CROP AND HERBICIDE ROTATION EFFECTS ON WEED POPULATION DYNAMICS AND THE CHARACTERIZATION OF IMIDAZOLINONE-RESISTANT SMOOTH PIGWEED (*Amaranthus hybridus*)

by

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(ABSTRACT)

Shifts in weed populations to herbicide-resistant biotypes are occurring more frequently. In two adjacent field studies from 1991 through 1994, crop rotations and herbicide programs affected control and densities of common lambsquarters, common ragweed, smooth pigweed, redroot pigweed, jimsonweed, goosegrass, stinkgrass, large crabgrass, smooth crabgrass, fall panicum, and yellow nutsedge. Generally, the continuous use of the same herbicide or herbicides with similar selectivities resulted in proliferation of tolerant weed species. Corn, tomato, and soybean yields were affected mostly by crop rotations, rainfall, and weed control. Herbicide rotations or combinations must include herbicides that are efficacious on the target weed species to preclude weed shifts.

Approximately 5 million smooth pigweed plants in Painter, VA were treated with imazethapyr or nicosulfuron from 1992 to 1994, and no ALS-inhibitor-resistant plants were identified. Smooth pigweed in Marion, MD and Oak Hall, VA, and livid amaranth in Warren County, NJ were reportedly escaping control from imazaquin or imazethapyr. In greenhouse studies, control of smooth pigweed from Marion and Oak Hall was 3 to 18%

by 560 or 1120 g ai/ha imazaquin. Control of smooth pigweed from Painter was 81% by 70 g ai/ha imazethapyr. Control of livid amaranth from New Jersey was 8 to 15% by 560 g/ha imazethapyr.

Field, greenhouse, and laboratory studies were conducted on Marion [resistant (R) biotype] and Painter [susceptible (S) biotype] smooth pigweed to characterize herbicide resistance in the R biotype. The R biotype was resistant at high levels to imazaquin and imazethapyr, and was cross-resistant at low levels to rimsulfuron and chlorimuron in the greenhouse. Both biotypes were equally susceptible to ASC-67040, CGA-152005, flumiclorac, halosulfuron, lactofen, metribuzin, nicosulfuron, pendimethalin, primisulfuron, pyrithiobac, and thifensulfuron in field or greenhouse studies. ALS enzyme assays confirmed target site-based resistance to imazaquin, imazethapyr, and rimsulfuron but not to chlorimuron in the R biotype. Metabolism of ^{14}C-chlorimuron was more rapid in the R than in the S biotype which may explain the low level of whole plant resistance to chlorimuron in the R biotype. The occurrence of herbicide resistance and patterns of cross-resistance in weeds cannot be predicted.

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Chapter I

Introduction and Literature Review

1. Weed shifts

The rise in importance of herbicides during the last 40 years has led to the continuous use of the same herbicide or family of herbicides in some cropping systems. The use of the same herbicide year after year increases selection pressures in the plant community for certain weed populations (30, 37). Selection pressures cause shifts in the weed community from one species to another (or to another biotype of the same species in the case of herbicide resistance) by eliminating susceptible weed species or biotypes from the existing population. As a result, tolerant species or resistant biotypes flourish and produce offspring (30, 46). As the tolerant weed species or resistant biotypes are repeatedly selected, they eventually dominate the population.

Weed population shifts have occurred most readily in the presence of a control measure that promotes high selection pressure on the population (9, 12, 13, 15, 16, 22, 28, 33, 39, 40, 53, 72, 74, 77). The control measure could be from an herbicide, a tillage operation, crop rotations, or other agronomic factors; but it must be used continuously in the management scheme to cause a shift in the weed population (28, 30, 47, 58). Perennial weeds and small-seeded annual weeds that germinate near the soil surface generally dominate weed populations under no-tillage systems; whereas annual weeds that germinate from various depths are favored by conventional tillage systems (9, 13, 15, 16, 40, 53, 72, 77). Crop rotations can also influence weed population shifts due to diverse

cultural practices, competitive ability, and herbicide use associated with different crops (9, 12, 13, 33, 39, 72, 74). Weed species shifts are highly dependent on the species present and often their susceptibility to the herbicides being used. Population fluctuations, rather than shifts, have been observed in response to various management strategies; but most often shifts are observed following the continuous use of a control measure (9, 13, 15, 19, 20, 22, 40, 53, 72, 77). Weed species shifts are most often observed in cropping systems in which the same herbicide or herbicides with similar selectivities are used continuously.

Increasingly over the past 25 years, selection pressures generated by an herbicide have selected a resistant biotype of the same species (28, 36, 37, 38, 42, 54). Resistance was first identified in the late 1960's when common groundsel (*Senecio vulgaris* L.) could no longer be controlled by atrazine [6-chloro-*N*-ethyl-*N'*-(1-methylethyl)-1, 3, 5-triazine-2, 4-diamine] (42, 60). Since that time, resistance in weeds has been identified to an increasing number of herbicides and herbicide families (23, 36, 37, 42, 54). Of particular interest is the relatively recent identification of resistance to acetolactate synthase (ALS)[1]-inhibitor herbicides with only 3 to 5 years of selection.

Sulfonylureas and imidazolinones are ALS-inhibitor herbicides that exhibit many of the characteristics that have promoted herbicide resistance in plants in the past (27, 28, 37, 47, 58). Various sulfonylurea herbicides are used commercially in corn (*Zea mays* L.), peanut (*Arachis hypogaea* L.), rice (*Oryza sativa* L.), small grains, soybean [*Glycine max* (L.)

[1] Abbreviations: ALS, acetolactate synthase; I_{50}, Inhibitor concentration required to cause a 50% reduction in enzyme activity; ST, sulfonylurea tolerant.

Merr.], fallow-land, and non-agricultural uses (8, 75). Rimsulfuron {*N*-[[(4,6-dimethoxy-2-pyrimidinyl)amino]carbonyl]-3-(ethylsulfonyl)-2-pyridinesulfonamide} is being developed for use in potato (*Solanum tuberosum* L.) and has selectivity in tomato (*Lycopersicon esculentum* L.) (1, 2). Various imidazolinone herbicides are used commercially in peanut, small grains, soybean, selected vegetable crops, and non-agricultural uses (6, 8). Genetic modification of crop plants allows the use of these herbicides in crops that were previously susceptible (24, 26, 50, 67, 68). Imidazolinone-resistant and -tolerant corn allows the use of imazethapyr {2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-5-ethyl-3-pyridinecarboxylic acid} in that crop. The development of sulfonylurea-tolerant (ST)[1] soybean allows the application of additional sulfonylurea herbicides as well as higher rates of previously registered sulfonylurea herbicides. Additional sulfonylurea and imidazolinone herbicides are being developed for use in corn, soybean, and some other crops (3, 5). Other commercial and experimental ALS-inhibitor herbicides for use in corn, soybean, and cotton (*Gossypium hirsutum* L.) belong to the triazolopyrimidine and pyrimidinyl thiobenzoate chemical families (4, 18). From this partial list of crops tolerant to ALS-inhibitors and the growing number of herbicides acting at this site of action, the potential for ALS-inhibitors to be used continuously in numerous crop rotations is high.

At least 17 weed species have biotypes resistant to one or more ALS-inhibitor herbicides (38, 45, 61). Most of these biotypes were selected under monoculture cropping systems in which the selection agent was a single herbicide used continuously in the system. Given

the likelihood for ALS-inhibitor herbicides to be used in each crop of a rotation, the probability for weed population shifts from a susceptible species (or biotype) to a naturally tolerant species (or a resistant biotype) is high.

2. Herbicide Susceptibility, Tolerance, and Resistance

Susceptibility, tolerance, and resistance are terms used to describe plant responses to herbicides. Susceptibility is defined as a lack of capacity of a plant to withstand herbicide treatment so that the plant is damaged by the herbicide (37). Tolerance has traditionally been defined as the ability of a plant to withstand herbicide treatment that may be overcome by high dosages (37). This response is likely due to naturally occurring variability within a species or larger taxonomic group, and is considered the wild type for that species or group. Resistance is generally defined as a decreased response of a population of plant species to an herbicide relative to the wild type of the same species as a result of the application of the herbicide (37, 46). The resistant population often withstands substantially higher concentrations of the herbicide relative to the wild type population. Two factors must be involved for resistance to develop in plant populations: heritable variation for the trait and natural selection (46). Natural selection generally involves multiple applications of an herbicide.

The number of herbicide-resistant weed species has increased exponentially over the last 15 years and continues to rise (36, 37, 38, 61). Herbicide resistance has generally developed under continuous selection pressure from an herbicide in a monoculture cropping system (36, 37, 38, 44, 56, 61). Herbicides select for these populations by

controlling the susceptible individuals and allowing resistant individuals to produce offspring and dominate the weed population in as little as 3 to 5 years (38, 44, 56, 61).

Little evidence exists to support the theory that herbicides can increase mutation rates in weeds and thereby confer herbicide resistance as a result of their application (46). Rather, resistant biotypes are presumed to be present in the weed population prior to the application of the herbicide, and continuous use of the herbicide selects the resistant biotype (30, 36, 37, 46). Much genetic and phenotypic variation occurs naturally in most weed populations, and it is conceivable that a mutation for resistance is present prior to the first application of an herbicide (35, 46).

Weed biotypes have been identified resistant to one or more of 14 herbicides or herbicide families, including the recent identification of biotypes resistant to the sulfonylurea and imidazolinone herbicides (36, 37, 61). The continuous use of sulfonylurea and imidazolinone herbicides, generally in monoculture cropping systems, has selected for biotypes of at least 17 weed species with resistance to ALS-inhibitor herbicides (36, 38, 41, 44, 45, 55, 56, 59, 61, 63, 71)[2]. This list includes prostrate pigweed (*Amaranthus blitoides* S. Wats.), redroot pigweed (*Amaranthus retroflexus* L.), Palmer amaranth (*Amaranthus palmeri* S. Wats.), and common waterhemp (*Amaranthus rudis* Sauer). Of these, only Palmer amaranth and common waterhemp have resistant populations in the United States. Selection for herbicide-resistant biotypes of *Amaranthus* spp. is likely to occur because of their generally high susceptibility to sulfonylurea and

[2] Barrentine, W.L. 1993. Personal communication. Delta Res. and Ext. Ctr., Stoneville, MS 38776.

imidazolinone herbicides and their generally high fecundity (11, 35, 43, 48, 51). Biotypes of smooth pigweed (*Amaranthus hybridus* L.) are resistant to the triazine herbicides (10, 29), and recently to ALS-inhibiting herbicides (45, 66).

Imazaquin {2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-3-quinolinecarboxylic acid} and imazethapyr {2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-5-ethyl-3-pyridinecarboxylic acid} are selective imidazolinone herbicides for soybean which control numerous broadleaf and certain grass weeds. The mode of action of these herbicides is the inhibition of ALS (EC 4.1.3.18) (also called acetohydroxyacid synthase), the first common enzyme in the biosynthesis of the branched chain amino acids - valine, leucine, and isoleucine (34, 64, 65, 69, 71). Since their commercial introduction in the late 1980's (75), imazaquin and imazethapyr use increased to 47% of the soybean area in the major soybean producing states by 1992 (78). There are three other classes of commercially available herbicides that inhibit ALS: the sulfonylureas, the triazolopyrimidines, and the pyrimidinyl thiobenzoates (25, 34, 57, 61, 72). Soybean is planted on over 50% of the harvested cropland on Virginia's portion of the Delmarva, and many farmers on the Delmarva grow soybean continuously (7). In areas with similar cultural practices, the likelihood of imazaquin, imazethapyr, or another ALS-inhibitor to be used year after year is high.

Of the ALS-inhibitor-resistant weed populations, only common cocklebur (*Xanthium strumarium* L.), Palmer amaranth, and common waterhemp populations developed from imidazolinone herbicide use. All other ALS-inhibitor-resistant biotypes were selected with

sulfonylurea herbicides. One biotype of rigid ryegrass (*Lolium rigidum* Gaudin), which was selected with diclofop {(±)-2-[4-(2,4-dichlorophenoxy)phenoxy]propanoic acid}, is an exception (44, 55, 56, 61).

Tolerance to the sulfonylurea and imidazolinone herbicides is usually due to enhanced metabolism in the tolerant plant species relative to the susceptible species; although differential uptake can contribute to this tolerance (14, 49, 52, 61, 72, 75, 78). Tolerant soybean seedlings metabolized chlorimuron with a half-life of only 1 to 3 h, while two susceptible weed species, common cocklebur and redroot pigweed, metabolized chlorimuron with half-lives of greater than 30 h (14). Metabolism of nicosulfuron in tolerant corn was nearly complete (above 90%) by 20 h after application (52, 70). However, metabolism of nicosulfuron in susceptible johnsongrass was not detectable by 24 h after application and was less than 30% by 72 h after application (52). Differential metabolism is the mechanism of resistance to ALS-inhibitors in an acetyl coenzyme A carboxylase-inhibitor-resistant biotype of rigid ryegrass (17, 61). However, all of the other ALS-inhibitor-resistant weed biotypes, in which the resistance mechanism has been determined, are resistant because of an altered ALS that is insensitive to the herbicides (17, 21, 61, 62, 63, 72, 73).

3. Herbicide Cross Resistance and Multiple Resistance

The most widely accepted and most effective, mechanistically based definitions of herbicide cross resistance and multiple resistance are presented here. Cross resistance is defined as the expression of a mechanism that endows the ability to withstand herbicides

from different chemical classes (31). It may be conferred by a single gene, or by two or more genes influencing a single mechanism. The most common form of cross resistance is a modification of the target site of an herbicide which also confers resistance to herbicides from a different chemical class with the same site of action. An example is the selection of aryloxyphenoxypropionic acid herbicide- resistant acetyl-coenzyme A carboxylase which is also less sensitive to cyclohexanedione herbicides. Another example is the selection of triazine resistant D1 protein that is also less sensitive to triazinones. A third example is the selection of sulfonylurea resistant ALS which is also less sensitive to other classes of ALS-inhibitors.

The ALS-inhibitor-resistant weed biotypes documented to date are often cross-resistant at varying levels to members of the same herbicide family as the selection agent, but display varying patterns of cross-resistance to members of other ALS-inhibitor herbicide families (21, 32, 38, 44, 55, 56, 61, 63). The ratios (resistant/susceptible) of the inhibitor concentration required to cause a 50% reduction in enzyme activity (I_{50})[1] for ALS from kochia [*Kochia scoparia* (L.) Schrad.], which was selected with chlorsulfuron, were 5 to 30 for sulfonylureas and triazolopyrimidines (61). The I_{50} ratios (resistant/susceptible) for ALS from this biotype of kochia were 2 to 6 for the imidazolinones. The I_{50} ratios (resistant/susceptible) for ALS from perennial ryegrass (*Lolium perenne* L.), which was selected with chlorsulfuron, were 8 to 80 for the sulfonylureas and triazolopyrimidines; and were >2 to >20 for the imidazolinones (61). The I_{50} ratios (resistant/susceptible) for ALS from three populations of common chickweed [*Stellaria media* (L.) Vill.], which

were selected with chlorsulfuron, were 7 to >300 for the sulfonylureas and triazolopyrimidines, and were 2 to 7 for the imidazolinones (61). The I_{50} (resistant/susceptible) ratios for ALS from common cocklebur, which was selected with imazaquin, were >40 for imazaquin and 1 for the sulfonylureas and triazolopyrimidines (61). The varying patterns of cross-resistance suggest that each ALS-inhibitor-resistant weed population should be analyzed separately for its level of resistance and its patterns of cross-resistance.

A less common form of cross resistance is that conferred by a mechanism other than resistant enzyme target sites (31). These resistance mechanisms can include reduced herbicide uptake, reduced translocation, reduced herbicide activation, enhanced herbicide detoxification, changes in intra- or intercellular compartmentation, and enhanced repair of herbicide-induced damage. This form of cross resistance has occurred to date only in blackgrass (*Alopecurus myosuroides* Huds.) and rigid ryegrass.

Multiple resistance is defined as the expression (within individuals or populations) of more than one resistance mechanism which confers the ability to withstand herbicides from different chemical classes (31). Multiple resistance is also rare, having been confirmed only in blackgrass, and rigid ryegrass. Blackgrass and rigid ryegrass biotypes are resistant to acetyl coenzyme A carboxylase inhibitor herbicides, ALS-inhibitors, mitotic inhibitors, photosystem II inhibitors, substituted ureas, the chloroacetamide herbicides, the isoxalolidonones, the 1-4 triazoles and the carbamothioates. Managing these weed populations is complex given the limited number of effective tools available to control

these grass weeds.

4. Objectives of This Research

The objective of the first portion of this research was to determine the effects of crop rotations and herbicide programs on weed populations over a four year period. One herbicide program was a lack of herbicide mode of action rotation. This program included no herbicide rotation or rotations of herbicides with the same mode of action. Other herbicide programs were rotations of herbicides with different modes of action and combinations of herbicides with different modes of action. A secondary objective of these studies was to monitor the plots for the development of herbicide resistant weed populations.

Several populations of *Amaranthus* spp. were identified which were escaping control by imidazolinone herbicides in fields where the herbicides were initially effective. There were several objectives for the second portion of this research. The first objective was to determine if herbicide resistant individuals were present in a known ALS-inhibitor susceptible population of smooth pigweed. The second objective was to determine if the aforementioned *Amaranthus* spp. populations were controlled by high rates of imidazolinone or sulfonylurea herbicides. The third objective was to confirm and characterize herbicide resistance and cross resistance in a population of smooth pigweed. The final objective was to determine the mechanism (or mechanisms) involved in herbicide resistance and cross-resistance if it occurred in this population of smooth pigweed.

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Chapter II

Crop Rotation and Herbicide Program Effects on Populations of Several Broadleaf Weeds

Abstract. Studies from 1991 through 1994 investigated the effects of several crop rotations and herbicide programs on populations of common lambsquarters, common ragweed, redroot pigweed, smooth pigweed, and jimsonweed at two adjacent sites. The effects on crop yields were also determined. In a split-plot arrangement of treatments, crop rotations were the main plots and included continuous corn, continuous soybean, corn-soybean, and corn-tomato-soybean. Herbicide programs were the split-plots and included continuous use of acetolactate synthase (ALS)-inhibitor herbicides, continuous use of non-ALS-inhibitor herbicides, annual rotations between ALS- and non-ALS-inhibitor herbicides, combinations of ALS- and non-ALS-inhibitor herbicides in the same year, and no herbicide. Weed control and weed population densities generally were affected by an interaction between crop rotations and herbicide programs. After 4 years, common lambsquarters control was lowest and densities were highest where fomesafen was used alone continuously for 4 years or in rotation with other herbicides. Although common ragweed densities were low at site two, control was generally lowest from treatments that included only ALS-inhibitor herbicides at both sites. Common ragweed densities were highest at site one in 1992 and 1993 following continuous applications of ALS-inhibitor herbicides. Jimsonweed densities were also low at site two, but control at

site one in tomato was low. Jimsonweed control from fomesafen and the combination of butylate plus atrazine in soybean and corn, respectively, was variable. Pigweed spp. densities decreased as the study progressed, and in 1993, control was over 90% from all treatments, except the combination of butylate plus atrazine. Corn and soybean yields varied with year and site, and yields of these crops and tomato were related to rainfall and weed control. **Nomenclature:** Atrazine, 6-chloro-*N*-ethyl-*N'*-(1-methylethyl)-1,3,5-triazine-2,4-diamine; butylate, *S*-ethyl bis(2-methylpropyl)carbamothioate; fomesafen, 5-[2-chloro-4-(trifluoromethyl)phenoxy]-*N*-(methylsulfonyl)-2-nitrobenzamide; common lambsquarters, *Chenopodium album* L. #[1] CHEAL; common ragweed, *Ambrosia artemisiifolia* L. # AMBEL; jimsonweed, *Datura stramonium* L. # DATST; redroot pigweed, *Amaranthus retroflexus* L. # AMARE; smooth pigweed, *Amaranthus hybridus* L. # AMACH; corn, *Zea mays* L. 'ICI 8532IT' in 1991, 1992 and 1993, and 'Pioneer 3245IR' in 1994; soybean, *Glycine max* (L.) Merr. 'W20-STS' in 1991, 'STS-9122' in 1992, 'Asgrow 3200 STS' in 1993, and 'Asgrow 4045 STS' in 1994; tomato, *Lycopersicon esculentum* L. 'Floradade'.

Additional index words: ALS-inhibitor herbicides, crop rotations, herbicide programs, herbicide rotations, imidazolinone herbicides, sulfonylurea herbicides, weed shifts, atrazine, butylate, fenoxaprop, fluazifop-P, fomesafen, imazaquin, imazethapyr, metribuzin, nicosulfuron, rimsulfuron, trifluralin, *Amaranthus hybridus, Amaranthus*

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[1] Letters following this symbol are a WSSA-approved computer code from Composite List of Weeds, Revised 1989. Available from WSSA, 1508 West University Ave., Champaign, IL 61821-3133.

retroflexus, Ambrosia artemisiifolia, Chenopodium album, Datura stramonium, Glycine max, Lycopersicon esculentum, Zea mays, AMACH, AMARE, AMBEL, CHEAL, DATST.

INTRODUCTION

The rise in importance of herbicides during the last 40 years has led to the continuous use of the same herbicide or family of herbicides in some cropping systems. The use of the same herbicide year after year increases selection pressures in the plant community for certain weed populations (24, 27). Selection pressures cause shifts in the weed community from one species to another (or to another biotype of the same species in the case of herbicide resistance) by eliminating susceptible weed species or biotypes from the existing population and allowing tolerant species or resistant biotypes to flourish and produce offspring (24, 33). As the tolerant weed species or resistant biotypes are repeatedly selected, they eventually dominate the population.

Weed population shifts have occurred most readily in the presence of a control measure that promotes high selection pressure on the population (8, 9, 10, 11, 12, 17, 23, 25, 29, 30, 36, 44, 45, 47). The control measure could be from an herbicide, a tillage operation, crop rotations, or other agronomic factors, but it must be used continuously in the management scheme to cause a shift in the weed population (23, 24, 34, 38). Perennial weeds and small-seeded annual weeds that germinate near the soil surface generally

dominate weed populations under no-tillage systems. Annual weeds that germinate from various depths are favored by conventional tillage systems (8, 10, 11, 12, 30, 36, 44, 47). Crop rotations can also influence weed species shifts due to diverse cultural practices, competitive ability, and herbicide use associated with different crops (8, 9, 10, 25, 29, 44, 45). Weed species shifts are highly dependent on the species present and often their susceptibility to the herbicides being used. Population fluctuations rather than shifts have been observed in response to various management strategies, but most often shifts are observed following the continuous use of a control measure (8, 10, 11, 14, 15, 17, 30, 36, 44, 47). Weed species shifts are most often observed under monoculture cropping systems in which the same herbicide or herbicides with similar selectivities are used continuously.

Increasingly over the past 25 years, selection pressures generated by an herbicide have selected for a resistant biotype of the same species (23, 26, 27, 28, 31, 37). Resistance was first identified in the late 1960's when common groundsel (*Senecio vulgaris* L.) could no longer be controlled by atrazine (31, 39). Since that time, resistance in weeds has been identified to an increasing number of herbicides and herbicide families (18, 26, 27, 31, 37). Of particular interest is the relatively recent identification of resistance to ALS-inhibitor herbicides with only 3 to 5 years of selection.

Sulfonylureas and imidazolinones are ALS-inhibitor herbicides that exhibit many of the characteristics that have promoted herbicide resistance in plants in the past (22, 23, 27, 34, 38). Various sulfonylurea herbicides are used commercially in corn, peanut (*Arachis*

hypogaea L.), rice (*Oryza sativa* L.), soybean, small grains, fallow-land, non-agricultural uses (7, 46). Rimsulfuron {*N*-[[(4,6-dimethoxy-2-pyrimidinyl)amino]carbonyl]-3-(ethylsulfonyl)-2-pyridinesulfonamide} is being developed for use in potato (*Solanum tuberosum* L.) and has selectivity in tomato (1, 2). Various imidazolinone herbicides are used commercially in peanut, small grains, soybean, selected vegetable crops, and non-agricultural uses (6, 7). Genetic modification of crop plants allows the use of these herbicides in crops that were previously susceptible (19, 21, 35, 42, 43). Imidazolinone-resistant and -tolerant corn allows the use of imazethapyr {2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-5-ethyl-3-pyridinecarboxylic acid} in that crop, and the development of sulfonylurea-tolerant (ST)[2] soybean allows the application of additional sulfonylurea herbicides as well as higher rates of previously registered sulfonylurea herbicides. Additional sulfonylurea and imidazolinone herbicides are being developed for use in corn, soybean, and other crops (3, 5). Other commercial and experimental ALS-inhibitor herbicides for use in corn, soybean, and cotton (*Gossypium hirsutum* L.) belong to the triazolopyrimidine and pyrimidinyl thiobenzoate chemical families (4, 13). From this partial list of crops tolerant to ALS-inhibitors and the growing number of herbicides acting at this site of action, the potential for ALS-inhibitors to be used continuously in numerous crop rotations is high.

At least 17 weed species have biotypes identified to be resistant to one or more ALS-

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[2] Abbreviations: CC, continuous corn; CS, corn-soybean; CTS, corn-tomato-soybean; EPOST, early postemergence; LPOST, late postemergence; SS, continuous soybean; ST, sulfonylurea tolerant.

inhibitor herbicides (28, 32, 40, 41). Most of these biotypes were selected under monoculture cropping systems in which the selection agent was a single herbicide used continuously in the system. Given the likelihood for ALS-inhibitor herbicides to be used in each crop of a rotation, the probability for weed population shifts from a susceptible species (or biotype) to a tolerant species (or a resistant biotype) is high.

The first objective of this research was to determine the effects of crop rotations and herbicide programs on population dynamics of common lambsquarters, common ragweed, pigweed spp., and jimsonweed. A secondary objective of this research was to monitor the plots for the development of herbicide-resistant weed populations.

MATERIALS AND METHODS

Field studies were conducted in separate, adjacent sites in 1991, 1992, 1993, and 1994 on a State sandy loam soil (Typic Hapludults) at Painter, VA. Soil organic matter was 1%, and pH was 6.2 to 6.4 and 5.9 to 6.3 at sites one and two, respectively. Studies were conducted under a conventional tillage system. The seedbed was prepared with a chisel plow followed by tandem disking two to three times, and was tilled twice with a field cultivator containing double rolling baskets after application of PPI herbicides. In 1993, an 8.6-cm rain following incorporation of the PPI herbicides delayed crop planting 11 d. Therefore, both sites were tilled two more times with the field cultivator to control weeds that had germinated during this time. Fertilizer was applied to all crops according to

Virginia Polytechnic Institute and State University recommendations (16).

Experiments were conducted using a randomized complete block design with a split-plot arrangement of treatments and four replications. Crop rotations were the main plots and included continuous corn (CC)[2], continuous soybean (SS)[2], corn-soybean (CS)[2], and corn-tomato-soybean (CTS)[2]. The CTS rotation was not included at site two. Herbicide mode of action programs were the split-plots and included continuous use of ALS-inhibitor herbicides, continuous use of non-ALS-inhibitor herbicides, annual rotation between ALS-inhibitor and non-ALS-inhibitor herbicides, combinations of ALS-inhibitor and non-ALS-inhibitor herbicides in the same year, and no herbicide. Split-plots were 7.6 by 12.2 m in 1991 and 7.6 by 10.7 m in 1992, 1993, and 1994. These relatively large split-plots were used to minimize effects of weed seed movement between plots. Data were collected from the center 4.6 by 7.6 m of each split plot.

Corn and soybean rows were spaced 76 cm apart, and tomato rows were spaced 1.5 m apart. Corn varieties were imidazolinone-tolerant ICI 8532IT planted at 56,800 seeds/ha on May 17, 1991, May 14, 1992 and 59,300 seeds/ha on May 24, 1993; and imidazolinone-resistant Pioneer 3245IR planted at 59,300 seeds/ha on May 10, 1994. Imidazolinone-tolerant or -resistant varieties were included to permit the application of imazethapyr as the ALS-inhibitor herbicide for corn. Soybean varieties were sulfonylurea tolerant (ST)[2] - W-20 STS in 1991, STS-9122 in 1992, Asgrow 3200 STS in 1993, and Asgrow 4045 STS in 1994 planted at approximately 60 kg/ha each year on approximately the same dates corn was planted. ST varieties were included to permit the application of

nicosulfuron {2-[[[[(4,6-dimethoxy-2-pyrimidinyl)amino]carbonyl]amino]sulfonyl]-*N,N*-dimethyl-3-pyridinecarboxamide} for grass control as part of the ALS-inhibitor herbicide program for soybean. Floradade tomato were transplanted 46 cm apart in the row on May 18, 1992 with a commercial one row transplanter. Floradade tomato was selected because of its popularity[3] and availability.

Herbicides were broadcast with a commercial tractor-mounted sprayer which had an effective spray width of 7.6 m. The sprayer dispersed a water carrier at 230 L/ha with a pressure of 207 kPa through flat fan nozzles[4]. PPI herbicides were applied on May 16, 1991, May 12, 1992, May 12, 1993, and May 9, 1994. Application dates of early postemergence (EPOST)[2] and late postemergence (LPOST)[2] herbicides varied with treatment and year, but were based on weed size of less than 10 cm (usually less than 5 cm) and 10 to 20 cm for EPOST and LPOST, respectively.

The herbicides used in the three crops to form the herbicide programs are listed in Table 1 with their modes of action, application timings, and rates. The continuous ALS-inhibitor, continuous non-ALS-inhibitor, and combination herbicide programs are detailed in the table. Other herbicide programs included an annual rotation, which was a rotation between the ALS-inhibitor component(s) and the non-ALS-inhibitor component(s), and an untreated control. Imazethapyr was selected as the ALS-inhibitor component, and

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[3] Wilson, H. P. 1992. Personal communication. Eastern Shore Agric. Res. and Ext. Center, Virginia Polytech. Inst. and State Univ., Painter.

[4] Teejet 8003 flat fan spray nozzles, Spraying Systems Co., North Ave. Wheaton, IL 60188.

butylate and atrazine were selected as non-ALS-inhibitor components for corn. Imazaquin {2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-3-quinolinecarboxylic acid} and nicosulfuron were selected as ALS-inhibitor components; and fomesafen, fluazifop-P {(R)-2-[4-[[5-(trifluoromethyl)-2-pyridinyl]oxy]phenoxy]propanoic acid}, and fenoxaprop {(±)-2-[4-[(6-chloro-2-benzoxazolyl)oxy]phenoxy]propanoic acid} were selected as non-ALS-inhibitor components for soybean. Fomesafen was always applied as a mixture (1991) or sequentially (1992 to 1994) with fluazifop-P plus fenoxaprop. Fluazifop-P plus fenoxaprop was a prepackage mixture in 1992, 1993, and 1994. Rimsulfuron was the ALS-inhibitor component, and trifluralin [2,6-dinitro-*N,N*-dipropyl-4-(trifluoromethyl)benzenamine] and metribuzin [4-amino-6-(1,1-dimethylethyl)-3-(methylthio)-1,2,4-triazin-5(4*H*)-one] were the non-ALS-inhibitor components for tomato. Herbicides were applied at commercial rates or an anticipated commercial rate for rimsulfuon. Methylated seed oil[5] was applied at 1% v/v with imazethapyr and nicosulfuron. Crop oil concentrate[6] was applied at 1% v/v with fomesafen plus fluazifop-P plus fenoxaprop. A non-ionic surfactant[7] was applied at 0.25% v/v with rimsulfuron

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[5] Sun-it II or Scoil, methylated oil additive, AGSCO, Inc. Grand Forks, ND 58206-0458.

[6] Agridex, a mixture of 83% paraffinic mineral oil and 17% polyoxyethylene sorbitan fatty acid ester, Helena Chemical Co. 5100 Poplar Ave. Memphis, TN 38137.

[7] Ortho X-77, Valent U.S.A. Corp., 1333 N. California Blvd., Walnut Creek, CA 94596. Nonionic surfactant with 80% principal functioning agents as: alkylarylpolyoxy ethylene glycols, free fatty acids, and isopropanol.

plus metribuzin.

Visual estimates of percent weed control were made 89 ± 11 days after the PPI treatments were applied each year. Weed control was based on a scale of 0 to 100% where 0 equals no control and 100 equals complete control (death of plants) compared to an untreated control (20). Variability in rating dates had little or no effect on weed control ratings since these were late season ratings. Weed population densities were determined by species 48 ± 14 days after the PPI treatments were applied each year at site one. Densities were determined 54 ± 11 days after the PPI treatments were applied each year at site two. Weed densities were determined by counting the number of plants in a 50 by 50 cm area at three random locations in each split-plot. Weed densities were determined when weeds were 20 to 50 cm tall. This timing ensured that a majority of viable seeds had germinated, and minimized population reductions from competition. Corn yields were determined on October 24, 1991, October 1, 1992, October 1, 1993, and September 28, 1994 by harvesting the center six rows of each ten-row split-plot with a commercial harvester. Soybean yields were determined October 24, 1991; October 2 (site two) and October 16 (site one), 1992; November 13, 1993; and October 11, 1994 by harvesting the center four rows of each ten-row split-plot with a commercial harvester. Tomato yields were determined at site one on July 30, 1992 by hand harvesting and weighing all of the fruit present on the plants from 3 m of the center row of each split-plot.

Statistical analyses. The three population counts by weed species from each split plot were averaged prior to analyses. Data were analyzed statistically by ANOVA, and means

were separated using the appropriate Fisher's LSD test at the 0.05 level.

RESULTS AND DISCUSSION

Moisture varied with year and rainfall totals for the growing season were low in 1991 and 1993 (Table 2). Rainfall in May, 1991 was only 2 cm, and weed control was generally low that year. Rainfall in July and August, 1993 was low, but high rainfall amounts during May and early June resulted in generally good activity from all herbicides. Additionally, 2.5 cm irrigation was applied in August, 1993 to relieve moisture stress that developed as a result of low rainfall during July and August.

Increases in weed densities generally occurred with all species when control from herbicides was low, and shifts were often less severe when herbicides were rotated annually. Weed control was generally affected by an interaction of crop rotations and herbicide programs or by herbicide programs alone. Where the interaction of crop rotations and herbicide programs was significant, the means represent the individual treatments and are presented with the appropriate LSD value. Where only the main effect of herbicide programs was significant, the means represent the herbicide program averaged over the crop rotations and are presented with the appropriate LSD value.

Common lambsquarters. The interaction of crop rotations and herbicide programs was significant for control (Table A.1) and common lambsquarters densities (Table A.2) in all years at both sites. Common lambsquarters control from butylate plus atrazine, treatments

including imazaquin, and trifluralin plus metribuzin was generally 90% or higher (Table 3). Control at both sites from fomesafen plus fluazifop-P plus fenoxaprop was always low, but was improved by the addition of imazaquin and by rotating with other herbicides. Similarly, rotating with fomesafen plus fluazifop-P plus fenoxaprop often reduced common lambsquarters control from other herbicides. For example, common lambsquarters control in 1994 from butylate plus atrazine in the CTS rotation was 79% where it followed fomesafen plus fluazifop-P plus fenoxaprop compared with 90% where it followed imazaquin plus nicosulfuron. In the CS rotation, common lambsquarters control in 1993 from imazethapyr averaged 56% at both sites where it was rotated with fomesafen plus fluazifop-P plus fenoxaprop compared with an average 67% where it was rotated with imazaquin plus nicosulfuron. Control from imazethapyr in 1993 at site one was also lower in the CS rotation where it was rotated with fomesafen plus fluazifop-P plus fenoxaprop than in CC, whether it was applied continously or in rotation with butylate plus atrazine. Common lambsquarters control from imazethapyr alone in CC averaged 14% in 1991, but averaged 70 to 85% at both sites from 1992 to 1994 likely due to rainfall received prior to and following applications in 1992, 1993, and 1994 (Table 2). However, the addition of butylate to imazethapyr improved control in all but one year at both sites (Table 3).

Common lambsquarters densities generally increased in all crop rotations as the studies progressed, except in 1993 (Table 4). By 1994, densities were higher in the SS untreated control than in other crop rotation untreated controls at site one. However, there were no

differences among the untreated controls at site two. High common lambsquarters densities were generally associated with the inclusion of fomesafen plus fluazifop-P plus fenoxaprop. In 1991, common lambsquarters densities were 31 and 78 plants/m^2 at sites one and two, respectively from fomesafen plus fluazifop-P plus fenoxaprop. By 1994, densities were 189 and 218 plants/m^2 at sites one and two, respectively, where fomesafen plus fluazifop-P plus fenoxaprop was applied continuously in the SS rotation. Densities were also high in 1994 in the CS rotation where fomesafen plus fluazifop-P plus fenoxaprop was applied in rotation with imazethapyr. Densities in 1994 at site two were lower in the SS rotation where fomesafen plus fluazifop-P plus fenoxaprop was rotated with imazaquin plus nicosulfuron than in the CS rotation where fomesafen plus fluazifop-P plus fenoxaprop was rotated with imazethapyr. The difference between these treatments in 1994 at site one was not significant. However, control from imazethapyr was high enough to keep common lambsquarters densities below 40 plants/m^2 after 1991 where it was applied continuously. Densities were lower, an average 66 plants/m^2, where fomesafen plus fluazifop-P plus fenoxaprop was rotated with imazaquin plus nicosulfuron in the SS rotation or with butylate plus atrazine in the CS rotation, compared with an average 204 plants/m^2 where fomesafen plus fluazifop-P plus fenoxaprop was applied for 4 consecutive years in SS. Higher control from imazaquin plus nicosulfuron and butylate plus atrazine compared with imazethapyr may explain generally higher densities where fomesafen plus fluazifop-P plus fenoxaprop was rotated with imazethapyr compared with where they were rotated with either combination.

Common ragweed. Visual estimates of percent control of common ragweed were not made in 1991 since population densities were low and inconsistent (Table 6). However, densities were higher and more consistent in subsequent years. The interaction of crop rotations and herbicide programs was significant for control in all years at both sites (Table A.1). Common ragweed control was generally 90% or higher from fomesafen plus fluazifop-P plus fenoxaprop in all years except in the CTS rotation in 1993, when it was below 85% (Table 5). Generally, control from ALS-inhibitor herbicides was not adequate (less than 75%), and control from imazaquin plus nicosulfuron was always lowest. Common ragweed control from other herbicides was variable. Common ragweed control from imazaquin plus nicosulfuron was sometimes higher where the combination was rotated with other herbicide programs. For example, in SS at site two in 1993, control from imazaquin plus nicosulfuron was 51% where the combination was rotated with fomesafen plus fluazifop-P plus fenoxaprop, but was 39% where the combination was used continuously. Common ragweed control from these treatments did not differ at site one in 1993. Control from imazaquin plus nicosulfuron was higher in 1994 at site two, but not at site one, where the combination was rotated with imazethapyr in the CS rotation than where the combination was applied annually in SS. However, common ragweed control from continuous applications of imazaquin plus nicosulfuron averaged 58% in 1994 compared with 25% in 1992 and 1993.

The interaction of crop rotations and herbicide programs was significant in 1991 and 1994 at both sites and 1993 at site two for common ragweed densities (Table A.2). The

main effect of herbicide programs was significant in 1992 at both sites and 1993 at site one (Table A.2). Common ragweed densities were low and variable at both sites in 1991 and at site two in 1992, 1993, and 1994; and there were few differences between treatments. In 1992, densities were highest in the untreated controls at both sites, and were higher from the continuous ALS-inhibitor program than from other herbicide programs at site one. Common ragweed densities were 45 plants/m^2 in the untreated controls at site one in 1993. There were 25 plants/m^2 from the continuous ALS-inhibitor program and 13 plants/m^2 from the annual rotation program in which ALS-inhibitor herbicides were applied that year. These values compare with 1 and 8 plants/m^2 from the continuous non-ALS-inhibitor and combination programs, respectively at site one in 1993. At site one in 1994, average densities were 41 plants/m^2 in the untreated controls and 26 plants/m^2 from the continuous ALS-inhibitor program in the CTS rotation. Densities at site one from continuous ALS-inhibitor programs in other crop rotations declined from 25 plants/m^2 in 1993 to an average of 8 plants/m^2 in 1994. The decline in common ragweed densities in 1994 from these treatments, particularly where imazaquin plus nicosulfuron was applied continuously for 4 years, is likely due to higher control in 1994 (Table 5).

Pigweed. Pigweed species included smooth and redroot pigweed, but were evaluated as pigweed spp. because the two species are difficult to distinguish, particularly at early growth stages. The interaction of crop rotations and herbicide programs was significant in 1991 at site one and in 1992 and 1993 at both sites for control (Table A.1). The main effect of herbicide programs was significant in 1991 at site two (Table A.1). Control of

jimsonweed densities was significant in 1992 at both sites and in 1993 at site one; otherwise the main effect of herbicide programs was significant (Table A.2). Visual control estimates were not made at site two in 1992 due to low jimsonweed densities (Table 10). Jimsonweed control and densities fluctuated for most herbicides or herbicide combinations, except that imazethapyr consistently gave 90% or higher jimsonweed control. All herbicides or herbicide combinations applied to tomato at site one in 1992 controlled less than 50% of jimsonweed, and densities in tomato were generally high. Control in 1994 was 90% or higher from all treatments likely due to low jimsonweed densities.

Crop injury. Initial corn injury did not exceed 15% from any herbicide in any year, and corn recovered quickly from any initial injury (data not presented). Fomesafen injured soybean 14 to 35% depending on year, but soybean recovered quickly from fomesafen injury (data not presented). Nicosulfuron injured soybean 24 to 45% depending on year. Injury symptoms and growth reduction from nicosulfuron treatments were evident through much of the season (data not presented). Tomato injury from the split application of rimsulfuron (15%) was higher than from a single application of rimsulfuron in combination with metribuzin following PPI trifluralin (9 %), but tomato recovered quickly from rimsulfuron injury (data not presented). Tomato injury from trifluralin followed by metribuzin did not exceed 6%.

Crop yields. Yields of corn, soybean, and tomato were analyzed separately, and the interaction of crop rotations and herbicide programs was significant at both sites in all

Soybean yields were often related to weed control. The combination of imazaquin plus fomesafen plus fluazifop-P plus fenoxaprop consistently provided the highest overall weed control, and when yields differed within a crop rotation, those from soybean receiving this combination were highest (Table 11). Few consistent differences occurred among herbicide programs or crop rotations for soybean yields from 1991 to 1993. Within the SS rotation, soybean receiving fomesafen plus fluazifop-P plus fenoxaprop in 1994 gave higher yields where the combination was rotated with imazaquin plus nicosulfuron than where it was applied for 4 consecutive years. Soybean receiving fomesafen plus fluazifop-P plus fenoxaprop every other year in the CS rotation gave higher yields in 1994 than those receiving the combination for 4 consecutive years in SS. Soybean receiving imazaquin plus fomesafen plus fluazifop-P plus fenoxaprop in 1994 gave higher yields in the CS rotation where the combination was rotated with other herbicides than in SS where the combination was applied for 4 consecutive years. Lower soybean yields from imazaquin plus nicosulfuron treatments compared with imazaquin plus fomesafen plus fluazifop-P plus fenoxaprop treatments may be due to lower weed control from imazaquin plus nicosulfuron rather than soybean injury from nicosulfuron, since yield differences were not always significant. Higher soybean yields where crops or herbicide programs were rotated are also likely due to a combination of weed control and other beneficial cultural factors associated with crop rotations.

Tomato yields in 1992 were highest from the split application of rimsulfuron in the continuous ALS-inhibitor program and from trifluralin plus metribuzin in the continuous

non-ALS-inhibitor program. Yields from tomato treated with trifluralin plus metribuzin in the continuous non-ALS-inhibitor program, following the application of butylate plus atrazine to corn, were higher than those from tomato treated with trifluralin plus metribuzin in the annual rotation program or with trifluralin plus metribuzin plus rimsulfuron in the combination program. Both of the latter combinations followed applications of imazethapyr in corn.

In summary, weed control was generally affected by crop rotations and herbicide programs or by herbicide programs alone. Weed species shifts to tolerant species were observed where the same herbicides or herbicides with similar selectivities were used continuously, as seen in previous research (8, 17, 24, 29, 43, 44). Weed shifts that occurred were shifts to tolerant species rather than herbicide resistant biotypes. These results indicate the importance of rotating to or including herbicides with high activity on the target weed species to delay or preclude weed population shifts.

Crop yields fluctuated with year due to rainfall amounts and weed control. Yields of corn and soybean treated with the same herbicide program were often increased by rotating the crop, and soybean yields were often high where overall weed control was high.

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Table 1. ALS- and Non-ALS-inhibitor herbicide components used in the specified crop to form the herbicide mode of action programs.

Crop	Mode of action	Herbicide(s)[a]	Timing[b]	Rate[c]
				g/ha
Corn	ALS-inhibitor	Imazethapyr	EPOST	70
	Non-ALS-inhibitor	Butylate + atrazine	PPI + PPI	3360 + 1120
	Combination[d]	Butylate + imazethapyr	PPI + EPOST	3360 + 70
Soybeans	ALS-inhibitor	Imazaquin + nicosulfuron	PPI + EPOST	140 + 35
	Non-ALS-inhibitor	Fomesafen + fluazifop + fenoxaprop[e]	EPOST + LPOST + LPOST	426 + 140 + 46
	Combination	Imazaquin + fomesafen + fluazifop + fenoxaprop	PPI + EPOST + LPOST + LPOST	140 + 426 + 140 + 46
Tomato	ALS-inhibitor	Rimsulfuron + rimsulfuron	10 DATP + 21 DATP	35 + 35
	Non-ALS-inhibitor	Trifluralin + metribuzin	PPI + 10 DATP	560 + 280
	Combination	Trifluralin + metribuzin + rimsulfuron	PPI + 10 DATP + 10 DATP	560 + 280 + 35

[a] Methylated seed oil was applied at 1% v/v with imazethapyr and nicosulfuron. Crop oil concentrate was applied at 1% v/v with fomesafen + fluazifop + fenoxaprop. A non-ionic surfactant was applied at 0.25% v/v with rimsulfuron and metribuzin.

[b] PPI = preplant incorporated; EPOST = early postemergence based on weed size of less than 10 cm (usually less than 5 cm); LPOST = late postemergence based on weed size of 10 to 20 cm; DATP = days after transplanting.

[c] Rates applied were commercial use rates or an anticipated commercial use rate for rimsulfuron.

[d] Combination refers to application of the components as a mixture, or sequential applications in the same year. The annual rotation mode of action program and untreated control are not included.

[e] Fomesafen was applied as a mixture (1991) or sequentially (1992 to 1994) with fluazifop + fenoxaprop.

Table 2. Monthly precipitation from April through August in 1991, 1992, 1993, and 1994 at Painter, VA.

Month	1991	1992	1993	1994
	cm			
April	8	6	9	3
May	2	12	12	7
June	6	8	8	7
July	7	13	2	17
August	7	21	3	9
Total	30	60	34	43

Table 3. Crop and herbicide rotation effects on common lambsquarters control from 1991 to 1994 at two sites in Painter, VA.[a]

Year	Crop[b]	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			%							
1991	C	Imazethapyr	13	81	71	85	15	86	68	84
1992	C	Imazethapyr								
1993	C	Imazethapyr								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	93	91	99	91	80	85	96	92
1992	C	Butylate + atrazine								
1993	C	Butylate + atrazine								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	15	94	68	83	14	93	68	90
1992	C	Butylate + atrazine								
1993	C	Imazethapyr								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	86	93	79	90	88	93	71	93
1992	C	Butylate + imazethapyr								
1993	C	Butylate + imazethapyr								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	0	0	0	0	0	0	0	0
1992	C	Untreated control								
1993	C	Untreated control								
1994	C	Untreated control								
1991	S	Imazaquin + nicosulfuron	85	99	98	97	83	92	99	99
1992	S	Imazaquin + nicosulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	S	Imazaquin + nicosulfuron								

[a] Visual control ratings were made 82, 100, 98, and 78 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively.
[b] Crops were: C, corn; S, soybeans; T, tomato.

Table 3 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			%							
1991	S	Fomesafen + fluazifop-P + fenoxaprop[c]	6	15	26	23	10	16	31	25
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + nicosulfuron	90	30	99	35	89	23	93	36
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + nicosulfuron								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop	76	89	91	91	64	86	86	87
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	S	Untreated control	0	0	0	0	0	0	0	0
1992	S	Untreated control								
1993	S	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	9	99	63	97	13	97	71	99
1992	S	Imazaquin + nicosulfuron								
1993	C	Imazethapyr								
1994	S	Imazaquin + nicosulfuron								
1991	C	Butylate + atrazine	93	25	96	34	81	25	92	44
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + atrazine								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	C	Imazethapyr	15	16	46	31	11	25	65	34
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Imazethapyr								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								

[c] Fomesafen was applied as a mixture (1991) or sequentially (1992 to 1994) with fluazifop-P + fenoxaprop.

Table 3 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			%							
1991	C	Butylate + imazethapyr	76	91	69	94	79	96	73	88
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + imazethapyr								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	C	Untreated control	0	0	0	0	0	0	0	0
1992	S	Untreated control								
1993	C	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	11	80	99	85	-[d]	-	-	-
1992	T	Rimsulfuron + rimsulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	92	97	73	79	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	11	95	99	90	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Imazaquin + nicosulfuron								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	89	95	93	93	-	-	-	-
1992	T	Trifluralin + metribuzin + rimsulfuron								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	0	0	0	0	-	-	-	-
1992	T	Untreated control								
1993	S	Untreated control								
1994	C	Untreated control								
LSD (0.05) (crop rotation x herbicide program)[e]			8	8	6	7	7	7	6	5

[d] The corn-tomato-soybean crop rotation was not included at site two.
[e] The LSD value is for the interaction of crop rotations and herbicide programs.

LSD (0.05) (crop rotation x herbicide program)[e]	25	38	20	51	46	62	23	74

[d] The corn-tomato-soybean crop rotation was not included at site two.
[e] The LSD value is for the interaction of crop rotations and herbicide programs.

Year	Crop[b]	Herbicide(s)	1991	1992	1993	1994				
			plants/m²							
1991	C	Imazethapyr	15	36	24	7	97	26	24	10
1992	C	Imazethapyr								
1993	C	Imazethapyr								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	1	2	0	0	7	5	1	0
1992	C	Butylate + atrazine								
1993	C	Butylate + atrazine								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	15	0	16	6	85	5	32	0
1992	C	Butylate + atrazine								
1993	C	Imazethapyr								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	16	9	14	1	43	2	26	1
1992	C	Butylate + imazethapyr								
1993	C	Butylate + imazethapyr								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	22	97	70	154	143	243	93	276
1992	C	Untreated control								
1993	C	Untreated control								
1994	C	Untreated control								
1991	S	Imazaquin + nicosulfuron	1	0	0	0	10	5	0	1
1992	S	Imazaquin + nicosulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	S	Imazaquin + nicosulfuron								

[a] Population counts were taken 35, 58, 37, and 63 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site one; and 43, 62, 47, and 64 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site two.
[b] Crops were: C, corn; S, soybeans; T, tomato.

Table 4 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m²							
1991	S	Fomesafen + fluazifop-P + fenoxaprop[c]	31	32	26	189	78	52	23	218
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + nicosulfuron	2	14	10	76	5	13	2	55
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + nicosulfuron								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop	5	9	2	1	27	13	0	4
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	S	Untreated control	10	137	83	286	133	292	128	317
1992	S	Untreated control								
1993	S	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	29	1	17	0	121	1	19	0
1992	S	Imazaquin + nicosulfuron								
1993	C	Imazethapyr								
1994	S	Imazaquin + nicosulfuron								
1991	C	Butylate + atrazine	4	19	2	54	7	25	0	80
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + atrazine								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	C	Imazethapyr	20	22	61	112	106	81	48	167
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Imazethapyr								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								

[c] Fomesafen was applied as a mixture (1991) or sequentially (1992 to 1994) with fluazifop-P + fenoxaprop.

Table 4 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m²							
1991	C	Butylate + imazethapyr	30	9	16	5	54	29	27	14
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + imazethapyr								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	C	Untreated control	43	124	123	234	129	280	73	247
1992	S	Untreated control								
1993	C	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	27	13	6	5	-[d]	-	-	-
1992	T	Rimsulfuron + rimsulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	2	1	5	6	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	18	1	0	1	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Imazaquin + nicosulfuron								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	14	2	0	2	-	-	-	-
1992	T	Trifluralin + metribuzin + rimsulfuron								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	12	124	71	189	-	-	-	-
1992	T	Untreated control								
1993	S	Untreated control								
1994	C	Untreated control								
LSD (0.05) (crop rotation x herbicide program)[e]			25	38	20	51	46	62	23	74

[d] The corn-tomato-soybean crop rotation was not included at site two.
[e] The LSD value is for the interaction of crop rotations and herbicide programs.

Table 5. Crop and herbicide rotation effects on common ragweed control from 1992 to 1994 at two sites in Painter, VA.[a]

Year	Crop[b]	Herbicide(s)	Site one			Site two		
			1992	1993	1994	1992	1993	1994
			%					
1991	C	Imazethapyr	61	60	70	71	73	73
1992	C	Imazethapyr						
1993	C	Imazethapyr						
1994	C	Imazethapyr						
1991	C	Butylate + atrazine	72	82	78	85	84	95
1992	C	Butylate + atrazine						
1993	C	Butylate + atrazine						
1994	C	Butylate + atrazine						
1991	C	Imazethapyr	61	58	75	84	76	94
1992	C	Butylate + atrazine						
1993	C	Imazethapyr						
1994	C	Butylate + atrazine						
1991	C	Butylate + imazethapyr	73	59	74	80	74	76
1992	C	Butylate + imazethapyr						
1993	C	Butylate + imazethapyr						
1994	C	Butylate + imazethapyr						
1991	C	Untreated control	0	0	0	0	0	0
1992	C	Untreated control						
1993	C	Untreated control						
1994	C	Untreated control						
1991	S	Imazaquin + nicosulfuron	12	26	56	23	39	59
1992	S	Imazaquin + nicosulfuron						
1993	S	Imazaquin + nicosulfuron						
1994	S	Imazaquin + nicosulfuron						

[a] Visual control ratings were made 100, 98, and 78 days after PPI treatments were applied in 1992, 1993, and 1994, respectively. Common ragweed populations were low and inconsistent in 1991, so visual control ratings were not made.

[b] Crops were: C, corn; S, soybeans; T, tomato.

Table 5 continued.

Year	Crop	Herbicide(s)	Site one			Site two		
			1992	1993	1994	1992	1993	1994
			%					
1991	S	Fomesafen + fluazifop-P + fenoxaprop[c]	99	90	97	99	95	97
1992	S	Fomesafen + fluazifop-P + fenoxaprop						
1993	S	Fomesafen + fluazifop-P + fenoxaprop						
1994	S	Fomesafen + fluazifop-P + fenoxaprop						
1991	S	Imazaquin + nicosulfuron	93	29	94	99	51	99
1992	S	Fomesafen + fluazifop-P + fenoxaprop						
1993	S	Imazaquin + nicosulfuron						
1994	S	Fomesafen + fluazifop-P + fenoxaprop						
1991	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop	99	89	96	99	95	99
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1991	S	Untreated control	0	0	0	0	0	0
1992	S	Untreated control						
1993	S	Untreated control						
1994	S	Untreated control						
1991	C	Imazethapyr	6	53	53	26	74	68
1992	S	Imazaquin + nicosulfuron						
1993	C	Imazethapyr						
1994	S	Imazaquin + nicosulfuron						
1991	C	Butylate + atrazine	99	76	94	99	88	99
1992	S	Fomesafen + fluazifop-P + fenoxaprop						
1993	C	Butylate + atrazine						
1994	S	Fomesafen + fluazifop-P + fenoxaprop						
1991	C	Imazethapyr	95	61	95	98	76	99
1992	S	Fomesafen + fluazifop-P + fenoxaprop						
1993	C	Imazethapyr						
1994	S	Fomesafen + fluazifop-P + fenoxaprop						

[c] Fomesafen was applied as a mixture (1991) or sequentially (1992 to 1994) with fluazifop-P + fenoxaprop.

Table 5 continued.

Year	Crop	Herbicide(s)	Site one			Site two		
			1992	1993	1994	1992	1993	1994
			%					
1991	C	Butylate + imazethapyr	97	66	98	98	80	99
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1993	C	Butylate + imazethapyr						
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1991	C	Untreated control	0	0	0	0	0	0
1992	S	Untreated control						
1993	C	Untreated control						
1994	S	Untreated control						
1991	C	Imazethapyr	51	28	65	-[d]	-	-
1992	T	Rimsulfuron + rimsulfuron						
1993	S	Imazaquin + nicosulfuron						
1994	C	Imazethapyr						
1991	C	Butylate + atrazine	40	80	73	-	-	-
1992	T	Trifluralin + metribuzin						
1993	S	Fomesafen + fluazifop-P + fenoxaprop						
1994	C	Butylate + atrazine						
1991	C	Imazethapyr	43	34	71	-	-	-
1992	T	Trifluralin + metribuzin						
1993	S	Imazaquin + nicosulfuron						
1994	C	Butylate + atrazine						
1991	C	Butylate + imazethapyr	74	84	73	-	-	-
1992	T	Trifluralin + metribuzin + rimsulfuron						
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1994	C	Butylate + imazethapyr						
1991	C	Untreated control	0	0	0	-	-	-
1992	T	Untreated control						
1993	S	Untreated control						
1994	C	Untreated control						
LSD (0.05) (crop rotation x herbicide program)[e]			10	10	7	11	9	5

[d] The corn-tomato-soybean crop rotation was not included at site two.
[e] The LSD value is for the interaction of crop rotations and herbicide programs.

Table 6. Crop and herbicide rotation effects on common ragweed densities from 1991 to 1994 at two sites in Painter, VA.[a,b]

Year	Crop[c]	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m^2							
1991	C	Imazethapyr	8	18	25	7	0	3	3	2
1992	C	Imazethapyr								
1993	C	Imazethapyr								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	1	3	1	1	0	0	1	0
1992	C	Butylate + atrazine								
1993	C	Butylate + atrazine								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	1	3	13	3	1	0	5	0
1992	C	Butylate + atrazine								
1993	C	Imazethapyr								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	5	3	8	12	1	1	6	4
1992	C	Butylate + imazethapyr								
1993	C	Butylate + imazethapyr								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	3	34	45	17	0	8	6	6
1992	C	Untreated control								
1993	C	Untreated control								
1994	C	Untreated control								
1991	S	Imazaquin + nicosulfuron	1	18	25	6	0	3	3	6
1992	S	Imazaquin + nicosulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	S	Imazaquin + nicosulfuron								

[a] Population counts were taken 35, 58, 37, and 63 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site one; and 43, 62, 47, and 64 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site two.

[b] Since the interaction of crop rotations and herbicide programs was not significant at the 0.05 level, means for 1992 and 1993 at site one and for 1992 at site two are of the herbicide program averaged over all crop rotations and are presented with the appropriate Fisher's LSD value (at the 0.05 level). The LSD values for other years are for the interaction of crop rotations and herbicide programs.

[c] Crops were: C, corn; S, soybeans; T, tomato.

Table 6 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m²							
1991	S	Fomesafen + fluazifop-P + fenoxaprop[d]	10	3	1	0	0	0	0	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + nicosulfuron	8	3	13	0	1	0	3	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + nicosulfuron								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop	1	3	8	0	0	1	0	0
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	S	Untreated control	2	34	45	68	1	8	11	11
1992	S	Untreated control								
1993	S	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	13	18	25	12	1	3	1	3
1992	S	Imazaquin + nicosulfuron								
1993	C	Imazethapyr								
1994	S	Imazaquin + nicosulfuron								
1991	C	Butylate + atrazine	6	3	1	0	1	0	0	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + atrazine								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	C	Imazethapyr	7	3	13	0	0	0	1	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Imazethapyr								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								

[d] Fomesafen was applied as a mixture (1991) or sequentially (1992 to 1994) with fluazifop-P + fenoxaprop.

Table 6 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m²							
1991	C	Butylate + imazethapyr	4	3	8	0	2	1	1	0
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + imazethapyr								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	C	Untreated control	7	34	45	43	1	8	28	34
1992	S	Untreated control								
1993	C	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	6	18	25	26	-[e]	-	-	-
1992	T	Rimsulfuron + rimsulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	7	3	1	5	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	7	3	13	5	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Imazaquin + nicosulfuron								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	9	3	8	5	-	-	-	-
1992	T	Trifluralin + metribuzin + rimsulfuron								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	2	34	45	35	-	-	-	-
1992	T	Untreated control								
1993	S	Untreated control								
1994	C	Untreated control								
LSD (0.05)			8	10	9	17	NS	5	10	12

[e] The corn-tomato-soybean crop rotation was not included at site two.

Table 7 continued.

Year	Crop	Herbicide(s)	Site one			Site two		
			1991	1992	1993	1991	1992	1993
			%					
1991	S	Fomesafen + fluazifop-P + fenoxaprop[d]	65	97	99	92	99	99
1992	S	Fomesafen + fluazifop-P + fenoxaprop						
1993	S	Fomesafen + fluazifop-P + fenoxaprop						
1994	S	Fomesafen + fluazifop-P + fenoxaprop						
1991	S	Imazaquin + nicosulfuron	98	98	99	99	98	99
1992	S	Fomesafen + fluazifop-P + fenoxaprop						
1993	S	Imazaquin + nicosulfuron						
1994	S	Fomesafen + fluazifop-P + fenoxaprop						
1991	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop	91	99	99	98	99	99
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1991	S	Untreated control	0	0	0	0	0	0
1992	S	Untreated control						
1993	S	Untreated control						
1994	S	Untreated control						
1991	C	Imazethapyr	95	99	99	99	99	99
1992	S	Imazaquin + nicosulfuron						
1993	C	Imazethapyr						
1994	S	Imazaquin + nicosulfuron						
1991	C	Butylate + atrazine	95	96	84	92	99	80
1992	S	Fomesafen + fluazifop-P + fenoxaprop						
1993	C	Butylate + atrazine						
1994	S	Fomesafen + fluazifop-P + fenoxaprop						
1991	C	Imazethapyr	97	99	99	99	99	99
1992	S	Fomesafen + fluazifop-P + fenoxaprop						
1993	C	Imazethapyr						
1994	S	Fomesafen + fluazifop-P + fenoxaprop						

[d] Fomesafen was applied as a mixture (1991) or sequentially (1992 to 1994) with fluazifop-P + fenoxaprop.

Table 7 continued.

Year	Crop	Herbicide(s)	Site one			Site two		
			1991	1992	1993	1991	1992	1993
			%					
1991	C	Butylate + imazethapyr	99	99	99	98	99	99
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1993	C	Butylate + imazethapyr						
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1991	C	Untreated control	0	0	0	0	0	0
1992	S	Untreated control						
1993	C	Untreated control						
1994	S	Untreated control						
1991	C	Imazethapyr	96	99	99	-[e]	-	-
1992	T	Rimsulfuron + rimsulfuron						
1993	S	Imazaquin + nicosulfuron						
1994	C	Imazethapyr						
1991	C	Butylate + atrazine	95	98	96	-	-	-
1992	T	Trifluralin + metribuzin						
1993	S	Fomesafen + fluazifop-P + fenoxaprop						
1994	C	Butylate + atrazine						
1991	C	Imazethapyr	99	98	99	-	-	-
1992	T	Trifluralin + metribuzin						
1993	S	Imazaquin + nicosulfuron						
1994	C	Butylate + atrazine						
1991	C	Butylate + imazethapyr	99	99	99	-	-	-
1992	T	Trifluralin + metribuzin + rimsulfuron						
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1994	C	Butylate + imazethapyr						
1991	C	Untreated control	0	0	0	-	-	-
1992	T	Untreated control						
1993	S	Untreated control						
1994	C	Untreated control						
LSD (0.05)			9	4	5	2	3	5

[e] The corn-tomato-soybean crop rotation was not included at site two.

Table 8. Main effects of herbicide mode of action programs on densities of pigweed spp. from 1991 to 1994 at two sites in Painter, VA.[a,b]

Herbicide mode of action program	Site one				Site two			
	1991	1992	1993	1994	1991	1992	1993	1994
	plants/m²							
Continuous ALS-inhibitor herbicides	8	0	0	0	1	0	0	0
Continuous non-ALS-inhibitor herbicides	7	2	1	1	2	2	3	2
Annual rotation between ALS and non-ALS inhibitor herbicides	6	1	0	0	1	0	0	0
Combination of ALS- and non-ALS-inhibitor herbicides	0	0	0	0	0	0	0	1
Untreated control	46	96	18	4	41	44	8	3
LSD (0.05) (herbicide program)	10	20	5	2	5	14	3	2

[a] Population counts were taken 35, 58, 37, and 63 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site one; and 43, 62, 47, and 64 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site two. Pigweed spp. included smooth and redroot pigweed.

[b] Means and LSD values are for the herbicide program averaged over four and three crop rotations at site one and two, respectively.

Table 9. Crop and herbicide rotation effects on jimsonweed control from 1991 to 1994 at two sites in Painter, VA.[a,b]

Year	Crop[c]	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992[d]	1993	1994
			%							
1991	C	Imazethapyr	95	94	93	95	93	-	96	97
1992	C	Imazethapyr								
1993	C	Imazethapyr								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	36	82	92	92	45	-	95	96
1992	C	Butylate + atrazine								
1993	C	Butylate + atrazine								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	96	86	94	91	99	-	95	96
1992	C	Butylate + atrazine								
1993	C	Imazethapyr								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	98	90	94	95	98	-	94	97
1992	C	Butylate + imazethapyr								
1993	C	Butylate + imazethapyr								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	0	0	0	0	0	-	0	0
1992	C	Untreated control								
1993	C	Untreated control								
1994	C	Untreated control								
1991	S	Imazaquin + nicosulfuron	91	97	99	95	87	-	96	97
1992	S	Imazaquin + nicosulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	S	Imazaquin + nicosulfuron								

[a] Visual control ratings were made 82, 100, 98, and 78 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively.

[b] Since the interaction of crop rotations and herbicide programs was not significant at the 0.05 level, means for 1991 and 1994 at site one and for 1993 and 1994 at site two are of the herbicide program averaged over all crop rotations and are presented with the appropriate Fisher's LSD value (at the 0.05 level). The LSD values for other years are for the interaction of crop rotations and herbicide programs.

[c] Crops were: C, corn; S, soybeans; T, tomato.

[d] Visual control ratings on jimsonweed were not made in 1992 at site two.

Table 9 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			%							
1991	S	Fomesafen + fluazifop-P + fenoxaprop[e]	49	68	86	92	88	-	95	96
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + nicosulfuron	82	60	98	91	76	-	95	96
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + nicosulfuron								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop	84	95	93	95	87	-	94	97
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	S	Untreated control	0	0	0	0	0	-	0	0
1992	S	Untreated control								
1993	S	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	96	99	94	95	99	-	96	97
1992	S	Imazaquin + nicosulfuron								
1993	C	Imazethapyr								
1994	S	Imazaquin + nicosulfuron								
1991	C	Butylate + atrazine	40	49	89	92	39	-	95	96
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + atrazine								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	C	Imazethapyr	98	45	92	91	98	-	95	96
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Imazethapyr								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								

[e] Fomesafen was applied as a mixture (1991) or sequentially (1992 to 1994) with fluazifop-P + fenoxaprop.

Table 9 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			%							
1991	C	Butylate + imazethapyr	98	99	96	95	94	-	94	97
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + imazethapyr								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	C	Untreated control	0	0	0	0	0	-	0	0
1992	S	Untreated control								
1993	C	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	96	13	99	95	-[f]	-	-	-
1992	T	Rimsulfuron + rimsulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	35	48	83	92	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	98	44	96	91	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Imazaquin + nicosulfuron								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	98	35	95	95	-	-	-	-
1992	T	Trifluralin + metribuzin + rimsulfuron								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	0	0	0	0	-	-	-	-
1992	T	Untreated control								
1993	S	Untreated control								
1994	C	Untreated control								
LSD (0.05)			9	13	5	2	10	-	3	2

[f] The corn-tomato-soybean crop rotation was not included at site two.

Table 10. Crop and herbicide rotation effects on jimsonweed densities from 1991 to 1994 at two sites in Painter, VA.[a,b]

Year	Crop[c]	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m^2							
1991	C	Imazethapyr	7	1	4	1	2	1	0	0
1992	C	Imazethapyr								
1993	C	Imazethapyr								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	11	9	2	1	3	10	0	0
1992	C	Butylate + atrazine								
1993	C	Butylate + atrazine								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	5	2	6	1	1	1	1	0
1992	C	Butylate + atrazine								
1993	C	Imazethapyr								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	4	5	2	3	4	1	1	0
1992	C	Butylate + imazethapyr								
1993	C	Butylate + imazethapyr								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	24	2	13	26	19	2	5	6
1992	C	Untreated control								
1993	C	Untreated control								
1994	C	Untreated control								
1991	S	Imazaquin + nicosulfuron	7	2	1	1	2	1	0	0
1992	S	Imazaquin + nicosulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	S	Imazaquin + nicosulfuron								

[a] Population counts were taken 35, 58, 37, and 63 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site one; and 43, 62, 47, and 64 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site two.

[b] Since the interaction of crop rotations and herbicide programs was not significant at the 0.05 level, means for 1991 and 1994 at site one and for 1991, 1993 and 1994 at site two are of the herbicide program averaged over all crop rotations and are presented with the appropriate Fisher's LSD value (at the 0.05 level). The LSD values for other years are for the interaction of crop rotations and herbicide programs.

[c] Crops were: C, corn; S, soybeans; T, tomato.

Table 10 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m²							
1991	S	Fomesafen + fluazifop-P + fenoxaprop[d]	11	11	2	1	3	1	0	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + nicosulfuron	5	5	3	1	1	4	1	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + nicosulfuron								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop	4	2	0	3	4	1	1	0
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	S	Untreated control	24	4	8	26	19	3	5	6
1992	S	Untreated control								
1993	S	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	7	4	1	1	2	0	0	0
1992	S	Imazaquin + nicosulfuron								
1993	C	Imazethapyr								
1994	S	Imazaquin + nicosulfuron								
1991	C	Butylate + atrazine	11	12	9	1	3	3	0	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + atrazine								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	C	Imazethapyr	5	6	11	1	1	2	1	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Imazethapyr								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								

[d] Fomesafen was applied as a mixture (1991) or sequentially (1992 to 1994) with fluazifop-P + fenoxaprop.

Table 10 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m²							
1991	C	Butylate + imazethapyr	4	2	1	3	4	1	1	0
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + imazethapyr								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	C	Untreated control	24	5	14	26	19	2	5	6
1992	S	Untreated control								
1993	C	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	7	19	10	1	-[c]	-	-	-
1992	T	Rimsulfuron + rimsulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	11	35	2	1	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	5	8	1	1	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Imazaquin + nicosulfuron								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	4	16	3	3	-	-	-	-
1992	T	Trifluralin + metribuzin + rimsulfuron								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	24	4	10	26	-	-	-	-
1992	T	Untreated control								
1993	S	Untreated control								
1994	C	Untreated control								
LSD (0.05)			12	9	6	7	6	4	2	2

[c] The corn-tomato-soybean crop rotation was not included at site two.

Table 11. Crop and herbicide rotation effects on corn, soybean, and tomato yields from 1991 to 1994 at two sites in Painter, VA.

Year	Crop[a]	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			kg/ha							
1991	C	Imazethapyr	2929	6976	3991	8256	1880	3720	3142	4334
1992	C	Imazethapyr								
1993	C	Imazethapyr								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	2786	6597	4191	6579	2905	5646	4329	4214
1992	C	Butylate + atrazine								
1993	C	Butylate + atrazine								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	2621	6270	4159	7507	2193	5435	3812	5424
1992	C	Butylate + atrazine								
1993	C	Imazethapyr								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	2987	6605	4224	7394	2496	4451	3519	4453
1992	C	Butylate + imazethapyr								
1993	C	Butylate + imazethapyr								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	416	435	565	333	305	159	683	148
1992	C	Untreated control								
1993	C	Untreated control								
1994	C	Untreated control								
1991	S	Imazaquin + nicosulfuron	1792	1609	723	1974	1798	2645	989	2475
1992	S	Imazaquin + nicosulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	S	Imazaquin + nicosulfuron								
1991	S	Fomesafen + fluazifop-P + fenoxaprop[b]	331	1475	1643	2364	713	2331	945	1611
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								

[a] Crops were: C, corn; S, soybeans; T, tomato.
[b] Fomesafen was applied as a mixture (1991) or sequentially (1992 to 1994) with fluazifop-P + fenoxaprop.

Table 11 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			kg/ha							
1991	S	Imazaquin + nicosulfuron	1766	2195	1278	2590	1766	2621	930	2682
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + nicosulfuron								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop	2263	3313	1525	3314	1525	3197	822	3308
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	S	Untreated control	0	629	195	747	168	931	0	834
1992	S	Untreated control								
1993	S	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	3963	1481	5261	2682	1852	2919	3020	2048
1992	S	Imazaquin + nicosulfuron								
1993	C	Imazethapyr								
1994	S	Imazaquin + nicosulfuron								
1991	C	Butylate + atrazine	3946	2013	6047	2944	3026	3018	4469	2842
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + atrazine								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	C	Imazethapyr	3935	2028	4900	2734	2291	3256	3885	2704
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Imazethapyr								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	C	Butylate + imazethapyr	4714	3636	5439	3871	2860	3895	4443	3622
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + imazethapyr								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								

Table 11 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			kg/ha							
1991	C	Untreated control	391	552	638	1199	240	826	328	989
1992	S	Untreated control								
1993	C	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	2508	35203	943	8963	-[c]	-	-	-
1992	T	Rimsulfuron + rimsulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	3219	31900	1786	8035	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	2088	21748	1147	7992	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Imazaquin + nicosulfuron								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	3701	26641	1686	8843	-	-	-	-
1992	T	Trifluralin + metribuzin + rimsulfuron								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	353	5088	607	212	-	-	-	-
1992	T	Untreated control								
1993	S	Untreated control								
1994	C	Untreated control								
LSD (0.05) (crop rotation x herbicide program) (corn)[d]			978	596	630	611	840	1019	692	1149
LSD (0.05) (crop rotation x herbicide program) (soybean)			221	532	425	360	104	611	198	889
LSD (0.05) (crop rotation x herbicide program) (tomato)			-	4258	-	-	-	-	-	-

[c] The corn-tomato-soybean crop rotation was not included at site two.
[d] The LSD values are for the interaction of crop rotations and herbicide programs. Yields for the individual crops were analyzed separately in each year.

Chapter III

Crop Rotation and Herbicide Program Effects on Annual Grass and Yellow Nutsedge (*Cyperus esculentus*) Populations

Abstract. Studies from 1991 through 1994 investigated the effects of several crop rotations and herbicide programs on populations of goosegrass, stinkgrass, large crabgrass, smooth crabgrass, fall panicum, and yellow nutsedge at two adjacent sites. In a split-plot arrangement of treatments, crop rotations were the main plots and included continuous corn, continuous soybean, corn-soybean, and corn-tomato-soybean. Herbicide programs were the split plots and included continuous use of acetolactate synthase (ALS)-inhibitor herbicides, continuous use of non-ALS-inhibitor herbicides, annual rotations between ALS- and non-ALS-inhibitor herbicides, combinations of ALS- and non-ALS-inhibitor herbicides in the same year, and no herbicide. Weed control and weed densities generally were affected by an interaction between crop rotations and herbicide programs. Goosegrass densities in 1994 were highest from the continuous use of ALS-inhibitor herbicides in the corn-tomato-soybean rotation, and were generally high in 1994 in the continuous corn and corn-tomato-soybean rotations. Stinkgrass control at both sites was lowest from imazethapyr, and densities were highest by 1994 where imazethapyr was applied alone for 4 years. Stinkgrass densities were also high where imazethapyr was applied in combination with butylate for 4 years and at site two where imazaquin plus nicosulfuron was applied for 4 years. Herbicide programs did not produce shifts in large

crabgrass densities, except that control was lowest and densities highest where butylate plus atrazine were applied for 4 years. Smooth crabgrass was present in significant densities at site one only where imazaquin plus nicosulfuron was used for 4 years or at site two from continuous ALS-inhibitor programs. Fall panicum densities were highest by 1994 where the combination of butylate plus atrazine was applied continuously for 4 years. Yellow nutsedge control was lowest and densities were highest at site two where the combination of fomesafen plus fluazifop-P plus fenoxaprop was applied continuously for 4 years. Yellow nutsedge densities by 1994 at site two were also high where these herbicides were applied with imazaquin for 4 years or where these herbicides were applied in rotations with imazaquin plus nicosulfuron or butylate plus atrazine. **Nomenclature:** Atrazine, 6-chloro-*N*-ethyl-*N'*-(1-methylethyl)-1,3,5-triazine-2,4-diamine; butylate, *S*-ethyl bis(2-methylpropyl)carbamothioate; fenoxaprop, (±)-2-[4-[(6-chloro-2-benzoxazolyl)oxy]phenoxy]propanoic acid; fluazifop-P, (R)-2-[4-[[5-(trifluoromethyl)-2-pyridinyl]oxy]phenoxy]propanoic acid; fomesafen, 5-[2-chloro-4-(trifluoromethyl)phenoxy]-*N*-(methylsulfonyl)-2-nitrobenzamide; imazaquin, 2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-3-quinolinecarboxylic acid; imazethapyr, 2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-5-ethyl-3-pyridinecarboxylic acid; nicosulfuron, 2-[[[[(4,6-dimethoxy-2-pyrimidinyl)amino]carbonyl]amino]sulfonyl]-*N,N*-dimethyl-3-pyridinecarboxamide; fall

panicum, *Panicum dichotomiflorum* Michx. #[1] PANDI; goosegrass, *Eleucine indica* (L.) Gaertn. # ELEIN; large crabgrass, *Digitaria sanguinalis* (L.) Scop. # DIGSA; smooth crabgrass, *Digitaria ischaemum* (Schreb. ex Schweig.) Schreb. ex Muhl # DIGIS; stinkgrass, *Eragrostis cilianensis* (All.) E. Mosher # ERACN; yellow nutsedge, *Cyperus esculentus* L. # CYPES; corn, *Zea mays* L. 'ICI 8532IT' in 1991, 1992 and 1993, and 'Pioneer 3245IR' in 1994; soybeans, *Glycine max* (L.) Merr. 'W20-STS' in 1991, 'STS-9122' in 1992, 'Asgrow 3200 STS' in 1993, and 'Asgrow 4045 STS' in 1994; tomato, *Lycopersicon esculentum* L. 'Floradade'.

Additional index words: ALS-inhibitor herbicides, crop rotations, herbicide programs, herbicide rotations, imidazolinone herbicides, sulfonylurea herbicides, weed shifts, atrazine, butylate, fenoxaprop, fluazifop-P, fomesafen, imazaquin, imazethapyr, metribuzin, nicosulfuron, rimsulfuron, trifluralin, *Digitaria ischaemum, Digitaria sanguinalis, Eleucine indica, Eragrostis cilianensis, Panicum dichotomiflorum, Glycine max, Lycopersicon esculentum, Zea mays,* CYPES, DIGIS, DIGSA, ELEIN, ERACN, PANDI.

INTRODUCTION

This research was introduced and the literature reviewed in the previous chapter. The

[1] Letters following this symbol are a WSSA-approved computer code from Composite List of Weeds, Revised 1989. Available from WSSA, 1508 West University Ave., Champaign, IL 61821-3133.

first objective of this research was to determine the effects of crop rotations and herbicide programs on the population dynamics of fall panicum, goosegrass, large crabgrass, smooth crabgrass, stinkgrass, and yellow nutsedge. A secondary objective was to monitor plots for the potential development of herbicide resistant weed populations.

MATERIALS AND METHODS

The materials and methods for this research are described in the previous chapter. The herbicides used in the three crops to form the herbicide programs are listed in Table 1 with their modes of action, application timings, and application rates. Three of the herbicide programs (the continuous use of ALS-inhibitor herbicides, continuous use of non-ALS-inhibitor herbicides, and combination of ALS- and non-ALS-inhibitor herbicides in the same year) are explained in the table. Other herbicide programs included an annual rotation between ALS- and non-ALS-inhibitor herbicides and no herbicide.

RESULTS AND DISCUSSION

Weed densities were determined prior to the application of fluazifop-P plus fenoxaprop in 1993, which explains unusually high densities of grass species in plots treated with these herbicides in 1993. Densities of all grass species in these plots decreased following application of fluazifop-P plus fenoxaprop in 1993. Increases in weed densities occurred

with all species where control from herbicides was low, and shifts were often less severe where herbicides were rotated annually. Weed control was generally affected by an interaction of crop rotations and herbicide programs or by herbicide programs alone. Where the interaction of crop rotations and herbicide programs was significant, the means represent the individual treatments and are presented with the appropriate LSD value. Where only a main effect (crop rotations or herbicide programs) was significant, the means represent the significant factor averaged over the other factor.

Goosegrass. The interaction of crop rotations and herbicide programs was significant in all years at both sites for control estimates (Table A.4). Goosegrass control from fomesafen plus fluazifop-P plus fenoxaprop was generally above 90%, but was lower and inconsistent from other treatments (Table 2). For goosegrass densities, the interaction of crop rotations and herbicide programs was significant in 1992 at site one, and in 1993 and 1994 at both sites (Table A.5). Otherwise, the main effect of herbicide programs was significant for goosegrass densities. Goosegrass densities in most treatments also fluctuated annually at both sites, and by 1994, densities in the untreated controls were less than 7 plants/m^2 (Table 3) due to competition from broadleaf weeds (see previous chapter). In 1994, goosegrass control was generally lower (Table 2) and densities generally higher (Table 3) in the continuous corn (CC)[2] and corn-tomato-soybean (CTS)[2] rotations where corn was planted compared with the continuous soybean (SS)[2] and corn-

[2] Abbreviations: CC, continuous corn; CS, corn-soybean; CTS, corn-tomato-soybean; SS, continuous soybean.

soybean (CS)[2] rotations where soybean was planted (Table 3). Goosegrass control at site one in 1994 was lowest (41%) and densities were highest (91 plants/m^2) from the continuous ALS-inhibitor program in the CTS rotation likely due to control below 65% in each of the previous seasons from this treatment. Goosegrass control from butylate plus atrazine averaged 61% at both sites in 1994, but at site one averaged 63% where the combination was rotated with other herbicides compared with 49% where it was applied continuously for 4 years (Table 2). However, densities from butylate plus atrazine were not always lowered by rotating the combination with other herbicides (Table 3), and the differences in control at site one did not occur at site two. Goosegrass control at site one in the CTS rotation was higher from the continuous non-ALS-inhibitor program than from the annual rotation program in all 4 years including 1994 when butylate plus atrazine was applied in both rotations (Table 2). As a result, goosegrass densities in the CTS rotation were 16 and 33 plants/m^2 in 1993 and 1994, respectively from the continuous non-ALS-inhibitor program. Densities in the CTS rotation were 31 and 51 plants/m^2 in 1993 and 1994, respectively from the annual rotation program (Table 3).

Stinkgrass. Stinkgrass control was not estimated in 1991 because stinkgrass densities were low, and stinkgrass was generally present only in plots treated with imazethapyr. The interaction of crop rotations and herbicide programs was significant in all years at both sites for control estimates (Table A.4). Stinkgrass control was lowest from imazethapyr (0 to 5% where it was used continuously), but was improved by rotating imazethapyr with other herbicides (Table 4). Butylate plus imazethapyr gave 85% to 86%

control in 1992, but control averaged 34% by 1994 where the combination was applied for 4 years. Stinkgrass control in 1994 from butylate plus imazethapyr was 68% in the CTS rotation where the combination was rotated with other herbicides. Control from continuous annual applications of butylate plus atrazine also diminished from an average 92% in 1991 to an average 66% in 1994. However, stinkgrass control at site one in 1994 was 76 to 83% in the CTS rotation where butylate plus atrazine was rotated with other herbicides.

Stinkgrass densities generally increased at both sites from 1991 to 1994 (Table 5), and control from imazaquin plus nicosulfuron declined as densities increased (Table 4). Stinkgrass control in 1992 at site one from imazaquin plus nicosulfuron was above 90% where densities were low [< 5 plants/m^2 (Table 5)]. Control at site two from imazaquin plus nicosulfuron was approximately 60% in the same year where densities were higher (10 to 20 plants/m^2). In 1993 at site one, imazaquin plus nicosulfuron gave approximately 83% stinkgrass control where the combination was rotated with other herbicides, but stinkgrass control was only 55% where the combination was applied for 3 consecutive years. At site two in 1993, stinkgrass control was less than 35% from imazaquin plus nicosulfuron whether these herbicides were rotated with other herbicides or applied continuously. In 1994, control from imazaquin plus nicosulfuron was between 10 and 36% at both sites whether the combination was rotated with imazethapyr or applied for 4 years.

Stinkgrass densities generally increased in the untreated controls at both sites from 1991

to 1994 (Table 5). Stinkgrass densities were high in 1992 at site two and in 1993 and 1994 at both sites where treatments did not give control. The main effect of herbicide programs was significant in 1992 at site one and in 1993 at site two for stinkgrass densities (Table A.5). The interaction of crop rotations and herbicide programs was significant at site one in 1991, 1993, and 1994 and at site two in 1991, 1992, and 1994 (Table A.5). High stinkgrass densities were often associated with imazethapyr or imazaquin plus nicosulfuron applications. In 1991 at site one, stinkgrass densities were low, and there were no significant differences between treatments. Densities at site two in 1991 were also low, and stinkgrass was generally only present where imazethapyr was applied. In 1992 at site one, stinkgrass densities were 4 plants/m^2 from the continuous ALS-inhibitor program. In 1992 at site two, densities were 55 plants/m^2 from two consecutive annual applications of imazethapyr, and were 19 plants/m^2 following two annual applications of imazaquin plus nicosulfuron.

In 1993, stinkgrass densities were affected by the late application timing of fluazifop-P plus fenoxaprop. There were no differences among treatments in 1993 at site one due to variability in stinkgrass densities and the late application of fluazifop-P plus fenoxaprop. Densities at site two in 1993 were highest from the continuous ALS-inhibitor program (84 plants/m^2), but were also 26 and 42 plants/m^2 from the annual rotation and combination programs, respectively. High stinkgrass densities in the combination program were due in part to the late application of fluazifop-P plus fenoxaprop.

High stinkgrass densities in 1994 were again associated with imazethapyr or imazaquin

plus nicosulfuron applications. Stinkgrass densities were 122 and 228 plants/m^2 in 1994 at sites one and two, respectively, where imazethapyr was applied alone for 4 years. There were 64 and 122 plants/m^2 at sites one and two, respectively, where butylate plus imazethapyr were applied for 4 years. Densities at site two in 1994 were 123 plants/m^2 where imazaquin plus nicosulfuron were applied for 4 years in SS, and there were 59 plants/m^2 where this combination was rotated with imazethapyr in the CS rotation. However, stinkgrass densities at site one in 1994 remained below 20 plants/m^2 from imazaquin plus nicosulfuron treatments in spite of 23 to 36% control from these treatments (Table 4).

Large crabgrass. Large crabgrass densities at site one were low in 1991, 1992, and 1993 (Table 7) and control estimates were not made in those years (Table 6). The main effect of herbicide programs was significant in 1991 at site two for control; otherwise the interaction of crop rotations and herbicide programs was significant (Table A.4). Control at site two in 1991 was 90% from the combination program, but was low from ALS-inhibitor herbicides (Table 6). Control from imazaquin plus nicosulfuron fluctuated from 39 to 83% at site two from 1991 to 1994, but was generally above 85% from treatments that included fluazifop-P plus fenoxaprop. Large crabgrass control from butylate plus atrazine at site two was 82% in 1991, but by 1994 averaged only 27% at both sites where the combination was applied for 4 years. However, control was higher than 70% from butylate plus atrazine in 1994 where these herbicides were rotated with other herbicides, regardless of the crop rotation. Conversely, control from butylate plus imazethapyr in

1994 was above 90% where this combination was applied for 4 years likely due to control from imazethapyr. Large crabgrass densities were 19 to 20 plants/m^2 in 1993 at site two in SS where fluazifop-P plus fenoxaprop were to be applied (Table 7), but these densities declined following application of these herbicides. In 1993 at site two and in 1994 at both sites, densities were 15 to 36 plants/m^2 where butylate plus atrazine were applied continuously. Densities in other treated plots were below 10 plants/m^2 in all years at both sites.

Smooth crabgrass. Smooth crabgrass was not observed in 1991, and densities were low in other years, so percent control estimates were not made (Table 8). The main effect of herbicide programs was significant in 1992 and 1994 at site two; otherwise, the interaction of crop rotations and herbicide programs was significant (Table A.6). Generally, smooth crabgrass was present only where imazaquin plus nicosulfuron or imazethapyr alone were applied. In 1992 at site one, smooth crabgrass occurred only in plots treated with imazaquin plus nicosulfuron and at site two in plots treated with only ALS-inhibitor herbicides. By 1994 at site one, smooth crabgrass densities were 90 plants/m^2 from four annual applications of imazaquin plus nicosulfuron, but were less than 10 plants/m^2 from all other treatments. Densities in 1994 at site one were 7 plants/m^2 from imazaquin plus nicosulfuron rotated with imazethapyr in the CS rotation and 5 plants/m^2 from imazethapyr rotated with other ALS-inhibitor herbicides in the CTS rotation. By 1994 at site two, densities were 19 plants/m^2 from the continuous ALS-inhibitor programs, while densities from other herbicide programs were 1 plant/m^2 or less.

Fall Panicum. As with smooth crabgrass, fall panicum densities were low at both sites, and control estimates were not made. For fall panicum densities, the main effect of herbicide programs was significant in 1991 at both sites (Table A.6). Otherwise the interaction of crop rotations and herbicide programs was significant (Table A.6). From 1991 to 1993, fall panicum densities were below 10 plants/m^2 in all treatments except in 1993 at site two where butylate plus atrazine were applied for 3 years (Table 9). By 1994, fall panicum densities were 28 and 47 plants/m^2 at sites one and two, respectively, where butylate plus atrazine were applied for 4 years. Densities in 1994 in all other treatments were 5 plants/m^2 or less, except densities were 14 plants/m^2 in CC at site two where butylate plus atrazine was rotated with imazethapyr.

Yellow nutsedge. Yellow nutsedge control estimates were made only at site two in 1992, 1993, and 1994 (Table 10) since densities were low and variable otherwise (Table 11). The interaction of crop rotations and herbicide programs was significant for yellow nutsedge control at site two in all years (Table A.4). For yellow nutsedge densities, the interaction of crop rotations and herbicide programs was significant in all years at both sites except 1993 at site one, when the main effect of crop rotations was significant (Table A.6). Yellow nutsedge control was lowest from fomesafen plus fluazifop-P plus fenoxaprop at site two, but in 1994 was improved by rotating with other herbicides regardless of the crop rotation (Table 10). Control from other herbicide combinations remained relatively constant from 1992 to 1994 except that control was an average 22% from the combination of imazaquin, fomesafen, fluazifop-P, and fenoxaprop in the SS and

the CS rotations in 1992 compared with 65% to 75% in 1993 and 1994, respectively. Treatments that did not include fomesafen plus fluazifop-P plus fenoxaprop controlled 70 to 90% of yellow nutsedge at site two.

Yellow nutsedge densities did not differ among treatments at site one in 1991. Densities in most treatments at site one fluctuated annually but remained below 10 plants/m^2. However, yellow nutsedge densities at site one in 1993 in the CTS rotation were 13 plants/m^2 and were higher than densities in other crop rotations. Densities in all herbicide programs in CC at site two remained below 15 plants/m^2 throughout the study since all corn herbicides controlled 70 to 90% of yellow nutsedge and common lambsquarters competed with yellow nutsedge in the untreated control.

Yellow nutsedge control was lowest from fomesafen plus fluazifop-P plus fenoxaprop, and densities in 1994 at site one were highest from this combination applied alone (9 plants/m^2) or with imazaquin (9 plants/m^2) for 4 years in SS. Densities at site one in 1994 were also high from butylate plus atrazine in the continuous non-ALS-inhibitor program in the CTS rotation (11 plants/m^2). Yellow nutsedge densities in 1991 at site two were 27 plants/m^2 from fomesafen plus fluazifop-P plus fenoxaprop but increased to 286 plants/m^2 by 1994 where this combination was applied for 4 years. In addition, yellow nutsedge densities in 1994 at site two were 60 to 99 plants/m^2 where fomesafen plus fluazifop-P plus fenoxaprop were applied in combination with imazaquin for 4 years or in rotation with imazaquin plus nicosulfuron or butylate plus atrazine.

In summary, weed species shifts to tolerant species were observed where the same

herbicides or herbicides with similar selectivities were used continuously or in rotation as other researchers have observed (1, 2, 5). An exception is the decreased control and increased densities of grass species following continuous applications of butylate. Densities of all grass species except smooth crabgrass increased from 1991 to 1994 following four annual applications of butylate plus atrazine, and densities of goosegrass and stinkgrass increased from 1991 to 1994 following four annual applications of butylate plus imazethapyr. Enhanced microbial degradation of butylate and other carbamothioate herbicides has been demonstrated in soils with a history of carbamothioate herbicide exposure (4, 6). This enhanced degradation can negatively affect weed control from these herbicides. Soil from plots at site one treated with butylate plus atrazine for 3 consecutive years and from untreated plots was collected following the 1993 season. Accelerated degradation of EPTC (*S*-ethyl dipropyl carbamothioate) was observed in the butylate-history soil (3). Accelerated degradation of butylate may be partially responsible for the diminished control of previously susceptible grass species in these treatments. Other weed species shifts that occurred were due to tolerant species rather than herbicide-resistant biotypes proliferating in the weed population since control of these species was low throughout the course of these studies. The results of this research indicate the importance of rotating to or including herbicides with high activity on the target weed species to delay or preclude weed population shifts. Weed population shifts could include shifts to herbicide-resistant biotypes of the same species, so these principles could also be applied to herbicide-resistance management.

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Table 1. ALS- and Non-ALS-inhibitor herbicide components used in the specified crop to form the herbicide mode of action programs.

Crop	Mode of action	Herbicide(s)[a]	Timing[b]	Rate[c]
				g/ha
Corn	ALS-inhibitor	Imazethapyr	EPOST	70
	Non-ALS-inhibitor	Butylate + atrazine	PPI + PPI	3360 + 1120
	Combination[d]	Butylate + imazethapyr	PPI + EPOST	3360 + 70
Soybeans	ALS-inhibitor	Imazaquin + nicosulfuron	PPI + EPOST	140 + 35
	Non-ALS-inhibitor	Fomesafen + fluazifop + fenoxaprop[e]	EPOST + LPOST + LPOST	426 + 140 + 46
	Combination	Imazaquin + fomesafen + fluazifop + fenoxaprop	PPI + EPOST + LPOST + LPOST	140 + 426 + 140 + 46
Tomato	ALS-inhibitor	Rimsulfuron + rimsulfuron	10 DATP + 21 DATP	35 + 35
	Non-ALS-inhibitor	Trifluralin + metribuzin	PPI + 10 DATP	560 + 280
	Combination	Trifluralin + metribuzin + rimsulfuron	PPI + 10 DATP + 10 DATP	560 + 280 + 35

[a] Methylated seed oil was applied at 1% v/v with imazethapyr and nicosulfuron, crop oil concentrate was applied at 1% v/v with fomesafen and fluazifop + fenoxaprop, and a non-ionic surfactant was applied at 0.25% v/v with rimsulfuron and metribuzin.

[b] PPI = preplant incorporated; EPOST = early postemergence based on weed size of less than 10 cm (usually less than 5 cm); LPOST = late postemergence based on weed size of 10 to 20 cm; DATP = days after transplanting.

[c] Rates applied were commercial use rates or an anticipated commercial use rate for rimsulfuron.

[d] Combination refers to application of the components as a mixture, or sequential applications in the same year. The annual rotation mode of action program and the untreated control are not included.

[e] Fomesafen was always applied as a mixture (1991) or sequentially (1992 to 1994) with fluazifop + fenoxaprop.

Table 2. Crop and herbicide rotation effects on goosegrass control from 1991 to 1994 at two sites in Painter, VA.[a]

Year	Crop[b]	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			%							
1991	C	Imazethapyr	12	64	56	56	15	49	60	65
1992	C	Imazethapyr								
1993	C	Imazethapyr								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	92	79	68	49	88	75	66	63
1992	C	Butylate + atrazine								
1993	C	Butylate + atrazine								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	16	83	65	64	21	80	64	68
1992	C	Butylate + atrazine								
1993	C	Imazethapyr								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	97	78	68	63	91	81	65	69
1992	C	Butylate + imazethapyr								
1993	C	Butylate + imazethapyr								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	0	0	0	0	0	0	0	0
1992	C	Untreated control								
1993	C	Untreated control								
1994	C	Untreated control								
1991	S	Imazaquin + nicosulfuron	59	69	68	93	54	88	76	90
1992	S	Imazaquin + nicosulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	S	Imazaquin + nicosulfuron								

[a] Visual control ratings were made 82, 100, 98, and 78 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively.
[b] Crops were: C, corn; S, soybeans; T, tomato.

Table 2 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			%							
1991	S	Fomesafen + fluazifop-P + fenoxaprop[c]	94	99	99	96	61	99	99	95
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + nicosulfuron	68	99	74	95	51	99	70	95
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + nicosulfuron								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop	91	99	97	94	78	98	97	95
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	S	Untreated control	0	0	0	0	0	0	0	0
1992	S	Untreated control								
1993	S	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	14	80	63	88	23	88	65	89
1992	S	Imazaquin + nicosulfuron								
1993	C	Imazethapyr								
1994	S	Imazaquin + nicosulfuron								
1991	C	Butylate + atrazine	94	99	83	95	91	99	68	95
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + atrazine								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	C	Imazethapyr	13	99	64	95	21	99	61	95
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Imazethapyr								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								

[c] Fluazifop-P + fenoxaprop was applied as a mixture (1991) or sequentially (1992 to 1994) with fomesafen.

Table 2 continued.

Year	Crop	Herbicide(s)	Site one 1991	Site one 1992	Site one 1993	Site one 1994	Site two 1991	Site two 1992	Site two 1993	Site two 1994
			%							
1991	C	Butylate + imazethapyr	96	99	76	93	88	99	69	93
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + imazethapyr								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	C	Untreated control	0	0	0	0	0	0	0	0
1992	S	Untreated control								
1993	C	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	13	46	64	41	-[d]	-	-	-
1992	T	Rimsulfuron + rimsulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	94	66	99	65	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	14	50	66	59	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Imazaquin + nicosulfuron								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	97	49	98	63	-	-	-	-
1992	T	Trifluralin + metribuzin + rimsulfuron								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	0	0	0	0	-	-	-	-
1992	T	Untreated control								
1993	S	Untreated control								
1994	C	Untreated control								
LSD (0.05) (crop rotation x herbicide program)[e]			18	13	10	6	7	7	6	3

[d] The corn-tomato-soybean crop rotation was not included at site two.
[e] The LSD values are for the interaction of crop rotations and herbicide programs.

Table 3. Crop and herbicide rotation effects on goosegrass densities from 1991 to 1994 at two sites in Painter, VA.[a,b]

Year	Crop[c]	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m²							
1991	C	Imazethapyr	7	10	0	17	28	4	5	21
1992	C	Imazethapyr								
1993	C	Imazethapyr								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	2	6	10	42	10	2	10	18
1992	C	Butylate + atrazine								
1993	C	Butylate + atrazine								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	7	4	3	34	26	2	6	32
1992	C	Butylate + atrazine								
1993	C	Imazethapyr								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	2	6	1	40	9	1	5	22
1992	C	Butylate + imazethapyr								
1993	C	Butylate + imazethapyr								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	9	3	10	5	37	31	27	1
1992	C	Untreated control								
1993	C	Untreated control								
1994	C	Untreated control								
1991	S	Imazaquin + nicosulfuron	7	3	30	1	28	4	0	2
1992	S	Imazaquin + nicosulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	S	Imazaquin + nicosulfuron								

[a] Population counts were taken 35, 58, 37, and 63 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site one; and 43, 62, 47, and 64 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site two.

[b] Since the interaction of crop rotations and herbicide programs was not significant at the 0.05 level, means for 1991 at sites one and two and for 1992 at site two are the means of the herbicide program averaged over all crop rotations and are presented with the appropriate Fisher's LSD value (at the 0.05 level). The LSD values for other years are for the interaction of crop rotations and herbicide programs.

[c] Crops were: C, corn; S, soybeans; T, tomato.

Table 3 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m²							
1991	S	Fomesafen + fluazifop-P + fenoxaprop[d]	2	0	19	0	10	2	23	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + nicosulfuron	7	0	10	0	26	2	0	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + nicosulfuron								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop	2	0	5	0	9	1	38	1
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	S	Untreated control	9	11	11	2	37	31	25	5
1992	S	Untreated control								
1993	S	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	7	3	3	5	28	4	5	1
1992	S	Imazaquin + nicosulfuron								
1993	C	Imazethapyr								
1994	S	Imazaquin + nicosulfuron								
1991	C	Butylate + atrazine	2	0	1	1	10	2	8	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + atrazine								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	C	Imazethapyr	7	0	3	0	26	2	4	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Imazethapyr								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								

[d] Fluazifop-P + fenoxaprop was applied as a mixture (1991) or sequentially (1992 to 1994) with fomesafen.

Table 3 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m^2							
1991	C	Butylate + imazethapyr	2	0	1	1	9	1	6	3
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + imazethapyr								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	C	Untreated control	9	14	14	1	37	31	34	2
1992	S	Untreated control								
1993	C	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	7	17	25	91	-[e]	-	-	-
1992	T	Rimsulfuron + rimsulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	2	5	16	33	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	7	7	31	51	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Imazaquin + nicosulfuron								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	2	8	31	42	-	-	-	-
1992	T	Trifluralin + metribuzin + rimsulfuron								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	9	29	18	6	-	-	-	-
1992	T	Untreated control								
1993	S	Untreated control								
1994	C	Untreated control								
LSD (0.05)			5	10	15	18	13	11	16	11

[e] The corn-tomato-soybean crop rotation was not included at site two.

Table 4 continued.

Year	Crop	Herbicide(s)	Site one			Site two		
			1992	1993	1994	1992	1993	1994
			%					
1991	S	Fomesafen + fluazifop-P + fenoxaprop[c]	99	98	95	99	94	95
1992	S	Fomesafen + fluazifop-P + fenoxaprop						
1993	S	Fomesafen + fluazifop-P + fenoxaprop						
1994	S	Fomesafen + fluazifop-P + fenoxaprop						
1991	S	Imazaquin + nicosulfuron	99	89	95	99	34	94
1992	S	Fomesafen + fluazifop-P + fenoxaprop						
1993	S	Imazaquin + nicosulfuron						
1994	S	Fomesafen + fluazifop-P + fenoxaprop						
1991	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop	99	97	95	97	92	95
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1991	S	Untreated control	0	0	0	0	0	0
1992	S	Untreated control						
1993	S	Untreated control						
1994	S	Untreated control						
1991	C	Imazethapyr	92	41	36	58	18	10
1992	S	Imazaquin + nicosulfuron						
1993	C	Imazethapyr						
1994	S	Imazaquin + nicosulfuron						
1991	C	Butylate + atrazine	99	89	95	99	78	95
1992	S	Fomesafen + fluazifop-P + fenoxaprop						
1993	C	Butylate + atrazine						
1994	S	Fomesafen + fluazifop-P + fenoxaprop						
1991	C	Imazethapyr	99	58	95	99	44	95
1992	S	Fomesafen + fluazifop-P + fenoxaprop						
1993	C	Imazethapyr						
1994	S	Fomesafen + fluazifop-P + fenoxaprop						

[c] Fluazifop-P + fenoxaprop was applied as a mixture (1991) or sequentially (1992 to 1994) with fomesafen.

Table 4 continued.

Year	Crop	Herbicide(s)	Site one			Site two		
			1992	1993	1994	1992	1993	1994
			%					
1991	C	Butylate + imazethapyr	99	76	95	99	65	94
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1993	C	Butylate + imazethapyr						
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1991	C	Untreated control	0	0	0	0	0	0
1992	S	Untreated control						
1993	C	Untreated control						
1994	S	Untreated control						
1991	C	Imazethapyr	96	80	54	-[d]	-	-
1992	T	Rimsulfuron + rimsulfuron						
1993	S	Imazaquin + nicosulfuron						
1994	C	Imazethapyr						
1991	C	Butylate + atrazine	91	99	83	-	-	-
1992	T	Trifluralin + metribuzin						
1993	S	Fomesafen + fluazifop-P + fenoxaprop						
1994	C	Butylate + atrazine						
1991	C	Imazethapyr	97	79	76	-	-	-
1992	T	Trifluralin + metribuzin						
1993	S	Imazaquin + nicosulfuron						
1994	C	Butylate + atrazine						
1991	C	Butylate + imazethapyr	95	93	68	-	-	-
1992	T	Trifluralin + metribuzin + rimsulfuron						
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1994	C	Butylate + imazethapyr						
1991	C	Untreated control	0	0	0	-	-	-
1992	T	Untreated control						
1993	S	Untreated control						
1994	C	Untreated control						
LSD (0.05) (crop rotation x herbicide program)[e]			6	15	12	8	12	7

[d] The corn-tomato-soybean crop rotation was not included at site two.
[e] The LSD values are for the interaction of crop rotations and herbicide programs.

Table 5. Crop and herbicide rotation effects on stinkgrass densities from 1991 to 1994 at two sites in Painter, VA.[a,b]

Year	Crop[c]	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m²							
1991	C	Imazethapyr	1	4	12	122	3	55	84	228
1992	C	Imazethapyr								
1993	C	Imazethapyr								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	0	1	7	19	0	2	11	11
1992	C	Butylate + atrazine								
1993	C	Butylate + atrazine								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	1	2	4	18	5	2	26	35
1992	C	Butylate + atrazine								
1993	C	Imazethapyr								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	0	1	7	64	3	4	42	122
1992	C	Butylate + imazethapyr								
1993	C	Butylate + imazethapyr								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	0	7	5	10	1	20	43	60
1992	C	Untreated control								
1993	C	Untreated control								
1994	C	Untreated control								
1991	S	Imazaquin + nicosulfuron	0	4	11	16	0	19	84	123
1992	S	Imazaquin + nicosulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	S	Imazaquin + nicosulfuron								

[a] Population counts were taken 35, 58, 37, and 63 days after PPI treatments were applied in 1991, 1992. 1993, and 1994, respectively at site one; and 43, 62, 47, and 64 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site two.

[b] Since the interaction of crop rotations and herbicide programs was not significant at the 0.05 level, means for 1992 at site one and for 1993 at site two are the means of the herbicide program averaged over all crop rotations and are presented with the appropriate Fisher's LSD value (at the 0.05 level). The LSD values for other years are for the interaction of crop rotations and herbicide programs.

[c] Crops were: C, corn; S, soybeans; T, tomato.

Table 5 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m^2							
1991	S	Fomesafen + fluazifop-P + fenoxaprop[d]	2	1	4	0	1	0	11	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + nicosulfuron	0	2	4	0	0	0	26	1
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + nicosulfuron								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop	0	1	3	0	0	0	42	0
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	S	Untreated control	0	7	15	26	1	27	43	72
1992	S	Untreated control								
1993	S	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	1	4	4	14	6	10	84	59
1992	S	Imazaquin + nicosulfuron								
1993	C	Imazethapyr								
1994	S	Imazaquin + nicosulfuron								
1991	C	Butylate + atrazine	0	1	1	0	0	0	11	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + atrazine								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	C	Imazethapyr	1	2	4	0	5	0	26	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Imazethapyr								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								

[d] Fluazifop-P + fenoxaprop was applied as a mixture (1991) or sequentially (1992 to 1994) with fomesafen.

Table 5 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m²							
1991	C	Butylate + imazethapyr	0	1	9	1	2	0	42	4
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + imazethapyr								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	C	Untreated control	0	7	8	6	0	42	43	91
1992	S	Untreated control								
1993	C	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	2	4	5	16	-[e]	-	-	-
1992	T	Rimsulfuron + rimsulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	0	1	13	6	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	2	2	11	12	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Imazaquin + nicosulfuron								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	1	1	14	7	-	-	-	-
1992	T	Trifluralin + metribuzin + rimsulfuron								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	0	7	17	24	-	-	-	-
1992	T	Untreated control								
1993	S	Untreated control								
1994	C	Untreated control								
LSD (0.05)			NS	4	NS	20	4	16	20	48

[e] The corn-tomato-soybean crop rotation was not included at site two.

Table 6. Crop and herbicide rotation effects on large crabgrass control in 1994 at site one and from 1991 to 1994 at site two.[a]

Year	Crop[b]	Herbicide(s)	Site one	Site two			
			1994	1991[c]	1992	1993	1994
			%				
1991	C	Imazethapyr	94	55	51	78	88
1992	C	Imazethapyr					
1993	C	Imazethapyr					
1994	C	Imazethapyr					
1991	C	Butylate + atrazine	30	82	66	55	24
1992	C	Butylate + atrazine					
1993	C	Butylate + atrazine					
1994	C	Butylate + atrazine					
1991	C	Imazethapyr	74	57	73	80	73
1992	C	Butylate + atrazine					
1993	C	Imazethapyr					
1994	C	Butylate + atrazine					
1991	C	Butylate + imazethapyr	94	90	84	88	93
1992	C	Butylate + imazethapyr					
1993	C	Butylate + imazethapyr					
1994	C	Butylate + imazethapyr					
1991	C	Untreated control	0	0	0	0	0
1992	C	Untreated control					
1993	C	Untreated control					
1994	C	Untreated control					
1991	S	Imazaquin + nicosulfuron	88	55	65	39	78
1992	S	Imazaquin + nicosulfuron					
1993	S	Imazaquin + nicosulfuron					
1994	S	Imazaquin + nicosulfuron					
1991	S	Fomesafen + fluazifop-P + fenoxaprop[d]	96	82	99	97	95
1992	S	Fomesafen + fluazifop-P + fenoxaprop					
1993	S	Fomesafen + fluazifop-P + fenoxaprop					
1994	S	Fomesafen + fluazifop-P + fenoxaprop					
1991	S	Imazaquin + nicosulfuron	94	57	97	49	94
1992	S	Fomesafen + fluazifop-P + fenoxaprop					
1993	S	Imazaquin + nicosulfuron					
1994	S	Fomesafen + fluazifop-P + fenoxaprop					
1991	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop	94	90	87	93	95
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop					
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop					
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop					
1991	S	Untreated control	0	0	0	0	0
1992	S	Untreated control					
1993	S	Untreated control					
1994	S	Untreated control					

[a] Visual control ratings were made 82, 100, 98, and 78 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively.

[b] Crops were: C, corn; S, soybeans; T, tomato.

[c] Means for 1991 at site two are the means of the herbicide program averaged over all crop rotations and are presented with the appropriate Fisher's LSD value (at the 0.05 level). The LSD values for other years are for the interaction of crop rotations and herbicide programs.

[d] Fluazifop-P + fenoxaprop was applied as a mixture (1991) or sequentially (1992 to 1994) with fomesafen.

Table 6 continued.

Year	Crop	Herbicide(s)	Site one	Site two			
			1994	1991	1992	1993	1994
				%			
1991	C	Imazethapyr	91	55	68	80	83
1992	S	Imazaquin + nicosulfuron					
1993	C	Imazethapyr					
1994	S	Imazaquin + nicosulfuron					
1991	C	Butylate + atrazine	95	82	98	66	95
1992	S	Fomesafen + fluazifop-P + fenoxaprop					
1993	C	Butylate + atrazine					
1994	S	Fomesafen + fluazifop-P + fenoxaprop					
1991	C	Imazethapyr	95	57	97	84	94
1992	S	Fomesafen + fluazifop-P + fenoxaprop					
1993	C	Imazethapyr					
1994	S	Fomesafen + fluazifop-P + fenoxaprop					
1991	C	Butylate + imazethapyr	95	90	92	91	95
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop					
1993	C	Butylate + imazethapyr					
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop					
1991	C	Untreated control	0	0	0	0	0
1992	S	Untreated control					
1993	C	Untreated control					
1994	S	Untreated control					
1991	C	Imazethapyr	95	-[e]	-	-	-
1992	T	Rimsulfuron + rimsulfuron					
1993	S	Imazaquin + nicosulfuron					
1994	C	Imazethapyr					
1991	C	Butylate + atrazine	75	-	-	-	-
1992	T	Trifluralin + metribuzin					
1993	S	Fomesafen + fluazifop-P + fenoxaprop					
1994	C	Butylate + atrazine					
1991	C	Imazethapyr	73	-	-	-	-
1992	T	Trifluralin + metribuzin					
1993	S	Imazaquin + nicosulfuron					
1994	C	Butylate + atrazine					
1991	C	Butylate + imazethapyr	95	-	-	-	-
1992	T	Trifluralin + metribuzin + rimsulfuron					
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop					
1994	C	Butylate + imazethapyr					
1991	C	Untreated control	0	-	-	-	-
1992	T	Untreated control					
1993	S	Untreated control					
1994	C	Untreated control					
LSD (0.05)			6	7	8	10	4

[e] The corn-tomato-soybean rotation was not included at site two.

Table 7. Crop and herbicide rotation effects on large crabgrass densities from 1991 to 1994 at two sites in Painter, VA.[a,b]

Year	Crop[c]	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m^2							
1991	C	Imazethapyr	2	1	2	3	8	2	0	1
1992	C	Imazethapyr								
1993	C	Imazethapyr								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	0	0	3	15	4	3	19	36
1992	C	Butylate + atrazine								
1993	C	Butylate + atrazine								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	2	0	1	6	8	3	0	6
1992	C	Butylate + atrazine								
1993	C	Imazethapyr								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	0	0	0	0	3	1	0	0
1992	C	Butylate + imazethapyr								
1993	C	Butylate + imazethapyr								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	0	6	4	4	16	15	43	24
1992	C	Untreated control								
1993	C	Untreated control								
1994	C	Untreated control								
1991	S	Imazaquin + nicosulfuron	3	1	3	0	8	1	3	2
1992	S	Imazaquin + nicosulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	S	Imazaquin + nicosulfuron								

[a] Population counts were taken 35, 58, 37, and 63 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site one; and 43, 62, 47, and 64 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site two.

[b] Since the interaction of crop rotations and herbicide programs was not significant at the 0.05 level, means for 1992 at site one and for 1991 at site two are the means of the herbicide program averaged over all crop rotations and are presented with the appropiate Fisher's LSD value (at the 0.05 level). The LSD values for other years are for the interaction of crop rotations and herbicide programs.

[c] Crops were: C, corn; S, soybeans; T, tomato.

Table 7 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m²							
1991	S	Fomesafen + fluazifop-P + fenoxaprop[d]	0	0	1	0	4	0	20	1
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + nicosulfuron	1	0	1	0	8	1	2	1
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + nicosulfuron								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop	2	0	3	1	3	1	19	2
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	S	Untreated control	6	6	16	47	16	51	88	63
1992	S	Untreated control								
1993	S	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	4	1	0	1	8	0	1	5
1992	S	Imazaquin + nicosulfuron								
1993	C	Imazethapyr								
1994	S	Imazaquin + nicosulfuron								
1991	C	Butylate + atrazine	0	0	1	0	4	0	3	2
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + atrazine								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	C	Imazethapyr	2	0	0	0	8	0	0	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Imazethapyr								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								

[d] Fluazifop-P + fenoxaprop was applied as a mixture (1991) or sequentially (1992 to 1994) with fomesafen.

Table 7 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m²							
1991	C	Butylate + imazethapyr	0	0	0	0	3	0	1	1
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + imazethapyr								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	C	Untreated control	1	6	5	8	16	42	55	33
1992	S	Untreated control								
1993	C	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	1	1	3	1	-[e]	-	-	-
1992	T	Rimsulfuron + rimsulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	0	0	4	4	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	1	0	2	3	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Imazaquin + nicosulfuron								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	0	0	4	1	-	-	-	-
1992	T	Trifluralin + metribuzin + rimsulfuron								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	2	6	5	23	-	-	-	-
1992	T	Untreated control								
1993	S	Untreated control								
1994	C	Untreated control								
LSD (0.05)			NS	4	5	16	7	12	20	12

[e] The corn-tomato-soybean crop rotation was not included at site two.

Table 8. Crop and herbicide rotation effects on smooth crabgrass densities from 1992 to 1994 at two sites in Painter, VA.[a,b]

Year	Crop[c]	Herbicide(s)	Site one			Site two		
			1992	1993	1994	1992	1993	1994
			plants/m^2					
1991	C	Imazethapyr	0	0	2	2	0	19
1992	C	Imazethapyr						
1993	C	Imazethapyr						
1994	C	Imazethapyr						
1991	C	Butylate + atrazine	0	1	0	0	0	1
1992	C	Butylate + atrazine						
1993	C	Butylate + atrazine						
1994	C	Butylate + atrazine						
1991	C	Imazethapyr	0	0	1	0	0	1
1992	C	Butylate + atrazine						
1993	C	Imazethapyr						
1994	C	Butylate + atrazine						
1991	C	Butylate + imazethapyr	0	0	0	0	0	0
1992	C	Butylate + imazethapyr						
1993	C	Butylate + imazethapyr						
1994	C	Butylate + imazethapyr						
1991	C	Untreated control	0	0	0	0	0	0
1992	C	Untreated control						
1993	C	Untreated control						
1994	C	Untreated control						
1991	S	Imazaquin + nicosulfuron	5	6	90	2	0	19
1992	S	Imazaquin + nicosulfuron						
1993	S	Imazaquin + nicosulfuron						
1994	S	Imazaquin + nicosulfuron						

[a] Population counts were taken 58, 37, and 63 days after PPI treatments were applied in 1992, 1993, and 1994, respectively at site one; and 62, 47, and 64 days after PPI treatments were applied in 1992, 1993, and 1994, respectively at site two.

[b] Since the interaction of crop rotations and herbicide programs was not significant at the 0.05 level, means for 1992 and 1994 at site two are the means of the herbicide program averaged over all crop rotations and are presented with the appropiate Fisher's LSD value (at the 0.05 level). The LSD values for other years are for the interaction of crop rotations and herbicide programs.

[c] Crops were: C, corn; S, soybeans; T, tomato.

Table 8 continued.

Year	Crop	Herbicide(s)	Site one			Site two		
			1992	1993	1994	1992	1993	1994
			%					
1991	S	Fomesafen + fluazifop-P + fenoxaprop[d]	0	0	0	0	0	1
1992	S	Fomesafen + fluazifop-P + fenoxaprop						
1993	S	Fomesafen + fluazifop-P + fenoxaprop						
1994	S	Fomesafen + fluazifop-P + fenoxaprop						
1991	S	Imazaquin + nicosulfuron	0	1	0	0	0	1
1992	S	Fomesafen + fluazifop-P + fenoxaprop						
1993	S	Imazaquin + nicosulfuron						
1994	S	Fomesafen + fluazifop-P + fenoxaprop						
1991	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop	0	1	0	0	0	0
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1991	S	Untreated control	0	1	0	0	0	0
1992	S	Untreated control						
1993	S	Untreated control						
1994	S	Untreated control						
1991	C	Imazethapyr	2	0	7	2	0	19
1992	S	Imazaquin + nicosulfuron						
1993	C	Imazethapyr						
1994	S	Imazaquin + nicosulfuron						
1991	C	Butylate + atrazine	0	0	0	0	0	1
1992	S	Fomesafen + fluazifop-P + fenoxaprop						
1993	C	Butylate + atrazine						
1994	S	Fomesafen + fluazifop-P + fenoxaprop						
1991	C	Imazethapyr	0	0	0	0	0	1
1992	S	Fomesafen + fluazifop-P + fenoxaprop						
1993	C	Imazethapyr						
1994	S	Fomesafen + fluazifop-P + fenoxaprop						

[d] Fluazifop-P + fenoxaprop was applied as a mixture (1991) or sequentially (1992 to 1994) with fomesafen.

Table 8 continued.

Year	Crop	Herbicide(s)	Site one			Site two		
			1992	1993	1994	1992	1993	1994
			%					
1991	C	Butylate + imazethapyr	0	0	0	0	0	0
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1993	C	Butylate + imazethapyr						
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1991	C	Untreated control	0	0	0	0	0	0
1992	S	Untreated control						
1993	C	Untreated control						
1994	S	Untreated control						
1991	C	Imazethapyr	0	0	5	-[e]	-	-
1992	T	Rimsulfuron + rimsulfuron						
1993	S	Imazaquin + nicosulfuron						
1994	C	Imazethapyr						
1991	C	Butylate + atrazine	0	0	1	-	-	-
1992	T	Trifluralin + metribuzin						
1993	S	Fomesafen + fluazifop-P + fenoxaprop						
1994	C	Butylate + atrazine						
1991	C	Imazethapyr	0	3	0	-	-	-
1992	T	Trifluralin + metribuzin						
1993	S	Imazaquin + nicosulfuron						
1994	C	Butylate + atrazine						
1991	C	Butylate + imazethapyr	0	0	0	-	-	-
1992	T	Trifluralin + metribuzin + rimsulfuron						
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop						
1994	C	Butylate + imazethapyr						
1991	C	Untreated control	0	3	0	-	-	-
1992	T	Untreated control						
1993	S	Untreated control						
1994	C	Untreated control						
LSD (0.05)			1	3	28	1	NS	10

[e] The corn-tomato-soybean crop rotation was not included at site two.

Table 9. Crop and herbicide rotation effects on fall panicum densities from 1991 to 1994 at two sites in Painter, VA.[a,b]

Year	Crop[c]	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m^2							
1991	C	Imazethapyr	2	0	0	0	0	0	0	0
1992	C	Imazethapyr								
1993	C	Imazethapyr								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	1	8	2	28	1	4	15	47
1992	C	Butylate + atrazine								
1993	C	Butylate + atrazine								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	3	0	0	4	0	1	0	14
1992	C	Butylate + atrazine								
1993	C	Imazethapyr								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	0	1	0	0	0	0	0	0
1992	C	Butylate + imazethapyr								
1993	C	Butylate + imazethapyr								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	8	0	0	4	2	0	2	1
1992	C	Untreated control								
1993	C	Untreated control								
1994	C	Untreated control								
1991	S	Imazaquin + nicosulfuron	2	0	0	0	0	0	0	0
1992	S	Imazaquin + nicosulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	S	Imazaquin + nicosulfuron								

[a] Population counts were taken 35, 58, 37, and 63 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site one; and 43, 62, 47, and 64 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site two.

[b] Since the interaction of crop rotations and herbicide programs was not significant at the 0.05 level, means for 1991 at both sites are the means of the herbicide program averaged over all crop rotations and are presented with the appropiate Fisher's LSD value (at the 0.05 level). The LSD values for the other years are for the interaction of crop rotations and herbicide programs.

[c] Crops were: C, corn; S, soybeans; T, tomato.

Table 9 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m²							
1991	S	Fomesafen + fluazifop-P + fenoxaprop[d]	1	0	0	1	1	0	0	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + nicosulfuron	3	0	0	0	0	0	0	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + nicosulfuron								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop	0	0	0	0	0	0	0	0
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	S	Untreated control	8	3	0	2	2	0	0	0
1992	S	Untreated control								
1993	S	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	2	0	0	0	0	0	0	1
1992	S	Imazaquin + nicosulfuron								
1993	C	Imazethapyr								
1994	S	Imazaquin + nicosulfuron								
1991	C	Butylate + atrazine	1	0	0	0	1	0	0	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + atrazine								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	C	Imazethapyr	3	0	0	0	0	0	0	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Imazethapyr								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								

[d] Fluazifop-P + fenoxaprop was applied as a mixture (1991) or sequentially (1992 to 1994) with fomesafen.

Table 9 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m²							
1991	C	Butylate + imazethapyr	0	0	0	0	0	0	0	0
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + imazethapyr								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	C	Untreated control	8	4	0	1	2	0	2	2
1992	S	Untreated control								
1993	C	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	2	0	0	0	-[e]	-	-	-
1992	T	Rimsulfuron + rimsulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	1	3	2	1	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	3	2	0	4	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Imazaquin + nicosulfuron								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	0	0	1	0	-	-	-	-
1992	T	Trifluralin + metribuzin + rimsulfuron								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	8	9	0	5	-	-	-	-
1992	T	Untreated control								
1993	S	Untreated control								
1994	C	Untreated control								
LSD (0.05)			3	4	NS	3	1	2	5	11

[e] The corn-tomato-soybean crop rotation was not included at site two.

Table 10. Crop and herbicide rotation effects on yellow nutsedge control from 1992 to 1994 at site two in Painter, VA.[a]

Year	Crop[b]	Herbicide(s)	1992	1993	1994
				%	
1991	C	Imazethapyr	87	70	86
1992	C	Imazethapyr			
1993	C	Imazethapyr			
1994	C	Imazethapyr			
1991	C	Butylate + atrazine	82	90	86
1992	C	Butylate + atrazine			
1993	C	Butylate + atrazine			
1994	C	Butylate + atrazine			
1991	C	Imazethapyr	89	78	83
1992	C	Butylate + atrazine			
1993	C	Imazethapyr			
1994	C	Butylate + atrazine			
1991	C	Butylate + imazethapyr	90	85	89
1992	C	Butylate + imazethapyr			
1993	C	Butylate + imazethapyr			
1994	C	Butylate + imazethapyr			
1991	C	Untreated control	0	0	0
1992	C	Untreated control			
1993	C	Untreated control			
1994	C	Untreated control			
1991	S	Imazaquin + nicosulfuron	83	90	90
1992	S	Imazaquin + nicosulfuron			
1993	S	Imazaquin + nicosulfuron			
1994	S	Imazaquin + nicosulfuron			
1991	S	Fomesafen + fluazifop-P + fenoxaprop[c]	0	0	0
1992	S	Fomesafen + fluazifop-P + fenoxaprop			
1993	S	Fomesafen + fluazifop-P + fenoxaprop			
1994	S	Fomesafen + fluazifop-P + fenoxaprop			
1991	S	Imazaquin + nicosulfuron	0	89	16
1992	S	Fomesafen + fluazifop-P + fenoxaprop			
1993	S	Imazaquin + nicosulfuron			
1994	S	Fomesafen + fluazifop-P + fenoxaprop			
1991	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop	20	70	68
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop			
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop			
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop			
1991	S	Untreated control	0	0	0
1992	S	Untreated control			
1993	S	Untreated control			
1994	S	Untreated control			

[a] Visual control ratings were made 100, 98, and 78 days after PPI treatments were applied in 1992, 1993, and 1994, respectively.

[b] Crops were: C, corn; S, soybeans; T, tomato.

[c] Fluazifop-P + fenoxaprop was applied as a mixture (1991) or sequentially (1992 to 1994) with fomesafen.

Table 10 continued.

Year	Crop	Herbicide(s)	1992	1993	1994
				%	
1991	C	Imazethapyr	78	85	90
1992	S	Imazaquin + nicosulfuron			
1993	C	Imazethapyr			
1994	S	Imazaquin + nicosulfuron			
1991	C	Butylate + atrazine	0	88	36
1992	S	Fomesafen + fluazifop-P + fenoxaprop			
1993	C	Butylate + atrazine			
1994	S	Fomesafen + fluazifop-P + fenoxaprop			
1991	C	Imazethapyr	0	75	33
1992	S	Fomesafen + fluazifop-P + fenoxaprop			
1993	C	Imazethapyr			
1994	S	Fomesafen + fluazifop-P + fenoxaprop			
1991	C	Butylate + imazethapyr	23	85	75
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop			
1993	C	Butylate + imazethapyr			
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop			
1991	C	Untreated control	0	0	0
1992	S	Untreated control			
1993	C	Untreated control			
1994	S	Untreated control			
LSD (0.05) (crop rotation x herbicide program)[d]			7	7	8

[d] The LSD values are for the interaction of crop rotations and herbicide programs.

Table 11. Crop and herbicide rotation effects on yellow nutsedge densities from 1991 to 1994 at two sites in Painter, VA.[a,b]

Year	Crop[c]	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m^2							
1991	C	Imazethapyr	4	5	2	3	5	2	13	4
1992	C	Imazethapyr								
1993	C	Imazethapyr								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	2	1	2	4	1	3	8	3
1992	C	Butylate + atrazine								
1993	C	Butylate + atrazine								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	3	7	2	2	8	2	3	11
1992	C	Butylate + atrazine								
1993	C	Imazethapyr								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	0	0	2	1	5	3	6	3
1992	C	Butylate + imazethapyr								
1993	C	Butylate + imazethapyr								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	2	0	2	0	2	1	6	4
1992	C	Untreated control								
1993	C	Untreated control								
1994	C	Untreated control								
1991	S	Imazaquin + nicosulfuron	2	2	1	0	0	1	1	0
1992	S	Imazaquin + nicosulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	S	Imazaquin + nicosulfuron								

[a] Population counts were taken 35, 58, 37, and 63 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site one; and 43, 62, 47, and 64 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site two.

[b] Since the interaction of crop rotations and herbicide programs was not significant at the 0.05 level, means for 1993 at site one are the means of the crop rotation averaged over all herbicide programs and are presented with the appropiate Fisher's LSD value (at the 0.05 level). The LSD values for other years are for the interaction of crop rotations and herbicide programs.

[c] Crops were: C, corn; S, soybeans; T, tomato.

Table 11 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m²							
1991	S	Fomesafen + fluazifop-P + fenoxaprop[d]	4	4	1	9	27	46	44	286
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + nicosulfuron	0	5	1	0	6	30	18	99
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + nicosulfuron								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop	5	5	1	9	4	17	15	93
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	S	Untreated control	0	0	1	1	7	10	22	18
1992	S	Untreated control								
1993	S	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	3	1	2	1	5	3	7	0
1992	S	Imazaquin + nicosulfuron								
1993	C	Imazethapyr								
1994	S	Imazaquin + nicosulfuron								
1991	C	Butylate + atrazine	2	6	2	2	7	57	23	60
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + atrazine								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								
1991	C	Imazethapyr	2	10	2	7	19	30	25	33
1992	S	Fomesafen + fluazifop-P + fenoxaprop								
1993	C	Imazethapyr								
1994	S	Fomesafen + fluazifop-P + fenoxaprop								

[d] Fluazifop-P + fenoxaprop was applied as a mixture (1991) or sequentially (1992 to 1994) with fomesafen.

Table 11 continued.

Year	Crop	Herbicide(s)	Site one				Site two			
			1991	1992	1993	1994	1991	1992	1993	1994
			plants/m^2							
1991	C	Butylate + imazethapyr	0	13	2	3	19	38	17	21
1992	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1993	C	Butylate + imazethapyr								
1994	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1991	C	Untreated control	2	0	2	2	24	7	21	11
1992	S	Untreated control								
1993	C	Untreated control								
1994	S	Untreated control								
1991	C	Imazethapyr	0	1	13	1	-[e]	-	-	-
1992	T	Rimsulfuron + rimsulfuron								
1993	S	Imazaquin + nicosulfuron								
1994	C	Imazethapyr								
1991	C	Butylate + atrazine	0	6	13	11	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + atrazine								
1991	C	Imazethapyr	4	28	13	3	-	-	-	-
1992	T	Trifluralin + metribuzin								
1993	S	Imazaquin + nicosulfuron								
1994	C	Butylate + atrazine								
1991	C	Butylate + imazethapyr	1	12	13	1	-	-	-	-
1992	T	Trifluralin + metribuzin + rimsulfuron								
1993	S	Imazaquin + fomesafen + fluazifop-P + fenoxaprop								
1994	C	Butylate + imazethapyr								
1991	C	Untreated control	1	2	13	2	-	-	-	-
1992	T	Untreated control								
1993	S	Untreated control								
1994	C	Untreated control								
LSD (0.05)			NS	11	8	6	17	26	17	43

[e] The corn-tomato-soybean crop rotation was not included at site two.

Chapter IV

Response of Smooth Pigweed (*Amaranthus hybridus*) and Livid Amaranth (*A. lividus*) to Several Imidazolinone and Sulfonylurea Herbicides

Abstract. Field studies in 1991 and 1992 investigated chlorimuron, imazaquin, imazethapyr, nicosulfuron, primisulfuron, and thifensulfuron on a population of smooth pigweed in Painter, VA with no history of acetolactate synthase (ALS)-inhibitor herbicides. Imazethapyr and nicosulfuron gave the highest smooth pigweed control. Therefore, these herbicides were used in field and greenhouse studies to investigate susceptibility of populations of smooth pigweed and livid amaranth to ALS-inhibitor herbicides. Approximately 5 million Painter smooth pigweed plants were treated with imazethapyr or nicosulfuron from 1992 to 1994 and no ALS-inhibitor-resistant plants were identified. Smooth pigweed seeds were collected from the Painter population and from populations with histories of six consecutive, annual applications of imazaquin in combination with trifluralin or pendimethalin in Marion, MD and Oak Hall, VA. Livid amaranth seeds were collected from a population in Warren County, NJ with a history of at least three consecutive, biannual applications of imazethapyr. The tolerance of the Painter, Marion, and Oak Hall smooth pigweed and the livid amaranth population to nicosulfuron and either imazaquin or imazethapyr was investigated in the greenhouse. Painter smooth pigweed was controlled by both imazethapyr and nicosulfuron, while the Marion and Oak Hall populations tolerated 560 and 1120 g ai/ha imazaquin, respectively,

herbicide-resistant biotypes of *Amaranthus spp.* is likely to occur because of their generally high susceptibility to sulfonylurea and imidazolinone herbicides and their generally high fecundity (3, 7, 11, 15, 16). Biotypes of smooth pigweed have been reported to be resistant to the triazine herbicides (5) and to ALS-inhibiting herbicides (13, 21).

The first objective of this research was to determine if biotypes resistant to ALS-inibitor herbicides could be identified in a population of smooth pigweed with no history of ALS-inhibitor use. The second objective was to determine if populations of smooth pigweed and livid amaranth with various histories of imidazolinone herbicide use could tolerate high rates of imazaquin or imazethapyr and nicosulfuron.

MATERIALS AND METHODS

Plant sources and field histories. Smooth pigweed populations from Painter and Oak Hall, VA, and Marion, MD, and a population of livid amaranth from Warren County, NJ were used in this research. Smooth pigweed from Painter was located on the Eastern Shore Agricultural Research and Extension Center, and had little or no history of ALS-inhibitor herbicide use. The Painter population was considered the wild type susceptible population. Smooth pigweed from Marion and Oak Hall were from fields which had received applications of imazaquin alone or in combination with pendimethalin or trifluralin each year since 1986. Smooth pigweed was no longer controlled by imazaquin

at these sites. Escapes were first observed in 1991 at both locations, but were not reported until 1993 and 1994 at Marion and Oak Hall, respectively. Smooth pigweed seeds were collected following the reports of high populations in 1993 and 1994 at Marion and Oak Hall, respectively. Livid amaranth seeds were collected from a field in Warren County, NJ that had received at least three consecutive biannual applications of imazethapyr (NJ1[2]) and from a field with no history of imazethapyr use (NJ2[2]). Detailed records from these sites were not available.

Initial field studies. Research was conducted in 1991 and 1992 to determine the control of smooth pigweed by several sulfonylurea and imidazolinone herbicides. Studies were conducted in Painter, VA on a State sandy loam soil (Typic Hapludults) with a pH of 6.1 and an organic matter content of 1% without a crop. Plot sizes were 1.5 or 2.0 by 3.0 m, and herbicides were applied POST with a backpack sprayer. Applications were made in water at 187 L/ha with a pressure of 234 kPa through flat fan spray tips[3]. Heights of smooth pigweed at application were 13 to 23 cm in 1991. In 1992, herbicides were applied when smooth pigweed was 8 to 13 cm and 20 to 25 cm.

Herbicides included 13 g ai/ha chlorimuron, 140 g ai/ha imazaquin, 70 g ai/ha imazethapyr, 35 g ai/ha nicosulfuron, 39 g ai/ha primisulfuron, and 4 g ai/ha

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[2] Abbreviations: NJ1, livid amaranth population with history of at least three biannual applications of imazethapyr; NJ2, livid amaranth population with no history of imazethapyr applications.

[3] Teejet 8003 flat fan spray tips, Spraying Systems Co., North Ave., Wheaton, IL 60188.

thifensulfuron. Treatments included a non-ionic surfactant[4] in both years, and 105 g ai/ha clethodim {(*E,E*)-(±)-2-[1-[[(3-chloro-2-propenyl)oxy]imino]propyl]-5-[2-(ethylthio)propyl]-3-hydroxy-2-cyclohexen-1-one} in 1991 to control annual grasses. Visual estimates of percent control were made 35 DAT in 1991, and 43 and 31 DAT to 8 to 13 cm and 20 to 25 cm smooth pigweed, respectively in 1992.

Statistical Analyses. Studies were conducted using a randomized complete block design with three replications. Data were analyzed by ANOVA, and means were separated using Duncan's multiple range test at the 0.05 significance level. The data were arcsine transformed to produce a near-normal distribution (1, 23), but are presented in untransformed percentages while the mean separation procedures reflect the differences found with the transformed data.

Large plot screens. Based on the results of the initial field studies, imazethapyr and nicosulfuron were selected to screen high natural populations of smooth pigweed in Painter for resistant individuals. Large areas of smooth pigweed were treated with 70 g/ha imazethapyr and 35 g/ha nicosulfuron in 1992, 1993, and 1994. Herbicides were applied POST with a tractor-mounted sprayer in water at 234 L/ha with a pressure of 207 kPa through flat fan spray tips[3] and included a non-ionic surfactant[4] at 0.25% v/v. The treated areas were monitored throughout the growing season for resistant plants (defined as

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[4] Ortho X-77, Valent USA Corp., 1333 N. California Blvd., Walnut Creek, CA 94596-8025. Nonionic surfactant with 80% principal functioning agents as: alkylarylpolyoxy ethylene glycols, free fatty acids, and isopropanol.

actively growing plants with little to no effects from the herbicide). In 1992, imazethapyr and nicosulfuron were each applied to three 502 m^2 areas in which smooth pigweed was 5 to 8, 15 to 20, or 30 to 36 cm tall. In 1993, imazethapyr and nicosulfuron were each applied to one 344 m^2 area and three 1255 m^2 areas when smooth pigweed was 13 to 18 cm tall. In 1994, both herbicides were applied to one 335 m^2 area when smooth pigweed was 15 to 20 cm tall. Mean smooth pigweed densities and the total number of treated plants were determined in 1992 for all of the plots by counting the number of plants in a 50 by 50 cm area at four random locations. In 1993 and 1994, smooth pigweed densities and the total number of plants treated were determined for each plot by counting the number of plants in a 50 by 50 cm area at five random locations in each plot.

Greenhouse studies. Greenhouse studies were conducted to determine if the smooth pigweed from Painter, Marion, or Oak Hall and livid amaranth from NJ could tolerate high rates of imazaquin or imazethapyr and nicosulfuron. A greenhouse soil mix consisting of State sandy loam, sand, and peat (23:13:1 by wt) was placed in flats (6.9 L) or styrofoam cups (470 ml). Smooth pigweed or livid amaranth seeds were sprinkled on the surface and covered with 0.6 cm of greenhouse soil mix. The seeds were germinated and the plants grown under natural sunlight or under natural sunlight supplemented with metal halide lamps in the greenhouse for a photoperiod of at least 14 h and were watered manually as needed. A complete fertilizer[5] was also applied weekly to maintain active growth.

[5] Peters 20-20-20 general purpose fertilizer, The Wetzel Seed Co. Inc., 1345 Diamond Springs Road, Virginia Beach, VA 23455.

Herbicides were applied POST in water at 210 to 234 L/ha with a pressure of 172 to 276 kPa through flat fan spray tips[6]. Smooth pigweed seeds from Painter or Marion in both studies and livid amaranth seeds in study one were planted in flats, and the plants were not thinned. In these studies, three representative plants were harvested to obtain fresh and dry weights. Smooth pigweed seeds from Oak Hall in both studies and livid amaranth seeds in study two were planted in styrofoam cups. NJ2 was included in study two to compare the response of NJ1 to that of a population with no history of imazethapyr use. The Oak Hall smooth pigweed was thinned to five uniform plants per cup, and the livid amaranth in study two was thinned to two plants per cup because of poor seed germination. In these experiments, all of the plants in the cup were harvested. Plants were maintained for 28 to 34 DAT when visual estimates of percent control were made. Control estimates were based on a scale of 0 to 100% where 0 equals no control and 100 equals complete control (death of plants) compared to an untreated control (4). Plants were then harvested, fresh weights were determined, and the plants were dried for a minimum of 48 h at 65 C before dry weights were determined.

Since Painter smooth pigweed was susceptible to both imazaquin and imazethapyr, it was treated with 0, 70, 140, 280, and 560 g/ha imazethapyr and 0, 35, 70, 140, and 280 g/ha nicosulfuron. Marion and Oak Hall smooth pigweed populations were selected with imazaquin. Therefore, Marion smooth pigweed was treated with 0, 70, 140, 280, and 560 g/ha imazaquin and 0, 35, 70, 140, and 280 g/ha nicosulfuron; and Oak Hall smooth

[6] Teejet 8001E or 8002E flat fan spray tips, Spraying Systems Co., North Ave. Wheaton, IL 60188.

pigweed was treated with 0, 70, and 1120 g/ha imazaquin and 0, 35, and 280 g/ha nicosulfuron. Since NJ1 was selected with imazethapyr, livid amaranth was treated with 0, 70 and 560 g/ha imazethapyr and 0, 35, and 280 g/ha nicosulfuron. Herbicides were applied with a non-ionic surfactant[4] at 0.25% v/v to 3- to 8-cm plants in all studies.

Statistical analyses. Randomized complete block designs were used in studies with the Painter and Marion smooth pigweed and in study one with livid amaranth. Completely randomized designs were used in studies with the Oak Hall smooth pigweed and study two with livid amaranth. Studies had three or four replications and were repeated. Following an analysis for homogeneity of variance, data from the two studies for each population were combined, except that data from the two livid amaranth studies could not be combined. Data were analyzed statistically by ANOVA, and means were separated using Duncan's multiple range test at the 0.05 significance level. Control estimates were arcsine transformed prior to analysis to produce a near-normal distribution (1, 23). The data are presented in untransformed percentages while the mean separation procedures reflect the differences found with the transformed data.

RESULTS AND DISCUSSION

Initial field studies. Painter smooth pigweed control was highest from imazethapyr and nicosulfuron with 88% or higher control (Table 1), so these herbicides were selected as the imidazolinone and sulfonylurea herbicides, respectively, for subsequent field screens

and greenhouse studies. Smooth pigweed control from imazaquin was also 80% or higher, and in 1991 was 98%. Control from thifensulfuron was variable with control of 8- to 13-cm plants in 1992 only 68%, but this may have been due to seed germination after application of thifensulfuron. Control from chlorimuron and primisulfuron was less than 60%. In 1992, smooth pigweed control was generally not affected by the size of the plants except with thifensulfuron.

Large plot screens. High numbers of smooth pigweed plants in Painter were treated with imazethapyr or nicosulfuron to determine if resistant individuals were present in the population. In 1992, the mean smooth pigweed density was determined for the entire treated area, and approximately 1.6 million plants were treated with imazethapyr or nicosulfuron (Table 2). In 1993 and 1994, the mean smooth pigweed density and the total number of plants treated were determined for each plot area. From these data, the total number of plants treated with imazethapyr and nicosulfuron was 856,000 and 737,000, respectively in 1993, and 37,000 and 73,000, respectively in 1994. The total number of plants treated in all 3 years with imazethapyr and nicosulfuron was 2.5 and 2.4 million, respectively. Among the almost 5 million treated plants in Painter, no resistant individuals were observed. A dominant or semi-dominant allele for resistance to sulfonylurea or imidazolinone herbicides was apparently not present in the plants treated from this population. Other research has indicated that resistance to ALS-inhibitor herbicides is due to single gene mutations, controlled by a dominant or semi-dominant allele, that are estimated to occur at a mutation rate of 10^{-6} (14, 19). Our research suggests that the

mutation for smooth pigweed resistance to ALS-inhibitor herbicides may actually occur at a lower frequency in nature than has been estimated, but these results are from only one population. Since this population is still susceptible, this research supports the theory that the mutation for resistance must be present in the population prior to application of the herbicide in order for a resistant population to develop. More research should be conducted on this and other susceptible populations to confirm these theories.

Greenhouse studies. Control of Painter smooth pigweed from imazethapyr and nicosulfuron was high (Table 3). Control of Painter smooth pigweed from 70 g/ha imazethapyr and 35 and 70 g/ha nicosulfuron was 81 to 85%, while control from other treatments was 90% or higher. All treatments reduced fresh and dry weights of this population similarly relative to the untreated control. While control of the Painter population was not complete from 70 g/ha imazethapyr and 35 and 70 g/ha nicosulfuron, this population is susceptible to these herbicides. Field studies indicated that this population is also susceptible to imazaquin (Table 1).

Marion smooth pigweed was not controlled, and fresh and dry weights were not reduced with as much as 560 g/ha imazaquin, which is eight times the registered rate (Table 4). Nicosulfuron (35 g/ha) controlled only 50% of Marion smooth pigweed and did not reduce fresh weights, while 70 g/ha nicosulfuron controlled 70% of this population and reduced fresh weights approximately 50%. Higher nicosulfuron rates were required to control 90% or more of the Marion population. All of the treated plants from the Marion population responded similarly to imazaquin and nicosulfuron indicating that the

seeds collected were homogeneous in their tolerance to these herbicides.

Two rates each of imazaquin and nicosulfuron were tested on the Oak Hall and livid amaranth populations. Imazaquin did not control the Oak Hall population at 70 g/ha and controlled only 18% of this population at 1120 g/ha (Table 5). Fresh and dry weights were not reduced by either rate of imazaquin. Nicosulfuron at 35 g/ha controlled 73% of this population, and reduced both fresh and dry weights. Control of the Oak Hall population by 280 g/ha nicosulfuron was essentially complete. Variability in the response of individual plants from the Oak Hall population to imazaquin and nicosulfuron compared to the Marion population indicates that the Oak Hall population is heterogeneous with regard to tolerance to these herbicides, but this observation was not measured quantitatively.

Control of NJ1 by 70 and 560 g/ha imazethapyr was 15% or less in both studies, and fresh and dry weights were not reduced by either rate (Table 6). Imazethapyr (70 g/ha) controlled 65%, and 560 g/ha controlled higher than 90% of NJ2 in study two (data not shown). Control of NJ1 by 35 g/ha nicosulfuron was 30 and 58% in studies one and two, respectively, and fresh weights of NJ1 were not reduced by this treatment in study one (Table 6). However, control of NJ2 by 35 g/ha nicosulfuron was only 25% in study two (data not shown). Livid amaranth appears to be tolerant to registered rates of nicosulfuron. Control of both populations of livid amaranth by 280 g/ha nicosulfuron was nearly complete.

Marion and Oak Hall smooth pigweed and NJ1 appear to be resistant to the

imidazolinone herbicides, but this conclusion should be confirmed with direct comparisons in the same studies to a susceptible biotype of each species. Marion and Oak Hall smooth pigweed also appear to have low levels of cross-resistance to nicosulfuron, which should also be verified with direct comparisons to a susceptible biotype. Further research has been conducted on the Marion population and is reported in a later chapter of this dissertation.

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Table 1. Smooth pigweed control from several ALS-inhibiting herbicides applied at various plant heights in 1991 and 1992 at Painter, VA.[a]

		1991[b]	1992[c]	
Herbicide	Rate	13 to 23 cm	8 to 13 cm	20 to 25 cm
	g/ha	%		
Chlorimuron	13	53 c	50 e	55 c
Imazaquin	140	98 a	80 bc	88 b
Imazethapyr	70	99 a	88 ab	88 b
Nicosulfuron	35	99 a	95 a	98 a
Primisulfuron	39	57 c	57 de	40 d
Thifensulfuron	4	95 b	68 cd	82 b
Untreated control	-	0 d	0 f	0 e

[a] Means within a column followed by the same letter do not differ at the 0.05 significance level by Duncan's multiple range test.

[b] All treatments in 1991 were applied to 13- to 23-cm smooth pigweed and included 105 g/ha clethodim and 0.25% v/v non-ionic surfactant. Visual estimates of percent control were made 35 DAT.

[c] All treatments in 1992 included 0.25% v/v non-ionic surfactant and were applied to 8- to 13- and 20- to 25-cm smooth pigweed. Visual estimates of percent control were made 43 and 31 DAT to 8- to 13- and 20- to 25-cm smooth pigweed, respectively.

Table 2. Number of smooth pigweed plants screened with imazethapyr and nicosulfuron for resistance in fields at Painter, VA.

Herbicide[a]	Rate	1992[b]	1993[c]	1994[d]	Total
	g/ha	number of plants treated x 1000 ± S. D.			
Imazethapyr	70	1606 ± 789	896 ± 174	37 ± 18	2539
Nicosulfuron	35	1606 ± 789	769 ± 269	73 ± 36	2448

[a] Herbicides were applied with 0.25% v/v non-ionic surfactant.

[b] Numbers of plants treated with imazethapyr and nicosulfuron in 1992 are the total from three 502 m^2 areas treated when smooth pigweed was 5 to 8, 15 to 20, or 30 to 36 cm. Mean smooth pigweed densities were 1067 plants/m^2.

[c] Numbers of plants treated in 1993 are totals from one 344 m^2 and three 1255 m^2 areas. Mean smooth pigweed densities were 262 and 220 plants/m^2 in the smaller areas treated with imazethapyr and nicosulfuron, respectively, and 203 and 176 plants/m^2 in the larger areas treated with imazethapyr and nicosulfuron, respectively.

[d] Numbers of plants treated in 1994 are the total from one 335 m^2 area with smooth pigweed densities of 112 and 218 plants/m^2 for areas treated with imazethapyr and nicosulfuron, respectively.

Table 3. Percent control, fresh weights, and dry weights of smooth pigweed from Painter, VA treated with imazethapyr and nicosulfuron POST in the greenhouse.[a,b,c]

Herbicide	Rate	Control	Fresh weight	Dry weight
	g/ha	%	g	
Imazethapyr	70	81 c	4.5 b	0.8 b
Imazethapyr	140	90 abc	2.3 b	0.6 b
Imazethapyr	280	92 ab	2.5 b	0.8 b
Imazethapyr	560	97 a	1.5 b	0.7 b
Nicosulfuron	35	85 bc	2.3 b	0.5 b
Nicosulfuron	70	83 c	4.0 b	0.8 b
Nicosulfuron	140	91 abc	1.6 b	0.5 b
Nicosulfuron	280	93 ab	1.3 b	0.5 b
Untreated control	-	0 d	54.1 a	9.5 a

[a] Means are an average from two studies with three replications, and those within a column followed by the same letter do not differ at the 0.05 significance level by Duncan's multiple range test.

[b] All treatments included 0.25% v/v non-ionic surfactant.

[c] Visual estimates of percent control were made and three plants/flat were harvested 28 DAT.

Table 5. Percent control, fresh weights, and dry weights of smooth pigweed from Oak Hall, VA treated with imazaquin and nicosulfuron POST in the greenhouse.[a,b,c]

Herbicide	Rate	Control	Fresh weight	Dry weight
	g/ha	%	g	
Imazaquin	70	2 d	13.7 a	3.4 a
Imazaquin	1120	18 c	11.1 ab	2.5 a
Nicosulfuron	35	73 b	6.6 bc	1.3 b
Nicosulfuron	280	91 a	4.7 c	0.9 b
Untreated control	-	0 d	12.8 a	3.2 a

[a] Means are an average from two studies with three replications, and those within a column followed by the same letter do not differ at the 0.05 significance level by Duncan's multiple range test.

[b] All treatments included 0.25% v/v non-ionic surfactant.

[c] Visual estimates of percent control were made and five plants/cup were harvested 28 DAT.

Table 6. Percent control, fresh weights, and dry weights of livid amaranth from NJ treated with imazethapyr and nicosulfuron POST in two greenhouse studies.[a,b]

		Study one[c]			Study two[d]		
Herbicide	Rate	Control	Fresh weight	Dry weight	Control	Fresh weight	Dry weight
		%	g		%	g	
Imazethapyr	70	0 c	45.9 a	5.0 ab	8 c	8.2 a	1.4 a
Imazethapyr	560	8 c	42.0 a	4.1 ab	15 c	9.0 a	1.7 a
Nicosulfuron	35	30 b	50.5 a	3.5 b	58 b	3.7 b	0.6 b
Nicosulfuron	280	94 a	2.7 b	0.4 c	99 a	0.2 c	0.1 b
Untreated control	-	0 c	51.6 a	5.8 a	0 d	7.1 a	1.3 a

[a] Means within a column followed by the same letter do not differ at the 0.05 significance level by Duncan's multiple range test. Each study had three replications.

[b] All treatments included 0.25% v/v non-ionic surfactant.

[c] Visual estimates of percent control were made and three plants/flat were harvested 31 DAT in study one.

[d] Due to poor seed germination, cups in study two were thinned to two plants/cup. Visual estimates of percent control were made and two plants/cup were harvested 28 DAT.

Chapter V

Characterization of Imidazolinone-Resistant Smooth Pigweed (*Amaranthus hybridus*)

Abstract. Following six consecutive, annual applications of imazaquin in combination with trifluralin or pendimethalin to several soybean fields on the Delmarva Peninsula, unacceptable smooth pigweed control was observed. Field and greenhouse studies were conducted to determine if this population was resistant to imazaquin and other herbicides. Seeds were collected from this population (R) and from another smooth pigweed population (S) known to be susceptible to acetolactate synthase (ALS)-inhibiting herbicides for use in greenhouse studies. Field studies on both populations were conducted on a course-textured soil with an organic matter content of less than 1.5%. In field studies pendimethalin, metribuzin, halosulfuron, and flumiclorac controlled less than 75% of both the R and S populations. Also, chlorimuron, primisulfuron, CGA-152005, and lactofen gave above 75% control, while thifensulfuron, nicosulfuron and ASC-67040 gave above 90% control of both populations. However, imazaquin and imazethapyr gave complete control of the S population while providing no control of the R population, and pyrithiobac controlled 99 and 90% of the R and S population, respectively. Greenhouse studies confirmed high levels of resistance to imazaquin and imazethapyr and low levels of cross-resistance to rimsulfuron and chlorimuron in the R population. Susceptibility of the R population to nicosulfuron, thifensulfuron, pyrithiobac, and pendimethalin was

comparable to that of the S population. **Nomenclature:** ASC-67040, 1-(4,6-dimethoxypyrimidin-2-yl)-3-[(3-trifluoromethyl-pyridin-2-yl)sulfonyl]urea; CGA-152005, 1-(4-methoxy-6-methyl-triazin-2-yl)-3-[2-(3,3,3-trifluoropropyl)-phenylsulfonyl]-urea; chlorimuron, 2-[[[[(4-chloro-6-methoxy-2-pyrimidinyl)amino]carbonyl]amino]sulfonyl]benzoic acid; flumiclorac, [2-chloro-4-fluoro-5-(1,3,4,5,6,7-hexahydro-1,3-dioxo-2*H*-isoindol-2-yl)phenoxy]acetic acid; halosulfuron, methyl 3-chloro-5-(4,6-dimethoxypyrimidin-2-ylcarbamoylsulfamoyl)-1-methylpyrazole-4-carboxylate; imazaquin, 2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-3-quinolinecarboxylic acid; imazethapyr, 2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-5-ethyl-3-pyridinecarboxylic acid; lactofen, (±)-2-ethoxy-1-methyl-2-oxoethyl 5-[2-chloro-4-(trifluoromethyl)phenoxy]-2-nitrobenzoate; metribuzin, 4-amino-6-(1,1-dimethylethyl)-3-(methylthio)-1,2,4-triazin-5(4*H*)-one; nicosulfuron, 2-[[[[(4,6-dimethoxy-2-pyrimidinyl)amino]carbonyl]amino]sulfonyl]-*N,N*-dimethyl-3-pyridinecarboxamide; pendimethalin, *N*-(1-ethylpropyl)-3,4-dimethyl-2,6-dinitrobenzenamine; primisulfuron, 2-[[[[[4,6-bis(difluoromethoxy)-2-pyrimidinyl]amino]carbonyl]amino]sulfonyl]benzoic acid; pyrithiobac, 2-chloro-6-[(4,6-dimethoxy-2-pyrimidinyl)thio]benzoic acid; rimsulfuron, *N*-[[(4,6-dimethoxy-2-pyrimidinyl)amino]carbonyl]-3-(ethylsulfonyl)-2-pyridinesulfonamide; thifensulfuron, 3-[[[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl)amino]carbonyl]amino]sulfonyl]-2-thiophenecarboxylic acid; trifluralin, 2,6-dinitro-*N,N*-dipropyl-4-

(trifluoromethyl)benzenamine; smooth pigweed, *Amaranthus hybridus* L. #[1] AMACH; soybeans, *Glycine max* (L.) Merr.

Additional index words: ALS-inhibitor resistance, herbicide resistance, imidazolinone resistance, sulfonylurea herbicides, ASC-67040, CGA-152005, chlorimuron, flumiclorac, halosulfuron, imazaquin, imazethapyr, lactofen, metribuzin, nicosulfuron, pendimethalin, primisulfuron, pyrithiobac, rimsulfuron, thifensulfuron, trifluralin, *Glycine max,* AMACH.

INTRODUCTION

Imazaquin and imazethapyr are selective imidazolinone herbicides for soybean which control numerous broadleaf and certain grass weeds. The mode of action of these herbicides is the inhibition of ALS (EC 4.1.3.18) (also called acetohydroxyacid synthase), the first common enzyme in the biosynthesis of the branched chain amino acids - valine, leucine and isoleucine (8, 20, 21, 23, 24). Since their commercial introduction in the late 1980's (27), imazaquin and imazethapyr use increased to 47% of the soybean area in the major soybean producing states by 1992 (28). There are three other classes of commercially available herbicides that inhibit ALS: the sulfonylureas, the triazolopyrimidines and the pyrimidinyl thiobenzoates (6, 8, 16, 18, 25).

There are at least 17 weed species with biotypes resistant to ALS-inhibiting herbicides

[1] Letters following this symbol are a WSSA-approved computer code from Composite List of Weeds, Revised 1989. Available from WSSA, 1508 West University Ave., Champaign, IL 61821-3133.

including prostrate pigweed (*Amaranthus blitoides* S. Wats.), redroot pigweed (*Amaranthus retroflexus* L.), Palmer amaranth (*Amaranthus palmeri* S. Wats.), and common waterhemp (*Amaranthus rudis* Sauer); all of which were selected by continuous, annual applications of ALS-inhibiting herbicides (9, 10, 12, 13, 14, 15, 17, 18, 19, 22, 26)[2]. Of the ALS-inhibitor-resistant weed biotypes, only common cocklebur (*Xanthium strumarium* L.), smooth pigweed, Palmer amaranth, and common waterhemp biotypes were selected with an imidazolinone herbicide. All other ALS-inhibitor-resistant biotypes were selected with sulfonylurea herbicides except one biotype of rigid ryegrass (*Lolium rigidum* Gaudin) which was selected with diclofop {(±)-2-[4-(2,4-dichlorophenoxy)phenoxy]propanoic acid} (12, 14, 15, 18).

The resistant weed populations documented to date are often cross-resistant at varying levels to members of the same herbicide family as the selection agent, but display varying patterns of cross-resistance to members of other ALS-inhibitor herbicide families (5, 7, 10, 12, 14, 15, 18, 19). These varying patterns of cross-resistance suggest that each population should be analyzed separately for its level of resistance and its patterns of cross-resistance.

Soybean is planted on over 50% of the harvested cropland on Virginia's portion of the Delmarva, and many farmers on the Delmarva grow soybean continuously year after year (2). A farmer near Marion, MD had grown soybean without rotation since 1986. Imazaquin (140 g ai/ha) was applied to these fields each year from 1986 to1993, and was

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[2] Barrentine, W.L. 1993. Personal communication. Delta Res. and Ext. Ctr., Stoneville, MS 38776.

often applied in combination with 560 to 840 g ai/ha trifluralin or 840 g ai/ha pendimethalin. The farmer observed unacceptable smooth pigweed control in 1992 and 1993. Seeds were collected in the fall of 1993 from plants in one of these fields. An initial greenhouse study conducted in the winter of 1993 indicated that this population of smooth pigweed tolerated eight times the registered postemergence rate of imazaquin, and was not completely controlled by 35 and 70 g ai/ha nicosulfuron (previous chapter).

The first objective of this research was to investigate the activity of various ALS-inhibitor herbicides and herbicides with other modes of action on this population and an imazaquin-susceptible population of smooth pigweed. The second objective was to determine if the smooth pigweed population in Marion was resistant to imazaquin and other ALS-inhibitor herbicides.

MATERIALS AND METHODS

Field studies. Field studies were conducted in 1994 in Marion, MD (R population) and in Painter, VA (S population) to investigate the tolerance of these smooth pigweed populations to commercial rates of various herbicides. The soil in Marion is a Fallsington and Dragston fine sandy loam (Typic Ochraquults) with a pH of 6.0 and 1.4% organic matter. The site in Marion was prepared with a chisel plow followed with a tandem disc three times, and soybean ('Hutcheson') were planted with a drill in rows spaced 18 cm apart. The soil in Painter is a State sandy loam (Typic Hapludults) with a pH of 6.1 and

1% organic matter. The site in Painter was prepared with a heavy tandem disc once followed twice by a field cultivator with a double rolling basket, but no crop was planted. The plot size in Marion was 2.1 m wide by 6.1 m long and in Painter was 2.4 m wide by 6.1 m long. Herbicide treatments in Marion were applied with a backpack sprayer, while the treatments in Painter were applied with a tractor-mounted plot sprayer. Applications were made at both locations in water at a rate of 234 L/ha with a pressure of 207 kPa through flat fan spray tips[3].

Various ALS-inhibitor herbicides including imidazolinone and sulfonylurea herbicides and the pyrimidinylthiobenzoate herbicide, pyrithiobac, were included to investigate the control of R and S smooth pigweed. These herbicides are currently being used or developed for use in soybean, corn (*Zea mays* L.), cotton (*Gossypium hirsutum* L.), small grains, turf, or certain vegetable crops Several non-ALS-inhibitor herbicides currently used for weed control in soybean were also included in field studies to investigate their activity on the R and S populations. Pendimethalin was included because it or another dinitroaniline herbicide, trifluralin, was applied in combination with imazaquin during the selection of the R population. Pendimethalin is registered for control of smooth pigweed and is recommended for the control of triazine-resistant smooth pigweed (2). Metribuzin was also included since it is registered for control of smooth pigweed in soybean. Also included were two non-ALS-inhibitor herbicides registered for POST control of smooth pigweed in soybean: the *N*-phenylphthalimide herbicide, flumiclorac, and the

[2] Teejet 8003 flat fan spray tips, Spraying Systems Co., North Ave. Wheaton, IL 60188

diphenylether herbicide, lactofen.

Pendimethalin (1120 g/ha), metribuzin (280 g ai/ha), and imazaquin (140 g/ha) were applied PRE. Imazaquin (140 g/ha), imazethapyr (70 g ai/ha), ASC-67040 (49 g ai/ha), CGA-152005 (20 g ai/ha), chlorimuron (9 g ai/ha), flumiclorac (29 g ai/ha), halosulfuron (35 g ai/ha), lactofen (70 g ai/ha), nicosulfuron (35 g/ha), primisulfuron (40 g ai/ha), pyrithiobac (70 g ai/ha), thifensulfuron (4 g ai/ha) and a combination of imazethapyr and lactofen at 70 g/ha each were applied POST to 5- to 10-cm smooth pigweed. All POST treatments included a non-ionic surfactant[4] at 0.25% v/v, and 30% liquid nitrogen at 1.0% v/v was included with imazethapyr. Visual estimates of percent control were made at 20 days after the POST treatments (DAPT)[5] in Marion, and at 32 DAPT in Painter. Control estimates were based on a scale of 0 to 100% where 0 equals no control and 100 equals complete control (death of plants) compared to an untreated control. Smooth pigweed densities were determined 20 and 32 DAPT in Marion and Painter, respectively by counting the number of plants in a 50- by 50-cm area at three random locations in each plot.

Statistical analyses. Studies at both Marion and Painter were conducted using a randomized complete block design with three or four replications, and were repeated at an

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[3] Ortho X-77, Valent USA Corp., 1333 N. California Blvd., Walnut Creek, CA 94596-8025. Nonionic surfactant with 80% principal functioning agents as: alkylarylpolyoxy ethylene glycols, free fatty acids, and isopropanol.

[4] Abbreviations: DAPT, days after postemergence treatments; DAT,days after treatment; GR_{50}, rate required to cause a 50% reduction in dry weights.

covered with 0.6 cm of greenhouse soil mix. The seeds were germinated and the plants grown under natural sunlight, or under natural sunlight supplemented with metal halide lamps in the greenhouse for a photoperiod of at least 14 h, and were watered manually as needed. A complete fertilizer[7] was also applied weekly to maintain active growth. Herbicides, except pendimethalin, were applied PRE or POST with a backpack sprayer in a water carrier at a rate of 234 L/ha with a pressure of 241 kPa through flat fan spray nozzles[2]. Pendimethalin was applied uniformly to the greenhouse soil mix, and the soil was mixed for 5 min to incorporate pendimethalin. The treated soil was placed in cups prior to planting seeds. Plants were maintained in all studies for 28 to 31 days after treatment (DAT[4]) when visual estimates of percent control were made, and five representative shoots were harvested and dried for a minimum of 48 h at 65 C before determining dry weights. Control estimates were based on a scale of 0 to 100% where 0 equals no control and 100 equals complete control (death of plants) compared to an untreated control.

Imazaquin, imazethapyr, several sulfonylurea herbicides, pyrithiobac, and pendimethalin were included in greenhouse studies to determine the level of resistance to the imidazolinone herbicides and the patterns of cross-resistance of the R biotype to other herbicides at the whole plant level. In one study, 0, 9, 18, 35, 70, 140, 280, and 560 g/ha imazaquin and 0, 4, 9, 17, 35, 70, 140, and 280 g/ha nicosulfuron were applied POST to

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[6] Peters 20-20-20 general purpose fertilizer, The Wetzel Seed Co. Inc., 1345 Diamond Springs Road, Virginia Beach, VA 23455

the regression curves were not different.

RESULTS AND DISCUSSION

Field studies. Moisture stress at Marion reduced activity of PRE treatments, but timely rains produced active growth of smooth pigweed prior to the application of POST treatments. Moisture conditions at Painter were good, and activity from both PRE and POST applications was high. Imazaquin (PRE or POST) and imazethapyr (POST) controlled the S population but did not control the R population (Table 1). POST applications of imazaquin and imazethapyr initially injured R smooth pigweed 26 and 14%, respectively, at 7 DAPT (data not presented). Initial injury included general chlorosis in the apical meristem region and growth inhibition. The R population recovered from initial injury by 20 DAPT (Table 1), and neither imazaquin nor imazethapyr reduced R smooth pigweed densities (Table 2). Since densities were not reduced by these compounds the R population is likely homogeneous resistant, and has been developing for several years.

The sulfonylurea herbicides and pyrithiobac had comparable activity on both R and S smooth pigweed (Table 1). Control of both populations by ASC-67040, nicosulfuron, and thifensulfuron was nearly complete. Control of both populations by CGA-152005, chlorimuron, halosulfuron, and primisulfuron was lower (63 to 91%) except CGA-152005 gave 96% control of the S population in study two. Smooth pigweed densities in study two at Painter were low and variable, which resulted in higher control estimates for some

78% and was generally below 50%. Both pendimethalin and metribuzin reduced R and S smooth pigweed densities below the untreated control (Table 2), but did not adequately control either population.

Control of R smooth pigweed by POST non-ALS-inhibitor herbicides was also generally comparable to control of S smooth pigweed. Control of R and S smooth pigweed by lactofen was 81 and 85%, respectively in study one at both sites, but control of S smooth pigweed was 95% while that of the R population was 79% in study two. High control in study two at Painter may be due to variable population densities within this study. Plant densities in both populations were reduced by lactofen, but control was not complete except in study two in Painter. However, a higher rate of lactofen may be adequate for controlling both populations of smooth pigweed. The addition of imazethapyr to lactofen improved control of the S population but not the R population, further indicating the lack of activity of imazethapyr on the R population. Control of both populations from flumiclorac was below 75% except in study two at Painter, which is likely due to the variable population densities within this study. Smooth pigweed densities were relatively high in plots treated with flumiclorac in all other studies suggesting that control of S smooth pigweed in study two was higher than would normally be expected due to variable population densities within that study.

In field studies, the R population was tolerant to registered rates of the imidazolinone herbicides. The R and S populations were controlled equally well by other herbicides. None of the non-ALS-inhibitor herbicides adequately controlled R or S smooth pigweed

at the rates tested.

Greenhouse studies. Greenhouse studies support the results from the field and confirm that the R biotype is resistant to imazaquin and imazethapyr. Injury to R smooth pigweed from 280 and 560 g/ha imazaquin and imazethapyr, respectively was as high as 20% at 11 DAT (data not presented). R smooth pigweed recovered from initial injury, and control and dry weights were not affected 28 to 30 DAT by as much as 560 g/ha imazaquin or imazethapyr (Table 3). The response of R smooth pigweed to imazaquin and imazethapyr is similar to the response of resistant biotypes of common waterhemp and Palmer amaranth to imazethapyr (10). S smooth pigweed control was 79% from 70 g/ha imazaquin and 97% from 70 g/ha imazethapyr, and dry weights were reduced by 9 and 4 g/ha imazaquin and imazethapyr, respectively. Although the S biotype was more susceptible to imazethapyr than to imazaquin, it was controlled by both herbicides.

Smooth pigweed biotypes responded differently to various sulfonylurea herbicides. At all rates of chlorimuron or rimsulfuron, control of S smooth pigweed was higher and dry weights were lower than control and dry weights of R smooth pigweed treated with these herbicides (Table 4). Chlorimuron at 18 g/ha gave 83% control of R smooth pigweed, and at 9 g/ha gave 87% control of S smooth pigweed. With rimsulfuron, R smooth pigweed control was 81% by 135 g/ha and S smooth pigweed control was 83% by 17 g/ha. The R biotype is cross-resistant at low levels to chlorimuron and rimsulfuron. Low levels of cross-resistance to imidazolinone herbicides has been observed in sulfonylurea-resistant weed biotypes (4, 12, 15, 18).

The R and S biotypes generally responded similarly to other sulfonylurea herbicides in the greenhouse. Nicosulfuron did not completely control either biotype, but dry weights of both biotypes were reduced approximately 75% by 17 g/ha (Table 5). Both biotypes responded similarly to most rates of thifensulfuron except control of the R biotype was higher and dry weights of the R biotype were lower from 0.6 g/ha thifensulfuron than control and dry weights of the S biotype. At 0.6 g/ha, the R biotype was more sensitive to thifensulfuron than the S biotype. Pyrithiobac PRE controlled approximately 90% of both biotypes at rates as low as 36 g/ha (Table 6). Thus, the R biotype was not cross-resistant to proposed use rates of this pyrimidinyl thiobenzoate herbicide. The difference in response of the biotypes to pyrithiobac observed at site one in the field studies (Table 1) is likely due to variability or rainfall.

Both biotypes responded similarly to pendimethalin except control of the R biotype was lower than that of the S biotype at 0.5 ppmw (Table 6). Differences in dry weights did not occur at this rate, but plants may have been too small at harvest to detect differences in dry matter accumulation. Pendimethalin did not completely control either biotype at rates as high as 2.0 ppmw. These observations confirm that pendimethalin does not have sufficient activity on smooth pigweed to provide complete control of either biotype.

A GR_{50} value could not be calculated for imazaquin or imazethapyr on the R biotype due to the lack of response to rates as high as 560 g/ha (Table 7). The GR_{50} values on the S biotype were 22 and 3 g/ha for imazaquin and imazethapyr, respectively which correlated to greater than 25- and 186-fold levels of resistance to imazaquin and imazethapyr,

respectively, at the whole plant level. Low levels of cross-resistance to rimsulfuron and chlorimuron were also observed with R smooth pigweed GR_{50} values 7- and 2-fold higher for rimsulfuron and chlorimuron, respectively, than the S smooth pigweed GR_{50} values. There were no differences in the regression curves for nicosulfuron and thifensulfuron indicating that the two biotypes responded similarly to these herbicides. The low level of cross-resistance to chlorimuron in the greenhouse was not observed in the field since control of both populations was similar in the field studies (Table 1). Rimsulfuron was not included in the field studies, but the cross-resistance of R smooth pigweed to rimsulfuron observed in the greenhouse could correlate to lower activity in the field.

In two of five greenhouse studies, the dry matter accumulation for the untreated controls of the two biotypes did not differ. In the other three studies, differences were significant at the 0.05 level with the S biotype accumulating more dry matter. In this non-competitive environment, these data suggest that the biotypes are equally well fit with regard to biomass accumulation, but more research needs to be conducted in this area.

The R smooth pigweed from Marion, MD is resistant to imazaquin and imazethapyr. An insensitive ALS enzyme has been the mechanism of resistance, when the mechanism has been determined, in all of the ALS-inhibitor-resistant weed populations that have been selected with ALS-inhibitors (18). It is likely that an insensitive ALS enzyme is the mechanism of resistance in this smooth pigweed biotype. However, there is a population of rigid ryegrass, that was selected with diclofop, that is resistant to diclofop and cross-resistant to several sulfonylurea and imidazolinone herbicides (4). The ALS-inhibitor-

resistance mechanism in this rigid ryegrass population was determined to be increased metabolism of chlorsulfuron in the resistant biotype.

This population of smooth pigweed developed during five consecutive annual applications of imazaquin in spite of the fact that imazaquin was applied in combinations with pendimethalin or trifluralin, both of which are reported to have good activity on smooth pigweed. In these studies, pendimethalin did not adequately control either biotype of smooth pigweed, and in this case did not prevent the development of an herbicide-resistant population. This case supports the findings of Wrubel and Gressel that present herbicide mixtures do not meet all of their criteria for resistance management and are not effective in preventing the development of resistance (28).

This is the first reported smooth pigweed biotype with resistance to ALS-inhibitor herbicides (13), and it adds to a growing list of weed species with biotypes resistant to the ALS-inhibitor herbicides. The continued introduction of more ALS-inhibitor herbicides could intensify the development of resistant biotypes.

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Table 1. Control of smooth pigweed from PRE and POST applications of ALS-inhibitor and non-ALS-inhibitor herbicides in adjacent field studies in Marion, MD [imidazolinone-resistant (R)] and Painter, VA [imidazolinone-susceptible (S)] in 1994.[a]

			Resistant		Susceptible	
Herbicide	Rate	Timing	I[b]	II	I	II
	g/ha		% control[c]			
Imazaquin	140	PRE	0 h	0 f	99 a	99 a
Imazaquin	140	POST[d]	5 h	3 f	99 a	99 a
Imazethapyr	70	POST	0 h	0 f	98 ab	99 a
ASC-67040	49	POST	98 a	99 a	99 a	99 a
CGA-152005	20	POST	89 b	91 bc	89 bc	96 ab
Chlorimuron	9	POST	88 bc	83 cd	76 cde	90 bc
Halosulfuron	35	POST	75 cde	72 d	63 ef	84 cd
Nicosulfuron	35	POST	99 a	98 ab	97 ab	99 a
Primisulfuron	40	POST	83 bcd	92 bc	78 cde	91 bc
Pyrithiobac	70	POST	99 a	99 a	86 cd	96 ab
Thifensulfuron	4	POST	98 a	99 a	91 abc	99 a
Imazethapyr + lactofen	70 + 70	POST	70 def	70 d	99 a	99 a
Pendimethalin	1120	PRE	49 g	48 e	34 g	78 d
Metribuzin	280	PRE	54 fg	48 e	73 def	83 cd
Flumiclorac	29	POST	65 ef	72 d	58 f	84 cd
Lactofen	70	POST	81 bcd	79 d	85 cd	95 ab
Untreated control	—	—	0 h	0 f	0 h	0 e

[a] Means followed by the same letter do not differ at the 0.05 significance level by Duncan's multiple range test.

[b] I and II refer to studies one and two, respectively.

[c] Visual estimates of percent control were made 20 and 32 days after POST treatments at Marion and Painter, respectively. PRE treatments were applied 24 days prior to the POST treatments.

[d] All POST treatments included a non-ionic surfactant at 0.25% v/v, and treatments with imazethapyr also included 30% liquid nitrogen at 1.0% v/v.

Table 2. Smooth pigweed populations following PRE and POST applications of ALS-inhibitor and non-ALS-inhibitor herbicides in adjacent field studies in Marion, MD [imidazolinone-resistant (R)] and Painter, VA [imidazolinone-susceptible (S)] in 1994.[a]

			Resistant		Susceptible	
Herbicide	Rate	Timing	I[b]	II	I	II
	g/ha		plants/m^2[c]			
Imazaquin	140	PRE	42 ab	29 a	0 d	0 b
Imazaquin	140	POST[d]	41 ab	26 ab	0 d	0 b
Imazethapyr	70	POST	53 a	21 abc	0 d	0 b
ASC-67040	49	POST	0 d	0 d	0 d	0 b
CGA-152005	20	POST	4 d	0 d	15 d	1 b
Chlorimuron	9	POST	3 d	7 cd	102 cd	17 b
Halosulfuron	35	POST	25 abcd	10 bcd	264 ab	67 ab
Nicosulfuron	35	POST	0 d	0 d	0 d	0 b
Primisulfuron	40	POST	7 d	1 d	106 cd	8 b
Pyrithiobac	70	POST	0 d	0 d	68 cd	0 b
Thifensulfuron	4	POST	0 d	0 d	16 d	0 b
Imazethapyr + lactofen	70 + 70	POST	10 cd	9 bcd	0 d	0 b
Pendimethalin	1120	PRE	8 d	7 cd	91 cd	10 b
Metribuzin	280	PRE	11 bcd	6 cd	17 d	4 b
Flumiclorac	29	POST	17 bcd	19 abc	171 bc	12 b
Lactofen	70	POST	1 d	5 cd	14 d	0 b
Untreated control	—	—	50 a	29 a	293 a	102 a

[a] Means followed by the same letter do not differ at the 0.05 significance level by Duncan's multiple range test.

[b] I and II refer to studies one and two, respectively.

[c] Population counts were taken 20 and 32 days after POST treatments in Marion and Painter, respectively. PRE treatments were applied 24 days prior to the POST treatments.

[d] All POST treatments included a non-ionic surfactant at 0.25% v/v, and treatments with imazethapyr also included 30% liquid nitrogen at 1.0% v/v.

Table 3. Percent control and dry weights of imidazolinone-resistant and -susceptible biotypes of smooth pigweed treated with imazaquin and imazethapyr in the greenhouse[a].

		Control		Dry weight	
Herbicide	Rate	Resistant	Susceptible	Resistant	Susceptible
	g/ha	%		g	
Imazaquin[b]	0	0	0	2.1	2.6
	9	3	27	2.1	1.8
	18	3	44	2.4	1.5
	35	0	62	2.0	1.1
	70	1	79	2.1	0.7
	140	6	84	1.9	0.7
	280	10	89	1.7	0.7
	560	12	93	1.8	0.6
LSD (0.05) (rate)[c]		NS	16	NS	0.8
LSD (0.05) (biotype)		10		0.5	
Imazethapyr[d]	0	0	0	3.7	3.8
	4	0	33	3.1	2.1
	9	0	70	4.0	1.0
	18	0	94	3.3	0.3
	35	0	96	3.8	0.5
	70	0	97	3.6	0.3
	140	0	99	3.5	0.4
	280	0	99	3.6	0.3
	560	9	99	2.9	0.2
LSD (0.05) (rate)		NS	10	0.9	0.9
LSD (0.05) (biotype)		6		0.6	

[a] All treatments included a non-ionic surfactant at 0.25% v/v.

[b] Visual estimates of percent control were made 29 DAT and plants were harvested 30 DAT for imazaquin.

[c] LSD values for rate indicate differences between rates within a biotype, and LSD values for biotype indicate differences between biotypes within a rate.

[d] Visual estimates of percent control were made and plants were harvested 28 DAT for imazethapyr.

Table 4. Percent control and dry weights of imidazolinone-resistant and -susceptible biotypes of smooth pigweed treated with rimsulfuron and chlorimuron in the greenhouse[a,b].

		Control		Dry weight	
Herbicide	Rate	Resistant	Susceptible	Resistant	Susceptible
	g/ha	%		g	
Rimsulfuron	0	0	0	4.1	3.8
	1	8	31	3.2	2.8
	2	14	43	3.0	2.1
	4	19	65	2.6	1.1
	8	31	69	2.4	1.1
	17	45	83	1.9	0.8
	34	62	89	1.6	0.4
	68	73	94	1.2	0.3
	135	81	97	1.1	0.2
LSD (0.05) (rate)[c]		16	16	0.9	0.9
LSD (0.05) (biotype)		10		0.6	
Chlorimuron	0	0	0	4.1	3.8
	0.6	10	26	3.4	2.5
	1.1	20	41	3.2	2.3
	2.2	35	61	2.7	1.8
	4.5	38	64	2.2	1.4
	9	56	87	1.6	0.5
	18	83	94	0.9	0.3
LSD (0.05) (rate)		16	16	0.9	0.9
LSD (0.05) (biotype)		11		0.6	

[a] All treatments included a non-ionic surfactant at 0.25% v/v.

[b] Visual estimates of percent control were made and plants were harvested 31 DAT for rimsulfuron and chlorimuron.

[c] LSD values for rate indicate differences between rates within a biotype, and LSD values for biotype indicate differences between biotypes within a rate.

Table 5. Percent control and dry weights of imidazolinone-resistant and -susceptible biotypes of smooth pigweed treated with nicosulfuron and thifensulfuron in the greenhouse[a].

		Control		Dry weight	
Herbicide	Rate	Resistant	Susceptible	Resistant	Susceptible
	g/ha	%		g	
Nicosulfuron[b]	0	0	0	2.0	2.6
	4	25	30	1.6	1.3
	9	27	28	1.3	1.6
	17	79	78	0.5	0.6
	35	83	82	0.5	0.5
	70	92	84	0.4	0.5
	140	92	86	0.4	0.7
	280	93	88	0.4	0.6
LSD (0.05) (rate)[c]		13	13	0.6	0.6
LSD (0.05) (biotype)		8		0.4	
Thifensulfuron[d]	0	0	0	3.7	3.8
	0.1	43	33	1.7	2.2
	0.3	42	43	1.6	1.8
	0.6	78	44	1.0	1.6
	1.1	94	87	0.5	0.6
	2.2	96	86	0.6	0.6
	4.5	97	96	0.3	0.4
LSD (0.05) (rate)		18	18	0.8	0.8
LSD (0.05) (biotype)		12		0.6	

[a] All treatments included a non-ionic surfactant at 0.25% v/v.

[b] Visual estimates of percent control were made 29 DAT and plants were harvested 30 DAT for nicosulfuron.

[c] LSD values for rate indicate differences between rates within a biotype, and LSD values for biotype indicate differences between biotypes within a rate.

[c] Visual estimates of percent control were made and plants were harvested 28 DAT for thifensulfuron.

Table 6. Percent control and dry weights of imidazolinone-resistant and -susceptible biotypes of smooth pigweed treated with pyrithiobac (PRE) and pendimethalin (PPI) in the greenhouse.

		Control		Dry weight	
Herbicide	Rate	Resistant	Susceptible	Resistant	Susceptible
	g/ha	%		g	
Pyrithiobac[a]	0	0	0	1.22	1.43
	36	93	89	0	0.03
	70	96	92	0	0
	140	96	94	0	0
LSD (0.05) (rate)[b]		8	8	0.18	0.18
LSD (0.05) (biotype)		5		0.12	
Pendimethalin	ppmw				
	0	0	0	1.0	1.3
	0.25	12	21	0.9	0.9
	0.5	54	73	0.2	0.1
	1.0	67	71	0.2	0.2
	2.0	88	89	0.1	0.1
LSD (0.05) (rate)		18	18	0.4	0.4
LSD (0.05) (biotype)		12		0.3	

[a] Visual estimates of percent control were made and plants were harvested 28 DAT for pyrithiobac and pendimethalin.

[b] LSD values for rate indicate differences between rates within a biotype, and LSD values for biotype indicate differences between biotypes within a rate.

Table 7. GR_{50} values for several imidazolinone and sulfonylurea herbicides on imidazolinone-resistant (R) and -susceptible (S) biotypes of smooth pigweed as determined by dry weights in greenhouse studies.

	GR_{50} values[a]		
Herbicide	Resistant	Susceptible	Ratio (R/S)[b]
	g/ha		
Imazaquin	>560	22	>25 +
Imazethapyr	>560	3	>186 +
Chlorimuron	4	2	2 +
Nicosulfuron	10	9	1
Rimsulfuron	13	2	7 +
Thifensulfuron	0.3	0.3	1

[a] GR_{50} values were determined by regressing dry weights, as a percent of the untreated control, on the logarithm of herbicide rate. Plants were harvested 28 to 31 DAT to determine dry weights.

[b] + = regression curves differ significantly, indicating a significant difference in the response of the two biotypes using 95% confidence limits.

Chapter VI

Imidazolinone-Resistance in Smooth Pigweed (*Amaranthus hybridus*) is Due to an Altered Acetolactate Synthase

Abstract. Seeds were collected from an imidazolinone-resistant (R) population of smooth pigweed in Marion, MD and from an imidazolinone-susceptible (S) population in Painter, VA, and grown in the greenhouse. Acetolactate synthase (ALS) enzyme was extracted from both biotypes and assayed in the presence of imazaquin, imazethapyr, CGA-152005, chlorimuron, halosulfuron, nicosulfuron, primisulfuron, pyrithiobac, rimsulfuron, and thifensulfuron to determine if an altered ALS was the mechanism of resistance in the R biotype and to determine if this biotype was cross-resistant to other ALS-inhibitor herbicides. The inhibitor concentration required to cause a 50% reduction in ALS activity (I_{50}) was calculated for each herbicide. ALS from the R biotype was 83-, 397-, and 12-fold more resistant to imazaquin, imazethapyr, and rimsulfuron, respectively than that from the S biotype. ALS from the R biotype was 4- and 3-fold less resistant to pyrithiobac and thifensulfuron, respectively, than that from the S biotype. ALS from both biotypes responded similarly to CGA-152005, chlorimuron, halosulfuron, nicosulfuron, and primisulfuron. Resistance in the R biotype was not due to overexpression of ALS but to an altered form of ALS that is insensitive to the imidazolinone herbicides and rimsulfuron. **Nomenclature:** CGA-152005, 1-(4-methoxy-6-methyl-triazin-2-yl)-3-[2-(3,3,3-trifluoropropyl)-phenylsulfonyl]-urea; chlorimuron, 2-[[[[(4-chloro-6-methoxy-2-

pyrimidinyl)amino]carbonyl]amino]sulfonyl]benzoic acid; halosulfuron, methyl 3-chloro-5-(4,6-dimethoxypyrimidin-2-ylcarbamoylsulfamoyl)-1-methylpyrazole-4-carboxylate; imazaquin, 2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-3-quinolinecarboxylic acid; imazethapyr, 2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-5-ethyl-3-pyridinecarboxylic acid; nicosulfuron, 2-[[[[(4,6-dimethoxy-2-pyrimidinyl)amino]carbonyl]amino]sulfonyl]-*N,N*-dimethyl-3-pyridinecarboxamide; primisulfuron, 2-[[[[[4,6-bis(difluoromethoxy)-2-pyrimidinyl]amino]carbonyl]amino]sulfonyl]benzoic acid; pyrithiobac, 2-chloro-6-[(4,6-dimethoxy-2-pyrimidinyl)thio]benzoic acid; rimsulfuron, *N*-[[(4,6-dimethoxy-2-pyrimidinyl)amino]carbonyl]-3-(ethylsulfonyl)-2-pyridinesulfonamide; thifensulfuron, 3-[[[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl)amino]carbonyl]amino]sulfonyl]-2-thiophenecarboxylic acid; smooth pigweed, *Amaranthus hybridus* L. #[1] AMACH.

Additional Index Words: ALS-inhibitor resistance, herbicide resistance, imidazolinone resistance, sulfonylurea herbicides, target-site resistance, CGA-152005, chlorimuron, halosulfuron, imazaquin, imazethapyr, nicosulfuron, primisulfuron, pyrithiobac, rimsulfuron, thifensulfuron, AMACH.

INTRODUCTION

[1] Letters following this symbol are a WSSA-approved computer code from Composite List of Weeds, Revised 1989. Available from WSSA, 1508 West University Ave., Champaign, IL 61821-3133.

Weed control is increasingly difficult due, in part, to the development of herbicide-resistant biotypes that cannot be controlled by herbicides that were once effective (8, 9, 12, 16, 17, 19, 21, 29). Weed biotypes have been identified resistant to one or more of 14 herbicides or herbicide families, including the recent identification of biotypes resistant to the sulfonylurea and imidazolinone herbicides (8, 9, 10, 13, 19). Sulfonylurea and imidazolinone herbicides inhibit ALS (EC 4.1.3.18), the first common enzyme in the biosynthesis of the branched chain amino acids - valine, leucine, and isoleucine (7, 18, 19, 22, 23, 24, 27). Additionally, the triazolopyrimidine, and pyrimidinyl thiobenzoate herbicides inhibit ALS (5, 19, 28).

Differential selectivity from sulfonylurea and imidazolinone herbicides is usually due to enhanced metabolism in the tolerant species relative to the susceptible species (2, 14, 15, 19, 25, 30, 31). This is the mechanism of resistance to ALS-inhibitors in an acetyl coenzyme A carboxylase-inhibitor-resistant biotype of rigid ryegrass (*Lolium rigidum* Gaudin) (3, 19). However, all of the other ALS-inhibitor-resistant weed biotypes, in which the resistance mechanism has been determined, are resistant because of an altered ALS that is insensitive to the herbicides (3, 4, 19, 20, 21, 28, 29). The ALS-inhibitor-resistant weed biotypes documented to date are often cross-resistant at varying levels to members of the same herbicide family as the selection agent, but display varying patterns of cross-resistance to members of other ALS-inhibitor herbicide families (4, 6, 10, 12, 13, 16, 17, 19, 21).

A biotype of smooth pigweed identified near Marion, MD as resistant to the

imidazolinone herbicides, imazaquin and imazethapyr, was reported previously (13). This biotype was greater than 25- and 186-fold more resistant to imazaquin and imazethapyr, respectively, than a S biotype. The R biotype displayed 7- and 2-fold levels of cross-resistance to rimsulfuron and chlorimuron, respectively, compared to the S biotype in the greenhouse. The R biotype did not appear to be cross-resistant to other sulfonylurea herbicides or pyrithiobac in field and greenhouse studies.

The objectives of this research were to determine if the mechanism of resistance in the R biotype of smooth pigweed is an altered ALS, as in most other weed biotypes resistant to ALS-inhibitor herbicides, and to determine if ALS from the R biotype was sensitive to other ALS-inhibitors.

MATERIALS AND METHODS

Plant material. Smooth pigweed seeds were collected in the fall of 1993 from several plants in a field in Marion, and from several plants on the Virginia Polytechnic Institute and State University Eastern Shore Agricultural Research and Extension Center in Painter, VA. The population at Marion (R) had received applications of imazaquin alone or in combination with pendimethalin [*N*-(1-ethylpropyl)-3,4-dimethyl-2,6-dinitrobenzenamine] or trifluralin [2,6-dinitro-*N,N*-dipropyl-4-(trifluoromethyl)benzenamine] each year since 1986. The population at Painter (S) had little or no history of ALS-inhibitor herbicide use, and was shown to be susceptible to numerous sulfonylurea and imidazolinone herbicides

(chapters IV and V). Seeds were collected by shaking seeds from randomly selected, mature plants at both locations.

ALS enzyme assays. Acetolactate synthase enzyme assays were conducted using the method of Singh et al. (26) with slight modifications. Flats (6.9 L) were filled with greenhouse soil mix consisting of State sandy loam soil (Typic Hapludults), sand, and peat (23:13:1 by wt). Smooth pigweed seeds from the R or S population were sprinkled on the soil surface and covered with 0.6 cm of greenhouse soil mix. The seeds were germinated and the plants grown under natural sunlight in the greenhouse with a photoperiod of at least 14 h and were watered as needed. A complete fertilizer[2] was applied weekly to maintain active growth. The apical meristem region, including the youngest unexpanded leaves, from 5- to 10-cm R and S smooth pigweed seedlings were harvested for ALS enzyme extraction. The plant tissue (10 g) was powdered in liquid nitrogen and homogenized in 100 mM potassium phosphate buffer (pH 7.5) containing 200 mM sodium pyruvate, 20 mM $MgCl_2$, 2 mM thiamine pyrophosphate, and 20 μM flavin adenine dinucleotide. The homogenate was filtered through a nylon cloth (53 μm mesh) and centrifuged at 15,000 *g* for 15 min. The supernatant was desalted on a 10 ml desalting column[3] before being used for assay procedures.

Standard reaction mixtures contained ALS in 50 mM potassium phosphate buffer (pH

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[2] Peters 20-20-20 general purpose fertilizer, The Wetzel Seed Co. Inc., 1345 Diamond Springs Road, Virginia Beach, VA 23455

[3] Econpack 10 DG™ desalting columns, Bio Rad Laboratories, Hercules, CA 94547.

7.0) containing 100 mM sodium pyruvate, 10 mM $MgCl_2$, 1 mM thiamine pyrophosphate, and 10 μM flavin adenine dinucleotide, and ALS-inhibitors at various concentrations. High concentrations of the combination of valine and leucine inhibits ALS enzyme activity, which in effect, prevents overproduction of these branched chain amino acids (26). A combination of valine and leucine was included at 0.008, 0.016, 0.031, 0.063, 0.125, 0.25, 0.5, and 1.0 mM as a standard. Imazaquin and imazethapyr were assayed at 0.78, 1.56, 3.13, 6.25, 12.5, 25, 50, and 100 μM. CGA-152005, chlorimuron, halosulfuron, primisulfuron, and pyrithiobac were assayed at 0.78, 1.56, 3.13, 6.25, 12.5, 25, 50, and 100 nM. Thifensulfuron was assayed at 0.008, 0.016, 0.031, 0.063, 0.125, 0.25, 0.5, and 1.0 nM. Rimsulfuron was assayed at 0.008, 0.016, 0.031, 0.063, 0.125, 0.25, 0.5, and 1.0 μM; and nicosulfuron was assayed at 0.08, 0.16, 0.31, 0.63, 1.25, 2.5, 5.0, and 10.0 μM.

The reaction mixtures were incubated at 37 C for two h, after which H_2SO_4 was added (final concentration was 1%) to stop the reaction and to decarboxylate the acetolactate formed in the reaction to acetoin. Direct acetoin formation was determined by the addition of 4 N NaOH instead of H_2SO_4 to a control with no inhibitor. The decarboxylation process was conducted for 15 min at 60 C. Creatine (0.17% final concentration) and 1-napthol (1.7% in 4 N NaOH final concentration) were added to the reaction mixtures which were then incubated for 15 min at 60 C and 15 min at room temperature. The absorbance of the resulting color complex was measured at 520 nm to determine the amount of acetoin formation. Protein concentrations were determined by the method of Bradford (1).

The ALS from the R biotype was resistant to high concentrations of both imazaquin and imazethapyr, whereas ALS activity from the S biotype was inhibited approximately 80% with as little as 10 to 15 μM of both herbicides (Figure 2). The R ALS was 83- and 397-fold more resistant to imazaquin and imazethapyr, respectively, than S ALS based on the I_{50} values for the two herbicides (Table 1). These results correspond well with the response observed at the whole plant level (previous chapter). Interestingly, based on enzyme assays and symptoms observed at the whole plant level the R biotype is more resistant to imazethapyr than to the selection agent, imazaquin. This response is likely due to a higher binding affinity for imazethapyr on S ALS compared with imazaquin.

Resistance to rimsulfuron at the whole plant level (previous chapter) was confirmed at the target site level since activity of S ALS was inhibited more than that of R ALS at lower rimsulfuron concentrations (Figure 3). Concentrations above 0.4 μM rimsulfuron inhibited enzyme activity from both biotypes similarly. The I_{50} of R ALS for rimsulfuron is 12-fold higher than that of S ALS (Table 1). The low level of resistance observed at the whole plant level to chlorimuron (previous chapter) was not confirmed at the target site level since ALS from both biotypes responded similarly to this herbicide (Figure 3). These results may suggest that the low level of cross-resistance to chlorimuron at the whole plant level in the greenhouse was due to differential uptake, translocation, or metabolism of chlorimuron in the two biotypes.

The ALS from the R biotype was more sensitive to thifensulfuron and pyrithiobac than ALS from the S biotype at concentrations below 0.4 and 40 nM, respectively, indicating

low levels of negative cross-resistance to these compounds at the target site level (Figure 4). The I_{50} values of the R biotype were also three- and four-fold lower than those of the S biotype for thifensulfuron and pyrithiobac, respectively (Table 1). These results may explain the higher control of the R biotype compared to the S biotype from 0.6 g/ha thifensulfuron in greenhouse studies (previous chapter). The negative cross-resistance to pyrithiobac at the target site level was not confirmed at the whole plant level, since all rates controlled both biotypes completely in the greenhouse (previous chapter). ALS from the R and S biotypes responded similarly to nicosulfuron and halosulfuron (Figure 5), and to primisulfuron and CGA-152005 (Figure 6). The I_{50} values from these herbicides were similar for both biotypes (Table 1). These data and the results from field and greenhouse studies indicate that these herbicides are equally effective for control of both biotypes.

Resistance in the R biotype of smooth pigweed was not affected by an overexpression of ALS since the specific activities of ALS from the R and S biotypes were similar (1.61 ± 0.10 and 1.56 ± 0.36 AU/mg protein/h for the R and S ALS, respectively).

Resistance to the imidazolinone herbicides and rimsulfuron in the R biotype is due to an altered target site (ALS) that is insensitive to these herbicides. The altered target site has been the mechanism of resistance, when the mechanism has been determined, in all of the ALS-inhibitor-resistant weed populations that have arisen under ALS-inhibitor selection pressure (3, 4, 6, 19, 20, 21). The selection agent for this biotype was also an ALS-inhibitor, imazaquin. The R biotype is not cross-resistant to other sulfonylurea herbicides, except chlorimuron at the whole plant level, or pyrithiobac. Varying patterns of cross-

resistance to other classes of ALS-inhibitors have been observed in other ALS-inhibitor-resistant weed biotypes (3, 4, 6, 19, 20, 21). Multiple gene mutations can affect ALS-inhibitor resistance (19). Some mutations confer resistance to one class of ALS-inhibitors, while other mutations confer resistance to two or more classes of ALS-inhibitors. Plants can also stack multiple resistance mutations which can confer resistance to many herbicides with different modes of action (19). Multiple resistance has occurred in biotypes of rigid ryegrass (3). Given our results and those of others, levels of resistance and patterns of cross-resistance should be determined separately for each population of ALS-inhibitor-resistant weed species.

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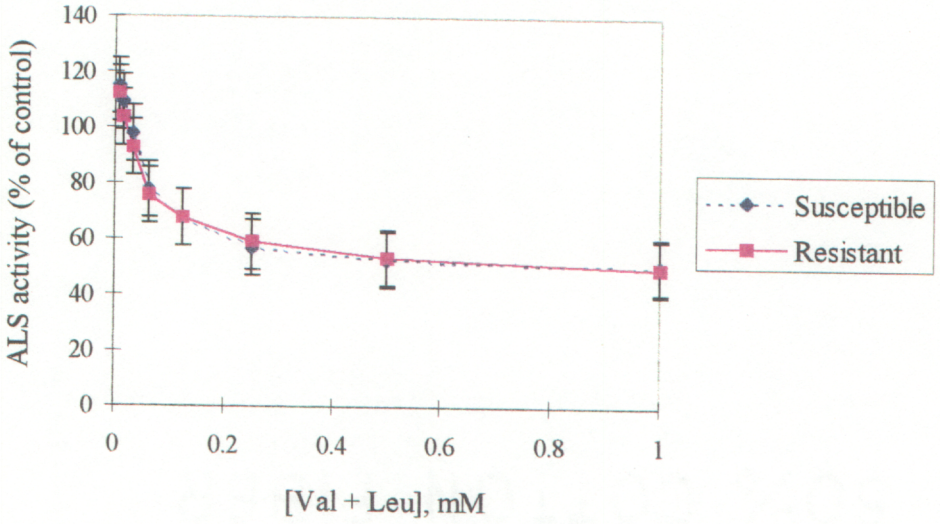
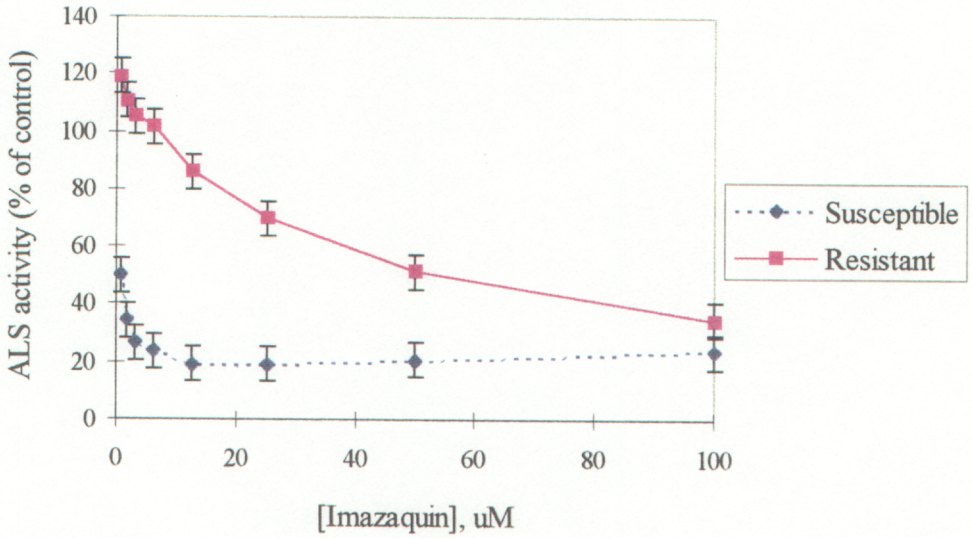


Figure 1. Inhibition of ALS activity isolated from imidazolinone-resistant and -susceptible smooth pigweed by a combination of valine and leucine. Data are means from two studies with one replication each and Fisher's LSD value (0.05 significance level) centered around them.



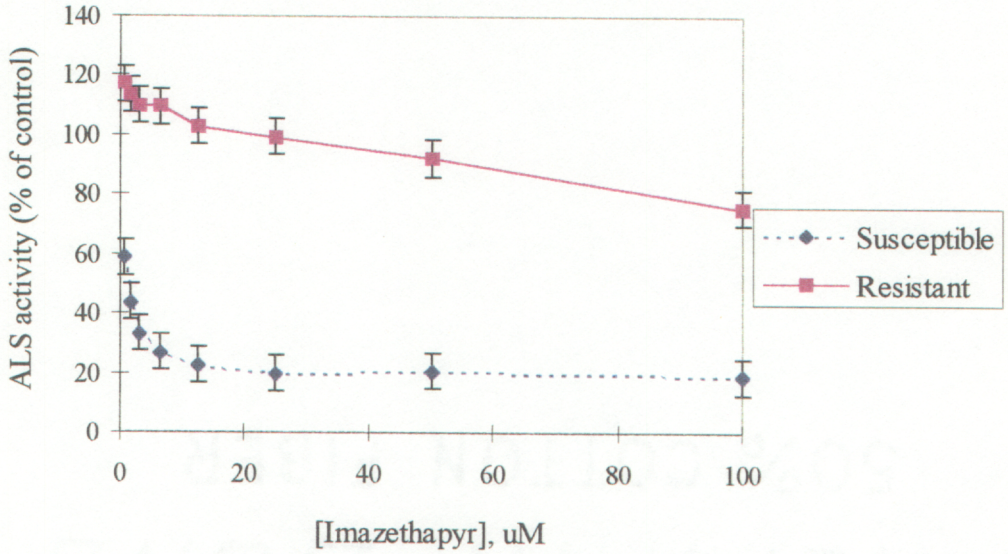
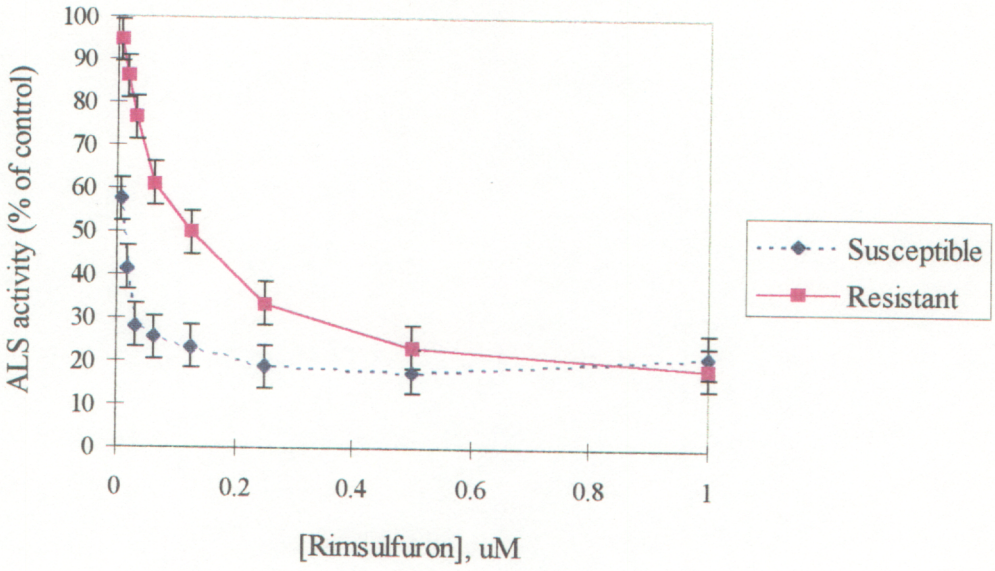


Figure 2. Inhibition of ALS activity isolated from imidazolinone-resistant and -susceptible smooth pigweed by imazaquin and imazethapyr. Data are means from two studies with two replications each and Fisher's LSD value (0.05 significance level) centered around them.



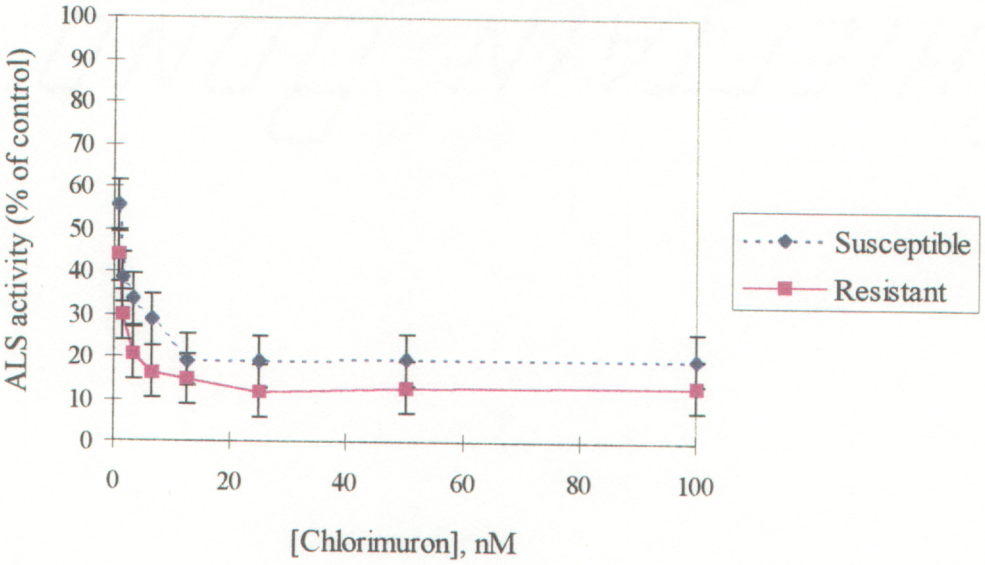


Figure 3. Inhibition of ALS activity isolated from imidazolinone-resistant and -susceptible smooth pigweed by rimsulfuron and chlorimuron. Data are means from two studies with two replications each and Fisher's LSD value (0.05 significance level) centered around them.

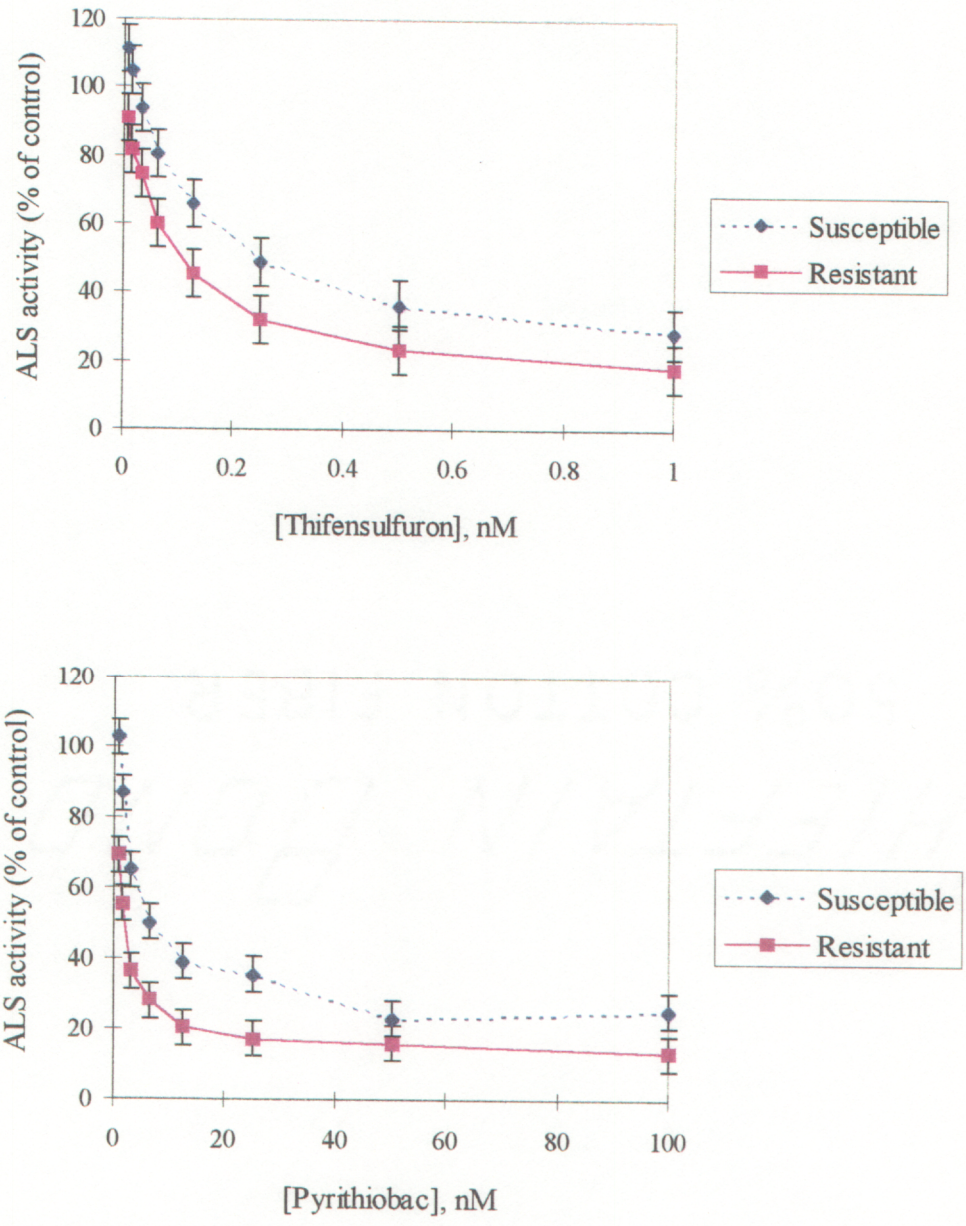


Figure 4. Inhibition of ALS activity isolated from imidazolinone-resistant and -susceptible smooth pigweed by thifensulfuron and pyrithiobac. Data are means from two studies and two replications each and with Fisher's LSD value (0.05 significance level) centered around them.

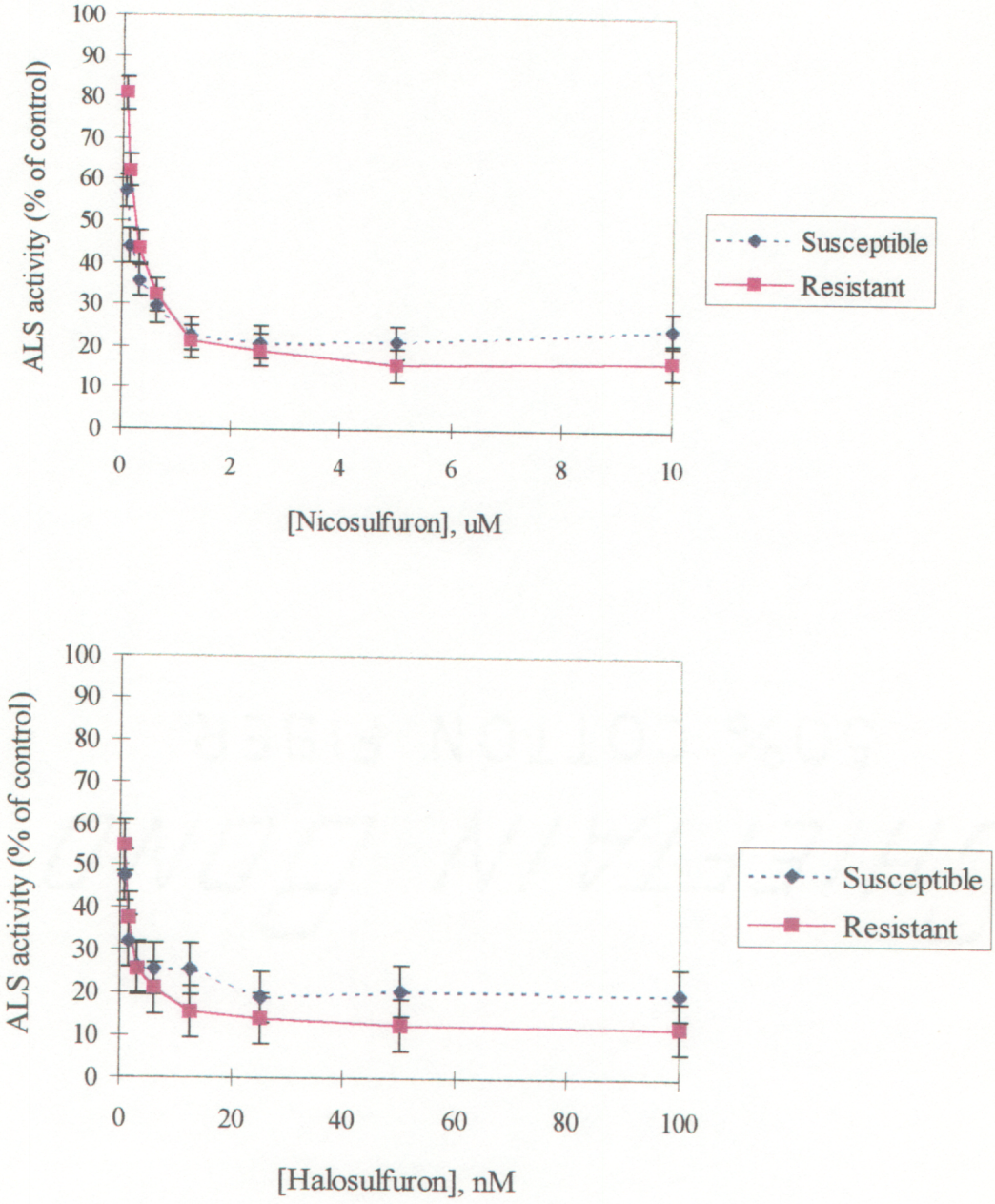
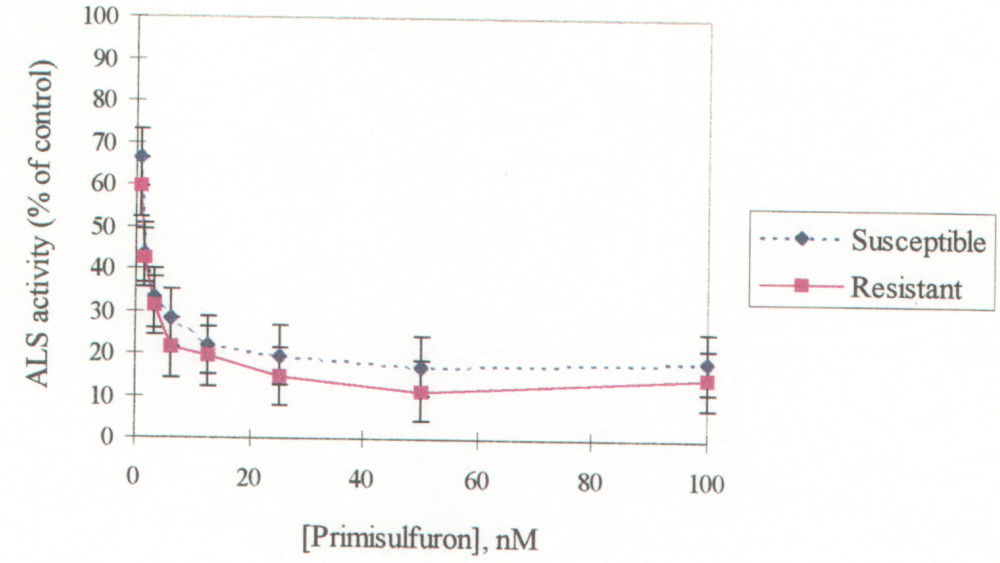


Figure 5. Inhibition of ALS activity isolated from imidazolinone-resistant and -susceptible smooth pigweed by nicosulfuron and halosulfuron. Data are means from two studies with two replications each and Fisher's LSD value (0.05 significance level) centered around them.



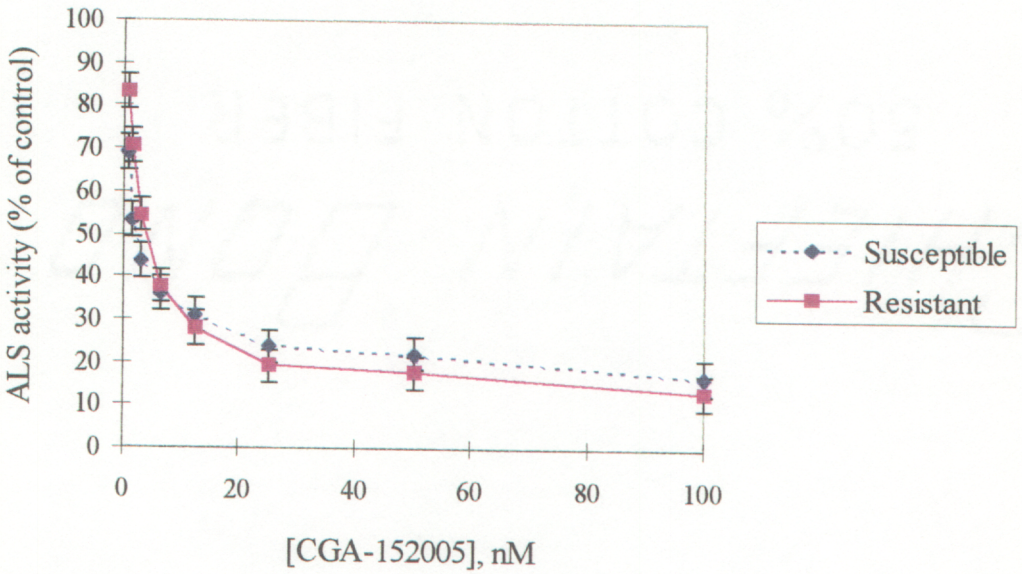


Figure 6. Inhibition of ALS activity isolated from imidazolinone-resistant and -susceptible smooth pigweed by primisulfuron and CGA-152005. Data are means from two studies with two replications each and Fisher's LSD value (0.05 significance level) centered around them.

Table 1. I_{50} values for various ALS inhibitor-herbicides on ALS isolated from imidazolinone-resistant (R) and -susceptible (S) biotypes of smooth pigweed.

	I_{50} values[a]		
Herbicide	Resistant	Susceptible	Ratio (R/S)
	nM		
Imazaquin	57410	690	83 +[b]
Imazethapyr	472750	1190	397 +
CGA-152005	3.4	2.3	1.5
Chlorimuron	0.5	1.0	0.5
Halosulfuron	0.9	0.5	1.8
Nicosulfuron	240	120	2
Primisulfuron	1.1	1.4	0.8
Pyrithiobac	1.8	7.0	0.26 ++[c]
Rimsulfuron	120	10	12 +
Thifensulfuron	0.1	0.3	0.33 ++
Valine + leucine	620000	600000	1

[a] I_{50} values were determined by regressing ALS-inhibition, expressed as a percent of the control, on the inhibitor concentration.

[b] + = regression curves differ significantly, indicating that R ALS is more tolerant than S ALS using 95% confidence limits.

[c] ++ = regression curves differ significantly, indicating that R ALS is less tolerant than S ALS using 95% confidence limits.

Chapter VII

Uptake, Translocation, and Metabolism of Chlorimuron and Nicosulfuron in Imidazolinone-Resistant and -Susceptible Smooth Pigweed (*Amaranthus hybridus* L.)

Abstract. A population of smooth pigweed on the Delmarva Peninsula was determined to be resistant to imazaquin and imazethapyr. This population exhibited low levels of cross-resistance to chlorimuron, but were not cross-resistant to nicosulfuron in greenhouse research. Previously conducted acetolactate synthase (ALS) enzyme assays showed no differences in the response of ALS from imidazolinone-resistant (R) and -susceptible (S) smooth pigweed to chlorimuron and nicosulfuron. The uptake, translocation, and metabolism of the ethyl ester of chlorimuron and nicosulfuron in R and S smooth pigweed was investigated. Five- to 10-cm R and S smooth pigweed seedlings were exposed to foliar-applied ^{14}C-labeled chlorimuron and nicosulfuron for 3, 6, 24, and 72 h. Chlorimuron uptake increased with time in both biotypes and was higher in the R biotype only at 6 h after application. Nicosulfuron uptake did not increase with time in the R biotype, and after 6 h, there were no differences between the R and S biotypes. Chlorimuron and nicosulfuron were translocated above and below the treated leaf, but not into the roots in both biotypes. Translocation of chlorimuron and nicosulfuron out of the treated leaf was also similar in both biotypes. The R biotype metabolized more chlorimuron at 3 h after application than the S biotype, but the S biotype metabolized

more chlorimuron at 24 and 72 h after application than the R biotype. The two-fold level of cross-resistance to chlorimuron at the whole plant level might be explained by more rapid metabolism in the R biotype. Metabolism of nicosulfuron decreased with time in the R and S biotypes and was not different between biotypes. **Nomenclature:** chlorimuron, ethyl 2-[[[[(4-chloro-6-methoxy-2-pyrimidinyl)amino]carbonyl]amino]sulfonyl]benzoic acid; imazaquin, 2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-3-quinolinecarboxylic acid; imazethapyr, 2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-5-ethyl-3-pyridinecarboxylic acid; nicosulfuron, 2-[[[[(4,6-dimethoxy-2-pyrimidinyl)amino]carbonyl]amino]sulfonyl]-*N,N*-dimethyl-3-pyridinecarboxamide; smooth pigweed, *Amaranthus hybridus* L. #[1] AMACH.

Additional index words. ALS-inhibitor resistance, herbicide resistance, herbicide tolerance, sulfonylurea herbicides, AMACH.

INTRODUCTION

The number of weed species with biotypes resistant to sulfonylurea and imidazolinone herbicides continues to increase, and these biotypes are often selected by the continuous use of a single control agent (7, 8, 9, 10, 13, 14, 15, 17, 24). The mode of action of these herbicides, as well as the triazolopyrimidine and pyrimidinyl thiobenzoate herbicides, is the

[1] Letters following this symbol are a WSSA-approved computer code from Composite List of Weeds, Revised 1989. Available from WSSA, 1508 West University Ave., Champaign, IL 61821-3133.

inhibition of ALS (EC 4.1.3.18) which is the first common enzyme in the biosynthesis of the branched chain amino acids valine, leucine, and isoleucine (6, 18, 19, 20, 22). Tolerance to the sulfonylurea and imidazolinone herbicides is usually due to enhanced metabolism in the tolerant plant species relative to the susceptible species; although differential uptake can contribute to this tolerance (2, 11, 12, 15, 21, 25, 26). Tolerant soybean seedlings metabolized chlorimuron with a half-life of only 1 to 3 h while two susceptible weed species, common cocklebur (*Xanthium strumarium* L.) and redroot pigweed (*Amaranthus retroflexus* L.), metabolized chlorimuron with half-lives of greater than 30 h (2). All of the ALS-inhibitor resistant weed populations, in which the resistance mechanism has been determined, are resistant because of an altered ALS that is insensitive to the ALS-inhibiting herbicides (3, 4, 15, 16, 17, 23, 24). An exception to this is a biotype of rigid ryegrass (*Lolium rigidum* Gaudin) which is resistant due to enhanced metabolism of the ALS-inhibitor herbicides (3).

Chlorimuron and nicosulfuron are sulfonylurea herbicides registered for use in soybeans [*Glycine max* (L.) Merr.] and corn (*Zea mays* L.), respectively. Chlorimuron controls many broadleaf weeds while nicosulfuron is most effective in controlling grass weeds, but both herbicides control smooth pigweed effectively (1, 5).

A biotype of smooth pigweed identified as resistant to the imidazolinone herbicides, imazaquin and imazethapyr, was reported previously (10). The mechanism of resistance in this biotype was also determined to be an altered ALS that is insensitive to the imidazolinone herbicides (Chapter 6). This biotype displayed a two-fold level of cross

resistance to chlorimuron at the whole plant level in greenhouse studies but no cross-resistance to nicosulfuron (10, chapter 5). However, ALS extracted from the resistant biotype was not cross-resistant to chlorimuron or nicosulfuron (Chapter 6).

The objective of this research was to determine if the cross-resistance to chlorimuron at the whole plant level was due to uptake, translocation, or metabolism.

MATERIALS AND METHODS

Radiolabeled chemicals. All studies were conducted using [pyrimidine-2-^{14}C] -chlorimuron and [pyrimidine-2-^{14}C] -nicosulfuron. The specific activities were 304 and 2301 kBq/mg for chlorimuron and nicosulfuron, respectively, and the radiochemical purities were 99 and 98.8% for chlorimuron and nicosulfuron, respectively.

Plant material. Smooth pigweed seeds were collected from several plants in a field in Marion, MD and from several plants at the Eastern Shore Agriculture Research and Extension Center in Painter, VA. The population at Marion (R)[2] had received applications of imazaquin alone or in combination with pendimethalin or trifluralin annually since 1986 in continuous soybeans. This biotype was confirmed resistant to imazaquin and imazethapyr, and cross-resistant to rimsulfuron {*N*-[[(4,6-dimethoxy-2-pyrimidinyl)amino]carbonyl]-3-(ethylsulfonyl)-2-pyridinesulfonamide} at the whole plant and enzyme levels (Chapters 5 and 6). It was also cross-resistant to chlorimuron at the whole plant level, but was susceptible to nicosulfuron (Chapter 5). The population at

Painter (S)[2] had little to no history of ALS-inhibitor herbicide exposure. It was shown to be susceptible to chlorimuron, imazaquin, imazethapyr, and nicosulfuron (Chapters 4 and 5); and was considered a representative wild type susceptible biotype.

Absorption and translocation studies. Smooth pigweed seeds (R and S) were planted in a commercial potting medium[3] in 470 ml styrofoam cups. The seeds were germinated and the plants grown under natural sunlight supplemented with metal halide lamps for a photoperiod of at least 14 h in the greenhouse, and were watered as needed. After germination, plants were thinned to one plant per cup. A complete fertilizer[4] was applied weekly to maintain active growth. Commercially formulated chlorimuron or nicosulfuron was applied POST to 10- to 15-cm tall plants at rates of 5 g ai/ha chlorimuron or 17 g ai/ha nicosulfuron. These rates are one-half the normal use rates of these herbicides (1). Commercially formulated herbicides were applied with a greenhouse bench sprayer calibrated to deliver 234 L/ha of water at 221 kPa pressure through a flat fan spray tip[5],

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[2] Abbreviations: HAA, hours after application; LSS, liquid scintillation spectrometry; R, resistant; Rf, ratio of front; S, susceptible; TLC, thin layer chromatography.

[3] Metro Mix 360, The Wetzel Seed Co. Inc., 1345 Diamond Springs Rd., Virginia Beach, VA 23455.

[4] Peters 20-20-20 general purpose fertilizer, The Wetzel Seed Co. Inc., 1345 Diamond Springs Rd., Virginia Beach, VA 23455.

[5] Teejet 8001E flat fan spray tip, Spraying Systems Co., North Ave., Wheaton, IL 60188.

and included a non-ionic surfactant[6] at 0.25% v/v. Application of the radiolabeled herbicides was done by the method described by Wilcut et al. (26). Immediately following application of the commercially formulated herbicides, 10 μl of ^{14}C-labeled-chlorimuron or -nicosulfuron was applied as a circular swipe on the third youngest leaf of each plant. The 10-μl solution contained approximately 2.63 kBq of ^{14}C-chlorimuron or -nicosulfuron in MeOH:H_2O (80:20, v/v) with a non-ionic surfactant[6] at 0.25% v/v. Plants were maintained in the greenhouse until harvested.

Plants were removed from soil 3, 6, 24, and 72 h after application (HAA)[2] of the radiolabeled herbicide, and the roots were rinsed and blotted dry. The plants were then divided into treated leaf, shoot above treated leaf, shoot below treated leaf, and roots. The treated leaves were rinsed in 20 ml of MeOH:H_2O (1:9, v/v) for 30 s to remove unabsorbed radioactivity. A 1 ml aliquot of the leaf rinse was added to 15 ml of scintillation fluid and radioactivity was quantified with liquid scintillation spectrometry (LSS)[2] in a commercial liquid scintillation spectrometer[7]. Herbicide absorption was calculated by subtracting the amount of ^{14}C recovered in the leaf rinse from the amount applied, and is expressed as a percent of the amount applied. The plant parts were weighed and dried for at least 48 h at 70 C and weighed again. Translocation of ^{14}C out

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[6] Ortho X-77, Valent USA Corp., 1333 N. California Blvd., Walnut Creek, CA 94596-8025. Nonionic surfactant with 80% principal functioning agents as: alkylarylpolyoxy ethylene glycols, free fatty acids, and isopropanol.

[7] LS-5800TA Model, Beckman Instrument Co., Fullerton, CA 92634.

of the treated leaf was measured by combusting the parts in a biological sample oxidizer[8] and counting the radioactivity, trapped as $^{14}CO_2$, by LSS. Radioactivity in plant parts other than the treated leaf was totaled and translocation out of the treated leaf was expressed as a percent of absorbed ^{14}C.

Metabolism studies. Plants were grown and treated with commercially formulated and radiolabeled herbicides as described for absorption and translocation studies. Since little radioactivity was translocated to the roots, only shoots were harvested at 3, 24, and 72 HAA of the radiolabeled herbicides. The plants were separated into parts, and the treated leaf rinsed as described earlier. Since a majority of the radioactivity remained in the treated leaf, only it was extracted for use in metabolism analysis. Metabolite and parent herbicide extraction was done with minor modifications of the methods of Brown and Neighbors (2). Treated leaves were frozen in liquid nitrogen, pulverized with mortar and pestle and homogenized in 10 ml of MeOH:H_2O (80:20, v/v). The homogenates were centrifuged at 2,000 x *g* for 10 min, and the supernatant was saved. The solid portion was extracted two more times with 10 ml of MeOH:H_2O (80:20, v/v). The combined supernatants (30 ml) were concentrated to 0.5 ml by evaporation. The concentrated extracts from the two replications of each treatment were combined to increase radioactivity levels and were cleaned by filtration through 0.2 μm nylon disposable filters[9]. Five μl of standard ^{14}C-chlorimuron or -nicosulfuron and 50 μl of each sample were

[8] B0306 Biological Sample Oxidizer, Packard Instruments Co., Downer Grove, IL 60515.

[9] Acrodisc disposable filters, Gelman Sciences, Ann Arbor, MI 48106.

loaded on silica gel thin layer chromatography (TLC)[2] plates[10]. Plates were run in duplicate and those loaded with chlorimuron were developed in a butanol:acetate:water (12:3:5, v/v/v) solvent system. Plates loaded with nicosulfuron were developed in a methylene chloride:methanol: ammonium hydroxide (6:1:0.1, v/v/v) solvent system.

Developed TLC plates were exposed to X-ray film[11] for 21 d, and the film was then developed to reveal position of radioactive bands on the plates. The radioactive bands were then scraped from the plate and the amount of radioactivity was determined by LSS. Metabolites were separated by their Rf[2] values and the radioactivity detected in each metabolite is expressed as a percent of the total radioactivity recovered during the TLC analysis.

Statistical analysis. Absorption, translocation, and metabolism studies were completely randomized designs with two replications of each treatment. Absorption and translocation data are from three studies, and metabolism data are from two studies. Data from separate studies were combined and analyzed statistically by ANOVA, and means were separated using Fisher's LSD test at the 0.05 significance level.

RESULTS AND DISCUSSION

Absorption and translocation studies. Chlorimuron was absorbed quickly into both R

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[10] Silica Gel 60 F_{254} precoated TLC plates, EM Science, 480 Democrat Rd., Gibbstown, NJ 08027.

[11] Kodak X-OMAT AR film, Eastman Kodak Co., Rochester, NY 14650.

and S smooth pigweed, with approximately 52% of the applied ^{14}C absorbed at 3 HAA of the radiolabeled chlorimuron (Figure 1). Chlorimuron absorption continued to increase with time to approximately 73% of the applied ^{14}C in the R and S biotypes at 72 HAA. Wilcut et al. observed similar and higher levels of chlorimuron absorption in various species (26). Nicosulfuron absorption was rapid in both biotypes but was higher in the R than in the S biotype at 3 HAA. Nicosulfuron absorption in the R biotype was 58% of the applied ^{14}C at 3 HAA but was only 50% at 6 HAA. The drop in absorption of nicosulfuron at 6 HAA in the R biotype is not understood; but absorption remained relatively constant at later harvest times, with similar levels of absorption at 3 and 72 HAA. Nicosulfuron absorption was 40% of the applied ^{14}C in the S biotype at 3 HAA and increased to 52% by 72 HAA. Absorption of nicosulfuron was similar in the two biotypes at 6, 24, and 72 HAA. In other research, 30 to 40% of applied nicosulfuron was absorbed by corn, a nicosulfuron-tolerant species, and johnsongrass [*Sorghum halepense* (L.) Pers.], a nicosulfuron-susceptible species (12, 21). Chlorimuron absorption was greater than that of nicosulfuron in both biotypes at 24 and 72 HAA, and was higher at 6 HAA in the R biotype.

Translocation of chlorimuron and nicosulfuron out of the treated leaf, as a percent of absorbed ^{14}C, was variable and did not increase from 3 to 72 HAA (Figure 2) in either biotype. Chlorimuron translocation out of the treated leaf was 16 to 37% of the absorbed ^{14}C in these studies, while that of nicosulfuron was 26 to 44% of the absorbed ^{14}C. Both herbicides were translocated apoplastically and symplastically, but were translocated into

the roots only slightly (data not presented). In previous research, chlorimuron translocation out of the treated leaf in numerous species was less than 20% of the applied ^{14}C (26). Simpson et al. showed that less than 5% of absorbed nicosulfuron was translocated out of the treated leaf in corn (21). Obrigawitch et al. showed that 20 to 30% of absorbed nicosulfuron was translocated out of the treated leaf in johnsongrass (12). While translocation of sulfonylurea herbicides out of the treated leaf is limited (12, 16, 21, 26), translocation may be contributing to nicosulfuron selectivity since it was translocated out of the treated leaf in susceptible johnsongrass and pigweed, but was not in tolerant corn.

Metabolism studies. Since a majority of the radioactivity was in the treated leaf (Figure 2), metabolism of ^{14}C-chlorimuron and -nicosulfuron was analyzed only in the treated leaf in both biotypes. Metabolites of chlorimuron and nicosulfuron were not identified.

Standard unmetabolized chlorimuron had an Rf value of 0.94 in our solvent system. Chlorimuron metabolism was greater at 3 HAA in the R than in the S biotype (Table 1). However, at 24 and 72 HAA chlorimuron metabolism was greater in the S than in the R biotype, but less than 50% of the extracted ^{14}C was unmetabolized chlorimuron at these harvest times in both biotypes. Selectivity with chlorimuron is due to rapid metabolism in tolerant species (2, 11, 26). Chlorimuron-tolerant soybean had as much as 70% of recovered ^{14}C as unmetabolized chlorimuron at 24 HAA and as much as 40% at 72 HAA (11). Both biotypes of smooth pigweed may be able to tolerate low levels of chlorimuron in the treated leaf at 24 and 72 HAA. If this is true, our results indicate that higher

tolerance of the R biotype is due to more rapid metabolism in that biotype.

In this research, two major metabolites of chlorimuron were observed with Rf values of 0.58 and 0.64, and a minor metabolite was observed with an Rf value of 0.51. At 72 HAA, the S biotype had accumulated more radioactivity at Rf 0.64 than the R biotype; otherwise, both biotypes accumulated similar amounts of each metabolite. Two major early metabolites in soybean are the chlorimuron-homoglutathione conjugate and the de-esterified free acid of chlorimuron, both of which are inactive against soybean ALS (2). The metabolites in these biotypes of smooth pigweed may correspond to the metabolites in soybean, but further research needs to be conducted to confirm this hypothesis. It is unlikely that the metabolites in R and S smooth pigweed are affecting the sensitivity of the two biotypes to chlorimuron.

Standard nicosulfuron had an Rf value of 0.25 in our solvent system (Table 2). There was another band observed in the nicosulfuron standard with an Rf value of 0.19 which contained 11% of the ^{14}C extracted in these studies. No nicosulfuron was metabolized to a greater extent over time than at 3 HAA in both biotypes, and the amount of parent nicosulfuron identified in the treated leaf samples was only slightly lower than that in the standard nicosulfuron (Table 2). A metabolite with an Rf value of 0.83 in the R biotype and 0.74 in the S biotype was observed at 3 HAA, but both metabolites appeared to be transient since they were essentially gone by 72 HAA. Bands were identified with an Rf value of 0.19 in both biotypes, but they correspond to the band identified in the standard nicosulfuron. These results indicate that little metabolism of nicosulfuron is occurring in

either biotype of smooth pigweed which may explain the high susceptibility of these biotypes to nicosulfuron. In previous research, less than 30% of nicosulfuron was metabolized in susceptible johnsongrass up to 72 HAA, while greater than 90% of nicosulfuron was metabolized in as little as 4 HAA in tolerant corn (12, 21). Our results concur with previous research indicating that little nicosulfuron metabolism occurs in susceptible plants.

Absorption and translocation of chlorimuron do not affect the susceptibility of either biotype to this herbicide. The higher amounts of nicosulfuron absorption at 3 HAA in the R biotype did not result in a differential response at the whole plant level (chapter 5). The two-fold level of cross-resistance to chlorimuron in the R biotype might be explained by the slightly more rapid metabolism in this biotype (Table 1). The S biotype had metabolized more chlorimuron than the R biotype at the later harvests, but chlorimuron may have been metabolized to non-toxic levels. The more rapid metabolism correlating with higher tolerance levels agrees with results from other researchers (2, 11, 12, 15, 21, 25, 26).

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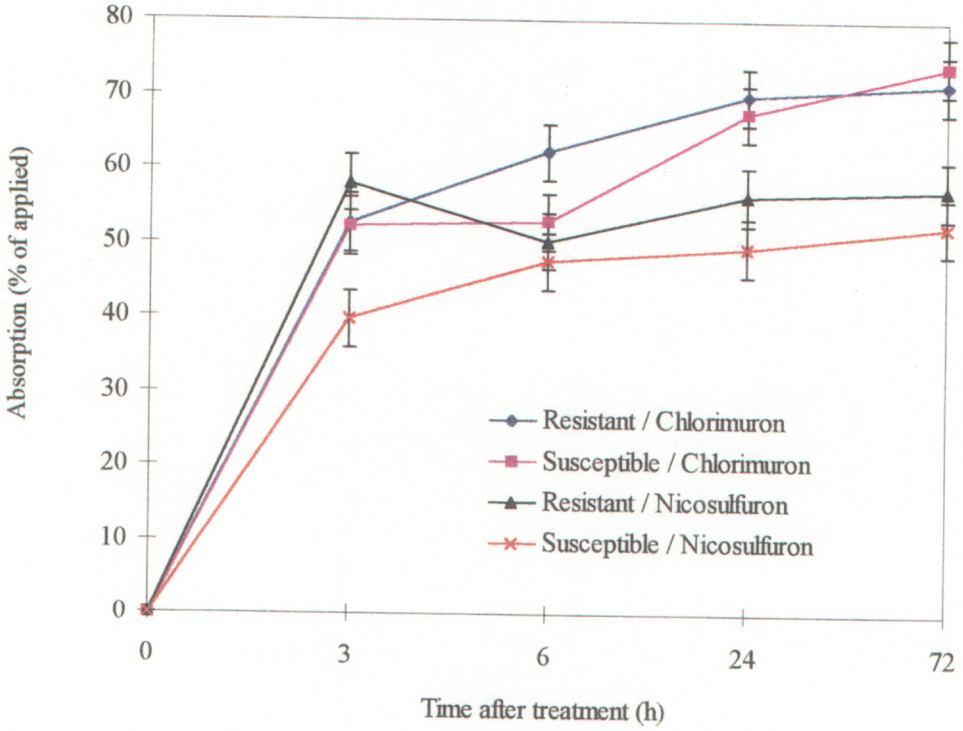

Figure 1. Absorption of foliar-applied ^{14}C-chlorimuron and -nicosulfuron by imidazolinone-resistant and -susceptible smooth pigweed as a percent of the applied radioactivity. Data points are the means from three studies with the Fisher's LSD value (0.05 significance level) centered around it.

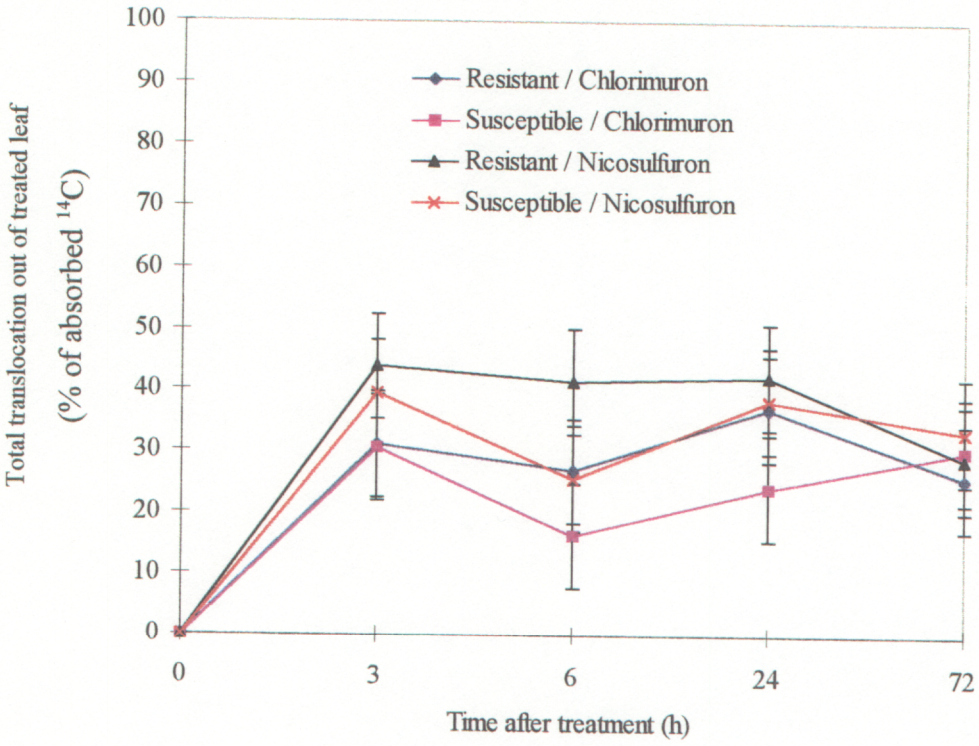

Figure 2. Time course of foliar-applied ^{14}C-chlorimuron and -nicosulfuron translocation out of the treated leaf as a percent of the absorbed ^{14}C in imidazolinone-resistant and -susceptible smooth pigweed. Data points are means from three studies with the Fisher's LSD value (0.05 significance level) centered around it.

Table 1. TLC analysis of the metabolism of foliar-applied ^{14}C-chlorimuron by imidazolinone-resistant and -susceptible smooth pigweed 3, 24, and 72 h after treatment.[a]

		Distribution of extracted ^{14}C			
Population	Time after treatment	Rf = 0.51	Rf = 0.58	Rf = 0.64	Rf = 0.94
	(h)	(% of ^{14}C extracted)			
Resistant	3	7.0	0.0	6.0	68.5
Resistant	24	6.5	10.5	16.8	48.5
Resistant	72	10.3	16.5	13.8	39.5
Susceptible	3	4.0	0.0	4.8	81.5
Susceptible	24	9.8	13.8	16.0	37.8
Susceptible	72	10.0	16.8	22.2	29.8
LSD (0.05)		5.5	4.3	7.7	8.2

[a] Approximately 96% of the ^{14}C extracted in the chlorimuron standard migrated to an Rf value of 0.94.

Table 2. TLC analysis of the metabolism of foliar-applied ^{14}C-nicosulfuron by imidazolinone-resistant and -susceptible smooth pigweed 3, 2^{4}, and 72 h after treatment.[a]

		Distribution of extracted ^{14}C				
	Time after			Sum		
Population	treatment	Rf = 0.19	Rf = 0.25	Rf=0.19+0.25	Rf = 0.74	Rf = 0.83
	(h)			(% of ^{14}C extracted)		
Resistant	3	10.3	52.0	62.3	3.0	20.5
Resistant	24	15.8	58.3	74.1	3.3	11.3
Resistant	72	14.3	65.3	79.6	2.3	2.5
Susceptible	3	11.8	54.3	66.1	16.0	7.0
Susceptible	24	13.5	63.5	77.0	9.5	3.8
Susceptible	72	11.3	65.8	77.1	3.3	4.0
LSD (.05)		3.7	5.9		1.7	2.2

[a] Approximately 72% of the ^{14}C extracted in the nicosulfuron standard migrated to an Rf value of 0.25, and 11% migrated to an Rf value of 0.19.

Chapter VIII

Summary and Conclusions

Crop rotations and herbicide programs affected weed population dynamics and crop yields from 1991 to 1994, but weed populations were influenced more by species and specific herbicides included. Generally, tolerant weed species proliferated where the same herbicide or herbicides with similar selectivities were used continuously. Common lambsquarters and yellow nutsedge control was lowest and densities were highest where fomesafen plus fluazifop-P plus fenoxaprop were applied for 4 consecutive years. Common ragweed control was generally lowest and densities were generally highest where only ALS-inhibitors were applied for 4 years. Stinkgrass control was lowest from imazethapyr, and densities were highest in 1994 where only imazethapyr was applied for 4 years. Smooth crabgrass densities were highest in 1994 from imazaquin plus nicosulfuron or from continuous ALS-inhibitor programs. Increased densities of numerous tolerant weed species following the continuous use of a weed control practice have been reported previously (1, 3, 5, 9, 12, 20). Densities of goosegrass, stinkgrass, large crabgrass, and fall panicum increased from 1991 to 1994 following four annual applications of butylate plus atrazine likely due, in part, to enhanced microbial degradation of butylate (13, 16, 22). Shifts in densities of other weed species were not observed due to variable control from herbicides included or to low densities of the weed species. Crop yields were generally affected by weed control, rainfall, and crop rotations. Corn and soybean yields

were generally high where overall weed control and rainfall were high and where crops were rotated.

Weed shifts can be to a tolerant species or to an herbicide-resistant biotype of the same species, but the weed community will adapt to the environment if the environment is not changed (2, 7, 8, 11). Herbicide resistant biotypes are presumed to be present in the weed population prior to the application of an herbicide, and the continuous use of an herbicide selects those resistant plants (8, 10, 11, 15). From 1992 to 1994, approximately 5 million Painter smooth pigweed plants were treated with imazethapyr or nicosulfuron and no resistant plants were identified in that population. A gene conferring herbicide resistance must occur in the weed population in order for resistance to develop (15).

Following six consecutive, annual applications of imazaquin in combination with trifluralin or pendimethalin, smooth pigweed was no longer controlled at two separate locations (Marion, MD and Oak Hall, VA). Additionally, unacceptable livid amaranth control from imazethapyr was observed following three consecutive, biannual applications of imazethapyr in Warren County, NJ. In greenhouse studies, Marion and Oak Hall smooth pigweed tolerated 560 and 1120 g ai/ha imazaquin, respectively while Painter smooth pigweed was controlled with 70 g/ha imazaquin. Warren County livid amaranth also tolerated 560 g ai/ha imazethapyr. Each of these populations was initially controlled with applications of imazaquin or imazethapyr.

Imazaquin and imazethapyr gave complete control of the Painter (S) population, but did not control the Marion (R) population in field studies. Control of the two populations

from other herbicides in field studies was comparable, but was not complete (less than 90%) from chlorimuron, CGA-152005, flumiclorac, halosulfuron, lactofen, metribuzin, primisulfuron, and pendimethalin. In greenhouse studies, the R biotype had >25- and >185-fold levels of resistance to imazaquin and imazethapyr, respectively compared to the S biotype based on dry weights. The R biotype also had two- and seven-fold levels of resistance to chlorimuron and rimsulfuron, respectively in greenhouse studies; but control of the two biotypes by nicosulfuron, pendimethalin, pyrithiobac, and thifensulfuron was comparable. Control of the two biotypes from pendimethalin was comparable but not complete; hence the inclusion of pendimethalin did not prevent the development of the imidazolinone-resistant population.

Resistance to imazaquin, imazethapyr, and rimsulfuron in the R biotype was due to an altered ALS enzyme which was 83-, 397-, and 12-fold more resistant to imazaquin, imazethapyr, and rimsulfuron, respectively than ALS from the S biotype. Negative cross-resistance to pyrithiobac and thifensulfuron was observed in the R-ALS, which was four- and three-fold less tolerant to pyrithiobac and thifensulfuron, respectively than S-ALS. ALS from the two biotypes responded similarly to CGA-152005, chlorimuron, halosulfuron, nicosulfuron, and primisulfuron. Target site modifications have been the resistance mechanisms in all but one of the ALS-inhibitor-resistant weed populations in which the resistance mechanism was determined (4, 10, 11, 18, 19, 20).

Although other mechanisms of herbicide-resistance have been observed in weed populations, such as enhanced metabolism and compartmentalization of the herbicide,

target-site-based resistance is most common and often produces the highest levels of resistance with any herbicide (6, 10, 11, 14, 17). Target-site-based resistance often involves a single amino acid substitution which can occur relatively freqently, but the occurrence of these substitutions cannot be predicted (7, 11, 14, 17). Several different amino acid substitutions can confer target-site-based resistance to ALS-inhibitor herbicides which result in varying patterns of cross-resistance to members of herbicide families other than that of the selection agent (18). The Marion biotype is highly resistant to the imidazolinone herbicides, imazaquin and imazethapyr, and is cross-resistant at low levels to rimsulfuron and chlorimuron.

Low levels of cross-resistance to chlorimuron in the R biotype are not due to a target site alteration or to uptake or translocation of chlorimuron. Uptake and translocation of chlorimuron were similar in both the R and S biotypes of smooth pigweed. Rather, cross-resistance to chlorimuron in the R biotype is likely due to slightly faster metabolism of chlorimuron compared with the S biotype. The cross-resistance to chlorimuron in the R biotype was not observed in field studies. Uptake and translocation of nicosulfuron in the two biotypes was similar, and little metabolism of nicosulfuron occurred in either biotype.

Given the results from this research and previous research, the development of herbicide resistance cannot be predicted accurately. However, herbicide resistance must be managed to maintain the effectiveness of our current herbicides for years to come. The results of the rotational studies and the development of the Marion smooth pigweed population in the presence of pendimethalin and imazaquin stress the need to rotate weed

management practices or to include multiple weed management practices in the same year which are effective on the target weed species. The herbicides or other weed management practices included must be effective on the target weed species to prevent or slow weed shifts. The inclusion of herbicides with multiple modes of action also helps to reduce the risk of selecting herbicide-resistant weed populations.

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Appendix

Table A.1. Analysis of main and interaction effects of crop rotations and herbicide programs on control of four annual broadleaf weeds from 1991 to 1994 at two sites in Painter, VA.[a]

Year	Factor	Site one				Site two			
		CHEAL[b]	AMBEL	AMASP	DATST	CHEAL	AMBEL	AMASP	DATST
		Significance[c]							
1991	Main effect of crop rotations	**	-	*	NS	*	-	NS	NS
	Main effect of herbicide programs	**	-	**	**	**	-	**	**
	Interaction of crop rotations x herbicide programs	**	-	**	**	**	-	NS	**
1992	Main effect of crop rotations	**	*	NS	**	**	NS	*	-
	Main effect of herbicide programs	**	**	**	**	**	**	**	-
	Interaction of crop rotations x herbicide programs	**	**	*	**	**	**	**	-
1993	Main effect of crop rotations	**	NS	**	NS	NS	NS	**	NS
	Main effect of herbicide programs	**	**	**	**	**	**	**	**
	Interaction of crop rotations x herbicide programs	**	**	**	*	**	**	**	NS
1994	Main effect of crop rotations	**	*	-	NS	**	**	-	NS
	Main effect of herbicide programs	**	**	-	**	**	**	-	**
	Interaction of crop rotations x herbicide programs	**	**	-	NS	**	**	-	NS

[a] Visual control ratings were made 82, 100, 98, and 78 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively.

[b] CHEAL = common lambsquarters, AMBEL = common ragweed, AMASP = redroot and smooth pigweed, DATST = jimsonweed.

[c] NS = not significant at p value of 0.05, * = p value of 0.05 level of significance, and ** = p value of 0.001 level of significance by Fisher's LSD test. - means no data.

Table A.2. Analysis of main and interaction effects of crop rotations and herbicide programs on densities of four annual broadleaf weeds from 1991 to 1994 at two sites in Painter, VA.[a]

Year	Factor	Site one				Site two			
		CHEAL[b]	AMBEL	AMASP	DATST	CHEAL	AMBEL	AMASP	DATST
		Significance[c]							
1991	Main effect of crop rotations	NS	NS	NS	NS	**	NS	NS	NS
	Main effect of herbicide programs	NS	NS	**	*	**	NS	**	**
	Interaction of crop rotations x herbicide programs	*	NS	*	*	**	NS	NS	NS
1992	Main effect of crop rotations	NS	NS	NS	*	*	NS	NS	NS
	Main effect of herbicide programs	**	**	**	**	**	*	**	*
	Interaction of crop rotations x herbicide programs	NS	NS	NS	*	NS	NS	*	*
1993	Main effect of crop rotations	*	NS	NS	NS	NS	NS	NS	NS
	Main effect of herbicide programs	**	**	**	**	**	**	**	**
	Interaction of crop rotations x herbicide programs	**	NS	NS	*	**	*	NS	NS
1994	Main effect of crop rotations	*	NS	NS	NS	NS	NS	NS	NS
	Main effect of herbicide programs	**	**	**	**	**	**	*	**
	Interaction of crop rotations x herbicide programs	**	**	NS	NS	**	*	NS	NS

[a] Population counts were taken 35, 58, 37, and 63 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site one; and 43, 62, 47, and 64 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site two.

[b] CHEAL = common lambsquarters, AMBEL = common ragweed, AMASP = redroot and smooth pigweed, DATST = jimsonweed.

[b] NS = not significant at p value of 0.05, * = p value of 0.05 level of significance, and ** = p value of 0.001 level of significance by Fisher's LSD test.

Table A.3. Analysis of main and interaction effects of crop rotations and herbicide programs on corn, soybean, and tomato yields from 1991 to 1994 at two sites in Painter, VA.

Year	Factor	Site one			Site two[a]	
		Corn	Soybean	Tomato	Corn	Soybean
		Significance[b]				
1991	Main effect of crop rotations	**	**	-	**	**
	Main effect of herbicide programs	**	**	-	**	**
	Interaction of crop rotations x herbicide programs	**	**	-	**	**
1992	Main effect of crop rotations	**	**	**	**	**
	Main effect of herbicide programs	**	**	**	**	**
	Interaction of crop rotations x herbicide programs	**	**	**	**	**
1993	Main effect of crop rotations	**	**	-	**	**
	Main effect of herbicide programs	**	**	-	**	**
	Interaction of crop rotations x herbicide programs	**	**	-	**	**
1994	Main effect of crop rotations	**	**	-	**	**
	Main effect of herbicide programs	**	**	-	**	**
	Interaction of crop rotations x herbicide programs	**	**	-	**	**

[a] The corn-tomato-soybean rotation was not included at site two, so tomato was not planted at this site.

[b] NS = not significant at p value of 0.05, * = p value of 0.05 level of significance, and ** = p value of 0.001 level of significance by Fisher's LSD test. - means no data.

Table A.4. Analysis of main and interaction effects of crop rotations and herbicide programs on control of three annual grasses and yellow nutsedge from 1991 to 1994 at two sites in Painter, VA.[a]

Year	Factor	Site one			Site two			
		ELEIN[b]	ERACN	DIGSA	ELEIN	ERACN	DIGSA	CYPES
		Significance[c]						
1991	Main effect of crop rotations	*	-	-	NS	-	NS	-
	Main effect of herbicide programs	**	-	-	**	-	**	-
	Interaction of crop rotations x herbicide programs	**	-	-	**	-	NS	-
1992	Main effect of crop rotations	**	**	-	**	**	*	**
	Main effect of herbicide programs	**	**	-	**	**	**	**
	Interaction of crop rotations x herbicide programs	**	**	-	**	**	**	**
1993	Main effect of crop rotations	*	**	-	**	*	*	**
	Main effect of herbicide programs	**	**	-	**	**	**	**
	Interaction of crop rotations x herbicide programs	**	**	-	**	**	**	**
1994	Main effect of crop rotations	**	**	**	**	**	**	**
	Main effect of herbicide programs	**	**	**	**	**	**	**
	Interaction of crop rotations x herbicide programs	**	**	**	**	**	**	**

[a] Visual control ratings were made 82, 100, 98, and 78 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively.

[b] ELEIN = goosegrass, ERACN = stinkgrass, DIGSA = large crabgrass, CYPES = yellow nutsdge.

[c] NS = not significant at p value of 0.05, * = p value of 0.05 level of significance, and ** = p value of 0.001 level of significance by Fisher's LSD test. - means no data.

Table A.5. Analysis of main and interaction effects of crop rotations and herbicide programs on densities of three annual grasses from 1991 to 1994 at two sites in Painter, VA.[a]

Year	Factor	Site one			Site two		
		ELEIN[b]	ERACN	DIGSA	ELEIN	ERACN	DIGSA
		Significance[c]					
1991	Main effect of crop rotations	NS	NS	NS	*	NS	NS
	Main effect of herbicide programs	*	NS	NS	**	*	**
	Interaction of crop rotations x herbicide programs	NS	NS	NS	NS	*	NS
1992	Main effect of crop rotations	NS	NS	NS	NS	NS	NS
	Main effect of herbicide programs	**	*	*	**	**	**
	Interaction of crop rotations x herbicide programs	*	NS	NS	*	**	**
1993	Main effect of crop rotations	*	NS	NS	*	NS	*
	Main effect of herbicide programs	NS	NS	**	**	**	**
	Interaction of crop rotations x herbicide programs	*	NS	*	*	NS	*
1994	Main effect of crop rotations	**	*	NS	*	*	NS
	Main effect of herbicide programs	**	**	**	NS	**	**
	Interaction of crop rotations x herbicide programs	**	**	*	*	**	**

[a] Population counts were taken 35, 58, 37, and 63 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site one; and 43, 62, 47, and 64 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site two.

[b] ELEIN = goosegrass, ERACN = stinkgrass, DIGSA = large crabgrass.

[c] NS = not significant at p value of 0.05, * = p value of 0.05 level of significance, and ** = p value of 0.001 level of significance by Fisher's LSD test.

Table A.6. Analysis of main and interaction effects of crop rotations and herbicide programs on densities of two annual grasses and yellow nutsedge from 1991 to 1994 at two sites in Painter, VA.[a]

Year	Factor	Site one			Site two		
		DIGIS[b]	PANDI	CYPES	DIGIS	PANDI	CYPES
		Significance[c]					
1991	Main effect of crop rotations	-	NS	NS	-	NS	NS
	Main effect of herbicide programs	-	**	NS	-	**	NS
	Interaction of crop rotations x herbicide programs	-	NS	NS	-	NS	*
1992	Main effect of crop rotations	*	NS	NS	NS	NS	NS
	Main effect of herbicide programs	**	**	**	**	*	**
	Interaction of crop rotations x herbicide programs	**	*	*	NS	*	*
1993	Main effect of crop rotations	NS	NS	*	NS	*	NS
	Main effect of herbicide programs	NS	NS	NS	NS	*	*
	Interaction of crop rotations x herbicide programs	*	NS	NS	NS	**	*
1994	Main effect of crop rotations	*	**	NS	NS	*	**
	Main effect of herbicide programs	**	**	*	**	**	**
	Interaction of crop rotations x herbicide programs	**	**	*	NS	**	**

[a] Population counts were taken 35, 58, 37, and 63 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site one; and 43, 62, 47, and 64 days after PPI treatments were applied in 1991, 1992, 1993, and 1994, respectively at site two.

[b] DIGIS = smooth crabgrass, PANDI = fall panicum, CYPES = yellow nutsedge.

[c] NS = not significant at p value of 0.05, * = p value of 0.05 level of significance, and ** = p value of 0.001 level of significance by Fisher's LSD test. - means no data.

VITA

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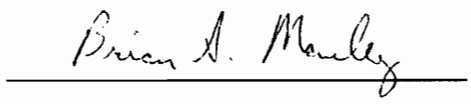

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