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DISSERTATION

**CHARACTERIZATION AND MANAGEMENT OF KOCHIA
EXHIBITING VARIABLE RESPONSES TO DICAMBA**

Submitted by

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In partial fulfillment of the requirements
for the Degree of Doctor of Philosophy

Colorado State University

Fort Collins, Colorado

Fall 1999

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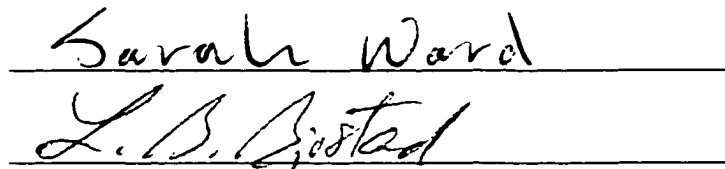
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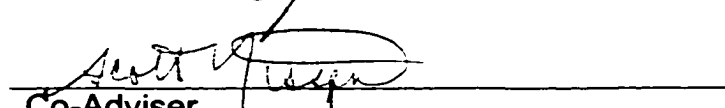
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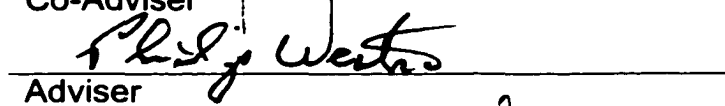
WE HEREBY RECOMMEND THAT THE DISSERTATION PREPARED
UNDER OUR SUPERVISION BY KIRK A. HOWATT ENTITLED
**CHARACTERIZATION AND MANAGEMENT OF KOCHIA EXHIBITING
VARIABLE RESPONSES TO DICAMBA** BE ACCEPTED AS FULFILLING IN
PART REQUIREMENTS FOR THE DEGREE OF DOCTOR OF PHILOSOPHY.

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ABSTRACT OF DISSERTATION
CHARACTERIZATION AND MANAGEMENT OF KOCHIA
EXHIBITING VARIABLE RESPONSES TO DICAMBA

Kochia (*Kochia scoparia* (L.) Schrader), at isolated locations, was identified as having variable responses to dicamba. These responses included lower observed injury and increased plant survival compared to susceptible populations. The response of kochia at these locations varied spatially in relation to field perimeters, area within a field, and plant within an area. In addition, injury varied among progeny from an individual plant. Kochia injury increased as dicamba rate increased for all kochia samples. In an effort to further characterize kochia and develop an objective method of characterizing kochia responses to dicamba, experiments were conducted to examine ethylene production as an herbicidal response and the response of kochia to ethylene. Ethylene was produced in response to dicamba treatments, but ethylene did not cause any symptoms on young vegetative kochia plants. Dicamba-susceptible kochia (accession S2) produced more ethylene than non-susceptible kochia (the Henry accession). The rate of ethylene evolution increased over time and as dicamba rate increased. Accession S2 produced as much as 470% of the ethylene generated by the Henry accession. Many herbicides proved to be effective

management tools in these experiments. Accessions varied in susceptibility to alternative herbicides, but there were no consistencies in the order of accessions when ranked from most to least injury. Smaller kochia was more injured for all herbicide treatments; however, systemic herbicides tended to retain more herbicidal activity as kochia size increased. Pre-emergence dicamba applications caused over 93% injury to kochia from the Henry accession compared to 84% injury for comparable post-emergence treatments. Accessions responded similarly to dicamba applications directed to the root zone or to the foliage. The difference in injury between kochia accessions was not affected by spray volume. Crop competition from winter wheat reduced kochia above ground biomass but had no effect on kochia population. Dicamba application reduced the number of kochia plants and caused lower kochia biomass production in some instances.

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DEDICATION

**At the darkest times
You lighten my load,
At the brightest times
Sharing with you increases the joy.**

This work is dedicated to Mary Beth.

Love always.

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LIST OF CHEMICALS

Common Name	Chemical Name
aminooxyacetic acid	carboxymethoxylamine
atrazine	6-chloro- <i>N</i> -ethyl- <i>N</i> -(1-methylethyl)-1,3,5-triazine-2,4-diamine
bromoxynil	3,5-dibromo-4-hydroxybenzonitrile
carfentrazone-ethyl	α ,2-dichloro-5-[4-(difluoromethyl)-4,5-dihydro-3-methyl-5-oxo-1 <i>H</i> -1,2,4-triazol-1-yl]-4-fluorobenzenepropanoic acid, ethyl ester
chlorsulfuron	2-chloro- <i>N</i> -[[4-methoxy-6-methyl-1,3,5-triazin-2-yl)amino]carbonyl]benzenesulfonamide
2,4-D	(2,4-dichlorophenoxy)acetic acid
dicamba	3,6-dichloro-2-methoxybenzoic acid
ethephon	(2-chloroethyl)phosphonic acid
fluroxypyr	[(4-amino-3,5-dichloro-6-fluoro-2-pyridinyl)oxy]acetic acid
MCPA	(4-chloro-2-methylphenoxy)acetic acid
pyridate	O-(6-chloro-3-phenyl-4-pyridazinyl) <i>S</i> -octyl carbonothioate

LIST OF ABBREVIATIONS

Abbreviation	Definition
ae	acid equivalent
ai	active ingredient
cm	centimeter
FW	fresh weight
g	gram
ha	hectare
hr	hour
L	liter
mM	millimolar
ng	nanogram

CHAPTER 1

KOCHIA

The Plant.

Kochia (*Kochia scoparia* (L.) Schrader) is a member of the Chenopodiaceae family. This herbaceous, dicotyledonous plant is a common weed in dryland and irrigated agriculture of the Northwest regions of North America causing yield losses in a number of crops (Black et al. 1969; Buhler et al. 1985; Dexter 1982; Durgan et al. 1990; Weatherspoon and Schweizer 1971). Unrestricted kochia growth severely interferes with crops and may reduce crop yield by more than 95% (Weatherspoon and Schweizer 1971).

Kochia is an annual weed that is thought to be native to southern and eastern Russia. It was introduced to North America from Europe as an ornamental plant because of its even conical shape, dense growth, and variegated red pigmentation on some stems (Holm et al. 1979). While kochia is an aggressive weed throughout the western United States and Canada, its worldwide distribution is rather limited. Kochia is primarily reported as a weed in North America, Argentina, and Afghanistan (Holm et al. 1979).

Morphology.

Kochia has highly variable morphology, which may be influenced by several genetic and environmental factors. The plant may reach a maximum reproductive height of 0.15 meters or may grow to over two meters tall (Eberlein and Fore 1984). Kochia growing under spatial stress is very tall and spindly, while kochia growing in the absence of competition remains shorter and takes up more lateral space (Eberlein and Fore 1984). Stems are rounded with vertical ribs becoming apparent as the plant matures. Stems are erect, pale green, and highly branched; however, branching is very dependent on the proximity and number of other plants. Lower leaves are linear to lanceolate in shape and 2.5 to 5 centimeters in length, while upper leaves are more oval and generally much smaller (Zimdahl 1983). The density of pubescence or even the presence of pubescence on kochia leaves varies, but hairs are normally present along the leaf margins (Zimdahl 1983). Kochia's branching taproot can extend into the soil three times as deep as the plant is tall (Hubbel 1931). Lateral branches can extend kochia's root profile to seven meters in diameter when the roots reach a depth of two meters (Davis et al. 1967).

Flowers produce a single seed (Zimdahl 1983). A thin perianth encloses the seed, and this seed often remains attached to the calyx (Eberlein and Fore 1984; Zimdahl 1983). The seed coat protects a plantlet that is coiled inside the seed (personal observation). This plantlet contains green and purple pigmentation, and its root and shoot primordia are already differentiated.

Fivehook bassia, *Bassia hisiopifolia* (Pallus) Kuntze, is another weed in the family Chenopodiaceae that very closely resembles kochia. Bassia can be distinguished from kochia by slight differences in seed coat texture but more characteristically by hooked sepals (Harrington 1964; Weber 1976).

Genetics.

Kochia is a diploid species with $2n$ chromosome number equal to 18 (Sivakumaran et al. 1991). The mating system was originally believed to be highly self-pollinating due to several studies that reported 0.5 to 13.1% cross-pollination (Mulugeta et al. 1992; Stallings et al. 1995; Thill et al. 1993). Examination of inbreeding coefficients, however, showed that kochia populations undergo a high degree of out-breeding and approximate random mating coefficients (Guttieri et al. 1998). Modeling of the distribution pattern of kochia pollen indicates that nuclear gene transfer can occur to distances of more than 150 meters from the paternal plant (Mulugeta et al. 1994).

Biology.

Kochia can produce an average of 12,000 seeds per plant in various competitive relationships (Thompson et al. 1994). The amount of seed produced can be as much as 23,000 seeds per plant under optimum growth conditions (Nussbaum et al. 1983). Most of the seeds produced drop to the ground before the plant senesces and becomes a tumbleweed (Fay et al. 1992).

Kochia seeds express very limited dormancy and do not require light signaling to germinate (Everitt et al. 1983). Germination can occur with only one accumulated degree day above 10° Celsius and therefore can germinate very early in the spring (Alan and Wise 1985). Kochia seeds also may germinate immediately following harvest from the plant. Limited dormancy influences the longevity of seed in the soil. Kochia seed germination can drop to 5% after one year in the soil and to less than 1% germination after two years (Burnside et al. 1981).

Kochia seed germination and emergence are favored at shallow depths. Kochia emergence is as high as 74% for seed that remains on the soil surface (Everitt et al. 1983). Seedling emergence is reduced even by shallow planting (57% emergence at 3 millimeter depth) (Everitt et al. 1983).

Plasticity of kochia morphology is also expressed in flowering. Kochia is a short day plant with a critical light period for floral initiation that varies among plants ranging from 13 to 15 hours (Bell et al. 1972). Plants can flower in as few as 57 days after emergence or may not flower until 100 days after emergence (Bell et al. 1972). Obligate out-crossing of individual flowers is believed to occur because the stigmas of a flower appear receptive to pollen several days before pollen is shed from the anthers on that flower (Thill et al. 1993). Self-fertilization of an individual kochia flower can still occur due to the fact that pollen may remain viable for up to 12 days (Mulugeta et al. 1994).

Although kochia seed generally drops at the base of the plant, seed dispersal may occur by means of tumbleweeds (Becker 1978; Fay et al. 1992).

Kochia tumbleweeds develop due to a loss of stem flexibility rather than the development of an abscission layer (Becker 1978). As the plant senesces, a loss of capillary water leads to reduced flexibility and increased brittleness, which results in the stem breaking under stress from strong winds or animal activity (Becker 1978).

Ecology.

The tumbleweed characteristic of kochia provides an effective means of rapid spread over large geographic regions (Becker 1978). Kochia was first collected in Wyoming before 1900 (Forcella 1985). By the 1930s kochia had spread east into Idaho, and kochia moved north into central Montana by the 1940s (Forcella 1985). In the 1950s, kochia was ubiquitous throughout Wyoming and Montana (Forcella 1985). A survey of Idaho alfalfa fields in the 1970s showed that kochia infested over 60% of the fields, and in nearly half of the fields, kochia covered more than 75% of the area (Calpouzos et al. 1980).

Once introduced to an area, kochia can tolerate a number of environmental stresses (Eberlein and Fore 1984; Steppuhn and Wall 1993). Kochia is well known for tolerance to high salinity levels (Biliski and Foy 1988; Steppuhn and Wall 1993). Possible mechanisms of this tolerance include a high affinity and selectivity for nutrient ions, high concentrations of osmoprotectants such as glycinebetaine, and cell wall enzymes that are tolerant to elevated salt levels (Curtin et al. 1993; Selvaraj et al. 1995; Thiyagarajah et al. 1996). Kochia can also absorb and tolerate contaminants such as copper, nickel, and cadmium

(Dreesen and Cokal 1984). Kochia's vast root system combined with its fairly high water use efficiency enables plant survival under drought conditions (Davis et al. 1967; Nussbaum et al. 1985). Kochia is tolerant to a wide range of soil pH and can germinate equally well at pH conditions between 2 and 12 (Everitt et al. 1983). These factors as well as a favorable CO₂ compensation point, early germination, rapid growth, and tumbleweed characteristic contribute to the weedy nature and competitiveness of kochia (Black et al. 1969; Nussbaum et al. 1985).

Kochia is grown as a forage crop in many areas of the world because of its salt and drought tolerance (Garduno 1990; Khassanov et al. 1994; Madrid et al. 1996; McArthur et al. 1996). The feed quality of kochia is very high and nearly equal to alfalfa (Durham and Durham 1979). Besides having a high protein content, good digestibility, and favorable leaf retention when baled, kochia pastures can support eight times as many animals as grass pastures (Coxworth et al. 1969; Durham and Durham 1979). Feeding kochia to animals must be well managed though due to documented pathology of livestock death from kochia toxicosis (Lorenz and Dewey 1988).

Kochia is also beneficial as a candidate for bioremediation projects. High salinity tolerance combined with contaminant absorption makes kochia a good plant to detoxify waste areas and reclaim mining sites (Biliski and Foy 1988; Dreesen and Cokal 1984). And atrazine-resistant kochia supports a rhizosphere that enhances mineralization of pesticides in contaminated soil (Perkovich et al. 1996).

Kochia produces many secondary metabolites (Dinan et al. 1998; Wen et al. 1995). Some compounds, such as saponins, need to be removed from kochia seed before feeding to livestock (Coxworth et al. 1969). Kochia may be cultivated in the future to take advantage of a secondary metabolite that aids mosquito control programs (Agelopoulos et al. 1999). 5-hexadecenoic acid is a precursor to a known mosquito oviposition pheromone. This precursor is found at fairly high levels in kochia and may be extracted and used in a program to regulate mosquito oviposition behavior (Agelopoulos et al. 1999).

Resistance to Herbicides.

Specific cases of herbicide-resistant kochia biotypes have evolved. The most prevalent resistant kochia biotype is ALS-resistance with over 500 kochia sites of confirmed resistance (Holt et al. 1993). ALS-resistant kochia was first reported concurrently in South Dakota, New Mexico, and Canada (Heap 1997). ALS-resistance quickly spread in the western United States and Canada and is now confirmed in nine states and two Canadian territories (Heap 1999).

Kochia resistant to triazine herbicides was first confirmed in 1982 (Heap 1997). Triazine resistance has been reported to be present in six states, which is likely an underestimation of the actual occurrence as is the case for ALS-resistance (Heap 1999). Auxinic herbicide resistance was reported in Montana and North Dakota kochia biotypes in 1995 (Heap 1997).

Kochia fitness varies as a result of resistance type. Fitness measurements include parameters of growth and reproduction that enable the

contribution of genetic material to future generations. Fitness was reported to be reduced in several ways for kochia biotypes that are resistant to triazines (Conrad and Radosevich 1979; Holt et al. 1991). This is not the case with ALS-resistant kochia, which often shows no fitness deviation from susceptible biotypes (Thompson et al. 1994b). ALS-resistant biotypes, on the other hand, may express greater fitness than susceptible plants for parameters such as germination, leaf weight, stem weight, and seed production (Thompson et al. 1994a; Thompson et al. 1994b; Christoffoleti 1993). Little is known about the fitness of auxinic herbicide-resistant kochia. Many other herbicides remain efficacious to resistant biotypes of kochia which can be used in combinations to manage kochia populations while reducing the risk of evolving resistance to additional herbicides (Gressel and Segel 1990).

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CHAPTER 2

MONITORING THE RESPONSE OF KOCHIA TO DICAMBA AT LOCATIONS EXHIBITING ATYPICAL INJURY

ABSTRACT

Kochia from isolated locations in Colorado, Kansas, Montana, and Nebraska was injured less than 88% by application of 140 g ae ha⁻¹ dicamba. Kochia response at these locations varied spatially. Kochia injury was lowest for samples collected from within an agronomic area while adjacent agronomic areas and waste areas seldom expressed less than 88% injury. Kochia injury also varied among the areas sampled within a field, the plants sampled within an area, and the progeny of an individual plant. The number of progeny surviving an individual dicamba rate varied from 0% to 100% survival. Kochia injury increased as dicamba rate increased from 70 g ae ha⁻¹ to 560 g ae ha⁻¹ dicamba. Seven locations had kochia that expressed less than 96% injury from 560 g ae ha⁻¹ dicamba, but these locations were susceptible to atrazine or chlorsulfuron.

INTRODUCTION

Kochia has been a weed in dryland and irrigated cropping systems of the Northwest regions of North America for many years causing yield losses and reducing harvest efficiency (Black et al. 1969; Buhler et al. 1985; Dexter 1982; Durgan et al. 1990; Weatherspoon and Schweitzer 1971). Since the mid-1960's, the synthetic, auxinic herbicide dicamba has been used to manage kochia populations. Due to its effectiveness on taller broadleaved weeds, soil and foliar activity, and reasonable cost compared to other herbicides, dicamba has been used alone or in tank-mixes year after year in many locations with no observable decline in its efficacy.

In 1993, a sample of kochia seed was delivered to the Colorado State University Weed Science Laboratory from a field near Morrill, NE. Kochia that produced this seed was reported to not be responding to dicamba application. This was after many years of annual auxinic herbicide treatment. Plants were grown from this seed sample in the greenhouse and evaluated for response to dicamba. It was found that a portion of the plants survived treatment with 1120 g ae ha⁻¹ dicamba. Interest in the possibility of resistance evolution in kochia to such an important herbicide prompted the screening of 200 kochia samples collected from across Colorado. This screening indicated that kochia injury from dicamba varied but samples were generally very susceptible. Eight of the collections expressed injury that was considered low at 280 g ae ha⁻¹ dicamba. It

was decided that continued investigation was necessary to understand the circumstances surrounding this issue.

A guide for determining atypical kochia control needed to be established. There was a possibility of misrepresenting the expected injury from dicamba since kochia had always exhibited variations in response to this herbicide. Sandoz Agro, Inc. compiled internal and published data from 1963 to 1994 to establish a set of baseline expectations. Baseline data were compiled using information from as many as 59 studies for each dicamba rate. It was found that kochia control with dicamba has tended to fluctuate by year. Average control for rates of dicamba (g ae ha^{-1}) were as follows: 82% at 70 g, 88% at 140 g, 92% at 280 g, and 96% at 560 g. These were used as the typical response of kochia to dicamba.

It was determined that kochia experiences a high degree of out-breeding (Guttieri et al. 1998). This could be a contributing reason why reduced kochia injury has not shown up to a larger extent after 30 years of dicamba use. If the reduced injury trait were recessive in kochia, evolution models would predict a very slight chance of increasing the resistant allele proportion in an out-crossing population (Jasieniuk et al. 1996). Also, susceptible plants that emerged after the dicamba soil residual effect wore off could contribute susceptible alleles to dilute non-susceptible alleles if flowering of the two plants occurred simultaneously (Everitt et al. 1983; Bell et al. 1972).

Experiments were established to gain a better understanding of the circumstances surrounding these issues with dicamba. Continued random

location sampling was replaced by a system that took advantage of County Extension personnel and industry representatives to pinpoint locations where kochia survived herbicide treatment. Experiments were designed to determine the response of kochia to three herbicides, examine the spatial structure of atypical dicamba response, and monitor temporal fluctuations in populations that may be used to gain a better understanding of kochia population dynamics.

MATERIALS AND METHODS

Lack of Control Locations.

Design. Experiments were conducted under greenhouse conditions. Temperature ranged from 25° C to 29° C with a day:night cycle of 16:8 hr. Sodium halide lamps supplemented day length. The experimental design was a randomized complete block with three replications. A factorial layout with two categories, kochia collection by herbicide, was used to examine treatment combinations. Eighty kochia collections from 19 agricultural locations were used as levels of kochia collection. These collections were acquired over a three-year period from 1996 to 1998. Six herbicide treatments were included in the experiment.

Plant Material. Kochia seeds used in this study were obtained from fields identified by industry personnel as having kochia that exhibited an unexplained response to dicamba. When possible, seed was collected from several areas in the cropping area as well as from areas adjacent to the field. At each sample

area, seed was collected from ten plants. The seed from all ten plants was placed in a coin envelope and labeled with the field location and sample area. A map was constructed at each location to identify the spatial relationships among the samples.

If plants would not be available at the end of the season, vegetative plants or plant cuttings were collected from areas of the field. These plants were delivered to the Colorado State University - Weed Research Facility, Fort Collins, Colorado 80523-1177. Plants and plant cuttings were potted in Metro-Mix 350 potting soil (Scotts-Sierra Horticultural Products Company, 14111 Scottslawn Rd., Marysville, Ohio 43041). Rootone® (Green Light Co., P.O. Box 17985, San Antonio, Texas 78217-1985) rooting hormone was used to improve transplant success. A small amount of rooting hormone was applied to plant roots before potting. The soil of whole plant transplants was kept moist. Plant cuttings were trimmed at approximately 8 cm from the tip. Two to three of the lowest leaves were removed and this area was dipped in rooting hormone. Cuttings were then planted in potting mix that was kept damp. Leaves of cutting transplants were regularly misted with water during the first week of establishment. Transplants remained in the greenhouse until seed was mature.

The experimental unit was a flat (model A 12-01, East Jordan Plastics, Inc., P.O. Box 575, East Jordan, Minnesota 49727) 10 cm wide by 15 cm long by 5 cm deep. The flat was filled with Sunshine® potting mix #1 (Sun Gro Horticulture, Inc., 15831 N.E. 8th Street, Bellevue, Washington 98008) to 0.5 cm of the top. Approximately 30 seeds of one kochia sample were distributed on top

of the potting mix across each flat area. Potting mix was sprinkled over the seeds so they were buried by approximately 0.3 cm of potting mix. Flats were kept moist with overhead irrigation and fertilized every two weeks with 100 ml of Miracle-Gro® (Scotts Miracle-Gro Products, Inc., P.O. Box 888, Port Washington, New York 11050) solution. The fertilizer solution was prepared by dissolving 3.5 g granular fertilizer (analysis 15-30-15) in 3.8 L water.

Two days before herbicide application, plants were thinned from the flats to reduce interference between plants and maximize spray coverage. Flats were thinned to a population of 20 plants except in instances where leaving that many plants would compromise adequate spray coverage. The actual number of plants that received herbicide treatment was recorded for each flat. Kochia plants were 3 cm to 7 cm tall at the time of herbicide application.

Herbicide Treatments. Herbicide treatments were administered with a chain-driven chamber sprayer. The sprayer was calibrated to deliver 94 L ha⁻¹ to 112 L ha⁻¹ by compressed air through an 8001 even flat fan tip. Herbicide treatments included an untreated control; 140 g ae ha⁻¹, 280 g ae ha⁻¹, and 560 g ae ha⁻¹ dicamba; 26 g ai ha⁻¹ chlorsulfuron with 0.25% V V⁻¹ non-ionic surfactant; and 1400 g ai ha⁻¹ atrazine with 3.5 L ha⁻¹ crop oil.

Evaluation. Plants were evaluated 28 days after herbicide application. This evaluation assessed plant injury. Each plant in a flat was given a score for percent injury from 0% (no injury) to 100% (complete death) compared to control plants. Scores for all plants in a flat were averaged to provide a single rating of plant injury.

Data Analyses. Data were analyzed using procedures for mixed effects models in SAS programming software (SAS Institute 1989). Standard diagnostic inquiries indicated no violations of the assumptions underlying analysis of variance testing. Mean separation was performed using single degree of freedom contrasts via the least squares means (lsmeans) statement in the mixed effects procedure of SAS (SAS Institute 1989).

Yearly Monitoring Locations.

Design. Experiments were conducted under greenhouse conditions. Temperature ranged from 25° C to 29° C with a day:night cycle of 16:8 hr. Sodium halide lamps supplemented day length. The experimental design was a randomized complete block with three replications. A factorial layout with two categories, kochia collection by herbicide, was used to examine treatment combinations. Ninety kochia collections were used as levels of kochia collection, and three herbicide treatments were included in the experiment. The experiment was conducted for each of five locations during 1996, 1997, and 1998 when kochia was present at the site.

Plant Material. Kochia seeds used in this study were obtained from fields identified by previous experiments as having kochia that exhibited lower than expected control with dicamba. Two locations were in Colorado. One of these was south of Burlington, Colorado. This site was located southwest of the intersection of County Road N and County Road 53 in Kit Carson County. The other Colorado site is located near Longmont, Colorado, southwest of the

intersection of Pike Road and County Road 1 in Boulder County. There were three locations in Nebraska. The first of these was west of Culbertson, Nebraska. The site was 4 km due west of the intersection of Highway 6 and Highway 34 on the north side of the road in Hitchcock County. The second Nebraska location was south of Henry, Nebraska. The site was southwest of the intersection of Holloway Road and Road D in Scotts Bluff County. The final site was near Morrill, Nebraska, which is also in Scotts Bluff County. This site was located north of the intersection of Highway 92 and County Road 10.

Each year, the sites were visited to collect kochia seed from plants that were still rooted. Seed was collected from 50 plants within the agricultural area of the designated location and from 40 plants in the site's waste areas and adjacent fields. Plants were sampled from all areas of the site but depended on the distribution of kochia at the site. Plants were randomly selected but not less than 25 m from other sampled plants. Seed from each plant was placed in a separate coin envelope and labeled with the field site and sample area. A map was constructed at each location to identify the spatial relationships among the sampled kochia plants.

The experimental unit was a flat (model A 12-01, East Jordan Plastics, Inc., P.O. Box 575, East Jordan, Minnesota 49727) 10 cm wide by 15 cm long by 5 cm deep. The flat was filled with Sunshine® potting mix #1 (Sun Gro Horticulture, Inc., 15831 N.E. 8th Street, Bellevue, Washington 98008) to 0.5 cm of the top. Approximately 30 seeds of one kochia sample were distributed on top of the potting mix across each flat area. Potting mix was sprinkled over the

seeds so they were buried by approximately 0.3 cm of potting mix. Flats were kept moist with overhead irrigation and fertilized every two weeks with 100 ml of Miracle-Gro® (Scotts Miracle-Gro Products, Inc., P.O. Box 888, Port Washington, New York 11050) solution. The fertilizer solution was prepared by dissolving 3.5 g granular fertilizer (analysis 15-30-15) in 3.8 L water.

Two days before herbicide application, plants were thinned from the flats to reduce interference between plants and maximize spray coverage. Flats were thinned to a population of 20 plants except in instances where leaving that many plants would compromise adequate spray coverage. The actual number of plants that received herbicide treatment was recorded for each flat. Kochia plants were 2 cm to 7 cm tall at the time of herbicide application.

Herbicide Treatments. Herbicide treatments were administered with a chain-driven chamber sprayer. The sprayer was calibrated to deliver from 94 L ha⁻¹ to 103 L ha⁻¹ by compressed air through an 8001 even flat fan tip. Herbicide treatments included a control, 70 g ae ha⁻¹ dicamba, and 140 g ae ha⁻¹ dicamba.

Evaluation. Plants were evaluated 28 days after herbicide application. This evaluation assessed plant injury. Each plant in a flat was given a score for percent injury from 0% (no injury) to 100% (complete death) compared to control plants. Scores for all plants in a flat were averaged to provide a single rating of plant injury. The individual plant injury rating was also used to generate the proportion of plants in each flat that were considered resistant to dicamba treatment. Plants receiving a score of less than 40% were considered resistant.

Data Analyses. Each location was analyzed separately because of location interactions and unequal data sets. Data were analyzed using procedures for mixed effects models in SAS programming software (SAS Institute 1989). Resistant plant proportions were transformed by the arcsine of the square root of data decimal values. This was done because of the binomial nature of these data. Standard diagnostic inquiries indicated no violations of the assumptions underlying analysis of variance testing. Mean separation was performed using single degree of freedom contrasts via the least squares means (lsmeans) statement in the mixed effects procedure of SAS (SAS Institute 1989). Non-transformed resistance proportions were presented in text and tables; however, all statistical analyses were performed with transformed proportion data.

RESULTS AND DISCUSSION

Lack of Control Locations.

From 1994 to 1998, kochia samples from locations in Colorado, Kansas, Montana, and Nebraska were subject to herbicide screening at Colorado State University. The number of locations that submitted samples varied from one location in 1998 to as many as 16 locations in 1995 (Table 2.1). Individual locations were not commonly sampled from more than one year unless the field was of particular interest due to the response to dicamba. Managers of the locations were invited to submit samples if circumstances resulted in another

unexplained lack of control, but the absence of sampling in subsequent years does not remove dicamba from suspicion as an agent of the location's lack of kochia control. Altered management practices, application techniques, or expectations may have kept managers from calling attention to their location again.

Experiments in the greenhouse identified several locations that contained areas where kochia was not responding as susceptible to dicamba. Kochia from these areas tended to differ in the proportion of surviving plants but a greater influence on the injury rating appeared to be the degree of vigor and growth following dicamba application. All plants showed immediate symptoms of epinasty, tissue rigidity, and in many instances partial or complete tissue death. Plants from some locations, however, were more likely to have healthy growth at the time of evaluation. Two whole plant responses to dicamba were identified that seemed to have different physiologic causes. The first response type was apical meristem death followed by prolific stem differentiation from axillary meristem regions. This "witches-brooming" biotype was common to only one location near Morrill, NE. The second response type was a continuum of plant injury with no localized tissue death. Plant response in this biotype ranged from slight arresting of normal growth to severely chlorotic plants that eventually produced single branches from one or a few leaf axils. Lower kochia injury ratings were again strongly related to new tissue growth, but the growth of the second biotype following dicamba application was similar to normal kochia plant architecture.

Due to the differences in kochia response, it was speculated that more than one mechanism might be influencing kochia injury responses. Examination of several kochia populations revealed that this might not be true. While the Morrill location showed the greatest occurrence and degree of witches-brooming, other locations have expressed this response in greenhouse experiments. Likewise, the Morrill location did not exclusively produce the witches-brooming response. Lower rates of dicamba tended to elicit the continuum response from the same collection of seed that originally demonstrated the witches-brooming response. Two separate mechanisms might be responsible for these responses, however, the responses could be different manifestations of the same physiologic process.

Fifteen of the 49 locations screened showed evidence of lower kochia injury from dicamba than baseline data would suggest (Table 2.1). Two of these locations appeared in two years. All kochia samples from the remaining 32 locations, over two-thirds of the total number of locations, were susceptible to dicamba at rates from 140 g ae ha⁻¹ to 560 g ae ha⁻¹. The locations were sampled because kochia management programs, which included dicamba applications, did not control kochia, but kochia progeny from these locations were susceptible to dicamba applications in the greenhouse.

Among the 24 locations from 1996 to 1998, eight locations contained at least one sample that had a lower injury rating than the baseline data for dicamba treatments when $\alpha = 0.1$ was used as the selection criterion (Table 2.2). Reduction in kochia response to dicamba was not consistent across dicamba

rates at each location, however. For instance, at location #2 of Yuma, CO, kochia injury was only different from dicamba baseline data at 140 g ae ha⁻¹ dicamba (df (1, 100), $t = 2.33$, $P = 0.022$). At higher dicamba rates, all kochia samples from this location responded similar to the baseline data (df (1, 100), $t = 0.69$, $P = 0.49$). This response to rate increase was also observed in many of the 1994 and 1995 locations. At other locations such as Cimerron, KS, kochia injury was lower than baseline data at 140 g ae ha⁻¹ (df (1, 100), $t = 3.07$, $P = 0.0027$) and 280 g ae ha⁻¹ dicamba (df (1, 100), $t = 2.05$, $P = 0.043$) but similar to baseline data when the rate was increased to 560 g ae ha⁻¹ dicamba (df (1, 98), $t = 1.24$, $P = 0.22$). Samples from five locations were different from baseline data at all rates of dicamba. These represent the most difficult to control kochia populations with injury ratings as low as 70% at 560 g ae ha⁻¹ dicamba, 26 percentage points lower than baseline data (df (1, 99), $t = 22$, $P = 0.0001$).

As stated earlier, the locations in Table 2.2 contained at least one sample area with low kochia injury rating. Many of the sample areas within a location were susceptible to all dicamba rates. This was explored by analyzing each sample area within a location. Two locations, Cimerron, KS, and McCook, NE, were presented as examples of dicamba response differences among areas at a location. At each location, samples were collected from the primary field area, in the field, and from external crop and non-crop areas, out of the field. This position effect had the greatest influence on the 140 g ae ha⁻¹ dicamba treatment (Table 2.3). For this treatment, injury was 16 percentage points lower for samples collected inside the field than for samples collected outside the field at

Cimerron ($df (1, 109)$, $t = 2.2$, $P = 0.030$). There was 18 percentage points difference between positions at McCook with sample areas in the field again being lower ($df (1, 66)$, $t = 2.25$, $P = 0.028$).

Eight areas were sampled from Cimerron (Table 2.4, Figure 2.1). Only three of these areas differed from dicamba baseline data ($df (1, 15)$, $t = 2.12$, $P = 0.051$, minimum difference), all of which were collected from inside the field perimeter. All areas, whether outside or inside the field, were not different from baseline data for 560 g ae ha⁻¹ dicamba ($df (8, 16)$, $F = 1.83$, $P = 0.15$).

Sixteen areas were sampled from McCook (Table 2.5, Figure 2.2). At this location, one of the samples from outside the field perimeter had an injury rating lower than baseline data for 140 g ae ha⁻¹ dicamba ($df (1, 32)$, $t = 2.86$, $P = 0.0074$). The difference was not consistent at the other rates of dicamba for this sample area ($df (1, 32)$, $t = 0.84$, $P = 0.41$). Six of the eight primary field samples exhibited lower kochia injury than the baseline data at one or more dicamba treatments ($df (1, 32)$, $t = 1.93$, $P = 0.063$, minimum difference). While response to the three dicamba rates varied depending on sample area, kochia from three of the sample areas responded lower than baseline data at all three dicamba treatments. These sample areas were clustered in one area of the field relative to other field sample areas (Figure 2.2).

Kochia injury from atrazine and chlorsulfuron was also investigated and locations with samples that deviated from 100% injury ($\alpha = 0.1$) were identified (Table 2.2). Locations were either highly susceptible to atrazine or contained large proportions of resistant seed. Atrazine resistance at seven locations

resulted in injury ratings that were lower than baseline data by 73% or more (df (1, 100), $F = 10.66$, $P = 0.0001$). Kochia response to chlorsulfuron treatment was more intermediate than response to atrazine. Injury was still reliant on the proportions of susceptible and resistant seed, but there were more samples that contained relatively equal amounts of chlorsulfuron-susceptible and -resistant seed compared to atrazine resistance. Seven locations had chlorsulfuron resistance that resulted in kochia injury at least 27% lower than the baseline data (df (1, 99), $F = 2.40$, $P = 0.018$).

Mechanisms of resistance to triazine (atrazine) and sulfonylurea (chlorsulfuron) herbicides have been identified and discussed by other authors (Holt et al. 1993; Saari et al. 1990). While a mechanism for resistance to dicamba has not been identified, the mechanism, assuming it exists, would probably differ from mechanisms of resistance to triazine and sulfonylurea herbicides due to differences in symptomology of the three herbicide classes. Even though the mechanisms of resistance differ, multiple-resistance is a possibility given the wide use of these herbicide classes. Resistance to each herbicide would develop independently of the others, but resistance to multiple herbicides could develop concurrently. Table 2.2 contained seven locations that demonstrated multiple-herbicide resistance evolution. Kochia populations with resistance to atrazine and chlorsulfuron demonstrated correlation due to the dichotomy of resistance expression in kochia (Pearson's $r = 0.45$, $P = 0.0001$, Figure 2.3). It was not very common, however, to evaluate collections with resistance to both of these herbicides. Kochia injury from dicamba and atrazine

(Pearson's $r = -0.07$, $P = 0.25$, Figure 2.4) and from dicamba and chlorsulfuron (Pearson's $r = 0.08$, $P = 0.22$, Figure 2.5) was not correlated. In general, the injury obtained from one herbicide was independent of the injury obtained from any other herbicide.

Yearly Monitoring Locations.

Of the five locations that were monitored over three years, the Henry, NE, location had the lowest average injury score from dicamba applications, 70% (df (1, 6384), $t = 15.41$, $P = 0.0001$). Average injury was 79% at both the Culbertson, NE, and Morrill, NE locations (df (1, 6384), $F = 0.20$, $P = 0.84$), which was slightly lower than the 81% injury from Longmont, CO, samples (df (1, 6384), $F = 2.74$, $P = 0.0062$). Burlington, CO, at 89% average injury, had the highest injury rating (df (6384), $F = 14.04$, $P = 0.0001$). Response to main effects within each location differed greatly, which resulted in several interactions with main effects. Since it was known at the outset of the experiment that the locations differed greatly in response to dicamba, the locations were analyzed separately for comparison of main and interaction effects.

Values for kochia injury and percentage resistant plants were highly correlated in a negative relationship (Pearson's $r = -0.86$, $P = 0.0001$, Figure 2.6). This is a reasonable result due to the method of resistance determination. Since all kochia plants treated with dicamba demonstrated injury, there was no clear dichotomy of susceptible and resistant plant responses. On the contrary, plants that survived dicamba application exhibited varying degrees of injury even

among progeny from an individual field plant sample. A division was created at 40% injury. Plants with injury lower than 40% were classified as resistant because symptoms were minor. Plants in this category had epinastic symptoms but did not turn chlorotic or manifest localized tissue death. As average injury rating decreased the range of progeny response overlapped further into the resistant category, which resulted in the strong negative correlation between the two values. Both data sets were analyzed with similar results; therefore, only injury data will be discussed for most effects. Additional tables that described main effects on resistance were included in *Appendix 2.D*.

Kochia injury in greenhouse experiments was affected by the year of sampling from Burlington, Henry, and Longmont (Table 2.6). Kochia samples from Culbertson (df (1, 166), $t = 2.85$, $P = 0.093$) and Morrill (df (1, 168), $t = 3.37$, $P = 0.068$) did not differ across years sampled. Samples collected in 1996 tended to have higher injury ratings than samples in the other years. This was confirmed with samples from Burlington (df (1, 112), $t = 3.53$, $P = 0.0006$) and Longmont (df (1, 247), $t = 9.11$, $P = 0.0001$) that had the greatest average injury in 1996. While injury declined from 1996 to 1997, samples in 1998 tended to be more injured than 1997 samples. In particular, differences in the Burlington (df (1, 108), $t = 6.38$, $P = 0.0001$) and Henry samples (df (1, 120), $t = 5.28$, $P = 0.0001$) support greater average injury in 1998 than 1997. Given the tendencies of all locations over the years sampled, it appeared that injury response formed a trough. Overall, there was a decrease in kochia injury from dicamba treatments, but the second sample year was lower than the first or third years.

The percentage of resistant plants was more difficult than injury to characterize over years. There was a tendency at many of the locations for more kochia plants to be categorized as resistant at the end of the three years of sampling than at the beginning (Table 2.7). Although inflated by low initial percentages, the portion of plants from Morrill and Longmont samples that expressed resistant tendencies increased substantially, four- and eight-fold respectively ($df (1, 157)$, $F = 28$, $P = 0.0001$, and $df (1, 247)$, $t = 9.09$, $P = 0.0001$). Samples from the Burlington and Culbertson locations did not differ from the first to the last sample year, however, and samples from Henry contained fewer resistant plants in 1998 than in 1997 ($df (1, 73)$, $F = 6.74$, $P = 0.011$).

Information about the percentage of resistant plants (Table 2.7) was contrasted to a summary of plant death (Table 2.8). Dead plants appeared to comprise a much larger category than resistant plants for the majority of locations within a year. With the exception of 1997 samples from Henry, dead kochia was at least three times more common than resistant kochia for each location by year combination. Many plants were in visible decline at the time of evaluation and would probably have died given more time. Inclusion of these plants with the dead plants increased the number of susceptible progeny by 50% to 100% over the number of dead plants alone (Table 2.9). Except for 1997 samples from Henry, the majority of progeny, at least 52% and at most 97%, from each location and year was susceptible to dicamba treatments.

The effect of dicamba rate was very consistent across locations (Table 2.10). Kochia injury increased as dicamba rate increased. Burlington samples had the smallest increase, 6 percentage points, in injury score as rate increased from 70 g ae ha⁻¹ to 140 g ae ha⁻¹ dicamba (df (1, 224), $F = 110$, $P = 0.0001$). At the Burlington location, kochia was very susceptible to 70 g ae ha⁻¹ dicamba, 88% injury. Kochia from the Henry location, which had the least susceptibility and subsequently the highest level of resistance, still responded to increased dicamba rate by exhibiting more injury, 67% injury at the low rate versus 77% injury at the high rate (df (1, 164), $F = 240$, $P = 0.0001$). Kochia from the Henry location, however, appeared to deviate from baseline data of 82% and 88% injury for 70 g ae ha⁻¹ and 140 g ae ha⁻¹ dicamba, respectively.

Field position of the plant sample also had a large effect on kochia injury (Table 2.11). Progeny from plants in the primary field area had lower injury ratings than progeny from plants collected on the outside of the field perimeter for four of the five locations. The minimum difference among these locations was an increase from 76% injury in the field to 83% injury out of the field (df (1, 168), $F = 15.24$, $P = 0.0001$). The one location where sample position did not influence kochia injury was Burlington (df (1, 226), $F = 3.38$, $P = 0.067$). Samples from positions outside of the field at all locations had values of kochia injury that were consistent with baseline data.

Individuality of plants was examined by the random effect of plant within year and position. Within position, the plant sampled greatly influenced the injury rating of progeny in the greenhouse experiments (df (175, 176), $F = 6.79$, $P =$

0.0001, minimum influence). This meant that the general population of kochia within field position contained plants that produced progeny with a wide range of injury response to dicamba and that this effect is more consistent within a plant than among plants. Maps of the locations for each year demonstrated the intermixed placement of plants that produced progeny that were largely susceptible to dicamba and those that produced fewer susceptible progeny (Figure 2.7 – Figure 2.11).

CONCLUSIONS

In 1993, an unsolicited kochia sample that was difficult to control with dicamba sparked interest in dicamba efficacy on kochia. This Morrill, NE, sample prompted a preliminary study of 200 Colorado kochia collections. This study indicated overwhelming susceptibility to dicamba in the general kochia population but led to the establishment of a protocol to investigate locations of unexplained kochia response to management practices that included dicamba.

In a period of five years, nearly 50 lack of control locations have been tested. Two-thirds of the locations were susceptible to dicamba treatments at all rates. The other one-third demonstrated varying degrees of dicamba injury, which were lower than expected for some of the dicamba rates used. Of all the kochia locations tested, over 250 total, 25 have demonstrated reduced dicamba injury. Most of these 25 locations were not randomly selected, but rather, they were locations from an area that included four states and were highlighted due to

kochia management difficulties. They represented the extreme cases of management concerns with dicamba in the region.

Kochia that exhibited lower injury from dicamba applications did exist at a relatively small number of locations. From all monitoring, it appeared that only six locations demonstrated consistent and severe kochia management concerns when dicamba was considered the primary management tool. Response of kochia at these locations varied among samples. Kochia with reduced injury was generally restricted to the interior of the field of concern and often sequestered to a portion of the field area.

Besides the continued usefulness of dicamba at the majority of these locations, triazine and sulfonyleurea herbicides remain effective management tools as well. These experiments demonstrated that each field is unique, especially in regard to dicamba response. This emphasized the need for testing individual locations where specific response to herbicides was questionable.

Five locations identified through this process that had atypical kochia response to dicamba were selected for additional monitoring. These experiments strengthened the conclusion from the lack of control locations that reduced kochia response to dicamba was localized. Lower injury ratings were almost exclusively from samples collected on the inside of the field perimeter, but not all kochia in an area produced progeny that responded similarly to dicamba. Kochia response varied among progeny within a plant as well as between plants, but all kochia positively responded to increased dicamba rate by expressing higher injury.

Portions of kochia populations at all locations expressed susceptibility to dicamba even though kochia had received yearly dicamba applications for 20 years or more. This fact and the differences in the response of kochia offspring between and within parental plants may have been due to kochia genetics and plant biology. More investigations into the genetics and heritability of the atypical kochia response to dicamba need to be performed to clarify the significance of current observations and results. Out-crossing may prevent the rapid increase of resistant plants in the population if the trait was recessive. The variable responses within progeny from a plant, however, may have indicated that the trait was regulated by multiple loci or perhaps by incomplete dominance. Also, since dicamba has a short soil residual effect, susceptible kochia plants emerged in the middle of the season and were able to contribute pollen and seed to the genetic diversity of the population.

Year of sampling had an inconsistent effect on injury rating. This could have been due in part to altered management practices. Evidence of reduced plant injury at the Burlington location was essentially lost over the course of monitoring from 1994 to 1998, which was believed to be due in part to changes in herbicide program. At the Morrill location, however, changes in management and prevention of seed production did not affect the overall location response.

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Table 2.1. Number of locations requesting greenhouse testing of *K. scoparia*.

Year	Locations		
	Number Tested	States Involved	Low Dicamba Effect ^a
1994 ^b	9	CO, KS, NE	5
1995 ^b	16	KS, MT, NE	4
1996	15	CO, MT	3
1997	8	KS, MT, NE	4
1998	1	NE	1

^a Locations with at least one sample area that did not respond as susceptible to one or more dicamba treatments.

^b Locations in 1994 (*Appendix 2.B*) and 1995 (*Appendix 2.C*) were investigated under a different experimental design and were not analyzed with the other years.

Table 2.2. *K. scoparia* locations, identified by lowest sample area injury rating 28 days after treatment, that did not respond as susceptible to herbicide treatment.

Location	KCHSC ^a Injury				
	Herbicide ^b Treatment (g ai ha ⁻¹)				
	Dicamba (140)	Dicamba (280)	Dicamba (560)	Chlorsulfuron (26)	Atrazine (1400)
	(%)				
Baseline Data	88	92	96	100	100
1996					
Cope, CO	ns ^c	ns	ns	35 ± 5 ^d	9 ± 5
Scobey, MT	ns	ns	ns	72 ± 5	ns
Sterling, CO	68 ± 5	67 ± 3	70 ± 3	ns	ns
Yuma, CO (1)	ns	ns	ns	ns	3 ± 5
Yuma, CO (2)	72 ± 5	ns	ns	ns	3 ± 5
Yuma, CO (3)	72 ± 5	ns	ns	ns	2 ± 5
Yuma, CO (4)	ns	ns	ns	ns	27 ± 5
Rep. 76, MT	ns	ns	ns	73 ± 5	ns
1997					
Big Bow, KS	ns	ns	ns	0 ± 5	0 ± 5
Cimerron, KS	61 ± 5	78 ± 3	ns	47 ± 5	20 ± 5
Colby, KS	71 ± 5	78 ± 3	90 ± 2	ns	ns
Forsyth, MT	76 ± 5	84 ± 3	87 ± 2	ns	ns
Henry, NE	60 ± 5	72 ± 3	76 ± 2	64 ± 5	ns
1998					
McCook, NE	48 ± 5	68 ± 3	88 ± 2	13 ± 5	ns
ANOVA ^e					
df	49, 100	49, 100	49, 98	49, 99	49, 100
F	6.93	7.56	8.64	62	66
P>F	0.0001	0.0001	0.0001	0.0001	0.0001

^a KCHSC, *Kochia scoparia*.

^b Spray solution adjuvants were included as indicated in the Materials and Methods section.

^c ns, not significant. The combination of location and treatment was not different from baseline performance on susceptible *K. scoparia* according to least squares means contrasts for $\alpha = 0.1$. Locations not included in the table were not different from baseline performance for any of the treatments.

^d Means $\pm$ standard errors for combinations of location and treatment that were different from baseline performance of that treatment according to least squares means contrasts for $\alpha = 0.1$.

^e Analysis of variance table for each treatment as a main effect.

Table 2.3. Position effect on *K. scoparia* injury 28 days after treatment with 140 g ae ha⁻¹ dicamba averaged across sample areas.

Position ^a	KCHSC ^b Injury	
	Location	
	Cimerron, KS	McCook, NE
	(%)	
Out	88 ± 5 a ^c	86 ± 4 a
In	72 ± 5 b	68 ± 4 b
ANOVA ^d		
df	1, 109	1, 14
F	5.24	5.40
P>F	0.024	0.036

^a Position was in reference to the cropping area. Out, adjacent field or non-crop area; In, primary crop area.

^b KCHSC, *Kochia scoparia*.

^c Means ± standard errors in each column followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^d Analysis of variance for the main effect.

Table 2.4. *K. scoparia* sample areas, identified by injury rating 28 days after treatment, that did not respond as susceptible to dicamba rate, Cimerron, KS, 1997.

KCHSC ^a Sample		KCHSC Injury		
Area ^b	Position ^c	Dicamba Rate		
		140 g ae ha ⁻¹	280 g ae ha ⁻¹	560 g ae ha ⁻¹
		(%)		
Baseline Data		88	92	96
1	Out	ns ^d	ns	ns
2	Out	ns	ns	ns
3	Out	ns	ns	ns
4	Out	ns	ns	ns
5	In	ns	ns	ns
6	In	67 ± 5 ^e	ns	ns
7	In	ns	83 ± 3	ns
8	In	61 ± 5	78 ± 3	ns
ANOVA ^f				
df		8, 16	8, 15	8, 16
F		4.32	5.41	1.83
P>F		0.0062	0.0025	0.15

^a KCHSC, *Kochia scoparia*.

^b Refer to map in Figure 2.1.

^c Position was in reference to the cropping area. Out, adjacent field or non-crop area; In, primary crop area.

^d ns, not significant. The combination of area and treatment was not different from baseline performance on susceptible *K. scoparia* according to least squares means contrasts for $\alpha = 0.1$.

^e Means $\pm$ standard errors for combinations of area and treatment that were different from baseline performance of that treatment according to least squares means contrasts for $\alpha = 0.1$.

^f Analysis of variance table for each treatment as a main effect.

Table 2.5. *K. scoparia* sample areas, identified by injury rating 28 days after treatment, that did not respond as susceptible to dicamba rate, McCook, NE, 1998.

KCHSC ^a Sample		KCHSC Injury		
Area ^b	Position ^c	Dicamba Rate		
		140 g ae ha ⁻¹	280 g ae ha ⁻¹	560 g ae ha ⁻¹
		(%)		
Baseline Data		88	92	96
1	Out	ns ^d	ns	ns
2	Out	ns	ns	ns
3	Out	ns	ns	ns
4	Out	ns	ns	ns
5	Out	ns	ns	ns
6	Out	69 ± 5 ^e	ns	ns
7	Out	ns	ns	ns
8	Out	ns	ns	ns
9	In	ns	ns	ns
10	In	ns	ns	ns
11	In	75 ± 5	ns	ns
12	In	69 ± 5	ns	ns
13	In	63 ± 5	68 ± 4	ns
14	In	48 ± 5	78 ± 4	88 ± 2
15	In	71 ± 5	78 ± 4	90 ± 2
16	In	62 ± 5	79 ± 4	88 ± 2
ANOVA ^f				
df		16, 32	16, 32	16, 32
F		7.56	4.53	4.82
P>F		0.0001	0.0001	0.0001

^a KCHSC, *Kochia scoparia*.

^b Refer to map in Figure 2.2.

^c Position was in reference to the cropping area. Out, adjacent field or non-crop area; In, primary crop area.

^d ns, not significant. The combination of area and treatment was not different from baseline performance on susceptible *K. scoparia* according to least squares means contrasts for $\alpha = 0.1$.

^e Means $\pm$ standard errors for combinations of area and treatment that were different from baseline performance of that treatment according to least squares means contrasts for $\alpha = 0.1$.

^f Analysis of variance table for each treatment as a main effect.

Table 2.6. Year effect on *K. scoparia* injury 28 days after treatment for each location averaged across positions and dicamba rates.

Year	KCHSC ^a Injury				
	Location				
	Burlington	Culbertson	Henry	Longmont	Morrill
	(%)				
1996	97 ± 1 a ^b	na ^c	na	93 ± 1 a	82 ± 1 a
1997	84 ± 1 c	78 ± 1 a	66 ± 2 b	77 ± 1 b	na
1998	92 ± 1 b	81 ± 1 a	78 ± 2 a	77 ± 1 b	78 ± 1 a
ANOVA ^d					
df	2, 114	1, 166	1, 120	2, 247	1, 168
F	44	2.85	28	55	3.37
P>F	0.0001	0.093	0.0001	0.0001	0.068

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^c na, not applicable. Seed was not available from the location.

^d Analysis of variance table for the main effect.

Table 2.7. Year effect on *K. scoparia* resistance expression for each location averaged across positions and dicamba rates.

Year	KCHSC ^a Resistance ^b				
	Location				
	Burlington	Culbertson	Henry	Longmont	Morrill
	(%)				
1996	0 ± 1 b ^c	na ^d	na	1 ± 1 b	3 ± 1 b
1997	4 ± 1 a	8 ± 2 a	18 ± 2 a	10 ± 1 a	na
1998	1 ± 1 b	10 ± 2 a	11 ± 2 b	8 ± 1 a	12 ± 1 a
ANOVA ^e					
df	2, 234	1, 148	1, 73	2, 202	1, 157
F	13.25	0.69	6.74	21	28
P>F	0.0001	0.41	0.011	0.0001	0.0001

^a KCHSC, *Kochia scoparia*.

^b Plants that expressed less than 40% injury at 28 days after treatment were classified as resistant.

^c Means ± standard errors in each column followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^d na, not applicable. Seed was not available from the location.

^e Analysis of variance table for the main effect.

Table 2.8. Year effect on *K. scoparia* death for each location averaged across positions and dicamba rates 28 days after treatment.

Year	KCHSC ^a Death				
	Location				
	Burlington	Culbertson	Henry	Longmont	Morrill
	(%)				
1996	62 ± 3 a ^b	na ^c	na	53 ± 2 a	27 ± 2 b
1997	45 ± 2 b	30 ± 2 b	20 ± 2 b	33 ± 2 b	na
1998	56 ± 2 a	43 ± 2 a	35 ± 2 a	23 ± 2 c	37 ± 2 a
ANOVA ^d					
df	2, 56	1, 117	1, 100	2, 173	1, 134
F	12.68	26	31	78	15.39
P>F	0.0001	0.0001	0.0001	0.0001	0.0001

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$. Mean separations were based on statistics that used data transformed by the arcsine of the square root of decimal values.

^c na, not applicable. Seed was not available from the location.

^d Analysis of variance table for the main effect when transformed data were used.

Table 2.9. Year effect on highly susceptible *K. scoparia* for each location averaged across positions and dicamba rates 28 days after treatment.

Year	Highly Susceptible ^a KCHSC ^b				
	Location				
	Burlington	Culbertson	Henry	Longmont	Morrill
	(%)				
1996	97 ± 2 a ^c	na ^d	na	86 ± 2 a	59 ± 2 a
1997	66 ± 2 c	52 ± 2 b	33 ± 3 b	55 ± 2 b	na
1998	89 ± 2 b	67 ± 2 a	62 ± 3 a	53 ± 2 b	65 ± 2 a
ANOVA ^e					
df	2, 43	1, 153	1, 142	2, 231	1, 153
F	84	20	55	75	2.23
P>F	0.0001	0.0001	0.0001	0.0001	0.0001

^a Plants that were dead or in declining condition near death were classified as highly susceptible.

^b KCHSC, *Kochia scoparia*.

^c Means ± standard errors in each column followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$. Mean separations were based on statistics that used data transformed by the arcsine of the square root of decimal values.

^d na, not applicable. Seed was not available from the location.

^e Analysis of variance table for the main effect when transformed data were used.

Table 2.10. Dicamba rate effect on *K. scoparia* injury 28 days after treatment for each location averaged across years and positions.

Dicamba	KCHSC ^a Injury				
Rate	Location				
	Burlington	Culbertson	Henry	Longmont	Morrill
(g ae ha ⁻¹)	(%)				
0	0 ± 0 c ^b	0 ± 0 c	0 ± 0 c	0 ± 0 c	0 ± 0 c
70	88 ± 1 b	72 ± 1 b	67 ± 1 b	77 ± 1 b	75 ± 1 b
140	94 ± 1 a	87 ± 1 a	77 ± 1 a	87 ± 1 a	84 ± 1 a
ANOVA ^c					
df	1, 224	1, 47	1, 164	1, 41	1, 18
F	110	380	240	430	330
P>F	0.0001	0.0001	0.0001	0.0001	0.0001

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^c Analysis of variance table for the main effect.

Table 2.11. Position effect on *K. scoparia* injury 28 days after treatment for each location averaged across years and dicamba rates.

Position ^a	KCHSC ^b Injury				
	Location				
	Burlington	Culbertson	Henry	Longmont	Morrill
	(%)				
Out	90 ± 1 a ^c	87 ± 1 a	81 ± 2 a	85 ± 1 a	83 ± 1 a
In	92 ± 1 a	72 ± 1 b	63 ± 1 b	79 ± 1 b	76 ± 1 b
ANOVA ^d					
df	1, 226	1, 166	1, 175	1, 247	1, 168
F	3.38	60	64	18.66	15.24
P>F	0.067	0.0001	0.0001	0.0001	0.0001

^a Position was in reference to the cropping area. Out, adjacent field or non-crop area; In, primary crop area.

^b KCHSC, *Kochia scoparia*.

^c Means ± standard errors in each column followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^d Analysis of variance table for the main effect.

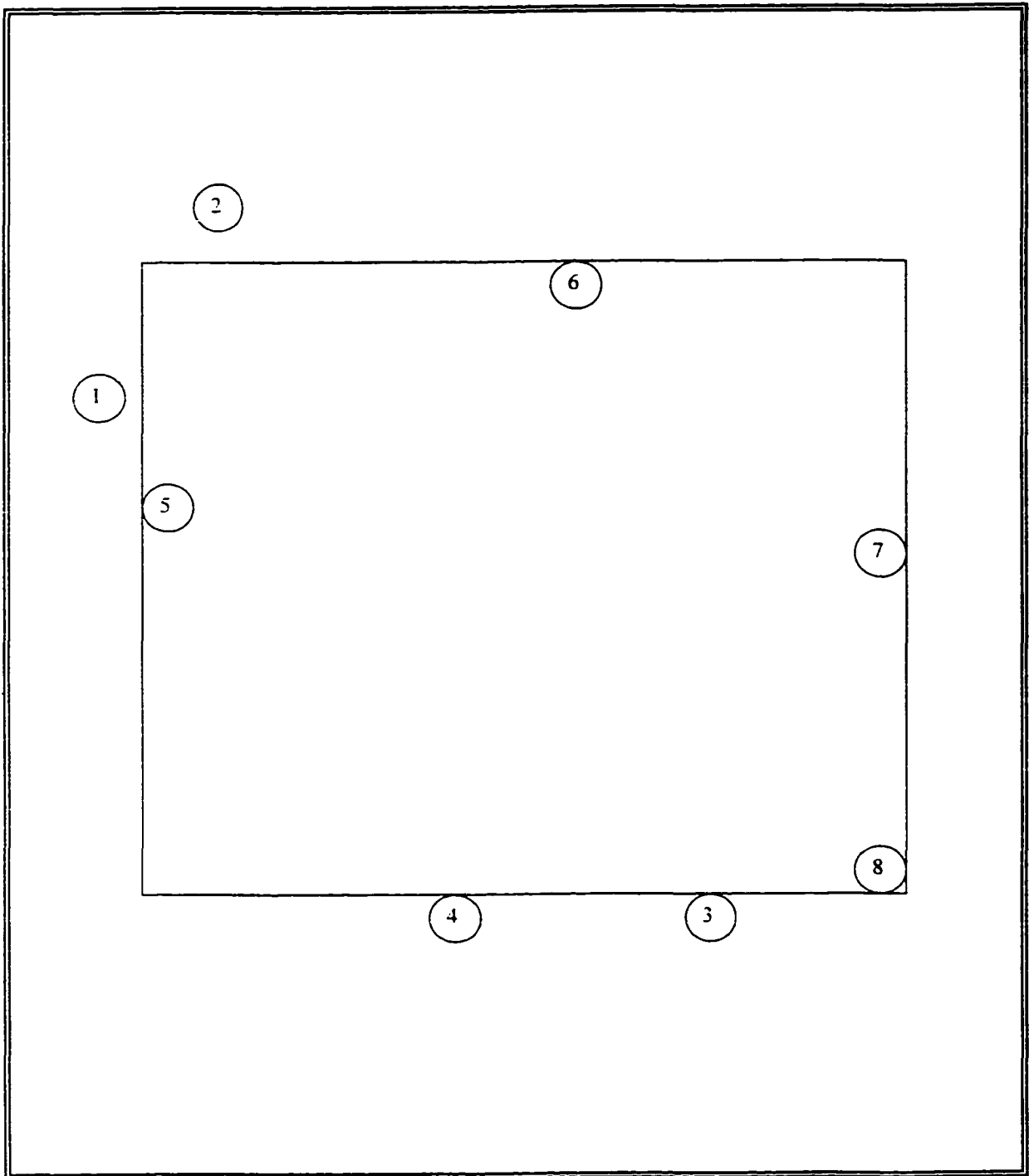


Figure 2.1. Map of *K. scoparia* sample areas from Cimerron, KS. The rectangle represents the primary crop area. Circles represent the sample areas with numbers corresponding to Table 2.4.

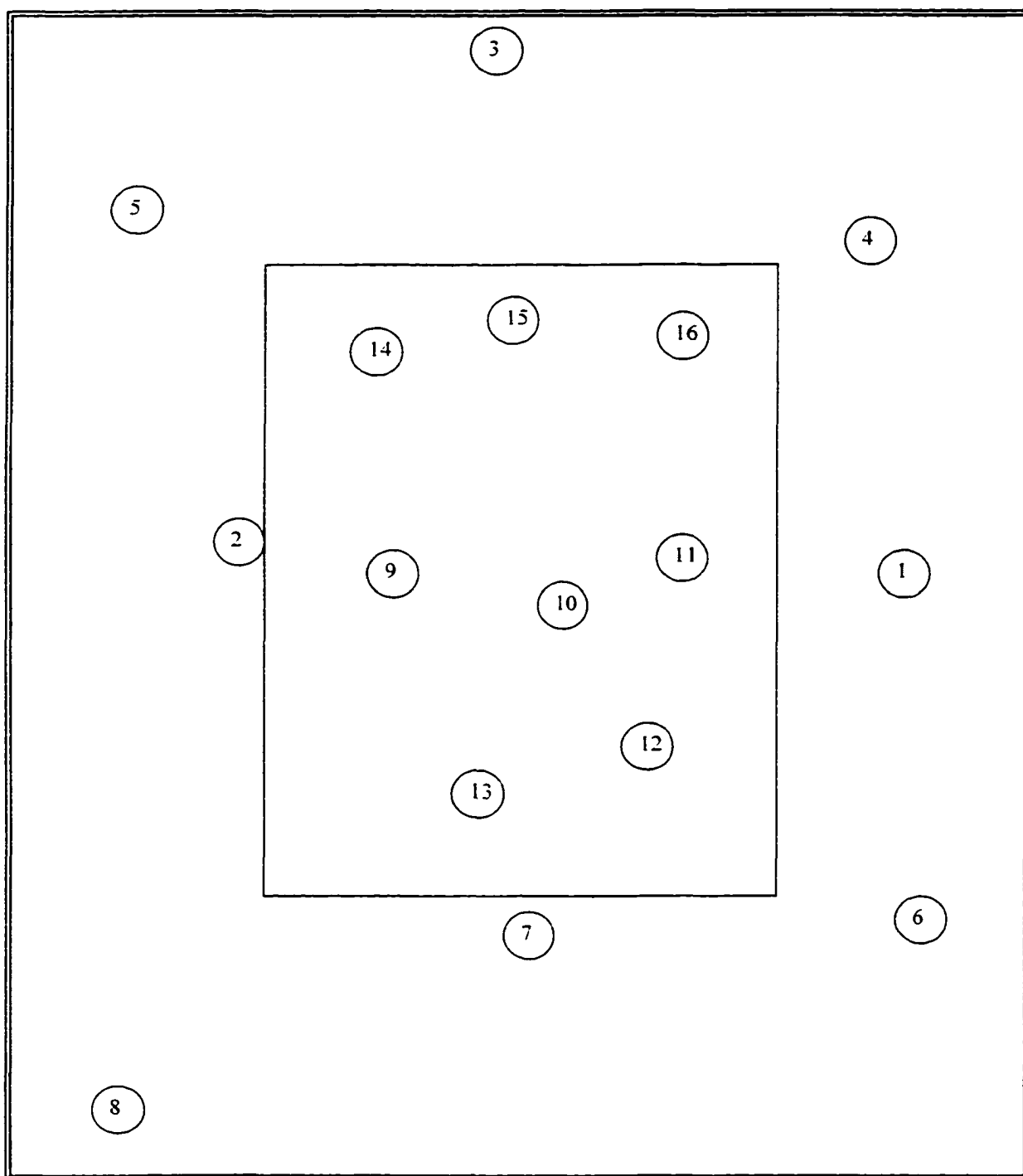


Figure 2.2. Map of *K. scoparia* sample areas from McCook, NE. The rectangle represents the primary crop area. Circles represent the sample areas with numbers corresponding to Table 2.5.

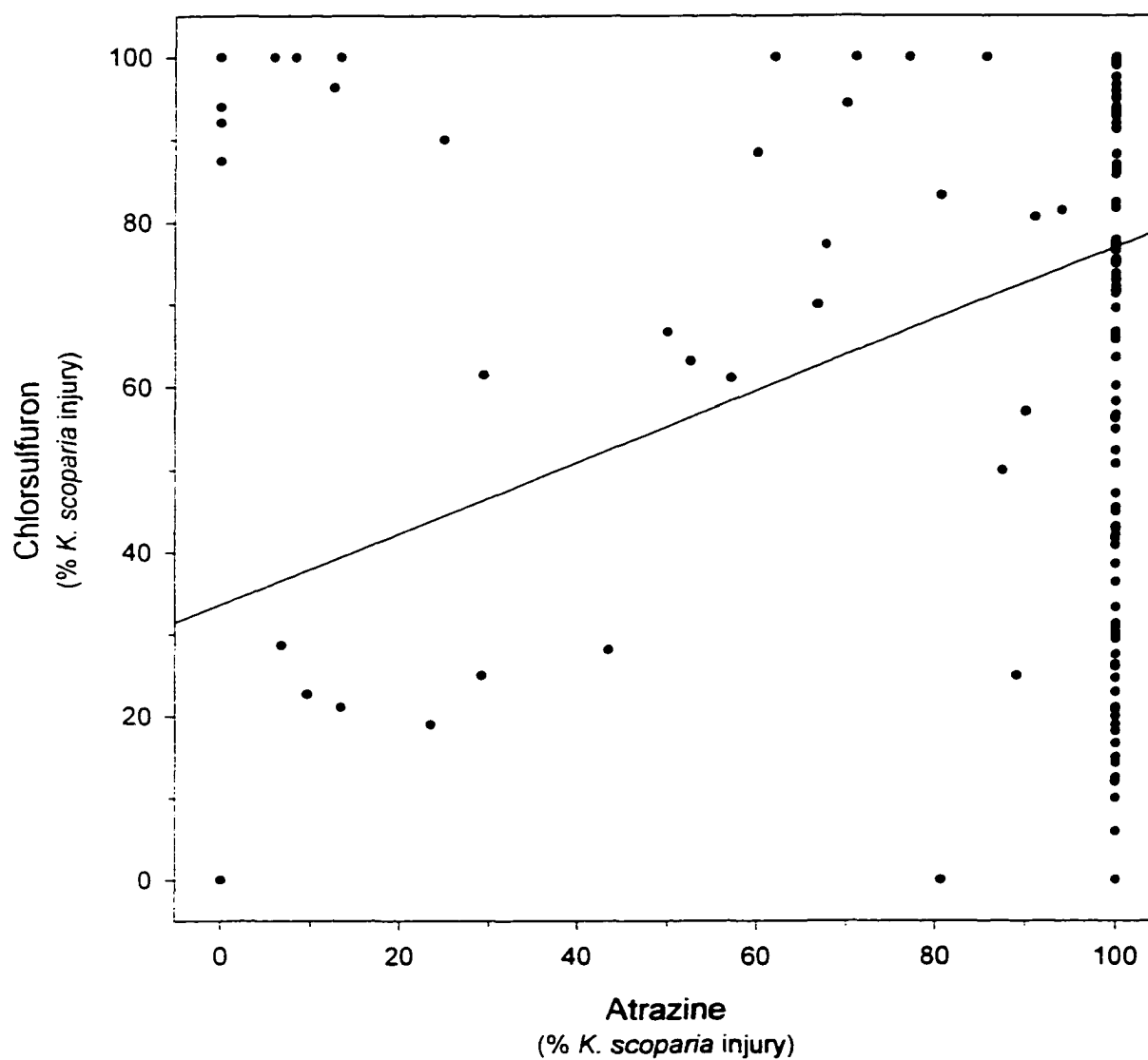


Figure 2.3. Correlation of *K. scoparia* injury from 26 g ai ha⁻¹ chlorsulfuron to injury from 1400 g ai ha⁻¹ atrazine. Each point represents a location by replication pair of data from the two herbicides. Pearson Correlation Coefficient = -0.86, $P = 0.0001$.

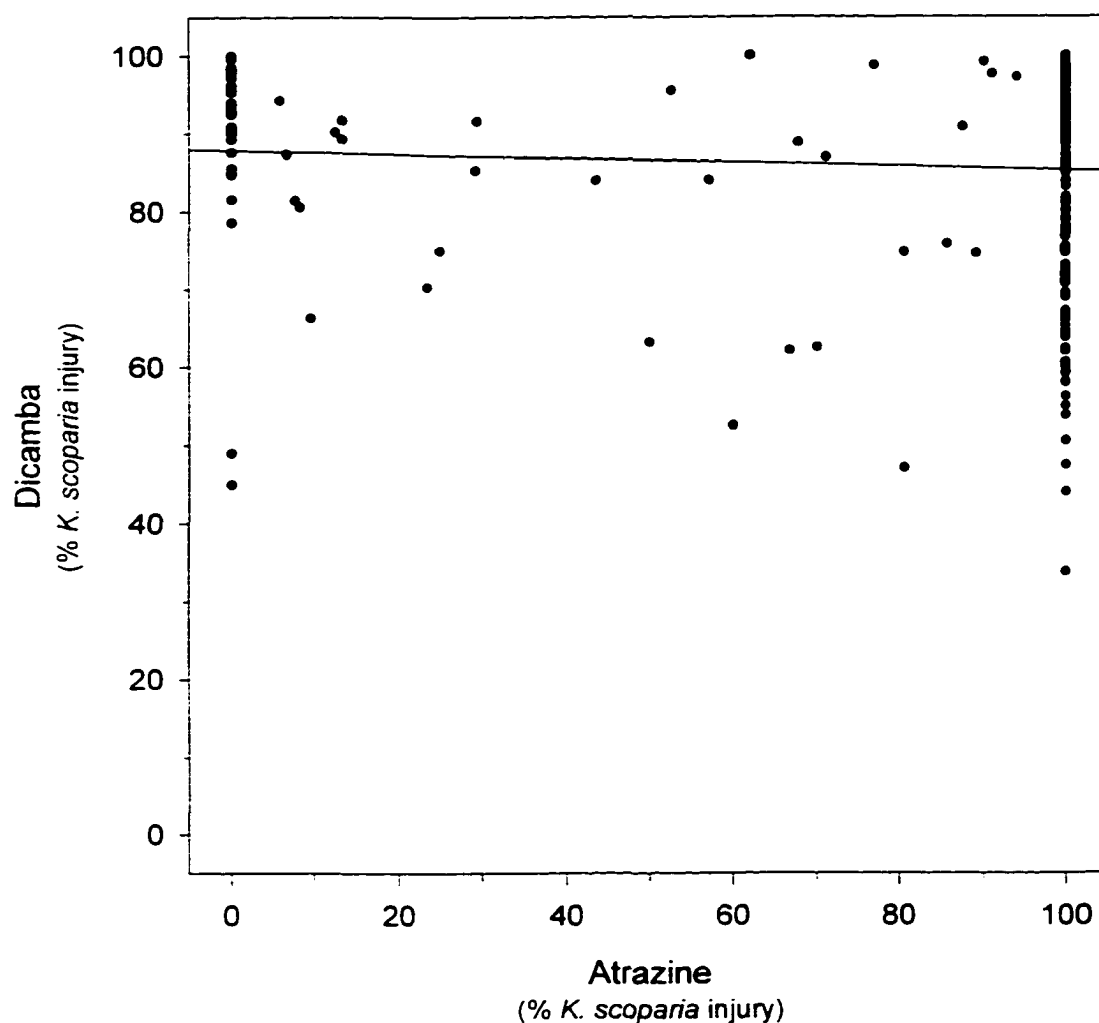


Figure 2.4. Correlation of *K. scoparia* injury from 140 g ae ha⁻¹ dicamba to injury from 1400 g ai ha⁻¹ atrazine. Each point represents a location by replication pair of data from the two herbicides. Pearson Correlation Coefficient = -0.07, P = 0.25.

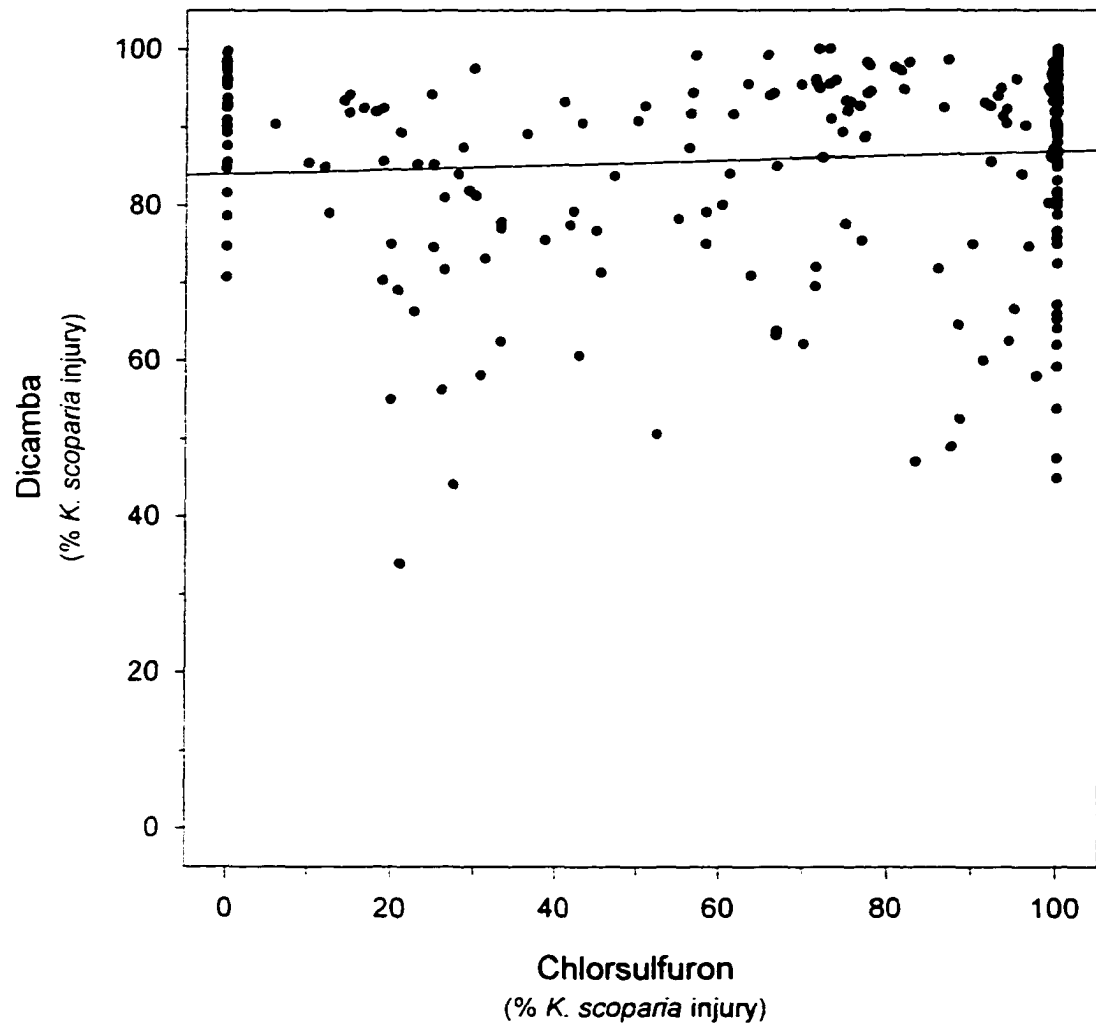


Figure 2.5. Correlation of *K. scoparia* injury from 140 g ae ha⁻¹ dicamba to injury from 26 g ai ha⁻¹ chlorsulfuron. Each point represents a location by replication pair of data from the two treatments. Pearson Correlation Coefficient = 0.08, P = 0.22.

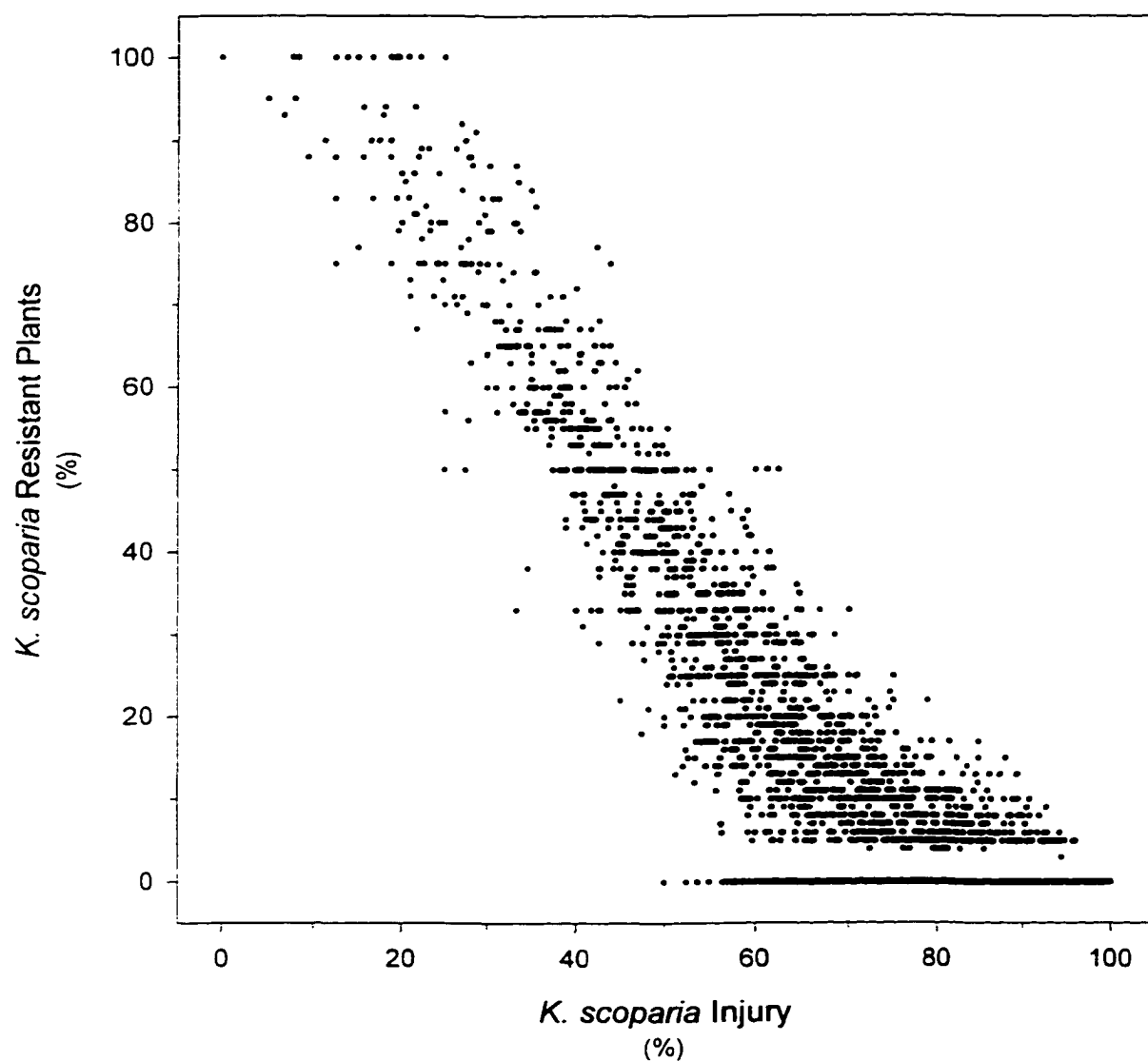


Figure 2.6. Correlation of the number of *K. scoparia* resistant plants to plant injury at 28 days after treatment with 70 g ae ha⁻¹ or 140 g ae ha⁻¹ dicamba. Each point represents location by replication data within year and dicamba rate. Pearson Correlation Coefficient = -0.86, $P = 0.0001$.

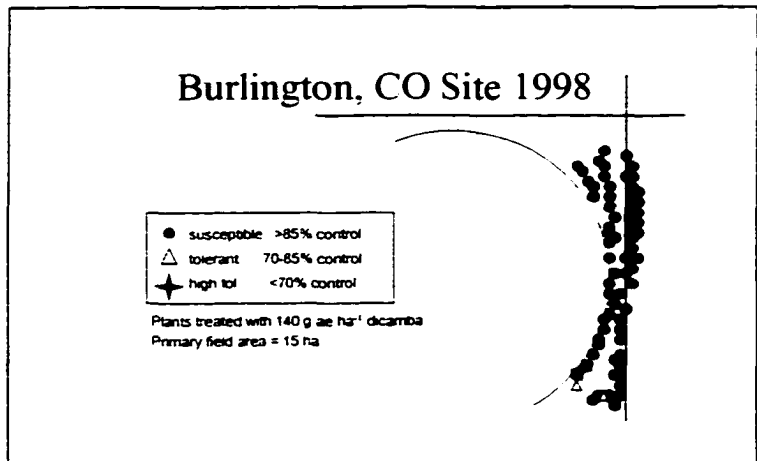
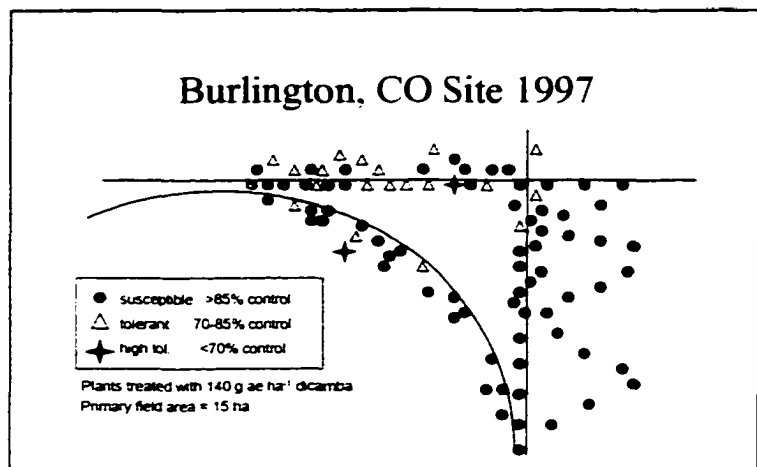
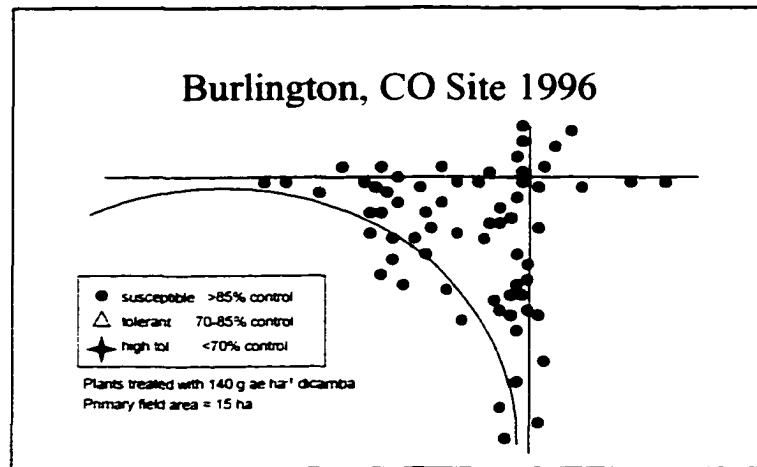


Figure 2.7. Sample locations of individual *K. scoparia* plants at Burlington, CO.

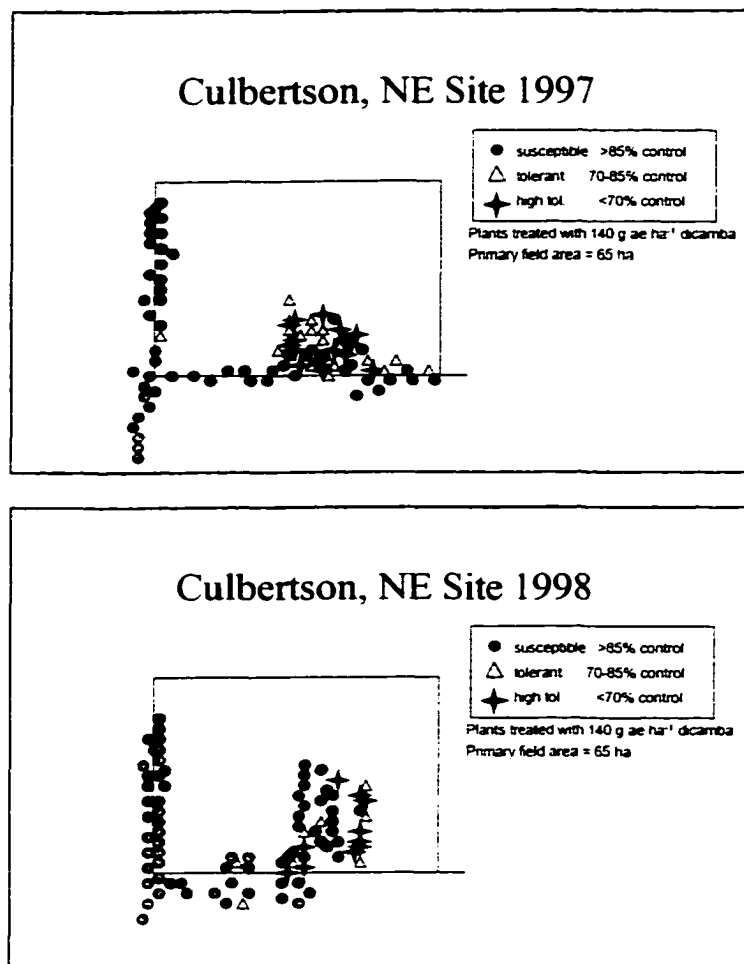


Figure 2.8. Sample locations of individual *K. scoparia* plants at Culbertson, NE.

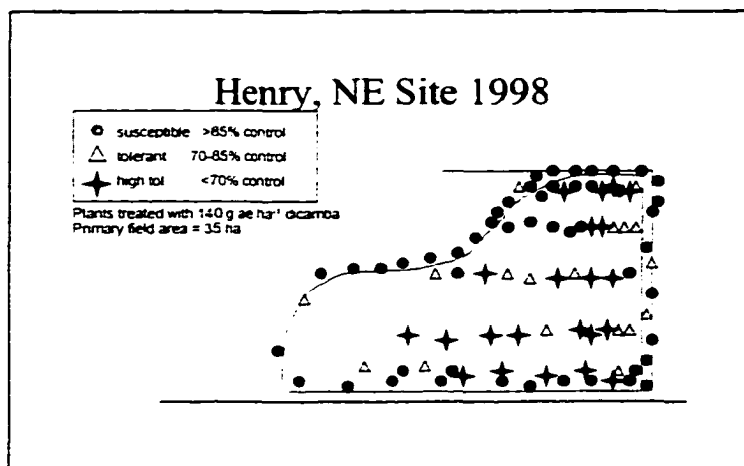
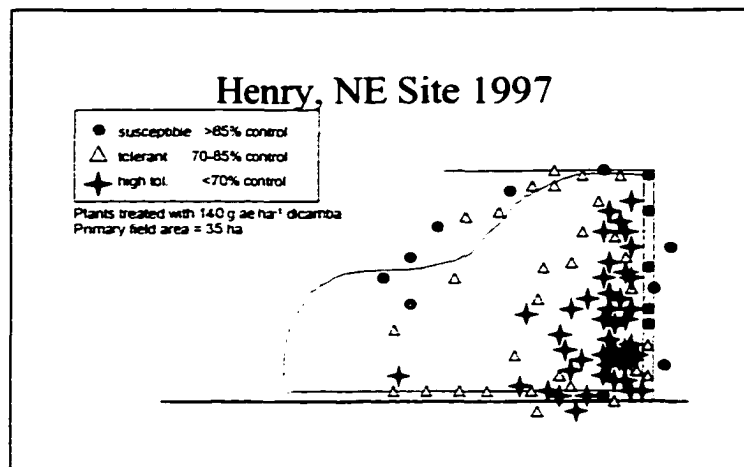


Figure 2.9. Sample locations of individual *K. scoparia* plants at Henry, NE.

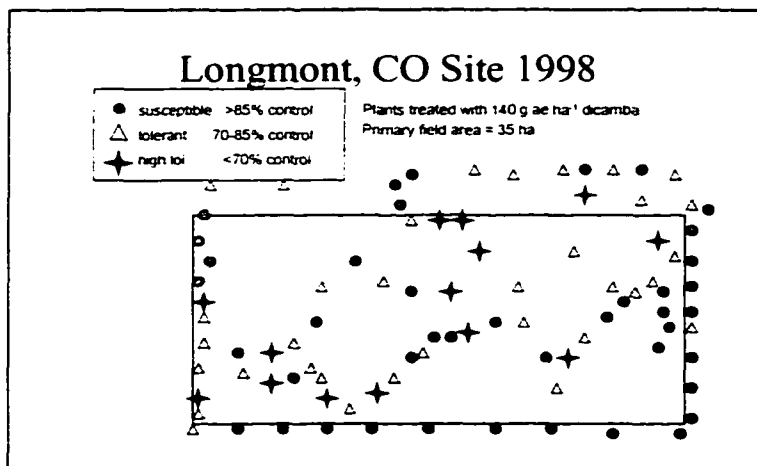
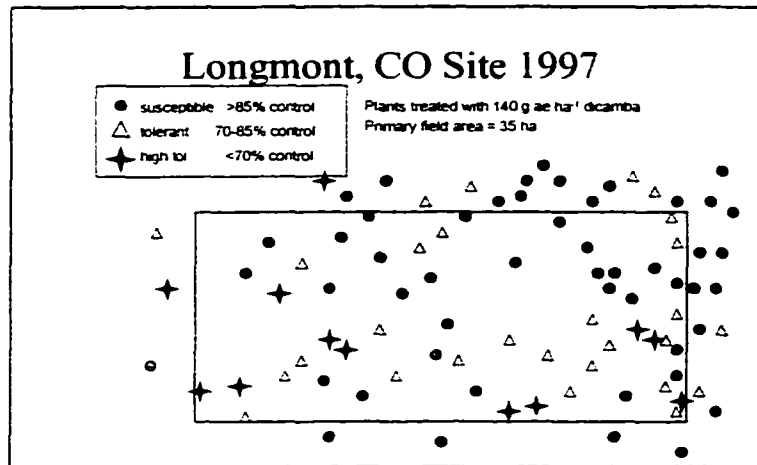
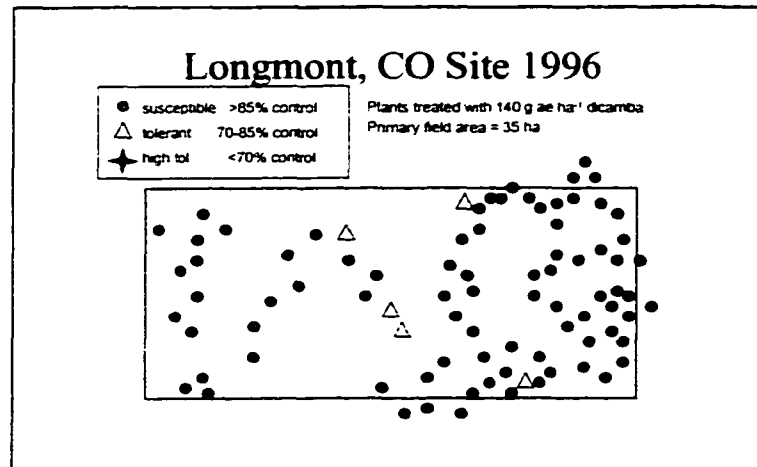


Figure 2.10. Sample locations of individual *K. scoparia* plants at Longmont, CO.

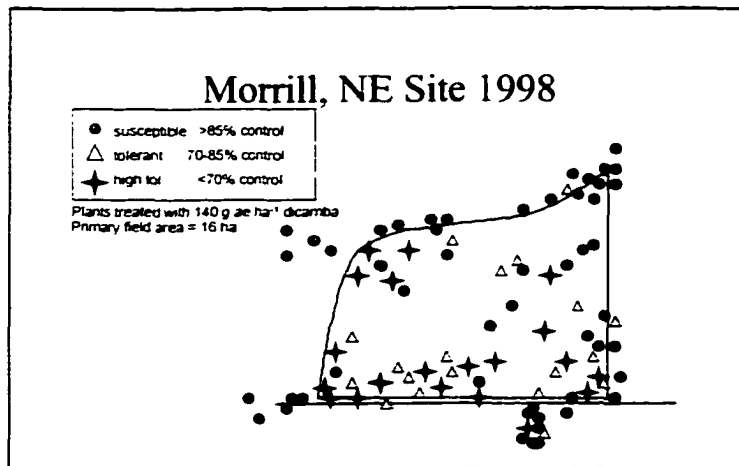
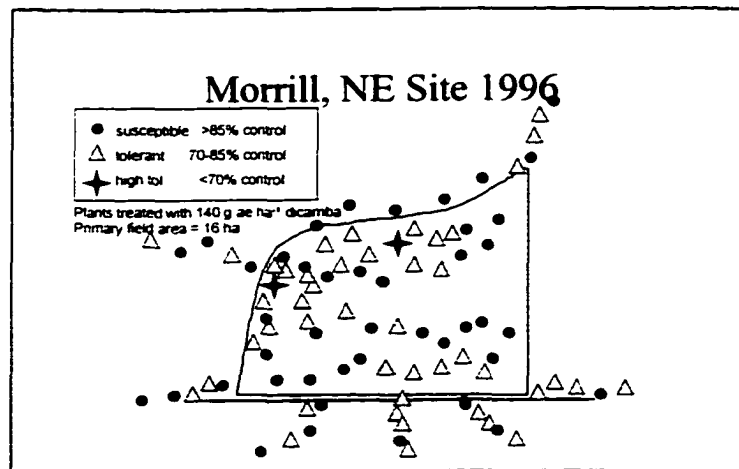


Figure 2.11. Sample locations of individual *K. scoparia* plants at Morrill, NE.

CHAPTER 3
KOCHIA ETHYLENE EVOLUTION IN RESPONSE TO DICAMBA
APPLICATION AND EFFECT OF ETHYLENE ON KOCHIA

ABSTRACT

Kochia treated with 1120 g ae ha⁻¹ ethephon and 560 g ae ha⁻¹ dicamba generated similar ethylene levels. Ethylene generated from ethephon did not produce epinastic symptoms. Dicamba treatment caused epinasty and increased ethylene production within six hours of application. Ethylene evolution from kochia plants occurred in dicamba-susceptible (S2) and non-susceptible (Henry) kochia accessions. Ethylene evolution following dicamba application increased with time up to 48 hours and as dicamba rate increased from 70 g ae ha⁻¹ to 560 g ae ha⁻¹. Ethylene evolution from the S2 accession was nearly 2.5 times higher than evolution from the Henry accession averaged across dicamba rates and harvest times. The greatest difference in ethylene evolution between accessions occurred at the 24-hr harvest and 140 g ae ha⁻¹ dicamba. At this combination, ethylene evolution from the S2 accession was 470% of the ethylene measured from the Henry accession.

INTRODUCTION

Dicamba is a synthetic, auxinic herbicide used to manage broadleaved weeds. While the mode of action is not known, it is believed to act like the plant hormone auxin (Devine et al. 1993). Early symptoms of auxinic herbicides include ethylene production and epinasty, which are also induced by auxin (Coupland 1994; Reid 1995). Ethylene has been implicated in the mechanism of action of several auxinic herbicides. Ethylene produced as a plant response to application of 2,4-D was believed to slow the rate of stem elongation in soybean seedlings (Holm and Abeles 1968). Dicamba caused whole plant responses in common purslane that resulted in epinasty and defoliation (Stacewicz-Sapuncakis et al. 1973). Tomato, sunflower, and wild mustard are examples of plants that are very sensitive to ethylene in the absence of an auxinic herbicide (Grossmann and Schmulling 1995; Hall et al. 1985; Peniuk et al. 1993). For these plants, ethylene generated in response to auxinic herbicides was determined to be a major cause of herbicidal effects. Other plants, however, such as tobacco, chickweed, and yellow starthistle, are relatively insensitive to the effects of ethylene (Coupland and Jackson 1991; Keller and Van Volkenburgh 1997; Sabba et al. 1998).

Plants have been identified with resistance to auxinic herbicides. Yellow starthistle resistant to picloram was identified in Washington, a wild mustard biotype was determined to be resistant to 2,4-D, and a chickweed biotype was reported resistant to mecoprop (Barnwell and Cobb 1989; Callihan 1990, Peniuk

et al. 1993). These plants were not all sensitive to ethylene; however, part of the resistance characterization was reduced ethylene evolution in the resistant accession in response to auxinic herbicide application (Peniuk et al. 1993; Fuerst et al. 1996). Yellow starthistle resistance was not due to differences in absorption, translocation, or metabolism (Fuerst et al. 1996). Altered absorption, translocation, and metabolism are not believed to be the cause of differential response of kochia to dicamba. These experiments were conducted to determine the sensitivity of kochia to ethylene and to examine ethylene evolution as a plant response to dicamba application.

MATERIALS AND METHODS

Plant Material. Kochia seeds used in this study were obtained from two collections that were maintained at the Colorado State University - Weed Research Facility, Fort Collins, Colorado 80523-1177. These two accessions were chosen because they represent a wide range of natural response to dicamba. In previous experiments, accession 95-8 (S2) was very susceptible to an application of 140 g ae ha⁻¹ dicamba while the accession denoted as Henry showed fewer injury symptoms and had a higher survival rate.

The experimental unit was a flat (model A 12-01, East Jordan Plastics, Inc., P.O. Box 575, East Jordan, Minnesota 49727) 10 cm wide by 15 cm long by 5 cm deep. The flat was filled with Sunshine® potting mix #1 (Sun Gro Horticulture, Inc., 15831 N.E. 8th Street, Bellevue, Washington 98008) to 0.5 cm of the top. Approximately 30 seeds of one kochia accession were distributed on

top of the potting mix across each flat area. Potting mix was sprinkled over the seeds so they were buried by approximately 0.3 cm of potting mix. Flats were kept moist with overhead irrigation and fertilized every two weeks with 100 ml of Miracle-Gro® (Scotts Miracle-Gro Products, Inc., P.O. Box 888, Port Washington, New York 11050) solution. The fertilizer solution was prepared by dissolving 3.5 g granular fertilizer (analysis 15-30-15) in 3.8 L water.

Two days before chemical application, flats were thinned to 20 plants flat⁻¹ to reduce interference between plants and maximize spray coverage. In instances where leaving plants at that density compromised adequate spray coverage, additional plants were removed. The actual number of plants that received chemical treatment was recorded for each flat. Kochia plants were 3 cm to 6 cm tall at chemical application.

Response to Ethylene.

Design. Experiments were conducted under greenhouse conditions. Temperature ranged from 25° C to 29° C with a day:night cycle of 16:8 hr. Sodium halide lamps supplemented day length. The experimental design was a randomized complete block with three replications. A factorial layout with two categories, kochia accession by chemical, was used to examine treatment combinations. Two kochia accessions were used as levels of accession and six treatments were used as chemical levels. The experiment was repeated.

Chemical Treatments. Chemical treatments were administered with a chain-driven chamber sprayer. The sprayer was calibrated to deliver 98 L ha⁻¹ by

compressed air through an 8001 even flat fan tip. Chemical treatments included an untreated control; 280 g ae ha⁻¹ dicamba; 280 g ae ha⁻¹ fluroxypyr; and ethephon at 504 g ae ha⁻¹, 1008 g ae ha⁻¹, and 2016 g ae ha⁻¹.

Evaluation. Two evaluations were conducted to monitor the response of the two kochia accessions to chemical applications. At seven days after treatment, plants in each flat were given an average score for epinastic symptoms. Plants were rated on a scale from 0 to 5 where 0 = no epinasty, 1 = slight leaf epinasty, 2 = moderate leaf epinasty, 3 = severe leaf or slight stem epinasty, 4 = severe leaf and moderate stem epinasty, and 5 = severe leaf and stem epinasty. A second evaluation was performed 28 days after chemical application. This evaluation was a visual assessment of plant injury. Each plant in a flat was given a score for percent injury from 0% (no injury) to 100% (complete death) compared to control plants. Scores for all plants in a flat were averaged to provide a single rating of plant injury.

Data Analyses. Data were analyzed using procedures for mixed effects models in SAS (SAS Institute 1989). Epinasty rating and percent injury data were transformed by the arcsine of the square root of data decimal values. This was done because of the binomial nature of these data. Bartlett's test for variance homogeneity indicated that transformed data from each of the experiments could be pooled for epinasty (df (35, 35), $F = 1.15$, $P = 0.69$) and plant injury data (df (29, 29), $F = 1.24$, $P = 0.56$). Standard diagnostic inquiries of transformed data indicated no violations of the assumptions underlying analysis of variance testing. Mean separation was performed using single degree of

freedom contrasts via the least squares means (lsmeans) statement in the mixed effects procedure (SAS Institute 1989). Non-transformed data were presented in graphs and tables; however, all statistical analyses were performed with transformed data.

Ethylene Evolution.

Design. Plants for these experiments were maintained under greenhouse conditions as previously described. The experimental design was a randomized complete block with four replications. A factorial layout with two categories, accession by chemical, was used to examine treatment combinations over five time points. Two kochia accessions were used as levels of accession and seven chemical treatments were investigated. The experiment was repeated.

Chemical Treatments. Aminooxyacetic acid (AOA) (Sigma Chemical, St. Louis, Missouri) was applied to foliage 24 hours before dicamba application as part of one treatment to inhibit ethylene production. This treatment was applied to run-off with a misting bottle using a 0.5 mM AOA solution. Other chemicals were applied with a chain-driven chamber sprayer. The sprayer was calibrated to deliver 117 L ha⁻¹ by compressed air through an 8001 even flat fan tip. Chemical treatments included an untreated control; dicamba at rates of 70 g ae ha⁻¹, 140 g ae ha⁻¹, 280 g ae ha⁻¹, and 560 g ae ha⁻¹; 1120 g ae ha⁻¹ ethephon; and 280 g ae ha⁻¹ dicamba after AOA application.

Plant Harvest and Ethylene Measurement. Ethylene production was measured at 0 hr, 6 hr, 12 hr, 24 hr, and 48 hr after treatment application. Two

intact plants including the soil surrounding the roots were removed from a flat. Plants were suspended in water to soak soil from the roots while the stems and leaves remained dry. Excess water was blotted from the roots with a paper towel and the fresh weight of the two plants was recorded. Root systems were placed in a 10-ml vial filled with water, which was placed in a 58-ml culture tube and sealed with a rubber septum. The plants were allowed to incubate in the culture tube for two hours.

After incubation, two 1-ml gas samples were removed from the culture tube headspace with a gas-tight syringe. Samples were analyzed for ethylene by gas chromatography using a model 5890 Hewlett-Packard gas chromatograph equipped with a flame-ionization detector. The 2-m by 0.31-cm stainless steel column was packed with 80 to 100 mesh HaysepT polymer (Alltech Associates, Inc., 2051 Waukegan Road, Deerfield, Illinois 60015-1899). Nitrogen was used as the carrier gas and the column was operated at an isothermic 80° C. A 1-ppm concentration of ethylene gas was used as an external standard.

Ethylene measurements were recorded and converted to ng with a calibration curve. The calibration curve was generated using several dilutions from standard cylinders containing 100 ppm $\pm$ 2% ethylene (Scott Specialty Gases, 6141 Easton Road, P.O. Box 310, Plumsteadville, Pennsylvania 18949-0310) and 1 ppm $\pm$ 2% ethylene (Matheson Gas Products, Inc., 166 Keystone Drive, Montgomeryville, Pennsylvania 18963). The equation for this line in the area of biological interest was:

$$(y + 0.0034) \div 0.0904 = x$$

where y was the area under the peak of the chromatograph trace and x was the amount of introduced ethylene in ng (Figure 3.1). Ethylene evolution was expressed as ng ethylene g⁻¹ fresh weight hr⁻¹.

Data Analyses. Data were analyzed using procedures for mixed effects models in SAS (SAS Institute 1989). A natural logarithmic transformation of data was performed to satisfy the assumption of equal variance underlying analysis of variance testing. Bartlett's test for variance homogeneity indicated that transformed data from each of the experiments could be pooled (df (279, 279), $F = 1.04$, $P = 0.75$). Mean separation was performed using single degree of freedom contrasts via the least squares means (lsmeans) statement in the mixed effects procedure of SAS (SAS Institute 1989). Non-transformed data were presented in graphs and tables; however, all statistical analyses were performed with transformed data.

RESULTS AND DISCUSSION

Response to Ethylene.

Epinasty. The kochia accessions used in this experiment differed in epinastic response to chemical treatments. Over all treatments, accession S2 produced a greater epinastic response than the Henry accession (df (1, 48), $F = 88$, $P = 0.0001$). This is consistent with symptom expression of other dicamba susceptible and non-susceptible kochia accessions treated with dicamba in previous experiments.

Chemical treatments were the largest determinant of epinastic response (df (5, 12), $F = 580$, $P = 0.0001$, Table 3.1). This was primarily due to the inability of ethephon treatments, even at $2016 \text{ g ae ha}^{-1}$, to cause any epinasty (df (1, 12), $t = 0.00$, $P = 1.0$). On the contrary, dicamba (df (1, 12), $t = 29$, $P = 0.0001$) and to a greater extent fluroxypyr (df (1, 12), $t = 6.89$, $P = 0.0001$) caused severe leaf and stem epinasty. Plants treated with dicamba or fluroxypyr were so severely twisted that they became interwoven and pressed against the soil surface, while plants in the ethephon treatments did not show the slightest leaf cupping.

An interaction existed between kochia accession and chemical treatment (df (5, 48), $F = 36$, $P = 0.0001$, Table 3.2). Examined separately, the mean separations for each accession were identical. The interaction was attributed to a combination of kochia's non-response to ethephon and differences in herbicidal activity of dicamba and fluroxypyr on the two kochia accessions. While there was no difference in epinasty scores between accessions treated with ethephon (df (1, 48) $t = 0.00$, $P = 1.0$), the epinastic responses due to dicamba and fluroxypyr treatments were dependent on kochia accession. Accession S2 exhibited more epinasty symptoms than the Henry accession for both herbicide treatments (df (1, 48), $t = 10.04$, $P = 0.0001$).

Plant Injury. Analysis of plant injury 28 days after treatment was generally consistent with that of epinasty scores at 7 days after treatment. Plant injury differed between the kochia accessions (df (1, 40), $F = 12.49$, $P = 0.0010$). The Henry accession, with 34% average injury, was less injured than the S2 accession, 37% injury.

As with epinasty scores, plant injury was not different from the control plants for any of the ethephon treatments (df (1, 10), $t = 0.37$, $P = 0.72$, Table 3.1). Treatment effects were due to dicamba and fluroxypyr (df (4, 10), $F = 960$, $P = 0.0001$). The results of these treatments mirrored the results from epinasty data. Dicamba, 86% injury, was different from the control and ethephon treatments, 0% injury (df (1, 10), $t = 38$, $P = 0.0001$), and different from fluroxypyr, 91% injury (df (1, 10), $t = 3.68$, $P = 0.0043$).

Plant injury was affected by an interaction between kochia accession and treatment (df (4, 40), $F = 9.56$, $P = 0.0001$, Table 3.3). This interaction involved the response of each accession to the dicamba treatment. All other treatments elicited similar responses in plant injury from the two accessions (df (4, 40), $t = 1.19$, $P = 0.24$). The Henry accession, however, displayed less injury from the dicamba treatment than the S2 accession (df (4, 40), $t = 7.02$, $P = 0.0001$). This resulted in a unique difference between accessions treated with dicamba, which was expected due to the nature of the kochia accessions chosen.

Ethylene Evolution.

Ethephon treatment resulted in similar amounts of ethylene measured from the S2 and Henry accessions (df (1, 58), $t = 1.27$, $P = 0.21$). Ethylene levels from ethephon were not different from the ethylene response of accession S2 when treated with 560 g ae ha⁻¹ dicamba (df (1, 58), $t = 0.80$, $P = 0.43$). Plants treated with ethephon generated large quantities of ethylene at the 0-hr harvest, 49 ng (g FW)⁻¹ hr⁻¹ (df (1, 233), $t = 10.56$, $P = 0.0001$). Ethylene

detected from ethephon treated plants rose sharply between the 0-hr and 6-hr harvests (df (1, 466), $t = 4.85$, $P = 0.0001$). Ethylene evolution in the ethephon treatment reached a maximum of $100 \text{ ng (g FW)}^{-1} \text{ hr}^{-1}$ 6 hours after treatment and then gradually declined. Ethylene was still generated at $47 \text{ ng (g FW)}^{-1} \text{ hr}^{-1}$ 48 hr after treatment, which was higher than the control plants, $8 \text{ ng (g FW)}^{-1} \text{ hr}^{-1}$ (df (1, 233), $t = 10.07$, $P = 0.0001$). As in the preceding experiment, neither of the accessions manifested any visual injury or epinasty from treatment with ethephon.

Plants pre-treated with AOA did not prevent ethylene evolution. In the S2 accession, ethylene evolution from plants treated with AOA and 280 g ae ha^{-1} dicamba, $59 \text{ ng (g FW)}^{-1} \text{ hr}^{-1}$ ethylene, did not differ from plants treated with 280 g ae ha^{-1} dicamba, $68 \text{ ng (g FW)}^{-1} \text{ hr}^{-1}$ ethylene (df (1, 58), $t = 0.55$, $P = 0.58$). Ethylene evolution from Henry accession kochia plants treated with 280 g ae ha^{-1} dicamba was reduced by AOA pre-treatment from $27 \text{ ng (g FW)}^{-1} \text{ hr}^{-1}$ to $21 \text{ ng (g FW)}^{-1} \text{ hr}^{-1}$ ethylene.

Ethylene produced in response to dicamba application was dependent on accession (df (1, 3), $F = 270$, $P = 0.0005$). For all dicamba rates, the S2 accession reached maximum evolution by 12 hours after treatment and then sustained that level of ethylene production up to the 48-hr harvest (Figure 3.2). Average ethylene evolution for accession S2 was $53 \text{ ng (g FW)}^{-1} \text{ hr}^{-1}$. This was nearly 2.5 times higher than the average ethylene evolution for the Henry accession, $22 \text{ ng (g FW)}^{-1} \text{ hr}^{-1}$. Ethylene evolution from the Henry accession

tended to increase over the duration of the experiment for all dicamba rates and showed no indication that a plateau was reached (Figure 3.3).

An increase in ethylene production was associated with each increase in dicamba rate averaged across accessions (df (4, 344), $F = 170$, $P = 0.0001$, Table 3.4). Each doubling of dicamba rate resulted in approximately 5 ng (g FW)⁻¹ hr⁻¹ more ethylene produced. The greatest increase was between 140 g ae ha⁻¹ and 280 g ae ha⁻¹ dicamba, an increase from 41 ng (g FW)⁻¹ hr⁻¹ to 48 ng (g FW)⁻¹ hr⁻¹ ethylene (df (1, 344), $t = 2.10$, $P = 0.036$).

Graphical representation for combined effects of kochia accession and dicamba rate suggested that responses of each accession diverge as dicamba rate was increased (Figure 3.4). These data do not support this trend as an interaction (df (4, 344), $F = 1.96$, $P = 0.10$, Table 3.5), possibly because ethylene evolution from the accessions differed in the absence of dicamba treatment (df (1, 52), $t = 6.52$, $P = 0.0001$). Accession S2 always produced more ethylene than the Henry accession, and there was a trend toward higher ethylene production for both accessions as dicamba rate increased.

The amount of time elapsed between dicamba application and ethylene sampling also affected ethylene evolution (df (4, 344), $F = 190$, $P = 0.0001$, Table 3.6). Longer herbicide exposure periods generally meant that more ethylene was detected. This was not related to the amount of incubation time for ethylene generation, since the incubation time was always 2 hr. The largest increase occurred between 6-hr and 12-hr harvests when ethylene levels rose from 26 ng (g FW)⁻¹ hr⁻¹ to 47 ng (g FW)⁻¹ hr⁻¹ (df (1, 344), $t = 5.19$, $P = 0.0001$). Average

ethylene production was still increasing at the final harvest, $59 \text{ ng (g FW)}^{-1} \text{ hr}^{-1}$ during the 48-hr harvest compared to $47 \text{ ng (g FW)}^{-1} \text{ hr}^{-1}$ during the 24-hr harvest (df (1, 344) , $t = 4.79$, $P = 0.0001$).

Interaction between kochia accession and time after dicamba application influenced ethylene evolution (df (4, 344) , $F = 31$, $P = 0.0001$, Table 3.7). Initial increase in ethylene was due to increased response from each accession. The increase in ethylene production between the 6-hr and 12-hr harvests averaged across accessions (Table 3.6) was largely regulated by accession S2. Between these harvests, S2 accession ethylene generation increased from $29 \text{ ng (g FW)}^{-1} \text{ hr}^{-1}$ to $70 \text{ ng (g FW)}^{-1} \text{ hr}^{-1}$ (df (1, 344) , $t = 8.17$, $P = 0.0001$, Table 3.7), while evolution from the Henry accession remained unchanged (df (1, 344) , $t = 1.88$, $P = 0.061$). At the 48-hr harvest, both accessions contributed to the increase in average ethylene response. The interaction of accession and time after treatment was also influenced by the similarity of ethylene evolution at the 0-hr harvest (df (1, 52) , $t = 1.41$, $P = 0.16$) and by the divergence of ethylene measurements at later harvests (df (1, 52) , $t = 2.48$, $P = 0.016$, minimum difference). Accession S2 produced much more ethylene than the Henry accession beginning with the 12-hr harvest (Figure 3.5).

Analyses of data for individual main effects or combinations of two main effects holding other effects constant were robust for this experiment due to the magnitude of F value. The combination of kochia accession by dicamba rate by time after treatment, however, was a significant interaction (df (16, 344) , $F = 2.41$, $P = 0.0019$). Examination of the data indicated this interaction was

probably due to the influence of the 0-hr harvest, the herbicide control, or both. Combinations of dicamba rate and time after treatment that did not include the 0-hr harvest or the herbicide control seemed to have similar ethylene relationships between kochia accessions. Representative data that illustrated ethylene evolution as time increased holding the dicamba rate at 140 g ae ha⁻¹ indicated that evolution from the Henry accession may decline for a short period after the 6-hr harvest (Figure 3.6). This recession in ethylene evolution was apparent for all dicamba treatments at the 12-hr harvest, the 24-hr harvest, or both (Figure 3.3). Similar illustration of dicamba rates at the 12-hr harvest was representative of other harvests excluding the 0-hr harvest (Figure 3.7).

CONCLUSIONS

Plant species differ in their response to ethylene. Ethylene, induced by dicamba, was a determined cause of epinasty and defoliation in common purslane (Stacewicz-Sapuncakis et al. 1973). Yellow starthistle and chickweed, however, were not sensitive to ethylene (Coupland and Jackson 1991; Sabba et al. 1998). Data suggested that kochia also was insensitive to ethylene. Ethylene was generated from ethephon application to kochia but did not cause epinasty. The level of ethylene evolution from kochia treated with 1120 g ae ha⁻¹ ethephon was similar to ethylene production from dicamba-susceptible kochia treated with 560 g ae ha⁻¹. Ethylene level due to ethephon, however, did not affect young vegetative kochia tissue while dicamba treatments induced epinasty within 6 hr

on accession S2. This was in agreement with work conducted on yellow starthistle that indicated very little epinasty from ethephon application (Sabba et al. 1998). If ethylene was a contributing factor to kochia response following auxinic herbicide application, it relied on the herbicide or some herbicide induced plant response to cause symptoms. The results of these experiments indicated that ethylene production was a symptom of dicamba activity. Ethylene production was lower in the Henry accession as a characteristic manifestation of lower dicamba activity. Biotypes of yellow starthistle, wild mustard, and chickweed, resistant to auxinic herbicides also were characteristically low ethylene generators compared to susceptible biotypes (Barnwell and Cobb 1989; Callihan 1990, Peniuk et al. 1993). Regardless of sensitivity to ethylene, ethylene regulation in these plants was altered.

Differences in kochia accession response to dicamba might be exploited in the future to classify kochia accessions. Many more accessions with known and stable responses to auxinic herbicides need to be examined to determine if ethylene production is a good predictor of plant injury. Development of a field bioassay based on ethylene production to characterize kochia populations may be possible by taking advantage of differences in ethylene evolution as effected by dicamba rate and time after treatment. Development of a bioassay that reliably predicts kochia response might be confounded by the intermediate nature of kochia's response to auxinic herbicides in the field.

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Table 3.1. Dicamba, fluroxypyr, and ethephon effects on plant epinasty and injury ratings averaged across *K. scoparia* accessions.

Chemical	Rate	Epinasty Score ^a	Plant Injury ^b
	(g ae ha ⁻¹)		(%)
Control		0.0 ± 0.0 c ^c	0 ± 0 c
Dicamba	280	3.4 ± 0.2 b	86 ± 1 b
Fluroxypyr	280	4.2 ± 0.1 a	91 ± 1 a
Ethephon	504	0.0 ± 0.0 c	1 ± 1 c
Ethephon	1008	0.0 ± 0.0 c	0 ± 0 c
Ethephon	2016	0.0 ± 0.0 c	0 ± 0 c
ANOVA ^d			
df		5, 12	4, 10
F		580	960
P>F		0.0001	0.0001

^a Epinasty evaluated seven days after treatment (0 = no epinasty, 5 = severe leaf and stem epinasty).

^b Plant injury evaluated 28 days after treatment.

^c Means ± standard errors in each column followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$. Mean separations were based on statistics that used data transformed by the arcsine of the square root of data decimal values.

^d Analysis of variance table for the main effect when transformed data were used.

Table 3.2. Combined effects of chemical application and *K. scoparia* accession on plant epinasty rating seven days after application.

Chemical	Rate	KSHSC ^a Epinasty Score ^b	
		Accession	
		S2	Henry
(g ae ha ⁻¹)			
Control		C ^c 0.0 ± 0.0 a ^d	C 0.0 ± 0.0 a
Dicamba	280	B 4.3 ± 0.2 a	B 2.5 ± 0.2 b
Fluroxypyr	280	A 5.0 ± 0.1 a	A 3.3 ± 0.1 b
Ethephon	504	C 0.0 ± 0.0 a	C 0.0 ± 0.0 a
Ethephon	1008	C 0.0 ± 0.0 a	C 0.0 ± 0.0 a
Ethephon	2016	C 0.0 ± 0.0 a	C 0.0 ± 0.0 a
ANOVA ^e			
df		5, 48	
F		36	
P>F		0.0001	

^a KCHSC, *Kochia scoparia*.

^b Epinasty scale: 0 = no epinasty, 5 = severe leaf and stem epinasty.

^c Means ± standard errors in each column preceded by the same upper case letter were not different according to least squares means contrasts for $\alpha = 0.05$. Mean separations were based on statistics that used data transformed by the arcsine of the square root of data decimal values.

^d Means ± standard errors in each row followed by the same lower case letter were not different according to least squares means contrasts for $\alpha = 0.05$. Mean separations were based on statistics that used data transformed by the arcsine of the square root of data decimal values.

^e Analysis of variance table for the interaction effect when transformed data were used.

Table 3.3. Combined effects of chemical application and *K. scoparia* accession on plant injury rating 28 days after application.

Chemical	Rate	KSHSC ^a Plant Injury	
		Accession	
		S2	Henry
	(g ae ha ⁻¹)	(%)	
Control		B ^b 0 ± 0 a ^c	C 0 ± 0 a
Dicamba	280	A 92 ± 2 a	B 80 ± 2 b
Fluroxypyr	280	A 92 ± 2 a	A 90 ± 2 a
Ethephon	504	B 1 ± 1 a	C 1 ± 1 a
Ethephon	1008	B 0 ± 0 a	C 1 ± 1 a
Ethephon	2016	B 0 ± 0 a	C 0 ± 0 a
ANOVA ^d			
df		4, 40	
F		9.56	
P>F		0.0001	

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column preceded by the same upper case letter were not different according to least squares means contrasts for $\alpha = 0.05$. Mean separations were based on statistics that used data transformed by the arcsine of the square root of data decimal values.

^c Means ± standard errors in each row followed by the same lower case letter were not different according to least squares means contrasts for $\alpha = 0.05$. Mean separations were based on statistics that used data transformed by the arcsine of the square root of data decimal values.

^d Analysis of variance table for interaction effect when transformed data were used.

Table 3.4. Dicamba rate effect on ethylene evolution from *K. scoparia* averaged across accessions and sample times.

Dicamba Rate	Ethylene Evolution
(g ae ha ⁻¹)	(ng (g FW) ⁻¹ hr ⁻¹)
0	9.7 ± 2.1 e ^a
70	35.4 ± 2.1 d
140	40.6 ± 2.1 c
280	47.9 ± 2.1 b
560	52.7 ± 2.1 a
ANOVA ^b	
df	4, 344
F	170
P>F	0.0001

^a Means ± standard errors followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$. Mean separations were based on statistics that used natural logarithm transformed data.

^b Analysis of variance table for the main effect when transformed data were used.

Table 3.5. Combined effects of dicamba rate and *K. scoparia* accession on ethylene evolution averaged across time after dicamba application.

Dicamba Rate	Ethylene Evolution	
	KCHSC ^a Accession	
	S2	Henry
(g ae ha ⁻¹)	(ng (g FW) ⁻¹ hr ⁻¹)	
0	C ^b 13.1 ± 2.8 a ^c	E 6.3 ± 2.8 b
70	B 53.0 ± 2.8 a	D 17.8 ± 2.8 b
140	AB 59.7 ± 2.8 a	C 21.4 ± 2.8 b
280	A 68.4 ± 2.8 a	B 27.4 ± 2.8 b
560	A 69.4 ± 2.8 a	A 36.1 ± 2.8 b
ANOVA ^d		
df	4, 344	
F	1.96	
P>F	0.10	

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column preceded by the same upper case letter were not different according to least squares means contrasts for $\alpha = 0.05$. Mean separations were based on statistics that used natural logarithm transformed data.

^c Means ± standard errors in each row followed by the same lower case letter were not different according to least squares means contrasts for $\alpha = 0.05$. Mean separations were based on statistics that used natural logarithm transformed data.

^d Analysis of variance table for the interaction effect when transformed data were used.

Table 3.6. Time after dicamba application effect on ethylene evolution from *K. scoparia* averaged across accessions and treatments.

Time After Dicamba Application	Ethylene Evolution
(hr)	(ng (g FW) ⁻¹ hr ⁻¹)
0	8.2 ± 2.0 d ^a
6	26.1 ± 2.0 c
12	46.9 ± 2.0 b
24	46.7 ± 2.0 b
48	58.5 ± 2.0 a
ANOVA ^b	
df	4, 344
F	190
P>F	0.0001

^a Means ± standard errors followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$. Mean separations were based on statistics that used natural logarithm transformed data.

^b Analysis of variance table for the main effect when transformed data were used.

Table 3.7. Combined effects of time after dicamba application and *K. scoparia* accession on ethylene evolution averaged across dicamba rates.

Time After Dicamba Application (hr)	Ethylene Evolution	
	KCHSC ^a Accession	
	S2	Henry
	(ng (g FW) ⁻¹ hr ⁻¹)	
0	D ^b 8.8 ± 2.7 a ^c	C 7.6 ± 2.7 a
6	C 29.4 ± 2.7 a	B 22.8 ± 2.7 b
12	B 70.2 ± 2.7 a	B 23.5 ± 2.7 b
24	AB 71.9 ± 2.7 a	B 21.4 ± 2.7 b
48	A 83.3 ± 2.7 a	A 33.8 ± 2.7 b
ANOVA ^d		
df	4, 344	
F	31	
P>F	0.0001	

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column preceded by the same upper case letter were not different according to least squares means contrasts for $\alpha = 0.05$. Mean separations were based on statistics that used natural logarithm transformed data.

^c Means ± standard errors in each row followed by the same lower case letter were not different according to least squares means contrasts for $\alpha = 0.05$. Mean separations were based on statistics that used natural logarithm transformed data.

^d Analysis of variance table for the interaction effect when transformed data were used.

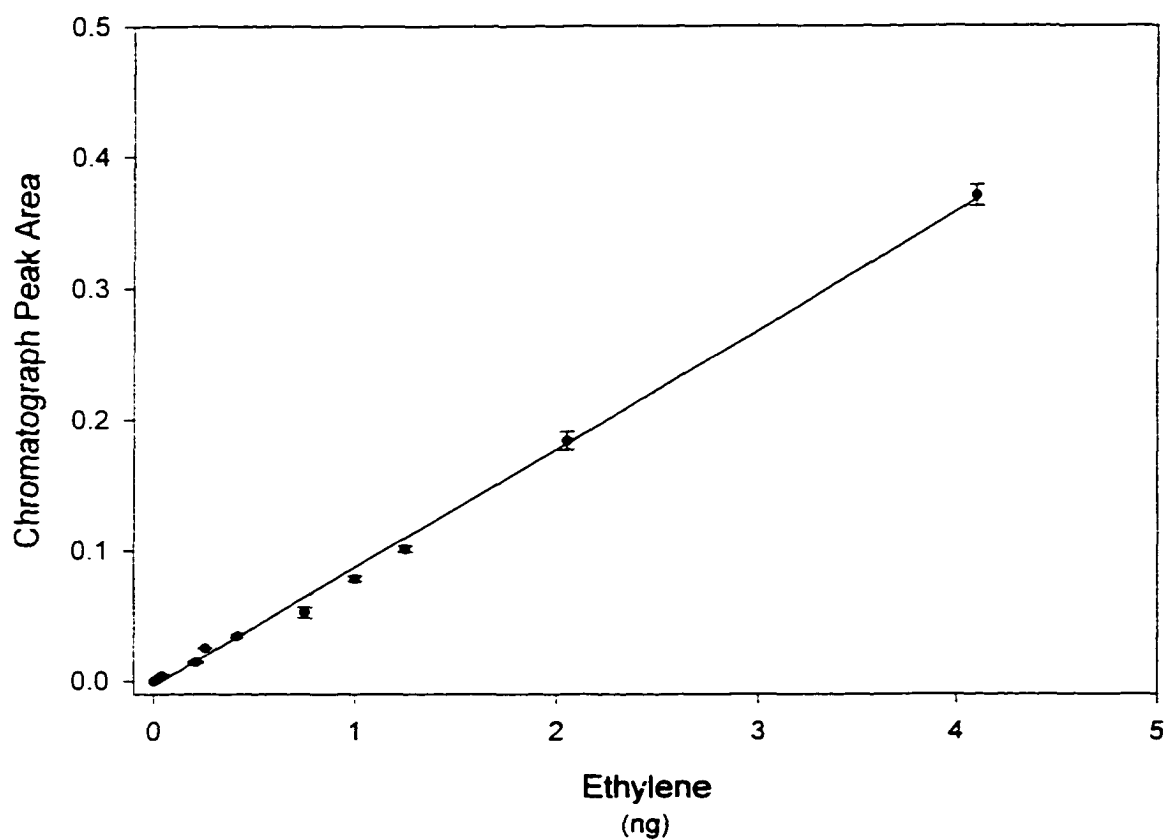


Figure 3.1. Calibration curve for ethylene on the 5890 Hewlett-Packard gas chromatograph equipped with a flame-ionization detector. Values were the means $\pm$ standard errors from three experiments. Regression fit a line to the data, equation $y = 0.0904 x - 0.0034$, with an adjusted R^2 value of 0.99.

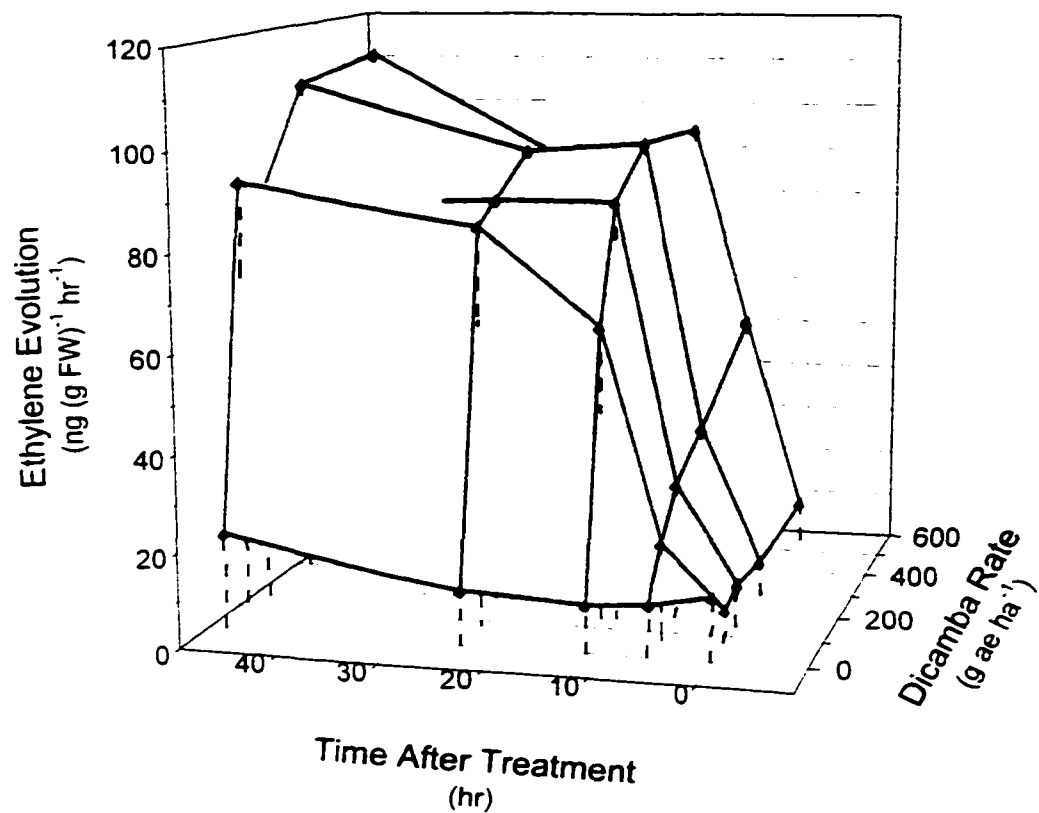


Figure 3.2. Combined effects of dicamba rate and time after herbicide treatment on ethylene evolution from the S2 accession of *K. scoparia*. Values of the mesh-plot vertices were the mean of eight replications from two experiments.

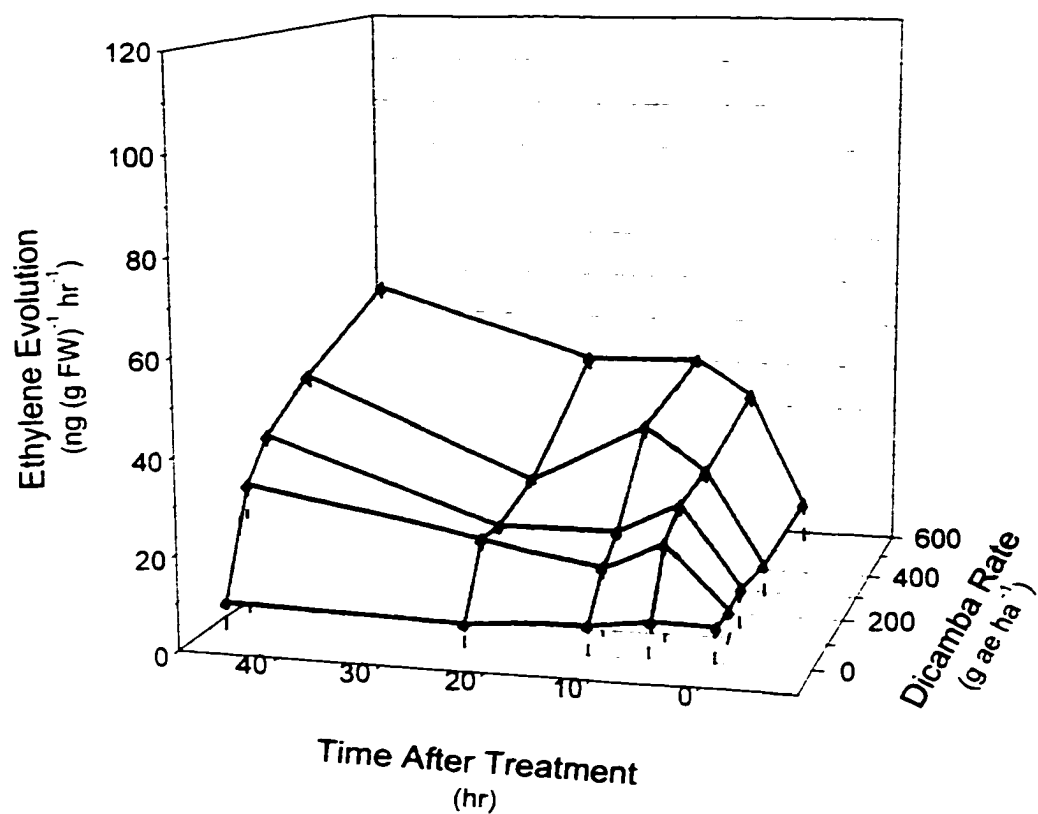


Figure 3.3. Combined effects of dicamba rate and time after herbicide treatment on ethylene evolution from the Henry accession of *K. scoparia*. Values of the mesh-plot vertices were the mean of eight replications from two experiments.

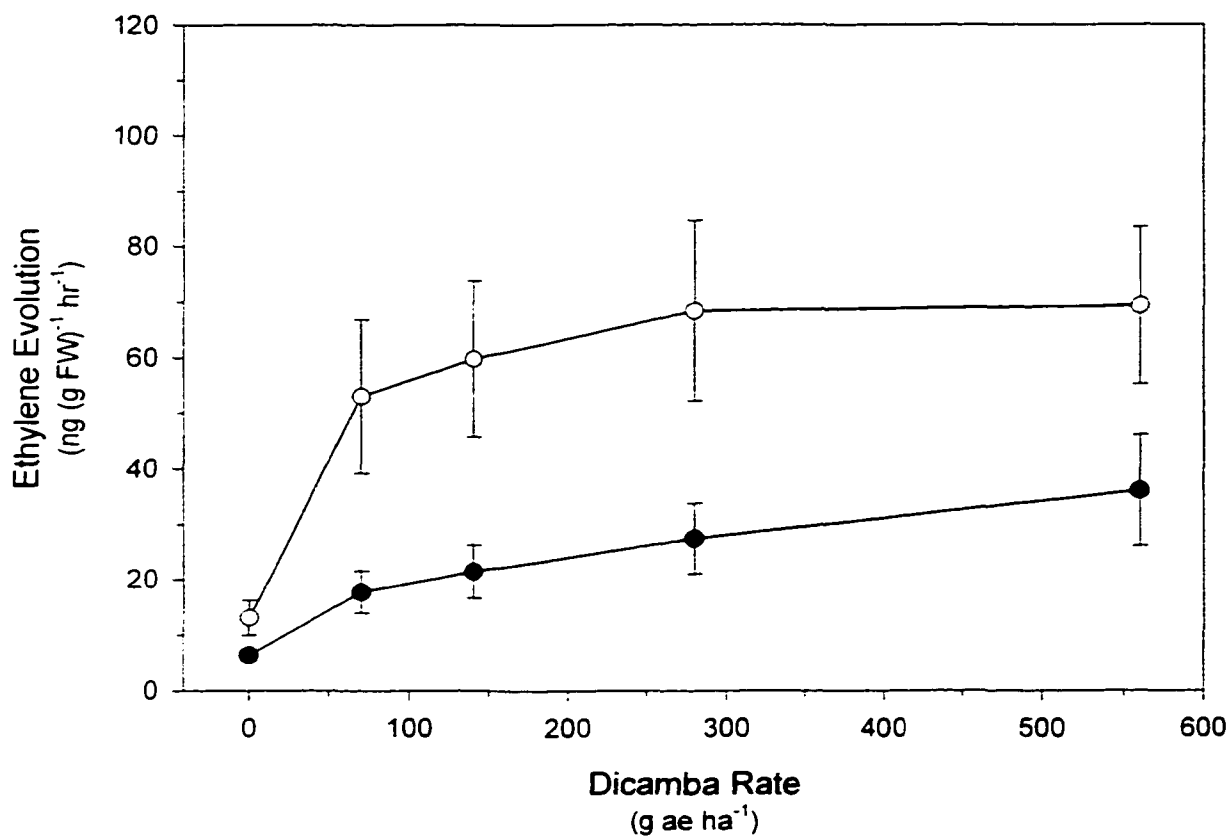


Figure 3.4. Dicamba rate effect on ethylene evolution from the S2 accession (open circles) and from the Henry accession (closed circles) averaged across time points. Values were the means $\pm$ standard errors of eight replications from two experiments.

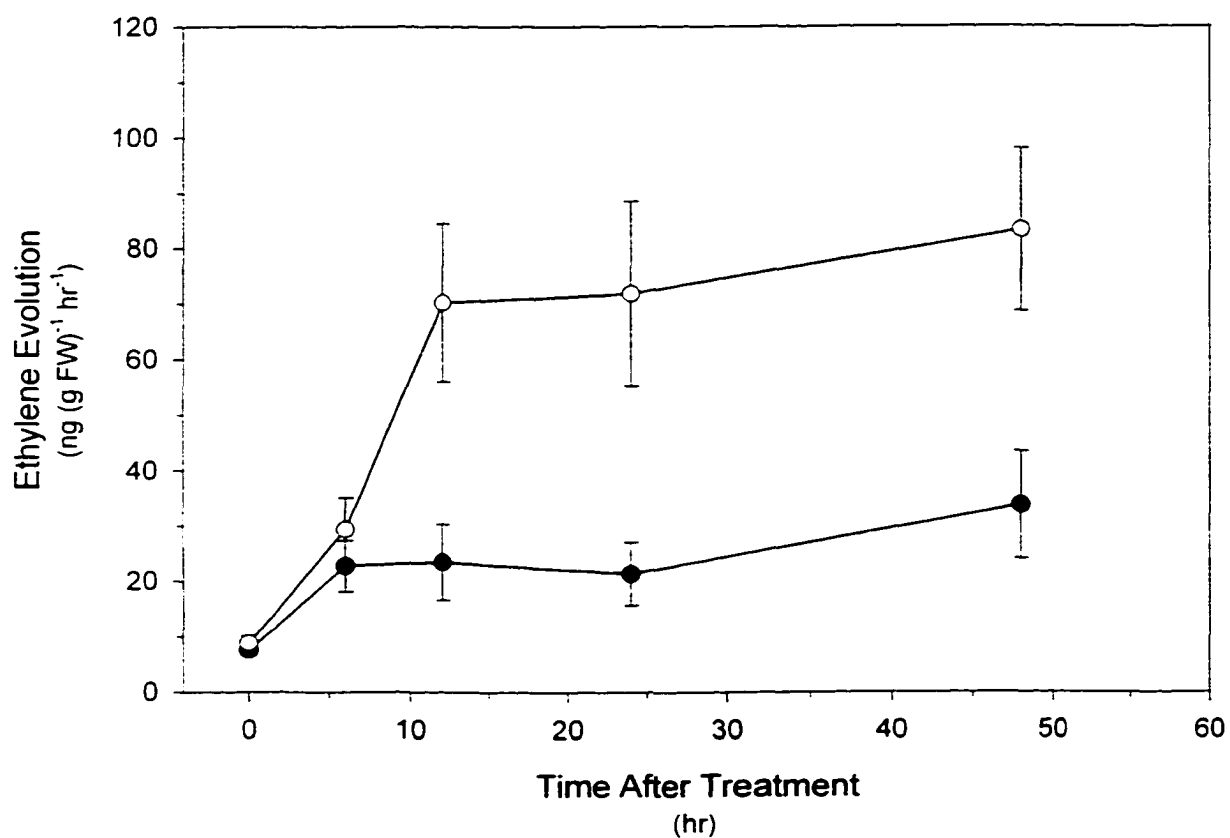


Figure 3.5. Time after herbicide treatment effect on ethylene evolution from the S2 accession (open circles) and from the Henry accession (closed circles) averaged across dicamba rates. Values were the means $\pm$ standard errors of eight replications from two experiments.

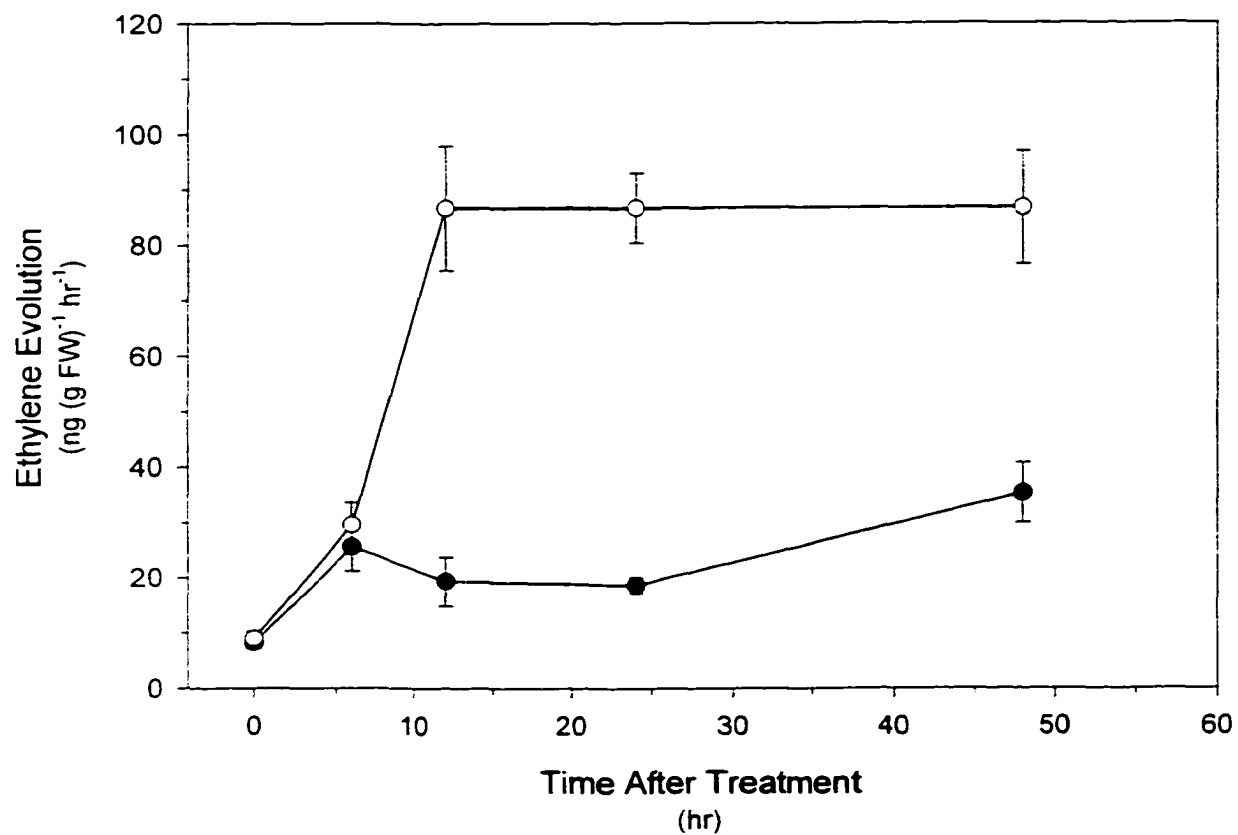


Figure 3.6. Time after herbicide treatment effect on ethylene evolution from the S2 accession (open circles) and from the Henry accession (closed circles) treated with 140 g ae ha⁻¹ dicamba. Values were the means $\pm$ standard errors of eight replications from two experiments.

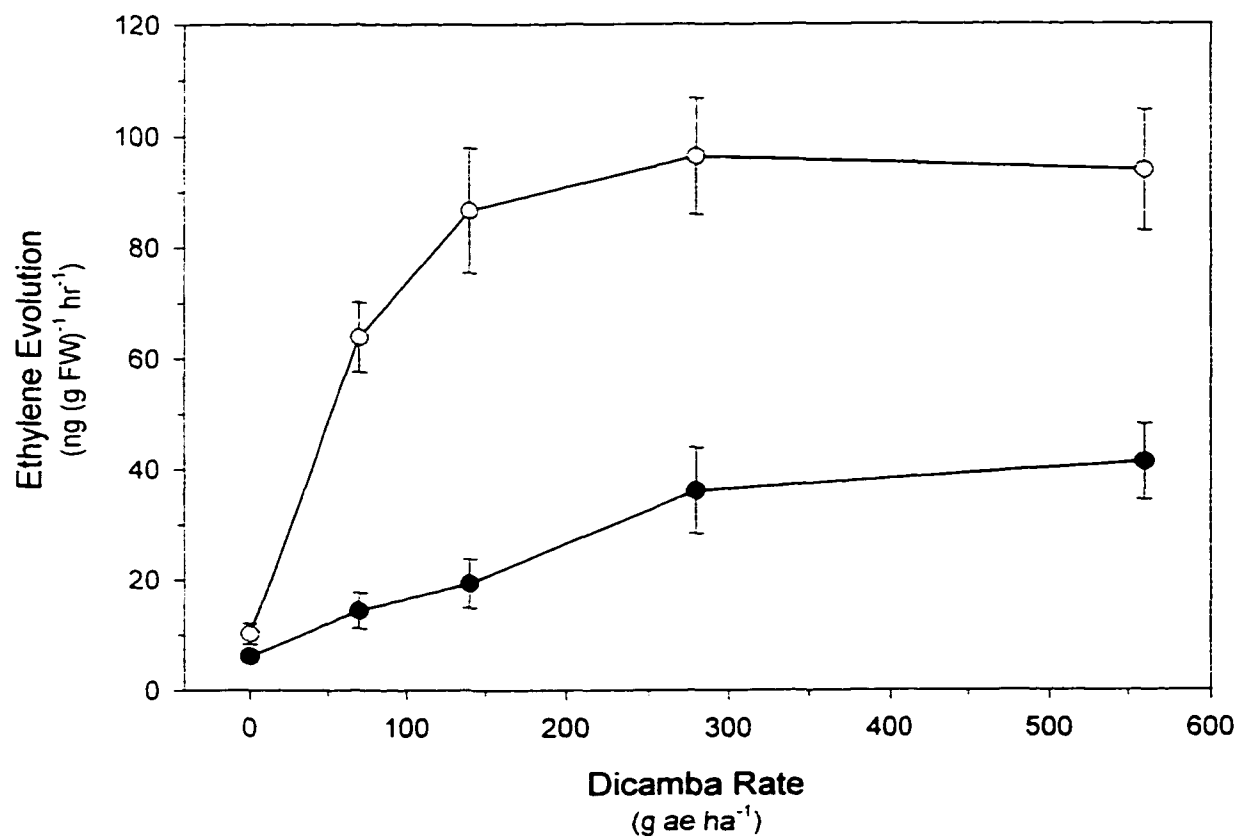


Figure 3.7. Dicamba rate effect on ethylene evolution from the S2 accession (open circles) and from the Henry accession (closed circles) at 12 hours after dicamba treatment. Values were the means $\pm$ standard errors of eight replications from two experiments.

CHAPTER 4
EFFECTS OF WEED MANAGEMENT ASPECTS ON DICAMBA-
SUSCEPTIBLE AND NON-SUSCEPTIBLE KOCHIA

ABSTRACT

Experiments were conducted to examine the effects of several aspects of kochia management on accessions of kochia that differed in response to dicamba. Differences in plant injury among five kochia accessions were inconsistent across twelve herbicide treatments. Bromoxynil provided the highest (97% injury) and most consistent injury across kochia accessions and growth stages. Plant injury decreased as kochia height at spray application increased, but injury to the accessions did not differ within plant height. Kochia accessions expressed more injury as dicamba rate was increased, but the accessions differed in the degree of injury due to post-emergence dicamba application. All kochia accessions were very susceptible (over 93% injury) to pre-emergence dicamba application regardless of response to post-emergence dicamba application. Plants within an accession were similarly affected by post-emergence dicamba exposure to roots or shoots. Kochia injury was not affected by spray volume. Crop competition reduced kochia above ground biomass

production in one season by 91%, but dicamba application was necessary to reduce kochia plant population.

INTRODUCTION

Weed management systems include several components. Many herbicides are effective on typical kochia populations (Alley and Humburg 1980; Bell et al. 1972; Blacksaw 1990; Brattain and Fay 1980; Nalewaja et al. 1990). It is not known how the atypical kochia populations respond to herbicides other than dicamba. Cross-resistance or multiple-resistance may significantly reduce the usefulness of these herbicides.

While many herbicides are available for kochia control, application factors can affect the efficacy of any herbicide. These include many aspects, one of which is spray volume. Spray volume is necessary to provide adequate coverage of the plant. Very dense stands of weeds that result in plant overlap can essentially reduce the amount of chemical reaching an individual plant. Lower application volumes may be favored because of the saved time or economic advantage, but should not be used at the expense of weed control. The growth stage of weeds at the time of application can greatly affect herbicide efficacy. Performance problems can arise if the label recommendations are not followed. This is a factor that can cause weed escapes, which are often mistaken as resistant plants. An informal survey of growers and chemical applicators revealed that they often do not know the growth stage of the weeds

that were sprayed. More often treatments are applied on the basis of crop stage and the window of opportunity for field application.

Crop selection may be used in a weed management program to facilitate herbicide rotation or take advantage of competition. Competition is a significant factor affecting plant growth that can be used to reduce the effects of weeds. While kochia is an efficient competitor, crops such as barley can effectively compete with kochia (Black et al. 1969; Dahl et al. 1982; Scragg and McElvie 1976). The result is reduced growth and vigor of both plants; however, the added effect of a selective herbicide may significantly reduce the competitiveness of the weed.

Experiments were established in the field and greenhouse to examine aspects of kochia management. The purposes of these experiments were to determine the effects of alternative herbicides, kochia growth stage, organ of dicamba exposure, spray volume, and crop competition on dicamba-susceptible and non-susceptible kochia accessions.

MATERIALS AND METHODS

Multiple Herbicide Screening.

Plant Material. Kochia seeds used in these studies were obtained from five different collections that were maintained at the Colorado State University - Weed Research Facility, Fort Collins, Colorado 80523-1177. The inclusion of these five kochia accessions enabled examination of kochia that exhibited a

range of response to dicamba herbicides as determined by previous greenhouse experiments. Dicamba at 140 g ae ha⁻¹ provided 95% control of highly susceptible accessions 94-7 (S1) and 95-8 (S2). The same rate of dicamba provided 50% control of accessions 93-82 (T1) and 93-145 (T2) and less than 40% control of the Sato accession.

Greenhouse Experiment.

Design. Experiments were conducted under greenhouse conditions. Temperature ranged from 25° C to 29° C with a day:night cycle of 16:8 hr. Sodium halide lamps supplemented day length. The experimental design was a randomized complete block with three replications. A factorial layout with three categories, accession by growth stage by herbicide, was used to examine treatment combinations. Five kochia accessions were included. There were three levels of growth stage and 13 herbicide treatments. The greenhouse experiment was repeated. Experiments were initiated in the summer of 1997 and the spring of 1999.

The experimental unit was a flat (model A 12-01, East Jordan Plastics, Inc., P.O. Box 575, East Jordan, Minnesota 49727) 10 cm wide by 15 cm long by 5 cm deep. The flat was filled with Sunshine® potting mix #1 (Sun Gro Horticulture, Inc., 15831 N.E. 8th Street, Bellevue, Washington 98008) to 0.5 cm of the top. Approximately 15 seeds of one kochia accession were distributed on top of the potting mix across each flat area. Potting mix was sprinkled over the seeds so they were buried by approximately 0.3 cm of potting mix. Flats were kept moist with overhead irrigation and fertilized every two weeks with 100 ml of

Miracle-Gro® (Scotts Miracle-Gro Products, Inc., P.O. Box 888, Port Washington, New York 11050) solution. The fertilizer solution was prepared by dissolving 3.5 g granular fertilizer (analysis 15-30-15) in 3.8 L water.

Growth Stages. Herbicide treatments were applied to each kochia accession at three growth stages. These growth stages were at kochia heights of 5 cm, 10 cm, and 15 cm. At 5 cm, plants had 8 to 14 true leaves. Vegetative buds in the leaf axils were present on plants at the 10-cm growth stage, and 15-cm tall plants had branches of various lengths. Each herbicide treatment was applied to separate kochia flats for the three growth stages.

Two days before herbicide application, plants were thinned from the flats. Five plants of similar size at the desired growth stage were left in each flat. Plants were removed such that the remaining plants were distributed across the flat area. This thinning technique increased the similarity of plant health and growth stage within and between experimental units, enabled a consistent plant stand, and maximized spray coverage.

Herbicide Treatments. Herbicide treatments were administered with a chain-driven chamber sprayer. The sprayer was calibrated to deliver 103 L ha⁻¹ by compressed air through an 8001 even flat fan tip. Herbicide treatments included an untreated control, 420 g ai ha⁻¹ bromoxynil, 840 g ai ha⁻¹ bromoxynil+MCPA (1:1), 1053 g ai ha⁻¹ pyridate with 2.3 L ha⁻¹ crop oil, 17 g ai ha⁻¹ and 26 g ai ha⁻¹ carfentrazone-ethyl with 4.7 L ha⁻¹ of a 28% urea ammonium nitrate solution, 1064 g ae ha⁻¹ 2,4-D ester, 140 g ae ha⁻¹ and 560 g ae ha⁻¹ dicamba, 140 g ae ha⁻¹ and 420 g ae ha⁻¹ fluroxypyr, 1400 g ai ha⁻¹

atrazine with 2.3 L ha⁻¹ crop oil, and 26 g ai ha⁻¹ chlorsulfuron with 0.25% V V⁻¹ non-ionic surfactant.

Evaluation and Harvest. Evaluations and harvests were conducted 28 days after herbicide treatments. Each plant was visually evaluated for percent injury from 0% (no injury) to 100% (complete death) compared to control plants. The evaluation scores for the five plants of an experimental unit were averaged to yield a single measure of visual injury for each flat. Fresh weight of all five plants was recorded at harvest and dry weight of all five plants was recorded following oven drying.

Data Analyses. Data were analyzed using procedures for mixed effects models in SAS programming software (SAS Institute 1989). Bartlett's test for variance homogeneity indicated that data from each of the experiments could be pooled (df (538, 539), $F = 1.09$, $P = 0.33$). Standard diagnostic inquiries indicated no violations of the assumptions underlying analysis of variance testing. Mean separation was performed using single degree of freedom contrasts via the least squares means (lsmeans) statement in the mixed effects procedure of SAS (SAS Institute 1989). Pearson Correlation Coefficients were used to correlate objective measures of fresh and dry weights to the more subjective measure of plant injury (SAS Institute 1989).

Field Experiment.

Design. Two field experiments were planted to complement results obtained in the greenhouse; however, kochia was only established in one of these experiments. Plots were established in the spring of 1997 at the Colorado

State University - Agricultural Research, Development, and Education Center (ARDEC) located north of Fort Collins, Colorado, at the intersection of Larimer County Road 54 and the east frontage road of Interstate 25. The soil at ARDEC was a Fort Collins loam, Ustollic Haplargid (Fort Collins series) with pH = 7.9 and 2.2% organic matter.

The experimental design was a randomized complete block in a split-block arrangement with three replications. Plots were 3 m wide by 3 m long. Factorial combinations of the three growth stages with the 13 herbicide treatments described in the greenhouse experiments were randomized within replication. The five kochia accessions described in the greenhouse experiments were blocked within the growth stage by herbicide treatment layout.

Kochia seeds were planted 0.5 cm deep into a prepared seedbed. Seeds from each kochia accession were planted in separate rows that ran through each plot in a tier. Rows were spaced on 60-cm centers. Within a row, five to seven seeds were planted on 30-cm centers. Before herbicide application, plants were thinned to reduce plant overlap and improve the consistency of plant size within a growth stage.

Herbicide Treatments. Herbicide treatments were applied with a CO₂ backpack sprayer. Spray volume was calibrated at 120 L ha⁻¹ delivered through 11001 flat fan tips. Herbicide application was conducted on the 2nd of July in 4 km hr⁻¹ to 6 km hr⁻¹ wind which traveled parallel to the direction of spraying. The air temperature was 19° C with 42% relative humidity under a cloudless sky. Soil

temperature was 15° C and soil moisture content was excellent. Herbicide treatments were consistent with those applied in the greenhouse experiment.

Weather conditions were monitored by the Colorado Agriculture Meteorological Network at the ARDEC facilities. Weather data were included in *Appendix 4.A*.

Evaluation. Plants were evaluated for visual injury 28 days after herbicide applications. A single injury score was assigned to each kochia accession within a growth stage by herbicide plot. Plant injury was rated compared to control plants.

Data Analyses. Data were analyzed using procedures for mixed effects models in SAS programming software (SAS Institute 1989). Standard diagnostic inquiries indicated no violations of the assumptions underlying analysis of variance testing. Mean separation was performed using single degree of freedom contrasts via the least squares means (lsmeans) statement in the mixed effects procedure of SAS (SAS Institute 1989).

Plant Organ Exposed to Dicamba.

Design. Experiments were conducted under greenhouse conditions. Temperature ranged from 25° C to 29° C with a day:night cycle of 16:8 hr. Sodium halide lamps supplemented day length. The experimental design was a randomized complete block with three replications. A factorial layout with four categories, soil by accession by organ exposed by herbicide, was used to examine treatment combinations. There were two field soils used in the

experiment, and two kochia accessions were used as levels of accession. Four plant organs were investigated and three herbicide treatments were used. The experiment was repeated.

Soils. The field soils used in this experiment differed in texture and organic matter to represent a range of Colorado soil types. A sandy loam soil was obtained from a field near Nunn, Colorado. This soil contained 1.2% organic matter and had pH = 7.6. The other soil was a clay loam from a field near Fort Collins, Colorado. The clay loam soil contained 2.1% organic matter and had pH = 7.9. The soils were sieved through a mesh screen with one-cm wire spacing and allowed to air dry.

Plant Material. Kochia seeds used in this study were obtained from two collections that were maintained at the Colorado State University - Weed Research Facility, Fort Collins, Colorado 80523-1177. These two accessions were chosen because they represent a wide range of natural response to dicamba. In previous experiments, accession 95-8 (S2) was very susceptible to an application of 140 g ae ha⁻¹ dicamba while the accession denoted as Henry showed fewer injury symptoms and had a higher survival rate.

The experimental unit was a flat (model A 12-01, East Jordan Plastics, Inc., P.O. Box 575, East Jordan, Minnesota 49727) 10 cm wide by 15 cm long by 5 cm deep. Soils were mixed with slow release fertilizer (analysis 17-6-12 plus minors, Scotts-Sierra Horticultural Products Co., 14111 Scottslawn Road, Marysville, Ohio 43041) at a rate of 15 g fertilizer kg⁻¹ soil. The flat was filled with 800 g of one of the air-dried soil mixtures. A shallow furrow was made

lengthwise in the center of the flat. Thirty seeds of one kochia accession were counted and distributed the length of the furrow. The furrow was then closed which buried the kochia seeds approximately 0.4 cm deep. Flats were sub-irrigated to field capacity and then irrigated with overhead sprinklers to prevent soil crusting. Periodic sub-irrigation replenished soil moisture.

Plant Organs Exposed. Four organs were exposed to herbicide in this experiment. These organs represented the area of the plant that was directly exposed to the herbicide treatments. The first treatment included the emerging radicals, hypocotyls, and cotyledons (germinating seeds). This was achieved with a pre-emergence application of herbicide within six hours of planting. The remaining treatments were applied to plants that had true leaves. The second organ investigated was herbicide exposure to the roots only (roots). The above ground portion of plants in this group was protected from herbicide application by plastic bags that were placed over the top of the plants and held close to the base of the plants by toothpicks. After the spray solution dried, the plastic bags were removed. The third exposure treatment involved applying dicamba only to the stems and leaves of plants (shoots). Herbicide was excluded from the soil environment by a one cm thick layer of vermiculite. After the spray solution dried, the vermiculite was removed. The fourth organ of exposure was the entire plant (roots and shoots). No barriers were placed on the plant environment, which enabled herbicide exposure via roots, stems, and leaves.

Herbicide Treatments. Herbicide treatments were administered with a chain-driven chamber sprayer. For pre-emergence applications, the sprayer was

calibrated to deliver 187 L ha⁻¹ by compressed air through an 8001 even flat fan tip. Herbicide treatments included an untreated control, 140 g ae ha⁻¹ dicamba, and 280 g ae ha⁻¹ dicamba. Pre-emergence applications were incorporated with irrigation. The number of plants emerging from pre-emergence treatments was recorded every two days for two weeks after planting. This determined the number of seeds that were germinable; and therefore, the number of plants exposed to the herbicide.

Two days before post-emergence herbicide application, plants were thinned from the flats to reduce interference between plants and maximize spray coverage. The actual number of plants that received herbicide treatment was recorded for each flat. Kochia plants were 2 cm to 5 cm tall at the time of post-emergence herbicide application.

Post-emergence herbicide treatments were administered with the chain-driven chamber sprayer calibrated to deliver 98 L ha⁻¹ by compressed air through an 8001 even flat fan tip. Herbicide treatments included an untreated check, 140 g ae ha⁻¹ dicamba, and 280 g ae ha⁻¹ dicamba.

Evaluation. Plants were evaluated 28 days after herbicide application. This evaluation assessed plant injury. Each plant in a flat was given a score for percent injury from 0% (no injury) to 100% (complete death) compared to control plants. Scores for all plants in a flat were averaged to provide a single rating of plant injury.

Data Analyses. Data were analyzed using procedures for mixed effects models in SAS programming software (SAS Institute 1989). Bartlett's test for

variance homogeneity indicated that data from each of the experiments could be pooled (df (92, 95), $F = 1.45$, $P = 0.076$). Standard diagnostic inquiries indicated no violations of the assumptions underlying analysis of variance testing. Mean separation was performed using single degree of freedom contrasts via the least squares means (lsmeans) statement in the mixed effects procedure of SAS (SAS Institute 1989).

Spray Volume.

Design. Experiments were conducted under greenhouse conditions. Temperature ranged from 25° C to 29° C with a day:night cycle of 16:8 hr. Sodium halide lamps supplemented day length. The experimental design was a randomized complete block with three replications. A factorial layout with three categories, accession by spray volume by herbicide, was used to examine treatment combinations. Two kochia accessions were used as levels of accession. Three spray volumes were investigated and three herbicide treatments were used. The experiment was repeated.

Plant material. Kochia seeds used in this study were obtained from two collections that were maintained at the Colorado State University - Weed Research Facility, Fort Collins, Colorado 80523-1177. These two accessions were chosen because they represent a wide range of natural response to dicamba. In previous experiments, accession 95-8 (S2) was very susceptible to an application of 140 g ae ha⁻¹ dicamba while the accession denoted as Henry showed fewer injury symptoms and had a higher survival rate.

The experimental unit was a flat (model A 12-01, East Jordan Plastics, Inc., P.O. Box 575, East Jordan, Minnesota 49727) 10 cm wide by 15 cm long by 5 cm deep. The flat was filled with Sunshine® potting mix #1 (Sun Gro Horticulture, Inc., 15831 N.E. 8th Street, Bellevue, Washington 98008) to 0.5 cm of the top. Approximately 30 seeds of one kochia accession were distributed on top of the potting mix across each flat area. Potting mix was sprinkled over the seeds so they were buried by approximately 0.3 cm of potting mix. Flats were kept moist with overhead irrigation and fertilized every two weeks with 100 ml of Miracle-Gro® (Scotts Miracle-Gro Products, Inc., P.O. Box 888, Port Washington, New York 11050) solution. The fertilizer solution was prepared by dissolving 3.5 g granular fertilizer (analysis 15-30-15) in 3.8 L water.

Two days before herbicide application, plants were thinned from the flats to reduce interference between plants and maximize spray coverage. Flats were thinned to a population of 20 plants except in instances where leaving that many plants would compromise adequate spray coverage. The actual number of plants that received herbicide treatment was recorded for each flat. Kochia plants were 3 cm to 5 cm tall at the time of herbicide application.

Herbicide Treatments. Herbicide treatments were administered with a chain-driven chamber sprayer. Flats received an appropriate herbicide dose in one of three spray volumes. The sprayer was calibrated to deliver 51 L ha⁻¹, 98 L ha⁻¹, and 196 L ha⁻¹ by compressed air through an 8001 even flat fan tip. Herbicide treatments included an untreated control, 140 g ae ha⁻¹ dicamba, and 280 g ae ha⁻¹ dicamba.

Evaluation. Plants were evaluated 28 days after herbicide application.

This evaluation assessed plant injury. Each plant in a flat was given a score for percent injury from 0% (no injury) to 100% (complete death) compared to control plants. Scores for all plants in a flat were averaged to provide a single rating of plant injury.

Data Analyses. Data were analyzed using procedures for mixed effects models in SAS programming software (SAS Institute 1989). Bartlett's test for variance homogeneity indicated that data from each of the experiments could be pooled (df (35, 35), $F = 1.08$, $P = 0.82$). Standard diagnostic inquiries indicated no violations of the assumptions underlying analysis of variance testing. Mean separation was performed using single degree of freedom contrasts via the least squares means (lsmeans) statement in the mixed effects procedure of SAS (SAS Institute 1989).

Interaction of Herbicide and Crop Competition.

Design. Experiments were established in the fall of 1995 and 1996 at the Colorado State University - Agricultural Research, Development, and Education Center (ARDEC) located north of Fort Collins, Colorado, at the intersection of Larimer County Road 54 and the east frontage road of Interstate 25. The soil at ARDEC was a Fort Collins loam, Ustollic Haplargid (Fort Collins series) with pH = 7.9 and 2.2% organic matter.

The experimental design was a randomized complete block in a split-block arrangement with three replications. Plots were 3 m wide by 4 m long. The

presence or absence of wheat was blocked within replication. Factorial combinations of two kochia accessions and three herbicide treatments were randomized within wheat treatment blocks.

Plant Material. Winter wheat cv. TAM 107 was planted into the split-block arrangement at a seeding rate of 45 kg ha⁻¹. Fertilizer was applied to the entire study area. Granular fertilizer (analysis 46-0-0) was broadcast at 56 kg ha⁻¹ nitrogen. This application occurred after planting and immediately preceding the next precipitation event for incorporation. Blocks that were not to have winter wheat were kept free of undesired plants by mechanical methods.

Kochia accessions used in this study were obtained from two collections that were maintained at the Colorado State University - Weed Research Facility, Fort Collins, Colorado 80523-1177. These accessions were chosen because they represent a wide range of natural response to dicamba. In previous experiments, accession 95-8 (S2) was very susceptible to an application of 140 g ae ha⁻¹ dicamba while the accession denoted as Sato showed fewer injury symptoms and had a higher survival rate. In the spring of 1996, kochia plants were started in Jiffy-7® peat pellets (Jiffy Products, 951 Swanson Drive, Batavia, Illinois 60510) in the greenhouse. Plants were transplanted into the appropriate field plots when the risk of freezing temperatures was low. In the spring of 1997, kochia seeds were planted directly into the proper plots. Seeds were planted 0.5 cm deep into a prepared seedbed. Ten plants were monitored in each plot. In both years, desired kochia plants were marked with flags to discern treatment kochia plants from the native population.

Herbicide Treatments. Herbicide treatments were applied with a CO₂ backpack sprayer. Spray volume was calibrated at 120 L ha⁻¹ delivered through 11001 flat fan tips. Treatments included an untreated control, 70 g ae ha⁻¹ dicamba, and 140 g ae ha⁻¹ dicamba. In 1996, herbicide application was conducted on the 13th of May in 1 km hr⁻¹ to 3 km hr⁻¹ wind. The air temperature was 19° C with 67% relative humidity under a partly cloudy sky (estimated 5% cloud cover). Soil temperature was 12° C and soil moisture content was good. Kochia plants were 2 cm to 4 cm tall, and wheat was in the boot stage, 20 cm to 25 cm in height. In 1997, herbicide application was conducted on the 20th of May in no wind. The air temperature was 11° C with 80% relative humidity under a cloudless sky. Soil temperature was 10° C and soil moisture was good. Kochia plants were 1 cm to 2 cm tall, and wheat was just reaching the jointing stage, 10 cm to 25 cm in height.

Weather conditions were monitored by the Colorado Agriculture Meteorological Network at the ARDEC facilities. Weather data were included in *Appendix 4.A*.

Evaluation and Harvest. Plants were evaluated for visual injury 28 days after herbicide applications. A single injury score from 0% (no injury) to 100% (plant death) was assigned to each plot. Plant injury rating was comparative to control plants. At grain maturity, the height and number of surviving kochia plants was recorded. Kochia plants were harvested at the soil surface for fresh weight measurements and dry weights were obtained after oven drying.

Data Analyses. Data were analyzed using procedures for mixed effects models in SAS programming software (SAS Institute 1989). Bartlett's tests for variance homogeneity indicated that data from each of the experiments could be pooled for plant injury (df (23, 23), $F = 1.24$ $P = 0.61$) and for plant survival (df (35, 35), $F = 1.63$, $P = 0.16$). Bartlett's tests also indicated that plant height (df (35, 35), $F = 2.61$, $P = 0.0056$) and plant dry weight data (df (35, 35), $F = 1600$, $P = 0.0001$) could not be pooled between experiments. Standard diagnostic inquiries of plant height and weight within year and combined data for surviving plants and plant injury indicated no violations of the assumptions underlying analysis of variance testing. Mean separation was performed using single degree of freedom contrasts via the least squares means (lsmeans) statement in the mixed effects procedure of SAS (SAS Institute 1989).

RESULTS AND DISCUSSION

Multiple Herbicide Screening.

Correlation between visual kochia injury and harvest weight was interpreted to determine the most reasonable parameter of plant response. While there was a fairly strong relationship between injury and harvest weight (Pearson's $r = -0.76$, $P = 0.0001$), there were some inconsistencies that complicated the use of harvest weight. These inconsistencies resulted from inherent symptomology differences between auxinic herbicides and other herbicides included in the experiments. Therefore it was decided that the visual

injury evaluation was a better description of the actual plant response than harvest weight.

Kochia injury was dependent on kochia accession for greenhouse (df (4, 897), $F = 4.34$, $P = 0.0018$, Table 4.1) and field experiments (df (4, 331), $F = 21$, $P = 0.0001$). While mean separation grouping changed the ranking of accessions from most injured to least injured between the experiments, there were only slight differences in the absolute values of kochia injury for accessions within an experiment or between experiments for an accession. These differences were probably of no practical use. The large experiment size enabled separation of means with small differences that would not have meaning in production situations.

The growth stage of kochia at herbicide application greatly influenced plant injury in the greenhouse (df (2, 879), $F = 86$, $P = 0.0001$, Table 4.2) and field (df (2, 5), $F = 67$, $P = 0.0003$). For both experiments, kochia responded to each increase in growth stage with a decrease in injury (df (1, 5), $t = 3.9$, $P = 0.012$, minimum difference). Plants growing under field conditions tended to be more sensitive to herbicides.

Herbicide treatment also greatly affected plant injury (Table 4.3), especially in the greenhouse (df (11, 897), $F = 89$, $P = 0.0001$). The effects of most herbicide treatments were moderately consistent from the greenhouse to the field. While mean separation groups differed between experiments, the injury rating for most herbicides was stable. Some notable increases in herbicide effect from the greenhouse to the field include pyridate which increased from 83%

injury to 92% injury and both carfentrazone-ethyl treatments which increased by more than 50% of their original injury rating. The most puzzling, however, was atrazine. Plant injury from atrazine treatment increased from 81% injury in the greenhouse to 99% injury in the field, even though two of the accessions contained atrazine resistant seed. Not all treatments elicited higher injury in the field. Kochia injury caused by bromoxynil + MCPA decreased from 95% injury in the greenhouse to 82% injury in the field.

There were interactions in both experiments between growth stage and herbicide treatment which were illustrated with the greenhouse data (df (22, 897), $F = 9.06$, $P = 0.0001$, Table 4.4). Generally, plant response to systemic herbicides such as dicamba and atrazine was more consistent across growth stages than plant response to contact herbicides such as pyridate and carfentrazone-ethyl. This seemed to be particularly true of auxinic herbicides and exemplified by the 560 g ae ha⁻¹ dicamba treatment (df (1, 993), $t = 1.77$, $P = 0.076$). Plant injury from contact herbicides tended to decrease as growth stage increased. Kochia injury from pyridate decreased 13 percentage points from the 5-cm to the 10-cm growth stages (df (1, 993), $t = 2.82$, $P = 0.0050$) and 21 percentage points between the 10-cm and 15-cm growth stages (df (1, 993), $t = 4.81$, $P = 0.0001$).

There was also an interaction between kochia accession and herbicide treatment (df (44, 897), $F = 4.51$, $P = 0.0001$). Greenhouse data were again used to demonstrate the nature of this interaction (Table 4.5). This interaction is quite intricate because the five accessions responded differently to individual

herbicide treatments. Accessions have the same injury rating for some treatments. This was true with bromoxynil (df (1, 993), $t = 0.38$, $P = 0.71$). In the pyridate treatment, accessions T1 and T2 had the highest injury (df (1, 993), $t = 3.0$, $P = 0.0027$), but these same accessions had the lowest injury when treated with atrazine (df (1, 993), $t = 6.34$, $P = 0.0001$). And the Sato accession had a lower injury rating than accessions S1 and S2 in the dicamba treatment (df (1, 993), $t = 2.33$, $P = 0.020$).

The response of Sato accession kochia in the greenhouse experiment was not different from the response of accessions S1 or S2 to either rate of fluroxypyr (df (1, 993), $t = 0.38$, $P = 0.71$, Table 4.5). Even though Sato accession injury from fluroxypyr tended to be lower than the S1 or S2 accession injury, the results do not support cross-resistance between dicamba and fluroxypyr for the Sato accession. In the field, however, the Sato accession was less injured than the S1 accession, 80% and 93% respective injury, when treated with 140 g ae ha⁻¹ fluroxypyr (df (1, 427), $t = 2.98$, $P = 0.0030$, Table not shown). This difference supports cross-resistance between dicamba and fluroxypyr. The amount of injury difference between susceptible and resistant expression is greater for dicamba than fluroxypyr. This was supported by visual observations in non-reported field experiments. In these field experiments, when injury from dicamba was low, injury from fluroxypyr was also low, but the fluroxypyr treatment tended to be more injured than the dicamba treatment.

In the greenhouse experiment, accessions T1 and T2, which previously were determined to express low levels of injury, did not differ from accessions S1

and S2 for either rate of dicamba. In the field however, accessions T1 and T2 were less injured than accession S2 at both rates of dicamba (df (1, 427), $t = 2.34$, $P = 0.019$, Table not shown).

Plant Organ Exposed to Dicamba.

Soil had a slight effect on observed plant injury in this experiment (df (1, 175), $F = 9.79$, $P = 0.021$). While significant, this difference was small. Plants growing in the Nunn soil had an injury rating of 85% while plants growing in the Fort Collins soil exhibited 88% injury. Due to the small difference in plant injury between soils and the fact that there no significant soil interactions, it was questioned whether this difference had practical meaning. It may be indicating a more substantial difference that could occur if additional soils were investigated.

Kochia accession had a large influence on plant injury (df (1, 175), $F = 180$, $P = 0.0001$). While the S2 accession had an average plant injury rating of 94%, plants from the Henry accession only expressed 79% injury. Plant injury was also effected by dicamba rate. Averaged across accessions and organs exposed, plants were more injured by 280 g ae ha⁻¹ dicamba, 89% injury, than by 140 g ae ha⁻¹ dicamba, 83% injury (df (1, 175), $F = 5.2$, $P = 0.0001$).

The combination of accession and dicamba rate formed an interaction (df (1, 175), $F = 10.69$, $P = 0.0013$, Table 4.6). Injury on plants from the Henry accession was 74% for the 140 g ae ha⁻¹ dicamba treatment. This was lower than the 84% injury exhibited by plants treated with 280 g ae ha⁻¹ dicamba (df (1, 175), $t = 5.94$, $P = 0.0001$). The S2 accession, on the other hand, did not

respond to the dicamba rate increase by expressing more injury, 93% versus 95% (df (1, 175), $t = 1.38$, $P = 0.17$). This resulted from the inherent susceptibility of the S2 accession to dicamba. While the S2 accession tended to respond to the rate increase, the lack of difference between the two treatments caused the interaction effect.

The plant organ of herbicide exposure affected plant injury scores (df (3, 175), $F = 48$, $P = 0.0001$, Table 4.7). Exposure to germinating seeds resulted in the highest plant injury, 96% (df (1, 175), $t = 4.01$, $P = 0.0001$). Injury when plant roots and shoots were exposed to dicamba, 90%, was lower than for exposure to germinating seeds, but exposure to roots and shoots elicited more plant injury than exposure to roots only, 81%, or exposure to shoots only, 79% (df (1, 175), $t = 5.15$, $P = 0.0001$). Plant injury was not different for dicamba exposure to roots only or exposure to shoots only (df (1, 175), $t = 1.55$, $P = 0.12$).

The combination of dicamba treatment and organ of herbicide exposure did not interact (df (3, 175), $F = 2.43$, $P = 0.069$, Table not shown). Terms of this combination, therefore, were used to strengthen information about root and shoot absorption of dicamba. The injury of plants treated with 140 g ae ha⁻¹ dicamba exposed to the roots and shoots, 88%, was not different from plants treated with 280 g ae ha⁻¹ dicamba exposed to the shoots only, 84% (df (1, 175), $t = 1.67$, $P = 0.097$, Table not shown). These treatments were considered to be equivalent on the whole plant level because of the total amount of dicamba entering the plant environment. The injury rating of plants treated with 140 g ae ha⁻¹ dicamba exposed to the roots and shoots was also similar to plants treated with 280 g ae

ha⁻¹ dicamba exposed to the roots only, 85% (df (1, 175), $t = 1.39$, $P = 0.17$, Table not shown). Examination of these treatments by accession gave similar results. These results supported the evidence of equal root and shoot absorption from the plant organ of exposure main effect (Table 4.7).

Plant organ of exposure interacted with kochia accession (df (3, 175), $F = 15.29$, $P = 0.0001$, Table 4.8). As with the interaction of kochia accession and dicamba rate, the interaction between accession and organ of exposure seemed to be due to exceptional sensitivity of the S2 accession to dicamba. There was much less variation in plant injury among organ of exposure treatments for the S2 accession than for the Henry accession. This resulted in similar injury values between the roots and shoots treatment and the germinating seeds treatment and between the roots and shoots treatment and the roots only treatment for the S2 accession. In the Henry accession, both of these comparisons produced differences.

Spray Volume.

Kochia accession had a large effect on the observed plant injury. S2 accession plants expressed more injury than Henry accession plants, 89% compared to 83% injury (df (1, 54), $F = 32$, $P = 0.0001$, Table not shown). The increase in dicamba rate from 140 g ae ha⁻¹ to 280 g ae ha⁻¹ dicamba also resulted in an increase in plant injury, 83% and 89% injury respectively (df (1, 2), $F = 21$, $P = 0.044$). There was, however, no effect of spray volume on plant injury (df (2, 4), $F = 1.20$, $P = 0.39$). Injury of plants ranged from 85% for volumes

of 51 L ha⁻¹ and 98 L ha⁻¹ to 87% injury at the 196 L ha⁻¹ spray volume. There were no significant interactions for this experiment.

Interaction of Herbicide and Crop Competition.

Kochia Height. The height of kochia plants was not effected by kochia accession in either year of this experiment. This was the first experiment in which accession did not result in separation of a parameter response. In the first season, the Sato accession tended to have shorter plants than the S2 accession, 27 cm tall compared to 31 cm tall (df (1, 24), $F = 2.54$, $P = 0.12$). In the second season, plants were much taller. Sato plants were 70 cm in height while S2 accession plants were 67 cm (df (1, 20), $F = 1.29$, $P = 0.27$).

As with the effect of accession on height in the first season, kochia plants growing in winter wheat, 31 cm, were not different from kochia growing without crop competition, 27 cm (df (1, 24), $F = 2.45$, $P = 0.13$). In the second season though, adequate moisture enabled more plant growth and the effect of crop competition was substantial. Kochia that had to compete with wheat was 27% shorter than kochia growing in a relatively non-competitive environment, 58 cm and 79 cm respectively (df (1, 4), $F = 33$, $P = 0.0046$).

Dicamba rate was the only main effect with consistent effect in both seasons (Table 4.9). In the first season, only plants treated with 140 g ae ha⁻¹ dicamba were shorter than plants in the control plots (df (2,24), $t = 3.01$, $P = 0.0061$). Due to compact growth of kochia in this season, the height of plants treated with 70 g ae ha⁻¹ dicamba was not different from the height of control

plants (df (1, 24), $t = 1.07$, $P = 0.29$) or the height of plants treated with 140 g ae ha⁻¹ dicamba (df (1, 24), $t = 1.94$, $P = 0.065$). In the second season, both dicamba rates caused shorter plants than the control (df (1, 20), $t = 4.13$, $P = 0.0005$, minimum difference), but the heights of plants treated with each rate were not different from each other (df (1, 20), $t = 1.29$, $P = 0.21$).

Herbicide rate and crop competition interacted in the second season (df (2,20), $F = 3.65$, $P = 0.045$, Table 4.10). Kochia plants growing with winter wheat were shorter when treated with 140 g ae ha⁻¹ dicamba than control plants (df (1, 20), $t = 4.49$, $P = 0.0002$). There was no difference, however, in height of plants growing non-competitively in the control or 140 g ae ha⁻¹ dicamba plots (df (1, 20), $t = 1.34$, $P = 0.19$). Under both crop competition regimes, the lower rate of dicamba tended to have more influence on plant height than the higher rate.

The combined effects of kochia accession and crop competition produced an interaction in the first season (df (1, 24), $F = 10.07$, $P = 0.0041$, Table 4.11). Two factors generated this interaction. First, only S2 accession plants were shorter in plots with no winter wheat than in plots with winter wheat (df (1, 24), $t = 3.35$, $P = 0.0027$). And second, for kochia growing with crop competition, Sato accession plants were shorter than S2 accession plants (df (1, 24), $t = 2.24$, $P = 0.035$). This interaction was further explained by examining the interaction of kochia accession, crop competition, and dicamba rate (df (2, 24), $F = 4.65$, $P = 0.020$, Table 4.12). Dicamba rate tended to have a greater effect on plant height on the S2 accession when no winter wheat was present but on the Sato accession when plants were growing with winter wheat.

Kochia Harvest Weight. The harvest dry weight per kochia plant was no different between accessions for either season of the experiment. In the first season, the S2 accession tended to have more weight, 1.58 g pl⁻¹ versus 1.00 g pl⁻¹ (df (1, 4), $F = 3.35$, $P = 0.14$). But in the second season, the Sato accession tended to be heavier, 41.82 g pl⁻¹ compared to 31.23 g pl⁻¹ (df (1, 24), $F = 1.92$, $P = 0.18$).

Crop competition varied in effect between seasons (Table 4.13). Winter wheat, severely stunted by growing conditions, had no effect on kochia weight during the first season (df (1, 3), $F = 3.25$, $P = 0.18$). On the contrary, the most important influence on the second season's harvest weight was crop competition (df (1, 24), $F = 63$, $P = 0.0001$). In this season, vigorously growing wheat caused kochia harvest weight to be 91% lower than control plants. The magnitude of this effect masked other main effects in this year of the experiment.

Dicamba rate affected plant weight in the first season (df (2, 17), $F = 9.06$, $P = 0.0020$, Table 4.14). Both dicamba rates caused lower kochia harvest weight than the control treatment (df (1, 17), $t = 3.2$, $P = 0.0052$), but there was no difference in weight between the two rates (df (1, 17), $t = 0.84$, $P = 0.41$).

There were interactions in both seasons that involved dicamba rate effects. In the first season, dicamba rate interacted with crop competition (df (2, 17), $F = 11.94$, $P = 0.0005$, Table 4.15). This interaction resulted from stable plant weights among dicamba treatments within the winter wheat plots. This effect was attributed to wheat competition because the weight of kochia growing with winter wheat was 61% lower than kochia growing in the non-wheat

treatment (df (1, 13), $t = 4.92$, $P = 0.0003$). This interaction was further examined through the interaction of dicamba rate, crop competition, and kochia accession (df (2, 17), $F = 16.25$, $P = 0.0001$, Table 4.16). This interaction showed that the S2 accession was more sensitive to the influence of crop competition than the Sato accession. There also was an unexplained effect of increasing dicamba rate for the S2 accession under wheat influence. While all other combinations of crop competition and kochia accession tended to decrease in weight with increasing dicamba rate, accession S2 increased in weight as dicamba rate increased (df (1, 17), $t = 2.32$, $P = 0.033$).

In the second season, dicamba rate interacted with kochia accession (df (2, 24), $F = 4.00$, $P = 0.032$, Table 4.17). In this season, it was the Sato accession that produced the atypical response to increased dicamba rate. The interaction of dicamba rate, crop competition, and kochia accession was used to more completely explain this effect (df (2, 24), $F = 3.93$, $P = 0.033$, Table 4.18). The interaction was the result of plant responses in the non-wheat treatment. While S2 accession plant weight tended to decrease as rate increase, Sato accession weight tended to increase. The interactions with dicamba rate in each season not only involved a different accession exhibiting the atypical dose response but also involved different winter wheat treatments.

Surviving Plants. The percentage of surviving kochia plants depended on kochia accession. Eighty-eight percent of Sato accession plants survived to harvest compared to 65% survival of the S2 accession (df (1, 58), $F = 25$, $P = 0.0001$). Surviving plants were not all vigorously growing. Dicamba application

reduced the apparent health of plants, particularly of accession S2 plants. There were varying degrees of plant size and residual herbicide injury. For a plant to be classified as a survivor, it needed only to have green tissue.

Dicamba rate also influenced the number of surviving plants (df (2, 58), $F = 9.03$, $P = 0.0004$). Plant survival in the control herbicide treatment averaged across winter wheat level and kochia accession was 90%. Application of 70 g ae ha⁻¹ dicamba reduced the number of plants in each plot, 70% survival (df (1, 58), $t = 3.52$, $P = 0.0008$). Increasing the rate of dicamba to 140 g ae ha⁻¹ dicamba did not change the number of surviving plants, 69%, compared to 70 g ae ha⁻¹ dicamba (df (1, 58), $t = 0.30$, $P = 0.77$).

While crop competition greatly affected plant height and weight, it did not influence plant survival (df (1, 58), $F = 1.76$, $P = 0.19$). Kochia survival was 80% in plots without winter wheat and 73% in plots with winter wheat. This meant that even though kochia plants in wheat were shorter or weighed less than plants growing independently, the kochia plants were still present in the crop environment competing for water, nutrients, and light. Eliminating kochia from the cropping system relied on dicamba treatment.

Plant Injury. Crop competition affected plant injury (df (1, 40), $F = 19.79$, $P = 0.0001$). Injury expression for plants in non-wheat plots was 43% compared to 66% injury for plants growing in winter wheat. This effect could have been the result of greater spray coverage on kochia plants that were in the open environment of non-wheat plots. Kochia plants growing under the crop canopy were partially shielded from dicamba treatment by the wheat plants.

Kochia plants responded differently depending on accession (df (1, 40), $F = 16.74$, $P = 0.0002$). Accession S2 showed 67% injury while Sato plants only expressed 44% injury. There was no difference in plant injury between 70 g ae ha⁻¹ and 140 g ae ha⁻¹ dicamba treatments, 53% and 58% injury respectively (df (1, 40), $F = 0.91$, $P = 0.35$).

CONCLUSIONS

These experiments examined the effects of several weed management components on kochia. This was done with special consideration of kochia that exhibited different responses to dicamba. Many herbicides remain very effective for kochia management. Of the herbicides included in the experiments, bromoxynil provided the most consistent efficacy across environments, growth stages and kochia accessions investigated. Growth stage of kochia at treatment was an important determiner of injury. Smaller kochia expressed more injury than taller kochia. Systemic herbicides were less influenced by growth stage of kochia at treatment and maintained their performance better than contact herbicides as growth stage increased. Other than instances of known resistance, there were no consistencies in accession response across herbicide treatments.

Soil type did not affect dicamba performance for a limited range of soil types and organic matter contents. All kochia, regardless of past response to dicamba, was very susceptible to pre-emergence dicamba treatments. Differences between the Henry accession and accession S2 in the pre-

emergence treatment became more pronounced in post-emergence treatments. There was no difference in plant injury between plants that encountered dicamba through their roots and plants that encountered dicamba through their leaves and stems.

Kochia injury following treatments of dicamba was not dependent on spray volume. Carrier volumes consistent with the range suggested by dicamba labels did not affect the injury observed within each kochia accession. Kochia injury differences between accessions remained consistent across volumes.

Crop competition greatly reduced kochia height and weight in favorable growing conditions but did not reduce kochia population. Dicamba treatment also affected kochia harvest parameters and was integral in reducing kochia population. The selected crop, winter wheat, was a good competitor to use as a model for eastern Colorado agriculture. The results may have been different if another crop system was used. Crop rotations in many of the locations that have been investigated for atypical kochia response to dicamba included corn. A corn system is under investigation at North Dakota State University to effects on kochia.

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Table 4.1. *K. scoparia* accession effect on plant injury rating averaged across growth stages and herbicide treatments 28 days after treatment, Fort Collins, CO.

KCHSC ^a Accession	Plant Injury	
	Greenhouse Data	Field Data
	(%)	
S1	76 ± 1 ab ^b	82 ± 1 c
S2	78 ± 1 a	88 ± 1 a
T1	72 ± 1 c	85 ± 1 b
T2	72 ± 1 c	78 ± 1 d
Sato	74 ± 1 bc	79 ± 1 d
ANOVA ^c		
df	4, 897	4, 331
F	4.34	21
P>F	0.0018	0.0001

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^c Analysis of variance table for the main effect.

Table 4.2. *K. scoparia* growth stage effect on plant injury rating averaged across herbicide treatments and accessions 28 days after treatment, Fort Collins, CO.

Growth Stage	KCHSC ^a Plant Injury	
	Greenhouse Data	Field Data
	(%)	
5-cm	84 ± 1 a ^b	89 ± 1 a
10-cm	73 ± 1 b	84 ± 1 b
15-cm	67 ± 1 c	74 ± 1 c
ANOVA ^c		
df	2, 897	2, 5
F	86	67
P>F	0.0001	0.0003

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^c Analysis of variance table for the main effect.

Table 4.3. Herbicide treatment effect on *K. scoparia* plant injury rating averaged across growth stages and accessions 28 days after treatment, Fort Collins, CO.

Herbicide ^a	KCHSC ^b Plant Injury	
	Greenhouse Data	Field Data
(g ai ha ⁻¹)	(%)	
Control	0 ± 0 h ^c	0 ± 0 h
Bromoxynil (420)	97 ± 2 a	91 ± 3 ab
Bromoxynil + MCPA (840)	95 ± 2 a	82 ± 3 cd
Pyridate (1053)	83 ± 2 cd	92 ± 3 ab
Carfentrazone-ethyl (17)	44 ± 2 g	75 ± 3 de
Carfentrazone-ethyl (26)	52 ± 2 f	83 ± 3 cd
2,4-D ester (1064)	55 ± 2 f	61 ± 3 g
Dicamba (140)	67 ± 2 e	72 ± 3 ef
Dicamba (560)	85 ± 2 bc	84 ± 3 bc
Fluroxypyr (140)	79 ± 2 d	90 ± 3 bc
Fluroxypyr (420)	89 ± 2 b	92 ± 3 ab
Atrazine (1400)	81 ± 2 cd	99 ± 3 a
Chlorsulfuron (26)	66 ± 2 e	64 ± 3 fg
ANOVA ^d		
df	11, 897	11, 24
F	89	16.34
P>F	0.0001	0.0001

^a Spray solution adjuvants were included as indicated in the Materials and Methods section.

^b KCHSC, *Kochia scoparia*.

^c Means $\pm$ standard errors in each column followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^d Analysis of variance table for the main effect.

Table 4.4. Combined effects of herbicide treatment and *K. scoparia* growth stage on plant injury rating averaged across accessions 28 days after treatment, greenhouse data.

Herbicide ^a	KCHSC ^b Plant Injury		
	Growth Stage		
	5-cm	10-cm	15-cm
(g ai ha ⁻¹)	(%)		
Bromoxynil (420)	100 ± 3 a ^c	98 ± 3 a	93 ± 3 a
Bromoxynil + MCPA (840)	99 ± 3 a	99 ± 3 a	88 ± 3 b
Pyridate (1053)	99 ± 3 a	86 ± 3 b	65 ± 3 c
Carfentrazone-ethyl (17)	72 ± 3 a	33 ± 3 b	26 ± 3 b
Carfentrazone-ethyl (26)	84 ± 3 a	42 ± 3 b	29 ± 3 c
2,4-D ester (1064)	61 ± 3 a	53 ± 3 a	52 ± 3 a
Dicamba (140)	69 ± 3 a	66 ± 3 a	65 ± 3 a
Dicamba (560)	90 ± 3 a	84 ± 3 a	82 ± 3 a
Fluroxypyr (140)	82 ± 3 a	79 ± 3 a	76 ± 3 a
Fluroxypyr (420)	94 ± 3 a	88 ± 3 ab	84 ± 3 b
Atrazine (1400)	84 ± 3 a	84 ± 3 a	75 ± 3 a
Chlorsulfuron (26)	70 ± 3 a	62 ± 3 a	65 ± 3 a
ANOVA ^d			
df	22, 897		
F	9.06		
P>F	0.0001		

^a Spray solution adjuvants were included as indicated in the Materials and Methods.

^b KCHSC, *Kochia scoparia*.

^c Means $\pm$ standard errors in each row followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^d Analysis of variance table for the interaction effect.

Table 4.5. Combined effects of herbicide treatment and *K. scoparia* accession on plant injury rating averaged across growth stages 28 days after treatment, greenhouse data.

Herbicide ^a	KCHSC ^b Plant Injury				
	Accession				
	S1	S2	T1	T2	Sato
(g ai ha ⁻¹)	(%)				
Bromoxynil (420)	96 a ^c	97 a	98 a	96 a	98 a
Bromoxynil + MCPA (840)	92 a	96 a	92 a	99 a	99 a
Pyridate (1053)	75 c	77 bc	94 a	88 ab	80 bc
Carfentrazone-ethyl (17)	41 a	44 a	41 a	43 a	50 a
Carfentrazone-ethyl (26)	47 a	51 a	54 a	49 a	57 a
2,4-D ester (1064)	64 ab	67 a	47 c	53 bc	47 c
Dicamba (140)	74 a	72 a	64 ab	69 a	56 b
Dicamba (560)	90 a	90 a	84 ab	85 ab	77 b
Fluroxypyr (140)	79 a	80 a	79 a	80 a	76 a
Fluroxypyr (420)	89 a	89 a	91 a	90 a	86 a
Atrazine (1400)	97 a	98 a	51 b	60 b	98 a
Chlorsulfuron (26)	67 ab	77 a	74 a	51 c	61 bc
SEM ^d	4	4	4	4	4
ANOVA ^e					
df	44, 897				
F	4.51				
P>F	0.0001				

^a Spray solution adjuvants were included as indicated in the Materials and Methods.

^b KCHSC, *Kochia scoparia*.

^c Means $\pm$ standard errors in each row followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^d Standard error of the means for values within a column.

^e Analysis of variance table for the interaction effect.

Table 4.6. Combined effects of dicamba rate and *K. scoparia* accession on plant injury rating averaged across soils and plant organs exposed 28 days after treatment.

Dicamba Rate	KCHSC ^a Plant Injury	
	Accession	
	S2	Henry
(g ae ha ⁻¹)	(%)	
140	A ^b 93 ± 1 a ^c	B 74 ± 1 b
280	A 95 ± 1 a	A 84 ± 1 b
ANOVA ^d		
df	1, 175	
F	10.69	
P>F	0.0013	

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^c Means ± standard errors in each row followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^d Analysis of variance table for the interaction effect.

Table 4.7. Plant organ of herbicide exposure effect on *K. scoparia* plant injury rating averaged across soils, accessions, and herbicide rates 28 days after treatment.

Plant Organ Exposed	KCHSC ^a Plant Injury (%)
Germinating Seeds	96 ± 1 a ^b
Roots Only	81 ± 1 c
Shoots Only	79 ± 1 c
Roots and Shoots	90 ± 1 b
ANOVA ^c	
df	3, 175
F	48
P>F	0.0001

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^c Analysis of variance table for the main effect.

Table 4.8. Combined effects of plant organ exposed and *K. scoparia* accession on plant injury rating averaged across soils and dicamba rates 28 days after treatment.

Plant Organ Exposed	KCHSC ^a Plant Injury	
	Accession	
	S2	Henry
	(%)	
Germinating Seeds	A ^b 99 ± 2 a ^c	A 93 ± 2 b
Roots Only	BC 93 ± 2 a	C 70 ± 2 b
Shoots Only	C 90 ± 2 a	C 67 ± 2 b
Roots and Shoots	AB 95 ± 2 a	B 84 ± 2 b
ANOVA ^d		
df	3, 175	
F	15.29	
P>F	0.0001	

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^c Means ± standard errors in each row followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^d Analysis of variance table for the interaction effect.

Table 4.9. Dicamba rate effect on *K. scoparia* plant height averaged across accessions and winter wheat treatment at winter wheat grain maturity, Fort Collins, CO.

Dicamba Rate	KCHSC ^a Plant Height	
	Year	
	1996	1997
(g ae ha ⁻¹)	(cm)	
0	34 ± 2 a ^b	82 ± 3 a
70	30 ± 2 ab	59 ± 3 b
140	23 ± 2 b	65 ± 3 b
ANOVA ^c		
df	2, 24	2, 20
F	4.65	16.00
P>F	0.020	0.0001

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^c Analysis of variance table for the main effect.

Table 4.10. Combined effects of dicamba rate and winter wheat competition on *K. scoparia* plant height averaged across accessions at winter wheat grain maturity, Fort Collins, CO, 1997.

Dicamba Rate	KCHSC ^a Plant Height	
	Winter Wheat Presence	
	No Wheat	Wheat
(g ae ha ⁻¹)	(cm)	
0	A ^b 86 ± 4 a ^c	A 78 ± 4 a
70	B 73 ± 4 a	B 45 ± 4 b
140	AB 78 ± 4 a	B 52 ± 4 b
ANOVA ^d		
df	2, 20	
F	3.65	
P>F	0.045	

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column preceded by the same upper case letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^c Means ± standard errors in each row followed by the same lower case letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^d Analysis of variance for the interaction effect.

Table 4.11. Combined effects of winter wheat competition and *K. scoparia* accession on plant height averaged across dicamba rates at winter wheat grain maturity, Fort Collins, CO, 1996.

Winter Wheat Presence	KCHSC ^a Plant Height	
	Accession	
	S2	Sato
	(cm)	
No Wheat	B ^b 25 ± 3 a ^c	A 29 ± 3 a
Wheat	A 38 ± 3 a	B 25 ± 3 b
ANOVA ^d		
df	1, 24	
F	10.07	
P>F	0.0041	

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column preceded by the same upper case letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^c Means ± standard errors in each row followed by the same lower case letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^d Analysis of variance for the interaction effect.

Table 4.12. Combined effects of dicamba rate, winter wheat competition, and *K. scoparia* accession on plant height at winter wheat grain maturity, Fort Collins, CO, 1996.

Dicamba Rate	KCHSC ^a Plant Height	
	Accession	
	S2	Sato
(g ae ha ⁻¹)	(cm)	
No Wheat		
0	A ^b 39 ± 5 a ^c	A 31 ± 5 a
70	A 27 ± 5 a	A 27 ± 5 a
140	B 8 ± 5 b	A 29 ± 5 a
Wheat		
0	A 36 ± 5 a	A 29 ± 5 a
70	A 41 ± 5 a	A 26 ± 5 b
140	A 38 ± 5 a	A 19 ± 5 b
ANOVA ^d		
df	2, 24	
F	4.65	
P>F	0.020	

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column within winter wheat treatment preceded by the same upper case letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^c Means ± standard errors in each row followed by the same lower case letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^d Analysis of variance for the interaction effect.

Table 4.13. Winter wheat competition effect on *K. scoparia* plant dry weight averaged across accessions and dicamba rates at winter wheat grain maturity, Fort Collins, CO.

Winter Wheat Presence	KCHSC ^a Dry Weight	
	Year	
	1996	1997
	(g plant ⁻¹)	
No Wheat	1.48 ± 0.19 a ^b	67.18 ± 5.41 a
Wheat	1.11 ± 0.19 a	5.87 ± 5.41 b
ANOVA ^b		
df	1, 3	1, 24
F	3.25	63
P>F	0.18	0.0001

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^c Analysis of variance table for the main effect.

Table 4.14. Dicamba rate effect on *K. scoparia* plant dry weight averaged across accessions and winter wheat treatments at winter wheat grain maturity, Fort Collins, CO.

Dicamba Rate	KCHSC ^a Dry Weight	
	Year	
	1996	1997
(g ae ha ⁻¹)	(g plant ⁻¹)	
0	1.82 ± 0.21 a ^b	37.93 ± 6.62 a
70	1.12 ± 0.21 b	27.05 ± 6.62 a
140	0.94 ± 0.21 b	44.59 ± 6.62 a
ANOVA ^c		
df	2, 17	2, 24
F	9.06	1.79
P>F	0.0020	0.19

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^c Analysis of variance for the interaction effect.

Table 4.15. Combined effects of dicamba rate and winter wheat competition on *K. scoparia* plant dry weight averaged across accessions at winter wheat grain maturity, Fort Collins, CO, 1996.

Dicamba Rate	KCHSC ^a Dry Weight	
	Winter Wheat Presence	
	No Wheat	Wheat
(g ae ha ⁻¹)	(g plant ⁻¹)	
0	A ^b 2.61 ± 0.26 a ^c	A 1.02 ± 0.26 b
70	B 1.04 ± 0.26 a	A 1.20 ± 0.26 a
140	B 0.78 ± 0.26 a	A 1.11 ± 0.26 a
ANOVA ^d		
df	2, 17	
F	11.94	
P>F	0.0005	

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column preceded by the same upper case letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^c Means ± standard errors in each row followed by the same lower case letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^d Analysis of variance for the interaction effect.

Table 4.16. Combined effects of dicamba rate, winter wheat competition, and *K. scoparia* accession on plant dry weight at winter wheat grain maturity, Fort Collins, CO, 1996.

Dicamba Rate	KCHSC ^a Dry Weight	
	Accession	
	S2	Sato
(g ae ha ⁻¹)	(g plant ⁻¹)	
No Wheat		
0	A ^b 3.96 ± 0.36 a ^c	A 1.25 ± 0.36 a
70	B 1.16 ± 0.36 a	A 0.93 ± 0.36 b
140	B 0.52 ± 0.36 a	A 1.04 ± 0.36 b
Wheat		
0	B 0.73 ± 0.36 a	A 1.32 ± 0.36 a
70	A 1.74 ± 0.36 a	A 0.67 ± 0.36 a
140	AB 1.40 ± 0.36 a	A 0.81 ± 0.36 a
ANOVA ^d		
df	2, 17	
F	16.25	
P>F	0.0001	

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column within winter wheat treatment preceded by the same upper case letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^c Means ± standard errors in each row followed by the same lower case letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^d Analysis of variance for the interaction effect.

Table 4.17. Combined effects of dicamba rate and *K. scoparia* accession on plant dry weight averaged across winter wheat treatments at winter wheat grain maturity, Fort Collins, CO, 1997.

Dicamba Rate	KCHSC ^a Dry Weight	
	Accession	
	S2	Sato
(g ae ha ⁻¹)	(g plant ⁻¹)	
0	A ^b 47.68 ± 9.36 a ^c	B 28.18 ± 9.36 a
70	B 16.67 ± 9.36 a	AB 37.43 ± 9.36 a
140	AB 29.34 ± 9.36 b	A 59.84 ± 9.36 a
ANOVA ^d		
df	2, 24	
F	4.00	
P>F	0.032	

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column preceded by the same upper case letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^c Means ± standard errors in each row followed by the same lower case letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^d Analysis of variance for the interaction effect.

Table 4.18. Combined effects of dicamba rate, winter wheat competition, and *K. scoparia* accession on plant dry weight at winter wheat grain maturity, Fort Collins, CO, 1997.

Dicamba Rate	KCHSC ^a Dry Weight	
	Accession	
	S2	Sato
(g ae ha ⁻¹)	(g plant ⁻¹)	
No Wheat		
0	A ^b 87.37 ± 13.24 a ^c	B 45.53 ± 13.24 b
70	B 29.55 ± 13.24 b	B 69.32 ± 13.24 a
140	AB 54.72 ± 13.24 b	A 114.60 ± 13.24 a
Wheat		
0	A 7.98 ± 13.24 a	A 8.84 ± 13.24 a
70	A 3.80 ± 13.24 a	A 5.54 ± 13.24 a
140	A 3.96 ± 13.24 a	A 5.07 ± 13.24 a
ANOVA ^d		
df	2, 24	
F	3.93	
P>F	0.033	

^a KCHSC, *Kochia scoparia*.

^b Means ± standard errors in each column within winter wheat treatment preceded by the same upper case letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^c Means ± standard errors in each row followed by the same lower case letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^d Analysis of variance for the interaction effect.

APPENDICES

Appendix 2.A. Dicamba rate effect on 200 collections of *K. scoparia* from Colorado.

Site	KCHSC Injury			Site	KCHSC Injury		
	Dicamba Rate (g ae ha ⁻¹)				Dicamba Rate (g ae ha ⁻¹)		
	140	280	560		140	280	560
	————— (%) —————				————— (%) —————		
145	50	50	95	157	80	90	100
20	30	85	90	161	8	90	100
82	50	70	95	85	80	95	100
71	40	80	100	115	75	99	100
100	50	80	90	125	75	99	100
143	60	70	90	79	80	95	100
144	60	70	90	114	75	100	100
72	30	100	100	121	80	95	100
83	70	80	80	154	90	90	95
70	40	95	99	174	90	90	95
74	50	85	99	64	89	90	97
73	40	99	100	43	80	96	100
77	50	90	100	84	80	100	99
75	50	90	100	123	80	99	100
76	50	90	100	127	80	99	100
97	70	80	100	131	90	90	99
172	60	90	100	105	80	100	100
155	60	95	100	106	80	100	100
92	80	100	80	108	80	100	100
137	60	100	100	129	80	100	100
158	70	90	100	132	90	90	100
170	70	90	100	134	80	100	100
113	75	90	100	138	90	90	100
148	70	95	100	141	95	90	95
151	75	95	95	146	80	100	100
159	70	95	100	160	90	90	100
165	70	95	100	171	90	90	100
166	70	95	100	175	90	90	100
94	70	99	100	179	85	95	100
110	70	99	100	19	90	95	99
18	80	90	100	112	90	95	99
78	80	90	100	122	90	99	95
86	70	100	100	66	90	95	100
98	80	90	100	119	90	95	100
99	80	90	100	130	90	95	100
104	70	100	100	149	95	90	100
109	75	95	100	152	90	95	100
124	70	100	100	156	90	95	100

Site	KCHSC Injury			Site	KCHSC Injury		
	Dicamba Rate (g ae ha ⁻¹)				Dicamba Rate (g ae ha ⁻¹)		
	140	280	560		140	280	560
	(%)				(%)		
164	85	100	100	21	95	99	100
177	85	100	100	22	95	100	100
181	85	100	100	33	95	100	100
17	85	98	100	41	95	100	100
3	90	98	100	44	95	100	100
128	90	99	99	45	95	100	100
6	90	99	100	62	95	100	100
11	90	99	100	67	95	100	100
42	90	99	100	81	95	100	100
120	90	99	100	142	95	100	100
126	90	99	100	162	95	100	100
10	91	99	100	163	95	100	100
26	90	100	100	168	95	100	100
29	90	100	100	169	95	100	100
87	90	100	100	176	95	100	100
93	90	100	100	178	95	100	100
95	90	100	100	180	100	95	100
96	90	100	100	182	95	100	100
103	90	100	100	183	95	100	100
107	90	100	100	187	95	100	100
111	90	100	100	192	95	100	100
118	90	100	100	193	100	95	100
133	90	100	100	198	95	100	100
135	90	100	100	4	96	100	100
136	90	100	100	38	97	99	100
139	100	90	100	25	98	99	100
147	90	100	100	37	98	100	99
153	95	95	100	50	98	99	100
167	90	100	100	46	100	100	99
185	90	100	100	9	98	100	100
186	95	95	100	12	98	100	100
188	95	95	100	15	99	99	100
189	95	95	100	30	99	99	100
195	90	100	100	49	98	100	100
199	90	100	100	61	99	99	100
200	90	100	100	68	99	99	100
8	92	100	100	69	99	99	100
23	97	95	100	116	99	99	100
5	95	98	100	7	99	100	100
28	95	98	100	13	99	100	100

Site	KCHSC Injury			Site	KCHSC Injury		
	Dicamba Rate (g ae ha ⁻¹)				Dicamba Rate (g ae ha ⁻¹)		
	140	280	560		140	280	560
	(%)				(%)		
16	99	100	100	191	100	100	100
24	99	100	100	194	100	100	100
27	99	100	100	196	100	100	100
35	99	100	100	197	100	100	100
36	99	100	100				
39	99	100	100				
40	99	100	100				
48	99	100	100				
53	99	100	100				
59	99	100	100				
60	100	99	100				
63	99	100	100				
65	99	100	100				
88	99	100	100				
90	99	100	100				
117	99	100	100				
1	100	100	100				
2	100	100	100				
14	100	100	100				
31	100	100	100				
32	100	100	100				
34	100	100	100				
47	100	100	100				
51	100	100	100				
52	100	100	100				
54	100	100	100				
55	100	100	100				
56	100	100	100				
57	100	100	100				
58	100	100	100				
80	100	100	100				
89	100	100	100				
91	100	100	100				
101	100	100	100				
102	100	100	100				
140	100	100	100				
150	100	100	100				
173	100	100	100				
184	100	100	100				
190	100	100	100				

Appendix 2.B. Herbicide effect on K. scoparia from 1994 lack of control locations 28 days after treatment.

Location	KCHSC Injury				
	Herbicide (g ai ha ⁻¹)				Atrazine (1400)
	(140)	Dicamba (280)	(560)	Chlorsulfuron (26+26)	
Longmont, CO					
1	70	80	85	25	10
2	80	90	95	10	100
3	75	100	100	30	100
4	70	80	99	25	0
Stratton, CO					
5	95	100	100	10	100
6	95	100	100	10	100
7	95	100	100	50	100
8	90	100	100	0	100
9	95	95	100	0	100
10	90	100	100	40	100
11	80	100	100	70	100
12	95	100	100	20	100
Burlington, CO					
13	85	95	100	0	0
14	95	75	80	0	0
15	70	95	100	0	100
16	80	100	100	80	100
17	90	100	100	60	60
18	85	100	100	70	60
19	90	100	100	20	100
20	95	95	95	30	100
21	85	85	95	10	10
Scottsbluff, NE					
22	80	80	90	95	100
23	75	80	85	85	80
24	50	50	70	100	100
25	40	60	80	100	100
26	30	50	75	50	100
27	25	55	70	10	100
28	50	65	95	100	100
29	65	75	95	100	100

Location	KCHSC Injury				
	Herbicide (g ai ha ⁻¹)				
	(140)	Dicamba (280)	(560)	Chlorsulfuron (26+26)	Atrazine (1400)
Eustis, NE					
30	80	95	100	100	0
31	85	100	100	100	50
Culbertson, NE					
32	80	95	100	100	100
33	20	40	65	100	0
34	90	95	95	60	100
Wakeeney, KS					
35	70	85	90	50	0
36	80	85	95	50	0
37	80	95	100	80	10
Rush Center, KS					
38	50	80	90	80	50
39	85	95	100	30	100
40	80	95	100	60	100
41	45	70	100	70	95
Sheridan Lake, CO					
42	95	100	100	60	100
43	95	100	100	50	50
44	95	95	100	50	100
45	95	100	100	20	100

Appendix 2.C. Herbicide effect on *K. scoparia* from 1995 lack of control locations
28 days after treatment.

Location	KCHSC Injury				
	Herbicide (g ai ha ⁻¹)				
		Dicamba		Chlorsulfuron	Atrazine
	(140)	(280)	(560)	(26)	(1400)
Morrill, NE					
1	95	95	100	100	100
2	100	99	100	100	100
3	90	80	90	100	100
4	95	95	100	40	100
5	75	80	95	100	100
7	85	90	95	90	100
8	95	100	100	100	100
9	50	75	95	100	100
10	80	90	95	100	100
11	80	90	90	100	100
12	90	90	100	40	100
Culbertson, NE					
14	85	85	90	90	50
15	85	95	99	95	95
16	70	80	90	90	30
17	90	100	100	20	10
18	95	95	100	100	10
19	80	85	100	90	70
20	95	100	100	100	50
21	100	100	100	100	90
Oakley, KS					
22	90	100	100	60	80
23	80	70	80	85	100
Quinter, KS					
24	95	100	100	0	100
26	95	95	100	60	100
Leoti, KS					
27	100	100	100	50	75
28	95	100	100	95	100
29	99	100	100	30	100
Garden City, KS					
30	90	95	98	50	85
31	95	100	100	90	100
32	100	100	99	10	80
33	95	100	100	0	0

Location	KCHSC Injury			
	Herbicide (g ai ha ⁻¹)			Chlorsulfuron (26)
	(140)	Dicamba (280)	(560)	
Montana-076				
3	50	57	85	80
37	80	75	80	0
7	70	85	85	0
36	80	87	85	0
9	80	82	92	0
6	78	87	90	6
4	77	88	95	40
38	88	87	90	0
10	80	90	95	68
8	82	93	93	0
35	85	93	100	0
30	90	93	95	3
22	92	92	97	0
13	95	92	95	0
2	93	93	97	30
29	90	95	99	0
24	90	95	100	0
31	93	95	97	0
15	93	95	97	0
16	93	95	98	0
34	92	95	100	0
26	90	97	100	0
17	93	97	97	0
28	90	98	99	0
1	95	95	99	53
23	95	95	100	7
20	95	95	100	0
21	95	95	100	0
25	95	97	100	0
12	95	99	100	43
19	95	100	100	0
33	95	100	100	0
11	95	100	100	60
27	97	100	100	0
5	97	100	100	57
32	98	100	100	13
14	98	100	100	0
18	100	100	100	0

Location	KCHSC Injury			
	Herbicide (g ai ha ⁻¹)			Chlorsulfuron (26)
	(140)	Dicamba (280)	(560)	
Montana-077				
5	53	73	92	0
6	63	73	90	10
7	72	70	88	0
4	60	88	93	60
11	82	85	87	82
8	75	88	100	75
1	88	85	95	50
3	85	88	98	0
9	85	92	98	40
12	87	93	95	70
2	90	90	100	0
10	95	100	100	0
Montana-074				
2	83	95	98	0
Montana-078				
2	87	90	92	100
1	87	88	95	88

Appendix 2.D. Tables of dicamba rate and field position effects on *K. scoparia* resistance expression.

Table 2.D.1. Dicamba rate effect on *K. scoparia* resistance expression for each location averaged across years and positions.

Dicamba	KCHSC ^a Resistance ^b				
Rate	Location				
	Burlington	Culbertson	Henry	Longmont	Morrill
(g ae ha ⁻¹)	(%)				
70	3 ± 1 a ^c	14 ± 1 a	21 ± 1 a	10 ± 1 a	9 ± 1 a
140	1 ± 1 b	4 ± 1 b	8 ± 1 b	3 ± 1 b	5 ± 1 b
ANOVA ^d					
df	1, 222	1, 66	1, 34	1, 48	1, 23
F	17.15	68	110	81	47
P>F	0.0001	0.0001	0.0001	0.0001	0.0001

^a KCHSC, *Kochia scoparia*.

^b Plants that expressed less than 40% injury at 28 days after treatment were classified as resistant.

^c Means ± standard errors in each column followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^d Analysis of variance table for the main effect.

Table 2.D.2. Position effect on *K. scoparia* resistance expression for each location averaged across years and dicamba rates.

Position ^a	KCHSC ^b Resistance ^c				
	Location				
	Burlington	Culbertson	Henry	Longmont	Morrill
	(%)				
Out	2 ± 1 a ^d	2 ± 1 b	6 ± 2 b	4 ± 1 b	3 ± 1 b
In	1 ± 1 a	15 ± 1 a	23 ± 2 a	9 ± 1 a	11 ± 1 a
ANOVA ^e					
df	1, 234	1, 148	1, 143	1, 202	1, 157
F	0.53	41	54	13.76	19.68
P>F	0.47	0.0001	0.0001	0.0003	0.0001

^a Position was in reference to the cropping area. Out, adjacent field or non-crop area; In, primary crop area.

^b KCHSC, *Kochia scoparia*.

^c Plants that expressed less than 40% injury at 28 days after treatment were classified as resistant.

^d Means ± standard errors in each column followed by the same letter were not different according to least squares means contrasts for $\alpha = 0.05$.

^e Analysis of variance table for the main effect.

Appendix 4.A. Colorado Meteorological Network monthly average maximum temperature and total precipitation for experiment seasons at the ARDEC weather station (elevation = 1560 m, latitude = 40.65° N, and longitude = 105.00° W) and for the 100 year average at the Fort Collins weather station (elevation = 1560 m, latitude = 40.59° N, and longitude = 105.14° W).

Month	1995-96		1996-97		100 Year Average	
	Temp	Precip	Temp	Precip	Temp	Precip
	(°C)	(mm)	(°C)	(mm)	(°C)	(mm)
Aug	31	25	28	14	29	35
Sep	21	60	21	24	24	33
Oct	18	67	17	7	18	28
Nov	12	0	9	7	11	15
Dec	7	0	7	0	6	12
Jan	4	0	2	5	5	9
Feb	8	0	4	3	7	13
Mar	10	0	13	5	10	29
Apr	16	4	12	37	16	49
May	21	115	21	30	21	71
Jun	27	72	27	37	26	46
Jul	28	55	29	128	29	40
Total		398		297		380