Wild oat resistance to triallate in Montana
by Wade Eugene Malchow

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in Agronomy
Montana State University

Abstract:
Wild oats is the most important weed in the continuous malt barley production area of Fairfield, Montana. Continuous use of the wild oat herbicide triallate in barley production has resulted in selection for triallate resistant wild oats.

Sixty-four percent of fields sampled in 1992 were determined to contain triallate resistant seed. Fields that were treated with triallate in 1992 and treated with imazamethabenz in 1993 had a significant increase in triallate resistant wild oat seed which may increase the probability of selecting for wild oats that are resistant to triallate and imazamethabenz. A survey of field histories indicated that resistant fields had been treated with triallate for more years than susceptible fields, and had been planted to barley 99% of the time between 1988 and 1992, while susceptible fields were in alternative crop 19% of the time.

Resistant fields within the study region were in patches approximately 1.47 km in diameter. Patches of resistant fields indicates that resistance was introduced or selected for in particular fields and may have dispersed to adjacent fields by seed or pollen.

Sampling individual plants within triallate treated fields indicated that approximately two-thirds of the plants were resistant and were in patches approximately 7.5 meters in diameter. Only one-third of the seed sampled from within a field that was not treated with triallate were resistant and were found to be randomly distributed. Triallate resistant wild oats may not be able to form patches without the presence of the selecting herbicide due to a lack of fitness compared to susceptibles.

Triallate resistant wild oat seed was at a very low frequency on roadsides indicating that wild oats are not being moved, and/or are not able to persist without the presence of triallate due to a decreased fitness compared to susceptible wild oat plants.

Triallate resistant wild oats were determined to be resistant to difenzoquat, which is not consistently used in the Fairfield area. One wild oat field sample screened with triallate, diclofop-methyl, imazamethabenz, and all subsequent combinations was controlled by imazamethabenz but was resistant to triallate, diclofop-methyl, and triallate plus diclofop-methyl.

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This thesis has been read by each member of the thesis committee and has been found to be satisfactory regarding content, English usage, format, citations, bibliographic style, and consistency, and is ready for submission to the College of Graduate Studies.

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TABLE OF CONTENTS

TABLE OF CONTENTS - CONTINUED

LIST OF TABLES

Table

LIST OF FIGURES

LIST OF FIGURES - CONTINUED

LIST OF FIGURES - CONTINUED

ABSTRACT

Wild oats is the most important weed in the continuous malt barley production area of Fairfield, Montana. Continuous use of the wild oat herbicide triallate in barley production has resulted in selection for triallate resistant wild oats.

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CHAPTER 1

LITERATURE REVIEW

Wild Oat

Wild oats (*Avena fatua* L.) is an annual grass native to Asia , but has become common throughout much of western North America since its arrival with early settlers as impurities in grain seed. Wild oats is a serious problem in spring grain crops, but can also be found along roadsides, in pastures and waste areas (Whitson et al., 1991).

Wild oat belongs to the family Poaceae. Wild oat resembles cultivated oat (*Avena sativa* L.) but can be distinguished by greater height, loose drooping panicles, a geniculate awn, and basal seed scars the shape of a sucker mouth. It is believed that *A. sativa* and *A. fatua* may have shared a common ancestor in *A. sterilis* (Griffiths and Johnston, 1956).

Wild oats are a major weed problem in arable lands, especially in cereals, in the United Kingdom, Belgium, Denmark, France, Germany, Netherlands, Spain, Greece, Italy, North Africa, the former Soviet Republics, Australia and New Zealand (Bowler, 1973; Dadd, 1957; Thurston, 1954). In addition, wild oats are by far the most troublesome weed of the cultivated portion of the Northern Great Plains region of North America (Wood, 1953). The total area infested with wild oats in Canada and the United States is estimated at over 25 million ha (Nalewaja, 1970).

Wild oats can reduce the yield of wheat (*Triticum aestivum* L.) (Bell and Nalewaja, 1968; Bowden and Friesen, 1967), barley (*Hordeum vulgare* L.) (Bell and Nalewaja, 1968; McBeath et al., 1970), flax (*Linum usitatissimum* L.) (Bowden and Friesen, 1967), and rapeseed (*Brassica napus* L.) (Dew and Keys, 1976). Projected annual losses of wheat and barley were estimated at 2.2 and 6.4 million tons in Europe and North America, respectively (Nalewaja, 1977). Wild oat is Montana's most costly weed problem (Fay and Stewart, 1981). The wild oat problem in Montana is intensifying because combines have the ability to move wild oat seed, while large tractors, tillage equipment and drills enable growers to

seed earlier and to recrop more, creating more favorable conditions for wild oat growth (Fay and Stewart, 1981).

Wild Oat Interference

No known studies have shown beneficial effects of wild oats on crops, however many studies have demonstrated that wild oats reduce small grain yields. Reports on yield reductions resulting from wild oats vary. This may be due to variable edaphic conditions among years and sites. Bowden and Friesen (1967) showed that 33 and 84 wild oat plants m^{-2} decreased wheat yields by 16 and 27%, respectively, when previous year summerfallow plots were fertilized or by 22 and 37%, respectively, when the plots were unfertilized. In England, average wild oat seedling densities of 174 and 157 m^{-2} reduced spring barley yields by 16 and 20% respectively (Wilson and Peters, 1982). Wild oat infestations of 150-200 plants m^{-2} were estimated to reduce barley yield 26% and between 33 and 39% in wheat (Bell and Nalewaja, 1968; Chancellor and Peters, 1974). It has been found that the competitive effects of wild oats increase the longer they are allowed to remain in the crop, however, reports on the time of onset of wild oat competition vary. Studies in England (Chancellor and Peters, 1974) and in the United States (Sharma and Hunter, 1974), demonstrate that wild oat competition began at the four leaf stage of barley and wheat. Reports from Canada are quite variable. In Manitoba, wild oat competition in wheat began prior to crop emergence at wild oat densities of 110 plants m^{-2} or higher (Bowden and Friesen, 1967), while in Saskatchewan, wild oat competition in wheat began between the two to four-leaf stage of the wild oats (Sharma and Hunter, 1974).

More recently the competitive effects of spring small grains and wild oats have been re-assessed in order to develop predictive models for determining economic weed control thresholds. In developing these models many competitive aspects of barley, wheat, and wild oats have been studied to determine model parameters. Carlson and Hill (1985) measured the grain yield of wheat (*Triticum aestivum* L. 'Anza') at various wild oat and wheat densities. Wheat yields were reduced by wild oat competition in all experiments. Wheat yields were reduced by 64% at the highest wild oat density of 300 plants m^{-2}.

However, increasing crop density had an effect in reducing yield losses when weed densities were low (below 200 wild oat plants m^{-2}).

Experiments conducted in Idaho investigated the growth and development of wild oats and barley in mixture and monoculture. Barley was more competitive with wild oat than wild oat was with barley when barley and wild oats were planted in mixed culture additively. Barley in mixture with wild oats produced a barley yield that was 34% lower than barley yielded in monoculture (Morishita and Thill, 1988a). Wild oats and barley had growth and development patterns that were similar to each other when grown in monoculture, partially explaining the intensity of wild oat competition in spring grains. A later study demonstrated that wild oat interference reduced tiller head formation, accounting for the reduction in barley yield (Morishita and Thill, 1988b). Morishita and Thill, (1988b) explained that the reduction in tiller head formation was due to wild oat's ability to reduce total water and turgor potential in barley at the boot stage of growth.

Dunan and Zimdahl (1991) used density replacement series experiments to study the effects of intra- and interspecific competition and the effects of density and proportion of wild oats on barley. Barley was found to be a stronger competitor than wild oats. Barley's intraspecific competition was greater than its interspecific competition with wild oats. Wild oats, on the other hand were the opposite, having greater competitive effects on barley than on other wild oats. Barley was an earlier emerger than wild oats, establishing a photosynthetic area that was twice that of wild oat four days after emergence. During most of the growing season barley had higher shoot weight, root weight, and leaf area. However, other wild oat growth parameters such as leaf area and leaf area rate of expansion were not different from barley by the end of the growing season. Similar experimentation in Idaho determined that barley biomass was affected most by intraspecific competition, while wild oat biomass was affected most by interspecific competition (Evans et al., 1991).

O'Donovan et al. (1985) investigated the relationship between barley and wheat yield loss to the relative emergence time of wild oat using multiple regression analysis on data from field experiments in

Alberta. A wild oat density of 50 plants m^{-2} emerging at the same time as the barley reduced barley yield by approximately 10%, while 300 plants m^{-2} reduced yield approximately 45%. Results indicated that for every day wild oats emerged before the crop, yield loss increased by approximately 3%. The yield loss diminished by 3% for every day wild oat emerged after the crop. It was also determined that yield loss was generally greater for wheat than it was for barley. Cousens et al. (1987) attempted to refine the analysis of the Canadian data by using a non-linear model, which avoids problems inherent in multiple regression, such as predicting yield loss when no weeds are present, resulting in a better description of the data. Barley was concluded to be a better competitor than wheat and is less affected by late-emerging wild oats. Dunan et al. (1994) used modeling techniques to analyze the competitive abilities of wild oats and irrigated barley, and to determine the effects of various types of simulated wild oat control measures. Dunan et al. (1994) confirmed results found by O'Donovan et al., (1985) and Cousens et al., (1987) involving crop yield losses and wild oat time of emergence. Through the use of model simulations it was also determined that the use of more competitive crop varieties showed promise for reducing the effect of wild oats in irrigated barley.

Allowing for the greater competitive ability of barley over wild oat as previously discussed, wild oats has still been able to persist as a problem in barley production. Wild oats, even with a lower competitive ability early in the growing season, has shown no difference as compared to barley in growth characteristics such as leaf area and leaf area expansion rates by the end of the growing season (Dunan and Zimdahl, 1991).

Seed Characteristics

The ability of wild oat seed to move along the surface of the soil, a secondary dispersal behavior, has been extensively studied. The geniculate awn present on the dorsal side of wild oat florets can propel seeds up to 27 cm from a starting position in 14 days through the twisting and untwisting of the awn caused by changes in humidity (Somody et al., 1985). The movement of wild oat florets allows for

deposition of seed in soil surface cracks and subsequent drilling down into the soil (Somody et al., 1985). Wild oats left to overwinter on the soil surface had lower germination rates compared to buried seed (Somody et al.; 1984).

Miller et al. (1990) studied the effect of burial depth on seed longevity, in which burial depths ranged from 4 to 34 cm. The number of viable seed decreased rapidly during the first 7 months from 99% to 21% and 15% at two different study sites, respectively. The number of viable seeds decreased gradually to less than 1% after 168 months of burial. Research in Great Britain has indicated that only a small proportion of the population of wild oat seed produced on a given year may persist into the second season following seed rain. Wild oat seed may remain viable for up to six years in cultivated soils (Thurston, 1961), resulting in the threat of re-infestation. Miller (1990) concluded the percentage of dormant viable seed was influenced more by the length of burial than by depth, however more viable seed was recovered from deep than shallow depths. A European experiment, investigating the effects of depth of burial on wild oat seed viability over time, noted an initial decline in viable wild oat seed (Wilson, 1978). Fall season tine and deep plowing cultivations were compared with tine cultivation resulting in slightly higher germination rates than did deep plowing. Deep plowing usually deposits seed at deeper depths which would allow wild oat to remain dormant until a later cultivation. In order to reduce wild oat seed bank populations faster, deep tillage should be avoided unless seed will be left at lower depths and never returned to near the surface.

Peaks of wild oat emergence occur in the spring and early fall (Malzew, 1930). Rate of germination is related to soil temperature, but in a complex manner. The optimal temperature ranges reported for germination vary widely, from 10 C to 27 C (Friesen and Shebeski, 1961; Mather, 1946; Paterson et al., 1976; Sharma et al., 1976). The initiation of germination is more likely driven by a combination of factors, such as soil moisture, soil temperature, oxygen levels, and even possibly light exposure and seed maturity. Variable time of germination allows wild oats to emerge for a protracted

period of time. Possible prolonged germination coupled with dormancy capable of lasting several years allows wild oat to persist and make control efforts difficult.

Genetic Variability

The genetic variability of wild oat may be found in differences in germination, vernalization, growth, reproduction, dormancy, and herbicide response (Adkins et al., 1986; Darmency and Aujas, 1986; Price et al., 1985; Miller et al., 1977; Price et al., 1983; Miller et al., 1982). Imam and Allard (1965), conducted research designed to permit quantification of three aspects of genetic variability within wild oat. The objectives of this work were to study the genetic variability between geographically different regions, variation between sites within regions, and variation within individual sites. Concurrently this work provided information as to the mating system of this species. Lemma color and rachilla pubescence, dominant traits in wild oat, were used to determine the frequency of outcrossing. The estimates of outcrossing ranged from slightly higher than 1% to nearly 12% for *Avena fatua.*

In order to determine the geographical variation, Imam and Allard (1965) measured quantitative characters such as percent emergence, days to flowering, number of tillers per plant, height, and seed number per largest panicle in three regions, two regions containing three sites and one region containing one site. A comparison of regions showed striking differences in plant tillering and plant height resulting in a conclusion that wild oats are differentiated geographically and that the differentiations observed appeared to be those which would provide each population with adaptive properties needed to meet the requirements of the environment. A comparison of the three sites within two regions indicated appreciable genetic differentiation between sites within regions. Particularly large differences were found in percent emergence, reflecting varying degrees of seed dormancy. Genetic variability was also measured among families whose seed parents were single plants taken from the same site. Some families at each site showed no seed dormancy (as measured by percent emergence), while each site included at least a few families that had pronounced dormancy levels. Families from each site also varied widely with respect to

days to flowering, mean plant height, and mean tillering ability, indicating that families from each site were highly heterogeneous for the measured characters. Family to family variability was also measured using the family component of variance computed from the between-families term from the analysis of variance for each site. The family component of variance was statistically significant for all characters measured except number of seed per largest panicle, also leading to the conclusion that wild oats at any single site include plants of many different genotypes. Significant variation was also detected within progeny of 10 different individuals. Divergence within individual families was produced by selection of progeny of the 10 individual parents for the extremes in three types of traits: prostrate *vs.* erect growth habit, early *vs.* late flowering time, and short vs. tall. Results indicated that individual wild oat plants in natural populations are often heterozygous for genes governing quantitative characters and that much of the variability observed within individual families can be ascribed to segregation.

In Imam and Allard's (1965) discussion of their results, genetic variation was described to take the form of a mosaic where localized differentiation were superimposed on patterns of differentiation associated with larger geographical areas. The nature of the differentiation from site to site and region to region in wild oats suggest that the variations observed had developed in response to and are maintained by natural selection. One possible explanation offered for the variation in region and site is that populations composed of mixtures of genotypes differing in morphology, physiological requirements and phenology may exploit the available moisture, light and nutrients more efficiently than a genetically uniform population. In addition, Imam and Allard (1965) suggested that the genetic heterogeneity in wild oats increases the probability that some genotypes will do well regardless of season-to-season fluctuations in environment. The authors go on to hypothesize that a simple and effective system for the exploitation of complex mosaic habitats, at least for annual plants, would be the development of "all purpose" genotypes with ample phenotypic plasticity to handle the range of local conditions. Varying soil types, fertility, and moisture regimes within fields and areas associated with disturbances in pastures and right-of-ways are examples of the varying habitats wild oats are able to inhabit.

Imam and Allard (1965) also provide the following explanation of how the genetic system of wild oats appears to equip the species for the occupation of complex habitats. The occurrence of regular low levels of outcrossing between different coexisting genotypes sets the stage for the recombination that is needed for genotypic diversity development for increasing adaptation to specific environmental niches. At the same time inbreeding provides that most of the individuals in a population are homozygous, thus providing for the maintenance of genotypes that are well adapted to specific niches. Wild oats may have evolved a diverse genetic system in which appropriate compromises between the high recombinational potential of outbreeding combined with inbreeding to hold together desirable complexes of genes (Imam and Allard, 1965).

Marshall and Jain (1969) conducted similar research comparing genetic polymorphism in *Avena fatua* and *A. barbata* in two regions of California. *A. fatua* was found to be more polymorphic than *A. barbata* in both regions studied. In areas of both regions where species were in mixed stands, each species had less polymorphism than when each species was in a pure stand. It was believed that these two species had broadly overlapping requirements and the niche occupied by each species is likely to have less mixed than pure stands because of selection imposed by mutual interference. It was concluded that the observed polymorphisms were adaptive, and differences in the degree of environmental heterogeneity account in part for the differences between populations and regions in degree of polymorphism. Marshall and Jain (1969) also reasoned that phenotypic plasticity may be a more important adaptive mechanism for the *Avena* species than genetic diversity contrary to the hypotheses proposed by Imam and Allard (1965).

More recent work by Darmency and Aujas (1987) studied the genetic structure of a wild oat population and its possible shifts over time. Forty-seven phenotypes were observed over five years for inheritance of seed morphology, seed avenin pattern, leaf esterase pattern, and vernalization requirements. Only five phenotypes were consistently present in all samples. The five phenotypes represented an average of 78% of the population. However, the remaining 22% of the population was made of 42 other

minor phenotypes that represent a background of phenotypic diversity which may be important genetic potential for further population adaptation.

Wild oat is a hexaploid species (O'Mara, 1961). Whether there is a relationship between ploidy level and genetic diversity and overall adaptiveness of a plant is not known. However it may be postulated that plants with higher ploidy levels may be more capable of adaptation due to the increased copies of chromosomes and subsequent increased mutation sites. The converse to this argument would be that increased mutation potential could produce increased lethal mutations as well as those improving fitness.

Resistance

The World Health Organization defines resistance as "the development of an ability in a strain of an organism to tolerate doses of a toxicant which would prove lethal to the majority of individuals in a normal (susceptible) population of the species" (World Health Organization Expert Committee on Insecticides, 1957). Mullin and Scott (1992) define cross-resistance as, "resistance to one compound that confers by a common mechanism resistance to another xenobiotic", while multiple resistance is "resistance to more than one or a class of compounds due to coexistence of separate mechanisms."

Resistance of a wide range of organisms to control measures, particularly chemical controls, has become a widespread problem in medicine and agriculture. Recently, bacterial strains resistant to all available antibacterial agents were identified among clinical isolates of some bacterial species (Tomasz, 1994). Selection of resistant bacteria began on a global scale in the early 1940's, with the introduction of the first penicillins into clinical use (Tomasz, 1994). The problem appears to be expanding quickly. *E. coli* were not resistant to fluoroquinolones (a common family of antibiotic) used between 1983 and 1990. However, 28% of isolates were found to be resistant in 1993. This change corresponded to an increase in the rate of flouroquinolone use from 1.4% of patients in 1983 through 1985 to 45% of patients in 1991 through 1993 (Murray, 1994). Currently, 60% to 90% of all isolates of coagulase-negative Staphylococci are resistant to methicillin, another antibiotic (Tomasz, 1994). Over-use, particularly prophylactic use of

antibiotics, and unenforced infection-control or sanitation measures appear to be speeding the rates of resistance development (Murray, 1994).

Resistance has been documented for over 500 arthropod species, particularly flies, caterpillars, beetles and mites (Georghiou, 1990). The first documented case of insecticide resistance in arthropods was 1908 in Washington for the San Jose scale (*Quadraspidiotus perniciosus*) to lime sulfur (Melander, 1914). More recently, Colorado potato beetle, *Leptinotarsa decemlineata*, has developed so many resistance mechanisms that its control is no longer assured even with novel insecticides (Mullin and Scott, 1992). Incidence of resistance in the field appears to be correlated with the length of time an insecticide has been used. Fungicide resistance in plant pathogens has been documented since 1940 but has increased dramatically over the last 15 years with the introduction of systemic fungicides (Eckert, 1988). Resistance has been documented in at least 150 species of plant pathogens (Croft, 1986; Eckert, 1988).

Herbicide resistance has a more recent agricultural history than insecticide and fungicide resistance. Herbicides have been used extensively for weed control since the late 1940's. The use of herbicides for the last 40 years has selected for resistant biotypes within formerly susceptible plant populations. The first North American report of herbicide resistance was in 1970 by Ryan (1970) for common groundsel (*Senecio vulgaris*) in Washington state. It was actually discovered in 1968 , due to a complaint in a conifer nursery after frequent and massive doses of simazine and atrazine had failed to control common groundsel. Since then, and particularly in recent years, the number of reported cases of herbicide resistant weed species has rapidly increased, as has the variety of herbicides to which resistance has evolved. At least 57 weed species have evolved resistance to triazine herbicides, a class of herbicides primarily used for weed control in corn (Holt et al., 1993). In addition, at least 60 species have biotypes resistant to one or more herbicides from 14 other herbicide classes (Holt, 1994; LeBaron, 1991). One or more resistant species has arisen in 31 states of the United States, four provinces of Canada, 18 countries in Europe, and Israel, Japan, Australia, and New Zealand (Holt and LeBaron, 1990).

Herbicide Resistance

Herbicide resistance is defined as the inherited ability of a previously herbicide susceptible plant to not be controlled by the herbicide (Gressel, 1990). Many different mechanisms of resistance are possible. Herbicide resistance in weeds is most often attributed to an alteration of the site of action of the herbicide, i.e. structural change of a herbicide binding site. Resistance may also result from differences in herbicide uptake, translocation, or a plant's ability to metabolize a compound or, conversely, not metabolize and therefore prevent activation of the herbicide (Warwick and Marriage, 1982).

The intense selection pressure of the continuous use of herbicides over the past four decades has resulted in the selection of resistant biotypes. Natural selection was defined by Maxwell and Mortimer (1994) as the differential contribution of individuals to future generations through unequal reproduction and survival. Some progeny have a greater likelihood of survival than others living in a specific environment (Maxwell and Mortimer, 1994). The specific environment, in this case, includes the presence of a herbicide(s) among other farming practices.

The following is a description of the requirements for resistance evolution taken from Maxwell and Mortimer (1994):

> Heritable (genetic) variation for a resistance trait and natural selection are the two precursors for the evolution of herbicide resistance. Given the existence of genetic variation in a species, the rate of evolution will be determined by the type of inheritance exhibited by the species along with the intensity of the selection pressure. Persistent application of a herbicide to a population would be considered an example of recurrent selection in which there is a progressive shift in average fitness of populations of weeds exposed to herbicide.
>
> The efficiency of the herbicide, the frequency of use and the duration of effect contribute to selection intensity. Efficiency of the herbicide is a measure of the relative mortality of the target weed species and the relative reduction in seed production. The frequency of use refers to how persistently the herbicide is used, resulting in selection of resistant types. Selection duration is a measure of the period of time over which phytotoxicity is imposed by a herbicide. For example, a herbicide that has a short selection duration used on a species that germinates over a long period of time would apply decreased selection intensity as compared to a herbicide that would select against all of the germinating cohorts. Both intensity and duration interact to give seasonal variation in the selection pressure imposed on a weed species according to their phenology and growth. Many other factors, such as the mode of inheritance, number of resistant traits, mating behavior, and gene flow also affect the rate of herbicide resistance evolution.

Genetic variation is essential for the evolution of herbicide resistance. Genetic variability may take the form of variation inherently present in a species such as polymorphism in wild oat (Imam and Allard, 1965; Darmency and Aujas, 1987; Marshall and Jain, 1969), or spontaneous gene mutation (Gasquez and Compoint, 1981; Warwick and Marriage, 1982), which would be necessary if the population did not already possess the alleles for resistance. The rates at which herbicide resistance mutations occur are unknown and are likely to differ with species and herbicide class (Jasieniuk and Maxwell, 1994). In addition, large weed populations would have a higher probability of occurrence of resistance mutations than smaller populations. Genetic variability may contribute to herbicide resistant mutations prior to exposure to herbicides. A genetic mutation that confers resistance that has an initial frequency within the population due to genetic variability or large numbers within that population would allow for rapid development of a resistant population (Jasieniuk et al., 1994a).

Adaptation , in response to a herbicide, is only possible if the genes encoding for resistance have a sufficiently large phenotypic effect to allow the survival of a few individuals in a single generation (Jasieniuk et al., 1994a). Polygenic inheritance of resistance would require a longer time for development because individuals would need to recombine for many generations in order to acquire multiple resistance alleles (Jasieniuk and Maxwell, 1994). Gressel (1994), on the other hand, states that initial low dose rates of a herbicide facilitate rapid evolution of resistance. The nature of polygenic inheritance is such that there are small increments of increase in resistance in the population over time as herbicide dose rates increase (Gressel, 1994).

In self-fertilizing species, a resistance mutation associated with a recessive allele would have greater likelihood of establishing (Charlesworth, 1992). As Jasieniuk (1994a) notes, the rapidly increasing frequency of homozygotes would result in the selection of alleles by the herbicide. Resistance to trifluralin in yellow foxtail (Setaria viridis), a 99% self-pollinating plant, was determined to be controlled by a single recessive allele (Jasieniuk et al., 1994b).

The interaction between genetic diversity for the herbicide resistance trait and the intensity of selection for resistance will be the major driving factors influencing the onset of resistance (Maxwell and Mortimer, 1994). Once the trait is present in the population, whether the trait is dominant or recessive and the plant is a cross- or self-pollinator, the continuity of use and the efficacy of the herbicide will determine the rate of expansion of the resistant population.

Gene flow may drastically influence the spread of resistance across a region and within individual agricultural fields. Resistant pollen and seed provide a source of resistant genes. Success of gene flow by wind-mediated pollen dispersal, as well as insect and animal dispersal, are dependent on the amount of viable pollen released, distance between possible mates, and if release of pollen corresponds to time of receptive stigmas. Ghersa et al. (1991) observed that diclofop-resistant *Lolium multiflorum* extruded anthers and subsequently released pollen later than the susceptible biotype, suggesting that neighboring plants had a higher probability of pollination with susceptible pollen.

Seed may be moved from field to field via agricultural equipment (McCanny and Cavers, 1988; Maxwell and Ghersa, 1992). Fay and Stewart (1981) suggested that in Montana, the combine is the single most important reason for the spread of wild oats. Gene flow through seed movement within fields was thought to be small (Howard et al., 1991).

The speed at which a resistant population will become established is dependent upon the relative fitness of resistant biotypes as compared to their susceptible counterparts. Herbicide rotations of various modes of action and various cultural practices may allow for time periods when resistant and susceptible biotypes will compete without selection by the herbicide. Differing fitness levels between resistant and susceptible types may allow the susceptible type to leave more offspring than another (Holt and Hill, 1994). Holt and Hill (1994) also suggest that the susceptible type's superior ability to survive and reproduce without the presence of the herbicide will have an evolutionary advantage relative to the resistant type in that environment. A gene that replaces another in a population may be less adaptive initially if it has some physiological disadvantages relative to the original gene (Haldane, 1960). In

example, the normal regulation of an enzyme may render it resistant to a toxicant but may interfere with its original function. The new genotype may be less efficient and subsequently have reduced reproduction relative to the susceptible wild type in the absence of selection by the toxicant (Uyenoyama, 1986). Decreased reproduction of the new biotype will decrease the frequency of the new gene in the absence of the selection agent. The decrease in fitness may correspond to less advantageous resource allocation and relative survivorship of the plant throughout its life cycle (Roush et al., 1990; Holt, 1990), taking the form of altered seed germination processes, plant development, or seed quality. Without fitness differences between resistant and susceptible biotypes, management of the evolved biotypes will become difficult. As described by Mathews (1994), "Unless there is a substantial fitness penalty, resistant biotypes will not be eliminated from a population by selective mortality in the absence of herbicide application within an economically viable period of time."

Wild Oat Resistance to Herbicides

The inherit genetic variability of the wild oat species has allowed for variation in the responses of some wild oat populations to herbicides. Jacobsohn and Andersen (1968) reported large differences in response to the herbicides diallate, triallate, and barban among lines of wild oats collected in the Red River Valley of North Dakota. Continuous distribution patterns of treated plant heights and dry weights as a percent of the control for all three herbicides suggest that response to the herbicides is quantitatively inherited, or multigenic. Jacobsohn and Andersen (1968) concluded that wild oat populations could develop resistance to herbicides in the Red River Valley under the selection pressure of herbicide applications, and that these populations could develop from genotypes already present without need for mutation or recombination of genes. Jana and Naylor (1982), in an attempt to ascertain the possible extent of wild oat tolerance to triallate, determined that wild oat populations with a history of triallate use (13 years) were more tolerant to triallate than fields with no history of triallate use. Jana and Naylor (1982) hypothesized that the slow evolution of wild oat tolerance to triallate was due to: 1) nonuniform

seed maturation and seed germination, 2) a relatively large soil seed bank, and 3) multigenic inheritance to triallate resistance. Work conducted by Price et al. (1983) with the herbicides barban and bromoxynil demonstrated that wild oat populations from areas historically unexposed to those herbicides may differ in their response, and therefore may have genetic variability controlling the response to herbicides. A phytotoxicity rating (PTR), which was significantly correlated with number of juvenile tillers, decreased leaf stages, decreased dry weights, decreased seed production, increased number of fertile tillers, delayed flowering, and diminished height and dimensions of flag leaves was selected as an index of herbicide response for analysis. Between-family variances in PTR indicated that wild oat populations were indeed polymorphic and that their response to herbicides was more variable than could be explained by expected mutation rates (Price et al., 1983).

The work that had been conducted previous to the 1980's investigated the initial frequency of resistance in populations as a result of genetic variation or phenotypic plasticity. More recently research has focused on the evolution of resistant populations.

Thai et al. (1985) studied the reaction of eight wild oat populations to triallate that were collected from across Saskatchewan. Four of the populations had received triallate for ten years and four had no known use of any herbicides. Emergence, survival to produce first leaf, mesocotyl length, first-leaf length, and seedling height were measured. In the presence of triallate at a rate of 1.0 mg a.i. kg^{-1} of soil resistant oats had a higher seedling emergence and percent survival to produce the first leaf, longer mesocotyl and taller seedlings. Significant differences in triallate reaction were found within and among triallate exposed and unexposed populations. Within population variation for seedling emergence and survival was higher in herbicide unexposed than exposed populations. An additional screening of the wild oat populations with the herbicides asulam, difenzoquat, flamprop, sethoxydim, and diclofop revealed that families contained progenies resistant to both triallate and diclofop. Later work (Joseph et al., 1990), exemplified the difference in resistance levels between resistant and susceptible types. Photosynthesis was significantly reduced in seedlings of both biotypes after treatment with diclofop at a

rate of 0.70 kg a.i. ha^{-1}, but the resistant type was able to recover. Joseph et al. (1990) hypothesized that the nearly complete recovery of the resistant biotypes nine days after herbicide treatment implied that translocation or herbicide metabolism differences might be involved in the resistance response.

Somody et al. (1984) screened 88 accessions of wild oat from ten western states of the United States for resistance to diallate, triallate, barban, diclofop, difenzoquat, flamprop, and MSMA (monosodium methanearsonate), individually and in some combinations to determine the variation in herbicide response of accessions from various areas around the country. Resistance was not confined to areas that were treated with herbicides, indicating that resistance can occur without selection due to genetic variability inherent in the samples. Some accessions were found to be resistant to more than one herbicide, but none were resistant to all the herbicides. Cross-resistant combinations included: triallate, diallate, and MSMA; difenzoquat and barban; diclofop and barban; and flamprop and MSMA.

In the fall of 1990 complaints were received about the lack of wild oat control provided by diclofop-methyl and, a newly recommended aryloxyphenoxy propionate herbicide, fenoxaprop-ethyl in three wild oat populations in Manitoba (Heap et al., 1994). Field histories indicated that these wild oat populations had received repeated applications of diclofop-methyl and sethoxydim, a cyclohexanedione, over the previous 10 years. Heap et al. (1994) attempted to determine the levels of cross-resistance in these populations. Samples were treated with diclofop-methyl, fenoxaprop-p-ethyl, fluazifop-p-butyl, and quizalifop-ethyl (aryloxyphenoxy propionate herbicides), and clethodim, sethoxydim, tralkoxydim (cyclohexanedione herbicides), all of which are inhibitors of acetyl-coenzyme A carboxylase (ACCase). The three populations were resistant to diclofop-methyl, fenoxaprop-p-ethyl, and sethoxydim. It was concluded that resistances had evolved independently because of the contrast in cross-resistance patterns and the many kilometers between the sampled fields. Heap et al. (1994) suggested that different mutations in the gene coding acetyl CoA carboxylase may have been able to confer varying degrees of resistance to the ACCase inhibitors.

Diclofop resistant wild oats were discovered in the Pacific Northwest in the Willamette Valley of Oregon in 1990 (Seefeldt et al., 1992). Seefeldt et al. (1992) reported two populations of wild oat that were resistant to treatments of diclofop, and fenoxaprop with 2,4-D and MCPA , and an additional population that was resistant to diclofop only. Eight more fields were later confirmed to be resistant to diclofop (Seefeldt et al., 1992). In 1994 Seefeldt et al. (1994) further investigated cross resistance of eight diclofop resistant wild oat biotypes. All biotypes were resistant to diclofop. Only four were also resistant to fenoxaprop plus 2,4-D and MCPA. Seefeldt et al. (1994) proposed that the variable response to these herbicides was due to different mutations of the gene coding for ACCase (Seefeldt et al., 1994). The diclofop resistant wild oats were generally resistant to other aryloxyphenoxy propionate herbicides, however only one biotype was resistant to all the ACCase inhibitors. Seefeldt et al. (1994) hypothesized that because wild oat is a hexaploid, mutations of different isoforms of ACCase might occur. Multiple alleles for ACCase, combined with multiple mutations at the site of action and possibly more than one mechanism of resistance, could explain the amount of variation in cross-resistance.

Resistance in wild oat has not been confined to the aryloxyphenoxy propionate and cyclohexanedione herbicides. In 1990, O'Donovan et al. (1994) investigated complaints of poor triallate performance in Alberta. Triallate is a thiocarbamate herbicide. Fifteen of 34 fields sampled were highly resistant to triallate when applied at a rate of 1.7 kg ha^{-1} (equivalent to the recommended field rate). It was later discovered that all the triallate resistant populations were also resistant to difenzoquat, a pyrazolium herbicide structurally dissimilar to triallate, at a rate of 1.7 kg ha^{-1} (twice the recommended field rate) (O'Donovan et al., 1994). Triallate at 3.4 kg ha^{-1} had little or no effect on the resistant populations. Early difenzoquat symptoms included chlorosis and necrosis seven days after application, however triallate resistant populations resumed growth by 21 days while triallate susceptible populations ceased growth at seven days after treatment. Field histories indicated that all populations resistant to triallate and difenzoquat had histories of continuous barley and triallate for approximately 15 years, however, the use history of difenzoquat was not mentioned. O'Donovan et al. (1994) suggested that,

because difenzoquat is rarely used for wild oat control in the fields where resistance developed, continuous triallate use selected for resistance to difenzoquat and triallate.

Recent research suggests that triallate and other thiocarbamates are metabolized to more active compounds which inhibit very long-chain fatty acid synthesis in pea (Pisum sativum L.) (Abulnaja and Harwood, 1991). Wheat cultivars tolerant to triallate had more ^{14}C-metabolites than their susceptible counterparts (McMullan and Nalewaja, 1991). O'Donovan et al. (1994) suggests that differential triallate metabolism could play a role in the triallate resistance mechanism.

Spatial Distribution of Weeds

The spatial distribution of weeds within fields as a result of agronomic practices and soil physical and chemical properties has been studied in order to improve the economic efficiency of weed management. Interest in reducing herbicide use and more accurate herbicide application has driven research to develop an understanding of the distribution of weeds in fields to improve the economic return on crops. Spatial statistics may be useful in describing the spatial structure of weeds, particularly a weed's establishment and spread.

Using a simple weed threshold model, Thurston et al. (1990) determined that the spatial distribution of weeds in a field could have a substantial effect on the economic weed density threshold. A weed density of 10 plants m^{-2} spread uniformly across the field resulted in a yield loss of 15% per hectare, but if that density of weeds was compressed into 10% of the field to create patches with densities of 100 plants m^{-2}, yield loss was reduced to 5% per hectare. Brain and Cousens (1990) studied *Bromus sterilis* in winter wheat to determine the effect of the weed distribution on predicting the yield loss assuming either a random or negative binomial distribution of the weeds. The assumption of random distribution under-estimated the yields in the presence of aggregation. However, the error at lower weed densities, where practical control decisions are made, would be minimal. Wiles et al. (1992), looking at a multi-weed situation with the option of various post-emergence herbicide treatments, tested a model assuming either a

negative binomial distribution (patchy) or regular distribution of weeds within field. Overall, the cost of assuming a regular distribution was low. Errors most typically encountered resulted in recommending more intensive control than was needed. Wiles et al. (1992) suggested that determining the actual distribution of weeds would be costly, however, modeling weed distributions in crops of high value or low competitive ability may be worthwhile depending on how decision makers react to risk. Mortenson et al. (1993) determined that a negative binomial distribution best characterized weed seedling spatial distributions found in soybeans.

Spatial statistics, first developed in the mining industry to describe spatial variation, develop maps, and improve sampling of mineral deposits, are being adopted by field ecologists in studies of trees, pathogens, seed banks, soil characteristics, and emerged weed populations where spatial autocorrelation has made the use of classical statistics troublesome (Yost et al., 1982; Noe and Baker, 1991; Cohen and Spies, 1990; Chellemi et al., 1988; Robertson et al., 1988; Halstead et al. 1990; Donald, 1994; Mortensen et al., 1993). The spatial distribution of common lambsquarter (Chenopodium album) in no-till soybean was analyzed by Cardina et al. (1995) using both classical and spatial statistics. The spatial structure of the weed population was described using semivariograms, which is a graph of the average variance between two points over distance, indicated spatial autocorrelation at distances near 16 m, indicating that point (density) pairs up to that distance apart were spatially dependent, or likely to be similar.

Holt and LeBaron (1990) have discussed in which states of the United States herbicide resistant weeds are distributed. The spatial structure of resistant weeds across a region and within fields has not been investigated to this point. Spatial statistics may be useful in determining how resistance spread or continues to spread across a region and within fields. Aggregation of fields containing herbicide resistant weeds may indicate specific introduction or resistance selection sites. The amount or pattern of aggregation may result from mediated gene flow or some agronomic practice(s).

Fairfield Bench

The Fairfield Bench is a region of approximately 20,000 ha that is situated on the eastern side of the Rocky Mountains approximately 56 km northwest of Great Falls, Montana. The elevation of the area is approximately 1188 m above sea level. The mean annual precipitation is 30 cm, 18 cm, or 60%, of which comes in the agriculturally important period of April to July. The area has 120 frost free days with the average last spring freeze being May 25 and the average first fall freeze being September 25. The mean annual air temperature is 24 C with a mean annual minimum of -14 C and a mean annual maximum of 53 C (Caprio and Nielsen, 1992). The Greenfield Irrigation District manages the irrigation needs of the Fairfield Bench. Irrigation water is taken from the Sun River, a tributary to the Missouri River. Flood irrigation is the dominate form of irrigation, however pivot and wheel line are also used .

The majority of the soil of the Fairfield Bench is classified as Rothiemay loam (Clark, 1982). This soil occupies terraces and fans with zero to two percent slopes. The Ap horizon of this soil is grayish brown to brown with a fine granular structure, is very friable, slightly sticky and plastic, has few pebbles, and is mildly alkaline (Clark, 1982).

Farms on the Fairfield Bench range in size from 200 to over 600 hectares and are family owned. The Fairfield area has been raising continuous barley since the 1960's. In the spring the previous year's stubble is burned off which allows the ground to warm in the early spring for planting and reduces disease due to raising repeated barley crops. Barley is planted early in order to take advantage of spring moisture and in order to ensure that harvest takes place before fall rains occur which cause the crop to sprout while in the field, decreasing crop quality. Traditionally triallate is applied to fields prior to seeding for wild oat control, however, the wild oat post-emergence herbicide imazamethabenz has become more popular since its release in 1989. The post emergent herbicide diclofop is not popular in the area because of the potential for crop injury, and difenzoquat is not commonly used because of erratic weed control. The crop is irrigated two to three times per growing season depending on moisture conditions. The crop is typically

swathed when the barley is in the hard dough stage and allowed to dry prior to being threshed with a combine.

The barley crop is marketed for malt in beer production when crop quality is high. The eight year average malt barley price in Montana is $2.42 per 21.7 kg (1 bu)[1], however Anheuser-Busch® contracts for malt barley in 1993 were for $6.25 per 45 kg (2 bu). The average yield for irrigated barley in Teton county, which the Fairfield Bench belongs to, in 1993 was 3650 kg ha-1 (67 bu/acre)[1]. The majority of growers raise their crops under contract with Anheuser-Busch®, the only brewing company that has an elevator in the immediate area. The brewing company has a zero tolerance for wild oats in barley marketed as malt. Barley that does not meet malt standards is sold as feed barley to elevators outside the area. The eight year average price for feed barley in Montana is $1.87 per 21.7 kg (1 bu)[1].

The edaphic conditions of the Fairfield Bench region and the inexpensive control of wild oats through the use of triallate have allowed for continual production of high quality malt barley for the past 15 to 20 years. However, the recent development of wild oat populations that are resistant to triallate have begun to interrupt malt barley production by decreasing crop quality because of wild oat seed being a contaminant in the barley seed, which is grounds for rejection of the crop by the company. Resistance to difenzoquat and diclofop methyl, two of the three major alternative post emergent herbicides has also evolved in the region. In some fields, imazamethabenz is the only remaining wild oat control tool.

Objectives

The objectives of the following work on wild oat resistance to triallate were to: 1) determine the frequency and distribution of triallate resistant wild oat populations on the Fairfield Bench of Montana; 2) determine what correlations may exist between farming practices and the occurrence, level, and increase of resistance in fields sampled in 1992 and 1993; 3) determine the level of accuracy in sampling for resistance in 1992; 4) determine the level of pattern and variability of resistance on a regional and field

[1] Montana Agricultural Statistics Service, vol. 31, 1994.

scale; 5) assess the spread of resistance via roadways and irrigation ditches; and 6) initiate experiments to determine the possibility of cross-resistance to other wild oat herbicides in wild oat populations from the Fairfield Bench.

CHAPTER 2

FREQUENCY OF RESISTANCE AND AGRONOMIC PRACTICES INFLUENCING RESISTANCE

Introduction

The total area infested with wild oats in Canada and the United States is estimated at over 25 million ha (Nalewaja, 1970). Montana farmers indicated that wild oats were by far their most important weed problem and, along with weather, the most significant problem in small grain crop production (Fay and Rhode, 1980). Wild oats can cause significant yield reductions in barley and wheat at high densities and when they emerge at the same time or before the crop (O'Donovan et al., 1985; Dew, 1972).

In north central Montana, where there are few rotation crops, wild oat management options are restricted to fallowing or applying herbicides (Taylor and Burt, 1984). Several post emergent herbicides are available for wild oat control in irrigated malt barley and are preferred as management tools because they allow a more accurate assessment of infestation prior to treatment than pre-emergent herbicides. However, postemergent treatments are more expensive and produce more erratic results than triallate, the most common pre-emergent herbicide for wild oat control in small grains in Montana (Fay and Rhode, 1980).

The pre-emergent herbicide triallate is typically applied as a liquid or granular formulation and incorporated in the soil prior to planting for the most effective control of wild oats in small grain crops (Banting, 1970). Approximately 80% of Montana growers apply triallate for wild oat control (Fay and Rhode, 1980). Triallate is a thiocarbamate herbicide applied in either of two formulations: 1.4 to 1.7 kg a.i. ha^{-1} of the 4 EC liquid formulation, or 1.4 to 1.7 kg a.i. ha^{-1} of the 10G granular formulation (Whitson et al., 1983). Triallate is soil incorporated to prevent loss by volatilization and to improve herbicide contact with the emerging wild oat stem apex (Miller, 1990).

Several studies have shown that continuous treatment of wild oats with triallate can result in increased levels of tolerance to the herbicide (Jana and Naylor, 1982; Somody et al., 1984; Thai et al.,

1985). Triallate-tolerant wild oats have also demonstrated tolerance to some postemergent herbicides (Somody et al., 1984; Thai et al., 1985). In recent work wild oats in areas of the Pacific Northwest and Canada have been found to be resistant to the postemergent herbicides diclofop-methyl, fenoxaprop-p-methyl, and sethoxydim (Seefeldt et al., 1994; Heap et al., 1994). Of 34 fields sampled in Alberta in 1990, 15 were found to be highly resistant to triallate at a rate of 1.7 kg ha^{-1}. All triallate resistant populations were also resistant to difenzoquat applied at 1.7 kg ha^{-1} (O'Donovan et al., 1994).

Decreasing triallate performance resulted in complaints from malt barley producers on the Fairfield bench (north central Montana) in 1992. The objective of this study was to determine the frequency of triallate resistance in fields on the Fairfield bench in 1992 and 1993, and to determine what relationships may exist between the occurrence of resistance and farming practices used in the resistant fields.

Materials and Methods

Frequency of Resistance - 1992 Sampling

The collecting of initial samples took place in 1992. The sampling region consisted of 18130 ha (70 sections) of the Fairfield Bench. The sampling region was divided into seventy 259 ha sub-sampling areas along section lines. These were divisions created by roads, and usually form boundaries of fields. Within each sub-sampling area, four fields were randomly chosen from aerial photographs. A total of 280 fields were selected, however some fields had been converted to pasture so a total of 237 fields were sampled for resistance (Figure 1, see pocket in back).

Each field was sampled by a collector walking across the field four times in a "W" pattern, avoiding collection near field edges and in obvious herbicide skips. Every 12 m the collector removed the seed from a single panicle nearest to the collector. Each field sample was considered a bulk composite sample. Field samples were sun-dried in the greenhouse and threshed to remove glumes, awns, break up florets, and homogenize the sample as much as possible. Prior to screening, a subsample from the field

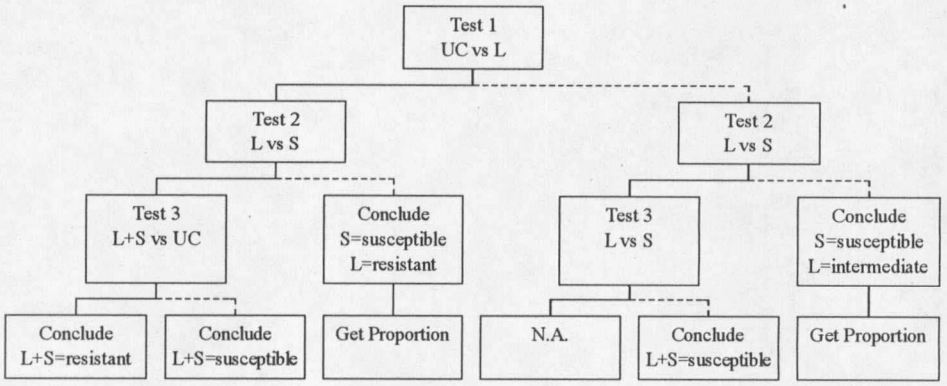
bulk composite samples (approx. 200 seed), were imbibed in tap water for 24 h, pierced on the dorsal side to stimulate germination, then placed in a growth chamber at 19 C to determine germination rate.

The response to triallate of the 237 fields sampled was assessed. An aqueous solution of liquid triallate was applied at 1.1 kg a.i. ha^{-1} per 2.54 cm of greenhouse soil to be added to steel flats 45 cm by 61 cm by 9 cm deep filled with 6 cm of untreated greenhouse soil. Treated soil was mixed thoroughly in a rotary mixer. Seven wild oat field samples and one known susceptible wild oat biotype were planted along the surface in rows of 15 seeds. Rows were 2.54 cm apart. Treated flats received 2.54 cm of the triallate incorporated soil while untreated flats received 3 cm of untreated greenhouse soil. The experiment was a randomized complete block design with three replicates. Flats were placed in a greenhouse receiving 16 h artificial light and set for day/night temperatures of 22/16 C, respectively. Flats were watered daily to field capacity. Twenty-eight days after planting, wild oat seedlings were clipped at soil surface. Seedlings where the shoot had not emerged from the coleoptile were classified as small plants, conversely seedlings where the shoot had emerged from the coleoptile were classified as large plants. This distinction was made in order to decrease variability within reps of samples due to the possibility that resistant and susceptible seed may both be present within a sample. Individual triallate treated plant fresh weights were measured for thirteen randomly chosen field samples that contained both large and small plants in order to determine if a significant difference (0.05 level of significance) existed between large and small classes of plants using a t-test. Seedlings of each size class from a row within a flat were counted and weighed to determine mean fresh weight per plant for each size class. The mean of the three replicate mean fresh weights per plant was then calculated. The treated size class means were compared to each other and to the untreated mean fresh weight per plant of a field sample using t-tests at a 5% level of significance to determine if a sample was resistant or susceptible according to a series of decisions (Figure 2). F- and t-tests (0.05 level of significance) were conducted using PROC TTEST in SAS[2] .

[2] Statistical Analysis System Institute, Inc., Cary, NC (Version 6.0) 27512.

1993 Sampling

Eighteen of the 236 fields sampled in 1992 were randomly selected and resampled for triallate resistance. The 18 fields had histories of triallate in 1992 and 1993, or triallate in 1992 and imazamethabenz in 1993. Eleven fields had received triallate in 1992 and 1993, while 7 had received triallate in 1992 and imazamethabenz in 1993.



Solid line represents no difference. Dashed line represents a significant difference. L = large class, S = small class, UC = untreated control, N.A. = Not Applicable.

Figure 2. The series of decisions used to determine if samples were resistant (R), susceptible (S), or a proportion of R and S.

Resistance Occurrence vs. Farming Practices

A survey of the farmers with fields sampled in 1992 was conducted to determine if there was a relationship between management practices and the occurrence of triallate resistant wild oats. The survey was distributed to growers who participated in a grower meeting conducted at Fairfield, MT in December, 1992. Any grower that was present and owned a field that had been sampled in 1992 was asked to complete the survey and mail it back to Montana State University.

The survey included determining the farming history of each field. Questions included: years of triallate use; the pattern of triallate use over past years; formulation used; rate normally applied; implement used to incorporate triallate; planting date of the crop; in what market the crop was sold (i.e. barley for malt, seed, or feed); possible changes in planting date in recent years; form of irrigation used;

and cropping history from 1988 to 1992. A total of 47 fields were associated with respondents, 30 of the fields had been tested as triallate resistant in 1992 and 17 had triallate susceptible wild oats. This was not a random sample because of the method of distribution. The sample size was low, therefore significant differences between resistant and susceptible fields would be difficult to detect. A single factor ANOVA with a 5% level of significance was used to determine if a difference existed between the resistant and susceptible populations in the number of years of triallate use. The remainder of the questions were analyzed using a Chi-square test of similar population distributions, and a 5% level of significance was used to determine differences.

Results and Discussion

Frequency of Resistance

Two hundred thirty-seven fields were sampled across the Fairfield Bench in 1992. One sample did not contain an adequate amount of mature wild oat seed to allow for resistance testing. Of the 236 samples tested in the greenhouse with triallate applied at 1.1 kg ha^{-1}, 123 were determined to be resistant, 27 intermediate, and 86 were susceptible. Resistant seedlings, although delayed in growth, were equivalent to the untreated control by the end of a 28 day growth period. Susceptible wild oats either never emerged or never progressed past coleoptile emergence. Of the 236 fields sampled and tested for resistance in 1992, 64% contained resistant or intermediate seed while 37% of the samples contained seed that was all susceptible. The intermediate response class was made up of plants within a sample that were significantly smaller ($P<0.05$) than the untreated control and significantly larger than the treated small class seedlings. Intermediate seedlings displayed some of the classic triallate symptoms: decreased height, darker green leaf color, and leaves that were shorter and wider than the untreated, however, these plants continued to grow and appeared capable of reproduction. If triallate resistance is polygenic in wild oats, the plants with intermediate response may be the result of populations accumulating resistant alleles. The intermediate response could also be the result of a partially dominant single gene for resistance. The

possibility also exists that many types of resistance have developed, some more fit and successful than others, these intermediate plants may be a different form of resistance that is less fit than that found in the samples classified as resistant. Intermediate samples were considered to be resistant under field conditions due to the increased efficacy of triallate in greenhouse conditions, which may be due to improved incorporation and increased exposure of germinating wild oats to the triallate treatment.

In some samples large and small plants were present. Large plants were those where the shoot had emerged from the coleoptile, while small plants were those wild oat seedlings where the shoot had not emerged from the coleoptile. The frequency distribution for all fresh weights per plant depicts a large number of plants in the smallest size class (0.03 g) (Figure 3). The mean of large and small plants was found to be significantly different using a t-test at 0.05 level of significance ($P<0.01$) (Table 1). The distinction between large and small size classes was made in order to decrease variability within samples due to both resistant and susceptible seed being present within field samples and allow for the determination of the proportion of resistant seed within samples.

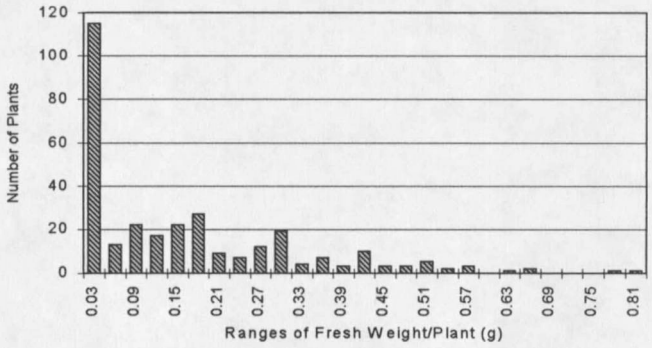


Figure 3. Distribution of triallate treated individual plant weights (g).

Table 1. Mean and standard deviation of large and small plant classes, t-test ($P<0.01$).

	Large Plants	Small Plants
Mean	0.199 g	0.004 g
Standard Deviation	0.185	0.003

One hundred fifty samples contained resistant seed; 58, or 39%, contained 100% resistant seed and 92, or 61%, were made up of a proportion of resistant and susceptible seed. The proportion of resistance in those samples containing resistant and susceptible seed ranged from 15% to 95% resistant seed (Figure 4). Susceptible seed in a sample from a field that had been treated with triallate indicates escapes from the effect of triallate are common. Susceptible escapes in these fields can result from skips in application, uneven incorporation, and /or germination after the triallate has volatilization from the soil. The fields that were 100% resistant may have had more effective and thorough applications, or less likely, those fields evolved resistance earlier and have received continuous triallate for a long enough period of time, allowing the resistant plants enough time to spread over the entire field. Complete versus partial resistant fields may represent different stages in the evolution or spread of resistance in fields.

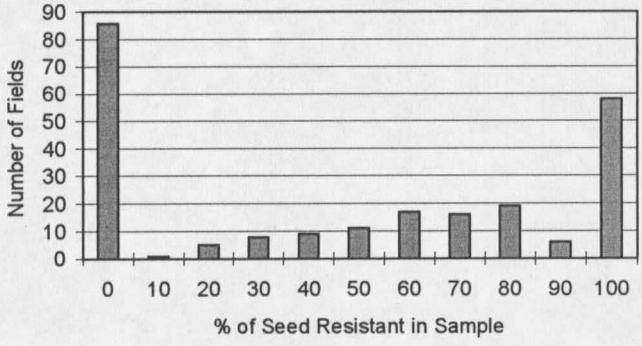


Figure 4. Frequency of different proportions of resistance present in the fields sampled in 1992 on the Fairfield Bench.

1993 Sampling

A mean increase of 24% triallate resistant seed was found within the eleven fields receiving triallate in 1992 and 1993 (Table 2). No difference in the mean percent of resistance in these eleven fields was detected between years 1992 and 1993 (P=0.22) due to one of the fields (field number 93543) which appeared to be a reversal to completely susceptible. Seven of the 11 fields treated both years with triallate

increased in the level of resistance detected, in three of the fields the samples indicated that there was a decrease in resistance while one field remained at 100% resistant in 1992 and 1993.

The 7 fields that received triallate in 1992 and imazamethabenz in 1993 averaged a 51% increase in triallate resistant seed production (Table 3). Two of the seven field samples were susceptible in 1992 and found to be resistant in 1993. A significant difference in the mean percent of resistance between years did exist for this group of seven fields (P=0.007). No significant difference in percent resistance was detected (P=0.22) between the two mean increases between field history types (triallate/triallate and triallate/imazamethabenz). Changing wild oat herbicide from triallate to imazamethabenz did not decrease the amount of triallate resistant seed sampled from fields. A significant increase in resistant seed took place, indicating that resistant types may be at least as fit or more fit than the susceptibles in the absence of triallate and presence of imazamethabenz. The fact that resistant plants remained and may have increased in the system when they were not exposed to triallate suggests that the probability of selection for cross or multiple resistance to imazamethabenz may increase because imazamethabenz resistance may be selected for from a population of wild oats that is maintaining its resistance to triallate.

Table 2. Change in percent resistant (R) seed in samples from fields receiving triallate in 1992-93.

Field #	1992	1993	Change
	% R Seed		
93022	0	94	+94
93033	73	94	+21
93082	60	100	+40
93152	82	72	-10
93274	61	98	+36
93332	67	100	+33
93463	95	84	-11
93543	67	0	-67
93563	69	100	+31
93194	100	100	0
93273	0	100	+100
	Mean = 61% SD = 32.9	Mean = 85% SD = 29.7	Mean = 24% SD = 47.4

Table 3. Change in percent resistant (R) seed in samples from fields receiving triallate in 1992 and imazamethabenz in 1993

Field #	1992	1993	Change
	% R Seed		
93054	57	84	+27
93071	0	82	+82
93121	59	98	+39
93321	0	100	+100
93464	90	100	+10
93484	70	100	+30
93602	33	100	+67
	Mean = 44% SD = 34.6	Mean = 95% SD = 8.2	Mean = 51% SD = 32.8

Eighteen fields were resampled in 1993 of the original 236 fields from 1992. In 1992, no resistance was detected in 4 fields and 14 contained triallate resistant wild oat seed. When the same fields were resampled in 1993, resistance was detected in the 4 fields where no resistance was detected in 1992. No resistant seed was detected in field 93543 (Table 2) when resampled in 1993. Resistance was detected in the remaining 13 fields again in 1993 although proportions did vary. The "W" method of within field sampling and the screening method were successful in detecting resistance in 13 of 14 fields where resistant seed had been detected in 1992. The 4 fields that were susceptible in 1992 and resistant in 1993 showed an average increase of 94% resistance indicating that resistance may be the result of introduction of resistant seed rather than mutation events. Field sample 93543, which contained 67% resistant seed in 1992, was the only sample to go from resistant to susceptible when sampled again in 1993. This could be explained by a field that is dominated by susceptible types with a very low number of resistant wild oats possibly contained within a limited number of small patches which may have been walked through in 1992 but missed in 1993. The "W" within field sampling method and bulk seed testing technique used here are appropriate when sampling a large number of fields where time is a factor and the primary objective is to determine if resistance is present in a field. A more precise method of sampling would be required when working with a smaller number of fields, and the objective is to estimate the frequency of resistance within a field.

The rate of resistant wild oat population growth within fields can be estimated over time by comparing the mean percent of resistance within fields over years. In 1991 a survey of wild oat resistance to triallate was conducted on the Fairfield Bench consisting of 44 fields[3]. Although the 1991 data is based on visual plant injury, it may be used to represent the percent resistance within the fields surveyed and compared to the mean percent resistance determined within the fields treated with triallate in both 1992 and 1993 (Figure 5). The mean percent resistance within fields increases by 32% from 1991 to 1992 and by 24% from 1992 to 1993. The mean percent resistance within fields appears to be increasing exponentially assuming a 74.5% mean rate of increase between years. It may be estimated that in 1989, when reports of poor triallate performance first began, the mean percent resistance within fields was 11%.

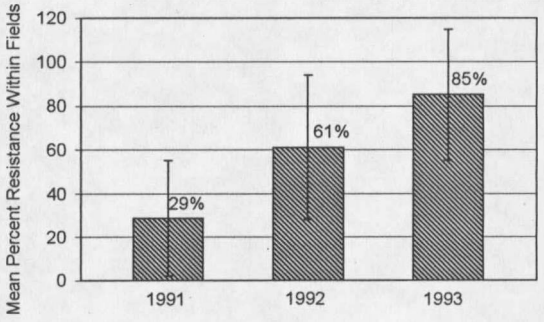


Figure 5. Mean percent resistance within fields for 1991 to 1993. Bars represent one standard deviation of the mean.

Resistance Occurrence vs. Farming Practices

Two hundred and thirty-six fields were tested for the occurrence of triallate resistant wild oat seed in 1992. These fields were managed by approximately 93 growers. Approximately 25% of those farmers who had fields sampled in 1992 responded to the farming practices survey in 1993, representing 20% of the fields sampled in 1992. Thirty of the 47 fields that farming practice histories were obtained for were determined to be resistant to triallate, while 17 were susceptible in 1992.

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[3] Wild Oat Resistance to FarGo in Montana, Report to Dr. Doug Ryerson of Monsanto Corp. by Fay, Trunkle, and Christianson, 1992.

The survey indicated that years of triallate use, cropping history, and incorporation implement were significantly different for resistant and susceptible fields. From 1988 through 1992, 91% of the total fields surveyed were continuous malt barley. The mean number of years that triallate had been used for susceptible fields was 12 years while resistant fields were significantly lower (P=0.03) at 16 years of triallate use (Figure 6). Triallate was used every year in 96% of the fields referenced in the survey (Figure 7). Triallate has been the primary herbicide used for wild oat control in the Fairfield area since the early 1970's. Stephenson et al. (1990) observed that in western Ontario, triazine resistant weeds presented little problem because agronomic practices involved crop rotation, only moderate atrazine use, use of many postemergent herbicides, and inter-row cultivation. Eastern Ontario on the other hand relied predominately on atrazine for weed control in continuous corn production with little cultivation and triazine resistance was a major problem. In a recent study conducted in Alberta, O'Donovan et al. (1994) were unable to relate years of triallate use to resistance due to a lack of farm records, but were able to approximate that in resistant fields triallate had been used continuously for 15 years or longer in continuous barley production.

The five year cropping history was significantly different (P=0.01) for resistant and susceptible fields. An alternative crop or summerfallow was used by the resistant fields only 1% of the time while susceptible fields interrupted the continuous barley cycle 19% of the time between 1988 and 1992 (Figure 8).

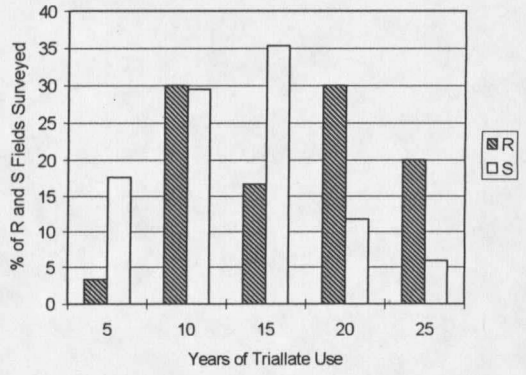

Figure 6. Frequency of years of triallate use on resistant (R) and susceptible (S) fields. Single factor ANOVA (P = 0.03).

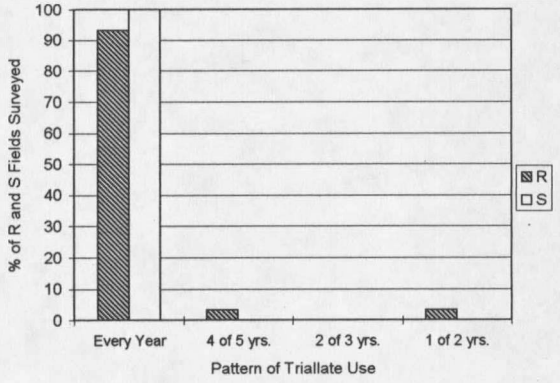

Figure 7. The frequency of resistant (R) and susceptible (S) fields receiving triallate. Chi-square = 1.115 (P = 0.573).

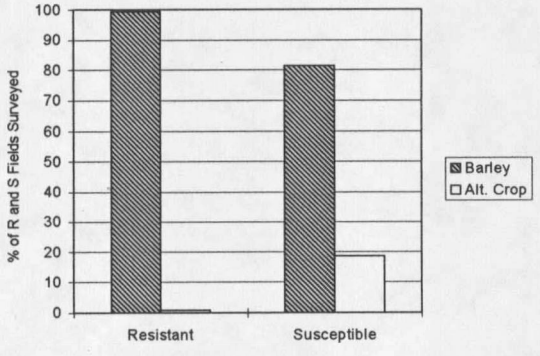

Figure 8. The crops planted from 1988-92 in surveyed fields was a significant factor. Chi-square = 6.68 (P = 0.01).

The implement used to incorporate triallate was significantly different for resistant and susceptible fields in the survey (P=0.019). Fields where a chisel plow was used for incorporation of triallate were more likely to be resistant, while susceptible fields were split between chisel plow and field cultivator (Figure 9). A preplant incorporated herbicide must be incorporated evenly without skips in order to be effective in controlling the target weed. A well incorporated herbicide treatment would have a greater selection intensity than a poorly incorporated herbicide treatment. A field cultivator typically uses shovels as wide as 30 cm, while a chisel plow would have shovel sweeps typically less than 30 cm. In a one pass incorporation of triallate, although not suggested but often implemented, a chisel plow may result in more thorough incorporation than a field cultivator, leading to increased selection for resistant wild oats.

The remaining variables help describe other triallate use characters and farming practices although none of them showed significant differences between resistant and susceptible fields. The granular and liquid formulations of triallate were both used (Figure 10) at the 1.7 kg ha^{-1} and 1.4 kg ha^{-1} rates (Figure 11). There was no significant correlation between formulation or rates of triallate use and the occurrence of resistance. Flood and wheel line types of irrigation account for 76% of the fields surveyed, while pivot, flood, and wheel line in combinations make up the remainder (Figure 12). The malt barley crop in surveyed fields was planted the first and second week of April 73% of the time (Figure 13). Approximately half of the growers (55%) felt that planting dates were progressively moved earlier over previous years due to new crop varieties and seasonal moisture conditions which had created crop quality problems due to late maturity associated with late planting in the past. The barley grown on the surveyed fields was all qualified to be marketed as malt barley, however, two fields had been contracted as seed barley prior to 1988. The wild oat field samples from these two fields were determined to be susceptible to triallate in 1992 and were not sampled in 1993.

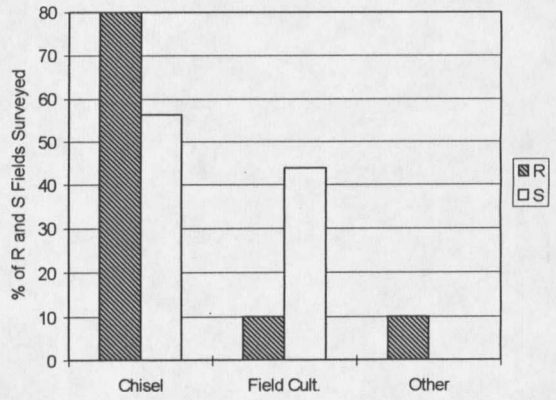

Figure 9. The form of tillage used to incorporate triallate on resistant (R) and susceptible (S) fields. Chi-square = 7.88 (P=0.019).

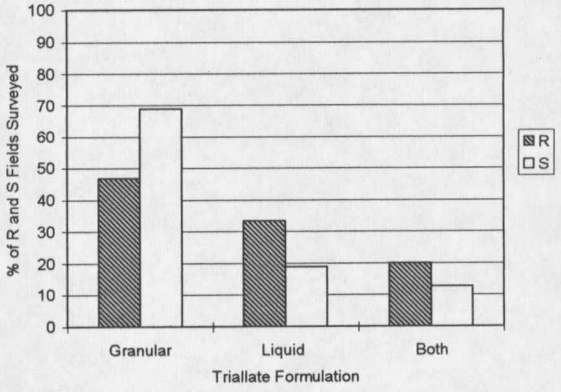

Figure 10. Formulation of triallate applied to resistant (R) and susceptible (S) fields. Chi-square = 2.059 (P = 0.357).

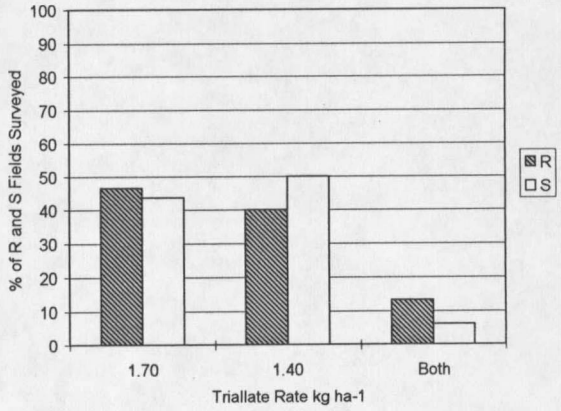

Figure 11. Triallate rate applied to the resistant (R) and susceptible (S) fields. Chi-square = 0.741 (P = 0.69).

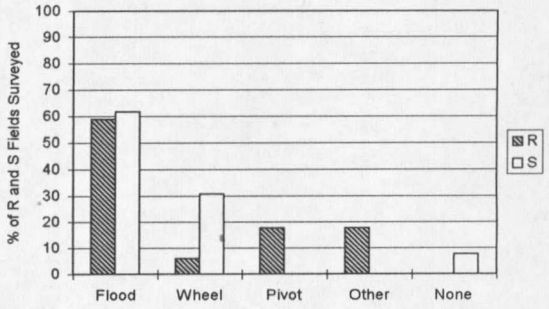

Figure 12. The type of irrigation used in resistant (R) and susceptible (S) fields. Chi-square = 8.643 (P = 0.071).

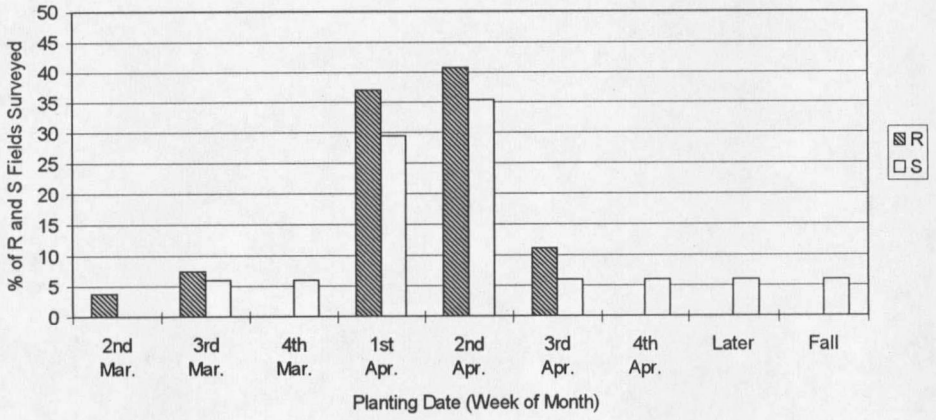

Figure 13. Planting date of the barley on resistant (R) and susceptible (S) fields. Chi-square = 7.59 (P=0.475).

This study demonstrates a significant difference between resistant and susceptible fields for years of continuous triallate use (mean years for resistant fields being 16) as well as a cropping history. This further supports the theory that rotating herbicides and crops, avoiding mono-crop and continuous single herbicide usage systems, may reduce the selection for, and/or spread, of herbicide resistance. Alternative crops may allow for the incorporation of different herbicide chemistries and differential selection through competition for resources and wild oat life cycle disruptions.

CHAPTER 3

REGIONAL AND WITHIN FIELD PATTERN OF RESISTANCE

Introduction

The spatial distribution of weeds within fields as a result of agronomic practices and soil physical and chemical properties has been studied in order to improve the economic efficiency of weed management. Interest in reducing herbicide use and more accurate herbicide application has driven research to develop an understanding of the distribution of weeds in fields to improve the economic return on crops. Spatial statistics may be useful in describing the spatial structure of weeds, particularly a weed's establishment and spread.

Using a simple weed threshold model, Thornton et al. (1990) determined that the spatial distribution of weeds in a field had a substantial effect on the calculation of the economic threshold. A weed density of 10 plants m^{-2} spread uniformly across the field resulted in a yield loss of 15% per hectare, but if that density of weeds was compressed into 10% of the field to create patches with densities of 100 plants m^{-2}, yield loss was reduced to 5% per hectare. Brain and Cousens (1990) studied *Bromus sterilis* in winter wheat to determine the effect of the weed distribution on predicting the yield loss assuming either a random or negative binomial distribution of the weeds. The assumption of random distribution under-estimated the yields in the presence of aggregation. However, the error at lower weed densities, where practical control decisions are made, would be minimal. Wiles et al. (1992), looking at a multi-weed situation with the option of various post-emergence herbicide treatments, tested a model assuming either a negative binomial distribution (patchy) or regular distribution of weeds within field. Overall, the cost of assuming a regular distribution was low. Errors most typically encountered resulted in recommending more intensive control than was needed. Wiles et al. (1992) suggested that determining the actual distribution of weeds would be costly, however modeling weed distributions in crops of high value or low

competitive ability may be worthwhile depending on how decision makers react to risk. Mortenson et al. (1993) determined that a negative binomial distribution best characterized weed seedling spatial distributions found in soybeans.

Spatial statistics, first developed in the mining industry to describe spatial variation, develop maps, and improve sampling of mineral deposits, are being adopted by field ecologists in studies of trees, pathogens, seed banks, soil characteristics, and emerged weed populations where spatial autocorrelation has made the use of classical statistics troublesome (Yost et al., 1982; Noe and Baker, 1991; Cohen and Spies, 1990; Chellemi et al., 1988; Robertson et al., 1988; Halstead et al. 1990; Donald, 1994; Mortensen et al., 1993). The spatial distribution of common lambsquarter (*Chenopodium album*) in no-till soybean was analyzed by Cardina et al. (1995) using both classical and spatial statistics. The spatial structure of the weed population was described using semivariograms, which are plots of the average variance between sampling points over distance between points. Semivariograms indicated spatial autocorrelation at distances near 16 m, indicating that point (density) pairs up to that distance apart were spatially dependent, or likely to be similar when within 16 m of one another.

Our region of study was a 18,130 ha irrigated continuous barley production area approximately 56 km northwest of Great Falls, MT. Wild oat (*Avena fatua*) is an important grass weed in barley production in this area. Wild oat is typically controlled in the barley crop through the application of the herbicide triallate prior to planting in the spring. However, populations of wild oat across the study region have developed resistance to the herbicide. Spatial statistics may be useful in determining how resistance spread or continues to spread across the region and within fields. Aggregation of fields containing herbicide resistant weeds may indicate specific introduction or resistance selection sites. The amount and pattern of aggregation may result from mediated gene flow or some agronomic practice(s). The objectives of this work were to determine the frequency and spatial structure of resistance at two scales, within a region and within a field.

Materials and Methods

Sampling and Testing of Region

Wild oats were sampled in 236 randomly selected fields in 1992 within the study region. Wild oat populations were sampled by a collector walking across each field in a "W" pattern. Seed from a single wild oat panicle was taken from wild oats nearest to 12 m intervals producing a bulk composite sample for the field. Field samples were dried in the sun and threshed to remove glumes, awns, break up florets, and homogenize the sample as much as possible. Prior to screening, sub samples from each of the field bulk composite samples were imbibed in tap water for 24 h, pierced on the dorsal side to stimulate germination, then placed in a growth chamber at 19 C to determine germination rate in preparation for planting.

The response to triallate of the 236 field samples was assessed. An aqueous solution of liquid triallate was applied at 1.1 kg a.i. ha^{-1} per 2.54 cm of greenhouse soil to be added to steel flats 45 cm by 61 cm by 9 cm deep filled with 6 cm of untreated greenhouse soil. Treated soil was mixed thoroughly in a rotary mixer. Seven wild oat field samples and one known susceptible wild oat biotype were planted in rows of 15 seeds. Rows were 3 cm apart in each flat. Treated flats received 2.54 cm of the triallate treated soil while untreated flats received 2.54 cm of untreated greenhouse soil. The experiment was a randomized complete block design with three replicates. Flats were placed in a greenhouse receiving 16 h artificial light and set for day/night temperatures of 22/16 C, respectively. Flats were watered daily to field capacity. Wild oat seedlings were clipped at the soil surface twenty eight days after planting. Seedlings where the shoot had not emerged from the coleoptile were classified as small plants, conversely seedlings where the shoot had emerged from the coleoptile were classified as large plants. A significant difference had been detected between the mean size of small and large plants using a t-test ($P<0.01$) (Table 1, Chap. 2) This distinction was made in order to decrease variability within reps of samples due to the possibility that resistant and susceptible seed may both be present within a sample and allow for the determination of the proportion of resistance within a sample. Seedlings of each size class from a row

within a flat were counted and weighed to determine mean fresh weight per plant for each size class. The mean of the three replicate mean fresh weights per plant was then calculated. The treated size class means were compared to each other and to the untreated mean of a field sample using t-tests at a 5% level of significance to determine if a sample was resistant or susceptible according to a series of decisions (Figure 2, Chap. 2). F- and t-tests were conducted with PROC TTEST in the analysis package SAS. A significant difference between the treated and the untreated fresh weight per plant indicated that the sampled seeds were susceptible to triallate.

The level of resistance was determined for each field tested. Sampled fields were located on a map made of aerial photos (6.25 cm = 1 km). A grid (1 grid increment = 0.1 km) was laid over the map in order to provide each field with an XY coordinate at the field's geometric center. The spatial structure of resistance across the region was analyzed using a data set containing the percent resistant seed and XY coordinate for each field.

Spatial Statistics for Region

Spatial autocorrelation, the likelihood that samples taken near each other are more similar than those taken at a further distance, was analyzed for the regional data set using the semivariance statistic:

$$\gamma_h = \frac{1}{2N_h}\sum_{i=1}^{N}\left[Z(Xi)-Z(Xi+h)\right]^2$$

where γ_h is the semivariance for sample sites separated by distance h (in grid units), Z(x) and Z(x+h) are percent of resistance at points x and x+h, and N is the number of pairs of sample sites separated by distance h (Cardina et al., 1994). The calculated semivariance was plotted against distance (h) between pairs of all sample points to create a semivariogram. All of the pairs of points separated by distance h were used to calculate the value of γ. GS+[4], a geostatistical software package, was used to construct a series of unidirectional, or anisotropic, semivariograms (with regard to direction) and an omni-directional, or isotropic, semivariogram (without regard to direction). Uni-directional semivariograms will determine

[4] Gamma Design Software, Plainwell, MI, (Version 1.1) 49080.

if anisotropy is present in the data. Anisotropy would indicate that there is more spatial autocorrelation along a particular axis, i.e. north-south rather than east-west, which may indicate an elliptical shape to the average patch and a direction of resistance spread. Uni-directional semivariograms were drawn for sample pairs oriented in one of four directions (0^0, 45^0, 90^0 and 135^0) to determine the direction with the strongest spatial autocorrelation. The 0^0 direction corresponds to the north-south axis and 90^0 to west-east. An omni-directional semivariogram was constructed to determine if any spatial autocorrelation exists for all directions combined. The range of influence, or distance up to which sample points are similar, may be interpreted from the omni-directional semivariogram. The least squares procedure of GS+ was used to fit various models to the semivariogram. The model selected by GS+ was then used to estimate values of percent resistance at non-sampled points across the region (called kriging), taking into account the percent resistance at neighboring sampling points and the spatial variance structure of the region. GS+ was then used to create a contour map of estimated levels of resistance for the entire sampling region (Figure 14).

Sampling Within Field

A systematic grid method of sampling was used at 3 sampling sites in 1993 (Figure 1, see pocket in back). Site one was in the untreated portion of a continuous barley field suspected to contain resistance in 1992 but the field was not sampled in 1992. Site two was placed in the triallate treated (1.4 kg a.i. ha^{-1}) portion of the same field. The third site was in a field which had received triallate at 1.4 kg ha^{-1} in 1992 and 1993 and was sampled in 1992 and found to be 100% resistant. A 3600 m^2 grid containing 144, 25 m^2 cells, was placed at each site. Seed from an entire panicle was taken from a single wild oat plant nearest to the center of each cell within the blocks. It is assumed that the individual plants from each cell represent the resistance status of the population of wild oats within the cell and that the sampled wild oat plant's location was the center of the cell.

Screening Within Field

An alternative resistance screening method was used because of the large amount of greenhouse space and time required for a mass screening. Seed samples were screened for triallate resistance using a petri dish bioassay developed by Colliver et al. (1994), where dose response curves indicated that a rate of 1 µL of triallate at 100 ppm and 0.2% surfactant, per seedling in a petri dish containing five seedlings, provided the best delineation between susceptible and resistant seed. Seeds were prepared by removing the lemma and palea from the seed, the caryopsis were pierced on the dorsal side, and the seeds were surface sterilized in a 1.3% bleach solution. Samples were imbibed in distilled water for 4 days in total darkness. Twenty seedlings with coleoptiles 5 mm to 15 mm in length were selected and the lengths recorded. Ten seedlings were divided into two petri dishes to be treated by placing 1µL triallate 100 ppm solution on the tip of each coleoptile using a 10 µL syringe. The remaining ten seedlings were placed into two petri dishes and left untreated. All petri dishes were 100 x 25 mm and contained one disk of #4 Whatman® 9 cm filter paper and 2 ml of distilled water. The vapor phase characteristics of triallate require that the petri dish size, filter paper type, and amount of water added to dishes not vary. Petri dishes were sealed and placed in a growth chamber at 19 C in total darkness. Seven days later shoot length was recorded for the ten replicate seeds for each treatment. Mean shoot growth after seven days following treatment for the treated and untreated portions of each sample were compared using a t-test at 0.05 level of significance. Treated means significantly different than the untreated means were considered susceptible. Results were mapped for each cell and the frequency of resistance determined for each site.

Within Field Spatial Statistics

Spatial point pattern analysis was used to determine if the resistant or susceptible cells within a grid were aggregated, regular, or completely spatially random. If the stochastic process governing the position of resistant or susceptible cells can be characterized as a homogeneous Poisson process, then the process is said to be completely spatially random (CSR), which is the null hypothesis. Coordinate data

sets for resistant and susceptible cell locations for the three sites were analyzed using a program developed by Hamilton et al. (1995). The procedure of spatial point pattern analysis follows the standard notation and terminology of Cressie (1991) and is demonstrated and outlined by Hamilton et al. (1995).

Allow B to denote the site of interest, v(B) will denote the area of B, and let N(B) be a random variable denoting the number of resistant or susceptible cells in B. Two important first order properties of the spatial point process are the mean $\mu(B)=E\{N(B)\}$ and the intensity at point $s \in B$, $\lambda(s) = \mu'(s)$, for which a definition is provided in Cressie (1991). Under CSR, $\lambda=(s)$ and $\mu(B)=\lambda*\nu(B)$. In this study region is rectangular and the density of the resistant or susceptible cells under CSR can be estimated by $\lambda = \frac{N(B)}{\nu(B)}$ (Hamilton et al, 1995).

The second order properties of a spatial process are the second moment, $\mu_2(B_1*B_2) = E\{N(B_1)*N(B_2)\}$, and the second order intensity at points s_1,s_2, $\lambda_2(s_1,s_2) = \frac{d^2\mu_2(s_1 * s_2)}{ds_1 * ds_2}$ for which a definition is provided in Cressie (1991). For a stationary, isotropic point process, λ_2 depends only on the distance between s_1 and s_2: in which case. the distance will be denoted by r and the second order intensity will be denoted by $\lambda_2(r)$ (Hamilton et al., 1995).

The K and L functions are used for a measure of crowding. The K function, K(r), a measure of relative crowding; specifically, $K(r)=\lambda$-1(E) {number of extra events within distance r of an arbitrary event}. The K function is related to the second order properties of the underlying process; specifically, for stationary, isotropic point process, $\lambda_2(r)=[\lambda^2/2\pi r]K'(r)$. Under CSR, $K(r) = \pi r^2$, and the standardized K function, defined by $L(r)\equiv[K(r)/\pi]^{1/2}$-r, solves to zero. The function L tends to be greater than zero when the true process leads to aggregation, and less than zero when the true process leads to a regular pattern (Ripley, 1977; Cressie, 1991). Under CSR, the sample estimate of K(r) is subject to Poisson variation, for which the variance stabilizing transformation is the square root. Thus the sample estimate of L(r) should have a homogenous variance, except for large r where the necessity of correcting for study region edge

effects, causes an increased variance. For these reasons, the analysis is based on L(·) instead of unstandardized K(·). To estimate K(·), the edge correction method of Ripley (1976) was used. The estimator, denoted by K, was calculated using the following equation from Cressie(1991):

$$K(h) \equiv \lambda^{-1} \sum_{i=1}^{N} \sum_{j=1}^{N} w(s_i, s_j)^{-1} I(\|s_i - s_j\| \le h) \ /N, h > 0$$

where $w(s_i, s_j)$ is the proportion of the circumference of a circle centered at s_i, passing through s_j, and that is inside the study region A. K was calculated for each r in the set R = {2,3,4,...,R}, where R is half of the narrowest dimension of B. Then K was transformed into L, an estimate of the standardized function L (Hamilton et al., 1995).

The L is plotted for all point pair distances. In order to make appropriate hypothesis tests, simultaneous tolerance envelopes around the null hypothesis (CSR) corresponding to tolerance probabilities of 0.90, 0.95, and 0.99 have been plotted (Hamilton et al., 1995). The simultaneous tolerance envelopes described in Hamilton et al. (1995) differ from the conventional methods in three ways. First, the probability level 1-α is simultaneous for all r in R, secondly, each boundary is smoothed by applying the Lowess smoother (Cleveland, 1979), and thirdly, the upper and lower envelope boundaries are calculated separately because of the asymmetry of deviations from the null L (Hamilton et al., 1995). Hamilton et al. (1995) have outlined the series of steps to create the tolerance envelopes of any specified probability level (Appendix).

All three sites were also analyzed using spatial autocorrelation in order to confirm the results of the spatial point pattern analysis method. The GS+ analysis package was used to analyze data sets for all three sites. Isotropic, or omni-directional, semivariograms were created for each site to determine if any spatial autocorrelation existed within the sites.

Results and Discussion

Regional Pattern

On the Fairfield bench, 236 fields were sampled and tested for resistance to triallate. It was determined that 63.5% of the fields contained triallate resistant seed in 1992. This large percentage of fields containing resistant seed may indicate that wild oat resistance to triallate may have been present for a long period of time, and/or resistance has quickly spread across the region. The percent of resistance for each field sample was printed on the study region map (Figure 1, see pocket in back).

Uni-directional (anisotropic) semivariogram analysis demonstrated no anisotropy. The presence of anisotropy indicates that patches are elliptical rather than circular in form. An elliptical form may signify a direction of spread of resistance. Anisotropy is measured along the major axis, at the angle of maximum variation (long axis of an ellipse), and the minor axis, perpendicular to the major axis. The range of influence along the major and minor axes were essentially identical for all the models indicating that autocorrelation has the same range of influence along all four axes (0^0, 45^0, 9^0, 135^0) (Table 4). Whatever patches that may exist will on average have a circular rather than an elliptic form.

Table 4. Comparison of models for best fit of uni-directional semivariogram. C_0 is the nugget, or y-intercept of the semivariogram and represents the measurement error, C_0+C is the sill, or asymptote of the semivariogram, the major axis is the range of influence, or the length of possible ellipses, and the minor axis is the range of influence, or length, of the short axis of the ellipse (perpendicular to the major axis).

	Parameters					
	C_0	C_0+C	Major axis range (1 unit = 0.1 km)	Minor axis range (1 unit = 0.1 km)	RSS	r^2
Linear	1551	1801	96.5	145.6	1.254×10^7	0.072
Linear/sill	958	1703	13.06	13.4	1.075×10^7	0.247
Spherical	903	1702	16.8	16.81	1.080×10^7	0.252
Exponential	279	1702	4.05	4.05	1.075×10^7	0.260
Gaussian	1043	1701	14.34	14.35	1.088×10^7	0.250

Omni-directional (without regard to direction) semivariogram analysis demonstrated that the spherical model best described the regional data with an r^2 of 0.637 (Table 5). With the spherical model:

$$\gamma(h) = C_0 + C\{1.5(h/A_0) - 0.5(h/A_0)^3 \text{ , for } h \leq A_0$$

$\gamma(h)$ represents the semivariance at some point pair distance h, C_0 represents the nugget effect (y intercept on the semivariogram), or the amount of measurement error, C_0+C represents the sill (or asymptote on the semivariogram), and A_0 represents the range or distance up to which point pairs are autocorrelated (Table 5). When autocorrelation is present, point pairs near each other are dependent or similar. The autocorrelation occurs up to a distance or range after which the correlation asymptotes represented by the sill, where point pair variation has stabilized and points at greater distances have became as dissimilar as can occur for the data set. The best fit omni-directional semivariogram for the region indicated that, point pairs were autocorrelated up to a range of 14.7 units (1 unit = 0.1 km), at which point the model indicates a constant variance or asymptote (Figure 15). This may be interpreted as a 1.47 km zone of influence, where fields within 1.47 km of each other are likely to have similar proportions of resistance. Spatial autocorrelation ceases beyond 1.47 km. The kriged contour map of the area depicts what may be considered resistant and susceptible patches of fields (Figure 14). An initial explanation may be that pattern of resistance up to 1.47 km may be the result of the clumping of land ownership \ management and that a subset of growers on the Fairfield bench accounted for the majority of resistant fields. However, it was determined that all owners who owned more than one field that was sampled, had at least one resistant and one susceptible field. An alternative explanation could be that the slight patchiness of resistant fields may be attributed to the slow expansion of resistance from initial sources by pollen and seed dispersion.

Table 5. Omni-directional (Isotropic) best fit analysis. C_0 represents the nugget effect (y-intercept on the semivariogram), or the amount of measurement error, C_0+C represents the sill (or asymptote on the semivariogram), and A_0 represents the range of influence (or distance up to which point pairs are autocorrelated) in map units (1 unit = 0.1km).

	Parameters				
	C_o	C_o+C	A_o	RSS	r^2
Linear	1540	>1789	>111.5	1.128×10^6	0.188
Linear/sill	844	1702	12.8	4.909×10^5	0.647
Spherical	705	1701	14.7	5.043×10^5	0.637
Exponential	163	1702	4.1	5.159×10^5	0.629
Gaussian	884	1701	12.7	5.200×10^5	0.626

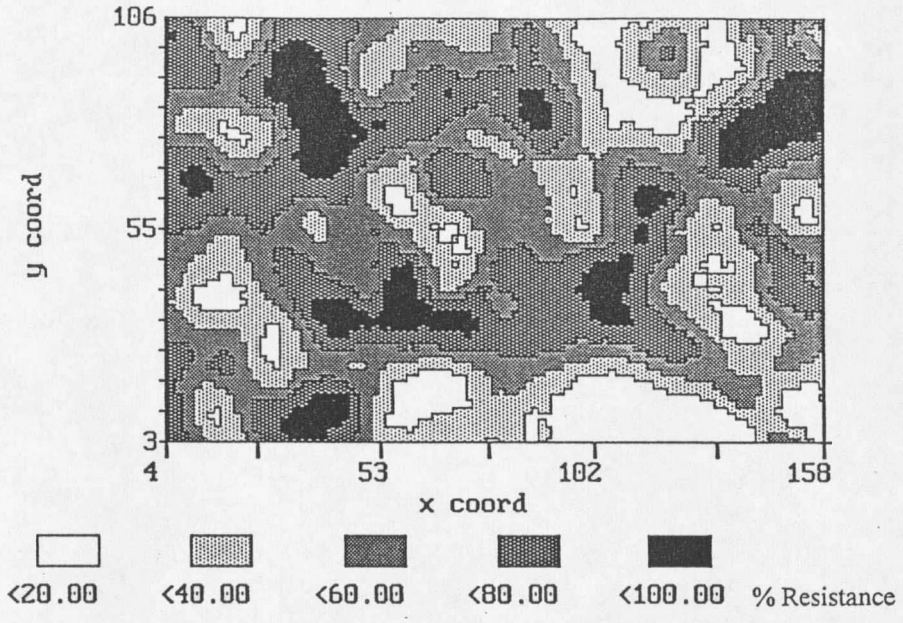

Figure 14. Kriged map of region.

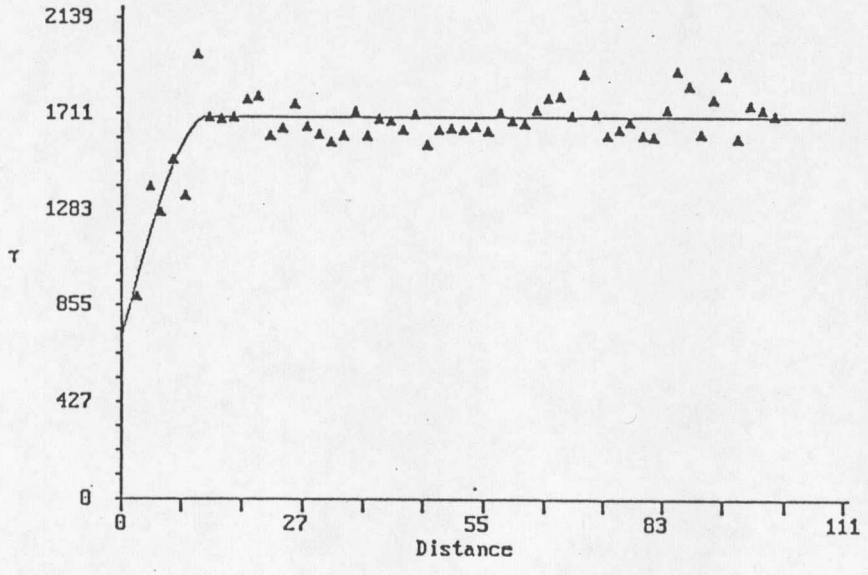

Figure 15. Omni-directional (Isotropic) semivariogram of regional resistance data. Lambda represents semivariance and distance is in map units (1 unit = 0.1 km).

The kriged map also shows that resistance has been either slower to develop in, or migrate to, the southeast portion of the sampling region which is predominately dryland crop /fallow winter wheat (Figure 14). The reduced extent of triallate resistance in the southeast region of the study area may be due to the reduced number of continuous years that triallate would be used in a crop/fallow rotation, or the effect of tillage, and/or the lack of avenues for physical migration of resistant wild oat seed.

Within Field Pattern of Resistance

The screening of seed from individual wild oat plants allowed for the opportunity to observe what variation in response to triallate might occur in seed produced on a single plant. Most samples contained either all resistant or all susceptible seed with variation in treated plant size no different than that found in the untreated seedlings. However, triallate response was highly variable. Some plants produced both susceptible and resistant seed. In these cases, a portion of the sample would stop growth immediately after triallate was applied but the remaining seed from the sample would continue to grow. Nineteen treated samples (6% of the total sampled plants) had variation in treated plant fresh weight higher than in the untreated (Figure 16). The presence of resistant and susceptible seed on the same plant could be the result of a heterozygous resistant mother plant or an outcrossing event, assuming resistance is the result of a single dominant trait. Recent work in Canada with domestic oats (*Avena sativa*) and wild oats suggested that wild oats have an outcrossing rate of up to 12% (Murray, 1994). Outcrossing in wild oats, historically thought to be insignificant, may play a significant role in the mechanism for spreading herbicide resistance.

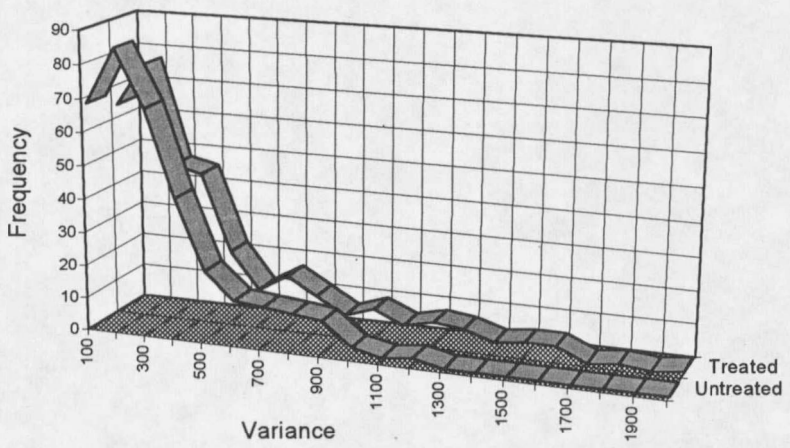

Figure 16. The distribution of variances for the treated and untreated portions of all the samples from the three systematic grid sampling sites.

The frequency of resistant for the grid in the untreated area (Site 1) was 35% , while 65% were susceptible (Figure 17). Sixty-four percent of the cells within the triallate treated area (Site 2) were resistant, while 36% of the cells were determined to be susceptible (Figure 17). In the third sampling site, within a field that had been determined to be resistant in 1992 and was known to be treated with triallate in 1992 and 1993, 66% of the cells were resistant and 34% were susceptible (Figure 18). Both triallate treated sampling areas were comprised of 64% and 66% resistant cells. Limited time and resources allowed only two triallate treated areas to be tested, so it cannot be determined if all triallate treated areas have a 66% frequency of resistant wild oats. This effect may be the result of triallate allowing one third of the susceptibles to escape due to heterogeneous herbicide distribution in the soil profile resulting from single pass incorporation. The selection pressure of triallate treatments for resistant wild oats would be considered low with the successful reproduction of susceptible wild oats in the presence of a triallate treatment. This may partially explain the 15 years of continuous triallate use that was required to select for an observable frequency of triallate resistant wild oats. Even after many years of triallate use and the continual selection of resistant wild oat seed, susceptible seed is still present and reproducing with or

without the presence of triallate. A comparison of the treated area and untreated area of the same field demonstrates that susceptible plants that survive the triallate treatment produce a significant amount of seed, due to the susceptibility of two thirds of the plants in untreated site 1. The fact that only one third of the population was resistant in the untreated grid may be evidence that resistant types are not as fit without the presence of the herbicide, or that the susceptible seed bank is twice as large as that of the resistant types, because susceptible plants may not have been exposed to triallate in prior years due to poor herbicide incorporation. If there is a fitness difference between resistant and susceptible types without the presence of the herbicide, the population may be rapidly shifted back to susceptible plants. Therefore, maintenance of susceptible wild oat populations may be exploited in a resistance recovery program in this region.

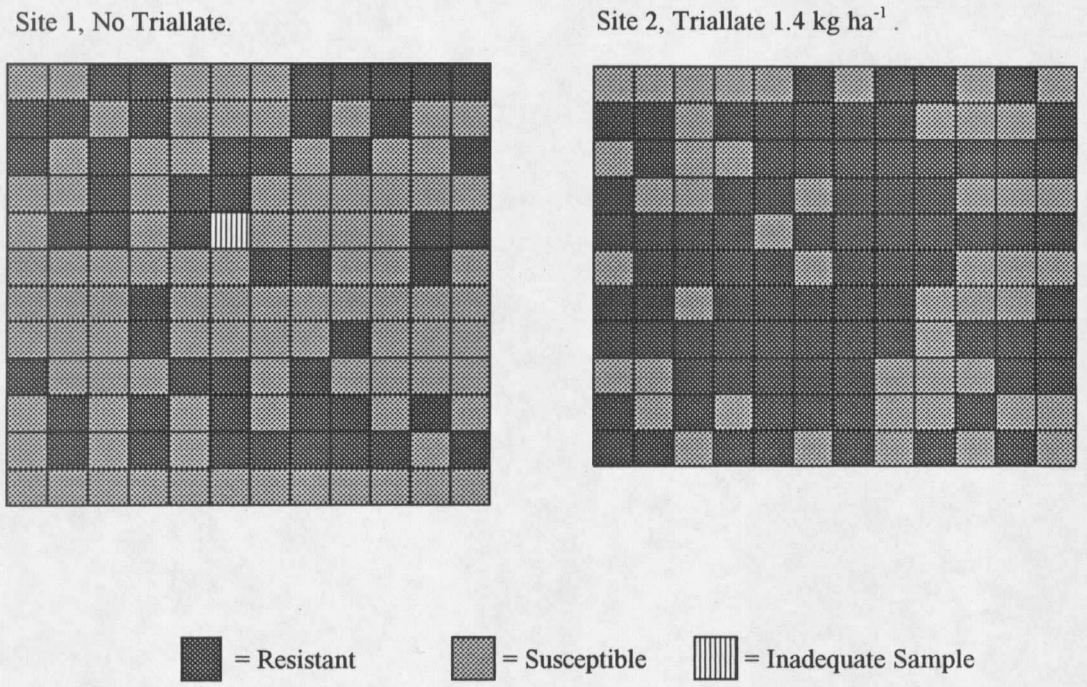

Figure 17. Map of sampling grids within the triallate treated and untreated areas of the same field.

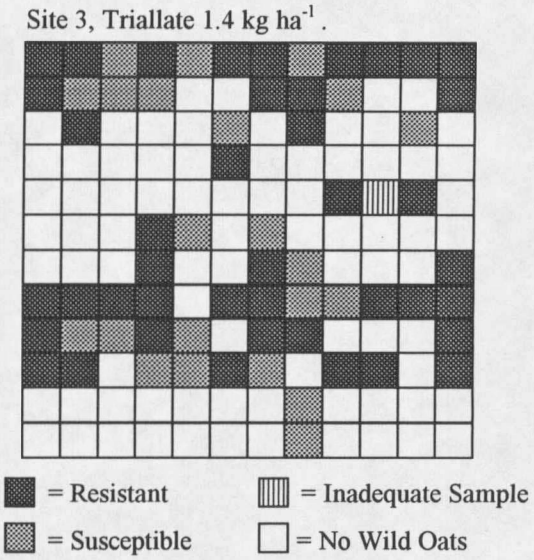

Figure 18. Map of sampling grid within a field determined to be resistant when sampled in 1992.

Pattern Analysis Within Field

The L function, an estimator of the K function, which is a measure of relative crowding, and confidence envelopes of 90%, 95%, and 99% were plotted over distance between point pairs for the resistant and susceptible cells of the three grids. The null hypothesis in this test is that events are completely spatially random (CSR), thus the L function solves to zero. An alternative to the null is when the L function takes on positive values then events are tending towards aggregation. However if the function takes on negative values then events are tending towards regularity. We believe there is little or no evidence to reject the null hypothesis of spatial randomness for the resistant and susceptible cells within the grid at site 1 (triallate untreated portion of the field) and the susceptible cells at sites 2 and 3 (triallate treated fields) (Figures 19, 20, 21, 22). Plots of the L function for the resistant cells of both sites two and three cross confidence envelopes, indicating that aggregation of resistance may exist in triallate treated field. The plot of the L function of site 2 (triallate treated area), crossed the positive (upper) 90% confidence envelopes at the distance of 7.5 m, 22.5m, and 27.5 m (Figure 23). The L function crosses the positive 90% and 95% confidence envelope at 7.5 m for site 3 which was also treated with triallate (Figure 24).

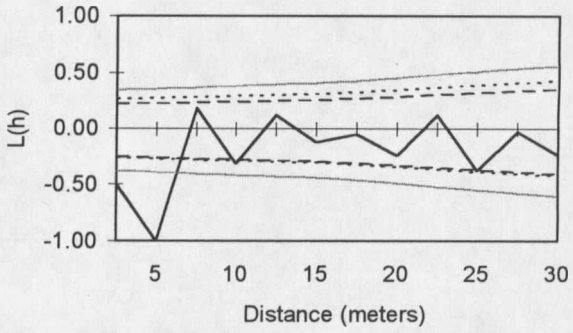

Figure 19. Plot of the L Function for site 1, no triallate, resistant cells.

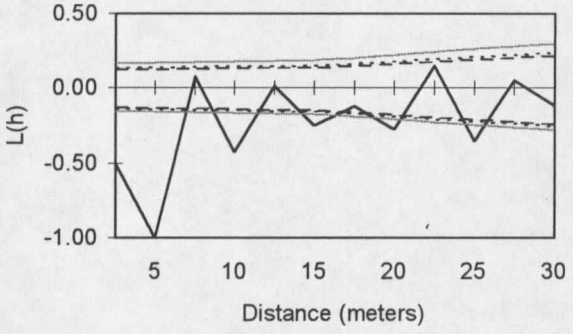

Figure 20. Plot of the L function for site 1, no triallate, susceptible cells.

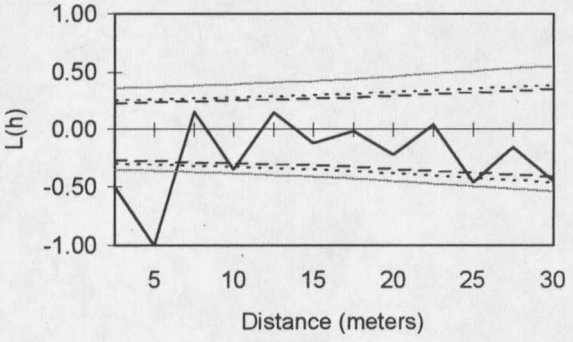

Figure 21. Plot of the L function for site 2, triallate treated, susceptible cells.

Legend

L Function

(+) and (-) 90% envelope

(+) and (-) 95% envelope

(+) and (-) 99% envelope

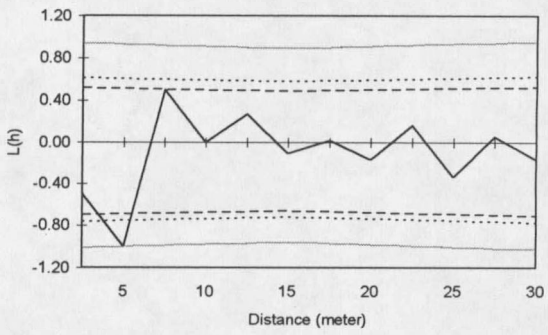

Figure 22. Plot of the L function for site 3, triallate treated, susceptible cells.

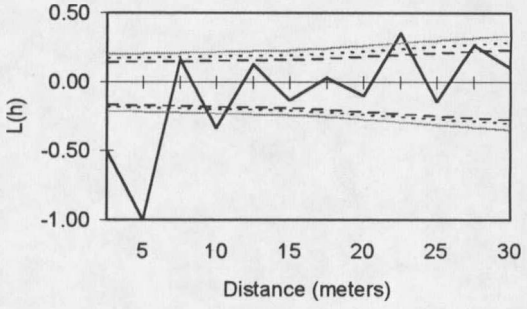

Figure 23. Plot of the L function for site 2, triallate treated, resistant cells.

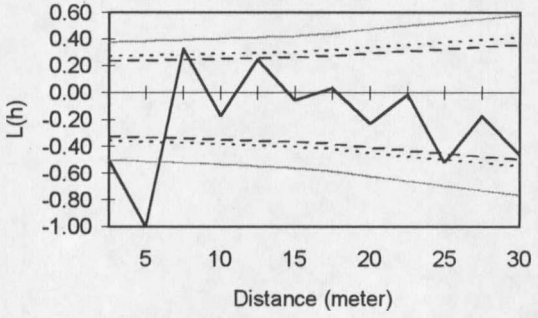

Figure 24. The plot of the L function for site 3, triallate treated, resistant cells.

Legend

L Function

(+) and (-) 90% envelope

(+) and (-) 95% envelope

(+) and (-) 99% envelope

The significant negative L value in all of the spatial point pattern analysis plots indicates regularity at 5 m intervals. These negative peaks are an artifact of the sampling method and do not necessarily indicate pattern at that scale. Spatial point pattern analysis assumes one determines all the occurrences of an event and their locations within a sampling area. In the systematic method of sampling, cells were established in a 5 m grid with the wild oat plant nearest the center of each cell being sampled. The wild oat plants were assumed to come from the center of the cells and together represent the population of the sampling area. This creates a regular pattern at the sampling scale.

The spatial point pattern analysis of sites 2 and 3 indicated that there may be aggregation of resistant wild oats at 7.5 m. This may be interpreted as resistant patches of wild oats of approximately 7.5 m in size at these sites. Because spatial point pattern analysis tests specific hypotheses, we are able to associate the patches with a particular size. Peaks in the L function for site 2 at distances 22.5 m and 27.5 m may be interpreted as the distance between aggregated patches of resistance. However, in the case of site 2, the peak in the L function at 22.5 m likely indicates a second larger scale of patch that may be observed in the map of the site 2 (Figure 24). The possibility that resistance may be aggregated or "patchy" in sites 2 and 3 indicates that resistance may have been established in the field for some time and was able to reproduce and form patches in the continuous presence of the selecting herbicide triallate. The null hypothesis (complete random spatial distribution of resistance) was not rejected for the resistant and susceptible cells in the sampling area that was not treated with triallate (site 1). This may indicate that: 1) the resistance event recently occurred throughout the sampled area and has not yet had time to spread; or 2) resistant types have been present for some time and, without the presence of the selection of triallate have been displaced due to decreased fitness compared to the susceptible and are not able to maintain an aggregated or patchy form at the scale sampled here.

Analysis of the three sites using the omni-directional semivariogram approach, as was used for the regional study, depicts no autocorrelation (Figures 25, 26, 27). However, because of the relatively small number of samples within the grids and the limited area covered by the grids, the linearity of the

semivariograms could be interpreted as complete autocorrelation or none at all. An observation of the map of site 1 (Figure 17) may lead one to interpret that there were enough susceptible cells present to be completely autocorrelated. Site 2 would appear to be a large patch of resistant cells, again completely autocorrelated. Site 3 had such a small population of wild oats that the majority of the cells were empty, thus leading one to interpret no autocorrelation. A greater area of sampling points may have allowed for a greater variance in point pairs and determination of a range of influence. Spatial point pattern analysis may be more appropriate for detecting aggregation under the constraints of limited sampling.

No spatial pattern of resistance may indicate that resistance originated randomly and simultaneously throughout the fields sampled and has either not been present long enough to form patches or has been displaced due to decreased fitness and has become unable to form cohesive patches. Patchiness of resistance indicates that resistance occurred or was introduced at a lower frequency and has been present within the field for sufficient time to form cohesive patches in the presence of triallate. The conclusion that there is no pattern or aggregation to resistance in an untreated field may indicate that resistant wild oats are less fit than susceptible plants without the presence of triallate. Management strategies that remove triallate from the Fairfield agricultural system and replace it with a crop rotation and/or a wild oat herbicide with a different site of action or metabolizing mechanism may be able to capitalize on fitness differences, encouraging the displacement of triallate resistant wild oats with susceptibles in the population so farmers can return to triallate.

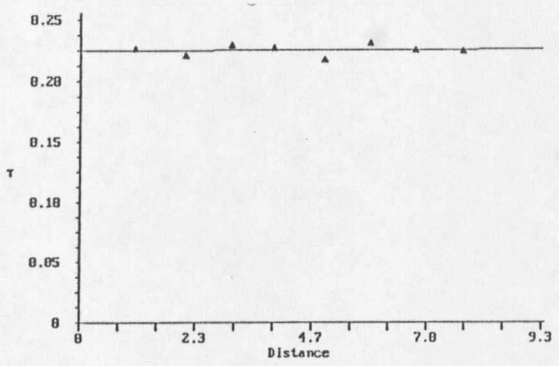

Figure 25. Omni-directional (Isotropic) semivariogram for site 1 sampling grid. Lambda represents semivariance and distance is in grid units (1 unit = 5 m).

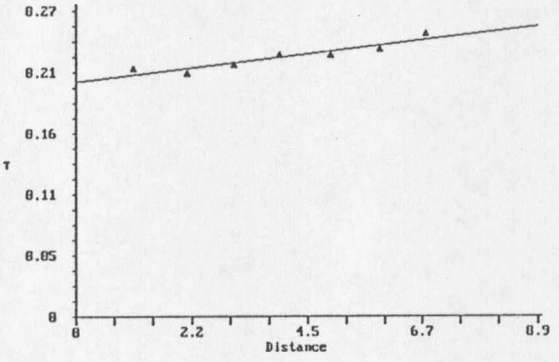

Figure 26. Omni-directional (Isotropic) semivariogram for site 2 sampling grid. Lambda represents semivariance and distance is in grid units (1 unit = 5 m).

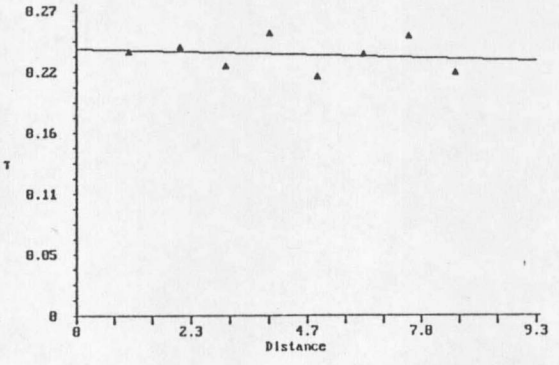

Figure 27. Omni-directional (Isotropic) semivariogram for site 3 sampling grid. Lambda represents semivariance and distance is in grid units (1 unit = 5 m).

CHAPTER 4

POSSIBLE MECHANISMS OF THE SPREAD OF TRIALLATE RESISTANT WILD OAT SEED ON THE FAIRFIELD BENCH

Introduction

The spread of weed seed has been studied as it may pertain to dispersing weed populations. McCanny and Cavers (1988) determined that approximately 2% of the seeds of proso millet remaining on parent plants at the time of harvest were carried more than 50 m by corn harvesters and that harvesters were capable of moving proso millet seed between fields. Maxwell and Ghersa (1992) used a theoretical model to determine the relative importance of weed competition and seed dispersal in determining crop yield reductions. Simulations demonstrated that the dispersal of seed of a weed species into a field may have a greater influence on crop yield than the relative competitive ability of the weed with the crop. The difference in effect was decreased if the weed species had a uniform or a high frequency random distribution. Maxwell and Ghersa (1992) concluded that harvest machines may be important mechanisms for dispersing weed seeds harvested with the crop.

The spread of weed seeds from field to field and across the region may also have a great impact on the management of herbicide resistant weeds. Several factors can be identified that may play a role in wild oat seed dispersal on the Fairfield Bench in north central Montana. Combines move from field to field , crops are swathed early so that a large proportion of wild oat seed does not drop to the soil prior to harvest, trucks tarped and untarped haul harvested crops across the region to market, irrigation canals move water throughout the area, and refuges of wild oats are maintained in disturbed areas along irrigation canals and roads. These characteristics may allow for the introduction of resistant wild oat seed into previously susceptible fields. The persistence of wild oat populations in unmanaged areas such as irrigation canal banks and roadways may also allow for the harboring of resistant genes that later serve as an inoculum via pollen or seed to later contaminate fields. The objective of this research was to

investigate the existence of resistant wild oats along roadways and irrigation canals in a region that was known to have fields that contained wild oats resistant to triallate.

Materials and Methods

Road Sampling

The Fairfield Bench study area is a region of irrigated continuous malt barley production in north central Montana. In the summer of 1992 wild oats were sampled along 14, 1.6 km sections of gravel road representing 9% of the total road length (252 km) on the Fairfield Bench. Road sections were selected to represent an even geographic distribution across the region. Road section selection was also constrained by the requirement that wild oats be present. The location of sampled sections of road are shown on the regional map (road sites R1 to R14, Figure 1, see pocket in back). Seed was stripped from the panicle of the nearest single wild oat plant every 25 m to form a composite sample for each section of road. Further sampling was performed in the summer of 1993 in order to further investigate and confirm the existence of resistance on road sections. Two 1.6 km sections of road were resampled and one additional section of road was sampled in 1993. The first resampled road site (road site R2, Figure 1, see pocket in back) was in the northwest quadrant of the study region and was determined to be resistant in 1992 and was amongst resistant fields. The second resampled road site (road site R14, Figure 1, see pocket in back) was in the northeast quadrant of the study area, and was determined to be susceptible in 1992 even though it was among resistant fields. The third site chosen to be sampled in 1993 was not sampled in 1992, however, this site was chosen because it was a new asphalt road that served as a major traffic artery across the study region and most grain trucks would pass along it on their way from the fields to the grain elevator (road site R15, Figure 1, see pocket in back).

In 1993 a 0.25 m^2 ring was randomly placed at three locations along both shoulders of each of the three road sections to be sampled. The seed was removed from the panicles of all of the wild oat plants within the ring to create a composite sample.

Irrigation Canal Sampling

Three different pairs of irrigation canal banks were sampled and tested for wild oat resistance to triallate in 1993. Irrigation canal site 1 (IRC1, Figure 1, see pocket in back) was adjacent to a field that had been determined to be susceptible in 1992. Irrigation canal site 2 (IRC2, Figure 2, see pocket in back) was adjacent to an alfalfa field and a local highway and was down stream from a field determined to be resistant in 1992. The adjacent alfalfa field contained some wild oats which were sampled and tested for resistance. Irrigation canal site 3 (IRC3, Figure 1, see pocket in back) was adjacent to a field that was determined to be resistant in 1992 and on the other side a spring wheat field that had not previously been sampled in 1992, but was sampled in 1993.

Three sampling locations along each side of the irrigation canal banks were randomly selected. At each location a pair of 0.25 m^2 rings were randomly placed across from each other on the banks. The seed was removed from the panicles of all of the wild oat plants within the ring to create a composite sample. Due to the patchiness of wild oats along canals, two rings along site 1, four rings along site 2, and one ring along site 3 did not contain wild oats. In addition to the sampling rings a composite sample was formed by walking the banks of the canals stripping seed from the nearest single wild oat panicle every 15 m for the same distance covered for sampling with the rings.

Testing for Triallate Resistance

The response to triallate for the road and irrigation canal samples was assessed by using an aqueous solution of liquid triallate applied at 1.1 kg a.i. ha^{-1} per 2.54 cm of greenhouse soil that was added to steel flats 45 cm by 61 cm by 9 cm following placement of 6 cm of untreated greenhouse soil in the bottom of each flat. Treated soil was mixed thoroughly in a rotary mixer before adding to each flat. Seven wild oat road or irrigation samples and one known susceptible wild oat biotype were planted in rows of 15 seeds. Rows were 3 cm apart. Treated flats received 2.54 cm of the triallate incorporated soil while untreated flats received 2.54 cm of untreated greenhouse soil. Planted flats were placed in a greenhouse receiving 16 h artificial light and set for day/night temperatures of 22/16 C, respectively, in a

randomized complete block design with three replicates. Flats were watered daily to field capacity. Twenty-eight days after planting wild oat seedlings were clipped at the soil surface and weighed immediately. Seedlings where the shoot had not emerged from the coleoptile (a primary symptom of triallate) were classified as small plants, conversely seedlings where the shoot had emerged from the coleoptile were classified as large plants. This initial distinction was made in order to decrease variability within replications of samples due to the possibility that resistant and susceptible seed may both be present within a bulk sample. Seedlings of each size class from within a row within a flat were counted and weighed to determine mean fresh weight per plant for each size class. The mean of the three replicate mean fresh weights per plant was then calculated. The treated size class means were compared to each other and to the untreated mean of a sample using t-tests to determine if a sample was resistant or susceptible according to a series of decisions (Figure 2, Chap. 2). F- and t-tests were conducted in SAS.

Results and Discussion

Road Samples

Only one (7%) of the 14 road sections sampled in 1992 contained wild oats resistant to triallate (Table 3). Only one road sample (road site R2, Figure 1, see pocket in back) of the ten road samples taken from road sections adjacent to resistant fields contained resistant seed, indicating that triallate resistant wild oats are either being moved to the roadsides at a low frequency and/or the triallate resistant wild oats are being displaced due to decreased fitness compared to the susceptible type.

Road site 2 was resistant and adjacent to resistant fields in 1992. When this same section of road was resampled in 1993 it was found to contain triallate resistant seed. Road site 13 was susceptible and adjacent to resistant fields in 1992 and was found to have a low frequency of resistance in 1993. Road site 15, sampled only in 1993, was recently paved and is a major truck route to the local elevator during harvest. Road site 15 was determined to be susceptible in 1993 and was adjacent to resistant fields (Figure 1, see pocket in back).

Sixty three percent of the fields sampled in the study area were determined to contain resistant wild oat seed in 1992. Only 7% of the road sites sampled contained resistant seed in the same year and only 10% of those road samples adjacent to triallate resistant fields contained triallate resistant seed. One may expect the frequency of resistance along roads to be the same as non-triallate treated fields if roads are receiving wild oat seeds. If resistance is lower than in untreated fields then the resistant biotype may be less able to persist along roads due to decreased fitness where they must compete with perennial grasses and susceptible wild oats without the presence of triallate. The frequency of seed being moved from fields to roadsides is not known. However, once seed is present on the roadside it is possible for it to be moved about by road graders. The Teton county road maintenance program involves grading roads twice a year, during which gravel on the shoulder and in the barrow pit is pulled to the center of the road and pushed along for an unknown distance in order to smooth the road and then the remainder is deposited off one of the road shoulders to be used at a later date to smooth the road. Resistant wild oat seed deposited or produced on the road shoulder may be moved along roads where it may eventually be able to enter fields by natural dispersal and by traffic on road approaches to fields. Resistant genes may be harbored in wild oat populations existing along roadways and may serve as future contaminants of susceptible populations within fields.

Table 6. Road and irrigation canal bank sections sampled in 1992 and 1993.

	Years Sampled	Total Sections	No. R Sections	No. S Sections
Roads	1992	14	1 (R2)	13
	1993	3	2 (R2, R14)	1
Irrigation Canal Banks	1993	3	0	3

Irrigation Canals

Wild oat seed from the ring samples and composite samples from irrigation canal banks were susceptible whether they were adjacent or downstream from resistant fields (Table 3). The sample from the alfalfa field adjacent to site 2 (IRC2) was determined to contain only susceptible seed and the spring wheat field adjacent to site 3 was determined to contain resistant seed.

Triallate resistant wild oats have either not yet moved to the irrigation canal banks or are unable to persist there due to a decreased fitness compared to susceptible biotypes when not in the presence of triallate. Dispersal of wild oat seed to canal banks could only occur by natural dispersal mechanisms. In addition, irrigation canals are not subject to constant disturbance and activity as are the roadways. Due to the decreased traffic and disturbance it is possible that resistant types have not been introduced to the canal banks. Most canal banks are also dominated by perennial grass species making it difficult for wild oats to get established. The danger of resistant wild oat plants persisting along canal banks is the possibility of the introduction of resistant seed to fields downstream via water movement. Typically, however, irrigation water only flows into fields, not out, so even though resistant wild oats may exist within the field there is little chance of the seed flowing out.

At the present time triallate resistant wild oat seed appears to be moving out of fields and onto roadsides and canal banks at a low frequency, either because the seed is not moving or is not fit enough to survive once moved. Field machinery and untarped trucks moving from fields onto roads may not be depositing resistant wild oat seed along the roadways at rates initially hypothesized.

CHAPTER 5

PRELIMINARY STUDIES OF CROSS RESISTANCE IN WILD OAT

Introduction

Prior to the introduction of the post emergent wild oat herbicides, wild oat was one of the most difficult weeds to manage in small grain production in the United States (Nalewaja and Arnold, 1970). Projected annual losses of wheat and barley as a result of wild oat infestations have been estimated at 2.2 and 6.4 million tons in Europe and North America, respectively (Nalewaja, 1977). In recent years many wild oat populations have been documented to be resistant to one or more wild oat herbicides, particularly the post-emergence applied herbicides.

Heap et al. (1994) determined the levels of cross-resistance in three wild oat populations. In the fall of 1990 complaints were received about the lack of wild oat control provided by diclofop-methyl, and a newly recommended aryloxyphenoxy propionate herbicide, fenoxaprop-ethyl in Manitoba. Field histories indicated that these wild oat populations had received repeated applications of diclofop-methyl and sethoxydim, a cyclohexanedione, over the previous 10 years. The three populations were resistant to diclofop-methyl, fenoxaprop-p-ethyl, and sethoxydim, all of which are inhibitors of acetyl-coenzyme A carboxylase. In contrast a fourth population was also screened and found to be resistant to the aryloxyphenoxy propionate herbicides but was not resistant to sethoxydim. It was concluded that because of the contrast in cross-resistance patterns and the distance between these sites that resistance evolved independently. It would follow that slightly different mutations in the acetyl CoA carboxylase enzyme may have been able to confer varying degrees of resistance to the ACCase inhibitors.

Diclofop resistant wild oats were discovered in the Pacific Northwest in the Willamette Valley of Oregon in 1990 (Seefeldt et al., 1990). Seefeldt (1990) reported two populations of wild oat that were resistant to the herbicides diclofop methyl and fenoxaprop that was mixed in a single formulation with 2,4-D and MCPA , and an additional population that was resistant to diclofop methyl only. Eight more

fields were later confirmed to be resistant to diclofop (Seefeldt et al., 1992). In 1994 Seefeldt et al. (1994) further investigated cross resistance of eight diclofop resistant wild oat biotypes. All biotypes were significantly resistant to diclofop as compared to the susceptible. Only four wild oat biotypes were resistant to fenoxaprop. Seefeldt et al. (1994) suggests that the variable response to these herbicides may be due to mutations of the gene coding for ACCase (Seefeldt et al., 1994). The diclofop methyl resistant oats were generally resistant to other aryloxyphenoxy propionate herbicides, however only one biotype was resistant to all the ACCase inhibitors. Seefeldt et al. (1994) goes on to hypothesize that because wild oat is a hexaploid, mutations of different isoforms of ACCase might occur. Multiple alleles for ACCase, combined with multiple mutations at the site of action and more than one mechanism of resistance, could explain the amount of variation in cross-resistance.

Resistance in wild oat has not been confined to the aryloxyphenoxy propionate and cyclohaxanedione herbicides. In 1990, O'Donovan et al. (1994) investigated complaints of poor triallate performance in Alberta. Triallate belongs to a group of herbicides called thiocarbamates. Fifteen of 34 samples were highly resistant to triallate used at a rate of 1.7 kg ha^{-1} (equivalent to the recommended field rate). It was later discovered that all the triallate resistant populations were also resistant to difenzoquat, a pyrazolium herbicide structurally dissimilar to triallate, at a rate of 1.7 kg ha^{-1} (twice the recommended field rate) (O'Donovan et al. 1994). Triallate at 3.4 kg ha^{-1} had little or no effect on the resistant populations. Rates of 6.8 kg ha^{-1} triallate created differences among and within populations. Early difenzoquat symptoms included chlorosis and necrosis seven days after application, however triallate resistant populations resumed growth by 21 days while triallate susceptible populations ceased growth at seven days after treatment. Field histories indicated that all populations resistant to triallate and difenzoquat had histories of continuous cropping, mainly barley, and continuous triallate use for approximately 15 years or longer. The use history of difenzoquat was not mentioned. O'Donovan et al. (1994) suggested that because difenzoquat is rarely used for wild oat control in the fields where resistance developed that continuous triallate use selected for resistance to difenzoquat and triallate.

Recent research suggests that triallate and other thiocarbamates are metabolized to more active compounds which inhibit very long-chain fatty acid synthesis in pea (*Pisum sativum* L.) (Abulnaja and Harwood, 1991). O'Donovan et al. (1994) suggests that differential triallate metabolism could play a role in the triallate resistance mechanism in wild oat. Susceptible wild oats were found to have more ^{14}C-triallate metabolites than their triallate resistant counterparts from the Fairfield Bench (Colliver et al., 1994).

The objective of this work was to determine if triallate resistant wild oats from the Fairfield Bench were cross-resistant to any other wild oat herbicides.

Materials and Methods

1992 Field Study

Sixty-seven wild oat field samples were collected from complaint fields by Monsanto personnel in 1991. In 1992, 23 of the 67 field samples were randomly selected and seeded into single rows one meter apart at the Post Research Farm near Bozeman, MT. Seeds were pierced on the dorsal side using a sterilized needle to break dormancy before planting. Six wild oat herbicide treatments and an untreated control were applied across one meter portions of the rows of wild oats. Treatments were as follows: triallate preplant incorporated at 1.4 kg a.i. ha^{-1}, trifluralin preplant incorporated at 0.45 kg a.i. ha^{-1}, triallate and trifluralin preplant incorporated at 1.4 and 0.45 kg a.i. ha^{-1} respectively, diclofop-methyl applied post-emergence (3-leaf) at 1.1 kg a.i. ha^{-1} plus crop oil, imazamethabenz applied post-emergence (3-leaf) at 0.5 kg a.i. ha^{-1} plus surfactant, and difenzoquat applied post-emergence (3-leaf) at 1.1 kg a.i. ha^{-1}. Treatments were applied using a CO^2 backpack sprayer applying 178 L of water per ha. The experiment was a randomized complete block design with three replicates.

1994 Greenhouse Study

Eight fields sampled in 1992 were resampled in 1993 to investigate possible cross resistance of triallate and diclofop-methyl and imazamethabenz. Each field was sampled by a collector walking across the field in a "W" pattern, avoiding collection near field edges and in obvious herbicide skips. A collector removed wild oat seed from a single wild oat panicle every 12 m. A sub-sample of 200 seeds was removed from a bulk composite sample representing the field. The sub-samples were imbibed in tap water for 24 h, pierced on the dorsal side to stimulate germination and planted in flats.

Treatments were triallate (1.26 kg a.i. ha^{-1}), imazamethabenz (0.52 kg a.i. ha^{-1}), diclofop-methyl (1.1 kg a.i. ha^{-1}), triallate (1.26 kg a.i. ha^{-1}) plus imazamethabenz (0.52 kg a.i. ha^{-1}), triallate (1.26 kg a.i. ha^{-1}) plus diclofop-methyl (1.1 kg a.i. ha^{-1}), diclofop-methyl (1.1 kg a.i. ha^{-1}) plus imazamethabenz (0.52 kg a.i. ha^{-1}), triallate (1.26 kg a.i. ha^{-1}) plus diclofop-methyl (1.1 kg a.i. ha^{-1}) plus imazamethabenz (0.52 kg a.i. ha^{-1}), and an untreated control. Triallate was preplant incorporated while diclofop-methyl and imazamethabenz were sprayed postemergence at the three leaf stage. Crop oil and surfactant were applied with diclofop-methyl and imazamethabenz, respectively. An aqueous solution of liquid triallate was applied at 1.26 kg ai ha^{-1} per 2.54 cm of greenhouse soil to be added to steel flats 45 cm by 61 cm by 9 cm deep filled with 6 cm of untreated greenhouse soil. The eight wild oat field samples and one known susceptible wild oat biotype were planted along the surface in rows of 15 seeds. Rows were 2.5 cm apart. Treated soil was mixed thoroughly in a rotary mixer. Triallate treated flats received 3 cm of the triallate incorporated soil while untreated flats and treatments not involving triallate received 3 cm of untreated greenhouse soil.

The experiment was a randomized complete block design with three replicates. Flats were placed in a greenhouse receiving 16 h artificial light and set for day/night temperatures of 22/16 C. Flats were watered daily to field capacity. Twenty-one days after application of the postemergent herbicides wild oat seedlings were counted in each row and clipped at the soil surface. Mean fresh weight for each row of plants was determined. The mean of the three replicate mean fresh weights per plant was calculated. The

treated means were compared to the untreated mean of each sample using t-tests to determine if a sample was resistant or susceptible (using a 0.05 level of significance). F- and t-tests were conducted in SAS.

Results and Discussion

1992 Field Study

Samples with less than 30% visual injury were considered resistant. Trifluralin, triallate and trifluralin, diclofop-methyl, and imazamethabenz all provided excellent control of all wild oat field samples collected in 1991, indicating that resistance to triallate in wild oat does not initially confer resistance to trifluralin, diclofop-methyl, and imazamethabenz . Triallate alone and difenzoquat provided erratic control (Figure 28). Thirteen samples (57%) were resistant to triallate and 10 (43%) were cross resistant to difenzoquat. Five samples (22%) were resistant to difenzoquat that were susceptible to triallate. Therefore, some samples that are resistant to triallate are also resistant to structurally dissimilar difenzoquat as was the case with the Alberta samples (O'Donovan et al., 1994). Difenzoquat has been used very little in the study region (Fairfield Bench) because it can injure spring barley. Resistance to both herbicides in this case may be classified as cross or multiple resistance due to the limited amount of knowledge of the mechanisms of these herbicides. Holt (1993) defines cross resistance as, following exposure to a herbicide, a weed population evolved resistance to herbicides from chemical classes to which it has never been exposed. Holt (1993) goes on to suggest that the most common form of cross resistance occurs when different herbicide chemistries act on the same target site. Wild oats collected and tested in Canada were resistant to triallate and difenzoquat (O'Donovan et al., 1994). The area where resistance had developed in Canada had a long history of continuous barley with triallate use. In Alberta, difenzoquat was rarely used for wild oat control in the fields where resistance had developed, suggesting that continuous triallate use selected for resistance to difenzoquat as well as to triallate (O'Donovan et al., 1994).

Resistance to difenzoquat, but not triallate, when selection has been under triallate is very unusual. Malt barley raised for seed in other areas such as Idaho and Wyoming where difenzoquat is used more readily may have been the site where selection for difenzoquat resistance occurred and then migrated to the Fairfield area as barley seed contaminant. Another hypothesis may be that resistance to both herbicides was selected through the continuous use of triallate. If resistance to triallate is the result of a common mechanism of herbicide degradation associated with the accumulation of resistant alleles, and difenzoquat requires fewer alleles than triallate for degradation, then plants would be difenzoquat resistant without being triallate resistant. The mechanism of triallate resistance is not known, however, there is evidence that thiocarbamate metabolites inhibit very long-chain fatty acid synthesis in pea (Abulnaja and Harwood, 1991). It is also possible that many different types of resistance to triallate and even difenzoquat have developed and we are observing the results of a mosaic of resistance mechanisms.

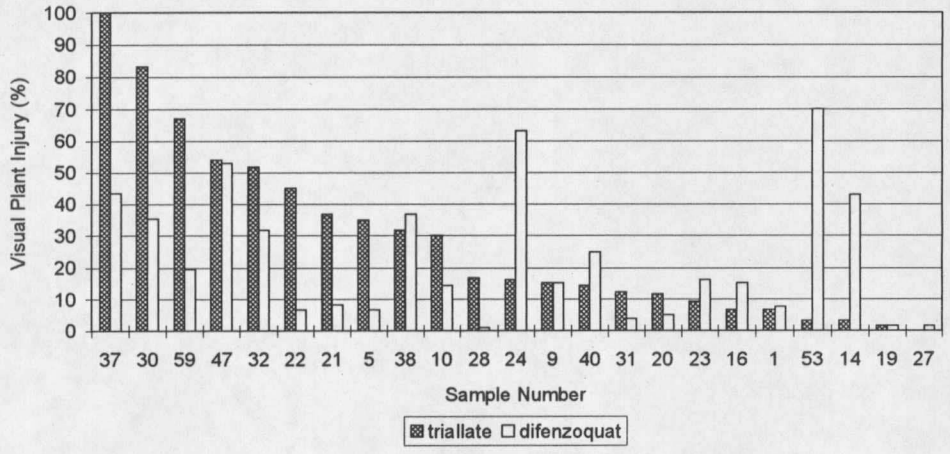

Figure 28. Visual plant injury in percent for the 23 field samples collected in 1991.

1994 Greenhouse Study

The greenhouse study compared triallate, diclofop-methyl, and imazamethabenz alone and in combinations. Samples were not resistant to imazamethabenz or any herbicidal combination containing imazamethabenz. Five of the eight samples were resistant to triallate but none of the other herbicides or combinations (Figures 29-33). Two of the samples showed no resistance to any of the herbicides or

combinations even though one of them, field number 054, had been tested to be resistant to triallate in 1992 (Figure 34, 35). Triallate was applied at 1.26 kg a.i. ha^{-1} in this study rather than 1.1 kg a.i. ha^{-1} as in the field study. The increased triallate rate may have decreased the mean fresh weight per plant sufficiently to cause previously resistant plants to be susceptible at the 1.26 kg a.i. ha^{-1} rate. One sample (Figure 36) was resistant to triallate, diclofop-methyl, and triallate plus diclofop-methyl. The grower managing this field had used a commercial combination herbicide containing fenoxaprop-ethyl and MCPA prior to 1992 for several years, and prior to that, continuous triallate. Wild oats resistant to diclofop methyl in Washington were also resistant to fenoxaprop (Seefeldt et al., 1994). Diclofop methyl and fenoxaprop both belong to the aryloxyphenoxy propionate (AOPP) herbicide family which inhibit acetyl CoA carboxylase (ACCase). Triallate resistance may have been selected during the continuous triallate use period. Wild oat resistance to diclofop-methyl and triallate would indicate multiple resistance which is defined by Holt (1993) as resistance to herbicides from more than one chemical class with different mechanisms of action.

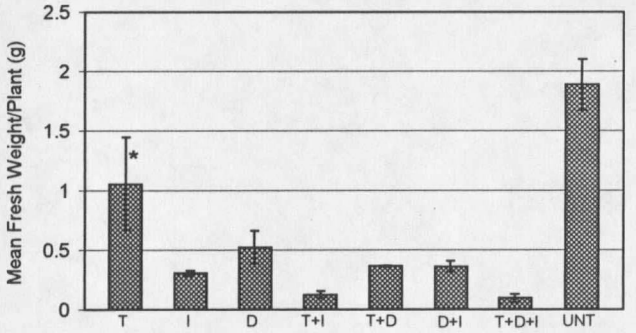

Figure 29. Field 93121, field no. 121, collected in 1993. T = triallate, I = imazamethabenz, D = diclofop-methyl. * Not significantly different than the untreated. Bars are the standard error of the mean.

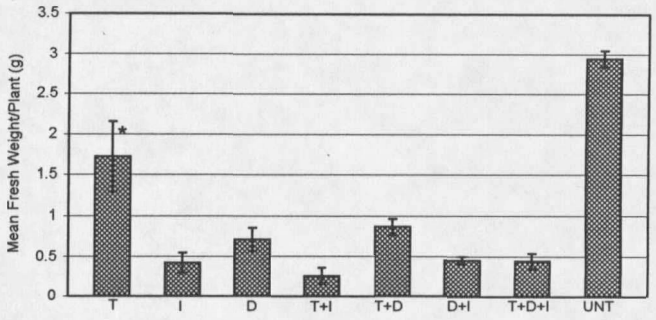

Figure 30. Field 93464, field no. 464, collected in 1993. T = triallate, I = imazamethabenz, D = diclofop-methyl. * Not significantly different than the untreated. Bars are the standard error of the mean.

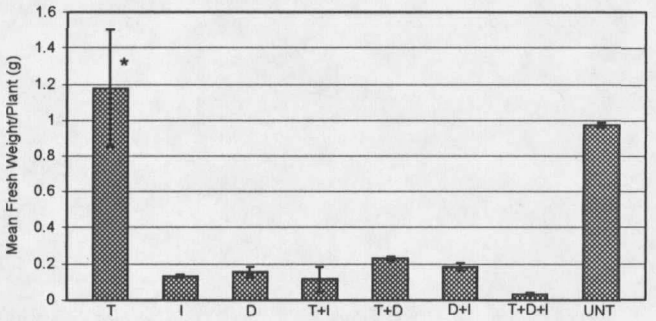

Figure 31. Field 93484, field no. 484, collected in 1993. T = triallate, I = imazamethabenz, D = diclofop-methyl. * Not significantly different than the untreated. Bars are the standard error of the mean.

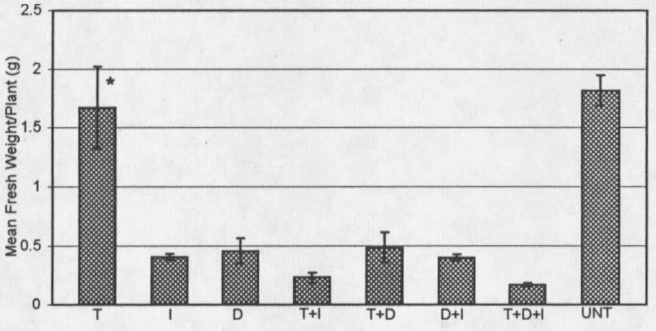

Figure 32. Field 93593, field no. 593, collected in 1993. T = triallate, I = imazamethabenz, D = diclofop-methyl. * Not significantly different than the untreated. Bars are the standard error of the mean.

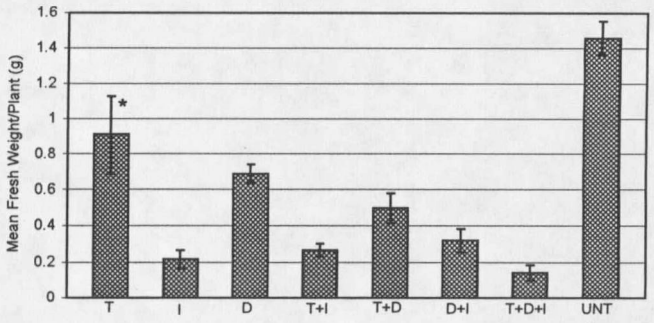

Figure 33. Field 93602, field no. 602, collected in 1993. T = triallate, I = imazamethabenz, D = diclofop-methyl. * Not significantly different than the untreated. Bars are the standard error of the mean.

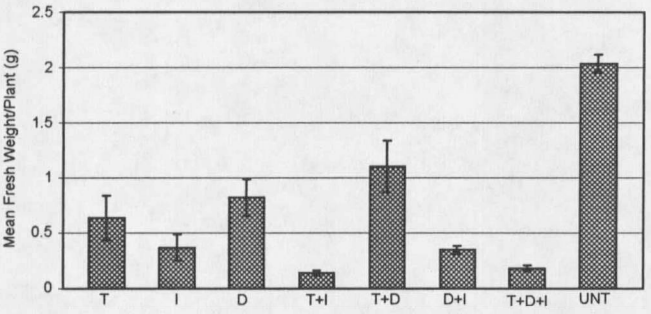

Figure 34. Field 93054, field no. 054, collected in 1993. T = triallate, I = imazamethabenz, D = diclofop-methyl. * Not significantly different than the untreated. Bars are the standard error of the mean.

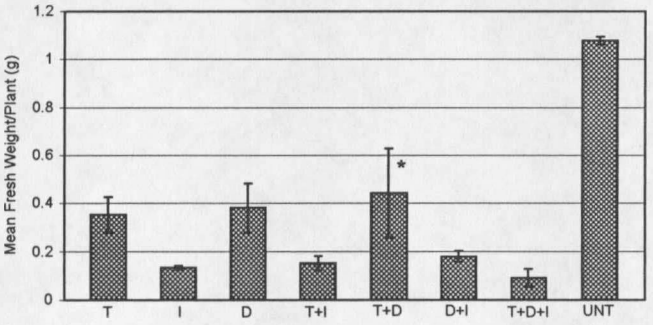

Figure 35. Field 93321, field no. 321, collected in 1993. T = triallate, I = imazamethabenz, D = diclofop-methyl. * Not significantly different than the untreated. Bars are the standard error of the mean.

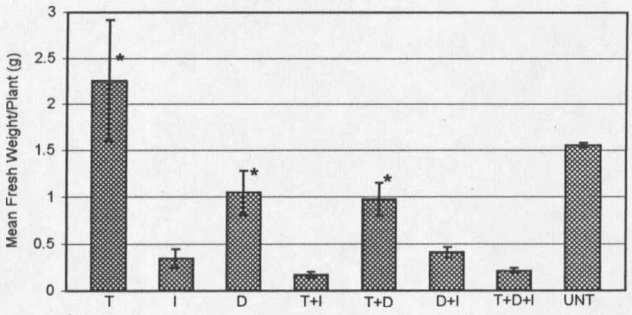

Figure 36. Field 93071, field no. 071, collected in 1993. T = triallate, I = imazamethabenz, D = diclofop-methyl. * Not significantly different than the untreated. Bars are the standard error of the mean.

Growers must continually rotate herbicides in order to slow the onset of resistance. The danger in just moving from one continuous single herbicide use program to another continuous program with a different herbicide will be the development of weed biotypes that exhibit cross- or multiple resistance. It is evident that most growers switched to imazamethabenz in 1993 after the disclosure of triallate resistance (Table 7). Imazamethabenz will need to be rotated and managed carefully due to the fact that it is resistance prone because imazamethabenz, a member of the imidazolinones, has a very specific site of action. The mechanism of action of imazamethabenz is the inhibition of acetolactate synthase (ALS), which is a key enzyme in the synthesis of isoleucine, leucine, and valine. However, plant death may result from the disruption of photosynthate translocation and overall hormone imbalance rather than an insufficient supply of amino acids. Kochia (*Kochia scoparia*) resistance to chlorsulfuron, another ALS inhibitor belonging to the sulfonylurea family, was detected in a Kansas wheat field that had been treated with chlorsulfuron for five consecutive years (Primiani et al., 1990). Imazamethabenz, like chlorsulfuron, may develop resistance after a limited number of uses.

Table 7. History and resistance rating of fields .

Field Number	92 Result	'92 wild oat herbicide	'93 wild oat herbicide
071	R	triallate	imazamethabenz
054	R	triallate/imazamethabenz	imazamethabenz
121	R	triallate	imazamethabenz
321	S	triallate	imazamethabenz
464	R	triallate	imazamethabenz
484	R	triallate	imazamethabenz
593	R	triallate	triallate
602	R	triallate/imazamethabenz	triallate/imazamethabenz

The Fairfield Bench malt barley production area now contains wild oats that are resistant to triallate, difenzoquat, and diclofop-methyl. Imazamethabenz is one of the only remaining chemical wild oat control tools that remains effective in some small grain fields on the Fairfield Bench. Careful management involving crop rotations and cultivation will be necessary to prevent the loss of imazamethabenz to resistance and aid in controlling resistant wild oat populations. Fields that have not yet developed resistance to triallate, difenzoquat, diclofop methyl, or imazamethabenz should carefully rotate the remaining herbicide options and crops as to prolong or prevent the development of resistance to the remaining wild oat herbicides.

APPENDIX

TOLERANCE ENVELOPES

Steps in the simulation technique for placing a 95% tolerance envelope around the L function under the null hypothesis of complete spatial randomness (CSR).[5]

1. Observe N events in region B.

2. Calculate $\hat{K}(r)$, r R, which is the observed K function, and the standardized $\hat{K}$ function,
$$L(r) = [\hat{K}(r) / \pi]^{1/2} - r.$$

3. Start computer simulation; set i = 1.

4. Simulate n cell location events from a uniform distribution on B.

5. Calculate $\hat{K}(r)$ for the simulated events; denote the result by $K_i(r)$.

6. If I = 200, go to 7; otherwise, set i = i+1, then got to 4.

7. Find $L_i(r) = [K_i(r) / \pi]^{1/2} - r$, i = 1,...,200.

8. $V_L(r) = Var\{L_i(r), i = 1,...,200\}$.

9. Using Lowess with span = optimum window width, smooth $V_L(r)$. Denote the smooth by $V_{LS}(r)$.

10. $Max_i = max\{ L_i(r) / [V_{LS}(r)]^{1/2}, r \varepsilon R \}$.

11. UPPER = 97.5%-tile of $\{Max_1, ..., Max_{200}\}$.

12. $Min_i = min\{ L_i(r) / [V_{LS}(r)]^{1/2}, r \varepsilon R \}$.

13. Lower = 2.5%-tile of $\{ Min_1, ..., Min_{200}\}$.

14. Upper Boundary (r) = $[V_{LS}(r)]^{1/2}$ * UPPER.

15. Lower Boundary (r) = $[V_{LS}(r)]^{1/2}$ * LOWER.

16. L(r) departs significantly from CSR if it crosses a boundary at any r ε R. (0.05 level of significance).

[5] Hamilton et al., 1995, in press.

Literature Cited

Abulnaja, K.O. and J.L. Harwood. 1991. Thiocarbamate herbicides inhibit fatty acid elongation in a variety of monocotyledons. Phytochemistry 30:1445-1447.

Adkins, S.W., M. Loewen, and S.J. Symons. 1986. Variation within pure lines of wild oats (*Avena fatua*) in relation to degree of primary dormancy. Weed Sci. 34:859-864.

Banting, J.D. 1970. Effect of diallate and triallate on wild oat and wheat cells. Weed Sci. 18:80-84.
Bell, A.R. and J.D. Nalewaja. 1968. Competition of wild oat in wheat and barley. Weed Sci. 16:505-509.

Bowden, B.A. and G. Friesen. 1967. Competition of wild oats (*Avena fatua* L.) in wheat and flax. Weed Res. 7:349-359.

Bowler, D.J. 1973. Economic benefits from controlling wild oat. Span 16:79-81.

Brian, P. and R. Cousens. 1990. The effect of weed distribution on prediction of yield loss. J. Appl. Ecol. 27:735-742.

Caprio, J.M. and G.A. Nielsen. 1992. Climate atlas of Montana. Extension Bulleti 113.

Cardina, J., D.H. Sparrow, and E.L. McCoy. (In Press). Analysis of spatial distribution of common lambsquarters (*Chenopodium album*) in no-till soybean (*Glycine max*).

Carlson, H.L. and J.E. Hill. 1985. Wild oat (*Avena fatua*) competition with spring wheat: plant density effects. Weed Sci. 33:176-181.

Chancellor, R.J. and N.C.B. Peters. 1974. The time of the onset of competition between wild oats (*Avena fatua* L.) and spring cereals. Weed Res. 14:197-202.

Charlesworth, B. 1992. Evolutionary rates in partially self-fertilizing species. Am. Nat. 140:126-148.

Chellemi, D.O., K.G. Rohrbach, R.S. Yost, and R.M. Sonoda. 1988. Analysis of the spatial pattern of plant pathogens and diseased plants using geostatistics. Phytopathol. 78:221-226.

Clark, C.O., D.H. Farnsworth, F.T. Miller, and B.N. Weight. 1982. Soil survey of Cascade County, Montana. National Cooperative Soil Survey, U.S.D.A., S.C.S., and Mont. Ag. Expt. Sta.

Cleveland, W.S. 1979. Robust locally weighted regression and smoothing scatterplots. J. American Statistical Assoc. 74:829-836.

Cohen, W.B., and T.A Spies. 1990. Semivariograms of digital imagery for analysis of conifer canopy structure. Remote Sens. Environ. 34:1267-178.

Colliver, C.T., A.J. Kern, W.E Dyer, and B.D. Maxwell. 1994. Mechanism of triallate resistance in wild oats (*Avena fatua* L.): Preliminary studies. West. Soc. Weed Sci. Proc. 47:103.

Cousens, R., P. Brain, J.T. O'Donovan and P.A. O'Sullivan. 1987. The use of biologically realistic equations to describe the effects of weed density and relative time of emergence on crop yield. Weed Sci. 35:720-725.

Cressie, N.A.C. 1991. Statistics for spatial data. New York: Wiley.

Croft, B.A. Keeping ahead of the wolf: pest resistance to agricultural pesticides. In: USDA Yearbook of Agriculture. Research for Tomorrow, edited by U.S. Dep. Agric., Washington, D.C.: U.S. Dep. Agric., 1986, p. 168-174.

Dadd, C.V. 1957. Wild oats. Field Crop Abstr. 10:1-10.

Darmency, H. and C. Aujas. 1986. Polymorphism for vernalization requirement in a population of *Avena fatua*. Can. J. Bot. 64:730-733.

Darmency, H. and C. Aujas. 1987. Character inheritance and polymorphism in a wild oat (*Avena fatua*) population. Can. J. Bot. 65:2352-2356.

Dew, D.A. 1972. An index of competition for estimating crop loss due to weeds. Can. J. Plant Sci. 52:921-927.

Dew, D.A. and C.H Keys. 1976. An index of competition for estimating loss of rape due to wild oats (*Avena fatua*). Can. J. Plant Sci. 56:1005-1006.

Donald, W.W. 1994. Geostatistics for mapping shoot and root growth across a Canada thistle (*Cirsium arvense*) patch. Weed Ssci. Soc. Am. Proc. 34:72.

Dunan, C.M. and R.L. Zimdahl. 1991. Competitive ability of wild oats (*Avena fatua*) and barley (Hordeum vulgare). Weed Sci. 39:558-563.

Dunan, C.M., F.D. Moore, and P. Westra. (In Press). A plant process-economic model for wild oats management decisions in irrigated barely. Agricultural Systems.

Dyer, W.E, P.W. Chee, and P.K. Fay. 1992. Low temperature seed germination characteristics of sulfonylurea-resistant Kochia scoparia L. accessions. Proc. West. Soc. Weed Sci. 117-118.

Eckert, J.W. Historical development of fungicide resistance in plant pathogens. In: Fungicide Resistance in North America, edited by C.J.Delp., St. Paul: APS Press, 1988, p. 1-3.

Evans, R.M., D.C. Thill, L. Tapia, B. Shafii, and J.M. Lish. 1991. Wild oat (*Avena fatua*) and spring barley (*Hordeum vulgare*) density affect spring barley grain yield. Weed Technol. 5:33-39.

Fay, P.K. and V.R. Stewart. 1981.The number one weed problem in Montana: controlling wild oats *Avena fatua*. Capsule Inf. Ser. Mont. Agric. Exp. Stn. 3:ill.

Fay, P.K., and G. Rohde. 1980. Weed control in small grains. Bozeman: Montana Agr. Exp. Sta. Jan.: (Abstract)

Friesen, G. and L.H. Shebeski. 1961. The influence of temperature on the germination of wild oat seeds. Weeds 9:634-638.

Gasquez, J. and J.P. Compoint. 1981. Enzymatic variations in populations of *Chenopodium album* L. resistant and susceptible to triazines. Agroeco. 7:1-10.

Georghiou, G.P. Managing Reistance to Agrochemicals: From Fundamental Research to Practical Strategies. In: ACS Symp. Ser. No. 21, edited by Green, M.B., LeBaron, H.M., and Moberg, W.K. Washington, D.C.: American Chemical Society, 1990, p. 18-41.

Ghersa, C.M., M.A., Ghersa, M.L. Roush, and S.R. Radosevich. 1991. Fitness studies of italian ryegrass r esistant to diclofop-methyl: 2. Pollen phenology and gene flow. Proc. West Soc.Weed Sci. 44:42

Gressel, J. Need herbicide resistance have evolved? Generalizations from around the world, Adelaide: Crop Science Society of South Australia, Inc., 1990. pp. 173-184.

Griffiths, D.J. and T.D. Johnston. 1956. Origin of common wild oats, *Avena fatua* L. Nature 178:99-100.

Haldane, J.B.S. 1960. More precise expressions for the cost of natural selection. J. Genet. 57:351-360.

Halstead, S.J., K.L. Gross, and K.A. Renner. 1990. Geostatistical analysis of the weed seed bank. Proc. North Cent. Weed Sci. Sco. 45:123-124.

Hamilton, M.A., K.R. Johnson, A.K. Camper, P. Stoodley, G.J. Harkin, R.J. Gillis, and P.A. Shope. (1995). Analysis of bacterial spatial patterns at the initial stage of biofilm formation. The Biometric. J. (In Press).

Heap, I.M., B.E. Murray, H.A. Loeppky, and I.N. Morrison. 1994.Resistance to aryloxyphenoxypropionate and cyclohexadione herbicides in wild oat (*Avena fatua*). Weed Sci. 41:232-238.

Holt, J.S. 1994. History of identification of herbicide-resistant weeds. Weed Technol. 6:615-620.

Holt, J.S. and D.C. Thill. Growth and Productivity of Resistant Plants. In: Herbicide Resistance in Plants. Biology and Biochemistry, edited by Powles, S.B. and Holtum, J.A.M. Boca Ranton: CRC Press, 1994, p. 299-316.

Holt, J.S. and H.M. LeBaron. 1990. Significance and distribution of herbicide resistance. Weed Technol. 4:141-149.

Holt, J.S. Fitness and Ecological Adaptibility of Herbicide-Resistant Biotypes. In: Managing Resistance to Agrochemicals: From Fundamental Research to Practical Strategies, edited by Green, M.B., LeBaron, H.M. and Moberg, W.K. Washington, D.C.: American Chemical Society, 1990, p. 419-429.

Holt, J.S., S.B. Powles, and J.A.M. Holtum. 1993. Mechanisms and agronomic aspects of herbicide resistance. Annu. Rev. Plant Physiol. Plant Mol. Biol. 44:203-229.

Howard, C.L., A.M. Mortimer, P. Gould, P.D. Putwain, R. Cousens, and G.W. Cussans. 1991. The dispersal of weeds: Seed movement in arable agriculture. Brighton Crop Potection Conference - Weeds.821-828.

Imam, A.G. and R.W. Allard. 1965. Population studies in predominatly self-pollinated species. VI. Genetic variability between and within natural populations of wild oats from differing habitats in California. Genetics 51:49-62.

Jacobsohn, R., and R.N. Andersen. 1968. Differential response of wild oat lines to dillate, triallate, and barban. Weed Sci. 16:491-494.

Jana, S. and J.M. Naylor. 1982. Adaptation for herbicide tolerance in populations of *Avena fatua* Weed species, cropland, Canada. Can. J. Bot. 60:1611-1617.

Jasieniuk, M., A.L. Brule-Babel, and I.N. Morrison. 1994. Inheritance of trifluralin resistance in green foxtail (*Setaria viridis*). (In Press).

Jasieniuk, M., A.L. Brule-Babel, and I.N. Morrison. 1994. The evolution and genetics of herbicide resistance in agricultural weeds. (In Press).

Jasieniuk, M., and B.D. Maxwell. 1994. Population genetics and the evolution of herbicide resistance in weeds. (In Press).

Joseph, O.O., S.L.A. Hobbs, and S. Jana. 1990. Diclofop resistance in wild oat (*Avena fatua*). Weed Sci. 38:475-479.

LeBaron, H.M. Distribution and seriousness of herbicide-resistant weed infestations worldwide. In: Herbicide Resistance in Weeds and Crops, edited by Casley, J.C., Cussans, G.W. and Atkins, R.K. Oxford: Butterworth-Heinemann, Ltd., 1991, p. 27-43.

Mallory-Smith, C.A., D.C. Thill, M. Alcocer-Ruthling, and C.R. Thompson. 1992. Growth comparisons of sulfonylurea resistant and susceptible biotypes. Proceedings of the First International Weed Control Congress 6:858-864.

Malzew, A.I. 1930. Wild and cultivated oats. Bull. Appl. Bot. Gener, Plant Breed. Suppl. 38:Leningrad.

Marshall, D.R and S.K. Jain. 1969. Genetic polymorphism in natural populations of *Avena fatua* and *A. barbata*. Nature 221:276-278.

Mather, H.J. 1946. The control of wild oats in Alberta. Alta. Dept. Agric. Circ. 71

Matthews, J.M. Management of Herbicide Resistant Weed Populations. In: Herbicide Resistance in Plants: Biology and Biochemistry, edited by Powles, S.B. and Holtum, J.A.M. Boca Ranton: CRC Press, 1994, p. 317-335.

Maxwell, B.D. and C. Ghersa. 1992. The influence of weed seed dispersion versus the effect of competition on crop yield. Weed Technol. 6:196-204.

Maxwell, B.D. and A.M. Mortimer. Selection for Herbicide Resistance. In: Herbicide Resistance in Plants: Biology and Biochemistry, edited by Powles, S.B. and Holtum, J.A.M. Boca Raton, Ann Arbor, London, Tokyo: Lewis Publishers, CRC Press Inc., 1994, p. 1-25.

Maxwell, B.D. and C.M. Ghersa. 1992. The influence of weed seed dispersion versus the effect of competition on crop yield. Weed Technol. 6:192-204.

McBeath, D.K., D.A. Dew, and H.A Friesen. 1970. Competition between barley and wild oats as affected by nitrogen, barban and time of seeding. [*Avena fatua*]. Can. J. Plant Sci. 50:541-550.

McCanny, S.J. and P.B. Cavers. 1988. Spread of proso millet(*Panıceum miliacem*) in Ontario, Canada. 2. Dispersal by combines. Weed Res. 28:67-72.

McMullan, P.M. and J.D Nalewaja. 1991. Triallte absorption and metabolism in relationship to tolerance in wheat (*Triticum aestivum* and *Triticum durum*). Can. J. Plant Sci. 71:1081-1088.

Melander, A.L. 1914. J. Econ. Entomol. 7:167-173.

Miller, S.D. Triallate. In: Systems for weed control in North America, edited by Donald, W.W. Champaign: Weed Science Society of America, 1990, p. 266-280.

Miller, S.D., J.D. Nalewaja, and C.E.G. Mulder. 1982. Morphological and physiological variation in wild oat *Avena fatua*, dormancy, herbicide resistance, varieties, weeds. Agron. J. 74:771-775.

Miller, S.D., J.D. Nalewaja, E. Pacholak, and C.E.G Mulder. 1977. MSMA [monosodium, methanearsonate] for weed [*Avena fatua, Setaria*] control in wheat. Proc. Annu. Meet. North Cent. Weed Control Conf. 32:133-135.

Miller, S.D.,and J.D. Nalewaja. 1990. Influence of burial depth on wild oats (*Avena fatua*) seed longevity. Weed Technol. 4:514-517.

Morishita, D.W. and D.C. Thill. 1988a. Factors of wild oat (*Avena fatua*) interference on spring barley (*Hordeum vulgare*) growth and yield. Weed Sci. 36:37-42.

Morishita, D.W. and D.C. Thill. 1988b. Wild oat (*Avena fatua*) and spring barley (*Hordeum vulgare*) growth and development in monoculture and mixed culture. Weed Sci. 36:43-48.

Mortensen, D.A., G.A. Johnson, and L.J. Young. 1993. Weed distribution in agricultural fields. pgs. 113-124 In: Robert, P.C., R.H. Rust, and W.E. larson (eds.) Soil specific crop management. American Society of Agronomy, Madison, WI.

Mullin, C.A., and Scott, J.G. Biomolecular Basis for Insecticide Resistance. In: Molecular Mechanics of Insecticide Resistance, edited by Mullin, C.A. and Scott, J.G. Washington, D.C.: ACS Symp. Series 505

Murray, B.G. 1994. Ph.D. Thesis. University of Manitoba.

Murray, B.E. 1994. Can antibiotic resistance be controlled? New Engl. J. Med. 330:1229-1230.

Nalewaja, J.D. 1970. Wild oat: A persistent and competitive weed. Weeds Today 1(2):10-13.

Nalewaja, J.D. 1977. Wild oats: global gloom. West. Soc. of Weed Sci. Proc.30:21-32.

Noe, J.P., and K.R. Baker. 1985. Relation of within-field spatial variation of plant-parasitic nematode population densities and edaphic factors. Phytopathol. 75:247-252.

O'Donovan, J.T., E.A. De-St-Remy, P.A. O'Sullivan, D.A. Dew, and A.K. Sharma. 1985.Influence of the relative time of emergence of wild oat (*Avena fatua*) on yield loss of barley (*Hordeum vulgare*) and wheat (*Triticum aestivum*). Weed Sci. 33:498-503

O'Donovan, J.T., M.P. Sharma, K.N Harker, D. Maurice, M.N. Baig, and R.E. Blackshaw. 1994. Wild oat (*Avena fatua*) populations resistant to triallate are also resistant to difenzoquat. Weed Sci.42:195-199.

Paterson, J.G., W.J.R. Boyd, and N.A Goodchild. 1976. Effect of temperature and depth of burial on the persistence of seed of *Avena fatua* L. [wild oat] in Western Australia. J. Appl. Ecol. 13:841-847.

Price, S.C., J.E. Hill, and R.W. Allard. 1983. Genetic variability for herbicide reaction in plant populations Wild oat, *Avena barbata*, *Avena fatua*, godetia, *Clarkia williamsonii*, toxicity, Sierra Nevada in California. Weed Sci. 31:652-657.

Price, S.C., R.W. Allard, J.E. Hill, and J. Naylor. 1985. Associations between discrete genetic loci and genetic variabiblity for herbicide reaction in plant populations. Weed.sci.33:650-653.

Primiani, M.M., J.C. Cotterman, and L.L. Saari. 1990. Resistance of kochia (*Kochia scoparia*) to sulfonylurea and imidazolinone herbicides. Weed Technol. 4:169-172.

Ripley, B.D. 1976. The second-order analysis of stationary point process. J. Appl. Probabiltiy. 13:255-266.

Robertson, P.G., M.A. Huston, F.C. Evans, and J.M. Tiedje. 1988. Spatial variability in a successional plant communtiy: Patterns of nitrogen availibilty. Ecology 69:1517-1524.

Roush, M.L., S.R. Radosevich, and B.D. Maxwell. 1990. Future outlook for herbicide-resistance research. Weed Technol. 4:208-214.

Ryan, G.F. 1970. Resistance of common groundsel to simazine and atrazine. Weed Sci. 18:614.

Seefeldt, S.S., D.R. Gealy, B.D. Brewster ,and E.P. Fuerst. 1992. Diclofop resistant wild oat (*Avena fatua* L.) in the Willamette Valley of Oregon: Distribution, case histories, and cross-resistance to other herbicides. Abstr. Weed. Soc. Am. 33:19

Seefeldt, S.S., D.R. Gealy, B.D. Brewster, and Fuerst E.P. 1994. Cross-resistance of several diclofop-resistant wild oat (*Avena fatua*) biotypes from the Willamette Valley of Oregon. Weed Sci. 42:430-437.

Seefeldt, S.S., D.R. Gealy, E.P. Fuerst, B.D. Brewster, and A.P. Appleby. 1992. Investigations of diclofop-methyl resistant wild oat (*Avena fatua* L.) biotypes from Oregon. Abstr. Weed. Soc. Am. 32:89

Sharma, H.C. and J.H. Hunter. 1974. Effect of time of removal of wild oat competition in wheat. Res. Rep. Wheat Comm. (West. Sect.)p. 13.

Sharma, M.P., D.K. McBeath, and W.H Vanden-Born. 1976. Studies on the biology of wild oats [*Avena fatua*]. I. Dormancy, germination and emergence. Can .J .Plant Sci. 56:611-618.

Somody, C.N., J.D. Nalewaja, and S.D. Miller. 1984. The response of wild oat (*Avena fatua*) and *Avena sterilis* accessions to photoperiod and temperature [Morphology, seed dormancy, growth rate]. Weed Sci 32:206-213.

Somody, C.N., J.D. Nalewaja, and S.D. Miller. 1984. Wild oat (*Avena fatua*) seed environment and germination [North Dakota]. Weed Sci. 32:502-507.

Somody, C.N., J.D. Nalewaja, and S.D. Miller. 1985. Self-burial of wild oat florets. Agron. J. 77:359-362.

Stephenson, G.R., M.D. Dykstra, R.D. McLaren, and A.S. Hamill. 1990. Agronomic practices influencing triazine-resistant weed distribution in Ontario. Weed Technol. 4:199-207.

Taylor, C.R. and O.R. Burt. 1984. Near-optimal management strategies for controlling wild oats in spring wheat. Amer. J. Agric. Econ. 66:50-60.

Thai, K.M., S. Jana, and J.M. Naylor. 1985. Variability for response to herbicides in wild oat (*Avena fatua*) populations. Weed Sci. 33:829-835.

Thornton, P.K., R.H. Fawcett, J.B. Dent, and T.J. Perkins. 1990. Spatial weed distribution and economic thresholds for weed control. Crop Prot. 9:337-342.

Thurston, J.M. 1954. A survey of wild oat plants (*Avena fatua* and *A. ludoviciana*) in England and Wales in 1951. Ann. Appl. Biol. 41:619-636.

Thurston, J.M. 1961. The effect of depth of burying and frequency of cultivation on survival and germination of seeds of wild oats (*Avena fatua* L. and *Avena ludoviciana* Dur.). Weed Res. 1:19-31.

Tomasz, A. 1994. Multiple-antibiotc-resistant pathogenic bacteria. New Engl. J. Med. 330:1247-1251.

Uyenoyama, M.K. Plieotropy and the evolution of genetic systems conferring resistance to pesticides. In: Pesticide Resistance: Strategies and Tactics for Management, Washington, D.C.: National Academy Press, 1986, p. 207-221.

Warwick, S.I. and P.B. Marriage. 1982. Geographical variation in populations of *Chenopodium album* resistant and susceptible to atrazine. II. Photoperiod and reciprocal transplant studies Herbicide resistance. Can. J. Bot. 60:494-504.

Warwick, S.I. and P.B. Marriage. 1982. Geographical variation in populations of *Chenopodium album* resistant and susceptible to atrazine. I. Between and within-population variation in growth restponse to atrazine. Can. J. Bot. 60:483-493.

Whitson, T.D., Burrill, L.C, Dewey, S.A., et al. Weeds of the West, Jackson:University of Wyoming, 1991.

Wiles, L.J., Wilkerson, G.G., Gold, H.J., and H.D. Coble. 1992. Modeling weed distribution for improved postemergence control decisions. Weed Sci.40:546-553.

Wilson, B.J. 1978. The long term decline of a population of *Avena fatua* L. with different cultivations associated with spring barley cropping. Weed Res. 18:25-31.

Wilson, B.J. and N.C.B. Peters. 1982. Some studies of competition between *Avena fatua* L. and spring barley. I. The influence of *Avena fatua* on yield of barley. Weed Res. 22:143-148.

Wood, H.E. 1953. The occurence and the problem of wild oats in the great plains region of North America. Weeds 2:292-294.

World Health Organization Expert Committee on Insecticides. 1957. 7th Report. W.H.O. Tech. Rept. Ser. 125 pp.

Xunhong, C. 1992. Geostatistical methods for the study of pressure compartments; A case study in the Hilight oil field, Powder River Basin, Wyoming.

Yost, R.S., G.Uehara, and R.L. Fox. 1982. Geostatsistical analysis of soil chemical properties of large land areas. II. Kriging. Soil Sci. Soc. Am. J. 46:1033-1037.

FAIRFIELD BENCH

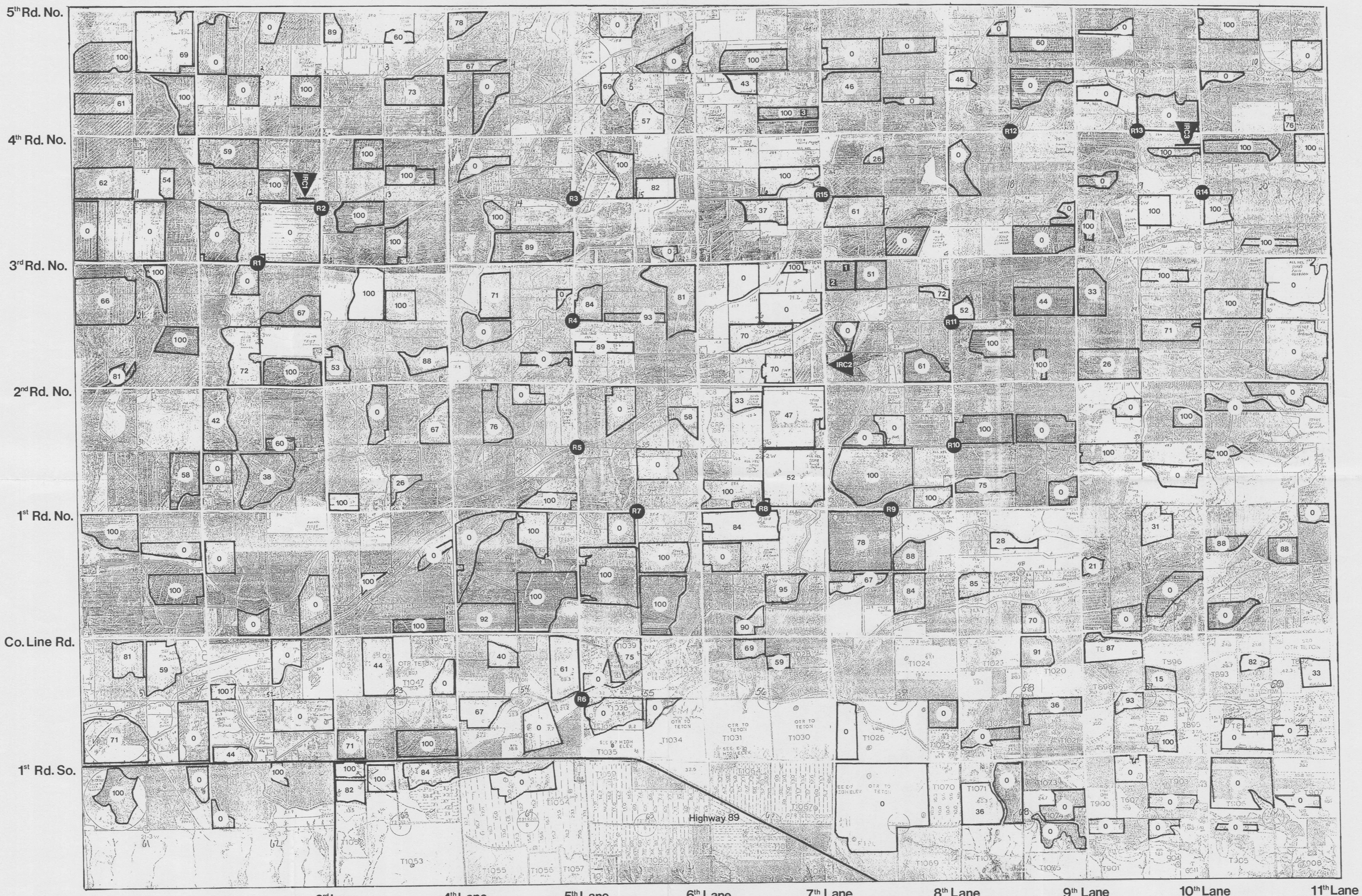

FIGURE 1. FAIRFIELD BENCH STUDY REGION