Growth and Seed return of Auxinic Herbicide Resistant

Wild Mustard (*Sinapis arvensis*).

A Thesis

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by

Daniel James Debreuil

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GROWTH AND SEED RETURN OF AUXINIC HERBICIDE

RESISTANT WILD MUSTARD (Sinapis arvensis)

BY

DANIEL JAMES DEBREUIL

A Thesis/Practicum submitted to the Faculty of Graduate Studies of the University of Manitoba in partial
fulfillment of the requirements for the degree of

MASTER OF SCIENCE

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ABSTRACT

Debreuil, D.J., M.Sc., The University of Manitoba, 1994. Modelling the Evolution of Auxinic Herbicide Resistance in Wild Mustard (Sinapis arvensis) grown with wheat. Major Professor: Dr. I.N. Morrison, Department of Plant Science.

In field trials conducted at Portage la Prairie in 1992 and 1993 the growth and seed return of an auxinic herbicide resistant (R) wild mustard was compared to that of a susceptible (S) biotype in a wheat crop. In the absence of herbicide, the S wild mustard biotype accumulated shoot dry matter more quickly than the R biotype throughout most of the growing season in both years. However, only in 1993 did the S biotype set substantially more seed than the R biotype (3120 seeds plant^{-1} vs. 2520). The recommended dosage (420 g ha^{-1}) of 2,4-D for wild mustard control killed all S plants in both years and severely inhibited the growth and seed return of R plants. However, R seed rain calculated on a square meter basis (20 wild mustard plants m^{-2}) was still very high: 9000 and 5700 seeds m^{-2} in 1992 and 1993, respectively. The recommended dosage (300 g ha^{-1}) of dicamba did not inhibit the growth and seed return of either S or R wild mustard to the same extent as 2,4-D. The results of this study indicate that the R wild mustard exhibits a functional level of resistance to recommended dosages of the auxin-type herbicides, 2,4-D and dicamba, when growing in wheat. Furthermore, the study indicates that the recommended dosage of 2,4-D selects very highly for R genes within a wild mustard population.

Inserting the results into Gressel and Segel's herbicide resistance rotational model (1990) indicates a very rapid development of resistance in a wild mustard population unless the initial frequency of R individuals is set very low (i.e., 10^{-30}). Since the initial mutation frequency is the parameter in the model about which the least is known, it may be that a stable, heritable mutation conferring resistance to auxin-type herbicides in wild mustard occurs very infrequently. Given the widespread use of auxin-type herbicides over the last 30 to 40 years and the paucity of plants resistant to these herbicides, a very low, heritable mutation frequency may be a reasonable assumption.

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My parents, Barbara and Lucien. Your encouragement was strong throughout my university career. The road was rocky at times. Yet you had the patience to let me make my own mistakes. From my point of view, it was easy, as I knew I had the support at home, when ever I wanted it. That was extremely important, as it gave me the confidence to explore on my own time. I hope that Mary-Jane and I can provide our children a similar type of environment. An environment flexible enough to allow the children to develop and learn on their own time. An environment that places equal emphasis on social and academic learning. Thank you, Mom and Dad.

Mary-Jane, four years ago, we together made the decision to go ahead with graduate school. We knew it would be tough, but we knew it would be worthwhile. Today, we still feel this way. Mary-Jane, thank you for your patience, devotion and encouragement. We did it.

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INTRODUCTION

Wild mustard, *Sinapis arvensis* L., occurs in all Canadian provinces as a weed of field and horticultural crops. A long soil seedbank life, a competitive growth habit, and high fecundity all contribute to the weedy nature of wild mustard and ensure that it remains a continuing problem (Mulligan and Bailey 1975). Up to the mid-fifties wild mustard was the worst weed of cultivated land on the Canadian prairies (Pavlychenko and Harrington 1934; Friesen and Shebeski 1960). The commercialization and the subsequent extensive usage of the phenoxy herbicides 2,4-D and MCPA in the early 1950s reduced the seriousness of the wild mustard problem. Nevertheless, it still ranks as the fourth most abundant weed of field crops on the prairies (Thomas and Wise 1988).

In 1991, a population of wild mustard from Gilbert Plains, Manitoba was confirmed to be resistant to the herbicides 2,4-D, MCPA, dichlorprop, mecoprop, dicamba and picloram (Heap and Morrison 1992). From field trials, it was determined that the R wild mustard was about 20 times more resistant to 2,4-D, and more than 30 times more resistant to dicamba, than a known S wild mustard population.

The objectives of the research in this thesis were 1) to investigate the growth and seed return of auxin-type herbicide resistant wild mustard in a wheat crop when treated with 2,4-D or dicamba and 2) to utilize this information in modelling the evolution of herbicide resistance in this weed.

CHAPTER 1

LITERATURE REVIEW

1.1 Wild Mustard.

Introduction. Wild mustard, *Sinapis arvensis* L., is a member of the Cruciferae family. It occurs in all Canadian provinces, principally as a weed of field and horticultural crops. Wild mustard was the most common weed identified by Friesen and Shebeski (1960) in 142 Manitoba grain fields surveyed during the years 1956 to 1958. Wild mustard has since been effectively controlled in cereal grains by 2,4-D (*2,4-dichlorophenoxyacetic acid*) and MCPA (*4-chloro-2-methylphenoxyacetic acid*). Nevertheless, it still ranks as the fourth most abundant weed in Manitoba (Thomas and Wise 1988).

Biology. Mulligan and Bailey (1975) describe wild mustard as an annual, broad-leaved plant. Seedlings initially form a rosette and later develop into an erect plant, 30-100 cm in height. Stems are typically hairy, profusely branched, and purple at the junctions between the branches. In mature plants the bottom leaves are lobed, hairy and petioled. The upper leaves are not lobed or petioled, are smaller than the lower leaves, and are alternately arranged.

Wild mustard has bright yellow flowers, each with four petals, borne in racemose clusters at the ends of stems and branches. Flowers open first at the bottom of each cluster. Insects are attracted to light reflected from the brightly colored

flowers (Mulligan and Bailey 1975). Visiting insects are essential for pollen dispersal because wild mustard is self-incompatible.

After flowering, distinctive pods are formed, with inflorescences often bearing ripened pods at the bottom and new buds at the top. Wild mustard is distinguished from other cruciferous plants as having a viable seed in the beak of each silique (Mulligan and Bailey 1975; Frankton and Mulligan 1987). Although it is an outcrossing species, wild mustard is described as being morphologically and cytologically quite uniform throughout its Canadian range (Mulligan and Bailey 1975).

Wild mustard is a prolific seed producer. In a non-competitive environment individual plants can produce well over 3500 seeds (Mulligan and Bailey 1975). Plants grown in cultivated fields will produce as many as 2000 to 3500 seeds (Mulligan and Bailey 1975). Blackshaw *et al.* (1987) reported wild mustard plants in competition with rapeseed (*Brassica napus*) and lamb's quarters (*Chenopodium album*) produced between 400 to 600 seeds. Edwards (1980) reported that wild mustard produced up to 750 seeds plant^{-1} when competing with cereals.

In undisturbed land wild mustard seeds can remain dormant and viable for up to sixty years (Mulligan and Bailey 1975). However, wild mustard seed dormancy can be broken with germination taking place when favourable growing conditions arise.

Goudey *et al.* (1986) reported a maximum wild mustard seed germination of 51% with independent treatments of temperature, light and nitrogen. However, up to 92% germination occurred when wild mustard seeds were treated with combinations of the three "dormancy breaking" treatments. The most successful treatment included 10mM KNO_3 + NH_4Cl, plus exposure to red light (10 minutes), and a 96 hour exposure to 20 C after 48 hours of treatment at 5 C.

From experiments with excised embryos and using seeds exposed to sulphuric acid to remove the seed coat, Edwards (1968) determined that seed dormancy in wild mustard is regulated by an inhibitor produced by the embryo under low oxygen concentrations.

Edwards (1980) reported wild mustard seedling emergence to be correlated with soil temperature. Results from field trials, conducted over a three year period, indicated that seedlings emerge when the mean weekly soil temperature at a 10 cm depth was above 4.4 C. From the same study it was also determined that annual emergence of wild mustard was just 2.5% of the total wild mustard seed in the seed bank.

Kropac (as cited in Edwards 1980) determined that average annual seedbank death and decay for wild mustard was 17.9%. To this value Edwards added the wild mustard emergence value of 2.5% (of the total wild mustard seedbank) to obtain a total annual wild mustard seedbank loss of 20.4%. A previously determined

value for unemerged, germinated seed brought the total annual wild mustard seedbank loss to 22.8%.

The ability of wild mustard seed to germinate and emerge when environmental conditions are favourable ensures long-term survival and contributes to its weedy nature. Detailed studies on mechanisms controlling seed bank dynamics under prairie conditions are lacking. Future investigations would provide valuable insights and allow for more accurate estimates when used in predictive models to ascertain rates of resistance enrichment within weed populations.

Weedy nature. Due to its tall stature, robust growth habit and large root system (Pavlychenko and Harrington 1934), wild mustard is a strongly competitive weed (Friesen and Shebeski 1960; Alex 1970; Richardson 1980; Blackshaw *et al.* 1987; Wall *et al.* 1991).

Burrows and Olsen (1955a) reported that wild mustard interfered with the growth, tillering and yield of wheat grown in Manitoba, by competing for moisture, light and nutrients. For example, 25 wild mustard plants m^{-2} decreased the grain yield of wheat seeded at 135 kg ha^{-1} by 16%. Richardson (1980) observed sparse wild mustard infestations ($<$ 5 plants m^{-2}) reduced dwarf barley yield by up to 41% in the United Kingdom. Similarly, 13 wild mustard plants m^{-2} reduced sunflower yields by 35% (Nalewaja *et al.* as cited in Friesen (1988)) and, 20 wild mustard plants m^{-2} reduced the yield of field pea (*Century*) by up to 35% (Wall *et al.* 1991).

Dry conditions have been observed to reduce the competitiveness of wild mustard. Edwards (1980) reported that drought conditions decreased wild mustard plant numbers and seed production per plant. This report concurs with the observations of Friesen and Shebeski (1960) who noted that wild mustard infestations were less severe in Manitoba grain fields in 1958 than in 1956 or 1957. The researchers attributed the decreased wild mustard densities to extremely dry conditions occurring early in the 1958 growing season. Moreover, Wall *et al.* (1991) reported pea yield losses due to wild mustard competition were least in 1988 as compared to 1987 and 1989. They too attribute the reduced competition in 1988 to the fact that it was a year with below average precipitation. Similarly, Blackshaw *et al.* (1987) reported that canola yield losses due to wild mustard competition were less in dry years than in moist years.

Wild mustard is a contaminant. Wild mustard is an alternate host for insect and fungal pests of cruciferous crops, particularly *Brassica rapa* and *Brassica napus*. White rust (*Albugo candida* (Pers.) O. Ktze.) is an economically important disease of *Brassica rapa*. The pathogen causing white rust can successfully infect wild mustard, and in favourable conditions allow for proliferation of the disease (Downey and Rimmer 1993). Moreover, early germinating wild mustard plants can nurture overwintered adult crucifer flea beetles until such time as the canola emerges later in the season (Philip and Mengersen 1989).

Wild mustard seed is considered a contaminant in harvested crops including canola, rapeseed and domestic mustard. More than five percent by weight of wild

mustard seed in these crops results in them being assigned a sample grade at the primary elevator (Canadian Grain Commission 1993).

Wild mustard seed is high in glucosinolates, saponins and tannins, and is therefore unpalatable to livestock (Kingsbury 1963). The ingestion of wild mustard seed by livestock can cause serious illness, and lead to premature death (Kingsbury 1963; Mulligan and Bailey 1975; Frankton and Mulligan 1987). Feed grain must have less than 1% by weight of wild mustard seed (Canada Feeds Act 1983).

Wild mustard is included as a "primary noxious" weed in the Federal Seeds Act and Regulations of Canada. The regulations accompanying the act limit the number of wild mustard seeds permitted in commercial seed. Pedigreed canola seed growers must comply with a zero tolerance level regarding wild mustard seed contamination, as stipulated by the Canada Seeds Act, 1986 (Louise Cook[1], pers. comm.).

1.2 Auxin-type Herbicides for Wild Mustard Control

Auxin-type herbicide groups. The auxin-type herbicides currently used in Western Canada are grouped by their chemical structure and include phenoxyalkanoic acids, benzoic acids, aromatic carboxymethyl derivatives, pyridine derivatives and quinoline carboxylic acids (Cobb 1992). The most widely used groups are the

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phenoxyalkanoic acids (phenoxy) and the benzoic acids which include 2,4-D (phenoxy) and dicamba (*3,6-dichloro-2-methoxybenzoic acid*) (benzoic acid). 2,4-D is highly effective in controlling wild mustard, and is widely used to control this weed. Dicamba is less effective than 2,4-D on wild mustard, but is more effective on other problem weeds like wild buckwheat and green smartweed.

History. The phenoxyalkanoic acids, 2,4-D and MCPA, were the first of the auxin-type herbicides to be used in commercial grain production. In 1941, both herbicides were synthesized independently and were subsequently kept secret until the end of the Second World War (Peacock 1978; Cobb 1992). In 1945, 2,4-D was first marketed in the United States for agricultural use by the American Chemical and Paint Company. One year later the Imperial Chemical Company marketed MCPA as a 1% active dust (Peacock 1978). The introduction of these products revolutionized modern agriculture.

With the introduction of 2,4-D and MCPA an effective, inexpensive selective chemical weed killer was available to the farmer for the first time. Farming became more productive with less reliance on labour to control weeds. Almost fifty years have passed and the "phenoxy" herbicides 2,4-D and MCPA are still among the world's most effective and widely used herbicides (Cobb 1992; Devine *et al.* 1993).

Herbicide properties. Auxin-type herbicides are synthetic analogues of natural plant hormones (Ashton and Crafts 1981; Cobb 1992; Devine *et al.* 1993).

Synthetic auxins stimulate the plant enzyme 1-aminocyclopropane carboxylic acid synthase which induces ethylene evolution and uncontrolled growth in susceptible plants. In some plants auxin-induced epinasty is linked with ethylene evolution; however, this does not occur in all species (Barnwell and Cobb 1989).

Auxin-type herbicides are usually foliar-applied, and are absorbed through the leaf cuticle into the apoplast. Examining cuticles from 8 different species (7 leaf cuticles and 1 tomato fruit cuticle), Norris (1974) determined that there was no correlation between cuticle thickness and penetration of 2,4-D. For example, among those plants tested the cuticle of the tomato fruit was thickest; yet 2,4-D penetrated it most easily. From further analysis Norris (1974) concluded that penetration into the plant by 2,4-D was more directly related to the composition of cutin and wax of the cuticle than the cuticle thickness.

Peniuk et al. (1993) reported rapid penetration of both 2,4-D and dicamba into leaves of phenoxy-susceptible and -resistant populations of wild mustard[2]. Using radiolabelled herbicides, they determined that there was greater than 95% penetration within 12 hours of application for both populations.

Auxinic herbicides penetrate the plasmalemma where they are readily translocated to the meristematic tissues via the phloem (Ashton and Crafts 1981; Devine 1989). Chang and Vanden Born (1968) used radiolabelled dicamba to measure

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[2]. Seed from the same population of R wild mustard studied in this thesis (see Materials and Methods).

absorption and translocation in four species, including wild mustard, under controlled environment conditions. In wild mustard, dicamba was quickly absorbed into the leaf and translocated from the leaf to meristematic tissue. The relative susceptibility of the four species was correlated with the amount of dicamba absorbed and translocated.

The site of action of auxin-type herbicides is purported to be an auxin binding protein. However, the details of the cause-and-effect sequence started after binding of an auxin herbicide to a plasmalemma auxin-binding protein are not known (Devine *et al.* 1993). The precise mechanism of action for auxinic herbicides is not known because the current understanding of auxin and its effects on plant physiology is incomplete. Auxin exists within the plant at very low doses with different tissues having various amounts. The phytotoxic action of auxinic herbicides occurs as a result of their ability to mimic endogenous auxin. The enormously high relative concentrations of exogenous auxin as a result of herbicide treatment creates an imbalance relative to intracellular auxin concentrations, resulting in a perturbation of normal growth. The ongoing mobilization of metabolic reserves for excessive growth eventually leads to the loss of cellular functions, cellular integrity, and repair capacities. Symptoms of auxin imbalance are stem and petiole curling and elongation, stem and petiole thickening, and the formation of irregular adventitious roots. Eventually plant tissues begin to desiccate and/or disintegrate. Hence, the herbicidal effect of auxin-type herbicides is to supply an overdose of auxin to the plant, causing death (Devine *et al.* 1993).

Phenoxy herbicides and wild mustard. "*Sinapis arvensis is killed by the herbicide 2,4-D. The successful control of this weed in grain crops helped bring about the acceptance of 2,4-D by the majority of cereal grain growers in Western Canada*" (Alex 1970).

Wild mustard is very susceptible to most phenoxy herbicides. From the early 1950's, wild mustard has been controlled effectively with the phenoxy herbicides, 2,4-D and MCPA. As such, the weed is no longer considered a problem in the United Kingdom (Edwards 1980; Richardson 1980). Although phenoxy herbicides have reduced the wild mustard problem in Western Canada compared to what it was forty years ago, the weed remains a major problem in crops such as canola which are sensitive to these herbicides (Thomas 1984).

Wild mustard is more susceptible to phenoxy herbicides at early stages of development than at later stages. Burrows and Olson (1955a) observed a high mortality of wild mustard when it was sprayed at the early four-leaf stage (5 to 12 cm in height) with 280 (g a.i. ha^{-1}) of 2,4-D. Efficacy of the herbicide was substantially less at later leaf stages. In a separate experiment, 280 g ha^{-1} rate applied at the four-leaf stage was more effective than a 420 g ha^{-1} rate at the late five-leaf stage (22-30 cm in height). Currently it is recommended that 2,4-D be applied at 420 g a.i. ha^{-1} to wild mustard at the 2 to 4-leaf stage (Manitoba Agriculture 1991).

Wild mustard is not as susceptible to dicamba as it is to 2,4-D. In Western Canada, dicamba alone is not recommended for selective control of broadleaf weeds in cereals. Even the highest dosage (140 g a.i. ha[-1]) of dicamba registered for use in Western Canada for selective weed control in cereals does not provide consistently good control of wild mustard (Dwight Willoughby[3], pers. comm.). In growth chamber experiments, wild mustard was not killed by dicamba at dosages from 70 to 240 g a.i. ha[-1] twenty days after treatment (Chang and Vanden Born 1971). From the same experiment the ED_{50} value (equivalent dose of dicamba required to reduce growth by half) for wild mustard was determined to be 35 g a.i. ha[-1].

Dicamba at 300 g a.i. ha[-1] is registered for the control of wild mustard in row-crop corn in Eastern Canada. In Western Canada dicamba is mixed with 2,4-D or MCPA for adequate wild mustard control.

1.3 Herbicide Resistance

Resistance is defined as a genetic change within a pest population in response to selection by a toxicant that may impair control in the field (Holt and LeBaron 1990). Resistant (R) weeds will survive and grow when exposed to an otherwise lethal herbicide dosage (Gressel and Segel 1982). Resistance is either the result of a reduction in herbicide uptake and translocation at the whole plant level or

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[3]. Dwight Willoughby, Sandoz Agro Canada, Winnipeg.

enhanced herbicide detoxification or a modification at the site of action resulting in reduced sensitivity to a herbicide (Warwick 1991).

Resistant weed populations evolve through a process of natural selection. Over time R weeds, which can withstand herbicide treatment, increase in frequency within the population (Gressel and Segel 1990; Holt and LeBaron 1990). Repeated use of the same herbicide or herbicides with a similar mode of action will result in a shift in the population such that, in time, the resistant weeds will predominate.

Resistant biotypes can be cross-resistant to several compounds with a similar mechanism of action (Holt and LeBaron 1990). Usually these compounds are chemically related; however, this is not always the case. O'Donovan *et al.* (1994) identified fifteen populations of R wild oat from central Alberta with cross resistance to the active ingredients triallate *{S-(2,3,3-trichloro-2-propenyl)bis(1-methylethyl)carbmothiate}* and difenzoquat *{1,2-dimethyl-3,5-diphenyl-1-1H-pyrazolium}*, two chemically unrelated compounds.

Weed biotypes may develop multiple resistance when resistance to herbicidal mode(s) of action is selected through multiple or sequential herbicide exposure. In 1988, dinitroaniline-resistant populations of green foxtail were identified in southwestern Manitoba as a result of repeated exposure to these herbicides (Morrison *et al.* 1989b). Farmers with dinitroaniline-resistant green foxtail then became increasingly reliant on ACCase inhibiting herbicides for control, for reasons of efficacy and crop safety. In 1992 a green foxtail population resistant to both

trifluralin and ACCase inhibiting herbicides was identified in Manitoba (Heap 1994; Morrison and Devine 1994).

Herbicide tolerance is defined as a low degree of resistance that is rate dependent (Holt and LeBaron 1990). Tolerance refers to the natural and normal variation in response to pesticides or other control practices occurring within a species. A weed population is tolerant if it displays an inherent ability to withstand a normally lethal herbicide dosage without prior exposure to that particular herbicide.

1.4 Selection - A Measurement of Relative Fitness

Selection is a result of differences in genotypic fitness. The selection intensity exerted by a herbicide is the most important factor determining the rate of R enrichment in a weed population. The higher the selection intensity the more frequent R alleles become in the population.

Selection is a function of rate, frequency of usage, efficacy on targeted weeds and persistence (Gressel and Segal 1982). Herbicide characteristics that correlate with "intense selection" include a single target site, overlapping season-long control, and the ability to control a wide range of targeted weeds.

Fitness is defined as the proportion of genes an individual organism contributes to the gene pool of a population (Holt 1990). Fitness is, therefore, a measure of the

survival and reproduction (fecundity) of each genotype in the population under selection. Population genetics theory measures selection by relative fitness of genotypes in the presence of a particular selecting agent such as a herbicide. Relative fitness is a quantitative estimate of the reproductive success of the weaker genotype compared to the more fit genotype. A fitness value of 1 is assigned to the most prolific genotype. The difference between the relative fitness of the most fit genotype and that of a less fit genotype is termed the selection coefficient, and is directly proportional to the intensity of selection against the less fit genotype (Jasieniuk *et al.* 1994a).

Fecundity. Seed production reflects the change in relative numbers of resistant and susceptible plants in weed populations in the next generation. Weed seed return measurements provide reliable estimates of reproductive fitness, taking into account S individuals that escape herbicide injury, as well as plants that are injured but are still capable of some S weed seed production.

"Knockdown" studies refer to estimates of herbicide efficacy based on observations of initial mortality measured as relative differences in weed densities or shoot dry matter. The assumption that a particular phenotype (S or R) has a higher fitness value on the basis of shoot dry matter production alone is likely subject to considerable error. In order to predict rates of increase or decline in resistance due to herbicide rotation or the complete cessation of use of a particular herbicide group, measurements of relative fitness must incorporate the differential

survival and fecundity of resistant and susceptible phenotypes (Jasieniuk et al. 1994a).

The percentage decrease in seed yield from the direct comparison of a herbicide treated resistant weed population as compared to a treated, susceptible population is determined to be the "effective kill". Gressel and Segel (1978) proposed that seed return measurements (effective kill) should be used to quantify selection. Selection pressure is calculated as [(R seed return$_{treated}$/R seed return$_{untreated}$) / (S seed return$_{treated}$/S seed return$_{untreated}$)]. To date the only study in which selection pressure has been calculated on this basis is one conducted by Beckie and Morrison (1993) in which they determined herbicide selection pressures on S and R green foxtail over a range of trifluralin and ethalfluralin dosages.

1.5 Modelling Weed Resistance To Herbicides

Mathematical population genetic equations (models) have been developed to predict the rate of enrichment of R individuals within a weed population (Gressel and Segel 1978, 1982, 1990; Maxwell et al. 1990; Mortimer et al. 1990). Weed resistance models are based on factors in the weed-crop ecosystem that influence evolutionary processes in populations of plants. The models can be used to investigate a particular parameter (factor) by varying the value of that parameter while assigning constant values to the other parameters. Unfortunately, scientifically verified, appropriate, quantitative data respecting many of the

parameters used in resistance models are lacking. This shortcoming must be acknowledged in interpreting model results as it limits the usefulness of models as predictive tools.

To verify the applicability of weed resistance models in the global sense, verification of individual parameters and overall results should be conducted with a number of resistant weed species with different mechanisms of resistance and different growth and reproductive patterns. Some of the parameters in the models are easier to verify than others. For instance, the relative fitness of R plants can be estimated by measuring seed return. Seed return measurements provide the best indication of relative fitness. Reproductive fitness encompasses fitness at all levels including germination, establishment, growth and reproduction (Holt 1990; Warwick 1991; Jasieniuk *et al.* 1994a).

Beckie and Morrison (1993) determined that the selection pressure imposed by trifluralin and ethalfluralin on dinitroaniline-susceptible and -resistant green foxtail were more appropriately described by measurements of effective kill than by percentage seedling mortality four weeks after herbicide application. They stated that determining weed seed return was essential in order to "*fully understand the evolution and population dynamics of resistant green foxtail populations*". Beckie and Morrison (1993) also determined the fitness of S and R green foxtail in the absence of herbicide treatment. The data were then incorporated into Gressel and Segel's monoherbicide (1978) and herbicide rotational models (1990). Complete verification of the models using the green foxtail data was not possible because

seed bank longevity and initial mutation frequency were not known. Nevertheless, given the calculated selection pressure and fitness parameters and reasonable assumptions of seed bank longevity and initial mutation frequency, model simulations indicated that herbicide rotation will not delay the rate of appearance of dinitroaniline-resistant green foxtail, with exception to the number of seasons (years) trifluralin is not used.

Gressel and Segel's (1990) weed resistance model incorporating rotational strategies is:

$$H_{p,q} = [1 + s(af_{on} - 1)]^p [1 - s(1 - f_{off})]^q$$

H, is the overall enrichment factor indicating the increase in resistance following a period of p 'on' seasons of herbicide application and q 'off' seasons without herbicide. Log_{10} of the enrichment factor (log $H_{p,q}$) corresponds to the negative exponent (base 10) of the initial frequency of resistance. Estimates of initial resistance frequency, or mutation rate, are based on studies conducted mainly with organisms other than higher plants (Duesing 1983). Nevertheless, these mutation frequencies are often assumed to be applicable to higher plants.

s, is the fraction of seeds leaving the seedbank each year. In general, it may be assumed that there is an exponential decline of a seed population in soil to which no new seeds are being added. The components contributing to the decline include the number of emerged seedlings, the number of unemerged seedlings

which decay in the soil, and the number of seeds lost due to death and decay prior to germination (Edwards 1980).

a, refers to the selection pressure. Selection pressure is the most influential factor affecting the rate of enrichment of R individuals within a population (Gressel and Segel 1978, 1982, 1990; Maxwell et al 1990).

Fitness, *f*, is the competitive reproductive fitness of the resistant biotype relative to the susceptible one. It is the overall relative robustness of resistant individuals during germination, establishment, growth, pollination, and seed production. *fon* denotes the relative fitness of R individuals in the presence of herbicide (generally assumed to be 1.0), while *foff* is the relative fitness of R ones in the absence of herbicide.

1.6 Phenoxy Herbicide Resistant Wild Mustard

In 1991, a population of wild mustard from Gilbert Plains, Manitoba was confirmed to be resistant to the phenoxyalkanoic herbicides 2,4-D, MCPA, dichlorprop (*2-(2,4-dichlorophenoxy)propanoic acid*), mecoprop (*2-(4-chloro-2-methylphenoxy)propanoic acid*), and the benzoic acid herbicide dicamba (Heap and Morrison 1992). Phenoxy herbicides, and benzoic acids act as auxin-type herbicides, and have a similar mechanism of action.

Resistance ratios (R/S) derived from GR_{50} values (the dosage of herbicide required to reduce shoot dry matter by 50% relative to the untreated control) indicated that the R wild mustard was 20 times more resistant to 2,4-D than a S wild mustard population, and more than 30 times more resistant to dicamba (Heap and Morrison 1992).

Currently, resistant wild mustard has been identified in nine fields near Gilbert Plains, Manitoba (Friesen 1993). While unproven, it would appear that resistant wild mustard seed was spread from an original site by the use of shared harvesting equipment.

The herbicide use pattern of the field from which the R mustard originated has a history of extensive auxin-type herbicide usage. Target^{TM} (MCPA:mecoprop:dicamba)(4.4:1:1) was applied annually between 1981 and 1990 (TABLE 1-1). In addition other phenoxy herbicides were applied prior to 1981. Initially the wild mustard was effectively controlled by Target^{TM} indicating that continued use of the herbicide caused a build-up of R biotypes. The R wild mustard evolved in a continuous cereal production system in which either wheat or barley was planted every year.

Mechanism of resistance. Studies were conducted at the University of Guelph to determine the mechanism(s) of resistance to auxinic herbicides in wild mustard. No difference occurred between S and R populations with respect to absorption, translocation, exudation and metabolism of foliar applied 2,4-D, dicamba or

TABLE 1-1. Herbicide use pattern selecting for auxinic herbicide-resistant wild mustard.

Year	Crop	Herbicide
1990	Barley	Target™ (MCPA[1])
1989	Barley	Target™ (MCPA[1])
1988	Barley	Target™
1987	Wheat	Target™
1986	Wheat	Target™
1985	Wheat	Target™
1984	Wheat	Target™
1983	Wheat	Target™
1982	Wheat	Target™
1981	Wheat	Target™

[1]. A follow up application of MCPA was applied due to poor wild mustard control from the initial application of Target™.

picloram (Peniuk *et al.* 1993). The results indicate that these factors do not contribute directly to the mechanism of auxinic herbicide resistance in wild mustard.

Differences in ethylene evolution were determined between S and R populations. Within 4 and 44 hours after 2,4-D (75 g a.e. ha^{-1}) treatment S mustard emitted between two- and six-times as much ethylene as R mustard. The results strongly imply that resistance to auxinic herbicides may be due to an altered target site of action (Peniuk *et al.* 1993). More detailed studies of the ethylene evolution pathway of the S and R populations and kinetic analysis of the auxin-binding-protein(s) are required to confirm this hypothesis.

Mode of inheritance of resistance. Resistance to the auxin-type herbicides in wild mustard is conferred by a single, completely dominant gene (Jasieniuk *et al.* 1994b). Reciprocal crosses were made between the resistant population described in this study and a susceptible population from Minto, Manitoba to obtain F_1 hybrids. F_1 hybrids were then selfed to produce F_2 populations and backcrossed to the susceptible parental phenotype. At the 2-4 leaf stage, F_1, F_2, and backcross populations were screened for resistance to dicamba at three rates (50, 200, and 400 g ha^{-1}). F_1 and reciprocal F_1 progeny survived dicamba treatment at all rates and exhibited degrees of injury similar to the resistant parental population. Chi-square values indicated that F_2 progeny segregated in a 3:1 ratio of resistant to susceptible phenotypes at all dicamba rates, and that backcrossed progeny segregated in a 1:1 ratio of resistant to susceptible phenotypes. The response of

the F_1, F_2, and backcross populations to treatment with dicamba indicates that resistance in wild mustard is determined by a single, completely dominant allele.

1.7 Weed Populations Resistant To Phenoxy Herbicides

Globally there have been very few reported cases of phenoxy or auxin-type herbicide resistant weeds. This contrasts with triazine and sulfonyl-urea herbicides where there are hundreds of cases of these chemicals selecting for resistant populations (LeBaron and McFarland 1990).

A review of the literature indicates a number of 'phenoxy tolerant' weed populations. Thus, a distinction must be made between a resistant weed population and a tolerant weed population. Tolerant weed populations are identified by an absence of previous phenoxy-herbicide exposure. Reports of such weed populations are frequent and should not be confused with phenoxy R populations which were selected after repeated exposure. Examples of weed populations that are identified as being 'tolerant', but not resistant, to auxin-type herbicides include: MCPA tolerant scentless mayweed (Ellis and Kay 1975); mecoprop tolerant chickweed (Barnwell and Cobb 1989); and 2,4-D tolerant lamb's quarters (Hume and Shirriff 1989). All these weed populations have an inherent ability to withstand a normally lethal phenoxy herbicide dosage without prior exposure.

Erechtities hieracifolia or fireweed of Hawaii was the first weed reported to have evolved phenoxy herbicide resistance (Hanson 1956; Harper 1956). The population was first reported by Noel Hanson, a senior agronomist with the Hawaiian State Agricultural Experiment Station who noted poor control of the weed in sugarcane fields. Support for Hanson's claim comes from the following evidence: 1) fireweed was once easily controlled with 2,4-D; 2) each field was sprayed with 2,4-D an average of five times per cropping season; 3) after eleven years, 2,4-D did not control fireweed. Hanson reported, *"There is evidence that strains of broad-leaved plants with a relatively high inherited tolerance for 2,4-D have been selected chemically in the spraying process since 1945"*.

A population of *Daucus carota* L. or wild carrot was reported as being resistant to 2,4-D, 4-(2,4-DB) *[4-(2-methyl, 4-chlorolphenoxy) butyric acid]* and 4-(MCPB) *[4-(2,4-dichlorophenoxy) butyric acid]*, but not to 2,4,5-T *[2,4,5-trichlorophenoxyacetic acid]* or 2,4,5-TP *[2,4,5-trichlorophenoxypropionic acid]* (Whitehead and Switzer 1963). This population was targeted for eradication along the railway lines in the province of Ontario. The R population evolved from repeated exposure to 2,4-D as the herbicide was applied at high dosages over a number of years. After initial treatment both S and R plants showed typical signs of auxin overdose, i.e., epinasty and uncontrolled growth (Whitehead and Switzer 1963). After three weeks the S population died while the resistant population recovered from the 2,4-D, 4-(2,4-DB) and 4-(MCPB) treatments.

Sphenoclea zeylanica, commonly known as gooseweed, is a competitive weed in rice fields grown in the Philippines. Gooseweed is normally susceptible to 2,4-D up to the eight-leaf stage. Gooseweed was controlled effectively until 1980. In Bulacan Province poor control with 2,4-D occurred in a population able to resist 2,4-D up to the 7-8 leaf stage (Sy and Mercado 1983). The R gooseweed occurred in fields where 2,4-D had been applied every season over the previous 10 years. A later study conducted by Mercado *et al.* (1990) determined that at the 8- to 12-leaf stage the cuticle of resistant plants was thicker than the cuticle of S plants. Although the evidence is slight, the mechanism of resistance was deemed to be due to reduced herbicide uptake.

Populations of nodding thistle (*Cardus nutans* L.) and giant butter-cup (*Ranunculus acris* L.) were confirmed to be resistant to MCPA in New Zealand (Harrington and Popay 1987; Bourdot *et al.* 1989; Bourdot *et al.* 1990). Over successive years, both pasture weeds were exposed repeatedly to phenoxy herbicides. The R nodding thistle and R giant buttercup populations were 6.7- and 4.9-times more resistant to MCPA than a susceptible population (Bourdot *et al.* 1989). Initial treatments of MCPA caused both S and R populations of giant buttercup to exhibit typical symptoms of an auxin overdose, only to recover soon after.

Nodding thistle and giant buttercup have biennial and perennial life cycles, respectively. As such, both can produce large amounts of seed. Annual application of 2,4-D imposes a very high selection intensity for R plants as both weeds are exposed at least twice before setting seed. This very high selection for

R biotypes is provided as an explanation for the occurrence of these weed populations.

The few reported cases of weed populations resistant to phenoxy herbicides all display a long history of intensive phenoxy herbicide exposure. Only the R gooseweed from the Philippines and the R wild mustard from Manitoba evolved in a field cropping situation.

Recently, Coupland (1994) reviewed resistance to auxinic herbicides and listed 19 different instances of weeds resistant to auxin-type herbicides worldwide, including the resistant wild mustard in this study.

CHAPTER 2

MATERIALS & METHODS

Seed source. Wild mustard seed was originally collected in 1990 in a barley field near Gilbert Plains, Manitoba from plants that survived an application of a commercial mixture of MCPA, mecoprop, and dicamba. This seed was subsequently increased in isolation (approximately 200 m in all directions from other wild mustard plants) on the University of Manitoba, Plant Science Field Research Laboratory at Portage la Prairie, Manitoba during the 1991 growing season. To ensure that only R plants survived to produce seed, the plot was sprayed with 420 g ha^{-1} of 2,4-D. The R wild mustard population appeared to be near-homozygous for resistance as there was very little plant death in the seed increase plot or in the subsequent field experiments. Susceptible wild mustard seed originated from a field near Portage la Prairie and was separated from chaff and screenings.

Field experiments. A field experiment was conducted in 1992 and repeated in 1993 on the University of Manitoba, Plant Science Field Research Laboratory at Portage la Prairie, Manitoba. The experiment was arranged as a split plot with four replications. The main plot treatments consisted of the two populations; susceptible (S) and resistant (R). Seven randomized sub-plot treatments included an untreated control, 2,4-D dimethylamine at 105, 210, and 420 g ha^{-1}, and dicamba dimethylamine at 75, 150 and 300 g ha^{-1}. 2,4-D at 420 g ha^{-1} is recommended for wild mustard control in cereal crops in western Canada, while

300 g ha^{-1} of dicamba is recommended for wild mustard control in corn in eastern Canada. Dicamba applied alone is not recommended for wild mustard control in cereal crops in western Canada because the highest recommended dosage to avoid crop injury is 140 g ha^{-1}. Individual main plots were 35m X 4m; subplot treatments were 5m X 8m.

Soil at the experimental site was a Neuhorst clay loam (Udic Haploboroll) [25% sand, 44% silt, and 31% clay] with an organic matter content of 7.5% and a pH of 7.4. In both years the experimental area had been cropped to spring wheat and confirmed to be free of wild mustard. Prior to seeding, ammonium nitrate phosphate fertilizer (23-23-0) was broadcast at a rate of 70 kg ha^{-1} N and 70 kg ha^{-1} P$_2$O$_5$ and incorporated.

Wild mustard seed was broadcast using a small-plot cone seeder with seed delivery tubes pulled from the openers and hanging freely. The wild mustard seed was then shallowly incorporated using a cultivator and spring-tooth harrows. The wild mustard was seeded five days prior to the wheat in an attempt to synchronize emergence. With the objective of establishing a wild mustard population density of 20 wild mustard plants m^{-2}, approximately 400 S and 200 R wild mustard seeds m^{-2}, respectively, were broadcast in 1992. In 1993, 250 S and 350 R seeds m^{-2} were broadcast. Seeding rates were based on preliminary germination and emergence experiments (TABLES A-1 and A-2). Based on three field studies in the U. K., Edwards (1980) reported that intra-specific competition between wild

mustard plants and cereal crops appeared to be significant only in areas with densities above 20 plants m^{-2}.

Roblin spring wheat at 60 kg ha^{-1} was seeded 4 to 5 cm deep in rows 15 cm apart using a double-disk press drill May 19 and May 15 in 1992 and 1993, respectively. Wheat emerged May 25 and May 23 in 1992 and 1993, respectively. The majority of S wild mustard emerged 2 to 3 days prior to the wheat crop in 1992, and with the wheat in 1993. Resistant wild mustard emerged 2 to 3 days later than S plants.

Within each sub plot, twenty-four 0.5 m^2 permanent quadrats were established, twelve for each wild mustard population. Wild mustard was hand-thinned to 10 plants per quadrat (equivalent to 20 plants m^{-2}). Other broadleaf weeds were removed by hand at this time and quadrats were maintained throughout the growing season.

The herbicide treatments (2,4-D at 105, 210 and 420 g ha^{-1}, and dicamba at 75, 150, and 300 g ha^{-1}) were applied on June 19, 1992 and June 15, 1993 using a bicycle-wheel plot sprayer equipped with flat fan nozzles delivering 108 L ha^{-1} at 275 kPa. At the time of herbicide application in 1992, the S wild mustard had 10 leaves and was just beginning to bolt, while R plants had 7 to 10 leaves. In 1993, the S plants had 6 leaves, while R plants had 4 to 6 leaves.

Natural infestations of wild oat (*Avena fatua* L.) and green foxtail (*Setaria viridis* L.) were controlled with a 2:1 formulated mixture of fenoxaprop-ethyl *{(ethyl 2-[4-[(6-chloro-2-benzoxaazoly)oxy]phenoxy]propanoic acid}* and the herbicide safener fenchlorazole-ethyl *{ethyl-1-(2,4-dichlorophenyl)-5-trichloromethyl-1H-1,2,4-triazole-3 carobxylate}* at 270 g ha^{-1} on June 8, 1992 and June 10, 1993. Flea beetles (*Phyllotreta* spp.) feeding on wild mustard seedlings were controlled by foliar applications of carbofuran *{2,3-dihydro-,2-dimethyl-7-benzofuranol methylcarbamate}* at 130 g ha^{-1} on May 30 and June 8 in 1992 and on June 5 and June 10 in 1993.

Commencing 20 days after emergence (DAE) two quadrats within each sub plot (one for each wild mustard population) in each of the 4 replicates were destructively sampled. The 10 wild mustard plants in each quadrat were cut at ground level and each plant separated into leaves, support structures (stems and branches), and reproductive (flowers and pods) components. Leaf areas were determined using a leaf area meter[4]. Dry weights were determined after the parts were oven dried at 80 C for 48 hours. Sampling occurred weekly up to 69 DAE, with the final sample taken 95 DAE in 1992, and the final two samples taken 83 and 97 DAE in 1993. The sampling interval was lengthened towards the end of the growing season to facilitate handling and measuring large plants.

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[4]Portable leaf area meter, model LI-3000, Li-Cor Inc., 4421 Superior street, box 4425, Lincoln, NE 68504.

At the final sampling date, four quadrats within each sub plot (two for each wild mustard population) within each of the 4 replicates were harvested. At this time wild mustard plants were mature and beginning to dry down. Sampling occurred just prior to shattering of the lower pods (siliques). Pods were removed from branches and support structures, air dried, and weighed. The pods were then threshed by hand and the seed separated from the chaff using a hand sieve. Total weight and thousand-seed weight were determined for each sample and used to calculate the total number of wild mustard seeds produced. The wheat in these same quadrats was also harvested and grain yield (g m^{-2}) determined after threshing and cleaning.

Statistical procedures. The experimental design was a split-plot. Leaf area and seed return data were subjected to analysis of variance (SAS)[5]. Differences among treatments were not significant when analyzed as a split plot design. Therefore, to obtain additional error degrees of freedom for the main plot treatments the data were re-analyzed as a randomized complete block. The variable 'yearpop' was assigned to a wild mustard population in a particular year. A Bartlett's test for homogeneity of variances was performed to determine if the populations could be pooled between years (Gomez and Gomez 1984). Variances were determined to be homogeneous and the data was pooled. Means were separated statistically using standard errors. Standard errors provide information about the inherent variability of the dataset, valuable information that is not readily apparent when means are presented followed by asterisks, letters or probability

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[5]. SAS, Version 5. 1985. SAS Inst., Inc., Box 8000, Cary, NC 27511-8000.

levels (Morse and Thompson 1981). When comparing two means, three times the numerical value of the standard error is approximately equivalent to a LSD test at the 0.05 level of significance (Morse and Thompson 1981). Therefore, in the Results and Discussion portion of this thesis, significant differences between yearpops will refer to differences exceeding three times the standard error.

Growth curves were fitted to the wild mustard data set for total shoot dry matter by regression procedures (SAS). A quadratic model was used to describe the response of both S and R biotypes. The model fitted was

$$y = a_o + b_1x + c_2x^2$$

where y is wild mustard shoot dry matter (g plant^{-1}), b_o is the intercept, b_1 is the linear regression coefficient, b_2 is the curvilinear coefficient, and x is the number of days after wild mustard emergence. Differences between years, particularly for S shoot dry matter in the 2,4-D treated plots, precluded combining data between years.

CHAPTER 3

RESULTS AND DISCUSSION

3.1 Emergence and Weather.

Soil moisture conditions were satisfactory for wild mustard seed germination and seedling emergence in 1992, and very good in 1993. In 1992, only 19 mm of rain fell during the 40 days following seeding, with the first rain occurring 22 days after planting. In 1993 52 mm of rain fell the week prior to planting, favouring better germination and seedling establishment than in 1992. However, then no rain fell until 24 days after planting when 17 mm fell.

In 1992, precipitation in May and June was 22% and 59% of the long term average precipitation, respectively (TABLE 3-1). However, precipitation in July 1992 was above average: 142% of the long-term average. Soil moisture was not a limiting factor in 1993 as precipitation was normal or above-normal throughout the growing season.

In both years, wild mustard emergence was variable (particularly in 1992) and S plants generally emerged 1 to 3 days before R plants. As a result, S plants were 1 to 3-leaf stages in advance of R plants up to bolting. The S mustard emerged 2 to 3 days before the wheat in 1992 and at the same time as the wheat in 1993. Hence, the R mustard emerged with the wheat in 1992 and 1 to 3 days after the wheat in 1993.

TABLE 3-1. May to August precipitation, and average maximum and minimum daily air temperatures at the University of Manitoba Research Station at Portage la Prairie, Manitoba in 1992 and 1993.

	Precipitation		Temperature			
			Max		Min	
	mm	% of 30-yr mean[1]	C	% of 30-yr mean	C	% of 30-yr mean
1992						
May	12	22	20.7	113	4.9	102
June	44	59	22.0	94	9.0	84
July	109	142	22.1	85	10.8	80
August	50	63	23.2	93	9.9	84
1993						
May	53	98	19.2	105	4.3	90
June	68	91	22.3	95	9.3	87
July	114	148	23.5	90	12.5	93
August	89	113	24.0	96	12.0	102

[1]30yr mean from 1961 to 1990; Environment Canada Climate Center, 266 Graham Ave, Winnipeg, MB.

Between years there were only minor differences in daily maximum temperatures averaged over each month from May to August. However, there were differences between years in minimum temperatures. In July and August 1992 the daily average minimum temperatures were 10.8 and 9.9 C, respectively. In comparison in July and August 1993, average minimum temperatures were 12.5 and 12.0 C. Thus, in 1993 the minimum temperatures were 1.7 and 2.1 C higher in the second half of the growing season than in 1992.

Due to variable wild mustard emergence in 1992, it was decided to delay herbicide application for several days past the time when the largest plants were at the recommended stage of treatments i.e., fourth leaf stage. Wind and rain caused an additional delay of 1.5 weeks. Therefore, spraying was conducted approximately two weeks later than optimum for some of the earliest emerging wild mustard plants. At the time of spraying in 1992, some of the S and R wild mustard plants were at the 10 and 7-10 leaf stages, respectively, and were beginning to bolt (equivalent to Harper and Berkenkamp growth stage (HB) 3.1)(Harper and Berkenkamp 1975). This is past the 2 to 4 leaf stage (equivalent to HB 1.2 to 2.4) which is the recommended growth stage to apply 2,4-D (Man. Dept. Agric. 1991).

3.2 Growth Analysis Results

Leaf area. Leaf areas for S and R wild mustard plants harvested 55 days after emergence (DAE) are presented in TABLE 3-2. Although samples were taken throughout the season, only the data for this one sampling date are discussed. This date was chosen because relationships in leaf area between mustard populations and herbicide treatments were most apparent at this time. By 55 DAE [25 and 34 days after herbicide treatment (DAT) in 1992 and 1993, respectively] herbicide phytotoxicity symptoms were fully expressed. Refer to Table A-3 for all the mean leaf area results for S and R wild mustard.

In the untreated control plots the data were variable from one year to the next. In 1992 R plants had greater leaf area than S plants while the opposite occurred in 1993. Differences between S and R plants in 1993 were statistically significant (p = 0.10) as S plants had a leaf area of 678 cm^2 and R plants, 463 cm^2.

In 1992, the leaf area of S plants treated with 2,4-D at the recommended rate of 420 g a.i. ha^{-1} was 19% of the untreated control. In 1993, at this dosage S plants were completely dead and desiccated by 55 DAE. In comparison, the leaf areas of R plants were 67% and 45% of the untreated control in 1992 and 1993, respectively. Although leaf areas were reduced for both populations, S plants were more sensitive to 2,4-D than the R plants.

TABLE 3-2. Leaf area at 55 days after emergence (DAE) for 1992 and 1993 and standard errors for the pooled data.

Year & Pop	Control treatment	2,4-D g a.i. ha^{-1}			Dicamba g a.i. ha^{-1}		
		105	210	420	75	150	300
LEAF AREA (cm^2)							
92S	453	221	114	84	344	258	222
92R	509	440	398	339	569	343	593
93S	678	65	76	0	564	385	121
93R	463	278	287	207	525	506	472
S. E.	79	67	42	48	96	59	47

The recommended rate of dicamba (300 g a.i. ha^{-1}) reduced the leaf area of S plants to 49% and 18% of the untreated control in 1992 and 1993, respectively. Surprisingly, leaf areas of treated R plants were slightly greater than the leaf areas of corresponding plants in the untreated control plots in both years. These results indicate that both S and R populations of mustard were less sensitive to dicamba than to 2,4-D and confirm the observations of Heap and Morrison (1992) who reported R/S (GR$_{50}$) ratios for dicamba of 100 and 30 in the growth room and field, respectively. By contrast, they reported corresponding R/S (GR$_{50}$) ratios of 18 and 26, for 2,4-D.

Generally, 55 DAE leaf areas of wild mustard plants treated with 2,4-D and dicamba were higher in 1992 than 1993. In 1992, the herbicide treatments were applied 30 DAE compared to 21 DAE in 1993. Hence in 1992, the plants were sampled 25 days after treatment, and in 1993 34 days after treatment. The larger leaf areas observed in 1992 in comparison to 1993 may be ascribed to the shorter time between herbicide application and leaf area assessment. More importantly, the plants were in a more advanced growth stage in 1992 and, therefore, less susceptible to herbicidal injury (see Literature Review). In this experiment phenoxy herbicide injury symptoms were observed on wild mustard plants the day after treatment.

In both years S wild mustard plants emerged before R plants, prompting an instant differential of 1-3 leaf stages until bolting. More importantly the R plants emerged with the wheat in 1992 and 1 to 3 days after the wheat in 1993. Since the R

plants were at a competitive disadvantage to the wheat, relative to S plants, it is not surprising that differences in leaf area between S & R plants in 1993 were statistically significant (p = .10) at 55 DAE.

Leaf area accounts for the number of leaves, as well as the size of each leaf. In 1993 bolting occurred at 41 DAE versus 62 DAE in 1992, at this point the wild mustard populations each had the same number of leaves. The significant difference (p=.10) in leaf area between S and R plants at 55 DAE in 1993 was therefore, a result of the S wild mustard plants having larger leaves and not more leaves than the R wild mustard plants. Clearly, the R wild mustard population was at a competitive disadvantage relative to the S population.

Shoot dry matter accumulation. To facilitate comparisons between wild mustard populations a quadratic function was fitted to the growth analysis data using nonlinear regression procedures (SAS v.5) . Wild mustard growth is indeterminate and the quadratic function provided the best fit for the data.

Susceptible and resistant wild mustard shoot dry matter accumulation, in the absence of herbicide, is presented graphically in FIGURE 3-1. To facilitate comparisons of shoot dry matter accumulation at various sampling dates, means are also presented in TABLE 3-5. The dry weight of S wild mustard increased more quickly than that of R plants from emergence to 69 DAE in 1992, and 83 DAE in 1993. Generally, the difference between S and R plants increased at successive dates. By 69 DAE, the shoot biomass of S plants was two and three
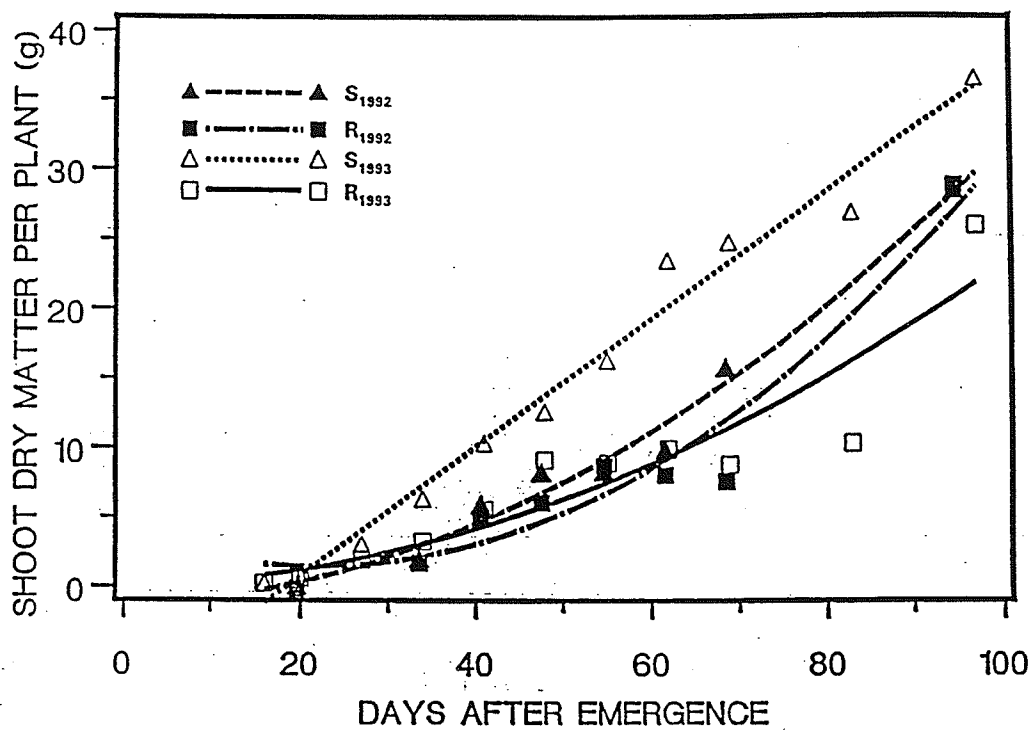
FIGURE 3-1. Shoot dry matter accumulation of S and R wild mustard in the absence of herbicide in wheat. Mean values for each sampling date and the quadratic function are plotted. Refer to TABLES 3-3 and 3-4 for parameter estimates.

TABLE 3-3.   Quadratic equation parameter estimates (standard errors in parentheses) for wild mustard shoot dry matter accumulation in 1992.

g ha^{-1}	a	b	c	R^2
1992 S				
Control	-1.48 (5.59)	0.030 (0.21)	0.0030 (0.0017)	0.71
2,4-D 105	-2.45 (2.39)	0.16 (0.089)	-0.00045 (0.00075)	0.52
2,4-D 210	-0.79 (1.57)	0.076 (0.059)	-0.00029 (0.00051)	0.29
2,4-D 420	-0.42 (1.21)	0.080 (0.045)	-0.00084 (0.00038)	0.21
dicamba 75	-2.88 (3.07)	0.17 (0.11)	-0.00020 (0.00097)	0.55
dicamba 150	-2.01 (1.53)	0.15 (0.056)	-0.00088 (0.00048)	0.37
dicamba 300	-4.17 (1.94)	0.25 (0.072)	-0.0015 (0.00061)	0.52
1992 R				
Control	3.88 (4.34)	-0.21 (0.16)	0.005 (0.0014)	0.80
2,4-D 105	-5.04 (2.11)	0.27 (0.078)	-0.00073 (0.00066)	0.80
2,4-D 210	-2.94 (1.72)	0.16 (0.064)	0.000032 (0.00054)	0.83
2,4-D 420	-4.63 (1.41)	0.28 (0.052)	0.0016 (0.00044)	0.72
dicamba 75	-4.54 (3.41)	0.23 (0.13)	-0.000081 (0.0012)	0.72
dicamba 150	1.03 (4.20)	-0.037 (0.15)	0.0024 (0.0013)	0.64
dicamba 300	-7.70 (2.19)	0.40 (0.081)	-0.0020 (0.00068)	0.78

TABLE 3-4. Quadratic equation parameter estimates (standard errors in parentheses) for wild mustard shoot dry matter accumulation in 1993.

g ha^{-1}	a	b	c	R^2
1993 S				
Control	-8.65 (3.06)	0.47 (0.13)	-0.00015 (0.0012)	0.85
2,4-D 105	4.28 (0.78)	-0.07 (0.028)	0.00029 (0.00022)	0.68
2,4-D 210	4.41 (0.72)	-0.095 (0.025)	0.00050 (0.00020)	0.67
2,4-D 420	4.47 (0.49)	-0.11 (0.017)	0.00067 (0.00014)	0.78
dicamba 75	-3.48 (4.38)	0.21 (0.15)	-0.000045 (0.0012)	0.66
dicamba 150	3.87 (2.38)	-0.078 (0.084)	0.0016 (0.00067)	0.72
dicamba 300	4.18 (1.45)	-0.094 (0.052)	0.0011 (0.00042)	0.38
1993 R				
Control	-0.15 (2.47)	0.025 (0.11)	0.0020 (0.00095)	0.72
2,4-D 105	0.38 (0.51)	0.08 (0.00)	-0.00025 (0.000092)	0.39
2,4-D 210	1.25 (1.72)	0.051 (0.060)	-0.00024 (0.00048)	0.13
2,4-D 420	0.14 (1.32)	0.071 (0.047)	-0.00045 (0.00037)	0.15
dicamba 75	-7.10 (2.75)	0.37 (0.097)	-0.0015 (0.00078)	0.80
dicamba 150	-5.09 (2.79)	0.30 (0.099)	-0.0011 (0.00079)	0.78
dicamba 300	-9.05 (2.75)	0.49 (0.097)	-0.0031 (0.00078)	0.65

times higher than that of R plants in 1992 and 1993, respectively. Apparent fitness differences between S and R mustard populations may be the result of differences between populations, or it may be the result of a fitness cost associated to the auxinic herbicide resistance gene. Despite the cause, its effects of delaying R mustard emergence relative to S, is extremely important. Timing of weed emergence versus the crop and the resulting weed pressure has been studied extensively. Generally, if the weed emerged before the crop, a distinct competitive advantage would be enjoyed by the weed. Moreover, if the weed emerged after the crop the weed's ability to survive and reproduce would be severely hampered. Since the R mustard emerged after the crop in 1993 it may help to explain why it was further behind the S wild mustard population in contrast to the observations made in 1992 when the R wild mustard population emerged with the wheat.

From pod initiation (HB 4.3 - approximately 62 and 48 DAE in 1992 and 1993, respectively) to final harvest, wild mustard shoot dry matter increased approximately three-fold for both populations in both years (TABLE 3-5). However, wild mustard growth (accumulation of shoot biomass) during the interval between the next-to-last and last sampling dates differed between populations. These sampling dates were 69 and 95 DAE in 1992, and 83 and 97 DAE in 1993. Shoot dry matter of the S population during this interval increased by 1.8 and 1.4-fold while dry matter of the R population increased by 3.8 and 2.5-fold, in 1992 and 1993, respectively. The faster growth rate of R plants late in the season resulted in no significant differences in shoot dry matter between S and R populations by final harvest in the absence of herbicide treatment.

TABLE 3-5. Control treatment shoot dry matter accumulation (g plant^{-1}) for 1992 and 1993 and standard errors for the pooled data.

DAE	92 S	92 R	93 S	93 R	S. E.
20	0.1	0.1	0.7	0.5	0.1
27	-	-	3.0	1.9	0.5
34	2.1	2.0	6.3	3.2	0.6
41	6.0	5.3	10.3	5.5	0.9
48	8.3	7.3	12.5	9.0	1.0
55	8.4	8.9	16.2	8.8	1.3
62	9.9	8.3	23.4	9.9	2.9
69	15.8	7.8	24.7	8.7	2.8
83	-	-	26.8	10.2	5.0
95	28.4	29.4	-	-	5.4
97	-	-	36.4	25.8	7.3

The indeterminate nature of wild mustard is reflected by heavy growth up to the end of the growing season. Exhibited by both populations in 1992 and 1993, the late season dry matter accumulation does not conform well to a sigmoidal shaped growth curve, typical of many annual plants. Similar to the wild mustard, dog mustard (*Erucastrum gallicum*) displayed a "*marked increase in shoot dry matter*" from 70 DAE to final harvest (Wall et al 1994). Although the dog mustard was grown on fallow and not under crop competition, this observation is important as it supports observations made in this study.

In *Brassica napus*, Morrison (1987) observed increases in shoot dry matter of 2.2 to 2.4-fold between HB 4.3 and HB 5.3. Tayo and Morgan (1979) attributed large, late season canola dry matter increases to elevated production of total plant photosynthate from canola siliques and developing seed. They postulated that new green pods and seed rich in photosynthetically active chlorophyll resulted in exponential dry matter accumulation in canola following pod initiation. Comparisons of *Brassica napus* and *Sinapis arvensis* are appropriate due to similarities in morphological structure and growth patterns in similar environments (Blackshaw and Dekker 1988).

Partitioning. For the control treatment, above-ground dry matter was partitioned into the various components on a percentage basis (i.e., % leaf, % support, and % pod) (FIGURE 3-2). Leaves initially comprised 100% of above-ground dry matter, but by final harvest the leaves had fully senesced and fallen from the
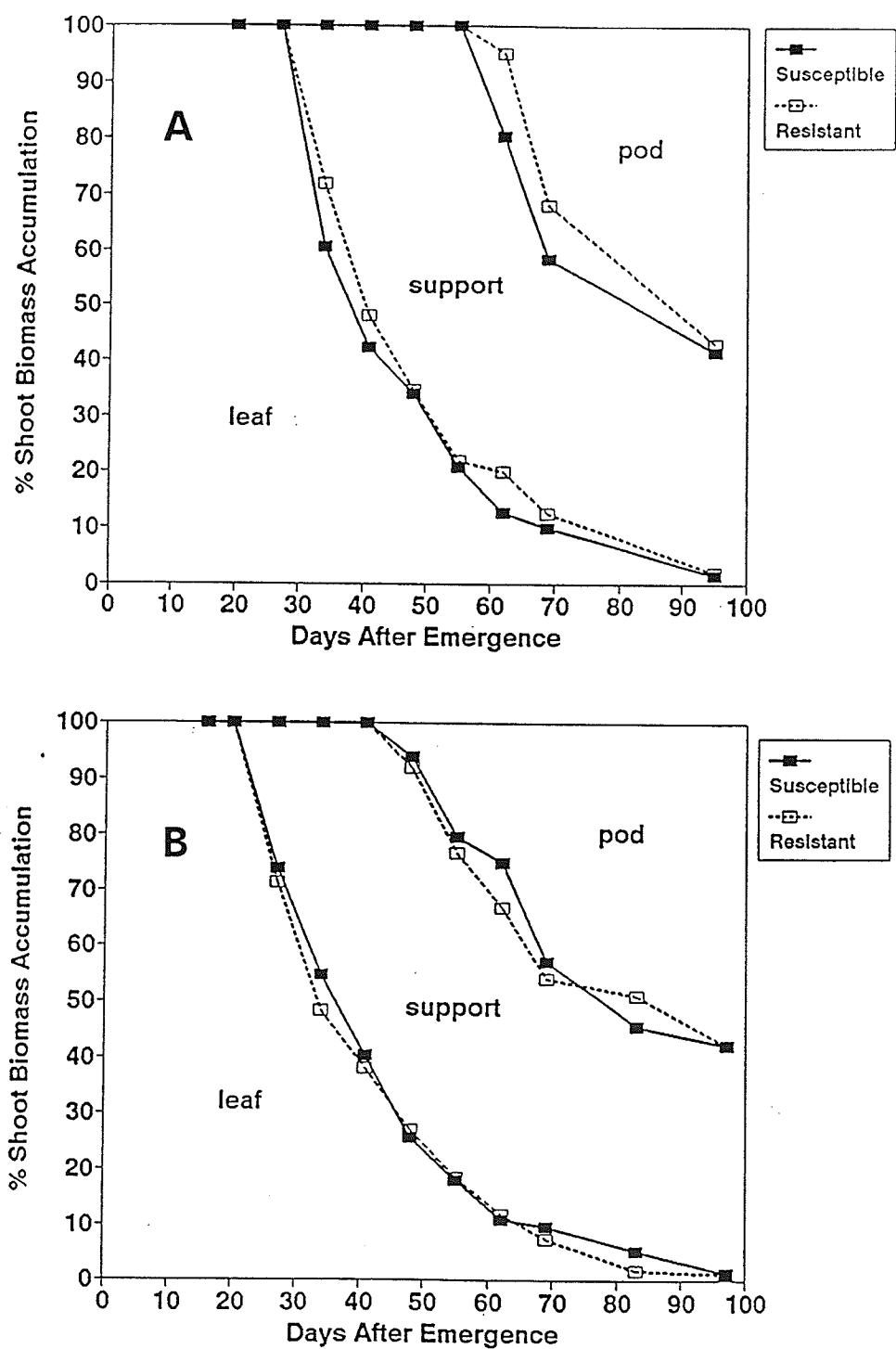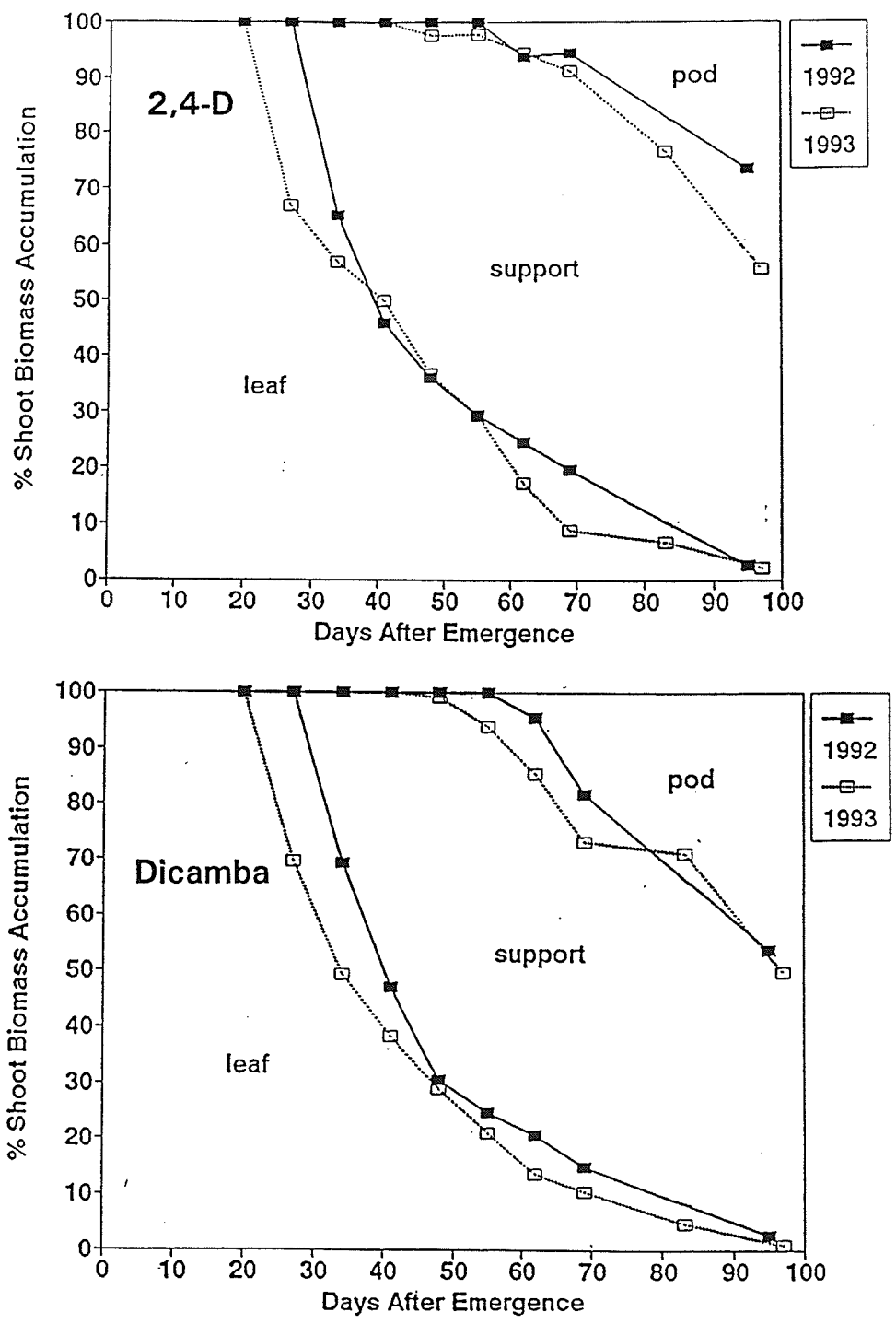
FIGURE 3-2. Control treatment wild mustard S and R shoot biomass partitioning for 1992 (A) and 1993 (B). Plants are divided into percent leaf, support and pod.

FIGURE 3-3. Shoot biomass partitioning of S and R wild mustard plants treated with 420 g ha^{-1} 2,4-D and 300 g ha^{-1} dicamba in 1992 and 1993. Plants are divided into percent leaf, support and pod.

stems. The central stem did not become a distinct structure until elongation or bolting occurred approximately 25 to 30 DAE. Support structures then developed rapidly and comprised the majority of above-ground biomass for a period of approximately 40 to 75 DAE. Even at final harvest, support structures comprised approximately 40 to 45% of total shoot dry matter. Pods were distinguished at sampling dates 62 and 48 DAE in 1992 and 1993, respectively. By final harvest, pods and their contents comprised over 50% of total shoot dry matter.

Interestingly, although actual values (g plant^{-1}) for shoot dry matter in the absence of herbicide differed substantially between populations at intermediate sampling dates (TABLE 3-5), biomass partitioning expressed as a percentage basis differed only slightly between populations and years (FIGURE 3-2). Biomass partitioning differences between populations were not substantial, in either year. However, initiation of support structures and pods occurred approximately 1 and 2 weeks later, respectively, in 1992 as compared to 1993. The slower rate of growth and development in 1992 as compared to 1993 probably was due to drier conditions, early in the 92 season. Lower minimum temperatures in the second half of the 92 summer also contributed to slower wild mustard growth rates (TABLE 3-1).

Shoot dry matter accumulation of S and R wild mustard plants treated with 2,4-D is presented in FIGURE 3-4. The recommended rate of 2,4-D (420 g a.i. ha^{-1}) reduced shoot dry matter of wild mustard S and R populations substantially. S mustard was completely dead and desiccated by 69 and 55 DAE in 1992 and 1993, respectively. At final harvest, the shoot dry matter of R plants was reduced
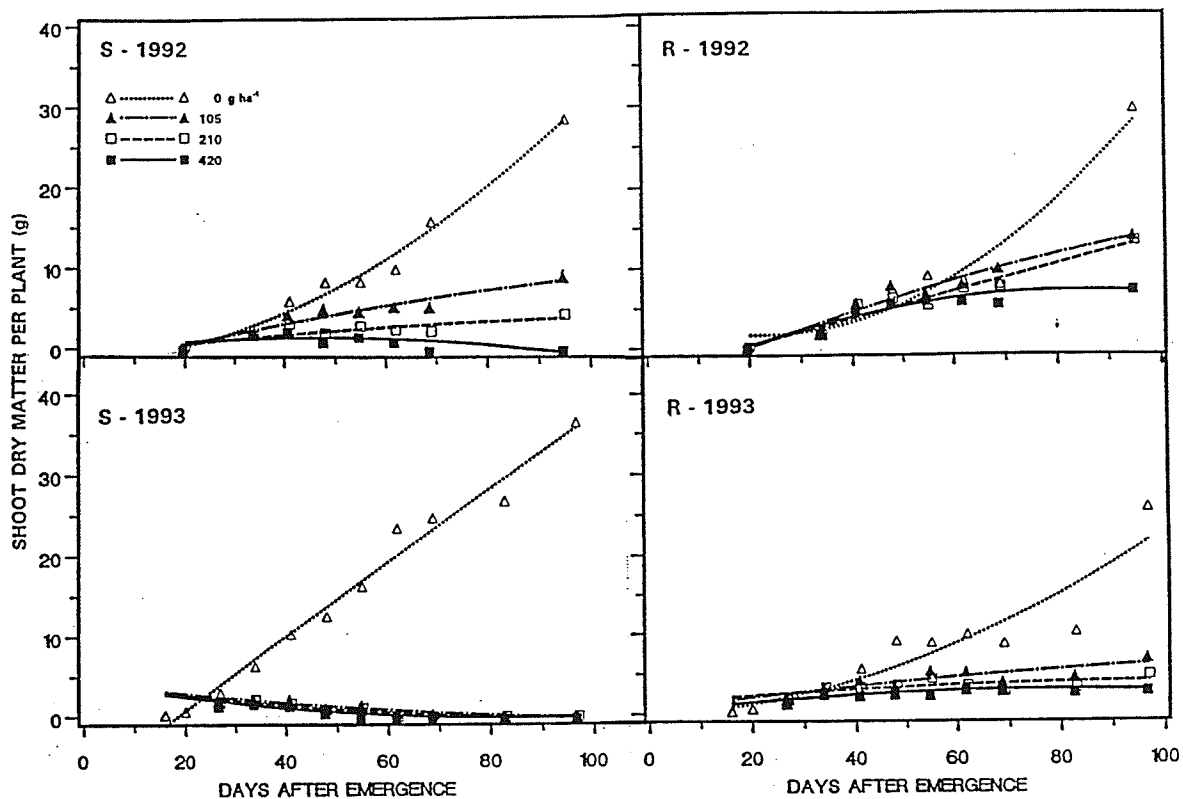
FIGURE 3-4. Shoot dry matter accumulation of S and R wild mustard treated with 2,4-D (0, 105, 210, and 420 g ha⁻¹) in wheat. Mean values for each sampling date and the quadratic function are plotted. Refer to TABLES 3-3 and 3-4 for parameter estimates.

to 25% and 11% of R plants in the untreated control plots. Actual values (mean ± S.E. g plant[-1]) were 7.36 ± 0.64 and 2.95 ± 1.06 g per plant in 1992 and 1993, respectively.

The data indicate that S wild mustard is satisfactorily controlled under field conditions by the recommended dosage of 2,4-D. Furthermore, although R plants exhibit a functional level of resistance to the recommended dosage of 2,4-D under field conditions, growth (as measured by accumulation of shoot dry matter) is greatly inhibited by the herbicide. In 1993, the herbicide was applied at an earlier growth stage than in 1992. As a result 2,4-D was more injurious to both S and R plants as indicated by complete control of S plants in 1993 at all three dosages of 2,4-D and by the R shoot dry matter values (g plant[-1]) at 420 g ha[-1].
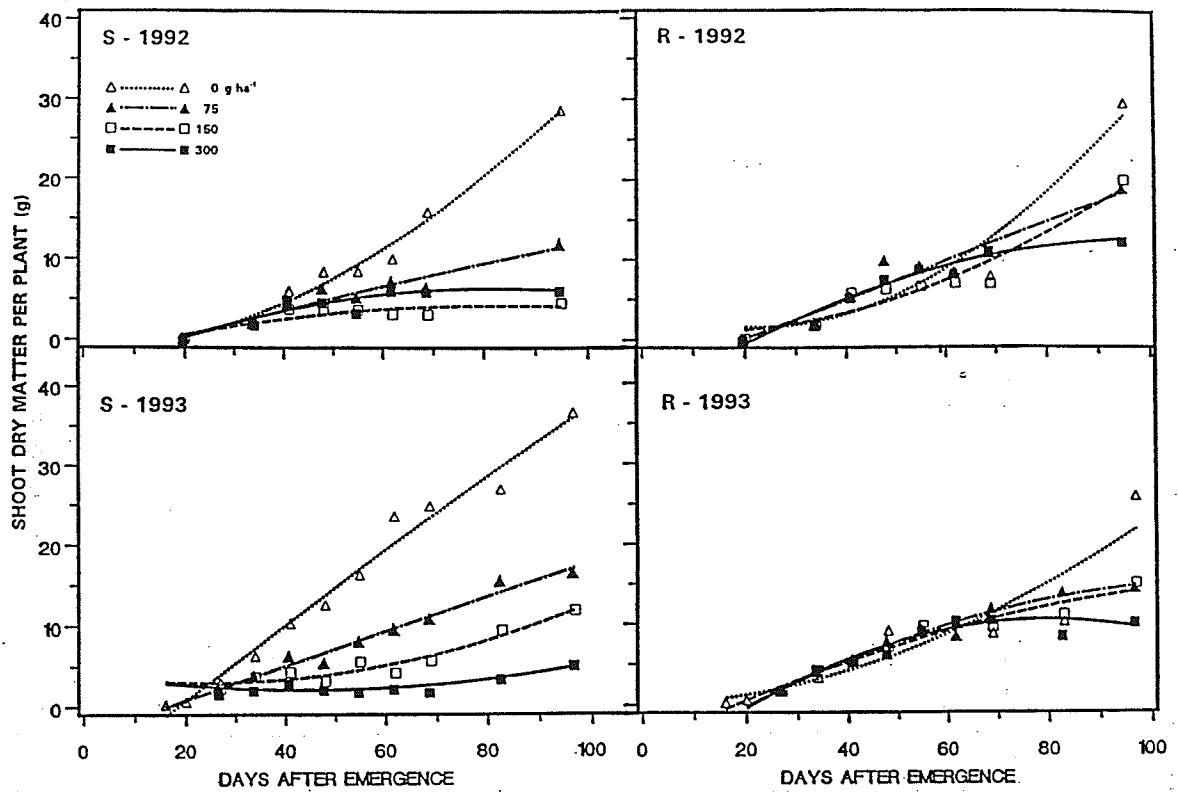
In general, there were only minor differences between years in shoot dry matter partitioning of R wild mustard plants treated with 420 g a.i. ha[-1] of 2,4-D (FIGURE 3-3). As with R plants in the untreated plots, support structures were initiated one week later in 1992 (27-34 DAE) as compared to 1993 (20-27 DAE). However, at final harvest the percentage of shoot biomass partitioned into pods and their contents differed somewhat between years. Even though R plants accumulated twice as much actual shoot biomass (g plant[-1]) in 1992 as compared to 1993, a smaller percentage of total shoot biomass was comprised of pods and their contents in 1992 (approximately 25% versus 40%). This may be an indication of the plasticity of wild mustard growth and the result of plant response to herbicide

stress, as accumulation of shoot biomass by R plants in 1993 was inhibited to a greater extent than in 1992 (i.e., greater herbicidal activity in 1993).

In comparison to R plants in the untreated plots, R plants treated with 2,4-D at 420 g a.i. ha^{-1} partitioned a smaller percentage of total shoot biomass into pods and their contents. This occurred in both 1992 and 1993 (FIGURES 3-2 and 3-3).

Shoot dry matter accumulation patterns of S and R wild mustard plants treated with intermediate dosages of 2,4-D (105 and 210 g ha^{-1}) were intermediate between those for the untreated check and the recommended dosage (FIGURE 3-4). In 1992, when 2,4-D activity was less, a response to dosage was noted. However in 1993, when herbicidal activity was greater, dry matter accumulation for both S and R populations at the intermediate dosages was almost the same as at the recommended rate. In fact in 1993, S wild mustard was killed by all three dosages of 2,4-D. Biomass partitioning for the intermediate rates is not presented.

Shoot dry matter accumulation of S and R wild mustard plants treated with dicamba is presented in FIGURE 3-5. The recommended dose of dicamba (300 g ha^{-1}) reduced shoot dry matter of S and R populations substantially, but not to the same extent as the recommended dosage of 2,4-D. Unlike 2,4-D, dicamba did not satisfactorily control S wild mustard. At final harvest, the top-growth of S plants was reduced to 22% and 16% of the untreated control in 1992 and 1993, respectively, while the top-growth of R plants was reduced to 42% and 40%. Actual values (mean ± S.E. g plant^{-1}) for S shoot dry matter were 6.18 ± 1.74

FIGURE 3-5. Shoot dry matter accumulation of S and R wild mustard treated with dicamba (0, 75, 150, and 300 g ha⁻¹) in wheat. Mean values for each sampling date and the quadratic function are plotted. Refer to TABLES 3-3 and 3-4 for parameter estimates.

and 5.82 ± 1.54, and for R shoot dry matter 12.30 ± 1.74 and 10.30 ± 1.54 in 1992 and 1993, respectively.

There were only minor differences between years in shoot dry matter partitioning by R wild mustard plants treated with 300 g ha^{-1} of dicamba (FIGURE 3-3). Similar to R plants in the untreated control and 2,4-D (recommended dosage) treated R plants, support structures were initiated one week later in 1992 (27-34 DAE) as compared to 1993 (20-27 DAE). Unlike 2,4-D (420 g ha^{-1}) treated R plants, by final harvest there were no differences between years in biomass partitioning of R plants treated with dicamba. Similar to R plants treated with 2,4-D and in comparison to R plants in the untreated control, R plants treated with dicamba partitioned a smaller percentage of their total shoot biomass into pods and their contents (FIGURES 3-2 and 3-3). However, this response to herbicide stress was not as accentuated with dicamba as compared to 2,4-D.

Shoot dry matter of S and R wild mustard plants treated with 75 and 150 g ha^{-1} dicamba generally were intermediate between those for the untreated check and the recommended dosage (FIGURE 3-5). The only exception was in 1992 when at final harvest 150 g ha^{-1} of dicamba reduced the shoot dry matter of S plants to 4.50 ± 6.00 g plant^{-1} in comparison to 6.19 ± 1.74 g plant^{-1} when dicamba was applied at 300 g ha^{-1}.

Wheat yield. In the untreated S wild mustard plots wheat yields were 116 and 100 g m^{-2} in 1992 and 1993, respectively (TABLE 3-6). In comparison, wheat

TABLE 3-6. Wheat yield (g m^{-2}) for 1992 and 1993 and standard errors for the pooled data.

Year Pop	92S	92R	93S	93R	S.E.
Herb. trt. **g ha^{-1}**					
Control trt.	116	215	100	143	23
2,4-D 105	289	310	171	152	28
2,4-D 210	326	271	159	154	22
2,4-D 420	312	287	139	198	21
dicamba 75	199	211	140	132	22
dicamba 150	196	200	181	125	17
dicamba 300	207	240	113	102	11
S.E.	17	17	17	15	

yields in the untreated R wild mustard plots were 215 and 143 g m^{-2}. Wheat yield differences between untreated S and R wild mustard populations were substantial in both years and statistically significant in 1992. The lower wheat yields in S plots probably reflect the greater competitiveness of S wild mustard throughout most of the growing season (competitiveness as measured by shoot biomass accumulation). In the untreated control in 1992, at 69 DAE S wild mustard shoot biomass was significantly greater than R (15.8 vs. 7.8 g plant^{-1}) (TABLE 3-5). Similarly, in 1993 S wild mustard shoot biomass was significantly greater than R for most of the growing season. However in 1993, the wheat crop was severely infected by several wheat diseases including tan spot and fusarium head blight which reduced yield potential and probably partially masked some of the effects of wild mustard competition on wheat yield.

In both years, as previously discussed, the recommended dosage of 2,4-D greatly reduced S and R wild mustard growth and competitiveness (as measured by shoot biomass accumulation) in comparison to the untreated control. Reduced wild mustard competitiveness corresponded with increased wheat yields and the increase was statistically significant in three of four instances (S92, R92, R93) (TABLE 3-6). The recommended dosage of dicamba did not reduce wild mustard growth and competitiveness to the same extent as 2,4-D, and in only one instance (S92) were wheat yields in these plots significantly higher than the untreated control. Wheat yields in plots treated with intermediate dosages of 2,4-D or dicamba were somewhat variable and did not consistently follow a dose-response pattern (i.e., an expected dose-response pattern = higher herbicide dosages

corresponding to reduced wild mustard growth and competitiveness and therefore increased wheat yields). However, wheat yields generally were higher in 2,4-D treated plots as compared to dicamba treated plots - undoubtedly resulting from better wild mustard control.

Seed return. Susceptible wild mustard seed yield in the untreated control plots was 2730 and 3120 seeds per plant in 1992 and 1993, respectively. The corresponding seed return from R plants was 2925 and 2520 seeds per plant, respectively (TABLE 3-7). The pooled S.E. of the mean seed yield per plant is 570. Therefore, no significant differences in seed yield occurred between populations or years in the untreated plots. Hence, in the absence of herbicide the relative fitness based on seed return is close to 1. Notwithstanding, that there were apparent fitness differences observed between S and R plants during the vegetative growth stages. In some years, the late season growth surge of the R wild mustard may jeopardize seed production and quality. After all, the wheat growing season dictates when the crop, including weeds are harvested. If the wheat matures quickly and is immediately swathed or harvested, than so is the wild mustard (ready or not). The R wild mustard might not have completed pod fill, and the pods that were filled may have a higher proportion of immature seed in contrast to the earlier pod filling S wild mustard. Obviously, over time there would be years where this scenario would hold true. Hence, decreasing the R wild mustard's ecological fitness relative to S. In this study the wild mustard was harvested when the wheat was ready to swath. In 1993, the wheat ripened early due to heavy disease pressure. Not surprisingly, 1993 was also the year that the

TABLE 3-7. Seed return data (seed no. plant^{-1}) for 1992 and 1993 and standard errors for the pooled data.

Year & Pop	Control treatment	2,4-D g a.i. ha^{-1}			dicamba g a.i. ha^{-1}		
		105	210	420	75	150	300
92S	2730	635	245	0	895	285	555
92R	2925	1185	680	450	1455	1480	980
93S	3120	0	0	0	1530	1160	405
93R	2520	530	255	285	1315	1220	840
S. E.	570	180	130	95	340	285	140

S wild mustard yielded substantially more seed than R. Since seed viability was not measured in this study, we cannot conclude for certain that the R seed was more immature than the S wild mustard. However, it would be a reasonable assumption.

Susceptible wild mustard plants treated with 420 g ha^{-1} 2,4-D were killed in both years and did not produce any seed (TABLE 3-7). In comparison, R seed return was reduced to 15% and 11% of the untreated control in 1992 and 1993, respectively. Although R seed return was decreased substantially, actual R seed rain calculated on a square meter basis (20 plants m^{-2}) was 9000 seeds m^{-2} in 1992 and 5700 seeds m^{-2} in 1993. Practically, this is a very high seed return, as the highest wild mustard density reported in a weed survey conducted in Manitoba wheat fields during the early 1980's was 53 plants m^{-2} (Thomas and Wise 1988).

Differences in seed return between S and R plants at 420 g ha^{-1} 2,4-D for 1992 and 1993 are considered significant, biologically, because S plants produced no seed (Dr. Roger Rimmer, pers. comm.)[6].

Wild mustard seed return in plots treated with 105 and 210 g ha^{-1} 2,4-D generally was less than from untreated control plots but more than from those treated at the recommended dosage (TABLE 3-7). Exceptions to this occurred in 1993 when

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[6]. Professor of oilseed pathology and biometrics, University of Manitoba, R3T 2N2.

S plants were killed at all dosages of 2,4-D. Also in 1993, R plants treated with 210 g ha^{-1} of 2,4-D set slightly less seed than R plants treated with 420 g ha^{-1} 2,4-D.

3.3 Determination of Fitness

Using fecundity as an ultimate measure of fitness, the results on seed return can be incorporated into predictive models which indicate the rate of enrichment of R individuals within a weed population. Under herbicide 'on' conditions the definition of selection intensity (SI) is 1.0 minus the relative fitness of S plants. Since the relative fitness of S plants approaches 0 when 2,4-D is used, the selection intensity imposed by the herbicide is extremely high, approaching a value of 1.0. In other words, the herbicide is so highly efficacious that only resistant individuals remain to set seed. Since selection intensity is "*the most influential factor affecting the rate of R enrichment*" (Gressel and Segel 1978, 1982, 1990) one could predict on the basis of these results that resistance evolution would occur rapidly.

Resistance models calculate the relative frequencies of S and R individuals within a population. However, actual weed population densities and seed return numbers are important in the field. For example, the herbicide selection intensity imposed by the recommended dosage of 2,4-D would still be 1.0 even if R seed return was only 25 seeds m^{-2}. Obviously, this level of seed set would not result in the same

damaging effect on crop production as 9000 seeds m^{-2}. Therefore, the high seed return of R plants, in combination with the high selection intensity imposed by 2,4-D, would result in a very rapid enrichment of the R biotype within the population and a serious weed control problem.

Seed return of S plants treated with the recommended dosage of dicamba was reduced to 20% and 13% of the untreated control in 1992 and 1993, respectively (TABLE 3-7). In comparison, R seed return was reduced to 33% of the untreated control in both years. As with 2,4-D, wild mustard seed return at the lower two dosages of dicamba generally was intermediate between the untreated control and the recommended dosage. However, the seed return of S plants treated with 150 g ha^{-1} of dicamba in 1992 appears to be somewhat anomalous, as it does not conform to this generality.

Although the seed return and shoot dry matter of S plants was reduced substantially at the recommended dosage of dicamba, actual seed rain calculated on a square meter basis (20 plants m^{-2}) was 11100 seed m^{-2} and 8100 seeds m^{-2} in 1992 and 1993, respectively. R plants set nearly twice as many seeds as S plants, producing 19600 seeds m^{-2} and 16800 seeds m^{-2}, respectively in the two years. The recommended dosage of dicamba did not provide satisfactory control of either wild mustard population in either year.

Since dicamba did not adequately control S plants, relative differences in seed rain between S and R plants were small. The relative fitness based on seed return of

S plants treated with 300 g ha^{-1} dicamba was 0.57 and 0.48 in 1992 and 1993, respectively (TABLE 3-8). In comparison, the relative fitness of S plants treated with the recommended dosage of 2,4-D was 0 in both years.

The selection intensity (SI) values for the recommended dosage of dicamba are 0.43 and 0.52 in 1992 and 1993, respectively. In terms of evolutionary ecology, these SIs for R individuals are very high. However, in terms of the development of herbicide resistance, they are low as the models indicate that it would take years of continuous herbicide exposure for R individuals to predominate in the population. Also, dicamba alone is a relatively ineffective herbicide on wild mustard and probably would not be widely used frequently in a rotation to control this weed.

TABLE 3-8. Wild mustard seed return (no. m^{-2}, based on 20 plants m^{-2}) for dosages of 2,4-D and dicamba, and associated relative fitness of S, selection intensity, and selection pressure[a].

Herbicide Treatment g ai ha^{-1}	Seed Return m^{-2}			Relative Fitness of S	Selection	
	R	S	S.E.		Intensity	Pressure
1992						
control	58500	54600	3670			
2,4-D 105	23700	12700	6870	0.54	0.46	1.74
2,4-D 210	13600	4900	6000	0.36	0.64	2.59
2,4-D 420	9000	0	3700	0.00	1.00	153
dicamba 75	29100	17900	4800	0.62	0.38	1.52
dicamba 150	29600	5700	10200	0.19	0.81	4.84
dicamba 300	19600	11100	3900	0.57	0.43	1.65
1993						
control	50400	62400	11000			
2,4-D 105	10600	0	3400	0.00	1.00	210
2,4-D 210	5100	0	1300	0.00	1.00	101
2,4-D 420	5700	0	1570	0.00	1.00	113
dicamba 75	26300	30600	10900	1.16	-0.16	1.06
dicamba 150	24400	23200	5300	0.95	0.05	1.30
dicamba 300	16800	8100	3000	0.48	0.52	2.57

[a]In the presence of herbicide, the Relative Fitness of S plants is calculated as S seed return/R seed return, and the Selection Intensity is 1.0 minus the relative fitness of S. Selection Pressure is calculated as [(R seed return$_{treated}$/R seed return$_{untreated}$) / (S seed return$_{treated}$/S seed return$_{untreated}$)]. However, if S seed return$_{treated}$ = 0, the selection pressure value is undefined. Therefore, for the 420 g ha^{-1} 2,4-D treatment in 1992 and the 420, 210, and 105 g ha^{-1} 2,4-D treatments in 1993, the value for S seed return$_{treated}$ was assigned 0.1% of S seed return$_{untreated}$.

3.4 Modelling Herbicide Resistance

Relative fitness herbicide 'off'. As previously mentioned, the results indicate that in terms of seed yield there were no significant differences between populations or years. In 1992, the actual mean seed return of S and R populations were quite similar, therefore for the purposes of fitting the data to a predictive model (Gressel and Segel 1990a), a value of 1.0 was assigned to the parameter for fitness of R plants under herbicide 'off' conditions ($f_{off (1992)} = 1.00$). Although the differences were not statistically significant in 1993, R plants set only 81% as much seed as S plants. Therefore in presenting two possible scenarios, the fitness of R in 1993 under herbicide 'off' conditions was assigned a value of 0.81 ($f_{off (1993)} = 0.81$).

Relative fitness herbicide 'on'. Selection pressure based on seed return was calculated prior to inserting these values in Gressel and Segel's (1990) herbicide rotational model. Selection pressure is calculated as [(R seed return$_{treated}$/R seed return$_{untreated}$) / (S seed return$_{treated}$/S seed return$_{untreated}$)]. However, selection pressure values for the recommended dosage of 2,4-D in both years (and for the intermediate 2,4-D treatments in 1993) are undefined due to the fact that the S plants were completely killed. In order to utilize the model it was necessary to assign a seed return of 0.1% to the susceptible untreated control plants (equivalent to 99.9% effective kill).

Calculated selection pressures were very high for the recommended dosage of

2,4-D (420 g ha^{-1}) in both years; 153 and 113 in 1992 and 1993, respectively (TABLE 3-8). In contrast, selection pressures were low for the recommended dosage of dicamba (300 g ha^{-1}) in both years; 1.65 and 2.57 in 1992 and 1993, respectively.

Calculated selection pressures for intermediate dosages of 2,4-D were also low in 1992 when S plants were not completely controlled, and high in 1993 when S plants were killed at all three dosages of 2,4-D. Selection pressures for 2,4-D in 1993 did not follow a dose-response pattern, as the calculation of selection pressure was influenced only by R seed return and since a small constant value was assigned to S seed return over all three dosages. Selection pressures for intermediate dosages of dicamba were low in both years and followed a dose-response pattern in 1993, but not in 1992. In 1992, S plants treated with 150 g ha^{-1} of dicamba set one-half as much seed as S plants treated with 300 g ha^{-1}. This anomaly results in selection pressure values that do not follow a typical dose-response pattern.

Fraction of seeds leaving the seed bank. In applying the model, s, which is the fraction of seeds leaving the seedbank each year, was assigned a value of 0.228. This value was based on Edwards (1980) study, as discussed in the literature review (Chapter 1.1).

Number of years, herbicide on (p), herbicide off (q). The p 'on' years and the q 'off' years can occur in any order during the p + q year period specified. A typical

herbicide rotation pattern for phenoxy herbicide usage in Manitoba might be 2 years 'on' (phenoxy-herbicide applied) and 1 year 'off' (no phenoxy herbicide). This is an approximation, based on extensive use of phenoxy herbicides in Manitoba cereal crops since the late 1950's.

Initial frequency of R plants. Auxin-type herbicides are purported to have multiple modes/sites of action (Gressel and Segel 1982). It is generally thought by molecular biologists that a single gene encodes for a single specific enzyme or protein. Thus, if one believes that auxin-type herbicides have multiple target sites, mutations at several gene loci would be necessary for the expression of herbicide resistance. The initial frequency of resistant individuals would be the product of the probability for each mutation (or the sum of the exponents). Theoretically, the probability of multiple mutations conferring resistance is much lower than the probability of one mutation. This hypothesis may explain the paucity of weed populations resistant to auxin-type herbicides even after many years of worldwide use (Gressel and Segel 1990). Alternatively, auxin-type herbicides may act at a single site within the plant. In this case, the scarcity of phenoxy-resistant weed populations may be a result of mutations conferring resistance either being lethal or occurring at very low frequencies (Jasieniuk et al. 1995).

The development of phenoxy-resistance within a previously unexposed wild mustard population was modeled using various rotational scenarios and three values (10^{-6}, 10^{-9}, 10^{-30}) for the initial frequency of phenoxy-resistant individuals (TABLE 3-9). The value of 10^{-6} represents an estimate of the initial frequency of

TABLE 3-9. Model simulation #1. Predicted proportions of R wild mustard in a previously unexposed population when treated with 2,4-D (420 g ha^{-1}) two out of every three years. Calculated using Gressel and Segel's (1990) herbicide rotational model using selection pressure and fitness parameters determined in this study in 1992 and 1993.

	Initial Frequency of R Plants		
	10^{-6}	10^{-9}	10^{-30}
	proportion of R individuals		
1992[a]			
2 years on 1 year off	0.001	0.000	0.000
4 years on 2 years off	1.000	0.002	0.000
6 years on 3 years off	1.000	1.000	0.000
18 years on 9 years off	1.000	1.000	0.010
20 years on 10 years off	1.000	1.000	1.000
1993[b]			
2 years on 1 year off	0.000	0.000	0.000
4 years on 2 years off	0.457	0.000	0.000
6 years on 3 years off	1.000	0.309	0.000
8 years on 4 years off	1.000	1.000	0.000
20 years on 10 years off	1.000	1.000	0.020
22 years on 11 years off	1.000	1.000	1.000

[a]1992 results: Herbicide selection pressure = 154, R relative fitness herbicide off = 1.0, fraction of seeds leaving the seed bank = 0.228 (both years).

[b]1993 results: Herbicide selection pressure = 113, R relative fitness herbicide off = 0.81.

R individuals within a population if the inheritance of resistance is a monogenic dominant trait (Gressel and Segel 1982).

Given the selection pressure, relative fitness, and seedbank mortality values listed in TABLE 3-9 and initial R plant frequencies of 10^{-6}, 10^{-9} and 10^{-30}, the model predicts the proportion of R individuals in the population will approach 1.0 (completely resistant population) at 6 and 9, 9 and 12, and 30 and 33 years, using 1992 and 1993 data, respectively. As expected, resistance is predicted to occur more quickly using the greater selection pressure and relative fitness values from 1992 as compared to 1993.

Gressel and Segel (1982) stated that most farmers will notice resistant weeds within a field when the frequency of resistant individuals is between 10 and 30%. It is notable that the model predicts a very rapid increase (exponential increase) in R individuals after the frequency has reached 1 in 1000. In all scenarios listed in TABLE 3-9, after the frequency of R individuals has reached at least 1 in 1000 the model predicts the population will be 100% resistant after an additional two years of herbicide selection. This is a result of the very high selection pressures imposed by 2,4-D at the recommended rates as observed in both 1992 and 1993.

As indicated by Gressel and Segel (1990), selection pressure is a more important parameter than fitness of R individuals in the absence of herbicide (f_{off}) in influencing the enrichment of R individuals within a population. Only a minor decrease in the predicted proportion of R individuals occurs in one scenario when

f_{off} is varied from 1.0 to 0.81 (Compare values in TABLES 3-9 and 3-10). In contrast, when f_{off} is held constant at 1.0 and selection pressure is decreased from 154.3 to 113.2, substantial decreases in the proportion of R individuals in all three scenarios occurs. In other words, with lower selection pressure additional years of herbicide treatment can occur before the population becomes 100% resistant. However, this strategy may not be very effective in general in delaying or avoiding the occurrence of herbicide resistance since most modern herbicides impose a very high selection pressure on target weeds (typically >95% effective kill). Furthermore, circumstantial evidence to date indicates that the initial frequency of resistant individuals in most weed populations probably is between 10^{-6} and 10^{-9}, not 10^{-30}, and minor decreases in selection pressure or fitness (f_{off}) will not substantially delay the development of resistance (Jasieniuk et al 1994a).

The model indicates that if the initial frequency of R individuals was one in a million (10^{-6}), resistance to 2,4-D would occur very quickly - within 6 and 9 years given the 1992 and 1993 parameter values, respectively (TABLE 3-9). Given the rarity of auxin-type herbicide resistance and considering the extensive usage of 2,4-D and other auxin-type herbicides, this is not a reasonable prediction. If the initial frequency of R individuals was 10^{-9}, the model predicts resistance in 9 to 12 years. Again, this is an unreasonable result. A more realistic estimate of the development of resistance results if the initial frequency of R individuals is set at 10^{-30}. Then the proportion of R individuals within the field would approach 1.0 after 30 years (20 years on, 10 years off). This is a more realistic scenario given the extensive use of auxin-type herbicides and the rarity of phenoxy herbicide resistance.

TABLE 3-10. Model simulation #2. Predicted proportions of R wild mustard in a previously unexposed population when treated with 2,4-D (420 g ha^{-1}) two out of every three years. Calculated using Gressel and Segel's (1990) herbicide rotational model using selection pressure and fitness parameters indicated below.

	Initial Frequency of R Plants		
	10^{-6}	10^{-9}	10^{-30}
	proportion of R individuals		
f_{off} = 0.81, other parameters same as 1992 (TABLE 3-8)[a].			
2 years on 1 year off	0.001	0.000	0.000
4 years on 2 years off	1.000	0.002	0.000
6 years on 3 years off	1.000	1.000	0.000
18 years on 9 years off	1.000	1.000	0.007
20 years on 10 years off	1.000	1.000	1.000
Selection pressure = 113, other parameters same as 1992 (TABLE 3-8)[b].			
2 years on 1 year off	0.000	0.000	0.000
4 years on 2 years off	0.499	0.000	0.000
6 years on 3 years off	1.000	1.000	0.000
18 years on 9 years off	1.000	1.000	0.000
20 years on 10 years off	1.000	1.000	0.310

[a]Herbicide selection pressure = 154, R relative fitness herbicide off = 0.81, fraction of seeds leaving the seed bank = 0.228 (both years).

[b]Herbicide selection pressure = 113, R relative fitness herbicide off = 1.0.

Considering that phenoxy herbicide resistance has occurred in only one instance and in only one weed species across the Prairies after 20 to 30 years of selection pressure (herbicide on), the initial frequency of resistant individuals may be even lower than 10^{-30}. Thus, the mutation conferring resistance to auxin-type herbicides in broadleaf weeds in general and wild mustard in particular is probably quite rare.

3.5 General Summary and Conclusions

Fitness in the absence of herbicide. Although planted at the same time the R wild mustard emerged 1 to 3 days after the S wild mustard. This conferred a fitness disadvantage on R plants which was especially costly in 1993 when the wheat crop emerged at the same time as the S wild mustard population but 1 to 3 days before the R population. Except at the last sampling dates, the S wild mustard produced substantially more dry matter than the R wild mustard throughout the growing season. Moreover, the S wild mustard had a more detrimental effect on wheat yield than R mustard. Taken together, this evidence suggests that R plants may be generally less competitive (and less fit) than S ones.

Pods and their contents comprised over 50% of wild mustard total shoot dry matter by the final harvest date (FIGURE 3-2). Both populations nearly tripled their dry matter from pod initiation to final harvest in both 1992 and 1993. The R population accumulated most of this dry matter during the last two weeks of the growing season. Curiously, substantial differences in shoot dry matter partitioning did not exist between populations. Therefore, during the last weeks of the

growing season the R wild mustard had a higher photosynthetic net assimilation rate than the S wild mustard. The R wild mustard plants had small leaves and were probably light starved from competing with the taller wheat plants. Late in the season when the wheat leaves were fully seneseced, R wild mustard plants would have the opportunity to intercept more sunlight, thus increasing their net assimilation rate. The biomass accumulation rate for the R wild mustard population was at its maximum between the last and second last sampling dates in both years. The S wild mustard plants emerged either with or ahead of the wheat and were taller and had larger leaves than the R wild mustard. Towards the end of the growing season most of the S wild mustard plants were approaching maturity. Hence at this stage the plants were accumulating little additional biomass.

Practically, farmers are primarily focused on harvesting their crops when they (not the weeds) are mature. The crop, is harvested at the appropriate time irrespective of the growth stage of any weeds. Because of this fact, the experiment was stopped when the wheat was ready to swath (approximately 14% moisture). In 1993, the wheat was under heavy disease pressure and ripened quickly. The greatest difference in total biomass accumulation and seed rain between the S and R wild mustard populations was observed in 1993. The R wild mustard population may not have reached full maturity by the time the crop was harvested.

Peniuk (1994) reported that the seed of phenoxy herbicide resistant wild mustard was shrivelled and smaller than susceptible wild mustard seeds. Although there may be many contributing factors, shrivelled wild mustard seed may be the result

of pre-mature ripening and may indicate poor or sub-standard seed quality. The late season growth of the R wild mustard might have created a seed rain composed of non viable and/or immature seeds. Over time, this would reflect a substantial ecological advantage for the S wild mustard population in contrast to the R one.

Although there was no apparent fitness cost observed between the S and R wild mustard populations in terms of fecundity, there is strong evidence to suggest that the R wild mustard population is substantially less fit than the S wild mustard population when in competition with wheat. The S wild mustard germinated more quickly and competed more effectively with the wheat crop than the R wild mustard. Moreover, the late season growth of the R wild mustard population may jeopardize seed production and quality if harvested too early or exposed to an early season frost. Seed viability and seed chlorophyll percentages were not determined in this study. These observations would have provided a higher degree of precision to the general assessment of relative ecological fitness between wild mustard populations. However, even without these results, it is pausible to assume that in some years the R wild mustard would set substantially more immature seeds than S plants, thereby decreasing its overall ecological fitness relative to S.

Fitness in the presence of herbicide. The recommended dosage of 2,4-D (420 g ha^{-1}) killed all S plants in both years of the study, and severely inhibited the growth and seed return of R plants. Shoot dry matter accumulation and seed return of treated R plants was reduced by 75 to 90% compared to the untreated control.

However, R seed rain calculated on a square meter basis (20 plants m^{-2}) was still very high, i.e., 9000 and 5700 seeds m^{-2} in the two years of this study, sufficient to establish or maintain a very dense infestation of wild mustard. Lower dosages of 2,4-D (105 and 210 g ha^{-1}) affected R growth and seed return to a lesser extent (60 to 80% reductions). Also, in one of the two years, some S wild mustard survived and set seed when treated with these lower 2,4-D dosages.

Dicamba did not inhibit the growth and seed return of either S or R wild mustard to the same extent as 2,4-D. In both years of the study, some S wild mustard survived and set seed even when treated with the recommended dosage of dicamba (300 g ha^{-1}). Dicamba at 300 g ha^{-1} reduced S shoot dry matter and seed return by 80 to 90%, while R was reduced by 60 to 65%. Unlike some populations of ACCase inhibitor resistant wild oat which are not visibly affected by recommended dosages of fenoxaprop-p-ethyl or sethoxydim (Heap et al. 1993), the R wild mustard used in this study exhibits a functional level of resistance to recommended dosages of 2,4-D and dicamba in the field but is still greatly affected by these herbicides.

Although R wild mustard was injured by the herbicides, it still interfered with the wheat crop. Wheat yields in R wild mustard plots treated with 2,4-D were higher than in plots where wild mustard growth was not as inhibited (ie. untreated control and dicamba treated plots). In general, crop yield loss due to weed interference is density dependent and wheat yield losses occurred in this study at the relatively low density of 20 R wild mustard plants m^{-2}. If herbicide resistant weeds are not

identified while still sparsely scattered in the field and alternative control measures not implemented, infestations can rapidly increase to densities well above 20 plants m^{-2} and can cause significant crop yield losses (Morrison and Devine 1994).

Modelling for Resistance. The results of this study indicate a very high selection pressure for R wild mustard at the recommended dosage of 2,4-D. Although R wild mustard growth and seed set was greatly inhibited by the recommended dosage of 2,4-D, S plants were completely killed and set no seed. Inserting the results into Gressel and Segel's herbicide resistance rotational model (1990) indicates a very rapid development of resistance in a wild mustard population unless the initial frequency of R individuals is very low (i.e., 10^{-30}). Since the initial mutation frequency is the parameter in the model about which the least is known, it may be that a stable, heritable mutation conferring resistance to auxin-type herbicides in wild mustard occurs very infrequently. Given the widespread use of auxin-type herbicides over the last 30 to 40 years and the paucity of plants resistant to these herbicides, a very low, heritable mutation frequency may be a reasonable assumption.

Although both S and R wild mustard populations were severely inhibited by all rates of 2,4-D, the R seed rain observed from the commercial treatment of 2,4-D ranged from 200-400 seeds per plant. One can clearly see how such populations can become a significant weed problem, even if there is a small fitness cost in the absence of phenoxy herbicide. Reports from Gilbert Plains, suggest that the

phenoxy resistant wild mustard is well established, infesting farms with heavily populated stands.

Wild mustard is an important weed of field crops on the Prairies. Long soil seedbank life, competitive growth habit, and high fecundity all contribute to the weedy nature of wild mustard and ensure a continuing problem. Prior to the introduction and widespread usage of phenoxy herbicides, wild mustard was the worst weed of cultivated land on the Prairies. For three decades farmers depended almost exclusively on phenoxy herbicides for effective, selective control of wild mustard in cereal crops and certain other special crops. The development of sulfonylurea and imidazolinone herbicides during the mid- to late 1980's finally provided farmers with an alternative to phenoxy herbicides for wild mustard control. However, wild mustard populations resistant to ALS inhibitors have recently been identified in Manitoba and Alberta. It is possible that subsequent mutation and/or pollen movement followed by selection could give rise to 'double-resistant' wild mustard populations (plants resistant to both auxin-type herbicides and ALS inhibitors) and the resurgence of wild mustard as an extremely serious weed problem across the Prairies.

TABLE A-1. Percent emergence of three populations of wild mustard under two seeding rates.

Seeding rate	Resistant	Portage susceptible	Moran Farms susceptible
no. m^{-2}			
600	43	29	11
1200	53	21	6

Emergence testing was conducted outdoors from May 5-11, 1992. To simulate actual experiment conditions seeds were shallowly incorporated into soil taken from the potential experimental site at Portage la Prairie, Manitoba. Percent emergence was averaged based on two replicates.

TABLE A-2. Percent germination of three wild mustard populations after scarification with 12 N H_2SO_4 under three different time intervals.

Scarification (H_2SO_4, minutes)	Resistant	Portage Susceptible	Moran Farms Susceptible
control	60	60	35
1min	70	66	60
2min	86	41	7
3min	96	23	2

All wild mustard seed was kept at 5 C for 48 hours prior to a 96 hour 20 C treatment. This procedure is recommended by Goudey et al (1990). Percent germination was averaged, based on two replicates.

TABLE A-3. 1992 susceptible and resistant wild mustard leaf area cm^{-2} results.

DAE	27		34		41		48		55		62		69		95	
Pop	S	R	S	R	S	R	S	R	S	R	S	R	S	R	S	R
Control	136	158	345	351	578	546	777	698	453	510	232	410	220	134	49	42
2,4-D g ha⁻¹																
105	163	148	285	289	379	429	404	705	221	440	128	376	74	211	15	28
210	172	121	250	253	264	557	179	513	114	398	74	364	24	184	3	35
420	209	148	288	292	228	521	88	528	84	339	45	341	0	108	0	14
dicamba g ha⁻¹																
75	165	159	306	319	461	548	570	899	344	569	281	382	215	245	20	30
150	158	139	274	313	329	556	348	532	258	343	205	266	166	151	6	50
300	169	123	314	338	445	531	479	704	222	593	n/a	n/a	249	267	12	18

TABLE A-4. 1993 susceptible and resistant wild mustard leaf area cm^{-2} results.

DAE	27		34		41		48		55		62		69		97	
Pop	S	R	S	R	S	R	S	R	S	R	S	R	S	R	S	R
Control	390	219	745	357	1035	664	724	558	677	463	695	323	560	124	33	23
2,4-D g ha⁻¹																
105	222	182	280	359	203	429	61	259	65	278	12	206	67	114	0	4
210	176	171	245	376	144	363	42	238	76	287	0	158	0	69	0	15
420	214	155	227	321	147	276	23	205	0	207	0	168	0	56	0	6
dicamba g ha⁻¹																
75	224	178	460	448	532	596	420	486	564	525	443	288	264	258	24	18
150	252	158	448	448	418	472	280	413	385	506	325	221	59	186	16	15
300	218	188	271	462	292	446	163	372	121	472	n/a	n/a	61	180	17	8

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