A STUDY OF DICLOFOP-METHYL TOLERANCE IN OATS (AVENA SATIVA)

A Thesis

Submitted to the Faculty

of

Graduate Studies

The University of Manitoba

by

Thomas Dale Warkentin

In Partial Fulfillment of the Requirements for the Degree

of

Master of Science
Department of Plant Science

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THOMAS DALE WARKENTIN

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ABSTRACT

Warkentin, Thomas Dale. M.Sc., The University of Manitoba, May 1986. A study of diclofop-methyl tolerance in oats (Avena sativa). Major Professors; R.I.H. McKenzie, G. Marshall.

Field experiments examined the feasibility of achieving selective wild oat (Avena fatua L.) control in an Australian oat (Avena sativa L.) cultivar, Savena 1 from the cross: West x (West x New Zealand Cape/23) / 28. Diclofop-methyl (2- [4- (2,4-dichlorophenoxy) phenoxy] propanoic acid) was applied at rates of 0.4 to 0.7 kg/ha at the 3 and 5-leaf stages in the presence (84 to 132 culms/m²) and absence of wild oats. Although all rates of diclofop-methyl caused initial chlorosis and necrosis to the crop, the subsequent control of wild oats permitted increased crop tillering. Wild oat control and crop yield response was maximized when diclofop-methyl was applied at the 3-leaf stage. Final crop grain yield was increased by up to 32% and 22% during 1984 and 1985 respectively. In the absence of wild oat competition, Savena 1 shoot dry weight at flowering was reduced only where diclofop-methyl was applied at the 0.6 and 0.7 kg/ha rates, however, final grain yield was not reduced by any treatment.

A field screening experiment assessed the tolerance of 240 oat genotypes to the application of 0.4 and 0.7 kg/ha diclofop-methyl. Only nine genotypes showed any significant degree of tolerance and none reached the level attained by Savena 1 and a closely related Australian line [Irwin x (West x New Zealand Cape / 42)) x West] / 24. The nature of the inheritance of diclofop-methyl tolerance in Savena 1 oats was examined (1983 - 1985) by crossing and backcrossing (BC) Savena 1 with

four diclofop-methyl susceptible, but agronomically superior, lines from the Agriculture Canada, Winnipeg breeding program. Field trials in which the resulting F3, BC1F2 and BC1F3 lines were treated with 0.4 and 0.7 kg/ha diclofop-methyl were rated visually for herbicide tolerance. Inheritance appeared to be controlled by two genes with susceptibility being dominant to tolerance. Two other sources of diclofop-methyl tolerance in Avena reported in the literature were tested. Neither possessed a level of tolerance comparable to Savena 1.

INTRODUCTION

Oats (Avena sativa L.) is the third ranked cereal crop in Canada and the fifth in North America in terms of area in production. (Anon., 1982b). The average harvested area of oats in Canada over the last five years (1980-1984) has been 1.49 million hectares with an average production of 3.039 million tonnes. The major use of the oats crop is as a feed source for livestock (Anon., 1984a).

A major restriction to the expansion of oat acreages is that imposed by weed control. Wild oats is one of the most economically harmful annual grass weeds of cultivated land in many areas of the world, especially in North America, Europe, and Australia (O'Donovan and Sharma, 1983). The chemical control of wild oats is possible in all major field crops of western Canada except oats (Anon., 1986a). Wild oat control in cultivated oats has been unsuccessful thus far because of the close genetic relationship of these two species.

This project investigated the feasibility of developing a herbicide-tolerant oat cultivar. A diclofop-methyl (Hoegrass) tolerant oat variety named 'Savena 1' developed by Barr (South Australia Department of Agriculture, Adelaide) was the source of the tolerance trait. Diclofop-methyl provides effective control of wild oats, green foxtail, yellow foxtail and barnyard grass which are important weed problems of western Canada (Anon., 1986b).

The objectives of this study were as follows:

 To determine the efficacy of diclofop-methyl in controlling wild oats in Savena 1 oats.

- To screen a number of oat genotypes for possible tolerance to diclofop-methyl.
- 3. To consider the potential of incorporating diclofop-methyl tolerance into oat genotypes adapted to western Canada, and to study possible mechanisms of inheritance of this trait.

1.0 REVIEW OF LITERATURE

1.1 The Oats Crop

Avena sativa L. and A. byzantina L. are the most common cultivated oat species on a world scale. A. sativa is the oats species of temperate regions, including North America, while A. byzantina is grown as a winter crop in Mediterranean climates (Rajhathy and Thomas, 1974).

In the five year period from 1980 to 1984, the average harvested area of oats in Canada was 1.49 million hectares (Anon., 1984a). This made oats the fourth most widely grown crop, and third most widely grown cereal, in Canada. In the 1970's, oats was ranked third in seeded area among crops, but has since been surpassed by rapeseed. Oat production and acreage in Canada has actually been declining since the 1930's (Anon., 1976, 1984a, 1964, 1964-1965).

By far the major use of oats produced in Canada is as a feed source (Anon., 1984a). Oats used for feed (plus waste and dockage) accounted for 89.5 per cent of the average annual disposition (aside from carryover) from 1974-1983. Oats are fed to horses, cattle, poultry and hogs (Martin et al., 1976). A small proportion (2.0 per cent) was used for human food, where its major uses were for the production of rolled oats and breakfast cereals (Western and Graham, 1961). The remainder was exported or used for seed. Canadian oat exports comprised a minimal proportion of total production in the ten year period from 1973-1982. During this time exports varied from 12,000 to 491,000 tonnes annually. A single country did not annually import a large amount of Canadian oats (Anon., 1984a).

Oats is an important crop on a world scale. In 1982 it was the ninth most widely grown crop in the world, and seventh ranked cereal crop (Anon., 1982b). Within the oat producing countries, Canada ranked third in average area seeded (1.89 million ha) and average amount produced (3.68 million tonnes) during the ten year period from 1974-1983 (Anon., 1984a). As a percentage of production, world trade in oats has been minimal (Anon., 1982c). Most oats has been used in the country in which it was produced.

Martin et al., (1976) described some of the agronomic traits of oats. Oats fits well into many crop rotations whether in monoculture or in a companion cropping situation and is adapted to a wide range of soil types. Oats can be damaged by hot, dry weather just prior to heading. The most prevalent diseases of oats in North America are stem rust (Puccinia graminis f.sp. avenae), crown rust (Puccinia coronata), loose smut (Ustilago avenae), and covered smut (Ustilago kolleri). These authors suggested that the reasons for declining oat production worldwide is the fact that other feed grains, maize, sorghum, and barley out yield oats, and the replacement of horses and other work animals (consumers of oats) with motorized equipment.

1.2 Weed Problems in Oats

Every field crop has associated weed problems. Over the past 40 years, the use of herbicides has become an important method of weed control in Western Canada. The 1986 Guide to Chemical Weed Control (Manitoba) recommended several herbicides for use on oats. These herbicides control all of the important annual broadleaved weeds of

Manitoba as well as the grass weeds green and yellow foxtail. Thus, the major weed problems of oats are a small group of annual grass weeds.

Barnyard grass (Echinochloa crusgalli L.), green foxtail (Setaria viridis L.), yellow foxtail (Setaria glauca L.), Persian darnel (Lolium persicum Boiss. and Hohen.) and wild oats were the annual grass weeds listed in recent weed control guides of Manitoba, Saskatchewan and Alberta. Chemical control of barnyard grass (using sodium TCA) and green and yellow foxtail (using sodium TCA or propanil) is possible in oats crops. There are no chemical control measures listed for the control of wild oats or Persian darnel in oats.

In the 1981 Weed Survey of Cultivated Land in Manitoba, Thomas (1982) found the following proportion of fields surveyed to be infested with these annual grass weeds.

	Weed	Frequency (%)
1	Green foxtail	80.9
	Wild oats	73.3
	Barnyard grass	6.8
	Yellow foxtail	<1.0
	Persian darnel	<1.0
	Volunteer corn	<1.0

A more recent survey of agricultural land in Saskatchewan, produced the following results (Thomas, 1985).

	Frequency Level*				
	Absent	0.1-25	26-50	51-75	76-100
Weed		(numbe	r of dis	tricts)	•
Wild oats	0	1	4	14	24
Green foxtail	1	7	16	11	8
Barnyard grass	23	19	1	0	0
Persian darnel	18	25	0	0	0

^{*}The number of fields in which a species occurred, expressed as a percentage of the total number of fields surveyed.

Producers in both Manitoba and Saskatchewan rated wild oats as the most troublesome weed on their farm (Thomas, 1983). In addition, wild oats occurred in 60% and 38% of surveyed fields in Alberta and the Peace River region of British Columbia, respectively. In Manitoba in 1981, approximately 60% of surveyed fields were treated with a wild oat herbicide (Thomas, 1983). Dew (1978) estimated that the cost of wild oat infestations in terms of crop losses and herbicide expenditures was \$280 million annually in Western Canada alone.

It can be seen that wild oats is a widely distributed and serious weed problem in Western Canada. It is especially serious in oat production because of the lack of a selective herbicide for its control.

Wild oats (<u>Avena fatua</u>) is the major uncontrolled weed problem of oat crops in Britain (Taylor and Codd, 1985) and throughout North America (Shands and Chapman, 1961). Barr (Personal Communication, 1983) stated that <u>Avena sterilis</u> and <u>A. barbata</u>, as well as <u>A. fatua</u>, are weed problems of cultivated oats in Australia.

Since green and yellow foxtail can be chemically controlled in oats

using propanil (Anon., 1986b), barnyard grass by sodium TCA (Anon., 1980); and Persian darnel and volunteer corn are very minor weeds in Western Canada (Thomas, 1982, 1985), the following discussion will concentrate on wild oats, the most serious weed problem in oats.

1.2.1 Wild Oat Competition

Wild oats decreases crop growth and yield by competing for mineral nutrients, water, and light (Eddowes, 1972). Competition between wild oats and crops is a complex system. Chancellor and Peters (1976) and O'Donovan and Sharma (1983) list the factors involved as follows: the crop and cultivar seeded, the crop density, the wild oat density, the date of sowing, the period of wild oat emergence relative to the crop, the soil fertility level, and the climatic conditions. Chancellor and Peters (1976) summarized several reports on the effect of chemical removal of wild oats (infestations ranged from medium to heavy) on the yield enhancement of various crops. Yield increases ranged from 0-344% in wheat, 0-110% in barley, 17-148% in flax, and 10-107% in peas. Studies on the competitiveness of wild oats in cultivated oats are not available in the literature. For this reason, discussion will center on the other cereal crops of Western Canada. O'Donovan and Sharma (1983) reported on the following yield reductions caused by wild oats (150-200 plants/ m^2): 26% (barley), 33-39% (wheat), 46% (rapeseed), and 86% (flax). Even at 12 plants/ m^2 , wheat and flax yields were significantly reduced.

Dew (1972) developed an index for wild oat competition in barley, wheat, and flax. Data was obtained from replicated studies in which these three crops were grown in competition with wild oats that had been

broadcast seeded to produce various weed densities. Regression equations were calculated based on: expected weed free yield of the crop, the weed population, and the index of competition. The competition indices obtained were: 0.0230 (barley), 0.0339 (wheat), and 0.0601 (flax); ie. barley is the best competitor with wild oats, flax is the poorest. A modified index calculated by O'Donovan et al., (1985) quantified wheat and barley yield losses accounting for the time of emergence of wild oats relative to the crop. For every day wild oats emerged prior to the crop, yield loss increased by approximately 3 per cent. An index has not been calculated for wild oat competition in cultivated oats but Pavlychenko and Harrington (1934) determined the competitive ability of spring crops in the following order: barley > rye > wheat > oats > flax. This was based on uniformity of germination under moisture stress, ability to rapidly develop a large assimilation surface, number of stomata, and size and profile of the root system. Chancellor and Peters (1976) reported a similar order of competitiveness.

Another factor that contributes to the weediness of wild oats is its seed characteristics. Chancellor (1976) summarized several studies and concluded that wild oat seeds can remain viable in cultivated soils for 2-9 years, and generally longer under untilled grass conditions.

Avena fatua, A. byzantina, and A. sterilis all display some degree of post-harvest dormancy (Chancellor, 1976). Therefore, a tillage operation after harvest will not eliminate all wild oats in a field.

Each floret of Avena fatua disarticulates (shatters) at maturity (Thomas and Jones, 1976). Since seeds mature in sequence from the top of the

panicle downward, a large proportion of seed will be returned to the soil before a crop is removed by combine harvester.

Wild oats not only reduce the quantity of a crop such as oats but also reduce its quality. Wild oat seeds can downgrade oats. The Canadian Grain Commission states that the maximum allowable quantity of wild oats is 1% in No. 1 C.W., 2% in No. 2 C.W., 4% in No. 1 Feed, 8% in No. 2 Feed, 12% in No. 3 Feed, and over 12% grades as mixed grain (Anon., 1984b).

1.2.2 Wild Oat Control

The deleterious effects of wild oats on cereal production necessitate the use of some type of control measure. Decreasing the number of seeds that return to the soil was considered the major objective of wild oat control by Elliot (1976). Methods of controlling wild oats can be classified as either 'cultural' or 'chemical'.

1.2.2.1 <u>Cultural Control of Wild Oats</u>. 'Cultural control' refers to the management practices used by a farmer to reduce a weed population. Cussans and Wilson (1976) and Hunter (1983) discussed some of the techniques used and some of their shortcomings. Delayed seeding was the most widely used cultural control method reported. Delaying seeding, tilling to stimulate wild oat germination, followed by another tillage operation, or direct seeding, was the procedure used. However, delayed seeding reduces the yield potential of annual crops. The 1986 Field Crop Recommendations for Manitoba Guide states that wheat, oats and barley should be seeded as early as soil conditions allow. These authors also stated that the additional tillage dries the soil and

breaks down soil structure. Sowing a competitive crop was a second cultural control method discussed. The competitiveness of certain annual crops was discussed in Section 1.2.1. Fall tillage, to promote germination before winter, was considered useful if temperatures were warm. Shallow seeding combined with adequate fertility can allow crops to emerge before wild oats, thus significantly reducing wild oat competition (Hunter, 1983). The use of wild oat free seed was an obvious precaution. Removing a crop as green feed prevented wild oats from setting seed but was often found to be impractical. Burning straw or summerfallowing were not considered useful cultural control methods.

Thomas (1983) reported that the use of most cultural control methods for wild oat control in Western Canada has been limited in recent years. Cussans and Wilson (1976) suggested that cultural control practices were more applicable to the containment of small weed populations than for large populations. In the last 25 years, the use of chemical wild oat control has increased greatly in importance.

1.2.2.2 Chemical Control of Wild Oats. Holroyd et al., (1976) described the requirements of an 'ideal' wild oat herbicide as follows. All species of Avena should be susceptible at all stages of growth; herbicide activity and persistance in the soil should be such as to control seeds which germinate after treatment. Adverse effects on the crop should be minimal, even when the crop is very closely related genetically. Treatments should be easy to apply and cost should be appreciably less than the expected return.

The herbicides barban (Carbyne 2 EC), triallate (Avadex BW), difenzoquat (Avenge 200C), flamprop-methyl (Mataven), and

diclofop-methyl (Hoegrass) are currently recommended for the control of wild oats in wheat and/or barley (Anon., 1986b). All of these herbicides provide good wild oat control (except barban) and good crop tolerance when applied at the proper rate and stage of growth of the crop.

Due to genetic similarities, there has never been a herbicide recommended for control of wild oats in cultivated oats in Canada. The herbicide chlorfenprop-methyl was released in the United Kingdom (trade name: Bidisin) in the late 1970's for wild oat control in certain oat cultivars (Fryer and Makepeace, 1978). Stryckers et al. (1972) found that seven oat cultivars tested were tolerant, while five were very susceptible to chlorfenprop-methyl. In addition, some biotypes of Avena fatua were tolerant (Taylor and Codd, 1985) as were A. sterilis and A. strigosa. Use of chlorfenprop-methyl has been discontinued (Taylor and Codd, 1985).

1.3 <u>Diclofop-Methyl</u>

Diclofop-methyl is the active ingredient of the herbicides:
Hoegrass (Canada), Hoelon (U.S.A.) and Illoxan (other countries). It
was discovered in 1971 by Hoechst AG (Kocher, 1983). The full chemical
name of this compound is 2-(4-(2, 4-dichlorophenoxy)-phenoxy)-methylpropanoate. This name is generally shortened to 'diclofop-methyl' or
'diclofop' for common use. The structure of the molecule is as follows:

Empirical formula: $C_{16}^{H}_{14}Cl_{2}^{0}_{4}$

Diclofop-methyl is used as a selective herbicide in many dicotyledonous crops as well as in wheat and barley for the control of certain annual graminaceous weeds. The major weeds controlled by diclofop-methyl are: wild oat species (Avena spp.), wild millets (Echinochloa spp., Setaria spp.), rye grass (Lolium spp.) and volunteer corn (Zea mays) (Kocher, 1983).

1.3.1 Effect of Diclofop-methyl on Plant Structures

Several researchers have reported on the injury symptoms caused by diclofop-methyl. These symptoms can be divided into effects on: shoots, roots, and cell ultrastructure.

Hoerauf and Shimabukuro (1979) reported on the visual symptoms of susceptible wild oat and resistant wheat to foliar applications of diclofop-methyl (0.84 kg/ha). Symptoms could first be detected three days after treatment.

- (a) On wheat: Discrete chlorotic spots occurred only on the parts of the 2nd and 3rd leaves exposed to the herbicide. New leaf growth was not injured. This limited chlorosis did not affect dry matter accumulation measured 15 days after treatment.
- (b) On wild oats: The 2nd and 3rd leaves became entirely chlorotic and seven days after spraying they became necrotic. New growth was

inhibited and internodes failed to develop. Shoot dry weight ranged from 39-51% of control plants 15 days after treatment.

Brezeanu et al., (1976) observed similar symptoms. Donald and Shimabukuro (1980) reported on growth inhibition in wild oats after two days and chlorosis after three days. Hoerauf and Shimabukuro (1979) found that herbicide placement greatly affected symptom expression.

Greatest injury occurred when the herbicide droplet was applied to the leaf sheath. Application to the center of the 2nd leaf alone caused chlorosis and necrosis in this area but new growth was not affected.

These authors suggested that it is important to apply diclofop-methyl in a way so as to contact the lower portions of the plant.

Kocher (1983) reported that diclofop-methyl strongly inhibited root growth of susceptible plants. Rates as low as 10⁻⁷ M in aqueous solution inhibited primary root growth of Avena sativa and A. fatua seedlings by 50 per cent. This rate stimulated adventitious root emergence from the oat crown but inhibited their elongation. A rate of 10⁻⁶ M was found to inhibit both emergence and elongation of adventitious roots. In early studies with the herbicide, Crowley et al., (1978) found that growth inhibition of wild oats three weeks after planting was greater when roots grew through treated soil (0.4, 4, 16 mg diclofop-methyl/kg dry soil), than when shoots grew through a treated layer of soil. Root injury was associated with reduced ⁴⁵Ca uptake.

Morrison et al., (1981) found that diclofop-methyl severely affected root tip anatomy. Cells in the vascular region were affected within one day, and within four days tissue destruction was general

throughout the central cylinder.

Chloroplasts are the organelles most affected by diclofop-methyl (Kocher, 1983; Brezeanu et al., 1976). Membrane damage, as well as abnormal formation of new chloroplasts, occurs. Brezeanu et al., (1976) found the following injury symptoms to chloroplasts: disruption of the cisternae tissue that connects grana, swelling of thylakoids, change of shape of the entire chloroplast from discoid to spherical, disorganization of the entire thylakoid system, and bursting of the chloroplast envelope releasing contents into the cytoplasm.

Brezeanu et al., (1976) found diclofop-methyl damage to mitochondria to be limited, while Cohen and Morrison (1981) found increased swelling of membranes. However, mitochondria were not thought to be the primary site of diclofop-methyl phytotoxicity.

Other cellular symptoms noted were: separation of the plasmalemma from cell walls, the appearance of vesicles in the vacuole (Kocher, 1983; Brezeanu et al., 1976), and injury to the tonoplast (Kocher, 1983). The accumulation of vesicles was thought to be due to the presence of lipid material that would normally be used for thylakoid production.

1.3.2 Effect of the Environment on Diclofop-methyl Activity

Kocher (1983) summarized the effects of environmental conditions on diclofop-methyl activity. Weed control using diclofop-methyl was found to be most effective at temperatures suited for vigorous growth of the target weeds. This herbicide was not affected by rainfall shortly after application. A 15 mm artificial rain 0.5 hours after spraying wild oats did not significantly reduce weed control. Moisture stress conditions,

however, reduced the effectiveness of diclofop-methyl. Dortenzio and Norris (1980) found that diclofop-methyl activity was reduced 15-50% (as measured by reduction in wild oat dry weight) when soil was held at 2-3% above wilting point as compared to near field capacity. Akey and Morrison (1983) obtained similar results. Soils were maintained at -6.5 bars and -0.3 bars for 5 days after spraying. Wild oat control was 38% poorer in the stressed plants, as measured by shoot dry weights. Reduced translocation of diclofop-methyl to the youngest leaves, the tillers, and shoot apex of wild oats in the stressed vs. unstressed plants was the explanation given.

1.3.3 Mode of Action and the Basis of Selectivity of Diclofop-methyl

Uptake of diclofop-methyl by wheat, barley, wild oats, and green foxtail occurs over a period of four days or more after application, with most rapid uptake occurring in the first 12-24 hours (Kocher, 1983). Only a small proportion of applied diclofop-methyl is translocated in plant tissue. Using ¹⁴C-labelled diclofop-methyl, Kocher (1983) found translocation to be as follows: wheat, 1.7%, wild oats, 0.9% and green foxtail, 0.8%. Brezeanu et al., (1976) reported that both wild oat and wheat translocated 4% of applied diclofop-methyl in the four days after treatment. Boldt and Putnam (1980) found that less than 2% of applied diclofop-methyl was translocated out of the treated leaf within five days after treatment in the five species tested, proso millet (Panicum miliaceum L.), cucumber (Cucumis sativus L.), soybean (Glycine max L.), longspine sandbur (Cenchrus longispinus (Hack.)), and barnyard grass. Kocher (1983) developed autoradiographs of leaves from different species treated with labelled diclofop-methyl.

Material translocated above and below the site of application was located mainly within the conducting tissues and decreased with increasing distance from the site of application. Greatest damage to susceptible weeds occurred when the herbicide was applied near the base of the shoot.

Several studies have been conducted comparing retention, uptake, and translocation of diclofop-methyl in resistant and sensitive species. Herbicidal selectivity of diclofop-methyl between cereals (wheat and barley) and wild oats cannot be explained by differential retention and uptake (Todd and Stobbe, 1977; Donald and Shimabukuro, 1980; Boldt and Putnam, 1980) or differential translocation (Kocher, 1983; Brezeanu et al., 1976; Boldt and Putnam, 1980).

Chow (1982) treated wild oats with 1.1 kg/ha diclofop-methyl at the 3-leaf stage and measured physiological responses 6-7 days later. This time period may be too long to assess primary effects of the herbicide, however some of his findings were noteworthy. Diclofop-methyl was found to reduce ³²P incorporation into lipids (40% reduction relative to control), DNA (45%) and RNA (30%). Chow (1982) stated that the reduced phospholipid content would have an important impact on electron transport, oxidative phosphorylation, and energy-linked transport of ions across membranes. Fedtke (1982) postulated that the site of action of diclofop-methyl is located in a lipophilic compartment which is most likely the plasma membrane. At this location, several possible sites for the binding of the herbicidally active acid may exist.

Hoppe (1985) found that diclofop-methyl caused an early and pronounced inhibition of the incorporation of $^{14}\mathrm{C}$ -acetate into leaf

lipids of the sensitive plant species maize, wild oat, and barnyard grass and in the resistant species wheat. This inhibition could be detected 0.5-4 hours after herbicide application (10^{-7} M). In wheat, recovery occurred within 4 days. It was noted that in tolerant bean (<u>Phaseolus vulgaris</u>), sugarbeet (<u>Beta vulgaris</u>), and soybean (<u>Glycine max</u>) fatty acid biosynthesis was unaffected by diclofop-methyl.

Other physiological responses to diclofop-methyl have been observed. Chow (1982) found that one week after treatment, serine (+53% of control plants) and threonine (+85%) accumulated in wild oat plants. It was suggested that metabolic pathways of these two amino acids to end products may have been blocked. Inhibition of photosynthesis was seen as a secondary response of the plant due to chloroplast membrane damage (Chow, 1982; Kocher, 1983; Brezeanu et al., 1976).

Mitochondrial activity was only reduced when diclofop-methyl rates were very high (0.5 mM) (Kocher, 1983). At these rates, both wheat and wild oats were affected.

Morrison et al., (1981) considered mitotic index as a possible explanation for diclofop-methyl injury. They exposed wheat and wild oats to diclofop-methyl at rates of 0.15-3.0 μM for 8-24 hours. The mitotic index (a measure of the percentage of cells dividing at a given time) of adventitious root tips of wheat was not affected after 8 hours, but was significantly reduced after 24 hours at the high rates. Wild oat root tips showed a significant decrease in their mitotic index after 8 hours at rates of 0.30-3.0 μM. They suggest that diclofop-methyl probably arrests cells in the interphase stage of the cell cycle. However, these researchers caution that simply stating that

diclofop-methyl prevents mitosis does not explain its primary mode of action.

Differing pathways of metabolism of diclofop-methyl in tolerant versus susceptible species appears to be the basis of selectivity of this herbicide. Shimabukuro et al., (1979) found that in both wheat and wild oats, diclofop-methyl was hydrolyzed rapidly to diclofop acid.

After 24 hours, only 3% of the diclofop-methyl applied to wheat, and 4% applied to wild oats remained as diclofop-methyl, the remainder was in the acid form. They proposed the following degredation pathways for the two species.

(after Shimabukuro et al., (1979).

Goreka et al., (1981) and Hoppe (1985) also concluded that irreversible aryl-hydroxylation is the mechanism used by wheat and barley to detoxify diclofop-methyl. Twenty hours after treatment, wheat coleoptiles contained only 10% of the applied product in a potentially active form (parent ester, free acid, or ester conjugate), while oats contained 70% in one of these forms. Shimabukuro et al., (1979) noted that the ester conjugate in Avena spp., although not toxic itself, can

readily be reconverted to diclofop acid. Both the acid and parent ester forms are biologically active (Donald and Shimabukuro, 1980; Shimabukuro et al., 1978).

In 1981, Boldt and Putnam found that although soybeans were tolerant to diclofop-methyl, they did not conjugate diclofop acid to the same extent as monocots. Hoppe (1985) proposed that dicot crops displayed a different mechanism of tolerance than that of tolerant monocots. He stated that dicot tolerance (in species tested) probably lies at the site of action of diclofop-methyl, since fatty acid biosynthesis in chloroplasts was not inhibited by the herbicide. On the contrary, in maize (a sensitive monocot) a 60% inhibition was observed.

1.3.4 Summary

Diclofop-methyl is an effective herbicide for the selective control of graminaceous weeds in wheat, barley and dicot crops. Herbicide damage by diclofop-methyl consists of:

- ultrastructural cell damage, primarily chloroplast membrane damage;
- growth inhibition due to chloroplast damage and a reduced mitotic rate.

The outward symptoms of this damage are:

- 1. chlorosis and necrosis of leaves,
- inhibition of new shoot and root growth.

Diclofop-methyl selectivity between tolerant and susceptible monocots has been found to be due to differential detoxification mechanisms. In all plants tested, diclofop-methyl is rapidly hydrolyzed to the acid form. In wheat and barley, the acid is detoxified by aryl-hydroxylation. Susceptible species convert diclofop acid to an

ester conjugate. The ester is not toxic, however it can be reconverted to the acid form which has a lethal effect on susceptible plants.

1.4 Herbicide Tolerance in Plants

The evolution of herbicide tolerant plants is an example of the biological flexibility and adaptibility of living organisms. Modern organic chemicals have revolutionized crop production, and despite their limitations they are considered to be a major and increasing part of agricultural technology in the decades ahead (LeBaron and Gressel, 1982). Resistance to agricultural chemicals began to occur after they came into widespread use. LeBaron and Gressel (1982) reported that by 1980, 428 species of arthropods had become resistant to one or more insecticides that were once effective against them. As well, disease resistance to fungicides has increased markedly since 1967. Weed resistance to herbicides which once provided excellent control has come about more slowly than resistance to insecticides and fungicides. This is due to the longer reproductive cycles of plants as compared to insects or fungi.

1.4.1 <u>Herbicide Resistance vs Tolerance</u>

The terms 'herbicide resistance' and 'herbicide tolerance' are sometimes misused or used interchangeably. LeBaron and Gressel (1982) refer to tolerance as "the natural and normal variability to pesticides and other agents which exist within a species and can easily and quickly evolve". They state that the term 'tolerance' can also be used to make comparisons between species. Resistance, as defined by the FAO, is a more drastic lack of response of a population of animal or plant species

to a pesticide or control agent as a result of their repeated application. Resistance should not be confused with natural tolerance or low susceptibility due to a normal physiological or behavioristic property of an unselected population. The working definition of these terms used by LeBaron and Gressel (1982) is as follows. "A resistant weed is one that survives and grows normally at the usually effective dose of a herbicide. Resistance is the maximum tolerance that can be achieved."

1.4.2 Origins of Herbicide Resistance

Resistance to major herbicide families has been reported in biotypes of many weed species that were previously controlled by the herbicide. Bandeen et al., (1982) described the discovery and distribution of herbicide resistant weeds in North America. A similar review of herbicide resistant weeds from outside North America was produced by Gressel et al. in 1982 (Table 1).

It can be seen that the triazines are the herbicide family to which the largest number of tolerant biotypes has been reported. The first documented case of triazine tolerance occurred approximately 10 years after these herbicides were in widespread use. Ryan (1970) found that large doses of simazine did not control Senecio vulgaris (common groundsel) plants in a conifer nursery in Washington State. Most reported cases of triazine tolerance occurred on land that had been treated annually for more than 10 years. This was usually on corn fields, tree nurseries, or railway right-of-ways. Infestations generally appeared as scattered plants in a field where otherwise good weed control existed. These plants then tended to spread rapidly through a field (Bandeen et al., 1982). The size of infestations reported by

TABLE 1. Herbicide families to which tolerant weed biotypes have arisen.

	Number of Weed Species Reported with Tolerant Biotypes				
Herbicide Family		Outside North America ²			
Triazines	17	21			
Phenoxys	7	4			
Dalapon and TCA	8	_			
Carbamates and thiocarbamates	1	_			
Glyphosate	1	_			
Urea and uracils	1	1			
Amitrole	1	-			
Trifluralin	1	_			
Benzonitriles	-	1			
Bipyridiliums	_	3			

¹Bandeen <u>et al</u>., 1982.

²Gressel <u>et al</u>., 1982.

Bandeen et al., (1982) ranged from 2 - 250,000 ha. These researchers stated that there are still vast agricultural areas of North America where there have been no reports of triazine resistant weeds despite extensive use of these herbicides. They suggested that the reasons for this may be the fact that corn/soybean rotations, combined with herbicide rotations, are used as opposed to continuous corn treated with atrazine. Also, on much of this land (S.E. States and the Mid-West cornbelt) at least one cultivation is conducted annually.

A second herbicide family to which many resistant biotypes have evolved are the phenoxys. Sexsmith (1964) first detected differential 2,4-D sensitivity in <u>Cardaria chalapensis</u> (hoary cress) biotypes in southern Alberta in 1951. Since that time, several other examples of phenoxy tolerance have been reported however, not as many as with the triazines considering the length of time phenoxys have been used and their widespread distribution.

1.4.3 Factors Affecting the Appearance of Herbicide Resistance

Gressel (1978) considers three major factors that affect the appearance and rate of appearance of herbicide resistance. First, the frequency of resistance genes in the plant population is important. Gressel (1978) suggested that the frequency of resistant individuals in an untreated population would be between 10^{-10} and 10^{-5} but that prediction was difficult and depended on the number of genes controlling resistance, dominance, and the ploidy of the plant involved. Chaleff and Parsons (1978) isolated a picloram tolerant Nicotiana (tobacco) line. Tolerance was due to a single dominant nuclear gene with a frequency of about 10^{-5} . Faulkner (1982) reported on the screening of the USDA flax

collection (1541 samples) for atrazine tolerance. Only one line was found with tolerance two times that of a standard cultivar. This population was obviously not large enough to detect useful genes.

The selection pressure, or kill rate, of the herbicide is a second factor that affects the appearance of herbicide resistance. The greater the rate, below 100 per cent, the more rapidly resistant strains arise. This is one reason why resistance to triazines has occurred to a greater extent than to phenoxys. Triazines have a higher kill rate and greater persistence.

Finally, Gressel (1978) states that the fitness of the resistant biotype is important in its ultimate survival. 'Fitness' is the ability of a resistant line to compete with sensitive plants in the absence of herbicidal selection pressure (Gressel, 1978). Reduced fitness is a common phenomenon in resistant lines of bacteria, fungi, insects, and plants. It can take on several forms: slower germination and establishment, less vigorous growth, less plasticity with respect to the environment, and reduced seed yield (Gressel, 1978). Conard and Radosevich (1979) found that resistant plants of Amaranthus, Chenopodium, and Senecio were only about half as fit (in terms of seed production) as sensitive plants. The recently released, triazine-tolerant canola cultivar, OAC Triton, is less fit than triazine sensitive cultivars such as Regent (Anon., 1986a).

1.4.4 Rationale for Developing Herbicide-Tolerant Crop Cultivars

Faulkner (1982) describes his rationale for the development of herbicide tolerant crop cultivars as well as some of the associated limitations.

- Herbicide tolerant crops make it possible to control weeds that were previously impossible or very expensive to control in a given crop.
- 2. It is less expensive to produce a new cultivar than it is to develop a selective herbicide. Faulkner (1982) estimated this cost to be 1-5% of that of a new herbicide.
- A herbicide tolerant crop provides an additional alternative to a crop rotation.
- Weed control in companion-cropping situations, where both crops must tolerate the herbicide, can be improved.
- 5. Herbicide tolerant crops allow for the removal of seeds that reduce the grade of a crop, for example wild mustard seeds in canola, or wild oat seeds in cultivated oats.

A possible disadvantage of herbicide resistant crops that was introduced in Section 1.4.2, is a reduced level of fitness. This need not be a universal rule. Erickson et al., (1985) isolated several Chlamydomonas reinhardi (algae) mutants with triazine tolerance. Two of these mutants did not exhibit reduced rates of photosynthetic electron transport-characteristic of higher plants tolerant to triazines (Arntzen et al., 1982). Faulkner (1982) suggested that a second disadvantage of herbicide tolerant crops is that they could cause weed problems themselves, either as volunteers in succeeding crops or through outcrossing to closely related weeds. To reduce this risk, it was recommended that tolerance be sought to a single herbicide and in species where an alternative means of control existed.

1.4.5 Techniques of Incorporating Herbicide Tolerance into Crops

There are three broad classes of methods being studied in breeding crop cultivars for herbicide tolerance. These are: conventional plant breeding techniques, cell culture techniques, and molecular biology or genetic engineering techniques.

1.4.5.1 Conventional Plant Breeding. Faulkner (1982) described the general strategy used in breeding for herbicide tolerance by conventional methods. It involved: i) locating a gene(s) for tolerance and, ii) incorporating the gene into a susceptible, but agronomically superior cultivar.

Tolerance genes could be located by chance, however a more structured approach generally involved the screening of large collections of a crop(s) with a given herbicide. Faulkner (1982) suggested that screening of wild or primitive species related to a given crop could also be useful if these species could be successfully crossed to the crop in question. If a tolerant biotype was detected, it could be used in a breeding program.

Incorporating the tolerance trait into a cultivar generally involved crossing, via emasculation and pollination, the tolerant biotype with a susceptible but agronomically superior cultivar.

Table 2 describes conventional plant breeding programs from which herbicide-tolerant cultivars were produced. Various breeding strategies were used. Faulkner used recurrent selection to develop paraquat and dalapon tolerant varieties of Lolium perenne, a cross-pollinating species (Wright and Faulkner, 1981). Triazine tolerance was transferred into Brassica campestris, B. napus (Beversdorf et al., 1980) and

Herbicide tolerant cultivars produced by conventional plant breeding techniques. TABLE 2.

Reference	Year	Crop	Herbicide	Variety Name
Wright, C. E. & J. S. Faulkner	1981	Perennial rye grass Perennial rye grass	paraquat dalapon	Causeway No name given
Beversdorf, W. D. et al.	1980	Canola	triazines	OAC Triton
Souza Machado, V. et al.	1983	Rutabaga	atrazine	A/Laur
Kerr, F. A. & F. I. Cook	1983	Tomato	metribuzin	Wondervee
Tseng, S. T. et al.	1984	Rice	molinate & thiobencarb	L-202
Barr, A. R.	1985	Oats	diclofop-methyl	Savena 1
Hartwig, E. E.	1985	Soybean Soybean	2, 4-DB metribuzin	Tracy Tracy-M

B. napus var. napobrassica (rutabaga) (Souza Machado et al., 1983) via backcrossing. Barr (1985) (oats) and Hartwig (1985) (soybeans) selected for tolerance in segregating generations using the pedigree breeding method. It should be noted that Kerr and Cook (1983) and Tseng et al., (1984) released cultivars with herbicide tolerance, but that in each program, tolerance was not the primary objective. The method used to incorporate the trait was not reported.

Table 3 lists several other studies of herbicide tolerance in which crosses were conducted via emasculation and pollination, followed by analysis of segregating generations. An exception is Pinthus et al., (1972) who used mutagenesis in an attempt to induce herbicide tolerance. These studies did not result in the production of cultivars, rather most were designed as inheritance studies only. Table 3 lists the generation to which the inheritance study was carried.

The efficiency of selection in any breeding program depends on the mode of inheritance of the desired trait. The inheritance of herbicide tolerance can involve various levels of genetic complexity. Simple inheritance, whereby tolerance is controlled by one major dominant or recessive gene, has been shown to occur by Souza Machado et al., (1982) (metribuzin tolerance in tomato), Hayes et al., (1965) (tolerance to the chlorosis reaction of DDT and barban in barley), Grogan et al., (1963) (maize sensitivity to triazines), and Edwards et al., (1976) (soybean sensitivity to metribuzin). The following authors found triazine tolerance to be maternally inherited: Beversdorf et al., (1980) (in rapeseed), Souza Machado et al., (1983) (in rutabaga), and Scott and Putwain (1981) (in Amaranthus retroflexus). The literature

TABLE 3. Herbicide tolerance studies using conventional plant breeding techniques.

Reference	Year	Crop/Weed	Herbicide	Generation to which inheritance was studied
Hayes, J. D. et al.	1965	Barley	DDT, barban	F3
Karim, A. and A. D. Bradshaw	1968	Wheat, rapeseed, mustard	Simazine	
Comstock, V. E. and R. N. Andersen	1968	Flax	Atrazine	F3, BC2F2
Stafford, R. G. et al.	1968	Flax	MCPA	
Pinthus, M. J. et al.	1972	Wheat Tomato	Terbutryn Diphenamid	M3-M5 M2-M4
Schooler, A. B. et al.	1972	Foxtail barley	Siduron	F2
Devine, T. E. et al.	1975	Bird's Foot Trefoil	2,4-D	
Edwards, C. J. J. et al.	1976	Soybean	Metribuzin	F2, BC1F2
Geadelmann, J. L. and R. N. Andersen	1977	Corn	Diclofop-methyl	F2
Scott, K. R. and P. D. Putwain	1981	Common groundsel	Simazine	F2
Souza Machado, V. et al.	1982	Tomato	Metribuzin	F2, BC1F1
Busch, R. <u>et al</u> .	1984	Wheat	Difenzoquat	F4

also contains examples of polygenic inheritance of herbicide tolerance. These include: Comstock and Anderson (1968) (flax tolerance to atrazine), Faulkner (1974) (perennial ryegrass tolerance to paraquat), Schooler et al., (1972) (foxtail barley tolerance to siduron), and Gaedelmann et al. (1977) (maize tolerance to diclofop-methyl).

1.4.5.2 Cell Culture. In recent years, the technology of cell culture has been used as a tool to select herbicide tolerant crop lines 'in vitro'. Herbicides that interfere with basic metabolic activities can be expected to inhibit the growth of cultured cells as well as of the whole plant (Chaleff and Ray, 1984). Meredith and Carlson (1982) outlined a commonly used procedure as follows. A callus culture is established from the tissue of a plant. The callus is dispersed in a liquid medium. Using an appropriate enzymatic treatment, cell walls can be removed releasing protoplasts. These protoplasts are in many ways unicellular organisms which have the potential to develop into entire plants in the appropriate nutrient medium. Selecting herbicide resistant cell lines may be accomplished by adding the herbicide to the culture medium. Most cells will be killed, however, in the population, occasional spontaneous mutations can occur, producing herbicide tolerant variants which survive and proliferate.

This technique is prone to many problems and situations where apparent success becomes a failure. Meredith and Carlson (1982) described the following situations that researchers have faced.

- Tolerance occurring in cultured cells was lost when the cells were grown away from the herbicide for a generation,
- ii) plants could not be regenerated from apparently tolerant callus,

- iii) regenerated plants did not express the tolerance trait,
- iv) plants expressed the trait but the trait was not heritable.

Despite these difficulties, there are successful examples, listed in Table 4, in which herbicide tolerant plants were regenerated and were able to transmit the trait to following generations. It should be noted that this table is not necessarily a complete list. Chaleff and Ray (1984) developed chlorsulfuron tolerant tobacco plants that were not affected by a foliar application of 100 ppm chlorsulfuron. Normal plants were severely inhibited by 3 ppm. They suggested that this magnitude of difference is large enough to be referred to as 'resistance'.

1.4.5.3 Molecular Biology. The most recent research in the area of breeding herbicide resistant crops has involved techniques of molecular biology and genetic engineering. Duesing (1985) reported that these procedures involved the isolation of the specific gene that confers herbicide tolerance on a crop or weed species, and its transfer to a sensitive crop. Gene transfer could be accomplished using Agrobacterium tumefaciens vectors (Fraley et al., 1985).

This type of research is being conducted for glyphosate tolerance by at least two different research teams. Calgene researchers isolated a mutant gene from a glyphosate tolerant strain of Salmonella typhimurium bacteria. This gene codes for 5-enolpyruvylshikimate-3-phosphate (EPSP synthase), the enzyme which glyphosate inhibits. The mutant gene caused the EPSP synthase enzyme to differ by a single amino acid. This change made it less inhibited by glyphosate (Comai et al., 1985). Calgene recently received a U.S. patent for this gene, the first

TABLE 4. Herbicide resistance in plant cell cultures.

Reference	Year	Crop	Herbicide
Chaleff, R. S. & M. F. Parsons	1978	Tobacco	picloram
Radin, D. N. & P. S. Carlson	1978	Tobacco	phenmedipham bentazon
Chaleff, R. S. & T. B. Ray	1984	Tobacco	chlorsulfuron sulfometuron-methyl

gene engineered for crop agriculture (Anon., 1985). It was named 'GlyphoTol'. The GlyphoTol gene has already been successfully introduced, via Agrobacterium rhizogenes vectors, into cells of soybean, cotton, tomato, tobacco, and certain tree species. Calgene and DeKalb-Pfizer Genetics have agreed to develop and market glyphosate tolerant varieties of hybrid corn (Fishbein, 1985). Field trials with some of those crops are to begin in 1986.

Monsanto researchers have a similar objective but are using a different strategy. They have induced petunia plants to overproduce EPSP synthase. The 'hybrid' gene, that caused this overproduction, was then transferred into crop plants via plasmid vectors (Marx, 1985).

Studies of this nature are also being considered for other important herbicide families including the triazines and sulfonylureas (Marx, 1985).

2.0 MATERIALS AND METHODS

2.1 The Effect of Rate and Stage of Application of Diclofop-Methyl Applied to Savena 1 Oats

The source of diclofop-methyl tolerance studied in this project was a recently registered Australian oat named 'Savena 1'. The pedigree of Savena 1 is West x (West x New Zealand Cape/23)/28 and was developed by Barr (Personal Communication, 1985) of the South Australia Department of Agriculture. The tolerance trait was derived from New Zealand Cape. It was being used as a source of resistance to the cereal cyst nematode (Heterodera avenae) in a backcrossing program to the variety West. Progeny of this cross were also found to display tolerance to diclofop-methyl. Barr (1985) screened many oat genotypes from 1980 to 1985, using several graminicides, and found the diclofop-methyl tolerance of Savena 1 to have the greatest practical value.

2.1.1 1984 Study

Field plots were established at the University of Manitoba research site at Portage la Prairie, Manitoba (soil type: Neuhorst clay loam - 25% sand, 44% silt, 31% clay, organic matter 8.5%, pH 7.4) to study the effect of rate and stage of application of diclofop-methyl applied to Savena 1 oats. The experiment was placed on land that had been fallow in 1983 and was fertilized with 290 kg/ha granular 27-27-0 fertilizer broadcast and incorporated in spring.

The experiment was designed as a randomized complete block with four replicates and ten treatments per replicate. One guard row plot was placed at each end of each replicate. Individual plots were

measured to a size of 2.8 x 5.0 m. A 2.0 m alley separated each of the four replicates. Approximately 800 grams of wild oat seeds (percentage germination rate = 86%) collected in 1983 were hand-broadcast over each replicate of the experiment. This was approximately $30\text{--}60~\text{seeds/m}^2$ and was applied to provide a moderate to heavy infestation of competitive weeds. Wild oats were incorporated to 4-5 cm by using double disk cultivation. The soil was then levelled using diamond harrows.

The experiment was seeded using an International Harvester field drill of 2.4 m in width, the 16 seed runs were spaced 15 cm apart. The drill was calibrated to seed Savena 1 oats at a rate of 55-60 kg/ha. The entire experimental area was seeded to Savena 1 oats on May 14 to a depth of 3-5 cm. Seed was placed into moist soil.

Ten treatments, including two controls were used. The treatments consisted of four rates of diclofop-methyl 0.4, 0.5, 0.6, and 0.7 kg/ha (formulated as Hoegrass (R) 284 g/l E.C.) applied at the 3-leaf and 5-leaf stages of wild oats. The treatments were randomized in each of the four blocks. Treatments applied to the 3-leaf stage were sprayed on June 7, those at the 5-leaf stage on June 14. Plots were sprayed using a bicycle plot sprayer equipped with a four nozzle (Teejet SS80015) boom. The sprayer was calibrated to deliver 115 l/ha solution at a pressure of 40 psi (275 Kpa) and a walking speed of 6 km/hr.

An overall treatment of bromoxynil octanoate plus MCPA ester (Buctril M) at 0.56 kg/ha was applied to the experiment on June 11 to provide broadleaf weed control.

Plots were visually rated for crop tolerance on a weekly basis for three weeks beginning one week after diclofop-methyl application. The

rating scale used was similar to that used by most weed control researchers in Western Canada. A single digit rating, ranging from 1-9, was given to each plot. On this scale, '9' represented complete tolerance, '1' complete kill and '7' was regarded as the minimum 'commercially acceptable' rating. Morphological injury symptoms were also recorded.

Dry matter sampling of each plot was conducted once the wild oats were fully flowering (July 18-19). This allowed for the easy identification and separation of weed and crop. Sampling consisted of pulling all plants from a 1 m^2 area from a representative location near the rear of each plot. Oats and wild oats were separated, roots were removed with pruning shears, oats and wild oats were placed in separate bags and air dried until weights were constant. Air dry weights of oats and wild oats were then measured. Counts were also taken of the oats and wild oats culms/ m^2 .

Plots were sampled for oat grain yield in a similar manner.

Harvesting was conducted on August 8-9. This corresponded to the growth stage when the majority of the oat peduncles had turned to a yellow-brown color. Two 1 m² samples were pulled from a representative location near the front of each plot. Roots were removed and samples were placed in bags and air dried. Seed was threshed and dried to a constant moisture level. Due to the presence of wild oats and some volunteer wheat seeds in the samples, subsamples were taken and dockage was removed by hand. The per cent dockage by weight was subtracted from the gross weight of the sample.

Analysis of variance and Duncan's Multiple Range Test were

conducted for oat dry weight, wild oat dry weight, oat culm counts, wild oat culm counts, and oat grain yield.

2.1.2 1985 Study

The 1984 study was repeated in 1985. The procedure used was identical with the following exceptions: seeding date - May 21; spraying dates - June 12 (3-leaf), June 20 (5-leaf), June 24 (overall treatment); dry matter sampling dates - July 30-31, and grain harvesting dates - August 21-22. A modification of the grain harvesting procedure was also implemented in 1985. A hand sickle was used to cut stems above the soil level. Since only grain yield was being measured, the height of cutting was not critical. This system was more efficient than pulling plants and removing roots with pruning shears. As well, wild oats and any volunteer cereal plants were removed prior to bagging of samples. This was done to avoid contamination of grain samples.

The following measurements were taken on plots in 1985 that were not recorded in 1984: i) days to heading, and ii) average plant height after heading.

In 1985, Savena 1 oats were exposed to a substantial stem rust (Puccinia graminis f. sp. avenae) and crown rust (Puccinia coronata f. sp. avenae) infestation. Symptoms were noted on July 23 and the entire experiment was treated with the fungicide propiconazole (25% E.C.) (trade name: Tilt) on July 24. The fungicide was applied at a rate of 0.5 l/ha using a sprayer attached to the 3-point hitch of a tractor with the boom 40-50 cm above the crop canopy. The solution was applied in a volume of 120 l/ha at 300 Kpa using Teejet SS80015 nozzles.

2.1.3 The Effect of Rate and Stage of Application of Diclofop-methyl Applied to Savena 1 Oats Under Weed-Free Conditions

A second activity study was conducted in 1985. The purpose of this study was to examine the effects of diclofop-methyl on Savena 1 oats in the absence of weed competition, ie. a crop tolerance study. This experiment utilized the same design and treatments as the experiment in Section 2.1.2, except that it was seeded on land that had not previously been infested with wild oats. The only other differences in methodology were: i) dry matter sampling date: July 29, and ii) grain harvesting date: August 22.

2.2 The Evaluation of Various Avena sativa Genotypes for Tolerance to the Application of 0.4 and 0.7 kg/ha Diclofop-Methyl

An experiment was conducted in the summer of 1984 at the
Agriculture Canada Station at Glenlea to evaluate a total of 240 oat
genotypes for possible tolerance to diclofop-methyl. The material
tested was a diverse group of genotypes which consisted of: i) 157
advanced lines from oat breeding programs across Canada and the North
Central United States, ii) 81 entries from the Agriculture Canada
Historical Oat Collection - a group of cultivars that are currently
being grown in Canada as well as cultivars that have been grown in the
past, iii) the line (Irwin x (West x New Zealand Cape/42)) x West/24; it
was also sent to Winnipeg by Barr, and iv) Savena 1.

The soil type at the Glenlea station is Osborne heavy clay (4% sand, 25% silt, 71% clay, organic matter 5.1%, pH 7.4).

Six replicates of each of the 240 lines were seeded in two physically separate blocks in the field with three replicates in each

block. This allowed for the use of two different spray rates. Seeding was conducted using a 'Seedmatic' drill. This drill had six seed runs and produced one meter row plots, with 23.5 cm row spacings, followed by a one meter wide alley. Approximately 30 seeds were planted in each row. Control varieties were randomly assigned to every 29th and 30th plot. Controls consisted of Savena 1 and Harmon, a commonly grown oat cultivar in Western Canada.

Plots were seeded on May 9 and sprayed on June 5 when the majority of the rows were in the 3-leaf stage. Approximately one-half (13) of the pairs of control plots were covered with plastic at the time of spraying to prevent herbicide contact with the plants and thus provided unsprayed control plots. Spraying was conducted using a bicycle plot sprayer with a seven nozzle boom (as in Section 2.1.1). One block was treated with diclofop-methyl at a rate of 0.4 kg/ha, the other with 0.7 kg/ha.

Individual rows were rated on June 18 and June 27 (13 and 22 days after spraying) using the rating system described earlier (Section 2.1.1). Ratings were recorded on a portable TRS80 computer and uploaded into the Agriculture Canada main frame computer for future analysis.

The most tolerant entries were harvested after heading to determine dry matter weights. Only entries in which at least two of three replicates (at either rate) had visual ratings of '4' or greater on June 27 were harvested. In addition, all covered checks were harvested. The harvesting procedure consisted of cutting out a representative 50 cm section from each row, drying the sample, and weighing it.

2.3 Inheritance of Diclofop-Methyl Tolerance

In an attempt to study the inheritance of diclofop-methyl tolerance, the following crosses and reciprocal backcrosses were made by Dr. R. McKenzie, Agriculture Canada, Winnipeg.

Savena 1 x 0T216 Savena 1 x 0T228 Savena 1 x 0T231 Savena 1 x 0T233

(Savena 1 x OT216) x OT216 OT216 x (Savena 1 x OT216) (Savena 1 x OT228) x OT228 OT228 x (Savena 1 x OT228) (Savena 1 x OT231) x OT231 OT231 x (Savena 1 x OT231) (Savena 1 x OT223) x OT233 OT233 x (Savena 1 x OT233)

Crosses were made in 1983 (fall) and backcrosses in 1984 (spring).

OT216, OT228, OT231, and OT233 were advanced lines of differing genetic backgrounds from the Winnipeg breeding program. Reciprocal backcrosses were conducted to test for possible cytoplasmic inheritance of the tolerance trait.

The following numbers of F1 plants were grown. Several were used in making backcrosses.

Cross	Number of F1 Plants Grown
Savena 1 x OT216	19
Savena 1 x OT228	24
Savena 1 x OT231	26
Savena 1 x OT233	17

From this point onward, the progeny of one of these individual crossed or backcrossed plants will be referred to as a 'line'. Records of each line were kept by the pedigree method.

From the F1 and backcross (BC) seeds, F2 and BC1F1 plants were

grown in the field at Glenlea in 1984. Seeding was conducted on May 18 using the procedure described in Section 2.2. One to three rows were grown from each line (1-15 seeds/row), depending on the amount of seed available. Small plastic label stakes were placed into the soil between closely adjacent plants when in the seedling stage. This allowed for the identification and harvest of individual plants at maturity. Plants were pulled from the soil at maturity (August 20-27), labelled, allowed to dry, and threshed. This seed was then used in the inheritance study conducted in 1985.

A portion of the F2 plants from each of the four crosses were grown in rows adjacent to the screening experiment (Section 2.2). One to five rows were grown (approximately 20 seeds/row) from lines with the greatest seed availability. Half of the rows were sprayed with diclofop-methyl at a rate of 0.4 kg/ha and half at 0.7 kg/ha. Spraying was conducted in the same operation as the screening experiment, when plants were in the 3-leaf stage. Individual plants were rated for crop tolerance 20 days after spraying using the system described earlier (Section 2.1.1). Surviving plants were individually harvested at maturity as described above.

The 18 plants which produced the greatest amount of seed (at least two from each of the four crosses) were advanced one generation in a growth room pot study during the winter of 1984. Approximately 15 seeds of each of the 18 entries were planted per pot in each of two pots. The soil mixture used was 2 parts clay loam: 1 part sand: 1/2 part peat. Plants were thinned to 12 per pot at the 2-leaf stage. At the 2 1/2 - 3 leaf stage all pots were sprayed with diclofop-methyl (0.7 kg/ha) using

a laboratory bench sprayer (Teejet 8002 nozzle) in a water volume of 141 l/ha. Individual plants were rated for tolerance 16 days after spraying. The five highest rating plants per entry were allowed to grow to maturity, the remainder were cut and discarded. Seed from individual plants was harvested at maturity.

2.3.1 1985 Inheritance Study

In 1985 a field experiment was conducted at Glenlea to examine the inheritance of diclofop-methyl tolerance.

The following material was studied in 1985:

- 1. F3 lines from F2 plants grown in 1984,
- 2. BC1F2 lines from BCF1 plants grown in 1984,
- BC1F3 lines from BC1F2 plants that had been advanced a generation in New Zealand in the winter of 1984-85,
- F3 lines from F2 plants which had been sprayed with diclofop-methyl in 1984 and,
- 5. F4 families from F2 plants sprayed in the summer of 1984 and again as F3 lines in the winter of 1984-1985.

Description in Table 5.

Four replicates of each line were grown, two replicates in one block of the field, two in a second block. The separate blocks allowed for the treatment of plots with two different rates of diclofop-methyl. The two replicates of each line in a given cross were randomized. One meter row plots were seeded on May 15 using the Seedmatic drill described in Section 2.2. The width of the experiment was 100 plots. Control plots consisted of one row of Savena 1 adjacent to one row of the susceptible parent involved in the cross that surrounded the check

TABLE 5. Material examined in 1985 study of diclofop-methyl tolerance in oats.

Cross	Generation	Number of Lines Studied
Savena 1 x OT216	F3	84
Savena 1 x OT228	F3	150
Savena 1 x OT231	F3	150
Savena 1 x OT233	F3	150
Savena 1 x OT216 ²	BC1F2	77
OT228 x (Savena 1 x OT228)	BC1F2	31
Savena 1 x OT228 ²	BC1F2	87
OT231 x (Savena 1 x OT231)	BC1F2	34
Savena 1 x OT231 ²	BC1F2	62
OT233 x (Savena 1 x OT233)	BC1F2	20
Savena 1 x OT233 ²	BC1F2	62
OT216 x (Savena 1 x OT216) Savena 1 x OT2282	BC1F3	50
	BC1F3	50
OT231 x (Savena 1 x OT231) Savena 1 x OT233 ²	BC1F3	50
Savena 1 x U1233	BC1F3	50
Savena 1 x OT216 Savena 1 x OT228	F3 ^a	11
Savena 1 x OT228 Savena 1 x OT231	F3 ^a	24
	F3 ^a	33
Savena 1 x OT233	F3 ^a	7 5

 $^{^{\}mathrm{a}}\mathrm{Sprayed}$ with diclofop-methyl in the F2 generation.

rows. Control plots were placed after every 20th plot, ie. after every plot number ending in 10, 30, 50, 70, 90 (controls were not given plot numbers). The F4 families from the material advanced in the growth room were seeded in the block to be treated with the higher rate of the herbicide. A single plot (20-25 seeds) was sown from each of the five plants grown to maturity.

When the majority of plants were in the 2-leaf stage, notes were taken on missing rows, thin rows, and rows that appeared to have emerged late.

Spraying was conducted when the majority of the plants were in the 2½-leaf stage. The two replicates treated with 0.4 kg/ha diclofop-methyl were sprayed on June 6, the two replicates treated with 0.7 kg/ha on June 7. Spraying was conducted with a bicycle plot sprayer with a 7 nozzle boom as described in Section 2.1.1. Approximately one-fifth of the pairs of check plots were covered with plastic at the time of spraying to prevent herbicide contact, and thus provide unsprayed control plots (as in Section 2.2). An overall treatment of Buctril M (0.45 kg/ha) was applied to the entire experiment on June 11 to control broadleaved weeds present. The rate used was 80% of the recommended rate for oats (Anon., 1986a), to avoid the possibility of injury symptoms appearing on the oats.

Plots were visually rated for crop tolerance on June 19 (0.4 kg/ha plots) and June 21 (0.7 kg/ha plots). The rating scale and TRS80 computer data collection system were used (see Section 2.2). Control plots were rated in the same manner on June 18. A second rating was conducted on July 3-4 (0.7 kg/ha plots) and July 4-8 (0.4 kg/ha plots).

To account for plots that were non-uniform with respect to diclofop-methyl tolerance, a modified rating system was used for the second rating. Plots that appeared uniform were rated as previously described. Non-uniform plots were given a two digit rating. The first digit represented the tolerance of the majority of plants in the row, the second digit represented the tolerance of the remaining plants. A single 'non-representative' plant in a plot was ignored since its presence may have been due to a seeding error, late emergence, or being missed by the spray treatment. Control plots were also rated in this way on July 2.

To provide additional information to the 1985 inheritance study a late-seeded trial was conducted at Glenlea. A single replicate of lines from the F3, BC1F2, and BC1F3 crosses described in Table 5 with sufficient seed, was sown using the Seedmatic drill (Section 2.2) at Glenlea on August 16. Control plots consisted of one row of Savena 1 adjacent to one row of the susceptible parent of the cross that surrounded the check rows. Control plots were placed after every 20th plot.

In addition a small number of seeds from growth cabinet crosses between Savena 1 and OT216, OT228, OT231, and OT233 were sown in rows adjacent to the BCIF3 lines. These seeds would produce F1 plants.

Plots were sprayed with diclofop-methyl at a rate of 0.7 kg/ha on September 10. Oats were in the two to three leaf stage. Spraying was conducted with a bicycle plot sprayer and seven nozzle boom as described in Section 2.1.1. A portion of the control plots were covered with plastic at the time of spraying.

Plots were noted for crop tolerance on October 7. The ratings consisted only of the following: 'susceptible', 'segregating/ intermediate' and 'tolerant'.

2.4 <u>Alternative Oat Genotypes Reported to Show Diclofop-Methyl Tolerance</u>

During the course of this project, alternative sources of diclofop-methyl tolerance were tested. Somody et al., (1984) screened a large number of Avena fatua and A. sterilis accessions from the United States with several graminicides. Accession 762 was reported to have the highest level of diclofop-methyl tolerance among 88 accessions tested with only a 17 per cent reduction in fresh weight two to three weeks after spraying (0.8 kg/ha). A small quantity of seed of this accession was obtained from these researchers.

The seed source was increased before further investigations could be conducted. One seed of Accession 762 was planted in each of three pots and grown to maturity in a growth cabinet during the winter of 1984. At maturity seeds were removed from the three plants and stored in separate envelopes. This seed was then labelled AC762-1, AC762-2, and AC762-3.

In anticipation of obtaining useful diclofop-methyl tolerance from this <u>Avena fatua</u> accession, the following crosses were made during the winter and spring of 1985.

```
OT231
         x AC762-1
AC762-1 x
            OT231
OT233
         X
            AC762-2
AC762-2
            OT233
         X
OT228
         X
            AC762-3
AC762-3
         X
            0T228
(OT231 \times AC762-1) \times
                      OT231
(0T233 \times AC762-2) \times
                       OT233
(OT228 x AC762-3) x OT228
Savena 1 x AC762-2
Savena 1 x AC762-3
```

In the summer of 1985, a greenhouse study was conducted to compare the diclofop-methyl tolerance of AC762-1, AC762-2, AC762-3, Savena 1, Harmon (a diclofop-methyl susceptible Avena sativa cultivar), Elen (an A. sativa cultivar from the United Kingdom reported to have diclofopmethyl tolerance (Taylor and Codd, 1985)), and #35, a uniformly tolerant BC1F3 line selected in the 1985 inheritance study (pedigree: OT231 x (Savena 1 x OT231)). Approximately 100 seeds of each experimental line were placed on moist filter paper and stored in a refrigerator for four days. Avena fatua seeds were first dehulled by hand. These measures were taken to break dormancy, a trait that had been detected in preliminary studies (not reported). Ten pots of each of the seven entries were seeded in a soil mixture of 2 parts clay loam : 1 part sand: 1/2 part peat. Pots were thinned to four plants/pot at the 2-leaf stage. A completely randomized design was used. From the time of emergence to the end of the experiment, pots were rotated on the greenhouse bench once every three days to minimize edge effects. Five pots of each entry were sprayed (the remaining five were unsprayed controls), with diclofop-methyl (0.7 kg/ha) at the 3-leaf stage using a cabinet sprayer (Teejet 8002 nozzle) in a water volume of 137 l/ha. Plants were harvested on an individual plant basis 20 days after

treatment, bagged, dried, and weighed. Per cent reduction in dry matter was calculated and analysis of variance was conducted to determine whether the seven plant types differed with respect to diclofop-methyl tolerance.

3.0 RESULTS

3.1 The Effect of Rate and Stage of Application of Diclofop-Methyl Applied to Savena 1 Oats

3.1.1 <u>1984 Study</u>

Visual ratings of Savena 1 crop tolerance one and three weeks after application of diclofop-methyl are listed in Appendix Table 1. These ratings indicated that Savena 1 oats were injured by diclofop-methyl application (one week rating) both at the 3-leaf and 5-leaf stages, however, after three weeks, ratings for all treatments had increased and most treatments were at or near the commercially acceptable '7' rating.

One week after diclofop-methyl application, chlorotic patches covering half or more than half of the leaf were visible on the youngest leaves of Savena 1 plants (third leaf in the 3-leaf treatments, fifth and sixth leaf in the 5-leaf treatments) in all plots. Older leaves displayed lesser amounts of chlorosis (Plate 1a). A slight amount of necrosis occurred on leaf tips of many plants. Heights of sprayed plants were somewhat reduced (visual assessment) in comparison to unsprayed plants. These injury symptoms increased in magnitude as the diclofop-methyl rate was increased (Plates 1b and 1c). Two weeks after spraying, Savena 1 plants appeared to have recovered. The youngest leaves were green and maturity was approximately equal to the unsprayed controls. By the third week, very little chlorosis or necrosis remained visible. A height reduction of approximately 15-25% had occurred. Plots treated at the 5-leaf stage appeared more vigorous than those treated at the 3-leaf stage.

Plate 1a. Diclofop-methyl injury symptoms on Savena 1 oats one week after treatment (3-leaf stage, 0.4 kg/ha). Chlorosis.

Plate 1b. Diclofop-methyl injury symptoms on Savena 1 oats one week after treatment (3-leaf stage, 0.4 kg/ha). Chlorosis and necrosis.

Plate 1c. Diclofop-methyl injury symptoms on Savena 1 oats one week after treatment (3-leaf stage, 0.7 kg/ha). Chlorosis and necrosis.







At heading, sprayed plots and unsprayed plots did not differ significantly in terms of oat dry weight per m^2 (Table 6). Differences were detected in oat culm counts per m^2 . A leaf stage by rate interaction existed. At the 3-leaf stage, lower rates increased culm counts while at the 5-leaf stage, the higher herbicide rates increased culm counts.

Culm counts and dry weight of wild oats were reduced by all herbicide treatments by comparison with the control. The extent of reduction was greater in plots treated at the 3-leaf stage than at the 5-leaf at the 0.4 and 0.5 kg/ha rates. The two highest rates of diclofop-methyl, applied at the 5-leaf stage, resulted in wild oat control equal to that at the 3-leaf stage. Wild oat control, measured as dry weight of wild oats per m² in sprayed vs. unsprayed plots, ranged from 73% (0.4 kg/ha, 5-leaf stage) to 100% (0.4 kg/ha, 3-leaf stage).

Grain yields of all sprayed plots significantly exceeded the unsprayed controls. Yield increases ranged from 18% (0.6 kg/ha, 3-leaf stage) to 33% (0.5 kg/ha, 5-leaf stage). Yields did not differ significantly at the two application stages. When controls were removed from the analysis, yields were found to be significantly greater when the herbicide was applied at rates of 0.4 or 0.5 rather than 0.6 or 0.7 kg/ha.

3.1.2 <u>1985</u> Study

Visual ratings of Savena 1 crop tolerance and associated injury symptoms were similar to those described in Section 3.1.1 (Appendix Table 1).

In 1985, as in 1984, oat dry weights (per m^2) at heading did not

The effect of rate and stage of application of diclofop-methyl on wild oat control and Savena 1 oat tolerance (1984 Study). TABLE 6.

		Ö	Oat Assessments		Wild Oat Assessments	ssessments
Diclofop- methyl Rate (kg/ha)	Leaf Stage of Application1	Culm Count ² (m ²)	Dry Weight ² (g/m ²)	Grain Yield ³ (g/m ²)	Culm Count ²	Dry Weight ²
0.0	Control	490.6 c	611.8	247.6 C	131.8 a	160.1.8
0.4	က	613.5 a	649.5	319.5 ab		
0.5	က	581.5 ab	604.5		ာ (၁) (၁)	
9.0	က	515.8 bc	534.4	292.5 b		0 8 · 9 0 · 8 · 9
0.7	ဇာ	489.3 c	510.3	304.0 ab		
Mean (Mean (3-leaf)	550.0	574.7	307.1	5.0	3.0
0.4	ស	488.8 c	573.3	308.3 ab	44.0 b	43.4 b
0.5	လ	515.8 bc	609.5	328.0 a		
9.0	വ	561.0 abc	622.0	298.5 b	26.0 bc	
0.7	വ	574.5 ab	600.5	295.8 b	28.3 bc	19.6 bc
Mean (Mean (5-leaf)	535.0	601.3	307.7	34.1	29.5
L.S.D.		74.3	n.s. 81.3	25.9	24.8	25.1

Means within columns followed by the same letter do not differ significantly at P=0.05 according to Duncan's Multiple Range Test.

¹Wild oat leaf stage. ²Taken on July 18–19 when wild oats were flowering. ³At maturity.

differ significantly among sprayed or unsprayed treatments (Table 7). A rate effect was detected in that five of the eight herbicide treatments significantly exceeded the control in oat culm count per m^2 .

As in the 1984 study, diclofop-methyl treatments significantly reduced wild oat culm counts and dry weights. Reduction in dry weight of wild oats as compared to control plots ranged from 66% (0.4 kg/ha, 3-leaf stage) to 93% (0.7 kg/ha, 3-leaf stage). In 1985 there were no significant differences between treatments at the 3-leaf and 5-leaf stages. When control plots were removed from the analysis the 0.4 kg/ha treatment at the 3-leaf stage was found to be equal in wild oat dry weight to the 0.4 kg/ha treatment at the 5-leaf stage, but significantly greater than all other treatments.

Only two treatments (0.4 and 0.5 kg/ha, 3-leaf stage) exceeded the control in terms of oat grain yield at maturity. Yield increases in these treatments were 22% and 18%, respectively. In this trial, a significant leaf stage effect was observed. Grain yields tended to be greater in plots treated at the 3-leaf stage.

The two additional agronomic traits measured in 1985, plant height and days to heading, were significantly affected by diclofop-methyl application. The height of Savena 1 oat plants at maturity was significantly reduced by the herbicide at all four rates and at both treatment stages. Average height reductions ranged from 5.7 cm (0.4 kg/ha, 3-leaf stage) to 11.5 cm (0.7 kg/ha, 3-leaf and 5-leaf stage). The effects tended to increase with increasing diclofop-methyl rate. Heading of Savena 1 oats was delayed somewhat by the herbicide application. All treatments were significantly later in reaching

The effect of rate and stage of application of diclofop-methyl on wild oat control and Savena 1 oat tolerance (1985 Study). TABLE 7.

)	Oat Assessment	ınt		Wild Oat	Wild Oat Assessment
Diclofop- methyl Rate (kg/ha)	Leaf Stage of Application ¹	Culm Count ² (m ²)	Dry Weight ² (g/m ²)	Grain Yield ³ (g/m ²)	Height ³ (cm)	Days to Heading	Culm Count ²	Culm Count ² Dry Weight ² (m ²) (g/m ²)
0.0	Control	444.5 C	725.8	212.6 c	84.5 a	52.0 c	84.0 8	160.4 a
0.4	က	494.3 abc	737.6	259.3 a	78.8 b	53.0 ab		
0.5	က	514.8 ab	753.2	250.3 ab	74.3 C	53.5 a		
9.0	က	543.3 a	754.1	240.4 abc	75.8 bc	53.0 ab	17.3 b	23.4 b
0.7	က	491.8 abc	675.0	209.0 c	73.0 с	53.3 ab		
Mea	Mean (3-leaf)	511.1	730.0	239.8	75.5	53.2	21.8	28.8
0.4	വ	482.5 bc	755.5	219.4 bc	75.8 bc	53.0 ab	30.5 b	34.4 h
0.5	ເລ	514.0 ab	764.0	208.1 c	75.0 bc	52.5 bc	23.3 b	
9.0	വ	506.8 ab	750.4	220.0 bc	74.3 C	53.0 ab	22.5 b	19.3 b
0.7	വ	525.8 ab	788.8	204.9 c	73.0 c	53.3 ab	19.3 b	15.9 b
Меа	Mean (5-leaf)	507.3	764.7	213.1	74.5	53.0	23.9	23.4
L.S.D.		52.4 n.s	n.s. 87.8	31.3	3.6	0.7	25.3	42.5

Means within columns followed by the same letter do not differ significantly at P = 0.05 according to Duncan's Multiple Range Test.

 $^{1}_{2}\mbox{wild}$ oat leaf stage. $^{2}_{2}\mbox{Taken}$ on July 30-31 when wild oats were flowering. $^{3}_{4}\mbox{At}$ maturity.

flowering than control plots. This delay ranged from 0.5 (0.5 kg/ha, 5-leaf stage) to 1.5 days (0.5 kg/ha, 3-leaf stage).

3.1.3 The Effect of Rate and Stage of Application of Diclofop-methyl Applied to Savena 1 oats under Weed-Free Conditions

Visual ratings of Savena 1 oats are presented in Appendix Table 1. This experiment assessed the effects of diclofop-methyl on Savena 1 oats without the interference of wild oat competition. At flowering, total shoot dry weights in plots treated with the two highest rates of diclofop-methyl (0.6 and 0.7 kg/ha) at the 3-leaf stage and the highest rate (0.7 kg/ha) at the 5-leaf stage were the only treatments to significantly reduce dry matter in comparison to unsprayed controls (Table 8). Overall, herbicide-induced reductions in dry matter ranged from 4% (0.5 kg/ha, 3-leaf stage) to 18% (0.7 kg/ha, 3-leaf stage). Savena 1 culm counts per m² were not significantly affected by the herbicide treatments.

Plant height and days to heading were affected in a similar manner as described in Section 3.1.2. Diclofop-methyl significantly reduced plant height in six of eight treatments. Height reductions ranged from 2.5 cm (0.5 kg/ha, 3-leaf stage) to 8.3 cm (0.7 kg/ha, 5-leaf stage). Treated plots were delayed by 0.7 to 2.0 days in time to flowering.

None of the diclofop-methyl treated plots differed significantly from the control in grain yield at maturity. Among treated plots, greater yields were obtained in plots treated with lower herbicidal rates. The 0.4 and 0.5 kg/ha treatments at the 3-leaf stage produced significantly greater yields than those treated with 0.7 kg/ha at either leaf stage.

The effect of rate and stage of application of diclofop-methyl on Savena 1 oat tolerance under weed-free conditions (1985 Study). TABLE 8.

Oat Assessments

te Stage of Application1 Control 3 3 3 3 3 Aean (3-leaf) Mean (5-leaf)				
Control 3 3 3 3 3 Mean (3-leaf) 5 5 5 5	$\begin{array}{ll} \text{Culm} & \text{Dry Weight}^2 \\ \text{unt}^2 \text{ (m}^2\text{)} & \text{(g/m}^2\text{)} \end{array}$	Grain Yield3 (g/m²)	Height ³ (cm)	Days to Heading
3 3 3 3 Mean (3-leaf) 5 5 5 5 5	521.5 876.6 a	247.4 abc	80.3 8	51.8 C
3 3 3 Mean (3-leaf) 5 5 5 5 5	563.8 831.0 a	275.8 a	75.3 abc	
3 3 Mean (3-leaf) 5 5 5 5 5 5	552.5 842.2 a	267.6 ab	77.8 ab	52.8 b
3 Mean (3-leaf) 5 5 5 5 5 Mean (5-leaf)	526.3 735.8 b	249.8 abc	72.3 c	
Mean (3-leaf) 5 5 5 5 5 5	531.8 722.9 b	229.3 c	72.3 c	53.8 a
5 5 5 5 Mean (5-leaf)	543.6 783.0	255.7	74.4	53.0
5 5 5 Mean (5-leaf)	557.8 783.7 ab	250.1 abc	71.5 c	52.5 b
5 5 Mean (5-leaf)	578.5 814.6 ab	246.0 abc	74.8 bc	
5 Mean (5-leaf)	586.0 831.9 a	235.4 bc	74.0 bc	
Mean (5-leaf)	534.5 735.8 b		72.0 c	
	564.2 791.5	240.4	73.1	52.8
L.S.D. n.s. 57.5	57.5 86.0	29.8	4.9	0.5

Means within columns followed by the same letter do not differ significantly at P = 0.05 according to Duncan's Multiple Range Test.

1 2 2 Taken on July 29 when Savena 1 was flowering. 3 At maturity.

3.2 The Evaluation of Various Avena sativa Genotypes for Tolerance to the Application of 0.4 and 0.7 kg/ha Diclofop-Methyl

The average visual ratings of each of the 240 genotypes treated with diclofop-methyl at 0.4 and 0.7 kg/ha are listed in Appendix Table 2. Average dry matter weights (measured at heading) and visual ratings of the 11 genotypes displaying the greatest diclofop-methyl tolerance are listed in Table 9. All other genotypes displayed severe injury symptoms with at least two of three ratings less than '4' at both rates. Although Savena 1 was the most tolerant genotype in the study, dry matter weight reductions of 34% (0.4 kg/ha rate) and 75% (0.7 kg/ha rate) occurred.

3.3 <u>Inheritance of Diclofop-Methyl Tolerance</u>

In 1985 a large study was undertaken at Glenlea in which F3 lines from crosses of the tolerant cultivar Savena 1 with four diverse, susceptible cultivars were tested. Backcrosses of F1's to the respective susceptible parent were also made and BC1F2 and BC1F3 progenies were studied. Diclofop-methyl injury symptom expression was greater four weeks after spraying (second rating) than two weeks after spraying (first rating), and was also more readily visible in the block treated with the higher rate of the herbicide. Thus, the two replicates in this block, rated on July 3-4, were the basis of the analysis which follows.

Lines from each cross were classified as being either
'susceptible', 'tolerant', or 'segregating/intermediate' with respect to
diclofop-methyl tolerance. Classification of plots as 'susceptible' or

TABLE 9. Shoot dry weights and visual ratings of the 11 genotypes displaying the greatest diclofop-methyl tolerance of 240 genotypes tested.

		Diclofop-	methyl Rate	
	0.4 k	g/ha	0.7 k	kg/ha
	Dry Weight ^a (g)	Visual Rating ^b	Dry Weight ^a (g)	Visual Rating ^b
Savena 1	34	6.2	13	5.2
Irwin ^C	30	5.7	11	4.7
Beacon	34*	4.3	9	3.7
Anthony	15	3.7	8	3.3
Hinoat	16*	4.0	6	3.3
Cartier	29*	4.7	_	2.3
Scotian	18	4.0		2.7
Woodstock	35*	4.0		2.3
Abegweit	31*	4.0	_	2.3
Victory	23*	4.0	_	2.3
ND810917	36*	3.7	_	2.7
Harmon (control)		1.8	-	1.4
Unsprayed Checks:		***************************************		
Harmon	84	9.0	64	9.0
Savena 1	59	9.0	52	9.0

aDry weight (g) of a representative 0.5 m row harvested on July 10, (average of 3 replicates, except Savena 1 and Harmon - 13 replicates).

b_{Visual} rating 22 days after spraying (number of replicates, as above).

 $c_{Irwin} = ((Irwin X (West X New Zealand Cape/42)) X West)/24$

^{*}Harvested six days later than others (July 16), therefore somewhat biased.

'tolerant' was based on the ratings of the susceptible and tolerant parents (Table 10). A particular hybrid line was classified as 'susceptible' if the two replicates of that line rated: 1, 2, 13, 14, or 15 at the 0.7 kg/ha rate. If only one of the two replicates was assigned one of these five ratings, data for this line treated at the 0.4 kg/ha rate was used. The line was considered susceptible if at least one of these two ratings was also one of the five listed above. Lines were classified as 'tolerant' if the two ratings at the 0.7 kg/ha rate were 5, 6, 7, or 8 (uniformly tolerant). If one of the two replicates was uniformly tolerant (the other non-uniform, for example, 73), the 0.4 kg/ha data was examined; if both of these replicates were uniformly tolerant, the line was considered 'tolerant'. However, if only one rated 5, 6, 7, or 8, the single replicate in the Glenlea (late) trial was used.

Lines which were neither susceptible nor tolerant were classified as 'segregating/intermediate'. Examples of tolerant, susceptible, and segregating plots are presented in Plates 2a and 2b. The classification of lines from each of the crosses is presented in Table 11.

The data in Table 11 were examined for goodness of fit to various theoretical genetic models, ie. hypotheses were tested to determine whether control of diclofop-methyl tolerance could best be explained by one dominant gene, one recessive gene, two genes or three or more genes. The possibility of cytoplasmic inheritance (as described by Beversdorf et al., (1980) for triazine tolerance in rapeseed) was discounted because the reciprocal backcrosses produced similar ratios of susceptibility to segregating/intermediate lines.

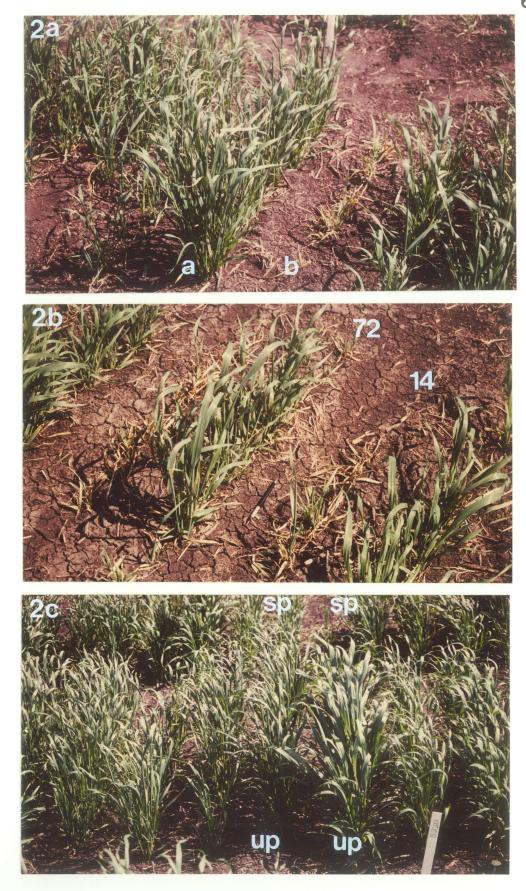
TABLE 10. Visual ratings of control plots of parental varieties treated with 0.7 kg/ha diclofop-methyl.

Susceptib	ole Parents	Toler:	ant Parent
Visual Rating	Number of Plots	Visual Rating	Number of Plots
1	50	5	6
2	10	6	43
13	11	7	40
14	20	57	2
15	10	64	4
		74	2
		75	4
Total	101	Total	101

Plate 2a. Examples of diclofop-methyl tolerant ('a', Savena 1) and susceptible ('b', OT228) plots (0.7 kg/ha).

Plate 2b. Examples of plots segregating for diclofop-methyl tolerance and their associated visual ratings.

Plate 2c. Examples of F3 lines, treated with diclofop-methyl (0.7 kg/ha), derived from F2 plants that had survived diclofop-methyl treatment in 1984. Note: This pertains to all plots shown except those labelled 'up' (unsprayed parent) or 'sp' (sprayed parent).



Classification of lines from crosses of Savena 1 with four susceptible parents with respect to diclofop-methyl tolerance. TABLE 11.

			Number of Lines	Lines		
Cross	Generation	Susceptible	Tolerant	Segregating/ Intermediate	Missing ^a	Total
	F3 F3 F3 F3 BC1F2	7 14 4 4	5 12 13 46	71 122 120 99		84 150 150 150
Savena 1 X 0T2282 Savena 1 X 0T2282 0T231 X (Savena 1 X 0T231) Savena 1 X 0T2312 0T233 X (Savena 1 X 0T233) Savena X 0T2332	BC1F2 BC1F2 BC1F2 BC1F2 BC1F2	6 9 21 3	0 2 0 1 2 1 1	69 69 14 15 38	00000	77 31 87 62 20
OT216 X (Savena 1 X OT216) Savena 1 X OT228 ² OT231 X (Savena 1 X OT231) Savena 1 X OT233 ²	BC1F3 BC1F3 BC1F3 BC1F3	22 21 8	= ผพพ	27 27 40 41	· •••	50 50 50
Savena 1 X 0T216 Savena 1 X 0T228 Savena 1 X 0T231 Savena 1 X 0T233	F3b F3 F3b F3b	0000	3 22 23 66	ଷ ଷ ଚ ଷ	0011	24 24 33 75

 $^{
m a}$ Missing lines include those whose rating was ambiguous.

 $^{^{}m b}_{
m Sprayed}$ with diclofop-methyl in the F2 generation.

The model which most closely fit the data was one in which diclofop-methyl tolerance was controlled by two genes. In a two gene model where each gene has some independent influence on tolerance, one would expect 1/4 of the BC1F2 lines to be homozygous for susceptibility and 3/4 to be segregating for one or the other or both genes controlling tolerance. The BC1F3 crosses should contain 1/64 tolerant, 25/64 susceptible, and 38/64 segregating lines. Most of the BC1F2 and BC1F3 data fit a two gene model. Chi-square (goodness of fit) and P values are listed in Table 12. The three sets of reciprocal backcrosses studied in the BC1F2 were initially tested separately and all fit the model with P > .01. For this reason, they were combined.

If two genes controlled tolerance, F3 plots would represent nine different F2 genotypes. If each of the two genes displayed an independent effect, one would expect only 1/16 of the lines to be as tolerant as Savena 1, 1/16 susceptible, and the remainder segregating or intermediate for tolerance. The OT216, OT228, and OT231 crosses fit this model, while the OT233 cross did not (Table 13).

In 1984, F2 plants from Savena 1 crosses with OT216, OT228, OT231, and OT233 were treated with diclofop-methyl at 0.4 and 0.7 kg/ha. The individual plant ratings are presented in Table 14, and ratings of the parents of these crosses scored on a plot basis in Table 15.

Prior to analysis, F2 data were grouped in the following manner based on the ratings of the parents of the crosses:

(a) At the 0.4 kg/ha rate, ratings 1 and 2 were classified as 'susceptible'; 3, 4, and 5 as 'intermediate'; and 6 and 7 as 'tolerant'. These values were chosen because the average rating of

TABLE 12. Chi-square testing of BC1F2 and BC1F3 lines to a two gene model for diclofop-methyl tolerance.

Cross						
	Generation	Segregating/ Intermediate	Susceptible	Tolerant	x ²	Q.
Savena 1 X OT216 ²	BC1F2	o: 40 ^a e: 57.75	37 19.25	1 1	21.82	<.001
OT228 X (Savena 1 X OT228) Savena 1 X OT228 ²	BC1F2	o: 96 ^a e: 87	20 29	1 1	3.72	.0510
OT231 X (Savena 1 X OT231) Savena 1 X OT231 ²	BC1F2	o: 66 ^a e: 72	30 24	l l	2.00	.1020
OT233 X (Savena 1 X OT233) Savena 1 X OT233 ²	BC1F2	o: 55 ^a e: 61.5	27 20.5	i I	2.75	.05 - 10
OT216 X (Savena 1 X 0T216)	BC1F3	o: 27 e: 29.69	22 19.53	1 0.78	0.62	.70 - 07.
Savena 1 X OT228 ²	BC1F3	o: 27 e: 29.69	21 19.53	2 0.78	2.25	.3050
OT231 X (Savena 1 X OT231)	BC1F3	o: 40 e: 29.69	8 19.53	2 0.78	12.29	.001 - 101
Savena 1 X OT233 ²	BC1F3	0: 41 e: 29.69	7 19.53	2 0.78	14.25	<.001

o: observed value

e: expected value

degrees of freedom: 1 (BC1F2), 2 (BC1F3)

 $^{
m a}$ These values include tolerant lines (Table 11) which were considered to be misclassifications.

TABLE 13. Chi-square testing of F3 lines to a two gene model for diclofop-methyl tolerance.

		Numbe	Number of Lines		
Cross	Susceptible	Segregating/ Intermediate	Tolerant	x ²	Ф
Savena 1 X 0T216	o: 7 e: 5.19	71 72.63	5 5.19	0.68	06 07.
Savena 1 X 0T228	0: 14 e: 9.25	122 129.5	12 9.25	3.69	.1020
Savena 1 X 0T231	0: 16 e: 9.31	120 130.38	13 9.31	7.10	.0105
Savena 1 X 0T233	0: 4 e: 9.31	99 130.38	46 9.31	155.17	<.001

o: observed value

e: expected value

degrees of freedom:

TABLE 14. Visual rating summary of crop tolerance of F2 plants to diclofop-methyl.

					1	Numbe	er of	Pla	ants	
						Visu	ıal F	Ratir	ngs	
	cross	Rate (kg/ha)	1	2	3	4	5	6	7	Total
Savena 1	X 0T216	0.4	20	22	11	5	4	1	0	63
		0.7	32	24	9	5	2	0	Ŏ	72
Savena 1	X 0T228	0.4	94	45	31	20	5	10	0	205
		0.7	108	74	30	24	13	5	0	254
Savena 1	X OT231	0.4	67	55	32	18	9	8	3	192
		0.7	149	56	22	24	10	1	0	262
Savena 1	X OT233	0.4	38	42	49	28	22	21	13	213
		0.7	82	34	51	20	18	8	0	213

TABLE 15. Average crop tolerance ratings (1984) of parents used in crosses.

Ave	rage ^a Visual Rating	
Parent	Diclofop-methy 0.4	l rate (kg/ha) 0.7
OT228	1.7	1.3
OT231	1.7	1.0
OT233	1.7	1.0
0T216 ^b	-	-
Savena 1	6.3	5.3

^aAverage of three replicates (one meter row plots).

 $^{^{}m b}{
m OT216}$ was not included in this test.

the susceptible and tolerant parents were 1.7 and 6.3, respectively.

(b) At the 0.7 kg/ha rate, a rating of 1 was classified as 'susceptible'; 2, 3, and 4 as 'intermediate'; and 5 and 6 as 'tolerant'. The parents averaged 1.1 (susceptible) and 5.3 (tolerant).

Because this grouping system did not precisely match the parental ratings, it was decided that a more accurate analysis would be obtained if the data for the two herbicidal rates was combined.

F2 data were used in an effort to determine whether tolerance was dominant to susceptibility, or vice versa. If tolerance was dominant, one would expect 9/16 of the F2 plants to be tolerant. By examining Table 14 it can be seen that this did not occur. However, if susceptibility was dominant to tolerance, 1/16 of the plants should have been tolerant, 9/16 susceptible, and 6/16 intermediate (assuming each gene has some effect alone). Combined data were analyzed using chi-square goodness of fit testing (with two degrees of freedom) to a 1/16 tolerant:6/16 intermediate:9/16 susceptible model (Table 16). The OT216, OT228, and OT231 crosses appeared to fit this model, while the OT233 cross did not.

The material which was treated in the F2 generation and grown out and treated as F3 lines in 1985 was not used directly in the explanation of diclofop-methyl tolerance. However, the fact that none of these lines was susceptible in the F3 generation, and nearly all were uniformly tolerant also suggests that tolerance was controlled by recessive genes (Plate 2c). The small number of lines that were found

TABLE 16. Chi-square testing of F2 plants to a two recessive gene model for diclofop-methyl tolerance.

			Number	of Plants	***	
	Cross	Tolerant	Intermediate	Susceptible	x ²	P
Savena	1 X OT216	o: 3	58	74		
		e: 8.44	50.63	75.94	4.63	.0510
Savena	1 X OT228	o: 28	184	247		
		e: 28.69	172.13	258.19	1.32	.5070
Savena :	1 X OT231	o: 22	161	271		
		e: 28.38	170.25	255.38	2.89	.2030
Savena :	1 X OT233	o: 60	204	162		
		e: 26.63	159.75	239.63	102.42	<.001

o: observed value

e: expected value

degrees of freedom: 2

to segregate, were probably intermediate in tolerance in the F2 generation, but survived to produce enough seed to be tested in 1985. It would appear from the data in Table 11 that most plants that were of only intermediate tolerance in the F2, either did not survive or produced only a small amount of seed.

Similar results were obtained for the F4 plots originally treated in the F2 and again as F3 lines in the growth room in the winter of 1984-85. Based on the five plant progenies of each of the 18 families tested, the majority were found to be uniformly tolerant, the remainder segregated for tolerance. Segregation was probably due to either a lack of adequate selection pressure in the F2 and F3 generations or selected plants being intermediate for tolerance in both generations.

3.4 <u>Alternative Oat Genotypes Reported to Show</u> <u>Diclofop-Methyl Tolerance</u>

Figure 1 depicts the dry matter reductions caused by 0.7 kg/ha diclofop-methyl to AC762-1, AC762-2, AC762-3, Savena 1, Harmon, Elen, and #35 measured 20 days after treatment. The Avena fatua Accession 762 and the A. sativa cultivar Elen, both reported to possess diclofop-methyl tolerance (Somody et al., 1984 and Taylor and Codd, 1985), were severely injured by the herbicide. Average dry matter reductions of 20 individual plants of each entry were 52% (AC762-1), 62% (AC762-2), 49% (AC762-3), and 48% (Elen). Savena 1 (22%) and #35 (14%) displayed much smaller dry matter reductions.

Due to the lack of tolerance displayed by AC762, further studies into the inheritance of its reputed tolerance were discontinued.

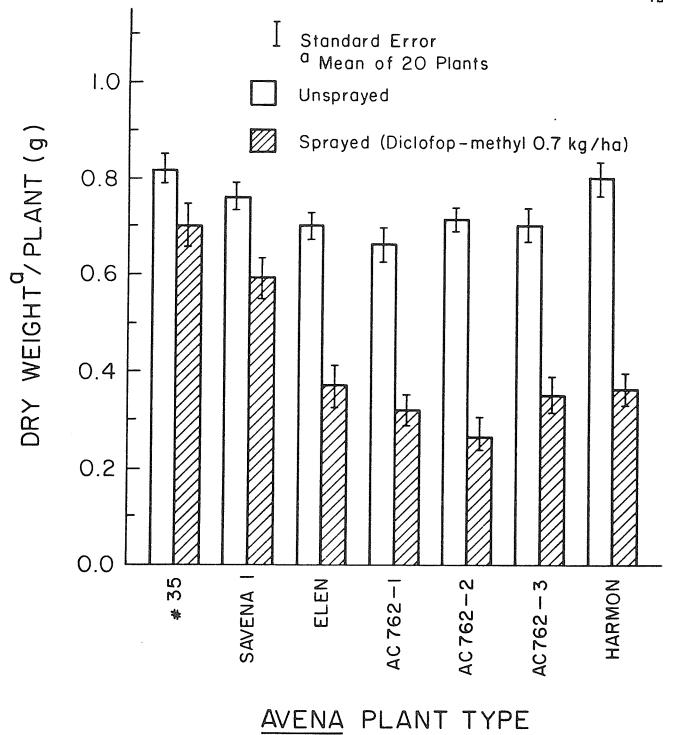


Figure 1. The effect of diclofop-methyl (0.7 kg/ha) on the shoot dry weight, 20 days after the treatment, of seven Avena entries.

4.0 DISCUSSION

4.1 The Effect of Rate and Stage of Application of Diclofop-Methyl Applied to Savena 1 Oats

The three experiments analyzing Savena 1 tolerance to diclofop-methyl in the presence or absence of wild oat weed competition will be discussed together.

The injury symptoms displayed by Savena 1 were similar to, but less severe than, diclofop-methyl injury to wild oats as described in Section 1.3.1. However, unlike wild oat plants, Savena 1 appeared normal in growth and development two to three weeks after herbicide application.

Diclofop-methyl application, in the experiments containing wild oat competition, resulted in an increased number of culms/m² of Savena 1 in many of the plots. A possible explanation for this phenomenon, is that the chemical removal of wild oats provided increased space for tillering of the crop. The tillering response seemed to be a factor of both herbicidal rate, leaf stage of application, and the corresponding levels of wild oat control and crop damage. In situations where wild oat control was near 100% and crop damage was minimal (for example, the 0.4 and 0.5 kg/ha 3-leaf treatments in 1984; and the 0.7 (1984) and 0.5, 0.6, and 0.7 kg/ha (1985) 5-leaf treatments), Savena 1 was able to tiller to fill the space previously occupied by wild oats. However, in situations where wild oat control was excellent but crop damage was significant (for example, the 0.7 kg/ha 3-leaf treatment in 1984), the crop seemed to be unable to increase its tillering rate. Finally, when wild oat control was only fair (0.4 and 0.5 kg/ha (1984) 5-leaf; and

0.4 kg/ha (1985) 3-leaf and 5-leaf treatments) insufficient space was provided for Savena 1 to tiller extensively even though it may have had the physiological capacity to do so.

This rationale is consistent with the 1985 weed-free data. In this experiment, herbicide application did not result in an increase in space for crop tillering (since wild oats were not present as competition). Correspondingly, Savena 1 culm counts in treated plots did not differ significantly from control plots.

In the two experiments with wild oat competition, the dry weight of Savena 1 oats at heading did not differ significantly in sprayed vs. unsprayed plots. The additional culms, in plots mentioned above, tended to be thinner and lighter than in control plots. The 1985 weed-free experiment most closely measured the effect of diclofop-methyl damage to Savena 1 plants. By flowering, plants in all plots except the 0.6 and 0.7 kg/ha 3-leaf and 0.7 kg/ha 5-leaf treatments had recovered to a level equal to that of the unsprayed check.

Seasonal differences were quite obvious in regard to wild oat control. Treatments applied at the 3-leaf stage in 1984 resulted in nearly 100% control, while in 1985 these treatments produced 63-93% control. In 1984, even the 0.4 kg/ha treatment provided excellent weed control while in 1985, only the 0.7 kg/ha rate controlled over 90% of the wild oat population. The major reason for these differences appear to be the weather conditions in the two seasons. During the week prior to, and two to three weeks after spraying, conditions in 1984 were ideal for diclofop-methyl activity, as described in Section 1.3.2.

Temperatures were warm and adequate moisture was available for vigorous

plant growth (Figure 2). Moisture conditions were again adequate in 1985, however temperatures were cooler. This favoured slower herbicidal activity and greater wild oat recovery.

Wild oat control (measured as % reduction in dry weight compared to control plots) was only fair at the 5-leaf stage both in 1984 (73-88% control) and 1985 (79-90% control). The explanation for this differs for the two seasons. The reason for only moderate control in 1985 is probably the same as the situation described above for the 3-leaf stage. Cool conditions favored recovery over herbicidal phytotoxicity. Weather conditions were suitable for control in 1984 however, at the time of spraying, the wild oat plants were somewhat further advanced in maturity than those sprayed at the '5-leaf stage' in 1985. In 1984, wild oat plants treated on the second spray date were in the 5-leaf to early 6-leaf stage and many had two tillers. In the 1985 treatment, most wild oats had only started to tiller and were in the early 5-leaf stage. Thus, in 1984 the wild oat population was past the optimum stage for diclofop-methyl control. Despite this situation, the two higher rates provided control that was statistically equal to that at the 3-leaf stage.

Selective control of wild oats in Savena 1 oats allowed for significant increases in grain yields in many sprayed treatments as opposed to unsprayed, weedy controls in 1984 and 1985. In 1984 all sprayed plots yielded greater than unsprayed plots (P = 0.05), despite initial injury symptoms. The two treatments that produced significant yield increases in 1985 (0.4 and 0.5 kg/ha, 3-leaf stage) were among the best treatments in 1984 also. One explanation for the presence of fewer

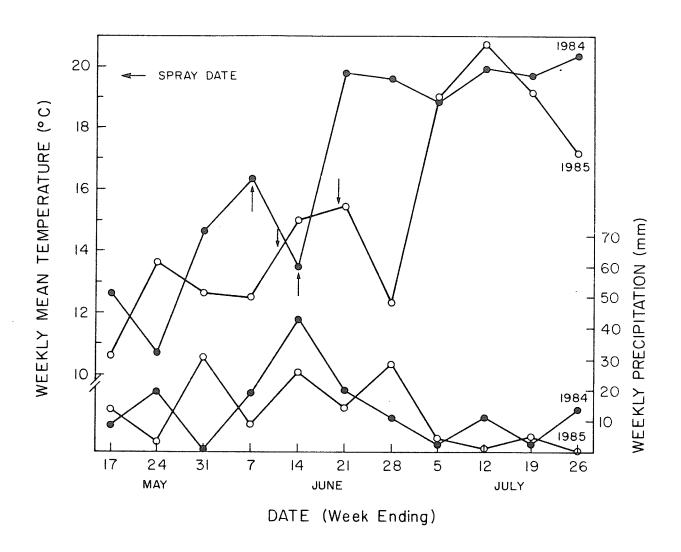


Figure 2. Mean weekly temperatures and total weekly precipitation during the 1984 and 1985 herbicide spraying seasons at Portage la Prairie, Manitoba.

herbicide treatments with yield increases over controls in 1985, was the fact that the wild oat population was smaller than in 1984. The average number of wild oat culms per m^2 in control plots was 84.0 (1985) and 131.8 (1984).

Grain yields in all plots in 1985, including the weed-free trial, were reduced by the presence of stem and crown rust. Both of these diseases can cause extensive damage to oats in Manitoba (Martens et al., 1984). Agrios (1978) reported that stem rust causes losses by reducing foliage and root development as well as yield and quality of grain. This may also have reduced the size of yield differences between diclofop-methyl sprayed and unsprayed treatments. It is possible that in a rust-free season, certain treatments may have yielded significantly less than the control.

Height reductions caused by diclofop-methyl in both 1985 experiments increased as rate increased and were independent of leaf stage of application. This phenomenon is consistent with Hoerauf and Shimabukuro (1979) who reported that wild oat internodes failed to develop after treatment, and Morrison et al., (1981) who showed that treated wild oats had a reduced mitotic index. These wild oat symptoms were expressed to a lesser extent by Savena 1 oats.

The slight delay in time to flowering was also a result of a stress situation. It should be noted that this delay was not accentuated as plants progressed from flowering to maturity.

4.2 <u>The Evaluation of Various Avena sativa Genotypes for</u> <u>Tolerance to the Application of</u> 0.4 and 0.7 kg/ha Diclofop-Methyl

The screening experiment suggested that the level of diclofop-methyl tolerance present in Savena 1 exceeded that of any of the other genotypes tested. It was superior in average visual rating and shoot dry weight at both rates studied. The majority of the other genotypes displayed diclofop-methyl injury symptoms characteristic of wild oats (Section 1.3.1). Symptoms included chlorosis and necrosis of shoot tissue within the first week after herbicide application. These increased in severity during the second and third weeks. The Australian line containing Irwin in its pedigree was the only genotype with tolerance comparable to Savena 1. It was not studied further because its resistance derived from the same source as that of Savena 1. There was also a lack of sufficient seed for further trials. None of the other genotypes tested displayed a level of tolerance sufficient to be used as a sole source of diclofop-methyl tolerance. Possibly, the genotypes listed in Table 9, that displayed a degree of tolerance, may possess genes that complement the action of those in Savena 1. If so, these genotypes could be useful in a breeding program. This is an area of study that could be undertaken in the future.

4.3 <u>Inheritance of Diclofop-Methyl Tolerance</u>

In the 1985 study of the inheritance of diclofop-methyl tolerance in F3, BC1F2, and BC1F3 lines from Savena 1 crosses, susceptible and tolerant lines could be rated with greater confidence than intermediate

or segregating lines. These last two groups were difficult to distinguish. For this reason, they were combined as the 'segregating/intermediate' class.

By examining Table 11 it can be seen that neither the susceptible nor the tolerant control plots were always assessed visual ratings that suggested complete susceptibility or complete tolerance, respectively. A total of 41/101 of the susceptible parental plots (the four parents were combined) were given ratings (13, 14, 15) that suggested segregation for tolerance when in fact these plots were the susceptible controls of this experiment. This 'misclassification' was generally due to the presence of a small number of partially green plants in an otherwise dead plot. This may have been a result of incomplete spray coverage of closely bunched plants, or possibly late germination of one or more seeds in a plot. For this reason, some apparently 'non-uniform' plots were classified as 'uniformly susceptible'.

The 'incorrect' ratings of Savena 1 control plots was usually due to a degree of height differences among plants in a plot (height reduction is a symptom of diclofop-methyl injury, Section 1.3.1). To account for these two digit ratings, a small amount of non-uniformity was accepted as tolerance in the hybrid lines, ie. some lines with apparent segregation (for example, 73 or 64 ratings) in one replicate (at the 0.7 kg/ha rate) were classified as being tolerant. Plate 2a compares parental responses to diclofop-methyl.

Although the two gene model appears to be the best explanation of the inheritance of diclofop-methyl tolerance, certain crosses did not fit this model. The Savena 1 x $0T216^2$ BC1F2 data contains more

susceptible lines and fewer segregating/intermediate lines than expected in a two gene model. This is difficult to explain because the OT216 data fit a two gene model in the F2 and BC1F3 generations. The excess susceptible plots may have been due to incorrect ratings caused by excess herbicide application. This may have been caused by wind interference or a change in walking speed at the time of spraying.

In the OT231 x (Savena 1 x OT231) BC1F3 cross, an insufficient number of susceptible lines were present to fit the two gene hypothesis. Again, the OT231 cross seemed to fit this model in the F2 and BC1F2 generations. The presence of fewer than expected susceptible lines may have been due to insufficient herbicide application, for the reasons mentioned earlier. In addition, this lack of fit may be partially explained as being a product of the rating system used. Had '16' ratings been considered as part of the susceptible category (Section 3.3.1) the results for this cross would have changed from 8 susceptible, 40 segregating/intermediate, 2 tolerant (P: .001 - .01) to 14 susceptible, 34 segregating/intermediate, 2 tolerant (P: .10 - .20). When assessing ratings, the difference between '15' and '16' was negligible. It should be noted that shifts of this magnitude would not have occurred for any other cross tested.

Perhaps the rating system used was too complex. However, when the experiment was initiated, little was known of the inheritance of this trait, therefore it was difficult to determine what the most appropriate rating scheme should be. A standard method did not exist to assess the inheritance of herbicide tolerance. Each herbicide that is studied in this regard, that possesses a unique mode of action, would probably

require a unique rating system. Considering the difficulties of conducting an experiment such as this in the field, it may have been best to simply assign one of four ratings, to each plot, either susceptible, tolerant, intermediate, or segregating.

Finally, crosses involving OT233 did not fit the two gene model in either the F2, F3, or BC1F3 generations. A possible explanation is that one or more loci in OT233 modify the expression of tolerance caused by the other two genes.

Various models involving epistasis and gene interaction as described by Strickberger (1976) were considered. The model which most closely fit the data was one in which two gene pairs controlled tolerance, with susceptibility dominant to tolerance. The presence of either gene pair in the homozygous recessive state produced an intermediate level of tolerance. Because the F2 test was not large, and due to the inherent difficulties in this experiment, caution should be taken in accepting this model. Other two gene models that produce similar ratios may also have merit.

A valuable piece of information regarding dominance or recessiveness that was not obtained was a test of F1 plants. Some F1 plants were included in the Glenlea (late) trial. However, due to cold temperatures and frost during the two to three week period after herbicide treatment, it was not possible to obtain an accurate rating of tolerance. Under these conditions herbicidal activity was not sufficient to completely kill the susceptible parents.

4.4 Alternative Oat Genotypes Reported to Show Diclofop-Methyl Tolerance

The greenhouse study investigating alternative sources of diclofop-methyl tolerance showed that both AC762 and Elen were severely injured by the herbicide. The three AC762 entries responded similarly suggesting that the seed source was homogeneous. The tolerance of AC762 was reported by Somody \underline{et} \underline{al} ., (1984) on the basis of a single replicate. Perhaps herbicide coverage was inadequate or conditions were better suited to plant recovery than herbicidal activity. The variety Elen, although tested on more than one occasion, was reported to display inconsistency in its level of tolerance to diclofop-methyl. The plants examined in the present study appeared quite susceptible. The 22% reduction in the dry matter weight of Savena 1 measured 20 days after treatment may be comparable to 18 and 16% reductions measured 47 and 39 days after treatment, respectively, of plots sprayed with 0.7 kg/ha diclofop-methyl in the field (Section 3.1.3). The OT231 x (Savena 1 x OT231) line (entry #35) selected for uniform tolerance in the 1985 inheritance study displayed the greatest tolerance of the seven entries screened. The fact that it exceeded Savena 1 in tolerance could possibly be attributed to superior agronomic traits as compared to Savena 1, or to transgressive segregation for tolerance.

5.0 SUMMARY AND GENERAL DISCUSSION

The development of a crop cultivar with tolerance to a herbicide that is otherwise lethal to the species in question, is a strategy that can allow for the selective control of a problematic weed species in a genetically similar crop. Breeding programs with this goal have been conducted using conventional plant breeding, cell culture, and genetic engineering techniques. To date, field crop varieties with herbicide tolerance have only been produced via the crossing and selection procedures of conventional breeding.

One cultivar produced in this way was Savena 1, a diclofop-methyl tolerant oat. It was studied in this project with regard to its level of tolerance to diclofop-methyl and the inheritance of this trait. As well, oat genotypes more adapted to North America were tested for tolerance to this herbicide.

Herbicide activity studies revealed that Savena 1 was tolerant, but not resistant, to diclofop-methyl. It was found that if a substantial wild oat population was present in the Savena 1 stand, yield advantages could be obtained by chemically removing it with the herbicide. When crop tolerance was studied, in the absence of wild oat competition, the higher rates of diclofop-methyl studied caused dry matter reductions (measured at heading), however by maturity grain yields were not adversely affected. Based on the three experiments conducted, an optimum wild oat control recommendation for Savena 1 would be as follows:

i) Time: diclofop-methyl should be applied from the 2½ - 5 leaf stage

of Savena 1 but before extensive wild oat tillering.

ii) Rate: A rate of 0.5 - 0.6 kg/ha should be optimal for Savena 1 crop tolerance and adequate wild oat control.

Of the two application stages studied, the 3-leaf may be preferable to the 5-leaf. When the two 1985 experiments were analyzed without the control plots, grain yields were significantly greater at the 3-leaf than the 5-leaf stage, over all rates. It should be noted that Savena 1 crop tolerance was not studied in the one to two leaf stages.

A rate of 0.5 - 0.6 kg/ha was considered optimal because in each of the three experiments grain yields at 0.7 kg/ha (over both leaf stages) were significantly less than yields at the 0.4 - 0.6 kg/ha rates. The most suitable rate of diclofop-methyl for wild oat control in Savena 1 oats may depend on the growing conditions. Assuming that wild oats are treated during the 2½ - 5-leaf stage and before extensive tillering, a rate of 0.4 - 0.5 kg/ha should be adequate under conditions suitable for vigorous plant growth; if cool temperatures or moisture stress conditions exist, a rate of 0.5 - 0.6 kg/ha would be more suitable.

The screening experiment suggested that for this project, the most useful source of diclofop-methyl tolerance available was that displayed by Savena 1. Many of the genotypes tested originated from nearby provinces and states and should therefore have been more agronomically adapted to Manitoba than Savena 1, however they did not possess comparable herbicide tolerance. Savena 1 is adapted to Australian climatic conditions. In Manitoba it was found to be relatively low yielding (varieties recommended for Manitoba yield approximately 450-550 g/m², Brown, personal communication, 1986; while Savena 1 yielded

205-328 g/m^2 in 1984-1985, Tables 6-8), and susceptible to stem and crown rust. Its usefulness would be as a source of diclofop-methyl tolerance in crosses with adapted but susceptible genotypes.

In crosses of Savena 1 with OT216, OT228, and OT231 diclofop-methyl tolerance appears to be controlled by two recessive genes. The presence of either gene alone probably results in an intermediate level of tolerance. The OT233 x Savena 1 crosses behaved differently than the other three. These crosses had a greater number of tolerant progeny in the F2 and F3 generations and fewer susceptible progeny in the BC1F3 generation.

Since, diclofop-methyl tolerance appears to be relatively simply inherited, it should be possible to obtain lines uniform in tolerance and equal to Savena 1 after a few generations of adequate selection pressure. From observations in experiments in which hybrid lines were treated with the herbicide, both in 1984 and 1985, a rate of 0.4 kg/ha is not adequate to remove plants with intermediate levels of tolerance. A rate of 0.7 kg/ha, or possibly greater, would be required.

The activity of diclofop-methyl does not appear to be specific to a single location in plant tissues (Section 1.3). This herbicide may act at more than one site in plasma membranes as well as in the nucleus of cells to inhibit mitosis. For an <u>Avena</u> genotype to be tolerant, two or more gene mutations may be required to allow the detoxification or conjugation of diclofop-methyl. For this reason, it is not surprising that the inheritance of tolerance to this herbicide was not simple. Savena 1 may lack a gene required to produce a level of tolerance comparable to wheat or barley.

Currently, Savena 1 oats may not possess a level of diclofop-methyl tolerance sufficient for use as an agronomically viable cultivar in Western Canada. To warrant the expense of the herbicide application, tolerance levels would probably have to be improved. However, it is possible that progeny of Savena 1 crosses, either because of superior agronomic adaptation to Western Canada or transgressive segregation, may display more tolerance than Savena 1. This appeared to be the case with entry #35 tested in the greenhouse study. Further investigation would be required in future generations when sufficient seed was available for yield trials. Improved levels of tolerance may also be obtained in a crossing program between Savena 1 and one of the genotypes listed in Table 9. These genotypes displayed intermediate levels of tolerance which may complement that of Savena 1. If Elen does indeed possess some tolerance, and if it is derived from a different source than Savena 1, its tolerance could possibly be combined with that of Savena 1 in a breeding project. It may also be possible to detect useful tolerance by further screening of large Avena collections.

To clarify or confirm the results obtained in this inheritance study, a smaller investigation could be performed under greenhouse or growth room conditions. A study in which P1, P2, F1, F2, B1 and B2 (backcross 1 and backcross 2) generations are grown out at the same time would allow for the measurement of heritibility (broad and narrow sense), as well as providing an estimate of the number of genes controlling the trait (Strickberger, 1976). Such an experiment would be most reliable if conducted under strictly controlled conditions with precise herbicidal application using a laboratory sprayer. Individual

plants should be harvested for dry matter to allow for the objective calculation of means and variances of the six populations.

The development of an oat cultivar with a high level of tolerance to a grass-killing herbicide could be of benefit to Canadian farmers in several ways. Such a cultivar would allow for efficient wild oat control in oats resulting in increased yields and reduced soil contamination with weed seeds, increased quality (grade) of oats sold for food and feed, and more effective use of oats as a companion crop in cereal-legume stands. In addition, if an effective means of grassy weed control were available, more producers might grow oats as an alternative to wheat or barley in their crop rotation. Oats are less susceptible than wheat or barley to ergot (Claviceps purpurea), common root rot and spot blotch (Cochliobolus sativus), and Fusarium head blight (Fusarium spp.) (Anon., 1986a). The above advantages could warrant the expense of a diclofop-methyl application.

A concern that could be raised in regard to a herbicide tolerance study of this nature is the question of whether a diclofop-methyl tolerant oat could cause weed problems itself, either as a volunteer in a succeeding crop or through outcrossing to <u>Avena fatua</u>. Neither situation should pose serious problems under proper management practices. New Zealand Cape (the source of diclofop-methyl tolerance in Savena 1) is susceptible to other wild oat herbicides tested. These include diallate, triallate (Avadex BW), trifluralin (Treflan), and difenzoquat (Avenge 200C) (Barr, 1983). Therefore, if a cereal crop was grown following Savena 1 (or a Savena 1 hybrid) control measures for volunteer oats would be available. This would also be the case for

broadleaf crops through the use of either triallate, trifluralin, or sethoxydim (Poast). Even if diclofop-methyl tolerant tame oats did outcross to Avena fatua producing diclofop-methyl tolerant wild oat plants, this process would tend to occur slowly since both species are naturally self-pollinated, and the resulting tolerant wild oats, if they displayed sufficient fitness to set seed, could still be controlled in the following crop by the herbicides listed above.

Two points of caution mentioned by researchers in regard to this problem are as follows. In breeding herbicide tolerant crop cultivars, tolerance should be sought to a single herbicide only, and only in situations where an alternative means of control exists (Faulkner, 1982). Barr (1983) suggested producers use a herbicide rotation in conjunction with their crop rotation to reduce the likelihood of the appearance of herbicide resistant weed biotypes.

Once genetic engineering techniques are improved it may be possible to achieve greater herbicide tolerance in oats through the direct insertion of a gene for tolerance. It may be beneficial to seek tolerance to a herbicide that acts at a single site in the plant. Herbicides such as glyphosate (Steinrucken and Amrhein, 1980) and the sulfonylureas (Ray, 1984) inhibit single enzymes that control the production of specific amino acids. Tolerance may require only a single gene alteration. In such a situation tolerance would probably be simply inherited and conducive to use in a plant breeding project.

LIST OF REFERENCES

- Agrios, G. N. 1978. Pages 372-396. In <u>Plant Pathology</u>, second edition. Academic Press, New York.
- Akey, W. C. and I. N. Morrison. 1983. Effect of moisture stress on wild oat (<u>Avena fatua</u>) response to diclofop. Weed Sci. 31: 247-253.
- Anonymous. 1964. Handbook of Agricultural Statistics Part 1 Field Crops 1908-63. Pages 24-35, Dominion Bureau of Statistics, Agriculture Division, Crops Section, Ottawa.
- Anonymous. 1964-1965. Field Crop Reporting Service. Statistics Canada, Crops Section, Catalogue No. 22-002.
- Anonymous. 1976. Canadian Grain Industry, Statistical Handbook. Canada Grains Council, Winnipeg.
- Anonymous. 1980. Guide to Chemical Weed Control in Alberta. Alberta Agriculture.
- Anonymous. 1982a. Chemical Weed Control in Cereal, Oilseed, and Pulse Crops. Saskatchewan Agriculture.
- Anonymous. 1982b. Pages 105-133. Food and Agriculture Organization of the United Nations, Rome.
- Anonymous. 1982c. Pages 126-127. Food and Agriculture Organization of the United Nations, Rome.
- Anonymous. 1984a. Canadian Grain Industry, Statistical Handbook. Canada Grains Council, Winnipeg.
- Anonymous. 1984b. Official Grain Grading Guide. Canadian Grain Commission, Winnipeg.
- Anonymous. Sept/Oct 1985. Agricultural Biotechnology News. Page 4. Algene secures patent for glyphosite tolerant gene.
- Anonymous. 1986a. Field Crop Production Recommendations for Manitoba. Manitoba Agriculture.
- Anonymous. 1986b. Guide to Chemical Weed Control. Manitoba Agriculture.
- Arntzen, C. J., K. Pfister, and K. E. Steinback. 1982. The mechanism of chloroplast triazine resistance: alterations in the site of herbicide action. Pages 185-214. In Herbicide Resistance in Plants. H. M. LeBaron and J. Gressel, eds. Wiley, New York.

- Bandeen, J. D., G. R. Stephenson, and E. R. Cowett. 1982. Discovery and distribution of herbicide-resistant weeds in North America. Pages 9-30. In Herbicide Resistance in Plants. H. M. Le Baron and J. Gressel, eds. Wiley, New York.
- Barr, A. R. 1983. Personal communication.
- Barr, A. R. 1985. Personal communication.
- Barr, A. R. 1985a. Tolerance of oat genotypes to Hoegrass (R) (diclofop-methyl). In <u>Proceedings Second International Oat Conference</u>, Aberystwyth, Wales (In press).
- Beversdorf, W. D., J. Weiss-Lerman, L. R. Erickson, and V. Souza Machado. 1980. Transfer of cytoplasmically inherited triazine resistance from bird's rape to cultivated oilseed rape (Brassica campestris and B. napus). Can. J. Genet. Cytol. 22:167-172.
- Boldt, P. F. and A. R. Putnam. 1980. Selectivity mechanisms for foliar applications of diclofop-methyl. I. Retention, absorption, translocation and volatility. Weed Sci. 28:474-477.
- Boldt, P. F. and A. R. Putnam. 1981. Selectivity mechanisms for foliar applications of diclofop-methyl. II. Metabolism. Weed Sci. 29:237-241.
- Brezeanu, A. G., D. G. Davis, and R. H. Shimabukuro. 1976.

 Ultrastructural effects and translocation of
 methyl-2-(4-(2,4-dichlorophenoxy)-phenoxy) propanoate in wheat
 (Triticum aestivum) and wild oat (Avena fatua). Can. J. Bot.
 54:2038-2048.
- Brown, P.D. 1986. Personal communication.
- Busch, R., R. Behrens, Anwar Ageez, and M. Elakkad. 1984. Inheritance of resistance and agronomic effect of difenzoquat herbicide on spring wheat. Agronomy Abstracts, p. 60.
- Chaleff, R. S. and M. F. Parsons. 1978. Direct selection <u>in vitro</u> for herbicide-resistant mutants of <u>Nicotiana tabacum</u>. Proc. Natl. Acad. Sci. USA, 75:5104-5107.
- Chaleff, R. S. and T. B. Ray. 1984. Herbicide-resistant mutants from tobacco cell cultures. Science 223:1148-1151.
- Chancellor, R. J. and N. C. B. Peters. 1976. Competition between wild oats and crops. Pages 99-112. In <u>Wild Oats in World Agriculture</u>. D. Price Jones, ed. Agricultural Research Council, London.
- Chancellor, R.J. 1976. Seed behavior. Pages 65-88. In <u>Wild Oats in World Agriculture</u>, D. Price Jones, ed. Agricultural Research Council, London.

- Chow, P. N. P. 1982. Wild oat (<u>Avena fatua</u>) herbicide studies: 1. Physiological response of wild oat to five post-emergence herbicides. Weed Sci. 30:1-6.
- Cohen, A. S. and I. N. Morrison. 1981. <u>In vitro</u> sensitivity of wheat and oat mitochondria to the selective herbicide, diclofop-methyl. Pest. Biochem. Physiol. 16:110-119.
- Comai, L., D. Facciotti, W. R. Hiatt, G. Thompson, R. E. Rose, and D. M. Stalker. 1985. Expression in plants of a mutant <u>aroA</u> gene from <u>Salmonella typhimurium</u> confers tolerance to glyphosate. Nature 317:741-744.
- Comstock, V. E. and R. N. Andersen. 1968. An inheritance study of tolerance to atrazine in a cross of flax (<u>Linum usitatissimum</u> L.). Crop Sci. 8:508-509.
- Conard, S. G. and S. R. Radosevich. 1979. Ecological fitness of <u>Senecio vulgaris</u> and <u>Amaranthus retroflexus</u> biotypes susceptible and resistant to atrazine. J. Appl. Ecol. 16:171-177.
- Crowley, J., J. T. O'Donovan, and G. N. Prendeville. 1978.

 Phytotoxicity of soil-applied dichlorfop-methyl and its effect on uptake of ⁴⁵Ca in wild oats, barley, and wheat. Can. J. Plant Sci. 58:395-399.
- Cussans, G. W. and B. J. Wilson. 1976. Cultural control. Pages 127-142. In <u>Wild Oats in World Agriculture</u>. D. Price Jones, ed. Agricultural Research Council, London.
- Devine, T. E., R. R. Seaney, D. L. Linscott, R. D. Hagin, and N. Brace. 1975. Results of breeding for tolerance to 2,4-D in birds foot trefoil. Crop Sci. 15:721-724.
- Dew, D. A. 1972. An index of competition for estimating crop loss due to weeds. Can. J. Plant Sci. 52:921-927.
- Dew, D. A. 1978. Estimating crop losses caused by wild oats. Pages 15-18. In Wild Oat Action Committee Seminar Proceedings, Regina, Sask.
- Donald, W. W. and R. H. Shimabukuro. 1980. Selectivity of diclofop-methyl between wheat and wild oat: growth and herbicide metabolism. Physiol. Plant. 49:459-464.
- Dortenzio, W. A. and R. F. Morris. 1980. The influence of soil moisture on the foliar activity of diclofop. Weed Sci. 28:534-539.
- Duesing, J. H. 1985. Potential and limitations of genetic engineering for triazine resistance in crops. WSSA Abstracts, 25:77.

- Eddowes, M. 1972. Objectives of weed control in arable crops, principles and practice. Proc. 11th British Weed Cont. Conf. 3:887-892.
- Edwards, C. J. Jr., W. L. Barrentine, and T. C. Kilen. 1976. Inheritance of sensitivity to metribuzin in soybeans. Crop Sci. 16:119-120.
- Elliot, J. G. 1976. Objectives and systems of control. Pages 113-118.

 In <u>Wild Oats in World Agriculture</u>. D. Price Jenes, ed.

 Agricultural Research Council, London.
- Erickson, J. M., M. Rahire, and J. D. Rochaiz. 1985. Herbicide resistance and cross resistance: changes at three distinct sites in the herbicide binding protein. Science 228 (4696), 204-207.
- Faulkner, J. S. 1974. Heritability of paraquat tolerance in <u>Lolium</u> perenne L. Euphytica 23:281-288.
- Faulkner, J. S. 1982. Breeding herbicide-tolerant crop cultivars by conventional methods. Pages 235-256. In <u>Herbicide Resistance in Plants</u>. H. M. Le Baron and J. Gressel, eds., Wiley, New York.
- Fedtke, C. 1982. Pages 177-183. <u>Biochemistry and Physiology of Herbicide Action</u>, Springer-Verlag, Heidelberg.
- Fishbein, G. W., editor. 1985. Calgene, Pfizer unit sign pact on tolerant corn seed. Genetic Engineering Letter 5(5), p. 4.
- Fraley, R., S. Rogers, R. Horsch, R. Weigand, T. Mozer, and D. Shah. 1985. The potential for introduction of herbicide resistance into crop plants by genetic engineering. WSSA Abstracts 25:78.
- Fryer, J. D. and R. J. Makepeace, editors. 1978. <u>Weed Control Handbook Volume 2/Recommendations</u>, eighth edition. Blackwell Scientific Publications, Oxford.
- Geadelmann, J. L. and R. N. Andersen. 1977. Inheritance of tolerance to Hoe 23408 in corn. Crop Sci. 17:601-603.
- Goreka, K., R. H. Shimabukuro, and W. C. Walsh. 1981. Aryl hydroxylation: A selective mechanism for the herbicides diclofop-methyl and clofop-isobutyl, in gramineous species. Physiol. Plant. 53:55-63.
- Gressel, J. 1978. Genetic herbicide resistance: projections of appearance in weeds and breeding for it in crops. In Plant Regulation and World Agriculture. Pages 85-109. T. K. Scott, ed. Plenum Press, New York.
- Gressel, J., H. U. Ammon, H. Fogelfors, J. Gasquez, Q. O. N. Kay, and H. Kees. 1982. Discovery and distribution of herbicide-resistant

- weeds outside North America. Pages 31-55. In <u>Herbicide Resistance</u> in Plants. H. M. Le Baron and J. Gressel, eds. Wiley, New York.
- Grogan, C. O., E. F. Eastin, and R. D. Palmer. 1963. Inheritance of susceptibility of a line of maize to simazine and atrazine. Crop Sci. 3:451.
- Hartwig, E. E. 1985. Identification and utilization of variation in herbicide tolerance in soybean breeding. WSSA Abstracts 25:77.
- Hayes, J. D., R. K. Pfeiffer, and M. S. Rana. 1965. The genetic response of barley to DDT and barban and its significance in crop protection. Weed Res. 5:191-206.
- Hoerauf, R. A. and R. H. Shimabukaro. 1979. The response of resistant and susceptible plants to diclofop-methyl. Weed Res. 19:293-299.
- Holroyd, J., R. J. Chancellor, W. G. Richardson, B. J. Wilson, P. J. Lutman, D. R. Tottman, and P. Ayres. 1976. Chemical control. In Wild Oats in World Agriculture. Pages 143-210. D. Price Jones, ed. Agricultural Research Council, London.
- Hoppe, H. H. 1985. Differential effect of diclofop-methyl on fatty acid biosynthesis in leaves of sensitive and tolerant plant species. Pestic. Biochem. Physiol. 23:297-308.
- Hunter, J. H. 1983. Cultural control of wild oats. Pages 43-52. In Wild Oat Symposium: Proceedings. A. E. Smith, ed. Regina, Sask.
- Karim, A. and A. D. Bradshaw. 1968. Genetic variation in simazine resistance in wheat, rape, and mustard. Weed Res. 8:283-291.
- Kerr, F. A. and F. I. Cook. 1983. Wondervee tomato. Can. J. Plant Sci. Rev. Can. Phytotechnie 63:1103-1105.
- Kocher, H. 1983. Mode of action of the wild oat herbicide diclofop-methyl. In <u>Proceedings of the Wild Oat Symposium</u>, A. E. Smith, ed. Regina, Sask.
- LeBaron, H. M. and J. Gressel, editors. 1982. <u>Herbicide Resistance in Plants</u>. Wiley, New York.
- Martens, J. W., W. L. Seaman, and T. G. Atkinson, editors. 1984.

 <u>Diseases of Field Crops in Canada</u>, The Canadian Phytopathological Society.
- Martin, J. H., W. H. Leonard, and D. L. Stamp. 1976. <u>Principles of Field Crop Production</u>, Macmillan Publishing Co., Inc., New York.
- Marx, J. L. 1985. Plant gene transfer becomes a fertile field. Science 230 (4730):1148-1150.

- Meredith, C. P. and P. S. Carlson. 1982. Herbicide resistance in plant cell cultures. In <u>Herbicide Resistance in Plants</u>. Pages 275-291. H. M. LeBaron and J. Gressel, eds. Wiley, New York.
- Morrison, I. N., M. G. Owino, and E. H. Stobbe. 1981. Effects of diclofop on growth, mitotic index, and structure of wheat (<u>Triticum aestivum</u>) and wild oat (<u>Avena fatua</u>) adventitious roots.. Weed Sci. 29:426-432.
- O'Donovan, J. T. and M. P. Sharma. 1983. Wild oats, competition and crop losses. Pages 27-42. In Wild Oat Symposium: Proceedings. A. E. Smith, ed. Regina, Sask.
- O'Donovan, J. T., E. A. de St. Remy, D. A. O'Sullivan, D. A. Dew, and A. K. Sharma. 1985. Influence of the relative time of emergence of wild oat (<u>Avena fatua</u>) on yield loss of barley (<u>Hordeum vulgare</u>) and wheat (<u>Triticum aestivum</u>). Weed Sci. 33:498-503.
- Pavlychenko, T. K. and J. B. Harrington. 1934. Competitiveness efficiency of weeds and cereal crops. Can. J. of Research 10:77-94.
- Pinthus, M. J., Y. Eshel, Y. Shchori. 1972. Field and vegetable crop mutants with increased resistance to herbicides. Science 177:715-716.
- Radin, D. N. and P. S. Carlson. 1978. Herbicide-tolerant tobacco mutants selected 'in situ' recovered via regeneration from cell culture. Genet. Res. Camb. 32:85.
- Rajhathy, T. and H. Thomas. 1974. <u>Cytogenetics of Oats (Avena L.)</u>. The Genetics Society of Canada, Ottawa.
- Ray, T.B. 1984. Site of chlorsulfuron: inhibition of valine and isoleucine biosynthesis in plants. Plant Physiol. 75: 827-831.
- Ryan, G. F. 1970. Resistance of common groundsel to simazine and atrazine. Weed Sci. 18:614-616.
- Schooler, A. B., A. R. Bell, and J. O. Nalewaja. 1972. Inheritance of siduron tolerance in foxtail barley. Weed Sci. 20:167-169.
- Scott, K. R. and P. D. Putwain. 1981. Maternal inheritance of simazine resistance in a population of <u>Senecio vulgaris</u>. Weed Res. 21:137-140.
- Sexsmith, J. J. 1964. Morphological and herbicide susceptibility differences among strains of hoary cress. Weed Sci. 12:19-22.
- Shands, H. L. and W. H. Chapman. 1961. Culture and production of oats in North America. Pages 465-529. In <u>Oats and Oat Improvement</u>. F. A. Coffman, ed. American Society of Agronomy, Madison, Wisconsin.

- Shimabukuro, M. A., R. H. Shimabukuro, W. S. Nord, and R. A. Hoerauf. 1978. Physiological effects of methyl 2-(4-(2,4-dichlorophenoxy) phenoxy) propionate on oat, wild oat and wheat. Pestic. Biochem. Physiol. 8:199-207.
- Shimabukuro, R. H., W. C. Walsh, and R. A. Hoerauf. 1979. Metabolism and selectivity of diclofop-methyl in wild oat and wheat. J. Agric. Food Chem. 27:615-623.
- Steinrucken, H.C. and N. Amrhein. 1980. The herbicide glyphosate is a potent inhibitor of 5-enolpyruvylshikimic acid-3-phosphate synthase. Biochem. Biophys. Res. Commun. 94: 1207-1212.
- Somody, C. N., J. D. Nalewaja, and S. D. Miller. 1984. Wild oat (<u>Avena fatua</u>) and <u>Avena sterilis</u> morphological characteristics and response to herbicides. Weed Sci. 32:353-359.
- Souza Machado V., A. Ali, and J. Shupe. 1983. Herbicide-resistant rutabagas: a novel approach to weed control. Highlights of Agricultural Research in Ontario 6(4):8-10.
- Souza Machado V., S. C. Phatak, and I. L. Nonnecke. 1982. Inheritance of tolerance of the tomato (<u>Lycopersicon esculentum Mill.</u>) to metribuzin herbicide. Euphytica 31:129-138.
- Stafford, R. G., V. E. Comstock, and J. H. Ford. 1968. Inheritance of tolerance in flax (<u>Linum usitatissimum</u> L.) treated with MCPA. Crop Sci. 8:423-426.
- Strickberger, M. W. 1976. Pages 202-220. <u>Genetics</u>, second edition. Macmillan Publishing Co., Inc., New York.
- Strykers, J., M. van Himme, and E. van Bockstaele. 1972. Susceptibility of <u>Avena</u> spp. and varieties of chlorfenprop-methyl. Meded Rijksuniversiteit-Gent 17, 32-35.
- Taylor, H. F. and Codd, T. M. 1985. The chemical control of wild oats in oats a progress report. In <u>Proceedings Second International Oat Conference</u>, Aberystwyth, Wales (In press).
- Thomas, A. G., editor. 1982. The 1981 weed survey of cultivated land in Manitoba. Published with the cooperation of Manitoba Department of Agriculture.
- Thomas, A. G. 1983. Field and questionnaire surveys of cereal and oilseed crops in Western Canada. Pages 17-26. In <u>Wild Oat Symposium: Proceedings</u>. A. E. Smith, ed. Regina, Sask.
- Thomas, A. G. 1985. Weed survey system used in Saskatchewan for cereal and oilseed crops. Weed Sci. 33:34-43.
- Thomas, H. and I. T. Jones. 1976. Origins and identification of weed species of <u>Avena</u>. Pages 1-18. In <u>Wild Oats in World Agriculture</u>. D. Price Jones, ed. Agricultural Research Council, London.

- Todd, B. G. and E. H. Stobbe. 1977. Selectivity of diclofop-methyl among wheat, barley, wild oat (<u>Avena fatua</u>) and green foxtail (<u>Setaria viridis</u>). Weed Sci., 25:382-385.
- Tseng, S. T., H. L. Carnahan, C. W. Johnson, J. J. Oster, J. E. Hill, and S. C. Scardaci. 1984. Registration of cultivar L-202 rice (Oryza sativa). Crop Sci. 24:1213-1214.
- Western, D. E. and W. R. Graham, Jr. 1961. Marketing, processing, uses and composition of oats and oat products. Pages 552-578. In Oats and Oat Improvement. F. A. Coffman, ed. American Society of Agronomy, Madison, Wisconsin.
- Wright, C. E. and J. S. Faulkner. 1981. Effective selection for tolerance to grass-killing herbicides in perennial ryegrass (<u>Lolium perenne</u> L.). Pages 210-212. In Proc. XIV International Grasslands Congress. J. A. Smith and V. W. Hayes, eds. Westview Press, Boulder, Col.

APPENDIX TABLE 1. Average visual ratings of Savena 1 crop tolerance in 1984 and 1985 activity studies.

Diclofop- methyl rate (kg/ha)	Leaf stage of application ^b				¹ Visual F after tre	_	
		1	004				985
			984		85	(wee	d-free)
		1 Week	3 weeks	1 week	3 weeks	1 week	3 weeks
0.0	control	0 0	0.0				
		9.0	9.0	9.0	9.0	9.0	9.0
0.4	3	6.0	7.0	6.5	7.8	7.0	7.8
0.5	3	6.0	6.0	6.3	7.0	6.0	8.0
0.6	3	6.0	6.0	5.5	7.0	5.3	7.0
0.7	3	5.0	5.8	4.5	6.0	5.0	6.3
0.4	5	5.8	8.0	6.3	7.3	7.0	8.0
0.5	5	5.8	7.8	6.3	7.3	6.0	
0.6	5	5.8	7.3	6.0	7.0		7.0
0.7	5	5.3	7.0			6.0	7.3
			7.0	5.3	6.5	6.0	7.0

 $^{^{\}mathbf{a}}$ Average of four replicates for all diclofop-methyl treatments (eight for controls).

bwild oat leaf stage for 1984 and 1985 experiments with wild oat competition, Savena 1 leaf stage for 1985 (weed-free) experiment.

APPENDIX TABLE 2. Average visual ratings (22 days after treatment) of three replicates of 240 oat genotypes tested for diclofop-methyl tolerance at two rates.

1984 Western Cooperative Oat Test

	Average Rating		
Variety or Station Number	at 0.4 kg/ha	at 0.7 kg/ha	
Rodney	2.0	1.0	
Fidler	2.0	1.0	
Dumont	1.7	1.3	
Calibre	1.3	1.0	
Cascade	1.0	1.0	
W80093	1.7	1.3	
W80474	1.7	1.0	
OT220W	1.7	1.0	
W81129	1.7	1.0	
W81146	1.7	1.3	
W82056	2.3	1.0	
W82639	1.7	1.7	
S081136	2.7	2.0	
\$082004	1.7	1.0	
S082013	3.7	2.0	
S082030	1.3	1.3	
393-29	3.3	2.0	
388-121	2.3	1.7	
421-69	1.0	1.0	
421-72	1.3	1.0	

1984 Eastern Cooperative Oat Test

Variety or Station Number	Average Rating at 0.4 kg/ha at 0.7 kg/ha		
	at 0.4 kg/ha	at 0.7 kg/ha	
Elgin	2.0	1.3	
Lamar	1.7	1.0	
Oxford	2.7	2.3	
Shaw	1.7	1.3	
Terra	1.3	1.0	
Woodstock	3.7	2.3	
CG084-2	2.0	1.0	
0A540-19	1.3	1.3	
0A551-1	1.3	1.0	
0A555-1	2.0	1.3	
0A569-1	1.7	2.0	

APPENDIX TABLE 2 (continued). Average visual ratings (22 days after treatment) of three replicates of 240 oat genotypes tested for diclofop-methyl tolerance at two rates.

OA583-1	1.7	1 2
0A629-6	1.7	1.3
0A447-43	2.0	1.7 2.0
Q0206.60	1.0	1.0
Q0209.32	1.7	2.0
AS81-1	2.0	1.3
AS81-2	1.3	1.0
0A447-27	1.7	
0A516-2	1.7	1.3
0A518-11	1.3	1.3
Q0191.70	1.3	1.7
Q0199.27	2.3	1.3
Q0199.60		1.7
Q0505.2	1.0	1.0
Q0447-11	2.0	1.0
0A501-1	2.3	2.3
Ogle	2.0	1.0
Q0186.10	1.0	1.0
Q0508.3	3.0	2.7
Q 0000,3	2.0	1.0

1984 Oat Rust Area Test

	Average Rating		
Variety or Station Number	at 0.4 kg/ha	at 0.7 kg/ha	
Dumont	1.7	1.0	
Fidler	1.7	1.0	
OT231	1.7	1.7	
W82269	1.3	1.0	
W32393	1.7	1.0	
W82404	2.0	1.3	
W82586	1.3	1.0	
W82678	1.0	1.3	
Steele	2.0	1.7	
S082060	1.7	1.3	
S083082	1.3	1.0	
S083084	2.0	1.0	
S083100	1.3	1.0	
W83020	2.0	1.3	
W83056	1.3	1.0	
W83069	1.7	1.0	
W83073	1.3	1.0	
W83080	1.0	1.0	
W83100	2.0	1.0	

APPENDIX TABLE 2 (continued). Average visual ratings (22 days after treatment) of three replicates of 240 oat genotypes tested for diclofop-methyl tolerance at two rates.

W83101	1.3	1.3
W83113	1.0	1.7
W83176	1.7	1.0
W83230	2.0	1.3
W83279	2.0	1.0
W83326	2.0	1.3
W83344	2.0	1.0
W83387	1.3	1.0
W83390	1.7	1.0
W83399	1.3	1.3
W83402	1.0	1.0
W83205	1.7	1.0
W83438	2.0	1.3
W83442	2.0	
W83452	2.0	1.0
W83460	_ · ·	1.0
W83512	1.7	1.0
M03017	1.3	1.0

1984 Uniform Midseason Oat Performance Nursery

Vanioty on State Coloration w		e Rating	
Variety or State Selection Number	at 0.4 kg/ha	at 0.7 kg/ha	
W1 X390-15	2.7	2.3	
Dal	2.7	2.3	
IL 75-5860	1.7	1.7	
IL 75-3402	2.0	2.0	
IL 79-5394	1.7	1.0	
IL 80-3072	2.0	1.7	
IL 79-1776	1.7	1.0	
IL 79-4924	1.0	1.0	
Ogle	1.3	1.0	
IA B605-1085	2.0		
Dumont	1.7	1.7	
W80474	1.7	1.3	
PA 8098-13900	2.3	1.3	
PA 8196-1338	1.7	1.7	
PA 8196-15	1.0	1.3	
SD 790400	2.0	1.3	
SD 810095		1.0	
SD 790188	1.3	1.0	
SD 800312	2.0	1.7	
Clintland 64	2.0	2.0	
CIINCIANU 04	2.7	2.7	

APPENDIX TABLE 2 (continued). Average visual ratings (22 days after treatment) of three replicates of 240 oat genotypes tested for diclofop-methyl tolerance at two rates.

2.0	2.0
2.0 2.0	· 1.3 1.7
1.7	1.0
3.0	2.3
3.0	1.3
4.0	1.7
1.3	1.3 2.3
2.0 1.0	2.0 1.7 1.0
	2.0 2.0 1.7 1.3 3.0 2.0 3.0 2.0 4.0 1.7 1.3 1.7 2.0

1984 Preliminary Oat Test

lines from OT224 X (Moore X OT220) and (Moore X OT220) X OT224 crosses

1004	Avera	ge Rating
1984 Accession Number	at 0.4 kg/ha	at 0.7 kg/ha
W84295	1.7	1.0
W84296	2.0	1.0
₩84297	1.7	1.0
W84298	1.7	1.0
W84299	1.7	1.0
W84300	1.7	1.7
W84301	1.7	1.3
W84302	2.7	2.0
W84303	2.0	1.3
W84304	1.7	1.3
W84305	2.0	1.0
₩84306	1.0	1.0
₩84307	1.0	1.0
W84308	1.3	1.0
W84309	2.0	1.3
W84310	2.0	1.3
W84311	2.0	1.0
W84312	1.3	1.3
₩84313	1.7	1.0
W84314	2.3	1.3

APPENDIX TABLE 2 (continued). Average visual ratings (22 days after treatment) of three replicates of 240 oat genotypes tested for diclofop-methyl tolerance at two rates.

W84316	2.0	1.3	
W84317	2.7	1.7 ·	
W84318	1.3	1.0	
W84319	1.3	1.3	
W84 320	1.7	1.0	
W84321	1.7	1.0	
W84322	1.0	1.0	
W84323	1.7	1.0	
W84325	2.0	1.0	
W84326	2.3	1.0	
W84329	1.7	1.3	
W84330	1.7	1.3	
W84331	1.7	1.0	
W84333	2.0	1.0	
W84334	1.3	1.0	
W84335	2.3		
₩84336	2.3	1.3	
W84339		1.3	
W84340	1.3	1.0	
W84341	1.7	1.3	
W84342	2.0	1.0	
	1.7	1.0	
W84343	2.0	1.0	
W84344	2.0	1.3	

Agriculture Canada Historical Oat Collection

		e Rating
Variety Name	at 0.4 kg/ha	at 0.7 kg/ha
Beaver	2.7	1.7
LaSalle	2.0	1.3
Valor	2.3	2.3
Dasix	2.3	1.3
Roxton	1.7	2.0
Exeter	2.0	2.0
Beacon	4.3	3.7
Clinton	2.3	1.7
Bambu	2.3	2.0
Big Four	2.0	1.7
Fortune	1.7	1.3
Abegweit	4.0	2.3
Lanark	2.3	1.7
Gopher	2.3	1.3
Victory	4.0	2.3

APPENDIX TABLE 2 (continued). Average visual ratings (22 days after treatment) of three replicates of 240 oat genotypes tested for diclofop-methyl tolerance at two rates.

Alaska	2.7	0.0	
Laurel	1.7	2.3	
0.A.C. 72	2.0	1.0 2.0	
0.A.C. 144	2.0	1.3	
Fundy	2.0	1.3	
Banner	2.0	1.7	
Legacy	2.0	1.7	
0.A.C. #3	3.3	2.3	
Larain	3.7	2.3	
Clintland	2.3	1.7	
Shefford	2.0	1.7	
Simcoe	2.3	1.7	
Scotian	4.0	2.7	
Glen	2.0	1.3	
Shield	2.7	2.3	
Russell	2.0	1.7	
Sixty Day	1.3	1.0	
Hajira Strain	2.3	2.3	
Cartier	4.7	2.3	
White Cross	1.7	1.0	
Liberty	1.3	1.0	
Gold Rain	2.7	2.0	
Ligowa	2.3	1.3	
Danish Island	2.7	2.3	
Thousand Dollar	3.0	1.7	
Danish Island	1.3	1.3	
Anthony	3.7	3.3	
Ajax	2.0	1.3	
Eagle	2.0	2.0	
Early Miller	2.0	1.3	
Erban	2.7	2.0	
Mabel	2.0	1.3	
Brighton	2.3	2.0	
Garry	2.0	1.7	
Vicar	2.0	1.3	
Rodney	2.0	1.0	
Tartar King	2.0	2.0	
Great Mogul	2.0	1.7	
Waverly	2.3	2.0	
Granary Filler	3.0	2.0	
C.D. 2492	2.0	2.0	
Cabot	2.7	2.0	
Cavell	2.7	2.0	
Dorval	2.0	1.3	
Foothill	2.0	2.0	
Fraser	2.0	1.7	
Gemini	1.7	1.3	

APPENDIX TABLE 2 (continued). Average visual ratings (22 days after treatment) of three replicates of 240 oat genotypes tested for diclofop-methyl tolerance at two rates.

Grizzly Harmon Hinoat	1.7	1.0	
Harmon	2.0		
		1.7	
Hinoat		4 + 1	
	4.0	3.3	
Hudson	1.3	2.0	
Kelsey	2.0	1.7	
Laurent	1.7	1.3	
Oxford	2.7	2.7	
Pendek	2.0	2.3	
Random	2.0	1.0	
Russell	2.0	2.0	
Scott	2.3	1.7	
Sentinal	2.0	1.3	
Sioux	3.0	2.0	
Stormont	3.0		
Terra			
Yamaska	1.7		
Fidler			
	Laurent Oxford Pendek Random Russell Scott Sentinal Sioux Stormont Terra Yamaska	Laurent 1.7 Oxford 2.7 Pendek 2.0 Random 2.0 Russell 2.0 Scott 2.3 Sentinal 2.0 Sioux 3.0 Stormont 3.0 Terra 1.0 Yamaska 1.7 Fidler 2.3 Cascade 1.3 Athabasca 2.0	Laurent 1.7 1.3 Oxford 2.7 2.7 Pendek 2.0 2.3 Random 2.0 1.0 Russell 2.0 2.0 Scott 2.3 1.7 Sentinal 2.0 1.3 Sioux 3.0 2.0 Stormont 3.0 2.7 Terra 1.0 1.0 Yamaska 1.7 1.3 Fidler 2.3 1.7 Cascade 1.3 1.3 Athabasca 2.0 1.0