RESPONSE OF TRIFLURALIN-RESISTANT GREEN FOXTAIL [*Setaria viridis* (L.) Beauv.] TO HERBICIDES

A Thesis

Submitted to the Faculty

of

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by

Hugh John Beckie

In Partial Fulfillment of the

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of

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Department of Plant Science

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ABSTRACT

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Response of trifluralin-resistant green foxtail [*Setaria viridis* (L.) Beauv.] to herbicides. Major Professor: Ian N. Morrison.

The response of trifluralin-resistant (R) green foxtail to herbicides was investigated at the whole-plant level under both controlled environmental and field conditions and at the cell level, providing a detailed description of the expression of resistance in R foxtail.

Differences in response between R and susceptible (S) foxtail to increasing dosages of trifluralin, applied as a preplant incorporated (PPI) treatment in rapeseed and as a preemergence incorporated (PEI) treatment in wheat, were 7- and 12-fold, respectively, based on density and biomass determinations 4 wk after emergence. Under cropped conditions, 9 and 14 times higher dosages of PPI- and PEI-trifluralin, respectively, were required to reduce R seed production by 50% than to reduce S seed production by the same amount. At the recommended trifluralin dosage in rapeseed (1.4 kg ha^{-1}), the density of S plants was reduced by 84% compared to untreated plots, whereas the density of R plants was reduced by only 4%. The effective kill (seed yield reduction) was 99% and 42%, respectively. At the recommended dosage in wheat (0.9 kg ha^{-1}), the density of S and R plants was reduced by 99% and 36%, respectively. The effective kill was 97% and 14%, respectively. These studies indicate that the expression of resistance in R foxtail is affected by the method of trifluralin incorporation and does not decline over the growing season under cropped conditions. Furthermore, since the selection pressure of trifluralin on green foxtail can be estimated from effective kill, the evolution and population dynamics of R foxtail under field conditions can be simulated more accurately using population models.

The response of R foxtail to herbicides belonging to several chemical groups indicated resistance to dinitroanilines and an unrelated mitotic disrupter. The response of R and S foxtail to increasing dosages of ethalfluralin, applied as a PPI treatment in rapeseed, indicated that the R biotype was 7 times more resistant to ethalfluralin than the S biotype. Under cropped conditions, 7 times more herbicide was required to reduce R seed production by 50% than to reduce S seed production by the same amount. At the recommended dosage in rapeseed (1.4 kg ha^{-1}), the density of R plants was reduced by 35%, whereas the density of S plants was reduced by 95%. The effective kill was 55% and 99%, respectively. The results indicate that ethalfluralin will not effectively control R foxtail. However, a number of other herbicides with different mechanisms of action are available to effectively control R foxtail.

The effect of trifluralin and ethalfluralin on R and S green foxtail was further examined using a petri dish bioassay and by determining mitotic indices of treated and untreated root tips. In the petri dish assay, radicle growth of R green foxtail exposed to trifluralin concentrations of up to 0.4 ppm (w/v) was not inhibited. Radicle growth of S foxtail was completely inhibited at this concentration. Radicle growth of both S and R biotypes were much more sensitive to ethalfluralin than to trifluralin. R foxtail was 9 times more resistant to trifluralin and 6 times more resistant to ethalfluralin than S foxtail. To screen seed stocks for resistance, best discrimination between R and S foxtail was achieved by measuring radicle length after incubation of germinated caryopses at 0.3 ppm trifluralin in the dark for 5 days at 22 C. To determine mitotic indices, squashes of S and R root meristems treated with increasing concentrations of trifluralin and ethalfluralin were examined by light microscopy. The R biotype was 10 times more resistant to both trifluralin and ethalfluralin than the S biotype as indicated by the mitotic indices. As in the bioassay, both biotypes

were much more sensitive to ethalfluralin than to trifluralin. The concentrations causing an increase in the number of cells in condensed prophase corresponded well with the concentrations required to inhibit radicle growth. These results indicate that differences in sensitivity may be related to a target-site modification.

Nomenclature: Ethalfluralin, *N* -ethyl-*N* -(2-methyl-2-propenyl)-2,6-dinitro-4-(trifluoromethyl)benzenamine; trifluralin, 2, 6-dinitro-*N*,*N* -dipropyl-4-(trifluoromethyl)benzenamine; green foxtail, *Setaria viridis* (L.) Beauv.; rapeseed, *Brassica napus* L. 'Westar'; spring wheat, *Triticum aestivum* L. 'Katepwa'.

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This thesis has been written in manuscript style. All manuscripts were prepared in accordance with the style requirements of Weed Technology. However, a subnumbering system was used for main section headings and subsection headings.

1. INTRODUCTION

Herbicide resistance in weeds is a relatively recent phenomenon. The reported cases of herbicide-resistant (R) weeds have increased sharply over the past decade. There are presently over 120 R biotypes that have evolved in various locations around the world, including most of the Canadian provinces, American states, and European countries. Resistance in weeds not only endangers the usefulness of both old and new herbicide classes, but threatens the viability of our agricultural systems. It is a symptom of monoculture/ monoherbicide practices that were frequently employed in the past. If the trend of increasing incidence of R biotypes is not altered, herbicide resistance could rapidly become a serious economic problem for agriculture in the future.

In Manitoba, trifluralin-resistant green foxtail was confirmed in growth chamber experiments during the winter of 1988-89 (Morrison et al. 1989). Most R populations originated in fields in southwestern Manitoba where trifluralin had been used repeatedly for nearly 20 yr. This was only the second reported case of a weed species being resistant to a dinitroaniline herbicide in North America; trifluralin-resistant goosegrass [*Eleusine indica* (L.) Gaertn.] was reported in South Carolina in the early 1980's (Mudge et al. 1984). In Manitoba, agricultural producers were concerned that R green foxtail populations could pose a serious threat to crop production, since trifluralin is used extensively on the prairies and green foxtail is the most abundant weed of cereal and oilseed crops.

The discovery of R foxtail prompted the need for research in many areas, including 1) the comparative response of R and susceptible (S) green foxtail to dinitroaniline herbicides and to herbicides belonging to other chemical groups under both controlled environmental and field conditions, 2) the relative fitness

of R compared to S populations, 3) the breeding system of green foxtail and the mode of inheritance of resistance, and 4) a determination of the mechanism of resistance. This thesis addresses the first of the above areas and reports on the results of dose-response experiments to determine if the expression of resistance in R green foxtail differs 1) under controlled environmental as compared to field conditions, 2) under cropped as compared to non-cropped conditions over the growing season, 3) with depth of herbicide incorporation in the soil, 4) among herbicides belonging to various chemical groups, and 5) at the cell level as compared to the whole-plant level. The response of a R weed species to herbicides under such a range of test conditions has rarely been documented in the literature.

When placed in the broader context of international research collaboration into herbicide resistance, these studies attempt to fill a large void in our understanding of the rate of enrichment of selected R weed biotypes. Predictive models have been developed that highlight the contributions of various factors that affect the population dynamics of R weeds. They have indicated that the selection pressure of the herbicide on the weed species is the most important factor influencing the rate of enrichment of R biotypes. Yet, studies that have quantified the true selection pressure (measured by relative seed production of R and S biotypes under cropped conditions at the end of the growing season) are mostly non-existent. Instead, estimates of selection pressure, inferred from initial reductions in weed density or biomass shortly after herbicide treatment, have been inserted into the models. However, such estimates could be very different from the true selection pressure, thereby contributing to inaccurate model results. More accurate simulations could aid immensely in designing preventative or management strategies, such as detailed crop/herbicide rotations, to regulate the proportion of R biotypes in the field.

An important component of the research program consisted of field experiments, which were conducted at the University of Manitoba Research Station at Portage la Prairie in 1989 and 1990. The objectives of two experiments were to verify the 1988-89 growth chamber results and to establish the extent to which R and S biotypes differed in their response to trifluralin, when applied as a pre-plant incorporated (PPI) treatment and a pre-emergence incorporated (PEI) treatment in rapeseed and wheat, respectively. In these experiments, particular importance was placed on determining the effective kill (seed yield reduction) of R and S green foxtail at recommended dosages, since this parameter is equated with the selection pressure of the herbicide.

As an extension of the work conducted at Portage la Prairie, in 1989 an experiment was conducted in a field in southwestern Manitoba, which was naturally infested with R green foxtail. This provided a comparison of the response between sown and naturally occurring R foxtail to PPI trifluralin under cropped and non-cropped conditions. The different climate and soil at this site extended the range of test conditions and therefore provided more comprehensive information on the response of R foxtail to trifluralin.

Little is known of the cross-resistance pattern of R green foxtail to other dinitroaniline herbicides and to herbicides belonging to other chemical families. In addition to providing an insight into the mechanism of resistance, identification of cross-resistance patterns provides possible alternatives for controlling R green foxtail where the problem has already developed. For these reasons, studies were undertaken to document the response of R green foxtail to other dinitroaniline herbicides and two unrelated mitotic disrupter herbicides. The extent of resistance to eight other herbicides belonging to six chemical families with different mechanisms of action was also investigated. Most of these herbicides are registered to control or suppress green foxtail in various

cereal and oilseed crops grown in Manitoba. In conjunction with these growth chamber studies, a field experiment compared the response of R and S biotypes to ethalfluralin, which is registered in western Canada to control various grass and broadleaf weeds when applied as a PPI treatment in rapeseed and other oilseed and specialty crops.

Another important component of the research program was the development of a simple and inexpensive bioassay to screen green foxtail seed samples to determine resistance to trifluralin or ethalfluralin. The detection of R green foxtail by growing plants in pots filled with herbicide-treated soil requires several weeks before definitive conclusions can be made. The successful development of a petri dish bioassay facilitates the identification of R foxtail so that control measures can be implemented quickly.

Resistance in foxtail was also examined at the cell level to ascertain differences between R and S plants at the location of herbicide action. The response of R and S green foxtail to trifluralin and ethalfluralin was determined by measuring the mitotic indices of treated and untreated S and R root meristems. This study will contribute to a more complete understanding of the expression of resistance in R green foxtail.

2. LITERATURE REVIEW

2.1. Introduction

This chapter reviews the literature on herbicide resistance in weeds. It is divided into three main sections. The first provides a general overview of the extent and nature of resistance in weeds. The second describes the evolution and dynamics of R[1] weed populations, including plant and herbicide attributes that affect the development of resistance. The extent to which green foxtail and dinitroaniline herbicides share these attributes will be examined. The final section outlines agronomic practices to delay or preclude the development of resistance in weeds.

2.2. The Extent and Nature of Herbicide Resistance in Weeds

Resistance is defined as a genetic change within a pest population in response to selection by a toxicant that may impair control in the field (Sawicki 1987). Thus, R weeds are those that survive and grow normally at field dosages that usually control the weed effectively (LeBaron and Gressel 1982). In contrast, herbicide tolerance is defined as a low degree of resistance that is dosage dependent (Holt and LeBaron 1990). It is usually the result either of differences in herbicide uptake and translocation at the plant level, or of differences in plant metabolism and herbicide detoxification or sensitivity of the site of action (Warwick 1991).

Herbicide resistance in weeds has a more recent history than either insecticide or fungicide resistance. In the past, resistance to herbicides evolved

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[1]Abbreviations: ACCase, acetyl coenzyme A carboxylase; AHAS, aceto-hydroxyacid synthase; ALS, acetolactate synthase; MT, microtubule; R, resistant; S, susceptible.

more slowly than pesticide resistance in insects and fungi. Presently, however, the rate of either evolution or detection of new cases of herbicide resistance in plants is equivalent to those in insects and fungi (Holt and LeBaron 1990). Since the first reported case of herbicide resistance (Ryan 1970), there are presently over 120 R weed biotypes. These include at least 57 species (40 dicots and 17 monocots) resistant to triazine herbicides and at least 45 biotypes (27 dicots and 18 monocots) resistant to 15 other classes of herbicides (LeBaron 1989; Holt and LeBaron 1990). Resistance to nontriazine herbicides has been a more recent occurrence, but is now being reported at a faster rate. R weeds may become a more serious economic problem within 5 to 10 yr than pest resistances to insecticides and fungicides due to the greater use of herbicides in agriculture (LeBaron and McFarland 1990).

In Manitoba, R biotypes of three weed species have been discovered recently in field crops. The occurrence of trifluralin-resistant green foxtail, primarily in southwestern Manitoba, was confirmed in dose-response experiments conducted in the growth chamber during the winter of 1988-89 (Morrison et al. 1989). In fields where R green foxtail was confirmed, farmers observed random patchiness of green foxtail escapes, poor green foxtail control yet good trifluralin activity on other weeds for which it is registered, worsening control of green foxtail each year with repeated trifluralin use, and/or escapes that did not follow any pattern relating to application or incorporation. The highest frequency of occurrence of R foxtail corresponded closely to areas where trifluralin was used most frequently in the past. R populations had been selected by trifluralin after nearly 20 yr of repeated use in both cereal and oilseed crops. Prior to the appearance of R green foxtail, the weed had been shown to be very sensitive to the herbicide (Rahman and Ashford 1970).

R biotypes of two other weed species were also recently discovered in Manitoba fields. In 1990, it was confirmed that several populations of wild oat (*Avena fatua* L.) were resistant to various herbicides belonging to the arloxyphenoxypropionates and cyclohexanediones (Heap and Morrison 1991). The mechanism of action of herbicides belonging to these chemical groups is the inhibition of the chloroplast enzyme, ACCase[1], which catalyzes fatty acid synthesis[2]. Wild mustard (*Sinapis arvensis* L.) that resists auxin-type herbicides including 2,4-D [(2,4-dichlorophenoxy)acetic acid] was also detected (Heap 1991, personal communication).

The appearance of R weeds should have been expected. Indeed, herbicide resistance was predicted by Blackman in 1950, shortly after the introduction of synthetic organic herbicides. Changes in the weed flora can occur in response to any agricultural manipulation (Haas and Streibig 1982; Radosevich and Holt 1984). R weed biotypes are a consequence of basic evolutionary processes. Biotypes within a species that are best adapted to a particular practice are selected for and will increase in the population (Gressel and Segel 1990a; Holt and LeBaron 1990). Once the weed population is exposed to a herbicide to which some naturally occurring R biotypes are present, the herbicide kills S plants and favors R ones. With repeated use of the herbicide over time on the same site, R biotypes come to dominate the population and the soil seed bank (Holt and LeBaron 1990). R biotypes are also generally cross-resistant to other chemically related compounds that have a similar mechanism of action (Holt and LeBaron 1990). Most reported cases of resistance are presumed to be due to separate instances of parallel evolution

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[2]ACCase resistance in a population of green foxtail has also been confirmed (Heap 1991, personal communication).

through selection (Gressel 1987).

The mechanism of resistance in weeds is often due to an altered site of action (Fuerst and Vaughn 1990). The highly resistant (100- to 10,000-fold) biotype of goosegrass has an altered tubulin subunit such that MTs[1] are insensitive to dinitroaniline herbicides (Vaughn 1986a,b; Vaughan and Vaughn 1987). Hyperstability of MTs in R cells, caused by the novel tubulin subunit, is responsible for dinitroaniline resistance (Vaughan and Vaughn 1987; Vaughn and Vaughan 1990). In constrast, there are no discernable differences between the tubulin subunits of R and S[1] green foxtail biotypes[3]. The mechanism of resistance in R green foxtail may be due to an alteration in a MT-associated protein (Smeda et al. 1991), similar to that postulated for another dinitroaniline-resistant biotype of goosegrass with an intermediate (50-fold) level of resistance (Vaughn et al. 1990).

Triazine resistance in most weeds is due to a loss of herbicide binding ability because of an alteration of the binding site (a 32 kilodalton protein) on the thylakoid membrane of the chloroplast (Arntzen et al. 1982; Rádosevich 1983). However, another triazine resistance mechanism involving herbicide detoxification through enhanced metabolism has been reported (Gronwald et al. 1989; LeBaron and McFarland 1990).

Sulfonylurea herbicides inhibit the ALS[1] enzyme, also referred to as AHAS[1] (Chaleff and Mauvais 1984; Ray 1984; Shaner et al. 1984), which catalyzes the first step in the biosynthesis of the branched chain amino acids. The mechanism of resistance is an altered site of action (ALS/AHAS enzyme), which is inhibited less in R than in S biotypes by chemical groups having this

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[3]Ellis, J. R. 1991. Personal communication. Jealott's Hill Research Station, Bracknell, Berkshire, U.K.

mechanism of action (i.e. the sulfonylureas, imidazolinones, and the sulfonanilides) (Saari et al. 1990).

There are some reported cases where the mechanism of resistance does not involve an alteration at the site of action. Mechanism(s) of bipyridylium resistance include rapid sequestration of the herbicide resulting in reduced herbicide concentration at the site of action in the chloroplast, and/or rapid enzymatic detoxification of superoxide and other toxic forms of oxygen due to elevated levels of superoxide dismutase (Fuerst and Vaughn 1990). The most likely mechanism of multiple herbicide resistance (the evolution of populations resistant to chemically unrelated herbicides with different mechanisms of action) is due to enhanced metabolic detoxification by microsomal cytochrome P_{450} mono-oxygenases (Kemp and Casely 1987; Kemp et al. 1990; Powles and Howat 1990). Examples include rigid ryegrass (*Lolium rigidum* Gaudin) in southern Australia (Heap and Knight 1986; Powles et al. 1990) and blackgrass (*Alopecurus myosuroides* Huds.) in England (Moss 1987; Kemp et al. 1990), which are resistant to wheat-selective herbicides.

2.3. Evolution and Dynamics of Herbicide-Resistant Weed Populations

The rate of appearance or enrichment of R plants in a population under monoculture and/or monoherbicide usage has been estimated by a simple population model (Gressel and Segel 1978, 1982):

$$N_n = N_o \left[1 + \frac{fa}{\beta} \right]^n$$

N_n is the proportion of R individuals in the population after n seasons of herbicide use, N_o is the initial frequency of R plants prior to herbicide use, f is

the relative ecological fitness of R compared to S biotypes, ß is the average seed bank longevity, and *å* is the selection pressure of the herbicide on the weed species. The model assumes that R individuals initially form an exceedingly small fraction of the population; these R plants increase by a constant factor each year (exponential increase). This exponential increase has been verified in the field (Nosticzius et al. 1979). Resistance in the field becomes detectable when the proportion of R individuals reaches about 10 to 30% of the population. N_O is the very low initial frequency of R individuals derived from natural mutations in the S population prior to any herbicide exposure. This initial frequency is the product of the frequency of natural mutations to the R biotype and the ecological fitness of the R biotype relative to the S biotype. N_O depends on several genetic factors including the number of genes involved (mono- versus polygenic traits, ploidy level) and the mode of inheritance of resistance (Duesing 1983). For example, N_O is estimated at 10^{-5} to 10^{-6} if resistance is a monogene dominant trait (Gressel and Segel 1982), inferred from the frequency of other naturally occurring nuclear-inherited mutations (Gressel and Segel 1978). This initial frequency provides the starting point for resistance, whereas the parameters within the brackets of the equation affect the rate of enrichment of resistance.

Ecological fitness (*f*) is defined as the ability of the R biotype to compete with the S biotype under nonselective (i.e. without herbicide) field crop conditions. Fitness differences between R and S biotypes are usually inferred from measures of relative plant productivity and/or competitiveness (Warwick 1991). Overall, *f* is the compounded fitness for each stage of growth and is measured as the relative seed production (per unit area) of the R biotype compared to the S biotype when the plants are grown in mixed culture (Gressel 1979). Relative fitness of R and S biotypes has been measured at various

stages in the life cycle of weed species. However, the ultimate measure is reproductive fitness, because it is the integrated product of fitnesses at all levels including germination, establishment, growth and reproduction (Gressel 1985).

Haldane (1960) postulated that selection has a genetic 'cost' which is 'charged against fitness'. This is exemplified by the triazine-resistant (TR) biotypes, which are generally much less fit than their corresponding wild (TS) type (Radosevich and Holt 1982; Holt 1990). Reduced fitness in R biotypes infers that R plants will be replaced by S individuals over time after the herbicide is absent. However, target site mutations or gene amplifications in enzymes present in low quantities may not exert such strong effects on fitness as was found with triazine resistance (Gressel and Segel 1990a). There have even been reports of comparable photosynthetic potential and growth between TR and TS populations, under both competitive and noncompetitive conditions (Rubin et al. 1985; Jansen et al. 1986; Schonfeld et al. 1987). Differences in fitness between sulfonylurea R and S biotypes of kochia [*Kochia scoparia* (L.) Schrad.] are less than that observed for TR weeds (Warwick 1991). Dinitroaniline-resistant and susceptible biotypes of goosegrass do not differ in most growth and development characteristics when grown under noncompetitive field conditions, with the exception of greater inflorescence weight in the S biotype (Murphy et al. 1986; Valverde et al. 1988). However, further studies indicated that the R biotype was indeed less competitive than S plants and responded to competition by reduced reproductive output (Valverde et al. 1988). There is little difference in fitness between R and S biotypes of blackgrass (Moss and Cussans 1989). If fitness of the R biotype is not less than the S biotype, resistance should decline slowly, or not at all (Gressel and Segel 1982; Rubin 1991).

If R plants are indeed less fit than S plants, however, there can be a strong dampening effect on the rate of evolution of resistance, but only when it can be expressed, i.e. when the herbicide is not present as the selecting agent. Thus in monoherbicide culture, this lack of fitness will be less important in retarding the rate of development of resistance when persistent herbicides are employed, than when shorter residual herbicides are used (Gressel and Segel 1982, 1990a). If R individuals have near-normal fitness, rotation of herbicides with different sites of action will be of little added value in delaying resistance. The only delay will be the number of seasons that the particular herbicide (or maybe chemically related herbicides) is not used (Gressel and Segel 1990a). In such instances, only lowering the selection pressure will delay resistance.

In addition to ecological fitness, S gene flow, specifically immigration of external pollen and seed genes into a population through space and time, has been identified as being a potentially important biological process in the evolution and dynamics of R weed populations (Maxwell et al. 1990; Roush et al. 1990). These authors indicate that S gene flow may significantly reduce the proportion of R individuals in a population of predominantly cross-pollinating species if there are large, nearby sources of the S biotype. However, gene flow probably will not effectively reduce the proportion of R individuals in predominantly self-fertilizing populations, unless the S sources are quite large and near the treated population. Gene flow would also be much more effective in slowing the rate of appearance of R individuals if resistance was a recessive rather than a dominant trait (Roush et al. 1990). However, gene flow due to seeds and pollen is usually minimal, in the range of metres per year (Sagar and Mortimer 1977; Hume and Archibold 1986), especially in cropped versus non-cropped situations (Warwick 1991).

The germination dynamics of the weeds, over the growing season and from the soil seed bank, is an important factor in the evolution of resistance. The average seed bank longevity of the weed species (ß) can be a major modifier of the rate of enrichment of R biotypes (Gressel and Segel 1982). The longer the seed is viable in the soil, the greater is the buffering effect of S seed from previous years, decreasing the rate of evolution of resistance. The model assumes that (different) constant proportions of susceptibles and resistants germinate, survive to the end of the season and that S and R plants have (different) constant seed yields. There are conflicting reports of differences in dormancy between seeds of R and S biotypes, depending on the species and growth conditions (Warwick 1991).

The selection pressure (å) of the herbicide on the weed species is governed by the dosage and frequency of use, its efficacy with particular weeds, and its persistence (Gressel and Segel 1982). A high selection pressure will result if a long residual herbicide (eg. triazines, dinitroanilines, sulfonylureas), which provides season-long control of weeds, is used or herbicides with shorter residual activity are used repeatedly (Gressel and Segel 1990a). A herbicide with a relatively long residence time in soil would be conducive for the selection of R biotypes because of the long selection pressure duration. Furthermore, since most S seedlings are killed, intraspecific competition and S seed production is minimized.

The selection pressure is the most important factor affecting the rate of evolution of resistance in weed biotypes (Gressel and Segel 1982). The selection pressure is not determined by the initial reductions in density or biomass typically measured by weed scientists, but rather by determination of the 'effective kill'. Effective kill is the reduction in weed seed yield of R and S biotypes due to the herbicide treatment measured at the end of the growing

season (Gressel and Segel 1978). The difference between the initial density or biomass reductions and the effective kill is governed by the interaction between herbicide persistence and late germination of weed seed after the herbicide is gone, as well as the capacity of surviving plants to compensate after herbicide thinning (Gressel and Segel 1982). S weed seeds can germinate after a rapidly degraded herbicide has disappeared. S plants then produce more seeds before the season is over, considerably lowering the selection pressure.

Subsequent to the development of the predictive model described above, the following model was proposed that considers what happens during the 'off' years when a particular herbicide is not used (Gressel and Segal 1990a,b):

$$H_{p,q} = [1 + \partial\ (\mathring{a}f_{on} - 1)]^{p}\ [1 - \partial\ (1 - f_{off})]^{q}$$

$H_{p,q}$ is the overall enrichment factor giving the increase in resistance following a period of p 'on' seasons of herbicide application and q 'off' seasons without herbicide; ∂, the fraction of seeds leaving the seed bank each year, replaces β, the average residence time as the factor describing seed bank characters. It is assumed that there are no differences in the longevity of S and R seeds in the soil seed bank. The remaining model parameters are the same as in the original model. This modified model more adequately describes the effects of herbicide rotations on delaying or precluding resistance, and produces various scenarios derived from different input values for the model parameters.

2.3.1. Herbicide and weed attributes affecting the evolution of resistance

The rate of appearance of resistance is governed by an interaction between specific attributes of both the herbicide and the weed species. Some of these were integrated, directly or indirectly, into the population models described in the previous section. Those attributes and others are examined in this section. R biotypes have evolved only in monoculture and/or

monoherbicide conditions at predictable rates for each compound and weed species (Gressel and Segel 1990a).

2.3.1.1. Herbicide attributes

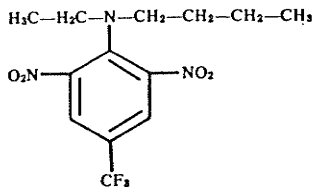
Characteristics of herbicides and their use that contribute to a high probability for the evolution of resistance are: 1) single target site and specific mechanism of action, 2) extremely active and effective in killing a wide range of weed species, 3) long soil residual and season-long control of germinating weeds, and 4) applied frequently and over several growing seasons without rotating, alternating, or combining with other types of herbicides (LeBaron and McFarland 1990). These characteristics would cause intense selection pressure for the evolution of resistance. The herbicide and use characteristics are correlated fairly well with the herbicides that have selected for resistance (Holt and LeBaron 1990).

Dinitroaniline herbicides have been classified as having a high risk for the selection of R biotypes (LeBaron and McFarland 1990). Dinitroaniline herbicides are non-systemic, soil-applied compounds that are principally grass killers but also control many broadleaf weeds (Appleby and Valverde 1989). Chemical structures and properties of some of these herbicides are shown in Figure 2-1.

Herbicides with a highly site-specific mechanism of action are more likely to be inactivated by a single gene mutation (which induces a change at the site of action) than herbicides that cause a more general disruption of plant growth and development, such as 2,4-D and other growth regulator-type herbicides with multiple sites of activity (Putwain 1982). Dinitroaniline herbicides act at a single target site and have a very specific mechanism of action. The specific site of action is tubulin, a protein in the cell that polymerizes to form MTs (Appleby and Valverde 1989). MTs have a number of functions in higher plant

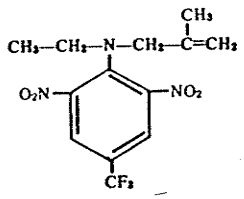
Benefin

MW=335
VP=7.8 X 10^{-5} mm Hg (25 C)
Water solubility (25 C)=0.10 ppmw



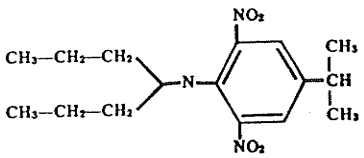
Ethalfluralin

MW=333
VP=8.2 X 10^{-5} mm Hg (25 C)
Water solubility (25 C)=0.30 ppmw



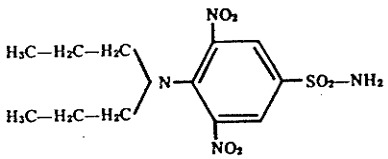
Isopropalin

MW=309
VP=3.0 X 10^{-5} mm Hg (25 C)
Water solubility (25C)=0.08 ppmw



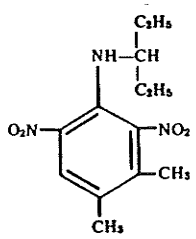
Oryzalin

MW=346
VP<1.0 X 10^{-8} mm Hg (25 C)
Water solubility (25 C)=2.6 ppmw



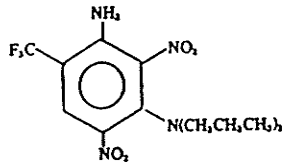
Pendimethalin

MW=281
VP=3.0 X 10^{-5} mm Hg (25 C)
Water solubility (25 C)=0.28 ppmw



Prodiamine

MW=350
VP=2.5 X 10^{-8} mm Hg (25 C)
Water solubility (25 C)=0.01 ppmw



Trifluralin

MW=335
VP=1.1 X 10^{-4} mm Hg (25 C)
Water solubility (25 C)=0.30 ppmw

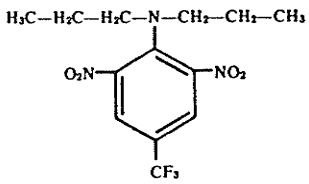


Figure 2-1. Chemical structures and properties of select dinitroaniline compounds (WSSA 1989).

cells, the most important of these being movement of chromosomes during cell division (spindle MTs), formation of the cell plate during cytokinesis (phragmoplast MTs), and determination of cell shape (cortical MTs) (Gunning and Hardham 1979; Clayton 1985). In S plants, dinitroaniline herbicides bind to tubulin which prevents their polymerization into MTs in the cytoplasm(Strachan and Hess 1983; Morejohn et al. 1987; Vaughn and Lehnen, Jr. 1991), thereby arresting mitosis.

Dinitroaniline herbicides are very effective in controlling a wide spectrum of weed species, a feature which has favored their extensive use. Susceptible weeds are controlled shortly after germination but prior to emergence from soil (Parka and Soper 1977). The major sites of uptake are the shoots of monocots and the hypocotyl or hypocotyl hook of dicots (Barrentine and Warren 1971; Hilton and Christiansen 1972; Friesen and Bowren 1973; Parka and Soper 1977; Derr and Monaco 1982). Dinitroaniline herbicides are also absorbed by the roots, but shoot exposure is more phytotoxic (Knake et al. 1967; Appleby and Valverde 1989). Most dinitroaniline herbicides are volatile and can be absorbed in the vapor phase by roots and shoots of germinating seedlings. Absorption of trifluralin as vapor is more phytotoxic than absorption from the soil solution (Swann and Behrens 1972; Harvey 1974). Translocation or metabolism of dinitroaniline herbicides within plants is typically minor (Golab et al. 1967; Strang and Rogers 1971; Parka and Soper 1977; Jacques and Harvey 1979a; Marquis et al. 1979; Ashton and Crafts 1981).

The effectiveness of dinitroaniline herbicides in controlling susceptible weed species is dependent on the activity (as measured by phytotoxicity) of these herbicides in soil. Their activity in soil, in turn, depends on the degree of adsorption of the herbicide molecules to soil colloids, which is affected by moisture and temperature. At relatively low soil moisture levels, increased

adsorption of trifluralin to soil colloids will decrease its phytotoxicity (Bode et al. 1973; Friesen and Bowren 1973; Harper et al. 1976; Moyer 1979). Soil temperature has been observed to have either no effect on trifluralin phytotoxicity (Mulder and Nalewaja 1978; Moyer 1979; Darwent 1980; Heath et al. 1985) or was positively correlated with phytotoxicity due to more herbicide present in the vapor phase (Rahman 1973; Jacques and Harvey 1979b).

Adsorption and activity of trifluralin and ethalfluralin in soil are also dependent on the organic matter content (Grover et al. 1979; Moyer 1979; Derr and Monaco 1982). As soil organic matter content is increased, adsorption of the herbicide to soil colloids is increased and consequently, the phytotoxicity is reduced (Friesen and Bowren 1973; Rahman 1973; Grover 1974; Horowitz et al. 1974; Weber et al. 1974; Pritchard and Stobbe 1980; Derr and Monaco 1982; Peter and Weber 1985).

Trifluralin and ethalfluralin are highly effective in controlling green foxtail. They are registered in western Canada to control green foxtail in oilseed crops such as rapeseed, when applied as a pre-plant incorporated (PPI) treatment. Trifluralin, when applied at 1.1 kg ha^{-1}, controlled over 95% of green foxtail in rapeseed (Chow 1976). The PPI treatment consists of incorporating the herbicide into the soil to a depth of 8 to 10 cm prior to seeding. Trifluralin can also be applied as a pre-emergence incorporated (PEI) treatment to control green foxtail in wheat and barley. Trifluralin, applied at 0.56 kg ha^{-1}, controlled over 95% of green foxtail in these two cereal crops (Rahman and Ashford 1970, 1972). The herbicide is applied to the soil and shallowly incorporated to a depth of 2 to 4 cm following seeding. The herbicide is present in the soil as a thin concentrated band above the depth of placement of the wheat or barley caryopses. The selective action of trifluralin for the control of green foxtail in wheat is due to differences in the sensitivity between the two species to

trifluralin and to morphological differences between seedlings of the weed and the crop (Rahman and Ashford 1970). The sensitive coleoptilar node of wheat remains closely associated with the caryopsis below the treated layer. The sensitive coleoptilar node of green foxtail is located at a soil depth of less than 1 cm regardless of depth of seed burial. If the green foxtail caryopsis is located beneath the treated layer, extension of the first internode (mesocotyl) will bring the coleoptile in contact with the herbicide. If the roots come into direct contact with trifluralin, which would occur when germination proceeds within the treated layer, a shortening of the roots and radial expansion near the tips would result. However, failure of emergence of trifluralin-damaged green foxtail seedlings is mainly the result of shoot effects (Knake et al. 1967). Negligible elongation and radial enlargement of the first internode and a marked swelling and shortening of the coleoptile is characteristic of shoot damage (Swann and Behrens 1972; Ashford et al. 1987).

Persistence of dinitroaniline herbicides in soil is another important component of the selection pressure of these herbicides. The persistence of trifluralin is enhanced with higher soil organic matter levels (Smith 1972; Pritchard and Stobbe 1980). Soil texture or pH, though, have generally no effect on persistence or activity of trifluralin (Probst et al. 1967; Menges and Hubbard 1970; Grover 1974; Harvey 1974; Horowitz et al. 1974; Weber et al. 1974; Grover et al. 1979; Moyer 1979; Gaynor 1985). Herbicide dissipation rates tend to be greater in relatively warm, moist soils (Probst et al. 1967; Horowitz et al. 1974; Zimdahl and Gwynn 1977; Golab et al. 1979; Hayden and Smith 1980). Chemical processes are mainly responsible for degradation of dinitroaniline herbicides in soil; microbial degradation is minor (Probst et al. 1967; Parr and Smith 1973). Losses to the atmosphere by volatilization, especially from moist soil, is the most important means of dissipation of

dinitroaniline herbicides from treated soils (Helling 1976). On the Canadian prairies, between 10 to 30% of the trifluralin detected at seeding time carries over in the soil to the next season (Smith 1982). The carryover of ethalfluralin residues to the next crop year is very similar to trifluralin (Hayden and Smith 1980).

2.3.1.2. Weed attributes

Weed species may possess certain attributes that favor the development of R biotypes. Species that tend to exhibit resistance to herbicides are those that possess characteristics such as the following: predominantly herbaceous annuals, widely distributed over agricultural habitat, rapid development of plants from seedling to maturity, wholly or partially self-fertile, high reproductive capacity, short seed viability in the soil, and complex genetic variability expressed as polymorphic phenotypes (Hill 1982; Warwick 1991). These attributes would be conducive to a rapid R population increase in response to selection.

Rigid ryegrass, an annual diploid grass weed of cereal and grain legume crops in Australia, has many of these qualities with the exception that it is an outcrossing species. It is widespread throughout the cropping zones of southern Australia (Powles and Howat 1990). When uncontrolled, its high fecundity and competitiveness result in rapid population increases (Heap and Knight 1986). Its relatively short seed bank longevity (Rubin 1991) further predisposes this weed to evolve R biotypes, which now pose a serious threat to sustainable agriculture in that region.

Green foxtail also possess most or all of the attributes that favor the evolution of R biotypes. A comprehensive review on the biology of this weed was published by Douglas et al. (1985). Green foxtail is an annual diploid (2n=18) grass weed. Since its introduction into Canada from Europe circa

1821, it has become the most abundant weed species of cereal and oilseed crops on the Canadian prairies (Douglas et al. 1985). It occurs on approximately 78%, 53%, and 26% of the cultivated area in Manitoba, Saskatchewan, and Alberta, respectively (Thomas and Wise 1985, 1987, 1988). Although easily controlled with herbicides, green foxtail continues to pose a serious problem in annual crops because of its rapid growth through the vegetative stage to flowering, the large number of seeds it produces, and its high phenotypic plasticity or polymorphism (Douglas et al. 1985). Green foxtail caryopses have a relatively short seed bank longevity of approximately 2 yr (Banting et al. 1973; Thomas et al. 1986). The species also has a very low degree of outcrossing (0.25 to 0.32%) (Jaseniuk 1991, personal communication), which makes possible the rapid buildup of a resistant population from a single seed or plant. This set of characteristics would favor the evolution of R green foxtail populations, especially in a high selection pressure environment. Triazine resistance at the chloroplast level was discovered in a few populations of green foxtail in France in 1981 (Gasquez and Compoint 1981). ACCase resistance in populations of green foxtail was recently discovered in Manitoba (Heap 1991, personal communication).

The competitiveness of green foxtail will affect the rate of appearance of R biotypes, since competition is a determinant of ecological fitness (Maxwell et al. 1990). The competitive effects of green foxtail depend on the associated crop, the weed density, the time of emergence relative to the crop, and environmental conditions following emergence (Dryden and Whitehead 1963; Blackshaw et al. 1981b). Crop yield reductions as high as 25% may occur as a result of green foxtail interference (Sturko 1978; Maurice and Morrison 1983). Barley is the most efficient competitor; spring rye, oats, rapeseed, and wheat rank as less

efficient competitors, followed by flax, which is the least competitive crop (Maurice and Morrison 1983; Douglas et al. 1985; Hoechst 1989).

In field surveys, green foxtail densities ranged from less than one to more than 1500 plants m^{-2} (Thomas and Wise 1987). In a survey conducted in Manitoba in the late 1970's, the average green foxtail density in infested fields prior to spraying was approximately 300 plants m^{-2}, with a maximum density of over 2600 plants m^{-2} (Thomas and Donaghy 1991).

The effect of green foxtail density on crop yields is highly variable. In cereal crops, densities of 1600 plants m^{-2} did not reduce yield in some years, while in other years less than 100 plants m^{-2} reduced yield significantly (Rahman and Ashford 1972; Blackshaw et al. 1981b).

Seed germination and seedling emergence of green foxtail is greatly affected by soil temperature and moisture (Vanden Born 1971; Banting et al. 1973; Blackshaw et al. 1981a). The low soil temperatures that typically occur during spring seeding reduce the potential competitiveness of green foxtail with wheat because of poor germination (Vanden Born 1971; Alex et al. 1972; Rahman and Ashford 1972; Banting et al. 1973). Freshly harvested seed is dormant. However, this dormancy disappears in less than 10 wk under field conditions (Banting et al 1973).

The main flush of green foxtail emerges in June, with subsequent germination and emergence associated with precipitation events (Banting et al. 1973). In field studies in Manitoba, Blackshaw et al. (1981a) also observed that emergence was related to average daily soil temperature. Most seedlings emerge from depths of 1 to 3 cm; delayed or prolonged emergence from greater depths probably accounts for the numerous flushes appearing throughout the season (Dawson and Bruns 1962; Alex et al. 1972; Maurice and Morrison 1983). Plants that emerge early in the growing season are generally the most

competitive. In wheat, green foxtail is most competitive when it emerges with the crop or shortly thereafter, regardless of planting date (Banting et al. 1973; Blackshaw et al. 1981b). Although green foxtail plants that emerge after the crop have less detrimental effects on crop yield, they can produce seed which will provide continued infestations in subsequent years (Vanden Born 1971; Rahman and Ashford 1972).

The critical period of green foxtail competition occurs during early stages of seedling growth (Sturko 1978). At the seedling stage, relatively high temperatures and light intensity are necessary for establishment and rapid growth of green foxtail (Vanden Born 1971). This C_4 plant is very sensitive to shading and low temperatures (Blackshaw et al. 1981b; Lee and Cavers 1981). Bubar and Morrison (1984) reported that plants growing in full sunlight produced up to 5 times more tillers, and about 8 times more dry matter than plants growing in wheat. Early seeded crops and agronomic practices that result in rapid establishment of a crop canopy, therefore, are beneficial in reducing the vigor and competitiveness of green foxtail (Rahman and Ashford 1972; Bubar and Morrison 1984).

2.4. Agronomic Practices to Delay or Preclude Resistance

The underlying principle of any management strategy is to reduce the selection pressure for the evolution of resistance (Holt and LeBaron 1990). This reduction can be accomplished by an integrated weed management approach, including the judicious use of herbicides with the minimum selection pressure giving cost-effective weed control (Gressel 1986). This is in sharp contrast with a recommendation of a total weed kill, advocated as late as 1976. The rationale was that a partial kill or stunting of the weeds would create a high selection pressure for resistance, but a total kill would give zero selection pressure

(Holliday et al. 1976). Less frequent use of long residual herbicides would delay the appearance of resistance by allowing S plants to reach maturity and produce enough seed each year to dilute out R seeds (Gressel and Segel 1990a). The adoption of minumum tillage practices, although beneficial for soil and water conservation, may require more frequent use of herbicides, thus increasing the selection pressure. Furthermore, the seed bank longevity under such a production system would be only 1 yr if weed seed is not buried (Gressel and Segel 1982). This would further increase the rate of enrichment of R biotypes.

Herbicide rotations were recommended over forty yr ago to prevent the occurrence of R weeds (Blackman 1950). Herbicides which act at different sites of action should be used in the rotation (Radosevich and Appleby 1973; Holliday et al. 1976; LeBaron and Gressel 1982; Parochetti et al. 1982) since a R biotype resistant to one chemical is generally cross-resistant to others with similar chemistry (mechanism of action). However, there are weed biotypes, such as rigid ryegrass, with multiple resistance to more than one class of herbicides. For these R biotypes, rotation of herbicides having different modes of degradation as well as different mechanisms of action may be useful (Holt and LeBaron 1990). Since many of these R populations are cross-resistant to different herbicides to varying degrees than other R populations (Heap and Knight 1986), the herbicide rotation program must be carefully planned, based on knowledge of the cross resistance pattern of each R population.

In the future, the use of chemical synergists may help combat R biotypes where resistance is due to herbicide degradation. Synergistic effects may be attributed to modification of either the uptake, translocation, or metabolism of the herbicide (Kemp and Casely 1989). For example, some synergistic compounds, such as 1-aminobenzotriazole (ABT), inhibit cytochrome P_{450}

mixed-function oxidases, which are responsible for herbicide detoxification in R biotypes of blackgrass and rigid ryegrass (Kemp et al. 1990). Synergists may also allow lower herbicide dosages to be applied, thereby lessening the selection pressure. For example, tridiphane [2-(3,5-dichlorophenyl-2-(2,2,2-trichloroethyl))oxirane] prevents grasses from metabolizing atrazine [6-chloro-*N* -ethyl-*N* -(1-methylethyl)-1,3,5-triazine-2,4-diamine], allowing control of both grassy and broadleaf weeds in corn (*Zea mays* L.) at lower dosages (Ezra et al. 1985; Lamoureux and Rusness 1986)

Tank mixtures of herbicides, which differ in their mechanisms of action but are active on the same spectrum of weeds, may also be useful (LeBaron and Gressel 1982). However, the effectiveness of this strategy may be limited by the availability of suitable mixture partners, differences in time of application or persistence of the herbicides, and their cost-effectiveness (Rubin 1991; Thill et al. 1991). There are also concerns that some mixtures may actually increase the selection pressure because of herbicide synergy (Radosevich et al. 1989). Inclusion of herbicides, to which the R biotype exhibits strong negative cross resistance (R biotype is more susceptible than the S biotype), in rotations or mixtures can be very effective in delaying the evolution of resistance (Gressel and Segel 1990a). For example, dinitroaniline-resistant goosegrass is more sensitive to chlorpropham [1-methylethyl 3-chlorophenylcarbamate] than the S biotype (Vaughn et al. 1987).

Cultural control practices are an integral component of cropping systems for reducing the propensity for the development of resistance. Crop rotations are beneficial in delaying resistance since this usually involves herbicide rotations and such rotations may alter the life cycle of the weed by changing conditions for plant growth (LeBaron and Gressel 1982). Inclusion of crops with different phenologies, such as winter wheat, or crops with greater

competitive efficiencies, would be especially important in retarding the rate of evolution of resistance (Parochetti et al. 1982). Suppression of growth and reproduction of R biotypes by crop competition is a key element in the management of resistance. Timely tillage operations can also be an effective weed control option.

The extent to which a particular set of agronomic practices can influence the rate of development and spread of R biotypes was recently documented in Ontario (Stephenson et al. 1990). TR weeds are only a minor problem in corn fields in southwestern Ontario, which has the longest history of corn production and triazine use of any area in the province. Predominant agronomic practices include crop rotation, atrazine use on 60% of the corn land, use of other postemergence herbicides, interrow cultivation, little silage corn, and little manure returned to the land. However, in eastern Ontario where corn (particularly grain corn) is relatively new as a major crop, more than 75% of corn land is infested with two or more TR weed species. Predominant agronomic practices include continuous corn, treatment of nearly all fields with atrazine, infrequent use of postemergence herbicides and cultivation, use of 25% of the corn for silage, and deposition of manure from corn silage on all cultivated land. As a consequence, TR biotypes are continuing to spread at the fastest rate in this region.

Management practices that promote susceptibility have been advocated for slowing the evolution of resistance. Manipulation of S genotype gene flow by having S source areas adjacent to the treated population (noted in section 2.3) or leaving untreated areas within the treated population may slow the development of resistance (Radosevich et al. 1989; Maxwell et al. 1990; Roush et al. 1990). However, there is a lack of scientific evidence to substantiate this hypothesis.

If a R weed population does develop, it should be detected as soon as possible. Indications that a weed population is resistant were outlined in section 2.2. It is important to remember, however, that poor weed control is often related to application problems, suboptimum weed stage for spraying, or environmental conditions. If the suspect plants are confirmed to be resistant, many of the practices outlined above can still be employed. However, the range of options available to the producer may be limited, such as the elimination or less frequent use of the herbicide(s) that selected the R biotype. Chemically related herbicides may also be ineffective in controlling the R population. The alternative herbicide(s) are usually less cost-effective. Practices that minimize the spread of R seeds via harvesting equipment or contaminated seed grain should also be followed. It is much easier to employ good agronomic practices to delay or prevent the appearance of R weed biotypes than to control them after they infest an area.

3. RESPONSE OF RESISTANT GREEN FOXTAIL (*Setaria viridis*) TO TRIFLURALIN

Abstract. The response of susceptible (S) and resistant (R) green foxtail biotypes to increasing dosages of trifluralin, applied as a preplant incorporated (PPI) treatment in rapeseed and as a preemergence incorporated (PEI) treatment in wheat, was investigated in field experiments located at Portage la Prairie in 1989 and 1990. Additionally, the response of these biotypes was compared under non-cropped conditions in the same experiments. A third, similar experiment including PPI treatments was located near Deloraine, Manitoba in 1989. Green foxtail density and shoot biomass 4 wk after emergence verified the occurrence of R foxtail. The difference in response between the biotypes to PPI trifluralin was approximately 7-fold under either cropped or non-cropped conditions, whereas the difference in sensitivity between the biotypes to PEI trifluralin was about 12-fold under these same conditions. Under cropped conditions, 9 and 14 times higher dosages of PPI- and PEI-trifluralin, respectively, were required to reduce R seed production by 50% than to reduce S seed production by the same amount. At the recommended trifluralin dosage in rapeseed (1.4 kg ha^{-1}) , the density of S plants 4 wk after emergence was reduced by 84% compared to untreated plots, whereas the density of R plants was reduced by only 4%. The effective kill (seed yield reduction) was 99% and 42%, respectively. At Deloraine, the initial reductions in density and biomass, as well as the effective kill of R green foxtail were similar to that observed at Portage la Prairie. When trifluralin was applied at the recommended dosage in wheat (0.9 kg ha^{-1}) , the density of S plants 4 wk after emergence was reduced by over 99% compared to less than 36% for R plants. The effective kill was 97% and 14%, respectively. These studies

indicate that the expression of resistance in R foxtail is affected by the method of trifluralin incorporation and does not decline over the growing season under cropped conditions. Furthermore, since the selection pressure of trifluralin on green foxtail can be estimated from effective kill, the evolution and population dynamics of R foxtail under field conditions can be simulated more accurately using population models. **Nomenclature:** Trifluralin, 2,6-dinitro-*N*,*N* -dipropyl-4-(trifluoromethyl)benzenamine; green foxtail,*Setaria viridis* (L.) Beauv. #[1] SETVI; rapeseed, *Brassica napus* L. 'Westar'; spring wheat, *Triticum aestivum* L. 'Katepwa'.

Additional index words: Herbicide resistance, *Brassica napus, Triticum aestivum,* SETVI.

3.1. Introduction

Green foxtail is the most abundant grass weed of cereal and oilseed crops on the Canadian prairies (Thomas and Wise 1985, 1987, 1988). Trifluralin, introduced in western Canada in the early 1970's, proved to be very effective in controlling green foxtail in crops including rapeseed when applied as a PPI treatment (Chow 1976), and in wheat as a PEI treatment (Rahman and Ashford 1970, 1972). Spring-applied trifluralin is recommended at 0.8 to 1.4 kg ha^{-1} and at 0.6 to 0.9 kg ha^{-1} to control green foxtail in rapeseed and wheat, respectively[2]. The lower dosages are specified on lighter textured soils with relatively low organic matter contents. The differences in dosages between the two crops relate to the differences in methods of incorporation.

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[1] Letters following this symbol are a WSSA-approved computer code from Composite List of Weeds, Revised 1989. Available from WSSA, 309 W. Clark St., Champaign, IL 61820.

[2] 1991 Guide to Chemical Weed Control, Manitoba Agriculture, 908 Norquay Bldg., Winnipeg, MB R3C OP8.

Dose-response experiments conducted in the growth chamber during the winter of 1988-89 verified the occurrence of trifluralin-resistant green foxtail from fields located primarily in southwestern Manitoba (Morrison et al. 1989). This was the first reported case of a weed species being resistant to a dinitroaniline herbicide in Canada. The R biotypes were about 5 times more resistant to trifluralin than the S biotypes when the herbicide was uniformly mixed throughout the entire volume of soil in the pot (simulated PPI treatment), and up to 10 times more resistant when trifluralin was incorporated into the upper 2 cm of soil (simulated PEI treatment).

The expression of resistance in R green foxtail to PPI- and PEI -trifluralin may be markedly different in the field, however, than under controlled environmental conditions. Climate and soil factors strongly influence the efficacy and persistence of trifluralin as well as the growth and competitiveness of both the weed and the crop. If the expression of resistance does differ in the field, either over the growing season, or with the method of herbicide incorporation, this could have important implications for the evolution and dynamics of R green foxtail populations and for recommendations of effective control measures.

There is little data available on effective kill, which is the reduction in weed seed yield over the growing season due to the herbicide treatment. Because effective kill is equated with the selection pressure of the herbicide on the weed, it is the most important parameter in mathematical models for predicting the rate of appearance of resistance in weed biotypes (Gressel and Segel 1982, 1990a). Accurate information on the effective kill could be used in population models, with the objective of formulating possible prevention or management strategies to regulate the rate of enrichment (or decline) of R biotypes in the field. This chapter reports on the results of field experiments to determine the

response of R green foxtail to PPI- and PEI-trifluralin under cropped and non-cropped conditions over the growing season, with primary emphasis on the determination of a true estimate of effective kill.

3.2. Materials and Methods

3.2.1. Site descriptions and preparations

3.2.1.1. Portage la Prairie

The response of known S and R green foxtail biotypes to PPI- and PEI-trifluralin was compared in field experiments conducted at the University of Manitoba Research Station at Portage la Prairie, Manitoba in 1989 and 1990. The soil at the site is a Neuhorst clay loam (25% sand, 44% silt, and 31% clay) with an organic matter content of 7.5% and a pH of 7.4. The land where the experiments were located was sown to spring wheat in the previous year. Fertilizer was broadcast at a rate of 50 kg ha^{-1} N and 50 kg ha^{-1} P_2O_5 (based on soil fertility test results) and disked into the plot area to a depth of 8 to 10 cm prior to seeding.

3.2.1.2. Deloraine

The response of a naturally occurring R green foxtail population to PPI trifluralin in the presence and absence of crop competition was investigated in an experiment established in a field near Deloraine, Manitoba (49° 11′ N; 100° 30′ W) which was uniformly infested with green foxtail that had previously been determined to be trifluralin-resistant using the petri dish bioassay (Beckie et al. 1990). The seed of the R biotype used in the experiments at Portage la Prairie originated from this field. The soil at the site (NW 13-4-23-W1, 10 km north of Deloraine) is a Ryerson clay loam with an organic matter content of 4.0% and a pH of 7.2. The land where the experiment was located was sown to barley (*Hordeum vulgare* L.) in the previous year. Fertilizer N was deep-banded at a

rate of 60 kg ha^{-1} and 40 kg ha^{-1} P_2O_5 was placed with the seed (based on soil fertility test results). Seeding of the trial was contracted with Ag-Quest Inc.[3]

3.2.2. Experimental design

The experiments were arranged in a split-split block (Portage la Prairie) or split-block (Deloraine) design with four replicates. The randomized main plot treatments consisted of eight (Deloraine) and nine (Portage la Prairie) herbicide dosages ranging from 0 to 3 kg ai ha^{-1}. The highest dosage is more than twice the recommended dosage of PPI trifluralin in rapeseed and over three times the recommended dosage of PEI trifluralin in wheat. One-half of each main plot was seeded to crop, whereas the remaining half was left unseeded (Figure 3-1). At the Portage la Prairie site, the S green foxtail biotype was sown on either the front or back half of each block (chosen randomly) and the R biotype was sown on the remaining portion. The dimensions of individual main plots were 5 by 10 m (Portage la Prairie) and 3 by 12 m (Deloraine).

3.2.3. PPI trifluralin

Trifluralin (545 g ai L^{-1} emulsifiable concentrate) was applied on May 18, 1989 and May 14, 1990 at Portage la Prairie and on May 15, 1989 at Deloraine. The herbicide was applied with a bicycle sprayer fitted with flat-fan[4] nozzles on a 2-m boom that delivered a spray volume of 120 L ha^{-1}. Immediately following application, the herbicide was incorporated into the soil to a depth of 8 to 10 cm using a tandem disk, the second pass being perpendicular to the first.

Westar canola was seeded using a double-disk press drill at a rate of 6 kg viable seed ha^{-1} on May 23 in 1989 and in 1990 at Portage la Prairie and on May 20 at Deloraine. The seed was placed 2 to 3 cm deep in rows 15 cm apart.

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[3]Ag-Quest Inc., Box 144, Minto, MB.

[4]Tee Jet 80015. Spraying Systems Co., North Ave. and Schmale Rd., Wheaton, IL 60187.

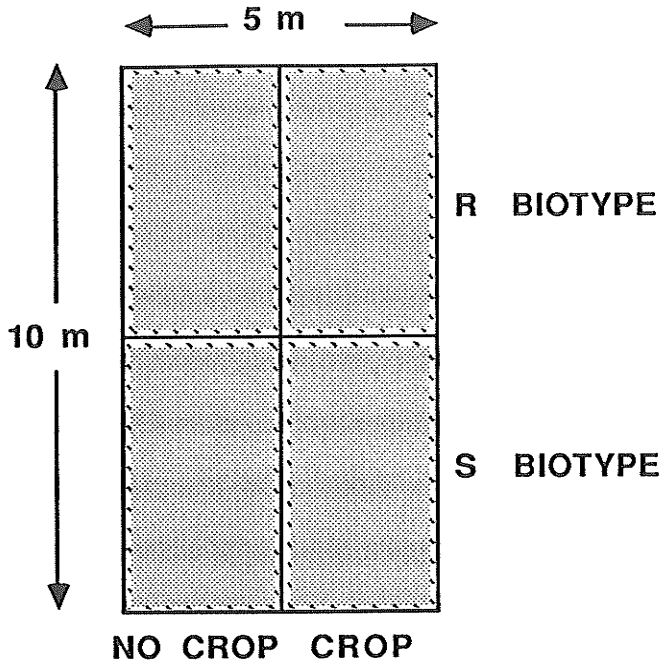

Figure 3-1. Layout and dimensions of a main plot in experiments at Portage la Prairie.

Following seeding at Portage la Prairie, the S and R green foxtail biotypes were hand-broadcast at a rate of 5000 viable seeds m^{-2} and incorporated into the soil to a depth of 2 to 4 cm with a spike-tooth harrow, the second pass being perpendicular to the first. At the Portage la Prairie site, broadleaf weeds were controlled using a mixture of ethametsulfuron [2-[[[[[4-ethoxy-6-(methylamino)-1,3,5-triazin-2-yl]amino]carbonyl]amino]sulfonyl]benzoic acid] (20 g ai ha^{-1}) and clopyralid [3,6-dichloro-2-pyridinecarboxylic acid] (100 g ai ha^{-1}) applied on June 16 in 1989. In 1990, weed infestations were light and controlled by hand-weeding. Grass weeds were also removed by hand in both years.

At both sites, flea beetles (*Phyllotreta* sp.) were controlled using granular terbufos [phosphorodithioic acid S- [[[1, 1-dimethylethyl]thio]methyl]0,0-diethyl-ester] (10%) pre-mixed with the canola seed (1:1), followed by a foliar

application of carbofuran [2,3-dihydro-2,2-dimethyl-7-benzofuranol methyl-carbamate] (0.13 kg ai ha^{-1}) applied on June 14 in 1989 and June 19 in 1990 at Portage la Prairie and on June 8 in 1989 at Deloraine.

3.2.4. PEI trifluralin

Katepwa wheat was seeded with a double-disk press drill at a rate of 100 kg viable seed ha^{-1} on May 23 in 1989 and May 24 in 1990. The seed was placed 5 to 6 cm deep in rows 15 cm apart. Immediately following seeding, trifluralin was applied to the soil and incorporated to a depth of 2 to 4 cm by two harrow passes at right angles to each other. S and R green foxtail were then broadcast and incorporated as described previously. Broadleaf weeds were controlled using thifensulfuron [3-[[[[(4-methoxy-6-methyl-1,3-5-triazin-2-yl)-amino]carbonyl]amino]sulfonyl]-2-thiophenecarboxylate] (10 g ai ha^{-1}) applied on June 16 in 1989 and June 19 in 1990. Flamprop methyl [methyl*N*- benzoyl-*N*-(3-chloro-4-fluorophenyl)-2-amino-propionate] (0.26 kg ai ha^{-1}) was applied on June 16 in 1989 to control wild oat (*Avena fatua* L. # AVEFA). Light infestations in 1990 were controlled by hand-weeding.

3.2.5. Sample collection and processing

Green foxtail and crop samples were collected 4 wk after green foxtail emergence and again at foxtail maturity, prior to general seed shatter. Shoot samples were collected from four 0.25- by 0.25-m quadrats per sub-subplot (Portage la Prairie) or subplot (Deloraine). At crop maturity, four 0.25-m^2 areas within each crop subplot were harvested by hand, and threshed with a plot combine. The number of plants in each sample were counted at both sampling dates. The number of green foxtail and wheat tillers were counted for samples collected at maturity; green foxtail panicles also were counted and separated from the vegetative shoot parts. Samples were weighed after they were oven dried at 80 C for 24 h. The panicles were compressed later by a belt thresher

and the seed was cleaned using a fanning mill. One thousand green foxtail-seed weight was determined for each sub-subplot (Portage la Prairie) or subplot treatment (Deloraine).

3.2.6. Data analyses

For consistency, all data were expressed as a percentage of the untreated controls and the results were combined for the 2 yr (Portage la Prairie) upon confirmation of homogeneity of variances (Gomez and Gomez 1984). Dose-response curves were fitted to the green foxtail data using non-linear regression procedures (Freund and Littell 1986). An exponential decay model was used to describe the response of the S biotype and of the naturally occurring R biotype (Deloraine) to trifluralin, whereas a quadratic model best described the response of the R biotype to the herbicide at Portage la Prairie (Beckie et al. 1990). A symmetrical sigmoidal model (Brain and Cousens 1989) provided a comparable fit to the S biotype data but gave very large asymptotic standard errors when fitted to the R biotype data. Therefore, the sigmoidal model was not chosen because the regression curves that represent the dose-response of S and R biotypes could not be plotted on the same graph. The sigmoidal model requires that dosages be transformed to logarithmic values, thereby preventing simultaneous graphing of the quadratic curve, which is plotted using non-transformed dosages on the abscissa (x) axis. Crop response to trifluralin was described by linear and non-linear (quadratic model) regression. Regression analysis was performed using individual datapoints, but means were plotted. Regression equations were statistically compared when required, using the parameter estimates as described by Ratkowsky (1983). The coefficients of determination (R^2) were calculated as described by Kvalseth (1985). The *t* test was used to compare the means of plant variables from untreated plots.

3.3. Results and Discussion

3.3.1. PPI trifluralin

3.3.1.1. Portage la Prairie

There was no apparent difference in the time of emergence of S and R green foxtail seedlings in untreated plots. Emergence of both biotypes occurred within a week of crop emergence in both years. At the time the experiments were established, soil moisture conditions were good for seed germination, seedling emergence, and a high level of trifluralin activity (Morrison et al. 1990). In addition, in both years, June rains were 50% higher than the long-term average (Table 3-1) and favored foxtail and crop establishment. Mean air temperatures in May and June were normal to above normal (see Appendix Figure 1).

Four wk after foxtail emergence, marked differences occurred between the

Table 3-1. May to August precipitation and mean temperatures at the University of Manitoba Research Station at Portage la Prairie, Manitoba in 1989 and 1990.

	Precipitation		Temperature	
	mm	% of 30-yr mean[a]	C	% of 30-yr mean
1989				
May	24	45	14.7	131
June	124	152	16.5	97
July	32	44	21.8	108
August	65	88	18.8	100
1990				
May	34	64	10.9	97
June	134	164	18.0	106
July	54	74	19.2	95
August	43	57	19.9	106

[a]30-yr mean from 1951 to 1980; Environment Canada Climate Center, 266 Graham Ave, Winnipeg, MB.

S and R biotypes in their response to trifluralin in plots sown to rapeseed (Figure 3-2). The R biotype was 7 times more resistant to the herbicide than the S biotype, based on ED_{50}[5] values from density and biomass determinations (Table 3-2). In the untreated cropped plots, no differences were observed between S and R foxtail densities. Although these plant densities (3100 m^{-2}) are higher than those reported in field surveys (Thomas and Wise 1988), over 5000 plants m^{-2} were observed in some fields that were naturally infested with R green foxtail (personal observation, June 1989).

As trifluralin dosages increased, rapeseed density and dry matter were affected little (Figure 3-3), as indicated by the coefficients of determination (R^2) (Table 3-3). The crop response was the same in plots sown either to S or R foxtail.

Under non-cropped conditions (Plate 1 and Figure 3-2), there was a comparable difference between the biotypes in their sensitivity to trifluralin as compared to cropped conditions (Table 3-2). As expected, foxtail density and shoot dry matter were higher under non-cropped as compared to cropped conditions due to the absence of crop competition. As in the untreated cropped plots, no differences were observed between the density or biomass of S or R biotypes in untreated non-cropped plots.

At green foxtail maturity, the differences between the biotypes in their response to trifluralin were slightly reduced in cropped plots compared to the earlier sampling date (Figure 3-4). Rapeseed density and dry matter increased marginally with increasing dosages (Figure 3-5), as a consequence of greater foxtail control with higher dosages. Both crop variables, density and dry matter,

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[5]Abbreviations: ED_{50}, effective dosage required to reduce the plant variable (eg. density, biomass) by 50% relative to the control.

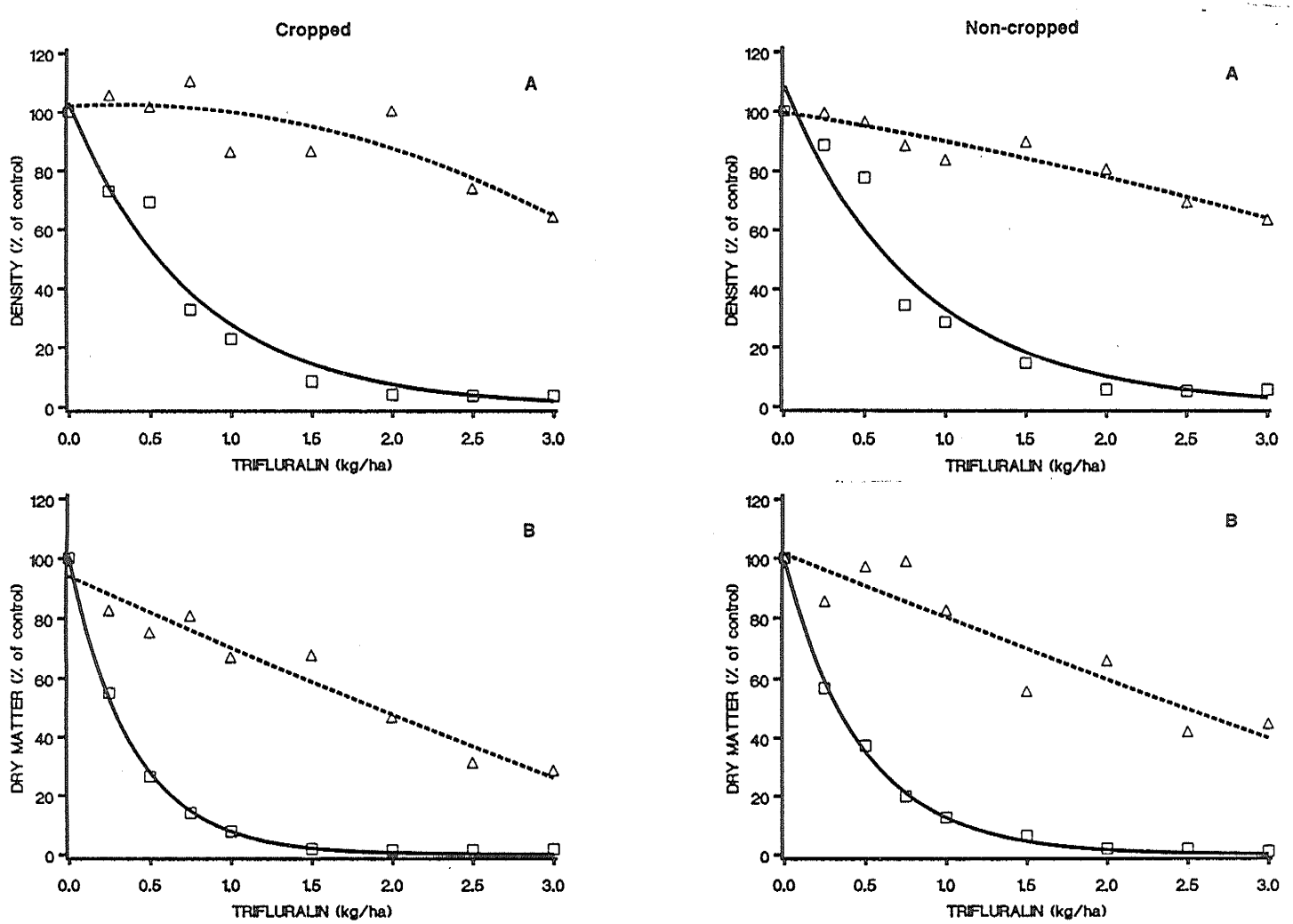

Figure 3-2. The effect of increasing dosages of PPI trifluralin on R (dashed line) and S (solid line) green foxtail density (A) and dry matter production (B) 4 wk after emergence under cropped and non-cropped conditions at Portage la Prairie in 1989 and 1990. See Table 3-2 for equations and parameter estimates.

Table 3-2. Parameter estimates (standard errors in parentheses) and ED_{50}'s of the equations for the regression curves for the response of S and R green foxtail to PPI trifluralin under cropped and non-cropped conditions at Portage la Prairie in 1989 and 1990.

Plant variable[a]	a[b]	b	c	R^{2c}	ED_{50}	R/S
4 wk after emergence						
Cropped conditions						
S density	103.3(4.7)	-1.3(0.1)		0.84**	0.5	
R density	102.2(7.4)	2.7(13.3)	-5.1(4.3)	0.19**	3.4	7
S dry wt	100.5(3.1)	-2.6(0.2)		0.93**	0.3	
R dry wt	94.1(4.9)	-24.4(8.7)	0.6(2.8)	0.61**	2.0	7
Non-cropped conditions						
S density	108.4(5.3)	-1.2(0.1)		0.81**	0.6	
R density	99.6(6.5)	-8.8(11.2)	-1.1(3.7)	0.19**	3.9	6
S dry wt	99.2(4.2)	-2.1(0.2)		0.87**	0.3	
R dry wt	101.4(4.6)	-21.8(10.2)	0.4(3.5)	0.36**	2.4	8
Maturity						
Cropped conditions						
S density	114.4(6.4)	-1.4(0.2)		0.79**	0.5	
R density	93.9(5.4)	-30.0(9.8)	4.3(3.3)	0.42**	2.4	5
S dry wt	99.7(3.1)	-2.8(0.2)		0.93**	0.3	
R dry wt	95.4(6.7)	-29.9(12.1)	1.8(4.0)	0.48**	1.8	6
S seed wt	98.9(4.1)	-3.1(0.3)		0.89**	0.2	
R seed wt	92.9(9.2)	-24.0(16.6)	-0.4(5.6)	0.37**	1.9	9
Non-cropped conditions						
S density	110.8(4.1)	-1.2(0.1)		0.89**	0.6	
R density	100.0(5.0)	4.7(5.8)	-5.5(2.3)	0.25**	3.5	6
S dry wt	100.5(5.9)	-0.6(0.1)		0.62**	1.1	
R dry wt	92.6(5.9)	13.9(10.3)	-7.3(3.4)	0.15**	3.6	3
S seed wt	102.3(7.8)	-0.6(0.1)		0.49**	1.1	
R seed wt	94.6(7.8)	10.1(13.0)	-5.3(4.2)	0.06*	4.1	4

[a]Mean values ± standard error for plant variables (per 1-m^2 basis, wt in g) in control plots: **4 wk, cropped conditions** Density: S 3 140(280), R 3 100 (340); Dry wt: S 84(12), R 100(13); **4 wk, non-cropped conditions** Density: S 3 970(370), R 4 300(340); Dry wt: S 159(15), R 155(12); **Maturity, cropped conditions** Density: S 1 610(150), R 2 380(90); Dry wt: S 270(47), R 300(11); Seed wt: S 44(9), R 50(5); **Maturity, non-cropped conditions** Density: S 2 760(240), R 3 080(140); Dry wt: S 725(79), R 808(70); Seed wt: S 203(17), R 220(17).

[b]Exponential function equation: $y = a\, e^{bx}$ where a = intercept (% of control) and ab = initial slope; quadratic function equation: $y = a + bx + cx^2$ where a = intercept (% of control), b = linear coefficient, and c = curvilinear coefficient; y is the plant variable (% of control) and x is the trifluralin dosage (kg ha^{-1}).

[c]Coefficient of determination: significant at the 5% level (*), 1% level (**).

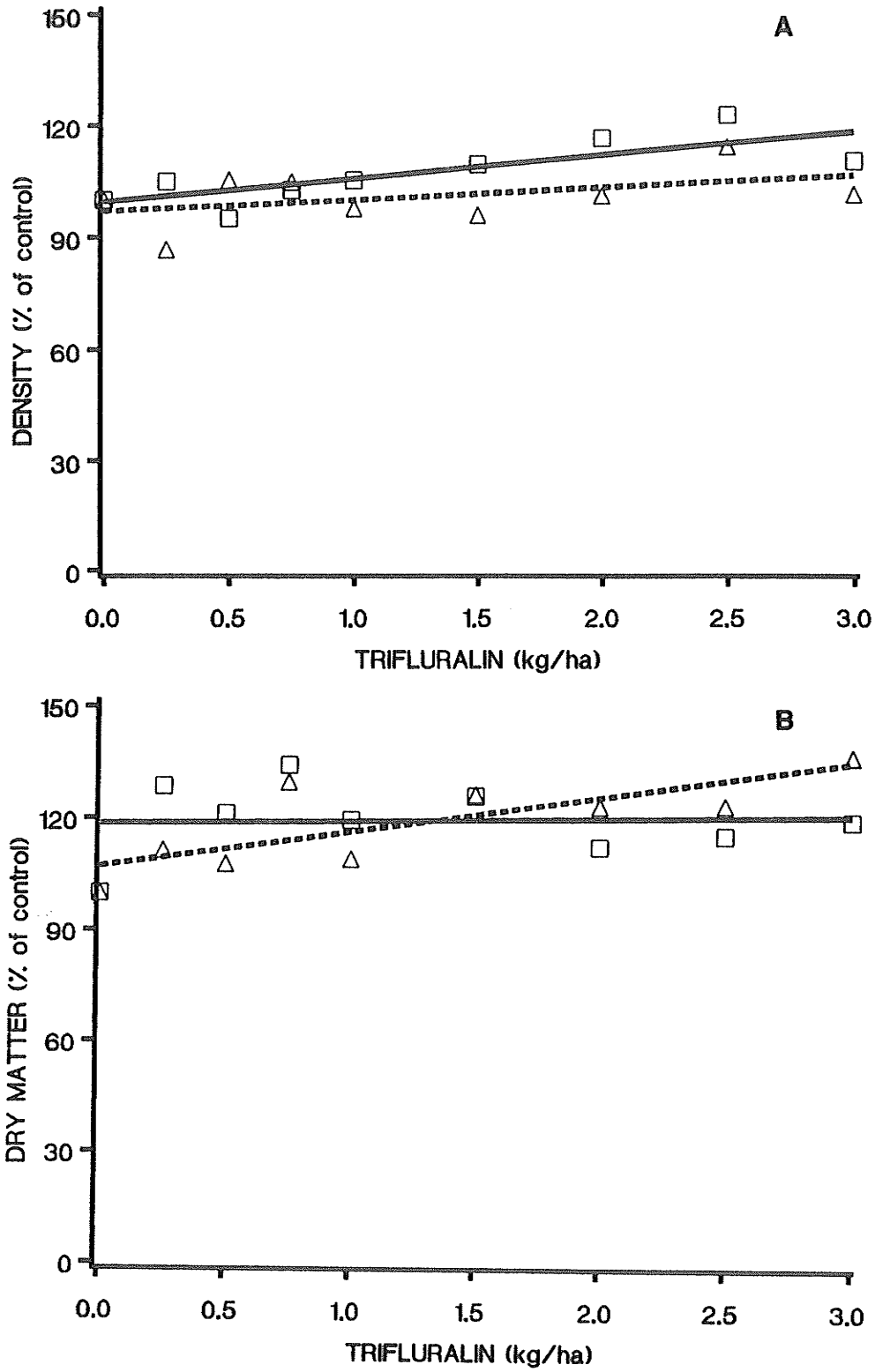

Figure 3-3. The effect of increasing dosages of trifluralin applied as a PPI treatment on rapeseed density (A) and dry matter production (B) 4 wk after foxtail emergence, in plots sown to R (dashed line) and S (solid line) biotypes at Portage la Prairie in 1989 and 1990. See Table 3-3 for equations and parameter estimates.

Table 3-3. Parameter estimates (standard errors in parentheses) of the equations for the regression curves for the response of rapeseed to PPI trifluralin at Portage la Prairie in 1989 and 1990.

Crop variable	a[a]	b	R^{2b}
4 wk after emergence[c]			
S[d] density[e]	99.6 (4.9)	6.0 (3.0)	0.06*
R density	97.0 (4.0)	3.0 (2.5)	0.02
S dry wt	118.6 (7.6)	0.7 (4.9)	0.01
R dry wt	107.2 (7.9)	9.2 (5.0)	0.05
Maturity			
S density	108.5 (3.9)	7.7 (2.4)	0.14**
R density	88.9 (4.2)	4.6 (2.6)	0.04
S dry wt	111.6 (4.3)	9.4 (2.7)	0.18**
R dry wt	89.5 (6.1)	10.6 (3.8)	0.10**
S seed wt	114.7 (7.9)	8.8 (4.9)	0.05
R seed wt	109.2 (10.2)	11.1 (6.6)	0.05

[a]Linear function equation: $y = a + bx$ where a = intercept (% of control), b = slope, y is the crop variable (% of control), and x is the trifluralin dosage (kg ha^{-1}).

[b]Coefficients of determination: * significant at the 5% level; ** significant at the 1% level.

[c]First sampling date: 4 wk after green foxtail emergence (stem extension to early bud crop development stages); second sampling date: green foxtail maturity (crop density and shoot dry matter) and crop maturity (yield).

[d]Crop in competition with trifluralin-susceptible (S) or trifluralin-resistant (R) green foxtail biotypes.

[e]In control plots, mean values ± standard errors (per 1 m^2, wt in g) for select variables were: **4 wk** Density: S 107(10), R 107 (8); **Maturity** Seed wt: S 205(38), R 165(23).

were significantly lower in treated plots of R foxtail as compared to S foxtail, presumably due to the greater degree of interference by the former biotype. However, crop yields were similar in R and S foxtail plots, and were not significantly affected by increasing trifluralin dosages.

Under non-cropped conditions (Figure 3-4), the difference in sensitivity between S and R plants to trifluralin, based on density determinations, was maintained since the first sampling date (R/S=6). However, this difference in

Plate 1. Effect of 3.0 kg ai ha^{-1} PPI trifluralin on R (foreground) and S (background) green foxtail 4 wk after emergence at Portage la Prairie in 1989.

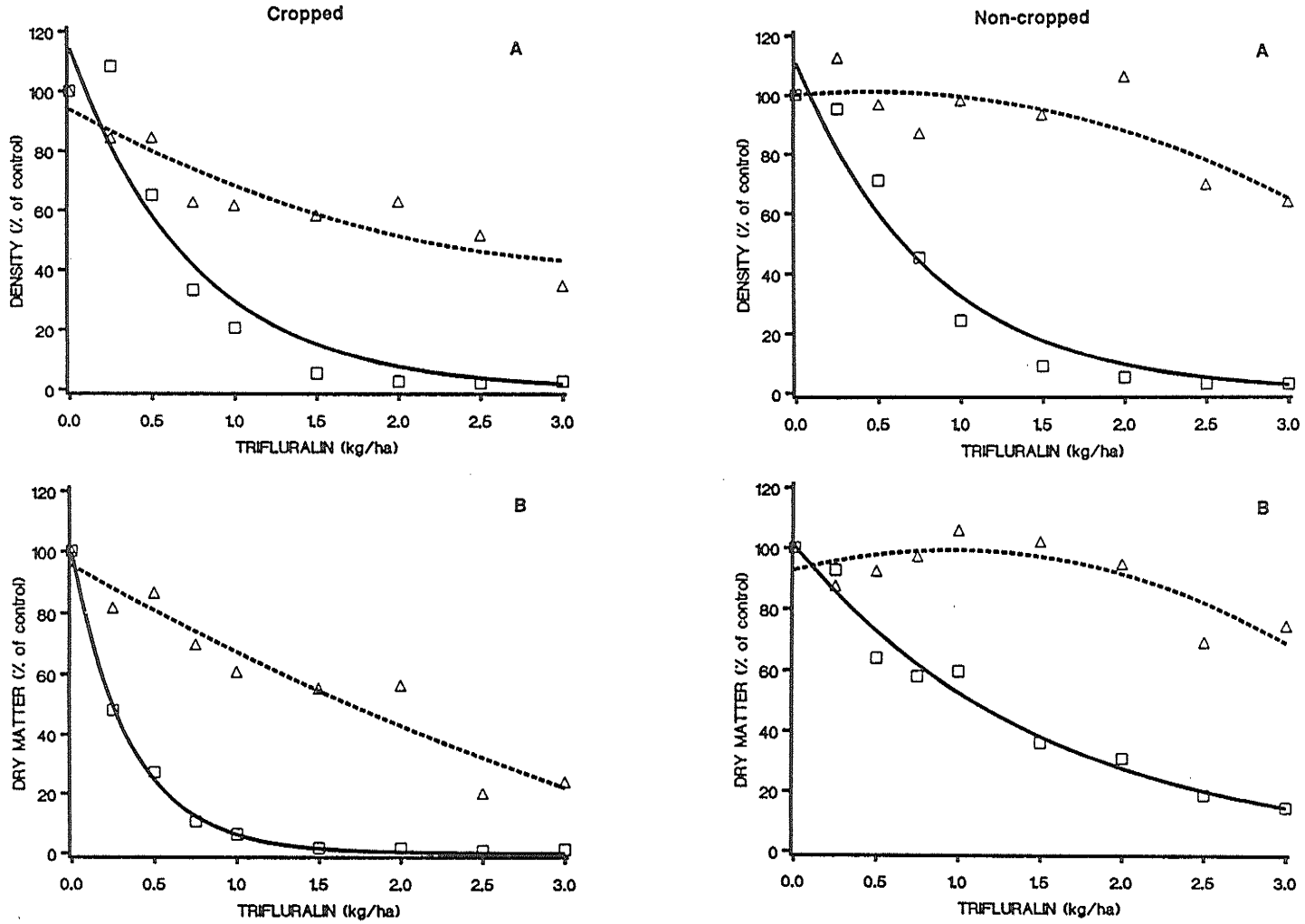

Figure 3-4. The effect of increasing dosages of PPI trifluralin on R (dashed line) and S (solid line) green foxtail density (A) and dry matter production (B) at maturity under cropped and non-cropped conditions at Portage la Prairie in 1989 and 1990. See Table 3-2 for equations and parameter estimates.

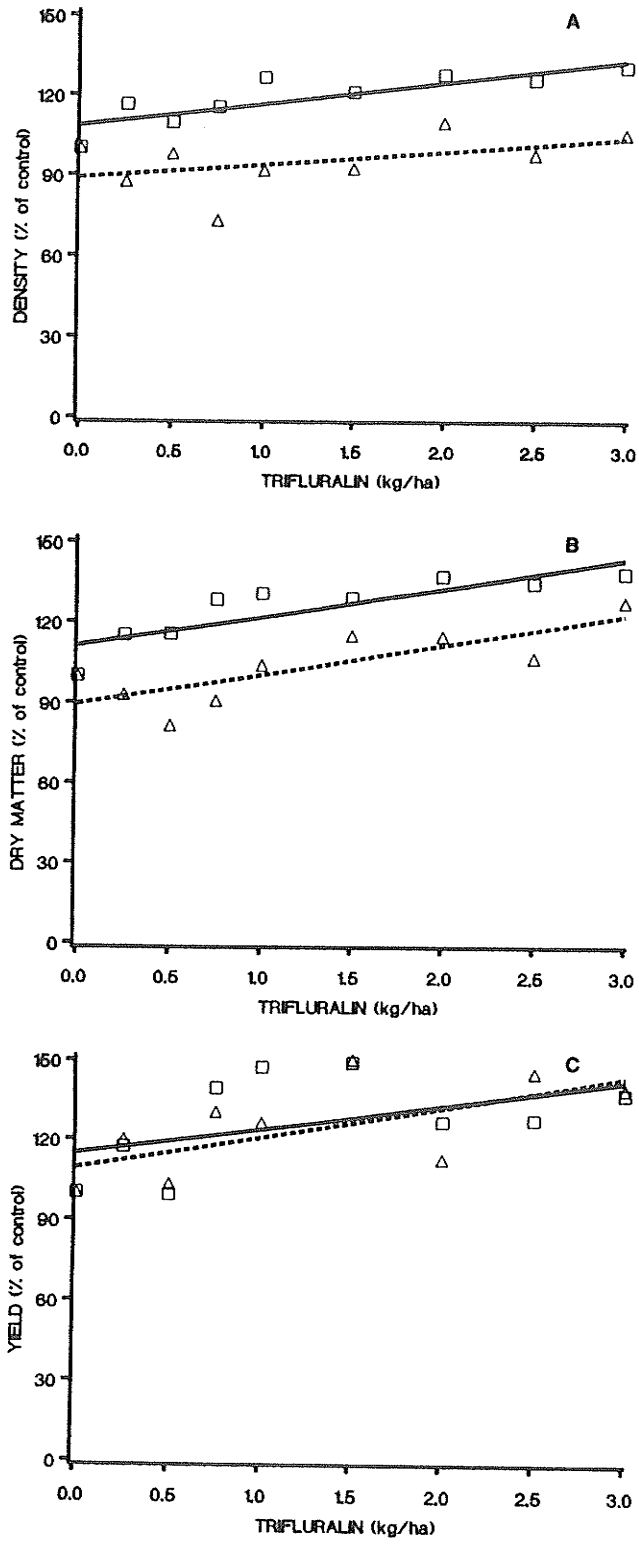

Figure 3-5. The effect of increasing dosages of trifluralin applied as a PPI treatment on rapeseed density (A), dry matter production (B), and yield (C) at maturity, in plots sown to R (dashed line) and S (solid line) biotypes at Portage la Prairie in 1989 and 1990. See Table 3-3 for equations and parameter estimates.

sensitivity between the biotypes, when calculated from biomass determinations, was less than at the earlier sampling date. This was due primarily to enhanced tillering of S plants at higher dosages (Figure 3-6), as a consequence of decreasing foxtail density.

Under cropped conditions, 9 times more herbicide was required to reduce R seed production by 50% than to reduce S seed production by the same amount (see Figure 3-7, Table 3-2, Appendix Figure 2, and Appendix Table 1). However, under non-cropped conditions, enhanced tillering of the S biotype at higher trifluralin dosages markedly reduced the effect of increasing dosages of the chemical on S seed return. No significant differences were observed in seed return between the S and R biotypes in untreated plots under either

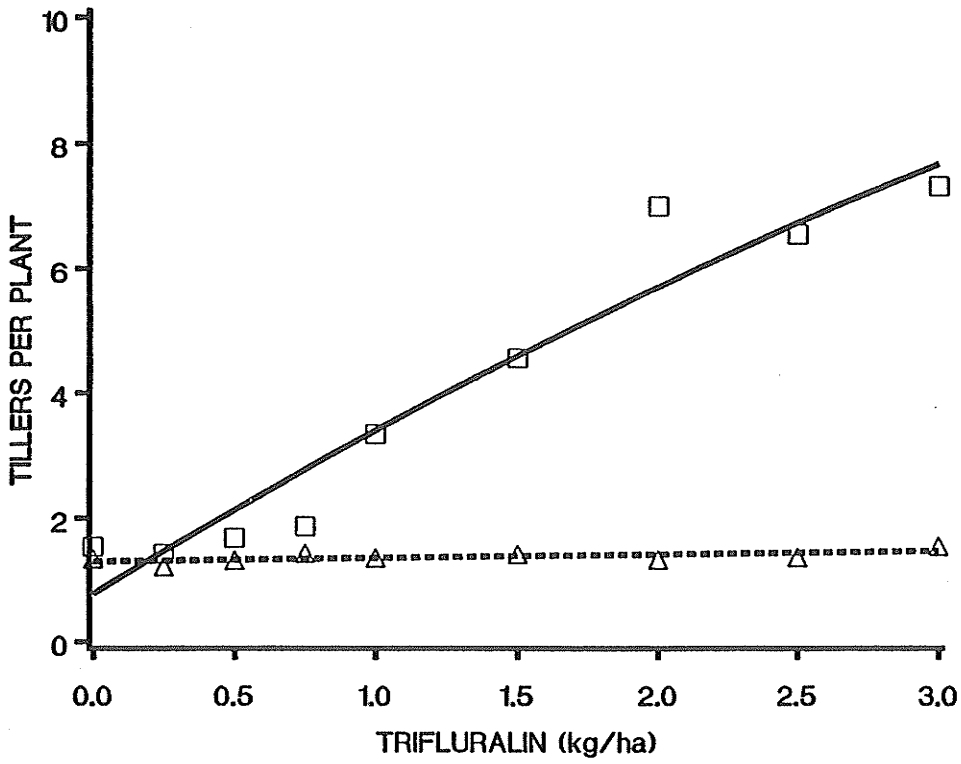

Figure 3-6. The effect of increasing dosages of PPI trifluralin on R (dashed line) and S (solid line) green foxtail tillers per plant at maturity under non-cropped conditions at Portage la Prairie in 1989 and 1990. R^2 for R and S biotypes = 0.05 and 0.48**, respectively.

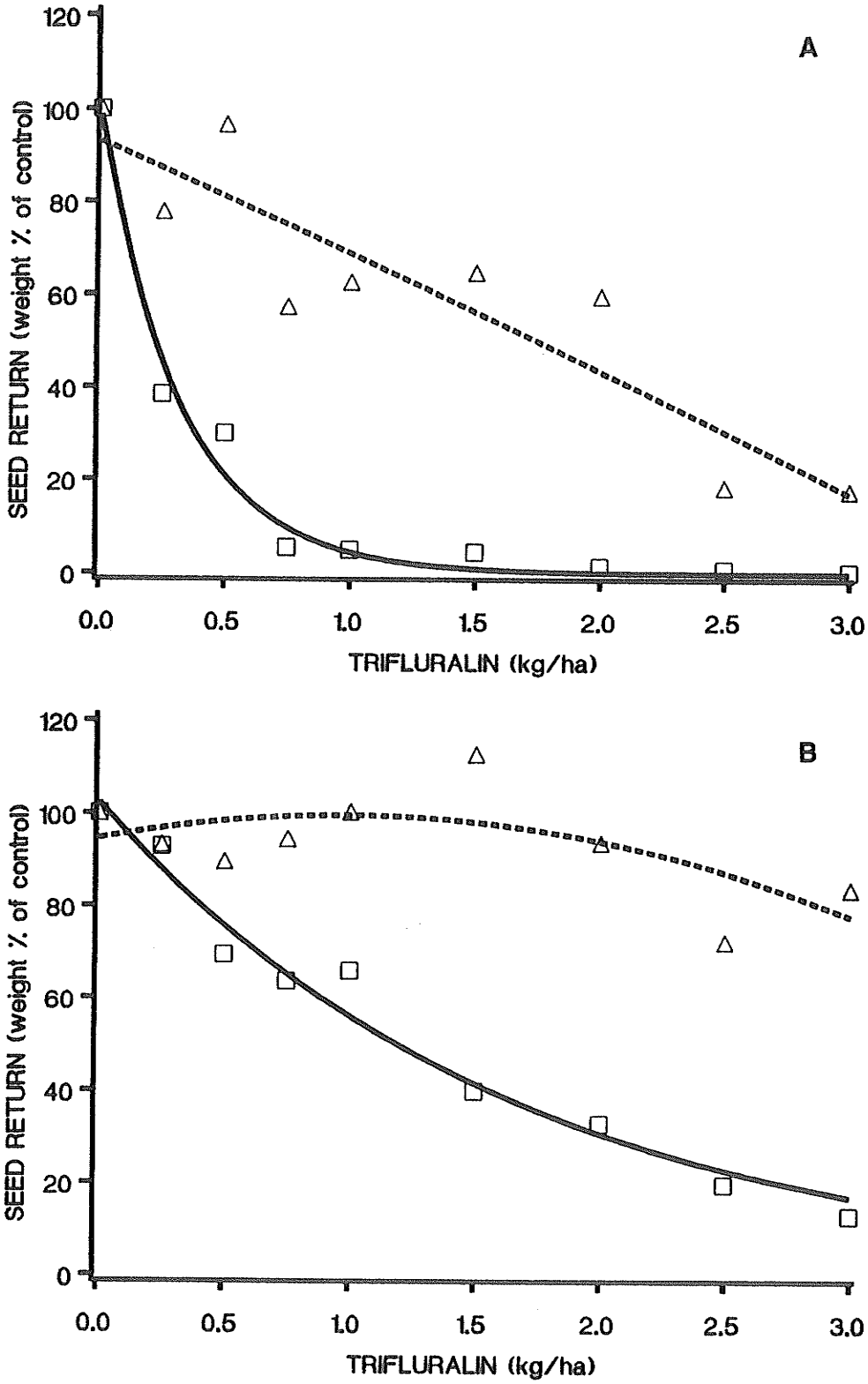

Figure 3-7. The effect of increasing dosages of PPI trifluralin on R (dashed line) and S (solid line) green foxtail seed production under cropped (A) and non-cropped (B) conditions at Portage la Prairie in 1989 and 1990. See Table 3-2 for equations and parameter estimates.

cropped or non-cropped conditions. This similar reproductive vigor may indicate a negligible difference in relative fitness between these two biotypes.

Calculated from the dose-response equations (see Table 3-2), at the recommended trifluralin dosage in rapeseed (1.4 kg ha^{-1})[6], the density of S plants 4 wk after emergence was reduced by 84% compared to untreated plots, whereas the density of R plants was reduced by only 4% (Table 3-4). The biomass of S and R biotypes were reduced to a greater extent - 97% and 39%, respectively. Similarly, seed return (weight per unit area) of S and R foxtail were reduced by 99% and 42%, respectively. Therefore, half of the potential R seed production is returned to the seed bank to germinate the following year.

Table 3-4. Percent reduction in S and R green foxtail variables in response to PPI trifluralin at the recommended dosage in rapeseed (1.4 kg ha^{-1}) at Portage la Prairie in 1989 and 1990.

	Cropped		Non-cropped	
	S	R	S	R
	% reduction[a]			
4 wk after emergence				
Density	84	4	79	15
Dry wt	97	39	94	28
Maturity				
Density	83	40	80	4
Dry wt	98	43	59	2
Seed wt	99	42	56	2
Seed no	99	44	57	1

[a]Percent reduction calculated from the regression equations at the application dosage of 1.4 kg ha^{-1}.

[6]Highest recommended dosage.

At the recommended dosage, the effective kill (seed yield reduction) of S and R green foxtail is equal to or greater than the initial reductions in plant density or dry weight. For herbicides with less residual activity, the effective kill might be expected to be significantly less than the initial reductions in density or biomass due to late germination of weed seeds after the herbicide is dissipated, as well as the capacity of surviving plants to compensate after herbicide thinning (Gressel and Segel 1982). In population models which describe the rate of enrichment of R weed biotypes, inaccurate results will be obtained if the selection pressure is equated with the initial weed control rather than estimated from effective kill. In this experiment, the selection pressure (seed yield of R plants, expressed as a proportion of the control, that survive the recommended herbicide dosage divided by the relative seed yield of surviving S plants) of trifluralin on green foxtail under cropped conditions was: (1-0.42)/(1-0.99) = (0.58/0.01) = 58.

3.3.1.2. Deloraine

Green foxtail seedlings emerged at approximately the same time as the crop. Although total precipitation in May was below the long-term average (Table 3-5), most of the rainfall occurred from the 16th to the 25th when the

Table 3-5. May to August precipitation and mean temperatures at Deloraine, Manitoba in 1989.

Month	Precipitation		Temperature	
	mm	% of 30-yr mean[a]	C	% of 30-yr mean
May	43	77	13.8	124
June	116	135	16.3	97
July	18	27	21.8	112
August	27	38	19.7	109

[a]30-yr mean from 1951 to 1980; Environment Canada Climate Center, 266 Graham Ave, Winnipeg, MB.

experiment was established (see Appendix Figure 3). Hence, the combination of adequate soil moisture conditions and above normal temperatures favored foxtail seed germination and emergence, as well as trifluralin activity in soil. Similarly, in June, total precipitation was above normal and the mean temperature was nearly normal, providing suitable conditions for growth of both green foxtail and rapeseed.

A soil residue level of 0.5 kg ha^{-1} trifluralin was detected at seeding time, prior to application of herbicide treatments[7]. The soil was sampled using eight cores, 7.5 cm in diameter by 10.5 cm deep, randomly selected from each control plot. The cores from each plot were combined, air dried at room temperature for 48 h, and stored in plastic bags in the dark at -30 C until they were analyzed by gas liquid chromatography (Smith 1981; Grover et al. 1988). The high residue level in the soil was the result of trifluralin being applied at the oilseed dosage (0.8 kg ha^{-1}) in the fall of 1987 and half of that dosage in the spring of 1988, in conjunction with unusually dry soil moisture conditions that year. Therefore the ED_{50}'s calculated from the experiment results were adjusted to correct for this pre-existing residue.

The expression of resistance in green foxtail 4 wk after emergence was similar in both cropped and non-cropped plots (Figure 3-8), as indicated by the dose-response of density and shoot dry matter. ED_{50}'s under both cropped and non-cropped conditions were 1.3 kg ha^{-1} (density) and 1.0 kg ha^{-1} (biomass) (Table 3-6). These values are noticeably lower than those determined in the Portage la Prairie experiment. These differences are probably due to the lower organic matter content of the Deloraine soil, which would enhance trifluralin

[7]Soil residue analysis performed by A. Smith, Agriculture Canada, Regina, SK S4P 3A2.

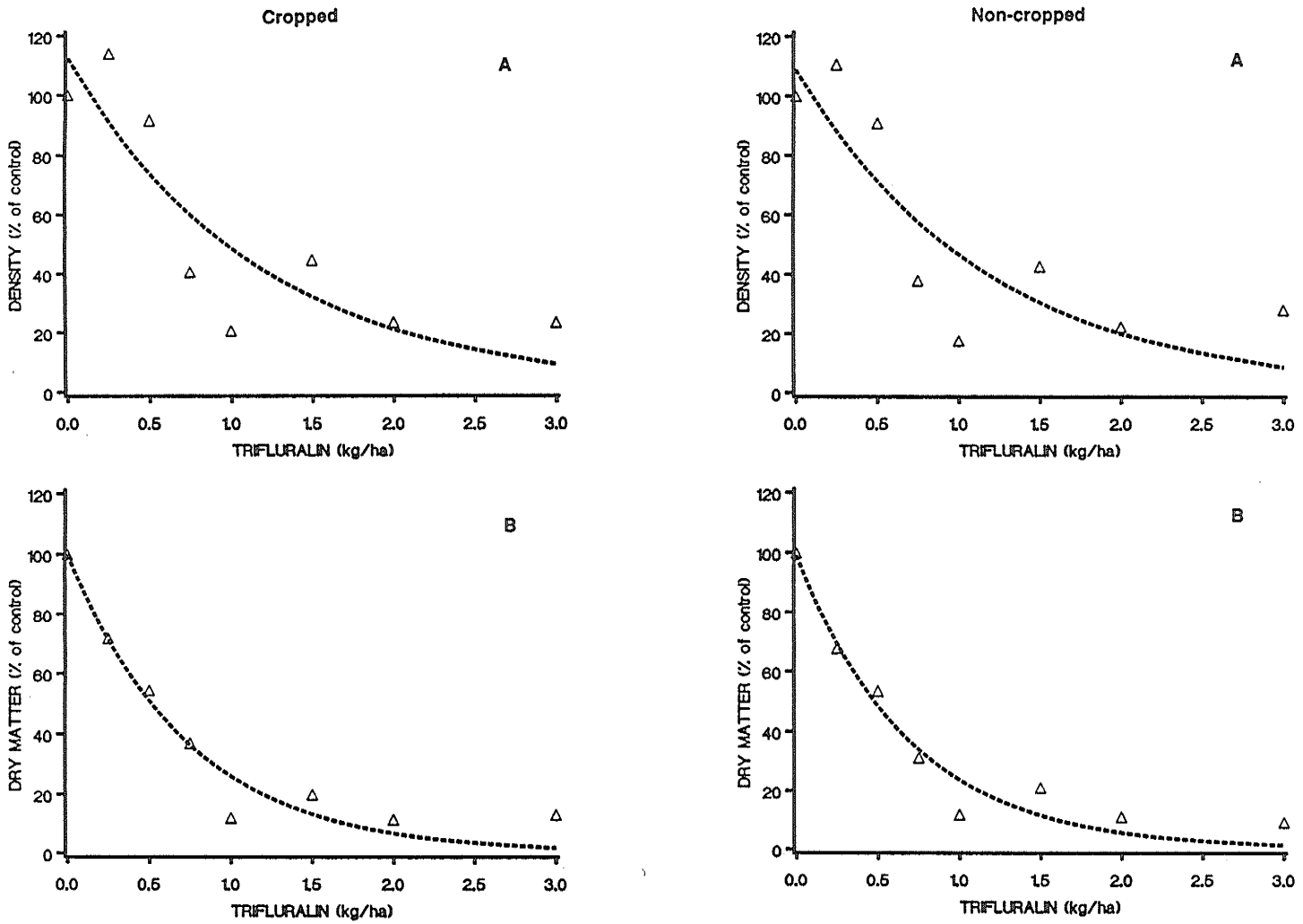

Figure 3-8. The effect of increasing dosages of PPI trifluralin on R green foxtail density (A) and dry matter production (B) 4 wk after emergence under cropped and non-cropped conditions at Deloraine in 1989. See Table 3-6 for equations and parameter estimates.

Table 3-6. Parameter estimates (standard errors in parentheses) and ED_{50}'s of the equations for the regression curves for the response of R green foxtail to PPI trifluralin under cropped and non-cropped conditions at Deloraine in 1989.

Plant variable[b]	a[c]	b	c	R^2[d]	ED_{50}[a] (kg ha^{-1}) Inter-polated	ED_{50}[a] (kg ha^{-1}) Adjusted
4 wk after emergence						
Cropped conditions						
Density	112.5(10.9)	-0.8(0.2)		0.58	0.8	1.3
Dry wt	100.0(5.3)	-1.4(0.1)		0.87	0.5	1.0
Non-cropped conditions						
Density	109.0(10.0)	-0.8(0.2)		0.61	0.8	1.3
Dry wt	99.0(6.0)	-1.4(0.2)		0.84	0.5	1.0
Maturity						
Cropped conditions						
Density	108.4(9.1)	-1.2(0.2)		0.72	0.6	1.1
Dry wt	106.1(7.9)	-1.5(0.2)		0.79	0.5	1.0
Seed wt	85.9(15.8)	-1.1(0.4)		0.34	0.6	1.1
Non-cropped conditions						
Density	108.9(8.6)	-0.5(0.1)		0.55	1.4	1.9
Dry wt	99.5(3.8)	-0.3(0.1)		0.70	2.6	3.1
Seed wt	100.2(11.9)	51.8(20.9)	-15.3(6.8)	0.18		

[a]ED_{50}'s were calculated by the addition of the carryover residue level of 0.5 kg ha^{-1} detected at seeding time to the application dosages (not determined for seed wt (non-cropped conditions) due to the positive slope of the regression curve).

[b]Mean values ± standard error for plant variables (per 1-m^2 basis, wt in g) in control plots: **4 wk, cropped conditions** Density 5 330(270), Dry wt 74(8); **4 wk, non-cropped conditions** Density 5 310(210), Dry wt 80(7); **Maturity, cropped conditions** Density 2 860(360), Dry wt 380(32), Seed wt 2.8(0.5); **Maturity, non-cropped conditions** Density 3 290(240), Dry wt 669(52), Seed wt 33(4).

[c]Exponential function equation: $y = a\, e^{bx}$ where a = intercept (% of control) and ab = initial slope; quadratic function equation: $y = a + bx + cx^2$ where a = intercept (% of control), b = linear coefficient, and c = curvilinear coefficient; y is the plant variable (% of control) and x is the trifluralin dosage (kg ha^{-1}).

[d]All coefficients of determination are significant at the 1% level.

phytotoxicity (Grover et al. 1979; Moyer 1979). Nevertheless, these ED_{50}'s are higher than the recommended dosage of trifluralin in rapeseed for this soil type (0.8 kg ha^{-1}).

A comparison of the results from cropped and non-cropped areas indicated that the response of the resistant foxtail to trifluralin was not affected by the crop during the first 4 wk of growth. No differences in either foxtail density (5300 plants m^{-2}) or biomass were observed between control cropped and non-cropped plots. Similar to the results at Portage la Prairie (Figure 3-9, Table 3-7), rapeseed density and biomass 4 wk after foxtail emergence were unaffected by increasing trifluralin dosages. At the recommended dosage of

Table 3-7. Parameter estimates (standard errors in parentheses) of the equations for the regression curves for the response of rapeseed to PPI trifluralin at Deloraine in 1989.

Crop variable	a[a]	b	c	R^{2b}
4 wk after emergence[c]				
Density[d]	110.0 (6.8)	-0.3 (4.6)		0.01
Dry wt	111.8 (8.4)	1.3 (5.8)		0.01
Maturity				
Density	105.0 (8.9)	52.2 (15.2)	-16.5 (6.2)	0.14**
Dry wt	109.6 (15.7)	63.2 (28.0)	-14.3 (9.0)	0.25**
Seed wt	90.0 (21.8)	37.5 (36.6)	-4.0 (12.1)	0.17**

[a]Linear function equation: $y = a + bx$ where a = intercept (% of control), b = slope; quadratic function equation: $y = a + bx + cx^2$ where a = intercept (% of control), b = linear coefficient, and c = curvilinear coefficient. In both equations, y is the crop variable (% of control) and x is the trifluralin dosage (kg ha^{-1}).

[b]Coefficients of determination: * significant at the 5% level; ** significant at the 1% level.

[c]First sampling date: 4 wk after green foxtail emergence; second sampling date: green foxtail maturity (crop density and shoot dry matter) and crop maturity (yield).

[d]In control plots, mean values ± standard errors (per 1 m^2, wt in g) for select variables were: **4 wk** Density 98(14); **Maturity** Seed wt 66(17).

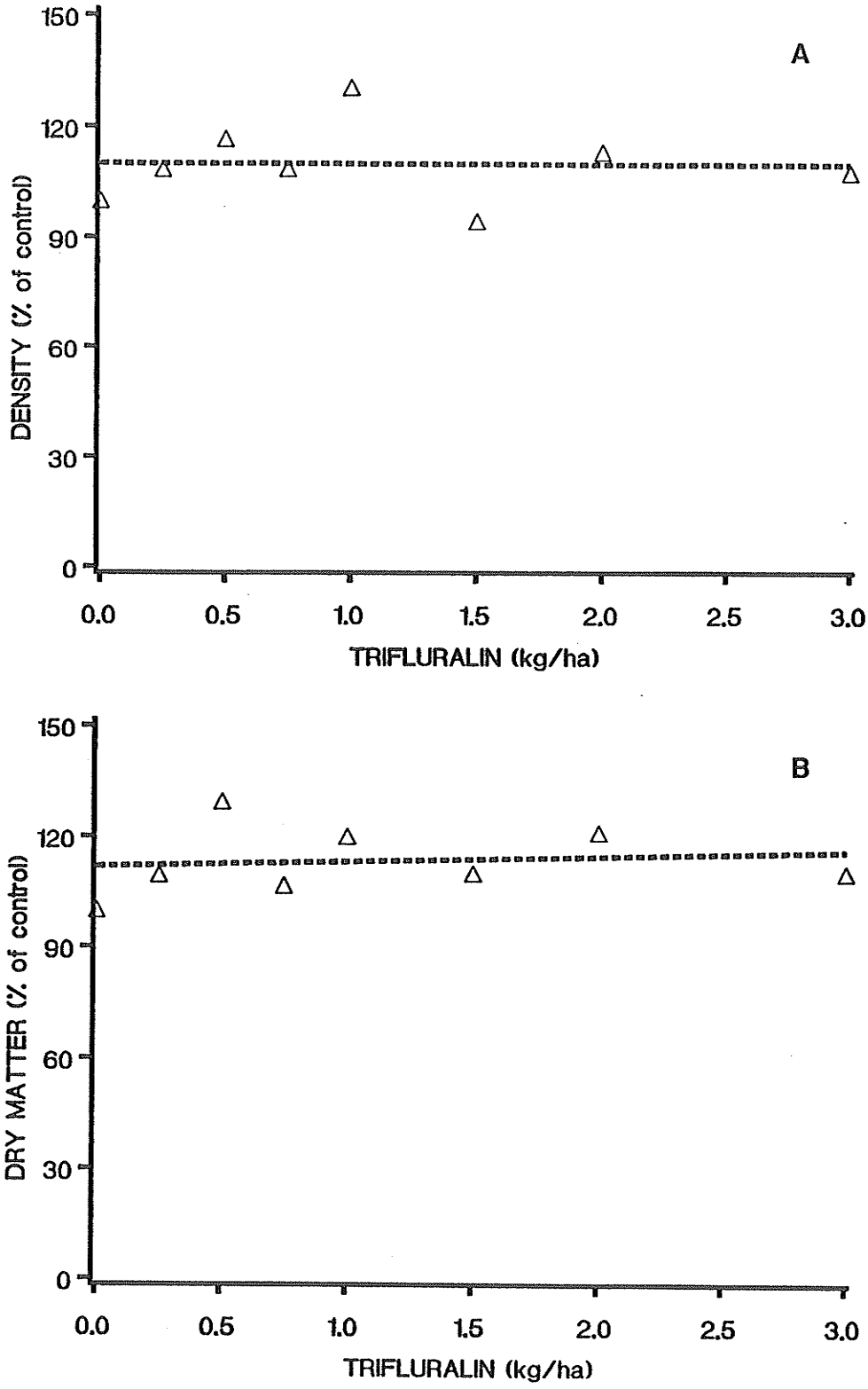

Figure 3-9. The effect of increasing dosages of trifluralin applied as a PPI treatment on rapeseed density (A) and dry matter production (B) 4 wk after foxtail emergence at Deloraine in 1989. See Table 3-7 for equations and parameter estimates.

trifluralin in rapeseed (0.8 kg ha^{-1}), the density and biomass of green foxtail were reduced by 12% and 34%, respectively, compared to the untreated control (Table 3-8).

At green foxtail maturity, the ED_{50} values determined from the dose-response of foxtail density and biomass in rapeseed plots (Figure 3-10) were similar to those calculated for the first sampling date. In the control plots, the density of foxtail was about half that recorded 4 wk after emergence, with the reduction likely due to natural density-dependent mortality. Rapeseed density and biomass generally increased as trifluralin dosages increased, almost certainly the result of a reduction in green foxtail interference (Figure 3-11). However, rapeseed yields increased the most, such that at 3 kg ha^{-1} trifluralin the yield was 1.5 times greater than in control plots.

Table 3-8. Percent reduction in R green foxtail variables in response to PPI trifluralin at the recommended dosage in rapeseed (0.8 kg ha^{-1}) at Deloraine in 1989.

	Cropped	Non-cropped[a]
Variable	% reduction[b]	
4 wk after emergence		
Density	12	16
Dry wt	34	35
Maturity		
Density	25	6
Dry wt	32	8
Seed wt	38	
Seed no	35	

[a]No reduction in seed variables due to the positive slope of the regression curves.

[b]Percent reduction calculated from the regression equations at the application dosage of 0.3 kg ha^{-1} due to the carryover residue level of 0.5 kg ha^{-1} detected at seeding time.

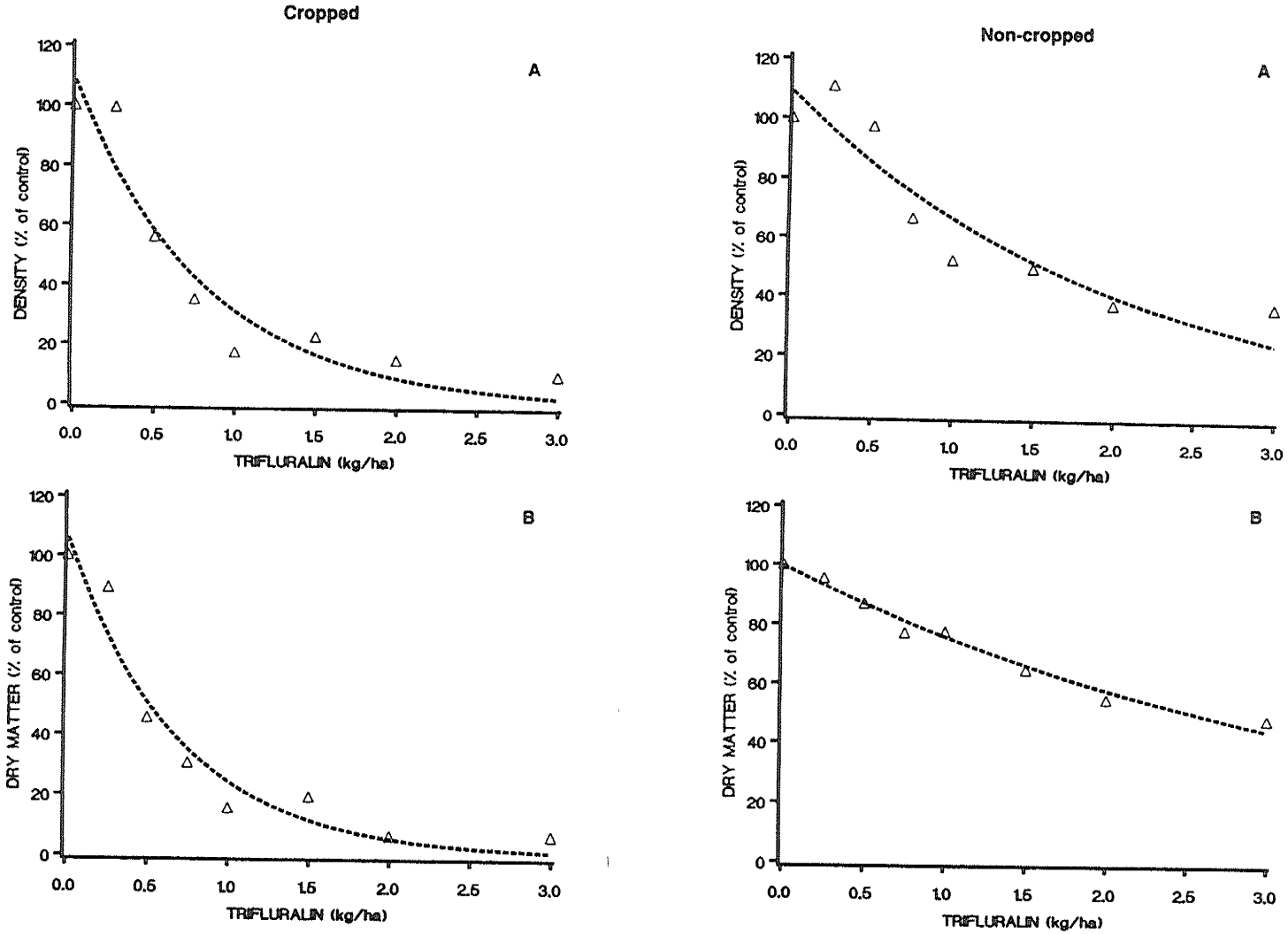

Figure 3-10. The effect of increasing dosages of PPI trifluralin on R green foxtail density (A) and dry matter production (B) at maturity under cropped and non-cropped conditions at Deloraine in 1989. See Table 3-6 for equations and parameter estimates.

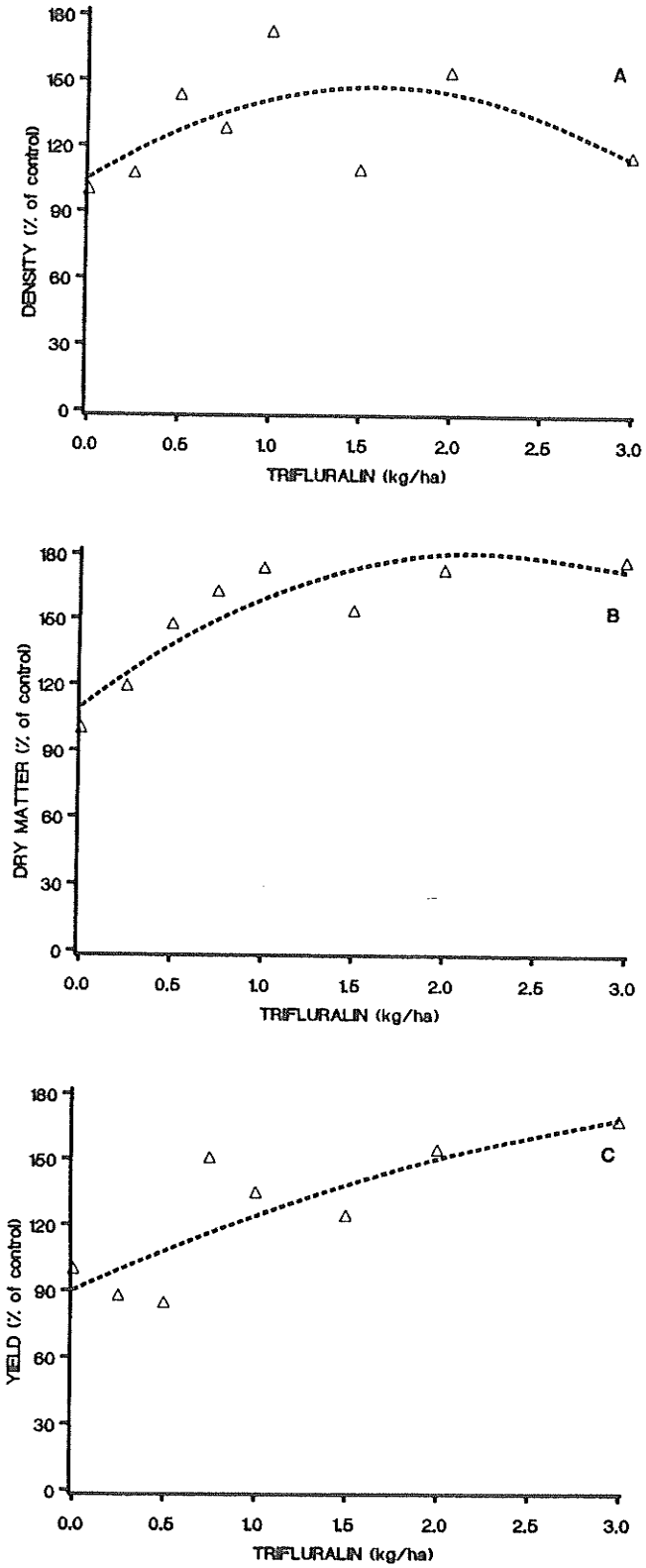

Figure 3-11. The effect of increasing dosages of trifluralin applied as a PPI treatment on rapeseed density (A), dry matter production (B), and yield (C) at maturity at Deloraine in 1989. See Table 3-7 for equations and parameter estimates.

Under non-cropped conditions (Figure 3-10), the ED_{50} for the dose-response of foxtail density was greater than that for the first sampling date. The value for dry matter had tripled, because of enhanced tillering (Figure 3-12) associated with decreased plant density caused by herbicide thinning. Therefore, the response of foxtail biomass to trifluralin was much less sensitive when measured at maturity compared to the first sampling date.

Trifluralin affected foxtail seed production differently under cropped and non-cropped conditions (Figure 3-13, Appendix Figure 4, and Appendix Table 2). Where foxtail were competing with rapeseed, seed return declined exponentially as the dosage increased. In contrast, in the non-cropped plots

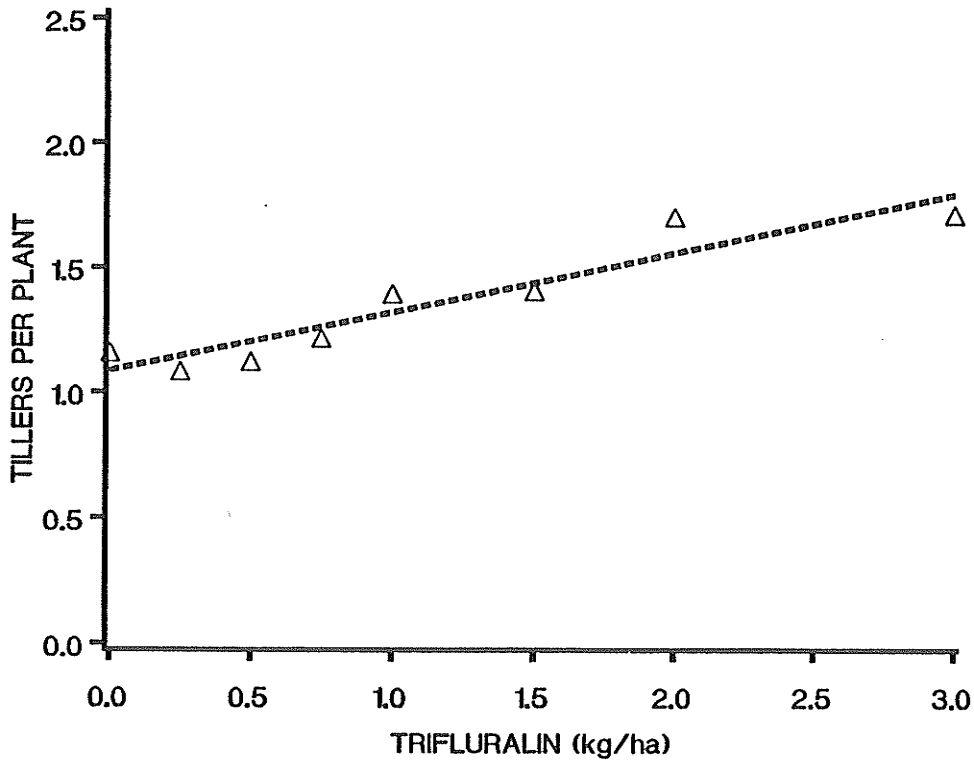

Figure 3-12. The effect of increasing dosages of PPI trifluralin on R green foxtail tillers per plant at maturity under non-cropped conditions at Deloraine in 1989 ($R^2 = 0.49^{**}$).

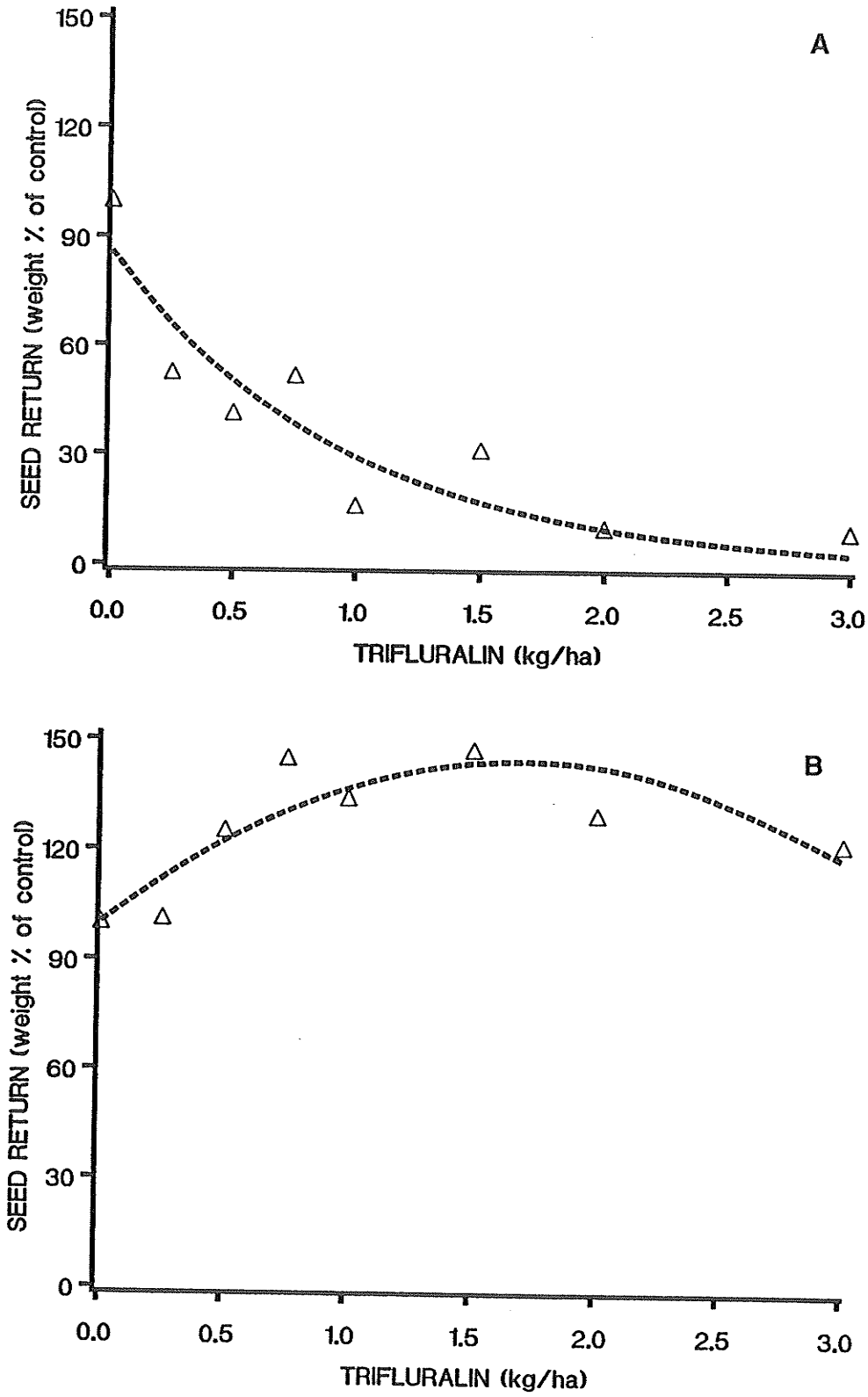

Figure 3-13. The effect of increasing dosages of PPI trifluralin on R green foxtail seed production under cropped (A) and non-cropped (B) conditions at Deloraine in 1989. See Table 3-6 for equations and parameter estimates.

seed return exceeded that of the control plots. The differences in seed yield response in the cropped versus non-cropped plots emphasizes the importance of crop competition in reducing foxtail vigor. The ED_{50} for seed production in cropped plots was similar to that determined for density and biomass, indicating that the expression of resistance was maintained over the the growing season, similar to that observed at Portage la Prairie. The effective kill at the recommended dosage of trifluralin in rapeseed was 38% (seed weight per unit area) (Table 3-8).

The initial reductions in density and biomass, as well as the effective kill of R green foxtail under cropped conditions (Table 3-8) coincide closely with the results from the PPI trifluralin experiment at Portage la Prairie (Table 3-4). This is an important finding since there are noticeable differences in climate, soils (i.e. organic matter content) and nature of green foxtail infestation (natural versus sown) between the two sites. Consequently, differences in the growth and competitiveness of both green foxtail and rapeseed, as well as trifluralin dissipation and activity in the soil, could be expected. The similar values for effective kill of R green foxtail at Deloraine (38%) and Portage la Prairie (42%) provides greater confidence in the usefulness of these values for computing the selection pressure of trifluralin on foxtail.

3.3.2. PEI trifluralin

Where trifluralin was applied as a PEI treatment, the differences between R and S biotypes were greater than where the chemical was applied as a PPI treatment. Four wk after foxtail emergence in plots sown to wheat, the R biotype was 12 times more resistant to trifluralin than the S biotype based on either density or dry matter determinations (Figure 3-14, Table 3-9). The difference in effect between PPI and PEI treatments appears primarily to result from the latter

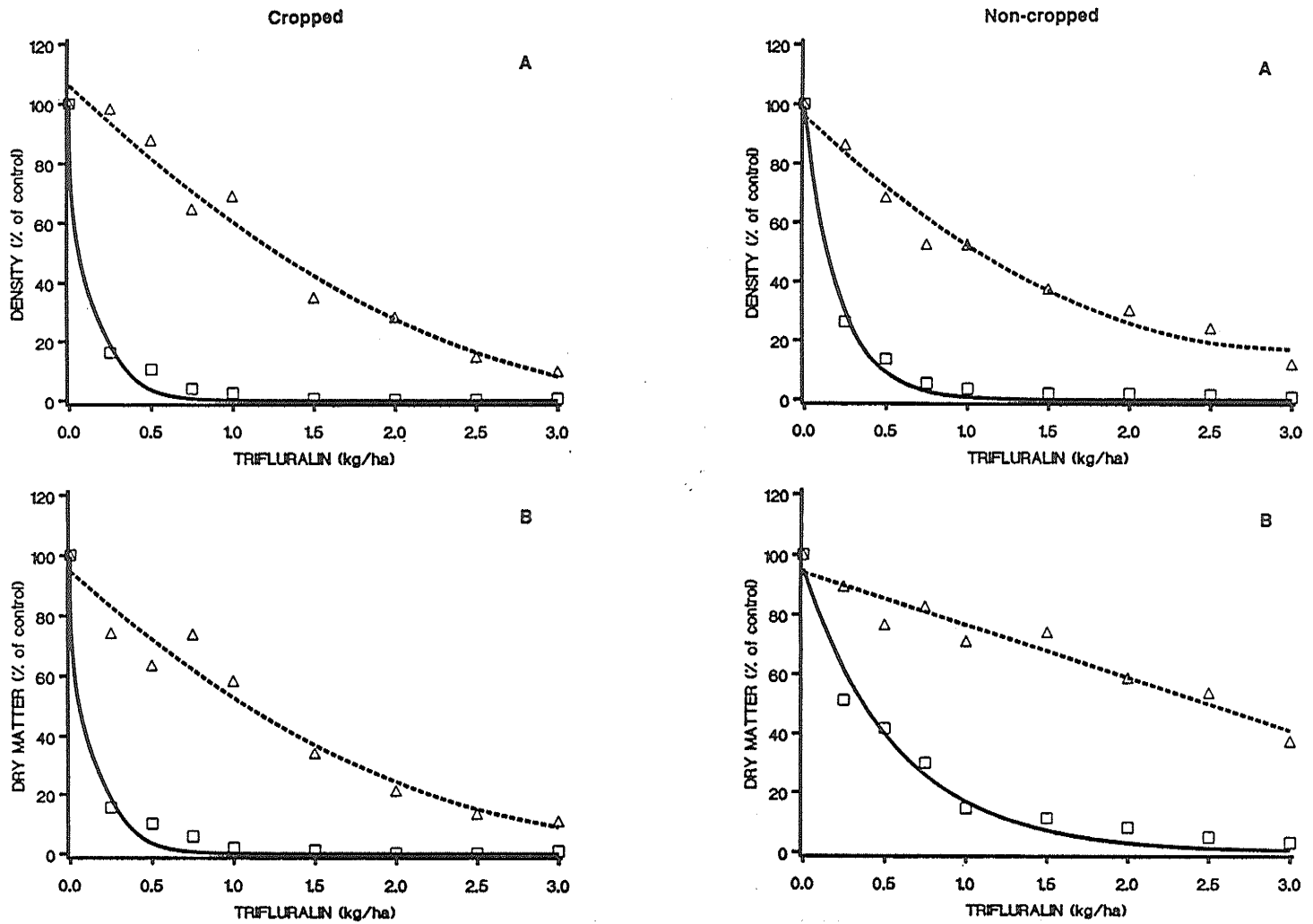

Figure 3-14. The effect of increasing dosages of PEI trifluralin on R (dashed line) and S (solid line) green foxtail density (A) and dry matter production (B) 4 wk after emergence under cropped and non-cropped conditions at Portage la Prairie in 1989 and 1990. See Table 3-9 for equations and parameter estimates.

Table 3-9. Parameter estimates (standard errors in parentheses) and ED_{50}'s of the equations for the regression curves for the response of S and R green foxtail to PEI trifluralin under cropped and non-cropped conditions at Portage la Prairie in 1989 and 1990.

Plant variable[a]	a[b]	b	c	R^{2c}	ED_{50}	R/S
4 wk after emergence						
Cropped conditions						
S density	99.7(1.5)	-6.7(0.3)		0.98**	0.1	
R density	106.3(6.4)	-52.4(10.9)	6.6(3.6)	0.66**	1.2	12
S dry wt	99.7(2.2)	-6.7(0.4)		0.96**	0.1	
R dry wt	94.7(4.6)	-48.4(6.6)	6.6(2.3)	0.71**	1.2	12
Non-cropped conditions						
S density	99.4(2.1)	-4.8(0.2)		0.96**	0.1	
R density	96.1(4.3)	-52.6(7.3)	8.7(2.4)	0.75**	1.1	11
S dry wt	95.1(5.9)	-1.7(0.2)		0.73**	0.4	
R dry wt	94.0(6.9)	-17.6(12.1)	-0.1(3.9)	0.31**	2.7	7
Maturity						
Cropped conditions						
S density	99.9(1.2)	-4.9(0.2)		0.99**	0.1	
R density	105.3(4.4)	-72.1(7.5)	14.0(2.2)	0.73**	0.9	9
S dry wt	99.4(3.2)	-4.3(0.3)		0.92**	0.2	
R dry wt	105.3(7.1)	-45.7(12.3)	6.1(4.0)	0.52**	1.4	7
S seed wt	99.2(3.8)	-3.9(0.3)		0.89**	0.2	
R seed wt	96.5(11.0)	-9.8(18.8)	-2.5(6.1)	0.16**	2.8	14
Non-cropped conditions						
S density	99.2(1.9)	-4.5(0.2)		0.97**	0.2	
R density	95.3(4.6)	-60.4(8.0)	11.8(2.6)	0.70**	1.0	5
S dry wt	87.1(4.9)	-0.4(0.1)		0.47**	1.7	
R dry wt	94.8(7.1)	-14.7(12.3)	2.7(4.0)	0.06*	>3.0	
S seed wt	96.1(8.1)	-0.4(0.1)		0.26**	2.0	
R seed wt	94.6(9.6)	4.4(16.8)	-1.1(5.5)	0.01	>3.0	

[a]Mean values ± standard error for plant variables (per 1-m^2 basis, wt in g) in control plots: **4 wk, cropped conditions** Density: S 2 740(340), R 2 850 (230); Dry wt: S 57(10), R 64(10); **4 wk, non-cropped conditions** Density: S 3 950(310), R 4 570(440); Dry wt: S 229(28), R 264(25); **Maturity, cropped conditions** Density: S 2 080(280), R 2 250(270); Dry wt: S 116(20), R 115(21); Seed wt: S 22(5), R 16(4); **Maturity, non-cropped conditions** Density: S 4 030(240), R 4 100(370); Dry wt: S 811(81), R 810(68); Seed wt: S 264(28), R 242(17).

[b]Exponential function equation: $y = a\,e^{bx}$ where a = intercept (% of control) and ab = initial slope; quadratic function equation: $y = a + bx + cx^2$ where a = intercept (% of control), b = linear coefficient, and c = curvilinear coefficient; y is the plant variable (% of control) and x is the trifluralin dosage (kg ha^{-1}).

[c]Coefficient of determination: significant at the 5% level (*), 1% level (**).

having a greater effect on S foxtail than the former. That is, ED_{50} values for S green foxtail (for density and dry matter determinations) are reduced to a greater extent than for R foxtail, when trifluralin is applied as a PEI treatment in wheat. Neither wheat density nor shoot biomass were affected by trifluralin (Figure 3-15, Table 3-10).

Similar to cropped conditions, R foxtail was about 12 times more resistant to trifluralin than S plants in non-cropped plots, based on density determinations 4 wk after emergence (Figure 3-14, Table 3-9). However, the R biotype was only 7 times more resistant to trifluralin than the S biotype on the basis of biomass determinations. Smaller differences in herbicide tolerance between R and S biotypes for biomass versus density were attributed mainly to enhanced growth of S plants due to less interplant competition at higher dosages. No differences in density or dry matter between S and R foxtail occurred in either untreated cropped or non-cropped plots.

The response of S and R green foxtail dry matter to PPI- and PEI- trifluralin closely parallels the results reported for pot experiments conducted in the growth chamber (Morrison et al. 1989) (see section 3-1). Although climate and soil factors are known to influence the effectiveness of the herbicide in controlling green foxtail, as well as the growth and competitiveness of both foxtail and the crop, the relative sensitivity of S and R biotypes to trifluralin was very similar in both studies. As in the growth chamber experiments, the method of trifluralin placement, which affects the spatial herbicide concentration in the soil, altered the expression of resistance.

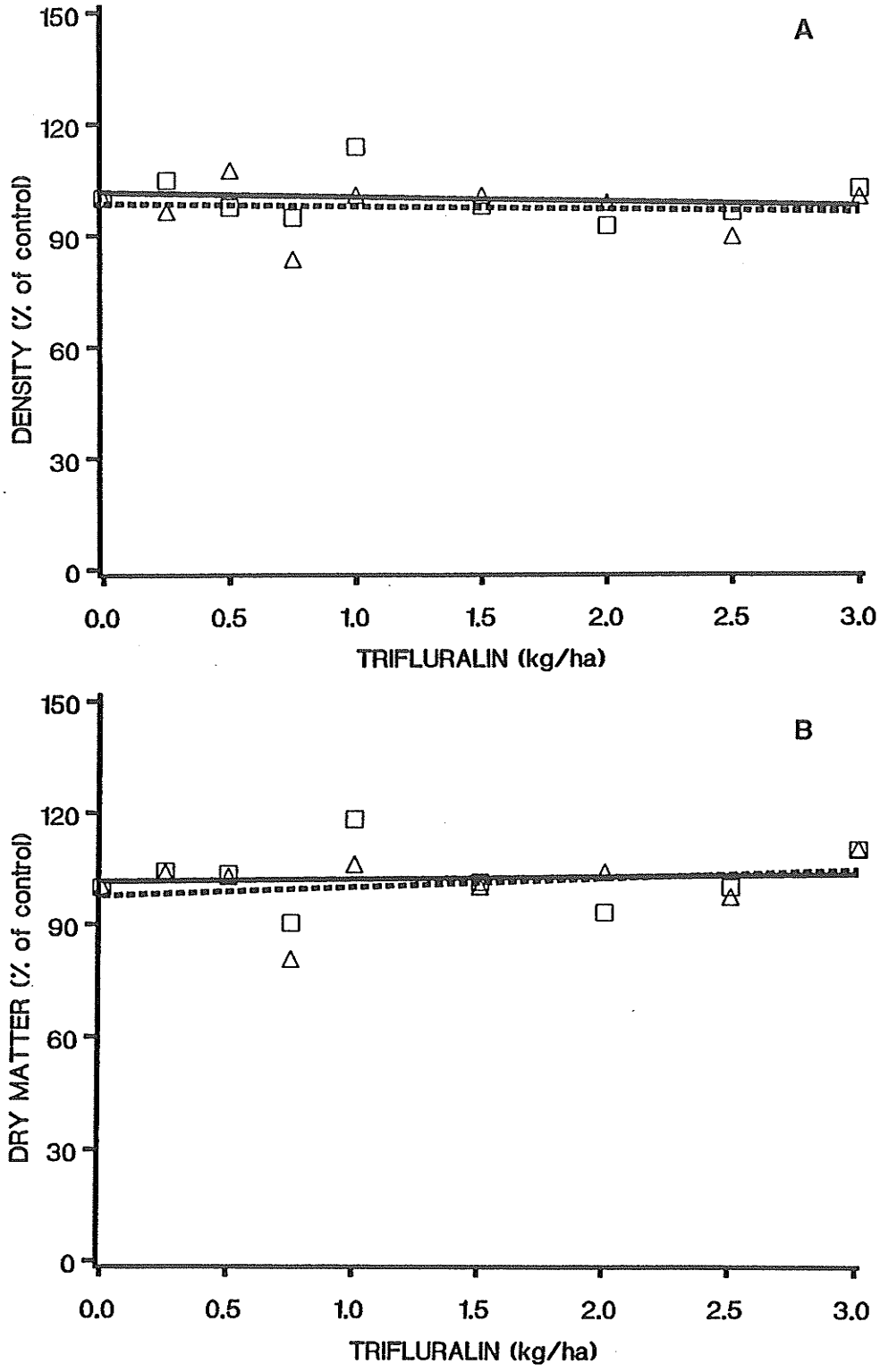

Figure 3-15. The effect of increasing dosages of trifluralin applied as a PEI treatment on wheat density (A) and dry matter production (B) 4 wk after foxtail emergence, in plots sown to R (dashed line) and S (solid line) biotypes at Portage la Prairie in 1989 and 1990. See Table 3-10 for equations and parameter estimates.

Table 3-10. Parameter estimates (standard errors in parentheses) of the equations for the regression curves for the response of wheat to PEI trifluralin at Portage la Prairie in 1989 and 1990.

Crop variable	a	b	R^{2a}
4 wk after emergence [b]			
S density[c]	101.5 (3.5)	-1.4 (2.2)	0.01
R density	98.5 (3.0)	-1.0 (1.9)	0.01
S dry wt	101.5 (3.4)	1.1 (2.1)	0.01
R dry wt	97.7 (3.7)	2.7 (2.3)	0.02
Maturity			
S density	104.3 (3.4)	-4.1 (2.1)	0.05
R density	98.1 (3.4)	-4.1 (2.1)	0.05
S dry wt	102.0 (3.0)	0.4 (1.9)	0.01
R dry wt	91.5 (3.1)	3.5 (1.9)	0.04
S seed wt	104.6 (3.0)	-0.3 (1.9)	0.01
R seed wt	105.0 (2.8)	3.1 (1.8)	0.04

[a]Coefficients of determination are not significant at the 5% level.

[b]Stem elongation to booting crop development stages.

[c]In control plots, mean values ± standard errors (per 1 m^2, wt in g) for select variables were: **4 wk** Density: S 274(25), R 269 (11); **Maturity** Seed wt: S 398(46), R 373(44).

By foxtail maturity under cropped conditions, the differences in sensitivity between the S and R biotypes were not as evident as earlier in the season (Figure 3-16). Wheat density, biomass, and yield were similar in plots sown to either S or R green foxtail and no change in any of these variables occurred as trifluralin dosages increased (Figure 3-17). The fact that neither wheat nor rapeseed yield were affected by green foxtail in plots treated with increasing trifluralin dosages may be due primarily to the relatively rapid crop stand establishment because of favorable climatic conditions. In other studies, trifluralin at dosages higher than 0.25 kg ha^{-1} caused significant reductions in wheat yields (Morrison et al. 1991).

Differences in response between the biotypes to trifluralin in the non-

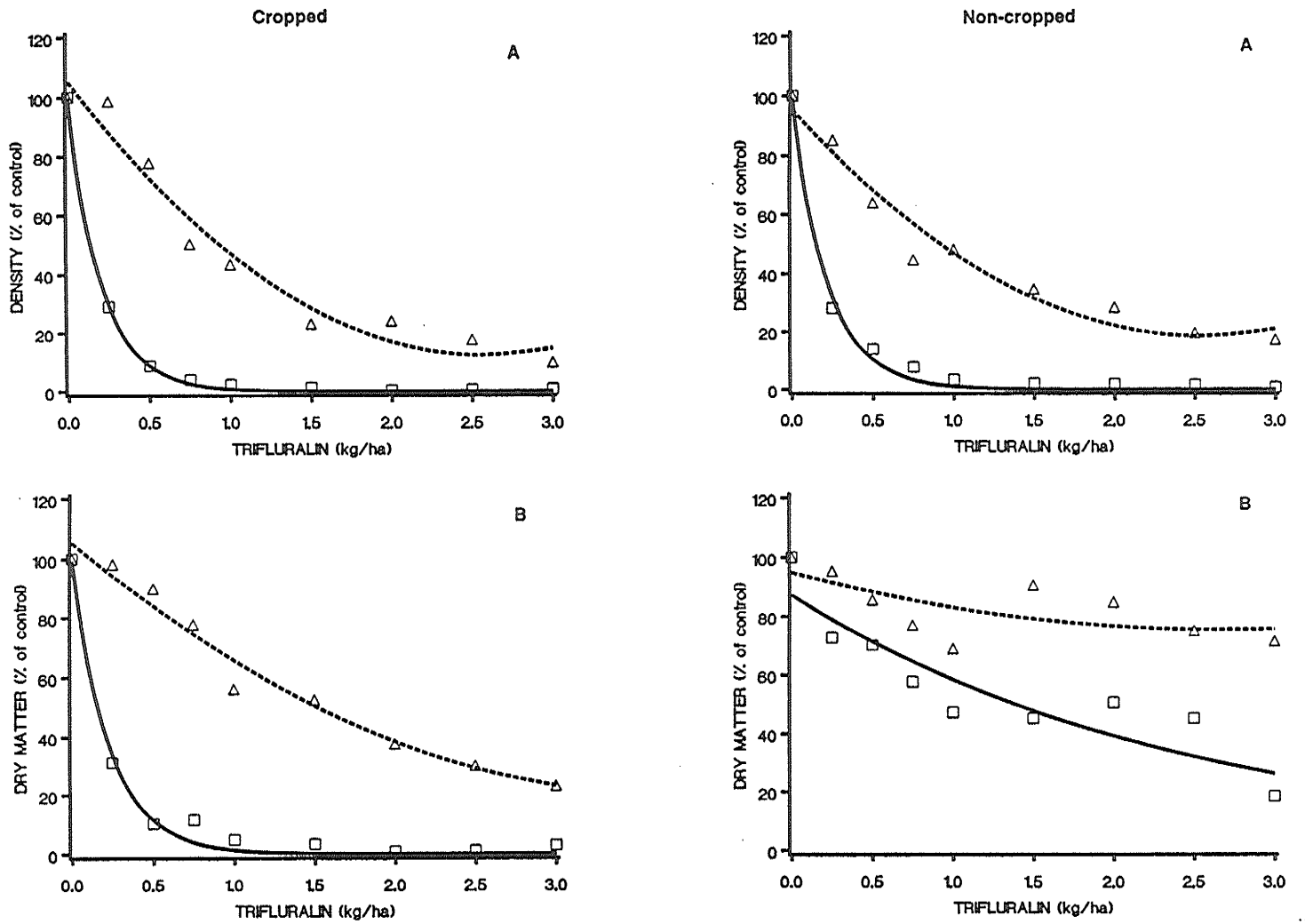

Figure 3-16. The effect of increasing dosages of PEI trifluralin on R (dashed line) and S (solid line) green foxtail density (A) and dry matter production (B) at maturity under cropped and non-cropped conditions at Portage la Prairie in 1989 and 1990. See Table 3-9 for equations and parameter estimates.

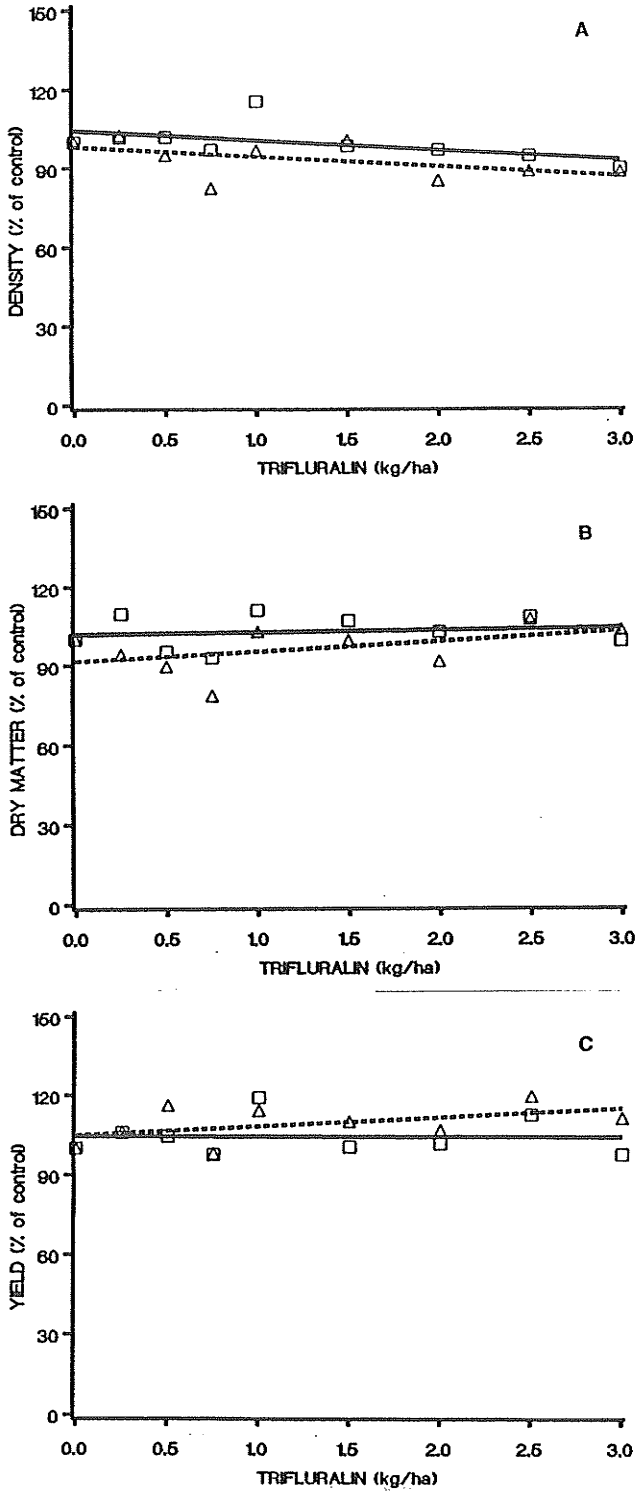

Figure 3-17. The effect of increasing dosages of trifluralin applied as a PEI treatment on wheat density (A), dry matter production (B), and yield (C) at maturity, in plots sown to R (dashed line) and S (solid line) biotypes at Portage la Prairie in 1989 and 1990. See Table 3-10 for equations and parameter estimates.

cropped plots (Figure 3-16) were less than those reported for the cropped plots. Enhanced tillering of S foxtail at higher dosages reduced the biomass differences between the biotypes (Figure 3-18).

The results of the PEI study are similar to those reported for the PPI experiment in that there was a greater level of resistance of the R biotype (14-fold) under cropped conditions on the basis of seed return (Figure 3-19, Appendix Figure 5, and Appendix Table 3), than measured from density or biomass. Therefore, the expression of resistance in R green foxtail to trifluralin under field crop conditions did not decline over the growing season. Under

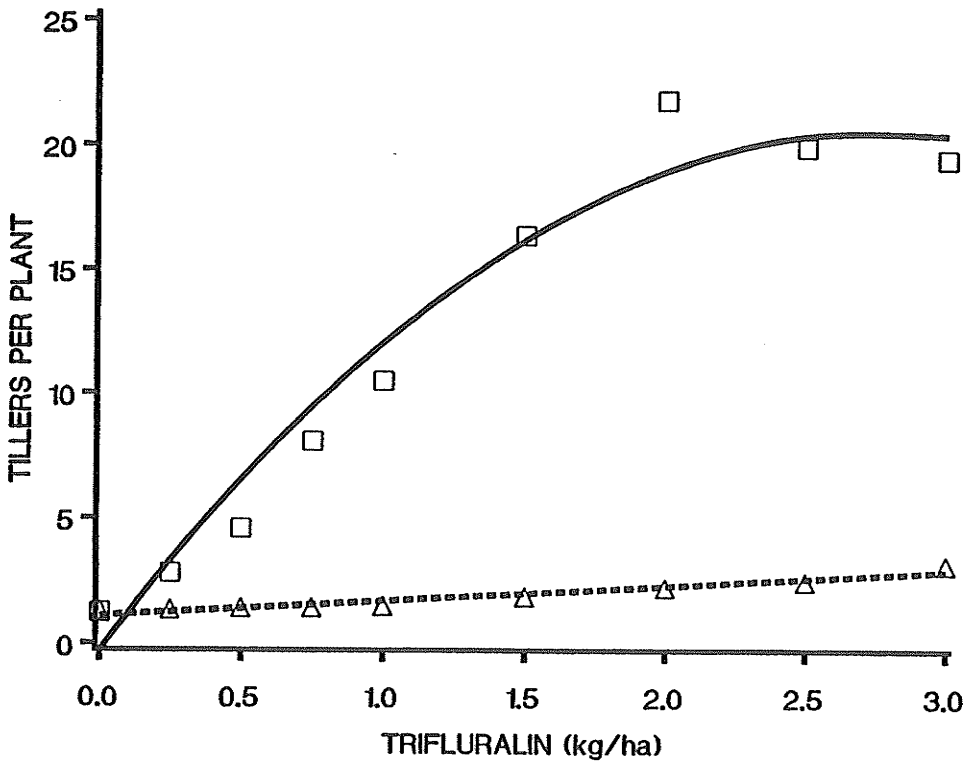

Figure 3-18. The effect of increasing dosages of PEI trifluralin on R (dashed line) and S (solid line) green foxtail tillers per plant at maturity under non-cropped conditions at Portage la Prairie in 1989 and 1990. R^2 for R and S biotypes = 0.44** and 0.37**, respectively.

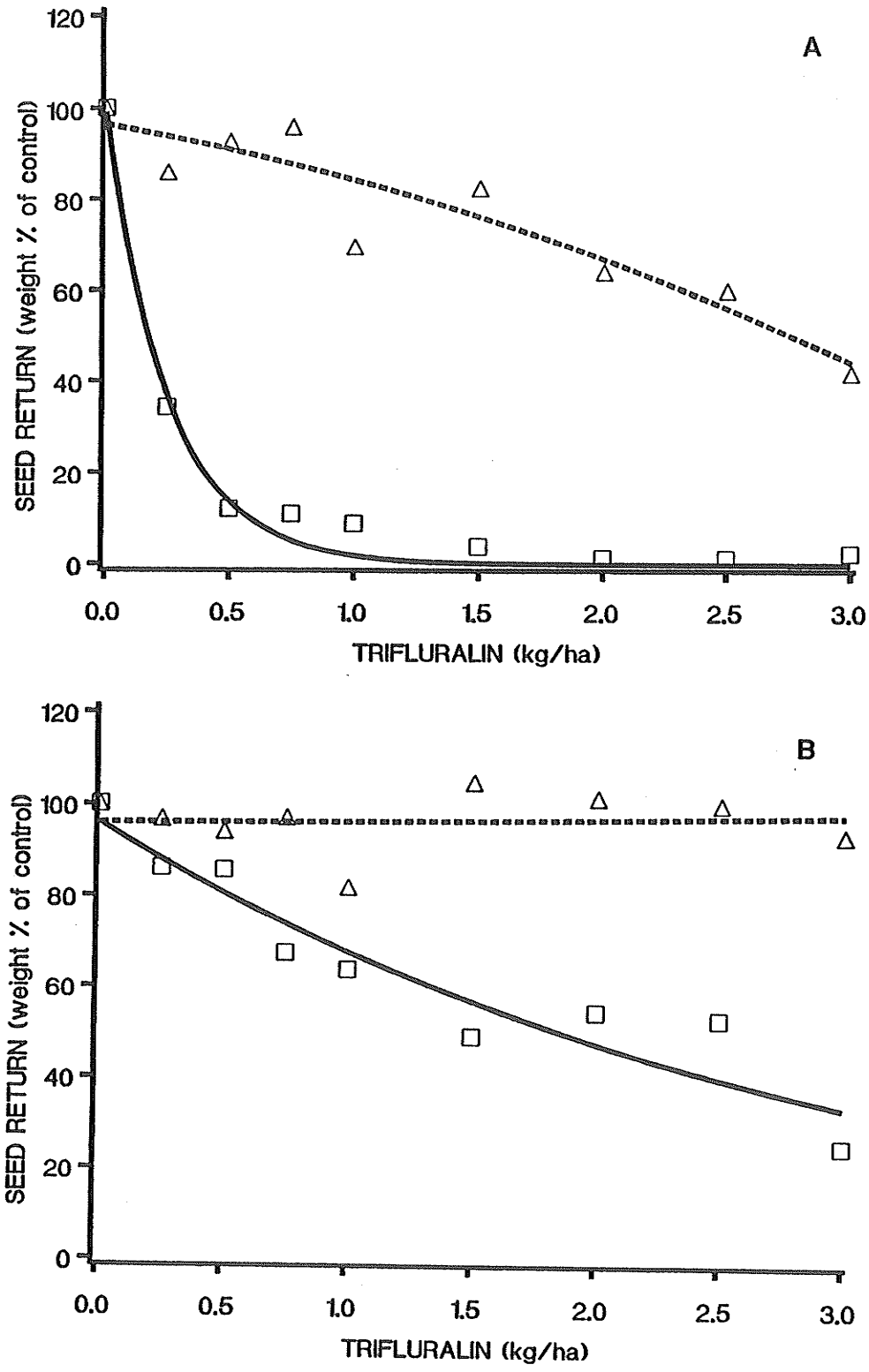

Figure 3-19. The effect of increasing dosages of PEI trifluralin on R (dashed line) and S (solid line) green foxtail seed production under cropped (A) and non-cropped (B) conditions at Portage la Prairie in 1989 and 1990. See Table 3-9 for equations and parameter estimates.

non-cropped conditions, a comparison of the response of R and S foxtail seed return to increasing dosages of trifluralin was not possible because seed production of the R biotype was not reduced by the chemical. S and R seed return were similar in either untreated cropped or non-cropped plots, as reported for the PPI experiment.

At the recommended trifluralin dosage in wheat (0.9 kg ha^{-1}), the density and biomass of S plants were reduced by over 99%, whereas that of R plants was reduced by 36% and 44%, respectively (Table 3-11). Seed return (weight per unit area) of S and R biotypes were reduced by 97% and 14%, respectively. Therefore in this experiment the effective kill was less than the initial reductions in density or biomass. In contrast, the effective kill of R and S foxtail determined from the PPI experiment (99% and 42%, respectively) was greater than their initial reductions in density or biomass. Nevertheless, the results from all three

Table 3-11. Percent reduction in S and R green foxtail variables in response to PEI trifluralin at the recommended dosage in wheat (0.9 kg ha^{-1}) at Portage la Prairie in 1989 and 1990.

	Cropped		Non-cropped	
	S	R	S	R
	% reduction			
4 wk after emergence				
Density	99	36	99	44
Dry wt	99	44	80	22
Maturity				
Density	99	49	98	49
Dry wt	98	31	39	16
Seed wt	97	14	30	2
Seed no	97	15	27	2

experiments clearly indicate that the effective kill is not markedly lower than (and can even exceed) the initial weed control. This is in contrast to what would be expected with less persistent herbicides.

The selection pressure of PEI trifluralin on green foxtail under cropped conditions was: (1-0.14)/(1-0.97) = (0.86/0.03) = 29. In contrast, the selection pressure of PPI trifluralin on foxtail was twice as great (58), attributed largely to the greater effective kill of S foxtail. Therefore the rate of enrichment of trifluralin-resistant biotypes may be greater when trifluralin is applied as a PPI treatment in rapeseed than when trifluralin is applied as a PEI treatment in wheat.

Even though the expression of resistance (R/S ratio) in R green foxtail is greater when trifluralin is applied as a PEI treatment in wheat than as a PPI treatment in rapeseed, the selection pressure was lower in the former than the latter. Thus, the level of resistance can not be used as an indicator of the rate of evolution of R foxtail populations under selection pressure by trifluralin. Rather, the effective kill must be known to more fully understand the evolution and population dynamics of R foxtail populations.

3.4. Modelling the Rate of Enrichment of R Green Foxtail

The rate of enrichment of R green foxtail was modelled using values for the selection pressure, computed from the effective kill of R and S green foxtail reported in the preceding section. These values were inserted into two population models which were developed by Gressel and Segel (1978, 1990a, b) (see Chapter 2 [p. 9-14] for model descriptions). The original model (Model I) described the rate of enrichment of R biotypes under monoculture, monoherbicide usage, whereas the modified model (Model II) accounts for the effect of herbicide rotations on delaying or precluding the appearance of

resistance.

Different input values for the remaining model parameters were substituted into the model equations to represent various scenarios occurring in the field. The initial frequency of R individuals, N_o, derived from natural mutations in the S population prior to any herbicide exposure, was set at 10^{-6} (for a dominant monogenic trait) or 10^{-12} (for a recessive monogenic trait) (Gressel and Segel 1990a). However, these are only crude estimates because of the lack of information. It is not clear that one need actually consider whether mutations to resistance are dominant or recessive as there may be only a small frequency difference between the two types in diploid organisms; recombination can increase homozygous recessive frequencies in populations considerably (Williams 1976). Regardless, N_o will not affect the rate of enrichment of resistance, only the starting point for resistance.

The predicted exponential rates of enrichment of R green foxtail by PPI- and PEI-trifluralin are illustrated in Figures 3-20 and 3-21, respectively, using Model I, with different scenarios derived from different input values of N_o and fitness, f. It is assumed that there is no carryover of trifluralin residues from one year to the next. The scale on the left indicates the proportion of R individuals in the population, starting from a theoretically expected frequency of a dominant or recessive monogene. The scale on the right indicates the increase in resistance from any unknown initial frequency of resistants in the population (enrichment factor). It is assumed that the fitness of R green foxtail biotypes is either slightly less than ($f = 0.8$) or the same ($f = 1.0$) as that of S biotypes[8]. The R and S biotypes used in the field experiments had similar productivity, which may indicate a negligible difference in fitness between these two biotypes. The

[8]Heap, I. 1991. Personal conversation. Univ. Manitoba, Winnipeg, MB.

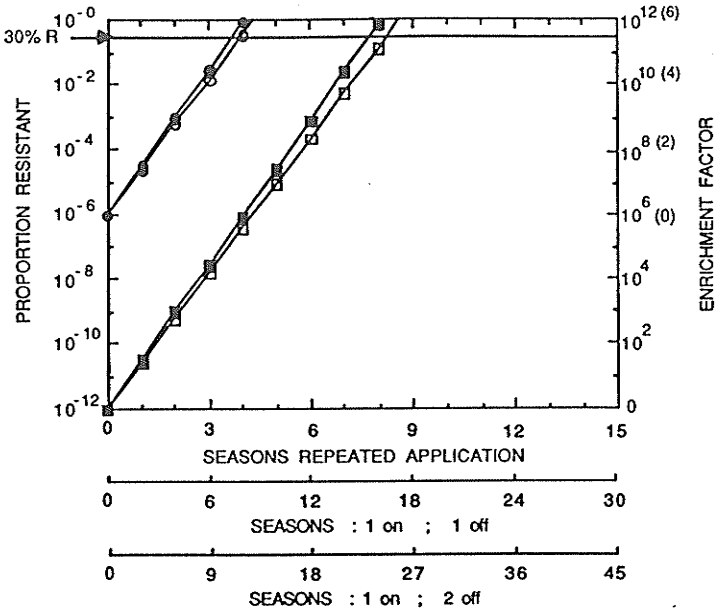

Figure 3-20. The predicted rate of evolution of R green foxtail when trifluralin is applied as a PPI treatment in rapeseed at the recommended dosage (Model I). The initial frequency, N_o, = 10^{-6} (dominant monogene trait) or 10^{-12} (recessive monogene trait; the fitness differential, *f*, between the R and S green foxtail biotypes is either 0.8 (open symbols) or 1.0 (closed symbols); the seed bank longevity, ß, is 2 yr. The selection pressure, å, =58 (see section 3.3.1).

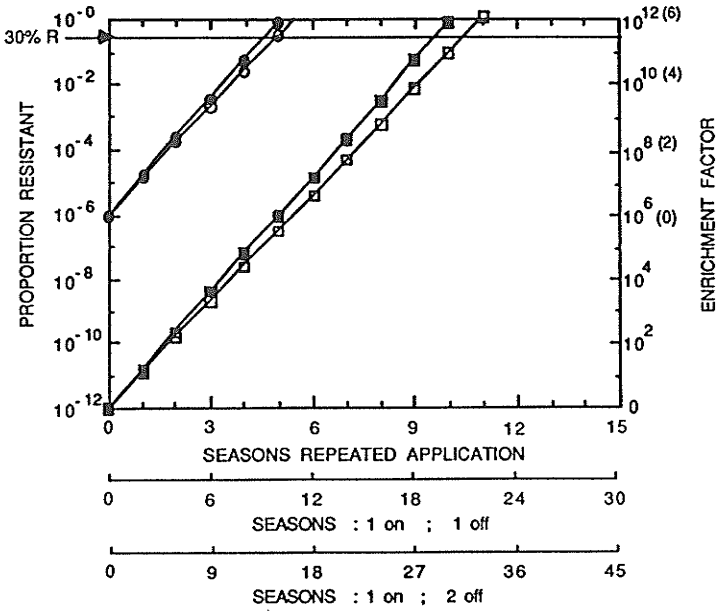

Figure 3-21. The predicted rate of evolution of R green foxtail when trifluralin is applied as a PEI treatment in wheat at the recommended dosage (Model I). The selection pressure, å, =29 (see section 3.3.2). Other model parameters are listed in Figure 3-20.

average number of years that green foxtail remains viable in the seed bank, ß, was set at 2 yr, based on data reported by Banting et al. (1973) and Thomas et al. (1986) on seed longevity.

The model indicates that R green foxtail would become evident in rapeseed treated annually with PPI trifluralin after 4 (N_o =10^{-6}) or 8 yr (N_o = 10^{-12}). R biotypes are usually not noticeable until they comprise 10 to 30% of the population (Gressel and Segel 1982). Producers will not detect R green foxtail, therefore, until it is a serious problem. The time it would take for R biotypes to become evident in wheat fields treated annually with PEI trifluralin would be slightly longer, due to the lower selection pressure. The nearly parallel slopes of the lines of similar fitness confirm the fact that the initial frequency only influences the starting level of resistance and not the rate of enrichment. If a different initial field frequency is chosen, the frequency scale can be adjusted, or else the right-hand scale can be used (Gressel and Segel 1982).

It is clear that at these very high selection pressures, there is little difference in the rate of enrichment of resistance if the R biotype has near-normal (f = 0.8) or normal fitness (f = 1.0) as compared to S biotypes. Differential fitness between the biotypes would be relatively more important in dampening the rate of enrichment of resistance if the selection pressure was lower, such as might occur with less persistent herbicides. This is because the reduced fitness of the R biotype can only be expressed after the herbicide is inactive. If trifluralin is alternated with another herbicide that controls R and S green foxtail equally (assuming no carryover), the model predicts that resistance would be delayed by a factor of two. Similarly, if trifluralin was employed every third season, resistance would theoretically be delayed by a factor of three (Gressel and Segel 1982). Departure from model

assumptions (since green foxtail has a very low degree of outcrossing) may reduce the accuracy of the simulations because the models are based on the Hardy-Weinberg law of population genetics.

Model II was used to more accurately simulate the rate of development of resistance when trifluralin is not used in certain seasons. The fitness of R green foxtail in the 'off' season when trifluralin is not used is either 0.8 or 1.0; f_{on} is generally assumed to be 1.0 (Gressel and Segal 1990a, b). In contrast, Model I used an average fitness differential for all generations treated. The fraction of seeds leaving the seed bank each year, ∂, is equivalent to β^{-1} (Gressel and Segel 1990b), which is equal to 0.5.

The enrichment of R individuals in the population (enrichment factor) when trifluralin is applied as a PPI treatment in rapeseed (Figure 3-22) or as a PEI treatment in wheat (Figure 3-23) is markedly influenced by the specific herbicide rotation. When trifluralin is not used over a season (no selection pressure) the rate of disappearance of resistance is due only to decreased fitness of the R biotype. Thus, if trifluralin is used once every 4 yr (1 on:3 off), there will be a slight decline in the enrichment factor (proportion of resistant individuals) if the R biotype is 80% as fit as the S biotype. However, this rate of decline is much less than the rate of increase obtained when trifluralin is present. There will be no decline in the enrichment factor if there is no fitness differential between the biotypes. In any event, there is little difference in the rate of enrichment of resistance at f = 0.8 or 1.0, due to the relatively high selection pressure (similar to Model I). In fact, if the curves derived from Model II are smoothed, the results are very similar to the lines generated from Model 1 for corresponding scenarios, due to the high selection pressure coupled with near-normal to normal fitness of R green foxtail. Model I, however, will not adequately account for events in the 'off' years during herbicide rotations if the

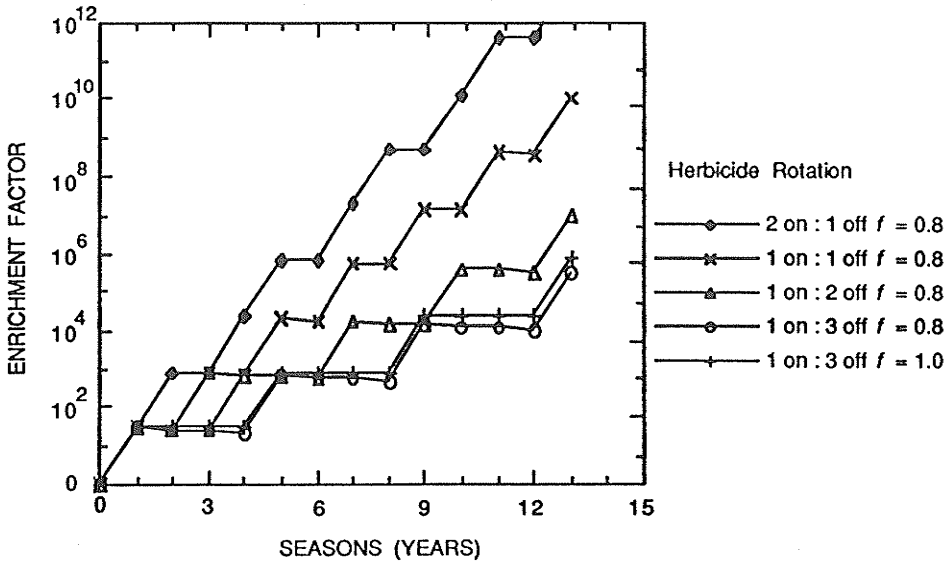

Figure 3-22. The predicted rate of evolution of R green foxtail when trifluralin is applied as a PPI treatment in rapeseed at the recommended dosage (Model II). The fraction of seeds leaving the seed bank each year, ∂, =0.5; the selection pressure, å,= 58; the fitness differential between the R and S biotypes in the absence of trifluralin, f_{off}, = 0.8 or 1.0; f_{on} = 1.0.

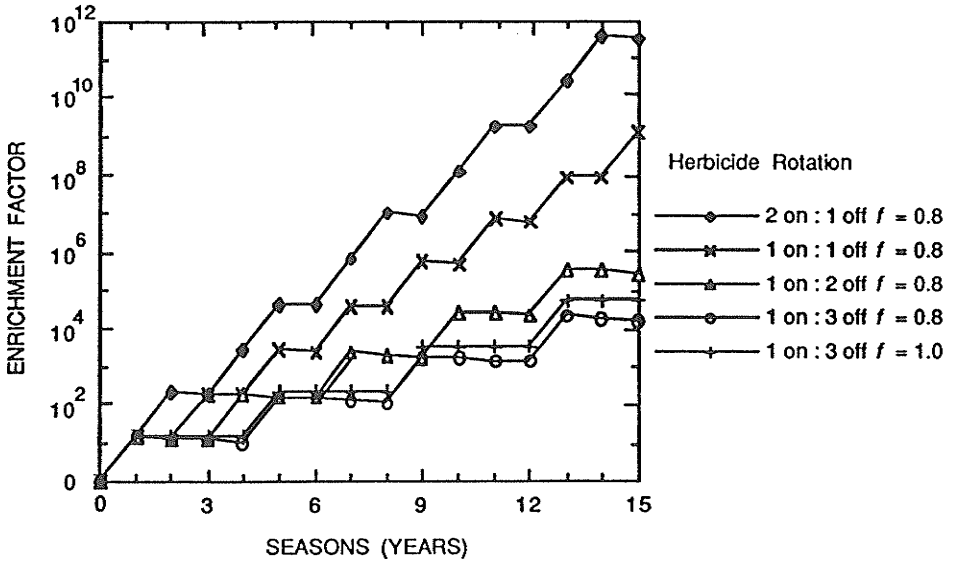

Figure 3-23. The predicted rate of evolution of R green foxtail when trifluralin is applied as a PEI treatment in wheat at the recommended dosage (Model II). The selection pressure, å, = 29. Other model parameters are listed in Figure 3-22.

fitness of R weed biotypes is low, such as with triazine resistance (Gressel and Segel 1990a). Therefore, rotations (or mixtures) may not truly delay the rate of appearance of R green foxtail, where the fitness of the R biotype is near-normal. The only delay will be for the number of years that trifluralin is not used. The only alternative is to lower the selection pressure by using this persistent herbicide less frequently in the cropping rotation.

R green foxtail has been shown to exhibit negative cross resistance to two herbicidal compounds (Smeda et al. 1991), although they are not registered in western Canada. These herbicides are propham [1-methylethyl-phenylcarbamate] and chlorpropham [1-methylethyl 3-chlorophenylcarbamate]. Identification and inclusion of herbicides to which R green foxtail shows such cross resistance could further delay the evolution of R biotypes.

The results of these models emphasize the importance of obtaining meaningful data, such as the selection pressure, to insert into the model equations, in order to more accurately predict the evolution and population dynamics of R green foxtail. This would allow specific herbicide rotation programs to be implemented that would be designed to delay resistance for a minimum specified period if trifluralin was to be used periodically in the cropping system. The model results would thus estimate the useful lifetime of the herbicide for controlling green foxtail. Since trifluralin exerts a very high selection pressure, which is the most influential agronomic variable having the largest effect on the evolution of resistance, and given the presumed small fitness differential between S and R biotypes, this herbicide must be used less frequently in the rotation to delay the appearance of trifluralin-resistant green foxtail.

Verification of these model results can only be achieved from knowledge of the field histories of trifluralin usage. Complete documentation is required from

the time that trifluralin was first applied on the particular field, since these models become less accurate when the R biotype comprises a large percentage of the population (Gressel and Segel 1990b). In addition, current research on the relative fitness of R and S biotypes and the mode of inheritance of resistance will provide more accurate input model parameters for better estimating the rate of evolution of R green foxtail.

3.4.1. A case study

In order to run the model using actual crop/herbicide rotations, a letter was sent to approximately 40 producers, who had fields with populations of R foxtail, requesting information on the cropping history and herbicide usage. Of ten replies received, only one had detailed information on the complete field history of trifluralin usage (Table 3-12). In this field, trifluralin was first applied in 1974 and R foxtail was first observed in the spring of 1988.

The rate of enrichment of R foxtail is plotted in Figure 3-24, using the same values for the model II parameters as described previously. The enrichment factor was approximately 1 X 10^6 in 1987 and increased to almost 1 X 10^8 in 1988. If resistance is controlled by a single dominant gene, and if the estimates of *No* (10^{-6}) and *f* (0.8 to 1.0) are correct, then the model results indicate that R foxtail would be observed in 1987 or 1988. This would coincide with the time that resistance was first noticed in the field.

This single simulation does not validate the model, but emphasizes its usefulness in formulating preventative management strategies to regulate the enrichment of R foxtail and thus considerably delay the appearance of this biotype as well as extend the product's effectiveness and market lifetime. This can be achieved mainly by altering the selection pressure, by changing either the frequency that the product is used in the rotation or the dosage of

Table 3-12. Cropping history and trifluralin usage associated with the occurrence of R green foxtail at Snowflake, Manitoba.

Year	Crop	Trifluralin dosage[1]
		kg ha^{-1}
1974	Rapeseed	0.8
1975	Wheat	
1976	Wheat	
1977	Wheat	
1978	Rapeseed	0.8
1979	Wheat	
1980	Rapeseed	0.8
1981	Wheat	
1982	Flax (*Linum usitatissimum* L.)	
1983	Rapeseed	0.8
1984	Rye (*Secale cereale* L.)	
1985	Flax	
1986	Wheat	
1987	Wheat	0.3 (half dosage)
1988	Rapeseed	0.8

[1]Ethalfluralin [*N*-ethyl-*N*-(2-methyl-2-propenyl)-2, 6-dinitro-4-(trifluoromethyl)benzenamine] has never been applied on this field.

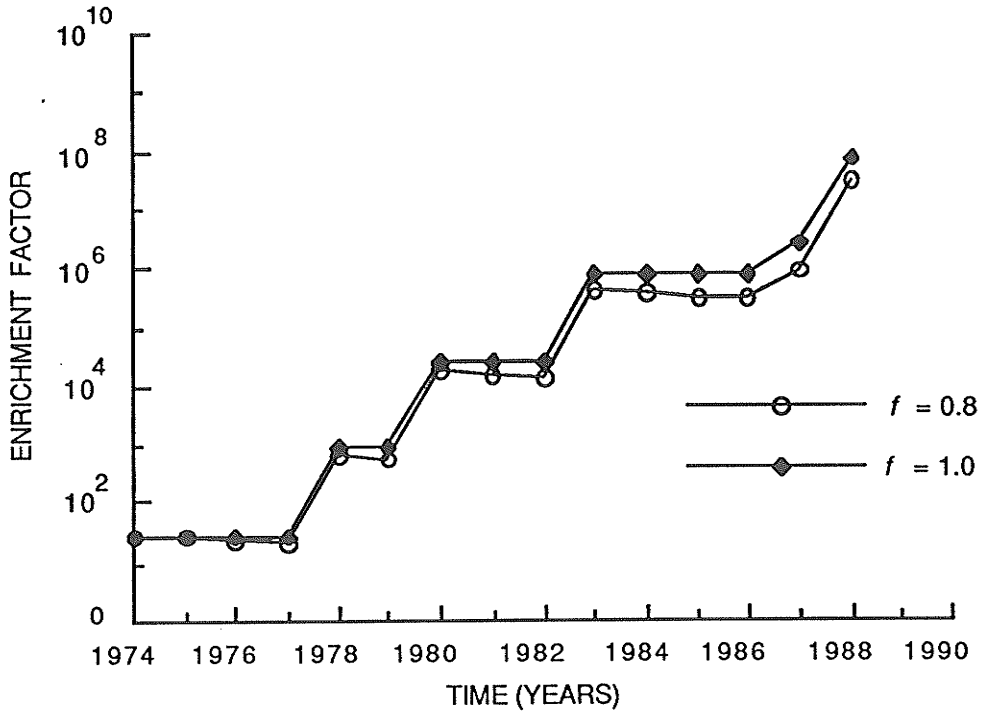

Figure 3-24. The simulated rate of enrichment of R foxtail in a field near Snowflake, Manitoba (see Figures 3-22 and 3-23 for model parameters).

application. However, this must be accompanied by agronomic practices that promote vigorous crop growth, such as proper fertilization and good seed bed preparation. The model results also emphasize the need for more research to quantify the factors that affect the evolution of R biotypes. In addition, producers must keep detailed records of herbicide usage on their fields so that model simulations can be performed using actual crop/herbicide rotations. Only then can these models be adequately validated.

4. CROSS-RESISTANCE PATTERN OF TRIFLURALIN-RESISTANT GREEN FOXTAIL (*Setaria viridis*)

Abstract. The response of susceptible (S) and resistant (R) green foxtail biotypes to herbicides belonging to several chemical groups was compared to determine the cross-resistance pattern of the R biotype. Dose-response experiments conducted in the growth chamber indicated that trifluralin-resistant green foxtail was cross-resistant to other dinitroaniline herbicides. Based on differences in shoot dry matter accumulation, differences between R and S plants were as follows: trifluralin 7-fold, ethalfluralin 6, oryzalin 6, benefin 4, isopropalin 4, pendimethalin 4, and prodiamine 4. The R biotype was twice as resistant as the S biotype to dithiopyr, a chemically unrelated mitotic disrupter herbicide. There were no differences in response between the biotypes to nine other herbicides belonging to seven chemical families. The response of S and R green foxtail to increasing dosages of ethalfluralin, applied as a preplant incorporated (PPI) treatment in rapeseed, was investigated in a field experiment located at Portage la Prairie in 1989 and 1990. Additionally, the response of these biotypes was compared under non-cropped conditions in the same experiment. Under both cropped and non-cropped conditions, the R biotype was about 7 times more resistant to ethalfluralin than the R biotype based on density determinations 4 wk after emergence. Under cropped conditions, 7 times higher dosage was required to reduce R seed production by 50% than to reduce S seed production by the same amount. The initial reductions in density of R and S plants at the recommended dosage of ethalfluralin in rapeseed (1.4 kg ha^{-1}) was 35% and 95%, respectively. The effective kill (seed yield reduction) of R and S biotypes was 55% and 99%, respectively. The results indicate that ethalfluralin will not effectively control R foxtail. However, there

are a number of other herbicides with different mechanisms of action that can be used to effectively control R foxtail, thereby reducing any adverse effects on crop production in western Canada. **Nomenclature:** Benefin, *N*-butyl-*N*-ethyl-2,6-dinitro-4-(trifluoromethyl)benzenamine; dithiopyr, *S,S*-dimethyl 2-(difluoromethyl)-4-(2-methylpropyl)-6-(trifluoromethyl)-3, 5-pyridinedicarbothioate; ethalfluralin, *N*-ethyl-*N*-(2-methyl-2-propenyl)-2, 6-dinitro-4-(trifluoromethyl)benzenamine; isopropalin, 4-(1-methylethyl)-2,6-dinitro-*N*,*N*-dipropylbenzenamine; oryzalin, 4-(dipropylamine)-3, 5-dinitrobenzenesulfonamide; pendimethalin, *N*-(1-ethylpropyl)-3, 4-dimethyl-2, 6-dinitrobenzenamine; prodiamine, *N*,*N*-dipropyl-2, 4-dinitro-6-(trifluoromethyl)phenylenediamine; trifluralin, 2,6-dinitro-*N*,*N*-dipropyl-4-(trifluoromethyl)benzenamine; green foxtail, *Setaria viridis* (L.) Beauv. # SETVI; rapeseed, *Brassica napus* L. 'Westar'. **Additional index words:** Herbicide resistance, *Brassica napus*, *Triticum aestivum*, SETVI.

4.1. Introduction

Green foxtail is the most abundant grass weed of annual crops grown on the Canadian prairies (Thomas and Wise 1985, 1987, 1988). The weed is effectively controlled by several herbicides including trifluralin, which is commonly used in the cropping system. However, the occurrence of trifluralin-resistant green foxtail, primarily in southwestern Manitoba, was confirmed in dose-response experiments conducted in the growth chamber (Morrison et al. 1989) and subsequently was verified in the field (Chapter 3). Under both cropped and non-cropped conditions, the R biotype was 7 times more resistant than the S biotype to PPI applications of trifluralin. The effective kill of R and S green foxtail in rapeseed at the recommended dosage of trifluralin was 42% and 99%, respectively.

R weed biotypes usually are resistant to other chemicals in the same herbicide family (Holt and LeBaron 1990), although the levels of resistance can not be accurately predicted. For example, a R biotype of kochia [*Kochia scoparia* (L.) Schrad.] is over 400 times more resistant to chlorsulfuron [2-chloro-*N*- (((4-methoxy-6-methyl-1, 3, 5-triazin-2-yl)amino)carbonyl)benzene-sulfonamide] than S plants, but only twice as resistant to another sulfonylurea herbicide, metsulfuron [2- (((((4-methoxy-6-methyl-1, 3 ,5-triazin-2-yl)amino)carbonyl)amino)sulfonyl)benzoic acid] (Saari et al. 1990). A R biotype that is resistant to a particular herbicide may exhibit related, though usually lower degrees of resistance to other herbicides having the same mechanism of action[1]. These observations form the basis of recommendations for preventing or delaying the appearance of resistance by rotating herbicides with different sites of action (Gressel and Segel 1982, 1990a). However, this strategy is compromised by weed biotypes with multiple resistance to herbicides having different mechanisms of action (Heap and Knight 1986; Moss 1987). The mechanism of resistance in these biotypes does not involve target site modifications, but rather enhanced metabolic detoxification by mixed-function oxygenases (Kemp and Casely 1987; Kemp et al. 1990; Powles and Howat 1990).

Little is known of the extent to which R green foxtail is resistant to other dinitroaniline herbicides and to herbicides belonging to other chemical families. Identification of the cross-resistance pattern will indicate alternative herbicides that control R foxtail and may provide an insight into the mechanism of resistance.

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[1]LeBaron, H. M. 1986. Resistance of weeds to herbicides. Presented to the Illinois Custom Spray Operators Training School. University of Illinois, Urbana, IL.

The first objective of the present studies was to document the levels of resistance of trifluralin-resistant green foxtail to other dinitroaniline herbicides and two unrelated mitotic disrupter herbicides. The second objective was to determine whether or not R foxtail exhibited resistance to eight other herbicides belonging to six chemical families which differ in their mechanisms of action from the dinitroanilines. Most of these herbicides are registered to control or suppress green foxtail in cereal and oilseed crops grown in Manitoba. In conjunction with these growth chamber studies, a field experiment compared the response of S and R foxtail under both cropped and non-cropped conditions to ethalfluralin, which is registered in western Canada to control various grass (including green foxtail) and broadleaf weeds when applied as a PPI treatment in rapeseed and other oilseed and specialty crops. This experiment was initiated after preliminary studies in the growth chamber had confirmed that trifluralin-resistant foxtail was cross-resistant to this dinitroaniline herbicide.

4.2. Materials and Methods

4.2.1. Growth chamber experiments

4.2.1.1. Preemergence herbicides

The herbicides which were applied preemergence included the dinitroanilines (see Figure 2-1); two chemically unrelated mitotic disrupter herbicides - pronamide (3,5-dichloro(*N*-1,1-dimethyl-2-propynyl)benzamide) and dithiopyr; and EPTC (Table 4-1). The soil was a silty clay loam (13% sand, 39% silt, 28% clay) with an organic matter content of 7.9% and a pH of 7.7. The dinitroaniline herbicides and EPTC were applied to the soil with a spray bottle at dosages equivalent to 0 to 1.2 kg ai ha^{-1}. Pronamide was applied at dosages equivalent to 0 to 1.6 kg ai ha^{-1}, and dithiopyr at 0 to 200 g ai ha^{-1}. The herbicides were incorporated into the soil in a rotary mixer for 10 min. After

Table 4-1. Herbicides used in the cross resistance studies, categorized by chemical family[a].

Family name	Common name	Chemical name
1. Amide	propanil	*N* -(3,4-dichlorophenyl)propanamide
2. Aryloxyphenoxy-propionate	diclofop methyl	methyl 2-(4-(2,4-dichlorophenoxy)phenoxy)propanoic acid
	fenoxaprop-p-ethyl	ethyl 2-(4-((6-chloro-2-benzoxazolyl)oxy)phenoxy)propanoic acid
	fluazifop-butyl	butyl 2-(4-((5-(trifluoromethyl)-2-pyridinyl)oxy)phenoxy)propanoic acid
3. Chlorinated aliphatic acid	dalapon	2,2-dichloropropanoic acid
4. Cyclohexanedione	sethoxydim	2-(1-(ethoxyimino)butyl)-5-(2-(ethylthio)propyl)-3-hydroxy-2-cyclohexen-1-one
5. Substituted urea	linuron	*N* '-(3,4-dichlorophenyl)-*N* -methoxy-*N* -methylurea
6. Thiocarbamate	EPTC	*S* -ethyl dipropylcarbamothioate

[a]All herbicides were applied postemergence except EPTC. Chemical names of the dinitroaniline herbicides and the unrelated mitotic disrupter herbicides are listed in the text.

a 24-h equilibration period, 25 viable seeds of S and R green foxtail (same biotypes as those used in the field experiment plus a second R population from Nesbitt, Manitoba) were planted 2 cm deep into treated soil in separate 9 cm-diam plastic pots. In addition, six viable caryopses of Katepwa wheat, which served as a reference, were planted in each pot in a similar manner using the same herbicide treatments.

Pots were placed in a growth chamber with a 16-h photoperiod and a 22/16 C day/night temperature setting. The light intensity was approximately 780 $\mu E\ m^{-2}\ s^{-1}$. The experiments, which were conducted twice, were arranged in a randomized complete block design with four replicates per treatment. Plants were watered daily. The shoots of green foxtail and wheat in each pot were harvested 30 d after seeding, oven dried for 24 h at 80 C, and weighed.

4.2.1.2. Postemergence herbicides

Seven herbicides belonging to five chemical families were applied as postemergence treatments (Table 4-1). S and R green foxtail and Katepwa wheat caryopses were planted, as described in the previous section, into untreated soil in 9 cm-diam pots which subsequently were placed in the growth chamber. When the plants were in the two to three leaf stage, the herbicides were applied at 1/4, 1/2 and 1 times the recommended dosage (four replicates per treatment) using a cabinet sprayer with a flat fan (80015 Teejet) nozzle which delivered a spray volume of 124 L ha^{-1}. Fourteen days after treatment, the shoots of the surviving plants in each pot were harvested, oven dried, and weighed. To determine the percentage growth reduction by the herbicide treatments, the post-treatment gain in dry weight was determined. This change in dry weight was then expressed as a percentage of the unsprayed control plants.

4.2.2. Field experiment

The response of S and R green foxtail to PPI ethalfluralin was investigated in 1989 and 1990 at the University of Manitoba Research Station at Portage la Prairie, Manitoba. The soil description and site preparations have been described previously (Chapter 3).

The experiment was arranged in a split-split block design with four replicates. The randomized main plot treatment consisted of nine dosages ranging from 0 to 3 kg ai ha^{-1}. The highest dosage is slightly over twice the recommended dosage of PPI ethalfluralin in rapeseed[2]. The right half of each main plot was seeded to crop, whereas the left half was left unseeded. The S green foxtail biotype was sown on either the front or back half of each block (replicate) (chosen randomly), and the R biotype was sown on the remaining area. The dimensions of the individual main plots were 5 by 10 m.

Ethalfluralin (50% water dispersible granule) was applied on May 18 in 1989 and May 14 in 1990. The herbicide was applied with a bicycle sprayer fitted with flat-fan[3] nozzles on a 2-m boom that delivered a spray volume of 120 L ha^{-1}. Immediately following application, the herbicide was incorporated into the soil to a depth of 8 to 10 cm with a tandem disk, the second pass at a right angle to the first.

Westar canola was seeded using a double disk press drill at a rate of 6 kg viable seed ha^{-1} on May 23 in 1989 and in 1990. The seed was placed 2 to 3 cm deep in rows 15 cm apart. Following seeding the S and R green foxtail biotypes were hand-broadcast at a rate of 5000 viable seeds m^{-2} and

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[2]1991 Guide to Chemical Weed Control, Manitoba Agriculture, 908 Norquay Bldg., Winnipeg, MB R3C OP8.

[3]Tee Jet 80015. Spraying Systems Co., North Ave. and Schmale Rd., Wheaton IL 60187.

incorporated into the soil to a depth of 2 to 4 cm with a spike-tooth harrow, the second pass being at a right angle to the first. Broadleaf and grass weeds were controlled as described previously (Chapter 3). Sample collection and processing procedures are also outlined in Chapter 3.

4.2.3. Data analyses

All data were expressed as a percentage of the untreated controls and the results were combined for the duplicated experiments (growth chamber) or for the 2 yr (field experiment) upon confirmation of homogeneity of variances (Gomez and Gomez 1984). Dose-response curves were fitted to the green foxtail data using non-linear regression procedures (Freund and Littell 1986). The quadratic model was used to describe the response of both S and R biotypes to the herbicides used in the growth chamber studies. However, the exponential decay model best described the response of S foxtail to two dinitroaniline herbicides. For the field data, an exponential decay model was used to describe the response of the S biotype to ethalfluralin, whereas a quadratic model best described the response of the R biotype to the herbicide (Beckie et al. 1990). Crop data were fitted with linear and quadratic regression models. Regression analysis was performed using individual datapoints, but means were plotted. Regression equations were statistically compared when required, using the parameter estimates as described by Ratkowsky (1983). The coefficients of determination (R^2) were calculated as described by Kvalseth (1985). For the field data, the *t* test was used to compare the means of plant variables from untreated plots.

4.3. Results and Discussion

4.3.1. Growth chamber experiments

Because the two R populations responded similarly to all herbicides, only the results of the R (Deloraine) population used in the field experiment are presented. The R biotype was cross-resistant to all of the dinitroaniline herbicides. The response of R and S foxtail to isopropalin is shown in Figure 4-1, with the remaining dinitroaniline dose-response graphs illustrated in Appendix Figure 6. (see Table 4-2 for parameter estimates and ED_{50}'s[4]). The difference in response between the biotypes to trifluralin under field conditions (Chapter 3) was similar to their relative response to the herbicide under

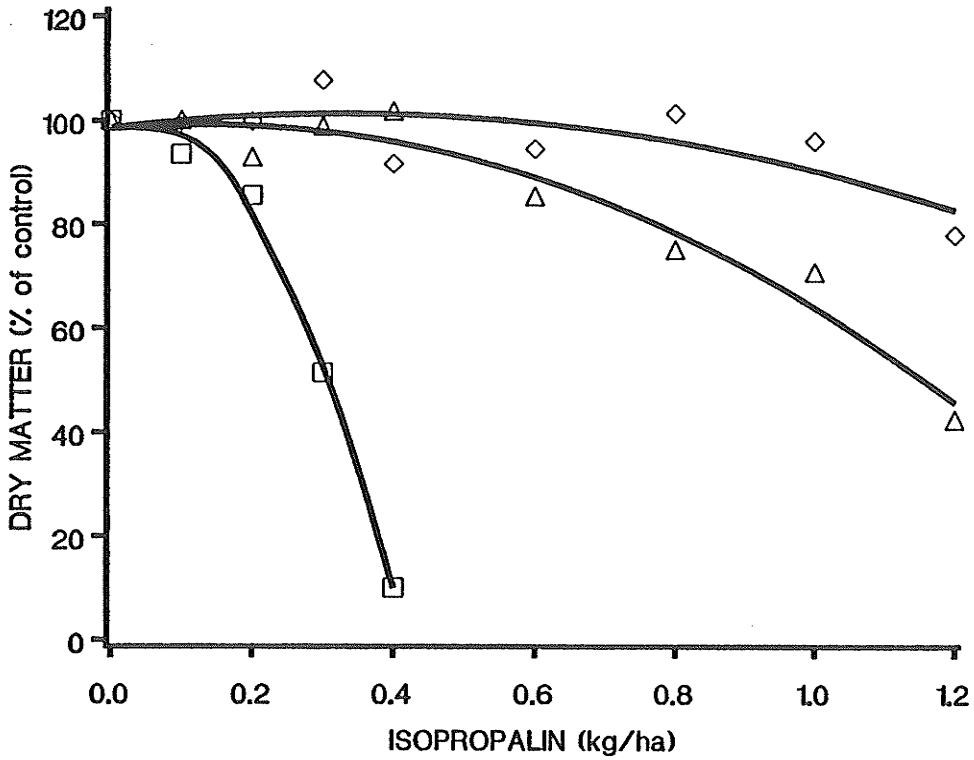

Figure 4-1. The effect of increasing dosages of isopropalin on S (square symbols) and R (triangle symbols) green foxtail and 'Katepwa' wheat (diamond symbols).

[4]Abbreviations: ACCase, acetyl coenzyme A carboxylase; ED_{50}, effective dosage required to reduce the plant variable (eg. density, biomass) by 50% relative to the control.

Table 4-2. Parameter estimates (standard errors in parentheses) and ED_{50}'s of the equations for the regression curves for the shoot dry matter response of S and R green foxtail and 'Katepwa' wheat to dinitroaniline and unrelated mitotic disrupter herbicides.

Herbicide[a]		a[b]	b	c	R^{2c}	ED_{50}	R/S
Dinitroanilines						kg ha^{-1}	
Trifluralin	R[d]	101.6 (5.1)	-12.8 (21.3)	-60.6 (17.0)	0.83	0.82	
	S	100.3 (6.8)	-392 (197)	-448 (989)	0.85	0.12	7
	W	101.7 (4.3)	-47.8 (19.1)	-17.5 (15.2)	0.82	0.83	
Ethalfluralin	R	101.6 (5.0)	54.4 (61.3)	-759 (152)	0.87	0.30	
	S	100.2 (15.4)	-970 (1460)	142 (145)	0.71	0.05	6
	W	108.9 (3.9)	-206 (20)	101 (19)	0.88	0.34	
Oryzalin	R	102.0 (2.6)	-93.6 (11.5)	23.3 (9.5)	0.88	0.66	
	S	99.6 (3.0)	-6.4 (0.4)		0.94	0.11	6
	W	99.1 (4.2)	-11.2 (17.9)	-10.0 (14.5)	0.27	>1.2	
Benefin	R	103.9 (4.8)	-24.9 (20.9)	-51.9 (16.9)	0.81	0.81	
	S	99.1 (5.2)	-65.5 (83.4)	-846 (269)	0.87	0.21	4
	W	103.9 (3.4)	-26.5 (14.8)	-35.9 (12.0)	0.83	0.91	
Isopropalin	R	98.4 (3.7)	11.9 (16.0)	-46.7 (13.5)	0.61	1.2	
	S	98.9 (4.6)	50.5 (55.1)	-682 (133)	0.86	0.31	4
	W	98.7 (3.3)	15.8 (14.6)	-24.6 (11.9)	0.19	>1.2	
Pendimethalin	R	102.3 (3.7)	-31.7 (18.8)	-67.4 (18.1)	0.87	0.68	
	S	100.3 (9.4)	147 (341)	-3110 (1670)	0.77	0.15	4
	W	104.1 (4.4)	-4.0 (18.5)	-39.6 (15.0)	0.63	1.1	

Table 4-2 (continued).

Prodiamine	R	102.9	(3.8)	-121	(17)	34.9	(14.0)	0.84	0.51	
	S	104.3	(4.2)	-5.9	(0.4)			0.93	0.12	4
	W	98.2	(4.6)	6.5	(21.0)	-38.6	(17.1)	0.50	>1.2	
Unrelated mitotic disrupters										
Dithiopyr	R	105.4	(6.0)	-482	(204)	-1530	(1420)	0.81	0.087	
	S	106.7	(8.6)	-903	(554)	-6900	(7080)	0.86	0.044	2
	W	99.0	(3.9)	53.5	(100.6)	-1490	(483)	0.71	0.20	
Pronamide	R	96.8	(3.7)	51.0	(11.7)	-70.6	(7.0)	0.93	1.3	
	S	96.9	(3.4)	47.9	(11.3)	-68.2	(6.7)	0.94	1.3	1
	W	100.3	(2.7)	-5.4	(0.4)			0.98	0.13	

[a]All herbicides were applied preemergence. Plants were harvested 30 d after seeding.

[b]Exponential function equation: $y = a\, e^{bx}$ where a = intercept (% of control) and ab = initial slope; quadratic function equation: $y = a + bx + cx^2$ where a = intercept (% of control), b = linear coefficient, and c = curvilinear coefficient. In both equations, y is shoot dry matter (% of control) and x is the herbicide dosage (kg ha^{-1}).

[c]All coefficients of determination are significant at the 1% level.

[d]S = trifluralin-susceptible, R = trifluralin-resistant green foxtail biotypes, W = 'Katepwa' wheat.

controlled environmental conditions, based on the R/S (ED_{50}) ratios. The R biotype exhibited a similar level of resistance to ethalfluralin and oryzalin, but was more sensitive to the other herbicides. As expected, the R biotype was most resistant to the herbicide that had selected for it in the field.

The R biotype also was less sensitive than the S biotype to an unrelated mitotic disrupter herbicide (Plate 2 and Figure 4-2); R plants were twice as resistant to dithiopyr than S plants (Table 4-2). However, no difference in response for the two biotypes to pronamide was evident. The mechanism of action of dithiopyr may be to alter microtubule polymerization and stability by interacting with a microtubule-associated protein and/or microtubule organizing centers rather than interacting directly with tubulin (Armbruster et al. 1991; Lehnen Jr. and Vaughn 1991). There are no differences in either molecular

Plate 2. Effect of dithiopyr, applied preemergence at 0, 50, 100, 150, and 200 g ai ha^{-1} (left to right), on R (top row) and S (bottom row) green foxtail 30 d after seeding.

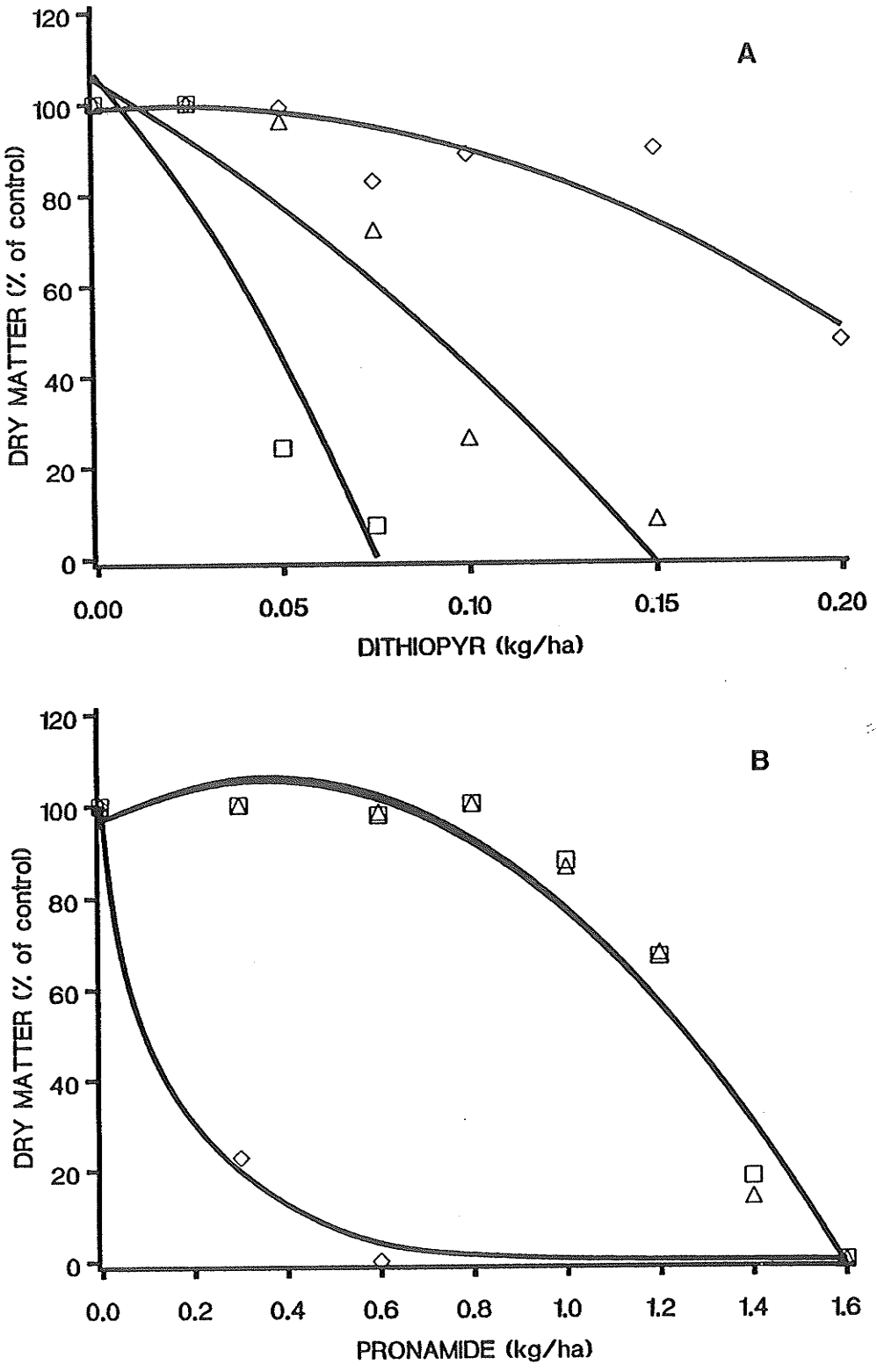

Figure 4-2. The effect of increasing dosages of dithiopyr (A) and pronamide (B) on S (square symbols) and R (triangle symbols) green foxtail and 'Katepwa' wheat (diamond symbols).

weight or abundance of the tubulin subunits between these R and S biotypes[5]. Since the R biotype is cross-resistant to dithiopyr and also exhibits a very high level of resistance to DCPA (dimethyl 2, 3, 5, 6-tetrachloro-1, 4-benzene-dicarboxylate) (>50-fold), an alteration in a microtubule-associated protein may be responsible for resistance in green foxtail (Smeda et al. 1991).

There was no difference in response between the biotypes to herbicides belonging to the other six chemical families (see Appendix Figure 7 and Appendix Table 4). Therefore, an effective strategy to manage R green foxtail would be to rotate herbicides with different sites of action from among these chemical families, with the specific rotation dependent upon the crops grown. However, with the recent discovery of ACCase[4]-herbicide resistant populations of green foxtail in Manitoba (Heap 1991, personal communication), producers must be mindful that there is the potential for resistance in green foxtail to these herbicides. The probability that these new R foxtail biotypes will evolve can be reduced by practicing meaningful herbicide rotations in the cropping system using compounds with different sites of action.

4.3.2. Field experiment

There was no difference in the relative time of emergence of S and R green foxtail seedlings in untreated plots. Both emerged within a week of rapeseed seedling emergence. Soil moisture and temperature conditions were generally conducive for good seed germination, seedling establishment and ethalfluralin efficacy in both years (Chapter 3). The R biotype exhibited cross resistance to ethalfluralin (Plate 3 and Figure 4-3), verifying the results of the growth chamber study. Four wk after green foxtail emergence in cropped plots, the R biotype was 7 to 9 times more resistant to the herbicide than the

[5]Ellis, J. R. 1991. Personal communication. ICI Seeds, Jealott's Hill Research Station, Bracknell, Berkshire, U. K. RG12 6EY.

Plate 3. Effect of 3.0 kg ai ha^{-1} ethalfluralin on R green foxtail in rapeseed 4 wk after weed emergence at Portage la Prairie in 1990. Note crop thinning caused by the herbicide treatment.

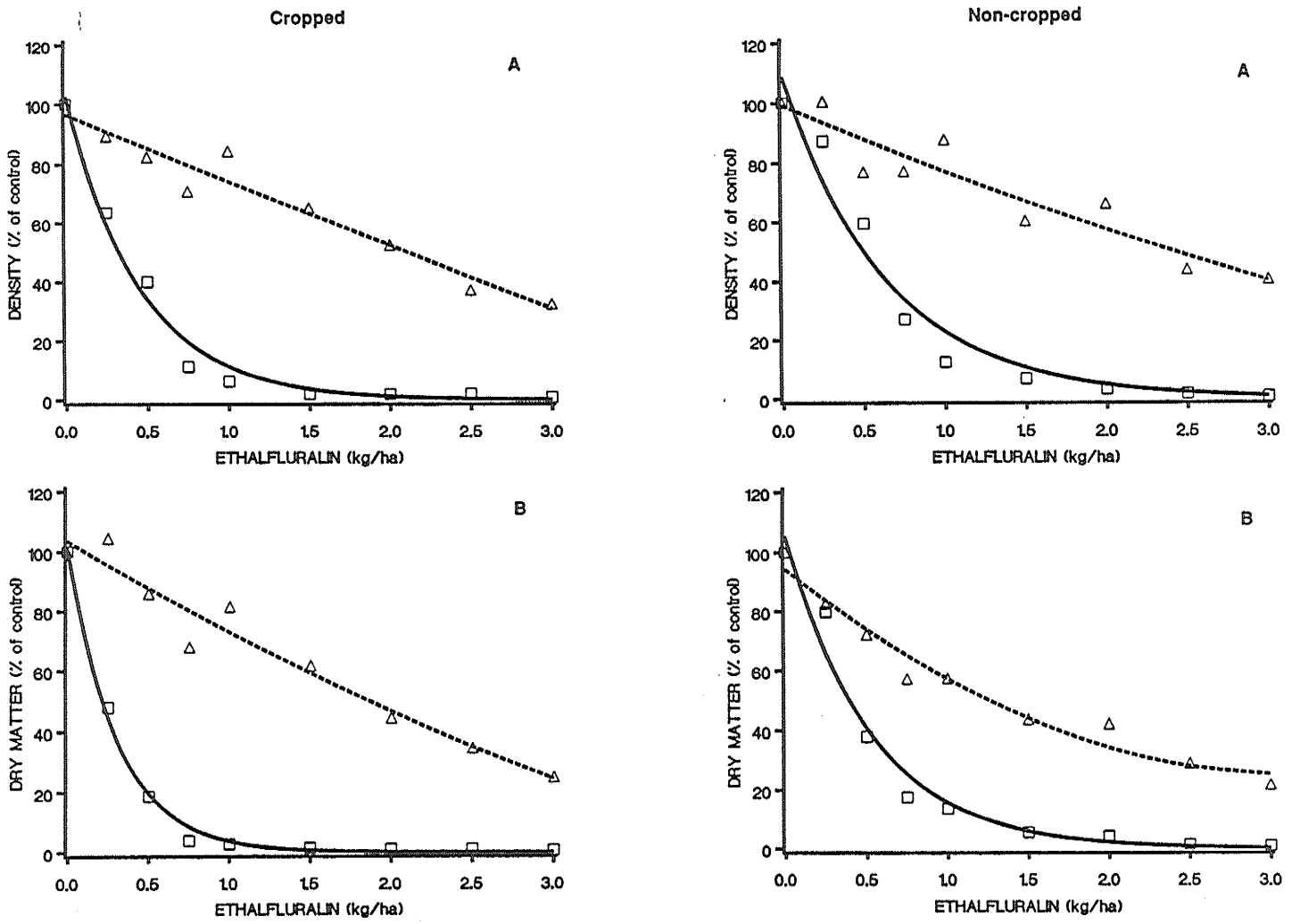

Figure 4-3. The effect of increasing dosages of ethalfluralin on R (dashed line) and S (solid line) green foxtail density (A) and dry matter production (B) 4 wk after emergence under cropped and non-cropped conditions at Portage la Prairie in 1989 and 1990. See Table 4-3 for equations and parameter estimates.

S biotype, based on density and biomass determinations (Table 4-3). These differences in sensitivity between the biotypes are similar to their relative response to the herbicide under growth chamber conditions and also to their response to PPI trifluralin (Chapter 3), even though ethalfluralin is more active on both biotypes than trifluralin. No significant differences were observed between S and R green foxtail density or biomass in untreated crop plots (see Table 4-3).

Rapeseed density was affected by ethalfluralin differently in S and R foxtail plots (Figure 4-4 and Table 4-4). Rapeseed density in S green foxtail plots increased as herbicide dosages increased, reflecting the decline in green foxtail competition, but decreased at higher-than-recommended dosages (>1.4 kg ha^{-1}) due to crop injury. However, rapeseed in R foxtail plots showed no response to increasing dosages of the herbicide. Rapeseed dry matter in S and R foxtail plots were similar. Biomass increased slightly with increasing herbicide dosages because of the decline in green foxtail vigor but decreased at high dosages because of crop injury.

Under non-cropped conditions, the R biotype exhibited a similar level of resistance as under cropped conditions, based on density determinations, but was less resistant on the basis of shoot dry matter determinations (Figure 4-3 and Table 4-3).

Differences in response of the two foxtail biotypes to increasing dosages of ethalfluralin, under cropped conditions (Figure 4-5), were maintained between the first sampling date and foxtail maturity. Rapeseed density in herbicide-treated S foxtail plots were generally higher than in corresponding R foxtail plots due to less interference by the former biotype, with the largest difference occurring in plots treated with ethalfluralin at close to the recommended dosage

Table 4-3. Parameter estimates (standard errors in parentheses) and ED_{50}'s of the equations for the regression curves for the response of S and R green foxtail to ethalfluralin under cropped and non-cropped conditions at Portage la Prairie in 1989 and 1990.

Plant variable[a]	a[b]	b	c	R^{2c}	ED_{50}	R/S
4 wk after emergence						
Cropped conditions						
S density	102.6(3.8)	-2.2(0.2)		0.90	0.3	
R density	96.6(6.7)	-22.6(12.0)	0.2(3.9)	0.42	2.2	7
S dry wt	101.0(4.8)	-3.3(0.3)		0.85	0.2	
R dry wt	103.5(6.2)	-32.5(11.1)	2.1(3.6)	0.55	1.8	9
Non-cropped conditions						
S density	108.8(3.9)	-1.6(0.1)		0.90	0.4	
R density	99.2(7.2)	-23.3(12.6)	1.2(4.1)	0.34	2.4	6
S dry wt	105.5(4.1)	-1.9(0.1)		0.89	0.4	
R dry wt	94.3(5.1)	-44.1(8.8)	7.0(2.9)	0.60	1.4	4
Maturity						
Cropped conditions						
S density	108.1(4.7)	-1.9(0.2)		0.87	0.4	
R density	95.3(4.7)	-5.8(7.9)	-4.7(2.7)	0.48	2.6	7
S dry wt	99.2(2.2)	-4.7(0.2)		0.96	0.2	
R dry wt	89.8(4.7)	-38.0(8.3)	5.7(2.7)	0.60	1.5	8
S seed wt	102.4(6.2)	-3.3(0.4)		0.78	0.2	
R seed wt	89.9(7.4)	-40.4(13.0)	5.8(4.2)	0.44	1.4	7
Non-cropped conditions						
S density	106.1(3.6)	-1.5(0.1)		0.91	0.4	
R density	91.9(4.7)	-14.1(7.9)	-1.4(2.6)	0.54	2.6	6
S dry wt	105.8(5.1)	-0.6(0.1)		0.68	1.2	
R dry wt	92.4(4.6)	6.9(8.0)	-5.9(2.6)	0.30	3.5	3
S seed wt	99.5(6.4)	-0.6(0.1)		0.56	1.1	
R seed wt	93.9(4.5)	11.9(8.8)	-7.6(3.0)	0.25	3.4	3

[a]Mean values ± standard error for plant variables (per 1-m^2 basis, wt in g) in control plots: **4 wk, cropped conditions** Density: S 3 310(460), R 3 270 (290); Dry wt: S 87(11), R 102(16); **4 wk, non-cropped conditions** Density: S 4 690(550), R 4 250(410); Dry wt: S 169(20), R 198(23); **Maturity, cropped conditions** Density: S 2 020(200), R 2 140(210); Dry wt: S 345(37), R 351(44); Seed wt: S 54(9), R 61(10); **Maturity, non-cropped conditions** Density: S 3 780(350), R 3 730(410); Dry wt: S 810(85), R 864(86); Seed wt: S 208(26), R 223(25).

[b]Exponential function equation: $y = a\, e^{bx}$ where a = intercept (% of control) and ab = initial slope; quadratic function equation: $y = a + bx + cx^2$ where a = intercept (% of control), b = linear coefficient, and c = curvilinear coefficient; y is the plant variable (% of control) and x is the ethalfluralin dosage (kg ha^{-1}).

[c]All coefficients of determination are significant at the 1% level.

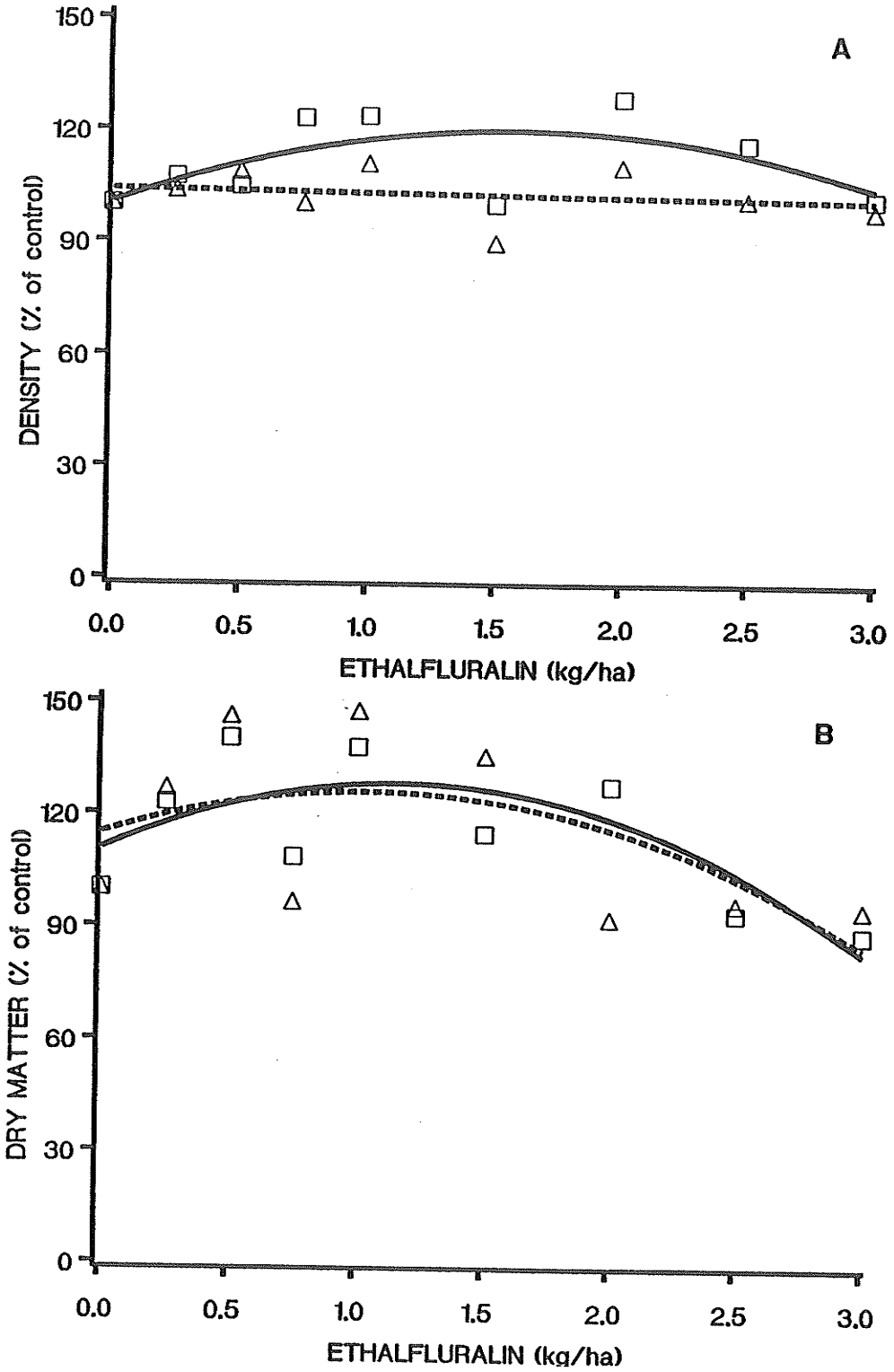

Figure 4-4. The effect of increasing dosages of ethalfluralin applied as a PPI treatment on rapeseed density (A) and dry matter production (B) 4 wk after foxtail emergence, in plots sown to R (dashed line) and S (solid line) biotypes at Portage la Prairie in 1989 and 1990. See Table 4-4 for equations and parameter estimates.

Table 4-4. Parameter estimates (standard errors in parentheses) of the equations for the regression curves for the response of rapeseed to ethalfluralin at Portage la Prairie in 1989 and 1990.

Crop variable	a[a]	b	c	R^{2b}
4 wk after emergence[c]				
S[d] density[e]	100.4 (6.9)	24.2 (12.2)	-7.7 (4.0)	0.06*
R density	103.8 (5.6)	-1.1 (3.6)		0.01
S dry wt	110.6 (10.5)	30.2 (18.9)	-13.2 (6.2)	0.11**
R dry wt	114.8 (14.8)	21.4 (26.4)	-10.6 (8.7)	0.05
Maturity				
S density	106.2 (5.9)	41.2 (10.6)	-13.6 (3.4)	0.20**
R density	93.9 (3.6)	0.6 (2.3)		0.01
S dry wt	111.0 (5.6)	29.2 (10.6)	-8.4 (3.4)	0.13**
R dry wt	106.0 (7.2)	17.7 (13.4)	-3.6 (4.4)	0.07*
S seed wt	109.0 (9.8)	30.4 (18.4)	-9.6 (6.0)	0.05
R seed wt	110.1 (10.6)	13.6 (19.7)	-2.3 (6.4)	0.04

[a]Linear function equation: $y = a + bx$ where a = intercept (% of control), b = slope; quadratic function equation: $y = a + bx + cx^2$ where a = intercept (% of control), b = linear coefficient, and c = curvilinear coefficient. In both equations, y is the crop variable (% of control), and x is the ethalfluralin dosage (kg ha^{-1}).

[b]Coefficients of determination: * significant at the 5% level; ** significant at the 1% level.

[c]First sampling date: 4 wk after green foxtail emergence (stem extension to early bud crop development stages); second sampling date: green foxtail maturity (crop density and shoot dry matter) and crop maturity (yield).

[d]Crop in competition with trifluralin-susceptible (S) or trifluralin-resistant (R) green foxtail biotypes.

[e]In control plots, mean values ± standard errors (per 1 m^2, wt in g) for select variables were: **4 wk** Density: S 97(5), R 102 (9); **Maturity** Seed wt: S 206(38), R 187(27).

(Figure 4-6). However, no differences in crop biomass or yield between S and R foxtail plots were evident. The response of crop density in S and R foxtail plots to increasing dosages of ethalfluralin was similar to the earlier sampling date. Rapeseed biomass in both S and R foxtail plots increased slightly, whereas yield was not significantly affected by increasing dosages.

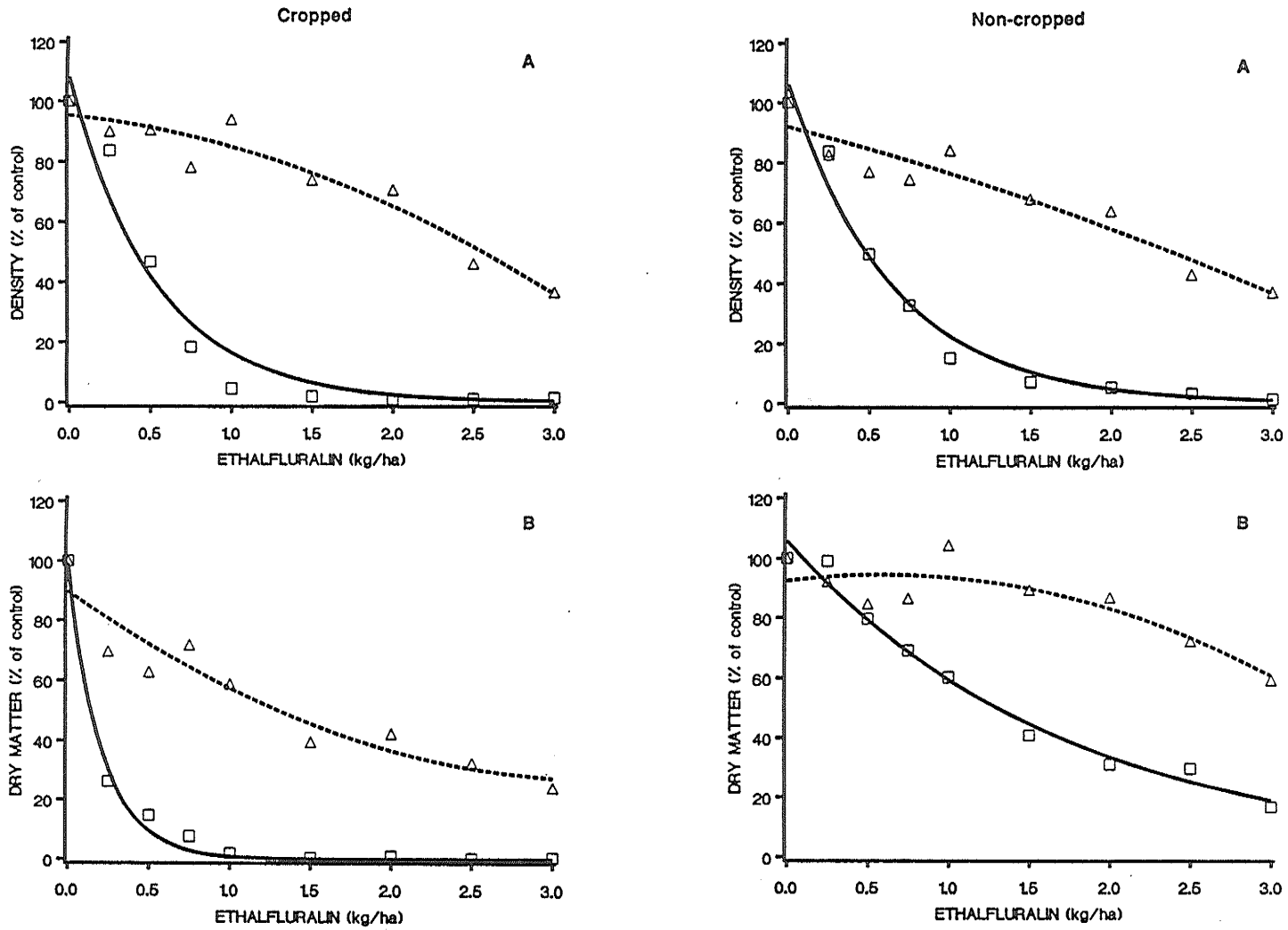

Figure 4-5. The effect of increasing dosages of ethalfluralin on R (dashed line) and S (solid line) green foxtail density (A) and dry matter production (B) at maturity under cropped and non-cropped conditions at Portage la Prairie in 1989 and 1990. See Table 4-3 for equations and parameter estimates.

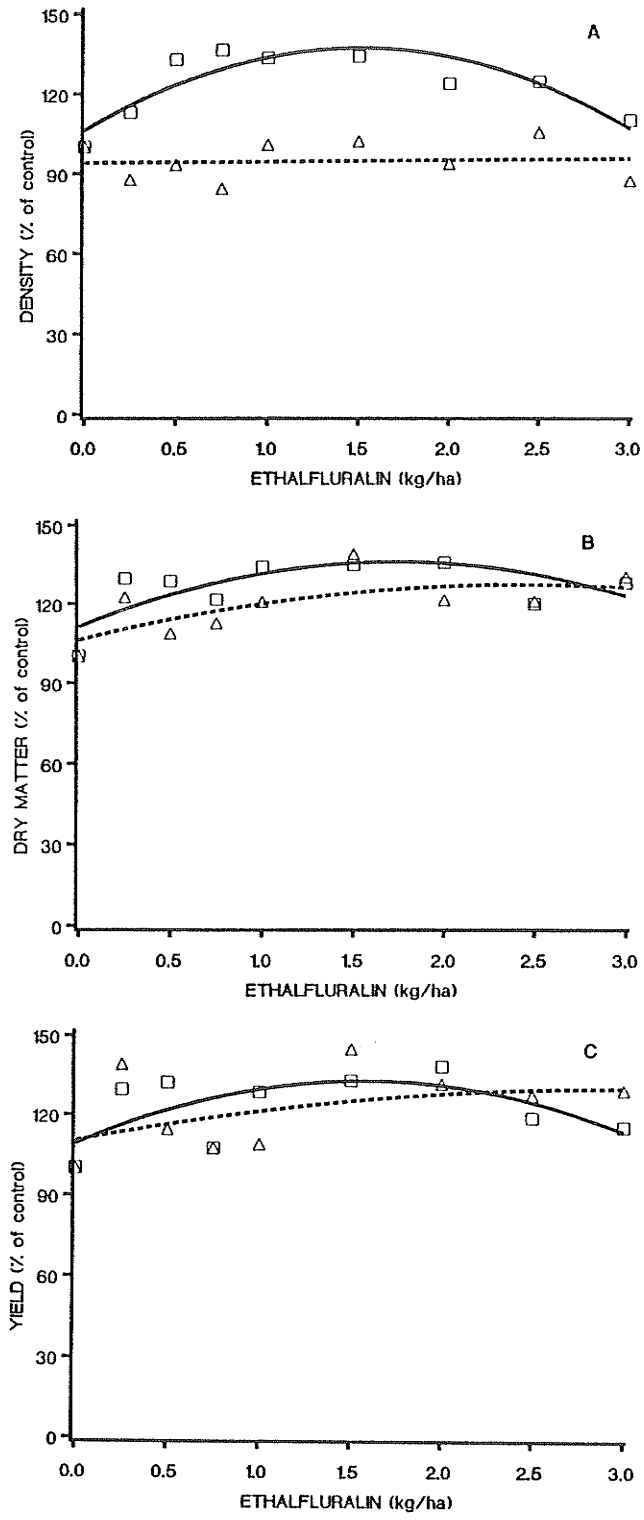

Figure 4-6. The effect of increasing dosages of ethalfluralin applied as a PPI treatment on rapeseed density (A), dry matter production (B), and yield (C) at maturity, in plots sown to R (dashed line) and S (solid line) biotypes at Portage la Prairie in 1989 and 1990. See Table 4-4 for equations and parameter estimates.

Under non-cropped conditions (Figure 4-5) enhanced tillering of surviving S plants in plots treated at higher dosages (Figure 4-7) contributed to a lower R/S ratio when determined from biomass compared to density measurements.

Seven times as much herbicide was required to reduce R seed production by 50% than to reduce S seed production by the same amount under cropped conditions (see Figure 4-8, Appendix Figure 8, and Appendix Table 5). Therefore based on relative seed return, the difference in sensitivity between S and R green foxtail to ethalfluralin under cropped conditions was maintained over the growing season. Under non-cropped conditions, increased tillering of the S biotype at higher dosages markedly reduced the effect of increasing dosages of the chemical on S seed return. As in the trifluralin field experiments,

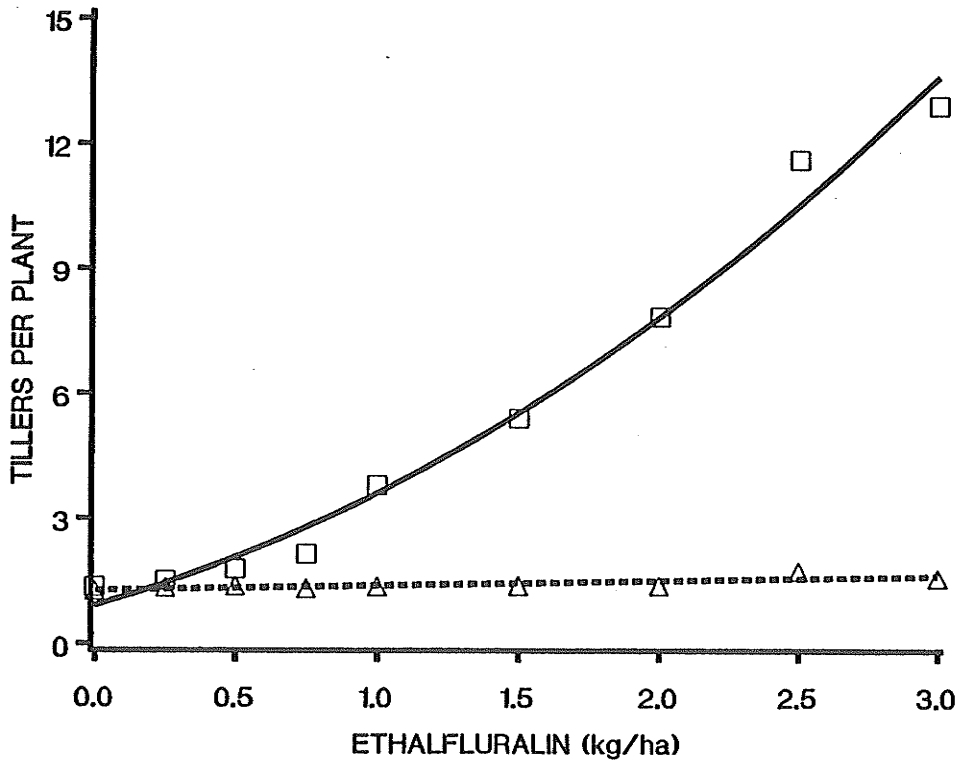

Figure 4-7. The effect of increasing dosages of ethalfluralin on R (dashed line) and S (solid line) green foxtail tillers per plant at maturity under non-cropped conditions at Portage la Prairie in 1989 and 1990. R^2 for R and S biotypes = 0.17** and 0.53**, respectively.

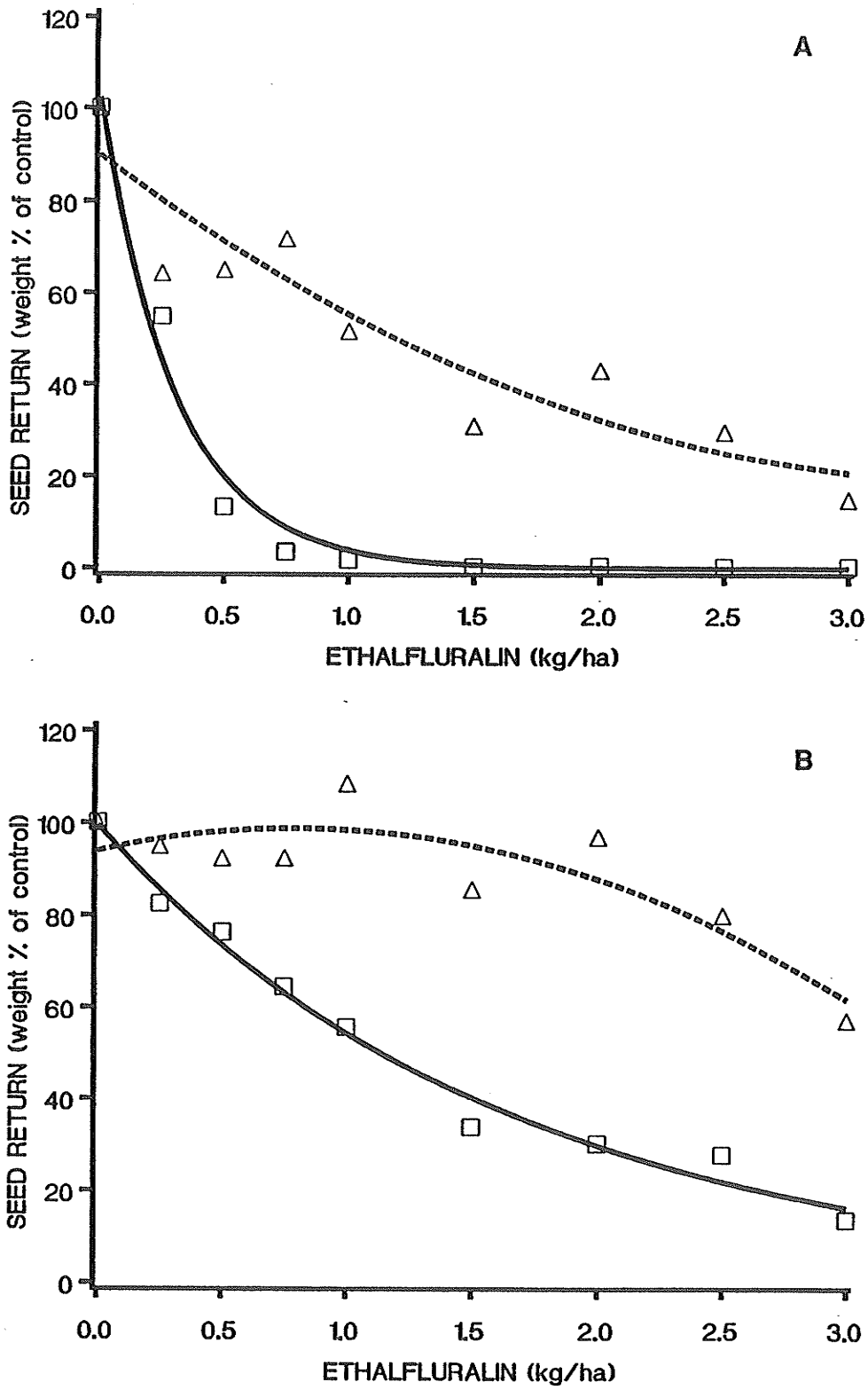

Figure 4-8. The effect of increasing dosages of ethalfluralin on R (dashed line) and S (solid line) green foxtail seed production under cropped (A) and non-cropped (B) conditions at Portage la Prairie in 1989 and 1990. See Table 4-3 for equations and parameter estimates.

no differences between seed return of S and R biotypes were observed in untreated plots under either cropped or non-cropped conditions.

Calculated from the dose-response equations (see Table 4-3), at the recommended ethalfluralin dosage in rapeseed (1.4 kg ha^{-1}) the density of S plants 4 wk after emergence was reduced by 95% compared to untreated plots, whereas the density of R plants was reduced by 35% (Table 4-5). Biomass of S and R foxtail was reduced by 99% and 38%, respectively. The effective kill (seed yield reduction) of S foxtail was 99% compared to 55% for R foxtail. Therefore, the initial reductions in density or biomass do not exceed the effective kill, similar to PPI trifluralin (Chapter 3). The selection pressure of ethalfluralin on green foxtail under cropped conditions is: (1-0.55)/(1-0.99) = (0.45/0.01) = 45. This is less than the corresponding value for PPI trifluralin (58), due to the greater reduction of R seed yield at the recommended dosage

Table 4-5. Percent reduction in S and R green foxtail variables in response to ethalfluralin at the recommended dosage in rapeseed (1.4 kg ha^{-1}) at Portage la Prairie in 1989 and 1990.

	Cropped		Non-cropped	
	S	R	S	R
	% reduction[a]			
4 wk after emergence				
Density	95	35	88	31
Dry wt	99	38	93	54
Maturity				
Density	92	22	88	31
Dry wt	86	52	53	9
Seed wt	99	55	57	4
Seed no	99	55	49	2

[a]Percent reduction calculated from the regression equations at the application dosage of 1.4 kg ha^{-1}.

of ethalfluralin than trifluralin.

4.4. Modelling the Rate of Enrichment of R Green Foxtail by Ethalfluralin

The rate of enrichment of R green foxtail by ethalfluralin is shown in Figures 4-9 and 4-10 for Model I and II, respectively. The rate of enrichment is slightly lower than for PPI trifluralin because of the reduced selection pressure (45 versus 58). However, despite a lower selection pressure by ethalfluralin, the conclusions pertaining to trifluralin apply to ethalfluralin as well. Herbicide rotations will not provide a real added delay in the appearance of resistance of R green foxtail because of the high selection pressure and small fitness differential, if any, between R and S biotypes. The only delay will be for the number of seasons that ethalfluralin (or trifluralin) is not used. In terms of the rate of evolution of green foxtail resistance, a herbicide rotation alternating ethalfluralin and trifluralin for weed control in rapeseed would be very similar to only using trifluralin.

Although these model results (and those from Chapter 3) suggest that ethalfluralin and trifluralin should be used sparingly in the cropping system to delay or preclude the appearance of R foxtail, some producers that already have fields infested with the weed have continued to use these herbicides to control other weed species. In those fields, a postemergence herbicide will be required to effectively control R foxtail. Regardless of whether or not fields are infested with R foxtail, trifluralin and ethalfluralin should be used only periodically in the herbicide rotation, since they may select for other R weed biotypes. This applies to other herbicides as well. Meaningful herbicide rotations are critical for reducing the propensity for resistance in weeds. By determining the response of R biotypes to herbicides that normally control the species, herbicide rotations can be designed to maximize the market lifetime of

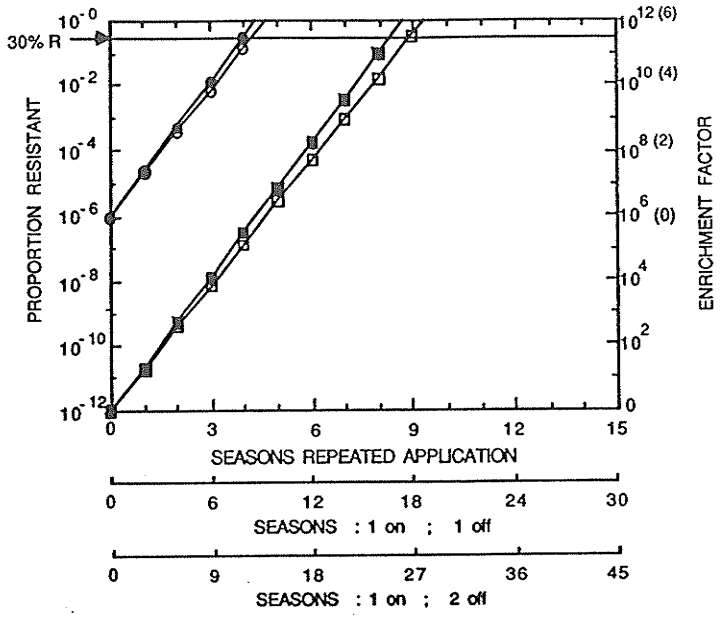

Figure 4-9. The predicted rate of evolution of R green foxtail when ethalfluralin is applied as a PPI treatment in rapeseed at the recommended dosage (Model I). The initial frequency, N_o , $=10^{-6}$ (dominant monogene trait) or 10^{-12} (recessive monogene trait; the fitness differential, *f*, between the R and S green foxtail biotypes is either 0.8 (open symbols) or 1.0 (closed symbols); the seed bank longevity, ß, is 2 yr. The selection pressure, å, = 45 (see section 4.3.2).

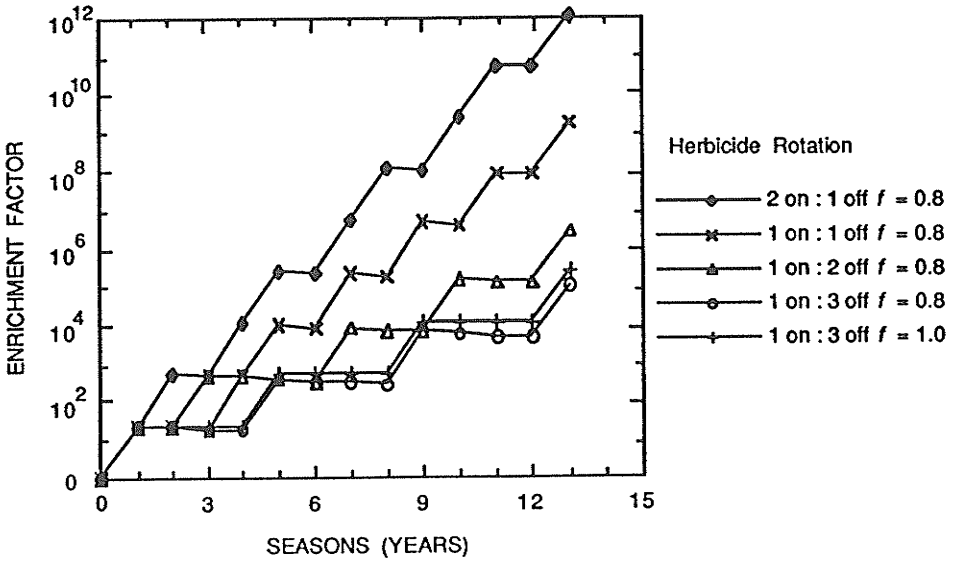

Figure 4-10. The predicted rate of evolution of R green foxtail when ethalfluralin is applied as a PPI treatment in rapeseed at the recommended dosage (Model II). The fraction of seeds leaving the seed bank each year, ∂, =0.5; the selection pressure, å, = 45; the fitness differential between the R and S biotypes in the absence of ethalfluralin, f_{off}, = 0.8 or 1.0; f_{on} = 1.0.

cost-effective herbicides and minimize the adverse effects of R weeds on crop production.

5. RESPONSE OF RESISTANT GREEN FOXTAIL (*Setaria viridis*) TO TWO DINITROANILINES: BIOASSAY AND MITOTIC INDEX[1]

Abstract. The response of resistant (R) and susceptible (S) green foxtail to trifluralin and ethalfluralin was examined using a petri dish bioassay, and by determining mitotic indices of treated and untreated root tips. In the petri dish assay, radicle growth of R green foxtail exposed to trifluralin concentrations of up to 0.4 ppm (w/v) was not inhibited. Radicle growth of S foxtail was completely inhibited at this concentration. Radicle growth of both S and R biotypes was much more sensitive to ethalfluralin than to trifluralin. R foxtail was nine times more resistant to trifluralin and six times more resistant to ethalfluralin than S foxtail. Shoots were more sensitive to trifluralin or ethalfluralin than roots, with shoot growth of both R and S biotypes being inhibited at trifluralin concentrations of 0.2 ppm or more and at ethalfluralin concentrations of 0.05 ppm or more. Best discrimination between R and S green foxtail biotypes was achieved by measuring radicle length after incubation of germinated caryopses at 0.3 ppm trifluralin in the dark for 5 d at 22 C. To determine mitotic indices, squashes of S and R root meristems treated with increasing concentrations of trifluralin and ethalfluralin were examined by light microscopy. The R biotype was 10 times more resistant to both trifluralin and ethalfluralin than the S biotype as indicated by the mitotic indices. As in the petri dish assay, both biotypes were much more sensitive to ethalfluralin than to trifluralin. The herbicide concentrations causing an increase in the number

[1]Parts of this chapter were included in the following publication: Beckie, H. J., L. F. Friesen, K. M. Nawolsky, and I. N. Morrison.1990. A rapid bioassay to detect trifluralin-resistant green foxtail (*Setaria viridis*). Weed Technol. 4:505-508.

of cells in condensed prophase (prometaphase) corresponded well with the concentrations required to inhibit radicle growth. **Nomenclature:** Ethalfluralin, *N* -ethyl-*N* -(2-methyl-2-propenyl)-2, 6-dinitro-4-(trifluoromethyl)benzenamine; trifluralin, 2, 6-dinitro-*N,N*- dipropyl-4-(trifluoromethyl)benzenamine; green foxtail, *Setaria viridis* (L.) Beauv. # SETVI.

Additional index words: Herbicide resistance, mitotic index, SETVI

5.1. Introduction

The occurrence of trifluralin-resistant green foxtail, primarily in southwestern Manitoba, was confirmed initially in dose-response experiments conducted in the growth chamber during the winter of 1988-89 (Morrison et al. 1989). Since then, R foxtail has been shown to be resistant to other dinitroaniline herbicides and to an unrelated mitotic disrupter herbicide (Chapter 4). The initial identification of R foxtail was done by growing plants for several weeks in pots filled with trifluralin-treated soil under defined temperature and light regimes in a growth chamber (Morrison et al. 1989). This procedure was both time consuming and dependent on ready availability of growth cabinets or chambers, which are expensive to operate. To screen large numbers of green foxtail seed samples to determine resistance, a simple, inexpensive bioassay was required.

Petri dish bioassays are simple, rapid, and useful for determining herbicide concentrations either in aqueous solution or in soil (Lavy and Santelmann 1986). They have been used to determine the response of seedlings to varying concentrations of trifluralin present in both these media (Rahman and Ashford 1970; Jacques and Harvey 1974). This type of assay also has been used as a rapid screening technique for testing seed stocks for resistance (Moss 1987). When the petri dish bioassay is used, the chosen plant parameter of the R biotype should be less sensitive to varying concentrations of the herbicide than

the S biotype. The herbicide concentration providing the largest difference in response between the biotypes is used to test seed stocks for resistance.

Seedlings of susceptible plant species exposed to mitotic disrupter herbicides have root tips that are typically club-shaped rather than tapered, because of larger isodiametric cells caused by the loss of cortical and phragmoplast MTs[2] (Vaughn 1990). Swollen root tips are an excellent diagnostic symptom for determining if a herbicide is causing mitotic disruption (Vaughn and Koskinen 1987; Hess 1989). Growth in root tips cease after dinitroaniline herbicide treatment because cell division is disrupted and consequently, no new cells are produced in the meristem. The concentrations at which dinitroaniline herbicides disrupt mitosis are quite low and correlate well with those reported to inhibit root growth (Hess 1989).

With the loss of spindle MTs from treated meristem cells, chromosomes can not align at the equator during metaphase. Instead, the chromosomes coalesce in the middle of the cell and appear as a clump of densely stained chromatin (Vaughn et al. 1990). This appearance is the result of the chromosomes continuing to shorten, thicken, and uncoil even though dinitroaniline herbicides prevent their normal movement to the metaphase configuration (Hess 1989). This aberrant mitotic figure is usually referred to as condensed prophase, reflecting the appearance of the chromosomes (Hess 1989), or prometaphase (Vaughn 1986a).

To determine the response of S and R biotypes to dinitroaniline herbicides at the site of herbicidal action, root tip squashes of herbicide-treated R and S biotypes have been used to measure changes in the mitotic indices (proportion of meristem cells in mitosis) (Vaughn 1986a, Vaughn et al. 1987; Vaughn et al.

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[2]Abbreviations: ED_{50}, herbicide concentration required to reduce the plant variable (eg. radicle, shoot) by 50% relative to the control; MT, microtubule.

1990). Mitotic indices are a convenient measure of the effect of a mitotic disrupter since the disruption of mitosis generally results in a longer time in mitosis and therefore a higher mitotic index (Vaughn et al. 1990). The mitotic indices of S biotype root meristems following herbicide treatment will tend to be greater than those of R biotype meristems because more mitotic cells in the former biotype will be in condensed prophase. In root meristem tissue of many species, the number of metaphase, anaphase, and telophase mitotic figures decreases to zero within 3 to 6 h of treatment; aberrant mitotic figures appear immediately after treatment and increase during the first 6 to 8 h to as many as 20 to 30% of the total meristematic cells (Hess 1987). In contrast, the number of cells in mitosis at any one time in untreated tissue is commonly only 4 to 8 % (Hess 1989).

Mitotic indices have been used to provide an insight into the mechanism of action of mitotic disrupter herbicides. The stages of mitosis that require the function of MTs (metaphase, anaphase, and telophase) are absent in dinitroaniline-treated meristems, whereas the stage that does not require MTs (prophase) is unaltered (Lignowski and Scott 1972). This suggests that the effect of dinitroaniline herbicides on mitosis is related either to MT function or to their absence from meristem cells. The latter explanation was verified by Bartels and Hilton (1973), who reported that spindle MTs were absent in wheat and corn root meristem cells that had been treated with trifluralin or oryzalin. These observations led to further studies on the effects of dinitroaniline herbicides on the MT constituents (tubulin), which clarified the precise mechanism of action. The mechanism of action of phosphoric amide herbicides is the same as that of the dinitroanilines, and was deduced in a similar manner (Sumida and Udea 1976; Hess 1989; Vaughn and Lehnen, Jr. 1991).

This chapter decribes the response of R and S green foxtail to trifluralin and ethalfluralin as determined at the whole-plant level (petri dish bioassay) and at the cell level (mitotic index).

5.2. Materials and Methods

5.2.1. Germination procedure

For all experiments, green foxtail caryopses of the R and S populations were germinated on a thin layer of silica sand saturated with distilled water in trays covered with plastic wrap. The populations were determined to be R or S following criteria described by Morrison et al. (1989). The trays were placed in germination cabinets in the dark for 28 to 30 h at 25 C for the petri dish bioassay and 72 h at 22 C for the mitotic index study.

5.2.2. Petri dish bioassay

The lids of 9-cm diam glass petri dishes were lined with two filter papers[3]. A 4-ml aliquot of test solution was applied uniformly over the filter paper. Ten germinated caryopses with visible radicles not exceeding 1 mm in length were placed on the saturated filter paper. The lids were covered with the bottoms, and the petri dishes were sealed with parafilm and incubated in a dark germination cabinet for 5d at 22 C. At the end of the incubation period for the petri dish bioassay, the lengths of the radicle and shoot were measured to the nearest millimeter.

Aqueous solutions with concentrations as high as 0.6 ppm of trifluralin (500 g ai L^{-1} emulsifiable concentrate) or ethalfluralin (50% water dispersible granule) were prepared by serial dilution for a preliminary dose-response experiment to determine the concentration of each herbicide providing the

[3]Whatman #1, Whatman Int. Ltd., Maidstone, U.K.

largest difference in shoot or radicle growth between R and S biotypes. Distilled water containing no herbicide was the control. Five green foxtail populations from Manitoba (two S and three R) were used in the trifluralin dose-response experiment (see Appendix Table 6). One R and one S biotype were used in the ethalfluralin dose-response experiment. Results from these preliminary dose-response experiments indicated that a solution containing 0.3 ppm of trifluralin or 0.05 ppm of ethalfluralin was optimum and that radicle length was a suitable test parameter (data not shown).

Prior to repeating the dose-response experiment, physical and environmental factors were investigated to assess their effect on the growth of S and R biotypes treated with trifluralin. In one experiment, plastic petri dishes were used instead of glass. In another experiment, glass microfibre filter paper[4] was substituted for cellulosic paper[3]; in a third, the effect of light was investigated by incubating petri dishes in a germination cabinet with a 16-h photoperiod for 5 d at 22 C. The light intensity was approximately 14 $\mu E\ m^{-2}\ s^{-1}$.

After repeating the dose-response experiments using the standard procedure, twelve S and R populations were randomly selected and tested to verify the accuracy of the bioassay procedure. Except for this final experiment, which had five replicates, all treatments were replicated four times (one petri dish per replicate). Each experiment was conducted twice, and results were combined upon confirmation of homogeneity of variances (Gomez and Gomez 1984. Prior to analysis, all datapoints in each experiment, including values from control treatments, were transformed by subtracting the observed baseline growth of the radicle or shoot (5 mm) that occurred regardless of treatment.

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[4]Whatman G F/A

5.2.3. Mitotic index study

A 4-ml aliquot of test solution was applied uniformly over the two filter papers, which lined the lids of the glass petri dishes. Trifluralin concentrations ranged from 0.05 to 1.0 ppm, whereas ethalfluralin concentrations were 0.025 to 0.6 ppm. Distilled water was the control. Ten germinated caryopses with radicles approximately 1 cm long were placed on the saturated filter paper. The lids were covered with the bottoms, and the petri dishes were sealed with parafilm and incubated in a dark germination cabinet for 24 h at 22 C. All treatments were replicated 3 times (one petri dish per replicate). The experiments were repeated twice and results were combined upon confirmation of homogeneity of variances (Gomez and Gomez 1984).

A preliminary study of the mitotic cycle was initiated prior to the experiments to obtain some knowledge of the synchrony in the stages of mitosis of root meristem cells of S and R green foxtail. The highest mitotic indices generally occurred from 11 am to 1 pm CST for the seedlings that were incubated in the dark. Therefore, to discern the effects of the herbicide treatment, the seedlings were treated at 9 am and harvested 24 h later. This ensured that the root tips would be exposed to trifluralin before the period of relatively high mitotic activity and would be sampled when the background levels of mitosis were low (Vaughn 1986a).

Root tip squashes were prepared using the procedures described by Holmsen and Hess (1984), but with some minor modifications. At sampling time, the roots were rinsed with deionized water and the apical 1 cm of each root tip was excised. The root tips were incubated in cold (4 C) fixative [ethanol:glacial acetic acid (3:1, v/v)] for 24 h. After the incubation period, the fixative was replaced with five changes of deionized water and the root meristem cell walls were softened for 5 h at 27 C in a 1% (w/v) solution of

pectinase[5] in 0.1 M acetate buffer, pH 4.0. The root tips were later rinsed with deionized water and hydrolyzed for 15 min in 1N HCl at 60 C. The tips were rinsed again, and the apical 1 mm was excised and placed on a microscope slide. The apical meristem was stained with acetocarmine (0.1% w/v), a cover slip was applied, and the meristem was squashed. The slides were heated gently (60 C) on a slide warming tray for 30 s to improve the staining of the tissue. Meristem cells were examined by light microscopy using an oil immersion lens (total magnification of 1250X). One thousand cells from each of three root tips per replicate (nine root tips per treatment) were counted to determine the proportion of cells in mitosis.

5.2.4. Data analyses

Data analyses involved the calculation of standard errors. The dose-response experiments were analyzed using nonlinear regression procedures (Freund and Littell 1986). The response of the radicle of R foxtail to trifluralin was best described by a quadratic model. In contrast, the response of the radicle of S foxtail to trifluralin and of the radicle of the R biotype to ethalfluralin, as well as the shoot of both biotypes to trifluralin and ethalfluralin was described by an exponential decay model (Milthorpe and Moorby 1974).

The response of mitotic indices of R biotype root meristems to trifluralin and ethalfluralin was best described by a quadratic model (Beckie et al. 1990) whereas the response of mitotic indices of S biotype meristems to the herbicides was described by a rectangular hyperbolic model (Cousens 1985). Regression analysis was performed using individual datapoints, but means were plotted. The coefficients of determination (R^2) were calculated as described by Kvalseth (1985).

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[5]Sigma Chemical Co., St. Louis, MO.

5.3. Results and Discussion

5.3.1. Petri dish bioassay

In all experiments, a 'time lag' between exposure to trifluralin and growth inhibition, similar to that noted by Appleby and Valverde (1989), was observed. Regardless of treatment, growth of both radicle and shoot occurred during the germination period and for a short period after initial exposure to the herbicides. This baseline growth (5 mm ± 1 mm) was determined to be the total growth of the radicle or shoot of S foxtail that occurred in the presence of 0.6 ppm trifluralin or ethalfluralin. S foxtail also grew a similar amount, even when a much higher concentration of 1.5 ppm trifluralin or ethalfluralin was used in experiments confirming this observation.

The type of petri dish, filter paper composition, and presence of light did not have a marked effect on radicle length (Table 5-1). However the variability in radicle length, as indicated by the coefficients of variation, tended to be lowest when glass dishes were used with Whatman #1 filter paper and incubated in the dark. Trifluralin activity probably was affected by plastic dishes, which displayed a yellowish tinge after use, and light. This may explain the increased variability observed. Considering the chemical properties of trifluralin, specifically the high octanol-water partition coefficient (log P = 5.07 at 25 C; WSSA 1989), it is likely that some of the chemical partitioned into plastic. Trifluralin photodecomposes in the presence of light (Rahman and Ashford 1970; WSSA 1989), which may have contributed to increased variability.

In the trifluralin dose-response experiment, the largest difference in radicle length between the R and S biotypes occurred at concentrations of 0.2 to 0.4 ppm (Figure 5-1). Radicle growth was not inhibited for the three R populations at these concentrations. However, radicle growth of S green foxtail

Table 5-1. The effect of petri dish type, light, and filter paper composition on green foxtail radicle length and experiment variability using a solution of 0.3 ppm trifluralin (standard errors in parentheses).

	Plastic dish		Glass dish					
	Dark, Whatman #1		Dark, Glass fiber		Light, Whatman #1		Dark, Whatman#1	
Population	Length	CV[a]	Length	CV	Length	CV	Length	CV
	(% of control)[b]							
S1[c]	2(1)	21	1(1)	22	2(1)	22	1(1)	15
R1	110(6)	27	103(3)	18	95(4)	26	111(4)	18
R2	116(4)	21	102(4)	20	106(4)	24	107(3)	14
R3	101(6)	33	94(4)	24	90(5)	36	109(3)	18

[a]Coefficient of variation.

[b]Radicle length of the four populations for the control treatment ranged from 30 to 42 mm with the largest standard error being 3 mm.

[c]S = trifluralin-susceptible, R = trifluralin-resistant green foxtail populations; see Appendix Table 6 for the location of origin of each population.

was completely inhibited. At 0.6 ppm, radicle growth of R foxtail was partially inhibited, but not to the same extent as for the two S populations. The slight increase in radicle length of R foxtail, compared to the control, at solution concentrations of up to 0.3 ppm may not be biologically relevant. Rather, this may be caused by experimental error and the method of presentation (Brain and Cousens 1989).

Radicle growth of S and R biotypes was much more sensitive to ethalfluralin than to trifluralin. In the ethalfluralin dose-response experiment, the largest difference in radicle length between the R and S biotype occurred at concentrations of 0.05 to 0.1 ppm (Figure 5-2). At 0.05 ppm, radicle growth of the R biotype was not inhibited whereas radicle growth of the S biotype was greatly inhibited. At 0.1 ppm, radicle growth of the R biotype was slightly

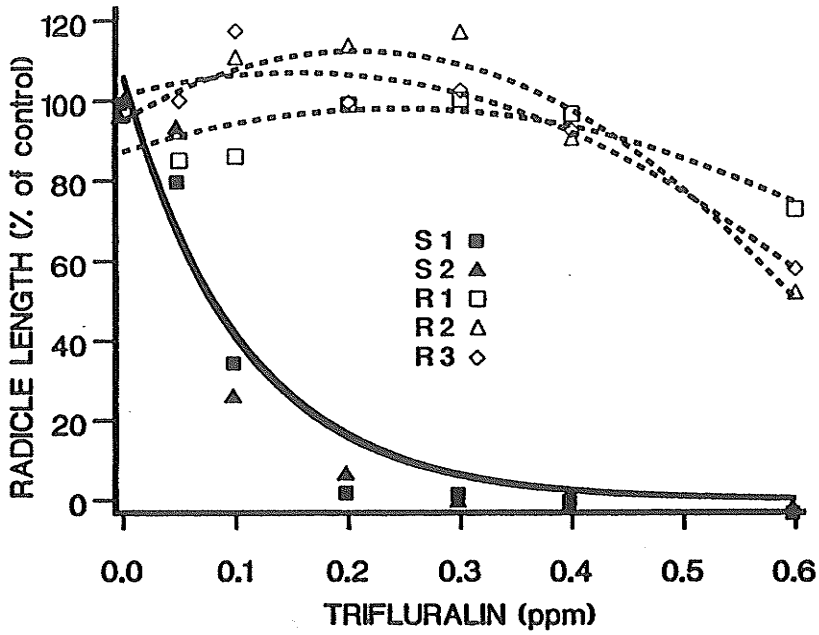


Figure 5-1. Radicle growth of three resistant (dashed lines) and two susceptible (solid lines) green foxtail populations as influenced by trifluralin concentration. See Table 5-2 for equations and parameter estimates.

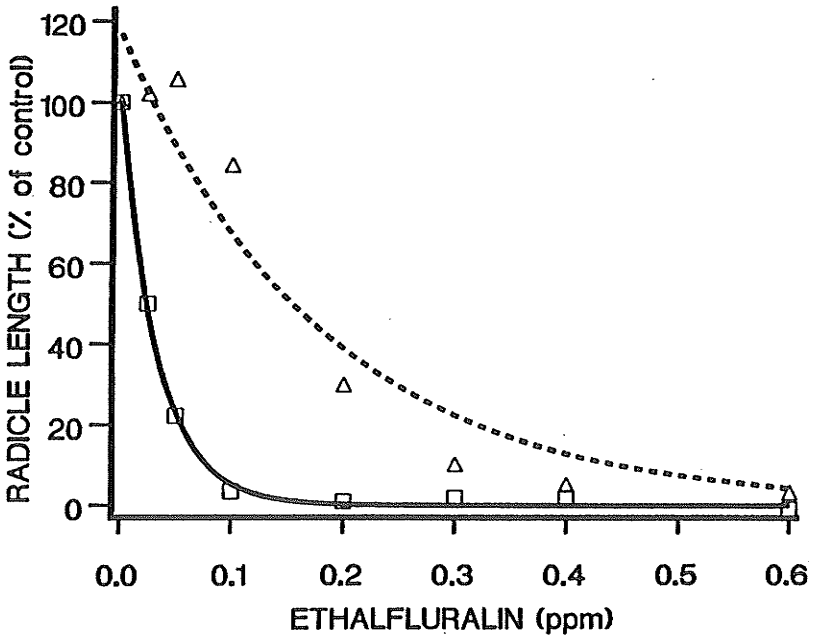


Figure 5-2. Radicle growth of a resistant (dashed line) and a susceptible (solid line) green foxtail biotype as influenced by ethalfluralin concentration. See Table 5-2 for equations and parameter estimates.

inhibited whereas radicle growth of the S biotype was completely inhibited. At 0.3 ppm, radicle growth of both biotypes were greatly inhibited. As computed from the ED_{50}[2] values, (Table 5-2) there was a nine-fold difference in response between S and R biotypes to trifluralin and a six-fold difference in response to ethalfluralin. These differences are comparable to those found in the growth chamber and field experiments where about a 7-fold difference in sensitivity to trifluralin and ethalfluralin was observed.

The largest difference in shoot length between the R and S biotypes occurred at 0.05 ppm trifluralin (Figure 5-3), although this difference was less

Table 5-2. Parameter estimates (standard errors in parentheses) and ED_{50}'s of the equations for the regression curves for the response of S and R green foxtail radicle growth to trifluralin and ethalfluralin (Figures 5-1 and 5-2).

Population	*a* [a]		*b*		*c*		R^{2b}	ED_{50}[c]	R/S
Trifluralin								ppm	
S1	105.5	(3.4)	-9.6	(0.8)			0.94	0.07	
S2	106.3	(4.8)	-9.3	(1.0)			0.89	0.07	
R1	87.2	(2.9)	85.9	(27.6)	-180.7	(45.2)	0.32	0.61	
R2	95.1	(3.6)	162.5	(32.9)	-396.6	(55.5)	0.66	0.63	9
R3	101.3	(3.7)	71.3	(32.7)	-241.0	(53.1)	0.58		
Ethalfluralin									
S1	100.5	(3.6)	-29.3	(2.3)			0.96	0.02	
R1	116.9	(4.7)	-5.5	(0.6)			0.92	0.13	6

[a]Exponential function equation: $y = a\ e^{bx}$ where a = intercept (% of control) and ab = initial slope; quadratic function equation: $y = a + bx + cx^2$ where a = intercept (% of control), b = linear coefficient, and c = curvilinear coefficient. In both equations, y is the length of the radicle (% of control) and x is the herbicide concentration (ppm).

[b]All coefficients of determination are significant at the 1% level.

[c]The value for R3 was beyond the highest concentration. For all populations for the control treatment, radicle length ranged from 28 to 36 mm with the largest standard error being 2 mm.

than that observed for radicle length at 0.2 to 0.4 ppm. At concentrations higher than 0.05 ppm, the differences in shoot length between R and S foxtail decreased sharply. At 0.2 ppm or more, shoot growth was greatly inhibited for both the R and S biotypes. The sensitivity of the coleoptilar node of green foxtail to trifluralin likely resulted in the greater sensitivity of shoot growth to the herbicide compared to the radicle (Rahman and Ashford 1970; Appleby and Valverde 1989). There was a much smaller difference in response between the S and R biotypes to ethalfluralin than to trifluralin (Figure 5-4). The largest difference in shoot length between the S and R biotypes occurred at 0.025 ppm. The difference in response between the biotypes to ethalfluralin was much less than for trifluralin-treated shoots at 0.05 ppm. Growth of both S and R biotypes was greatly inhibited at 0.1 ppm ethalfluralin. The maximum standard error of the means for radicles and shoots at all concentrations was 6%. The greater sensitivity of shoot as compared to radicle growth to these herbicides was reflected in lower R/S ratios (Table 5-3).

The results indicate that radicle length is the appropriate parameter to measure response to either trifluralin or ethalfluralin and to discriminate between R and S green foxtail biotypes. Shoot length is not a useful parameter as trifluralin and ethalfluralin inhibited shoot growth of both R and S biotypes at very low concentrations, which would greatly reduce the ability to distinguish between R and S biotypes because of larger experimental error (data not shown). In contrast, relatively large differences in radicle length existed between R and S biotypes over a range of trifluralin and ethalfluralin concentrations. Since these differences are largest when trifluralin is used, this herbicide should be used to best discriminate between R and S biotypes.

The trifluralin concentration resulting in the largest difference in radicle growth between R and S biotypes with the smallest variability in radicle length

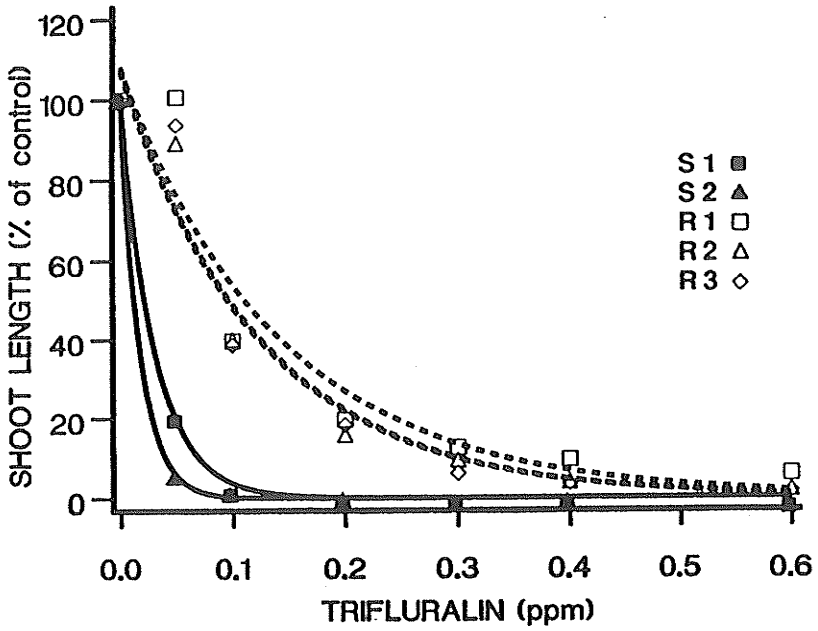


Figure 5-3. Shoot growth of three resistant (dashed lines) and two susceptible (solid lines) green foxtail populations as influenced by trifluralin concentration. See Table 5-3 for equations and parameter estimates.

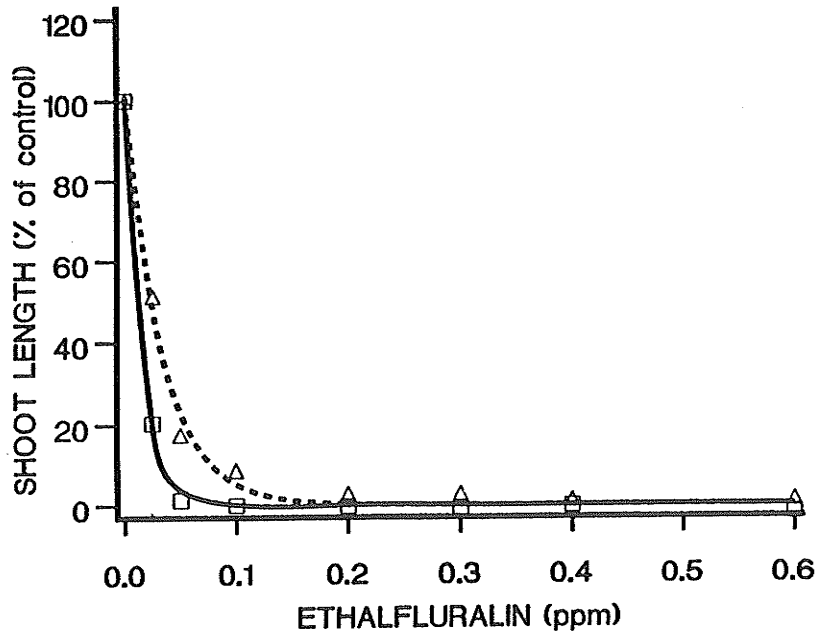


Figure 5-4. Shoot growth of a resistant (dashed line) and a susceptible (solid line) green foxtail biotype as influenced by ethalfluralin concentration. See Table 5-3 for equations and parameter estimates.

Table 5-3. Parameter estimates (standard errors in parentheses) of the equations for the regression curves for the response of S and R green foxtail shoot growth to trifluralin and ethalfluralin (Figures 5-3 and 5-4).

Population	a[a]	b	R^2[b]	ED_{50}	R/S
Trifluralin				ppm	
S1[c]	100.1 (1.2)	-33.0 (1.2)	0.99	0.02	
S2	100.0 (0.7)	-57.8 (2.4)	0.99	0.01	
R1	107.7 (4.1)	-7.0 (0.6)	0.90	0.10	
R2	106.9 (3.3)	-8.0 (0.5)	0.94	0.09	
R3	108.2 (4.4)	-7.9 (0.7)	0.90	0.09	6
Ethalfluralin					
S1	100.1 (0.6)	-65.6 (1.3)	0.99	0.01	
R1	100.6 (1.5)	-29.6 (1.0)	0.99	0.02	2

[a]Exponential function equation: $y = a\ e^{bx}$ where a = intercept (% of control) and ab = initial slope, y is the length of the shoot (% of control) and x is the herbicide concentration (ppm).

[b]All coefficients of determination are significant at the 1% level.

[c]For all populations for the control treatment, shoot length ranged from 54 to 59 mm with the largest standard error being 2 mm.

among and within replicates for each biotype was 0.3 ppm. Therefore, this concentration was selected for the reliable detection of R foxtail with the radicle as the test parameter, using glass petri dishes and two Whatman #1 filter papers and incubating in the dark for 5 d at 22 C (Plate 4).

Twelve green foxtail populations were randomly selected and tested with trifluralin to verify the accuracy of this procedure (Table 5-4 and Appendix Table 6). Radicle growth of eight populations was not inhibited when exposed to trifluralin, whereas complete inhibition of growth was observed for the other four populations. Identification of the R and S populations matched the growth chamber results of Morrison et al. (1989). These consistent results indicate that the petri dish bioassay can detect accurately and reliably R green foxtail.

Table 5-4. Radicle length of four susceptible (S1-S4) and eight resistant (R1-R8) green foxtail populations using a solution of 0.3 ppm trifluralin (standard errors in parentheses).

Population	Radicle length[a]
	(% of control)
S1	1 (1)
S2	1 (1)
S3	2 (1)
S4	4 (1)
R1	113 (3)
R2	111 (4)
R3	119 (4)
R4	119 (3)
R5	112 (3)
R6	109 (3)
R7	99 (3)
R8	110 (5)

[a]Radicle length for all the populations for the control treatment ranged from 26 to 38 mm, with the largest standard error being 2 mm; see Appendix Table 6 for location of origin of each population.

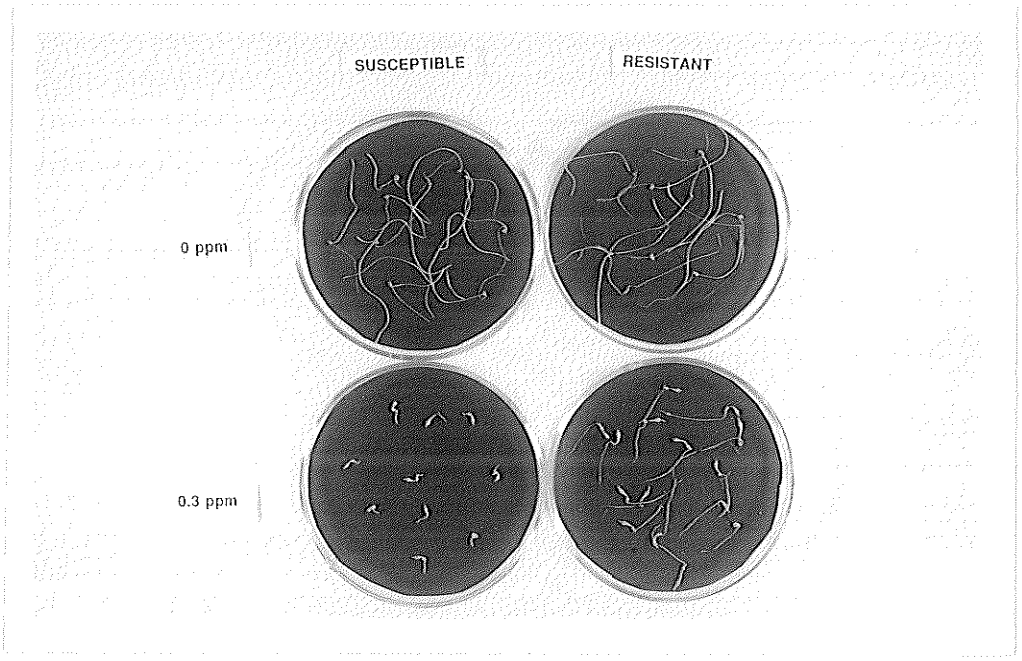
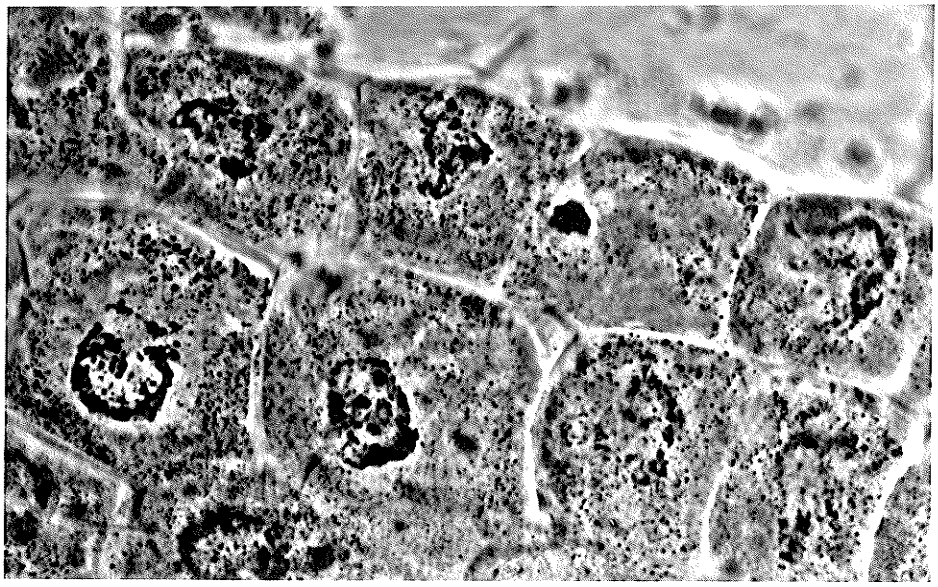


Plate 4. Petri dish bioassay showing the effects of 0.3 ppm trifluralin on radicle and shoot growth of susceptible and resistant green foxtail biotypes.

5.3.2. Mitotic index study

Aberrant mitotic figures were visible in S root tips treated with dinitroaniline herbicides at all concentrations and in R root tips, but only at relatively higher concentrations. Cells in condensed prophase were observed for S biotype root meristems treated with 0.1 ppm ethalfluralin (Plate 5), whereas R biotype root meristems treated with the same concentration showed only normal mitotic figures. The effect of trifluralin and ethalfluralin on the mitotic index of S and R green foxtail root meristems is shown in Figure 5-5. There is a substantial increase in the frequency of dividing cells observed for both biotypes over the concentration ranges of the herbicides. There are marked differences, however, in the response of the two biotypes to each herbicide and in the collective response of both biotypes to each herbicide. The S biotype was 10 times more sensitive to both trifluralin and ethalfluralin than the R biotype, as indicated by the ED_{50} ratio (Table 5-5). For S root meristems, the mitotic index increased by over 50% at 0.1 ppm compared to the untreated control, and had nearly plateaued by 0.2 ppm. In contrast, R root meristems treated with trifluralin showed a gradual increase in mitotic index over the concentration range. There were no differences in mitotic indices between S and R biotype root meristems only at the highest concentration of 1 ppm.

Ethalfluralin was more effective than trifluralin in increasing the mitotic index of both S and R biotypes. The mitotic index of S root meristems was increased by over 50% at 0.025 ppm. Little change in mitotic indices occurred at concentrations greater than 0.1 ppm. In contrast, the R biotype required approximately 0.2 ppm ethalfluralin to obtain a 50% rise in the mitotic index. At 0.3 ppm, there were no differences in mitotic indices between S and R biotype root meristems. The herbicide concentrations required to increase the mitotic



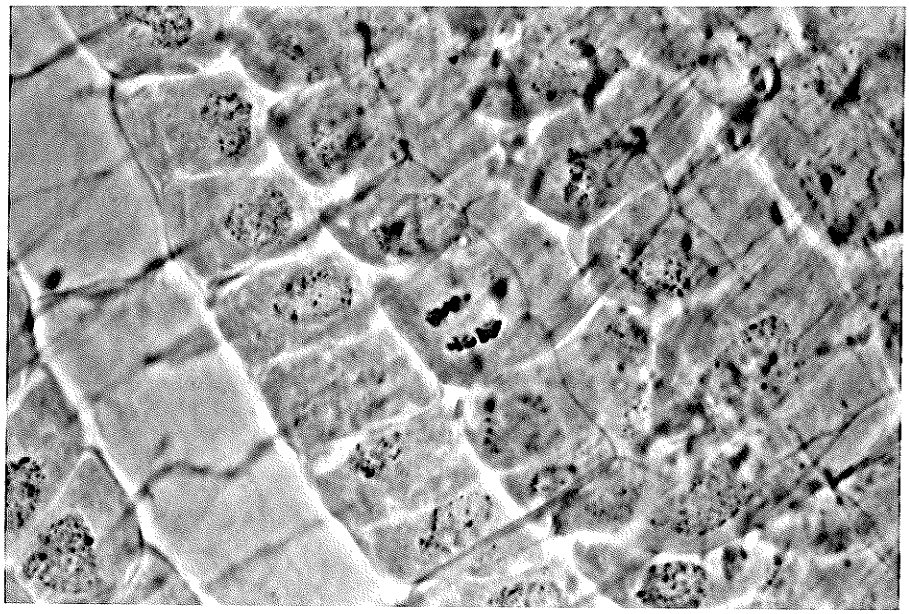


Plate 5. Photomicrograph (reverse phase) of a condensed prophase mitotic figure of a S biotype root meristem (top) and a telophase mitotic figure of a R biotype root meristem (bottom) treated with 0.1 ppm ethalfluralin (total magnification = 1250X).

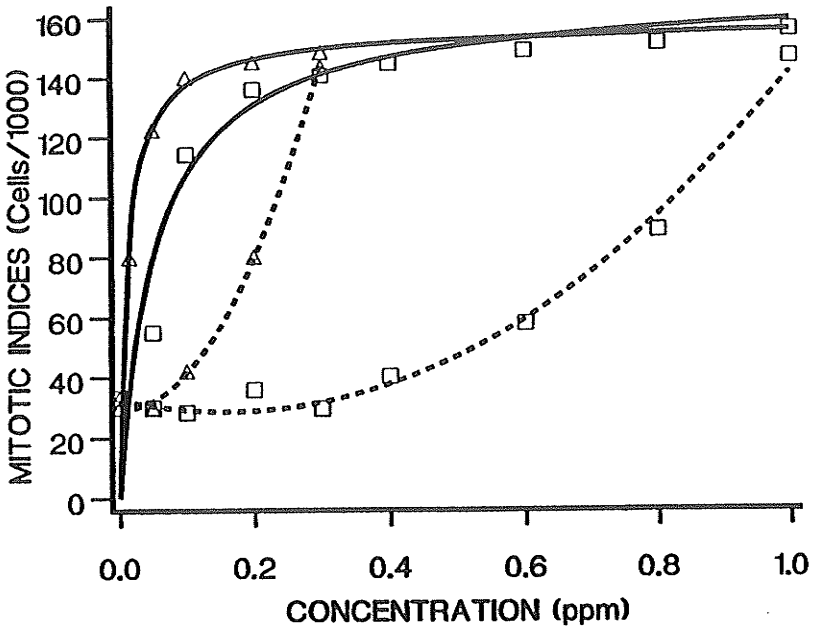


Figure 5-5. Mitotic index of S (solid lines) and R (dashed lines) green foxtail biotypes as influenced by trifluralin (square symbols) and ethalfluralin (triangle symbols). See Table 5-5 for equations and parameter estimates.

Table 5-5. Parameter estimates (standard errors in parentheses) of the equations for the regression curves for the dose-response experiment (Figure 5-5).

Biotype[a]	a[b]	b	c	R^{2c}	ED_{50}[d]	R/S
					ppm	
Trifluralin						
S	3030 (670)	166 (9)		0.82	0.08	
R	33 (2)	-51 (14)	158 (15)	0.92	0.78	10
Ethalfluralin						
S	11400 (5880)	156 (15)		0.78	0.02	
R	30 (4)	-13 (84)	1310 (290)	0.96	0.21	10

[a]See Appendix Table 6 for place of origin of the R and S biotypes.

[b]Quadratic function equation (R biotype): $y = a + bx + cx^2$ where a = intercept, b = linear coefficient, and c = curvilinear coefficient; rectangular hyperbolic function equation (S biotype): $y = (ax)/(1+((ax)/b))$ where a = initial slope and b = asymptote. In both equations, y is the mitotic index and x is the herbicide concentration (ppm).

[c]All coefficients of determination are significant at the 1% level.

[d]Herbicide concentration required to increase the mitotic index by 50%.

index of S and R green foxtail correspond well with those required to inhibit radicle growth (Beckie et al. 1990).

This study supports the findings of other workers who have concluded that the mode of action of dinitroaniline herbicides is the inhibition of MT polymerization (Lignowski and Scott 1972; Bartels and Hilton 1973). In root meristems of S foxtail treated with these two dinitroaniline herbicides, only prophase and condensed prophase mitotic figures were observed. These are the only mitotic stages not requiring spindle MTs. This study again verifies that the R biotype is resistant to trifluralin and ethalfluralin, in agreement with the findings obtained from whole-plant level studies. The level of resistance in R green foxtail is not much higher when measured at the cell level than at the whole-plant level under controlled environmental conditions. This study has shown that changes in the mitotic indices of S and R green foxtail root meristems in response to varying concentrations of dinitroaniline herbicides can be used to determine the expression of resistance in green foxtail at the site of herbicide activity on an individual plant basis.

Identification of R green foxtail biotypes using the petri dish bioassay will assist in determining the nature and extent of the resistance problem in the northern Great Plains and the Canadian prairies. Green foxtail seed samples can be screened rapidly and reliably to detect R populations. Producers can be informed of a potential problem in a timely fashion and can undertake effective weed management practices to control R green foxtail.

6. SUMMARY AND CONCLUSIONS

The response of susceptible (S) and resistant (R) green foxtail to dinitroaniline herbicides and to herbicides belonging to other chemical families was compared under controlled environmental conditions. Their response to two dinitroanilines (trifluralin and ethalfluralin) commonly used on the prairies was also investigated under field conditions. Furthermore, the response of S and R green foxtail to these two herbicides was compared in a petri dish bioassay and mitotic index study. These studies were performed under diverse experimental conditions with many different herbicides, thereby contributing to a more complete description of the expression of resistance in green foxtail.

The response of S and R green foxtail to varying dosages of PPI- and PEI-trifluralin under field conditions verified the growth chamber results of Morrison et al. (1989). The level of resistance of R foxtail 4 wk after emergence, to PPI trifluralin (7-fold) and PEI trifluralin (12-fold) under either cropped or non-cropped conditions, was similar to the results of the growth chamber studies. Therefore, even though climate and soil factors can influence the effectiveness of the herbicide in controlling green foxtail, as well as the growth and competitiveness of both the weed and the crop, the level of resistance in R foxtail to trifluralin was very similar in both studies. The method of trifluralin placement (PPI or PEI treatment), which affects the spatial herbicide concentration in the soil, altered the expression of resistance in green foxtail.

The response of R foxtail seed production to PPI- and PEI-trifluralin under cropped conditions indicated that the degree of resistance was similar to determinations earlier in the season. However, the effective kill (seed yield reduction) differed from the initial reductions in density or biomass. Only the initial weed control, though, is usually measured by weed scientists. When

trifluralin was applied as a PPI treatment in rapeseed, the initial weed control (as measured by density and shoot dry matter reductions) at the recommended dosage did not exceed the effective kill. However, when trifluralin was applied as a PEI treatment in wheat the effective kill was somewhat less than the initial knockdown. For less persistent herbicides, it would be expected that the effective kill would be markedly less than the initial reductions in density or biomass because of germination of weed seed after the herbicide is dissipated, as well as growth compensation of surviving plants after herbicide thinning.

The initial reductions in density and biomass as well as the effective kill of R green foxtail at the recommended dosage of PPI trifluralin in rapeseed were similar at both Portage la Prairie and Deloraine, Manitoba. This is a significant finding since there are differences in climate (temperature and precipitation), soils (principally organic matter content), and nature of green foxtail infestation (sown versus naturally occurring) between the two locations. Consequently, differences in the growth and competitiveness of both green foxtail and the crop, as well as trifluralin dissipation and activity in the soil, could be expected. The similar effective kill of R green foxtail at these two locations provides greater confidence in the usefulness of the data for estimating the true selection pressure of the herbicide on foxtail. More such field data is needed to determine the true selection pressure of other herbicides on weed species to better understand the population dynamics of R weed biotypes.

From the effective kill results, the selection pressure of PPI trifluralin on foxtail in rapeseed (58) was calculated to be twice as high as PEI trifluralin on foxtail in wheat (29). Since the selection pressure is the most important parameter in population models for predicting the rate of evolution of herbicide-resistant biotypes, one would expect a more rapid rise in the proportion of R

individuals in a foxtail population when trifluralin is repeatedly applied as a PPI treatment in rapeseed than when applied as a PEI treatment in wheat.

These different rates of development of resistance were demonstrated by model simulations, which predicted the population dynamics of R green foxtail under cropped conditions using the experiment values for selection pressure plus known or assumed values for the other parameters. The model results indicated that herbicide rotations will not provide a real added delay in the rate of appearance of R green foxtail, except for the number of seasons that trifluralin is not used. The only practical solution is to use trifluralin less frequently in the cropping system to lessen the selection pressure and to delay the rate of evolution of resistance. The model simulations could potentially assist producers in designing herbicide rotation programs that would permit trifluralin to be used sparingly, while extending the useful lifetime of the herbicide in effectively controlling green foxtail.

R green foxtail is not only trifluralin-resistant, but dinitroaniline-resistant as well. The level of resistance of R foxtail to the other dinitroanilines is slightly less than for trifluralin, which had originally selected the biotype in the field. Such cross resistance to chemically similar herbicides with the same mechanism of action was not unexpected, although the degree of resistance was unpredictable. Of even greater significance was green foxtail resistance to dithiopyr, a chemically-unrelated mitotic disrupter herbicide with a different specific site of action. Resistance to this herbicide and to DCPA may indicate that the mechanism of resistance in green foxtail involves an alteration in a microtubule-associated protein (Smeda et al. 1991). R green foxtail was not resistant to nine other herbicides belonging to seven chemical families. Therefore, since most of these herbicides are registered to control or suppress green foxtail in cereal, oilseed, or other crops in Manitoba, the producer has a

sufficient arsenal of different herbicides with different sites of action to effectively control R green foxtail, if used in conjunction with good agronomic practices to combat resistance, such as well-planned herbicide and crop rotations. This will reduce any adverse effects of R foxtail on crop production in western Canada.

The R biotype was cross-resistant to ethalfluralin under both cropped and non-cropped field conditions. The difference in response between R and S foxtail 4 wk after emergence (7-fold), was similar to that observed under growth chamber conditions. A similar level of resistance was also determined from green foxtail seed return at the end of the growing season. The effective kill exceeded the initial weed control, similar to the results of the PPI trifluralin experiment. However, the selection pressure of ethalfluralin on green foxtail in rapeseed (45) was somewhat less than trifluralin (58), since the former herbicide reduced seed production of R green foxtail more effectively than trifluralin. Despite the lower selection pressure, ethalfluralin will still not be a suitable alternative herbicide for combatting R green foxtail. Cross resistance of R green foxtail to ethalfluralin must be taken into account when designing a herbicide rotation program, since it will also select for the R biotype.

A simple and inexpensive petri dish bioassay was proven to accurately detect R green foxtail. The assay is currently used commercially to test seed stocks for resistance. The identification of R green foxtail will assist in determining the nature and extent of the resistance problem on the Canadian prairies. R foxtail have been discovered not only in Manitoba, but in Saskatchewan and Alberta as well. Since green foxtail seed samples can now be screened rapidly, producers can be quickly notified of a potential problem and can undertake effective weed management practices to control R green foxtail.

Resistance of green foxtail to trifluralin and ethalfluralin was demonstrated at the site of herbicide action. As determined from mitotic indices of treated and untreated root tips, the R biotype was 10 times more resistant than the S biotype to both trifluralin and ethalfluralin, even though both biotypes were much more sensitive to ethalfluralin. The expression of resistance at the cell level is not markedly different from determinations under controlled environmental or field conditions. Mitotic indices provide an additional tool to verify and quantify resistance to mitotic disrupter herbicides on an individual plant basis.

These studies, consisting of both basic and applied research, will be of practical benefit to both producers and research and extension workers. The simple test to detect R foxtail will facilitate rapid screening of seed samples for resistance so that problem fields can be quickly identified. Combined with information on alternative herbicides that effectively control R foxtail, producers can effectively manage R foxtail. However in the longer term producers must employ suitable agronomic practices, particularly crop/herbicide rotations, to effectively delay or even preclude resistance. The results of the field experiments, in conjunction with scenarios generated from the population models, clearly indicate that long residual herbicides with a high effective kill, such as trifluralin or ethalfluralin, must be used less frequently in the herbicide rotation in order to lessen the selection pressure. These studies indicate that unlike short residual herbicides, the effective kill of trifluralin and ethalfluralin is not markedly less and may even exceed the initial reductions in density or biomass, due to season-long persistence of these herbicides in soil. The response of R foxtail seed return to trifluralin and ethalfuralin under cropped conditions indicates that the expression of resistance does not decline over the growing season.

Herbicide resistance in weed species is a relatively new and serious problem facing producers and weed scientists. Continued basic and applied research will be needed to contain the problem within tolerable limits, so that R weed biotypes do not seriously threaten future crop production. Of particular importance is research directed at quantifying those factors that affect the population dynamics of R weeds, such as selection pressure and ecological fitness. This would greatly increase the predictive value of population models in describing the evolution and dynamics of R biotypes. Research focusing on the breeding system, mode of inheritance of resistance, and relative fitness of R green foxtail, combined with the field data on the selection pressure of trifluralin and ethalfluralin, will inevitably result in more accurate model simulations of the dynamics of R populations. This, in turn, will facilitate the formulation of management strategies, such as detailed crop/herbicide rotations, to maintain the proportion of R foxtail in the field population at a low level. Another management strategy that must be seriously examined is to lower the selection pressure, not only by reducing the frequency of herbicide application, but also the dosage of application. Such an approach would allow sufficient S plants to mature and produce seed before the end of the growing season, yet still maintain cost-effective weed control. Adoption of this practice would require more research on economic weed competition thresholds. If these management strategies are not adopted and current monoherbicide/ monoculture farming practices are continued, the future usefulness of currently available herbicides will be jeapordized along with the sustainability of crop production on the Canadian prairies.

LITERATURE CITED

Alex, J. F., J. D. Banting, and J. P. Gebhardt. 1972. Distribution of *Setaria viridis* in western Canada. Can. J. Plant Sci. 52:129-138.

Appleby, A. P., and B. E. Valverde. 1989. Behavior of dinitroaniline herbicides in plants. Weed Technol. 3:198-206.

Armbruster, B. L., W. T. Molin, and M. W. Bugg. 1991. Effects of the herbicide dithiopyr on cell division in wheat root tips. Pestic. Biochem. Physiol. 39:110-120.

Arntzen, C. J., K. Pfister, and K. E. Steinback. 1982. The mechanism of chloroplast triazine resistance: alterations in the site of herbicide action. p. 185-214 *in* H. M. LeBaron and J. Gressel, eds. Herbicide Resistance in Plants. John Wiley and Sons, Inc., New York.

Ashford, R., R. B. McKercher, and F. A. Holm. 1987. Trifluralin. *in* W. W. Donald, ed. Systems of Weed Control in Wheat in North America. Cpt. 17. Weed Sci. Soc. Am., Champaigne, IL.

Ashton, F. M., and A. S. Crafts. 1981. Mode of Action of Herbicides. John Wiley & Sons, New York.

Banting, J. D., E. S. Molberg, and J. P. Gebhardt. 1973. Seasonal emergence and persistence of green foxtail. Can. J. Plant Sci. 53:369-376.

Barrentine, W. L., and G. F. Warren. 1971. Shoot zone activity of trifluralin and nitralin. Weed Sci. 19:37-41.

Bartels, P. G., and J. L. Hilton. 1973. Comparison of trifluralin, oryzalin, pronamide, propham, and colchicine treatments on microtubules. Pestic. Biochem. Physiol. 3:462-472.

Beckie, H. J., L. F. Friesen, K. M. Nawolsky, and I. N. Morrison. 1990. A rapid bioassay to detect trifluralin-resistant green foxtail (*Setaria viridis*). Weed Technol. 4:505-508.

Blackman, G. E. 1950. Selective toxicity and the development of selective weedkillers. J. Roy. Soc. Arts. 98:499-517.

Blackshaw, R. E., E. H. Stobbe, C. F. Shaykewich, and W. Woodbury. 1981a. Influence of soil temperature and soil moisture on green foxtail (*Setaria viridis*) establishment in wheat (*Triticum aestivum*). Weed Sci. 29:179-184.

Blackshaw, R. E., E. H. Stobbe, and A. R. W. Sturko. 1981b. Effect of seeding dates and densities of green foxtail (*Setaria viridis*) on the growth and productivity of spring wheat (*Triticum aestivum*). Weed Sci. 29:212-217.

Bode, L. E., C. L. Day, M. L. Gebhardt, and C. E. Goering. 1973. Prediction of trifluralin diffusion coefficients. Weed Sci. 21:485-489.

Brain, P., and R. Cousens. 1989. An equation to describe dose responses where there is stimulation of growth at low doses. Weed Res. 29:93-96.

Bubar, C. J., and I. N. Morrison. 1984. Growth responses of green and yellow foxtail (*Setaria viridis* and *S. lutescens*) to shade. Weed Sci. 32:774-780.

Chaleff, R. S., and C. J. Mauvais. 1984. Acetolactate synthase is the site of action of two sulfonylurea herbicides in higher plants. Science 224:1443-1445.

Chow, P. N. P. 1976. Dinitroaniline herbicides for grassy weed control in rapeseed. Can. J. Plant Sci. 6:705-713.

Clayton, L. 1985. The cytoskeleton and the plant cell cycle. p. 113-131 *in* J. A. Bryant and D. Francis, eds. The Cell Division Cycle in Plants. Cambridge University Press, Cambridge.

Cousens, R. 1985. A simple model relating yield loss to weed density. Ann. Appl. Biol. 107:239-252.

Darwent, A. L. 1980. Effects of soil temperature on the phytotoxicity of trifluralin to wild oats. Can. J. Plant Sci. 60:929-938.

Dawson, J. H., and V. F. Bruns. 1962. Emergence of barnyardgrass, green foxtail and yellow foxtail seedlings from various soil depths. Weeds 10: 136-139.

Derr, J. F., and T. J. Monaco. 1982. Ethalfluralin activity in cucumber. Weed Sci. 30:498-502.

Douglas, B. J., A. G. Thomas, I. N. Morrison, and M. G. Maw. 1985. The biology of canadian weeds. 70. *Setaria viridis* (L.) Beauv. Can. J. Plant Sci. 65:669-690.

Dryden, R. D., and C. W. Whitehead. 1963. The effect of TCA on green foxtail in competition with cereals. Can. J. Plant Sci. 43:451-456.

Duesing, J. 1983. Genetic analysis of herbicide resistance. Proc. North Cent. Weed Control Conf. p. 143-147, Columbus, Ohio.

Ezra, G., J. H. Dekker, and G. R. Stephenson. 1985. Tridiphane as a synergist for herbicides in corn (*Zea mays*) and proso millet (*Panicum milliaceum*). Weed Sci. 33:287-290.

Freund, R. J., and R. C. Littell. 1986. SAS system for regression. SAS Inst. Inc., Cary, NC, p. 133-141.

Friesen, H. A., and K. E. Bowren. 1973. Factors affecting the control of wild oats in rapeseed with trifluralin. Can. J. Plant Sci. 53:199-205.

Fuerst, E. P., and K. C. Vaughn. 1990. Mechanisms of paraquat resistance. Weed Technol. 4:150-156.

Gasquez, J., and J. P. Compoint. 1981. Observation de chloroplastes résistants aux triazines chez une panicoidée. *Setaria viridis* L. Agronomie 1:923-926.

Gaynor, J. D. 1985. Dinitroaniline herbicide persistence in soil in southern Ontario. Can. J. Soil Sci. 65:587-592.

Golab, T., W. A. Althaus, and H. L. Wooten. 1979. Fate of [^{14}C] trifluralin in soil. J. Agric. Food Chem. 27:163-179.

Golab, T., R. J. Herberg, S. J. Parka, and J. B. Tepe. 1967. Metabolism of carbon-14 trifluralin in carrots. J. Agric. Food Chem. 15:638-641.

Gomez, A. K., and A. A. Gomez. 1984. Chi-square test. p. 458-477 *in* Statistical Prodedures for Agricultural Research. John Wiley and Sons, Toronto.

Gressel, J. 1979. Genetic herbicide resistance: projections on appearance in weeds and breeding for it in crops. *in* T. K. Scott, ed. Plant Regulation and World Agriculture. Plenum Press, New York.

Gressel, J. 1985. Herbicide tolerance and resistance: alteration of site of activity. p. 159-189 *in* S. O. Duke, ed. Weed Physiology. CRC Press, Boca Raton, FL.

Gressel, J. 1986. Modes and genetics of herbicide resistance in plants. p. 54-73 *in* E. H. Glass, ed. Pesticide Resistance: Strategies and Tactics for Management. National Academy Press, Washington, D. C.

Gressel, J. 1987. Appearance of single and multi-group herbicide resistances, and strategies for their prevention. Br. Crop Prot. Conf.-Weeds 2: 479-488.

Gressel, J., and L. A. Segel. 1978. The paucity of plants evolving genetic resistance to herbicides: possible reasons and implications. J. Theor. Biol. 75: 349-371.

Gressel, J., and L. A. Segel. 1982. Interrelating factors controlling the rate of appearance of resistance: the outlook for the future. p. 325-347 *in* H. M. LeBaron and J. Gressel, eds. Herbicide Resistance in Plants. John Wiley and Sons, Inc., New York.

Gressel, J., and L. A. Segel. 1990a. Modelling the effectiveness of herbicide rotations and mixtures as strategies to delay or preclude resistance. Weed Technol. 4:186-198.

Gressel, J., and L. A. Segel. 1990b. Herbicide rotations and mixtures: effective strategies to delay resistance. p. 430-458 *in* M. B. Green, H. M. LeBaron, and W. K. Moberg, eds. Managing Resistance to Agrochemicals: From Fundamental Research to Practical Strategies. ACS Symp. Ser. 421, ACS, Washington, DC.

Gronwald, J. W., R. N. Andersen, and C. Yee. 1989. Atrazine resistance in velvetleaf (*Abutilon theophrasti*) due to enhanced atrazine detoxification. Pestic. Biochem. Physiol. 34:149-163.

Grover, R. 1974. Adsorption and desorption of trifluralin, triallate, and diallate by various adsorbents. Weed Sci. 22:405-408.

Grover, R., J. D. Banting, and P. M. Morse. 1979. Adsorption and bioactivity of diallate, triallate and trifluralin. Weed Res. 19:363-369.

Grover, R., A. E. Smith, S. R. Shewchuk, A. J. Cessna, and J. H. Hunter. 1988. Fate of trifluralin and triallate applied as a mixture to a wheat field. J. Environ. Qual. 17:543-550.

Gunning, B. E., and A. R. Hardham. 1979. Microtubules and morphogenesis in plants. Endeavour 3:112-117.

Haas, H., and J. C. Streibig. 1982. Changing patterns of weed distribution as a result of herbicide use and other agronomic factors. p. 57-80

in H. M. LeBaron and J. Gressel, eds. Herbicide Resistance in Plants. John Wiley and Sons, Inc., New York.

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

Harper, L. A., A. W. White, R. R. Bruce, A. W. Thomas, and R. A. Leonard. 1976. Soil and microclimate effects on trifluralin volatilization. J. Environ. Qual. 5:236-242.

Harvey, R. G. 1974. Soil adsorption and volatility of dinitroaniline herbicides. Weed Sci. 22:120-124.

Hayden, B. J., and A. E. Smith. 1980. Comparison of the persistence of ethalfluralin and trifluralin in Saskatchewan field soils. Bull. Environ. Contam. Toxicol. 25:508-511.

Heap, I., and R. Knight. 1986. The occurrence of herbicide cross-resistance in a population of annual ryegrass (*L. rigidum*). Aust. J. Agric. Res. 37:149-156.

Heap, I., and I. N. Morrison. 1991. Herbicide-resistant weeds. Manitoba Agriculture Agdex No. 818, Winnipeg, MB.

Heath, M. C., R. B. McKercher, and R. Ashford. 1985. Influence of high soil temperature, ammonium ion, and rapeseed residue on trifluralin phytotoxicity to wheat. Can. J. Plant Sci. 65:151-161.

Helling, C. S. 1976. Dinitroaniline herbicides in soils. J. Environ. Qual. 5:1-15.

Hess, F. D. 1987. Herbicide effects on the cell cycle of meristematic plant cells. p. 183-203 *in* C. L. Foy, ed. Rev. Weed Sci., V. 3. Weed Sci. Soc. Am., Champaign, IL.

Hess, F. D. 1989. Herbicide interference with cell division in plants. p. 85-104 *in* P. Boger and G. Sandmann, eds. Target Sites of Herbicide Action. CRC Press, Inc., Boca Raton, FL.

Hill, R. J. 1982. Taxonomy and biological considerations of herbicide-resistant and herbicide-tolerant biotypes. p. 81-98 *in* H. M. LeBaron and J. Gressel, eds. Herbicide Resistance in Plants. John Wiley and Sons, Inc., New York.

Hilton, J. L., and M. N. Christiansen. 1972. Lipid contribution to selective action of trifluralin. Weed Sci. 20:290-294.

Hoechst. 1989. Western Canada's worst weed: green foxtail. Hoechst Canada Inc. publ. , Regina, SK.

Holliday, R. J., P. D. Putwain, and A. Dafni. 1976. The evolution of herbicide resistance in weeds and its implications for the farmer. Proc. Br. Crop Prot. Conf.- Weeds:937-946.

Holmsen, J. D., and F. D. Hess. 1984. Growth inhibition and disruption of mitosis by DCPA in oat (*Avena sativa*) roots. Weed Sci. 32:732-738.

Holt, J. S. 1990. Fitness and ecological adaptability of herbicide-resistant biotypes. p. 419-429 *in* M. B. Green, H. M. LeBaron, and W. K. Moberg, eds. Managing Resistance to Agrochemicals: From Fundamental Research to Practical Strategies. ACS Symp. Ser. 421, ACS, Washington, DC.

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

Horowitz, M., N. Hulin, and T. Blumenfeld. 1974. Behaviour and persistence of trifluralin in soil. Weed Res. 14:213-220.

Hume, L., and O. W. Archibold. 1986. The influence of a weedy habitat on the seed bank of an adjacent cultivated field. Can. J. Bot. 64:1879-1883.

Jacques, G. L., and R. G. Harvey. 1974. A simple bioassay technique for dinitroaniline herbicides in soils. Proc. North Cent. Weed Control Conf. 29:93.

Jacques, G. L., and R. G. Harvey. 1979a. Vapor absorption and translocation of dinitroaniline herbicides in oats (*Avena sativa*) and peas (*Pisum sativum*). Weed Sci. 27:371-374.

Jacques, G. L., and R. G. Harvey. 1979b. Dinitroaniline herbicide phytotoxicity as influenced by soil moisture and herbicide vaporization. Weed Sci. 27:536-539.

Jansen, M. A. K., J. H. Hobe, J. C. Wesselius, and J. J. S. van Rensen. 1986. Comparison of photosynthetic activity and growth performance in triazine-resistant and -susceptible biotypes of *Chenopodium album.* Physiol. Vegetale 24:475-484.

Kemp, M. S., and J. C. Casely. 1987. Synergistic effects of 1-aminobenzotriazole on the phytotoxicity of chlorotoluron and isoproturon in a resistant population of blackgrass (*Alopecuris myosuroides*). Br. Crop Prot. Conf.-Weeds 3:895-899.

Kemp, M. S., and J. C. Casely. 1989. Synergists to combat herbicide resistance. *in* G. Cussans and J. C. Casely, eds. Herbicide Resistance in Weeds and Crops. Abstracts of papers presented at the 11th Long Ashton Int. Symp., Bristol, U.K.

Kemp, M. S., S. R. Moss, and T. H. Thomas. 1990. Herbicide resistance in *Alopecurus myosuroides.* p. 376-393 *in* M. B. Green, H. M. LeBaron, and W. K. Moberg, eds. Managing Resistance to Agrochemicals: From Fundamental Research to Practical Strategies. ACS Symp. Ser. 421, ACS, Washington, DC.

Knake, E. L., A. P. Appleby, and W. R. Furtick. 1967. Soil incorporation and site of uptake of preemergence herbicides. Weeds 15:228-232.

Kvalseth, T. O. 1985. Cautionary note about R^2. Am. Stat. 39:279-285.

Lamoureux, G. L. , and D. G. Rusness. 1986. Tridiphane [2-(3,5-dichlorophenyl)-2-(2,2,2-trichloroethyl)oxirane] an atrazine synergist: enzymatic conversion to a potent glutathione S-transferase inhibitor. Pestic. Biochem. Physiol. 26:323-342.

Lavy, T. L., and P. W. Santelmann. 1986. Herbicide bioassay as a research tool. p. 201-217 *in* N. D. Camper, ed. Research Methods in Weed Science. South. Weed Sci. Soc., Champaign, IL.

LeBaron, H. M. 1989. Distribution and seriousness of herbicide resistant weed infestations worldwide. *in* G. Cussans and J. C. Casely, eds. Herbicide Resistance in Weeds and Crops. Abstracts of papers presented at the 11th Long Ashton Int. Symp., Bristol, U.K.

LeBaron, H. M., and J. Gressel. 1982. Herbicide Resistance in Plants. John Wiley and Sons, Inc., New York.

LeBaron, H. M., and J. McFarland. 1990. Overview and prognosis of herbicide resistance in weeds and crops. p. 336-352 *in* M. B. Green, H. M. LeBaron, and W. K. Moberg, eds. Managing Resistance to Agrochemicals: From Fundamental Research to Practical Strategies. ACS Symp. Ser. 421, ACS, Washington, DC.

Lee, S. M., and P. B. Cavers. 1981. The effects of shade on growth, development, and resource allocation patterns of three species of foxtail (*Setaria*). Can. J. Bot. 59:1776-1786.

Lehnen Jr., L. P., and K. C. Vaughn. 1991. Immunofluorescence and electron microscopic investigations of the effects of dithiopyr on onion root tips. Pestic. Biochem Physiol. 40:58-67.

Lignowski, E. M., and E. G. Scott. 1972. Effect of trifluralin on mitosis. Weed Sci. 20:267-270.

Marquis, L. Y., R. H. Shimabukuro, G. E. Stolzenberg, V. J. Feil, and R. G. Zaylskie. 1979. Metabolism and selectivity of fluchloralin in soybean roots. J. Agric. Food Chem. 27:1148-1156.

Maurice, D. C., and I. N. Morrison. 1983. How to control green foxtail. Manitoba Agriculture: Agdex No. 642., Winnipeg, MB.

Maxwell, B. D., M. L. Roush, and S. R. Radosevich. 1990. Predicting the evolution and dynamics of herbicide resistance in weed populations. Weed Technol. 4:2-13.

Menges, R. M., and J. L. Hubbard. 1970. Phytotoxicity of bensulide and trifluralin in several soils. Weed Sci. 18:244-247.

Milthorpe, F. L., and J. Moorby. 1974. An Introduction to Crop Physiology. Cambridge University Press, London, p. 225.

Morejohn, L. C., T. E. Bureau, J. Mole-Bajer, A. S. Bajer, and D. E. Fosket. 1987. Oryzalin, a dinitroaniline herbicide, binds to plant tubulin and inhibits microtubule polymerization in vitro. Planta 172:252-264.

Morrison, I. N., H. J. Beckie, and K. M. Nawolsky. 1990. The occurrence of trifluralin resistant green foxtail (*Setaria viridis*) in western Canada. *in* G. Cussans and J. C. Casely, eds. Herbicide Resistance in Weeds and Crops. Abstracts of papers presented at the 11th Long Ashton Int. Symp., Bristol, U.K.

Morrison, I. N., K. M. Nawolsky, M. H. Entz, and A. E. Smith. 1991. Differences among certified wheat seedlots in response to trifluralin. Agron. J 83:119-123.

Morrison, I. N., B. G. Todd, and K. M. Nawolsky. 1989. Confirmation of trifluralin-resistant green foxtail (*Setaria viridis*) in Manitoba. Weed Technol. 3:544-551.

Moss, S. R. 1987. Herbicide resistance in blackgrass (*Alopecurus myosuroides*). Br. Crop Prot. Conf.-Weeds 8C:879-886.

Moss, S. R., and G. W. Cussans. 1989. The development of herbicide-resistant populations of *Alopecurus myosuroides* (black-grass) in England. *in* G. Cussans and J. C. Casely, eds. Herbicide Resistance in Weeds and Crops. Abstracts of papers presented at the 11th Long Ashton Int. Symp., Bristol, U.K.

Moyer, J. R. 1979. Soil organic matter, moisture, and temperature: effect on wild oats control with trifluralin. Can. J. Plant Sci. 59:763-768.

Mudge, L. C., B. J. Gossett, and T. R. Murphy. 1984. Resistance of goosegrass (*Eleusine indica*) to dinitroaniline herbicides. Weed Sci. 32:591-594.

Mulder, C. E. G., and J. D. Nalewaja. 1978. Temperature effect of phytotoxicity of soil-applied herbicides. Weed Sci. 26:566-570.

Murphy, T. R., B. J. Gossett, and J. E. Toler. 1986. Growth and development of dinitroaniline-susceptible and -resistant goosegrass (*Eleusine indica*) under noncompetitive conditions. Weed Sci. 34:704-710.

Nosticzius, A., T. Muller, and G. Czimber. 1979. The distribution of *Amaranthus retroflexus* in monoculture cornfields and its herbicide resistance. Bot. Kozl. 66:299.

Parka, S. J., and O. F. Soper. 1977. The physiology and mode of action of the dinitroaniline herbicides. Weed Sci. 25:79-87.

Parochetti, J. V., M. G. Schnappinger, G. F. Ryan, and H. A. Collins. 1982. Practical significance and means of control of herbicide-resistant weeds. p. 309-323 *in* H. M. LeBaron and J. Gressel, eds. Herbicide Resistance in Plants. John Wiley and Sons, Inc., New York.

Parr, J. F., and S. Smith. 1973. Degradation of trifluralin under lab conditions and soil anaerobiosis. Soil Sci. 115:55-63.

Peter, C. J., and J. B. Weber. 1985. Adsorption and efficacy of trifluralin and butralin as influenced by soil properties. Weed Sci. 33:861-867.

Powles, S. B., J. A. M. Holtum, J. M. Matthews, and D. R. Liljegren. 1990. Herbicide cross-resistance in annual ryegrass (*Lolium rigidum Gaud*). p. 394-406 *in* M. B. Green, H. M. LeBaron, and W. K. Moberg, eds. Managing Resistance to Agrochemicals: From Fundamental Research to Practical Strategies. ACS Symp. Ser. 421, ACS, Washington, DC.

Powles, S. B., and P. D. Howat. 1990. Herbicide-resistant weeds in Australia. Weed Technol. 4:178-185.

Pritchard, M. K., and E. H. Stobbe. 1980. Persistence and phytotoxicity of dinitroaniline herbicides in Manitoba soils. Can. J. Plant Sci. 60: 5-11.

Probst, G. W., T. Golab, R. J. Herberg, F. J. Holzer, S. J. Parka, C. Van Der Schans, and J. B. Tepe. 1967. Fate of trifluralin in soils and plants. J. Agric. Food Chem. 15:592-599.

Putwain, P. D. 1982. Herbicide resistance in weeds-an inevitable consequence of herbicide use? Proc. Br. Crop Prot. Conf. -Weeds:719-728.

Radosevich, S. R. 1983. Herbicide resistance in higher plants. p. 453-479 *in* G. P. Georghiou and T. Saito, eds. Pest Resistance to Pesticides. Plenum Press, New York.

Radosevich, S. R., and A. P. Appleby. 1973. Relative susceptibility of two common groundsel (*Senecio vulgaris* L.) biotypes to six *s* -triazines. Agron. J. 65: 553-555.

Radosevich, S. R., and J. S. Holt. 1982. Physiological responses and fitness of susceptible and resistant weed biotypes to triazine herbicides. p. 163-183 *in* H. M. LeBaron and J. Gressel, eds. Herbicide Resistance in Plants. John Wiley and Sons, Inc., New York.

Radosevich, S. R., and J. S. Holt. 1984. Weed Ecology. Implications for Vegetation Management. John Wiley & Sons, Inc., New York.

Radosevich, S. R., B. D. Maxwell, and M. L. Roush. 1989. Managing herbicide resistance through fitness and gene flow. *in* G. Cussans and J. C. Casely, eds. Herbicide Resistance in Weeds and Crops. Abstracts of papers presented at the 11th Long Ashton Int. Symp., Bristol, U.K.

Rahman, A. 1973. Effects of temperature and soil type on the phytotoxicity of trifluralin. Weed Res. 13:267-272.

Rahman, A., and R. Ashford. 1970. Selective action of trifluralin for control of green foxtail in wheat. Weed Sci. 18:754-759.

Rahman, A., and R. Ashford. 1972. Control of green foxtail in wheat with trifluralin. Weed Sci. 20:23-27.

Ratkowsky, D. A. 1983. Nonlinear Regression Modelling. Marcel Dekker, New York.

Ray, T. B. 1984. Site of action of chlorsulfuron. Plant Physiol. 75:827-831.

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

Rubin, B. 1991. Herbicide resistance, progress, and prospects. p. 387-414 *in* J. L. Caseley, G. W. Cussans, and R. K. Atkin, eds. Herbicide Resistance in Weeds and Crops. Butterworth-Heinemann Ltd.

Rubin, B., T. Yaacoby, and M. Schonfeld. 1985. Triazine resistant grass weeds: cross resistance with wheat herbicide, a possible threat to cereal crops. Br. Crop Prot. Conf.-Weeds:1171-1178.

Ryan, G. F. 1970. Resistance of common groundsel to simazine and atrazine. Weed Sci. 18:614-616.

Saari, L. L., J. C. Cotterman, and M. M. Primiani. 1990. Mechanism of sulfonylurea herbicide resistance in the broadleaf weed, *Kochia scoparia*. Plant Physiol. 93:55-61.

Sagar, G. R., and A. M. Mortimer. 1977. An approach to the study of the population dynamics of plants with special reference to weeds. Appl. Biol. 1:1-47.

Sawicki, R. M. 1987. Definition, detection and documentation of insecticide resistance. p. 105-117 *in* M. G. Ford, D. W. Holloman, B. P. S. Khambay, and R. M. Sawicki, eds. Combating Resistance to Xenobiotics. Ellis Horwood, Ltd., Chichester, UK.

Schonfeld, M., T. Yaacoby, O. Michael, and B. Rubin. 1987. Triazine resistance without reduced vigor in *Phalaris paradoxa*. Plant Physiol. 83:329-333.

Shaner, D. L., P. C. Anderson, and M. A. Stidham. 1984. Imidazolinones. Potent inhibitors of acetohydroxyacid synthase. Plant Physiol. 76: 545-546.

Smeda, R. J., K. C. Vaughn, and I. N. Morrison. 1991. Cross-resistance and hyper-sensitivity of trifluralin-resistant green foxtail [*Setaria viridis* (L.) Beauv.]. Abstr. Weed Sci. Soc. Am. 140:47.

Smith, A. E. 1972. Persistence of trifluralin in small field plots as analyzed by a rapid gas chromatographic method. J. Agric. Food Chem. 20:829-831.

Smith, A. E. 1981. Comparison of solvent systems for the extraction of atrazine, benzoylprop, flamprop, and trifluralin from weathered field soils. J. Agric. Fd. Chem. 29:111-115.

Smith, A. E. 1982. Herbicides and the soil environment in Canada. Can. J. Soil Sci. 62:433-460.

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

Strachan, S. D., and F. D. Hess. 1983. The biochemical mechanism of action of the dinitroaniline herbicide oryzalin. Pestic. Biochem. Physiol. 20: 131-150.

Strang, R. H., and R. L. Rogers. 1971. A microradioautographic study of ^{14}C-trifluralin absorption. Weed Sci. 19:363-369.

Sturko, A. R. W. 1978. Green foxtail (*Setaria viridis*) competition in semidwarf and normal height spring wheat. M.Sc. thesis, University of Manitoba, Winnipeg, MB. 45 pp.

Sumida, S., and M. Udea. 1976. Effect of O-ethyl O-(3-methyl-6-nitrophenyl) *N -sec* -butylphosphorothioamidate (S-2846), an experimental herbicide, on mitotis in *Allium cepa*. Plant Cell Physiol. 17:1351-1354.

Swann, C. W., and R. Behrens. 1972. Phytotoxicity of trifluralin vapors from soil. Weed Sci. 20:143-146.

Thill, D. C., C. A. Mallory-Smith, L. L. Saari, J. C. Cotterman, M. M. Primiani, and J. L. Saladini. 1991. Sulfonylurea herbicide resistant weeds: discovery, distribution, biology, mechanism, and management. *in* J. L. Caseley, G. W. Cussans, and R. K. Atkin, eds. Herbicide Resistance in Weeds and Crops. Butterworth-Heinemann Ltd.

Thomas, A. G., J. D. Banting, and G. Bowes. 1986. Longevity of green foxtail seeds in a Canadian prairie soil. Can. J. Plant Sci. 66:189-192.

Thomas, A. G., and D. I. Donaghy. 1991. A survey of the occurrence of seedling weeds in spring annual crops in Manitoba. Can. J. Plant Sci. 71:811-820.

Thomas, A. G., and R. Wise. 1985. Dew's Alberta weed survey 1973-1977. Weed Survey Ser. Publ. No. 85-3. Agriculture Canada, Regina.

Thomas, A. G., and R. Wise. 1987. Weed survey of Saskatchewan cereal and oilseed crops 1986. Weed Survey Ser. Publ. No. 87-1. Agric. Canada, Regina.

Thomas, A. G., and R. Wise. 1988. Weed survey of Manitoba cereal and oilseed crops 1986. Weed Survey Ser. Publ. No. 88-1. Agric. Canada, Regina.

Valverde, B. E., S. R. Radosevich, and A. P. Appleby. 1988. Growth and competitive ability of dinitroaniline-herbicide resistant and susceptible goosegrass (*Eleusine indica*) biotypes. Proc. West. Soc. Weed Sci. 41:81.

Vanden Born, W. H. 1971. Green foxtail: seed dormancy, germination and growth. Can. J. Plant Sci. 51:53-59.

Vaughan, M. A., and K. C. Vaughn. 1987. Taxol treatment of *Eleusine* indicates hyper-stabilized tubulin may cause dinitroaniline resistance. Plant Physiol. 83 (Suppl.):643.

Vaughn, K. C. 1986a. Cytological studies of dinitroaniline-resistant *Eleusine*. Pestic. Biochem. Physiol. 26:66-74.

Vaughn, K. C. 1986b. Dinitroaniline resistance in goosegrass [*Eleusine indica* (L.) Gaertn.] is due to an altered tubulin. Abstr. Weed Sci. Soc. Am. 26:77.

Vaughn, K. C. 1990. Mitotic disrupter herbicides. Abstr. Weed Sci. Soc. Am. 304:102.

Vaughn, K. C., and W. C. Koskinen. 1987. Effects of trifluralin metabolites on goosegrass (*Eleusine indica*) root meristems. Weed Sci. 35:36-44.

Vaughn, K. C., and L. P. Lehnen, Jr. 1991. Mitotic disrupter herbicides. Weed Sci. 39:450-457.

Vaughn, K. C., M. D. Marks, and D. P. Weeks. 1987. A dinitroaniline-resistant mutant of *Eleusine indica* exhibits cross-resistance and super-sensitivity to antimicrotubule herbicides and drugs. Plant Physiol. 83:956-964.

Vaughn, K. C., and M. A. Vaughan. 1990. Structural and biochemical characterization of dinitroaniline-resistant *Eleusine*. p. 364-375 *in* M. B. Green, H. M. LeBaron, and W. K. Moberg, eds. Managing Resistance to Agrochemicals: From Fundamental Research to Practical Strategies. ACS Symp. Ser. 421, ACS, Washington, DC.

Vaughn, K. C., M. A. Vaughan, and B. J. Gossett. 1990. A biotype of goosegrass (*Eleusine indica*) with an intermediate level of dinitroaniline herbicide resistance. Weed Technol. 4:157-162.

Warwick, S. I. 1991. Herbicide resistance in weedy plants: physiology and population biology. Annu. Rev. Ecol. Syst. 22:95-114.

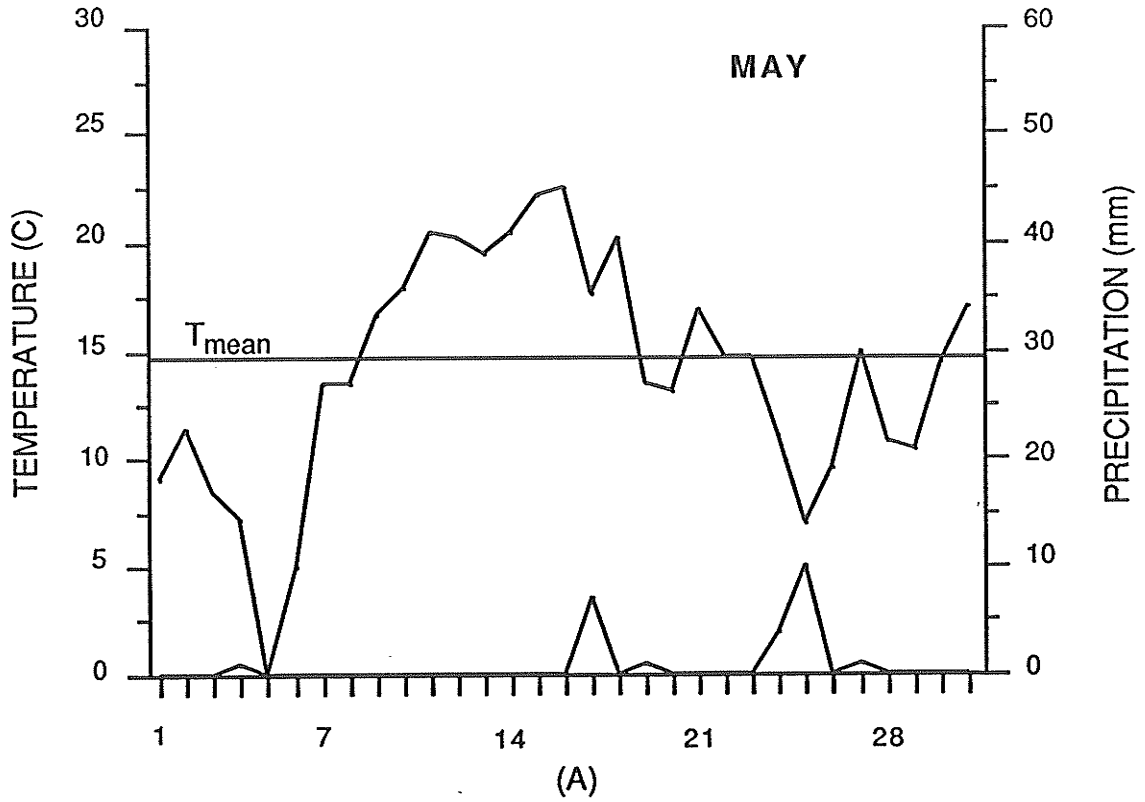
Weber, J. B., S. B. Weed, and T. W. Waldrep. 1974. Effect of soil constituents on herbicide activity in modified-soil field plots. Weed Sci. 22:454-459.

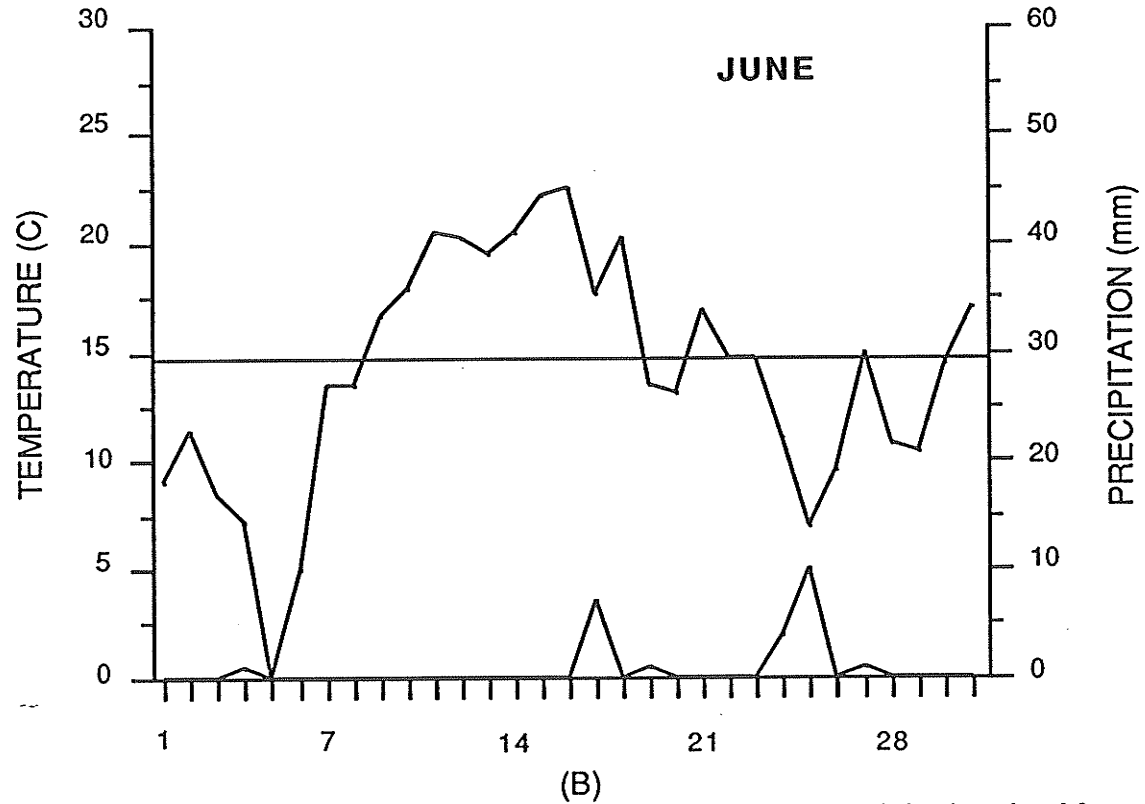
Williams, K. L. 1976. Mutation frequency at a recessive locus in haploid and diploid strains of a slime mould. Nature 260:785-786.

WSSA. 1989. Herbicide Handbook of the Weed Science Society of America, 6th ed. Weed Sci. Soc. Am., Champaign, IL.

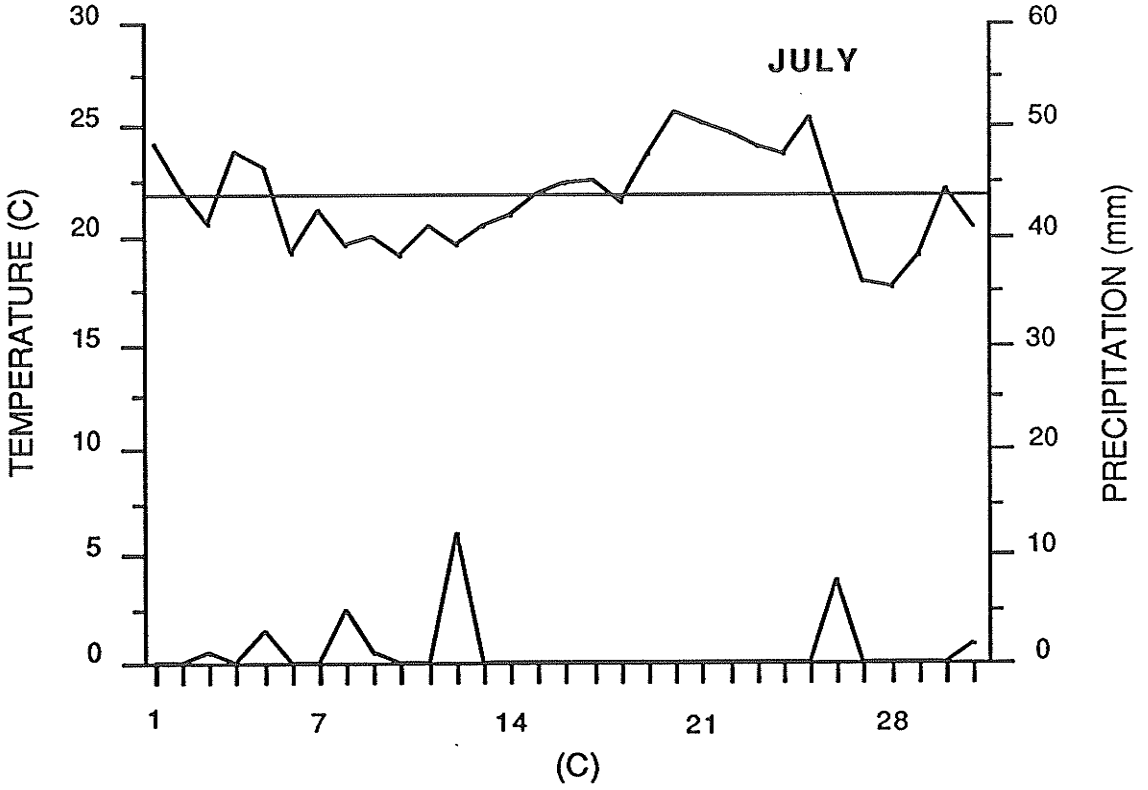
Zimdahl, R. L., and S. M. Gwynn. 1977. Soil degradation of three dinitroanilines. Weed Sci. 25:247-251.

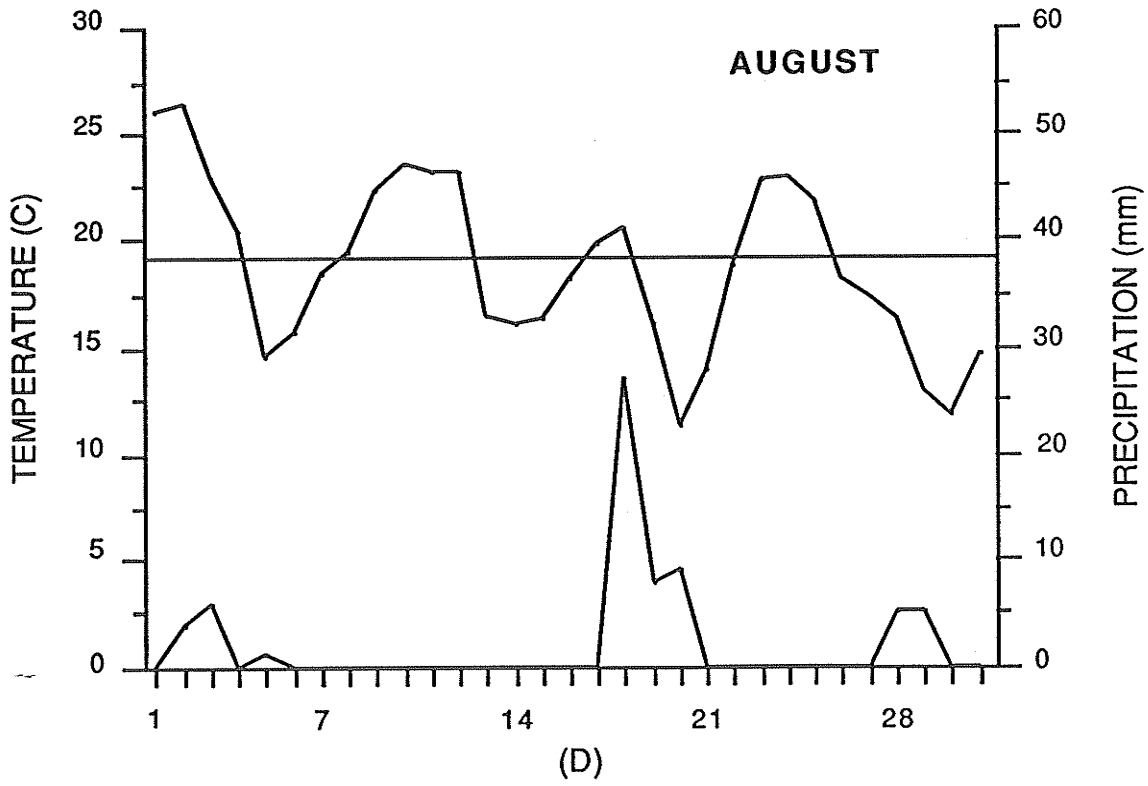
APPENDIX





Appendix Figure 1. Daily mean air temperatures and precipitation for May to August of 1989 (A-D) and 1990 (E-H) at Portage la Prairie.





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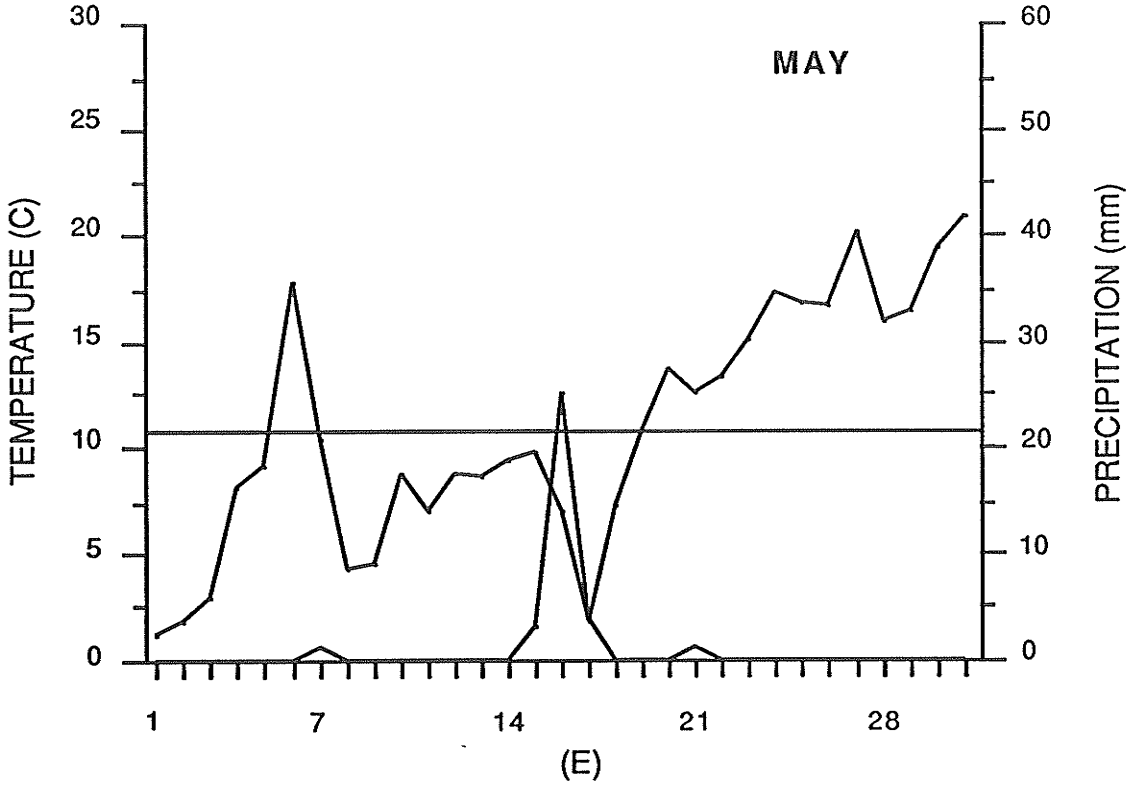

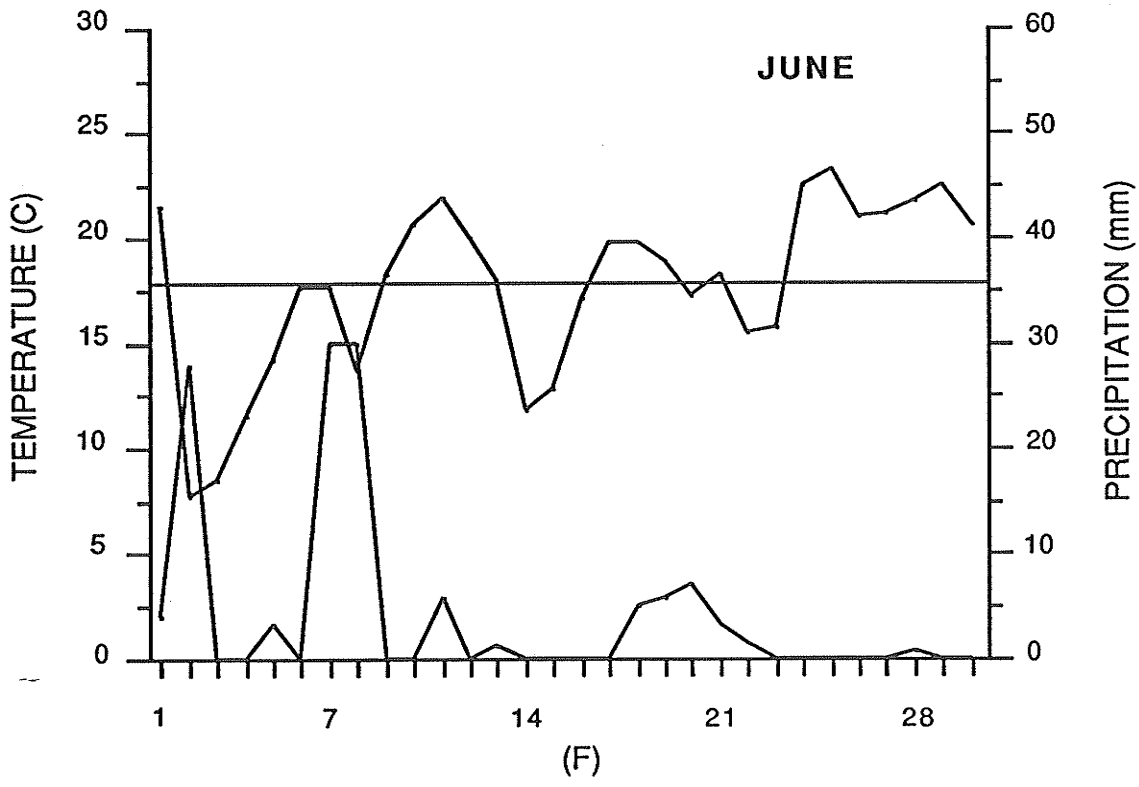

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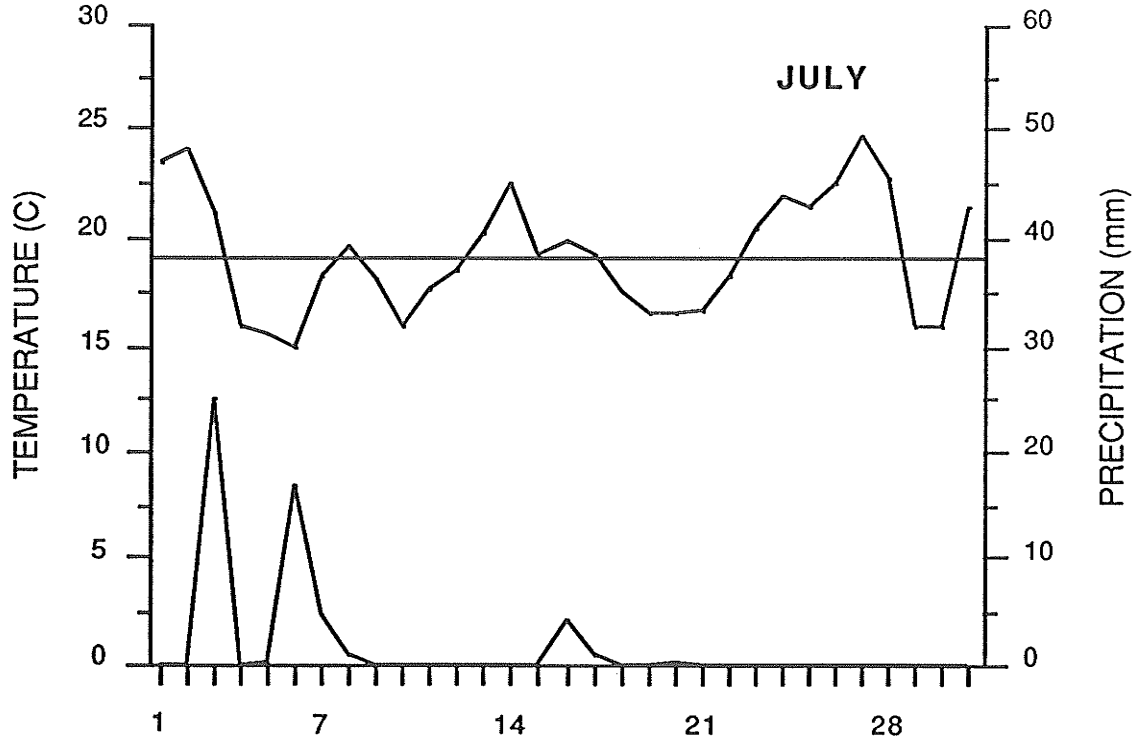
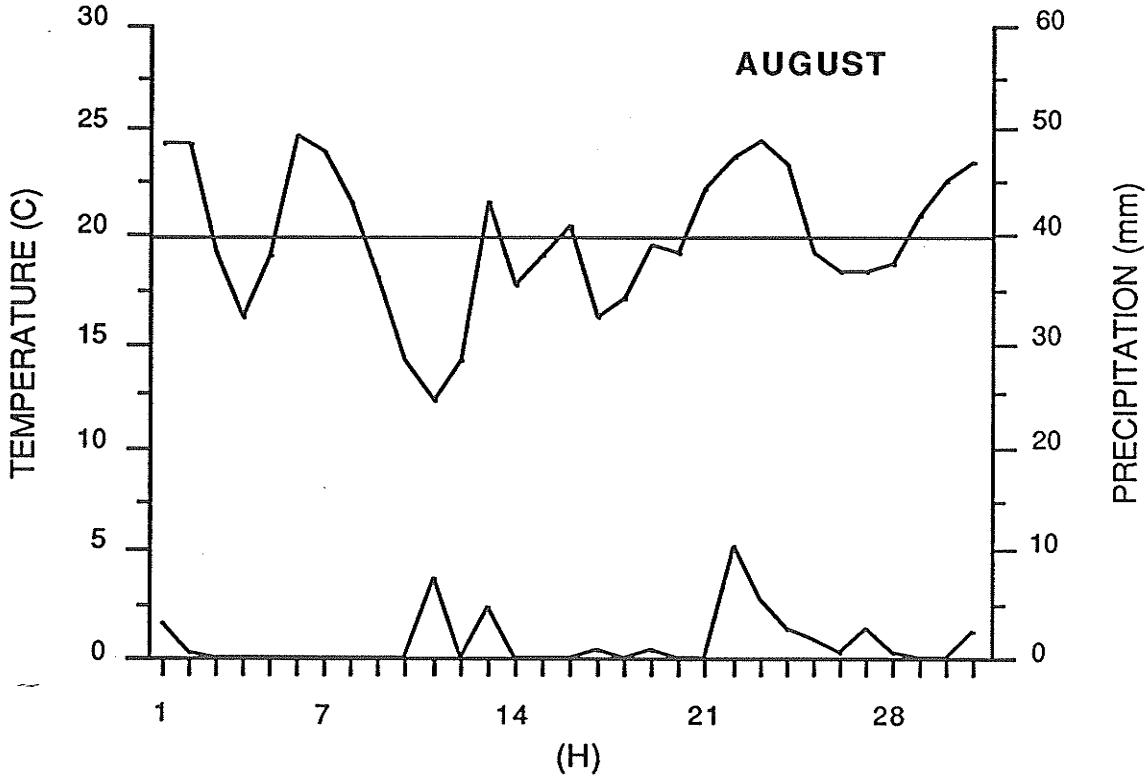


(G)



(H)

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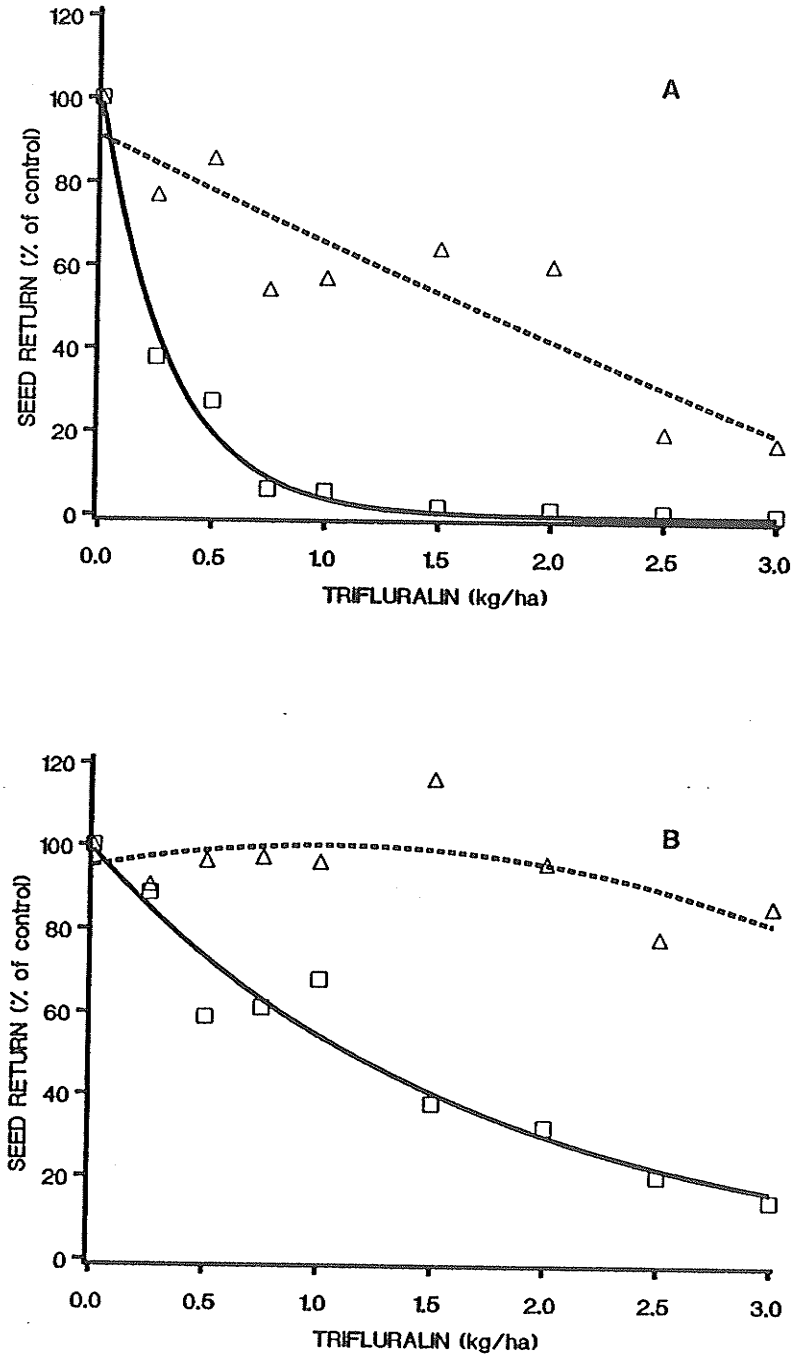
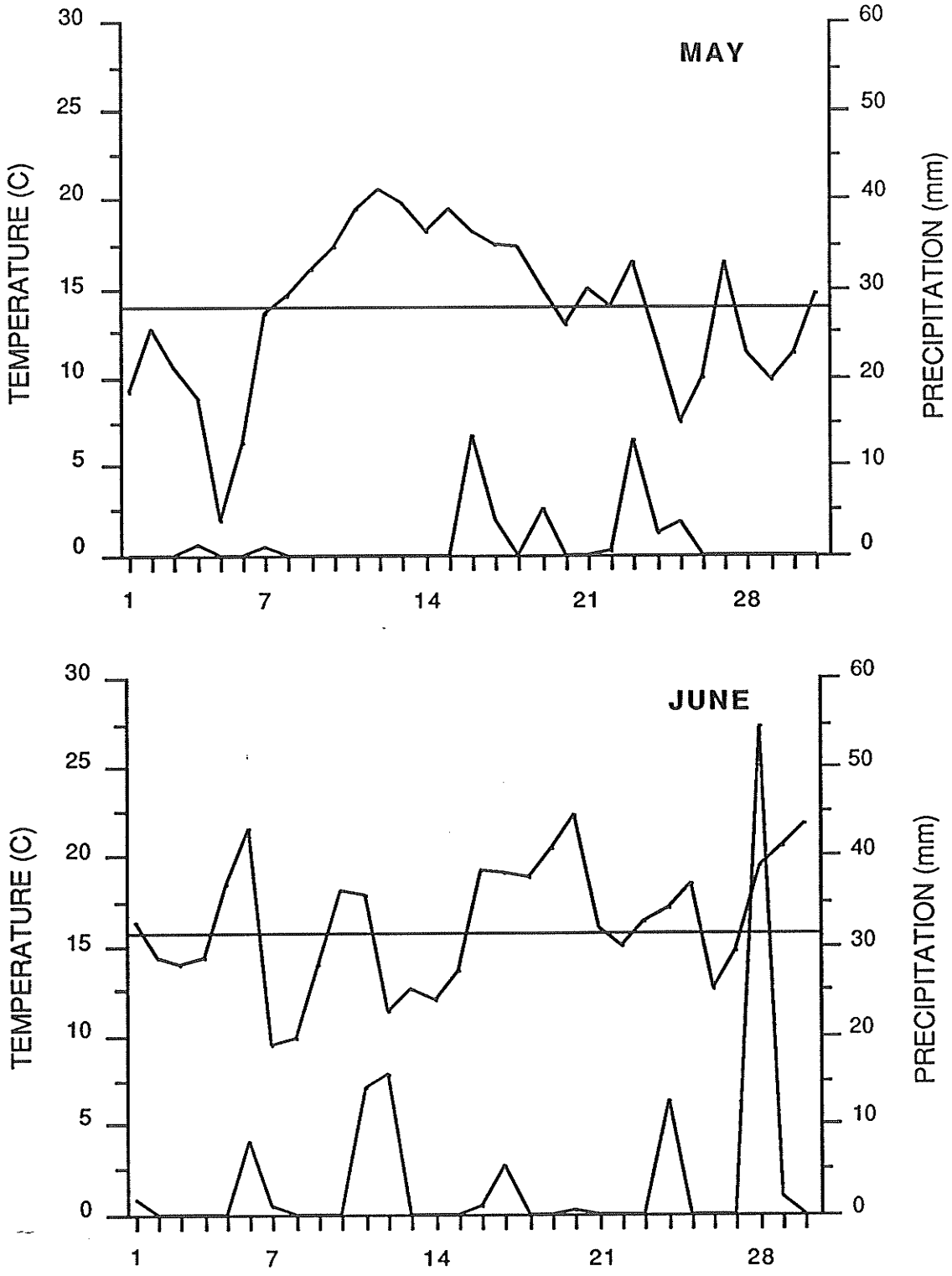
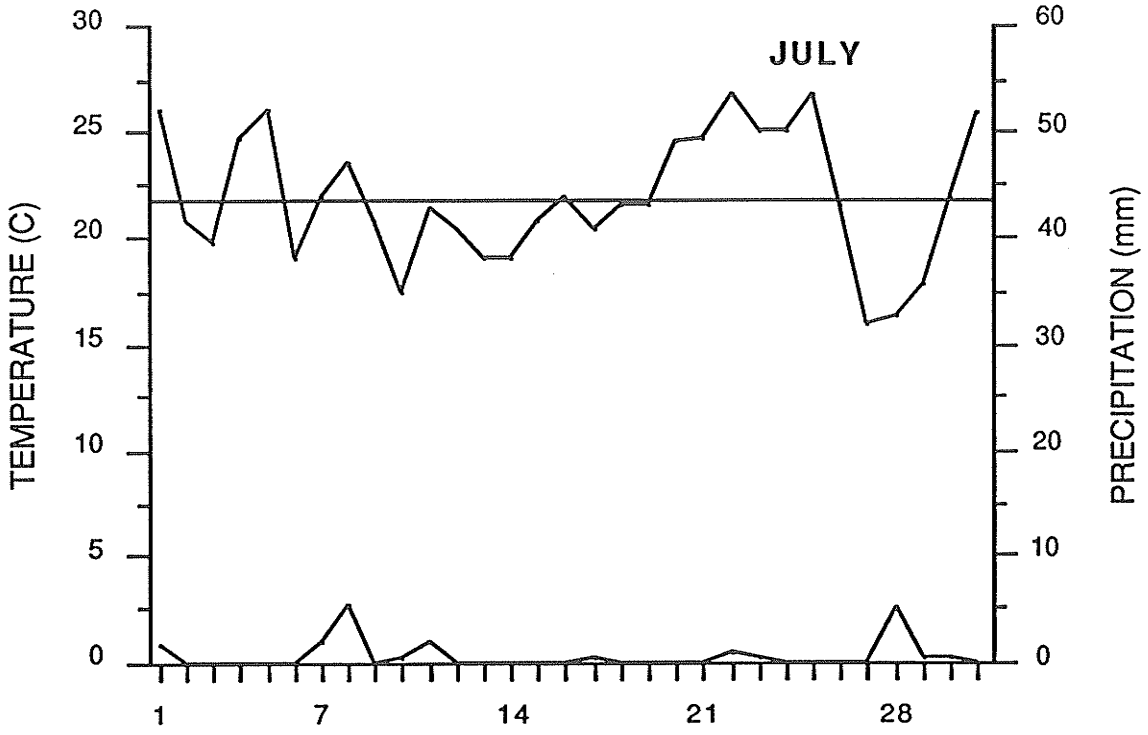
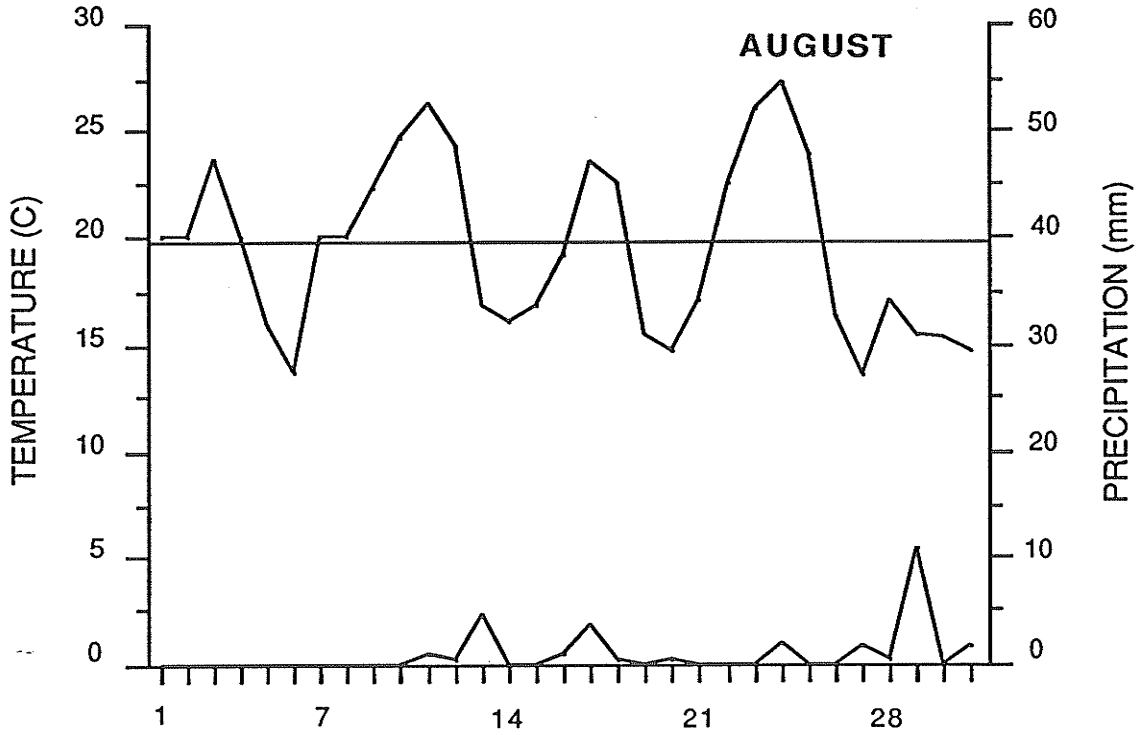


Appendix Figure 2. The effect of increasing dosages of PPI trifluralin on R (dashed line) and S (solid line) green foxtail seed number (1-m^2 basis) under cropped (A) and non-cropped (B) conditions at Portage la Prairie in 1989 and 1990. See Appendix Table 1 for equations and parameter estimates.



Appendix Figure 3. Daily mean air temperatures and precipitation for May to August of 1989 at Deloraine, Manitoba.





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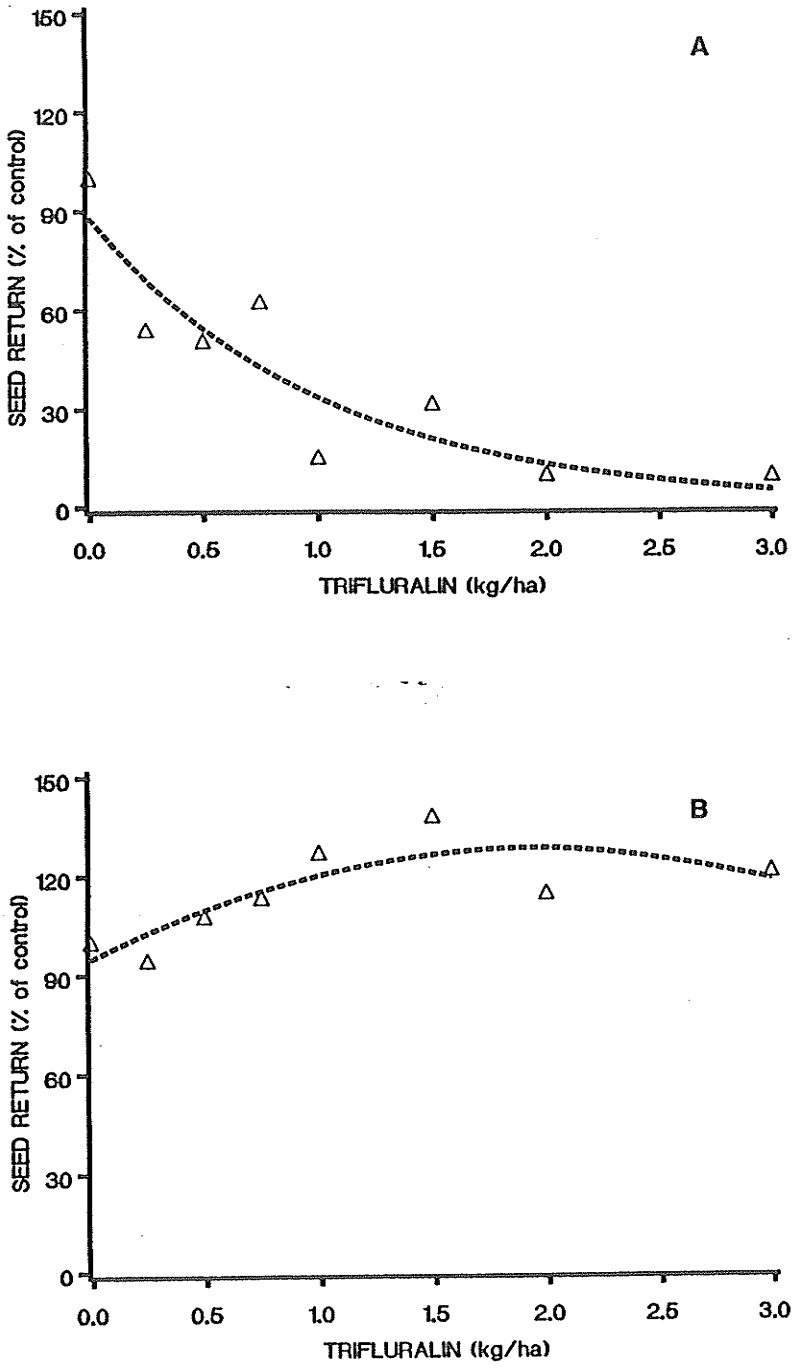
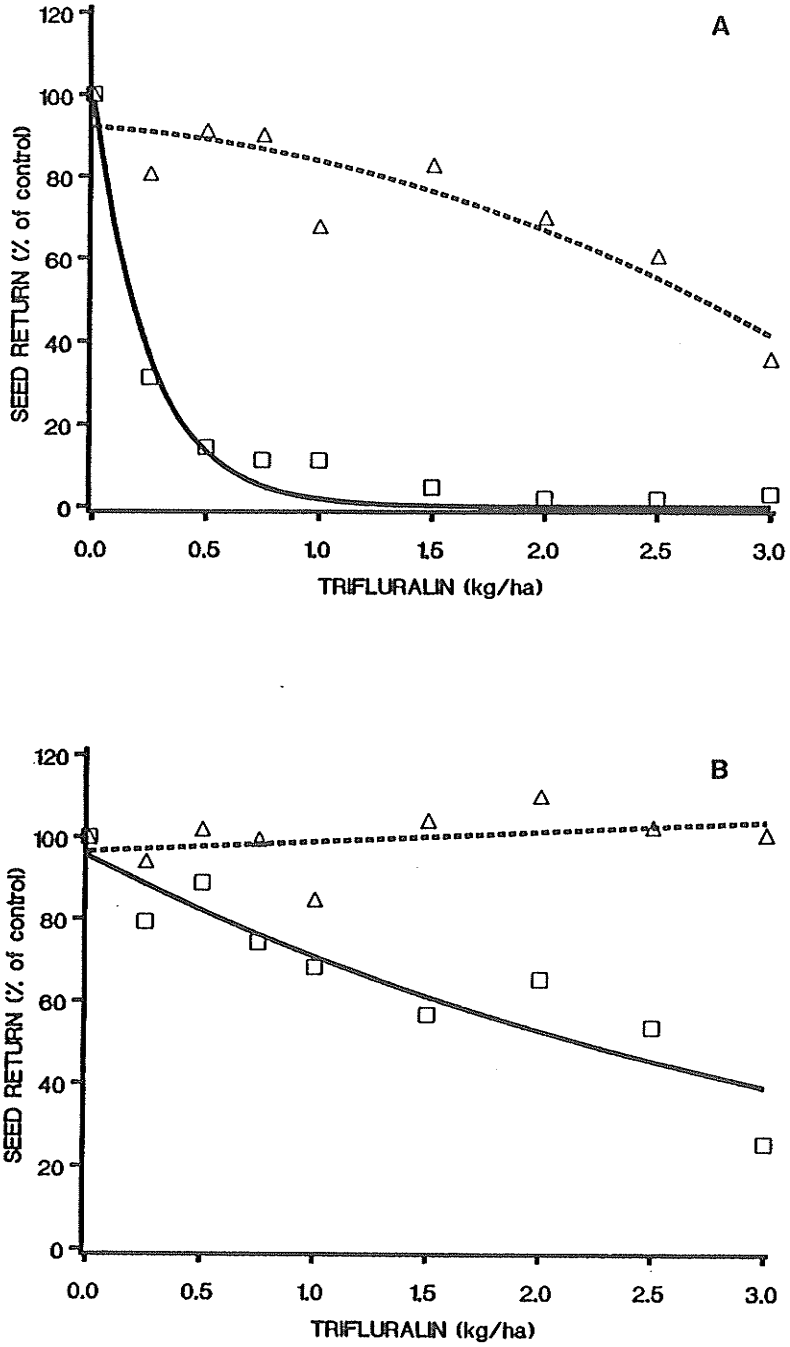
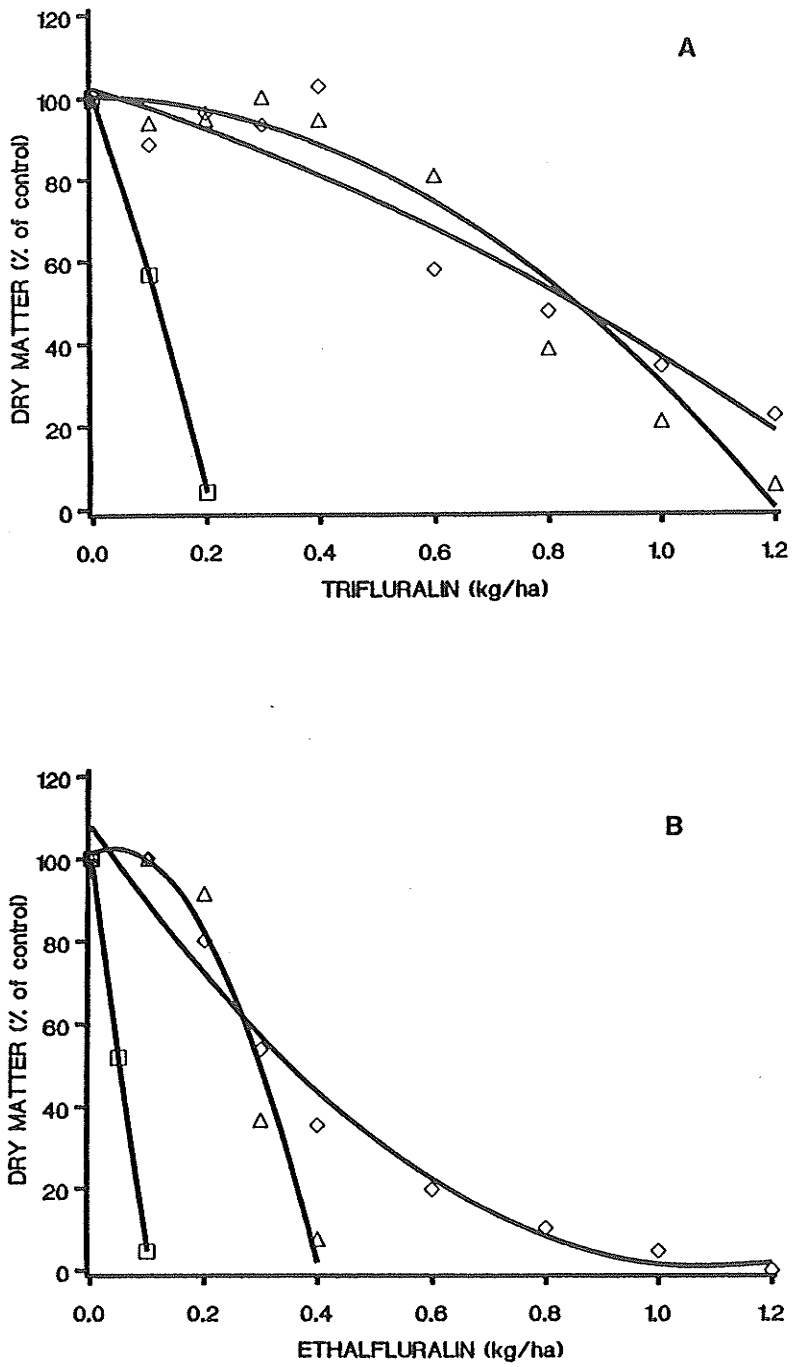


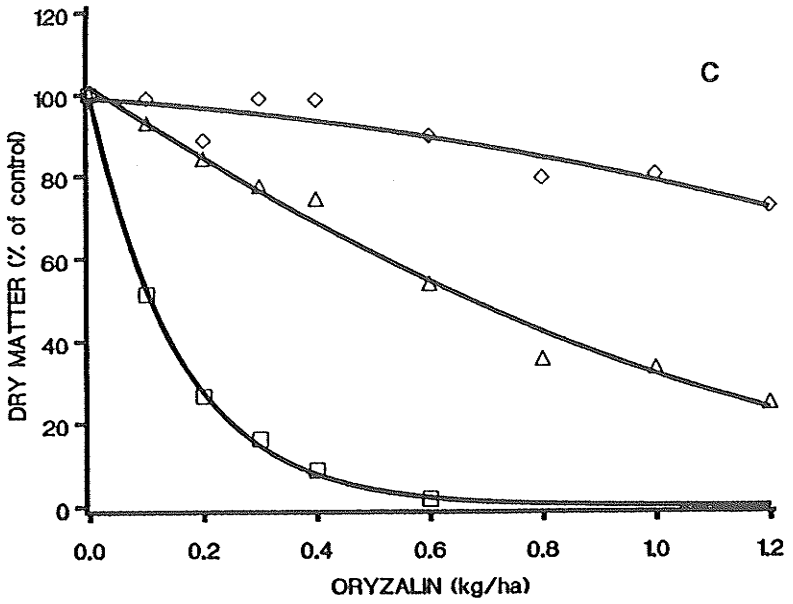
Appendix Figure 4. The effect of increasing dosages of PPI trifluralin on R green foxtail seed number (1-m^2 basis) under cropped (A) and non-cropped (B) conditions at Deloraine in 1989. See Appendix Table 2 for equations and parameter estimates.

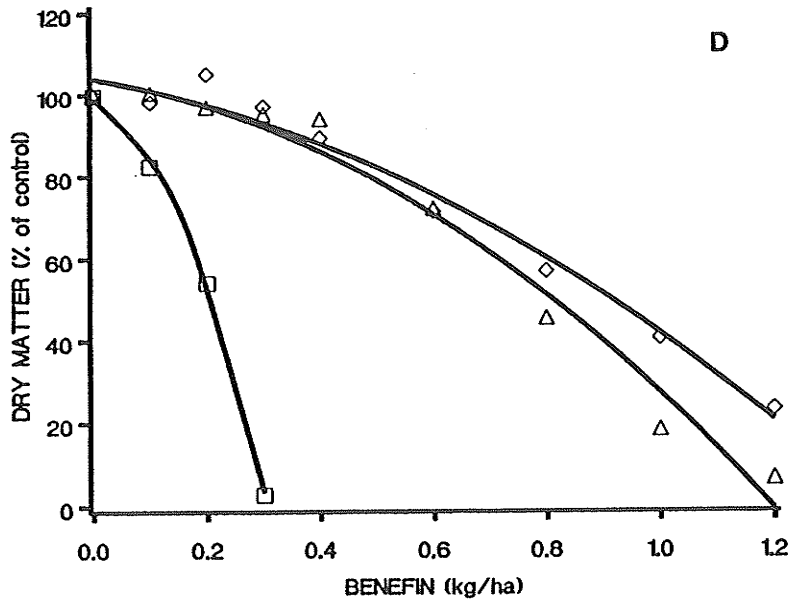


Appendix Figure 5. The effect of increasing dosages of PEI trifluralin on S (solid line) and R (dashed line) green foxtail seed number (1-m^2 basis) under cropped (A) and non-cropped (B) conditions at Portage la Prairie in 1989 and 1990. See Appendix Table 3 for equations and parameter estimates.



Appendix Figure 6. Response of S (square symbols) and R (triangle symbols) green foxtail and 'Katepwa' wheat (diamond symbols) to trifluralin (A), ethalfluralin (B), oryzalin (C), benefin (D), prodiamine (E), and pendimethalin (F).





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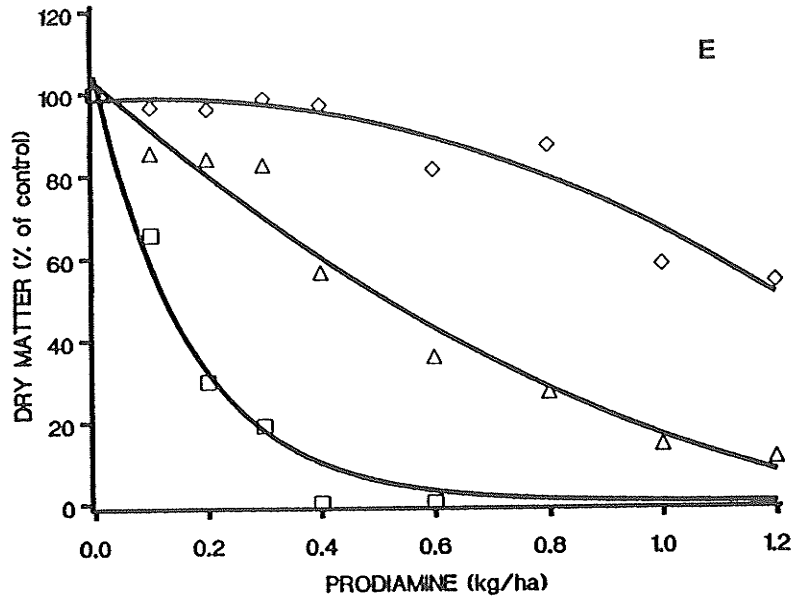

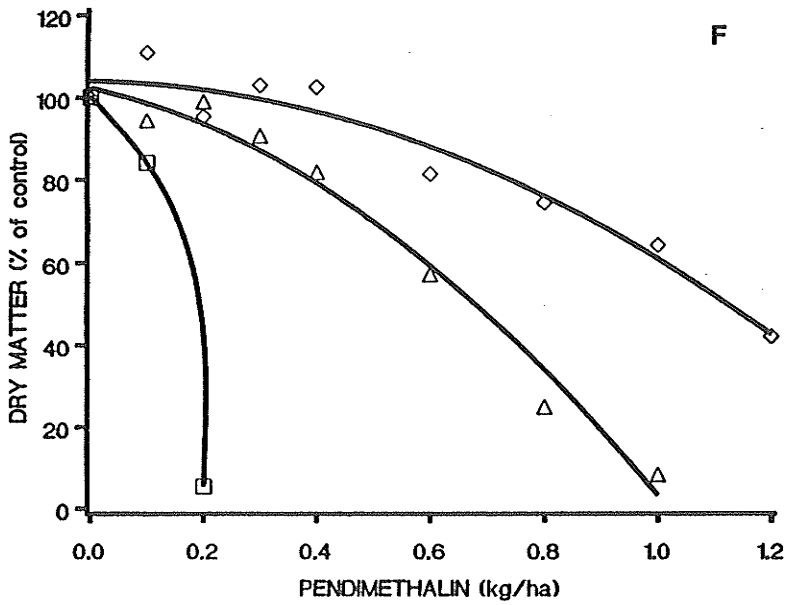

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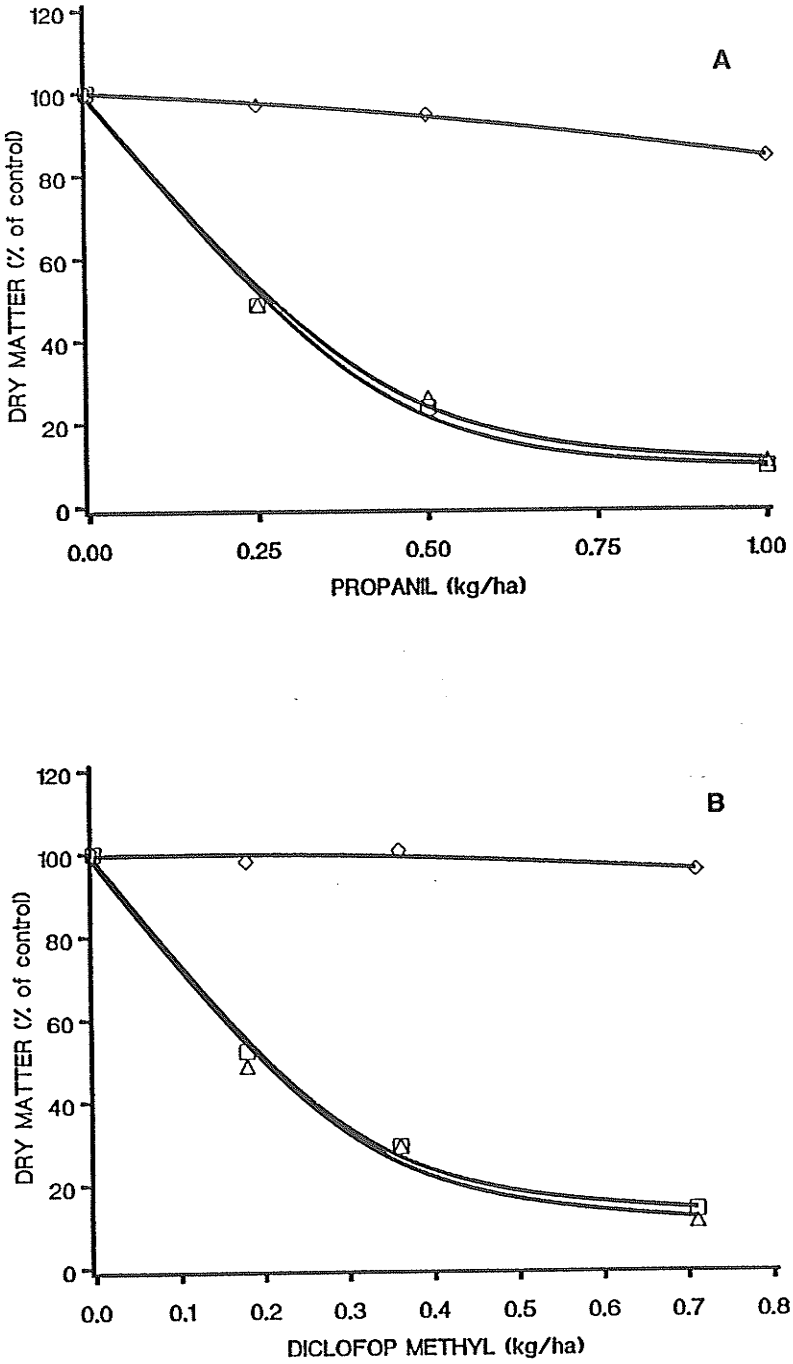
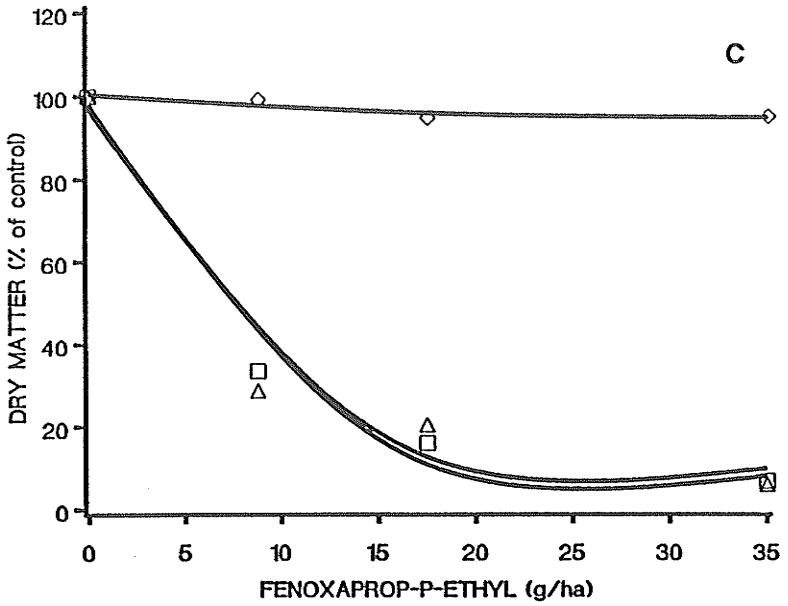
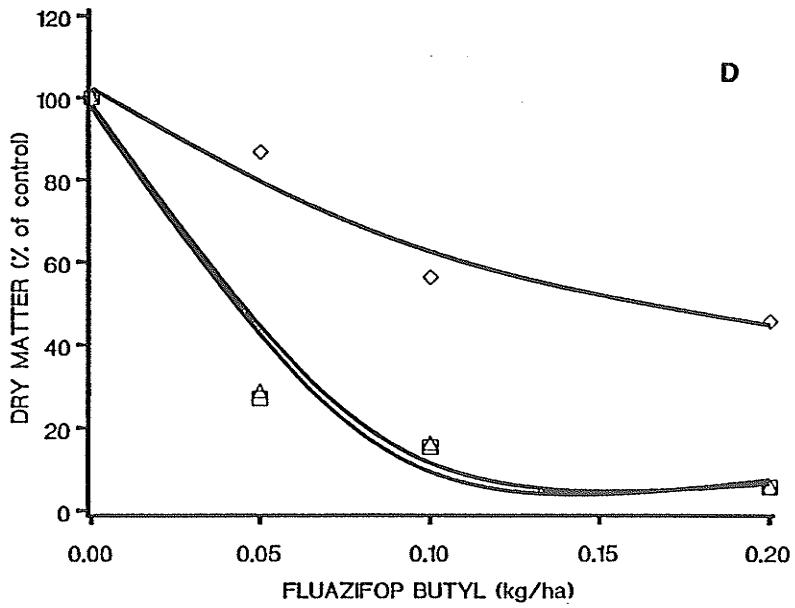
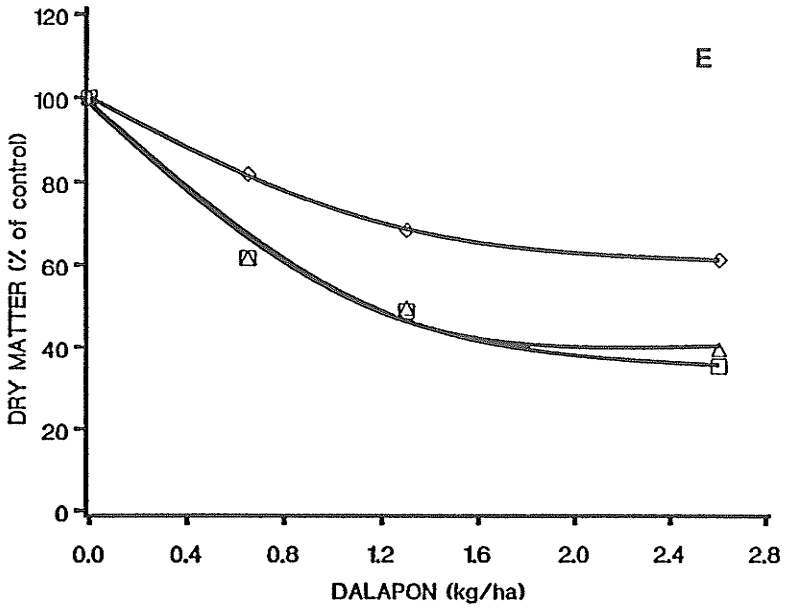


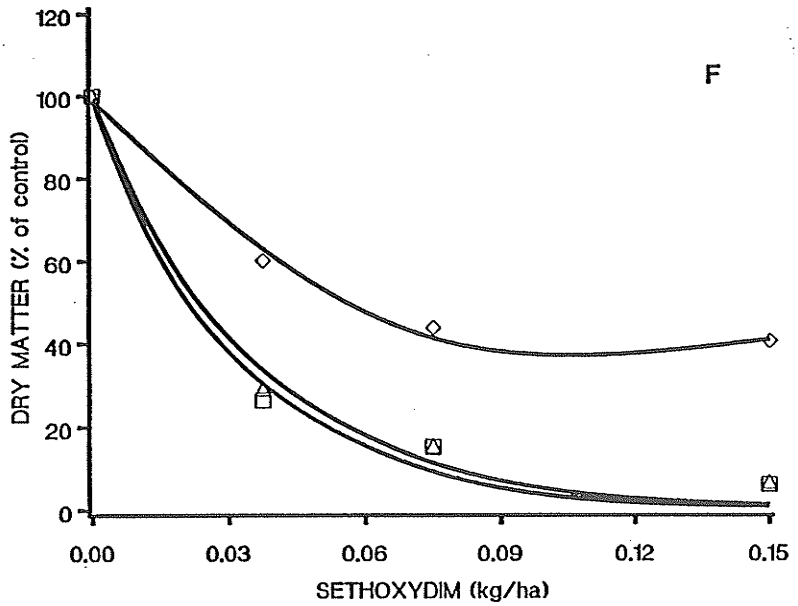
Appendix Figure 7. Response of S (square symbols) and R (triangle symbols) green foxtail and 'Katepwa' wheat (diamond symbols) to propanil (A), diclofop methyl (B), fenoxaprop-p-ethyl (C), fluazifop-butyl (D), dalapon (E), sethoxydim (F), linuron (G), and EPTC (H).



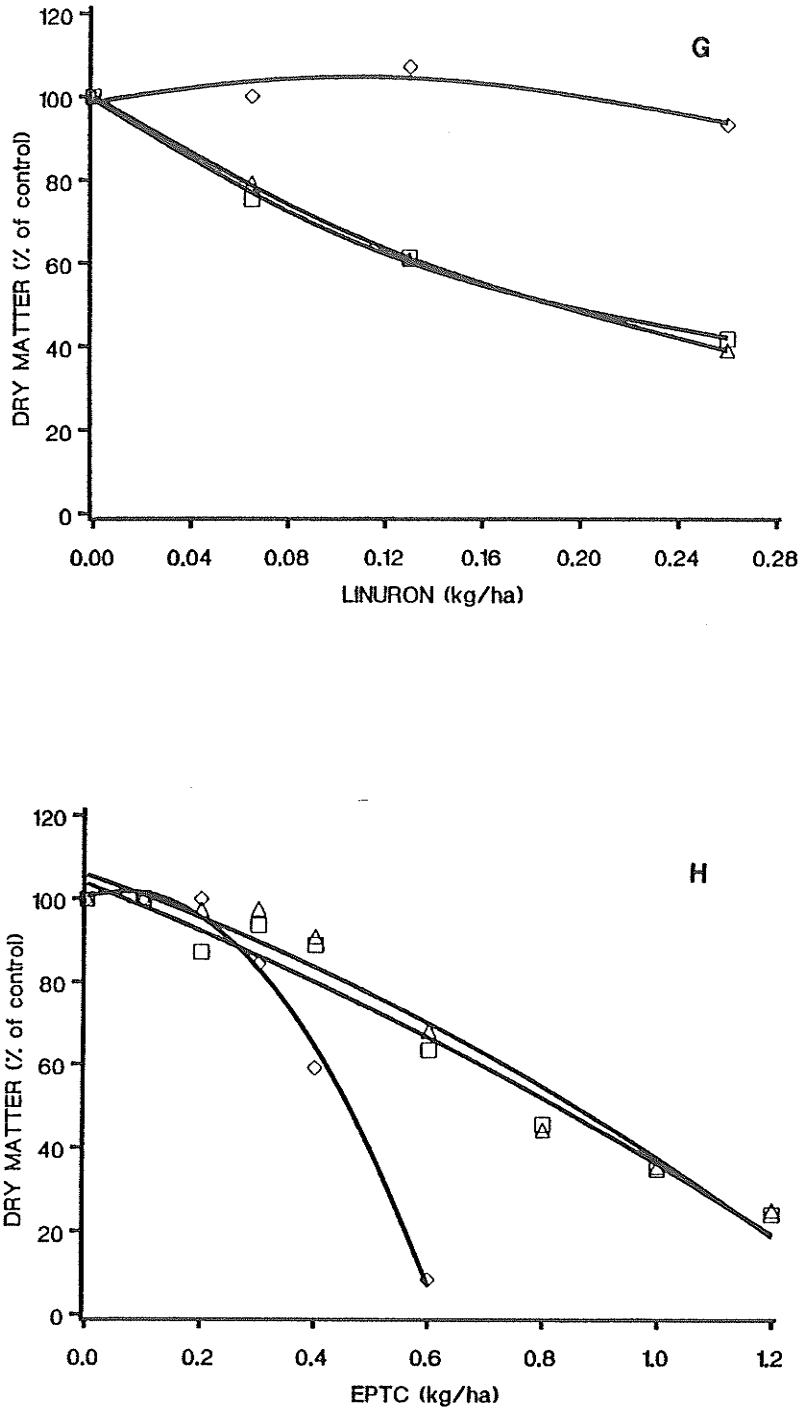


Appendix Figure 7 (continued).

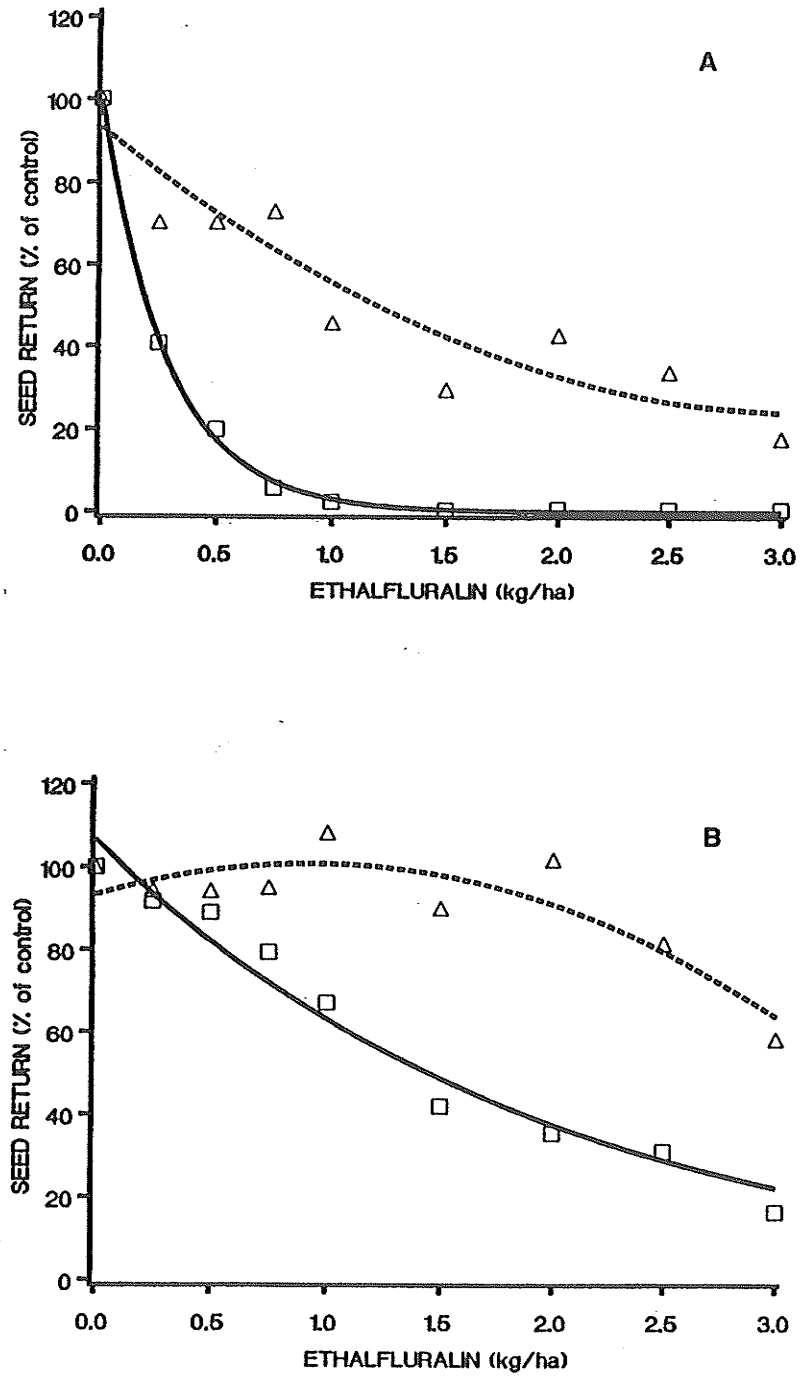




Appendix Figure 7 (continued).



Appendix Figure 7 (continued).



Appendix Figure 8. The effect of increasing dosages of ethalfluralin on S (solid line) and R (dashed line) green foxtail seed number (1-m^2 basis) under cropped (A) and non-cropped (B) conditions at Portage la Prairie in 1989 and 1990. See Appendix Table 5 for equations and parameter estimates.

Appendix Table 1. Parameter estimates (standard errors in parentheses) and ED_{50}'s of the equations for the regression curves for the response of S and R green foxtail seed number to PPI trifluralin under cropped and non-cropped conditions at Portage la Prairie in 1989 and 1990.

Plant variable[a]	a[b]	b	c	R^{2c}	ED_{50}[d]	R/S
Cropped conditions						
S seed no	98.9(3.8)	-3.2(0.3)		0.90**	0.2	
R seed no	90.8(9.1)	-26.1(16.3)	0.8(5.5)	0.35**	1.8	9
Non-cropped conditions						
S seed no	98.8(7.0)	-0.6(0.1)		0.53**	1.1	
R seed no	95.1(6.7)	9.4(11.4)	-4.7(3.7)	0.06*	4.3	4

[a]Mean values ± standard error for seed no (per 1-m^2 basis) in control plots: **Cropped conditions** S 43 000(7 300), R 60 000(9 800); **Non-cropped conditions** S 285 000(27 000), R 279 000 (37 000).

[b]Exponential function equation: $y = a\, e^{bx}$ where a = intercept (% of control) and ab = initial slope; quadratic function equation: $y = a + bx + cx^2$ where a = intercept (% of control), b = linear coefficient, and c = curvilinear coefficient; y is seed no (% of control) and x is the trifluralin dosage (kg ha^{-1}).

[c]Coefficient of determination: significant at the 5% level (*), 1% level (**).

[d]ED_{50} is the trifluralin dosage required to reduce seed no by 50% relative to the control.

Appendix Table 2. Parameter estimates (standard errors in parentheses) and ED_{50}'s of the equations for the regression curves for the response of R green foxtail seed number to PPI trifluralin under cropped and non-cropped conditions at Deloraine in 1989.

Plant variable[b]	a[c]	b	c	R^{2}[d]	ED_{50}[a] (kg ha^{-1})	
					Inter-polated	Adjusted
Cropped conditions						
Seed no	88.0(17.3)	-1.0(0.4)		0.31	0.7	1.2
Non-cropped conditions						
Seed wt	94.7(10.7)	33.4(19.2)	-8.6(6.2)	0.13		

[a]Effective dosage required to reduce seed no by 50% relative to the control; ED_{50}'s were calculated by the addition of the carryover residue level of 0.5 kg ha^{-1} detected at seeding time to the application dosages (not determined for seed no (non-cropped conditions) due to the positive slope of the regression curve).

[b]Mean values ± standard error for seed no (per 1-m^2 basis) in control plots: **Cropped conditions** 5 420(1 060); **Non-cropped conditions** 64 200 (7 900).

[c]Exponential function equation: $y = a\, e^{bx}$ where a = intercept (% of control) and ab = initial slope; quadratic function equation: $y = a + bx + cx^2$ where a = intercept (% of control), b = linear coefficient, and c = curvilinear coefficient; y is seed no (% of control) and x is the trifluralin dosage (kg ha^{-1}).

[d]All coefficients of determination are significant at the 1% level.

Appendix Table 3. Parameter estimates (standard errors in parentheses) and ED_{50}'s of the equations for the regression curves for the response of S and R green foxtail seed number to PEI trifluralin under cropped and non-cropped conditions at Portage la Prairie in 1989 and 1990.

Plant variable[a]	a[b]	b	c	R^{2c}	ED_{50}[d]	R/S
Cropped conditions						
S seed no	98.7(4.1)	-4.0(0.4)		0.87**	0.2	
R seed no	92.3(9.4)	-4.7(15.9)	-4.1(5.2)	0.20**	2.8	14
Non-cropped conditions						
S seed no	95.3(5.9)	-0.3(0.1)		0.35**	2.0	
R seed no	96.5(6.9)	2.1(12.2)	-0.1(4.0)	0.01	>3.0	

[a]Mean values ± standard error for seed no (per 1 m^2 basis) in control plots: **Cropped conditions** S 35 900(10 000), R 29 000(6 900); **Non-cropped conditions** S 376 000(60 000), R 332 000(48 000).

[b]Exponential function equation: $y = a\, e^{bx}$ where a = intercept (% of control) and ab = initial slope; quadratic function equation: $y = a + bx + cx^2$ where a = intercept (% of control), b = linear coefficient, and c = curvilinear coefficient; y is seed no (% of control) and x is the trifluralin dosage (kg ha^{-1}).

[c]Coefficient of determination: significant at the 5% level (*), 1% level (**).

[d]ED_{50} is the trifluralin dosage required to reduce seed no by 50% relative to the control.

Appendix Table 4. Parameter estimates (standard errors in parentheses) and ED_{50}'s of the equations for the regression curves for the shoot dry matter response of S and R green foxtail and 'Katepwa' wheat to herbicides belonging to various chemical groups.

Herbicide[a]		a[b]	b	c	R^2[c]	ED_{50}[d]
						kg ha^{-1}
Propanil	R[e]	98.5 (4.0)	-211 (19)	124 (18)	0.89**	0.28
	S	98.7 (6.0)	-220 (30)	131 (27)	0.80**	0.27
	W	99.8 (4.4)	-8.1 (23.6)	-7.6 (21.9)	0.20**	>1.2
Diclofop methyl	R	98.0 (5.3)	-287 (38)	233 (52)	0.83**	0.21
	S	98.8 (5.7)	-284 (40)	232 (53)	0.80**	0.21
	W	99.5 (2.7)	2.6 (20.6)	-11.1 (29.6)	0.03	>0.7
Fenoxaprop-p-ethyl[f]	R	97.0 (9.4)	-7.2 (1.1)	0.13 (0.04)	0.70**	7.9
	S	98.0 (5.7)	-7.4 (0.9)	0.14 (0.02)	0.87**	7.9
	W	100.4 (5.2)	-0.36 (0.78)	0.01 (0.02)	0.02	>35
Fluazifop butyl	R	97.4 (5.9)	-1310 (160)	4310 (760)	0.86**	0.04
	S	98.3 (5.1)	-1280 (130)	4090 (670)	0.90**	0.04
	W	102.3 (4.2)	-510 (124)	1110 (650)	0.79**	0.15
Dalapon	R	98.5 (8.2)	-58.0 (16.6)	13.7 (6.2)	0.52**	1.2
	S	99.0 (7.8)	-56.5 (16.5)	12.4 (6.1)	0.62**	1.2
	W	100.1 (2.7)	-33.4 (8.2)	7.1 (3.2)	0.71**	>2.6
Sethoxydim	R	97.6 (6.0)	-1780 (230)	7960 (1630)	0.88**	0.02
	S	97.5 (5.3)	-1860 (110)	8420 (400)	0.92**	0.02
	W	99.0 (4.0)	-1150 (140)	5100 (900)	0.83**	0.06

Appendix Table 4 (continued).

Linuron	R	100.1 (8.5)	-362 (171)	490 (614)	0.47**	0.18
	S	99.2 (8.0)	-383 (156)	626 (569)	0.46**	0.19
	W	98.6 (2.7)	109 (54)	-494 (192)	0.24**	>0.26
EPTC	R	105.7 (3.3)	-46.8 (14.7)	-21.3 (12.2)	0.84**	0.82
	S	103.6 (4.6)	-53.6 (20.8)	-13.9 (17.5)	0.72**	0.82
	W	100.7 (2.9)	39.3 (23.4)	-325 (37)	0.93**	0.46

[a]All herbicides except EPTC were applied postemergence. Plants were harvested 14 d after postemergence herbicide treatment; plants subjected to the EPTC preemergence treatment were harvested 30 d after seeding.

[b] Quadratic function equation: $y = a + bx + cx^2$ where a = intercept (% of control), b = linear coefficient, and c = curvilinear coefficient, y is shoot dry matter (% of control) and x is the herbicide dosage (kg ha^{-1}).

[c]Coefficient of determination: * significant at the 5% level; ** significant at the 1% level.

[d]ED_{50} is the herbicide application dosage required to reduce shoot dry matter by 50%.

[e]S = trifluralin-susceptible, R = trifluralin-resistant green foxtail biotypes, W = 'Katepwa' wheat.

[f]ED_{50} values for R, S, and W are in g ai ha^{-1}.

Appendix Table 5. Parameter estimates (standard errors in parentheses) and ED_{50}'s of the equations for the regression curves for the response of S and R green foxtail seed number to ethalfluralin under cropped and non-cropped conditions at Portage la Prairie in 1989 and 1990.

Plant variable[a]	a[b]	b	c	R^{2c}	ED_{50}[d]	R/S
Cropped conditions						
S seed no	99.8(5.0)	-3.5(0.4)		0.84	0.2	
R seed no	93.1(7.2)	-44.9(12.9)	7.3(4.2)	0.48	1.4	7
Non-cropped conditions						
S seed no	106.6(6.2)	-0.5(0.1)		0.59	1.3	
R seed no	93.3(4.8)	15.7(8.6)	-8.6(2.9)	0.27	3.4	3

[a]Mean values ± standard error for seed no (per 1-m^2 basis) in control plots: **Cropped conditions** S 84 400(15 700), R 109 000 (18 000); **Non-cropped conditions** S 313 000(41 000), R 329 000(43 000).

[b]Exponential function equation: $y = a\, e^{bx}$ where a = intercept (% of control) and ab = initial slope; quadratic function equation: $y = a + bx + cx^2$ where a = intercept (% of control), b = linear coefficient, and c = curvilinear coefficient; y is seed no (% of control) and x is the ethalfluralin dosage (kg ha^{-1}).

[c]All coefficients of determination are significant at the 1% level.

[d]ED_{50} is the ethalfluralin dosage required to reduce seed no by 50% relative to the control.

Appendix Table 6. Location of origin of R and S green foxtail populations used in the bioassay and mitotic index study.

Population	Seed sample[a]	Location	Legal land location (W1)	Producer
Bioassay				
Table 5-1				
S1		Portage la Prairie	Univ. of Man. Research Station	
R1	8816	Pierson	SE 8-3-29	Drier Farms
R2	8894	Gilbert Plains	NW 11-25-22	B. Randell
R3	88112	Deloraine	NE 14-4-23	C. Beernaert
Table 5-2				
Trifluralin dose-response experiment				
S1	same as above			
S2	8806	Reston	25-9-29	B. Clark
R1	8816			
R2	8894			
R3	88123	Belmont	NE 20-4-15	H. Dubyts
Ethalfluralin dose-response experiment				
S1	same as above			
R1	Field experiments		NW 13-4-23	B. Day
Table 5-4				
S1	same as above			
S2	8806			
S3	88146	Pierson	5-3-28	B. Riddel
S4	8835	Holland	NE 1-8-12	D. Patenaude
R1	8812	Reston	NW 23-7-28	G. Caldwell
R2	8816			
R3	8874	Killarney	SE 31-1-16	V. Martens
R4	8894			
R5	88107	Deloraine	N1/2 13-4-23	B. Day
R6	88112			
R7	88117	Deloraine	S1/2 23-3-23	B. McMechan
R8	88123			
Mitotic index study				
Table 5-5				
S1	same as above			
R1	Field experiments			

[a]Collection of seed samples is described in Morrison et al. (1989).