AN ABSTRACT OF THE THESIS OF

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Title: Postemergence Herbicides and Take-All Disease of Winter

Wheat: Alterations in the Incidence and Severity of

Disease and Crop Growth.

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Eight postemergence herbicides were evaluated to determine

their influence on the incidence and severity of take-all disease

caused by the fungus Gaeumannomyces graminis var. tritici in winter

wheat. Mecoprop (($\pm$)-2-(4-chloro-2-methylphenoxy)propanoic acid),

difenzoquat (1,2-dimethyl-3,5-diphenyl-1H-pyrazolium), dinoseb amine

(amine salt of 2-(1-methylpropyl)-4,6-dinitrophenol), and diclofop-

methyl (methyl ester of ($\pm$)-2-[4-(2,4-dichlorophenoxy)

phenoxy]propanoic acid) sometimes reduced the severity of take-all

disease on the seminal and crown roots. The herbicides also reduced

the incidence of 'whiteheads' associated with take-all injury. In

additional evaluations, mecoprop, difenzoquat, and dinoseb did not

affect severity of disease on the roots, but whiteheads again were reduced. Diclofop-methyl applied at 1.12 kg ai/ha in early January often reduced the severity of take-all disease on seminal and crown roots. Higher rates of this herbicide, however, occasionally increased disease severity.

The number of seminal roots produced by plants treated with diclofop-methyl often depended upon both application rate and the level of take-all stress. Crown root and tiller production were stimulated by diclofop-methyl. Low rates of difenzoquat, dinoseb, and mecoprop often increased tillering as well. The responses of the wheat plant to all four herbicides generally were greatest in plots with take-all disease. Fresh weights of individual tillers generally were reduced in response to herbicide application. Stimulations of tillering in diseased plots treated with diclofop-methyl progressively declined when applications were delayed after early January.

Grain yields were greater in diseased plots treated with mecoprop, difenzoquat, and dinoseb than in the untreated check in 1982. Grain yield was either reduced or unaffected by diclofop-methyl, regardless of the level of take-all disease present. Herbicides did not affect grain yield in 1983.

In vitro growth of three isolates of Gaeumannomyces graminis var. tritici was not inhibited by diclofop-methyl at concentrations below 10 μM.

Data suggest that herbicides reduced take-all disease by altering wheat growth, allowing diseased plants to compensate for

take-all injury. Susceptibility of root tissues to infection also
may have been reduced in some cases.

POSTEMERGENCE HERBICIDES AND TAKE-ALL DISEASE IN WINTER WHEAT:

ALTERATIONS IN THE INCIDENCE AND SEVERITY OF DISEASE

AND CROP GROWTH

by

Ray M. Geddens

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In closing, I would like to thank two very special people. I will always cherish the memory of my dear friend, Bill Morrow. Without his friendship and constant good humor, I doubt that I would have survived the harrowing experience of adolescence. Finally, I will be eternally grateful to Lori Johnson who has given me love and hope for the future.

Postemergence Herbicides and Take-All Disease of Winter Wheat:

Alterations in the Incidence and Severity of Disease and Crop Growth.

INTRODUCTION

Take-all, caused by the fungus <u>Gaeumannomyces graminis</u> (Sacc.)

Arx and Olivier var. <u>tritici</u> Walker, is a major root disease of wheat

and other small grains in most areas of the world where these crops

are grown. Take-all is a constant threat to winter wheat production

in the Willamette Valley of western Oregon. The mild, wet winters in

this region favor not only spread of the disease but growth of

numerous species of weeds. Successful wheat production for Oregon

growers requires management of both take-all and weeds.

The phenoxyalkanoic herbicide, diclofop-methyl, commonly is

used by Northwestern wheat growers for control of wild oats and

annual ryegrass. Some wheat growers have suspected that

postemergence applications of diclofop-methyl and other herbicides

applied to winter wheat sometimes increase injury from take-all

disease. Phytotoxicity, noninjurious alterations in wheat growth, or

changes in microenvironment produced by herbicides applied for weed

control could predispose the plant to injury from diseases such as

take-all. Alternatively, growth stimulation or toxicity toward plant

pests may enhance the ability of the crop plant to withstand disease

stress. Interactions between other herbicides and plant diseases

have been well documented. Several herbicides increased take-all

injury in winter- and spring-sown cereals in Europe.

The research presented in Chapter 1 was undertaken to determine the influence of herbicides commonly used for postemergence weed control in winter wheat on the incidence and severity of take-all. Wheat growth was monitored in an effort to explain alterations in take-all and crop productivity associated with herbicide applications.

The research presented in Chapter 2 was undertaken to determine whether diclofop-methyl, applied at several rates and application dates, could affect take-all injury and winter wheat growth. Correlations between altered disease injury and plant growth, should they occur, would suggest possible mechanisms of interaction between the herbicide and disease. The potential for direct interaction between herbicide and pathogen in the soil would be evaluated by determining the sensitivity of the fungus to diclofop-methyl in vitro.

Chapter 1. Effects of postemergence herbicides on the incidence and severity of take-all disease in winter wheat.

Abstract. Eight postemergence herbicides were evaluated in 1982 to determine their influence on the severity of take-all disease caused by the fungus Gaeumannomyces graminis var. tritici in winter wheat. Mecoprop ((+)-2-(4-chloro-2-methylphenoxy)propanoic acid), difenzoquat (1,2-dimethyl-3,5-diphenyl-1H-pyrazolium), dinoseb amine (amine salt of 2-(1-methylpropyl)-4,6-dinitrophenol), and diclofop-methyl (methyl ester of (+)-2-[4-(2,4-dichlorophenoxy)phenoxy] propanoic acid) sometimes reduced the severity of take-all disease on the seminal and crown roots. All herbicides reduced the incidence of 'whiteheads' associated with take-all injury. Grain yields in diseased plots treated with mecoprop, difenzoquat, and dinoseb were greater than the untreated check. Mecoprop, difenzoquat, dinoseb, and diclofop-methyl were selected for further evaluation in 1983. The herbicides did not affect severity of disease on the roots, but whiteheads again were reduced. Low rates of any of the four herbicides often increased tiller density in diseased plots. The herbicides, however, had little effect on tillering in healthy wheat. Fresh weights of individual tillers generally were reduced in response to herbicide application. Differences among treatments diminished as the season progressed. Herbicides did not affect grain yield in 1983. Data suggest that herbicides reduced take-all disease by altering wheat growth, allowing diseased plants to compensate for take-all injury. Susceptibility of root tissues to infection also may have been reduced in some cases.

INTRODUCTION

Take-all, caused by the fungus <u>Gaeumannomyces graminis</u> (Sacc.)
Arx and Olivier var. <u>tritici</u> Walker, is a major root disease of wheat
and other small grains in most areas of the world where these crops
are grown (5). Take-all is a constant threat to winter wheat
production in the Willamette Valley of western Oregon. The mild, wet
winters in this region favor not only spread of the disease but
growth of numerous species of weeds. Successful wheat production for
Oregon growers requires management of both take-all and weeds.

Interactions between herbicides and plant diseases have been
well documented (1,2,6,10,14,19,20,21,27,29,40). Most of the
information related to the effects of herbicides on take-all disease
of cereal crops comes from Europe. In England, Salt reported that
mecoprop applied to wheat doubled the percentage of straws severely
infected with take-all (33). Later research, however, indicated that
neither mecoprop nor a MCPA ((4-chloro-2-methylphenoxy)acetic
acid)/TBA (2,3,6-trichlorobenzoic acid) mix affected the incidence of
take-all when compared with an infected, but unsprayed check (34).
Brooks and Dawson found that minimum-tillage wheat planted into
stubble that had been killed with paraquat (1,1'-dimethyl-4,4'-
bipyridinium ion) had fewer symptoms of both take-all and
strawbreaker footrot (<u>Pseudocercosporella herpotrichoides</u> (Fron)
Dei.) than wheat planted into conventionally tilled soil (7).
Paraquat did not affect either growth of the take-all fungus in vitro
or saprophytic survival in infested debris. They concluded that
restricted movement of take-all inoculum in minimum-tillage fields,
not paraquat use directly, accounted for disease reductions. Nilsson

reported several occasions in Sweden where herbicides intensified take-all. In three field trials conducted between 1963 and 1978, he found increased injury from take-all in barley treated with mecoprop (25,28). He also found that mecoprop stimulated in vitro growth of the fungus and disease incidence in spring wheat grown in the greenhouse (25). He attributed the increased level of take-all in treated plants to root malformations that allowed easier penetration of the pathogen into the root. In additional field experiments, Nilsson reported that a mixture of dichlorprop ((+)-2-(2,4-dichlorophenoxy)propanoic acid), MCPA, ioxynil (4-hydroxy-3,5-diiodobenzonitrile), and bromoxynil (3,5-dibromo-4-hydroxybenzonitrile) increased the incidence of both take-all and eyespot in winter wheat (26). A mixture of brompyrazon (5-amino-4-bromo-2-phenylpyridazin-3-one) and tricuron (= isonoruron; 3-(1 or 2-hexahydro-4,7-methanoindanlyl)-1,1-dimethylurea) increased take-all symptoms without affecting eyespot. In England, Tottman and Thompson observed an increase in take-all severity in field plots sprayed with either mecoprop, ioxynil, dicamba (3,6-dichloro-2-methoxybenzoic acid), or a dicamba/2,3,6-TBA/mecoprop/MCPA mixture (37). The percentage of roots infected by take-all was 50% to 60% greater in herbicide-treated plots than in the unsprayed check. Finally, Burgiel in Poland noted a decrease in the incidence of both take-all and strawbreaker footrot symptoms in winter wheat treated with a variety of herbicides (8). Applications of a mixture of nitrofen (2,4-dichloro-1-(4-nitrophenoxy)benzene) and linuron (N'-(3,4-dichlorophenyl)-N-methoxy-N-methylurea) consistently reduced footrot over three years of tests. Take-all reductions generally were small.

Some Pacific Northwest wheat growers have suspected that postemergence herbicides applied to winter wheat sometimes increase injury from take-all disease. Research was undertaken to determine the influence of herbicides commonly used for postemergence weed control in winter wheat on the incidence and severity of take-all. Wheat growth was monitored in an effort to explain alterations in take-all and crop productivity associated with herbicide applications.

MATERIALS AND METHODS

General. Two field experiments were established at Hyslop Research

Farm, near Corvallis, Oregon, in 1982 and 1983. Levels of take-all

disease were established by incorporating coarsely ground oat seed,

either sterile or colonized with the take-all fungus, into the soil

prior to seeding. Inoculum was prepared using a single, highly

virulent isolate of Gaeumannomyces graminis var. tritici ('Jakes

Hill' isolate, Dept. of Botany and Plant Pathology, Oregon State

University). All plots received the same total quantity of ground

oats, but the proportion of colonized oats varied. Inoculum was

spread evenly over each plot by hand, then incorporated to a depth of

8 to 12 cm with a Rototerra power tiller.

Both experiments were conducted on a Woodburn silt loam (fine-

silty, mixed, mesic Aquultic Argixeroll). 'Stephens' winter wheat

was planted in mid-October at 100 kg seed/ha at a depth of 3 to 5 cm

in 18-cm rows. Both experiments received a preemergence application

of diuron (N'-(3,4-dichlorophenyl)-N,N-dimethylurea) at 1.8 kg ai/ha

and a postemergence application of bromoxynil at 0.6 kg ai/ha to

eliminate weed competition as a factor influencing crop growth and

disease development. In 1983, chlorsulfuron (2-chloro-N-[[(4-

methoxy-6-methyl-1,3,5-triazin-2-yl)amino]carbonyl]benzene-

sulfonamide) at 16.8 g ai/ha was applied for additional broadleaf

weed control. All herbicide treatments were applied with a unicycle

sprayer equipped with compressed air and a 2.4-m boom. Herbicides

were applied in 234 L/ha water carrier at 124 kPa nozzle pressure.

Fertility regimes for both experiments were based on previous crop

management. In 1982, 336 kg/ha 16-20-0 was applied preplant

incorporated (PPI), with an additional 123 kg N/ha as urea in a spring topdressing. In 1983, 224 kg/ha 16-20-0 PPI and 118 kg N/ha as urea in the spring were applied.

Take-all disease on the roots could be assessed reliably only by direct examination. Two root cores per plot, each core a 12-cm square centered on the row, were dug to a depth of 10 cm. After soaking overnight in water, cores were washed of soil and debris, then stored in a freezer until evaluation. Take-all severity was measured by assigning each sampling unit, whether whole plants in 1982 or single tillers in 1983, a percent attack (PA) value. The PA represented a visual estimate of the percentage of total root length with black lesions characteristic of take-all infection. Estimates of both disease severity on the roots and whitehead incidence followed the logistic rating scale proposed by Zadoks (41). After disease assessments, plants were saved for measurement of root growth. Relative root weight was measured by weighing the same portion of the root system of each plant or tiller in relation to the crown. Roots were cut 6 cm below the first node with crown roots. Culms were removed 1 cm above this node. Samples were placed in an oven at 60 C for 72 h prior to weighing. Root weights were determined once for each experiment.

The incidence of take-all disease was visually estimated in late summer as the percentage of grain heads in each plot with the bleached, 'whitehead' appearance often associated with take-all injury. Plots were harvested in late summer with a small-plot combine.

Data were evaluated in an analysis of variance with partitioning of error terms as appropriate for the experimental design. When indicated as statistically significant at the 10% or lower level of probability in the analysis of variance, main effects or interaction means were separated using Fisher's protected LSD (F-LSD).

When appropriate, the sum of squares for main effects and interactions from the analysis of variance were partitioned into single degree-of-freedom, orthogonal contrasts for treatment comparison or regression analysis (11). Orthogonal polynomial coefficients were used for regression analysis where treatments represented graded levels of a quantitative variable, such as herbicide application rates or inoculation rates.

Screening Of Cereal Herbicides (1982). A split-plot experiment in five replications with two levels of disease inoculum as main plots and nine herbicide treatments as subplots was installed in October, 1982. Inoculum rates were 0 and 100 kg/ha colonized, ground oat seed. Herbicide treatments included barban (4-chloro-2-butynyl-3-chlorophenylcarbamate), dicamba, diclofop-methyl, difenzoquat, dinoseb, mecoprop, metribuzin (4-amino-6-(1,1-dimethylethyl)-3-(methylthio)-1,2,4-triazin-5(4H)-one), and terbutryn (N-(1,1-dimethylethyl)-N'-ethyl-6-(methylthio)-1,3,5-triazine-2,4-diamine), plus an untreated check. All herbicides except mecoprop were registered for weed control in wheat in Oregon at the time of this research. Mecoprop was selected for evaluation because of the many published reports from Europe describing its effect on take-all.

The experimental site was fallow in the previous year. The soil had a 1:2 soil:water pH of 5.8, and 23 mg soil organic matter kg^{-1} soil in the surface 20 cm. Levels of major nutrients were adequate for wheat production. Herbicide treatments were applied on January 11, 1983, to 2.4 m by 7.6 m plots. Root growth and take-all severity were evaluated on April 4 and July 6. At each sampling date, two plants were randomly selected from each core for evaluation. Take-all severity was estimated and root numbers were counted for both the seminal and crown root systems. In addition to root numbers, the numbers of mature and immature tillers per plant were counted. Immature tillers were designated as those tillers that did not elongate.

The incidence of whiteheads was visually assessed on June 27. Plots were harvested on August 1. Grain was cleaned of debris prior to yield and test weight measurement.

Rates of disease development over the seminal and crown roots in inoculated plots were calculated as suggested by Vanderplank (39). Apparent infection rates for each plot were calculated as follows:

apparent infection rate =
$$[\ln (100-PA1)/(100-PA2)]/(t2-t1)$$

where PA1 and PA2 represent the percent attack values and t1 and t2 the number of days since planting for sample dates 1 and 2, respectively. An insignificant PA of 0.0001% was added to all values before calculating the infection rates in order to avoid errors when PA values equalled zero. Vanderplank also suggested that a

modification of this procedure may be useful to compensate for new host tissues produced during the period over which disease development was measured (39). Corrected infection rates were calculated as follows:

corrected apparent infection rate =
$$[\ln [(n2/n1) \times (100-PA1)/(100-PA2)]]/(t2-t1)$$

where n1 and n2 represent the number of either seminal or crown roots present at sample dates 1 and 2, respectively.

Evaluation Of Selected Herbicides (1983). Herbicides with the greatest influence on take-all disease and crop growth in 1982 were selected for further evaluation in 1983. The experiment used a split-split plot design in four replications. Levels of take-all disease were established by incorporating 0, 45, or 90 kg/ha ground, colonized oat seed into each main plot. The four herbicides evaluated were diclofop-methyl, dinoseb, mecoprop, and difenzoquat as subplots. Sub-sub-plots were three rates of each herbicide, plus an untreated check. Herbicide rates corresponded to multiples of 0.5, 1.0, and 1.5 times a standard rate commonly used for weed control in wheat in Oregon (Table 1.5).

The experimental site had been planted with a variety of perennial grasses in the previous year. The soil had a 1:2 soil:water pH of 7.0, and 23 mg soil organic matter kg^{-1} soil in the surface 15 cm. Levels of major nutrients were adequate for wheat production. Individual plots were 3.1 m by 6.1 m. Herbicides were applied to the wheat on January 11, 1984, when one to two tillers

were present. Fresh weight samples were collected on April 23 (two
to three nodes), June 4 (early anthesis), and July 3 (medium milk).
Two subsamples of 0.3 m of row each were taken from each plot.
Plants were cut 5 cm above the soil surface. Fresh weight and tiller
number per subsample were immediately determined. Root cores were
dug on May 10 ($\pm$ 7 days). All plants present in each core were
evaluated in this experiment, rather than selecting individual plants
as previously described. PA values designating take-all severity
were measured on a single-tiller basis. PA per tiller and the
percentage of tillers per core with disease were calculated from this
information. Relative root dry weight was measured on a single-
tiller basis. The incidence of whiteheads was estimated at the time
of the final fresh weight sampling. Plots were harvested on August
1.

RESULTS

Take-All Severity And Infection Rates. Of the herbicides evaluated
in 1982, only mecoprop, difenzoquat, diclofop-methyl, and dinoseb
reduced the severity of root infection by take-all (Table 1.1).
Mecoprop produced the most consistent effects, reducing the severity
of infection on both the seminal and crown roots. Restriction of the
rate of disease development over the growing season from mecoprop
application was evident from the reduction in apparent infection
rates for both root systems. Difenzoquat also reduced disease
severity for both seminal and crown roots, but reductions were
restricted to the late-season evaluation. Difenzoquat reduced the
rate of disease progress on the seminal roots only. Diclofop-methyl
reduced root infection on the seminal roots when evaluated late in
the season and on the crown roots at the early evaluation date.
Dinoseb reduced the level of infection only on the crown roots at the
final evaluation date. Neither diclofop-methyl nor dinoseb affected
the apparent infection rates. Terbutryn increased the severity of
take-all, but only on the crown roots at the early sampling date.
Correction of apparent infection rates for new root production had
little effect on the relative ranking of herbicides. Uncorrected
rates suggest that disease proceeds more slowly on the crown roots
than on seminal roots. After correction, however, infection rates on
the seminal and crown root systems often were similar.

In 1982, mecoprop, difenzoquat, diclofop-methyl, as well as
barban and terbutryn, reduced the incidence of strawbreaker footrot,
but only in uninoculated plots. Footrot was more severe in

uninoculated plots than in plots inoculated with the take-all

pathogen (Appendix Table 4).

None of the four herbicides evaluated in 1983 altered either

the incidence or severity of take-all disease in root evaluations.

Whitehead Incidence. All of the herbicides evaluated in both 1982

and 1983 reduced the incidence of whiteheads when compared to a

diseased, but unsprayed check. Mecoprop, difenzoquat, and dinoseb

had the greatest effect in 1982 (Table 1.1). Whitehead reductions in

1983 were directly proportional to the rate of herbicide applied,

regardless of the specific herbicide (Table 1.5, Figure 1.1). The

significant 'inoculation x rate' interaction (Table 1.6) indicated

that the effect of herbicide rate on disease incidence, however,

changed as inoculation rates increased. The nonsignificant three-way

interaction suggested that the herbicides were sufficiently alike in

their effects on disease incidence at each inoculation rate to allow

pooling of their values into a general herbicide rate effect. Two

points were apparent when the regression coefficients of the

equations (Figure 1.1) were examined. When no herbicides were

applied, disease incidence was approximately twice as great in the

plots receiving 90 kg/ha as in those receiving only 45 kg/ha of

inoculum. Also, the efficacy of herbicide applications in reducing

disease incidence became greater as inoculation rates, and presumably

take-all stress, increased.

Root Growth. In 1982, none of the herbicides affecting take-all

severity on the roots altered the number of either seminal or crown

roots present per plant (Table 1.2,1.3). Though not statistically

significant, crown root numbers measured on July 6 in inoculated

plots treated with diclofop-methyl, dinoseb, metribuzin, and terbutryn were substantially higher than in the inoculated, but unsprayed check (Table 1.3). The consistency of terbutryn in stimulating crown root growth in both uninoculated and inoculated plots may explain its effect on yield (Table 1.4). Difenzoquat apparently stimulated crown root growth, but only in the uninoculated plots. Barban reduced the number of crown roots early in the season, without affecting root symptoms. Root number was not evaluated in the 1983 trial. Only mecoprop increased relative root dry weight of diseased plants (Table 1.2). Barban reduced root weight, possibly through its effect on crown root number. In 1983, herbicides had no consistent effect on relative root weight of individual tillers.

Shoot Growth. None of the herbicides affecting take-all severity on the roots in 1982 influenced the number of tillers produced per plant (Table 1.2, 1.3). Barban reduced the number of mature tillers when evaluated on April 4 (Table 1.2). Reductions were similar in both uninoculated and inoculated plots. Suppression of tiller production may be responsible for the reduction of both crown root number and root weight in plots treated with this chemical. More immature tillers were found in dicamba-treated plots on July 6 (Table 1.3). The number of immature tillers was higher primarily in inoculated plots treated with this herbicide. Both barban and dicamba affected healthy and diseased plants in a similar manner. Symptoms of phytotoxicity from dicamba were noted early in the season in both diseased and healthy plots. Treated plants were stunted, with twisted, abnormal foliage. Dicamba injury also was reflected in

grain yield and test weight reductions in both healthy and diseased plots (Table 1.4).

Of the herbicides responsible for reductions in take-all on the roots, mecoprop, difenzoquat, and dinoseb stimulated grain yields in diseased plots (Table 1.4). Mecoprop and difenzoquat also increased test weights. Yield also was higher than the untreated check in plots receiving terbutryn, which had caused a small increase in take-all on the roots earlier in the season.

In 1982, inoculation of plots with the take-all pathogen reduced grain yield approximately 30%, when averaged over all herbicide treatments (Figure 1.6).

Crop growth and development were monitored more closely in 1983 than in the previous year. Growth reductions from take-all infection were considered important symptoms of disease, though more subtle than root lesions and whiteheads. The effects of herbicides on plant growth were most conspicuous at the first sampling date, April 23 (Table 1.5). Differences among the herbicide treatments diminished at later sampling dates. A severe epidemic of leaf blight (Septoria tritici Rob. in Desm.) in the spring may have contributed to the high variability in fresh weights noted at later sampling dates.

Though tiller density, fresh weight accumulation, and tiller weight were affected by herbicide application, the interactions between the level of take-all disease (reflected in the rate of inoculation), the specific herbicides applied, and the rate of application were complex (Table 1.6).

In regard to tiller density, the significant 'herbicide x rate' interaction (Table 1.6) indicated that the effect of increasing

application rate depended upon the specific herbicide applied. The response of tiller density to increasing application rates of each herbicide were sufficiently similar at each level of inoculation to allow the pooling of data for the analysis (Figure 1.3). Difenzoquat stimulated tiller production at all rates, while mecoprop inhibited it. Diclofop-methyl and dinoseb stimulated tillering at all rates when compared with the untreated check, but the stimulations were lower at rates above 1.0X.

The significant 'inoculation x rate' interaction in the analysis of tiller density (Table 1.6) further indicated that the effect of increasing application rates on tillering also depended on the rate of inoculation with the pathogen. The responses of tiller density to increasing application rates at each level of inoculation were sufficiently similar for each herbicide to allow pooling of data for the analysis (Figure 1.2). In unsprayed plots, tiller densities tended to decrease as more take-all inoculum was added to the soil. When plots were uninoculated, increasing the rate of herbicide application had little effect on tillering. If plots were inoculated, however, application of herbicides tended to increase tiller densities to values comparable to the uninoculated, unsprayed check. The greater the level of stress from take-all, reflected in the rate of inoculation, the greater the magnitude of the response to herbicide application. The maximal response of tillering to herbicide application in inoculated plots occurred at the 1.0X rate.

With minor variations, fresh weight production per unit area responded to increasing rate of herbicides in a manner similar to tiller density (Table 1.5).

The significant 'herbicide x rate' interaction in the analysis of tiller weights (Table 1.6) indicated that the effect of increasing application rate on this variable depended upon the specific herbicide applied. The relationship between herbicides and their rates of application were sufficiently alike at each level of inoculation to allow the pooling of data for the analysis (Figure 1.4). All herbicides tended to reduce tiller weights. Difenzoquat substantially reduced the weight of individual tillers at rates above 0.5X. Reductions for the other herbicides were small, even at the highest rate.

None of the herbicides evaluated in 1983 affected grain yield.

In regard to the efficacy of artificial inoculation in generating disease, visible symptoms of take-all injury were evident by the harvest in all inoculated plots. In both 1982 and 1983, severity of take-all disease on the roots and the incidence of both diseased roots and whiteheads were directly proportional to the rate of inoculum incorporated into the soil prior to planting (Figure 1.5). Grain yields were inversely proportional to inoculation rate (Figure 1.6). Yield in the absence of take-all inoculum was higher in 1982 than in 1983. Inoculum also was more detrimental to yield in 1982.

DISCUSSION

Interpretation of much of the published research on interactions between herbicides and take-all disease is difficult. Generalizations regarding herbicidal effects on disease sometimes were based upon in vitro toxicity tests, with little consideration for the complexity introduced into the field by the host plant, soil, and climate. The short-term, possibly transient, effects of herbicide treatment noted in greenhouse experiments sometimes were extrapolated to the field situation. Conclusions often were based on disease responses to herbicide rates much higher than would normally be applied.

In regard to take-all, reports in the literature quantified disease in many different ways. The distinction between disease incidence and severity as emphasized by James (17) often was not clear. The critical role of host root growth to survival and spread of soil-borne pathogens often was ignored (16). Inconsistent results both within and between cropping seasons were observed in several studies. These inconsistencies may have been related to the highly variable natural infestations under which the experiments were conducted.

Experimental procedures reported in the literature often were questionable. Experiments devoted to other purposes were sometimes used for evaluation of herbicide effects on take-all. In one case, observations on disease in a portion of the field outside the experimental area were incorporated into the analysis. Adequate experimental checks were not included in some experiments. Two check treatments, both diseased/unsprayed and healthy/sprayed, are

necessary to reliably distinguish additive from interactive effects
of herbicide and disease.

Many of these problems as well as suggestions for future
research have been discussed by Domsch (9). Personal experience
suggests that interactions in the field are often so complex as to be
overwhelming, even when all factors of interest are under strict
control of the experimenter. We do not claim to have solved all of
these problems. Control over the experimental material often is
gained only at the expense of increasing artificiality. Cognizance
of these problems, however, has dictated many of the procedures
utilized in this research.

Herbicides applied for weed control could affect a soil-borne
disease like take-all in numerous ways (1,19). Herbicides present in
the soil may affect the take-all pathogen directly by influencing its
survival prior to infection. Three isolates of the take-all fungus
were sensitive to in vitro concentrations of diclofop-methyl greater
than approximately 5 ppm (see Chapter 2). Both difenzoquat (38) and
dinoseb (24,30) have demonstrated fungicidal properties in relation
to plant diseases. Dinoseb, however, also has been implicated as a
factor predisposing plants to disease injury (32). The effects of
the herbicides evaluated in this research on survival of the take-all
fungus in the soil prior to infection of the host are not known.

Alternatively, herbicides may influence disease indirectly by
affecting host growth. For example, the susceptibility of tissues to
infection may be altered by herbicides. Herbicides with growth
regulatory properties also may inhibit or stimulate root and shoot

growth, and in turn influence the ability of the plant to compensate for loss of diseased tissues.

Based upon the increase in relative root weight from mecoprop application noted in 1982, mecoprop may affect root morphology more than root and shoot growth. Mecoprop did not affect either tiller or root numbers in 1982. This herbicide also did not affect tiller development in wheat with take-all as much as the other herbicides evaluated in 1983. Increased root weight with little effect on root number suggests that mecoprop stimulated either the size of individual roots or the degree of secondary branching. Direct examination of roots from treated plants support this conclusion. When examined under a dissecting microscope prior to weighing, roots of mecoprop-treated plants were thicker, with longer, more extensively branched secondary roots, than roots from the untreated check.

Several researchers have noted alterations in root growth and morphology from mecoprop application (12,25,28,35). These short-term toxicity studies indicated that mecoprop reduced elongation of primary roots and injured the meristems of secondary roots. Root weight often was reduced by this acute injury. Nilsson attributed the higher incidence of take-all in mecoprop-treated cereals to root injury allowing easier penetration of the root by the pathogen (25). The long-term effects of root pruning, however, are unpredictable. Stunted secondary roots may branch more prolifically and ultimately produce more root tissue than would normally occur.

The related phenoxy herbicide, 2,4-D, apparently affects root morphology in a manner similar to that noted for mecoprop (18).

Enlarged cortical cells were observed in the region behind the apical meristem in roots of plants treated with this herbicide. Mecoprop may affect development of the root cortex as well. Penetration of the cortex by the take-all fungus is a prerequisite to further invasion of the vascular tissues within the stele. Altered cortical growth resulting from mecoprop application may slow the rate of cellular penetration and colonization by the pathogen. Reductions in apparent infection rates on the seminal and crown roots by mecoprop treatment support this hypothesis. Possible alterations include proliferation of cellular layers within the cortex, the degree or rate of senescence in cortical tissues, or thickening of cell walls. In an analogous situation, reduction in the number of root knot nematode galls in onions treated with DCPA was attributed to alteration in the epidermal tissues of the root making stylet penetration and gall establishment more difficult (3).

Rates of disease development, reflected in the apparent infection rates, should be lower in plants less susceptible to infection. Treatments that stimulated crop growth, however, could reduce ratings of disease severity without affecting the absolute amount of infected tissue. Diseased tissues in this case would be diluted by new root growth. For this reason, infection rates were corrected for differences in root number. Only mecoprop consistently retarded the corrected rate of disease development on both the seminal and crown roots. This suggests that reductions in take-all disease sometimes observed in plots treated with difenzoquat, diclofop-methyl and dinoseb were produced in some way other than by alterations in host susceptibility.

Vegetative growth of diseased plants in 1982 apparently was sensitive to applications of several herbicides. When crop development was followed more closely in 1983, growth alterations were noted for all four of the herbicides evaluated that year. The growth response to herbicide application depended on the level of take-all induced stress. This suggests that infection by the pathogen predisposes the plant to react to the herbicides in a manner different from that of a healthy plant.

Tiller production in infected plants apparently was very sensitive to herbicide application, especially to difenzoquat, diclofop-methyl, and dinoseb. Both difenzoquat and diclofop-methyl have been reported to stimulate tiller production in winter wheat (36). Dose-response studies with diclofop-methyl have demonstrated that this herbicide can stimulate the growth of primary and secondary tillers, the total number of tillers per plant, and crown root production (Appendix Tables 24, 27).

Delay or abortion of tillers in wheat apparently is a general response to stress (22). Root and shoot growth in wheat also are highly interdependent (23). Take-all infection of the roots reduces the availability of water and nutrients to the shoots. Ultimately, shoot growth will be inhibited in diseased plants (4). When averaged over all herbicide treatments, inoculation of plots with the take-all pathogen in 1982 reduced the number of mature tillers at both sampling dates (Table 1.2, 1.3). Immature tiller numbers also were reduced, but only early in the spring. By mid-summer, the number of immature tillers per plant was higher in inoculated plots than

uninoculated plots. This suggests that take-all disease slowed the rate of tiller initiation and development.

Results from 1983 support this conclusion. Inoculation with the take-all pathogen tended to reduce tiller density early in the spring, with little effect on tiller weight. By late summer, however, tiller densities in inoculated plots were similar to those in uninoculated plots, but tiller weights were reduced (Appendix Tables 15,16).

These data suggest that herbicides applied early in the vegetative phase of wheat development may stimulate the initiation of tiller buds suppressed by early-season take-all infection. The effect of herbicides applied to wheat infected by take-all may be the restoration of the plant's normal pattern of development. Herbicides applied to healthy plants would have less effect on tiller production because fewer tiller buds would be inhibited by root stress.

Take-all becomes more severe in late spring when soil temperatures rise and vegetative growth of the crop ceases (7). Summers in Western Oregon usually are very dry. The combination of increased disease pressure and drought stress makes the establishment of a healthy root system early in the season critical to successful wheat production. Earlier tillering and root development in infected plants treated with postemergence herbicides may allow deeper penetration of roots into the soil before late-season disease and drought stress begins. Improved root health could account for the reduction in whiteheads observed in plots treated with these herbicides.

The results of these experiments must be interpreted with
caution. The herbicides were tested against a single cultivar of
winter wheat inoculated with a single isolate of the pathogen. The
choice of site and inoculation procedure insured that the influence
of indigenous strains of the fungus would be minimized. Both
experiments were conducted at the same site, under near optimal
management and environmental conditions. Unlike many wheat-producing
regions, the Willamette Valley of western Oregon usually has ample
rainfall through most of the growing season. Soil moisture reserves
are usually adequate to maintain healthy growth throughout the two to
three dry months prior to harvest. Prolonged moisture stress could
dramatically alter the response of the plant to herbicides and
disease. Late-season herbicide applications, or high application   .
rates may injure the crop and in turn affect disease (see Chapter 2).
The long-term effects of herbicides on the amount of inoculum
returned to the soil in the form of infested plant debris after
harvest may be important. Small reductions in the amount of diseased
tissue attributable to herbicide application may not consistently
affect final yield, but could substantially reduce the amount of
take-all inoculum left in the field. Finally, interactions between
herbicides, take-all, and other plant diseases undoubtedly are
complex. Reductions in the incidence of strawbreaker footrot from
applications of substituted-urea and triazine herbicides have been
reported by other researchers (13,15). Results from the 1982
experiment suggest that this pathogen may be sensitive to other
herbicides as well. Reductions in footrot incidence noted in plots
inoculated with the take-all fungus are not without precedence as

well. Antagonism between <u>Pseudocercosporella herpotrichoides</u> and

other pathogenic fungi infecting the roots and lower shoots of

cereals has been reported (31).

Regardless of mechanism of interaction, application of these

herbicides at recommended rates under climatic and management

conditions similar to those encountered in this research should not

increase disease injury from take-all. In some instances, take-all

disease and injury to the wheat may be reduced.

Table 1.1. Effects of postemergence herbicides on the incidence and severity of take-all disease and the rate of disease development in winter wheat (1982).

Treatment	kg ai/ha	Estimated PA[1] (April 4) Seminal roots 0	Crown roots 100	Seminal roots 0	Crown roots 100 Inoculum (kg/ha)	Estimated PA (July 6) Seminal roots 0	100	Crown roots 0	100	Apparent infection rates[2] Seminal roots Uncorrected	Corrected	Crown roots Uncorrected	Corrected	Whiteheads/ plot[3] (%) Inoculum 0	100
Check	-	0	13.6	0	4.8 bcd[4]	0.5	84.1 a[4]	2.2	78.7 a[4]	.028 ab[4]	.028 a[4]	.018 ab[4]	.026 abc[4]	0	52 a[5]
barban	0.4	0	13.3	0	2.6 def	0.5	83.7 a	0.5	79.5 a	.025 b	.025 a	.019 ab	.028 ab	0	33 bc
dicamba	0.3	0	9.4	0.1	6.2 abc	0.6	73.6 ab	1.9	75.8 ab	.016 c	.016 b	.017 ab	.025 abc	0	33 bc
diclofop-methyl	1.4	1.8	7.5	0.5	1.1 ef	2.9	70.7 b	3.5	71.8 ab	.026 b	.027 a	.016 ab	.023 bc	0	40 b
difenzoquat	1.1	0	14.8	0	3.5 cde	0.1	52.3 c	0.4	66.0 b	.014 cd	.014 bc	.015 b	.021 cd	0	18 d
dinoseb	1.7	0.5	13.4	0.1	3.0 def	1.3	84.5 a	0.9	66.5 b	.025 b	.025 a	.015 b	.025 abc	0	27 c
mecoprop	2.5	0	11.9	0	0.4 f	2.8	36.3 d	0.8	41.3 c	.007 d	.008 c	.007 c	.015 d	0	5 e
metribuzin	0.3	0	11.3	0	7.1 ab	0.7	83.0 ab	0.5	79.8 a	.028 ab	.027 a	.020 ab	.028 ab	0	31 bc
terbutryn	1.8	0.8	15.6	0	8.1 a	0	83.6 a	0.1	74.4 ab	.034 ab	.033 a	.021 a	.030 a	0	36 b

[1]Disease severity measured as a 'percent attack,' denoting the visually estimated percentage of the respective root system with black lesions characteristic of take-all disease.

[2]Rates of disease development on the roots, denoted by the apparent infection rate, either uncorrected or corrected for new root production; rates were calculated for inoculated plots only; computational procedure described in the text.

[3]Disease incidence denoted by the visually estimated percentage of grain heads in each plot with the bleached, 'whitehead' appearance.

[4]Means within a column followed by the same letter are not significantly different at the 10% level of probability as determined by the F-LSD.

[5]Means within a column followed by the same letter are not significantly different at the 5% level of probability as determined by the F-LSD.

Table 1.2. Effects of postemergence herbicides on the vegetative growth of winter wheat with take-all disease when evaluated on April 4 (1982).

Treatment	kg ai/ha	Seminal roots/plant			Crown roots/plant			Relative root wt (mg)[1]			Tillers/plant					
											Immature[2]			Mature		
		0	100		0	100		0	100		0	100		0	100	
							Inoculation rate (kg/ha)									
				M.E.H.[3]			M.E.H.			M.E.H.			M.E.H.			M.E.H.
Check	-	5.8	5.6	5.7	28.9	26.6	27.7 ab[5]	241	215	228 b	2.7	2.2	2.4	3.4	2.8	3.1 ab
barban	0.4	5.4	5.2	5.3	20.9	26.3	23.6 c	152	190	171 c	2.5	2.8	2.6	2.4	2.6	2.5 c
dicamba	0.3	5.9	5.7	5.8	30.1	25.8	27.9 ab	308	211	260 b	2.8	2.4	2.6	4.5	2.9	3.7 a
diclofop-methyl	1.4	5.1	5.5	5.3	27.4	29.4	28.4 ab	215	204	210 bc	2.9	2.7	2.8	3.0	3.0	3.0 bc
difenzoquat	1.1	5.8	5.3	5.5	30.7	30.7	30.7 a	226	215	220 bc	2.6	2.4	2.5	4.2	3.2	3.7 a
dinoseb	1.7	5.7	5.8	5.7	29.7	25.8	27.7 ab	233	200	217 bc	3.1	2.0	2.5	3.0	2.9	2.9 bc
mecoprop	2.5	5.9	5.7	5.8	32.9	28.1	30.5 a	376	280	328 a	2.7	1.7	2.2	3.5	2.8	3.2 ab
metribuzin	0.3	5.7	5.5	5.6	24.0	29.4	26.7 bc	167	258	212 bc	2.1	2.7	2.4	2.6	3.2	2.9 bc
terbutryn	1.8	6.0	6.2	6.1	27.9	28.6	28.2 ab	231	237	234 b	3.1	2.8	2.9	3.4	2.7	3.0 bc
M.E.I.[4]		5.7	5.6		28.0	27.8		239	223		2.7a[6]	2.4b		3.3 a	2.9 b	

[1]Relative root weight measured as the dry weight of tissues extending 1 cm above and 6 cm below the crown of the plant.

[2]Tillers that did not elongate were designated as immature.

[3]Main effects of herbicide application; effect of herbicide when averaged over both inoculation rates.

[4]Main effects of inoculation; effects of inoculation with the take-all fungus when averaged over both inoculation rates.

[5]Means within a column followed by the same letter are not significantly different at the 10% level of probability as determined by the F-LSD; absence of letters indicates no significant effect.

[6]Main effects of inoculation on numbers of both immature and mature tillers significantly different at the 5% level of probability.

Table 1.3. Effects of postemergence herbicides on the vegetative growth of winter wheat with take-all disease when evaluated on July 6 (1982).

Treatment	kg ai/ha	Seminal roots/plant			Crown roots/plant			Tillers/plant Immature[1]			Mature		
		0	100		0	100		0	100		0	100	
				Inoculation (kg/ha)									
				M.E.H.[2]			M.E.H.			M.E.H.			M.E.H.
Check	-	5.6	5.5	5.5	61.5	52.9	57.2	0.4	0.5	0.5 bc[4]	4.6	3.8	4.2
barban	0.4	5.7	5.5	5.6	54.2	54.3	54.3	0.6	0.7	0.6 b	4.4	4.0	4.2
dicamba	0.3	5.5	5.4	5.4	55.6	53.4	54.5	0.6	1.3	0.9 a	5.1	3.8	4.4
diclofop-methyl	1.4	5.7	5.7	5.7	63.1	61.2	62.1	0.6	0.5	0.5 bc	5.0	4.5	4.8
difenzoquat	1.1	5.9	5.4	5.6	72.4	53.7	63.1	0.4	0.4	0.4 c	5.3	3.5	4.4
dinoseb	1.7	6.0	5.6	5.8	56.3	65.2	60.8	0.3	0.4	0.3 c	4.4	4.8	4.6
mecoprop	2.5	5.7	5.8	5.8	58.0	57.8	57.9	0.4	0.4	0.4 c	4.6	4.4	4.5
metribuzin	0.3	5.9	5.6	5.7	52.8	63.4	58.1	0.2	0.6	0.4 bc	4.3	4.3	4.3
terbutryn	1.8	5.9	5.7	5.8	66.0	68.4	67.2	0.2	0.6	0.4 bc	5.1	5.0	5.0
M.E.I.[3]		5.7	5.6		60.0	58.9		0.4 a	0.6 b		4.7	4.2	

[1]Tillers that did not elongate were designated as immature.

[2]Main effects of herbicide application; effects of herbicides when averaged over both inoculation rates.

[3]Main effects of inoculation rate; effect of inoculation with the take-all fungus when averaged over all herbicide treatments.

[4]Means within a column or row followed by the same letter are not significantly different at the 10% level of probability as determined by the F-LSD; absence of letters indicates no significant effect.

Table 1.4. Effects of postemergence herbicides on grain yield and test weights of winter wheat with take-all disease (1982).

Treatment	kg ai/ha	Grain yield (kg/ha)		Test weight (kg/L)	
		Inoculation rate (kg/ha)			
		0	100	0	100
Check	-	7880 abc[1]	4940 d	58.1 ab	55.4 cd
barban	0.4	7510 c	5310 bcd	58.5 ab	56.2 bc
dicamba	0.3	5850 d	4370 e	54.3 c	54.7 d
diclofop-methyl	1.4	7710 bc	4790 de	58.9 a	56.2 bc
difenzoquat	1.1	8320 a	5660 bc	58.1 ab	57.8 ab
dinoseb	1.7	8170 ab	5600 bc	57.4 b	55.8 cd
mecoprop	2.5	7730 bc	6310 a	58.5 ab	58.5 a
metribuzin	0.3	8170 ab	5170 cd	58.5 ab	56.2 bc
terbutryn	1.8	8360 a	5800 ab	58.9 a	56.6 bc

[1]Means within a column followed by the same letter are not significantly different at the 5% level of probability as determined by the F-LSD.

Table 1.5. Effects of application rate for four postemergence herbicides on vegetative growth and disease incidence in winter wheat with take-all disease (1983).

Treatment	Relative rate (X standard)	Actual rate (kg ai/ha)	Tillers/0.3 m row[1]			Fresh wt/0.3 m row(g)			Tiller weight (g)			Whiteheads/plot (%)[2]		
			Inoculation rate (kg/ha)											
			0	45	90	0	45	90	0	45	90	0	45	90
diclofop-methyl	0	0	35.5	35.6	34.8	143	141	142	4.02	3.95	4.08	0	17.6	45.0
	0.5	0.70	39.8	37.4	36.9	161	150	143	4.04	4.03	3.88	0	11.3	30.0
	1.0	1.40	33.4	40.8	42.4	128	150	156	3.94	3.60	3.78	0	8.3	23.1
	1.5	2.10	36.3	37.9	34.6	131	160	129	3.62	4.17	3.76	0	6.4	20.0
difenzoquat	0	0	39.4	38.5	33.3	176	163	143	4.41	4.21	4.11	0	26.3	43.8
	0.5	0.56	42.0	38.5	36.9	181	160	151	4.36	4.12	4.34	0	17.5	41.9
	1.0	1.12	44.0	41.0	46.3	166	140	165	3.76	3.45	3.58	0	15.6	25.6
	1.5	1.68	43.5	42.9	47.1	136	137	140	3.14	3.30	2.98	0	8.1	24.4
dinoseb	0	0	38.4	32.4	32.9	155	136	139	4.04	4.26	4.38	0	27.5	33.1
	0.5	0.84	38.8	35.3	37.6	164	156	156	4.21	4.35	4.05	0	16.3	40.0
	1.0	1.68	37.4	40.5	40.8	146	172	164	3.95	4.24	4.08	0	12.5	27.5
	1.5	2.52	40.0	36.4	36.1	170	154	140	4.23	4.27	3.90	0	23.1	27.5
mecoprop	0	0	40.4	37.4	36.9	165	151	148	4.10	4.10	4.11	0	27.5	40.6
	0.5	1.23	36.4	39.9	39.0	147	159	152	4.12	3.93	3.93	0	23.1	25.6
	1.0	2.46	34.5	37.3	39.6	138	148	162	3.94	4.01	4.06	0	18.9	17.5
	1.5	3.69	35.5	35.5	38.8	128	134	143	3.55	3.81	3.69	0	11.4	21.3

[1]Vegetative growth evaluated on April 23.

[2]Disease incidence denoted by visually estimated percentage of grain heads in each plot with bleached, 'whitehead' appearance; evaluated on July 3.

Table 1.6. Effects of application rate for four postemergence herbicides on vegetative growth and disease incidence in winter wheat with take-all disease. Analysis of Variance.

Source	df	Tillers/0.3 m row			Fresh weight/0.3 m row (g)			Tiller weight (g)			Whiteheads/plot (%)		
		MS	F	p	MS	F	p	MS	F	p	MS	F	p
Block	3	183.0	2.28	0.18	70119.7	29.10	0.00	36.77	67.43	0.00	729.8	1.68	0.27
Inoculation rate	2	9.3	0.12	0.89	503.2	0.21	0.82	0.15	0.28	0.76	29759.6	68.35	0.00
Error (main plot)	6	80.3	-	-	2410.0	-	-	0.55	-	-	435.4	-	-
Herbicide type	3	353.1	7.15	0.00	2412.4	1.83	0.17	2.11	7.36	0.00	289.1	0.64	0.59
Inoculation x herbicide	6	42.7	0.86	0.53	1207.2	0.91	0.50	0.16	0.55	0.76	329.6	0.73	0.63
Error (split plot)	27	49.4	-	-	1321.2	-	-	0.29	-	-	449.8	-	-
Herbicide rate	3	210.3	3.94	0.01	3779.7	4.43	0.01	4.33	18.60	0.00	2068.7	19.78	0.00
Inoculation x rate	6	116.0	2.17	0.05	2006.1	2.35	0.04	0.29	1.25	0.29	628.3	6.01	0.00
Herbicide x rate	9	94.4	1.77	0.08	1098.2	1.29	0.25	1.36	5.85	0.00	94.6	0.90	0.52
Inoculation x herbicide x rate	18	33.0	0.62	0.88	814.6	0.96	0.52	0.15	0.65	0.85	119.5	1.14	0.32
Error (split split plot)	108	53.4	-	-	852.4	-	-	0.23	-	-	104.6	-	-
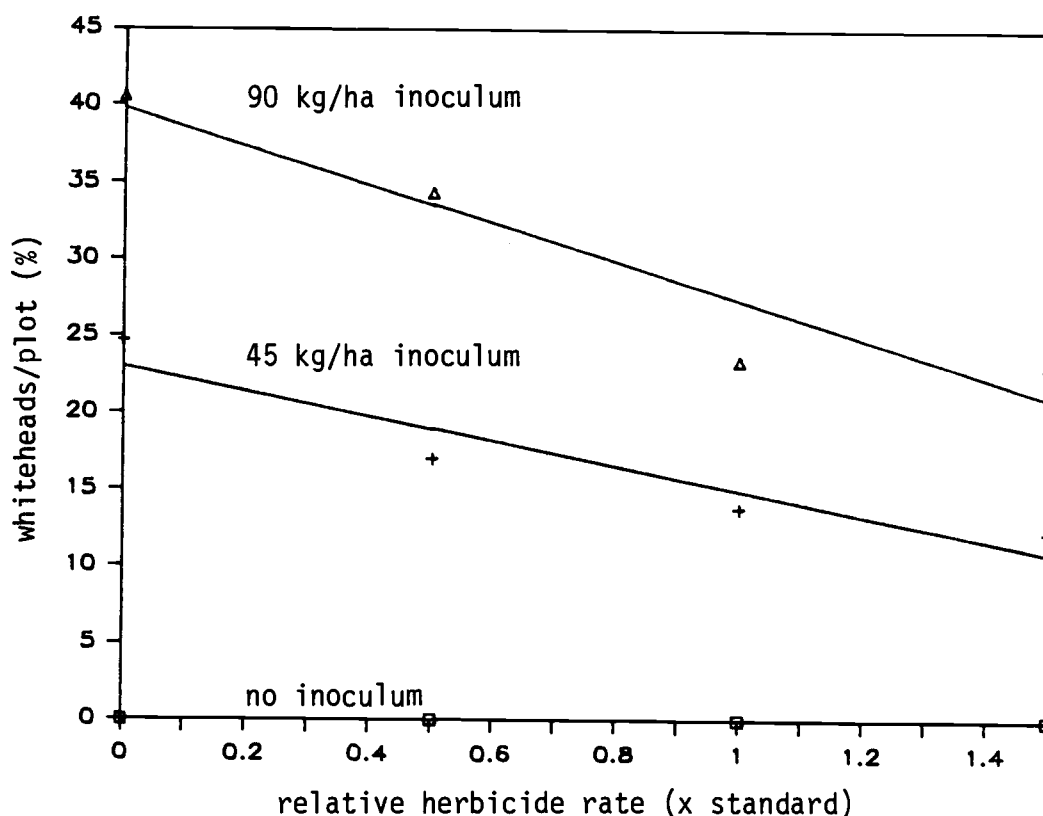
Figure 1.1. Effect of relative rates of herbicide application on the incidence of take-all disease in winter wheat at Hyslop Farm (1983). Plots were inoculated prior to planting with no inoculum (□), 45 kg/ha (+), and 90 kg/ha (∆) ground, colonized oats. The effect of increasing application rate on disease incidence was similar for four postemergence herbicides. Partitioning of the 'inoculation x rate' interaction (Table 1.6) into polynomial components indicated that the linear effect of relative rate on disease incidence changed when inoculation rates increased (p<.01).

no inoculum

     no 'whiteheads' observed

45 kg/ha inoculum

     whiteheads/plot(%)=23.1-8.1(relative rate) r^2=0.89

90 kg/ha inoculum

     whiteheads/plot(%)=39.9-12.6(relative rate) r^2=0.90
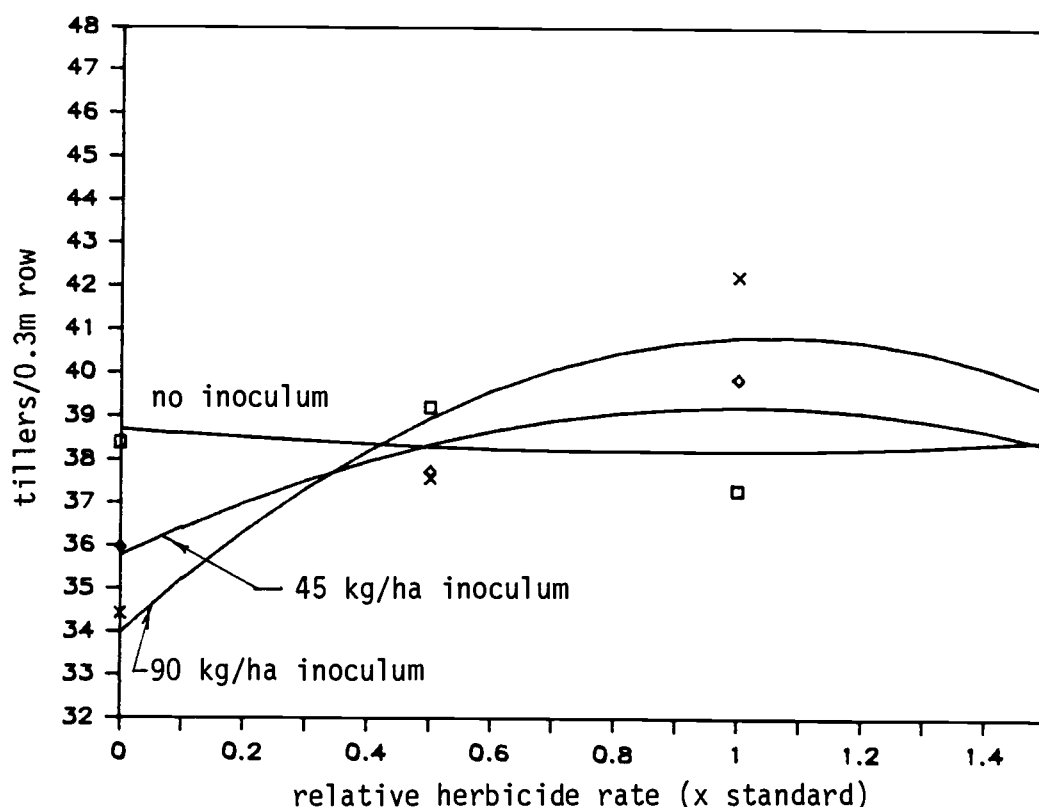
Figure 1.2. Effect of relative rates of herbicide application on tiller density of winter wheat with take-all disease (1983). Plots were inoculated prior to planting with no inoculum (□), 45 kg/ha (◊), and 90 kg/ha (X) ground, colonized oats. Partitioning of the 'inoculation x rate' interaction (Table 1.6) into polynomial components indicated that the quadratic response of tiller density to relative herbicide rate changed when inoculation rates increased ($.05 < p < .10$).

no inoculum

tillers/0.3m row=$38.7-1.2$(rel. rate)$+0.7$(rel. rate)^2 $r^2 = 0.07$

45 kg/ha inoculum

tillers/0.3m row=$35.8+7.0$(rel. rate)-3.5(rel. rate)^2 $r^2 = 0.89$

90 kg/ha inoculum

tillers/0.3m row=$34.0+13.1$(rel. rate)-6.2(rel. rate)^2

$r^2 = 0.86$
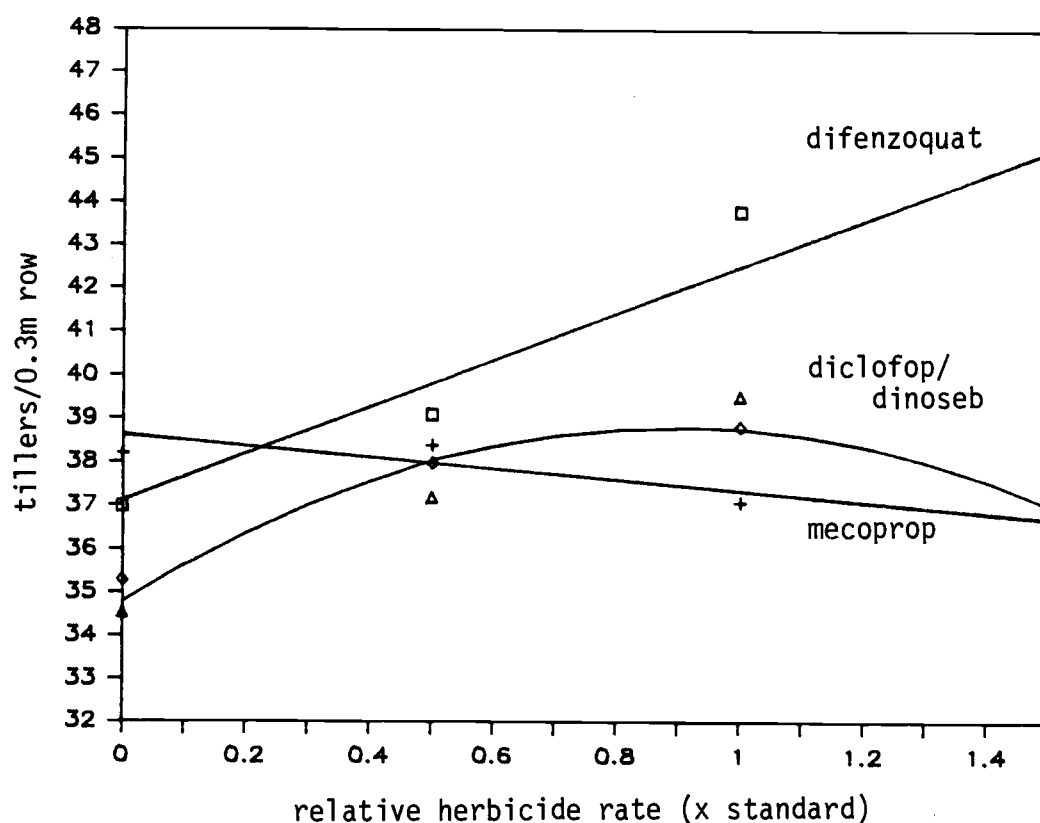
Figure 1.3. Effect of relative application rate of four post-emergence herbicides on tiller density of winter wheat (1983). Difenzoquat (□), mecoprop (+), diclofop-methyl (◊), and dinoseb (△) were evaluated. Partitioning of the 'herbicide x rate' interaction (Table 1.6) into orthogonal contrasts for the herbicides and polynomial components for rates indicated that the response of tiller density to relative application rate depended upon the herbicide applied. The linear response of tiller density to difenzoquat ($p < .01$) and mecoprop ($p < .10$) rates were different from each other and from the mean quadratic response to diclofop-methyl and dinoseb. There was no significant difference between responses to diclofop-methyl and dinoseb.

difenzoquat tillers/0.3m row=37.1+5.4(rel. rate) r^2=0.94

mecoprop tillers/0.3m row=38.5-1.2(rel. rate) r^2=0.83

diclofop-methyl/dinoseb

tillers/0.3m row=34.8+9.0(rel. rate)-5.0(rel. rate)^2

r^2=0.96
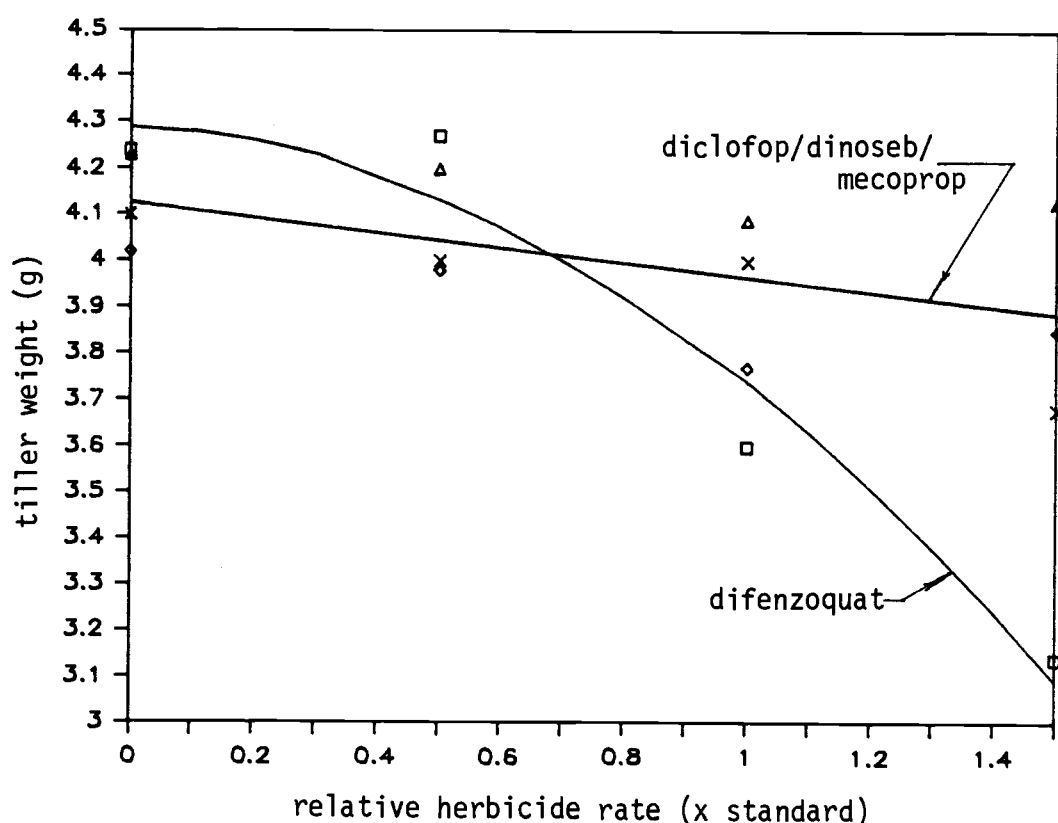
Figure 1.4. Effect of relative application rate of four post-emergence herbicides on tiller weight of winter wheat (1983). Difenzoquat (□), mecoprop (X), diclofop-methyl (◇), and dinoseb (△) were evaluated. Partitioning of the 'herbicide x rate' interaction (Table 1.6) into orthogonal contrasts for the herbicides and polynomial components for rates indicated that the response of tiller weight to relative application rate depended upon the herbicide applied. The quadratic response of tiller weight to difenzoquat rate was significantly different from responses to the other herbicides (p<.05). Responses to rates of diclofop-methyl, dinoseb, and mecoprop were similar.

difenzoquat tiller weight=4.3-0.1(rel. rate)-0.5(rel. rate)^2

$$r^2=0.95$$

diclofop-methyl/dinoseb/mecoprop

tiller weight=4.1-0.2(rel. rate) $r^2=0.99$
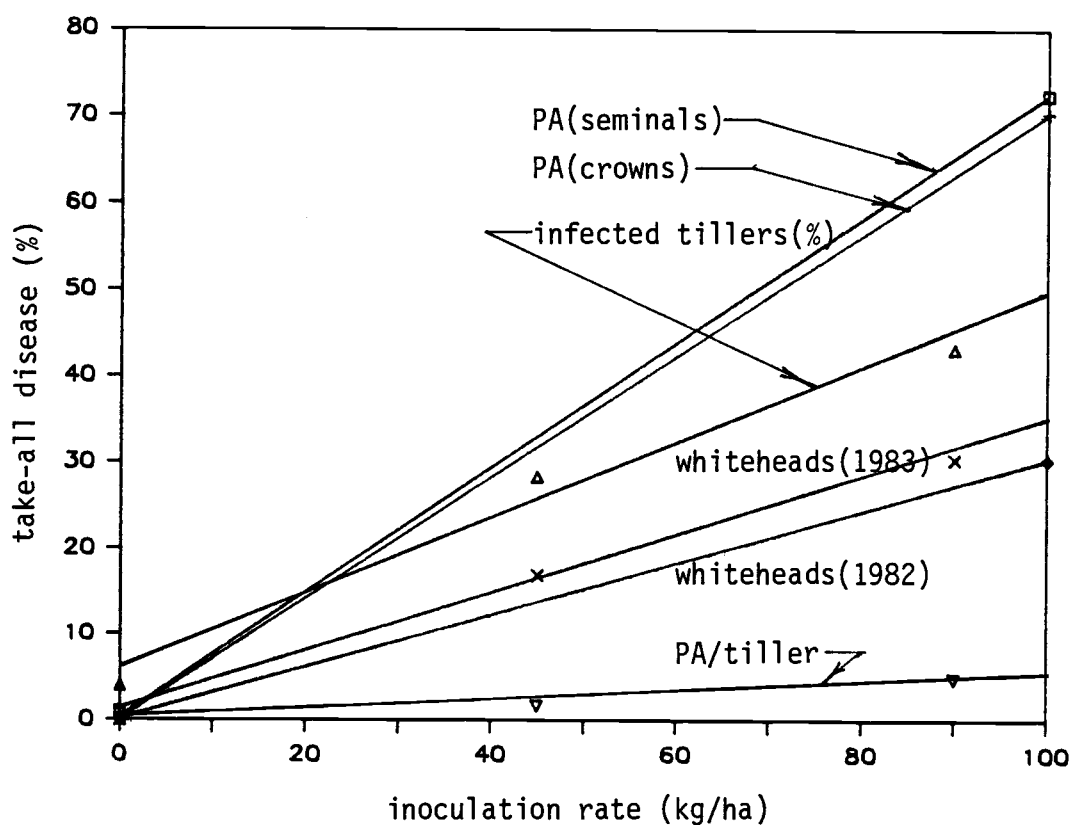
Figure 1.5. Effect of rate of inoculation with ground oats colonized by Gaeumannomyces graminis var. tritici isolate 'Jakes Hill' on disease incidence and severity in winter wheat grown at Hyslop Farm in 1982 and 1983.

1982

□ PA(seminal roots)=0.20+0.15(inoc. rate)

+ PA(crown roots)=0.23+0.15(inoc. rate)

◇ whiteheads(%)=0.00+0.30(inoc. rate)

1983

▽ PA/tiller=-0.17+0.05(inoc. rate) r^2=0.96

△ infected tillers(%)=5.58+0.44(inoc. rate) r^2=0.98

X whiteheads(%)=0.58+0.34(inoc. rate) r^2=1.00
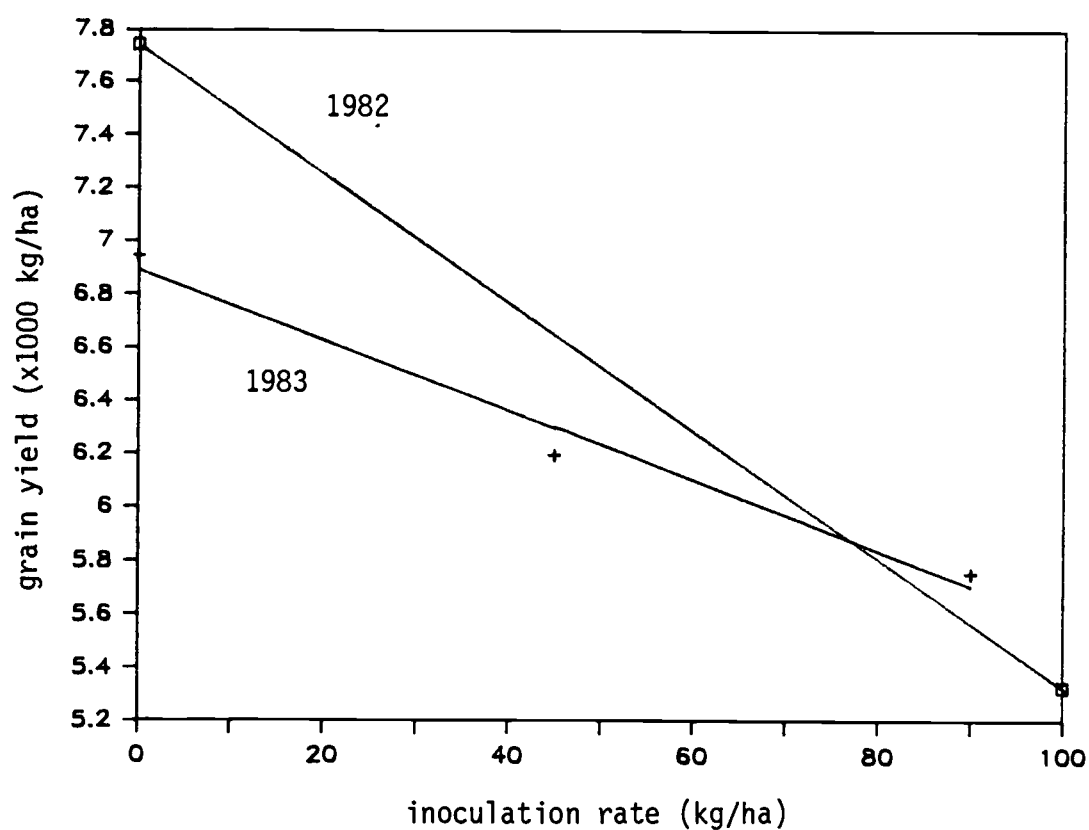
Figure 1.6. Effect of rate of inoculation with ground oats colonized by Gaeumannomyces graminis var. tritici isolate 'Jakes Hill' on grain yield in winter wheat grown at Hyslop Farm in 1982 and 1983.

1982

□ yield(kg/ha)=7750-25(inoc. rate)

1983

+ yield(kg/ha)=6900-13(inoc. rate) r^2=0.98

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Chapter 2. Effects of the herbicide diclofop-methyl on
Gaeumannomyces graminis var. tritici and take-all disease in winter
wheat.

Abstract. In vitro growth of three isolates of Gaeumannomyces
graminis var. tritici, the fungus responsible for take-all disease of
winter wheat, was not inhibited by diclofop-methyl (methyl ester of
(+)-2-[4-(2,4-dichlorophenoxy)phenoxy]propanoic acid) at
concentrations below 10 μM. Postemergence application of diclofop-
methyl at 1.12 kg ai/ha often reduced the severity of take-all
disease on seminal and crown roots of winter wheat. Higher rates,
however, sometimes increased disease severity. Diclofop-methyl
applied in early January either reduced or did not affect the
incidence of 'whiteheads' associated with take-all injury. Disease
incidence progressively increased when herbicide applications were
delayed beyond this time. The number of seminal roots per plant
often depended upon both application rate and the level of take-all
stress. Crown root and tiller production often were stimulated by
diclofop-methyl. Stimulations in tillering in diseased plots
progressively declined when applications were delayed after early
January. Grain yield was either reduced or unaffected by diclofop-
methyl, regardless of the level of take-all disease present.
Diclofop-methyl applied to winter wheat in early winter at
recommended rates did not increase injury from take-all disease. In
some situations, the herbicide may have stimulated vegetative growth
of infected plants and compensated for growth inhibition caused by

take-all. High rates and late-season applications of diclofop-methyl, however, sometimes intensified take-all injury.

INTRODUCTION

The phenoxyalkanoic herbicide, diclofop-methyl, controls grass weeds in a variety of crops. Diclofop-methyl has been used by Oregon wheat growers since 1978 to remove wild oats and annual ryegrass from winter wheat.

Wheat is highly tolerant of diclofop-methyl (1,29,32). Wheat tolerance has been attributed to the rapid, irreversible conversion of the herbicide to nontoxic, hydroxylated derivatives within the plant (9,25). Diclofop-methyl, however, can sometimes injure wheat (31). This suggests that detoxification in wheat is not always complete. Phytotoxicity, noninjurious alterations in wheat growth, or changes in microenvironment produced by diclofop-methyl applied for weed control may predispose the plant to injury from other stress factors, such as plant diseases. Alternatively, growth stimulation of the crop or toxicity toward the pathogen may reduce disease injury.

Take-all disease, caused by the fungus Gaeumannomyces graminis var. tritici, is one of the most destructive of the soil-borne diseases of small grains (3). The mild climate and prolonged, wet winters in western Oregon favor the pathogen and make take-all a major problem in winter wheat production in this area. Several herbicides increased take-all injury in winter- and spring-sown cereals in Europe (16,17,18,23,30). No interactions between diclofop-methyl and take-all disease, however, have been described in the literature. In Oregon, unconfirmed incidents of increased take-all injury in winter wheat treated with postemergence applications of

diclofop-methyl have been reported since the introduction of the herbicide.

This research was undertaken to determine whether diclofop-methyl applied at several rates and application dates could affect take-all injury and winter wheat growth in the absence of weed competition. Correlations between altered disease injury and plant growth, should they occur, could suggest possible mechanisms of interaction between the herbicide and disease. The potential for direct interaction between herbicide and pathogen in the soil would be evaluated by determining the sensitivity of the fungus to diclofop-methyl in vitro.

MATERIALS AND METHODS

Laboratory Research.

An experiment was designed to determine whether diclofop-methyl incorporated into an artificial growth medium was toxic to virulent isolates of Gaeumannomyces graminis var. tritici obtained from three locations in the northwestern United States. Isolate GGT-MT-1 from Montana (Mary Kleis, Plant Path. Dept., Montana State University, Bozeman, MT), an unspecified isolate from Washington (Dr. R. J. Cook, Regional Cereal Disease Lab., ARS, Pullman, WA), and 'Jakes Hill' isolate from Oregon (Botany/Plant Path. Dept., Oregon State University, Corvallis, OR), were maintained on colonized, dehydrated oat seed until needed. Colonized oats were ground in a Wiley mill and used to infect wheat seedlings, from which the cultures were reisolated. Active cultures were maintained on potato dextrose agar (PDA) at 20 C without illumination until treatments were made.

Treatments consisted of a factorial combination of three take-all isolates grown on PDA amended with six concentrations of diclofop-methyl. The 18 treatments were replicated four times, in a randomized, complete block design.

A stock solution of 1.06 g technical-grade diclofop-methyl (American Hoechst Corp., Agricultural Div., Somerville, NJ) in 10 ml absolute ethanol was diluted in a logarithmic series with additional ethanol. One ml from each dilution was filtered into 300 ml autoclaved PDA, and the mixture stirred for several minutes. One ml of ethanol alone was filtered into PDA to serve as a check. Final concentrations of diclofop-methyl in agar were 0 (check), 10^{-1}, 10^{0}, 10^{1}, 10^{2}, and 10^{3} μM. Twenty-milliliter aliquots of treated agar

were pipetted into plastic Petri plates and allowed to cool. Disks 4 mm in diameter were removed from the margin of active colonies of the isolates and placed in the center of treated plates. Disks for each isolate within a single replication were removed from the margin of the same colony. After inoculation, plates were placed in an incubator maintained at 25 C, without illumination.

Radial growth was measured every 24 h by marking the point of maximum hyphal growth along four perpendicular radii originating at the center of the inoculum disk. Colony extension after 48 h was used as the baseline against which further growth increments were measured. Measurements were continued for 3 days after the baseline was established. Data values for each plate were based upon the mean of the four subsample measurements of radial growth, in millimeters of growth per day, taken each day. Growth increments in plates treated with diclofop-methyl were analyzed as a percentage of growth in the ethanol check for each day.

Field Research.

Features common to all field experiments. The effects of diclofop-methyl on take-all disease were evaluated in both natural and artificial infestations. Artificial infestations were generated by incorporating coarsely ground oat seed, either sterile or colonized with the 'Jakes Hill' isolate of the take-all fungus, into the soil of each plot prior to planting. All plots received the same total quantity of ground oats, with only the proportion of colonized oats changing at different levels of inoculation. Inoculum was spread evenly over each plot by hand, then incorporated to a depth of 8 to 12 cm with either a hand rake or a Rototerra tractor-powered tiller.

Experiments at both Amity and Corvallis were conducted on a Woodburn silt loam (fine-silty, mixed, mesic, Aquultic Argixeroll). 'Stephens' winter wheat was planted in mid-October in all experiments at seeding rates of 100 to 120 kg/ha on 18-cm rows. All herbicide treatments were applied with a unicycle sprayer, equipped with compressed air, and a 2.4-m boom. Herbicides were applied in 234 L/ha water carrier at 124 kPa nozzle pressure.

Take-all severity, measured as the percentage of root tissues with symptoms of the disease, could be determined reliably only by direct examination of the roots of treated plants. Two root cores per plot, each core a 12-cm square centered on the row, were dug to a depth of 10 cm. After soaking overnight in water, cores were washed of soil and debris, then stored in a freezer until evaluation. Two to four plants per core were selected for disease evaluation. Take-all severity was measured by assigning a percent attack (PA) value to the seminal and crown root systems of each plant. The PA represented a visual estimate of the percentage of total root length with black lesions characteristic of take-all infection. Total numbers of seminal and crown roots per plant also were determined at this time.

Take-all incidence, measured as the percentage of plants with symptoms of disease, was visually estimated in late summer as the percentage of the grain heads in each plot with a bleached 'whitehead' appearance often associated with take-all injury. The rating scale of Zadoks was used for all estimates of disease incidence and severity (33). Plots were harvested in late summer with a small-plot combine.

Data were analyzed in an analysis of variance, with appropriate
partitioning of treatment effects and error terms for each
experimental design. When treatment differences were indicated as
statistically significant at a 10% or lower level of probability in
the analysis of variance, main effects and interaction means were
separated using Fisher's protected LSD (F-LSD). Responses to rates
of a quantitative variable, such as herbicide rate, were
characterized by regression analysis using orthogonal polynomial
coefficients.

Effects of diclofop-methyl on take-all disease in a natural
infestation. An experiment, established near Amity, Oregon, in
February, 1982, included a factorial combination of diclofop-methyl,
at 0 and 1.12 kg ai/ha, and benomyl (methyl-1-(butylcarbamoyl)-2-
benzimidazole carbamate), at 0 and 1.12 kg ai/ha. The four
treatments were replicated 12 times in a randomized, complete block
design. The treatment containing both pesticides was applied as a
tank-mix. This early experiment was designed to detect any changes
in take-all injury resulting from diclofop-methyl application. The
potentially interactive effects of other root- and foot-rotting
pathogens, especially strawbreaker footrot (Pseudocercosporella
herpotrichoides (Fron.)Dei.), with take-all and crop growth were not
known. Benomyl, therefore, was included as a treatment factor to
control footrot and allow more accurate assessment of take-all
injury.

The Amity location was selected after a pretreatment assay of
the test area confirmed the presence of take-all. Wheat had been
grown at this site for the previous two years. Analysis indicated a

1:2 soil:water pH of 5.9 and 33 mg soil organic matter kg^{-1} soil in

the surface 20 cm. Levels of all major nutrients were adequate for

wheat production. Fall applications of 224 kg/ha 10-20-20 complete

fertilizer and spring top-dressing of nitrogen (rate unknown) were

made. Plots of 2.4 m by 7.6 m were laid out perpendicular to the

rows, with blocks arranged in a three by four rectangle.

Pesticide treatments were applied on February 23, 1982, when

the wheat had two tillers present. Blanket applications of diuron,

at 1.8 kg ai/ha on Mar 11, and bromoxynil, at 0.6 kg ai/ha on Mar 23,

were applied to the entire experimental area.

Root cores were dug on April 8 (four tillers) and June 17

(anthesis). The incidence of whiteheads was not measured in this

experiment. Plots were harvested on August 2.

Effects of diclofop-methyl rate on the incidence and severity of

take-all disease in an artificial infestation. An experiment

established at Hyslop Research Farm, near Corvallis, Oregon, in

October, 1982, included a factorial combination of diclofop-methyl,

at 0, 1.12, and 2.24 kg ai/ha, with soil-incorporated oat inoculum,

at 0, 10, and 100 kg/ha. The nine treatments were replicated six

times in a randomized, complete block design.

The experimental site was fallow the previous year. The soil

had a 1:2 soil:water pH of 5.9 and 31 mg soil organic matter kg^{-1}

soil in the surface 20 cm. Levels of major nutrients were adequate

for wheat production. Additional fertilizer requirements were

supplied by pre-plant incorporation of 336 kg/ha 16-20-0, and a

spring top-dressing of 123 kg N/ha as urea applied on March 10, 1983.

Inoculum was hand-raked into the 3.0 m by 7.6 m plots. Stephens

winter wheat was planted on October 19, 1982. Diuron at 1.8 kg ai/ha
was applied on November 3 for supplemental weed control. Diclofop-
methyl treatments were made on December 8 when one to two tillers
were present.

Root cores were dug on March 25 (three to four tillers) and
June 30 (early to medium milk), 1983. In addition to take-all and
root number assessments, the number of tillers per plant was
determined at both root sampling dates. The incidence of whiteheads
in each plot was visually evaluated on June 27. Two independent
assessments were made from the front and back of each plot. Plots
were harvested on August 1.

Effects of diclofop-methyl application date on take-all incidence in
an artificial infestation. A field experiment, established at Hyslop
Farm in October, 1983, included six diclofop-methyl application
dates, plus an untreated check, in factorial combination with two
rates of inoculation, 0 and 90 kg/ha ground, colonized oats.
Application dates for the herbicide were approximately 3 weeks apart.
The same herbicide rate, 1.12 kg ai/ha, was applied at all treatment
dates. Application dates and inoculum rates were arranged in a
split-plot design, with inoculum levels as main plots. The 14
treatments were replicated four times in a randomized, complete block
arrangement.

The experimental area had been planted with a range of
perennial grasses in the previous year. The soil had a 1:2
soil:water pH of 7.0 and 23 mg soil organic matter kg^{-1} soil in the
surface 15 cm. Levels of major nutrients were adequate for wheat
production. A complete fertilizer application of 240 kg/ha 16-20-0

was incorporated into the soil prior to planting. In addition, a spring top-dressing of 120 kg N/ha as urea was applied on March 5, 1984. Stephens winter wheat was planted on October 19, 1983, in 3 m by 6 m plots. The entire area was oversprayed with diuron at 1.8 kg ai/ha on October 27, and with a mixture of chlorsulfuron at 16.8 g ai/ha and bromoxynil at 0.6 kg ai/ha on December 1.

Diclofop-methyl treatments were applied on January 10, 1984 [one to two tillers/83 days after planting (DAP)], February 2 (two to three tillers/106 DAP), February 27 (two to three tillers/131 DAP), March 20 (five to six tillers/152 DAP), April 4 (one node/167 DAP), and April 27 (three node/190 DAP).

The severity of take-all disease on roots was not evaluated in this experiment. Plant growth, however, integrating the reactions of the crop plant to both herbicide and disease, was monitored. Fresh weights were measured on June 12 (early anthesis) and July 10 (late milk). Two subsamples of 0.3 m of row each were taken from each plot. Plants were cut 5 cm above the soil surface. Fresh weight and tiller number per subsample were determined in the field. The incidence of whiteheads was evaluated at the time of the final fresh weight sampling. Plots were harvested on August 1.

Greenhouse Research.

Three greenhouse experiments, established in 1983 at the Crop Science Department, Oregon State University, examined the effects of diclofop-methyl application rates on the severity of take-all disease under controlled environmental conditions. Treatments included a factorial combination of three diclofop-methyl rates, 0, 1.12, or 2.24 kg ai/ha, with several rates of ground oat inoculum for

generating take-all disease. Treatments were replicated either five or six times in a randomized, complete block design. The major differences between the experiments were soil types and inoculation rates. Soils collected from two different locations in the Willamette Valley of western Oregon were used in these experiments. Soil from the top 20 cm of a Willamette silt loam was collected at the North Willamette Experiment Station near Canby, Oregon. Soil analysis indicated a 1:2 soil:water pH of 5.1, 36 mg soil organic matter kg^{-1} soil and adequate levels of major nutrients. Finely ground, colonized and sterile oats were mixed with soil to give final inoculum concentrations of 0 and 0.5% w/v (1% w/v = 1 g inoculum in 100 cm^3 soil). Ground sterile oats were added to the inoculum to insure that all pots received the same quantity of organic matter. Checks received sterile oats alone. Two additional experiments used soil from the surface layers of an unidentified sandy clay loam collected near Corvallis, Oregon. This soil was either unamended or mixed in equal proportions with peat and sand. Inoculum was added to the unamended soil in concentrations of 0, 0.15%, and 0.30% w/v, and to the amended soil in concentrations of 0, 0.5%, and 1.0% w/v. The fertility status of the soil from Corvallis was not analyzed.

Soils were screened through 0.6-cm mesh hardware cloth to remove large debris prior to mixing with inoculum. Treated soil was mixed thoroughly, then dispensed in units of 350 ml into plastic pots 6.7 cm in diameter and 25 cm deep to which vermiculite had been added to improve drainage. Four seeds of Stephens winter wheat were planted equidistantly in each pot, and approximately 2 cm deep. Cones were placed in drainage pans and maintained on the greenhouse

bench under high-intensity fluorescent lamps with a photoperiod of 12 h. Greenhouse temperatures were maintained near 20 C ($\pm$ 2 C). Plants were watered as needed and fertilized every 2 weeks with full-strength Long-Ashton solution (10).

Plants were treated with diclofop-methyl approximately 3 weeks after planting. The herbicide was applied in 300 L/ha water carrier with a compressed air sprayer at a pressure of 190 kPa, using a single nozzle 36 cm above the soil surface. All plants were sprayed with the same volume of water, with the check treatments receiving distilled water alone.

Plants were harvested when 7 to 9 weeks old. Soil and debris were washed from the roots and entire plants were stored in plastic bags in the refrigerator until evaluation. Take-all severity on root systems of individual plants was visually estimated following the same procedure described for the field experiments. Wheat response to take-all disease and diclofop treatment was monitored by counting the number of seminal and crown roots produced by each plant.

Data were analyzed in a similar manner to that described for the field experiments. Poor seedling emergence in replications of some treatments, especially those with the high rate of inoculum, resulted in an unbalanced data set. A computer program capable of handling unbalanced data was used when necessary for analysis.

RESULTS AND DISCUSSION

Interactions between herbicides and plant diseases can sometimes be explained by direct fungicidal or stimulatory effects of the chemical on the pathogen prior to infection. Isolates of Gaeumannomyces graminis var. tritici from Montana and Washington responded to diclofop-methyl in a similar manner, but both were more sensitive than the Oregon isolate (Figure 2.1). Radial growth of the Montana and Washington isolates in vitro was not affected at doses below approximately 10 μM, or 3.41 ppm. Growth of the Oregon isolate was stimulated at concentrations below approximately 100 μM. Concentrations higher than these estimated threshold values reduced growth, but did not prohibit it altogether.

These results suggest that fungal growth would not be inhibited by the diclofop-methyl at concentrations in the soil solution of less than 5 ppm. At very low concentrations, growth of pathogen strains similar to the Oregon isolate may be stimulated.

Extrapolation of data from in vitro toxicity tests such as this to the field must be undertaken with caution. The complexity of the soil environment makes estimation of herbicide concentrations in the soil solution very difficult. Leaching studies on several Oregon soils demonstrated that most soil-applied diclofop-methyl was retained in the top 5 cm of the profile (11). Only 31% of the active herbicide had leached out of the top centimeter after 87 days. Limited movement in the soil suggests that inhibition of pathogen growth by diclofop-methyl would occur only in the top several centimeters of soil. The majority of the crop roots and

epidemiologically important concentrations of take-all inoculum, however, are found at lower depths.

Direct effects of diclofop-methyl on the take-all fungus may be mediated by factors other than in vitro sensitivity to the herbicide. The reaction of the saprophytically incompetent take-all pathogen to the herbicide may be greater in the soil where the organism is constantly weakened by competition and predation from other microorganisms. Dormant mycelia sequestered within colonized debris, however, may be less sensitive to the herbicide than the active fungus. Alteration of growth in other soil microflora that serve as biological control agents against the take-all fungus could affect the disease as well.

In field and greenhouse studies, applications of diclofop-methyl at a rate of 1.12 kg ai/ha to naturally infested and artificially inoculated soils reduced the severity of take-all on the seminal root system in eight of 11 assessments in five different experiments. Differences between treatments, however, were rarely statistically significant.

Diclofop-methyl applied to the unamended sandy clay loam soil reduced severity of disease on seminal roots, but only at the 1.12 kg ai/ha rate (Figure 2.3). Responses to increasing herbicide rates were similar at all inoculation rates including the uninoculated check. In order to duplicate the field situation as closely as possible, soils used in greenhouse experiments were never sterilized prior to use. The take-all fungus is a common contaminant in soils collected from Western Oregon, especially soils where wild or cultivated grasses have grown (28). Data suggest that a small

resident population of the pathogen in the uninoculated soil reacted to diclofop-methyl in a manner similar to the 'Jakes Hill' isolate.

The effect of diclofop-methyl on seminal root infection in the Willamette silt loam soil changed when pots were artificially inoculated with the take-all fungus. In inoculated pots, take-all became less severe as diclofop-methyl rates increased (Figure 2.2). The reaction in uninoculated pots was reversed. The different responses in take-all symptoms in inoculated and uninoculated soils may be due to the presence of take-all strains with different reactions to diclofop-methyl. The Canby site was selected after confirming that take-all had been reported in earlier wheat crops at that location. The response in the uninoculated soils may reflect the reaction of a small resident population of the pathogen. This strain represented only a small proportion of the population present in inoculated pots, where the 'Jakes Hill' strain predominated.

The severity of take-all on the crown roots also was reduced by the low rate of diclofop-methyl in eight of 11 assessments. Reductions in take-all severity were generally less than 10 percentage points. Treatment differences were statistically significant, however, in only two cases.

Interactions between diclofop-methyl and inoculation rates in their effects on crown root symptoms were noted in two soils evaluated in the greenhouse. The response of crown root infection to increasing diclofop-methyl rates in the Willamette silt loam soil was similar to that noted on seminal roots (Figure 2.2). Take-all injury was reduced by diclofop-methyl applied to inoculated pots, but increased in uninoculated pots.

The severity of crown root infection in response to diclofop-methyl application changed at each level of inoculation in the unamended sandy clay loam soil (Figure 2.3). Disease was negligible in the uninoculated soil. Diclofop-methyl applied at 1.12 kg ai/ha either slightly increased or decreased the severity of disease on crown roots, depending upon the level of inoculum in the soil. When applied at the 2.24 kg ai/ha rate, more disease was present in treated than in untreated pots, regardless of the concentration of inoculum.

In general, the responses of infected seminal and crown root systems to diclofop-methyl rate were highly correlated within an experiment. The mechanism of interaction between the herbicide and disease apparently affected both root systems in a similar manner.

Rates of diclofop-methyl greater than 1.2 kg ai/ha sometimes increased disease severity (Figure 2.3). Symptoms of herbicide injury, such as large chlorotic areas on the foliage, generally were seen in both healthy and diseased plants treated with diclofop-methyl at high rates. In one field study, reduction in the growth of secondary roots was correlated with diclofop-methyl rate (Appendix Table 8). Reductions in secondary root growth were observed in both uninoculated and inoculated plots. In another experiment, the occurrence of stunted, discolored crown roots was correlated with increasing rates of both diclofop-methyl and take-all inoculum, suggesting an interaction between herbicide and disease in generating this unusual injury. Inhibition of root growth by diclofop-methyl has been reported in several weed and crop species (8,12).

Diclofop-methyl did not affect the incidence of whiteheads in the artificially inoculated 1982 experiment. In 1983, whitehead incidence in inoculated plots was reduced by diclofop-methyl applied in early January, but increased when applications were delayed later into the growing season (Figure 2.4). Reductions in the incidence of whiteheads by diclofop-methyl applied in early winter also have been observed in related research (see chapter 1).

Wheat shoot and root growth was monitored in an effort to explain the small, but consistent reductions in take-all disease following diclofop-methyl application. In several experiments, the number of seminal and crown roots in herbicide-treated plants differed from that observed in untreated plants. In cases where seminal root production was affected, root number was found to depend not only upon the rate of diclofop-methyl applied, but also upon the level of take-all inoculation.

In the artificially inoculated 1982 field experiment, increasing diclofop-methyl rates above 1.12 kg ai/ha reduced the number of seminal roots on plants in uninoculated plots (Figure 2.5). Increasing diclofop rates had little effect if the inoculation rate, and presumably disease stress, was low. Increasing herbicide rate at the highest level of inoculation, however, increased the number of seminal roots present.

A similar response to herbicide application was observed in tiller production in related research (see chapter 1, figure 1.2). Increasing rates of four postemergence herbicides, including diclofop-methyl, stimulated tiller production, but primarily in

inoculated plots. The herbicides applied to healthy wheat had little effect on tiller number.

Treatment effects noted above, however, were reversed in the Willamette silt loam soil tested in the greenhouse (Figure 2.6). Root number was stimulated by diclofop-methyl applied to uninoculated pots, but was sharply reduced in inoculated pots, when compared with the diseased but untreated check. The data, however, suggest that increasing the rate of herbicide from 1.12 to 2.24 kg ai/ha in inoculated pots stimulated root production. Root numbers for both diclofop-methyl rates on inoculated pots were similar to those in uninoculated pots receiving the same herbicide rate. The apparently anomalous value for the inoculated but unsprayed treatment cannot be explained.

The small differences between treatments in their effects on seminal root number should not be discounted. The seminal roots, varying in number from three to six, are the first roots produced by the wheat plant (14,19). In root amputation studies, however, the seminal root system alone accounted for 40 to 60% of final yield (22). These roots were especially important during the first half of the developmental cycle of the crop, prior to full establishment of the crown root system (22). The combination of diclofop-methyl and take-all disease in our research apparently affected either the initiation of a dormant primordium sensitive to stress or the persistence of roots after initiation. The nature and significance of this interaction is in doubt until additional data become available.

Crown root production was either stimulated or unaffected by
diclofop-methyl. The herbicide affected crown root production in a
similar manner regardless of the level of take-all present.
Stimulation of crown root production was noted in both the greenhouse
(Figure 2.7) and the field (Figure 2.8).

The close interrelationship between root and shoot growth
suggested that altered rooting characteristics should affect, or be
affected by, shoot growth. Diclofop-methyl, in fact, stimulated
tiller production in the field (Figure 2.9). Analysis of the data
indicated that the herbicide increased tiller number regardless of
the level of take-all present. The response of tiller production to
progressively later application dates changed substantially,
depending upon whether plots were inoculated or not (Figure 2.10).
When take-all was present, early diclofop-methyl applications
stimulated tillering. Plots treated later in the season generally
produced tiller numbers similar to the untreated, but diseased check.
This tillering response in inoculated plots may have been correlated
with the response described earlier for the incidence of whiteheads.
Delaying diclofop-methyl application beyond mid-January reduced
tillering, but increased take-all symptoms. This observation
suggests that the herbicide may influence take-all through its effect
on tiller growth or related processes within the plant, such as crown
root growth. Tillering in uninoculated plots was stimulated with
progressively later application dates up to a maximum for the late-
March treatment. Tillering in plots treated after this date was
similar to that in the untreated, uninoculated plots.

Fresh weight accumulation by the crop was evaluated only in relation to time of diclofop-methyl application. Fresh weights per unit of row generally responded to application timing in a manner similar to tiller production. Changes in fresh weight, in fact, probably reflected alterations in tiller density. Weights of individual tillers, calculated from tiller density and fresh weight data, were similar to the unsprayed checks in uninoculated plots at all application dates except the final one (Figure 2.11). In inoculated plots, however, tiller size was increased by diclofop-methyl at all application dates, but decreased slowly with progressively later application timing.

Grain yield responsed to increasing rates of diclofop-methyl in a similar manner at all levels of inoculation. The herbicide either had no effect or reduced yield (Figure 2.12). Yield reductions were small and directly proportional to herbicide rate. In cases where yield was reduced by diclofop-methyl application, the herbicide may have stimulated vegetative growth at the expense of reproductive growth since tiller density is only one component of final yield.

Inoculation of test plots with the take-all fungus consistently reduced grain yield (data not shown). In general, measurements of plant growth were indirectly proportional to inoculation rates. The incidence and severity of disease, however, were directly proportional to the amount of inoculum added to each plot.

Should the interaction between diclofop-methyl rate and timing, and the level of take-all infection prove to be a general phenomenon, this would indicate that the pathogen predisposes the infected plant to react to the herbicide in a manner different from that of a

healthy plant. Results from related research support this hypothesis (chapter 1).

The entire seminal root system in plants heavily infected with take-all is smaller than that of a healthy plant (2). Reduced water and nutrient uptake through diseased seminal roots attached to the main tiller presumably suppress the initiation of tiller buds in the axils of main tiller leaves. In any case, severe injury to the seminal roots from take-all disease, often associated with infection early in the season, reduces tiller production (26). Delay or abortion of tillers in wheat appears to be a general response to root stress (13,20). Many of the crown roots found on a mature, well-tillered wheat plant are those associated with specific tillers (14). Reduced tillering from take-all infection, therefore, could reduce crown root number as well.

Diclofop-methyl and other postemergence cereal herbicides, such as difenzoquat, sometimes stimulate tillering in wheat (31). It is possible that herbicide applications early in the vegetative phase of wheat development stimulate the initiation of tiller buds suppressed by early-season take-all infection. Tillers produce their own crown roots shortly after initiation, thus becoming somewhat independent from the main tiller (14). The effect of diclofop-methyl on plants infected with take-all, then, may simply be the restoration of the plant's normal pattern of development. Herbicide applications to healthy plants should have less effect because fewer tiller buds would be suppressed by root stress from take-all infection.

Time of infection in relation to the developmental cycle of the crop may be critical in determining the response of the plant to

take-all. Infection of crown roots late in the season after tillering is underway retards the growth of individual tillers (26). Growth inhibition in infected tillers at this time may result in transfer of assimilates from shoots to the roots. This phenomenon could account for the increased crown root production frequently observed in mild or late-season infestations of take-all (5,7,21). Skou suggested that increased crown root production in response to take-all infection may be a transient phenomenon, and that diseased plants eventually are weakened to the extent that they lose their capacity to generate new roots (27).

The direction, whether inhibition or stimulation, and magnitude of a growth response to herbicide application often will depend upon the concentration of the chemical at the primary site of activity within the plant. Diclofop-methyl is rapidly hydrolyzed to the parent acid soon after application in both sensitive and tolerant species (24). Both diclofop-methyl and diclofop acid are toxic to sensitive species. Shoots are generally more sensitive to the ester and roots to the acid (24). Evidence suggests that the specific mechanisms of activity for the two compounds may differ (24). Root responses may be of little consequence in postemergence applications to unstressed plants, however, since little of the herbicide applied to the foliage is translocated to other parts of the plant (4,12). Translocation patterns between shoots and roots, however, may be altered by vascular blockage of roots infected by the take-all pathogen. Altered sinks and rates of symplastic transport in plants with take-all could result in different concentrations of active herbicide reaching sites of meristematic activity near the crown.

Interference with IAA-stimulated cell elongation is a major mechanism of growth inhibition by the diclofop-methyl, although other physiological functions also may be impaired (24). The effects of diclofop-methyl on growth of wheat with take-all disease may be related to this anti-auxin activity. Growth regulators that reduce the activity of endogenous auxins stimulate root growth in some plant species (15). Pathogens also have been implicated in disruptions of hormonal balances within a host plant (6). Biosynthetic processes in the root tips of plants could be affected by take-all infection through reduction in the quantity of assimilates passing through regions of the root blocked by the pathogen. Vascular disruption also could reduce transport of hormones, particularly auxins, from roots to shoots. The presence of both herbicide and disease in a single plant may affect hormonal regulation of plant growth in a different manner from either factor alone.

Diclofop-methyl, applied to winter wheat in early winter at rates of 1.12 kg ai/ha or less, did not increase injury from take-all disease. Disease sometimes was more severe when the herbicide was applied at high rates or late in the growing season. The incidence and severity of take-all infection often were reduced by herbicide application, but reductions were small. Diclofop-methyl probably had little direct effect on survival of the pathogen in the soil. The herbicide may have reduced susceptibililty of root tissues to infection, but no unequivocal evidence was found to support this conclusion. It seems more likely that diclofop-methyl stimulated vegetative growth, allowing the infected plant to more easily compensate for take-all injury. The observed reductions in take-all

symptoms are compatible with this conclusion. More roots present on treated plants could result in an apparent reduction in the relative percentage of infected tissue, without necessarily reducing the absolute amount of infection.
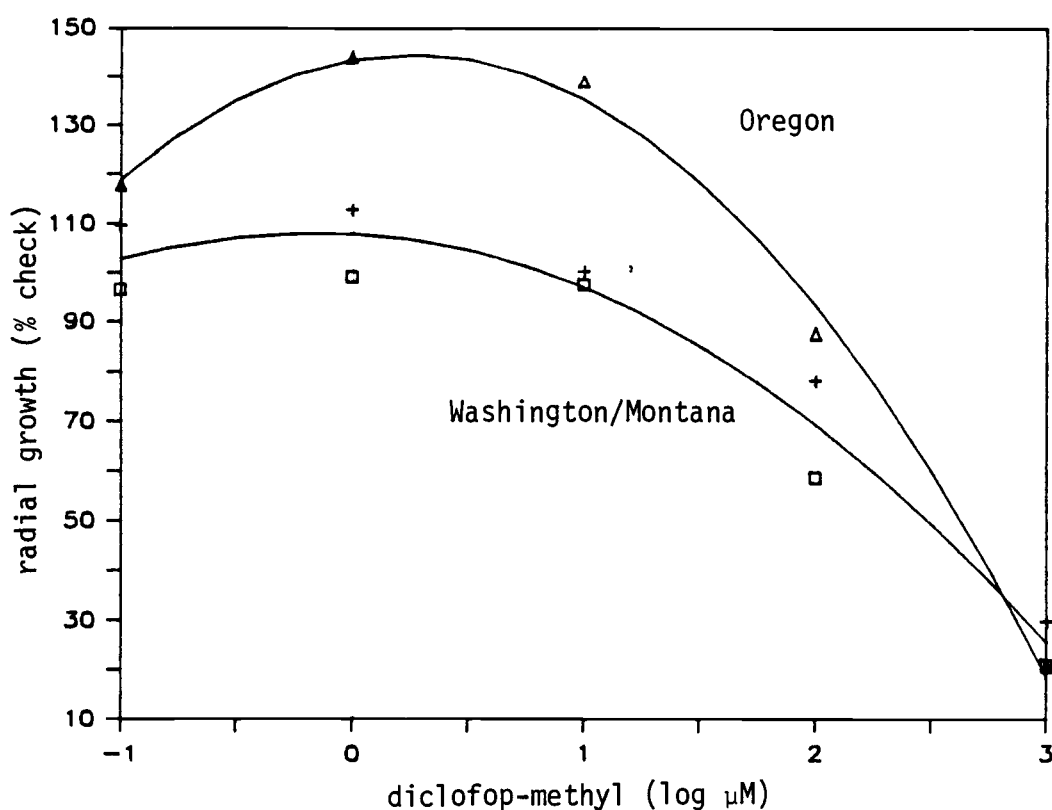
Figure 2.1. Effect of diclofop-methyl on in vitro growth of
Gaeumannomyces graminis var. tritici isolates from Oregon (Δ),
Washington (+), and Montana (□). The quadratic response to
herbicide concentration was similar for the Washington and
Montana isolates. The mean quadratic response of these isolates
was different from that of the Oregon isolate ($.01<p<.05$).
Equation describing the mean response of the Washington and Oregon
isolates:

radial growth (% check)=108.0-2.9(log conc.)-8.2(log conc.)^2 r^2=1.00
Equation describing response of Oregon isolate:
radial growth (% check)=143.6+8.1(log conc.)-16.7(log conc.)^2
$$r^2=0.99$$
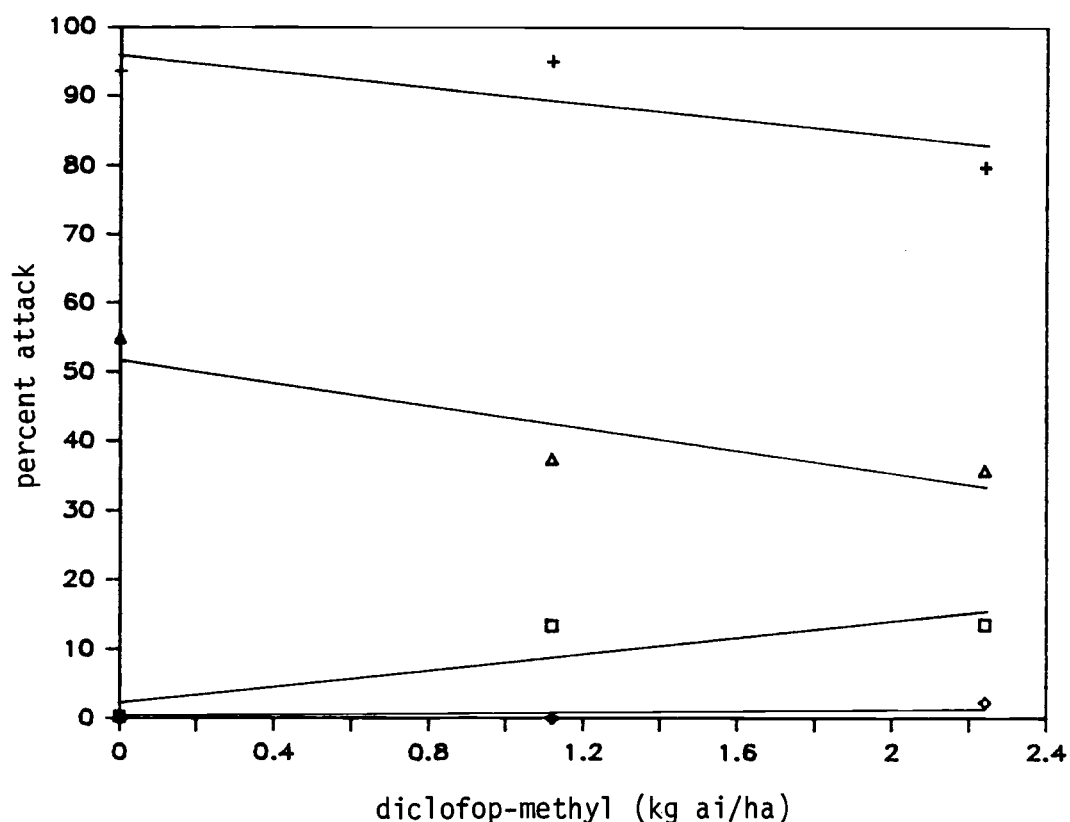
Figure 2.2. Effect of diclofop-methyl on the severity of take-all disease on the seminal and crown roots of winter wheat grown in the Canby soil with no inoculum and 0.5% w/v ground, colonized oats. The linear response of disease severity to herbicide rate changed on both the seminal roots (.01<p<.05) and crown roots (p<.01) when pots were inoculated with the take-all fungus.

seminal roots/no inoculum (□):

PA=2.4+5.9(rate) r^2=0.76

seminal roots/0.5% w/v inoculum (+):

PA=96.4-6.2(rate) r^2=0.67

crown roots/no inoculum (◇):

PA=-0.4+1.0(rate) r^2=0.75

crown roots/0.5% w/v inoculum (△):

PA=52.4-8.6(rate) r^2=0.82
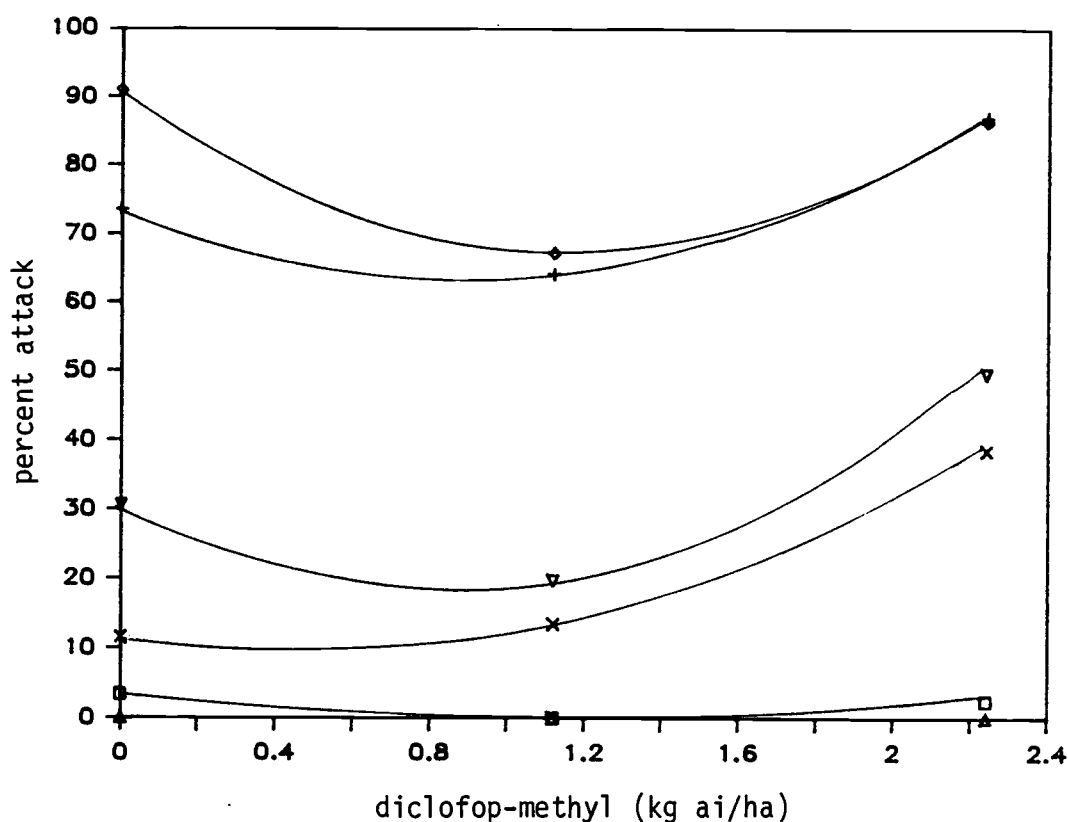
Figure 2.3. Effect of diclofop-methyl on the severity of take-all disease on the seminal and crown roots of winter wheat grown in the unamended Corvallis soil with no inoculum, 0.15%, and 0.30% w/v ground, colonized oats. The quadratic response of disease severity to herbicide rate changed for both the seminal roots (.05<p<.10) and crown roots (.05<p<.10) when pots were inoculated with the take-all fungus.

seminal roots

  no inoculum (□): $3.4-5.6(\text{rate})+2.3(\text{rate})^2$

  0.15% w/v (+): $73.7-23.1(\text{rate})+13.0(\text{rate})^2$

  0.30% w/v (◊): $91.0-40.4(\text{rate})+17.2(\text{rate})^2$

crown roots

  no inoculum (△): $0.1-0.2(\text{rate})+0.1(\text{rate})^2$

  0.15% w/v (X): $11.6-8.6(\text{rate})+9.2(\text{rate})^2$

  0.30% w/v (▽): $30.7-28.1(\text{rate})+16.3(\text{rate})^2$
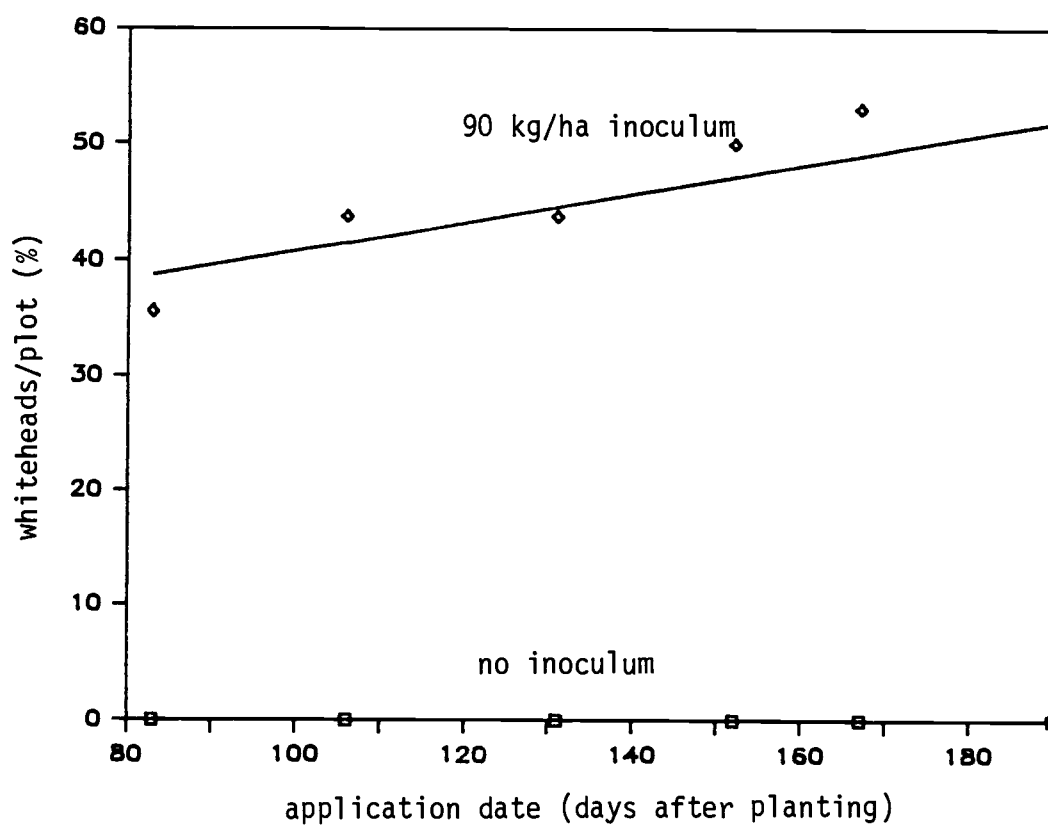
Figure 2.4. Effect of diclofop-methyl application date on the
incidence of take-all disease in winter wheat at Hyslop
Farm (1983). Plots were inoculated prior to planting with no
inoculum (□), and 90 kg/ha (◊) ground, colonized oats. The
linear response of disease incidence to application timing
changed when plots were inoculated with the take-all fungus
(p<.05).
Equation describing response in uninoculated soil:
            no whiteheads observed
Equation describing response in the inoculated soil:
  whiteheads/plot (%)=28.5+0.1(DAP) r^2=0.65
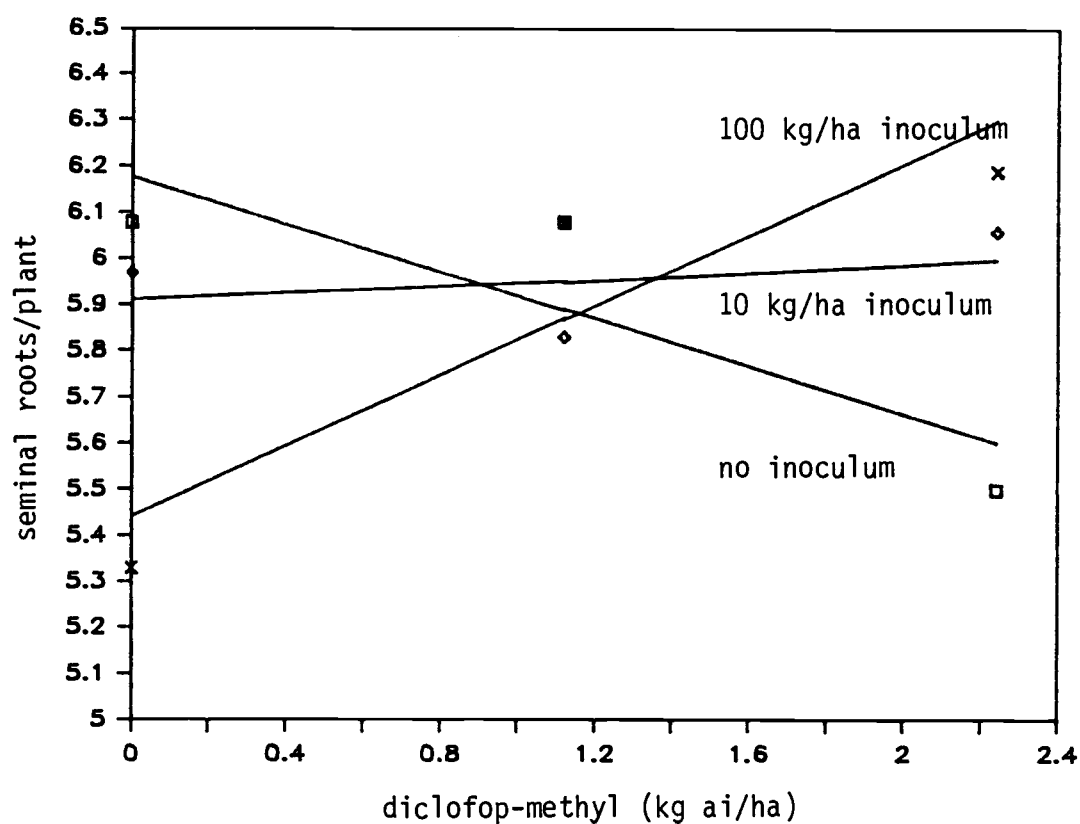
Figure 2.5. Effect of diclofop-methyl on seminal root growth
in wheat with take-all disease at Hyslop Farm (1982). Plots
were inoculated prior to planting with no inoculum (□),
10 kg/ha (◊), and 100 kg/ha (X) ground, colonized oats. Plots
were evaluated on April 5. The linear response of root number
to herbicide rate changed as inoculation rates increased (p<.01).
No inoculum: sem. roots/plant=6.2-0.3(rate) r^2=0.75
10 kg/ha inoculum: sem. roots/plant=5.9+0.1(rate) r^2=0.14
100 kg/ha inoculum: sem. roots/plant=5.4+0.4(rate) r^2=0.84
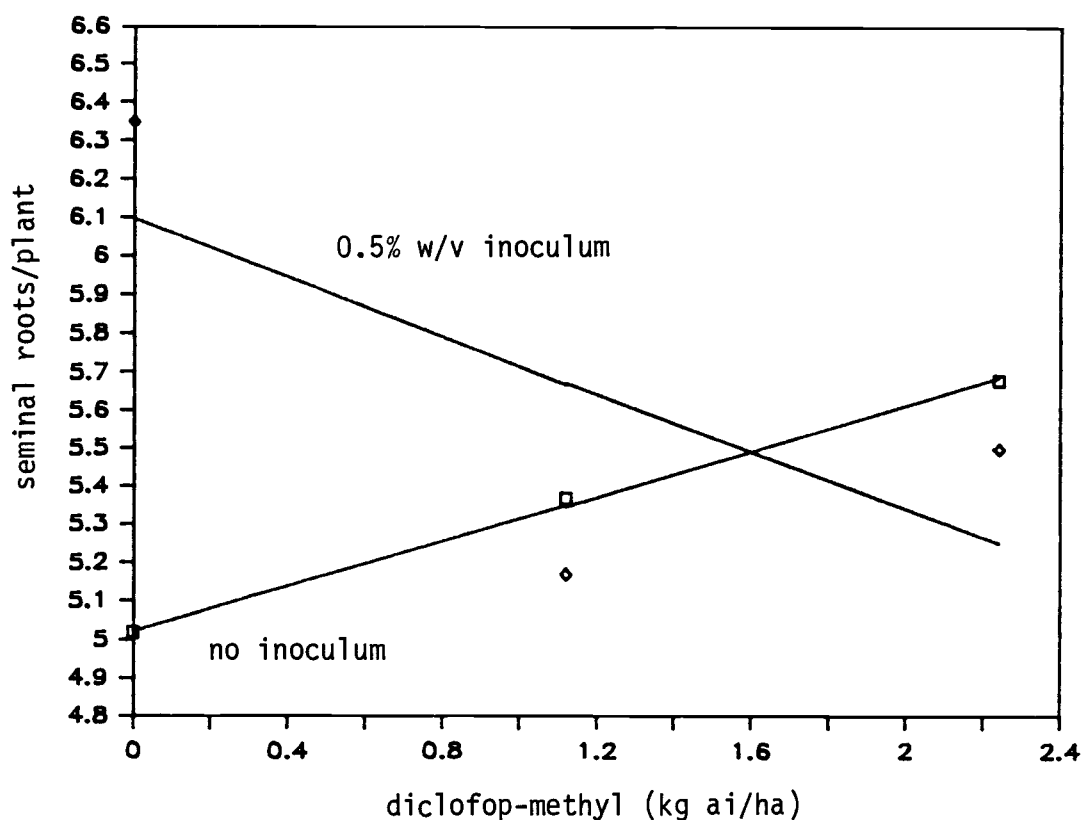
Figure 2.6. Effect of diclofop-methyl on seminal root growth of winter wheat in the Canby soil with no inoculum (□) and 0.5% w/v (◇) ground, colonized oats. The linear response of root number to herbicide rate changed when pots were inoculated with the take-all fungus (.01<p<.05).

Equation describing the response in the uninoculated soil:

$$\text{sem. roots/plant} = 5.0 + 0.3(\text{rate}) \quad r^2 = 1.00$$

Equation describing the response in the inoculated soil:

$$\text{sem. roots/plant} = 6.1 - 0.4(\text{rate}) \quad r^2 = 0.49$$
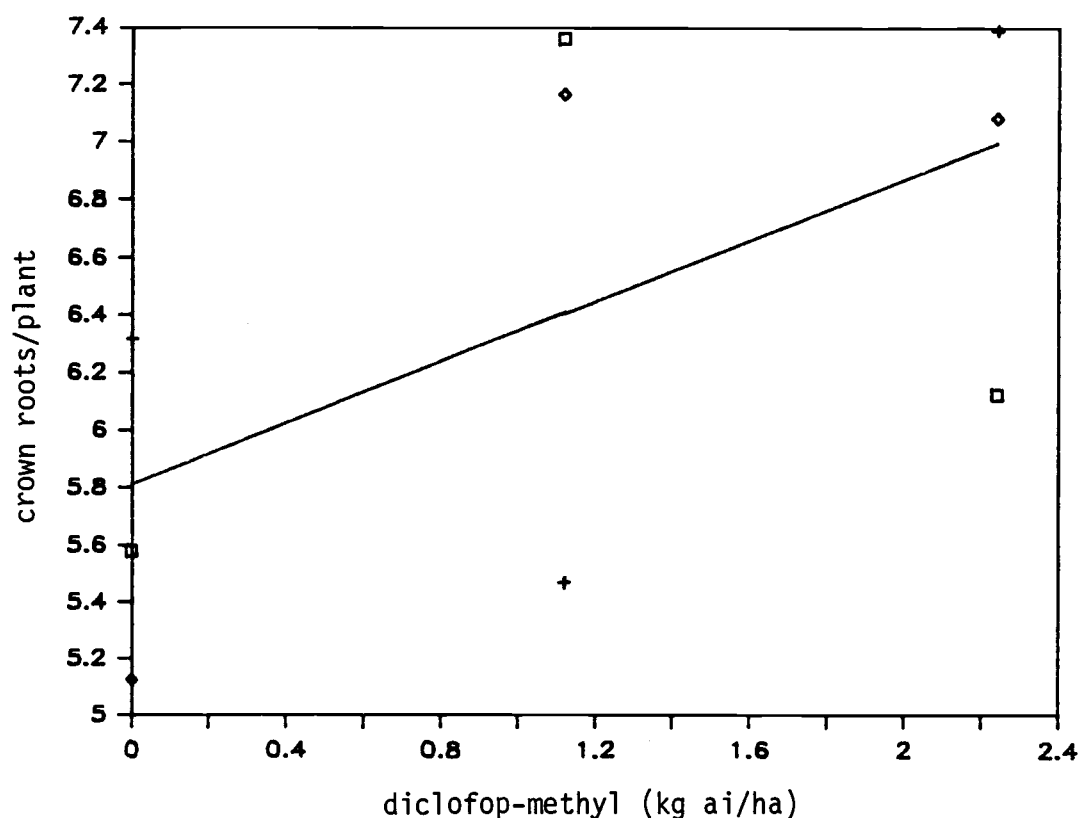
Figure 2.7. Effect of diclofop-methyl on crown root growth of winter wheat grown in the unamended Corvallis soil with no inoculum (□), 0.15% w/v (+), and 0.30% w/v (◇) ground, colonized oats. The linear response of root number to herbicide rate was similar at all inoculation rates (p<.05). Diclofop-methyl treatment values were averaged over all inoculation rates for regression analysis.

Equation describing the mean response of crown root number to diclofop-methyl rate:

$$\text{crown roots/plant} = 5.8 + 0.5(\text{rate}) \quad r^2 = 0.87$$
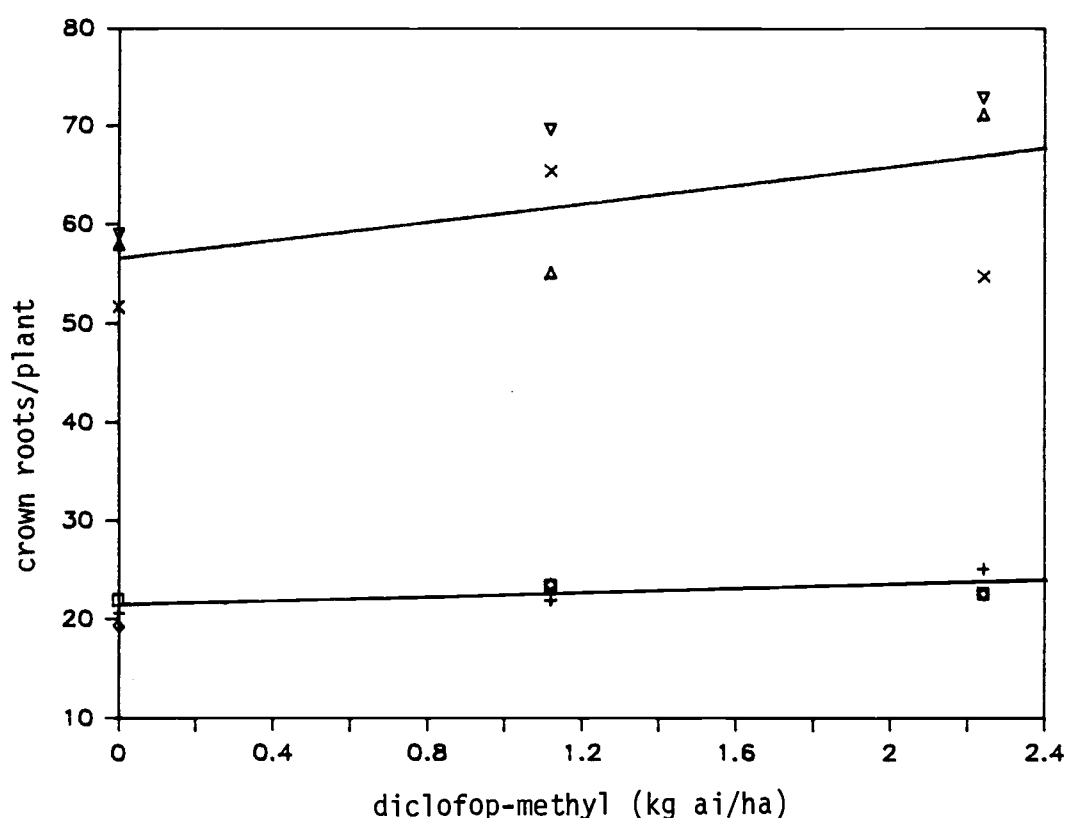
Figure 2.8. Effect of diclofop-methyl on crown root growth of winter wheat at Hyslop Farm (1982). Plots were inoculated prior to planting with no inoculum, 10 kg/ha, and 100 kg/ha ground, colonized oats. Plants were sampled on April 5 and July 6. The linear response of root number to herbicide rate was similar at all inoculation rates on both April 5 ($.05 < p < .10$) and July 6 ($.01 < p < .05$). Diclofop-methyl treatment values were averaged over all inoculation rates for regression analysis at each date.

Equation describing the mean response of crown root number to diclofop-methyl rate in plots with no inoculum (□), 10 kg/ha (+), and 100 kg/ha (◇) inoculum on April 5:

$$\text{crown roots/plant} = 21.0 + 1.3(\text{rate}) \quad r^2 = 0.87$$

Equation describing the mean response of crown root number to diclofop-methyl rate in plots with no inoculum (△), 10 kg/ha (X), and 100 kg/ha (▽) inoculum on July 6:

$$\text{crown roots/plant} = 57.0 + 4.5(\text{rate}) \quad r^2 = 0.94$$
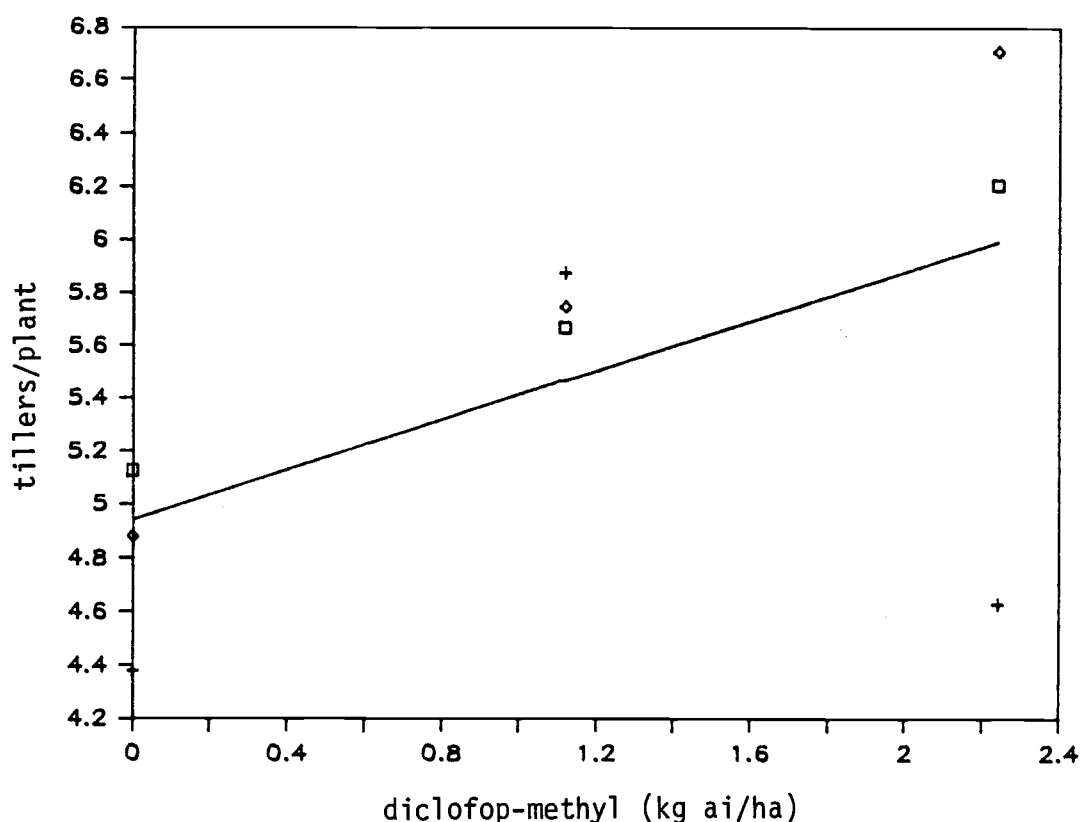
Figure 2.9. Effect of diclofop-methyl on tiller production of
winter wheat at Hyslop Farm (1982). Plots were inoculated
prior to planting with no inoculum (□), 10 kg/ha (+), and
100 kg/ha (◇) ground, colonized oats. Plants were sampled on
July 6. The linear response of tiller number to herbicide rate
was similar at all inoculation rates ($.01 < p < .05$). Diclofop-
methyl treatment values were averaged over all inoculation
rates for regression analysis.

Equation describing the response of tiller number per plant to
diclofop-methyl rate:

$$tillers/plant = 4.9 + 0.5(rate) \quad r^2 = 0.81$$
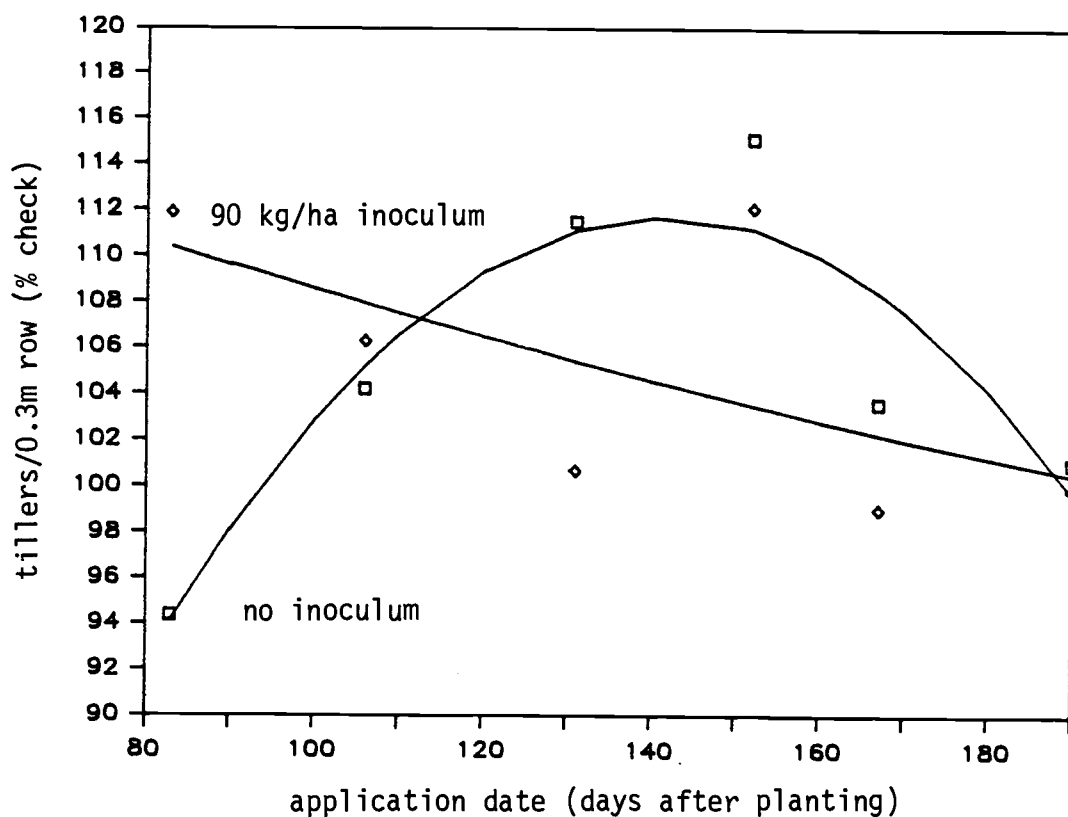
Figure 2.10. Effect of diclofop-methyl application date on tiller density of winter wheat at Hyslop Farm (1983). Plots were inoculated prior to planting with no inoculum (□) and 90 kg/ha (◇) ground, colonized oats. The quadratic response of tiller density to application timing changed when plots were inoculated with the take-all fungus (.05<p<.10).

Equation describing the response in uninoculated soil:
tillers/0.3m row(% check)=8.96+1.45(DAP)-0.01(DAP)^2 r^2=0.85

Equation describing the response in the inoculated soil:
tillers/0.3m row(% check)=121.34-0.15(DAP)+0.0002(DAP)^2 r^2=0.38
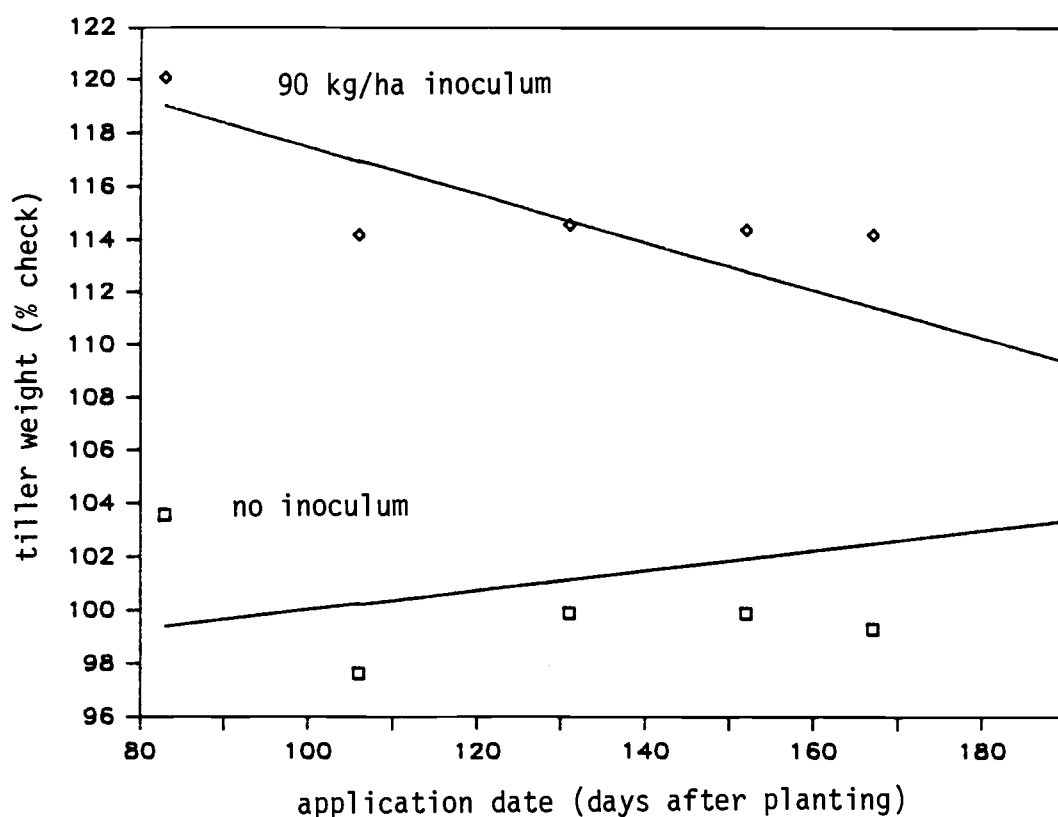
Figure 2.11. Effect of diclofop-methyl application date on tiller weight of winter wheat at Hyslop Farm (1983). Plots were inoculated prior to planting with no inoculum (□) and 90 kg/ha (◇) ground, colonized oats. The linear response of tiller weight to application timing changed when plots were inoculated with the take-all fungus ($.01 < p < .05$).

Equation describing the response in uninoculated soil:
  tiller weight(% check)=96.28+0.04(DAP) r^2=0.14

Equation describing the response in the inoculated soil:
  tiller weight(% check)=126.62-0.09(DAP) r^2=0.71
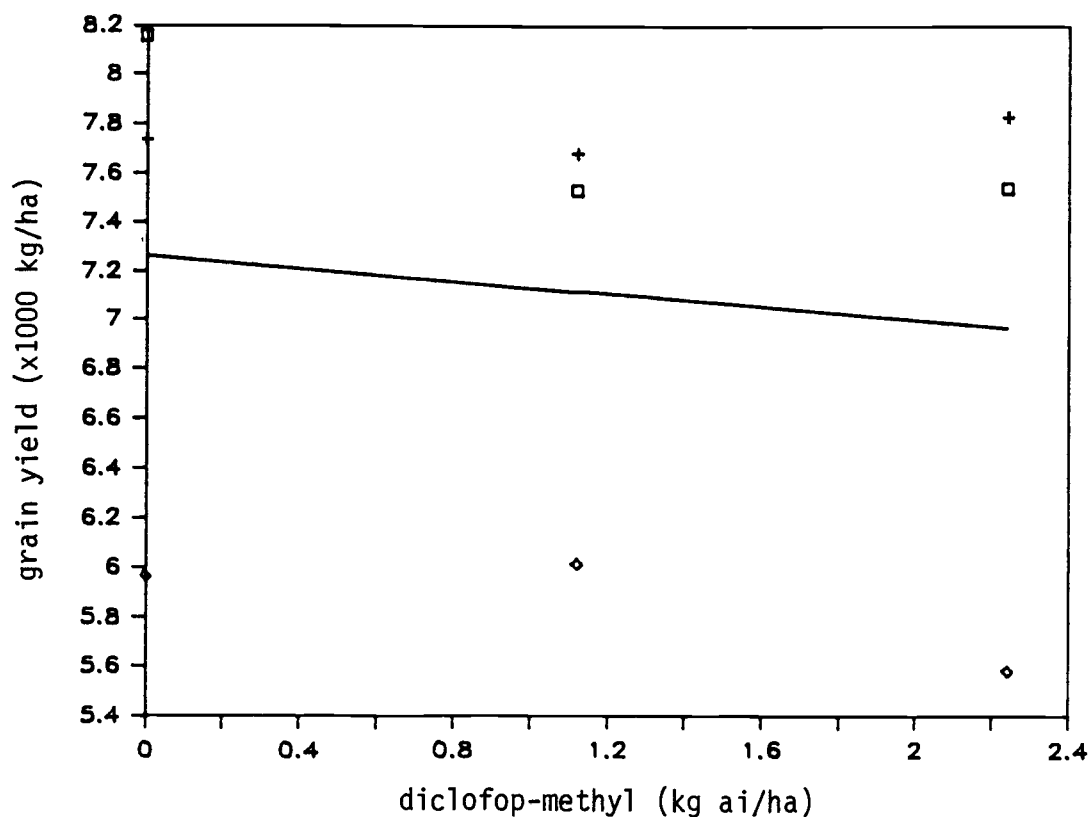
Figure 2.12. Effect of diclofop-methyl on grain yield of winter
wheat at Hyslop Farm (1982). Plots were inoculated prior to
planting with no inoculum (□), 10 kg/ha (+), and 100 kg/ha (◊)
ground, colonized oats. The linear response of yield to
herbicide rate was similar at all inoculation rates (.05<p<.10).
Diclofop-methyl treatment values were averaged over all inoculation
rates for regression analysis.
Equation describing the response of grain yield to diclofop-
methyl rate:

grain yield(kg/ha)=7269-137(rate) r^2=0.95

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APPENDIX

Appendix Table 1. Effects of diclofop-methyl and benomyl on take-all disease[1], strawbreaker footrot[2], and reproductive growth of winter wheat (Amity, 1982).

| treatment[3] | take-all severity[4] | | | | footrot incidence[4] June 17 | yield components[5] July 15 | | | | grain yield[5] August2 |
| | April 8 | | June 17 | | | | | | | |
	PA (seminal)	PA (crown)	PA (seminal)	PA (crown)		tillers/ 0.3m row	spikelets/ spike	kernels/ spikelet	1000-grain weight (g)	(kg/ha)
check	11	5	34	17	53	23.0	15.1	2.5	40.8	5850
diclofop-methyl	10	3	21	10	50	23.0	15.2	2.6	45.9	5850
benomyl	15	7	39	17	15	20.7	15.3	2.5	40.6	5690
diclofop-methyl + benomyl (tank-mix)	13	5	36	16	24	23.6	14.9	2.6	42.9	6100

[1] Take-all disease caused by the fungus, Gaeumannomyces graminis var. tritici; severity of take-all presented as a percent attack (PA) = visual estimate of the percentage of the respective root tissues with black lesions characteristic of take-all disease; root cores were dug on the dates indicated.

[2] Strawbreaker footrot caused by the fungus, Pseudocercosporella herpotrichoides; footrot incidence presented as a percent of plants with culm lesions characteristic of footrot.

[3] Both diclofop-methyl and benomyl applied on February 23 at 1.12 kg ai/ha; data are the means of 72 observations (three plants per core, two cores per replication, and 12 replications per treatment).

[4] Statistical analysis presented in Appendix Table 2.

[5] Statistical analysis presented in Appendix Table 3.

Appendix Table 2. Effects of diclofop-methyl and benomyl on take-all disease and strawbreaker footrot of winter wheat: analysis of variance (Amity, 1982).

| | | April 8 | | | | | | June 17 | | | | | | | | |
| | | PA (seminal) | | | PA (crown) | | | PA (seminal) | | | PA (crown) | | | footrot incidence | | |
source of variation	d.f.	MS	F	p	MS	F	p	MS	F	p	MS	F	p	MS	F	p
block	11	264.172	1.98	0.06	73.709	1.40	0.22	943.81	2.18	0.04	300.88	1.51	0.17	0.024	0.29	0.98
diclofop-methyl	1	11.218	0.08	0.77	29.063	0.55	0.46	683.39	1.58	0.22	203.36	1.02	0.32	0.009	0.11	0.74
benomyl	1	175.969	1.32	0.26	65.054	1.23	0.28	1206.16	2.79	0.10	122.15	0.61	0.44	1.229	14.86	0.00
diclofop-methyl x benomyl	1	5.926	0.04	0.83	0.649	0.01	0.91	289.22	0.69	0.42	95.40	0.48	0.49	0.038	0.46	0.50
experimental error	33	133.174	-	-	52.822	-	-	432.80	-	-	199.10	-	-	0.083	-	-

[1] Statistical analysis for data presented in Appendix Table 1.

Appendix Table 3. Effects of diclofop-methyl and benomyl on yield components and grain yield of winter wheat: analysis of variance (Amity, 1982).[1]

source of variation	d.f.	tillers/0.3m row			spikelets/spike			kernels/spike[2]			1000-grain weight (g)			grain yield (kg/ha)		
		MS	F	p	MS	F	p	MS	F	p	MS	F	p	MS	F	p
block	11	19.633	2.34	0.03	1.000	1.68	0.12	34.972	2.51	0.02	86.768	2.78	0.01	2.266	5.75	0.00
diclofop-methyl	1	22.825	2.72	0.11	0.350	0.59	0.45	5.267	0.38	0.54	163.910	5.25	0.03	0.414	1.05	0.31
benomyl	1	8.585	1.02	0.32	0.110	0.19	0.67	1.172	0.84	0.77	31.850	1.02	0.32	0.018	0.05	0.83
diclorop-methyl x benomyl	1	25.085	2.99	0.09	0.880	1.48	0.23	20.935	1.50	0.23	23.102	0.74	0.40	0.429	1.09	0.30
experimental error	33	8.400	-	-	0.594	-	-	13.932	-	-	31.236	-	-	0.394	-	-

[1] Statistical analysis for data presented in Appendix Table 1.

[2] No statistical analysis for kernels/spikelet; data presented in Appendix Table 1 for kernels/spikelet derived from mean values for spikelets/spike and kernels/spike.

Appendix Table 4.   Effects of postemergence herbicides and take-all disease on the incidence of culm lesions of strawbreaker footrot in winter wheat (1982).

| Treatment | Footrot lesions/plant | |
	No take-all[1]	Take-all
Check	1.10 ab[2]	0.20 n.s.
barban	0.20 d	0.25
dicamba	0.80 bc	0.40
diclofop-methyl	0.35 cd	0.15
difenzoquat	0.45 cd	0.50
dinoseb	1.50 a	0.05
mecoprop	0.50 cd	0.20
metribuzin	0.75 bcd	0.40
terbutryn	0.40 cd	0.25

[1]Plots inoculated prior to planting with 100 kg/ha of ground oat seed either sterile ('no take-all') or colonized by the take-all fungus ('take-all').

[2]Means within a column followed by the same letter are not significantly different at the 0.10 level of probability as determined by the F-LSD.

Appendix Table 5. Effects of diclofop-methyl on disease severity and crown root growth of six winter wheat cultivars grown in microplots inoculated with Gaeumannomyces graminis var. tritici, the fungus causing take-all disease (Hyslop Farm, 1982).

cultivar	take-all severity[1]				crown root growth			
	uninoculated		inoculated		uninoculated		inoculated	
	check	d-m	check	d-m	check	d-m	check	d-m
	----percent of root tissues with lesions----				--------------crown roots/plant---------------			
Daws	<1[2]	1	29	29	95	63	77	54
Hill 81	<1	<1	25	30	88	81	129	89
Hyslop	1	1	46	32	99	73	85	134
McDermid	5	<1	25	20	77	72	105	57
Nugaines	<1	<1	21	31	164	125	105	81
Stephens	3	<1	37	22	71	62	45	102

[1] Statistical analysis for data on disease severity presented in Appendix Table 7; data on root growth were not analyzed because of the highly unbalanced data set; ground sterile oats ('uninoculated') or ground oats colonized by the take-all fungus ('inoculated') were incorporated prior to planting into the circular microplots, 36 cm in diameter (0.1 m^2), at 100 kg/ha; diclofop-methyl applied on March 3 at 0 ('check') and 1.12 kg ai/ha ('d-m').

[2] Data are the means of 21 observations (three subsamples per replication, seven replications per treatment).

Appendix Table 6. Effects of diclofop-methyl on tiller development of six winter wheat cultivars grown in microplots inoculated with Gaeumannomyces graminis var. tritici, the fungus causing take-all disease (Hyslop Farm, 1982).

cultivar	shoot growth[1]				fertile tiller production				fertile tiller weight			
	uninoculated		inoculated		uninoculated		inoculated		uninoculated		inoculated	
	check	d-m	check	d-m	check	d-m	check	d-m	check	d-m	check	d-m
	--shoot fresh weight/plant (g)--				--number of headed tillers/plant--				-fresh weight/headed tiller (g)-			
Daws	96[2]	136	73	119	24	23	19	20	8.49	8.38	7.58	7.71
Hill 81	162	113	109	110	23	22	44	20	9.44	9.77	8.77	8.38
Hyslop	191	119	122	114	33	28	28	28	9.51	9.63	9.74	9.14
McOermid	152	113	113	116	33	22	28	22	7.84	14.19	7.56	6.10
Nugaines	112	93	90	111	44	36	31	28	6.26	5.64	5.34	5.33
Stephens	142	121	127	138	23	21	24	24	9.74	9.94	8.90	10.19

[1] Statistical analyses for all data presented in Appendix Table 7; ground, sterile oats ('uninoculated') or ground oats colonized by the take-all fungus ('inoculated') incorporated into circular microplots prior to planting at 100 kg/ha; diclofop-methyl applied on March 3 at 0 ('check') and 1.12 kg ai/ha ('d-m'); plants were harvested on July 15 by cutting shoots 5 cm above the soil surface.

[2] Data are the means of 21 observations (three subsamples per replication, seven replications per treatment).

Appendix Table 7. Effect of diclofop-methyl on disease severity and shoot growth of six winter wheat cultivars grown in microplots inoculated with Gaeumannomyces graminis var. tritici, the fungus causing take-all disease: analysis of variance (Hyslop Farm, 1982).

source of variation	df	Disease Severity[1]			Shoot Growth[2]			Fertile Tiller Prod.[2]			Fertile Tiller Wt.[2]		
		MS	F	p	MS	F	p	MS	F	p	MS	F	p
block[3]	6	3386.7	4.18	0.00	70820	6.86	0.00	342.8	1.15	0.33	47.9	1.04	0.41
cultivar	5	641.6	0.79	0.56	125363	12.15	0.00	1865.7	6.28	0.00	190.4	4.12	0.00
inoculation	1	95590.2	118.08	0.00	126635	12.27	0.00	251.5	0.85	0.36	174.0	3.76	0.05
cultivar x inoculation	5	755.7	0.93	0.46	22712	2.20	0.06	921.4	3.10	0.01	47.9	1.04	0.40
diclofop-methyl (dm)	1	576.4	0.71	0.40	118741	11.51	0.00	3271.1	11.02	0.00	23.7	0.51	0.47
cultivar x dm	5	662.7	0.82	0.54	20300	1.97	0.09	506.0	1.70	0.14	23.3	0.50	0.77
inoculation x dm	1	168.0	0.21	0.65	6507	0.63	0.43	20.2	0.07	0.79	46.9	1.01	0.32
cultivar x inoculation x dm	5	430.1	0.53	0.75	13353	1.29	0.27	614.0	2.07	0.07	57.5	1.24	0.29
experimental error	138	809.6	-	-	10320	-	-	296.9	-	-	46.1	-	-
Sampling Error	336	168.3	-	-	16472	-	-	349.8	-	-	43.1	-	-

[1] Statistical analysis for data presented in Appendix Table 5.

[2] Statistical analysis for data presented in Appendix Table 6.

[3] Notes on experimental design:
 1) 6 cultivars x 2 inoculation rates x 2 herbicide rates = 24 treatments (full factorial)
 2) 24 treatments arranged in a randomized, complete block, with 7 replications
 3) Experimental unit = microplot
 4) 3 plants per microplot = subsamples

Appendix Table 8. Effects of diclofop-methyl on disease incidence and severity, and vegetative growth of winter wheat grown in field plots inoculated with Gaeumannomyces graminis var. tritici, the fungus causing take-all disease (Hyslop Farm, 1982).

inoculation rate (kg/ha)	diclofop-methyl (kg ai/ha)[3]	March 25[1]						June 30[2]					WH[6]
		SPA[4]	CPA	SNO	CNO	TILNO	crown root[5] growth	SPA	CPA	SNO	CNO	TILNO	
no inoculum													
	0	0	0	6.1	22.0	5.2	91	7.7	9.6	5.7	58.1	5.1	90
	1.12	2.1	0	6.1	23.4	6.6	69	18.3	13.8	5.7	55.1	5.7	1
	2.24	0.3	0	5.5	22.6	5.4	51	6.7	2.8	5.5	71.3	6.2	0
10													
	0	2.1	1.2	6.0	20.6	5.0	89	22.3	24.8	5.5	51.7	4.4	1
	1.12	0	0.3	5.8	22.0	5.4	72	5.0	7.1	5.9	65.5	5.9	3
	2.24	0	0	6.1	25.1	5.8	52	21.2	14.5	5.4	54.8	4.6	2
100													
	0	14.1	4.5	5.3	19.3	5.1	89	66.5	67.1	5.8	59.0	4.9	46
	1.12	10.5	2.5	6.1	23.5	5.2	79	65.4	62.3	5.8	69.7	5.8	42
	2.24	14.4	2.6	6.2	22.6	5.1	55	80.0	74.4	5.7	72.9	6.7	50

[1] Statistical analysis for data evaluated on March 25 presented in Appendix Table 9.

[2] Statistical analysis for data evaulated on June 30 and 'whiteheads' presented in Appendix Table 10.

[3] Diclofop-methyl applied on December 8.

[4] Take-all severity measured as a percent attack for the seminal (SPA) and crown (CPA) root systems; the number of seminal roots per plant (SNO) and crown roots per plant (CNO), and tillers per plant (TILNO) also evaluated in plants from root cores dug on the dates indicated.

[5] Crown root growth indicates the visual estimate of the percent of the crown root system with secondary root development; alterations in crown root morphology evaluated for March 25 sample only.

[6] The incidence of take-all disease visually estimated on June 27 as the percent of grain heads per plot with a bleached, 'whitehead' appearance (WH).

Appendix Table 9. Effects of diclofop-methyl on disease severity and vegetative growth of winter wheat grown in field plots inoculated with Gaeumannomyces graminis var. tritici, the fungus causing take-all disease: analysis of variance for data collected on March 25 (Hyslop Farm, 1982)[1].

source	d.f.	SPA			CPA			SNO			CNO			TILNO			crown root growth		
		MS	F	p	MS	F	p	MS	F	p	MS	F	p	MS	F	p	MS	F	p
block	5	121.84	0.62	0.69	7.81	0.67	0.65	1.24	1.28	0.29	84.31	1.24	0.31	1.90	0.25	0.94	0.01	0.33	0.89
inoculation	2	2701.40	13.74	0.00	158.48	13.61	0.00	0.10	0.11	0.90	12.28	0.18	0.84	5.68	0.76	0.47	0.03	0.58	0.57
diclofop-methyl	2	18.92	0.10	0.91	17.40	1.49	0.24	0.57	0.59	0.56	123.02	1.81	0.18	5.36	0.72	0.49	1.87	43.16	0.00
inoculation x diclofop	4	56.02	0.28	0.89	6.10	0.52	0.72	2.83	2.93	0.03	36.48	0.54	0.71	3.90	0.52	0.72	0.01	0.33	0.85
exp. error	40	196.67	-	-	11.65	-	-	0.97	-	-	67.95	-	-	7.48	-	-	0.04	-	-
samp. error	108	225.14	-	-	15.70	-	-	0.71	-	-	34.88	-	-	4.25	-	-	0.04	-	-

[1] Statistical analysis for data presented in Appendix Table 8.

Appendix Table 10. Effects of diclofop-methyl on disease incidence and severity, and vegetative growth of winter wheat grown in field plots inoculated with <u>Gaeumannomyces graminis</u> var. <u>tritici</u>, the fungus causing take-all disease: analysis of variance for data collected on June 30 and 'whiteheads' (Hyslop Farm, 1982).

source	d.f.	SPA			CPA			SNO			CNO			TILNO			WH		
		MS	F	p	MS	F	p	MS	F	p	MS	F	p	MS	F	p	MS	F	p
block	5	122.2	0.16	0.98	372.6	0.55	0.74	0.67	0.71	0.62	1220.5	3.04	0.02	5.85	1.53	0.20	149.7	1.67	0.16
inoculation	2	39338.9	51.50	0.00	37820.5	56.28	0.00	0.28	0.30	0.74	884.8	2.20	0.12	7.11	1.86	0.17	11948.6	133.52	0.00
diclofop-methyl	2	373.7	0.49	0.62	336.7	0.50	0.61	0.70	0.76	0.48	962.8	2.40	0.10	12.40	3.23	0.05	32.9	0.37	0.70
inoc. x diclofop	4	1024.3	1.34	0.27	713.5	1.06	0.39	0.14	0.15	0.96	590.3	1.47	0.23	4.48	1.17	0.34	37.7	0.42	0.79
exp. error	40	763.9	-	-	672.1	-	-	0.93	-	-	401.8	-	-	3.83	-	-	89.5	-	-
samp. error	54	601.4	-	-	416.2	-	-	0.79	-	-	498.3	-	-	4.48	-	-	_[2]	-	-

[1] Statistical analysis for data presented in Appendix Table 8.

[2] Sampling error not determined for 'whiteheads' data.

Appendix Table 11. Effects of diclofop-methyl on disease severity and vegetative growth of winter wheat grown in different soils inoculated with the take-all fungus, Gaeumannomyces graminis var. tritici (1983).

inoc rate[4]	diclofop-methyl (kg ai/ha)	sandy loam, unamended[1]						sandy loam, amended[2]						silt loam[3]					
		SPA[5]	CPA	SNO	CNO	STWT (mg)	RTWT (mg)	SPA	CPA	SNO	CNO	STWT (mg)	RTWT (mg)	SPA	CPA	SNO	CNO	STWT (mg)	RTWT (mg)
no inoculum																			
	0	3.4	0.1	6.0	5.6	256	135	23.2	2.6	5.3	13.0	423	167	0.3	0	5.0	10.5	448	232
	1.12	0.2	0	6.5	7.4	332	166	11.1	1.6	5.4	10.6	387	184	13.4	0	5.4	12.2	454	216
	2.24	2.5	0.1	5.8	6.1	275	111	0.8	0	4.7	10.9	481	253	13.6	2.3	5.7	12.9	490	252
0.15% w/v																			
	0	73.6	11.6	5.8	6.3	105	44	-	-	-	-	-	-	-	-	-	-	-	-
	1.12	64.1	13.5	5.0	5.5	92	26	-	-	-	-	-	-	-	-	-	-	-	-
	2.24	87.0	38.6	4.9	7.4	116	32	-	-	-	-	-	-	-	-	-	-	-	-
0.30% w/v																			
	0	91.0	30.7	5.3	5.1	59	25	-	-	-	-	-	-	-	-	-	-	-	-
	1.12	67.2	19.7	6.4	7.2	113	44	-	-	-	-	-	-	-	-	-	-	-	-
	2.24	86.5	49.7	5.5	7.1	94	29	-	-	-	-	-	-	-	-	-	-	-	-
0.50% w/v																			
	0	-	-	-	-	-	-	85.0	45.3	5.3	8.7	118	37	93.6	55.0	6.4	14.1	256	88
	1.12	-	-	-	-	-	-	90.2	48.3	5.7	7.5	133	36	95.1	37.5	5.2	16.2	312	112
	2.24	-	-	-	-	-	-	95.6	52.2	5.6	9.8	150	54	79.8	37.8	5.5	14.1	286	88
1.0% w/v																			
	0	-	-	-	-	-	-	74.5	23.5	6.1	8.0	154	42	-	-	-	-	-	-
	1.12	-	-	-	-	-	-	94.8	25.0	6.2	7.8	129	45	-	-	-	-	-	-
	2.24	-	-	-	-	-	-	93.4	33.0	6.3	8.1	137	45	-	-	-	-	-	-

[1] Unidentified sandy loam from Corvallis, Oregon; data are the means of six replications; analysis presented in Appendix Table 12.

[2] Sandy loam from Corvallis amended with 1/3 peat, 1/3 sand, to 1/3 soil; data are the means of three to six replications (unbalanced); analysis presented in Appendix Table 13.

[3] Willamette silt loam from Canby, Oregon; data are the means of five replications; analysis presented in Appendix Table 14.

[4] 1% w/v = 1 g ground, colonized oat seed to 100 cm^3 soil.

[5] SPA = percent attack (seminal roots); CPA = percent attack (crown roots); SNO = number of seminal roots; CNO = number of crown roots; STWT = dry weight of shoots; RTWT = dry weight of roots

Appendix Table 12. Effects of diclofop-methyl on disease severity and vegetative growth of winter wheat grown in a sandy loam soil inoculated with Gaeumannomyces graminis var. tritici, the fungus causing take-all disease: analysis of variance (1983)[1].

source	d.f.	logit(SPA)[2]			logit(CPA)[2]			SNO			CNO			STWT			RTWT		
		MS	F	p	MS	F	p	MS	F	p	MS	F	p	MS	F	p	MS	F	p
inoculation[3]	2	40.73	42.24	0.00	2.05	45.12	0.00	4.18	4.28	0.02	55.73	12.37	0.00	0.472	30.61	0.00	0.135	59.11	0.00
diclofop-methyl	2	1.39	1.44	0.25	0.01	0.29	0.75	0.21	0.22	0.81	6.40	1.42	0.25	0.006	0.41	0.66	0.006	2.43	0.10
inoc. x diclofop	4	1.21	1.25	0.31	0.01	0.19	0.94	0.36	0.37	0.83	4.71	1.05	0.40	0.004	0.26	0.90	0.003	1.35	0.27
exp. error	38	0.96	-	-	0.05	-	-	0.98	-	-	4.51	-	-	0.015	-	-	0.002	-	-

[1] Statistical analysis for data presented in Appendix Table 11.

[2] Logit transformation for SPA and CPA prior to analysis: logit (PA) = ln (100/(100-(PA+.001)))

[3] Analysis for unbalanced, completely randomized experiment conducted with 8MOP4V(General Univariate and Multivariate ANOVA), 8MOP Statistical Software, 1983 revised version, University of California. Berkeley.

Appendix Table 13. Effects of diclofop-methyl on disease severity and vegetative growth of winter wheat grown in an amended sandy loam soil inoculated with Gaeumannomyces graminis var. tritici, the fungus causing take-all disease: analysis of variance (1983)[1].

source	d.f.	SPA			CPA			SNO			CNO			STWT			RTWT		
		MS	F	p	MS	F	p	MS	F	p	MS	F	p	MS	F	p	MS	F	p
inoculation	1	48467.7	347.07	0.00	13222.0	212.35	0.00	0.76	1.36	0.26	62.66	7.26	0.01	0.241	0.04	0.84	0.141	278.44	0.00
diclofop-methyl	2	186.1	1.33	0.28	247.8	3.98	0.03	0.48	0.86	0.44	9.09	1.05	0.37	0.004	0.00	1.00	0.0003	0.17	0.84
inoc. x diclofop	2	463.9	3.32	0.05	327.5	5.26	0.01	1.94	3.47	0.05	5.65	0.65	0.53	0.003	0.00	1.00	0.0024	1.33	0.28
exp. error	23[2]	139.7	-	-	62.3	-	-	0.56	-	-	8.63	-	-	6.008	-	-	0.0018	-	-

[1] Statistical analysis for data presented in Appendix Table 11.

[2] One missing value calculated; degrees of freedom for experimental error reduced by one prior to calculation of means square error.

Appendix Table 14. Effects of diclofop-methyl on disease severity and vegetative growth of winter wheat grown in an Willamette silt loam soil inoculated with Gaeumannomyces graminis var. tritici, the fungus causing take-all disease: analysis of variance (1983)[1].

source	d.f.	SPA			CPA			SNO			CNO			STWT			RTWT		
		MS	F	p	MS	F	p	MS	F	p	MS	F	p	MS	F	p	MS	F	p
inoculation	2	35104.0	147.57	0.00	5110.2	23.98	0.00	3.08	4.14	0.02	0.05	0.02	0.98	0.222	115.23	0.00	0.066	132.70	0.00
diclofop-methyl	2	1134.5	4.77	0.01	1741.9	8.17	0.00	1.44	1.94	0.16	7.31	2.92	0.06	0.007	3.66	0.03	0.002	4.02	0.02
inoc. x diclofop	4	317.4	1.33	0.27	497.2	2.33	0.07	1.40	1.88	0.13	5.61	2.24	0.08	0.004	2.00	0.11	0.002	3.90	0.01
exp. error	45	237.9	-	-	213.1	-	-	0.74	-	-	2.50	-	-	0.002	-	-	0.0005	-	-

[1] Statistical analysis for data presented in Appendix Table 11.

Appendix Table 15. Effects of four postemergence herbicides on vegetative growth of winter wheat with take-all disease when evaluated on June 4 (Hyslop Farm, 1983).

	Fresh weight/0.3m row (g)				Tillers/0.3m row				Fresh weight/tiller (g)			
	relative application rate (x standard)[1]											
	0	0.5	1.0	1.5	0	0.5	1.0	1.5	0	0.5	1.0	1.5
no inoculum												
diclofop-methyl	346[2]	362	361	338	35.3	36.6	39.4	36.8	9.84	10.05	9.22	9.21
dinoseb	351	364	357	343	35.8	37.0	35.9	33.1	9.85	9.91	10.30	10.47
mecoprop	341	360	295	361	35.9	37.3	30.3	36.6	9.57	9.68	9.80	9.98
difenzoquat	358	361	323	349	37.8	38.1	33.5	38.8	9.48	9.50	9.81	9.07
45 kg/ha inoculum												
diclofop-methyl	314	353	318	331	36.3	36.1	33.3	35.1	8.87	9.81	9.57	9.50
dinoseb	359	342	347	322	37.0	35.6	35.1	32.0	9.92	9.72	9.93	10.16
mecoprop	343	320	347	333	36.9	33.5	36.5	33.5	9.29	9.57	9.50	9.95
difenzoquat	347	353	372	351	36.0	36.9	42.6	39.8	9.66	9.64	8.73	8.83
90 kg/ha inoculum												
diclofop-methyl	305	329	342	326	35.3	35.4	37.0	35.9	8.76	9.33	9.25	9.16
dinoseb	369	311	347	349	39.0	34.8	39.3	35.6	9.39	8.95	8.76	9.76
mecoprop	353	328	339	340	37.5	33.9	34.8	35.4	9.39	9.78	9.70	9.66
difenzoquat	358	326	320	295	39.5	35.5	35.1	34.4	9.11	9.20	9.12	8.61

[1] See Table 1.5 for actual application rates.

[2] All data are the means of eight observations (two subsamples per replication and four replications per treatment); statistical analysis presented in Appendix Table 17.

Appendix Table 16. Effects of four postemergence herbicides on vegetative growth of winter wheat with take-all disease when evaluated on July 3 (Hyslop Farm, 1983).

	Fresh weight/0.3m row (g)				Tillers/0.3m row				Fresh weight/tiller (g)			
	relative application rate (x standard)[1]											
	0	0.5	1.0	1.5	0	0.5	1.0	1.5	0	0.5	1.0	1.5
no inoculum												
diclofop-methyl	321[2]	359	351	337	33.9	38.4	37.6	38.4	9.48	9.36	9.44	8.78
dinoseb	341	329	373	376	37.4	34.3	38.5	39.5	9.25	9.60	9.67	9.55
mecoprop	321	363	339	355	34.8	38.5	35.3	37.4	9.32	9.44	9.56	9.47
difenzoquat	369	371	366	322	39.3	38.8	40.1	36.6	9.44	9.61	9.30	8.86
45 kg/ha inoculum												
diclofop-methyl	303	288	315	296	36.4	33.9	35.5	36.5	8.27	8.65	8.91	8.12
dinoseb	329	303	293	297	37.6	36.8	35.1	37.5	8.75	8.23	8.35	7.99
mecoprop	281	303	287	299	33.6	37.0	34.0	35.0	8.45	8.16	8.45	8.52
difenzoquat	275	298	289	341	35.8	37.0	38.6	40.9	7.70	8.07	7.49	8.47
90 kg/ha inoculum												
diclofop-methyl	274	290	284	312	38.3	37.8	37.3	39.0	7.12	7.77	7.57	8.13
dinoseb	267	268	301	277	37.1	39.3	38.3	32.1	7.17	6.90	7.84	8.65
mecoprop	273	268	276	269	36.1	35.1	31.6	36.9	7.59	7.64	8.68	7.37
difenzoquat	281	278	292	262	34.6	36.9	42.1	38.4	8.12	7.60	7.04	6.95

[1] See Table 1.5 for actual application rates.

[2] All data are the means of eight observations (two subsamples per replication and four replications per treatment); statistical analysis for data presented in Appendix Table 17.

Appendix Table 17. Effects of four postemergence herbicides on vegetative growth of winter wheat with take-all disease when evaluated on June 4 and July 3: analysis of variance (Hyslop Farm, 1983)[1].

| | | June 4 | | | | | | | | | July 3 | | | | | | | | |
| | | FRWT[2] | | | TILNO | | | TILWT | | | FRWT | | | TILNO | | | TILWT | | |
source[3]	d.f.	MS	F	p	MS	F	p	MS	F	p	MS	F	p	MS	F	p	MS	F	p
block	3	3022.4	0.60	0.64	341.5	6.73	0.02	25.19	20.70	0.00	713.6	12.76	0.01	104.8	2.55	0.15	20.12	28.34	0.00
inoc. (I)	2	6881.1	1.37	0.32	0.6	0.01	0.99	7.31	6.01	0.04	166395.0	123.89	0.00	37.9	0.92	0.45	100.19	141.11	0.00
main plot error	6	5014.4	-	-	50.7	-	-	1.22	-	-	1343.1	-	-	41.0	-	-	0.71	-	-
herb. (H)	3	2448.0	0.69	0.56	78.6	1.91	0.15	5.33	5.66	0.00	2013.8	0.64	0.60	126.8	6.77	0.00	2.12	0.93	0.44
I x H	6	3132.3	0.89	0.52	43.5	1.06	0.41	1.02	1.08	0.40	1257.2	0.40	0.87	16.0	0.86	0.54	0.57	0.25	0.96
subplot error	27	3529.5	-	-	41.2	-	-	0.94	-	-	3170.0	-	-	18.7	-	-	2.29	-	-
rate (R)	3	1461.5	0.44	0.72	27.6	0.81	0.49	0.58	0.85	0.47	2207.5	0.79	0.50	21.0	0.70	0.55	0.37	0.33	0.81
I x R	6	3401.8	1.03	0.41	42.3	1.24	0.29	0.10	0.14	0.99	1337.8	0.48	0.82	11.3	0.37	0.89	0.67	0.59	0.74
H x R	9	2759.5	0.83	0.59	26.3	0.77	0.64	1.79	2.62	0.01	1248.1	0.44	0.91	37.8	1.26	0.27	1.39	1.22	0.29
I x H x R	18	2406.5	0.73	0.78	40.3	1.18	0.29	0.68	0.99	0.47	3526.8	1.26	0.23	41.9	1.40	0.15	1.84	1.61	0.07
sub-subplot error	192	3314.8	-	-	34.1	-	-	0.68	-	-	2805.9	-	-	30.0	-	-	1.14	-	-
sampling error	383	3162.1	-	-	30.8	-	-	0.84	-	-	2827.3	-	-	29.4	-	-	1.03	-	-

[1] Statistical analysis for data presented in Appendix Tables 15 and 16.

[2] FRWT = fresh weight/0.3m row (g); TILNO = tillers/0.3m row; TILWT = fresh weight/tiller (g).

[3] Treatments were arranged in a split-split plot design, with inoculation rates as main plots, herbicide type as subplots, and relative herbicide application rate as sub-subplots; sampling error was based upon two subsamples per replication.

Appendix Table 18. Effects of four postemergence herbicides on disease incidence and severity, root growth, and grain yield of winter wheat grown in field plots inoculated with Gaeumannomyces graminis var. tritici, the fungus causing take-all disease (Hyslop Farm, 1983).[1]

treatment	take-all severity[2]				take-all incidence				root dry weight/tiller				grain yield[3]			
	relative application rate (x standard)[4]															
	0	0.5	1.0	1.5	0	0.5	1.0	1.5	0	0.5	1.0	1.5	0	0.5	1.0	1.5
	PA/tiller				-tillers with disease (%)-				mg				kg/ha			
no inoculum																
diclofop-methyl	0.10[5]	0.05	0.40	0	2.9[6]	3.4	12.1	1.0	138[5]	133	154	158	7120[7]	7170	6870	6650
dinoseb	0.19	0	0	0	4.9	2.8	0.5	3.4	153	145	148	148	7100	6900	6670	6970
mecoprop	0.04	0.03	0.50	0.04	1.8	2.7	9.9	5.8	145	120	136	144	6940	7000	6900	7100
difenzoquat	0.04	0.01	0.02	0	3.5	1.8	4.0	3.3	138	135	150	126	7000	6940	6800	7020
45 kg/ha inoculum																
diclofop-methyl	3.12	3.13	1.24	0.32	44.4	52.9	29.4	15.6	145	138	126	155	6290	6570	6440	6190
dinoseb	0.23	2.12	0.60	2.42	15.5	25.8	14.3	40.8	144	177	153	155	5870	6200	6350	5940
mecoprop	4.22	0.89	0.78	2.11	35.5	27.7	17.4	19.6	146	150	157	158	5890	5820	6100	6790
difenzoquat	1.64	0.66	1.54	1.42	30.3	26.5	19.9	38.4	146	136	143	130	6020	6240	6140	6290
90 kg/ha inoculum																
diclofop-methyl	6.50	4.56	3.95	4.38	51.2	49.2	46.2	39.7	133	146	151	134	5550	5600	5790	5700
dinoseb	6.38	3.05	6.12	2.92	35.3	42.2	46.3	43.4	154	156	148	159	5790	6000	5770	6220
mecoprop	2.43	9.66	6.51	2.43	37.5	45.7	49.3	37.3	164	159	148	167	5570	6120	5700	5520
difenzoquat	4.42	2.82	6.66	3.92	42.5	33.8	50.8	40.9	137	154	127	139	5650	5570	5800	5740

[1] Statistical analysis for all data are presented in Appendix Table 19.

[2] Take-all severity and incidence, and root weight evaluated for plants from root cores dug on May 10 ($\pm$ 7 days).

[3] Plots were harvested on August 1.

[4] See Table 1.5 for actual application rates.

[5] Data are the means of 80-240 observations (10-30 tillers per subsample, two subsamples per replication, four replications per treatment).

[6] Data are the means of eight observations (two subsamples per replication, four replications per treatment).

[7] Data are the means of four replications.

Appendix Table 19. Effects of four postemergence herbicides on disease incidence and severity, root growth, and grain yield of winter wheat grown in field plots inoculated with Gaeumannomyces graminis var. tritici, the fungus causing take-all disease: analysis of variance (Hyslop Farm, 1983)[1].

source	d.f.	take-all severity			take-all incidence			root dry weight/tiller			grain yield[2]		
		MS	F	p	MS	F	p	MS(x 10^{-3})	F	p	MS	F	p
block	3	2.09	0.07	0.97	128.2	0.07	0.97	6.48	5.56	0.04	565.8	5.24	0.04
inoculation	2	734.96	24.40	0.00	50169.0	26.73	0.00	1.65	1.41	0.31	5137.9	47.60	0.00
main plot error	6	30.12	-	-	1877.1	-	-	1.16	-	-	107.9	-	-
herbicide	3	6.27	0.14	0.94	673.5	0.75	0.53	4.32	7.52	0.00	8.0	0.06	0.98
inoc. x herb.	6	1.13	0.03	1.00	200.7	0.22	0.97	1.26	2.20	0.07	53.0	0.41	0.86
subplot error	27	45.15	-	-	902.6	-	-	0.57	-	-	128.6	-	-
rate	3	11.93	0.47	0.70	75.0	0.13	0.94	0.14	0.22	0.88	31.7	0.85	0.47
inoc. x rate	6	14.59	0.58	0.75	728.2	1.26	0.28	0.95	1.44	0.21	41.3	1.11	0.36
herb. x rate	9	10.16	0.40	0.93	675.7	1.17	0.32	0.77	1.18	0.32	24.9	0.67	0.73
inoc. x herb. x rate	18	22.41	0.89	0.60	370.7	0.64	0.86	0.79	1.21	0.27	47.5	1.28	0.21
sub-subplot error	108	25.28	-	-	577.8	-	-	0.66	-	-	37.1	-	-
sampling error	192	31.69	-	-	710.1	-	-	0.76	-	-	_3	-	-

[1] Statistical analysis for data presented in Appendix Table 18.

[2] Statistical analysis for yield data based on bushels/A, rather than kg/ha.

[3] Sampling error not calculated for yield data because of single subsample per replication.

Appendix Table 20. Effect of application date of diclofop-methyl on vegetative growth and yield of winter wheat with take-all disease (Hyslop Farm, 1983)[1].

application date[4]	DAP[5]	Fresh weight/ 0.3m row (g) [2]		Tillers/ 0.3m row [2]		Fresh weight/ tiller (g) [2]		Grain yield [3]	
		---------------------------------inoculation rate (kg/ha)--							
		0	90	0	90	0	90	0	90
check	-	359	232	40.3	37.5	8.97	6.18	7490	6070
January 10	83	343	253	37.5	37.4	9.14	6.79	7500	6150
February 2	106	333	245	37.0	39.6	9.03	6.29	7590	5850
February 27	131	335	250	38.4	39.1	8.72	6.36	7490	5620
March 20	152	336	236	36.9	36.4	9.11	6.42	7570	5720
April 4	167	362	234	40.4	40.3	8.97	5.89	7390	5840
April 27	190	329	237	36.1	39.4	9.16	6.05	8050	6090

[1] Statistical analysis for all data presented in Appendix Table 21.

[2] Vegetative growth evaluated on July 10.

[3] Plots harvested on August 1.

[4] Diclofop-methyl applied at 1.12 kg ai/ha at all application dates.

[5] DAP - days after planting.

Appendix Table 21. Effects of application date of diclofop-methyl on vegetative growth and yield of winter wheat with take-all disease: analysis of variance (Hyslop Farm, 1983)[1].

source	d.f.	fresh weight/ 0.3m row (g)			tillers/ 0.3 m row			fresh weight/ tiller (g)			grain yield (bu/A)[2]		
		MS	F	p	MS	F	p	MS	F	p	MS	F	p
block	3	1685.3	0.55	0.68	55.96	1.96	0.30	0.18	0.06	0.98	40.0	0.43	0.75
inoculation	1	291006.0	95.44	0.00	5.58	0.20	0.69	209.00	66.08	0.00	8692.8	93.54	0.00
mainplot error	3	3049.0	-	-	28.53	-	-	3.16	-	-	92.9	-	-
application date	6	6054.3	0.30	0.93	22.49	1.08	0.39	0.48	0.48	0.82	52.7	1.25	0.31
inoc. x date	6	1361.0	0.67	0.67	16.31	0.78	0.59	0.37	0.36	0.90	25.3	0.60	0.73
subplot error	36	2023.4	-	-	20.89	-	-	1.01	-	-	42.3	-	-
sampling error	56	2529.8	-	-	20.17	-	-	1.09	-	-	.3	-	-

[1] Statistical analysis for data presented in Appendix Table 20.

[2] Statistical analysis for yield data based on bushels/A, rather than kg/ha.

[3] Sampling error not calculated for yield data because of single subsample per replication.

Appendix Table 22. Effects of microdroplet application of diclofop-methyl to the leaf blade and collar on vegetative growth of winter wheat (1985)[1].

diclofop-methyl concentration (mM)[2]	shoot dry weight (mg)		root dry weight (mg)		plant dry weight (mg)		shoot/root ration	
	leaf[3]	collar	leaf	collar	leaf	collar	leaf	collar
check	39[4]	48	40	53	79	101	0.98	0.95
2.5	72	69	59	54	131	123	1.21	1.33
5.0	64	60	51	39	115	99	1.26	1.53
10.0	51	53	36	35	87	88	1.58	1.52
20.0	47	44	29	24	76	68	1.66	1.81
40.0	33	41	18	17	51	58	1.92	2.45
80.0	31	26	18	15	49	41	1.73	1.82
160.0	34	32	19	20	53	52	1.89	1.60

[1] Plants were maintained in a growth chamber at 14 C, approximately 250 $\mu E \cdot m^{-2} \cdot sec^{-1}$, and 14-h photoperiod; plants were watered as needed with 1/5 strength Hoagland's No. 2 nutrient solution; plants were treated when 13 days old, and harvested 11 days later; statistical analysis presented in Appendix Table 23.

[2] Triton AG-98 added to all treatment solutions, including the check, at 0.10% v/v.

[3] Four 10 μl-droplets of treatment solution applied to the adaxial surface of either the leaf blade or collar.

[4] Data are the means of three replications.

Appendix Table 23. Effects of microdroplet application of diclofop-methyl to the leaf blade and collar on vegetative growth of winter wheat: analysis of variance (1985)[1].

source	d.f.	shoot dry weight (mg)			root dry weight (mg)			plant dry weight (mg)			shoot/root ratio		
		MS(x 10^{-4})	F	p	MS(x 10^{-4})	F	p	MS(x 10^{-4})	F	p	MS(x 10^{-2})	F	p
block	2	1.50	0.81	0.46	1.22	1.08	0.35	5.27	1.02	0.37	0.62	0.07	0.93
droplet position	1	0.00	0.00	0.99	0.25	0.22	0.64	0.23	0.05	0.83	11.97	1.37	0.25
rate	7	12.50	6.72	0.00	14.04	12.46	0.00	49.83	9.66	0.00	82.53	9.42	0.00
position x rate	7	0.47	0.25	0.97	0.75	0.67	0.70	2.13	0.41	0.89	8.81	1.01	0.45
experimental error	30	1.86	-	-	1.13	-	-	5.16	-	-	8.76	-	-

[1] Statistical analysis for data presented in Appendix Table 22.

Appendix Table 24. Effects of microdroplet application of diclofop-methyl to the leaf blade and collar on tiller development of winter wheat (1985)[1].

diclofop-methyl (mM)[3]	main stem[2]		tiller 1		tiller 10		tiller 11		tiller 2		tiller 20		tiller 21		tiller 3		tiller 4	
	leaf[4]	collar	leaf	collar	leaf	collar	leaf	collar	leaf	collar	leaf	collar	leaf	collar	leaf	collar	leaf	collar
check	6.7	6.7	3.0	3.8	0.7	2.1	0.5	1.1	3.0	3.0	1.7	1.6	0.4	0.4	2.4	2.1	1.3	0.8
0.63	6.8	6.7	3.4	3.8	1.7	2.1	1.2	1.3	2.5	3.2	0.7	1.7	0.3	0.3	2.5	2.3	1.5	1.0
1.25	6.8	6.8	3.9	3.9	1.6	1.7	1.2	1.1	3.3	3.2	1.7	1.2	0.6	0.6	2.5	2.3	1.3	0.8
2.50	6.6	6.4	3.8	3.8	2.2	1.3	1.2	0.7	3.3	3.3	1.7	1.7	0.5	0.4	2.2	2.2	1.1	1.5
5.00	6.6	6.1	3.9	2.8	2.3	0	1.4	0	3.1	2.7	1.7	0	0.6	0	2.3	0	1.2	0
10.00	6.5	5.3	3.7	2.2	2.2	0.7	1.2	0.2	3.0	1.1	1.4	0	0.2	0.1	2.2	0	1.7	0
20.00	6.8	6.6	3.7	3.3	1.5	1.0	0.8	0.2	3.1	2.9	1.7	1.0	0.4	0.4	2.3	0.7	1.2	0.3
40.00	6.7	6.5	3.8	3.9	0.9	0.6	0.7	0	3.1	2.3	1.6	1.5	0.2	0.2	2.3	1.2	1.1	0.5

[1] Statistical analysis presented in Appendix Tables 25 and 26.

[2] Data represent the number of leaves on each tiller at the time of evaluation; system for naming primary and secondary tillers was based on Klepper et al., 1982; plants were treated with herbicide when 25 days old and harvested 36 days later; data are the means of three replications.

[3] Triton AG-98 added to all treatment solutions, including the check, at 0.19% v/v.

[4] Four 5-μl droplets of treatment solution applied to the adaxial surface of the first two leaves (10 μl per leaf) on either the blade or collar.

Appendix Table 25. Effects of microdroplet application of diclofop-methyl to the leaf blade and collar on vegetative growth of winter wheat: analysis of variance (1985).[1]

source	d.f.	main stem			tiller 1			tiller 10			tiller 11		
		MS	F	p	MS	F	p	MS	F	p	MS	F	p
block	2	0.439	1.47	0.25	2.608	5.69	0.01	9.74	16.50	0.00	4.67	14.36	0.00
droplet position	1	1.110	3.71	0.06	0.585	1.28	0.27	2.52	4.27	0.05	2.43	7.48	0.01
rate	7	0.479	1.60	0.17	0.603	1.31	0.28	0.78	1.32	0.28	0.52	1.61	0.17
position x rate	7	0.254	0.85	0.56	0.839	1.83	0.12	1.92	3.25	0.01	0.62	1.90	0.10
experimental error	30	0.300	-	-	0.459	-	-	0.59	-	-	0.33	-	-

[1] Statistical analysis for data presented in Appendix Table 24; analysis continued in Appendix Table 26.

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Appendix Table 26. Effects of microdroplet application of diclofop-methyl to the leaf blade and collar on vegetative growth of winter wheat: analysis of variance (1985)[1].

source	d.f.	tiller 2			tiller 20			tiller 21			tiller 3			tiller 4		
		MS	F	p	MS	F	p	MS	F	p	MS	F	p	MS	F	p
block	2	3.833	5.32	0.01	2.471	6.11	0.01	0.796	4.45	0.02	0.958	6.22	0.01	1.503	6.54	0.00
droplet position	1	1.401	1.94	0.17	2.297	5.68	0.02	0.120	0.67	0.42	11.603	75.29	0.00	3.968	17.28	0.00
rate	7	0.956	1.33	0.27	0.814	2.01	0.09	0.102	0.57	0.77	1.756	11.39	0.00	0.633	2.76	0.02
position x rate	7	0.863	1.20	0.33	1.108	2.74	0.03	0.065	0.36	0.92	1.343	8.71	0.00	0.298	1.30	0.29
experimental error	30	0.721	-	-	0.404	-	-	0.179	-	-	0.154	-	-	0.230	-	-

[1] Statistical analysis for data presented in Appendix Table 24; analysis continued from Appendix Table 25.

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Appendix Table 27. Effects of microdroplet application of diclofop-methyl to the leaf blade and collar on vegetative growth of winter wheat (1985)[1].

diclofop-methyl (mM)[2]	seminal roots/ plant		seminal root dry weight (g)		crown roots/ plant		crown root dry weight (g)		tillers/ plant		shoot weight (g)	
	leaf[3]	collar	leaf	collar	leaf	collar	leaf	collar	leaf	collar	leaf	collar
check	5.0[4]	5.0	0.19	0.22	21.7	26.3	0.32	0.47	7.3	8.3	1.27	1.69
0.63	4.7	5.7	0.17	0.20	26.7	25.0	0.37	0.29	7.0	8.3	1.62	1.46
1.25	5.3	5.0	0.18	0.28	28.7	26.0	0.35	0.35	8.3	8.0	1.70	1.72
2.50	5.3	5.0	0.24	0.19	27.7	24.7	0.46	0.43	8.7	9.7	1.97	1.55
5.00	5.3	5.3	0.25	0.19	24.3	11.0	0.45	0.13	9.0	2.3	1.73	0.62
10.00	5.3	5.0	0.24	0.13	25.7	9.0	0.36	0.08	8.3	2.3	1.72	0.57
20.00	5.3	5.0	0.23	0.18	27.7	17.7	0.45	0.15	7.7	5.7	1.61	0.66
40.00	4.7	5.3	0.15	0.11	26.0	17.0	0.45	0.16	7.3	4.7	1.71	0.71

[1] Statistical analysis presented in Appendix Table 28.

[2] Triton AG-98 added to all treatment solutions, including check, at 0.19% v/v; plants were treated with herbicide when 25 days old and harvested 36 days later.

[3] Four 5-μl droplets of treatment solution applied to the adaxial surface of the first two leaves (10 μl per leaf) on either the blade or collar.

[4] Data are the means of three replications.

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Appendix Table 28. Effects of microdroplet application of diclofop-methyl to the leaf blade and collar on vegetative growth of winter wheat: analysis of variance (1985)[1].

source	d.f.	seminal roots/plant MS (x 10^{-1})	F	p	seminal root dry weight (g) MS (x 10^{-2})	F	p	crown roots/plant MS	F	p	crown root dry weight (g) MS (x 10^{-1})	F	p	tillers/plant MS	F	p	shoot weight (g) MS	F	p
block	2	1.46	0.69	0.51	6.15	15.49	0.00	186.19	17.44	0.00	2.81	27.11	0.00	46.31	15.34	0.00	2.35	23.05	0.00
droplet position	1	0.21	0.10	0.76	0.39	0.98	0.33	520.08	48.71	0.00	2.46	23.71	0.00	38.52	12.83	0.00	3.55	34.81	0.00
rate	7	0.68	0.32	0.94	0.59	1.48	0.21	86.43	8.10	0.00	0.27	2.65	0.03	11.00	3.66	0.01	0.41	4.00	0.00
position x rate	7	4.02	1.89	0.11	0.62	1.57	0.18	75.32	7.06	0.00	0.47	4.55	0.00	14.95	4.98	0.00	0.53	5.19	0.00
exp. error	30	2.13	-	-	0.40	-	-	10.68	-	-	0.10	-	-	3.00	-	-	0.10	-	-

[1] Statistical analysis for data presented in Appendix Table 27.