

# The Effect of Plant Growth Regulators and Nitrogen on Fusarium Head Blight of the Spring Wheat Cultivar Max

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## ABSTRACT

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Application of the growth regulators chlormequat and ethephon and fertilization with nitrogen had no significant effect on the progress of head blight caused by *Fusarium graminearum* on the red spring wheat (*Triticum aestivum*) cultivar Max in greenhouse experiments in which heads were inoculated at anthesis with suspensions of macroconidia. A factorial field experiment was conducted in Quebec in 1991 with three variables: nitrogen (0 or 140 kg/ha), growth regulator (none, ethephon, or chlormequat), and inoculation (none, inoculation with macroconidia at anthesis, or inoculation 1 wk after anthesis). Because of dry conditions that year, no head blight symptoms developed on plants in the field. Seed infection by *F. graminearum* was evaluated with a *Fusarium* selective medium. Nitrogen and growth regulators had no effect on seed infection. In 1992, a 3 × 3 factorial experiment was conducted in Ontario using the same growth regulator treatments and three inoculation treatments (no inoculation, heads inoculated with macroconidia, or rows infested with *Fusarium*-colonized corn kernels that produced ascospores). Both ethephon and chlormequat increased the incidence of spikelet infection, but only in treatments inoculated with infested corn. Chlormequat also increased the incidence of seed infection in noninoculated treatments and treatments with infested corn. These data suggest that growth regulators and nitrogen do not change the inherent susceptibility of wheat heads to *F. graminearum*, but shortened plants may be subject to higher inoculum doses because they are closer to ejected ascospores. The dwarfed plant architecture also may influence the microclimate and production of perithecia and ascospores by the perfect stage on the soil surface.

One of the most important diseases of wheat (*Triticum aestivum* L.) in eastern Canada is Fusarium head blight or head scab caused by *Gibberella zeae* (Schwein.) Petch (anamorph = *Fusarium graminearum* Schwabe). The fungus infects flowering heads of wheat during late June to early July and colonizes developing wheat seed, thereby producing the mycotoxins deoxynivalenol and zearalenone (14). Cultural practices have been shown to influence the incidence of Fusarium head blight. Significantly higher infection and mycotoxin levels were found in wheat planted after corn than in wheat planted after soybeans or barley (16). Recent research in Quebec has focused on intensive cereal management through the use of nitrogen and growth regulators (1). In a study of crop management practices over eight site years in the Maritime provinces of Canada, Martin et al (8) reported that sup-

plemental nitrogen and the plant growth regulator ethephon increased the incidence of natural infection by *F. graminearum* in wheat cultivars. A similar result was found in a naturally infected field trial at Macdonald College in 1990. Application of 480 g/ha of ethephon (Cerone) significantly increased the incidence of seed infection by *Fusarium* spp. in the cultivar Columbus, in both hand- and machine-harvested seed (7). The predominant species, in order of frequency, were *F. sporotrichioides* Sherb., *F. graminearum*, *F. poae* (Peck) Wollenweb., and *F. equiseti* (Corda) Sacc., much as reported by Duthie et al (6).

No previous studies have critically examined the effect of plant growth regulators on Fusarium head blight of inoculated plants in the greenhouse or field. The objective of our work was to determine whether growth regulators affect the inherent susceptibility of the plant or the plant architecture and microclimate, thus indirectly exerting an influence on inoculum production and dispersal. In this paper, we report the effect of the growth regulators ethephon

(Cerone) and chlormequat (Cycocel) and nitrogen application on disease progress on wheat inoculated with macroconidia applied to the heads in the greenhouse. We also report the effect of inoculations of macroconidia or ascospores to wheat treated with these two growth regulators in two field trials. All experiments were performed on the spring wheat cultivar Max, which is susceptible and widely grown in Quebec (5).

## MATERIALS AND METHODS

**Fungal isolates.** *F. graminearum* isolate 7, a single-spore isolate collected in 1990 at the Emil Lods Experimental Farm on the Macdonald Campus, tested for pathogenicity and deposited at the Biosystematics Research Centre, Agriculture Canada, Ottawa, was used in all greenhouse trials and in the 1991 field trial. Isolate 7 produced sparse perithecia on PDA after 2–3 mo in culture. Another isolate of *F. graminearum*, DAOM 178148, from the Biosystematics Research Centre, was used in the 1992 field trial because it produced more perithecia in culture. Both isolates were highly virulent in greenhouse tests on cv. Max.

**Preparation of inoculum.** Macroconidia for inoculation of heads of wheat in the greenhouse and field experiments were produced in carboxymethylcellulose medium (CMC) (4). A 4-mm-diameter plug from the margin of a 5-day-old culture of *F. graminearum* on PDA was placed in 500 ml of CMC medium in a flask and incubated on a rotary shaker (100 rpm) for 7 days at 24 C with 16 hr per day of combined fluorescent and incandescent lighting. Conidia were harvested from the flask by filtration through two layers of sterile cheesecloth. Conidial density was determined with a hemacytometer. Ascospore inoculum was produced on corn kernels. After 500 g of corn was autoclaved in 1-L glass jars for 45 min on two consecutive days, 10 ml of macroconidial suspension was placed in each jar, which was then sealed with a canning lid in which two 6-mm-diameter holes had been punched. A 70-mm-diameter

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filter disk was placed on the inside of the lid to permit air exchange while maintaining sterility. The jars were shaken to distribute spores, then incubated at 20 C under long-wave UV and fluorescent light for about 2 mo.

**Preparation of plants for greenhouse tests.** Seeds of cv. Max were planted three per 12-cm-diameter plastic pot filled with 50% peat medium and 50% pasteurized greenhouse soil. The pots were placed on a temperature-controlled growth bench at 22 C, with incandescent and fluorescent lighting. After 2 wk, plants were thinned to one per pot.

**Inoculum density test.** To determine the volume of spore suspension that would be deposited on a wheat head using an artist's airbrush at 103 kPa of pressure, 10 wheat heads were each uniformly sprayed for 5 sec at a distance of 5 cm with a known concentration of conidia, and then each head was placed in a flask with 99 ml of sterile water. Flasks were agitated on a rotary shaker for 3 min, and the suspension was dilution-plated on PDA amended with 100 µg/ml of chloramphenicol. This test established that approximately 70 µl of suspension was deposited on each head. To determine the inoculum density that would result in symptoms on 50% of the spikelets per head in the greenhouse, the main stem heads of plants were inoculated at anthesis with conidial suspensions at inoculum densities of 0,  $2.78 \times 10^2$ ,  $9.26 \times 10^2$ ,  $2.78 \times 10^3$ ,  $9.26 \times 10^3$ ,  $2.78 \times 10^4$ ,  $9.26 \times 10^4$ , and  $2.78 \times 10^5$  conidia per milliliter, equivalent to 0,  $2.0 \times 10^1$ ,  $6.6 \times 10^1$ ,  $2.0 \times 10^2$ ,  $6.6 \times 10^2$ ,  $2.0 \times 10^3$ ,  $6.6 \times 10^3$ , and  $2.0 \times 10^4$  conidia per head, respectively. Sprayed plants were covered with plastic bags and incubated at room temperatures (22–24 C) for 48 hr under fluorescent lights (12 hr/day). Bags were then removed, and the plants were placed in a greenhouse at 16–25 C, supplemented with 16 hr of light from sodium vapor lamps. Disease was measured daily by counting the number of infected spikelets per head. The experiment was arranged in a randomized complete block design on a greenhouse bench with 12 replicates per treatment and was performed twice.

**Effect of plant growth regulators in the greenhouse.** Eight plants with the first node detectable, Zadoks growth stage 31 (ZGS 31) (17), were sprayed with chlormequat at a rate of 1.2 kg a.i./ha in 345 L/ha of water per hectare, using an automatic spray chamber. Eight more plants with swollen boots (ZGS 45) were sprayed with ethephon at a rate of 480 g a.i./ha in 345 L/ha of water. Eight untreated plants served as controls. Plants were arranged on a greenhouse bench in a randomized complete block design. All plants were treated with approximately 100 ml of 20-20-20 N-P-K fertilizer every week and were watered daily. At anthesis, plants were inoculated

by spraying the main stem head with a suspension containing  $9 \times 10^5$  conidia per milliliter with one drop of Tween 20, previously determined to cause approximately 50% infection after 1 wk. Plants were treated as described in the previous section, and the incidence of infection was assessed daily by counting the percentage of infected spikelets per head. Treatment effects were determined by comparing the areas under the disease progress curve (AUDPC) (3). This experiment was conducted twice.

**Effect of nitrogen fertilizer and ethephon in the greenhouse.** Wheat plants were grown as described above. After 2 wk, plants were thinned to one per pot and 16 pots were fertilized with ammonium nitrate at 140 kg N/ha or approximately 0.034 g N per plant. Another 16 pots were treated with water. When plants reached ZGS 45, eight unfertilized and eight fertilized plants were sprayed with ethephon and inoculated as previously described.

**Field experiment, 1991.** In 1991, the effects of nitrogen fertilization and growth regulator application on *Fusarium* head blight were studied in inoculated field plots of cv. Max in a fine sandy loam soil in the plant pathology field on Macdonald Campus. The field had been fallow the previous year and was plowed in the fall. In the spring, oat straw (5 t/ha) was disked into the field 2 wk prior to sowing to immobilize the nitrogen. The experiment was arranged as a  $3 \times 3 \times 2$  factorial in a randomized split-plot design with four replicates (blocks). The main plots were inoculation treatments: no inoculation and inoculation 1 or 2 wk after anthesis. Subplots were growth regulator treatments: no treatment and treatment with chlormequat or ethephon. Sub-subplots were nitrogen treatments: no nitrogen or ammonium nitrate. Each plot was 3.8 m long, with 11 rows spaced 10 cm apart. Each main plot was separated by a 9-m border plot of cv. Max, and each block was separated by a 3-m bare walkway. Plots were seeded on 7 May by a tractor seeder at 450 seeds per square meter. One week after seeding, half of the plots were hand-fertilized with nitrogen in the form of ammonium nitrate at 140 kg N/ha. When the wheat reached ZGS 31, chlormequat plots were sprayed at a rate of 1.2 kg a.i./ha. When plants in the ethephon plots reached ZGS 45, they were sprayed with 480 g a.i./ha of ethephon. All growth regulators were applied with a backpack sprayer. Plots not treated with growth regulators served as controls. On 9 July, 1 wk after anthesis, the heads of plants in one treatment were inoculated with macroconidia of *F. graminearum* using spray bottles filled with a suspension of  $6 \times 10^5$  conidia per milliliter. In another treatment, heads were inoculated 2 wk after anthesis. Uninoculated plots served as control treat-

ments. All plots were irrigated with a lawn sprinkler for 15 min every 2 hr during the day, for 2 wk after inoculation. The incidence of infection by *F. graminearum* was assessed by plating 100 seeds harvested from each plot on selective medium developed to induce rapid sporulation of *Fusarium* spp. The medium contained agar (20 g/L), potato-dextrose broth (12 g/L), pentachloronitrobenzene (1.5 g/L), and chloramphenicol (0.5 g/L). Seeds were surface-disinfested for 6 min in 0.5% sodium hypochlorite, rinsed three times in sterile distilled water, blotted on sterile paper towels, and plated, 10 per plate. Plates were incubated at room temperature (22–24 C) for 5 days. The *Fusarium* colonies originating from the seeds sporulated rapidly, and *F. graminearum* was identified by the carmine-red color of the colony and the shape and size of the macroconidia (10).

**Field experiment, 1992.** In 1992, a similar field experiment was conducted at the Central Experimental Farm of Agriculture Canada in Ottawa, where an automatic misting system was available to create more favorable conditions for disease. A different isolate of *F. graminearum* (DAOM 178148) was used, one that produced perithecia more profusely than the isolate used in 1991. The nitrogen applications were omitted in 1992, since the nitrogen level in the field was already high (>100 kg/ha). The land was previously cropped to red clover, which had been plowed under the previous fall, and in the spring was disked and cultivated. The experiment was a  $3 \times 3$  factorial arranged in a split-plot design with four replicates (blocks). Each plot was 3 m long, with eight rows 22 cm apart. Main plots were inoculation treatments (noninoculated and macroconidia or ascospore inoculation). Subplots were growth regulator treatments (no growth regulator and chlormequat or ethephon). Main plots were separated by a 3-m border of cv. Fielder, and blocks were separated by a 3-m walkway. Wheat was seeded on 15 May. The herbicide bromoxynil was applied to all plots in mid-June to control broadleaf weeds. The growth regulators were applied as in the 1991 experiment. Macroconidia inoculum was applied with a backpack sprayer at anthesis on 14 July as previously described. On 10 June, when the wheat was approximately 15 cm high, 500 g of *Fusarium*-colonized corn kernels was applied to the ascospore inoculation treatments. By the end of June, corn kernels were covered with purple-black perithecia containing mature ascospores. After anthesis and until disease symptoms appeared, plots were irrigated with an automatic mist system, which turned on for 30 sec every 5 min from 6 a.m. to 8 p.m. Disease was assessed at the soft dough stage in early August by counting the number of infected spikelets

per head on 50 heads arbitrarily chosen from the middle of each plot. Seed infection was assessed by plating 100 seeds from each plot on *Fusarium*-selective medium, as previously described.

**Statistical analysis.** All greenhouse tests were done twice. Data were analyzed with analysis of variance procedures to determine the significance of treatment effects and their interactions (12). A protected least significant difference (LSD) test at the 0.05 level was carried out to compare treatments. Orthogonal contrast tests were used on data from the 1992 field trial.

## RESULTS

**Inoculum density test.** In the greenhouse, approximately 50% of the spikelets per head were diseased when plants were inoculated with a conidial suspension of  $9.26 \times 10^3$  conidia per milliliter, with a calculated conidial load of  $6.66 \times 10^2$  conidia per head. The relationship between inoculum density and the percentage of spikelets diseased was described by the linear regression  $Y = -97.02 + 35.25X$ , where  $Y$  is the arcsine transformation of the percentage of spikelets per head showing symptoms and  $X$  is the log (inoculum density + 1), where inoculum density is expressed as conidia per milliliter. The  $R^2$  of the equation was 0.91.

**Effect of plant growth regulators in the greenhouse.** All plants inoculated with  $6.67 \times 10^2$  conidia per head showed *Fusarium* head blight symptoms 5–6 days after inoculation. Two weeks after inoculation, the percentage of spikelets diseased per head ranged from 60 to 80% in all treatments. There were no significant differences in AUDPC values among growth regulator treatments in either experiment. The overall mean AUDPC for experiments 1 and 2 was 469 and 439, respectively.

**Effect of nitrogen fertilizer and ethephon in the greenhouse.** The application of ammonium nitrate and ethephon did not affect the AUDPC of macroconidia-inoculated wheat in the greenhouse. The overall mean AUDPC for experiments 1 and 2 was 448 and 472, respectively. There was no significant interaction between nitrogen and growth regulators.

**Field experiment, 1991.** The summer of 1991 was dry. During the 2-wk period after inoculation (6–20 July), there were only 11 mm of rain, with high temperatures frequently above 30 C during the second week. No symptoms of *Fusarium* head blight were observed in the field, but seed infection ranged from 2 to 20% in treatments inoculated with macroconidia. Only inoculation treatments had a significant effect on seed infection (Table 1); there were no significant interactions between variables. There was no seed infection in the noninoculated treatment, 10% infection in the treatment inoculated at anthesis, and 8% infection

in the treatment inoculated 1 wk after anthesis. Nitrogen showed no significant main effect ( $P = 0.93$ ) or interactions. Growth regulator treatment did not have a significant main effect ( $P = 0.06$ ).

**Field experiment, 1992.** The summer of 1992 was much wetter than the summer of 1991 (July rainfall was approximately 150% of normal), and disease severity was much higher than in 1991. Incidence of spikelet infection ranged from 2–4% in noninoculated treatments to 7–22% in inoculated treatments. The incidence of seed infection ranged from 12–31% in noninoculated treatments to 74–85% in inoculated treatments. Both growth regulators and inoculation treatments had significant effects on spikelet infection and interacted with each other (Table 2). In plots treated with *Fusarium*-colonized corn, where the inoculum source was ascospores, incidence of scabby spikelets was higher in treatments sprayed with chlormequat or ethephon than in treatments with no growth regulator. However, no significant differences among growth regulator treatments were seen in noninoculated plots or plots inoculated with macroconidia. Both inoculation and growth regulator treatments also had a significant effect on incidence of seed infection, but the interaction between the two variables was not

significant. When compared with the nontreated control, chlormequat significantly increased seed infection both in the noninoculated plots and in plots treated with *Fusarium*-colonized corn (ascospores) (Table 3). Growth regulators had no effect on seed infection in plots where the heads were directly inoculated with macroconidia.

## DISCUSSION

Nitrogen and growth regulators had no effect on head blight when heads of wheat plants in the greenhouse were inoculated with macroconidia of *F. graminearum*. Although efforts were made to use a realistic inoculum dose, the postinoculation conditions favorable for infection (48 hr of continuous wetness) in the greenhouse and resulting disease severity may have masked any physiological changes in resistance mediated by nitrogen or growth regulators. If growth regulators and nitrogen do not significantly change the inherent susceptibility of anthers or developing seed to the fungus, direct inoculation of the head with macroconidia would not show any differences.

Nitrogen also failed to affect disease in the field in 1991. Although 1991 was a dry year and no visible symptoms

**Table 1.** The effect of growth regulators and inoculation treatment on seed infection of spring wheat (cv. Max) by *Fusarium graminearum* in the 1991 field trial

Inoculation treatment	Incidence of seed infection (%)		
	Control	Ethephon	Chlormequat
Water at anthesis	0	0	0
Macroconidia at anthesis	9.1 a <sup>z</sup>	7.4 a	11.7 a
Macroconidia 1 wk after anthesis	6.1 a	6.5 a	9.8 a

<sup>z</sup> Treatments within a row followed by the same letter are not significantly different according to a protected least significant difference test,  $P = 0.05$ .

**Table 2.** The effect of growth regulators and inoculation treatment on spikelet infection of spring wheat (cv. Max) by *Fusarium graminearum* in the 1992 field trial

Inoculation treatment	Incidence of spikelet infection per head (%)		
	Control	Ethephon	Chlormequat
Noninoculated	1.6 a <sup>z</sup>	1.6 a	1.6 a
Ascospore inoculation from perithecia on colonized corn	9.4 a	13.4 b	13.6 b
Inoculation of heads at anthesis with macroconidia	13.1 a	10.7 a	13.6 a

<sup>z</sup> Treatments within a row followed by the same letter are not significantly different according to orthogonal contrasts,  $P = 0.05$ .

**Table 3.** The effect of growth regulators and inoculation treatment on seed infection of spring wheat (cv. Max) by *Fusarium graminearum* in the 1992 field trial

Inoculation treatment	Incidence of seed infection (%)		
	Control	Ethephon	Chlormequat
Noninoculated	11.3 a <sup>z</sup>	16.2 a	22.6 b
Ascospore inoculation from perithecia on colonized corn	75.8 a	82.2 a	87.1 b
Inoculation of heads at anthesis with macroconidia	75.2 a	77.4 a	80.6 a

<sup>z</sup> Treatments within a row followed by the same letter are not significantly different according to orthogonal contrasts,  $P = 0.05$ .

developed, we believe that differences in seed infection would reflect any differences in susceptibility mediated by the nitrogen. The level of nitrogen in the plots was not measured after planting, but the wheat was taller in fertilized plots than in nonfertilized plots, so nitrogen was probably a limiting factor. Literature on the effect of nitrogen on *Fusarium* diseases is contradictory. Supplementary nitrogen increased the incidence of head blight in naturally infested fields (8) and also increased root rot of winter wheat seedlings caused by *F. graminearum* (11). Others found that head blight and mycotoxin levels were not affected by the level of applied nitrogen but were greater when the preceding crop was maize (16). The form of nitrogen is also important, as shown in previous studies on other diseases. Wheat fertilized with urea had less head blight than that fertilized with ammonium nitrogen (15). In another study, *Fusarium* root rot severity was greater with use of ammonium nitrogen than with nitrate nitrogen (11).

Application of growth regulators in the field in 1991 had no effect on seed infection by *F. graminearum*, possibly because of unfavorable conditions that year. Growth regulators had no effect in the 1992 field trial in spray-inoculated plots, when conditions were favorable for disease development because of high rainfall and misting. In all head-inoculation treatments, macroconidia were applied directly to the head, thus eliminating the effects of growth regulators on microclimate in the canopy and position of susceptible heads relative to the inoculum.

The use of *Fusarium*-colonized corn kernels in the field more closely simulated natural inoculum in the field. In eastern Canada, the fungus readily forms the perfect stage, *G. zeae*, which produces black-purple perithecia in the spring on crop debris infected the previous year. Ascospores are thought to be more important than macroconidia in the epidemiology of this disease in eastern Canada (14). Ascospores are forcibly discharged during evening hours in response to falling temperatures and rising relative humidity and can be detected above the wheat canopy during a 3-wk period that coincides with anthesis (T. C. Paulitz, unpublished). In treatments that used mature perithecia, chlormequat signifi-

cantly increased both spikelet and seed infection, although not in treatments inoculated with macroconidia. Chlormequat also increased seed infection in non-inoculated plots, which probably received inoculum that had drifted from inoculated plots. Etephon also increased spikelet infection in our study.

The difference in disease between treatments in which heads were inoculated with macroconidia and treatments receiving ascospore inoculum from perithecia suggests that plant growth regulators increase head blight by decreasing head height and placing it closer to the inoculum source. Plants treated with chlormequat and etephon were on average 18 and 12 cm shorter, respectively, than nontreated plants. Martin et al (8) observed that etephon increased *Fusarium* head blight in field plots with natural inoculum. Bockmann (2) reported similar results with chlormequat and *F. culmorum* and *Septoria nodorum* on wheat and suggested that one of the causes was the shorter distance that spores would have to travel to reach leaves and heads. Mesterhazy (9) showed that dwarf genotypes of wheat were more susceptible to head blight, based on natural infection in field plots. Artificial inoculation tests did not show significant differences, suggesting that plant height is a morphological trait that influences natural infection but does not change plant resistance. The inoculum potential of ascospores and macroconidia is similar (13), so the difference between the two treatments is probably due to differences in inoculum density, not inoculum potential or physiological differences in the plants. Another possibility is that the dwarf habit of wheat treated with growth regulators may influence the microenvironment of crop debris on the soil, leading to greater production of perithecia and ascospores.

Further field studies should be done, using the *Fusarium*-colonized corn inoculation method. The method is easy to use and does not require exotic media or special equipment to produce ascospore inoculum. This method should also be used more extensively in cultivar trials to select for cultivars that may escape head blight because of growth habit or plant architecture. These results also have important implications for disease control in more intensive cereal manage-

ment using growth regulators or dwarf cultivars.

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