# Interactive Effects of Plant Growth Regulators and Fungicides on Epidemics of Dollar Spot in Creeping Bentgrass

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## ABSTRACT

Burpee, L. L., Green, D. E., and Stephens, S. L. 1996. Interactive effects of plant growth regulators and fungicides on epidemics of dollar spot in creeping bentgrass. Plant Dis. 80:1245-1250.

Studies were conducted to assess interactions among gibberellin inhibitor plant growth regulators (PGRs) and fungicides on dollar spot of creeping bentgrass caused by *Sclerotinia homoeocarpa.* The PGRs paclobutrazol and flurprimidol were fungistatic to *S. homoeocarpa* in vitro at significantly lower concentrations ($EC_{50}$ < 0.5 μg/ml) than was the PGR trinexapac-ethyl ($EC_{50}$ > 15 μg/ml). In the field, paclobutrazol, flurprimidol, and trinexapac-ethyl were applied to creeping bentgrass at the respective rates of 0.16, 0.33, and 0.19 kg a.i./ha either alone or as pretreatments 4 days prior to applications of the fungicides chlorothalonil, iprodione, propiconazole, and thiophanate-methyl at 9.6, 3.1, 0.4, and 3.1 kg a.i./ha, respectively. In plots treated with the PGRs alone, rates of dollar spot epidemics ($dy/dt$, where $y$ = amount of disease and $t$ = time) were significantly lower in bentgrass treated with flurprimidol in 1993 and 1994 or with paclobutrazol in 1994 compared to nontreated turf and turf treated with trinexapac-ethyl. Similar effects were detected from analysis of areas under disease progress curves (AUDPC). The trinexapac-ethyl treatment had no significant effect on epidemic rates or values of AUDPC compared to nontreated controls in 1993 and 1994. Based on values of AUDPC and/or time to reach 5% disease severity, pretreatment of turf with flurprimidol significantly enhanced the efficacy of chlorothalonil, iprodione, and propiconazole in 1994 and each of the four fungicides tested in 1995. Pretreatment with paclobutrazol resulted in effects similar to those observed with the flurprimidol pretreatment in 1994, but paclobutrazol did not enhance the efficacy of chlorothalonil or thiophanate-methyl in 1995. Pretreatment with trinexapac-ethyl significantly enhanced the efficacy of chlorothalonil, iprodione, and propiconazole in 1994, but had no effect on fungicide efficacy in 1995. Foliar clipping weights were significantly greater in plots treated with any of the PGRs 28 days after treatment in 1993 compared to nontreated controls. In 1994, canopy heights of bentgrass were significantly lower in PGR-treated plots 10 and 18 days after treatment compared to nontreated controls.

Synthetic plant growth regulators (PGRs) have been used by turfgrass managers for more than 40 years to regulate the canopy height of grasses and reduce mowing frequency (20). The PGRs that are registered for use on turfgrasses include six compounds classified as either mitotic inhibitors or inhibitors of gibberellin biosynthesis (5). The latter group includes paclobutrazol, flurprimidol, and trinexapac-ethyl. Use of gibberellin biosynthesis inhibitors has stimulated interest in PGRs among turfgrass managers because of a reduced potential for phytotoxicity compared with applications of mitotic inhibitors (20). Furthermore, applications of some gibberellin inhibitors have resulted in additional beneficial effects, including improved leaf color (13) and increased stand density (6).

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Accepted for publication 22 July 1996.

Publication no. D-1996-0816-04R


Reports of the effects of PGRs on several turfgrass diseases describe increases in disease severity after PGR treatment (7,8,19). This increase has been attributed generally to reduced growth rates and recuperative potential of treated grasses (20), but stimulatory effects of PGRs on sporulation and spore germination of pathogens (15) may play a role. Triazole and pyrimidine PGRs are fungistatic in vitro and are chemically related to several fungicides (9,14,16). However, the disease suppressive potential of these and related PGRs has not been elucidated.

Dollar spot, caused by *Sclerotinia homoeocarpa* F.T. Bennett, is a foliar disease of turfgrasses grown in temperate and tropical environments (4). The disease can be particularly severe on closely mowed turf (canopy height <1.3 cm), where infection results in necrotic, white to straw-colored leaves and tillers occurring in foci usually <5 cm in diameter. Fungicide applications are often required to achieve acceptable control of dollar spot on turf that also may be treated with PGRs, e.g., creeping bentgrass (*Agrostis palustris* Huds.) in golf course fairways. The objectives of our research were to determine the effects of some commonly used gibberellin inhibitor PGRs on (i) the growth of *S. homoeocarpa* in vitro, (ii) the severity of dollar spot of creeping bentgrass, and (iii) the efficacy of fungicides that are applied to control dollar spot.

## MATERIALS AND METHODS

**Laboratory studies.** Commercial formulations of the PGRs, paclobutrazol (TGR Turf Enhancer 50WP, O.M. Scott and Sons, Marysville, OH), flurprimidol (Cutless 50W, DowElanco, Indianapolis, IN), and trinexapac-ethyl (Primo 1.0E, Ciba Geigy Corp., Greensboro, NC), were diluted to concentrations of 0.1, 1.0, 10, and 100 μg/ml in cooled (50°C) autoclaved potato-dextrose agar (PDA) and poured into 9-cm-diameter petri dishes. Six-mm-diameter mycelial plugs from the margins of a colony of *S. homoeocarpa* (isolate S084) growing on PDA were transferred to the center of each dish and incubated at 24°C in the dark. Colony diameters were measured to the nearest millimeter at intervals of 24, 48, and 72 h after transfer. Each treatment was replicated three times, and the experiment was repeated once. Effective concentration (EC) values, representing reductions of mycelial growth by 50 and 90%, were determined for each replicate by regressing probit transformed percent inhibition of growth against $\log_{10}$ concentrations of the PGRs. Replicate values of $EC_{50}$ and $EC_{90}$ were subjected to analysis of variance, and means were separated using Duncan's least significant difference test at $\alpha \leq 0.05$.

**Field studies.** Experiments were conducted in 1993, 1994, and 1995 on a sward of creeping bentgrass cv. Penncross at the Georgia Experiment Station, Griffin. The

**Table 1.** Effective concentration (EC) of plant growth regulators (PGRs) required to reduce mycelial growth of *Sclerotinia homoeocarpa* by 50 and 90%

| PGR | $EC_{50}$[y] | $EC_{90}$[y] |
|---|---|---|
| Paclobutrazol | 0.10 b[z] | 0.82 b |
| Flurprimidol | 0.21 b | 2.96 b |
| Trinexapac-ethyl | 15.89 a | 29.64 a |

[y] μg/ml.
[z] Within a column, values followed by the same letter are not significantly different at $\alpha$ = 0.05 according to Duncan's LSD.

turfgrass was established from seed planted in September 1990 on a sand:peat mix (90:10 wt/wt) and maintained as prescribed for bentgrass golf greens (2), with the exception of mowing height in 1994 and 1995. The turf was mowed three times per week at heights of 0.4 cm in 1993 and 1.6 cm in 1994 and 1995, which approximated standards for golf greens and fairways, respectively (2). The fertility regime included monthly applications of 24-4-10 (N-P-K) at rates of 24.4 kg N/ha, with the exception of May, June, and July, when ureaformaldehyde was applied at 12.2 kg N/ha/month. Fertilizer was not applied in August, January, or February. Overhead irrigation was applied periodically to prevent drought stress.

Plots of creeping bentgrass (1 × 2 m) were treated with the PGRs paclobutrazol, flurprimidol, or trinexapac-ethyl on 1 September 1993 at label rates of 0.16, 0.33, and 0.19 kg a.i./ha, respectively. Materials were applied in 700 liters of water per ha with a wheel-mounted, $CO_2$-pressured boom sprayer at 138 kPa. The PGR treatments and a nontreated control were arranged in a randomized complete block design with four replicates.

The PGR treatments were repeated on 2 May 1994 at a different site within the bentgrass sward. Applications were made in subplots (1 × 2 m) of a split-plot design with four replicates. The fungicides chlorothalonil (Daconil 2787 500F, ISK Biosciences, Mentor, OH), iprodione (Chipco26019 50WP, Rhone-Poulenc Ag Co., Research Triangle Park, NC), propiconazole (Banner 1.1E, Ciba Geigy Corp., Greensboro, NC), or thiophanate-methyl (Cleary's 3336 50WP, W.A. Cleary Chemical Co., Somerset, NJ) were applied in the main plots (5 × 2 m) at label rates of 9.6, 3.1, 0.4, and 3.1 kg a.i./ha, respectively. Fungicides were selected as main plots to validly analyze the effects of PGRs within rather than among fungicides. The application equipment and dilutions were the same as described for the 1993 experiment. Fungicides were applied 4 days after the PGRs to limit any potential for phytotoxicity. Nontreated main and subplots served as controls.

On 12 May 1995, fungicide and PGR plus fungicide treatments were repeated as individual plots (1 × 2 m) in four separate experiments (one for each fungicide) in randomized complete block designs with four replicates each. The sites for these studies within the bentgrass sward were different from those used in the 1993 and 1994 experiments. Treatments were applied as described for the 1994 experiment.

In each year, the turf was inoculated with *S. homoeocarpa* (isolate S084) 4 h after the PGR (1993) or fungicide (1994 and 1995) treatments by hand dispersal of rye grain infested with the fungus into the plots at a rate of approximately 7 g of grain per $m^2$. The inoculum was prepared as described for grain infested by *Rhizoctonia solani* (3). The turf received approximately 4 mm of irrigation nightly at 2000 h throughout each experiment to ensure long durations of foliar wetness for disease development.

Disease severity was assessed by counting the number of dollar spot foci per plot at 1- to 3-day intervals for the first 11 to 20 days after inoculation, at which time

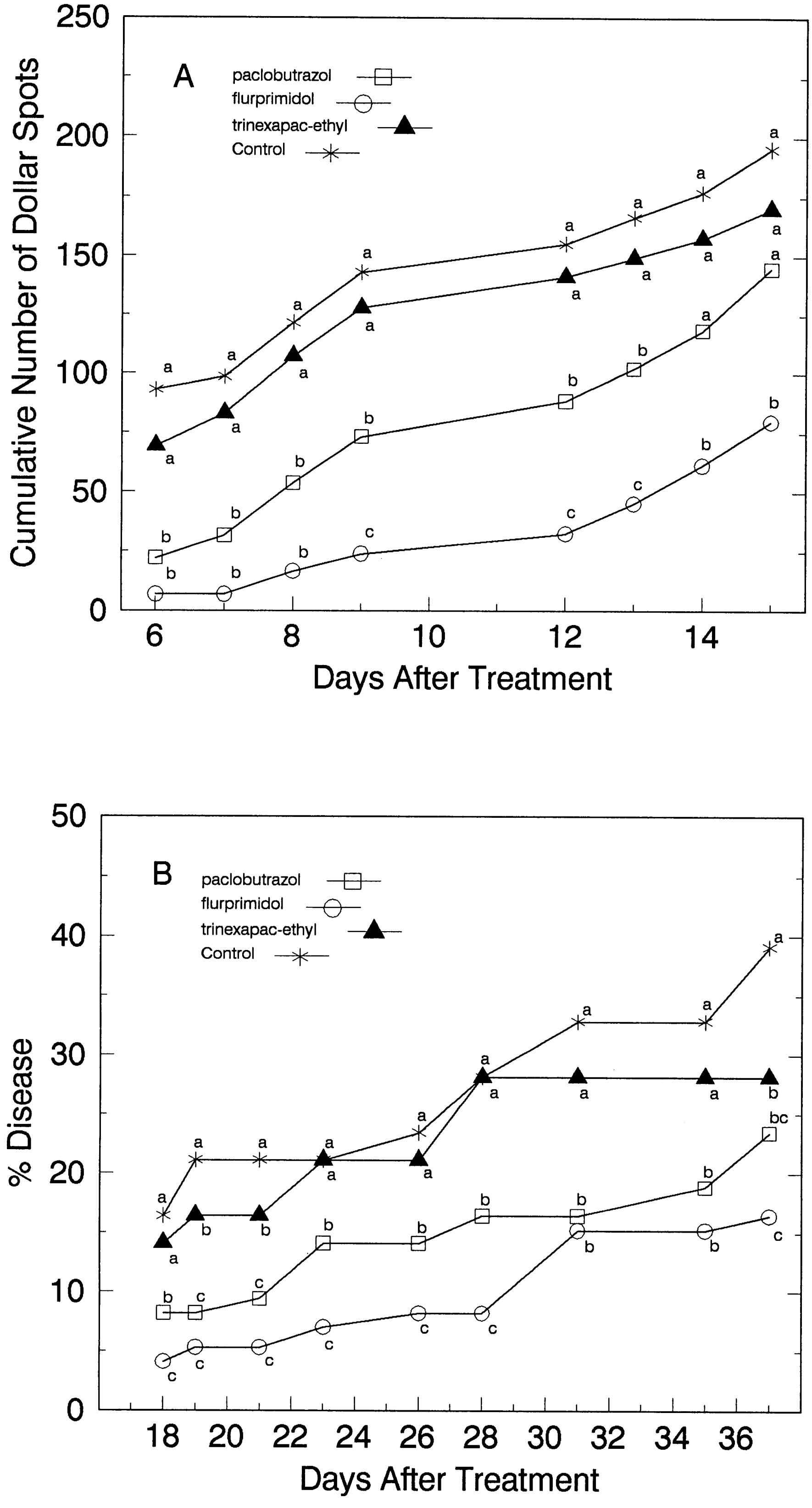


**Fig. 1.** Epidemics of dollar spot in plots of creeping bentgrass treated with plant growth regulators in 1993. **(A)** early epidemics based on counts of dollar spot foci and **(B)** late epidemics based on visual estimates of disease severity.

counting became prohibitive due to the large number of spots formed each day. After these periods, disease severity was visually assessed at 1- to 7-day intervals for up to 37 days after PGR (1993) or fungicide treatment (1994 and 1995) by estimating the percent necrotic turf area per plot using the Horsfall-Barratt rating scale (11).

An area under a disease progress curve (AUDPC) was calculated (17) for each treatment replicate using the formula $\Sigma[(y_i + y_{i+1})(t_{i+1} - t_i)/2]$, for which $i = 1,2,3,...,n - 1$, $y_i$ is the severity of disease, and $t_i$ is the time of the $i$th rating. Separate values of AUDPC were calculated based on cumulative numbers of dollar spots and visual estimates of percent area of necrotic turf in each plot. Estimates of the time required for epidemics to reach a threshold of 5% disease severity were derived from the Horsfall-Barratt ratings. Data were subjected to analysis of variance, and means were statistically separated using Duncan's least significant difference test (18) at $\alpha \leq 0.05$. Rates of disease increase were determined from slopes of disease progress curves generated from linear, logistic, Gompertz, or monomolecular regression models. Selection of a model was based on coefficients of determination, standard errors for $Y$, and visual inspection of residual plots (18).

Effects of the PGRs on turfgrass growth were determined by measuring oven-dry weight of foliar clippings in 1993 and height of foliar canopies in 1994. Clippings were removed from each plot at 14, 28, and 42 days after treatment with a reel mower equipped with a collection basket and set to a mowing height of 0.4 cm. On each collection date, the turf canopy had been allowed to grow for 5 to 7 days since a previous mowing. Clippings were dried in paper bags at 100°C for 24 h and weighed. Canopy heights were measured with a ruler at five random locations on a diagonal transect across each plot at 10, 18, and 32 days after treatment. Data were subjected to analysis of variance, and means were separated using Duncan's least significant difference test at $\alpha \leq 0.05$.

## RESULTS

**Laboratory studies.** Each of the PGRs significantly suppressed mycelial growth of *S. homoeocarpa* within the range of dilutions tested. The $EC_{50}$ and $EC_{90}$ values for paclobutrazol and flurprimidol were significantly less than those for trinexapac-ethyl (Table 1). After 72 h of incubation, mycelial plugs removed from agar containing 10 or 100 μg/ml of each PGR produced colonies on unamended PDA, indicating a fungistatic rather than fungicidal effect of the PGRs.

**Field studies.** In 1993, severity of dollar spot (numbers of disease foci) was significantly less in plots treated with flurprimidol or paclobutrazol than in nontreated plots or in plots treated with trinexapac-ethyl from 6 to 14 days after treatment (Fig. 1A). Disease severity was significantly less in plots treated with flurprimidol than in other plots from 9 to 15 days after treatment, while the trinexapac-ethyl treatment had no significant effect on the incidence of dollar spot during this period.

Later in the epidemics, severity of dollar spot was significantly less in plots treated

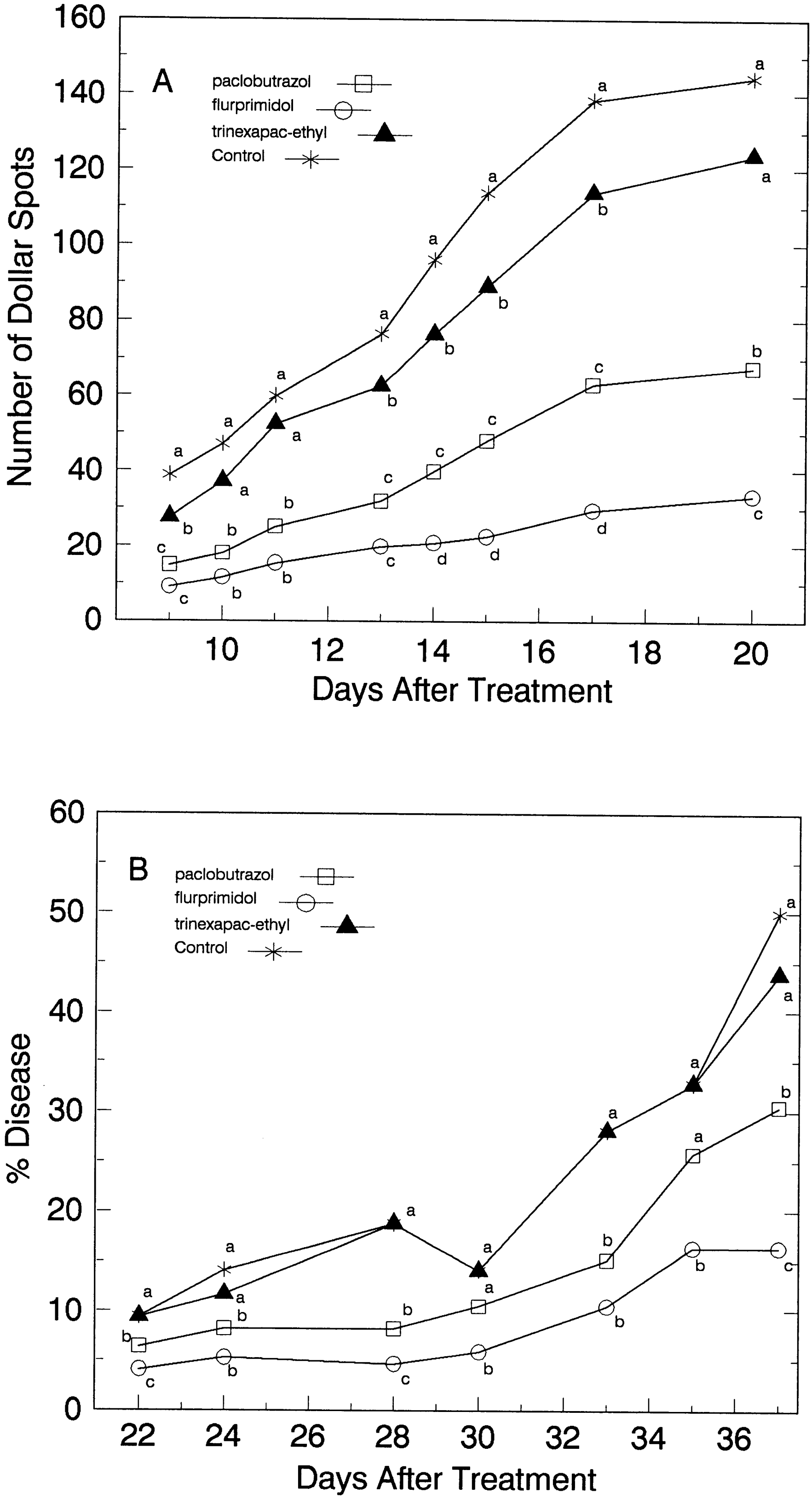


**Fig. 2.** Epidemics of dollar spot in plots of creeping bentgrass treated with plant growth regulators in 1994. **(A)** early epidemics based on counts of dollar spot foci and **(B)** late epidemics based on visual estimates of disease severity.

with paclobutrazol or flurprimidol than in nontreated plots or plots treated with trinexapac-ethyl from 18 to 35 days after treatment (Fig. 1B). During this period, the effects of paclobutrazol and flurprimidol were not significantly different from each other on most rating dates, and the trinexapac-ethyl treatment had no significant effect on disease severity.

Factorial analysis of data from the 1994 experiment revealed significant effects of fungicides, PGRs, and fungicide × PGR interactions on each of the measured variables associated with dollar spot epidemics (Table 2). Due to the interactions, data from plots treated with the PGRs alone (no fungicide) were analyzed to obtain unbiased effects of these compounds on dollar spot. Interactive effects were revealed by statistically separating the mean effects of the PGR treatments (subplots) within each fungicide treatment (main plot). Effects of the PGRs on dollar spot were similar to those observed in 1993, except that trinexapac-ethyl produced significant disease suppression from 13 to 17 days after treatment (Fig. 2A). Significantly fewer dollar spots developed in plots treated with paclobutrazol or flurprimidol than in nontreated plots or plots treated with trinexapac-ethyl from 9 to 20 days after treatment. Flurprimidol was significantly more disease suppressive that pachlobutrazol from 14 to 20 days after treatment. From 22 to 37 days after treatment, the severity of dollar spot was significantly less in plots treated with paclobutrazol or flurprimidol than in nontreated plots or plots treated with trinexapac-ethyl, but disease was not significantly suppressed by the trinexapac-ethyl treatment (Fig. 2B).

Rates of dollar spot epidemics, based on counts of disease foci up to 20 days after PGR treatment, were significantly suppressed by flurprimidol in 1993 and 1994, and by paclobutrazol in 1994 (Table 3). Similarly, the paclobutrazol and flurprimidol treatments resulted in significantly lower AUDPC values than did trinexapac-ethyl or the nontreated control for early epidemics in 1993 and 1994 (Table 4). The trinexapac-ethyl treatment had no significant effect on rates (Table 3) or AUDPC (Table 4) of early epidemics in either year. None of the PGR treatments significantly affected epidemic rates from 18 to 37 and from 22 to 37 days after treatment in 1993 and 1994, respectively. However, values of AUDPC for these periods were significantly lower for the paclobutrazol and flurprimidol treatments than for trinexapac-ethyl or the nontreated control in both years (Table 4).

In 1994, pretreatment with paclobutrazol or flurprimidol resulted in a significant increase in the number of days required to reach 5% dollar spot severity in plots treated with chlorothalonil compared to plots treated with the fungicide alone (Table 5). Each of the PGRs produced a similar increase in plots treated with iprodione, but not in plots treated with propiconazole or thiophanate-methyl. In 1995, days to 5% disease were significantly increased by pretreatment with flurprimidol in plots treated with iprodione, propiconazole, or thiophanate-methyl (Table 6). Pretreatment with paclobutrazol or trinexapac-ethyl had no significant effect on days to 5% disease in any of the fungicide-treated plots.

In 1994, pretreatment of turf with any of the PGRs in plots treated with chlorothalonil, iprodione, or propiconazole resulted in

**Table 2.** Significance level (P) for effects of plant growth regulators (PGRs) and fungicides on dollar spot in 1994

| Factor | Days to 5% disease | Disease severity post PGR treatment: 9 days | 10–33 days[x] | 35 days | 37 days | AUDPC[y] |
|---|---|---|---|---|---|---|
| Fungicide[z] | <0.01 | <0.01 | <0.01 | 0.05 | 0.03 | <0.01 |
| PGR | <0.01 | <0.01 | <0.01 | <0.01 | <0.01 | <0.01 |
| PGR × fungicide | 0.02 | NS | <0.01 | 0.03 | 0.03 | <0.01 |

[x] Significance level for each of 12 rating dates between 10 and 33 days after treatment.
[y] Area under disease progress curve.
[z] Fungicides were applied 4 days after PGRs.

**Table 3.** Effect of plant growth regulators on rates (*dy/dt*) of dollar spot epidemics in plots of creeping bentgrass

| Treatment | Rate (kg a.i./ha) | (*dy/dt*) early epidemic[w,x] 1993 | 1994 | (*dy/dt*) late epidemic[w,y] 1993 | 1994 |
|---|---|---|---|---|---|
| Paclobutrazol | 0.16 | 12.2 a[z] | 5.3 b | 0.012 a | 0.093 a |
| Fluprimidol | 0.33 | 7.5 b | 2.2 c | 0.010 a | 0.093 a |
| Trinexapac-ethyl | 0.19 | 10.5 a | 9.2 a | 0.009 a | 0.093 a |
| Nontreated control | – | 10.7 a | 10.7 a | 0.016 a | 0.091 a |

[w] Values of *dy/dt*, where $y$ = amount of disease and $t$ = time for early epidemics, were based on cumulative numbers of dollar spots per plot from 6 to 15 and from 9 to 20 days after treatment in 1993 and 1994, respectively. Values of *dy/dt* for late epidemics were based on visual estimates of percent disease per plot from 18 to 37 and from 22 to 37 days after treatment in 1993 and 1994, respectively.
[x] Rate parameters were determined by regressing nontransformed disease units against time.
[y] Rate parameters were determined by regressing transformed disease severity units against time. Transformations were based on monomolecular and exponential models in 1993 and 1994, respectively.
[z] Within a column, values followed by the same letter are not significantly different at $\alpha = 0.05$ according to Duncan's LSD.

**Table 4.** Effect of plant growth regulators on area under disease progress curves (AUDPC) for dollar spot in plots of creeping bentgrass

| Treatment | Rate (kg a.i./ha) | AUDPC – early epidemic[x] 1993 | 1994 | AUDPC – late epidemic[y] 1993 | 1994 |
|---|---|---|---|---|---|
| Paclobutrazol | 0.16 | 622.8 b[z] | 402.1 b | 335.1 bc | 213.8 b |
| Fluprimidol | 0.33 | 227.6 b | 215.4 b | 231.4 c | 134.1 b |
| Trinexapac-ethyl | 0.19 | 1,131.4 a | 761.4 a | 521.5 ab | 329.7 a |
| Nontreated control | – | 1,300.6 a | 952.6 a | 610.5 a | 359.4 a |

[x] Values were based on cumulative numbers of dollar spots per plot from 6 to 15 and from 9 to 20 days after treatment in 1993 and 1994, respectively.
[y] Values were based on visual estimates of percent disease per plot from 18 to 37 and from 22 to 37 days after treatment in 1993 and 1994, respectively.
[z] Within a column, values followed by the same letter are not significantly different at $\alpha = 0.05$ according to Duncan's LSD.

**Table 5.** Interaction of fungicides and plant growth regulators (PGRs) on elapsed time after treatment to reach 5% dollar spot severity in plots of creeping bentgrass in 1994

| Treatment | Days to 5% disease: Chlorothalonil | Iprodione | Propiconazole | Thiophanate-methyl |
|---|---|---|---|---|
| Paclobutrazol | 28.8 a[y] | 26.3 a | 31.5 a | 32.5 a |
| Fluprimidol | 28.8 a | 28.3 a | 32.5 a | 33.0 a |
| Trinexapac-ethyl | 26.3 ab | 22.8 b | 29.5 a | 32.0 a |
| No PGR[z] | 24.8 b | 19.5 c | 31.0 a | 32.0 a |

[y] Within a column, values followed by the same letter are not significantly different at $\alpha = 0.05$ according to Duncan's LSD.
[z] Fungicide treatment alone.

significantly lower values of AUDPC from 5 to 16 days after fungicide treatment compared to plots treated with fungicide alone (Table 7). Similar effects were detected 18 to 33 days after fungicide treatment in plots treated with flurprimidol plus chlorothalonil, iprodione, or propiconazole and in plots treated with paclobutrazol plus iprodione or propiconazole (Table 7). The PGRs had no significant effect on dollar spot in plots treated with thiophanate-methyl compared with plots treated with the fungicide alone.

In 1995, pretreatment with flurprimidol resulted in significantly lower values of AUDPC for early epidemics (5 to 13 days after fungicide treatment) in plots treated with each fungicide, except thiophanate-methyl, compared to plots treated with the fungicides alone (Table 8). Similar effects were observed in plots treated with paclobutrazol plus propiconazole compared to plots treated with the fungicide alone. Values of AUDPC calculated for late epidemics (17 to 37 days after fungicide treatments) were significantly lower for plots treated with each PGR plus propiconazole, but not with other fungicides, than for plots treated with fungicide alone (Table 8).

In 1993, foliar clipping weights were significantly higher from plots treated with each PGR 28 days after treatment, but not 14 or 42 days after treatment, compared to nontreated controls (Table 9). Canopy heights in 1994 were significantly suppressed by each PGR 10 and 18 days after

**Table 6.** Interaction of fungicides and plant growth regulators (PGRs) on elapsed time after treatment to reach 5% dollar spot severity in plots of creeping bentgrass in 1995

| | Days to 5% disease | | | |
|---|---|---|---|---|
| **Treatment** | **Chlorothalonil** | **Iprodione** | **Propiconazole** | **Thiophanate-methyl** |
| Paclobutrazol | 27.3 a[y] | 18.3 b | 23.8 ab | 19.3 ab |
| Fluprimidol | 22.3 a | 28.0 a | 31.0 a | 21.3 a |
| Trinexapac-ethyl | 22.5 a | 17.0 b | 20.0 b | 20.0 ab |
| No PGR[z] | 24.5 a | 17.0 b | 18.3 b | 17.0 b |

[y] Within a column, values followed by the same letter are not significantly different at $\alpha = 0.05$ according to Duncan's LSD.
[z] Fungicide treatment alone.

**Table 7.** Interactions of fungicides and plant growth regulators (PGRs) on area under disease progress curves (AUDPC) for dollar spot of creeping bentgrass in 1994

| | AUDPC – early epidemic[x] | | | | AUDPC – late epidemic[x] | | | |
|---|---|---|---|---|---|---|---|---|
| **Treatment[w]** | **Chlorothalonil** | **Iprodione** | **Propiconazole** | **Thiophanate-methyl** | **Chlorothalonil** | **Iprodione** | **Propiconazole** | **Thiophanate-methyl** |
| Paclobutrazol | 41.3 b[y] | 77.9 b | 6.1 b | 8.4 a | 149.6 ab | 153.2 bc | 65.9 b | 47.4 a |
| Fluprimidol | 41.8 b | 35.4 b | 3.5 b | 7.4 a | 99.3 b | 140.0 c | 37.5 b | 42.1 a |
| Trinexapac-ethyl | 37.1 b | 111.5 b | 4.1 b | 9.3 a | 169.0 a | 222.0 ab | 76.7 a | 56.8 a |
| No PGR[z] | 132.4 a | 364.8 a | 20.8 a | 15.1 a | 173.4 a | 245.8 a | 71.4 a | 48.9 a |

[w] The PGRs paclobutrazol, fluprimidol, and trinexapac-ethyl were applied 4 days prior to fungicides at rates of 0.16, 0.33, and 0.19 kg a.i./ha, respectively. The fungicides chlorothalonil, iprodione, propiconazole, and thiophanate-methyl were applied at 9.6, 3.1, 0.4, and 3.1 kg a.i./ha, respectively.
[x] Values for early epidemics were based on cumulative numbers of dollar spots per plot from 5 to 16 days after fungicide treatment. Values for late epidemics were based on visual estimates of percent disease per plot from 18 to 33 days after fungicide treatment.
[y] Within a column, values followed by the same letter are not significantly different at $\alpha = 0.05$ according to Duncan's LSD.
[z] Fungicide treatment alone.

**Table 8.** Interactions of fungicides and plant growth regulators (PGRs) on area under disease progress curves (AUDPC) for dollar spot of creeping bentgrass in 1995

| | AUDPC – early epidemic[x] | | | | AUDPC – late epidemic[x] | | | |
|---|---|---|---|---|---|---|---|---|
| **Treatment[w]** | **Chlorothalonil** | **Iprodione** | **Propiconazole** | **Thiophanate-methyl** | **Chlorothalonil** | **Iprodione** | **Propiconazole** | **Thiophanate-methyl** |
| Paclobutrazol | 19.8 a[y] | 39.0 a | 0.3 b | 13.5 a | 133.5 a | 192.7 a | 57.9 b | 158.7 a |
| Fluprimidol | 3.7 b | 12.7 b | 0.2 b | 7.9 a | 125.9 a | 169.8 a | 62.9 b | 162.8 a |
| Trinexapac-ethyl | 22.3 a | 41.3 a | 2.7 ab | 13.7 a | 147.0 a | 265.4 a | 87.8 b | 125.3 a |
| No PGR[z] | 28.0 a | 46.2 a | 13.2 a | 15.2 a | 144.0 a | 323.4 a | 134.4 a | 175.1 a |

[w] The PGRs paclobutrazol, fluprimidol, and trinexapac-ethyl were applied 4 days prior to fungicides at rates of 0.16, 0.33, and 0.19 kg a.i./ha, respectively. The fungicides chlorothalonil, iprodione, propiconazole, and thiophanate-methyl were applied at 9.6, 3.1, 0.4, and 3.1 kg a.i./ha, respectively.
[x] Values for early epidemics were based on cumulative numbers of dollar spots per plot from 5 to 13 days after fungicide treatment. Values for late epidemics were based on visual estimates of percent disease per plot from 17 to 37 days after fungicide treatment.
[y] Within a column, values followed by the same letter are not significantly different at $\alpha = 0.05$ according to Duncan's LSD.
[z] Fungicide treatment alone.

**Table 9.** Effect of plant growth regulators (PGRs) on foliar clipping weight and height of the foliar canopy of creeping bentgrass

| | | Clipping wt (g) on days after treatment[x] | | | Canopy height (mm) on days after treatment[y] | | |
|---|---|---|---|---|---|---|---|
| **Treatment** | **Rate (kg a.i./ha)** | **14** | **28** | **42** | **10** | **18** | **32** |
| Paclobutrazol | 0.16 | 10.8 a[z] | 10.4 a | 6.2 a | 7.1 b | 8.6 b | 12.8 a |
| Flurprimidol | 0.33 | 8.8 a | 10.3 a | 5.9 a | 7.1 b | 7.8 b | 12.8 a |
| Trinexapac-ethyl | 0.19 | 10.5 a | 9.7 a | 6.0 a | 7.4 b | 7.2 b | 14.8 a |
| Control | – | 10.5 a | 7.2 b | 5.4 a | 15.1 a | 13.2 a | 15.2 a |

[x] Data collected in 1993.
[y] Data collected in 1994.
[z] Within a column, values followed by the same letter are not significantly different at $\alpha = 0.05$ according to the Scott-Knott cluster analysis procedure.

treatment, but not 32 days after treatment, compared to nontreated controls.

## DISCUSSION

Our findings show that some gibberellin inhibitor PGRs inhibit growth of *S. homoeocarpa*, suppress dollar spot, and enhance the efficacy of dollar spot fungicides. These results are related to the chemistry of the materials in question. Paclobutrazol (a triazole) and flurprimidol (a pyrimidine) are chemically related to fungicides that inhibit demethylation of lanosterol or 24-methylene dihydrolanosterol in the fungal sterol biosynthesis pathway (5). Our results confirm previous findings (14,16), indicating that these PGRs are fungistatic in vitro. In plants, paclobutrazol and flurprimidol inhibit gibberellin biosynthesis by interfering with the oxidation of kaurene to kaurenoic acid in the isoprenoid pathway (5). Trinexapac-ethyl (a cyclohexadione) inhibits gibberellin later in the biosynthetic pathway by suppressing the conversion of $GA_{20}$ to $GA_1$ (1). To our knowledge, this compound has not been reported to be a fungistat. Each of the three PGRs has inhibited turfgrass shoot growth for up to 5 weeks (12).

Suppression of dollar spot by paclobutrazol and flurprimidol is probably a result of fungistatic activity, but the possibility of disease-suppressive environmental effects resulting from inhibition of shoot growth (e.g., changes in canopy temperature and duration of leaf wetness) cannot be ruled out. However, the fact that trinexapac-ethyl was significantly less fungistatic than paclobutrazol or flurprimidol in vitro, and only marginally disease suppressive, suggests that growth regulatory effects of PGRs are not as significant as fungistatic effects in limiting disease.

Fungicidal control of dollar spot was enhanced by pretreatment of turf with the PGRs tested. This was particularly evident with flurprimidol and probably results from the fungistatic effect of this material and the rate of application. More consistent effects of paclobutrazol and trinexapac-ethyl on fungicide efficacy may be detected at higher application rates. It is also possible that PGR × fungicide interactions were not simply a result of fungistasis. Growth regulation may have increased the efficacy of fungicides by limiting removal of systemic and nonsystemic material by mowing. Results of studies with stained or labeled fungicide residues, or the reversal of growth inhibition by gibberellic acid, may help to separate fungistatic from growth regulatory effects of PGRs on fungicide efficacy.

The reduced efficacy of propiconazole and thiophanate-methyl in 1995 (<19 days to reach 5% disease severity) compared to 1994 (>30 days to reach 5% disease) may have resulted from contamination of the turf plots by a fungicide-resistant strain of *S. homoeocarpa*. An isolate, cross-resistant to DMI and benzimidazole fungicides, was being studied in turfgrass near the PGR experiments. If contamination occurred, the enhancement of the efficacy of propiconazole by pretreatment with paclobutrazol or flurprimidol suggests that the development of DMI resistance in *S. homoeocarpa* is a quantitative response, with the existence of strains that vary in sensitivity to increases in concentrations of triazole or pyrimidine compounds (14). Given the report of isolation of DMI-resistant strains of *S. homoeocarpa* from golf course turf in Michigan and Ohio (10), the role that triazole or pyrimidine PGRs may play in the possible selection of such strains needs to be addressed.

Measurement of clipping weight in 1993 did not reflect the growth-suppressive effects of the PGRs on creeping bentgrass. In fact, clipping weights were higher for PGR-treated plots, which probably resulted from high levels of disease in nontreated plots and possibly an increase in tillering that may occur in PGR-treated grasses (19). Measurements of canopy height in 1994 showed that the PGRs significantly suppressed the growth of bentgrass for at least 18 days after treatment.

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