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Persistence of Triazole Growth Retardants on Stem Elongation of *Rhododendron* and *Kalmia*

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Abstract. Triazole growth retardant chemicals may inhibit stem elongation of woody ornamental species for several years after application. Potted plants of large-leaf *Rhododendron catawbiense* and *Kalmia latifolia* were treated with a single spray application of paclobutrazol or uniconazole in the 2nd year from propagation. They were transplanted into the field the next spring. The elongation of stems was measured in the year of application and in the next 2–4 years. Treatments with a wide range of doses were applied in 1991, 1992, or 1995. For all except the most dilute applications, stem elongation was retarded in the year after application. At the highest doses, stem growth was inhibited for 2 years after application. The results were fit to a model of growth regulator action which assumed that stem elongation was inversely related to the amount of growth regulator applied. For paclobutrazol, the dose per plant that inhibited stem elongation half as much as a saturating dose was tenfold that for uniconazole, about 0.5 and 0.05 mg, respectively. For both chemicals, the dose-response coefficient decreased exponentially with time after application, with an exponential time constant of about 2 year^{-1}. A dose of growth regulator which reduced stem elongation by half immediately after application would only inhibit 12% of stem elongation the next year. However, a tenfold greater dose would result in less than half the stem elongation of untreated plants in the next year.

Key Words. Growth retardants—*Kalmia*—Paclobutrazol—*Rhododendron*—Stem elongation—Uniconazole

A growth retardant chemical, in combination with a cold and day length-forcing treatment, can induce *Rhododendron* to flower a year after propagation, as first shown by Stuart (1961). The triazole growth regulators reduce stem elongation more effectively than the chemicals used previously (Davis et al. 1988). Two of these, paclobutrazol and uniconazole, promoted flowering of field-grown *Rhododendron* and *Kalmia* (Gent 1995a, Ranney et al. 1994, Wilkinson and Richards 1991), whereas other chemicals had inconsistent effects. However, paclobutrazol applied to *Rhododendron* also reduced stem elongation in the year after application (Ranney et al. 1994). A drench of 50 mg plant^{-1} essentially inhibited any growth of *Rhododendron* the next year (Wilkinson and Richards 1991). After application of paclobutrazol, stem elongation was reduced for a year in *Vitis* (Reynolds and Wardle 1990) and for 2 years in *Malus* (Williams 1984). A dose of 1–10 mg of uniconazole reduced the growth in dry weight of various other woody landscape species, when measured 100 days after application (Warren et al. 1991). The persistent inhibition of growth by triazole growth regulators could be a problem when woody ornamentals are transplanted into the landscape. A dose that promotes flowering of *Rhododendron* and *Kalmia* may severely inhibit stem elongation for several years and delay establishment of the plant in the landscape.

What factors cause the effect of triazole growth retardant to persist for several years in woody plants? Inhibition of stem elongation in herbaceous plants such as *Chrysanthemum, Poinsettia,* and annual bedding plants persists for only a few weeks after application of growth retardant. Stem elongation of *Chrysanthemum* gradually returned to control growth rates about 15 days after the application of ancymidol (Fisher et al. 1996) or daminozide or uniconazole (Tayama and Carver 1992). The duration of the growth-retarding effect was roughly proportional to the concentration of regulator applied initially (Fisher et al. 1996). These authors could not distinguish between a linear or exponential time course for the return to control growth rates. Using sequential harvests after application of daminozide, Dicks and Charles

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Edwards (1973) determined that the rate of stem elongation was related to the concentration of daminozide remaining in the stem. The initial concentration was proportional to the amount of daminozide applied, and the concentration in the stem decreased exponentially with time.

$$\text{Concentration (time)} = \text{concentration (0)} \cdot \exp(-K_t \cdot \text{time}) \quad (1)$$

Daminozide did not appear to be metabolized in *Chrysanthemum* (Dicks and Charles Edwards 1973); the decrease in the concentration of growth regulator in plant tissue appeared to be primarily the result of dilution by new biomass. This process can be rapid in herbaceous plants, where the biomass doubles every week or two. Growth of woody plants is generally slower and often occurs in discrete flushes rather than continuously. Thus, the dilution of growth regulators by new biomass would take longer in woody than in herbaceous plants. As much as 27 days after applying [^{14}C]paclobutrazol to *Malus*, 85% of the extractable ^{14}C was identical to the original compounds (Sterrett 1985), indicating that triazole growth regulators are metabolized slowly in plant tissue, Thus, the effect of growth regulators may persist in woody plants because of their slow growth rate.

Stem elongation decreases to a limiting value as the dose of growth retardant is increased. The inhibition of stem elongation was related to the concentration of daminozide in the stem of *Chrysanthemum* by the modified inverse relation shown in Equation 2 (Dicks and Charles Edwards 1973).

$$\text{Stem elongation} = \frac{\text{control stem elongation}}{1 + K_c \cdot \text{concentration}} \quad (2)$$

A similar but more flexible function, using a power of this inverse relation, accounted for the dependence of leaf elongation of *Cyperus* on the concentration of paclobutrazol (Kawabata and DeFrank 1994). These functions predict that the inhibition of stem elongation will be in proportion to the dose of growth retardant only when $K_c \cdot \text{concentration} < 1$. When $K_c \cdot \text{concentration} >> 1$, stem elongation is almost completely inhibited, and a further increase in the concentration of growth retardant would have little effect. A high dose of growth retardant would continue to inhibit stem elongation severely until the concentration in plant tissue was diluted into the range of linear response. Biomass would have to increase severalfold to dilute the concentration into this range. For plants with a slow growth rate, such as woody perennials, this dilution of growth retardant in plant tissue could take several years. Because paclobutrazol and uniconazole have much greater dose-response coefficients, K_c, than daminozide (Davis et al. 1988), these triazole growth regulators can easily be applied at doses exceeding the range for linear response of stem elongation. This may explain why the effects of uniconazole lasted longer than those of daminozide in *Chrysanthemum* (Tayama and Carver 1992). Eight weeks after treatment, the height of bedding plants was reduced by high concentrations but not by low concentrations of uniconazole, whereas daminozide had no effect (Keever and Foster 1991). *Rhododendron* and *Kalmia* respond to triazole growth regulators at doses on the order of 1 mg plant^{-1} (Gent 1995a). Consequently, it could take several doublings in size to grow out of the effect of triazole growth regulators applied at a dose of 10–100 mg plant^{-1}. Because of their slow growth, it could take years for these plants to grow out of effects of such a dose of triazole growth regulators.

This report describes the time course of inhibition of stem elongation of *Rhododendron* and *Kalmia* for 2–4 years after spray application of paclobutrazol and uniconazole. The data were fit to a modified inverse relation, in which the dose response decreased exponentially with time, to derive the initial dose response and the time constant for return of stem elongation to the rates of growth seen in untreated plants.

Materials and Methods

Plant Material and Growth Conditions

The large leaf *Rhododendron catawbiense* Michx. cultivars Boursault and Roseum Elegans and the *Kalmia latifolia* L. cultivars Carousel and Yankee Doodle were used in this study. All plants were propagated and potted in 8-liter pots at Prides Corner Farm, Lebanon, CT, a commercial nursery. The *Rhododendron* were grown in a mix of 32:32:32:5 v/v hardwood bark:softwood bark:peat:sand. The *Kalmia* were grown in a mix of 50:33:17 v/v peat:hardwood bark:sand. In March of the year of treatment, each pot was top dressed with an Osmocote 9-month timed release formulation of 17:3:8 w/w N:P:K plus minor elements, at a rate of 36 g for the *Rhododendron* and 24 g for the *Kalmia.* During the growing season, plants were spaced two pot diameters apart in full sun and watered at regular intervals. Plants were protected over the winter in hoop houses that were covered with white polyethylene film in late October and uncovered in late March.

Plants were treated and grown at Lockwood Farm, Hamden, CT, the experimental farm of the Connecticut Agricultural Experiment Station. In the year after application of growth retardant, the plants were transplanted in the field. A plot was plowed, and 10:4:8 N:P:K fertilizer and powered sulfur were incorporated to give high fertility and adjust the pH to 5.0. Plants were set in rows with 0.6 m between plants in the row and 1 m between rows. The soil surface was covered with a layer of wood chips. Insecticides, fungicides, and herbicides were applied according to normal production practices.

Application of Growth Regulators

The growth regulators used were paclobutrazol (2*RS*,3*RS*-1-[4-chlorophenyl]-4,4-dimethyl-2-[1,2,4-triazol-1-yl]-pentan-3-ol in the Bonzi formulation (Uniroyal Chemical Co., Naugatuck, CT), and uni-

conazole (*E*-1-[4-chlorophenyl]-4,4-dimethyl-2-[1,2,4-triazol-1-yl]-1-penten-3-ol in the Sumagic formulation (Valent Chemical Co., Walnut Creek, CA).

The plants were treated in the 2nd year after propagation, except the *Kalmia* Carousel treated in April 1992 were in the 3rd year. Each plant received just one dose of growth retardant. In each year, a batch of solution was applied in its entirety to a group of plants. The solution was applied to leaves and stems as a timed, directed spray, with repeated applications, to equalize the volume applied to each plant and to reduce runoff to a minimum. Six or more plants of each cultivar were not sprayed, to serve as controls.

Growth retardant was applied in 1991, 1992, and 1995. In 1991, the doses per plant were 5 or 20 mg of paclobutrazol doses per plant, applied as 0.2 liter of solution with concentrations of 25 or 100 mg liter^{-1} on July 18, 1991. In 1992, the treatments were solutions of paclobutrazol applied at concentrations of 4, 10, and 30 mg liter^{-1} or solutions of uniconazole applied at 1.5, 4, and 12 mg liter^{-1}. Each plant was sprayed on one of three dates; April 23 before the first flush of growth in the spring; June 19 before the second flush of growth; or August 25, 1992 before the third flush of growth. A volume of 0.05 liter was applied per plant on April 23, resulting in doses per plant of 0.2, 0.5, and 1.5 mg of paclobutrazol or 0.075, 0.2, or 0.6 mg of uniconazole. A volume of 0.1 liter was applied on later dates, resulting in doses per plant of 0.4, 1.0, and 3.0 mg of paclobutrazol or 0.15, 0.4, or 1.2 mg of uniconazole. In 1995, each plant was sprayed with a volume of 0.05 liter on June 19, after the first flush of growth. The doses per plant were 0.25, 0.5, 1.0, 2.5, 5.0, and 10.0 mg of paclobutrazol or 0.05, 0.1, 0.25, 0.5, 1.0, and 2.5 mg of uniconazole.

The experiments in 1991 and 1992 were randomized block designs. In 1991, the main plots were cultivar, and subplots were the doses applied. In 1992, the main plots were cultivar, subplots were the date of application, and sub-subplots were the doses applied. In the years after treatment in 1992, rows of each cultivar were grouped by dose and date of application. These plots were located randomly in the field. The experiment in 1995 was a completely randomized design, with cultivars as main plots and treated plants located randomly within these plots. In the year after application, the cultivars were planted in alternating rows, and plants with different treatments were located randomly in these rows.

Measurements

In April, three to five stems on each plant were marked with white paint just below the terminal bud. In October, the length of the annual growth was measured for three stems of each plant. Typically, the growth was measured for the longest leader on each of three different branches resulting from pruning in the first year of propagation. If there were only two branches, a side shoot was measured. This procedure was repeated in each year after application of growth regulator.

Model

The length of stem elongation in each year was related by the following model to the amount of growth regulator applied and year after application:

$$\text{Growth (amount, year)} = \text{initial growth} + \frac{\text{growth (0,year)}}{1 + K_c \cdot \text{amount} \cdot \exp(-K_t \cdot \text{year})} \qquad (3)$$

where K_c, in units of mg^{-1} plant, was the dose-response coefficient to growth regulator in the year of application, and K_t, in units of year^{-1}, was the coefficient for the exponential decrease in the effect of the growth regulator with time. Growth(0,year) corresponded to the stem elongation of the control or untreated plants in each year. In the year of application, year = 0, some initial growth was not affected by growth regulator, and this was added as a constant to the model. In the years after application, regression indicated that the constant corresponding to initial growth was not significantly different than zero.

Analysis

The data were fit to Equation 3 using a nonlinear regression routine in SYSTAT (Version 6, SPSS Inc., Chicago). All regressions used equal weights for all data, included the unsprayed controls, and the three stem lengths for each plant were treated as independent measures. Regressions were done separately for each year of application, cultivar, and chemical. First, the data for each year were fit using $K_t = 0$ and varying the parameters, *initial growth*, *growth*(*year*,*0*), and K_c, to determine the significance of dose response in each year. Then the regression was repeated with fixed values for the parameters, *initial growth* and *growth*(*year*,*0*), to determine the 95% confidence interval for K_c. Finally the data for the year of application and the following years were fit simultaneously to define K_t and the 95% confidence interval for the regression coefficient. When regression could only define either an upper or lower limit to the confidence interval, SYSTAT reported unreasonably wide confidence limits. Regression coefficients were also determined by minimizing the sum of squares of deviations using the SOLVER algorithm in Microsoft EXCEL (Version 7, Microsoft Corp., Redmond, WA). The upper or lower bounds for the regression coefficients were determined separately, by varying the coefficients from the optimum values until the *F* statistic for regression decreased by 3.

Results

Treatments in 1991

After the application of paclobutrazol at 5 or 20 mg plant^{-1}, stem elongation of all four cultivars was inhibited more in 1992 than in 1991, the year of application. In 1991 stem elongation was similar with doses of 5 and 20 mg of paclobutrazol. In 1992, stem elongation of *Rhododendron* Roseum Elegans was inhibited more by a dose of 20 than 5 mg plant^{-1} (Fig. 1). In 1992, 5 mg of paclobutrazol inhibited stem elongation of *Kalmia* Yankee Doodle more than that of *Rhododendron* (Fig. 2). When stem elongation was analyzed separately in each year, the dose-response coefficient decreased from year to year. Because stem elongation in 1991 was similar with doses of 5 and 20 mg of paclobutrazol, no upper bound could be determined for the response coefficient. In 1992, the upper bounds for the dose-response coefficients were less than 0.6 mg^{-1} plant, except for *Kalmia* Yankee Doodle, 1.0 mg^{-1} plant. In 1993, there was a significant dose response only for *Rhododendron* Roseum Elegans and *Kalmia* Yankee Doodle, with dose response coefficients of <0.08 and <0.17 mg^{-1} plant, respectively. Stem elongation of *Kalmia* Yankee Doodle was inhibited more severely than that of other cultivars

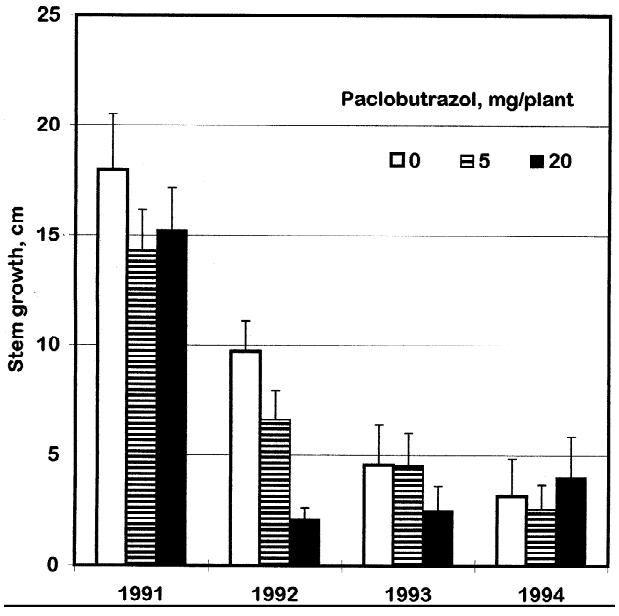

Fig. 1. Stem elongation of *Rhododendron* Roseum Elegans in 1991 and in the subsequent years after application of paclobutrazol in 1991. *Shading* indicates treatments in 1991, and *I bars* are the least significant difference within years.

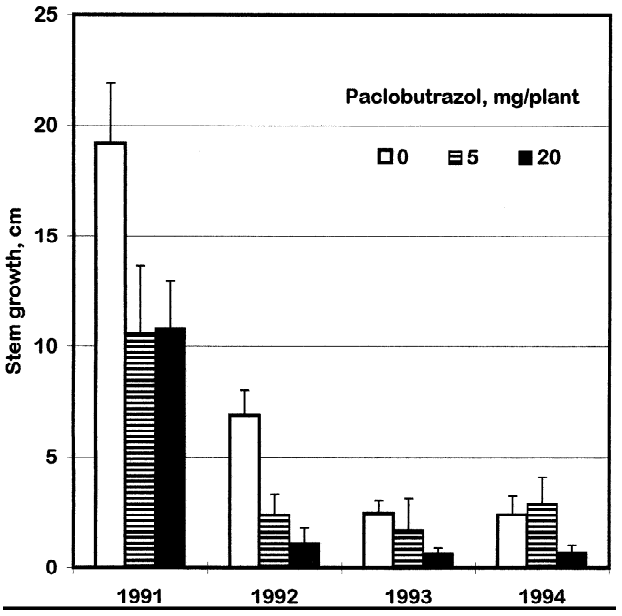

Fig. 2. Stem elongation of *Kalmia* Yankee Doodle in 1991 and in the subsequent years after application of paclobutrazol in 1991. *Shading* indicates treatments in 1991, and *I bars* are the least significant difference within years.

in the years after application. Stem elongation was inhibited 3 years after application only for this cultivar treated with a dose of 20 mg plant^{-1}. This was the only combination of treatment and cultivar which resulted in smaller leaves than the controls. The experiment illustrated that triazole growth regulators could inhibit stem elongation for at least 3 years after a spray application.

Treatments in 1992

The initial growth of stems, that amount of stem elongation which was not inhibited after application of growth regulators, depended on the date of application in 1992. These results have been reported previously (Gent 1995a). Across application dates, a single dose-response coefficient was sufficient to predict the response of that part of stem elongation which was sensitive to growth retardant. These coefficients are presented in Table 1. Nonlinear regression indicated that half the maximum inhibition of stem elongation in 1992 was achieved with a dose per plant of less than 0.7 mg of paclobutrazol or 0.04 mg of uniconazole, except for *Kalmia* Carousel. For all cultivars and for both chemicals, there was a significant response of stem elongation in 1993 to application of growth regulator in 1992. Stem elongation was inhibited more in the year after application because growth was inhibited for the entire year. There was no significant initial growth, as determined by nonlinear regression. Half the maximum inhibition of stem elongation in 1993 was achieved with a dose per plant of less than 5.0 mg of paclobutrazol or 0.4 mg of uniconazole, with the exception of *Kalmia* Carousel. The cultivars differed in response. The *Kalmia* Carousel were 3 years after propagation when treated in April 1992, and these larger plants had the lowest dose response. In contrast, *Kalmia* Yankee Doodle was the cultivar most affected by both chemicals, and it grew very little in the year after application, even at low doses.

Data for 3 years of stem elongation were included in regression to determine the time course of the dose-response coefficient. The coefficient for the exponential decrease with time varied from 1.3 to 2.7 year^{-1} (Table 1), corresponding to a 4- to 15-fold decrease in the dose-response coefficient per year. In general, the effect of growth retardant persisted for a shorter time in *Rhododendron* than in *Kalmia.* The initial dose-response coefficients for uniconazole were 4–20 times those for paclobutrazol, but the exponential decrease with time in the response coefficient was similar for the two chemicals.

Treatments in 1995

The modified inverse response of stem elongation to concentration of growth regulator was seen most clearly in the response of stem elongation to six doses over a 40- or 50-fold range applied in 1995. The responses to uniconazole and the predicted response based on fitting the data to Equation 3 are shown for *Rhododendron* Bour-

Table 1. Coefficients for predicting the response to paclobutrazol or uniconazole of stem elongation of *Rhododendron* and *Kalmia* in 1992 through 1994, after spray application at one of three concentrations in April, June, or August 1992.

	Regression coefficients				
	K_c (mg^{-1} plant)				
Chemical and cultivar	1992	1993	1994	all years	K_t (year^{-1})
Paclobutrazol					
Rhododendron Boursault	1.5 ± 0.5***[a]	0.20 ± 0.04***	N.S.	1.5 ± 0.4***	2.0 ± 0.3
R. Roseum Elegans	3.5 ± 0.4***	0.36 ± 0.12***	N.S.	3.6 ± 1.0**	2.3 ± 0.4
Kalmia Carousel	0.50 ± 0.22**	0.14 ± 0.08**	N.S.	0.50 ± 0.14***	1.3 ± 0.7
K. Yankee Doodle	6.0 ± 2.0***	1.7 ± 0.4***	N.S.	6.3 ± 1.4***	1.5 ± 0.3
Uniconazole					
Rhododendron Boursault	21.5 ± 9.8*	3.1 ± 0.4***	0.11 ± 0.18*	30.1 ± 13.0***	2.3 ± 0.4
R. Roseum Elegans	29.2 ± 13.8***	2.6 ± 0.7***	N.S.	35.8 ± 18.0**	2.7 ± 0.5
Kalmia Carousel	2.2 ± 0.9**	0.46 ± 0.23***	N.S.	2.3 ± 0.6**	1.7 ± 0.8
K. Yankee Doodle	35.2 ± 11.5***	5.0 ± 1.2***	0.46 ± 0.34**	36.8 ± 9.9**	2.1 ± 0.3

[a] N.S., *, **, ***, not significant or significant at $p < 0.05$, $p < 0.01$, $p < 0.001$, respectively, as determined by nonlinear regression. Range indicates Wald confidence interval at $p < 0.05$.

sault and *Kalmia* Carousel, in Figs. 3 and 4, respectively. A dose of about 0.12 plant^{-1} mg of uniconazole gave half the maximum inhibition of stem elongation of *Rhododendron* Boursault in the year of application (Table 2). However, only about 5 cm of the total of 15 cm of stem elongation in 1995 was sensitive to application of growth regulator (Fig. 3). A modified inverse response to the dose of growth regulator was also seen in the year after application, but 3.0 mg plant^{-1} of uniconazole was required to reduce stem elongation to half that of the control.

In 1995, *Kalmia* Carousel was more responsive to paclobutrazol and uniconazole than was *Rhododendron* Boursault. A dose of 0.07 plant^{-1} mg of uniconazole gave half the maximum inhibition of stem elongation of *Kalmia* Carousel, and all but 4 cm of the total of 19 cm of stem elongation in 1995 was sensitive to application of growth regulator (Fig. 4). In 1996, about 0.6 mg plant^{-1} of uniconazole was required to reduce stem elongation to half that of the control.

For both cultivars, the dose-response coefficients for paclobutrazol were much less than for uniconazole. The effect of paclobutrazol on *Rhododendron* Boursault was not significant in the year after application.

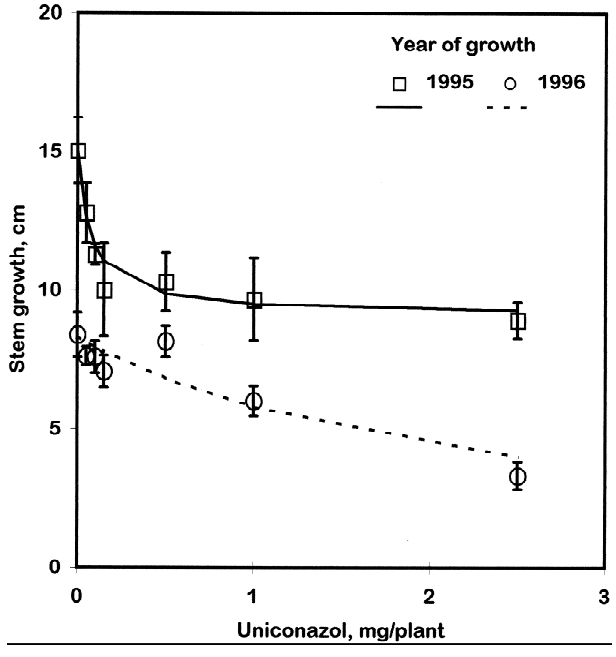

Fig. 3. Stem elongation of *Rhododendron* Boursault in 1995 and 1996 after application of uniconazole in 1995. *Symbols* indicate the mean, *I bars* indicate the standard error, and the *curves* represent the best fit from Equation 3.

Discussion

The time course of growth regulator action on *Rhododendron* and *Kalmia,* as well as the dose dependence in each year, was predicted by the model (Equation 3). This model would likely predict the extended period of inhibition of stem elongation reported elsewhere for *Vitis* (Reynolds and Wardle 1990) and *Rhododendron* (Wilkinson and Richards 1991). The dose-response coefficients, relating the amount of growth regulator to the inhibition of stem elongation, varied among cultivars and among the experiments reported here. The dose-response coefficients were assumed to decrease exponentially with time. The coefficient for this decrease with time, about 2 year^{-1}, was fairly consistent across experiments.

A time constant of 2 year^{-1} would reduce the dose-response coefficient by a factor of 7 in the year after application compared with that in the year of application. A dose of growth regulator which inhibited stem elongation to half that of untreated plants in the year of application would only inhibit 12% of the stem elonga-

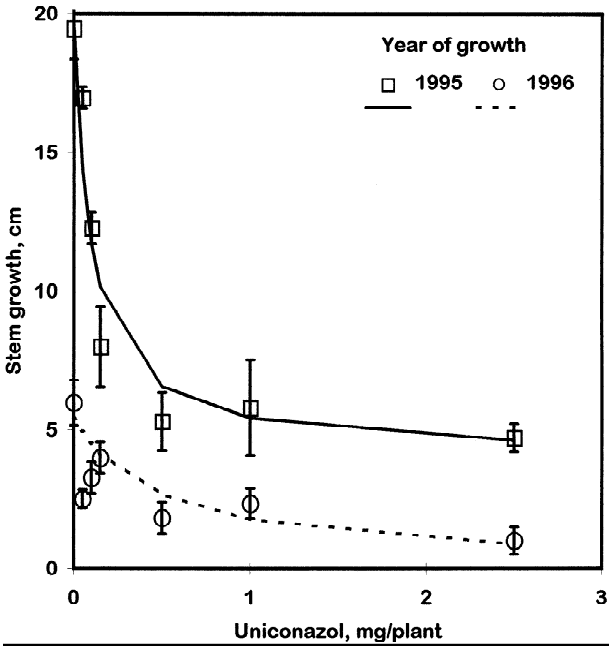

Fig. 4. Stem elongation of *Kalmia* Carousel in 1995 and 1996 after application of uniconazole in 1995. *Symbols* indicate the mean, *I bars* indicate the standard error, and the *curves* represent the best fit from Equation 3.

tion in the next year. Thus, the effect of growth retardant would persist for less than a year if it were applied in a dose small enough to result in a proportional response of stem elongation. However, if the dose were tenfold greater than that required to reduce stem elongation to half that of controls, the stem elongation in the next year would still be less than half that of untreated plants. This behavior was nearly always seen in the present experiments after application of a large dose of paclobutrazol or uniconazole. A time constant of 2 year^{-1} would predict a 55-fold decrease in the dose-response coefficient by 2 years after application. Few significant effects were seen in the 2nd year after application, except for high doses applied to the most sensitive cultivars.

Even for untreated plants, stem elongation was less in the year after application, when plants grew in the field, than in the year of application, when they grew in pots. In part, the difference in growth was the result of greater water stress in field-grown than potted plants. Potted plants were watered regularly, whereas plants in the field were watered only in periods of drought. This stress may have slowed the growth in biomass and dilution of growth retardant and prolonged the inhibition of stem elongation. On the other hand, drought stress would decrease the sensitivity to growth retardant and the dose-response coefficients. Thus, the change in growth conditions did not necessarily emphasize the persistence of effects of triazole growth retardants. In part, growth differed between the year of application and the following years because flowering affected vegetative growth. Vegetative shoots often failed to initiate at stem apices that flowered. All plants were strictly vegetative when growth retardants were applied, but in the subsequent years, many stems terminated in a flower raceme. Often, there was no new vegetative growth on one or two of the three stems selected for measurement. Thus, the average stem elongation was reduced by stems that did not grow at all.

There was variation in the dose-response coefficient in the year of application. The plants were most responsive to paclobutrazol in 1991, when the volume of solution applied was greater than in other years. The plants were least responsive in 1995, when the smallest volume of solution was used. Thus, efficacy of the chemicals increased with the volume of spray applied, as noted by Barrett et al. (1994) for uniconazole applied to *Chrysanthemum.* It is likely that the more intensive response obtained with an application of 0.2 liter plant^{-1} was caused by increased wetting of basal shoots and increased dripping of solution onto the soil. Because these compounds are translocated strictly acropetally (Reed et al. 1989; Richardson and Quinlan 1986), application to basal stems and the root zone is likely to be more efficacious than application to leaves.

The responses to paclobutrazol and uniconazole were qualitatively similar, but uniconazole was effective at lower doses than paclobutrazol. Based on the regression analysis of the *Rhododendron* treated in 1992 and 1995, the efficacy of uniconazole was 10- to 20-fold that of paclobutrazol. *Kalmia* differed less in response to the two chemicals. For *Kalmia,* the efficacy of uniconazole was four- to eightfold that of paclobutrazol.

Kalmia Yankee Doodle treated with 20 mg of paclobutrazol in 1991 took 4 years to grow out of the effect on stem elongation. This was the only case where the size of leaves was reduced by the treatment, and flower bud formation was prevented. Biomass accumulation, as determined by visual observation, was much less for these plants than for those given other treatments. In all other cases, the primary effect of the treatment with paclobutrazol or uniconazole was inhibition of stem elongation. Growth parameters other than stem elongation were not affected by triazole growth regulators. Leaf size, stem diameter, and flower size and color were similar for treated and control plants. Paclobutrazol also had no phytotoxic symptoms when applied to florist *Azalea,* in contrast to other growth retardant chemicals (Whealy et al. 1988).

Whereas stem elongation showed a saturating response to dose of growth retardant, the number of flowers/plant generally showed a linear response. After application in April or June 1992, stem elongation showed a quadratic, saturating response to paclobutrazol or uniconazole in 8 of 16 comparisons, whereas the number of

Table 2. Coefficients for predicting the response to paclobutrazol or uniconazole of stem elongation of *Rhododendron* and *Kalmia* in 1995 and 1996, after spray application at one of six concentrations in June 1995.

	Regression coefficients			
	K_c (mg^{-1} plant)			
Chemical and cultivar	1995	1996	both years	K_t (year^{-1})
Paclobutrazol				
Rhododendron Boursault	0.44 ± 0.35**[a]	N.S.	0.44 ± 0.36**	>2.1
Kalmia Carousel	1.7 ± 0.9***	1.2 ± 0.7***	1.7 ± 0.7***	<1.3
Uniconazole				
Rhododendron Boursault	8.0 ± 6.3**	0.32 ± 0.13***	8.0 ± 5.1**	3.2 ± 0.8
Kalmia Carousel	14.4 ± 8.3***	1.8 ± 1.4**	14.4 ± 7.0***	2.1 ± 1.1

[a] N.S., *, **, ***, not significant or significant at $p < 0.05$, $p < 0.01$, $p < 0.001$, respectively, as determined by nonlinear regression. Range indicates Wald confidence interval at $p < 0.05$. An inequality indicates the confidence interval is bounded in only one direction.

flowers/plant showed a quadratic response in only 3 of 16 comparisons (Gent 1995a). Thus, nearly complete inhibition of stem elongation was achieved more easily than complete expression of flowering. After application in 1995, the number of flowers was not increased significantly for *Rhododendron* Boursault, and flowering of *Kalmia* Carousel was increased only with high doses of paclobutrazol and uniconazole (Gent 1995b). The lack of response in 1995 was caused, in part, by flowering of untreated controls, which was not seen in plants treated in 1991 and 1992. In 1992, some flowering was induced when the dose of growth retardant was less than or equal to half the saturating dose for inhibition of stem elongation. This dose would not inhibit stem elongation severely in the subsequent year. Thus, there is a dose of paclobutrazol or uniconazole which can induce flowering when untreated plants would normally not flower but which will not severely inhibit stem elongation the next year. Based on the model presented here, the doses per plant for *Rhododendron* and *Kalmia* in the 2nd year from propagation would be less than 0.5 mg of paclobutrazol or 0.05 mg of uniconazole when applied as a spray application to wet the plant completely.

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