J. Amer. Soc. Hort. Sci. 121(5):861–868. 1996.

Modeling the Stem Elongation Response of Poinsettia to Chlormequat

P.R. Fisher[1] and R.D. Heins
Department of Horticulture, Michigan State University, East Lansing, Michigan 48824

J.H. Lieth
Department of Environmental Horticulture, University of California, Davis, California 95616

Additional index words. Euphorbia pulcherrima, plant growth, height control, dose response, growth retardant, plant growth regulator, three-phase function, Richards function, simulation

Abstract. **Stem elongation response to a single foliar application of the growth retardant chlormequat chloride [(2-chloroethyl) trimethylammonium chloride] for poinsettia (*Euphorbia pulcherrima* Klotz.) was quantified. Growth retardant applications did not affect final leaf count or timing of visible bud, first bract color, or anthesis. There was a statistically significant effect of growth retardant concentration on stem elongation, with a range from 289 ± 15 mm (mean ± 95% confidence intervals) for the control plants to 236 ± 17 mm at 4000 ppm. The growth-retarding effect during the first day after the application was not significantly different between 500 and 4000 ppm, and concentration primarily affected the duration of growth-retarding activity. A dose response function was incorporated into a three-phase mathematical function of stem elongation of single-stem poinsettia to predict elongation of treated and untreated plants. The model was calibrated using a data set from plants receiving 0, 500, 1000, 1500, 2000, 3000, and 4000 ppm, with a resulting R^2 of 0.99. Validation of the dose response model against an independent data set resulted in an r^2 of 0.99, and predicted final stem length was within 12 mm of observed final length.**

A three-phase mathematical function has been used to model shoot elongation of single-stem poinsettia (*Euphorbia pulcherrima* Klotz.) (Fisher et al., 1996). In calibrating the model, aside from nyctoperiod and minor greenhouse temperature fluctuations, environmental factors were constant throughout the experiment. For a stem elongation model to be useful for simulation and practical application, however, it is necessary to incorporate the effect of perturbations such as growth retardant applications and temperature effects. Growth retardant chemicals are used widely in the commercial production of poinsettia and other flowering potted plants for height control (Ecke et al., 1990). A simulation model may be a useful horticultural tool because there is increasing pressure to minimize the use of growth retardants (Ludolph, 1992) and maximize their efficacy.

Although many researchers have examined the impact of growth retardant applications on poinsettia final height and flowering (for example, Holcomb et al., 1992; Holcomb and Rose, 1992; Larson, 1967; McDaniel, 1986; McDaniel and Wilson, 1990), published data are not suitable for predicting short term changes in stem elongation rate. These data provide a basis for determining the effects of growth retardant type and concentration on final plant height. It is difficult to generalize these results, however, because growth retardant efficacy can be affected by a range of environmental conditions, including soil media composition (Barrett, 1982), timing (Gilbertz, 1992), nutrient status, application technique and frequency, chemical type, concentration, and temperature (Larson, 1967).

The objectives of this project were to quantify the dose response of poinsettia to a single foliar application of chlormequat chloride growth retardant [(2-chloroethyl) trimethylammonium chloride] and incorporate the dose response into a three-phase model of stem elongation ($f_{3\text{-}phase}$, Fisher et al., 1996). Separate data sets were collected for model calibration and validation. A methodology developed by Lieth and Reynolds (1986) for modifying the Richards function (Richards, 1959) to incorporate the effect of daminozide applications on chrysanthemum [*Dendranthema grandiflora* (Ramat.) Kitamura] was adapted to model a different chemical (chlormequat), species (poinsettia), and base function ($f_{3\text{-}phase}$).

Materials and Methods

Modeling an environmental perturbation. In the Lieth and Reynolds (1986) approach, the Richards equation (Eq. [1]),

$$\frac{dH_u}{dt} = \frac{kH_u(A^n - H_u^n)}{(nA^n)} \quad [1]$$

with the solution (Eq. [2]), commonly termed the Richards function,

$$H_u = \frac{H_o A}{\sqrt[n]{H_0^n + (A^n - H_0^n)e^{-kt}}} \quad [2]$$

was fitted to data from plants grown in constant conditions. In the Richards function, t represents time, A defines the upper asymptote of untreated plant height H_u, and parameters H_0, n, and k determine the lower asymptote and shape of the growth curve with respect to A (Table 1). The estimated values of A, H_0, n, and k describe the asymptotic growth characteristics of H_u in the absence of environmental change.

The Richards function was modified by Lieth and Reynolds (1986) to simulate the response to an environmental perturbation. The response function was termed $g(t)$, and the modified Richards equation was written as

$$\frac{dH}{dt} = \frac{dH_u}{dt} g(t) \quad [3]$$

where $g(t)$ is a function that has a value ≥0. If $g(t) = 0$, there is no growth, a $g(t)$ of 1 means that the treated plant grows at the same

Received for publication 7 Aug. 1995. Accepted for publication 7 Mar. 1996. We thank University Outreach at Michigan State Univ. for providing funding for this project. The cost of publishing this paper was defrayed in part by the payment of page charges. Under postal regulations, this paper therefore must be hereby marked *advertisement* solely to indicate this fact.

[1]Current address: Dept of Environmental Horticulture, Univ. of California, Davis, CA 95616.

rate as a control plant, and a $g(t)$ <1 or >1 would indicate that the treated plant is growing slower or faster than the control, respectively.

Larsen and Lieth (1993) modeled the dose response to daminozide by formulating two alternative $g(t)$ functions (Fig. 1, Eqs. [4] and [5]). A foliar spray application at time t_{ev} was assumed to immediately reach a maximum effect, or amplitude, that declined over time. Before the spray application ($t < t_{ev}$), $g(t)$ had a value of 1 (no effect). After t_{ev}, $g(t)$ was a function that had a value less than 1. When a linear decay function ($g_L(t)$) was used, the chemical was assumed to have an immediate maximum effect (M_L) that declined over a period termed persistence (P, days) until g(t) = 1 at time t_{rec}. The linear model is represented below (Eq. [4]), and is represented graphically (Fig. 1a):

$$g_L(t) = \begin{cases} 1 & \text{...for } t \le t_{ev} \\ M_L + [1 - M_L)/P](t - t_{ev}) & \text{...for } t_{ev} < t \le t_{rec} \\ 1 & \text{for } t \quad < t \end{cases} \qquad [4]$$

In the exponential decay curve ($g_E(t)$, Eq. [5] and Fig. 1b), M_E referred to the amplitude of the response and C_E represented the rate of decay.

$$g_E(t) = \begin{cases} 1 & \text{..for } t \le t_{ev} \\ 1 - (1 - M_E)e^{[-C_E(t - t_{ev})]} & \text{..for } t > t_{ev} \end{cases} \qquad [5]$$

Larsen and Lieth (1993) obtained estimates of A, n, H_0 and k in the Richards function by fitting Eq. [2] to data from untreated plants. The modified Richards function (Eq. [3]) was then fitted to data from the growth retardant-treated plants, requiring only the estimation of the $g(t)$ parameters, because the parameter estimates of A, n, H_0, and k were fixed based on the untreated plants.

Three-phase methodology. The $f_{3\text{-}phase}$ poinsettia stem elongation model for modeling elongation under constant temperature conditions and variable flower initiation dates is described in detail by Fisher et al. (1996). Equations were chosen (Eq. [6]) that provided the empirical qualities desired to develop a sigmoid curve. Increasing growth rate during the initial lag phase was

Table 1. List of abbreviations and parameters.

Symbol	Description	Units
α	Initial stem length in the lag phase of $f_{3\text{-}phase}$	mm
β	Rate constant in lag phase of $f_{3\text{-}phase}$	per day
γ	Gradient of linear phase of $f_{3\text{-}phase}$	mm·d^{-1}
δ	Asymptotic potential growth during plateau phase of $f_{3\text{-}phase}$	mm
Δt_{SV}	Time from t_S to t_V	days
Δt_{VP}	Time from t_V to t_P	days
a	Parameter used to estimate duration or persistence of $g(t)$	days
A	Asymptotic height parameter in the Richards function	mm
b	Parameter used to estimate duration or persistence of $g(t)$	days/ppm
C_E	Recovery parameter in exponential $g_E(t)$ dose response function	per day
$Conc$	Concentration of chlormequat	ppm
D	Duration parameter in exponential $g_E(t)$ dose response function (inverse of C_E)	days
DIF	Average day minus average night temperature	°C
$f_{3\text{-}phase}$	Three-phase stem elongation model	
f_{LAG}	Function during lag phase of $f_{3\text{-}phase}$	
f_{LIN}	Function during linear phase of $f_{3\text{-}phase}$	
f_{PLA}	Function during plateau phase of $f_{3\text{-}phase}$	
$g_E(t)$	Exponential dose response function	
$g_L(t)$	Linear dose response function	
G_E	Complete stem elongation model incorporating $f_{3\text{-}phase}$ and $g_E(t)$	
G_L	Complete stem elongation model incorporating $f_{3\text{-}phase}$ and $g_L(t)$	
$g(t)$	Environmental perturbation function	
H	Predicted stem length	mm
H_0	Initial stem length parameter in the Richards function	mm
H_f	Predicted final stem length in $f_{3\text{-}phase}$	mm
H_u	Predicted stem length of untreated plant	mm
k	Rate parameter in the Richards function	per day
k_{PLA}	Rate parameter in plateau phase	per day
M_E	Initial amplitude effect of exponential $g_E(t)$ dose response function	
M_L	Initial amplitude effect of linear $g_L(t)$ dose response function	
n	Point of inflection in the Richards function	
P	Persistence parameter in linear $g_L(t)$ dose response function	days
t	Time variable	days
t_0	Time of transplanting	days
t_L	Time when lag phase ends and linear phase begins in $f_{3\text{-}phase}$	days
t_P	Time when linear phase ends and plateau phase begins in $f_{3\text{-}phase}$	days
t_{ev}	Time of growth retardant application	days
t_{rec}	Time at which plant recovers from linear $g_L(t)$ dose response	days
t_S	Time when short days (flower-initiating nyctoperiods) begin	days
t_V	Time when visible bud occurs	days

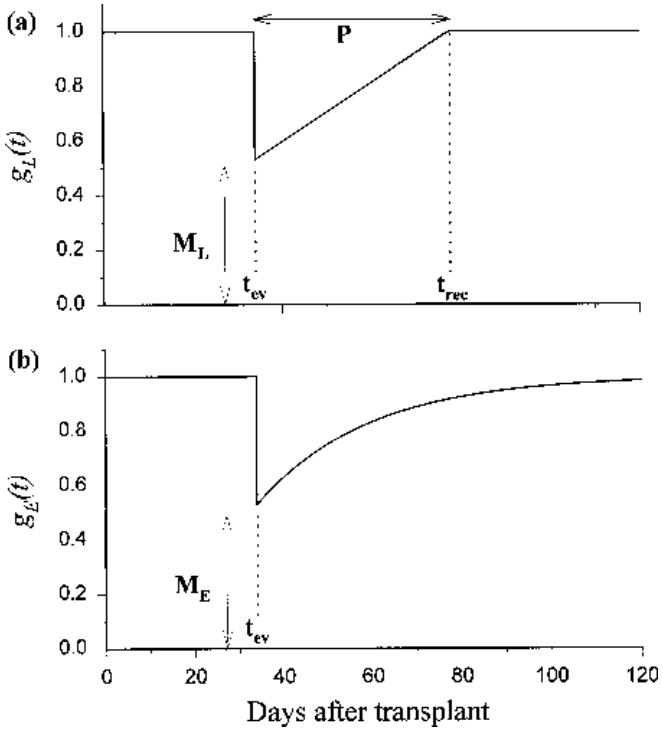

Fig. 1. Alternative *g(t)* dose response models proposed by Larsen and Lieth (1993), where *g(t)* represents the elongation rate of the treated plants relative to an untreated control. (**a**) a linear $g_L(t)$ model where the initial effect of a growth retardant application at time t_{ev} is represented by M_L, and the declining growth retardant effect persists for *P* days. (**b**) an exponential dose response model with declining growth retardant effect after an initial amplitude of M_E.

described with an exponential function ($f_{LAG}(t)$), in which α (mm) is the observed starting length and β (per day) is a rate constant. Constant growth rate during the linear phase was described with a linear function ($f_{LIN}(t)$) with elongation rate γ (mm·d^{-1}). A monomolecular function ($f_{PLA}(t)$) was used to describe decreasing elongation rate during the plateau phase, where the proportion of growth remaining when the plateau phase begins is represented by δ (mm), and k_{PLA} (per day) represents the rate at which the plant reaches the asymptote.

$$f_{3-phase}(t) = \begin{cases} f_{LAG}(t) = \alpha + e^{\beta t} - 1 & \text{...for } t \le t_L \\ f_{LIN}(t) = f_{LAG}(t_L) + \gamma t & \text{...for } t_L < t \le t_P \\ f(t) = f(t_P) + \delta[1 - e^{-k_{PLA}(t - t_P)}] & \text{for } t > t_P \end{cases} \quad [6]$$

where the function and the first derivative were continuous at the transition points (t_L and t_P)

$$\begin{aligned} f_{LAG}(t_L) &= f_{LIN}(t_L) \\ f_{LIN}(t_P) &= f_{PLA}(t_P) \\ f'_{LAG}(t_L) &= f'_{LIN}(t_L) \\ f'(t) &= f'(t) \end{aligned} \quad [7]$$

and the first derivative $f'_{3-phase}$ is

$$f'_{3-phase}(t) = \begin{cases} f'_{LAG}(t) = \beta e^{\beta t} & \text{...for } t \le t_L \\ f'_{LIN}(t) = \gamma & \text{...for } t_L < t \le t_P \\ f'(t) = k\delta e^{-k_{PLA}(t - t_P)} & \text{for } t > t_P \end{cases} \quad [8]$$

In the elongation model described by Fisher et al. (1996), t_P was assumed to occur some number of days, Δt_{VP}, after the timing of visible bud (t_V), because at t_V the apical flower bud is visible and all internodes on the stem have begun to elongate. t_V was in turn assumed to occur Δt_{SV} days after the start of short days (t_S) (when flower-initiating nyctoperiods begin). The model was therefore assigned parameters to estimate Δt_{VP} and calculate t_P based on the timing of visible bud:

$$t_P = t_v + \Delta t_{VP} \quad [9]$$

The $f_{3-phase}$ function (Eq. [6]) was fitted to data for control (untreated) plants to obtain a curve for elongation in the absence of a growth retardant application. The resulting model was then modified to incorporate the effect of a dose response function *g(t)* on plant height *H* by

$$H = \int f'3\text{–}phase(t)g(t)dt \quad [10]$$

and

$$\frac{dH}{dt} = \int f'3\text{–}phase(t)g(t) \quad [11]$$

The parameters in the exponential dose response function $g_E(t)$ (Eq. [5]) were changed to create a new variable *D* that was the inverse of C_E:

$$g_L(t) = \begin{cases} 1 & \text{..for } t \le t_{ev} \\ 1 - (1 - M_E)e^{[-\frac{1}{D}(t - t_{ev})]} & \text{..for } t > t_{ev} \end{cases} \quad [12]$$

By using the version of $g_E(t)$ in Eq. [12], high values of *P* (Eq. [4]) and *D* both indicated a slower recovery from the growth retardant (i.e., a greater duration of effect).

Experimental design for calibration data set. Rooted cuttings of 'Annette Hegg Dark Red' poinsettia were transplanted 17 Aug. 1994 into 15-cm-diameter (1000-cm^3) pots. Plants were grown in a Michigan State University (MSU, East Lansing) greenhouse at an average light integral of 11.7 ± 2.7 mol·m^{-2}·d^{-1} (means are displayed ± standard error unless otherwise stated), a daily temperature of 21.4 ± 0.1 °C and a DIF (average day minus average night) temperature of 0.9 ± 0.1 °C. Night-interruption lighting was supplied from 2200 to 0200 HR until 27 Sept. Black cloth was pulled from 1730 until 0800 HR from 27 Sept. until 25 Oct. after which plants were exposed to natural nyctoperiods.

A foliar spray of chlormequat (10 ml/plant) was applied with a hand sprayer to four sides of the plant to achieve thorough coverage. This spray volume ensured complete coverage of all leaf surfaces without runoff, although some spray drifted onto the media surface. Chlormequat was applied on 20 Sept., 34 days after transplanting, at six rates: 500, 1000, 1500, 2000, 3000, and 4000 ppm plus an unsprayed control. Five plants per treatment were positioned randomly on a single subirrigated bench and were surrounded by untreated and unmeasured boundary plants. Plants were placed at 35 × 35-cm spacing to avoid light-quality effects from leaves overlapping between plants. Lateral shoots were removed every 2 weeks and plants were grown as single, i.e., unpinched, stems to facilitate height measurements. Height from the pot rim to the stem apex was measured twice weekly from transplanting until 1 week before application, daily from 1 week before application until 2 weeks after application, and twice weekly from 2 weeks after application until 2 weeks after anthesis. Leaf number was recorded weekly. Dates of first bract color (first color), externally visible flower buds (visible bud), and anthesis were recorded for each plant.

Experimental design for validation data set. In a separate experiment at an MSU greenhouse during 1993, 40 rooted cuttings of 'Annette Hegg Dark Red' were planted in 15-cm-diameter pots on 20 Aug., and groups of 10 plants received chlormequat spray applications (10 ml/plant) at 0, 1000, 2000, or 3000 ppm 24 days after planting. Plants were not pinched, and lateral shoots were removed 15, 30, and 68 days after transplanting to leave a single stem. Night-interruption lighting was supplied until 36 days after transplanting, and was followed by 14-h nyctoperiods until anthesis. Average light integral was 11.3 ± 2.5 mol·m^{-2}·d^{-1}, and daily and DIF temperatures averaged 21.5 ± 0.1 °C and -0.2 ± 0.1 °C. Plants were subirrigated and grown at 35×35-cm spacing in a completely randomized design. Plant height to the stem apex and leaf count were recorded several times each week until anthesis and date of visible bud was also recorded.

Analysis. The $f_{3\text{-}phase}$ model (Eq. [6]) was fitted to the control plants in the calibration experiment to obtain untreated estimates for β, γ, Δt_{VP}, and δ using PROC NLIN, the nonlinear regression procedure of SAS (SAS Institute, 1988). α was assigned the value of the observed transplant height for each plant.

Elongation from t_{ev} until the end of the experiment was compared against growth retardant concentration using linear regression to confirm that increasing growth retardant concentration reduced elongation. The assumption in $g_L(t)$ and $g_E(t)$ functions that growth retardants had an immediate effect was tested using ANOVA to compare the growth rates of the treated and control plants between t_{ev} and the first measurement date on $t_{ev} + 1$. Effects of the chlormequat application on leaf unfolding rate and the time to first color, visible bud, and anthesis were compared by ANOVA to investigate whether development rate was affected in addition to stem elongation rate.

The $f_{3\text{-}phase}$ function (Eq. [6]) was fitted separately to data from each treatment between t_0 (time of transplanting) and t_{ev} using SAS PROC NLIN, to estimate β for each growth retardant treatment. Estimating β by treatment did not affect predicted elongation rate after t_L (or t_{ev}), but accounted for random variation in initial elongation rate before t_L and improved fitting of the dose response models. Parameters γ, Δt_{VP}, and δ were assumed to be equal to the parameter estimates for untreated control plants, and these estimates were entered as constants in the model.

Computation of H using Eq. [10] required a numerical simulation procedure that used a rectilinear integration method with an integration interval of 0.5 days. The resulting data set of predicted H, using parameters for γ, Δt_{VP}, and δ from control plants and the estimates of β by treatment, was used to estimate $g(t)$ parameters for each concentration. The $g(t)$ parameter estimates were graphed and a function was fitted to these data to estimate the dose response as a function of concentration. The $f_{3\text{-}phase}$ control parameters were combined with the $g_L(t)$ and $g_E(t)$ dose response functions to derive complete models, termed G_L and G_E respectively, for the elongation of single-stem poinsettia beginning at time of transplanting (t_0). A simulation program with a time step of 0.5 days was used to generate data sets of predicted heights from both models. Goodness of fit of the predicted heights against the entire observed data set, including treated and untreated plants, was then quantified.

Finally, the model was validated against the 1993 data set. The $f_{3\text{-}phase}$ model (Eq. [6]) was fit to the control plants in the validation experiment to obtain untreated estimates for β, γ, Δt_{VP}, and δ using PROC NLIN, and α was assigned the value of the observed transplant height for each plant. Predicted heights were obtained by simulation of the $g(t)$ models from the calibration experiment combined with the untreated elongation curve to form G_L and G_E models for the validation data set. Predicted heights from the G_L and G_E model were then compared for goodness of fit against the observed height.

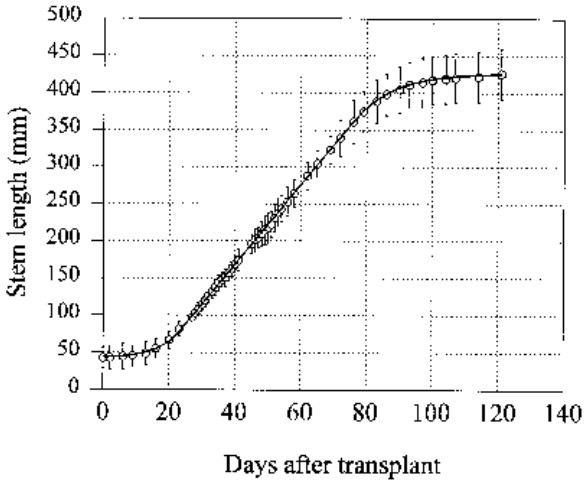

Fig. 2. Plot of observed and predicted average stem length over time for the untreated plants during the calibration experiment: ❍ = mean observed height ± 95% confidence intervals, solid line = fit of the $f_{3\text{-}phase}$ function.

Results

Fitting the $f_{3\text{-}phase}$ function to control data. Elongation followed a three-phase pattern (Fig. 2), with an initial lag in elongation followed by a near-linear period of growth and a final plateau stage. Fitting the $f_{3\text{-}phase}$ function to the height of untreated control plants resulted in an R^2 of 0.996 (Fig. 2, Table 2). Final height (H_f), including the transplant height (α), was estimated to be 426 mm, equal to the observed final height of 426 ± 17 mm. Plants were estimated to begin the linear phase of elongation 23 days after transplant, when they were 78 ($f_{LAG}(t_L)$) mm in length and elongated at over 5 mm·d^{-1} (γ). The linear phase was estimated to end 15 (Δt_{VP}) days after visible bud (or 35 days after t_s) at a height of 365 ($f_{LIN}(t_P)$) mm, after which an additional 63 mm of elongation (δ) was predicted during the plateau phase.

Table 2. Analysis of variance and parameter estimates from fitting the $f_{3\text{-}phase}$ model to the untreated control data from the calibration experiment.

Source	df	Sum of squares	Mean square
Regression	4	13103843	3275961
Residual	200	57586	288
Uncorrected total	204	13161429	
Corrected total	203	3208912	$R^2 = 0.996$
Estimated parameters		**Estimate ± ASE [z]**	**Units**
β		0.154 ± 0.003	per day
γ		5.36 ± 0.10	mm·d^{-1}
Δt_{VP}		15.4 ± 3.1	days
δ		63 ± 20	mm
Observed factors			
α		43 ± 14	mm
Calculated factors			
t_L		23.1	days
t_P		76.5	days
$f_{LAG}(t_L)$		78	mm
$f_{LIN}(t_P)$		365	mm
k_{PLA}		0.0852	per day
H_f		427	mm

[z] ASE = asymptotic standard error.

Growth retardant treatments. There was a statistically significant effect (P = 0.0005) of growth retardant concentration on elongation after t_{ev}, and height decreased with increasing chlormequat concentration with a range from 289 ± 15 mm for the control plants to 236 ± 17 mm at 4000 ppm. Growth retardant applications did not affect timing of visible bud, first color, or anthesis (which averaged 20.1 ± 0.1 (Δt_{SV}), 16.1 ± 0.4, and 50.7 ± 0.7 days, respectively, after the start of short days on day 41). Final leaf count (average 25.7 ± 0.2 leaves) was also not affected by growth retardant concentration.

Fitting the g(t) dose response models. We found that estimating β separately for each treatment improved fitting of the dose response functions because this ensured that predicted and observed height were very similar (within 5 mm) at time t_{ev}. Elongation that occurred before growth retardants were applied at t_{ev} was not significantly different between treatments (data not shown). However, we found that the Larsen and Lieth (1993) protocol for fitting the *g(t)* models using SAS PROC NLIN was highly sensitive to even slight differences between observed and predicted height at t_{ev}. Estimating β by treatment did not affect predicted elongation rate after t_{ev} because in all cases, t_L, the start of the linear phase, was estimated to occur before t_{ev} and γ, Δt_{VP}, and δ were assumed equal to the estimates from control data. Estimates of β were 0.154 ± 0.003, 0.141 ± 0.001, 0.156 ± 0.002, 0.164 ± 0.002, 0.155 ± 0.003, 0.176 ± 0.004, and 0.153 ± 0.003 per day (estimate ± asymptotic standard error) for the control, 500, 1000, 1500, 2000, 3000, and 4000 ppm treatments respectively.

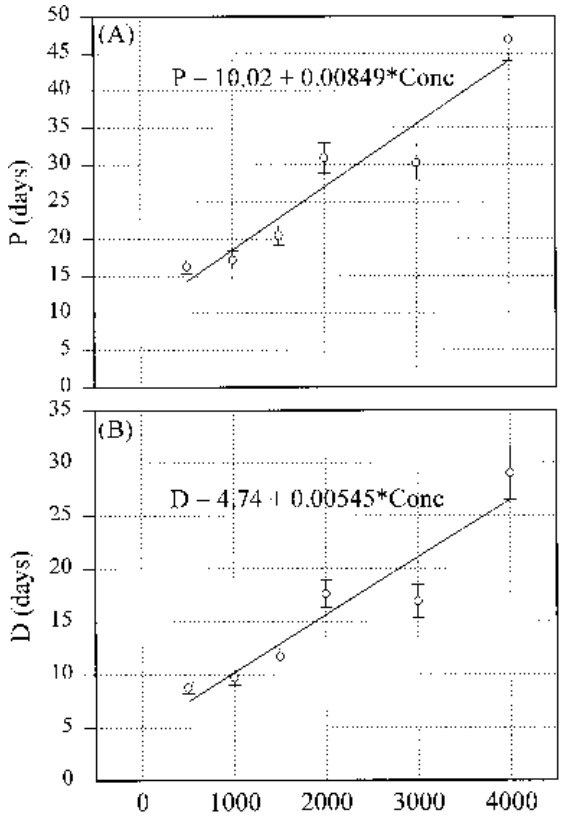

Fig. 3. Parameter estimates (❍) for (**a**) the persistence P in the linear *g(t)* model and (**b**) the duration parameter D in the exponential *g(t)* models respectively. The solid lines represent the linear regression (Eq. [13]) fit to the parameter estimates where the parameter a represents the y-intercept constant and b represents the gradient constant.

Table 3. Estimates of a and b used to calculate persistence P and duration D parameters in the $g_L(t)$ and $g_E(t)$ models respectively.

	$g_L(t)$ (Linear model)	$g_E(t)$ (Exponential model)
Amplitude	M_L = 0.53	M_E = 0.53
a (days)	10.558 ± 1.278	5.523 ± 0.784
b (days/ppm)	0.00824 ± 0.0006	0.00493 ± 0.0004
R^2	0.989	0.989

We found a high degree of correlation between amplitude (M_L and M_E for Eqs. [4] and [12] respectively) and duration parameters P and D when fitting the *g(t)* functions to the treated plants. Estimating both the amplitude and recovery parameters by nonlinear regression was therefore not a reliable method for modeling the dose response.

Because of correlation between amplitude and duration parameters, we chose to set amplitude parameters (M_E and M_L) to a constant (0.53) based on observed elongation immediately after t_{ev}. Growth retardant treatments elongated an average 53% as rapidly during the first day after application compared with the control (3.5 ± 0.5 mm and 6.6 ± 0.8 mm respectively, P = 0.027). Chlormequat concentration between 500 and 4000 ppm did not, however, significantly affect initial elongation (3.6 ± 1.6, 2.8 ± 1.2, 4.8 ± 1.0, 2.8 ± 0.9, 3.8 ± 1.2, and 3.0 ± 1.0 mm for 500, 1000, 1500, 2000, 3000, and 4000 ppm respectively). We assumed that the main effect of growth retardant concentration was to increase the duration of effect (P and D), and we estimated P and D by nonlinear regression.

Both *g(t)* models were fitted to each concentration to obtain a data set of estimates of P and D (Fig. 3). Both P and D increased with increasing chlormequat concentration between 500 and 4000 ppm. For example, the growth retardant effect was predicted to persist for 16 days at 500 ppm and 47 days at 4000 ppm by the linear $g_L(t)$ model. Parameters P and D were modeled as a linear function of concentration using the equation

$$X = a + b \times Concn \quad [13]$$

where X represents P or D for the $g_L(t)$ and $g_E(t)$ models respectively, and *Conc* was the chlormequat concentration in ppm. a and b have units of days and days/ppm respectively

Initial estimates of parameters a and b in Eq. [13] were calculated from linear regression of the estimated P and D parameters versus concentration (Fig. 3). The resulting estimates were used as starting values for fitting the $g_L(t)$ and $g_E(t)$ models separately to the entire data set of treated plants in order to estimate a and b by nonlinear regression. Of the remaining parameters in the complete G_L and G_M models, the amplitude parameters (M_L and M_E) were set to 0.53, based on observed elongation rate between t_{ev} and t_{ev}+1; the $f_{3\text{-}phase}$ model parameters γ, Δt_{VP}, and δ were set to the estimates for the untreated control plants (Table 2) and β was set to the value previously estimated for each treatment.

The resulting two *g(t)* models (Table 3) both had an R^2 of 0.989, and the coefficient of variation of parameter estimates was also similar. At 1500 ppm, the $g_L(t)$ model predicted greater growth-retarding effect than the $g_E(t)$ model during the first 18 days after t_{ev} (Fig. 4a), whereas the $g_E(t)$ model predicted greater growth retarding effect as the $g_L(t)$ model neared 1. The relative growth-retarding ($g(t)$) effect predicted by the models never differed by more than 0.1 for all concentrations between 500 and 4000 ppm and all days after application (Fig. 4a). The $g_L(t)$ model predicted that a 500 ppm growth retardant applied 34 days after transplant would persist for 15 days and reduce height by 6% compared with an untreated plant (Fig. 4b); at 4000 ppm persistence would be 44

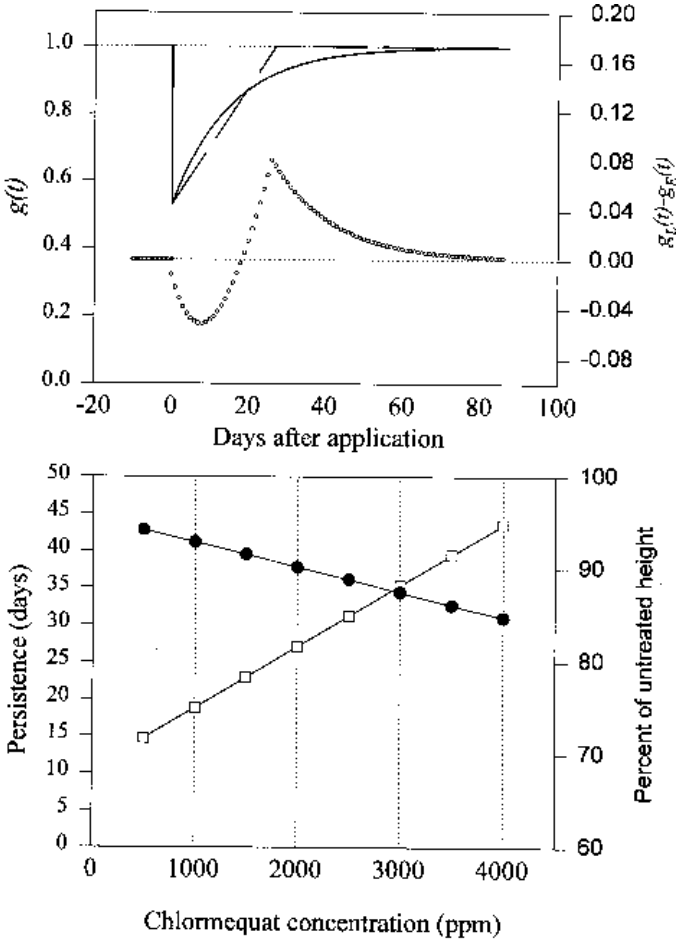

Fig. 4. (**a**) Comparison of $g_L(t)$ (dashed line) and $g_E(t)$ (solid line) dose response models assuming a 1500 ppm chlormequat application, ❍ = difference between models ($g_L(t)$ minus $g_E(t)$). (**b**) Final height of chlormequat-treated plants relative to that of untreated plants for a growth retardant applied t_{ev}=34 days after transplant (●), and growth retardant persistence for various concentrations (*Conc*) using the $g_L(t)$ model (❑).

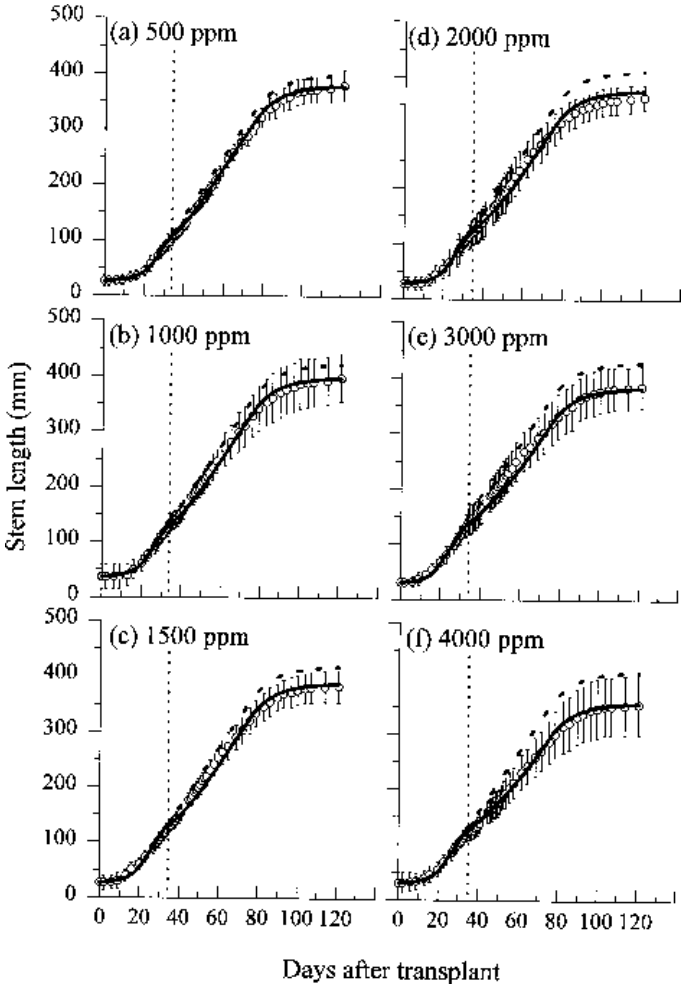

Fig. 5. (**a**–**f**) Plot of observed average stem length (❍) for growth retardant treatments over time during the calibration experiment and predicted height from the exponential $g_E(t)$ model (solid line). The dashed line represents heights predicted by the $f_{3\text{-}phase}$ model in the absence of a growth retardant application.

days and height reduction 15%. There was little difference between the predictions of the G_L model and those of the G_M model, and at a given chlormequat concentration the final heights predicted by both models were within 3 mm, or 1% (the G_L model predicting slightly more elongation). Predictions by both models were within 9 mm of the average final height for each treatment, with the greatest deviation for the 2000 ppm treatment (Figs. 5 and 6).

Model validation. The $f_{3\text{-}phase}$ function was fitted to the untreated control plants (Table 4, Fig. 7a) in the validation experiment. The control plants elongated more rapidly during the lag phase compared with the control plants in the calibration experiment, with a significantly higher estimate of β and an earlier estimate of t_L. Elongation of the untreated validation plants was 26% slower during the linear phase compared with the calibration experiment. However, validation experiment transplants were 7 cm longer than the calibration cuttings. Therefore, although total predicted final height (H_f) of the control plants was only 3 cm shorter in the validation experiment compared with the calibration experiment, validation plants were 10 cm shorter when only elongation after transplant was considered. The estimate of δ (21 ± 19 mm) had a high asymptotic standard error, which was probably because few data points were collected from the plateau phase. The δ parameter for the validation plants was estimated to be only one-third of the length of the plateau in the calibration experiment, in part because the estimate of Δt_{VP} was 9 days later than that in the calibration experiment.

Heights were predicted for the growth retardant-treated plants using the $g(t)$ models (Table 3) from the calibration experiment combined with the $f_{3\text{-}phase}$ parameters from the untreated validation plants (Table 4), simulating with a 0.5-d time step. The estimates of β were not significantly different between growth retardant treatments, and the estimate of β for untreated control plants was used in all cases. The height predicted by G_L (not displayed) and G_E models (Fig. 7 b–d) closely tracked the observed height (r^2 = 0.967) and was always within the 95% confidence intervals. However, both models overpredicted final height of the growth retardant-treated plants, by 3, 9, and 8 mm (G_E model) for the 1000, 2000, and 3000 ppm, respectively, and by 6, 12, and 11 mm (G_L model).

Discussion

The untreated control plants exhibited a three-phase elongation pattern in both experiments. However, between years there were differences in the length of each phase and the elongation rate. Validation control plants elongated 10 cm less after transplant than the calibration plants. Flower-initiating nyctoperiods were begun five days earlier in the validation experiment than in the calibration experiment, and the $f_{3\text{-}phase}$ model would predict about 26 mm less

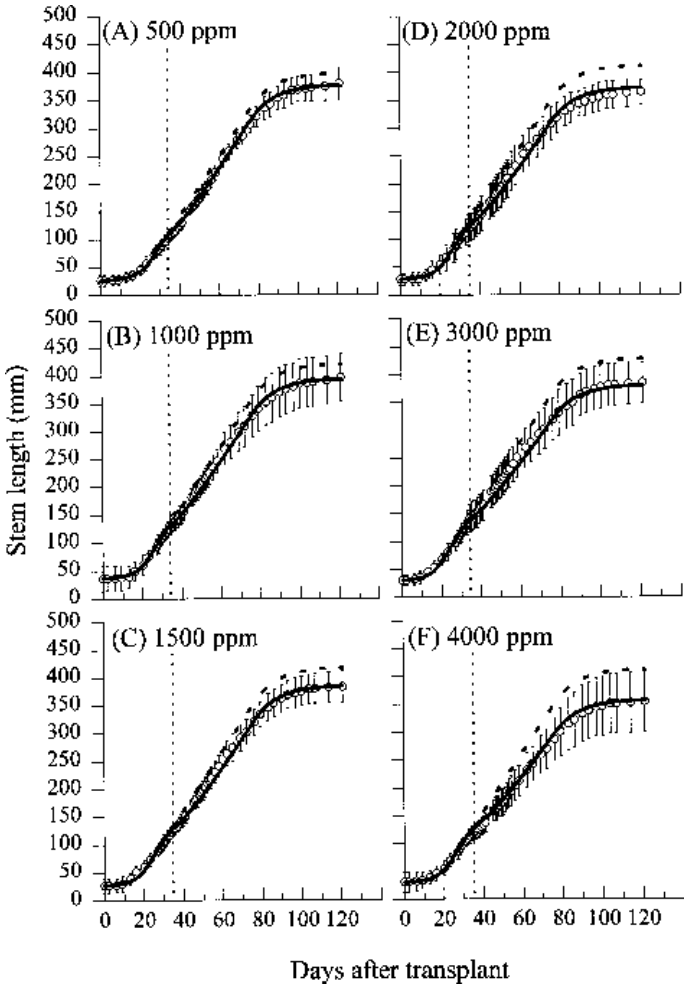

Fig. 6. (**a**–**f**) Plot of observed average stem length for growth retardant treatments over time during the calibration experiment (❍) and predicted height from the linear $g_L(t)$ model (solid line).

elongation in the validation experiment, based on the effect of flower-initiation date described by Fisher et al. (1996). DIF temperature was 1 °C lower during the validation experiment, which would also have slightly reduced elongation (Berghage and Heins, 1991). Differences in transplant size and vigor also may have affected elongation. Few data were collected during the plateau phase in the validation experiment, and plants may have elongated more than estimated.

The dose response *g(t)* models are highly empirical. In the equation describing the effect of chlormequat concentration on duration of effect (Eq. [13]), *a* represents the intercept or duration when concentration *Conc* = 0, and *b* represents the effect on duration of increasing *Conc*. Because *a* does not equal zero duration at *Conc* = 0, the model should not be extrapolated beyond concentrations of 500 to 4000 ppm. The model predicts that initial retarding effect is independent of concentration between 500 to 4000 ppm: for all concentrations in this range, the maximum growth retarding effect is predicted to be 53% of the untreated control.

This type of experiment and methodology does not effectively identify the most biologically valid or mechanistic formulation of *g(t)*. In Larsen and Lieth's (1993) research on daminozide applications to chrysanthemum, $g_L(t)$ and $g_E(t)$ equations resulted in a close fit to observed data. The researchers noted close correlation of amplitude and recovery factors, a similar effect on absolute growth rate of the two *g(t)* models, variability in data, and a lack of knowledge of the physiology of daminozide activity. As a result, although their methodology was an effective empirical model, Larsen and Lieth (1993) noted that it was less effective at selecting the most biologically valid form of *g(t)*. The most appropriate form of *g(t)* is likely to vary depending on chemical type, translocation pathway, and plant species. Either Larsen and Lieth (1993) *g(t)* model may be appropriate for daminozide on chrysanthemum, because translocation and activity are rapid (Dicks, 1972) and the effect declines over time (Dicks, 1972; Dicks and Charles-Edwards, 1973; Tayama and Carver, 1992).

Increased knowledge of growth retardant physiology would be necessary to develop a more mechanistic model. Chlormequat primarily retards height by inhibiting internode elongation (Steffens, 1980), although the chemical inhibited subapical cell expansion and division in chrysanthemum, and promoted transverse stem growth, resulting in short, thick stems (Sachs and Kofranek, 1963). The principal mechanism of action for chlormequat has been associated with the inhibition of gibberellin biosynthesis resulting in a reduction in the endogenous content of gibberellins (Grossman, 1992). Effects on gibberellin activity, IAA metabolism, ethylene production, and sterol synthesis also may be involved, however (Steffens, 1980).

Chlormequat is absorbed rapidly, with more than 90% of ^{14}C-labeled chemical applied to a wheat leaf being taken up within 24 h (Arissian et al., 1991). Rapid absorption supports the *g(t)* assumption of immediate growth-retarding effect after t_{ev}. Chlormequat has been described as readily translocated in the xylem and phloem and is highly water-soluble (Krishnamoorthy, 1981; Smith et al., 1982). However, Arissian et al. (1991) found that over 85% of the chemical remained in a treated wheat leaf 10 days after a foliar application. This result suggests that as a plant elongates over time, the zones of cell division and elongation may become physically distant from the original application sites that contain the highest concentration of chlormequat.

Table 4. Analysis of variance and parameter estimates from fitting the $f_{3\text{-}phase}$ model to the untreated control data from the validation experiment.

Source	df	Sum of squares	Mean square
Regression	4	17817477	4454369
Residual	320	40502	127
Uncorrected total	324	17857979	
Corrected total	323	2138957	R^2 = 0.997
Estimated parameters		Estimate ± ASE[z]	Units
β		0.169 ± 0.003	per day
γ		3.95 ± 0.44	mm·d^{-1}
Δt_{VP}		24.4 ± 3.6	days
δ		21 ± 19	mm
Observed factors			
α		110 ± 11	mm
Calculated factors			
t_L		18.6	days
t_P		79.9	days
$f_{LAG}(t_L)$		133	mm
$f_{LIN}(t_P)$		375	mm
k_{PLA}		0.186	per day
H_f		396	mm

[z] ASE = asymptotic standard error.

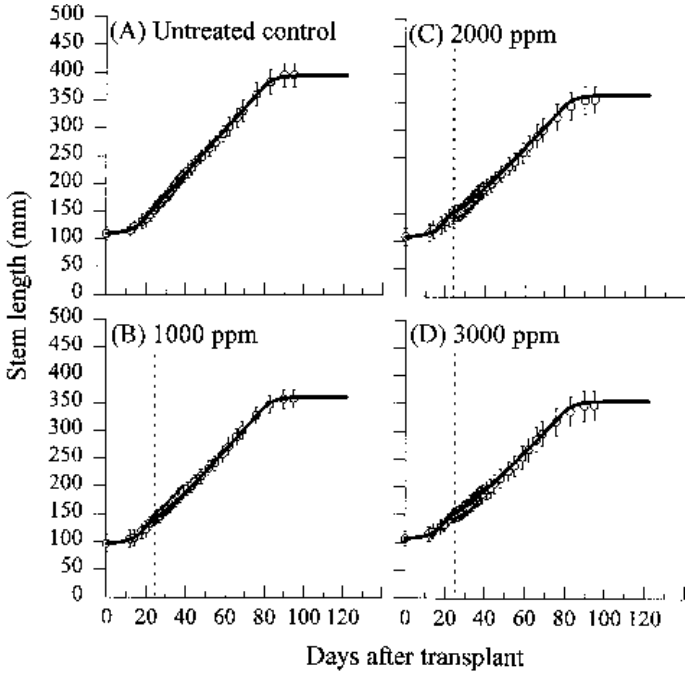

Fig. 7. (**a**–**d**). Plot of the validation data set showing observed average stem length for control and growth retardant treatments over time (❍) and predicted height from the exponential *g(t)* model combined with the $f_{3\text{-}phase}$ function fit to the untreated validation control plants (solid line).

Smith et al. (1982) reviewed research on the rate of degradation of chlormequat chloride and found contradictory evidence. Some research suggested rapid degradation (for example, 20% to 30% metabolism within 24 h in barley and chrysanthemum shoots (Schneider, 1967)) whereas other reports indicated that the compound may be stable in wheat plants for four or more weeks after application. Decomposition rate of chlormequat is affected by temperature, with little decomposition below 4 °C or above 40 °C, and the chemical is broken down by soil microorganisms if applied as a drench (Steffens, 1980). Growth-retarding activity may not simply decline as metabolism of chlormequat increases. Smith et al. (1982) suggested the possibility that the growth effects attributed to the parent compound may occur as a result of a combination or balance of parent compound and metabolites.

We chose the *g(t)* models based on achieving the desired empirical qualities for the intended use of the model. The initial amplitude M_L or M_E can be approximated as the observed reduction in elongation immediately after t_{ev}, whereas P and D must be estimated by nonlinear regression. Data were highly variable within growth retardant treatments when examined over short periods; for example, the first one to three days after t_{ev}. However, a statistical comparison of initial elongation did not indicate that initial elongation rate was affected by concentrations between 500 and 4000 ppm. When we used nonlinear regression to estimate M_L or M_E in addition to the duration equation (Eq. [13]), the amplitude estimates were 0.75 ± 0.01 (± asymptotic standard error) and 0.71 ± 0.02 for the G_L and G_E models, respectively. Although the resulting models overall had a slightly higher R^2 (0.990) than the G_L and G_E models in Table 3, the predicted heights during the first 10 days after application did not fit as well to observed data, especially in the validation experiment. Using nonlinear regression to estimate amplitude therefore less effectively predicted short-term effects of chlormequat, which was our primary intended use of the model, than assuming a 47% initial reduction.

The resulting model closely fit the observed data during calibration and validation trials. Further validation of this model would be necessary to incorporate variables that are important in commercial horticulture. Effect of plant pinching, cultivar, average and DIF temperatures, date and method of application, and number of applications should be considered. Several other growth retardants are applied to poinsettia (Ecke et al., 1990), including ancymidol, daminozide (applied in combination with chlormequat), paclobutrazol, and uniconazole. Therefore, a comprehensive growth retardant dose response model would need to make considerable simplifying assumptions to accommodate this large combination of possible situations. The model could be used to aid height-control decisions, by predicting the final effect of a single foliar application of chlormequat on percent height reduction (Fig. 4b) and dynamically simulating the effect of a proposed or actual application on elongation over time.

Literature Cited

Arissian, M., D. Perrissin-Fabert, A. Blouet, J. Morel, and A. Guckert. 1991. Effect of imazaquin on absorption, translocation, and pattern of distribution of chlormequat chloride in winter wheat. J. Plant Growth Regulat. 10:1–4.

Barrett, J.E. 1982. Chrysanthemum height control by ancymidol, PP333, and EL-500 dependent on medium composition. HortScience 17:896–897.

Berghage, R.D. and R.D. Heins. 1991. Quantification of temperature effects on stem elongation in poinsettia. J. Amer. Soc. Hort. Sci. 116:14–18.

Dicks, J.W. 1972. Uptake and distribution of the growth retardant, aminozide, in relation to control of lateral shoot elongation in *Chrysanthemum morifolium*. Ann. Appl. Biol. 72: 313–326.

Dicks, J.W. and D.A. Charles-Edwards. 1973. A quantitative description of inhibition of stem growth in vegetative lateral shoots of *Chrysanthemum morifolium* by N-Dimethylaminosuccinamic acid (Daminozide). Planta 112:71–82.

Ecke, P., O.A. Matkin, and D.E. Hartley. 1990. The poinsettia manual. 3rd ed. Paul Ecke Publications, Encinitas, Calif.

Fisher, P.R., R.D. Heins, and J.H. Lieth. 1996. Quantification of the relationship between phases of stem elongation and flower initiation in poinsettia. J. Amer. Soc. Hort. Sci. (In press.)

Gilbertz, D.A. 1992. Chrysanthemum response to timing of paclobutrazol and uniconazole sprays. HortScience 27:322–323.

Grossman, K. 1992. Plant growth retardants: Their mode of action and benefit for physiological research, p. 788–797. In: C.M. Karssen, L.C. von Loon, and D. Vreugdenhil (eds.). Progress in plant growth regulation, Kluwer, Netherlands.

Holcomb, E.J., L. Gohn, and J.C. Shriver. 1992. 1991 poinsettia trials: Fertigation and growth retardant trials. Pennsylvania Flower Growers Bul. 411.

Holcomb, E.J. and M. Rose. 1992. Poinsettia height reduction using multiple applications of Sumagic. Pennsylvania Flower Growers Bul. 413.

Krishnamoorthy, H.N. 1981. Plant growth substances. McGraw-Hill, New Delhi.

Larsen, R.U. and J.H. Lieth. 1993. Shoot elongation retardation owing to daminozide in chrysanthemum: I. Modeling single applications. Scientia Hort. 53:109–125.

Larson, R.A. 1967. Chemical growth regulators and their effects on poinsettia height control. North Carolina Agr. Expt. Sta. Tech. Bul. 180.

Lieth, J.H. and J.F. Reynolds. 1986. Plant growth analysis of discontinuous growth data: A modified Richards function. Scientia Hort. 28:301–304.

Ludolph, D. 1992. Height control of ornamental plants without chemical growth retardants. Ohio Florists Assn. Bul. 748:1–4.

McDaniel, G.L. 1986. Comparison of paclobutrazol, flurprimidol, and tetcyclasis for controlling poinsettia height. HortScience 21:1161–1163.

McDaniel, G.L. and S. Wilson. 1990. Comparison of split application vs. single rate application of uniconazole on poinsettia. Tennessee Farm Home Sci. 155:11–15.

Richards, F.J. 1959. A flexible function for experimental use. J. Expt. Bot. 10:290–300.

Sachs, R.M. and A.M. Kofranek. 1963. Comparative cytohistological studies on inhibition and promotion of stem growth in *Chrysanthemum morifolium.* Amer. J. Bot. 50:772–779.

Statistical Analysis Systems Institute. 1988. SAS/STAT users guide, version 6.03. SAS Institute, Cary, N.C.

Schneider, E.F. 1967. Conversion of the plant growth retardant (2-chloroethyl) trimethyl ammonium chloride to choline in shoots of chrysanthemum and barley. Can. J. Biochem. 45:395–400.

Smith, A.R., T.H. Thomas, and J.F. Garrod. 1982. Specificity and mode of action of BTS 44584 and chlormequat chloride in wheat and soybeans: II. Distribution and persistence. Ann. Applied Biol. 101:349–357.

Steffens, G.L. 1980. Applied uses of growth substances—Growth inhibitors, p. 397–408. In: F. Skoog (ed.). Plant growth substances 1979. Springer-Verlag, Berlin.

Tayama, H.K. and S.A. Carver. 1992. Residual efficacy of Uniconazole and Daminozide on potted 'Bright Golden Anne' chrysanthemum. HortScience 27:124–125.