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Temperature and the Growth of Plant Cells

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Abstract

In cell elongation, the juvenile cell vacuolates, takes up water, and expands by irreversible extension of the growth-limiting primary walls. This process was elaborated analytically by Lockhart in the mid-1960s. His growth equation does not, however, include the influence of the environmental temperature at which cell growth takes place. In this article we consider a phenomenological model including temperature in the equation of growth. Also, by introducing the possible influence of growth regulators treated here as external perturbations, linear and nonlinear solutions are found. A comparison of experimental and theoretical results permits qualitative and quantitative conclusions concerning change in the magnitude of the cell wall yielding coefficient Φ as a function of both time and temperature (with or without external perturbations), which has acquired reasonable values throughout.

Key words: Cell wall extensibility; Growth stimulators/inhibitors; Modified growth equations; Temperature

Introduction

Plant cell development consists of two interrelated processes: growth and differentiation. Growth can be described as a three-step model: (1) cell cycle (new cells are formed), (2) cell elongation, and (3) cessation of cell enlargement (cell maturation). In (step 2) the juvenile cell vacuolates, takes up water, and expands by irreversible extension of the growth-limiting primary walls. The growth process is based on irreversible extension of the whole organism as a result of the increase in the quantity and size of cells, the mass of protoplast, and the cell walls (Fogg 1975; Kutschera 2000 and articles cited therein; see for a review). Growth of any plant organ can be split into three basic phases: the initial phase of slow growth, the intense growth phase and, eventually, the final phase of slow growth. Such regularity can be represented by a sigmoid curve that characterizes the course of individual cell growth, the growth of plant organs, and the growth of the plant as a whole. Plant growth is influenced by physical (abiotic) and biotic factors of the environment (Wright 1966; Trewavas 1991; Edelmann 1995). The external factors that fundamentally influence plant growth are temperature, light, water and soil factors, pH, and atmosphere composition. A rise in temperature gradually increases the intensity of growth (which is also due to acceleration of chemical reactions by raising the temperature). However, after the optimum

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temperature has been exceeded a rapid decrease in the intensity of plant growth begins (caused by dysfunction of the plasmalemma). The experimental relations between temperature, turgor pressure, and the growth of plant cells have been investigated by Proseus and others (2000).

The problem of plant growth raises several basic questions: What limits the growth rate? What is the role of temperature in the growth rate of real plants? The first question is broadly discussed in a review article (Cosgrove 1986) where the prevailing concept of plant cell growth results from turgor-driven yielding of the cell wall, which is a physical description of how plant cells increase in size during growth and morphogenesis. Cosgrove (1986) considers plant cell enlargement as resulting from two independent processes. First, water absorption will increase the volume of the cells, given water content of 85%–95%. Second, wall yielding generates the driving force of water uptake.

Growth regulators are of fundamental importance in growth and development (see also Cleland 1986). These substances stimulate or inhibit the processes of growth. Natural regulators acting on the growth process—both stimulatory and inhibitory—are the plant hormones auxins, gibberellins, cytokinins, ethylene, and abscisic acid.

To the most intensely studied abiotic factors belong heavy metal compounds (for example, cadmium). Cadmium evokes a number of parallel and/or consecutive events at the molecular, physiological, and morphological levels (Sanita di Toppi and Gabbrielli 1999). Excess Cd causes a number of toxic symptoms in plants, like growth retardation, inhibition of photosynthesis, induction and inhibition of enzymes, altered stomatal action and water relations, efflux of cations, and generation of free radicals (Ros and others 1992; Prasad 1995; Skorzynska-Polit and Baszynski 2000). Cadmium uptake, translocation and localization in maize shoots at the tissue and cellular levels were recently investigated by Wojcik and Tukiendorf (2005), who also demonstrated that growth of maize seedlings depended strongly on Cd concentration.

The objective of the present article is to introduce the notion of temperature to the growth equations via the state equation. In our opinion the Lockhart model is a good candidate for the temperature extension: in its original form it deals with the cardinal thermodynamic quantities like the cell volume and pressure. The only factor lacking is temperature. The extension, however, is straightforward through the state equation. Such an approach, as used in our article, is fully justified (Stanley 1971) and makes possible the creation of a new phenomenological (thermodynamic) model of growth. After preparing the proper equations, we adjust them to the experimental data and quantitatively describe the elongation of maize versus time, parameterized by temperature. Two major solutions of these equations are found: (1) a linear solution for unperturbed growth and (2) linear and nonlinear solutions of growth influenced by external perturbation (growth hormones and abiotic factors), which in fact both reflect different conditions of experiments performed by the authors. Moreover, we notice that the foregoing major consequence of our model is a good working mathematical description of the most essential features of the mechanical properties of the cell wall (regarding wall yielding as a function of time and temperature).

Materials and Methods

The experiments were carried out with 4-day-old maize seedlings (*Zea mays* L.) grown on Hoagland's medium (Hoagland and Arnon 1950) at 27°C. Seeds of maize were cultivated in darkness. We have chosen five seedlings of the same (2 cm) length. The initial length of the segments in all experiments was 2 mm. All segments were cut along the coleoptile axis from the same part of it, about 3 mm below the cap. This fragment of maize coleoptile elongates most intensely and is obviously free from cell divisions. Individual coleoptile segments were transferred to an aerated solution containing standard micro- and macroelements.

The segments were grown in darkness, and manipulations of plants (elongation measurements and transfer to test solutions) were carried out under green light (sunlight transmitted through a green filter) because coleoptile growth does not respond to this light spectrum. Two kinds of experiment were performed: (1) an unperturbed experiment(control): The coleoptile segments were divided into eight groups growing at different temperatures from 5°C, increased by 5°C up to 40°C. Each group was represented by 5 segments. The experiment was carried out within 2 h, and the measurements were taken every 30 min. (2) an experiment in which perturbation was present: The coleoptile segments were divided into eight groups growing at temperatures ranging from 5°C, increased by 5°C up to 40°C. The experiment was carried out within 2 h, and measurements were taken every 30 min. In the *linear response case*, auxin (indole -3- acetic acid, IAA), was applied at concentrations of 10^{-4} M; as an inhibitor, we used $CdCl_2$ (10^{-4} M), see Figure 1. In the *nonlinear response case*, the auxin was applied as a stimulator

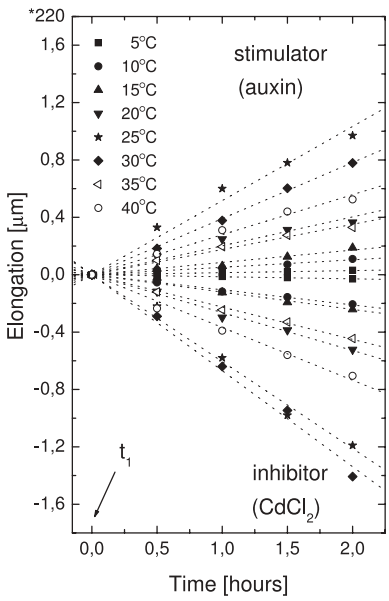


Figure 1. Experimental results: Perturbed (by the application of inhibitor/stimulator of a constant concentration one at a time $t = t_1$) elongation of maize (*Zea mays* L.) coleoptile segments versus time at eight different temperatures (the linear response).

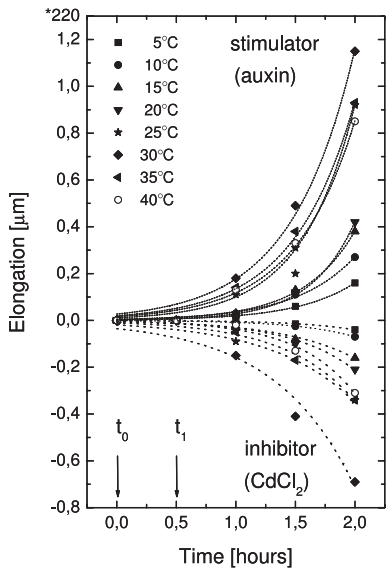


Figure 2. Experimental results: Perturbed (by the application of inhibitor/stimulator with an increasing concentration starting at a time $t = t_1$) elongation of maize (*Zea mays* L.) coleoptile segments versus time at eight different temperatures (the nonlinear response).

after $t_1 = 0.5$ h (2.5×10^{-5} M) of unperturbed growth, then after 1 h (5×10^{-5} M), and after 1.5 h (10^{-4}M); as an inhibitor, we used $CdCl_2$ after $t_1 = 0.5$ h (2.5×10^{-5} M) of unperturbed growth, then successively after 1 h (5×10^{-5} M), and 1.5 h (10^{-4} M) (see Figure 2). The values presented in Figures 1 and 2 are average values from five individual measurements. In all experimental cases, control measurements were performed. The elongation was measured by a microscope. Magnification was 75 times with a stereomicroscope (Zeiss ID 03, Germany) plus a micrometer screw (Polish Optical Manufacture, Warsaw, Poland). The standard deviation was estimated as not exceeding 10 µm.

Temperature-modified Growth Equations

In the model of plant cell growth the cell turgor pressure remains in dynamic balance between wall extension, which tends to dissipate turgor pressure, and water uptake, acting to restore it (Cosgrove 1986). In the 1960s Lockhart (1965) proposed a simple differential equation:

$$\frac{1}{V}\frac{dV}{dt} = \Phi(P - Y) \qquad (1)$$

It states that the relative growth rate (here V denotes the cell volume) depends linearly on hydrostatic pressure (P) in excess of a critical turgor (Y). All these are linked by a wall extensibility coefficient (Φ). In this model of growth, turgor pressure coordinates water uptake with cell wall extension. (It is well established that the driving force for water uptake is water potential difference across the cell membrane).

However, plants do not grow in an imaginary space disconnected from the real world. (As we noted at the beginning of this article, one of the external factors fundamentally influencing plant growth is temperature). Plants usually grow in definite conditions where one of the most important growth factors is heat—more precisely, temperature. In our approach we divide, as is usual in thermodynamics, the whole system into the investigated sample (here: a plant cell) and ''the rest of the world,'' a thermostat (the environment) that remains at a constant temperature T. By introducing the physical model (Figure 3) we are far beyond the oversimplified picture in which we interpret the movement of a piston as *only* reflecting the compressibility/extensibility properties of the water solution inside the plant cell. On the contrary, we incorporate a number of basic chemical and biochemical processes that accelerate or decelerate growth as a function of temperature (kinetics of chemical reactions, metabolism, photosynthesis [biomass production], protein denaturing, and so on). Because both types of processes act simultaneously, although with different intensity at distinct temperature ranges, crossover from one type of

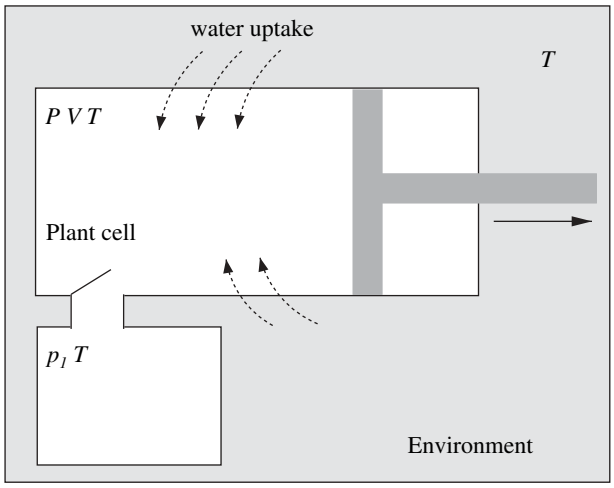


Figure 3. The ''Gedanken experiment'' set-up: The movement of the piston in the cylinder (plant cell) reflects extensibility properties of the cell wall. The additional container with pressure p_1 and the valve is bound to the action of an inhibitor or a stimulator that can be opened at a time $t_0 < t = t_1$ to release pressure p_1. The whole system is immersed into a thermostat (environment) at a temperature T.

behavior to the other can be expected. Thus, there should be a delicate balance among all those factors and consequently a specific, well-defined critical temperature at which the growth rate is optimal.

To bring temperature into Eq. (1), we utilize the state equation in the form, $P = Y + \frac{\gamma T}{V}$ where T is the absolute temperature (in Kelvin scale) and Y = const. The latter assumption needs a comment. There are few empirical data (Proseus and others 2000) revealing the turgor threshold Y dependence on temperature T. Because of the sparseness of information, we are not able to propose an exact model function for $Y(T)$. Nevertheless, in zeroth approximation we may surely assume $Y(T)$ = const. Based on Proseus and others (2000), we acknowledge that the $Y(T)$ dependence is much smaller than $P(T)$ and thus may be neglected (the assumption Y = const. still holds). V is generally interpreted as cell volume, but in the context of numerical recalculation of experimental data in light of our model we refer V to the entire organ, the coleoptile. The pressure of cell solution is proportional to T, and this is a natural consequence of the molecular energy increase with temperature. The reciprocal proportionality of P to V we introduce as in the first approximation, which gives the linear solution $V = V(t)$ of Eq. (1). This assumption goes along with the fact that the experiments are performed just in the linear range of the sigmoidal growth curve. It is, however, evident that in more precise calculations one should solve Eq. (1) for subsequent reciprocal powers of the volume V by the iteration method. Such an extension would be even more suitable nonetheless also more complicated. However, such high accuracy in cases of biological experiments in which we deal with relatively high statistical error is superfluous. In the case of external perturbations our approach yields solutions that are clear and easy to interpret and are in good agreement with the elongation experiments.

Accordingly, we propose the following equation

$$\frac{1}{V}\frac{dV}{dt} = \Phi\gamma\frac{T}{V} \tag{2}$$

Although Eq. (2) is a novel attempt to account for the effect of temperature on cell growth, its simplifying assumptions require explanation: Because the effect of T on osmotic pressure is large, we, in fact, make its role implicit in determining the hydrostatic pressure P within the cell (P stands for the net pressure) and γ is actually RS, where S is the quantity of solute within the cell.

Simple calculus yields the general expression for the volume V we have been looking for:

$$V = V_0 + \gamma T\int \Phi dt, \tag{3}$$

where $V_0 = V(t = t_0)$ stands for the initial volume of the cell. By defining $\xi(t) = \xi_t := \int \Phi(T, t)dt$ Eq. (3) can be rewritten as $V = V_0 + \gamma T\xi_t$, which expresses the solution of the nonperturbed case. Next, we construct the elongation function as

$$\text{Elong}(\tau, t) \equiv V - V_0 = \gamma T\xi_t = \gamma(\tau + 273.15)\xi_t \tag{4}$$

and τ is the measured temperature in Celsius. Eq. (3) is the solution of the unperturbed Lockhart equation and expresses change of the volume in the course of time. Notice, that it linearly increases with temperature $T = \tau + 273.15°C$. However, from empirical data (see for example, Figures 4 and 5) we realize that after reaching a critical temperature τ^* the volume [here represented by the elongation function, Eq. (4)] decreases for high temperatures. We conclude that the state equation itself is not able to describe completely the temperature dependence of elongation and cell wall extensibility. Hence, we propose the following model that allows for the calculation of the cell wall extensibility as a function of time and temperature. We bring in the temperature dependence of the elongation function as a model assumption (phenomenological Ansatz):

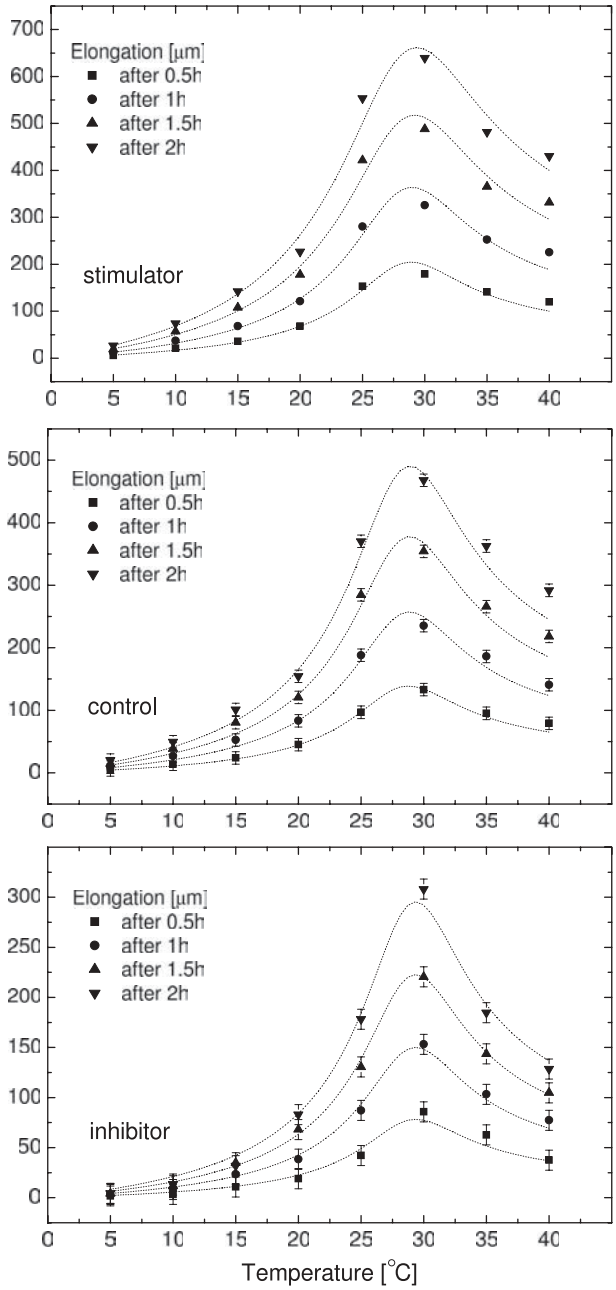


Figure 4. The experimental results for the linear case (Lorentzian fit).

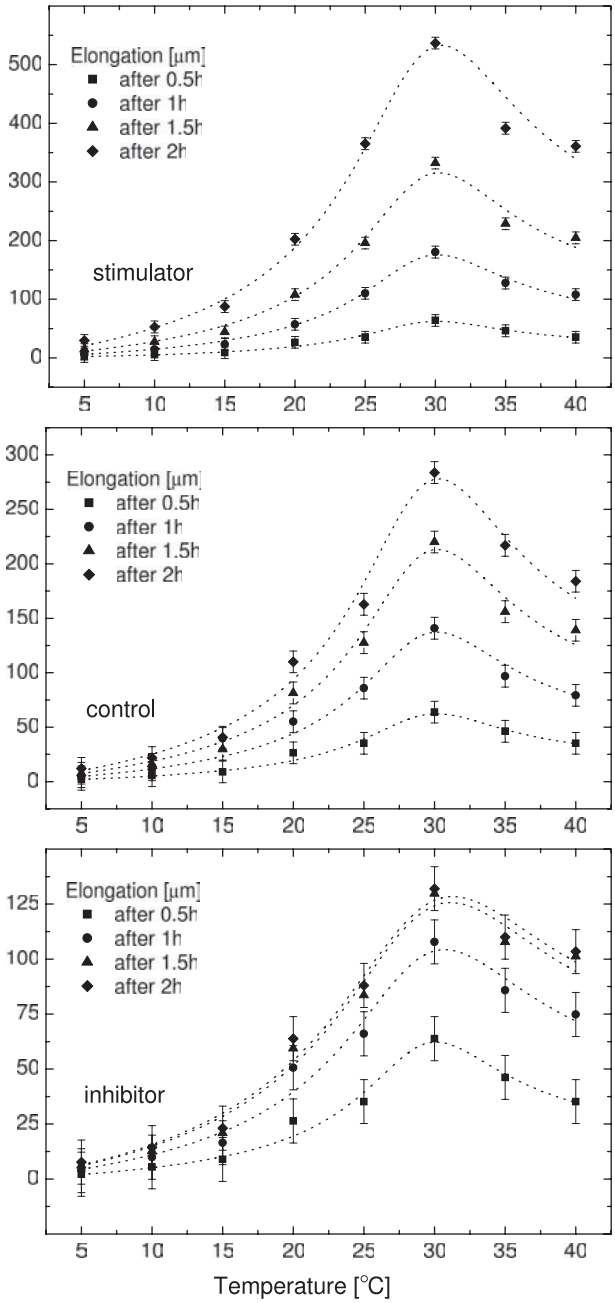


Figure 5. The experimental results for the nonlinear case (Lorentzian fit).

$$\mathrm{Elong}(\tau, t) = \frac{\phi_0(t)\tau}{\sqrt{\alpha(t)^2 + (\tau - \tau^*)^2}}, \tag{5}$$

where τ is the temperature in Celsius. We have adopted a Lorentz-like distribution (which normally describes resonance phenomena), not only because it best suits as a fitting function to the authors' experimental data but also for the important reasons described below.

The outlined system (plant cell) in the energetical context behaves like most systems described by differential equations where both dissipative and extortive forces are present. In such systems there always exists a variable that is optimal in certain conditions (like the resonance frequency $\omega = \omega^*$ for the harmonic oscillator). In our case, the factor enforcing the transition from accelerating to decelerating growth is temperature τ. As in the analogy of the harmonic oscillator, our case also

includes a critical (''resonance'') temperature $\tau = \tau^*$. However, considering the resonance temperature, we are very well aware that such magnitude is meaningless unless we treat it in the energetical context, that is, multiplying temperature by the Boltzmann constant $T \rightarrow k_B T$. This is in accordance with the fact that the optimum temperature of growth (τ^*) corresponds to the maximum energy absorption due to activation of internal biochemical processes. Consequently, in the authors' opinion the system can be described by the resonance curve—Lorentz distribution function—however, modified by the factor τ. Multiplication of the Lorentz distribution by τ is fully justified by empirical data, according to which growth should cease altogether at $\tau = 0°C$. This is also in accordance with the fact that the Celsius scale is a natural temperature scale for plants.

Even though the intuitive explanation for Eq. (5) has been presented above, nevertheless it should be derived from the first principles. A tempting way to obtain such a dependence is to combine the application of stochastic resonance in biological systems where random perturbations (here: temperature fluctuations) play a useful role in enhancing energy absorption in nonlinear systems (here: the whole complexity of basic processes stimulating plants to grow)(see for example, Hänggi 2002). This mathematically very difficult task is presently under study.

The parameters ϕ_0 and α in Eq. (5) are interpreted as the peak height and half-width of the Lorentz-like curve, respectively. Accordingly, by comparing Eqs (4) and (5) we get

$$\begin{aligned}
\xi_t &= \frac{1}{\gamma(\tau + 273.15)} \frac{\phi_0 \tau}{\sqrt{\alpha^2 + (\tau - \tau^*)^2}} \\
&\Downarrow \\
&\Phi(\tau, t) - \Phi(\tau, t_i) \\
&= \frac{\tau}{\gamma(\tau + 273.15)} \frac{d}{dt} \left(\frac{\phi_0}{\sqrt{\alpha^2 + (\tau - \tau^*)^2}} \right) \\
&= \frac{\tau}{\gamma(\tau + 273.15)} \\
&\quad \left(\frac{\phi_0'}{\sqrt{\alpha^2 + (\tau - \tau^*)^2}} - \frac{\phi_0 \alpha \alpha'}{\left(\alpha^2 + (\tau - \tau^*)^2\right)^{3/2}} \right),
\end{aligned} \tag{6}$$

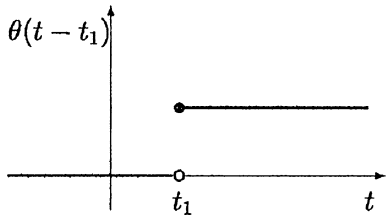
where primes denote, as usual, time derivatives. The above equation represents the exact form of the analytically derived cell wall yielding coefficient $\Phi(\tau, t)$ in the unperturbed case (with respect to its initial value at t_i for a juvenile cell). To calculate this quantity numerically, we need to know $\phi_0(t)$, $\alpha(t)$ and τ^* from fits of the experimental data. Knowledge of the (constant) γ value is found by fulfilling the condition that in most cases the difference P-Y is of the order of 0.1 MPa (see for example, Cosgrove 1985; Taylor and Cosgrove 1987; Triboulot and others 1997). We note, however, that in numerical calculations the exact value of γ is not of great importance, because it changes the amplitude (the peak value) of the calculated cell wall yielding coefficient, but the order of magnitude remains the same.

External Stimulations

As mentioned in the introductory paragraphs, plant growth can be stimulated by phytohormones or inhibited by heavy metal compounds. Stimulators or inhibitors, however, can also be applied at a certain period of time after incubation, say, at time $t = t_1$ (the experiment starts at t_0). From the mathematical point of view, it means (see also Figure 3) that we perform the following transformation (we exploit step changes in the pressure P to mimic the fact of turning on (the application of) growth phytohormones or abiotic factors at a given time t_1):

$$P \rightarrow P + p_1 \theta(t - t_1) \tag{7}$$

where $\theta(t - t_1)$ is the Heaviside theta step function that we couple to the valve placed



in the container with pressure p_1 (triggered at t_1).

Switching on the pressure $p_1 < 0$ can be interpreted as the start of the action of the plant growth inhibitor, whereas $p_1 > 0$ can be seen as the plant growth stimulator. The Heaviside theta function has such a property that it is zero for times less than t_1 (the second term in Eq. (7) vanishes what meaning is ''no added stimulator/inhibitor''). For times equal to or greater than t_1, the theta function equals one. It corresponds to the opening of the container with additional positive (stimulator) or negative (inhibitor) pressure.

Empirical data reveal that the growth rate exhibits a jump at the time (here: t_1) of application of the stimulator/inhibitor (see for example, Cosgrove 1985; Karcz and Burdach 2002). In the

Lockhart equation, the growth rate denotes the l.h.s. of Eq. (1) and equals Φ *(P–Y)*. It means that either Φ or *(P–Y)* should significantly change (in the same manner) at this point. Data in the literature (Cosgrove 1985; Kutschera 2000) demonstrate that the cell wall extensibility Φ does not undergo such abrupt changes in time. Thus we are left with the second factor in Eq. (1), *P–Y* as the candidate for modeling the stimulator/inhibitor action on growth. However, some of mechanical features related to cell wall loosening during growth are implicitly inserted into the coefficient Φ.

From the biological point of view, auxin acts mainly on the cell wall by loosening hydrogen bonds in the hemicelullose. It causes the initial underpressure in plant cells (lowering of water potential) that gives rise to water absorption. This additional water influx is reflected in Eq. (7) by the positive second term ($p_1 > 0$) indirectly reproducing the effect of the cell wall loosening.

Linear Perturbation

The assumptions $p_1 = b_1/V$ and $b_1 = const.$ will modify Eq. (2) in the following way:

$$\frac{1}{V}\frac{dV}{dt} = \Phi(T,t)\left(\frac{\gamma T}{V} + \frac{b_1}{V}\theta(t-t_1)\right) \Downarrow$$
$$V = V_0 + \gamma T\xi_t + b_1 \underbrace{\int \Phi(T,t)\theta(t-t_1)dt}_{I} \tag{8}$$

We need to find the value of the integral I relevant to our situation (satisfying the condition $I = 0$ for $t < t_1$ as in the unperturbed case; see Appendix A at http://www.springerlink.com for details). Hence

$$V = V_0 + \gamma T\xi_t + b_1\vartheta(\xi_t - \xi_{t_1}), \tag{9}$$

and thus the defined earlier elongation function reads

$$\mathrm{Elong}(\tau,t) = V - V_0 = \begin{cases} \gamma T\xi_t & t < t_1 \\ \gamma T\xi_t + b_1(\xi_t - \xi_{t_1}) & t \geq t_1 \end{cases} \tag{10}$$

Comparing Eqs (5) and (10), we obtain for $t > t_1$

$$\xi_t = \frac{1}{\gamma(\tau+273.15)+b_1}\cdot\frac{\phi_0\tau}{\sqrt{\alpha^2+(\tau-\tau^*)^2}} + \underbrace{\frac{b_1\xi_{t_1}}{\gamma(\tau+273.15)+b_1}}_{\text{const}} \tag{11}$$

Accordingly, the final equation for the cell wall yielding Φ *(τ, t)* (with respect to its initial value at t_i for a juvenile cell) in the case of the linear perturbation reads

$$\Phi(\tau,t) - \Phi(\tau,t_i) = \frac{\tau}{\gamma(\tau+273.15)+b_1}\cdot \left(\frac{\phi_0'}{\sqrt{\alpha^2+(\tau-\tau^*)^2}} - \frac{\phi_0\alpha\alpha'}{\left(\alpha^2+(\tau-\tau^*)^2\right)^{3/2}}\right) \tag{12}$$

Eq. (12) is presented in its general form. However, if one wishes to calculate its numerical value, the explicit dependences for the height $\phi_0(t)$ and the half-width $\alpha(t)$ on time are required. In fact, we obtain the set of both coefficients for fixed times from the fitting procedure to the Lorentz-like distribution, Eq. (5), of the elongation data.

Nonlinear Perturbation

Now, let us investigate another case, where p_1 is a constant. Then Eq. (2) changes to

$$\frac{1}{V}\frac{dV}{dt} = \Phi\left(\gamma\frac{T}{V} + p_1\theta(t-t_1)\right) \tag{13}$$

and hence the solution for plant cell volume in the case of nonlinear perturbation reads (for detailed derivation see Appendix A at http://www.springerlink.com):

$$V = \begin{cases} V_0 + \gamma T\xi_t & t<t_1 \\ V_0 e^{p_1(\xi_t - \xi_{t_1})} + \gamma T\xi_{t_1} & t \geq t_1 \end{cases} \tag{14}$$

Now, similar to the preceding two cases, based on the fit of the experimental data to the Lorentz-like distribution, we can draw conclusions about cell wall yielding Φ both through time and dependent on temperature. Hence for $t \geq t_1$,

$$\mathrm{Elong} = V - V_0 = V_0\left(e^{p_1(\xi_t - \xi_{t_1})} - 1\right) + \gamma T\xi_{t_1} \tag{15}$$

On the other hand we recall Eq. (5) and compare it with the above equation. Thus we get

$$V_0\left(e^{p_1(\xi_t-\xi_{t_1})} - 1\right) + \gamma(\tau+273.15)\xi_{t_1} = \frac{\phi_0\tau}{\sqrt{\alpha^2+(\tau-\tau^*)^2}} \tag{16}$$

The value of ξ_{t_1} can be established by the unperturbed solution at point $t = t_1$; hence the solution of Eq. (16) eventually reads

$$\Phi(\tau,t)-\Phi(\tau,t_i)=\frac{1}{p_1}\frac{\tau}{V_0-\gamma(\tau+273.15)\xi_{t_1}+\frac{\phi_0\tau}{\sqrt{\alpha^2+(\tau-\tau^*)^2}}}\cdot\left(\frac{\phi_0'}{\sqrt{\alpha^2+(\tau-\tau^*)^2}}-\frac{\phi_0\alpha\alpha'}{\left(\alpha^2+(\tau-\tau^*)^2\right)^{3/2}}\right) \quad (17)$$

The expression above represents the final form of the relative cell wall extensibility coefficient $\Phi(\tau, t)$ (with respect to its initial value at t_i for a juvenile cell) for the case of nonlinear perturbation.

Experimental and Theoretical Results

Our model predicts two kinds of solutions, namely linear ones and nonlinear ones. Indeed, our experimental data reflect both cases (Figures 1 and 2). In Figure 1 we see the linear response [according to Eq. (10)] of the empirical elongation (with the control subtracted) to the external perturbation (with a stimulator/inhibitor applied only once at time $t = t_1 = 0$ h). Similarly, in Figure 2 we observe a nonlinear response [in accordance with Eq. (15)] of elongation (also here the control has been subtracted) to an external perturbation (stimulator/inhibitor applied first at time $t = t_1 = 1/2$ h, and successively increased at subsequent half-hours).

In Figures 4 and 5 the experimental results are plotted against changing temperature at fixed times. The Lorentz-like curves (Eq. 5) were fitted to each data series with the help of the nonlinear Levenberg–Marquardt algorithm. (The same interpolation method was also used in all subsequent fits.) It is important that for the linear case, as presented in Figure 4, the peak heights ϕ_0 increase linearly with increasing time. In contrast, for the nonlinear case (Figure 5) the peak heights ϕ_0 increase exponentially or logarithmically with increasing times, depending on whether a stimulator or inhibitor was applied. This property is clearly visible, especially for the optimum (peak) temperatures; the distances between the subsequent maxima vary linearly (as for both controls in Figures 4 and 5, or in the linear case Figure 4), and exponentially or logarithmically (stimulator/inhibitor; see Figure 5).

Because the Lorentz-like distribution function is characterized by three parameters: optimum temperature τ^*, curve peak height ϕ_0, and curve half-width α, we determined these values by fitting them to the experimental data (see Figures 4 and 5) for each series. Having acquired the set of coefficients, we made further progress by assuming a special kind of behavior for $\phi_0(t)$ and $\alpha(t)$ in the course of time. That is, in all linear cases (both controls, plus the linear response as in Figure 4) we have fitted linear functions; in the case of the applied stimulator/inhibitor (with increasing concentrations, see Figure 5), we have fitted an exponent or a logarithm, respectively. The optimal temperature was 29.3°C. After establishing the dependences of ϕ_0 and α on time, we insert them into Eq. (6) to find $\Phi(\tau, t)$ in the unperturbed case. Next, if we want to find $\Phi(\tau, t)$ in the linearly perturbed case, we insert $\phi_0(t)$ and $\alpha(t)$ into Eq. (12). Eventually, if we wish to find $\Phi(\tau, t)$ in the nonlinearly perturbed case, we insert them into Eq. (17). The results of numerical computations are presented in the form of 3D plots (see Figures 6, 7, 8, 9, and 10). All these figures

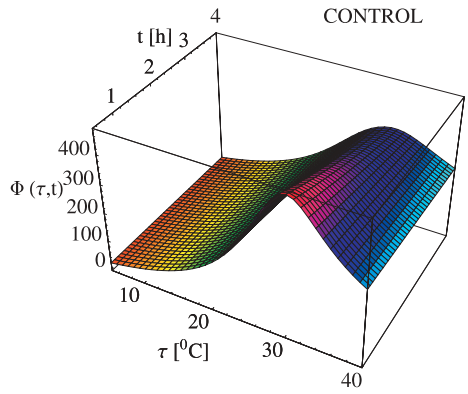


Figure 6. Theoretical results: The calculated cell wall yielding $\Phi[\mu m^3 J^{-1}\cdot h^{-1}]$ as a function of time t and temperature τ for the case of unperturbed growth (control).

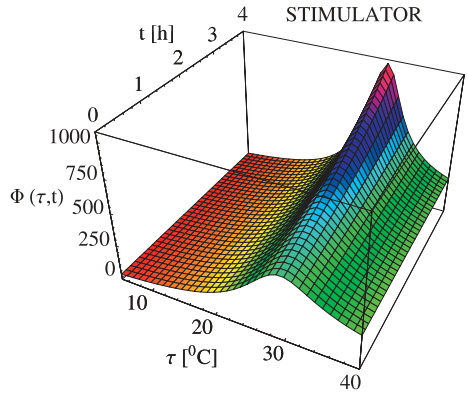


Figure 7. Theoretical results: The calculated cell wall yielding $\Phi[\mu m^3 J^{-1}\cdot h^{-1}]$ as a function of time t and temperature τ for the case of linearly perturbed growth (stimulator).

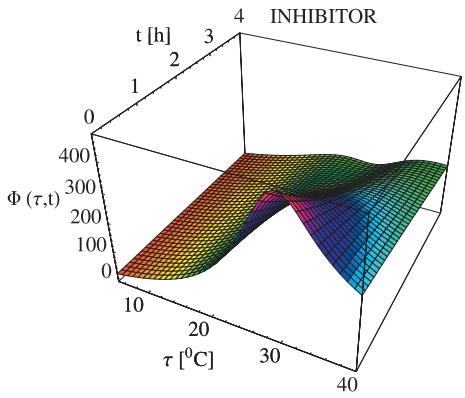


Figure 8. Theoretical results: The calculated cell wall yielding $\Phi[\mu m^3 J^{-1} \cdot h^{-1}]$ as a function of time t and temperature τ for the case of linearly perturbed growth (inhibitor).

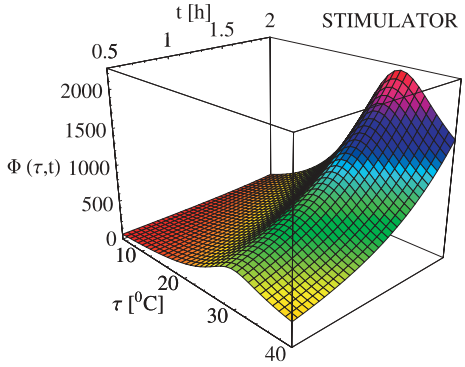


Figure 9. Theoretical results: The calculated cell wall yielding $\Phi[\mu m^3 J^{-1} \cdot h^{-1}]$ as a function of time t and temperature τ for the case of nonlinearly perturbed growth (stimulator). The exponential increase of the crest along the time axis is clearly visible.

present the model-calculated coefficient Φ *(τ, t)* for the cell wall extensibility, as a function of both time and temperature. The unperturbed growth (control, Figure 6) characterizes a weak dumping along the time axis at the crest of the optimum temperature and even lower dumping at both edges at low and high temperatures. Figures 7 and 8 demonstrate the linear response of Φ on the (applied once) stimulator and inhibitor, respectively, whereas Figures 9 and 10 clearly show the theoretically calculated nonlinear (exponential) response to continuously applied stimulator or inhibitor. The pronounced exponential increase manifests itself particularly at the crest and in its close vicinity. An interesting result of the exponential decrease is shown in Figure 10, where a peculiar kind of bifurcation appears. This solution is due to the balance in the last factor of Eq. (17), where if the second term in

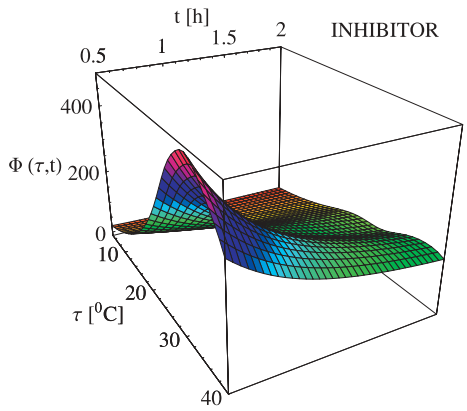


Figure 10. Theoretical results: The calculated cell wall yielding $\Phi[\mu m^3 J^{-1} \cdot h^{-1}]$ as a function of time t and temperature τ for the case of non-linearly perturbed growth (inhibitor). The exponential decay of the crest along the time axis is clearly visible.

parentheses is big enough, the maximum at the crest changes into the minimum at the bifurcation line. This kind of behavior may have an interesting biological interpretation: namely, the growing plant cell wall can choose either means of optimum growth when an increasing concentration of inhibitor is applied. Also, from a practical point of view, the nonlinear solution of Eq. (13) may have a very simple interpretation, as it can report on growth inhibition with increasing pollution of the investigated area (for example, the acid rains falling constantly in a given industrial environment increases, as in our solutions, the concentration of the poisonous factor in the soil). In all cases the constants b_1 (linear response) and p_1 (nonlinear response) have been calculated to obtain continuous solutions at $t = t_1$, where the perturbation is switched on. This is in accordance with the fact that even after application of an intense stimulus, cell wall extensibility should not have steps (discontinuities) over time but should rather be smooth. Figures 11 and 12 present the calculated (see Appendix B at http://www.springerlink.com) radical change of behavior after the application of external perturbations (in a form of stimulator/inhibitor) at time $t = t_1$.

Conclusions

Accepting as a starting point the time-dependent differential Lockhart growth equation, we bring the notion (magnitude) of environmental temperature into the equation. Such a temperature-modified equation of growth not only describes the existing

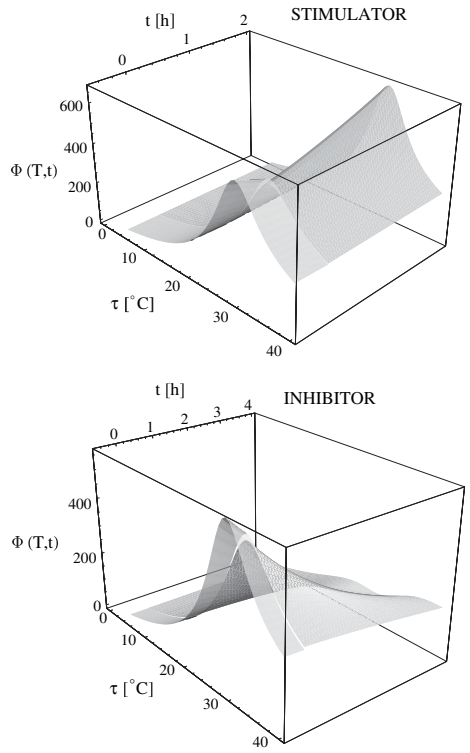


Figure 11. The calculated cell wall yielding $\Phi[\mu m^3 J^{-1} \cdot h^{-1}]$ as a function of time t and temperature τ in the case of linearly perturbed growth (stimulator/inhibitor). A characteristic (theoretically calculated) kink at time point t_1 at which the external perturbation (stimulator/inhibitor) has been applied is clearly visible.

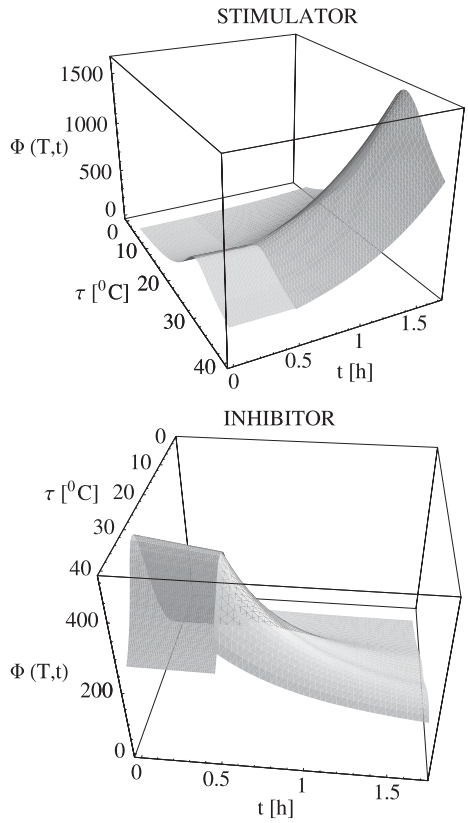


Figure 12. The calculated cell wall yielding $\Phi[\mu m^3 J^{-1} \cdot h^{-1}]$ as a function of time t and temperature τ in the case of nonlinearly perturbed growth (stimulator/inhibitor). A characteristic (theoretically calculated) kink at time point t_1 at which the external perturbation (stimulator/inhibitor) has been applied is visible.

growth data but also allows for the theoretical determination of the optimum temperature for the growth rate. Moreover, by taking into account the temperature-dependent growth experiments, which also include growth stimulators/inhibitors, we perform further investigations and put forward a ''linear/nonlinear response'' theory that incorporates our empirical data and gives a reasonable theoretical description of the action of plant phytohormones and abiotic factors on plant growth rate. One of the most interesting findings of our model is that based on the simple analytical predictions we are able to draw qualitative and, especially, the quantitative conclusions about the cell wall extensibility coefficient $\Phi(\tau, t)$ itself. We stress that the experiments performed by the authors confirmed, with a very high degree of exactness (in all cases the determination coefficients (R^2) exceeding 0.99), the theoretically anticipated solutions and thus strongly support our model equations.

In this article we started with the original Lockhart equation, in which cell wall yielding was studied in a time-irreversible (non-elastic) regime. Our temperature-modified model follows this line and, accordingly, does not take into account cell wall elastic properties at low temperatures. Hence, further improvements to the model should be made to consider both (elastic and non-elastic) components of cell wall extensibility. Indeed, this is the issue that is under investigation. Nevertheless, it seems that the broad applicability of our analytical model to many phenomena relevant to plant cell growth, confirmed by exactness of the fits of experimental data to the theoretical solutions, gives a new tool for investigation of plant cell growth in the aspect of its thermodynamic features.

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Appendix A

Evaluation of the integral I

We wish to integrate the function $\Phi\ (T,\ t)\cdot\theta(t - t_1)$ introduced in Eq. (8). If $t < t_1$ then $I = \int \Phi(T,t)\cdot 0dt$ generally is equal to a constant c_1. We put $c_1 = 0$ as such solution corresponds to the unperturbed case. If $t \geq t_1$, then $I = \int \Phi(T,t)\cdot 1dt = \int \Phi(T,t)dt + c_2 = \xi_t + c_2$. Now, as I is a primordial function (in common sense), it must satisfy the continuity condition $I(t_1^-) = I(t_1^+)$. Hence we get the constant $c_2 = \xi_{t_1}$ and the following expression for I:

$$I \equiv \vartheta\left(\xi_t - \xi_{t_1}\right) = \begin{cases} 0 & \text{for } t < t_1 \\ \xi_t - \xi_{t_1} & \text{for } t \geq t_1 \end{cases} \tag{18}$$

Derivation of Eq. (17)

Wishing to solve the problem of switching on the nonlinear perturbation, we need first to find a solution for the homogeneous differential equation for Eq. (13):

$$\frac{dV}{dt} = \Phi p_1 V\theta(t - t_1) \tag{19}$$

Second, we perform a variation of the constant and finally put the calculated constant into the original (inhomogeneous) equation. From Eq. (19), we get

$$V = V_0 e^{p_1\vartheta(\xi_t - \xi_{t_1})}, \tag{20}$$

where V_0 stands, as usual, for the initial volume of a plant cell. Next, let the constant V_0 vary, compute its time-derivative, and compare such calculated expression with Eq. (13). We finally get the form of ''the constant'' V_0:

$$V_0(t) - V_0(t_0) = \gamma T \underbrace{\int \Phi(T,t) e^{-p_1\vartheta(\xi_t - \xi_{t_1})} dt}_{I_1} \tag{21}$$

Now, let us find the analytical form of the integral I_1. If $t < t_1$ then $I_1 = \int \Phi(T,t)\cdot \mathrm{ldt} = \xi_t$. If $t \geq t_1$ then

$$\begin{aligned} I_1 &= \int \Phi(T,t)\exp\left(-p_1\cdot\left(\xi_t - \xi_{t_1}\right)\right)dt \\ &= -\frac{1}{p_1} e^{-p_1\cdot\left(\xi_t - \xi_{t_1}\right)} + c \end{aligned} \tag{22}$$

The constant c in the above equation may be determined from the continuity condition for I_1 integral: $I_1(t_1^+) = -1/p_1 + c$. Thus $c = 1/p_1 + \xi_{t_1}$ and

$$I_1 = \begin{cases} \xi_t & \text{for } t > t_1 \\ -\frac{1}{p_1}\mathrm{e}^{-p_1\left(\xi_t - \xi_{t_1}\right)} + \frac{1}{p_1} + \xi_{t_1} & \text{for } t \geq t_1 \end{cases} \tag{23}$$

Putting the obtained form of I_1 into Eq. (21) leads us to the final expression for the volume V:

$$V = \begin{cases} V_0 + \gamma T\xi_t & t < t_1 \\ V_0 e^{p_1(\xi_t - \xi_{t_1})} + \gamma T\xi_{t_1} & t \geq t_1 \end{cases}, \tag{24}$$

which we have been seeking.

Appendix B

Having obtained expressions for the cell wall yielding coefficient $\Phi\ (\tau,\ t)$ for all cases considered in the article, we may now estimate the b_1 and p_1 coefficients bound with the action of phytohormones or abiotic factors. Nature gives us many reasons to assume that cell wall yielding should be a continuous function of time and temperature despite the application of perturbations. (Infinite changes in magnitudes do not exist in descriptions of real biological and physical systems). The continuity condition provides a method for solving for b_1 in the linear case (now denoted as LC) and p_1 in the nonlinear case (NC):

$$[\Phi(\tau,t_1) - \Phi(\tau,t_i)]_{UC} = [\Phi(\tau,t_1) - \Phi(\tau,t_i)]_{LC} \tag{25}$$

The temperature τ is fixed, and we denote the unperturbed case as UC. Many calculations of Eq. (25) lead us to the equation

$$\begin{aligned} &\frac{B^2(\tau)\left[\Phi_0'(t_1)\right]_{UC} - A[\alpha,(t_1)]_{UC}}{B^2(\tau)\left[\Phi_0'(t_1)\right]_{LC} - A[\alpha,(t_1)]_{LC}} \\ &= \frac{\gamma(\tau + 273.15)}{\gamma(\tau + 273.15) + b_1} \Rightarrow b_1(\tau) \end{aligned} \tag{26}$$

from which we are able to calculate $b_1\ (\tau)$ numerically for each fixed temperature τ. We have denoted

$$\begin{aligned} B(\tau) &= \sqrt{[\alpha^2(t_1)]_{UC} + (\tau - \tau^*)^2} \\ &= \sqrt{[\alpha^2(t_1)]_{LC} + (\tau - \tau^*)^2} \end{aligned}$$

and

$$A = [\phi_0(t_1)]_{UC}\cdot\left[\alpha^2(t_1)\right]_{UC} = [\phi_0(t_1)]_{LC}\cdot\left[\alpha^2(t_1)\right]_{LC}$$

The same reasoning leads to the analogous derivations for $p_1(\tau)$ in the nonlinear case, where we start from the following continuity condition:

$$[\Phi(\tau, t_1) - \Phi(\tau, t_i)]_{UC} = [\Phi(\tau, t_1) - \Phi(\tau, t_i)]_{NC} \qquad (27)$$

Again, tedious calculations lead us to

$$\frac{\gamma(\tau + 273.15)}{p_1 \cdot V_0} = \frac{\left[\phi_0'(t_1)_{UC}\right] \cdot b^2(\tau) - a \cdot [\alpha\prime(t_1)]_{UC}}{\left[\phi_0'(t_1)_{NC}\right] \cdot b^2(\tau) - a \cdot [\alpha\prime(t_1)]_{NC}} \Rightarrow p_1(\tau) \qquad (28)$$

From Eq. (28) we may numerically calculate the value of p_1 for each fixed temperature. The parameter a in the above equation expressed by the product

$$[\phi_0(t_1)]_{UC} \cdot [\alpha(t_1)]_{UC} = [\phi_0(t_1)]_{NC} \cdot [\alpha(t_1)]_{NC}$$

is constant. In contrary,

$$b = b(\tau) = \sqrt{[\alpha^2(t_1)]_{UC} + (\tau - \tau*)^2} = \sqrt{[\alpha^2(t_1)]_{NC} + (\tau - \tau*)^2}$$

depends on temperature, and this fact leads us to conclusion that p_1 also must depend on τ.

After performing these calculations, we have fitted the appropriate functions and inserted them into the expressions for the cell wall yielding $\phi\ (\tau, t)$ in all cases. The final step was to plot $\phi(\tau, t)$ throughout the whole time range, including time t_1 when the perturbation was applied. It is very interesting that all predictions about the behavior of b_1 or p_1 in the course of time have been fulfilled. We expected that absolute values of $b_1(\tau)$ and $p_1(\tau)$ should increase linearly or exponentially in time (the explanation is obvious: growth regulators are chemical compounds that act more intensely as the temperature rises; the thermal vibrations intensify).

Indeed, having obtained the set of calculated values b_1 in the linear case and p_1 in the nonlinear case we have fitted linear functions for auxin ($b_1 > 0$) or $CdCl_2$ ($b_1 > 0$), applied once, and exponents for auxin or $CdCl_2$, applied with increasing concentrations ($p_1 > 0$ or $p_1 < 0$, respectively).