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Photocontrol of Extension Growth: A Biophysical Approach [and Discussion]

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Photocontrol of extension growth: a biophysical approach

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The analysis of plant growth as a physical process is briefly reviewed. Growth requires the coordinated uptake of water and the irreversible expansion of the cell wall. Any agent that affects the growth rate must act on one or more of the parameters governing water absorption (e.g. the hydraulic conductivity or the difference in osmotic pressure of the cell contents and the water source) or cell wall expansion (e.g. wall extensibility or the yield threshold). When the hydraulic conductivity of the pathway for water transport is small enough to impede the rate of cell enlargement, a substantial gradient in water potential within the growing tissue will develop to sustain the absorption of water. In such a case, the analysis shows that turgor pressure is a key indicator for determining whether an agent acts predominantly on the osmotic properties of the tissue or on the cell wall properties. Furthermore, the dynamic response to a slight perturbation from steady-state conditions is shown to be a function of parameters for both the water relations and cell wall expansion of the tissue.

Blue irradiation of etiolated seedlings causes a large inhibition of stem elongation with lag times as short as 30 s and half-times as short as 20 to 25 s. The biophysical mechanism of blue-light suppression of growth was studied in cucumber and sunflower seedlings by means of direct and indirect measurements of turgor pressure. The results indicate that (*a*) blue light suppresses growth by influencing the cell wall properties of the growing tissue, and (*b*) the hydraulic conductivity of the growing tissue is large enough for it not to limit the rate of cell enlargement.

Introduction

The transition of a plant from dark to light conditions is followed by a number of pronounced changes in the development of the plant. Leaves expand very little in the dark, but upon onset of irradiation they unfold and expand to their full size. Just the opposite action occurs in stems where the high rate of elongation in etiolated seedlings is greatly inhibited by light. These morphogenetic developments are accompanied by many other changes at the cell, organelle, and biochemical levels. In this report I wish to focus on efforts to study the mechanism by which light alters the growth rate of shoots, with particular emphasis on the physical approach to this problem.

The effect of light on shoot growth has been the subject of numerous studies. Since the discovery of auxin and other growth regulators, many investigations have dealt with the hypothesis that light alters growth by the mediation of one or more of the plant growth regulator systems (Black & Vlitos 1972). It is indeed clear by now that light has effects on the levels of endogenous auxin and gibberellins (Briggs 1963; Iino 1982; Murray & Acton 1974), on the levels of growth antagonists (Franssen & Bruinsma 1982), on the transport of hormones through plant tissues (Pickard & Thimann 1964; Sherwin & Furuya 1973; Thornton & Thimann 1967), on the number of auxin receptor sites (Walton & Ray 1981) and on the sensitivity of tissues to exogenously applied hormones (Kende & Lang 1964; Russel & Galston

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1969). What is not clear is to what degree these various biochemical responses are incidental to the change in growth rate or fully account for the light growth response. Indeed, there is disagreement about whether endogenous hormone levels normally exert a controlling influence on the growth rate of a plant tissue (Trewavas 1981). It is a fundamental problem with this experimental approach that it is difficult to quantitatively relate a particular biochemical change induced by irradiation to the growth response. As an example, some workers dispute whether the amount of auxin redistribution during phototropism of corn coleoptiles is sufficient to account for the observed change in growth rate (Firn & Digby 1980; Hall *et al.* 1980). One important reason for this controversy is that auxin itself does not directly control growth; rather, it acts on one or more cellular parameters that govern the growth rate.

A way around this dilemma is to investigate growth from a biophysical perspective, in which the physical parameters directly responsible for growth are measured. This approach offers the possibility of quantitatively accounting for the entire growth response in terms of changes in four biophysical parameters that govern the growth rate of a tissue.

Plant growth as a physical process

The growth of plant organs entails the irreversible enlargement of cells. Cleland & Rayle (1977) have calculated that if the stem cells of a redwood tree simply duplicated themselves without undergoing the usual phase of prolonged cell enlargement, the tallest redwood would stand less than two feet in height! For irreversible cell enlargement to occur, two fundamental processes are required. The cells must take up water to generate the additional cell volume, in that growing cells are more than 90% water; and the cell wall surrounding the protoplast must irreversibly expand to accommodate the absorbed water. Conceptually, water uptake and irreversible wall expansion are distinct processes, but in growing tissues they are tightly linked with one another. Each process will be considered separately below, and then their interdependence will be discussed.

Water absorption during plant growth

As far as has been established, water transport is a passive physical process that (with certain important restrictions) occurs down a gradient in chemical potential, or equivalently, water potential. The rate of water uptake by a cell, given any specific water potential difference between its contents and the environment, depends on the hydraulic conductivity of the pathway for water transport (L), such that

$$\mathrm{d}V_\mathrm{w}/\mathrm{d}t = L(\Delta\psi) = L(P - \Delta\pi), \tag{1}$$

where $\mathrm{d}V_\mathrm{w}/\mathrm{d}t$ is the rate of water uptake, $\Delta\psi$ is the difference in water potential between the cell and the water source, P is the turgor pressure of the cell, and $\Delta\pi$ is the difference in osmotic pressure between the cell contents and the water source. To a first approximation, $\mathrm{d}V_\mathrm{w}/\mathrm{d}t$ is equal to the growth rate of the cell (assuming zero transpiration). Equation (1) assumes an ideal semipermeable membrane.

When growth of multicellular tissues is considered, the mathematical treatment of water transport becomes rather more complicated (Molz & Boyer 1978; Molz & Ferrier 1982). The hydraulic conductivity of both the cell wall pathway and the cell-to-cell pathway for water transport must be considered (figure 1). Providing that conditions of 'local equilibrium' obtain

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(i.e. water in the cell wall space is in equilibrium with water in the nearest cells), water transport will have diffusion-type kinetics. In such a case, there will develop within a growing tissue a gradient in water potential that is induced by the expansion of the cell wall and that at the same time sustains the absorption of water by the growing cells. Models of elongation in a cylindrical piece of tissue such as a stem predict that the water potential gradient would be

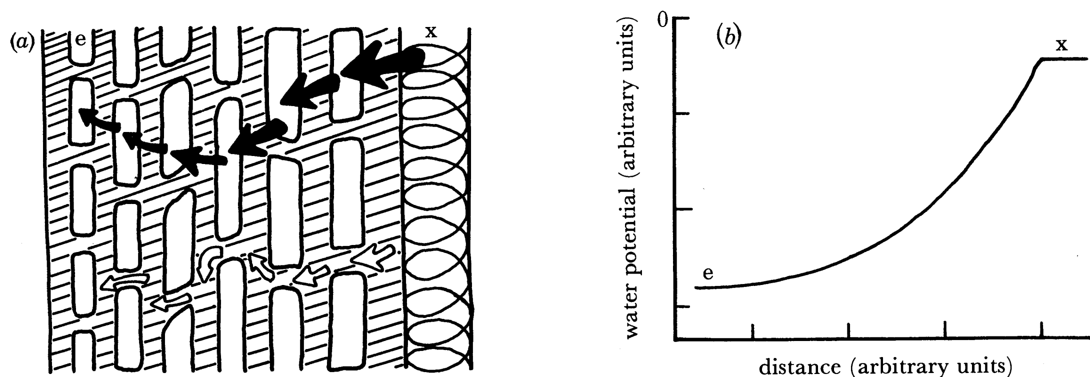

FIGURE 1. (*a*) Diagram showing the pathway for water flow during the growth of a multicellular tissue. Water may move from the xylem (x) toward the epidermis (e) via the cell-to-cell pathway (solid arrows) and the cell wall pathway (open arrows). (*b*) Theoretical water potential profile in a growing cylinder of tissue such as a stem. The profile is drawn radially from the xylem (right side) to the epidermis (left side). The size of the gradient depends on the growth rate of the tissue and the diffusivity of the tissue for water flow. (Redrawn after Molz & Boyer (1978).)

steep near the water source (the xylem) and flatter in the regions further from the xylem (see figure 1). The cells closest to the xylem must transport the water that is absorbed during their own growth as well as that absorbed by cells lying further from the centre. For this reason the gradient in water potential is steeper in this region to support the higher rate of water flow. Toward the outside of the tissue, the gradient is flatter because less water is transported.

Cell wall expansion during plant growth

In non-growing plant tissue, cells out of osmotic equilibrium would absorb water and consequently increase in turgor pressure until $\Delta\psi$ became zero (i.e. $P = \Delta\pi$), at which point net water flow would stop. However, in a growing cell, the cell walls are loosened in some fashion and the cell wall yields to the stress generated by the pressure of the cell contents. This irreversible stretching or deformation of the cell wall dissipates P (because water has a low compressibility) and hence sustains the gradient in water potential necessary for water absorption. Thus growth begins with stress relaxation of the cell wall (i.e. wall loosening), which concommittantly brings about relaxation of the turgor pressure and a lowering of the water potential of the cells. The depressed water potential permits uptake of water, which generates additional cell volume and expands the cell wall. If stress relaxation of the cell wall is stopped, then water absorption will cause the stress in the cell wall and its opposing force (turgor pressure) to increase until osmotic equilibrium is re-established.

Wall expansion is thought to be a rheological process in which the cell wall is physically distended by the forces in the wall generated by turgor pressure. A number of studies (Lockhart 1965; Cleland 1967; Green *et al.* 1971) have suggested that the rate of cell-wall expansion is

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proportional to the amount by which turgor pressure exceeds a certain minimum value, such that

$$\mathrm{d}V_{\mathrm{cw}}/\mathrm{d}t = m(P-Y), \quad (2)$$

where Y is the minimum turgor pressure necessary for growth (the yield threshold), m is a parameter termed the cell wall extensibility, and $\mathrm{d}V_{\mathrm{cw}}/\mathrm{d}t$ is the rate of expansion of cell wall, expressed in volumetric units. It should be emphasized here that the term 'cell wall extensibility' as used in this context does not refer to the elastic extensibility measured by various mechanical means, but refers to the coefficient relating the rate of irreversible cell wall expansion to the 'effective' turgor pressure $(P-Y)$. Equation (2) is an empirically derived relation and is somewhat deceptive in that m and Y are not simple physical parameters describing time-independent rheological properties of the cell wall (Green *et al.* 1971; Green & Cummins 1974; Ray & Ruesink 1962). Rather, they seem to depend on the rate of cellular or biochemical processes that loosen the cell wall and are coupled to the respiration rate (i.e. they are energy dependent). The nature of the wall-loosening and subsequent expansion processes have yet to be established and the physical meanings of m and Y are not clear, except as operationally defined in (2). It is probable that they represent the result of a complex cellular process and it thus may be misleading to represent this process in such a simple fashion. Further insight into the physical and biochemical nature of wall extension and its relation to turgor pressure is vitally needed.

In contrast, the water transport equation has a strong basis in the theory of irreversible thermodynamics and is well supported experimentally (Molz & Ferrier 1982; Dainty 1976). The parameters of (1) are well defined and may be measured in a number of independent ways. Of course, the values of L and $\Delta\pi$ depend upon the maintenance of membrane integrity and solute accumulation, which are uphill processes energetically and thus in the long term also depend on metabolic energy, although only indirectly.

Predictions of the physical theory

Under conditions of steady-state growth, the rates of water uptake and irreversible wall expansion are assumed to be equal to each other and we may combine (1) and (2) to obtain the general equation governing the rate of plant growth $(\mathrm{d}V/\mathrm{d}t)$:

$$\mathrm{d}V/\mathrm{d}t = Lm(\Delta\pi - Y)/(L+m). \quad (3)$$

The growth rate $(\mathrm{d}V/\mathrm{d}t)$ thus depends on the parameters controlling both water transport and irreversible wall expansion. These two processes are linked by the common dependent variable, turgor pressure, which is eliminated to obtain (3). Although turgor pressure does not explicitly appear in (3), an understanding of its role in coordinating wall expansion and water absorption is crucial for an understanding of cell expansive growth.

If water transport is a very rapid process in comparison with cell wall expansion (more specifically, if the hydraulic conductivity is much greater than the wall extensibility), the turgor pressure (and water potential) in a growing cell is negligibly affected by the expansion of the cell wall (Cosgrove 1981*a*). Water can enter the cell quickly enough to maintain the turgor pressure (almost) at the equilibrium value $(\Delta\pi)$. Consequently the hydraulic conductivity cannot exert a controlling influence on the rate of growth; the yielding properties of the cell wall are then of paramount importance. On the other hand, if water uptake is a slow process, wall expansion may occur at a sufficiently rapid rate to dissipate turgor pressure significantly

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(figure 2). The lower turgor pressure in turn decreases the rate of wall expansion (equation (2)) and the hydraulic conductivity in this case may exert a significant control on the growth rate by influencing steady-state turgor pressure.

As indicated by this analysis, the process of wall expansion tends to decrease turgor pressure, whereas the process of water uptake tends to increase it. In principle, the turgor pressure of

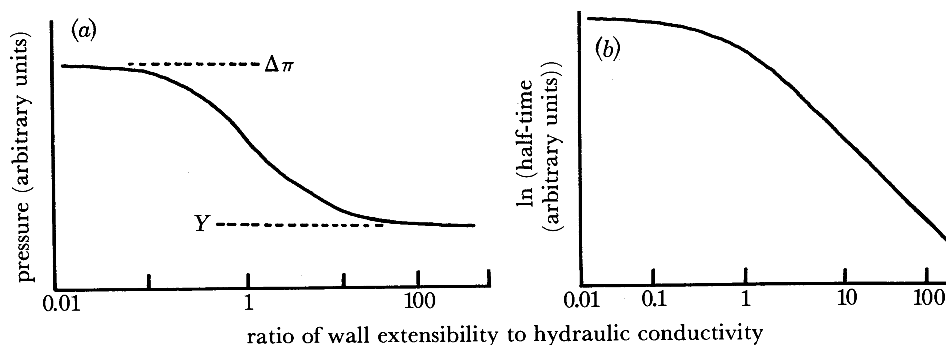

FIGURE 2. (*a*) Turgor pressure as a function of the ratio m/L. When wall extensibility (m) is much smaller than hydraulic conductivity (L), the tissue is essentially at osmotic equilibrium and $P = \Delta\pi$. At the other extreme, turgor pressure is dissipated by rapid expansion of the cell wall, so that $P = Y$. Within the range 0.1–10, the turgor pressure is determined by the balance point between water uptake and wall expansion. (*b*) Half-time of tissue as a function of the ratio m/L. Only when the value of wall extensibility approaches or exceeds the value of hydraulic conductivity is the half-time affected by the cell wall properties. Note the logarithmic scales. (After Cosgrove (1981*a*).)

a growing cell must be less than that of an equivalent non-growing cell; it must lie somewhere in the range between $\Delta\pi$ and Y (see figure 2), the exact value being determined by the balance point between wall expansion and water uptake (Cosgrove 1981*a*). In single isolated cells, water transport is so rapid that the cell is essentially in osmotic equilibrium with its bathing solution (Cosgrove 1981*a*). In multicellular tissue, however, it is possible for the hydraulic conductivity to decrease sufficiently (because of the long pathway for transport) for the cell layers remote from the water source to be far from osmotic equilibrium (Molz & Boyer 1978). Indeed, several reports support this view in that they show that growing tissues of various plant organs have low water potentials (Ray & Ruesink 1963; Boyer 1968; Molz & Boyer 1978; Michelena & Boyer 1982).

In such tissues we can predict that if an agent inhibits growth, it ought to change the steady-state balance point of turgor pressure upwards or downwards (toward $\Delta\pi$ or Y), depending on whether it acts primarily on the water transport characteristics of the tissue (L, $\Delta\pi$) or on the yielding properties of the cell wall (m, Y). Turgor pressure in principle should increase if the agent acts on the cell wall properties, but decrease if the agent acts on the osmotic properties (Cosgrove 1981*a*). The *amount* by which turgor pressure changes depends on the size of the span between $\Delta\pi$ and Y, and whether the balance point is already very close to $\Delta\pi$ or Y (see figure 2). For example, if the growth rate were controlled entirely by the wall properties (i.e. if hydraulic conductivity were much larger than wall extensibility), the change in turgor pressure brought about by a change in wall extensibility would be very small. Turgor pressure would be most responsive when wall extensibility and hydraulic conductivity were equal in magnitude.

A second prediction that may be derived from the physical analysis of plant growth concerns

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the time course for re-establishment of the steady state after a slight perturbation (Cosgrove 1981 *a*). Consider first a non-growing cell. If the cell is brought out of osmotic equilibrium with an external solution (either by changing the water potential of the solution or the water potential of the cell contents), water will be induced to flow across the cell membranes until equilibrium is re-established. The rate of water flow will be large at first and will decrease exponentially as the equilibrium point is reached. The half-time for the transient flow of water is determined entirely by the physical parameters governing water transport (the hydraulic conductivity, the osmotic pressure of the cell contents, the volumetric elastic modulus of the cell, and the cell geometry) (see Dainty 1976; Zimmermann & Steudle 1978; Molz & Ferrier 1982).

Now consider a growing cell. The water potential of the cell is affected not only by the process of water transport, but also by the process of cell wall expansion. If wall expansion is slow (such that the growing cell is close to osmotic equilibrium), the half-time for the re-establishment of steady state after a perturbation is hardly affected by the wall expansion process; the half-time will be the same as in a non-growing cell. If wall expansion is more rapid, i.e. if wall extensibility is similar to or larger than hydraulic conductivity, the half-time will be much faster for the growing cell (see figure 2). This half-time will be governed not only by the water relations parameters, but also the parameters controlling wall expansion, as shown by the analysis of Cosgrove (1981 *a*).

These two predictions based on the physical theory of plant growth have been used to investigate the inhibition of elongation growth by blue irradiation.

Case study: blue light inhibition of growth

Irradiation of dark-grown dicotyledonous seedlings with blue light induces a very rapid decrease in the elongation rate of the stem (Meijer 1968; Gaba & Black 1979). The latent period between the start of irradiation and the start of the growth inhibition is as short as 20 s in some species; a more typical value is about 60 s (Cosgrove 1981 *b*). A large response (say 25–75% inhibition) requires that the growing tissue itself be irradiated with a high fluence rate of blue light (1–5 W m^{-2} are typical values; see Cosgrove (1981 *b*) and Cosgrove & Green (1981)). It has been shown from a number of criteria that this blue-light response is mediated by a specific blue light photoreceptor, distinct from phytochrome (Cosgrove 1982; Gaba & Black 1979; Holmes & Schafer 1981; Thomas & Dickinson 1979).

The interesting aspect of this light response, from a growth physiologist's point of view, is that it occurs so rapidly. In certain species the growth inhibition has approximately the form of an exponential decay in the growth rate (see figure 3; see also Cosgrove (1981 *b*) and Cosgrove & Green 1981). In cucumber the half-time for the inhibition is between 15 and 30 s. Thus the full growth inhibition is complete in less than 5 min after the start of the irradiation. This is an important advantage for studying the mechanism of this growth response, in that little time is available for secondary responses to complicate the investigation. In contrast, red-light induced inhibitions of growth generally have longer lag times (Meijer 1968; Morgan *et al.* 1980; Vanderhoef & Briggs 1978) and probably involve multiple response mechanisms (Iino 1982).

The theoretical model of plant growth discussed above ignored the fact that transpirational water loss may also influence growth. For example, if blue light stimulated the opening of

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stomata in the epidermis of the stem or leaves, this might increase transpiration enough to cause the turgor pressure, and consequently the growth rate, to fall. This mechanism must be considered because blue light has been shown to stimulate stomatal opening in leaves (Zieger *et al.* 1981). To test this hypothesis, cucumber seedlings were mounted in a chamber that permitted the growing region of the stem to be submerged in aerated water. The growth rate

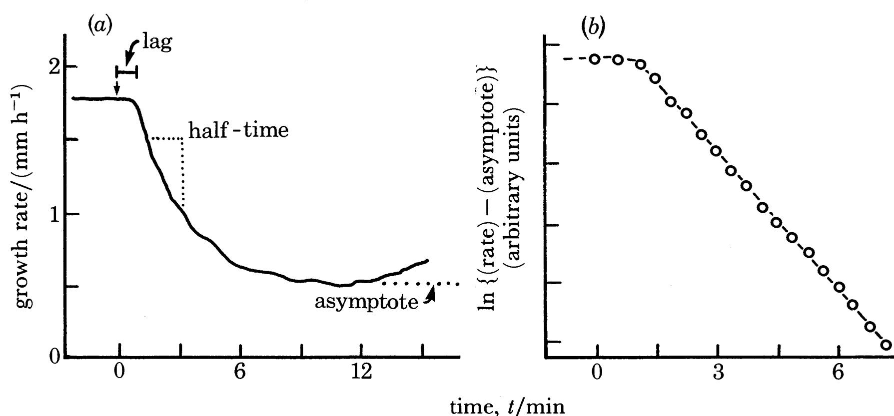

FIGURE 3. Time course of growth inhibition by blue light. (*a*) At the point indicated by the arrow, a dark-grown sunflower seedling was irradiated with 16 s of blue light (2.6 W m^{-2}). After a lag of *ca.* 1 min, the growth rate declined in an approximately exponential fashion to a low value (asymptote). (*b*) The half-time of the inhibition may be calculated from the slope of the data replotted as ln (rate minus asymptotic rate) against time. (Redrawn from Cosgrove & Green (1981).)

was measured continuously with a displacement transducer as described elsewhere (Cosgrove 1982). When a stable growth rate in the dark was attained, the seedling was irradiated with blue light (3 W m^{-2}). If the hypothesis of stomatal opening were true, blue light should have had no effect on the growth rate of such submerged seedlings, where transpiration was essentially eliminated. In fact, a typical growth inhibition was observed (data not shown). Thus blue light must act by altering one or more of the parameters in (3), e.g. $\Delta\pi$, L, m or Y.

A possible change in the concentration of intracellular solutes by blue irradiation was examined by measuring the osmolality of expressed cell sap from the growing region of cucumber seedlings exposed to blue light (2 W m^{-2}) for various lengths of time. As shown in figure 4, no effect of blue light was observed; blue light does not cause changes in bulk π.

In view of the rapid exponential kinetics of the light-growth response, it is possible that blue light causes the plasmalemma to become 'leaky' to intracellular solutes. In such a case, solutes would build up in the cell-wall free space, decreasing the gradient in osmotic pressure across the cell membrane and consequently decreasing turgor pressure and growth rate. Because free space water constitutes less than 5% of the total volume of the tissue (Cosgrove & Cleland 1983*a*), such leakage might involve the leakage of only a very small proportion of the intracellular solutes. Furthermore, cell sap expressed from stem tissue represents a volume-weighted average of the combined intracellular and extracellular solutions, so that a redistribution of solutes within the tissue would not be detected by bulk π measurements in any case. Such a redistribution, however, would be detected by turgor pressure measurements.

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Turgor pressure measurements

As pointed out in the theory section, turgor pressure is a key indicator for the mechanism by which an agent affects the plant growth. The analysis predicts a decrease in turgor pressure if blue light acts on the osmotic properties of the tissue, an increase if blue light acts on the cell wall. This prediction has been tested now by two different methods with similar results.

Qualitative changes in turgor pressure during the inhibition of stem elongation by blue light were first measured by using an indirect technique that measures the mechanical rigidity of

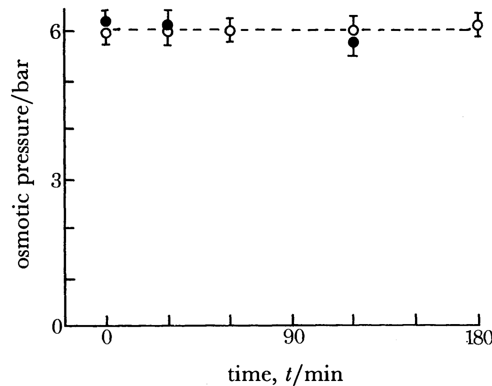

FIGURE 4. Osmotic pressure of cell sap expressed from 1 cm sections cut from the growing stem region of cucumber seedlings irradiated with blue light (2 W m^{-2}) for various length of time. Open circles are data from irradiated plants, filled circles from dark controls. Points plotted are means ± s.d. of 10 samples. (1 bar = 10^5 Pa.)

plant tissue (Virgin 1955). An intact stem was firmly mounted in the middle of the growing region and forced to vibrate at its resonance frequency. The more rigid the stem (i.e. the greater the elastic modulus), the higher is the resonance frequency. The cell walls of young growing tissue are very soft and weak; most of the stiffness of the tissue results from the hydrostatic pressure (turgor pressure) of the cell contents, which puts the cell wall in tension (Falk *et al.* 1958; Nilsson *et al.* 1958). In effect, such plant tissues have a hydraulic skeleton or support structure (Wainwright 1970). The rigidity of such tissue is thus largely a function of the turgor pressure of the cells. Changes in turgor pressure are reflected in changes in the resonance frequency of the stem (Virgin 1955; Nilsson *et al.* 1958).

Cucumber and sunflower seedlings were connected under dim green light to an apparatus which permitted simultaneous measurements of the growth rate and the resonance frequency of the elongating stem (Cosgrove & Green 1981). When growth was suppressed by blue light, the rigidity of the stem, measured by the resonance method, increased very slightly (figure 5). This resonance method is useful for indicating the *direction* of change in turgor pressure, but has several weaknesses. It is not very quantitative, it is sensitive to the geometry of the tissue, and it may be sensitive to changes in the mechanical properties of the cell wall (Nilsson *et al.* 1958; Uhrstrom 1969). Despite these limitations, we estimated that when the growth rate was inhibited 50% by blue irradiation, the turgor pressure increased by less than 0.2 bar (2×10^4 Pa). That is, it hardly changed at all.

Recently these results were confirmed by using the pressure probe technique (Hüsken *et al.* 1978). In these experiments, the turgor pressure of an individual cell in the outer cortical region

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of the stem of an intact cucumber seedling was monitored at the same time that the growth rate of the whole stem was measured. When stem elongation was inhibited by blue irradiation, no change in turgor pressure was detected (Cosgrove, in preparation). The practical resolution of the pressure probe method is approximately 0.1 bar (10^4 Pa).

These results admit two interpretations. First, it could be that blue light causes a simultaneous and proportional change in both the osmotic and the cell wall properties of the tissue. Thus

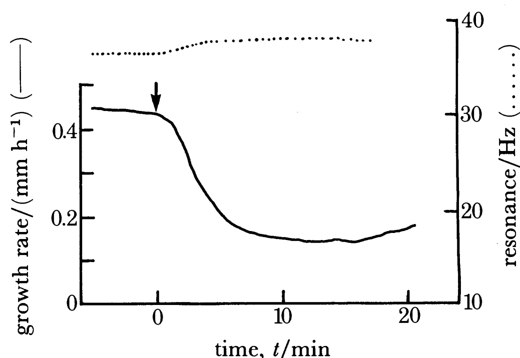

FIGURE 5. Changes in growth rate and resonance frequency in a sunflower seedling in response to a single 16 s pulse of blue light (4 W m^{-2}). The growth rate (solid line) is strongly inhibited whereas the resonance frequency of the stem (broken line) increases only very slightly. The arrow indicates when the light was given.

TABLE 1. EFFECT OF LIGHT DOSE ON GROWTH INHIBITION IN SUNFLOWER SEEDLINGS

(The growth response of seedlings to a 16 s pulse of blue light was broken down into three parameters that completely describe the response. Significance was tested by two-way analysis of variance (from Cosgrove & Green 1981).)

light dose / J m^{-2}	mean asymptote† (percentage of dark rate)	mean half-time‡ / s	mean lag time‡ / s
144	29	141	62
40	36.5	160	55
8	45	136	65

† Significant effect of light dose at the 0.001 probability level.
‡ No significant effect.

the balance point between wall expansion and water uptake would be unaltered and the turgor pressure would remain unchanged. It would be a remarkable coincidence indeed if such different processes were affected in such a fashion. However, this hypothesis may be rejected for the following reason. The half-time of inhibition appears to be identical with the half-time for readjustment in growth rate after perturbation (Cosgrove & Green 1981). If blue light decreased both the wall extensibility and the hydraulic conductivity of the growing tissue, the half-time of the growth response would be strongly affected. However, the half-time was experimentally found to be independent of the degree of inhibition and the light dose (table 1).

The second interpretation of the turgor pressure data is that the hydraulic conductivity of the tissue is so large that it does not limit growth. In such a case the gradient in water potential that sustains growth is insignificant and the turgor pressure of the growing tissue is only negligibly lower than in an equivalent non-growing tissue. Likewise, the half-time of the tissue is unaffected by wall expansion. At present this interpretation seems most in agreement with the results of the experiments.

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These data indirectly support the hypothesis that blue light acts on the cell wall properties to inhibit growth. This suggests that the yielding properties of the wall (m or Y) are under remarkably tight control by cellular processes. Two observations make it unlikely that the blue light effect is mediated by auxin. First, the lag time of the light response is as short as 20 s whereas the lag time for auxin stimulation of growth is 10–15 min. The latent period with auxin treatment evidently does not represent the time required for auxin uptake but rather the time required for the intermediate steps between auxin uptake and the change in the cell-wall yielding properties (Ray 1974). Although direct data are lacking, it is reasonable to expect that if we could somehow cause an instantaneous decrease in auxin concentration, transport or sensitivity, a minimum lag of 10–15 min would follow before a decrease in the growth rate would be seen. Thus the large disparity between the lag time for response to blue irradiation and auxin treatment (20 s compared with 10–15 min) argues against involvement of auxin in the rapid light response. Second, it has been shown by other investigators (Uhrström 1969; Burström *et al.* 1967; Göring *et al.* 1975) and confirmed in this laboratory that auxin induces a rather large decrease in the rigidity of the growing tissue (measured by the resonance frequency method). In principle, this change in rigidity might be due to a loss of turgor pressure or an *elastic* 'loosening' of the cell wall. Direct measurements with the pressure probe technique show that auxin does not affect turgor pressure over this short period (Cosgrove & Cleland 1983*b*); therefore the decrease in tissue rigidity upon auxin treatment must be an effect on the mechanical characteristics of the cell wall. In contrast, blue light has only a negligible effect on the rigidity of the growing tissue. These observations taken collectively suggest that auxin and blue light affect the cell wall by different mechanisms.

The observation that rapid and large suppression of elongation by blue light has only a negligible effect on the turgor pressure of the growing tissue deserves some comment. Several studies (Ray & Ruesink 1963; Boyer 1968; Molz & Boyer 1978; Michelena & Boyer 1982) have shown that growing tissues have water potentials in the range of -2 to -5 bar (-2 to -5×10^5 Pa). These low water potentials have been thought to result from the process of cell-wall expansion driving the tissue far out of osmotic equilibrium. When the water potential of growing cucumber stem tissue was estimated from measurements of turgor pressure and osmotic pressure, water potential values of -3 to -4 bar (-3 to -4×10^5 Pa) were obtained (Cosgrove, in prep.). This observation seems to be at variance with the conclusion reached above from the blue-light studies that the water potential gradient that supports growth in cucumber stems is small. However, Cosgrove & Cleland (1983*a*) have recently found that the cell-wall free space of growing stem tissues contains solute concentrations high enough to account for most of this low water potential. This finding resolves the apparent paradox posed by the conclusion that hydraulic conductivity does not limit growth and observations of low water potentials in growing tissue. This point is discussed further by Cosgrove & Cleland (1983*a*, *b*).

The process of cell-wall loosening is poorly understood. It must involve the breakage and perhaps reformation of load-bearing bonds within the cell wall. The nature of these critical bonds has not been established, nor is it clear whether the wall may be loosened in more than one way. The acid growth hypothesis for auxin action (see Cleland & Rayle 1977) proposes that the pH of the cell wall is a major control point for cell wall expansion. But this hypothesis is far from a complete or even sufficient model of cell wall expansion and cannot explain growth induced by cytokinin and gibberellin in at least some tissues (Ross & Rayle 1982; Stuart & Jones 1978). This is an area that requires further experimental investigation.

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Conclusion

The analysis of plant growth as a physical process offers a powerful framework within which the mechanism of action of any agent that alters the growth rate may be investigated. Growth is shown to be controlled by two parameters governing water uptake (hydraulic conductivity and the osmotic pressure difference between the cell contents and the water source) and two operationally defined parameters describing the yielding characteristics of the cell wall (wall extensibility and yield threshold). The analysis shows that turgor pressure is a key indicator for the mechanism by which an agent affects growth. Kinetic analysis of the re-establishment of the steady state after a perturbation in the growth rate of a tissue is another useful experimental technique.

The rapid inhibition of stem elongation by blue light was investigated within this framework. The experimental results support the conclusions that (*a*) blue irradiation suppresses growth by acting on the yielding characteristics of the cell wall, and (*b*) the hydraulic conductivity of growing cucumber stems is sufficiently large for it not to impede the rate of cell enlargement. Further research into the mechanism of stress relaxation (loosening) and expansion of the cell wall is needed.

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Discussion

A. W. Galston (*Plant Breeding Institute, Trumpington, U.K.*). Many years ago, Blaauw, in the Netherlands, investigated the 'light growth reaction' (*Lichtwachstumsreaktion*) in various tissues, including grass coleoptiles and *Phycomyces* sporangiophores. In etiolated coleoptiles, exposure to a pulse of blue light was followed by a temporary depression of growth rate, then an overshoot above the control dark value, and a return to the original growth rate. In *Phycomyces*, because of the sporangium lens effect, light induced a temporary rise in growth rate, followed by an

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undershoot, and a return to normal. After the completion of this entire cycle, there was no net effect of light on total growth in either organism. Dr Cosgrove's curves show no such details. Can he rationalize the differences between his results and Blaauw's?

D. Cosgrove. The figures I presented only show the *inhibition* of the growth rate, as that is the part of the response that we have studied most intensely. We have also followed the recovery of the high growth rate after a pulse of blue light; typically the recovery is slower than the inhibition, and there are pronounced oscillations in the growth rate (see our earlier published figures of the responses in pea, mustard, sunflower and cucumber seedlings). These oscillations frequently lead to an overshoot above the previous dark growth rate, but in general the overshoot is insufficient in magnitude and duration to cancel out completely the inhibitory effect of light. Such overshoots have been noted by other workers such as Gaba & Black.

Regarding Blaauw's results, I think the differences he has mentioned are due to the different organisms involved. It is well known that the *Phycomyces* growth response to a step-up in fluence rate is transient and is linked with the sporangiophore's phototropic behaviour. In dicot stems, in contrast, the growth inhibition persists as long as the stems are irradiated. Blaauw also examined the light growth response in sunflower hypocotyls, and found a pattern of response similar to ours.

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