Light Responses of Phycomyces

Light catalytically alters the distribution of the cell's regulated growth in time and in space.

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The sporangiophore of the mold *Phycomyces* is celebrated for the fact that light modifies its rapid growth in easily measurable and sometimes dramatic ways. The sporangiophore is a cylindrical tube, in effect a single cell, that elongates into the air at high speed (between 2 and 4 mm/hour), carrying a package of reproductive spores at its end. It aims itself toward a source of light and, under constant conditions, holds this aim and elongates in that direction for many hours at an essentially constant speed. If we do something to the light, the plant's growth changes—in speed, in direction, or in both; such changes are its light responses.

To an increase in light intensity, the cell responds by a temporary, damped spurt in speed of growth, with no change in its direction; this is the light-growth response (Fig. 1). To a change in the light's angular position, the cell responds by bending to reaim itself toward the new position. If the light moves slowly in a circle around the cell, the cell wraps itself into a helix in an endless attempt to follow the light; this is phototropism, in which the swing of the cell's axis is the mechanically amplified result of a small but persistent difference in growth rates across it. In practice, phototropic experiments are usually initiated not by motion of a target light source but by radiation incident normal to the cell's long axis and made asymmetric around it (Fig. 2).

Different forms of response to other situations are either negative variants or combinations of these two types. For example, the cell bends away from, instead of toward, a source of ultraviolet light (negative phototropism). Again, a flash of high-intensity light given during the course of normal positive bending causes a temporary period of reversed (negative) bending (Fig. 3); this is an instance of phototropic inversion which, although procedurally a light-growth response superimposed upon a phototropic response, is not at all a simple addition of them. How does a system of such apparent simplicity give rise to this range of behavior? Implicated are interconnected problems in photoreception, intracellular regulation, and cell enlargement (*1*).

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The Growth System

The light responses are changes in motion (spurts, bends) of an elongating thin-walled tube that is under internal pressure sustained by the uptake of water at the cell's base. When used for experiment, the sporangiophore is already several centimeters long, and it may extend finally to 15 centimeters or more. Growth, sensitivity to light, and response are all restricted to a zone (for brevity, the "tip") some 3 millimeters long at the cell's extremity, immediately below the spore mass. The tip has the form of a slightly tapered cylinder about 30 diameters long. It is not obviously different from the rest of the cell, with which it is continuous, except for its extensile wall and tapered profile.

The distribution of local growth activity along the tip is shown by the motion of small markers applied to its surface; the integral of this activity over the tip's length is the cell's observed speed of elongation. Activity rises to an early maximum in the upper quarter of the tip and then declines to zero at about 3 millimeters from the spore mass. A material element of the wall, originally near the top of the tip, passes down the tip as if along a production line, increasing in length by a factor of 20 to 30 during its travel time of some 2 hours; thereafter the element is immobile in the wall beneath the growing zone, where layers of secondary wall are deposited on it from within. As an automatic result of its own growth, the tip as a whole moves upward in step with the cell's elongation, thereby maintaining its characteristic length, distribution of growth activity, and terminal position in the cell.

Submicroscopically, the wall of the tip has a structural network built of chitin, a long-chain polymer of glucosamine. Chitin chains appear to be formed at the wall's inner surface, with a preferred orientation transverse to the cell's axis, like hoops around a barrel. As a piece of the wall undergoes its enormous axial extension in passage down the tip, the outer mesh of the network is pulled out longitudinally while new transverse chains continue to be deposited beneath it (Roelofsen's "multinet growth," *2*). Hence the growing wall preserves its characteristic optic and elastic anisotropy.

We do not yet fully understand, in the case of any plant cell, how mechanical stress on the wall interacts with the chemistry of growth, or what initially determines the directional property of growth (*3*). It is convenient, but perhaps misleading, to think of the growing wall as repeatedly yielding to longitudinal stress. But, in *Phycomyces*, extension is consistently at a small angle from the longitudinal direction, so that the tip twists as it elongates. As a conspicuous result the spore mass is revolved about its axis while being pushed upward. Twist appears in the light responses, complicates the geometry of phototropic bending, and is of much interest in itself, but I shall not discuss it further.

Two distinct parts of the cell that actively contribute to growth—the tip and the base—are separated by a relatively great distance that increases at the rate of 3 millimeters per hour. Nevertheless, the pressure and volume requirements for normal elongation are met by water uptake at the base, for steady growth goes on for days. By the same test, the supply of all solute molecules, ultimately derived from the basal substrate and used in the tip, must also be adequate, even though transport by simple diffusion is out of the question. Rapid cytoplasmic steaming (absolutely faster than the cell's extension) is conspicuous along the cell and may perform this transport func-

tion. Both water uptake and transport appear unaffected by light; if their rates are indeed fixed, either may check the initial acceleration of growth during the light-growth response (Fig. 1).

Light is not necessary for any process involved in growth, for the same steady-state speed of elongation occurs in complete darkness as in light. This fact means, formally, that the action of light on growth is a positive catalysis and, mechanistically, that growth of this cell is strictly limited by light-insensitive processes. All models of the system are built from this foundation.

General Features of the Responses

The various light responses almost certainly have the same photochemical basis. Spectral studies of response—action spectra both for growth acceleration and for bending—imply that the tip contains traces of a presumptive yellowish pigment that absorbs over the wavelength range from 230 to 500 nanometers (*4, 5*). There is no clear evidence that more than one pigment is implicated. The chemical nature of the pigment is disputed and, more important for many purposes, we do not know where it is located radially within the cell (*6*). But such studies clearly exclude both the chlorophyll system (absent from a mold in any case) and the phytochrome system through which red light influences the development of higher plants in many ways. The responses of *Phycomyces* belong in that separate area that is sometimes termed "blue light photobiology." The distinction is clear, but not its meaning.

If the cell is considered as a stimulus-response system, light is a stimulus when there is (i) a sufficient change in the received radiant flux, or (ii) a sufficient unequal distribution of flux within the cell. The double definition is necessitated by a basic difference between the light-growth response and the simple phototropic response. All the responses vary, within limits, with the size of the stimulus and are therefore inherently graded and not all-or-nothing. Irradiation of the whole tip is not necessary to cause response; light thrown on a small fraction of its length or grazing its periphery can speed growth or produce bending (*7*). Thus receptor and response mechanisms occur close together throughout the growing zone, and small regions of it have some reactive autonomy. There is no evidence of (or apparent need for) a long-range growth mediator such as auxin, an intercellular coordinator and hormone in higher plants.

There is a remarkable delay of 2 minutes or more between the onset of stimulation and the appearance of response in all the light responses; it varies with the size of the stimulus and with the state of the cell's recovery from prior stimulation (*8*); it is perceptibly longer for bending response than for simple acceleration of growth. We have no idea what processes occupy and require this long latent period; they are not a specific consequence of stimulation by light, since delay is roughly comparable when bending is induced by other agents such as applied droplets of water (*9*).

One is tempted to consider that light acts to increase the directional yield of the cell wall under the driving force of the cell's internal pressure. On this basis, immediate energy for the light-growth response would be in the stressed wall of the whole cell. A spurt in extension should be accompanied by a small change in the cell's shape (a gain in length at the expense of diameter) and, if the osmotic pump works at a fixed rate, by a drop in internal pressure. Neither change, if measurable, has been observed.

The pure light-growth response follows an increase in light intensity applied symmetrically about the cell, and is fundamentally an event in *time*. It has the form of a strongly damped oscillation: a steep rise in elongation speed, a maximum at about 5 minutes (when the speed may easily have doubled), and a return to the standard speed after about 15 minutes (Fig. 1). The cell does not bend, because growth is symmetric around its axis. After a recovery period following the spurt, the cell is said to be adapted to the higher intensity, for its behavior is just as it was before it was stimulated; it will now respond only if the intensity is again raised. As in the case of vision, the cell is sensitive and responsive over a prodigious range of intensity, at each level of which it restabilizes its growth. A decrease in intensity from any level provokes a corresponding damped fall in growth speed, to be followed by recovery. The adapted cell is clearly in a state of poise—not of fatigue or indifference.

Different levels of adaptation represent different steady states, which all nevertheless generate growth at the same rate. One may model such a system by supposing that an enzyme that catalyzes cell extension is some increasing function of the light flux, and that the rate of supply of the enzyme's substrate to the tip is fixed. If one assumes that the concentration of catalyst rises rapidly when the flux rises, the kinetics of response will largely be determined by changes in the available substrate. Thus growth resembles the flow from a reservoir (the substrate) drained through a valve, the aperture of which is controlled by light; growth in darkness implies a bypass or alternate channel, unregulated by light. An increase in flux opens the valve and briefly increases the flow, which then decays toward its original rate and toward a new steady state with a more-open valve and a level in the

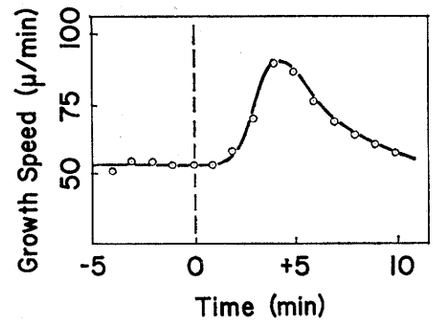

Fig. 1. Light-growth response following an increase in light intensity. Speed of growth is the cell's measured elongation per minute. At zero time, the light flux symmetrically incident on the cell was increased tenfold and maintained.

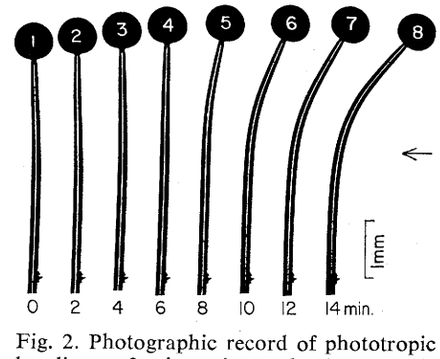

Fig. 2. Photographic record of phototropic bending at 2-minute intervals. Asymmetric irradiation from the right (arrow) begins at zero time; note the delay before bending begins. Only the cell's distal 4 millimeters are shown. Identifying numbers appear on the terminal spore mass. Starch-grain markers (bottom right of cell) are in the unresponsive region below the growing zone. The central axial white line is due to the cell's lens action on the beam of red (phototropically inactive) light by which the photographs were taken; this beam's axis is normal to the plane of the paper.

reservoir lower than it was initially (*10*).

The cell's level of adaptation must be considered the momentary state of the whole system, which is determined not only by the light intensity but also by such variables as available metabolites, wall stress, internal pressure, and the past history of the growing wall. The process of adjustment to a greater flux (light adaptation) runs a limited course in time and ends, practically if not theoretically; the reverse adjustment is dark adaptation. Different criteria of response give different estimates of the durations of these processes, which may require more than 1 hour if almost-full replacement of the tip's assembly line is to be accomplished.

Little attention has been paid to the processes that check the growth spurt so rapidly. There is evidence that for the extremity of the tip the supply of a metabolite may become immediately limiting, as is formally postulated in the model.

I have recently studied the way in which local growth activity during the light-growth response is distributed along the tip. Just after the maximum of the response, growth at the very top of the tip is depressed and sometimes abolished, although the remainder of the tip continues rapid growth. At this instant, the top of the tip is being thrust away from its base of supply at about twice its steady-state speed. This local decrease in growth appears to be a novel form of self-regulation by speed itself; I strongly suspect that it is promptly supplemented by a general factor such as decrease in internal pressure and hence in wall stress throughout the tip. The several possible sources of negative feedback in this situation have not been isolated; nor have the probable phase differences in recovery along the tip been adequately examined (*11*). A model that realistically accommodates a changing distribution of response along the tip is certain to be distressingly complex.

Phototropism

If the light-growth response is fundamentally a change in growth with *time*, phototropism is a difference in growth in *space*. The pure phototropic response results from an unchanged flux acting asymmetrically. Hence, for the cell as a whole there is no change in its average steady states, no adaptation, no time-limited course of response, but a spatial asymmetry of growth that can last indefinitely. Many phototropic responses in practice also contain a growth spurt because there has been an incidental change in flux in the process of making the flux asymmetric, but these two main modes of response are related only in the common photocatalytic action of light. Phototropism is not based upon light-growth responses of different magnitude across the cell, as has often been argued: in uncomplicated phototropism there is no light-growth response.

The chief problems in simple phototropism are to understand how the asymmetries of growth and the regularities of bending arise. Let us consider one of the simplest experimental situations: A cell was initially growing vertically between two equal and opposite horizontal beams of light. One beam is now extinguished and the other is doubled in intensity. After a delay of several minutes, the cell begins to bend toward the light at an angular speed that is remarkably constant for any one cell (Fig. 2). The flux through the cell is unchanged, but it is now acting asymmetrically on the growth mechanism. Optics determine whether bending is positive (toward the light) or negative (away from the light) (*5*, *12*, *13*, *14*). In air, the tip acts as a cylindrical converging lens of high transparency. Light entering from one side is refracted and passes through the cell in a converging bundle focused just outside the back wall. Light is unequally and complexly distributed within the front and back halves of the cell, but the result is faster growth of the back half than of the front—hence bending toward the light. Paradoxically, the flux through the back half must be the lesser because of slight losses by absorption from, and scattering in, the front half, but this difference is normally more than offset by some aspect of the lens action. Thus normal positive bending results from small attenuation (weak absorption) acting against a great effect of the lens (strong convergence).

The balance of these two factors can be upset in different ways, with the expected result: for example, for ultraviolet light, absorption is much stronger, convergence is almost the same as for visible light, and bending is negative because the front half of the cell now grows faster than the back. There is no special inhibition of growth here, for the light-growth response to ultraviolet light is a normal spurt. Alternatively, if the lens effect is changed by immersion of the cell in mineral oil (wherein it acts as a diverging lens), bending in ordinary light is negative instead of positive. These negative responses are analytically interesting but have no place in the plant's normal reproductive life.

In the simple experimental situation that I have just described (visible light, a single horizontal beam, cell growing vertically in air), the bending speed of many cells is about 5 degrees per minute; this speed corresponds to a difference in growth speed of 10 to 15 percent between the cell's two halves. Measurement shows that the average speed of growth around the cell's whole periphery during bending is the same as the speed of elongation before bending commenced. Thus, positive bending really involves two factors: (i) an increase in growth of the back half of the cell, and (ii) a corresponding decrease in growth of the front half.

This compensatory shift in speeds shows that, in steady-state phototropism, growth is conserved: the front must slow as the back speeds. In a sense, the response of the front is a local inhibition of growth by light; but inhibition is indirect and does not contradict the idea that basically light in this system only promotes growth.

One might expect the cell to bend slowly toward a dim light and faster toward a bright one, but in fact over a 1000-fold range of light intensity the cell bends at its standard speed. The explanation is that light acts in a constant ratio across the cell and that the cell's growth output is fixed. Both factors are, in the steady state, independent of the absolute flux (*15*, *16*).

Bending speed and the asymmetry of irradiation that causes it are both maximal in experiments with one beam of light. With two or more horizontal beams from varied azimuths, the net asymmetry is reduced and bending speed is a vector in the expected resultant direction and of magnitude proportionately reduced; here the cell integrates multiple patterns of illumination within it, the magnitude of the lens action being in effect reduced (*14*). All experiments of the type discussed have the defect of short duration, for the cell reaches its goal after bending through only 90 degrees; moreover, the incidence of light on the curving cell changes undesirably with time. The full proof that phototropism is a steady-state process without limit in time is given by Dennison's experiments in which the light moved in pace with bending, and growth took the form of a virtually endless helix (*17*).

Phototropic Inversion

Simple phototropism is not a spatially uniform steady state, since the action of light and the resultant growth are both unequal across the cell. It follows that there must be local differences in adaptation within the bending cell. A difference in adaptation is only revealed by a difference in response, the stimulus being a change in flux. If, then, we change the level of light intensity while a cell is engaged in bending, the responses of its front and back halves should differ if their local states of adaptation are different. Such an experiment in fact produces phototropic inversion.

Consider a cell bending toward a light of unit intensity. Let the intensity suddenly increase tenfold. Bending continues at the standard speed for several minutes, then very suddenly it stops and reverses its direction, becoming negative. In turn, this negative bending slows and stops, and positive bending gradually resumes again. The increase in intensity of the light, which does not in any way alter the optical balance across the cell, has temporarily caused the front half to grow faster than the back. Most remarkably, if in a similar experiment the change in intensity is a decrease instead of an increase, the same cycle of temporary reversal followed by recovery occurs. These are the two types of phototropic inversion (*13, 15*). Figure 3 gives a photographic record of inversion following a superimposed pulse of high-intensity light.

The light-increase inversion is literally a light-growth response evoked in a phototropically bending cell, with the unexpected feature that the response of the cell's near side (initially slower growing) is temporarily greater than that of the far side (initially faster growing). This difference in response signifies different local states of adaptation across the cell in the plane of bending; they were established there during the prior bending, for separate experiments show that the negative bending is in every instance in the same predetermined plane, regardless of the direction of the high-intensity beam (Fig. 3; *18*). Hence the recent history of local regions of the cell is a factor affecting their local states of adaptation.

An asymmetric modification of the kinetic model that I have described for the light-growth response imitates the inversions (*10*). It is only necessary to set up two parallel reaction chains, sharing a common supply of metabolite, upon one of which (the cell's far side) unilateral light has a continuously greater photocatalytic effect (because of the cell's optics). Then during steady bending there is inequality in the levels of two local reservoirs. If, as before, light controls a valve draining each reservoir, it is found that either opening or closing of the two light-responsive valves yields temporal inequalities in flow that mimic both inversions. The process of adaptation is in reality a complex of changes; simplifying assumptions are built into the model, but its general structure gains credibility from reproduction of such special behavior.

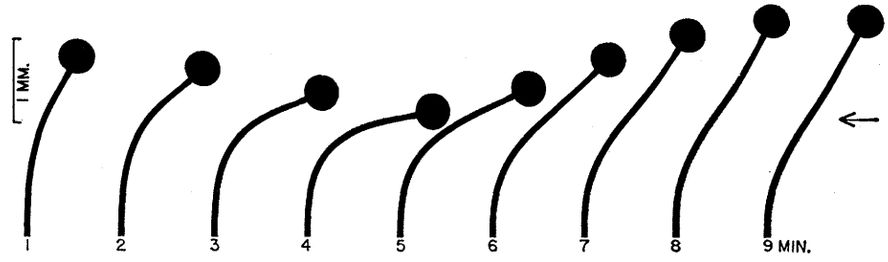

Fig. 3. Serial photographs of normal positive phototropic bending (first four pictures), followed by inversion (last five pictures). Continuous low-intensity irradiation from the right (arrow) commenced at −6 minutes. At zero time, the bending cell received a 15-second flash of high-intensity light from behind (perpendicular to plane of paper). Reversed bending is in the same plane (that of the paper) as initial bending. Later, normal positive bending resumes (not shown).

Regional Response

The responses so far considered result from irradiation of and response by the whole tip. There are difficulties if we seek to resolve the responses more highly. For example, bending has been treated, for simplicity, as if rate differences were lumped in two halves of the cell. In fact there are two continuous distributions in space of differential elements of growth rate: (i) a longitudinal distribution that can only be determined from the study of applied markers, and (ii) an angular distribution around the periphery of the tip's cross-section. The angular distribution is uniform in straight growth, but in a bending cell it is a cosine function demanded by the geometry of bending. The way in which the lens effect is translated into this regular periodic function is not clear; there must be a process of smoothing, perhaps in part purely physical in nature. Thus except in the rough the mechanism of the lens effect is not understood. This trouble is magnified in phototropic studies in which light is incident at large angles to the plane of the cell's cross-section (*13, 19*).

Interesting investigations by Delbrück and his associates have suggested that light reception and response have different local distributions along the tip. Thus, when the whole tip was repeatedly stimulated at 5-minute intervals, less than half its length was found responsive to light (*11*). In other experiments stimulations of very small zones of the tip led to the conclusion that the receiving and responding systems must be in separate structures that move complexly in relation to each other (*20*).

Unfortunately, these modes of experimentation maximize the uncertainties in our knowledge of adaptation. I am cautiously optimistic that other interpretations of this behavior can ultimately be made, perhaps in terms of phase differences in the recovery of responsiveness along the tip. It is likely that phase differences *across* the tip underlie the phototropic oscillations discovered by Dennison (*21*). Much more must be known about the time course of recovery after stimulation, as well as about interactions between small stimulated and unstimulated parts of the same pressurized tube, before response can be understood at this microscopic level.

Summary

The various growth spurts and bends obtained in simple programs or irradiation can be interpreted, up to a point, in terms of optics, symmetry, and the cell's intrinsically regulated growth. Simple kinetic models imitate many gross features of the responses, and imply that light catalyzes some step in the formation or extension of the cell wall. This step may well be a nearly terminal one, perhaps involving slippage of chitin chains over one another. The most pervasive concept in these studies

is that of adaptation, of which the steady-state level is primarily set by the light flux. But, since adaptation is tested by a growth response, every "dark" process contributing to cell enlargement is also implicated. From these sources comes the prompt negative feedback after an increase in light flux, as well as the conservation of growth seen in bending. Use of light by the plant seems to be only for operation of a crude guidance system for spore dispersal. For the investigator, light is a tool that displaces or unsteadies the mechanisms of cell extension and gives glimpses of their otherwise-concealed complex interplay.

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The Brain Drain: A U.S. Dilemma

The nature and extent of the brain drain, its effects on welfare, and its implications are analyzed.

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The migration of highly skilled individuals from the rest of the world to the United States, often called "the brain drain," puts U.S. society and policy makers on the horns of a genuine dilemma: On the one hand the United States is morally and politically committed to assist the development of the poorer regions of the world, and anything retarding this process, such as the loss of high-level manpower resources through emigration, runs contrary to the declared foreign policy of the nation. On the other hand, the United States has considered it to be in its national interest to restrict general immigration and make it selective through a set of laws and regulations that favor individuals with high levels of training. Furthermore, the country has a tradition of respecting personal liberty, welcoming the poor and oppressed, and avoiding coercion, so that under certain circumstances students are permitted to become immigrants even though laws and visa regulations would otherwise require them to leave the United States after completion of their studies.

In recent years countries throughout the world have awakened to the brain drain, as is evidenced by frequent articles in the foreign and U.S. press (*1*); the authors expand and popularize whatever empirical evidence regarding the magnitude of the migratory flows has been assembled by international agencies, national governments, and scholars. The catchy phrase "brain drain" has penetrated the public consciousness, and its implications are frequently discussed among intellectuals. The U.S. Department of State in June 1966 held a conference during which government officials, representatives from private organizations, and scholars discussed the issues surrounding the brain drain. The United Nations, the Pan American Health Organization, and the Organization for Economic Cooperation and Development are preparing studies and conferences to assess the magnitude of the problem and to arrive at policy stands. Recently, Walter F. Mondale, U.S. Senator from Minnesota, spoke (*2*) of the problem on the Senate floor; he summarized the government's dilemma by quoting Assistant Secretary of State Charles Frankel:

> This is one of the steady, trying, troublesome diplomatic issues confronted by [our] government . . . one of the most important problems faced not just by the Department of State, but more important, by the United States and by mankind as a whole.

Before the United States can develop a program to deal with the complex phenomenon so conveniently labeled the brain drain, its nature and magnitude must be understood more clearly than hitherto.

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