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11. We thank J. B. Dumser and C. M. Williams for helpful suggestions regarding this paper. Supported in part by PHS research grant 09556 from the National Institute of Allergy and Infectious Diseases.

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Phycomyces: Interference Between the Light Growth Response and the Avoidance Response

Abstract. Phycomyces *sporangiophores show a growth response to a light stimulus and an avoidance bending response to a physical barrier. A blue-light stimulus administered in conjunction with a barrier interferes with the avoidance bending response. This interference begins after a latency of about 3 minutes and continues for a period of 4 to 5 minutes.*

The most puzzling and least studied sensory pathway in *Phycomyces* is the avoidance response. By a yet unknown mechanism, a sporangiophore senses the presence of a solid barrier a short distance away and will bend away from it after a latency period of about 3 minutes (*1*). We have found that this response can be inhibited by a blue-light stimulus given in combination with the avoidance stimulus. Such interference is evidence of a significant physiological interaction between these two sensory pathways in the regulation of cell growth.

Although genetic studies by Bergman *et al.* (*2*) indicate that the two pathways share a common element, previous work has provided no physiological evidence for such interaction. A study by Cohen *et al.* (*3*) reports that the avoidance bending response is not affected by steady illumination, even at light intensities as high as 20 mW/cm^2, conditions under which the sporangiophore has lost the ability to respond to any light stimulus. Similarly, Ortega and Gamow (*4*) report that, after a saturating 10-minute light stimulus at 82 mW/cm^2, a normal avoidance growth response (bilateral barrier stimulus) still occurs.

In the work we report here, the avoidance stimulus was a plastic cover slip placed about 1 mm from a stage IV sporangiophore and kept at a constant distance throughout each experiment. Sporangiophores were grown in white light and adapted to red light for at least 5 minutes before each experiment. The blue-light stimulus was either a 1-minute pulse or a step (continuous light) at 315 μW/cm^2, with light from a 100-W tungsten bulb filtered through a Corning 5-61 filter. The light was split into two equal beams that struck the sporangiophore on each side at an angle of 30° from the horizontal. The angle of bending was measured in red light by use of a microscope with a goniometer eyepiece.

The application of a blue light step stimulus at 3, 7, or 10 minutes after the avoidance stimulus causes a temporary reversal of avoidance bending (Fig. 1, curves c, d, and e). Table 1 shows that, regardless of the time the light stimulus is applied and the amount of forward bending that has occurred, the interference period begins about 3 minutes after the light stimulus is applied and has a duration of 4 to 5 minutes. Similar results were obtained with 1-minute light pulse stimuli.

Not only does a light stimulus interfere with an ongoing avoidance response but if given earlier it can also interfere with the onset of this response (Fig. 1, curves a and b). Table 2 shows that the avoidance latency period, defined as the interval between application of the barrier and the start of avoidance bending, is 4 to 5 minutes longer in the presence of a light stimulus than in the absence of blue light. The light stimulus clearly interferes with the onset of avoidance bending, and the additional delay is roughly the same as the interference periods described above.

We also found that a succession of 1-minute light pulse stimuli, given at 10-minute intervals during an avoidance response, elicits a corresponding succession of bending reversals. Each reversal begins about 3 minutes after the pulse and lasts 4 to 5 minutes. When two pulses are spaced only 3 minutes apart, there is an overlap of the two interference periods, resulting in an unusually long reversal.

From the fact that the interference caused by a 1-minute pulse of light is comparable to that caused by a step, we conclude that the interference is not due to the presence of light per se but is instead a result of the growth response to

Table 1. The time of onset and duration of the interference period and the magnitude of the reversal induced by a light stimulus applied during an ongoing avoidance response. Steps were continuous blue light at 315 μW/cm^2; pulses were 1-minute flashes of the same light. Light stimuli were begun 3, 7, or 10 minutes after the barrier stimulus was applied. Values are means ± standard errors of the means. The number of experiments is given in parentheses. Time of onset is the interval between the beginning of the light stimulus and the beginning of the reversal. Interference duration is the duration of reversed bending.

Light stimulus	Time of interference onset (min)	Interference duration (min)	Reversal magnitude (deg)
Step (t = 3)	3.38 ± 0.13 (4)	3.75 ± 0.48 (4)	2.45 ± 0.61 (4)
Step (t = 7)	2.63 ± 0.24 (4)	5.50 ± 0.64 (4)	5.25 ± 1.08 (4)
Step (t = 10)	2.13 ± 0.23	5.00 ± 0.35 (4)	7.75 ± 0.92 (4)
Mean, all steps	2.71 ± 0.23	4.75 ± 0.35	
Pulse (t = 3)	3.00 ± 0.00 (5)	4.33 ± 1.64 (2)	6.25 ± 0.25 (2)
Pulse (t = 7)	2.67 ± 0.17 (3)	4.83 ± 0.66 (3)	8.33 ± 2.19 (3)
Mean, all pulses	2.88 ± 0.08	4.58 ± 0.79	

Table 2. The avoidance latency following light treatments. Dark indicates barrier applied in the absence of blue light; step, barrier applied 1 minute after the beginning of a blue light step at 315 μW/cm^2; and pulse, barrier applied immediately following a 1-minute pulse of the same light. Latency is the interval between application of the barrier and the beginning of avoidance bending. Values are means ± standard errors of the means. The number of experiments is given in parentheses.

Light treatment	Latency (min)
Dark	3.01 ± 0.78 (15)
Step	8.07 ± 0.80 (7)
Pulse	7.13 ± 0.37 (3)

0036-8075/79/1019-0357$00.50/0

light. The latter typically shows a 3-minute latency, a 4- to 5-minute period of rapid growth, and a final decrease in growth rate to baseline levels (*1*)—a pattern that coincides with the light-induced period of interference we have observed.

Our evidence for interference between the light growth response and the avoidance response does not contradict the findings of Ortega and Gamow (*4*), because they did not apply the avoidance stimulus until 12 minutes after the light stimulus. Our experiments show that a light stimulus is capable of causing interference only for a period of 3 to 8 minutes after the beginning of the light stimulus. Our results are likewise compatible with those of Cohen *et al.* (*3*), since they used steady illumination, which does not cause a light growth response.

Why does a bilateral light stimulus cause a reversal of avoidance bending in addition to simple inhibition? The phenomenon of temporary tropic reversals induced by light stimuli has previously been noted for the phototropic and geotropic bending response (*5*). Castle (*6*) has proposed a theory to explain such reversals, and his model can be extended to include reversals of the avoidance response.

Castle postulates that there is a substance, M, that is continually supplied and is distributed equally on both sides of the sporangiophore growing zone, forming M pools. The M substance is consumed during growth, being depleted equally on both sides during straight growth. When a sporangiophore bends, the M substance is depleted faster on the convex side because that side is growing more rapidly. When a light stimulus is applied, it triggers a period of rapid growth, and, since the rate of growth is proportional to the size of the M pools, the side with the larger M pool grows faster, causing a reversal of the original bending.

Castle's model implies that the magnitude and the speed of onset of the reversal should be proportional to the degree of imbalance in the two M pools, the imbalance being in turn directly related to the amount of bending that has occurred prior to the light stimulus. Our results are in accordance with this prediction. The latency of the reversal decreases and the magnitude of reversal increases as the elapsed time increases between the avoidance and the light stimuli (Table 1). This more rapid onset and greater magnitude may be a consequence of the greater M pool imbalance. No reversal is observed when a light stimulus is applied before avoidance bending has occurred

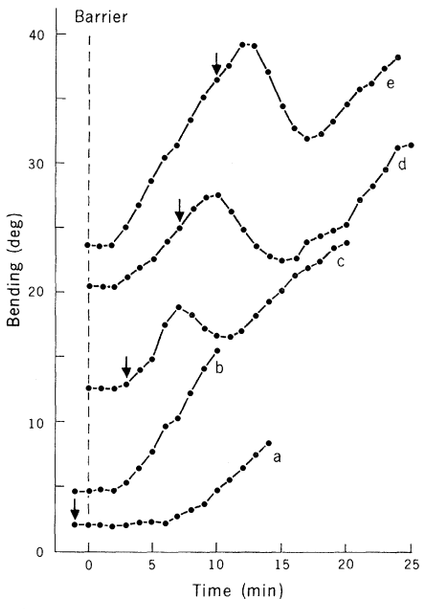

Fig. 1. Stage IV sporangiophores are placed 1 mm from a barrier stimulus at $t = 0$ minutes. A blue light step at 315 μW/cm^2 was applied 1 minute before the barrier (a), at $t = 3$ (c), at $t = 7$ (d), and at $t = 10$ (e), as shown by the arrows. No light step was applied in (b). Curves a through e represent the average of seven, six, four, four, and four experimental runs, respectively. All curves are displaced along the bending scale.

(Fig. 1, curve a). In this case there has been no previous bending to cause an imbalance in the M pools, and the interference period is seen as an extension of the latency of the avoidance response.

The significance of this work lies in the discovery of the light-induced delay of onset, a phenomenon unique to the avoidance response. The existence of an element common to both phototropic and avoidance pathways has been shown by the isolation of mutants in which both pathways are blocked (*2*). The delay of onset indicates that this common element is nonlinear and cannot function simultaneously in both pathways when they each carry saturating stimuli.

Our results raise the question of whether this interference is reciprocal. If so, one would expect a bilateral avoidance reponse to interfere with phototropism.

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Hydrostatic Pressure Reversibly Blocks Membrane Control of Ciliary Motility in *Paramecium*

Abstract. *A hydrostatic pressure of only 68 atmospheres prevented swimming* Paramecium caudatum *from "avoiding" or reversing direction; 170 atmospheres stopped or decreased forward velocity by more than 75 percent. A decompression of 40 atmospheres invoked a single reversal, even at 280 atmospheres. In contrast, 170 atmospheres did not significantly affect swimming behavior of paramecium "models" that were reactivated in a solution containing adenosine triphosphate and magnesium ions after their membranes had been disrupted by Triton X-100.*

In a survey of pressure effects on the activity of ciliates and flagellates, Kitching (*1*) reported that 100 to 200 atm slowed or stopped swimming paramecia. Our studies were designed (i) to characterize more fully the pressure-related changes in the swimming behavior of paramecia and (ii) to determine whether pressure induces alterations in the ciliary machinery or in the electrically excitable plasma membrane that controls the frequency and direction of ciliary beat. The bioelectric properties of the paramecium's surface membrane are closely coupled to the activity of its locomotory cilia (*2*). A depolarizing influx of calcium ions causes ciliary reversal and backward swimming; with hyperpolarization, the frequency of ciliary beating and rate of forward swimming increase (*2*). By comparing the effects of pressurization and decompression on normal paramecia with their effects on paramecium "models" [paramecia treated with Triton X-100 so that their membranes were no longer functional barriers (*3*)], we deter-

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