WHITE NOISE ANALYSIS OF *PHYCOMYCES* LIGHT GROWTH RESPONSE SYSTEM

III. PHOTOMUTANTS

E. D. LIPSON

From the Division of Biology, California Institute of Technology, Pasadena, California 91125

ABSTRACT Wiener kernels have been measured for the light growth response of a number of mutants of *Phycomyces* which show abnormal phototropism (*mad* mutants). Representative mutants were chosen from the six complementation groups (*madA* to *madF*) associated with the light response pathway. One group, *madA*, associated with the input part of the pathway, exhibits an essentially normal response provided it is tested above its moderate threshold. The groups *madB* and *madC* appear more defective, in that their kernel amplitudes are very small even above their thresholds. Their similarity to each other suggests a close functional connection between the respective genes. The remaining three groups (*madD*, *madE*, and *madF*) have all been associated with the output of the pathway. The kernels for all three indicate a gain reduction, which depends gradually on intensity. These three groups appear to have the same absolute threshold as wild-type. None of the mutants studied shows special behavior at high intensity that could be evidence of alterations in the photoreceptor complex.

INTRODUCTION

A number of representative photomutants will be examined in this paper, using as a basis the results of the two preceding papers (Lipson, 1975*a*, *b*; herein referred to as papers I and II).

Mutants with abnormal phototropism are designated *mad*. They have been classified by Bergman et al. (1973) in terms of various inputs and outputs. The scheme they proposed is shown by the sensory pathways of Fig. 1. The central pathway applies to the light growth response and phototropism of mature sporangiophores. The two other light-dependent outputs, namely, carotenogenesis and sporangiophore initiation, both pertain to the mycelial stage of development. The two other input channels mediate geotropism and the avoidance response (Cohen et al., 1975) of sporangiophores.

Mad mutants abnormal with respect to all photoresponses are designated class 1.1. Those *mad* mutants with normal mycelial photoresponses and normal geotropism and avoidance are assigned to class 1.2. Finally, class 2 contains the *mad* mutants with abnormal growth output to all three input modes.

Using heterokaryons produced by microsurgery, Ootaki et al. (1974) have performed

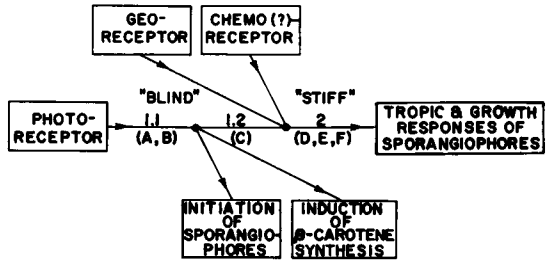

FIGURE 1 Physiological classification scheme for *mad* mutants, adapted from Bergman et al. (1973). The classes are denoted by numbers (1.1, 1.2, 2). The letters refer to the complementation (*madA*, *madB*, . . . , *madF*) determined by Ootaki et al. (1974).

complementation tests on several mutants from each class. As indicated in Fig. 1, they discovered two genes, denoted *madA* and *madB*, within class 1.1 and one denoted *madC* for class 1.2. In class 2 they reported two genes, *madD* and *madE*. Recently an additional class 2 gene, called *madF*, has been determined by E. P. Fischer (personal communication).

Fig. 2 shows a composite of phototropic threshold curves for *mad* mutants measured by Bergman et al. (1973) and Ootaki et al. (1974). The curves depict the phototropic-geotropic equilibrium bending angle as a function of absolute intensity. Implicit in these curves is the fact that no strain is absolutely blind. The two *madA* mutants have thresholds about 10^4 times higher than those of C2 or wild-type. The *madB* and *madC* mutants have even higher thresholds. The thresholds of *madD* and *madE* are indeterminate because of the shallowness of the curves; the threshold of *madF* is evidently the same as that of wild-type.

METHODS

The mutant strains used in this work are listed in Table I. The experimental procedures were the same as in paper I. All experiments were performed with the standard noise pattern and dynamic range defined in paper II. The "log mean" intensities (as defined in paper I) used were $I_0 = 10^{-3}$, 10^{-4}, 10^{-6}, and 10^{-9} W/cm^2.

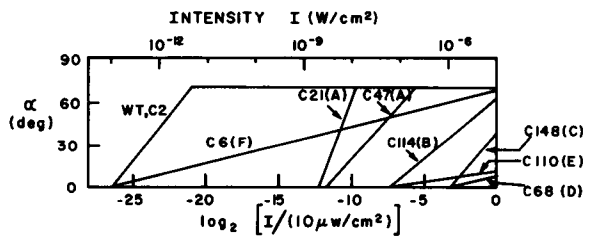

FIGURE 2 Phototropic threshold curves for *mad* mutants (Bergman et al., 1973; Ootaki et al., 1974). For comparison the normal curve common to C2 and NRRL1555 (WT) is shown. As in Fig. 1, the letters A, B, . . . , F are abbreviations for the respective complementation groups. The upper intensity scale in physical units is pertinent to the present experiments. The lower scale with $\log_2$ units is in common usage in *Phycomyces* research (Bergman et al., 1969).

TABLE I

STRAINS OF *PHYCOMYCES BLAKESLEEANUS* USED IN THIS WORK

Class	Strain	Genotype*	Origin
-	C2†	carA5 (-)	NRRL1555
1.1	C21	madA7 (-)	NRRL1555
1.1	C47	madA35 (-)	NRRL1555
1.1	C114	madB106 (-)	NRRL1555
1.2	C148	carA5 madC119 (-)	C2
2	C68	madD59 (-)	NRRL1555
2	C110	madE102 (-)	NRRL1555
2	C6	carA12 carR27 madF48 (-)	NRRL1555

*Based on phenotype. Mutants with abnormal phototropism are designated mad. Those with abnormal carotene complement are designated car. The car genes (here carA and carR) and the mad genes (A to F) were determined by Ootaki et al. (1973, 1974).

†The albino strain C2 has photophysiology identical to the yellow wild-type strain NRRL1555 (-).

RESULTS

madA Mutants

The two *madA* strains C21 and C47 have somewhat different threshold behavior, as indicated in Fig. 2. Whereas both have thresholds over four decades above that of wild-type, the steeper curve for C21 saturates at lower intensity than that for C47. Fig. 3 shows the kernels of these two strains compared to C2. The log-mean intensity was the standard $I_0 = 10^{-6}$ W/cm^2, which is above their threshold regions.

The mutant kernels above threshold are substantially normal in shape and magnitude. Closer examination of the kernels and the data in Table II reveals the following trends in order of increasing threshold (C2, C21, C47): (*a*) decreasing mean velocity, (*b*) increasing latency and time of h_1 peak, (*c*) decreasing h_1 amplitude, and (*d*) increasing h_2 amplitude. The same trends apear for C2 alone when one compares results for $I_0 = 10^{-6}$ W/cm^2 to those for $I_0 = 10^{-9}$ W/cm^2 (Table II, rows 3 and 12). Thus for both mutants and wild-type these properties seem to reflect the proximity of the operating level I_0 to the threshold of the strain. In other words, the slight abnormalities of the *madA* strains at $I_0 = 10^{-6}$ W/cm^2 are due to their high thresholds. Separate experiments on C47 (data not shown) confirmed the expectation that below the *madA* phototropic threshold the light growth response is negligible.

The adaptation property of the light growth response has the effect, in the model, that the elements in the sensory pathway following the adaptive element are essentially unaware of the absolute intensity. The observation that *madA* strains are practically

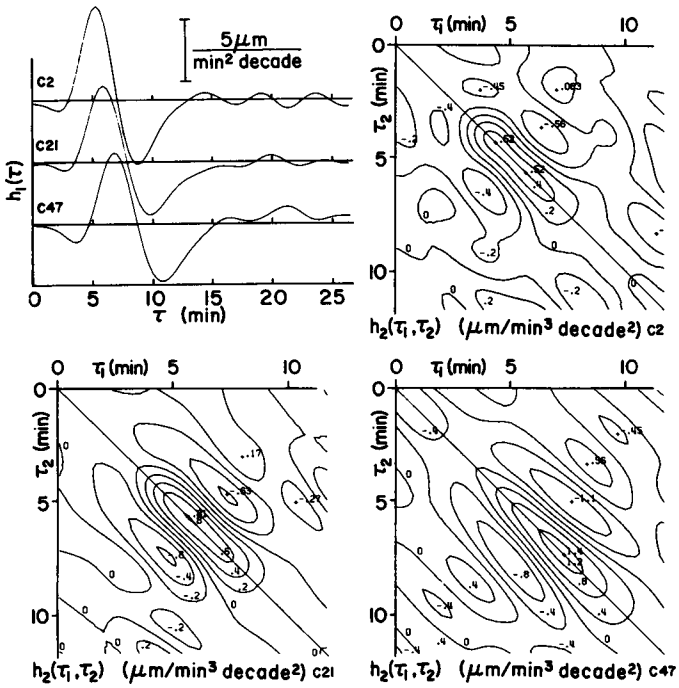

FIGURE 3

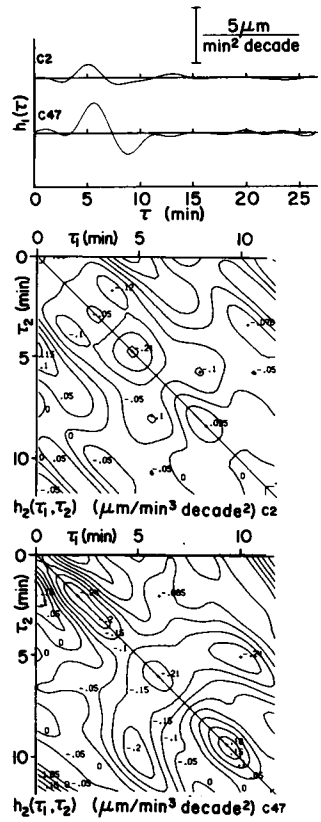

FIGURE 4

FIGURE 3 Kernels for C2 and for *madA* mutants, C21 and C47, at normal intensity $I_0 = 10^{-6}$ W/cm^2.

FIGURE 4 Kernels for C2 and for *madA* mutant C47 at high intensity $I_0 = 10^{-3}$ W/cm^2.

normal above their elevated threshold implies then that their defect occurs no later in the pathway than the adaptation step, which itself is likely to be very early. In particular, the *madA* defect might be associated with the pigment itself. The existence of a pigment mutant would help in identifying and locating the elusive *Phycomyces* photoreceptor.

One test for the integrity of the pigment in a given strain is to examine the diminution of the response characteristics at high intensity, where pigment inactivation overtakes regeneration. Recall from paper II that the high intensity range was defined by a critical intensity $I_c = k/c$ where c is the inactivation cross section and k is the regeneration rate constant of the photoreceptor. A change in either of these parameters would shift the high intensity range. For example, reduction of the inactivation cross-section c would raise I_c and so permit the mutant to "see" better than wild-type at a given high intensity.

In Fig. 4 and in Table II the kernels for C47 at the high intensity $I_0 = 10^{-3}$ W/cm^2

TABLE II

COMPARISON OF MUTANT RESULTS AT SEVERAL INTENSITY LEVELS

Kernels in Fig.	Log-mean intensity	Strain	No. of exp.	Mean velocity	MSE of model response*			Time of h_1 peak	Peak amplitude	
					Zero order	First order	Second order		h_1	h_2
	W/cm^2			$\mu m/min$	$(\mu m/min)^2$	%	%	*min*	$\frac{\mu m/min^2}{decade}$	$\frac{\mu m/min^3}{decade^2}$
3	10^{-6}	C2	7	52	108	22.0	19.3	5.3	7.7	0.52
3	10^{-6}	C21(*madA*)	5	43	95	23.1	21.1	6.0	6.3	0.81
3	10^{-6}	C47(*madA*)	4	33	113	41.2	38.2	6.8	5.7	1.37
4	10^{-3}	C2	4	66	3.5	59.4	51.2	5.1	1.1	−0.21
4	10^{-3}	C47(*madA*)	2	38	22	49.3	47.1	5.6	2.8	−0.21
5	10^{-4}	C2	3	60	21	19.3	15.3	5.0	3.8	(−0.2)
5	10^{-4}	C114 (*madB*)	3	36	13	49.1	49.3	7.0	1.8	0.33
5	10^{-4}	C148(*madC*)	2	39	8.4	46.5	46.5	6.3	1.4	(0.0)
6	10^{-6}	C68(*madD*)	2	40	11	38.6	36.6	5.0	2.1	0.11
6	10^{-6}	C110 (*madE*)	3	46	27	17.1	14.6	5.0	3.6	0.28
6	10^{-6}	C6(*madF*)	4	46	28	35.8	31.7	4.8	3.8	0.30
7	10^{-9}	C2	3	47	49	39.5	35.6	6.6	3.8	0.59
7	10^{-9}	C68(*madD*)	3	47	4.6	44.5	39.1	5.7	1.3	0.17
7	10^{-9}	C110 (*madE*)	2	41	21	37.4	32.8	6.7	2.3	0.52
7	10^{-9}	C6(*madF*)	4	46	1.8	65.6	58.4	6.8	0.6	0.16
8	10^{-6}	NRRL 1555	4	48	103	18.9	17.6	5.3	7.7	0.65

*Mean square errors between experimental and model response records. MSE for zero-order model (h_0) is in absolute units. MSEs for first-order (h_1) and second-order (h_1, h_2) models are given as percentages of zero-order MSE.

are compared to those for C2. Comparing the first-order kernels at $I_0 = 10^{-3}$ W/cm^2 to those in Fig. 4 at 10^{-6} W/cm^2, one finds that C2 is down by a factor of 7, whereas C47 is down only by a factor of 2. In paper II the reduction of the response of C2 at high intensity was analyzed in terms of a model for pigment kinetics, leading in particular to estimates for the pigment parameters. The same fitting procedure has been applied to the C47 data. The resulting parameter values are compared for C47 and C2 in Table III. Taken at face value they imply that *madA* mutants, here represented by C47, have an inactivation cross-section about half that of C2 and a regeneration rate constant somewhat larger. However, considering the large errors of the values in Table III, particularly for C47, the differences between the respective parameters are not highly significant. Moreover the results are no doubt affected by the nearness of the absolute threshold of C47 to 10^{-6} W/cm^2, one of the reference intensity levels used. In any case these possible differences in the pigment parameters are miniscule when set against the enormous difference in absolute thresholds. As a result two hypotheses regarding *madA* may be rejected: the shift in threshold cannot be due to a reduction by a factor of 10^4 of either the total extinction coefficient or the quantum yield for inactivation of the chromophore. Otherwise I_c would have been raised by the same factor. On the other hand, the possibility that the photoreceptor concentration is reduced by 10^4 (resulting perhaps from a leaky nonsense mutation) cannot be ruled out. Such a reduction would raise the threshold but leave the critical intensity unchanged, since the latter is an intrinsic property of the photoreceptor. However, if the quantity, the quality, and the pigment kinetics of the photoreceptor are all unaffected in *madA* strains, then the threshold shift would be due to a reduced efficiency in the link between receptor and the light responses. Further understanding is hindered by our ignorance of the receptor pigment, the nature of its inactivation, and its link to the responses in *Phycomyces*. If the processes of bleaching and adaptation occur separately but in sequence, then one would expect the *madA* defect to lie between the two steps. Further analysis of the *madA* strains by physiological, biochemical, and genetic techniques should help to clarify the important early steps of the light response pathway.

TABLE III

COMPARISON OF PIGMENT PARAMETERS FOR C47 and C2

Strain	Regeneration rate constant k	Inactivation rate ($@ I_o$) cI_o	Inactivation cross section ϵ^I_{488}	Partial extinction coefficients for inactivation: ϵ^I_{488}	ϵ^I_{455}	Critical intensity ($@$ 488 nm) I_c
			X 10^{17}	X 10^{-4}	X 10^{-4}	
	min^{-1}	min^{-1}	cm^2	liter/mol·cm	liter/mol·cm	$\mu W/cm^2$
C47	1.5 ± 1.3	2.5 ± 1.5	1.7 ± 1.0	0.4 ± 0.3	0.6 ± 0.4	600 ± 300
C2	0.37 ± 0.08	6.1 ± 0.5	4.0 ± 0.4	1.1 ± 0.1	1.5 ± 0.2	60 ± 10

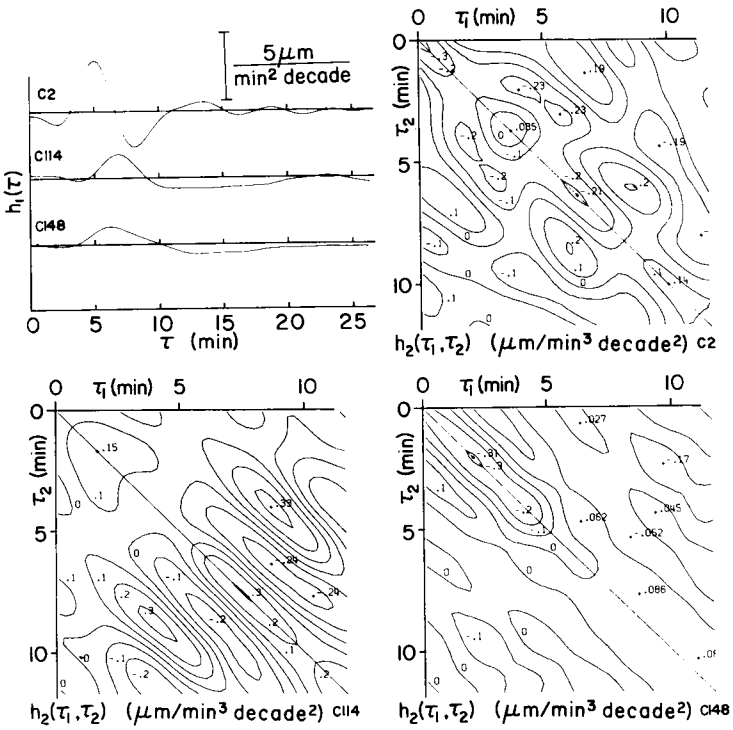

FIGURE 5 Kernels for C2 and for mutants C114 (*madB*) and C148 (*madC*) at moderate intensity $I_0 = 10^{-4}$ W/cm^2.

madB and madC Mutants

In Fig. 5 the kernels for C114 (*madB*) and C148 (*madC*) are compared to C2, all at $I_0 = 10^{-4}$ W/cm^2. That intensity was chosen because of the high phototropic threshold of both strains (Fig. 2). It was verified separately that both strains have negligible response at $I_0 = 10^{-6}$ W/cm^2. Despite the fact that I_0 was above threshold, the kernels are small. Thus while *madA* strains are practically normal above threshold, this does not appear to be the case for *madB* or *madC*. Experiments at 10^{-3} W/cm^2 (data not shown) indicated that the response was considerably less than at 10^{-4} W/cm^2, thus ruling out the possibility that either strain might have a significantly higher I_c and concomitant larger response at high intensity. Thus even at their optimal intensity range both strains have severely degraded responses. Like C47, both strains here have significantly lower mean growth velocity (Table II). Note that all kernels, as shown, have been scaled up by a factor $45/\bar{V}$, where $\bar{V}$ is the mean velocity in micrometers per minute as given in Table II. Without this normalization the kernel magnitudes of the *madB* and *madC* mutants would be proportionately still smaller. As with *madA*, both strains have longer latency than C2. Again this probably reflects the closeness of their thresholds to I_0. There are two minor differences between the *madB* and the *madC* mutants. First the h_1 kernels have slightly different shape, that of C148 being more

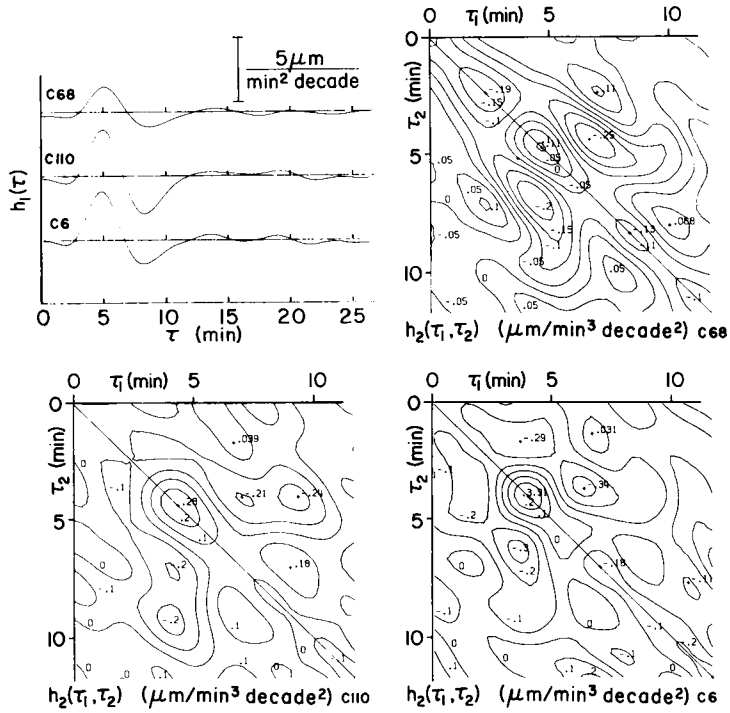

FIGURE 6 Kernels for class 2 mutants C68 (*madD*), C110 (*madE*), and C6 (*madF*) at normal intensity $I_0 = 10^{-6}$ W/cm^2. The corresponding kernels for C2 have been shown in Fig. 3.

shallow on the downslope. Secondly, the h_2 kernels are rather different, that of C114 appearing more normal and sizable. However, unlike all other cases studied in these three papers, the second-order model response in both cases has no better mean-square-error than the first-order. Thus the importance of the difference in the h_2 kernels must be downrated.

madD, madE and madF Mutants

Class 2 ("stiff") mutants show abnormal growth and tropic responses to other stimuli besides light, and are therefore presumed to have their defects near the *output* of the common growth response pathway (Fig. 1). In this class, three complementation groups, *madD, madE,* and *madF,* have been identified. In Fig. 6 kernels at $I_0 = 10^{-6}$ W/cm^2 are shown for representatives of these three groups, respectively, C68, C110, and C6. One notices at once that the shapes of the kernels are normal (see Fig. 3 for the standard kernels of C2). However the magnitudes are all attenuated significantly. For both C110 and C6 the h_1 and h_2 amplitudes are about half of that for C2, whereas for C68 the h_1 and h_2 amplitudes are down fourfold (see last two columns of Table II). The observation that both h_1 and h_2 scale proportionately is in accord with the notion that the defects occur at the output. The defects are thus interpretable simply as gain changes in one or more output stages. In the electronic analog circuit in Fig. 6 of

paper I the class 2 defects could be simulated by suitably reducing the gain of the output amplifier, or more generally the gains of any of the stages following the nonlinear element(s).

The class 2 kernels were studied also at 10^{-9} W/cm^2 (Fig. 7) to clarify whether the shallow threshold curves of C68 (*madD*) and C110 (*madE*) actually have intercepts at the same threshold as wild-type. Bergman et al. (1973) had interpreted the class 1 and class 2 threshold curves in terms of a pathway consisting of a logarithmic transducer preceded and followed by linear transducers. They suggested that class 1 mutants have the gain of the input linear transducer attenuated, while the class 2 mutants have the gain of the output linear transducer attenuated. Essential to their model was the assumption that the threshold of class 2 mutants is the same as that for wild-type. While in Fig. 2 the validity of this assumption is clear for C6, it is by no means clear for C68 or C110 because of the shallowness and uncertainty of the corresponding threshold curves. Examining the last columns of Table II for $I_0 = 10^{-6}$ and 10^{-9} W/cm^2 one finds that the kernel amplitudes for C68 and C110 change comparably to the C2 amplitudes. This supports the belief that class 2 mutants have the same threshold as wild-type.

The present results for C6 appear anomalous when compared to the C6 threshold curve in Fig. 2. One would have expected the C6 kernels to be comparable to those for C2 at 10^{-6} W/cm^2 and to be greater at 10^{-9} W/cm^2 than those for C68 and C110. Instead, at 10^{-6} W/cm^2 the kernels are only about half of those for C2. At 10^{-9} W/cm^2 the C6 kernels are *smaller* than those for C68 and C110. Conversely, one wonders why the responses for C68 and C110 are so sizable both at 10^{-6} and 10^{-9} when the phototropic response curves are so shallow.

At this point it is worth remembering that the light growth response is presumed to be an averaged expression of the local response responsible for phototropism. Furthermore, remember that the ordinate for the phototropic response curve of Fig. 2 is actually an equilibrium angle for the competing processes of positive phototropism to a horizontal light beam and of negative geotropism. From the present results it would appear that the relation between the light growth response and this equilibrium angle is complex, at least insofar as absolute intensity dependence is concerned. A simpler relation may exist between the light growth response and the phototropic bending rate. Measurements of phototropic bending rate for the stiff mutants as a function of intensity would be helpful in clarifying this issue.

Comparison of Kernels for C2 and Wild-Type

In papers I and II all experiments were performed on the albino strain C2 rather than the wild-type NRRL1555. In the present paper C2 was used as the standard for comparison of photomutants. Although C2 is missing 99% of the bulk-pigment β-carotene (Meissner and Delbrück, 1968) it seems to have photophysiology virtually identical to wild-type. This observation has been used to discount the hypothesis that β-carotene is the receptor pigment. In Fig. 2 the phototropic response curves for C2 and wild-type coincide. As a footnote to these papers on the white noise analysis of the

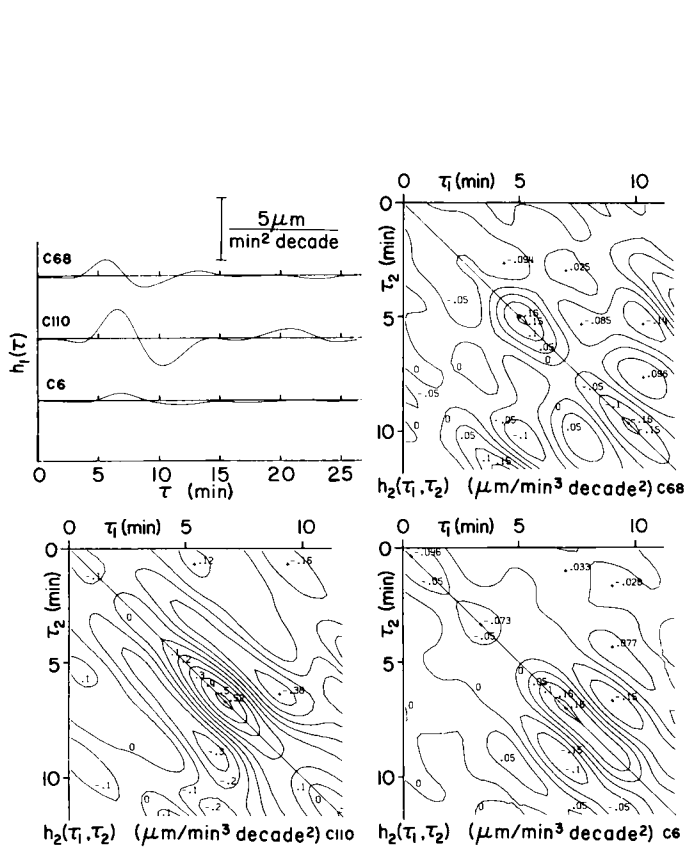

FIGURE 7

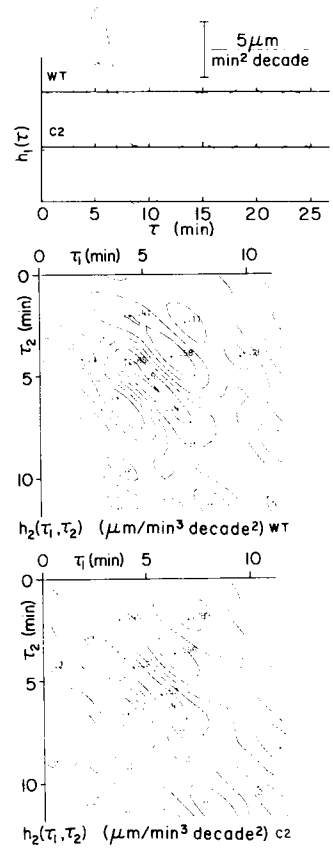

FIGURE 8

FIGURE 7 Kernels for class 2 mutants C68 (*madD*), C110 (*madE*), and C6 (*madF*) at low intensity $I_0 = 10^{-9}$ W/cm^2. The corresponding kernels for C2 are in Fig. 4 of paper II.

FIGURE 8 Comparison of kernels for wild type (WT) strain NRRL1555 and the albino strain C2.

light growth response it is worth comparing the Wiener kernels for the two strains. Fig. 8 shows that the kernels for the normal reference level $I_0 = 10^{-6}$ W/cm^2 are virtually identical for the two strains. This finding justifies the use of C2 data and kernels as if they were those of wild-type. The virtual absence of the bulk screening pigment in C2 makes that strain advantageous over wild-type for most photobiological work, particularly spectrophotometric work (Poff and Butler, 1974) in search of the photoreceptor.

DISCUSSION

The purpose in isolating and studying behavioral mutants is to permit a partial genetic dissection of the stimulus-response pathway(s) responsible for the behavior. The determination of physiological classes and complementation groups (Fig. 1) and mea-

surement of phototropic thresholds (Fig. 2) are important steps in this direction for *Phycomyces* research. The measurement of Wiener kernels for the fundamental light growth response of the mutants provides a deeper characterization of their defects.

The relationship between the light growth response and phototropism is still being worked out experimentally and theoretically (Dennison and Bozof, 1974).[1] The fact that all phototropically abnormal mutants have defective light growth responses provides genetic support for the presumed connection between the responses. Furthermore models of phototropism should be guided by and account for the relationships between abnormalities of the two response modes in the various mutants. The phototropic-geotropic equilibrium bending angle (Fig. 2), while valuable for determining phototropic thresholds, is probably not the best variable to compare to the light growth response characteristics. The interaction between geotropism and phototropism is complex and poorly understood. A better basis for comparison would be phototropic bending rates at a number of intensity levels.

On the whole, the defects found in the kernels are not as gross and varied as might have been expected. Aside from differences in gain (amplitude), the mutant kernels all have the bipolar shape and general time course exhibited by the kernels in papers I and II. The amplitudes of the kernels are abnormal and depend on absolute intensity in ways that are characteristic for the strain.

The responses of the two *madA* strains C21 and C47 were essentially normal provided they were adapted and tested at intensities above their thresholds. At high intensity the response of C47 was not appreciably different from that of wild-type. Thus the *madA* defect does not seem to affect the pigment inactivation and regeneration processes. In other words the *madA* gene does not seem directly associated with the pigment. Similarly none of the other mutants studied showed any evidence of alterations in the photoreceptor that would affect their behavior at high intensity.

The responses of C114 (*madB*) and C148 (*madC*) remained abnormally small even above their very high thresholds. On the whole the two representatives of *madB* and *madC* are very similar to one another and very different from all other strains studied. This may seem surprising, since the two complementation groups do not even fall in the same physiological class (Fig. 1). However the distinction between classes 1.1 and 1.2 is based solely on *mycelial* photoresponses whereas the pathway of Fig. 1 otherwise is based on the growth responses of the mature sporangiophore. Thus for the developmental stage studied here there is actually no known physiological separation between *madB* and *madC*. Both are quite blind but have normal output systems. The similarity between *madB* and *madC*, as opposed to *madA*, may imply that the two loci are closely related functionally. Aside from the difference in mycelial photoresponses one might even suspect the possibility of interallelic complementation, i.e. that *madB* and *madC* might really belong to the same gene. As part of the genetic mapping in progress

[1] Dennison, D. S., and K. W. Foster. 1975. Intracellular rotation and the phototropic response of *Phycomyces*. In preparation.

(Eslava et al., 1975) it will be particularly valuable to establish the degree of linkage of these two complementation groups.

Kernels for the three class 2 mutants studied, C68 (*madD*), C110 (*madE*), and C6 (*madF*), have normal time course but diminished amplitude depending progressively on intensity. This feature is compatible with a reduction of the gain of one or more output stages. The sizable amplitude of the class 2 mutant kernels at low intensity supports the hypothesis that class 2 mutants have the same absolute threshold as wild-type.

The limited scope of the distortions in the available mutants indicates that more photomutants are needed. All of the present mutants were obtained after mutagenization with nitrosoguanidine and screening for abnormal phototropism using the "glass bottom box" method (Bergman et al., 1973). To obtain a broader range of mutants different mutagenesis procedures and selection methods are needed.

Two phenotypes that would be especially valuable are (*a*) total blindness at any intensity and (*b*) alteration of the photoreceptor complex. The former should be obtainable by the standard screening method with different mutagens and more intense effort, using temperature sensitivity if such a mutation should happen to be lethal.

A search for pigment mutants is now in progress in this laboratory. The procedure includes screening for "bright-seeing" mutants that show phototropism at high intensity where wild-type fails to respond. Conversely "bright-blind" mutants are being sought by screening for absence of phototropism at moderate intensity where wild-type does still bend. The availability of pigment mutants would provide an optimum assay for biochemical and spectrophotometric studies in search of the photoreceptor. In addition pigment mutants might be helpful for comparative photophysiology, in particular to investigate the role of the photoreceptor in adaptation.

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