tropic translational librations, but a prominent rotational libration of 8° around the chain axis.

The chain orientation in this molecule is consistent with the previously reported crystal structures of two 1,3-diglycerides (*18*) and the α,β chain conformation in three triglyceride crystal structures (*19*) where the hydrocarbon chains are extended. This result is a possibility which was excluded in early interpretations of x-ray powder spacings from glycerides (*20*).

From work on the structure of *rac*-glycerol 1,2-(di-11-bromoundecanoate)-3-(*p*-toluenesulfonate) (*21*), it appears that the short, more rotund shape of a phospholipid polar head group is important in determining the packing arrangement. Yet, the presence of extended chain packing, as demonstrated by this diester, suggests another possible stable phospholipid acyl chain configuration for biological membranes, especially if nonpolar lipid-protein interactions are significant.

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$$R = \frac{\Sigma||F_{obs}| - |F_{calc}||}{\Sigma|F_{obs}|}$$

where F is the structure factor.
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Reversible Inhibition of Chloroplast Movement by Cytochalasin B in the Green Alga Mougeotia

Abstract. *Light-oriented chloroplast movement is reversibly inhibited by cytochalasin B. The photoperception is not influenced by this inhibitor. These results support the assumption that contractile protein fibrils are essential for this intracellular movement.*

In the filamentous alga *Mougeotia*, the single large plate-shaped chloroplast performs orientation movements with respect to light. In blue or white light of high intensity, the chloroplast orients its edge to the light, whereas in white light of low or medium intensity or in red light it orients its face to the light (*1*). We now discuss the latter response, which is mediated mainly by phytochrome ("low intensity movement").

Compared with our knowledge about the nature of the photoreceptor and some details about its localization (*1*), much less is known about the mechanism of movement. Microtubules that have been demonstrated in the cytoplasm (*2*) appear not to be involved in the movement of the chloroplast, but fibril structures have been found and are assumed to be involved in the movement (*3*).

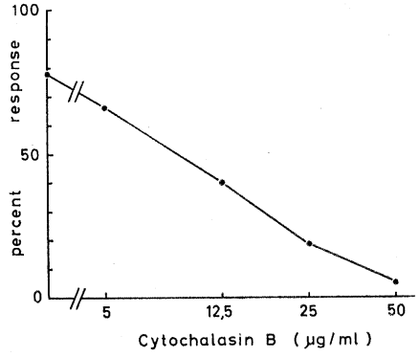

Fig. 1. Light-induced chloroplast movement as influenced by cytochalasin B. On the ordinate, the percentage of cells is given in which orientation of the chloroplast is found 40 minutes after a pulse of red light.

Cytochalasin has been reported to be a very specific inhibitor of contractile protein fibrils (*4*). Therefore we have investigated the effect of this substance on chloroplast movement. The *Mougeotia* cell is particularly suitable for investigating possible roles of protein fibrils in the mechanism of chloroplast movements. If the movement is induced by a short exposure to light (even less than 1 minute), it proceeds during a period of complete darkness following this light (*1*); moreover, the effect of such a short light induction can be stored by the cell for some time if the response is blocked (*5*). We therefore can separate experimentally the effect of an inhibitor on the photochemical reaction from that on the response.

Preparations of *Mougeotia* were first treated by appropriate irradiation so as to be oriented in profile position (*6*). Before the inductive irradiation was applied, the "MXS" culture medium (*7*) was removed and replaced by medium containing cytochalasin B (Serva) in increasing concentrations (0, 5, 12.5, 25, and 50 µg/ml). Fifteen minutes later, all samples were subjected to unpolarized red light for 60 seconds; this pulse was equivalent to the physiological effect of 1500 erg/cm² sec of 683-nm light (*8*). Immedi-

ately after the treatment with light, the samples were stored in complete darkness for 40 minutes. The percentage of cells in which the chloroplast had turned to face position was scored. From induction until scoring, all manipulations were conducted in green safelight (*6*).

With increasing concentrations of cytochalasin B, the response dropped to nearly zero (Fig. 1); the result is the same if the inhibitor was applied only 5 minutes before induction rather than 15 minutes before. The effective concentrations of cytochalasin and the rapid effect agree with results in other systems (*9*).

We then tested the reversibility of the inhibition. After the response was scored, the samples were rinsed with inhibitor-free medium (all manipulations in green safelight). The response induced by a second treatment with red light compares well with that of the controls for cytochalasin concentrations of 12.5 μg/ml or less. It is only after inhibition with a concentration of 50 μg/ml that the response is not restored completely (Fig. 2).

Another experiment was designed to ascertain whether photoperception or the mechanism of movement is affected by cytochalasin. Immediately after the induction, the inhibitor was replaced by control medium, and the normal response was obtained during the following dark period of 40 minutes in spite of the fact that the inhibitor was acting during the induction. This result shows that the mechanism of movement rather than the photochemical process is blocked by cytochalasin B, and confirms the reversibility of this inhibition.

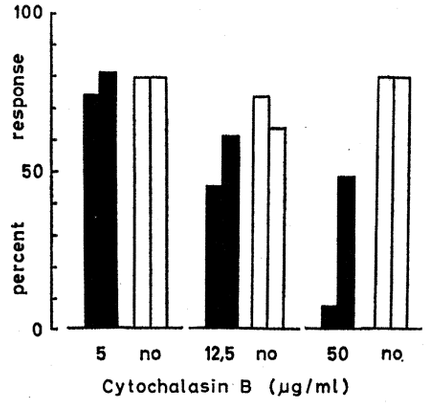

Fig. 2. Light-induced chloroplast movement with and without cytochalasin B. In each pair of black columns, the left column shows the response after the first induction under the influence of different concentrations of cytochalasin; the right column shows the response of the same sample after the inhibitor is replaced by control medium and treated again by light. White columns, controls without cytochalasin B. Ordinates are as in Fig. 1.

From these experiments, which confirm similar results of Schönbohm [in preparation, as suggested by (*3*)], we conclude that contractile protein fibrils are essential for the mechanism of chloroplast movement in *Mougeotia*.

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Hyperprolinemia and Prolinuria in a New Inbred Strain of Mice, PRO/Re

Abstract. *A hyperprolinemia was discovered, in a new inbred strain of mice, which was equivalent to about a sevenfold elevation above the concentration of proline in the blood of either of the original parental lines, or of 12 other inbred strains with diverse genetic constitution. In addition, mice of this PRO/Re strain exhibited a marked prolinuria, whereas the other 14 inbred strains had no proline detectable in their urine.*

Genetic aberrations that affect amino acid metabolism are documented for various organisms. Studies on mutations in *Neurospora* (*1*), for example, which block the biosynthesis of tryptophan (*2*) helped to establish the biochemical link between gene action and control of enzyme activity. Approximately 40 hereditary disorders of amino acid metabolism in humans are known, many of which are due to defective regulation of liver enzyme function (*3, 4*); two such disorders involve the heterocyclic amino acids proline and hydroxyproline. Hyperprolinemia and hydroxyprolinemia are rare "inborn errors of metabolism" characterized by an elevation of the concentration of proline or hydroxyproline in plasma. These amino acids also accumulate in the urine. At least two variations of hyperprolinemia are known in man (*5*). Type I hyperprolinemia is characterized by a hereditary renal disease, congenital anomalies of the genitourinary tract, and mild mental retardation. Type II hyperprolinemia has been accompanied by mild mental retardation, but neither the patient nor any of his relatives had renal disease.

Recently we investigated various inbred strains and mutant stocks of mice for inherited diseases of enzyme control that affect amino acid metabolism. During these studies, one particular line (*6*), since designated PRO/Re, was observed to excrete in the urine a substance that produced a yellow spot on paper chromatograms treated with ninhydrin.

Abnormal excretion of proline in the urine of PRO mice was first detected by the resolution of amino acids in urine with paper chromatography. Fresh, untreated urine, in amounts varying from 10 to 30 μl, was spotted on Whatman No. 1 paper and developed with a water-saturated phenol solvent for about 24 hours in a descending chromatography system. The amino acids were detected by means of the ninhydrin reaction; proline produced its characteristic yellow spot. In other experiments, an isatin reagent, 0.2 percent solution of isatin in *n*-butanol acidified to 5 percent glacial acetic acid, was utilized to detect proline on either paper chromatograms or paper electrophoretigrams.

Figure 1 is a paper chromatogram on which proline, after being resolved from the urine of two PRO/Re mice, was treated with the isatin reagent to give the characteristic blue-colored product. A proline standard was present in the same chromatographic position as the substance detected as urine proline.