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Inhibitor Studies on Formation of Giant Mitochondria in *Nitella flexilis*

By

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With 7 Figures (2 Plates)

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Summary

Foissner I. 1983. Inhibitor studies on formation of giant mitochondria in *Nitella flexilis*. — Phyton (Austria) 23 (1): 19—29, with 7 figures (2 plates). — English with German summary.

The formation of giant mitochondria in the characean alga *Nitella flexilis* was investigated with the aid of inhibitors. Experiments with 3-(3,4-dichlorophenyl)-1,1-dimethylurea (DCMU) showed that elongation occurs when photosynthesis is reduced. Inhibitors of complexes III and IV of the respiratory chain also induce the formation of giant mitochondria but not the inhibitor of complex I, rotenone, or the uncoupling agent 2,4-dinitrophenol (DNP). It is supposed that reduced photosynthesis and reduced cytochromal activity act on mitochondrial length indirectly via a common metabolic process. The formation of giant mitochondria during treatment with DCMU is sensitive to inhibitors of protein synthesis in cytoplasm or organelles. Elongation probably proceeds via blockage of mitochondrial fission and enhanced readiness to fuse.

Zusammenfassung

Foissner I. 1983. Inhibitor-Studien zur Bildung von Riesenmitochondrien in *Nitella flexilis*. — Phyton (Austria) 23 (1): 19—29, mit 7 Abbildungen auf 2 Tafeln. — Englisch mit deutscher Zusammenfassung.

Die Bildung von Riesenmitochondrien in der Characee *Nitella flexilis* wurde mit Hilfe von Inhibitoren untersucht. Versuche mit 3-(3,4-Dichlorphenyl)-1,1-Dimethylharnstoff (DCMU) ergaben bei eingeschränkter Photosynthese eine Verlängerung der Mitochondrien. Auch Inhibitoren der Komplexe

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III und IV der Atmungskette führen zu Riesenmitochondrien, nicht aber der Inhibitor des Komplexes I, Rotenon, oder der Entkoppler 2,4-Dinitrophenol (DNP). Eingeschränkte Photosynthese und verminderte Cytochromaktivität beeinflussen die Mitochondrienlänge vermutlich indirekt über einen gemeinsamen Stoffwechselweg. Die Bildung von Riesenmitochondrien durch DCMU ist gegenüber Stoffen, die Proteinsynthese im Cytoplasma oder in den Organellen hemmen, empfindich. Die Verlängerung beruht offenbar auf einer Blockade der Mitochondrienteilung und erhöhter Neigung zum Verschmelzen.

1. Introduction

Giant mitochondria have been described in a number of organisms and cells. They were detected in the internodal cells of the characean algae *Nitellopsis obtusa* (JAROSCH 1961), and *Nitella flexilis* (FOISSNER 1981), both of which plants were grown in a natural environment and were harvested during the winter season. Culture experiments with *N. flexilis* showed that elongation of mitochondria occurs when the light intensity is strongly reduced (FOISSNER 1981). The present study was undertaken to elucidate the metabolic background of giant mitochondria-formation with the aid of inhibitors.

2. Material and Methods[1])

Nitella flexilis was collected from the Hellbrunner Bach in Salzburg and determined according to WOOD & IMAHORI (1965). The plants were cultivated in Forsberg-Medium II (FORSBERG 1965) at a light intensity of about 15×10^1 μEinstein.$m^{-2}.s^{-1}$ (14 h light, 8 h dark) and a temperature of 20° C. Only dactyls, i. e. internodes of the branchlets were used during these experiments. They were isolated at least 3 d prior to incubation and maintained under experimental conditions (40 μEinstein.$m^{-2}.s^{-1}$, 14 h photoperiod, 20° C) in APW (1×10^{-3} M NaCl, 1×10^{-4} M KCl, 1×10^{-4} M $CaCl_2$). The incubation lasted 2 d (DCMU, CCCP, antimycin A, NaN_3), 3 d (DNP, rotenone; CAP, actidione) and 8 d (KCN).

DCMU, KCN, NaN_3, DNP, CAP and actidione were dissolved in APW. Stock solutions of antimycin A (10^{-2}% in 2% DMSO), CCCP (10^{-2}% in 10% DMSO) and rotenone (10^{-2}% in 2.5% DMSO) were diluted with APW. KCN was purchased from Merck; all other chemicals from Serva.

The light intensity was determined with a quantum-photometer (Lambda Instruments, Nebraska) inside the water of the culture vessels.

The length of the mitochondria was measured under the light microscope in the living cell. The term giant mitochondria was used for organelles

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[1]) Abbreviations: APW = artificial pond water, CAP = d-threo chloramphenicol, CCCP = carbonylcyanide.m-chloro-phenylhydrazone, DCMU = 3-(3,4-dichlorophenyl)-1,1-dimethylurea, DMSO = dimethylsulfoxide, DNP = 2,4-dinitrophenol.

exceeding 5 μm in length. The statistical analysis was performed according to MÜHLENBERG (1976).

The method for preparing dactyls for electron microscopy has been described previously (FOISSNER 1981).

3. Results

3.1. Effects of DCMU, CCCP, KCN, antimycin A and NaN_3

Light microscopy. The cytoplasm of the characean internodes consists of a stationary layer including rows of chloroplasts (cortex) and the streaming endoplasm. Mitochondria of the former were easy to observe because of their relatively fixed position immediately beneath the cell wall. In order to measure the length of endoplasmic mitochondria it was necessary to choose a site where chloroplasts were absent or at least contained few and small starch granules. Active movement of mitochondria (see for instance JAROSCH 1978) did not occur in *Nitella flexilis*.

All above cited inhibitors induced formation of giant mitochondria in the endoplasm as well as in the cortex. The proportion of giant mitochondria in the total number of mitochondria was estimated not to exceed 20% (compare FOISSNER 1981). Lengths up to 50 μm had been observed whereas in control cells they very rarely exceeded 2 μm. The inhibitor concentrations, the mean values of the maximum mitochondrial lengths per cell and their statistical analysis are given in table 1. It must be noted that during each experiment about each fifth cell did not respond to the addition of an inhibitor by forming giant mitochondria which possibly reflects a different physiological status. Giant mitochondria were mostly threadlike. Branched organelles were present mainly in the endoplasm whereas ringlike forms occurred preferentially in the cortex. The diameter of mitochondria was generally independent of their length and did not exceed 1 μm. Higher values had been obtained with KCN-treatment. These mitochondria often appeared disc- or plate-like.

Electron microscopy. DMSO at those concentrations used to dissolve CCCP and antimycin had no effect on cellular fine structure in comparison with APW.

The fine structure of mitochondria in cells treated with DCMU, antimycin or NaN_3 was the same as in control cells (Figs. 1—4). No structural difference could be found between short and giant mitochondria. Both had the same density of the matrix and the same arrangement of the cristae. Sections of cells incubated with KCN showed mitochondria at different degrees of swelling (Fig. 7). The matrix appeared more or less electron-transparent. The intracristal space was sometimes very small, with membranes lying closely against one another; on the other hand sometimes it appeared extremely enlarged. The number of cristae was reduced. Their complete absence left mitochondria which consisted only of a concentric

Table 1

Statistics of the effects of DCMU, CCCP, KCN, antimycin A and NaN_3. All values are based on the maximum mitochondrial length per cell, given in μm. Plus signs indicate significance at 5% between controls and treated cells (U-test). Control solutions: APW (for DCMU, KCN, NaN_3), 1×10^{-4}% DMSO (for CCCP), 2×10^{-1}% DMSO (for antimycin A). $\bar{x}$ = arithmetic mean, M = median, SD = standard deviation, $SE_{\bar{x}}$ = standard error of the mean. CV = coefficient of variation, n = number of cells analysed

Inhibitor	concentration (%)	$\bar{x}$	M	SD	$SE_{\bar{x}}$	CV	range	U-test	n
DCMU	0	2.0	2.0	0.0	0.0	0.0	2–2		8
(pH 6.0–6.2)	1×10^{-4}	9.0	9.0	6.1	1.8	68.0	2–20	+	12
	1×10^{-5}	13.3	16.0	6.7	2.0	50.2	2–24	+	11
	1×10^{-6}	2.7	2.0	1.5	0.4	55.2	2–6	–	12
CCCP	0	3.7	2.0	2.2	0.8	60.8	2–8		7
(pH 5.4)	1×10^{-5}	all cells dead							8
	1×10^{-6}	8.7	8.0	1.9	0.7	21.9	6–12	+	7
	1×10^{-7}	2.0	2.0	0.0	0.0	0.0	2–2	–	6
KCN	0	2.7	2.0	1.1	0.4	40.9	2–5		6
(pH 6.2–6.5)	1×10^{-3}	19.2	20.0	7.9	2.7	40.9	2–30	+	12
	1×10^{-4}	15.0	19.0	7.3	2.6	49.0	2–24	+	8
	1×10^{-5}	18.5	14.0	14.1	4.5	76.3	2–50	+	10
	1×10^{-6}	12.2	11.0	6.1	4.7	50.1	2–24	+	8
Antimycin A	0	4.8	2.0	3.1	0.9	64.4	2–10		13
(pH 6.0)	1×10^{-3}	19.1	19.0	8.1	2.3	42.3	2–32	+	12
	1×10^{-4}	15.2	19.5	7.5	2.6	49.3	2–24	+	8
	1×10^{-5}	18.4	15.0	14.1	4.4	76.6	2–50	+	10
	1×10^{-6}	12.2	11.0	6.1	2.2	50.1	2–25	+	8
NaN_3	0	2.4	2.0	0.7	0.2	29.0	2–4		8
(pH 5.1)	1×10^{-1}	5.4	5.0	3.4	1.3	62.5	2–12	+	7
	1×10^{-2}	7.6	8.0	4.8	1.4	63.3	2–18	+	11
	1×10^{-3}	5.6	2.0	5.2	1.7	92.9	2–18	–	9

layer of two membranes. The disc-like mitochondria observed with the light microscope turned out to be cup-shaped mitochondria in many cases. Swollen mitochondria which were mainly disc-like occurred occasionally in cells treated with CCCP (Fig. 5). They were then preferentially located between the chloroplasts. However, most mitochondria, including the giant ones, had a normal fine structure. A close association existed also between chloroplasts and mitochondria-like organelles with an extremely electron-dense matrix. They had been observed very rarely in CCCP-treated cells. Mitochondria with a similar "condensed" conformation were demonstrated

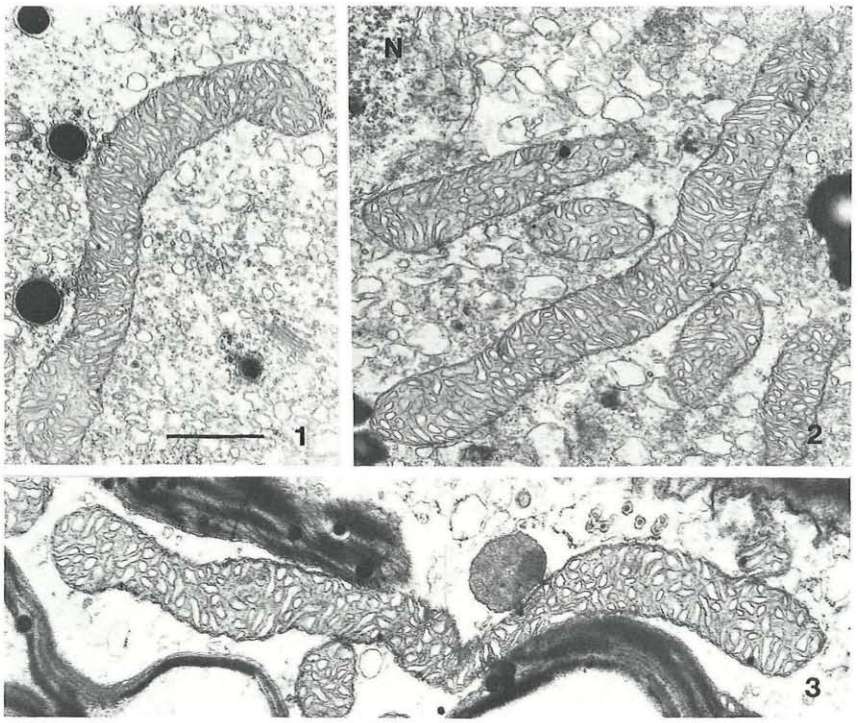

Plate 1

Figs. 1—3. *Nitella flexilis*, dactyl cell. Formation of giant mitochondria with normal fine structure by 1×10^{-4}% DCMU (Fig. 1), 1×10^{-2}% NaN_3 (Fig. 2), 1×10^{-6}% antimycin A (Fig. 3). N = nucleus. The bar indicates 1 μm

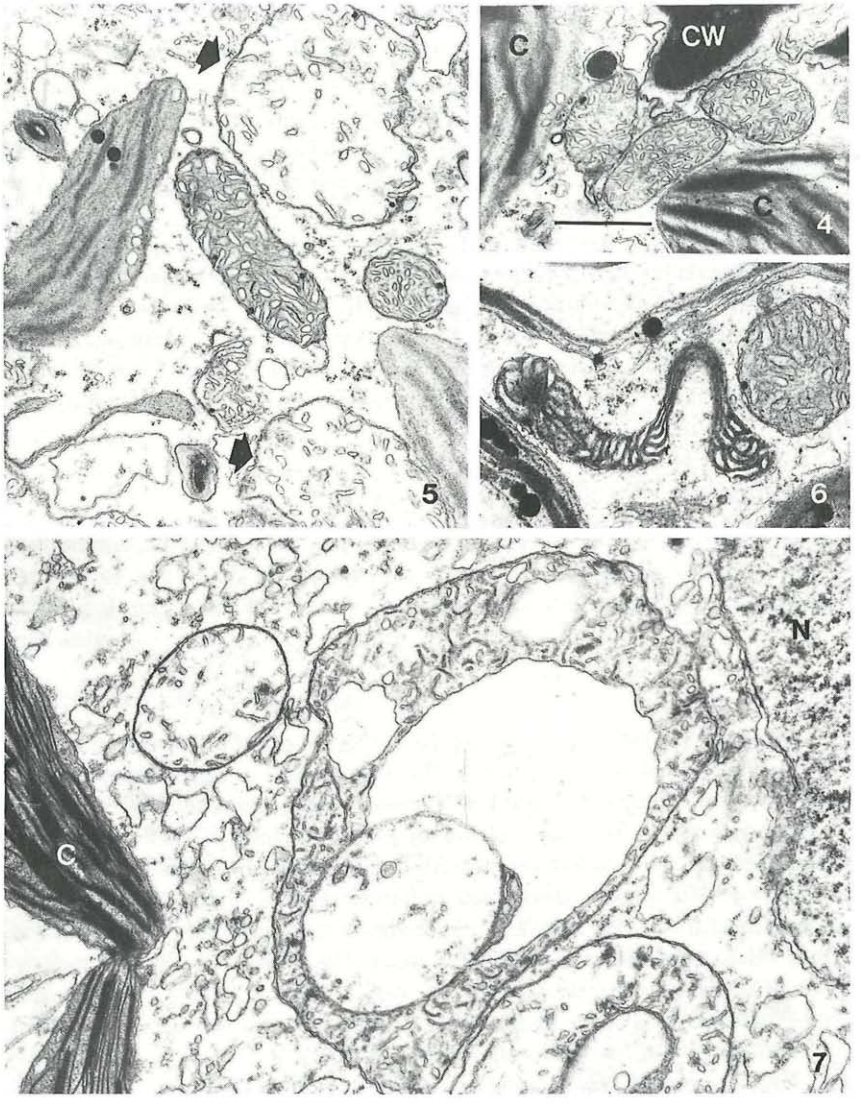

Plate 2

Fig. 4. *Nitella flexilis*, control cell. Normal mitochondria. C = chloroplast, CW = cell wall. The bar indicates 1 μm

Figs. 5—6. *Nitella flexilis*, dactyl cell. Treatment with 1×10^{-6}% CCCP

Fig. 5. Swollen mitochondria with clear matrix (arrows) and mitochondria with normal fine structure. Cytoplasm with numerous enlarged vesicles

Fig. 6. Mitochondrion-like organelle with darkly stained matrix

Fig. 7. *Nitella flexilis*, dactyl cell. Treatment with 1×10^{-3}% KCN. Swollen mitochondria. Cytoplasm vacuolated. C = chloroplast, N = nucleus

in de-aerated or CCCP-treated *Saccharomyces* cells (LUZIKOV 1973, cited in LLOYD 1974; see also THOMSON *et al.* 1972).

The residual cytoplasm was best preserved in cells incubated with DCMU, though severe disorganization of chloroplasts and mitochondria after DCMU-treatment has been reported in *Euglena* sp. (CALVAYRAC *et al.* 1979). The cytoplasm could not be distinguished from that of control cells. It appeared slightly less dense after treatment with antimycin or NaN_3 because of an enlargement of vesicles. The increased number of vesicles observed during CCCP-incubation was apparently due to enhanced activity of the dictyosomes. Irregularily shaped vacuoles were very prominent in KCN-poisoned cells. Chloroplasts had a normal fine structure and contained starch grains in all cells. A few damaged chloroplasts with various degrees of disorganization were observed only after KCN-treatment.

3.2. Effects of rotenone and DNP

Neither rotenone (1×10^{-2}—1×10^{-8}%) nor DNP (3×10^{-3}—3×10^{-7}%) affected the mitochondrial length in *Nitella flexilis*. DNP was used in concentrations which do not influence photosynthetic ATP-formation (NEUMANN & JAGENDORF 1964). Studies were performed with the light microscope only.

3.3. Effects of actidione and CAP

In order to test the necessity of protein synthesis for formation of giant mitochondria, actidione or CAP was added to a 1×10^{-5}% solution of DCMU. The influence of these substances was only studied with the light microscope. The incubation time was 3 days. 80% of the control cells in 1×10^{-5}% DCMU then had mitochondrial lengths of over 15 μm. Formation of giant mitochondria was prevented in the presence of 1×10^{-4}% actidione or 1×10^{-2}% CAP. These concentrations are sufficient to inhibit protein synthesis in the cytoplasm (MCMAHON 1975) and in the organelles (mitochondria and chloroplasts) respectively (DAVIS & MERRETT 1975). Cells in these solutions contained only mitochondria with a maximum length of 2 μm. The influence of 10^{-2}% CAP dissolved in 10^{-5}% DCMU was deadly for more than 50% of the cells. Neither CAP without DCMU, nor actidione without DCMU — i. e. only dissolved in APW — affected the mitochondrial lengths. Contrary results exist, for instance, in *Euglena* cells (NEUMANN & PARTHIER 1973) and yeast cells (KELLERMANN *et al.* 1969).

4. Diskussion

The inhibitors, which were tested for their ability to induce formation of giant mitochondria in *Nitella flexilis*, affect either photosynthesis and/or respiration.

DCMU specifically inhibits the Hill reaction at those concentrations which induce elongation of mitochondria (IZAWA 1977, CHEVALLIER & DOUCE 1976). That mitochondria are not directly influenced by DCMU has been shown in a series of papers (BISHOP 1958, DOWNTON & TREGUNNA 1968, CHAPMAN & GRAHAM 1974, GAUVRIT 1978). The interpretation of giant mitochondria-formation in *Nitella flexilis* as a result of reduced photosynthesis is thus confirmed (FOISSNER 1981). The existence of small starch granules in the chloroplasts even at the highest concentration indicates that photosynthesis is not completely restricted. This agrees with the observation that elongation occurs only at reduced light intensity but not in absolute darkness (FOISSNER, not published).

The results obtained with DCMU are further strengthened by CCCP-treatment which acts as an uncoupler of photophosphorylation (GOOD & IZAWA 1973, FUJIU *et al.* 1978) and inhibitor of photosynthetic electron transport (GOOD 1977). We have to keep in mind, however, that CCCP and its analogues act on mitochondria in the same way, i. e. either uncoupling or by inhibition of the respiratory chain (SLATER 1967, LÜTTGE *et al.* 1971, GOOD 1977). This fact is discussed below.

As in the case of CCCP it is difficult to determine the main inhibitory site of KCN. Besides inhibition of photosynthesis, a direct influence on mitochondria is very likely (GOOD & IZAWA 1973, CHEVALLIER *et al.* 1977). At the concentrations tested, cytochrome oxidase is probably strongly inhibited (SLATER 1967).

Antimycin causes elongation of mitochondria at concentrations which reduce O_2-uptake but not O_2-evolution (CHEVALLIER *et al.* 1977). Giant mitochondria after treatment with antimycin have also been observed in root tips of higher plants (WRISCHER & DEVIDE 1965) and in bleached *Euglena* cells (CALVAYRAC & BUTOW 1971), a further indication that photosynthesis is not affected.

NaN_3 inhibits the electron transport in mitochondria (LLOYD 1974).

Apart from DCMU and CCCP, which are assumed to act mainly on photosynthesis in this study, all substances that induced the elongation of mitochondria share one property: they inhibit either complex III (antimycin) or complex IV (CN, NaN_3) of the respiratory chain (see HANSON & DAY 1980). Thus it is concluded that formation of giant mitochondria in *Nitella flexilis* is directly or indirectly related to inactivation of these respiratory complexes. This conclusion is in accordance with findings of other authors (e. g. WRISCHER & DEVIDE 1965, CALVAYRAC *et al.* 1971, DAVISON *et al.* 1972, VARTAPETIAN *et al.* 1977). It is tempting to also attribute formation of giant mitochondria during reduced photosynthesis (treatment with DCMU, CCCP, low light intensity) to restricted cytochromal activity though entirely different processes could be decisive in this case. Assertions in this respect are extremely difficult to make in view of the complex interactions between chloroplasts and mitochondria (HEBER 1974, EVANS

& CARR 1979, GRAHAM & CHAPMAN 1979, GRAHAM 1980). Since DCMU was found to let dark respiration proceed in the light, an inhibitory effect on the cytochromes is very unlikely (JACKSON & VOLK 1970). The fact that normal and giant mitochondria of dim-light-grown algae stain with Janus Green B and subsequently bleach in anaerobic conditions indicates the activity of at least complex IV (LAZAROW & COOPERSTEIN 1953, FOISSNER 1981).

Because of this we have to look for metabolic processes which are influenced both by reduced photosynthesis and by inhibition of the cytochromes. The conclusion that enhanced glycolysis causes mitochondria to elongate would be consistent with those of other investigators (e. g. VARTAPETIAN *et al.* 1977). However, the question arises why neither rotenone nor DNP, both of which are assumed to increase the rate of glycolysis (MOHR & SCHOPFER 1978), have an effect on mitochondrial length. This could either contradict the above hypothesis or reflect the relative insensitivity of plant mitochondria to these substances (DUCET & LANCE 1978). The discrepancy cannot yet be solved the more so because the effect of CCCP could depend not only on uncoupling in chloroplasts but also on uncoupling in mitochondria, which would then favour the „glycolysis hypothesis". Heterotrophic metabolism with concomitant enhancement of glycolysis is unlikely to occur in characean cells (FORSBERG 1965). Furthermore, according to the results obtained, for instance by PELLEGRINI (1978), the change from photoautotrophic to heterotrophic metabolism is expressed by an increase in total mitochondrial volume per cell and not by elongation of single mitochondria (compare FOISSNER 1981).

Other metabolic processes that could be involved in the formation of giant mitochondria include alternate respiration (SOLOMOS 1977, BLEIN 1980) or restricted oxidation of exogenous NADH (PALMER 1976) or NADPH (HANSON & DAY 1980). Their possible role remains to be investigated (compare CALVAYRAC & BUTOW 1971).

A fact which must also be considered is that only a number of mitochondria (20% at the maximum) becomes elongated. This possibly reflects the existence of a heterogenous population of mitochondria (AVERS & KING 1960, FLETCHER 1972, and others). One population is assumed to be mainly concerned with anabolic functions (protein metabolism), in which photorespiration also takes a part (HALLDAL & HOLMEN 1979). Since photorespiration is inhibited at the same time as photosynthesis (HOCH *et al.* 1963), a relation to giant mitochondria formation is possible.

Elongation of mitochondria induced by DCMU is prevented in the presence of actidione or CAP. This indicates that protein synthesis in the cytoplasm and in the mitochondria is involved (KROON *et al*, 1972, CASKEY 1973). Protein synthesis in chloroplasts presumably plays a less decisive role in the present case. Protein synthesis could be necessary for the growth of the mitochondria and the simultaneous blockage of division (see LEFORT

1964) and/or for fusion of the mitochondria (see JAROSCH 1978). If the extreme length of the mitochondria is due to fission blockage, we have to assume an extremely rapid growth rate. Otherwise we would not have been able to observe mitochondria with a length of over 30 μm after only 2 days incubation with an appropriate inhibitor, since the doubling of mitochondrial substance normally requires 14 hours (KUROIWA 1968). Fusion, on the other hand, would better account for the existence of branched or ring-like mitochondria. The light microscopic observations suggest the involvement of both processes. Mitochondria between the chloroplasts generally cannot fuse because of their relatively fixed position. Elongation must thus occur by growth and blockage of fission. Since the space between the chloroplasts is limited, the mitochondria after having reached a certain length will incline back and fuse with themselves (ring-like forms). Fusion between different mitochondria is facilitated among mitochondria of the endoplasm which are in constant passive movement and hence easily come in contact with each other. This explains why branched organelles occur preferentially in the endoplasm.

Severe deleterious effects on cellular, especially mitochondrial structure were only observed after KCN-treatment. Such degenerative changes following the application of KCN have been reported e. g. from root tips of higher plants (WRISCHER & DEVIDE 1965). With all other substances the cytoplasm and the mitochondria are generally well preserved. Thus the giant mitochondria in *Nitella* should be regarded as adaptated organelles rather than pathological ones.

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6. References

AVERS C. J. & KING E. E. 1960. Histochemical evidence of intracellular enzymatic heterogeneity of plant mitochondria. — Amer. J. Bot. 47: 220—225.

BISHOP N. I. 1958. The influence of the herbicide, DCMU, on the oxygen-evolving system of photosynthesis. — Biochim. Biophys. Acta 27: 205—206.

BLEIN J. P. 1980. Cyanide stimulation of respiration of *Acer pseudoplatanus* cells in batch suspension culture and activation of the alternative pathway. — Plant Science Letters 19: 65—71.

CALVAYRAC R., BOMSEL J.-L. & LAVAL-MARTIN D. 1979. Analysis and characterization of 3-(3,4-dichlorophenyl)-1,1-dimethylurea (DCMU)-resistant *Euglena*. I. — Plant Physiol. 63: 857—865.

— & BUTOW R. A. 1971. Action de l'antimycine A sur la respiration et la structure des mitochondries d'*Euglena gracilis* Z. — Arch. Mikrobiol. 80: 62—69.

CALVAYRAC R., VAN LENTE F. & BUTOW R. A. 1971. *Euglena gracilis*: formation of giant mitochondria. — Science 173: 252—254.

CASKEY C. T. 1973. Inhibitors of protein synthesis. In: Metabolic Inhibitors, vol. IV (Eds. HOCHSTER R. M., KATES M. & QUASTEL J. H.): 131—177. — Academic Press, New York, London.

CHAPMAN E. A. & GRAHAM D. 1974. The effect of light on the tricarboxylic acid cycle in green leaves. I. — Plant Physiol. 53: 879—885.

CHEVALLIER D. & DOUCE R. 1976. Interactions between mitochondria and chloroplasts in cells. I. — Plant Physiol. 57: 400—402.

— — & NURIT F. 1977. Interactions entre mitochondries et chloroplastes dans la cellule. II. — Can. J. Bot. 55: 1650—1659.

DAVISON M. J., DOWNIE J. A. & GARLAND P. B. 1972. Giant mitochondria in iron- or copper-deficient *Candida utilis*. — Biochem. J. 129: 46P.

DAVIS B. & MERRETT M. J. 1975. The glycolate pathway and photosynthetic competence in *Euglena*. — Plant Physiol. 55: 30—34.

DOWNTON W. J. S. & TREGUNNA E. B. 1968. Photorespiration and glycolate metabolism: a re-examination and correlation of some previous studies. — Plant Physiol. 43: 923—929.

DUCET G. & LANCE C., (eds.) 1978. Plant Mitochondria. — Elsevier/North-Holland Biomedical Press, Amsterdam, New York, Oxford.

EVANS E. H. & CARR N. G. 1979. The interaction of respiration and photosynthesis in microalgae. In: Encyclopedia of Plant Physiology, New Series, vol. VI (Eds. GIBBS M. & LATZKO E.): 163—173. — Springer, Berlin, Heidelberg, New York.

FLETCHER J. S. 1972. Heterogeneous population of mitochondria in higher plant cells. — Nature 238: 466—467.

FOISSNER I. 1981. Licht- und elektronenmikroskopische Untersuchungen an Riesenmitochondrien von *Nitella flexilis*. — Phyton (Austria) 21: 39—51.

FORSBERG C. 1965. Nutritional studies of *Chara* in axenic cultures. — Physiol. Plant. 18: 275—290.

FUJII S., SHIMMEN T. & TAZAWA M. 1978. Light-induced changes in membrane potential in *Spirogyra*. — Plant & Cell Physiol. 19: 573—590.

GAUVRIT C. 1978. Effects of some substituted urea herbicides on oxidative phosphorylation in potato tuber mitochondria (*Solanum tuberosum* L.). In: Plant Mitochondria (Eds. DUCET G. & LANCE C.): 199—206. — Elsevier/North-Holland Biomedical Press, Amsterdam, New York, Oxford.

GOOD N. E. 1977. Uncoupling of electron transport from phosphorylation in chloroplasts. In: Encyclopedia of Plant Physiology, New Series, vol. V (Eds. TREBST A. & AVRON M.): 429—436. — Springer, Berlin, Heidelberg, New York.

— & IZAWA S. 1973. Inhibition of photosynthesis. In: Metabolic Inhibitors, vol. IV (Eds. HOCHSTER R. M., KATES M. & QUASTEL J. H.): 179—214. — Academic Press, New York, London.

GRAHAM D. 1980. Effects of light on "dark" respiration. In: The Biochemistry of Plants, vol. II (Ed. DAVIES D. D.): 525—579. — Academic Press, Inc., New York, London, Toronto, Sydney, San Francisco.

— & CHAPMAN E. A. 1979. Interactions between photosynthesis and

respiration in higher plants. In: Encyclopedia of Plant Physiology. New Series. vol. VI (Eds. GIBBS M. & LATZKO E.): 150—162. — Springer, Berlin, Heidelberg, New York.

HALLDAL P. & HOLMEN A. T. 1979. The effect of DCMU (3-(3,4-dichlorophenyl)-1,1-dimethylurea) on photosynthesis, glycolate excretion ("photorespiration") and amino acid metabolism in the blue-green alga *Anacystis*. — Physiol. Plant. 47: 195—199.

HANSON J. B. & DAY, D. A. 1980. Plant mitochondria. In: The Biochemistry of Plants, vol. I (Ed. TOLBERT N. E.): 315—358. — Academic Press, New York, London, Toronto, Sydney, San Francisco.

HEBER U. 1974. Metabolic exchange between chloroplasts and cytoplasm. — Ann. Rev. Plant Physiol. 25: 393—421.

HOCH G., OWENS O. v. H. & KOK B. 1963. Photosynthesis and respiration. — Arch. Biochem. Biophys. 101: 171—180.

IZAWA S. 1977. Inhibitors of electron transport. In: Encyclopedia of Plant Physiology, New Series, vol. V (Eds. TREBST A. & AVRON M.): 266—282. — Springer, Berlin, Heidelberg, New York.

JACKSON W. A. & VOLK R. J. 1970. Photorespiration. — Ann. Rev. Plant Physiol. 21: 385—432.

JAROSCH R. 1961. Das Characeen-Protoplasma und seine Inhaltskörper. — Protoplasma 53: 34—56.

— 1978. Mitochondrien-Dynamik auf Grund periodischer Auf- und Abwindung von Filamenten. — Phyton (Austria) 19: 123—136.

KELLERMANN G. M., BIGGS D. R. & LINNANE A. W. 1969. Biogenesis of mitochondria. XI. — J. Cell Biol. 42: 378—391.

KROON A. M., AGSTERIBBE E. & VRIES H. DE 1972. Protein synthesis in mitochondria and chloroplasts. In: The mechanism of protein synthesis and its regulation (Ed. BOSCH L.): 539—582. — Elsevier/North Holland, Amsterdam.

KUROIWA T., HIZUME M. & KAWANO S. 1978. Studies on mitochondrial structure and function in *Physarum polycephalum*. IV. — Cytologia 43: 119—136.

LAZAROW A. & COOPERSTEIN S. J. 1953. Studies on the mechanism on Janus-green B staining of mitochondria. — Exp. Cell Res. 5: 56—97.

LEFORT M. 1964. Modifications du chondriome dans les cellules etiolées de l'*Euglena gracilis* (KLEBS). — C. R. Acad. Sci. Paris 258: 4318—4321.

LLOYD D. 1974. The mitochondria of microorganisms. — Academic Press, London, New York, San Francisco.

LÜTTGE U., BALL E. & WILLERT K. v. 1971. Gas exchange and ATP-levels of higher plants as affected by FCCP and DCMU in *in vitro* experiments. — Z. Pflanzenphysiol. 65: 326—335.

MCMAHON D. 1975. Cycloheximide is not a specific inhibitor of protein synthesis in vivo. — Plant Physiol. 55: 815—821.

MOHR H. & SCHOPFER P. 1978. Lehrbuch der Pflanzenphysiologie. — Springer, Berlin, Heidelberg, New York.

MÜHLENBERG M. 1976. Freilandökologie. — UTB Quelle & Meyr, Heidelberg.

NEUMANN J. & JAGENDORF A. T. 1964. Dinitrophenol as an uncoupler of photosynthetic phosphorylation. — Biochem. Biophys. Res. Commun. 16: 562—567.

— & PARTHIER B. 1973. Effects of nalidixic acid, chloramphenicol, cycloheximide, and anisomycin on structure and development of plastids and mitochondria in greening *Euglena gracilis*. — Exp. Cell Res. 81: 255—268.

PALMER J. M. 1976. The organization and regulation of electron transport in plant mitochondria. — Ann. Rev. Plant Physiol. 27: 133—157.

PELLEGRINI M. 1978. The giant mitochondria of *Euglena gracilis* Z. In: Plant Mitochondria (Eds. DUCET G. & LANCE C.): 59—60. — Elsevier/North-Holland Biomedical Press, Amsterdam, New York, Oxford.

SLATER E. C. 1967. Application of inhibitors and uncouplers for a study of oxidative phosphorylation. — Methods Enzymol. 10: 48—57.

SOLOMOS T. 1977. Cyanide-resistant respiration in higher plants. — Ann. Rev. Plant Physiol. 28: 279—297.

THOMSON W. W., RAISON J. K. & LYONS J. M. 1972. The induction of energized configurational changes in plant mitochondria, *in vivo*. — Bioenergetics 3: 531—538.

VARTAPETIAN B. B., ANDREEVA I. N., KOZLOVA G. I. & AGAPOVA L. P. 1977. Mitochondrial ultrastructure in roots of mesophyte and hydrophyte at anoxia and after glucose feeding. — Protoplasma 91: 243—256.

WOOD R. D. & IMAHORI K. 1965. Monograph of the *Characeae*. — Cramer, Weinheim.

WRISCHER M. & DEVIDE Z. 1965. Mitochondrien-Veränderungen pflanzlicher Zellen bei Störung von Atmungsprozessen. — Z. Naturforsch. 20b: 260—263.