Forty years of plant mitochondriology

from A. L. Moore

IT has taken approximately 40 years since the first paper was published describing their oxidative capacities for a symposium to be held specifically devoted to plant mitochondria.*

Although the cytochromes of plant mitochondria are spectrally and functionally similar to those of animal mitochondria, there is still confusion about the role of the multiple *b* cytochromes in electron transport. B T. Storey (University of Pennsylvania, Philadelphia) suggested that cytochromes *b*-556 and *b*-560 are part of the respiratory chain whereas cytochrome *b*-565 only associated with the main chain in the energised state and in the presence of antimycin A. This suggestion was based upon the kinetic behaviour of *b*-565, in particular its complex oxidation/reduction kinetics during coupled substrate oxidation. However a few papers were presented which disagreed with this interpretation and it was suggested that a possible Q-cycle mechanism could explain the complicated nature of *b*-565 reduction under oxidative conditions.

P. R. Rich (University of Cambridge) gave a detailed analysis of the paramagnetic centres associated with higher plant and *poky N. crassa* mitochondria. Of particular importance was the finding that no EPR-detectable component was directly attributable to the alternative oxidase and furthermore it was suggested that there is no evidence for a direct involvement of iron sulphur centres or transition metals in a paramagnetic state in the terminal oxidation system. Although the HiPIP and ferredoxin-type centres are analogous to their mammalian counterparts, an EPR signal characteristic of a high-spin ferric iron with a *g* value at 4.3 was independently reported by Rich and M.-F. Henry (University of Amsterdam). Henry presented evidence in favour of this iron-containing protein's being involved in the cyanide insensitive respiratory pathway although the redox behaviour of this signal under conditions in which the alternative oxidase was operating was questioned. Obviously further work on the function of this component is required before its possible role in alternative oxidations can be better understood.

The topic which generated much interest and led to lively round-table discussions was the relationship between the malate oxidoreductases and NADH dehydrogenases. The process of malate oxidation is much more complex in plant than in animal mitochondria. Malate can be oxidised by two malate oxidoreductases and the NADH produced may be oxidised by at least four different NADH dehydrogenase systems. One of these dehydrogenases is located on the outer surface of the mitochondrial inner membrane.

Although there is general agreement that the oxidation of malate is mediated by an NAD^+-linked malic enzyme and malate dehydrogenase, the exact location of these enzymes is still controversial. J. M. Palmer (University of London) took the view that the oxidation of malate can provide NADH for oxidation by both the internal and external NADH dehydrogenases and that malate reduces external NAD^+ primarily by the activity of externally located malic enzyme and malate dehydrogenase. In contrast, however, J. T. Wiskich (University of Adelaide) supported the view that the external NADH dehydrogenase was not involved in the oxidation of malate in the absence of added NAD whereas in its presence a transmembrane transhydrogenase has been postulated to operate, which is considered to transfer reducing equivalents from internal NADH to external NAD thus connecting internal malate oxidation to the external dehydrogenase. D. A. Day (University of California) presented evidence on the malic enzyme distribution in potato mitochondria from which he concluded that malic enzyme is not present in the intermembrane space of these mitochondria and furthermore that all malate oxidation occurs in the matrix. Perhaps in this respect, the most interesting findings reported at this symposium were the detailed investigations of M. Neuburger (Université de Grenoble) on the transport of NAD^+ through the inner membrane of purified mitochondria. Convincing evidence was presented which indicated that highly purified intact plant mitochondria possess a NAD-translocator. Most workers readily agreed that the existence of such a carrier would explain many of the conflicting reports in the literature on malate oxidation and the further characterisation of this carrier is eagerly awaited.

The sessions on the molecular and cellular aspects of cyanide resistant respiration attracted considerable attention. The exact nature of the alternative oxidase has remained elusive for many years. However, as W. D. Bonner (University of Pennsylvania, Philadelphia) pointed out, a considerable amount of evidence has accumulated which allows a fuller understanding of its branchpoint from the main respiratory pathway and the nature of the products of the alternative oxidase with oxygen. Bonner suggested that a modified protonmotive ubiquinone cycle satisfies the kinetics of cytochrome interactions, and that the alternative pathway accepts electrons non-protonmotively from this cycle. This proposal suggests that a pool of ubiquinone in the succinic dehydrogenase region of the respiratory chain is performing as the branchpoint for the alternative pathway. Bonner concluded that the alternative oxidase could either be a species of ubiquinone or an EPR-silent metalloprotein that facilitates the reaction of the ubiquinone species with oxygen. In this regard a great advance in our knowledge of the nature of the alternative oxidase was obtained from the studies of Rich and S. Huq (Imperial College, London) who presented results on the isolation and partial purification of the alternative oxidase from *Arum maculatum*. Both workers used a hydroxamic acid-sensitive quinol oxidation as an assay for the oxidase. Remarkably, Rich found that in a deoxycholate solubilised preparation the quinol oxidase activity remained very stable, which seems to suggest that the lability of the oxidase in intact mitochondria may be due to the connection between the dehydrogenases and oxidase, and not the alternative oxidase itself. Both preparations contained minimal amounts of cytochromes aa_3, *c* and *b*. It was of particular interest to note that the overall product of oxygen reduction by the quinols was water.

A. M. Lambowitz (University of St. Louis) reviewed the current status of the biosynthesis of the alternative oxidase in *Neurospora* mitochondria. An important development in this area has been the isolation of antimycin-sensitive mutants with defects in the biosynthesis of the alternative oxidase. All cytochrome system activities seem unaffected in the mutants and the only apparent defect is in the induction of the alternative oxidase. Lambowitz suggested that the results indicate that there are at least two polypeptides unique to the alternative oxidation system. □

*The First International Symposium on Plant Mitochondria was held at the Luminy campus in Marseilles on 31 July–4 August.

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Erratum

In the article 'Halogenated hydrocarbon effects (*News & Views* **274**, 533; 1978), in the last paragraph, column 1, on page 534, the first line should read "Both the PCBs and the polybrominated biphenyls (PBBs) induce hepatic effects". In the second paragraph, column 2, line 4 ff. should read "Poland . . . believes this to be a receptor for TCDD and consequently for other inducers of aryl hydrocarbon hydroxylase . . .".

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