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Introduction

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This volume contains a series of papers that review recent developments in our understanding of the structure and function of plant mitochondria. Plant mitochondria are characterized by the possession of an externally located NADH dehydrogenase and an internal rotenone-resistant route through to oxygen and, in the majority of plants, a cyanide- and antimycin-resistant alternative oxidase. Other characteristic features of plant mitochondria include the capability to oxidize glycine, a key intermediate of the photorespiratory pathway, under illuminating conditions, suggesting that plant respiratory activity may contribute, under some conditions, to energy conservation in photosynthetic cells.

The first paper reviews current information on the properties of the alternative oxidase with particular emphasis on its structural features. Detailed analysis of published plant alternative oxidase sequences reveal the presence of several highly conserved regions and motifs, two of which are demonstrated to correspond to the motifs involved in ligating the binuclear iron cluster in methane monooxygenase. A structural model for the active site of the alternative oxidase is developed which suggests the presence of a hydroxo-bridged di-iron center in the active site and which opens up exciting possibilities for future structure–function studies. The nature of the alternative oxidase has been a question that many a laboratories have puzzled over for a considerable number of years, and it is a relief to know it is more than just a black box!

Regulation of the alternative oxidase is discussed in the next two papers of this series both in terms of activation and control by redox state. In the review by Day and Wiskich, the role of the redox state of the alternative oxidase in determining its activity in plant tissues is considered in detail particularly with respect to regulation by organic acid activators. Certain organic acids, most notably pyruvate and glyoxylate, do appear to activate the alternative oxidase in certain tissues which appears to account for the so-called substrate specificity of this enzyme. Activation by organic acids both on succinate dehydrogenase and the alternative oxidase is further considered in the article by Krab. This article emphasizes that in order to quantitatively understand how electron flux is partitioned between the cytochrome and alternative pathways it is necessary to have a knowledge not only of the kinetics of the quinol-oxidizing pathways but also of the quinone-reducing pathways, and it is the interplay between these different pathways that will define the extent to which the alternative oxidase is operative. Kinetic modelling of the plant respiratory network is discussed in terms of metabolic control analysis.

In the fourth article Soole and Menz discuss functional molecular aspects of the multiple NADH dehydrogenases of plant mitochondria. Although, in terms of subunit complexity, complex I of plant mitochondria is similar to the mammalian and fungal enzymes, there does appear to be a marked difference in the assembly of this complex since, in plants, two mitochondrial gene products (nad7 and nad9) are present in the peripheral arm. In beef heart and *Neurospora* both of these subunits are nuclear-encoded, implying that the assembly pathway for plant complex I differs from that of *Neurospora* or that assembly is independent of the origin of the polypeptide. The nature and role of the rotenone-insensitive matrix and cytosolic located NAD(P)H dehydrogenases are discussed in terms of their subunit composition and possible physiological role.

The articles by Oliver and Raman and Gardestrom and Lernmark discuss the structure, protein chemistry, and regulation of activity of glycine decarboxylase,

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the major protein in mitochondria isolated from leaf tissue. Oliver and Raman's article discuss the reaction mechanism, structure, and expression of the glycine decarboxylase complex. This abundant protein is nuclear-encoded by four single copy genes, the expression of which is under both temporal and spatial control. In contrast to animal tissues, where the complex has an essential function and mutants in this complex result in a disease called nonketotic hyperglycinemia, in plants it appears that the glycine decarboxylase complex has no essential nonphotorespiratory function. Importantly, the complex represents one of the very few developmental systems known in plant mitochondria and since the four different subunits that make up this complex readily dissociate and reassociate *in vitro*, it is an attractive model system to study subunit structure and protein:protein interactions. In the second of these two articles, Gardestrom and Lemmark discuss the regulation of glycine decarboxylase particularly with respect to the mechanism of re-oxidation of NADH, one of its major co-factors. Respiration in photosynthetic cells is discussed in terms of effects on gas exchange and focus on recent results indicating that mitochondrial ATP production and/or electron transport are crucial for optimal photosynthetic metabolism.

In the article by Braun and Schmitz, the bifunctional activity (electron transport and processing peptidase) of the plant cytochrome *c* reductase complex is reviewed in detail. It focuses on the structure, function, activity, and mitochondrial targeting of the 10 subunits of the complex. An interesting question concerning the physical relationship between the respiratory and proteolytic polypeptides is considered, with the conclusion that there is no significant physiological interrelation between both of these activities. The authors speculate that the occurrence of a bifunctional cytochrome *c* reductase/processing peptidase complex represents the original situation in eukaryotes and that the subunits of the peptidase became detached from the bc_1 complex in mammals and fungi after gene duplication has occurred.

In the last two articles of this series, the important role of plant mitochondria in cytoplasmic male sterility (CMS), a plant mitochondrial dysfunction, is reviewed. CMS is strongly correlated with rearrangements in the mitochondrial genome that create novel open reading frames. The article by Rhoads *et al.* focuses on the structure and function of URF13, a mitochondrially encoded gene product of 13 kDa found only in maize plants containing the Texas male-sterile cytoplasm thought to be responsible for both cytoplasmic male-sterility and susceptibility to fungal pathogens. URF13 acts as a receptor that specifically binds T-toxin to produce hydrophilic pores in the inner mitochondrial membrane. Topological studies reveal that URF13 contains three membrane-spanning α-helices, two of which are amphipathic and contribute to pore formation. Cross-linking experiments in conjunction with site-directed mutagenesis has established that the URF13 tetramer has a central core consisting of a four α-helical bundle which undergoes a conformational change following interaction with T-toxin. Such evidence indicates that URF13 functions as a ligand-gated, pore-forming T-toxin receptor in *cms-T* mitochondria and as such severely impairs the efficiency of mitochondrial oxidative phosphorylation.

In the article by Conley and Hanson, the question of how alterations in plant mitochondrial genomes disrupt pollen development is discussed. It focuses on Petunia as a system to study CMS and pollen development and concentrates on reproduction and mitochondrial function in this genus. In petunia mitochondria, CMS is associated with the presence of an aberrant *pcf* gene consisting of sequences from the first half of ATP synthase subunit 9, portions of both exons of the cytochrome oxidase subunit 2 (*cox II*), and an unidentified open reading frame (*urfS*), and the genes for NADH dehydrogenase subunit 3 (*nad3*) and ribosomal protein S12 (*rps12*). Although the molecular mechanism by which CMS might be caused has not been conclusively demonstrated for any system, several hypotheses by which mitochondrial dysfunction might disrupt pollen development are discussed. These include engagement of the alternative oxidase and impaired functioning of the ATP synthase, both of which affect ATP production. Whether altered efficiency in mitochondrial energy transduction is at the root of CMS in all species, or whether defects in numerous mitochondrial activities can produce sterility, will only be revealed by further analysis of the respiratory and phosphorylative properties of mitochondria from CMS lines of other species.