Disruption of the CYTOCHROME C OXIDASE DEFICIENT1 Gene Leads to Cytochrome c Oxidase Depletion and Reorchestrated Respiratory Metabolism in Arabidopsis

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Résumé en anglais	Cytochrome c oxidase is the last respiratory complex of the electron transfer chain in mitochondria and is responsible for transferring electrons to oxygen, the final acceptor, in the classical respiratory pathway. The essentiality of this step makes it that depletion in complex IV leads to lethality, thereby impeding studies on complex IV assembly and respiration plasticity in plants. Here, we characterized Arabidopsis (Arabidopsis thaliana) embryo-lethal mutant lines impaired in the expression of the CYTOCHROME C OXIDASE DEFICIENT1 (COD1) gene, which encodes a mitochondria-localized PentatricoPeptide Repeat protein. Although unable to germinate under usual conditions, cod1 homozygous embryos could be rescued from immature seeds and developed in vitro into slow-growing bush-like plantlets devoid of a root system. cod1 mutants were defective in C-to-U editing events in cytochrome oxidase subunit2 and NADH dehydrogenase subunit4 transcripts, encoding subunits of respiratory complex IV and I, respectively, and consequently lacked cytochrome c oxidase activity. We further show that respiratory oxygen consumption by cod1 plantlets is exclusively associated with alternative oxidase activity and that alternative NADH dehydrogenases are also up-regulated in these plants. The metabolomics pattern of cod1 mutants was also deeply altered, suggesting that alternative metabolic pathways compensated for the probable resulting restriction in NADH oxidation. Being the first complex IV-deficient mutants described in higher plants, cod1 lines should be instrumental to future studies on respiration homeostasis.

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