Nitrite–nitric oxide control of mitochondrial respiration at the frontier of anoxia

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Résumé en anglais	Actively respiring animal and plant tissues experience hypoxia because of mitochondrial O2 consumption. Controlling oxygen balance is a critical issue that involves in mammals hypoxia-inducible factor (HIF) mediated transcriptional regulation, cytochrome oxidase (COX) subunit adjustment and nitric oxide (NO) as a mediator in vasodilatation and oxygen homeostasis. In plants, NO, mainly derived from nitrite, is also an important signalling molecule. We describe here a mechanism by which mitochondrial respiration is adjusted to prevent a tissue to reach anoxia. During pea seed germination, the internal atmosphere was strongly hypoxic due to very active mitochondrial respiration. There was no sign of fermentation, suggesting a down-regulation of O2 consumption near anoxia. Mitochondria were found to finely regulate their surrounding O2 level through a nitrite-dependent NO production, which was ascertained using electron paramagnetic resonance (EPR) spin trapping of NO within membranes. At low O2, nitrite is reduced into NO, likely at complex III, and in turn reversibly inhibits COX, provoking a rise to a higher steady state level of oxygen. Since NO can be re-oxidized into nitrite chemically or by COX, a nitrite–NO pool is maintained, preventing mitochondrial anoxia. Such an evolutionarily conserved mechanism should have an important role for oxygen homeostasis in tissues undergoing hypoxia.
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