NAD(P)H- AND SUPEROXIDE-DEPENDENT NITRIC OXIDE DEGRADATION BY POTATO TUBER MITOCHONDRIA

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Nitric oxide (NO) has emerged as a ubiquitous signaling molecule in biological systems. Besides its synthesis, NO degradation is important to control steady-state levels of this radical in the cells. Some mechanisms for NO consumption have been addressed in mammalian mitochondria, such as the non-enzymatic reaction of NO with superoxide to form peroxynitrite. In the present work, we analyzed the NO degradation activity of mitochondria isolated from potato tubers. NO and O_2 concentrations in the reaction medium were followed using electrochemical sensors connected to a free radical analyzer. NO degradation was faster in mitochondria energized with NAD(P)H or malate than those with succinate. Therefore, oxygen consumption was transiently or persistently inhibited in NAD(P)H- and succinate-energized mitochondria, respectively. NO degradation, observed even at very low concentrations of NAD(P)H, was abolished under anoxia and in the presence of superoxide dismutase, indicating that NO was consumed by its reaction with superoxide. NO consumption was stimulated by antimycin-A, which favors electron leakage from complex III. NO degradation was partially prevented by EGTA and greatly enhanced when mitochondria were ruptured. These results suggest that complex I and Ca^{2+}-dependent external NAD(P)H dehydrogenases contribute to this aerobic NO degradation. Concluding, NAD(P)H leads to an increased superoxide generation by respiratory chain, stimulating NO consumption in potato tuber mitochondria.
Supported by Fapesp.

Nitric oxide – Plant mitochondria – Superoxide