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Nitric oxide scavenging modulates mitochondrial dysfunction induced by hypoxia/reoxygenation

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Abstract:
The objective of the present study was to delineate the role of excessive accumulation of mitochondrial nitrogen species contributing to oxidative stress induced by hypoxia/reoxygenation in isolated mitochondria. The present study shows that incubation of isolated rat heart mitochondria under hypoxic, but not anoxic conditions, followed by reoxygenation decreases the rate of mitochondrial oxygen consumption, mitochondrial membrane potential, and calcium retention capacity. These alterations were prevented, at least in part, by 2-(4-carboxyphenyl)-4,4,5,5-tetramethylimidazoline-1-oxyl-3-oxide (carboxy-PTIO), a nitric oxide (NO) scavenger, N^G-nitro-L-arginine-methyl ester (L-NAME), a broad-spectrum NO synthase inhibitor, or tempol, a superoxide dismutase mimetic and catalytic scavenger of peroxynitrite-derived radicals. In conclusion, these findings suggest a crucial role for nitric oxide pathways in cardiac oxidative stress induced by hypoxia/reoxygenation.

Key words:
mitochondria, respiration, membrane potential, nitric oxide, peroxynitrite

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