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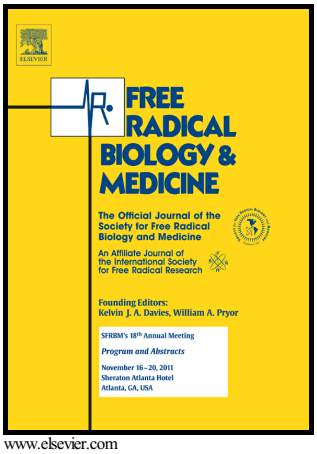

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Attenuation of oxidative damage by targeting mitochondrial complex I in neonatal hypoxic-ischemic brain injury.

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Abstract

Background

Establishing sustained reoxygenation/reperfusion ensures not only the recovery, but may initiate a reperfusion injury in which oxidative stress plays a major role. This study offers the mechanism and this mechanism-specific therapeutic strategy against excessive release of reactive oxygen species (ROS) associated with reperfusion-driven recovery of mitochondrial metabolism.

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