Pathophysiology of Ischemic Stroke: Role of Oxidative Stress

Por: Orellana-Urzua, S (Orellana-Urzua, Sofia)[1]; Rojas, I (Rojas, Ignacio)[1]; Libano, L (Libano, Lucas)[1]; Rodrigo, R (Rodrigo, Ramon)[2]

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Abstract

Stroke is the second leading cause of mortality and the major cause of adult physical disability worldwide. The currently available treatment to recanalize the blood flow in acute ischemic stroke is intravenous administration of tissue plasminogen activator (t-PA) and endovascular treatment. Nevertheless, those treatments have the disadvantage that reperfusion leads to a highly harmful reactive oxygen species (ROS) production, generating oxidative stress (OS), which is responsible for most of the ischemia-reperfusion injury and thus causing brain tissue damage. In addition, OS can lead brain cells to apoptosis, autophagy and necrosis. The aims of this review are to provide an updated overview of the role of OS in brain IRI, providing some bases for therapeutic interventions based on counteracting the OS-related mechanism of injury and thus suggesting novel possible strategies in the prevention of IRI after stroke.

Palabras clave

Palabras clave de autor: Ischemia-reperfusion; oxidative stress; reactive oxygen species; ischemic stroke; haemorrhagic stroke; antioxidant

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Información del autor

Dirección para petición de copias:
Universidad de Chile Univ Chile, Mol & Clin Pharmacol Program, Inst Biomed Sci, Fac Med, Independencia 1027, Santiago 8380453, Region Metropol, Chile.

Dirección correspondiente: Rodrigo, R (autor correspondiente)

+ Univ Chile, Mol & Clin Pharmacol Program, Inst Biomed Sci, Fac Med, Independencia 1027, Santiago 8380453, Region Metropol, Chile.

Direcciones:

+ [1] Univ Chile, Fac Med, Santiago, Chile

+ [2] Univ Chile, Mol & Clin Pharmacol Program, Inst Biomed Sci, Fac Med, Independencia 1027, Santiago 8380453, Region Metropol, Chile

Direcciones de correo electrónico:rrodrigo@med.uchile.cl

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