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NADPH oxidase inhibition as a neuroprotective measure against carbon monoxide toxicity

Carbon monoxide (CO) neurotoxicity is known to cause severe and detrimental damage to the brain through hypoxia, ischaemia, and neural cell degeneration and necrosis. Exposure to CO has been observed to generate an overproduction of reactive oxygen species (ROS) and induce oxidative stress leading to compromised and damaged neural systems. The current treatment recommended for CO poisoning is oxygen therapy by using hyperbaric oxygen supplementation (aside from the UK) yet neuroprotection strategies are a definitive step forward in both the treatment and understanding of CO-induced oxidative stress. By using live cell imaging of cultures of neurons and astrocytes, neuroprotection strategies can be investigated and applied for high risk groups in society. Notably, inhibition of NADPH oxidase has been shown to protect neurons and astrocytes against oxidative stress by drastically reducing cell death. NADPH oxidase inhibition is effective after CO-exposure and therefore investigation into the timings and procedures for NADPH oxidase inhibition is critical for generating effective treatment and protection. The fundamental understanding and improvement of a prevention strategy for CO poisoning and exposure is a progressive step forward leading from the findings of NADPH oxidase inhibition as well as the effects of mitochondrial antioxidants. Involvement of a combination of inhibitors can also be explored with mitochondrial antioxidants being a preventative strategy and NADPH oxidase inhibition being a treatment strategy for CO-induced oxidative stress.

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