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Reperfusion Injury: Mechanism and Therapeutic Strategies for Future Directions

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Commentary

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ABOUT THE STUDY

Reperfusion injury, sometimes called Ischemia-Reperfusion Injury (IRI) or reoxygenation injury, is the tissue damage caused when blood supply returns to tissue after a period of ischemia or lack of oxygen.

Reperfusion of ischemic tissues frequently results in microvascular damage, particularly as a result of enhanced capillary and arteriole permeability that boosts fluid diffusion and filtration through the tissues. Following reperfusion, activated endothelial cells create an imbalanced amount of reactive oxygen species and nitric oxide that leads to an inflammatory reaction. Damage from reperfusion injury is partially caused by the inflammatory response. In response to tissue damage, white blood cells, which are brought to the location by the freshly returned blood, release a variety of inflammatory substances such as interleukins and free radicals.

The reestablished blood flow causes damage to cellular proteins, DNA, and the plasma membrane by reintroducing oxygen within the cells. More free radicals may be released as a result of damage to the cell membrane. Such reactive radicals may also indirectly activate apoptosis through redox signaling. Additional ischemia may result from white blood cells adhering to the endothelium of tiny capillaries and blocking them.

The biochemistry of hypoxic brain injury following stroke involves reperfusion injury significantly. Similar failure mechanisms are engaged in brain failure after cardiac arrest recovery; research into the regulation of these mechanisms is continuing. Pressure sores and diabetic foot ulcers are examples of chronic wounds that develop and fail to heal as a result of repeated episodes of ischemia and reperfusion damage. Continuous pressure restricts blood flow, causing ischemia, and reperfusion causes the inflammation. Repeating this process eventually causes enough tissue damage to result in a wound.

Oxygen deprivation and the subsequent stoppage of oxidative phosphorylation in the mitochondria are the main causes of the acute phase of ischemia-reperfusion damage. Reperfusion (increase in oxygen level) occurs after tissue damage caused by the overall energy deficit during ischemia when the injury is intensified. Thought to be the most susceptible enzyme to tissue ischemia/reperfusion is mitochondrial complex I, but the mechanism of harm varies depending on the tissue. For instance, complex I redox-dependent inactivation mediates brain ischemia/reperfusion damage.

It was discovered that low oxygen levels cause the mitochondrial complex I to become inactive and lose its natural cofactor, flavin mononucleotide. The enzyme catalysis the physiological process of NADH oxidation by ubiquinone when oxygen is present, delivering electrons to the respiratory chain in the process. Succinate levels dramatically rise as a result of ischemia. When succinate is present, mitochondria catalyze reverse electron transfer, directing a portion of the succinate's electrons upstream to the FMN of complex I. Reverse electron transfer causes complex I FMN to decrease, ROS production to increase, the decreased cofactor to disappear, and mitochondrial energy production to be impaired. The use of FMN precursor riboflavin can reduce the loss of FMN caused by complicated I and I/R damage.

However, metabolism and membrane stability are only two aspects of hypothermia's therapeutic action. Another school of thinking focuses on the ability of hypothermia to stop reperfusion injuries, which are those that happen when blood flow returns to the brain. In actuality, even after circulation is restored, an individual who has experienced an ischemia insult continues to have damage. Many now believe that the reason hypothermia improves patient outcomes after a restriction of blood flow to the brain is because it lowers intracranial pressure and free radical generation.