The Role of Oxidative Stress in Traumatic Brain Injury Recovery

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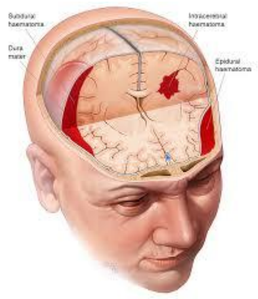
INTRODUCTION

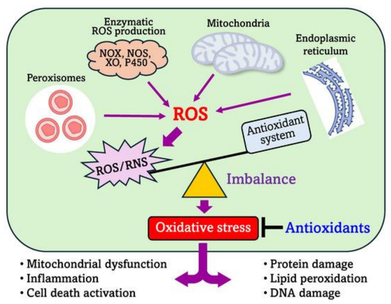
Traumatic brain injury (TBI) is a major cause of long-term neurological impairment. Common causes of TBI include falls, accidents and other injuries. TBI outcomes are mediated primarily through physical injury to brain tissue, and secondarily through oxidative stress.

Figure 1 – TBI



Oxidative stress happens when reactive oxygen/nitrogen species (ROS/RNS) are produced. This review critically examines the oxidative stress mechanisms in TBI and evaluates the current evidence of safety and efficacy of antioxidant treatment for TBI.

Figure 2 – Oxidative stress in TBI



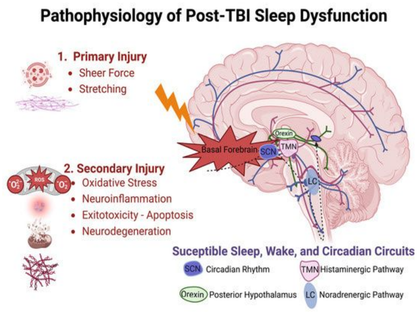
This figure shows that cellular health depends on maintaining a balance between the generation of ROS and antioxidant defense systems.

METHODS

A literature review of both clinical and animal studies on oxidative stress in TBI. The literature reviewed included:

1. Literature on the mechanism of oxidative stress and its effect on TBI prognosis and progression.
2. Literature on the efficacy of anti-oxidant therapies on TBI prognosis and progression

Figure 3 Pathophysiology of TBI Associated Sleep Dysfunction



This figure is an example of a primary brain injury that involves stretching, pressure, and tearing, followed by a secondary brain injury that causes more oxidative stress and neuroinflammation

Focused on 3 mechanisms:

- ROS overproduction + mitochondrial damage
- Brain swelling + neuronal injury
- Reduced tissue repair

Goal: To fully understand how antioxidants help prevent oxidative stress, protect neurons, lower brain damage, and possibly improve TBI patients' chances of recovery.

RESULTS

A. Mechanism of oxidative stress and effect on TBI progression

Mitochondrial damage → excess ROS production

ROS causes:

* Blood–brain barrier breakdown
- Neuron death
- Stroke-like injury
- Oxidative stress limits tissue **repair and neurogenesis**

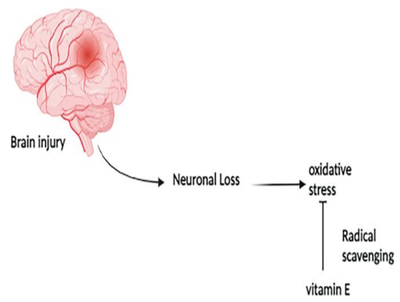
B. Efficacy of antioxidant treatments for TBI

Evaluated antioxidant treatments:

- quercetin
- vitamin E
-curcumin
- DHA (Docosahexaenoic Acid) amino acid
  helps brain repair
- NAC (N-acetylcysteine)

C. Antioxidants (NAC, DHA, quercetin) **reduce damage** in experimental studies
- Clinical outcomes remain inconsistent

Figure 4 - Antioxidants and Traumatic Brain Injury



Overall, oxidative stress is a significant cause of secondary damage in traumatic brain injury (TBI), and antioxidants appear to be beneficial, yet further study is needed.

CONCLUSION

In TBI, oxidative stress is a major cause of further injury. Some antioxidants to be appear beneficial in i*n-vivo* and *in -vitro* studies, but clinical outcomes remain inconsistent.

Future directions:

- More clinical studies are needed
- Better dosing + timing
- Multi-target therapy
- Improved brain delivery
- Potential to improve recovery + quality of life

Managing dose, using different routes, along with improving movement across the barrier between blood and brain, should be the main goals of future research. For TBI patients, effective antioxidant therapy may significantly improve their quality of life and recover.

Recourses :

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