A mechanistic link between oxidative stress and neuronal death in Alzheimer's Disease: Quest for a cure

Accumulation of free radicals mediated damage is considered an important factor in aging and neurodegenerative diseases. This is evidenced by decreased expression of cellular antioxidants and increased oxidative damage to proteins. While in normal conditions, these damaged proteins are recycled through autophagy, this process fails in the brain of people with Alzheimer's Disease (AD). Thioredoxin-1 (Trx1) is a small antioxidant protein responsible for reducing the oxidized proteins. Reports of Trx1 depletion in aging and in some neurodegenerative diseases suggest a critical role for this protein. However, many aspects of Trx1 functions remain unknown.

Using cellular and animal models to study the biology and novel therapeutic interventions for neurodegenerative diseases, we have shown that depletion of Trx1 is associated with structural and functions changes in lysosomes, nucleus and cellular cytoskeleton. These changes are also reported in AD patients. We are currently examining whether pharmacological replacement of Trx1 may reverse some of these changes. This research is partially supported by Alzheimer's Society of Canada.

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Register on Zoom: https://tinyurl.com/28pp3f8u

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