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Reducing Oxidative Stress with Multifunctional Radical Quenchers

Mitochondria are intracellular organelles responsible for a number of metabolic transformations and regulatory functions. They produce much of the ATP employed by eukaryotic cells. They are also the major source of free radicals and reactive oxygen species that cause oxidative stress.

Mitochondrial defects are damaging, particularly to neural and muscle tissues which have high energy demands. Energetic defects have been implicated in forms of movement disorders, cardiomyopathy, myopathy, blindness, and deafness. Currently, there are no effective treatments for mitochondrial diseases.

Researchers at the Biodesign Institute of Arizona State University have developed compounds to treat diseases associated with decreased mitochondrial function resulting in diminished ATP production and/or oxidative stress and/or lipid peroxidation. The compounds discovered are multifunctional radical quenchers, which prevent ROS-dependent cell death scavenging oxygen free radicals and lipid peroxidation by quenching C-centered lipid radicals.

These compounds represent a novel class of potential therapeutics for a variety of diseases associated with decreased mitochondrial function, including Friedreich's ataxia, Leber's Hereditary Optic Neuropathy, Kearns-Sayre Syndrome, Mitochondrial Encephalomyopathy with Lactic Acidosis and Stroke-like Episodes.

Potential Applications

- Mitochondrial diseases:
  - Friedreich's ataxia, Leber's Hereditary Optic Neuropathy, Kearns-Sayre Syndrome, Mitochondrial Encephalomyopathy with Lactic Acidosis and Stroke-Like Episodes (Leigh syndrome)

Benefits and Advantages

- More effective than idebenone or a-tocopherol (antioxidants)

For more information about the inventor(s) and their research, please see Dr. Hecht's departmental webpage