RAPID RESEARCH

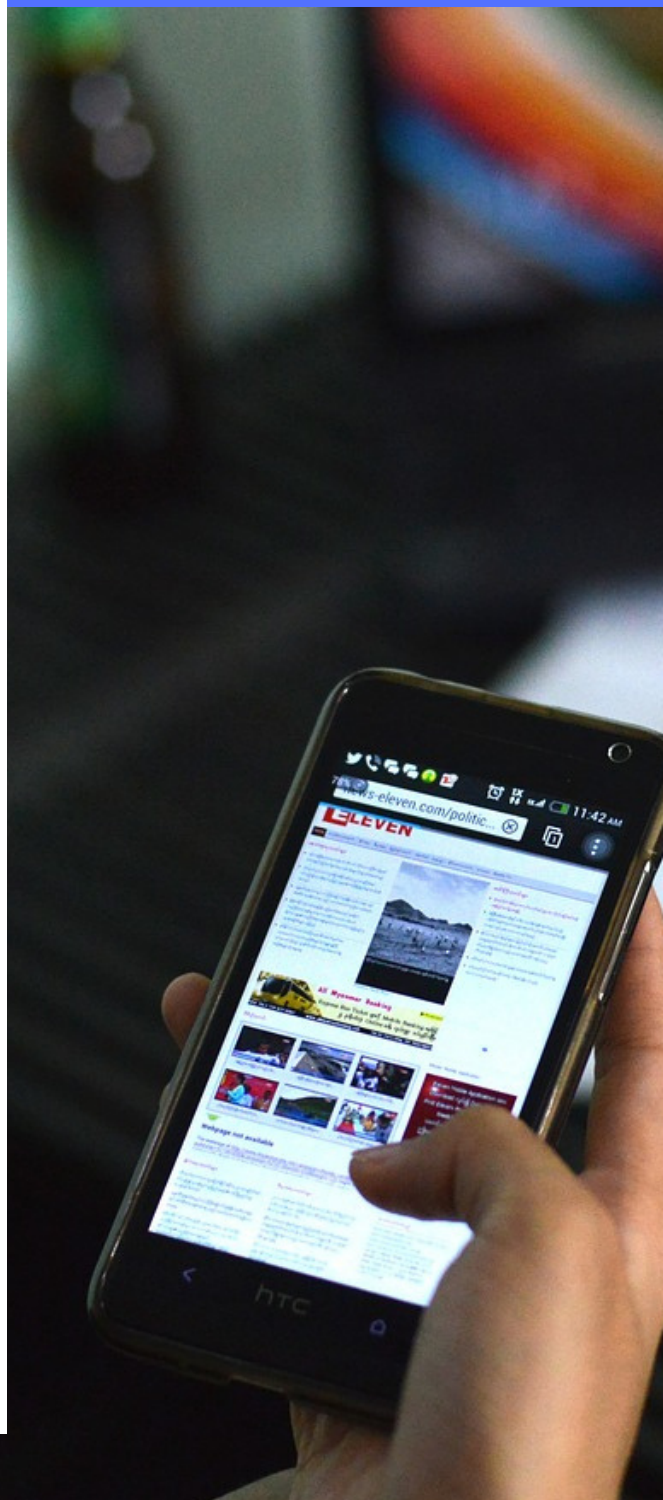
@physicaltherapyresearch

May 2022

Inside This Week:

Tendinopathy Intrinsic Factors

- Mitochondrial Transplant to Help Tendinopathy
- Mitochondrial Dysfunction & Treatments in Tendinopathy
- Oxidative Stress in Tendinopathy

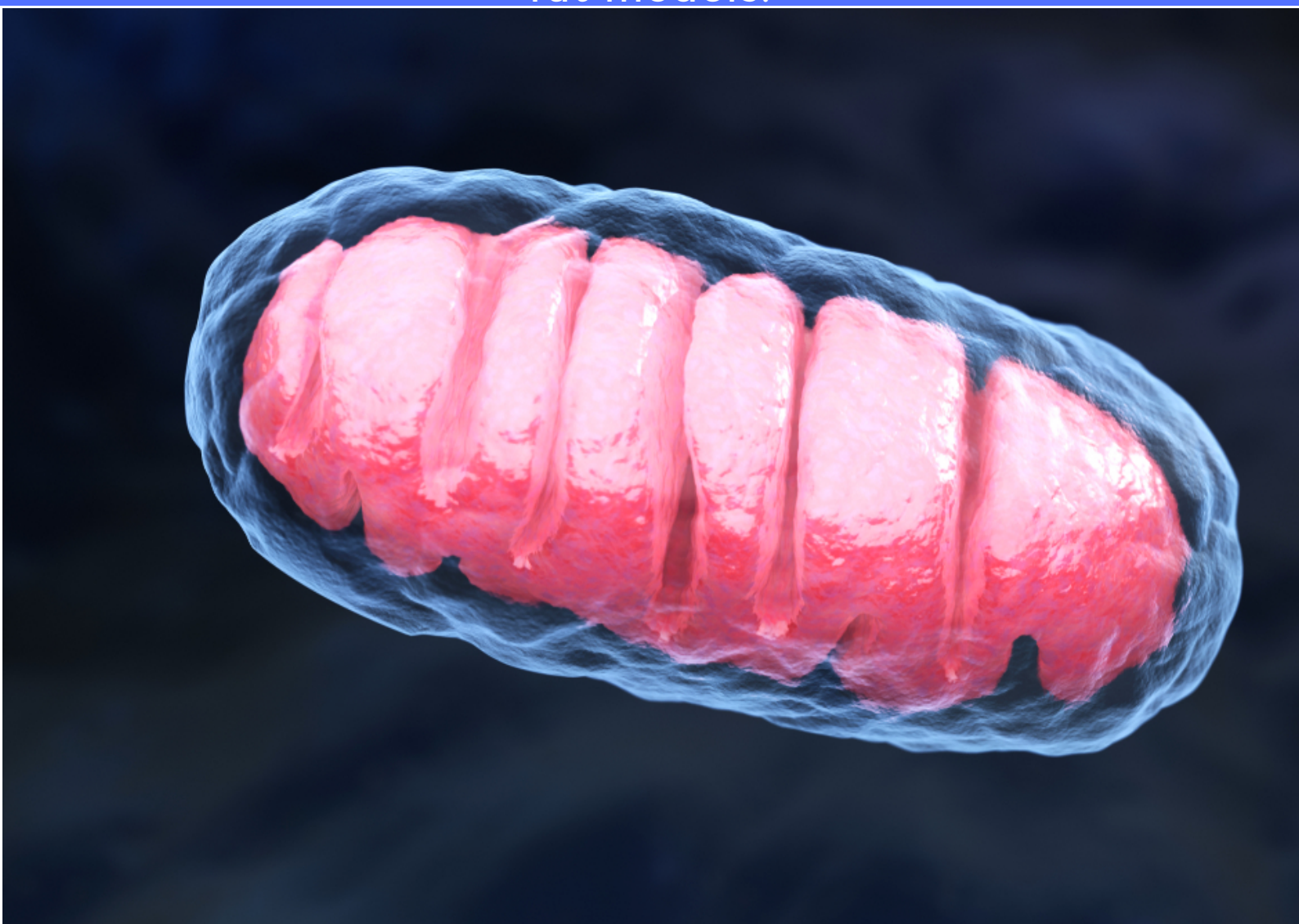


MAY 2022

MITOCHONDRIAL TRANSPLANT TO HELP TENDINOPATHY

Click for Full Text (Lee et al. 2021)

This research assessed whether mitochondrial transplantation could induce anti-inflammatory activity and modulate the metabolic state of a tendinopathy in rat models.



KEY FINDINGS

Tenocytes were treated with TNF-α for 24 h to establish an in vitro tendinopathy model.

Mitochondria were transferred into tenocytes by centrifugation and into tendon cells for the in vivo models.

In Vitro:

- Levels of Tenomodulin and Collagen I in damaged tenocytes were restored
- The dysregulation of oxidative stress and mitochondrial membrane potential was attenuated.
- Activated mitochondrial fission markers were down regulated.
- Apoptosis signaling pathway proteins were restored to the pre-damage levels.

In Vivo:

- Tendon thickness increased significantly at 1 and 2 weeks.
- Swelling subsided at 1 and 2 weeks after local injection
- Inflammatory and fission marker levels reduced significantly.
- Collagen production was restored.

MAIN TAKEAWAYS

This study provides the first evidence for the therapeutic effects of mitochondrial transfer in tendinopathy.

Exogenous mitochondria were successfully delivered into damaged tenocytes (in vitro) and tendons (in vivo).

They modulated inflammation and apoptosis and recovered the collagen component of the extracellular matrix.

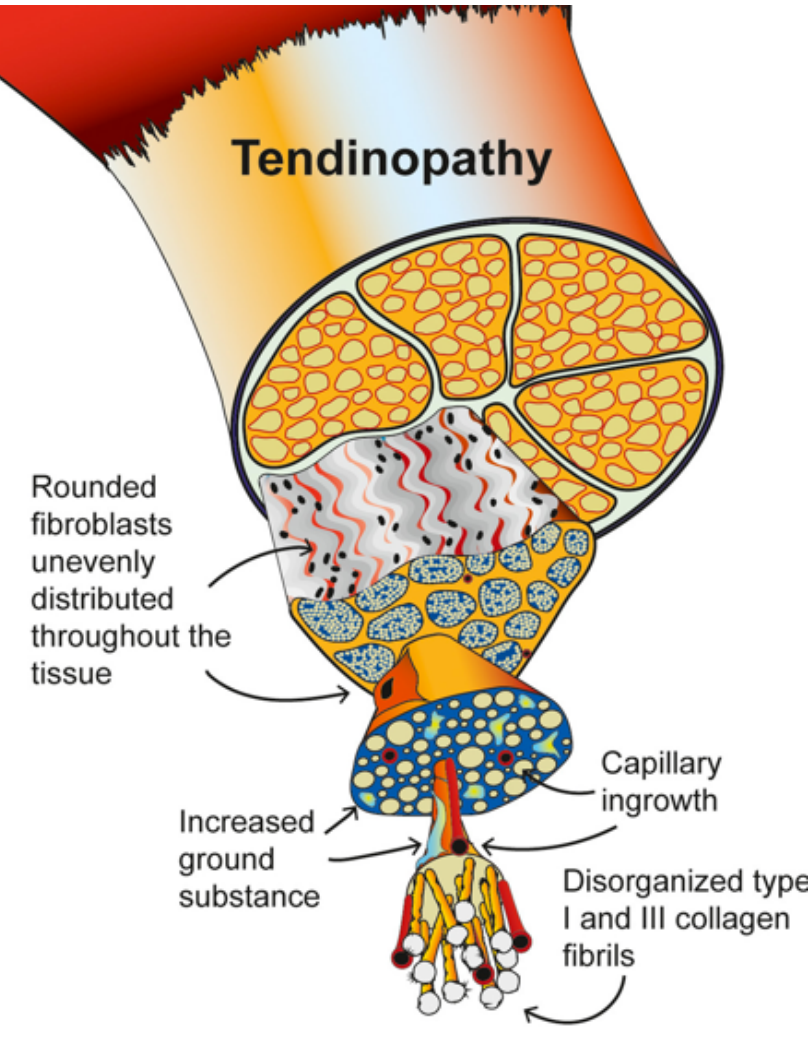
Exogenous mitochondrial transfer may become a novel approach for the treatment of tendinopathy.

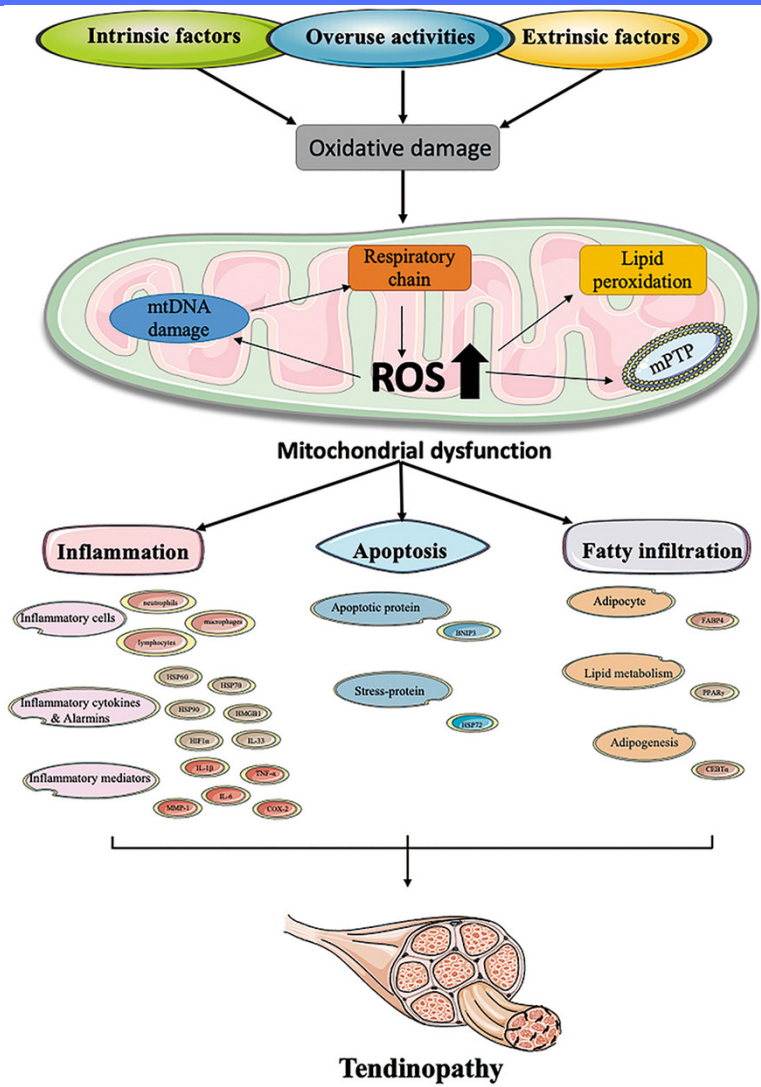
MITOCHONDRIAL DYSFUNCTION & TREATMENTS IN TENDINOPATHY

MAY 2022

Click for Full Text (Zhang et al. 2021)

This review summarizes the potential mechanism of mitochondrial dysfunction in tendinopathy and potential therapeutic benefits of mitochondrial protectants in the treatment of tendinopathy.





KEY FINDINGS

Potential Mechanisms:

- Inflammation
- Apoptosis & Cell Death
- Fatty Infiltration

Mitochondrial Protectants as Treatment

- **Szeto-Schiller peptide-31 (SS-31)**
  - Improves mitochondrial function.
  - Potentially promotes ATP synthesis and oxidative phosphorylation, reducing the levels of Reactive Oxygen Species (ROS), thus preventing apoptosis and inflammation.
- **Nicotinamide mononucleotide (NMN)**
  - Protects mitochondria via NAPDH synthesis
- **Cyclosporin A (CsA),**
  - Inhbits PTP complex; prevents cell death from oxidative stress.

MAIN TAKEAWAYS

Mitochondrial dysfunction may be associated with the development of tendinopathy.

Damaged tendon's poor intrinsic regenerative potential, make it difficult for tendons to heal once injured.

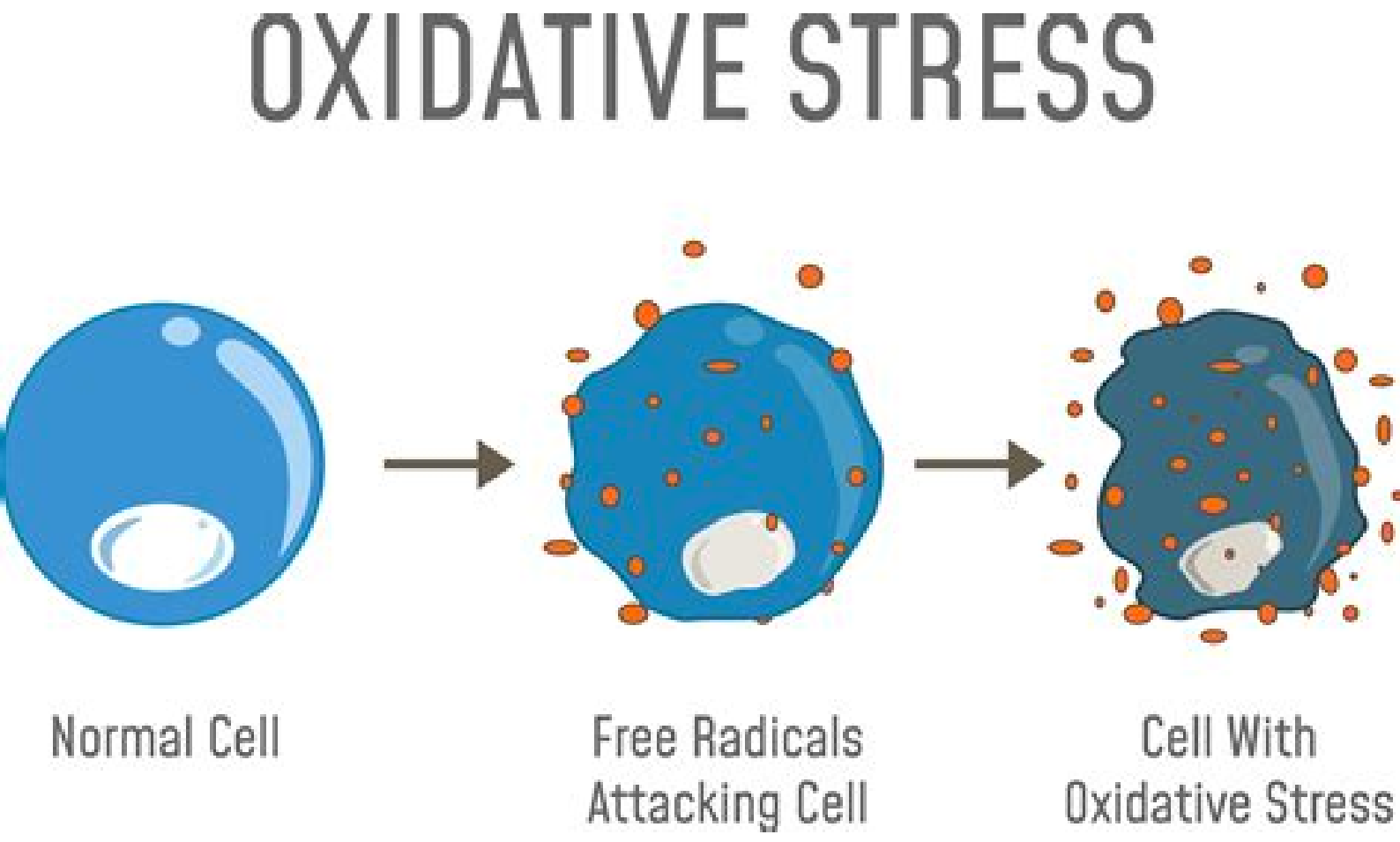
Several mitochondria-targeted drugs are being used in clinical trials and may be effective to treat and delay the development of tendinopathy through protection of mitochondria.

MAY 2022

OXIDATIVE STRESS IN TENDINOPATHY

Click for Full Text (Lui et al. 2022)

This Systematic Review aimed to summarize the clinical and pre-clinical evidence about the potential relationship of oxidative stress and tendon disorders.



KEY FINDINGS

81 articles included.

Oxidative Stress in Degenerative Tendinopathy

- Reactive oxygen species (ROS) are continually generated during normal cell metabolism, environmental and physiological stimulus.
- ROS can damage lipid, proteins and DNA in cells and tissues.
- Sustained oxidative stress is a major contributing factor to tendon fibrosis, adhesion, and pathological changes in tendinopathy

Potential Sources and Mechanisms of Oxidative Stress in Tendon:

- The tendon constantly generates ROS after exercise and traumatic injury
- Both acute and chronic tendon injuries can induce oxidative stress and inflammation in tendon.
- Overuse and Overloading up-regulates genes related to ROS production.
- Deficiency of mTOR pathway
- Metabolic risk factors (obesity, diabetes, hypercholesterolemia)
- Use of fluoroquinolone antibiotics

MAIN TAKEAWAYS

Oxidative stress due to excessive ROS production might contribute to the pathogenesis of acute tendon injury and degenerative tendinopathy.

Tendon injury, overuse, metabolic risk factors and drug exposure are potential sources of ROS in tendon disorders.

Further research to understand the molecular mechanisms of oxidative stress-induced tendon damage and develop novel anti-oxidative therapies for the promotion of tendon healing is required.

GIVE US YOUR FEEDBACK!

MEMBERS

We are on a mission to make research more accessible, easier to interpret, and quicker to implement.

Help us by giving 1 minute of your time to leave feedback for us.

We would greatly appreciate any feedback you have, as it helps us continually improve!

Leave Review