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NZ study links mitochondrial targeted antioxidant with protecting genomic DNA

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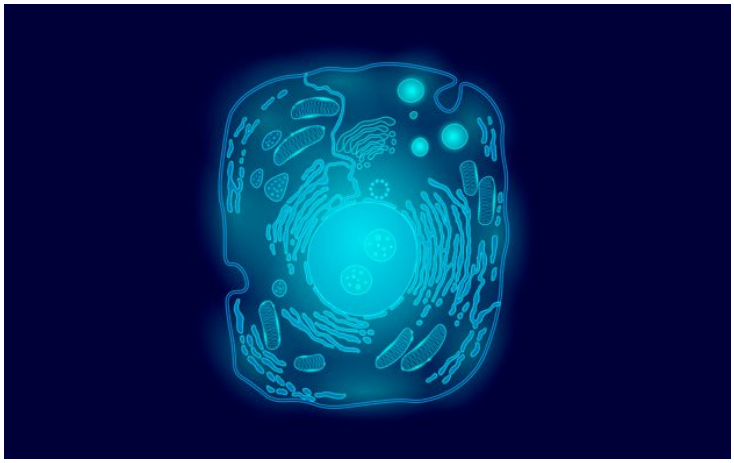
Novel antioxidant that specifically targets mitochondria, sees attenuation in exercise induced mitochondria DNA (mtDNA) damage in lymphocytes and muscle in exercising humans



New Zealand's researchers discovered that healthy male participants who take a novel antioxidant that specifically targets the powerhouse of the cells, mitochondria, sees attenuation in exercise induced mitochondria DNA (mtDNA) damage in lymphocytes and muscle.

Mitochondria has been shown to have numerous roles in a cell from contributing cellular networks for biosynthetic pathways, to stem cell function, mitophagy, proteolysis and apoptotic cell death. Mitochondrial dysfunction and disease, while complex is linked to each other. Production of mitochondrial reactive species occurs during and after exercise in humans. Studies have shown that this can increase mtDNA damage. Understanding this form of mtDNA damage can be important in preserving the integrity of the mitochondrial genome.

The study published on August 6th, 2020 in Redox Biology investigated whether a bout of high-intensity intermittent exercise (HIIE) damaged mtDNA and whether MitoQ, a commercially available supplement could prevent this damage.

"The majority (if not all) of exercise studies have used pleiotropic, non-selective antioxidants with unknown tissue distribution in an attempt to infer mechanistic conclusions relating to redox signalling from oxidative stress biomarkers.", said lead author Dr Josh Williamson from Sport and Exercise Research Institute at Ulster University.

"Mitochondrial targeted antioxidants (in this instance MitoQ) offer exciting new opportunities for research which may have important implications for physiological and pathological outcomes. As a result, the aim of this study was to determine whether high intensity intermittent exercises damage mitochondrial DNA, and more importantly, can MitoQ supplementation offer a prophylactic effect to the mitochondrial genome".

The researchers found that acute MitoQ treatment did not impact on any biomarkers. However, chronic MitoQ treatment attenuated lymphocyte mtDNA damage, human muscle mtDNA damage caused by HIIE.

"We believe the findings of this study are of great importance as it disclose valuable bioavailability features of MitoQ that will aid the optimization of the design of future studies. Further, the study adds to our understanding of molecular adaptations of

exercise. As a final noteworthy point, the notion that a protective effect of MitoQ was only unmasked by exercise, reinforces the value of interrogating multiple physiological states when appraising the efficacy of an antioxidant."