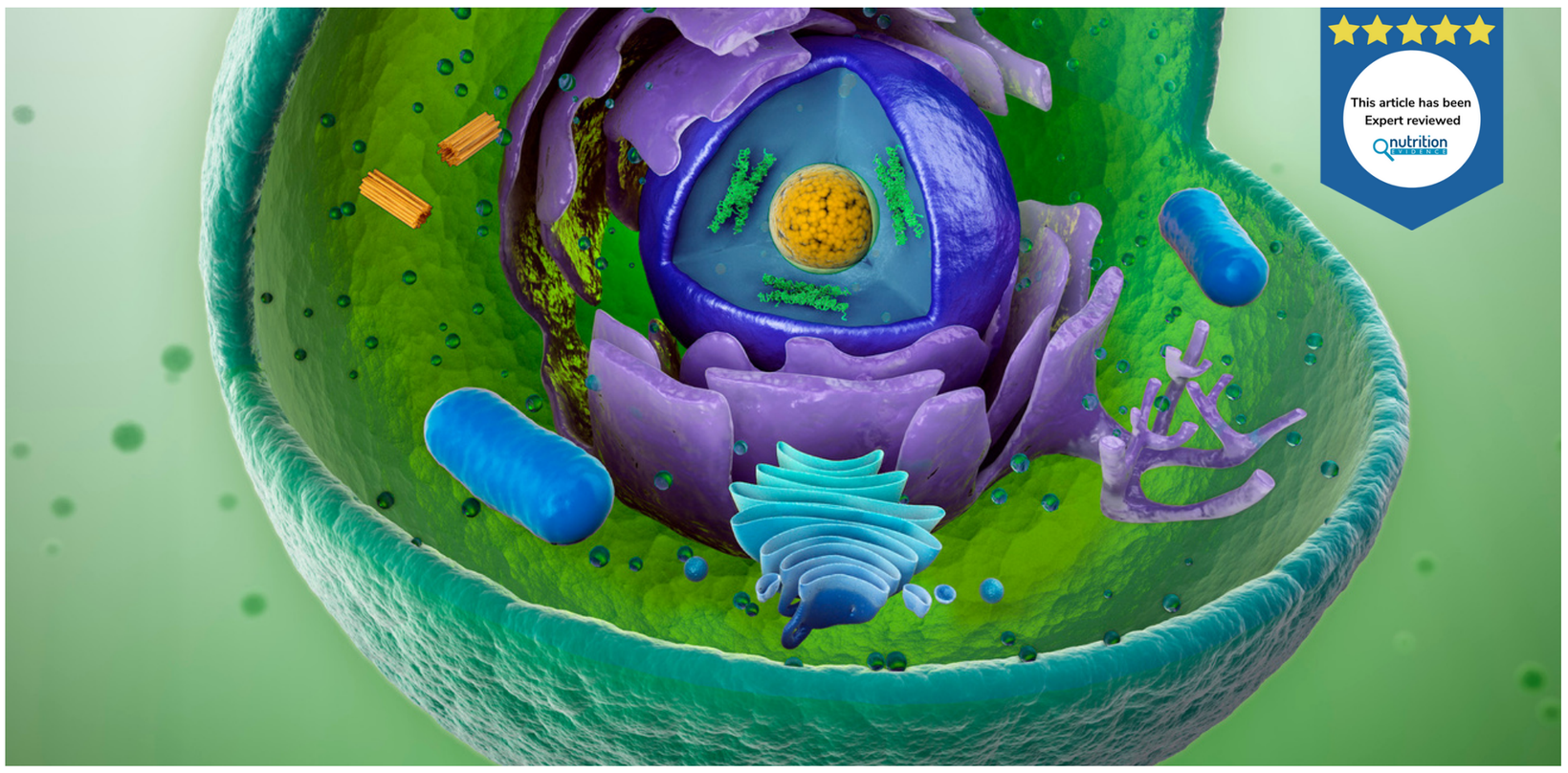
Mitochondrial Function & Health



COMBINED EPIGALLOCATECHIN-3-GALLATE AND RESVERATROL SUPPLEMENTATION FOR 12 WK INCREASES MITOCHONDRIAL CAPACITY AND FAT OXIDATION, BUT NOT INSULIN SENSITIVITY, IN OBESE HUMANS: A RANDOMIZED CONTROLLED TRIAL

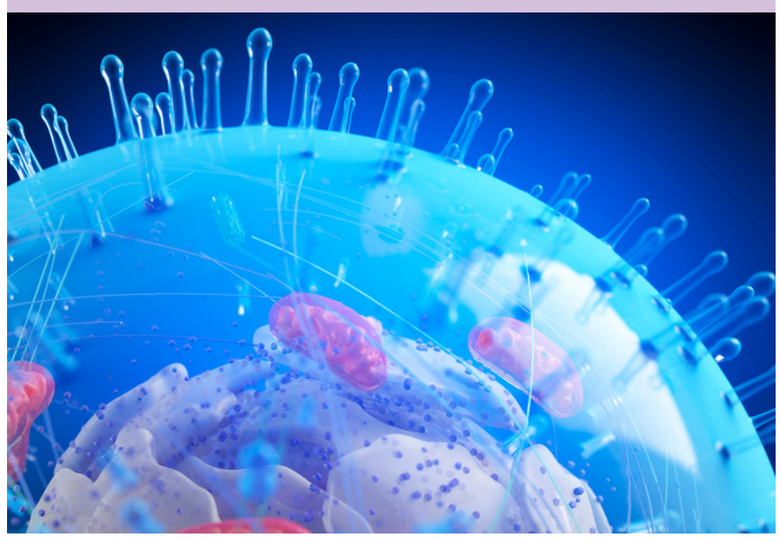
Most, J ; Timmers, S ; Warnke, I ; Jocken, JW ; van Boekschoten, M ; de Groot, P ; Bendik, I ; Schrauwen, P ; Goossens, GH ; Blaak, EE
The American journal of clinical nutrition. 2016;104(1):215-27

The prevalence of obesity and related chronic diseases is continuously increasing. Insulin resistance is a major risk factor for the progression of obesity toward chronic metabolic diseases, including cardiovascular disease and type 2 diabetes. Polyphenols were identified as dietary ingredients with antioxidant properties decades ago. Epigallocatechin-3-gallate (EGCG), which is most abundant in green tea, and resveratrol (RS), which is present in grape skins, have been implicated in the prevention of body weight gain and improvements in markers of insulin sensitivity in human and animal studies. The aim of this randomised control study was to investigate the longer-term effect of EGCG and RES (EGCG+RES) supplementation on metabolic profile, mitochondrial capacity, fat oxidation, lipolysis, and tissue-specific insulin sensitivity. 38 overweight and obese men and women received supplementation with either EGCG+RES (282 and 80 mg/d, respectively) or a placebo for 12 weeks. Before and after the intervention, oxidative capacity, lipid metabolism and insulin sensitivity were measured. EGCG+RES supplementation did not affect the fasting plasma metabolic profile. Although whole-body fat mass was not affected, visceral adipose tissue mass decreased after the intervention compared with placebo. EGCG+RES supplementation significantly increased oxidative capacity in muscle fibres. Fat oxidation and energy expenditure were not significantly affected by EGCG+RES. Finally, EGCG+RES had no effect on insulin-stimulated glucose disposal, suppression of endogenous glucose production, or lipolysis. The authors concluded that 12 weeks of EGCG+RES supplementation increased mitochondrial capacity and stimulated fat oxidation compared with placebo, and this may improve physical condition and play a role in the prevention of weight gain and worsening of insulin resistance in the long term.

A HIGH-CARBOHYDRATE DIET LOWERS THE RATE OF ADIPOSE TISSUE MITOCHONDRIAL RESPIRATION

Bikman, BT ; Shimy, KJ ; Apovian, CM ; Yu, S ; Saito, ER ; Walton, CM ; Ebbeling, CB ; Ludwig, DS
European journal of clinical nutrition. 2022;76(9):1339-1342

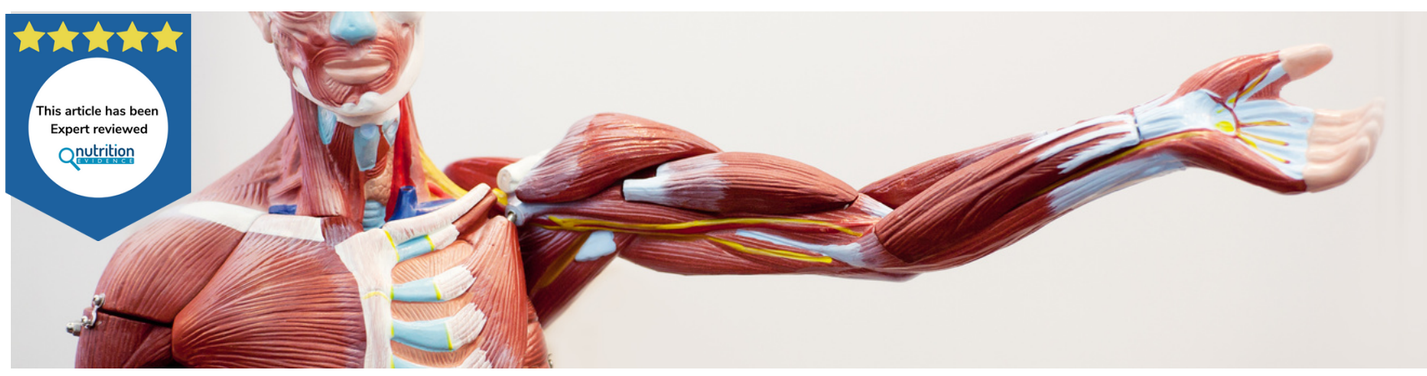
The hormone insulin plays a fundamental role in cellular nutrient signalling, including mitochondrial function.
The aim of this study was to test the hypothesis that a high-carbohydrate diet would lower measures of mitochondrial respiration in adipose tissue, consistent with the carbohydrate-insulin model of obesity.
This study is an ancillary study of the Framingham State Food Study, in which the primary outcome was total energy expenditure. Data of twenty-seven participants were included in this report.
Results show that a high-carbohydrate diet lowers mitochondrial respiratory function.
Authors conclude the study's sample may not reflect mitochondrial activity in all body fat depots.
Thus, further research is required in order to replicate the study's findings, conduct quantitative energetic studies, examine generalizability to other populations and experimental conditions, and explore translation to the prevention and treatment of obesity.



EFFECT OF MITOCHONDRIAL-TARGETED ANTIOXIDANTS ON GLYCAEMIC CONTROL, CARDIOVASCULAR HEALTH, AND OXIDATIVE STRESS IN HUMANS: A SYSTEMATIC REVIEW AND META-ANALYSIS OF RANDOMIZED CONTROLLED TRIALS

Mason, SA ; Wadley, GD ; Keske, MA ; Parker, L
Diabetes, obesity & metabolism. 2022;24(6):1047-1060

Reactive oxygen species (ROS) are free radical oxygen molecules produced by mitochondria, which cause molecular damage known as oxidative stress. Chronic diseases such as diabetes, heart disease, cancer, and Parkinson's disease are more likely to develop when ROS levels are elevated. Mitochondrial-targeted antioxidants (mitoAOX) may be effective in treating chronic diseases by targeting mitochondrial ROS. In this systematic review and meta-analysis, 19 randomised controlled trials were included to evaluate the effects of mitoAOXs on glycaemic control, cardiovascular health, and oxidative stress in humans. The evidence is limited, but there were improvements in endothelial function, blood pressure, oxidative stress, and functional capacity. Due to the heterogeneity of studies included in this study, there is a need for larger, longer-term robust studies to investigate mitoAOXs' effects on mitochondrial ROS and markers of oxidative stress in different clinical populations.



UROLITHIN A IMPROVES MUSCLE STRENGTH, EXERCISE PERFORMANCE, AND BIOMARKERS OF MITOCHONDRIAL HEALTH IN A RANDOMIZED TRIAL IN MIDDLE-AGED ADULTS

Singh, A ; D'Amico, D ; Andreux, PA ; Fouassier, AM ; Blanco-Bose, W ; Evans, M ; Aebischer, P ; Auwerx, J ; Rinsch, C - Cell reports. Medicine. 2022;3(5):100633

A gradual decline in muscle mass and strength with aging is natural, however, environmental factors such as diet and exercise dictate the trajectory of the decline. Exercise and healthy nutrition are the primary interventions to prevent and manage age-associated decline in muscle health and metabolic diseases. This study was designed as a proof-of-concept investigation of the efficacy of long-term oral supplementation with urolithin A (UA) on physiological endpoints in middle-aged adults. This study is a randomised, double-blind, placebo-controlled study of an overweight middle-aged population with a high body mass index and average physical endurance. Results showed improved lower-body muscle strength in the hamstring skeletal muscle at both doses of UA. Furthermore, it positively impacted aerobic endurance and physical-performance measures such as walking distance. Authors conclude that supplementation with UA is safe and increases circulating levels of UA.