

Mitochondrial Biogenesis: A Great Evaluation, Adaptation in Mitochondria Complex Powerhouse of the Cell ATP Generator

Roopali Kashyap

M.Sc. Biotechnology, Barkatullah University

Corresponding author's email: [roopali605kashyap\[at\]gmail.com](mailto:roopali605kashyap[at]gmail.com)

Abstract: *The scientific study pointed the aging of the mitochondrial cells lead to the myopathies of the older person generally. It is generally due to anaerobic respiration in the human, but in trees it is opposite to it. Sometimes it is proven that the mutation of the genome also the cause behind the aging. So, the reason beneath the stress is the older cells, describing the effects of exercise on mitochondrial biogenesis and its relevance for adipocyte function. Mitochondrial genesis the more blood vessels due to more blood capillary carrying oxygen with speedup action of more ATP production. Thus, the resulting impairment of aerobic ATP production results in increased glucose uptake, and glycolysis flux and lessen the anaerobic respiration*

Keywords: human mitochondrial genome, metabolic pathways, bio genesis, stress.

1. Introduction

Mitochondrial biogenesis is a major adaptation of skeletal muscle to exercise training and is induced by a complex interplay between numerous signaling pathways that respond to metabolic, mechanical, and hypoxic stresses that are generated within the myocyte during contraction. Manipulation of mitochondrial biogenesis can be a therapeutic strategy amenable to pharmaceutical intervention is under evaluation. Mitochondrial dysfunction occurs when the mitochondria don't work as well as they should due to another disease or condition.¹ Many conditions can lead to secondary mitochondrial dysfunction and affect other diseases, including Alzheimer's disease, muscular dystrophy, Lou Gehrig's disease, diabetes, and cancer. May 31, 2018, vitamins help mitochondria. B vitamins and lipid acid are essential in the tricarboxylic acid cycle, while selenium, α -troposphere, Coenzyme Q10, caffeine, and melatonin are suggested to boost the electron transfer system function. Carnitine is essential for fatty acid beta-oxidation. Selenium is involved in mitochondrial biogenesis. Together, Leigh syndrome and MELAS are the most common mitochondrial myopathies.² The prognosis of Leigh syndrome is generally poor, with survival generally being a matter of months after disease onset test for mitochondrial damage? They include biochemical tests on urine, blood, and spinal fluid. A muscle biopsy to examine the mitochondria and test enzyme levels magnetic resonance imaging (MRI) of the brain and spine.

Mitochondria are the cells' powerhouse, but also their suicidal weapon store. Dozens of lethal signal transduction pathways converge on mitochondria to cause the permeabilization of the mitochondrial outer membrane, leading to the cytosolic release of pro-apoptotic proteins and to the impairment of the bioenergetics functions of mitochondria. Resveratrol — a polyphenolic compound from grapes and wine — has recently been shown to improve mitochondrial function by stimulating the sirtuin 1 (SIRT1)-dependent deacetylation of the transcriptional co-activator peroxisome proliferator-activated receptor- γ .³ The topic of mitochondria may seem dry and uninteresting to the uninitiated, but this book makes mitochondria come to life with vivid descriptions accessible even to those with not training in biology. From infertility to ageing to cancer and neurological disease, Dr. Lee Know will teach you that mitochondria play a central role in much that we care about in health and disease. Stephanie Seneff, Ph.D., senior research scientist, MIT Computer Science and Artificial Intelligence Laboratory. The circular 16,569 bp human mitochondrial genomes encoding 37 genes, i.e., 28 on the H-strand and 9 on the strand. Mitochondria contain their genome, an indication that they are derived from bacteria through endosymbiosis. However, the ancestral endosymbiont genome has lost most of its genes, so that, the mitochondrial genome (mitogenome) is one of the most reduced genomes across organisms.

Mitochondrial Biogenesis

Salvage 3- Lack of exercise and Physical activity

Mitochondrial Biogenesis:

Exercise is one of the most powerful signals for PGC 1-alpha

A protein encoded by PPARGC1A gene (Peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC-1 α))

PGC 1-alpha:

Is the primary signal for **Mitochondria to Reproduce and Multiply**, a process called **Mitochondrial biogenesis**

(PGC-1 α) is a protein encoded by the PPARGC1A gene, known as human accelerated region 20 (HAR20).

PGC-1 α is a transcriptional co activator that regulates the genes involved in energy metabolism. It is the master regulator of mitochondrial biogenesis.



PPARGC1A



Figure 2: Show that mitochondrial biogenesis also deals with aerobic exercise

2. Mitochondrial history

The human mitochondrial genome is a circular DNA molecule of about 16 kilobases. It encodes 37 genes: 13 for subunits of respiratory complexes, I, III, IV and V, 22 for mitochondrial tRNA (for the 20 standard amino acids, plus an extra gene for leucine and serine), and 2 for rRNA. One mitochondrion can contain two to ten copies of its DNA. As in prokaryotes, there is a very high proportion of coding DNA and an absence of repeats. Mitochondrial genes are transcribed as multigenic transcripts, which are cleaved and polyadenylated to yield mature mRNAs. Not all proteins necessary for mitochondrial function is encoded by the mitochondrial genome; most are coded by genes in the cell nucleus and the corresponding proteins are imported into the mitochondrion.⁴ The exact number of genes encoded by the nucleus and the mitochondrial genome differs between species. Most mitochondrial genomes are circular, although exceptions have been reported. In general, mitochondrial DNA lacks introns, as is the case in the human mitochondrial however, introns have been observed in some eukaryotic mitochondrial DNA, such as that of yeast and protists, including *Dictyostelium discoideum*. Between protein-coding regions, tRNAs are present. During transcription, the tRNAs acquire their characteristic L-shape that gets recognized and cleaved by specific enzymes.

Mitochondrial tRNA genes have different sequences from the nuclear tRNAs but lookalikes of mitochondrial tRNAs have been found in the nuclear chromosomes with high sequence similarity. The dysfunction occurs when the mitochondria don't work as well as they should due to another disease or condition. Many conditions can lead to secondary mitochondrial dysfunction and affect other diseases, including Alzheimer's disease, muscular dystrophy, Lou Gehrig's disease, diabetes and cancer. May 31, 2018 vitamins help mitochondria.

B vitamins and lipid acid are essential in the tricarboxylic acid cycle, while selenium, α -troposphere, Coenzyme Q10, caffeine, and melatonin are suggested to boost the electron transfer system function. Carnitine is essential for fatty acid beta-oxidation. Selenium is involved in mitochondrial biogenesis. Together, Leigh syndrome and MELAS are the **most common mitochondrial** myopathies. The prognosis of Leigh syndrome is generally poor, with survival generally being a matter of months after **disease** onset test for mitochondrial damage? **They include** biochemical **tests** on urine, blood and spinal fluid. A muscle biopsy to examine the **mitochondria** and **test** enzyme levels magnetic resonance imaging (MRI) of the brain and spine.⁵

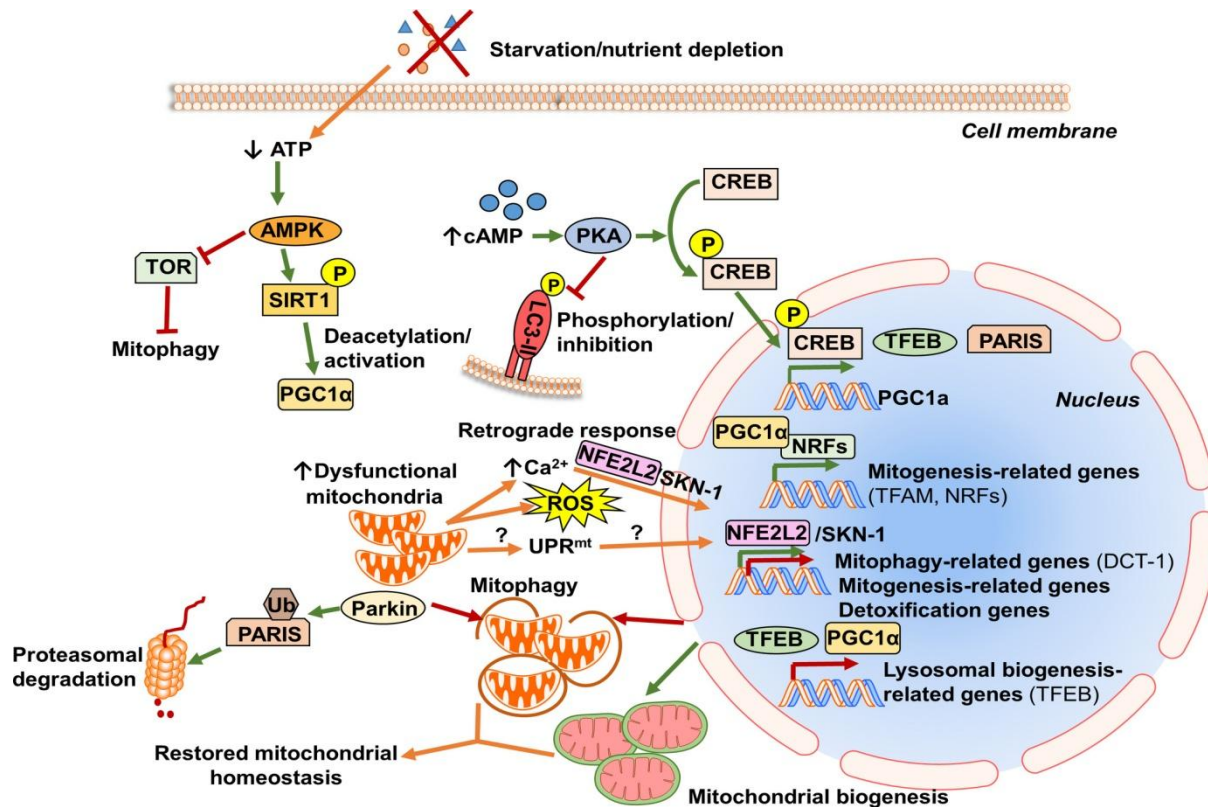


Figure 1: Mitochondrial biogenesis

3. Mitochondrial Adaptations to Aerobic Training

Mitochondria are the cells' powerhouse, but also their suicidal weapon store. Dozens of lethal signal transduction pathways converge on mitochondria to cause the permeabilization of the mitochondrial outer membrane, leading to the cytosolic release of pro-apoptotic proteins and to the impairment of the bio energetic functions of mitochondria.⁶ Resveratrol— a polyphenolic compound from grapes and wine — has recently been shown to improve mitochondrial function by stimulating the sirtuin 1 (SiRT1)-dependent deacetylation of the transcriptional co-activator peroxisome proliferator-activated

So, you should eat grapes must..... The best trainers can educate their clients as well as help them achieve their goals. When your clients who love to lift weights and do intense HIIT workouts don't want to jog or spending 30 minutes on the stair climber, how do you explain your reasoning.⁷ Aerobic workouts are important for overall fitness, even for non-endurance athletes. They improve cardiovascular fitness, increase the time to fatigue, burn fat, and promote recovery in muscle tissue. Help your resistant clients understand these benefits on a deeper level, so they are more motivated to fit in essential, aerobic workouts. What Do the Mitochondria Actually Do? Most people at least remember from high school or college biology that the mitochondria are the "powerhouses of the cell." In other words, they produce energy, and cells in muscle tissue are loaded with them. Aerobic training helps mitochondria make energy more efficiently.

Through a series of adaptations exercise improves the speed and efficiency of these processes:

- Glycolysis. During the process of glycolysis enzymes in the mitochondria break down glucose molecules, changing them into compounds called pyruvates. This reaction does not require oxygen and produces two molecules of ATP, the body's energy currency.
- The Krebs cycles. Pyruvates then move on to the Krebs cycle, a series of reactions again catalyzed by enzymes. The end of the cycle results in two more molecules of ATP.
- The electron transport chain. The other molecules produced in the Krebs cycle move to the electron transport chain, other series of chemical reactions resulting in a whopping 28 molecules of ATP.

All together the mitochondria produce 32 ATP molecules for energy with just one cycle of reactions. While the first step is anaerobic, most of the process requires oxygen. So, the more oxygen that gets to muscle cells, and the more quickly this happens, the faster and more efficiently the mitochondria can produce energy.⁸ Aerobic Mitochondrial Adaptations There seem to be multiple ways in which the mitochondria adapt in response to aerobic exercise, all of which ultimately lead to greater fitness: Aerobic exercise uses oxygen, which directly relates to how the mitochondria function. The more oxygen you pump to your muscle cells, and their mitochondria through aerobic workouts, the faster and better they work to produce energy. There is also an increase in mitochondrial enzymes.⁹ These proteins that are necessary for the metabolic reactions in the mitochondria seem to be increased when the body undergoes aerobic exercise.

The overall density of mitochondria in muscle tissue increases in response to aerobic workouts. More mitochondria mean greater use of oxygen to produce more

ATP and energy. Aerobic exercise also leads to an increase in myoglobin in muscle tissue. This is a protein that stores oxygen and transports it into cells, so that, mitochondria can use it to make more ATP molecules.¹⁰ Working out aerobically has been shown to trigger angiogenesis, the growth of new blood vessels. Blood vessels transport blood and oxygen to muscles, and with more of them the mitochondria can get and use oxygen more quickly and

efficiently. Could Mitochondrial Adaptations Help You Live Longer? There is also some research that suggests ageing in humans are related to mitochondrial changes.¹¹ Ageing is a complicated process that is not fully understood, and many factors that are involved, but studies show that damage to mitochondrial DNA, reduced ability to produce ATP, and resulting muscle weakness could be important culprits.¹²

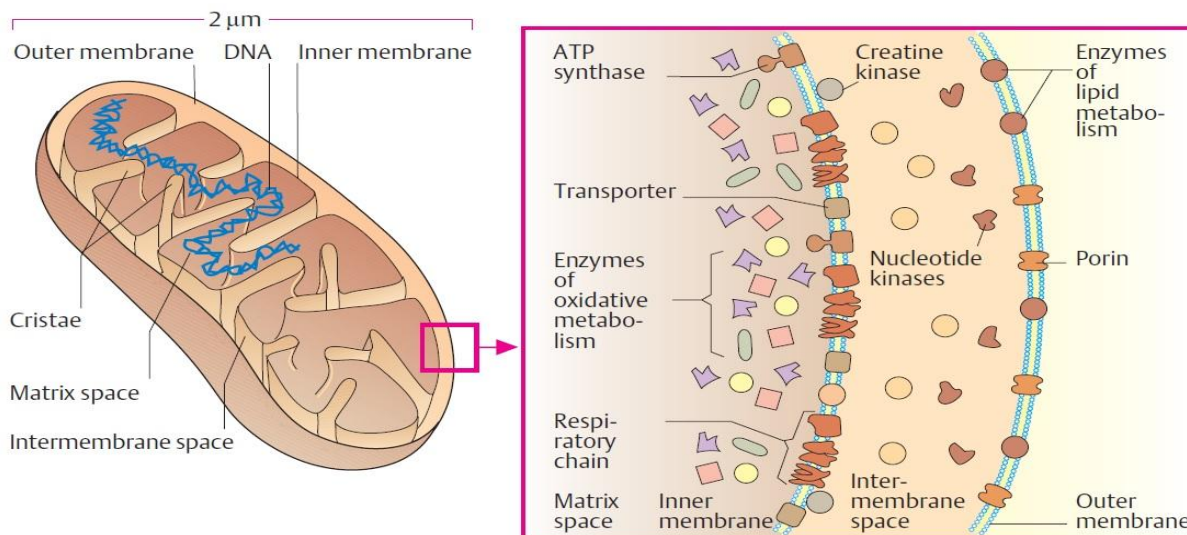


Figure 2

4. Results and Discussion

A number of studies have revealed that the reduced expression of PPAR coactivator-1 α (PGC-1 α), a key regulator of mitochondrial bio genesis, and mitochondrial dysfunction in patients with insulin resistance, type 2 diabetes, and in morbidly obese prediabetic patients.¹³ A

number of changes can occur to mitochondria during the ageing process. Tissues from elderly patients show a decrease in enzymatic activity of the proteins of the respiratory chain. Mitochondrial dysfunction has also been shown to occur in amyotrophic lateral sclerosis.¹⁴

Core regulatory molecules or complex	Effect on mitochondrial	Related disease model	Reference
Mitochondrial transcription factor A (TFAM)	Controls mtDNA copy number	Lysosomal storage disorders, sphingolipidoses	[46]
Peroxisome proliferator coactivator-1 alpha (PGC1 α)	Induces mitochondrial biogenesis, mitochondrial remodeling, respiration, gluconeogenesis and glucose transport, fatty acid oxidation, peroxisomal remodeling, and detoxification of reactive oxygen species	Muscle wasting myopathies; Cardiac ischemia-reperfusion injury; Parkinson's disease; Huntington's disease; melanoma; obesity; Retinal pigment epithelium (RPE)-associated retinal degeneration; Non-alcoholic fatty liver disease (NAFLD)	[39, 40, 42-45, 47, 52-57]
Mitochondrial respiratory chain complex I	Initial and rate limiting enzyme in electron transfer chain	Parkinson's disease (PD)	[50]
Mitochondrial respiratory chain complex II	Junction between oxidative phosphorylation and electron transport	Diabetes, obesity, and metabolic syndrome	[41]
Dynamin-related protein 1 (Drp1)	Key regulator of mitochondrial fission	Lethal dilated cardiomyopathy	[49]
PEN-induced putative kinase 1 (PINK1)	Recruits parkin resulting in ubiquitination of mitochondrial proteins	Parkinson's disease (PD)	[38, 47]
GCN5-like Protein 1 (GCN5L1)	A putative nutrient-sensing regulator, controls mitochondrial removal by autophagy	Fatty liver, Type 2 diabetes	[51]
STIP1 homology and U-Box containing protein 1 (STUB1)	Promotes ubiquitin-mediated protein degradation	Neurodegenerative diseases	[48]

literature review of mit/tfe family mitochondrial biogenesis and mitophagy -mediated

5. Conclusions

It is concluded imperative to aerobic exercise because it allows for heighten including PGC-1 α protein. ATP levels are also significant because more ATP must be generated to create the increase in mitochondrial content. It is apparent in other types of training as well as use of critical value for aerobic exercise because it generates enhanced performance. The process of mitochondria receiving oxygen for biogenesis needs constant rate of oxygen entering the body, so without this (during anaerobic exercise) mitochondria production. It is reduced during detraining due to decreased mitochondrial enzyme activity and a decreased mitochondrial ATP production rate. With aging comes a decrease in PGC-1 α , and increased triggering of the apoptotic pathway caused by ROS formation, therefore decreasing biogenesis. Extra factors that effect during exercise are Rev-erg-A, Quercetin, and Huntington's dRev-erb-A helps regulate skeletal muscle oxidative capacity which also regulates mitochondrial biogenesis. Quercetin imitates the same effect that exercising has on the skeletal muscles. Huntington's disease is a mitochondrial defect that lowers the anaerobic threshold while exercising by increasing the lactate produced. The importance by exercise is that exercise does aid mitochondrial production of understanding the process of mitochondrial biogenesis induced.

6. Competing Interests

No conflict of interest exists in this publication.

References

- [1] Lund by, C., Jacobs, R.A. (2016). Adaptations of Skeletal Muscle Mitochondria to Exercise Training. *Experimental Physiology*, 101 (1), 17-22
- [2] Margolis, L.M., Paitakes, S.M. (2013). Optimizing Intramuscular Adaptations to Aerobic Exercise: Effects of Carbohydrate Restriction, and Protein Supplementation on Mitochondrial Biogenesis. *Advanced Nutrition*, 4 (6), 657-664
- [3] Eliana, A., Brooks, K. (2013) Effect of Aerobic Exercise on Mitochondrial DNA and Ageing. *Journal of Exercise Science & Fitness*. 11 (1) 1-5
- [4] Compartmentation of metabolism within mitochondria and plastids Featured Course Caroline G. Bowsher, Alyson K. Tobin *Journal of Experimental Botany*, Volume 52, Issue 356, April 2001, Pages 513–527, <https://doi.org/10.1093/jexbot/52.356.513> Published: 01 April 2011:
- [5] Progress in Molecular Biology and Translational Science, 2015 Molecular and Cellular Regulation of Adaptation to Exercise Thomas Tsiloulis, Matthew J. Watt, I.
- [6] Progress in Molecular Biology and Translational Science, 2015
- [7] Reactive Oxygen Species and Oxidative Stress Author links open overlay panel Rhian M. Touyz <https://doi.org/10.1016/B978-0-12-386525-0.00069-X> Get rights and content

- [8] Translational models, methods, and concepts in studies of ageing and longevity Monty Montano, in *Transnational Biology in Medicine*, 2014:11.6 Mitochondrial dysfunction.
- [9] Strategy, and Drug Research. A. Dykens, in *Comprehensive Medicinal Chemistry II*, 2007 2.27.9.2.2 Pharmacological approach
- [10] Mitochondrial Dysfunction and Chronic Disease: Treatment With ... <https://www.ncbi.nlm.nih.gov/pmc>
- [11] Mitochondria—Fundamental to Life and Health — NCBI — NIH <https://www.ncbi.nlm.nih.gov/pmc>.
- [12] Which Foods Help Your Mitochondria? — MitoQ <https://www.mitoq.com/blog/whi...>
- [13] How exercise -- interval training in particular -- helps your ... <https://www.sciencedaily.com/201...>
- [14] Enhanced mitochondrial biogenesis is associated with the ameliorative action of creatine supplementation in rat soleus ... MA Gowayed · 2020 · 07-Nov-2019 · <https://www.ncbi.nlm.nih.gov/p>
- [15] <https://www.sciencedirect.com/pii> Feeding mitochondria: Potential role of nutritional components to improve critical illness convalescence — Science Direct

Certified Personal Trainer the Certified Fitness Trainer program is designed to equip graduates with the practical day-to-day skills necessary, as well as the theoretical knowledge needed to excel as a personal trainer serving the ageing.