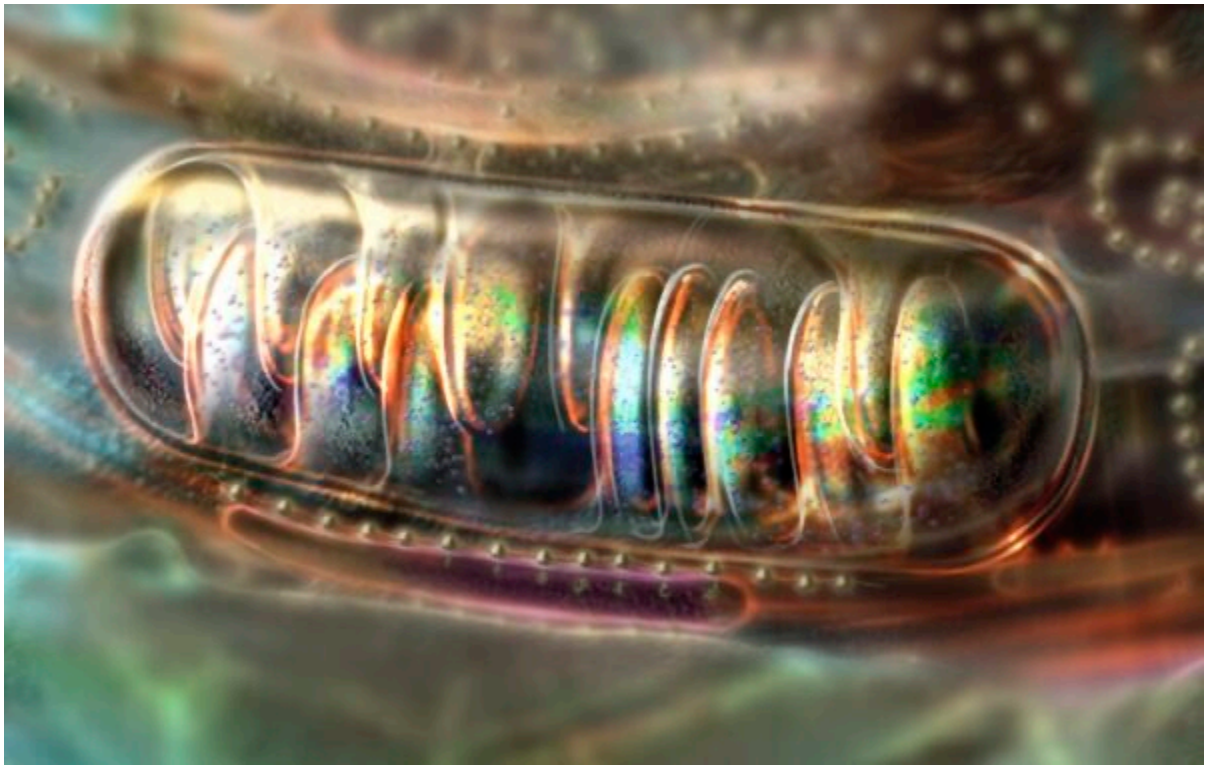


Mitochondrial Testing

Academy of Nutritional Medicine

with Gilian Crowther, Director of Research, AONM

30th October 2024

https://aonm.org/mitochondrial-testing/

Mitochondrial testing with AONM/MMD

1. **Brief introduction to the mitochondria**

2. **ATP Profile: Total ATP, Mitochondrial ATP, Glycolytic ATP, Reserve Capacity**

3. **Mitochondrial Health Index:**
   Basal respiration rate, mitochondrial ATP turnover, proton leak, maximum respiration rate, reserve capacity, non-mitochondrial rate, calculation of the overall Mitochondrial Health Index

4. **Supplementary biomarkers:**
   Ratio of mtDNA to nDNA (mtDNA:nDNA)
   PGC-1α
   Nrf-2
   Mitochondrial 4977 deletion mutant (mt4977del)
   Lactate/pyruvate ratio
   Mitochondrial Fuel Pathways
   OxPhos

ATP Profile

XXX
Max-Mustermann Straße 5
xxx Berlin

MMD

MMD GmbH & Co. KG
Breiter Weg 10a
39104 Magdeburg
Prof. Dr. Brigitte König
CEO/ Scientific Director
Prof. Dr. Gerhard Jorch
Medical Director

Tel. office: +49 391 535 37 97
Tel. laboratory: +49 391 611 72 09
Fax: +49 391 535 38 45
E-Mail: info@mmd-web.de
Web: www.mmd-web.de

Patient AW
Date of birth 01.01.1990
Entry on 23.07.2021
Order No.:

Date of sample 22.07.2021
Sample type CPDA vacutainer
Results status **Final report**
Validated by Prof. Dr. Brigitte König
Cell type PBMC
Results status on 23.07.2021

ATP profile

Test	Result	Unit	Reference range	Result [%]
Total ATP	0.8	fmol/cell		
Mitochondrial ATP capacity	0.4	fmol/cell		50
Glycolytic ATP capacity	0.5	fmol/cell		63
Reserve ATP capacity	0.10	fmol/cell		13

Reference range total ATP								
fmol/cell	<0.8	0.8 - 1.0	1.0 - 1.2	1.2 - 1.4	1.4 - 1.6	1.6 - 2.0	2.0 - 2.5	2.5 - 3.0	3.0 - 5.0

Reference range mitochondrial ATP capacity					
fmol/cell	<0.8	0.8 - 1.0	1.0 - 1.2	1.2 - 1.4	>1.4

Reference range glycolytic ATP capacity					
fmol/cell	<0.8	0.8 - 1.0	1.0 - 1.2	1.2 - 1.4	>1.4

Reference range reserve ATP capacity								
fmol/cell	<0.2	0.2 - 0.3	0.3 - 0.4	0.4 - 0.6	0.6 - 0.9	0.9 - 1.0	1.0 - 1.2	1.2 - 1.5	>1.5

Total ATP

ATP profile

Test	Result	Unit	Reference range	Result [%]
Total ATP	0.8	fmol/cell		

Reference range total ATP								
fmol/cell	<0.8	0.8 - 1.0	1.0 - 1.2	1.2 - 1.4	1.4 - 1.6	1.6 - 2.0	2.0 - 2.5	2.5 - 3.0	3.0 - 5.0

Total ATP

This is the quantity of ATP that the cells produce at rest via both mitochondrial and non-mitochondrial pathways. Total ATP is all the adenosine triphosphate (our cells' energy currency) available to the cell. This makes it possible to assess the relative performance of mitochondrial respiration (mitochondrial ATP capacity) versus anaerobic glycolysis (glycolytic ATP capacity).

Mitochondrial and glycolytic ATP capacity

Test	Result	Unit	Reference range	Result [%]
Mitochondrial ATP capacity	0.4	fmol/cell		50

Reference range mitochondrial ATP capacity

fmol/cell	<0.8	0.8 - 1.0	1.0 - 1.2	1.2 - 1.4	>1.4

Mitochondrial ATP capacity measures the capacity to synthesise adenosine triphosphate (ATP) in the patient's mitochondria in a defined basal state. This is calculated by determining the absolute ATP production that is inhibited by addition of the ATP synthase inhibitor oligomycin (see figure above).

Glycolytic ATP capacity	0.5	fmol/cell		63

Reference range glycolytic ATP capacity

fmol/cell	<0.8	0.8 - 1.0	1.0 - 1.2	1.2 - 1.4	>1.4

ATP can also be produced in the cytosol, outside the mitochondria (though still inside the cell). This parameter measures the glycolytic capacity for ATP production: the maximum quantity of ATP that the cells are able to produce at rest via non-mitochondrial pathways, i.e. anaerobic glycolysis. This makes it possible to assess the relative performance of anaerobic glycolysis versus mitochondrial respiration. It is important to have a high glycolytic capacity in the cells so that sufficient precursors for the Krebs Cycle can be made to then be cycled into the ETC, and also so that the cytosolic production of ATP (glycolysis) can be upregulated if needed, when immune cells need to address pathogens, etc.

Reserve ATP capacity

ATP profile

Test	Result	Unit	Reference range	Result [%]
Reserve ATP capacity	0.10	fmol/cell		13

Reference range reserve ATP capacity									
fmol/cell	<0.2	0.2 - 0.3	0.3 - 0.4	0.4 - 0.6	0.6 - 0.9	0.9 - 1.0	1.0 - 1.2	1.2 - 1.5	>1.5

ATP synthesis is generally presumed to be coupled almost entirely to two metabolic processes: oxidative phosphorylation and glycolysis. There is however another essential metabolic process that interconverts the three adenine nucleotides (ATP, ADP and AMP) using adenylate kinase according to metabolic needs. Adenylate kinase catalyses a reversible reaction: 2 ADP > ATP + AMP. This is a vital factor in regulating the energy charge in cells, providing an open system able to accept, store and supply energy to cells as needed. The marker “Reserve ATP capacity” indicates how dynamically the cell is able to perform this catalytic interconversion.

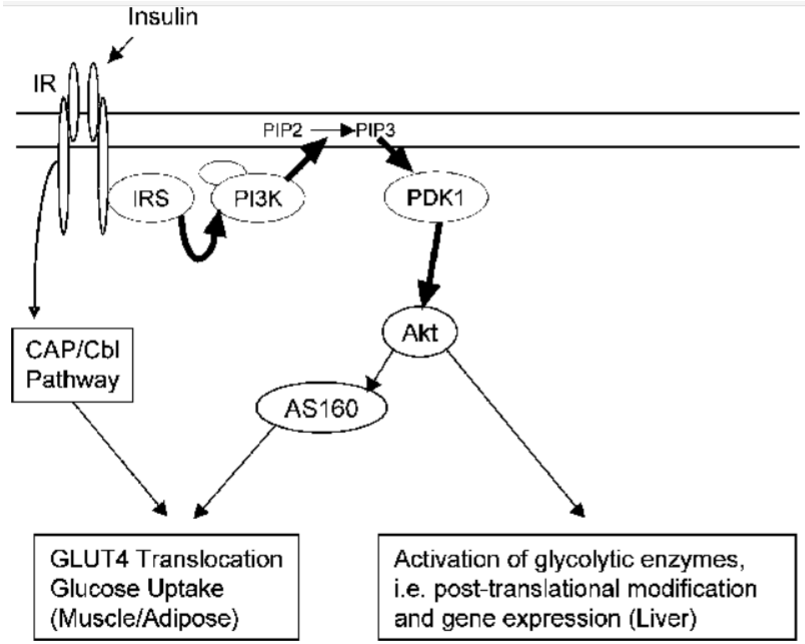
Here, the reserve ATP capacity is 13 %/ 0.10 fmol/ cell. The patient's result is in the very low range. The optimal would be between 0.6 to 0.9 fmol/cell.
13 % means that the cell is unable to perform dynamic catalytic interconversion between the three adenine nucleotides (ATP, ADP and AMP) according to metabolic needs.

Impaired glycolytic ATP capacity: Could insulin resistance be an issue? In 95% of the US population, Dr. Pizzorno said recently*

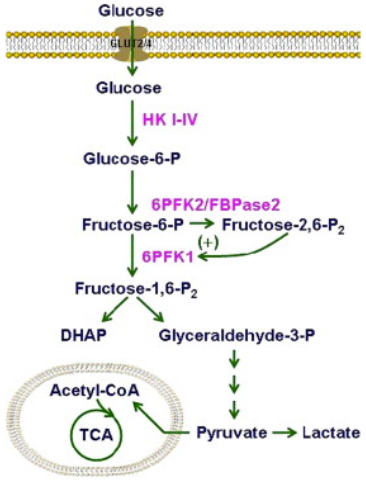
Insulin plays a significant role in this array of glycolysis regulation. In the short term, insulin, through insulin signaling pathways controls glucose entry and regulates the levels of F-2,6-P_2, a key regulator of glycolysis.

ScienceDirect.com
https://www.sciencedirect.com › article › abs › pii

Regulation of glycolysis—role of insulin - ScienceDirect.com



Scheme outlining insulin signaling and the regulation of glycolysis. In various tissues, insulin controls distinct components of glycolysis. IR, insulin receptor; IRS, insulin receptor substrate; PI3K, phosphotidylinositol-3-kinase; PIP2, phosphotidylinositol-,4,5-phosphate; PIP3, phosphotidylinositol-3,4,5-phosphate; GLUT4, glucose transport 4; and PDK, phosphotidylinositol-dependant kinase.

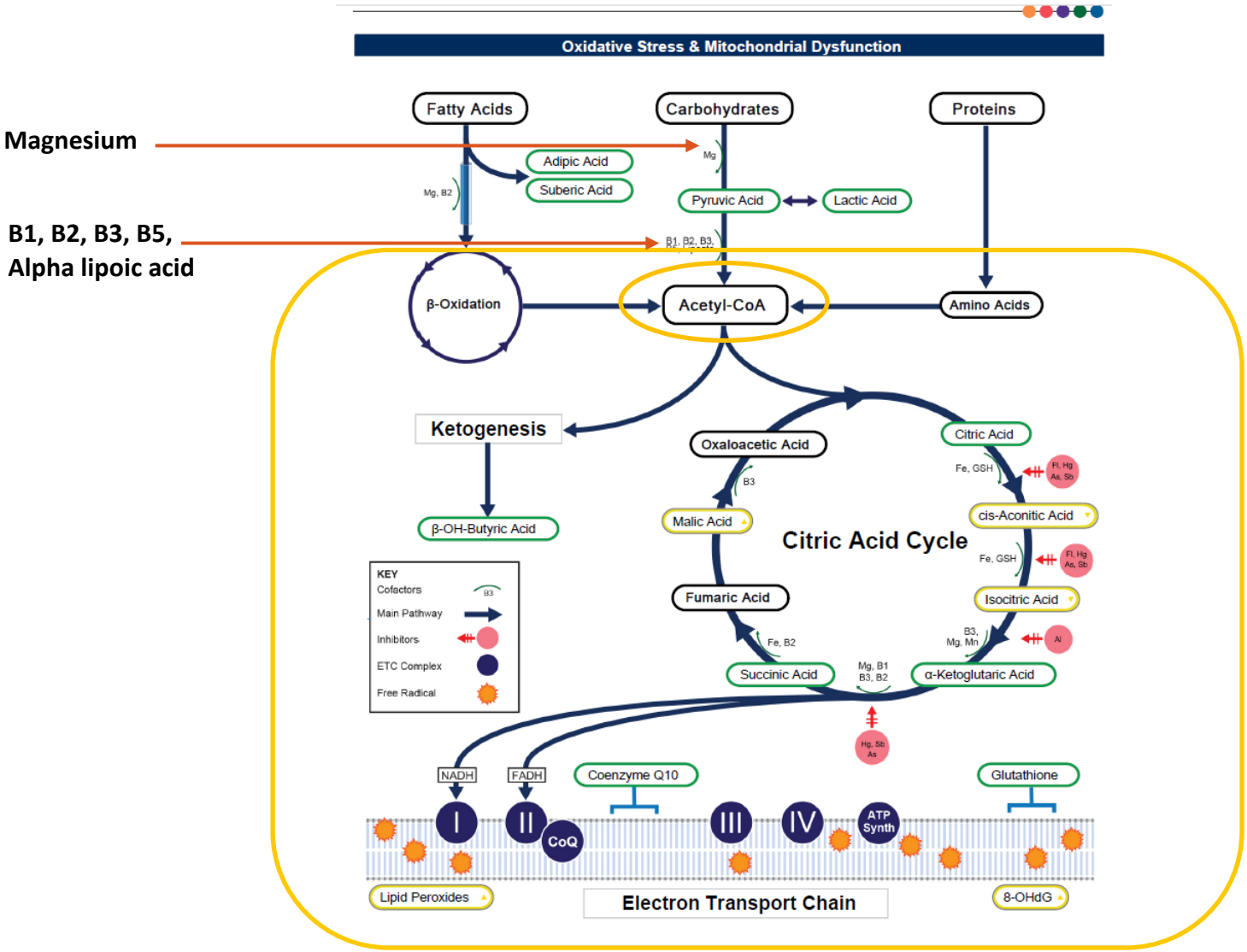


Download: Download full-size image

Figure 1. Major steps of glycolysis. Glycolysis is the pathway for the generation of pyruvate/lactate from glucose. Depending on cell types in which glycolysis occurs, glucose uptake is mediated mainly by glucose transporter 2 (GLUT2) or GLUT4. Following glucose uptake, rates of glycolysis are determined at steps of glucose phosphorylation, which is catalyzed by hexokinase II or hexokinase IV (glucokinase, GK), and the generation of fructose-1,6-bisphosphate, which is catalyzed by 6-phosphofructo-1-kinase (6PFK1). The latter is activated by fructose-2,6-bisphosphate (F2,6P_2), whose production is controlled by 6-phosphofructo-2-kinase/fructose-2,6-bisphosphatase (6PFK2/FBPase2). DHAP, dihydroxyacetone phosphate; TCA, tricarboxylic acid cycle.

Source: 1. Wu C, Khan SA, Lange AJ. Regulation of glycolysis-role of insulin. Exp Gerontol. 2005 Nov;40(11):894-9;
2. Hers HG. Mechanisms of blood glucose homeostasis. J Inherit Metab Dis. 1990;13(4):395-410; Nutrition Medicine Institute (NMI) Summit, London, "Mitochondrial Nutrition for Energy, the Brain, and Healthy Ageing"< Oct. 11/12, 2024

Impaired glycolytic ATP capacity: Cofactors may be lacking to get pyruvic acid into the mitochondria



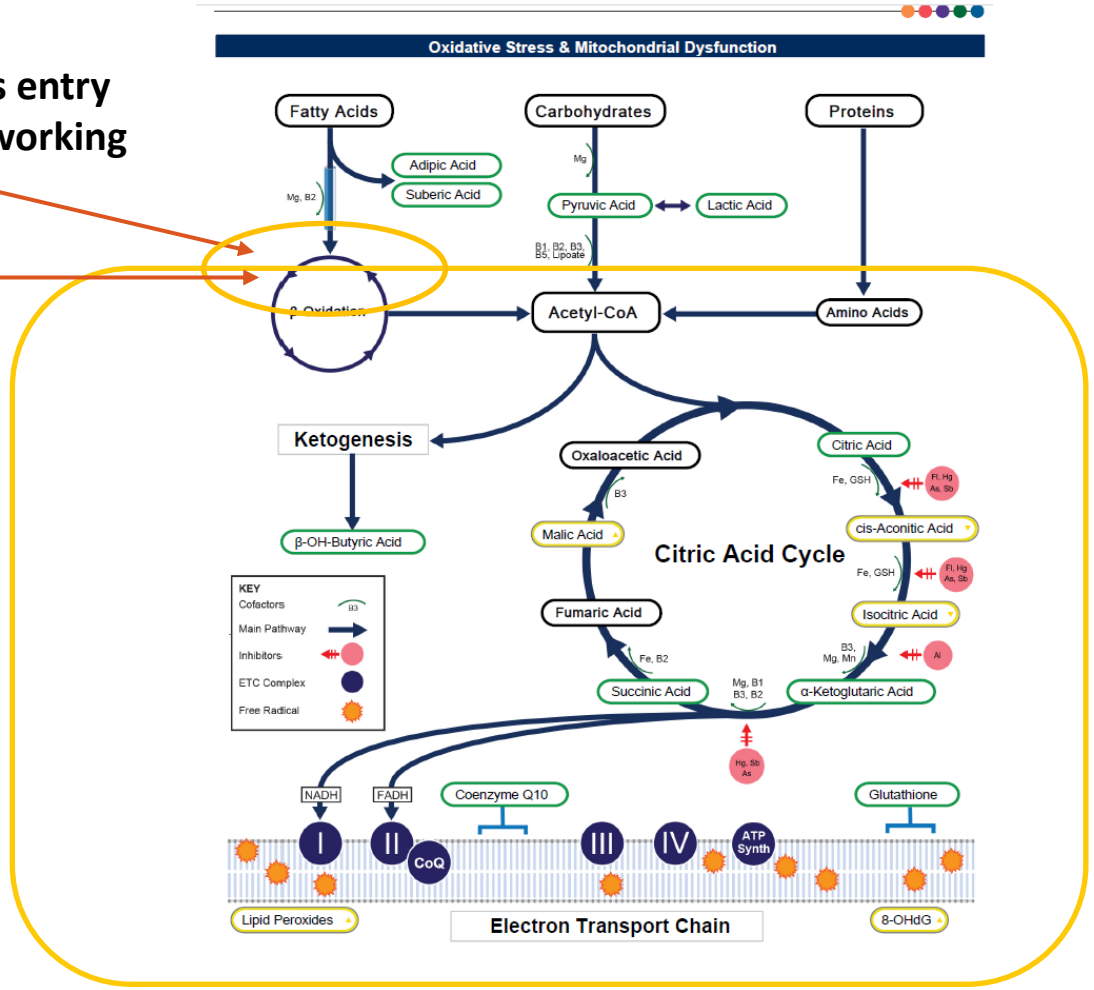
Source: ©Genova Diagnostics ▪ A.L. Peace-Brewer, PhD, D(ABMLI), Lab Director ▪ CLIA Lic. #34D0655571 ▪ Medicare Lic #34-8475 (with permission)

If mitochondrial ATP capacity on the ATP profile is stronger: fatty acids are getting in

Mitochondrial ATP capacity	1.33	fmol/cell	84
Glycolytic ATP capacity	1.12	fmol/cell	70

Then this entry point is working better

Carnitine!



There are however mitochondrial fatty acid oxidation disorders (FAODs) (inherited metabolic diseases) that affect the transport of fatty acids into mitochondria:

- **Carnitine transporter defect**
- **Carnitine-acylcarnitine translocase (CACT) deficiency**
- **LCHAD deficiency**
- **Etc.**

Source: ©Genova Diagnostics ▪ A.L. Peace-Brewer, PhD, D(ABMLI), Lab Director ▪ CLIA Lic. #34D0655571 ▪ Medicare Lic #34-8475 (with permission)

Mitochondrial testing with AONM/MMD

1. **Brief introduction to the mitochondria**

2. **ATP Profile:** **Total ATP, Mitochondrial ATP, Glycolytic ATP, Reserve Capacity**

3. **Mitochondrial Health Index:**
   Basal respiration rate, mitochondrial ATP turnover, proton leak, maximum respiration rate, reserve capacity, non-mitochondrial rate, calculation of the overall Mitochondrial Health Index

4. **Supplementary biomarkers (next time!):**
   Ratio of mtDNA to nDNA (mtDNA:nDNA)
   PGC-1α
   Nrf-2
   Mitochondrial 4977 deletion mutant (mt4977del)
   Lactate/pyruvate ratio
   Phase 2:
   Number of mitochondria
   Intact mitochondria versus Non-intact mitochondria

Mitochondrial Health Index: top page

Requisition:Mitochondrial Health Index / PBMCs

Sample type: Blood in CPDA vials

Summary

	Patient´s value	Target value (optimal)
Mitochondrial Health Index (MHI)	0.00	>2.5
Mitochondrial Bioenergetics		
Coupling efficiency, %	86	90-95
Reserve respiration capacity, %	0	>400
Cellular oxygen consumption profile		
Non-mitochondrial respiration as a share of total respiration, %	32	<10
Proton leak as a share of total respiration, %	10	5-10
Share of respiration used for mitochondrial ATP generation, %	58	>90
ATP turnover rate (mitochondrial oxygen utilisation)		
ATP base turnover, %	100	<20
ATP reserve, %	0	>80
Basal oxygen consumption rate in pmol oxygen/min	28.75	
Potential maximum oxygen consumption rate in pmol oxygen/min	22	>500
Cellular energy phenotype		
At rest	Resting	Resting
On energy demand	Resting	Energetic/Aerobic
Metabolic potential, mitochondrial percentage	84	>350
Metabolic potential, glycolysis percentage	151	>350
Oxygen consumption/glycolysis on energy demand	Strong preference for anaerobic glycolysis	

Optimal	Slightly high / low	Moderately high/low	Very high/low	Extremely high/low

Summary relating to mitochondrial dysfunction: selected markers

	None	Slight	Moderate	Considerable	Extreme
Mitochondrial dysfunction					√
Cellular imbalance				√	

Indications of					
Increased formation of oxygen radicals in the cell	√	No Yes	Insufficient ATP formation on energy demand	√	No Yes
Increased formation of oxygen radicals in the mitochondria	✓	No Yes	Limited glucose utilisation		No Yes
Restricted function of the electron transport chain in the mitochondria	√	No Yes	Limited fatty acid oxidation		No Yes
Limited number of functionally intact mitochondria	√	No Yes	Acute inflammation, active chronic inflammation/ autoimmune disease	✓	No Yes

Further diagnostic opportunities for personalised therapy

Investigate minerals and further mitochondrial cofactors
Investigate mitochondrial mass (mtDNA:nDNA/number of mitochondria) and analyse mitochondrial mutations that influence ATP generation (e.g., the common deletion mt4977bp).

- **Upregulated ROS in the cells**
- **Compromised function of the electron transport chain**
- **Limited no. of functionally intact mitochondria**
- **Insufficient ATP on demand**

Comparison of various tests

Patient	xxxxxxx
Date of birth	19.07.1941
Sample taken	15.06.2021
Receipt of sample	16.06.2021
Test completed	16.06.2021
Final result	**16.06.2021**
Validated by	Prof. Dr. Brigitte König
Medical Director	Prof. Dr. Gerhard Jorch

Comparison with previous values

	28.10.2020	19.05.2021	Current value 16.06.2021	Target value (optimal)
Mitochondrial Health Index (MHI)	1.87	1.54	1.90	>2.5
Mitochondrial bioenergetics				
Coupling efficiency, %	94.76	84.62	93.80	100
Reserve respiration capacity, %	242.93	291.35	468.73	>400
Cellular oxygen consumption profile				
Non-mitochondrial respiration as a share of total respiration, %	33.66	32.09	35.32	<10
Proton leak as a share of total respiration, %	3.76	10.45	4.96	
Share of respiration for mitochondrial ATP generation, %	62.58	57.46	59.72	>90
ATP turnover rate (mitochondrial oxygen utilisation)				
ATP base turnover, %	27.35	21.62	16.30	<20
ATP reserve, %	72.65	78.38	83.70	>80
Maximum possible oxygen consumption rate, pmol oxygen/min	90.78	123.10	180.06	>300
Cellular energy phenotype				
At rest	Resting	Resting	Resting	Resting
On energy demand	Energetic	aerobic	aerobic	Energetic/aerobic
Metabolic potential, % - Mitochondria	262.44	297.81	401.74	>350
Metabolic potential, % - glycolysis	312.43	252.29	334.84	>350
Oxygen consumption/glycolysis ratio on energy demand	Slight preference for anaerobic glycolysis	Slight preference for the mitochondria	Slight preference for the mitochondria	

Maximum possible oxygen consumption rate has doubled; many markers are showing improvement

Mitochondrial testing with AONM/MMD

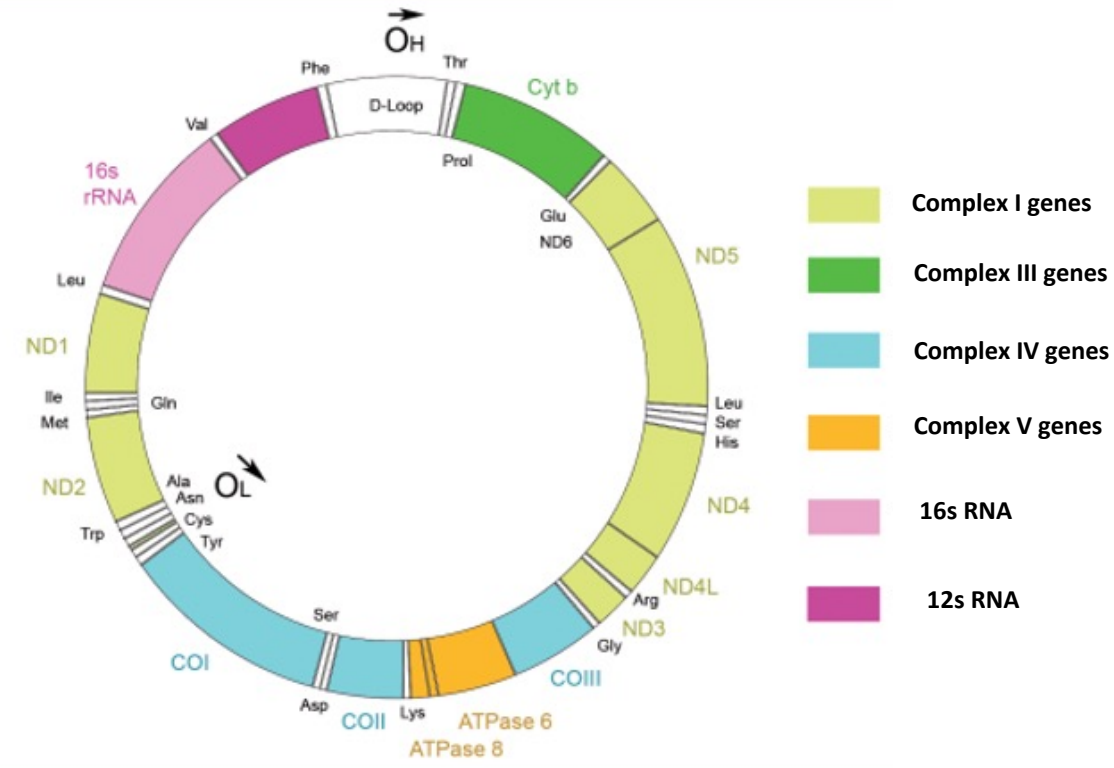
1. **ATP Profile:** Total ATP, Mitochondrial ATP, Glycolytic ATP, Reserve Capacity

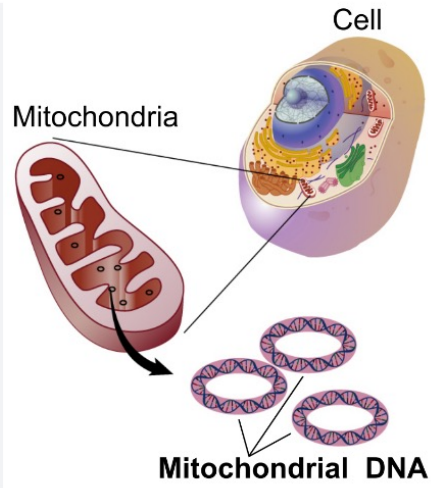
2. **Mitochondrial Health Index:**
   Basal respiration rate, mitochondrial ATP turnover, proton leak, maximum respiration rate, reserve capacity, non-mitochondrial rate, calculation of the overall Mitochondrial Health Index

3. **Supplementary biomarkers:**
   Ratio of mtDNA to nDNA (mtDNA:nDNA)
   PGC-1α
   Nrf-2
   Mitochondrial 4977 deletion mutant (mt4977del)
   Lactate/pyruvate ratio

Mitochondria have their own DNA

mtDNA:nDNA





Source: MMD GmbH & Co KG Author Prof. Dr. Brigitte König; Hoffmann A, Spengler D. The Mitochondrion as Potential Interface in Early-Life Stress Brain Programming. Front Behav Neurosci. 2018 Dec 6;12:306; https://en.wikipedia.org/wiki/Mitochondrial_DNA: Images free to use under Commons License

Ratio of mitochondrial DNA to nuclear DNA shows the mitochondrial mass in the cell

mtDNA:nDNA

DNA tests:

Ratio of mitochondrial DNA to nuclear DNA

Example 1:

Orthomolecular and mitochondrial medicine		
DNA		
Test	Result	Reference range
ratio mtDNA:nDNA	202	
Number of mitochondrial DNA copies per 1 copy of nuclear DNA		

Reference range ratio mtDNA:nDNA						
0 - 100	100 - 150	150 - 200	200 - 250	250 - 300	300 - 500	>500

The ratio of mitochondrial DNA to nuclear DNA is normal, though towards the lower end of the reference range.
Nuclear DNA remains stable at a unit of 1, but mitochondrial DNA will increase proportionally to the number of mitochondria in the cell.
It is important to note though that this does not mean that the mitochondria being detected are healthy/intact.

mtDNA:nDNA – numbers pathologically high/low

mtDNA:nDNA

Example 2:

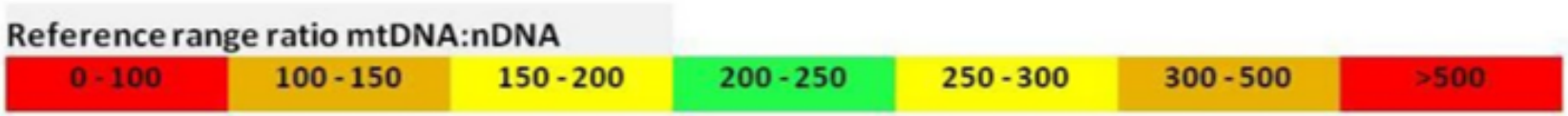
Ratio mtDNA:nDNA 1039

Number of mitochondrial DNA copies per 1 copy of nuclear DNA

Example 3:

ratio mtDNA:nDNA 115

Number of mitochondrial DNA copies per 1 copy of nuclear DNA



Too high (see example 2):
The cell is trying to counteract the lack of energy by increasing the number of mitochondria.

Too low (see example 3):
The cell is unable to counteract the lack of energy by increasing the number of mitochondria.

PGC-1-alpha is central for the induction of new mitochondria

PGC-1-alpha

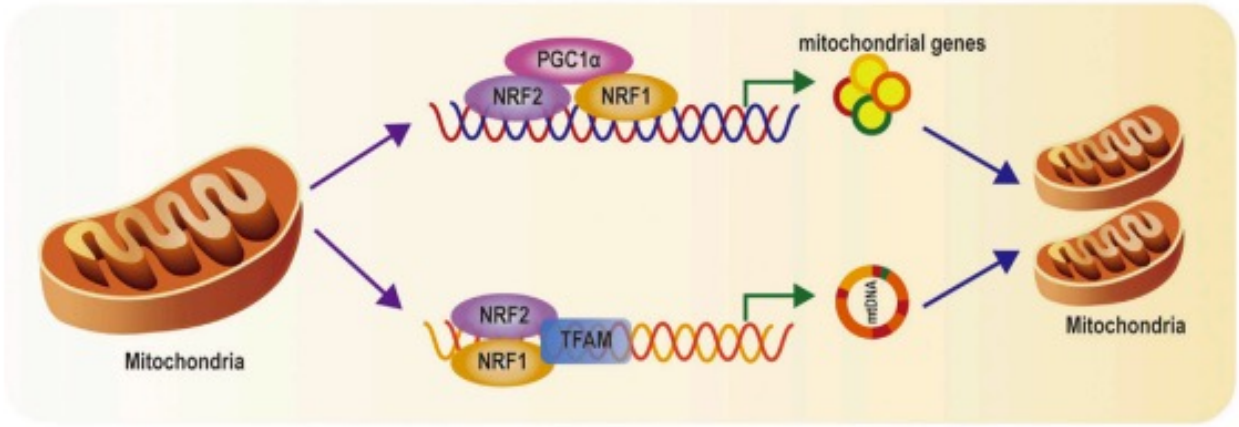


FIGURE 2 | Mitochondrial biogenesis pathways: When PGC-1α is activated, PGC-1a activates NRF1 and NRF2, and subsequently TFAM, which regulate genes involved in subunits of mitochondrial respiratory chain complexes, import of nuclear-encoded mitochondrial proteins, and mtDNA replication and transcription.

1

- PGC-1α regulates mitochondrial biogenesis but also has effects on mitochondrial functions beyond biogenesis.
- Mitochondrial quality control mechanisms, including fission, fusion, and mitophagy, are regulated by PGC-1α.
- PGC-1α-mediated regulation of mitochondrial quality may affect age-related mitochondrial dysfunction and insulin sensitivity.

2

Source: 1. Chen L, Qin Y, Liu B, Gao M, Li A, Li X, Gong G. PGC-1α-Mediated Mitochondrial Quality Control: Molecular Mechanisms and Implications for Heart Failure. Front Cell Dev Biol. 2022 May 27;10:871357; 2. Halling JF, Pilegaard H. PGC-1α-mediated regulation of mitochondrial function and physiological implications. Appl Physiol Nutr Metab. 2020 Sep;45(9):927-936.

The test for PGC-1-alpha measures its relative expression

PGC-1-alpha

RNA profile

Test	Unit	Result
PGC-1-alpha	Relative expression (to GAPDH)	0.000953

GAPDH: glyceraldehyde-3-phosphate dehydrogenase

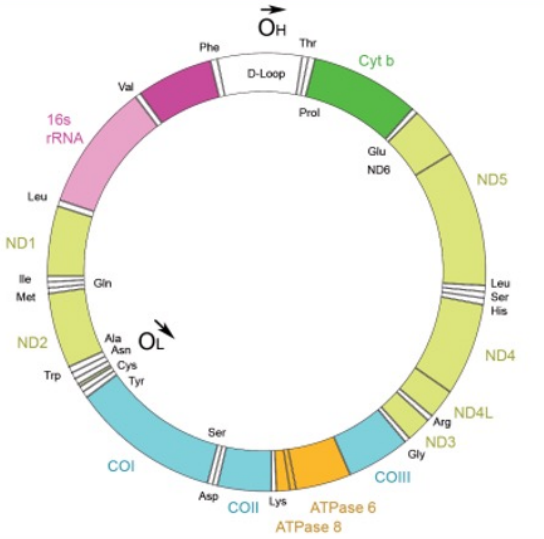
Interpretation: "Basic values of the peripheral blood leucocytes"

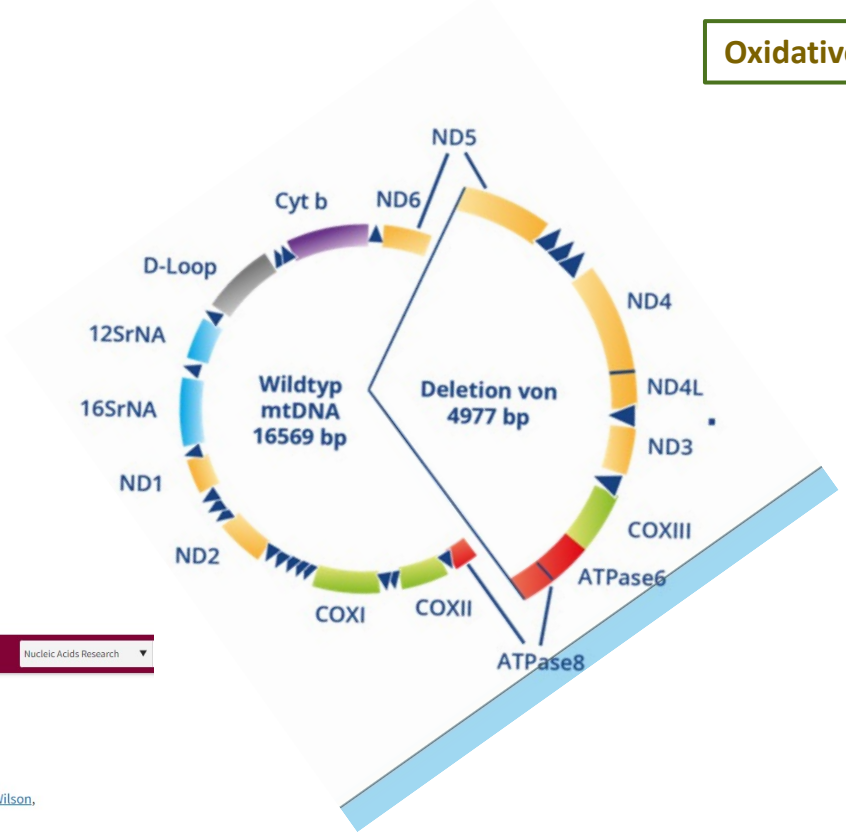
PGC-1-alpha expression is barely detectable. This indicates extremely low/absent new mitochondrial formation.

If this is the case, and mtDNA:nDNA is low too, then initiatives should be taken to increase PGC-1-alpha *(list of inducers available)*

The “common deletion” mDNA^{4977} is caused by oxidative stress

Oxidative stress





Nucleic Acids Research

Issues | Section browse | Advance articles | Submit | Purchase | About | Nucleic Acids Research

Volume 37, Issue 8
1 May 2009

Article Contents

Abstract
INTRODUCTION
MATERIALS AND METHODS
RESULTS
DISCUSSION
FUNDING
ACKNOWLEDGEMENTS
REFERENCES

JOURNAL ARTICLE

Oxidative stress induces degradation of mitochondrial DNA

Inna Shokolenko, Natalia Venediktova, Alexandra Bochkareva, Glenn L. Wilson, Mikhail F. Alexeyev

Nucleic Acids Research, Volume 37, Issue 8, 1 May 2009, Pages 2539–2548,
https://doi.org/10.1093/nar/gkp100
Published: 05 March 2009 **Article history**

PDF | Split View | Cite | Permissions | Share

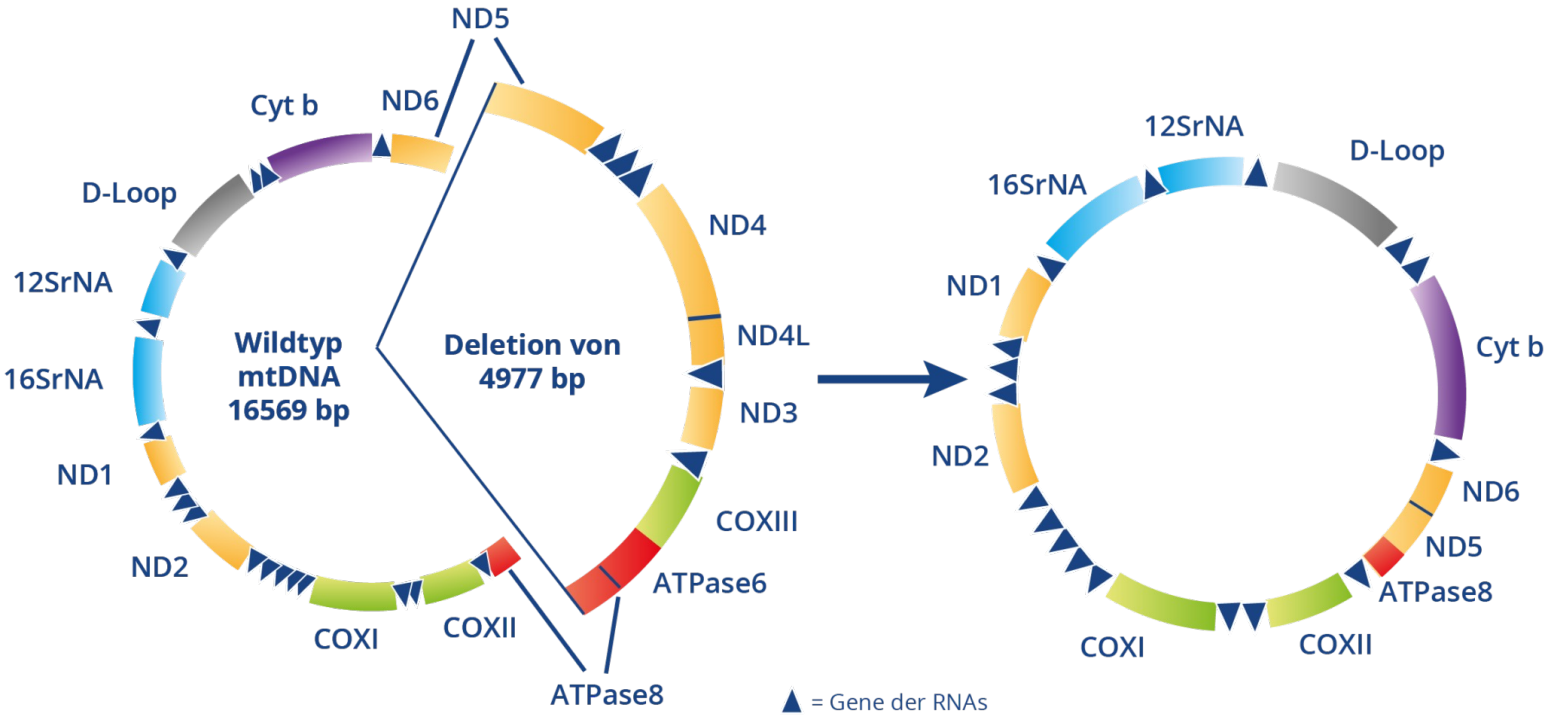
Abstract

Mitochondrial DNA (mtDNA) is located in close proximity of the respiratory chains, which are the main cellular source of reactive oxygen species (ROS). ROS can induce oxidative base lesions in mtDNA and are believed to be an important cause of the mtDNA mutations, which accumulate with aging and in diseased states. However, recent studies indicate that cumulative levels of base substitutions in mtDNA can be very low even in old individuals. Considering the reduced complement of DNA repair pathways available in mitochondria and higher susceptibility of mtDNA to oxidative damage than nDNA, it is presently

Source: MMD GmbH & Co KG Author Prof. Dr. Brigitte König

This can be measured, and shows the degree of oxidative stress the mitochondria are suffering …

Oxidative stress



Before deletion
Wildtype mtDNA = 16569 base pairs

After deletion
mtDNA = 11562 base pairs

Source: MMD GmbH & Co KG Author Prof. Dr. Brigitte König

… as well as any damage to mitochondrial DNA

Oxidative stress

Deletion mutant 4977

Example 1:

Mitochondrial 4977 Deletion mutant (mt4977del)

1.03E+06

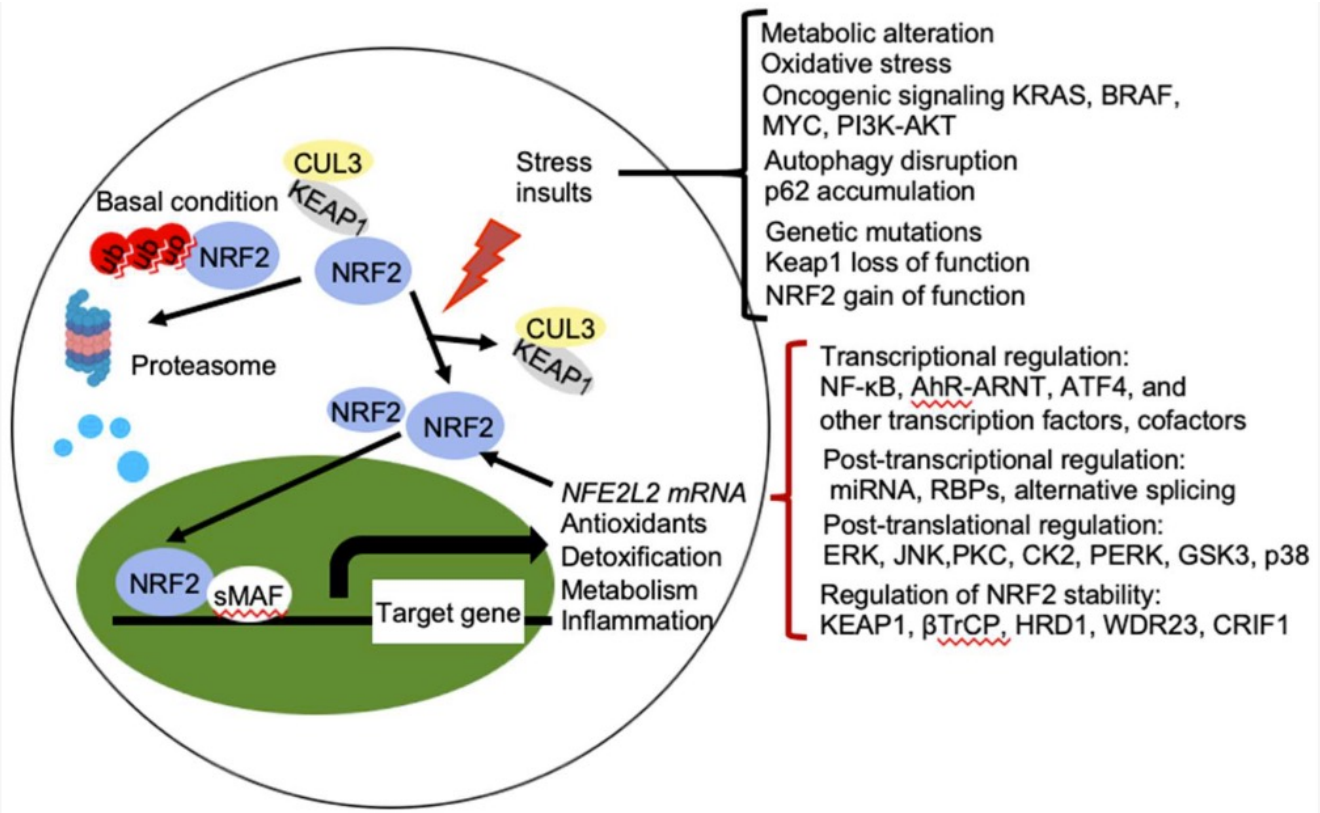
Number of copies of non-mutated mtDNA to 1 copy mt4977del

The mitochondrial deletion mutant mt4977bp is noticeably enhanced. This indicates oxidative stress and damage to mitochondrial DNA.

Among mtDNA deletions, one of the most vital that causes huge destruction of almost one third in length of the mitochondrial genome is the 4977-bp mtDNA deletion ($mDNA^{4977}$). This is one of the best-described large-scale mtDNA deletions, and has been found to accumulate in numerous disorders (literature available upon request). It is often known as a “common deletion” due to the frequency with which it has been reported. The deleted region encodes seven polypeptides essential for the OXPHOS pathway: four for Complex I, one for Complex IV, and two for Complex V. **This can cause complete failure of ATP production in the mitochondria affected.**

One initiative is to check Nrf-2: our cells' master antioxidant regulator

Nrf-2



"Nuclear factor-erythroid factor 2-related factor 2 (Nrf2) is a critical transcription factor that regulates the expression of over 1000 genes in the cell under normal and stressed conditions. **Nrf2 has been historically considered as a crucial regulator of antioxidant defense to protect against various insult-induced organ damage"**

Source: Zang H, Mathew RO, Cui T. The Dark Side of Nrf2 in the Heart. Front Physiol. 2020 Jul 9;11:722.

Problem if it is undetectable and you have evident oxidative stress

Nrf-2

RESULTS

Sample type: Blood in CPDA vials

Requisition:
RNA

Summary

RNA profile

Test	Unit	Result
Nrf-2	Relative expression (to GAPDH)	Not detectable

GAPDH: glyceraldehyde-3-phosphate dehydrogenase

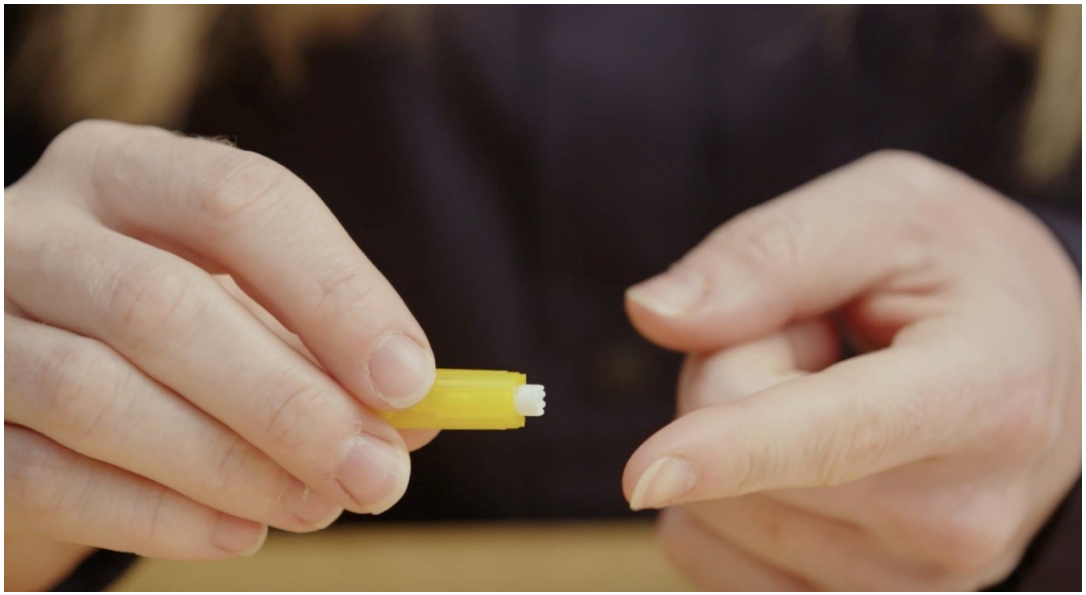
Interpretation: "Basic values of the peripheral blood leucocytes"

Nrf-2 expression is not detectable, indicating extremely low/absent defence against reactive oxygen metabolites in the cell.

Nrf-2

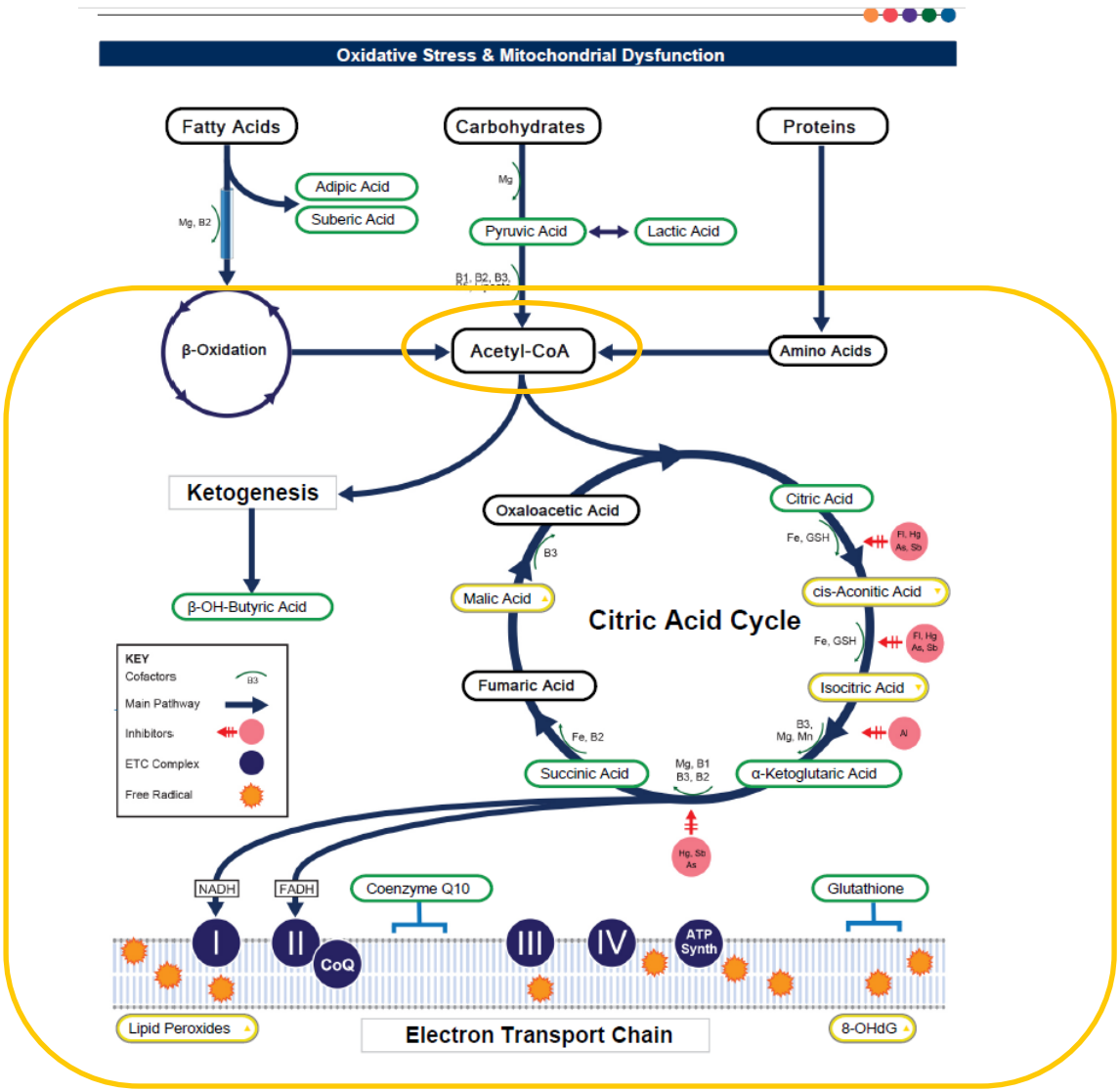
NRF-2, nuclear factor erythroid 2-related factor 2, is the master regulator of our antioxidant system to protect cells from reactive oxygen species. Nrf-2 activates Phase II detoxification – particularly glutathione-S-transferase and other antioxidant enzymes, including SOD-2, catalase and glutathione peroxidase. It is crucial to have adequate levels of this in the mitochondria.

This test of mitochondrial oxidation levels can also be done as a fingerprick test



Simple, can be done as a follow-up, or to check on your physical workup regime: are you over-training?

Pyruvate is the pathway into the mitochondria, as you will remember ....



Source: ©Genova Diagnostics ▪ A.L. Peace-Brewer, PhD, D(ABMLI), Lab Director ▪ CLIA Lic. #34D0655571 ▪ Medicare Lic #34-8475 (with permission)

Lactate/Pyruvate Index: shows what nutrients are being used as fuel for the mitochondria

Lactate/Pyruvate Plus

The higher the value of lactate compared to pyruvate, the more glycolysis is occurring. A higher level of pyruvate compared to lactate is a prerequisite for successful transfer of substrates in the mitochondria for oxidative phosphorylation.

The normal range for immune cells usually ranges from 1.0 – 0.7. Examples are calculated below

Ratio	Basal metabolic rate
>2.0	The cell is primarily using carbohydrates and preferentially converting them to lactate.
>1.2 – 2.0	The cell is primarily using carbohydrates and partially converting them to lactate.
1 – 1.2	The cell is primarily using carbohydrates and transporting them into the mitochondria.
0.8 – 1.0	The cell is using carbohydrates, fatty acids and amino acids. The carbohydrates are primarily being transported into the mitochondria.
<0.8	The cell is primarily using fatty acids as fuel.

Source: https://www.agilent.com/cs/library/usermanuals/public/103344-400.pdf; Prof. Dr. rer. nat. Brigitte König, MMD Labor; *mitochondrial research by Martin D. Brand and others*

Lactate/Pyruvate Index: shows what macronutrients are being used as fuel for the mitochondria

Lactate/Pyruvate Plus

Cell type:
Peripheral blood mononuclear cells (PBMC)

Lactate/Pyruvate ratio PLUS

Test	Result	Interpretation
Lactate/Pyruvate in dormant cells	1.61	Your immune cells are primarily metabolising carbohydrates and partially (30%) converting them to lactate
Lactate/Pyruvate in activated cells	2.43	The cells are primarily using carbohydrates and converting around 80% of them to lactate

This result:
Under pressure, the fuel is largely not going into the mitochondria, it is being recycled into lactate. The buildup can be very painful (fibromyalgia-type symptoms).

The normal range for immune cells usually ranges from 1.0 – 0.7. Examples are calculated below

Ratio	Basal metabolic rate
>2.0	The cell is primarily using carbohydrates and preferentially converting them to lactate.
>1.2 – 2.0	The cell is primarily using carbohydrates and partially converting them to lactate.
1 – 1.2	The cell is primarily using carbohydrates and transporting them into the mitochondria.
0.8 – 1.0	The cell is using carbohydrates, fatty acids and amino acids. The carbohydrates are primarily being transported into the mitochondria.
<0.8	The cell is primarily using fatty acids as fuel.

MITOCHONDRIAL TESTS

ACADEMY OF NUTRITIONAL MEDICINE

PATIENT INFORMATION		BARCODE (Lab use only)		Please send results to: ☐ myself ☐ my practitioner	
Patient FIRST NAME*:				**ORDERING DR/PRACTITIONER INFORMATION**	
Patient SURNAME*:				Dr. / Practitioner name:	
DATE OF BIRTH (DD/MM/YYYY)*:				Clinic:	
Sex assigned at birth* (please circle): male female		Time of Blood Draw*:		Street Address:	
Street Address:		Date of blood draw (DD/MM)*:		Postcode:	City:
Postcode:	City:	Material/Quantity	☐ CPDA	County:	Country:
County:	Country:			Tel no:	
Tel no:		**AONM HELPLINE:**		Email:	
Email*:		**+44 (0) 3331 210 305**			

☑	#TEST NUMBER	NAME OF TEST		MATERIAL	PRICE
☐	M1	ATP Profile:	Total ATP, Mitochondrial ATP, Glycolytic ATP, Reserve ATP	CPDA x1	£125
☐	M2	Mitochondrial Health Index (MHI):	Basal respiration rate, mitochondrial ATP turnover, proton leak, maximum respiration rate, reserve capacity, non-mitochondrial rate, calculation of the overall Mitochondrial Health Index	CPDA x1	£195
☐	M3 (M1+M2)	Combination of ATP Profile and MHI (M1 & M2)		CPDA x2	£285

SUPPLEMENTARY BIOMARKERS ON REQUEST (can normally only be done along with the ATP Profile and/or MHI)

☐	M4	Ratio of mtDNA to nDNA	1 additional CPDA (max. 2)	£70
☐	M5	PGC-1α	1 additional CPDA (max. 2)	£50
☐	M6	Nrf-2	1 additional CPDA (max. 2)	£50
☐	M7 (M4+M5+M6)	Combination of Ratio of mtDNA to nDNA, PGC-1α, and Nrf-2 (M4, M5, M6)	1 additional CPDA (max. 2)	£135
☐	M8	Lactate/pyruvate ratio (must be ordered at same time as MHI)	1 additional CPDA (max. 2)	£70
☐	M9	Mitochondrial 4977 deletion mutant (mt4977del)	1 additional CPDA (max. 2)	£70
☐	M10 (M3+M7+M8+M9)	Combination of all above (M3, M7, M8, M9)	CPDA x2	£485
☐	M11	Intact vs. non-intact mitochondria (must be ordered at same time as MHI + M4 + M9)	CPDA x2	£25
☐	M12	Mitochondrial Fuel Pathways (must be ordered at same time as MHI + M4 + M9)	CPDA x2	£195

Add £50 for courier delivery (to send from UK). Please Request shipping prices from elsewhere.

Tests plus courier. Total:

Tests of telomere length indicate a patient's biological (rather than chronological) age

Patient	
Geburtsdatum	28.11.1967
Probennahme	05.03.2024
Probeneingang	06.03.2024
Untersuchungsende	02.04.2024
Endbefund	**02.04.2024**
Validiert durch	Prof. Dr. Brigitte König
Ärztliche Leitung	Prof. Dr. Gerhard Jorch

MMD GmbH & Co. KG | Breiter Weg 10 a | 39104 Magdeburg

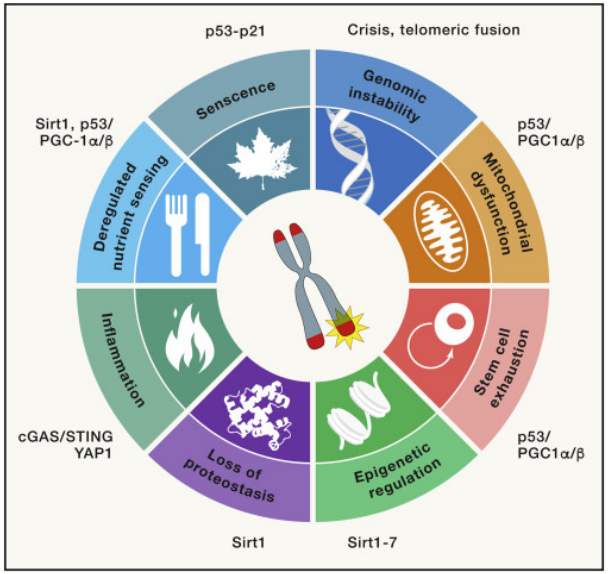
[Clinic]

RESULTS

Sample type: Whole blood	Test requested: Telomere length

Relative telomere length (telomere to single copy gene ratio, T/S)	0.724
Absolute telomere length	7.23 kb
Chronological age	57
Age calculated by telomere length, i.e. biological age	33

Test results/interpretation: The calculated absolute telomere length (7.23 kilobases) corresponds to an age of between 30-35 years. The result means that the telomeres are longer than for 50% of people in the respective age group. This is a very encouraging result.



Relevance of telomere dysfunction for features of cellular aging

Source: https://doi.org/10.1016/j.cell.2020.12.028

Fingerprick tests also available

MMD

AONM ACADEMY of NUTRITIONAL MEDICINE

MITOCHONDRIAL TESTS USA 3

CAPILLARY BLOOD DRAW (FINGERPRICK)

PATIENT INFORMATION		BARCODE (Lab use only)	Please send results to: ☐ myself ☐ my practitioner
Patient FIRST NAME*:			ORDERING DR/PRACTITIONER INFORMATION
Patient SURNAME*:			Dr. / Practitioner name:
DATE OF BIRTH (DD/MM/YYYY)*:			Clinic:
Sex* (please circle): male female		Date of Fingerprick (DD/MM)*:	Street Address:
Street Address:			
ZIP:	City:	Please fill 2x rings for each test, up to a total of 9 rings for ALL tests.	ZIP: City:
County:	Country:		County: Country:
Tel no:		AONM HELPLINE: +44 (0) 3331 210 305	Tel no:
Email*:			Email:

☑	#Test number	Name of test	Price (individual tests)	Price (ALL)
☐	MFP1	Oxidative stress measured using the mt4977 deletion mutant	£80	
☐	M6	Nrf-2 (Master antioxidant regulator)	£50	
☐	M15	8-OH-dG-DNA (a predominant ROS lesion)	£58	£352
☐	MFP8	Telomere length	£149	
☐	MFP7	Fingerprick Test Kit (incl. delivery to USA by Royal Mail)	£15	

Courier (shipping) costs to be determined. Tests plus courier. Total: _____

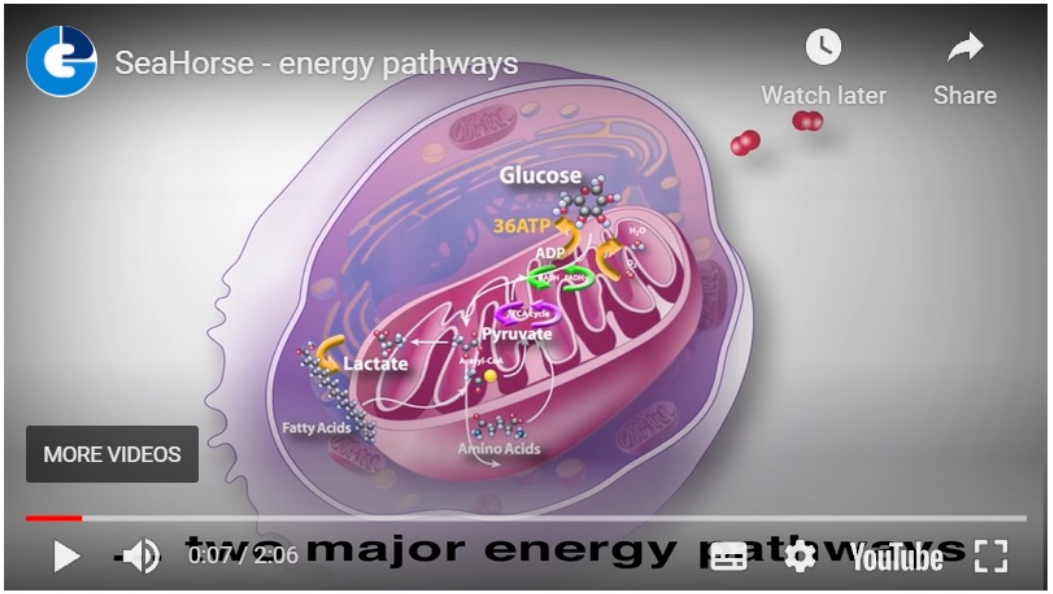
Many videos about the Seahorse technology available, and over 8,000 studies* for which the Seahorse has been used

HOW THE SEAHORSE XF WORKS

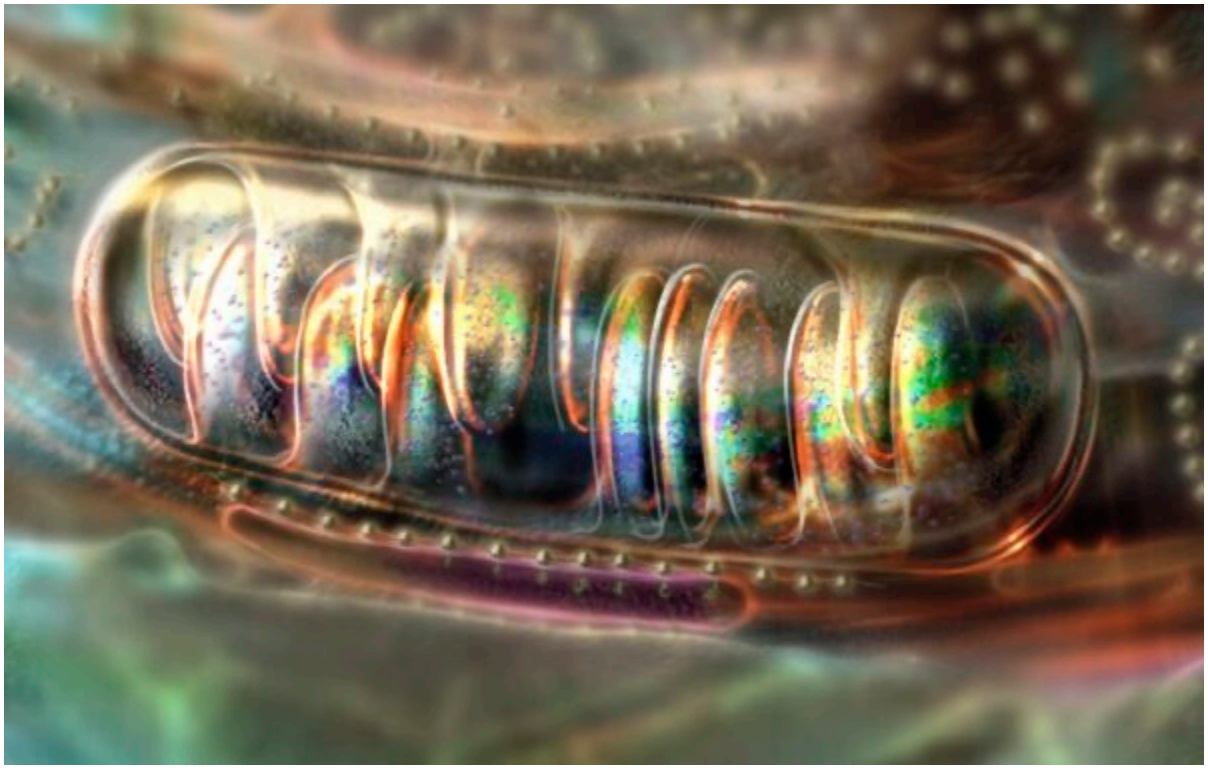
Videos are at the bottom of this page:
https://aonm.org/mitochondrial-testing/

The tests require only one vial of blood in a CPDA[i] tube. The laboratory uses

SEAHORSE: ENERGY PATHWAYS



* https://www.agilent.com/search/?N=4294836537



Thanks very much for your attention!

info@aonm.org, 0333 121 0305

gilian@aonm.org, 0786 772 6387