Patient	Xxxx
Date of birth	Xxxx
Sample taken	21.03.2018
Receipt of sample	26.03.2018
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Final result	
Validated by	Prof. Dr. Brigitte König

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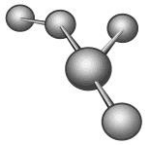
MMD



AONM
ACADEMY OF NUTRITIONAL MEDICINE

Xxxxx Clinic
Xxxxx
Xxxxx

RESULTS

Sample type: Blood in CPDA vials

Requisition:
Mitochondrial Health Index / PBMCs

Summary

	Patient's value	Target value (optimal)
Mitochondrial Health Index (MHI)	0.77	**>2.5**
Mitochondrial Bioenergetics		
Coupling efficiency, %	100.00	**100**
Reserve respiration capacity, %	324.52	**>400**
Cellular oxygen consumption profile		
Non-mitochondrial respiration as a share of total respiration, %	71.00	**<10**
Proton leak as a share of total respiration, %	0.00	
Share of respiration used for mitochondrial ATP generation, %	29.00	**>90**
ATP turnover rate (mitochondrial oxygen utilisation)		
ATP base turnover, %	26.00	**<20**
ATP reserve, %	74.00	**>80**
Potential maximum oxygen consumption rate in pmol oxygen/min	22.99	**>300**
Cellular energy phenotype		
At rest	Resting/glycolytic	**Resting**
On energy demand	energetic	**Energetic**
Metabolic potential, mitochondrial percentage	189.87	**>350**

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Metabolic potential, glycolysis percentage	135.97	**>350**
Oxygen consumption/glycolysis on energy demand	Moderate mitochondrial preference	

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

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Interpretation

	None	Slight	Moderate	Considerable	Extreme
Mitochondrial dysfunction					✓
Cellular imbalance					✓

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

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).

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DETAILED RESULTS

MITOCHONDRIAL HEALTH INDEX (MHI)

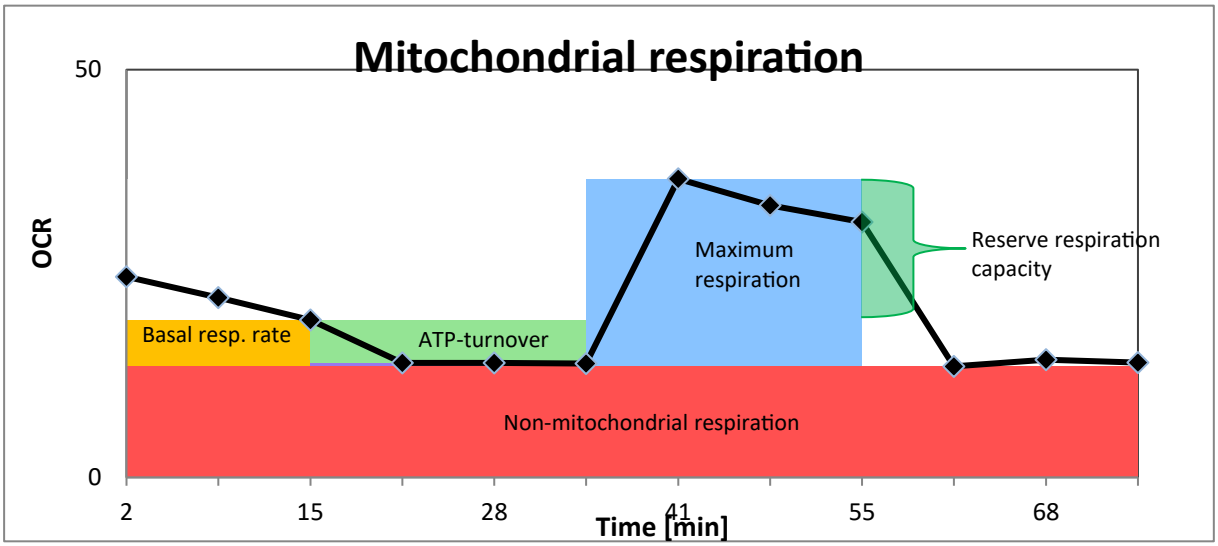
The MHI is a sensitive indicator of the reaction of immune cells (PBMCs) to oxidative stress, and for the changing metabolic programmes that they serve depending on the role they need to play in the case of inflammation, immune defence and immune health. The MHI is is also an indicator for the current "health" of the cell. It is interactively composed the following parameters.

YOUR RESULTS

Mitochondrial Health Index (MHI)	Extremely low

Parameter	Evaluation	Reference values	Results
Mitochondrial Health Index (MHI)	Optimal	>2.5	
	Slightly low	2.0-2.5	
	Moderately low	1.5-2.0	
	Considerably low	1.0-1.5	
	Extremely low	<1.0	**0.77**

YOUR RESULTS PROFILE



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MITOCHONDRIAL BIOENERGETICS

Coupling efficiency, %	**Optimal**
Reserve respiration capacity, %	**Slightly low**

COUPLING EFFICIENCY

Coupling efficiency is a metric for the transformation of oxygen into the energy currency ATP. The cause of reduced coupling efficiency is a proton leak. A proton leak accounts for any oxygen in the mitochondria that is not being used for ATP synthesis. (see also p. 6, oxygen consumption profile).

YOUR RESULTS

Parameter	Evaluation	Reference values in %	Result in %
Coupling efficiency, %	**Optimal**	99-100	**100.00**
	Slightly low	95-99	
	Moderately low	90-95	
	Considerably low	70-90	
	Extremely low	<70	

Interpretation of your results:

The coupling efficiency is optimal.

RESERVE RESPIRATION CAPACITY

Reserve respiration capacity shows the extent to which the existing mitochondria can use further oxygen for generating energy. Low reserve respiration capacity can be due to a) insufficient utilisation of fuels (glucose, fatty acids); b) high resting metabolism due to ROS and RNS; c) non-intact complexes of the electron transport chain; d) altering metabolic status due for example to the immune cells adapting their role as a result of infection (viral, bacterial), anti-tumour immune responses, autoimmune disease, etc.

YOUR RESULTS

Parameter	Evaluation	Reference values, %	Result, %
Reserve respiration capacity, %	Optimal	>400	
	Slightly low	300-400	**324.52**
	Moderately low	250-300	
	Considerably low	200-250	
	Extremely low	<200	

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Interpretation of your result:

The reserve capacity of the existing mitochondria is optimal. Looking at your overall results, consider the following drivers: a) insufficient use of fuels (glucose, fatty acids); b) high resting metabolism due to ROS and RNS; c) insufficient provision of the immune cells with necessary minerals, vitamins, etc.; d) defects in the electron transport chain.

Further diagnostic options

Investigate the mitochondrial mutations that influence ATP generation (e.g., common deletion mt4977bp; full sequencing).

Investigate the mitochondrial utilisation of the fuels fatty acids and glucose.

Investigate oxidised lipids, proteins, nuclear and mitochondrial DNA in the immune cells to assess the damage that has already occurred, and for targeted use of antioxidants (see the section “OXYGEN CONSUMPTION PROFILE“).

Investigate the intracellular reactive oxygen metabolites for the targeted use of antioxidants and/or therapeutics (see the section “OXYGEN CONSUMPTION PROFILE“).

CELLULAR OXYGEN CONSUMPTION PROFILE

The individual parameters of the oxygen consumption profile of the (resting) immune cells give an overview of the different ways in which the oxygen being delivered to the cells is being consumed. The oxygen should be being used largely for the generation of mitochondrial ATP by the immune cells being analysed (peripheral mononuclear blood cells). A proton leak accounts for any oxygen in the mitochondria that is not being used for ATP synthesis. The causes of a proton leak are for example a) high concentration of damaging free radicals; b) a lack of redox equivalents; c) inhibitors of ATPases, including ATP synthase; d) a fatty acid composition of the mitochondria that is suboptimal.

The oxygen consumption outside the mitochondria consists of at least two components: a) oxygen consumption at the surface of the cell; b) basal oxygen consumption for prooxidative processes (e.g., flavoenzymes), and the maintenance of various membrane pumps.

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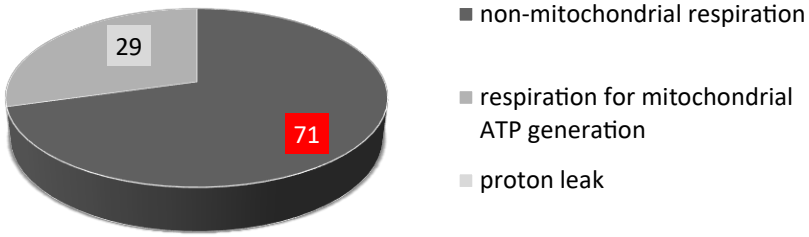
YOUR RESULTS

Optimal
Slightly high
Moderately high
Very high
Extremely high

How the oxygen is being consumed, %



Parameter	Evaluation	Reference values, %	Result, %
Non-mitochondrial respiration as a share of total respiration, %	Optimal	0-10	
	Slightly high	10-20	
	Moderately high	20-30	
	Very high	30-50	
	Extremely high	>50	**70.59**

Interpretation of your results

Your immune cells are using only 29% of the oxygen directly for generating mitochondrial energy. 71% of the oxygen is being used for non-mitochondrial processes. The non-mitochondrial oxygen consumption, independent of whether it is used for respiration at the surface of the cell and/or prooxidative processes, is having a negative effect on the MHI (see the MHI). No proton leak is detectable.

Recommendation

Investigate oxidised lipids, proteins, nuclear and mitochondrial DNA in the immune cells to assess the damage that has already occurred, and for targeted use of antioxidants.

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Investigate the intracellular reactive oxygen metabolites for the targeted use of antioxidants and/or therapeutics.

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ATP TURNOVER (MITOCHONDRIAL OXYGEN CONSUMPTION)

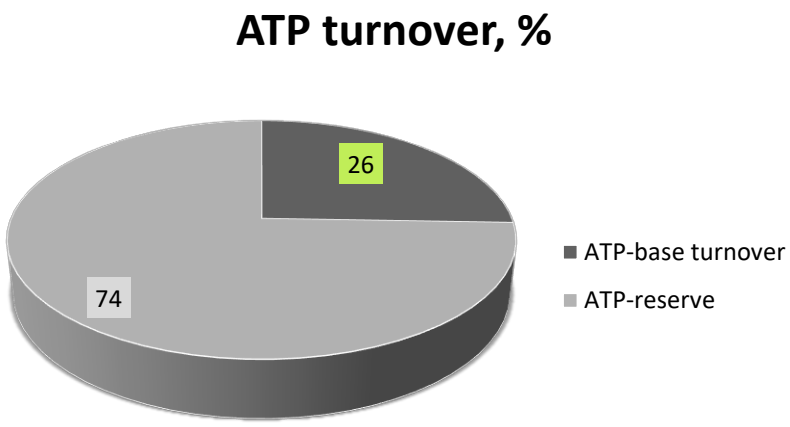
This parameter shows how much of the total possible ATP turnover of the mitochondrion is already being used at rest (in %).

Causes of a high pro rata ATP base turnover compared to the total potential ATP turnover are – in addition to oxidative processes – specific inhibitors of the individual complexes of the electron transport chain (such as heavy metals), toxins such as pesticides or lipopolysaccharides, and/or oxygen radicals.

Causes of a limited ATP reserve are a) insufficient utilisation of fuels (glucose, fatty acids); b) reduced mitochondrial mass (number of mitochondria); c) non-intact complexes of the electron transport chain; d) altering metabolic status due for example to repurposing of the immune cells as a result of infection (viral, bacterial), anti-tumour immune responses, autoimmune disease, etc.

YOUR RESULT

Optimal
Slightly high
Moderately high
Very high
Extremely high



Parameter	Evaluation	Reference values	Result
ATP consumption, base turnover, %	Optimal	0-20	
	Slightly high	21-25	**25.55**
	Moderately high	26-35	
	Considerably high	36-45	
	Extremely high	>45	

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Parameter	**Evaluation**	**Reference values, pmol/min**	**Result, pmol/min**
Maximum possible oxygen consumption rate, pmol oxygen/min	Optimal	>500	
	Slightly low	300-500	
	Moderately low	200-300	
	Very low	100-200	
	Extremely low	<100	**22.99**

Interpretation of your result:

Your immune cells are using 25.6 % of their possible oxygen consumption capacity for their base energy balance. This value is slightly high. This indicates a load on the immune cells that is disrupting cell regulation.

The maximum useable oxygen volume (in pmol oxygen/min) that can be converted into energy (ATP) by the mitochondria is 22.99 pmol/min. This potential oxygen consumption rate is, from an absolute perspective, considered to be extremely low On enery demand, after subtraction of the basal cellular oxygen consumption (5.88 pmol/min) noch 17.32 pmol oxygen/min remaining for mitochondrial ATP generation. This means the absolute potential ATP turnover rate is extremely low.

Against the backdrop of the other results, several factors may be responsible for the non-optimal absolute potential ATP turnover rate, either alone or in combination: a) insufficient mitochondrial mass: b) the limited utilisation of fatty acids and particularly of glucose; c) insufficient provision of the immune cells with the requisite minerals, vitamins, etc.; d) a defective electron transport chain.

Further diagnostic options

Investigate the mitochondrial mass (mtDNA:nDNA, i.e. number of mitochondria), and analyse the mitochondrial mutations that are influencing ATP generation (e.g., common deletion mt4977bp; full sequencing).

Investigate the mitochondrial use of fatty acids and glucose as fuels.

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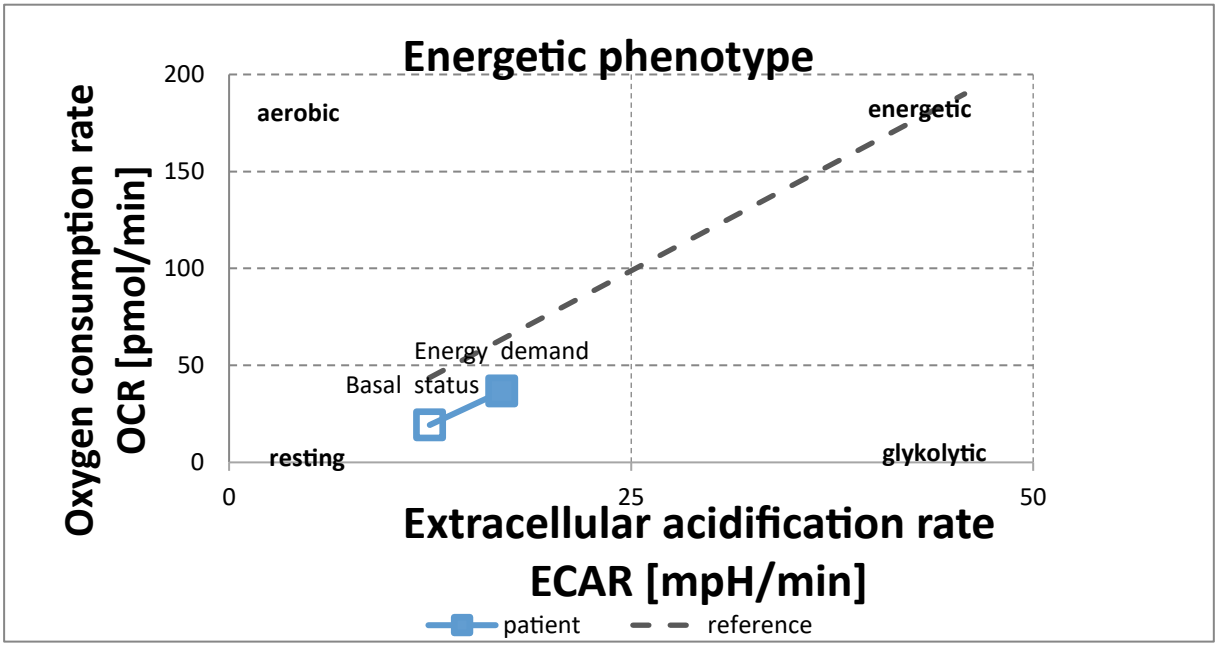
CELLULAR ENERGY PHENOTYPE

Our cells have two methods of energy generation available to them: mitochondrial respiration and anaerobic glycolysis. A person's energy phenotype is determined by which avenue of energy generation is primarily being used. A cellular energy phenotype comprises a phenotype at rest, a phenotype under stress, where it is experiencing energy demand, and the potential to use either mitochondrial respiration or anaerobic glycolysis when put under conditions of energy demand. The parameters of the cellular energy phenotype reveal the ability and/or the preferred avenue the cells use to cover their energy requirements. The parameters in the following are a sensitive early indicator of metabolic changes to the cells and their functions when immune reactions take place.

YOUR RESULTS

YOUR PROFILE



YOUR RESULTS PROFILE – CELLULAR ENERGY PHENOTYPE

At rest	**Resting/glycolytic**
On energy demand	**Glycolytic**
Metabolic potential, mitochondrial (%)	**Extremely low**
Metabolic potential, glycolysis (%)	**Extremely low**
Oxygen consumption/glycolysis ratio on energy demand	**Moderate mitochondrial preference**

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BIOENERGETISCHER PHÄNOTYP

Parameter	Reference values	Result
Bioenergetic phenotype at rest	Resting	**Resting/glycolytic**
Bioenergetic phenotype on energy demand	Energetic	**Glycolytic**

Parameter	Evaluation	Referenzwerte in %	Ergebnis in %
Metabolic potential, %	Optimal	>350	
-Mitochondria-	Slightly low	300-350	
	Moderately low	250-300	
	Considerably low	200-250	
	Extremely low	<200	**189.87**

Parameter	Evaluation	Reference values, %	Result, %
Metabolic potential, %	Optimal	>350	
-glycolysis	Slightly low	300-350	
	Moderately low	250-300	
	Considerably low	200-250	
	Extremely low	<200	**135.97**

Parameter	Evaluation	Reference values	Result
Oxygen consumption/glycolysis ratio on energy demand	Very strong preference for the mitochondria	>1.7	
	Strong preference for the mitochondria	1.5-1.7	
	Moderate preference for the mitochondria	1.3-1.5	**1.40**
	Slight preference for the mitochondria	1.1 – 1.3	
	Balanced	0,9-1,1	
	Slight preference for anaerobic glycolysis	0,8-0,9	
	Moderate preference for anaerobic glycolysis	0,7-0,8	
	Strong preference for anaerobic glycolysis	0,5-0,7	
	Very strong preference for anaerobic glycolysis	<0,5	

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Interpretation of the results of your cellular energy phenotype:

With immune cells one assumes that on energy demand they are able to use anaerobic glycolysis and the mitochondria in approximately equal proportions.

The base setting of your immune cells is glycolytic. This suggests the presence of acute inflammation/active chronic inflammation. From an overall perspective, the metabolic potential of the mitochondria is extremely low (189.87%).

Looking at all the results as a whole, several factors may be responsible for the reduced metabolic potential, either alone or in combination: a) limited utilisation of fatty acids and especially of glucose; b) insufficient mitochondrial cofactors; c) oxidative stress; d) dysfunctional complexes of the electron transport chain.

Further diagnostic options

Investigate mitochondrial utilisation of the fuels fatty acids and glucose.

Bestimmung von oxidierten Lipiden, proteins, nuclear and mitochondrial DNA in the immune cells to estimate the damage that has already taken place and for the targeted use of antioxidants (see the section “OXYGEN CONSUMPTION PROFILE“).

Investigate the intracellular reactive oxygen metabolites in order to target the use of antioxidants and/or therapeutics (see the section “OXYGEN CONSUMPTION PROFILE“).
Check the individal complexes of the electron transport chain for targeted individualised therapy.