

**Mitochondrien, Midichloria mitochondrii, Rickettsia**  
Gruppe [α-Proteobakterien](#), Ordnung [Rickettsiales](#), Gattung [Midichloria](#),  
group [α-proteobacteria](#), genus [Midichloria](#)

**Mitochondrien** <http://www.biokurs.de/skripten/bs11-57.htm>  
**Zellatmung** <http://www.biokurs.de/skripten/12/bs12-23.htm>

**A. Mitochondria dysfunktion** (The more the mitochondrial dysfunction the stronger the inflammation)

**B. Mitochondrial diseases** [http://en.wikipedia.org/wiki/Category:Mitochondrial\\_diseases](http://en.wikipedia.org/wiki/Category:Mitochondrial_diseases)  
Finsterer J. (2004) **Mitochondriopathies**. *Eur J Neurol*. 11(3), 163-86. <http://www.ncbi.nlm.nih.gov/pubmed/15009163>

**Mitochondrial diseases - Biochemistry**

"A special feature of mitochondria is their dual genetic control of mitochondrial DNA (mtDNA) and nuclear DNA (nDNA). 99% of all structural and functional proteins of the mitochondrion and the most proteins necessary for transcription, translation and replication of the mitochondrial genome are encoded by the nuclear DNA (nDNA). Only 1% of the proteins are encoded in the mitochondrial DNA (mtDNA)".

Source: [http://www.neuro.med.tu-dresden.de/mitolab/index.php?option=com\\_content&task=view&id=13&Itemid=30](http://www.neuro.med.tu-dresden.de/mitolab/index.php?option=com_content&task=view&id=13&Itemid=30)

**The mineralocorticoid aldosterone synthase enzymes and the 11 betahydroxylase are localized in mitochondria, as well as the cholesterol side-chain cleavage enzyme**

Source: <http://en.wikipedia.org/wiki/File:Steroidogenesis.svg>

**Diagnostics: TNF alpha, Interferon gamma inducible protein 10 (IP10), Histamin, IL6, IL8, lactate / pyruvate, methylmalonic acid, PH status, ketone bodies in urine or serum, intrazellulär ATP, Malondialdehyd-LDL, fasting blood glucose, homocysteine, arginine, vitamin D3**

**Drug therapy: CoQ10, L-Carnitine, Vitamin B1, B6, B12, Folic acid, Vitamin D, Vitamin E, Magnesium, Fatty Acids, Glutathione, N-Acetylcysteine, Selenium, Polyphenols, Curcumin, antichemokin, therapy <http://www.kabilahsystems.de/antizyt-chem.pdf>**

**A. Mitochondrien-Dysfunktion** (Je stärker die Mitochondrien-Dysfunktion desto stärker die Entzündung) Die **Bestimmung des intrazellulären ATP** als Marker einer mitochondrialen Dysfunktion. <http://www.daszahnzentrum.de/pdf/Intrazellulaerer%20ATP.pdf>

**B. Mitochondriopathie** <http://de.wikipedia.org/wiki/Mitochondriopathie>  
**Mitochondriale Myopathien** Licht- und ev. Elektronenmikroskopie, Messungen v. Enzymaktivitäten, genetische Untersuchungen. <http://www.dgm.org/muskelerkrankungen/mitochondriale-myopathien>

**Krankheitsbilder Mitochondriopathien – Biochemie**

„Eine Besonderheit der Mitochondrien ist ihre duale genetische Kontrolle durch mitochondriale DNA (mtDNA) und nukleäre DNA (nDNA). 99% aller strukturellen und funktionellen Proteine des Mitochondriums sowie die meisten für Transkription, Translation und Replikation des mitochondrialen Genoms erforderlichen Proteine werden durch die nukleäre DNA (nDNA) kodiert. Nur 1% der Proteine ist in der mitochondrialen DNA (mtDNA) verschlüsselt.“ Quelle:

[http://www.neuro.med.tu-dresden.de/mitolab/index.php?option=com\\_content&task=view&id=13&Itemid=30](http://www.neuro.med.tu-dresden.de/mitolab/index.php?option=com_content&task=view&id=13&Itemid=30)

**Die Mineralocorticoid-Enzyme Aldosteronsynthase und die 11 betahydroxylase sind in Mitochondrien lokalisiert, ebenso das Cholesterol side-chain cleavage enzyme**

Quelle: <http://en.wikipedia.org/wiki/File:Steroidogenesis.svg>

**Diagnostik: TNF alpha, Interferon gamma inducible protein 10 (IP10), Histamin, IL6, IL8, Laktat /Pyruvat, Methylmalonsäure, PH-Status, Ketonkörper im Urin oder Serum, intrazelluläres ATP, Malondialdehyd-LDL, Blutzucker, Homocystein, Arginin, Vitamin D3**

**Medikamententherapie: CoQ10, L-Carnitin, Vitamin B1, B6, B12, Folic acid, Vitamin D, Vitamin E, Magnesium, Fettsäuren, Glutathion, N-Acetylcystein, Selen, Polyphenole, Curcumin, Antizytokine, Therapie <http://www.kabilahsystems.de/antizyt-chem.pdf>**

“Die **Medizin des 21. Jahrhunderts** wird eine Mitochondrien – Filamenten – Mizellen \* – Beziehungs \* – Medizin sein oder sie wird nicht sein” (Huismans BD 2014).  
<http://www.xerlebnishaft.de/mitochondrien.pdf> -> Midichloria mitochondrii

"The **medicine of the 21st century** will be a medicine of mitochondria, filaments, micelles \* and relationships \*, or will not be viable" (Huismans BD 2014).  
<http://www.xerlebnishaft.de/mitochondrien.pdf> -> Midichloria mitochondrii

**Persistenz des Pathogens -> Chronische Entzündung -> Mitochondrien Dysfunktion**

Gefahren Modell



**Persistence of the pathogen -> Chronic Inflammation -> mitochondrial dysfunction**

Danger model



➔ **Atmung und Gärung im Krebsgeschehen. Respiration and fermentation management in cancer** <http://www.xerlebnishaft.de/krebsstammzelltherapie.pdf>

Karnkowska A, Vacek V, Zubáčová Z et al. (2016) **A eukaryote without a mitochondrial organelle**. Current Biology, DOI: <http://dx.doi.org/10.1016/j.cub.2016.03.053>

[http://www.cell.com/current-biology/abstract/S0960-9822\(16\)30263-9](http://www.cell.com/current-biology/abstract/S0960-9822(16)30263-9)

**“This is the first example of a eukaryote lacking any form of a mitochondrion, demonstrating that this organelle is not absolutely essential for the viability of a eukaryotic cell”.**

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“**RESULTS: In this population, we found that 9.2 in 100,000 people have clinically manifest mtDNA disease, making this one of the commonest inherited neuromuscular disorders. In addition, a further 16.5 in 100,000 children and adults younger than retirement age are at risk for development of mtDNA disease. INTERPRETATION: Through detailed pedigree analysis and active family tracing, we have been able to provide revised minimum prevalence figures for mtDNA disease. These estimates confirm that mtDNA disease is a common cause of chronic morbidity and is more prevalent than has been previously appreciated** ».
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**Boles RG, Zaki EA** (2009) **Diagnostic methods and kits for functional disorders**

US 20090269758 A1 <http://www.google.com/patents/US20090269758#classifications>

« **The present invention relates to methods for the diagnosis of functional disorders in humans. A method of the invention, in certain embodiments, comprises the detection of one or more polymorphisms in mitochondrial DNA of a human. The current invention further provides kits for use in a method of the invention.**»

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„**Anticonvulsants, Psychotropics, Cholesterol Medications, Analgesics and Anti-inflammatories, Antibiotics** Antibiotics, (specifically tetracycline, minocycline, chloramphenicol, and aminoglycosides), can be harmful to the mitochondria because they inhibit mtDNA translation and protein synthesis. They can cause hearing loss as well as cardiac and renal toxicity. - See more at: <http://www.mitoaction.org/blog/medication-exposures-mitochondrial-toxicity#sthash.Wqb0nicT.dpuf> , **Steroids, Anesthesia, Surgery, Environmental Agents Tobacco smoke (primary or secondary inhalation) and alcohol are both potentially toxic for patients with mitochondrial diseases. Other environmental factors may not be as controllable, but patients should be aware of their toxicity. These include rotenone (chemical used in insecticides and pesticides) and fat soluble chemicals with benzene rings such as hair dye and paint fumes. Ketogenic diet, Endogenous Stress Related Hormones, CoEnzyme Q10 can become an oxygen radical and cause trouble if the dosage is too high. The most common dosage is 10 - 20 mg/kg/day.**»

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« **The release of such mitochondrial 'enemies within' by cellular injury is a key link between trauma, inflammation and SIRS (systemic inflammatory response syndrome)** »

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„**Der medikationsorientierte Einsatz von mitotropen Mikro-nährstoffen wie Coenzym Q10 und L-Carnitin kann nicht nur das Risiko für unerwünschte Arzneimittelwirkungen verringern und die Lebensqualität der behandelten Patienten verbessern, sondern auch das pharmakologische, immunologische und metabolische Wirkprofil eines Arzneimittels erweitern. Darüber hinaus beinhaltet eine auf die Medikation ausgerichtete Supplementierung von Vitaminen und anderen Mikronährstoffen ein hohes Potenzial, Arznei- und Therapiekosten im Gesundheitssystem einzusparen.**“

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## Midichloria mitochondrii

**Midichloria Bakterien** parasitieren Mitochondrien. Sie verwenden sie als Energiequelle [sie fressen ATP] und sie verwenden sie als Vermehrungsmedium.

**Midichloria bacteria** seem to consume the mitochondria they parasite, possibly using them as a source of energy and/or molecules to multiply. Quelle, source : <http://en.wikipedia.org/wiki/Midichloria>

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«... **To our knowledge, this is the only known example of a bacterium that is able to enter the mitochondria of animals. Our recently published electron microscopic data indicates that the bacterium enters mitochondria between the inner and outer membranes, and then proceeds to consume the inner mitochondrial matrix. ...**»



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“**Most strikingly, pre-mitochondrion was predicted to possess a plastid/parasite type of ATP/ADP translocase that imports ATP from the host, which posits pre-mitochondrion as an energy parasite that directly contrasts with the current role of mitochondria as the cell’s energy producer. In addition, pre-mitochondrion was predicted to encode a large number of flagellar genes and several cytochrome oxidases functioning under low oxygen level, strongly supporting the previous finding that the mitochondrial ancestor was likely motile and capable of oxidative phosphorylation under microoxic condition.**”

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## Rickettsien, rickettsia

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