

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
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

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

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”.

Arts WF, Scholte HR, Bogaard JM et al. (1983) NADH-CoQ reductase deficient myopathy: **successful treatment with riboflavin**. Lancet 2, 581–582

Bresolin N, Doriguzzi C, Ponzetto C et al. (1990) **Ubidecarenone** in the treatment of mitochondrial myopathies: a multi-center double-blind trial. J Neurol Sci 100, 70–78

Goto Y, Nonaka I, Horai A. (1990) A mutation in tRNA^{Leu(UUR)} gene associated with the MELAS subgroup of mitochondrial encephalomyopathies. Nature 348, 651–653

Stanley CA, De Leeuw S, Coates PM et al. (1991) Chronic cardiomyopathy and weakness or acute coma in children with a defect in carnitine uptake. Ann Neurol 30, 709–716

Campos Y, Huertas R, Lorenzo G et al. (1993) Plasma **carnitine insufficiency** and effectiveness of L-carnitine therapy in patients with mitochondrial myopathy. Muscle Nerve 16, 150–153

Dougherty FE, Ernst SG, Aprille JR. (1994) Familial occurrence of intestinal obstruction in children with the syndrome of mitochondrial encephalomyopathy, lactic acidosis, and stroke-like episodes (MELAS). J Pediatr 125, 758–761

9

Folmer O, Black M, Hoeh W, Lutz R, Vrijenhoek R (1994) DNA primers for amplification of mitochondrial cytochrome c oxidase subunit I from diverse metazoan invertebrates. Mol. Mar. Biol. Biotechnol. 3, 294–299. <http://www.ncbi.nlm.nih.gov/pubmed/7881515>

Chen RS, Huang CC, Chu NS. (1997) **Coenzyme Q10** treatment in mitochondrial encephalomyopathies. Short-term double-blind, crossover study. Eur Neurol 37, 212–218

Lam CW, Lau CH, Williams JC et al. (1997) Mitochondrial myopathy, encephalopathy, lactic acidosis and stroke-like episodes (MELAS) triggered by **valproate** therapy. Eur J Pediatr 156, 562–564

Chan A, Reichmann H, Kogel A et al. (1998) Metabolic changes in patients with mitochondrial myopathies and effects of **coenzyme Q10 therapy**. J Neurol 245, 681–685
Wallace D C, Brown M D, Lott M T (1999) **Mitochondrial DNA variation in human evolution and disease**. Gene 238, 211-230.

Andreu AL, Hanna MG, Reichmann H et al. (1999) Exercise intolerance due to mutations in the cytochrome b gene of mitochondrial DNA. N Engl J Med 341, 1037–1044

Barbiroli B, Iotti S, Lodi R. (1999) Improved brain and muscle mitochondrial respiration with **CoQ**. An in vivo study by 31P-MR spectroscopy in patients with mitochondrial cytopathies. Biofactors 9, 253–260

Krahenbühl S, Brandner S, Kleinle S et al. (2000) **Mitochondrial cytopathies represent a risk factor for valproate-induced fulminant liver failure**. Liver 20, 346–348

Klopstock T, Querner V, Schmidt F et al. (2000) A placebo-controlled crossover trial of **creatine in mitochondrial diseases**. Neurology 55, 1748–1751

Spelbrink JN, Li FY, Tiranti V et al. (2001) Human mitochondrial DNA deletions associated with mutations in the gene encoding Twinkle, **a phage T7 gene 4-like protein localized in mitochondria**. Nat Genet 28, 223–231

[DiMauro S.](http://www.ncbi.nlm.nih.gov/pubmed/11735374) (2001) Lessons from mitochondrial DNA mutations. Semin Cell Dev Biol. 12(6), 397-405. <http://www.ncbi.nlm.nih.gov/pubmed/11735374>

Rose G, Passarino G, Carrieri G, Altomare K, Greco V, Bertolini S et al. (2001) **Paradoxes in longevity: sequence analysis of mtDNA haplogroup J in centenarians**. Eur J Hum Genet 9, 701-707 <http://www.ncbi.nlm.nih.gov/pubmed/11571560>

« The general picture that emerges from the study is that the J haplogroup of centenarians is surprisingly similar to that found in complex diseases, as well as in Leber Hereditary Optic Neuropathy. This finding implies that the same mutations could predispose to disease or longevity, probably according to individual-specific genetic backgrounds and stochastic events. This data reveals another paradox of centenarians and confirms the complexity of the longevity trait. »

Deschauer M, Zierz S. (2002) Defekte der intergenomischen Kommunikation: Mutationen der Kern-DNA und multiple Deletionen der mitochondrialen DNA bei chronisch progressiver externer Ophthalmoplegie. Akt Neurol 30, 103–106

[Morales CT¹, Atencio DP, Oca-Cossio J, Diaz F.](http://www.ncbi.nlm.nih.gov/pubmed/14573777) (2003) Techniques and pitfalls in the detection of pathogenic mitochondrial DNA mutations. J Mol Diagn. 5(4), 197-208. <http://www.ncbi.nlm.nih.gov/pubmed/14573777>

Chinnery PF, Bindoff LA. (2003) 116th ENMC international workshop: the treatment of mitochondrial disorders, 14–16th March 2003, Naarden, The Netherlands. Neuromusc Disord 13, 757–764

Hanisch F, Zierz S. (2003) **Only transient increase of serum CoQ subset 10 during long-term CoQ10 therapy in mitochondrial ophthalmoplegia**. Eur J Med Res 8, 485–491

Lamperti C, Naini A, Hirano M et al. (2003) Cerebellar ataxia and coenzyme Q10 deficiency. Neurology 60, 1206–1208

Chinnery PF, DiMauro D, Shanske S et al. (2004) Risk of developing a mitochondrial DNA deletion disorder. Lancet 364, 592–596

Jakobs S (2004) Mitochondrien - Dynamische Kraftwerke der Zelle. Max-Planck-Institut für biophysikalische Chemie. Karl-Friedrich-Bonhoeffer-Institut MPIbpc News 10(12) http://www3.mpibpc.mpg.de/groups/hell/other_publications/MPInews_dec04_Jakobs.pdf

DiMauro S, Mancuso M, Naini A. (2004) **Mitochondrial encephalomyopathies. Therapeutic Approach**. Ann NY Acad Sci 1011, 232–245

- [Wong LJ](http://www.ncbi.nlm.nih.gov/pubmed/15126301). (2004) Comprehensive molecular diagnosis of mitochondrial disorders: qualitative and quantitative approach. *Ann N Y Acad Sci.* 1011, 246-58.
<http://www.ncbi.nlm.nih.gov/pubmed/15126301>
- Martí R, Spinazzola A, Tadese S et al. (2004) Definitive diagnosis of mitochondrial neurogastrointestinal encephalomyopathy by biochemical assays. *Clin Chem* 50, 120–124
- Taylor RW, Turnbull DM (2005) **Mitochondrial DNA mutations in human disease.** *Nature Publishing Group.* 6, 389-402
<http://www.nature.com/scitable/content/mitochondrial-dna-mutations-in-human-disease-14018738>
- Wong L-JC, Boles RG (2005) **Mitochondrial DNA analysis in clinical laboratory diagnostics.** *Clin Chim Acta* 354, 1-20. <http://www.ncbi.nlm.nih.gov/pubmed/15748595>
- Koga Y, Akita Y, Nishioka J et al. (2005) **L-arginine** improves the symptoms of strokelike episodes in MELAS. *Neurology* 64, 710–712
- Kornblum C, Schröder R, Müller K et al. (2005) Creatine has no beneficial effect on skeletal muscle energy metabolism in patients with single mitochondrial DNA deletions. *Eur J Neurol* 12, 300–309
- Quinzii CM, Kattah AG, Naini A et al. (2005) Coenzyme Q deficiency and cerebellar ataxia associated with an aprataxin mutation. *Neurology* 64, 539–541
- Pineda M, Ormazabal A, López-Gallardo E et al. (2006) Cerebral folate deficiency and leukoencephalopathy caused by a mitochondrial DNA deletion. *Ann Neurol* 59, 394–398
- Chinnery PF, Majamaa K, Turnbull D et al. (2006) **Treatment for mitochondrial disorders.** *Cochrane Database Sys Rev* 1, CD004426
- Horvath R, Hudson G, Ferrari G et al. (2006) Phenotypic spectrum associated with mutations of the mitochondrial polymerase gamma gene. *Brain* 129, 1674–1684
- Horvath R, Schneiderat P, Schoser BG et al. (2006) Coenzyme Q10 deficiency and isolated myopathy. *Neurology* 66, 253–255
- de Bivort BL, Chen C-C, Perretti F et al. (2007) Metabolic implications for the mechanism of mitochondrial endosymbiosis and human hereditary disorders. *J.Theor. Biol.*
<http://www.necsi.edu/research/sysbio/jtbio/jtbio.pdf>
- Bourdon A., Minai L, Serre V et al. (2007) Mutation of RRM2B, encoding p53-controlled ribonucleotide reductase (p53R2), causes severe mitochondrial DNA depletion. *Nat Genet* 39, 776–780
- Gempel K, Topaloglu H, Talim B et al. (2007) The myopathic form of **coenzyme Q10 deficiency** is caused by mutations in the electron transferring flavoprotein dehydrogenase (ETFDH) gene. *Brain* 130, 2037–2044
- Schaefer AM, McFarland R, Blakely EL et al. (2008) **Prevalence of mitochondrial DNA disease in adults.** *Ann Neurol* 63, 35–39 <http://www.ncbi.nlm.nih.gov/pubmed/17886296>
“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 ».
- [Pagliarini DJ, Calvo SE, Chang B et al.](http://dx.doi.org/10.1016/j.cell.2008.06.016) (2008) A Mitochondrial Protein Compendium Elucidates Complex I Disease Biology. *Cell* 134(1), 112–123 [Switch to Standard View](http://dx.doi.org/10.1016/j.cell.2008.06.016)
DOI: <http://dx.doi.org/10.1016/j.cell.2008.06.016>
- Hirano M, Kunz WS, DiMauro S. (2008) **Mitochondrial diseases.** In: Engel J, Pedley TA, eds. *Epilepsy: a comprehensive Textbook.* Philadelphia: Lippincott Williams & Wilkins; 2621–2630

Verny C, Amati-Bonneau P, Letournel F et al. (2008) **Mitochondrial DNA A3243G mutation involved in familial diabetes, chronic intestinal pseudo-obstruction and recurrent pancreatitis.** Diabetes Metab 34, 620–626

[Lyamzaev KG](#), [Nepryakhina OK](#), [Saprunova VB](#) et al. (2008) **Novel mechanism of elimination of malfunctioning mitochondria (mitoptosis): Formation of mitoptotic bodies and extrusion of mitochondrial material from the cell.** [Biochimica et Biophysica Acta \(BBA\) - Bioenergetics 1777\(7–8\)](#), 817–825. DOI: 10.1016/j.bbabi.2008.03.027
<http://www.sciencedirect.com/science/article/pii/S0005272808000832>

Gardner A, Boles R G (2008) **Mitochondrial energy depletion in depression with somatization.** Psychother Psychosom. 77(2), 127-9.

Gardner A, Boles R G (2008) **Symptoms of somatization as a rapid screening tool for mitochondrial dysfunction in depression.** Biopsychosoc Med. 22, 2, 7.

Feddersen BL, DE LA Fontaine L, Sass JO et al. (2009) **Mitochondrial neurogastrointestinal encephalomyopathy mimicking anorexia nervosa.** Am J Psychiatry 166, 494–495

Jones CN, Millerb Ch, Tenenbaum A et al. (2009) **Antibiotic effects on mitochondrial translation and in patients with mitochondrial translational defects.** Mitochondrion, 429-437

Blakely EL, Trip SA, Swalwell H et al. (2009) A new mitochondrial transfer RNAPro gene mutation associated with myoclonic epilepsy with ragged-red fibers and other neurological features. Arch Neurol 66, 399–402

Di Fonzo A, Ronchi D, Lodi T et al. (2009) The mitochondrial disulfide relay system protein GFER is mutated in autosomal-recessive myopathy with cataract and combined respiratory-chain deficiency. Am J Hum Genet 84, 594–604

Leitlinien der Arbeitsgemeinschaft für Pädiatrische Stoffwechselstörungen (APS) (2009) **Diagnostik und Therapieansätze bei Mitochondriopathien im Kindes- und Jugendalter.** Leitlinien-Entwicklungsstufe 2

http://www.mito-center.org/mito-center.org/fileadmin/user_upload/LLUpdateAWMF2009Final.pdf
Flowchart http://www.mito-center.org/mito-center.org/fileadmin/user_upload/MitoFlowchar.pdf

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.**»

Quinzii CM, Hirano M (2010) **Coenzyme Q and mitochondrial disease.** Disabil Res Rev 16, 183–88

Mitochondrial Toxicity (2010) <http://www.mitoaction.org/blog/medication-exposures-mitochondrial-toxicity>

„**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.**»

Giordano C, Pichiorri F, Blakely EL et al. (2010) Isolated distal myopathy of the upper limbs associated with mitochondrial DNA depletion and polymerase gamma mutations. Arch Neurol 67, 1144–1146

[Zhang Q, Raouf M, Chen Y](#) et al. (2010) **Circulating mitochondrial DAMPs cause inflammatory responses to injury.** *Nature*. 464(7285), 104-7. doi: 10.1038/nature08780.

<https://www.ncbi.nlm.nih.gov/pubmed/20203610>

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

Zsurka G, Hampel KG, Nelson I et al. (2010) Severe epilepsy as the major symptom of new mutations in the mitochondrial tRNA(Phe) gene. *Neurology* 74, 507–512

Ghezzi D, Sevrioukova I, Invernizzi F et al. (2010) Severe X-linked mitochondrial encephalomyopathy associated with a mutation in apoptosis-inducing factor. *Am J Hum Genet* 86, 639–649

Larsson NG (2010) Somatic mitochondrial DNA mutations in mammalian aging. *Annual Review of Biochemistry* 79, 683–706

Park CB, Larsson NG (2011) Mitochondrial DNA mutations in disease and aging. *The Journal of Cell Biology* 193 (5), 809-818

Kukat C, Wurm CA, Spähr H, Falkenberg M, Larsson N-G, Jakobs S (2011) Super-resolution microscopy reveals that mammalian mitochondrial nucleoids have a uniform size and frequently contain a single copy of mtDNA. *Proceedings of the National Academy of Science of the United States of America* 108 (33), 13534-13539

[Konradi Chr, Sullivan SE, Clay](#) HB (2012) **Mitochondria, oligodendrocytes and inflammation in bipolar disorder: evidence from transcriptome studies points to intriguing parallels with multiple sclerosis.** *Neurobiology of disease* 45(1), 37-47

<http://www.ncbi.nlm.nih.gov/pubmed/21310238>

« **Experimental paradigms for multiple sclerosis demonstrate a tight link between energy metabolism, inflammation and demyelination.** »

Mutschler R (2012) **Praxistipps: Regenerative Mitochondrien-Medizin- von der Forschung in die Praxis.** in: Zeitschrift für Orthomolekulare Medizin 3/2012

http://www.google.de/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&ved=0CDAQFjAA&url=http%3A%2F%2Fwww.researchgate.net%2Fprofile%2FRainer_Mutschler%2Fpublication%2F236951119_Regenerative_Mitochondrien-Medizin-von_der_Forschung_in_die_Praxis%2Ffile%2F72e7e51a5e34c23a9b.pdf&ei=kTu-U_3iKsnBO462gJAB&usq=AFQjCNGZDe31y_3iAKZs3no84ajkvSYkiw&bvm=bv.70138588.d.ZWU

Kukat Chr, Wurm, ChrA, Spähr H, Falkenberg M et al. (2012) **Einblicke in die Nanowelt der Mitochondrien und die Organisation ihres Erbgutes.** Insights into the nanoworld of mitochondria and the organisation of their genome. *Forschungsbericht - Max-Planck-Institut für Biologie des Alterns.* http://www.mpg.de/5039005/Nanowelt_Mitochondrien?c=5732389

Gröber A (2012) **Mitochondriale Toxizität von Arzneimitteln.** *MMP* 35(12) 445-457

http://www.google.de/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&ved=0CCsQFjAA&url=http%3A%2F%2Fwww.mikronaehrs.toff.de%2Fpdf%2FGroe_Mitotox_von%2520Arzneimitteln_MMP_2012.pdf%3Fv%3D2&ei=6L2VU4HnKsq7Qbz0IHwDA&usq=AFQjCNE5bXm3Y113EGda_eqlwhKxnWJrwg&bvm=bv.68445247.d.ZGU

„**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.**“

Houtkooper RH et al. (2013) **Mitochondrial protein imbalance as a conserved longevity mechanism.** *Nature* 497, 451-457

<http://www.the-scientist.com/?articles.view/articleNo/35673/title/Inhibit-Mitochondria-to-Live-Longer/>

Pall M (2013) The NO/ONOO- cycle as the cause of complex mitochondrial/energy metabolism dysfunction, *Seminar Klinische Mitochondrien- und Umweltmedizin.*

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3856065/>

[Kalghatgi S, Spina CS, Costello JC](#) et al. (2013) **Bactericidal Antibiotics Induce Mitochondrial Dysfunction and Oxidative Damage in Mammalian Cells.** *Sci Transl Med.* Jul 3, 5(192), 192ra85.

doi: [10.1126/scitranslmed.3006055](https://doi.org/10.1126/scitranslmed.3006055) <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3760005/>
http://www.google.de/url?sa=t&rct=j&q=&esrc=s&source=web&cd=5&ved=0CFoQFjAE&url=http%3A%2F%2Fwww.bu.edu%2Fabl%2Ffiles%2Fstm_kalghatgi.pdf&ei=breVU4qRDMKu7AadvYHwDA&usq=AFQjCNF_ZSDL-OJQ_jXjBSiLFHH926mOjQ&bvm=bv.68445247,d.ZGU

Kaipparettu BA, Ma Y, Park JH et al. (2013) **Crosstalk from Non-Cancerous Mitochondria Can Inhibit Tumor Properties of Metastatic Cells by Suppressing Oncogenic Pathways.** PLOSone <http://dx.doi.org/10.1371/journal.pone.0061747>
<http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0061747>

Nykky J, Vuento M, Gilbert L (2014) **Role of Mitochondria in Parvovirus Pathology.** PLOS one 9(1), e86124 doi: 10.1371/journal.pone.0086124. eCollection 2014.
<http://www.ncbi.nlm.nih.gov/pubmed/24465910>
<http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0086124>

[Ray A](#), [Martinez BA](#), [Berkowitz LA](#), [Caldwell GA](#), [Caldwell KA](#) (2014) **Mitochondrial dysfunction, oxidative stress, and neurodegeneration elicited by a bacterial metabolite in a C. elegans Parkinson's model.** *Cell Death Dis.* 5, e984. doi: 10.1038/cddis.2013.513.
<http://www.ncbi.nlm.nih.gov/pubmed/24407237>

Ye K et al. (2014) **Extensive pathogenicity of mitochondrial heteroplasmy in healthy human individuals.** PNAS, doi:10.1073/pnas.1403521111
<http://www.pnas.org/content/suppl/2014/07/02/1403521111.DCSupplemental/pnas.1403521111.sapp.pdf>
<http://www.pnas.org/content/early/2014/07/02/1403521111/suppl/DCSupplemental>

Nicholson G (2014) **Mitochondrial dysfunction and chronic disease: treatment with natural supplements,** *Altern. Therapies Health Medicine* 20(1), 18-25. [pdf_doc](#)

Nicolson GL, Settineri R, Ellithorpe RR (2014) Review Article Open Access. **Neurodegenerative and Fatiguing Illnesses, Infections and Mitochondrial Dysfunction: Use of Natural Supplements to Improve Mitochondrial Function.** *Functional Foods in Health and Disease* 4(1), 23-65
<http://functionalfoodscenter.net/files/81861886.pdf>

Koslik HJ, Hamilton G, Golomb BA (2014) **Mitochondrial Dysfunction in Gulf War Illness Revealed by 31 Phosphorous Magnetic Resonance Spectroscopy: A Case Control Study.** PLoS ONE 9(3), e02887 <http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0092887>

Metzger MJ, Reinisch C, Sherry J, Goff SP (2015) **Horizontal transmission of clonal cancer cells causes leukemia in soft-shell clams.** *Cell* doi:10.1016/j.cell.2015.02.042.
<http://www.cell.com/cell/pdf/S0092-8674%2815%2900243-3.pdf>
« We therefore analyzed mitochondrial DNA (mtDNA) sequences and polymorphic microsatellite repeat loci and found that the genotypes of the neoplastic cells do not match those of their hosts. Our findings suggest that horizontal transmission of cancer cells is more widespread in nature than previously supposed“.

[West AP](#), [Khoury-Hanold W](#), [Staron M](#) et al. (2015) **Mitochondrial DNA stress primes the antiviral innate immune response.** *Nature* doi:10.1038/nature14156 <http://www.ncbi.nlm.nih.gov/pubmed/25642965>
<http://www.nature.com/nature/journal/vaop/ncurrent/full/nature14156.html>
„Here we show that moderate mtDNA stress elicited by TFAM deficiency engages cytosolic antiviral signalling to enhance the expression of a subset of interferon-stimulated genes. Mechanistically, we find that aberrant mtDNA packaging promotes escape of mtDNA into the cytosol, where it engages the DNA sensor cGAS (also known as MB21D1) and promotes STING (also known as TMEM173)-IRF3-dependent signalling to elevate interferon-stimulated gene expression, potentiate type I interferon responses and confer broad viral resistance. Furthermore, we demonstrate that herpesviruses induce mtDNA stress, which enhances antiviral signalling and type I interferon responses during infection. Our results further demonstrate that mitochondria are central participants in innate immunity, identify mtDNA stress as a cell-intrinsic trigger of antiviral signalling and suggest that cellular monitoring of mtDNA homeostasis cooperates with canonical virus sensing mechanisms to fully engage antiviral innate immunity.“

Morris G, Berk M (2015) **The many roads to mitochondrial dysfunction in neuroimmune and neuropsychiatric disorders.** *BMC Medicine.* 13(38) 1-24
<http://www.biomedcentral.com/1741-7015/13/68>

Reddy P, Ocampo A et al. (2015) **Selective elimination of mitochondrial mutations in the germline by genome editing.** Cell, doi:10.1016/j.cell.2015.03.051.
<http://www.cell.com/cell/pdf/S0092-8674%2815%2900371-2.pdf>
<http://www.cell.com/abstract/S0092-8674%2815%2900371-2>
« **This strategy represents a potential therapeutic avenue for preventing the transmission of human mitochondrial diseases.**»

Boles RG, Zaki EA, Kerr JR et al. (2015) **Increased prevalence of two mitochondrial DNA polymorphisms in functional disease: Are we describing different parts of an energy-depleted elephant? Mitochondrion.** pii: S1567-7249(15)00048-3. doi: 10.1016/j.mito.2015.04.005. [Epub ahead of print] <http://www.ncbi.nlm.nih.gov/pubmed/25934187>

Kulinski B (2015) **Mitochondrien: Symptome, Diagnose und Therapie.**
<http://www.narayana-verlag.de/Mitochondrien-Bodo-Kuklinski/b17290>

Ma H, Folmes CD, Wu J et al. (2015) **Metabolic rescue in pluripotent cells from patients with mtDNA disease.** Nature, doi:10.1038/nature14546. <http://www.ncbi.nlm.nih.gov/pubmed/26176921>
<http://www.nature.com/nature/journal/vaop/ncurrent/full/nature14546.html>
<http://www.ipscell.com/tag/metabolic-rescue-in-pluripotent-cells-from-patients-with-mtdna-disease/>

Latorre-Pellicer A et al. (2016) **Mitochondrial and nuclear DNA matching shapes metabolism and healthy ageing.** Nature, doi:10.1038/nature18618
<http://www.nature.com/nature/journal/vaop/ncurrent/full/nature18618.html>

Aufschnaiter A, Kohler V, Dissl J, Buettner S (2016) **Mitochondrial lipids in neurodegeneration.** Cell and Tissue Research. <http://paperity.org/p/77456795/mitochondrial-lipids-in-neurodegeneration>
https://www.researchgate.net/publication/305630088_Mitochondrial_lipids_in_neurodegeneration

Grier J, Hirano M, Karaa A, Shepard E, Thompson JLP (2018) **Diagnostic odyssey of patients with mitochondrial disease. Results of a survey.** Neurology Genetics 4 (2) ArticleOpen Access.
http://ng.neurology.org/content/4/2/e230?utm_source=STAT+Newsletters&utm_campaign=06b3c09970-MR&utm_medium=email&utm_term=0_8cab1d7961-06b3c09970-149720033

- ➔ **Q10 und L-Carnitin, Mitochondrienfunktion** http://www.kabilahsystems.de/q10_und_l.pdf
- ➔ **L-Arginin** <http://www.erlebnishaft.de/l-arginin.pdf>
- ➔ **Methyl- und Harnstoff Zyklus** <http://xerlebnishaft.de/bildmethyl-arginin.pdf>
- ➔ **Zytoskelett** <http://www.xerlebnishaft.de/zytoskelett.pdf>
- ➔ **Fibromyalgie** http://www.erlebnishaft.de/chronic_fatigue.pdf
- ➔ **Krebsstammzelltherapie** <http://www.xerlebnishaft.de/krebsstammzelltherapie.pdf>

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>

Beninati T, Lo N, Sacchi L, Genchi C, Noda H, Bandi C (2004) **A novel alpha-proteobacterium resides in the mitochondria of ovarian cells of the tick Ixodes ricinus.** Appl. Environ. Microbiol. 70, 2596-2602. <http://www.ncbi.nlm.nih.gov/pubmed/15128508>

Lo N, Beninati T, Sacchi L (2004) [Emerging rickettsioses]. *Parassitologia*. 46(1-2), 123-6.
<http://www.ncbi.nlm.nih.gov/pubmed/15305700>

«... 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. ...»

Sacchi L, Bigliardi E, Corona S et al. (2004) **A symbiont of the tick *Ixodes ricinus* invades and consumes mitochondria in a mode similar to that of the parasitic bacterium *Bdellovibrio bacteriovorus***. *Tissue Cell* 36, 43-53. <http://www.ncbi.nlm.nih.gov/pubmed/14729452>
http://link.springer.com/chapter/10.1007/978-90-481-9837-5_90#page-1

Lo N, Beninati T, Sacchi L, Bandi C (2006) **An alpha-proteobacterium invades the mitochondria of the tick *Ixodes ricinus***. In *Insect Symbiosis II*, edited by K. Bourtzis and T. Miller, CRC Press Boca Raton. <http://www.crcnetbase.com/doi/abs/10.1201/9781420005936.ch2>

Sassera D, Beninati T, Bandi C et al. (2006) **'Candidatus *Midichloria mitochondrii*', an endosymbiont of the tick *Ixodes ricinus* with a unique intramitochondrial lifestyle**. *Int J Syst Evol Microbiol*. Nov, 56(Pt 11), 2535-40. <http://www.ncbi.nlm.nih.gov/pubmed/17082386>
„'Candidatus *M. mitochondrii*' is the first bacterium to be identified that resides within animal mitochondria.“

Epis S, Sassera D, Beninati T, Lo N, Beati L, Plesman J, Rinaldi L, McCoy KD, Torina A, Sacchi L, Clementi E, Genchi M, Magnino S, Bandi C (2008) ***Midichloria mitochondrii* is widespread in hard ticks (*Ixodidae*) and resides in the mitochondria of phylogenetically diverse species**. *Parasitology*. 135, 485-494. <http://www.ncbi.nlm.nih.gov/pubmed/18205982>

Sassera D, Lo N, Bouman EAP et al. (2008) "Candidatus *Midichloria*" Endosymbionts Bloom after the Blood Meal of the Host, the Hard Tick *Ixodes ricinus* ▽ *Appl Environ Microbiol*. 74(19), 6138–6140. Published online Aug 8, 2008. doi: [10.1128/AEM.00248-08](https://doi.org/10.1128/AEM.00248-08) PMID: PMC2565945
<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2565945/>

Sassera S, Lo N, Epis S, D'Auria G, Montagna M, Comandatore F, Horner D, Peretó J, Luciano AM, Franciosi F, Ferri E, Crotti E, Bazzocchi C, Daffonchio D, Sacchi L, Moya A, Latorre A, Bandi C (2011) **Phylogenomic evidence for the presence of a flagellum and *cbb3* oxidase in the free-living mitochondrial ancestor**. *Molecular Biology and Evolution*. 10.1093/molbev/msr159 [PubMed] [Europe PMC] [Abstract] <http://www.ncbi.nlm.nih.gov/pubmed/21690562>

“We have sequenced the genome of *Candidatus Midichloria mitochondrii*, a novel and phylogenetically divergent member of the Rickettsiales. We found that it possesses unique gene sets found in no other Rickettsiales, including 26 genes associated with flagellar assembly, and a *cbb(3)*-type cytochrome oxidase. Phylogenomic analyses show that these genes were inherited in a vertical fashion from an ancestral α -proteobacterium, and indicate that the FMA [free-living mitochondrial ancestor] possessed a flagellum, and could undergo oxidative phosphorylation under both aerobic and microoxic conditions. These results indicate that the FMA played a more active and potentially parasitic role in eukaryogenesis than currently appreciated and provide an explanation for how the symbiosis could have evolved under low levels of oxygen.” <http://www.uniprot.org/proteomes/UP000006639>

Mariconti Mara (2012) **Doctoral Thesis. *Midichloria mitochondrii* as an emerging infectious agent: molecular and immunological studies on the intra-mitochondrial symbiont of the hard tick *Ixodes ricinus***. https://air.unimi.it/retrieve/handle/2434/216312/263086/phd_unimi_R08510.pdf

Williams-Newkirk AJ, Rowe LA, Mixson-Hayden TR, Dasch GA (2012) **Presence, genetic variability, and potential significance of "*Candidatus Midichloria mitochondrii*" in the lone star tick *Amblyomma americanum***. *Exp Appl Acarol*. 2012 Nov;58(3):291-300. doi: 10.1007/s10493-012-9582-5. Epub 2012 Jun 8. PMID: 22678102 [Related citations](#)

Najm N-A, Silaghi C, Bell-Sakyi L et al. (2012) **Detection of bacteria related to *Candidatus Midichloria mitochondrii* in tick cell lines**. *Parasitol Res* 110, 437–442 DOI 10.1007/s00436-011-2509-y <http://www.ncbi.nlm.nih.gov/pubmed/21748354>

Epis S, Mandrioli M, Genchi M et al. (2013) **Localization of the bacterial symbiont *Candidatus Midichloria mitochondrii* within the hard tick *Ixodes ricinus* by whole-mount FISH staining**. *Ticks and Tick-borne Diseases* 4, 1–2, 39–45 <http://www.sciencedirect.com/science/article/pii/S1877959X1200060X>

Mariconti M, Epis S, Gaibani P et al. (2012) **Humans parasitized by the hard tick *Ixodes ricinus* are seropositive to *Midichloria mitochondrii*: is *Midichloria* a novel pathogen, or just a marker of tick bite?** *Pathogens and Global Health* 106(7), 391-396 <http://www.ncbi.nlm.nih.gov/pubmed/23265610>
“...This implies that the immunology of the response toward the saliva of *I. ricinus* is to be reconsidered on the basis of potential effects of *M. mitochondrii* and poses the basis for the development of novel markers for investigating the exposure of humans and animals to this tick species.”

Montagna M, Sasser D, Epis S et al. (2013) “**Candidatus *Midichloriaceae*” fam. nov. (Rickettsiales), an ecologically widespread clade of intracellular alphaproteobacteria.** *Appl Environ Microbiol.* 79(10), 3241-8 <http://www.ncbi.nlm.nih.gov/pubmed/23503305>
<http://aem.asm.org/content/79/10/3241.full>

Bazzocchi C, Mariconti M, Sasser D (2013) [Molecular and serological evidence for the circulation of the tick symbiont *Midichloria* \(Rickettsiales: *Midichloriaceae*\) in different mammalian species.](#) *Parasit Vectors.* 6, 350. doi: 10.1186/1756-3305-6-350. PMID: 24330701 [Free Article](#)

Boscaro V, Petroni G, Ristori A et al. (2013) “**Candidatus *Defluviella procrastinata*” and “Candidatus *Cyrtobacter zanobii*”, two novel ciliate endosymbionts belonging to the “*Midichloria* clade”.** *Microb. Ecol.* 65, 302–310. doi:10.1007/s00248-012-0170-3.

Wang Z, Wu M (2014) **Complete Genome Sequence of the Endosymbiont of *Acanthamoeba* Strain UWC8, an Amoeba Endosymbiont Belonging to the “Candidatus *Midichloriaceae*” Family in Rickettsiales.** doi: 10.1128/genomeA.00791-14 *Genome Announc.* 2(4) e00791-14
<http://genomea.asm.org/content/2/4/e00791-14.full.pdf+html>

Wang Z, Wu M (2014) **Phylogenomic Reconstruction Indicates Mitochondrial Ancestor Was an Energy Parasite.** *PLoS ONE* 9(10): e110685. doi:10.1371/journal.pone.0110685
<http://www.plosone.org/article/fetchObject.action?uri=info:doi/10.1371/journal.pone.0110685&representation=PDF>
<http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0110685>
“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.”

Pfanner N (2015) **Publications of Pfanner Lab.**
<http://www.biochemie.uni-freiburg.de/ag/pfanner/publications>

Gofton AW, Oskam CL, Lo N et al. (2015) **Inhibition of the endosymbiont “Candidatus *Midichloria mitochondrii*” during 16S rRNA gene profiling reveals potential pathogens in *Ixodes* ticks from Australia.** *Parasit Vectors.* 8(1), 345. doi: 10.1186/s13071-015-0958-3.
<http://www.ncbi.nlm.nih.gov/pubmed/26108374>

- ➔ **Midichloria** <http://en.wikipedia.org/wiki/Midichloria>
- ➔ **Midichloria mitochondrii**
<http://www.ncbi.nlm.nih.gov/pubmed/?term=Midichloria+mitochondrii>
- ➔ **Rickettsiales, Midichloriaceae.** Related citations for PubMed
http://www.ncbi.nlm.nih.gov/pubmed?cmd=Link&dbFrom=PubMed&from_uid=24330701&holding=f1000%2Cf1000m%2Cisrctn

Rickettsien, rickettsia

BEATI L, PETER O, BURGDORFER W, AESCHLIMA A, RAOULT D (1993) **Confirmation that *Rickettsia helvetica* sp. nov. Is a Distinct Species of the Spotted Fever Group of Rickettsiae.** INTERNATIONAL JOURNAL OF SYSTEMATIC BACTERIOLOGY, July 1993, p. 521-526
International Union of Microbiological Societies 0020-7713/93/030521-06\$02.00/0 Vol. 43, No. 3
<http://www.microbiologyresearch.org/docserver/fulltext/ijsem/43/3/ijis-43-3-521.pdf?expires=1480493538&id=id&accname=guest&checksum=FBB9E8E4B3FADC24DC04AC2C2A253D69>
<https://www.researchgate.net/publication/15097497> Confirmation that *Rickettsia helvetica* sp nov Is a Distinct Species of the Spotted Fever Group of Rickettsiae
[Swiss agent, *Rickettsia helvetica*]

Andersson SG et al. (1998) **The genome sequence of *Rickettsia prowazekii* and the origin of mitochondria.** Nature 396 (6707), 133–40. doi:10.1038/24094. PMID 9823893

Nilsson¹ K, Lindquist O, Ai Jie Liu AJ et al (1999) ***Rickettsia helvetica* in *Ixodes ricinus* Ticks in Sweden.** J. Clin. Microbiol. 37(2) 400-403 <http://jcm.asm.org/content/37/2/400.full>

Emelyanov VV (2003) **Mitochondrial connection to the origin of the eukaryotic cell.** Eur J Biochem 270 (8), 1599–618. doi:10.1046/j.1432-1033.2003.03499.x. PMID 12694174

Paddock CD, Finley RW, Wright CS et al. (2008) ***Rickettsia parkeri* rickettsiosis and its clinical distinction from Rocky Mountain spotted fever.** Clin Infect Dis 47(9) 1188-96
<http://cid.oxfordjournals.org/content/47/9/1188>

Fredricks DN (2009) **Introduction to the Rickettsiales and Other Intracellular Prokaryotes.** *The Prokaryotes* pp 457-466 http://link.springer.com/referenceworkentry/10.1007%2F0-387-30745-1_18#page-1

Parola P, Paddock CD, Socolovschi C (2013) **Update on Tick-Borne Rickettsioses around the World: a Geographic Approach.** Clinical Microbiology Reviews 26(4), 657-702
<http://www.ncbi.nlm.nih.gov/pubmed/24092850>
<http://www.researchgate.net/publication/257350867> Update on Tick-Borne Rickettsioses around the World a Geographic Approach

Myers T, Lalani T, Dent M, et al. (2013) **Detecting *Rickettsia parkeri* infection from eschar swab specimens.** Emerg Infect Dis 19(5), 778-80 http://wwwnc.cdc.gov/eid/article/19/5/12-0622_article

Ekenna O, Paddock CD, Goddard J (2014) **Gulf coast tick rash illness in Mississippi caused by *Rickettsia parkeri*.** J Miss State Med Assoc 55(7) 216-9

Peacock BN, Gherezghiher TB, Hilario JD, Kellermann GH (2015) **New insights into Lyme disease.** Redox Biol. 5, 66-70. doi: 10.1016/j.redox.2015.03.002. Epub 2015 Mar 16.
<https://www.ncbi.nlm.nih.gov/pubmed/25838067>
«These results indicate that there is an imbalance of reactive oxygen species and cytosolic calcium in Lyme borreliosis patients. The results further suggest that oxidative stress and interrupted intracellular communication may ultimately contribute to a condition of mitochondrial dysfunction in the immune cells of Lyme borreliosis patients.»

Biggs HM, Behravesch CB, Bradley KK et al. (2016) **Diagnosis and management of tickborne Rickettsial diseases: Rocky Mountain spotted fever and other spotted fever group rickettsioses, ehrlichioses, and anaplasmosis -- United States.** MMWR Recomm Rep 65(2), 1-44
<http://www.cdc.gov/mmwr/volumes/65/rr/rr6502a1.htm>
http://www.cdc.gov/mmwr/volumes/65/rr/rr6502a1.htm?s_cid=rr6502a1_e

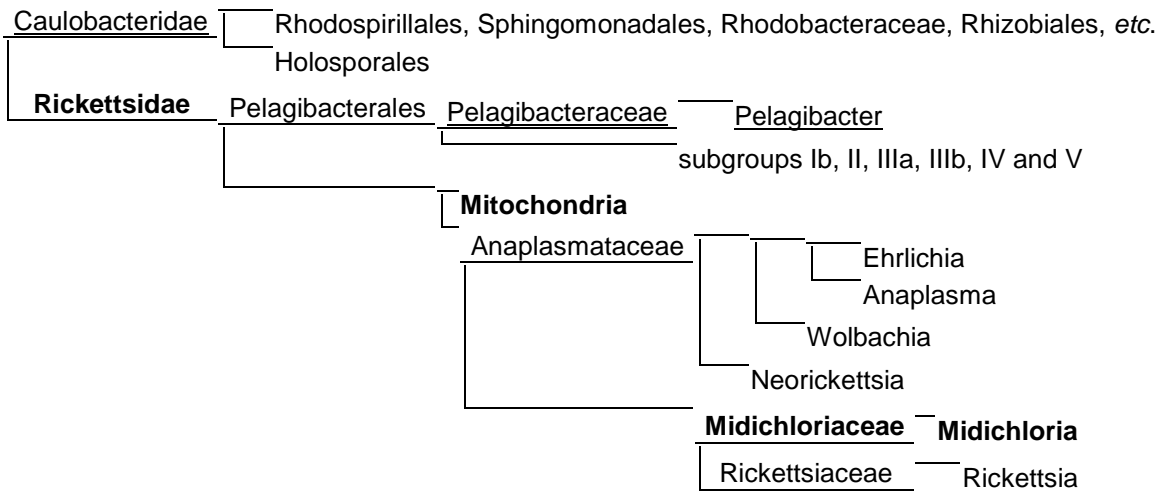
Drexler NA, Dahlgren FS, Heitman KN, et al. (2016) **National surveillance of spotted fever group rickettsioses in the United States, 2008-2012.** Am J Trop Med Hyg 94(1) 26-34
<http://www.ajtmh.org/content/94/1/26>

Piller C (2016) **The 'Swiss Agent': Long-forgotten research unearths new mystery about Lyme disease.** <https://www.statnews.com/2016/10/12/swiss-agent-lyme-disease-mystery/>

Rickettsia (2016) <https://en.wikipedia.org/wiki/Rickettsia> <https://de.wikipedia.org/wiki/Rickettsien>

Phylogeny of Rickettsiales

Magnetococcidae Magnetococcales Magnetococcaceae *Magnetococcus marinus*



Quelle, Source of origin: Ferla MP, Thrash JC, Giovannoni SJ, Patrick WM (2013) [New rRNA gene-based phylogenies of the Alphaproteobacteria provide perspective on major groups, mitochondrial ancestry and phylogenetic instability](https://doi.org/10.1371/journal.pone.0083383). PLoS ONE 8 (12), e83383. doi:10.1371/journal.pone.0083383. PMC 3859672. PMID 24349502 **Quelle, Source of origin:** <http://en.wikipedia.org/wiki/Midichloria>

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