

Q10 und L-Carnitinm, Mitochondrienfunktion

Mitochondrien <http://www.xerlebnishaft.de/mitochondrien.pdf>

Leitbefund: Lactazidose – Sonderform der metabolischen Azidose. <http://de.wikipedia.org/wiki/Laktazidose>
Bestimmung der organischen Säuren im Urin und der Aminosäuren im Serum, evtl. Muskelbiopsie.
ATP intrazellulär.

Main findings: lactic acidosis – as a special form of metabolic acidosis.
http://en.wikipedia.org/wiki/Lactic_acidosis. Determination of organic acids in urine and determination of serum amino acids, possibly muscle biopsy. Measurement of the intracellular ATP.

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[Cordero MD](#), [Alcocer-Gómez E](#), [Culic O](#) et al. (2013) NLRP3 Inflammasome is activated in Fibromyalgia: the effect of Coenzyme Q10. *Antioxid Redox Signal.* [Epub ahead of print]
<http://www.ncbi.nlm.nih.gov/pubmed/23886272>

[Shill DD](#), [Southern WM](#), [Willingham TB](#) et al. (2016) Mitochondria-specific antioxidant supplementation does not influence endurance exercise training-induced adaptations in circulating angiogenic cells, skeletal muscle oxidative capacity or maximal oxygen uptake. *The Journal of Physiology* 594(23) 7005-7014
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« In conclusion, endurance exercise training induced increases in multiple CAC types, and this adaptation is not modified by MitoQ supplementation. Furthermore, we demonstrate that a

mitochondrial-targeted antioxidant does not influence skeletal muscle or whole-body aerobic adaptations to exercise training. »

Rossmann MJ, Santos-Parker JR, Steward CAC et al. (2018) Chronic Supplementation With a Mitochondrial Antioxidant (MitoQ) Improves Vascular Function in Healthy Older Adults.

<https://doi.org/10.1161/HYPERTENSIONAHA.117.10787>

[Hypertension.HYPERTENSIONAHA.117.10787](https://pubs.aha.org/journal/Hypertension/HYPERTENSIONAHA.117.10787)

<http://hyper.ahajournals.org/content/early/2018/04/13/HYPERTENSIONAHA.117.10787>

„These findings in humans extend earlier preclinical observations and suggest that MitoQ and other therapeutic strategies targeting mitochondrial reactive oxygen species may hold promise for treating age-related vascular dysfunction.“

[Pfuhlmann K](#), [Schriever SC](#), [Baumann P](#) et al. (2018) Celastrol Induced Weight Loss is

Driven by Hypophagia and Independent From UCP1. [Diabetes](#). pii: db180146. doi:

10.2337/db18-0146. [Epub ahead of print] <https://www.ncbi.nlm.nih.gov/pubmed/30158241>

„Celastrol ist eine chemische Verbindung, die aus den Wurzelextrakten von *Tripterygium wilfordii* und *Celastrus repleii* isoliert wird. Celastrol ist ein pentacyclisches Triterpenoid und gehört zur Familie der Chinonmethide. [Wikipedia \(Englisch\)](#)“

<https://www.google.de/search?q=Celastrol&hl=de&btnG=Google+Search>

Medline search

<http://www.unboundmedicine.com/medline?in=kw%257CAnaplasma%2520phagocytophilum&in=jn%257C&in=au%257C>

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