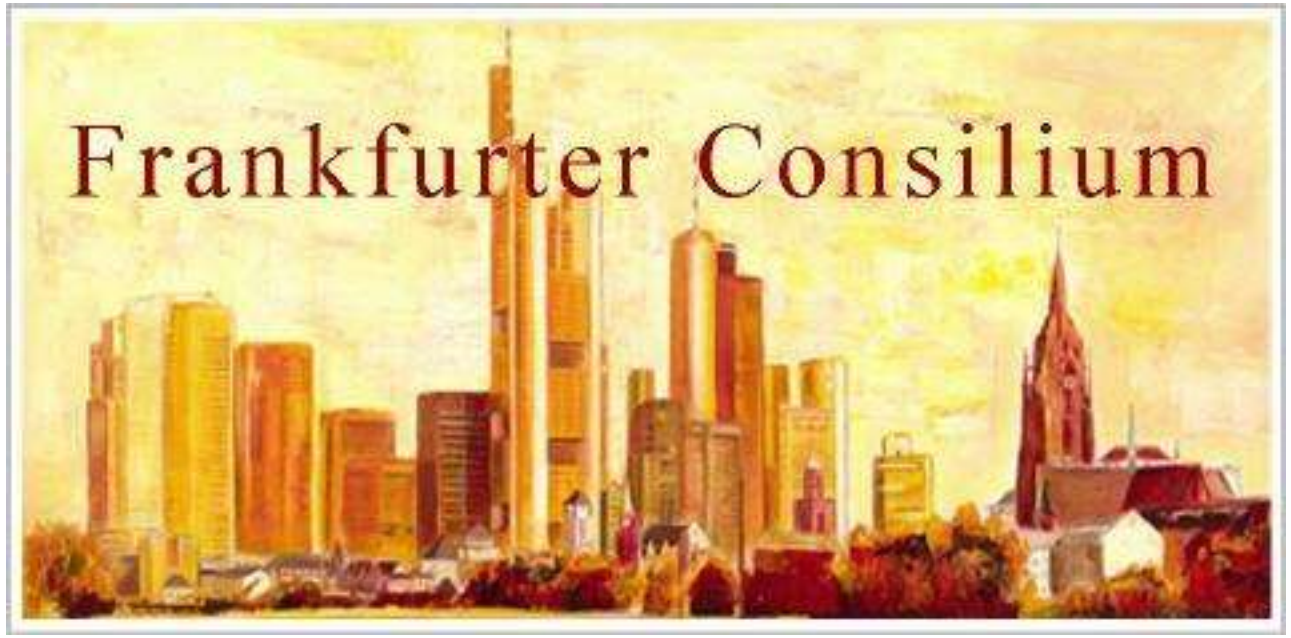
Bioenergetik

Resonanz – Regulation – Regeneration

Die neue Dimension der Biowissenschaften



Burkhard Poeggeler

Frankfurter Consilium, 23. November 2016

Altern,

Krankheit und Tod

entkommen

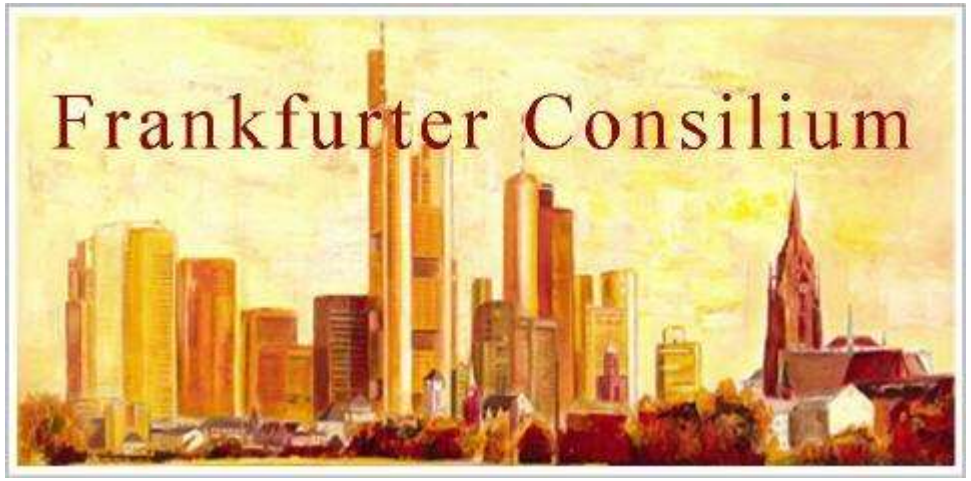
Gewidmet Gerhard Ohlenschläger dem Vordenker der aus
der Regulationsmedizin und Gesundheitsmedizin hervorgegangenen Endobolomics

Gesundheit

für alle

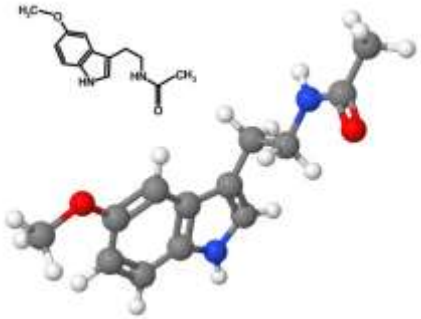
zeitlebens

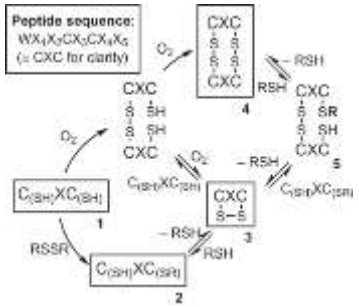
Gerhard Ohlenschläger

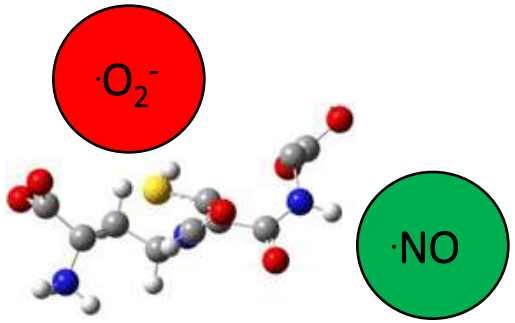


Philosoph, Wissenschaftler,
Forscher, Entdecker und Produktentwickler,
Mediziner, Arzt, Lehrer und Heiler,
Freund und Mentor des Frankfurter Consilium

Begründer der Regulationsmedizin
zusammen mit Prof. Dr. Dr. Jürgen Schole, Prof. Dr. Gerhard Uhlenbruck und Prof. Dr. Fritz Albert Popp

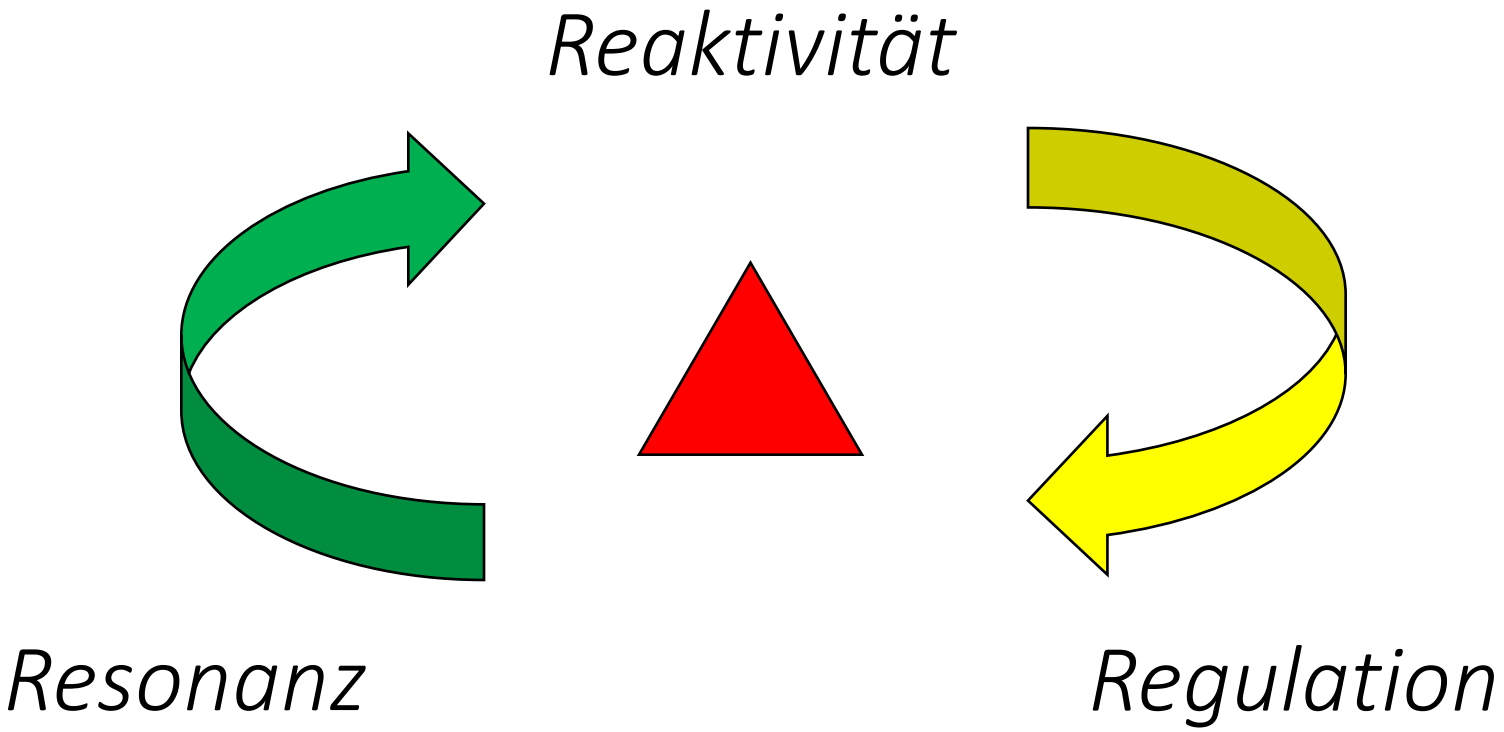




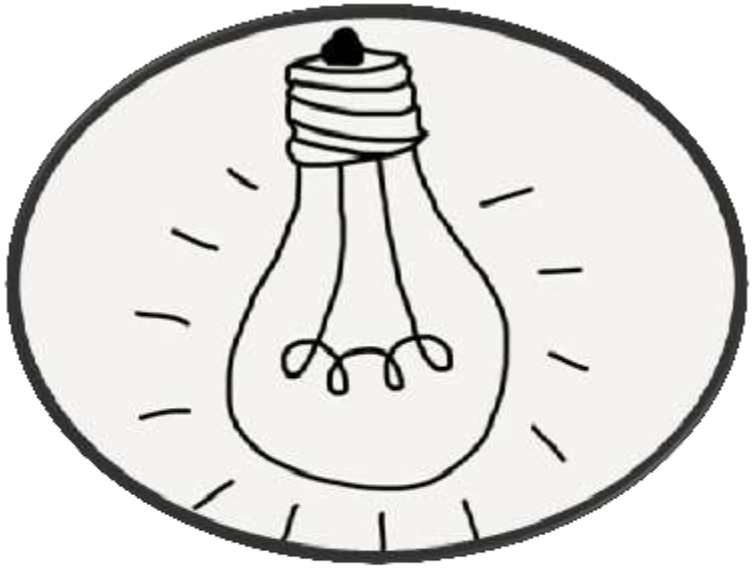


Forschungsschwerpunkte

Praxis der Anwendung der Naturwirkstoffe
Biochemie, Physiologie und Pathophysiologie
Molekulare Biophysik
Redoxregulation
Signaltransduktion
Therapien der Komplementärmedizin


Reaktivität
Resonanz
Regulation

Redoxregulation



Radikalreaktion *Signaltransduktion*

Die Resonanz erzeugt aufgrund der Reaktivität eine Regulation mit Synchronisation: Evolution von passiven zu aktiven Rhythmen

Dr. Ingrid Arnold und Prof. Dr. Gerhard Ohlenschläger

“Nur der, der sich die Gegenwart anders vorstellen kann als die existierende, verfügt über Zukunft.“

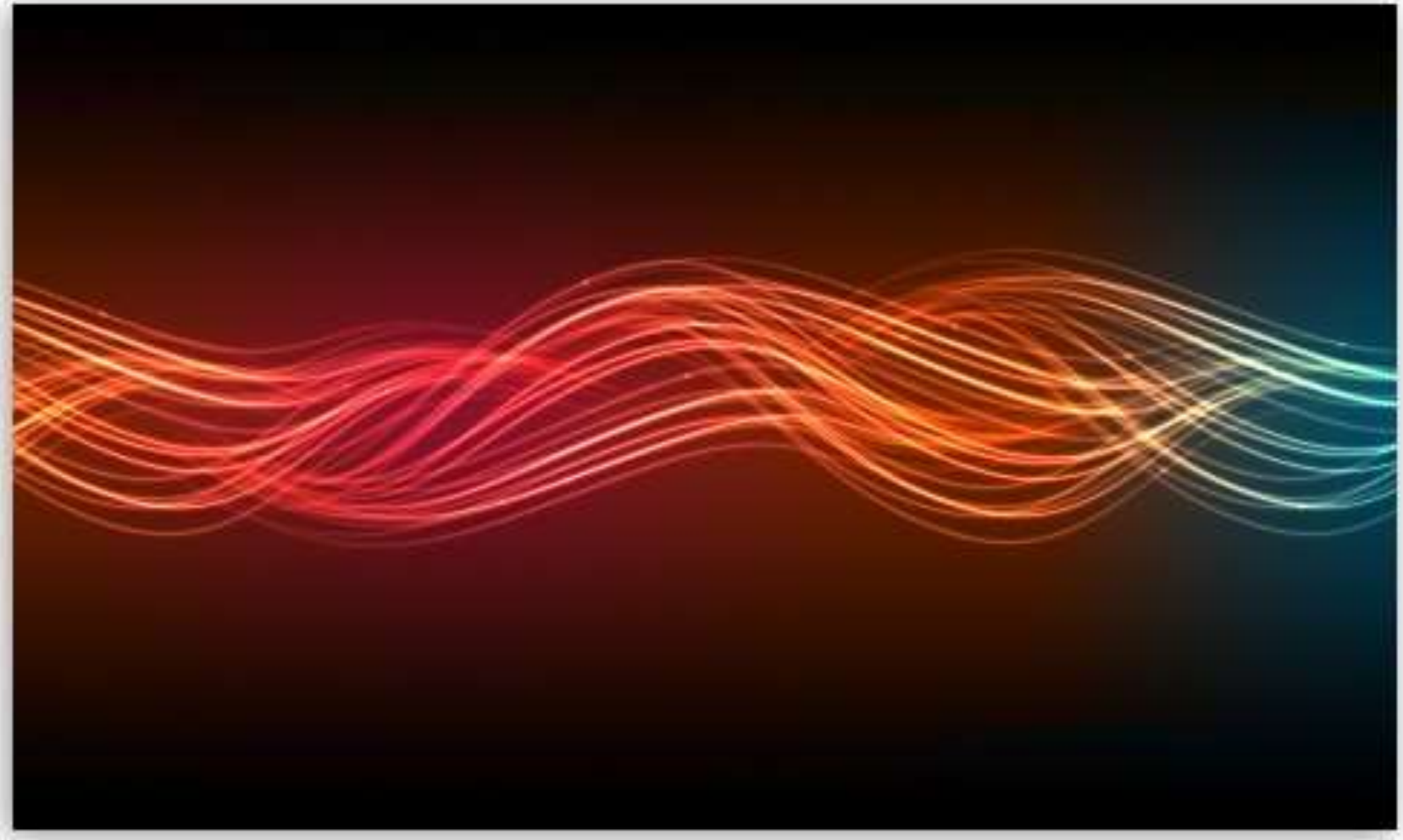
Prof. Dr. Theodor W. Adorno: Es ist Zeit

Einspruch und Reflexion

Prof. Dr. Otto Pöggeler: die existenzielle Zeit
Prof. Dr. Dr. Jürgen Schole: die essentielle Regulationsmedizin
Nobelpreisträger Prof. Dr. Dr. Ilya Prigogine: die evolutionäre Selbstorganisation
Nobelpreisträger Prof. Dr. Manfred Eigen: die nichtlineare Thermodynamik
Nobelpreisträger Profs. Drs. mult. Robert Furchtgott, Ferid Murad und Louis Ignarro: We say NO

Prof. Dr. Brigitte König: das einzigartige Mitochondrium

Bioenergetic Biomatrix Bioresonance



Master Mechanistic Modulation

Symbiotic Supersynergetic Synchronisation

Seit 1950 und 1980: Regulationsmedizin und Gesundheitsmedizin (Schole, Ohlenschläger und Uhlenbruck)

1956: Free Radical Theory of Aging (Harman)

1993: Bioenergetik (Poeggeler)

1994: Mitochondriale Medizin (Luft)

1995: SI System der Radikalreaktionen (Poeggeler)

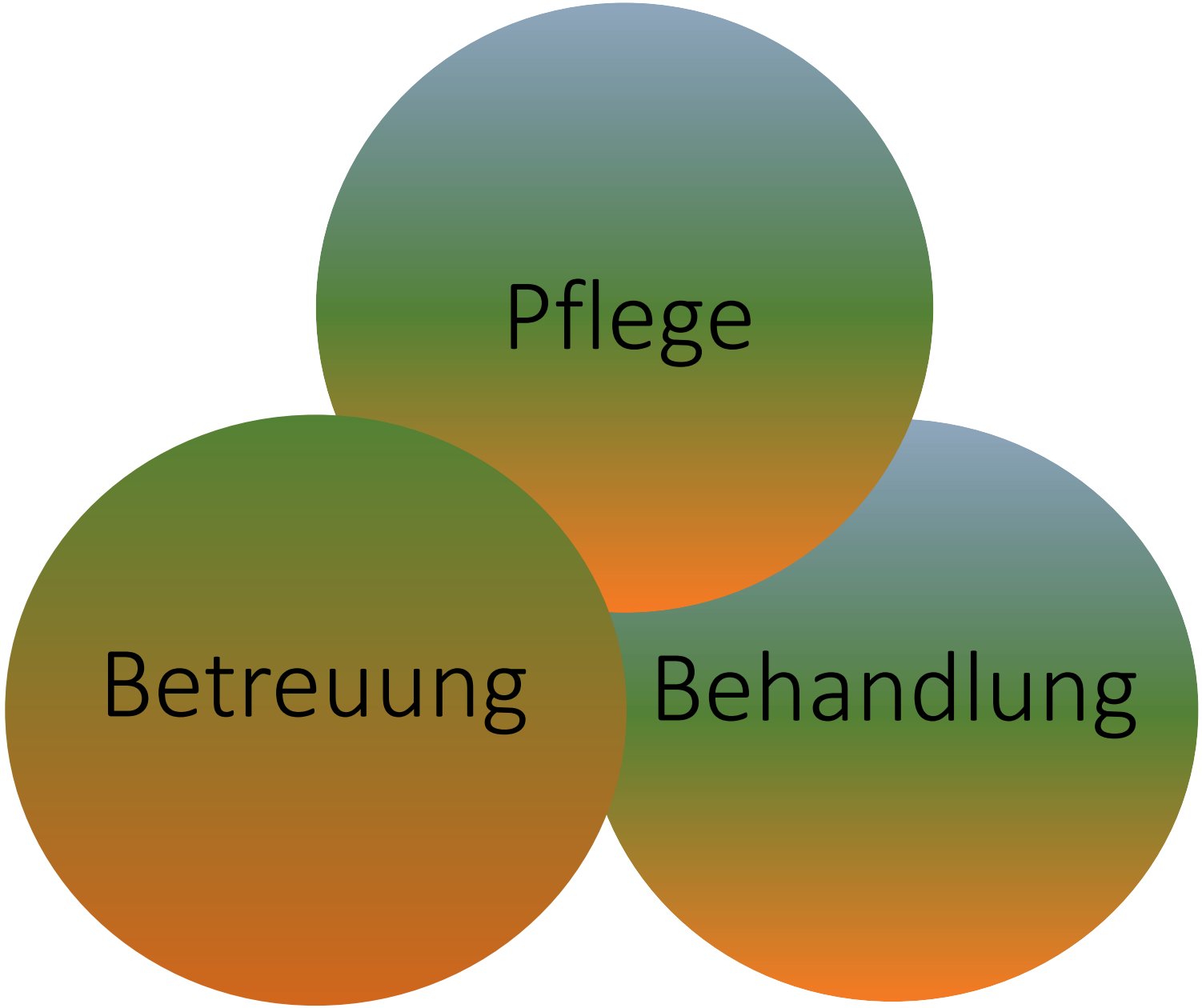
2001: Metabolische Mitochondriale Medizin (Fosslien)

2004: Metabolom und Mitochondrien als Modulatoren von Gesundheit und Altern (Poeggeler)

2014:Endobolomics (Poeggeler)

2015: Symbiotik und Biomimese mit Gesundheit durch die Biomatrix (Poeggeler)

2016: BIG – das breite individuelle Gesundheitsmanagement – als Nutritive Holistic Healthcare (Poeggeler)


Pflege
Betreuung
Behandlung

Deine Lebensmittel sollen Deine Heilmittel
und Deine Heilmittel Deine Lebensmittel sein
Hippokrates

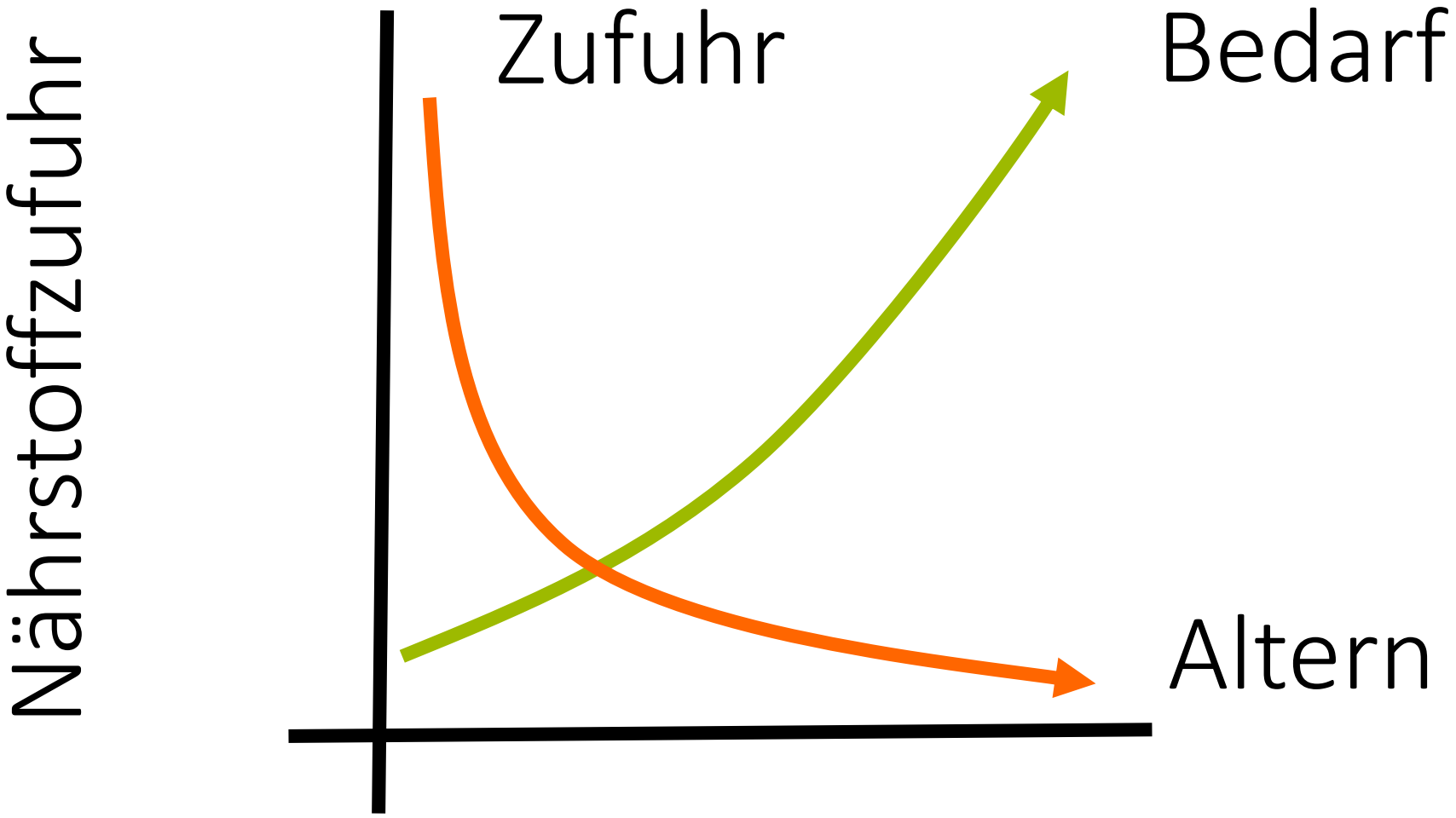
Von der orthomolekularen Medizin zur biomolekularen Gesundheitsmedizin

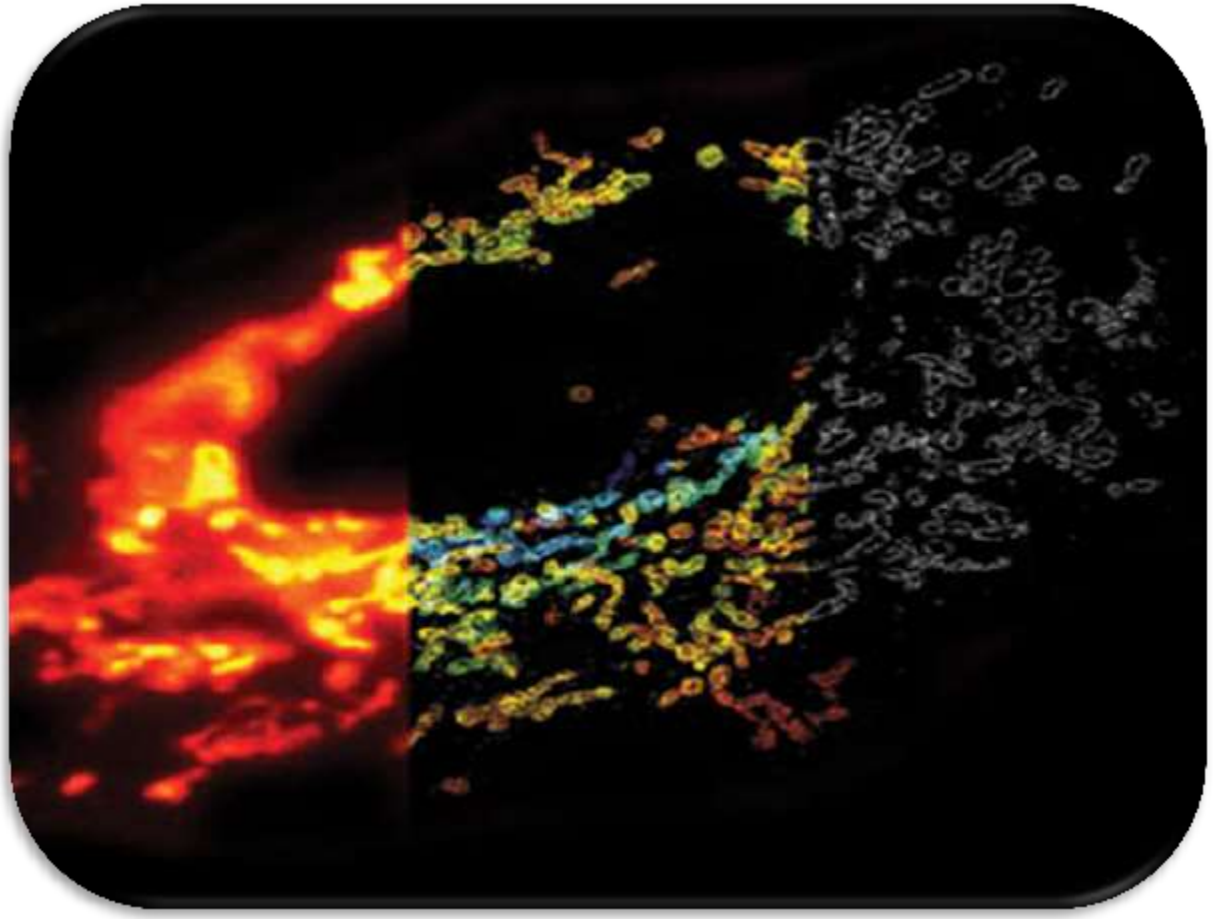
Don`t eat your body – feed your body
The orthoorganic agenda

Nährstoffergänzung aus und für die Biomatrix

Mögest Du leben solange Du willst
und es wollen solange Du lebst
Keltischer Trinkspruch

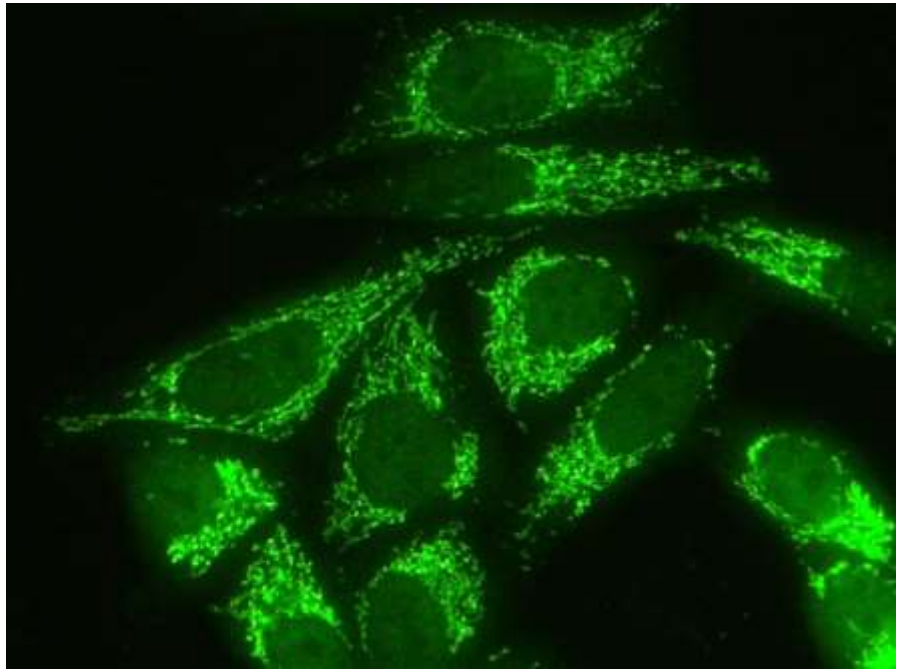
Herausforderung Altern als zentrale Aufgabe der Bioenergetik


Nährstoffzufuhr
Zufuhr
Bedarf
Altern



Quelle: Burkhard Poeggeler

Nur die bedarfsgerechte Nährstoffergänzung ermöglicht eine Erhaltung der Mitochondrien

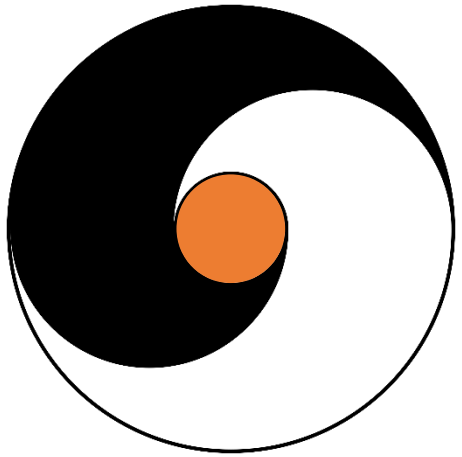


Mitochondriale Aktivität, Effizienz und Kapazität steigern

Kapazität

Regeneration

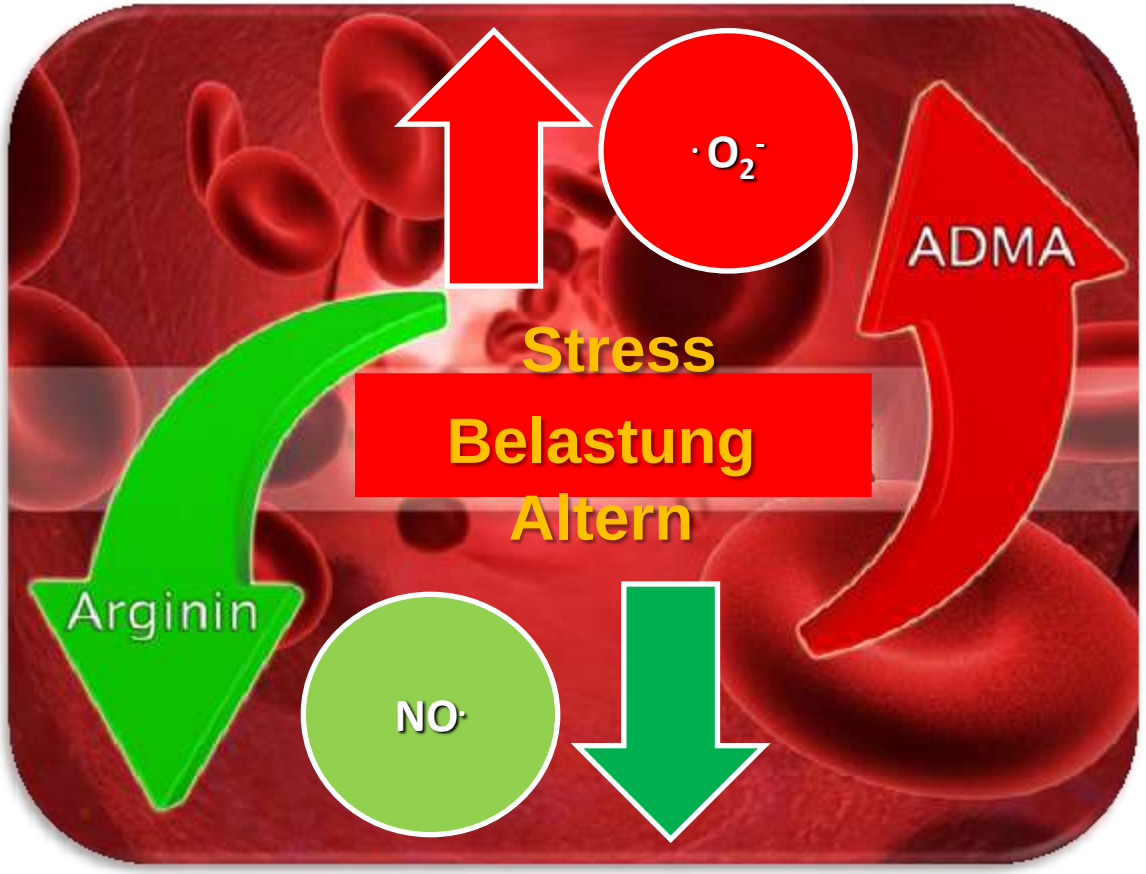
Ernährung



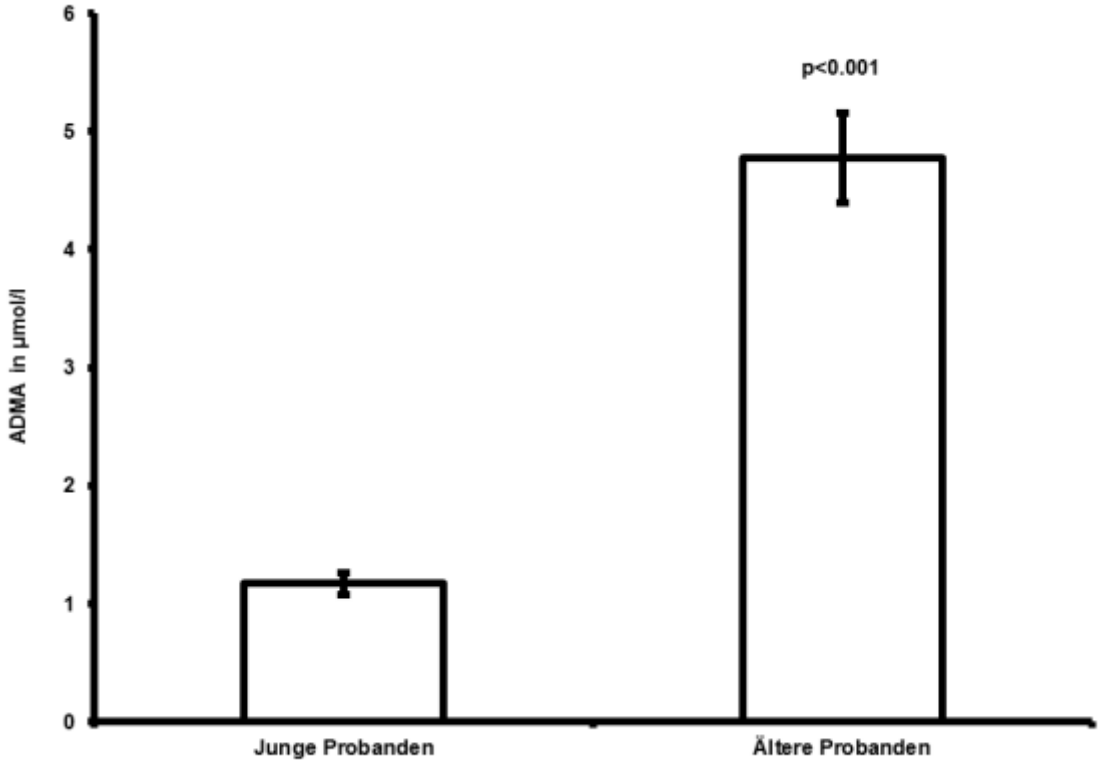
Altern

Degeneration

Belastung



Quelle: Burkhard Poeggeler



Quelle: Burkhard Poeggeler

L-Arginin

B Vitamine

Antioxidantien

neu denken

anders handeln

besser arbeiten

natürlich

ganzheitlich

dauerhaft

Zahlen …

Fakten …

Tatsachen …

Quellen: WHO, EUROSTAT, RKI

Lebenserwartung

79,7 Jahre in Deutschland

Frauen: 82,4 Jahre

Männer: 77,0 Jahre

Gesundheitsspanne

57,4 Jahre in Deutschland

Frauen: 57,7 Jahre

Männer: 57,1 Jahre

Das bedeutet 20 bis 25 Jahre

Krankheit

Leiden

Schmerzen

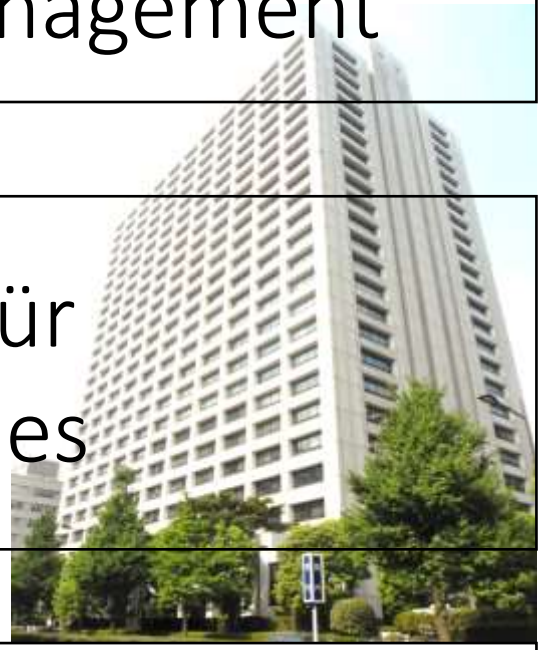
Die Gesundheitsspanne sinkt permanent in Europa
Die Gesundheitsspanne steigt in Japan

Die Schere zwischen Lebenserwartung
und Gesundheitsspanne geht immer weiter auf

Wohlhabende leben länger und gesünder
als Durchschnittsverdiener und Ärmere

Das breite individuelle Gesundheitsmanagement

Das japanische Ministerium für Gesundheit, Arbeit und Soziales



Ernährung

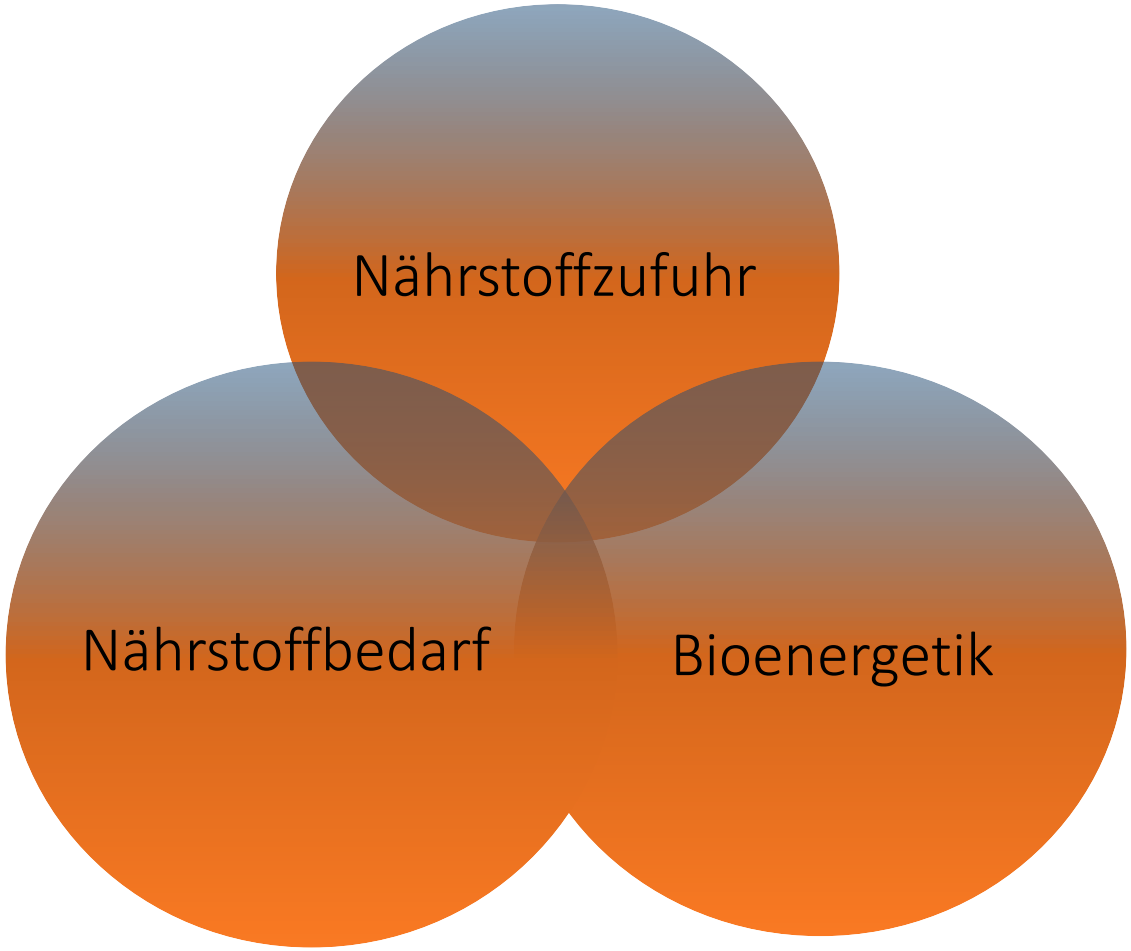
Aktivität

Verhalten

https://www.bundestag.de/blob/412864/e1e21b33e7bfd7f8eca1bcf9513add5b/wd-9-008-16-pdf-data.pdf

Burkhard Poeggeler, Quellen: Anfrage an die Bundesregierung vom Januar 2016 und Wikipedia Commons

Belastung
Umweltbedingte Exposition
Altern
Persönliche Entwicklung
Stress
Epigenetische Disposition


Nährstoffzufuhr
Nährstoffbedarf
Bioenergetik

Aktivität
Ernährung
Verhalten

Fatale

Fehleinschätzungen

und Folgen für die Bioenergetik

1

Unsere Ernährung ist gesund.

Unsere Ernährung macht krank.

Nachweis: Unsere Gesundheitsspanne sinkt.

Kosten

Fett	Salz	Zucker
2.9 Mia	5.3 Mia	8.6 Mia

Gärung

versus

Atmung

Review

openheart

Added sugars drive nutrient and energy deficit in obesity: a new paradigm

James J DiNicolantonio,[1] Amy Berger[2]

To cite: DiNicolantonio JJ, Berger A. Added sugars drive nutrient and energy deficit in obesity: a new paradigm. *Open Heart* 2016;3:e000469. doi:10.1136/openhrt-2016-000469

Received 8 May 2016
Accepted 7 July 2016

ABSTRACT

Obesity has traditionally been thought of as a state of caloric imbalance, where the intake of calories exceeds the expenditure or 'burning' of calories. However, a more nuanced appreciation for the complex biochemistry and physiology of cellular energy generation suggests that obesity is a state of hormonal imbalance causing increased shunting of food energy into adipose tissue for storage, resulting in decreased satiety and ultimately leading to increased caloric intake. Adding to this hypothesis, we propose that obesity is also a state of nutrient and energy deficit, leading to decreased fatty acid mobilisation and oxidation, the result of which may be a natural disinclination towards physical activity. Added sugars (sucrose, a.k.a. table sugar and high-fructose corn syrup) may provide energy (4 kcal/g) but at current intakes they do not facilitate—and may even hinder—the production of energy. Not only do added sugars displace nutritionally superior foods in the diet, but they may also deplete nutrients from other foods that have been consumed, as well as from body stores, in order to enable their proper oxidation and liberate their calories as energy. Additionally, the consumption of added sugars damages the mitochondria and hence impairs energy generation. Moreover, overconsuming added sugars may result in a kind of 'internal starvation' (via leptin and insulin resistance) leading to further hunger signals in the body. Added sugars promote nutrient and energy deficit and through this novel pathway promote obesity.

stress).[1] Because of this refinement, added sugars now behave like drug-like substances.[2 3] Although edible, added sugars cannot be considered a 'food', nor can their consumption be equated to eating foods that contain natural amounts of sugar, but which also provide fibre, vitamins, minerals and other phytonutrients that combat oxidative stress produced by the small amounts of fructose present. As the 'dose makes the poison', the food industry has made it possible for consumers to easily 'overdose' on added sugars, making this an issue of concern for public health worldwide, wherever there is significant consumption of processed, highly refined and sugar-dense foods.

Added sugars are not food

> Sugar is not among the recommended foods. Its recent rationing will not provoke a hardship, for sugar supplies nothing in nutrition but calories, and *the vitamins provided by other foods are sapped by sugar to liberate these calories.*[4] (Emphasis added) (Wilder—*Handbook of Nutrition*)

The definition of food is, 'Material that contains essential nutrients, which are assimilated by an organism to produce energy, stimulate growth and maintain life'.[5] Many types of

Glucoseaufnahme

Glucoseverwertung

Glucoseverbrennung

2

Die allgemeine Bevölkerung ist gesund.

Die allgemeine Bevölkerung ist krank.

Nachweis: Der Arzneimittelkonsum nimmt zu.

1992
2004
2016

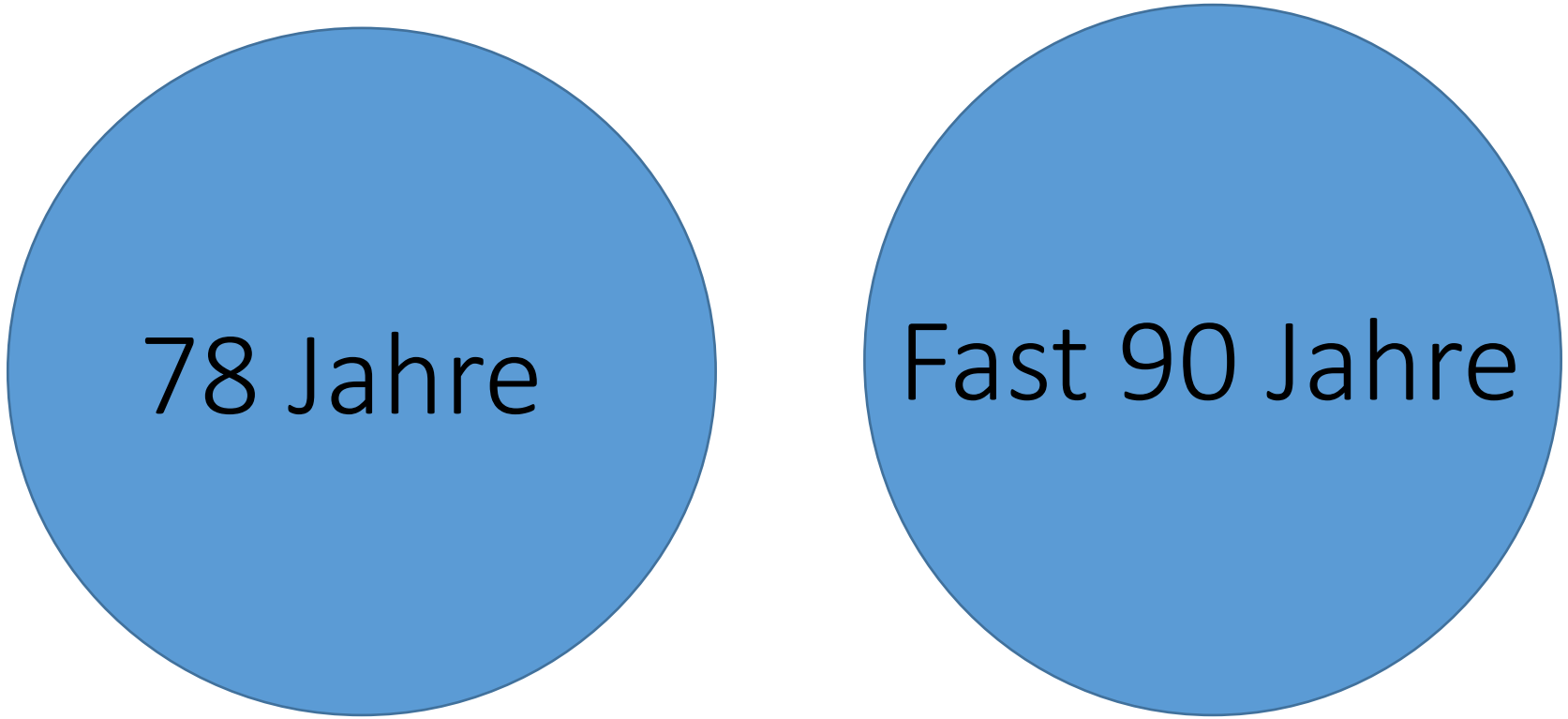
3

Kosten senken geht nur durch Einsparen.

Nutzen ermöglicht Kosten dauerhaft zu senken.

Nachweis: Das japanische Gesundheitswesen hat Erfolg.

Japan



Gesundheitsspanne Lebenserwartung

Die

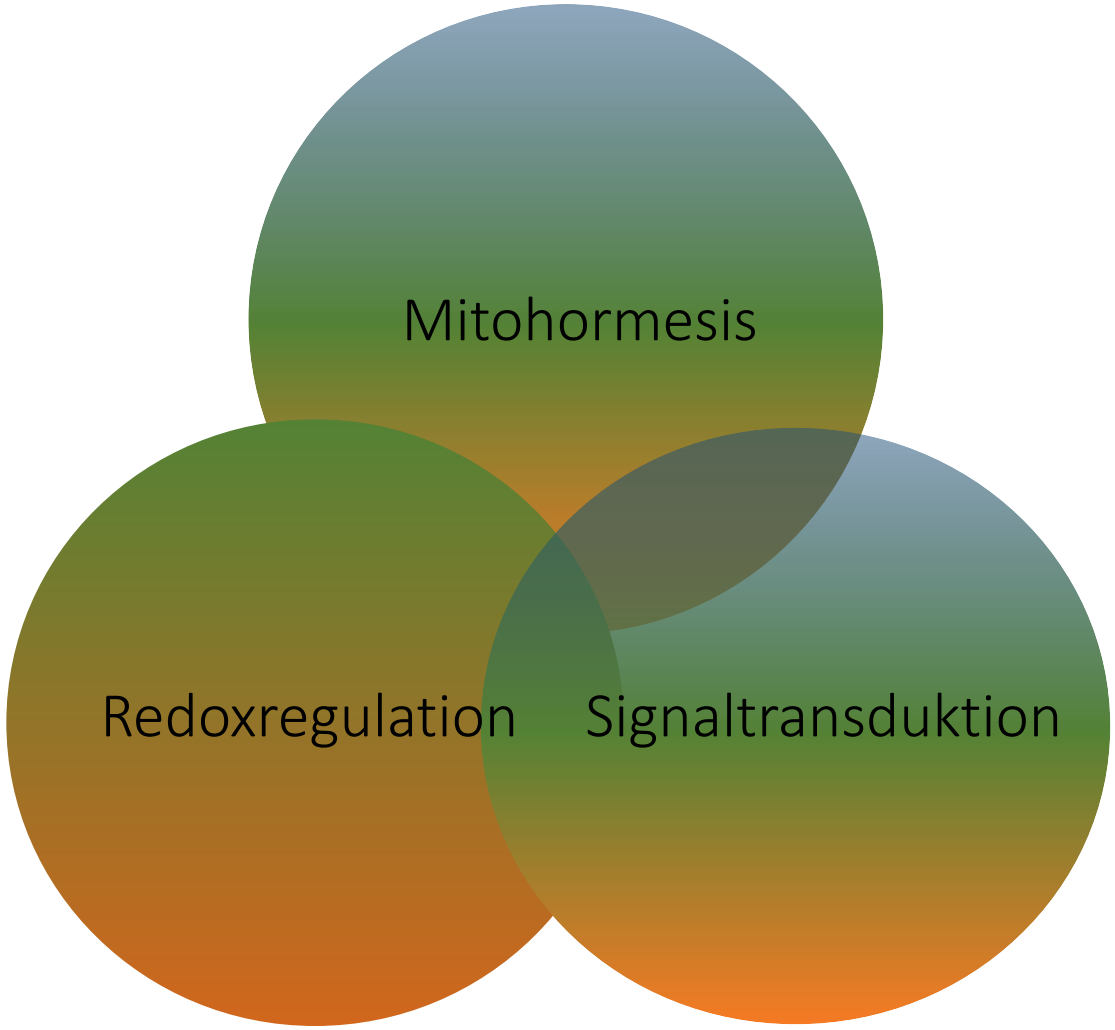
Herausforderung

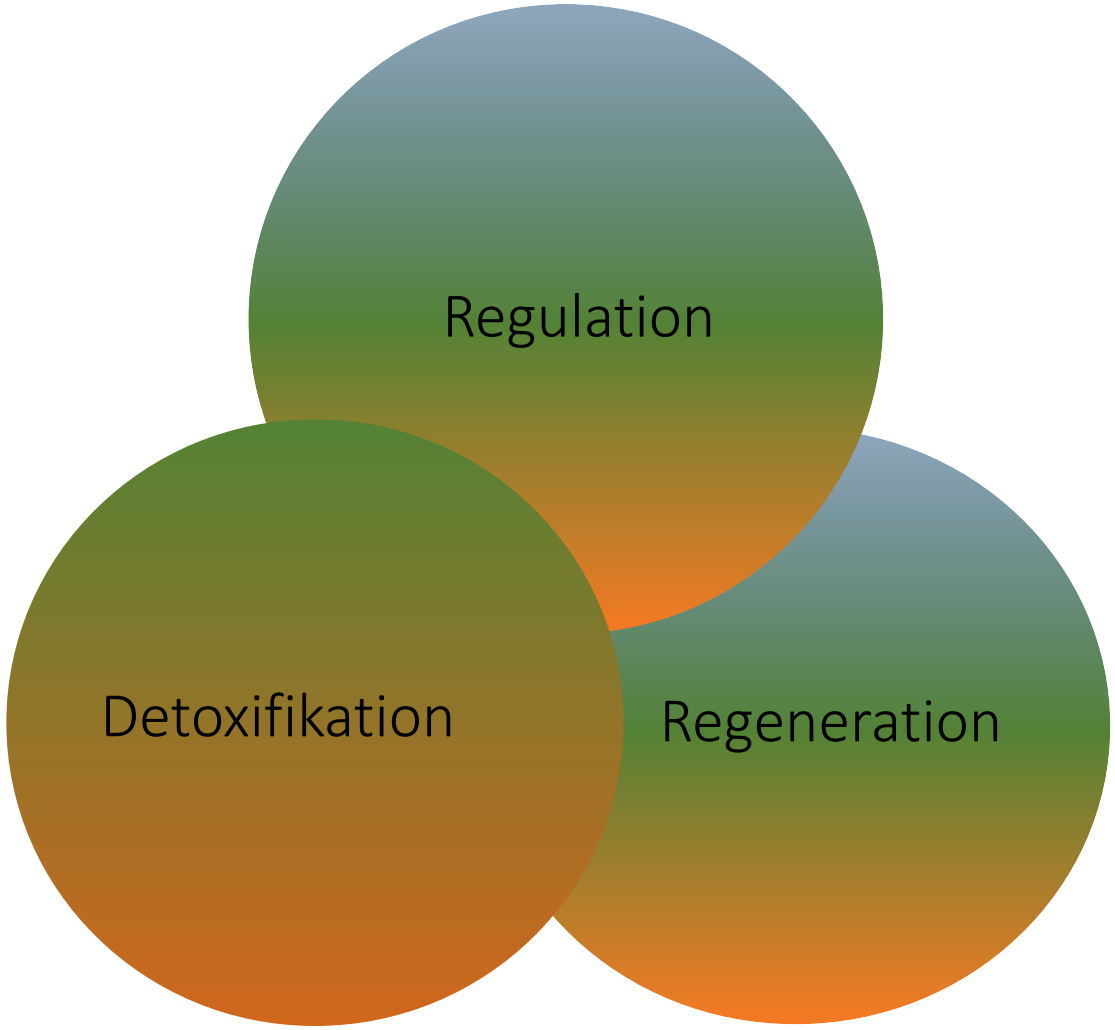
Gesund Altern

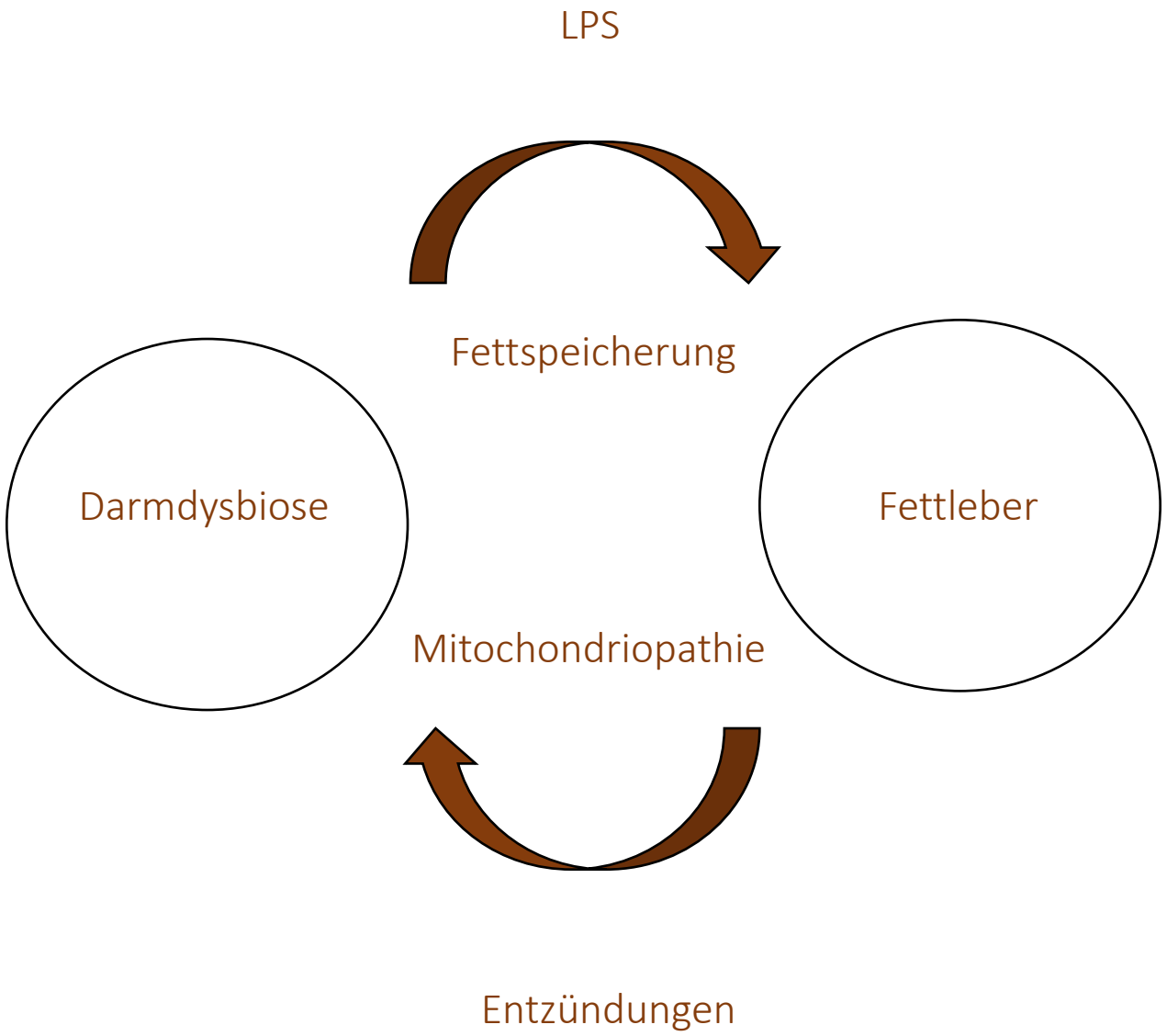
Der erfolgreiche Ansatz

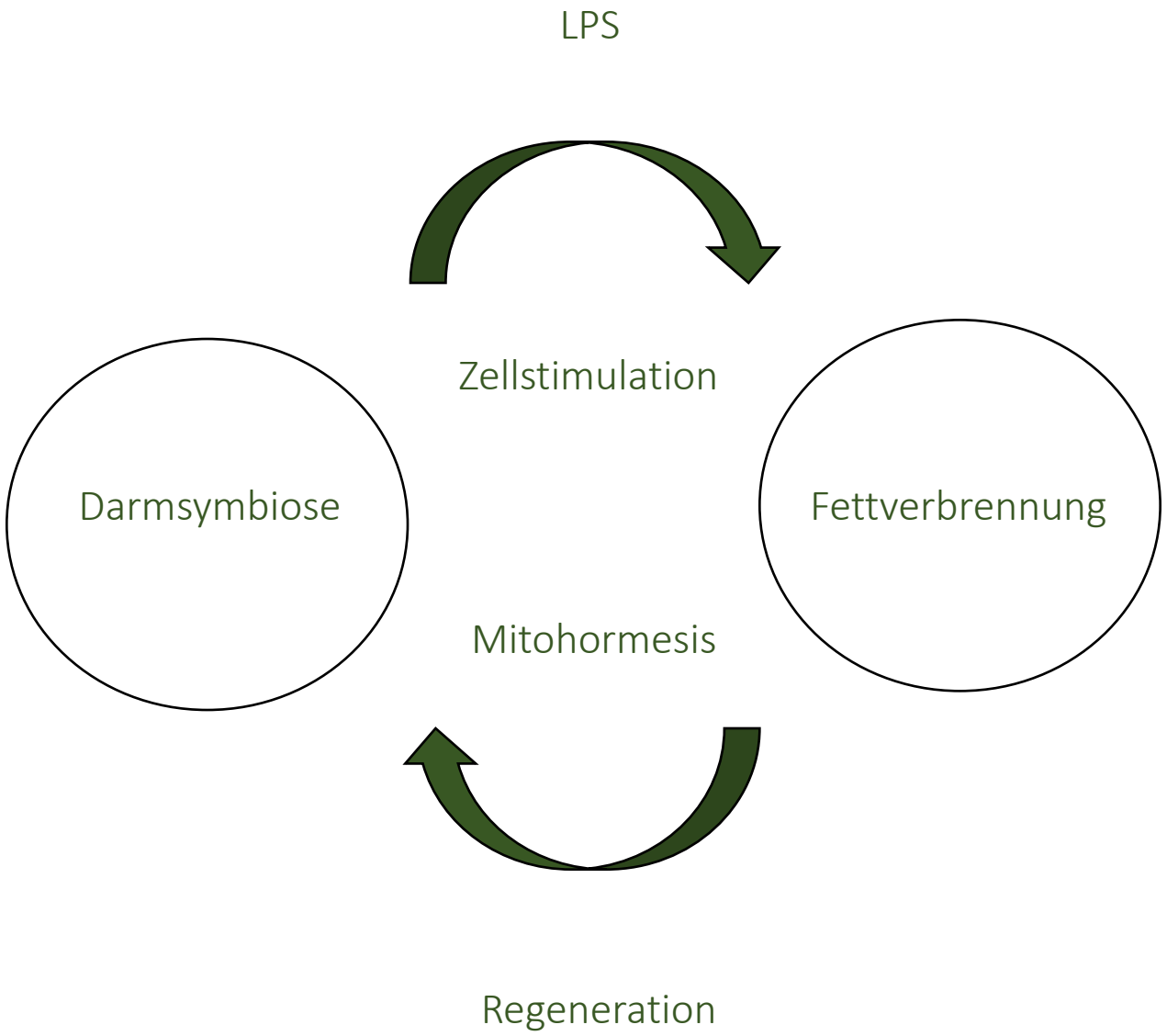
Bioenergetik und

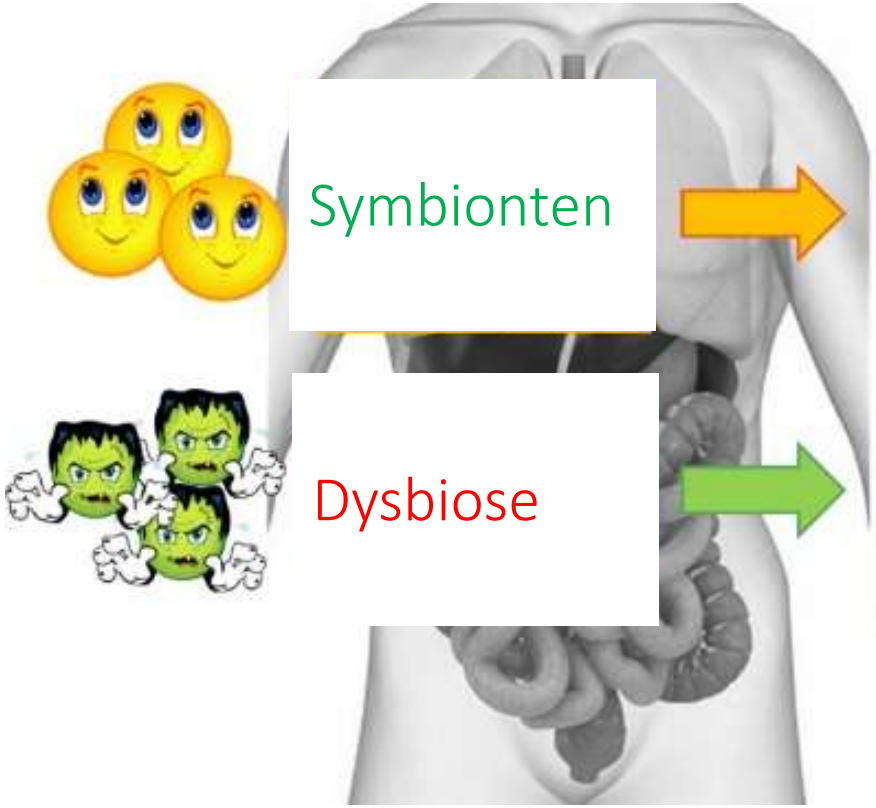
Regeneration


Mitohormesis
Redoxregulation
Signaltransduktion


Regulation
Detoxifikation
Regeneration


LPS
Fettspeicherung
Darmdysbiose
Fettleber
Mitochondriopathie
Entzündungen


LPS
Zellstimulation
Darmsymbiose
Fettverbrennung
Mitohormesis
Regeneration



Symbionten → Gesundheit

Belastung

Dysbiose → Krankheit

Alterung

Enteric connective tissue	increased overall connective tissue content altered composition of collagen types increased cross-linking of collagen fibers increased elastin fibers ('elastosis coli') disturbed connective tissue metabolism
Enteric musculature	thickening of circular and longitudinal muscle ('myochosis coli') deranged architecture of muscle fibers focal loss of myofilaments fibrotic degeneration
Enteric nervous system	oligo-neuronal hypoganglionosis imbalance of neurotransmitters/receptors deficiency in neurotrophic factors nerve fiber remodeling increased pain-mediating neurotransmitter systems loss of interstitial cells of Cajal (intestinal pacemakers)
In vivo intestinal motility	increased intraluminal pressure increased motility index increased high-amplitude propagated contractions retrograde propagations visceral hypersensitivity
In vitro intestinal motility	altered spontaneous contractility pattern decreased relaxation due to impaired nitrergic input increased contraction due to enhanced cholinergic input

Wedel et al., 2015

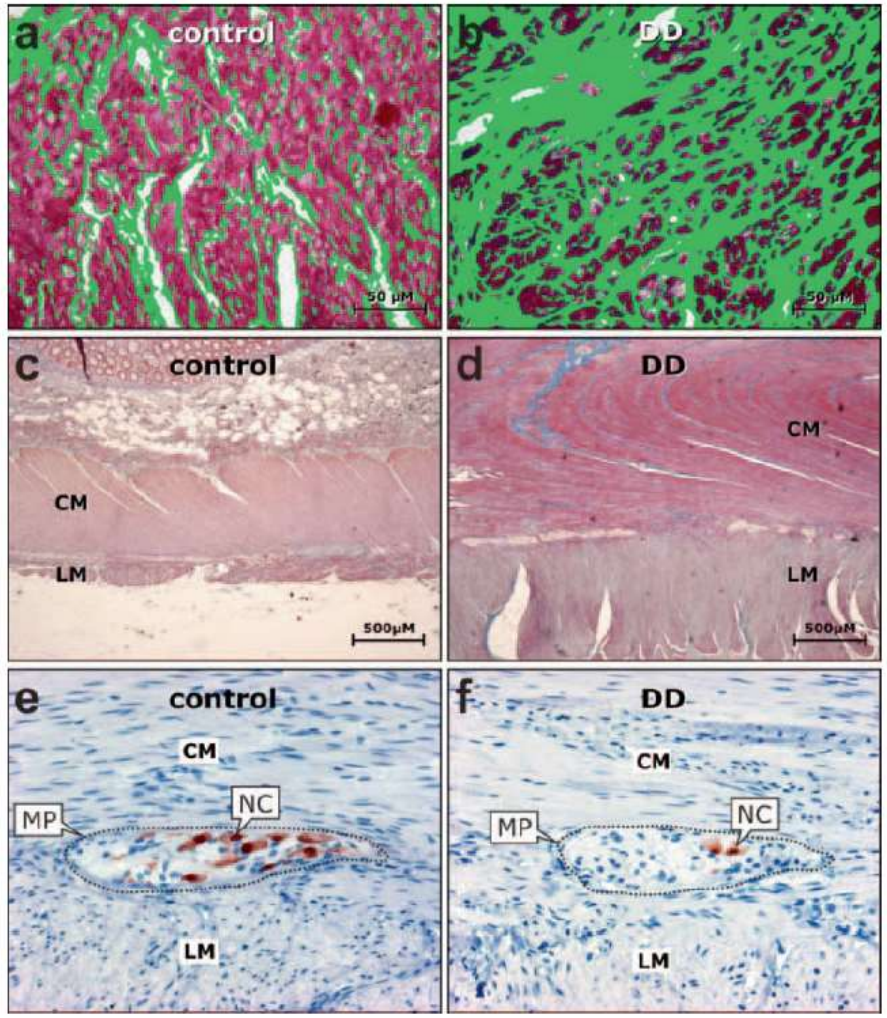
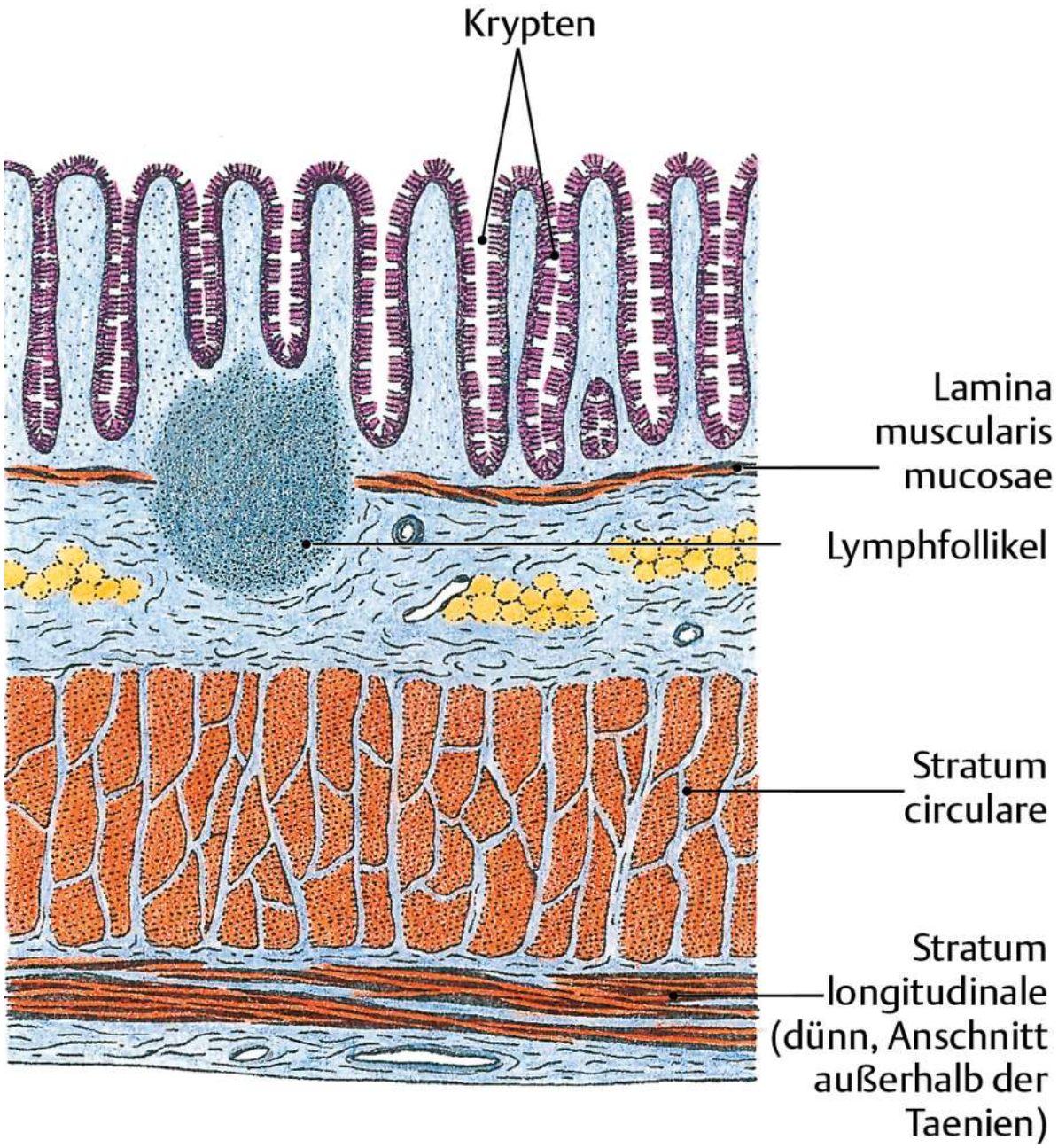


Fig. 2. Alterations of the **a, b** enteric connective tissue, **c, d** enteric musculature, and **e, f** enteric nervous system in diverticular disease (DD) compared to controls. Within the muscle layers (red colored tissue), the relative portion of connective tissue (green colored area) is significantly increased in **b** DD compared to **a** controls. Both the circular (CM) and longitudinal (LM) muscle layer are considerably thickened in **d** DD compared to **c** controls. Enteric ganglia of the myenteric plexus (MP) located between the CM and LM exhibit a significantly decreased nerve cell (NC) density in **f** DD compared to **e** controls. **a, b** Azan staining with false-color conversion of connective tissue elements (modified from [27]); **c, d** Azan staining, (modified from [27]); **e, f** anti-Hu C/D immunohistochemistry, hematoxylin counterstain (modified from [33]).

Der Darm macht dicht

Theorie …



Copyright
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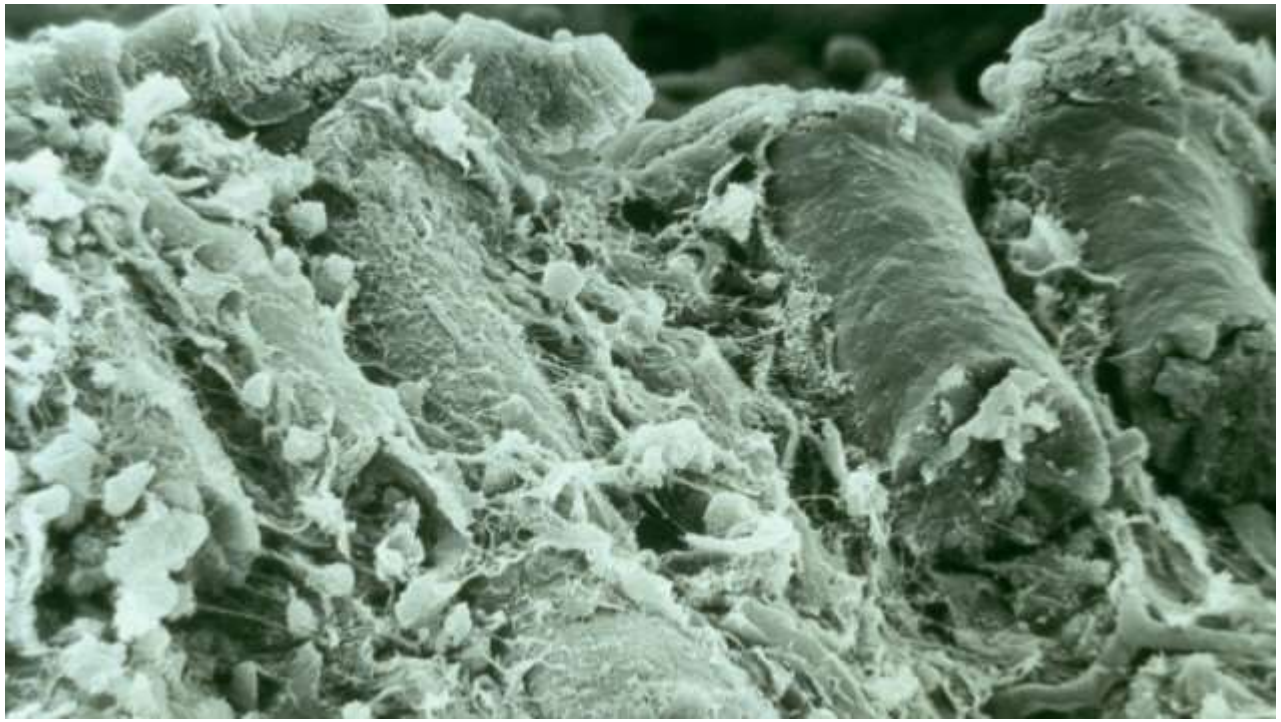
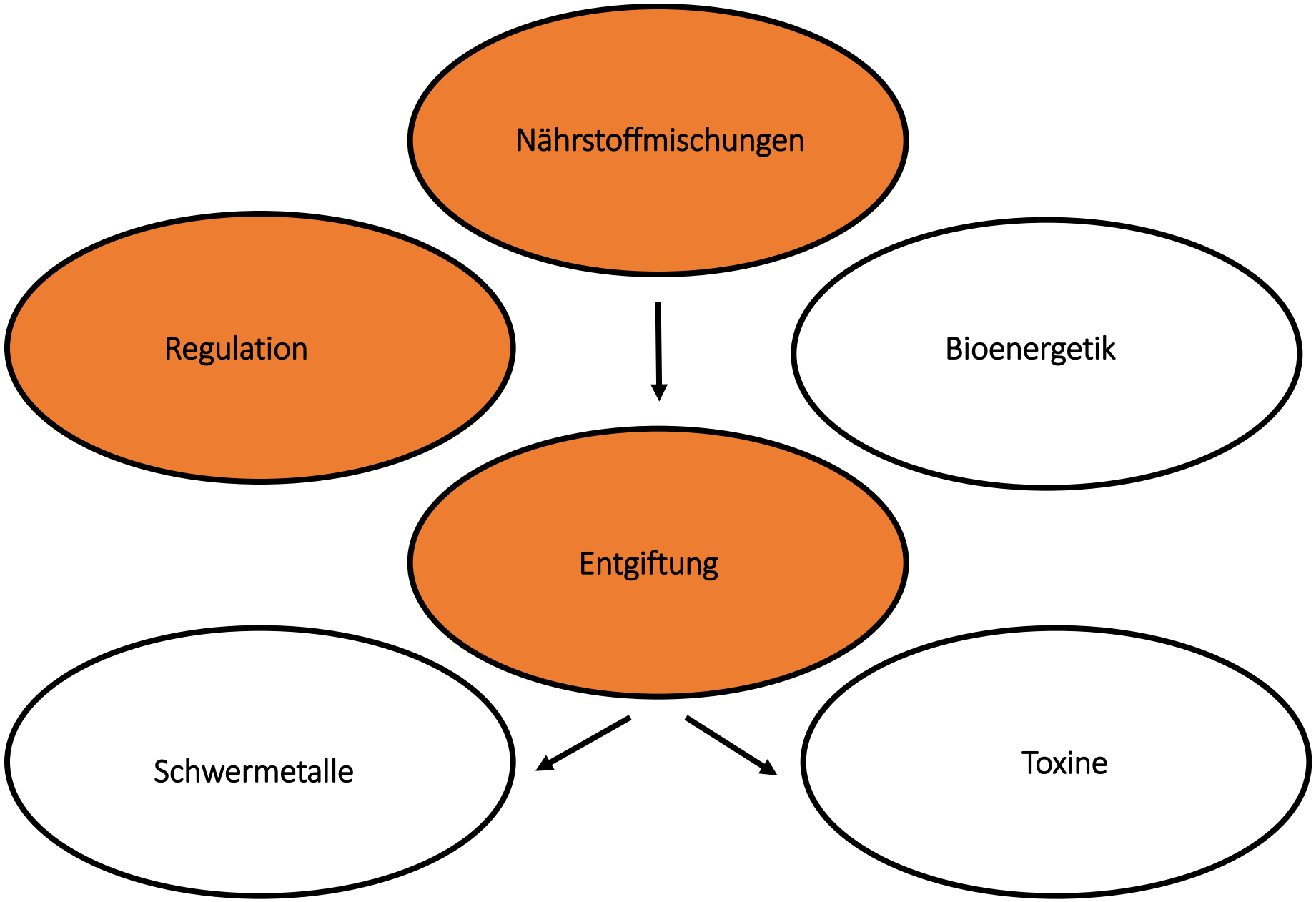


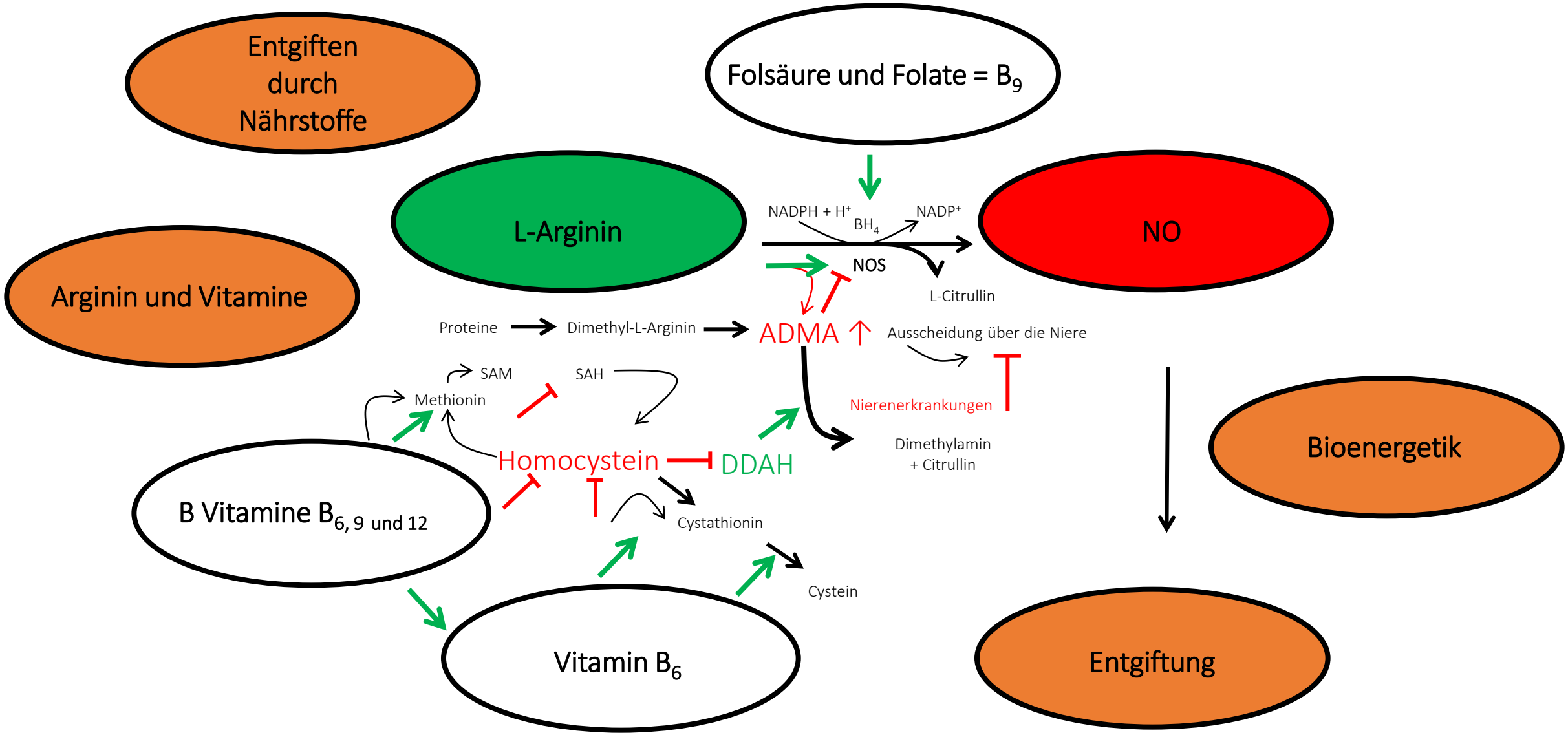
… und Realität

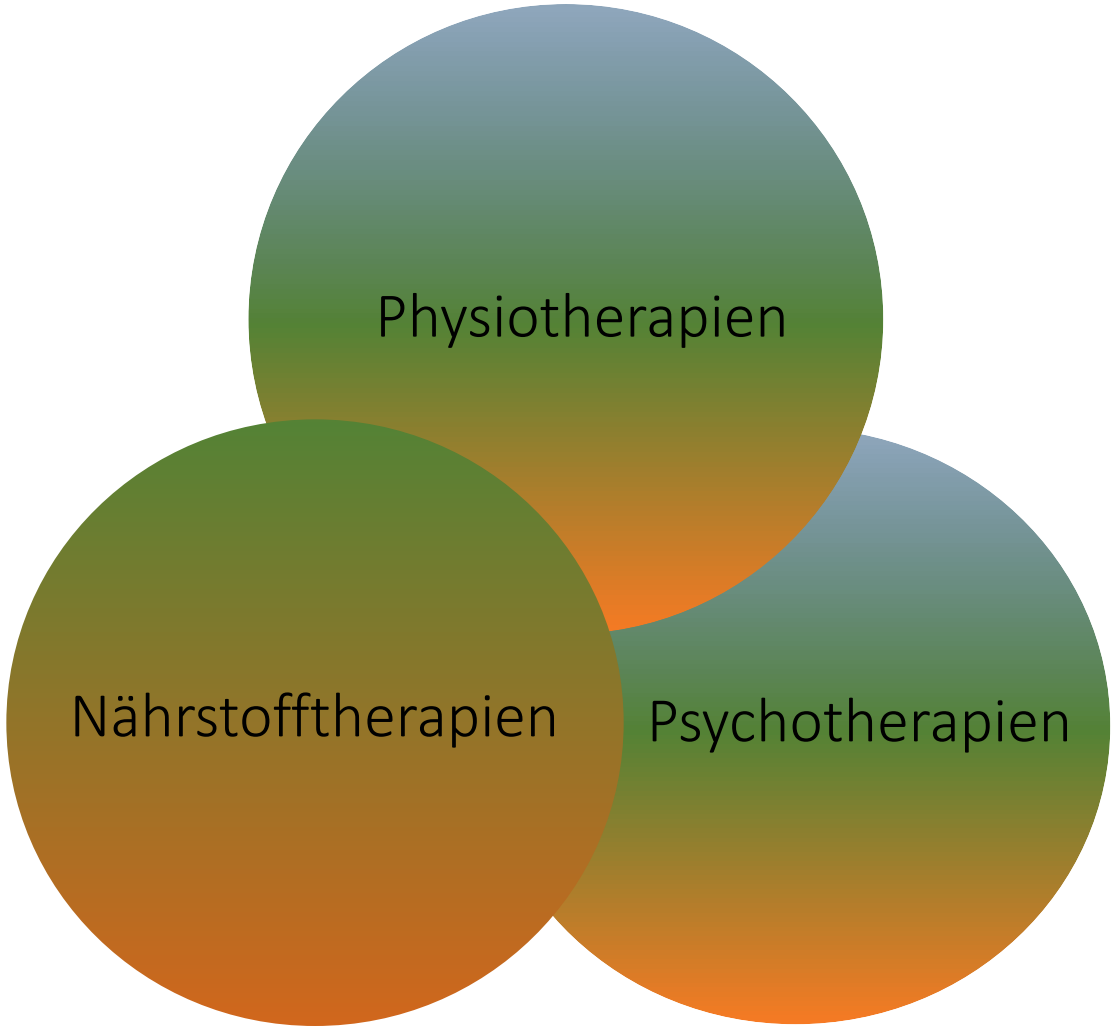
Zusammenbruch

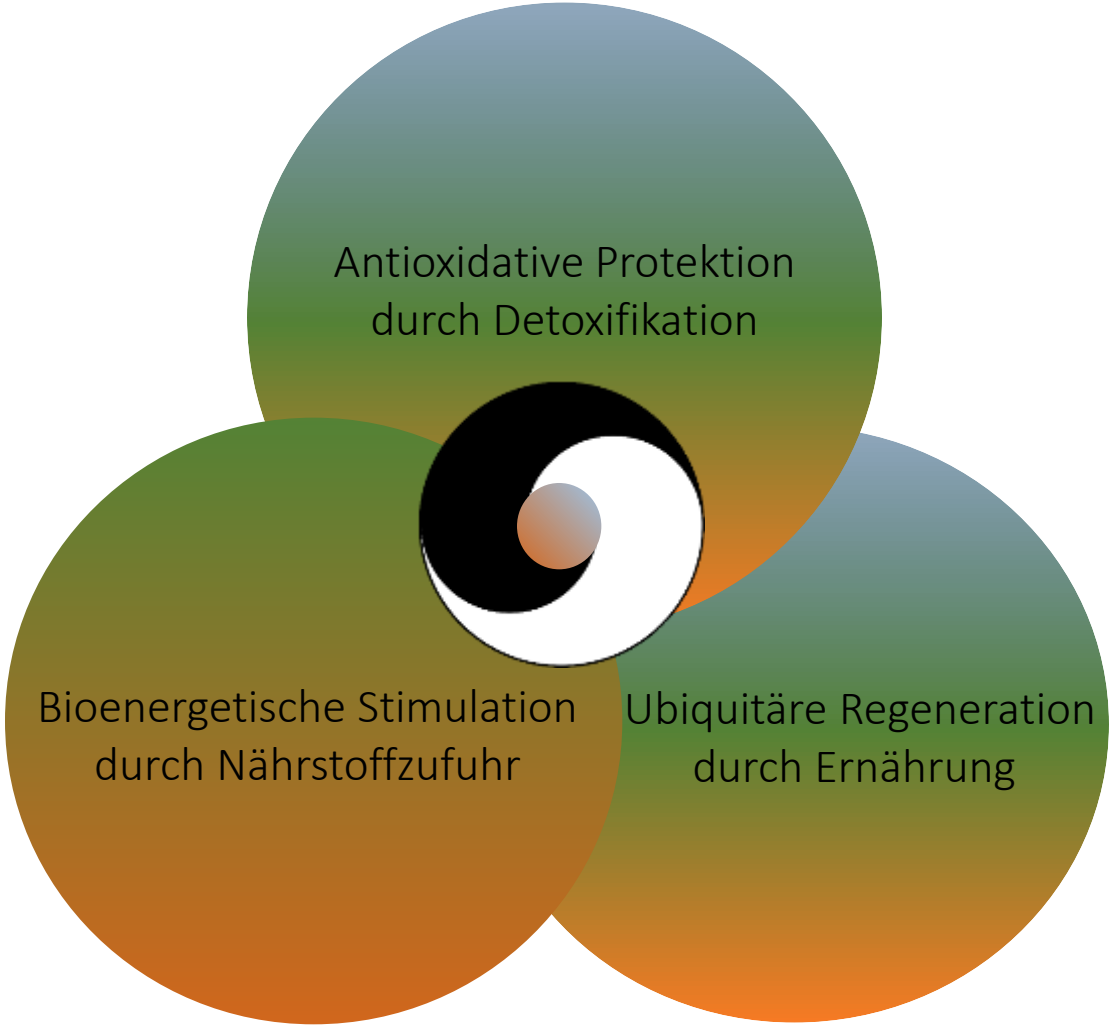
des

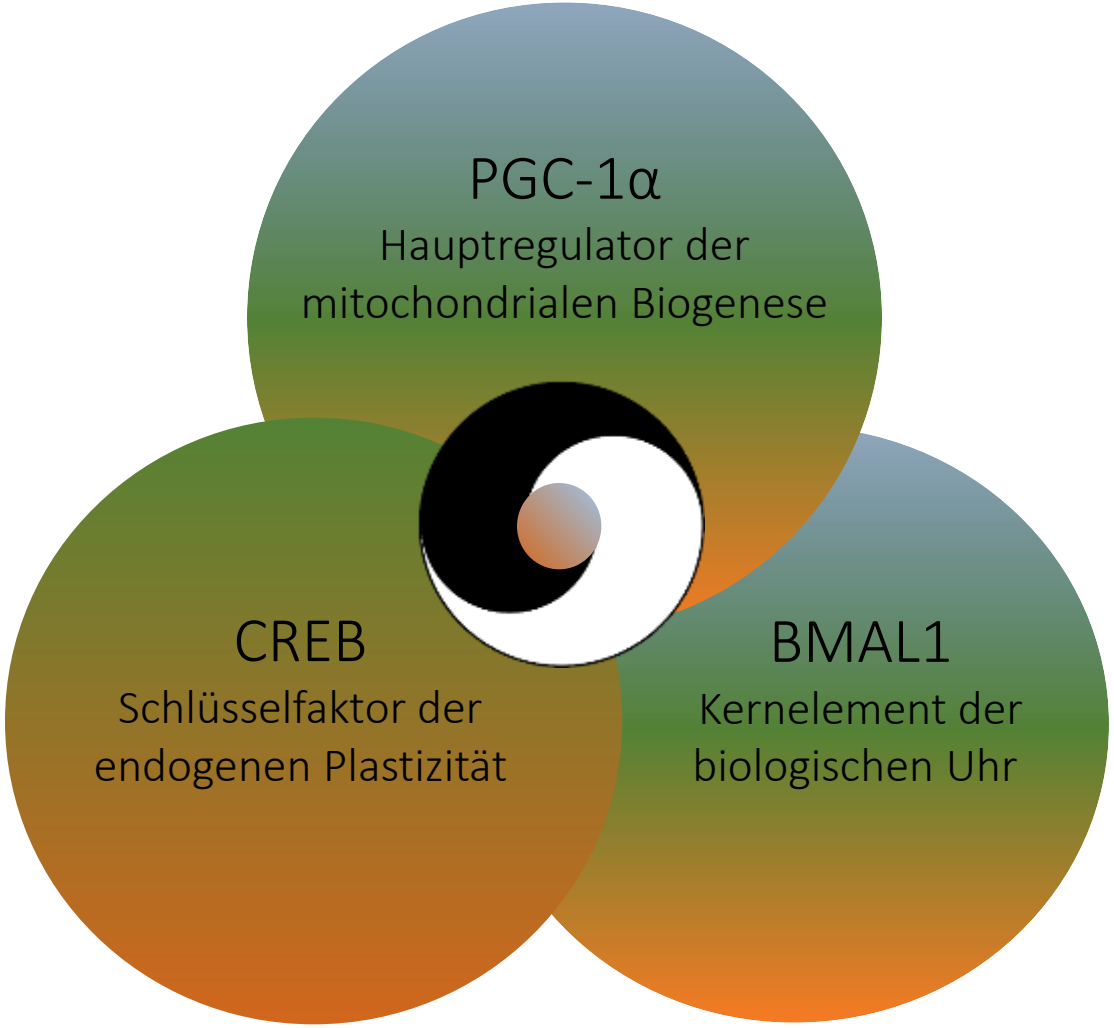
Energiestoffwechsels

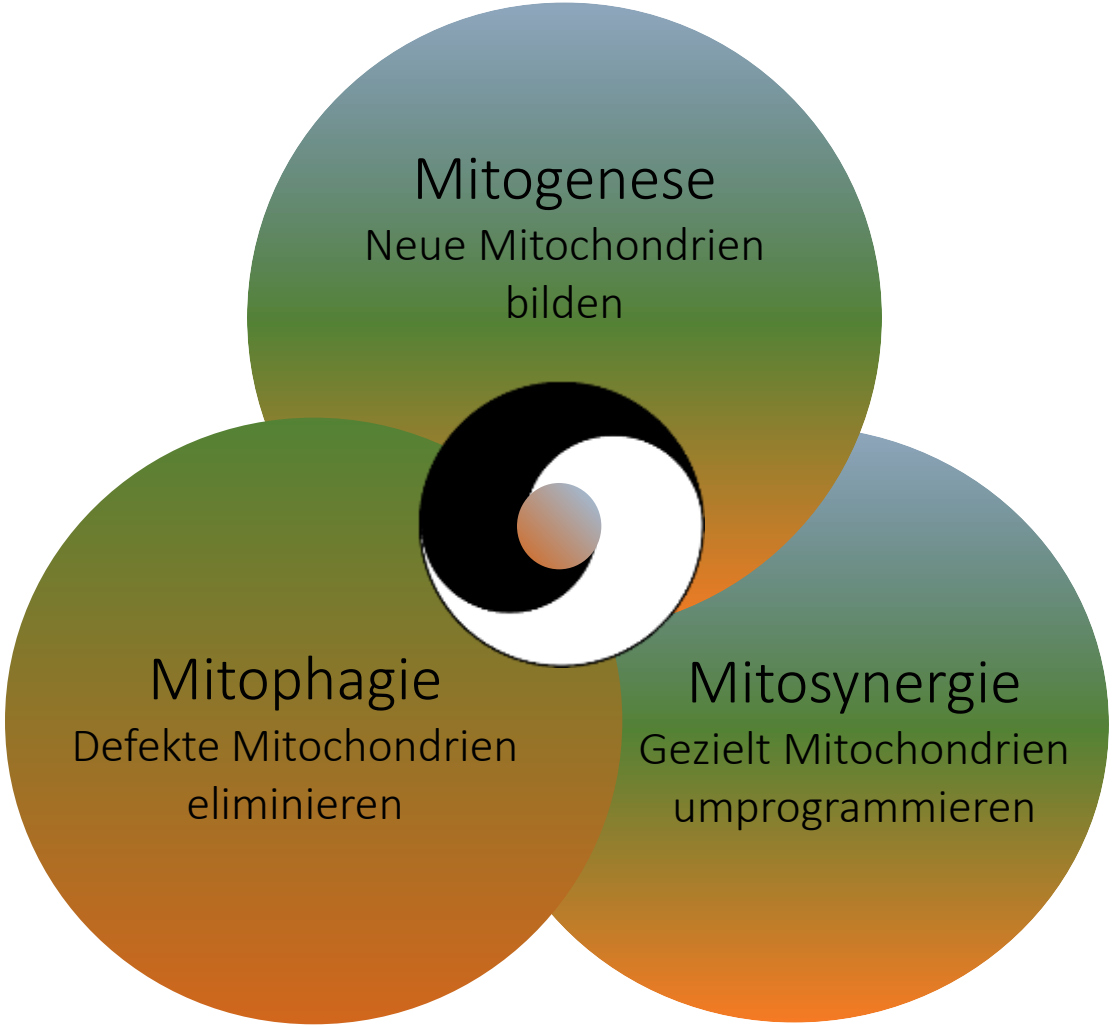

Nährstoffmischungen
Regulation
Bioenergetik
Entgiftung
Schwermetalle
Toxine


Entgiften durch Nährstoffe
Folsäure und Folate = B_9
L-Arginin
NO
Arginin und Vitamine
NADPH + H^+
BH_4
$NADP^+$
NOS
L-Citrullin
Proteine
Dimethyl-L-Arginin
ADMA ↑
Ausscheidung über die Niere
SAM
SAH
Methionin
Nierenerkrankungen
Homocystein
DDAH
Dimethylamin + Citrullin
Bioenergetik
B Vitamine $B_{6, 9 \text{ und } 12}$
Cystathionin
Cystein
Vitamin B_6
Entgiftung


Physiotherapien
Nährstofftherapien
Psychotherapien


Antioxidative Protektion
durch Detoxifikation
Bioenergetische Stimulation
durch Nährstoffzufuhr
Ubiquitäre Regeneration
durch Ernährung


PGC-1α
Hauptregulator der mitochondrialen Biogenese
CREB
Schlüsselfaktor der endogenen Plastizität
BMAL1
Kernelement der biologischen Uhr


Mitogenese
Neue Mitochondrien bilden
Mitophagie
Defekte Mitochondrien eliminieren
Mitosynergie
Gezielt Mitochondrien umprogrammieren

Nährstoffergänzung

aus und für

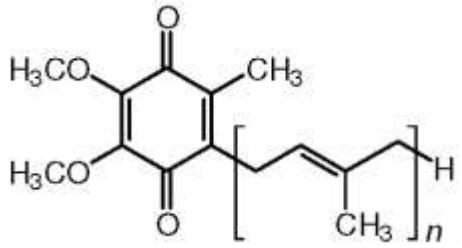
die Biomatrix

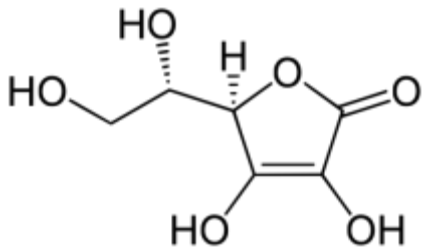
Bioenergetische Stimulation

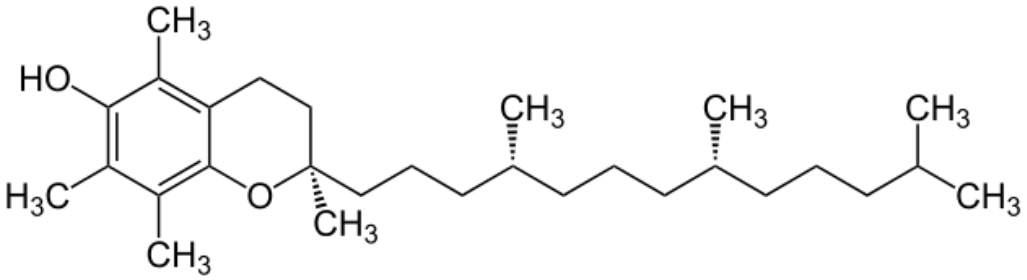
Antioxidative Protektion

Ubiquitäre Regeneration

Eradikation mit Protektion und Detoxifikation







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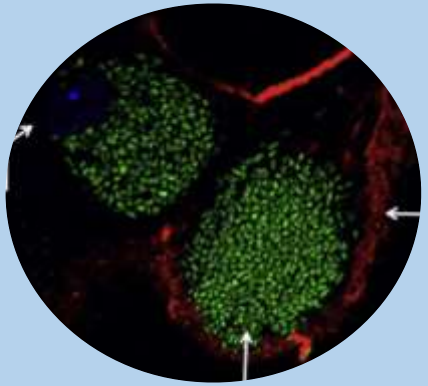


Alzheimer's disease - a neurospirochetosis. Analysis of the evidence following Koch's and Hill's criteria

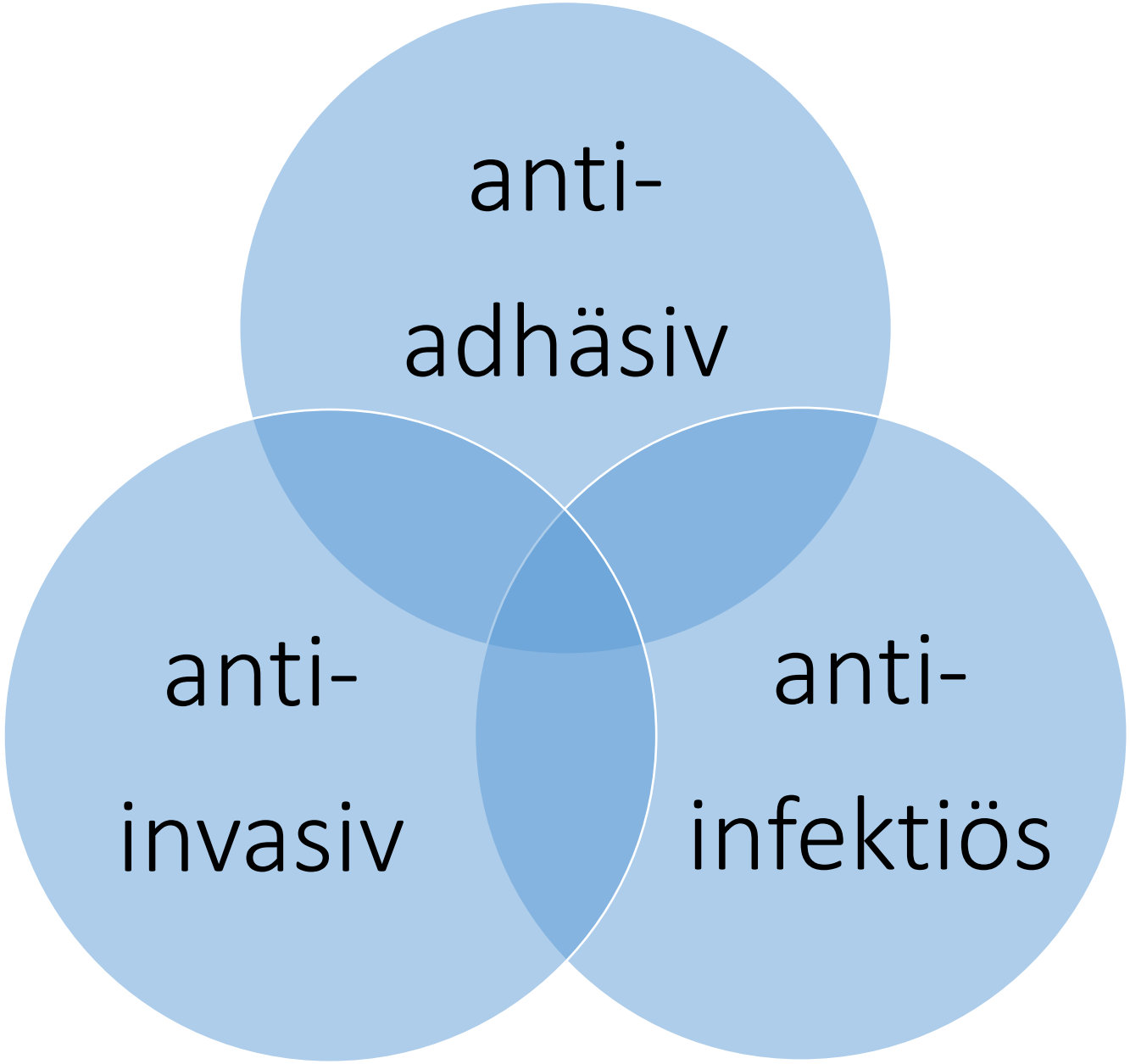
Judith Miklossy

Abstract

It is established that chronic spirochetal infection can cause slowly progressive dementia, brain atrophy and amyloid deposition in late neurosyphilis. Recently it has been suggested that various types of spirochetes, in an analogous way to *Treponema pallidum*, could cause dementia and may be involved in the pathogenesis of Alzheimer's disease (AD). Here, we review all data available in the literature on the detection of spirochetes in AD and critically analyze the association and causal relationship between spirochetes and AD following established criteria of Koch and Hill. The results show a statistically significant association between spirochetes and AD ($P = 1.5 \times 10^{-17}$, OR = 20, 95% CI = 8-60, N = 247). When neutral techniques recognizing all types of spirochetes were used, or the highly prevalent periodontal pathogen Treponemas were analyzed, spirochetes were observed in the brain in more than 90% of AD cases. *Borrelia burgdorferi* was detected in the brain in 25.3% of AD cases analyzed and was 13 times more frequent in AD compared to controls. Periodontal pathogen Treponemas (*T. pectinovorum, T. amylovorum, T. lecithinolyticum, T. maltophilum, T. medium, T. socranskii*) and *Borrelia burgdorferi* were detected using species specific PCR and antibodies. Importantly, co-infection with several spirochetes occurs in AD. The pathological and biological hallmarks of AD were reproduced *in vitro* by exposure of mammalian cells to spirochetes. The analysis of reviewed data following Koch's and Hill's postulates shows a probable causal relationship between neurospirochetosis and AD. Persisting inflammation and amyloid deposition initiated and sustained by chronic spirochetal infection form together with the various hypotheses suggested to play a role in the pathogenesis of AD a comprehensive entity. As suggested by Hill, once the probability of a causal relationship is established prompt action is needed. Support and attention should be given to this field of AD research. Spirochetal infection occurs years or decades before the manifestation of dementia. As adequate antibiotic and anti-inflammatory therapies are available, as in syphilis, one might prevent and eradicate dementia.

Keywords: Alzheimer's disease, bacteria, *Borrelia burgdorferi*, dementia, infection, Lyme disease, periodontal pathogen, spirochetes, *Treponema*, syphilis

Redoxaktive bioenergetische Naturwirkstoffe zur Prävention und Therapie


anti-
adhäsiv
anti-
invasiv
anti-
infektiös

Hauptkeime der Parodontose:

Porphyromonas gingivalis

Andere fusiforme Bakterien

Spirochäten.

mm Hg

Die Austauschergänzung

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Brain & Development 28 (2006) 141–146

BRAIN & DEVELOPMENT
Official Journal of the Japanese Society of Child Neurology

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Review article

Paradigm shift in treatment of Wilson's disease: Zinc therapy now treatment of choice

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Received 26 October 2004; received in revised form 1 August 2005; accepted 1 August 2005

Abstract

Zinc therapy has replaced penicillamine as first-line therapy for Wilson's disease. New guidelines reflect the paradigm shift in treatment that has occurred in recent years. In the old paradigm, Wilson's disease was seen as genetic disorder associated with the accumulation of copper in the liver and in other organs once the liver had become overloaded with copper. When left untreated, the disease was regarded as uniformly fatal. The old treatment guidelines advised, 'decoppering' with penicillamine because this chelating agent was considered effective in restoring most patients to health. Before the start of treatment, patients were warned that their symptoms could worsen during the first weeks or months of therapy, so as to prevent them from abandoning penicillamine therapy in dismay. In the new paradigm, Wilson's disease is seen as a hereditary disorder associated with copper intoxication. The essence of symptomatic Wilson's disease is poisoning by free copper in the blood, that is, by copper that is not bound to ceruloplasmin. This form of copper is toxic, whereas accumulated copper and copper that is bound to ceruloplasmin or metallothionein is not. The treatment of symptomatic Wilson's disease is no longer aimed at 'decoppering', the removal of accumulated copper, but at the normalization of the free copper concentration in blood, to reverse the copper poisoning. This can be achieved safely and effectively with zinc therapy. Zinc induces metallothionein, a highly effective detoxification protein that binds copper. Oral zinc therapy leads to storage of metallothionein-bound copper in the mucosa of the gut and to the excretion of copper via the stools. New treatment guidelines advise against the use of chelating agents as initial treatment because they may aggravate copper intoxication and cause iatrogenic deterioration.
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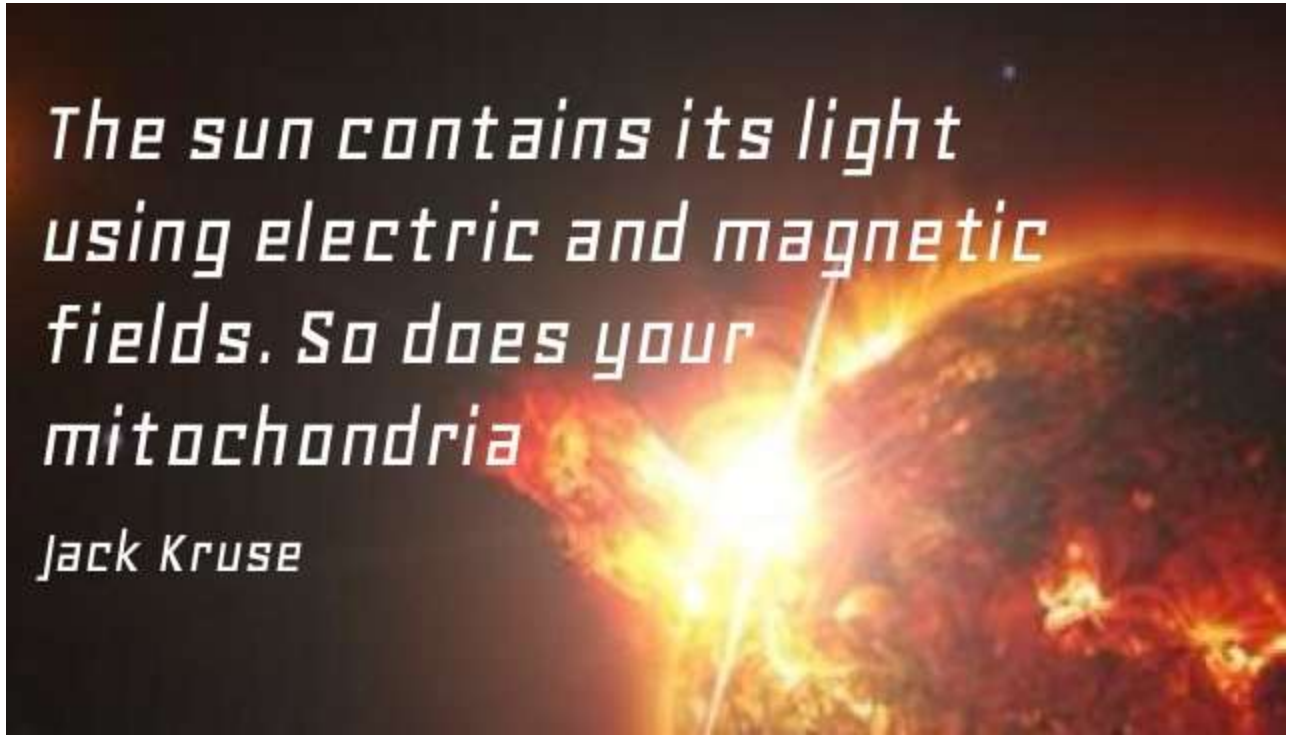
Keywords: Wilson's disease; Penicillamine; Zinc therapy; Paradigm shift; Copper

Energie und Kraft für unseren Körper und Geist

Gesundheit aus der Natur

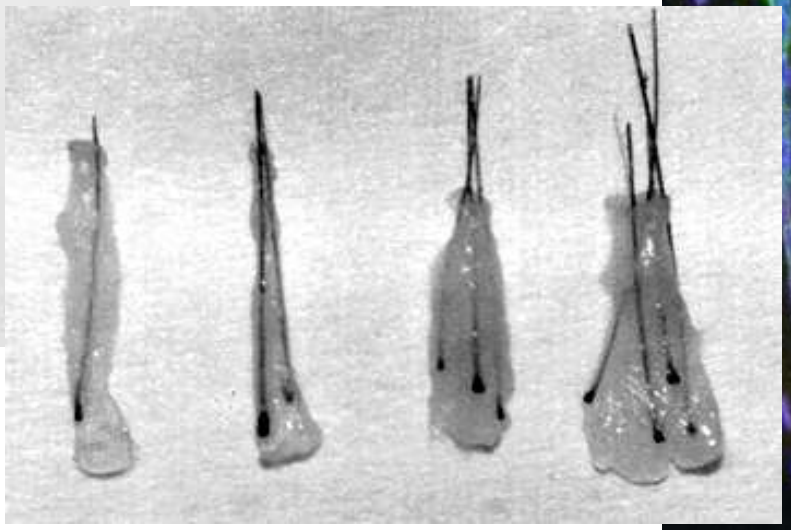
Naturwirkstoffe ermöglichen Gesundheit

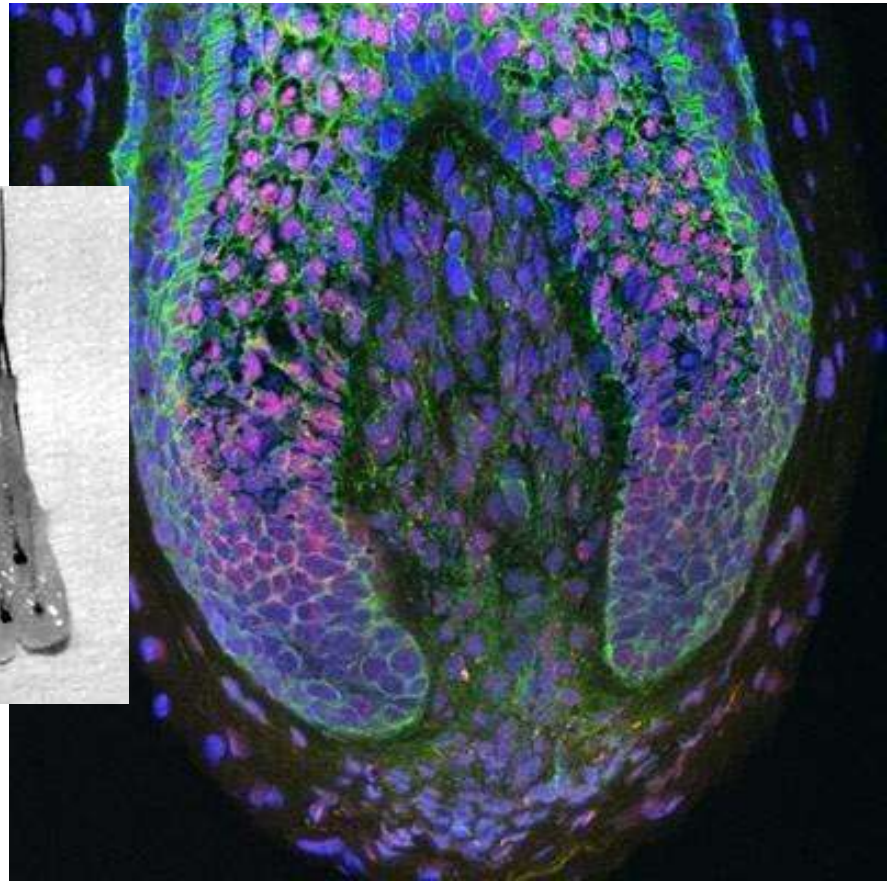

The sun contains its light using electric and magnetic fields. So does your mitochondria
Jack Kruse

Die humane Haarfollikelorgankultur

als optimale Modellsysteme für die

Forschung und Praxis der Bioenergetik





A simple assay for the study of human hair follicle damage induced by ionizing irradiation

Burkhard Poeggeler[1]*, Enikö Bodó[1,2]*, Roger Nadrowitz[3], Juergen Dunst[3] and Ralf Paus[1,4]*

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[2]Agricultural and Molecular Research Institute, College of Nyíregyháza, Nyíregyháza, Hungary;
[3]Department of Radiation Therapy, University of Lübeck, Lübeck, Germany;
[4]School of Translational Medicine, University of Manchester, Manchester, UK
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**These two authors contributed equally to this study.*

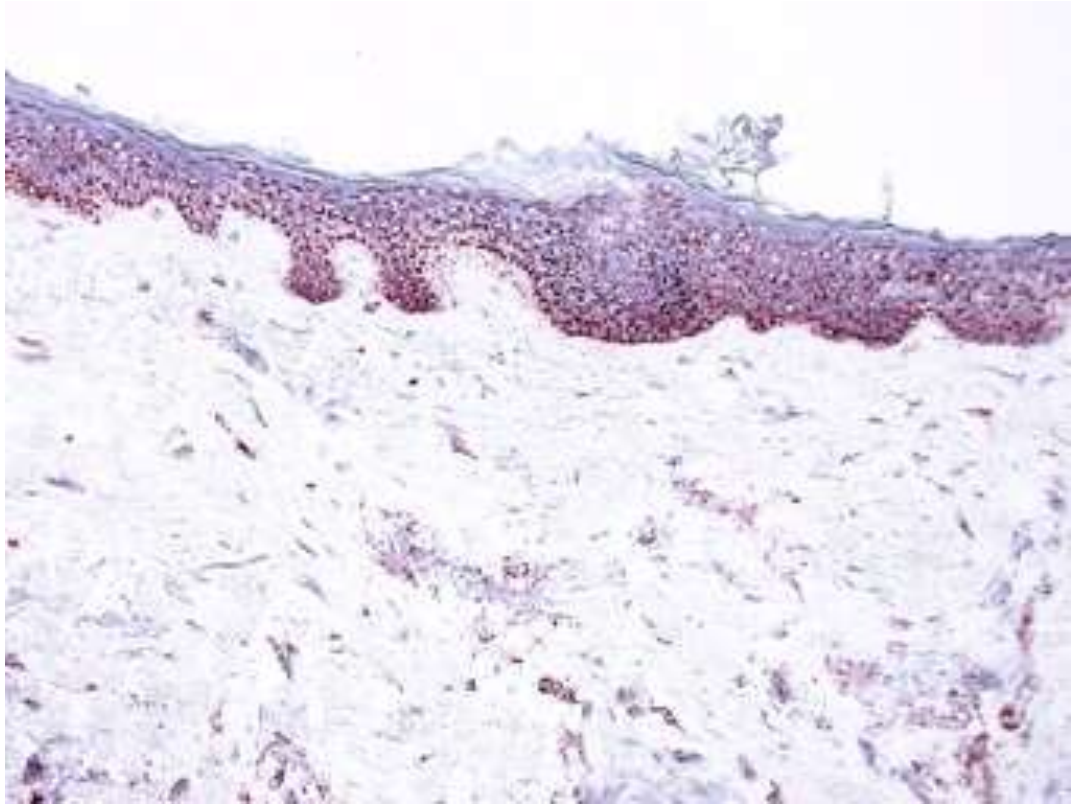
Abstract: Due to its rapidly proliferating matrix keratinocytes, the hair follicle is highly sensitive to ionizing irradiation (IR)-induced skin damage and thus an instructive and clinically relevant model organ for investigating the effects of IR on rapidly dividing epithelial-mesenchymal interaction systems. Here, we have assessed the impact of IR on organ-cultured human scalp hair follicles. We show that IR significantly inhibits the proliferation and induces apoptosis of hair follicle matrix keratinocytes, disrupts normal hair follicle pigmentation, and upregulates a number of quantitative toxicity and viability markers (oxidative stress indicators, DNA oxidative damage, LDH release). This introduces human hair follicle organ culture as an excellent novel research tool for radiobiology and invites exploitation as a preclinical assay system for testing candidate radioprotectants.

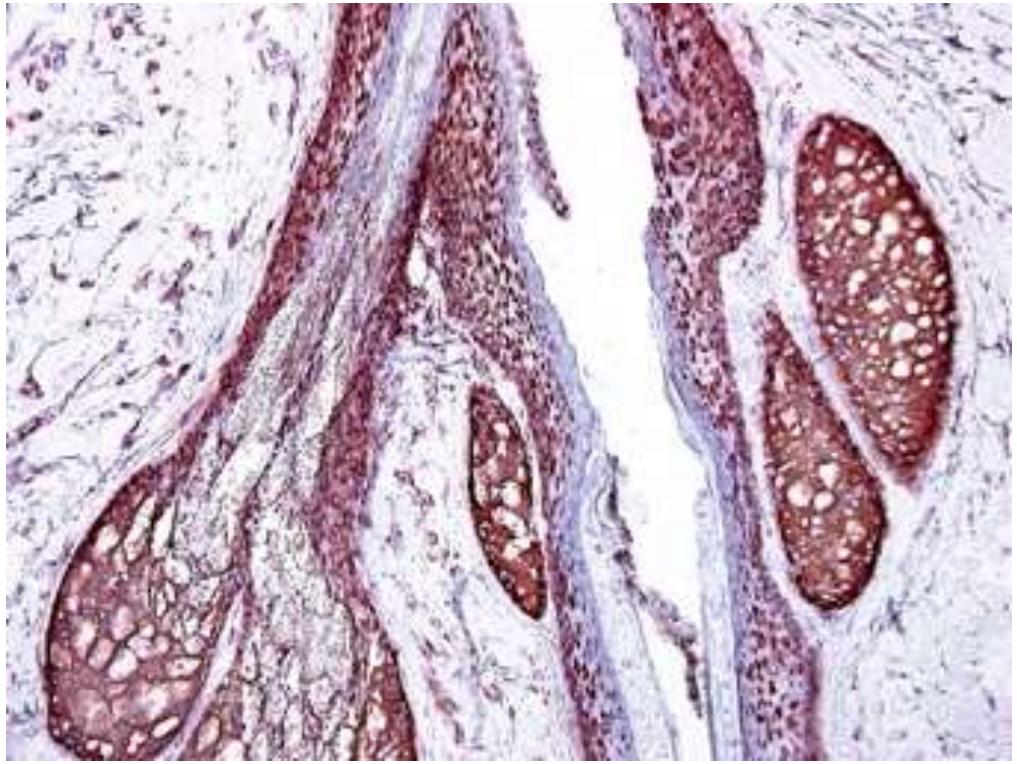
Key words: apoptosis – cancer – pigmentation – proliferation – radiation

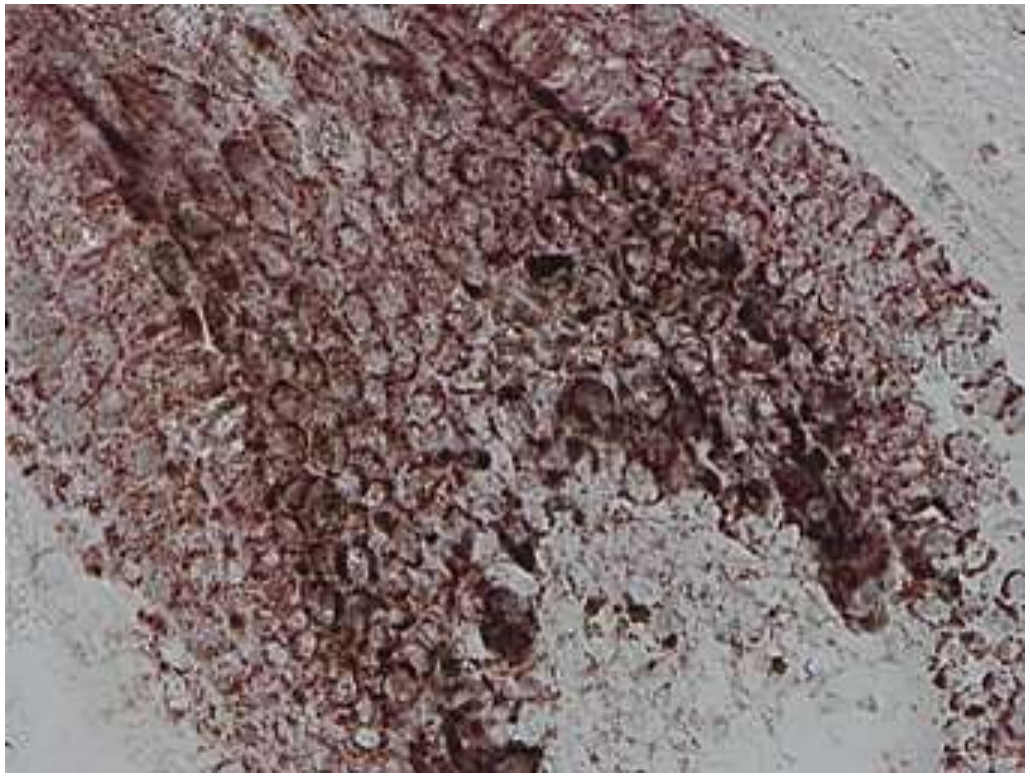
Haut und Haare

als Zugang

zur Bioenergetik



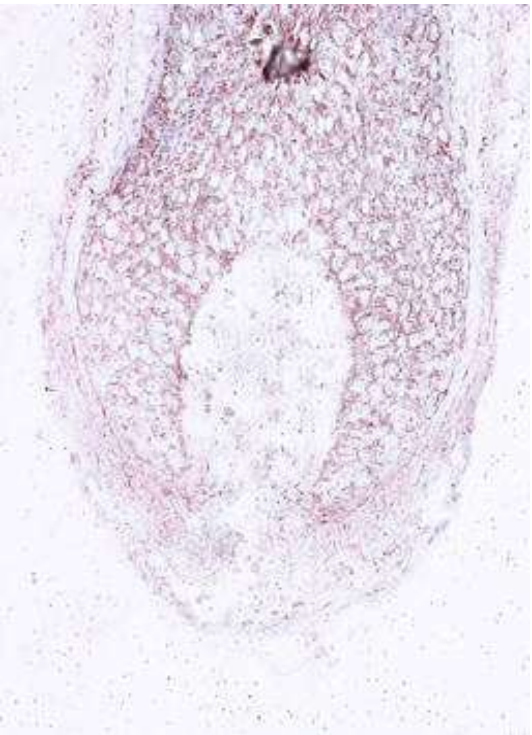


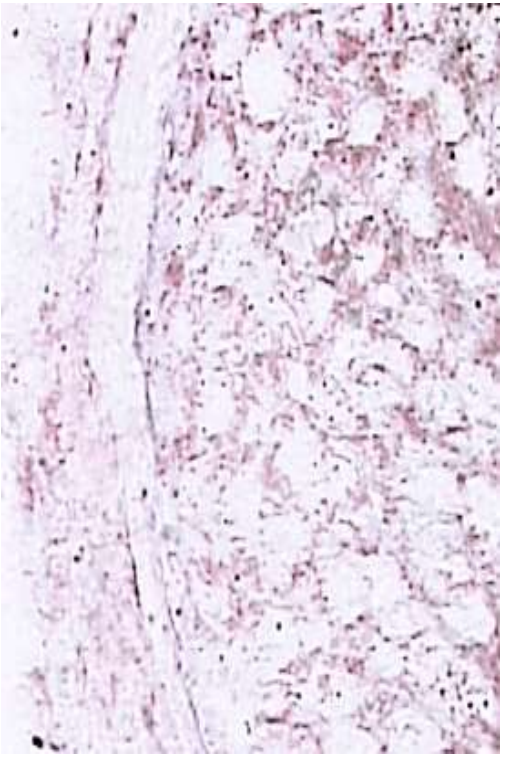


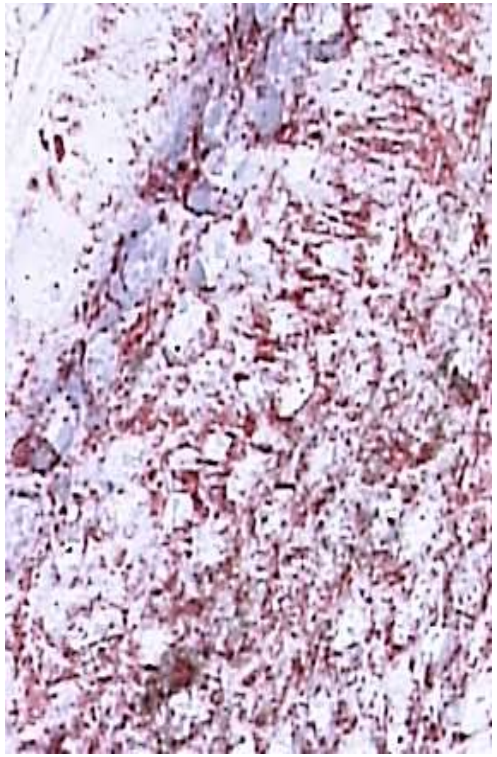
TPS: Trophic Prosurvival Signaling

STASIS: Stress and Senescence

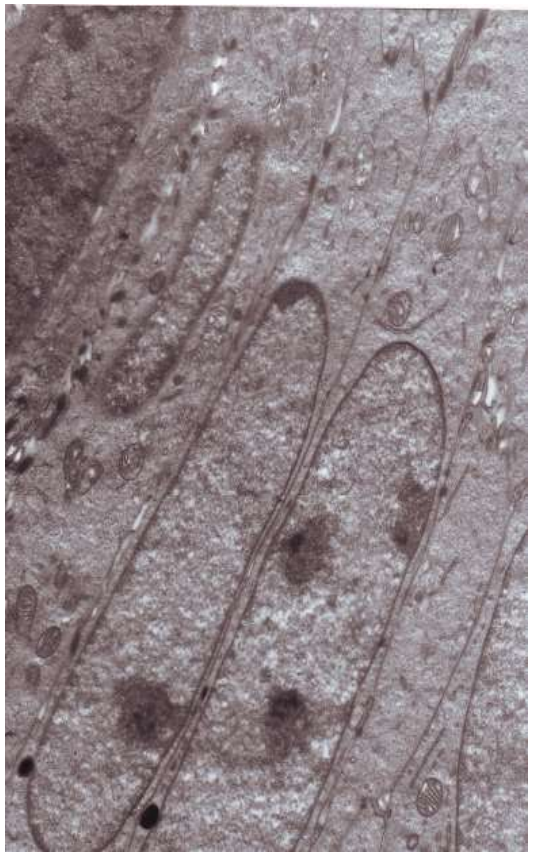
Induced Aberrant Signaling







Die Tiamat 3X Technology



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Review

Cite this article: Busch KB, Kowald A, Spelbrink JN. 2014 Quality matters: how does mitochondrial network dynamics and quality control impact on mtDNA integrity? *Phil. Trans. R. Soc. B* **369**: 20130442. http://dx.doi.org/10.1098/rstb.2013.0442

One contribution of 9 to a Theme Issue 'What cost mitochondria? Maintenance and evolution of mtDNA'.

Subject Areas:
cellular biology, genetics, evolution, molecular biology

Keywords:
mitochondrial dynamics, mtDNA, mitophagy, nucleoids, TFAM, OXPHOS

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Quality matters: how does mitochondrial network dynamics and quality control impact on mtDNA integrity?

Karin B. Busch[1], Axel Kowald[2] and Johannes N. Spelbrink[3,4]

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[2]Centre for Integrated Systems Biology of Ageing and Nutrition, Institute for Ageing and Health, Newcastle University, Campus for Ageing and Vitality, Newcastle upon Tyne NE4 5PL, UK
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[4]FinMIT Centre of Excellence, Institute of Biomedical Technology and Tampere University Hospital, Pirkanmaa Hospital District, 33014 Tampere, Finland

Mammalian mtDNA encodes for 13 core proteins of oxidative phosphorylation. Mitochondrial DNA mutations and deletions cause severe myopathies and neuromuscular diseases. Thus, the integrity of mtDNA is pivotal for cell survival and health of the organism. We here discuss the possible impact of mitochondrial fusion and fission on mtDNA maintenance as well as positive and negative selection processes. Our focus is centred on the important question of how the quality of mtDNA nucleoids can be assured when selection and mitochondrial quality control works on functional and physiological phenotypes constituted by oxidative phosphorylation proteins. The organelle control theory suggests a link between phenotype and nucleoid genotype. This is discussed in the light of new results presented here showing that mitochondrial transcription factor A/nucleoids are restricted in their intramitochondrial mobility and probably have a limited sphere of influence. Together with recent published work on mitochondrial and mtDNA heteroplasmy dynamics, these data suggest first, that single mitochondria might well be internally heterogeneous and second, that nucleoid genotypes might be linked to local phenotypes (although the link might often be leaky). We discuss how random or site-specific mitochondrial fission can isolate dysfunctional parts and enable their elimination by mitophagy, stressing the importance of fission in the process of mtDNA quality control. The role of fusion is more multifaceted and less understood in this context, but the mixing and equilibration of matrix content might be one of its important functions.

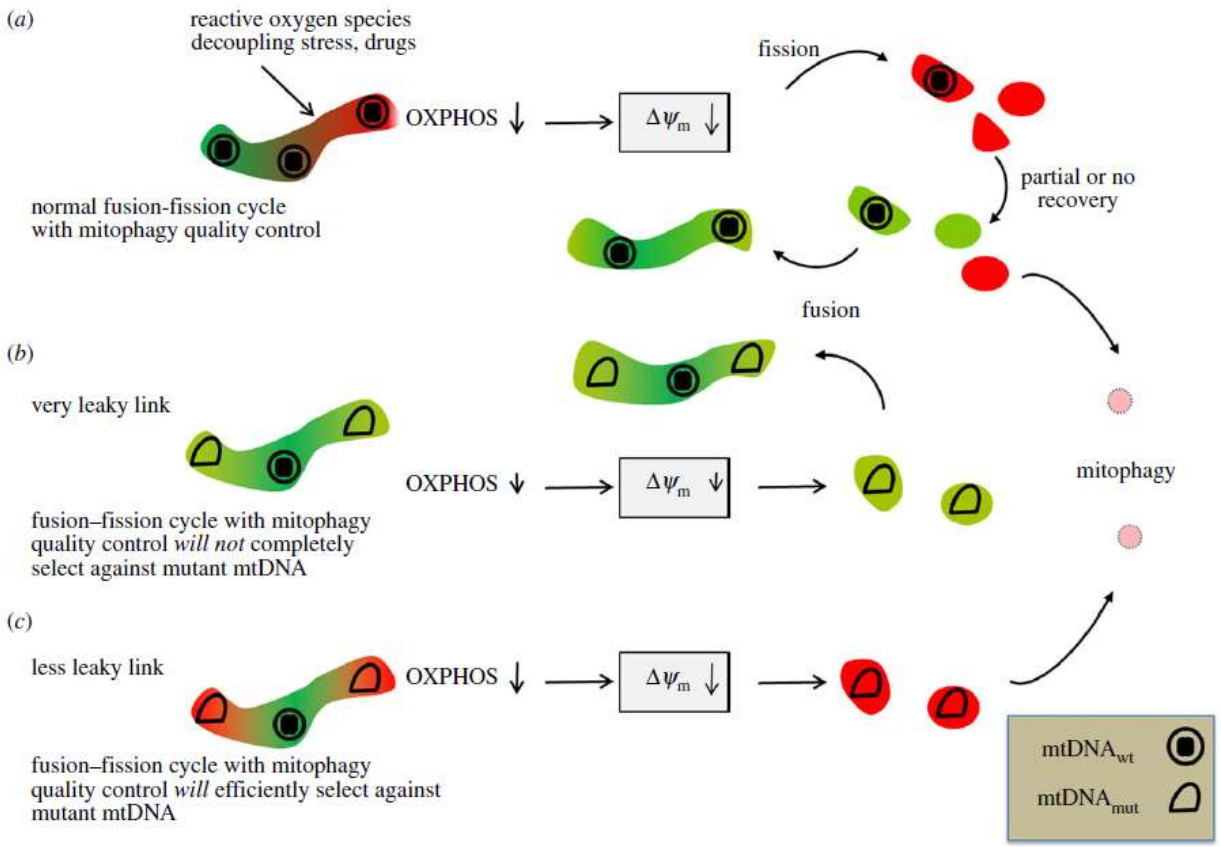


Figure 6. Genotype–phenotype linkage as the critical parameter to eliminate dominant negative mtDNA. (*a*) Mitochondria with wild-type mtDNA can undergo (spontaneous or induced) depolarization, but recovery followed by fusion brings them back into the dynamic mitochondrial network. (*b*) In a very leaky link, the presence of mutant next to wild-type mtDNA in the same mitochondrion would allow intramitochondrial functional complementation. As long as the decrease of proton motive force (*pmf*) and membrane potential, respectively, is not too harsh, elimination by mitophagy will not work and mutant mtDNA can escape the quality control. This spares biomaterial on cost on stringency. (*c*) In a less leaky link, with closer genotype-phenotype coupling, fission can generate fragments with low *pmf*, unable to fuse again, prone to be eliminated by mitophagosomes.

Drei Population von Mitochondrien

Gesunde Mitochondrien mit hohem Membranpotenzial

Belastete Mitochondrien mit normalem Membranpotenzial

Defekte Mitochondrien mit niedrigem Membranpotenzial

Defekte Mitochondrien müssen eliminiert werden

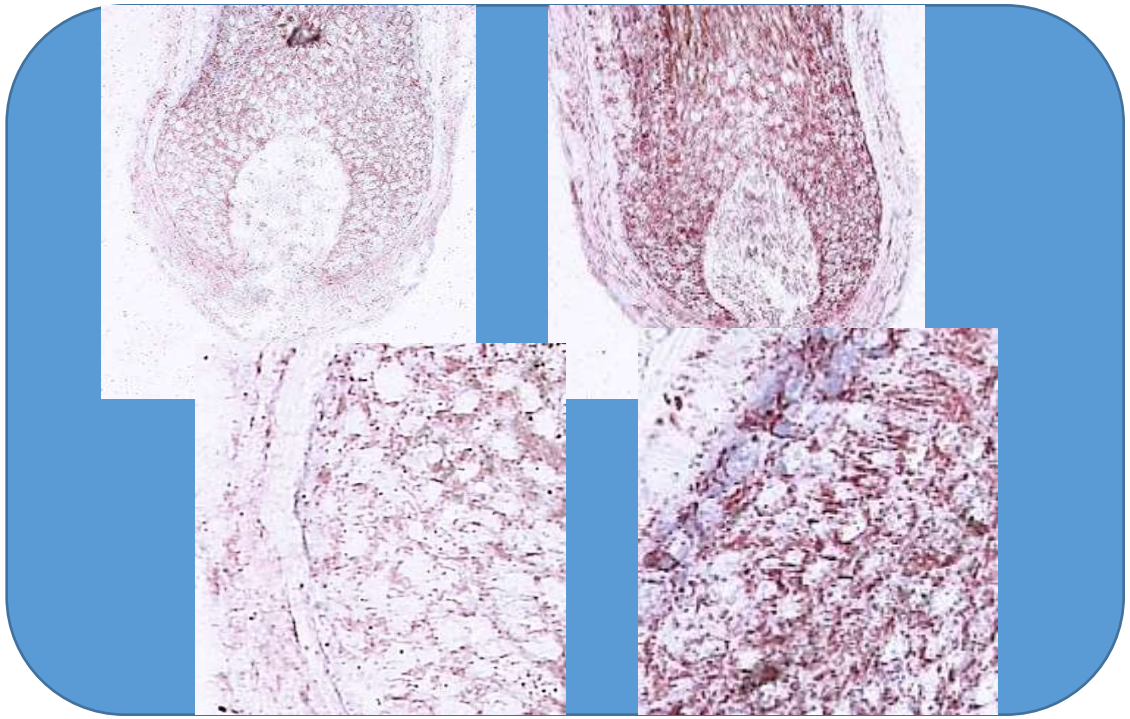
Erreicht durch Mitophagie mit Tiamat 3X Technology

RQ-Wert und VO_2Max

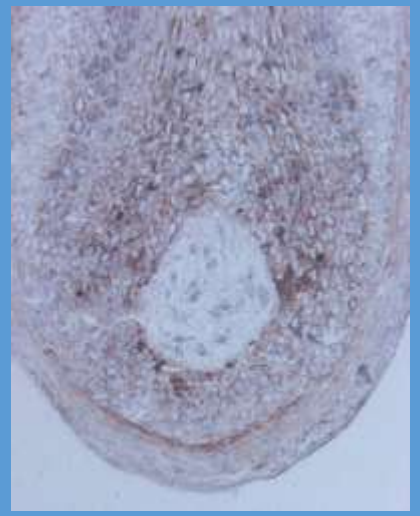
können signifikant

erhöht werden

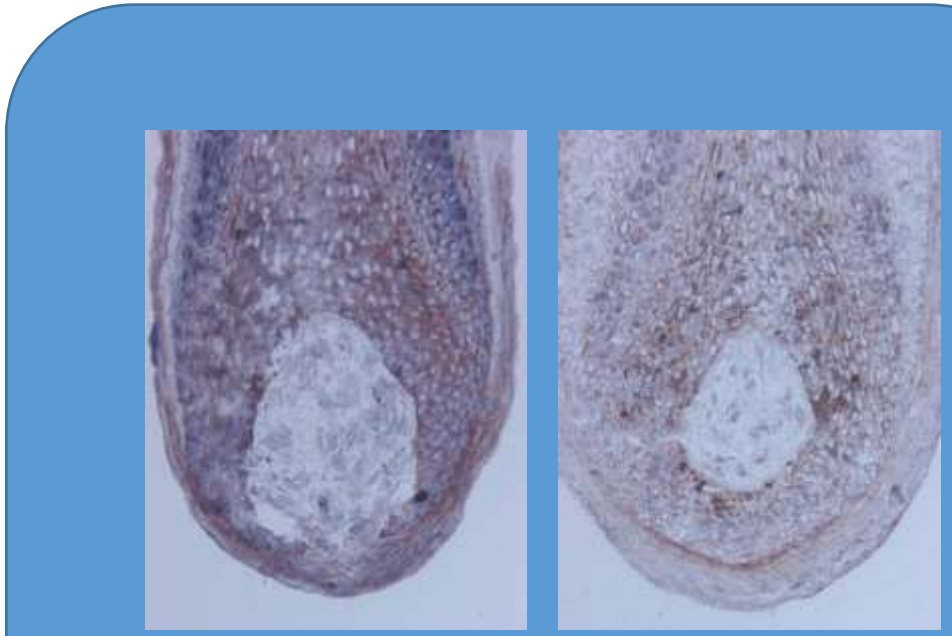
Bioenergetisierung
durch Nährstoffergänzung aus und für die Biomatrix
in den Haarwurzeln (NBT Reduktion in den Mitochondrien)



Protektion gegen oxidativen Stress durch Nährstoffergänzung aus und für die Biomatrix in den Haarwurzeln (8-OHdG)



Detoxifikation mit Entzündungshemmung durch Nährstoffergänzung aus und für die Biomatrix in den Haarwurzeln (Makrophagen)



	Placebo Beginn	Tiamat Beginn	Placebo 3 Monate	Tiamat 3 Monate
NBT Reduktion nmol/min/mg Protein	189 ± 19	208 ± 16	196 ± 21	**876 ± 73***
Eisen-III-Reduktion nmol/min/mg Protein	92 ± 9	107 ± 8	119 ± 11	**681 ± 59***
Cytochrome-c-Oxidation nmol/min/mg Protein	56 ± 4	52 ± 4	59 ± 5	**274 ± 23***

	Placebo Beginn	Tiamat Beginn	Placebo 3 Monate	Tiamat 3 Monate
Stickstoffmonoxid (NO) nmol/min/mg Protein	2.1 ± 0.3	1.9 ± 0.3	1.8 ± 0.2	**5.6 ± 0.4***
Superoxidanionradikale nmol/min/mg Protein	0.4 ± 0.1	0.6 ± 0.1	0.7 ± 0.1	**0.1 ± 0.1***
Peroxynitrit nmol/min/mg Protein	0.4 ± 0.1	0.3 ± 0.1	0.5 ± 0.1	0.1 ± 0.1*

	Placebo Beginn	Tiamat Beginn	Placebo 3 Monate	Tiamat 3 Monate
Toxizität LDH (Absorption)	0.25 ± 0.03	0.21 ± 0.02	0.27 ± 0.03	0.07 ± 0.01*
Peroxide nmol/mg Protein	26 ± 3	27 ± 2	28 ± 2	5 ± 1*
DNS Schäden (8-OH-dG) Zahl der positiven Zellen	3.9 ± 0.3	3.4 ± 0.2	3.6 ± 0.3	1.2 ± 0.3*
Apoptose (p53) Zahl der positiven Zellen	4.9 ± 0.5	4.6 ± 0.4	4.8 ± 0.4	1.1 ± 0.1*
Zelltod (TUNEL) Zahl der positiven Zellen	7.3 ± 0.5	7.7 ± 0.6	8.2 ± 0.8	3.1 ± 0.2*
Proliferation (Ki-67) Zahl der positiven Zellen	24 ± 1.8	23 ± 1.1	25 ± 1.6	33 ± 1.5*

Das metabolische Syndrom

Gewicht: - 15.9 kg/3 Monate

↓ Systolischer Blutdruck: - 27 mmHg/3 Monate

↓ ADMA Konzentrationen: - 57 %/3 Monate

Das maligne metabolische Syndrom

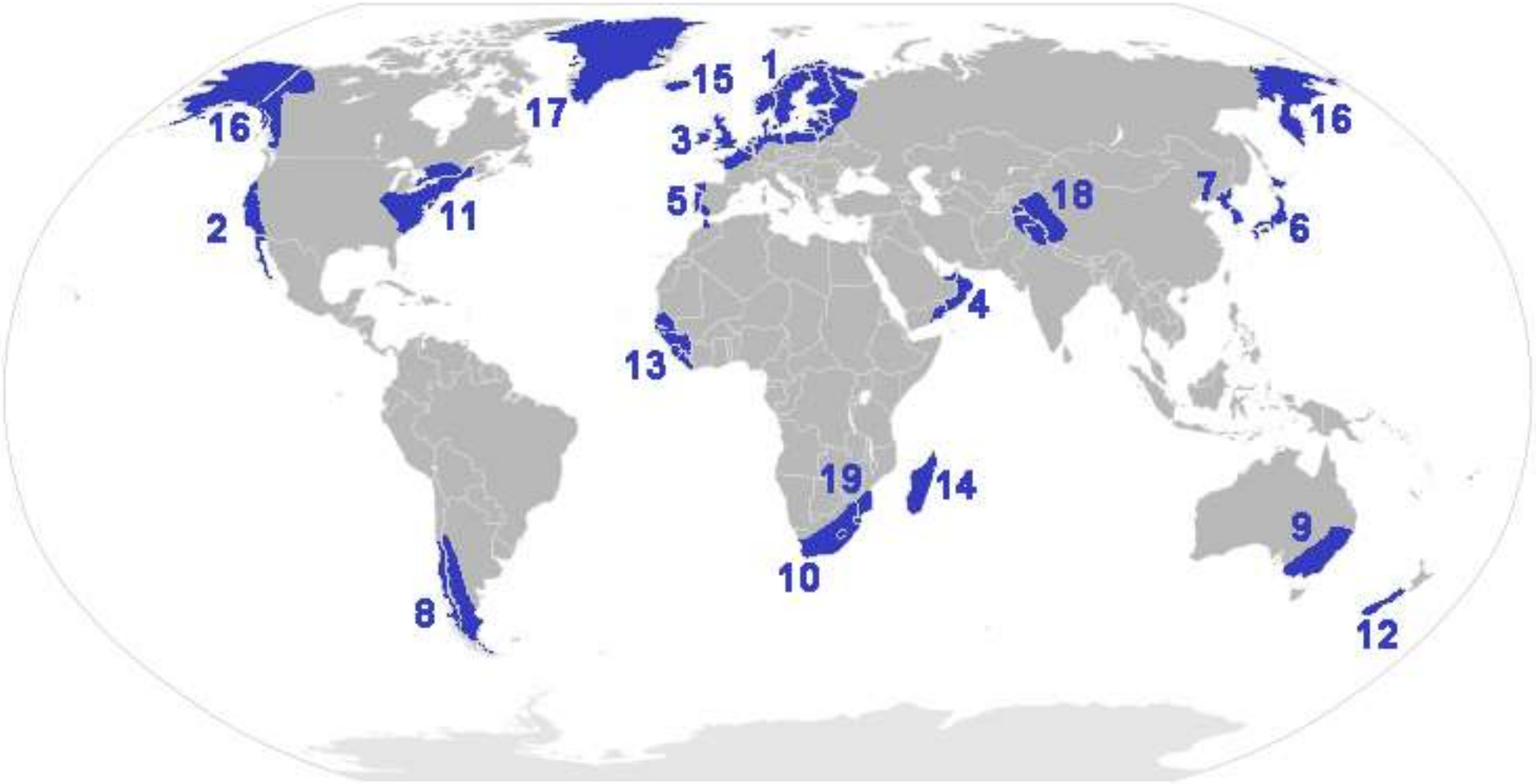
Prostatakrebs

80 % Responder
20 % Remission

nach 3 Jahren

Okinawa

105 Jahre jung und gesund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Kalorienarme

und nährstoffreiche

Ernährung

Gesundheit erhalten

Gemeinsam gestalten

Gesellschaft verändern

Herzlichen Dank für Ihre Aufmerksamkeit

Ihr Burkhard Poeggeler