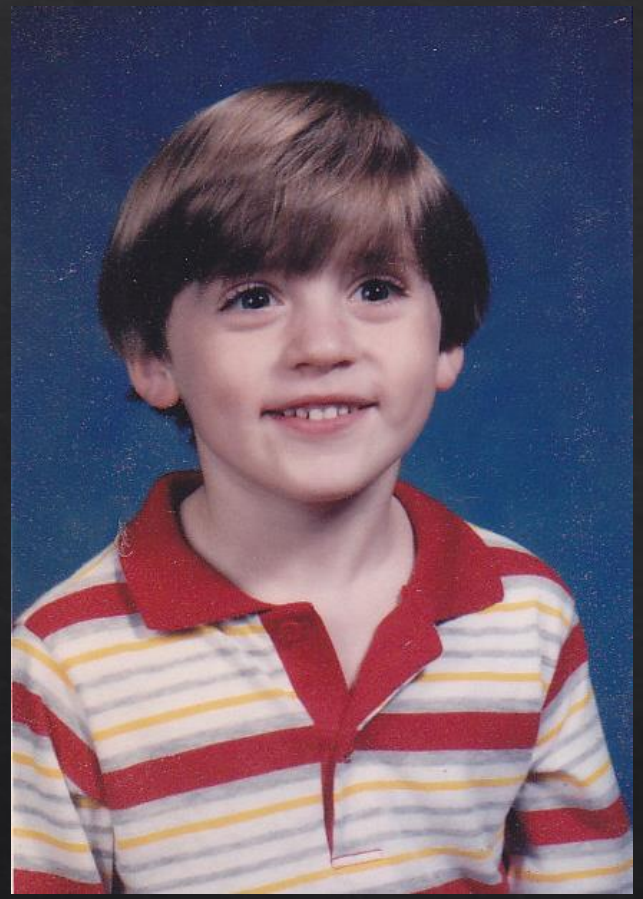
Cardiac Metabolism: Why Cancer of the Heart is So Rare

Dr. Stephen Hussey MS, DC

Wise Traditions Conference 2022

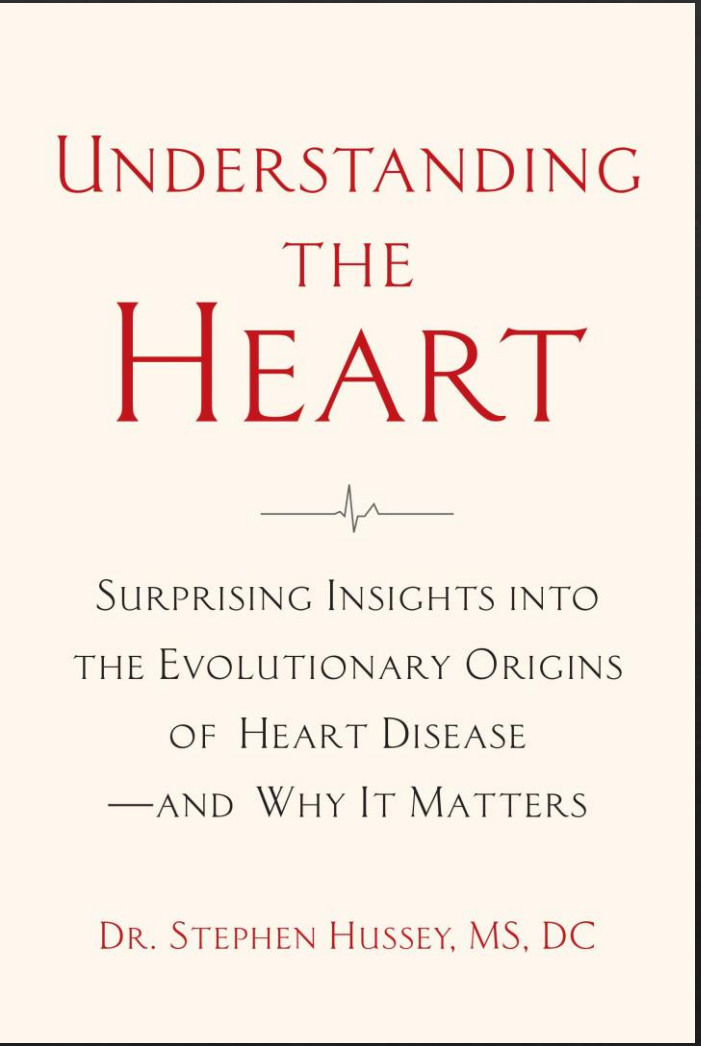
My Story



1. Age 2 – Diagnosed with asthma
2. Many allergies – from cats, to grass, to foods
3. Chronic Hives – Had to take prednisone
4. Irritable Bowel Syndrome – IBS
5. Age 9 – Type 1 Diabetes – heavily predisposed to heart disease
6. Age 18 – Doctor tried to put me on BP medication
7. Started to pay attention to lifestyle in college and became passionate about health
8. Age 22 – Doctor tried to put me on statin drug
9. Medical education

- Evolutionary origins of heart disease
- 4th phase water
- The heart is not the main mover of blood – heart failure
- The true function of the heart
- Atherosclerosis is clotting, not an accumulation of cholesterol
- Cholesterol and statin drugs
- Metabolic heart attacks
- High blood pressure
- True heart healthy diet
- Best biomarkers for heart disease
- The Autonomic Nervous System
- Chiropractic
- Dental health
- My heart attack at the age of 34

@resourceyourhealth



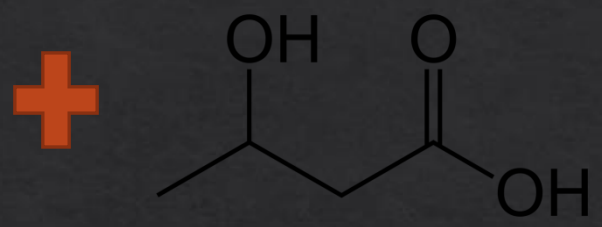
Heart Cancer Stats

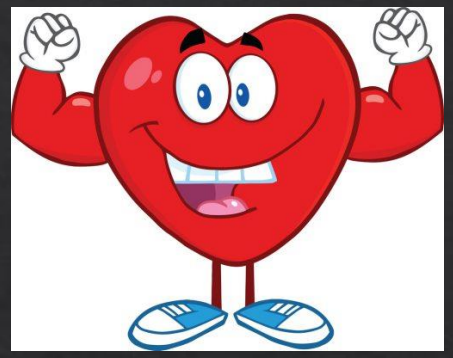
- Vast majority of tumors originating in heart are benign
- Study: 12,000 heart cancer cases, 7 were primary cardiac tumors
- Dr. Robert Cusimono, cardiac surgeon, 12 cases a year
- Primary cardiac tumors are 0.3 to 0.7% of all cardiac tumors
- Metastasis from other tumors is 30 times more likely
- When malignant, survival rate at 9 to 12 months is only 10%

1. Moynihan, T. J. (2019, January 9). Heart cancer: Is there such a thing? Retrieved from https://www.mayoclinic.org/heart-cancer/expert-answers/faq-20058130
2. Matters of the Heart: Why Are Cardiac Tumors So Rare? (n.d.). Retrieved from https://www.cancer.gov/types/metastatic-cancer/research/cardiac-tumors
3. Leja, M. J., Shah, D. J., & Reardon, M. J. (2011). Primary Cardiac Tumors. Texas Heart Institute Journal, 38(3), 261-262. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3113129/

Metabolic

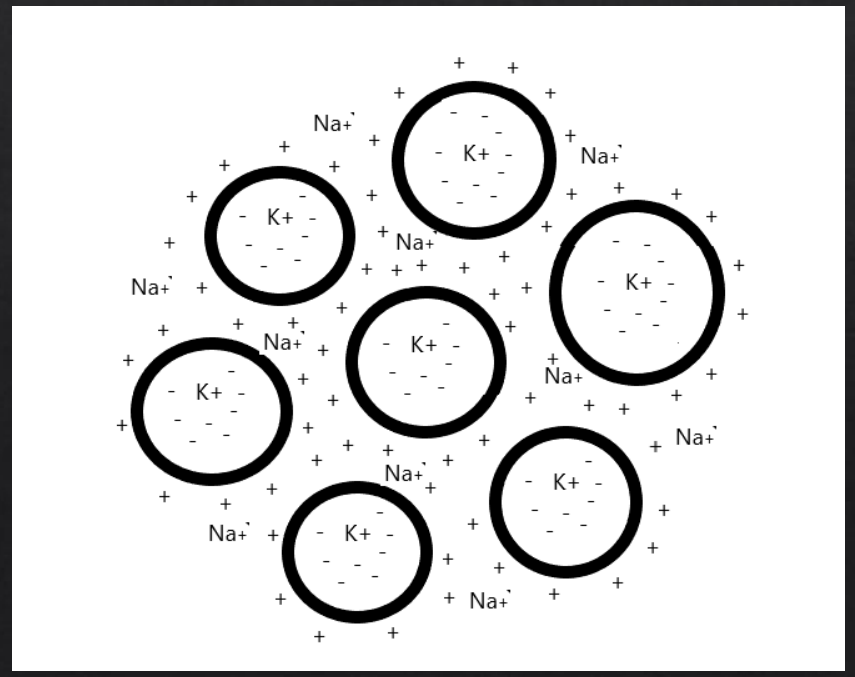
- Metabolic Theory of Cancer
  - Metabolic Heart Attacks



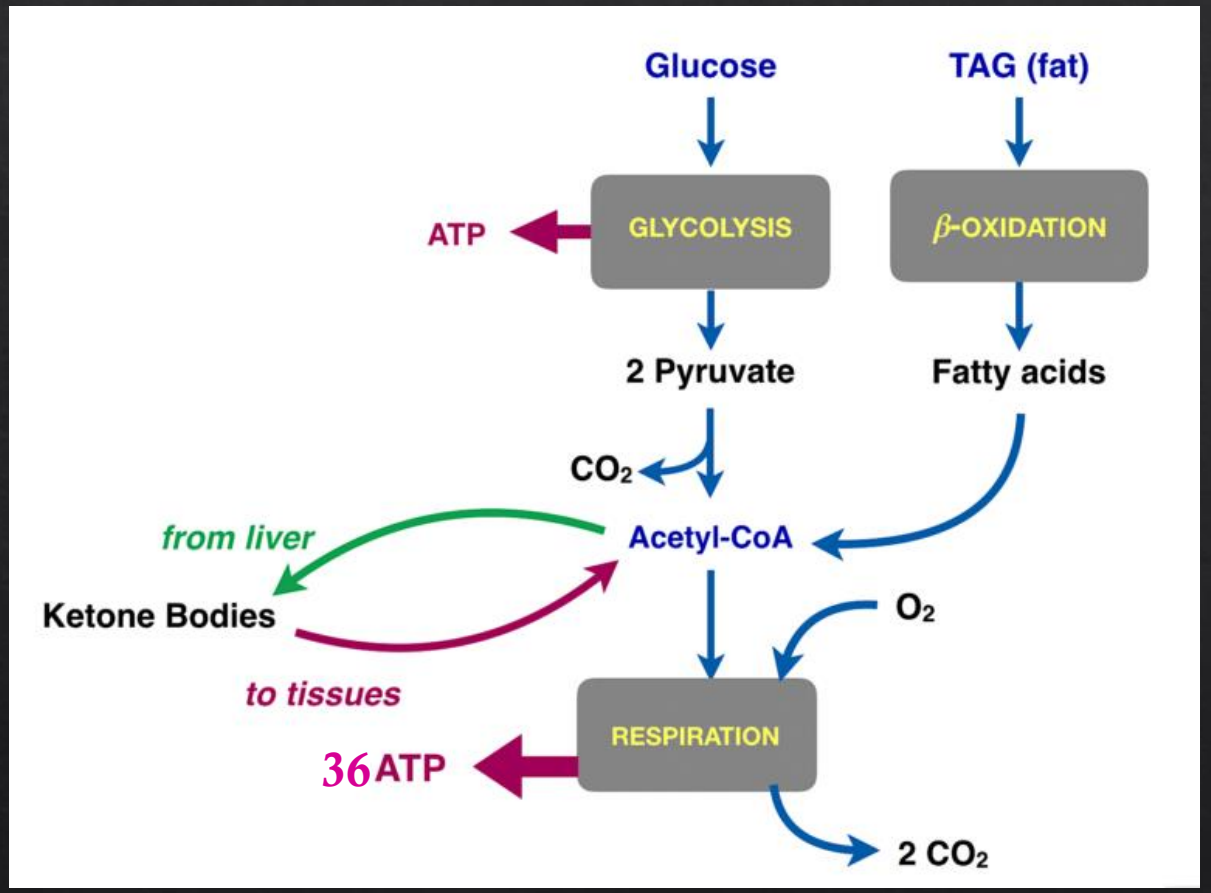


Biophysics/Quantum

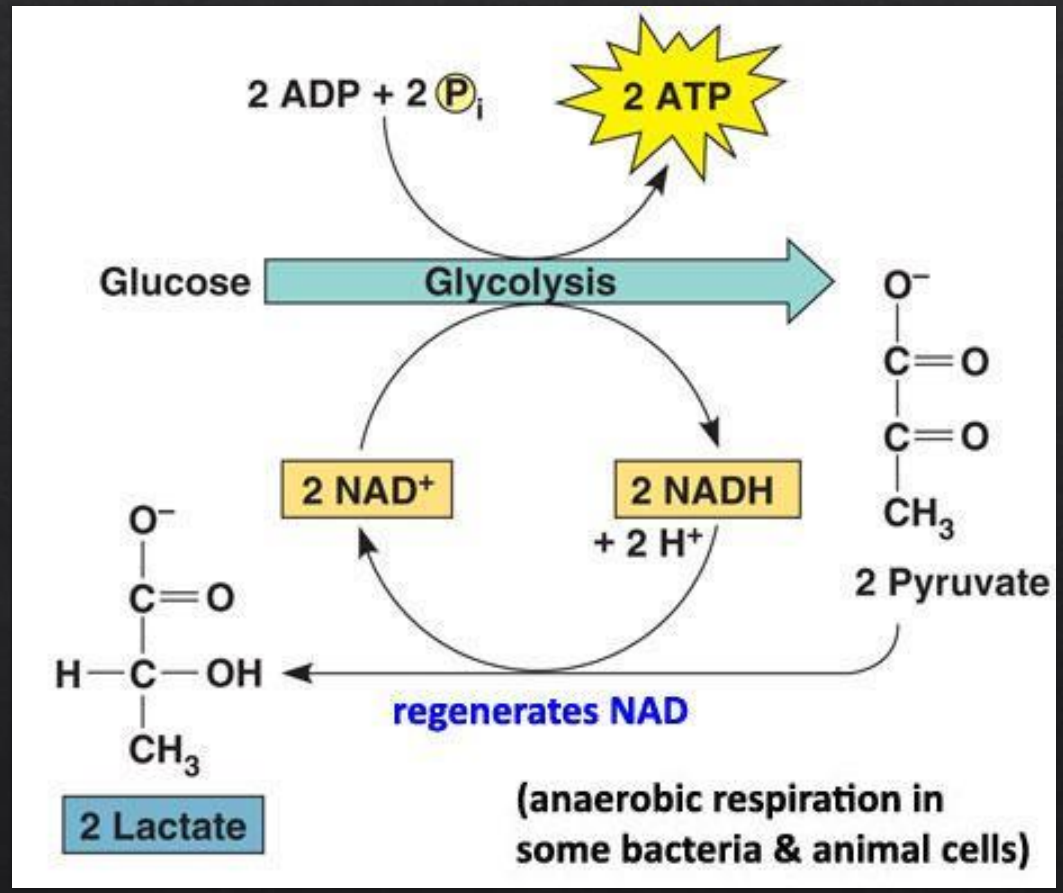
- Cells, Gels, and Electromagnetism



Metabolic Theory of Cancer



Oxidative Phosphorylation - 89% of total cellular energy

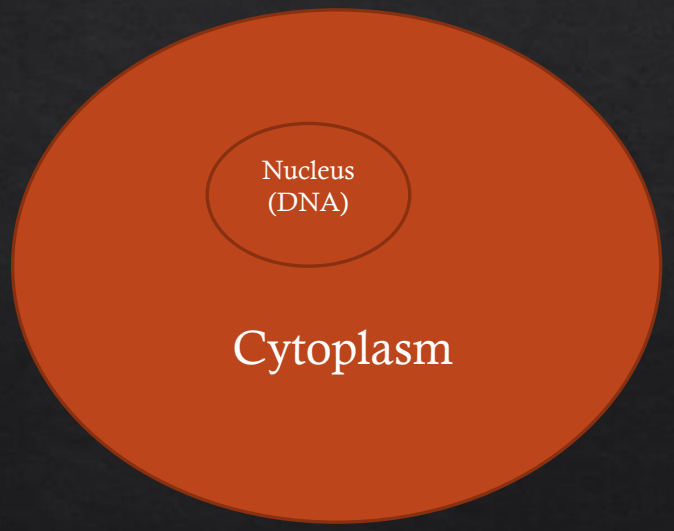


Cancer shows large increase in uptake of glucose

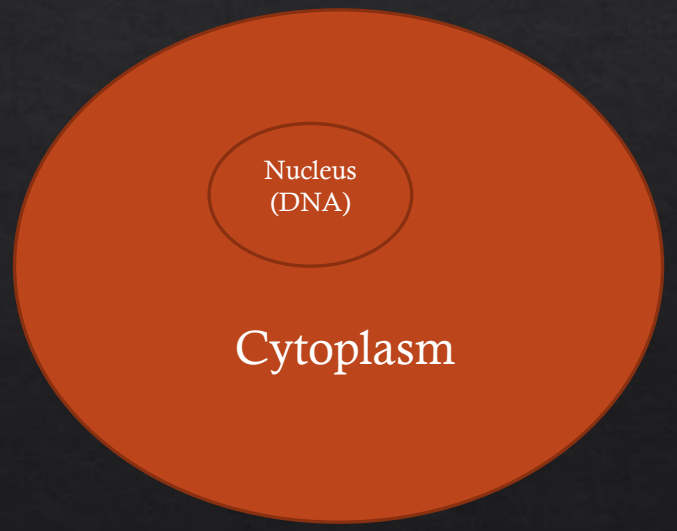
Metabolic Theory of Cancer

- "Although no specific gene mutation or chromosomal abnormality is common to all cancers, nearly all cancers express elevated fermentation, regardless of their tissue or cellular origin."

Healthy Cell

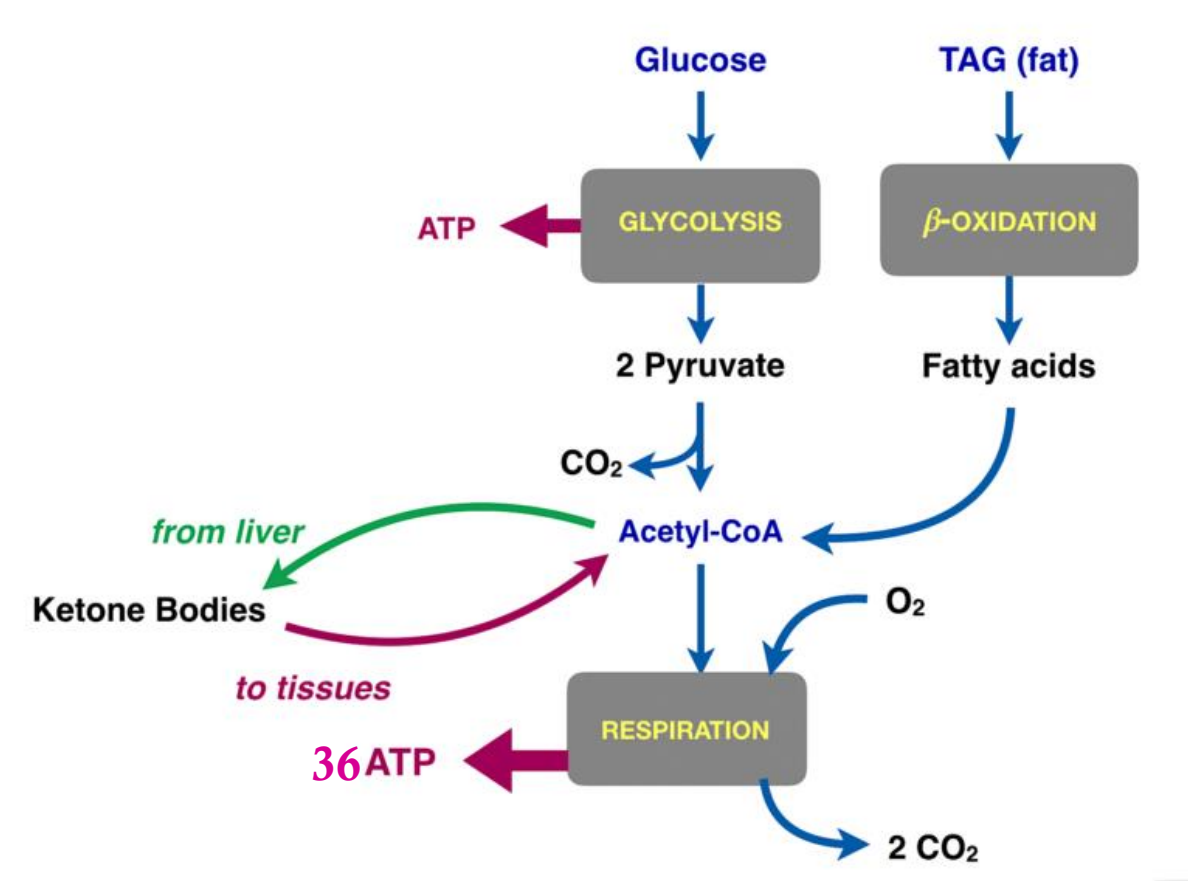


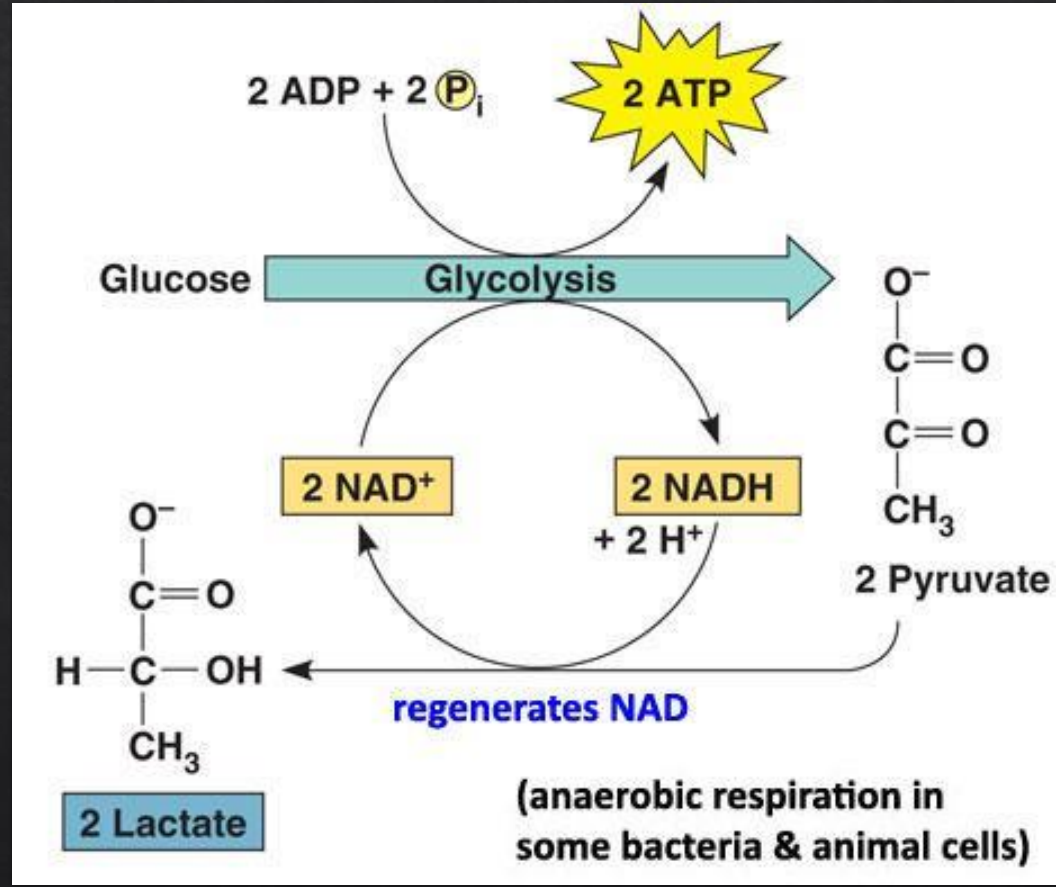
Cancer Cell



1. Seyfried, T. (2012). *Cancer as a Metabolic Disease: On the Origin, Management, and Prevention of Cancer*. John Wiley & Sons.
2. Koura M., Isaka H., Yoshida M. C., Tosu M., Sekiguchi T. (1982). Suppression of tumorigenicity in interspecific reconstituted cells and cybrids. Gann 73, 574–580.

Metabolic Theory of Cancer





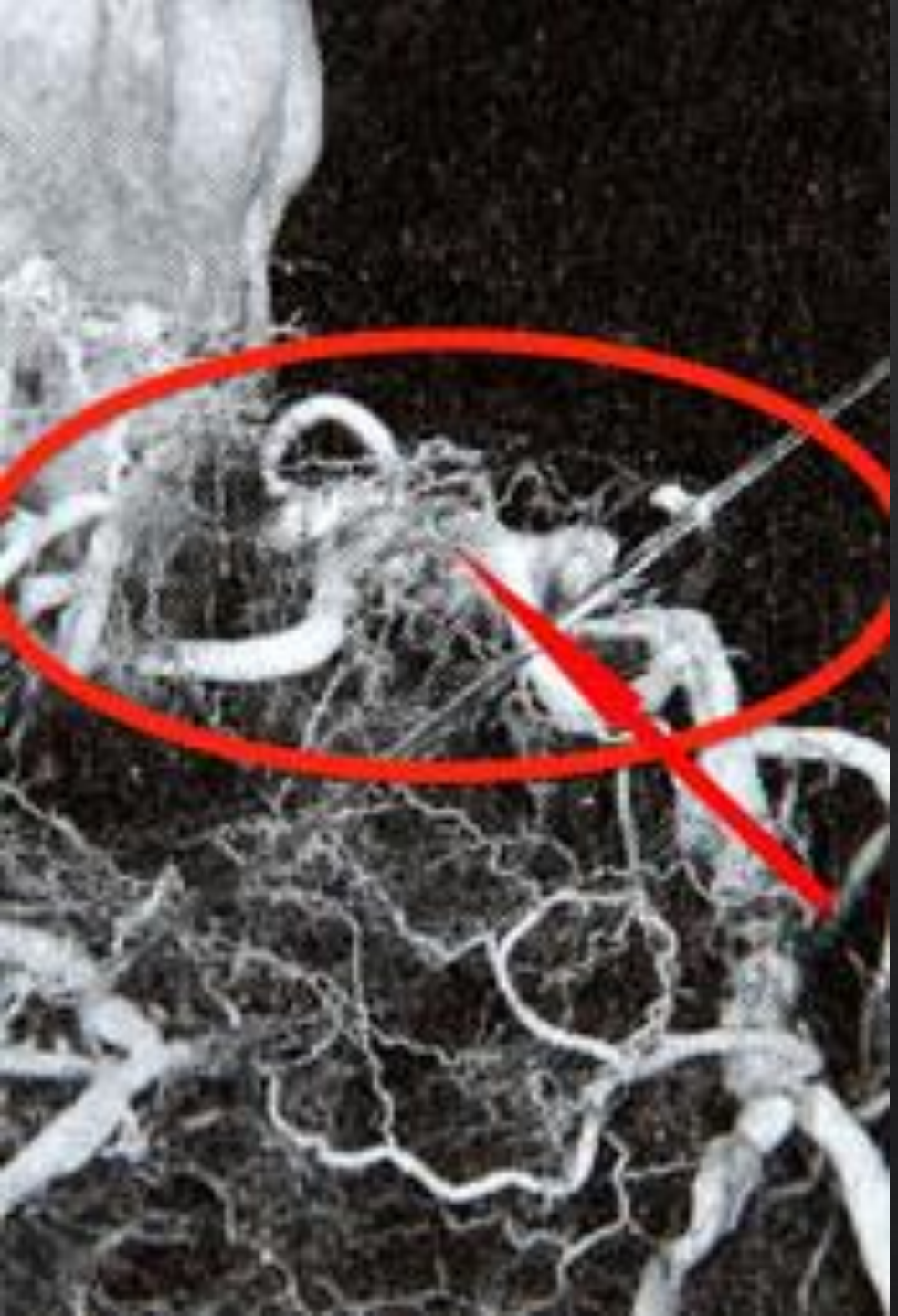
Why does this not happen often in heart tissue?

Metabolic Heart Attacks

Myocardial Infarction with non-obstructed coronary arteries (MINOCA)

- In 5% to 20% of cases, an angiogram will show “non-obstructive coronary artery disease,” which means that the arteries are less than halfway blocked.
- More common in women than men and affects up to 187,000 people in the United States each year.

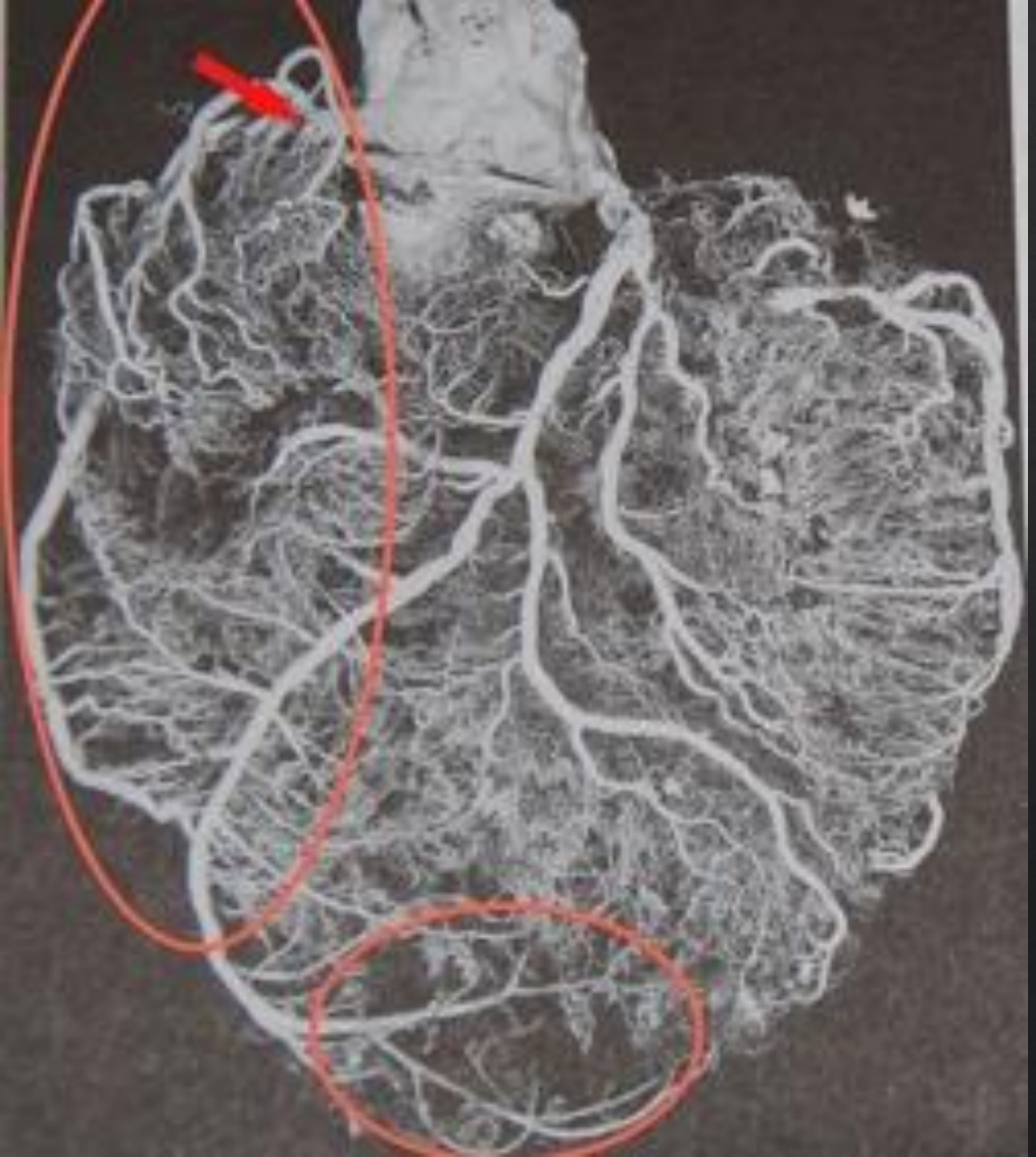
MINOCA Study | Women's Heart Attack Research Program. (n.d.). Retrieved from https://med.nyu.edu/research/womens-heart-attack-research-program/minoca-study



Plastic Cast Study

- "In the presence of atherosclerotic stenosis with a lumen/diameter reduction greater than 70% there was a dramatic increase in the diameter and length of collaterals…"
- "**Any** severely obstructed coronary artery lesion, even multiple ones, was **always** found associated with enlarged collaterals."
- "The anastomotic index in these instances ranged from 5 to 33 with a mean value of 16 associated with a single stenosis and 22 in multiple severe stenosis"

Baroldi, G., & Silver, M. D. (2004). *The Etiopathogenesis of Coronary Heart Disease: A Heretical Theory Based on Morphology* (2nd ed.). Georgetown, Texas: Eurekah.com/Landes Bioscience.



Baroldi Autopsies

- In accident victims of all ages, almost 40% had several severe stenoses of the coronary arteries. These people had in general never complained of heart problems.
- Critical stenoses of the coronary vessels were found in 2/3 of all the patients who had <u>not</u> died of heart disease.
- Of those who their heart attack was their first cardiac complaint, most had one or more severe stenoses of the coronary vessels.

Baroldi, G., & Silver, M. D. (2004). *The Etiopathogenesis of Coronary Heart Disease: A Heretical Theory Based on Morphology* (2nd ed.). Georgetown, Texas: Eurekah.com/Landes Bioscience.

What Can Cause a Heart Attack Without a Blockage?

Three Imbalances – Poor Metabolism, Oxidative Stress, Imbalance in the Stress Response of Autonomic Nervous System

Metabolic Health (Insulin Sensitivity)

88% of the American population is metabolically unhealthy!

METABOLIC SYNDROME AND RELATED DISORDERS
Volume XX, Number XX, 2018

Pp. 1–7
DOI: 10.1089/met.2018.0105

Prevalence of Optimal Metabolic Health in American Adults: National Health and Nutrition Examination Survey 2009–2016

Joana Araújo, PhD,[1] Jianwen Cai, PhD,[2] and June Stevens, PhD[1,3]

Abstract

Background: Several guidelines for cardiometabolic risk factor identification and management have been released in recent years, but there are no estimates of current prevalence of metabolic health among adults in the United States. We estimated the proportion of American adults with optimal cardiometabolic health, using different guidelines.

Methods: Data from the National Health and Nutrition Examination Survey 2009–2016 were analyzed ($n = 8721$). Using the most recent guidelines, metabolic health was defined as having optimal levels of waist circumference (WC <102/88 cm for men/women), glucose (fasting glucose <100 mg/dL and hemoglobin A1c <5.7%), blood pressure (systolic <120 and diastolic <80 mmHg), triglycerides (<150 mg/dL), and high-density lipoprotein cholesterol (≥40/50 mg/dL for men/women), and not taking any related medication.

Results: Changing from ATP III (Adult Treatment Panel III) guidelines to more recent cut points decreased the proportion of metabolically healthy Americans from 19.9% (95% confidence interval [CI]: 18.3–21.5) to 12.2% (95% CI: 10.9–13.6). Dropping WC from the definition increased the percentage of adults with optimal metabolic health to 17.6%. Characteristics associated with greater prevalence of metabolic health were female gender, youth, more education, never smoking, practicing vigorous physical activity, and low body mass index. Less than one-third of normal weight adults were metabolically healthy and the prevalence decreased to 8.0% and 0.5% in overweight and obese individuals, respectively.

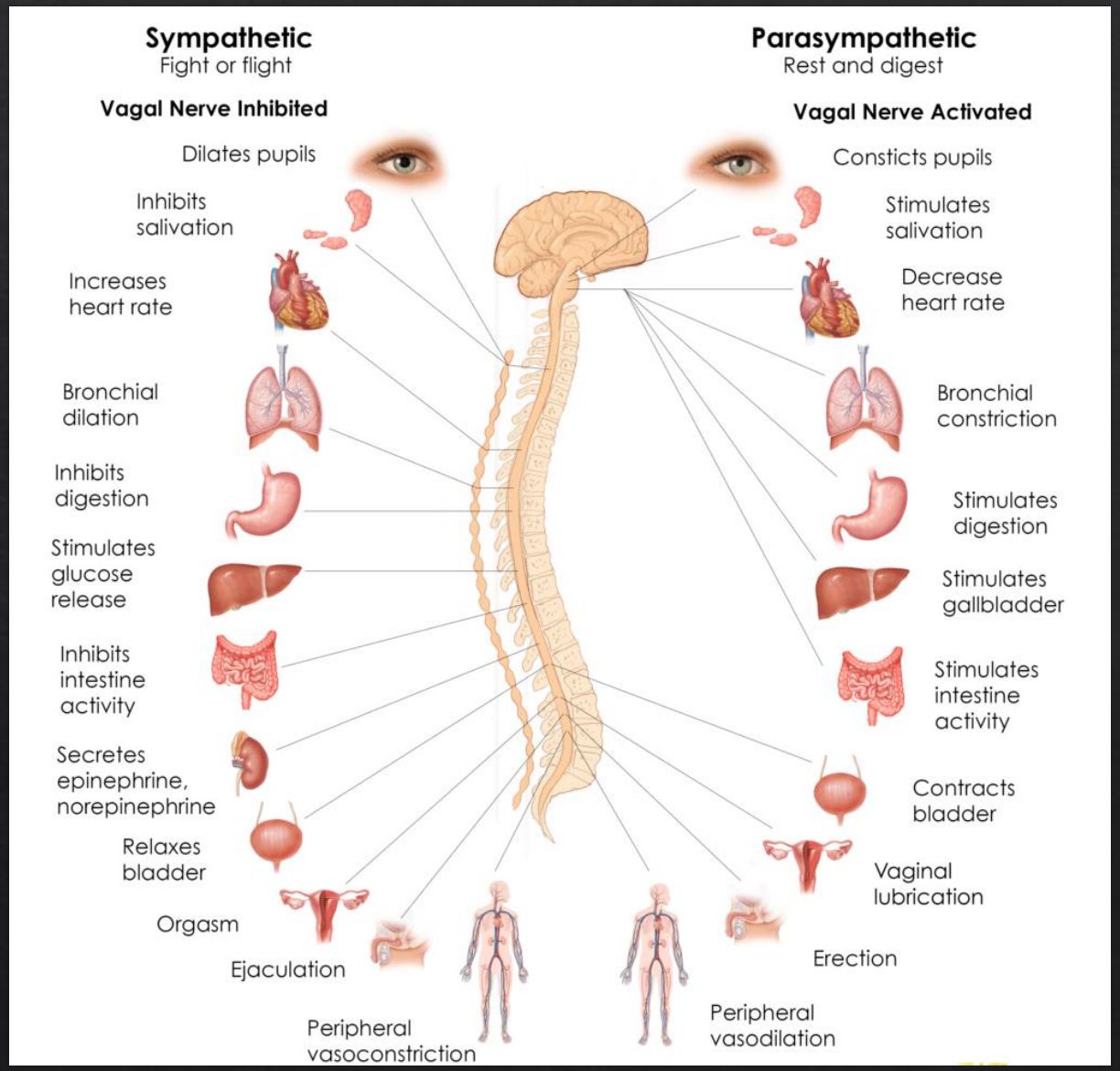
Conclusions: Prevalence of metabolic health in American adults is alarmingly low, even in normal weight individuals. The large number of people not achieving optimal levels of risk factors, even in low-risk groups, has serious implications for public health.

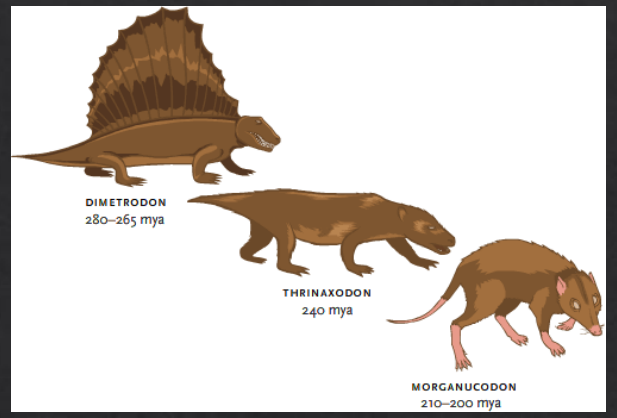
Keywords: metabolic health, risk factors, prevalence

“Electron-hungry process” Oxidative Stress

- Properties of Free Radicals
  - Naturally made in the process of burning any fuel source (fat, carbs, protein)
  - Normally neutralized by endogenous antioxidants like glutathione
  - Highly reactive
  - Very short half-life
  - Generate new radicals by chain reaction
  - Cause damage to cells, tissues, and EZ
  - Heavy metals, endotoxemia, high blood sugar, AGE’s, environmental toxins, seed oils, EMF

Autonomic Nervous System Imbalance





- The separate nuclei vagus system is the one that was passed on to all mammals
- DMN – Responsible for stimulating sympathetic nervous system
- NA – Vagal break that evolved to keep our complex brains and emotions from over stimulating the DMN (parasympathetic)
- NA supplies visceral efferent to the heart

Research Article | Article

INTERACTIONS O GLYCOGEN AND F HEARTS

JACOB A. BLUKOO-ALLOTEY, NORMA H. VINCENT, and SYDNEY ELLIS

Journal of Pharmacology and Experimental Therapeu

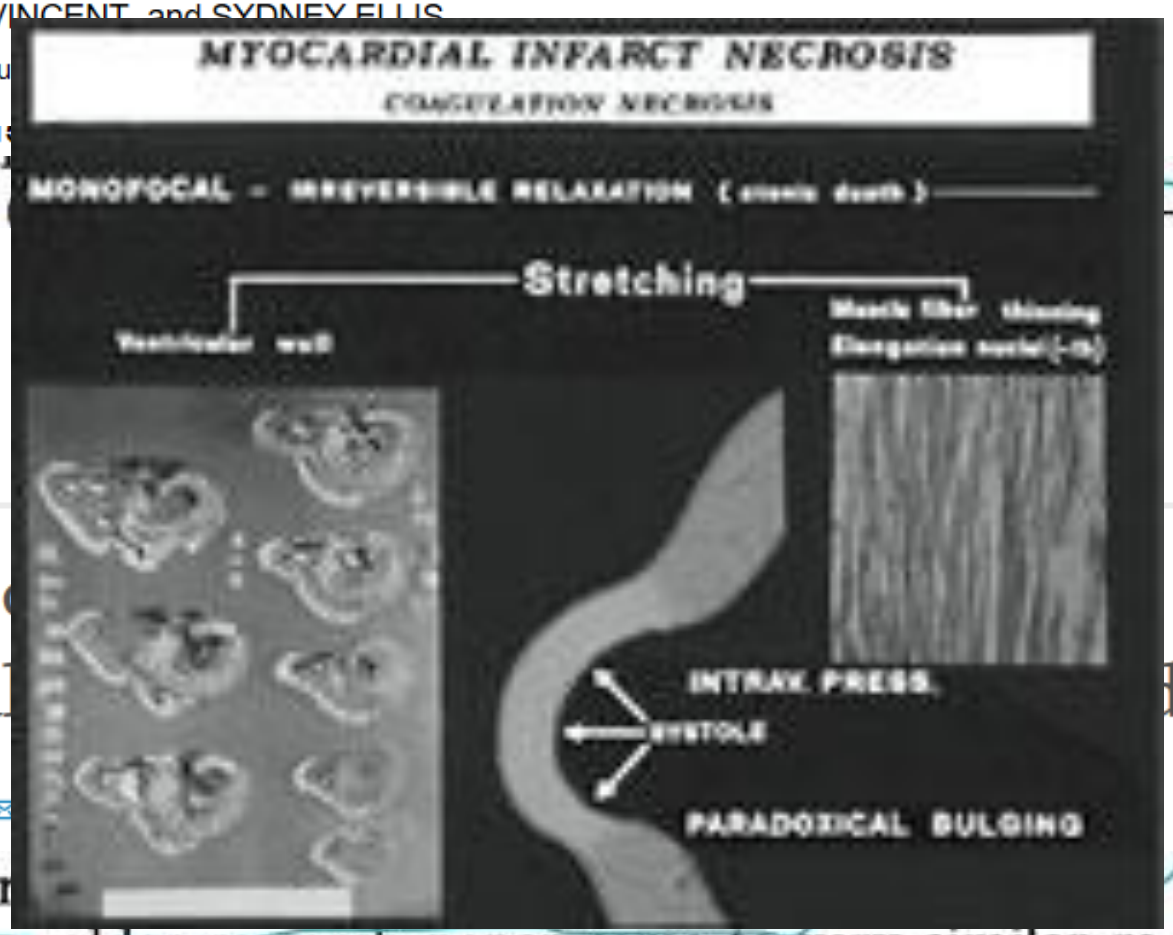
First published January 1

Different morphological types of myocardial cell death in man.

Baroldi G

Recent Advances in Studies on Cardiac Structure and Metabolism, 31 Dec 1974, 6:383-397

PMID: 1105714



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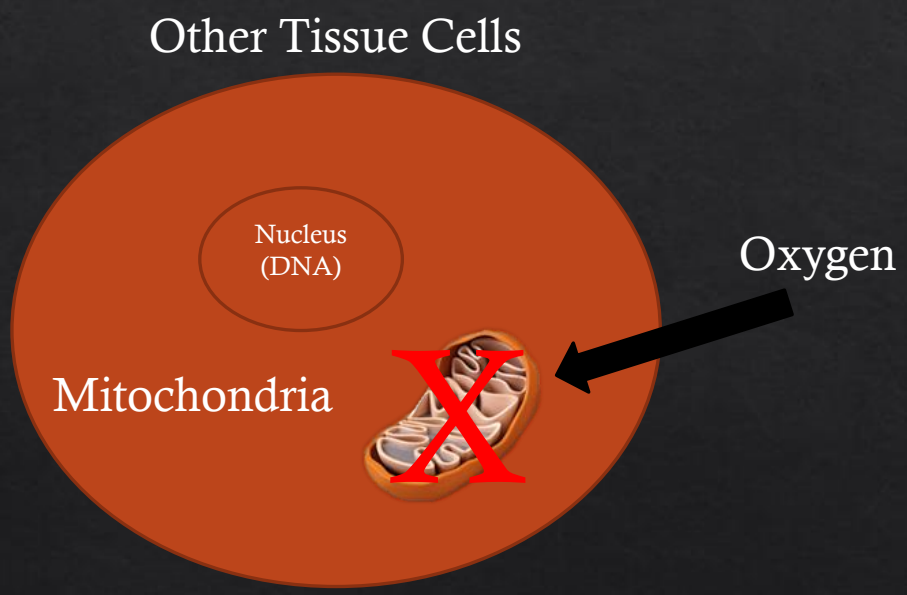
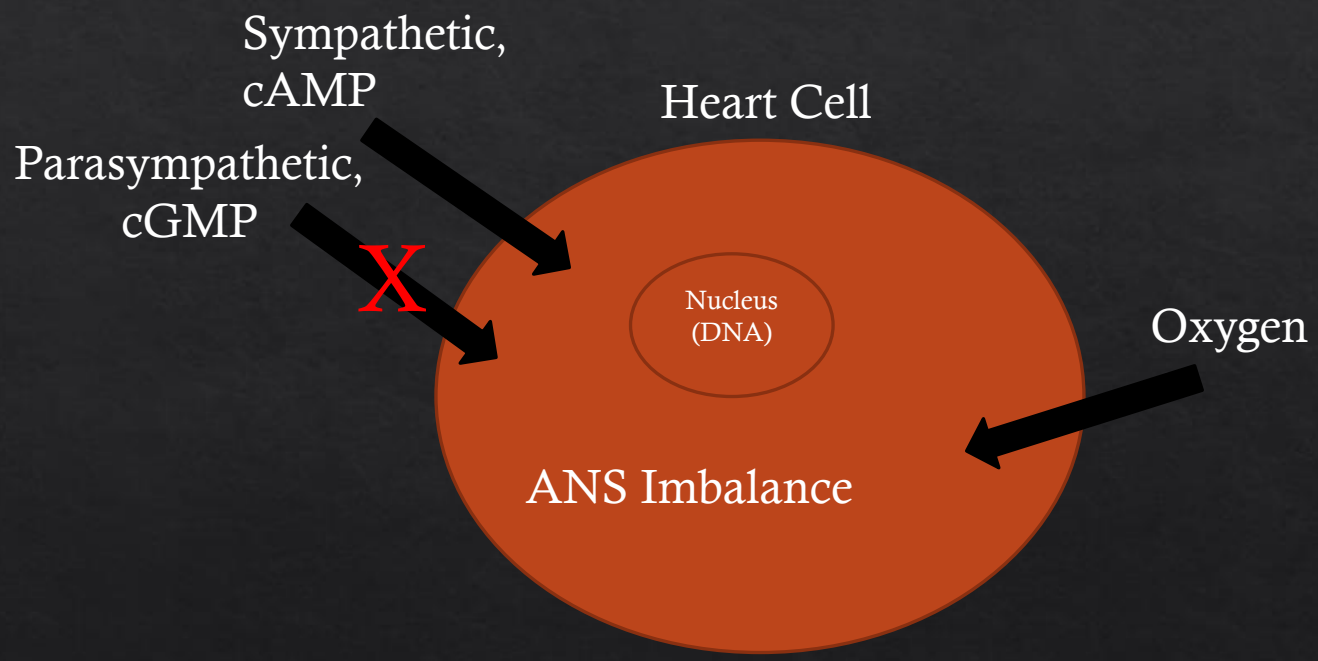
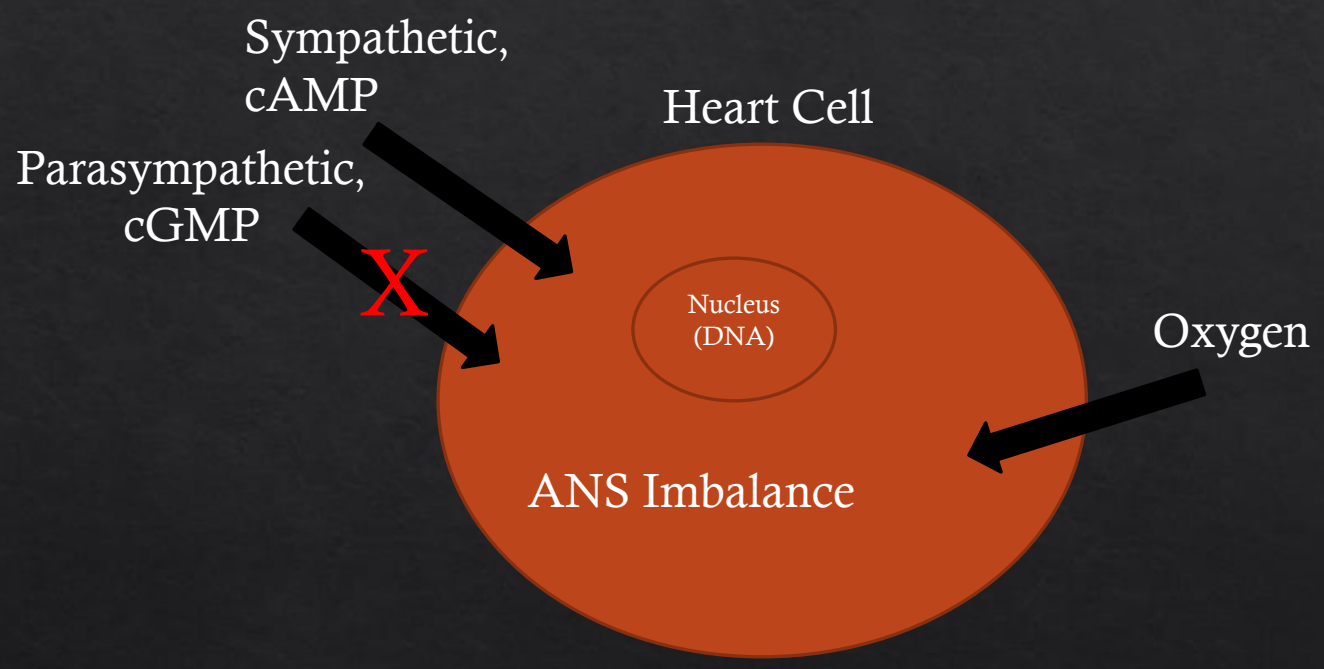
Two Paths to Aerobic Fermentation





Division Deficiency



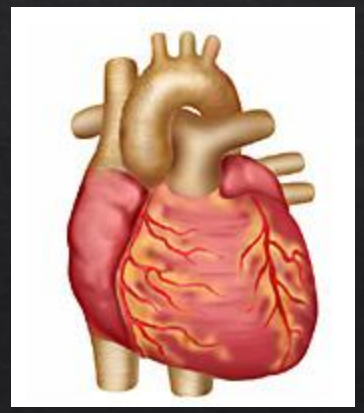
- After the 1st week of life, heart cells mature and lose the ability to replicate
- Thought to be because of the unique high metabolic activity of the heart

- Warburg said, "cells that die can never become tumorigenic".

-P. Sdek, P. Zhao, Y. Wang, C.-j. Huang, C. Y. Ko, P. C. Butler, J. N. Weiss, W. R. MacLellan. **Rb and p130 control cell cycle gene silencing to maintain the postmitotic phenotype in cardiac myocytes**. *The Journal of Cell Biology*, 2011; 194 (3): 407 DOI: 10.1083/jcb.201012049
-Warburg, O. (1956). On the Origin of Cancer Cells. *Science*, *123*(3191), 309-314. https://doi.org/10.1126/science.123.3191.309

What Does This Have to do With Cardiac Metabolism?

- Fatty acid oxidation, or fat burning, makes about 70% of the ATP that your heart produces.
- Add burning ketones to the mix and it can improve heart efficiency by 28%.



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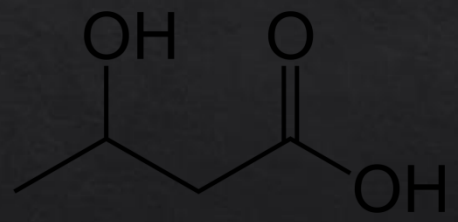

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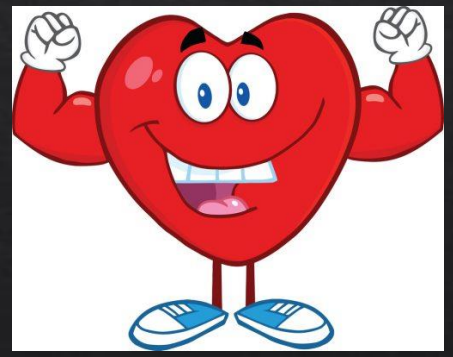


-Dedkova, E. N., & Blatter, L. A. (2014). Role of β-hydroxybutyrate, its polymer poly-β-hydroxybutyrate and inorganic polyphosphate in mammalian health and disease. *Frontiers in Physiology*, *5*. doi:10.3389/fphys.2014.00260
-Kashiwaya, Y., Sato, K., Tsuchiya, S., Thomas, S., & Fell, D. A. (1994). Control of glucose utilization in the perfused rat heart. *Journal of Biological Chemistry*, *269*, 25502-25514.

Heart Protection

- Fatty acids packaged in chylomicrons
- Heart has direct signaling pathway to fat cells
- Heart prefers fatty acids and ketones for fuel!

-Woodall, B. P., Gresham, K. S., Woodall, M. A., Valenti, M., Cannavo, A., Pfleger, J., Chuprun, J. K., Drosatos, K., & Koch, W. J. (2019). Alteration of myocardial GRK2 produces a global metabolic phenotype. *JCI Insight*, *4*(10). https://doi.org/10.1172/jci.insight.123848

Regulation of Substrate Oxidation in Isolated Myocardial Cells by β-Hydroxybutyrate

V. Chen, G. Wagner and J.J. Spitzer

Department of Physiology, Louisiana State University Medical Center, New Orleans, Louisiana, U.S.A.

Table 1 The effect of βOHB on lactate, octanoate or palmitate oxidation by isolated myocytes

βOHB concentration (mM)	0	0.4	1	2	5
Lactate (7.5 mM) oxidation; N=10	6.01 ± 0.30	---	4.14 ± 0.31(−30%)[c]	2.96 ± 0.31(−50%)[c]	2.38 ± 0.34(−60%)[c]
Octanoate (2 mM) oxidation; N=8	1.61 ± 0.05	---	1.50 ± 0.05(−7%)	1.44 ± 0.06(−10%)	1.34 ± 0.05(−17%)[b]
Palmitate (0.4 mM) oxidation; N=5	0.92 ± 0.09	0.73 ± 0.08(−21%)[a]	0.60 ± 0.07(−35%)[c]	---	0.60 ± 0.09(−35%)[b]
Palmitate (1 mM) oxidation; N=5	1.19 ± 0.09	0.82 ± 0.09(−31%)[b]	0.75 ± 0.07(−37%)[b]	---	0.73 ± 0.09(−39%)[b]

Values are means ± SE nmoles/mg protein/min

[a,b,c] $P < 0.05$, $P < 0.01$ and $P < 0.001$ vs 0 mM βOHB

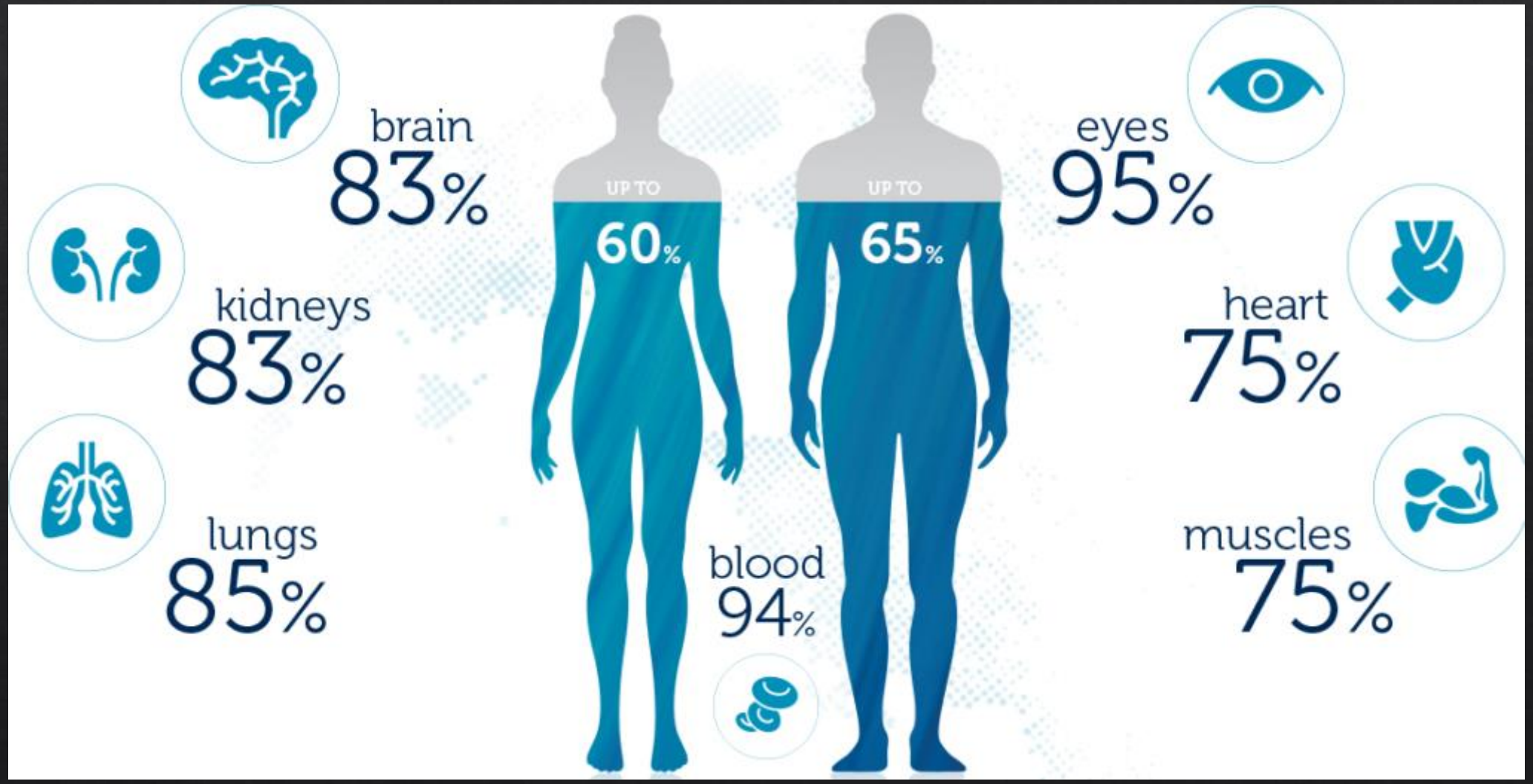
Summary

The role of ketone bodies in myocardial substrate oxidation was examined using freshly isolated Ca^{2+}-tolerant heart myocytes. β-hydroxybutyrate (βOHB) inhibited lactate oxidation by the myocytes by 30–60%, and the inhibition was concentration dependent. Palmitate oxidation was also markedly decreased, whereas octanoate oxidation was only minimally affected by the presence of βOHB. Lactate, octanoate, or palmitate had little, if any, effect on βOHB oxidation. βOHB oxidation was reduced by 22–28% in myocytes isolated from chronically diabetic rats, whereas the oxidation of palmitate remained similar to the controls. However, βOHB still inhibited palmitate oxidation to the same extent as in the control cells. Our data support the role of βOHB as a physiologic regulator of myocardial substrate metabolism.

Evolution was not accounting for the ANS imbalance

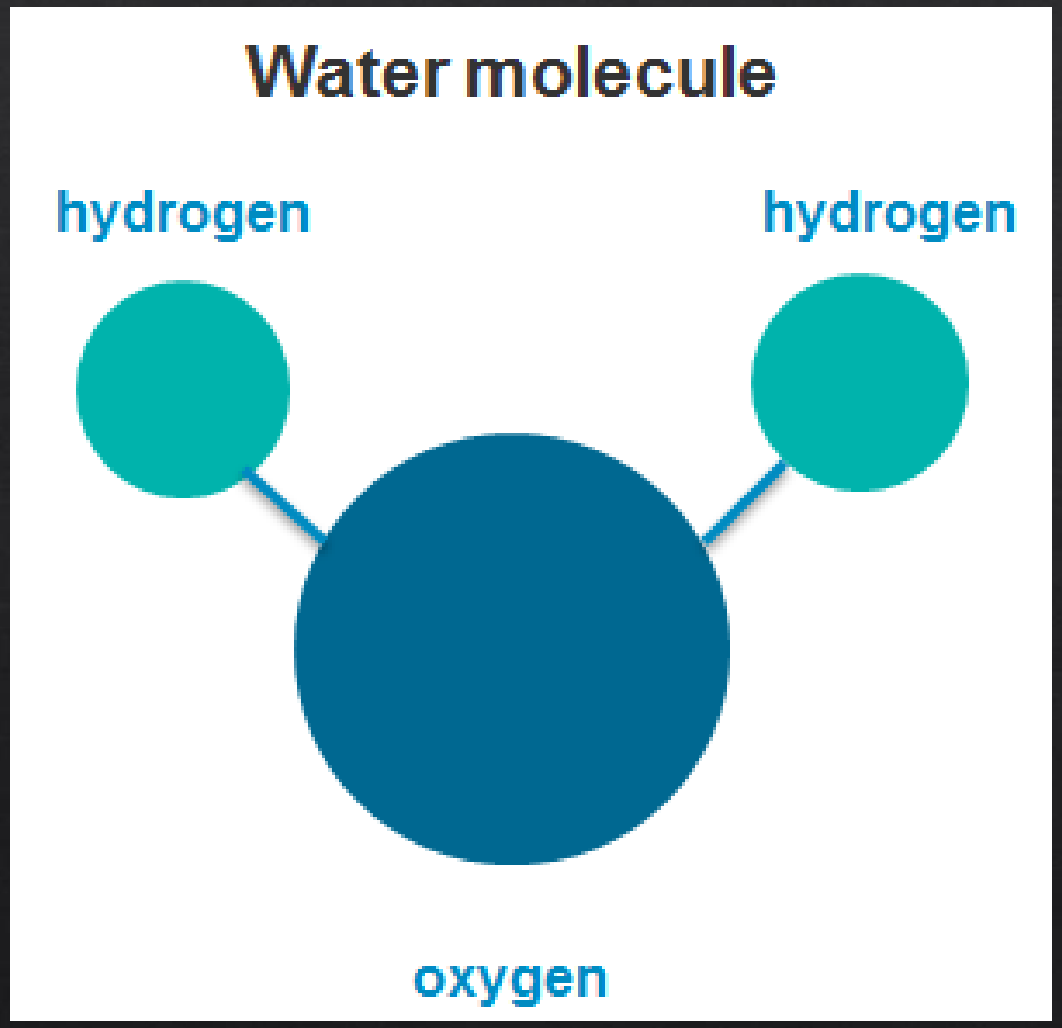
Biophysics/Quantum

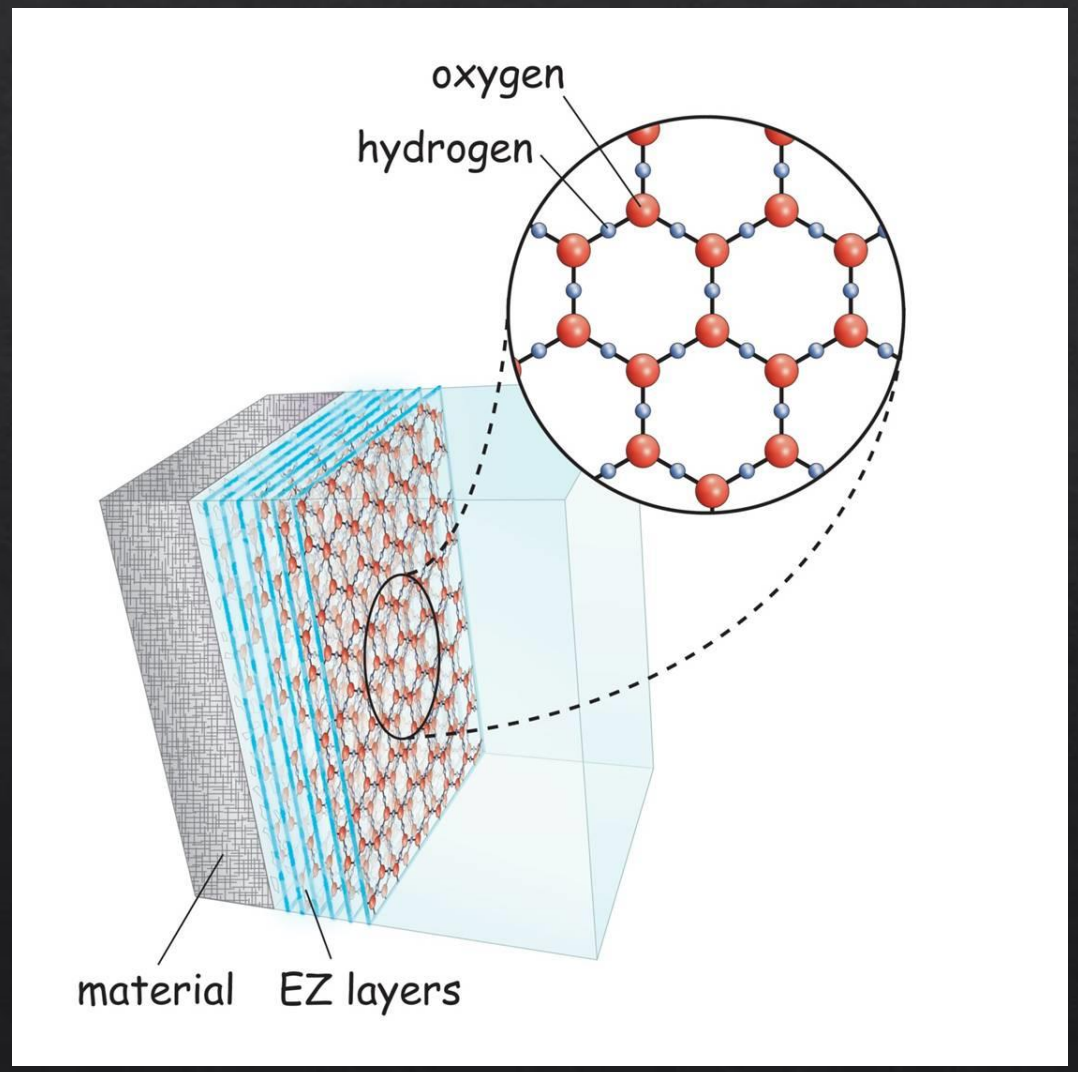
Human Body of Water



Exclusion Zone Water (water, hydrophilic surface, radiant energy)

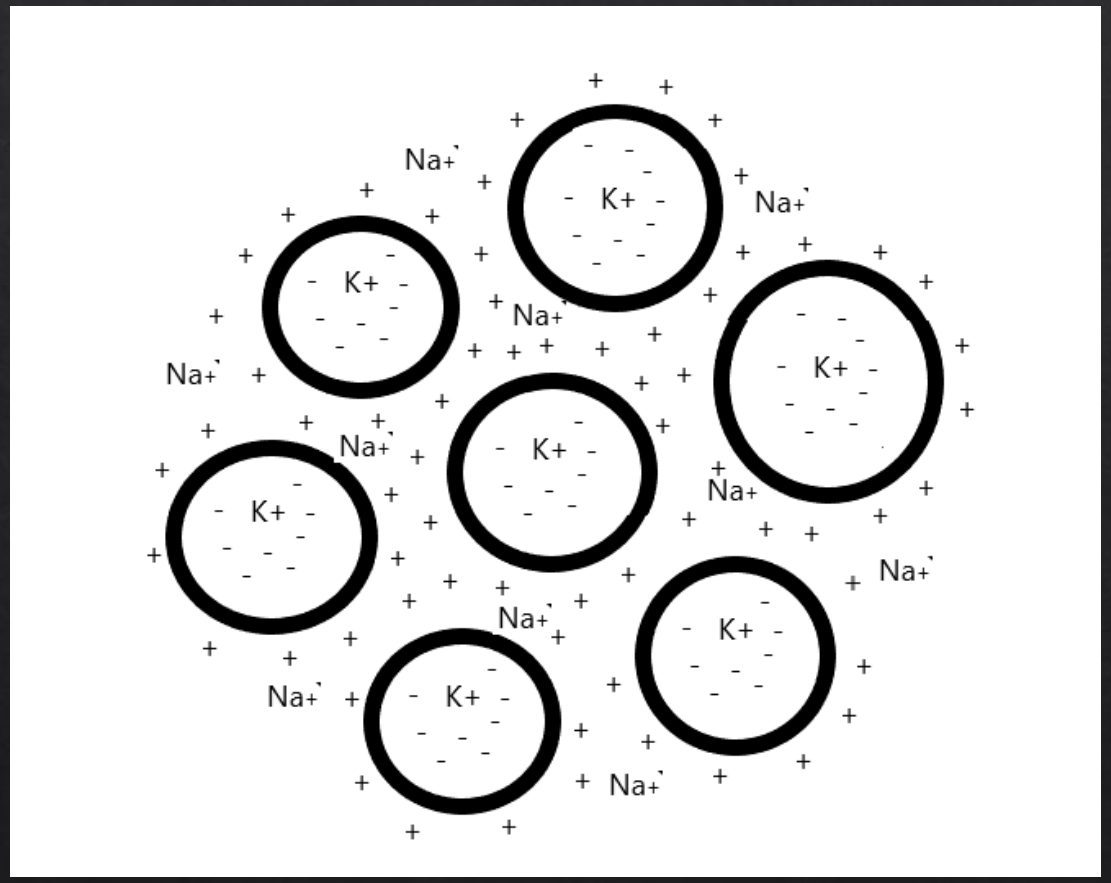
H_3O_2





Pollack, G.H. (2013) The fourth phase of water: Beyond solid, liquid, and vapor. Seattle, WA. Ebner & sons.

Hofmeister Series - $Mg^{2+} > Ca^{2+} > Na^{+} > K^{+} > Cl^{-} > NO_3^{-}$



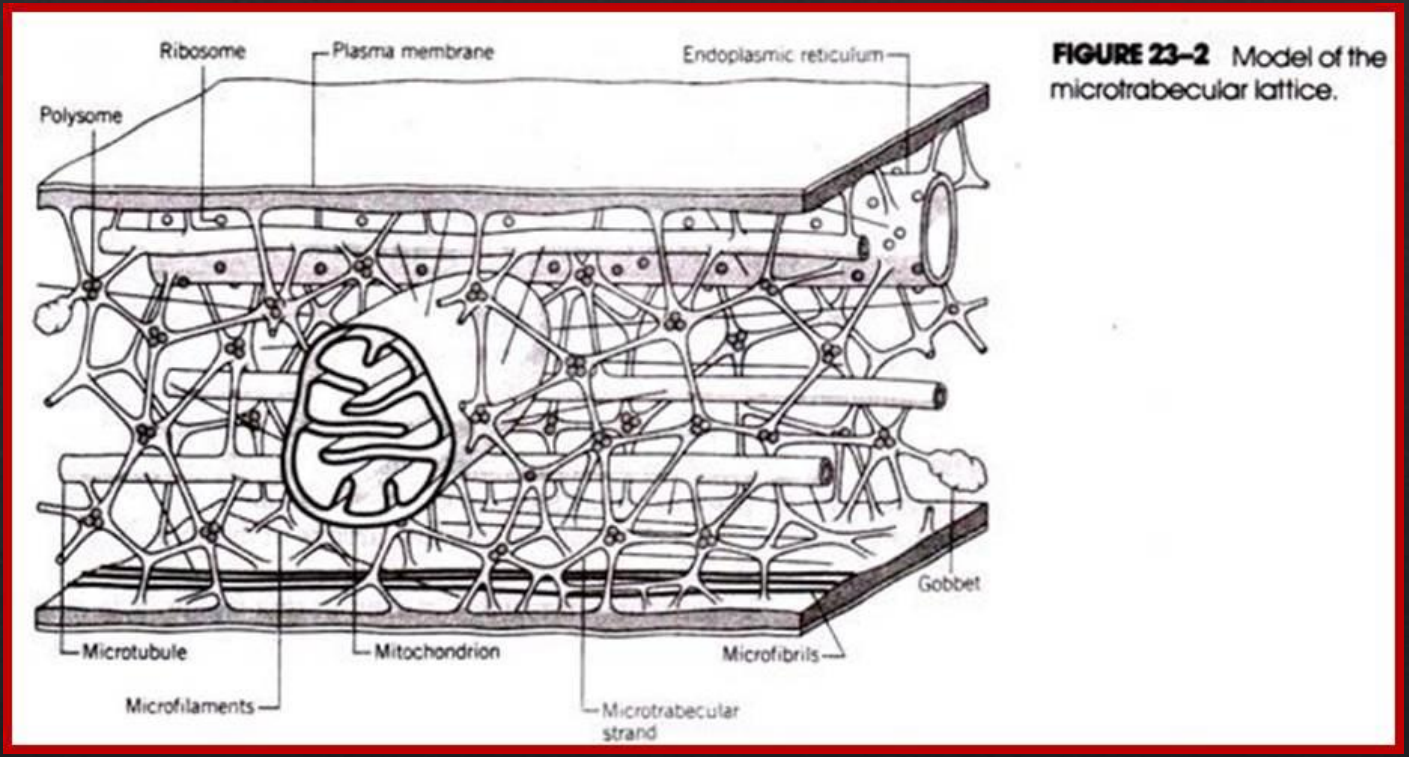
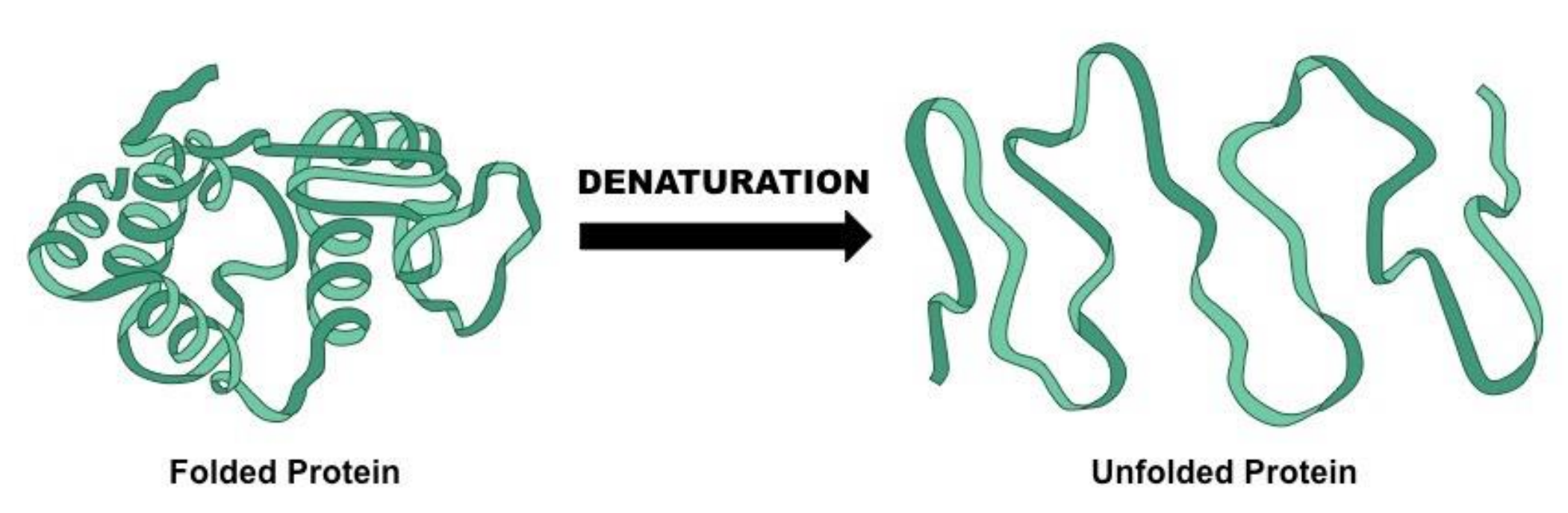


FIGURE 23–2 Model of the microtrabecular lattice.

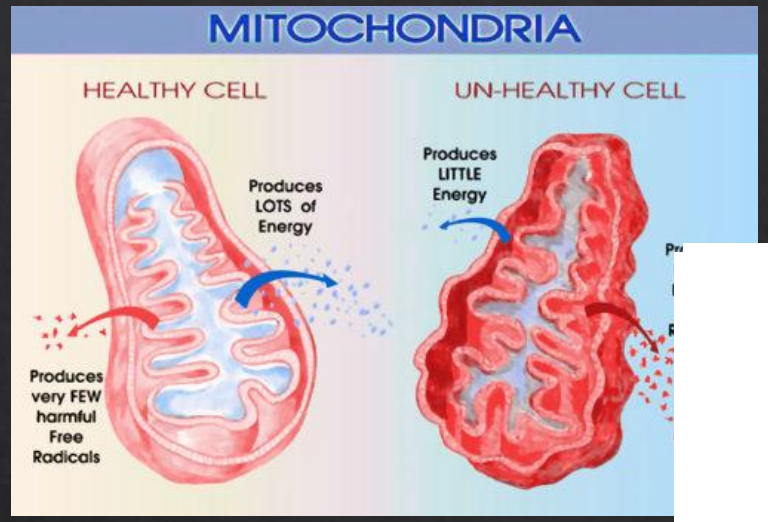
-Gershon, N. D., Porter, K. R., & Trus, B. L. (1985). The cytoplasmic matrix: its volume and surface area and the diffusion of molecules through it. Proceedings of the National Academy of Sciences, 82(15), 5030-5034. doi:10.1073/pnas.82.15.5030

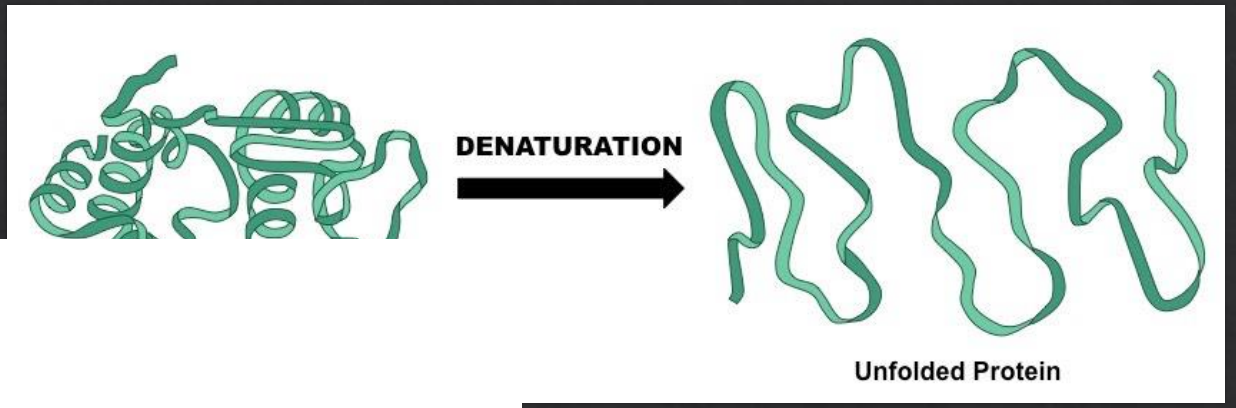
-Clegg, J. S. (2018). On the Internal Environment of Animal Cells. Microcompartmentation, 1-16. doi:10.1201/9781351074575-1

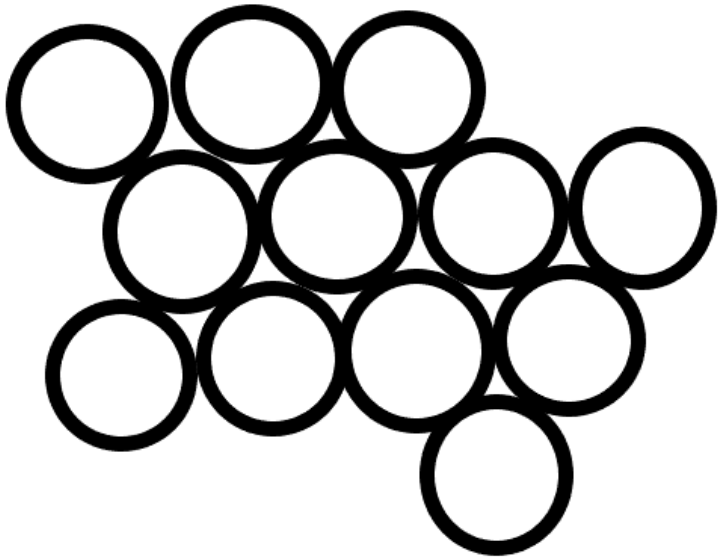
Maintaining unfolded proteins that form microtrabecular lattice (hydrophilic surface) requires lots of ATP

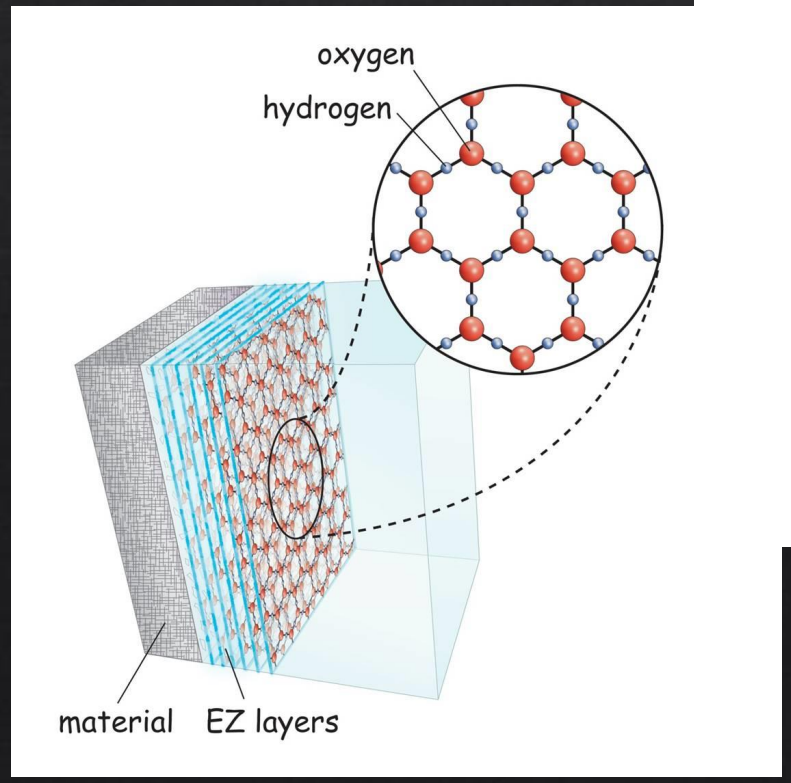


-López-Beltrán, E. A., Maté, M. J., & Cerdán, S. (1996). Dynamics and Environment of Mitochondrial Water as Detected by H NMR. Journal of Biological Chemistry, 271(18), 10648-10653. doi:10.1074/jbc.271.18.10648









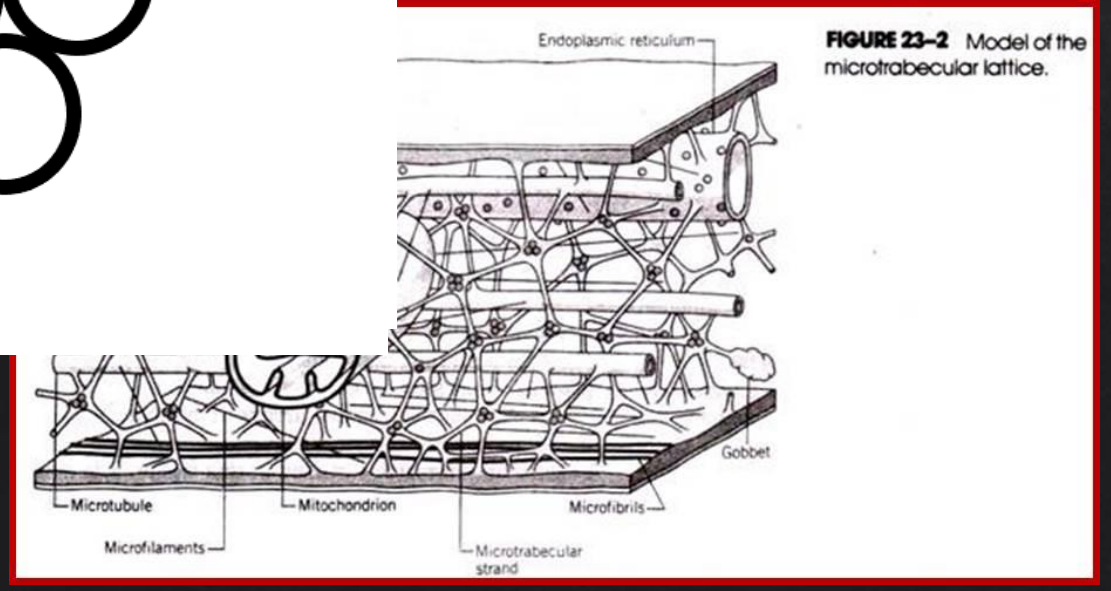


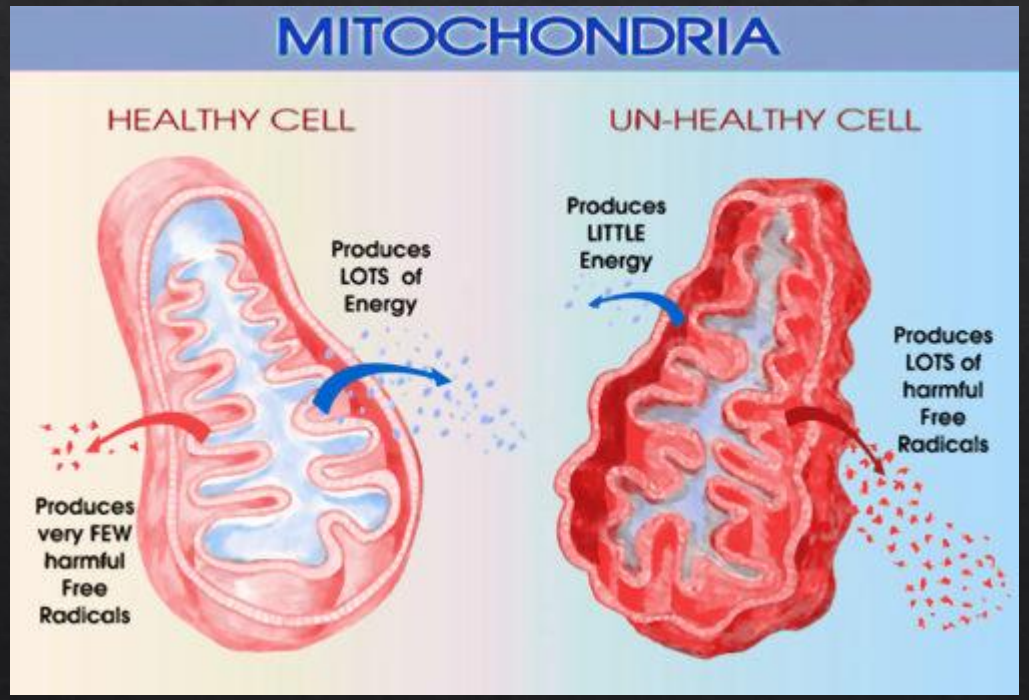
FIGURE 23–2 Model of the microtrabecular lattice.

“The irreversible injuring of respiration is followed, as the second phase of cancer formation, by a long struggle for existence by the injured cells to maintain their structure, in which a part of the cells perish from lack of energy, while another part succeeds in replacing the irretrievably lost respiratory energy by fermentation energy. Because of the morphological inferiority of fermentation energy, the highly differentiated body cells are converted by this into undifferentiated cells that grow wildly — the cancer cells.”

-Otto Warburg

Mitochondria

- Heart tissue is one of the densest with mitochondria
- Heart muscle tissue prefers fatty acids and ketones (more ATP)
- Presence of ketones has been shown to increase mitochondrial respiration, oxidative phosphorylation, by 128%



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The heart emits an electromagnetic field five thousand times stronger than the brain

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Research Article Volume 4 Issue 2

Exposure to electromagnetic fields induces oxidative stress and pathophysiological changes in the cardiovascular system

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duration, and prolonged P-R and QT-c intervals. A serious histopathological changes in the heart were seen in experimental animals exposed to EMFs, these changes includes increases the number of apoptotic cells, dark brown stain muscle fiber nuclei, marked cell vacuolation, hyperemia muscle fiber degeneration, distortion of some cardiac myocytes, mononuclear cellular infiltration and histological structure of the myocytes spaces were seen. Ultra structural of the myocardial tissue and sarcomere in experimental animals exposed to EMFs showed that lose of area in sarcomeres, irregular structural of myocardial cells, and ruptures of sarcomeres, lose of mitochondria cristae, blebs of mitochondria.

Conclusion: It can be concluded that exposure of human and experimental animals to EMFs have been a negative effect on the heart and blood vessels by causing a histopathological changes and disturbances in the functions of the organs of the cardiovascular system.

Heart Cancer Stats

- Vast majority of tumors originating in heart are benign
- Study: 12,000 heart cancer cases, 7 were primary cardiac tumors
- Dr. Robert Cusimono, cardiac surgeon, 12 cases a year
- Primary cardiac tumors are 0.3 to 0.7% of all cardiac tumors
- Metastasis from other tumors is 30 times more likely
- When malignant, survival rate at 9 to 12 months is only 10%

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3. Leja, M. J., Shah, D. J., & Reardon, M. J. (2011). Primary Cardiac Tumors. Texas Heart Institute Journal, 38(3), 261-262. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3113129/

Gradual inhibition of parasympathetic activity

Depletion of NO due to oxidative stress

Stressful event leading to sudden increase in sympathetic activity

Surge of adrenaline causes heart cells to revert to burning glucose instead of ketones

Burning glucose causes build-up of lactic acid in cardiac cells

Lactic acid prevents calcium from entering cells, therefore they cannot contract

Pressure build-up causes "paradoxical bulging"

This causes localized edema, dysfunctional cell walls, and necrosis.
HEART ATTACK!

https://www.resourceyourhealth.com/post/it-s-not-blockages-so-what-really-causes-heart-attacks

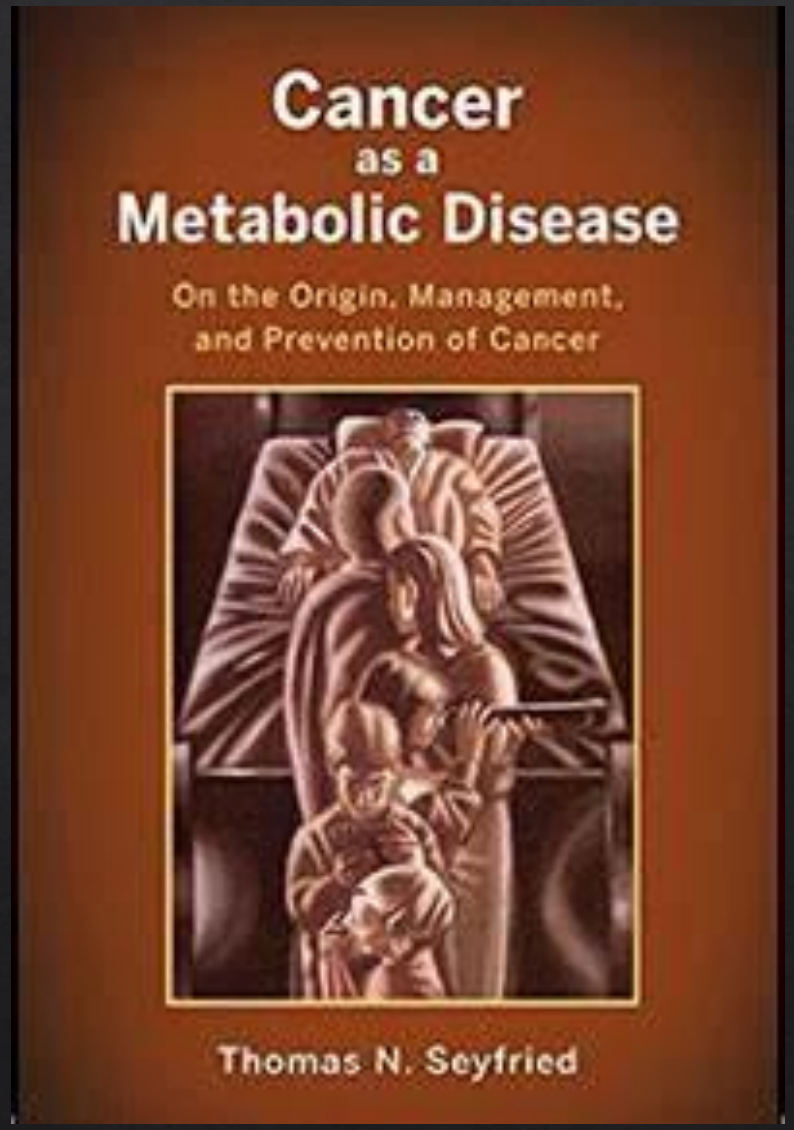
Preventing Metabolic Heart Attacks

Cellular Fermentation

Depletion of Nitric Oxide

ANS Imbalance

"Ketone bodies and fats are nonfermentable fuels in mammalian cells."



Oxidative metabolism of myocardium as influenced by fatty acids and epinephrine[1]

DAVID R. CHALLONER[2] AND DANIEL STEINBERG
Laboratory of Metabolism, National Heart Institute, National Institutes of Health, Bethesda, Maryland

RESULTS

β-Hydroxybutyrate (10 mM) was shown to increase oxygen uptake in both arrested and beating hearts (Table 1). The absolute magnitude of the increase was similar in the two preparations (arrested, +0.04; beating, +0.07 ml O_2/min per g dry wt). However, the percentage increment in the case of the beating hearts was much smaller and of marginal statistical significance.

Fluoroacetate (0.22 mM), an inhibitor of the Krebs cycle (32), reduced the qO_2 of the arrested heart by a small but statistically significant amount (Table 2, *exp. 1*). The addition of octanoate in the presence of fluoroacetate (Table 2, *exp. 2*), resulted in a highly significant elevation of qO_2, from 0.12 to 0.24 ml O_2/min per g dry wt. This stimulation was of the same magnitude as that seen previously with octanoate in the absence of fluoroacetate (8).

Oligomycin, an inhibitor of oxidative phosphorylation in mitochondria (13, 22, 26, 27), caused a highly significant decrease in oxygen consumption in the beating heart (Table 3, *exp. 1*; Fig. 1). This was accompanied by a

Octanoate stimulated qO_2 in the presence of oligomycin (Table 3, *exp. 3*). The magnitude of this stimulation was similar to that seen previously with octanoate in the absence of oligomycin (8). The octanoate effect in the

TABLE 1. *Effect of β-hydroxybutyrate on qO_2 of beating and arrested perfused rat hearts*

Exp.	β-Hydroxybutyrate, 10 mM	qO_2, ml/min per g dry wt	N	P
1 (Beating)	—	0.51±0.02	8	0.06
	+	0.58±0.03		
2 (Potassium-arrested)	—	0.14±0.01	7	<0.01
	+	0.18±0.01		

In each experiment every heart served as its own control, being perfused for 15 min with one solution and then 15 min with the other. The sequence of the two perfusion solutions being compared was alternated on successive hearts.

TABLE 3. *Effect of oligomycin with and without octanoate on qO_2 of the perfused rat heart*

Exp.	Perfusate Additions: Oligomycin, μg/ml	Perfusate Additions: Octanoate, 2 mM	qO_2, ml/min per g dry wt	N	P
1* (Beating)	—	—	0.40±0.02	6	≪0.001
	5	—	0.20±0.01	8	
2 (Arrested)	—	—	0.17±0.01	6	0.5
	5	—	0.18±0.01		
3 (Arrested)	5	—	0.20±0.01	6	<0.002
	5	+	0.29±0.01		

Strategies to Improve Metabolic Health

- Exercise (specifically resistance training and HIIT)
- Air Filter
- Avoid Toxins and Chemicals
- Avoid Artificial Sweeteners
- Minimize Endotoxemia
- Get Enough Mineral Salt
- Get Enough Sleep
- Don’t Be Sedentary
- Intermittent Fasting
- Reduce stress
- Diet (avoid seeds oils, excess fructose, processed grain, and processed sugar)

Preventing Metabolic Heart Attacks

~~Cellular Fermentation~~

Depletion of Nitric Oxide (Oxidative Stress)

ANS Imbalance

Article

β-Hydroxybutyrate Elicits Favorable Mitochondrial Changes in Skeletal Muscle

Brian A. Parker [1], Chase M. Walton [1], Sheryl T. Carr [1], Jacob L. Andrus [1], Eric C. K. Cheung [1], Michael J. Duplisea [1], Esther K. Wilson [1], Carrie Draney [2], Daniel R. Lathen [2], Kyle B. Kenner [2], David M. Thomson [1], Jeffery S. Tessem [2] and Benjamin T. Bikman [1,*]

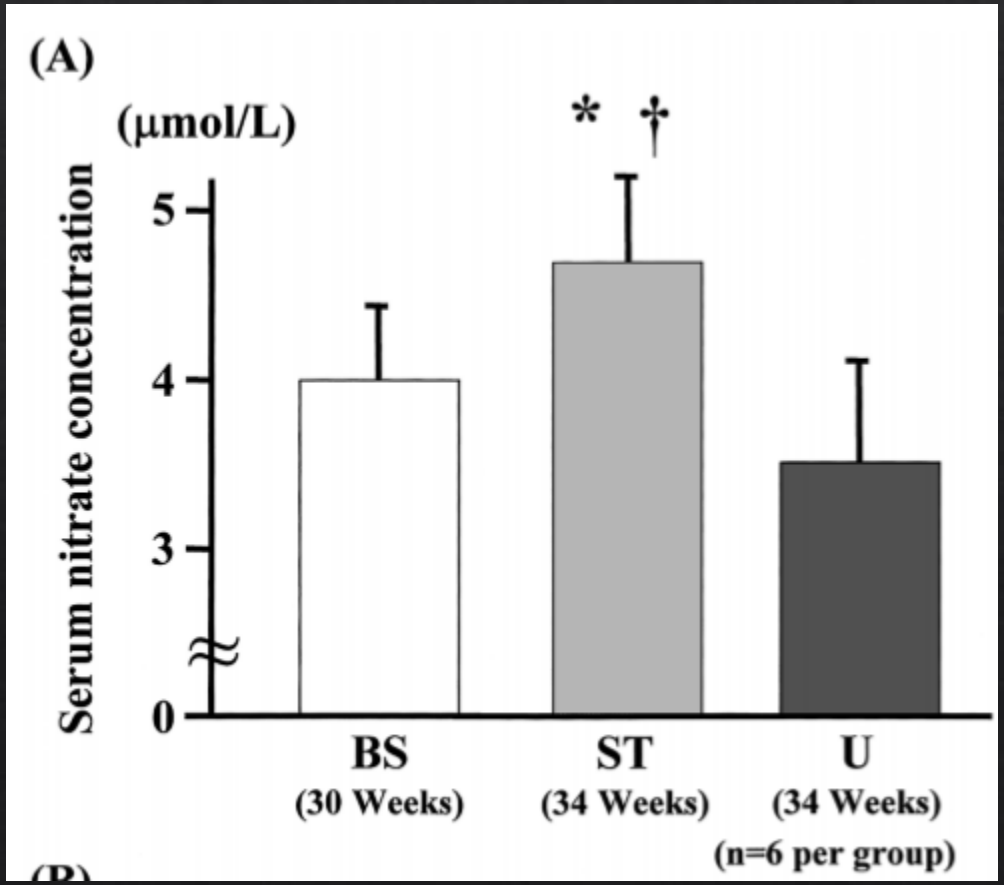
β-hydroxybutyrate (β-HB) on muscle cell mitochondrial physiology. In addition to increased cell viability, murine myotubes displayed beneficial mitochondrial changes evident in reduced H_2O_2 emission and less mitochondrial fission, which may be a result of a β-HB-induced reduction in ceramides. Furthermore, muscle from rats in sustained ketosis similarly produced less H_2O_2 despite an increase in mitochondrial respiration and no apparent change in mitochondrial quantity. In sum,

EXPERIMENTAL INVESTIGATIONS

Circ J 2005; **69:** 722–729

Repeated Sauna Therapy Increases Arterial Endothelial Nitric Oxide Synthase Expression and Nitric Oxide Production in Cardiomyopathic Hamsters

Yoshiyuki Ikeda, MD; Sadatoshi Biro, MD; Yasuyuki Kamogawa, MD; Shiro Yoshifuku, MD; Hideyuki Eto, MD; Koji Orihara, MD; Bo Yu, MD; Takashi Kihara, MD; Masaaki Miyata, MD; Shuichi Hamasaki, MD; Yutaka Otsuji, MD; Shinichi Minagoe, MD; Chuwa Tei, MD



Repeated Thermal Therapy Improves Impaired Vascular Endothelial Function in Patients With Coronary Risk Factors

Masakazu Imamura, MD,* Sadatoshi Biro, MD,* Takashi Kihara, MD,* Shiro Yoshifuku, MD,* Kunitsugu Takasaki, MD,* Yutaka Otsuji, MD, FACC,* Shinichi Minagoe, MD,* Yoshifumi Toyama, MD† Chuwa Tei, MD, FACC*

Kagoshima, Japan

Table 2. Changes in Clinical Parameters After Two Weeks of Sauna Treatment

Conclusions. Repeated thermal therapy improves impaired endothelial function in patients with coronary risk factors, suggesting a preventive role for thermal therapy for atherosclerosis.

Hematocrit (%)	47.6 ± 2.9	47.2 ± 2.3	NS
Total cholesterol (mg/dl)	214 ± 44	208 ± 34	NS
Triglyceride (mg/ml)	268 ± 327	221 ± 159	NS
HDL cholesterol (mg/dl)	51 ± 11	50 ± 11	NS
Uric acid (mg/dl)	6.8 ± 1.8	6.6 ± 1.5	NS
Fasting plasma glucose (mg/dl)	99 ± 25	94 ± 16	<0.05
TBARS (nmol/ml)	2.8 ± 0.6	2.9 ± 0.6	NS
Resting arterial diameter (mm)	3.9 ± 0.3	3.9 ± 0.3	NS
Reactive hyperemia (%)	398 ± 170	352 ± 215	NS
%FMD (%)	4.0 ± 1.7	5.8 ± 1.3	<0.001
%NTG (%)	18.7 ± 4.2	18.1 ± 4.1	NS

Values are expressed as the mean ± SD.

HDL cholesterol = high-density lipoprotein cholesterol; TBARS = thiobarbituric acid reactive substances; %FMD = percentage of flow-mediated dilation; %NTG = percentage of nitroglycerin-induced dilation.

Preventing Metabolic Heart Attacks

~~Cellular Fermentation~~

~~Depletion of Nitric Oxide~~

ANS Imbalance

Balance the Autonomic Nervous System

- Sunlight/Infrared Sauna
- Proper Sleep
- Laughter
- Positive Social Relationships
- Massage
- Singing
- Deep Breathing
- Chewing Food Thoroughly
- Acupuncture
- Yoga
- Tai Chi
- Meditation
- Prayer
- Fasting
- Eat Enough Omega-3's
- Heal the Gut
- Gargling
- Acupuncture
- Stimulate Gag Reflex
- Cold Therapy
- Coffee Enemas
- Satiety
- Maintain Serotonin Levels (gut)
- PEMF Therapy
- Grounding
- Music
- High Fat, Metabolically Flexible Diet

Nichols, H. (2017, December 29). 32 Science-Backed Ways to Stimulate Your Vagus Nerve - Well-Being Secrets. Retrieved from https://www.well-beingsecrets.com/vagus-nerve-stimulation/

Omega-3 fatty acid deficiencies in neurodevelopment, aggression and autonomic dysregulation: Opportunities for intervention

JOSEPH R. HIBBELN, TERESA A. FERGUSON, & TANYA L. BLASBALG

National Institute on Alcohol Abuse and Alcoholism, Bethesda, USA

Omega-3 fatty acid tissue composition and heart rate variability

Several studies have suggested that omega-3 fatty acid supplementation leads to increased heart rate variability. Four double-blind placebo-controlled clinical trials of adult subjects have documented increases in heart rate variability after treatments with omega-3 fatty acids during 24-hour monitoring (Christensen et al., 1996, 1997, 1998; Christensen, Christensen, Dyerberg, & Schmidt, 1999). Even

currently using these fatty acids to treat their patients, while positive findings would indicate that omega-3 fatty acids can increase heart rate variability, which is associated with a decreased risk of sudden cardiac death. Positive findings would suggest that a low omega-3 level is an important factor linking depression and aggression to increased cardiovascular risk.

Effects of high-carbohydrate and high-fat dietary treatments on measures of heart rate variability and sympathovagal balance

Richard M. Millis [a,*], Rachel E. Austin [a], Vernon Bond [d], Mezbah Faruque [b], Kim L. Goring [c], Brian M. Hickey [e], Raymond Blakely [f], Ronald E. DeMeersman [g]

Conclusion

In summary, metabolism of isocaloric high-carbohydrate and high-fat beverages were found to increase resting energy expenditure and heart rate and shift cardiac sympathovagal balance toward greater sympathetic activation. The sympathetic response was differentiable by the type of test beverage with the high-carbohydrate beverage producing greater sympathetic modulation. Across a wide physiological range of respiratory quotients, indicative of the relative oxygen consumption-carbon dioxide production rates, the metabolism of a high-carbohydrate beverage was associated with relatively higher sympathetic modulation of heart rate, resting energy expenditure and respiratory quotient. The metabolism of stored and ingested fat was associated with relatively lower sympathetic modulation, energy

Table 2
Effects of high-carbohydrate and high-fat treatments.

Pretreatment control (n=12)		Range	Mean±SD
Heart rate variability low frequency/high frequency ratio		0.21–0.70	0.37±0.18
Heart rate (bpm)		47.5–80.0	61.9±11.5
Respiratory quotient		0.67–0.85	0.74±0.05
Resting energy expenditure (Cal/d)		1692–2656	2080±422
Axillary body temperature (°F)		95.2–97.9	96.7±0.8
Systolic arterial pressure (Torr)		103–153	129±15
Diastolic arterial pressure (Torr)		57–81	70±6
High-carbohydrate (n=6)	**Range**	**Mean±SD**	***P*-value***
HRV low frequency/high frequency	0.64–1.83	1.18±0.43	0.011*
Heart rate (bpm)	69.5–86.1	78.5±7.2	0.001*
Respiratory quotient	0.93–0.99	0.96±0.05	<0.001*
Resting energy expenditure (Cal/d)	1873–3114	2599±471	0.001*
Axillary body temperature (°F)	96.6–97.3	97.2±0.4	0.654
Systolic arterial pressure (Torr)	125–150	133±9	0.208
Diastolic arterial pressure (Torr)	61–81	72±8	0.181
High-fat (n=6)	**Range**	**Mean±SD**	***P*-value**
HRV low frequency/high frequency	0.45–1.59	0.86±0.43	0.008*
Heart rate (bpm)	67.1–86.5	74.5±8.3	<0.001*
Respiratory quotient	0.74–0.91	0.80±0.05	0.010*
Resting energy expenditure (Cal/d)	1699–2851	2398±498	0.008*
Axillary body temperature (°F)	96.6–97.5	97.0±0.4	0.426
Systolic arterial pressure (Torr)	117–156	134±14	0.662
Diastolic arterial pressure (Torr)	67–83	75±7	0.018*

SD=standard deviation.
*Difference from control statistically significant at $P \leq 0.05$.

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Complementary Therapies in Medicine

Volume 45, August 2019, Pages 190-197

Recovery from sauna bathing favorably modulates cardiac autonomic nervous system

Tanjaniina Laukkanen [a, b], Jukka Lipponen [c], Setor K. Kunutsor [d, e], Francesco Zaccardi [f], Claudio Gil S. Araújo [g], Timo H. Mäkikallio [h], Hassan Khan [i], Peter Willeit [j], Earric Lee [k], Sanna Poikonen [l], Mika Tarvainen [c], Jari A. Laukkanen [a, b, k]

Conclusions

This study demonstrates that a session of sauna bathing induces an increase in HR. During the cooling down period from sauna bathing, HRV increased which indicates the dominant role of parasympathetic activity and decreased sympathetic activity of cardiac autonomic nervous system. Future randomized controlled studies are needed to show if HR and HRV changes underpins the long-term cardiovascular effects induced by regular sauna bathing.

Preventing Metabolic Heart Attacks

~~Cellular Fermentation~~

~~Depletion of Nitric Oxide~~

~~ANS Imbalance~~

Summary

- Cancer of the heart is one of the rarest cancers.
- This is in large part due to the unique metabolism of the heart combined with the inability of the heart cells to divide.
- However, a forced change in the preferred metabolism of the heart could lead to something acutely worse than cancer, myocardial infarction.
- The presence and preference of ketones seems to be protective (metabolically and biophysically) for the heart.
- A metabolically flexible diet and radiant energy exposure can help balance the imbalances that predispose us to metabolic heart attacks.

Take home

- Heart disease and heart attacks are about much more than cholesterol/LDL.
- Understanding why the heart is resistant to a certain pathology is just as important as understanding why it commonly gets another pathology.
- This gives us new insights into heart metabolism, preventing heart disease, and creating overall health.
- Eat in a way that keeps ketones around, expose your body to radiant energy, and keep your body in an electromagnetically appropriate environment.
- Ferment your food, not your cellular fuel!

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