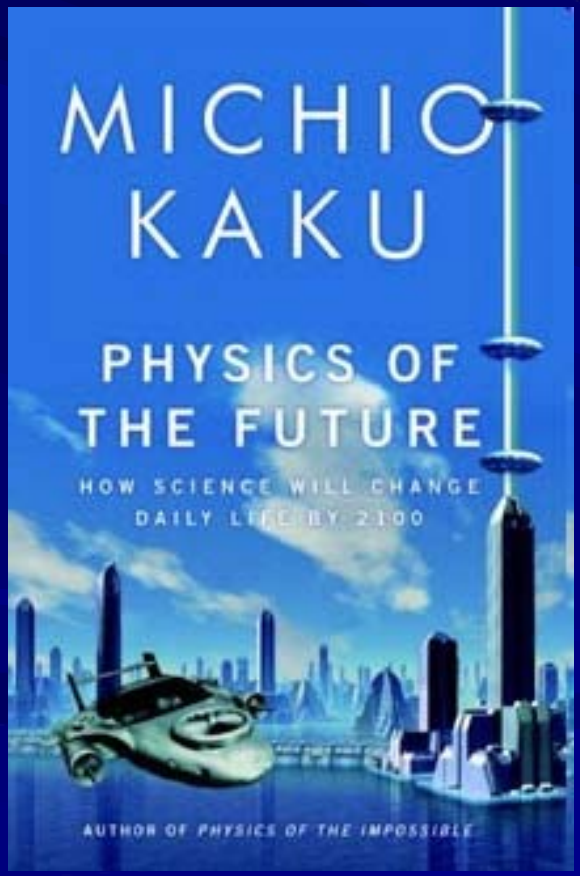
Dr Gary Gordon

MD DO MD(H)

Energize the Brain Heal the Body

Dr. Garry F. Gordon, MD, DO, MD(H)
Gordon Research Institute (GRI)
Payson, Arizona USA

Anti-Ageing Conference London (AACL)
September 19 – 21, 2013



Based on interviews with over three hundred of the world's top scientists, who are already inventing the future in their labs, Kaku—in a lucid and engaging fashion—presents the revolutionary developments in medicine, computers, quantum physics, and space travel that will forever change our way of life and alter the course of civilization itself.

Dr. Kaku's astonishing revelations include:

Sensors in your clothing, bathroom, and appliances will monitor your vitals, and nanobots will scan your DNA and cells for signs of danger, allowing life expectancy to increase dramatically.

You will control computers and appliances via tiny sensors that pick up your brain scans.

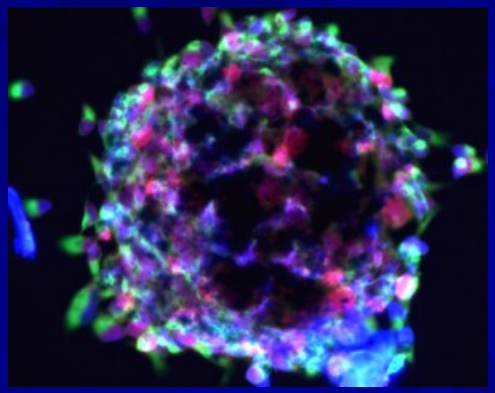
http://mkaku.org/home/?p=988

FutureMed
SINGULARITY UNIVERSITY

Exploring and driving the future of medicine

and the development of exponentially advancing technologies to address humanity's challenges

Cancer Treatments Made to Order: *Personalized Treatment for Cancer with frequent monitoring, captures and sequences malignant cells, detects changes and optimizes therapy*



Daily Test Compares Gene Expression Before, After Exercise: *The $100 genome and related technologies will increase understanding and use of the base genomic code and the ability to inexpensively measure gene expression in normal and diseased tissues*

Toothbrush upgrade – Monitoring chronic illness and disease through Exhaled Breath**:** *as capabilities of equipment only found in hospitals and laboratories, more test devices will have a size and price range that makes them practical for home use.*

Elizabeth Holmes: The Breakthrough of Instant Diagnosis – A Drop of Blood

THE WALL STREET JOURNAL.
U.S. EDITION Sunday, September 8, 2013 As of 12:04 PM EDT

by Joseph Rago

Ms. Elizabeth Holmes, a 29-year-old chemical and electrical engineer and entrepreneur, dropped out of Stanford as an undergraduate after founding a life sciences company called Theranos in 2003. Her inventions could upend the industry of laboratory testing and might change the way we detect and treat disease.

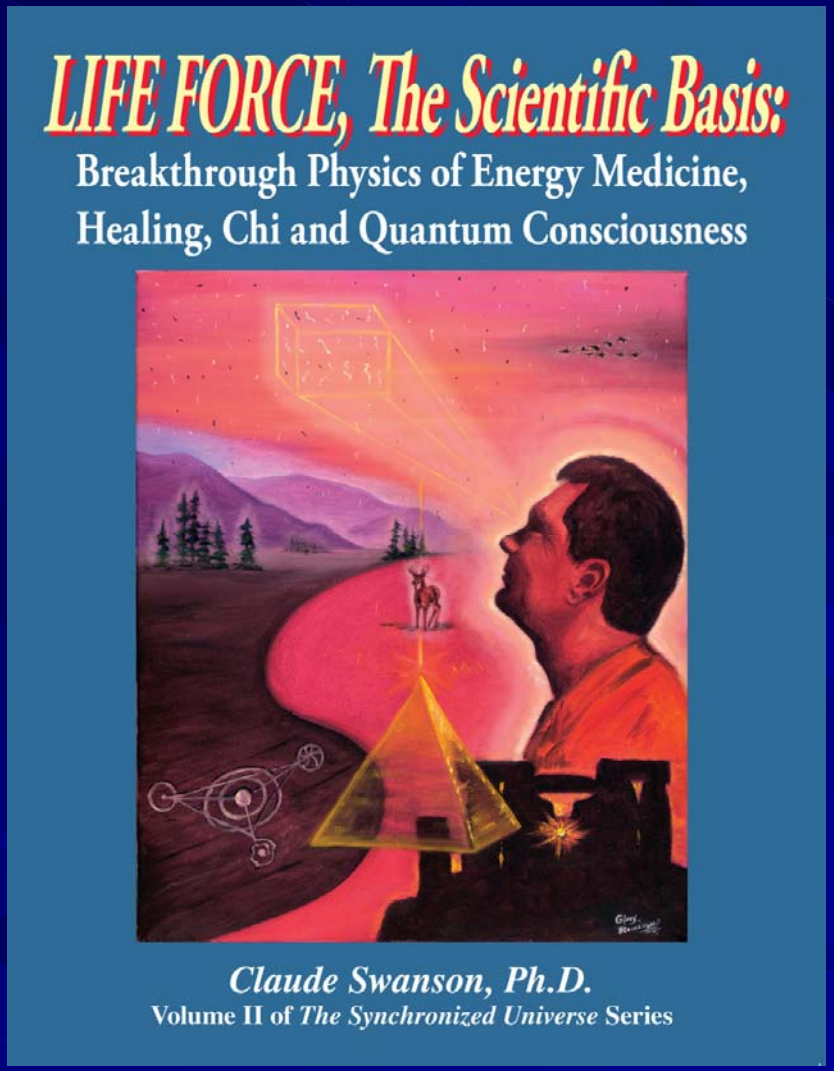
Theranos devices automate and miniaturize more than 1,000 laboratory tests, from routine blood work to advanced genetic analyses. **Theranos's processes are faster, cheaper and more accurate than the conventional methods and require only microscopic blood volumes, not vial after vial of the stuff.**

A Theranos technician first increases blood flow to your hand by applying a wrap similar to one of those skiing pocket warmers, then uses a fingerstick to draw a few droplets of blood from the capillaries at the end of your hand. The blood wicks into a tube in a cartridge that Ms. Holmes calls a "nanotainer," which holds microliters of a sample, or about the amount of a raindrop. The nanotainer is then run through the analyzers in a Theranos laboratory.

Results are usually sent back to a physician, but a full blood work-up—metabolic and immune markers, cell count, etc.—was in my inbox by the time I walked out the door.

http://online.wsj.com/article/SB10001424127887324123004579055003869574012.html

Life Force – A scientific revolution in Energy Medicine!



CH11-BIOPHOTONS, ENTANGLEMENT AND DNA

- The Biophoton Intention Experiment
- Discovery of Biophotons
- Herbert Frohlich and the Body Electromagnetic
- DNA -The Blueprint of Life....
- Laser Light from the Cells DNA
- Biophotons and Negentropy
- Flat Biophoton Spectrum
- Early Cancer Detection with Biophotons, XE "biophotons

http://synchronizeduniverse.com/

How Pig Guts Became the Next Bright Hope for Regenerating Human Limbs

Discover — SCIENCE FOR THE CURIOUS

By Adam Piore, Scott Lewis
Monday, September 26, 2011

The strange sensation in his right thigh muscle began as a faint pulse. Slowly, it was becoming more pronounced. Some people would have thought it impossible, but Cpl Isaias Hernandez could feel his quadriceps getting stronger.
The muscle was growing back.

A remarkable substance extracted from pigs enables the body to regenerate lost tissue, including fingertips and big chunks of muscle. They are called cryptic peptides, or "crypteins," and explain much of ECM's unique regenerative phenomena.

The peptides have potent antimicrobial effects and important signaling abilities… during scaffold breakdown, information held within the structural ECM molecules, recruit all-purpose "stem cells" that can develop into any type of tissue.

Biological scaffolds made of extracellular matrix, or ECM; the cylinder at far left mimics the shape of the trachea.

http://discovermagazine.com/2011/jul-aug/13-how-pig-guts-became-hope-regenerating-human-limbs#.Uik1lT968ng

Your genomic future:
Personalised Medicine is Here

05 September 2013 by Peter Aldhous

NewScientist

Only six other children in the world are known to have the same condition as Lillian Yuska *(Image: Children's Hospital of Wisconsin)*

Thanks to genome sequencing, parents Danielle and Erik have a name for the mysterious condition that they feared would take the life of their 7-year-old daughter, Lillian, and they have an idea of what her outlook might be.

Born prematurely, Lillian struggled to feed, suffered from chronic vomiting and diarrhoea, and succumbed to repeated infections. After shuttling for years from specialist to specialist, the Yuskas now know that Lillian has trichohepatoenteric syndrome-2, caused by a mutation in a gene ...

Genome sequencing is bringing a medical revolution for families with rare diseases, and the rest of us will benefit too.

Dr. Garry F. Gordon, MD, DO, MD(H)

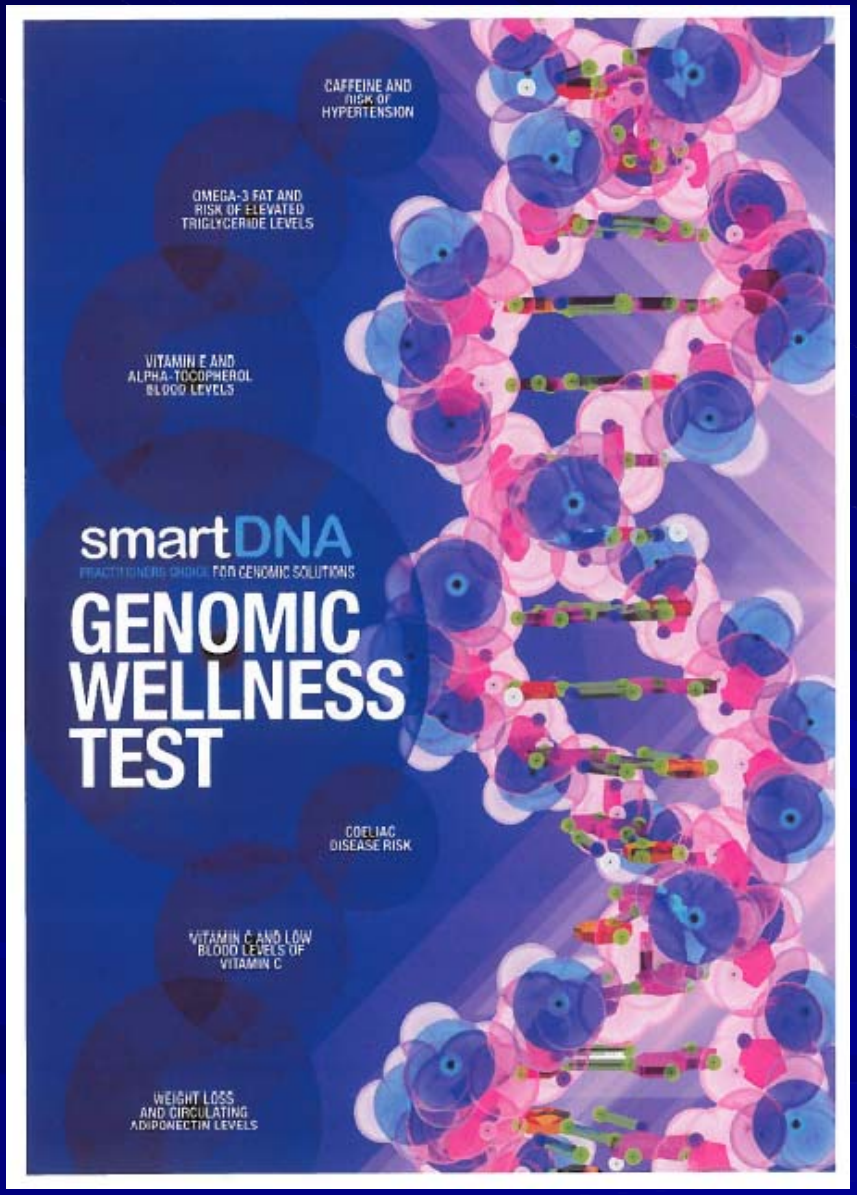
- President of Gordon Research Institute
- Doctor of Osteopathy 1958, Chicago College of Osteopathy
- Honorary MD 1962, University of California Irvine
- Radiology Residency 1964, Mt. Zion, San Francisco
- "Father of Chelation Therapy"
- Past Board Member of Arizona Homeopathic Medical Examiners
- Co-Founder of the American College for Advancement in Medicine (ACAM)
- Past Medical Director of Mineralab
- Board of Directors Member for IOMA (International Oxidative Medicine Association)
- Treasurer AHIMA (Arizona Homeopathic Integrative Medical Association)
- Author of numerous books including latest entitled "Detox With Oral Chelation"

Personal Health History

I became interested in Chelation therapy because I was very ill for the first 30 years of my life. As a young man, I had not been able to be athletic in any way and was not allowed to participate in physical education or sports.

I have suffered from a myriad of debilitating, chronic conditions, genetic and environmental in origin… including:

- Congenital Atrioventricular Block (CAVB)
- Achlorhydria (Hypochlorhydria) with associated malabsorption
- Severe Magnesium Deficiency
- Mercury Toxicity (dental amalgams)
- Chronic Fatigue and Myalgia
- Strabismus (concomitant estropia, or “cross-eyed”)
- Pathologic disfluency (“stuttering”)
- Atrial Fibrilation (AF)



PATIENT NAME: Dr Garry Gordon
PATIENT DOB: 3-01-1935
PATIENT SEX: Male

Test results and gene summary

Patient Name: Dr Garry Gordon
Patient ID Code: 5567
Aliquot Number: 447
Patient DOB: 3-01-1935
Submission Number: SDNMS274577
Patient Gender: Male
Specimen Source: Saliva
Clinic Address: 600 N. Beeline Hwy Payson AZ 85541 USA
Requesting Practitioner: Dr Garry Gordon
Sample Collected: 1-08-2013
Sample Received: 28-08-2013
Sample Reported: 13-09-2013

IMPORTANT NOTIFICATION FOR PRACTITIONERS: The Action Steps contained within this report are provided as guide for practitioners to discuss and review with their clients. The practitioner should consider the overall health status of their client before making recommendations.

Support Definitions

STAY BALANCED	No risk allele has been inherited
MODERATE RISK	One risk allele has been inherited which has affected the enzyme activity.
HIGH RISK	One or both risk alleles have been inherited with known effects on enzyme activity.
GENE x NUTRIENT INTERACTION	Outcome is dependent on dietary intake

Lipid Metabolism

Gene and SNP ID	Genotype / Haplotype	Result and Interpretation	Action steps and comments
Lipid Metabolism			
APOE rs429358	CT	HIGH CARDIOVASCULAR DISEASE RISK – LMT C.1 The APOE E3/E4 genotype	• Review Table 1 in relation to soluble fibre, fish oil, energy sources, effects of alcohol and exercise for individuals with this genotype. • If the individual smokes they should stop. • Alcohol may raise LDL-C, decrease HDL-C/HDL2 and increase sdLDL formation. This effect is stronger for males. This should be assessed via cholesterol profile monitoring. • Olive oil has been reported to increase sdLDL formation in this APOE 3/4 genotype. This should be assessed and monitored via cholesterol profile. • Low saturated fat intake and reduced intake of processed carbohydrates and foods containing high amounts of antioxidants should be considered. • Supplementation with omega-3-fatty acids is recommended, however fish oil has been reported to suppress HDL-C and raise calculated LDL-C. • If statins are prescribed then supplement with Co-enzyme Q10. • Niacin has been reported to lower triglyceride levels. • Plant sterols and soluble fibre have been reported to have beneficial effects.
APOE rs7412	CC		

PATIENT NAME: Dr Garry Gordon
PATIENT DOB: 3-01-1935
PATIENT SEX: Male

Lipid Metabolism - HDL

Gene and SNP ID	Genotype / Haplotype	Result and Interpretation	Action steps and comments
PUFA Dietary Fat			
APOA1 rs670	AG	HIGHER HDL-C level in the blood.	• From this individual's cholesterol profile determine if their HDL-C level is protective, if it is NOT protective, • then increase PUFA intake to >8 % of calories . • Monitor the individuals HDL-C blood level with a cholesterol profile. • Review the LPL, LIPC, and CETP haplotype in this section of the report in relation to increasing HDL-C and APOA1 levels via exercise.
Saturated Fats			
LPL rs320	TT	HIGHER HDL-C levels in the blood in response to lower dietary fat intake.	• From this individual's cholesterol profile determine if their HDL-C level is protective, if it IS NOT then • Review the APOA1 genotype action steps. • Review dietary fat intake. Lower dietary saturated fat intake will elevate HDL-C level. • Review the LPL, LIPC, and CETP haplotype in relation to increasing HDL-C and APOA1 levels via exercise.
LPL rs328	CC		
HDL-C level			
ABCA1 rs2230806	GG	LOWER HDL-C level in the blood.	• From this individual's cholesterol profile determine if their HDL-C level is protective, if it IS NOT then • Review the APOA1 genotype actron steps. • Review dietary fat intake. Lower dietary saturated fat intake will elevate HDL-C level. • Review the LPL, LIPC, and CETP haplotype in relation to increasing HDL-C and APOA1 levels via exercise.
CETP rs5882	AG	HIGHER HDL-C level in the blood.	• From this individual's cholesterol profile determine if their HDL-C level is protective, if it IS NOT then • Review the APOA1 genotype action steps. • Review dietary fat intake. Lower dietary saturated fat intake will elevate HDL-C level.
CETP rs708272	AG		

		Result and interpretation	Action steps and comments
			• Review the LPL, LIPC, and CETP haplotype in relation to increasing HDL-C and APOA1 levels via exercise.
Physiogenomic			
LPL rs10096633	CC	INCREASED HDL-C level and APOA1 level in response to exercise.	• From this individual's cholesterol profile determine if their HDL-C level is protective, if it IS NOT then • Review the APOA1 genotype action steps in relation to dietary PUFA intake. • Refer to Table 2 and Table 3 to review the increase gained in HDL-C level and APOA1 level when exercise is >8 METS per week when compared to <8 METS per week. • Exercise >8 METS per week is recommended to assist with elevating HDL-C and APOA1 level.
LIPC rs1800588	CT		
CETP rs1532624	AC		

Lipid Metabolism - LDL

Gene and SNP ID	Genotype / Haplotype	Result and interpretation	Action steps and comments
LDL-C level			
APOB rs693	AG	INCREASED LDL-C in response to dietary saturated fat intake.	• From a cholesterol profile review the LDL-C level, if the LDL level is elevated then, • Review dietary saturated fat intake with the individual and recommend other healthy sources of fats such as plant or fish. • Additional information may be sought from a Liposcan or VAP test in relation to the individual's formation of small dense LDL's and oxidised LDL subfractions.
APOB100 rs754523	AG		
LDL-R rs688	CT		

PATIENT NAME: Dr Garry Gordon
PATIENT DOB: 3-01-1935
PATIENT SEX: Male

Lipid Metabolism - Triglycerides

Gene and SNP ID	Genotype / Haplotype	Result and interpretation	Action steps and comments
Triglyceride level			
APOCIII rs5128	CC	Not associated with high triglyceride level.	• Stay balanced and focus on diet and lifestyle
APOA5 rs12286037	CT	INCREASED risk of hypertriglyceridemia	• From this individuals cholesterol profile determine if their triglyceride level is normal. If it exceeds normal limits then, • Review dietary saturated fat intake • Consider the measurement of small dense LDL's and oxidised LDL subfractions.
APOA5 rs662799	TT	Not associated with high triglyceride level.	• Review dietary fat intake since individuals with this genotype have been reported to increase their BMI as total fat intake is increased. Women and men are affected equally.
NOS3 rs1799983	GG	NOT associated with high triglyceride level in response to low omega-3 intake.	• From this individual's cholesterol profile determine if their triglyceride level is elevated, please note that • Omega-3 PUFA has been reported to have an attenuated response to reducing triglyceride concentrations.

Lipid Metabolism - Fat Absorption

Gene and SNP ID	Genotype / Haplotype	Result and interpretation	Action steps and comments
Fat Absorption			
FABP2 rs1799883	AG	Increased fat absorption.	• Recommend reducing dietary fat. • Increased fat absorption may increase the risk of being overweight. • Evaluate dietary saturated fat intake.

PATIENT NAME: Dr Garry Gordon
PATIENT DOB: 3-01-1935
PATIENT SEX: Male

Liver detoxification

Gene and SNP ID	Genotype / Haplotype	Result and interpretation	Action steps and comments
Phase I detoxification			
CYP1B1 rs1056836	GG	INCREASED risk for pro-carcinogen activation.	• Assess urinary estrogen metabolites that comprehensively measure 2, 4 and 16 hydroxylated estrogens. • Consider functional pathology to measure the 2 and 4 methoxylated estrogens and the important ratios between these substances. • Review the MTHFR and COMT enzymes since they are important, if both enzymes have reduced activity then phenotypically poor methylation of hydroxylates estrogens may occur. Reduced methylation results in the accumulation of fat soluble 4 hydroxy estrones.
CYP1A1_M1 rs4646903	TT	NORMAL CYP1A1_M1 enzyme activity.	• This enzyme can be promoted to remove hydrocarbons and accumulated estrogens which do not increase the risk of breast cancer. • Nutrigenetic foods that increase enzyme activity are the brassicas. • It is important that the individual does not smoke or is exposed to fumes and chemicals during up-regulation of the CYP1A1 enzyme.
COMT rs4680	AG	REDUCED enzyme activity.	• Assess the individual's weight and discuss weight reduction if necessary. • Reduce alcohol consumption if high. • Review and assess the MTHFR enzyme activity. • Reduce stress as this may be a factor associated with reduced enzyme activity. • Discuss the measurement of urinary estrogen metabolites that comprehensively measure 2, 4 and 16 hydroxylated estrogens.
Phase II detoxification			
GSTP1 rs1695	GG	Reduced GSTP1 enzyme activity.	• Review the individual's exposure to water soluble environmental toxins, including many solvents, herbicides, fungicides, lipid peroxidases and heavy metals such as mercury.

PATIENT NAME: Dr Garry Gordon
PATIENT DOB: 3-01-1935
PATIENT SEX: Male

Physiogenomic analysis

Gene and SNP ID	Genotype / Haplotype	Result and interpretation	Action steps and comments
Obesity/Depression			
BDNF rs6265	AG	INCREASED risk of obesity and depression.	• Discuss the benefit of exercise in relation to the natural release of endorphins. • Moderate exercise instead of reaching for food may be beneficial for mood and weight management.
Exercise and BP			
EDN1 rs5370	GT	Normal blood pressure.	• Review exercise activities because it is important for maintaining good cardiovascular health.
Brain health			
KIBRA rs17070145	CT	INCREASED memory and cognitive flexibility.	• Review daily exercise; establish a regular sleep pattern, play brain games and meditation as these activities have been reported to assist in maintaining brain health.
BRAIN HEALTH			
BDNF rs6265	AG	HIGHEST ACTH and cortisol in response to stress.	Chronic stress can severely impair memory; try relaxation techniques such as meditation. Exercise may improve mood and general feelings. • Create systems so you don't have to remember mundane day to day activities • Keep a diary of appointments. • Be active in communication by acknowledging what the other person is saying. • Make associations, create links between new information and things you already know. • Repeat new information. • Use imagery to create memory. • Keep your brain fit by eating a nutritious diet rich in berries, nuts and omega 3's. • Reduce chronic stress.
HPA axis			
TH	CC	NORMAL	• Recommend that the individual stays

PATIENT NAME: Dr Garry Gordon
PATIENT DOB: 3-01-1935
PATIENT SEX: Male

APOE genetic test result

Gene and SNP ID	Haplotype	Indicator	Result and Interpretation
APOE rs429358	CT		HIGH CARDIOVASCULAR DISEASE RISK*
APOE rs7412	CC		The APOE E3/E4 genotype has a gene frequency of 25% of most populations and contributes to a highly increased risk of dyslipidemia and related arteriosclerosis. Please review the action steps and comments in relation to this result.

What does this APOE genetic test result mean?

HIGH CARDIOVASCULAR DISEASE RISK

This result indicates that this APOE E3/E4 genotype is associated with a high risk of cardiovascular disease. Individuals with an E3/E4 genotype may have increased triglycerides, increased LDL, oxidative stress, chronic inflammation and oxidised LDL and individuals HDL-C level maybe decreased.

ACTION STEPS and comments:

- Review Table 1 in relation to soluble fibre, fish oil, energy sources, effects of alcohol and exercise for individuals with this genotype.
- If the individual smokes they should stop.
- Alcohol may raise LDL-C, decrease HDL-C/HDL2 and increase sdLDL formation. This effect is stronger for males. This should be assessed via cholesterol profile monitoring.
- Olive oil has been reported to increase sdLDL formation in this APOE 3/4 genotype. This should be assessed and monitored via cholesterol profile.
- Low saturated fat intake and reduced intake of processed carbohydrates and foods containing high amounts of antioxidants should be considered.
- Supplementation with omega-3-fatty acids is recommended, however fish oil has been reported to suppress HDL-C and raise calculated LDL-C.
- If statins are prescribed then supplement with Co-enzyme Q10.
- Niacin has been reported to lower triglyceride levels.
- Plant sterols and soluble fibre have been reported to have beneficial effects.

*There are three common variants of the APOE gene: E2, E3, and E4. Since human cells have two copies of each gene, there are six APOE genotypes: LMT A,1 or E2/E2, LMT A,2 or E2/E3, LMT B,1 or E3/E3, LMT B,2 or E2/E4, LMT C,1 or E3/E4, and LMT C,2 or E4/E4. The frequencies of these gene variations differ across ethnicities.

PATIENT NAME: Dr Garry Gordon
PATIENT DOB: 3-01-1935
PATIENT SEX: Male

ACTION STEPS and comments:

- From this individual's cholesterol profile determine if their HDL-C level is protective, if it IS NOT then
- Review the APOA1 genotype action steps.
- Review dietary fat intake if the individuals HDL-C IS NOT protective since lower dietary saturated fat intake will elevate HDL-C level.
- Review this individual's Physiogenomic results for the LPL, LIPC and CETP gene polymorphisms since they are associated with an increase in HDL-C and APOA1 in response to exercise of >8 METS/week.

ABCA1 genetic test result

Gene and SNP ID	Genotype	Indicator	Result and Interpretation
ABCA1 rs2230806	GG		**LOWER HDL-C level** in the blood based on this ABCA1 genotype. This result does not mean that the individuals HDL-C is low or non-protective. Please review the action steps and comments in relation to this result.

What does this ABCA1 genetic test result mean?

This ABCA1 genotype has been reported to be associated with lower HDL-C blood levels. This means that this individual has an increased risk of having a lower HDL-C level. The ABCA1 gene is a major regulator of cellular cholesterol and phospholipid homeostasis. With cholesterol as its substrate, this protein functions as a cholesterol efflux pump in the cellular lipid removal pathway.

ACTION STEPS and comments:

- From this individual's cholesterol profile determine if their HDL-C level is protective, if it IS NOT then
- Review the APOA1 genotype action steps.
- Review dietary fat intake if HDL-C IS NOT protective since lower dietary saturated fat intake will elevate HDL-C level.
- Review this individual's Physiogenomic results for the LPL, LIPC and CETP gene polymorphisms since they are associated with an increase in HDL-C and APOA1 in response to exercise of >8 METS/week.

CETP genetic test result

Gene and SNP ID	Haplotype	Indicator	Result and Interpretation
CETP rs5882	AG		**HIGHER HDL-C level** in the blood based on this CETP haplotype. Please review the action steps and comments in relation to this result.
CETP rs708272	AG		

Type 2 Diabetes

The long-chain acyl CoA synthetase 1 (ACSL1) and acetyl-CoA carboxylase (ACC2) play a key role in fatty acid synthesis and oxidation. Disturbance of these pathways is associated with impaired insulin responsiveness and metabolic syndrome (MetS). Moreover the ACSL1 and ACC2 gene polymorphisms are modulated by dietary fat intake. Genetic variations detected in the Transcription factor 7-like 2 (TCF7L2) and the Wolfram Syndrome 1 (WFS1) have been reported to play a role in insulin function. The Fat mass and obesity associated (FTO) gene, glucose-6- phosphatase. catalytic. 2 gene (G6PC2) and the peroxisome proliferator-activated receptor-gamma (PPARG) gene are associated with an increased likelihood of developing type 2 diabetes due to a higher BMI (FTO), reduced control of blood glucose levels (PPARG and G6PC2) or reduced pancreatic beta cell function Solute carrier family 30 (zinc transporter), member 8 (SLC30A8). The practitioner may also refer to the weight management section if overweight is an issue since additional information is available which may be of assistance.

This result does not mean that the individual has diabetes. Assessment of the individual's metabolic health in association with these gene variants relating to dietary fat intake, dietary n-6 PUFA, insulin secretion and BMI will assist with reducing the risk of type 2 diabetes.

ACSL1 genetic test result

Gene and SNP ID	Genotype	Indicator	Result and Interpretation
ACSL1 rs9997745	GG		**Increased metabolic syndrome (MetS) risk,** elevated fasting glucose, insulin concentrations and increased insulin resistance based on this ACSL1 gene polymorphism. Please review the action steps and comments in relation to this result.

What does this ACSL1 genetic test result mean?

This individual has two copies of the risk allele. It was reported that GG homozygotes have an increased risk of metabolic syndrome, elevated fasting glucose, insulin concentrations and increased insulin resistance. ACSL1 plays an important role in fatty acid metabolism and triacylglycerol synthesis. Disturbance of these pathways may result in dyslipidemia and insulin resistance which are the hallmarks of MetS.

ACTION STEPS and comments:

- Assess dietary fat intake since MetS risk was abolished among individuals with this genotype consuming either a low fat diet (<35% energy) or a
- high PUFA diet (>5.5% energy)

ACC2 genetic test result

Gene and SNP ID	Genotype	Indicator	Result and Interpretation
ACC2 rs4766587	AG		**INCREASED risk of metabolic syndrome (MetS)** including increased BMI, abdominal obesity and impaired insulin sensitivity based on this ACC2 gene polymorphism. Metabolic syndrome risk is increased for individuals with this genotype consuming a high fat diet >35% energy; in particular a high intake of n-6 PUFA. Please review the action steps and comments in relation to this result.

What does this ACC2 genetic test result mean?

Metabolic syndrome risk is increased for individuals with this genotype consuming a high fat diet >35% energy; in particular a high intake of n-6 PUFA. The ACC2 gene plays a key role in fatty acid synthesis and oxidation pathways.

ACTION STEPS and comments:

- Assess dietary fat intake since MetS is positively impacted by a low fat diet <35% energy.
- Review dietary n-6 PUFA in the diet since it has the greatest impact on MetS.

G6PC2 genetic test result

Gene and SNP ID	Genotype	Indicator	Result and Interpretation
G6PC2 rs560887	CC		**LOWER fasting glucose level** based on the G6PC2 gene polymorphism analysed. Please review the action steps and comments in relation to this result.

What does this G6PC2 genetic test result mean?

The G6PC2 gene polymorphism has been reported to be associated with lower fasting glucose level. Reduced control of fasting blood glucose level is a predictor of CAD and all-cause mortality. SNP rs560887 maps to intron 3 of the G6PC2 gene which encodes glucose-6-phosphatase catalytic subunit-related protein (also known as IGRP), a protein selectively expressed in pancreatic islets. This G6PC2 SNP was reported to be associated with fasting plasma glucose and with pancreatic beta cell function in 3 populations; however, it was not associated with risk of type 2 diabetes or body mass index (BMI).

Co-enzyme Q10

In the body, CoQ10 must be converted to its usable form in the body. CoQ10 is the inactive form and Ubiquinol is the active form. Ubiquinol as the reduced active antioxidant form of CoQ10 is used in cellular energy processes, it is a strong lipid-soluble antioxidant, and it protects cells from oxidative stress which can cause damage to protein, lipids and DNA. The highest concentration of this essential nutrient is in the heart. Studies have shown that Ubiquinol has superior absorption replenishing the normal CoQ10 plasma concentration more effectively. The transformation from CoQ10 to ubiquinol requires the addition of 2 electrons and 2 hydrogen molecules. NAD(P)H dehydrogenase [quinone] is an enzyme that in humans is encoded by the NQO1 gene. This gene is a member of the NAD(P)H dehydrogenase (quinone) family and encodes a cytoplasmic 2-electron reductase. Recent evidence shows that the NQO1 enzymes maintain ubiquinone (CoQ10) in its quinol form, which can act as an antioxidant protecting membranes from oxidative stress. In vitro studies of the NQO1 rs1800566 polymorphism markedly affect enzyme function. Homozygous variant cells of the rs1800566 polymorphism have complete absence of the NQO1 protein and activity. The result predicted that 5-20% of individuals (depending upon ethnicity) would likely have diminished metabolic activation of bioreductive compounds such as CoQ10. This finding indicates that individuals with this variant may not be effective at reducing CoQ10 to its active form. This is important for individuals that have been prescribed a statin therapy since utilisation of CoQ10 may be reduced.

NQO1 genetic test result

Gene and SNP ID	Genotype	Indicator	Result and Interpretation
NQO1 rs1800566	CT		Reduced NQO1 enzymatic activity preventing the one electron reduction of quinones that results in the production of radical species. In-vitro analysis has shown that the enzyme activity is greatly reduced when the "T" allele is substituted in the NQO1 rs1800566 polymorphism. Please review the action steps and comments in relation to this result.

What does this NQO1 genetic test result mean?

This individual inherited the risk allele for reduced enzyme activity. This result indicates that CoQ10 reduction to its active form ubiquinol may be affected based on this gene polymorphism.

ACTION STEPS and comments:

- Synthetic antioxidants and extracts of cruciferous vegetables are potent inducers of NQO1.
- The bioavailability of CoQ10 may be compromised since the conversion of CoQ10 to ubiquinol may be compromised.
- Ubiquinol is the reduced form of CoQ10 and it may be more bioavailable.
- Individuals prescribed a statin drug may benefit from ubiquinol rather than CoQ10.

PATIENT NAME: Dr Garry Gordon
PATIENT DOB: 3-01-1935
PATIENT SEX: Male

Omega-3 and Omega-6 blood levels

A large study has reported that a polymorphism in the Fatty Acid Desaturase 1 (FADS1) gene which produces an enzyme involved in the processing of omega-3 and omega-6 fats had lower blood levels of arachidonic acid (AA), an omega 6 fat, as well as eicosapentanoic acid (EPA) an omega-3 fat.

FADS1 genetic test result

Gene and SNP ID	Genotype	Indicator	Result and Interpretation
FADS1 rs174547	CT		Decreased blood levels of Arachidonic Acid (AA) and Eicosapentanoic acid (EPA). AA is a long chain omega-6 acid and EPA is a long chain omega-3 acid. Please review the action steps and comments in relation to this result.

What does this FADS1 genetic test result mean?

This individual inherited the risk allele for reduced blood levels of AA and EPA based on this FADS1 genotype and as such they may have lower bloods levels of AA and EPA.

ACTION STEPS and comments:

- Review dietary omega-3 intake and omega-6 intake.
- Consider measuring Fatty Acid status including the ratio of omega-3 to omega-6.
- Review the dietary intake of omega-6 fatty acids from processed foods and improve the intake of omega-3 fatty acids since the current ratio is skewed more towards omega-6 fatty acids.

PATIENT NAME: Dr Garry Gordon
PATIENT DOB: 3-01-1935
PATIENT SEX: Male

Vitamin B12 metabolism

Vitamin B12 has functional roles including DNA regulation and synthesis and brain and nervous system health. A polymorphism in the FUT2 gene has been reported to be associated with lower blood levels of B12.

FUT2 genetic test result

Gene and SNP ID	Genotype	Indicator	Result and Interpretation
FUT2 rs602662	AG		LOWER levels of B12 in the blood when compared with individuals harboring the AA genotype. Please review the action steps and comments in relation to this result.

What does this FUT2 genetic test result mean?

This individual inherited the risk allele for reduced blood levels of vitamin B12 in the blood based on this FUT2 genotype.

ACTION STEPS and comments:

- This result does not mean that the individual's B12 levels are low.
- Review dietary intake of vitamin B12. Dietary sources of vitamin B12 for example are meat, fish, eggs and dairy products

PATIENT NAME: Dr Garry Gordon
PATIENT DOB: 3-01-1935
PATIENT SEX: Male

Vitamin D metabolism

Genetic variations detected in the DHCR7, CYP2R1 and GC genes will indicate if the individual being tested is genetically predisposed to normal, moderate or high level of vitamin D insufficiency. Vitamin D insufficiency has been linked to an increased risk of the following diseases: osteoporosis, fractures, autoimmune diseases such as MS, Crohn's disease, lupus and rheumatoid arthritis, diabetes, depression and mood problems, reduced immunity and some cancers.

DHCR7, CYP2R1 and GC genetic test result

Gene and SNP ID	Haplotype	Indicator	Result and Interpretation
GC rs2282679	AA		HIGH RISK of vitamin D insufficiency based on the genetic variants tested. Please review the action steps and comments in relation to this result.
DHCR7 rs12785878	GT		
CYP2R1 rs10741657	GG		

What does this DHCR7, CYP2R1 and GC genetic test result mean?

This individual has inherited a haplotype that is associated with lower levels of vitamin D (plasma 25-hydroxy-vitamin D) based on the genes analysed.

ACTION STEPS and comments:

- This result does not mean that the individual's vitamin D levels are out of balance.
- Based on this genotype this individual has an increased risk of vitamin D insufficiency when compared to individuals that do not have the same genetic polymorphism.
- Maintain a healthy diet with dietary sources of vitamin D such as cod liver oil, fish especially raw fish, eggs, mushrooms and fortified dairy products.
- Discuss the importance of sunshine exposure with the client and review their daily exposure to sunshine.

PATIENT NAME: Dr Garry Gordon
PATIENT DOB: 3-01-1935
PATIENT SEX: Male

Methylation

MTHFR genetic variations

The Methylenetetrahydrofolate Reductase (MTHFR) gene encodes MTHFR protein. A distinct combination of two MTHFR gene polymorphisms C677T and A1298C result in the produce an enzyme with 70% reduced activity. Other combinations produce enzymes with different levels of enzyme efficiency. In addition, individuals with particular combinations of these gene variants have higher requirements for vitamin B9 commonly referred to as folate, folic acid or folicin. Folate is required for numerous body functions including DNA synthesis and repair, cell division, and cell growth. A deficiency of folate can lead to anaemia in adults, and slower development in children. For pregnant women, folate is especially important for proper foetal development. Folate or vitamin B9 is a water soluble vitamin that is well regulated by the body; therefore an overdose is rare in natural food sources.

MTHFR genetic test result

Gene and SNP ID	Haplotype	Indicator	Result and Interpretation
MTHFR rs1801133	CT		35% REDUCED MTHFR enzyme activity. Please review the action steps and comments in relation to this result.
MTHFR rs1801131	AA		

What does this MTHFR genetic test result mean?

This individual has inherited the haplotype that is associated with 35% reduced enzyme activity. However, this haplotype is not associated with reduced folate metabolism or elevated plasma homocysteine. There is NO INCREASED RISK of reduced folate metabolism or elevated homocysteine level.

ACTION STEPS and comments:

- Review the individual's dietary folate intake and make dietary changes if required.
- Serology may be required to assess the individual's red cell folate level.

Folate cofactors

The folate cofactors will assist the practitioner in determining if the patient has one or more genetic variations associated with elevated homocysteine level. The MTR, MTRR, TCN2 and SLC19A1 dependent on B group vitamins to function correctly in the folate mediated one-carbon metabolism. The risk associated with polymorphisms in these genes is high homocysteine level and neural tube defect during pregnancy.

PATIENT NAME: Dr Garry Gordon
PATIENT DOB: 3-01-1935
PATIENT SEX: Male

Oxidative stress

Superoxide dismutase is an enzyme that protects cells from increased oxidative stress and free radical damage to cell structures like membranes, mitochondria, DNA and proteins. SOD2 rs4880 is sensitive to inadequate antioxidant intake including environmental exposures that relate to ROS production such as smoking and environmental toxins. Among the antioxidant enzymes involved in protecting against ROS, the GPX1 enzyme plays an important role via the reduction of H2O2 to H2O. The human GPX1 gene contains the rs1050450 SNP which results in a Pro200Leu substitution. GPX1 is a selenoprotein, meaning it incorporates selenium into its protein structure. This polymorphism reduces an individual's ability to utilise selenium. That means that selenium intake needs to be assessed to afford protection to hydrogen peroxide-sensitive tissues, particularly lung and breast tissues. Catalase is a common enzyme found in nearly all living organisms that are exposed to oxygen, where it functions to catalyze the decomposition of hydrogen peroxide to water and oxygen. Catalase has one of the highest turnover numbers of all enzymes; one molecule of catalase can convert millions of molecules of hydrogen peroxide to water and oxygen per second. The rs1001179 CAT polymorphism identified in the promoter region of the human catalase gene has shown that individuals with the variant GA or AA genotypes have significantly lower activity than those with GG genotypes.

MnSOD genetic test result

Gene and SNP ID	Genotype	Indicator	Result and Interpretation
MnSOD rs4880	CT		Reduced enzymatic activity in relation to risk of cardiomyopathy associated with iron overload. Please review the action steps and comments in relation to this result.

What does this MnSOD genetic test result mean?

This individual has inherited the risk allele associated with reduced enzyme activity specifically in relation to cardiomyopathy associated with iron overload based on this MnSOD genotype. Among the antioxidant enzymes involved in protecting against reactive oxygen species, the MnSOD gene plays an important role via the reduction of hydrogen peroxide to water and oxygen. There is little overall association between MnSOD and cancer risk, therefore this polymorphism should not be used as general marker for cancer.

ACTION STEPS and comments:

- Consider the results in relation to the individual's vitamin and mineral intake and/or dietary intake of antioxidant rich foods.

Phase I detoxification

Cytochrome P450 1A1 catalyses the 2-hydroxylation of estrone (E1) and estradiol (E2) in to the catecholamines 2- hydroxy estrone (2-OHE1) and 2-hydroxy-estradiol (2-OHE2). These hydroxy metabolites show reduced estrogenic effects behaving more like anti-estrogens when compared with 4-OH and 16-OH metabolites. CYP1A1 also activates pro-carcinogens such as polycyclic aromatic hydrocarbons (PAH) or heterocyclic aromatic amines (HA) present in tobacco smoke and grilled or broiled meat which have been reported to play a role in some cancers, lung and breast. The CYP 450 1A1 rs4646903 SNP increases enzyme activity. CYP1B1 is also part of the CYP 450 family of cytochromes. The CYP1B1 enzyme hydroxylates estrogens into mutagenic 4 hydroxyestrone which creates toxic intermediaries from hydrocarbons that can mimic estrogens and promote estrogen receptor activity. The CYP1B1 rs1056836 SNP is unregulated by xenoestrogens favouring the formation of 4 hydroxyestrone. This increases the risk of prostate cancers in men and breast cancer in females to increased 4 hydroxyestrone which is mutagenic. Both the MTHFR enzyme and COMT enzymes are methylating enzymes, if both enzymes are sub-functional then reduced methylation of hydroxylated estrogens may occur. Reduced methylation of hydroxylated estrogens may result in the accumulation of fat soluble 4 hydroxy estrone which can be further oxidised to catechol quinones which can be DNA damaging and promote oncogenes (cancer genes). The CYP1B1 rs1056836 SNP increases the risk of individuals exposed to hydrocarbon or xenoestrogens. Therefore it is important for individuals to reduce their exposure to xenoestrogens, chemicals and pollutants. Females with the CYP1B1 rs1056836 SNP CG or GG genotypes who smoke were found to have a 2.3 fold increased risk of breast cancer when compared to non-smokers. A threefold increase was reported for long term HRT users.

CYP1B1 genetic test result

Gene and SNP ID	Genotype	Indicator	Result and Interpretation
CYP1B1 rs1056836	GG		Increased risk for pro-carcinogen activation. This enzyme hydroxylates estrogens into mutagenic 4 hydroxyestrone creating toxic intermediates from hydrocarbons that can mimic estrogens and promote estrogen receptor activity. Please review the action steps and comments in relation to this result.

What does this CYP1B1 genetic test result mean?

This individual has inherited the risk allele associated with pro-carcinogen activation based on the CYP1B1 genotype.

Phase II detoxification

The Glutathione-S-transferase enzymes detoxify many water soluble environmental toxins, including many solvents, polycyclic aromatic hydrocarbons, steroids, herbicides, fungicides, lipid peroxidases and heavy metals such as mercury, cadmium and lead. Decreased glutathione conjugation capacity may increase toxic burden and increase oxidative stress. Copy Number Variations in the GSTT1 and GSTM1 enzymes are associated with less effective detoxification of potential carcinogens may confer an increased susceptibility to some cancers. If either or both the GSTT1 or GSTM1 enzymes are ABSENT they are assigned a Null genotype. If either copy is present, it is termed PRESENT. The GSTP1 gene encodes for an enzyme, glutathione S-transferase P1 (GSTP1) located in brain tissue, skin tissue and lung tissue which is involved in Phase II detoxification of carcinogens, xenobiotics, steroids, heavy metals and products of oxidative stress. The GSTP1 rs1695 polymorphism produces a variant enzyme with lower activity and less capability or effective detoxification.

GSTT1 and GSTM1 haplotype genetic test result

Gene and SNP ID	Haplotype	Indicator	Result and Interpretation
GSTT1	PRESENT		AVERAGE blood levels of vitamin C. Please review the action steps and comments in relation to this result.
GSTM1	PRESENT		

What does this GSTT1 and GSTM1 haplotype genetic test result mean?

This individual has not inherited the risk alleles associated with reduced blood levels of vitamin C based on this combined GSTT1 and GSTM1 haplotype. The GST enzymes modify the association between dietary vitamin C and serum ascorbic acid level. However, it is important to ensure that all individuals maintain the RDI for vitamin C.

ACTION STEPS and comments:

- Individuals should maintain a healthy diet and stay balanced.
- Review dietary intake of vitamin C. Sources of vitamin C are lemons, oranges, watermelons and strawberries.

Life-transforming Health Events

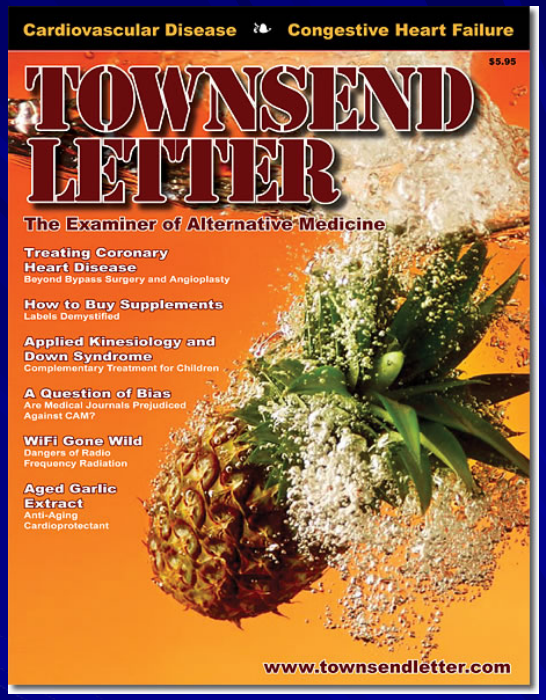
By age 29, I was so ill that I had to stop practicing medicine. It was at this time that I began to seek alternative medical therapies, embarking on a truly exciting journey of discovery that has dramatically improved my life and health.

- Vitamin C (Hi-potency)
- Chelated Minerals
- IV EDTA Chelation Therapy
- Oxidative / Ozone / UV / HBO
- Maca Root / Pueraria mirifica
- Zeolite (Clinoptilolite)
- Diet / non-GMO / Probiotics
- PEMF (pulsed electro-magnetic frequency)

Townsend Letter – July 2007

Chelation, Heavy Metals, Heart Disease, and Health: An Oral Detoxification Program That Is Now Essential for Optimal Health and Longevity

by Garry F. Gordon MD, DO, MD (H)



The Oral Detoxification Program: A Wide-Spectrum ProtocolCertain natural chelators can also be powerful antioxidants, but there is no single chelator that can meet all the needs of various tissues to bind different metals with different valences under different conditions of oxygen availability and differing pH levels. That is why I like my broadly based new program, the Oral Detoxification Protocol (ODP), which I am using on my patients and my horses. [See Part One of this article in the June issue of Townsend Letter for a complete introduction to my ODP.] With ODP, I am not relying on just one substance to lower the activity of metal-induced free radical mediated reactions.

I do not focus excessively on just one source of toxicity, whether it be vaccines or fillings or fish. Our genetics, environment, and diet are the interplay that largely determines the outcome from our ongoing continuous heavy metal exposures, which are all cumulative.

http://www.townsendletter.com/July2007/chelation0707.htm

Original Contribution | March 27, 2013

Effect of Disodium EDTA Chelation Regimen o n Cardiovascular Events in Patients With Previous Myocardial Infarction - The TACT Randomized Trial

JAMA The Journal of the American Medical Association

Gervasio A. Lamas, MD; Christine Goertz, DC, PhD; Robin Boineau, MD, MA, et al. for the TACT Investigators

JAMA. 2013;309(12):1241-1250. doi:10.1001/jama.2013.2107.

Importance Chelation therapy with disodium EDTA has been used for more than 50 years to treat atherosclerosis without proof of efficacy. Objective of study is to determine if an EDTA-based chelation regimen reduces cardiovascular events.

Conclusions and Relevance The 5-year estimate of reaching the primary end point shows that those given the EDTA chelation had a 18% lower risk (Hazard Ratio 0.82) which met the stringent level of statistical significance (p=0.035). The expectant lukewarm conclusion by authors was that the therapy "*modestly reduced the risk of adverse cardiovascular outcomes, many of which were revascularization procedures. These results provide evidence to guide further research but are not sufficient to support the routine use of chelation therapy for treatment of patients who have had an MI.*"

It should be mentioned that in the sub-group analysis, they saw much greater benefits in patients with diabetes (risk reduction of 39%, p=0.002) or with previous anterior MI (risk reduction of 37%, p=0.003) when given EDTA.

UNLEADED – The Movie

A documentary exposing the attempt to hide the revolutionary results of the 10 year $31 million NIH study to assess the impact of Chelation Therapy on heart disease and diabetes.

In 1999, cardiologist Roy Heilbron, MD and Angelique Hart, MD began participating in the $31 million double-blind NIH PACT & TACT studies on the effects of Chelation Therapy on heart disease, diabetes, and heart attacks.

On Nov. 4, 2012, Dr. Gervasio Lamas, the director of the TACT study presented the stunning results of 10 year study that involved over 130 clinics and 1,700 patients at the annual American Heart Association's 2012 Scientific Sessions.

Later that day, Elliott Antman, MD, Director of the AHA, announced that there was not enough information to recommend Chelation Therapy until more studies were done in the future, although TACT was designed to be the definitive study.

"Unleaded" takes a look at how heavy metals cause disease, and what Chelation Therapy can do towards possibly curing heart disease, diabetes, and many other diseases by removing heavy metals and toxins from our bodies.

For more information visit UnleadedMovie.com

Cardiovascular Disease • Chelation • Detoxification

MAY 2010 | ISSUE #322 | $7.50

Townsend Letter

Chelation Therapy
Nonsurgical Treatment of Heart Disease

The Salt Secret
How Salt Can Lower High Blood Pressure

The Awesome Foursome
Four Nutrients to Reverse Congestive Heart Failure

Ground Yourself
A Surprising Remedy for Many Ills

Milk and Obesity
Is There a Connection?

Beyond Chelation Therapy
Device Helps Reverse Disease

The Examiner of Alternative Medicine

WWW.TOWNSENDLETTER.COM

Chelation and Cardiovascular Disease

by Garry F. Gordon, MD, DO, MD(H)

I have spent over 35 years researching chelation therapy (CT), trying to determine how and why it helps patients with cardiovascular disease (CVD). I strongly believe that some form of CT should be a part of the treatment for anyone with CVD. My knowledge of CT has permitted me to safely advise against all bypass operations on my patients for over 30 years.

Due to my own CVD, I have intensively studied all aspects of it for most of the 50-plus years of medical practice. I have a complex medical history with lifelong heart disease issues that by age 29 had become nearly disabling: I avoided most physical activities until I was well in my 30s. When I first chelated, it was with with great results: hours after my eighth intravenous EDTA treatment, I felt like Superman! I could for the first time in my life run uphill without a racing heart, or chest pain, or fatigue.

I knew that this was working, but I jumped to the wrong conclusion: I thought that somehow CT must be reversing plaque, never dreaming that removing heavy metals could bring these benefits. My error probably set back the widespread acceptance of CT by decades, as knowledgeable invasive cardiologists often found that serious "obstructing" plaque was still present after CT.

I have since identified over 30 mechanisms of action of EDTA.[1,2] Any one or all of these working synergistically can explain why over 80% of patients get both subjective and objective improvement. However, it is still not possible to predict when sometimes more-dramatic benefits will occur, including occasional rapid saving of gangrenous legs, reversal of heart disease or blindness, or the occasional autistic child who within hours recovers speech. Since we have poisoned our planet, I believe that heavy metal detoxification is a big part of the explanation for the benefits seen, even in nonexposed patients.[3–11] All causes of morbidity and mortality have been shown to relate to how low lead levels are kept throughout life.[5,8,12]

There is no magic program that can remove all of our heavy metals or other toxins overnight. We need several years to decrease the body burden of lead, as bones will remodel over a period of 15 years. I recommend continuous use of one or more aids to detoxification such as chelators, high-dose vitamin C, fiber, lipoic acid, zeolite, saunas, and daily exercise.[13–27] These all provide benefits that greatly exceed any risks involved. For example, the various claims about chelation toxicity, such as harming the kidneys, although possible, are greatly exaggerated. In fact, repeated EDTA infusions often postpone indefinitely the need to start dialysis for many patients in early renal failure.[14]

I have acquired and reviewed thousands of articles and books about chelation and heavy metals; I have treated hundreds of patients, and seen many dramatic responses, yet I warn my patients that CT does not predictably by itself decrease plaque.[15–17] However, improved blood flow happens in over 80% of patients. With more treatment and improved compliance with my "FIGHT" Program (Food, Infection, Genetics, Heavy metals/Hormones, Toxins), over 95% will improve, even if the angiograms report that plaque size has increased. This experience and my radiology training confirm the limitation of angiograms, which fail to identify the existence of collateral circulation, as seen with a PET scan.[20]

Obstructing plaque or vascular calcium scores may appear worse after CT, yet the patient has dramatic subjective and objective improvement, and is now winning in competitive sports. I prefer noninvasive tests that more accurately assess the true status. They are useful and can motivate patients to try harder, as a poor response may just be a patient's failing to address all risk factors

In my own case, I had a mouthful of amalgam fillings, and part of my dramatic early response was due to removing heavy metals, which we now know interfere with healthy enzyme function and thus impair nitric oxide levels.[22] Improved nitric oxide function is another reason for the predictable improvement in blood flow seen with all noninvasive measurements, including segmental blood pressures, thermography, plethesmography, Bio Clip, and multifunction ECG.[24–27]

Researchers at California Institute of Technology have shown that average bone lead levels today are 1000 times higher than a few hundred

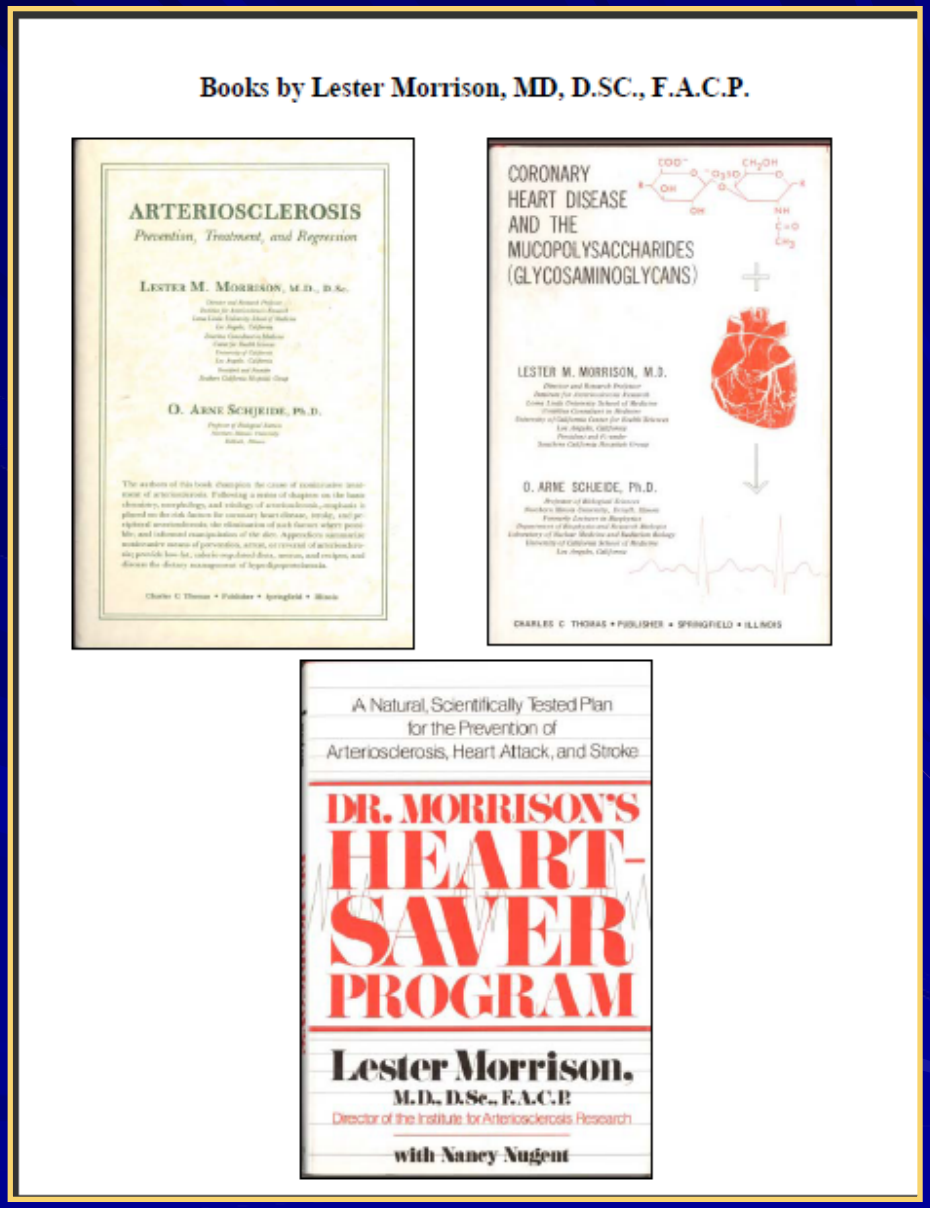
➤

Dr. Lester Morrison spent $10 million doing the research that led to his nutritional program that modifies viscosity and clotting.

He found a combination of several nutrients that act synergistically with his special mucopolysaccarides to help reverse arteriosclerosis and stop heart attacks.

The addition of EDTA made it far more effective and led to its incorporation into oral packets of nine pills. These packets include a strong multivitamin, a capsule each of omega-3 and primrose oil, a phosphatidyl serine with *Gingko biloba*, and three capsules containing the EDTA-enhanced institute formula.

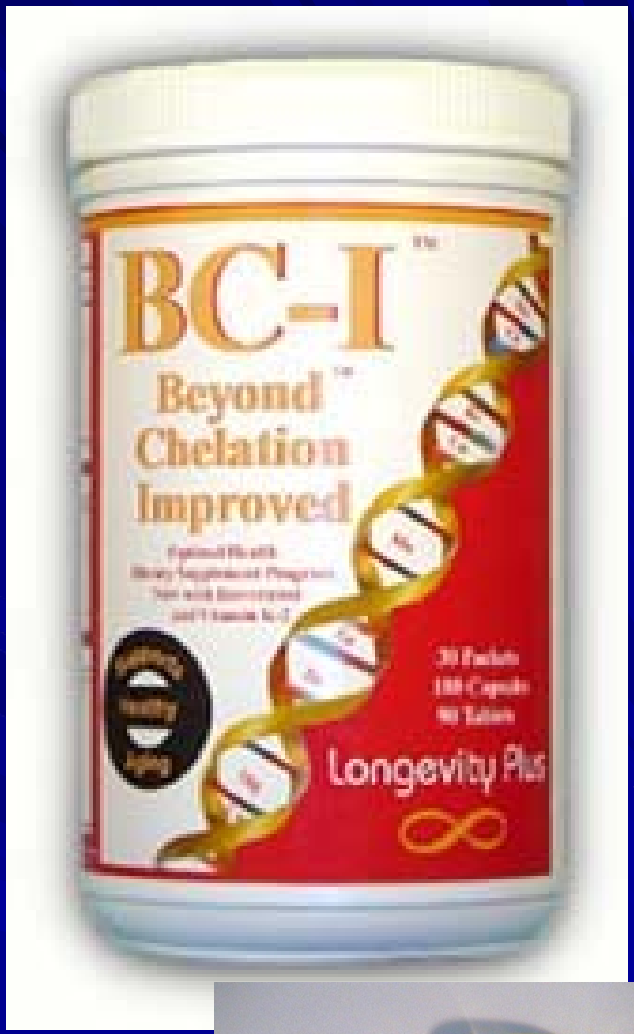
Dr. Morrison's two published studies reported an average 91% reduction in fatal heart attacks using his institute formula. This combination has been shown to lower viscosity using rheological testing. This is one reason that the packets help prevent fatal blood clots. Due to its weak benefits and side effects, I prefer these nine pills to aspirin, which I usually discontinue. ~ Garry Gordon, MD,DO,MD(H)



Beyond Chelation Improved (BC-I)

Each canister of Beyond Chelation Improved™ contains 30 packets. Each packet consists of:

- **3 *Beyond Any Multiple*™ caplets with Vitamin K2, Resveratrol, Tocotrienols, and Utah Sea Minerals**
- **3 *Essential Daily Defense*™ capsules (which deliver a combined total of 400 mgs of EDTA)**
- **1 Omega 3 marine lipid concentrate**
- **1 Evening Primrose Oil capsule**
- **1 Phosphatidyl Ginkgo Biloba capsule.**



Ninety Percent Reduction in Cancer Mortality after Chelation Therapy With Ca-EDTA

Walter Blumer, M.D. and Elmer Cranton, M.D.

ABSTRACT:

Mortality from cancer was reduced 90% during an 18-year follow-up of 59 patients treated with Calcium-EDTA. Only one of 59 treated patients (1.7%) died of cancer while 30 of 172 non-treated control subjects (17.6%) died of cancer (P=0.002).

Death from artherosclerosis was also reduced. Treated patients had no evidence of cancer at the time of entry into this study. Observations relate only to long-term prevention of death from malignant disease, if chelation therapy is begun before clinical evidence of cancer occurs. Control and treated patients lived in the same neighborhood, adjacent to a heavily traveled highway in a small Swiss city. Both groups were exposed to the same amount of lead from automobile exhaust, industrial pollution and other carcinogens.

Exposure to carcinogens was no greater for the studied population than exists in most other metropolitan areas throughout the world. Statistical analysis showed EDTA chelation therapy to be the only significant difference between controls and treated patients to explain the marked reduction in cancer mortality.

Arch Virol. 1982;73(2):171-83.

Disintegration of retroviruses by chelating agents

Wunderlich V, Sydow G.

Abstract

Exposure in vitro of various mammalian retroviruses to the chelating agents EDTA or EGTA in millimolar concentrations resulted in partial disintegration of viral membranes as measured by accessibility or even release of reverse transcriptase, an internal viral protein, without any other treatment usually required.

Among the viruses responding to chelators were mammalian type C viruses, primate type D viruses and bovine leukemia virus. The effect was dose-dependent. The avian type C virus AMV, however, was found to be not susceptible to the agents. Rauscher mouse leukemia virus treated in vitro with EDTA or EGTA showed reduced infectivity in mice.

The results are considered as evidence for some association of divalent cations with membranes of mammalian retroviruses. The disintegrating activity of EGTA suggests that Ca2+ is an integral constituent of viruses but Mg2+ may also be involved. These cations seem to be responsible for maintaining integrity of retroviral membranes which, after chelation of ions, are either disrupted or become permeable for the exogenous template of reverse transcriptase.

In addition, the disintegrating activity of trifluoperazine may indicate that a calmodulin-like protein occurs in retroviral membranes.

PMID: 6816193 [PubMed - indexed for MEDLINE]

Chelators as Life-Extending Substances

A number of studies confirm that chelating agents — particularly, EDTA — may have life-extending properties.

Johan Bjorksten and other scientists demonstrated the life-extending effects of EDTA on lowly rotifers (small multi-celled animals found in freshwater lakes and ponds).

In the Soviet Union in the 1970s, Dr. T.L. Dubina performed a series of studies with EDTA on the life span of rats. In most of the studies, the mean life span of female rats treated with EDTA was increased by nearly 50%, and in one study the maximum lifespan increased 18-25% over the control animals.

Other natural chelators include garlic, (10) Chlorella, (11) lactic acid, citric acid, and malic acid. Bjorksten demonstrated that lithium was also an effective aluminum chelator and crosslinkage inhibitor, stating that lithium continues to be the most effective electrolyte for aluminum detachment.

Bjorksten also believed that one of the benefits of exercise is that toxic heavy metals (especially aluminum) are chelated by the lactic acid that is generated.

Based on these and other studies, Bjorksten's associate, Prof. Donald Carpenter, calculated that the widespread use of chelation therapy would result in an average lifespan increase of over fifteen years.

TOXINS
Oxidative Stress
& Disease

Many diseases have been linked to oxidative stress….

The Environmental Working Group studies that have shown:

134 chemicals are shown to cause CANCER

151 chemicals cause BIRTH DEFECTS

154 are HORMONE DISRUPTORS

186 chemicals contribute to INFERTILITY

130 chemicals cause IMMUNE SYSTEM TOXICITY

158 chemicals are NEUROTOXINS

Autism now 1 in every 150 children.

57% increase in childhood brain cancer.

84% increase in acute lymphocytic leukemia in children (1975 – 2002)

About 7.3 million American couples have trouble becoming pregnant, or carrying to term, a 20% increase in the last 10 years. Sperm count decrease one percent every year.

"The combined evidence suggests that neurodevelopmental disorders caused by industrial chemicals has created a silent pandemic in modern society." ***~ Lancet, November 8, 2006.***

WATCH THE VIDEO: http://video.yahoo.com/watch/6431545/16676271

Lead Exposure on the Rise Despite Decline in Poisoning Cases

SCIENTIFIC AMERICAN™

By Mark Fischetti – Feb 17, 2013

Leaded gasoline and lead paint are gone, but other sources are keeping the danger high.Lead is still present in drinking water in many communities, where it can leach from lead pipes in homes, apartment buildings and municipal water system, or from brass fittings or solder used in plumbing.

Another 25,000 to 30,000 tons of lead enters the U.S. environment each year from hunting and shooting-range ammunition, fishing-line weights, discarded batteries and electronic waste, said Mark Pokras at Tufts University.

a coal-fired power plant in Dadong, Shanxi province

Coal-burning power plants in developed nations also generate some lead in emissions and more so in ash, and the steep rise in coal power in China has boosted levels worldwide because regulations are more lax. Larger lead particles fall to the ground within about 200 meters of the source (including tailpipes), but the smaller particles, about 0.5 micron in size, can remain airborne for a week before they settle out. According to Flegal, lead particles from China have been found in rainfall in Santa Cruz, Calif.

http://www.scientificamerican.com/article.cfm?id=lead-exposure-on-the-rise&page=2

Fukushima Radiation Found in Bluefin Tuna in California

Five months after the Fukushima disaster in Japan, researchers tested bluefin tuna caught off the coast of San Diego and found higher-than-normal levels of radiation.

By Alicia Chang | AP | May 29, 2012

Across the vast Pacific, the mighty bluefin tuna carried radioactive contamination that leaked from Japan's crippled nuclear plant to the shores of the United States 6,000 miles away — the first time a huge migrating fish has been shown to carry radioactivity such a distance.

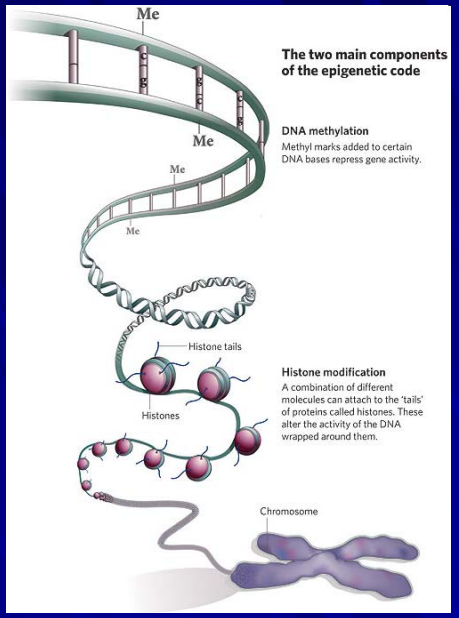
"We were frankly kind of startled," said Nicholas Fisher, one of the researchers reporting the findings online Monday in the *Proceedings of the National Academy of Sciences*.

The levels of radioactive cesium were 10 times higher than the amount measured in tuna off the California coast in previous years. But even so, that's still far below safe-to-eat limits set by the U.S. and Japanese governments.

http://healthland.time.com/2012/05/29/bluefin-tuna-carried-fukushima-radiation-across-the-pacific-to-calif/#ixzz1wOOUNKrd

Toxic and Heavy Metal Exposure Early In Life May Promote Disease Later in Life Via Epigenetics

Metals and Neurotocicology. J. of Nutr. 138,12,2007. Wright, RO, et al.



Minerals are necessary for normal cellular, metabolic and neurological function. It is well known that nutrient mineral deficiency can impair neurological development. Iron deficiency is a good example. However, it is also known that iron excess can also impair neurological development. Some transitional nutrients can cause later-life health disturbances when deficient in the diet, but in excess can be just as harmful. These include iron, copper, manganese and zinc as well as others. Heavy metals such as lead, cadmium, mercury, and arsenic are also neurotoxins and when present early in life can contribute to impaired neurodevelopment and detrimental health effects later in life and have been called the "fetal origins of disease." Suggesting that early environmental metal exposure can program later life gene expression, or fetal programming. The mechanism for this phenomenon is termed epigenetics. Epigenetics is the study of heritable changes in gene expression that occur without changes in DNA sequence, that unlike mutations, are reversible and responsive to environmental influences. DNA methylation is the most studied of the epigenetic process that regulated gene silencing.

Quinton Marine Plasma works similarly to Methylation Therapy with MSM, TMG and ACTIVE Folic Acid, B-6 and Sublingual B12 to undo the epigenetic changes that exposure to toxins like Bisphenol A are producing in our population. It is obvious that there is nothing in the world to offer this level of ULTRA TRACE MINERAL REPLETION.

~ Dr. Garry F. Gordon, MD, DO, MD(H)

Xenobiotics and their relationship to chronic illness

SPECIAL REPORT: INTEGRATIVE MEDICINE IN THE MARKETPLACE

Pioneering Research: David R. Jacobs, PhD, Explains the Relationship Between Xenobiotics and Type 2 Diabetes

Jeffrey Bland, PhD, FACN

Disclosure: Dr. Bland currently serves as chief science officer for Metagenics, a company that sells supplements and is president of its biotechnology unit, MetaProteomics, in Gig Harbor, Washington.

Editor's Note: The following is an adaptation of an August 2010 interview done by Dr Bland during his Functional Medicine Update, an audio journal now in its 28th year of publication. Integrative Medicine: A Clinician's Journal is pleased to publish this exclusive print presentation.

Jeff Bland: This interview is with a person whom most I've been following very carefully and closely. Every time 1 of his publications comes out, I can hardly wait to read it because I think the work is creating a whole new paradigm I'm speaking about David R. Jacobs, PhD.

Dr Jacobs is a Mayo Professor of Public Health, Division of Epidemiology and Community Health, at the University of Minnesota School of Public Health. He has what you might consider a fairly interesting background, although not one that you'd immediately connect to an investigator of type 2 diabetes. He is a mathematician from his undergraduate training and later earned a PhD in mathematical statistics at Johns Hopkins.

Working in epidemiology I mean he's a person looking at associations, and he has done an extraordinary amount of work in a whole variety of areas. Let me give you a thumbnail of some of the major ones.

I first became familiar with Dr Jacobs in regard to the work he has done on whole grains and refined grains intake and the relationship to chronic disease—one we epidemiologist associate work that I'm sure we'll touch upon in this interview. He's also looked at inflammatory processes and oxidation and how those relate to chronic disease. In addition, Dr Jacobs has done investigative work in periodontal disease and its relationship to the etiology of cardiovascular disease.

More to the point, the area that we're going to be speaking to quite a bit in this interview is related to Dr Jacobs' pioneering work in type 2 diabetes and his relationship to xenobiotics and persistent organic pollutants (POPs). Dr Jacobs will be weaving the story on how these remains into biological function.

Dr Jacobs, how did an undergraduate degree in mathematics ultimately translate into looking at the epidemiology and the association between POPs and type 2 diabetes?

David Jacobs: Statistics is, in a certain way, a branch of applied mathematics. An interesting little story is that when I was doing mathematical statistics, which is really heavy-duty math, the mathematical statisticians pretty much said, "Well, the biostatisticians, they're just applied, they don't really know the answers." But it turns out that the substance behind the numbers—that which creates the numbers—is, of course, extremely important, and I have striven throughout my career to really understand the substance. As really, I've much more interested in the substance than in the mathematical principles. That's the short answer.

JB: I think a hallmark of your work is the collaboration you've had with Duk-Hee Lee at the Medical University in South Korea looking at this nutrition connection, actually going back and looking at data from NHANES [the National Health and Nutrition Examination Survey] II and III. Maybe you could take us through that whole story because the way your whole hypothesis emerged sounds fascinating. I'd love to know where it came from.

DJ: Actually, the story with Duk-Hee Lee is really a fun story because I had written 3 papers with her before I met her. She is just an incredible, vigorous, and bright scientist. I came to know her through colleagues. You know, we English speakers control literature, for better or for worse. I am often called on for my English language skills to help people who have written in a foreign language or have a native language other than English. Ms Lee and I hit it right off because I not only fixed the English, but I made a few comments on the first paper that we worked on. Ultimately, she came to Minnesota and since then has been several times and has worked with me for months to years at a time. We've had a relationship in which we talk to each other by email probably at least once a week over the past 8 years or so. I think we have written something like 50 papers together.

The way that the science came about is that the way interest in a variety of things and it turned out that our skill sets

- POPs – Persistent organic pollutants
- Exotoxins from the environment
- Endotoxins from bacterial action in the gut

Bland J, *Integrative Medicine*, Oct/Nov 2010

Chronic exposure and mitochondrial dysfunction and insulin resistance

Chronic Exposure to the Herbicide, Atrazine, Causes Mitochondrial Dysfunction and Insulin Resistance

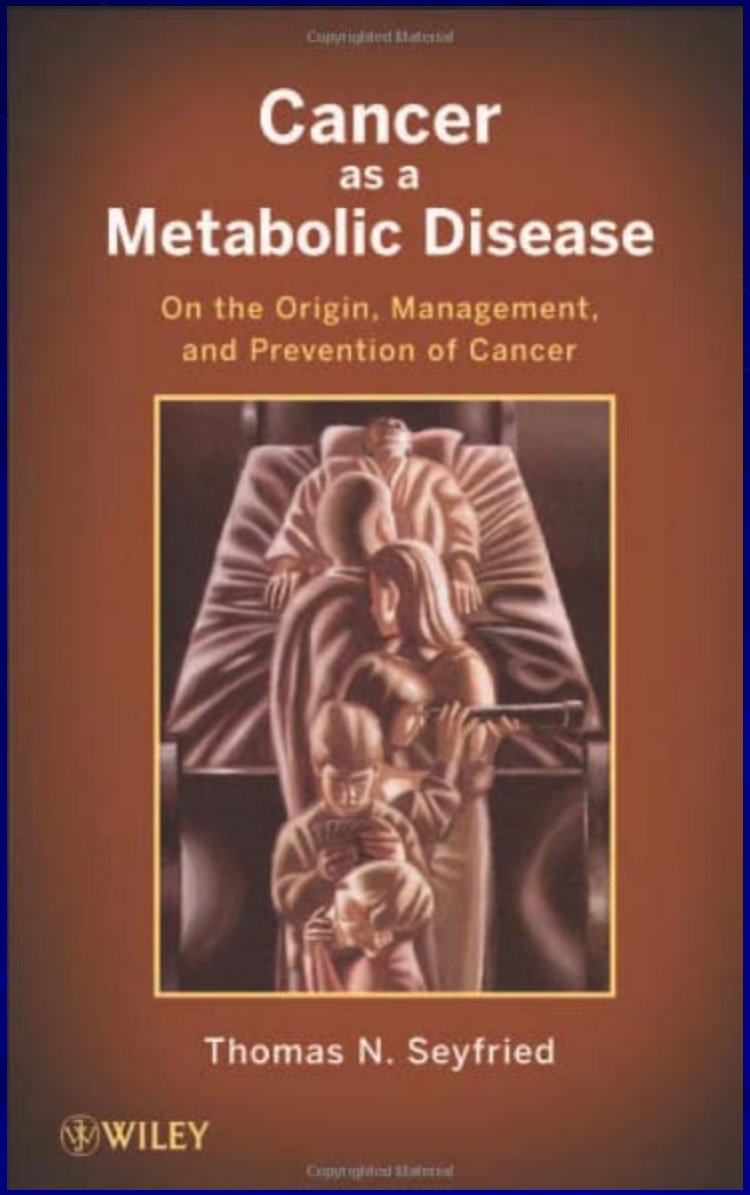
Soo Lim[1], Sun Young Ahn[2], In Chan Song[3], Myung Hee Chung[3], Hak Chul Jang[1], Kyong Soo Park[1], Ki-Up Lee[3], Youngmi Kim Pak[4]*, Hong Kyu Lee[5]*

Abstract

Introduction

- Uncoupling of mitochondrial bioenergetics with the production of free radicals and oxidative stress
- Energy deficit produces insulin dysregulation and resistance
- Glutathione mediated detoxification and antioxidation systems put under stress

PlosOne 2009; 4: 1186-90.



Cancer as a Metabolic Disease

[excerpts from pg(s) 5-6 and 17]

Radiation therapy is given to many cancer patients. Radiation will kill both cancer cells and normal cells.

Some normal cells that are not killed outright can be metabolically transformed into tumor cells.

Moreover, those tumor cells that survive the radiation treatment will sometimes grow back as more aggressive and less manageable cancers in the future.

Emerging evidence suggests that cancer is a metabolic rather than genetic disease.

Cancer is a disease of defective cellular energy metabolism, and most of the genomic defects found in cancer arise as secondary downstream effects of defective energy metabolism.

Curr Opin Clin Nutr Metab Care. 2006 Jul;9(4):339-45.

Oxidative metabolism in cancer growth.

Ristow M. Department of Human Nutrition, Institute of Nutrition, University of Jena, Jena, Germany.

Abstract

Recent evidence suggests that oxidative metabolism may have a key role in controlling cancer growth.

More than 80 years ago, Otto Warburg suggested that impaired oxidative metabolism may cause malignant growth. This assumption, later known as Warburg's hypothesis, has been experimentally addressed for many decades. It employs multiple approaches including cell lines, implanted xenografts and other animal models, by biochemical methods to quantify glycolytic and mitochondrial fluxes and signaling pathways including the rates of intermediate metabolism, respiration and oxidative phosphorylation.

The hallmarks of cancer growth, increased glycolysis and lactate production in tumors, have raised attention recently due to novel observations suggesting a wide spectrum of oxidative phosphorylation deficits and decreased availability of ATP associated with malignancies and tumor cell expansion. The most recent findings suggest that forcing cancer cells into mitochondrial metabolism efficiently suppresses cancer growth, and that impaired mitochondrial respiration may even have a role in metastatic processes.

Cancer a Redox Disease

By Dr. Mae-Wan Ho
December 4, 2012

Institute of Science in Society
science society sustainability

Cancer cells are universally disturbed in their electronic energy balance, an understanding that potentially revolutionizes cancer therapy and prevention.

An organism is energized by electrons (and protons) flowing through a liquid crystalline matrix that extends into the interior of every single cell.

The movement of electrons between chemical species is reduction (for the electron acceptor) and oxidation (for the electron donor). Reduction and oxidation always go together, hence 'redox' reactions.

Redox reactions are the heart of energy transduction in living organisms. Electrons move according to the *reduction potential* (also referred to as reduction-oxidation potential or redox potential), the affinity of a substance for electrons.

The redox potential for each substance is compared to that of hydrogen, which is set arbitrarily to zero at standard conditions of 25 °C, 1 atmosphere, and 1 M concentration.

Electrons – An Overlooked Key Nutrient

All physical things are comprised of atoms. An atom consists of a central nucleus which is positively charged, and electrons which are negatively charged in shells or orbits around that central nucleus.

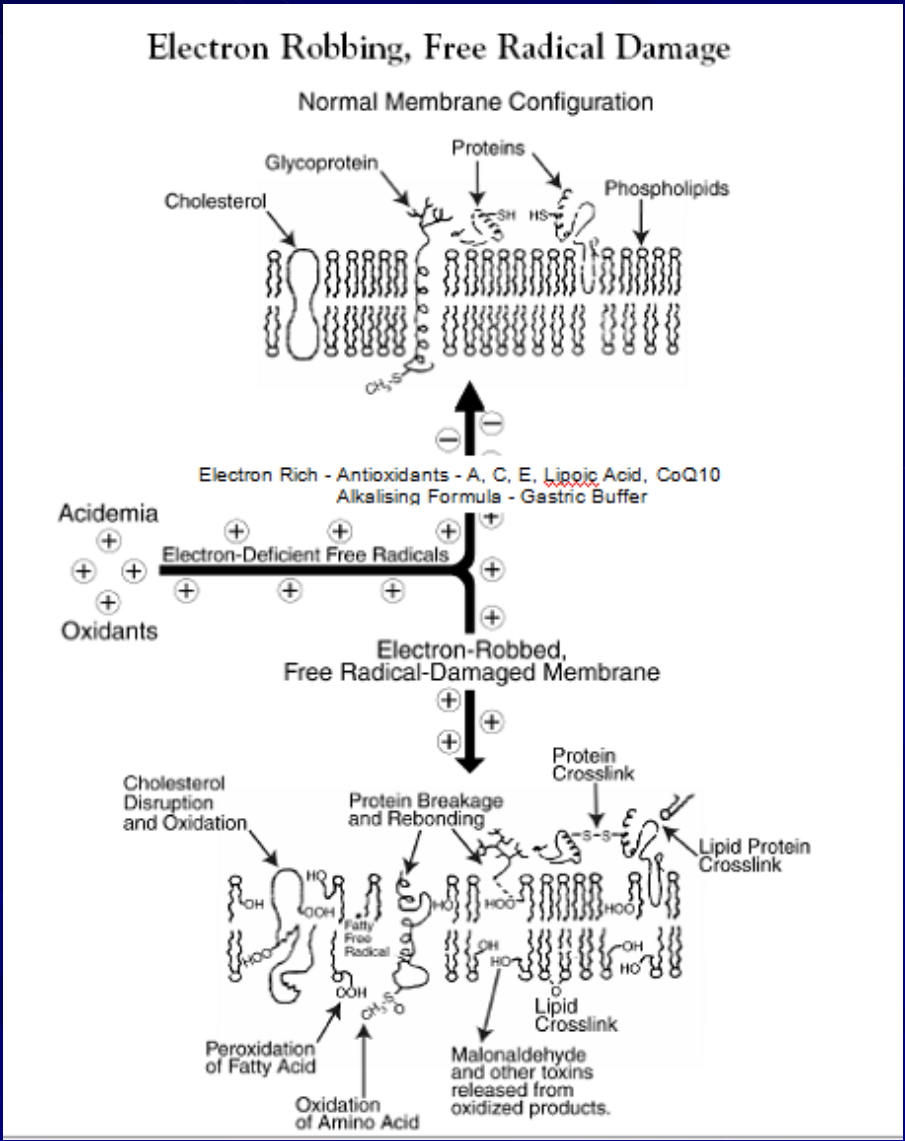
Atoms combine with one another because of their desire to lose, gain, or share electrons.

The phenomenon of electrons from one atom being shared with another atom is essential for construction of the complex biochemical compounds, organelles, cells, tissues, and organs comprising life.

The release of energy as electrons move from one energy level to another is responsible for the energy required in all body processes.

Modern living has created an electron-deficient environment that is creating electron-deficient bodies. Electron Deficiency is another way of saying something is Acidic.

Oxidation and Reduction



Electron excess and deficiency can also be understood in terms of oxidation and reduction.

An oxidant is a chemical that is deficient in electrons and tends to take them from others. If a compound has its electrons stolen by an oxidant, it is said to be oxidized.

A reducing agent is a chemical that donates electrons to another chemical. The chemical that receives the electrons is said to be reduced.

An oxidation-reduction chemical reaction is one in which some chemicals are receiving electrons and others are losing them.

Oxidation-reduction reactions occur continuously in the body.

Oxid Med Cell Longev. 2011;2011:194586.

Strategies for reducing or preventing the generation of oxidative stress

Poljsak B.
Laboratory for Oxidative Stress Research, Faculty of Health Sciences, University of Ljubljana, Zdravstvena Pot 5, 1000 Ljubljana, Slovenia. borut.poljsak@zf.uni-lj.si

Abstract

The reduction of oxidative stress could be achieved in three levels:
(1)by lowering exposure to environmental pollutants with oxidizing properties
(2)by increasing levels of endogenous and exogenous antioxidants, or
(3)by lowering the generation of oxidative stress by stabilizing mitochondrial energy production and efficiency.

Endogenous oxidative stress could be influenced in two ways: by prevention of ROS formation or by quenching of ROS with antioxidants. Recent evidence suggests that Antioxidant supplements often do not offer sufficient protection against oxidative stress, oxidative damage or increase the lifespan. The key to the future success of decreasing oxidative-stress-induced damage should thus be the suppression of oxidative damage without disrupting the well integrated antioxidant defense network.

Approach to neutralize free radicals with antioxidants should be changed into prevention of free radical formation. Thus, this paper addresses oxidative stress and strategies to reduce it with the focus on nutritional and psychosocial interventions of oxidative stress prevention, that is, methods to stabilize mitochondria structure and energy efficiency, or approaches which would increase endogenous antioxidative protection and repair systems.

http://www.ncbi.nlm.nih.gov/pubmed/22191011

Dr. Garry Gordon's F^2IGH^2T For Your Health Program

F^2 = Food and Focus - related aspect and leaky gut, and Focus (positive mental outlook): Acidophilus, Avoid food sensitivities (wheat, dairy) food supps to include Vitamin C and D

I = Infections - causing cancer, cardiovascular disease, autoimmune diseases: Ozone/UVB, HBO, Silver, Vit A, C and D including IV Vit C

G = Genetics - and epigenetics and methylation issues needed for detoxing B-12, MSM, TMG, 5'MTHF

H^2 = Heavy Metals and Hormones - Daily detoxification of mercury, lead; Hormonal balance and support for both men and women: Oral Chelation, Zeolite, DHEA, HRT, Melatonin, GH Support, Thyroid

T = Toxins - BPA, phtalates, and other toxins including household chemicals and everyday products: Exercise, IR/FIR Sauna, PEMF, Magnetics, Electrotherapy, cold (soft) lasers.

E^2 = Energy and Exercise - PEMF or pulsed electromagnetic frequency therapy that promotes healing through

Magnetically Induced Cellular Exercise, or MICE

FIGHT with M.I.C.E. = *Magnetically Induced Cellular EXERCISE*

Pulsed electromagnetic therapy recharges the body's 70+ trillion cells. Like physical exercise, it increases cellular bioporation, oxygenation, alkalinity, energy production, and nutrient uptake – while promoting vital autophagic processes and detox of harmful toxins and metals.

Multi-vitamin complex
Herbs & Minerals
Omega 3's
Zeolite

EDTA (calcium edta)
Vitamin C
Zeolite
Fiber

Dr. Gordon's Personal Daily Supplement Regimen

10 mins PEMF assisted Magnetically Induced Cellular Exercise twice per day

- Acetyl L-Carnitine (558 mg) 1 BID
- Adrenal Support, 1 BID
- Liquid Cellular Glutathione
- Liquid Colloidal Cellular Silver
- ACZ liquid zeolite
- Aloe caps for immune function
- B12 Sublingual, one at night
- Multivitamin and Chelation supplement
- Growth Hormonal Supplement With Resveratrol
- Lithium Orotate
- Herbal Brain Enhancer
- Boluoke Lumbrokinase
- D' Ribose
- DHEA 50 Milligram
- Benfotiamine
- 100% Chelated Magnesium Glycinate/Lysinate
- FibroBoost
- Phytosome Curcumins
- CoQ10, 100 mg one daily
- Mena Q7/Vitamin K2
- Quercetin Bromelain
- Trans-Resveratrol
- Pueraria mirifica (Herbal Remedy from Thailand)
- Hyal-Joint, 20 mg, one daily
- Immune System Support
- Kyodophilus probiotics
- L-5-HTP
- Master AntiOxidants
- Maximino
- Melatonin 10 mg, nightly
- N-Acetyl Cysteine (NAC)
- Omega 3 fish oil supplement
- Vitamin E
- Power Drink – Vitamin C, Maca, Organic Greens, stabilized rice bran and Fiber,
- Pregnenolone
- Stabilized R-Lipoic Acid
- Testosterone/Progesterone/Chry-H 150/5/200
- Thyroid 2 Grains, once daily
- Thyroid Support
- Vitamin D3, 5,000 Units
- Zeolite capsules

PEMF.US

Pulsed Electro-Magnetic Field

PMT-100

The Solution

With more than 40 years of clinical studies, researchers believe that the pulsed signal nudges the body's chemistry so the healing process may proceed more rapidly.

http://www.pemf.us

PEMF exercise slows aging

The resting potential of damaged and diseased cells is up to 80 percent lower than normal. This lowers metabolism and energy and makes the body more vulnerable to damage from disease-causing free radicals.

PEMF raises the body's supply of circulating electrons, thus serving as a potent antioxidant to *boost cellular energy.*

PEMF's antioxidant benefits, along with its proven ability to repair and regenerate tissue, make it a powerful anti-aging tool.

PEMF Therapy Increases Cellular Membrane Permeability and Cellular Metabolism

As early as 1940, it was suggested that magnetic fields affect the TMP and the flow of ions in and out of the cells and might therefore influence cellular membrane permeability.

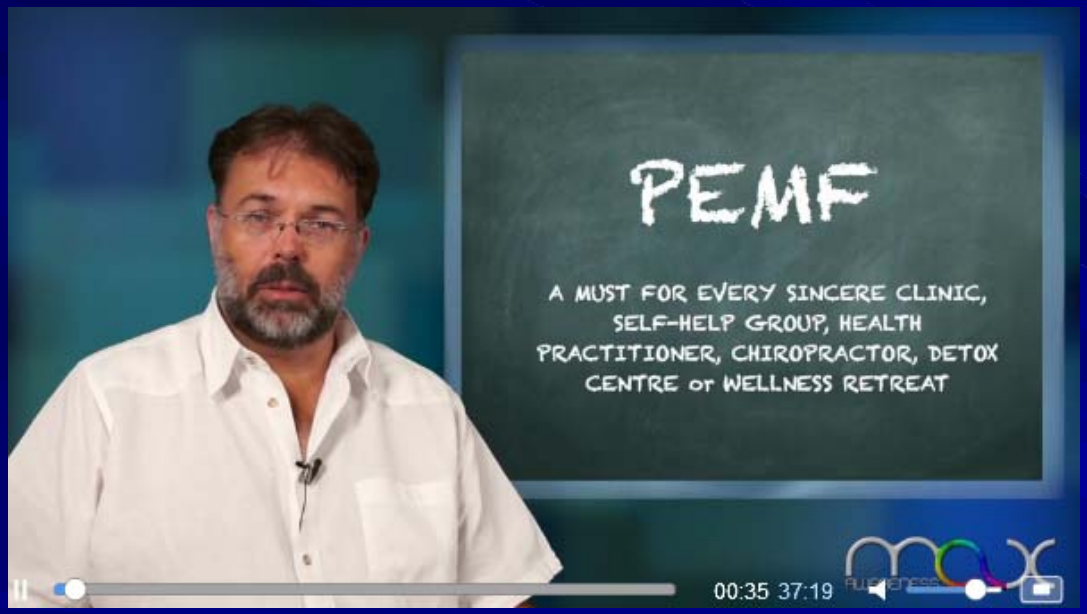
It has since been established that magnetic fields can influence ATP (Adenosine Tri-phosphate) production; increase the supply of oxygen and nutrients via the vascular and lymphatic systems; improve the removal of waste via the lymphatic system; and help re-balance the distribution of ions across the cell membrane.

Healthy cells in tissue have a voltage difference between the inner and outer membrane referred to as the membrane resting potential that ranges from -70 to -80 mV. This causes a steady flow of ions through its voltage-dependant ion channels.

As the magnetic field created fluctuates, it induces an electron flow or a current in one direction through the living tissue. As electrons always flow from a negative (cathode) to a positive (anode) potential, when the magnetic field vanishes, the direction of the electron flow is reversed. Therefore such induced polarized currents stimulate the exchange of ions across the cell membrane.

With over 8000 members this is the world's leading alternative cancer education program

TRULY HEAL! Health & Wellness Centers – 590+ revolutionary healing centers to open around the world!



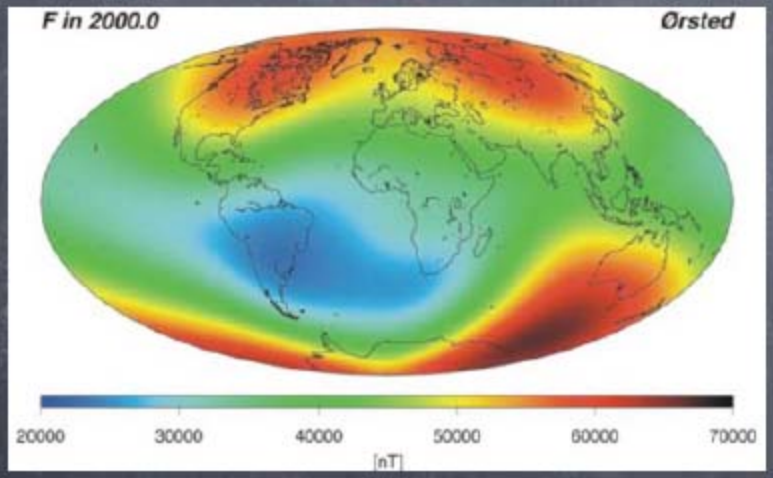
Magnetic fields move through the body freely as if it wasn't there—even the bones are essentially transparent. The body uses these fields to generate more cellular energy. This increased energy is needed to help the body heal and regain balance.

Visit Marcus Freudenmann's MAXAWARENESS site and learn more about how you can join the Truly Heal community

http://maxawareness.com/blog/pemf-review/

SOME SCIENCE

The intensity of pulse magnetic fields



- The geomagnetic field strength varies over the surface of the earth.
- It is shifting in gentle motions and increases in the area of fault-lines

The intensity of the magnetic field over South America has a minimum strength of 0.22 Gauss and reaches a maximum of 0.67 Gauss over South Australia, Russia and Canada.

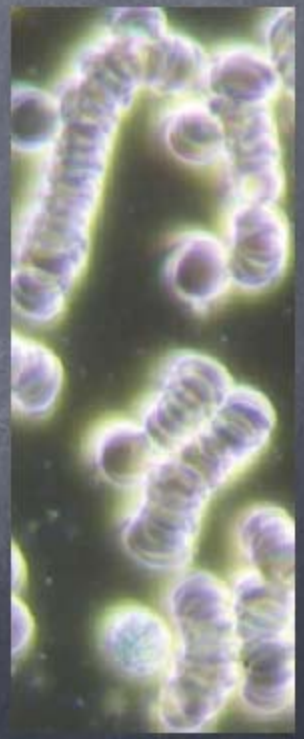
WHY DO WE NEED IT

NASA and the Russian Space Program depend on PEMF machines

General exhaustion of the body, caused by stress or chronic disease, will reduce the cell's membrane potential (usually between 70 to 90 mV).

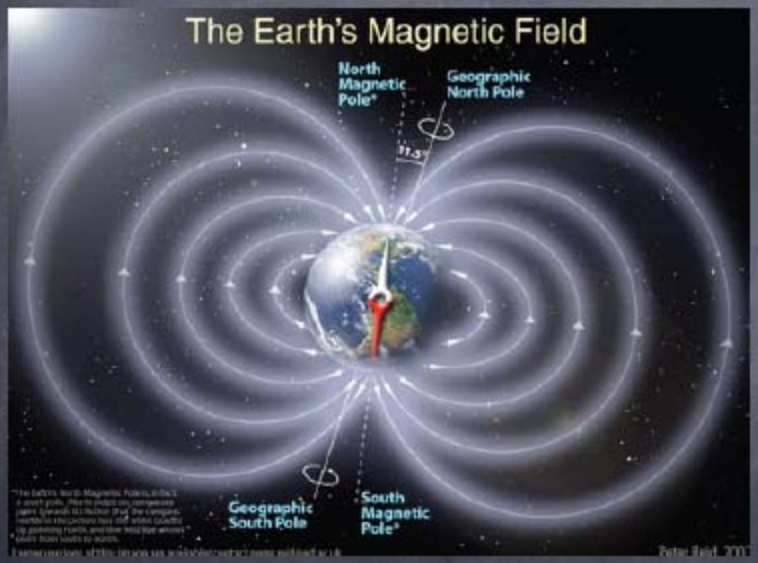
If this potential approaches the zero level, the cell dies. A cell uses 50% of its energy to maintain this potential.

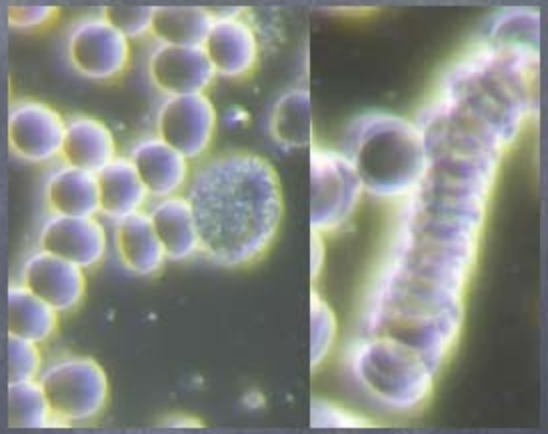
All PEMF devices are ion transport systems enabling selective movement of protons (H+ ions), These will then hyperpolarize the membrane which normalizes the cell membrane potential.

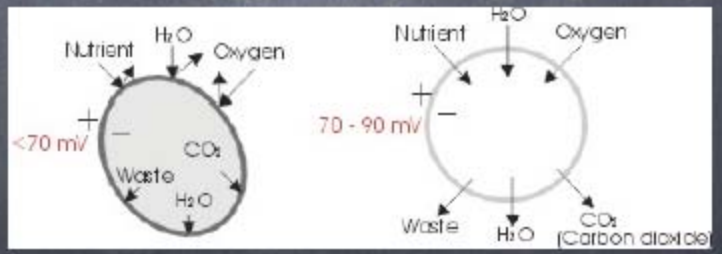


WITHOUT = NO LIFE

NASA and the Russian Space Program depend on PEMF machines

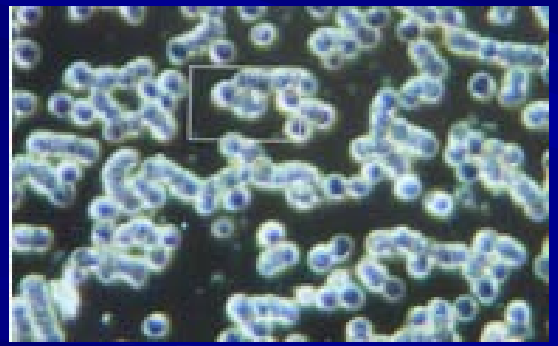






As soon as astronauts leave the magnetic field of the earth they deteriorate in energy

Energy medicine to the rescue… PEMF treatment at Arcadia Clinic in Germany



white blood cells (circled in red) in an anaerobic and inflamed body. About the size of a red blood cell, they are highly compressed and completely immobile. There was hardly any movement visible inside the cell.

Patient complained of severe fatigue and high levels of stress. Blood sample viewed under dark field microscope reveals red blood cells are sticky and clumped together indicating their energy and cell membrane charge is too low…

Sick cells such as these are unable to take in oxygen or therapeutic nutrients

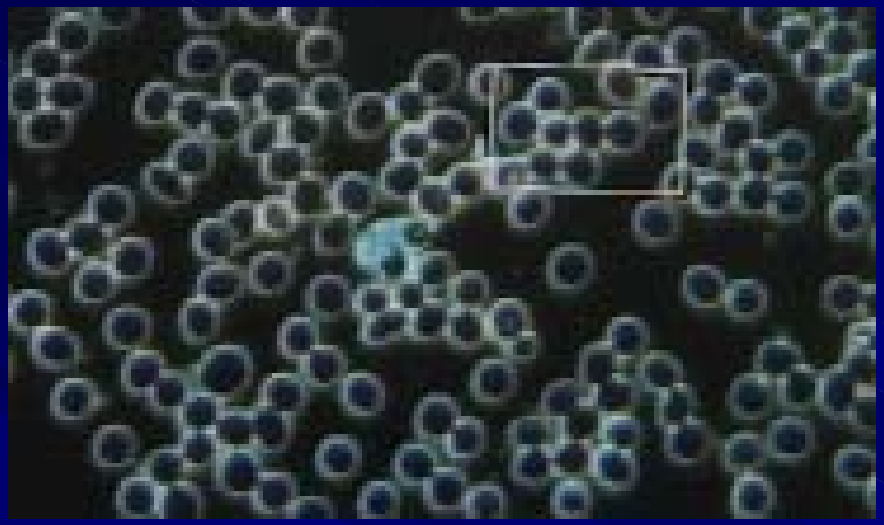
Pulsed Electro-Magnetic Frequency (PEMF) with the PMT-100

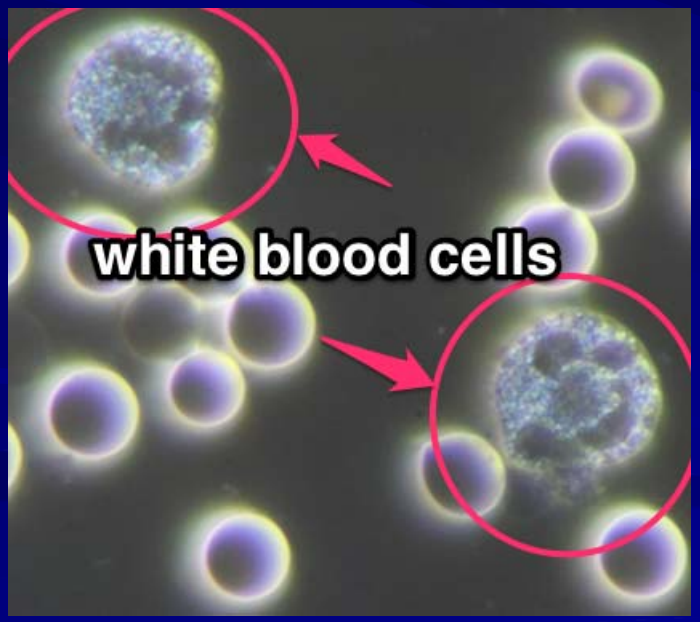





After first blood test results, patient has 15 minute PEMF session with PMT-100. The coil being placed over various areas of the body – lower torso and sacral area, leg and hip, back/spine, shoulders, breast and lymph areas, thyroid, etc.,. patient is able to personally control the strength of magnetic pulsations at each area for their own comfort level.

Post-treatment blood analysis – same patient just 20 minutes later!





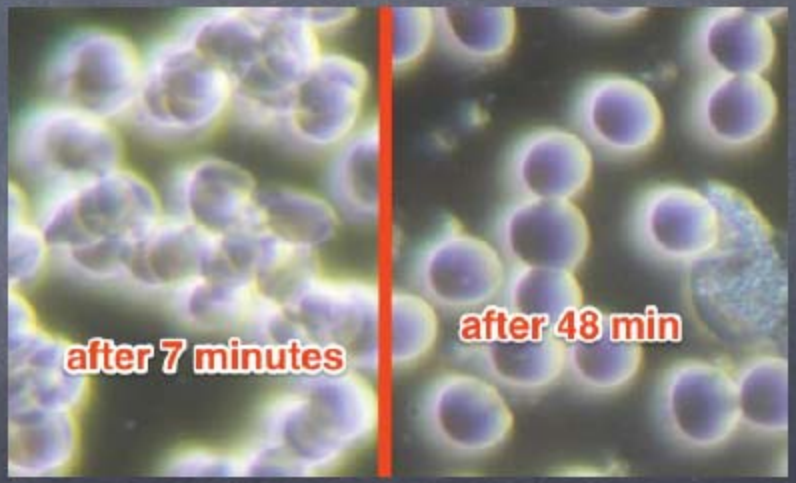
As you can see the white blood cell is about 3 times the size of the red blood cell and it moved very actively across the screen. Inside the cell the cytoplasm shimmered and moved vibrantly.

All cells held the membrane stability and mobility for around 45 min, and tested again the next day, in the morning, the blood appeared the same – still energized .

INCREASED ENDURANCE

We also noticed a dramatic improvement in the endurance of the cells. Normally after about 3 - 8 minutes under the dark field microscope we could observe a break down of the red blood cells.

After giving the patient just 20 min of treatment with the PMT100 all cells lasted without any deterioration over 45 minutes.
The patient immediately felt the change in energy




WARNING
Plug in both cable ends before turning main power on.
Turn main power off before unplugging any cable end.

OXFORD JOURNALS

JNCI JOURNAL OF THE NATIONAL CANCER INSTITUTE

Oxford Journals Medicine
JNCI J Natl Cancer Inst
Volume96, Issue24
Pp. 1805-1806.

Energy Boost: The Warburg Effect Returns in a New Theory of Cancer

Ken Garber

In 1930, German biochemist Otto Warburg, M.D., proposed that cancer was caused by altered metabolism—deranged energy processing—in the cell. Warburg, winner of a Nobel Prize in 1931, is now considered by many to be the greatest biochemist of the first half of the 20th century. His cancer theory, though, mostly fell on deaf ears.

Now Warburg's theory is enjoying a resurrection. Two prominent cancer biologists contend that a shift in energy production from oxidative phosphorylation to glycolysis—the so-called "Warburg effect"—is a fundamental property of cancer cells, not just a byproduct of the cell's transformation into cancer.

"We think it's a requirement of transformation," said University of Pennsylvania cancer biologist Craig Thompson, M.D. " You can't become fully transformed until you've had this shift." If Thompson is right, the implication is enormous: a whole new area of vulnerability for cancer cells, one that promises novel targeted treatments. " Can we exploit any of this for therapeutic reasons?" asked Chi Dang, M.D., Ph.D., a cell biologist at Johns Hopkins University Medical School in Baltimore who is doing similar work. "The answer is going to be yes."

PEMF's are like a spark plug or catalyst for energy production in the cell.

Just like a car needs oxygen, fuel and an ignition or spark plug, so does the human cell need fuel (glucose), oxygen and a "spark plug" or ignition. This ignition is PEMF or pulsed magnetic energy from both the earth and movement/exercise on the earth.

We can also think of PEMF as a battery recharger for the human cell. We now know that the voltage of a healthy cell is about 70-110 millivolts and when we get sick that voltage drops below 50 millivolts or less and cancer cells are 30 millivolts or less. Pulsed electromagnetic fields (PEMF) act like a catalyst and battery recharger for the human cells and these PEMF's are critical for human metabolism.

PEMF's also improve microcirculation, oxygenation (up to a 200% increase), help in nerve regeneration, pain management and many other health promoting benefits. There are over 1000 clinical studies and over 7000 research papers validating the therapeutic benefits of PEMFs.

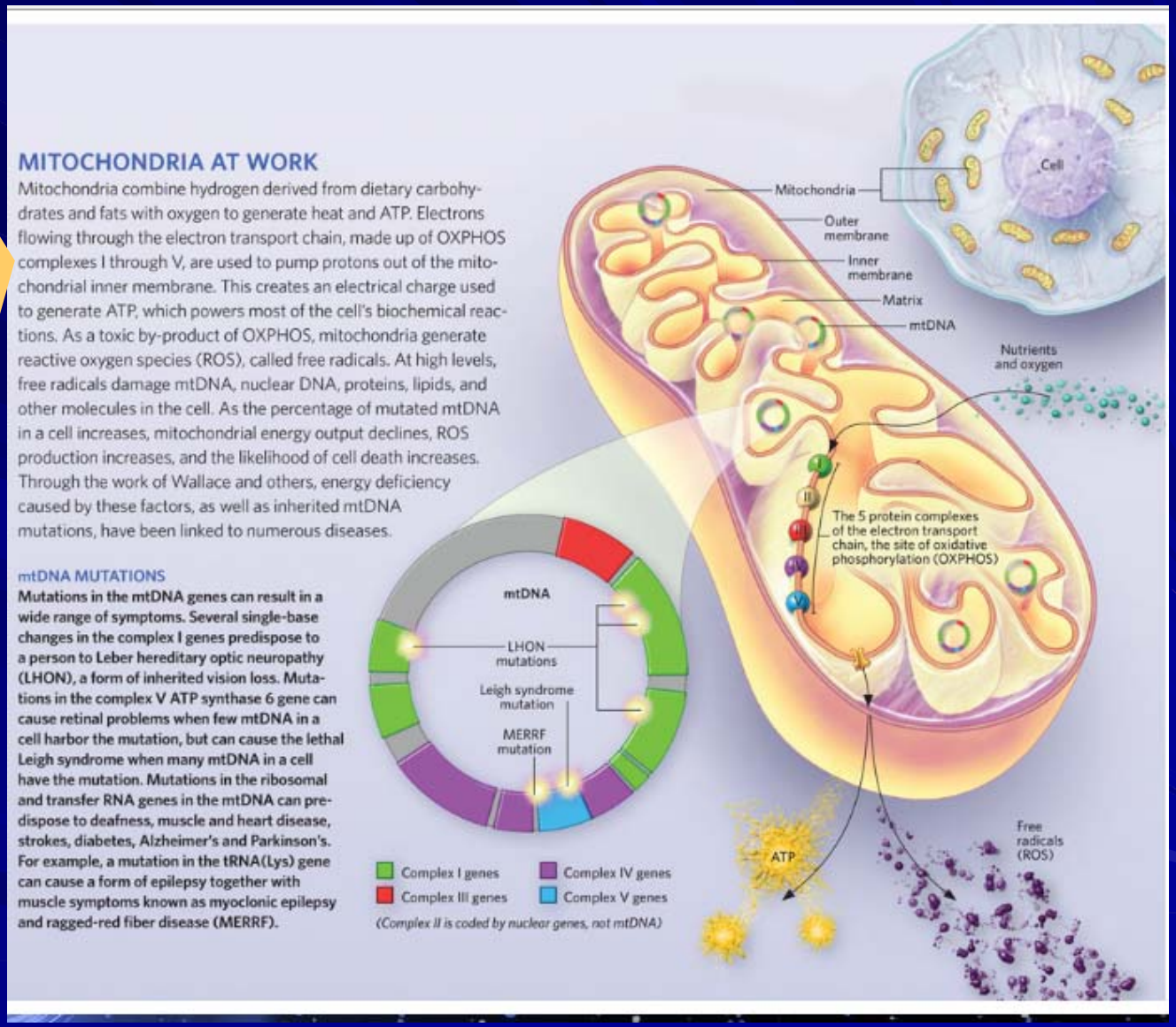
http://www.pemft.net/the-5th-element.html

Mitochondria – The Body's Powerhouse

Mitochondria combine hydrogen derived from dietary carbs and fats with oxygen to generate heat and ATP.

Electrons flowing through the electron transport chain, made up of OXPHOS complexes I through V, are used to pump protons out of the mitochondrial membrane.

This creates an ELECTRICAL CHARGE used to generate ATP, which powers most of the cell's biochemical reactions.



http://images.the-scientist.com/content/images/articles/58132/mitochondria_at_work.jpg

Cancer, Dental Heavy Metals & Lasers
Dr. Simona Pop

Criteria of research

Thirty people took part in the study:

- Ovarian Carcinoma, 19 women (24 -60 yrs)
  (CA-125 > 35 U/ml, Stage II, III,)
- Prostate cancer, 11 men, (27-55 yrs)
  (PSA 10 – 28 ng/ml)

- All the patients have been given vaccines in the past, had amalgam fillings, had prosthodontic treatment , orthodontic appliances or implants.
- At the time of the study, all the patients had complete dental metal replacement.
- All the patients had the Melisa test and the most positive responses were to: mercury, gold, platinum, palladium, silver, copper, titanium, tin, nickel, chromium, cobalt, cadmium, manganese, and thimerosal.

- All patients had undergone minimum one conventional treatment during a period of 10 to 25 years.
- The conventional treatment was unsatisfactory, some patients had had metastasis 0.5-3 years prior to the study.

- During the heavy metal detox treatments, from the total of 30 patients
- Half were in the control group without laser treatments,
- The other half were exposed to laser therapy (SLBP) one or more sessions.

Treatment

- All the patients in both groups were treated with detox treatment:
  - DMSA,
  - Vitamin C,
  - Glutathione,
  - Na Selenite
  - Mineral and Vitamin supplements.
- Half of them receive the laser therapy

Laser Therapy

- The treatment course consisted of 22 exposures distributed over 5 ½ weeks. There were four sessions per week.
- Another 22 sessions over 4 weeks were offered to patients who received the first course but showed minimal improvement after the first course.
- Ten patients completed the second course of 22 laser treatments

Laser Specifications

- soft laser
- category 1 or lower
- penetrates up to 17 mm
- improves the energy supply of the cell by direct effect on ATP- production
- improves cell regeneration - photonic effects take place in the cellular field

- The preliminary data show that increasing the number of laser sessions from 22 to 44 sessions did improve the efficacy of the treatments and resulted in total recovery in the majority of treated patients.

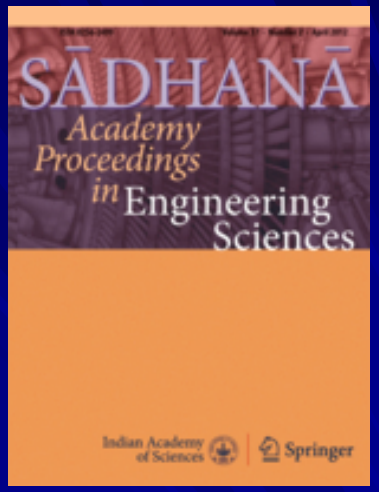
Conclusions

- The health in all patients treated by dental metal replacement and detoxification improved .
- Patients who received laser treatments had faster recovery.
- Patients who received the additional 22 laser sessions had complete health recovery in a short time.

Sadhana - June/August 2003, Volume 28, Issue 3-4, pp 639-656

Biomaterials and magnetism

D. Bahadur, Jyotsnendu Giri

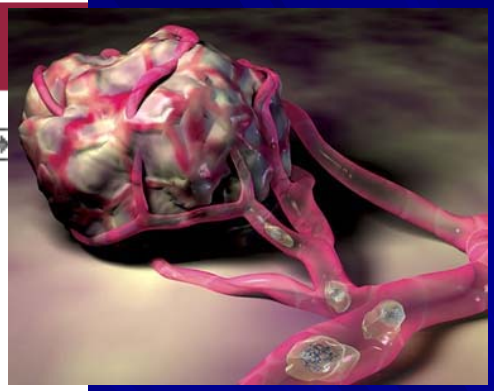


Magnetism, which is an intrinsic property of every atom, has a profound influence on living organisms. The haemoglobin in our blood is an iron complex and is magnetic in nature. There is now substantial evidence that all living organisms, including animals and humans, contain magnetic particles and act as magnetic receptors. It is established that the magnetism and magnetic materials have a strong role to play in health care and biological applications.

Of late the combination of fine particles and magnetism in the field of biology and biomaterials has been found useful in sophisticated bio-medical applications such as cell separation, drug delivery and magnetic intracellular hyperthermia treatment of cancer.

The activity of most pharmaceuticals or drugs against certain diseases or disease sites suffers from their inability to accumulate selectively in the pathological organ, tNo. or cells. When the drug or pharmaceutical agent is introduced into the body intravenously, it gets distributed throughout the body. Large quantities or doses have to be administered to get the required therapeutic concentration to a target site. As a result, many negative side effects may be caused by cytotoxic and/or antigenic drugs. Hence, the healthy tNo. gets exposed to higher concentrations of drugs. The situation becomes particularly critical in case of drugs having very low therapeutic indices (e.g. most anticancer drug).

Clinical Cancer Research



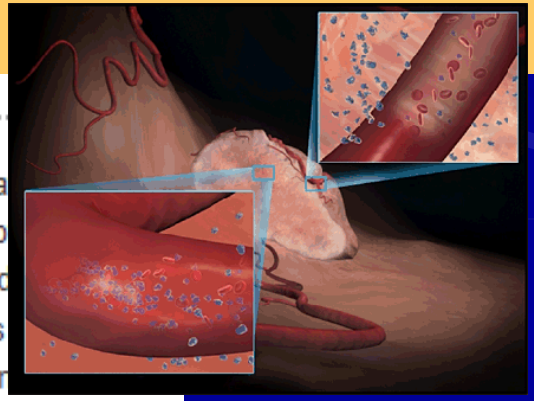
Resistance of Tumor Interstitial Pressure to the Penetration of Intraperitoneally Delivered Antibodies into Metastatic Ovarian Tumors

Michael F. Flessner, Jaewah Choi, Kimberly Credit, Ravi Deverkadra and Karla Henderson

+ Author A

Requests fo
Michael F. F
Mississippi
601-984-568

The results only partially support our hypothesis and imply that the microenvironment of the tumor is in itself a major barrier to delivery of charged macromolecules.



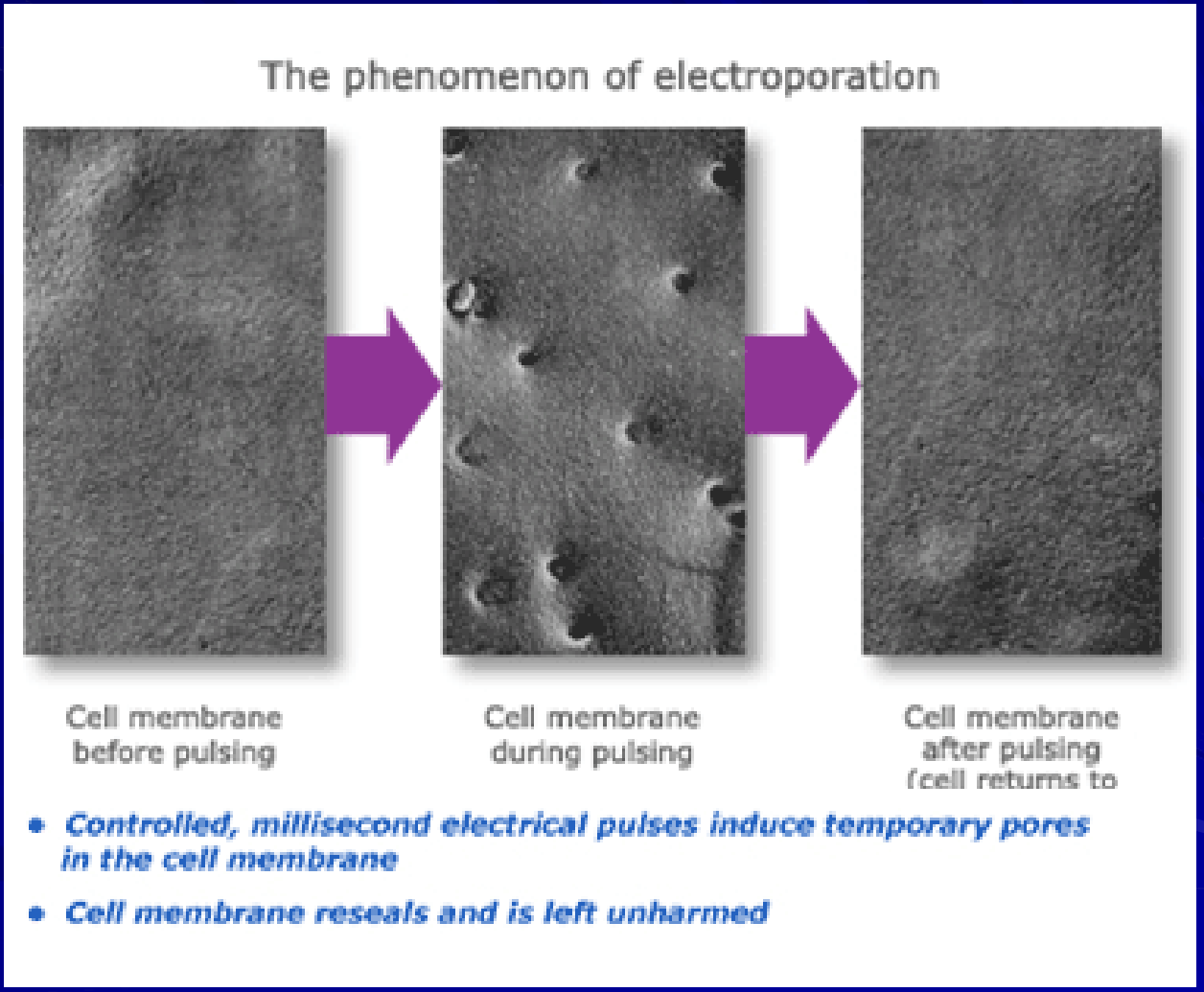
Abstract

Purpose: Despite evidence that regional chemotherapy improves the trea metastatic peritoneal ovarian carcinoma, monoclonal antibodies have no significant success in i.p. delivery. The present study was designed to add hypothesis that convective penetration of macromolecular antineoplastic agents on a positive pressure difference between the i.p. therapeutic solution and the tur

PEMF induces Electro-poration – Increasing Cellular (TMP) Transmembrane Potential

Applied PEMF stimulates electroporation of the cell membrane, where tiny pores or "ion channels" are opened during pulses.

This effect increases trans-membrane potential, electron transport, and free radical scavenging, which is significantly important for anti-agine and treating chronic diseases including cancer.



Photomed Laser Surg. 2010 Apr;28(2):159-60.

Mitochondrial mechanisms of photobiomodulation in context of new data about multiple roles of ATP

Karu T.

Various cellular responses to visible and IR-A radiation have been studied for decades in the context of molecular mechanisms of **laser phototherapy [also called photobiomodulation, low-level light therapy (LLLT)].** LLLT uses monochromatic and quasimonochromatic light in the
optical region of 600–1,000 nm to treat in a non-destructive and non-thermal fashion various soft-tissue and neurologic conditions.

This modality also was recently **used to reverse toxic effects of neurotoxins, to treat strokes and acute myocardial infarction, and to stimulate stem cell proliferation.** This multiplicity of conditions treated with photo-biomodulation has persuaded many unbelievers of the value of such an universal method.

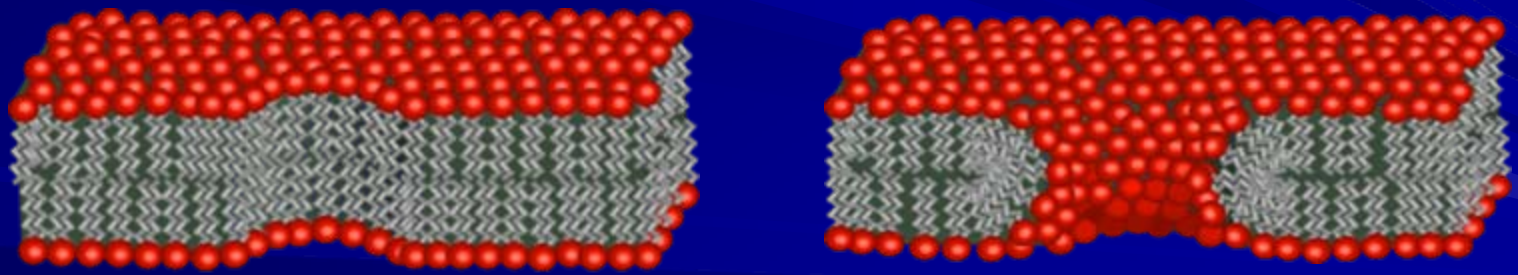
The **excitation of the photoacceptor molecule sets in motion cellular metabolism through cascades of reactions called cellular signaling or retrograde mitochondrial signaling.** Another signaling pathway starting from the mitochondria is connected with ATP. The **ATP extra-synthesis in isolated mitochondria and intact cells of various types, under irradiation with light of different wavelengths, is well documented**.

ATP is a universal fuel inside living cells that drives all biologic reactions. It is known that even small changes in the ATP level can significantly alter cellular metabolism.

Physical Mechanism of Electroporation

Electroporation allows cellular introduction of large highly charged molecules such as DNA which would never passively diffuse across the hydrophobic bilayer core. This phenomenon indicates that the mechanism is the creation of nm-scale water-filled holes in the membrane.

Although electroporation and dielectric breakdown both result from application of an electric field, the mechanisms involved are fundamentally different. In dielectric breakdown the barrier material is ionized, creating a conductive pathway. The material alteration is thus chemical in nature. In contrast, during electroporation the lipid molecules are not chemically altered but simply shift position, opening up a pore which acts as the conductive pathway through the bilayer as it is filled with water.



Schematic showing the theoretical arrangement of lipids in a hydrophobic pore (left) and a hydrophilic pore (right).

http://en.wikipedia.org/wiki/Electroporation#Electroporators

Bioelectrochemistry. Volume 79, Issue 2, October 2010, Pages 257–260

Electroporation and alternating current cause membrane permeation of photodynamic cytotoxins yielding necrosis and apoptosis of cancer cells

Nelly Traitcheva, Hermann Berg.

To increase the permeability of cell membranes for low doses of cytostatic drugs, two bioelectrochemical methods have been compared:

(a) electric pore formation in the plasma membranes by single electric impulses (electroporation), and

(b) reordering of membrane structure by alternating currents (capacitively coupled).

These treatments were applied to human leukemic K-562 cells and human lymphoma U-937 cells, yielding apoptotic and necrotic effects, determinated by flow cytometry.

Additional cell death occurs after exposure to light irradiation at wavelengths $\lambda > 600$ nm, of cells which were electroporated and had incorporated actinomycin-C or daunomycin (daunorubicine).

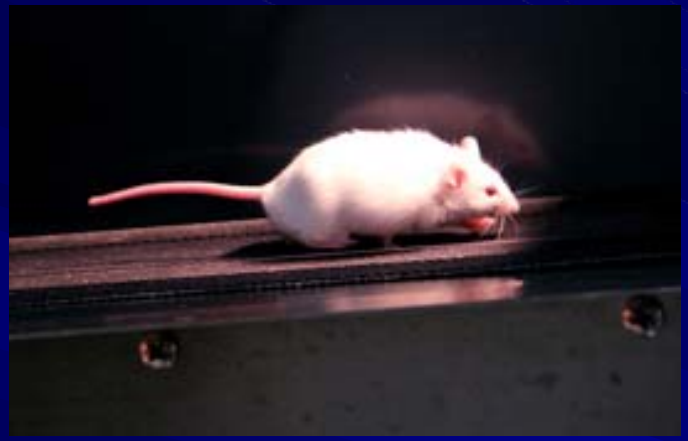
http://www.sciencedirect.com/science/article/pii/S1567539410000423

Cellular Workout: Autophagy

The cell's recycling system, may be responsible for the health benefits of exercise.

TheScientist
MAGAZINE OF THE LIFE SCIENCES

By Megan Scudellari | January 18, 2012



It's indisputable—exercise is good for you. But on a molecular level, scientists aren't really sure why.

Published online today in *Nature,* researchers show that a cellular housekeeping mechanism, called autophagy, could be the source of the beneficial effects of exercise, including protection against diabetes.

Targeting the pathway could mimic the health effects of exercise—all the perks with none of the sweat—and help treat type II diabetes, the authors suggest.

Autophagy is an internal recycling system that degrades damaged or unwanted organelles and proteins in a cell and produces energy. In animal models, this process has been shown to protect against cancer, neurodegenerative disorders, infections, diabetes, and more. "Exercise is known to protect against all these same diseases," said Beth Levine, a biologist at the University of Texas Southwestern Medical Center, "so it made sense to us that exercise might induce autophagy."

Exercise as Housecleaning for the Body

By GRETCHEN REYNOLDS, Columnist
New York Times
February 1, 2012

When ticking off the benefits of physical activity, few of us would include intracellular housecleaning. But a new study suggests that the ability of exercise to speed the removal of garbage from inside our body's cells may be one of its most valuable, if least visible, effects.

It's long been known that cells accumulate flotsam from the wear and tear of everyday living. Broken or misshapen proteins, shreds of cellular membranes, invasive viruses or bacteria, and worn-out, broken-down cellular components, like aged mitochondria, the tiny organelles within cells that produce energy, form a kind of trash heap inside the cell.

Through a process with the expressive name of autophagy, or "self-eating," cells create specialized membranes that engulf junk in the cell's cytoplasm and carry it to a part of the cell known as the lysosome, where the trash is broken apart and then burned by the cell for energy.

Without this efficient system, cells become choked with trash and malfunction or die.

Autophagy. 2007 Jan-Feb;3(1):28-31. Epub 2007 Jan 3.

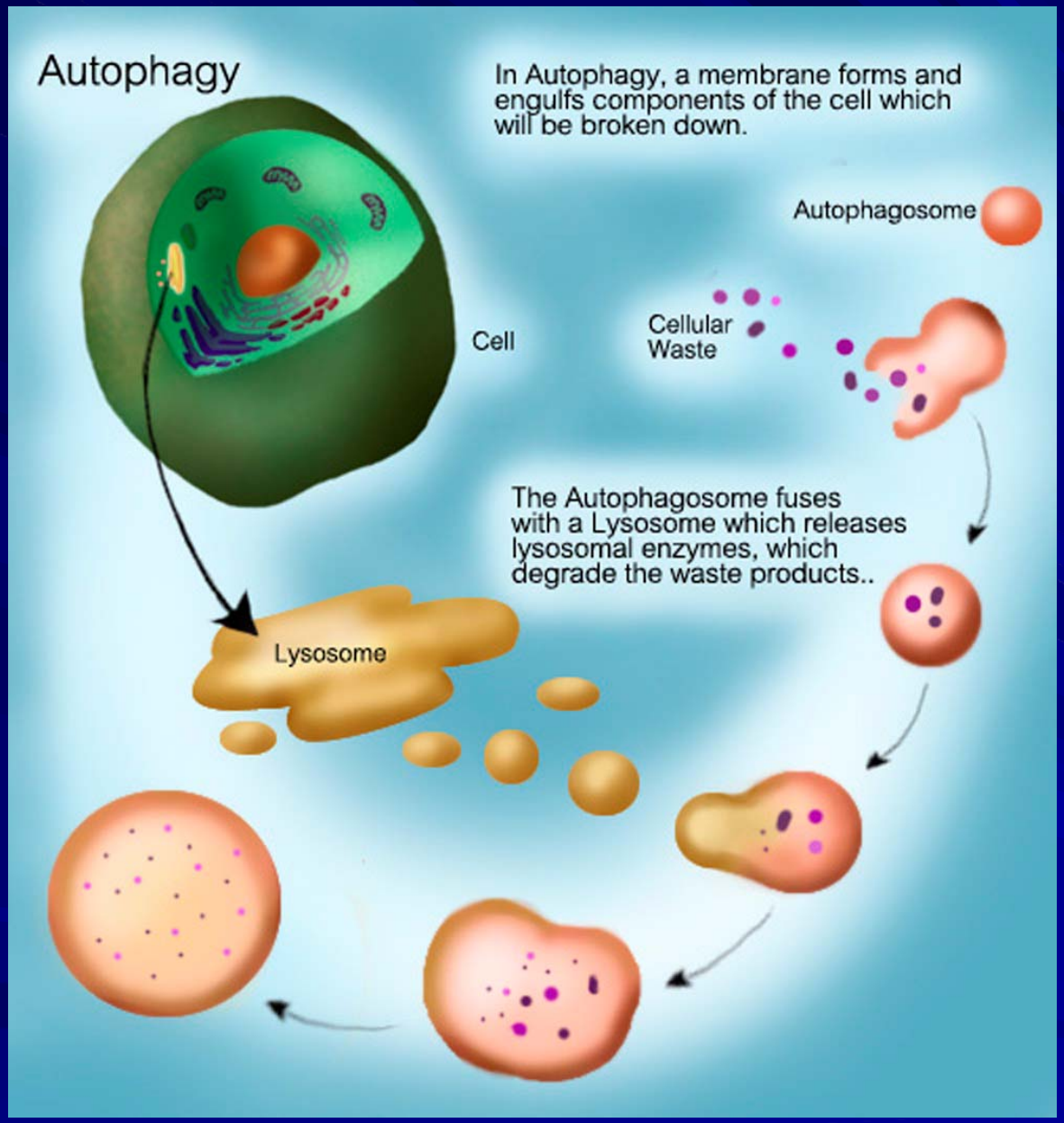
Role of autophagy in cancer: management of metabolic stress.

Jin S, White E.
Department of Pharmacology, University of Medicine and Dentistry of New Jersey, Robert Wood Johnson Medical School, 675 Hoes Lane, Newark, NJ 08854, USA.

Abstract

Human breast, ovarian, and prostate tumors display allelic loss of the essential autophagy gene beclin1 with high frequency, and an increase in the incidence of tumor formation is observed in beclin1(+/-) mutant mice. These findings suggest a role for beclin1 and autophagy in tumor suppression; however, the mechanism by which this occurs has been unclear.

We found that metabolic stress is a potent trigger of apoptotic cell death, defects in which enable long-term survival that is dependent on autophagy both in vitro and in tumors in vivo. These findings raise the conundrum whereby inactivation of a survival pathway (autophagy) promotes tumorigenesis. Interestingly, when cells with defects in apoptosis are denied autophagy, this creates the inability to tolerate metabolic stress, reduces cellular fitness, and activates a necrotic pathway to cell death. This necrosis in tumors is associated with inflammation and enhancement of tumor growth, due to the survival of a small population of injured cells in a microenvironment that favors oncogenesis. Thus, by sustaining metabolism through autophagy during periods of metabolic stress, cells can limit energy depletion, cellular damage, and cell death by necrosis, which may explain how autophagy can prevent cancer, and how loss of a survival function can be tumorigenic.



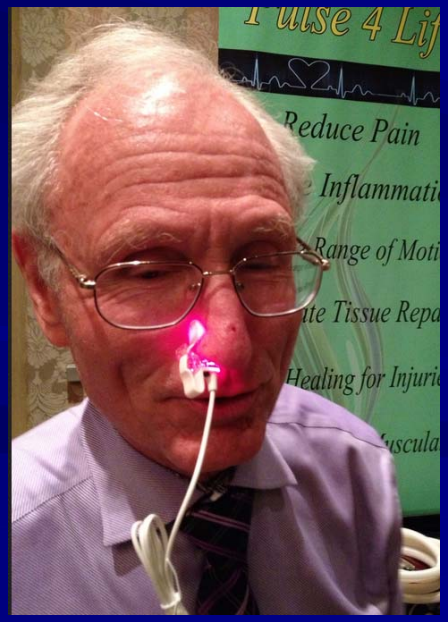
Recent developments reveal a crucial role for the autophagy pathway and proteins in immunity and inflammation. They balance the beneficial and detrimental effects of immunity and inflammation, and thereby may protect against infectious, autoimmune and inflammatory diseases.

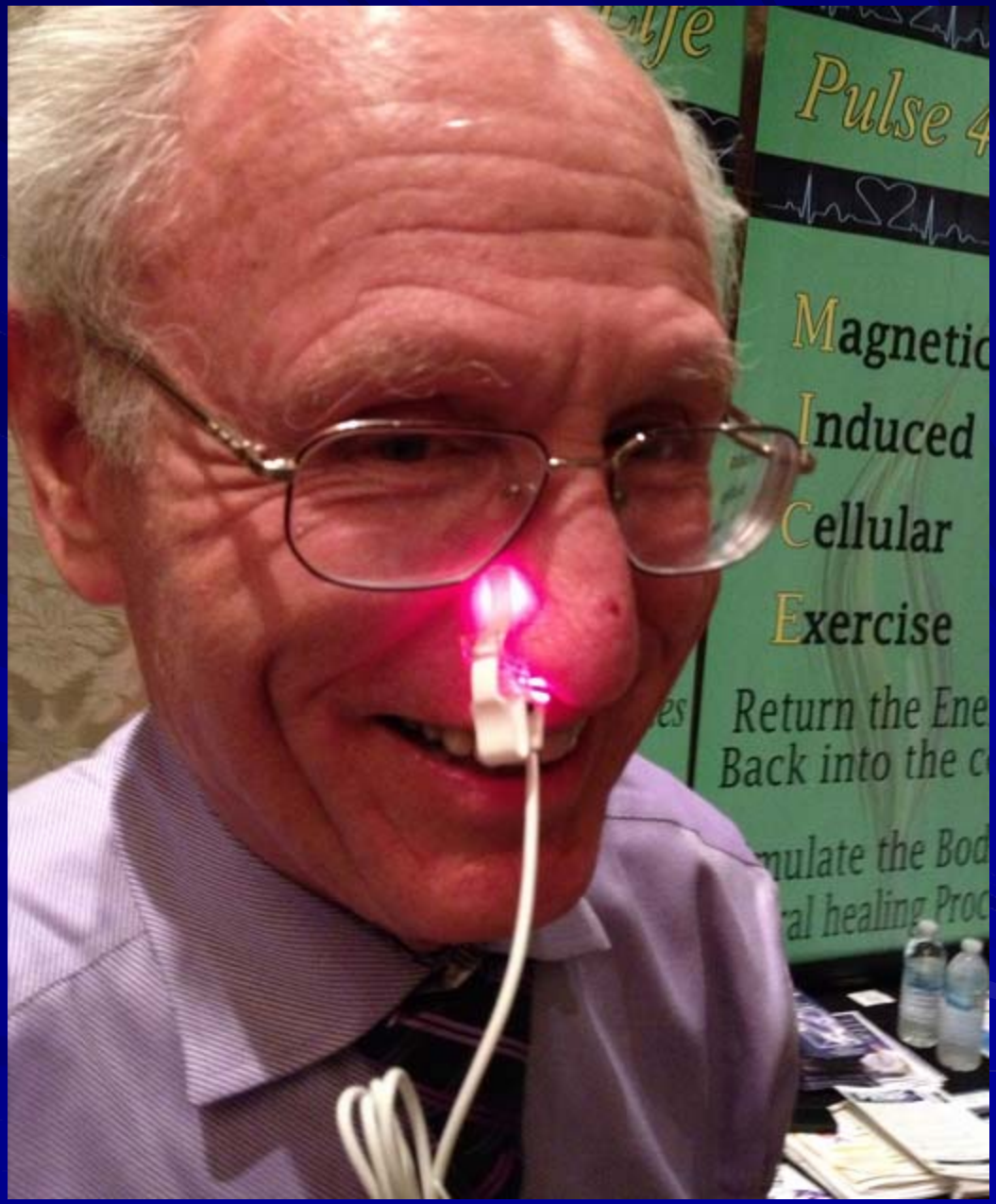
Autophagy helps the cell fight infection by some kinds of invading bacteria and viruses, by cleaning them out of the cell's interior without having to discard the entire cell.

Sustained autophagy may also increase longevity by protecting cells against free radical damage and mutations in DNA.

http://www.mesaschumacher.com/science-art-and-illustration/medical-illustration/?album=6&gallery=24


Reduce
Increase
Accelerate


Reduce Pain
Inflammati
Range of Moti
te Tissue Repa
Healing for Injuri

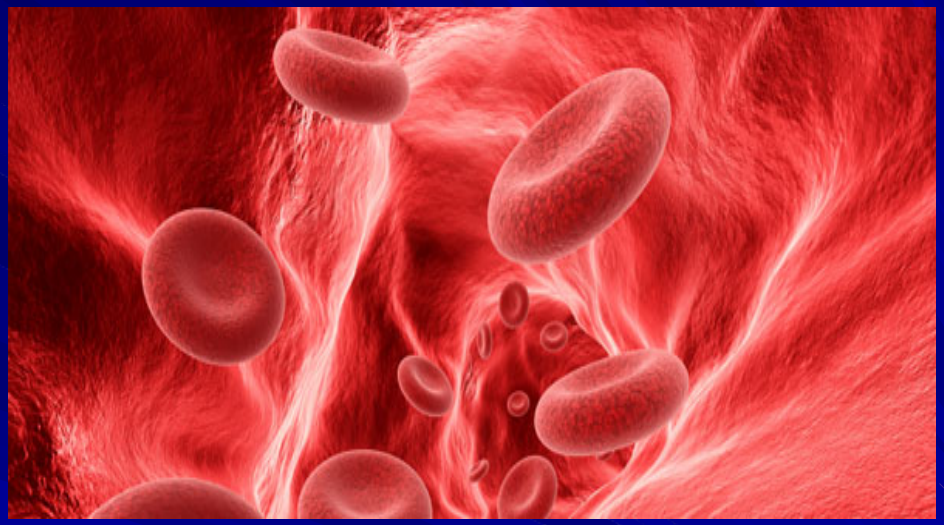

Pulse 4
Magnetic
Induced
Cellular
Exercise
Return the Ene
Back into the c
mulate the Bod
ral healing Proc

Natural Healing with Intranasal Light Therapy

MedicLights research inc.

Intranasal Light Therapy is a way to stimulate self healing and boost immunity by illuminating the blood capillaries through the nasal cavity.

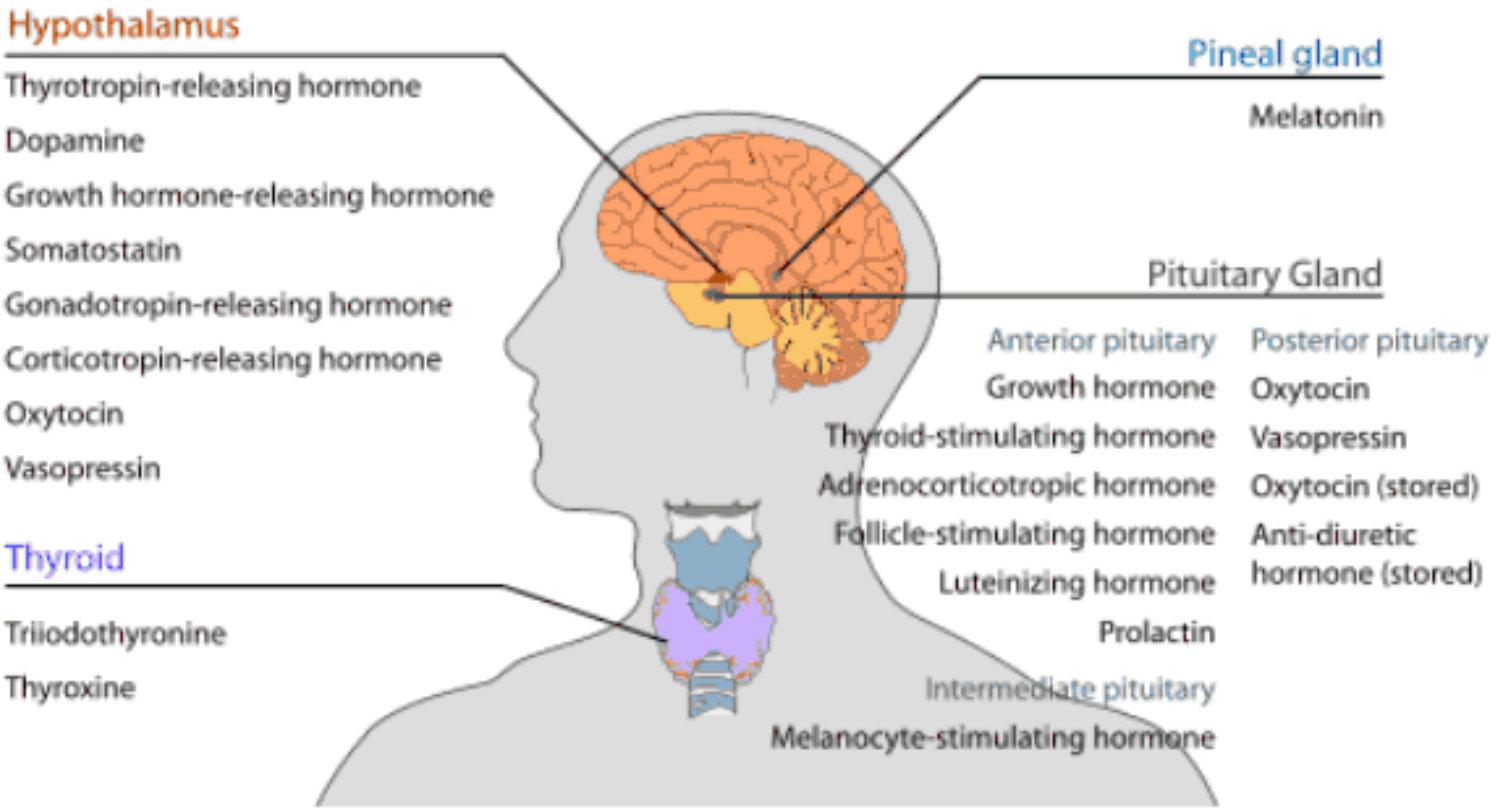
a small light diode of certain specifications designed to be inserted into either nostril for 25 minutes per day.



Intranasal light therapy oxygenates and purifies the blood, and stimulates restoration of body balance (homeostasis).

Homeostasic stimulation is achieved through the response of the mid-brain area, particularly the hypothalamus being in close proximity to the nasal cavity, and the stimulation of redox signaling molecules and their subsequent distribution through the nasal capillaries and the circulatory system.

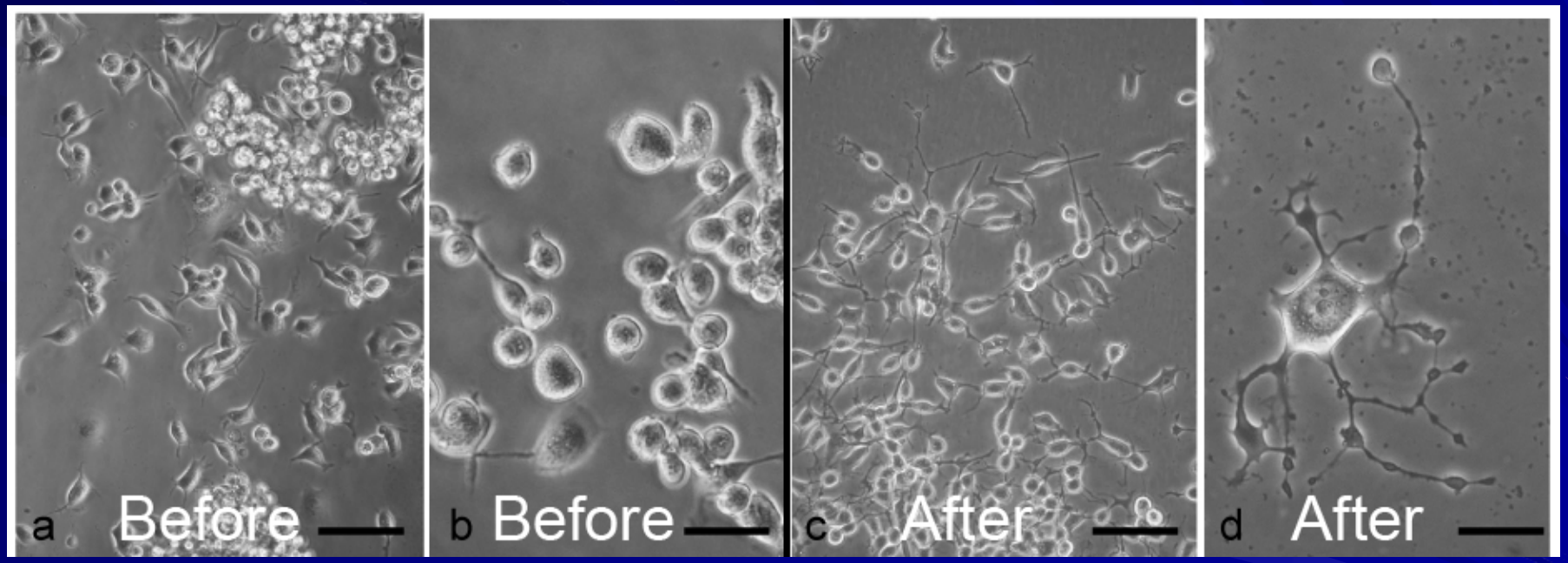
This is the "master control" for the endocrine system (pineal/pituitary/hypothalamus) for the entire glandular system of over



http://en.wikipedia.org/wiki/Endocrine_system

Tour of the brain http://www.alz.org/braintour/cortex.asp

Brain Cell Healing



In vitro post-oxidative stress. 670nm, 3 mW, 20 sec/day, 5 days

Source: Giuliani et al. Low infra red laser light irradiation on cultured neural cells: effects on mitochondria and cell viability after oxidative stress. BMC Com Alt Med 2009, 9:8.

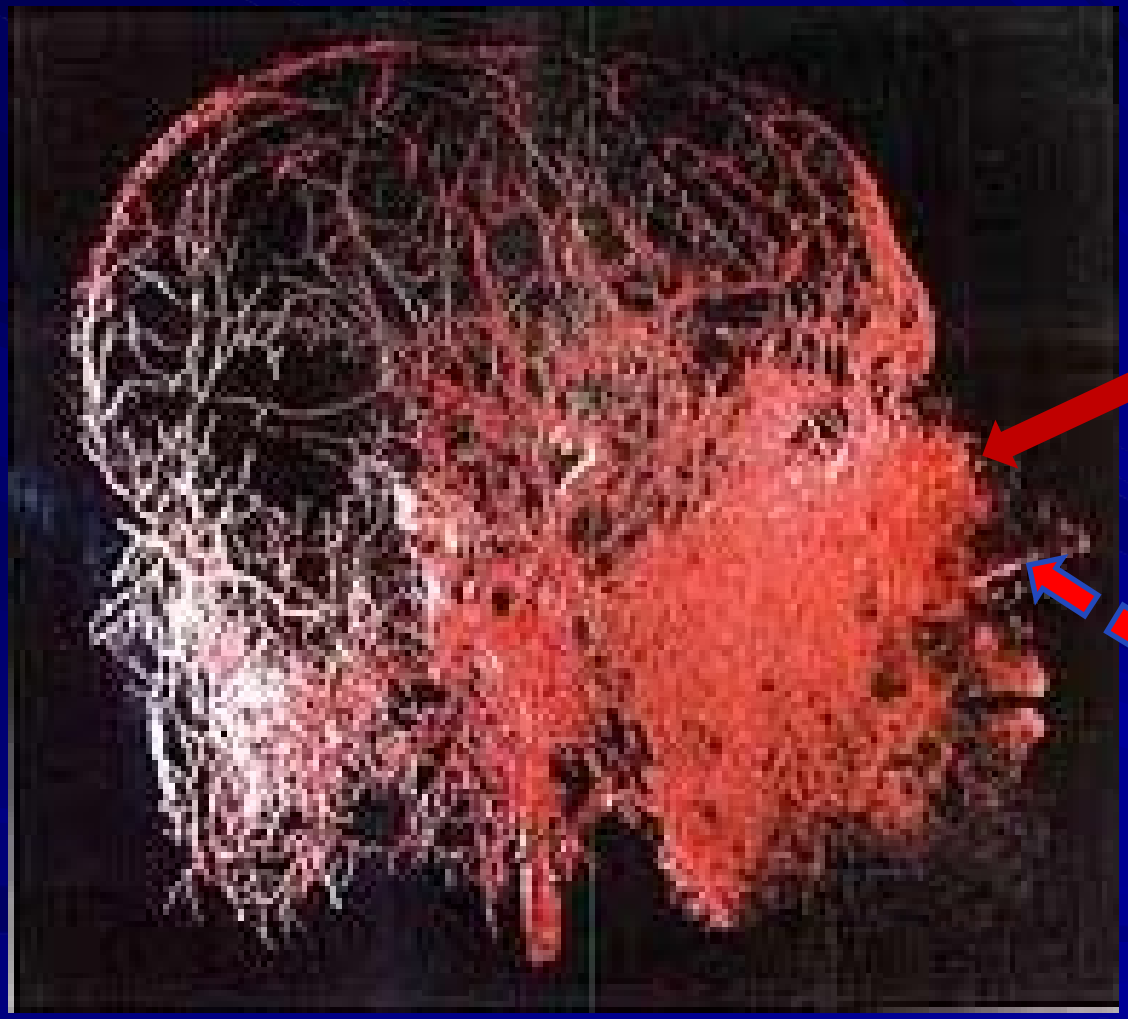
History of neurological evidence

With present Intranasal Low Level Laser Therapy parameters

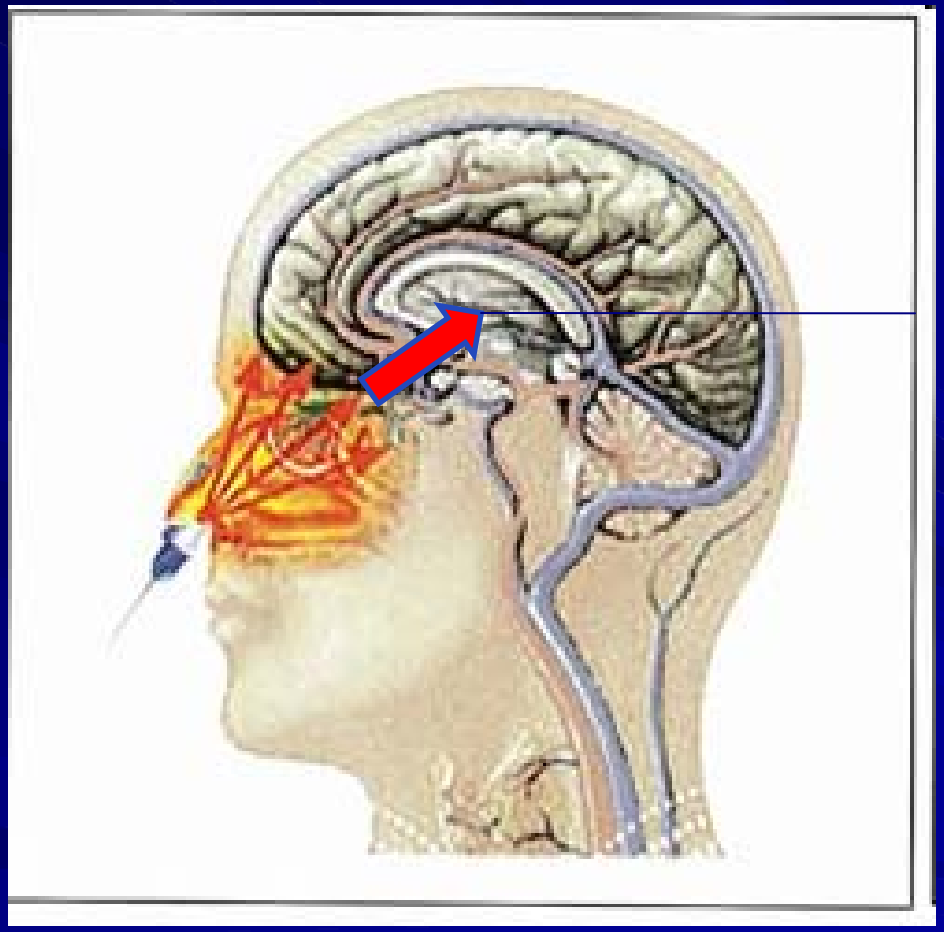
- **Facial pain, 1998**
- **Intractable headache, 1998**
- **Cerebral thrombosis, 1999**
- **Parkinson's disease, 1999**
- **Alzheimer's disease, 1999**
- **Mild cognitive impairment, 2000**
- **Insomnia, 2001**
- **Post-stroke conditions, 2003**
- **Migraine, 2003**
- **Traumatic brain injury, 2003**
- **Schizophrenia, 2000**
- **Vascular dementia, 2005**
- **Cerebral palsy, 2007**

Can we still improve?

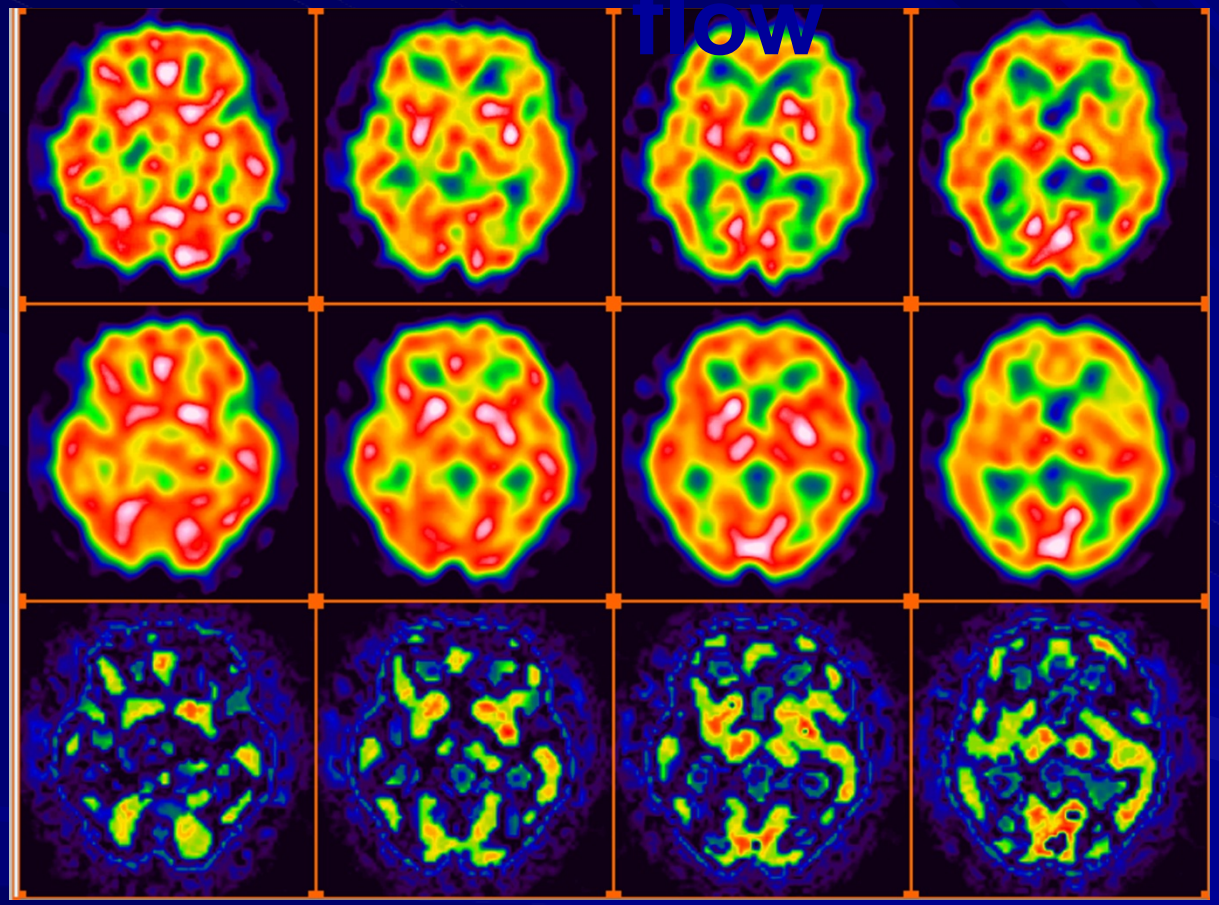
Blood vessels of the Human Head



Illuminating the Mid-brain



Improvement in brain blood flow



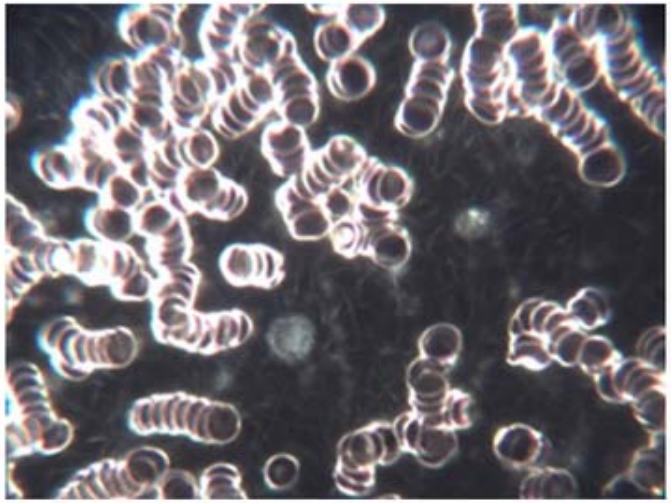
Before

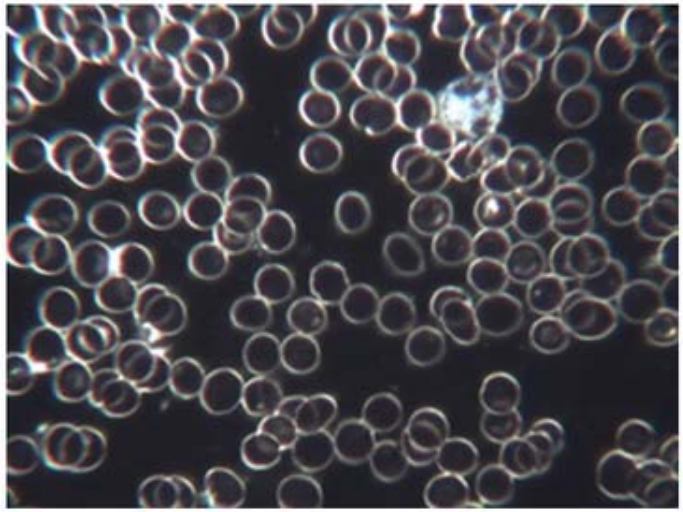
After

Net increase

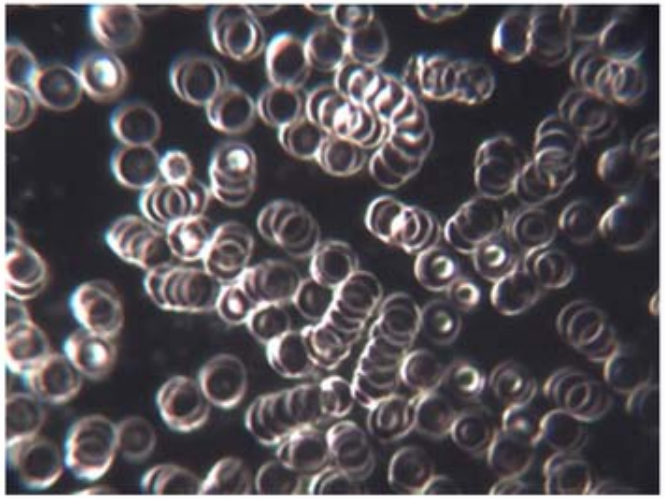
Stroke patient. 650 nm, 4 mW, 30 min/day, 7.2 J/cm²/day, 10 days

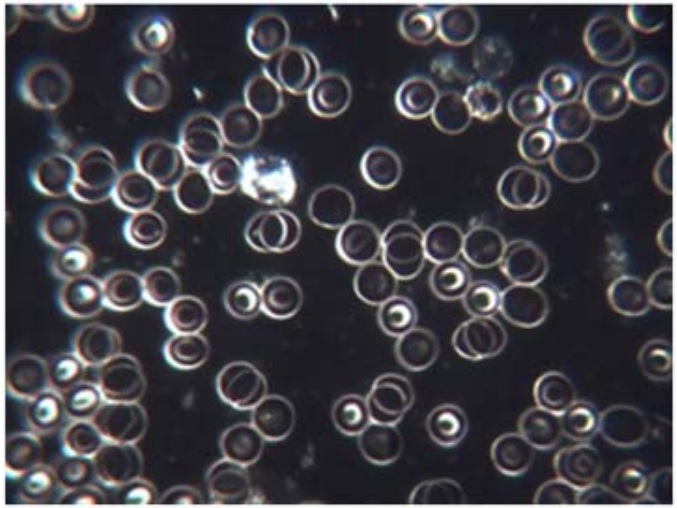
Source: Single photon emission computed photometry by Prof Xiao XC, 2005



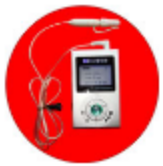


Left before and after 25 minute use of the Vielight on the right notice the red blood cells on the left are stacked with poor amount of surface area available for the exchange of oxygen

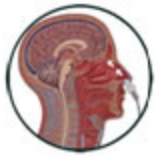




Intranasal Low Intensity Laser Therapy (ILILT)
blood purifying effects



ILILT



blood circulation and effects to whole body's blood

ILILT's Biostimulatory effects to blood

1. Increased ATP production by the mitochondria and increased oxygen consumption on the cellular level, which may result in muscle relaxation
2. Increased serotonin and increased endorphins
3. Increased anti-inflammatory effects through reduced prostaglandin synthesis
4. Improved blood circulation to the skin in cases like neuralgia and diabetes mellitus
5. Decreases permeability of the membrane of the nerve cells for Na/K causing hyperpolarisation
6. Increased lymphatic flow and decreased edema

Adjuvant therapy

- **Hypertension** <<Learn more
- **Hyperlipoidemia** <<Learn more
- **Hyperviscosity** <<Learn more
- **Stroke** <<Learn more
- **Sugar diabetes** << Learn more
- **Insomnia** << Learn more

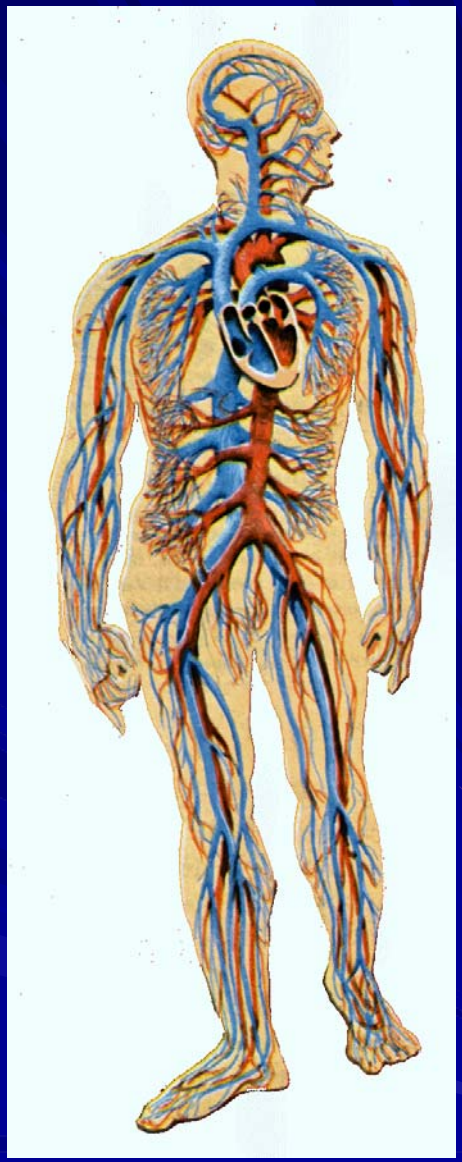
Health care

- Blood health care
- Improve natural immunity
- Anti-ageing
- protect the ischemic anemia

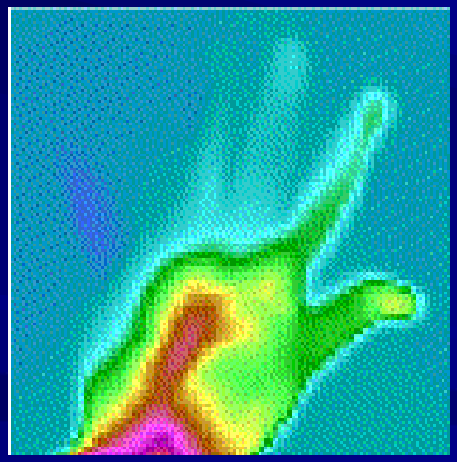
Healing Distributed through the Circulatory System

The combined roles of singlet oxygen, ROS, Redox Signalling and the activity of SOD best explains the mechanism behind the healing success of Intranasal Light Therapy. The key to the efficacy of the intranasal pathway is that it is essentially an in vivo method without the invasiveness of the older intravenous method.

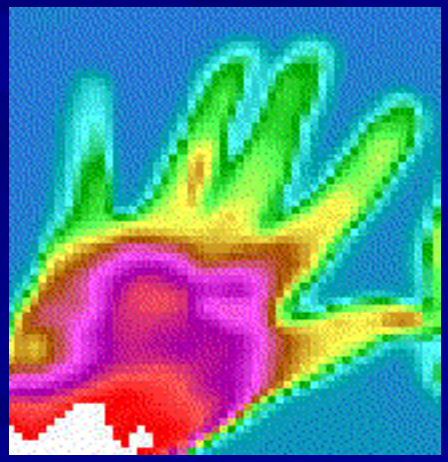
The rich vascular bed in the nasal cavity is an excellent starting point to carry and distribute Redox Signalling molecules throughout the body to stimulate the healing process.



Peripheral blood circulation



Before



After

Thermal texture map by Prof Liu SH

Electromagnetic Therapy

for energy production, nutrient uptake and cellular detoxification

In an article published in *Plos One*, November 2010, volume 5, issue 11 (Wang), page 4, Johns Hopkins' researchers found a 38% increase in ATP production in P12 cells that were placed in a static magnetic field device that we supplied.

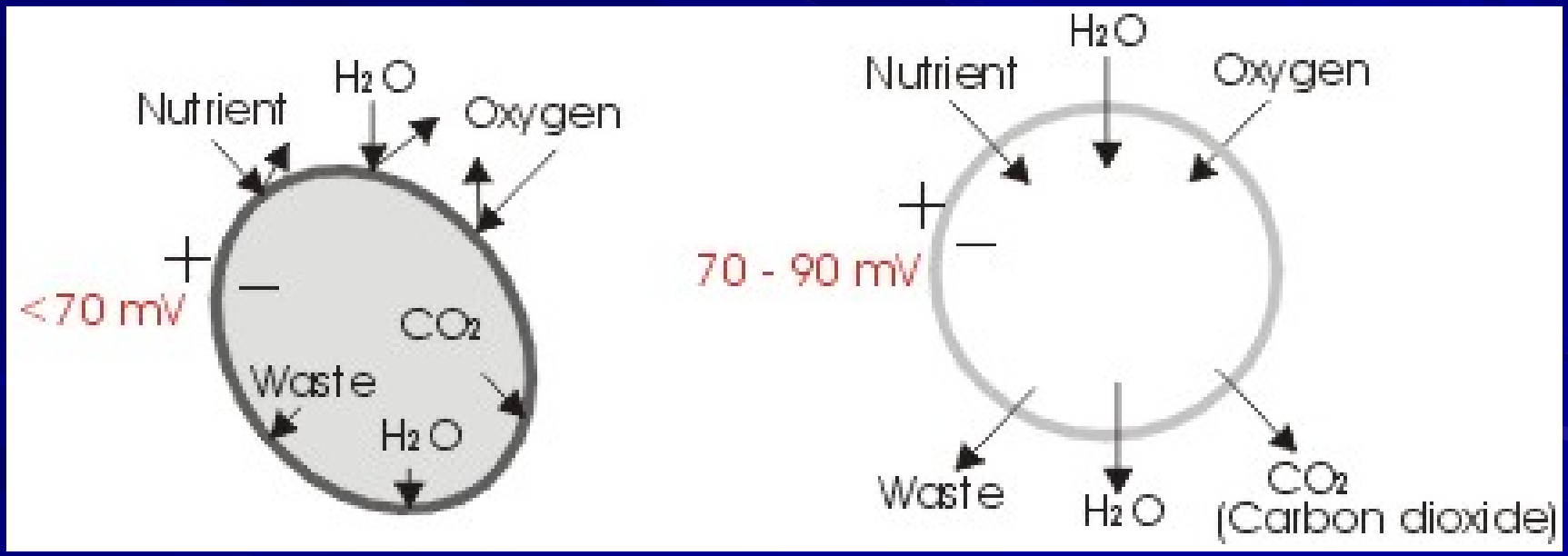
This increase could be much higher *in vivo* with the brain's pulsed DC electromagnetic field interacting with an enhanced earth-type field resulting in increased resonance of the mitochondria. All of this leading to enhance electron transfer in the creb cycle resulting in more ATP production.

↑ ATP equals ↑ Na+ K+ pump function which leads to ↑ charge of the cell wall, facilitating ↑ nutrient/drug uptake and waste and toxic metal excretion.

PEMF Exercise Therapy can Increase the Effectiveness of Anti-oxidants 100 Fold!

PEMF creates a Negative-Potential energy field to induces subtle current flows and generate a very large amount of negative ions inside human body. Negative Ions stimulate the activity of the Na+/K+-ATPase to enhance Na+/K+ pump and to maintain the cell potential at 70 – 90 mV.

Increasing cellular energy and membrane potential assists in uptake of oxygen, H2O, anti-oxidants and other critical nutrients into the cell…while toxins, cellular waste and carbon dioxide are purged.



Low energy "sick" cell < 70mV

Normal healthy cell = 70-90 mV

Dr. Gordon's Personal Daily Supplement Regimen

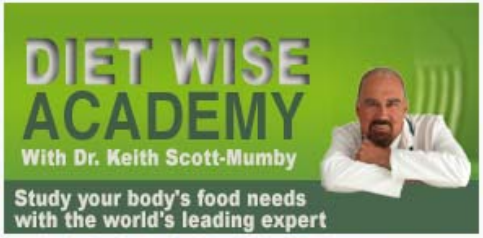
10 mins PEMF assisted Magnetically Induced Cellular Exercise twice per day

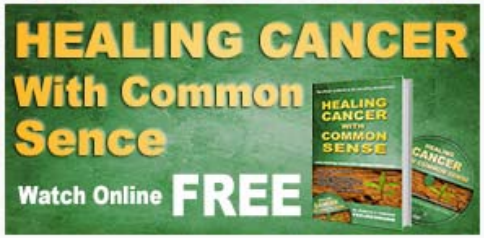
- Acetyl L-Carnitine (558 mg) 1 BID
- Adrenal Support, 1 BID
- Liquid Cellular Glutathione
- Liquid Colloidal Cellular Silver
- ACZ liquid zeolite
- Aloe caps for immune function
- B12 Sublingual, one at night
- Multivitamin and Chelation supplement
- Growth Hormonal Supplement With Resveratrol
- Lithium Orotate
- Herbal Brain Enhancer
- Boluoke Lumbrokinase
- D' Ribose
- DHEA 50 Milligram
- Benfotiamine
- 100% Chelated Magnesium Glycinate/Lysinate
- FibroBoost
- Phytosome Curcumins
- CoQ10, 100 mg one daily
- Mena Q7/Vitamin K2
- Quercetin Bromelain
- Trans-Resveratrol
- Pueraria mirifica (Herbal Remedy from Thailand)
- Hyal-Joint, 20 mg, one daily
- Immune System Support
- Kyodophilus probiotics
- L-5-HTP
- Master AntiOxidants
- Maximino
- Melatonin 10 mg, nightly
- N-Acetyl Cysteine (NAC)
- Omega 3 fish oil supplement
- Vitamin E
- Power Drink – Vitamin C, Maca, Organic Greens, stabilized rice bran and Fiber,
- Pregnenolone
- Stabilized R-Lipoic Acid
- Testosterone/Progesterone/Chry-H 150/5/200
- Thyroid 2 Grains, once daily
- Thyroid Support
- Vitamin D3, 5,000 Units
- Zeolite capsules

With over 8000 members this is the world's leading alternative cancer education program

HOW MUCH VITAMIN C SHOULD WE TAKE?

That really depends on your overall condition. If you have loads of inflammations and some infections, some fungi, Candida and bacteria imbalances in your gut you can take loads for a long time to get even. It takes a while to balance the system and to reactivate your immune system to full function.





http://maxawareness.com/blog/bio-enr-gy-c-bioenergy-c-vitamin-c/

Curr Pharm Des. 2011 Dec;17(35):3861-4.

Antioxidant pathways in Alzheimer's disease: possibilities of intervention

Viña J, Lloret A, Giraldo E, Badia MC, Alonso MD.
Department of Physiology, Faculty of Medicine, University of Valencia, Avda. Blasco Ibañez, 15. 46010 Valencia, Spain. jose.vina@uv.es

Abstract

Alzheimer's disease (AD) is closely related to the occurrence of oxidative stress. Extracellular plaques of amyloid b peptides (Aβ), a hallmark of the disease, have been postulated to be more protective than damaging in terms of oxidative stress because they may be chemical sinks in which heavy metals are placed.

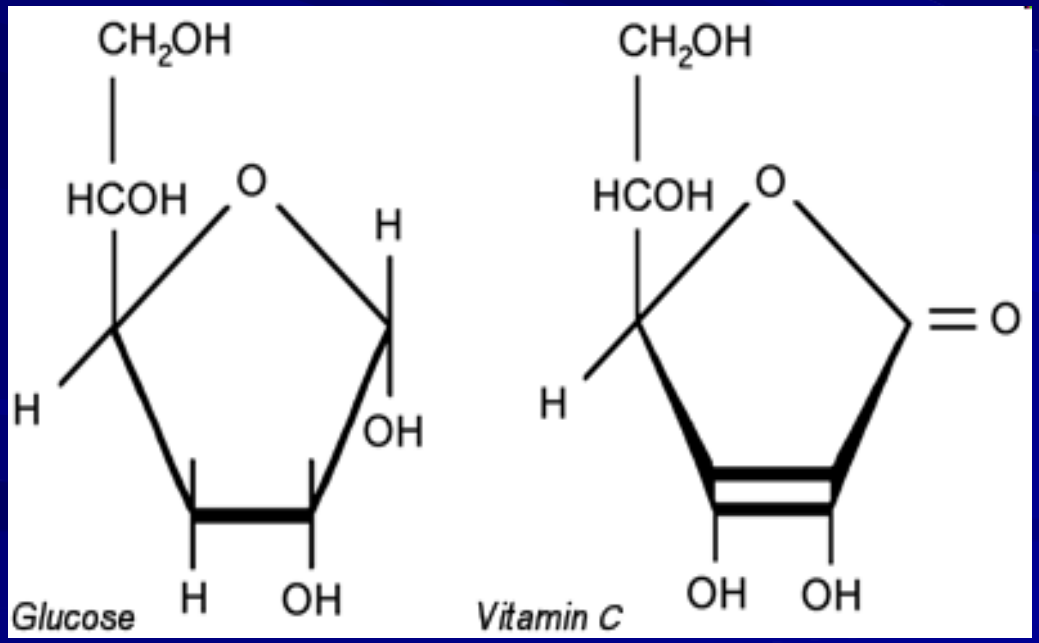
More than a decade ago we reasoned that damage due to Ab might be caused not by extracellular, but rather intracellular Ab peptide interacting with normal cell metabolism. Ab binds to mitochondrial membranes, interacts with heme and interferes with the normal electron flow through the respiratory chain. This results in a faulty mitochondrial energy metabolism and in an increased production of reactive oxygen species (ROS). The low mitochondrial energy metabolism may important to explain the hypo metabolism observed in AD patients in vivo (measured by positron emission tomography) and in isolated neurons incubated in the presence of Ab peptide. The increased ROS production results in oxidative stress.

Major efforts have been made to determine whether antioxidant supplementation could be a means of preventing, or even treating AD. We found that even though there is oxidative stress in AD, the administration of antioxidant vitamins, particularly vitamin E, is not effective in preventing the progression of the disease in all patients.

http://www.ncbi.nlm.nih.gov/pubmed/21933145

Vitamin C – Glucose Mimic "Trojan Horse" for Cancer Cells

Vitamin C is similar to Glucose in chemical configuration. Cancer cells have up to 24 times more receptor sites for glucose than healthy cells. By limiting dietary sources of glucose and supplementing with high doses of Vitamin C, cancer cells will up take in a disproportionate dose of Vitamin C, which can now act like a Trojan Horse – entering and destroying cancer cells from within.



Once inside the cell, Vitamin C metabolizes into hydrogen peroxide (H202) which selectively destroys cancer cells due to their relative deficiency of the enzyme catalase. Catalase metabolizes hydrogen peroxide into water and free oxygen in healthy cells, but is absent in cancer cells.

Vitamin C helps control gene activity in stem cells

NewsRx.com
08-09-13

Vitamin C affects whether genes are switched on or off inside mouse stem cells, and may thereby play a previously unknown and fundamental role in helping to guide normal development in mice, humans and other animals, a scientific team led by UC San Francisco researchers has discovered.

The more you study vitamin C, the more confident I am that it is a significant contributor to why at age 78 I feel this good! We all need *skin that does not sag* and *joints and tendons that do not give out from minor traumas* we all incur all the time. I just had a major wipe-out on my bicycle with head trauma, huge black eye, major joint trauma etc.,.. but I was riding my bike the next day! Please learn more about vitamin C for your own good!

I use 8000-16000 mg of BIOENERGY C and HRT ORAL AND TOPICALLY on the skin as C-PERFECTION so I DO NOT HAVE to buy STEM CELLS… I make my own!

Th
th
fe
vit
gr
or
Ju

In
int
ste

discovery emerged from an effort to compare different formulations of the growth medium, a kind of nutrient broth used to grow mouse embryonic stem cells in the lab.

http://www.newsrx.com/health-articles/3847178.html

Molecular & Cellular Biochemistry – Feb 2006

High-Dose Vitamin C Eradicates Cholesterol From Artery Walls

Researchers in New Delhi, India now demonstrate the cholesterol-eradicating effect of high-dose vitamin C in animals. Using rabbits that were force fed a high-cholesterol diet, or a high cholesterol diet plus low or high-dose vitamin C, the researchers conclusively showed the power of vitamin C to prevent narrowing of arteries with cholesterol plaque.

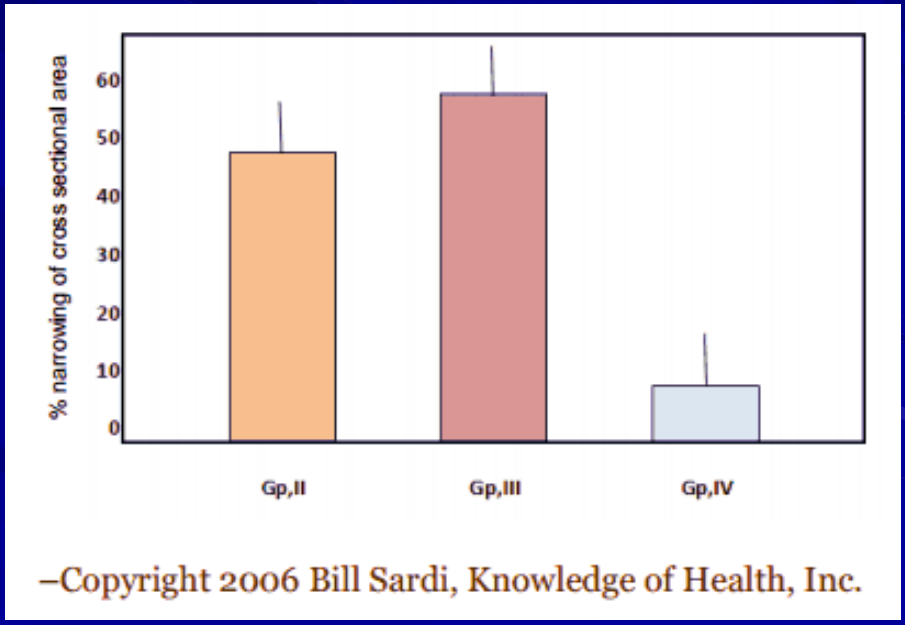
The low-dose group of rabbits were given the human equivalent of about 350 milligrams of vitamin C, and the high-dose group the human equivalent of 11,000 milligrams of vitamin C per day. The chart below shows the percentage of arterial narrowing by cholesterol.

Group II was fed cholesterol, no vitamin C.

Group III was fed cholesterol + low-dose vitamin C.

Group IV was fed cholesterol + high-dose vitamin C.

Arterial narrowing declined from about 40-50% to ~10% with high-dose vitamin C.



Bio En'R-G'y C

is an exciting new form of Ribose Nucleotide Activated (RNA) Vitamin C containing Riboperine metabolites that safely allows patients to take daily high doses without stomach upset, cramping, or diarrhea.

Each serving of Bio En'R-G'y C ‘s unique form of L-Ascorbate C crystals, has been further enhanced with 2000 mg of GMS-Ribose for increased bio-availablility.

Preliminary double blind, human trials on one or more of the ingredients of GMS-Ribose taken with Vitamin C have been shown to enhance the uptake of Vitamin C plasma levels above 30% of subjects on placebo.

MSM, methylsulfonylmethane (METH-əl-sul-FON-il-METH-ane) provides sulfur, a vital building block of joints, cartilage, skin, hair and nails, and methyl groups, which support many vital biochemical processes in the body, including energy production.

MSM is a naturally-occurring nutrient found in small amounts of many foods. As a dietary supplement, MSM is synthesized. When made correctly, it is identical to that found in nature. MSM can be taken alone or in combination with other joint health supplements, such as glucosamine and chondroitin.

GMS Ribose - a patented, proprietary blend of glycine complexed with methyl sulfone and Ribose providing methyl sulfur metabolites with Riboperine. Methyl sulfone is an important nutrient (the prime source of bio-available sulfur) used by the body for healthy and proper enzyme activity and natural hormone balance.

Methyl-sulfone is a natural form of organic sulfur found in all living organisms, including humans' body fluids and tissues. Sulfur along with Vitamin C is necessary for making collagen, the primary constituent of cartilage and connective tissue.

Inhibition of Reactive Oxidative Species (ROS) i.e. formation of Oxidative Stress by Beyond C™ - Bio En'R-G'Y C™

NIS LABS
Natural Immune Systems Inc

Background for this study: Oxidative stress is a condition in cells which is characterized by an excess of reactive oxygen species (ROS). An excess of these molecules leads to oxidative damage which plays a role in many disease processes

The method is based on challenging human cells with an inflammatory stimulus to produce damaging reactive oxygen radicals (Oxidative Stress) . Changes in the oxidative stress level in each cell are monitored by a ROS-sensitive dye.

Conclusions: Beyond C™/ Bio En'R-G'Y™ demonstrated a substantial inhibitory effect on the ROS (Oxidative Stress) formation in human neutrophil cells; displayed a maximum effect at a concentration of 1 ppm (v/v). At that dose, "Beyond C™"/ Bio En'R-G'Y™ inhibited approximately 68% of the oxidative stress caused by the peroxide challenge.

The level of ROS formation was not brought back to baseline at any of the dilutions tested, including 0.1 parts per trillion.

The ROS tests performed indicate that the compounds in the product are available to the interior of the cells.

Screening for Vitamin C in the Urine: Is it Clinically Significant?

James A. Jackson, MT(ASCP)CLS, Ph.D., BCLD; Kelly Wong, B.S.; Chad Krier, N.D., D.C.; Hugh D. Riordan, M.D.[1]

Humans cannot make vitamin C (ascorbic acid or ascorbate) and must obtain it through the diet or as supplements.[1] If taken orally several important things must occur to get an adequate supply of vitamin C to the tissues. The substance containing vitamin C must be digested, absorbed, metabolized and excreted. If given intravenously, the digestion and absorption process is, of course, bypassed.

Since vitamin C is a water-soluble vitamin, any excess in the blood should appear in the urine, providing there is normal renal function. Vitamin C disappears from the urine early in blood or tis-

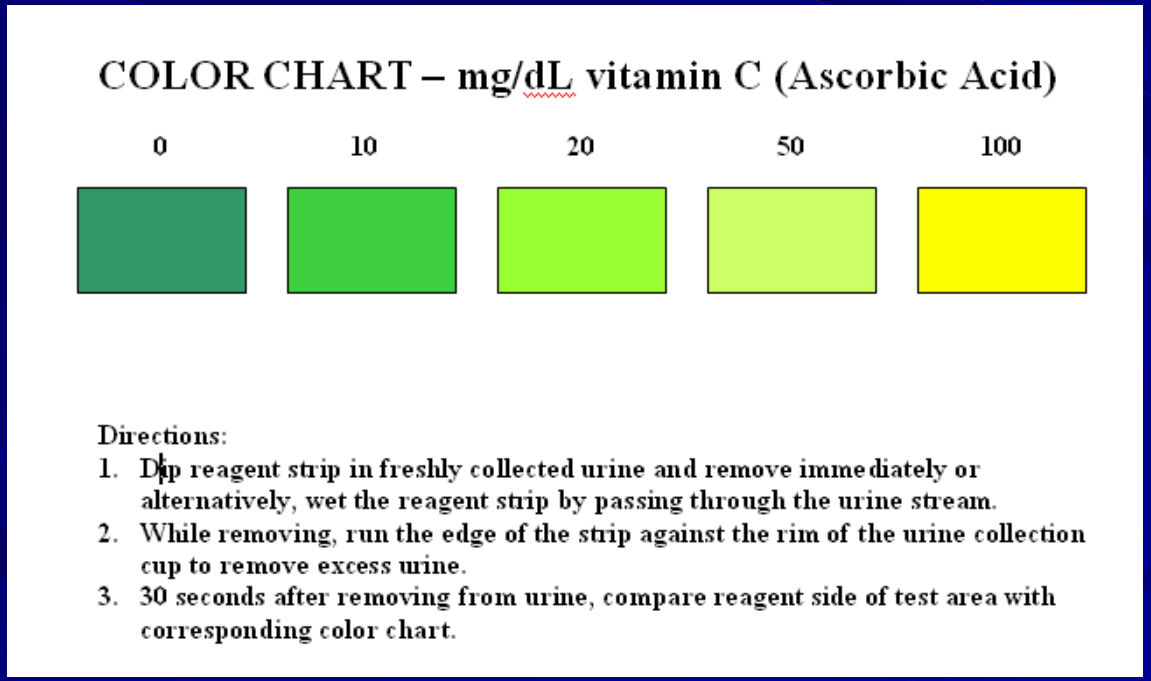
excretion of vitamin C.[9] However, certain medications such as aspirin, aminopyrine, barbiturates, hydantoins and paraldehyde as well as cold or heat stress are known to increase the excretion of vitamin C in the urine.[10,11]

When vitamin C stores are depleted, very little vitamin C appears in the urine after a test dose.[9] The U.S. RDA for vitamin C is 75 mg for females and 90 mg for males with an additional 35 mg if one smokes cigarettes.[12] It is important to remember that the RDA nutrient guide was designed to prevent deficiency diseases with a little nutrients to spare. It does not guarantee optimal or good health.

VitaChek-C fromTeco Diagnostics

Reagent Strips For Urinalysis
Ascorbic Acid (Vitamin C)

VitaChek-C Strips are designed for in-vitro measurement of urine Vitamin C and allow you to test Vitamin C levels multiple times a day from the privacy of your own home. Whether once a day or more, it is simple and easy to do.

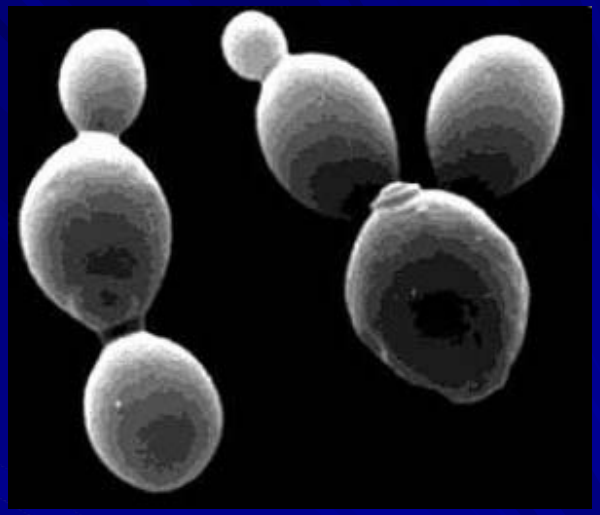


A BRIGHT SPOT on this urine stick test means you will have a brighter future!

Good News In Our DNA: Defects You Can Fix With Vitamins And Minerals

ScienceDaily (June 3, 2008)

ScienceDaily
Your source for the latest research news



Electron microscope image of budding yeast, Saccharomyces cerevisiae. UC Berkeley researchers insert variants of human enzymes into yeast to see if these enzymes can be tuned up with vitamins.

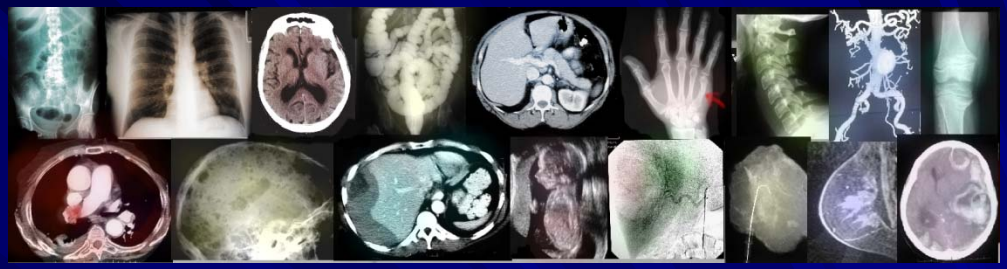
As the cost of sequencing a single human genome drops rapidly, with one company predicting a price of $100 per person in five years, soon the only reason not to look at your "personal genome" will be fear of what bad news lies in your genes.

University of California, Berkeley, scientists, however, have found a welcome reason to delve into your genetic heritage: to find the slight genetic flaws that can be fixed with remedies as simple as vitamin or mineral supplements.

"Our studies have convinced us that there is a lot of variation in the population in these enzymes, and a lot of it affects function, and a lot of it is responsive to vitamins," **Marini said.** ***"I wouldn't be surprised if everybody is going to require a different optimal dose of vitamins based on their genetic makeup, based upon the kind of variance they are harboring in vitamin-dependent enzymes."***

How to Turn on Telomerase Activity and Find the Fountain of Youth.

By Jeffrey Dach, MD



By now, it is should be obvious to you that activating telomerase, protects the telomeres from shortening and will slow or reverse the process of aging. On the contrary, knocking out or inhibiting telomerase activity results in shortened telomeres with acceleration of the aging process.

What Activates Telomerase ?

Among other things, the bioidentical hormones, 17 beta estradiol (estrogen) and testosterone activate telomerase. **The major mechanism for control and activation of telomorase is the hTERT promoter gene which stands for the human telomerase reverse transcriptase (hTERT) gene. When the hTERT gene is sequenced, and the code reviewed, it turns out there are two estrogen receptor elements in this gene. This explains why 17-beta estradiol activates telomerase.**

The Harvard study used Tamoxifen on genetically modified telomeres. **In the real world, tamoxifen is an estrogen blocker that occupies the cell receptors and turn OFF telomerase. Androgens were also found to turn on the hTERT gene and activate telomerase, and as expected, androgen blocker drugs inhibit telomerase.**

Bioidentical Hormones are the more logical choice…

http://www.wellsphere.com/genetics-article/bioidentical-hormones-reverse-aging-new-harvard-study-by-jeffrey-dach-md/1295172

Telomere dysfunction induces metabolic and mitochondrial compromise

nature International weekly journal of science

Nature 470, 359–365 (17 February 2011)
doi:10.1038/nature09787

Telomere dysfunction activates p53-mediated cellular growth arrest, senescence and apoptosis to drive progressive atrophy and functional decline in high-turnover tissues. The broader adverse impact of telomere dysfunction across many tissues including more quiescent systems prompted transcriptomic network analyses to identify common mechanisms operative in haematopoietic stem cells, heart and liver.

Consistent with PGCs as master regulators of mitochondrial physiology and metabolism, telomere dysfunction is associated with impaired mitochondrial biogenesis and function, decreased gluconeogenesis, cardiomyopathy, and increased reactive oxygen species.

In the setting of telomere dysfunction, enforced *Tert* or *PGC-1α* expression or germline deletion of *p53* (also known as *Trp53*) substantially restores PGC network expression, mitochondrial respiration, cardiac function and gluconeogenesis.

We demonstrate that telomere dysfunction activates p53 which in turn binds and represses *PGC-1α* and *PGC-1β* promoters, thereby forging a direct link between telomere and mitochondrial biology. We propose that this telomere–p53–PGC axis contributes to organ and metabolic failure and to diminishing organismal fitness in the setting of telomere dysfunction.

U.S. NEWS - NOVEMBER 28, 2010 — by Gautum Naik

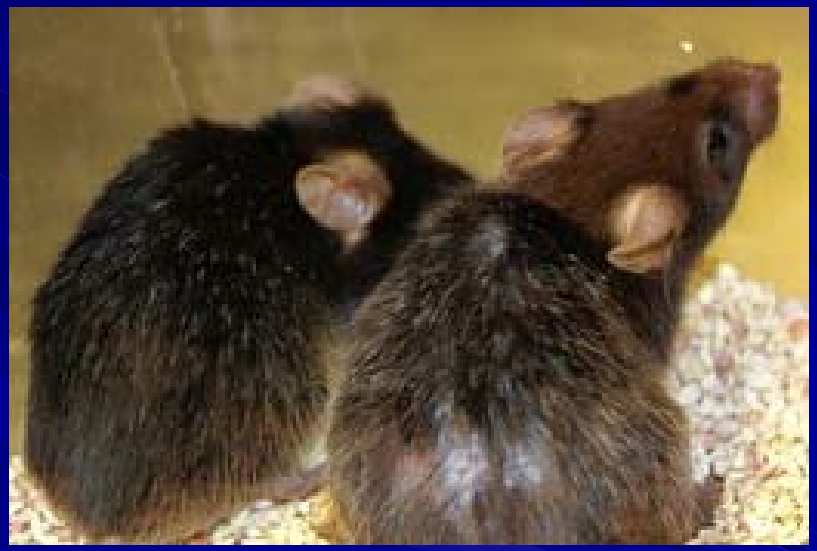
Aging Ills Reversed in Mice

Scientists Tweak a Gene and Rejuvenate Cells, Raising Hopes for Uses in Humans

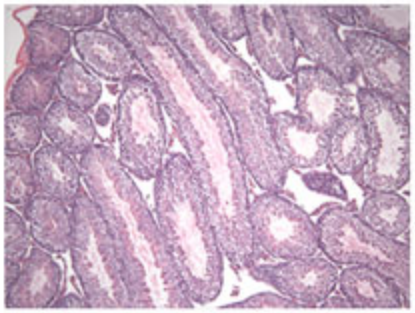
The research team led by Dr. Ronald DePinho of Dana Farber Cancer Institute made genetically engineered mice that aged prematurely.

The animals had short, dysfunctional telomeres and suffered a range of age-related problems, such as:

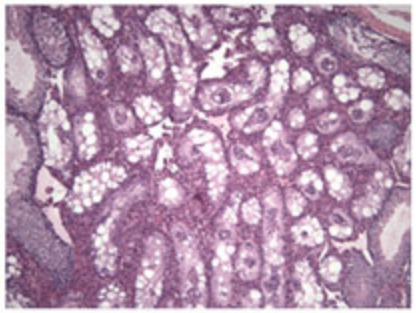
- atrophied spleens
- intestinal damage
- impaired sense of smell
- shrunken brains
- shrunken testes, depleted sperm count.
- Their telomeres had lengthened and the levels of telomerase increased, waking dormant brain stem cells, producing new neurons. The mice spleen, testes and brains were rejuvenated and grew in size.



Two mice involved in an experiment on age-related degeneration. Mouse on left, whose telomerase gene was activated, showed notable improvements.

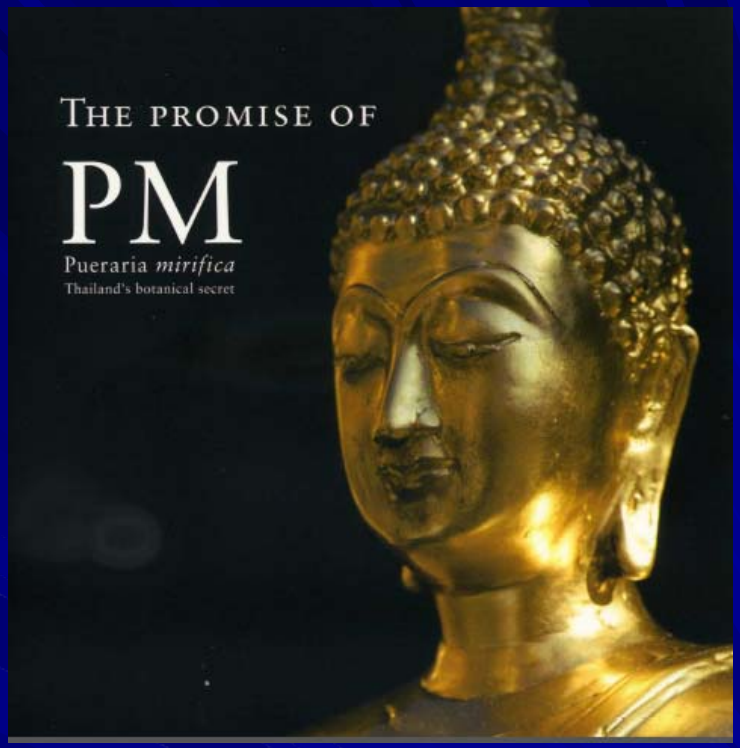


aged testicular tissue



telomerase activated

Pueraria mirifica (Thai Kudzu)



Studies have shown that this natural SERM, with Phase I, II and III, peer reviewed studies, shows benefit in HRT / menopause, with additional studies in…

- reducing CVD disease (cardioprotective)
- dyslipidemia
- remineralizes bone
- inhibits cancer growth
- restores vaginal integrity
- restores hair color
- builds collagen
- supports the prostate
- improves oxidative stress
- increases long and short-term memory
- extends telomeres
- reverses signs of aging,
- and has been shown to be completely safe below 10 mg / kg. body weight.

H.R.T. Plus (Herbal Remedy from Thailand)

The New Activated Herbal Remedy from Thailand (H.R.T.) containing Pueraria mirifica, a Bio-Identical PhytoEstrogen complex of PhytoEstrogen and Isoflavones.

Pueraria mirifica is an indigenous herb of Thailand, known as "Kwao Kreu" or "Kwao Kreu Kao" (White Kwao Kreu). It belongs to the Leguminosae, subfamily Papilionoideae, or the soy, bean & pea subfamily.

Active principles in this plant are found in the tuberous root, which looks like a chain of round-shaped bulbs of various sizes connected to the next one via small root throughout the entire length of the root. The shape and size of the tuberous root are diverse depending on the environment in which it exists.

Phytoestrogen-Puresterol (Pueraria mirifica) in the alleviation of climacteric symptoms

Author：Ta-Chin Lin [a], Tsung-Cheng Kuo [b]
a. Departments of Gynecology, Obstetrics and infertility, Kuo General Hospital
b. Superintendent, Kuo General Hospital, Tainan.

Abstract

Objective: Over the last several years, menopausal women have been seeking nonestrogen alternatives to hormone replacement in order to avoid the possible risks and side effects associated with conventional therapies. Most recently, women have increasingly looked to phytoestrogens to switch their menopausal therapy in a “natural” way. This clinical trial evaluates the estrogenic effects of the phytoestrogen-riched supplement Puresterol® in thirty females with climacteric symptoms.

Conclusions: From the clinical point of view, an oral dose of 80 mg of Pueraria Mirifica was found to be effective at alleviating climacteric symptoms. Due to the serious side effects associated with hormone replacement therapy, patients with climacteric symptoms currently prefer alternative phytoestrogen therapies to conventional menopausal management regimens. Our study illustrate that Pueraria Mirifica is a promising alternative for women suffering from menopausal symptoms.

Efficacy Comparison of Pueraria mirifica (PM) against Conjugated Equine Estrogen (CEE) with/without Medroxyprogesterone Acetate (MPA) in the Treatment of Climacteric Symptoms in Perimenopausal Women: Phase III Study

Verapol Chandeying MD*, Malinee Sangthawan MD**

J Med Assoc Thai 2007; 90 (9): 1720-6 *Full text. e-Journal: http://www.medassocthai.org/journal*

Perimenopausal women attending the Menopausal clinic of Hat Yai Regional Hospital were voluntarily recruited. The vasomotor symptoms such as hot flushes and night sweats, as well as other unpleasant symptoms, urogenital and psychological symptoms, were also assessed. Patients were voluntarily enrolled and randomly received daily 50 mg raw material of PM, Group A, or daily 0.625 mg of conjugated equine estrogen (CEE) with/without 2.5 mg of medroxyprogesterone acetate (MPA), Group B, depend on nonhysterectomized/ hysterectomized condition.

***Conclusion**: PM, containing phytoestrogens, has estrogenic effect as similar as CEE, and can alleviate the climacteric symptoms in perimenopausal women. PM demonstrates great promise in the treatment of climacteric symptoms. However, optimal doses should be clinically assessed to meet appropriate individual responses.*

But…doesn't Pueraria mirifica cause cancer?

Proceedings of the Nutrition Society (2009), **68** (OCE), E93 doi:10.1017/S0029665109990462

Scottish Section of The Nutrition Society, 7–8 April 2009

Phyto-oestrogens: do they have a role in breast cancer therapy?

S. Ramnarine[1], J. MacCallum[2] and M. Ritchie[2]

[1]*Edinburgh University, Edinburgh, UK and* [2]*Napier University, Edinburgh, UK*

Breast cancer is the most common malignancy in women[1]. Each year 44 335 women living in the UK are diagnosed with breast cancer[2]. Female sex hormones, such as oestrogen, are clearly implicated in breast cancer and conventional treatments include manipulation of the hormonal environment. There is both epidemiological[3,4] and experimental[5] evidence that plant oestrogens (phyto-oestrogens) in the diet may alter oestrogen metabolism, but the potential role of phyto-oestrogens in the management of breast cancer is unknown.

Ethnobotanical information has provided evidence of a potential anti-cancer effect of Kwai Krua root, *Pueraria Mirifica* (PM)[6,7], a herb commonly used in Thailand for enhancing female health. The herb contains a mixture of phyto-oestrogens including genistein (GEN), daidzein (DAI) and miroestrol. The aim of the present study was to evaluate the possible role of phyto-oestrogens, in particular those present in PM, in the management of breast cancer. The investigation was carried out using an *in vitro* model that included powdered extract of PM (concentrations ranging from 0.1 μg/ml to 1000 μg/ml); β-oestradiol (βE; concentrations 10^{-7}M–10^{-6}M); GEN (concentrations 10^{-8}M–10^{-4}M); DAI (concentrations 10^{-8}M–10^{-4}M); MCF-7 oestrogen receptor (ER)-positive breast cancer cell line (ATCC HTB-22). The effect of phyto-oestrogen exposure on cell growth over time was also monitored.

Tissue-culture techniques included: CyQuant™ assay (Invitrogen/Molecular Probes®), Paisley, UK); MTT and lactate dehydrogenase assays; immunostaining; flow cytometry; mRNA and protein extraction; RT–PCR; Western blotting; SDS gel electrophoresis. A powdered extract of Thai root PM was prepared at a stock concentration of 10 mg/ml in dimethyl sulfoxide and diluted in medium to various concentrations for use. βE, daidzein and GEN were prepared at stock concentrations in dimethyl sulfoxide and diluted as appropriate. MCF-7 ER-positive cells were maintained in culture for use.

Initial results indicated that ERα: ERβ mRNA expression appeared to change over time (0.5–24 h), suggesting a possible modulation of the receptors occurring at the level of the mRNA. This effect was particularly notable over the range of PM concentrations. GEN also produced similar effects on mRNA levels. Cell-growth studies over 4 d, as examined using the CyQuant™ assay, indicated low growth rates for the PM-treated cells (especially at 10 μg/ml) with an increasing effect over time that appeared to reach significance (n 4; ANOVA; $P = 0.088$) by day 3 or 4 when compared with untreated cells.

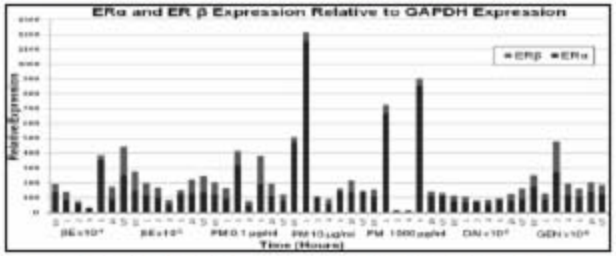


Fig. 1. For each treatment expression of ERα and ERβ mRNA is shown. Time points used were 0.5, 1, 2, 4, 6, and 24 h. Expression of ERα is higher for PM treatments (10 and 1000 μg/ml); PM at 0.1 μg/ml is in line with βE and GEN. Expression of ERβ is decreased for PM at 10 and 1000 μg/ml, and similar to βE and GEN for PM at 0.1 μg/ml. GAPDH, glyceraldehyde-3-phosphate dehydrogenase.

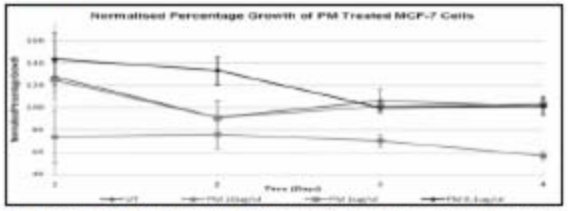
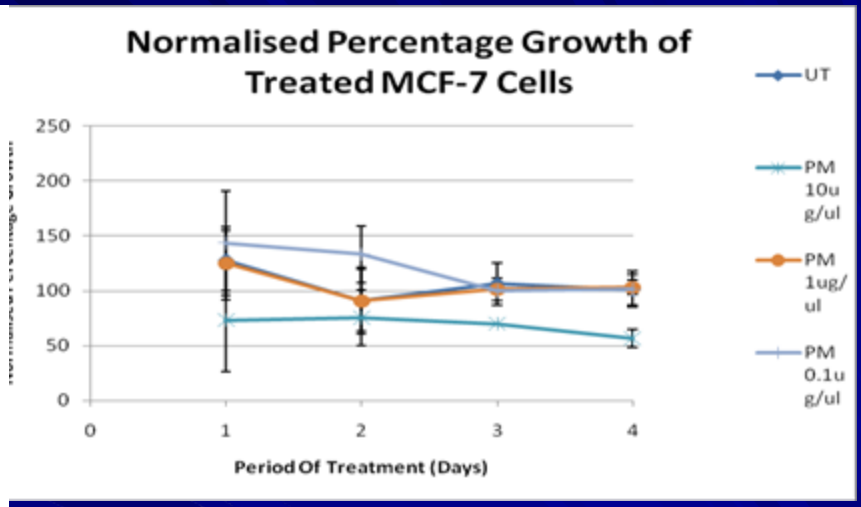


Fig. 2. Effects of PM on MCF-7 growth over 4 d. The maximum concentration used was 10 μg/ml, as higher concentrations were previously found to be directly toxic to the cells. Reduction in growth was significant ($P < 0.05$) for PM at 0.1 μg/ml over 4 d.



"These preliminary results are indicative of a potential anti-cancer action of PM that may be of use in the treatment of breast cancer. "

Inhibitory Potentials of Five Phytoestrogens from *Pueraria candollei* var. *mirifica* on CYP1A1 and CYP1A2 Proteins in Mouse Liver Microsomes and *in silico*

Waranya Chatuphonprasert[1,2], Kanokwan Jarukamjorn[1,*], Waraporn Putalun[1] and Thaweesak Juengwattanatrakul[3]

[1]*Research Group for Pharmaceutical Activities of Natural Products using Pharmaceutical Biotechnology (PANPB), Faculty of Pharmaceutical Sciences, National Research University-Khon Kaen University, Khon Kaen 40002, Thailand*

[2]*Faculty of Medicine, Mahasarakham University, Mahasarakham 44000, Thailand*

[3]*Faculty of Pharmaceutical Sciences, Ubon Ratchathani University, Ubon Ratchathani 34190, Thailand*

Abstract: *Pueraria candollei* var. *mirifica* (PM) is a Thai traditional medicinal plant for rejuvenation and estrogen replacement therapy in menopausal women. CYP1A1 and CYP1A2 proteins are the members of hepatic cytochrome P450 (CYP) enzymes to activate a procarcinogen, in which ethoxyresorufin *O*-demethylase (EROD) and methoxyresorufin *O*-demethylase (MROD) activities are the specific markers for CYP1A1 and CYP1A2, respectively. In the present study, the effects of five phytoestrogens isolated form the bark of PM tuberous roots namely miroestrol, deoxymiroestrol, kwakhurin, isomiroestrol, and methoxyisomiroestrol, on EROD and MROD activities were examined in mouse hepatic microsomes, compared to a typical CYP1A1/2 inducer and substrate beta-napthoflavone (BNF). The bindings of these five compounds to either CYP1A1 or CYP1A2 enzymes were analyzed using molecular docking with homology modeling technique. Rank of the median inhibitory concentration (IC_{50}) of these compounds on EROD activity corresponded to that of MROD, namely BNF > miroestrol > kwakhurin > deoxymiroestrol > methoxymiroestrol> isomiroestrol, respectively. Interestingly, the binding pose energy of these compounds to CYP1A1 and CYP1A2 proteins were consistent to those of inhibitory effects on EROD and MROD activities. The observations suggested for the first time that the active phytoestrogens from PM possessed inhibitory potentials on CYP1A1 and CYP1A2 *via* EROD and MROD activities, respectively. Furthermore, the binding energy of the compounds to CYP1A1 and CYP1A2 proteins might be a useful tool to predict the effects of a compound on these two CYP enzymes.

Keywords: *Pueraria candollei* var. *mirifica*, phytoestrogen, miroestrol, kwakhurin, CYP1A1, CYP1A2.

1. INTRODUCTION

Cytochrome P450 monooxygenase (CYP) is a supergene family of enzymes involved in the metabolism of numerous endogenous and exogenous compounds [1]. CYP plays important roles in the metabolism of many drugs and in the activation of several chemical toxicants and carcinogens in both humans and animals [2]. The subfamily 1A of CYP (CYP1A) consists of two enzymes: CYP1A1 and CYP1A2. CYP1A1 is not significantly expressed in the liver but constitutively expressed in several other extrahepatic tissues, whereas CYP1A2 is constitutively and inducibly expressed specifically in the liver [3]. CYP1A inactivates some chemical carcinogens and environmental contaminants by converting the substrates to more polar metabolites, resulting in increased excretion. In contrast, this metabolic activation may generate other potent carcinogens. For example, catalyzing the oxygenation of carcinogenic polycyclic aromatic hydrocarbons [4] which are found in combustion products [5], and the conversion of heterocyclic aromatic amines/amides to epoxide and other electrophilic reactive species (ultimate carcinogens) cause DNA or protein adducts, which lead to tumor formation and toxicity [6]. The activities of CYP1A1 and CYP1A2 are widely measured as a rate of the *O*-dealkylation of 7-ethoxyresorufin (ER) and 7-methoxyresorufin (MR) for EROD and MROD, respectively [7].

Pueraria candollei var. *mirifica* (PM) is a Thai traditional medicinal plant for rejuvenation and estrogen replacement therapy in menopausal women. The extensive researches have informed the pharmacological effects of this plant, such as stimulating effects on the luteinizing hormone (LH) and the follicle-stimulating hormone (FSH) in gonadectomized rats [8], inhibitory effect on ovulation in monkeys [9], and anti-oxidation properties in ovariectomized mice [10]. There are several phytoestrogens from tuberous roots of PM, *i.e.*, miroestrol, isomiroestrol, and deoxymiroestrol, and isoflavonoids, *i.e.*, puerarin, daidzin, daidzein, genistin, and genistein [11-12]. The crude extract of PM inhibited MROD activity in rats [13]. Moreover, miroestrol and

*Address correspondence to this author at the Faculty of Pharmaceutical Sciences, Khon Kaen University, Khon Kaen 40002, Thailand; Tel/Fax: +66-43-202305; E-mail: kanok_ja@kku.ac.th

"These observations suggested that the five phytoestrogens from PM might potentially decrease the risk of carcinogenesis due to inhibition of CYP1A oxidative metabolic activity pathway."

Possible effect of *Pueraria mirifica* on growth of primary culture of porcine endometrial cells and human endometrial cancer cells

Chatsri Deachapunya[a,*], Watchareewan Thongsaard[a], Piyanoot Tapaneeyaphan[a] and Sutthasinee Poonyachoti[b]
[a] Department of Physiology, Faculty of Medicine, Srinakharinwirot University,
[b] Department of Physiology, Faculty of Veterinary Medicine, Chulalongkorn University,

In conclusion, the water extract of *P. mirifica* had no effect on proliferation of normal endometrial epithelial cells and human endometrial cancer cell line except a slight inhibition of normal endometrial cell proliferation by high concentration of *P. mirifica*. The antiproferative effect of *P. mirifica* mediated via estrogen receptor requires further investigation. In contrast, 17β-estradiol increased the proliferation of both PE cells and Ishikawa cells in concentration-dependent manner. The presence of estrogen receptor and estrogen responsiveness of PE cells will serve as a non-pathogenic cell model used to screen

Vascul Pharmacol. 2008 Aug-Sep;49(2-3):106-10.

Puerarin reduces endothelial progenitor cells senescence through augmentation of telomerase activity.

Zhu J, Wang X, Shang Y, Xie X, Zhang F, Chen J, Fu G.
Department of Cardiology, Sir Run Run Shaw Hospital, College of Medicine, Zhejiang University, Hangzhou 310016, China.

Endothelial progenitor cells (EPCs) play an important role in both reendothelialization and neovascularization. Ex vivo expansion of EPCs might be useful for potential clinical cell therapy of ischemic diseases. However, ex vivo cultivation of EPCs leads to rapid onset of EPCs senescence, thereby severely limiting the proliferative capacity and clonal expansion potential. Therefore, we investigated whether puerarin might be able to prevent senescence of EPCs.

Puerarin dose dependently prevented the onset of EPCs senescence in culture. To get further insights into the underlying mechanisms of these effects induced by puerarin, we measured telomerase activity and determined the phosphorylation of serine/threonine protein kinase Akt by using western blot.

Puerarin significantly increased telomerase activity and phosphorylation of Akt, a downstream effector of phosphoinositide 3-kinase (PI-3K). Moreover, pretreatment with PI-3K blockers, either wortmannin or LY294002, significantly attenuated the puerarin puerarin-induced telomerase activity. Taken together, the results of the present study indicated that puerarin delayed the onset of EPCs senescence, which may be related to the activation of telomerase through the PI-3K/Akt pathway. The inhibition of EPCs senescence by puerarin in vitro may improve the functional activity of EPCs in a way that is important for potential cell therapy. PMID:18692596

Puerarin prevented senescence.

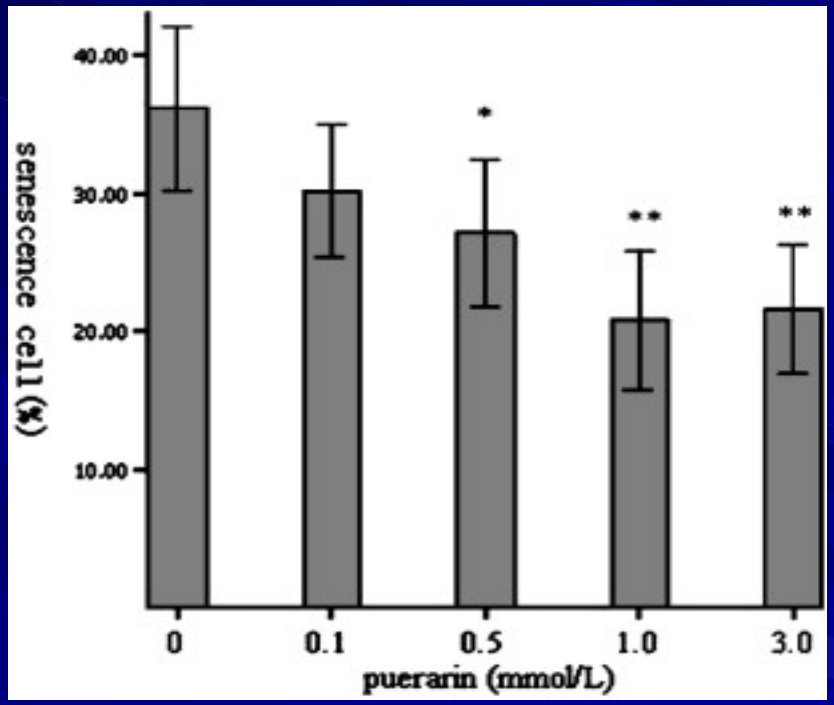


Fig. 1 Puerarin prevented EPCs' senescence. Freshly isolated mononuclear cells were cultivated in Medium 199 supplemented with 10% fetal-calf serum and VEGF. At day 4, cells were seeded in either indicated doses of puerarin in methylcellulose plates.

Puerarin induced telomerase in EPC

- Telomerase activities in EPC increases in proportional to the dose of puerarin.

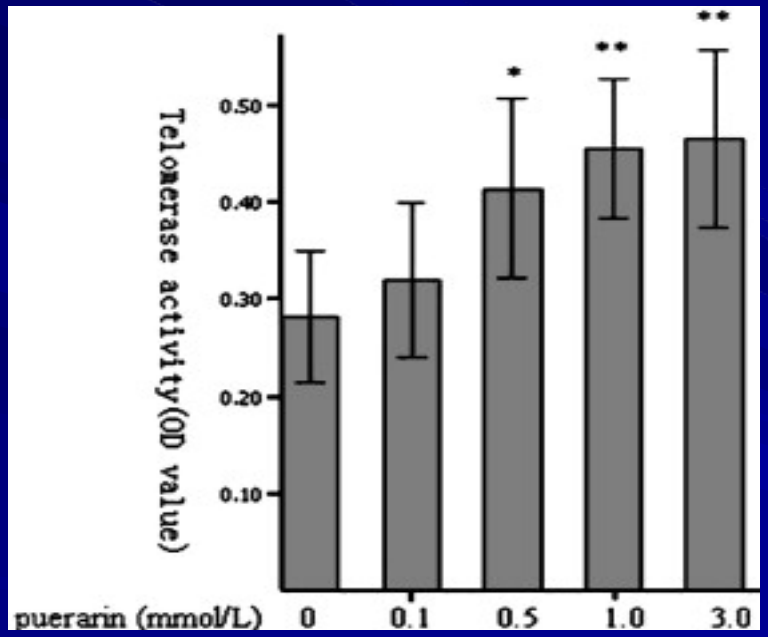


Fig. 4 Effects of puerarin on telomerase activity in EPCs. Freshly isolated mono-nuclear cells were cultivated in Medium 199 supplemented with 20% fetal-calf serum and VEGF. After 4 days of cultivation, EPCs were incubated with either indicated doses of puerarin for 24 h, and telomerase activity was measured by the TRAP assay.

Phytomedicine. Volume 15, Issues 1–2, 25 January 2008, Pages 38–43

Correlation of antioxidant activity and major isoflavonoid contents of the phytoestrogen-rich *Pueraria mirifica* and *Pueraria lobata* tubers

W. Cherdshewasart, W. Sutjit. Faculty of Science, Chulalongkorn University, Phyathai Road, Patumwan, Bangkok 10330, Thailand

Abstract

The antioxidant activity of wild *Pueraria mirifica* collected from 28 of the 76 provinces of Thailand and *Pueraria lobata* collected from China were assessed by 1,1-diphenyl-2-picrylhydrazyl (DPPH) assay.

***P. mirifica* tuberous extracts showed antioxidant activity as with *α*-tocopherol. Six plant samples exhibited stronger antioxidant activity than the mean value of the *P. mirifica* population. In addition, the mean value of the *P. mirifica* population indicated significantly lower antioxidant activity than *P. lobata*. The analysis of the antioxidant activity of isoflavonoids revealed that puerarin and daidzein exhibited the same level of antioxidant activity as *α*-tocopherol.**

The results showed convincingly that puerarin and daidzein in the plant tubers may play an important role in antioxidant activity. The correlation analysis between antioxidant activity and major isoflavonoid contents of plant tubers indicated a significant correlation only with puerarin and a significant lack of correlation with daidzin, daidzein and genistein.

Journal of Medicinal Plants Research
Vol. 5(31), pp. 6832-6838
23 December, 2011

PubMed.gov
U.S. National Library of Medicine
National Institutes of Health

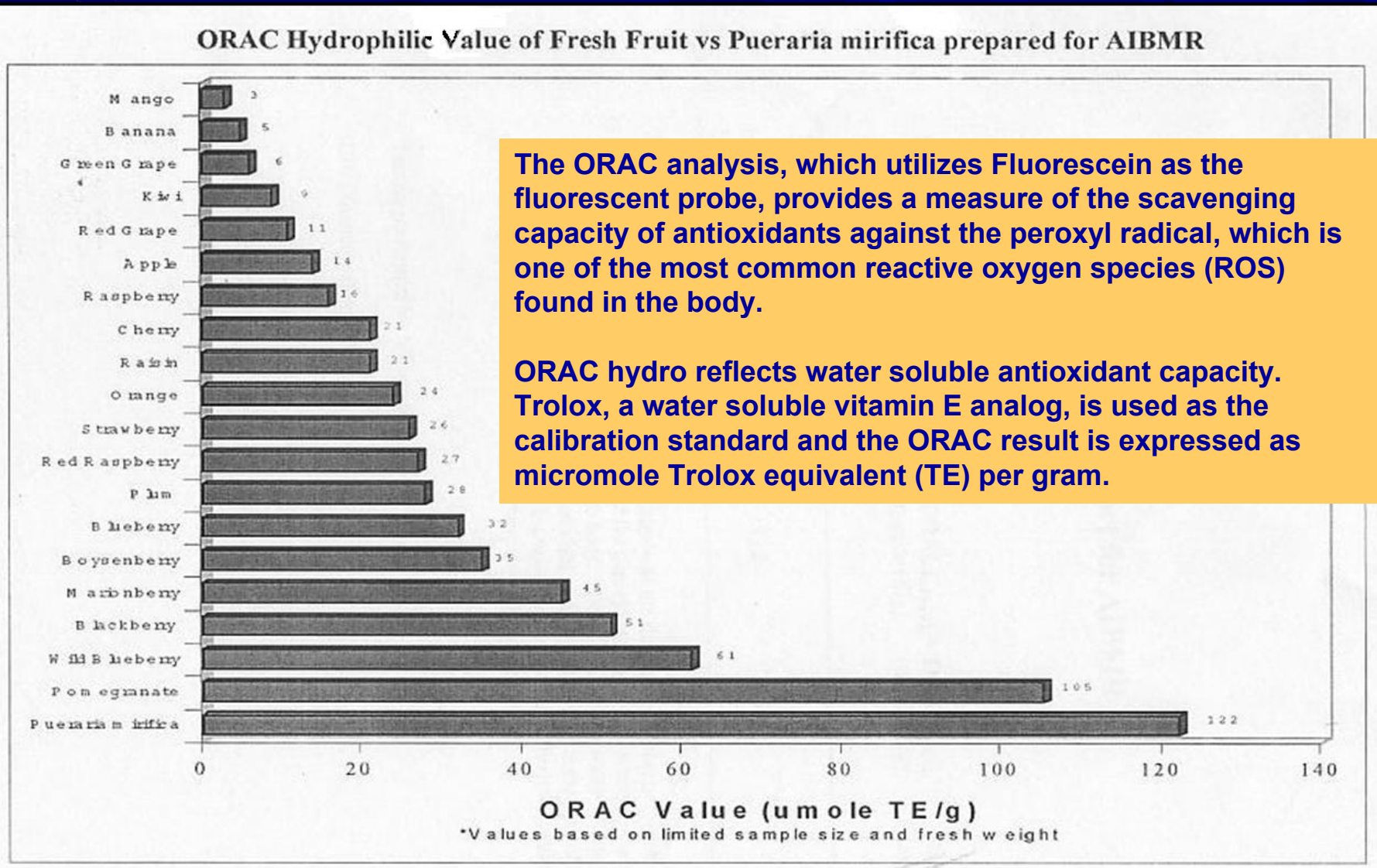
Estrogen-like activities and cytotoxicity effects of Thai herbal medicines as natural ingredients in anti-ageing

Yingham B, et al.

The objective of the study was to search for the appropriate herbal extracts by comparative analysis of their estrogenic and cytotoxic activities. Some potentially estrogenic activity of herbal extracts in the management of female disorder symptoms was investigated by E-screen assay.

The extract of *P. candollei* var *mirifica* exerted the strongest estrogenicity and gave the highest level in growth promoting activity. It significantly stimulated cell proliferation at concentrations of 0.1-50 µ/ml ($p<0.05$) whereas higher concentration (100 µg/ml) suppressed the growth of such cells. The maximal proliferative effect of this extract was achieved at 50 µg/ml which is higher than the effect displayed by 0.1 nM E2.

Results indicated that the phytoestogenic substances in the extracts exerted their estrogenic activities through estrogen receptor pathways.



The ORAC analysis, which utilizes Fluorescein as the fluorescent probe, provides a measure of the scavenging capacity of antioxidants against the peroxyl radical, which is one of the most common reactive oxygen species (ROS) found in the body.

ORAC hydro reflects water soluble antioxidant capacity. Trolox, a water soluble vitamin E analog, is used as the calibration standard and the ORAC result is expressed as micromole Trolox equivalent (TE) per gram.

Toxins, Telomeres and Cellular Aging

A typical cell can only accurately replicate itself approximately 50-times. Each time a cell divides, the DNA's end-cap, or telomere, gets slightly smaller. Eventually, the telomeres become so short that the chromosomes are damaged in the process and the cell can no longer divide. However, if the enzymes telomerase and polymerase are present in adequate quantities, telomeres are protected and can even grow longer!

Free radicals are unstable oxygen molecules created during the course of normal cellular function. If enough free radicals exist, they can damage other components, including DNA and proteins, causing cellular stress. Antioxidants are substances found in plants that absorb unstable oxygen molecules. If the body has adequate antioxidants present, it can minimize free radical damage.

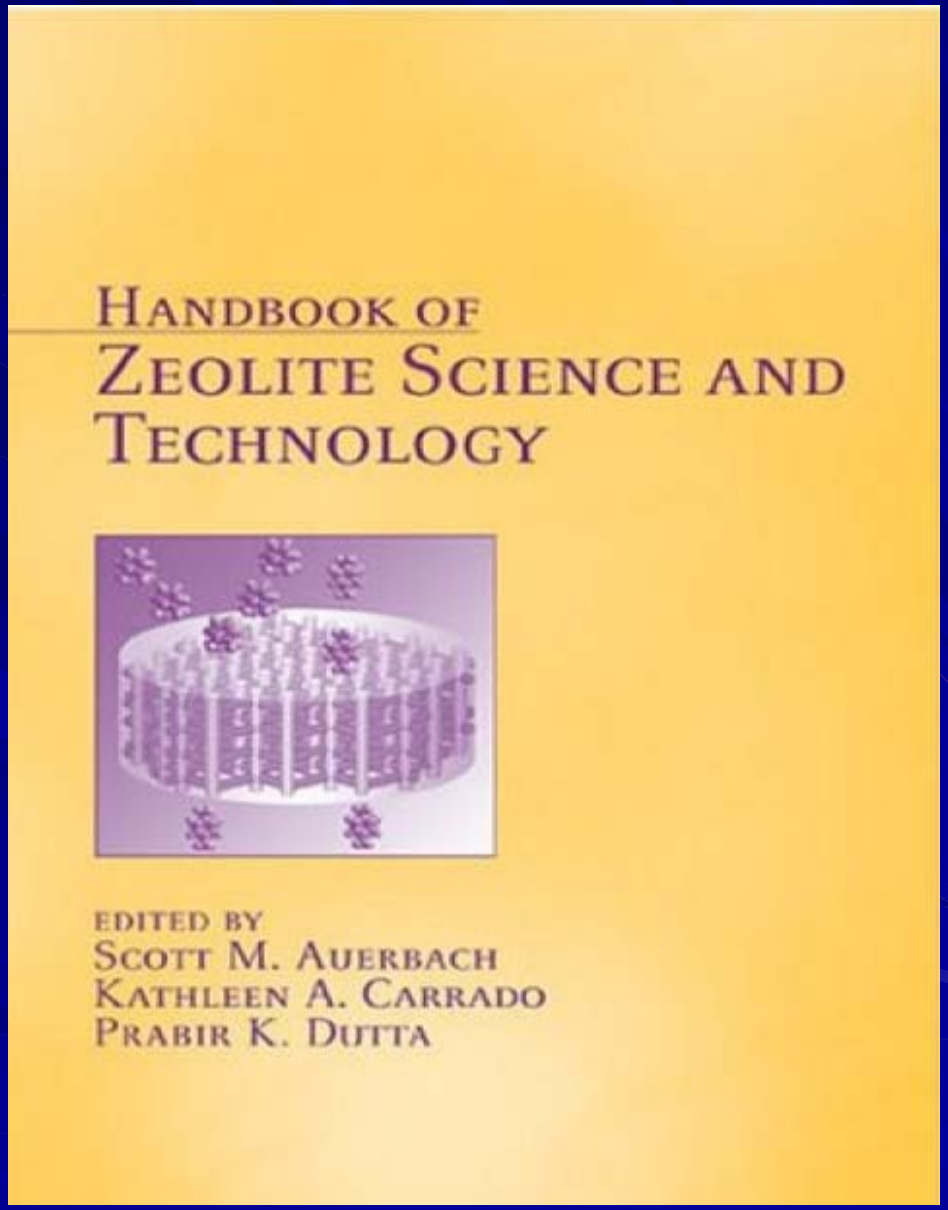
Toxins from chemicals, environment pollutants, processed foods and other sources all take their toll on the body. If these toxins are allowed to accumulate in the body, eventually cellular and organ functions suffer.

However, if cells are continually detoxed and fed proper nutrients, there is evidence that they may live indefinitely.

24 – Medical Applications of Zeolite

Kresimir Pavelic and Mirko Hadzija
Ruder Boskovic Institute, Zagreb, Croatia

Zeolites are among the most important inorganic cation exchangers. The aluminosilicate structure is negatively charged and attracts cations that come to reside inside the pores and channels. Zeolites have large empty spaces, or cages, within their structures that can accommodate large cations, such as Na+, K+, Br+, and Ca+, and even relatively large molecules and cationic groups, such as water, ammonia, carbonate ions, and nitrate ions. The basic structure of zeolites is biologically neutral (pg 1141).



Doyle *et al.*: ISOFLURANE SCAVENGING

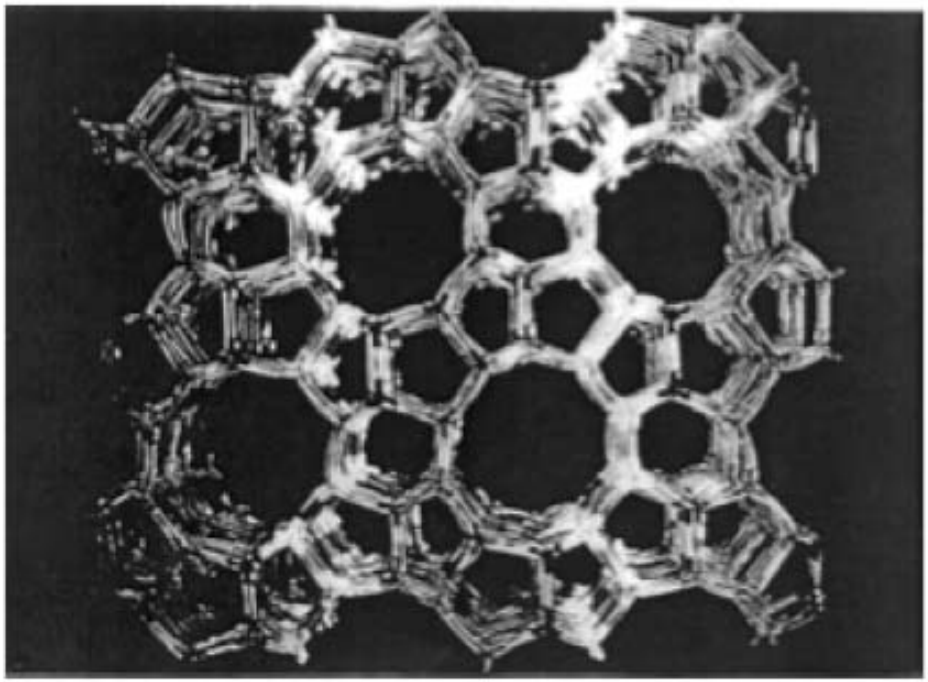


FIGURE 1 A repeated unit cell of the crystal structure of hydrophobic silica zeolite (MHZ™) molecular sieve; anesthetic agent is selectively captured in the honeycomb-shaped silica zeolite crystals. In just 500 g of silica zeolite, the total internal surface area available for capturing anesthetic would cover 70 football fields.

Size Matters…

100 g of zeolite internal surface is equal to 14 football fields.

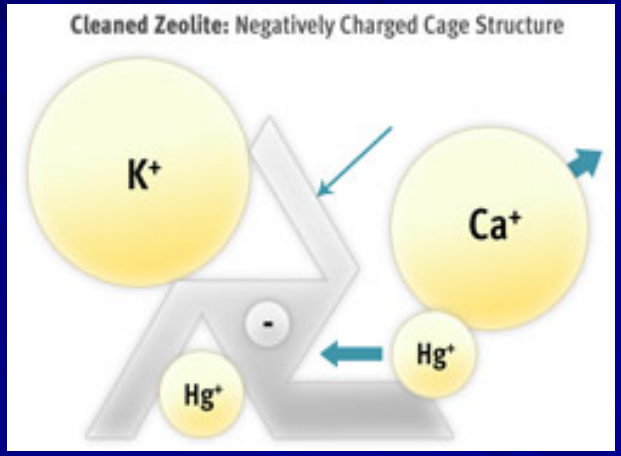
The internal surface area of the Micronized Hydro-Colloidal Zeolite crystal structure of only 7.15 g would cover the surface area of an entire 100 yard football field.

1 g of zeolite internal surface is equal to 14 yards of a football field.

100 mg (one ZeoGold capsule) = 1.4 yards of one football field.

Breakthrough in Heavy Metal Chelation, Zeolite Safely Removes Mercury, Lead and More.

One distinct advantage of Zeolite over many chelation methodologies is its highly selective attraction for toxic heavy metals with far less attraction for vital minerals like calcium, potassium and selenium. Zeolite actually prefers mercury and lead.



Smaller ions such as Mercury and Lead are pulled deeply into the cage structure of the zeolite and held securely for safe elimination.

Another advantage is that unlike acid-based chelating agents, Zeolite molecules irreversibly bind to the toxic element in three key ways, with the strength of the bond based upon:

- The toxin's charge density
- The toxin's average molecular size
- A phenomenon known as "molecular adaptive fit".

Zeolite Safely Removes: Mercury, Lead, Aluminum, Antimony, Arsenic, Barium, Bismuth, Cadmium, Cesium, Gadolinium, Gallium, Nickel, Niobium, Platinum, Rubidium, Thallium, Thorium, Tin, Tungsten, Uranium and more.

Acid-based chelators such as EDTA, DMSA and DMPS do not preferentially bind, nor irreversibly bind toxic heavy metals. The following selectivity scale of clinoptilolite zeolite for various heavy metal ions is backed by atomic absorption spectroscopy studies. As you can see, heavy metals are highest in preference of attraction.

Zeolite Selectivity Series

IV. Removal Of Heavy Metals and Organopoisoning

Heavy metals released in wastewater are among the most worrisome pollution problems due to their cumulative effects along the food chain. The natural zeolites clinoptilolite, phillipsite, and chabazite are particularly useful in selectively eliminating ammonia and heavy metals such as Cd^{2+}, Pb^{2+}, Zn^{2+}, Cu^{2+}, and particularly Cr^{3+}. Generally, clinoptilolite is stable in an acidic environment and shows high selectivity for many heavy metals.

V. Antimicrobial Effects

Tissue conditioners containing silver-exchanged zeolite showed a strong in-vitro antimicrobial effect on *Candida albicans*, and also on nasocomial respiratory infections of *S. aureus* and *P. aeruginosa*. All microbes were killed whether they have been immersed in saliva or not.

A new type of antibacterial temporary filling material in dentistry was incorporated into urethane acrylate monomer paste. These materials exhibited prominent in-vitro antibacterial activity against *Streptococcus mutans* and *Streptococcus mitis*.

XI. Effects on Diabetes Mellitus

Zeolites are of potential use in the treatment of diabetes. Our unpublished data concerning alloxan-induced diabetic mice showed that natural clinoptilolite could prevent of diminish some late complications of diabetes, namely, development of polyneuropathies. There is some indication that natural zeolite may sorb small amounts of glucose, and the hydrothermal transformation of natural, purified clinoptilolite using $FeSO_4$ has been shown to cause selectivity for glucose adsorption.

Clinoptilolite showed positive effects on many diabetic symptoms. Significant differences between zeolite-treated and nontreated diabetic mice were noticed only in the amount of total Ca in sera. Nontreated diabetic animals had 1.92 mM/L Ca in sera, wheras clinoptilolite-treated diabetic mice had a higher concentration of Ca in sera, ranging from 2.15 to 2.3 mM/L. Iron (Fe2+) containing, natural clinoptilolite interacts with glucose with formation of an iron-glucose complex in the clinoptilolite.

Biomedical Effects of Zeolite (Med Apps continued from pg 1141)

Zeolites have known biological properties alone with long-term chemical and biological stability; they reversibly bind small molecules such as oxygen and nitric oxide and have immunomodulatory activity.

It is known that environmental DNA can be stabilized by adsorption onto sand and clay particles, thereby becoming 100- to 1000-fold more resistant to deoxyribonuclease (DNase).

Such adsorbed DNA may retain its transforming ability for weeks and even months.

Since many biochemical processes are closely related to some zeolite properties (ion-exchange, adsorption, and catalysis), we believe that natural and synthetic zeolites may lead to significant advances in biology, medicine, and in the pharmaceutical industry in the near future.

Life Sci. 2013 May 20;92(17-19):903-10.

Dietary zeolite supplementation reduces oxidative damage and plaque generation in the brain of an Alzheimer's disease mouse model

Montinaro M, Uberti D, Maccarinelli G, Bonini SA, Ferrari-Toninelli G, Memo M.
Department of Biomedical Sciences and Biotechnologies, University of Brescia, 25123 Brescia, Italy.
mery.montinaro@med.unibs.it

Abstract

Oxidative stress is considered one of the main events that lead to aging and neuro-degeneration. Antioxidant treatments used to counteract oxidative damage have been associated with a wide variety of side effects or at the utmost to be ineffective. The aim of the present study was to investigate the antioxidant property of a natural mineral, the tribomechanically micronized zeolite (MZ).

The study showed that 24h of cell pretreatment with MZ (1) protected the cells by radical oxygen species (ROS)-induced cell death and moreover (2) induced a reduction of the mitochondrial ROS production following a pro-oxidant stimulation. Looking for an antioxidant effect of MZ in vivo, we found (3) an increased activity of the endogenous antioxidant enzyme superoxide dismutase (SOD) in the hippocampus of tg mice and (4) a reduction in amyloid levels and plaque load in MZ treated tg mice compared to control tg mice.

Our results suggest MZ as a novel potential adjuvant in counteracting oxidative stress and plaque accumulation in the field of neurodegenerative diseases.

http://www.ncbi.nlm.nih.gov/pubmed/23562853

J Clin Biochem Nutr. 2012 May;50(3):195-8. Epub 2011 Nov 29.

Natural zeolites *chabazite/phillipsite/analcime* increase blood levels of antioxidant enzymes.

Dogliotti G, Malavazos AE, Giacometti S, Solimene U, Fanelli M, Corsi MM, Dozio E.
Dipartimento di Morfologia Umana a Scienze Biomediche "Città Studi", University of Milan, Via Mangiagalli 31, 20133 Milan, Italy.

Abstract

Imbalance between reactive oxygen species generation and antioxidant capacity induces a condition known as oxidative stress which is implicated in numerous pathological processes. In this study we evaluated whether natural zeolites (chabazite/phillipsite/analcime) may affect the levels of different antioxidant enzymes (gluthatione peroxidase, superoxide dismutase, gluthatione reductase), total antioxidant status and oxidative stress in 25 clinically healthy men, both non-smokers and smokers. Measurements were performed on whole blood or on plasma samples before (T0) and after 4-weeks zeolites intake (T1).
At T1, gluthatione peroxidase, superoxide dismutase and gluthatione reductase increased compared to T0 levels, both considering all subjects as joint and after subdivision in non-smokers and smokers. Differently, a reduction in total antioxidant status was observed at T1. Anyway, total antioxidant status resulted higher than the reference values in both groups at each time point. A decrease in lipid peroxidation, a major indicator of oxidative stress assessed by monitoring thiobarbituric acid reactive substances, was observed in all subjects at T1. Our results suggested that natural zeolites may help to counteract oxidative stress in apparently healthy subjects exposed to different oxidative stress risk factors, such as smoking, thus representing a particular kind of food with potential antioxidant properties.

Anticancer Res. 2003 Mar-Apr;23(2B):1589-95.

Anticancer and antioxidative effects of micronized zeolite clinoptilolite.

Zarkovic N, Zarkovic K, Kralj M, Borovic S, Sabolovic S, Blazi MP, Cipak A, Pavelic K.
Ruder Boskovic Institute, Division of Molecular Medicine, Bijenicka 54, HR-10000 Zagreb, Croatia.

ABSTRACT

Treatment of cancer-bearing mice and dogs with micronized zeolite clinoptilolite (MZ) led to improvement of the overall health status, prolongation of life span and decrease of tumor size in some cases. It also reduced lipid peroxidation in the liver of mice.

MATERIALS AND METHODS:

The experiments were performed on various tumor cell cultures and tumor-bearing animals. Immunohistochemistry was used to analyze if MZ could interfere with Doxorubicin-induced lipid peroxidation and consequential production of 4-hydroxynonenal (HNE).

RESULTS:

MZ reduced the metabolic rate of cancer cells and increased binding of HNE to albumin in vitro. It selectively reduced generation of HNE in vivo in tumor stroma after Doxorubicin treatment leaving onset of lipid peroxidation intact in malignant cells. Combined treatment with Doxorubicin and MZ resulted in strong reduction of the pulmonary metastasis count increasing anticancer effects of Doxorubicin.

CONCLUSION:

Interference of MZ with lipid peroxidation might explain some of the beneficial effects of this particular zeolite in combined cancer therapy.

J Mol Med (Berl). 2001;78(12):708-20.

Natural zeolite clinoptilolite: new adjuvant in anticancer therapy.

Pavelić K, Hadzija M, Bedrica L, Pavelić J, Dikić I, Katić M, Kralj M, Bosnar MH, Kapitanović S, Poljak-Blazi M, Krizanac S, Stojković R, Jurin M, Subotić B, Colić M.
Ruder Bosković Institute, Division of Molecular Medicine, Zagreb, Croatia.

Abstract

Natural silicate materials, including zeolite clinoptilolite, have been shown to exhibit diverse biological activities. We report a novel use of finely ground clinoptilolite as a potential adjuvant in anticancer therapy.

Clinoptilolite treatment of mice and dogs suffering from a variety of tumor types led to improvement in the overall health status, prolongation of life-span, and decrease in tumors size. Local application of clinoptilolite to skin cancers of dogs effectively reduced tumor formation and growth.

In addition, toxicology studies on mice and rats demonstrated that the treatment does not have negative effects. In vitro tissue culture studies showed that finely ground clinoptilolite inhibits protein kinase B (c-Akt), induces expression of p21WAF1/CIP1 and p27KIP1 tumor suppressor proteins, and blocks cell growth in several cancer cell lines.

These data indicate that clinoptilolite treatment might affect cancer growth by attenuating survival signals and inducing tumor suppressor genes in treated cells.

PMID: 11434724 [PubMed - indexed for MEDLINE]

ZeoGold™ Has Superior DETOX Capacity and Performance

Generally, ZeoGold™ powder has superior DETOX capacity and performance for inorganic metallics vs. other zeolite DETOX products, because of the higher CEC capacity, ultrahigh surface area available for sorption and optimized particle size. The natural zeolites remove Pb or other metal cations present in water solutions and biological, aqueous milieu via:

a) ***exchange for ions (e.g., Na, K, Ca, H+) in the zeolite, crystallites for the Pb or other metal cation.***

b) ***by direct, surface sorption.***

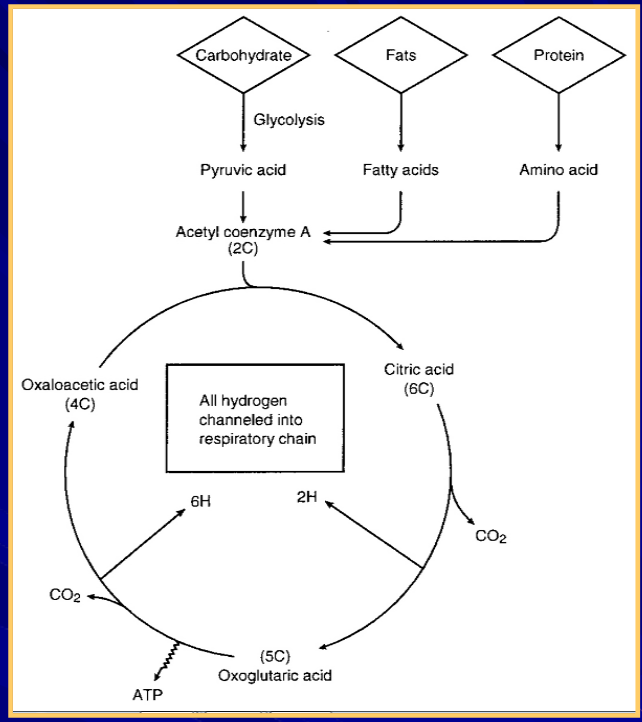
c) ***by physically, removing particulate forms of Pb or trace metals that get "trapped" in the zeolite, micro-crystals or pore structures.***

d) ***indirectly, by altering the intestinal tract microflora and/or bio-film layer that can alter the utilization or processing of trace metals.***

The mechanism for removal of Pb and other toxic, trace metal cations for ZeoGold™ is the same as for Clinoptilolite products, but superior DETOX performance can be expected from the ZeoGold™ doses (100 to 250 mg/day) than the Clinoptilolite products.

WHAT'S HYDROGEN GOT TO DO WITH IT?

Albert Szent-Gyorgyi, the Hungarian Nobel Prize winning biochemist who discovered Vitamin C, said that hydrogenrather than oxygen, is the fuel of life.

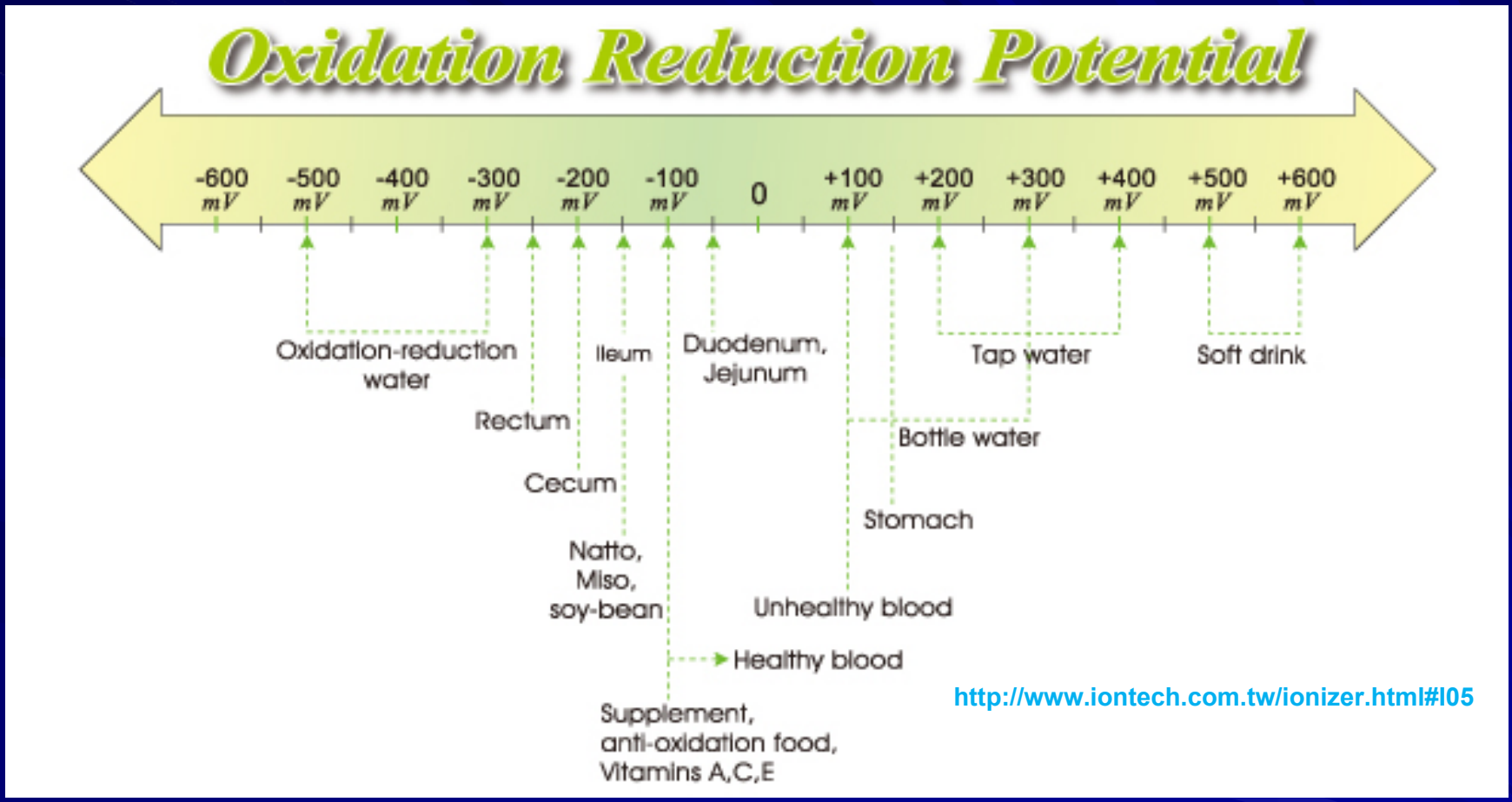


Hydrogen is the body's most needed nutrient, our Primordial ANTI-OXIDANT!

Everyone is deficient in H-. A machine called the BTA or Biological Terrain Analyzer developed by a Dr. Morrell which tests blood, saliva and urine for H+, H- and minerals found 100% of people low in H-, especially as they got older. They were all over oxidized. The absence of electrons causes numerous diseases.

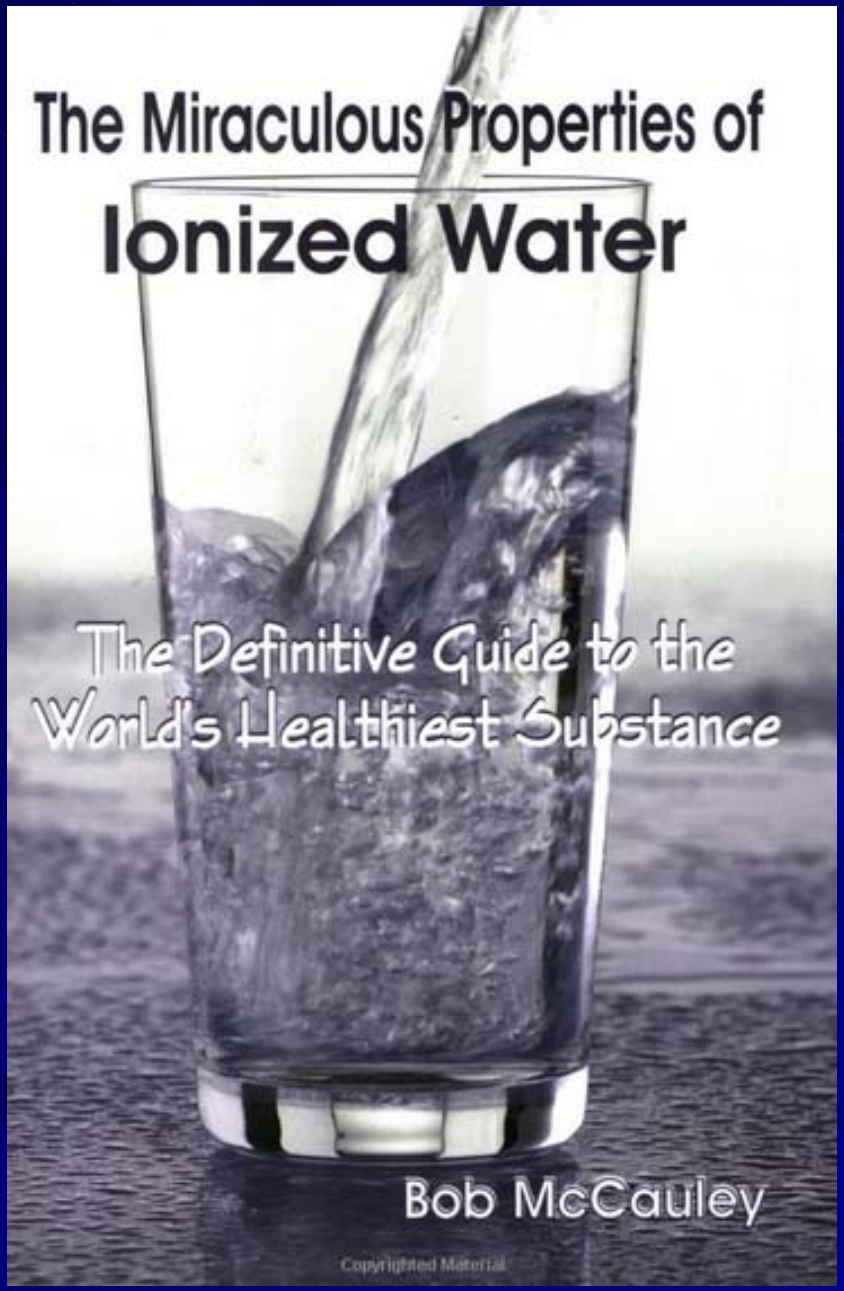
Electrons don't move in the body unless they are associated with hydrogen. A body in good health has abundant H- ionised molecules.

When you hydrate the cells they plump and become healthy and the body goes into an anabolic state - when the cells become dehydrated, the body goes into a catalytic state and eats its own muscles.



ORP (Oxidation Reduction Potential or Redox Potential)

ORP is a measure of the presence of oxidizing or reducing agents in a solution. The oxidation scale can go from about -1000 to +1000. Sources with a strong negative ORP are safer to consume.



Ionized Water

Water is our best defense against disease of every kind. Sixty percent or more of all chronic disease would be significantly reduced if people would simply keep themselves properly hydrated and alkalinized.

Alkaline Ionized Water has two antioxidant qualities, its negative charge and the presence of hydroxyl ions which are free radical scavengers. The body is starved for electrons and Alkaline Ionized Water contains an abundance of them, which nullify free radicals in the body.

All disease thrives in an acid environment in the body and will not flourish and thrive in an alkaline environment.

Drinking Alkaline Ionized Water gives you energy through better hydration and alkalization of the body and by providing the body with oxygen

Journal of Health Science, **50**(5) 456–465 (2004)

Studies on the Properties and Real Existence of Aqueous Solution Systems that are Assumed to Have Antioxidant Activities by the Action of "Active Hydrogen"

Atsushi Hiraoka,*,[a] Masumi Takemoto,[a] Takahiro Suzuki,[a] Atsuko Shinohara,[b] Momoko Chiba,[b] Mika Shirao,[c] and Yoshihiro Yoshimura[d]

[a]Department of Pathological Biochemistry, Kyorin University School of Health Sciences, 476 Miyashita-cho, Hachioji-shi, Tokyo 192–8508, Japan, [b]Department of Epidemiology and Environmental Health, Juntendo University School of Medicine, 2–1–1 Hongo, Bunkyo-ku, Tokyo 113–8421, Japan, [c]Department of Human Nutrition, Jissen Woemen's College, 1–13–1 Shinmei, Hino-shi, Tokyo 191–0016, Japan, and [d]Department of Analytical Chemistry, Faculty of Pharmaceutical Science, Hoshi University, 2–4–41 Ebara, Shinagawa-ku, Tokyo 142–8501, Japan

(Received March 2, 2004; Accepted June 9, 2004)

We evaluated the properties and real existence of an electrolyzed-reduced water, which we prepared, and three commercially purchased water goods, that are advertised to have antioxidant activities by the action of "active hydrogen," on the basis of the results of examinations for inhibitory effects on the oxidative reactions of biomolecules, quantitative analyses of the minerals, and the ESR spectral data in measurement of the scavenging ability for reactive oxygen species. The results suggested that all of the examined aqueous solution systems undoubtedly have antioxidant activities *in vitro* and that such effects are derived from ordinary molecular hydrogen (hydrogen gas) and/or (a) reductive vanadium ion(s). "Active hydrogen" seems to be absent as an effective component of the antioxidant activities of these aqueous solution systems.

Key words —— reduced water, antioxidant activity, oxygen-radical scavenger, ESR spectrometry, hydrogen, vanadium

nature medicine

Hydrogen acts as a therapeutic antioxidant by selectively reducing cytotoxic oxygen radicals

Ikuroh Ohsawa[1], Masahiro Ishikawa[1], Kumiko Takahashi[1], Megumi Watanabe[1,2], Kiyomi Nishimaki[1], Kumi Yamagata[1], Ken-ichiro Katsura[2], Yasuo Katayama[2], Sadamitsu Asoh[1] & Shigeo Ohta[1]

Acute oxidative stress induced by ischemia-reperfusion or inflammation causes serious damage to tissues, and persistent oxidative stress is accepted as one of the causes of many common diseases including cancer. We show here that hydrogen (H_2) has potential as an antioxidant in preventive and therapeutic applications. We induced acute oxidative stress in cultured cells by three independent methods. H_2 selectively reduced the hydroxyl radical, the most cytotoxic of reactive oxygen species (ROS), and effectively protected cells; however, H_2 did not react with other ROS, which possess physiological roles. We used an acute rat model in which oxidative stress damage was induced in the brain by focal ischemia and reperfusion. The inhalation of H_2 gas markedly suppressed brain injury by buffering the effects of oxidative stress. Thus H_2 can be used as an effective antioxidant therapy; owing to its ability to rapidly diffuse across membranes, it can reach and react with cytotoxic ROS and thus protect against oxidative damage.

Oxidative stress arises from the strong cellular oxidizing potential of excess reactive oxygen species (ROS), or free radicals[1–5]. Most of the superoxide anion radical ($O_2^{\bullet-}$) produced is generated in mitochondria by electron leakage from the electron transport chain and the Krebs cycle[6]. $O_2^{\bullet-}$ is also produced by metabolic oxidases, including NADPH oxidase and xanthine oxidase[7]. Superoxide dismutase converts $O_2^{\bullet-}$ into hydrogen peroxide (H_2O_2)[8], which is detoxified into H_2O by

RESULTS

H_2 selectively reduces •OH in cultured cells

H_2 reduces the •OH that is produced by radiolysis or photolysis of water[12]; however, whether H_2 can effectively neutralize •OH in living cells has not been directly investigated. As the cellular damage produced by spontaneous generation of •OH is not sufficient to be

The hydrogen highway to reperfusion therapy

Katherine C Wood & Mark T Gladwin

Hydrogen gas debuts as a selective antioxidant with explosive potential as cytoprotective therapy for ischemia-reperfusion injury and stroke.

Just when we thought we had exhausted our tool kit of therapeutic gases, Ohsawa *et al.*[1] provide evidence that inhaled hydrogen gas (H_2) has antioxidant and antiapoptotic activities that protect the brain against ischemia-reperfusion injury and stroke[1].

During the ischemic phase of thromboembolic stroke, a blood clot travels to and lodges in the distal blood vessels in the brain, blocking blood flow to the oxygen-starved tissue for a period of hours. This is followed by the reperfusion phase, when the blood clot is broken down by natural or pharmacological means and blood flow is restored. Although restoration of blood flow is critical, the reintroduction of molecular oxygen triggers a cytotoxic cascade during which reactive oxygen species are generated by the mitochondria. This burst of reactive oxygen species irrevocably drives downstream signaling networks that lead to cellular necrosis and apoptosis. For both stroke and myocardial infarction, there are now highly successful approaches to restore blood flow to the ischemic tissue. So far, however, we have completely failed to relieve this pathological cascade of oxidative damage after reperfusion injury. In this issue, Ohsawa *et al.*[1] report that highly diffusible hydrogen gas can target intracellular sources of reactive oxygen species and dose-dependently inhibit reperfusion-induced oxidative damage.

Numerous studies have consistently demonstrated a burst of reactive oxygen species on restoration of blood flow after a stroke[2,3]. Reactive oxygen species, such as superoxide, have been suggested to be the primary activator of the mitochondrial permeability transition pore, a large multiprotein conductance channel[4]. The opening of this channel causes a loss of membrane potential, mitochondrial swelling with membrane rupture, cytochrome C release and apoptotic cell death.

After ischemic damage to the mitochondrial electron transport chain, there is inefficient transfer of electrons to molecular oxygen, leading to the generation of superoxide. What's more, activation of superoxide-producing enzymes, such as xanthine oxidase and NADPH oxidase, following isch-

respiratory complexes I and III prevent reperfusion reactive oxygen species generation and improve cellular viability[5–7].

The lightweight gas diatomic hydrogen (H_2), a major component of interstellar space and the fuel that sustains the stars, is rare on Earth. Hydrogen gas directly and violently reacts with oxidizing elements such as chlorine and fluorine and is highly flammable, a property evident in the 1937 Hindenburg zeppelin fire and its use as propellant fuel for the space shuttle. Hydrogen gas is highly diffusible and reacts with hydroxyl radical to produce water[8].

Ohsawa *et al.* set out to see if hydrogen gas could be used as a therapeutic mitochondrial antioxidant to neutralize oxidative stress after ischemia-reperfusion injury[1]. To induce the production of reactive oxygen species, the authors treated cultured cells with a mitochondrial respiratory complex I inhibitor or subjected them to oxygen or glucose deprivation. After oxidative damage, cells underwent pathological mitochondrial depolarization, ATP depletion, DNA oxidation, lipid peroxidation, and cellular necrosis and apoptosis

THE ENHANCED ZEOLITE

with BioEn'R-G'y C and negatively charged micro-bubbles of Hydrogen!

Assisting in maintaining the electrical balance that enables cell structures to communicate and function properly.

When MicroHydro Zeolite CEA (cation exchange activator) is added to water, the pH shifts to a slightly alkaline state as multitudes of negative ions, as stable MICROBUBBLES, cascade into solution.

The effect is a rapid change of the oxidation-reduction potential (ORP) toward the high negative millivolt (mv) range.

Detoxification is a LIFETIME challenge

LEAD in bones requires years of continuous oral chelation with EDTA and/or Zeolite.

Because bones take an average of 15 years to fully regenerate, IV EDTA chelation therapy over several months only removes lead and other toxic metals from the body's blood and tissues, NOT from bones.

Harvard studies prove that bone lead leads to heart disease and cataracts, as Bones are the MAJOR storehouse of lead in the body.

For more information see the

507 References Supporting Oral EDTA

On the Gordon Research Institute Website at

www.gordonresearch.com

The Case Against Detoxing

Why detoxing can actually make your body more toxic…

Most toxins are stored in your fat cells. When you begin a detox program, you pull these toxins out of your fat cells and into your bloodstream, where they can travel through your body to your vital organs, your brain, invade your joints and tissues, triggering pain and inflammation, cause headaches, memory loss and premature brain aging.

And they can invade your heart, where they can cause blood pressure problems.

Because these toxins do not dissolve in water, your body cannot eliminate them easily. Before it eliminates them, it has to make them water-soluble. Your liver makes the toxins water-soluble so they can be excreted in the urine or via the bile. That's why your bile is full of toxins. Every day, your liver dumps bile into your intestines so the toxins can be eliminated through your stool.

The problem is that the toxins must first bind with fiber in your intestines. And if you don't eat enough fiber, the toxins are simply re-absorbed through your intestines, and sent right back into your body!

FIGHT for Your Health with Dr. Gordon's Power Drink

Beyond Fiber - 1 rounded tsp

Bio En'R-G'y C - 1 rounded tsp

MACA Powder - 1/2 tsp

Dr. Gordon's Organic Best of Greens - 1 rounded tsp

ZeoGold* - 1 capsule (twist open and dissolve in drink)

YouTube

Dr. Gordon is considered to be the Father of Chelation Therapy and carries the banner for "alternative medicine" in the United States. He is a member of many of the leading professional medical bodies, a medical researcher, consultant and a legal expert providing defense for alternative medical practices.

Garry Gordon - Zeolite (ZeoGold™)

http://www.youtube.com/watch?v=9HHis6uCsVU

fitgenes

Key Health Factors Addressed

- Diabetes and Insulin Resistance
- Cardiovascular Health and Cholesterol Regulation
- Fat Metabolism and Weight Management
- Bone Health
- Hormone Regulation
- Thyroid Function
- Sleep Patterns
- Relief from joint pain
- Healthier ageing
- Menopause balance
- Hypertension - Blood pressure

Personalized Genetic Profiling in Support of Your Health and Wellness Goals

THANK YOU

Garry F. Gordon MD, DO, MD(H)


ANTI AGEING CONFERENCE LONDON
10
YEARS
SEPTEMBER 19-21, 2013
LEADING 21ST CENTURY MEDICINE
AACL2013