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AAACL 2013

How Energy Medicine, Hydrogen and Lasers Can Save Your Life

Dr Garry Gordon's Abstract



AACL 2013



Anti Ageing Conference London 2013

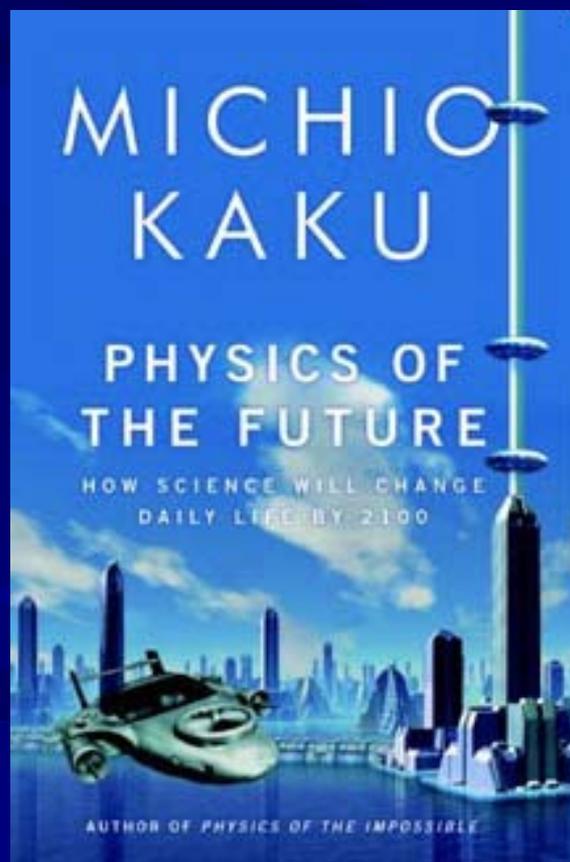
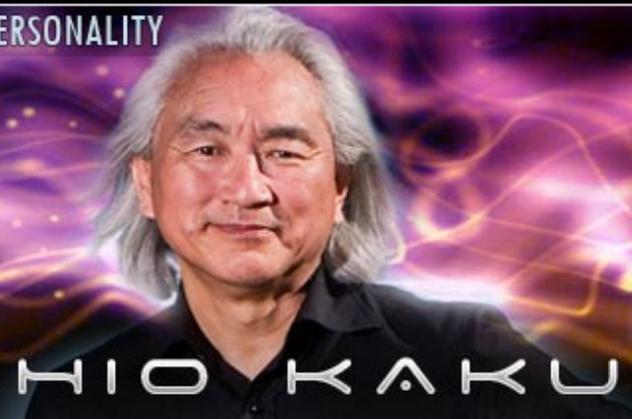
How Energy Medicine, Hydrogen and Lasers Can Save Your Life

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

**Anti-Ageing Conference London (AACL) 2013
Thursday Pre-conference Workshop
19th September 2013 - 8.00am - 1.00pm**

mk

DR. MICHIO KAKU



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.

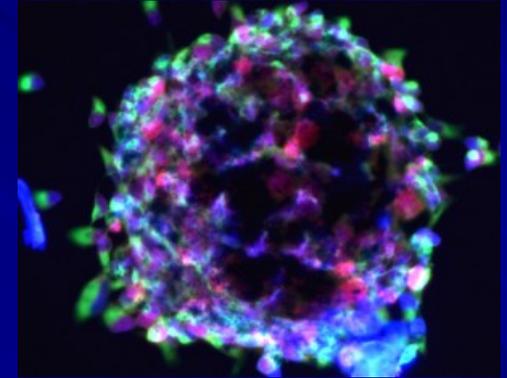
Dr. Kaku's astonishing revelations include:

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.

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

by Joseph Rago

THE WALL STREET JOURNAL.

U.S. EDITION

Sunday, September 8, 2013 As of 12:04 PM EDT

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>

HOW TO REGROW A SEVERED FINGER

1 Start to make 'pixie dust' by cutting open a pig's bladder and flattening it out.



2 Scrape away the layer of muscle before 'cleaning' the remaining collagen-rich tissue by shaking it in acid.

3 Dry out the paper-like 'extra-cellular matrix' and grind into powder form.

4 Regularly sprinkle powder on severed finger tip.



5 Within a few weeks, tip grows to normal length, complete with nail and 'fingerprint'.



Future Medicine Human Tissue Regeneration

Lee Spievack, a model aircraft enthusiast from Cincinnati in the U.S. who, in 2005, accidentally sliced an inch off the tip of his index finger with a model aeroplane propeller.

A collagen powder derived from pigs' bladders, appeared to provide a suitable "matrix" or framework, stimulating regrowth of the tissues and division of the cells, to enable Mr Spievack's finger to grow back - in just a month - flesh, tendon, skin, fingernail, fingerprints and all!

Dr Stephen Badylak of the University of Pittsburgh is the scientist who developed the pixie dust. It consists of a mixture of protein and connective tissue which is already used by surgeons to repair tendons.

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

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.

SCIENCE FOR THE CURIOUS
Discover



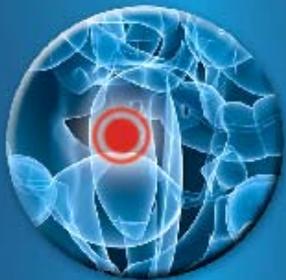
The peptides have potent antimicrobial effects and important signaling abilities... during scaffold breakdown, information held within the structural ECM molecules, recruit all-purpose “stem cells” that can develop into any type of tissue.

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

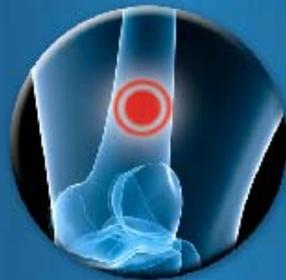
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Fast
Symptom Relief



*Uterine Fibroids
and Adenomyosis*



*Bone Tumor Pain
Palliation*



Brain Disorders

**ExAblate O.R. – The Operating Room
of the Future**

<http://www.insightec.com/>

InSightec® is the pioneer and global leader in MR guided Focused Ultrasound Surgery (MRgFUS).

ExAblate MRgFUS provides a personalized non-invasive treatment that can replace invasive procedures and offer therapeutic alternatives to millions of patients with serious diseases around the globe.





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Exploring the Evidence and Emerging
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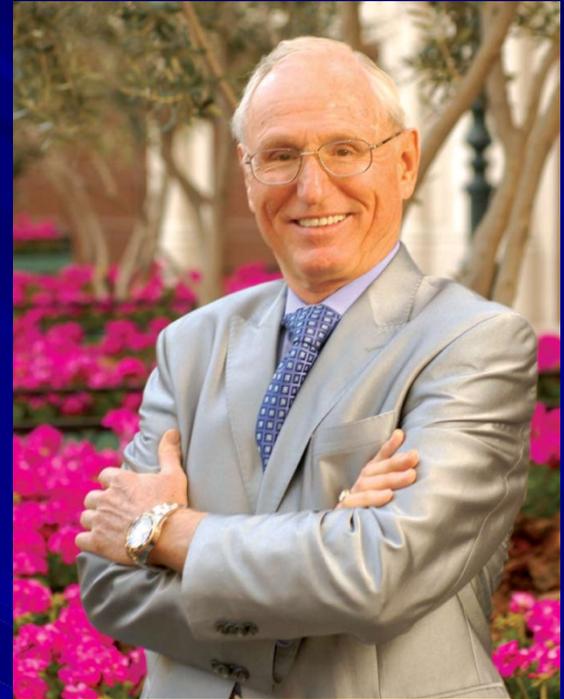
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Through the use of clinical nutrition, dietary modifications, and nutraceuticals, as well as therapeutic modalities such as homeopathy, acupuncture, microcurrent, laser, pulsed electromagnetic field, craniosacral therapy, and guided imagery.

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”**



Townsend Letter – July 2007

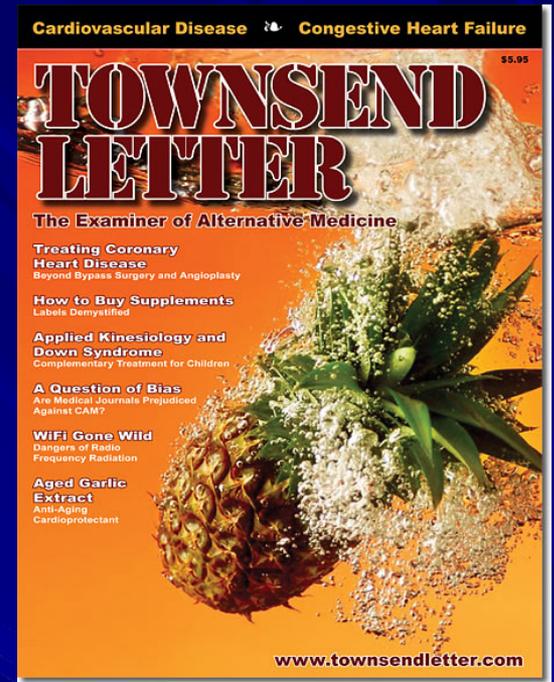
Chelation, Heavy Metals, Heart Disease, and Health: An Oral Detoxification Program That Is Now Essential for Optimal Health and Longevity

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

The Oral Detoxification Program: A Wide-Spectrum Protocol Certain 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>



Effect of Disodium EDTA Chelation Regimen on Cardiovascular Events in Patients With

Previous Myocardial Infarction - The TACT Randomized Trial

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

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

TOXINS

Oxidative Stress & Disease

**Many diseases have been linked to
oxidative stress....**

The Environmental Working Group studies that have shown:



From
Environmental Working
Group

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

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.

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.**



a coal-fired power plant in Dadong, Shanxi province

Fluoride Increases Heavy Metal Accumulation in Your Body

<http://www.youtube.com/watch?v=dKrcmOTmhxo>



Studies confirm that hydrofluorosilicic acid increases lead accumulation in bone, teeth, and other calcium-rich tissues.

The free fluoride ion actually acts as a *transport* of heavy metals, allowing them to enter into areas of your body they normally would not be able to go, such as into your brain.

Fluoride is... the most aggressive seeker of another electron. It's the most electromagnetically negatively charged element in the entire world.

"There is a current and growing body of peer reviewed scientific publications showing that fluoridated water causes gene damage leading to birth defects and cancer and that humans are genetically different in their sensitivity to levels of fluoride in their drinking water. "...

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>

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, MD, MCV

Diabetes. Dr. Bland currently serves as chief science officer for Allergan, a company that sells supplements and a provider of biotechnology and pharmaceuticals in El Segundo, California.

Editor's Note: The following is an adaptation of an August 2010 interview with Dr. Bland during his sabbatical sabbatical in Japan, an audio journal from the 20th year of publication, *Integrative Medicine: A Clinician's Journal* is pleased to publish this exclusive joint presentation.



David R. Jacobs, PhD is an author of 100+ books. His research focuses on the relationship between diet and health. He has written extensively on the relationship between diet and health. He has written extensively on the relationship between diet and health. He has written extensively on the relationship between diet and health.

Working in epidemiology means that a person looking at associations, and he has done an extraordinary amount of work in the whole history of man. Let me give you a thumbnail of some of the major ones.

I have been involved with Dr. Jacobs regarding the work he has done on whole grains and refined-grain intake and the relationship to chronic illness—some very important associations that I'm sure we'll touch upon in this interview. He's also looked at inflammatory processes and oxidation and how these 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 its relationship to xenobiotics and persistent organic pollutants (POPs). Do you mind first reviewing the story on how these chemicals are biological factors?

Dr. Jacobs has done an extraordinary degree of collaborative interdisciplinary work looking at the epidemiology and the association between POPs and type 2 diabetes?

David Jacobs: Statistically, population science 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 mathematics, they're just applied, they don't really know the answers." But it turns out that the substance behind the numbers—that which causes the numbers—is, of course, extremely important, and I have done throughout my career to really understand the substance. Actually, I'm much more interested in the substance than in the mathematical principles. That's the short answer.

It's a bit of a hallmark of your work is the collaboration you've had with individuals at the National University of Health Sciences looking at this xenobiotic connection, actually going back and looking at data from NHANES, the National Health and Nutrition Examination Survey, II and III. Maybe you could elaborate through that whole story because the way your role in epidemiology changed somewhat looking. I'd love to know where it came from.

Dr. Jacobs: The story with Dr. 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 read and literature, but her in the sense, I can often refer to the my English language skills to help people who have written in a foreign language or have a subtle language other than English. My Lee and I had a bright off because I can only read 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 here 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 e-mail 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 she was interested in a variety of things and I'm interested that we did not

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

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

COMMENTARY

Open Access

Mitochondrial oxidative stress drives tumor progression and metastasis: should we use antioxidants as a key component of cancer treatment and prevention?

Federica Sotgia^{1,2,3}, Ubaldo E Martinez-Outschoorn^{1,2,4} and Michael P Lisanti^{1,2,3,4*}

Abstract

The functional role of oxidative stress in cancer pathogenesis has long been a hotly debated topic. A study published this month in *BMC Cancer* by Goh et al., directly addresses this issue by using a molecular genetic approach, via an established mouse animal model of human breast cancer. More specifically, alleviation of mitochondrial oxidative stress, via transgenic over-expression of catalase (an anti-oxidant enzyme) targeted to mitochondria, was sufficient to lower tumor grade (from high-to-low) and to dramatically reduce metastatic tumor burden by >12-fold. Here, we discuss these new findings and place them in the context of several other recent studies showing that oxidative stress directly contributes to tumor progression and metastasis. These results have important clinical and translational significance, as most current chemo-therapeutic agents and radiation therapy increase oxidative stress, and, therefore, could help drive tumor recurrence and metastasis. Similarly, chemo- and radiation-therapy both increase the risk for developing a secondary malignancy, such as leukemia and/or lymphoma. To effectively reduce mitochondrial oxidative stress, medical oncologists should now re-consider the use of powerful anti-oxidants as a key component of patient therapy and cancer prevention.

Please see related research article: <http://www.biomedcentral.com/1471-2407/11/191>

Introduction

Mitochondrial oxidative stress has long been implicated in normal aging, and a host of human diseases, including cancer and neurodegenerative disorders, such as Alzheimer's disease. In support of this idea, vegetarians, who consume a diet rich in anti-oxidants, have reduced rates of cancer incidence, have longer life expectancies, and suffer less from dementia [1-3].

Similarly, breast cancer patients taking anti-oxidants showed reduced rates of recurrence, as well as less risk of mortality [4]. In fact, N-acetyl-cysteine (NAC), a powerful anti-oxidant, has anti-tumor properties, and has been recommended for melanoma chemo-prevention [5]. Finally, metformin therapy, a powerful anti-oxidant which reduces reactive oxygen species (ROS) production from mitochondrial complex I, has been associated with a lower risk of various epithelial cancers, in more than 11 studies [6,7].

A simple PubMed search reveals that nearly 9,000 articles have been published linking oxidative stress with cancer pathogenesis. Thus, it is surprising that anti-oxidants are not routinely used as a component of cancer therapy and prevention.

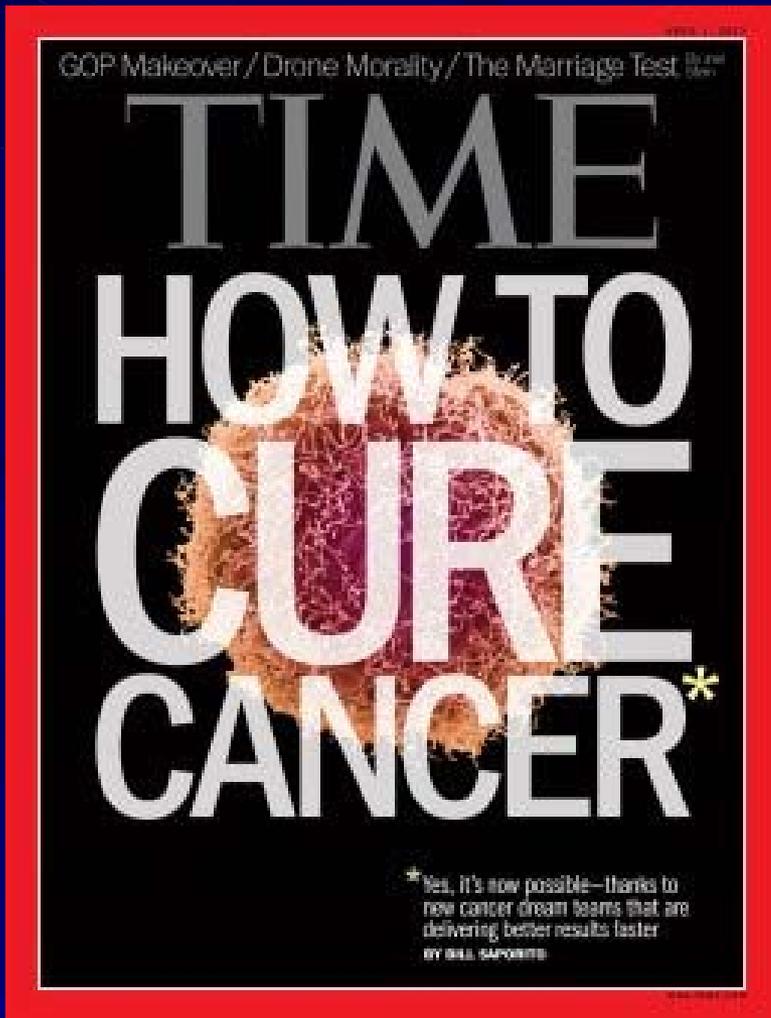
Genetic reduction of mitochondrial oxidative stress reduces tumor grade and inhibits metastasis

This month in *BMC Cancer*, Goh and colleagues [8] use an established mouse model of breast cancer tumor formation and metastasis (MMTV-PyMT) to explore the role of mitochondrial oxidative stress in cancer patho-

One out of every three adults in America has cancer, but most incidences are as yet undiagnosed and undetected.

According to the American Cancer Society, “the probability that an individual, in the course of their lifetime, will develop cancer or die from it” is 1 in 2 for men (50%), and 1 in 3 for women (33.3%). Extrapolating from the male-female ratio of 49:51, the chances that any individual, male or female, will get cancer in their lifetime is 41%!

Every human being has cancer cells existing in the body which are just seeking a low-oxygen environment where they can multiply into the full-blown disease.



The Conspiracy to End Cancer

by Bill Saporito

April 1, 2013

The hero scientist who defeats cancer will likely never exist. It will take not one hero but many.

Cancer is not just one disease, it is potentially thousands. And not all cancers are caused by just one agent — a virus or bacterium that can be flushed and crushed.

Cancer is an intricate, potentially lethal collaboration of genes gone awry, of growth inhibitors gone missing, of hormones and epigenomes changing and rogue cells breaking free. It works as one great armed force, attacking by the equivalent of air and land and sea and stealth...

Cancer research has traditionally involved a narrowly focused investigator beavering away, one small grant at a time. But advances in genetic profiling of malignancies and mutations that cause them are telling scientists and physicians they must stop treating lung or breast or colon or prostate cancer as distinct diseases.

<http://healthland.time.com/2013/04/01/the-conspiracy-to-end-cancer/>

New Ecosystem of Cancer Research: Cross Institutional Team Science - March 24, 2013 by 2012pharmaceutical

Today the physics of cancer are known; what remains is massive engineering.

Now the Cure for Cancer is possible thanks to the following innovations in the Division of Labor of the research process among integrative institutions.

1. New Cancer Dream Teams deliver better results faster, better understand the metabolic changes of pancreatic cells. Joint Lab work: Superior to any research ever known.

2. Drug agents in development for therapy **targeting the genetic mutations**

- > reactivate the body's immune system
- > cut off a tumor's blood or energy supply
- > restart apoptosis

3. New Biomarkers

Allows to identify, target and track cancer cells – PI3K mutation One pathway – three women's Cancers: Ovarian, endometrial, Breast CA.

4. Design and built of a **smart chip device to trap circulating tumor cells (CTCs) in a blood sample – early identification of metastasis**

Does breast screening do more harm than good?

NHS Choices – Tuesday October 30, 2012

The media reports that breast cancer screening is "harming thousands", with The Guardian claiming "breast cancer screening causes more damage than previously thought".



Breast cancer screening programmes are unable to predict the individual outcome (prognosis) if a person is found to have breast cancer. In some cases, the cancerous cells can spread rapidly, posing a significant risk to health. In others, the cancerous cells are much less aggressive, so the cancer has no impact on life expectancy. This uncertainty leads to what the researchers term 'overdiagnosis' – where women are given treatment and are exposed to all of its harms, but receive no benefit.

Therefore, **for every death prevented, there are estimated to be three cases of overdiagnosis**. This means that, in around 307,000 women aged 50-52 who are invited to screening each year in the UK, about 1,320 deaths from breast cancer will be prevented and about 3,960 women will be overdiagnosed. The panel noted that there is uncertainty around these estimates, and that they should be seen as approximate.

Voluntary mastectomies to reduce breast cancer risk becoming more common

By Adam Smeltz

May 15, 2013

Actress Angelina Jolie told the world on Tuesday that she prefers life over breasts, announcing a voluntary double mastectomy (*after testing positive for the genetic mutation BRCA1*) that dropped her inherited cancer risk from 87 percent to 5 percent.

Genetic testing for breast cancer has been available for about a decade, but has become popular only in the past five years, physicians said. They said a **blood test costing roughly \$3,000 identifies the gene defect**, which can raise the lifetime risk of breast cancer to 80 percent or higher.

<http://triblive.com/news/allegHENY/4018753-74/breast-cancer-women#axzz2WDQ5xAwx>

TRIB LIVE



Instead of having a “preventative surgery” what if we tried to prevent cancer by reducing the exposure to toxins that cause cancer? Whether or not you have the genetic markers for a disease, there are still many things that you can do to keep that disease from developing that do not include voluntary amputations.

Justices Strike Down Gene Patents

By JESS BRAVIN and BRENT KENDALL

THE WALL STREET JOURNAL.

U.S. EDITION

Thursday, June 13, 2013 As of 7:35 PM EDT

WASHINGTON—The Supreme Court unanimously held Thursday that human genes cannot be patented, even when isolated from the body, a ruling expected to quickly expand access to genetic testing while potentially allowing inventors to retain rights to artificially created DNA.

The decision marked the latest step in the court's decade long march to toughen the requirements for patents.

The justices repeatedly have declared that 21st-century innovation depends less on locking up intellectual-property rights than on expanding access to discoveries in order to spur further progress.

The case involved two genes, known as BRCA1 and BRCA2, where mutations can indicate significant likelihood of breast and ovarian cancer.

The actress Angelina Jolie recently thrust genetic testing into the spotlight when she disclosed that she possessed the cancer-associated mutation, prompting her to undergo a double mastectomy.



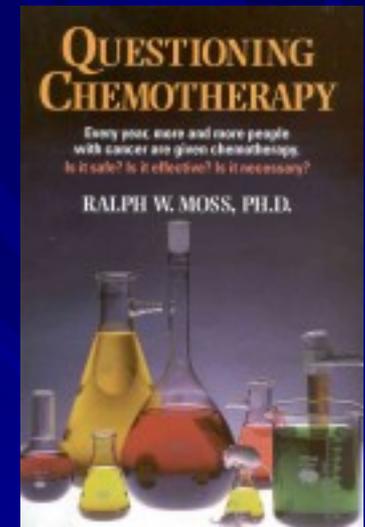
What Cancer Doctors say about Chemo Therapy?

Scientists based at McGill Cancer Centre sent a questionnaire to 118 lung cancer doctors to determine what degree of faith these practicing cancer physicians placed in the therapies they advise and administer.

They were asked to imagine that they had cancer and were asked which of six current trials they would choose. **Of 79 doctors who responded, 64 said they would NOT consent to be in any trial containing Cisplatin - one of the common chemotherapy drugs they were trialing** (currently achieving worldwide sales of about \$110,000,000 a year), and 58 of the 79 found that all the trials in question were **unacceptable due to the ineffectiveness of chemotherapy and its unacceptably high degree of toxicity.**

Chemotherapy's use is now rampant not just in the United States, where it began, but in Canada, Chile, Denmark, France, Germany, Italy, South Africa—actually throughout most of the industrialized, and some of the developing, world. In fact, in Great Britain, 60 different cytotoxic drugs—more than in the United States—are now licensed for use in cancer therapy.

With Questioning Chemotherapy, I hope to spark an international debate on the value of toxic drugs in the treatment of cancer.



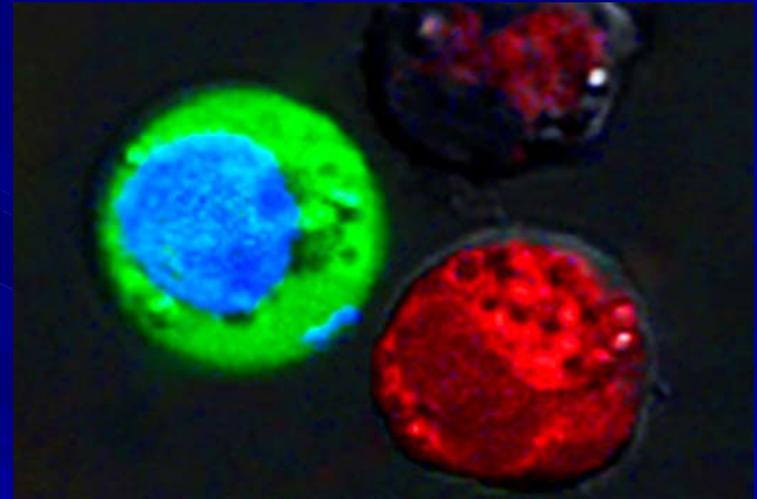
Physicists Kill Cancer Cells with NANOBUBBLES

Jade Boyd – February 2010



Activated by a pulse of laser light, nanobubbles can kill diseased cells while leaving healthy cells untouched.

Nanobubbles are created when gold nanoparticles are struck by short laser pulses. The short-lived bubbles are very bright and can be made smaller or larger by varying the power of the laser. Because they are visible under a microscope, nanobubbles can be used to either diagnose sick cells or to track the explosions (apoptosis) that are destroying them.

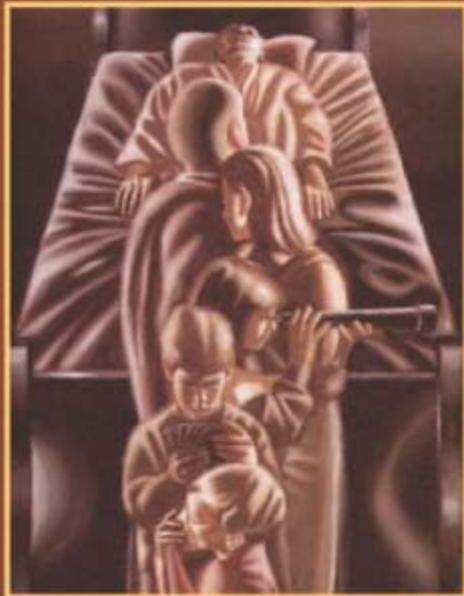


After the laser pulse, red-stained sick cells show evidence of massive damage from exploding nanobubbles, while blue-stained healthy cells remained intact, but with green fluorescent dye pulled in from the outside. (Credit: Plasmonic Nanobubble Lab/Rice University)

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Cancer as a Metabolic Disease

On the Origin, Management,
and Prevention of Cancer



Thomas N. Seyfried

 WILEY

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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.

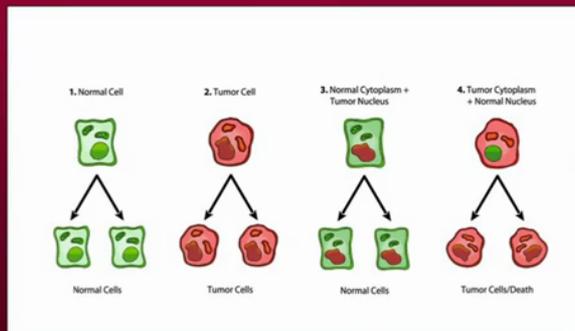
Cancer is a disease of defective cellular energy metabolism, and most of the genomic defects found in cancer arise as secondary downstream effects of defective energy metabolism.

Thomas Seyfried, Ph.D.—Targeting Energy Metabolism in Brain Cancer



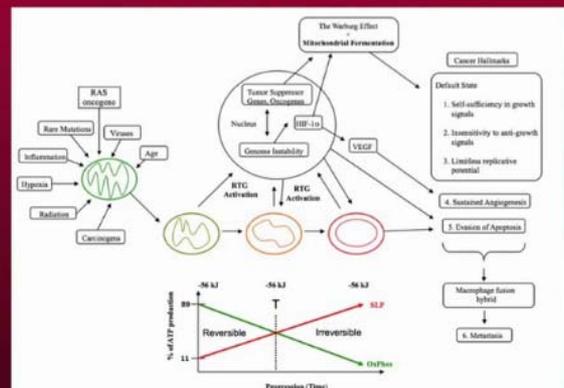
Is cancer a nuclear genetic disease or a mitochondrial metabolic disease? As a metabolic disorder involving the dysregulation of respiration, malignant brain cancer can be managed through changes in metabolic environment. Warburg was correct.

Role of the nucleus and mitochondria in the origin of tumors



Seyfried, *Cancer as a Metabolic Disease*, John Wiley & Sons, 2012

Cancer as a Metabolic Disease



Seyfried, *Cancer as a Metabolic Disease*, 2012, John Wiley & Sons



<https://www.youtube.com/watch?v=sBjnWfT8HbQ>



The Prime Cause and Prevention of Cancer **Dr. Otto Warburg – 1931 Nobel Laureate**

Dr. Warburg stated “Cancerous tissues are acidic, whereas healthy tissues are alkaline. Water splits into H^+ and OH^- ions, if there is an excess of H^+ , it is acidic; if there is an excess of OH^- ions, then it is alkaline.”

...tumors live in the body anaerobically.

...cell respiration is impaired if the active groups of the respiratory enzymes are removed from the food; and that cell respiration is repaired at once, if these groups are added again to the food. No way can be imagined that is scientifically better founded to prevent and cure a disease, the prime cause of which is an impaired respiration.

...the prevention of cancer requires no government help, and no extra money.

Healthy tissues are alkaline whereas cancerous tissues are acidic. Cancer does not survive in an alkaline state.

Mol Aspects Med. 2010 Feb;31(1):60-74. Epub 2009 Dec 6.

The Warburg effect and mitochondrial stability in cancer cells.

Gogvadze V, Zhivotovsky B, Orrenius S.

Institute of Environmental Medicine, Division of Toxicology, Karolinska Institutet, Box 210, Stockholm SE-17177, Sweden.



Abstract

The last decade has witnessed a renaissance of Otto Warburg's fundamental hypothesis, which he put forward more than 80 years ago, that mitochondrial malfunction and subsequent stimulation of cellular glucose utilization lead to the development of cancer.

Since most tumor cells demonstrate a remarkable resistance to drugs that kill non-malignant cells, the question has arisen whether such resistance might be a consequence of the abnormalities in tumor mitochondria predicted by Warburg.

The present review discusses potential mechanisms underlying the upregulation of glycolysis and silencing of mitochondrial activity in cancer cells, and how pharmaceutical intervention in cellular energy metabolism might make tumor cells more susceptible to anti-cancer treatment.

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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.

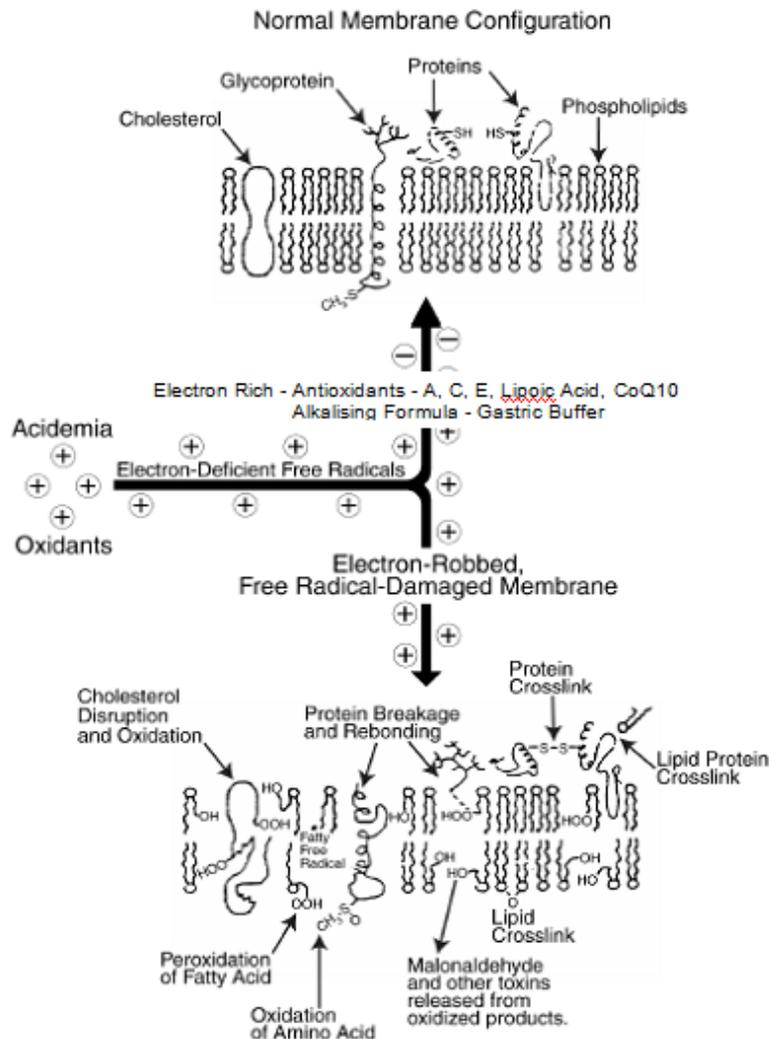
The movement of electrons between chemical species is reduction (for the electron acceptor) and oxidation (for the electron donor). **Reduction and oxidation always go together, hence 'redox' reactions.**

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.

Oxidation and Reduction

Electron Robbing, Free Radical Damage



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 (reductant)** 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.

CAUSE OF CANCER & pH

by Herman Aihara, author of "Acid & Alkaline"

If the condition of our extra cellular fluids, especially the blood, becomes acidic, our physical condition will first manifest tiredness, proneness to catching colds, etc. When these fluids become more acidic, our condition then manifests pains and suffering such as headaches, chest pains, stomach aches, etc.

According to Keiichi Morishita in his Hidden Truth of Cancer, If the Blood develops a more acidic condition, then our body inevitably deposits these excess acidic substances in some area of the body such so that the **blood will not be able to maintain an alkaline condition** which causes these areas such as the **cells to become acidic and lowers in oxygen.**

Some cells, instead of dying - as normal cells do in an acid environment - survive by becoming abnormal cells. Abnormal, or malignant cells **THRIVE** in an acidic and anaerobic (low oxygen) environment.

They do not correspond with brain function, nor with our own DNS memory code. This is cancer.

pH (Hydrogen potential) and 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 to describe Acidosis

Cancer Res Published OnlineFirst January 3, 2013.

Acidity generated by the tumor microenvironment drives local invasion

Veronica Estrella, Tingan Chen, Mark Lloyd, et al.

The morbidity and mortality associated with cancer is largely related to tumor invasion and formation of metastases. Extensive application of FDG-PET imaging to clinical cancers has clearly demonstrated the vast majority of malignant tumors metabolize glucose at high rates.

We propose **there is a direct, causative link between increased glucose metabolism and the ability of cancer cells to invade and metastasize.**

Elevated glucose metabolism is the proximate cause of increased acidity in the tumor microenvironment. Furthermore, most tumors develop an aberrant vasculature network that tends to be poorly organized and leaky, disrupting blood flow and hampering the delivery of oxygen. This has a two-fold effect on tumor acidity.

First, it subjects tumor regions to poor perfusion and hence, poor oxygenation (36). Low oxygenation increases glycolytic flux via the Pasteur Effect. Notably, **even in tumor regions with adequate oxygen supply, glycolysis and acid production are up regulated via the Warburg Effect.** Second, **poor perfusion hampers the ability of the microenvironment to remove tumor derived acid through diffusion.** Consequently, the extracellular pH of tumors is typically highly acidic, and this will inevitably result in acid diffusion into the surrounding stroma.



Swiss Med Wkly. 2012 Aug 17;142:w13659.

Reactive oxygen species: from health to disease.

Brieger K, Schiavone S, Miller FJ Jr, Krause KH.

Department of Pathology and Immunology, University of Geneva Faculty of Medicine and Geneva University Hospitals, Switzerland.



Abstract

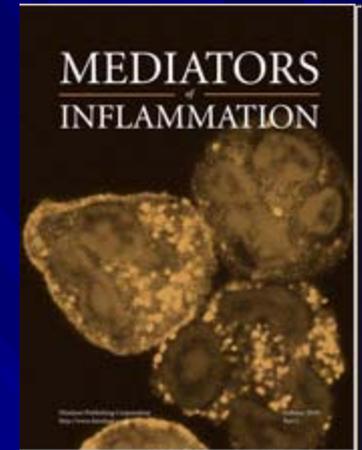
Upon reaction with electrons, oxygen is transformed into reactive oxygen species (ROS). It has long been known that ROS can destroy bacteria and destroy human cells, but research in recent decades has highlighted new roles for ROS in health and disease. Indeed, while **prolonged exposure to high ROS concentrations may lead to non-specific damage to proteins, lipids, and nucleic acids, low to intermediate ROS concentrations exert their effects rather through regulation of cell signalling cascades.** Biological specificity is achieved through the amount, duration, and localisation of ROS production. ROS have crucial roles in normal physiological processes, such as through redox regulation of protein phosphorylation, ion channels, and transcription factors. ROS are also required for biosynthetic processes, including thyroid hormone production and crosslinking of extracellular matrix. There are multiple sources of ROS, including NADPH oxidase enzymes; similarly, there are a large number of ROS-degrading systems. ROS-related disease can be either due to a lack of ROS (e.g., chronic granulomatous disease, certain autoimmune disorders) or a surplus of ROS (e.g., cardiovascular and neurodegenerative diseases). **For diseases caused by a surplus of ROS, antioxidant supplementation has proven largely ineffective in clinical studies, most probably because their action is too late, too little, and too non-specific.**

Mediators of Inflammation

Volume 2011 (2011), Article ID 891752, 7 pages
doi:10.1155/2011/891752

Clinical Study: Malondialdehyde in Exhaled Breath Condensate as a Marker of Oxidative Stress in Different Pulmonary Diseases

M. L. Bartoli, F. Novelli, F. Costa, L. Malagrino, L. Melosini, E. Bacci, S. Cianchetti, F. L. Dente, A. Di Franco, B. Vagaggini, and P. L. Paggiaro



Oxidative stress plays an important role in the pathogenesis of many chronic inflammatory lung disorders, particularly in COPD and asthma, where it is an important consequence of irritant-induced damage of bronchial epithelial cells...

Among the many biological targets of oxidative stress, membrane lipids are the most commonly involved class of biomolecules. Lipid peroxidation yields a number of secondary products able to boost oxidative damage. In addition to their cytotoxic properties, lipid peroxides are increasingly recognized as being important in signal transduction for a number of events in the inflammatory response.

Malondialdehyde (MDA) has been widely studied as a product of polyunsaturated fatty acid peroxidation. High MDA levels have been observed in several biological fluids from patients with different airway diseases including asthma, COPD, and bronchiectasis.

Circulation. 2011 Aug 15. [Epub ahead of print]



Atrial Sources of Reactive Oxygen Species Vary With the Duration and Substrate of Atrial Fibrillation: Implications for the Antiarrhythmic Effect of Statins.

Reilly SN, Jayaram R, Nahar K, Antoniadou C, Verheule S, Channon KM, Alp NJ, Schotten U, Casadei B.

Department of Cardiovascular Medicine, University of Oxford, John Radcliffe Hospital, Oxford, UK.

Background- An altered nitric oxide-redox balance has been implicated in the pathogenesis of atrial fibrillation (AF). Statins inhibit NOX2-NADPH oxidases and prevent postoperative AF but are less effective in AF secondary prevention; the mechanisms underlying these findings are poorly understood. Rac1 and NADPH oxidase activity and the protein level of NOX2 and p22phox were significantly increased in the left atrium of goats after 2 weeks of AF and in patients who developed post-operative AF in the absence of differences in leukocytes infiltration.

Conversely, in the presence of longstanding AF or atrioventricular block, uncoupled nitric oxide synthase activity (secondary to reduced BH(4) content and/or increased arginase activity) and mitochondrial oxidases accounted for the biatrial increase in reactive oxygen species. **Conclusions-** Upregulation of atrial NADPH oxidases is an early but transient event in the natural history of AF.

Changes in the sources of reactive oxygen species with atrial remodeling may explain why statins are effective in the primary prevention of AF but not in its management.

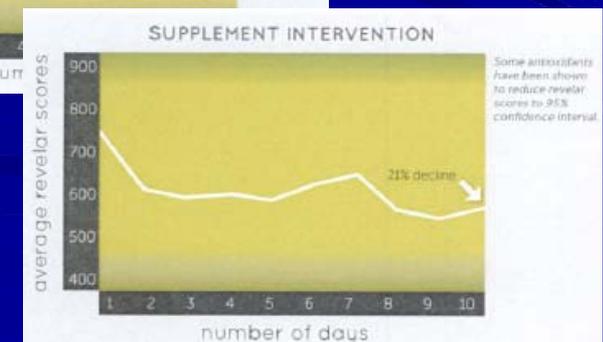
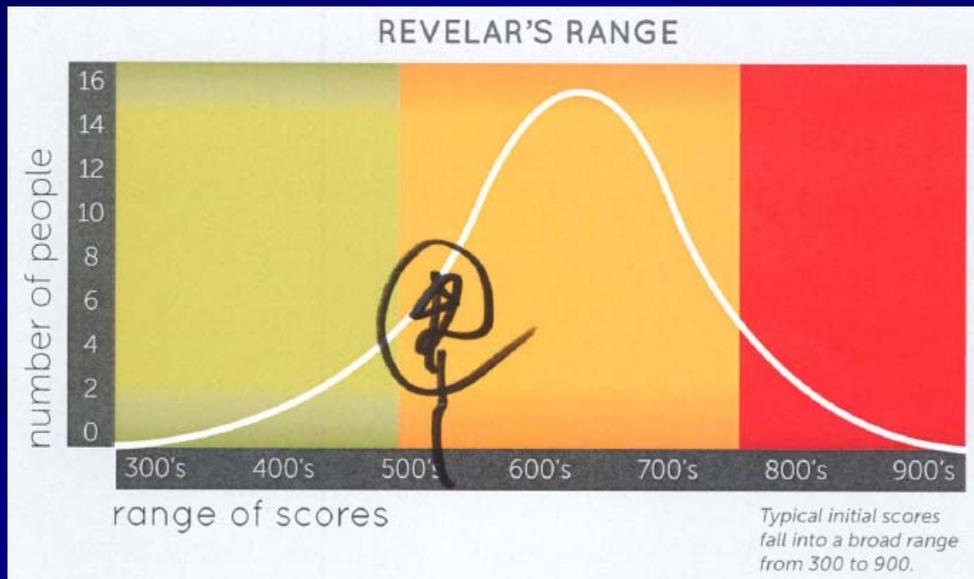
PMID: 21844076 [PubMed - as supplied by publisher]

REVELAR™

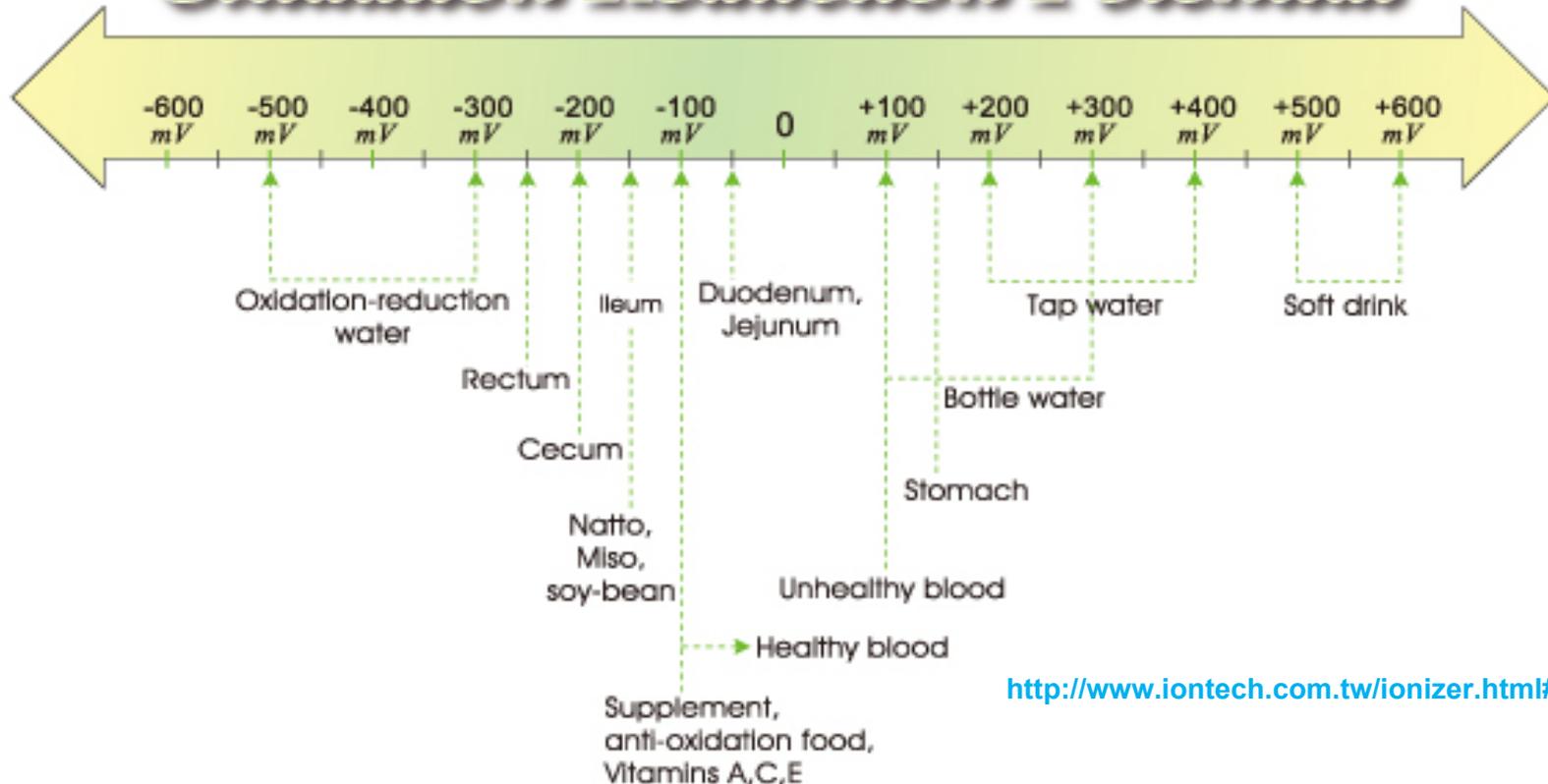


Antioxidant supplements can protect us from free radical damage. But which supplements and regimen really work?

Revelar provides the first accurate aldehyde measurement system that both detects and measures aldehydes in the breath. Aldehydes are known to be indicators of free radical damage also known as oxidative stress.



Oxidation Reduction Potential



<http://www.iontech.com.tw/ionizer.html#I05>

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.

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.

Dr. Garry Gordon's F²IGH²T For Your Health Program

F² = 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

H² = 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.

E² = Energy and Exercise - PEMF or pulsed electromagnetic frequency therapy that promotes healing through



Magnetically Induced Cellular Exercise, or MICE

F = Food

Many people are sensitive to specific foods which when eaten become stressful to the body. The immune system must then mount a response to these substances every time they are consumed.

40% of the population needs to be off all dairy, and 30% should eat a gluten-free diet". In people with chronic illness, these numbers are likely even higher.

IS SUGAR TOXIC?



New research coming out of some of America's most respected institutions is starting to find that sugar is a toxin and is a driving force behind some of this country's leading chronic diseases, including Cancer obesity, type II diabetes, hypertension and heart disease.

(CBS News) If you are what you eat, then what does it mean that the average American consumes 130 pounds of sugar a year? Sanjay Gupta reports on new research showing that beyond weight gain, sugar can take a serious toll on your health, worsening conditions ranging from heart disease to cancer. Some physicians go so far as to call sugar a toxin.

Since the 1970s, sugar consumption has gone down nearly 40 percent, but high fructose corn syrup has more than made up the difference. Dr. Robert Lustig, a pediatric endocrinologist at University of California, San Francisco, says they are both toxic because they both contain fructose – and that's what makes them sweet and irresistible... and dangerous.

Modern Wheat a "Perfect, Chronic, Poison" Doctor Says...



September 3, 2012

(CBS News) Modern wheat is a "perfect, chronic poison," according to Dr. William Davis, a cardiologist who has published a book all about the world's most popular grain.

Davis said that the wheat we eat these days isn't the wheat your grandma had: "It's an 18-inch tall plant created by genetic research in the '60s and '70s. This thing has many new features nobody told you about, such as there's a new protein in this thing called gliadin. It's not gluten. I'm not addressing people with gluten sensitivities and celiac disease. **I'm talking about everybody else because everybody else is susceptible to the gliadin protein that is an opiate.** This thing binds into the opiate receptors in your brain and in most people stimulates appetite, such that we consume 440 more calories per day, 365 days per year."

Lose the wheat... lose the weight

"If three people lost eight pounds, big deal," he said. "But **we're seeing**

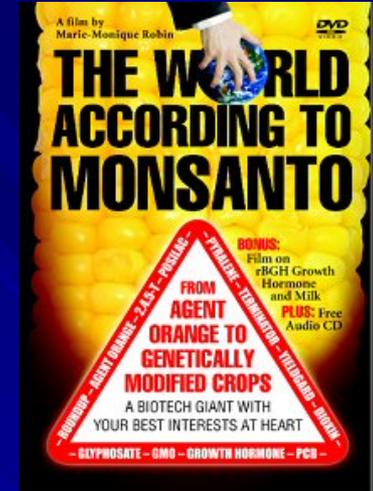
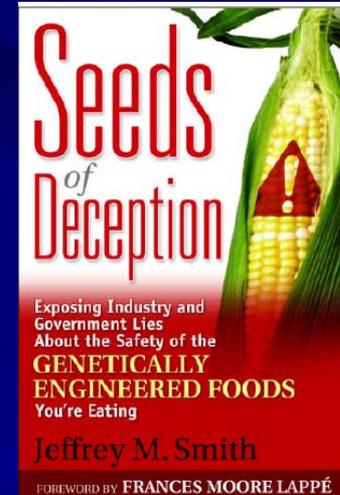
hundreds of thousands of people losing 30, 80, 150 pounds. Diabetics become no longer diabetic; people with arthritis having dramatic relief. People losing leg swelling, acid reflux, irritable bowel syndrome, depression, and on and on every day."



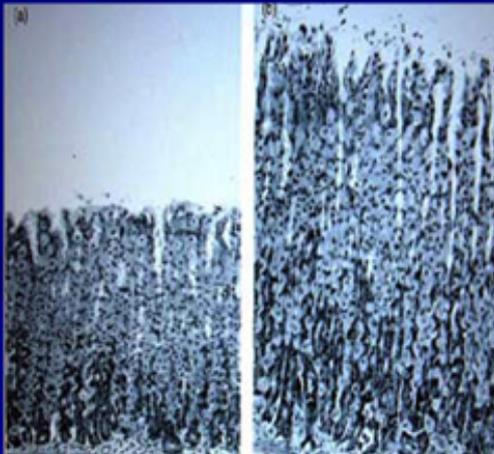
GM FOODS IN DIET SHOWS TOXIC REACTIONS IN THE DIGESTIVE TRACT

Monsanto's genetically modified Bt corn, engineered to kill the larvae of beetles, such as the corn rootworm, contains a gene copied from an insect-killing bacterium called *Bacillus thuringiensis*, or Bt.

Bt-toxin has been identified in the blood of both pregnant and non-pregnant women, as well as the umbilical blood of their babies. Researchers believe this can be explained by its presence in the normal diet.



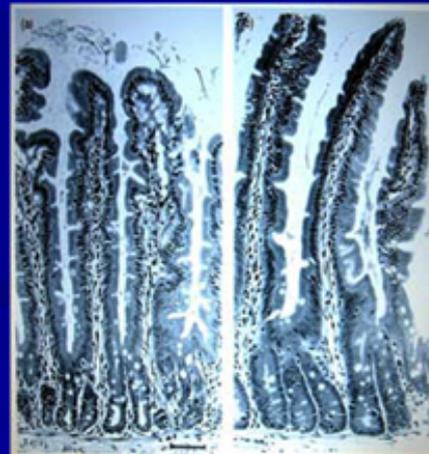
Stomach Lining



NON-GM

GM

Intestinal Wall



NON-GM

GM

Genetically engineered corn is present in the vast majority of all processed foods and drinks in the form of high fructose corn syrup, and you also ingest it when eating meat from animals fed Bt corn, which most livestock raised in confined animal feeding operations (CAFO, or so-called "factory farms") are.

Bt-crops may play a role in the rise in health problems such as gastrointestinal problems, autoimmune diseases, food allergies, and childhood learning disorders

GMO feed turns pig stomachs to mush! Shocking photos reveal severe damage caused by GM soy and corn

by Mike Adams, the Health Ranger – Natural News
Wednesday, June 12, 2013

If you have stomach problems or gastrointestinal problems, a new study led by Dr. Judy Carman may help explain why: pigs fed a diet of genetically engineered soy and corn showed a 267% increase in severe stomach inflammation compared to those fed non-GMO diets. In males, the difference was even more pronounced: a 400% increase.

The study is the first to show what appears to be a direct connection between the ingestion of GMO animal feed and measurable damage to the stomachs of those animals. Tests also showed abnormally high uterine weights of animals fed the GMO diets, raising further questions about the possibility of GMOs causing reproductive organ damage.

Humans have a similar gastrointestinal tract to pigs, and these GM crops are widely consumed by people, particularly in the USA, so it would be prudent to determine if the findings of this study are applicable to human

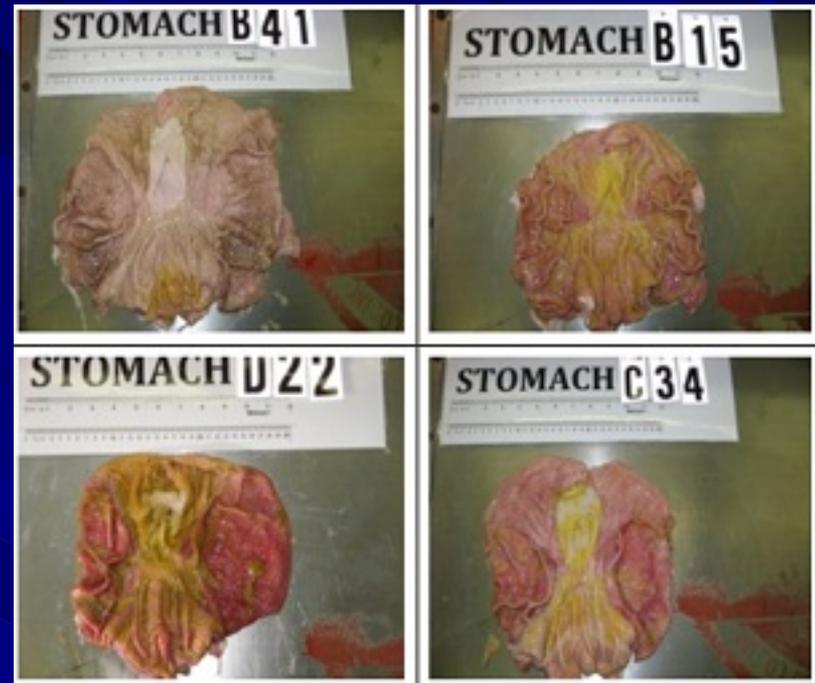


Figure 1. Different levels of stomach inflammation found (clockwise from top left: nil (from a non-GM fed pig, number B41), mild (from a non-GM-fed pig, number B15), moderate (from a GM-fed pig, number C34) and severe (from a GM-fed pig, number D22

Russia to Stop Import of U.S. Corn Linked to Cancer

A Russian consumer rights group said it would cease the purchase and import of American-grown, genetically modified (GM) corn after a study was released linking the GM food to breast cancer.



The study, conducted by researchers at the University of Caen in France, documented the effects of GM “Roundup-ready” corn on laboratory rats over a two-year period (the average life span of this rat species). **The results showed a significantly greater occurrence of breast cancer, kidney damage and liver damage in the test groups fed GM corn.**

Other test groups were given water containing trace amounts of Roundup or fed all-natural corn. **All groups that consumed Roundup showed elevated levels of cancer.**

Russia's Institute of Nutrition has contacted the European Union to for counsel, and the European Food Safety Authority is reviewing the study's validity. The research has already been peer reviewed.

Russia is not the first country to take issue with the GM corn. Genetically modified American corn is not sold in stores in the United Kingdom, though it is still used in feed for hens, pigs and cows.

Rats fed lifetime of GM corn grow horrifying tumors, 70% of females die early

by Mike Adams, the Health Ranger – Natural News
Wednesday, September 19, 2012



NaturalNews.com
Real News Powered by the People, Naturally.

Eating genetically modified corn (GM corn) and consuming trace levels of Monsanto's Roundup chemical fertilizer caused rats to develop horrifying tumors, widespread organ damage, and premature death, according to a recent study on **long-term effects** of consuming Monsanto's genetically modified corn.



Up to 50% of males and 70% of females suffered premature death.

Rats that drank trace amounts of Roundup (at levels legally allowed in the water supply) had a 200% to 300% increase in large tumors.

Rats fed GM corn and traces of Roundup suffered severe organ damage including liver damage and kidney damage.

The study fed these rats NK603, the Monsanto variety of GM corn that's grown across North America and widely fed to animals and humans. This is the same corn that's in your corn-based breakfast cereal, corn tortillas and corn snack chips.

http://www.naturalnews.com/037249_GMO_study_cancer_tumors_organ_damage.html

F = FOCUS

Focus refers to the power of the mind and the significant benefits of Mind-Body Medicine in our quest for health.

There are over 100 genes in the body that are activated by thoughts, feelings, and experiences.

We can have a dramatic and positive impact upon our health by simply taking control of our consciousness.

Understanding Your Mind, Mood, and Micronutrients: Treatment Studies

August 22, 2013

Hosted by Leslie Carol Botha

According to psychology researcher, Bonnie Kaplan, we are in a global mental illness epidemic. Indeed, scientists agree. In an article released July 24, 2013, Reuters reported that the rising costs of mental illness such as dementia, depression and addiction is overwhelming.

Estimated costs of brain disorders in Britain alone are at more than \$172 billion. Scientists are saying the same amount of funding is needed for research. Bonnie Kaplan will say that independent peer-reviewed research on broad spectrum micronutrient treatment for mental disorders has already been conducted, and published.

Kaplan will be joining host Leslie Carol Botha for a two-part series on 'Understanding Your Mind, Mood, and Micronutrients'. In the first interview Kaplan explained the social/historical context of how society got to this point. In the second interview she will focus on the empirical data (including treatment studies) that links mental illness to inadequate nutrient intake.



Oral folic acid and vitamin B-12 supplementation to prevent cognitive decline in community-dwelling older adults with depressive symptoms—the Beyond Ageing Project: a randomized controlled trial

Janine G Walker, Philip J Batterham, Andrew J Mackinnon, Anthony F Jorm, Ian Hickie, Michael Fenech, Marjan Kljakovic, Dimity Crisp, and Helen Christensen.

A randomized controlled trial (RCT) with a completely crossed $2 \times 2 \times 2$ factorial design comprising daily oral 400 μg FA + 100 μg vitamin B-12 supplementation (compared with placebo), physical activity promotion, and depression literacy with comparator control interventions for reducing depressive symptoms was conducted in 900 adults aged 60–74 y with elevated psychological distress (Kessler Distress 10–Scale; scores >15).

The 2-y intervention was delivered in 10 modules via mail with concurrent telephone tracking calls. Main outcome measures examined change in cognitive functioning at 12 and 24 mo by using the Telephone Interview for Cognitive Status–Modified (TICS-M) and the Brief Test of Adult Cognition by Telephone (processing speed); the Informant Questionnaire on Cognitive Decline in the Elderly was administered at 24 mo.

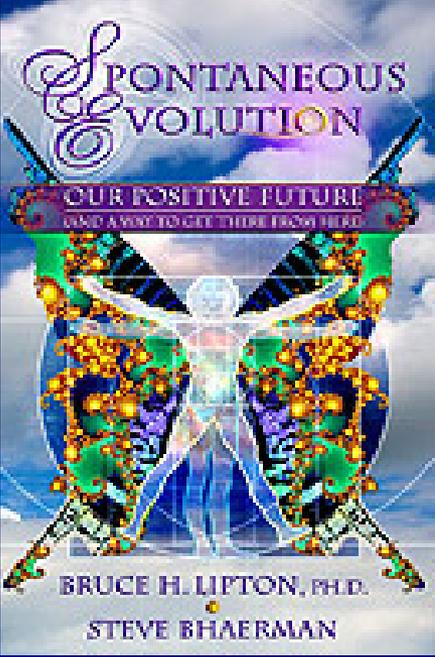
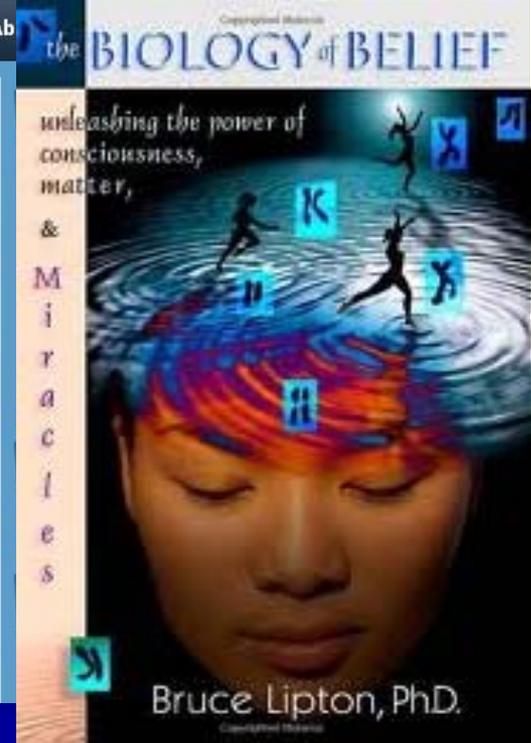
Conclusion: Long-term supplementation of daily oral 400 μg Folic Acid + 100 μg vitamin B-12 promotes improvement in cognitive functioning after 24 mo, particularly in immediate and delayed memory performance. This trial was registered at clinicaltrials.gov as [NCT00214682](https://clinicaltrials.gov/ct2/show/study/NCT00214682).

Bruce H Lipton, PhD

Uncovering The Biology of Belief

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The new sciences quantum physics and epigenetics are revolutionizing our understanding of the link between mind and matter. Biology of Belief shows that genes and DNA do not control our biology; that instead **DNA is controlled by signals from outside the cell, including the energetic messages emanating from our positive and negative thoughts.**

<http://www.bruce-lipton.com/>

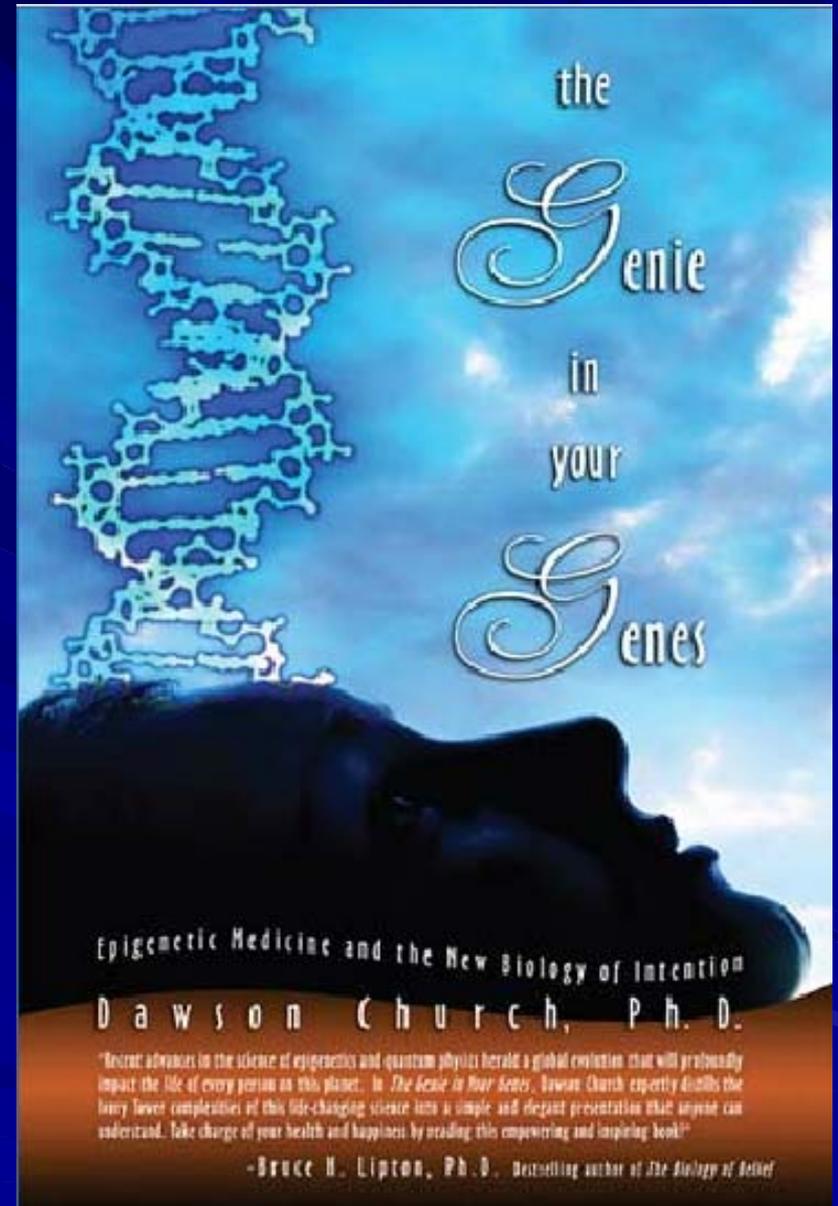
The Genie in Your Genes

Epigenetic Medicine and the New Biology of Intention

Is a breakthrough book linking consciousness to genetic change by Dawson Church, Ph.D.

For the first time, a single book summarizes a chain of remarkable scientific discoveries that in the new field of *Epigenetics* (*epi*=above, i.e. control *above* the level of the gene) that are the keys to healing.

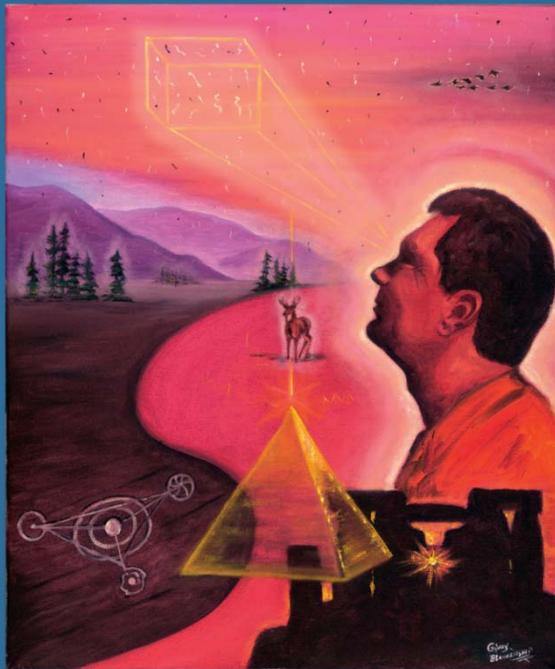
Citing hundreds of scientific studies, he shows how **beliefs and emotions can trigger the expression of specific DNA strands.**



Life Force – A scientific revolution in Energy Medicine!

LIFE FORCE, The Scientific Basis:

Breakthrough Physics of Energy Medicine,
Healing, Chi and Quantum Consciousness



Claude Swanson, Ph.D.
Volume II of *The Synchronized Universe Series*

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- Flat Biophoton Spectrum
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<http://synchronizeduniverse.com/>

Cellular communication – electrons and photons as messengers

Researchers have found that cells are in communication all the time. The DNA molecule, for example, radiates *and* absorbs in the millimeter wave band.

Can this be the source of the “Backster Effect”, of cell-to-cell communication?

Backster Effect – experiment postulating that plants can communicate with other lifeforms. By measuring the rate at which water rises from a philodendron's root into its leaves, using a polygraph to record altered electrical resistance signals from the plant taking up the water – surprisingly the graph tracing began to show a pattern typical of the response you get when you subject a human to emotional stimulation of short duration".

Now it has been proven that a “sick” cell radiates *something*, and when a healthy cell receives this radiation, it too becomes sick (Kaznachayev, 1967, 1981, 1982). The opposite also occurs, sick cells can be brought back to health with radiation from healthy cells.

Can this explain “energy healing”? Source, strength and intention of the energy being radiated?

(from p. 234 of *The Synchronized Universe* – Claude Swanson, PhD)

"The Inner Life of the Cell" (Molecular Dance)

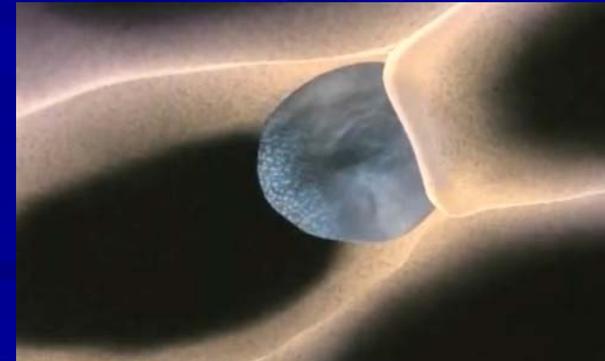
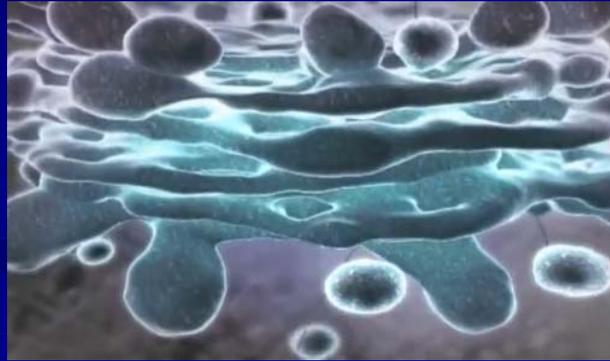
by Jeffrey Bland, PhD, FACN, CNS.



The Institute for Functional Medicine's 17th International Symposium on Functional Medicine opened with the talk "Looking Anew at Cancer".

...**emergent structures** of BOTH health and disease are locked into the potential of our genes and the cellular rhythm and dance of life are altered in response to different **environmental signals**.

Dr. Bland explains that **the cell is constantly in dynamic flux**; cellular signaling creates messages that induce changes internally and externally and **the cell is continually changing in response to its environment**.



I = INFECTIONS

Why is it that in today's society infections contribute to serious health problems such as autoimmune diseases, arteriosclerosis, and even cancer?

There is a clear correlation between infection and toxicity.

- Today, we have over 1000 times the amount of lead in our bones than our ancestors did just 400 years ago.
- Children are born already mercury toxic as a result of the toxicity of their mother.
- Toxins impair immune function and set the stage for chronic infections.

Let Them Eat Dirt

Early exposure to microbes shapes the mammalian immune system by subduing inflammatory T cells.

By Megan Scudellari | March 22, 2012



Maybe it's okay to let your toddler lick the swing set and kiss the dog. A new mouse **study suggests early exposure to microbes is essential for normal immune development**, supporting the so-called “hygiene hypothesis” which states that **lack of such exposure leads to an increased risk of autoimmune diseases.**

The finding, published (March 21) in *Science*, may help explain why there has been a rise in autoimmune diseases in sterile, antibiotic-saturated developed countries.

The mammalian immune system is dramatically influenced and shaped by exposure to microbes throughout life. **Epidemiological evidence suggests that early-life exposure to bacteria may be key in preventing two immune diseases: asthma and ulcerative colitis, a type of inflammatory bowel disease.**

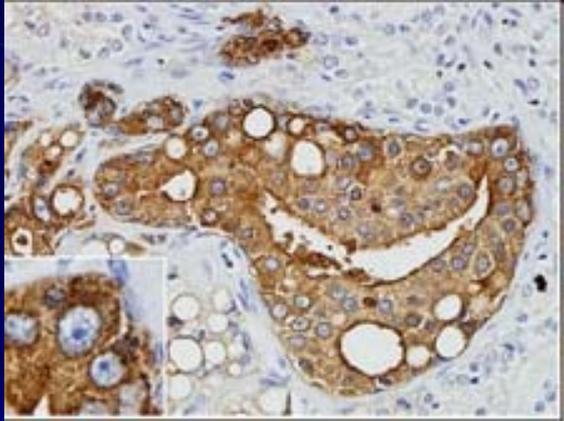
To explore that link, Dennis Kasper, Richard Blumberg, and colleagues at Harvard Medical School examined susceptibility to both diseases in germ free mice and normal lab mice. **“We were surprised to learn that germ free mice were extremely susceptible to both diseases,”** said Kasper, **“but normal, colonized mice were pretty resistant to both.”**

Infections Causing Human Cancer

By Harald zur Hausen.

Viral, bacterial, and parasitic diseases have accompanied humankind since the earliest times and have had more impact on history than any other single factor. Influenza and smallpox devastated cities and ruined empires, each claiming more lives worldwide than all wars combined. Plague killed approximately 200 million, and malaria still claims the life of a child every 30 seconds.

Other than the direct toll inflicted by infectious diseases, numerous studies have established a relationship between microorganisms and chronic conditions such as atherosclerosis, neurologic disorders, cancer, and obesity. The link between microorganisms and increasing numbers of diseases never before envisioned as having microbial etiology opens fascinating scientific, medical, and public health perspectives.



Human prostate cancer tissue. Brown, granular staining shows malignant epithelial cells that express XMRV proteins

Image: R. Schlaberg and H. M. Thaker

Viral cause for prostate cancer?

Prostate cancer is increasingly looking like an infectious disease, a new study shows, and may be sexually transmitted.

By Tabitha M. Powledge

[Published 7th September 2009 09:26 PM GMT]

Mounting evidence suggests that prostate cancer is an infectious disease caused by a recently identified virus. The latest report, published today (September 7) in the *Proceedings of the National Academy of Sciences*, found the virus was associated especially with aggressive prostate cancers and noted that "all individuals may be at risk" for infection.

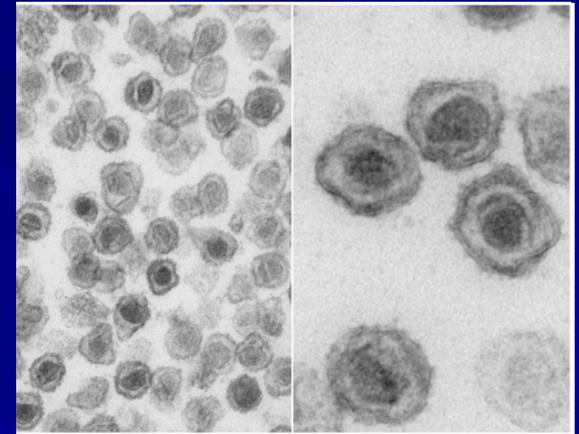
The notion that prostate cancer is an infectious disease like cervical cancer would not surprise most cancer experts, said Ila Singh of the University of Utah, the study's senior author. Almost 20% of visceral cancers are now proven infectious diseases, and there is a lot of indirect evidence from epidemiology and genetics that prostate cancer may be one of them.

The suspect is xenotropic murine leukemia-related virus (XMRV), a gammaretrovirus similar to viruses known to cause cancer in animals. Researchers at Columbia University and the University of Utah found the virus in more than a quarter of some 300 prostate cancer tissue samples, especially in malignant cells. That prostate cancer is a viral disease is not yet proven, but this is the third independent confirmation that XMRV infects prostate tissue.

XMRV Virus Linked to Chronic Fatigue

By AMY DOCKSER MARCUS

Researchers have linked an infectious virus known to cause cancer in animals to chronic-fatigue syndrome, a major discovery for sufferers of the condition and one that concerned scientists for its potential public-health implications.

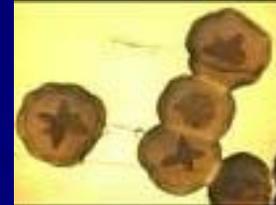
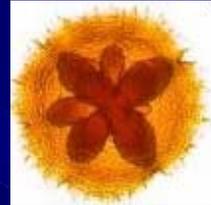
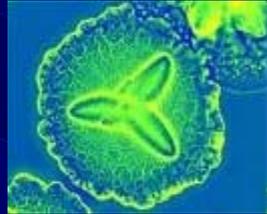


An estimated 17 million people world-wide suffer from chronic-fatigue syndrome, and the Centers for Disease Control and Prevention puts the U.S. figure at between one million and four million. CFS is characterized by debilitating fatigue and chronic pain, but there are no specific treatments, and the diagnosis is often made by ruling out other diseases. Thus there is disagreement in the medical community as to whether CFS is a distinct disease. A study showing a strong viral association with CFS could change that equation.

Researchers are just as concerned about the finding that nearly 4% of healthy people used as controls in the study were also infected with XMRV. If larger studies confirm these numbers, it could mean that as many as 10 million people in the U.S. and hundreds of millions of people around the world are infected with a virus that is already strongly associated with at least two diseases.

Oral Pathogen Linked to Periodontitis Boosts Heart Disease Risk

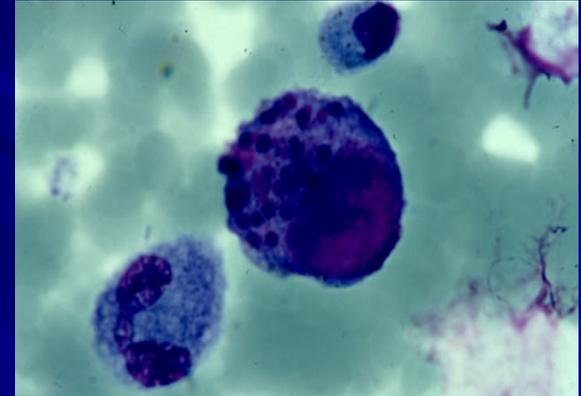
By Thomas S. May
June 12, 2007



HELSINKI, FINLAND -- -- *A. actinomycetemcomitans*, an oral pathogen that causes periodontitis, raises cardiovascular (CVD) risk by increasing blood serum levels of human heat-shock protein 60 (HSP60), researchers said here at the 76th Congress of the European Atherosclerosis Society (EAS).

Serum antibodies to human heat-shock protein 60 (HSP60) have been shown to be elevated in CVD, and *A. actinomycetemcomitans* may also contribute to elevated serum antibodies to human HSP60, according to lead investigator Susanna Paju, DDS, PhD, Research Associate in Oral Biology, Institute of Dentistry, University of Helsinki, Helsinki, Finland. "High serum antibodies to periodontal pathogens predict a risk for coronary heart disease and stroke, and our present study shows that they can also relate to elevated immune response against the host," Dr. Paju said.

Cytomegalovirus (CMV) May Cause High Blood Pressure



- A new study suggests for the first time that **cytomegalovirus (CMV)**, a common viral infection affecting between 60 and 99 percent of adults worldwide, is a cause of high blood pressure, a leading risk factor for heart disease, stroke and kidney disease.
- When coupled with other risk factors for heart disease, the virus can lead to the development of atherosclerosis, or hardening of the arteries.
- By the age of 40, most adults will have contracted the virus, though many will never exhibit symptoms. Once it has entered the body, CMV is usually there to stay, remaining latent until the immune system is compromised, when it then reemerges.
- This may eventually lead doctors to treating hypertension with anti-viral therapies or vaccines as part of the prescription.

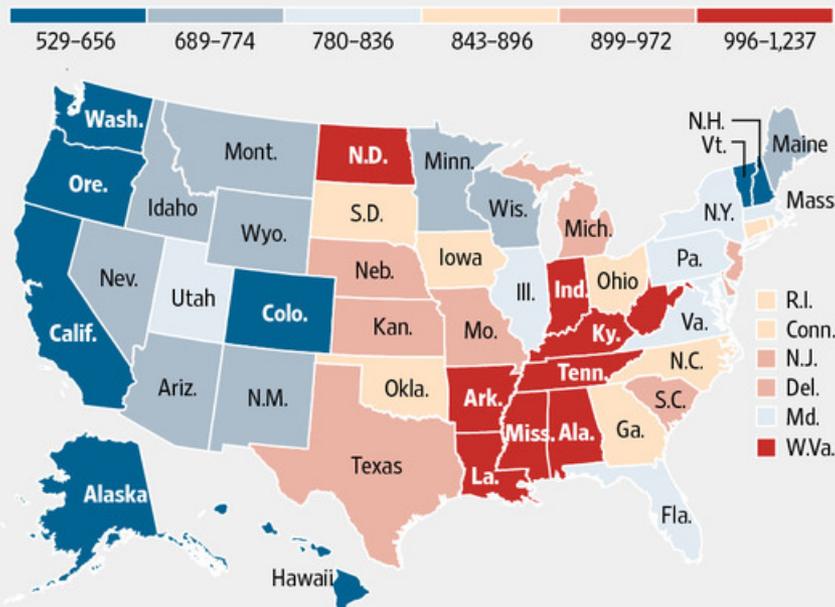
Persistent Resistance

The CDC has identified the most urgent drug-resistant organisms, as well as the areas of the country that prescribe the most antibiotics.

URGENCY LEVEL OF SELECT ANTIBIOTIC-RESISTANT BACTERIA

Urgent threats	Estimated annual impact	
	Infections	Deaths
Clostridium difficile	250,000	14,000
Drug-resistant Neisseria gonorrhoeae	246,000	<5
Carbapenem-resistant Enterobacteriaceae (CRE)	9,300	610

ANTIBIOTIC PRESCRIPTIONS PER 1,000 RESIDENTS, 2010



Source: Centers for Disease Control and Prevention

The Wall Street Journal

Antibiotics Losing Battle Against Bugs: Report

More than two million people in the U.S. develop infections every year that are resistant to antibiotics, and at least 23,000 of them die as a result

Serious threats

Drug-resistant Streptococcus pneumoniae	1,200,000	7,000
Drug-resistant Campylobacter	310,000	28
Drug-resistant Non-typhoidal Salmonella	100,000	40
Methicillin-resistant Staphylococcus aureus (MRSA)	80,000	11,000
Drug-resistant Shigella	27,000	<5
Extended spectrum β -lactamase producing Enterobacteriaceae (ESBLs)	26,000	1,700
Vancomycin-resistant Enterococcus (VRE)	20,000	1,300
Multidrug-resistant Acinetobacter	7,300	500
Multidrug-resistant Pseudomonas aeruginosa	6,700	440
Drug-resistant Salmonella Typhi	3,800	<5
Fluconazole-resistant Candida (a fungus)	3,400	220
Drug-resistant tuberculosis	1,042	50

Advanced Cellular Silver (ACS) 200

The leading evidence based silver prescribed today

ACS rapidly kills an enormous array of pathogenic microorganisms; literally oxidizing the cell wall of gram-positive and gram-negative bacteria, as well as killing naked virus, fungi and all without damaging human tissue.

ACS provides safe, effective and fast relief without harming beneficial gut flora.



A Broad-Spectrum Antimicrobial
ACS 200[®] achieves 99.99999% (complete) kill against *Borrelia burgdorferi*, *Candida albicans* and MRSA as proven via independently derived in vitro, kill-time studies.

Superior Silver
Powered by the same, patented silver technology as STERIPLEX[®] Ultra, now EPA-approved to kill anthrax spores.

Real Results
"I am absolutely thrilled with the results I personally received while using ACS 200[®]. It relieved my heart valve infection within 2 days when nothing else had."
—Dr. Loomis

Safety
ACS 200[®] has also been proven extremely safe in independent acute oral toxicity studies.

Independent Studies
Complete studies are available at www.resultsmresearch.com

Practitioner Endorsed
ACS 200[®] is prescribed by doctors in over 15 countries.

ACS 200[®] is significantly more effective in killing pathogens than competing brands. Compare the following laboratory kill-time studies.

Kill-time data compiled from individual manufacturer's published research.

B. burgdorferi	Titer	Log Reduction	Contact Time
ACS 200	3,400,000	5.95/99.99989%	< 8 minutes
ASAP Silver		No testing available	
Meso Silver		No testing available	

MRSA	Titer	Log Reduction	Contact Time
ACS 200	2,170,000,000	6.64/99.999984%	< 3 minutes
ASAP Silver	1,900,000	4.98/99.9989%	60 minutes
Meso Silver	1,200,000	Log not provided claimed kill	5 hours

S. aureus	Titer	Log Reduction	Contact Time
ACS 200	2,170,000,000	> 5.37/99.9996%	15 seconds
ASAP Silver	2,300,000	> 5.06/99.99914%	60 minutes
Meso Silver	830,000	Log not provided claimed kill	24 hours

C. albicans	Titer	Log Reduction	Contact Time
ACS 200	445,000,000	5.95/99.99989%	2 minutes
ASAP Silver	1,300,000	4.83/99.9985%	60 minutes
Meso Silver	12,000	Log not provided claimed kill	24 hours

G = GENETICS

Epi-genetics and methylation defects

Genetics can play a role in our ability to detoxify from numerous environmental insults. Optimizing methylation becomes a key strategy in improving the ability of the body to remove harmful substances that may have negative impacts on our health.

Epigenetics (“above genetics”) is the study of how environmental factors influence gene expression.

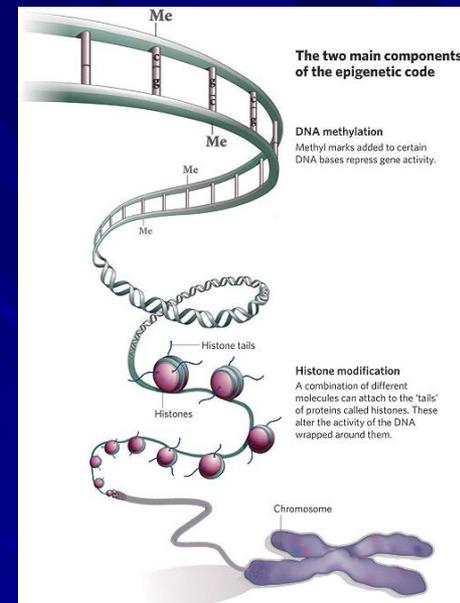
Toxic and Heavy Metal Exposure Early In Life May Promote Disease Later in Life Via Epigenetics

Metals and Neurotoxicology. 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)



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.

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

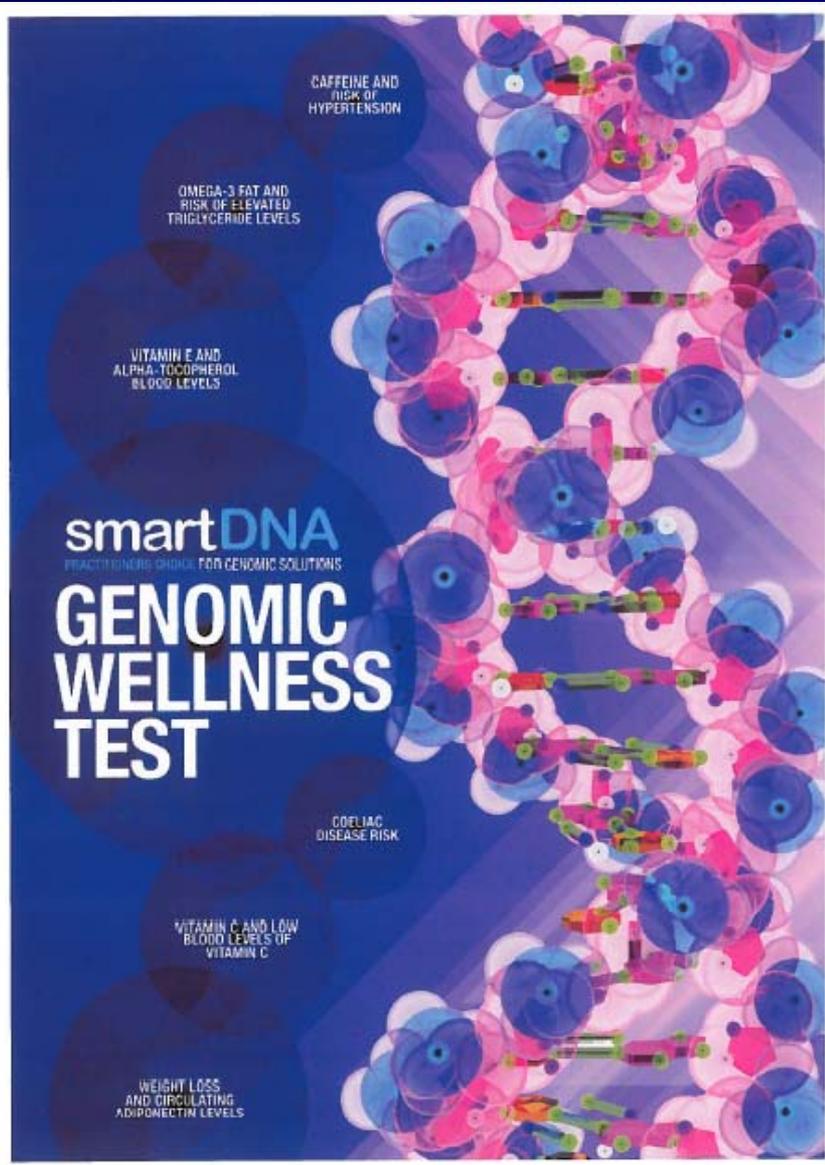
Test results and gene summary

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.



CAFFEINE AND RISK OF HYPERTENSION

OMEGA-3 FAT AND RISK OF ELEVATED TRIGLYCERIDE LEVELS

VITAMIN E AND ALPHA-TOCOPHEROL BLOOD LEVELS

smartDNA
PRACTITIONERS CHOICE FOR GENOMIC SOLUTIONS

GENOMIC WELLNESS TEST

COELIAC DISEASE RISK

VITAMIN C AND LOW BLOOD LEVELS OF VITAMIN C

WEIGHT LOSS AND CIRCULATING ADIPONECTIN LEVELS

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	<ul style="list-style-type: none"> 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		

Lipid Metabolism - HDL

Gene and SNP ID	Genotype / Haplotype	Result and Interpretation	Action steps and comments
PUFA Dietary Fat			
APOA1 rs970	AG	HIGHER HDL-C level in the blood. 	<ul style="list-style-type: none"> 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 individual's 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.	<ul style="list-style-type: none"> 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.
LPL rs328	CC		<ul style="list-style-type: none"> 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.
HDL-C level			
ABCA1 rs2230806	GG	LOWER HDL-C level in the blood. 	<ul style="list-style-type: none"> 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.
CETP rs5882	AG	HIGHER HDL-C level in the blood. 	<ul style="list-style-type: none"> 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.
CETP rs708272	AG		<ul style="list-style-type: none"> Review dietary fat intake. Lower dietary saturated fat intake will elevate HDL-C level.

Result and interpretation	Action steps and comments
<p>Physiogenomic</p> <p>LPL rs10098433 CC</p> <p>LIPC rs1800588 CT</p> <p>CETP rs1532624 AC</p>	<ul style="list-style-type: none"> Review the LPL, LIPC, and CETP haplotype in relation to increasing HDL-C and APOA1 levels via 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 compared to <8 METS per week. Exercise >8 METS per week is recommended to assist with elevating HDL-C and APOA1 level.

Lipid Metabolism - LDL

Gene and SNP ID	Genotype / Haplotype	Result and interpretation	Action steps and comments
LDL-C level			
APOB rs683	AG	INCREASED LDL-C in response to dietary saturated fat intake	<ul style="list-style-type: none"> 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 rs888	CT		

Lipid Metabolism - Triglycerides

Gene and SNP ID	Genotype / Haplotype	Result and interpretation	Action steps and comments
Triglyceride level			
APOCIII rs5126	CC	Not associated with high triglyceride level.	<ul style="list-style-type: none"> Stay balanced and focus on diet and lifestyle
APOA5 rs12286037	CT	INCREASED risk of hypertriglyceridemia	<ul style="list-style-type: none"> From this individual's 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.	<ul style="list-style-type: none"> 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 rs1799863	GG	NOT associated with high triglyceride level in response to low omega-3 intake.	<ul style="list-style-type: none"> 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 rs1796683	AG	Increased fat absorption.	<ul style="list-style-type: none"> Recommend reducing dietary fat Increased fat absorption may increase the risk of being overweight. Evaluate dietary saturated fat intake.

Type 2 Diabetes

Gene and SNP ID	Genotype / Haplotype	Result and Interpretation	Action steps and comments
Metabolic syndrome			
ACSL1 rs99977	GG	INCREASED metabolic syndrome (MetS) risk	<ul style="list-style-type: none"> Assess dietary fat intake and recommend either a low fat diet (< 35% energy) or a High PUFA diet (>5.5% energy)
ACC2 rs4766587	AA	INCREASED metabolic syndrome (MetS) risk	<ul style="list-style-type: none"> Assess dietary fat intake since MetS is positively impacted by a low fat diet <35% energy. Review n-6 PUFA in the diet since it has the greatest impact on MetS. Saturated and monounsaturated fat intake did not modulate MetS risk.
Glucose level			
G6PC2 rs590887	CC	LOWER fasting glucose level	<ul style="list-style-type: none"> Assessment of this individual's fasting plasma glucose and glycated haemoglobin A1C may be necessary. Review the portion size of carbohydrates in meals. Assess the intake of Low Glycaemic index carbohydrates in the diet. A very low carbohydrate is not necessary.
Insulin secretion			
TCF7L2 rs7903146	CC	NOT associated with decreased insulin secretion	<ul style="list-style-type: none"> The individual should stay balanced and maintain a healthy diet.
WFS1 rs10010131	AA		
Pancreatic Beta cell function			
SLC30A8 rs13266634	CT	DECREASED pancreatic beta cell function and impaired insulin secretion	<ul style="list-style-type: none"> Assessment of this individual's fasting plasma glucose and glycated haemoglobin A1C may be necessary. Review the portion size of carbohydrates in meals. Assess the intake of Low Glycaemic index and carbohydrates in the diet. A very low carbohydrate diet is not necessary.

Obesity risk

Gene and SNP ID	Genotype / Haplotype	Result and Interpretation	Action steps and comments
FTO rs9939609	AT	Associated with higher BMI	<ul style="list-style-type: none"> Assessment of this individual's fasting plasma glucose and glycated haemoglobin A1C may be necessary. Review the portion size of carbohydrates in meals. Assess the intake of Low Glycaemic index carbohydrates in the diet.
PPARG rs1801292	CC	Associated with higher BMI	<ul style="list-style-type: none"> Review dietary fat intake since individuals with this genotype consuming the highest quintile of total fat intake had a significantly higher BMI. MUFA intake was reported not to be associated with BMI for this genotype. In addition, the PUFA to saturated fat ratio does not affect body weight for individuals with this genotype. Review the portion size of carbohydrates in meals. Assess the intake of Low Glycaemic index carbohydrates in the diet. This does not mean a very low carb diet is necessary.

Co-enzyme Q10

Gene and SNP ID	Genotype / Haplotype	Result and Interpretation	Action steps and comments
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Co-enzyme Q10

NQO1 rs1800960	CT	Reduced NQO1 enzymatic activity.	<ul style="list-style-type: none"> Synthetic antioxidants and extracts of cruciferous vegetables are potent inducers of NQO1. The bioavailability of co-enzyme Q10 may be compromised since the conversion of co-enzyme Q10 to ubiquinol may be reduced. Individuals prescribed a statin drug may benefit from ubiquinol rather than co-Q10.
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Omega-3 and Omega 6

Gene and SNP ID	Genotype / Haplotype	Result and Interpretation	Action steps and comments
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Omega-3 and Omega-6

FADS1 rs174547	CT	Decreased blood levels of Arachidonic Acid and Eicosapentanoic Acid.	<ul style="list-style-type: none"> Review dietary omega-3 intake and omega-6 intake and improve the intake of omega-3 fatty acids if necessary. Consider measuring Fatty Acid status including the ratio of omega-3 to omega-6.
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Vitamins

Gene and SNP ID	Genotype / Haplotype	Result and Interpretation	Action steps and comments
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Vitamin B2

MTHFR rs1801133	CT	REDUCED impact of low blood levels of riboflavin on homocysteine level.	<ul style="list-style-type: none"> Recommend that the individual stays balanced and maintains a healthy diet.
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Vitamin B12

FUT2 rs602862	AG	LOWER levels of B12 in the blood.	<ul style="list-style-type: none"> 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.
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Vitamin C

SLC23A1 rs33972212	GG	Average blood levels of vitamin C.	<ul style="list-style-type: none"> Maintain a healthy diet and stay balanced by incorporating foods containing vitamin C, for example lemons, oranges, watermelons and strawberries.
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GSTT1	PRESENT	AVERAGE blood levels of vitamin C.	<ul style="list-style-type: none"> Individuals should maintain a healthy diet and stay balanced.
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GSTM1	PRESENT	AVERAGE blood levels of vitamin C.	<ul style="list-style-type: none"> Review dietary intake of vitamin C. Sources of vitamin C are lemons, oranges, watermelons and strawberries.
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Vitamin D

GC rs2282679	AA	HIGH RISK of vitamin D insufficiency.	<ul style="list-style-type: none"> This result does not mean that the individual's levels are out of balance.
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DHCR7 rs12785878	CT	HIGH RISK of vitamin D insufficiency.	<ul style="list-style-type: none"> Maintain a healthy diet with dietary sources of vitamin D such as cod liver oil, fish, eggs, mushrooms and fortified dairy products.
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CYP2R1 rs10741857	GG	HIGH RISK of vitamin D insufficiency.	<ul style="list-style-type: none"> Discuss the importance of sunshine exposure with the client and review their daily exposure to sunshine.
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Vitamin E

INTERGENIC	AC	INCREASED plasma	<ul style="list-style-type: none"> Maintain a healthy diet an
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PATIENT NAME: Dr Gary Gordon
 PATIENT DOB: 3-01-1935
 PATIENT SEX: Male

Gene and SNP ID	Genotype / Haplotype	Result and interpretation	Action steps and comments
rs12272004		levels of alpha-tocopherol.	incorporate foods containing naturally occurring sources of vitamin E such as eggs, nuts and leafy vegetables.

Methylation

Gene and SNP ID	Genotype / Haplotype	Result and interpretation	Action steps and comments
Methylation			
MTHFR rs1801133	CT	35%REDUCED MTHF Renzyme activity.	<ul style="list-style-type: none"> 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.
MTHFR rs1801131	AA		
Methylation co-factors			
MTR rs1805087	AA	HIGHER homocysteine level when B12 level is low.	<ul style="list-style-type: none"> Pathology testing may be necessary to assess the individual's B12 level. Review the MTHFR rs1801133 genotype. If it is "TT" then this variant has been reported to exert a greater effect in pregnant females.
MTRR rs1801394	GG	INCREASED risk of neural tube defects when vitamin B12 levels are low.	<ul style="list-style-type: none"> Review the MTHFR rs1801133 genotype. If it is "TT" then this variant has been reported to exert a greater effect in pregnant females. Pathology testing maybe necessary to measure the individuals B12 level.
TCN2 rs1801198	GG	LOWER holo-transcobalamin level and elevated homocysteine when vitamin B12 is low.	<ul style="list-style-type: none"> This result does not mean that the individual's levels are out of balance. Pathology testing maybe necessary to assess the individual's B12 level.
SLC19A1 rs4819130	TT	NOT associated with increased homocysteine level.	<ul style="list-style-type: none"> Individuals should maintain a healthy diet and stay balanced.

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

Choline Deficiency

Gene and SNP ID	Genotype / Haplotype	Result and interpretation	Action steps and comments
Choline			
MTHFD1 rs2236225	AG	Higher dietary choline requirements.	<ul style="list-style-type: none"> This result does not mean that the individual's levels are out of balance. Dietary sources of choline are eggs, cauliflower, almonds and peanut butter. Low dietary choline contributes to high homocysteine levels.

Oxidative stress

Gene and SNP ID	Genotype / Haplotype	Result and interpretation	Action steps and comments
Oxidative stress			
MnSOD rs4880	CT	REDUCED enzymatic activity.	<ul style="list-style-type: none"> Consider the results in relation to the individual's vitamin and mineral intake and/or dietary intake of antioxidant rich foods.
GPX1 rs1090499	CC	NORMAL enzyme activity.	<ul style="list-style-type: none"> Recommend that the individual stays balanced and maintains a healthy diet.
CAT rs1001179	AA	Reduced enzyme activity.	<ul style="list-style-type: none"> This genetic profile is particularly sensitive to antioxidant status, liberal consumption of dietary antioxidants and colourful vegetables and fruits are recommended.

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	AA	INCREASED risk for pro-carcinogen activation.	<ul style="list-style-type: none"> 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 estrogens.
CYP1A1_M1 rs464903	TT	NORMAL CYP1A1_M1 enzyme activity.	<ul style="list-style-type: none"> This enzyme can be promoted to remove hydrocarbons and accumulated estrogens which do not increase the risk of breast cancer. Nutrogenetic 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 rs4880	AG	REDUCED enzyme activity.	<ul style="list-style-type: none"> 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 rs1095	GG	Reduced GSTP1 enzyme activity.	<ul style="list-style-type: none"> 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.	<ul style="list-style-type: none"> 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.	<ul style="list-style-type: none"> Review exercise activities because it is important for maintaining good cardiovascular health.
Brain health			
KIBRA rs17070145	CT	INCREASED memory and cognitive flexibility.	<ul style="list-style-type: none"> 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.	<ul style="list-style-type: none"> 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			
TH1	CC	NORMAL	<ul style="list-style-type: none"> 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* 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 atherosclerosis. Please review the action steps and comments in relation to this result.
APOE rs7412	CC		

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 rs2230805	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?

The 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 rs5682	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		

Table 2: MEAN HDL-C (mg/dl) LEVELS PER COPY OF THE MINOR ALLELE AT SIGNIFICANT SNPs IN THE ENTIRE COHORT AND ACROSS MEDIAN LEVELS OF PHYSICAL ACTIVITY.

Gene rs number	MET – hours/week [Metabolic equivalent]	Number in each group	Mean (SD) HDL-C per allele, copy mg/dl		
			0 CC	1 CT	2 TT
LPL rs10096633	≤8.8	11,445	51.5	53.9	54.1
	>8.8	11,493	55.2	56.1	57.7
Delta [HDL-C & MET Physical activity]	N/A	N/A	3.7	2.2	3.6
LIPC rs1800588	≤8.8	11,445	51.3	53.0	54.4
	>8.8	11,491	54.4	56.8	59.3
Delta [HDL-C & Physical activity]	N/A	N/A	3.1	3.8	4.9
Gene rs number	MET – hours/week [Metabolic equivalent]	Number in each group	Mean (SD) HDL-C per allele, copy mg/dl		
			0 CC	1 CA	2 AA
CETP rs1532624	≤8.8	11,065	50.0	52.2	55.5
	>8.8	11,130	52.6	55.8	59.4
Delta [HDL-C & Physical activity]	N/A	N/A	2.6	3.6	3.9

Adapted from: Ahmad T et al. Physical Activity Modifies the Effect of LPL, LIPC and CETP polymorphisms on HDL-C levels and the Risk of Myocardial Infarction in Caucasian Women. *Circulation and Cardiovascular Genetics* 4(1), 74-80 (2011). The delta score in red refers to the mean increase in mg/dl for each genotype. For example LPL rs10096633 CC genotype indicates a 3.7 mg/dl increase in HDL-C when exercise is >8.8 METS.

Table 3: MEAN APOA1 (mg/dl) LEVELS PER COPY OF THE MINOR ALLELE AT SIGNIFICANT SNPs IN THE ENTIRE COHORT AND ACROSS MEDIAN LEVELS OF PHYSICAL

Gene rs number	MET – hours/week [Metabolic equivalent]	Number in each group	Mean (SD) HDL-C per allele, copy mg/dl		
			0 CC	1 CT	2 TT
LPL rs10096633	≤8.8	11,390	148.1	151.1	152.4
	>8.8	11,443	153.0	153.7	154.0
	Delta [HDL-C & MET Physical activity]	N/A	N/A	4.9	2.6
LIPC rs1800588	≤8.8	11,390	147.4	150.6	154.8
	>8.8	11,441	151.2	155.7	161.6
	Delta [HDL-C & Physical activity]	N/A	N/A	3.8	5.1
Gene rs number	MET – hours/week	Number in each group	Mean (SD) HDL-C per allele, copy mg/dl		
			0 CC	1 CA	2 AA
CETP rs1532624	≤8.8	11,065	145.9	149.3	152.9
	>8.8	11,130	149.2	153.8	158.1
	Delta [HDL-C & Physical activity]	N/A	N/A	3.3	4.5

Adapted From: Ahmad T et al. Physical Activity Modifies the Effect of LPL, LIPC and CETP polymorphisms on HDL-C levels and the Risk of Myocardial Infarction in Caucasian Women. *Circulation and Cardiovascular Genetics* 4(1), 74-80 (2011). The delta score in red refers to the increase in mg/dl for each genotype. For example the LPL rs10096633 CC genotype indicates a 4.9 mg/dl increase in mean ApoA1 level when exercise is >8.8 METS.

PATIENT NAME: Dr Garry Gordon
PATIENT DOB: 3-01-1996
PATIENT SEX: Male

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).

PATIENT NAME: Dr Gary Gordon
PATIENT DOB: 3-01-1995
PATIENT SEX: Male

ACC2 genetic test result

Gene and SNP ID	Genotype	Indicator	Result and Interpretation
ACC2 rs4788587	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 enzyme maintains ubiquinol (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 utilization 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.

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.

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 rs602652	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 the 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.

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.



Methylation

MTHFR genetic variations

The Methylene tetrahydrofolate 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 folacin. 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. This risk associated with polymorphisms in these genes is high homocysteine level and neural tube defect during pregnancy.



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 utilize 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 intermediates 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 GG 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 of 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.



Genetic
Engineering
& Biotechnology
News

GEN news highlights: Jan 21, 2011

Association between DNA Methylation and Obesity May Explain Related Chronic Diseases

A team of researchers at the Medical College of Georgia's Georgia Prevention Institute note a link between the presence of fat and chemical changes in DNA that may help explain the increased risk of chronic diseases in obese individuals. Specifically, they observed higher levels of methylation in a portion of the UBASH3A gene and lower levels in part of the TRIM3 gene.

The paper, "Obesity related methylation changes in DNA of peripheral blood leukocytes," is published online in *BMC Medicine*. The team observed that in comparison with the lean controls one CpG site in the UBASH3A gene showed higher methylation levels and one CpG site in the TRIM3 gene showed lower methylation levels in the obese cases... On the basis of those results they concluded that obesity is directly associated with methylation change in blood leukocyte DNA.

Both UBASH3A and TRIM3 genes are known to have roles in regulating the immune system, which is often dysregulated in obese individuals. The dysregulation of the genes can result in a level of chronic inflammation that contributes to diseases such as cardiovascular disease and diabetes. Increased methylation can further impact immune function by affecting gene-expression levels, which effects downstream functions of proteins produced by those genes.

<http://www.genengnews.com/gen-news-highlights/association-between-dna-methylation-and-obesity-may-explain-related-chronic-diseases/81244568/>

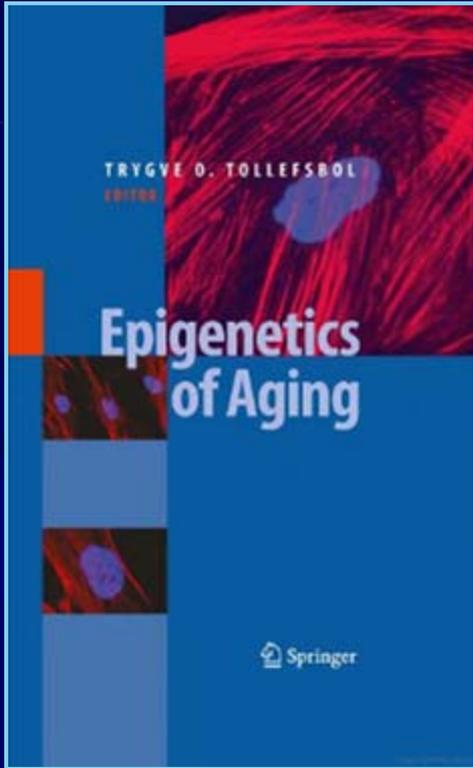


A pup of a different color

Supplementation of maternal diet with genistein and other compounds induced alterations in DNA methylation that were reflected in offspring coat color changes.
image: Randy Jirtle

Effects of nutrition on the epigenome of viable yellow agouti (Avy) mice. These female one year old Avy mice are isogenic. The mother of the mouse on the left ate a normal mouse diet while pregnant. In contrast, the mother of the mouse on the right ate a diet supplemented with methyl donors while pregnant [1]. The marked differences in the coat color and weight of these offspring resulted from a dissimilarity in the level of DNA methylation at the Agouti locus.





Epigenetics of Aging

2010, Part V, 315-326, DOI: 10.1007/978-1-4419-0639-7_17

DNA Methylation and Alzheimer's Disease

Thomas van Groen

Abstract

Epigenetics plays a direct and indirect role in the chances of developing Alzheimer's disease. The decreased DNA methylation status with increasing age of the amyloid precursor protein (APP) gene promoter will boost transcription of this gene, leading to higher levels of APP. Furthermore, both the BACE and PS1 genes show similar decreased promoter methylation with aging, causing higher levels – and activity – of β - and γ -secretases, increasing APP processing toward $A\beta$ production. Together, this increases the levels of $A\beta$ that will lead to the development of the pathology that is characteristic of sporadic AD.

Furthermore, **epigenetics plays a role through the nutritional status of the individual, i.e., through low folate and high homocysteine levels the DNA methylation level can be decreased. It is of interest to note that it has been shown that AD patients tend to have low levels of folate and high levels of homocysteine.** Finally, parental influences in the inheritance of AD have been demonstrated, likely caused by gene imprinting.

<http://itsnotmental.blogspot.com/2008/06/nutrition-genes-and-brain-dysfunctions.html>

Related to Cancer, Aging, Heart disease, Depression, Endothelial Dysfunction, Neuropathy...all involving 'sub-optimal' detoxification and supplementation of essential vitamins and minerals.

Nutrition, genes, and brain dysfunctions: Folate

Friday, June 6, 2008

Sometimes eating a healthy diet is not enough. In their search to answer why supplementing with folate helps some people with the brain symptoms of schizophrenia, NARSAD-funded research scientists are studying some people with schizophrenia who may have genetic defects that lower the amount of folate available in their bodies for their cells to use.

Folate, folic acid, Pteroylglutamic acid, are names for a water-soluble vitamin (B9) critical for the normal functioning of our cells, and for the development of neurons. We use folate, working synergistically with vitamin B12 (cobalamin) and vitamin C to help break down, use, and create new proteins that are then used to form red blood cells and help produce DNA.

Since folate is water-soluble, it is not stored in our bodies. Any excess is excreted out, so we need a daily fresh supply. Green leafy vegetables are rich in folate.

Folate is best known as the vitamin critical in pregnancy to prevent birth defects such as the neural tube defect—spina bifida, and to promote proper brain development. It also lowers homocysteine levels. High levels of homocysteine during pregnancy may increase the risk of the child developing brain disorders that may be later diagnosed as schizophrenia. [1, 2]

Poor diet may explain why some people with symptoms of schizophrenia are helped with folate supplementation, but their genes may explain why some people even with a healthy diet may need supplementation.

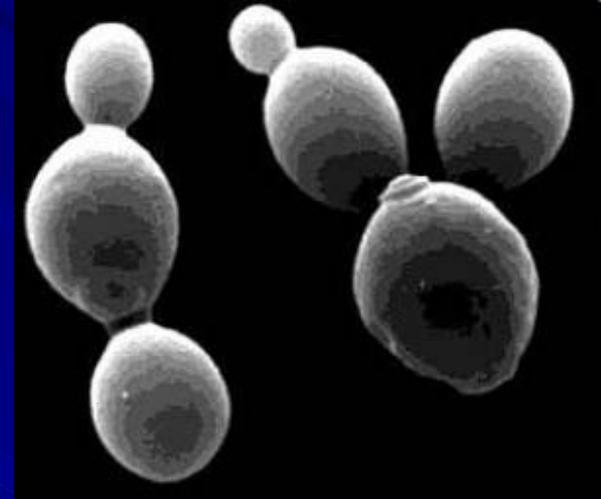
Good News In Our DNA: Defects You Can Fix With Vitamins And Minerals

ScienceDaily (June 3, 2008)

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."



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.

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

H = HEAVY METALS

Heavy metals are a major contributor to impaired health and are found in nearly everyone on the planet today.

People exposed to lead have developed cognitive difficulties. By age 80, 1 in 2 will develop Alzheimer's disease.

Could disturbed cognition and the loss of our ability to learn be directly related to heavy metal accumulation?

Made in China: Our Toxic, Imported Air Pollution

Mercury, sulfates, ozone, black carbon, flu-laced desert dust. Even as America tightens emission standards, the fast-growing economies of Asia are filling the air with hazardous components that circumnavigate the globe.

It is estimated that Asia is churning out 1,400 tons of Mercury emissions a year, and take as little as four days to reach North America.

Mercury plumes can wobble in latitude and altitude or park themselves in one spot for days on end. Emissions from China—and from the United States, and indeed from every industrial country—feed a network of air currents that, as equal-opportunity polluters, serve up toxic mercury around the world.

Commentary

Lead Poisoning-One Approach to a Problem That Won't Go Away

John D. Bogden,⁷ James M. Oleske,² Donald B. Louria¹

¹Department of Preventive Medicine and Community Health; and ²Department of Pediatrics, UMDNJ-New Jersey Medical School, Newark, NJ 07103-2714 USA

Patterson et al. (9) have compared current skeletal lead concentrations with those of Southwest American Indians who lived 700-1,000 years ago by use of museum samples. They found that the *present concentrations are about 500 to 1,000-fold greater than those of the museum samples, suggesting that current body lead burdens are about three orders of magnitude greater than those of our preindustrial ancestors*. Thus, it should not be surprising that adverse health effects have been associated with modestly increased bone lead stores in recent studies, including diminished academic achievement and aggressive behavior in children, and anemia, high blood pressure, and compromised renal function in adults (10-13).

Because lead is a ubiquitous and widespread contaminant, it will not be possible to eliminate additional environmental exposure of Americans of all ages. This inevitable exogenous exposure will be augmented by endogenous exposure as a result of past and ongoing bone lead accumulation. Dietary Calcium and Lead In the last 25 years, the blood lead concentration used to define poisoning or excessive exposure has fallen progressively from 40 to 30 to 25, and finally to 10 pg/dl (3).

Bone lead content assessed by L-line x-ray fluorescence in lead-exposed and non-lead-exposed suburban populations in the United States (blood lead/soil lead/lead-processing factories/pollution)

JOHN F. ROSEN*^t, ANNEMARIE F. CROCETTI*, KENNETH BALBI*, JULIE BALBI*, CHERYL BAILEY*, ISABELLA CLEMENTE*, NANCY REDKEY*, AND SARAH GRAINGER*

*Albert Einstein College of Medicine, Montefiore Medical Center, 111 E. 210th Street, Bronx, NY 10467; and ^tDepartment of Community Medicine, New York Medical College, Valhalla, NY 10595

Communicated by Clair Patterson, December 17, 1992

Current evidence linking release of bone Pb to blood is conclusive.

Studies of Pb workers, under conditions when there is a change in exposure, have demonstrated release of Pb from bone to blood. Blood Pb concentrations in retired workers are strongly influenced by bone Pb content; and two distinct kinetic compartments of Pb in bone have been described. These compartments have half-times of about 1 and 13 years, respectively. Significant contributions to blood Pb concentrations from bone stores have been documented.

Accumulated Lead Exposure and Risk of Age-Related Cataract in Men

Debra A. Schaumberg, ScD, MPH; Flavia Mendes, MD; Mini Balaram, MD; M. Reza Dana, MD, MPH; David Sparrow, DSc; Howard Hu, MD, MPH, ScD
JAMA. 2004;292:2750-2754.

Context Low-level lead exposure may increase the risk for a number of chronic age-related diseases. Several studies have documented the presence of lead in lenses with cataract. The intrusion of lead into the lens may alter lens redox status and cause protein conformational changes that decrease lens transparency.

Objective To determine the relationship of cumulative lead exposure with the development of cataract.

Design, Setting, and Participants Tibial (cortical) and patellar (trabecular) bone lead levels were measured by K x-ray fluorescence between 1991 and 1999 in a subset of participants in the Normative Aging Study (NAS), a Boston-based

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Dr. Gordon comments: Finally proof that bone lead levels are adversely affecting the health of our brain, as the eye is an extension of the brain. Therefore immune suppressing is occurring.

Results The mean age of the study participants was 69 years and cataract was identified in 122 men. The age-adjusted OR (95% CI) for cataract for men in the highest vs lowest quintile of tibia lead level was 2.68 (1.31-5.50). Further adjustment for pack-years of cigarette smoking, diabetes, blood lead levels, and intake of vitamin C, vitamin E, and carotenoids resulted in an OR of 3.19 (95% CI, 1.48-6.90). For patella lead level, there was an increased risk of cataract in the highest vs lowest quintile (OR, 1.88; 95% CI, 0.88-4.02), but the trend was not significant ($P = .16$). Blood lead levels, more indicative of short-term exposure levels, were not significantly associated with cataract (OR, 0.89; 95% CI, 0.46-1.72; $P = .73$).

Conclusions These epidemiological data suggest that accumulated lead exposure, such as that commonly experienced by adults in the United States, may be an important unrecognized risk factor for cataract. This research suggests that reduction of lead exposure could help decrease the global burden of cataract.

Men with high levels of bone lead 6 times more likely to die from heart disease

September 10th, 2009

WASHINGTON - Men with high levels of lead in bones are six times more likely to die from heart disease, according to a new study.

Researchers from the Harvard School of Public Health (HSPH) and the University of Michigan School of Public Health found that bone lead was associated with a higher risk of death from all causes, particularly from cardiovascular disease.

“The findings with bone lead are dramatic,” said Marc Weisskopf, assistant professor of environmental and occupational epidemiology at HSPH and lead author of the study.

The results showed that the risk of death from cardiovascular disease was almost six times higher in men with the highest levels of bone lead compared to men with the lowest levels.

The risk of death from all causes was 2.5 times higher in men with the highest levels of lead compared to those with the lowest levels.

Given that bone lead may be a better biomarker of cumulative lead exposure than blood lead, it may be the best predictor of chronic disease from exposure to lead in the environment.

The study appears in journal Circulation. (ANI)

NO SAFE LEVEL OF LEAD!

Blood lead levels and mortality

Archives of Internal Medicine (AMA Official Journal)

2002 Nov 25;162(21):2443-9

Lustberg M, Silbergeld E.

Department of Epidemiology and Preventive Medicine, University of Maryland

Despite declines in blood lead levels during the past 20 years, lead exposure continues to be a public health concern. Studies have linked lead exposure with increased risk for diverse health outcomes. Few studies have evaluated the association of lead exposure and mortality in the general population. METHODS: To evaluate the association of lead exposure and mortality in the United States, we used the recently released mortality follow-up data for participants of the Second National Health and Nutrition Examination Survey, a national cross-sectional survey of the general population conducted from 1976 to 1980. Survey participants aged 30 to 74 years with blood lead measurements were followed up through December 31, 1992 (n = 4292). **RESULTS:** After adjustment for potential confounders, individuals with baseline blood lead levels of 20 to 29 microg/dL (1.0-1.4 micromol/L) had 46% increased all-cause mortality (RR, 1.46; 95% confidence interval [CI], 1.14-1.86), 39% increased circulatory mortality (RR, 1.39; 95% CI, 1.01-1.91), and 68% increased cancer mortality (RR, 1.68; 95% CI, 1.02-2.78) compared with those with blood lead levels of less than 10 microg/dL (<0.5 micromol/L).

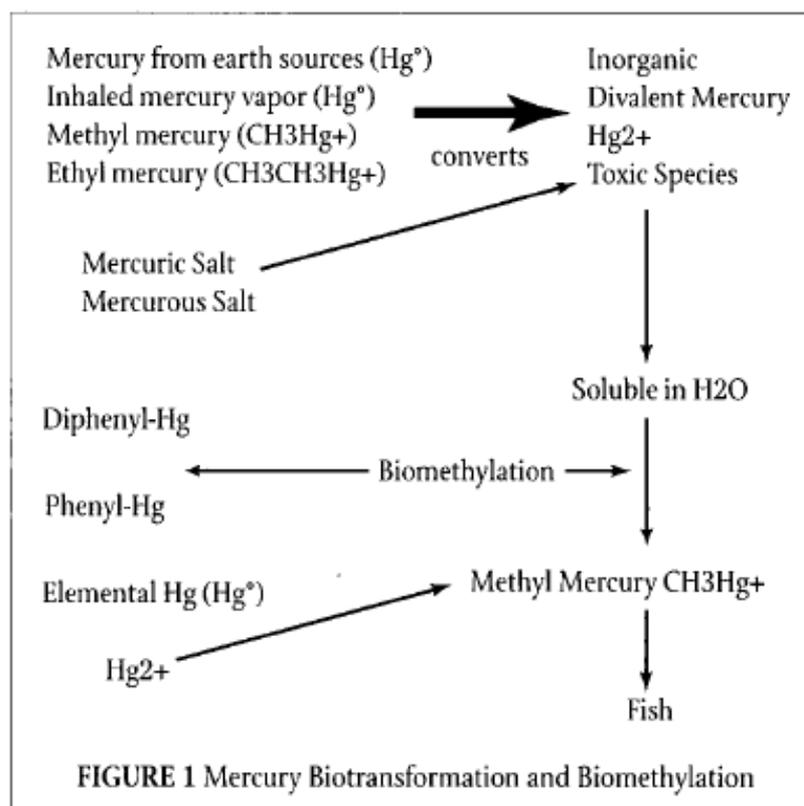
The Role of Mercury and Cadmium Heavy Metals in Vascular Disease, Hypertension, Coronary Heart Disease, and Myocardial Infarction

Mark C. Houston, MD, MS, FACP, FAHA

Mark Houston, MD, MS, FACP, FAHA, is editor-in-chief for the *Journal of the American Nutraceutical Association* and has published more than 70 clinical research studies and authored 3 books. He is clinical professor of medicine, Vanderbilt University School of Medicine; and director of the Hypertension Institute, Vascular Biology and the Life Extension Institute, Saint Thomas Hospital in Nashville, Tenn, where he also serves as medical director of Clinical Research and section chief of the Division of Nutrition.

INTRODUCTION

There is increasing concern regarding the overall health effects of exposure to various heavy metals in the environment. This is particularly true of mercury and less so with cadmium, lead, aluminum, and arsenic. The cardiovascular consequences of mercury and cadmium toxicity have not been carefully evaluated until recently. This paper will critically review the vascular consequences of mercury and cadmium toxicity in humans as it relates to hypertension, generalized atherosclerosis, coronary heart disease (CHD), myocardial infarction (MI), cerebrovascular accidents (CVA), carotid artery disease, renal dysfunction, and total mortality.



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 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.^{6,8,12}

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.¹⁸

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.¹³⁻¹⁷ 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.²⁸

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.²⁹ Improved nitric oxide function is another reason for the predictable improvement in blood flow seen with all noninvasive measurements, including segmental blood pressures, thermography, plethysmography, 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.

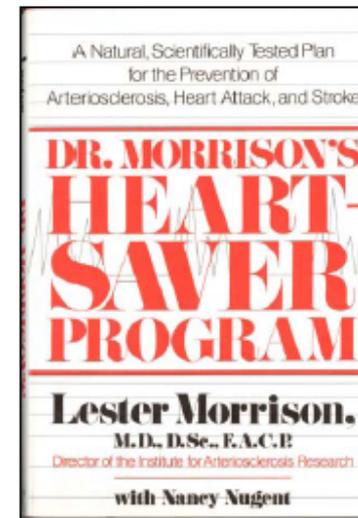
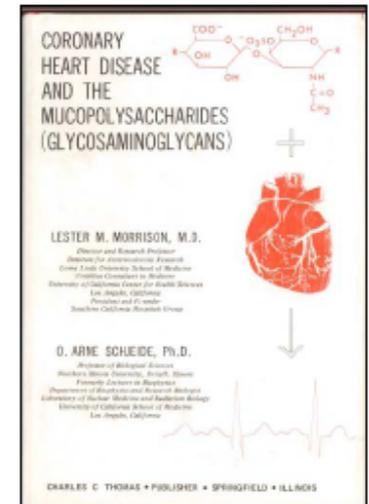
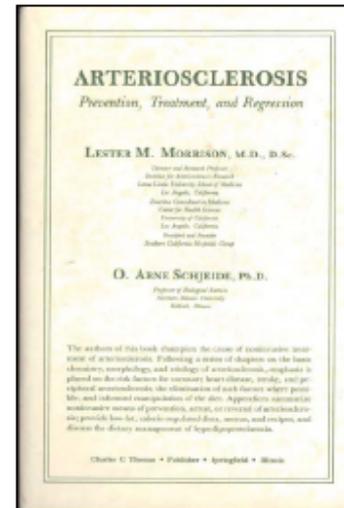
He found a combination of several nutrients that act synergistically with his special mucopolysaccharides to help reverse arteriosclerosis and stop heart attacks.

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)

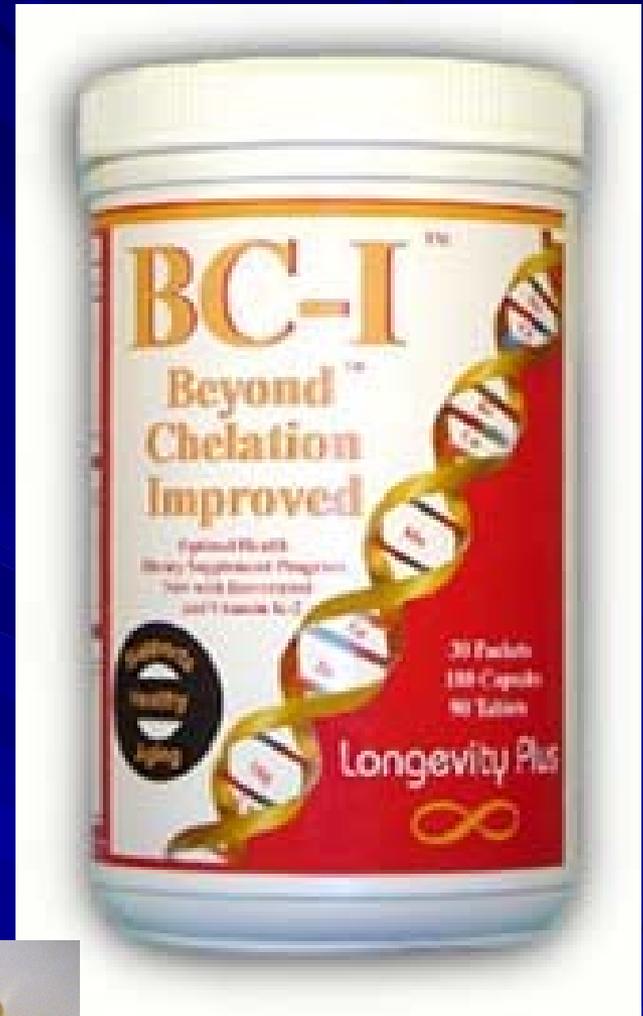
Books by Lester Morrison, MD, D.Sc., F.A.C.P.



Beyond Chelation Improved (BC-I)

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

- 3 *Beyond Any Multiple*™ caplets
- 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 arteriosclerosis 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.

PubMed.gov

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

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 Ca^{2+} is an integral constituent of viruses but Mg^{2+} 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]

<http://www.ncbi.nlm.nih.gov/pubmed/6816193>

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.

H = HORMONES

A hormone is a messenger molecule that works with receptors on cell membranes to tell a cell what to do.

They optimize our health potential by providing the body with the hormonal support it requires to perform at high levels of functioning.

Additionally, cholesterol is a precursor to hormone production. Without adequate levels of cholesterol, hormones are not produced.



Correcting Hormone Imbalance with Detoxification

Lyn Hanshew, MD

6/11/09

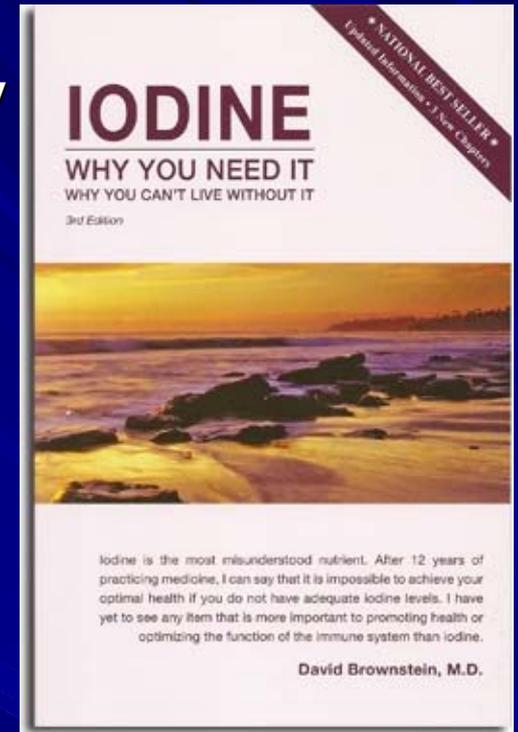
Environmental toxins such as heavy metals, pesticides, herbicides and volatile organic compounds are more pervasive than ever. From contaminated air and food, to pharmaceutical byproducts in water supplies, as our toxic exposure increases, so does our bio-accumulation of these same toxins. The body has limited ability to metabolize, mobilize and excrete these poisons. Stored toxins negatively impact the neurological, immune and endocrine systems and as significant damage is done, we develop symptoms and disease related to these impaired systems.

Let's examine symptoms related to a toxically impaired endocrine system. Hormones are messenger molecules that interact with receptors on the cell membranes to instruct the cell as to what to do. Common symptoms/diseases of deficient endocrine function include: Obesity, Diabetes, Hypercholesterolemia, Hyper or Hypo glandular function, Infertility, Fatigue, Chronic Fatigue, Fibromyalgia, Sexual dysfunction, Decreased libido, Impaired memory, Mood disorder, Sleep disturbance, Decreased cognitive function, Decreased cardiac function, Decrease muscle mass, Decreased bone mass, Osteopenia, Constipation, Cold hands/cold feet, and more. In conventional allopathic medicine, a patient is told that the "symptom" she is experiencing (such as one listed previously) is the "problem", and "Oh, have I got a pharmaceutical drug for you!" Pharmaceutical drugs do not correct the problem of poisoned endocrine pathways.

Specifically related to hormone production and regulation, Mercury and other toxins prevent the conversion of Free T4 (inactive) to Free T3 (active). The enzyme required for this conversion is the 5'-deiodinase enzyme. This enzyme is inactivated by Mercury, Arsenic, Cadmium and Lead.

■ Dr. David Brownstein, author of the book “Iodine, Why You Need It, Why You Can’t Live Without It”, states that “Approximately 1.5 billion people, about one-third of the earth’s population, live in an area of iodine deficiency as defined by the World Health Organization”.

■ Even though iodine is added to the salt supply, which can help prevent conditions such as goiter, it [iodized salt] is inadequate to prevent an iodine deficiency.

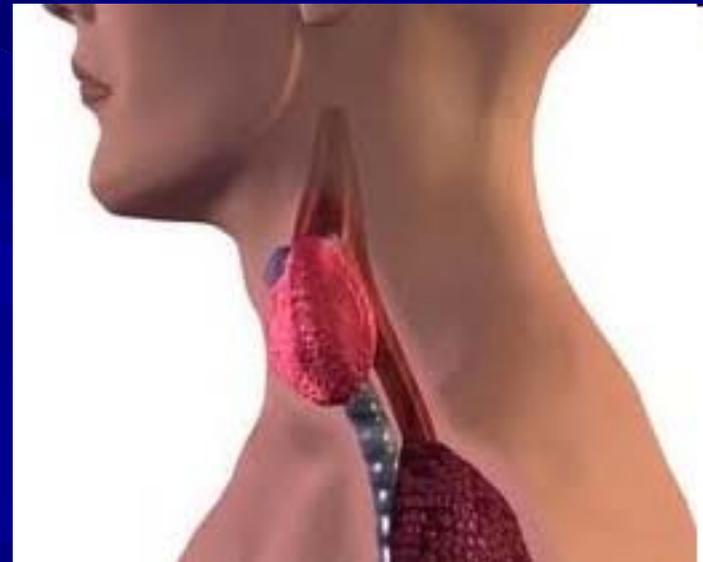


David Brownstein, M.D. is a Board-Certified family physician and is one of the foremost practitioners of holistic medicine. He is the Medical Director of the Center for Holistic Medicine in West Bloomfield, MI. Dr. Brownstein has lectured internationally to physicians and others about his success in using natural hormones and nutritional therapies in his practice.

Hypothyroidism – As the body's iodine levels fall, hypothyroidism may develop, since iodine is essential for making thyroid hormone.

Common symptoms of problem with thyroid due to low thyroid or hypothyroidism are:

- Fatigue and weakness
- Low basal temperature (cold intolerance)
- Dry and coarse skin
- Hair loss
- Cold hands and feet
- Weight gain
- Insomnia
- Constipation
- Depression
- Poor memory, forgetfulness, dementia
- Nervousness and tremors
- Immune system problems
- Heavy menstrual periods



From *International Journal of Impotence Research*

Are Declining Testosterone Levels A Major Risk Factor for Ill-Health in Aging Men? B. B. Yeap - 04/08/2009

As men grow older, testosterone levels fall, with a steeper decline in unbound or free testosterone compared with total testosterone concentrations. Lower testosterone levels have been associated with poorer cognitive function, and with impaired general and sexual health in aging men. Recently, lower testosterone levels have been linked with metabolic syndrome and type II diabetes, both conditions associated with cardiovascular disease, and shown to predict higher overall and cardiovascular-related mortality in middle-aged and older men.

However, reverse causation has to be considered, as systemic illness may result in reduced testosterone levels. Thus, the strength of these associations and the likely direction of causation need to be carefully considered. Furthermore, these conditions may overlap, for example aging, lower testosterone levels, erectile dysfunction and cardiovascular disease are interrelated.

Middle-Aged Men, Too, Can Blame Estrogen for That Waistline

By GINA KOLATA

Published: September 11, 2013

It is the scourge of many a middle-aged man: he starts getting a pot belly, using lighter weights at the gym and somehow just doesn't have the sexual desire of his younger years.

[Enlarge This Image](#)



Nathan Weber for The New York Times

Ben Iverson took part in a study of men aged 20 to 50 who agreed to have their testosterone production turned off for 16 weeks. The study is being repeated to measure vitality in older men.

The obvious culprit is testosterone, since men gradually make less of the male sex hormone as years go by. But a surprising new answer is emerging, one that doctors say could reinvigorate the study of how men's bodies age. Estrogen, the female sex hormone, turns out to play a much bigger role in men's bodies than previously thought, and falling levels contribute to their expanding waistlines just as they do in women's.

The New York Times

The new frontier of research involves figuring out which hormone does what in men, and how body functions are affected at different hormone levels.

While dwindling testosterone levels are to blame for middle-aged men's smaller muscles, falling levels of estrogen regulate fat accumulation, according to a study published Wednesday in The New England Journal of Medicine.

Estrogen is a major factor in male midlife woes. And both hormones are needed for libido.

Ensure Safe, Effective Bio-Identical Hormone Replacement: Select the Right Hormone Test for Your Patient.

By Lara Pizzorno, MDiv, MA, LMT.
Managing Editor, Longevity Medicine Review

Treating the sequelae of the age-and stress related decline in adult hormones with bio-identical hormone replacement (BHRT) can restore more youthful hormone levels and significantly alleviate symptoms associated with “normal” aging, optimizing health, happiness and quality of life. Successful and safe BHRT, however, necessitates laboratory testing to assess the patient’s current hormonal status, monitor treatment, and ensure that hormones are being metabolized in ways that reduce risks for cancer, cardiovascular disease, osteoporosis, other age-related diseases and declines in cognitive and sexual function.

24-Hour Urine Comprehensive Profile: Analytes & Reference Ranges for Women		
Analyte	Adult Reference Range*	
Creatinine	0.5 – 2.0 gm/24hr	
Total urine volume	1,200 – 3,000 mL	
Steroid		
	Amount excreted in µg/24 hr	
Sex Hormones	Luteal (Days 17-26)	Postmenopausal
Estrone (E1)	3.3 - 44.6	1.0 - 7.0
Estradiol (E2)	1.4 - 12.2	0 - 4
Estril (E3)	6.1 - 32.4	0 - 30
Total Estrogens	10.8 - 89.2	0 - 41
Estrogen Quotient (E3 / E1 + E2)	>1.0	
2-OH Estrone (Phase I metabolite)	3.8 - 38.1	0.2 - 5.4
16α-OH Estrone (Phase I metabolite)	2.1 - 7.9	0.15 - 3.5
2/16α Ratio (Ideal = 2 - 4)	1.8 - 5.5	0.6 - 5.0
4-OH Estrone (Phase I metabolite)	0.8 - 5.9	0.05 - 1.1
2-methoxyestrone (Phase II metabolite)	2.2 - 14.4	0.3 - 4.1
2-methoxyestradiol (Phase II metabolite)	0.2 - 2.2	0.03 - 0.54
Pregnanediol (progesterone metabolite)	1450 - 6140	200 - 1000
DHEA	100 - 2000	
Androsterone (DHEA metabolite)	500 - 3200	
Etiocholanolone (DHEA metabolite)	500 - 5000	
Testosterone	5.0 - 35.0	
5α-Androstanediol	3.0 - 35.0	
5β-Androstanediol	13.0 - 180.0	
Glucocorticoids		
Pregnanetriol (want ≥mid-range, indicator of substrate availability for cortisol pathway)	100 - 1500	
Cortisone (optimal range ~120-130 µg/24hr)	31 - 209	
Cortisol (optimal range ~90 µg/24hr)	30 - 170	
Cortisone: Cortisol Ratio (always interconverting)	Ideal = 0.7	
Tetrahydrocortisone (THE, cortisone metabolite)	1700 - 4200	
Allo-tetrahydrocortisol (5α-THF, cortisol metabolite)	400 - 2100	
Tetrahydrocortisol (THF, cortisol metabolite)	900 - 2600	
(THE + 5α-THF + THF) x2 = daily cortisol output	5000 - 7000	
11β-OH Androsterone (terminal metabolite of cortisol; low value confirms insufficiency)		
11β-OH Etiocholanolone (terminal metabolite of cortisol; low value confirms insufficiency)		

Hormones can be assayed using saliva, blood (serum), and urine. Each testing method has advantages and disadvantages. Which of the three hormone test methods, or which combination of tests, you will wish to utilize will depend upon what information you need in a given clinical situation. www.lmreview.com

The Wall Street Journal - Health Industry

U.S. Cell-Aging Researchers Awarded Nobel

Three Scientists Share Prize for Discovery of Enzyme That Has Opened New Avenue of Research Into Several Serious Diseases

by Gautam Naik – Oct 9, 2009

The scientists discovered the workings of telomerase, an enzyme that produces tiny units of DNA that seal off the tips of chromosomes. These DNA units, known as telomeres, act like the plastic caps at the ends of a shoelace, keeping the chromosomes from fraying and the genes inside them from unraveling.

As we age, though, these caps lose their ability to protect. One result is that some cells go into a state known as senescence, where they are alive but stop dividing. Researchers believe this may contribute to aging.

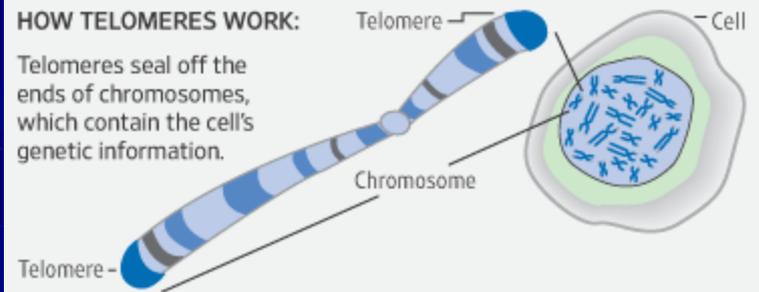
Their findings have sparked a new line of research into possible treatments for age-related maladies, such as cancer, blindness and cardiovascular disease.

Unraveling a Cellular Mystery

The Nobel Prize went to scientists who described the workings of the telomere, which helps protect cells from age-related damage.

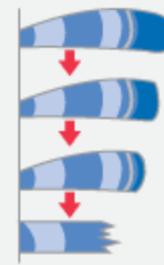
HOW TELOMERES WORK:

Telomeres seal off the ends of chromosomes, which contain the cell's genetic information.

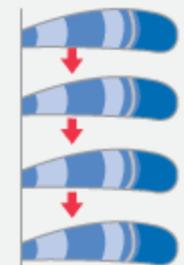
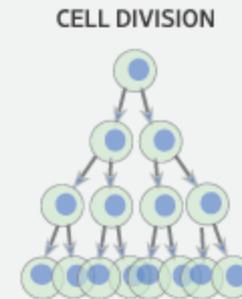


As cells replicate, telomeres can fray, allowing chromosomes to become damaged. This becomes increasingly common with age.

Scientists discovered a protein called telomerase, which maintains the telomeres, keeping chromosomes intact longer.



Chromosome tips without telomerase



Chromosome tips with telomerase

Source: The Nobel Committee for Physiology or Medicine, illustrations by Annika Röhl

<http://online.wsj.com/article/SB125472880070963893.html>

About TA-65[®]

TA-65[®] is a proven telomerase activator* that was originally discovered and patented by California biotech company Geron. Our clients take TA-65 capsules as part of a daily health regiment. TA-65 turns on the hTERT gene* which activates the enzyme telomerase which can lengthen your telomeres. You can be tested to measure your telomeres before, during and after taking TA-65 to show actual changes in telomere length.

How is TA-65[®] made?

TA-65 is a naturally occurring single molecule found in the small molecule from a medicinal plant. T.A. Sciences has developed a proprietary process to refine and purify TA-65. Our process begins with tons of plant material harvested from selected farms in one small region in China. In our plant extraction facility, the raw small molecule from a medicinal plant root is chopped up and refined. After initial extraction, the base ingredient is further purified and then sent to an outside government testing facility where it is tested for purity, heavy metals, and pesticides. The product is then sent to a FDA certified, state-of-the-art, laboratory for final purification that ends up with 90+% pure

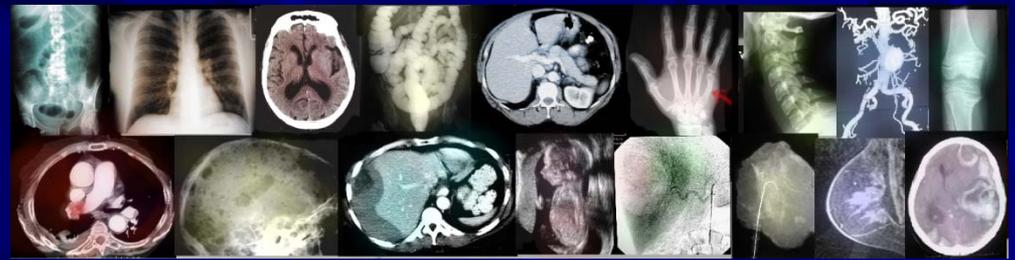
TA-65 Dosing Guideline

The statistics showing TA-65's efficacy in the ground breaking scientific paper published Sept. 8, 2010 in the peer-reviewed scientific journal *Rejuvenation Research* allows us to offer different dosing options. Below is the guideline for you to choose the appropriate dosage and price for your unique situation:

1. **250 units (1 capsule daily)** is efficacious for healthy adults in their 40's or 50's. Also 250 units can serve as a maintenance dose for older people who have been taking higher doses of TA-65 for several years and want to continue on a reduced cost program. Clients who took this dose were shown to have increased short telomere length and significantly improved immune system function. There are also anecdotal reports of increased endurance and other benefits. **Cost: US \$600.00 for each 3 month segment.**
2. **500 units (2 capsules daily)** has been proven to lengthen short telomeres, restore the immune system, and improve other important bio markers. Anecdotal reports included increased energy, endurance, vision improvements, sexual enhancement, and more. This medium strength dose is recommended for people who are generally in good health and want to be proactive in longevity and healthy aging. Many people in their 50's or 60's fall into this category. **Cost: US \$1,200.00 for each 3 month segment.**

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

By Jeffrey Dach, MD



By now, it 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 telomerase 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-1 α* 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-1 α* and *PGC-1 β* 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.

Aging Ills Reversed in Mice

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

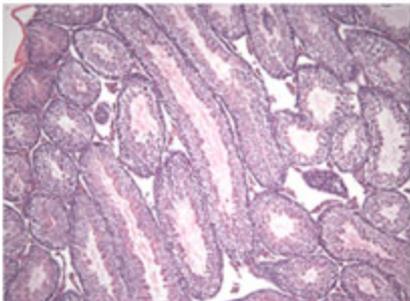
The research team led by Dr. Ronald DePinho of Dana Farber Cancer Institute made genetically engineered mice that aged prematurely.

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

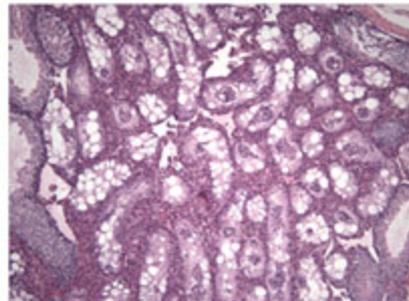
- atrophied spleens
 - intestinal damage
 - impaired sense of smell
 - shrunken brains
 - shrunken testes, depleted sperm count.
- Their telomeres had lengthened and the levels of telomerase increased, waking dormant brain stem cells, producing new neurons. **The mice spleen, testes and brains were rejuvenated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

THE PROMISE OF

PM

Pueraria mirifica

Thailand's botanical secret



Pueraria mirifica

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 cardiovascular disease , (cardioprotective), dyslipidemia, remineralizes bone, inhibits cancer growth, restores vaginal integrity, restores hair color, builds collagen, supports the prostate, improves oxidative stress, increases long term 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?

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

S. Ramnarine¹, J. MacCallum² and M. Ritchie²

¹Edinburgh University, Edinburgh, UK and ²Napier 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 Kwa 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 misocleol. 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-estradiol (βE, concentrations 10⁻⁷ µ-10⁻⁶ µ); GEN (concentrations 10⁻⁷ µ-10⁻⁶ µ); DAI (concentrations 10⁻⁷ µ-10⁻⁶ µ); 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: CyQuantSM 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 (0.5–24h), suggesting a possible modulation of the receptor 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 4d, as examined using the CyQuantSM 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 (x 4, ANOVA, P = 0.088) by day 3 or 4 when compared with untreated cells.

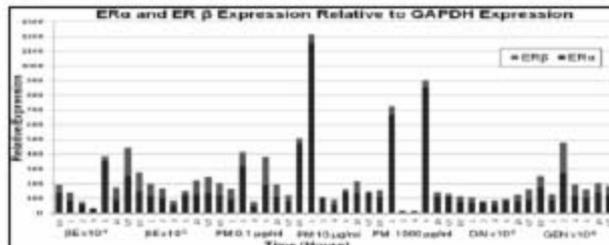


Fig. 1. For each treatment expression of ERα and ERβ mRNA is shown. Time points used were 0.5, 1, 2, 4, 6, and 24h. Expression of ERα is higher for PM treatments (10 and 1000 µg/ml); PM at 0.1 µg/ml is in line with βE and GEN. Expression of ERβ is decreased for PM at 10 and 1000 µg/ml, and similar to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

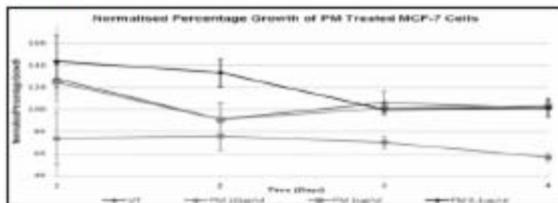
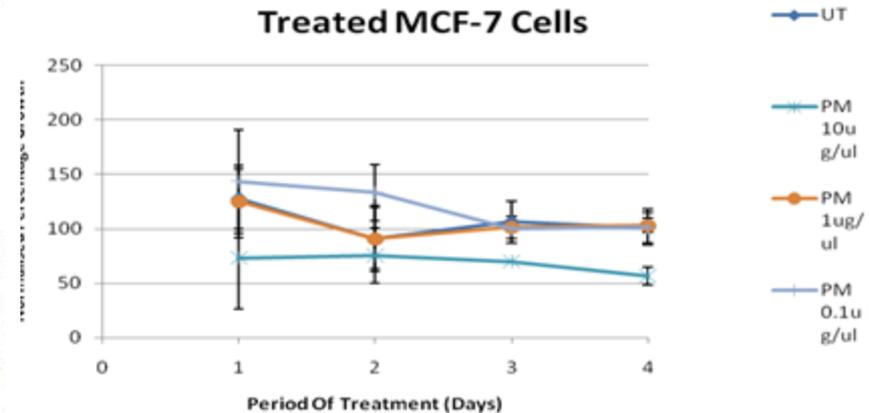


Fig. 2. Effect of PM on MCF-7 growth over 4d. The maximum concentration used was 10 µg/ml, as higher concentrations were previously found to be directly toxic to the cells. Reduction in growth was significant (P < 0.05) for PM at 0.1 µg/ml over 4d.

Normalised Percentage Growth of Treated MCF-7 Cells



“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*

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²Faculty of Medicine, Mahasarakham University, Mahasarakham 44000, Thailand

³Faculty of Pharmaceutical Sciences, Ubon Ratchathani University, Ubon Ratchathani 34100, 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 ethoxycresorufin O-demethylase (EROD) and methoxycresorufin O-demethylase (MROD) activities are the specific markers for CYP1A1 and CYP1A2, respectively. In the present study, the effects of five phytoestrogens isolated from 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-naphthoflavone (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 (IC₅₀) of these compounds on EROD activity corresponded to that of MROD, namely BNF > miroestrol > kwakhurin > deoxymiroestrol > methoxyisomiroestrol > 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-ethoxycresorufin (ER) and 7-methoxycresorufin (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

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“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 Tapaneeayaphan^a and Sutthasinee Poonyachoti^b

^a Department of Physiology, Faculty of Medicine, Srinakharinwirot University

^b Department of Physiology, Faculty of Veterinary Medicine, Chulalongkorn University,

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 antiproliferative effect of *P. mirifica* mediated via estrogen receptor requires further investigation. In contrast, 17 β -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

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.

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National Institutes of Health

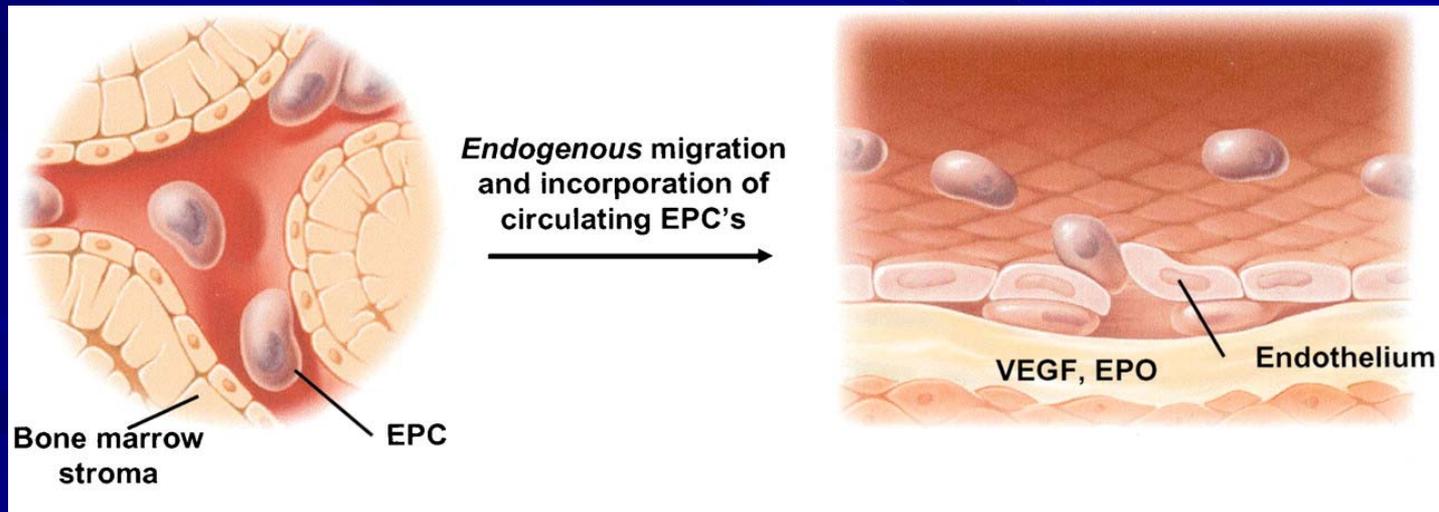
Endothelial progenitor cells (EPCs) play an important role in both reendothelialization and neovascularization. Ex vivo expansion of EPCs might be useful for potential clinical cell therapy of ischemic diseases. However, ex vivo cultivation of EPCs leads to rapid onset of EPCs senescence, thereby severely 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-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

Endothelial Progenitor Cells

- EPCs are rare Cells from bone marrow,
- EPC circulate in the blood with the ability to differentiate into endothelial cells



Puerarin prevented senescence.

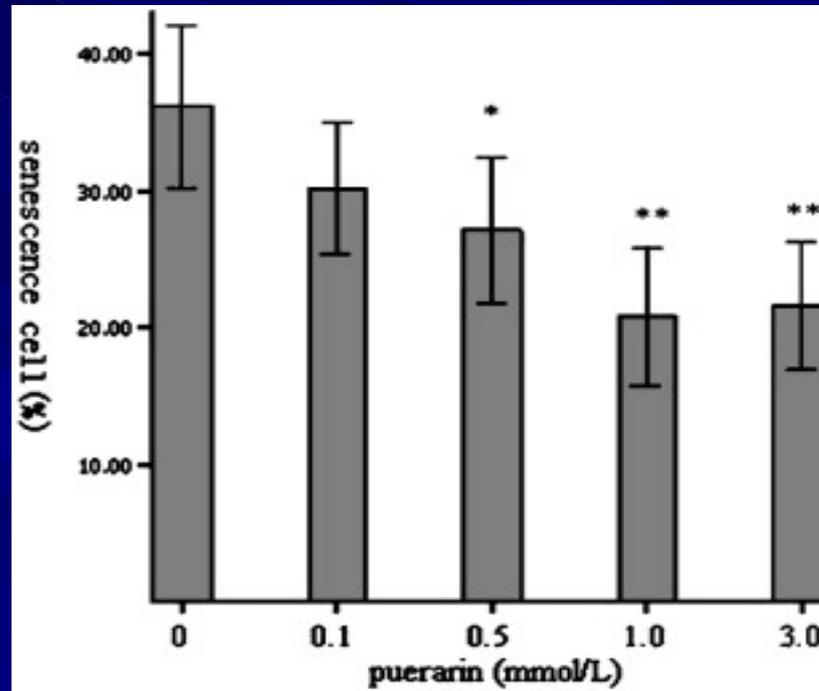


Fig. 1 Puerarin prevented EPCs' senescence. Freshly isolated mononuclear cells were cultivated in Medium 199 supplemented with 10% fetal-calf serum and VEGF. At day 4, cells were seeded in either indicated doses of puerarin in methylcellulose plates.

Puerarin promoted EPC proliferation

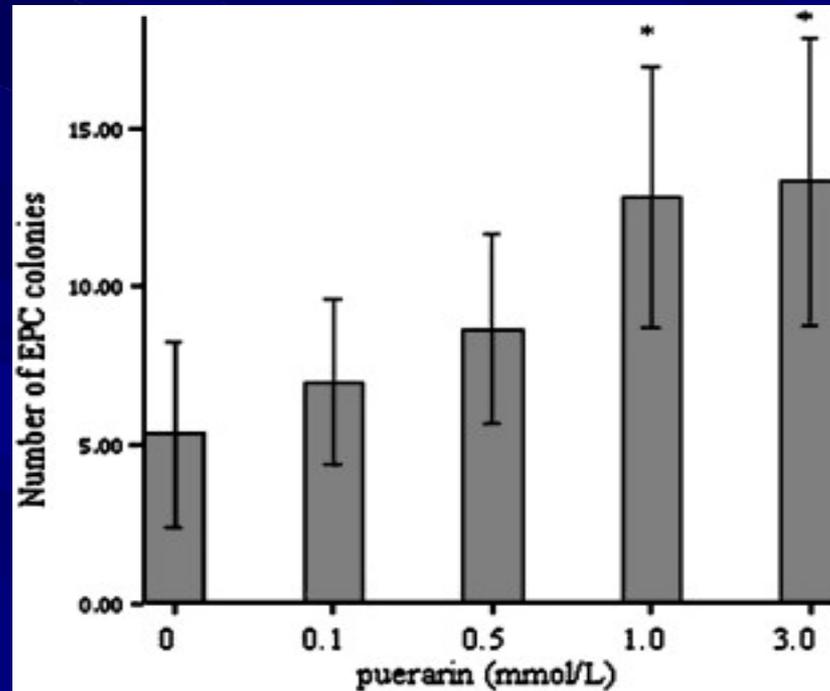


Fig. 2 Effects of puerarin on proliferation in EPCs. EPCs were treated with indicated concentrations of puerarin. Cells were harvested 7 days after culture. Cell proliferation was detected as described in the *Materials and Methods*. Data are mean \pm SD, $n = 5$; * $P < 0.05$, ** $P < 0.01$ vs control.

Puerarin induced telomerase in EPC

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

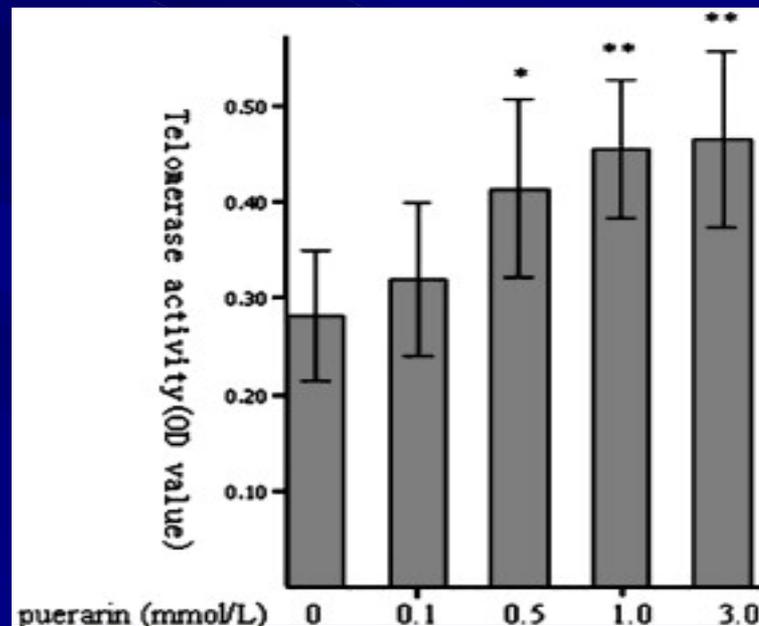


Fig. 4 Effects of puerarin on telomerase activity in EPCs. Freshly isolated mono-nuclear cells were cultivated in Medium 199 supplemented with 20% fetal-calf serum and VEGF. After 4 days of cultivation, EPCs were incubated with either indicated doses of puerarin for 24 h, and telomerase activity was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

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

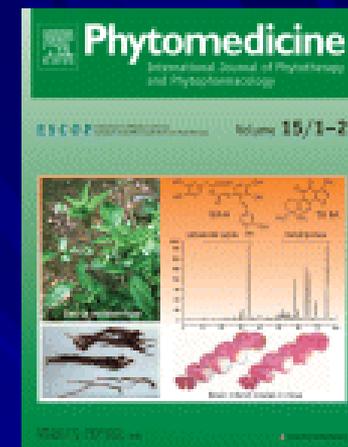
W. Cherdshewasart, W. Sutjit. Faculty of Science, Chulalongkorn University, Phyathai Road, Patumwan, Bangkok 10330, Thailand

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.



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

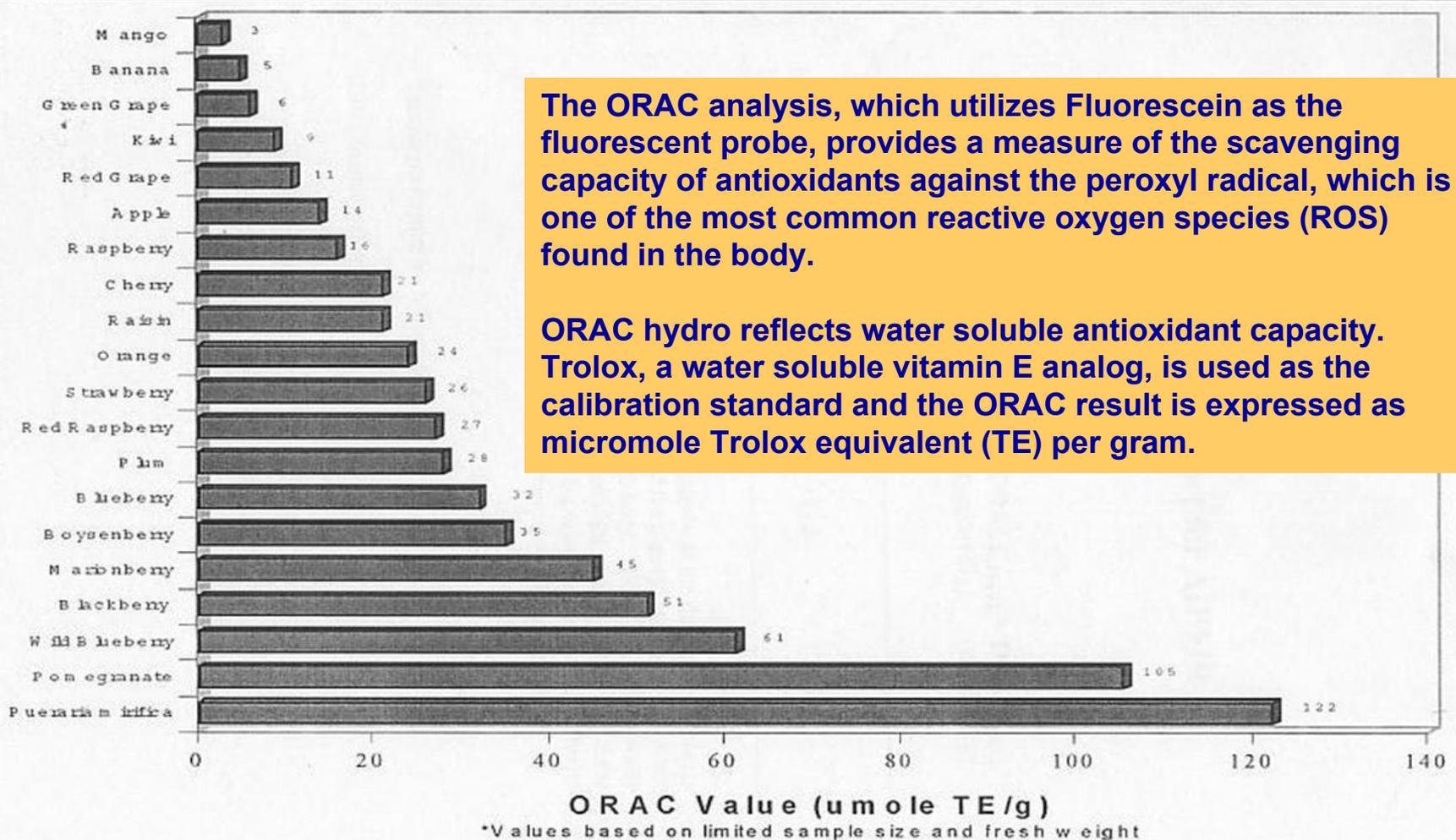
Yingham B, et al.

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 μ g/ml) suppressed the growth of such cells. The maximal proliferative effect of this extract was achieved at 50 μ g/ml which is higher than the effect displayed by 0.1 nM E2.

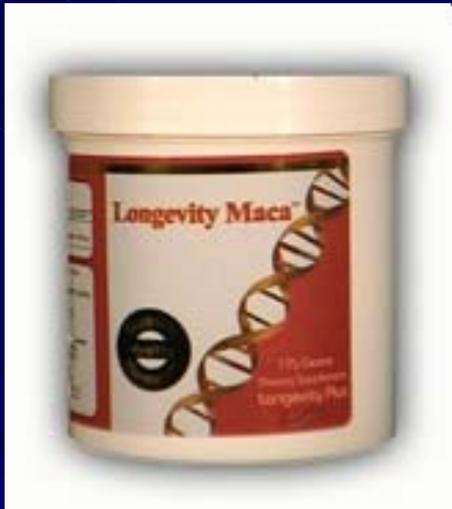
Results indicated that the phytoestrogenic substances in the extracts exerted their estrogenic activities through estrogen receptor pathways.

ORAC Hydrophilic Value of Fresh Fruit vs Pueraria mirifica prepared for AIBMR



The ORAC analysis, which utilizes Fluorescein as the fluorescent probe, provides a measure of the scavenging capacity of antioxidants against the peroxy 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.



Longevity Maca (*Lepidium meyenii* Walp) Powder

Maca's reputation as a powerful enhancer of strength and stamina and as a libido-fertility herb goes back more than 500 years, and today it is gaining worldwide attention for its effectiveness.

Maca is a radish-like root that grows in the mountains of Peru. Peruvian Maca Root naturally contains significant amounts of amino acids, carbohydrates, vitamins, and minerals.



Maca is both a hormone balancer and an adaptogen. It helps stimulate the pituitary gland, acting as a kind of tonic for the hormone system. When the pituitary gland functions optimally, the entire endocrine system becomes balanced, because the pituitary gland controls the hormone output of the other three glands.

TOXINS

The average US city is home to approximately
77,000 toxins
which we breathe, drink, ingest and absorb
on a daily basis.

Our bodies are designed to release accumulated toxins through bowel movements, urination, sweating or saliva; however, if we have poor digestion or constipation, our systems will find other ways to release toxins *such as* skin rashes, kidney and liver infections or colon cancer.

An ongoing detoxification program is critical to reduce toxic buildup and prevent disease.

The Environmental Working Group studies that have shown:



From
Environmental Working
Group

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>

Watch The Program Now ▶

HOME | INTERVIEWS | RISE IN AUTISM | SITE MAP | DISCUSSION

THE VACCINE WAR

Examining the emotionally charged debate over medical risks vs. benefits and a parent's right to make choices about her child vs. a community's common good...

Introduction »

Watch the Full Program Online



How Do You Feel About Childhood Vaccines?
Join the discussion

Interviews
Anthony Fauci, J.B. Handley, Paul Offit, Jenny McCarthy, Cynthia Cristofani, Robert Sears...

What's Behind the Rise in Autism?
...and other interesting readings and sites

Forum: Doctors and Viewers
Check out the posts and comments

POLL DID THIS REPORT CHANGE YOUR MIND?

SURVEY WHERE DO YOU STAND?

<http://www.pbs.org/wgbh/pages/frontline/vaccines/>

Genetically Modified Front Lawns and the mass spraying of neighborhoods and playgrounds with RoundUp!

Sunday, July 17, 2011 - by Mike Adams, the Health Ranger



NaturalNews.com
Natural Health, Natural Living, Natural News

Thanks to a recent admission by the USDA that it does not have the regulatory framework to even regulate GMOs, the world of biotech is set to unleash a tidal wave of genetically modified seeds upon the United States.

Scotts Miracle-Gro is now moving full speed ahead on its GMO yard grass product. This is a home consumer yard grass seed which, of course, resists glyphosate – or RoundUp.

RoundUp, in other words, may be coming soon to a neighborhood near you.

And it's not just the lawns, either: This combination of Scotts GMO grass and RoundUp chemicals could be used on playgrounds, schoolyards, community centers and parks.

Once this goes into production, there will be virtually no place your family can go in America that isn't contaminated with genetically modified grass seeds and toxic glyphosate chemicals.

Six Risky Chemicals You're Carrying In Your Body

by [Dr. Mercola](#) | January 07 2010



The U.S. Centers for Disease Control and Prevention has released its latest assessment of the chemicals people are carrying around in their bodies. The bio-monitoring study is the most comprehensive in the world, measuring 212 chemicals in the blood and urine of 8,000 Americans.

The CDC highlighted a few chemicals because they are both widespread -- **found in all or most people tested** -- and potentially harmful.

- **Polybrominated diphenyl ethers** (Better known as "flame retardants" or PBDEs)
- **Bisphenol A** (or BPA, which is found in most plastics)
- **PFOA** (perfluorinated chemicals used to create heat-resistant and non-stick coatings on cookware)
- **Acrylamide** (formed when carbohydrates are cooked at high temperatures)
- **Mercury/Methylmercury** (mostly from eating fish)
- **MTBE** (gasoline additive that although phased out in favor of ethanol, has contaminated many drinking water supplies)

It's not just the quality and quantity of our food that is making us sick... but the toxic materials we use to prepare, cook and store it too...

**David Ewing Duncan
cooks breakfast at home.
On the menu: PBDEs, phthalates,
PCBs, and a side of PFAs.**



A class of chemicals, called phthalates, is added to plastics, including some food wraps, for pliability.

These chemicals can cause cancer and other developmental problems in lab animals, even at relatively low doses.

FDA Bans BPA in Baby-Formula Packaging Agency Does not Take Action on the Chemical's Use in Food Cans - by Thomas M. Burton

THE WALL STREET JOURNAL.

U.S. EDITION Thursday, July 11, 2013 As of 5:11 PM EDT

The Food and Drug Administration said it will no longer allow the use of the chemical bisphenol-A, or BPA, in packaging for baby formula, but took no action on the controversial chemical's continued widespread use in food cans.

BPA has been linked to possible health problems of the brain, breast and prostate.

In 2008, the environmental group Natural Resources Defense Council asked the FDA to ban use of the chemical because of what it termed "serious adverse health effects."

In 2011, the American Medical Association deemed BPA an "endocrine-disrupting agent" and urged that "BPA-containing products with the potential for human exposure be clearly identified."

In 2012 The FDA banned the use of BPA in baby bottles and cups, but says it lacks the scientific information to ban BPA in packaging like cans, and only took action because manufacturers had abandoned use of it already in baby-formula packaging.

BPA (Bisphenol A) - Effects

- Endocrine disruptor, strongest effects during early development
- Estrogen mimic
- Obesity
- Neurological disorders
- Thyroid function
- Cancer risk: breast, prostate, neuroblastoma
- Reproductive anomalies – ovarian development, ...
- DNA alterations related to estrogen
- Heart disease, diabetes
- Growth, reproduction, development of aquatic organisms, including fish, invertebrates, amphibians,

Major Study of Teflon Chemical in People Suggests Harm To Immune System, Liver, Thyroid

Preliminary Data Released as Calif. Senate Votes to Ban PFCs in Food Packaging
May 13, 2008

WASHINGTON – A chemical used to make Teflon, food wrappers and dozens of other products may harm the immune system, liver and thyroid and cause higher cholesterol in children, according to the initial findings of a study of 69,000 people in West Virginia and Ohio who live near a DuPont manufacturing plant.

The health effects observed in the study population are strong indicators of health problems that might be caused by PFOA in average Americans. Toxic effects from PFOA were observed in study participants with blood levels of the chemical equal to those found in the more highly exposed individuals in the US population. PFOA (also called C8) is one of a class of perfluorinated chemicals, or PFCs, used to make Teflon and other nonstick products, oil-resistant paper packaging and stain-resistant fabrics.

A team from West Virginia University is leading the multi-year PFOA study, which is funded by a portion of a \$107.5 million settlement paid by DuPont to settle a lawsuit over dumping PFOA in the region's drinking water supplies. Last week, in the first public presentation of the C8 Health Project data, WVU scientists reported:

In animal studies, PFOA exposure has previously been linked with death of immune cells and weakening of the body's ability to protect itself from infection. The WVU study indicates higher levels of PFOA in people correlate with lower levels of a protein that helps the body fight bacteria, viruses, and other pathogens.

Higher PFOA levels in West Virginia and Ohio residents are associated with higher levels of two enzymes that can indicate liver damage, and with lower levels of a liver protein that is an important part of the body's defense against infection.

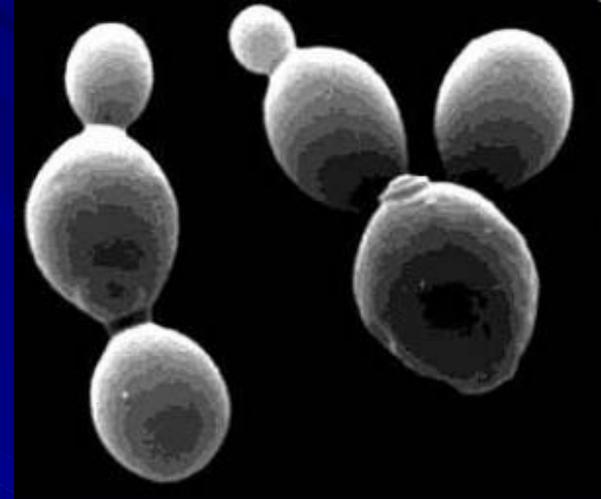
Good News In Our DNA: Defects You Can Fix With Vitamins And Minerals

ScienceDaily (June 3, 2008)

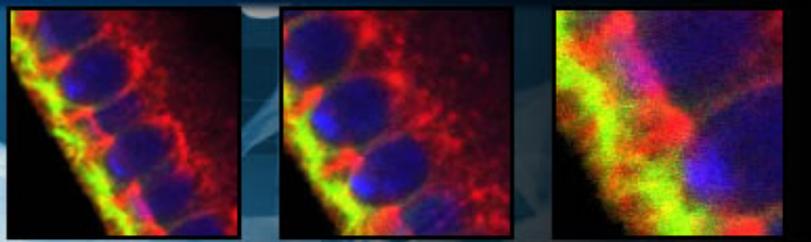
As the cost of sequencing a single human genome drops rapidly, with one company predicting a price of \$100 per person in five years, soon the only reason not to look at your "personal genome" will be fear of what bad news lies in your genes.

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."*



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.



Negative Effects of Plastic Additive Blocked by Nutrient Supplements

DURHAM, N.C. - Experiments in animals have provided additional and tantalizing evidence that what a pregnant mother eats can make her offspring more susceptible to disease later in life.

We have shown that during early fetal development, maternal nutrient supplements of methyl-donating substances (folic acid, choline, vitamin B12, and betaine) or genistein, found in soy products, can counteract the reduction in DNA methylation caused by BPA. Nevertheless, we have not yet tested if exposure to these nutrient supplements can reverse the negative effects of BPA in adulthood.

Methylation support has become vital to help deal with THE TOXINS Found in everyone.

Medical Maverick

Dr. Tsuneo Kobayashi

Originally published at www.japaninc.com December 2005

Melding East and West: a forerunner of cancer treatment and prevention.

by John Dodd



Over the last 30 plus years, he has become a thorn in the side of conventional cancer physicians, not least for his idiosyncratic behavior and treatment methods, which are based on a lifetime of experimentation and observation, and a belief in the holistic nature of the human body. He uses Chinese herbal medicines in addition to drugs for biochemical-modulation and apoptosis-inducing and cancer-vessel treatment, as well as sophisticated methods of applying **TMCA (tumor marker combination assay)**, heat therapy and immunology.

Proof Kobayashi Method Works: In the last 25 years, he has treated more than 20,000 early stage patients, and more than 2,000 mid-to-latter stage patients, who subsequently went into long term remission, with an average life span after treatment of seven years.

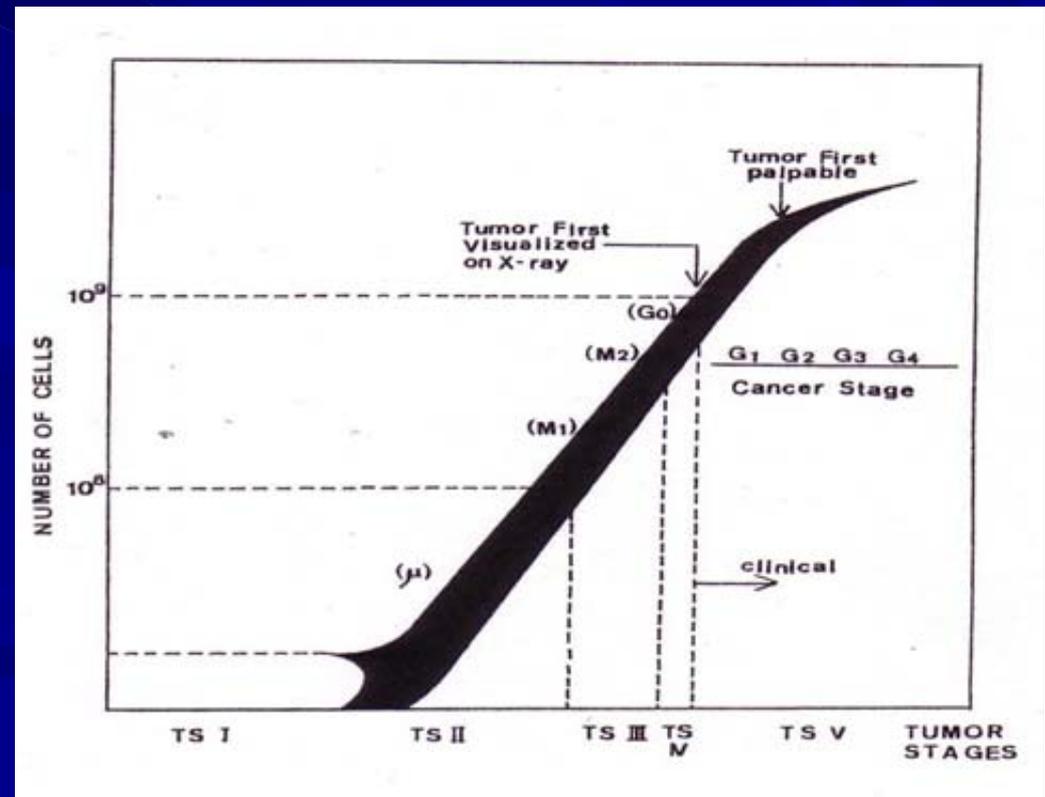
In comparison, the average life span of breast cancer patients in remission is 4.5 years (Source: US National Cancer Institute) and for prostate cancer patients it is around 2 years.

<http://www.euro-med.us/dr-kobayashi-story.pdf>

Tumor First Palpable typically after seven years of growth...

Regardless of the process by which normal cells become cancerous, as these toxic cells fatten and grow, they require more nutrients to survive. Over time, a network of blood cells and friendly neighbors start to emerge and the tumor grows into a palpable lump that until recently was one of the few clues for a cancer diagnosis. A tumor that is detectable by feel has been growing for approximately seven years.

By that time, more often than not, treatment is too late. Early detection significantly increases the likelihood of survival, and much research is geared toward detecting cancer-prone and individual cancer cells long before a tumor forms. Tumor markers, such as those developed by Dr. Kobayashi, are one method of early detection.

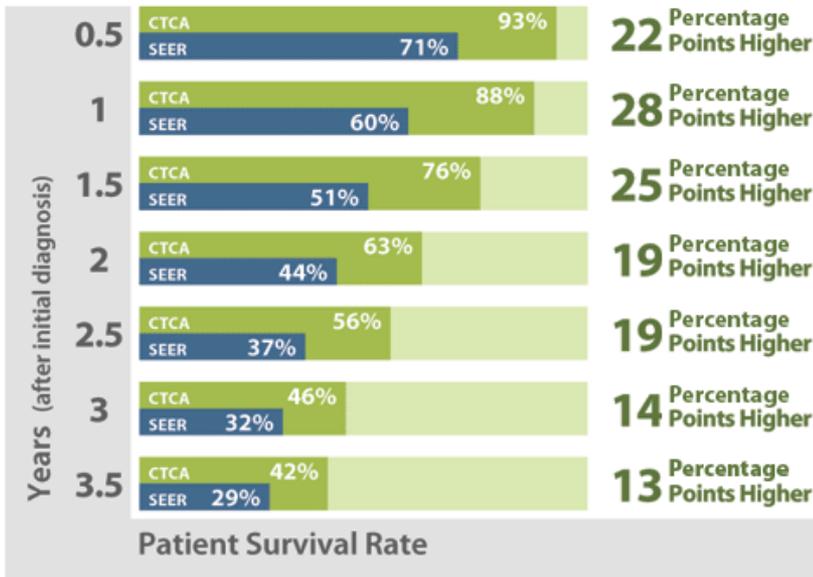




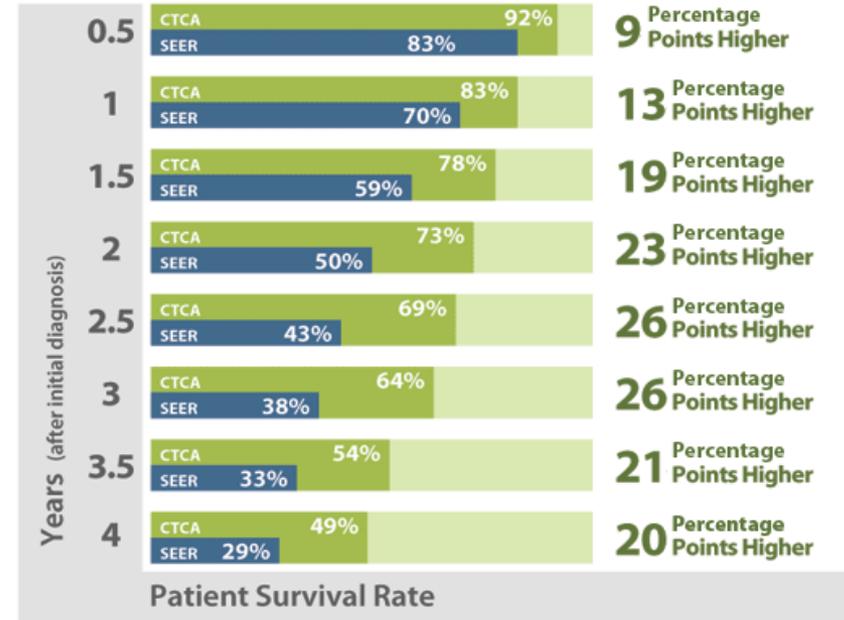
Cancer Treatment Statistics & Results

<http://www.cancercenter.com/cancer-statistics.cfm>

Advanced-Stage Breast Cancer Survival Rate



Advanced-Stage Prostate Cancer Survival Rate



Charts depict a comparison of advanced-stage (defined as distant metastasis) survival rates at CTCA hospitals to publicly available data from the National Cancer Institute Surveillance Epidemiology and End Results Program (SEER).

We all see cancer patients everyday now - and these vital statistics brought to you in a great format by CTCA Cancer Treatment Centers of America has the best organized website I have seen to help promote the value of alternative medicine!

CA Profile© (The Original Cancer Profile!)

American Metabolic Testing Laboratories, Inc.
1818 Sheridan Street, Suite 102
Hollywood, FL 33020
phone: 954. 929. 4814
fax: 954. 929. 4896

Measures molecular, cellular activity.

The Profile is composed of EIGHT tests:

- **HCG (IRMA)** : (intact HCG) human chorionic gonadotropic hormone, the pregnancy hormone and "malignancy hormone," according to Dr. Schandl. This test detects only intact HCG. That is, the alpha and beta intact subunits only.
- **HCG (IMM)** : (intact, beta, nicked beta and beta core subunits of HCG) human chorionic gonadotropic hormone, the pregnancy hormone and "malignancy hormone," according to Dr. Schandl. Normal: less 1.1
- **HCG (IMM) Urine** : a confirmatory determination for the presence of the true HCG hormone. It is important to find two of the three HCG tests positive in order to avoid misinterpretation.
- **HCG-Urine** may be the only one in the world to quantify this hormone at a super low detection limit; is immuno-specific, not the Phillipine acetone extraction method that will test for HCG, TSH, LH, and FSH all at the same time.
- **PHI** : phosphohexoses isomerase enzyme that regulates anaerobic metabolism; it is a neurokine, the autocrine motility/malignancy factor.
- **CEA** : carcinoembryonic antigen, is a broad spectrum cancer antigen.
- **GGTP** : gamma-glutamyl transpaptidase, a rather sensitive enzyme for monitoring the liver and bile system, and kidneys and heart also.
- **TSH** : thyroid stimulating hormone that can detect high or low thyroid activity.
- **DHEA-S** : dehydroepiandrosterone sulfate, the adrenal "anti-stress, pro-immunity, longevity hormone," according to Dr. Schandl. Most cancer patients and those, who are developing cancer have low DHEA blood levels.

Vitamins vs. Chemotherapy and Radiation for Cancer Therapy

by Reagan Houston, MS, PE

Townsend Letter – Aug/Sept 2009

Vitamins can strengthen the immune system to improve regular therapies and safely kill cancer. Here we compare cancer therapy by multivitamins with radiation and most chemotherapies. The late Abram Hoffer, MD, PhD, prescribed a regimen high in oral vitamin C plus other vitamins and minerals (Table 1). He also prescribed a diet low in meat, very low in sugar, but high in fruits, vegetables, and water. Most of his patients had failed prior surgery, radiation, and/or chemotherapy as prescribed by their oncologists. To all of his cancer patients, Hoffer offered the vitamin regimen, diet, and hope based on the results with earlier patients.

Table 1: Dr. Hoffer's Regimens (3), (6)

	Early	Later
Vitamin C mg	12,000	12,000
range	3,000-40,000	3,000-10,000
* Vitamin A, IU	10,000-50,000	
* Beta carotene	30K-75K IU	30,000 IU
Vitamin B complex	B50 to B100	1 or 2 of B100
Vitamin D-3	5,000 IU	To 19,000 IU
Vitamin E	300 IU	
Vitamin E succinate		800 IU
Selenium	600 mcg	400 to 600 mcg
Zinc as citrate	60 mg	60 mg
Coenzyme Q10		300 IU
Curcumin		300 mg
* Bioperin		15 mg
* Optional		

Table 2: Survival of Cancer Patients After Seeing Hoffer (11)

Type of Cancer	With Vitamins	Without Vitamins
Breast	70	3.7
Lung	17	2.0
Ovary	16	3.6
Pancreas	40	2.4
Uterus	99	4.0
All 30 types	45 months	2.6 months

Dr. Hoffer's results were excellent. Those who refused vitamins lived a median of only 2.6 months. The 101 who accepted vitamins lived 45 months after seeing Hoffer (Table 2).

Chemo, Radiation can Increase Malignancy of Tumors

Tumors consist of a diverse collection of cells, many of which are harmless (benign). The cancer stem cell is the most dangerous and is also very difficult to locate. There is only about one cancer stem cell for every 10,000 benign cells.



According to recent research, traditional cancer treatments may actually incite the growth of more of these dangerous cancer stem cells. These stem cells promote tumor growth as well as cause cancer to spread within the body.

In an interview with FoxNews.com, Dr. Chiang Li of Harvard Medical School in Boston, said **"Radiation and chemotherapy not only might create cancer stem cells, any pre-existing cancer stem cells in a tumor were very resistant to radiation and chemotherapy, so they remain as well"**.

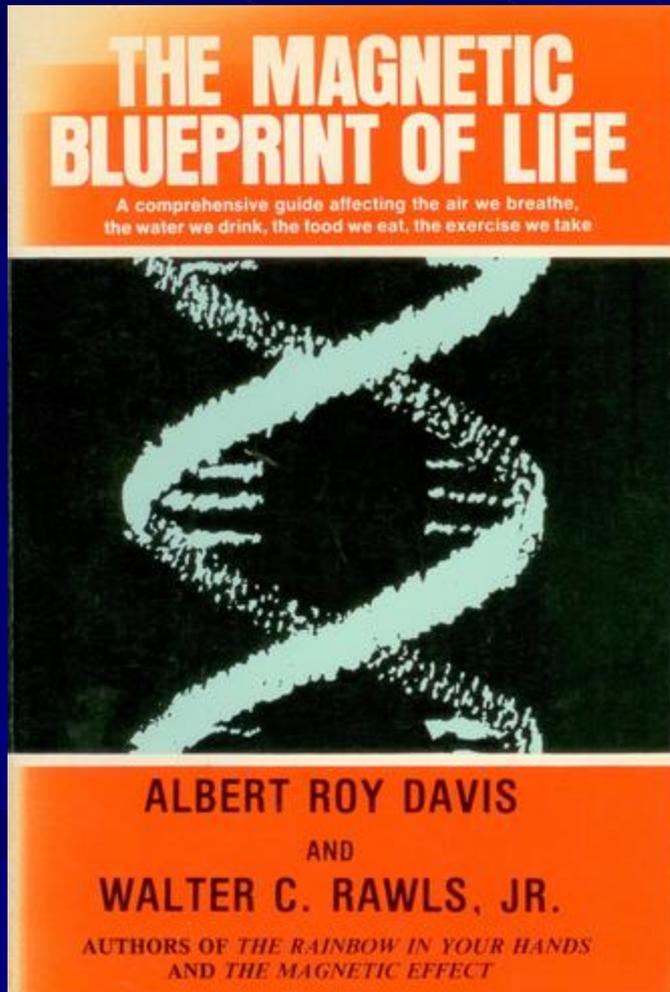
Studies continue to show that chemotherapy and radiation rarely provide a comprehensive, safe solution to cancer. Thus far, America's multi-billion-dollar search for a cure has provided only a handful of insufficient treatments that cause dozens of side effects.

ELECTROMAGNETISM

“The Missing Link?”

**Pulsed Electro-Magnetic Frequency (PEMF)
Low Level Laser Therapy (LLLT)
Intra-nasal Light Therapy (INLT)**

Evolution with Natural Magnetism – Through scientific method magnetism has been shown to exist in two separate energy forms, described as negative and positive, which are similar to negative and positive energy forms existing in all biological systems and organisms on the Earth.



It is known that **external energy forms of magnetism will affect the internal energy forms of biological systems, when programmed correctly.** Negative magnetism will arrest disease and infection, and positive magnetism will strengthen life forms.

The predominant factor in man's existence and his being, is the development of his brain and nervous system, his conscious awareness and perception of his being and his environment.

Magnetism, applied separately and distinctly, with known procedures and control factors, will increase the left and right hemisphere abilities of the brain. Life processes can be speeded up or slowed down. Nervous system structures and performances can be dulled or stimulated... **healthy or ill, the biological system will indicate its own built-in rejection or acceptance of the separate and distinct natural energy forms of magnetism** (excerpt from pg 11).

Fueled by Electro-Magnetic Energy ***We are only as healthy as our cells***

“By regenerating the cells in our bodies we can help our cells become and stay healthy with pulsed electromagnetic fields.

The earth creates magnetic fields, without which life would not be possible. Science teaches that everything is energy. All energy is electromagnetic in nature. All atoms, chemicals, and cells produce electromagnetic fields. Science has proven that our bodies actually project their own magnetic fields and our seventy trillion cells in the body communicate via electromagnetic frequencies.

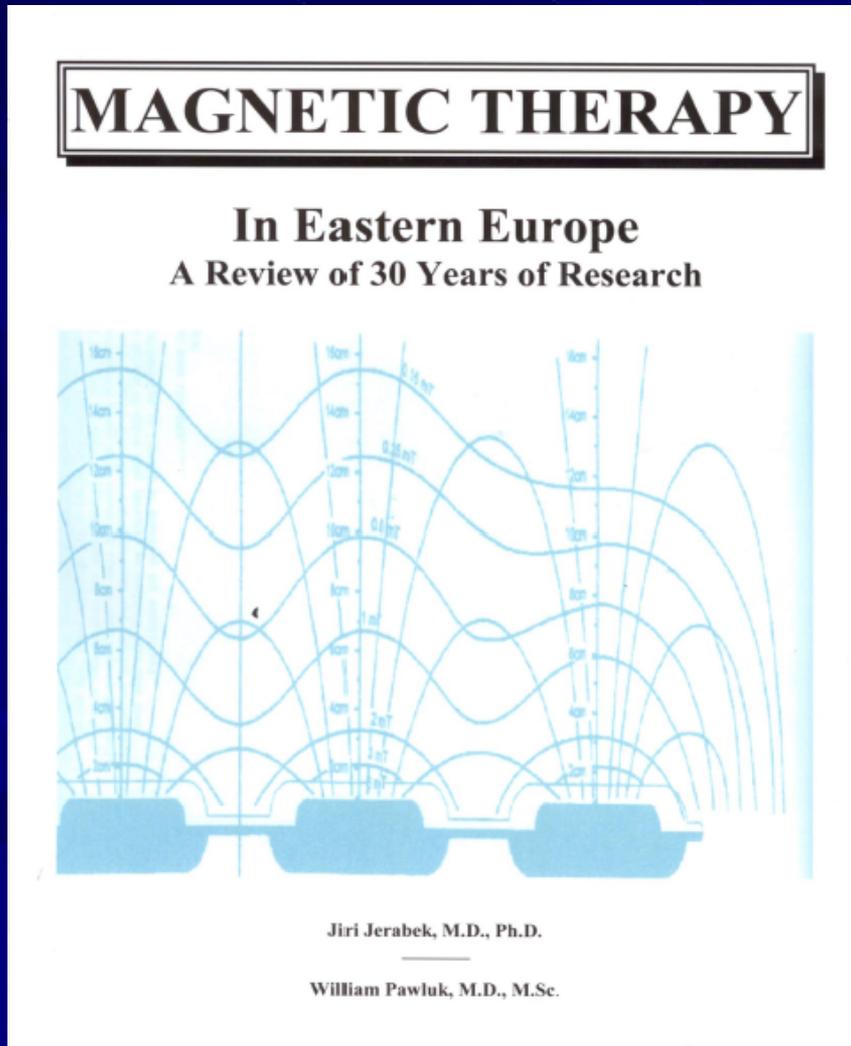
Disruption of electromagnetic energy in cells causes impaired cell metabolism. This is the final common pathway of disease. If cells are not healthy, the body is not healthy.”

William Pawluk, MD, MSc, and Donna Ganza, ND
Excerpt from 101 Great Ways to Improve Health

Magnetic Therapy in Eastern Europe: A Review of 30 Years of Research

By Jiri Jerabek, MD, PhD and William Pawluk, MD, MSc

The book presents information summarizing conditions studied, magnetic field strength and type of field used, frequency and duration of application and summary of actual results. There are detailed descriptions of many studies on both static (permanent) and frequency (pulsed) fields.



Controlled human studies described include:

- Atherosclerosis
- Brain neurosecretion
- Breast fissures
- Burns
- Carpal tunnel syndrome
- Cervicitis
- Chronic bronchitis
- Controlled Studies Animals
- Corneal trauma
- Edema
- Endometriosis
- Femoral artery surgery
- Fractures
- Increased circulation
- Infected skin wounds
- Ischemic heart disease
- Limb grafts
- Liver function

And more...

Bioelectromagnetics. 2009 Jan;30(1):21-8.

Prolonged weakening of the geomagnetic field (GMF) affects the immune system of rats.

Roman A, Tombarkiewicz B.

Department of Brain Biochemistry, Institute of Pharmacology, Polish Academy of Sciences, Kraków, Poland. roman@if-pan.krakow.pl



We found that the long-term shielding of the GMF could influence the functioning of the immune system in a sex-dependent manner.

The deprivation of the GMF delayed physiological thymus involution, that effect being more strongly expressed in females. The weakening of the GMF resulted in an increased number of peritoneal macrophages, especially in males.

The shielding of the GMF diminished the ability of macrophages to release NO and to synthesize O₂(-), those effects being more powerfully expressed in males and females, respectively.

It is proposed that the observed changes in the immune system occur as a consequence of the protective effect of GMF shielding on the circadian rhythm-dependent level of melatonin.

A role for the geomagnetic field in cell regulation.

Liboff AR.

Center for Molecular Biology and Biotechnology, Florida Atlantic University



Abstract

We advance the hypothesis that **biological systems utilize the geomagnetic field (GMF) for functional purposes by means of ion cyclotron resonance-like (ICR) mechanisms.**

Numerous ICR-designed experiments have demonstrated that living things are sensitive, in varying degrees, to magnetic fields that are equivalent to both changes in the general magnetostatic intensity of the GMF, as well as its temporal perturbations. We propose the existence of ICR-like cell regulation processes, homologous to the way that biochemical messengers alter the net biological state through competing processes of enhancement and inhibition. In like manner, **combinations of different resonance frequencies all coupled to the same local magnetic field provide a unique means for cell regulation.**

PMID:20707644 [PubMed - indexed for MEDLINE]

Power Failure

Does mitochondrial dysfunction lie at the heart of common, complex diseases like cancer and autism?

By Megan Scudellari

Over the last five years, a growing number of papers by researchers around the world have implicated dysfunctional mitochondria in many elusive diseases, including Parkinson's, autism, and aging.

Leading the charge is a respected and renowned member of the National Academy of Sciences, Dr. Douglas Wallace, founder of the field of human mitochondrial genetics.

“Every one of the diseases we can't solve is absolutely logical if we put energy at the center,” Dr. Wallace says.

Medicine fails to solve many of today's common, complex diseases, Wallace asserts, because the fundamental paradigm is wrong: the medical establishment has spent far too long focusing on anatomy and ignoring energy—specifically, mitochondria.



Dr. Garry Gordon's **FIGHT-EM with M.I.C.E.** = **Magnetically Induced Cellular *EXERCISE***

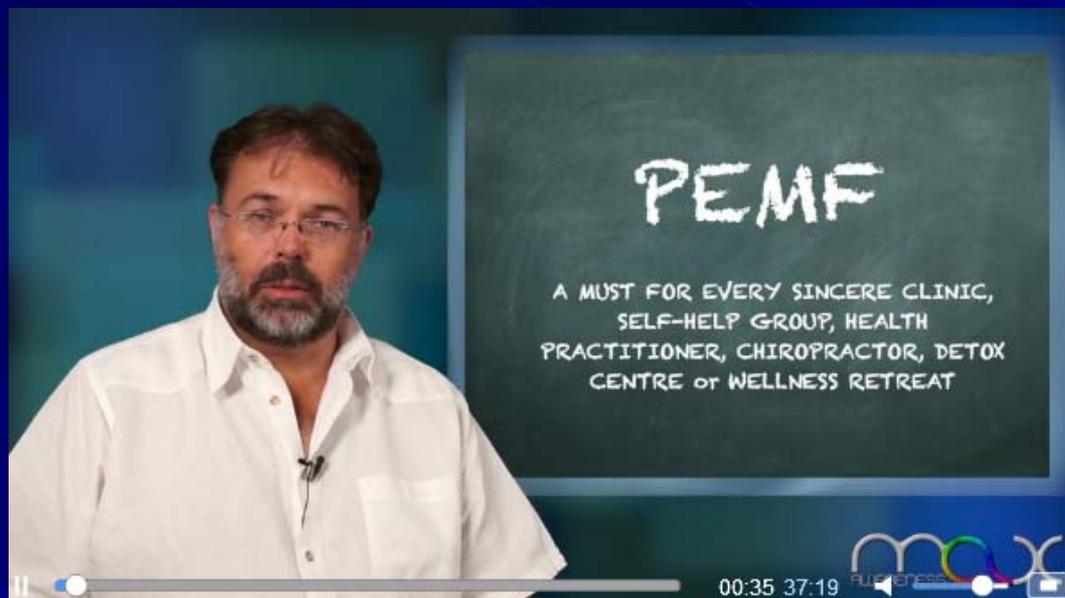
Pulsed electro-magnetic frequency (PEMF) therapy recharges the body's 70+ trillion cells. Like physical exercise, it increases cellular bioporation, oxygenation, alkalinity, energy production, and nutrient uptake – while promoting vital autophagic processes and detox of harmful toxins and metals.



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

EDTA (calcium edta)
Vitamin C
Zeolite
Fiber

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.

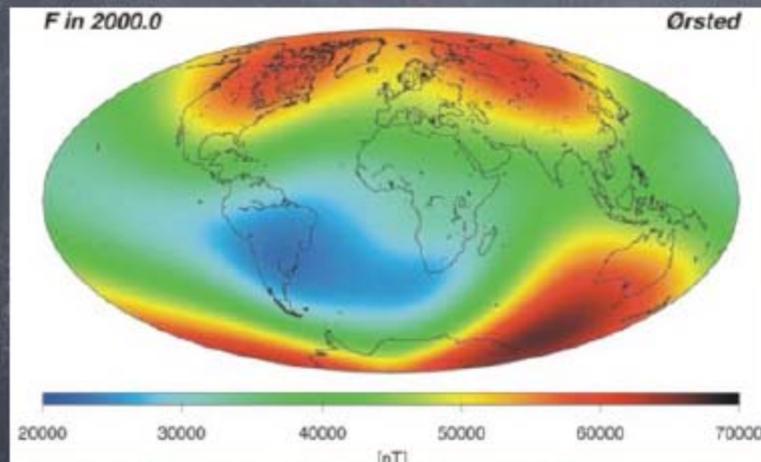
Visit Marcus Freudenmann's MAXAWARENESS site and learn more about how you can join the Truly Heal community

<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.

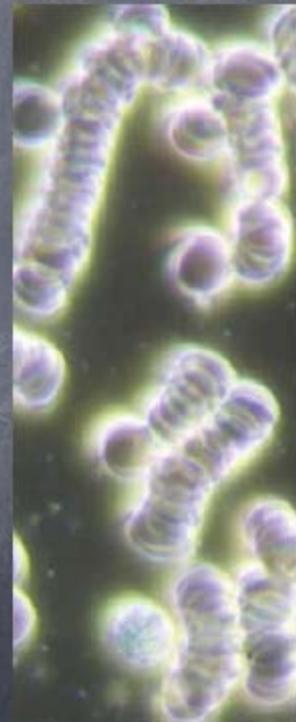
WHY DO WE NEED IT

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).

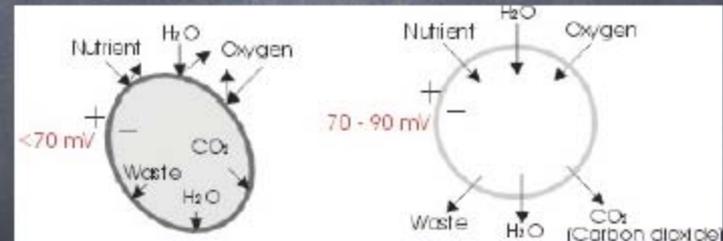
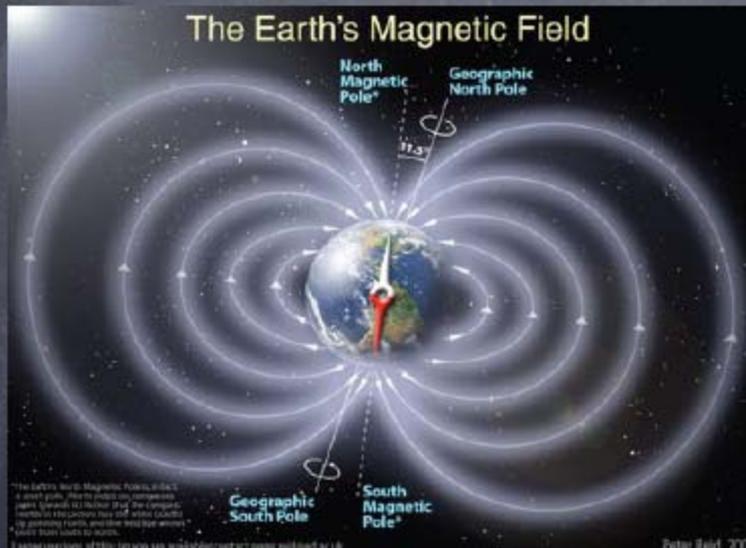
If this potential approaches the zero level, the cell dies. A cell uses 50% of its energy to maintain this potential.

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

This means in simple terms that the power of any "home" device does not penetrate deep enough to have an effect on your health. Rather save your money and enjoy an hour in the park.



POWER

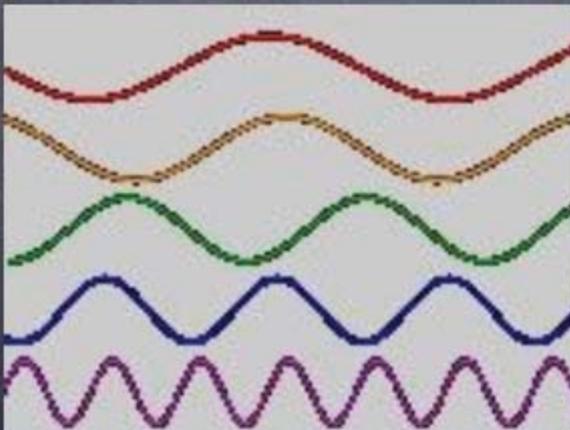
What you want is **THERAPEUTIC POWER** which is only available from 10 Gauss up!



Symbol	Meaning	Field strength in Tesla	Formerly in Gauss
T	Tesla	1	10000
mT	milli Tesla	0.001	10
µT	micro Tesla	0.000.001	0.01
nT	nano Tesla	0.000.000.001	0.000.01
pT	pico Tesla	0.000.000.000.001	0.000.000.01

FREQUENCY

Many independent studies done during the last 30 years clearly indicate that frequencies which are beneficial for human applications are mainly between 1 and 50 Hz



- However, it is still unclear which exact frequencies relates to specific diseases, body functions, cells, and bones.
- Some suggest that frequencies in the range of 1-15 Hz are more important, while others prefer frequencies in the 10-50 Hz range

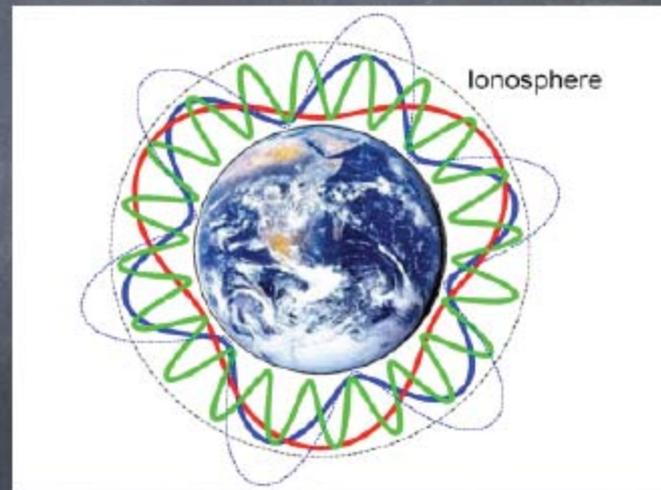
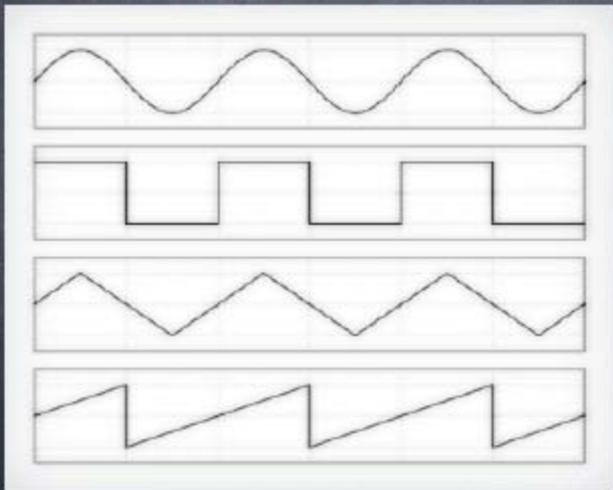
That's why you want a device that operates in all ranges between 1 and 50 Hz

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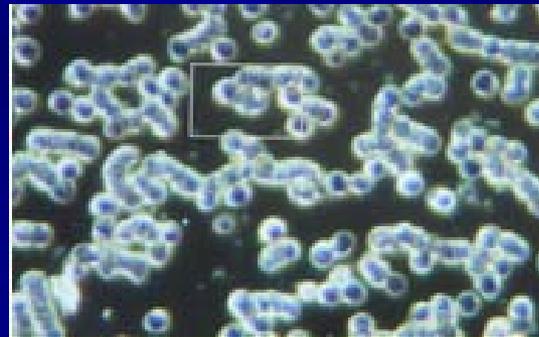
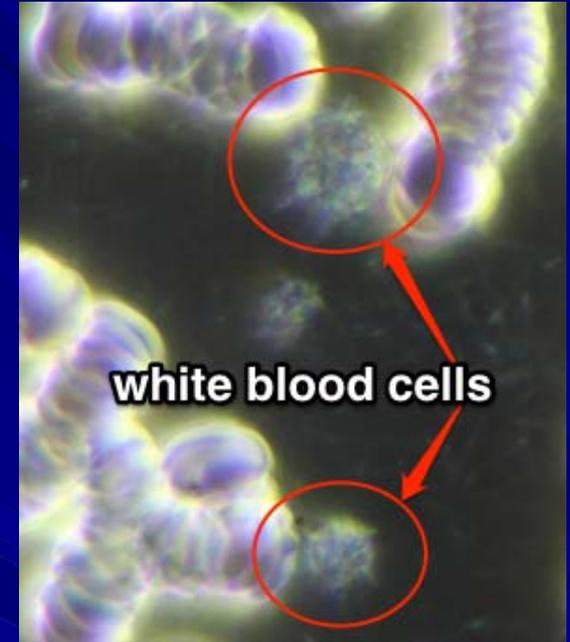
PULSE FORM

The mathematician Joseph Fourier discovered that **Sinusoidal** waves are the actual basic "building blocks" that make up nearly all other periodic waveforms, including square waves, triangle waves and sawtooth waves!



That's why you want a device that operates on sinus or square waves which cover a large portion of the whole spectrum.

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



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...

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.

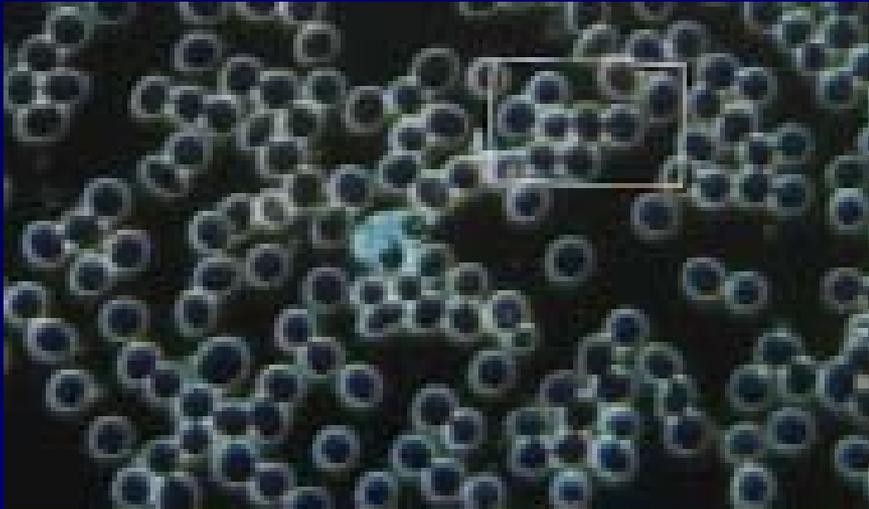
Sick cells such as these are unable to take in oxygen or therapeutic nutrients

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.

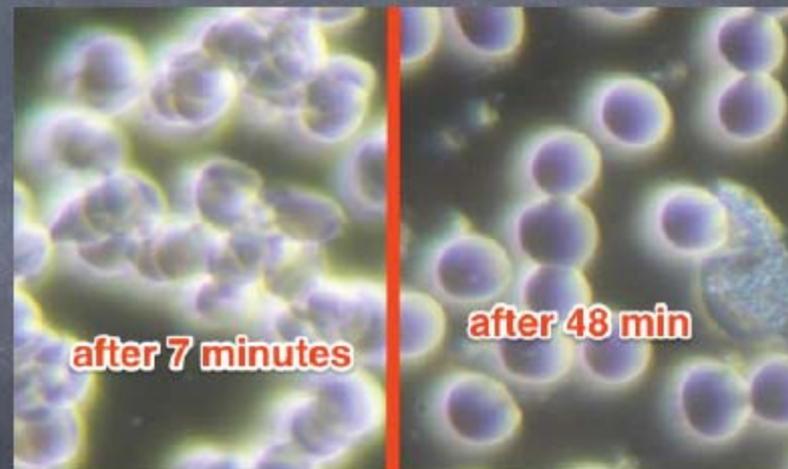
All cells held the membrane stability and mobility for around 45 min, and tested again the next day, in the morning, the blood appeared the same – still energized .

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.

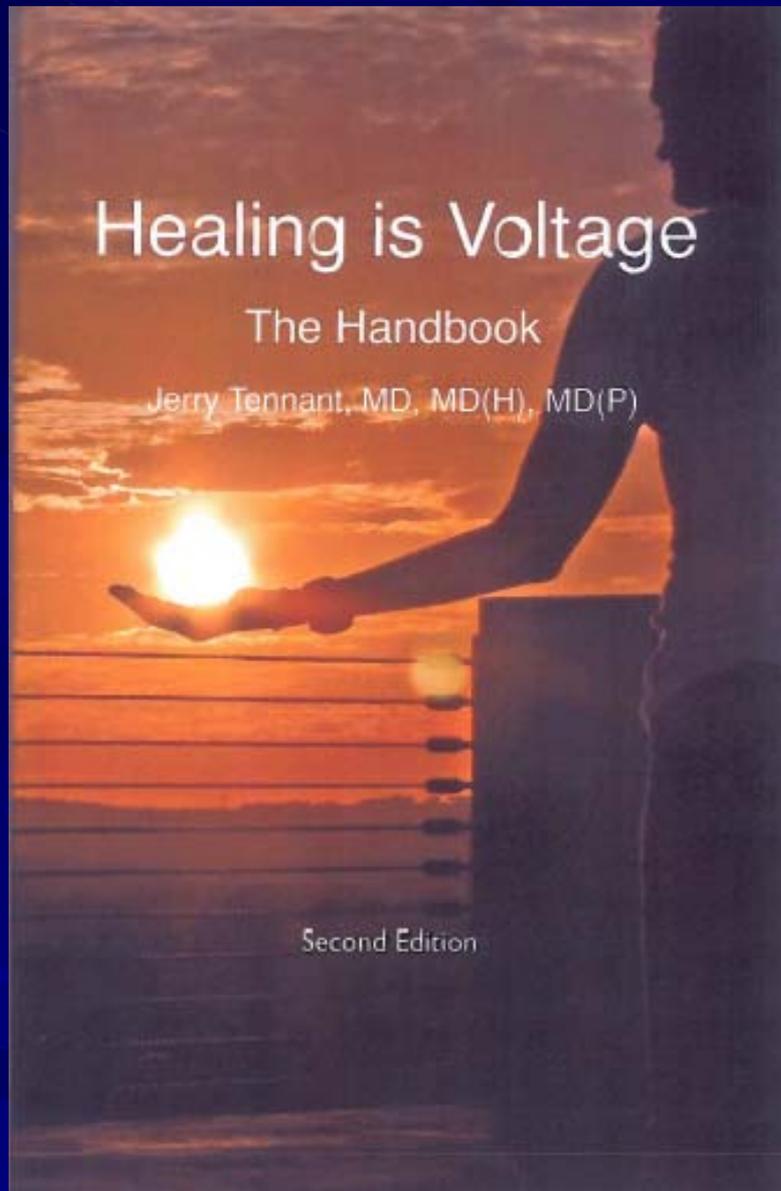
The patient immediately felt the change in energy





WARNING

Plug in both cables
and before turning
main power on
Turn both power off
before unplugging
any cable lead.



Healing is Voltage – The Handbook

Every cell in the body is designed to run at -20 to -25 millivolts.

To heal, we must make new cells. To make a new cell requires -50 millivolts.

Chronic disease occurs when voltage drops below -20 and/or you cannot achieve -50 millivolts to make new cells.

Thus chronic disease is always defined by having low voltage.

This book tells you how to measure your voltage in each organ, how to correct it, and how to determine why your voltage dropped enough to allow you to get sick.

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.”

The crisis of low energy is reflected in the following general chain reactions and results :

- low transmembrane potential
- increased accumulation of sodium ions inside the cell : Hypernatremia
- increased water molecules attached to sodium molecules inside the cell associated to hypernatremia
- inflammation
- increased volume of the cell and osmotic pressure inside the cell, damaging
- the cell membrane
- swelling of cell, followed by thinning of the cell membrane
- cell division

The above conditions further obstruct cell metabolism. When transmembrane potential drops below 15 mvolts, it leads to cell division and eventually causes cancerous cells to over populate.

So, we see naturally why the tumor grows or diffuses to adjacent areas and tissues, **a phenomenon known as “cancer diffusion”**, i.e., cancers ability to diffuse to adjacent healthy cells and tissues.

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.

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.

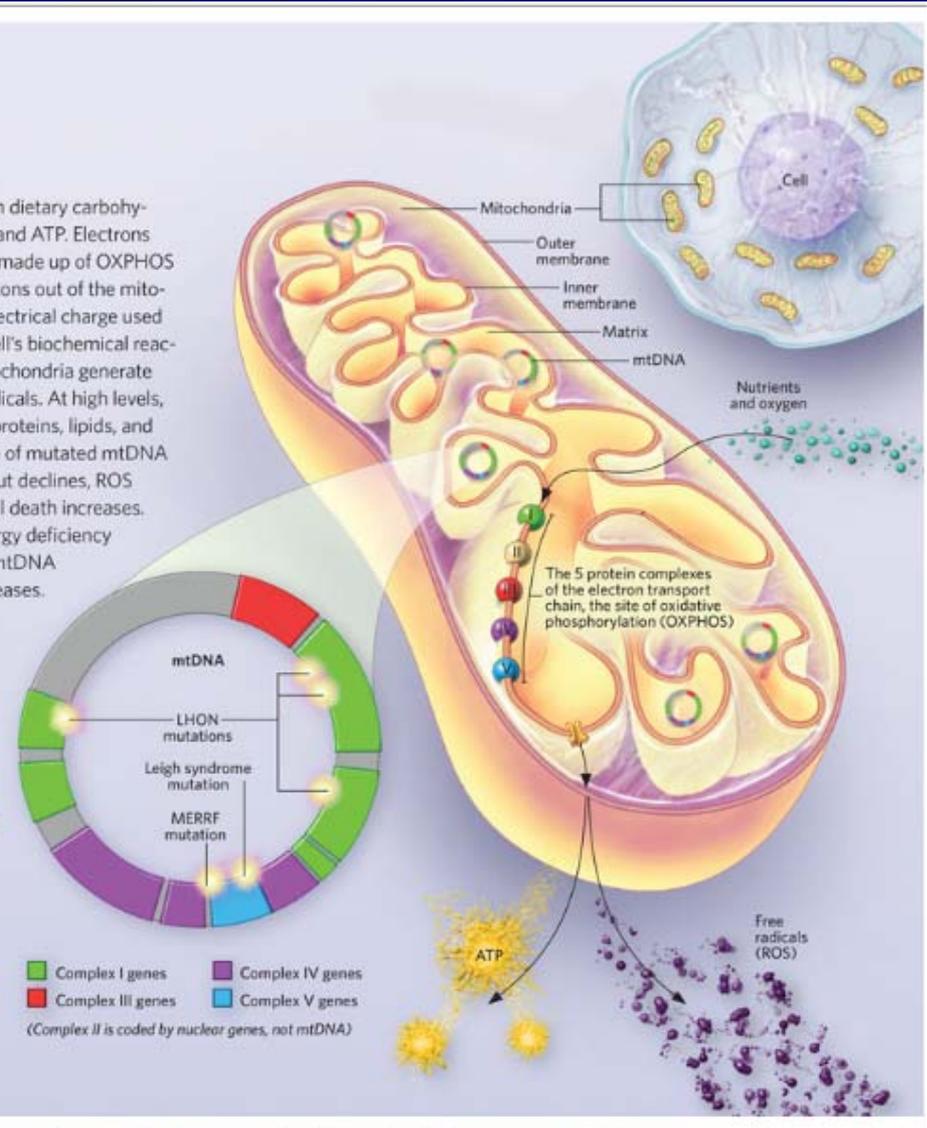
This creates an **ELECTRICAL CHARGE** used to generate **ATP**, which powers most of the cell’s biochemical reactions.

MITOCHONDRIA AT WORK

Mitochondria combine hydrogen derived from dietary carbohydrates 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 inner membrane. This creates an electrical charge used to generate ATP, which powers most of the cell’s biochemical reactions. As a toxic by-product of OXPHOS, mitochondria generate reactive oxygen species (ROS), called free radicals. At high levels, free radicals damage mtDNA, nuclear DNA, proteins, lipids, and other molecules in the cell. As the percentage of mutated mtDNA in a cell increases, mitochondrial energy output declines, ROS production increases, and the likelihood of cell death increases. Through the work of Wallace and others, energy deficiency caused by these factors, as well as inherited mtDNA mutations, have been linked to numerous diseases.

mtDNA MUTATIONS

Mutations in the mtDNA genes can result in a wide range of symptoms. Several single-base changes in the complex I genes predispose to a person to Leber hereditary optic neuropathy (LHON), a form of inherited vision loss. Mutations in the complex V ATP synthase 6 gene can cause retinal problems when few mtDNA in a cell harbor the mutation, but can cause the lethal Leigh syndrome when many mtDNA in a cell have the mutation. Mutations in the ribosomal and transfer RNA genes in the mtDNA can predispose to deafness, muscle and heart disease, strokes, diabetes, Alzheimer’s and Parkinson’s. For example, a mutation in the tRNA(Lys) gene can cause a form of epilepsy together with muscle symptoms known as myoclonic epilepsy and ragged-red fiber disease (MERRF).



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.

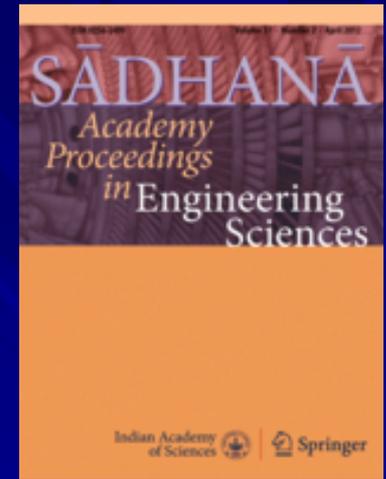
Biomaterials and magnetism

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, tissue 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 tissue 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

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

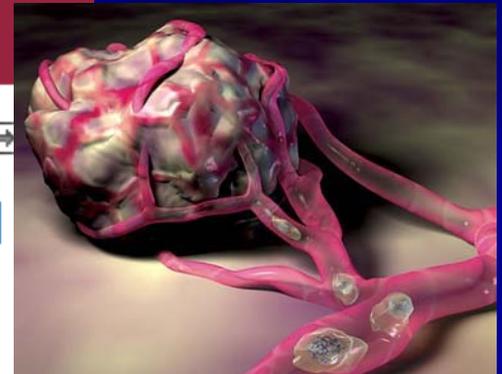
Michael F. Flessner, Jaewah Choi, Kimberly Credit, Ravi Deverkadra and Karla Henderson

Author A

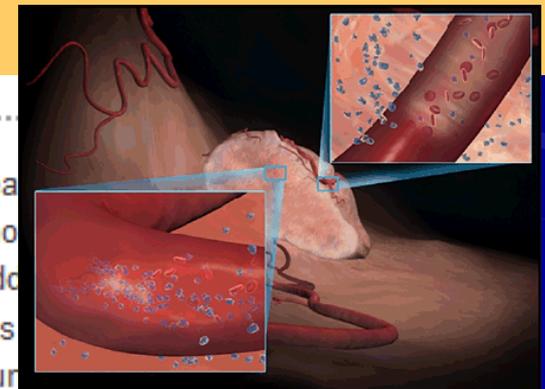
Requests for reprints to:
Michael F. Flessner, MD
Mississippi
601-984-5688

Abstract

Purpose: Despite evidence that regional chemotherapy improves the treatment of metastatic peritoneal ovarian carcinoma, monoclonal antibodies have not had significant success in i.p. delivery. The present study was designed to address the hypothesis that convective penetration of macromolecular antineoplastic agents occurs on a positive pressure difference between the i.p. therapeutic solution and the tumor.



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.

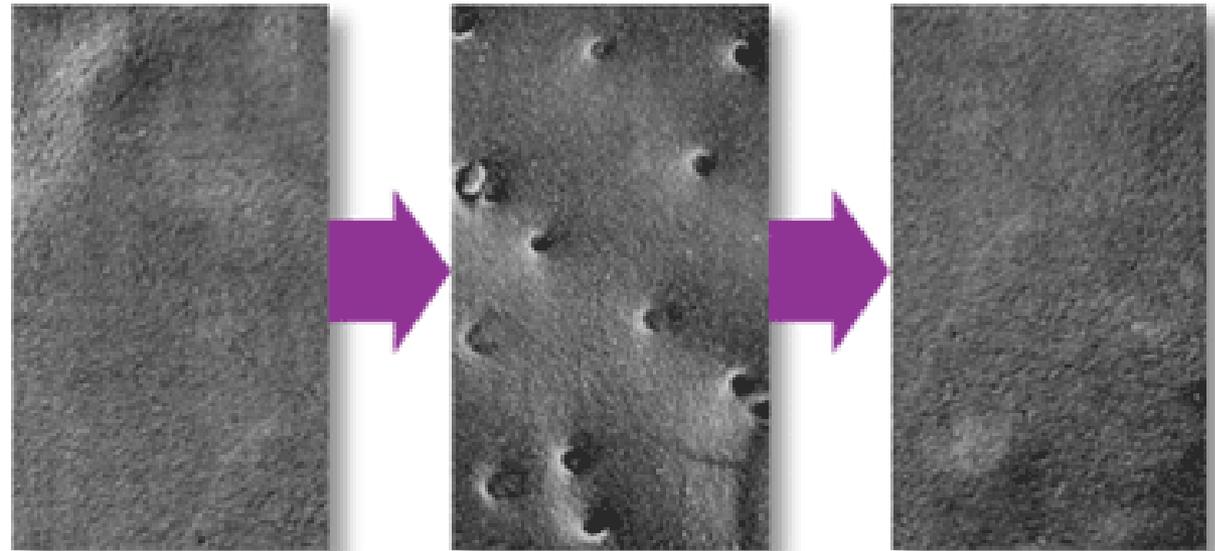


PEMF induces Electro-poration – Increasing Cellular (TMP) Transmembrane Potential

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-aging and treating chronic diseases including cancer.

The phenomenon of electroporation



Cell membrane before pulsing

Cell membrane during pulsing

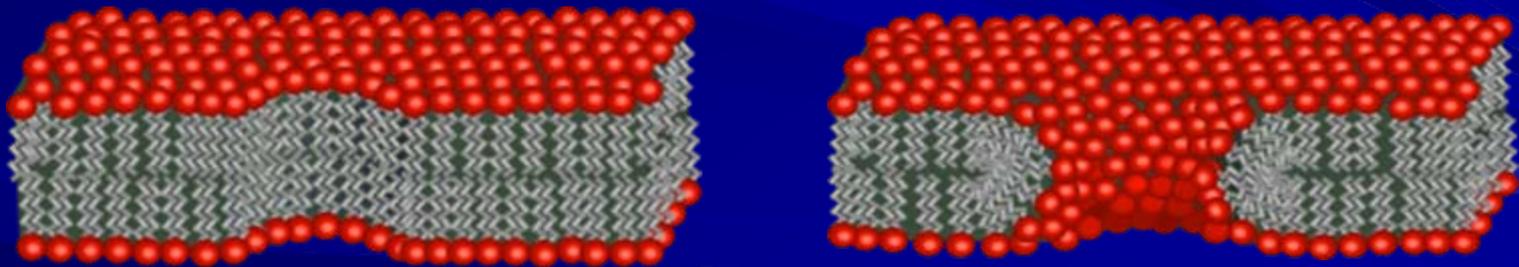
Cell membrane after pulsing
(cell returns to

- *Controlled, millisecond electrical pulses induce temporary pores in the cell membrane*
- *Cell membrane reseals and is left unharmed*

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).

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 Triphosphate) 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.

'If Physical Exercise Were a Drug, It Would Be Hitting the Headlines' ; Exercise Can Help Cancer Survivors, Says New Report

Macmillan Cancer Support - <http://www.macmillan.org.uk>

08-09-11

EXERCISE is a "wonder drug" for cancer survivors and may even prevent the disease coming back, according to a report published today. Macmillan Cancer Support said physical activity should be "prescribed" by doctors after "hard evidence" showed it can significantly help recovery and prevent other long-term illnesses.

The research also showed exercise had an impact on *preventing recurrence* of a few specific cancers.

- Women with **breast cancer** who exercise for **150 minutes a week at moderate intensity** have a **more than 40% lower risk of dying** and recurrence of disease compared to women who are active for less than one hour a week.
- Results of two studies on **bowel cancer** also show the **risk of dying or the disease coming back is cut by about 50%** in patients taking six hours a week of moderate intensity exercise.
- **Prostate cancer patients** have around a **30% lower risk of dying** from the disease and a **57% lower rate of disease progression** if they do three hours of moderate intensity exercise a week.

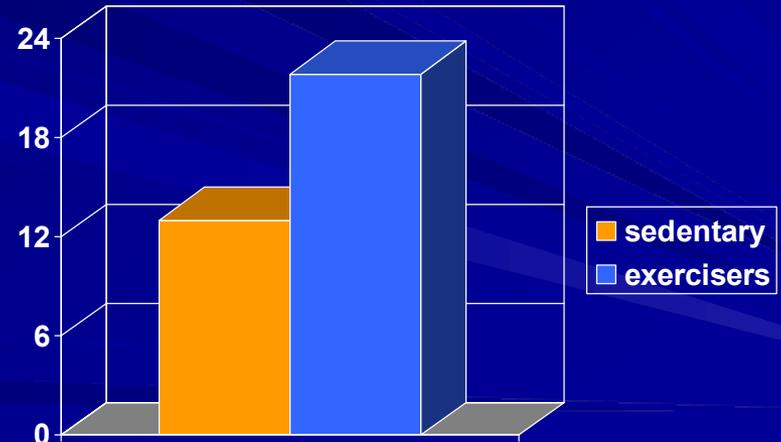
Exercise Associated With Longer Survival After Brain Cancer Diagnosis

ScienceDaily (June 21, 2011)

Brain cancer patients who are able to exercise live significantly longer than sedentary patients, scientists at the Duke Cancer Institute report.

The finding, published online June 20 in the *Journal of Clinical Oncology*, adds to recent research that exercise improves how cancer patients feel during and after treatments, and may also extend their lives. The study enrolled 243 patients at the Preston Robert Tisch Brain Tumor Center at Duke with advanced recurrent gliomas, lethal brain malignancies that typically result in a median life expectancy of less than six months.

The patients who reported participating in regular, brisk exercise - the equivalent of an energetic walk five days a week for 30 minutes, had significantly prolonged survival, living a median 21.84 months vs. 13.03 months for the most sedentary patients.



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 could become choked with trash and malfunction or die. In recent years, some scientists have begun to suspect that faulty autophagy mechanisms contribute to the development of a range of diseases, including diabetes, muscular dystrophy, Alzheimer's and cancer. The slowing of autophagy as we reach middle age is also believed to play a role in aging.

Autophagy in Human Health and Disease

Augustine M.K. Choi, M.D., Stefan W. Ryter, Ph.D., and Beth Levine, M.D. N Engl J Med 2013; 368:651-662
February 14, 2013



The NEW ENGLAND
JOURNAL of MEDICINE

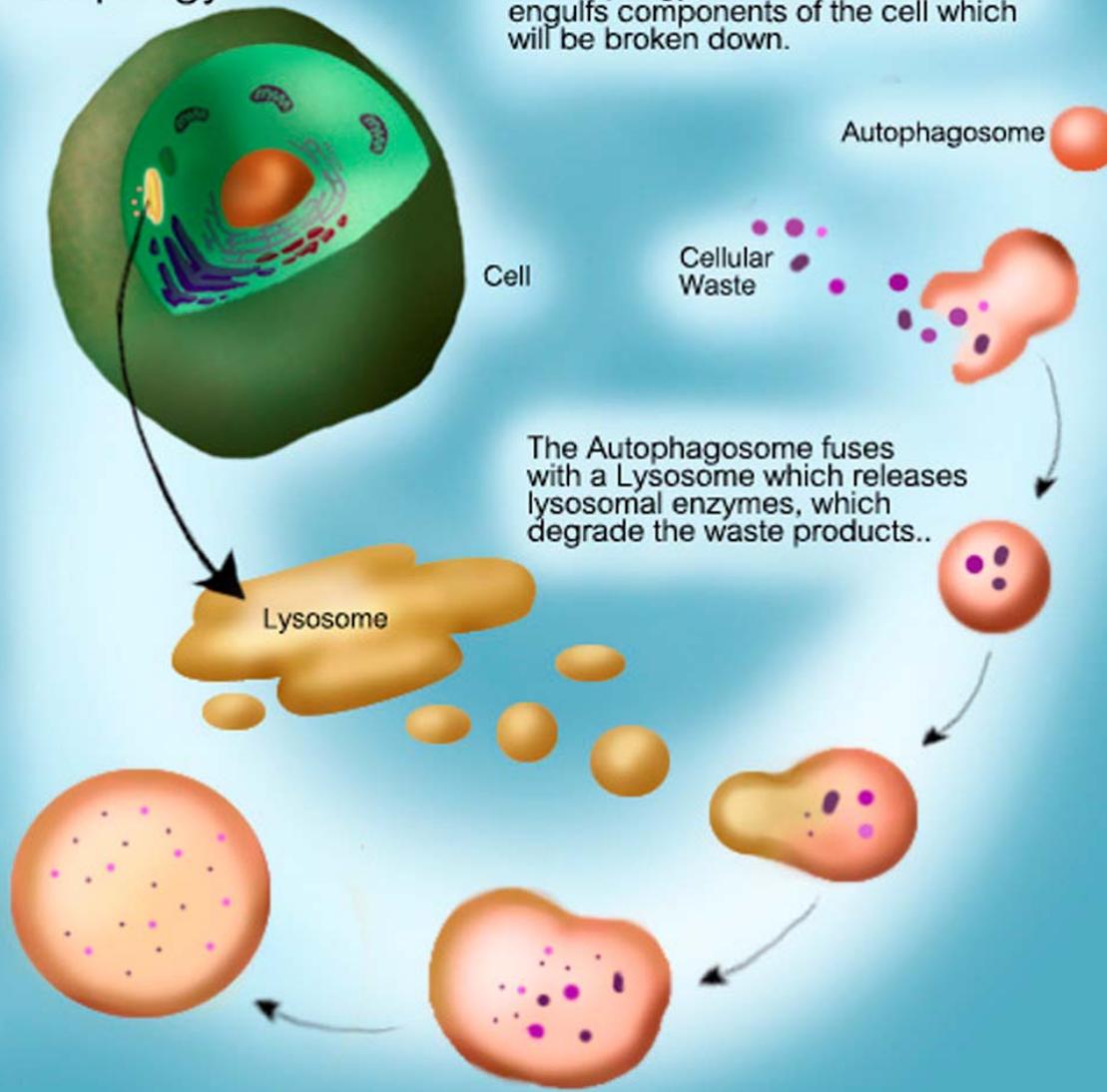
This review discusses the cellular process of autophagy (“self-eating”), which plays key roles in normal development of the immune system and adaptation to stress, as well as in a wide range of disease states.

During exercise, autophagy is increased in cardiac and skeletal muscle, adipose tissue, and pancreatic beta cells. In mice, exercise-induced autophagy provides protection against glucose intolerance associated with a high-fat diet

Without this efficient system, cells could become choked with trash and malfunction or die. In recent years, some scientists have begun to suspect that faulty autophagy mechanisms contribute to the development of a range of diseases, including diabetes, muscular dystrophy, Alzheimer’s and cancer. The slowing of autophagy as we reach middle age is also believed to play a role in aging.

Autophagy

In Autophagy, a membrane forms and engulfs components of the cell which will be broken down.



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.

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.****

Plant Physiol. 2007 January; 143(1): 291–299.

Degradation of Oxidized Proteins by Autophagy during Oxidative Stress in Arabidopsis

Yan Xiong, Anthony L. Contento, Phan Quang Nguyen, and Diane C. Bassham*



Upon encountering oxidative stress, proteins are oxidized extensively by highly reactive and toxic reactive oxidative species, and these damaged, oxidized proteins need to be degraded rapidly and effectively. There are two major proteolytic systems for bulk degradation in eukaryotes, the proteasome and vacuolar autophagy. In mammalian cells, the 20S proteasome and a specific type of vacuolar autophagy, chaperone-mediated autophagy, are involved in the degradation of oxidized proteins in mild oxidative stress.

Using two macroautophagy markers, monodansylcadaverine and green fluorescent protein-AtATG8e, we here show that application of hydrogen peroxide or the reactive oxidative species inducer methyl viologen can induce macroautophagy in Arabidopsis (*Arabidopsis thaliana*) plants. Macroautophagy-defective RNAi-*AtATG18a* transgenic plants are more sensitive to methyl viologen treatment than wild-type plants and accumulate a higher level of oxidized proteins due to a lower degradation rate. In the presence of a vacuolar H⁺-ATPase inhibitor, concanamycin A, oxidized proteins were detected in the vacuole of wild-type root cells but not RNAi-*AtATG18a* root cells.

Together, our results indicate that autophagy is involved in degrading oxidized proteins under oxidative stress conditions in Arabidopsis.

Bill Doyle: Treating cancer with electric fields



The [Tumor Treating Fields] patients can undergo all the activities of their daily life. There's none of the tiredness. There's none of what is called the 'chemo head.'

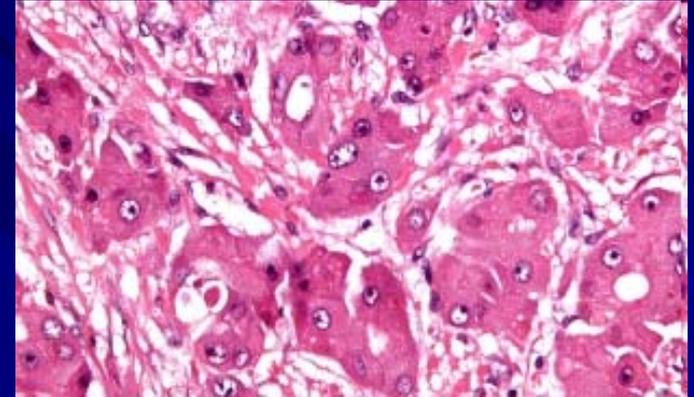
Surgery, chemotherapy and radiation are the best-known methods for treating cancer. At TEDMED, Bill Doyle presents a new approach, called Tumor Treating Fields, which uses electric fields to interrupt cancer cell division. Still in its infancy -- and approved for only certain types of cancer -- the treatment comes with one big benefit: quality of life. With his company Novocure, Bill Doyle works to bring breakthrough medical technologies to doctors and patients.

Electromagnetic Fields Shrink Tumors

New research shows that low-intensity fields can inhibit cancer cell proliferation.

By Bob Grant | The Scientist | January 11, 2012

Researchers have demonstrated that small doses of **electromagnetism can shrink liver and breast cancer cells without harming surrounding tissues**, according to a report published recently in the *British Journal of Cancer*.



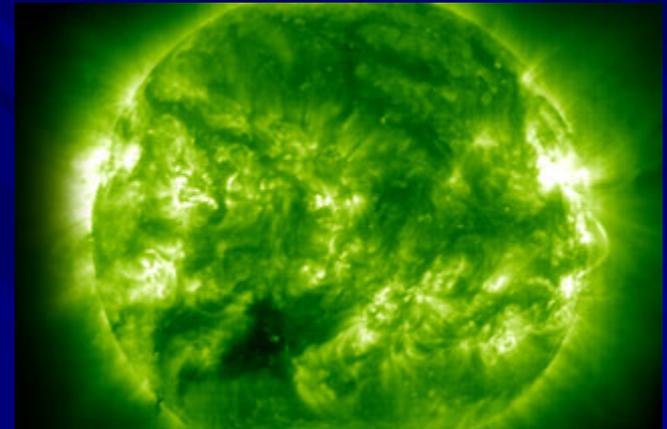
Very high magnification micrograph of fibrolamellar hepatocellular carcinoma
Wikimedia Commons, Nephron

An international team, led by University of Alabama at Birmingham oncologist Boris Pasche, has shown that low-intensity electromagnetic fields can slow the proliferation of and hepatocellular carcinoma (HCC) cells, which are involved with a deadly form of liver cancer, and breast cancer cells. *“This is a truly novel technique,”* Pasche told *The Guardian*. *“It is innocuous, can be tolerated for long periods of time, and could be used in combination with other therapies.”*

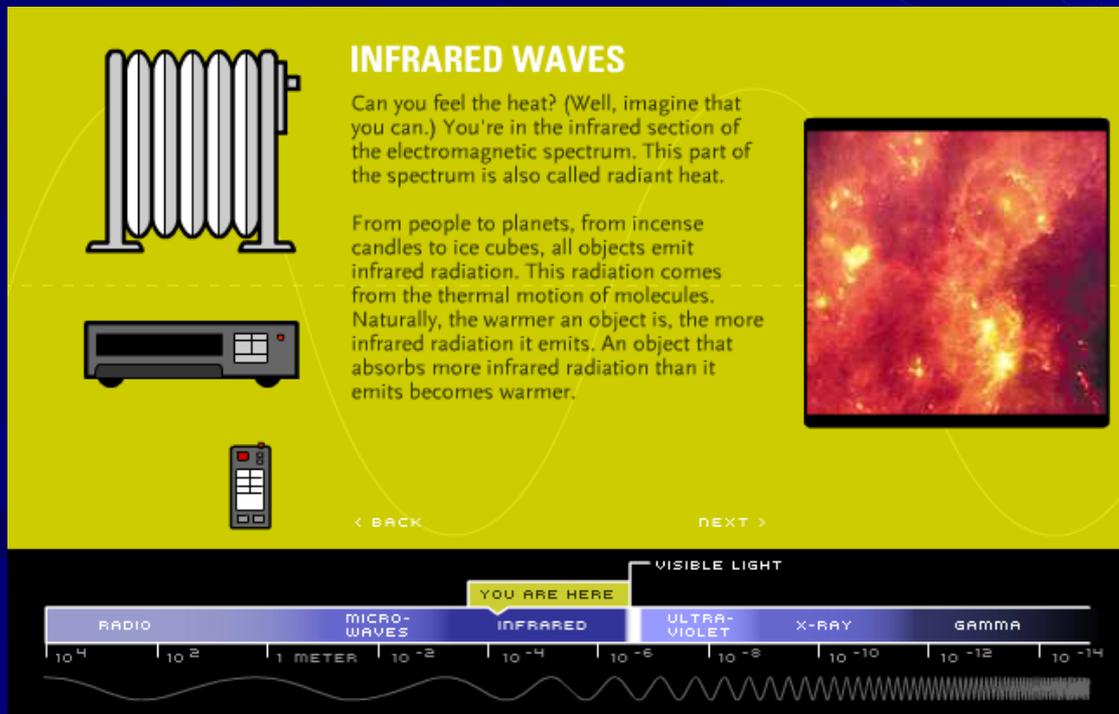
In August, Pasche and his colleagues published a *British Journal of Cancer* paper showing that they could slow tumor growth in some HCC patients by treating them with low-level electromagnetic fields on a regular basis. In total, 41 patients received the treatments... after 6 months of treatment, tumor growth in 14 of those patients had stabilized, and **none experienced negative side effects**.

Electromagnetic Spectrum

Light, heat, radio signals, and medical X-rays are all forms of electromagnetic radiation—waves moving through space that are delivered by mass-less particles called **photons**.



The only thing that differentiates one type of electromagnetic radiation from any other is the **energy carried by its photons**.



Electromagnetic energy is created at the atomic level, as electrons release energy while switching from higher- to lower-energy orbits, or while freeing themselves from atomic bonds. This movement of electrons results from the need to maintain energy balance within the atom under the input of some form of external energy.

Cells emit and absorb photons...

Photons are energy particles or waves of light in the electromagnetic spectrum. A bio-photon is a photon of light emitted from a biological system, and detected by biological probes as part of the general electromagnetic radiation of living biological cells.



Cells absorb photons and transform their energy into ATP (Adenosine Triphosphate), which is a form of energy that cells utilize. The resulting ATP is then used to power metabolic processes and to synthesize DNA and RNA (proteins & enzymes.) ATP is needed to repair and regenerate cellular components, foster an abundance of cell reproduction, and increase circulation thus restoring balance to the body.

Quantum biology-based energy medicine, including LED light therapy, low level laser therapy (LLLT), ultrasound resonance therapy, and pulsed electromagnetic frequency (PEMF) therapy, are all safe, effective, non-invasive modalities being used to restore cellular energy and functioning.

Effect of NASA light-emitting diode (LED) irradiation on wound healing

Harry T. Whelan, Robert L. Smits, Jr., Ellen V. Buchmann, Noel T. Whelan, et al.

Two approaches that specifically address the identified pathophysiological processes involved in wound healing are hyperbaric oxygen therapy and light therapy.

We believe that the use of NASA Light-Emitting Diodes (LED) for light therapy alone, and in conjunction with hyperbaric oxygen, will greatly enhance the natural wound healing process. This will save valuable time and resources for both patients and health care facilities.

Furthermore, improved wound healing will reduce the risk of infection for the patient, decrease the amount of costly dressings required, and more quickly return the patient to a pre-injury/illness level of activity.

Wound healing has three phases: first a substrate is laid down, then cells proliferate, and finally there is remodeling of tissue. It has been demonstrated that mitochondria are receptive to monochromatic near-infrared light and that laser light likely increases respiratory metabolism of certain cells. Processes such as fibroblast proliferation, attachment and synthesis of collagen and procollagen, growth factor production [including keratinocyte growth factor (KGF), transforming growth factor (TGF) and platelet-derived growth factor (PDGF)], macrophage stimulation, lymphocyte stimulation and greater rate of extracellular matrix production have been reported with laser light treatment.

Mitochondrial signal transduction in accelerated wound and retinal healing by near-infrared light therapy

Janis T. Eellsa, Margaret T.T. Wong-Rileyb, James VerHoevec, et al.

Mitochondrion. Volume 4, Issues 5–6, September 2004, Pages 559–567



Photobiomodulation by light in the red to near infrared range (630–1000 nm) using low energy lasers or light-emitting diode (LED) arrays has been shown to accelerate wound healing, improve recovery from ischemic injury in the heart, and attenuate degeneration in the injured optic nerve.

Recent evidence indicates that the therapeutic effects of red to near infrared light result, in part, from intracellular signaling mechanisms triggered by the interaction of NIR light with the mitochondrial photoacceptor molecule cytochrome c oxidase.

We have demonstrated that NIR-LED photo-irradiation increases the production of cytochrome oxidase in cultured primary neurons and reverses the reduction of cytochrome oxidase activity produced by metabolic inhibitors.

Gene discovery studies conducted using microarray technology documented a significant upregulation of gene expression in pathways involved in mitochondrial energy production and antioxidant cellular protection. These findings provide a link between the actions of red to near infrared light on mitochondrial oxidative metabolism in vitro and cell injury in vivo. NIR-LED photobiomodulation represents an innovative and non-invasive therapeutic approach for the treatment of tissue injury and disease processes in which mitochondrial dysfunction is postulated to play a role including diabetic retinopathy, age-related macular degeneration, Leber's hereditary optic neuropathy and Parkinson's disease.

Journal of Neurotrauma. November 2010, 27(11): 2107-2119.

Near Infrared Light Reduces Oxidative Stress and Preserves function in CNS Tissue Vulnerable to Secondary Degeneration following Partial Transection of the Optic Nerve

Melinda Fitzgerald, Carole A. Bartlett, Sophie C. Payne, Nathan S. Hart, Jenny Rodger, Alan R. Harvey, and Sarah A. Dunlop.

Traumatic injury to the central nervous system (CNS) is accompanied by the spreading damage of secondary degeneration, resulting in further loss of neurons and function. Partial transection of the optic nerve (ON) has been used as a model of secondary degeneration, in which axons of retinal ganglion cells in the ventral ON are spared from initial dorsal injury, but are vulnerable to secondary degeneration.

We have recently demonstrated that early after partial ON injury, oxidative stress spreads through the ventral ON vulnerable to secondary degeneration via astrocytes, and persists in the nerve in aggregates of cellular debris. In this study, **we show that diffuse transcranial irradiation of the injury site with far red to near infrared (NIR) light (WARP 10 LED array, center wavelength 670 nm, irradiance 252 W/m⁻², 30 min exposure), as opposed to perception of light at this wavelength, reduced oxidative stress in areas of the ON vulnerable to secondary degeneration following partial injury.**

The WARP 10 NIR light treatment also prevented increases in NG-2-immunopositive oligodendrocyte precursor cells (OPCs) that occurred in ventral ON as a result of partial ON transection. Importantly, normal visual function was restored by NIR light treatment with the WARP 10 LED array, as assessed using optokinetic nystagmus and the Y-maze pattern discrimination task. To our knowledge, this is the first demonstration that 670-nm NIR light can reduce oxidative stress and improve function in the CNS following traumatic injury *in vivo*.

Visible light-induced killing of bacteria as a function of wavelength: Implication for wound healing

Anat Lipovsky MSc, Yeshayahu Nitzan PhD, Aharon Gedanken PhD, Dr. Rachel Lubart PhD



Visible light (400–800 nm) at high intensity was previously found to kill bacteria that are frequently found in infected wounds, while low-power white light enhances bacterial proliferation. **The phototoxic effect was found to involve induction of reactive oxygen species (ROS) production by the bacteria.** The aim of the present study was to identify the most effective wavelengths in the visible range for inducing a bactericidal effect.

ROS production in *Staphylococcus aureus* and *Escherichia coli* as a function of wavelengths in the visible range (400–500, 500–800, 415, and 455 nm) was studied using the electron paramagnetic resonance (EPR) spin trapping technique.

ROS production following blue (400–500 nm) light illumination was found to be higher than that of red (500–800 nm). Within the blue range, light of 415 nm induced more ROS than 455 nm, which correlated with results obtained for the reduction in colony count of *S. aureus* and *E. coli* following illumination using equal intensities of these two wavelengths. At low fluencies, both 415 and 455 nm enhanced proliferation of *S. aureus* but reduced viability of *E. coli*.

Intense blue light, preferably at 415 nm, could be used for bacterial eradication. However, it should be noted that low intensity of visible light can be dangerous since it may promote proliferation of the microorganisms.

Photomed Laser Surg. 2010 Apr;28(2):159-60. doi: 10.1089/pho.2010.2789.

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

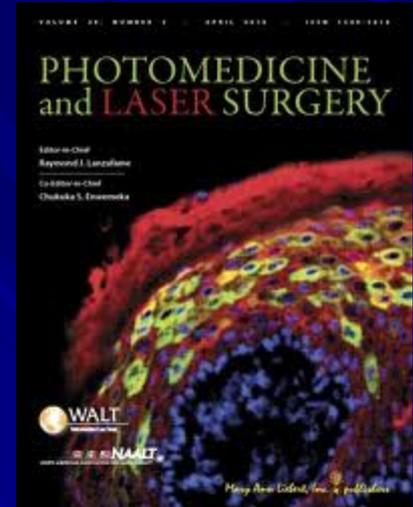
Karu T.

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 *600–1,000nm to treat in a nondestructive and nonthermal 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.**

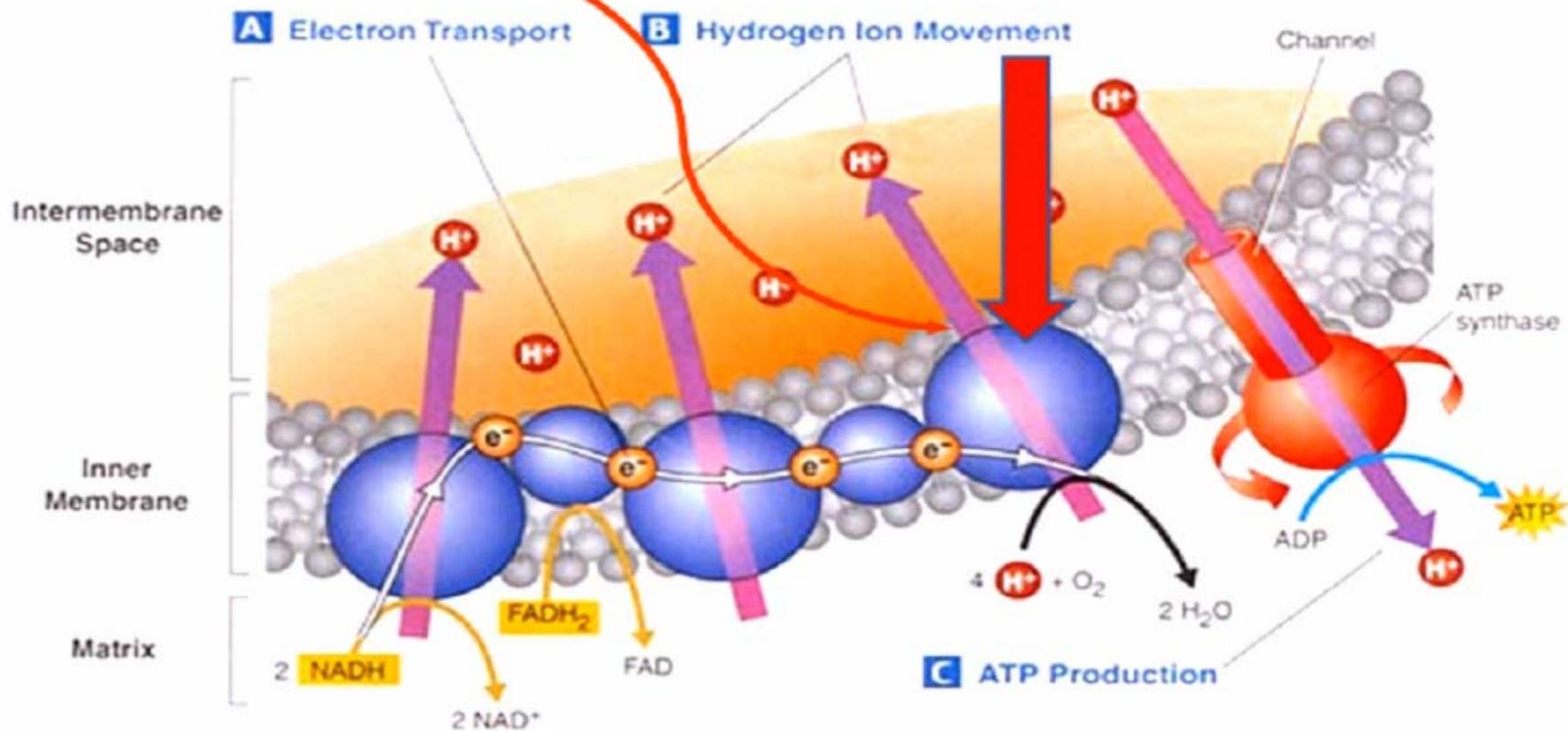
It is generally accepted that the mitochondria are the initial site of light action in cells, and cytochrome c oxidase (the terminal enzyme of the mitochondrial respiratory chain) is the responsible molecule.

This multiplicity of conditions treated with photobiomodulation has persuaded many unbelievers of the value of such an universal method.



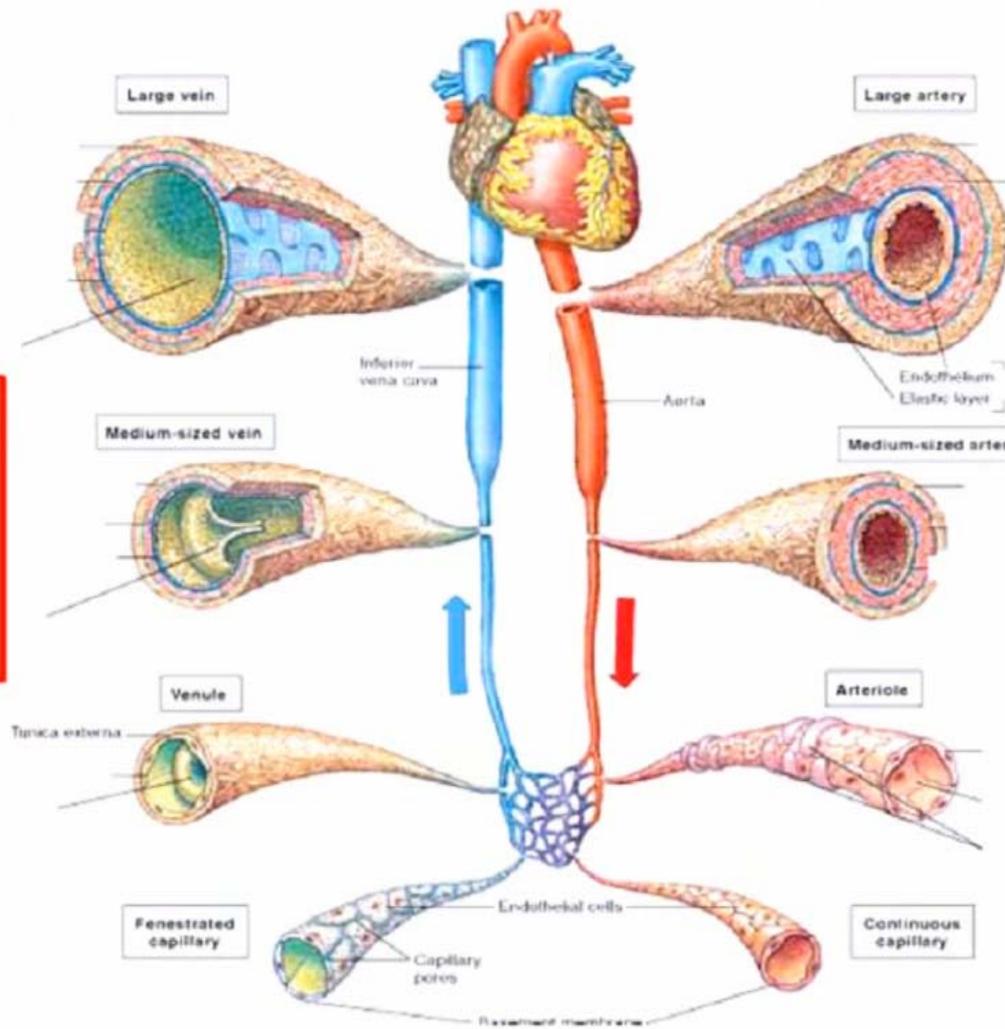
Light Activation on Mitochondria producing ATP

LIGHT (PHOTONS)



The Endothelium – Our “Third” Brain

Our Heart and Vessels: Veins and Arteries, Arteriole and Capillaries



Venous and Arterial System Health Are Dependent on:

Endothelium Integral Health

Why the Endothelium is considered an “Organ”

- *It is because of:*
- > it is Responsive to Stimuli: Photonic (Quantum), Ionic, osmotic, Biochemical: Hormonal and Enzymatic

- It is highly responsive to the photonic signaling from
Nitric Oxide

- There are many published materials about the **benefits of nitric oxide**, but it all started to gain a attention from the work of three scientists. In 1998, three American scientists were awarded the Nobel Prize in Medicine for discovering a vascular signaling molecule called nitric oxide. Why, you say is nitric oxide so important? Nitric oxide revolutionized conventional scientific reasoning because it was the first molecule discovered to literally communicate with another molecule. Why does that matter? When nitric oxide is made by the cells in our blood vessels, it signals the surrounding arterial tissues “telling” them to relax. **Nitric oxide optimizes circulation and is involved in virtually every organ system in our body.**

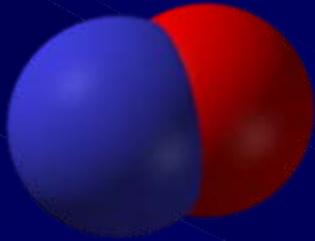
Nitric Oxide: The New Hero of Human Biology

By Marissa Kantor - February 08, 2005

A few extra minutes on the stairmaster might be more beneficial than you thought. A longer workout means more production of nitric oxide -- an enzyme that could save you from heart disease and other problems.

Three scientists, Louis Ignarro, Robert Furchgott and Ferid Murad, were the first to identify the artery-dilating properties of nitric oxide – and won them a nobel prize. Specifically, they identified the following process: the innermost layer of cells (**called the endothelium**) releases nitric oxide when triggered by the enzyme nitric oxide synthase.

Researchers are continuing to study the possible uses of nitric oxide and its link to heart disease prevention. In the meantime, scientists recommend that you maximize nitric oxide production in your body by following routines that hopefully are already familiar to you: a **low-fat diet, mild to moderate exercise, smoking cessation and better "cellular nutrition."** This includes consuming antioxidants like **vitamins A and C, which prevent the breakdown (oxidation) of nitric oxide in the body.**



What is Nitric Oxide (NO)?

NO is an important cellular signaling molecule and neurotransmitter involved in many physiological and pathological processes.

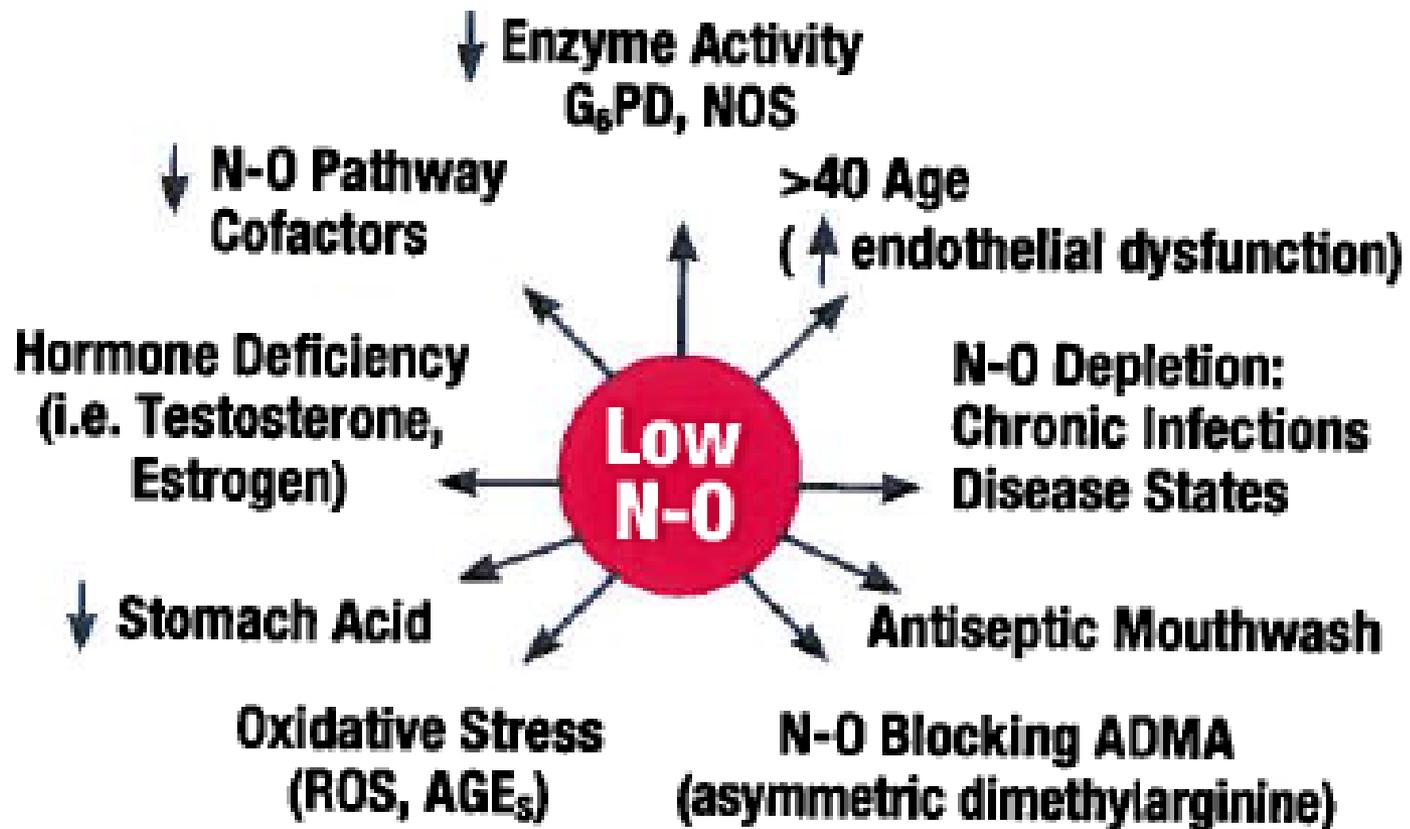
NO is a powerful vasodilator with a short half-life of a few seconds in the blood, but it affects blood flow and nerve responses very rapidly. Small increases in NO lead to increased circulation and to better sensory perception.

NO metabolism is necessary for normal circulation (venous, arterial, and lymph flows) and for the ability to sense pain, temperature, and pressure. Diabetic patients have deficits in circulation, which often lead to blindness, kidney dysfunction, heart disease, and ulcers in the lower legs.

Pharmaceuticals that work on increasing NO are nitro-glycerin, used for angina and other heart conditions, and Viagra® (sildenafil), the popular “magic blue pill” that is widely prescribed for erectile dysfunction.

Tissues that are hypoxic (deprived of good, normal circulation) can not produce as much NO as do normal, well-oxygenated tissues.

Some Causes of Low N-O



**Over the last 20 years,
research has linked the loss of NO
with many symptoms of aging:**

- Dementia
- Eye Disease
- Infection
- Insomnia
- Depression
- Osteoporosis
- Erectile Dysfunction
- Hypertension
- Atherosclerosis/Arteriosclerosis
- Diabetes
- Fatigue/Sedentary lifestyle
- Asthma/COPD/Lung disease
- Bladder dysfunction
- Kidney disease
- Premature-aged skin (AGEs)
- Poor wound healing
- Hormone imbalance
- Abnormal cell growth
- Poor neurotransmitter function
- Abnormal blood viscosity

Relevance of nitric oxide in pain mechanisms and pain management

Piotr K. Janicki MD, PhD; Magdalena Jeske-Janicka MD

Abstract

Nitric oxide (NO) may be involved in the mechanisms of pain generation and transmission throughout the central and peripheral nervous systems (including brain and spinal cord and perivascular tissue and peripheral nerve terminals) and locally released pain mediators (including formation of inflammation and vascular edema).

NO mechanisms are also involved in the analgesic activity of nonsteroidal anti-inflammatory drugs, opioids, and local anesthetics. These novel observations dictate new approaches to the pharmacologic treatment of migraine, neuropathic pain, and other forms of chronic, intractable pain that are resistant to classical pharmacotherapy.

The new strategies of pharmacologic pain treatment are increasing rapidly due to the availability of new drugs modulating the NO-activated cascade and soon may be available for clinical use.

<http://link.springer.com/article/10.1007%2Fs11916-998-0022-5?LI=true>



PEMF Therapy and Nitric Oxide Production

Many cells in the body produce nitric oxide; however, its production by the vascular endothelium is particularly important in the regulation of blood flow. Abnormal production of nitric oxide, as occurs in different disease states, can adversely affect blood flow and other vascular functions. Nitric oxide is one of the few gaseous signaling molecules known and is additionally exceptional due to the fact that it is a radical gas. It is a key vertebrate biological messenger, playing a role in biological processes.

The March/April 2009 Aesthetic Surgery Journal published a study:

“Evidence-Based Use of Pulsed Electromagnetic Field Therapy in Clinical Plastic Surgery” that summarizes the evolution in the understanding of the physiological effects of PEMF therapy on cells and tissues.

Studies emerged suggesting that PEMF could modulate the production of growth factors and began to focus on enzyme systems with well-characterized calcium (Ca²⁺) dependence.

Nitric oxide mediates the effects of pulsed electro magnetic field stimulation on the osteoblast proliferation and differentiation.

Diniz P, Soejima K, Ito G.

Department of Orthodontics, Kagoshima University Dental School, 8-35-1 Sakuragaoka, Kagoshima 890-8544, Japan.

Abstract

The purpose of this research was to investigate whether the effects of pulsed electro-magnetic field (PEMF) stimulation on the osteoblast proliferation and differentiation are mediated by the increase in the nitric oxide (NO, nitrogen monoxide) synthesis.

PEMF stimulation increased significantly the nitrite concentration in the -NMMA group on the 3rd, 7th, and 15th days of culture. However, this effect was partially blocked in the +NMMA group. The DNA content in the -NMMA group, but not in the +NMMA group, increased significantly on the 3rd and 7th days of culture. The AIPase activity in the P-NMMA and D-NMMA groups, but not in the P+NMMA and D+NMMA groups, also increased significantly.

In conclusion, the **PEMF stimulatory effects on the osteoblasts proliferation and differentiation were mediated by the increase in the NO synthesis.**

Exp Mol Med. 2002 Mar 31;34(1):53-9.

Enhanced expression of neuronal nitric oxide synthase and phospholipase C-gamma1 in regenerating murine neuronal cells by pulsed electromagnetic field.

Kim SS, Shin HJ, Eom DW, Huh JR, Woo Y, Kim H, Ryu SH, Suh PG, Kim MJ, Kim JY, Koo TW, Cho YH, Chung SM.
Department of Pathology, Ulsan University College of Medicine, Korea.



Abstract

Pulsed electromagnetic field (PEMF) has been shown to improve the rate of peripheral nerve regeneration. In the present study we investigated the expression of neuronal nitric oxide synthase (nNOS) and phospholipase C-gamma1 (PLC-gamma1) in regenerating rat laryngeal nerves during the exposure to PEMF after surgical transection and reanastomosis.

Axons were found to regenerate into the distal stump nearly twice faster in PEMF-exposed animals than in the control. Consistently, motor function was better recovered in PEMF-treated rats. The expression of nNOS and PLC-gamma1 was highly enhanced in the regenerated nerves.

Exercise Protects the Heart Via Nitric Oxide

Research , School of Medicine
May 4, 2011



EMORY

WOODRUFF
HEALTH
SCIENCES
CENTER

Exercise both reduces the risk of a heart attack and protects the heart from injury if a heart attack does occur. For years, doctors have been trying to dissect how this second benefit of exercise works, with the aim of finding ways to protect the heart after a heart attack.

Researchers at Emory University School of Medicine have identified the ability of the heart to produce and store nitric oxide as an important way in which exercise protects the heart from injury.

Nitric oxide, a short-lived gas generated within the body, turns on chemical pathways that relax blood vessels to increase blood flow and activate survival pathways. Both the chemical nitrite and nitrosothiols, where nitric oxide is attached to proteins via sulfur, appear to act as convertible reservoirs for nitric oxide in situations where the body needs it, such as a lack of blood flow or oxygen.

In experiments with mice, the researchers showed that four weeks of being able to run on a wheel protected the mice from having a blocked coronary artery; the amount of heart muscle damaged by the blockage was less after the exercise period. Importantly, the mice were still protected a week after the wheel was taken away.



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

Article by Martin Milner, ND featured in the May 2010 issue of Townsend Newsletter, along with article by Dr. Garry Gordon “Chelation and Cardiovascular Disease”

TOWNSEND LETTER – MAY 2010

Reversal of a Case of Advanced Coronary Artery Disease with Unstable Angina Using Pulsed Electromagnetic Field (PEMF) Cellular Exercise by Martin Milner, ND

It is wonderful to both the patient and physician when, after years of failed trials in both conventional and alternative medicine, a safe, natural method of cellular exercise makes dramatic change in a case of serious chronic disease. This case is an extraordinary example of reversing end-stage coronary artery disease with pulsed electromagnetic field cellular exercise (PEMF). The case also elucidates critical monitoring and decision-making horizons throughout patient management.

The Case

SH, a 65-year-old, very pleasant white Caucasian female, presented to our clinic with advanced coronary artery disease, diabetes, hypertension, and obesity. Her cardiac history began in 1996, when she went into cardiac arrest and was successfully defibrillated and brought back to life. She did lose sensation in two of her toes at discharge from this hospitalization. This loss of sensation was presumed to be a complication of chest defibrillation. During this hospitalization, significant ischemic heart disease was diagnosed on cardiac catheterization, and two stents were deployed into the left anterior descending and right circumflex coronary artery.

Progression to Advanced Coronary Artery Disease

As time progressed, her disease advanced, and a second angiogram involved the deployment of a third stent in her left anterior descending coronary artery. Her ischemic heart disease progressed further, and in 2005 she underwent three vessel coronary artery bypass graph surgery where the LAD stents were bypassed along with bypass surgery of the left circumflex and bypassing a new occlusion in the right anterior descending coronary artery. At the time of this

The Role of Nitric Oxide in Low Level Light Therapy (LLLT)

Hamblin MR

Wellman Center for Photomedicine, Massachusetts General Hospital, Department of Dermatology, Harvard Medical School, Harvard-MIT Division of Health Sciences and Technology

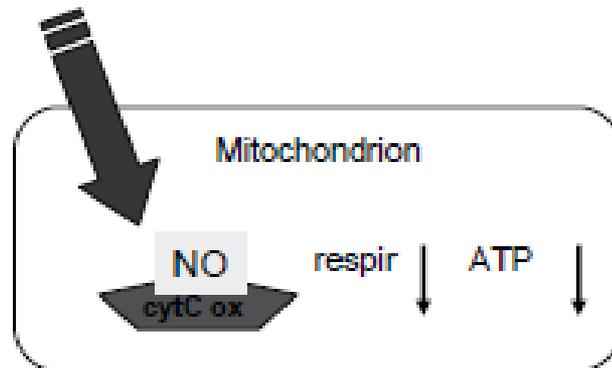
The use of low levels of visible or near infrared light for reducing pain, inflammation and edema, promoting healing of wounds, deeper tissues and nerves, and preventing tissue damage by reducing cellular apoptosis has been known for almost forty years since the invention of lasers.

Red and near-IR light is primarily absorbed by cytochrome c oxidase (unit four in the mitochondrial respiratory chain). Nitric oxide produced in the mitochondria can inhibit respiration by binding to cytochrome c oxidase and competitively displacing oxygen, especially in stressed or hypoxic cells. If light absorption displaced the nitric oxide and thus allowed the cytochrome c oxidase to recover and cellular respiration to resume, this would explain many of the observations made in LLLT.

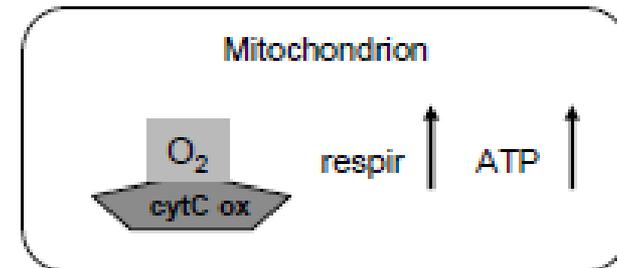
Studies suggest that mitochondria are responsible for the cellular response to red visible and NIR light; Increased proton electrochemical potential and ATP synthesis, increased RNA and protein synthesis, increase in polarographically measured oxygen uptake, major stimulation in the proton pumping activity, increased oxygen consumption, increased phosphate potential, energy charge and enhanced activities of NADH: ubiquinone oxidoreductase, ubiquinol: ferricytochrome c oxidoreductase and ferrocyanochrome C: oxygen oxidoreductase.

Effect of red or near IR light on cellular respiration, oxygenation

Red or NIR light



NO



Explains why:

Normal cells and tissue generally do not respond

Hypoxic cells, damaged cells, and tissue at risk of death respond well

Effects continue for long time after light is switched off

Released nitric oxide temporarily increases blood flow in illuminated area

Released nitric oxide reduces swelling by dilating lymphatics and increasing drainage

Michael Hamblin, Ph.D., R.Rox Anderson, M.D., Wellman Center for Photomedicine,
Massachusetts General Hospital, Boston, MA

Low Level Laser Energy

When our body absorbs the laser beam, a number of simultaneous chain reactions occur: blood flow invigoration, cell activity excitation, and intensification of inter-cell communication



Influences the permeability of cell membranes, the penetration of passage of the ions Ca^{++} , Na^{+} , K^{+} , and causes increased nerve activity

Increases ATP (adenosene triphosphate) levels, activates and stimulates enzymes in the target cells and cAMP molecules which carry inter-cell signals



Increases the synthesis of endorphins - hormones that relieve pain.

Increases S.O.D. (super oxide dismutase) which fights inflammation and reduces damage from free radicals

Activates immunization chain reactions; macrophage and mast cells that help in wound healing

Accelerates synthesis of collagen, elastin and keratinocytes – main components of the epidermis



Reduction in pain level

Anti-inflammatory activity

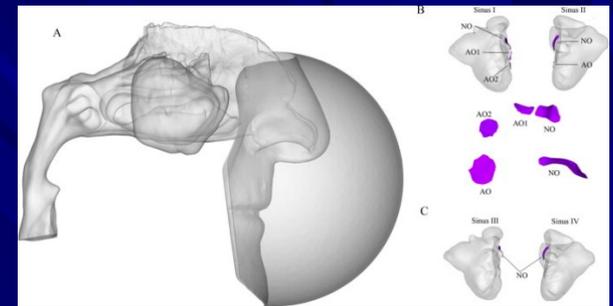
Faster wound healing and reduced scarring

Skin rejuventaing, anti-aging and wrinkle reduction

Nitric oxide and the paranasal sinuses.

Lundberg JO.

Karolinska Institutet, Department of Physiology and Pharmacology, Stockholm, Sweden. Jon.Lundberg@ki.se



Abstract

The discovery within the paranasal sinuses for the production of nitric oxide (NO) has altered the traditional explanations of sinus physiology... healthy paranasal sinus epithelium expresses an inducible NO synthase that continuously generates large amounts of NO, a pluripotent gaseous messenger with potent vasodilating, and antimicrobial activity.

This NO can be measured noninvasively in nasally exhaled breath. The role of NO in the sinuses is likely to enhance local host defense mechanisms via direct inhibition of pathogen growth and stimulation of mucociliary activity. The NO concentration in a healthy sinus exceeds those that are needed for antibacterial effects in vitro. In patients with primary ciliary dyskinesia (PCD) and in cystic fibrosis, nasal NO is extremely low. This defect NO generation likely contributes to the great susceptibility to chronic sinusitis in these patients. In addition, the low-nasal NO is of diagnostic value especially in PCD, where nasal NO is very low or absent. Intriguingly, NO gas from the nose and sinuses is inhaled with every breath and reaches the lungs in a more diluted form to enhance pulmonary oxygen uptake via local vasodilation. In this sense NO may be regarded as an "aerocrine" hormone that is produced in the nose and sinuses and transported to a distal site of action with every inhalation.

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Increase your Nitric oxide through Diet...

One way to increase your body's Nitric Oxide level is through the foods you eat. Beets, nuts, brown rice, artichokes, spinach, root vegetables, eggs, fish and poultry can help your body produce Nitric Oxide.



In certain regions of the world, such as the Mediterranean, the indigenous people have a low incidence of cardiovascular disease. Emerging research indicates the Mediterranean diet may promote Nitric Oxide production and help these people become more resistant to disease.

Additionally, foods rich in antioxidants, especially Vitamins C and E, have properties that can improve and help sustain Nitric Oxide levels.

The nitrate-nitrite-nitric oxide pathway was originally suggested and later demonstrated to be the mechanism for many of the health benefits from

vegetables. A telling point is that the DASH diet for reducing hypertension is higher in dietary nitrate. Though once vilified, without justification, nitrates and nitrites are now understood to play a very active and key role in causing healthy N-O formation that constitutes to a **clean, vibrant circulatory system... The Endothelium!**

Nitric Oxide Generating Capacity	Vegetable Varieties
Very low (<20)	Artichoke, asparagus, broad bean, eggplant, garlic, onion, green bean, mushroom, pea, pepper, potato, summer squash, sweet potato, tomato, watermelon
Low (20-<50)	Broccoli, carrot, cauliflower, cucumber, pumpkin, chicory
Middle (50-<100)	Cabbage, dill, turnip, savoy cabbage
High (100-<250)	Celeriac, Chinese cabbage, endive, fennel, kohlrabi, leek, parsley
Very high (>250)	Celery, cress, chervil, lettuce, red beetroot, spinach, rocket (rucola)



International Journal of Photoenergy
Volume 2012 (2012), Article ID 374861, 7 pages

Review Article: Photobiomodulation Process

Yang-Yi Xu,^{1,2} Timon Cheng-Yi Liu,¹ and Lei Cheng¹

Abstract

Photobiomodulation (PBM) is a modulation of laser irradiation, monochromatic light, hot color light such as red, orange or yellow, or cold color light such as green, blue or violet (LI) on biosystems.

Since its introduction in the early 1960s, laser has transformed phototherapy on biosystems. There is little research on PBM dynamics although its phenomena and mechanism have been widely studied. The PBM was discussed from dynamic viewpoint in this paper.

It was found that the primary process of cellular PBM might be the key process of cellular PBM so that the transition rate of cellular molecules can be extended to discuss the dose relationship of PBM.

There may be a dose zone in which low intensity LI (LIL at different doses) has biological effects similar to each other, so that biological information model of PBM might hold. LIL may self-adaptively modulate a chronic stress until it becomes successful.

Therapeutic Photobiomodulation: Nitric Oxide and a Novel Function of Mitochondrial Cytochrome C Oxidase

Robert O. Poyton, Kerri A. Ball

Institution: Department of Molecular, Cellular, and Developmental Biology,
University of Colorado. Published on February 20, 2011



Currently, light therapies are widely used in both human and veterinarian medicine. The application of light to clinical therapeutics includes:

- Photodynamic therapy, used to kill cancer cells;
- UVA therapies, used to treat a variety of skin diseases; and
- Photobiomodulation, used to promote cell growth and recovery from injury.

Photobiomodulation uses light emitting diodes (LEDs) or low energy lasers, which emit light in the visible red to near infrared range.

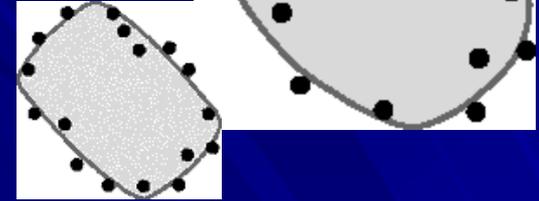
Three recent findings provide important new insights... First, nitric oxide has been implicated. Second, **cytochrome c oxidase**, an enzyme known to reduce oxygen to water at the end of the mitochondrial respiratory chain, has been shown to have a **new enzymatic activity -- the reduction of nitrite to nitric oxide.**

This nitrite reductase activity is elevated under hypoxic conditions but also occurs under normoxia. And third, **low intensity light enhances nitric oxide synthesis by cytochrome c oxidase without altering its ability to reduce oxygen.**

Ultrasonic Visualization And Stimulation Of Classical Oriental Acupuncture Points

Joie P. Jones, PhD - Young K. Bae, PhD

Medical Acupuncture, Vol 15, #2



Acupuncture has long been a major component of Oriental medicine and considerable evidence supports that acupuncture is effective in the treatment of pain and various disorders.

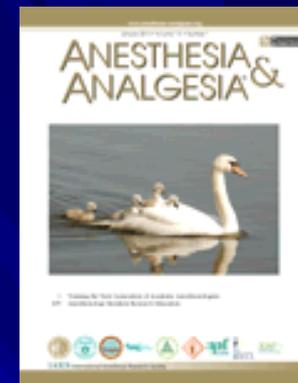
Pulses of ultrasonic energy can stimulate an classical acupoints, eliciting a response similar to that produced by standard needling. **Ultrasonic stimulation of the acupoints offers many advantages over conventional methods (no pain or sensation or temperature rise during treatment)** and provides an extremely useful tool for the scientific study and quantitative evaluation of acupuncture.

In addition, quantitative ultrasound methods have shown that **acupoints represent regions of enhanced ultrasonic attenuation, which change in size, shape, and location over short periods of time.** Our study also suggests that an ultrasonic acupuncture system could be developed that would locate the acupoint (using quantitative ultrasound methods), and then stimulate the acupoint (using pulses of higher ultrasonic energy). **"Ultrasonic Acupuncture" would seem to combine the best of Oriental medicine with the best of Western technology for the improvement of health care.**

Anesth Analg. 2007 Feb;104(2):301-7

Acupuncture Enhances Generation of Nitric Oxide and Increases Local Circulation

Tsuchiya M, et al. Departments of Biochemistry, Osaka City University Medical School, Abeno-Ku, Japan.



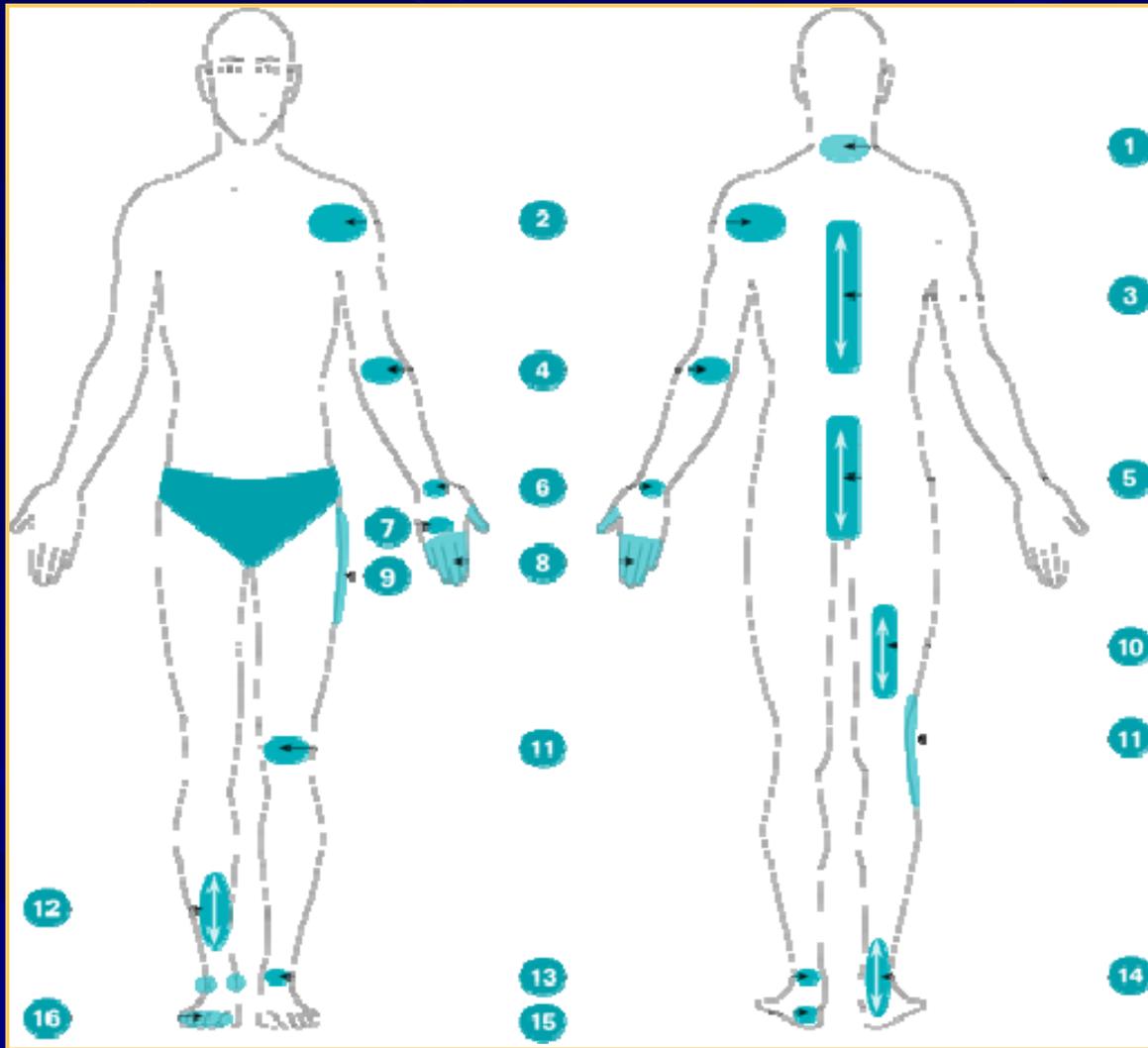
Abstract

Although it is widely used, the mechanisms and effects of acupuncture on pain are not completely understood. Recently, increased nitric oxide (NO) synthase activity has been found in meridians and acupoints. Because NO is a key regulator of local circulation, and because change in circulation can affect the development and persistence of pain, we propose that acupuncture might regulate NO levels. We studied the effects of acupuncture on local NO levels and circulation in a randomized, double-blind, crossover study with 20 volunteers, each of whom underwent one session each of real and noninvasive sham acupuncture in a single hand and forearm with a 1-wk interval between treatments.

NO concentration in the plasma from the acupunctured arm was significantly increased by 2.8 ± 1.5 micromol/L at 5 min and 2.5 ± 1.4 micromol/L at 60 min after acupuncture. Blood flow in palmar subcutaneous tissue of the acupunctured arm also increased, and this correlated with the NO increase. These changes were not observed in noninvasive sham-acupunctured hands and forearms. In conclusion, acupuncture increases the NO level in treated regions and thereby increases local circulation. These regulatory effects might contribute to pain relief provided by acupuncture.

Qi or Chi... According to the principles of traditional Chinese medicine

illness is caused when *qi* does not flow properly throughout the body. Acupuncturists determine whether *qi* is weak, stagnant or otherwise out of balance, which indicates the points to be stimulated.



Electroacupuncture is useful for conditions in which there is an accumulation of *qi*, such as in chronic pain conditions, or in cases where the *qi* is difficult to stimulate.

One advantage of electroacupuncture is that a practitioner does not have to be as precise with the insertion of needles. This is because the current delivered through the needle stimulates a larger area than the needle itself. The advantage of this procedure is that it can be used by people who have a fear of needles or a condition that prohibits them from being needed.

Nitric Oxide Formation by Ultrasound in Aqueous Solutions

Vladimír Mišík and Peter Riesz

Radiation Biology Branch, National Cancer Institute, National Institutes of Health, Bethesda, Maryland

In this study we demonstrate formation of nitric oxide in aqueous nitrogen-containing solutions exposed to 50 kHz cavitation-producing ultrasound (standard bath sonicator) using electron paramagnetic resonance detection of $\bullet\text{NO}$ by trapping with the sodium *N*-methyl-*D*-glucamine dithiocarbamate iron(II) complex ((MGD) 2Fe^{2+}) or by measuring the conversion of the nitronyl nitroxide, 2-(4-carboxyphenyl)-4,4,5,5-tetramethylimidazoline-3-oxide-1-oxyl (carboxy-PTIO), to the imino nitroxide, 2-(4-carboxyphenyl)-4,4,5,5-tetramethylimidazoline-1-oxyl. The (MGD) 2Fe^{2+} complex which was used in most experiments was suitable for $\bullet\text{NO}$ detection over a wide pH range (pH 3–7.8; the working range for carboxy-PTIO was pH 6–8.5), and the measured rate constant of (MGD) 2Fe^{2+} reaction with $\bullet\text{NO}$ was 2.3 times higher than for carboxy-PTIO. In air-saturated water the rate of $\bullet\text{NO}$ production by ultrasound was 0.5 $\mu\text{M}/\text{min}$.

The presence of dissolved oxygen was not essential for production of $\bullet\text{NO}$; the highest yields of $\bullet\text{NO}$ (1.2 μM $\bullet\text{NO}/\text{min}$) were found under an atmosphere of 40% N_2 and 60% argon. The formation of $\bullet\text{NO}$ by ultrasound in aqueous solutions can be understood in terms of combustion chemistry-type reactions occurring inside the “hot” collapsing cavitation bubbles. We also show that other N-containing molecules can serve as a source of nitrogen for $\bullet\text{NO}$ production. **The possibility of ultrasound-mediated $\bullet\text{NO}$ formation to alleviate hypoxia of tumors should be explored.**

Ultrason Sonochem. 2006 Jul;13(5):397-400. Epub 2005 Sep 15.

Ultrasound liberates nitric oxide (NO) from the caged NO compound N,N'-bis(carboxymethyl)-N,N'-dinitroso-p-phenylenediamine sodium salt.

Feril LB Jr, Kondo T.

Department of Radiological Sciences, Faculty of Medicine, Toyama Medical and Pharmaceutical University, 2630 Sugitani, Toyama 930-0194, Japan.

Abstract

To determine whether nitric oxide (NO) can be released from a cage compound N,N'-bis(carboxymethyl)-N,N'-dinitroso-p-phenylenediamine sodium salt (BNN 5 Na), we sonicated different concentrations of BNN 5 Na solutions containing an NO spin trap, (MGD)₂Fe²⁺, and then measured (MGD)₂Fe²⁺-NO signal using electron paramagnetic resonance (EPR).

We also investigated the role of cavitation by saturating the solutions with Ar, He or Xe gases before sonication. The result showed that ultrasound can liberate NO from caged NO compound at rates highest with Xe and lowest with He. These results suggest that high-temperature due to cavitations induced by ultrasound are capable of releasing NO from caged NO compounds.

This finding also opens up to a new possibility for the use of ultrasound in controlled release of active compounds (e.g. drugs, supplements) from caged forms for therapeutic purposes.

PMID: 16168699

Natural Healing with Intranasal Light Therapy

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



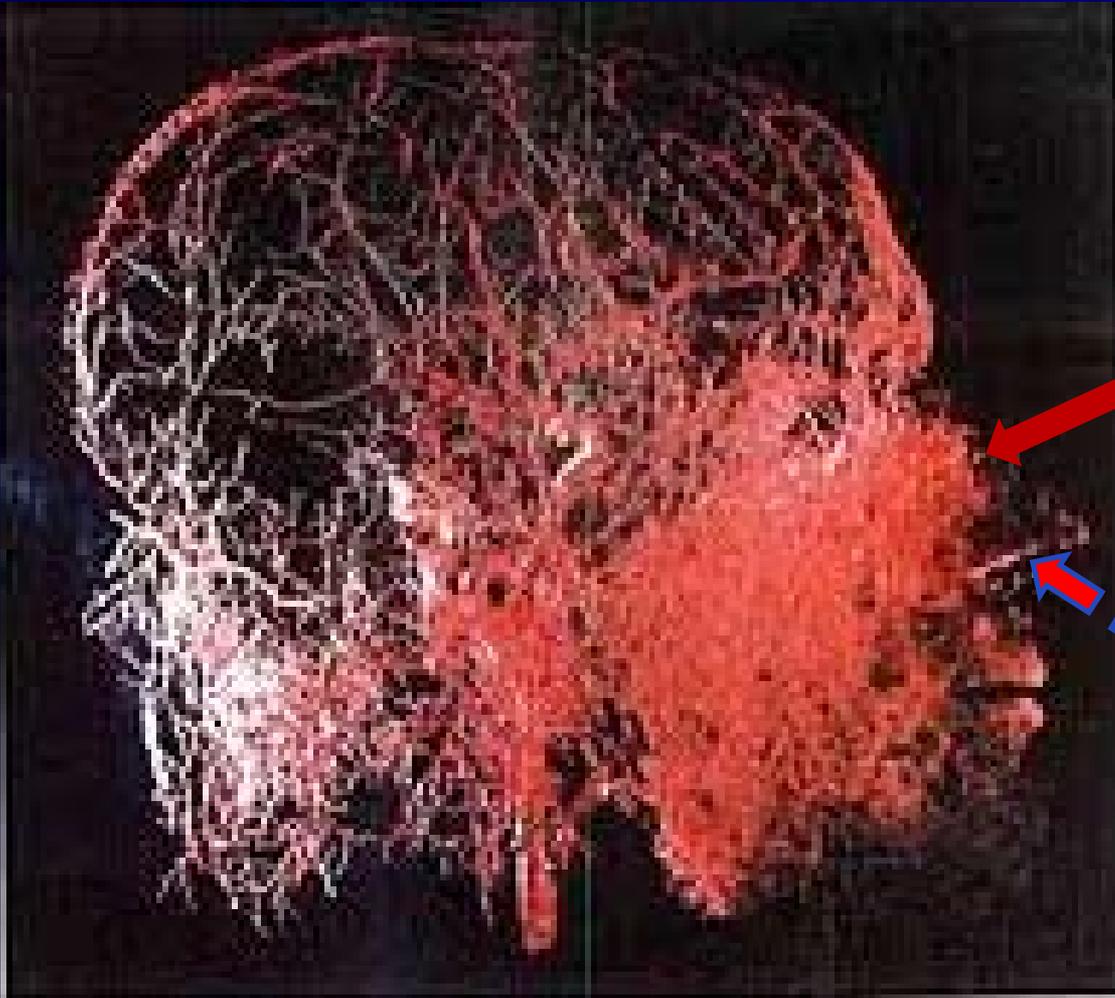
Intranasal light therapy stimulates restoration of body balance (homeostasis).

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



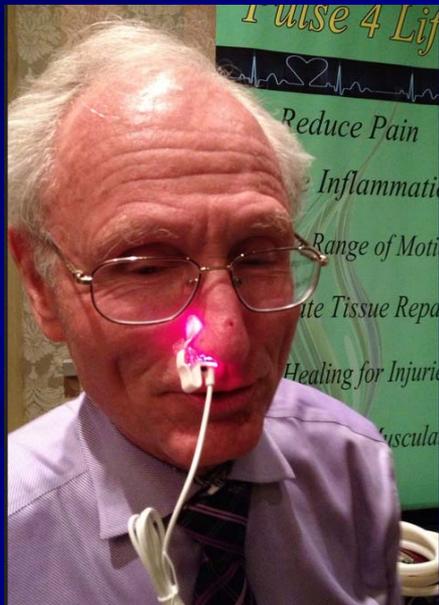
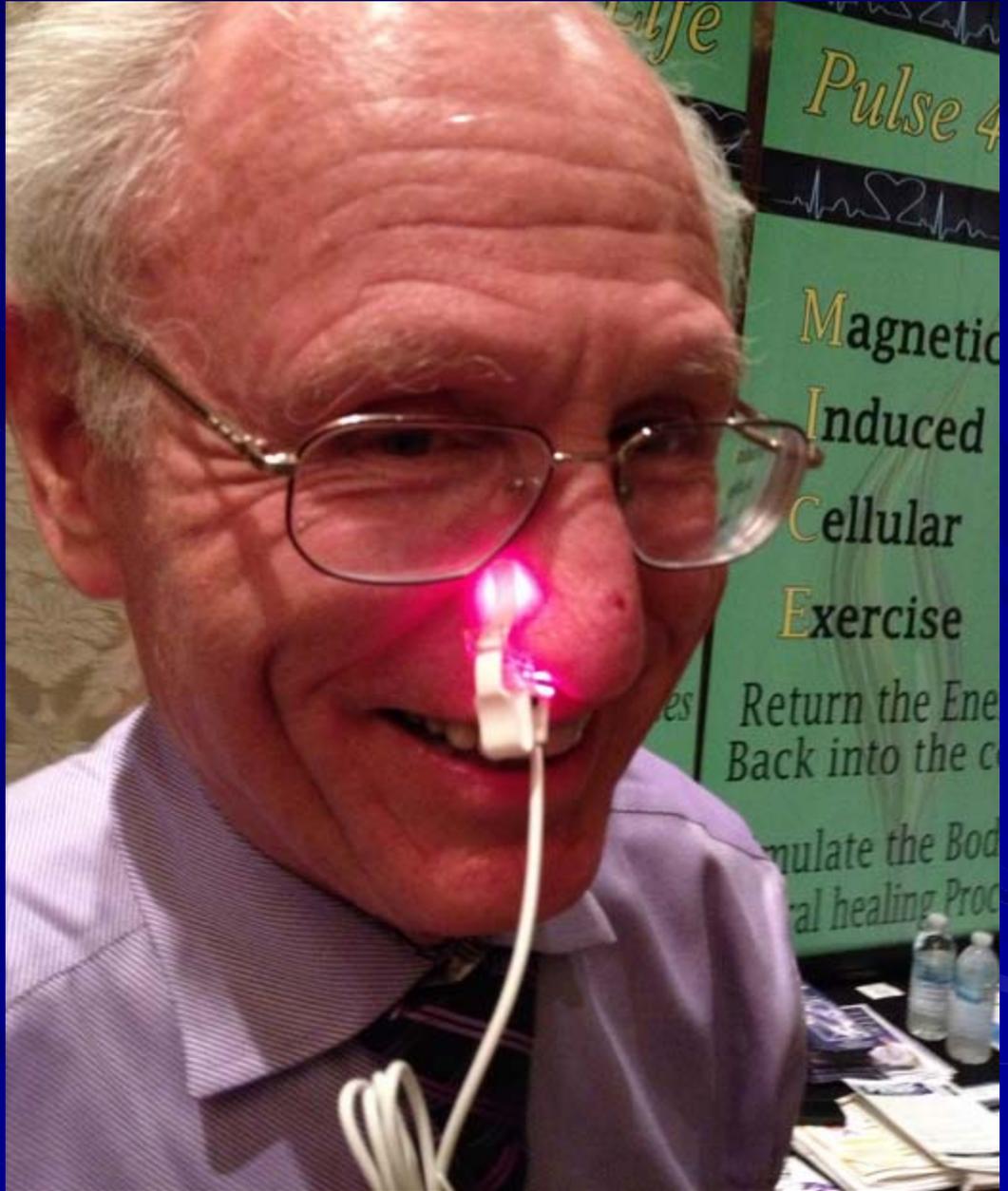
The facial area also responds directly to the light, often resulting in the immediate treatment of sinusitis, congestion, headache and, facial and pain in the neck area.

Blood vessels of the Human Head



The most concentrated area

Light source



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

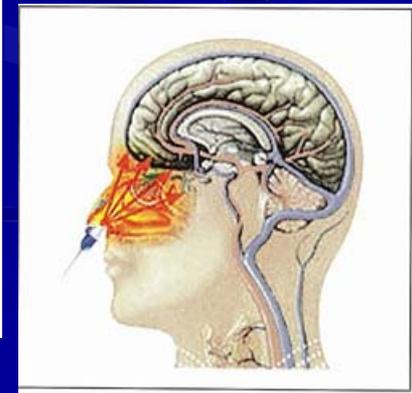
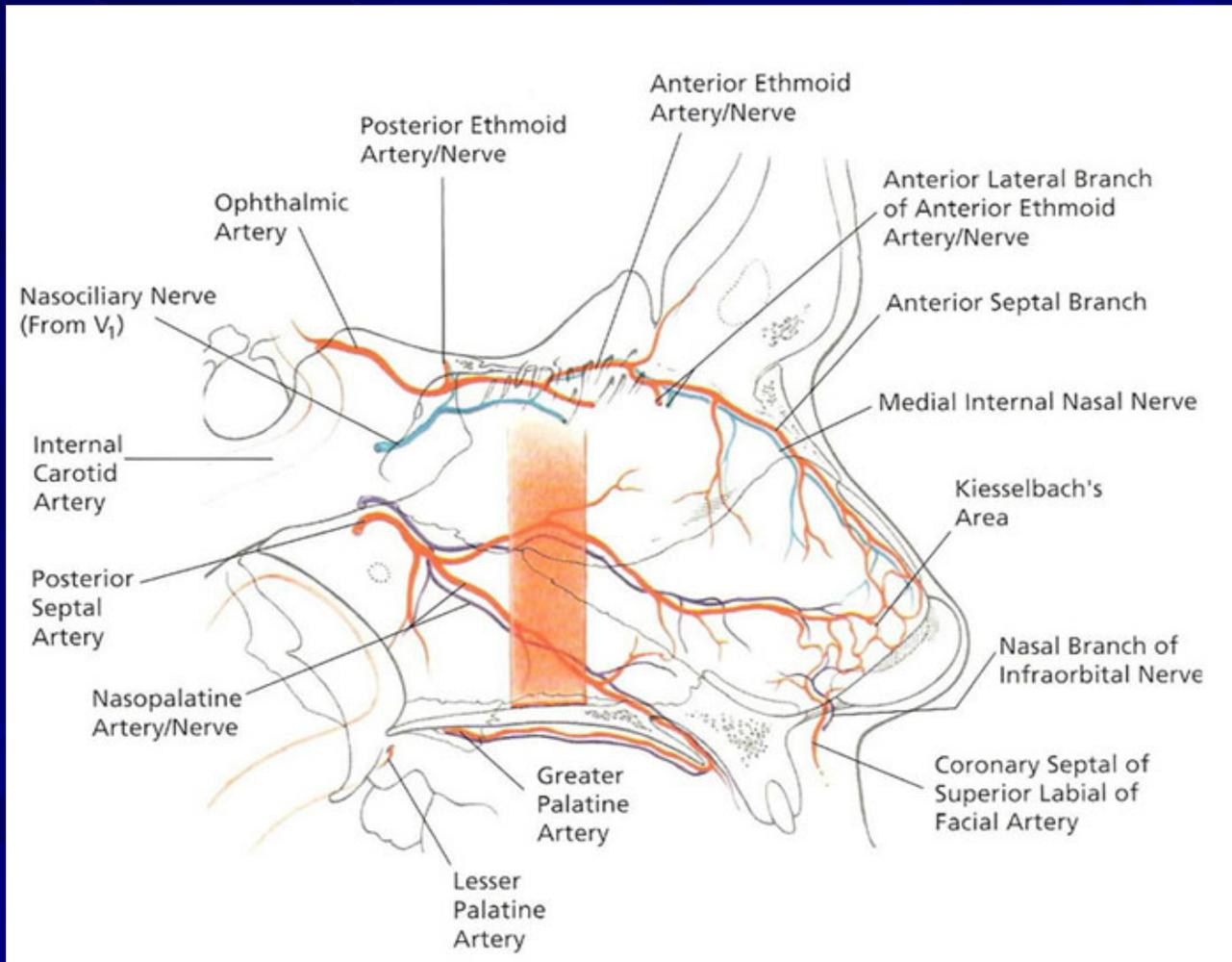
1. Increased ATP production by the mitochondria and increased oxygen consumption on the cellular level, which may result in muscle relaxation
2. Increased serotonin and increased endorphins
3. Increased anti-inflammatory effects through reduced prostaglandin synthesis
4. Improved blood circulation to 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

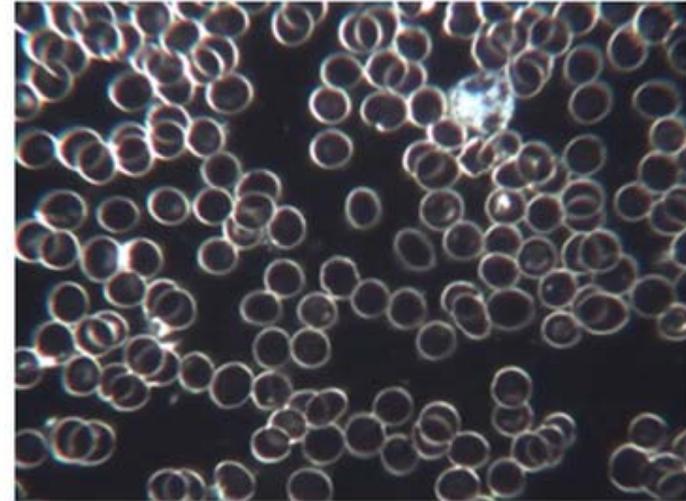
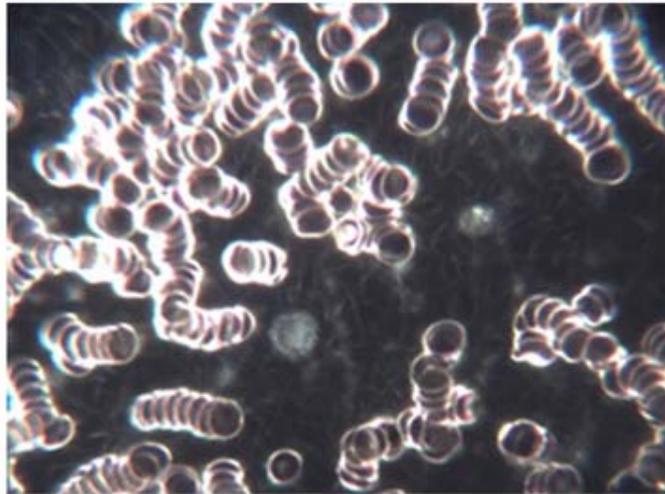
Adjuvant therapy

Health care

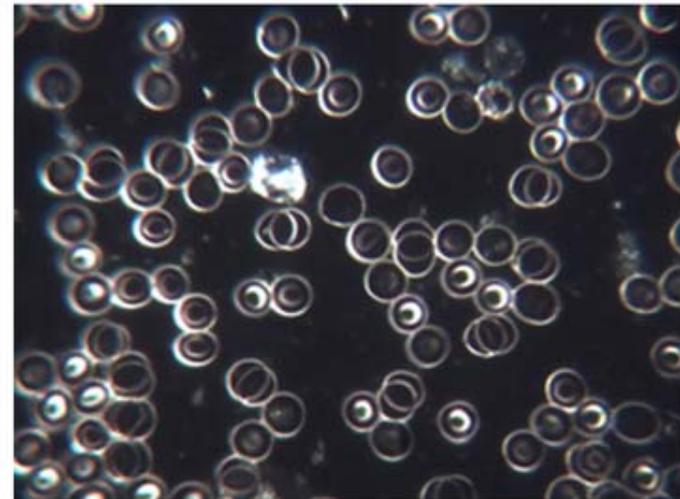
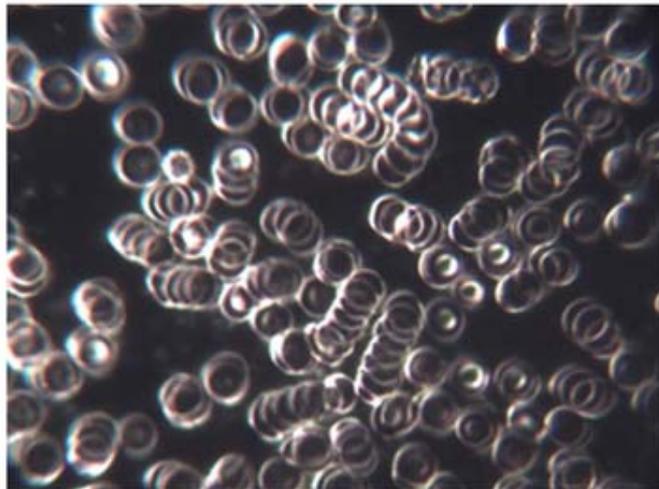
- **Hypertension** <<Learn more
- **Hyperlipoidemia** <<Learn more
- **Hyperviscosity** <<Learn more
- **Stroke** <<Learn more
- **Sugar diabetes** << Learn more
- **Insomnia** << Learn more
- Blood health care
- Improve natural immunity
- Anti-ageing
- protect the ischemic anemia

ILIT Biomechanism: blood mediation





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

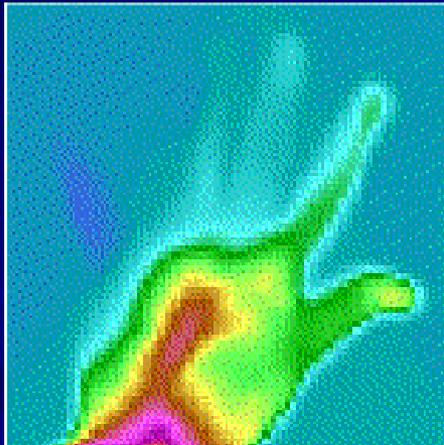


Healing Distributed through the Circulatory System

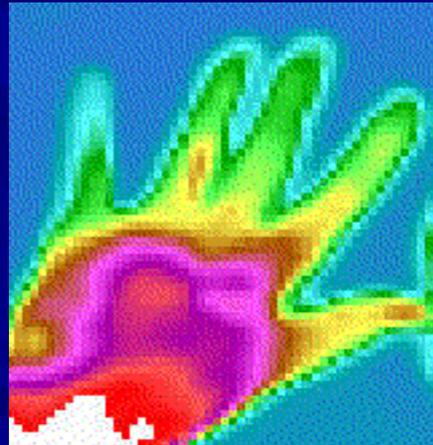
The combined roles of singlet oxygen, ROS, Redox Signalling and the activity of SOD best explains the mechanism behind the healing success of Intranasal Light Therapy. The key to the efficacy of the intranasal pathway is that it is essentially an in vivo method without the invasiveness of the older intravenous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

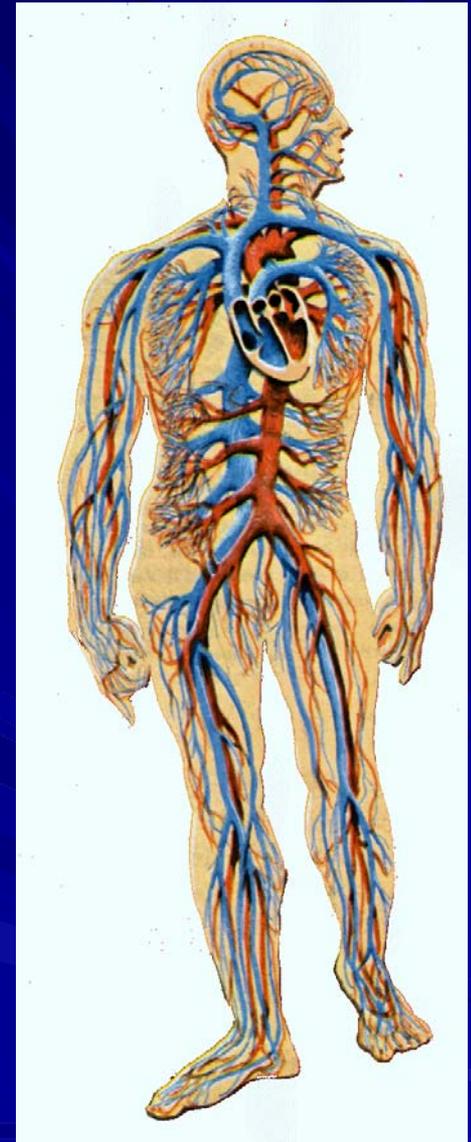
Peripheral blood circulation



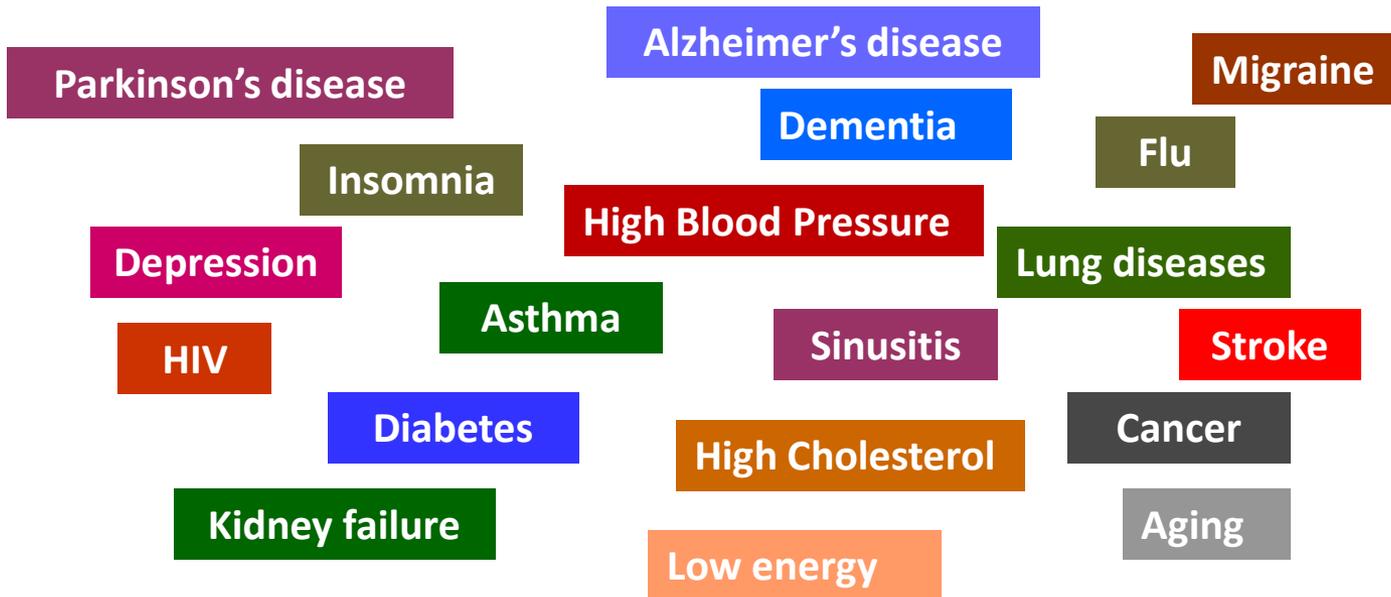
Before



After



Clinical Evidence for...



and more....

Summary Differences Between Three Models



	633 Red / Qi-Light	RadiantLife LT / 655 Prime	810 Infrared
Price	\$299	\$399	\$499
Light source	LED	Low level laser	LED
Wavelength	633 nm	655 nm	810 nm
Color	Red	Deep red	Invisible
Pulse mode	Continuous	Continuous	10 Hz
Battery life	2 months	3 months	2.5 months
Safety issues	None	Very small	None
Research studies	Few	Many	Few

J Altern Complement Med. 2007 Nov;13(9):955-67.

Can electrons act as antioxidants? A review and commentary.

Oschman JL. PMID: 18047442 [PubMed - indexed for MEDLINE]



It is well established, though not widely known, that the surface of the earth has a limitless and continuously renewed supply of free or mobile electrons as a consequence of a global atmospheric electron circuit.

Wearing shoes with insulating soles and/or sleeping in beds that are isolated from the electrical ground plane of the earth have disconnected most people from the earth's electrical rhythms and free electrons.

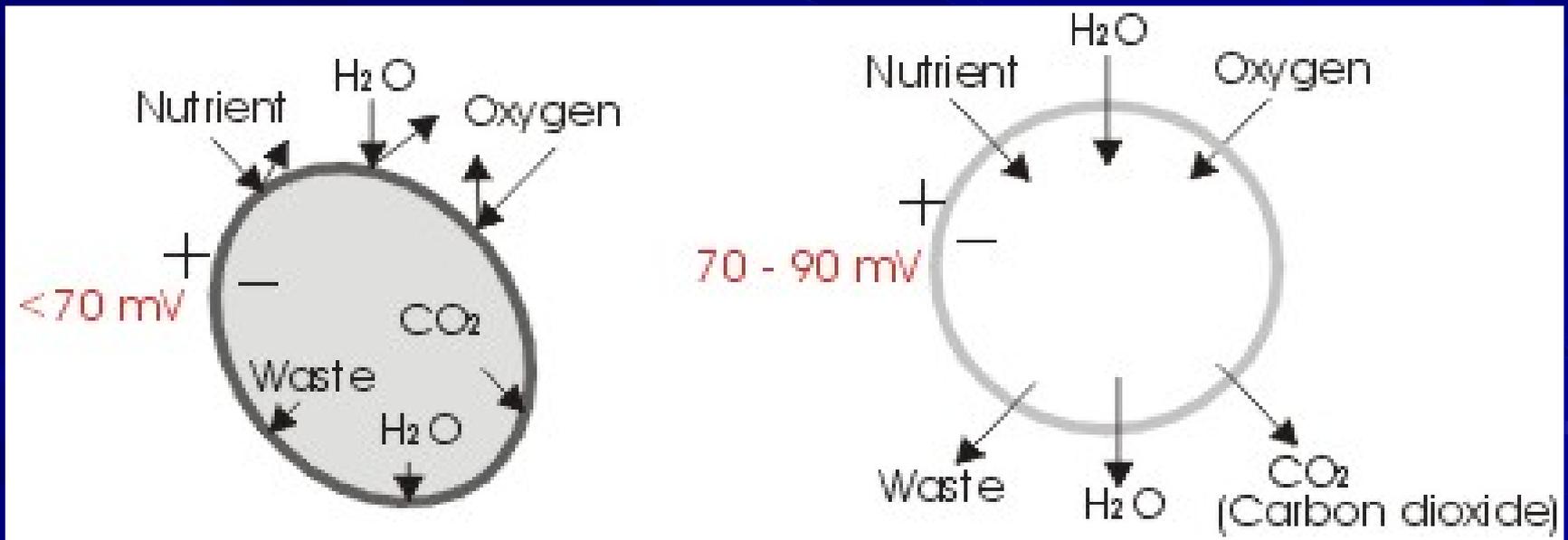
Studies have demonstrated that connecting the human body to the earth during sleep (earthing) normalizes the daily cortisol rhythm and improves sleep. It is also suggested that free electrons from the earth neutralize the positively charged free radicals that are the hallmark of chronic inflammation. The research summarized here and in subsequent reports provides a basis for a number of earthing technologies that restore and maintain natural electrical contact between the human body and the earth throughout the day and night in situations where going barefoot on the earth is impractical.

It is proposed that free or mobile electrons from the earth can resolve chronic inflammation and pain by serving as natural antioxidants.

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

It is observed that drug uptake after an exponentially decaying electro poration pulse of the initial field strength $E_0 = 1.4$ kV/cm and pulse time constants in the time range 0.5–3 ms, is faster than during PEMF-treatment, i.e., application of an alternating current of 16 kHz, voltage $U < 100$ V, $I = 55$ mA, and exposure time 20 min.

However, at the low a.c. voltage of this treatment, more apoptotic and necrotic cells are produced as compared to the electroporation treatment with one exponentially decaying voltage pulse.

Thus, **additional photodynamic action appears to be more effective than solely drugs and electroporation**, as typically applied in clinical electro chemotherapy, and somewhat more effective than the noninvasive pulsed electromagnetic fields (PEMFs), for cancer cells in general and animals bearing tumors in particular.

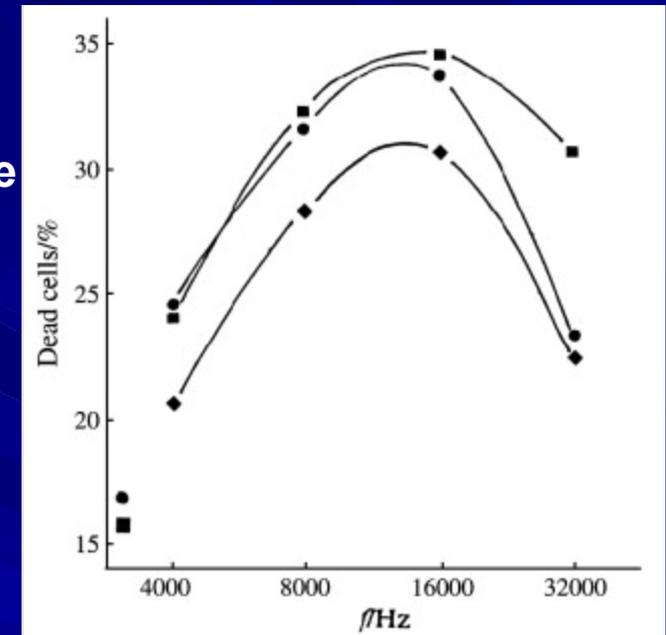
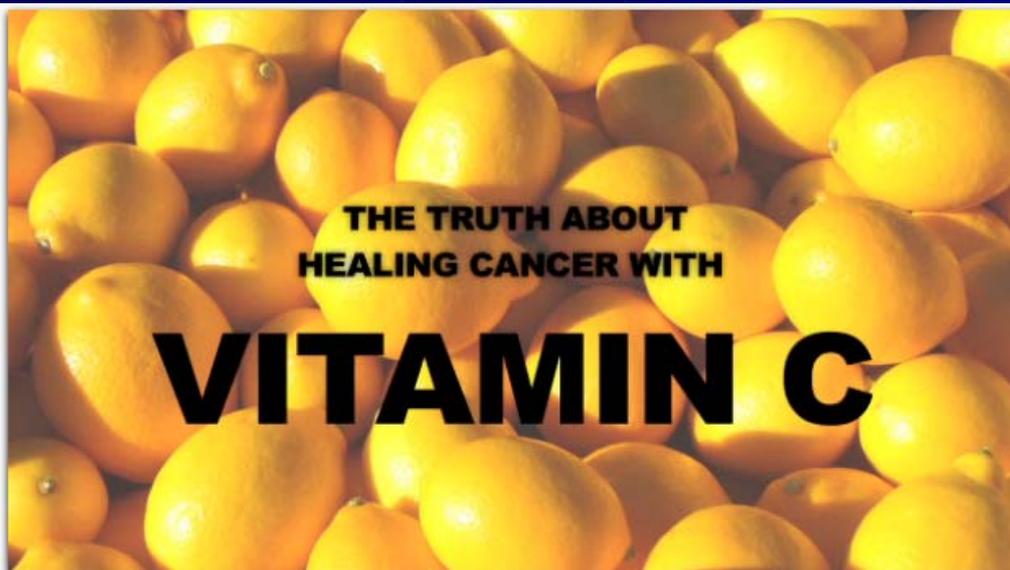


Fig. 1. Frequency dependence. % (death) as a function a.c. current frequency f for the number $N(\text{dead})$ of dead U-937 cells, determined by TP colouring: the percentage dead is defined by $\% \text{ dead} = 10^2 N(\text{dead})/N(\text{total})$. See Eq. (1) of the main text. Lethal effect by penetration of the drugs alone into the cells after 20 min, below left: (■), for actinomycin-C (1×10^{-5} M); (●), for daunomycin (1×10^{-5} M), both without a.c. field pulses. (◼) lethal synergism after 20 min exposure to a.c. field pulses in the presence of drugs: (◼) plus actinomycin-C (1×10^{-5} M) (◻) plus daunomycin (1×10^{-5} M).



CANCER is curable NOW

However it is **NOT** cured with Medicine or Drugs, but with **KNOWLEDGE**



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.

DIET WISE ACADEMY

With Dr. Keith Scott-Mumby



Study your body's food needs with the world's leading expert

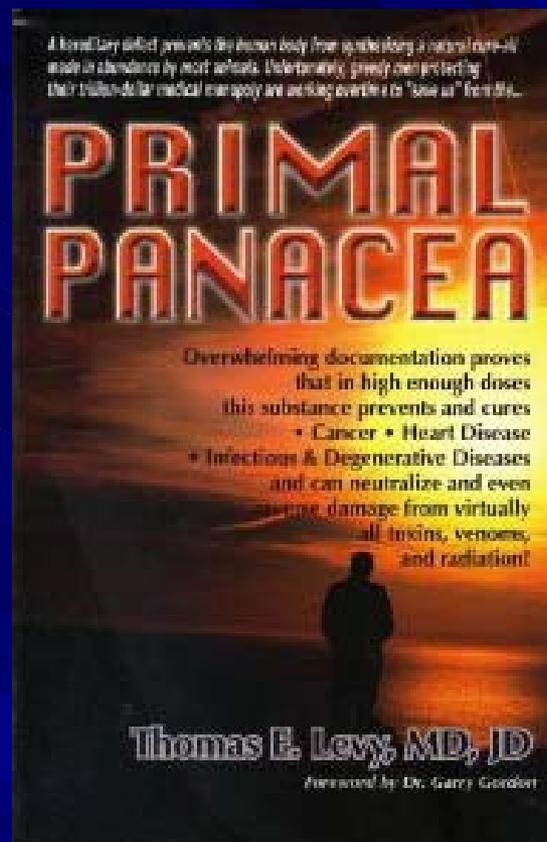
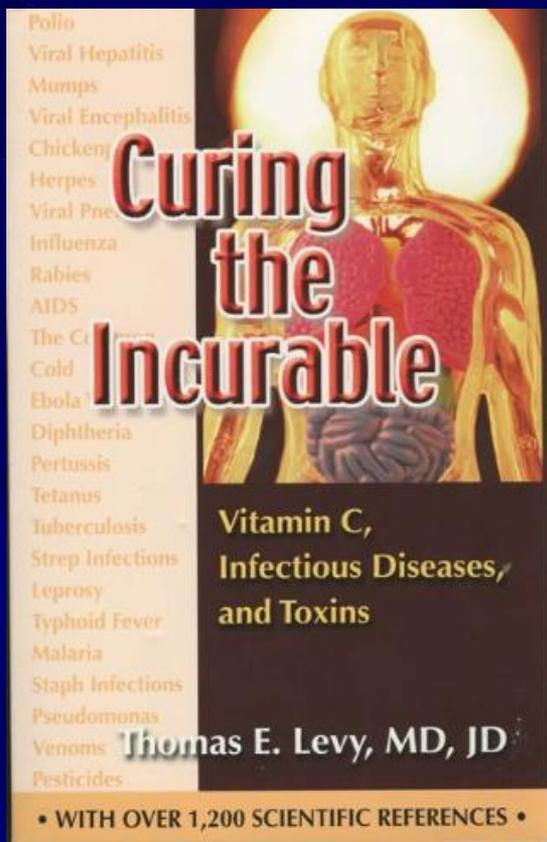


FREE 1 DAY CANCER WORKSHOP

HEALING CANCER With Common Sence

Watch Online **FREE**





VITAMIN C

Vitamin C, given at sufficiently high doses, by itself, can cure life-threatening infections and neutralize many otherwise fatal toxin exposures, according to author Thomas E. Levy, MD, JD in his extensively referenced book, *Vitamin C, Infectious Diseases, and Toxins: Curing the Incurable*, and his newest book "Primal Panacea".

Thomas Levy's books are unmatched in the medical literature. According to Dr. E. Cheraskin, more than 80,000 scientific papers and reports have been written about vitamin C since its chemical nature was first discovered early in the 20th century. The Vitamin C Foundation credits Levy with "doing an almost impossible feat of reading, analyzing and clearly explaining the meaning of the massive science behind vitamin C."

http://findarticles.com/p/articles/mi_m0ISW/is_2003_May/ai_100767885/

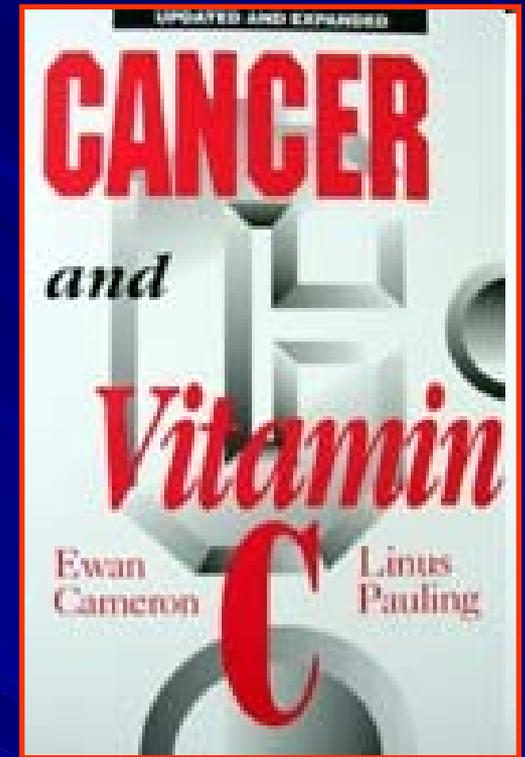
Vitamin C and Cancer

Linus Pauling & Ewan Cameron

A large number of studies have shown that increased consumption of fresh fruits and vegetables is associated with a reduced risk for most types of cancer.

Most have shown that higher intakes of vitamin C are associated with decreased incidence of cancers of the mouth, throat and vocal chords, esophagus, stomach, colon-rectum, and lung.

In general, prospective studies in which the lowest intake group consumed no more than 86 mg of vitamin C daily, have not found differences in cancer risk, while studies finding *significant cancer risk reductions found in people consuming at least 80 to 110 mg of vitamin C daily.*



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 β peptides ($A\beta$), 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 be 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.

Ascorbic acid enhances the expression of collagen and SVCT2 in cultured human skin fibroblasts.

Kishimoto Y, Saito N, Kurita K, Shimokado K, Maruyama N, Ishigami A.
Molecular Regulation of Aging, Tokyo Metropolitan Institute of Gerontology,
Tokyo 173-0015, Japan.



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

Abstract

Ascorbic acid (AA) is essential for collagen biosynthesis as a cofactor for prolyl and lysyl hydroxylase and as a stimulus for collagen gene expression. Many studies have evaluated the relationship between AA and collagen expression in short- and long-term effects on cells after a single administration of AA into the culture medium. However, no such study has monitored in detail the stability of AA in medium or the alterations of intracellular AA levels during a protracted interval. Therefore, we examined here intracellular AA levels and stability throughout its exposure to human skin fibroblasts *in vitro*.

Moreover, we determined the effects on type 1 and type 4 collagen and sodium-dependent vitamin C transporter (SVCT) gene expression when medium containing 100 μ M AA was replaced every 24h for 5 days to avoid depletion of AA. Throughout this long-term culture, intracellular AA levels remained constant; the expression of type 1 and type 4 collagens and SVCT2 mRNA was enhanced, and type 1 procollagen synthesis increased.

Thus, these results indicate that **human skin fibroblasts exposed to AA over time had rising levels of type 1/type 4 collagens and SVCT2 mRNA expression and type 1 procollagen synthesis.**

CMAJ. 2006 March 28; 174(7): 937–942.

Intravenously administered vitamin C as cancer therapy: three cases

Sebastian J. Padayatty, Hugh D. Riordan, Stephen M. Hewitt, Arie Katz, L. John Hoffer,
and Mark Levine



Early clinical studies showed that high-dose vitamin C, given by intravenous and oral routes, may improve symptoms and prolong life in patients with terminal cancer. Double-blind placebo-controlled studies of oral vitamin C therapy showed no benefit. Recent evidence shows that oral administration of the maximum tolerated dose of vitamin C (18 g/d) produces peak plasma concentrations of only 220 $\mu\text{mol/L}$, whereas intravenous administration of the same dose produces plasma concentrations about 25-fold higher. Larger doses (50–100 g) given intravenously may result in plasma concentrations of about 14 000 $\mu\text{mol/L}$. At concentrations above 1000 $\mu\text{mol/L}$, vitamin C is toxic to some cancer cells but not to normal cells in vitro. We found 3 well-documented cases of advanced cancers, confirmed by histopathologic review, where patients had unexpectedly long survival times after receiving high-dose intravenous vitamin C therapy. We examined clinical details of each case in accordance with National Cancer Institute (NCI) Best Case Series guidelines. Tumour pathology was verified by pathologists at the NCI who were unaware of diagnosis or treatment. In light of recent clinical pharmacokinetic findings and in vitro evidence of anti-tumour mechanisms, these case reports indicate that the role of high-dose intravenous vitamin C therapy in cancer treatment should be reassessed.

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1405876/?report=classic>

November 1996 chest radiography revealed multiple cannonball lesions ([Fig. 1](#)).

The patient declined conventional cancer treatment and instead chose to receive high-dose vitamin C administered intravenously at a dosage of 65 g twice per week starting in October 1996 and continuing for 10 months. She also used other alternative therapies: thymus protein extract, N-acetylcysteine, niacinamide and whole thyroid extract . In June 1997 chest radiography results were normal except for one remaining abnormality in the left lung field, possibly a pulmonary scar ([Fig. 2](#)).

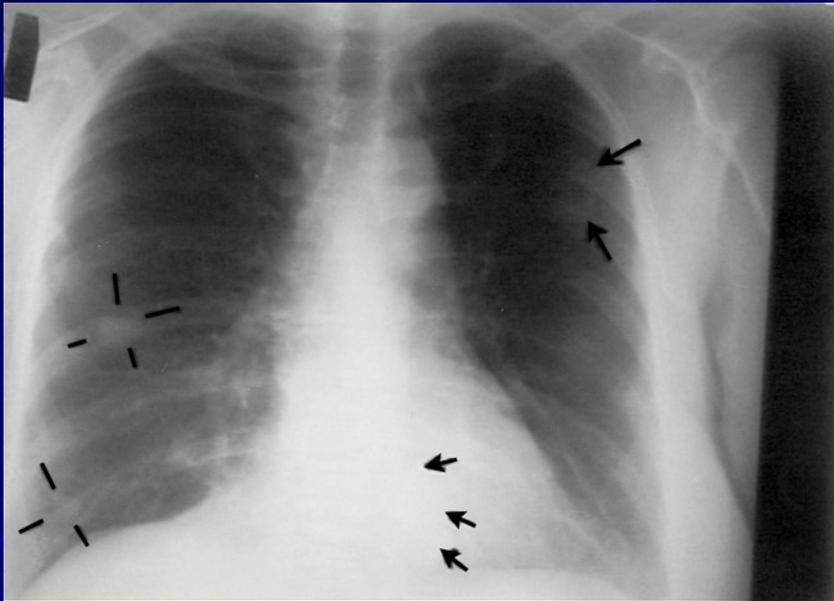


Fig. 1: Chest radiography, November 1996, about 1 month after intravenous vitamin C therapy was started. Cannonball lesions are evident in both lung fields, as indicated by the arrows and lines.



Fig. 2: Chest radiography, June 1997, showing regression of the lesions; the arrow indicates one residual abnormality.

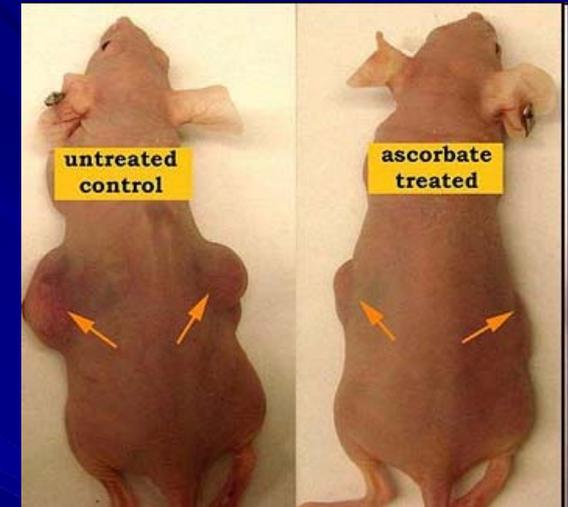
Vitamin C Injections Slow Tumor Growth in Mice

High-dose injections of vitamin C, also known as ascorbate or ascorbic acid, reduced tumor weight and growth rate by about 50 percent in mouse models of brain, ovarian, and pancreatic cancers, researchers from the National Institutes of Health (NIH) report in the August 5, 2008, issue of the *Proceedings of the National Academy of Sciences*.

In their laboratory experiments on 43 cancer and 5 normal cell lines, the researchers discovered that high concentrations of ascorbate had anticancer effects in 75 percent of cancer cell lines tested, while sparing normal cells.

In their paper, the researchers also showed that these high ascorbate concentrations could be achieved in people.

The team then tested ascorbate injections in immune-deficient mice with rapidly spreading ovarian, pancreatic, and glioblastoma (brain) tumors. The ascorbate injections reduced tumor growth and weight by 41 to 53 percent. In 30 percent of glioblastoma controls, the cancer had spread to other organs, but the ascorbate-treated animals had no signs of disseminated cancer. "These pre-clinical data provide the first firm basis for advancing pharmacologic ascorbate in cancer treatment in humans," the researchers conclude.



<http://www.nih.gov/news/health/aug2008/niddk-04.htm>

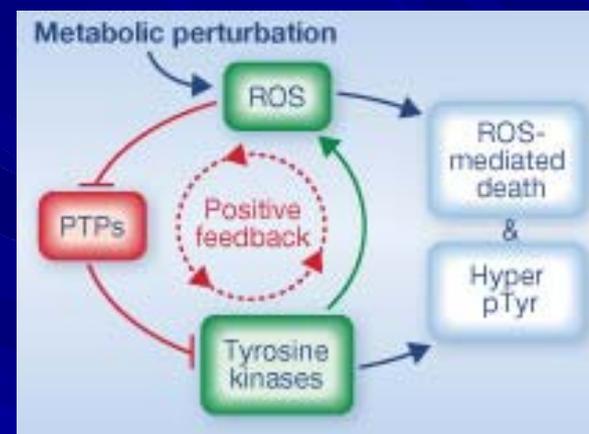
Image: Mark Levine

Glucose deprivation activates a metabolic and signaling amplification loop leading to cell death

Graham NA, Tahmasian M, Kohli B, et al.

In summary, cellular redox homeostasis is maintained by the balance between ROS generation by required metabolic functions and ROS elimination. Likewise, signaling homeostasis is controlled by balancing kinase and phosphatase activity.

Here, we demonstrate that the **cellular microenvironment (i.e., nutrient availability)** can alter the cellular redox balance, provoking a signaling-based positive feedback loop that amplifies ROS levels above a toxicity threshold resulting in cell death.

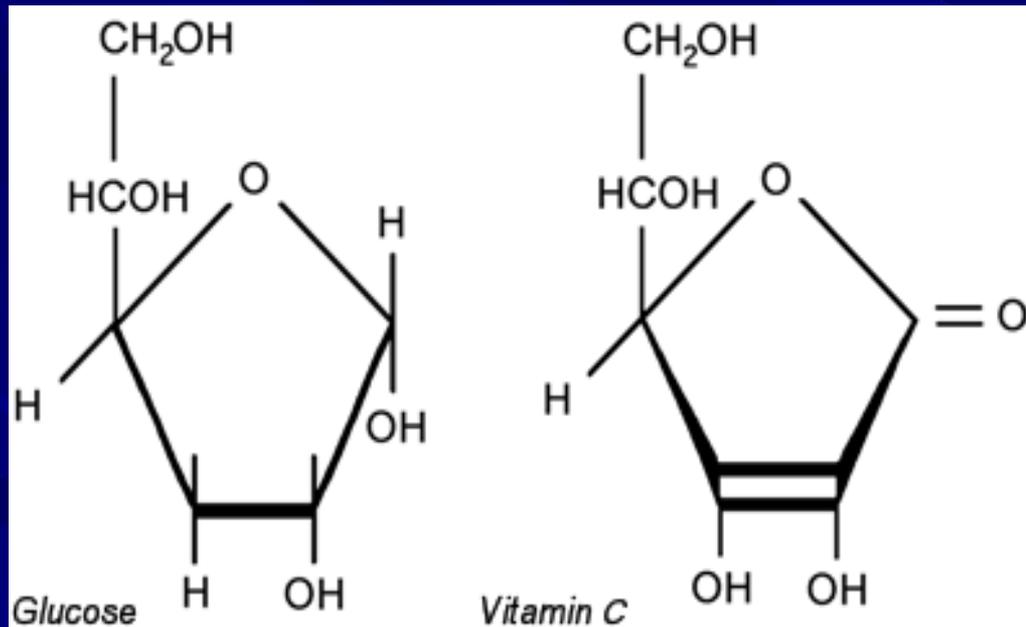


This positive feedback loop demonstrates the complex, systems-level integration of homeostatic control mechanisms for metabolism (e.g., redox balance) and TK signaling (e.g., PTPs).

Furthermore, this systems integration offers a scaffold for synergistic combinations of therapeutics targeting signaling, metabolism, and redox homeostasis.

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 (H₂O₂) 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.

High Dose Vitamin C – Overview

- Vitamin C is a nutrient found in food and dietary supplements. It is an antioxidant and also plays a key role in making collagen.
- High-dose vitamin C has been studied as a treatment for cancer patients since the 1970s
- High-dose vitamin C may be given by intravenous (IV) infusion or taken by mouth.
- Laboratory studies have shown that high doses of vitamin C may slow the growth and spread of prostate, pancreatic, liver, colon, and other types of cancer cells.
- Animal studies have shown that high-dose vitamin C treatment blocks tumor growth in certain models of pancreatic, liver, prostate, and ovarian cancers, sarcoma, and malignant mesothelioma.
- Some human studies of IV vitamin C or vitamin C taken by mouth in patients with cancer have shown improved quality of life, as well as improvements in physical, mental, and emotional functions, symptoms of fatigue, nausea and vomiting, pain, and appetite loss.
- Intravenous high-dose ascorbic acid has caused very few side effects in clinical trials.
- The U.S. Food and Drug Administration (FDA) has not approved the use of high-dose vitamin C as a treatment for cancer or any other medical condition.

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.

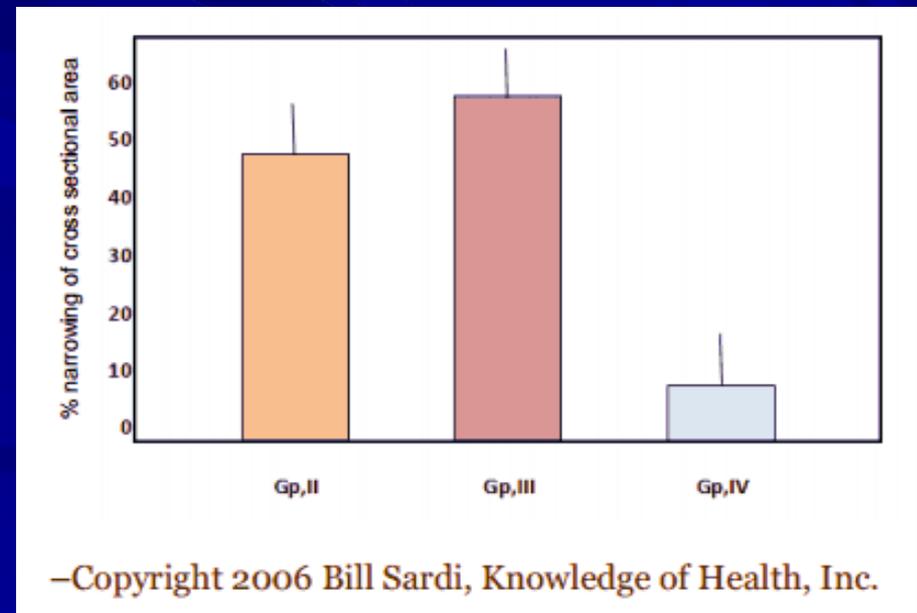
The low-dose group of rabbits were given the human equivalent of about 350 milligrams of vitamin C, and the high-dose group the human equivalent of 11,000 milligrams of vitamin C per day. The chart below shows the percentage of arterial narrowing by cholesterol.

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-availability.

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.

The vitamin C:K3 system - Enhancers and inhibitors of the anticancer effect.

Lamson DW, Gu YH, Plaza SM, Brignall MS, Brinton CA, Sadlon AE.

Abstract

The oxidizing anticancer system of vitamin C and vitamin K3 (VC:VK3, producing hydrogen peroxide via superoxide) was combined individually with melatonin, curcumin, quercetin, or cholecalciferol (VD3) to determine interactions. Substrates were LNCaP and PC-3 prostate cancer cell lines. Three of the tested antioxidants displayed differences in cell line cytotoxicity. Melatonin combined with VC:VK3 quenched the oxidizing effect, while VC:VK3 applied 24 hours after melatonin showed no quenching. With increasing curcumin concentrations, an apparent combined effect of VC:VK3 and curcumin occurred in LNCaP cells, but not PC-3 cells. Quercetin alone was cytotoxic on both cell lines, but demonstrated an additional 50-percent cytotoxicity on PC-3 cells when combined with VC:VK3. VD3 was effective against both cell lines, with more effect on PC-3. This effect was negated on LNCaP cells with the addition of VC:VK3. In conclusion, a natural antioxidant can enhance or decrease the cytotoxicity of an oxidizing anticancer system *in vitro*, but generalizations about antioxidants cannot be made.

The VC:VK3 combination generates H₂O₂ efficiently by redox cycling, such that a high level of VC by the intravenous route may not be necessary for cancer cell death. Since the VC:VK3 combination increases the cytotoxicity by six- to seven-fold over individual vitamin use, the oral route might suffice. Research on this concept proceeded through the usual route from *in vitro*, to *in vivo*, to human trial.

The VC:VK3 system has performed positively *in vitro* for prostate cancer, breast cancer, ovarian cancer, bladder cancer, hepatocarcinoma, and some leukemias.

Vitamin C is a powerful antibiotic, effective against both bacteria and viruses.

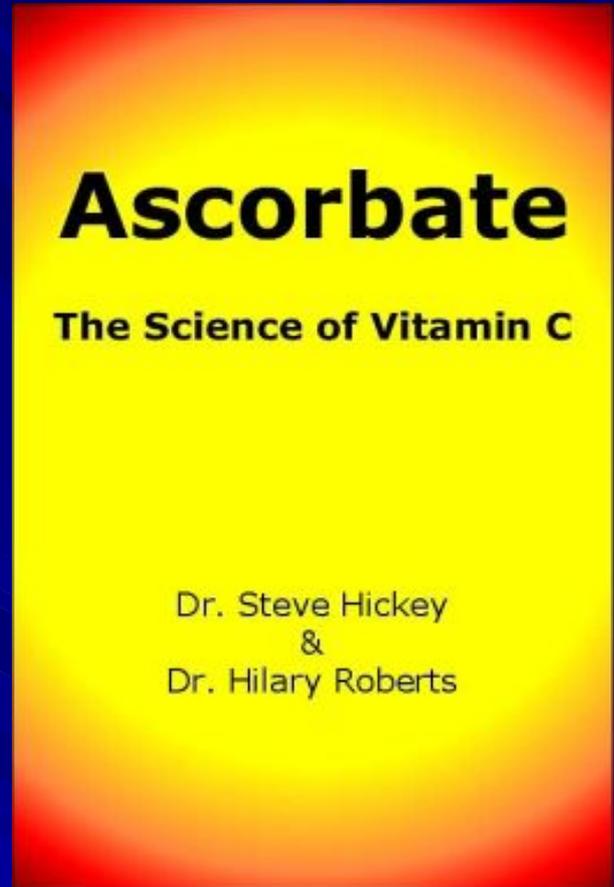
Infectious Disease is a big killer in both the developing world and industrialised societies, despite improvements in diet, hygiene, and the use of anti-biotics.

However, emerging viruses, such as HIV, SARS and Ebola, along with multiple drug resistant bacteria (e.g. MDR-TB, MRSA) are forcing us to think again.

Harmful infections result in damage to body tissues, caused by modified molecules called free radicals. Some physicians claim that large doses of vitamin C can act as the ultimate biological antioxidant, neutralizing the free radicals and quenching the infection.

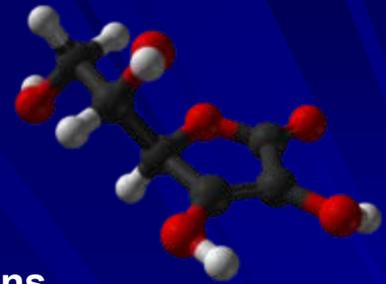
The action of free radicals is similar in a range of diseases, from the common cold through to the haemorrhagic fever Ebola, so they might all be suitable for treatment with vitamin C.

The only difference is the dose required.



RDA = “Ridiculous Dietary Allowance”

Steve Hickey, Hilary Roberts, Ph.D.



Many people consuming RDA levels of vitamins are likely to suffer from deficiency disease and premature death. Current official recommendations for nutrient intakes are inappropriate. **The recommended dietary intake for vitamin C owes more to politics and prejudice than to science, and the research behind the RDA values for vitamin C is *biased and insubstantial*.**

Low or adequate RDA dosages. Although ascorbate in small quantities prevents acute scurvy, the optimal intake is not established.

Age	Male	Female	Pregnant	Lactating
0-6 months	40 mg*	40 mg*		
7-12 months	50 mg*	50 mg*		
1-3 years	15 mg	15 mg		
4-8 years	25 mg	25 mg		
9-13 years	45 mg	45 mg		
14-18 years	75 mg	65 mg	80 mg	115 mg
19-50 years	90 mg	75 mg	85 mg	120 mg
51-70 years	90 mg	75 mg		
70+ years	90 mg	75 mg		

*Adequate Intake

An intake of 10mg a day may prevent scurvy, but might not be enough to prevent colds or chronic diseases like arthritis.

In order to find the optimal dose, it is necessary to examine a range of intakes – the research has not been done.

This book presents an open challenge to the government "experts," who support the out-of-date RDA approach to nutrition and thereby endanger the health of the entire population. The authors therefore assert that the RDA and the Codex justification for low intakes of vitamin C are both invalid and indefensible.

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.

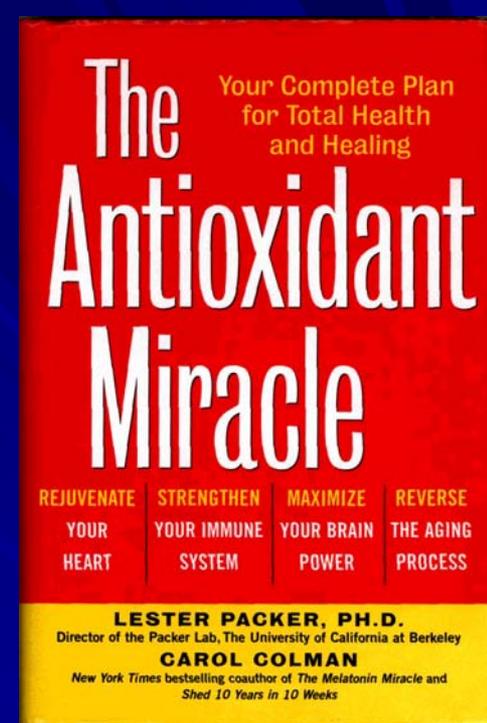
MSM is a naturally-occurring nutrient found in small amounts of many foods. As a dietary supplement, MSM is synthesized. When made correctly, it is identical to that found in nature. MSM can be taken alone or in combination with other joint health supplements, such as glucosamine and chondroitin.

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.

Using Multiple Pathways A Possible Explanation

1. Normally Glucose and Vitamin C are taken up by the Glucose pathway; as adapted from Lester Packer - *The Vitamin E. Ascorbate and Alpha Lipoate Antioxidant Defense System*. "Glucose and Vitamin C are taken up into the cells by the same transport system."
2. It is theorized by more than one researcher that some forms of C with **GMS Ribose** and **B.E.E.T.™** Metabolites do not use the Glucose pathway exclusively for transport but use multiple, entirely different, separate and unique pathways to the cell. Many nutrients utilize the Glucose pathway for absorption, however it appears that some forms of C do not have to **"WAIT IN LINE"** to be absorbed into the cell.



Vitamin C helps control gene activity in stem cells

NewsRx.com

08-09-13

NewsRx™

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!

stem-cell scientist Miguel Ramalho-Santos, PhD, who led the study. In fact, the unanticipated 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>

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



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.

Vitamin C (Beyond C™/BioEn'R-G'Y™) Challenge Test

Research Report RPN 9911

December 17, 2004

Silliker Inc.

Objective:

The objective of this study was to conduct a challenge test to assess the **micro-biological stability** of a product when challenged with one strain each of yeast, mold, lactic acid bacteria, Salmonella and Staphylococcus aureus.

Conclusions:

The AIBMR Life Sciences Beyond C Enhanced Vitamin C Powder with GMS Metabolite stored at ambient room temperature (73-77F) had a microbiological shelf-life of 28 days and **was microbiologically stable against challenge with Salmonella, Staphylococcus, lactic acid bacteria, yeast and mold for 28 days.**

Acute oral toxicity study of Beyond C™
With 14-day post-treatment observation period in the rat (limit test)
(Study code: PCDL-0410)

SUMMARY

TRUE COPY/IGAZOLT MASOLAT
PC&DL/GYEL
Valid in red only/Csak pirosan
ervenyes

General information:

Single oral limit dose of 2,000 mg/kg body weight of Beyond C™ (Lot number: G 4077) was applied to rats orally by gavage. Animals were observed for lethality and toxic symptoms for 14 days. Gross pathological examination was carried out on the 15th day.

Body Weight:

The body weight of the animals corresponded to their species and age throughout the study.

Evaluation:

No death occurred after oral administration of Beyond C™ at 2,000 mg/kg dose.
No toxic clinical symptoms were observed.
Scheduled autopsy carried out on Day 15 revealed no toxic gross pathological changes.

Conclusion:

No adverse effects were noted at single oral dose of 2,000 mg/kg Beyond C™ in male and female rats.

The trouble with high doses of vitamin C is that its almost impossible to take high doses orally – until now!

Most products on the market today will upset your stomach and bowels if you take too much.

But now there's a product called Bio E'nR-G'y C that's less likely to upset your gastrointestinal tract if you have to take up to 1 teaspoon of it per hour mixed in a beverage.

You can do a simple test to see if you are getting enough vitamin C. Longevity Plus also sells VitaChek C reagent strips for urine analysis. Buy these and test your urine. If the strip is bright yellow, then you have at least 100 mg/dl of vitamic C present in your blood. This is the optimal amount of vitamin C in your sytem."



Dr. Robert Jay Rowen's

SECOND OPINION

Vol. XX, No. 2

February 2010

HEALTH NOTES

How to Get the Benefits of Hydrogen Peroxide Without an IV

For years, I've told the readers of my newsletter that intravenous hydrogen peroxide can work miracles for many illnesses. Unfortunately, conventional medicine's attacks on the therapy have caused many doctors to stop using it. That's the bad news. The good news is there's a way you can get some of the same effects from another treatment that's not nearly as controversial.

Just in this case, I'm sure the hydrogen peroxide effect is helping with the reduction in tumor size. But I'm not so sure all of vitamin C's cancer-reducing effects are due to its ability to produce hydrogen peroxide. Vitamin C has the ability to save lives due to many other probable mechanisms of action, not just from its ability to produce hydrogen peroxide.

The trouble with vitamin C is that it's almost impossible to take high doses orally. Most vitamin C products on the market will upset your stomach and bowels if you take too much. So, for years, taking it intravenously was the only way to get adequate doses. But now, there's a product

(Continued on page 2)

Breakthrough Treatment Wipes Out Parkinson's, Alzheimer's, and Other Chronic Illness, Part 2

Last month, I showed you one of the most amazing breakthroughs in medicine I've seen in years. If you suffer from any disease that threatens your life or your ability to live life to the fullest, then you have to read the rest of the story.

The breakthrough is the controversial Stem Cell Therapy. But this Stem Cell Therapy is unlike any you've

“A recent study from NIH reported that high doses of IV Vitamin C can reduce the size of many cancers...”

aborted human baby. This is illegal in the U.S., but many offshore clinics have been using fetal cells from eastern European aborted pregnancies. But to get this therapy, you'll have to travel to an unregulated clinic in a foreign country and then pay \$25,000 or more for these cells.

Umbilical cord blood stem cells don't carry any moral or ethical problems. It's an easy routine procedure to collect cord blood from the cut cord. Once harvested, the placenta and umbilical cord cells are taken to a lab. There, lab technicians grow the cells in culture, and then doctors can administer them to patients.

Both of these procedures have some limitations. If there's not a good tissue-type match (like blood typing), the cells won't last as long as a perfect match. The cells will survive long enough to impart their growth factors into your diseased organs. But your body will eventually

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-

cretion 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.

Table 1. Comparison of plasma vitamin C with urine vitamin C in 6,537 patients.

Range of Plasma C mg/dL, (number of patients)*	Number (%)	
	0 - 20 mg/dL Urine C	30 - 40+ mg/dL Urine C
4.3 to 5.0 (n=36)	2 (6%)	34 (94%)
3.6 to 4.2 (n=55)	7 (13%)	48 (87%)
3.1 to 3.5 (n=80)	16 (20%)	64 (80%)
2.6 to 3.0 (n=187)	41 (22%)	146 (78%)
2.0 to 2.5 (n=627)	280 (46%)	347 (54%)
1.4 to 1.9 (n=1681)	939 (59%)	692 (41%)
0.6 to 1.3 (n=3059)	2303 (75%)	756 (25%)
0.1 to 0.5 (n=812)	687 (84%)	125 (16%)

*= plasma vitamin C measured by HPLC (normal value is 0.6 to 2.0 mg/dL), urine by VitaChek-C[®]

VitaChek-C from Teco Diagnostics

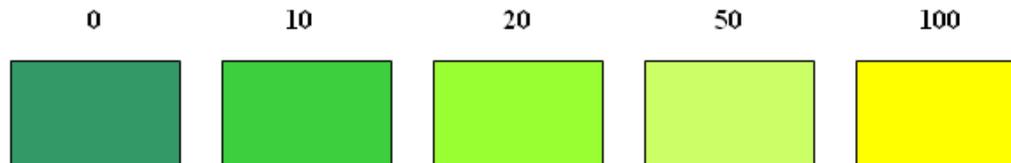
Reagent Strips For Urinalysis

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.



COLOR CHART – mg/dL vitamin C (Ascorbic Acid)



Directions:

1. Dip reagent strip in freshly collected urine and remove immediately or alternatively, wet the reagent strip by passing through the urine stream.
2. While removing, run the edge of the strip against the rim of the urine collection cup to remove excess urine.
3. 30 seconds after removing from urine, compare reagent side of test area with corresponding color chart.

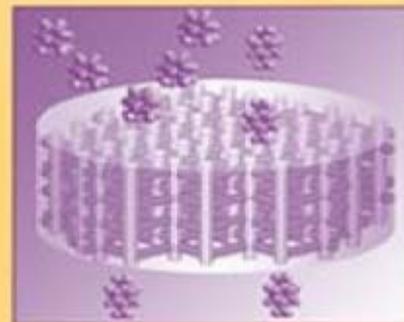
A BRIGHT SPOT
on this
urine stick test
means
you will have
a brighter future!

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).

HANDBOOK OF ZEOLITE SCIENCE AND TECHNOLOGY



EDITED BY
SCOTT M. AUERBACH
KATHLEEN A. CARRADO
PRABIR K. DUTTA

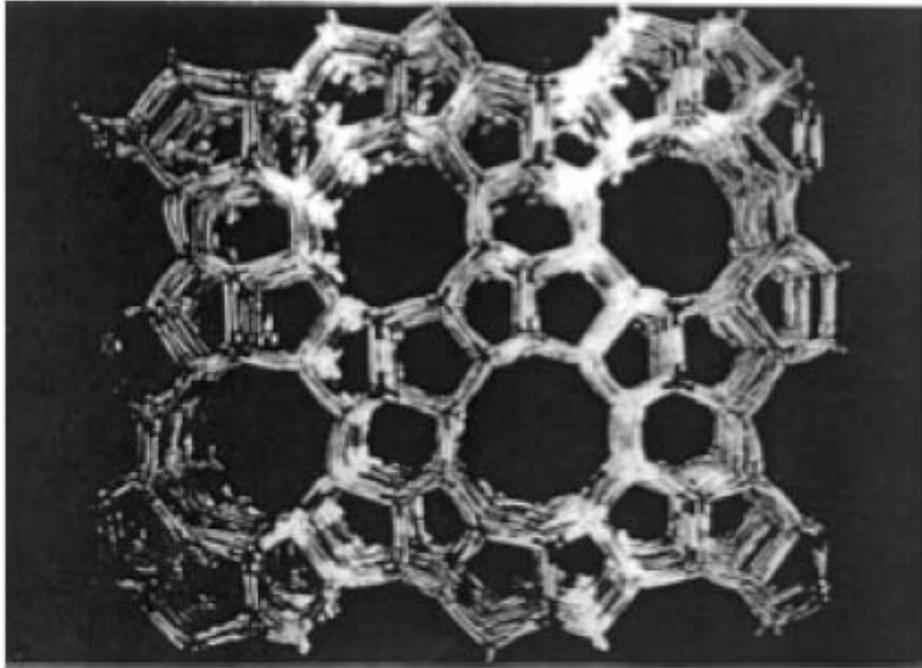


FIGURE 1 A repeated unit cell of the crystal structure of hydrophobic silica zeolite (MHZ™) molecular sieve; anesthetic agent is selectively captured in the honeycomb-shaped silica zeolite crystals. In just 500 g of silica zeolite, the total internal surface area available for capturing anesthetic would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

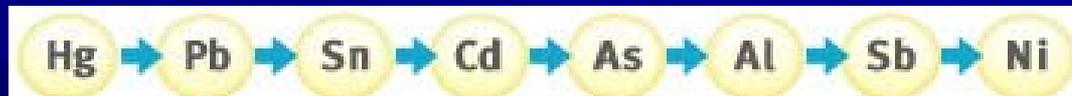
Breakthrough in Heavy Metal Chelation, Zeolite Safely Removes Mercury, Lead and More.

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.

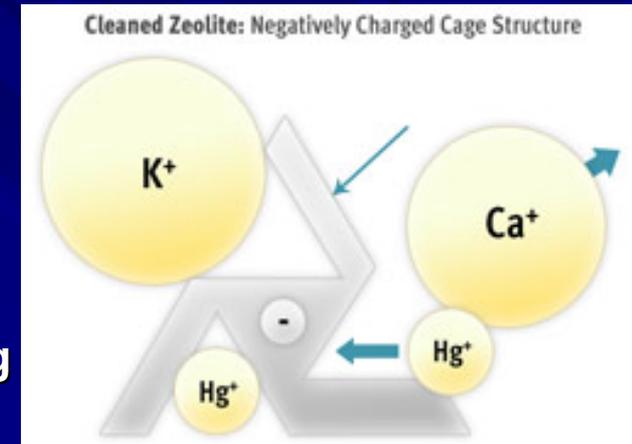
Another advantage is that unlike acid-based chelating agents, Zeolite molecules irreversibly bind to the toxic element in three key ways, with the strength of the bond based upon:

- 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



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

24 – Medical Applications of Zeolite (cont.) from pg 1146

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*.

24 – Medical Applications of Zeolite (cont.) from pg 1145

III. Radioprotection

Many researchers have demonstrated the ability of several natural zeolites to take up certain radionuclides (e.g., ^{90}Sr , ^{137}Cs , ^{60}Co , ^{45}Ca , and ^{51}Cr). Zeolite mordenite has effectively decontaminated soils contaminated with ^{137}Cs and ^{90}Sr .

Clinoptilolite shows a significant protective effect reducing radiocesium-137 accumulation in male broiler chickens exposed to alimentary contamination. The reduction of radiocesium in meat ranged between 60% and 70% and in edible organs it was greater than 50%.

Clinoptilolite supplementation in food eliminated ^{137}Cs deposition in some organs and tissues. After dietary administration with 2.5%, 5.0% and 10% zeolite, ^{137}Cs elimination increased and the radionuclide deposition in liver, kidneys, and femoral musculature decreased. The clinoptilolite decontamination effects were observed with preventive administration, as well as with sorbent administration from 24 h after a single contamination.

Clinoptilolite is an excellent sorber for both cesium and strontium ions and can be used for the treatment of radioactive wastewater and other decontamination purposes.

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 or 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, whereas clinoptilolite-treated diabetic mice had a higher concentration of Ca in sera, ranging from 2.15 to 2.3 mM/L. Iron (Fe^{2+}) containing, natural clinoptilolite interacts with glucose with formation of an iron-glucose complex in the clinoptilolite.

XVI. Toxicology of Clinoptilolite

In human medicine, zeolites have been used as anti-diarrheal remedies, for the external treatment of skin wounds and athletes foot, and in kidney dialysis for the removal of ammonia ions from body fluids. There were not many data showing the systemic effects of zeolites on physiological systems of the body.

The beneficial effects of zeolites on hematopoiesis, and various disease states, including tumors, have been observed. No toxic effects were observed in our toxicology study of clinoptilolite. The physical status of examined animals showed no evidence of any harmful reaction during the studies.

Clinoptilolite is well suited for these applications because of its large pore space, high resistance to extreme temps, and chemically neutral framework. The conclusion from all toxicology studies available is that natural clinoptilolite is not toxic and can be used in human as well as veterinary medicine.

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



Zeolites have known biological properties along 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.

Modified Natural Clinoptilolite Detoxifies Small Mammal's Organism Loaded with Lead II: Genetic, Cell, and Physiological Effects.

Topashka-Ancheva M, Beltcheva M, Metcheva R, Rojas JA, Rodriguez-De la Fuente AO, Gerasimova T, Rodríguez-Flores LE, Teodorova SE.



Abstract

The detoxification capacity of the clinoptilolite modification KLS-10-MA used as food additive in small mammals, chronically lead-exposed, was proven for the first time. The modified clinoptilolite was prepared based on natural Bulgarian clinoptilolite deposits. As a powder, it was mechanically mixed at 12.5% concentration with the conventional forage for small rodents. Lead in the form of aqueous solution of $Pb(NO_3)_2$ was diluted in the drinking water. In the ecotoxicological experiment covering 90 days, imprinting control region laboratory mice were used. They were allocated into four groups: group 1, (control): animals fed with conventional food for small rodents and water; group 2: animals fed with conventional food + clinisorbent KLS-10-MA and water; group 3: animals fed with conventional food and water + $Pb(NO_3)_2$; and group 4: animals fed with conventional food + KLS-10-MA and water + $Pb(NO_3)_2$. A group of non-exposed healthy animals was fed with conventional forage mixed with KLS-10-MA to prove eventual toxicity of the sorbent and influence on growth performance. The changes in the chromosome structure, mitotic index, erythrocyte form, erythropoiesis, and body weight gain were recorded. On day 90, the following relations were established: Pb-exposed and clinoptilolite-supplemented mice exhibited 2.3-fold lower chromosome aberrations frequency, 2.5-fold higher mitotic index, and 1.5-fold higher percentage normal erythrocytes 1.3-fold higher body weight compared to Pb-exposed and unsupplemented animals. The obtained data showed that the sorbent is practically non-toxic.

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 neurodegeneration. 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.

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 (glutathione peroxidase, superoxide dismutase, glutathione 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, glutathione peroxidase, superoxide dismutase and glutathione 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.

PubMed.gov

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

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.



Regulation of glutathione in inflammation and chronic lung diseases.

Department of Environmental Medicine, Division of Lung Biology and Disease Program, University of Rochester Medical Center, Rochester, NY 14642, USA.



Oxidant/antioxidant imbalance, a major cause of cell damage, is the hallmark for lung inflammation. Glutathione (GSH), a ubiquitous tripeptide thiol, is a vital intra- and extra-cellular protective antioxidant against oxidative stress, which plays a key role in the control of signaling and pro-inflammatory processes in the lungs.

Recent evidences have indicated that Nrf2 protein, which binds to the erythroid transcription factor (NF-E2) binding sites, and its interaction with other oncoproteins such as c-Jun, Jun D, Fra1 and Maf play a key role in the regulation of GCL. Alterations in alveolar and lung GSH metabolism are widely recognized as a central feature of many chronic inflammatory lung diseases. Knowledge of the mechanisms of GSH regulation could lead to the pharmacological manipulation of the production and/or gene transfer of this important antioxidant in lung inflammation and injury.

This article describes the role of AP-1 and ARE in the regulation of cellular GSH biosynthesis and assesses the potential protective and therapeutic role of glutathione in oxidant-induced lung injury and inflammation.

PMID: 16054171 [PubMed - as supplied by publisher]

Nutritional regulation of glutathione in stroke

College of Pharmacy and Nutrition, The Cameco MS Neuroscience Research Center,
University of Saskatchewan, 110 Science Place, Saskatoon, SK S7N 5C9, Canada.



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

In contrast to cardiovascular disease, the impact of nutritional status on the prevention and outcome of stroke has received limited investigation. We present a mechanism based on animal studies, clinical data, and epidemiological data by which protein-energy status in the acute stroke and immediate post-injury periods may affect outcome by regulating reduced glutathione (GSH), a key component of antioxidant defense.

As cysteine is the limiting amino acid for GSH synthesis, the GSH concentration of a number of nonneural tissues has been shown to be decreased by fasting, low-protein diets, or diets limiting in sulfur amino acids. The mechanism may also be relevant in brain since GSH in some brain regions is responsive to dietary sulfur amino acid supply and to the pro-cysteine drug, L-2-oxothiazolidine-4-carboxylate. The latter is an intracellular cysteine delivery system used to overcome the toxicity associated with cysteine supplementation. These findings may provide the mechanism to explain both the inverse correlation between dietary protein and stroke mortality and the documented association between suboptimal protein-energy status and diminished functional status following a stroke.

Finally, micronutrient deficiencies that may accompany protein-energy malnutrition, such as selenium, should also be investigated for their role in antioxidant defense in cerebral ischemia.

PMID: 12835106 [PubMed]

Glutathione status in critically-ill patients: possibility of modulation by antioxidants

Department of Anaesthesia and Intensive Care, KFC, Huddinge Hospital,
Stockholm, Sweden. Jan.Wernerman@anaesth.hs.sll.se



Muscle tissue serves as a protein reservoir which is mobilized to meet the specific metabolic needs associated with various catabolic conditions in human subjects, such as trauma and critical illness. Glutathione is one of the most abundant short-chain peptides and a major source of non-protein thiol in the body, and tissue glutathione concentration is related to its oxidative capacity.

Skeletal muscle is relatively unique with respect to a variety of metabolic properties, such as oxidative potential, patterns of amino acid utilization, and antioxidant enzyme activity. The glutathione concentration is not influenced by food intake, or by food deprivation. Moreover, there is no diurnal variation on muscle glutathione levels. Following elective surgery the muscle concentration of GSH (the reduced form) decreases by 40% 24 h post-operatively, while the concentration of GSSG (the oxidized form) remains unaltered. During critical illness a similar decrease in the GSH concentration is seen, but in addition a change in the redox status indicative of an elevated GSSG level occurs. Furthermore, correlations between the concentrations of glutamine as well as glutamate and GSH exist in these patients. From available evidence accumulated it is clear that glutathione plays a pivotal role in the maintenance of the intracellular redox status, the antioxidant vitamin levels, and the antioxidant enzyme functions under various metabolic conditions.

PMID: 10604202 [PubMed - indexed for MEDLINE]

Dr. David Perlmutter's glutathione Parkinson's video

<http://www.youtube.com/watch?v=uQRCpdcGwIU>



Video shows the use of intravenous glutathione in Parkinson's disease patients at the Perlmutter Health Center, Naples, Florida

66 yr. Military Veteran exposed to agent orange. Diagnosed with Parkinson's and prescribed Mirapex and Sinemet. Difficulty with speech, motor functioning and balance; walks with shuffling gait.

30 minutes after 3000 mg IV glutathione, there is a noticeable, albeit temporary improvement in patient .

Speech is clearer and smoother, no more stutter; walking and turning much improved as well, patient able to walk and turn without shuffling or losing balance.



L-Glutathione – The “Mother” of Antioxidants

Glutathione is a powerful antioxidant. It is water-soluble and is primarily synthesized in the liver. It is involved in DNA synthesis and repair, protein and prostaglandin synthesis, amino acid transport, metabolism of toxins and carcinogens, immune system function, prevention of oxidative cell damage and enzyme activation.

Cellular glutathione levels increase during exercise.

Glutathione deficiency is associated with aging, age-related macular degeneration (AMD), diabetes, lung and gastrointestinal disease, pre-eclampsia, Parkinson's disease, other neurodegenerative disorders and poor prognosis in AIDS.

Glutathione may inhibit the activity of enzymes that help the flu virus colonize cells lining the mouth and throat. Flu-infected mice fed glutathione-enriched drinking water have lower tissue virus levels than untreated mice. Human studies are needed to determine the effects on flu infection.

Lipoic acid may reduce the toxic effects of heavy metals

Submitted by shunsmuse on Tue, 03/19/2013 - 03:51

Toxicol Ind Health. 2013 Jan 4. Monitoring the toxic effects of Pb, Cd and Cu on hematological parameters of Wistar rats and potential protective role of lipoic acid and glutathione.

Nikolic R, Krstic N, Jovanovic J, Kocic G, Cvetkovic TP, Radosavljevic-Stevanovic N.

Faculty of Sciences and Mathematics, University of Nis, Nis, Serbia.

Heavy metal pollution is a serious environmental and health problem. The negative effects of heavy metals that can enter human body can be reduced by the addition of some supplements. In this study, the effects of lead (Pb), cadmium (Cd) and copper (Cu) on the hematological parameters in Wistar rats in the absence and presence of lipoic acid and glutathione were analyzed. Pb, Cd and Cu intoxication significantly affected the hematological parameters of treated animals. The main effects in the case of Pb and Cd intoxication were decreased values of erythrocytes, hemoglobin and hematocrit (up to 30% and 20% for these two metals, respectively) compared with the control group. Cu intoxication caused decrease in hematocrit, thrombocytes, mean cell volume values (c.a. 15%) and slight decrease in the erythrocyte number, while the value of hemoglobin increased (c.a. 7%). The treatment with lipoic acid and glutathione reduced the toxic effects of these metals in all cases.

Oral supplementation with antioxidant agents containing alpha lipoic acid: effects on postmenopausal bone mass.

Submitted by shunsmuse on Tue, 03/19/2013 - 03:43

Clin Exp Obstet Gynecol. 2012;39(4):489-93.

Mainini G, Rotondi M, Di Nola K, Pezzella MT, Iervolino SA, Seguino E, D'Eufemia D, Iannicelli I, Torella M. San Leonardo Hospital, Castellammare di Stabia, Naples, Italy.

Oxidative stress impacts many age-related degenerative processes, such as in postmenopausal bone loss and in antioxidant defenses that are significantly decreased in elderly osteoporotic women. The authors evaluated the effect of oral supplementation with antioxidant agents containing alpha lipoic acid (ALA) on bone mineral density (BMD) of osteopenic postmenopausal women.

Forty-four patients completed the one-year study: 23 in the ALA group, 21 in the control group. The treatment of ALA group led to a better estimated BMD compared to the control group (0.401 +/- 0.026 vs 0.388 +/- 0.025 g/cm²), although this difference barely achieved a statistical significance (p = 0.048).

These findings, although in a small population, could suggest that **oral supplementation with antioxidant agents containing ALA may mitigate bone loss in osteopenic postmenopausal women.**

R-Lipoic acid protects against oxidative stress in retinal neurons

Submitted by shunsmuse on Fri, 03/15/2013 - 17:43

Brain Res. 2013 Jan 4. pii: S0006-8993(13)00003-6. doi: 10.1016/j.brainres.2012.12.041.

Oxidative stress plays a key role in neurodegeneration of CNS neurons such as Alzheimer disease, Parkinson's disease and glaucoma. R- α -lipoic acid (R-LA) has been shown to have a neuroprotective effect through its antioxidant activity. However, the mechanism underlying its neuroprotection is totally unknown in retinal neurons.

In this study, we show that R-LA has a dramatic neuroprotective effect against oxidative stress-induced death of the retinal neuronal RGC-5 cell line. R-LA produced reactive oxygen species (ROS), including hydrogen peroxide.

Results suggest that ROS production triggered by R-LA might modify Kelch-like ECH-associated protein (Keap1), which in turn induces HO-1 expression through the PI3K signaling pathway. Furthermore, R-LA significantly attenuated cell death and accumulation of 4-hydroxy-2-nonenal (4HNE) in the retina induced by optic nerve injury in vivo through an HO-1 activity-dependent mechanism.

These data demonstrate for the first time that R-LA exerts a neuroprotective effect against oxidative stress in retinal neurons in vitro and in vivo by inducing HO-1 through Keap1/Nrf2 signaling.

Humates - Humic and Fulvic Acid



Humates contain both humic and fulvic acids. The fulvic acid is the chelator that carries the minerals. The humic acid acts as dilator increasing the cell wall permeability. This increased permeability allows easier transfer of minerals from the blood to the bone and cells.

Red blood cells have the capability of carrying higher percentages of oxygen when in the presence of humate. Healing of injuries, as a result of additional oxygen, is much quicker.

Literature reports additional transport of iodine from foods into the thyroid glands. Just as fulvic acid carries life-sustaining minerals to the body, it also captures and removes toxic metals from the body. Detoxification takes place within first three to four days of usage.

Humic substances, including peat and sodium humates, are known to exhibit anti-inflammatory properties. Inflammatory states of the cervix, especially cervical erosion (generally known as cervicitis) can be treated with humic preparations.

Humate takes an active part in the liver metabolism. The use of humate plays a role in the liver function and protects it somewhat from disease and/or disturbances

Detoxing and Chelating With Fulvic Acid

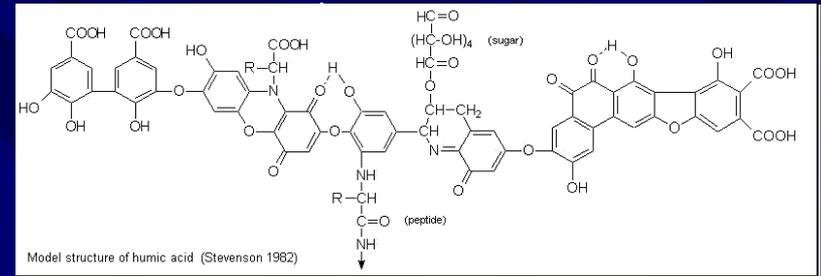
Even though not as touted and well known as zeolite products, fulvic acid has been around for a very long time. It has even been used in Ayurvedic Medicine, possibly the oldest accessible healing protocol in existence. It is a powerful overall detox and heavy metal chelation agent. It is well researched and used within the mainstream medicine halls of China, Russia, and India. The Tibetan Mountains offer possibly the richest source of pristine fulvic acid shales.



Fulvic acid is sometimes used as a liquid base for Zeolite molecules, even though it has its own chelation properties similar to zeolite. Fulvic acid has many other health restorative ramifications, which are supported by clinical reports. It has been used successfully in China to treat a variety of serous, stubborn lung disorders.

The lungs and the brain are the most obviously affected organs from chemtrail spraying. The health benefits observed and recorded for fulvic acid are too numerous to mention in this article. Check out the appropriate URL or link in the "sources area" below for a pdf medical report dealing with fulvic acid uses in clinics and hospitals. Fulvic acid is available and inexpensive.

Humates within the body work with DNA and cellular division.



It has been noted that the **humate tends to prevent cellular mutation during reproduction**. Several technical papers were noted during literature research for this paper regarding cancer research with humates. Natural humic acid administered prophylactically to rats can decrease significantly the amount of gastric mucus damage induced with ethanol. Humic acid also significantly accelerated the healing process of experimentally induced ulcers (52).

Humates exhibit anti-microbial and anti-viral properties, thus bolstering the immune system.

Dr. Daryl See, MD, formerly an Immunologist of UCI Medical School, suggests that the mechanism is related to the **humates ability to complex (assemble) sugars within the body**.

The abundance of these complexed sugars allows the body to manufacture glyco proteins (glyco nutrients) that attach to the killer and T cell acting as a modulator or communication link between the cells.

Visible benefit using Zeolite-based topical cream.

Zeolite has the ability to draw out impurities, and neutralize free radical damage that age the skin.



Zeolite as an antioxidant also helps to restore natural pH-value, balancing acid and irritated skin conditions.

WHAT'S HYDROGEN GOT TO DO WITH IT?

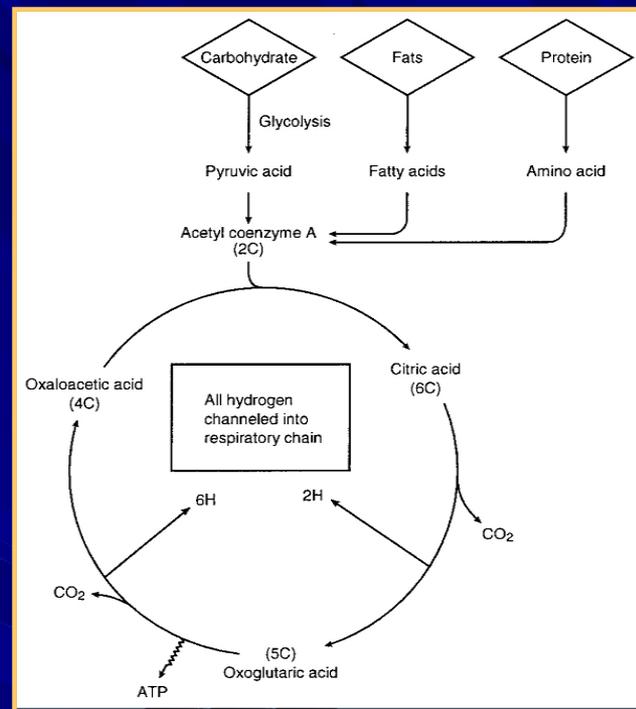
Albert Szent-Gyorgyi, the Hungarian Nobel Prize winning biochemist who discovered Vitamin C, said that hydrogen rather than oxygen, is the fuel of life.

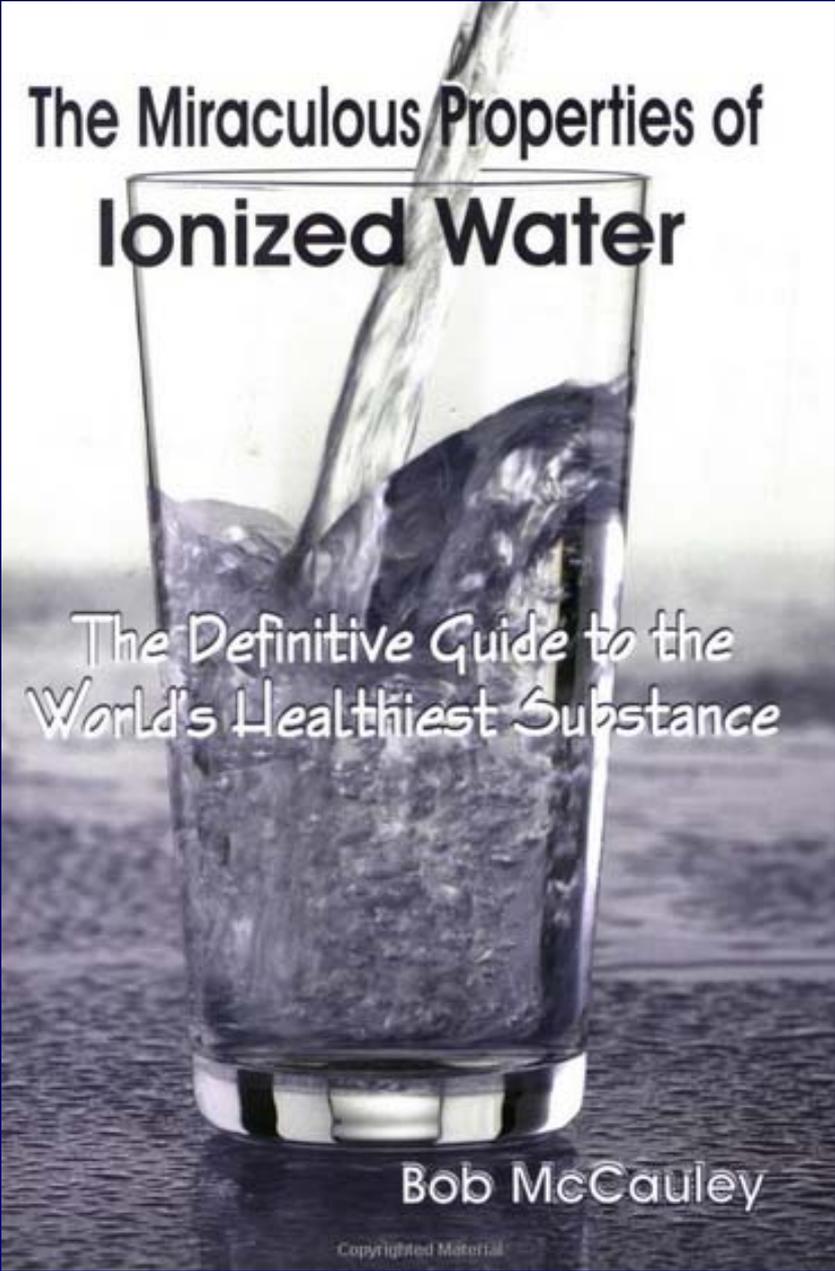
Hydrogen is the body's most needed nutrient, our Primordial ANTI-OXIDANT!

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.



A black and white photograph of a glass of water being poured. The water is captured in motion, splashing into the glass. The background is a soft, out-of-focus landscape. The text is overlaid on the image.

The Miraculous Properties of Ionized Water

*The Definitive Guide to the
World's Healthiest Substance*

Bob McCauley

Copyrighted Material

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 alkalized.

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

Biochim Biophys Acta. 2005 Sep 25;1756(1):1-24.

The role of pH dynamics and the Na⁺/H⁺ antiporter in the etiopathogenesis and treatment of cancer.

Two faces of the same coin--one single nature.

Harguindey S, Orive G, Luis Pedraz J, Paradiso A, Reshkin SJ.
Centro Médico La Salud, Independencia, 13-01004 Vitoria, Spain.



Conventionally, cancer represents a daunting and, frankly, confusing multiplicity of diseases (at least 100) that require an equally large variety of therapeutic strategies and substances designed to treat the particular tumor. However, when analyzed phenotypically, cancer is a relatively uniform disease of very conserved hallmark behaviors across the entire spectrum of tissue and genetic differences.

Cancers share common biochemical and physiological characteristics independent of the varied genetic backgrounds, and that there may be a common mechanism underlying both the neoplastic transformation/progression side and the antineoplastic/therapy side of oncology.

Hydrogen ion-dependent oncogenesis and parallel new avenues to cancer prevention and treatment using a H⁺-mediated unifying approach: pH-related and pH-unrelated mechanisms.

Cancer cells have an acid–base disturbance that is completely different than observed in normal tissues and that increases in correspondence with increasing neoplastic state: an interstitial acid microenvironment linked to an intracellular alkalosis.

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

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(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

Effectiveness of Hydrogen Rich Water on Antioxidant Status of Subjects with Potential Metabolic Syndrome—An Open Label Pilot Study

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Received 15 October, 2009; Accepted 6 November, 2009; Published online 24 February, 2010

Summary Metabolic syndrome is characterized by cardiometabolic risk factors that include obesity, insulin resistance, hypertension and dyslipidemia. Oxidative stress is known to play a major role in the pathogenesis of metabolic syndrome. The objective of this study was to examine the effectiveness of hydrogen rich water (1.5–2 L/day) in an open label, 8-week study on 20 subjects with potential metabolic syndrome. Hydrogen rich water was produced, by placing a metallic magnesium stick into drinking water (hydrogen concentration; 0.55–0.65 mM), by the following chemical reaction; $\text{Mg} + 2\text{H}_2\text{O} \rightarrow \text{Mg}(\text{OH})_2 + \text{H}_2$. The consumption of hydrogen rich water for 8 weeks resulted in a 39% increase ($p < 0.05$) in antioxidant enzyme superoxide dismutase (SOD) and a 43% decrease ($p < 0.05$) in thiobarbituric acid reactive substances (TBARS) in urine. Further, subjects demonstrated an 8% increase in high density lipoprotein (HDL)-cholesterol and a 13% decrease in total cholesterol/HDL-cholesterol from baseline to week 4. There was no change in fasting glucose levels during the 8 week study. In conclusion, drinking hydrogen rich water represents a potentially novel therapeutic and preventive strategy for metabolic syndrome. The portable magnesium stick was a safe, easy and effective method of delivering hydrogen rich water for daily consumption by participants in the study.

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 (H₂) has antioxidant and antiapoptotic activities that protect the brain against ischemia-reperfusion injury and stroke¹.

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 channel⁴. 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 viability⁵⁻⁷.

The lightweight gas diatomic hydrogen (H₂), 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 water⁸.

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 injury¹. 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

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

Ikuroh Ohsawa¹, Masahiro Ishikawa¹, Kumiko Takahashi¹, Megumi Watanabe^{1,2}, Kiyomi Nishimaki¹, Kumi Yamagata¹, Ken-ichiro Katsura², Yasuo Katayama², Sadamitsu Asoh¹ & Shigeo Ohta¹

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 (H₂) has potential as an antioxidant in preventive and therapeutic applications. We induced acute oxidative stress in cultured cells by three independent methods. H₂ selectively reduced the hydroxyl radical, the most cytotoxic of reactive oxygen species (ROS), and effectively protected cells; however, H₂ 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 H₂ gas markedly suppressed brain injury by buffering the effects of oxidative stress. Thus H₂ 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 (O₂[•]) produced is generated in mitochondria by electron leakage from the electron transport chain and the Krebs cycle⁶. O₂[•] is also produced by metabolic oxidases, including NADPH oxidase and xanthine oxidase⁷. Superoxide dismutase converts O₂[•] into hydrogen peroxide (H₂O₂)⁸, which is detoxified into H₂O by

RESULTS

H₂ selectively reduces •OH in cultured cells

H₂ reduces the •OH that is produced by radiolysis or photolysis of water¹²; however, whether H₂ 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 detectable, we induced O₂[•] production in BCL2 cultured cells. To do

**COMING
SOON!**

ZEO GOLD "Enhanced"



Medical Applications of Zeolite (pg 1141).

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 accommodate large cations, such as Na⁺, K⁺, Br⁺, and Ca⁺, and even large molecules and cationic groups, such as water, ammonia, carbonate ions, and nitrate ions. The basic structure of zeolites is biologically neutral.



ZEO GOLD ENHANCED

Advanced Zeolite Product containing an Advanced MicroHydro Colloidal Zeolite, with Cation Exchange ActiVator, Bio En'R-G'y C, GMS Ribose, And Glutathione Lipolate.

Zeo Gold uses a Purified, Micronized HydroColloidal certified Food Grade form of Zeolite. It was formed in petrified bubbles of the clinoptilolite volcanic rock providing ultra high surface area and higher CEC (cation exchange capacity) for more effective utilization that any other clinoptilolite with or without nano sizing.

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 range.

The resulting beverage provides exceptionally powerful electron donating properties, by donating more than 105 Active Nutritional Hydrogen Minus electrons for possible enzyme, ROS/free radical scavenging (oxidative stress), Detox, and Energy Enhancing functions.

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.

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)



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

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.

Dr. Charles Raison Awarded 2013 IMHR Pilot Grant \$20,000 from the Institute for Mental Health Research

Charles L. Raison, MD, Associate Professor of Psychiatry in the College of Medicine and Barry and Janet Lang Associate Professor in the College of Agriculture and Life Sciences, has been awarded a 2013 IMHR Pilot Grant of \$20,000 by the **Institute for Mental Health Research** to study, “Antidepressant Effects of Whole Body Hyperthermia (WBH)”.



These funds will augment existing extramural funding to help support an ongoing controlled study of mild whole body hyperthermia. This study builds upon prior open data from the research team suggesting that sensory pathways running from the brain to the body may be involved in the development of major depression and may hold promise as new ways of treating the disorder.

What's really remarkable in the work done so far is the evidence that is beginning to accumulate suggesting that it might be possible to treat this hugely costly mental illness by interventions based in the body, not in the brain," according to Dr. Raison. "Because sensory pathways evolved to have very specific effects on brain function and resultant behavior, we increasingly think they might be utilized to craft very specific and benign treatments for depression and anxiety. We have designed the hyperthermia study to begin really testing this possibility. ”



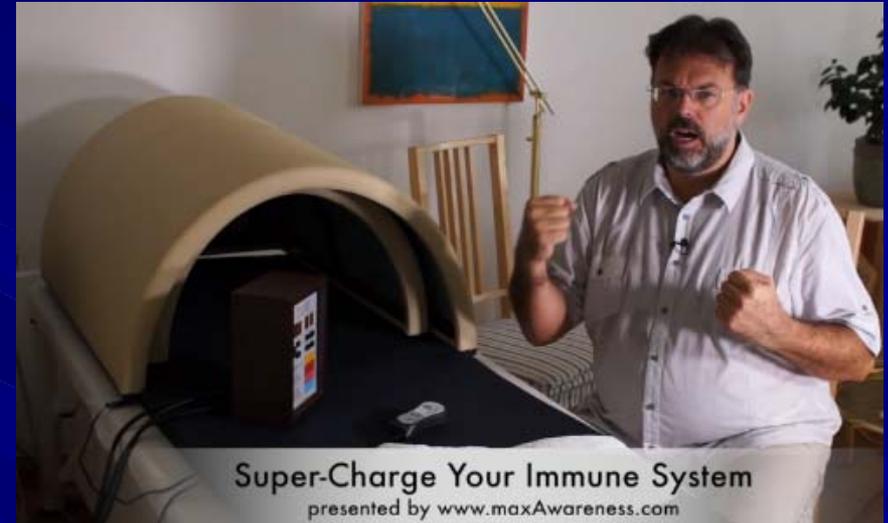
With over 8000 members this is the world's leading alternative cancer education program

FINALLY HYPERTHERMIA FOR ALL

By Marcus Freudenmann - 08-15-2013

Whole Body Hyperthermia is the most common sense treatment there is! It has NO toxic side effects what so ever while being extremely effective.

It increases your white blood cell count like no chemical substance ever can and arms your white blood cells with a protein that allows them to attack cancer cells more effectively. In combination with oxygen it increases hydrogen peroxide in your blood and it supports perfusion of anti cancer remedies into cancer cells.



NO EMF Sauna Dome 110V

The **FIR Dome Sauna** is made from the highest quality materials to ensure a lifetime of use and includes our advanced True Wave II™ heaters. The FIR Dome Sauna is comprised of two lightweight domes that make it very easy to store in a corner or closet. The FIR Dome Sauna can be setup anywhere and then stored away when not in use. The infrared mat allows you to have infrared heat on both the back and front of your body. Both the FIR Dome Sauna and infrared mat have a lifetime warranty.

<http://maxawareness.com/blog/finally-hyperthermia-for-all/>

MTS-7 Multiple Therapy System

6 in 1- Home System

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PEMF • LED Red Lights • LED Infrared Lights • E-Stim • Micro Current • Vibration Massage



Effective in treating the following conditions:

- Inflammation
- Pain Control
- Increased blood flow
- increased oxygen levels
- Temporary relief of minor muscle and joint pain
- Promotes the relaxation of muscle tissue
- Relief of post-surgical or post traumatic pain

For more information: info@nubiomed.us

PEMF – Home System

Advanced Magnetic Cellular Exercise

NuBioMed



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Use both attachments together or use them separately.

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For more information: info@nubiomed.us

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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.



4:42 / 7:32



Garry Gordon - Zeolite (ZeoGold™)

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

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[Genes](#) >
SKIV2L

The information on this page was *automatically extracted* from online scientific databases.

On this page: [Name](#) [Normal function](#) [Genetic changes](#) [Gene location](#) [Additional information](#)
[Other names](#) [About genes](#) [Glossary definitions](#)

What is the official name of the *SKIV2L* gene?

The official name of this gene is “superkiller viralicidic activity 2-like (*S. cerevisiae*).”

SKIV2L is the gene's official symbol. The *SKIV2L* gene is also known by other names, listed below.

Read more about gene names and symbols on the [About](#) page.

What is the normal function of the *SKIV2L* gene?

From [Entrez Gene](#) ⇨:

DEAD box proteins, characterized by the conserved motif Asp-Glu-Ala-Asp (DEAD), are putative RNA helicases. They are implicated in a number of cellular processes involving alteration of RNA secondary structure such as translation initiation, nuclear and mitochondrial splicing, and ribosome and spliceosome assembly. Based on their distribution patterns, some members of this family are believed to be involved in embryogenesis, spermatogenesis, and cellular growth and division. This gene encodes a DEAD box protein, which is a human homologue of yeast SKI2 and may be involved in antiviral activity by blocking translation of poly(A) deficient mRNAs. This gene is located in the class III region of the major histocompatibility complex. [provided by RefSeq, Jul 2008]

From [UniProt](#) ⇨:

Helicase; has ATPase activity. Component of the SKI complex which is thought to be involved in exosome-mediated RNA decay and associates with transcriptionally active genes in a manner dependent on PAF1 complex (PAF1C).



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- › Bone Health
- › Hormone Regulation
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For more information please contact Gordon Research Institute
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Garry F. Gordon MD, DO, MD(H)



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