Cancer
A Special Report

The Hidden Science

by

Thomas S. Ciraulo, MS, CL
Board Certified Holistic Health Practitioner
Nutrition/Wellness Coach, Educator, and Author of
"Learn the Simple Truth to Achieving Optimum Health"

E-mail: healthcoach9@gmail.com/ (516)-409-6978 / http://www.EnjoyAHealthyLife.com

Disclaimer:

The Information Provided In This Report Is Not Presented With Intention Of Diagnosing Or Prescribing, But Is Offered Only As Information For The Use In Maintaining And Promoting Health In Cooperation With A Physician. In The Event That The Information Presented In This Program Is Used Without A Physician's Approval, The Individual Will Be Diagnosing For Him/Herself. No Responsibility Is Assumed By The Author, Publisher, Or Distributors Of This Information Should Anyone Use The Information Contained Herein In Lieu Of A Doctor's Services. No Guarantees Of Any Kind Are Made For The Performance Or Effectiveness Of The Products Mentioned. This Research Is Being Made Available To Others In Their Quest To Explore And Do Their Own Research In The Area Of Cancer. The Findings Of The Research Found In This Report And The Findings From The Articles Found On PubMed.Gov Are No Indication Of Results That You May Achieve. You Have To Do Your Own Research Along With Your Physician To Determine What Would Work Best For You.

The following information is presented to help give you more ammunition in your fight with the war with Cancer. Some of the research found in this report may be a little technical, but I feel that you should know about it and discuss it with your health care provider. Many of the nutrients in the products discussed in this report are presented with the scientific studies I found on **www.PubMed.gov**. **PubMed.gov is a service of the U.S. National Library of Medicine and the National Institutes of Health.**

This report is intended to present to the reader, natural alternatives, with their scientific support, to fighting cancer. If you would like to discuss any of the information in this report I cam be contacted by calling **(516) 409-6978** or via e-mail at **healthcoach9@gmail.com**.

The sooner we start eating foods without chemicals and preservatives the healthier we will be and the less of a need to depend on medications. Yes, many prescriptions save many lives, but drugs, not just illegal drugs, but all drugs, have been known to KILL. They either kill your immune system and or liver and or your heart and or… and the list goes on and on.

Our bodies can take just so much before it gives out. So the sooner we all get back to basics and eating foods the way God/Nature intended, the sooner we could all enjoy better health.

I am going to start with the lymphatic system, which is a very integral part of your overall health.

Information on blood proteins of the blood and the role of the lymphatic system for removing them is quoted by Arthur C. Guyton in all 10 of his editions of **"Textbook of Medical Physiology,"** which is utilized by major medical schools for teaching. It quotes:

"The lymphatic system represents an accessory route by which fluid can flow from the interstitial spaces (the space that surrounds your cells) into the blood. Most important, the lymphatics can carry protein and large particulate matter away from the tissue spaces, neither of which can be removed by absorption directly into the blood capillaries. This return of proteins to the blood from the interstitial spaces is an essential function without which we would die within about 24 hours."

When Dr. Guyton refers to blood proteins and other particulate matter; he is referring to the "lymphatic load" which consists of blood proteins, fat, fluid, cellular wastes and toxins. All these include your dust particles, tattoo dye (80% of which is absorbed into the (lymphatics), cancer cells, etc. Remember, these are the large particulates. When a woman or anybody has lymph nodes or vessels removed in surgery, there is no path for these particulates to drain into,

so the trapped blood proteins attract water (because they are "hydrophilic") and this water then accumulates with the other wastes, causing tissue swelling, tightness, and eventually "lymphedema." Through manual lymphatic drainage and compression therapy, however, this fluid can be rerouted and the patients can learn to "drain" this fluid themselves. This technique now receives reimbursement through insurance companies with a ICD 9 and CPT codes! Many physicians don't know about the full impact of lymphedema, as many patients don't either, but they can contact the **National Lymphedema Network,** which now lobbies for legislation for coverage and has a site on the internet for cancer patients with lymphedema. Lots of info there for the cancer patient!

As far as the cells and cancer, look to **Dr. Otto Warburg, Nobel Peace Prize Winner in 1931 and 1944**, for his research published by the National Cancer Institute itself, on blood proteins (published in 1966) stating: that **the prime cause of cancer was "A LACK OF OXYGEN AND THAT CANCER CELLS CAN LIVE ON THE FERMENTATION OF GLUCOSE, WHICH OCCURS DUE TO THE LACK OF OXYGEN!"**

The following excerpts came from Wikipedia at **http://en.wikipedia.org/wiki/Otto_Heinrich_WarburgCancer hypothesis**

Main article: Warburg hypothesis

In 1924, Warburg hypothesized that cancer, malignant growth, and tumor growth are caused by the fact that tumor cells mainly generate energy (as e.g. adenosine triphosphate / ATP) by non-oxidative breakdown of glucose (a process called glycolysis). This is in contrast to "healthy" cells, which mainly generate energy from oxidative breakdown of pyruvate. Pyruvate is an end-product of glycolysis, and is oxidized within the mitochondria. Hence and according to Warburg, cancer should be interpreted as a mitochondrial dysfunction.

…"Cancer, above all other diseases, has countless secondary causes. But, even for cancer, there is only one prime cause. Summarized in a few words, the prime cause of cancer is the replacement of the respiration of oxygen in normal body cells by a fermentation of sugar." -- Dr. Otto H. Warburg in Lecture-Full article can be found here **http://healingtools.tripod.com/primecause1.html/**

…Warburg also wrote about oxygen's relationship to the pH of cancer cells internal environment. Since fermentation was a major metabolic pathway of cancer cells, Warburg reported that cancer cells maintain a lower pH, as low as 6.0, due to lactic acid production and elevated CO2. He firmly believed that there was a direct relationship between pH and oxygen. Higher pH means higher concentration of oxygen molecules while lower pH means lower concentrations of oxygen.

So, if you have the internal swamp of excess blood proteins, fats, cellular wastes and toxins (in other words, all that junk you dump into your body i.e.: additives, long chained fatty acids, tattoo dye, excessive salt etc.,) the cells cannot receive oxygen and become deformed and eventually die. Another point to make is that all that junk draws the excess water, causes the swelling of the tissues and even creates a "greater diffusion distance", meaning that the oxygen and nutrients can no longer reach the dying cells and tissue, which leaves a person susceptible to fibrosis (arthritis for example) and infections when that part of the body is injured.

Dr. H.S. Mayerson of the Tulane Medical School in LA was the first to "tag" these blood proteins using radioactive iodine. He determined how much of the blood proteins actually passed from the blood stream into the lymphatics based on a 24 hour time period, which is 1/2 of the circulating blood proteins (again quoted by Guyton in his 10th edition). Dr. Mayerson published his findings in the AMA's publication "Today's Health" in 1964. Mayerson also went on to help establish the **International Society of Lymphologists** in 1966, whose members are now viewed as the leading medical pioneers of that field.

Research by members of the ISL have now found that the lymphatics, even when damaged, can be regenerated with high pressures of oxygen, lymphatic drainage massage, exercise, deep breathing, etc. to the extent that they create a "plexus" or new network of vessels, most without valves, but with the above techniques, you can move the fluid through the lymph system. What is so exciting is that if we begin treatment at the "cellular" level, instead of treating symptoms with radiation, surgery, etc., by good nutrition (live foods) no additives, no sugars, etc., we can reverse fibrosis seen in arthritis, decrease the symptoms of such dis-ease states such as muscular dystrophy, multiple sclerosis,

etc. What we need to emphasize is that this is at the CELL LEVEL. Give the cell a nutritious, balanced environment, and it will function at optimum efficiency, you just have to remember to keep the solution it is in, CLEAN.

Now let's go to one of the members of the International Society of Lymphologists, Dr. C. Samuel West.

Dr. West held two doctorates in the natural healing arts, yet he was first a chemist and a lymphologist. This was the combination that enabled him to understand the life and death processes at the cell level.

Teaching chemistry during the day, Dr. West established an afternoon and evening clinic where he treated patients at the Naturopathic Clinic of Mesa for two years.
In the early part of 1974, while teaching chemistry, he learned that the proteins that make up the blood stream - namely albumin, globulin, and fibrinogen- can produce the conditions at the cell level that can cause death in just a few hours. This is the key to the life and death processes at the cell level.

In 1976, he discovered The Bio-Electric Gentle Bounce for Health and other bio-electric lymphacising techniques, which enabled people to relieve pain, speed up the healing process, and reverse injury and disease.

Basically, Dr. West was able to develop exercises, which can help your cells, go back to the **"Dry State"** and as we learned from Drs. Warburg and Guyton, the **"Dry State"** is where Cancer has difficulty or is unable to flourish.

As a result of these discoveries, as early as 1976 Dr. West was invited to lecture in approximately two-hundred and sixty-five cities per year all over the United States and in Canada for seventeen years.

In October-November of 1979, he went to Italy to attend the Seventh International Conference of Lymphology. He was pleased to learn at this Congress that the International Society of Lymphology is composed of some of the greatest surgeons, doctors, and scientists in the world.

Also at the ISL Conference, he learned that this society was "not a pure medical society", and that it embraced other professions as well. He applied for membership, and on February 22, 1980, Dr. West was accepted as the 379th member of The International Society of Lymphology.

So my question to you is, if the medical profession and scientific community know of these discoveries of Drs. Warburg, Guyton and West. WHY ARE MORE THAN 500,000 PEOPLE DYING OF CANCER EVERY YEAR AND COUNTLESS THOUSANADS OF PEOPLE HAVEING THEIR BODIES MUTALATED BY SURGERY EVERY YEAR???

I had to include an e-mail I received from one of my clients. It supports the fact that some doctors **DO** know that an acidic environment supports cancer growth and an **alkaline environment DOES NOT SUPPORT CANCER GROWTH.**

Here is the message I received:

Hi Tom --

Hope all is well with you.

As my nutritionist, I wanted to update you on my visit with Dr. X. Based on the MRI findings - being inconclusive - I am scheduled for a needle biopsy on Monday to make a further determination.

I did, however, tell Dr. X that my pH level was around 7.5 and he responded: **' then how can cancer live, I don't understand.'** I, of course, told him that I had a difficult two years, and while it is 7.5 now it was probably not until recently.

I will keep you posted, and let you know how Monday goes.

Thanks,
Andrea

Keep reading and hopefully you and your loved ones will not become a cancer statistic.

You have many options to help you keep your lymphatic system working at optimum efficiency. You can:

a) Have a message therapist do lymphatic massage.

b) Contact the **National Lymphedema Network, Latham Square, 1611 Telegraph Avenue, Suite 1111, Oakland, CA 94612-2138, Hotline : 1-800-541-3259 or 510-208-3200 Fax: 510-208-3110**

c) Contact the **Lymphology Association of North America (LANA) at L.A.N.A., PO Box 35288, Charlotte, N.C., 28235-5288, USA**, **e-mail: to:**mailto:lana@snonet.org

d) Contact the **Institute of Synergistic Arts and Sciences PO Box 1068 Springville, UT 84663 Phone: 801-491-9008**

e) Call my office, **ABC's of Nutrition and Wellness 516-409-6978** to learn how you can do simple lymphatic and breathing exercises in the privacy of your own home or e-mail me at **healthcoach9@gmail.com**.

I decided to split this report into two sections. The first section is called **Very Possible Solutions for Cancer** and the next section is **The Science** that supports the first section.

After doing extensive research I have decided to recommend the products mentioned in this next section, because of their potential benefits and the benefits that they have shown to me and to those who have used them. I believe in using products that work.

If you know of similar products that have worked for you or anyone else with similar benefits, by all means use them. I have included this section to show you that there are things that can be used in the fight against cancer. Also keep in mind that since we are all individuals, what may work for some, may not work for others and in many cases many of the results may be dose dependant.

Very Possible Solutions for Cancer

The following are my personal product preferences. What you are about to read could very possibly save your life. I am going to go over the basics that everyone should follow. **Anyone who has, had or even may be predisposed to cancer should, under the close supervision of his or her doctor, implement these**, **Very Possible Solutions for Cancer**.

Note: I am always searching for new products that offer the most benefit for the dollar. So, from time to time I may replace a new product with another or sometimes I just add a new product to this report to give you the option to make the decision for yourself. If you have one of my older reports and find that a product link does not work simply go to www.EnjoyAHealthyLife.com also known as www.abcsofhealth.com to learn about the products that I use.

You have to understand that the human body is a highly sophisticated piece of machinery. It has the ability to achieve and maintain optimum health. We, you and I, are the ones who prevent our bodies from working at its peak by what we put into our mouths, minds, and lungs. As far as the lungs are concerned, I am not talking about what is in the air that you have little to no control over. Unfortunately, inhaling these airborne toxins just forces you to maybe go the extra mile to ensure that you are taking products that can help in detoxifying your cells.

The products and companies mentioned can be found in the **Resources** section immediately following the **Very Possible Solutions for Cancer.**

There are countless products on the market today that can help you in your quest for optimum health, but I had to narrow it down to a select few. This should give you a good start in your research.

Another Note: In March of 2009 my mother was diagnosed with colon cancer and she and my brothers are old school and very intimidated by the medical profession. So, I had to find products that were not only potentially effective, but also easy to use and cost effective. My mother is currently taking two products: A coral calcium product, which she simply puts into her drinking water and another natural product from the same company.

Let's call it a coincidence. My mother was using the coral calcium sachets in her water for about three weeks before her surgery and she was taking the Ionyte on and off for about two weeks prior to her surgery.

Fact: Three Weeks prior to her surgery her CAT Scan was showing a mass of eight centimeters in size. Two weeks prior to her surgery a Barium enema was showing the mass to also be eight centimeters in size. After her surgery the surgical intern of the hospital told me that the pathology report was reporting that the mass removed from my mother was between seven and eight centimeters in size.

The Coral Calcium sachets contain:

Calcium Magnesium and over 70 different minerals and trace minerals in an ionic form (very useable by the body). It also helps to neutralize chlorine and provides you with clean fresh re-mineralized anti-oxidant water while raising the pH of your water.

Another Note: One of my cancer clients has been attempting to raise the pH level of her saliva for months and was not able to maintain a consistent 7.5 until she started using the coral calcium sachets.

Pure water is said to be neutral. The pH for pure water at 25 °C (77 °F) is close to 7.0. Solutions with a pH less than 7 are said to be acidic and solutions with a pH greater than 7 are said to be basic or alkaline. pH measurements are important in medicine, biology, chemistry, food science, environmental science, oceanography and many other applications. (Definition was obtained from www.wikipedia.com)

Your body does its best to maintain a pH of around 7.4 and when you drink acidic beverages like sodas and coffee and eat acidic foods like meats and chicken you need to balance out your diet with alkaline foods and beverages.

As you will find after reading this report, that I like products that have third party science to support the ingredients that are in the products. Well I found a product that contains Amalaki (Indian Gooseberry), Heritaki, Tulsi (Holy Basil), JuJube, Ginger, Schizandra, and Turmeric with enough scientific support to get me excited. You will learn some of the countless scientific articles that I found in "The Science" section of this report.

These Fruits and Herbs are a Synergistic blend of Ayurvedic, Chinese, and Traditional Superfoods. Together they provide nourishment like no other product on the market today.

There are basically 5 steps to obtaining and maintaining great health and they are:

1) Cleansing the blood and the cells
2) Detoxifying and removing toxins from your body
3) Neutralizing the toxins and acidity in the blood and at the cellular level
4) Provide Antioxidants to reduce free radicals
5) Restoring cellular health by providing nourishment to the cells.

Well according to what I have learned from listening to the formulators of this product it was formulated to help you address these 5 steps.

It is a **Preservative-free** formulation that takes nutrition to the next level. The formulation for the product is built on 5,000 years of Ayurvedic wisdom, which calls for multiple ingredients working together synergistically to maximize nutritional potency.

I don't know about you, but life is complicated enough so if I had a choice, I would prefer to drink one to three ounces of **this** product everyday rather than a handfull of pills or a not so pleasent tasting drink.

So, I present the following information for your review and research:

Now for the Basic Causes of Cancer:

1) Acidosis
2) Parasites
3) Elimination of oxygen in your cells
4) Excessive amounts of free radicals
5) Poor Nutrition
6) Congested colon

Very Possible Solutions for Cancer:

1) I found, in my opinion, a company whose products should be the front line of defense to every health program. Whether you have Cancer, Heart Disease, and Diabetes…or whatever health condition the medical profession is telling you that you have. You owe it to yourself to learn about the **HIGHLY AFFORDABLE PRODUCTS** from **Advanced Scientific Health (ASH)**. You can learn more by going to **http://www.ashnow.com/itisup2me** I make this statement, because as you will learn in the **Resource** section of this report and on their web site, their products help in raising the pH level in your body and this is very critical to achieving overall health.

Remember to keep your body as alkalized as possible by drinking alkaline beverages and eating alkalizing foods. If you like coffee I recommend the coffee by **Gano Excel**. They infuse the Ganoderma Mushroom into their coffee, which has shown to bring the pH level to that similar to the pH level of our blood, 7.3 to 7.5, and yes; it does still taste like coffee. The Gano Excel coffee also has over 165 different antioxidants as well as vitamins, minerals and amino acids.

a) To help me in keeping my body more alkaline than acidic I use the AquaLyte Coral Calcium S**achets**. **(see Resources for the link to this site)**

Each "tea-bag like" sachet contains 994 milligrams of coral minerals and will treat up to 2 quarts or liters.

When placed into drinking water or water-based beverages, the sachet will release beneficial ingredients like calcium, magnesium and other minerals in an ionic form for better absorption. All this while neutralizing most impurities, like chlorine and raising the pH level from an acidic to a more alkaline state.

Maintaining a proper, slightly alkaline pH level is considered the most important aspect of a healthy body. An imbalance of alkalinity creates a condition favorable to the growth of bacteria, viruses, yeasts and other harmful organisms. This combined with the accumulation of acid wastes are reported to be closely linked with degenerative disease, lack of vitality and aging in general.

One of the most impressive demonstrations is the power of the AquaLyte sachet is a simple OTO test that can be performed using a widely available do-it-yourself pool test kit.

AquaLyte sachets can neutralize the chlorine usually found in tap water. Untreated water will turn yellow after OTO drops are added, showing the chlorine content in the water. In just a few seconds after dropping a sachet into the water and stirring, the water will become clear, showing that the chlorine has been filtered through the sachet.

A good source to help you eat alkalizing foods is from **The Wholefood Farmacy** **www.MyWholefoodFarmacy.us.** They offer a wide variety of organic foods the whole family will love in easy and convenient packages.

Here is another very affordable and effective food source that you can use to alkalize your body, Sodium Bicarbonate. That's right, Baking Soda. If you were to read the box of the Arm & Hammer Baking Soda you will learn that it is recommended to take a 1/2 teaspoon in four ounces of water up to seven times per day.

To learn more on the importance of using sodium bicarbonate in your daily diet, go to your favorites search

engine and type in "sodium bicarbonate and cancer" or "baking soda and health". I have included some studies in the "Science" section of this report.

I have been finding studies that show that by raising the body's alkalinity it also raises the amount of oxygen that the blood carries.

From what I am finding sodium chloride (table salt) raises blood pressure, but sodium bicarbonate does not. Remember To Always Check With Your Healthcare Provider Before Adding Anything New To Your Diet.

To learn more about the use of sodium bicarbonate go to http://cancerfungus.com/. This is the site of Dr. Tullio Simoncini of Rome Italy. He has been able to accomplish amazing things with sodium bicarbonate in dealing with cancer.

NOTE: Go to your local pharmacy or health food store and by pH test strips. Alkalizing your body is very important, but you do not want to over do it. You could also go on the internet and search for alkalizing foods. There is a good book to buy by Dr. Theodore A. Baroody called Alkalize or Die.

2) Take an anti-parasite product that contains: Black Walnut hulls, Pau D'Arco bark, Valerian root extract and Colostrum. These ingredients not only help get rid of parasites they also help your body eliminate yeast, which can be building up in your colon. Tens of thousands of people go undiagnosed every year with yeast infections. The yeast competes with the healthy bacteria in your colon, which can weaken your immune system. Dr. Hulda Clark has had tremendous success with hundreds of patients who had cancer. Simply by eliminating the parasites and by teaching them and having them follow a healthy lifestyle program. You can learn more about the **Dr. Clark** programs\ by reading her book, **The Cure for All Cancers**. I recommend a product called **Para-Guard™** from **Food Science of Vermont**.

2 a) Another product that may be helpful in dealing with parasites is **Healose** from **NutriHarmony**.

2 b) The Amalaki (Indian Gooseberry), Haritaki, Tulsi (Holy Basil), Jujube, Schisandra, and Ginger found in the Zrii also have shown to have anti-parasitic capabilities.

2 c) For parasites you can also look into the **Model A** from **Wright Laboratories**.

3) Perform periodic deep breathing and lymphatic exercises on a rebounder (mini trampoline) as well as various localized lymphatic-stroking exercises daily. As stated earlier in this report, in 1966, Dr. Otto Warburg, who won the Nobel Peace Prize in 1931 and 1944, gave a lecture at the annual meeting of "Nobel Prize Winners" in Lineau, Germany. He stated that, **THE PRIME CAUSE OF CANCER WAS THE LACK OF OXYGEN. CANCER CELLS LIVE ON THE FERMENTATION OF GLUCOSE DUE TO THE LACK OF OXYGEN.** Lymphatic exercises help to remove trapped blood proteins and toxins that are preventing and/or minimizing the oxygen from getting to your cells.

4) Consume antioxidants. The following products either contain or provide antioxidant like benefits: **AquaLyte Coral Calcium sachets**, the **Ganoderma Lucidum and Excellium products** from **Gano Excel,** the **Zrii Juice from Zrii, LLC,** and **Farmacy Pro Power from The Wholefood Farmacy.** There are more, but you can decide for yourself when you visit the web sites in the **Resources** section.

5) Eat more raw (uncooked) **organic** green leafy vegetables and or take food concentrate supplements like **Garden and the Greens™, from NutriHarmony or Farmacy Pro Power from, The Wholefood Farmacy and Coconut Oil**. Besides chlorophyll, which helps clean the blood, foods also contain many antioxidants and the highly effective Spirulina as mentioned in the abstracts in this report. (My mother had breast cancer and her doctor at first recommended that she go on tamoxifen until I did some research and found out that it has only a 50 percent success rate and approx. 3 percent of women on tamoxifen develop uterine cancer. Coconut Oil provides medium-chain triglycerides, which studies have shown to be very beneficial for cardiovascular health.

The American Cancer Society is heading in the right direction. They recommend that you eat more fruits and vegetables and to take antioxidant supplements to help in your quest to reduce your risk of cancer. My

recommendation would be to eat more **RAW ORGANIC** fruits and vegetables, because organic produce is not grown with any pesticides, insecticides, or herbicides. We all know of the controversy of pesticides, insecticides, and herbicides. Organic produce is grown the way Mother Nature intended it to grow. It has not been genetically modified in a science lab. I guess organic farmers feel that they do not have to improve on Mother Nature.

Here is more evidence to prove that you should be eating organic foods. I read three different unrelated articles and put two and two together. Let's see if you develop the same opinion as I did.

Article number one: **Fruits, Vegetables Don't Prevent Breast Cancer**

Idea of nutrition stopping disease dealt another blow By Edward Edelson-HealthScout Reporter

TUESDAY, Feb. 13, (HealthScout) -- A diet rich in fruits and vegetables has no significant effect on the risk of breast cancer, says a study looking at data on more than 350,000 women.

Gee, I thought eating fruits and vegetables were good for you? Keep reading you'll be surprised as to what you learn next.

Article number two: **Pesticides Breeding Ground for Bacteria-Germs thrive in certain farm chemicals, study shows By Randy Dotinga-HealthScout Reporter -Thursday, Oct. 12, 2000 (HealthScout) -- Pesticides kill insects, but new research suggests they may be a breeding ground for different types of bugs -- bacteria**.

"Four out of 15 pesticides tested proved to be a very friendly environment for germs that cause human diseases," says Rick Holley, professor of food microbiology at the University of Manitoba in Canada.

..."I'd like to be able to say you could successfully wash away any problem bacteria, but the fact is that isn't 100 percent certain," Holley says, ...If a link is established, the findings could help explain why the incidents of illness carried by plants have doubled or tripled in the last decade, Holley says.
Gee, could it be that sickness and disease is rising because of the bacteria that may be coming from the fruits and vegetables that we eat? Keep reading and see if the next article will not scare the living non-organic produce out of you.

Article number three: **Highly pleomorphic staphylococci as a cause of cancer.-**Author: Wainwright **M-Source: Med Hypotheses; 54(1):91-4 2000 UI: 10790733**

"Abstract: **An extensive historical literature exists suggesting that bacteria and other non-virus microorganisms cause cancer…The literature linking highly pleomorphic bacteria with carcinogenesis is presented here in an attempt to add weight to the view that bacteria, notably those expressing the morphology of common species of staphylococci, cause cancer."**

Mesh Terms: Human Neoplasms/*microbiology Staphylococcus/classification/*isolation & purification-Language: ENG - Publication Type: JOURNAL ARTICLE -Title Abbreviation: Med Hypotheses -Year: 2000-Address: Department of Molecular Biology and Biotechnology, University of Sheffield, UK. Mail to:**mailto:M.Wainwright@Sheffield.ac.uk** Entry Month: 200006

O.K. people. Did you come up with the same conclusion as I did, which is: **You may be eating the nice looking fruit and/or vegetable that has the pesticide residue that may be harboring the bacteria that maybe infecting thousands of people with CANCER. Do ya think?**

DO YOUR BEST TO EAT ONLY ORGANIC FOODS!!!! A good way to start is by going to **www.MyWholeFoodFarmacy.us** they have many organic snacks you can introduce into your diet. You can learn more about The Whole Food Farmacy at the end of his report in the Resources Section.

Another important aspect in the war with cancer is to learn more about complementary medicine. More and more doctors are recognizing the important role that complementary medicine has to offer.

If you would like to learn more about complementary medicine, your local library and bookstores would have countless books on the subject. If you like, you could start with my book, ***"Learn the Simple Truth to Achieving Optimum Health".*** Don't take my word for it. Read what a highly respected breast cancer surgeon has to say:

"After reading Tom Ciraulo's book entitled, **"Learn the Simple Truth to Achieving Optimum Health"**, I felt I had acquired a lifetime of experience in a well presented, easy to understand manuscript. Tom is a renowned leader in the growing science of holistic medicine, and has enlightened his readers with enumerable everyday analogies, bringing the most salient points of achieving optimum health. This well researched, up to date book is a must for all. I highly recommend it for the experts and the novices interested in complementary medicine." **--Dwight De Risi, M.D. F.A.C.S., Surgical Oncologist, Specializing in Diseases of the Breast.**

Like Dr. De Risi said. **"This well researched, up to date book is a must for all. I highly recommend it for the experts and the novices interested in complementary medicine."** If you had, have or even feel that you maybe predisposed to a serious illness like cancer or any other debilitating/life threatening disease. Or you know others who fall into the above categories, you owe it to yourself and them to read **"Learn The Simple Truth To Achieving Optimum Health"** as well as learn as much as you can about complementary medicine. **You can learn more about my book at www.EnjoyAHealthyLife.com.**

5 a) Eat more **organic** (uncooked) fruits. I recommend **organic** because after reading the three different articles above I feel that the less pesticides that you consume the better you will be. I found that **The WholdFood Farmacy** has made eating organic whole foods simple, easy, and convenient. The Wholefood Farmacy has created a product, which you could basically call a One Stop Shopping Product, called **Farmacy Pro Power. The Farmacy Pro Power** contains your greens, and vegetables and enzymes and beneficial bacteria and more. It contains **8.5 billion pro-biotic cultures, Antioxidants equal to 8 to 10 servings of fruits and vegetables.** The product was formulated to help support your **Immune System, Digestive System, Enhance Energy**, help with **Cellular Repair,** and help you **Age Gracefully**.

6) Once again I am going to tell you to **STOP IMMEDIATELY ALL CONSUMPTION OF MILK, ICE CREAM, AND CHEESE**. That's right. Stop drinking milk and eating cheese, ice cream and yogurt. I know this may sound a little radical, but think about this for a moment. A mother cow is about 1,000 to 1,400 pounds and its main role is to feed its milk to its young to help it grow to over 1,000 pounds. Think about the powerful growth hormones that this milk contains. It has not been found that milk causes cancer, but the powerful growth hormones do help it to grow and grow and grow. Even organic milk has these powerful growth hormones.

Also found in non-organic milk are countless antibiotics, pesticides and artificial growth hormones.

Because of all of the infections a cow gets, you are drinking and/or eating millions of puss cells with every glass of milk or every piece of cheese that you consume. Did you know that it takes ten pounds of milk to equal one pound of cheese? So, the next time you eat that piece of cheese; just think of the concentration of growth hormones, antibiotics, pesticides, and pus that you are putting into your body.

Note on Dairy consumption. Recent studies are saying that milk may help with colon cancer. Actually, the studies are saying that it is the calcium that may be the beneficial ingredient. **Other studies are saying that milk consumption may promote ovarian and prostate cancers as well as other cancers.** My suggestion would be to get yourself a GOOD calcium supplement if you're concerned with the colon cancer and **STAY OFF OF ALL DAIRY PRODUCTS except for Whey. Based on what I have learned the Ion-Exchange Whey Protein from NutriHarmony offers a quality product. For those of you who would prefer to not to use whey, Food Science of Vermont has a great Soy based product called Soy Protein Powder.**

7) Make sure you start and maintain a good nutrition program that includes vitamins, antioxidants, major, trace and ultra trace minerals and anti-inflammatory nutrients. If you are taking a vitamin supplement now, does it have at least 65 trace minerals along with antioxidants and vitamins along with anti-inflammatory properties? If not, find one. The most efficient and effective way to consume a vitamin supplement is in a liquid form. It is the most absorbable by your body. Plus many people do not like to take pills all day.

8) Increase the amount of fiber that you put into your diet and take a colon cleanser like **Clearly Fiber™** from **Food Science of Vermont.** Many studies link an unhealthy colon to countless diseases. Fiber helps absorb many unwanted toxins as well as helps your body remove unwanted waste. When you go to your local health store or even the Internet you will see that there are many other high fiber and colon cleansing products on the market. Determine which one would be best for you and **USE IT**.

8 a) Take a digestive enzyme supplement to help your body minimize material that can congest your colon. In many cases this poorly digested material can reduce your body's ability to absorb nutrients as well as increase your chances of disease. Digestive enzymes help your body digest your food by replacing the natural enzymes (Protease for proteins, Amylase for carbohydrates Lipase for fats and Cellulase for plant fiber) found in raw foods that are destroyed by heat (cooking). I recommend **the Digestive Aids products from Food Science of Vermont.** They provide a variety of digestive aid products to suit your particular needs. You can also go to **http://www.goodhealth.nu/us/2281 to learn more about a very beneficial enzyme called** serrapeptase

8 b) Take a **Ganoderma Lucidum** supplement to help support your immune system. I use the Ganoderma Lucidum from **Gano Excel**. They have the largest organic Ganoderma Lucidum plantation in the world.

8 c) Take a supplement to help support the good bacteria in your colon like Fructo-oligosaccharides, a carbohydrate, which helps the healthy bugs (good bacteria), flourish. You should also take a good beneficial bacteria product, which can be found in any health food store or you can buy the **Mega Probiotic-ND™** from **Food Science of Vermont.**

9) Use filtered water even in the shower. Many municipalities put chlorine and fluoride in their water supply, plus depending on you plumbing system you water may be leaching lead from you pipes. To help avoid this you can buy a water filtration system like the **Countertop Water Processor** from a company called **Tidal Wave**. **Tidal Wave** also has a shower model called the **Deluxe Shower Head**. I recommend the Deluxe Shower Head, because your skin can absorb many of the impurities that are found in the water, which can add to ill health.

10) Take a supplement to help support the good bacteria in your colon like Fructo-oligosaccharides, a carbohydrate, which helps the healthy bugs (good bacteria), flourish. You should also take a good beneficial bacteria product, which can be found in any health food store. A good product would be **Mega-Probiotic-ND** by **Food Science of Vermont.**

There are a number of benefits that go along with these friendly bacteria:

- Vitamin production specifically B2, B5, B6, B12, folic acid, biotin and niacin.
- Decrease in lactose intolerance because of the enzyme lactase.
- Lower cholesterol levels
- Defense against food poisoning
- Inhibition of Candida, yeast and other fungal forms
- Healthier looking and feeling skin
- Better absorption of foods because of the enzymes they produce
- Increase in peristalsis (wave of contraction that moves food through the colon). Normalizes bowel movements.
- Increase in immunity through the secretion of acids and natural antibiotics.
- Help maintain a good hormonal balance.

11) Last, but not least, **Drink Plenty of Water. Not Just Any Water**. I am currently using the AquaLyte Coral Calcium sachets in my water that offers better hydration than your standard filtered or spring waters. To find my latest recommendations send an e-mail to **healthcoach9@gmail.com** and I will be happy to send you my latest updates.
I found the following article on serrapeptase at http://www.life-enthusiast.com/enzyme/serrapeptase.htm

Serrapeptase

In nature, serrapeptase is produced by a bacteria that lives in the intestinal tract of the silk worm. There is absolutely no way to extract enough serrapeptase from nature to support the world's consumption of serrapeptase. What is actually used to meet the global demand for serrapeptase is serratia peptidase. Serratia peptidase, is a proteolytic enzyme isolated from the non-pathogenic enterobacteria Serratia E15. This bacteria is cultured to produce, through batch fermentation, the necessary amount of serratia peptidase to meet global demand. At this point, however, the actual silk worm has nothing to do with the enzymes that make it into our (or anyone else's) products. And thus, silk worms are not harmed.

Additionally, after consumption, serratia peptidase is found in negligible amounts in the urine, suggesting that it is transported directly from the intestine into the bloodstream.(1, 2) Clinical studies show that serratia peptidase induces fibrinolytic, anti-inflammatory and anti-edemic (prevents swelling and fluid retention) activity in a number of tissues, and that its anti-inflammatory effects are superior to other proteolytic enzymes.(3) Besides reducing inflammation, one of Serratia Peptidase's most profound benefits is reduction of pain, due to its ability to block the release of pain-inducing amines from inflamed tissues.(4) Physicians throughout Europe and Asia have recognized the anti-inflammatory and pain-blocking benefits of this naturally occurring substance and are using it in treatment as an alternative to salicylates, ibuprofen, and other NSAIDs.(5)

Glossary Terms:

enterobacterium: any of a family (Enterobacteriaceae) of gram-negative straight rod bacteria (as a salmonella or a shigella) that ferment glucose and include saprophytes as well as some serious plant and animal pathogens.

pathogen: a specific causative agent (as a bacterium or virus) of disease

pathogenic: causing or capable of causing disease

References:

1. Moriya N, Nakata M, Nakamura M, Takaoka M, Iwasa S, Kato K, Kakinuma A. Intestinal absorption of serrapeptase (TSP) in rats. Biotechnol Appl Biochem. 1994; 20(Pt1):101-8.

2. Miyata, K. Intestinal absorption of Serratia Peptidase. J Appl Biochem. 1980;2:111-16.

3. Mazzone A, Catalani M, Costanzo M, Drusian A, Mandoli A, Russo S, Guarini E, Vesperini G. Evaluation of Serratia peptidase in acute or chronic inflammation of otorhinolaryngology pathology: a multicentre, double-blind, randomized trial versus placebo. J Int Med Res. 1990; 18(5):379-88.

4. Mazzone A, et al. Evaluation of Serratia peptidase in acute or chronic inflammation of otorhinolaryngology pathology: a multicentre, double-blind, randomized trial versus placebo. J Int Med Res. 1990; 18(5):379-88.

5. Aso T et al. Breast engorgement and its treatment: Clinical effects of Danzen an anti-inflammatory enzyme preparation. The world of Obstetrics and Gynecology (Japanese). 1981; 33:371-9.

Serrapeptase is an anti-inflammatory proteolytic (protein-dissolving) enzyme originally isolated from silkworms. Serrapeptase has been used in Europe to treat arterial blockages due to its ability to alleviate arterial inflammation and dissolve blood clots and arterial plaque. It has been used to treat arthritis, fibrocystic breast disease, carpal tunnel syndrome, and other inflammatory conditions. The ingredients in the Serrapeptase capsules are enterically coated for proper absorption.

Serrapeptase: Insect-Derived Enzyme Fights Inflammation

December 1999, *by Kimberly Pryor*

Our bodies have a love-hate relationship with inflammation. On the one hand, inflammation is a natural response, necessary to protect the body from invading organisms. On the other hand, inflammation can limit joint function, and destroy bone, cartilage and other articular structures.

An elusive goal of scientists and physicians has been to find a side-effect-free substance to reduce the pain and inflammation associated with fibrocystic breast disease, rheumatoid arthritis, idiopathic edema, carpal tunnel syndrome and post-operative swelling. It appears that the search may be nearing an end, thanks to an enzyme produced by the larval form of the silk moth.

Serrapeptase is an enzyme that is produced in the intestines of silk worms to break down cocoon walls. This enzyme is proving to be a superior alternative to the non-steroidal anti-inflammatory agents (NSAIDs) traditionally used to treat rheumatoid arthritis and osteoarthritis. Its uses have also been extended to the treatment of chronic sinusitis and postoperative inflammation, and some researchers believe the substance can play an important role in arterial plaque prevention and removal.

Harmful Effects of NSAIDs

NSAIDs, which include aspirin, ibuprofen, salicylates, and naproxen, are among the most commonly prescribed medications for inflammation resulting from rheumatoid arthritis, joint conditions, osteoarthritis, gouty arthritis, joint and muscle discomfort associated with systemic lupus erythematosus, and other musculoskeletal disorders.(1) In some cases, this overeliance on NSAIDs has proved deadly. Annually, 76,000 people are hospitalized from NSAID-induced gastrointestinal complications. The American Medical Association estimates that from 50-80 percent of those hospitalized for gastrointestinal bleeding are taking some form of NSAIDs. At this stage in the medication-induced bleeding, there is a ten percent chance of fatality.(2)

NSAIDs lethal effects result from the inhibition of the biosynthesis of prostaglandins. NSAIDs block cyclo-oxygenase, the enzyme responsible for catalyzing the reactions of arachidonic acid to endoperoxide compounds. This process results in the inhibition of gastric prostaglandin E, a hormone which protects the lining of the stomach from acid. After prolonged and frequent ingestion of NSAIDs, the stomach remains defenseless and at increased susceptibility to ulcers.(3-4) If an ulcer erodes into a blood vessel, bleeding results. An ulcer can destroy part of the stomach and duodenal walls, leaving a gap that requires immediate surgery.

In one study, 1,826 osteoarthritis or rheumatoid arthritis patients who had been taking NSAIDs for six months or more and who had been unable to tolerate continuous NSAID use because of adverse gastrointestinal symptoms were examined endoscopically for gastroduodenal lesions and ulcers. Clinically significant gastroduodenal lesions were found in 37.1 percent of the patients. Of those, 24 percent had ulcers. The prevalence of gastroduodenal ulcers increased with age, duration of osteoarthritis, and duration of current NSAID use. The authors of the study wrote: "These results provide further endoscopic confirmation of the association between NSAID use and gastroduodenal lesions and ulcers and support the contention that safer treatment alternatives to conventional NSAIDs are required."(5)

That advice is particularly wise in light of the other effects NSAIDs have on the gastrointestinal tract. In one group of 312 NSAID takers, 20 percent had levels of inflammation comparable to that previously reported in patients with inflammatory bowel disease.(6) Besides damaging the gastrointestinal tract, NSAIDs also interfere with and suppress bone repair and remodeling. One paper presented data obtained over a 12-year period, and outlined the effects of NSAIDs on the matrix synthesis and turnover in 650 arthritic and 180 non-arthritic human cartilages. The study showed that one category of NSAIDs that includes Naproxen, ibuprofen, indomethacin, and nimezulide significantly inhibited matrix synthesis and had toxic effects on cartilage metabolism.(7) Thus, it appears that the drugs many patients take to relieve their arthritic pains actually contributes to further destruction of their joints!

Additionally, NSAIDs have been shown to interfere with patients' sleep patterns. One study of 37 male and female subjects at the sleep laboratory at Bowling Green State University in Ohio demonstrated that aspirin and ibuprofen, in comparison to a placebo, increased the number of awakenings and the percentage of time spent awake. The drugs also decreased sleep efficiency, and delayed the onset of the deeper stages of sleep.(8)

Even insulin secretion is affected by NSAIDs. Neonatal rat pancreatic cells were examined partly to determine the effects of insulin secretion caused by prostaglandin E (PGE) and drugs that inhibit its synthesis—i.e. NSAIDs. Two NSAIDs, sodium salicylate (aspirin) and ibuprofen, at drug concentrations similar to those achieved therapeutically in humans, inhibited PGE synthesis up to 70-80 percent. Augmented insulin secretion accompanied the PGE inhibition. Both drugs shifted the glucose-insulin response curves to the left at low glucose concentrations and augmented maximal insulin release at high glucose concentrations.(9)

Other NSAID-induced side effects include kidney damage, blood dyscrasias and cardiovascular effects, complication of antihypertensive therapies involving diuretics or beta-adrenoceptor blockade, and adverse effects in patients with heart failure and cirrhosis.(10) In one instance, a woman treated for rheumatoid arthritis with the NSAID sulindac developed gallstones composed of sulindac metabolites.(11)

Interestingly, NSAIDs have also induced adverse psychiatric reactions. Five psychiatric outpatients—two with major depressive disorders, one with a bipolar disorder, one with a schizophrenic disorder and one with an anxiety disorder—were treated with NSAIDs due to rheumatoid arthritis, osteoarthritis, or other painful neuromuscular conditions. All five patients developed moderate to severe depression. Three patients became paranoid, and four either attempted or considered suicide. These psychiatric symptoms disappeared once the patients stopped taking NSAIDs. When the patients re-started the drugs, the symptoms returned.(12)

NSAIDs Roulette

Due to the detrimental effects of NSAIDs on the body, most physicians resort to a game of "NSAID musical-chairs," taking a patient off one NSAID as soon as side effects become evident or the drug stops working, then treating the patient with another of the 10 most widely prescribed propionic acid-derived NSAIDs.

To provide a more consistent form of treatment, researchers have long searched for a side-effect free anti-inflammatory agent. Researchers have recently focused on selective cyclo-oxygenase (COX-2) inhibitors, more precise versions of NSAIDs. Whereas previous NSAIDs reduced inflammation by inhibiting all cyclo-oxygenase activity, these new selective COX-2 inhibitors differentiate between the two forms of COX: COX-1 appears to regulate many normal physiologic functions and COX-2 mediates the inflammatory response. These selective inhibitors are believed to reduce inflammation without influencing normal physiologic functions by inhibiting only COX-2. By leaving COX-1 alone, the selective inhibitors result in fewer gastrointestinal side effects.

At first glance, these COX-2 inhibitors look like the solution to NSAID complications. Upon further inspection, however, celecoxib, a highly selective COX-2 inhibitor, can cause headaches, change in bowel habits, abdominal discomfort and dizziness in osteoarthritis patients. Fewer adverse effects are reported in rheumatoid arthritis patients, but because the drug is metabolized in the liver by cytochrome P-450 isozyme CYP2C9, serious drug interactions are possible. Fung and colleagues pointed out that more clinical studies are needed before the selective COX-2 inhibitors are put into widespread use.(13)

Another new drug, Enbrel, initially showed promise of treating the pain associated with rheumatoid arthritis. Currently, however, the FDA is advising physicians about safety concerns of the new drug. Thirty of the 25,000 patients treated with Enbrel since the drug's approval have developed serious infections, including sepsis. Several of those patients died as a result of the infections. Those at greatest risk when taking Enbrel appear to be patients with a history of chronic or recurrent infections, pre-existing infections, diabetes, or other conditions making them more susceptible to infection.(14)

The potentially lethal side effects associated with NSAIDs and other drugs indicate that a superior anti-inflammatory substance is needed.

A Natural Anti-Inflammatory

Serrapeptase, also known as Serratia peptidase, is a proteolytic enzyme isolated from the non-pathogenic enterobacteria Serratia E15. When consumed in unprotected tablets or capsules, the enzyme is destroyed by acid in the stomach. However, enterically-coated tablets enable the enzyme to pass through the stomach unchanged, and be absorbed in the intestine. Serrapeptase is found in negligible amounts in the urine, suggesting that it is transported directly from the intestine into the bloodstream.(15,16)

Clinical studies show that serrapeptase induces fibrinolytic, anti-inflammatory and anti-edemic (prevents swelling and fluid retention) activity in a number of tissues, and that its anti-inflammatory effects are superior to other proteolytic enzymes.(17)

Besides reducing inflammation, one of serrapeptase's most profound benefits is reduction of pain, due to its ability to block the release of pain-inducing amines from inflamed tissues.(18) Physicians throughout Europe and Asia have recognized the anti-inflammatory and pain-blocking benefits of this naturally occurring substance and are using it in treatment as an alternative to salicylates, ibuprofen and other NSAIDs.(19)

In Germany and other European countries, serrapeptase is a common treatment for inflammatory and traumatic swellings, and much of the research that exists on this substance is of European origin. One double-blind study was conducted by German researchers to determine the effect of serrapeptase on post-operative swelling and pain. This study involved sixty-six patients who were treated surgically for fresh rupture of the lateral collateral ligament of the knee. On the third post-operative day, the group receiving serrapeptase exhibited a 50 percent reduction of swelling, compared to the controls. The patients receiving serrapeptase also became more rapidly pain-free than the controls, and by the tenth day, the pain had disappeared completely.(20)

Cystic Breast Disease

Serrapeptase has also been used in the successful treatment of fibrocystic breast disease. In a double-blind study, 70 patients complaining of breast engorgement randomly were divided into a treatment group and a placebo group. Serrapeptase was superior to the placebo for improvement of breast pain, breast swelling and induration (firmness). 85.7 percent of the patients receiving serrapeptase reported moderate to marked improvement. No adverse reactions to serrapeptase were reported and the researchers concluded that "serrapeptase is a safe and effective method for the treatment of breast engorgement."(21,22)

Serrapeptase and Sinusitis

Due to its inflammatory properties, serrapeptase has been shown in clinical studies to benefit chronic sinusitis sufferers. In this condition, the mucus in patients' nasal cavities is thickened and hypersecreted. This thickening causes mucus to be expelled less frequently. Japanese researchers evaluated the effects of serratiopeptidase (30 mg/day orally for four weeks) on the elasticity and viscosity of the nasal mucus in adult patients with chronic sinusitis. Serratiopeptidase reduced the viscosity of the mucus, improving the elimination of bronchopulmonary secretions.(23)

Other clinical trials support serrapeptase's ability to relieve the problems associated with chronic sinusitis. In one study, 140 patients with acute or chronic ear, nose and throat pathologies were evaluated with either a placebo or the active serratia peptidase. Patients taking the serrapeptase experienced a significant reduction in severity of pain, amount of secretion, purulence of secretions, difficulty in swallowing, nasal dysphonia, nasal obstruction, anosmia, and body temperature after three to four days and at the end of treatment. Patients suffering from laryngitis, catarrhal rhinopharyngitis and sinusitis who were treated with serrapeptase experienced a significant and rapid improvement of symptoms after 3-4 days. Physicians assessed efficacy of treatment as excellent or good for 97.3 percent of patients treated with serrapeptase compared with only 21.9 percent of those treated with a placebo.(24)

Respiratory diseases are characterized by increased production of a more dense mucus modified in viscosity and elasticity. Traditionally, in respiratory diseases, muco-active drugs are prescribed to reestablish the physicochemical characteristics of the mucus in order to restore respiratory function. Some of these drugs, however, cause a functional depletion of mucus, whereas Serrapeptase alters the elasticity of mucus without depleting it.(25,27)

A powerful agent by itself, serrapeptase teamed with antibiotics delivers increased concentrations of the antimicrobial agent to the site of the infection. Bacteria often endure a process called biofilm formation, which results in resistance to antimicrobial agents. In an attempt to prevent this bacterial immunity, researchers have experimented with various means of inhibiting biofilm-embedded bacteria. Their search may have ended with serrapeptase. One study conducted by Italian researchers suggests that proteolytic enzymes could significantly enhance the activities of antibiotics against biofilms. Antibiotic susceptibility tests showed that serratiopeptidase greatly enhances the activity of the antibiotic, ofloxacin, and that it can inhibit biofilm formation.(28)

Another double-blind randomized study evaluated the effects of administering the antibiotic cephalexin in conjunction with serrapeptase or a placebo to 93 patients suffering from either perennial rhinitis, chronic rhinitis with sinusitis or chronic relapsing bronchitis. The serratia peptidase treated group experienced significant improvement in rhinorrhea, nasal stuffiness, coryza and improvement of the para-nasal sinus shadows.(29)

Researchers witnessed equally impressive results in the treatment of infections in lung cancer patients undergoing thoracotomy. Serrapeptase and cefotiam, an antibiotic with a broad spectrum of activity against both Gram-positive and Gram-negative microorganisms, were administered to 35 thoracotomy patients with lung cancer. The patients were divided into two groups. A single dose of cefotiam was administered to the 17 subjects in Group I. The 18 subjects in Group II received a combination of Cefotiam and serrapeptase. The level of the antibiotic in the tissues versus the blood was significantly higher in the serrapeptase group than the single dose group.(30)

Cardiovascular Implications

Hans A. Nieper, M.D., an internist from Hannover, Germany, studied the effects of serrapeptase on plaque accumulations in the arteries. The formation of plaque involves deposits of fatty substances, cholesterol, cellular waste products, calcium and fibrin (a clotting material in the blood) on the inner lining of the arteries. Excessive plaque results in partial or complete blockage of the blood's flow through an artery, resulting in arteriosclerosis, or hardening of the arteries, and an ensuing stroke or heart attack. The evidence to support serrapeptase's role in preventing plaque build-up is anecdotal. Still, further studies are called for in this area as Nieper's research indicated that the protein-dissolving action of serrapeptase will gradually break down atherosclerotic plaques.(31)

Conclusion

Regardless of whether serrapeptase is used for inflammatory diseases or to prevent plaque build up on the arteries, it is well-tolerated. Due to its lack of side effects and anti-inflammatory capabilities, serrapeptase is a logical choice to replace harmful NSAIDs. Thanks to the tiny larvae of the silk moth, researchers have taken a large step toward finding relief for inflammatory disease sufferers.

References

1. Raskin JB. Gastrointestinal effects of nonsteroidal anti-inflammatory therapy. Am J Med. 1999; 106 (5B):3S-12S.

2. No author listed. Regular Use of Pain Relievers Can Have Dangerous Results. Kaleidoscope Interactive News, American Medical Association media briefing. July 24, 1997.

3. Fung HB, Kirschenbaum, HL. Selective cyclooxygenase-2 inhibitors for the treatment of arthritis. Clin Ther. 1999; 21(7):1131-57.

4. Geis GS. Update on clinical developments with celecoxib, a new specific COX-2 inhibitor: what can we expect? Scand J Rheumatol Suppl. 1999; 109:31-7.

5. Cheatum DE, Arvanitakis C, Gumpel M, Stead H, Geis GS. An endoscopic study of gastroduodenal lesions induced by nonsteroidal anti-inflammatory drugs. Clin Ther. 1999; 21(6):992-1003.

6. Tibble JA, Sigthorsson G, Foster R, Scott D, Fagerhol MK, Roseth A, Bjarnason I. High prevalence of NSAID enteropathy as shown by a simple faecal test. Gut. 1999; 45(3):362-6.

7. Dingle JT. The effects of NSAID on the matrix of human articular cartilages. Z Rheumatol. 1999; 58(3):125-9.

8. Murphy PJ, Badia P, Myers BL, Boecker MR, Wright KP Jr. Nonsteroidal anti-inflammatory drugs affect normal sleep patterns in humans. Physiol Behav. 1994; 55(6):1063-6.

9. Metz SA, Robertson RP, Fujimoto WY. Inhibition of prostaglandin E synthesis augments glucose-induced insulin secretion in cultured pancreas. Diabetes. 1981; 30(7):551-7.

10. Raskin JB. Gastrointestinal effects of nonsteroidal anti-inflammatory therapy. Am J Med. 1999; 106 (5B):3S-12S.

11. Tokumine F, Sunagawa T, Shiohira Y, Nakamoto T, Miyazato F, Muto Y. Drug-associated cholelithiasis: a case of sulindac stone formation and the incorporation of sulindac metabolites into the gallstones. Am J Gastroenterol. 1999;94(8):2285-8.

12. Jiang HK, Chang DM. Non-steroidal anti-inflammatory drugs with adverse psychiatric reactions: five case reports. Clin Rheumatol. 1999;18(4):339-45.

13. Fung HB, Kirschenbaum, HL. Selective cyclooxygenase-2 inhibitors for the treatment of arthritis. Clin Ther. 1999; 21(7):1131-57.

14. FDA MedWatch: The FDA Medical Products Reporting Program. May 12, 1999. FDA Talk Paper.

15. Moriya N, Nakata M, Nakamura M, Takaoka M, Iwasa S, Kato K, Kakinuma A. Intestinal absorption of serrapeptase (TSP) in rats. Biotechnol Appl Biochem. 1994; 20(Pt1):101-8.

16. Miyata, K. Intestinal absorption of Serratia Peptidase. J Appl Biochem. 1980;2:111-16.

17. Mazzone A, Catalani M, Costanzo M, Drusian A, Mandoli A, Russo S, Guarini E, Vesperini G. Evaluation of Serratia peptidase in acute or chronic inflammation of otorhinolaryngology pathology: a multicentre, double-blind, randomized trial versus placebo. J Int Med Res. 1990; 18(5):379-88.

18. Mazzone A, et al. Evaluation of Serratia peptidase in acute or chronic inflammation of otorhinolaryngology pathology: a multicentre, double-blind, randomized trial versus placebo. J Int Med Res. 1990; 18(5):379-88.

19. Aso T et al. Breast engorgement and its treatment: Clinical effects of Danzen an anti-inflammatory enzyme preparation. The world of Obstetrics and Gynecology (Japanese). 1981; 33:371-9.

20. Esch PM, Gerngross H, Fabian A. Reduction of postoperative swelling. Objective measurement of swelling of the upper ankle joint in treatment with serrapeptase-a prospective study (German). Fortschr Med. 1989;107(4):67-8, 71-2.

21. Kee WH, Tan SL, Lee V, Salmon YM. The treatment of breast engorgement with Serrapeptase (Danzen): a randomized double-blind controlled trial. Singapore Med J. 1989;30(1):48-54.

22. Aso T et al. Breast engorgement and its treatment: Clinical effects of Danzen an anti-inflammatory enzyme preparation. The world of Obstetrics and Gynecology (Japanese). 1981; 33:371-9.

23. Majima Y, Inagaki M, Hirata K, Takeuchi K, Morishita A, Sakakura Y. The effect of an orally administered proteolytic enzyme on the elasticity and viscosity of nasal mucus. Arch Otorhinolaryngol. 1988;244(6):355-9.

24. Mazzone A, et al. Evaluation of Serratia peptidase in acute or chronic inflammation of otorhinolaryngology pathology: a multicentre, double-blind, randomized trial versus placebo. J Int Med Res. 1990; 18(5):379-88.

25. Tomoda K, and Miyatam K. Some information on the composition of trachael secretions before and after the administration of Danzen. Exper Ther. 1972; 477:9-16.

26. Kase Y, et al. A new method for evaluating mucolytic expectorant activity and its application to two proteolytic enzymes, serratiopeptidase and seaprose. Arznelrnitteltorachung. 1982; 32:374-378.

27. Marriott C. Modification in the rheological properties of mucus by drugs. Adv Exp Med Biol. 1982; 144:75-84.

28. Selan L, Berlutti F, Passariello C, Comodi-Ballanti MR, Thaller MC. Proteolytic enzymes: a new treatment strategy for prosthetic infections? Antimicrob Agents Chemother. 1993; 37(12):2618-21.

29. Perna L. Osservazioni Cliniche sui traitamento in osppio cieco con Serratio peptidasi nella neifre perenna naila ninite cronica nacutizzata con sinusopattia, nella bronchia cronica nacutizzata. Rlv Pat Clin Tuberc Penumol. 1985; 56:509-516.

30. Koyama A, Mori J, Tokuda H, Waku M, Anno H, Katayama T, Murakami K, Komatsu H, Hirata M, Arai T, et al. Augmentation by serrapeptase of tissue permeation by cefotiam (Japanese). Jpn J Antibiot. 1986; 39(3):761-71.

31. Brewer Science Library website. 1999.

Resources:

There are countless companies with countless products that can help you in your quest to maintain optimum health. To help you in your search I put together a list of companies and their products that I feel would help you in your quest for optimum health.

There are many great products on the market today, but for me, I prefer not to drink a liquid nutritional beverage that has preservatives. There are two preservatives in particular that are in most liquid drinks, as a matter of fact they are even in solid foods, and they are **Potasium Sorbate and Sodium Benzoate**.

I found out that Potassium Sorbate does not have any side effects at the levels found in most foods, but Potassium Sorbate is found in so many types of foods. So my question is, **"If you consume many of these foods in a given day. Would the Potasium Sorbate then become toxic???" Who knows???**

Here is disturbing information I found about **Sodium Benzoate** from www.wikipedia.com:

Safety and health

In combination with ascorbic acid (vitamin C, E300), **sodium benzoate and potassium benzoate may form benzene[7], a known carcinogen**. Heat, light, and shelf life can affect the rate at which benzene is formed.

Professor Peter Piper of the University of Sheffield claims that sodium benzoate by itself can damage and inactivate vital parts of DNA in a cell's mitochondria. Mitochondria consume oxygen to generate ATP, the body's energy currency. If they are damaged due to disease, the cell malfunctions and may enter apoptosis. There are many illnesses now tied to DNA damage, including Parkinson's and other neurodegenerative diseases, but above all, the aging process in general.[8][9][10][11][12]

Hyperactivity

Research published in 2007 for the **UK's Food Standards Agency** suggests that sodium benzoate (E211) is linked to hyperactive behaviour and decreased intelligence in children. According to the report, a high consumption of sodium benzoate is associated with a reduction in IQ of close to 5.5 points. [13] On 6 September 2007, the British Food Standards Agency issued revised advice on certain artificial food additives, including sodium benzoate (E211)[14][15][16].

Professor Jim Stevenson from Southampton University, and author of the report, said: "This has been a major study investigating an important area of research. The results suggest that consumption of certain mixtures of artificial food colours and sodium benzoate preservative are associated with increases in hyperactive behaviour in children…

Other health effects

People who suffer from asthma, or who have recurrent urticaria, may be sensitive to sodium benzoate and have allergic reactions. Sodium benzoate and tartrazine (E102) exacerbate the condition in between 10 and 40 percent of patients with chronic urticaria, and possibly a higher proportion still of aspirin sensitive individuals.[20]

References

7. FDA, 2006. "Data on Benzene in Soft Drinks and Other Beverages, " United States Food and Drug Administration. Accessed June 2nd at: http://www.cfsan.fda.gov/~dms/benzdata.html
8. Martin Hickman Caution: Some soft drinks may seriously harm your health *The Independent on Sunday* 27 May 2007
9. Martin Hickman E211 Revealed: Evidence highlights new fear over drinks additive *The Independent on Sunday* 27 May 2007
10. Leading article: Children deserve our doubts *The Independent on Sunday* 27 May 2007

11. Chris Mercer Fresh health fears hit benzoate in soft drinks BeverageDaily 29 May 2007
12. Piper PW Yeast superoxide dismutase mutants reveal a pro-oxidant action of weak organic acid food preservatives *Free Radic Biol Med* 1999 Dec;27(11-12):1219-27
13. Graham Tibbetts. "Artificial colourings as harmful as leaded petrol for children", *Telegraph*, 7 April 2008
14. Food Standards Agency issues revised advice on certain artificial colours 6 September 2007
15. Food Colorings and Hyperactivity "Myomancy" 7 September 2007
16. Agency revises advice on certain artificial colours Food Standards Agency 11 September 2007
17. BBC Europe-wide food colour ban call 10 April 2008
18. FSA Board discusses colours advice 10 April 2008
19. The Daily Mail [http://www.dailymail.co.uk/news/article-1021820/Diet-Coke-drop-additive-DNA-damage-fear.html DNA Damage Fear] 24 May 2008
20. E for additives, Maurice Hanssen with Jill Marsden, Thorsons 1987

This is the reason I prefer to consume products without Preservatives.

Anyway, here is a listing of companies for your review**. If you feel that there are too many choices to choose from, you can contact me at 516-409-6978 or healthcoach9@gmail.com and together we can decide which product or products would be best for you.**

www.PubMed.gov: This site is to provide you with the science that is available to educate you and your health care professional.

www.EnjoyAHelthyLife.com: If you have any questions or concerns regarding the information in this report

www.JustDrinkYourWater.com: To learn more about the importance of alkalinity, water, and Trace Minerals.

http://www.ashnow.com/itisup2me: Regardless of your current health condition, I truly feel that the products offered by **Advanced Scientific Health** should be the foundation to your health program.

Products to look for on this site:

- **MoRE**-This product has the ingredients that can help build and maintain one of the body's primary buildings blocks, collagen. It is crtical that our bodies have an ample supply of collagen.
- **Master Formula II-**Otto Warburg won a Nobel Prize for showing that cancer thrives in anaerobic (without oxygen), or acidic, conditions. Research by Keith Brewer, PhD and H.E. Satori has shown that raising the pH, or oxygen content, range of a cell to pH 8.0 creates a deadly environment for cancer. The pH scale ranges from 0 to 14, with numbers below 7 representing an acidic condition and above 7 representing an alkaline, or oxygenated, condition. When Master Formula II is taken up by cancer cells, it raises the pH, or oxygen content, of the cell. The cells that die are absorbed and eliminated by the body. There is a solution and it's a Nobel Prize Winning proven formula - Master Formula II. Master Formula II is designed to raise the pH level, increase the oxygen supply in the body, kill existing cancer cells, and prevent new cancer cells from forming.
- **Raanow**-People with high pH (oxygen), high GH, and high ascorbate live longer, stronger, slimmer, healthier lives. Learn these basics and age reversal will become obvious to you.
- **Cesium**-Cesium has been used to raise the pH of the body as an alternative cancer treatment of therapy for breast cancer, lung cancer, prostate cancer, colon cancer, pancreatic cancer, liver cancer, skin cancer, ovarian cancer, stomach cancer, cervical cancer, brain cancer, kidney cancer, testicular cancer, bone cancer, throat cancer, thyroid cancer, gastrointestinal cancer, cancers of the bladder and gallbladder, metastatic melanoma, and cancers in animals including feline, canine, and equine cancer.

Food Science of Vermont provides an array of nutritional supplements to help you address your particular needs. As you can see from the categories below: **To purchase Food Science of Vermont products go to www.EnjoyAHealthyLife.com and click on the FoodScience of Vermont link, or either call 516-409-6978 or send and e-mail to healthcoavh9@gmail.com.**

A, B, C, Bioflavonoids & E
Amino Acids
Antioxidants
Anxiety, Stress and Sleep Support
Books
Children Formulas
Coenzyme Q10
Digestive Aids
DMG Family of Products
Essential Fatty Acids
Herbal Extracts
Immune Support
Joint and Muscle Support
Men's, Women's and Specialty Products
Minerals
Multiple Vitamin/Mineral Formulas
Weight Management Products

Actually, I like their whole line of products. So, when you go to their web site you could decide for yourself the products that would be best for you and your family.

www.NutriHarmony.com/healthy1: If you are looking for whole-food supplements where the vitamins and minerals are made from plants and not in a lab, this is the site for you.

Products to look for on this site:

- **MRP (Meal Replacement Product)**
- **Garden and the Greens™**
- **Colonique™**
- **Healose™**
- **Real Food™ Multi-Vitamin/Multi-Mineral**
- **Ion-Exchange Whey Protein**
- **Enzymes**
- **Elemental Fiber™**

www.MyWholeFoodFarmacy.us: The first thing you should do when you get to this web site is to scroll down and watch the 22-minute video, especially if you are concerned about cancer. **The Wholefood Farmacy** is a must for those looking to eat foods that can enhance their health. You owe it to yourself and loved ones to learn and experience the foods offered on this web site.

Products to look for on this site:

- **Wholefoods**
- **Wholefood Beverages**
- **Stardust 2 Hydr8- Stardust 2 Hydr8** offers you all of the benefits of a saline or electrolyte solution without any needles or artificial additives. It is made from pure salt and bicarbonate which are the main components of extracellular fluid. Stardust 2 Hydra8 is mixed one tea spoon to a gallon of water creating a mildly saline solution - also referred to as an "electrolyte solution". Stardust 2 Hydr8 is a great way to help your body to operate at peak performance and to maintain proper hydration.
- **Farmacy Pro Power**
- **Soups**
- **Bath & Beauty**
- **Salt, Spices and Food Oils**

www.jwlabs.com: As I mentioned earlier Wright Laboratories offers the Royal R. Rife technology with their Model A. To contact Wright Laboratories call their Custom Service Department: **1-888-891-1122.**

Water!!!

There is water and there is water. I searched countless water products and water enhancing products and many water enhancers cost thousands of dollars. Well, I found a product that I truly believe in. Two critical elements to being healthy are to alkalize your body and to consume minerals and trace minerals. Well, I found an inexpensive product that than help with both.

The product is called **AquaLyte Coral Calcium**. It comes in little sachets that you drop into a quart or two of water. I was very impressed with the product very soon after using it.

Water is a necessity of life; why not drink water that could do the most good for you, your family, and your pets. It makes sense, doesn't it??? I think so, and that is why my family and I are using the **Aqualyte Coral Calcium** sachets.

I kept searching for a simple and easy way to bring up the alkalinity level in our bodies while providing essential minerals and trace minerals, and I believe I found it. From what I have learned; I feel it will help you and your family and anyone else who drinks water. The treated water would be good for every member of your family even your pets.

To learn more about the **AquaLyte Coral Calcium** sachets go to **www.JustDrinkYourWater.com**.

One night I was speaking with Fred Kaufman. Fred is the one who originally brought the original coral calcium into this country 14 years ago. From what he told me about the properties of the water after it is treated with the coral calcium sachets it will amaze you. I don't know if it is a coincidence or not, but something amazing happened to my daughter a few hours after drinking the treated water.

My daughter had white spots on her throat and the antibiotics she was taking were not helping. The doctor said that it might be a virus that was causing those white spots on her throat. She drank the **AquaLyte** treated water throughout the day and later that night she noticed that the spots were gone. In retrospect, I believe that the white spots might have been caused by a fungal infection after I visited the web site of Dr. Tullio Simoncini **http://cancerfungus.com/**. Dr. Simoncine believes that cancer is a fungus and he has been successfully treating it with sodium bicarbonate.

I came home the one night with aches and pains after a day of heavy lifting and I felt great the next morning. No aches and pains like I was expecting.

In the mean time if you have any questions send me an e-mail at **healthcoach9@gmail.com.**

I want to tell you about a great organization, **Environmental Working Group**. You've probably heard of their work - they're the ones responsible for getting a carcinogen removed from Teflon, spreading the word about how safe sunscreens really are, and creating an extensive cosmetics safety database.

You can join their email list at **www.ewg.org/signup** or check out their website at **www.ewg.org**.

They have a lot of great resources like:

- **Skin Deep, http://www.cosmeticsdatabase.com/** their cosmetics safety database
- **Food News**, **http://www.foodnews.org/** the insider's guide to which produce has the most pesticides
- **EWG's Fish List**, **http://www.ewg.org/safefishlist** which outlines which fish are the safest and how often you should eat them
- **10 Everyday Pollution Solutions**, **http://www.ewg.org/solutions** a list of small steps you can take to make a big difference
- **Parent's Buying Guide** http://www.cosmeticsdatabase.com/special/parentsguide/ to safe personal care products for kids

Join their email list to receive buying guides for safe children's products; info on cosmetics, sunscreens, and products to avoid; tips for making your food and water safer, and answers to your questions about keeping your family healthy. Sign up today at **www.ewg.org/signup.**

Environmental Working Group is a non-profit, non-partisan research organization dedicated to using the power of information to protect human health and the environment.

Here are some links that you may find interesting and as you can see after visiting these links that therea are countless was someone can work towards defeating cancer. I am providing these links for educational purposes only.

If you have any questions about the information in this report you can contact me at **(516) 409-6978** or by sending me e-mail at **healthcoach9@gmail.com**.

The links:

http://www.mnwelldir.org/docs/nutrition/sugar.htm
http://www.mnwelldir.org/docs/nutrition/diet.htm
http://www.mnwelldir.org/docs/cancer1/altthrpy.htm
http://www.mnwelldir.org/docs/cancer1/altthrpy2.htm
http://www.cancertutor.com doctor and Nobelist's quotes on cancer
http://www.cancer-coverup.com/brewer/default.html learn about "cesium" & Dr.. Keith Brewer

Here is another site where you can learn more about keeping your life as natural as possible,
http://www.neighborhood-network.org/

Articles

In this section I have decided to include various articles to help you in your quest for optimum health.

Here is an article that I had to include in this report It was found at the Hallelujah Acres web site http://www.hacres.com/home/home.asp

Tony Snow - Was It Cancer Or Chemotherapy That Killed Him?

by Andreas Moritz, author of the book *Cancer is not a Disease - It's a Survival Mechanism*

Former White House press secretary Tony Snow died in July 2008 at the age of 53, following a series of chemotherapy treatments for colon cancer. In 2005, Snow had his *colon removed* and underwent *6 months of chemotherapy* after being diagnosed with colon cancer. **Two years later (2007), Snow underwent surgery to remove a growth in his abdominal area, near the site of the original cancer.** "This is a very treatable condition," said Dr. Allyson Ocean, a gastrointestinal oncologist at Weill Cornell Medical College. "Many patients, because of the therapies we have, are able to work and live full lives with quality while they're being treated. *Anyone who looks at this as a death sentence is wrong"* **But of course we now know, Dr. Ocean was dead wrong!** The media headlines proclaimed Snow died from colon cancer, although they knew *he didn't have a colon anymore.* Apparently the malignant cancer had "returned" (from where?) and "spread" to the liver and elsewhere in his body. In actual fact, *the colon surgery severely restricted his normal eliminative functions, thereby overburdening the liver and tissue fluids with toxic waste.*

The previous series of chemo-treatments inflamed and irreversibly damaged a large number of cells in his body, and also impaired his immune system – a perfect recipe for growing new cancers. Now unable to heal the causes of the original cancer (in addition to the newly created ones), Snow's body developed new cancers in the liver and other parts of the body.

The mainstream media, of course, still insist Snow died from colon cancer, thus perpetuating the myth that it is only the cancer that kills people, not the treatment. Nobody seems to raise the important point that *it is extremely difficult for a cancer patient to actually heal from this condition while being subjected to the systemic poisons of chemotherapy and deadly radiation.*

Before Tony Snow began his chemo-treatments for his second colon cancer, he still looked healthy and strong. But after a few weeks into his treatment, *he started to develop a coarse voice, looked frail, turned gray and lost his*

hair. Did the cancer do all this to him? Certainly not! Cancer doesn't do such a thing, but chemical poisoning does.

Do the mainstream media ever report about the overwhelming scientific evidence that shows chemotherapy has zero benefits in the 5 year survival rate of colon cancer patients? *Or how many oncologists stand up for their cancer patients and protect them against chemotherapy treatment which they very well know can cause them to die far more quickly than if they received no treatment at all?*

Can you trustingly place your life into their hands when you know that most of them would not even consider chemotherapy for themselves if they were diagnosed with cancer? What do they know that you don't? *The news is spreading fast that in the United States physician-caused fatalities now exceed 750,000 each year. Perhaps, many doctors no longer trust in what they practice for good reasons.*

"MOST CANCER PATIENTS IN THIS COUNTRY DIE OF CHEMOTHERAPY. . . *Chemotherapy does not eliminate breast, colon or lung cancers. This fact has been documented for over a decade. Yet doctors still use chemotherapy for these tumors. . .* ***women with breast cancer are likely to die faster with chemo than without it."*** **–Alan Leven M.D.**

An investigation by the Department of Radiation Oncology, Northern Sydney Cancer Centre, Australia, into the contribution of chemotherapy to 5 year survival in 22 major adult malignancies, showed startling results: The overall contribution of curative and adjuvant cytotoxic chemotherapy to 5 year survival in adults was estimated to be 2.3% in Australia and 2.1% in the USA." [Royal North Shore Hospital Clin Oncol (R Coll Radiol) 2005 Jun:17(4):294.]

By comparison, a mere 2.3% contribution of chemotherapy to cancer survival does not justify the massive expense involved and the tremendous suffering patients experience because of severe, toxic side effects resulting from this treatment. *With a meager success rate of 2.3%, selling chemotherapy as a medical treatment (instead of a scam), is one of the greatest fraudulent acts ever committed.*

The average chemotherapy earns the medical establishment a whopping $300,000 to $1,000,000 each year, and has so far earned those who promote this pseudo-medication (poison) over 1 trillion dollars. *It's no surprise that the medical establishment tries to keep this scam alive for as long as possible.*
In 1990, the highly respected German epidemiologist, Dr. Ulrich Abel from theTumor Clinic of the University of Heidelberg, conducted the most comprehensive investigation of every major clinic study on chemotherapy drugs ever done. Abel contacted 350 medical centers and asked them to send him anything they had ever published on chemotherapy. He also reviewed and analyzed thousands of scientific articles published in the most prestigious medical journals. It took Abel several years to collect and evaluate the data.

Abel's epidemiological study, which was published on August 10, 1991 in The Lancet, should have alerted every doctor and cancer patient about the risks of one of the most common treatments used for cancer and other diseases. In his paper, Abel came to the conclusion that the overall success rate of chemotherapy was "appalling." *According to this report, there was no scientific evidence available in any existing study to show that chemotherapy can "extend in any appreciable way the lives of patients suffering from the most common organic cancers."*

Abel points out that chemotherapy rarely improves the quality of life. He describes chemotherapy as "a scientific wasteland" and states that even though there is no scientific evidence that chemotherapy works, neither doctor nor patient is willing to give up on it. The mainstream media has never reported on this hugely important study, which is hardly surprising, given the enormous vested interests of the groups that sponsor the media, that is, the pharmaceutical companies. A recent search turned up exactly zero reviews of Abel's work in the American journals, even though it was published in 1990. *I believe this is not because his work was unimportant—but because it is irrefutable.*

The truth of the matter would be far too costly for the pharmaceutical industry to bear, thus making it unacceptable. *If the mass media reported the truth that medical drugs, including chemotherapy drugs, are used to practically commit genocide in the U.S. and the world, their best sponsors (the pharmaceutical companies) would have to withdraw their misleading advertisements from television media, radio stations, magazines and newspapers.*

Many doctors go as far as prescribing chemotherapy drugs to patients for malignancies that are far too advanced for surgery, with the full knowledge that there are no benefits at all. Yet they claim chemotherapy to be an effective cancer treatment, and their unsuspecting patients believe that "effective" equals "cure." The doctors, of course, refer to the FDA's definition of an "effective" drug, one which achieves a 50% or more reduction in tumor size for 28 days.

Temporary tumor shrinkage through chemotherapy has never been shown to cure cancer or to extend life. *In other words, you can live with an untreated tumor for just as long as you would with one that has been shrunken or been eliminated by chemotherapy (or radiation).*

CHEMOTHERAPY HAS NEVER BEEN SHOWN TO HAVE CURATIVE EFFECTS FOR CANCER. *By contrast, the body can still cure itself, which it actually tries to do by developing cancer.* The "disease" is the body's attempt to cure itself of an existing imbalance. And sometimes, this healing response continues even if a person is subjected to chemotherapy (and/or radiation). ***Unfortunately, as the previously mentioned research has demonstrated, the chances for a real cure are greatly reduced when patients are treated with chemotherapy drugs.*** **The side effects of the treatment can be horrendous and heartbreaking for both patients and their loved ones, all in the name of trustworthy medical treatment.** *Although the drug treatment comes with the promise to improve the patient's quality of life, it is just common sense that a drug that makes them throw up and lose their hair, while wrecking their immune system, is doing the exact opposite.*

Chemotherapy can give the patient *life-threatening mouth sores. It attacks the immune system by destroying billions of immune cells (white blood cells). Its deadly poisons inflame every part of the body. The drug can slough off the entire lining of their intestines. The most common side effect experienced among chemo patients is their complete lack of energy.*

The new additional drugs now given to many chemo patients *may prevent the patient from noticing some of the side effects, but they hardly reduce the immensely destructive and suppressive effect of the chemotherapy itself. Remember, the reason chemotherapy can shrink some tumors is because it causes massive destruction in the body.*

If you have cancer, you may think that feeling tired is just part of the disease. This is rarely the case. *Feeling unusually tired is more likely due to anemia, a common side effect of most chemotherapy drugs. Chemo drugs can dramatically decrease your red blood cell levels, and this reduces oxygen availability to the 60-100 trillion cells of your body. You can literally feel the energy being zapped from every cell of your body—a physical death without dying. Chemo-caused fatigue has a negative impact on day-to-day activities in 89% of all patients. With no energy, there can be no joy and no hope, and all bodily functions become subdued.*

One long-term side effect is that these patients' bodies can no longer respond to nutritional or immune-strengthening approaches to cancerous tumors. *All of this may explain why cancer patients who do not receive any treatment at all, have an up to four times higher remission rate than those who receive treatment. The sad thing is that chemotherapy does not cure 96% to 98% of all cancers anyway.* ***Conclusive evidence (for the majority of cancers) that chemotherapy has any positive influence on survival or quality of life does not exist.***

To promote chemotherapy as a treatment for cancer is misleading, to say the least. *By permanently damaging the body's immune system and other important parts, chemotherapy has become the leading cause of treatment-caused diseases such as heart disease, liver disease, intestinal diseases, diseases of the immune system, infections, brain diseases, pain disorders, and rapid aging.*

Before committing themselves to being poisoned, cancer patients need to question their doctors and ask them to produce the research or evidence that shrinking a tumor actually translates to any increase of survival. *If they tell you that chemotherapy is your best chance of surviving, you will know they are lying or are simply misinformed*

As Abel's research clearly demonstrated, there is no such evidence anywhere to be found in the medical literature. **Subjecting patients to chemotherapy robs them of a fair chance of finding or responding to a real cure and DESERVES CRIMINAL PROSECUTION.**

Here is a recent article I found on www.sciencenews.org Here is a shortcut link to the article http://tinyurl.com/aqk7wj

Scientists find a soup of suspects while probing milk's link to cancer
Latest studies focus on estrogens, androgens and IGF-1

By Janet Raloff

March 28th, 2009; Vol.175 #7 (p. 5)

Got milk? Adults who answer yes may face a slightly heightened risk of cancer. Some emerging data may help scientists figure out why.

For more than a century, people thought that any beverage safe enough to serve to a weaning child couldn't hurt an adult. But test-tube studies and studies in adults over the past decade have linked cow's milk with an excess cancer risk in the prostate, and to a lesser extent in the breast and ovaries, notes oncologist Michael Pollak of McGill University in Montreal. Although scientists seeking to explain the link have fingered some suspects—such as milk's natural stew of hormones, growth factors and other biologically active chemicals—there's no "smoking gun," he says.

But a new study by researchers at the National Cancer Institute at Frederick, in Maryland, offers some ammunition.

Timothy Veenstra and his colleagues assayed grocery-store milk for 15 estrogens: estrone, estradiol and 13 metabolic derivatives of these female sex hormones.

Typically, hormones are produced in the body for use in the body. They act as orchestra conductors, telling genes when to turn on and off. But externally derived hormones add noise.

Estrogens can fuel the growth of many tumors, even in the prostate--and estrogen can do this at amazingly tiny concentrations. Identifying how estrogens' prevalence varies by milk type, and in what chemical form the hormones occur, required a new assay, which the NCI scientists describe in an upcoming issue of the Journal of Chromatography B.

Using that technique, they showed that the mélange of estrogens varies widely between milks. Whole milk contained the smallest quantity of estrogens, and amounts ascended from 2% to skim and buttermilk. In all of these milks, the majority of estrogens had undergone a minor chemical modification, rendering them less directly bioavailable and less hormonally active.

However, these modified, or conjugated, estrogens are not inert, and they can be converted back to their more potent parent compounds. What's more, the NCI scientists note, studies by others have shown that relative to free, bioavailable estrogens, conjugated ones take longer to get from the gut into the blood.

Veenstra's team concludes that compared with free estrogens, milk's conjugated ones "are likely to have longer half-lives."
Overall, skim milk had the smallest quantity of free estrogens. However, the conjugated type that dominated skim milk's profile, 2-hydroxyestrone, is known to be one of the most reactive and potentially risky of the metabolites, notes Xia Xu, a toxicologist on the NCI team. That metabolite's concentration in fat-free milk was second only to buttermilk's.

Dermatologist F. William Danby, who teaches at Dartmouth Medical School, also worries about other sex hormones in milk—the "male" androgens.
While estrogens may fuel tumor growth in reproductive tissues, certain androgens—ones that Danby refers to as 5alpha-reduced androgens—"have the capacity for increasing the number of estrogen receptors." In the January/February Dermato-Endocrinology, Danby notes that milk contains at least one receptor-proliferating androgen: 5alpha-pregnanedione.

Extra receptors, he explains, permit more estrogen—including any from milk—to unlock the cellular machinery that can turn tumor growth on. In other hormone systems, when excess hormone shows up, the body often cuts back on its production. Because the body has had relatively little evolutionary time to adapt to dietary sources of the 5alpha-reduced androgens, Danby says, no such feedback system has evolved.

"And this is probably the most important thing," he says. Milk-derived hormones "are being poured into a system that didn't anticipate them"—at least in adulthood.

One of the most provocative aspects of the milk story is its impact on insulinlike growth factor 1. Many studies have linked elevated concentrations of IGF-1 with cancer risk. Not only is milk a rich source of the substance, but people who drink milk also end up with more IGF-1 in their blood.

As with so much in science, however, the IGF-and-milk story is anything but simple, notes David Kleinberg, an endocrinologist at New York University School of Medicine.

Ordinarily, IGF-1 production is turned on when human growth hormone, produced in the pituitary gland, hits certain tissues. IGF-1 becomes the growth hormone's agent to locally trigger cell growth.

"We showed that IGF-1 can completely take the place of growth hormone" in breast tissue, Kleinberg says. In other words, IGF-1 can trigger cell growth without an outside cue.

Although estrogen is linked to breast development, it's impotent in the absence of IGF-1. Estrogen can amplify the cell-proliferating effects seen with IGF-1, his team has shown — both in the breast and prostate.

In the February Endocrine Reviews, Kleinberg and his colleagues note that when an excess of IGF-1 or estrogen occurs in the presence of the other, breast hyperplasia occurs—essentially cell division on overdrive.

"And when you get hyperplasia, it can put one at risk for breast cancer. Very slightly at risk," he says, "like maybe less than a doubling of risk."

But the real kicker: "There's a lot of interpersonal variability in our natural production of IGF-1," Pollak points out. "And even though we are sure that drinking more milk will increase your IGF-1 level, milk's contribution will still only account for a trivial part of the variation between people."

Which means genetics trumps milk intake. So people who naturally rank in the top quarter in terms of IGF-1 production and drink no milk, Pollak explains, "will still have a higher IGF-1 level than someone in the low quartile who drinks a quart a day."

Against this ambiguous backdrop, what's a milk drinker to do? Because the body of knowledge about this beverage's human bioactivity is still in its infancy, people may just have to employ the precautionary principle, Pollak says.

"In the absence of definitive [safety] data—or the presence of an adverse effect which may be small—you have to decide: Is there anything good about milk?" And other than developing children and malnourished adults, people probably don't need milk, he says. "I would never say anything stronger than that."

MORE THAN MILK-A new analysis measured the relative abundance of estrone (E1), estradiol (E2) and their metabolites in different types of milk. Bars show total estrogens versus free estrogens, which are directly bioavailable. These data reveal no obvious trends related to fat or protein content of milk, but some of the estrogens, including 2-hydroxyestrone (20HE1), are already known to be dangerous.Adapted from D.W. Farlow et al., Journal of Chromatography b 2009; DNY59/istockphoto

DAIRY COW HUSBANDRY AND HORMONES-Estrogen levels vary by type of milk, but when a cow is milked also affects the content. U.S. cows are milked 10 months a year (light blue) and are pregnant for nine (darker blue).

Estrone and estradiol levels increase in the cow (Plasma E1/E2) and her milk (Milk E1/E2) during pregnancy. The cows are dry, or not milked (yellow), for two months before giving birth, when estrogen levels are highest. One study has shown that estrogen levels are lower in milk from cows kept by a nomadic group in Mongolia that milks cows for human drinking just six months a year and only early in pregnancy.Sources: Graph from Zambito et al./Journal of Dairy Science; Calendar adapted from Akio Sato and Ganmaa Davasaambuu

On 3/26/09 I found a very appropriate article for this report at www.webmd.com here is the exact link to the article. http://www.webmd.com/health-ehome-9/pesticides-hormones-in-food?spon=2604_1?ecd=wnl_lbt_032509

Safer Food For a Healthier You
By Matthew Hoffman, MD
WebMD Feature
Reviewed by Michael W. Smith, MD

Pesticides in produce, hormones in milk, antibiotics in meat -- what are all these extra ingredients doing in our food?

Improved testing methods now allow researchers to detect and monitor a strange brew of unpleasant chemicals in our food and bodies. Although the amounts are small and there's controversy about whether or not they're harmful, their presence alone is disturbing to many --especially parents of small children.

"Modern production of foods incorporates a wide range of synthetic chemicals," says Jeff Gillman, PhD, associate professor of horticulture at the University of Minnesota and author of The Truth About Organic Gardening. "Many of these chemicals have the potential to be very damaging to humans if they are exposed to high concentrations, or to low concentrations over an extended period of time."

"More people are realizing there's a myriad of chemicals in conventionally produced food," says Craig Minowa, environmental scientist with the Organic Consumers Association, a nonprofit advocacy group. Although each has passed its own safety review, Minowa points out that "most of the studies on safety are done or supported by the companies themselves."

So what are the health effects of these unwanted ingredients?
Pickles, Lettuce, Mayo … Hold the Estrogen

Injecting hormones into young livestock can make them gain weight faster. More weight means more meat, which means more profit for the producer. Hormones also increase the production of milk by dairy cows.

Hormones have been used for decades in the meat and dairy industries. Synthetic estrogens and testosterone are the most common. Typically, farmers implant a pellet in a cow's ear at an early age; it releases hormones throughout the animal's life.

Initial concerns about estrogen-injected cows centered on a compound called diethylstilbestrol (DES). Nearly all beef cattle were treated with DES in the 1950s and 1960s. DES was also used as medicine, given to pregnant women to prevent miscarriages.

However, it was also discovered that DES caused a higher risk of vaginal cancer in the daughters of women who received the medicine. By the 1970s, over the protests of ranchers, diethylstilbestrol was phased out from use in medicine and agriculture.

It's also long been known that breast cancer risk increases with higher lifetime exposure to estrogen. These facts have led many to question whether the continued use of synthetic estrogens in livestock is safe.

Recombinant bovine growth hormone (rBGH) is a different class of hormone that increases the amount of milk dairy cows produce. Some suggest that although rBGH itself appears safe, it increases the amount of other chemicals in the body that might cause cancer. So far, there's no definitive proof one way or the other.

How much hormone is in a hamburger, and could it hurt you? The answer is, no one really knows. Studies show the added hormones do show up in beef and milk, pushing their estrogen and testosterone content to the high end of normal for cows. Whether that translates to increased risk for humans is the question.

"It really depends on how you look at the science," Minowa tells WebMD. "Many industry-funded studies show no risk, but there are independent studies that suggest" a potential cancer risk from hormones in milk.

Hormone-treated meat has long been suspected of contributing to early puberty in children, although the link has not been proven. There's no question that the age of puberty has been decreasing in the U.S. But some suggest that's due to improved nutrition and health, not to second helpings of hormones in children's diets.

The effects are very hard to study, experts say, because hormones are naturally present in both food and our bodies. Plus, the effects could be subtle and take years to show up.

The amount of hormone that enters a person's bloodstream after eating hormone-treated meat is small compared with the amount of estrogen a person produces daily. However, even low levels of hormones can have strong effects on some body processes.

Responding to the lack of certainty, the European Union has banned all hormones in beef, and Japan, Canada, Australia, New Zealand, and the EU have banned rBGH. No major studies are under way in the U.S. to evaluate the safety of hormones in meat and milk.
Produce and Pesticide Residue

Farmers use pesticides on many conventionally grown fruits and vegetables. The EPA sets limits on how much pesticide residue can remain on food. It's a complex process that's not easy to understand, incorporating variables such as the toxicity of the pesticide and how much of the food people generally eat. At the end, each of the 9,700 pesticides (at last count, in 1996) receives a number called a "tolerance."

The EPA, FDA, and USDA all play a role in ensuring pesticides on our food don't exceed the tolerances. In 1999, 40% of U.S. produce tested by the government contained pesticide residue. About 1% of domestically produced and 3% of the imported food had levels that violated standards.

While those numbers might seem reassuring, skeptics point out that no one could possibly test all the food grown or imported into the U.S. Even 1% of the total produce in the U.S. is a huge amount, Gillman points out.

And although pesticide tolerances are assumed to be safe, these chemicals are by their very nature toxic, and haven't been studied directly in people.

According to Minowa, the individual safety profiles of pesticides don't take into consideration any hazard from their combined effects. "Take a box of [cereal] off the shelf, and you can find residues from 32 pesticides," Minowa says. "Each one is within its tolerance, but what's the effect of those chemicals acting in combination in our bodies?"

According to FDA data analyzed by the nonprofit Environmental Working Group, the following fruits and vegetables tend to contain the highest levels of pesticide residue:

* Peaches
* Apples
* Sweet bell peppers
* Celery
* Nectarines
* Strawberries
* Cherries
* Pears
* Imported grapes
* Spinach
* Lettuce
* Potatoes

The foods with the least pesticide residues were:

* Avocados
* Frozen sweet corn
* Pineapples
* Mangos
* Asparagus
* Frozen peas
* Bananas
* Cabbage
* Broccoli
* Papayas

You can reduce your exposure to pesticides by buying organic for the high-pesticide items. Conventionally grown produce should be fine for those on the low-residue list, according to EWG.

Whether it's organic or conventional, you should take steps to reduce contamination of fresh food by pesticide or bacteria:

* Always wash fresh produce thoroughly.
* Peeling produce reduces pesticide residue and bacteria, although it also can remove valuable nutrients.

Antibiotics in Meat

Ranchers and farmers feed antibiotics in a daily low dose to their livestock. It's not to stop them from getting sick, but to make them gain weight.

But many doctors and researchers suspect that this practice is contributing to the rise of antibiotic-resistant bacteria, posing a serious danger to our health:

* A 2001 study in the New England Journal of Medicine showed that 84% of the Salmonella bacteria in supermarket ground beef were resistant to some antibiotics.
* Another study in 2002 suggested that some people caught resistant strains of Salmonella from eating pork that had been fed the antibiotic ciprofloxacin.
* The FDA estimates that use of antibiotics in chickens directly led to 11,000 people catching intestinal illnesses from antibiotic-resistant bacteria in 1999.

Partly because of these findings, several major fast food chains have refused to buy chicken treated with ciprofloxacin or similar antibiotics. Other companies continue to buy and sell antibiotic-treated meat, though.

There's no easy way to know if the meat you buy was raised with antibiotic feed. Companies aren't obligated to label their meat, or to provide consumers with the information.

"The best way to do that is to look for organic products, or to buy locally," says Minowa. "If you have a direct relationship with the farmer raising your food, you can just ask them."
Reduce Residues: Buy Local or Organic

Buying from local farmers' markets gets you the freshest produce possible. It also makes your food "greener" by reducing the wasted fuel, pollution, and greenhouse gases created by long-haul shipping.

"By buying local, you also have the ability to ask the farmer which pesticides he or she used on the crop as it was grown," says Gillman.

"Organic" is a term that's regulated by the USDA. Organic produce can't be treated with conventional pesticides, and must be grown in nearly pesticide-free soil. For these reasons, organic fruits and vegetables have much lower pesticide residues.

To be sold as organic, livestock must meet several criteria:

* They are fed only organic, vegetarian feed. They may not be fed meat from other slaughtered animals (a common component of conventional livestock feed).
* They are not treated with any antibiotics or hormones.
* The meat is not treated with radiation.
* They are raised under conditions that allow exercise and access to the outdoors.

The USDA can inspect farms for compliance. It's believed that the vast majority of organic farmers follow these practices.

The main drawback to organic food is expense. As you've noticed in the checkout lane, organic food nearly always costs more than conventionally produced food.

Is buying organic money well spent? Limited research suggests that some organic foods have more nutrients than conventional food. And then there's the issue of the environment. Gillman cautions that "organic practices aren't always 100% sustainable and green either," but they are usually "greener" than modern industrial farming.

To Minowa and many others in the organic food movement, "it's a matter of responsibility. Each bite that you consume, each dollar that you spend provides an opportunity to make positive change for a sustainable future."

The following story was printed from **FindArticles.com**, located at **http://www.findarticles.com/Natural Health**, Jan, 1999

I found this article by Sara Fremerman, which I believe would be very helpful for you.

13 Ways to Prevent Breast Cancer.
Author/s: Sarah Fremerman

YOU HAVE A 1-1N-8 CHANCE OF DEVELOPING BREAST CANCER IN YOUR LIFE. THIS PLAN SHOULD CHANGE THOSE ODDS.

WOMEN KNOW THE NUMBERS all too well--1 in 8. They show up every time we read about breast cancer or hear about some new drug that's supposed to cut the odds. But a woman's chances of getting breast cancer in her lifetime, in spite of new drugs and the millions poured into research, aren't shrinking.

Theories abound for ways to improve the odds--take Tamoxifen, exercise regularly, take antioxidants, don't live near toxic waste sites, even have a preventive mastectomy. Some make sense and could help; others seem far-fetched or even barbaric.

Natural Health has consulted five breast cancer experts, both those from the conventional fold and others more alternative in their approach, and asked them to help us build a program that would significantly lower your risk of getting the disease. The centerpiece of this plan is diet. It's the one thing you have direct control over. And, according to Mitchell Gaynor, M.D., director of medical oncology and integrative medicine at Strang Cancer Prevention Center in Manhattan, it has proven to be the most effective way to reduce your risk.

Other important suggestions about such things as exercise and going braless appear in "Cancer Dos and Don'ts" on page 95. But first, here are 13 simple ways you can redesign your diet and start protecting yourself today.

1 SAVOR SEAWEED

WHAT TO DO

Eat seaweeds such as kelp and nori often. Or consider taking blue-green algae such as spirulina (1 heaping teaspoon) and chlorella (3 g) in a glass of juice daily.

WHY

* Jane Teas, Ph.D., of the Harvard School of Public Health found that rats fed kelp had less breast cancer than rats that were not fed kelp. The high consumption of kelp may explain the lower incidence of breast cancer among Japanese women (who have one-third the risk of breast cancer of American women).

* Kelp, chlorella, and spirulina contain chlorophyll, which studies have shown to have anti-carcinogenic effects, as well as vitamin C and carotenoids, which fight free radicals.

2 HALVE THE FAT

WHAT TO DO

Limit your daily fat intake to 20 percent of your overall caloric intake.

WHY

* A diet high in fat (especially animal fat) is known to increase the risk of breast cancer. One study found that the risk of breast cancer increases for Japanese women who move from Japan (where daily fat intake is about 20 percent of total calories) to the United States (where daily fat intake is about 40 percent of total calories).

* According to Charles Simone, M.D., author of Breast Health (Avery Publishing Group, 1995), a high-fat diet produces chemicals in the intestine that bacteria convert to carcinogenic estrogens. These estrogens can then be stored in the fatty tissue of the breast, making cells in this area more susceptible to cancer growth.

3 PILE ON THE FIBER

WHAT TO DO

Get plenty of fiber from foods such as fruits and vegetables, beans, and whole grains.
WHY

* Robert Arnot, M.D., author of The Breast Cancer Prevention Diet (Little, Brown and Company, 1998) says that fiber interrupts the body's metabolism of estrogen and decreases the blood levels of estrogen. High levels of estrogen in the bloodstream correspond to a higher risk of breast cancer. High-fiber diets can decrease breast cancer risk by up to 54 percent.

4 Crunch Cruciferous Veggies

WHAT TO DO

Consume plenty of cruciferous vegetables--broccoli, Brussels sprouts, cabbage, turnips, bok choy, kale, and cauliflower. Steam them or eat them raw to best preserve their cancer-fighting nutrients.
WHY

* Cruciferous vegetables contain sulfurous compounds called indoles, which help eliminate estrogen from the body and prevent it from triggering the growth of breast cancer. According to Gaynor, author of Dr. Gaynor's Cancer Prevention Program (Kensington Books, 1999), only cruciferous vegetables are known to convert estrogen in the body from cancer-promoting forms to forms that actually protect against breast cancer. One particular indole, indole-3-carbinol (13C), inhibits the development of potentially cancerous cells in the breast.

5 GO FISH FOR FATTY ACIDS

WHAT TO DO

Eat at least three servings a week of cold-water fish such as tuna, salmon, halibut, mackerel, haddock, cod, and sardines. If you don't eat fish, you can also take fish oil capsules (2 to 10 g a day) or vegetarian supplements of algae-derived docosahexaenoic acid (300 mg daily).

WHY

* Omega-3 oils inhibit the effects of the compounds known as prostaglandins, which have been associated with the inflammation that suppresses the immune system's ability to identify tumors.

* In one major British study, researchers examined mortality data for breast and colorectal cancer in 24 European countries. A high consumption of animal fat was linked to more cases of cancer, while a higher consumption of fish and fish oil was linked to fewer cases of cancer.

* In a study from Finland, women with breast cancer were found to have lower levels of EPA and DHA, two omega-3 fatty acids, in their breast tissue than women with benign fibrocystic breast disease.

* North American Eskimo women, who eat a diet extremely rich in omega-3 oils, have no breast cancer at all.

6 Learn the Soy Secret

WHAT TO DO

Eat soy products such as tofu, miso, and tempeh regularly. (All soy products are not equally beneficial. Highly refined soy products, such as soymilk, soy burgers, and fake meats, contain much less genistein than traditional Asian soy products, and some may contain artificial preservatives. Soy oils and soy sauce are not good sources either: Soy oils contain unhealthy fats, and soy sauce is high in sodium.)

WHY

* Soybeans and other soy products contain genistein, a natural plant estrogen that binds to receptors in the breast, making it impossible for potentially cancer-causing forms of estrogen to connect with breast cells.

* Scientists are also investigating other benefits of soybeans. In one study, Seventh Day Adventist women, vegetarians who typically ate a lot of soy, were found to have a lower-than-normal risk of breast cancer. This may be because they have higher levels of the hormone DHEA, which is higher in women who are free of breast cancer.

* Gaynor says, "Soy does so many things--it is a weak estrogen that blocks estrogen receptors, decreases angiogenesis [the development of blood vessels that feed a tumor], increases apoptosis [cancer cell death], and contains enzymes that break down carcinogens in the body."

7 STOP AND SHOP ORGANIC

WHAT TO DO

Whenever possible buy organic food: fruits, vegetables, grains, dairy products, meat, and poultry.

WHY

Organic produce is free of pesticides such as DDT and other environmental toxins that have been linked to a higher

risk of breast cancer. Though DDT is banned in the United States, American manufactures export the pesticide to Third World countries, which often export tropical or out-of-season produce back to the United States. According to Devra Lee Davis, Ph.D., M.P.H., of the World Research Institute in Washington, D.C., organic fruits and vegetables contain higher levels of vitamins and minerals than nonorganic produce.
* Dairy products and meats that have been certified organic are free of (artificial) hormones like bovine growth hormone, a chemical fed to cows that has been shown to promote the growth of breast cancer cells.

8 STOCK UP ON SUPPLEMENTS

WHAT TO DO

Every day drink one cup of astragalus tea; take 200 mcg selenium; 30 to 100 mg of coenzyme [Q.sub.10]; 25 mg of grapeseed extract; 30 to 100 mg alpha lipoic acid; and a good multivitamin and mineral supplement.
WHY

* Astragalus. A 1990 study conducted at the M.D. Anderson Cancer Center in Houston found that taking astragalus daily increased the body's ability to kill cancer cells by tenfold.
* Selenium. A study by Larry Clark, Ph.D, M.P.H., associate professor at the University of Arizona showed that taking selenium could halve cancer rates, and an earlier study published in Holistic Medicine in 1989 concluded that the higher the blood selenium level, the lower the rate of breast cancer. Buy the organic form of selenium, selenomethionine, rather than selenite (the inorganic form).

* Co[Q.sub.10]. This nutrient protects against cancer by strengthening the immune system and zapping free radicals. However, there is no data that links Co[Q.sub.10] specifically to breast cancer prevention.

* Grapeseed extract. According to Gaynor, studies have shown that this antioxidant is 20 times more powerful than vitamin C and 50 times more powerful than vitamin E at scavenging free radicals.

* Alpha lipoic acid. This powerful antioxidant strengthens and regenerates other antioxidants in the body, especially vitamin E. Biochemist Richard Passwater, Ph.D., suggests that lipoic acid may even inhibit the activation of the gene that triggers cancer growth in cells.
9 MAKE THE MOST OF MUSHROOMS

WHAT TO DO

Regularly include medicinal mushrooms, especially the Japanese varieties maitake and shiitake, in your diet. Reishi, another medicinal mushroom, is slightly tough when cooked so you may want to buy it as a tea, tincture, or capsule.

WHY

* Studies have shown that maitake mushrooms stimulate immune function and also inhibit tumor growth. "Maitake D-fraction, the active ingredient in maitake, does not kill cancer cells directly," explains Cun Zhuang, Ph.D., who researches the anti-cancer effects of maitake. "It activates the immune system." According to Zhuang, maitake mushrooms have been shown to be particularly effective in protecting against breast cancer in mice. Some evidence suggests that maitake is effective against tumors in humans as well.

* Shiitake contains the polysaccharide called lentinan, which is known to boost the activity of the immune system.

* Reishi also contains polysaccharides.

10 THROW TEA PARTIES

WHAT TO DO

Drink one cup of green tea three times a day. Green tea contains about half the caffeine of coffee--you Can also buy decaffeinated green tea bags or try green tea capsules or tinctures.

WHY

* Green tea contains cancer-fighting antioxidants and polyphenols, which reduce the damage done by free radicals.

* In one study of women, drinking a lot of green tea--10 cups per day--significantly lowered the risk of cancer. Green tea may be another important protective factor responsible for the low rates of breast cancer among Japanese women.

11 CHOOSE THE RIGHT OILS

WHAT TO DO

Cook with virgin or extra-virgin olive oil. And use flaxseed oil in dishes that aren't heated (flaxseed oil is volatile and its chemical makeup is changed when it's exposed to light and heat). Avoid canola oil, safflower oil, corn oil, soybean oil, sesame oil, and margarine.

WHY

* Monounsaturated oils such as olive oil have been linked with lower rates of cancer. A study of women in Spain demonstrated a lower risk of breast cancer in those who were consuming the most olive oil. Lilian Thompson, Ph.D, a professor of nutritional sciences at the University of Toronto, has found that a daily close of flaxseed (1 tablespoon of oil or 3 tablespoons of seeds) can actually reduce breast cancer tumor size.

* Trans fats (denser fats, also called hydrogenated oils, found in margarine, for example) are linked with higher rates of cancer. A University of North Carolina study in 1997 confirmed a correlation between the consumption of the Trans fats in processed margarines and vegetable oils with an increase in breast cancer.

* Saturated fats (such as those in dairy products and red meat) cause the body to produce higher-than-normal levels of insulin, according to Arnot. Like certain types of estrogen, high levels of insulin can stimulate cancer cell growth in the breast. In a recent study, Pamela Goodwin, M.D., an associate professor at the University of Toronto, found a 283 percent increased risk of breast cancer in women with high insulin levels.

12 BEFRIEND PHYTONUTRIENTS

WHAT TO DO

Eat a wide variety of vegetables, fruits, grains, seeds, nuts, and legumes.

WHY

* These foods contain phytonutrients (plant nutrients such as polyphenols); compounds that protect against cellular damage and inhibit cancer growth. In one Harvard School of Public Health study, women who ate the most vegetables had a 48 percent lower incidence of breast cancer than those who ate the least; those who ate the most fruit had a 32 percent lower incidence than those who ate less fruit.

* Health researcher Robin Keuneke, author of Total Breast Health (Kensington Books, 1998), suggests cooking with herbs such as dill, which contains limonene, a phytochemical important for breast protection, and rosemary, which has antioxidant and anti-tumor properties.

13 EAT AN ALLIUM A DAY

WHAT TO DO

Eat plenty of allium vegetables--garlic, onions, leeks, and shallots. For optimal benefits (if you're brave), eat alliums raw.

WHY

* According to the National Cancer Institute, garlic is one of the best foods for protection against cancer. It contains the anti-cancer mineral selenium, which stimulates white blood cell production and induces apoptosis (cancer cell death).

* Onions and other allium vegetables offer similar therapeutic effects--alliums contain compounds that stimulate the production of enzymes that neutralize the free radicals linked with cancer. Alliums also contain saponins, which prevent cancer cells from multiplying.

* John Milner, Ph.D., head of the nutrition department at Pennsylvania State University used an aged garlic extract to successfully prevent the development of breast cancer tumors in rats exposed to carcinogens. Of the rats that received no garlic extract, 90 percent developed tumors. Only 35 percent of the garlic group developed tumors. And a 1995 study in Oncology Reports showed that the sulfur compounds in garlic extract inhibited the growth of precancerous humanbreast cells, and increased levels of an important detoxifying enzyme.

CANCER DOS AND DON'TS

DO

Get 4 hours of vigorous aerobic exercise a week. A study of 1,000 women showed that those who exercised 3.8 hours or more a week had less than half the rate of breast cancer of those who didn't exercise.
Stay within 12 pounds of your ideal body weight. In many studies, obesity has been correlated with a higher risk of breast cancer. Excess body fat produces estrogen, which can then be stored in breast tissue and trigger the growth of cancer cells.

Breast-feed infants. Breast-feeding interrupts ovulation and as such is thought to reduce the amount of time estrogen circulates in the body.

Go braless for a few hours each day. In Dressed to Kill: The Link Between Breast Cancer and Bras, (Avery Publishing Group, 1995), authors Sydney Ross Singer, Ph.D., and Soma Grismaijer found that women who wore a bra more than 12 hours a day had a 19 times greater risk of breast cancer than those who wore a bra fewer than 12 hours. The scientific credibility of this finding is still being debated.
Sleep in total darkness. Light inhibits your body's production of the hormone melatonin, and lower levels of melatonin have been correlated with a higher risk of breast cancer.

Spend time in the sunshine--15 minutes a day, 3 times a week. Sunlight helps the body to produce vitamin D, which has been linked to lower breast cancer rates.

Use hormone replacement therapy with caution. Some researchers link supplementation with DHEA and other hormones with an increased risk of breast cancer, though no conclusive information is available yet. Check with your doctor before embarking on any hormone replacement program.

DON'T

Drink excessive amounts of alcohol. According to Simone, women who have 2 to 4 alcoholic drinks per week have a 2 to 3 times higher risk of developing breast cancer than those who don't.

Use dark hair dyes for several years continuously. A 1980 study showed that women who dyed their hair to change its color (rather than to camouflage gray) were at a three times greater risk for breast cancer.
Smoke. One study of close to 85,000 women showed a higher risk of breast cancer in smokers than in nonsmokers.

SOME AGE-OLD ADVICE

Pre-pubescent girls: Eating plenty of organic yogurt at this age can reduce the risk of breast cancer by half, according to a study in the International Journal of Immunotheratry

Teens: Strenuous exercise during adolescence lowers the risk of breast cancer later in life. If possible, avoid taking birth control pills--they increase the risk of breast cancer later. Begin monthly serf-exams. Express feelings openly--talk out problems as they arise.

20s and 30s: Ideally the pill should be used for a prolonged period after the birth of a woman's first child. Having a child before the age of 35 lowers your risk of breast cancer.

40s: Experts disagree about whether it is necessary to get a baseline mammogram in your 40s. Check with your doctor. At this stage, doing a monthly breast self-exam is crucial.

50s and up: Get annual mammograms. Between the ages of 51 and 70, increase your daily dose of vitamin D to 400 IU. If you are 71 or older, you should be taking 600 IU per day.

RELATED ARTICLE: The Protective Mind

CERTAINLY OUR MINDS and emotions play a pivotal role in the prevention, recurrence, and remission of breast cancer. One recent study in the Journal of the National Cancer Institute reported that following breast cancer surgery, women showing the worst signs of stress and depression also experienced the most profound suppression of their immune systems.

In response to findings like this, cancer specialists like Jeremy Geffen, M.D., founder of the Geffen Cancer Center in Vero Beach, Fla., are addressing not only the physical, but also the spiritual aspects by combining conventional medicine with meditation, yoga, and massage.

The following are a few resources to help you use your mind to protect yourself from breast cancer.

The Center for Mind-Body Medicine. Phone: 202-966-7338. Web address: **http://www.healthy.net/cmbm.**

The Geffen Cancer Center. Phone: 561-770-5800. Web address: **http://www.geffencenter.com/** Stress Reduction Clinic at the University of Massachusetts Medical Center. Phone 508-856-2656.

Rituals of Healing: Using Imagery for Health and Wellness by Jeanne Achterberg, Ph.D. (Bantam Doubleday Dell, 1994).-**Sarah Fremerman is a frequent contributor to Natural Health.**

Some of you may have heard of the potential beneficial effects of apricot seeds for cancer. I found some information on the Internet recently that I felt you should know about, Apricot pits with vitamin B17 and their potential benefits in dealing with cancer.

For those of you who surf the Internet, there are many places where you could buy apricot pitsI saw them being sold for as low as $16.95 for two pounds to as much as $25.00 for one pound.

Whether you have a computer or not, you should first check with your local health food store to compare prices. Before you run out and buy them I would like you to read the following:

Apricot Seed Fact Sheet (Prunus armeniaca)

Description-Apricot seeds are used by Chinese herbalists to relieve bronchial problems. These seeds also known as bitter almonds are believed to serve as an expectorant and also help to stop coughing. In addition apricot seeds may act as a laxative. In traditional Chinese medicine, apricot seeds are classified as bitter and slightly warm.

How It May Benefit You... coughing and wheezing, bronchitis, asthma, emphysema, constipation.

Caution: If you have too much you may experience dizziness, nausea, vomiting, and headache and this can lead to more fatal symptoms and death.

WARNING: This herb contains a poisonous substance and should only be used under the supervision of an experienced herbalist.

Chinese medicine practitioners have advised using extreme caution in using apricot seeds to treat children or patients with diarrhea. **Some Chinese medicine practitioners believe that apricot seeds should not be taken with the herbs - astragalus, skullcap, or kudzu root.**

I went to Yahoo and typed "apricot pits" and found many sites with information about Vitamin B17. After reading the following excerpt, I feel that eating apricot pits would be an inexpensive way to potentially avoid and or beneficially deal with cancer.

Now that you know of the cautions, here is some information on why you may want to eat apricot pits. There are some who believe that one of the reasons for cancer is the lack of a vitamin called B17, which can be found in apricot pits.

Here is an excerpt from the book, Alternative Cancer Cures: The Nature of Cancer by Dr. Ernest T. Krebs Jr.:

"It is certainly a pleasure to be here at the Second Annual Convention of the Cancer Control Society, an outgrowth, as you know, of the International Association of Cancer Victims and Friends.

As I look back through the years marketing the emergence of these two fine Societies, I can recall the number of miraculous victories we have had in those intervening years; that it is as true today as it was eleven years ago that Laetrile, Vitamin B17, is the first and last final hope in the prophylacsis in therapy of cancer in man and animals. The reason for this is that Laetrile is a vitamin. It is the 17th of the B vitamins.

We hear a great deal about its use in terminal cancer, but the time to start with vitamin B17 is now before the disease become clinical. The time to start is the same with any matter of adequate nutrition and that is right now. You may start now by commencing to eat the seeds of all common fruits that you eat. The apricot and peach seed contain almost 2 percent of vitamin B17 by weight. The apple seed, although very small, is equally rich in Vitamin B17. So are the seeds of prunes, plums, cherries, and nectarines. The only common fruit on the hemisphere that lacks nitrilosidic seeds, are the citrus fruits. This lack has come about by artificial cultivation by breeding and hybridization, since the seeds of citrus fruits on the African continent still contain Vitamin B17.

Two more rich sources of Vitamin B17 are the simple cereal millet and buckwheat. Macadamia nuts, although expensive and exotic, are very rich in Vitamin B17 and so are bamboo shoots, mung beans, lima beans, butter beans and certain strains of garden peas. But for convenience, the simple source for your Vitamin B17 are the seeds of the common fruit.

We know something about the prophylactic dose of Vitamin B17. For example, we know the Hunza's represent a population that has been cancer free for over 900 years of its existence. This population has a natural diet, which supplies on the average between 50 to 75 milligrams of Vitamin B17 a day.

Hunzaland is a land that has sometimes been described as the "place where apricot is king." The Hunzakuts eat the fresh apricots for the three months they are in season and the remainder of the year they eat dried apricots. They never eat a dried apricot without enclosing the seed between them. This supplies them with better than average of 50 to 75 milligrams of Vitamin B17 a day.

There are many of us in the Western World who don't ingest this amount of Vitamin B17 in the course of an entire year. As a result we're in the midst of a fulminating deficiency of Vitamin B17 or nitriloside, the anti-neoplastic vitamin. Its absence from our diets accounts for the fact that cancer on our population has reached such a pandemisity as to account for its occurrence in one in every three American families…"

Remember to keep in mind that this report is to educate you on the potential resources that are available to anyone who is interested in beating Cancer.

I found the following information in a magazine called **EnergyTimes the May 2002 issue**. You could find this magazine in your local health store; it's free. The article is called **Cancer Weaponry** and it gives you foods, what substances they contain, and the latest research. Since people are always asking me about what they should eat, here are some examples:

Beans & Peas-Contain fiber, folate, lignins (type of fiber), phytosterols (plant fats), and saponins (phytochemicals with a sweet taste). The latest research-The fiber in beans has long been linked with lower cancer rates; now research shows that folate, a B vitamin, may reduce your colorectal cancer risk. Phytosterols, healthy plant fats, can slow the growth of prostate cancer.

Berries (Strawberries, Raspberries, et al)-Contain anthocyanadins (dark pigments), vitamin C. The latest research-Anthocyanadins and vitamin C are antioxidants that may help protect DNA against damage that leads to cancer. In lab studies, berries' anthocyanadins interfere with the metabolism of cancer cells. Research on black raspberries show they may lower your risk of esophageal cancer.

Cruciferous Vegetables (Broccoli, Cauliflower, et al) - Contain dithiolthiones, fiber, indoles, isothyocyanates, selenium, sulfurophane (chemical responsible for broccoli's sharp taste), and vitamin C. Latest research- Sulfurophane activates; over enzymes responsible for neutralizing toxins and indoles appear to inhibit the development of breast cancer cells. Selenium may fight prostate and stomach cancer, and deter colon cancer.

Carrots, Apricots, Sweet Potatoes, Cantaloupe - Contain Beta-carotenoids (red, orange and yellow pigments). Latest research-Carotenoid-rich foods stimulate immunity; daily consumption seems to protect against prostate and stomach cancer. Smokers who eat these fruits and vegetables may reduce their lung cancer chances. In one large study, women who ate the most carotenoids had a relatively low breast cancer risk.

Citrus Fruits (Oranges, lemons, Grapefruit)-Contain bioflavonoids (pigments), folate, hesperatin, limonene, narangenin, vitamin C. Latest research-Eating citrus fruits is linked to a lower rate of esophageal and stomach cancers. Men who consume the most vitamin C run the lowest risk of death from causes, including cancer. Bioflavonoids inhibit cancer-promoting hormones and enhance activity of cancer-fighting immune chemicals.

Fish-Contain omega –3 fatty acids: docosahexaenoic acid, eicosapentaenoic acid (EPA). Latest research-Omega-3 fats inhibit COX-2, an inflammation-stimulating chemical that may promote cancer by increasing production of free radicals, and molecules that damage DNA. Most studies show that eating fish lowers your risk of cancer.

Garlic - Allicin (sulfur-bearing compound) - Contains antioxidants, germanium, isoflavones, selenium. Latest research-More than a dozen studies show that garlic provides strong protection against stomach and colon cancer. Garlic can block creation of cancer-causing compounds in the digestive system; it also keeps cancer cells from proliferating.

Mushrooms: (Maitake, Lion's Head, Shiitake, et al)-Contains antioxidants, lignins, polysaccharides (long chain sugar molecules), and selenium. Latest research - Polysaccharides stimulate immunity-Lentinan, from shiitake, fights inflammation and is used in Asia to enhance cancer treatment and reduce side effects. Studies show that substances in Maitake can be useful against cancer.

Olive Oil - Contains antioxidants, lignans, phenols, secoiridoids, and squalene. Latest research-In a worldwide study, people who ate the most olive oil had the lowest rates of colon cancer. Squalene interferes with an enzyme that can activate cancer genes.

Soy - Contain daidzein, genistein, lecithin, phytosterols, and saponins. Latest research-The low rates of breast cancer in Asia have been linked to eating soy foods. Soy compounds fight cancer by inhibiting cancer-promoting enzymes and slowing the blood supply to tumors. They may also block the action of cancer-promoting hormones.
Spices (Rosemary & Turmeric)-Contain carnosol, diterpenes, rosmaricine (rosemary), and curcumin (turmeric). Latest research- Carnosol and curcumin both interfere with the COX-2 enzymes, chemicals that lead to inflammation. Rosemary also encourages production of detoxifying enzymes and keeps cancer –causing agents from attaching to DNA, while curcumin inhibits tumor development.

Tea (Black & Green)-Contains antioxidants, Epigallocatechin Gallate (EGCG), theaflavin, thearbigin. Latest research-Tea's antioxidants may reduce carcinogenic DNA damage. EGCG inhibits growth of cancer cells, and green tea has slowed breast cancer growth in lab studies. Tea drinkers have been shown to experience lower rates of lung cancer and melanoma.

Tomatoes - Contain Antioxidants, coumarins, flavonoids, lycopene (red pigment) and other carotenoids, vitamin C. Lycopene is best known for reducing the risk of prostate cancer. But research also links lycopene to lower rates of lung cancer; lycopene and vitamin C together may reduce breast cancer risk.

Whole Grains (Brown Rice, Whole Wheat, et al) - Contain B vitamins, fiber, saponins and natural vitamin E. Latest research-Whether or not dietary fiber lowers colorectal cancer risk is still controversial, but experts still recommend consuming larger amounts of fiber from cereals and whole grains. Natural vitamin E has been linked to lower prostate cancer rates.

Royal Raymond Rife, Jr. Technology

I included the following information on the Rife technology because I feel that you should know as much as you can about the various ways and techniques that are available to you regarding your health. If you want to learn more about the Rife Technology just type in "Royal Rife" into any search engine and you will be amazed as to how much information there is regarding his technology.

You will find people selling the Rife frequencies software for $100.00 or less and machines for as much as $3,000 and more. After reviewing many sites I settled on the Model A from Wright Laboratories. It is one of the most affordable and the company offers as much customer support as you would need. I will give you more details later in this report on how you can buy one of their machines.

If you decide to contact Wright Laboratories learn as much as you can through an Internet search before you contact them. The following information came from **http://www.jwlabs.com/.**

Has the Greatest Health Discovery in History Been Suppressed?

Royal Raymond Rife, Jr.
May 16, 1888 - August 11, 1971

Did a revolutionary microscope, invented in the 1920's, reveal a method of curing all types of germ-caused diseases? Can this technology, using radio frequencies, arrest most cancers, and actually cure them? Will this technology stop the dreaded AIDS virus, and halt the spread of Lyme's Disease, Butterfly Lupus, and other so-called "incurable" diseases?

The story of Royal Raymond Rife, genius scientist, can be likened to the most fascinating mystery. The Carl Zeiss Optical Company in Germany trained him for six years. He also worked for the Secret Service, U.S. Government. He became the inventor of powerful microscopes, leading to the discovery of a beneficial phenomenon dealing with viruses.

Rife received the backing of Mr. Timken, of the Timken Roller Bearing Company, who supplied funds to establish a laboratory in San Diego to finance his research.

Rife reasoned that if he was going to find a cure for diseases such as cancer, it was important to be able to see the live virus that caused the disease. In 1920, Royal Rife designed the first of several highly advanced microscopes, recognized as the most powerful in the world, and the only one that could be used to see viruses alive.

Rife's microscopes had resolutions and magnifications far more powerful than others of his day (or even today). Rife's Universal Microscope (1933) magnified 31,000 times; other microscopes of his day magnified only 3,000 times.

But Rife found that making a microscope with extreme magnification was not sufficient to see a colorless virus. Staining them with existing aniline dyes was unsuccessful because the virus was too small to absorb the colloidal particles.

Rife noted that the different frequencies of light caused certain microorganisms to luminate (light up) in their own resonant colors. So he invented a system of rotating prisms to select the appropriate light frequency (color), essentially staining the specimen with light.

Extrapolating from this resonant effect of light, he experimented with electromagnetic radio waves and discovered that for each type of virus, there was a particular resonant frequency that would cause it to burst into pieces and be destroyed.

He subjected test animals in his laboratory to lethal doses of pathogenic germs and found that he could invariably save their lives by subjecting their bodies for a few minutes to the electrical energy of the properly chosen frequency. Therefore, before the year 1930, he had built his first microscope and demonstrated that he could electronically kill pathogenic microorganisms.

This success demonstrated that any germ-created disease in man, animal or plant, could be quickly and painlessly eradicated! **This was Electronic Therapy - THE HEALTH TECHNOLOGY OF THE NEXT CENTURY!**

Can anyone imagine a more important discovery for mankind? It insured the virtual end to disease! (And at little cost, we might add.)

A primitive form of this technology is used today by the medical profession to treat certain types of leukemia. The patient's blood is pumped from the body and exposed to ultraviolet light. Unfortunately, this AMA-approved treatment does not treat the bone marrow, where blood cells originate. Also, it is painful and expensive. Rife's method did access the whole body, inside and out. It electrocuted the viruses and thus cured the so-called "incurable" diseases. Rife's method was totally harmless to the body, and extremely inexpensive.

Rife's success attracted the attention of many doctors and scientists. Dr. Arthur Kendall, a noted bacteriologist, contributed his "K Medium" which enabled the "filterable virus" portions of bacteria to be isolated and to continue reproducing.

Dr. Milbank Johnson was a strong supporter of Rife's work and arranged a dinner in honor of Rife and Kendall, attended by more than 30 of the most prominent people in the medical field.

Dr. Johnson set up a clinic where Rife treated 16 terminally ill cancer patients with his frequency instruments, and, after 3 months, the staff of 5 medical doctors and Dr. Alvin Foord, M.D., Pathologist for the group, pronounced 14 of the subjects clinically cured.

Dr. James Couche used Rife's frequency instrument for 22 years with continued success. He reported one case of a Mexican boy with osteomyelitis of the leg. The doctors had to scrape the bone every week, which was painful. After 2 weeks of treatment with the frequency instrument, the boy was completely healed with no reoccurrence.

RIFE'S DISCOVERY WAS SO REVOLUTIONARY, IT PROMISED TO ELIMINATE ALL GERM-CREATED DISEASE! And therein lay the basis for the suppression of his marvelous discoveries and inventions. The contributing factors to this suppression were scientific rivalries, institutional arrogance, one-man rule of the AMA, and vested interests of pharmaceutical companies.

Yet, in the years from 1934 to 1939, many doctors cured cancer, and other diseases using Rife's frequency instruments. Then, at the end of this period, extreme pressure was brought against the doctors to stop using this method, and their machines were confiscated.

In 1939, an engineer working with Rife to improve his instrument for commercial manufacture by the newly formed company, Beam Ray, brought suit against the company. This engineer obtained the powerful backing of the AMA, and the ensuing trial had a debilitating effect on Rife, leading eventually to alcoholism and depression. For all practical purposes, his aggressive research was over.

Two prestigious journals described Rife's revolutionary microscopes: The Journal of the Franklin Institute published an article, "The New Microscopes" in February 1944. The Smithsonian Institution published the same article in its Annual Report of the Board of Regents, year ending June 30, 1944, and republished it in 1945. The text is available in John Crane's book.

In 1950, John Crane became Rife's partner and worked to improve the frequency instrument and to document Rife's work. In 1958, he made a smaller frequency instrument that attached directly to the body. Doctors began using this smaller, lower cost, frequency instrument with success. Again, the health authorities surfaced and threatened the doctors, forcing them to shut down. Crane's office was raided and all machines, documents, and research records were removed, all without a search warrant.

The only way to investigate the Rife technology today is through personal research and experimentation.

NOTICE

Due to FDA regulations and various state laws, no medical claims can be made for the Royal R. Rife technology. All of the information expressed herein must be considered theoretical and unproven and for experimental research only.

Skeptical about Rife technology?

Great! That probably means you are "our kind" of people. A high degree of skepticism is a good thing, especially today. We welcome your skepticism. If you are skeptical and reading this page at all, you are probably like so many people who would like to believe in the Rife technology, but you need to have some really well grounded facts before you even open up to a dialog.

The more scientific you are about it, the better you will understand our message. But we are not really here to convince you of anything. Our job in this regard is to give a full disclosure to our users. We don't normally give much time to skeptics, for the reason that, in most cases, once you sit down in front of a really good instrument of this type, you won't be a skeptic anymore.

For what it is worth though, we will offer to you a few of the basic known quantities, in hopes that these will encourage you to call us for more information, or personal counseling.

The first thing is that Rife and his story are real historical events. Our link to the Smithsonian Annual Report for 1944 should prove that. Although not all of the things that have become popular diatribe are factual. Example: Some makers like to emphasize the persecution of Rife and Rife machine manufacturers, and use such exaggerations to create a sense of urgency by implying that it won't be available much longer.

If you have looked at a lot of web sites other than JWLABS, you have probably seen a lot of conflicting information, or plain disinformation. This confuses the important issues, which leads to skepticism in the minds of most reasonable people.

When we were first introduced to the Rife technique, we were skeptical too. In fact, we are among the most skeptical of people, in part because after you have been scammed enough times, you don't quickly accept much of anything, even when the proof is staring you in the face. At that time, (1986), all that Internet rhetoric and mumbo-jumbo, did not exist. We had to figure it out for ourselves.

One of the obvious and best known principals of the Rife technology likens it to shattering a glass with the right musical tones. A hundred years ago, there were probably a lot of people who would have a hard time believing that, unless they could see it for themselves. However, in the modern world, these principals are no longer doubted.

A lesser-known principal of Rife has to do with taking advantage of the difference in strength between virus and bacteria and that of a human cell. The difference can be as much as a million times. Radiation therapy attempts to take advantage of the difference in the strength of a normal human cell and a human cancer cell, which is a very small difference by comparison. The Rife transducer is in part, designed to exploit the difference between human cells and pathogens, by means of current. It only takes a few volts at miniscule amperage, to electrocute almost any pathogen, instantly. JWLABS machines deliver this electrocuting energy, which is at the same time a stimulus to the body, incorporating Rife's Mortal Oscillatory Rates. Even if you have doubts about the MORs, it is very difficult to be skeptical about the effects of microbial electrocution.

Electro-stimulators have been in use by the mainstream medical profession for many years. Their ability to revitalize and to reduce pain, are well established. It is also a matter of historical fact that much of that technology has been built on the work of Dr. Royal Raymond Rife, of San Diego, California.

You may examine additional information by reading our Rife FAQ page, which answers twice as many questions as are most often asked. This should at least take the edges off of your skepticism.

However, legitimate scientific or professional skepticism will probably not be satisfied in the present research environment of Rife technology. Although the theoretical principals of the therapy does not contradict established sciences and cannot be credibly refuted by the basic sciences of Pathology, Chemistry, Pharmacology, and Physics, it is still very often regarded as a fraudulent practice. This is likely due to the preponderance of poor quality devices or the low percentage of genuine instruments that are produced in the US.

It was predictable and inevitable that Rife technology would become popular amongst chiropractors, homeopaths, naturopaths, dentists, nurses, veterinarians, acupuncturists, etc, and this fact also contributes to the aura of the technology in the eyes of medical authority. These practices are often maligned by conventional medicine, not always justifiably. But the efficacy of the Rife technology has often been remarkable and for it to fall into the hands of unethical practitioners has both the effect of increasing the apparent success of otherwise medically unsuccessful practice, and of placing the technology in the same level of acceptability and credence as the practices in which it is so often used.

The general lack of regulation in some of these practices makes them a haven for quackery, which may be rightly seen as a tool of "calculated neglect" that encourages their vilification by conventional doctors and researchers. Homeopathic drugs, for example, are not subject to the same truth in advertising as conventional drugs, making them more vulnerable to invasion by unethical practitioners and thus attract ridicule unjustly, to those who are both sincere and efficacious. The end result of a lack of reasonable regulation being the appearance that these practices as a whole are a danger to the public, which is not a scientific judgment in the least, but more a means of manipulation of public sentiment.

Lack of reasonable regulation also means that products and equipment that is sold and marketed as Rife technology may be nothing of the sort. There are many false Rife machines, and the promoters of these fall into a spectrum of classifications from outright frauds to the genuinely mistaken. It is also quite common for false Rife machines to adopt all the same words and phrases as are used to describe genuine Rife instruments, making it impossible even for experienced users to determine either the hardware value or efficacy as opposed to price.

Dosages, amplitudes, waveforms, frequencies and session protocols all are without labeling requirements, acceptable standards, or authoritative training. This means that all of these critical factors are subject to arbitrary opinion and must certainly fall under the US House of Representatives definition of quackery, whether they are genuine or false machines and/or practitioners.

Further mass confusion concerning the Rife technology stems from the very fact of its basic truths. The results possible from the use of a very feeble machine is often quite impressive the first time it is used. This is similar to many drugs, where the maximum notable effect occurs upon the first use. The long-term effectiveness of the drug may taper off dramatically thereafter. Such a feeble machine does not display its diminished result and this is not evident to the user until after they have made a purchase. Feeble machines may be adequate to prove the theory, even in a scientific environment, but still fall short of having any real therapeutic or medicinal value. This one time only effect has been enough to convince even the most ardent skeptic and astute scientists and researchers should be aware that these effects are common and misleading.

Rife machines are further stigmatized by a lack of approval compared to the perceived credibility and public acceptance of products that are approved and widely marketed in advertising media. It is the opinion of many drug examiners and approval preparers for drug companies that the present fast track approval process is even more a danger to the public than any snake oil, as evidenced by the mass recalls and withdrawals from the market of well known drugs. In these cases, it is obvious that the approval process is seriously compromised and wholly inadequate. Unlike the use of transducers and Rife machines, which have been in use for as much as a hundred years, new,

unproven drugs are approved and placed on the market in as little as 5 months. It is simple logic to realize that until a product has been tested or used for at least 50 years, its long term side effects simply cannot be determined.

There is real doubt of the value of the official approval of Rife machines. Hostile regulation is probably not better than no regulation at all. The motive for regulation is lacking since it is generally accepted that the technology is grandfathered and therefore exempt from conventional regulation. It is subject to fair trade and truthful advertising, however, and The Federal Trade Commission suggests that the technology is not proven. This claim is arguable, since it has been proven many times in many countries, and it is very easily proven when challenged to do so. Such a challenge is not expected, because it is widely understood that the true Rife technique is massively effective, a fact that some of its opponents would prefer did not become public or common knowledge.

Frequently Asked Questions

Here you will find a collection of the most common questions regarding Rife, the machines and the therapy. They are presented in no particular order.

Why haven't I heard about Rife before?

There are a number of theories as to exactly what has suppressed the technology over the years. Most of them are exaggerated, glamorized, or simply untrue. What is true is that some people loose track of what they can say about their machines without getting into legal trouble. Others leap to conclusions about what the machines can really do. Occasionally, they run amuck of the law, and then claim they are being treated unfairly, or are being persecuted. When this happens they become overly cautious. So, the answer to this question is that users and manufacturers as well, have developed a tradition of not telling people about it, even when the need is great, purely out of a need for self preservation. Therefore, if you already know about it, it will not be deliberately hidden from you, but if you do not know about it, and do not seek it out, nobody is going to come knocking at your door, or send you unsolicited email. This keeps the profile down. Conventional medicine and other big business are not interested in the technology for the reason that they do not want to invest a lot of capital in a technology that they cannot patent, and cannot exclusively control. In a few cases, however, businessmen and industry have taken a clue from Rife technology, and developed their own, unique interpretations, which can be patented. The TENS unit, which has been approved by the FDA for many years, the bone growth stimulator, also approved are examples of Rife machines that you may have heard about. The Cybersonic, Ionic, Sonic, and other types of advanced tooth brushes are based on fundamental insights taken directly from the Rife technology. Outside of the US, there are many countries that have embraced the advent of frequency therapy. It seems to fall in and out of favor, depending on the administration or regime. This is probably because the technology, if it is done correctly, is fantastically effective, and tends to inhibit some sectors of the medical profession. The drug business is definitely the most obvious of these, but it affects every part of medicine to greater or to lesser degrees. Politics plays a role in the suppression of Rife machines.

Will it work for me?

It depends, in part, on what you are trying to accomplish with the machine. It also depends on whether or not the machine you get is able to do that job, since not all machines are equal. If you expect a limited function device, such as a zapper, or a tens unit to perform as a full function device, you may be disappointed.

There are profound differences between a broadcast device and a transducer, as there are distinct differences between an analog device and a digital device. Of these, only a very few are capable of everything a Rife machine is expected to do.

If the problem you have is something that is incurable, the Rife machine may be your best bet. There are a number of afflictions that can only be treated successfully with a full function instrument. But if all you expect is daily maintenance, lesser machines may do the job quite well. Unfortunately, you might spend thousands of dollars on an instrument that is not able to handle your particular problem, and yet, a simpler and much cheaper device may do the job easily.

Your success with any home remedy may depend on an accurate diagnosis. Once you have that, it helps a whole lot to be well educated about the nature of the problem, what causes it, what the symptoms are, and how it is normally treated.

Where you are located, where you grew up, and your personal habits often determine how difficult it might be to treat a given problem successfully. People who live in big cities often do not respond as well to the therapy as do those who live in remote or isolated regions. So the environment and your living conditions play an important part. This is true for many types of remedy, including the conventional allopathic approach.

Complications to a simple problem, underlying, disrelated problems, especially when they are located in the same general part of the body, contribute to ambiguities in the results you realize. If you contract an infection in unusual way, even if it is a very common infection, might make it very difficult to deal with as compared to the average case. A history of drug use, prescription or otherwise, alcohol, or steroids, usually reduces the chances or slows down your ability to recover, especially if traditional medicine has failed. Very advanced or very long term problems usually do not respond as well as newly contracted or untreated problems of the same type. Knowing exactly why the problem exists helps a great deal.

Conventional testing is the most common way users confirm their results. The therapy should not be used to replace most straight forward medical treatment. Be sure to see a doctor, or a specialist, and be sure to get copies of any and all records, test results, and X-rays that they routinely keep. The more you know and the more information you have that you can use to compare your problem with recorded cases, the better equipped you are to deal with a problem yourself.

There are very few users who do not do better with a JWLABS machine than they would have without it. Although we are not doctors, we will do everything we know how, to help you. One of the most important things that you can do to assure your success, regardless of the remedy, is to follow the instructions strictly and regularly.

If you use microscopy, or any other testing method, conventional or otherwise, the first thing you will be struck by is the fact that unlike most other therapies, the Rife machine starts working immediately. Amazing changes happen within seconds. Long before the changes are noticeable to you, marked, scientifically observable changes will happen in virtually every user.

This doesn't mean that it will cure you that fast. Normally it takes some time just to become accustomed to the effects that are produced. One case in a hundred does not respond as expected, or not at all. But by carefully examining the facts of the case and the circumstances, the symptoms and other complications, it is usually possible to determine with some degree of reliability, the reason why. If you can figure out why, often there is a way to make it work.

In the treatment of such things as gangrene, usually there is only time for a single treatment. It has to be done at high power, at the right frequencies, for extended session duration. Life and limb have been saved in more than one case, as reported. Influenza can be treated, but prevention by means of regular maintenance sessions is the preferred method, due to the potential for Herxheimenr (healing crisis) type reactions. Used in general healing for wounds and broken bones, accelerated healing about twice as fast as normal are typical reports in testimonials. Cancer responds according to the type. Some reports indicate that fast growing, terminal, inoperable tumors have been all but eliminated in 2 weeks. Thickly encapsulated, slow growing, benign tumors may not respond at all. There seems to be no reliable rules in the treatment of cancer. The machine does not kill human cells, and so it does not kill human cancer cells. The treatment is expected to remove the cause of the cancer, unless it is caused by a toxin, asbestos, or radiation. Removal of the cause, will usually bring remission in advanced cases, and may bring a complete cure in less advanced cases. Nothing works if it is simply too late and the systems of the body are already breaking down, as is observed in general systemic failure just prior to the death spiral.

Multiple Sclerosis and Fibromyalgia cases cover the full range from no results, to complete cessation of all symptoms. This has been done in as little as three months, and as long as two years.

In general, the machine works very fast. So fast, that the first few sessions are critical. With an analog, full function, double power instrument such as the ones we make, the first two or three sessions must be done with extreme caution, so as to avoid extreme reactions. Once this danger is past, regular sessions may begin. With lesser machines, the first few sessions may be the only time results are seen. Many people, new users and experienced practitioners alike have been confounded by the vast differences in the efficacy of different makes of Rife instrument and how difficult it is to tell which are good for a lifetime of therapy, and which will only work for a week or two.

How dangerous is Rife therapy?

It is best to use the analogy of the automobile, since most people are familiar with them. As a non-user, you have either been walking, or riding your bike. Now you are about to climb into a Ferrari F50, and get out onto a full competition race track. In other words, it is only as dangerous as you are. If you are a foolhardy know-it-all, or have declared yourself an "instant expert" you may very well kill yourself. But you are still going to have to do some very stupid things before you die.

If you are appropriately cautious and take the time to fully understand what you are doing before you put your foot to the floor, you will be fine. But in order for a car to take you down the highway, it must also have the power to take you over a cliff.

Injuries using the transducer are extremely rare however, for a number of important reasons. Obviously, if the machine you have is only capable of treating a quarter of a cubic inch at a time, there is no danger, because the machine is not powerful enough to hurt you. It is also not powerful enough to accomplish much except perhaps to prove that the technology works, even in its most ineffective forms. By contrast, both Model A and Model B have enough power to treat a horse, and can deliver enough current to thoroughly permeate every cell in your body. These devices are also sensitive enough to treat a mouse. Obviously, what is needed to treat the body lies somewhere inbetween.

One of the built in factors of a transducer that helps prevent injuries is that in order to hurt yourself, you would have to apply enough power to cause pain. So, for the same reason that you never use so much volume when using a headphone that it causes pain and thereby avoid doing harm to your hearing; you will avoid causing pain with the machine. Still, it is possible to do very dangerous things with the machine. For instance, if you were to place the electrodes of the machine over your temples, turn the machine up to maximum, and then switch the machine on, you could do serious harm to yourself. This and other potentially harmful mistakes are things that we are very aware of, and we always like to walk new users through their first few sessions so that we can be sure they get a reasonable understanding of the proper use of the machine.

Without question, the single most prominent of all dangers of the machines we make is the hazard of over use. Although not much of a problem for experienced users, the potential for over use among new users is something that we stress. The machine is not like a drug. It isn't something that you can over dose yourself using. It isn't putting new chemicals into your body and polluting it. Rather, it is going to continue to make changes to the chemicals already in your body, called endergonic reactions. It will continue to do this for as long as you keep using it. Although we always tell people that it is not recommended for systemic sessions more than 30 minutes every three days, many people exceed this. The reason they do, is either they did not believe what we warned them about, or they simply forget how much they have used it. Consider that a completely healthy person might use the machine everyday for an hour day after day, but after about ten days, it will hit them suddenly, and they will develop severe toxic reactions.

For new users, there is a real possibility of undiagnosed infection, which in the extreme case, can have the same effect in a matter of minutes, literally after a single session. The methods we use to prevent this from happening and the sort of sessions that we suggest in the beginning are tailored with this in mind.

If we have scared you, relax. When used properly, the machine is far less dangerous than a hair dryer, a toaster, or an electric razor. It is safe, but not recommended for children. Under the age of 13 must be with adult supervision. Other, more common cautions are included in the operating instruction booklet that comes with the machine. These cautions are covered with each customer verbally before they receive their machine. It is routine for us to interview each customer, to determine if there are any special dangers for that individual, based on their history.

Will it make my problems worse?

No. The machine will not make matters worse. Indirectly, however it is not a good idea to use the machine instead of having the sort of care your doctor suggests. Like using it to avoid a surgical procedure that you need.

For people suffering from viral infections like HIV, or hepatitis, it is best to refrain from using the machine for at least a few days before a viral load test, because using the machine will stir up pathogens, and other measurable blood factors, which is a normal effect of the device that will throw off the results of most tests of this kind. White cells are also brought out, and become much more active. Shattered virus may not be distinguishable from live virus. Increased antigens may appear to indicate changes that are not really correct. This could make it look a lot worse than it really is, and cause the doctor to make incorrect judgments about your condition. You will feel fine, but your doctor may wonder how it is that you are even walking around.

If tests are the means by which you are measuring your progress, just take leave of the machine for three to five days. The debris in your system will have time to clear out completely, and the test will reflect more accurately your true condition.

How do you avoid the FDA?

We don't. And neither should you. We cannot give legal advice, but it is our opinion that so long as what you are doing does not infringe on the domains of conventional medicine, and you are not misleading people in your presentations, you should have nothing to fear of the FDA

How can I use this in my medical practice?

Most conventional doctors can't. They are constrained by the rules of the AMA, and other local and state laws, which pretty much forbid anything that is not approved.

Other types of practices may be far less restricted however, and the technology is allowed in most other countries.

For those not in the medical field, there are no restrictions, but what you say about the therapy, and how it is presented, makes all the difference. If you have any doubts at all, the Model A was designed with this problem in mind. Model A may be used in a very special way that is non medical, and can be used and presented as entertainment.

Does the US government know about Rife machines?

Absolutely. And, it is fairly well known around the world. We gave the technology to the Department of Defense back in 1989. They responded very positively. But don't worry. Their interest was primarily in the medical aspects of the technology. It does not lend itself to being made into a weapon. Rife technology, in its true form, does not use any frequencies that can be harmful to the body or that have the capacity to kill human cells.

Go to our **TECH STUFF** link, for more technical information about the basic physics of Rife machines.

Rife used the frequency Instrument (the transducer) for therapy, which by design cannot kill human cells.

Tech Stuff - JWLABS technology

This is by no means intended to be a technical manual on Rife technology. The purpose of this page is to attempt to explain in simple terms, some of the questions laypersons rarely know to ask. For more common questions, please visit our Rife FAQ.

Probably the main issue we should discuss has to do with value versus hardware. Rife machine hardware, meaning the components that go into it, needs to be adequate to do the job, but nothing should be added to the machine that is

not actually required. So you will never see meaningless modalities on any JWLABS instrument that are intended merely to impress or to make the machine appear to have more value than it actually has. There are no exotic applicators that have any more valid efficacy, than those provided.

An examination of the long evolution of applicator technology will reveal that the means of delivering current to the body has gone through many changes over the one hundred year history of the technology.

Until medical quality electrode patches were invented, the means of application were crude and fairly limited. For the first fifty years, steel plates for the feet to stand on, and metal rods for the hands to grip, were about as good as could be expected. Though horribly inadequate, this was what Rife used from the earliest days. But these were only one step ahead of attaching wires to bolts installed in your neck!

Even today, would be Rife manufacturers use the rods and plates, and although it is one of the authentic ways, this method has been obsolete for many years.

There are various other experimental applicators, but these are mostly for show and do not actually add anything new to the therapy. So, it is a waste of money, if you pay more just for that.

The machine that uses less hardware, without sacrificing any of the output quality, makes it possible for more people to enjoy the benefits, because this makes the same therapy less expensive. This is not very easily done. Although we have finally achieved this with our Model A, it has taken decades to develop and test, and the machine has had to go through many different embodiments at great cost. We are satisfied now; that our new Model A can do everything our Model B can do therapeutically.

Another technical issue has to do with analog versus digital. Digital accuracy is certainly greater than is possible with analog, but it is the wrong sort of accuracy. The minor variations that are characteristic of an analog device must be simulated digitally, using still more complex hardware, in order for it to approximate analog. Of course, Rife never used anything digital, and the frequency tuning of most digital machines leaves a great deal to be desired. Unless a digital machine employs very complex programs, there are many frequencies that it will simply never be able to achieve properly, because it is effectively impossible to digitally simulate the perfectly smooth gradients of tuning that are inherent to an analog device of far less complexity.

In other words, if an analog device can split a single hertz into billionths, simply by turning a dial, a single hertz simulated at that level digitally would require at least a gigabyte of information. Multiply that by the ten thousand hertz that are traditionally used in the therapy, would require ten terabytes or about six modern PCs computers, loaded to the gills, to equal it digitally. Not impossible, but still a lot more than is feasible or affordable with the present state of digital programming technology. Again, this is easily achieved in analog by means of a series of simple potentiometers, albeit not cheap ones.

A common issue has to do with output power. There are limits to the amount of current energy the body can comfortably tolerate without harm. It has long been known that lower frequencies require far less energy to deliver effectively, because the amount of deliverable frequency power, or signal, than can be delivered drops off exponentially the higher the frequency that is used. For this and for other reasons, the lower frequencies are proportionally safer than higher ranges because they require far less electrical potential to drive them.

Frequency, harmonics, and resonance are often brought up as arguable points. Harmonics and resonances are not generated by digital technology, so digital will not be considered in this argument.

Rife discovered the importance of frequency when he found that microorganisms can be viewed in their native colors. He did this through the use of a series of prisms, and arrived at a system that could polarize the light spectrum for this purpose. In the strictest sense, these are the true native frequencies of the various microorganisms. But the frequencies of light can only be easily expressed by scientific notation, because they are so high. When he exposed them to the precise frequencies of light under the microscope, obviously, the microbes did not die, because the amount of deliverable energy by means of light is insignificant and far below the level that is needed to destroy them. So, he

was forced by practicality, to translate these light frequencies down the spectrum, to lower frequency ranges, so that enough energy could be safely delivered.

One of the ranges that will do the job is the radio transmission range. These are frequencies measured in megahertz, or millions of cycles per second. This range turned out to be ideal for treatment of the microscope, and will only destroy microbes when the precise frequency is achieved.

The big drawback of using radio waves to kill pathogens is that microwave begins as low as 33 megahertz. Unshielded microwave energy, obviously, is not desirable in any therapy instrument for the reason that it will kill off human cells just as readily as it can kill germs. Human brain cells are especially susceptible, as are certain nerves and tissues of the eye.

So, in order to make an instrument that will not do the user any harm, only the audio range is safe. The audio range is about the same as that of a piano. You can listen to a piano all day long and it will not harm you in the slightest way, in fact, music has other plainly observable therapeutic value all by its self.

The resonances of a signal are really overtones and undertones, just as they are observable on the piano. When you hit middle C, both high C and low C resonate. It is by this means that the mortal oscillatory rates actually work. Audio does not use the native frequencies of a microbe; they employ the resonances that are sympathetically generated. But resonance does not occur in the machine, they occur in the body. Also the same in a piano, it is not middle C that resonates when you press it, it is the other components of the piano that resonate.

Harmonics is what happens when you hit two piano keys at once. You do not get either frequency; instead, you get the harmonic frequency that is generated by the combined tones. For this reason, multiple signal generators are nonsense. If you introduce two different signals into the body, you will only get the harmonics they create, and thus you will never know what the effective frequency actually is that you are getting. In the worst case, the harmonics will reach frequencies approaching microwave.

Waveform. There are some very simple reasons why the only waveform a true Rife machine generates is a square wave. Introducing frequency into the body is like trying to play the piano underwater. The signal will be muted and sound very muffled. This is because as the waves of energy pass through the water, they are eroded by the natural kinetic resistance of the water. The Rife machine has a much more difficult medium to penetrate, and it is far more difficult to deliver the correct, coherent signal to the deepest tissues. The body has a lot of resistance and muffles the signal very rapidly. Literally, the signal flattens out the farther it travels in the medium of the body. In order to compensate for this, the square wave is used because it has the most total signal and so will maintain the frequency much farther through the medium than any other type of signal. The argument is that Rife used a sine wave. That is more correct than is immediately apparent. The instant a square wave enters the body; it is converted by the resistance of the body, into a sine wave. If you introduce a sine wave, it instantly goes flat, and does not deliver any frequency information/potential at all. This is true with all other waveforms as well, except a square wave.

We hope this clears up some of the technical questions you may have.

To contact Wright Laboratories call their Custom Service Department: **1-888-891-1122**

Before we get to the science portion of this report I would like to make an observation and a statement.

What drives me really crazy is when many women are having their breasts removed as a preventive because breast cancer runs in the family or they have the **"CANCER GENE".** Or if they have cancer in one breast to play it safe they are also having the other breast removed.

In my professional opinion that is absolutely absurd. Why do I say this??? Let me give you a couple of examples:

Let's say you have a flame near a piece of wood. Under normal circumstances the wood would be a little dry so when the flame is near enough to the wood it could catch on fire. If you kept the wood saturated with water it would make it nearly impossible for the wood to catch on fire, wouldn't it. Basically, by providing an environment that is not suitable for a fire the wood would not burn.

Here is another example: Mold. Sometimes no matter how you dry the shower walls, mold finds that little damp spot and starts to grow. Now since the bathroom is part of your house why isn't the mold growing in the rest of your house? Why, because the rest of the house is dry and is not providing a suitable environment for the mold to grow.

If we take the protocol of the medical profession, the first thing to do is to either remove the wall in the bathroom that has the mold or remove the entire bathroom and then put chemicals in the entire house to kill mold that may not even be there while you are still in the house.

By providing a clean and slightly alkaline environment in your blood and around your cells you would be providing an environment that would make it very difficult, if not impossible, for any cancer to grow. I am making this statement based on the findings of Nobel Prize winner Dr. Otto Warburg and the discoveries of Dr. Arthur C. Guyton and Dr. H.S. Mayerson. If you recall, I mentioned these gentleman earlier in this report.

So PLEASE, unless your doctor tells you that you are going to die within the week if you do not remove or have something cut out. Learn as much as you can before making that drastic of a decision.

This report is providing the science and information to give you a head start in taking your life/health back. Plus, there is so much more information on the Internet for your review/research.

On the other hand, if you decide that you do not want to change your lifestyle/eating habits that helped in the cancer development, then by all means, do what you have to do to save your life.

The Science

For thousands of years Chinese and Ayurvedic medicine has helped countless individuals. I guess based on the extended history of these two alternative forms of health that the scientific community decided to do some research for themselves.

I have selected seven foods for this report to see what the scientific community has found.

Amalaki (Indian Gooseberry) (Emblica officinalis)
Heritaki (Terminalia chebula)
Tulsi (Holy Basil) (Ocimum sanctum)
JuJube (Ziziphus jujuba Mill)
Ginger, (Zingiber officinale)
Schizandra (Schizandra chinensis)
and Turmeric (Curcuma longa)

Below, you will find a compilation of abstracts of the studies that I found at www.PubMed.gov.

These studies are presented here for educational purposes only.

Amalaki Fruit (Emblica Officinalis)

Yakugaku Zasshi. 2007 Oct;127(10):1701-7.
Effect of Anwala churna (Emblica officinalis GAERTN.): an ayurvedic preparation on memory deficit rats.
Vasudevan M, Parle M.

Department of Pharmacology, Nandha College of Pharmacy, Koorapalayam Pirivu, Pitchandampalayam, Erode-District, Tamilnadu-638052, India. vasumpharmacol@yahoo.co.uk

The present study was aimed at investigating the effects of Anwala churna (Emblica officinalis GAERTN.), an Ayurvedic preparation, on memory in rats. Anwala churna was administered orally in three doses (50, 100 and 200 mg/kg) for 15 days to different groups of young and aged rats. The elevated plus-maze and Hebb-Williams maze served as exteroceptive behavioral models for testing memory. Diazepam-, scopolamine-, and ageing induced amnesia served as the interoceptive behavioral models. Anwala churna (50, 100, and 200 mg/kg, p.o.) produced a dose-dependent improvement in memory scores of young and aged rats. Furthermore, it reversed the amnesia induced by scopolamine (0.4 mg/kg, i.p.) and diazepam (1 mg/kg, i.p.). Based on these results, Anwala churna may prove to be a useful remedy for the management of Alzheimer's disease due to its multifarious beneficial effects such as memory improvement and reversal of memory deficits. PMID: 17917427 [PubMed - indexed for MEDLINE]

J Ethnopharmacol. 1996 Feb;50(2):61-8.
Hypolipidaemic effect of fruit juice of Emblica officinalis in cholesterol-fed rabbits.
Mathur R, Sharma A, Dixit VP, Varma M.

Department of Home Science (Food and Nutrition), University of Rajasthan, Jaipur, India.

The lipid lowering and antiatherosclerotic effects of Emblica officinalis (Amla) fresh juice were evaluated in cholesterol-fed rabbits (rendered hyperlipidaemic by atherogenic diet and cholesterol feeding). E. officinalis fresh juice was administered at a dose of 5 ml/kg body weight per rabbit per day for 60 days. Serum cholesterol, TG, phospholipid and LDL levels were lowered by 82%, 66%, 77% and 90%, respectively. **Similarly, the tissue lipid levels showed a significant reduction following E. officinalis juice administration.** Aortic plaques were regressed. E. officinalis juice treated rabbits excreted more cholesterol and phospholipids, suggesting that the mode of absorption was affected. E. officinalis juice is an effective hypolipidaemic agent and can be used as a pharmaceutical tool in hyperlipidaemic subjects. PMID: 8866725 [PubMed - indexed for MEDLINE]

J Nutr Sci Vitaminol (Tokyo). 2005 Dec;51(6):413-8.
Influence of amla (Emblica officinalis Gaertn.) on hypercholesterolemia and lipid peroxidation in cholesterol-fed rats.
Kim HJ, Yokozawa T, Kim HY, Tohda C, Rao TP, Juneja LR.

Institute of Natural Medicine, Toyama Medical and Pharmaceutical University, 2630 Sugitani, Toyama 930-0194, Japan.

The effects of amla on low-density lipoprotein (LDL) oxidation and cholesterol levels were investigated in vitro and in vivo using Cu(2+)-induced LDL oxidation and cholesterol-fed rats. SunAmla and ethyl acetate (EtOAc) extract of amla significantly inhibited thiobarbituric acid (TBA)-reactive substance level in the Cu(2+)-induced LDL oxidation and the effects were stronger than those of probucol. In addition, the administration of SunAmla (at a dose of 20 or 40 mg/kg body weight/d) or EtOAc extract of amla (at a dose of 10 or 20 mg/kg body weight/d) for 20 d to rats fed 1% cholesterol diet significantly reduced total, free and LDL-cholesterol levels in a dose-dependent manner, and EtOAc extract of amla exhibited more potent serum cholesterol-lowering effect than SunAmla in the same amount. Furthermore, the oxidized LDL level in serum was markedly elevated in cholesterol-fed control rats as compared with normal rats, while it was significantly decreased by the administration of SunAmla or EtOAc extract of amla. Moreover, the serum TBA-reactive substance level was also significantly decreased after oral administration of SunAmla or EtOAc extract of amla. **These results suggest that amla may be effective for hypercholesterolemia and prevention of atherosclerosis.** PMID: 16521700 [PubMed - indexed for MEDLINE]

J Med Food. 2005 Fall;8(3):362-8.
Amla (Emblica officinalis Gaertn.) extracts reduce oxidative stress in streptozotocin-induced diabetic rats.
Rao TP, Sakaguchi N, Juneja LR, Wada E, Yokozawa T.

Bio-nutrition Division, Taiyo Kagaku Co. Ltd., Yokkaichi, Mie, Japan. tprao@taiyokagaku.co.jp

The antioxidant properties of amla extracts and their effects on the oxidative stress in streptozotocin-induced diabetes were examined in rats. Amla in the form of either the commercial enzymatic extract SunAmla (Taiyo Kagaku Co. Ltd., Yokkaichi, Japan) (20 or 40 mg/kg of body weight/day) or a polyphenol-rich fraction of ethyl acetate extract (10 or 20 mg/kg of body weight/day) was given orally for 20 days to the streptozotocin-induced diabetic rats. Amla extracts showed strong free radical scavenging activity. Amla also showed strong inhibition of the production of advanced glycosylated end products. The oral administration of amla extracts to the diabetic rats slightly improved body weight gain and also significantly alleviated various oxidative stress indices of the serum of the diabetic rats. The elevated serum levels of 5-hydroxymethylfurfural, which is a glycosylated protein that is an indicator of oxidative stress, were significantly reduced dose-dependently in the diabetic rats fed amla. Similarly, the serum level of creatinine, yet another oxidative stress parameter, was also reduced. Furthermore, thiobarbituric acid-reactive substances levels were significantly reduced with amla, indicating a reduction in lipid peroxidation. In addition, the decreased albumin levels in the diabetic rats were significantly improved with amla. Amla also significantly improved the serum adiponectin levels**. These results form the scientific basis supporting the efficacy of amla for relieving the oxidative stress and improving glucose metabolism in diabetes.** PMID: 16176148 PubMed - indexed for MEDLINE]

J Agric Food Chem. 2007 Sep 19;55(19):7744-52. Epub 2007 Aug 23.
Amla (Emblica officinalis Gaertn.) attenuates age-related renal dysfunction by oxidative stress.
Yokozawa T, Kim HY, Kim HJ, Tanaka T, Sugino H, Okubo T, Chu DC, Juneja LR.

Institute of Natural Medicine, University of Toyama, 2630 Sugitani, Toyama 930-0194, Japan. yokozawa@inm.u-toyama.ac.jp

To investigate the effects of amla on renal dysfunction involved in oxidative stress during the aging process, we employed young (2 months old) and aged (13 months old) male rats and administered SunAmla (Taiyo Kagaku Co., Ltd., Japan) or an ethyl acetate (EtOAc) extract of amla, a polyphenol-rich fraction, at a dose of 40 or 10 mg/kg body

weight/day for 100 days. The administration of SunAmla or EtOAc extract of amla reduced the elevated levels of serum creatinine and urea nitrogen in the aged rats. In addition, the tail arterial blood pressure was markedly elevated in aged control rats as compared with young rats, while the systolic blood pressure was significantly decreased by the administration of SunAmla or EtOAc extract of amla. Furthermore, the oral administration of SunAmla or EtOAc extract of amla significantly reduced thiobarbituric acid-reactive substance levels of serum, renal homogenate, and mitochondria in aged rats, suggesting that amla would ameliorate oxidative stress under aging. The increases of inducible nitric oxide synthase (iNOS) and cyclooxygenase (COX)-2 expression in the aorta of aging rats were also significantly suppressed by SunAmla extract or EtOAc extract of amla, respectively. Moreover, the elevated expression level of bax, a proapoptotic protein, was significantly decreased after oral administration of SunAmla or EtOAc extract of amla. However, the level of bcl-2, an antiapoptotic protein, did not show any difference among the groups. The expressions of renal nuclear factor-kappaB (NF-kappaB), inhibitory kappaB in cytoplasm, iNOS, and COX-2 protein levels were also increased with aging. However, SunAmla or EtOAc extract of amla reduced the iNOS and COX-2 expression levels by inhibiting NF-kappaB activation in the aged rats. **These results indicate that amla would be a very useful antioxidant for the prevention of age-related renal disease**. PMID: 17715896 [PubMed - indexed for MEDLINE]

Physiol Behav. 2007 May 16;91(1):46-54. Epub 2007 Feb 8.
Memory enhancing activity of Anwala churna (Emblica officinalis Gaertn.): an Ayurvedic preparation.
Vasudevan M, Parle M.

Pharmacology Division, Department of Pharmaceutical Sciences, Post Box - 38, Guru Jambheshwar University of Science and Technology, Hisar, (Haryana) -125 001, India.

Ayurveda means "the science of life". Ayur means "life" and Veda means "knowledge or science". It is the oldest medical system in the world. Its origins can be traced as far back as 4500 BC, to four ancient books of knowledge, (the "Vedas") and it is still officially recognized by the government of India. The present study was aimed at investigating the effects of Anwala churna (Emblica officinalis Gaertn.), an Ayurvedic preparation on memory, total serum cholesterol levels and brain cholinesterase activity in mice. Anwala churna was administered orally in three doses (50, 100 and 200 mg/kg) for fifteen days to different groups of young and aged mice. Elevated plus maze and passive avoidance apparatus served as the exteroceptive behavioral models for testing memory. Diazepam-, scopolamine- and ageing-induced amnesia served as the interoceptive behavioral models. Total serum cholesterol levels and brain cholinesterase activity also estimated. Anwala churna (50, 100 and 200 mg/kg, p.o.) produced a dose-dependent improvement in memory scores of young and aged mice. Furthermore, it reversed the amnesia induced by scopolamine (0.4 mg/kg, i.p.) and diazepam (1 mg/kg, i.p.). Interestingly, brain cholinesterase activity and total cholesterol levels were reduced by Anwala churna administered orally for 15 days. **Anwala churna may prove to be a useful remedy for the management of Alzheimer's disease on account of its multifarious beneficial effects such as, memory improving property, cholesterol lowering property and anticholinesterase activity.** PMID: 17343883 [PubMed - indexed for MEDLINE]

Phytomedicine. 2002 Mar;9(2):171-4.
Effect of bioactive tannoid principles of Emblica officinalis on ischemia-reperfusion-induced oxidative stress in rat heart.
Bhattacharya SK, Bhattacharya A, Sairam K, Ghosal S.

The tannoid principles of the fruits of Emblica officinalis have been reported to exhibit antioxidant activity in vitro and in vivo. In the present study, an emblicanin-A (37%) and -B (33%) enriched fraction of fresh juice of Emblica fruits (EOT) was investigated for antioxidant activity against ischemia-reperfusion (IRI)-induced oxidative stress in rat heart. Vitamin E (VE) was used as the standard antioxidant agent. IRI was induced in isolated rat heart by perfusing it with modified Kreb-Hensleitt's solution for 5 min, followed by a period of ischemia (stoppage of perfusion) for 10 min and then restoring the perfusion (reperfusion) for 15 min. IRI induced a significant decrease in the activities of cardiac superoxide dismutase, catalase and glutathione peroxidase, with a concomitant increase in lipid peroxidation. These IRI-induced effects were prevented by the administration of EOT (50 and 100 mg/kg body wt.) and VE (200 mg/kg body wt.) given orally twice daily for 14 days prior to the sacrifice of the animals and initiation of the perfusion

experiments**. The study confirms the antioxidant effect of E. officinalis and indicates that the fruits of the plant may have a cardioprotective effect**. PMID: 11995952 [PubMed - indexed for MEDLINE]

Skin Pharmacol Appl Skin Physiol. 2002 Sep-Oct;15(5):374-80.
Emblica cascading antioxidant: a novel natural skin care ingredient.
Chaudhuri RK.

Rona/EM Industries, Inc., Hawthorne, NY 10532, USA. rchaudhuri@emindustries.com

A standardized extract of Phyllanthus emblica (trade named Emblica) was found to have a long-lasting and broad-spectrum antioxidant activity. The product has no pro-oxidation activity induced by iron and/or copper because of its iron and copper chelating ability**. Emblica helps protect the skin from the damaging effects of free radicals, non-radicals and transition metal-induced oxidative stress. Emblica is suitable for use in anti-aging, sunscreen and general purpose skin care products.** Copyright 2002 S. Karger AG, Basel PMID: 12239434 [PubMed - indexed for MEDLINE]

J Ethnopharmacol. 1998 Sep;62(2):183-93.
Screening of some Indian medicinal plants for their antimicrobial properties.
Ahmad I, Mehmood Z, Mohammad F.

Department of Agricultural Microbiology, Institute of Agriculture, Aligarh Muslim University, India.

A total of 82 Indian medicinal plants traditionally used in medicines were subjected to preliminary antibacterial screening against several pathogenic and opportunistic microorganisms. Aqueous, hexane and alcoholic extracts of each plant were tested for **their antibacterial activity** using agar well diffusion method at sample concentration of 200 mg/ml. The results indicated that out of 82 plants, 56 exhibited antibacterial activity against one or more test pathogens. Interestingly, extracts of five plants showed strong and broad spectrum activity as compared to rest of 51 plant extracts which demonstrated moderate activity. On the whole the alcoholic extracts showed greater activity than their corresponding aqueous and hexane extracts. Among various extracts, only alcoholic extracts of **Emblica officinalis, Terminalia chebula**, Terminalia belerica, Plumbago zeylanica and Holarrhena antidysenterica **were found to show potentially interesting activity against test bacteria. These active crude alcoholic extracts were also assayed for cellular toxicity to fresh sheep erythrocytes and found to have no cellular toxicity**. PMID: 9741890 [PubMed - indexed for MEDLINE]

Mol Vis. 2004 Mar 12;10:148-54.
Inhibition of aldose reductase by tannoid principles of Emblica officinalis: implications for the prevention of sugar cataract.
Suryanarayana P, Kumar PA, Saraswat M, Petrash JM, Reddy GB.

Biochemistry Division, National Institute of Nutrition, Hyderabad, India.

PURPOSE:**Aldose reductase (AR) has been a drug target because of its involvement in the development of secondary complications of diabetes including cataract**. Although numerous synthetic AR inhibitors (ARI) have been tested and shown to inhibit the enzyme, clinically synthetic ARIs have not been very successful. Therefore, evaluating natural sources for ARI potential may lead to the development of safer and more effective agents against diabetic complications. In the present study we have assessed the inhibition of AR by constituents of Emblica officinalis both in vitro and in lens organ culture. METHODS: E. officinalis is widely used against many chronic ailments including diabetes. Aqeous extract of E. officinalis and its major constituent tannoids were tested for inhibition against both rat lens and purified recombinant human AR. ARI potential of isolated tannoids of E. officinalis were also investigated against osmotic stress in rat lens organ culture. RESULTS: E. officinalis extract inhibited rat lens and recombinant human AR with IC50 values 0.72 and 0.88 mg/ml respectively. Since E. officinalis is a rich source of ascorbic acid, we investigated whether ascorbic acid was responsible for AR inhibition by E. officinalis extract. However, ascorbic acid did not inhibit AR even at 5 mM concentration. Further, we demonstrate that the hydrolysable tannoids of E. officinalis were responsible for AR inhibition, as enriched tannoids of E. officinalis

exhibited remarkable inhibition against both rat lens and human AR with IC50 of 6 and 10 microg/ml respectively. The inhibition of AR by E. officinalis tannoids is 100 times higher than its aqueous extract and comparable to or better than quercetin. Furthermore, the isolated tannoids not only prevented the AR activation in rat lens organ culture but also sugar-induced osmotic changes. **CONCLUSIONS: These results indicate that tannoids of E. officinalis are potent inhibitors of AR and suggest that exploring the therapeutic value of natural ingredients that people can incorporate into everyday life may be an effective approach in the management of diabetic complications**. PMID: 15031705 [PubMed - indexed for MEDLINE]

Mol Vis. 2007 Jul 24;13:1291-7.
Emblica officinalis and its enriched tannoids delay streptozotocin-induced diabetic cataract in rats.
Suryanarayana P, Saraswat M, Petrash JM, Reddy GB.

Biochemistry Division, National Institute of Nutrition, Hyderabad, India.

PURPOSE: Aldose reductase (AR) has been a drug target because of its involvement in the development of secondary complications of diabetes including cataract. We have previously reported that the aqueous extract of Emblica officinalis and its constituent tannoids inhibit AR in vitro and prevent hyperglycemia-induced lens opacification in organ culture. The purpose of the current study was to investigate the effect of Emblica and its enriched tannoids on streptozotocin (STZ)-induced diabetic cataract in rats. METHODS: Diabetes was induced in Wistar-NIN rats by STZ (35 mg/kg body weight, intraperitoneally) and the animals were divided into three groups (Group II, III, and IV). The control rats (Group I) received only vehicle. While Group I and Group II animals received AIN-93 diet, rats in Groups III and IV received 0.2% of standardized mixture of Emblica tannoids and 2% of Emblica pericarp, respectively, in an AIN-93 diet for a period of eight weeks. Cataract progression due to hyperglycemia was monitored by slit-lamp biomicroscope and classified into four stages. At the end of the eight weeks, the animals were sacrificed and markers of the polyol pathway, oxidative stress, and alterations in protein content and crystallin profile in the lens were measured. Blood glucose and insulin levels were also determined. RESULTS: Both Emblica and its tannoids did not prevent STZ-induced hyperglycemia as assessed by blood glucose and insulin levels. However, slit lamp microscope observations indicated that these supplements delayed cataract progression. The present studies suggest that Emblica and its tannoids supplementation inhibited AR activity as well as sorbitol formation in the lens. The results also point out that Emblica and its tannoids might counter the polyol pathway-induced oxidative stress as there was a reversal of changes with respect to lipid peroxidation, protein carbonyl content, and activities of antioxidant enzymes. Emblica also prevented aggregation and insolubilization of lens proteins caused by hyperglycemia. **CONCLUSIONS: The results provide evidence that Emblica and an enriched fraction of Emblica tannoids are effective in delaying development of diabetic cataract in rats.** PMID: 17679931 [PubMed - indexed for MEDLINE]

Biomed Pharmacother. 2003 Sep;57(7):296-300.
Immunomodulatory effects of agents of plant origin.
Ganju L, Karan D, Chanda S, Srivastava KK, Sawhney RC, Selvamurthy W.

Immunomodulation Laboratory, Defence Institute of Physiology and Allied Sciences, Lucknow Road, Timarpur, Delhi 110054, India. lganju@rediffmail.com

The immunomodulatory properties of amla (Emblica officinalis) and shankhpushpi (Evolvulus alsinoides) were evaluated in adjuvant induced arthritic (AIA) rat model. Injecting Complete Freund's Adjuvant (CFA) in right hind paw of the animals induced inflammation. The crude extracts of both the herbs were administered intraperitonially following a repeated treatment profile. The anti-inflammatory response of both the extracts was determined by lymphocyte proliferation activity and histopathological severity of synovial hyperplasia. Both the extracts showed a marked reduction in inflammation and edema. At cellular level immunosuppression occurred during the early phase of the disease. There was mild synovial hyperplasia and infiltration of few mononuclear cells in amla or shankhpushpi treated animals. The induction of nitric oxide synthase (NOS) was significantly decreased in treated animals as compared to controls**. These observations suggest that both the herbal extracts caused**

immunosuppression in AIA rats, indicating that they may provide an alternative approach to the treatment of arthritis. PMID: 14499177 [PubMed - indexed for MEDLINE]

J Ethnopharmacol. 2002 Jun;81(1):5-10.
Cyto-protective and immunomodulating properties of Amla (Emblica officinalis) on lymphocytes: an in-vitro study.
Sai Ram M, Neetu D, Yogesh B, Anju B, Dipti P, Pauline T, Sharma SK, Sarada SK, Ilavazhagan G, Kumar D, Selvamurthy W.

Defence Institute of Physiology and Allied Sciences, Lucknow Road, Timarpur, New Delhi 110054, India. sairam64@rediffmail.com

The fruits extracts of Emblica officinalis (Amla) has been reported to have strong anti-oxidant properties. There is a paucity of studies on the immunomodulatory properties of fruit extracts of Amla in immuno-compromised states, with the emphasis on lymphocytes. Therefore, the aim of the study was to determine the anti-oxidant and immunomodulatory properties of Amla using chromium (VI) as an immunosuppressive agent. Chromium (Cr) treatment results in enhanced cytotoxicity, free radical production, lipid peroxidation and decreased glutathione peroxidase (GPx) activity and diminished glutathione (GSH) levels. There was a significant inhibition of both lipopolysaccharide and concanavalin-A-stimulated lymphocyte proliferation. Chromium also inhibited Con A stimulated interleukin-2 and gamma-interferon production significantly. Further, there was enhanced apoptosis and DNA fragmentation in the presence of Cr. Amla significantly inhibited Cr-induced free radical production and restored the anti-oxidant status back to control level. Amla also inhibited apoptosis and DNA fragmentation induced by Cr. Interestingly, **Amla relieved the immunosuppressive effects of Cr on lymphocyte proliferation and even restored the IL-2 and gamma-IFN production considerably**. PMID: 12020921 [PubMed - indexed for MEDLINE]

J Ethnopharmacol. 2004 Nov;95(1):83-5.
Evaluation of anti-pyretic and analgesic activity of Emblica officinalis Gaertn.
Perianayagam JB, Sharma SK, Joseph A, Christina AJ.

Pharmacognosy and Phytochemistry Division, Faculty of Pharmaceutical Sciences, Guru Jambheshwar University, Hisar-125001, India.

The present study was designed to investigate the anti-pyretic and analgesic activity of ethanol (EEO) and aqueous (AEO) extracts of Emblica officinalis fruits in several experimental models. A single oral dose of EEO and AEO (500 mg/kg, i.p.) showed significant reduction in brewer's yeast induced hyperthermia in rats. EEO and AEO also elicited pronounced inhibitory effect on acetic acid-induced writhing response in mice in the analgesic test. Both, EEO and AEO did not show any significant analgesic activity in the tail-immersion test**. These findings suggest that extracts of Emblica officinalis fruits possessed potent anti-pyretic and analgesic activity.** Preliminary phytochemical screening of the extracts showed the presence of alkaloids, tannins, phenolic compounds, carbohydrates and amino acids, which may be responsible for anti-pyretic and analgesic activities. PMID: 15374611 [PubMed - indexed for MEDLINE]

Heritaki (Terminalia chebula)

J Trace Elem Med Biol. 2006;20(4):233-9. Epub 2006 Oct 2.

Chemomodulatory effects of Terminalia chebula against nickel chloride induced oxidative stress and tumor promotion response in male Wistar rats.
Prasad L, Husain Khan T, Jahangir T, Sultana S.

Section of Chemoprevention and Nutrition Toxicology, Department of Medical Elementology and Toxicology, Jamia Hamdard (Hamdard University), Hamdard Nagar, New Delhi 110062, India.

Nickel, a major environmental pollutant is a known potent nephrotoxic agent. In this communication we report the chemopreventive effect of Terminalia chebula on nickel chloride (NiCl(2)) induced renal oxidative stress, toxicity and cell proliferation response in male Wistar rats. Administration of NiCl(2) (250micromoL Ni/kg body weight) to male Wistar rats resulted in an increase in the reduced renal glutathione content (GSH), glutathione-S-transferase (GST), glutathione reductase (GR), lipid peroxidation (LPO), H(2)O(2) generation, blood urea nitrogen (BUN) and serum creatinine with a concomitant decrease in the activity of glutathione peroxidase ($p<0.001$). Nickel chloride (NiCl(2)) treatment also induced tumor promotion markers, viz., ornithine decarboxylase (ODC) activity and thymidine [(3)H] incorporation into renal DNA ($p<0.001$). Prophylactic treatment of rats with T. chebula (25mg/kg body weight and 50mg/kg body weight) daily for one week resulted in the diminution of NiCl(2) mediated damage as evident from the down regulation of glutathione content, GST, GR, LPO, H(2)O(2) generation, BUN, serum creatinine, DNA synthesis ($p<0.001$) and ODC activity ($p<0.01$) with concomitant restoration of GPx activity. **Thus, the present investigation suggests that T. chebula extract could be used as therapeutic agent for cancer prevention as evident from this study where it blocks or suppresses the events associated with chemical carcinogenesis.** PMID: 17098582 [PubMed - indexed for MEDLINE]

Biol Pharm Bull. 2003 Sep;26(9):1331-5.

Antioxidant and free radical scavenging activities of Terminalia chebula.
Cheng HY, Lin TC, Yu KH, Yang CM, Lin CC.

Graduate Institute of Pharmaceutical Science, Kaohsiung Medical University, Taiwan.

Free radicals react with biological molecules and destroy the structure of cells, which eventually causes free-radical induced disease such as cancer, renal failure, aging, etc. In this study, 6 extracts and 4 pure compounds of Terminalia chebula RETZ. were investigated for anti-lipid peroxidation, anti-superoxide radical formation and free radical scavenging activities. The superoxide radical scavenging of the 4 pure compounds was further evaluated using electron spin resonance (ESR) spectrometry. **The results showed that all tested extracts and pure compounds of T. chebula exhibited antioxidant activity at different magnitudes of potency.** The antioxidant activity of each pure compound was derived from different pathways and was suggested to be specific. PMID: 12951481 [PubMed - indexed for MEDLINE]

Biol Pharm Bull. 2005 Sep;28(9):1639-44.

Antioxidant effects of aqueous extract of Terminalia chebula in vivo and in vitro.
Lee HS, Won NH, Kim KH, Lee H, Jun W, Lee KW.

Department of Food Science, College of Life & Environmental Sciences, Korea University, Seoul, Korea.

The ripe fruit of Terminalia chebula RETZIUS (T. chebula RETZ) (Combretsceae), which is a native plant in India and Southeast Asia, has traditionally been used as a popular folk medicine for homeostatic, antitussive, laxative, diuretic, and cardiotonic treatments. The objective of this study was to evaluate the protective effects of an aqueous extract of fruit of T. chebula on the tert-butyl hydroperoxide (t-BHP)-induced oxidative injury observed in cultured rat primary hepatocytes and rat liver. Both treatment and pretreatment of the hepatocytes with the T. chebula extract (TCE) significantly reversed the t-BHP-induced cell cytotoxicity and lactate dehydrogenase leakage. In addition, TCE exhibited in vitro ferric-reducing antioxidant activity and 2,2-diphenyl-1-picryhydrazyl free radical-scavenging activities. The in vivo study showed that pretreatment with TCE (500 or 1000 mg/kg) by gavage for 5 d before a single dose of t-BHP (0.1 mmol/kg i.p.) significantly lowered the serum levels of the hepatic enzyme markers aspartate aminotransferase and alanine aminotransferase and reduced the indicators of oxidative stress in the liver, such as the glutathine disulfide content and lipid peroxidation, in a dose-dependent manner. Histopathologic examination of the rat livers showed that TCE reduced the incidence of liver lesions, including hepatocyte swelling and neutrophilic infiltration, and repaired necrosis induced by t-BHP. Based on the results described

above, we speculate that TCE has the potential to play a role in the hepatic prevention of oxidative damage in living systems. PMID: 16141531 [PubMed - indexed for MEDLINE]

J Ethnopharmacol. 2002 Aug;81(3):327-36.

Inhibition of cancer cell growth by crude extract and the phenolics of Terminalia chebula retz. fruit.
Saleem A, Husheem M, Härkönen P, Pihlaja K.

Department of Chemistry, University of Turku, Kiinamyllynkatu 10, FIN-20014 Turku, Finland. amsale@utu.fi

A 70% methanol extract of **Terminalia chebula fruit, was studied for its effects on growth in several malignant cell lines including a human (MCF-7) and mouse (S115) breast cancer cell line, a human osteosarcoma cell line (HOS-1), a human prostate cancer cell line (PC-3) and a non-tumorigenic, immortalized human prostate cell line (PNT1A) using assays for proliferation ([(3)H]-thymidine incorporation and coulter counting), cell viability (ATP determination) and cell death (flow cytometry and Hoechst DNA staining). In all cell lines studied, the extract decreased cell viability, inhibited cell proliferation, and induced cell death in a dose dependent manner**. Flow cytometry and other analyses showed that some apoptosis was induced by the extract at lower concentrations, but at higher concentrations, necrosis was the major mechanism of cell death. ATP assay guided chromatographic fractionation of the extract yielded ellagic acid, 2,4-chebulyl-beta-D-glucopyranose (a new natural product), and chebulinic acid which were tested by ATP assay on HOS-1 cell line in comparison to three known antigrowth phenolics of Terminalia, gallic acid, ethyl gallate, luteolin, and tannic acid. Chebulinic acid (IC(50) = 53.2 microM +/- 0.16) > tannic acid (IC(50) = 59.0 microg/ml +/- 0.19) > and ellagic acid (IC(50) = 78.5 microM +/- 0.24), were the most growth inhibitory phenolics of T. chebula fruit in our study. PMID: 12127233 [PubMed - indexed for MEDLINE]

Indian J Exp Biol. 2004 Feb;42(2):174-8.

Protective effect of Terminalia chebula against experimental myocardial injury induced by isoproterenol.
Suchalatha S, Shyamala Devi CS.

Department of Biochemistry and Molecular Biology, University of Madras, Guindy Campus, Chennai 600 025, India.

Cardioprotective effect of ethanolic extract of Terminalia chebula fruits (500 mg/kg body wt) was examined in isoproterenol (200 mg/kg body wt) induced myocardial damage in rats. In isoproterenol administered rats, the level of lipid peroxides increased significantly in the serum and heart. A significant decrease was observed in the activity of the myocardial marker enzymes with a concomitant increase in their activity in serum. **Histopathological examination was carried out to confirm the myocardial necrosis. T. chebula extract pretreatment was found to ameliorate the effect of isoproterenol on lipid peroxide formation and retained the activities of the diagnostic marker enzymes.** PMID: 15282950 [PubMed - indexed for MEDLINE]

Phytother Res. 2007 May;21(5):476-80.

Evaluation of the growth inhibitory activities of Triphala against common bacterial isolates from HIV infected patients.
Srikumar R, Parthasarathy NJ, Shankar EM, Manikandan S, Vijayakumar R, Thangaraj R, Vijayananth K, Sheeladevi R, Rao UA.

Department of Physiology, Faculty of Medicine, Dr. ALM PG Institute of Basic Medical Sciences, University of Madras, Taramani Campus, Chennai, Tamilnadu, India 600 113.

The isolation of microbial agents less susceptible to regular antibiotics and the rising trend in the recovery rates of resistant bacteria highlights the need for newer alternative principles. Triphala has been used in traditional medicine

practice against certain diseases such as jaundice, fever, cough, eye diseases etc. In the present study phytochemical (phenolic, flavonoid and carotenoid) and antibacterial activities of aqueous and ethanol extracts of Triphala and its individual components (Terminalia chebula, Terminalia belerica and Emblica officinalis) were tested against certain bacterial isolates (Pseudomonas aeruginosa, Klebsiella pneumoniae, Shigella sonnei, S. flexneri, Staphylococcus aureus, Vibrio cholerae, Salmonella paratyphi-B, Escherichia coli, Enterococcus faecalis, Salmonella typhi) obtained from HIV infected patients using Kirby-Bauer's disk diffusion and minimum inhibitory concentration (MIC) methods. **T. chebula was found to possess high phytochemical content followed by T. belerica and E. officinalis in both aqueous and ethanol extracts. Further, most of the bacterial isolates were inhibited by the ethanol and aqueous extracts of T. chebula followed by T. belerica and E. officinalis by both disk diffusion and MIC methods. The present study revealed that both individual and combined aqueous and ethanol extracts of Triphala have antibacterial activity against the bacterial isolates tested**. Copyright 2007 John Wiley & Sons, Ltd. PMID: 17273983 [PubMed - indexed for MEDLINE]

Turmeric Root (Curcuma longa)

J Nat Prod. 2006 Mar;69(3):351-5.
Turmeric extracts containing curcuminoids prevent experimental rheumatoid arthritis.
Funk JL, Oyarzo JN, Frye JB, Chen G, Lantz RC, Jolad SD, Sólyom AM, Timmermann BN.

Arizona Center for Phytomedicine Research, Department of Medicine, Department of Cell Biology and Anatomy, University of Arizona, Tucson, 85724, USA. jfunk@u.arizona.edu

Turmeric has been used for centuries in Ayurvedic medicine as a treatment for inflammatory disorders including arthritis. On the basis of this traditional usage, dietary supplements containing turmeric rhizome and turmeric extracts are also being used in the western world for arthritis treatment and prevention. However, to our knowledge, no data are available regarding antiarthritic efficacy of complex turmeric extracts similar in composition to those available for use as dietary supplements. Therefore, the studies described here were undertaken to determine the in vivo efficacy of well-characterized curcuminoid-containing turmeric extracts in the prevention or treatment of arthritis using streptococcal cell wall (SCW)-induced arthritis, a well-described animal model of rheumatoid arthritis (RA). Arthritic index, a clinical measure of joint swelling, was used as the primary endpoint for assessing the effect of extracts on joint inflammation. An essential oil-depleted turmeric fraction containing 41% of the three major curcuminoids was efficacious in preventing joint inflammation when treatment was started before, but not after, the onset of joint inflammation. A commercial sample containing 94% of the three major curcuminoids was more potent in preventing arthritis than the essential oil-depleted turmeric fraction when compared by total curcuminoid dose per body weight. In conclusion, these data (1) document the in vivo antiarthritic efficacy of an essential oil-depleted turmeric fraction and (2) **suggest that the three major curcuminoids are responsible for this antiarthritic effect**, while the remaining compounds in the crude turmeric extract may inhibit this protective effect. PMID: 16562833 [PubMed - indexed for MEDLINE]

Mol Carcinog. 2006 May;45(5):320-32.
Antitumor action of curcumin in human papillomavirus associated cells involves downregulation of viral oncogenes, prevention of NFkB and AP-1 translocation, and modulation of apoptosis.
Divya CS, Pillai MR.

Department of Molecular Medicine, Regional Cancer Centre, Thiruvananthapuram, Kerala, India.

Curcumin (diferuloyl methane), the major yellow pigment from the rhizomes of turmeric (Curcuma longa Linn), has anticancer properties. Infection with high-risk human papillomaviruses (HPV) leads to development of cervical carcinoma, predominantly through the action of viral oncoproteins E6 and E7.**The present study aims at analyzing the antitumor and antiviral properties of curcumin,** on HPV associated cervical cancer cells. Our findings indicate curcumin to be cytotoxic to cervical cancer cells in a concentration-dependent and time-dependent manner. The cytotoxic activity was selectively more in HPV16 and HPV18 infected cells compared to non-HPV infected cells. Balance between tumor cell proliferation and spontaneous cell death via apoptosis had an important role in regulation of tumor cell growth. Curcumin-induced apoptosis in cervical cancer cells. Morphological hallmarks of

apoptosis such as nuclear fragmentation and internucleosomal fragmentation of DNA were observed. Curcumin also selectively inhibited expression of viral oncogenes E6 and E7, evident from RT-PCR and Western blotting data. Electrophoretic mobility shift assay revealed that activation of NFkappaB-induced by TNFalpha is down regulated by curcumin. Curcumin blocked IkBalpha phosphorylation and degradation, leading to abrogation of NFkappaB activation. Curcumin also down regulated the expression of COX-2, a gene regulated by NFkappaB. Binding of AP-1, an indispensable component for efficient epithelial tissue-specific gene expression of HPV was also selectively down regulated by curcumin**. These results provide attractive data for the possible use of curcumin in the management of HPV associated tumors**. (c) 2006 Wiley-Liss, Inc. PMID: 16526022 [PubMed - indexed for MEDLINE]

Am J Chin Med. 2005;33(3):449-57.
Effect of curcuma herbs on vasomotion and hemorheology in spontaneously hypertensive rat.
Goto H, Sasaki Y, Fushimi H, Shibahara N, Shimada Y, Komatsu K.

Department of Kampo Diagnostics, Institute of Natural Medicine, Toyama Medical and Pharmaceutical University, Japan. hiro510@ms.toyama-mpu.ac.jp

Curcuma herbs have a vasodilator effect. The effects of C. longa, which induces only endothelium-independent vasodilatation, and C. zedoaria, which induces both endothelium-dependent and -independent vasodilatation, were studied on vasomotion and hemorheology in spontaneously hypertensive rats. Spontaneously hypertensive eight-week-old male rats were assigned to five groups. For 12 weeks, the control group received standard chow. The 3%CL (C. longa) group received standard chow containing 3% (wt/wt) C. longa. The 1%CZ and 3%CZ (C. zedoaria) groups received standard chow containing 1% and 3% (wt/wt) C. zedoaria, respectively. The captoril group received standard chow and 100 mg/kg/day of captoril in drinking water. Blood pressure, vasomotion, hemorheology, etc. were examined. Systolic blood pressure of the 3%CZ and captoril groups decreased significantly as compared to the control group. Acetylcholine-induced endothelium-dependent relaxations of the 3%CZ and captoril groups were increased to a greater degree, significantly, than the control group. When testing xanthine oxidase-induced contraction, the 3%CZ group was significantly decreased as compared to the control group. Low shear stress of whole blood viscosity showed the 3%CL and 3%CZ groups to be decreased significantly compared to the control group. **Thus, Curcuma herbs have hypotensive and protective effect on the endothelium in spontaneously hypertensive rats. Especially, C. zedoaria is more effective than C. longa, and its mechanism is thought to be related to a radical scavenging effect and improvement of hemorheology**. PMID: 16047562 [PubMed - indexed for MEDLINE]

Curr Pharm Des. 2002;8(19):1695-706.
Chemotherapeutic potential of curcumin for colorectal cancer.
Chauhan DP.

Division of Gastroenterology, Department of Medicine, The University of California, San Diego, CA 92093-0688, USA. dchauhan@ucsd.edu

Colorectal cancer is one of the leading causes of cancer deaths in the Western world. More than 56,000 newly diagnosed colorectal cancer patients die each year in the United States. Available therapies are either not effective or have unwanted side effects. Epidemiological data suggest that dietary manipulations play an important role in the prevention of many human cancers. Curcumin the yellow pigment in turmeric has been widely used for centuries in the Asian countries without any toxic effects. Epidemiological data also suggest that curcumin may be responsible for the lower rate of colorectal cancer in these countries. Curcumin is a naturally occurring powerful anti-inflammatory medicine. The anticancer properties of curcumin have been shown in cultured cells and animal studies. Curcumin inhibits lipooxygenase activity and is a specific inhibitor of cyclooxygenase-2 expression. Curcumin inhibits the initiation of carcinogenesis by inhibiting the cytochrome P-450 enzyme activity and increasing the levels of glutathione-S-transferase. Curcumin inhibits the promotion/progression stages of carcinogenesis. The anti-tumor effect of curcumin has been attributed in part to the arrest of cancer cells in S, G2/M cell cycle phase and induction of apoptosis. Curcumin inhibits the growth of DNA mismatch repair defective colon cancer cells. Therefore, curcumin may have value as a safe chemotherapeutic agent for the treatment of tumors exhibiting DNA mismatch repair deficient and microsatellite instable phenotype. **Curcumin should be considered as a safe, non-toxic and easy to use chemotherapeutic agent for colorectal cancers arise in the setting of chromosomal instability as well as microsatellite instability.** PMID: 12171541 [PubMed - indexed for MEDLINE]

Cancer Lett. 2001 Oct 30;172(2):111-8.
Specific inhibition of cyclooxygenase-2 (COX-2) expression by dietary curcumin in HT-29 human colon cancer cells.
Goel A, Boland CR, Chauhan DP.

Division of Gastroenterology, Department of Medicine, The University of California San Diego, 9500 Gilman Drive, La Jolla, CA 92093-0688, USA.

Curcumin, a major yellow pigment and active component of turmeric, has been shown to possess anti-inflammatory and anti-cancer activities. Cyclooxygenase (COX)-2 plays an important role in colon carcinogenesis. To investigate the effect of curcumin on COX-2 expression, we treated HT-29 human colon cancer cells with various concentrations of curcumin. Curcumin inhibited the cell growth of HT-29 cells in a concentration- and time-dependent manner. Curcumin markedly inhibited the mRNA and protein expression of COX-2, but not COX-1. **These data suggest that a non-toxic concentration of curcumin has a significant effect on the in vitro growth of HT-29 cells, specifically inhibits COX-2 expression, and may have value as a safe chemopreventive agent for colon cancer.** PMID: 11566484 [PubMed - indexed for MEDLINE]

J Clin Rheumatol. 2004 Oct;10(5):236-245.
A 32-Week Randomized, Placebo-Controlled Clinical Evaluation of RA-11, an Ayurvedic Drug, on Osteoarthritis of the Knees.

Chopra A, Lavin P, Patwardhan B, Chitre D.

From the *Center for Rheumatic Diseases, Inlaks and Budhrani Hospital, Bharati Hospital Medical College (Deemed University), Pune, India; †Averion, Inc., Framingham, Massachusetts; the ‡School of Health Sciences, University of Pune, India; and §BIO-VED Pharmaceuticals, Inc., San Jose, California.

BACKGROUND:: The ancient Indian (Asian) Ayurvedic medicinal system uses herbomineral drugs to treat arthritis. Despite centuries of use, very few have been tested by drug trials. RA-11 (ARTREX, MENDAR), a standardized multiplant Ayurvedic drug (Withania somnifera, Boswellia serrata, Zingiber officinale, and Curcuma longa) is currently used to treat arthritis. OBJECTIVE:: The objective of this study was to evaluate the efficacy and safety of RA-11 in patients with symptomatic osteoarthritis (OA) of the knees. METHODS:: A total of 358 patients with chronic knee pain were screened free-of-cost in "arthritis camps" in an Indian metropolis. Ninety patients with primary OA of the knees (ACR classification; Arthritis Rheum 1986;29:1039-1049) were found eligible (postanalgesic washout pain visual analog score [VAS] >/=40 mm in either or both knees on body weight-bearing activities) to enroll into a randomized, double-blind, placebo-controlled, parallel efficacy, single-center, 32-week drug trial (80% power to detect 25% difference, P = 0.05, 2-sided). Concurrent analgesics/nonsteroidal antiinflammatory drugs and steroids in any form were not allowed. Lifestyle and/or dietary restrictions, as per routine Ayurveda practices, were not imposed. Pain VAS (maximum pain in each knee recorded by the patient during the preceding 48 hours) and modified WOMAC (Western Ontario McMaster University OA Index, Likert scale, version 3.0) were the primary efficacy variables. The WOMAC section on "physical function difficulty" was modified for Indian use and validated before the trial. Routine laboratory testing was primarily done to monitor drug safety. At baseline, the groups (active = 45, placebo = 45) were well matched for several measures (mean pain VAS: active = 6.17; placebo = 6.5). **RESULTS:: 1) Efficacy: Compared with placebo, the mean reduction in pain** VAS at week 16 (active = 2.7, placebo = 1.3) and week 32 (active = 2.8, placebo = 1.8) in the active group was significantly (P <0.05, analysis of variance [ANOVA]) better. Similarly, the improvement in the WOMAC scores at week 16 and week 32 were also significantly superior (P <0.01, ANOVA) in the active group. 2) Safety: Both the groups reported mild adverse events (AE) without any significant difference. 3) Withdrawals: Twenty-eight patients were discontinued. None reported drug-related toxicity. The majority failed follow up/compliance. No differences were observed between the groups. CONCLUSION:: This controlled drug trial demonstrates the potential efficacy and safety of RA- 11 in the symptomatic treatment of OA knees over 32 weeks of therapy. PMID: 17043520 [PubMed - as supplied by publisher]

Z Naturforsch [C]. 2006 Jan-Feb;61(1-2):6-10.
Seasonal variation and analgesic properties of different parts from Curcuma zedoaria Roscoe (Zingiberaceae) grown in Brazil.

Pamplona CR, de Souza MM, Machado Mda S, Cechinel Filho V, Navarro D, Yunes RA, Delle Monache F, Niero R.

Núcleo de Investigações Químico-Farmacêuticas (NIQFAR) e Programa de Mestrado em Ciências Farmacêuticas, Universidade do Vale do Itajaí (UNIVALI), 88.302-202 Itajaí, SC, Brazil.

This work describes the seasonal variation of curcumenol (1) and dihydrocurdione (2), two active terpenoids from different parts (roots, mother rhizome and rugous rhizome) of Curcuma zedoaria grown in Brazil. The analysis was carried out by high resolution gas chromatography, using external standards for determination. The results showed that both terpenoids are present in all the parts studied. However, C. zedoaria exhibited about three times more terpenoids in the mother rhizome in autumn than in other parts and seasons studied. The antinociceptive activity of the dichloromethane extracts from different parts and collected in different seasons was studied using the acetic acid-induced abdominal constriction model in mice. The extracts obtained from mother rhizome collected in autumn and winter at doses of 10 mg/kg body weight, i.p., caused considerable antinociceptive activity inhibiting 91.1 and 93.4% of the abdominal constrictions, respectively, whereas compounds 1 and 2 caused inhibitions of 64.0 and 46.0%, respectively**. These results confirm that both compounds contribute to explain the antinociceptive effect (*Anti-Pain Sensation)* of the plant but suggest that other compounds are also acting as analgesics. *An analgesic (colloquially known as a painkiller)-Wikipedia*** PMID: 16610209 [PubMed - indexed for MEDLINE]

Adv Exp Med Biol. 2007;595:379-405.
Protection from acute and chronic lung diseases by curcumin.
Venkatesan N, Punithavathi D, Babu M.

Faculte de Medecine, UMR-7561, CNRS UHP, Vandoeuvre lès Nancy, France. vnar12@yahoo.com

The aim of this review has been to describe the current state of the therapeutic potential of curcumin in acute and chronic lung injuries. Occupational and environmental exposures to mineral dusts, airborne pollutants, cigarette smoke, chemotherapy, and radiotherapy injure the lungs, resulting in acute and chronic inflammatory lung diseases. Despite major advances in treating lung diseases, until now disease-modifying efficacy has not been demonstrated for any of the existing drugs. Current medical therapy offers only marginal benefit; therefore, there is an essential need to develop new drugs that might be of effective benefit in clinical settings. Over the years, there has been increasing evidence that curcumin, a phytochemical present in turmeric (Curcuma longa), has a wide spectrum of therapeutic properties and a remarkable range of protective effects in various diseases. Several experimental animal models have tested curcumin on lung fibrosis and these studies demonstrate that curcumin attenuates lung injury and fibrosis caused by radiation, chemotherapeutic drugs, and toxicants. **The growing amount of data from pharmacological and animal studies also supports the notion that curcumin plays a protective role in chronic obstructive pulmonary disease, acute lung injury, acute respiratory distress syndrome, and allergic asthma, its therapeutic action being on the prevention or modulation of inflammation and oxidative stress. These findings give substance to the possibility of testing curcumin in patients with lung diseases.** PMID: 17569221 [PubMed - indexed for MEDLINE]

Cancer Lett. 1995 Jul 20;94(1):79-83.
Anti-tumour and antioxidant activity of natural curcuminoids.
Ruby AJ, Kuttan G, Babu KD, Rajasekharan KN, Kuttan R.

Amala Cancer Research Centre, Kerala, India.

Matural curcuminoids, curcumin, I, II and III isolated from turmeric (Curcuma longa) were compared for their cytotoxic**, tumour reducing and antioxidant activities**. Curcumin III was found to be more active than the other two as a cytotoxic agent and in the inhibition of Ehrlich ascites tumour in mice (ILS 74.1%). These compounds were also checked for their antioxidant activity which possibly indicates their potential use as anti-promoters. The amount of curcuminoids (I, II and III) needed for 50% inhibition of lipid peroxidation was 20, 14 and 11 g/m. Concentrations

needed for 50% inhibition of superoxides were 6.25, 4.25 and 1.9 micrograms/ml and those for hydroxyl radical were 2.3, 1.8 and 1.8 micrograms/ml, respectively. The ability of these compounds to suppress the superoxide production by macrophages activated with phorbol-12-myristate-13-acetate (PMA) indicated that all the three curcuminoids inhibited superoxide production and curcumin III produced maximum effect. These results indicate that curcumin III is the most active of the curcuminoids present in turmeric. Synthetic curcumin I and III had similar activity to natural curcumins. PMID: 7621448 [PubMed - indexed for MEDLINE]

Indian J Exp Biol. 2007 Sep;45(9):791-801.
Antischistosomal *(Anti-Parasite)* and liver protective effects of Curcuma longa extract in Schistosoma mansoni infected mice.
El-Ansary AK, Ahmed SA, Aly SA.

Medicinal Chemistry Department, National Research Centre, Dokki, Cairo, Egypt.

With a view to clarify the induction of the "Crabtree consequence" in liver cells of S. mansoni infected mice, the curative effect of oil extract of C. longa was tested and compared to praziquantel (PZQ) the effective drug against all schistosome species occurring in man. Protein, glucose, glucose-6-phopsphatase, AMP-deaminase, adensoine deaminase, urea concentration, pyravate kinase (PK), phosphoenol pyruvate carboxykinase (PEPCK) and PK/PEPCK ratio were estimated. In addition, worm burden and ova count in mice infected with S. mansoni were elucidated. The result showed that C. longa normalized the concentration of protein, glucose, AMP-deaminase and adenosine deaminase, which were changed by infection. Moreover, it lowered pyruvate kinase level, while PZQ-treatment induced more elevation of this enzyme**. PZQ was more effective in lowering worm burden while C. longa extract was more potent in reducing egg count.** PMID: 17907745 [PubMed - indexed for MEDLINE]

J Immunol. 2007 Jan 1;178(1):111-21.
Spleen tyrosine kinase (Syk), a novel target of curcumin, is required for B lymphoma growth.
Gururajan M, Dasu T, Shahidain S, Jennings CD, Robertson DA, Rangnekar VM, Bondada S.

Department of Microbiology, Immunology, and Molecular Genetics, University of Kentucky, Lexington, KY 40536, USA.

Curcumin (diferuloylmethane), a component of dietary spice turmeric (Curcuma longa), has been shown in recent studies to have therapeutic potential in the treatment of cancer, diabetes, arthritis, and osteoporosis. We investigated the ability of curcumin to modulate the growth of B lymphomas. Curcumin inhibited the growth of both murine and human B lymphoma in vitro and murine B lymphoma in vivo. We also demonstrate that curcumin-mediated growth inhibition of B lymphoma is through inhibition of the survival kinase Akt and its key target Bad. However, in vitro kinase assays show that Akt is not a direct target of curcumin. We identified a novel target for curcumin in B lymphoma viz spleen tyrosine kinase (Syk). Syk is constitutively activated in primary tumors and B lymphoma cell lines and curcumin down-modulates Syk activity accompanied by down-regulation of Akt activation. Moreover, we show that overexpression of Akt, a target of Syk, or Bcl-x(L), a target of Akt can overcome curcumin-induced apoptosis of B lymphoma cells. These observations suggest a novel growth promoting role for Syk in lymphoma cells. PMID: 17182546 [PubMed - indexed for MEDLINE]

Ann N Y Acad Sci. 2005 Nov;1056:206-17.
Curcumin: getting back to the roots.
Shishodia S, Sethi G, Aggarwal BB.

Cytokine Research Laboratory, Department of Experimental Therapeutics, The University of Texas M. D. Anderson Cancer Center, 1515 Holcombe Boulevard, Houston, TX 77030, USA.

The use of turmeric, derived from the root of the plant Curcuma longa, for treatment of different inflammatory diseases has been described in Ayurveda and in traditional Chinese medicine for thousands of years. The active component of turmeric responsible for this activity, curcumin, was identified almost two centuries ago. Modern science has revealed that curcumin mediates its effects by modulation of several important molecular targets, including transcription factors

(e.g., NF-kappaB, AP-1, Egr-1, beta-catenin, and PPAR-gamma), enzymes (e.g., COX2, 5-LOX, iNOS, and hemeoxygenase-1), cell cycle proteins (e.g., cyclin D1 and p21), cytokines (e.g., TNF, IL-1, IL-6, and chemokines), receptors (e.g., EGFR and HER2), and cell surface adhesion molecules. **Because it can modulate the expression of these targets, curcumin is now being used to treat cancer, arthritis, diabetes, Crohn's disease, cardiovascular diseases, osteoporosis, Alzheimer's disease, psoriasis, and other pathologies.** Interestingly, 6-gingerol, a natural analog of curcumin derived from the root of ginger (Zingiber officinalis), exhibits a biologic activity profile similar to that of curcumin. The efficacy, pharmacologic safety, and cost effectiveness of curcuminoids prompt us to "get back to our roots." PMID: 16387689 [PubMed - indexed for MEDLINE]

Biosci Biotechnol Biochem. 2007 Jun;71(6):1428-38.
Immunostimulating activity of crude polysaccharide extract isolated from Curcuma xanthorrhiza Roxb.
Kim AJ, Kim YO, Shim JS, Hwang JK.

Department of Biotechnology, Yonsei University, Korea.

Curcuma xanthorrhiza Roxb., commonly known as Javanese turmeric, has been reported to possess a variety of biological activities, including anti-inflammatory effects, anticarcinogenic effects, wound healing effects, and serum cholesterol-lowering effects. CPE, crude polysaccharide extract isolated from the rhizome of C. xanthorrhiza using 0.1 N NaOH, consisted of arabinose (18.69%), galactose (14.0%), glucose (50.67%), mannose (12.97%), rhamnose (2.73%), and xylose (0.94%), with an average molecular weight of 33,000 Da. In the present study, we investigated the effect of CPE on nitric oxide (NO), hydrogen peroxide (H2O2), tumor necrosis factor-alpha (TNF-alpha), and prostaglandin E2 (PGE2) production in RAW 264.7 cells. The uptake of fluorescein-labeled Escherichia coli was measured to determine whether CPE stimulates the phagocytic activity of RAW 264.7 cells. CPE significantly increased the phagocytosis of macrophages and the release of NO, H2O2, TNF-alpha, and PGE2 in a dose-dependent manner, and showed a similar activity to lipopolysaccharide (LPS). To study the mechanisms of CPE, we examined induction of iNOS and COX-2. NO and PGE2 were produced as a result of stimulation of inducible nitric oxide synthase (iNOS) and cyclooxygenase-2 (COX-2) respectively. Both modulations of iNOS and COX-2 expression by CPE were evaluated by Western immunoblotting and RT-PCR. Since transcription of these enzymes is under the control of nuclear factor-kappa B (NF-kappaB), we assessed the phosphorylation of inhibitor kappaBalpha (IkappaBalpha) through Western immunoblotting. CPE clearly induced phosphorylation of IkappaBalpha, suggesting a role as an NF-kappaB activator. Taking all this together, **we conclude that CPE isolated from Curcuma xanthorrhiza stimulates the immune functions of macrophages, which is mediated in part by specific activation of NF-kappaB.** PMID: 17587672 [PubMed - indexed for MEDLINE]

Am J Epidemiol. 2006 Nov 1;164(9):898-906. Epub 2006 Jul 26.
Curry consumption and cognitive function in the elderly.
Ng TP, Chiam PC, Lee T, Chua HC, Lim L, Kua EH.

Department of Psychological Medicine, National University of Singapore, Republic of Singapore. pcmngtp@nus.edu.sg

Curcumin, from the curry spice turmeric, has been shown to possess potent antioxidant and antiinflammatory properties and to reduce beta-amyloid and plaque burden in experimental studies, but epidemiologic evidence is lacking. The authors investigated the association between usual curry consumption level and cognitive function in elderly Asians. In a population-based cohort (n = 1,010) of nondemented elderly Asian subjects aged 60-93 years in 2003, the authors compared Mini-Mental State Examination (MMSE) scores for three categories of regular curry consumption, taking into account known sociodemographic, health, and behavioral correlates of MMSE performance. Those who consumed curry "occasionally" and "often or very often" had significantly better MMSE scores than did subjects who "never or rarely" consumed curry**. The authors reported tentative evidence of better cognitive performance from curry consumption in nondemented elderly Asians, which should be confirmed in future studies.** PMID: 16870699 [PubMed - indexed for MEDLINE]

Adv Exp Med Biol. 2007;595:1-75.
Curcumin: the Indian solid gold.
Aggarwal BB, Sundaram C, Malani N, Ichikawa H.

Department of Experimental Therapeutics, The University of Texas M.D. Anderson Cancer Center, Houston, TX 77030, USA. aggarwal@mdanderson.org

Turmeric, derived from the plant Curcuma longa, is a gold-colored spice commonly used in the Indian subcontinent, not only for health care but also for the preservation of food and as a yellow dye for textiles. Curcumin, which gives the yellow color to turmeric, was first isolated almost two centuries ago, and its structure as diferuloylmethane was determined in 1910. Since the time of Ayurveda (1900 Bc) numerous therapeutic activities have been assigned to turmeric for a wide variety of diseases and conditions, including those of the skin, pulmonary, and gastrointestinal systems, aches, pains, wounds, sprains, and liver disorders. Extensive research within the last half century has proven that most of these activities, once associated with turmeric, are due to curcumin. **Curcumin has been shown to exhibit antioxidant, anti-inflammatory, antiviral, antibacterial, antifungal, and anticancer activities and thus has a potential against various malignant diseases, diabetes, allergies, arthritis, Alzheimer's disease, and other chronic illnesses.** These effects are mediated through the regulation of various transcription factors, growth factors, inflammatory cytokines, protein kinases, and other enzymes. Curcumin exhibits activities similar to recently discovered tumor necrosis factor blockers (e.g., HUMIRA, REMICADE, and ENBREL), a vascular endothelial cell growth factor blocker (e.g., AVASTIN), human epidermal growth factor receptor blockers (e.g., ERBITUX, ERLOTINIB, and GEFTINIB), and a HER2 blocker (e.g., HERCEPTIN). Considering the recent scientific bandwagon that multitargeted therapy is better than monotargeted therapy for most diseases, curcumin can be considered an ideal "Spice for Life". PMID: 17569205 [PubMed - indexed for MEDLINE]

Adv Exp Med Biol. 2007;595:425-51.
Curcumin and autoimmune disease.
Bright JJ.

Neuroscience Research Laboratory, Methodist Research Institute, Clarian Health, Indianapolis, IN 46202, USA. jbright1@clarian.org

The immune system has evolved to protect the host from microbial infection; nevertheless, a breakdown in the immune system often results in infection, cancer, and autoimmune diseases. Multiple sclerosis, rheumatoid arthritis, type 1 diabetes, inflammatory bowel disease, myocarditis, thyroiditis, uveitis, systemic lupus erythromatosis, and myasthenia gravis are organ-specific autoimmune diseases that afflict more than 5% of the population worldwide. Although the etiology is not known and a cure is still wanting, the use of herbal and dietary supplements is on the rise in patients with autoimmune diseases, mainly because they are effective, inexpensive, and relatively safe. Curcumin is a polyphenolic compound isolated from the rhizome of the plant Curcuma longa that has traditionally been used for pain and wound-healing. **Recent studies have shown that curcumin ameliorates multiple sclerosis, rheumatoid arthritis, psoriasis, and inflammatory bowel disease in human or animal models**. Curcumin inhibits these autoimmune diseases by regulating inflammatory cytokines such as IL-1beta, IL-6, IL-12, TNF-alpha and IFN-gamma and associated JAK-STAT, AP-1, and NF-kappaB signaling pathways in immune cells. Although the beneficial effects of nutraceuticals are traditionally achieved through dietary consumption at low levels for long periods of time, the use of purified active compounds such as curcumin at higher doses for therapeutic purposes needs extreme caution. A precise understanding of effective dose, safe regiment, and mechanism of action is required for the use of curcumin in the treatment of human autoimmune diseases. PMID: 17569223 [PubMed - indexed for MEDLINE]

Phytother Res. 1999 Jun;13(4):318-22.
Efficacy of curcumin in the management of chronic anterior uveitis.
Lal B, Kapoor AK, Asthana OP, Agrawal PK, Prasad R, Kumar P, Srimal RC.

Department of Ophthalmology, K.G. Medical College, Lucknow, India.

Curcumin, obtained from rhizomes of Curcuma longa, was administered orally to patients suffering from chronic anterior uveitis (CAU) at a dose of 375 mg three times a day for 12 weeks. Of 53 patients enrolled, 32 completed the 12-week study. They were divided into two groups: one group of 18 patients received curcumin alone, whereas the other group of 14 patients, who had a strong PPD reaction, in addition received antitubercular treatment. The patients in both the groups started improving after 2 weeks of treatment. All the patients who received curcumin alone improved, whereas the group receiving antitubercular therapy along with curcumin had a response rate of 86%. Follow up of all the patients for the next 3 years indicated a recurrence rate of 55% in the first group and of 36% in the second group. Four of 18 (22%) patients in the first group and 3 of 14 patients (21%) in the second group lost their vision in the follow up period due to various complications in the eyes, e.g. vitritis, macular oedema, central venous block, cataract formation, glaucomatous optic nerve damage etc. None of the patients reported any side effect of the drug. **The efficacy of curcumin and recurrences following treatment are comparable to corticosteroid therapy, which is presently the only available standard treatment for this disease.** The lack of side effects with curcumin is its greatest advantage compared with corticosteroids. A double blind multi-centric clinical trial with this drug in CAU is highly desirable to further validate the results of the present study. PMID: 10404539 [PubMed - indexed for MEDLINE]

Biochem Pharmacol. 1995 Apr 18;49(8):1165-70.
Inhibition of human immunodeficiency virus type-1 integrase by curcumin.
Mazumder A, Raghavan K, Weinstein J, Kohn KW, Pommier Y.

Laboratory of Molecular Pharmacology, National Cancer Institute, Bethesda, MD 20892-4255, USA.

Curcumin (diferuloylmethane) is the yellow pigment in turmeric (Curcuma longa L.) that is widely used as a spice, food coloring (curry) and preservative. Curcumin exhibits a variety of pharmacological effects including antitumor, anti-inflammatory, and anti-infectious activities and is currently in clinical trials for AIDS patients. The effects of curcumin have been determined on purified human immunodeficiency virus type 1 (HIV-1) integrase. Curcumin has an inhibitory concentration50 (IC50) for strand transfer of 40 microM. Inhibition of an integrase deletion mutant containing only amino acids 50-212 suggests that curcumin interacts with the integrase catalytic core. Two structural analogs, methyl cinnamate and chlorogenic acid, were inactive. Energy minimization studies suggest that the anti-integrase activity of curcumin could be due to an intramolecular stacking of two phenyl rings that brings the hydroxyl groups into close proximity**. The present data suggest that HIV-1 integrase inhibition may contribute to the antiviral activity of curcumin.** These observations suggest new strategies for antiviral drug development that could be based upon curcumin as a lead compound for the development of inhibitors of HIV-1 integrase. PMID: 7748198 [PubMed - indexed for MEDLINE]

Res Virol. 1998 Jan-Feb;149(1):43-52.
Curcumin and curcumin derivatives inhibit Tat-mediated transactivation of type 1 human immunodeficiency virus long terminal repeat.
Barthelemy S, Vergnes L, Moynier M, Guyot D, Labidalle S, Bahraoui E.

Laboratoire de Synthèse, Physico-Chimie et Radiobiologie, Faculté de Pharmacie, Toulouse, France.

The transcription of HIV1 provirus is regulated by both cellular and viral factors. Various evidence suggests that Tat protein secreted by HIV1-infected cells may have additional action in the pathogenesis of AIDS because of its ability to also be taken up by non-infected cells. Curcumin [diferuloylmethane or 1,7-bis-(4-hydroxy-3-methoxyphenyl)-1,6-heptadiene-3,5-dione] is the yellow pigment in turmeric Curcuma longa (Linn). It exhibits a variety of pharmacological effects including antiinflammatory and antiretroviral activities. Here, we demonstrated that curcumin used at 10 to 100 nM inhibited Tat transactivation of HIV1-LTR lacZ by 70 to 80% in HeLa cells. In order to develop more efficient curcumin derivatives, we synthesized and tested in the same experimental system the inhibitory activity of reduced curcumin (C1), which lacks the spatial structure of curcumin; allyl-curcumin (C2), which possesses a condensed allyl derivative on curcumin that plays the role of metal chelator; and tocopheryl-curcumin (C3), which

enhances the antioxidant activity of the molecule. Results obtained with C1, C2 and C3 curcumin derivatives showed a significant inhibition (70 to 85%) of Tat transactivation. **Despite the fact that tocopheryl-curcumin (C3) failed to scavenge O2.-, this curcumin derivative exhibited the most activity; 70% inhibition was obtained at 1 nM, while only 35% inhibition was obtained with the curcumin.** PMID: 9561563 [PubMed - indexed for MEDLINE]

Jujube Fruit (Ziziphus jujuba Mill)

J Chromatogr A. 2006 Mar 10;1108(2):188-94. Epub 2006 Feb 3.
Simultaneous determination of saponins and fatty acids in Ziziphus jujuba (Suanzaoren) by high performance liquid chromatography-evaporative light scattering detection and pressurized liquid extraction.
Zhao J, Li SP, Yang FQ, Li P, Wang YT.

Institute of Chinese Medical Sciences, University of Macau, Taipa, Macau.

The seed of Ziziphus jujube Mill. var. spinosa (Bunge) Hu ex H. F. Chou, Suanzaoren in Chinese, is one of commonly used Chinese medicines. Saponins and fatty oil **contains several fatty acids in Suanzaoren are responsible for its therapeutic activities.** In this study, a new HPLC coupled with evaporative light scattering detection (ELSD) and pressurized liquid extraction (PLE) method was developed for the simultaneous quantitative determination of 11 major components of 2 saponins and 9 fatty acids, namely jujuboside A, jujuboside B, lauric acid, myristic acid, palmitic acid, palmitoleic acid, stearic acid, oleic acid, linoleic acid, arachidic acid and docosanoic acid in Suanzaoren. Simultaneous separation of these eleven compounds was achieved on a C18 analytical column. The mobile phase consisted of (A) 0.1% aqueous acetic acid and (B) methanol with 0.1% acetic acid using a gradient elution. The drift tube temperature of ELSD was set at 75 degrees C, and nitrogen flow-rate was 1.8l/min. All calibration curves showed good linearity (r(2)>0.9955) within test ranges. This method showed good reproducibility for the quantification of these eleven components in Suanzaoren with intra- and inter-day variations of less than 3.41 and 4.37%, respectively. The validated method was successfully applied to quantify 11 investigated components in nine commercial samples of Suanzaoren. PMID: 16458908 [PubMed - indexed for MEDLINE]

Am J Chin Med. 2007;35(3):517-32.
Mechanism of the anti-cancer activity of Zizyphus jujuba in HepG2 cells.
Huang X, Kojima-Yuasa A, Norikura T, Kennedy DO, Hasuma T, Matsui-Yuasa I.

Department of Food and Human Health Sciences, Graduate School of Human Life Science, Osaka City University, 3-3-138 Sugimoto, Sumiyoshi-ku, Osaka 558-8585, Japan.

The Zizyphus jujuba fruit has been used as a traditional Chinese medicinal herb and considered to affect various physiological functions in the body for thousands of years. However, its anti-cancer activity and mechanism of action remain to be elucidated. **We investigated the anti-cancer activity of Zizyphus jujuba Mill and its underlining mechanisms of action in human hepatoma cells (HepG2) and found that the extract of Z. jujuba decreased the viability of the cells.** Further extraction of the initial Z. jujuba extract with organic solvents revealed that the chloroform fraction (CHCl(3)-F) was the most effective. Interestingly, the CHCl(3)-F induced not only apoptosis but also G1 arrest at a low concentration (100 mug/ml) and G2/M arrest at a higher concentration (200 mug/ml) by cell cycle assay. Apoptosis, an increase in intracellular ROS (reactive oxygen species) level, a decline of mitochondrial membrane potential at low Z. jujuba concentrations, and a ROS-independent mitochondrial dysfunction pathway at high concentrations were all observed. CHCl(3)-F-induced G1 arrest in HepG2 cells was associated with an increase in hypohosphorylation of Rb and p27(Kip1), and a decrease of phosphorylated Rb. However, CHCl(3)-F-induced G2/M arrest in HepG2 cells correlated with a decrease of the p27(Kip1) levels and generation of the phosphorylation of p27(Kip1), however the hypohosphorylation of Rb protein remained. Collectively, our findings suggest that the CHCl(3)-F extract of Z. jujuba extract induced a concentration dependent effect on apoptosis and a differential cell cycle arrest in HepG2 cells. PMID: 17597510 [PubMed - indexed for MEDLINE]

Curr Eye Res. 2005 Jul;30(7):583-91.
Ocimum sanctum modulates selenite-induced cataractogenic changes and prevents rat lens opacification.
Gupta SK, Srivastava S, Trivedi D, Joshi S, Halder N.
Department of Pharmacology, All India Institute of Medical Sciences, New Delhi, India. skgup@hotmail.com

PURPOSE: To study the effect of **Ocimum sanctum (OS)** on selenite-induced morphological and biochemical changes in isolated rat lenses as well as on cataract incidence in rat pups. METHODS: Transparent rat lenses were divided into normal, selenite-only, and four treated groups. Selenite-only and treated group lenses were subjected to oxidative stress in vitro by incorporating sodium selenite (100 microM) in the culture medium. The effect of OS (70, 140, 280, and 560 microg/ml) was studied on the levels of reduced glutathione (GSH) and thiobarbituric acid reacting substances (TBARS) in selenite-challenged lenses. The lowest concentration of OS offering significant modulation on these two parameters was determined. Subsequently, the effect of prior and cotreatment with the lowest effective concentration of OS was studied on TBARS, GSH, and on lens antioxidant enzymes such as superoxide dismutase (SOD), glutathione peroxidase (GSHPx), catalase (CAT), and glutathione-S-transferase (GST). Changes in lens protein profiles under different incubation conditions were analyzed by SDS gel-electrophoresis. In vivo, cataract was induced by a single subcutaneous injection of sodium selenite (25 micromole/kg b.w.) to 9-day-old rat pups. The anticataract effect of OS (5 and 10 mg/kg b.w.) injected intraperitoneally 4 hr prior to selenite challenge was evaluated by the presence of lens nuclear opacity in rat pups on the 16th postnatal day. Insolubilization of lens proteins post-selenite injection was monitored for 4 days. RESULTS: The lenses in the selenite-only group developed cortical opacities in 24 hr. OS showed different degrees of positive modulation in selenite-induced morphological as well as biochemical changes. The lowest effective dose of OS that significantly modulated glutathione and thiobarbituric acid reacting substances was found to be 140 microg/ml. At this dose, a significant increase in antioxidant enzyme levels and preservation of normal lens protein profile was observed. OS at the dose of 70 microg/ml did not show any significant protection with respect to either morphology or biochemistry of lenses. In vivo, 5 and 10 mg/kg of OS reduced the incidence of selenite cataract by 20% and 60%, respectively, and prevented protein insolubilization as well. **CONCLUSIONS: Aqueous extract of OS possesses potential anticataract activity against selenite-induced experimental cataractogenesis.** The protective effect was supported by restoration of the antioxidant defense system and inhibition of protein insolubilization of rat lenses as well. PMID: 16020293 [PubMed - indexed for MEDLINE]

Tulsi Leaf (Ocimum sanctum)

Horm Metab Res. 2008 Jan;40(1):44-9. Epub 2007 Dec 18.
Prevention of Insulin Resistance by Ingesting Aqueous Extract of Ocimum sanctum to Fructose-fed Rats.
Reddy SS, Karuna R, Baskar R, Saralakumari D.

1Department of Biochemistry, Sri Krishnadevaraya University, Anantapur, Andhra Pradesh, India.

The study was aimed to examine if oral administration of the aqueous extract of the whole plant OCIMUM SANCTUM (OS) protects against the development of insulin resistance in fructose fed rats. Male Wister rats were randomly divided into four groups of eight animals each: group-S (starch diet), group-F (fructose diet), group-F+OS (fructose diet along with OCIMUM SANCTUM extract at a dose of 200 mg/kg), group-S+OS (starch diet along with OCIMUM SANCTUM). During the experimental period of 60 days body weight, plasma glucose, insulin, and triglycerides were measured at an interval of 15 days. Insulin sensitivity was assessed at the end of experimental period by measuring glucose-insulin index, which is the product of the areas under the curve of glucose and insulin during oral glucose tolerance test. The nontoxic nature of OS was revealed by unaltered body weight, plasma glucose, insulin, and triglyceride levels in group-S+OS when compared with group-S. A significant gain in body weight, hyperglycemia, hyperinsulinemia, hypertriglyceridemia, and insulin resistance were observed in group-F when compared with group-S. OS treatment prevented the observed fructose induced alterations in group-F+OS**. In conclusion, our results suggests that oral administration of OS aqueous extract could delay the development of insulin resistance in rats and may be used as an adjuvant therapy for treating diabetic patients with insulin resistance.** PMID: 18085503 [PubMed - in process]

J Endocrinol. 2006 Apr;189(1):127-36.
Ocimum sanctum leaf extracts stimulate insulin secretion from perfused pancreas, isolated islets and clonal pancreatic beta-cells.
Hannan JM, Marenah L, Ali L, Rokeya B, Flatt PR, Abdel-Wahab YH.

Diabetes Research Group, School of Biomedical Sciences, University of Ulster, Coleraine, Northern Ireland BT52 1SA, UK.

Ocimum sanctum leaves have previously been reported to reduce blood glucose when administered to rats and humans with diabetes. In the present study, the effects of ethanol extract and five partition fractions of O. sanctum leaves were studied on insulin secretion together with an evaluation of their mechanisms of action. The ethanol extract and each of the aqueous, butanol and ethylacetate fractions stimulated insulin secretion from perfused rat pancreas, isolated rat islets and a clonal rat beta-cell line in a concentration-dependent manner. The stimulatory effects of ethanol extract and each of these partition fractions were potentiated by glucose, isobutylmethylxanthine, tolbutamide and a depolarizing concentration of KCl. Inhibition of the secretory effect was observed with diazoxide, verapamil and Ca2+ removal. In contrast, the stimulatory effects of the chloroform and hexane partition fractions were associated with decreased cell viability and were unaltered by diazoxide and verapamil. The ethanol extract and the five fractions increased intracellular Ca2+ in clonal BRIN-BD11 cells, being partly attenuated by the addition of verapamil. **These findings indicated that constituents of O. sanctum leaf extracts have stimulatory effects on physiological pathways of insulin secretion which may underlie its reported antidiabetic action.** PMID: 16614387 [PubMed - indexed for MEDLIN

Phytomedicine. 2002 May;9(4):346-51. Extract of Ocimum canum lowers blood glucose and facilitates insulin release by isolated pancreatic beta-islet cells.
Nyarko AK, Asare-Anane H, Ofosuhene M, Addy ME.

Noguchi Memorial Institute for Medical Research, University of Ghana, Legon. anyarko@noguchi.mimcom.net

Aqueous extract of Ocimum canumSim, (Lamiaceae) is used by some Ghanaians to manage diabetes mellitus. In vivo modulation of levels of fasting blood glucose by 0. canum extract was evaluated in type-II diabetes mellitus using the C57BL/KsJ db/db genetically diabetic animal model, and its effects on glucose-stimulated insulin release in vitro were monitored using isolated rat pancreatic beta-islet cells. The results showed that fasting blood glucose levels and body weight decreased significantly ($p < 0.05$) in diabetic and non-diabetic C57BL/KsJ mice, which were administered aqueous extract of 0. canum. In vitro, the 0. canum extract significantly enhanced insulin release from isolated rat pancreatic beta-islet cells. Insulin release was found to be dependent on glucose concentration and increased with increasing O. canum concentration in the incubation medium up to an optimum extract concentration of 0.03 mg/ml. Release of the hormone decreased beyond this concentration of extract in the medium. Addition to the medium of Desmodium adscendens, a plant preparation used to manage inflammatory disorders, did not increase but rather inhibited insulin secretion by the pancreatic beta-islet cells. **These results could explain the use of 0. canum in Ghanaian folk medicine to manage diabetes mellitus.** PMID: 12120816 [PubMed - indexed for MEDLINE]

Mol Cell Biochem. 2005 Oct;278(1-2):177-84.
Effect of herbal polyphenols on atherogenic transcriptome.
Kaul D, Shukla AR, Sikand K, Dhawan V.

Department of Experimental Medicine & Biotechnology, Postgraduate Institute of Medical Education and Research, Chandigarh, India. dkaul_24@hotmail.com

The ancient Indian system of medicine supports the antiatherogenic properties of some herbs. The crosstalk amongst the genes coding for LDLR, LXRalpha, PPARs (alpha,gamma), CD-36 and c-myc may be important in atherogenesis because these genes control lipid metabolism, cytokine production and cellular activity within the arterial wall. Hence, we attempted for the first time to explore whether or not the polyphenols extracted from medicinal herbs had any effect on the transcription of these genes. Normal human mononuclear cells were cultured in the presence of polyphenols (and their HPLC purified sub-fractions) extracted from Green tea (Camellia sinensis), Neem (Azadirachta indica) and Tulsi (Ocimum sanctum). Transcriptional expression of these genes was measured by using RT-PCR and SCION IMAGE analysis software. These polyphenolic extracts were found to have the inherent capacity to inhibit the transcriptional expression of genes having direct involvement in atherogenic process. **On the basis of these results, we propose for the first time that HPLC purified polyphenolic fraction IV of Tulsi may have a profound antiatherogenic effect. (*arteriolosclerosis is any hardening (and loss of elasticity) of arterioles (small arteries), atherosclerosis is a hardening of an artery specifically due to an atheromatous plaque.)*** PMID: 16180103 [PubMed - indexed for MEDLINE]

Res Vet Sci. 2005 Aug;79(1):37-43. Epub 2004 Dec 21.

Immunotherapeutic potential of Ocimum sanctum (L) in bovine subclinical mastitis.
Mukherjee R, Dash PK, Ram GC.

Division of Medicine, Indian Veterinary Research Institute, Izatnagar, UP 243 122, India. reena@ivri.up.nic.in

Immunotherapeutic potential of aqueous extract of Ocimum sanctum (O. sanctum)leaf in bovine sub-clinical mastitis (SCM) was investigated. Somatic cell count (SCC), total bacterial count (TBC), milk differential leukocyte count (DLC), phagocytic activity and Phagocytic index and leukocyte lysosomal enzymes like myeloperoxidase and acid phosphatase content were evaluated after intramammary infusion of aqueous leaf extract of O. sanctum. The results revealed that the aqueous extract of O. sanctum treatment reduced the TBC and increased neutrophil and lymphocyte counts with enhanced phagocytic activity and phagocytic index. Similarly, the lysosomal enzymes contents of the milk polymorphonuclear cells (PMNs) were also enhanced significantly in animals treated with the extract. The results suggest that the crude aqueous extract of O. sanctum (leaf) possesses some biologically active principles that are antibacterial and immunomodulatory in nature. As such, the present wok substantiates the therapeutic use of medicinal herb and also emphasizes on the potential of the commonly available non-toxic substances to enhance the mammary immunity. PMID: 15894022 [PubMed - indexed for MEDLINE]

J Ethnopharmacol. 2003 May;86(1):113-6.
Lens aldose reductase inhibiting potential of some indigenous plants.
Halder N, Joshi S, Gupta SK.

Department of Pharmacology, All India Institute of Medical Sciences, Ansari Nagar, New Delhi 110029, India.

Cataract is the leading cause of blindness worldover. Diabetes is one of the major risk factors for cataractogenesis and aldose reductase (AR) has been reported to play an important role in sugar-induced cataract. In the present study, the AR inhibitory activity of Ocimum sanctum (OS), Withania somnifera (WS), Curcuma longa (CL), Azadirachta indica (AI) were studied together with their effect on sugar-induced cataractogenic changes in rat lenses in vitro. Aqueous extracts of the plants, procured from Dabur, India, were reconstituted with double distilled water to make various dilutions. AR inhibitory activity of these extracts and their anticataract potentials were evaluated in vitro in rat lenses. AR inhibitory activity of the aqueous extract of different plants was calculated considering the AR activity of normal rat lenses as 100%. The concentration of the plant extract that showed maximum AR inhibitory activity was selected to further study its effect on galactose-induced lens swelling and polyol accumulation in vitro. All the four plants were found to inhibit lens AR activity but to different extent. From dose-response curve, OS was found to be the most effective AR inhibitor followed by CL, AI and WS. The IC(50) values of OS, CL, AI and WS were calculated to be 20, 55, 57 and 89 microg/ml, respectively. OS showed a significant inhibition (38.05%) in polyol accumulation followed by CL and AI (28.4 and 25.04%, respectively). WS did not show any effect on polyol level in rat lenses. None of the plant extracts showed any significant effect on lens water content. **OS *(Ocimum sanctum)* possesses a significant anticataract activity in vitro and its anticataract potential could be related with its AR inhibitory effect.** PMID: 12686449 [PubMed - indexed for MEDLINE]

Schizandra Fruit (Schizandra chinensis)

Zhongguo Zhong Yao Za Zhi. 2004 Jun;29(6):568-9, 593.
[Primary research of pharmacological effects of PEC on mice]
[Article in Chinese]

Wang LW, Liu XM, Lu GH, Gao NN, Xiao PG.

Institute of Medicinal Plant Development, Chinese Academy of Medical Sciences, Beijing 100094, China.

OBJECTIVE: To study pharmacological effects of PEC (the oral liquid which consists of Panax quinquefolium, Epimedium brevicornum, Schisandra chinensis Bail and Cervus eplaphus) on mice. METHOD: Experiments were carried out through swimming test, step-through, spontaneous activity and sleeping time. RESULT: When 5-10 mL x kg(-1) of PEC was given orally for 7 days, it could prolong swimming duration of mice in water tank, and increase the

tolerant ability against oxygen-deficiency. PEC could also improve cognitive-deficiency induced by taking off sleep with force in mice after given orally for 7 days. The PEC could increase the spontaneous activity in mice, antagonize the inhabitation induced by Valium, and shorten the sleeping time caused by sodium pentobarbital. **CONCLUSION: PEC has strong potential neuro-pharmacological activities such as anti-fatigue, improving cognitive-deficiency in mice.** PMID: 15706926 [PubMed - indexed for MEDLINE]

Ginger Root (Zingiber officinale)

J Med Food. 2005 Summer;8(2):125-32.
Ginger--an herbal medicinal product with broad anti-inflammatory actions.
Grzanna R, Lindmark L, Frondoza CG.

RMG Biosciences, Inc.

The **anti-inflammatory properties of ginger** have been known and valued for centuries. During the past 25 years, many laboratories have provided scientific support for the long-held belief that ginger contains constituents with **antiinflammatory properties**. The original discovery of ginger's inhibitory effects on prostaglandin biosynthesis in the early 1970s has been repeatedly confirmed. This discovery identified ginger as an herbal medicinal product that shares pharmacological properties with non-steroidal anti-inflammatory drugs. Ginger suppresses prostaglandin synthesis through inhibition of cyclooxygenase-1 and cyclooxygenase-2. An important extension of this early work was the observation that ginger also suppresses leukotriene biosynthesis by inhibiting 5-lipoxygenase. This pharmacological property distinguishes ginger from nonsteroidal anti-inflammatory drugs. This discovery preceded the observation that dual inhibitors of cyclooxygenase and 5-lipoxygenase may have a better therapeutic profile and have fewer side effects than non-steroidal anti-inflammatory drugs. The characterization of the pharmacological properties of ginger entered a new phase with the discovery that a ginger extract (EV.EXT.77) derived from Zingiber officinale (family Zingiberaceae) and Alpina galanga (family Zingiberaceae) inhibits the induction of several genes involved in the inflammatory response. These include genes encoding cytokines, chemokines, and the inducible enzyme cyclooxygenase-2. This discovery provided the first evidence that ginger modulates biochemical pathways activated in chronic inflammation. Identification of the molecular targets of individual ginger constituents provides an opportunity to optimize and standardize ginger products with respect to their effects on specific biomarkers of inflammation. Such preparations will be useful for studies in experimental animals and humans. PMID: 16117603 [PubMed - indexed for MEDLINE]

Yakugaku Zasshi. 1992 Sep;112(9):645-55.
[Stomachic principles in ginger. II.

Pungent and anti-ulcer effects of low polar constituents isolated from ginger, the dried rhizoma of Zingiber officinale Roscoe cultivated in Taiwan. The absolute stereostructure of a new diarylheptanoid] [Article in Japanese]

Yamahara J, Hatakeyama S, Taniguchi K, Kawamura M, Yoshikawa M.

Kyoto Pharmaceutical University, Japan.

By using the effects on HCl/ethanol-induced gastric lesions in rats, beta-sesquiphellandrene (2), beta-bisabolene (3), ar-curcumene (4) and 6-shogaol (5) were isolated as anti-ulcer active principles in ginger, the dried rhizoma of Zingiber officinale Roscoe (Shokyo in Japanese) which was cultivated in Taiwan, together with nine known compounds and a new diarylheptanoid. The absolute stereostructure of the diarylheptanoid was characterized as (3S,5S)-dihydroxy 1-(4'-hydroxy-3',5'-dimethoxyphenyl)-7-(4"-hydrox y-3"-methoxyphenyl)heptane (15) on the basis of chemical and spectroscopic evidence which included the application of the benzoate chirality method. The pungent effects of several constituents isolated from ginger were examined. PMID: 1469612 [PubMed - indexed for MEDLINE]

Med Hypotheses. 1989 May;29(1):25-8.
Ginger (Zingiber officinale) and rheumatic disorders.
Srivastava KC, Mustafa T.

Department of Environmental Medicine, Odense University, Denmark.

Oxygenation of arachidonic acid is increased in inflamed tissues. In this condition products of two enzymic pathways--the cyclooxygenase and the 5-lipoxygenase producing respectively prostaglandins and leukotrienes--are elevated. Of the cyclooxygenase products, PGE2 and of the lipoxygenase products, LTB4 are the strongest candidates for mediating inflammation. Non-steroidal anti-inflammatory drugs which inhibit the cyclooxygenase, and corticosteroids are used to treat such disorders. Both types of drugs produce adverse side-effects on prolonged use. **Ginger is reported in Ayurvedic and Tibb systems of medicine to be useful in rheumatic disorders. Seven patients suffering from such disorders reported relief in pain and associated symptoms on ginger administration.** PMID: 2501634 [PubMed - indexed for MEDLINE]

BMC Complement Altern Med. 2007 Dec 20;7(1):44 [Epub ahead of print]
Ginger inhibits cell growth and modulates angiogenic factors in ovarian cancer cells.
Rhode J, Fogoros S, Zick S, Wahl H, Griffith KA, Huang J, Liu JR.

ABSTRACT: BACKGROUND: **Ginger (Zingiber officinale Rosc) is a natural dietary component with antioxidant and anticarcinogenic properties.** The ginger component [6]-gingerol has been shown to exert anti-inflammatory effects through mediation of NF-KB. NF-KB can be constitutively activated in epithelial ovarian cancer cells and may contribute towards increased transcription and translation of angiogenic factors. In the present study, we investigated the effect of ginger on tumor cell growth and modulation of angiogenic factors in ovarian cancer cells in vitro. METHODS: The effect of ginger and the major ginger components on cell growth was determined in a panel of epithelial ovarian cancer cell lines. Activation of NF-KB and and production of VEGF and IL-8 was determined in the presence or absence of ginger. RESULTS: Ginger treatment of cultured ovarian cancer cells induced profound growth inhibition in all cell lines tested. We found that in vitro, 6-shogaol is the most active of the individual ginger components tested. Ginger treatment resulted in inhibition of NF-kB activation as well as diminished secretion of VEGF and IL-8. CONCLUSION: Ginger inhibits growth and modulates secretion of angiogenic factors in ovarian cancer cells. The use of dietary agents such as ginger may have potential in the treatment and prevention of ovarian cancer. PMID: 18096028 [PubMed - as supplied by publisher]

Toxicol Lett. 2007 Sep 28;173(3):151-60. Epub 2007 Jul 28.
Pro-apoptotic effects of 1'-acetoxychavicol acetate in human breast carcinoma cells.
Campbell CT, Prince M, Landry GM, Kha V, Kleiner HE.

Department of Pharmacology, Toxicology & Neuroscience, Louisiana State University Health Sciences Center, Feist-Weiller Cancer Center, Shreveport, LA 71130, USA.

The tropical ginger compound, 1'-acetoxychavicol acetate (ACA) possesses cancer chemopreventive properties in several models but its effects on breast cancer have not been fully evaluated. In this study, the effects of ACA on human breast carcinoma-derived MCF-7 and MDA-MB-231 cell viability were assessed using trypan blue exclusion analysis. ACA significantly decreased cell viability in a time- and dose-dependent manner, with effective concentrations 10-50 microM. Apoptosis was confirmed by morphological examination of cells through light microscopy, 4,6-diamidino-2-phenylindole dihydrochloride staining, and annexin V/Alexa Fluor 488 staining visualized using flow cytometry. ACA also increased protein expression of the activated form of caspase-3 in MDA-MB-231 cells. Addition of antioxidants N-acetylcysteine, ascorbic acid, or trolox prevented the loss of viability caused by ACA using trypan blue uptake as a marker. **These results suggest ACA may have potential anticancer effects against breast carcinoma cells by inducing apoptosis.** PMID: 17766064 [PubMed - indexed for MEDLINE]

Dig Dis Sci. 2005 Oct;50(10):1889-97.
Pharmacological basis for the medicinal use of ginger in gastrointestinal disorders.
Ghayur MN, Gilani AH.

Department of Biological and Biomedical Sciences, The Aga Khan University Medical College, Karachi, 74800, Pakistan.

Ginger (rhizome of Zingiber officinale) has been widely used for centuries in gastrointestinal disorders, particularly dyspepsia, but its precise mode of action has yet to be elucidated. This study was undertaken to study the prokinetic action of ginger and its possible mechanism of action. Prokinetic activity of ginger extract (Zo.Cr) was confirmed in an in vivo test when it enhanced the intestinal travel of charcoal meal in mice. This propulsive effect of the extract, similar to that of carbachol, was blocked in atropine-pretreated mice, a standard cholinergic antagonist. Likewise, Zo.Cr showed an atropine-sensitive dose-dependent spasmogenic effect in vitro as well as in isolated rat and mouse stomach fundus tissues. In atropinized tissue, it showed spasmolytic activity as shown by the inhibition of 5-HT- and K+-induced contractions. A spasmolytic effect was also observed in other gut preparations either as noncompetitive inhibition of agonist dose-response curves, inhibition of high K+(80 mM)-induced contractions, or displacement of Ca2+ dose-response curves to the right, indicating a calcium antagonist effect. Phytochemical analysis revealed the presence of saponins, flavonoids, and alkaloids in the crude extract. **These data indicate that Zo.Cr contains a cholinergic, spasmogenic component evident in stomach fundus preparations which provides a sound mechanistic insight for the prokinetic action of ginger. In addition, the presence of a spasmolytic constituent(s) of the calcium antagonist type may explain its use in hyperactive states of gut like colic and diarrhea**. PMID: 16187193 [PubMed - indexed for MEDLINE]

J Cardiovasc Pharmacol. 2005 Jan;45(1):74-80.
Ginger lowers blood pressure through blockade of voltage-dependent calcium channels.
Ghayur MN, Gilani AH.

Department of Biological and Biomedical Sciences, The Aga Khan University Medical College, Karachi, Pakistan.

Ginger (Zingiber officinale Roscoe), a well-known spice plant, has been used traditionally in a wide variety of ailments including hypertension. We report here the cardiovascular effects of ginger under controlled experimental conditions. The crude extract of ginger (Zo.Cr) induced a dose-dependent (0.3-3 mg/kg) fall in the arterial blood pressure of anesthetized rats. In guinea pig paired atria, Zo.Cr exhibited a cardiodepressant activity on the rate and force of spontaneous contractions. In rabbit thoracic aorta preparation, Zo.Cr relaxed the phenylephrine-induced vascular contraction at a dose 10 times higher than that required against K (80 mM)-induced contraction. Ca2+ channel-blocking (CCB) activity was confirmed when Zo.Cr shifted the Ca2+ dose-response curves to the right similar to the effect of verapamil. It also inhibited the phenylephrine (1 microM) control peaks in normal-Ca2+ and Ca2+-free solution, indicating that it acts at both the membrane-bound and the intracellular Ca2+ channels. When tested in endothelium-intact rat aorta, it again relaxed the K-induced contraction at a dose 14 times less than that required for relaxing the PE-induced contraction. The vasodilator effect of Zo.Cr was endothelium-independent because it was not blocked by L-NAME (0.1 mM) or atropine (1 microM) and also was reproduced in the endothelium-denuded preparations at the same dose range**. These data indicate that the blood pressure-lowering effect of ginger is mediated through blockade of voltage-dependent calcium channels**. PMID: 15613983 [PubMed - indexed for MEDLINE]

Biochem Biophys Res Commun. 2007 Oct 12;362(1):218-23. Epub 2007 Aug 10.
Ginger ingredients reduce viability of gastric cancer cells via distinct mechanisms.
Ishiguro K, Ando T, Maeda O, Ohmiya N, Niwa Y, Kadomatsu K, Goto H.

Molecular Biology and Pathogenesis of Gastroenterology, Nagoya University School of Medicine, 65 Tsurumai-cho, Showa-ku, Nagoya, Aichi 466-8550, Japan. kio@med.nagoya-u.ac.jp

Ginger has been used throughout the world as spice, food and traditional herb. We found that 6-gingerol, a phenolic alkanone isolated from ginger, enhanced the TRAIL-induced viability reduction of gastric cancer cells while 6-

gingerol alone affected viability only slightly. 6-Gingerol facilitated TRAIL-induced apoptosis by increasing TRAIL-induced caspase-3/7 activation. 6-Gingerol was shown to down-regulate the expression of cIAP1, which suppresses caspase-3/7 activity, by inhibiting TRAIL-induced NF-kappaB activation. As 6-shogaol has a chemical structure similar to 6-gingerol, we also assessed the effect of 6-shogaol on the viability of gastric cancer cells. Unlike 6-gingerol, 6-shogaol alone reduced the viability of gastric cancer cells. 6-Shogaol was shown to damage microtubules and induce mitotic arrest. **These findings indicate for the first time that in gastric cancer cells, 6-gingerol enhances TRAIL-induced viability reduction by inhibiting TRAIL-induced NF-kappaB activation while 6-shogaol alone reduces viability by damaging microtubules**. PMID: 17706603 [PubMed - indexed for MEDLINE]

Yakugaku Zasshi. 2005 Feb;125(2):213-7.
[Antiobesity actions of Zingiber officinale Roscoe] [Article in Japanese]

Han LK, Gong XJ, Kawano S, Saito M, Kimura Y, Okuda H.

Department of Environmental and Symbiotic Sciences, Prefectural University of Kumamoto, Kumamoto City 862-8502, Japan. hanlikun@hotmail.com

Zingiber officinale Roscoe has been used as a folk medicine in China. An aqueous extract of Z. officinale Roscoe inhibited the hydrolysis of triolein emulsified with phosphatidylcholine by pancreatic lipase in vitro and it reduced the elevation of rat plasma triacylglycerol levels 1 and 2 h after oral administration of a lipid emulsion containing corn oil. These results suggest that the aqueous extract of Z. officinale Roscoe might inhibit the intestinal absorption of dietary fat by inhibiting its hydrolysis. **Therefore we investigated the antiobesity effects of the aqueous extract of Z. officinale Roscoe** by feeding a high-fat diet to mice for 8 weeks. Body weights at 2-8 weeks and final parametrial adipose tissue weights were significantly lower in mice fed the high-fat diet containing 3% aqueous extract of Z. officinale Roscoe than in the controls fed the high-fat diet. Feeding a high-fat diet containing 1% aqueous extract of Z. officinale Roscoe also significantly reduced final parametrial adipose tissue weights that were elevated in mice fed the high-fat diet alone. Our data suggest that the antiobesity effect of aqueous extract of Z. officinale Roscoe in mice fed a high-fat diet may be due in part to the inhibition of intestinal absorption of dietary fat by the active compounds of Z. officinale Roscoe. PMID: 15684576 [PubMed - indexed for MEDLINE]

Harefuah. 2006 Oct;145(10):738-42, 782.
[Herbal medicine in womens' life cycle] [Article in Hebrew]

Ben-Arye E, Oren A, Ben-Arie A.

The Complementary and Traditional Medicine Unit, Department of Family Medicine, Faculty of Medicine, Technion-Israel Institute of Technology, Haifa, Israel. eranben@netvision.net.il

Women use herbs and other traditional and complementary modalities to treat various ailments throughout their life circle. This article reviewed 19 randomized controlled trials, which studied efficacy and safety of various herbs in the treatment of premenstrual syndrome (PMS), nausea and vomiting in the first trimester of pregnancy and menopausal hot flushes. **Preliminary data support the efficacy of Chaste tree fruit (Vitex agnus) in the treatment of PMS, Ginger (Zingiber officinale) in the treatment of hyperemesis gravidarum and (Cimicifuga racemosa) in the treatment of menopausal hot flushes.** Additional and more rigorous studies are warranted in order to support the efficacy and safety of these herbal remedies. PMID: 17111709 [PubMed - indexed for MEDLINE]

J Ethnopharmacol. 1990 Jul;29(3):267-73.
Ginger (Zingiber officinale) in migraine headache.
Mustafa T, Srivastava KC.

Institute of Biology, Odense University, Denmark.

Migraine is considered as a neurological disorder with little convincing evidence of the involvement of some vascular phenomenon. Recent understanding of the mechanisms behind migraine pain generation and

perception have considerably helped the development of modern migraine drugs. Most migraine drugs in use, i.e., ergotamine and dihydroergotamine, iprazochrome, pizotifen and diazepam; and non-steroidal antiinflammatory drugs (i.e. aspirin, paracetamol, persantin, etc.) have side-effects and are prescribed with caution for a limited duration. Ginger is reported in Ayurvedic and Tibb systems of medicine to be useful in neurological disorders. It is proposed that administration of ginger may exert abortive and prophylactic effects in migraine headache without any side-effects. PMID: 2214812 [PubMed - indexed for MEDLINE]

Disclaimer: The information provided in this report is not presented with intention of diagnosing or prescribing, but is offered only as information for the use in maintaining and promoting health in cooperation with a physician.

In the event that the information presented here is used without a physician's approval, the individual will be diagnosing for him/herself.

No responsibility is assumed by the author, publisher, or distributors of this information. Should anyone use the information contained herein in lieu of a doctor's services.

No guarantees of any kind are made for the performance or effectiveness of these superfoods.

The studies for the abstract in this report were found at www.pubmed.gov. www.pubmed.gov is a service of the U.S. National Library of Medicine and the National Institutes of Health.

Hear is some information that may be contrary to what you have heard over the years. Speak to your healthcare provider before you drink another glass of milk or eat another piece of cheese or yogurt. I have done a lot of research to find you **clinical studies to show you that science is finding that milk DOES NOT do a body good.** Here is why:

The following references provide converging lines of evidence that focus upon one central point.
There are hundreds of millions of different proteins in nature, and only one hormone that is identical between any two species. That powerful growth hormone is insulin-like growth factor, or IGF-I. IGF-I survives digestion and has been identified as the KEY FACTOR in breast cancer's growth. IGF-I is identical in human and cow.

Br J Cancer. 2007; 96 Suppl:R2-6. **Republished from:** Br J Cancer. 2005 Jun 20; 92(12):2097-101.

Role of insulin-like growth factor 1 receptor signalling in cancer.-Larsson O, Girnita A, Girnita L.

Department of Oncology and Pathology, CCK R8:04, Karolinska Hospital, S-171 76 Stockholm, Sweden. olle.larsson@onkpat.ki.se

The insulin-like growth factor (IGF-1) signalling is highly implicated in cancer. In this signalling the IGF-1 receptor (IGF-1R) is unquestionable, the predominating single factor. IGF-1R is crucial for tumour transformation and survival of malignant cell, but is only partially involved in normal cell growth. This is in part due to the interactions with oncogenes. Recent findings suggest a close interplay with the p53/MDM2 pathway. Disturbances in components in the p53/MDM2/IGF-1R network may cause IGF-1R upregulation and growth advantage for the cancer cell. Targeting of IGF-1R is more and more seen as a promising option for future cancer therapy. Single chain antibodies and small molecules with selective effects on IGF-1R dependent malignant growth are of particular interest. Forthcoming clinical trials are welcome and will indeed be the only way to evaluate the impact of IGF-1R targeting in human cancer. PMID: 17393577 [PubMed]

Cancer Epidemiol Biomarkers Prev. 2007 Mar;16(3):598-605.

Direct inhibition of insulin-like growth factor-I receptor kinase activity by (-)-epigallocatechin-3-gallate regulates cell transformation. Li M, He Z, Ermakova S, Zheng D, Tang F, Cho YY, Zhu F, Ma WY, Sham Y, Rogozin EA,Bode AM, Cao Y, Dong Z.

Hormel Institute, University of Minnesota, 801 16th Avenue Northeast, Austin, MN 55912, USA.
Insulin-like growth factor-I receptor (IGF-IR) has been implicated in cancer pathophysiology. Furthermore, impairment of IGF-IR signaling in various cancer cell lines caused inhibition of the transformed phenotype as determined by the inhibition of colony formation in soft agar and the inhibition of tumor formation in athymic nude mice. Thus, the IGF-IR might be an attractive target for cancer prevention. We showed that the tea polyphenol, (-)-epigallocatechin-3-gallate (EGCG), is a small-molecule inhibitor of IGF-IR activity (IC50 of 14 micromol/L). EGCG abrogated anchorage-independent growth induced by IGF-IR overexpression and also prevented human breast and cervical cancer cell phenotype expression through inhibition of IGF-IR downstream signaling. Our findings are the first to show that the IGF-IR is a novel binding protein of EGCG and thus may help explain the chemopreventive effect of EGCG on cancer development. PMID: 17372258 [PubMed - in process] **NOTE: One place EGCG can be found is in green tea.**

Pharmacol Res. 2007 Feb 3;

Doping with growth hormone/IGF-1, anabolic steroids or erythropoietin: is there a cancer risk?
Tentori L, Graziani G.

Department of Neuroscience, University of Rome "Tor Vergata", Via Montpellier 1, 00133 Rome, Italy.

Anabolic steroid and peptide hormones or growth factors are utilized to increase the performance of athletes of professional or amateur sports. Despite their well-documented adverse effects, the use of some of these agents has significantly grown and has been extended also to non-athletes with the aim to improve appearance or to counteract ageing. Pre-clinical studies and epidemiological observations in patients with an excess of hormone production or in patients chronically treated with hormones/growth factors for various pathologies have warned about the potential risk of cancer development and progression which may be also associated to the use of certain doping agents. Anabolic steroids have been described to provoke liver tumours; growth hormone or high levels of its mediator insulin-like growth factor-1 (IGF-1) have been associated with colon, breast, and prostate cancers. Actually, IGF-1 promotes cell cycle progression and inhibits apoptosis (cell death) either by triggering other growth factors or by interacting with pathways which have an established role in carcinogenesis and cancer promotion. More recently, the finding that erythropoietin (Epo) may promote angiogenesis and inhibit apoptosis or modulate chemo- or radiosensitivity in cancer cells expressing the Epo receptor, raised the concern that the use of recombinant Epo to increase tissue oxygenation might favour tumour survival and aggressiveness. Cancer risk associated to doping might be higher than that of patients using hormones/growth factors as replacement therapy, since enormous doses are taken by the athletes often for a long period of time. Moreover, these substances are often used in combination with other licit or illicit drugs and this renders almost unpredictable all the possible adverse effects including cancer. **Anyway, athletes should be made aware that long-term treatment with doping agents might increase the risk of developing cancer**. PMID: 17349798 [PubMed - as supplied by publisher]
Cell Signal. 2007 Feb 8; [Epub ahead of print]

Insulin-like growth factor-I induces cyclooxygenase-2 expression via PI3K, MAPK and PKC signaling pathways in human ovarian cancer cells.-Cao Z, Liu LZ, Dixon DA, Zheng JZ, Chandran B, Jiang BH.

Mary Babb Randolph Cancer Center, Department of Microbiology, Immunology and Cell Biology, West Virginia University, Morgantown, WV 26506-9300, United States.

Elevated levels of insulin-like growth factor-I (IGF-I) are associated with ovarian carcinogenesis and progression. However, the molecular mechanisms by which IGF-I contributes to ovarian cancer development remain to be elucidated. Cyclooxygenase-2 (COX-2) is a crucial player in the pathogenesis of human malignancies. Herein we showed that IGF-I efficiently induced COX-2 expression and PGE(2) biosynthesis at physiologically relevant concentrations in human ovarian cancer cells. IGF-I treatment significantly increased COX-2 transcriptional activation. IGF-I also stabilized COX-2 mRNA through the COX-2 3'-untranslated region (3'-UTR), which appeared independent of the conserved AU-rich elements. We next investigated the signaling pathways involved in IGF-I-induced COX-2 expression. We found that PI3K inhibitor wortmannin or LY294002 blocked COX-2 expression induced by IGF-I. Wortmannin treatment or a dominant negative PI3K mutant significantly inhibited IGF-I-induced COX-2 mRNA stabilization, but only slightly decreased COX-2 transcriptional activation. We showed that ERK1/2

and p38 MAPKs were required for IGF-I-induced COX-2 expression and that activation of both pathways by IGF-I increased COX-2 transcriptional activation and its mRNA stability. IGF-I stimulated PKC activation in the cells and pretreatment with PKC inhibitor bisindolylmaleimide prevented IGF-I-induced COX-2 transcriptional activation and mRNA stabilization, and inhibited COX-2 mRNA and protein expression. Taken together, our data demonstrate that IGF-I induces COX-2 expression in human ovarian cancer cells, which is mediated by three parallel signaling cascades - PI3K, MAPK, and PKC pathways that differentially regulate COX-2 expression at transcriptional and post-transcriptional levels. PMID: 17341442 [PubMed - as supplied by publisher]

Cancer Res. 2007 Feb 15;67(4):1520-6.

Insulin-like growth factor-1 (IGF-1) induces WISP-2/CCN5 via multiple molecular cross-talks and is essential for mitogenic switch by IGF-1 axis in estrogen receptor-positive breast tumor cells.
Dhar K, Banerjee S, Dhar G, Sengupta K, Banerjee SK.

Cancer Research Unit, VA Medical Center, Kansas City, MO 64128, USA.

Previously, we have shown that the expression of Wnt-1-induced signaling protein-2 (WISP-2), also known as CCN5, can be regulated by multiple stimulants in estrogen receptor (ER)-positive breast tumor cells to exert their mitogenic action in these cells. Here, we show that insulin-like growth factor-1 (IGF-1), a strong mitogen, enhanced the expression of the WISP-2/CCN5 gene parallel with the induction of proliferation of ER-positive breast tumor cells. An additive effect was also seen in combination with estrogen. Perturbation of IGF-1-induced WISP-2/CCN5 expression by WISP-2-specific RNA interference impaired the mitogenic action of IGF-1 on ER-positive breast tumor cells. Furthermore, the studies have shown that the multiple molecular cross-talks and side-talks among IGF-1R, ER-alpha, and phosphatidylinositol 3-kinase (PI3K)/Akt signaling molecules are required to induce WISP-2/CCN5 mRNA by IGF-1 in ER-positive, noninvasive breast tumor cells. Because a pure anti-ER ICI 182,780 is not only able to suppress the up-regulation of WISP-2/CCN5 mRNA expression by IGF-1, it also suppresses the PI3K/Akt activity induced by IGF-1 in MCF-7 cells; we anticipate that the membrane ER receptor may participate in this event. Collectively, these studies propose for the first time that WISP-2/CCN5 is an integral signaling molecule in mitogenic action of IGF-1 axis in ER-positive human breast tumor cells. PMID: 17308090 [PubMed - indexed for MEDLINE]

Cancer Epidemiol Biomarkers Prev. 2006 Mar;15(3):449-55.

The insulin-like growth factor system and mammographic features in premenopausal and postmenopausal women.dos Santos Silva I, Johnson N, De Stavola B, Torres-Mejia G, Fletcher O, Allen DS,Allen NE, Key TJ, Fentiman IS, Holly JM, Peto J.

Department of Epidemiology and Population Health, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT, United Kingdom. isabel.silva@lshtm.ac.uk
High levels of circulating insulin-like growth factor-I (IGF-I) and its major binding protein (IGFBP-3) at pre-menopausal ages have been associated with an increased breast cancer risk. We conducted a cross-sectional study (215 premenopausal women and 241 after natural menopause) nested within the Guernsey prospective studies to examine the relationship between the IGF system and mammographic features of the breast. The mammographically dense area in the breast increased with increasing serum levels of IGF-I (P for linear trend, P(t) = 0.05), IGF-II (P(t) = 0.08), and IGFBP-3 (P(t) = 0.01) only in premenopausal women. IGF-II and IGFBP-3 serum levels were associated with increases in the mammographically lucent area in both premenopausal (P(t) = 0.01 and 0.04, respectively) and postmenopausal women (P(t) < 0.001 for both), but these associations were no longer statistically significant after adjustment for body mass index and waist circumference. Neither the IGF-I/IGFBP-3 nor the IGF-II/IGFBP-3 molar ratio was associated with any of these mammographic features. The number of A alleles at a polymorphic locus in the promoter region of the IGFBP-3 gene was associated with increasing mean IGFBP-3 levels in both premenopausal (P(t) = 0.01) and postmenopausal (P(t) <0.001) women but not with mammographically dense area. These results support the hypothesis that the IGF system may affect the amount of mammographically dense tissue in premenopausal women, possibly by promoting cell proliferation and inhibiting apoptosis in the fibroglandular tissue. The findings also show strong relations

between IGF-II and IGFBP-3 levels and the amount of mammographically lucent tissue, reflecting the associations between body adiposity and amount of fat tissue in the breast and between body adiposity and circulating levels of these growth factors.PMID: 16537700 [PubMed - indexed for MEDLINE]

Int J Oncol. 2006 Mar;28(3):723-30.

Insulin-like growth factor-I promotes migration in human androgen-independent prostate cancer cells via the alphavbeta3 integrin and PI3-K/Akt signaling.-Marelli MM, Moretti RM, Procacci P, Motta M, Limonta P.

Center for Endocrinological Oncology, Institute of Endocrinology, University of Milano, I-20133 Milano, Italy.

In its phase of androgen-independence, prostate carcinoma is characterized by a high proliferation rate and by a strong ability to give rise to metastases. **IGF-I has been shown to exert a potent mitogenic action on prostate cancer.** We investigated whether IGF-I might also affect the motility of prostate cancer cells and defined the mechanism of action. We found that IGF-I promotes the migratory capacity of androgen-independent prostate cancer cells through the activation of its specific receptor, IGF-IR. This effect was accompanied by a change in cell morphology (as revealed by scanning electron microscopy), and by a rearrangement of the actin cytoskeleton. The treatment of cells with the PI3-K inhibitor, LY294002, counteracted the pro-migratory activity of IGF-I. Experiments were then performed to clarify whether the integrin, alphavbeta3, could be involved in the action of IGF-I. We demonstrated that: a) the IGF-I-induced migration of cells is completely antagonized by an antibody specifically blocking the function of alphavbeta3; b) IGF-I increases alphavbeta3 immunofluorescence at the level of cell membranes, and this effect is counteracted by LY294002; and c) IGF-I increases alphavbeta3 protein levels. Our results demonstrate that IGF-I promotes the motility of androgen-independent prostate cancer cells by modulating alphavbeta3 integrin activation/expression; these effects are mediated by the PI3-K/Akt signaling pathway. **This study: a) supports a crucial role for IGF-I in the progression of the pathology towards the highly metastatic phase; and b) provides an additional rationale basis for the development of therapeutic strategies directed at the IGF-I/IGF-IR system in the treatment of androgen-independent prostate cancer.** PMID: 16465378 [PubMed - indexed for MEDLINE]
Med Hypotheses. 2005;65(6):1028-37. Epub 2005 Aug 24.

The possible role of female sex hormones in milk from pregnant cows in the development of breast, ovarian and corpus uteri cancers.-Ganmaa D, Sato A.

Department of Environmental Health, Medical University of Yamanashi, Tamaho, Yamanashi 409-3898, Japan.
The continued increase in incidence of some hormone-related cancers worldwide is of great concern. Although estrogen-like substances in the environment were blamed for this increase, the possible role of endogenous estrogens from food has not been widely discussed. We are particularly concerned about cows' milk, which contains a considerable quantity of estrogens. When we name cows' milk as one of the important routes of human exposure to estrogens, the general response of Western people is that "man has been drinking cows' milk for around 2000 years without apparent harm." However, the milk that we are now consuming is quite different from that consumed 100 years ago. Unlike their pasture-fed counterparts of 100 years ago, modern dairy cows are usually pregnant and continue to lactate during the latter half of pregnancy, when the concentration of estrogens in blood, and hence in milk, increases. The correlation of incidence and mortality rates with environmental variables in worldwide countries provides useful clues to the etiology of cancer. In this study, we correlated incidence rates for breast, ovarian, and corpus uteri cancers (1993-97 from Cancer Incidence in Five Continents) with food intake (1961-97 from FAOSTAT) in 40 countries. Meat was most closely correlated with the breast cancer incidence (r=0.827), followed by milk (0.817) and cheese (0.751). Stepwise multiple-regression analysis (SMRA) identified meat as the factor contributing most greatly to the incidence of breast cancer ([R]=0.862). Milk was most closely correlated with the incidence of ovarian cancer (r=0.779), followed by animal fats (0.717) and cheese (0.697). SMRA revealed that milk plus cheese make the greatest contribution to the incidence of ovarian cancer ([R]=0.767). Milk was most closely correlated with corpus uteri cancer (r=0.814), followed by cheese (0.787). SMRA revealed that milk plus cheese make the most significant contribution to the incidence of corpus uteri cancer ([R]=0.861). **In conclusion, increased consumption of animal-derived food may have adverse effects on the development of hormone-dependent cancers. Among dietary risk factors, we are most concerned with milk and dairy products, because the milk we drink today is produced from pregnant cows, in which estrogen and progesterone levels are markedly elevated**. PMID: 16125328 [PubMed - indexed for MEDLINE]

Med Hypotheses. 2001 Oct;57(4):510-4.

Is milk responsible for male reproductive disorders?-Ganmaa D, **Wang PY**, **Qin LQ**, **Hoshi K**, **Sato A**.

Department of Environmental Health, Medical University of Yamanashi, Tamaho, Yamanashi 409-3898, Japan.

The role of environmental compounds with estrogenic activity in the development of male reproductive disorders has been a source of great concern. Among the routes of human exposure to estrogens, we are particularly concerned about cows' milk, which contains considerable amounts of estrogens. The major sources of animal-derived estrogens in the human diet are milk and dairy products, which account for 60-70% of the estrogens consumed. Humans consume milk obtained from heifers in the latter half of pregnancy, when the estrogen levels in cows are markedly elevated. The milk that we now consume may be quite unlike that consumed 100 years ago. Modern genetically-improved dairy cows, such as the Holstein, are usually fed a combination of grass and concentrates (grain/protein mixes and various by-products), allowing them to lactate during the latter half of pregnancy, even at 220 days of gestation. **We hypothesize that milk is responsible, at least in part, for some male reproductive disorders.** Copyright 2001 Harcourt Publishers Ltd. PMID: 11601881 [PubMed - indexed for MEDLINE]

Med Hypotheses. 1997 Jun;48(6):453-61.-Dairy products and breast cancer: the IGF-I, estrogen, and bGH hypothesis.
Outwater JL, Nicholson A, Barnard N.-A. B. Princeton University 1996, Physicians Committee For Responsible Medicine, Washington, DC 20016, USA.

Research on the role of dietary factors in breast cancer causation has focused predominantly on fat intake. **While some studies have examined associations between breast cancer rates and consumption of whole milk, there has been less attention given to dairy products in general. Dairy products contain both hormones and growth factors, in addition to fat and various chemical contaminants that have been implicated in the proliferation of human breast cancer cells.** This literature review evaluates the epidemiological and mechanistic evidence linking dairy consumption with breast cancer risk. Publication Types: • Review-PMID: 9247884 [PubMed - indexed for MEDLINE]

"Human Insulin-like growth factor (IGF-I) and bovine IGF-I are identical. Both contain 70 amino acids in the identical sequence."-Judith C. Juskevich and C. Greg Guyer. SCIENCE, vol. 249. August 24, 1990.

If you believe that breastfeeding "works" to protect lactoferrins and immunoglobulins from digestion (and benefit the nursing infant), you must also recognize that milk is a hormonal delivery system. By drinking cow's milk, one delivers IGF-I in a bioactive form to the body's cells. When IGF-I from cow's milk alights upon an existing cancer...

Nat Med. 2005 Feb; 11(2):127-9. Epub 2005 Jan 30.

The breast cancer resistance protein BCRP (ABCG2) concentrates drugs and carcinogenic xenotoxins into milk.

Jonker JW, Merino G, Musters S, van Herwaarden AE, Bolscher E, Wagenaar E, Mesman E, Dale TC, Schinkel AH.-The Netherlands Cancer Institute, Division of Experimental Therapy, Amsterdam, The Netherlands.

Contamination of milk with drugs, pesticides and other xenotoxins can pose a major health risk to breast-fed infants and dairy consumers. Here we show that the multidrug transporter BCRP (encoded by ABCG2) is strongly induced in the mammary gland of mice, cows, and humans during lactation and that it is responsible for the active secretion of clinically and toxicologically important substrates such as the dietary carcinogen PhIP, the anticancer drug topotecan and the antiulcerative cimetidine into mouse milk. PMID: 15685169 [PubMed - in process]

Br J Cancer. 2005 Mar 8; A prospective study of serum insulin-like growth factor-I (IGF-I), IGF-II, IGF-binding protein-3 and breast cancer risk.

Allen NE, Roddam AW, Allen DS, Fentiman IS, Dos Santos Silva I, Peto J, Holly JM, Key TJ.-1Cancer Research UK Epidemiology Unit, University of Oxford, Radcliffe Infirmary, Oxford OX2 6HE, UK.

The associations between serum concentrations of insulin-like growth factor-I (IGF-I), IGF-II and IGF-binding proteins (IGFBP)-3 and risk of breast cancer were investigated in a nested case-control study involving 117 cases (70 premenopausal and 47 postmenopausal at blood collection) and 350 matched controls within a cohort of women from the island of Guernsey, UK. Women using exogenous hormones at the time of blood collection were excluded. Premenopausal women in the top vs bottom third of serum IGF-I concentration had a nonsignificantly increased risk for breast cancer after adjustment for IGFBP-3 (odds ratio (OR) 1.71; 95% confidence interval (CI): 0.74-3.95; test for linear trend, P=0.21). Serum IGFBP-3 was associated with a reduction in risk in premenopausal women after adjustment for IGF-I (top third vs the bottom third: OR 0.49; 95% CI: 0.21-1.12, P for trend=0.07). Neither IGF-I nor IGFBP-3 was associated with risk in postmenopausal women and serum IGF-II concentration was not associated with risk in pre- or postmenopausal women. **These data are compatible with the hypothesis that premenopausal women with a relatively high circulating concentration of IGF-I and low IGFBP-3 are at an increased risk of developing breast cancer.** British Journal of Cancer advance online publication, 8 March 2005; doi:10.1038/sj.bjc.6602471 **www.bjcancer.com.** PMID: 15756268 [PubMed - as supplied by publisher]

Med Hypotheses. 2004;62(1):133-42.-Estrogen: one of the risk factors in milk for prostate cancer.-Qin LQ, Wang PY, Kaneko T, Hoshi K, Sato A.-Department of Environmental Health,
School of Medicine, University of Yamanashi, Shimokato 1110, Tamaho, Yamanashi 409-3898, Japan. shinr@res.yamanashi-med.ac.jp

Studies to elucidate the cause of prostate cancer have met with little success to date. Epidemiological studies suggested that milk consumption is probably as one of the risk factors for prostate cancer. The studies thus focused on the fat and calcium in milk, but reached no definitive conclusion. **According to the measurements of estrogen levels in milk by different studies, it was suggested that estrogen in milk was a possible risk to cause prostate cancer.** One reason supporting this hypothesis is that Western diet (characterized by milk/dairy products and meat) causes a trend of increasing levels of estrogens, and Western males show a higher incidence rate of prostate cancer than Asia males.

Estrogen levels in prostate fluid are also correlated very well with the prostate cancer. During several decades, estrogens, together with testosterone, was commonly used to induce the rodent model of prostate cancer. Our hypothesis also was supported by the presence of estrogen receptors in the prostate gland and the genotoxic role of estrogens on the prostate gland, as possible mechanisms. **Therefore, if modern milk consumption does expose consumers to high levels of estrogen and plays an adverse role in prostate cancer, action should be taken to produce the noncontaminant milk.**-PMID: 14729019 [PubMed - indexed for MEDLINE]

J Natl Cancer Inst 2001 Sep 5;93(17):1330-6, Milk intake, circulating levels of insulin-like growth factor-I, and risk of colorectal cancer in men.

Ma J, Giovannucci E, Pollak M, Chan JM, Gaziano JM, Willett W, Stampfer MJ. Channing Laboratory, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA 02115, USAmailto:jing.ma@channing.harvard.edu

"BACKGROUND: Milk and dietary calcium may have antiproliferative effects against colorectal cancer, but milk intake also raises serum levels of insulin-like growth factor-I (IGF-I). A high ratio of IGF-I to IGF-binding protein-3 (IGFBP-3) has been linked to an increased risk of colorectal cancer. …CONCLUSION: Intake of dairy products was associated with a modest increase in circulating IGF-I levels, but intake of low-fat milk was associated with lower risk of colorectal cancer, particularly among individuals with high IGF-I/IGFBP-3. This subpopulation, which is at increased risk of colorectal cancer, might benefit the most from specific dietary intervention." PMID: 11535708 [PubMed - indexed for MEDLINE]

Am J Clin Nutr 2001 Oct;74(4):549-54- Dairy products, calcium, and prostate cancer risk in the Physicians' Health Study. Chan JM, Stampfer MJ, Ma J, Gann PH, Gaziano JM, Giovannucci EL.
Department of Nutrition, Harvard School of Public Health, Boston, USA **mailto:june.chan@channing.harvard.edu**

BACKGROUND: A high calcium intake, mainly from dairy products, may increase prostate cancer risk by lowering concentrations of 1,25-dihydroxyvitamin D(3) [1,25(OH)(2)D(3)], a hormone thought to protect against prostate cancer. The results of epidemiologic studies of this hypothesis are inconclusive. OBJECTIVE: We investigated the association between dairy product and calcium intakes and prostate cancer risk in the Physicians' Health Study, a cohort of male US physicians... **CONCLUSIONS: These results support the hypothesis that dairy products and calcium are associated with a greater risk of prostate cancer**.-PMID: 11566656 [PubMed - indexed for MEDLINE]

Med Hypotheses 1997 Jun;48(6):453-61-Dairy products and breast cancer: the IGF-I, estrogen, and bGH hypothesis.-Outwater JL, Nicholson A, Barnard N. A. B. Princeton University 1996, Physicians Committee For Responsible Medicine, Washington, DC 20016, USA.

"Research on the role of dietary factors in breast cancer causation has focused predominantly on fat intake. While some studies have examined associations between breast cancer rates and consumption of whole milk, there has been less attention given to dairy products in general. **Dairy products contain both hormones and growth factors, in addition to fat and various chemical contaminants that have been implicated in the proliferation of human breast cancer cells. This literature review evaluates the epidemiological and mechanistic evidence linking dairy consumption with breast cancer risk."** Publication Types: Review, Review, Academic, PMID: 9247884 [PubMed - indexed for MEDLINE]

Int J Health Serv 1996;26(1):173-85-Unlabeled milk from cows treated with biosynthetic growth hormones: a case of regulatory abdication. Epstein SS. School of Public Health West, University of Illinois, Chicago 60612, USA.

"Levels of insulin-like growth factor-1 (IGF-1) are substantially elevated and more bioactive in the milk of cows hyperstimulated with the biosynthetic bovine growth hormones rBGH, and are further increased by pasteurization. IGF-1 is absorbed from the gastrointestinal tract, as evidenced by marked growth-promoting effects even in short-term tests in mature rats, and absorption is likely to be still higher in infants. **Converging lines of evidence incriminate IGF-1 in rBGH milk as a potential risk factor for both breast and gastrointestinal cancers."** Publication Types: Review, Review, Tutorial PMID: 8932606 [PubMed - indexed for MEDLINE]

Adv Exp Med Biol 1999;472:29-42 Nutritional factors in human cancers. Giovannucci E. Channing Laboratory, Department of Medicine, Brigham and Women's Hospital, Boston, Massachusetts 02115, USA.

"A variety of external factors interacting with genetic susceptibility influence the carcinogenesis process. External factors including oxidative compounds, electrophilic agents, and chronic infections may enhance genetic damage. **In addition, various hormonal factors, which influence growth and differentiation, are critically important in the carcinogenic process.** Diet and nutrition can influence these processes directly in the gastrointestinal tract by providing bioactive compounds to specific tissues via the circulatory system, or by modulating hormone levels. **Differences in certain dietary patterns among populations explain a substantial proportion of cancers of the colon, prostate and breast. These malignancies are largely influenced by a combination of factors related to diet and nutrition.** Their causes are multifactorial and complex, but a major influence is the widespread availability of energy-dense, highly processed and refined foods that are also deplete in fiber. **These dietary patterns in combination with physical inactivity contribute to obesity and metabolic consequences such as increased levels of IGF-1, insulin, estrogen, and possibly testosterone. These hormones tend to promote cellular growth. For prostate cancer, epidemiologic studies consistently show a positive association with high consumption of milk, dairy products, and meats.** These dietary factors tend to decrease 1.25(OH)2 vitamin D, a cell differentiator, and low levels of this hormone may enhance prostate carcinogenesis. **While the nutritional modulation of growth-enhancing and differentiating hormones is likely to contribute to the high prevalence of breast, colorectal, prostate, and several other cancers in the Western world, these cancers are relatively rare in less economically developed countries, where malignancies of the upper gastrointestinal tract are quite common. The major causes of upper gastrointestinal tract cancers are likely related to various food practices or preservation**

methods other than refrigeration, which increase exposure to irritants or arcinogens." Publication Types: Review, Review, Academic PMID: 10736613 [PubMed - indexed for MEDLINE]

Oncogene 2001 Nov 8;20(51):7542-50-Insulin-like growth factor I stimulates motility in human neuroblastoma cells.

Meyer GE, Shelden E, Kim B, Feldman EL. Neuroscience Program, University of Michigan, 4414 Kresge III, 200 Zina Pitcher Place, Ann Arbor, MI 48109, USA.

"Motility is an important process that contributes to cancer cell spread. Growth factors are key regulators of motility in many cell types. Insulin-like growth factor I (IGF-I) causes SH-SY5Y human neuroblastoma cells to undergo dynamic morphological changes… These results delineate some of the proximal events in the signaling mechanism utilized by IGF-I to stimulate cell motility." - PMID: 11709726 [PubMed - indexed for MEDLINE]

Wien Med Wochenschr 2001;151(18-20):426-9-[Growth hormone in the elderly man] [Article in German]-Riedl M, Kotzmann H, Luger A.-Klinische Abteilung fur Endokrinologie und Stoffwechsel, Universitatsklinik fur Innere Medizin III, Wahringer Gurtel-18-20, A-1090 Wien.

"Many symptoms being part of the growth hormone deficiency syndrome in adults like decrease in muscle mass and bone mineral content, increase in fat mass, and skin atrophy are observed also with ageing. Indeed, short-term trials with growth hormone administration to persons over 60 years old revealed that many of these symptoms could be reversed by growth hormone. **However, recent reports of an association of high insulin-like growth factor-1 (IGF-1)-concentrations and increased risk of prostate, lung, colon and breast cancer as well as a possible decrease of insulin sensitivity prohibit currently the use of growth hormone in an attempt to reverse a normal ageing process..."** PMID: 11817251 [PubMed - indexed for MEDLINE]

Cell Growth Differ 2002 Feb;13(2):87-93-Insulin-like growth factor-I-induced migration of melanoma cells is mediated by interleukin-8 induction. Satyamoorthy K, Li G, Vaidya B, Kalabis J, Herlyn M. Wistar Institute, Philadelphia, Pennsylvania 19104, USA.

Urology 2002 Apr;59(4 Suppl 1):4-8-Mind-body effect: insulinlike growth factor-1; clinical depression; and breast, prostate, and other cancer risk-an unmeasured and masked mediator of potential significance?

Moyad MA, Pienta KJ.-Department of Urology, University of Michigan Medical Center, Ann Arbor, Michigan 48109-0330, USA. -mailto:moyad@umich.edu

"A possible relation may exist between higher insulinlike growth factor (IGF)-1 levels and the risk of premenopausal breast, prostate, or other cancers from recent prospective and case-control studies. Separately, a large prospective study has shown a potential association between chronic depression and cancer risk, whereas other preliminary studies have suggested a link between increasing IGF levels with major depression. … Depression has not been included in this list of potential factors that may need to be considered when analyzing IGF-1 data and cancer risks. The time seems ripe to at least define further the relation, if any, between IGF-1 and depression." - Publication Types: Review -Review, Tutorial-PMID: 11937431 [PubMed - indexed for MEDLINE]

By this time you are probably saying to yourself, "Enough already about milk and IGF-1". **The reason that I am over-emphasizing these clinical studies is to show you that there is science that basically says that cows' milk should be left for cows only. NOT HUMANS.**

One more thing about milk; Milk also contains somatic cells, (white blood cells) also known as pus cells. If you ever had a wound that had a yellowish white material oozing from it, that material contains somatic (pus) cells. Somatic cells are present in cows' milk because of the infections that cows get. The legal limit in the United States is 750,000 somatic cells per ml, which is approximately 3,000,000 somatic cells per 8 oz. glass of milk. **Since it takes 10 ounces of milk to equal 1 ounce of cheese, than YOU COULD BE EATING APPROXIMATELY 3.75 MILLION SOMATIC (PUS) CELLS WITH EVERY OUNCE OF CHEESE THAT YOU EAT. SOUNDS APPETIZING, DOESN'T IT?**

Aspartame and Cancer

I received this e-mail about Aspartame and Cancer on 6/18/07 and I felt it should be included in this report.

New Study - LOW DOSES Of Aspartame Cause CANCER-From Dr. Betty Martini, D.Hum. 6-15-7

Statement from Dr. Russell Blaylock, MD

Dear Betty,

My review of the first Ramazzini Study concluded that the study was one of the best designed, comprehensive, and conclusive studies done to date on the multipotent carcinogenic potential of aspartame.

This second study is even more conclusive, in that **it shows a dose-dependent statistically significant increase in lymphomas/ leukemia in both male and female rats exposed to aspartame. These two cancers are the fastest growing cancers in people under age 30.**

Also, of major concern is their finding of statistically significant increases in breast cancer in animals exposed to aspartame. With newer studies clearly indicating that toxic exposures during fetal development can dramatically increase the cancer risk of the offspring, this study takes on a very important meaning to all pregnant women consuming aspartame products. Likewise, small children are at considerable risk of the later development of these highly fatal cancers.

It should be appreciated that the doses used in these study fall within the range of doses seen in everyday users of aspartame. This study, along with the first study, should convince any reasonable scientific mind, as well as the public at large, that this product should be removed from the market. -Russell L. Blaylock, M.D.

New Study - Low Doses Of Aspartame Cause Cancer

Environmental Health Perspectives, the Journal of the U.S. National Institute of Environmental Health Sciences ranks first among more than 200 environmental science and occupational health journals and is read in 190 countries.

At New York's Mt Sinai School of Medicine DR. MORANDO SOFFRITTI was honored in April with the Irving J Selikoff Award for Outstanding contributions to the identification of environmental and industrial carcinogens, and his promotion of independent scientific research.

The prestigious Selikoff Award is only granted for groundbreaking cancer research. It was created 1993 by the Collegium Ramazzini, an academy of 180 internationally renowned experts in occupational and environmental health from over 30 nations. It has been awarded just twice before being presented to Dr. Soffritti.

His research was conducted for 36 months using 1,800 rats. It forced the conclusion that aspartame is a multipotential carcinogen. **Cancers aspartame produced included leukemia, lymphoma, kidney, and cranial peripheral nerves among others. Only the rats fed aspartame got malignant brain tumors.** This prodigious work was peer reviewed by 7 world experts.

This work confirmed studies presented to the FDA 25 years ago that documented a catalogue of brain, uterine, ovarian, testicular, mammary, pancreatic and thyroid tumors. Based on the evidence FDA denied approval of aspartame for 16 years from the time it was discovered. Then Don Rumsfeld, who was the CEO of NutraSweet's parent, the G. D. Searle Co., went to Washington to be newly elected Ronald Reagan's Secretary of Defense. The existing FDA Commissioner was asked to resign and aspartame was approved by Arthur Hull Hayes appointed by President Reagan. He did this over the objections of the FDA's scientific Board of Inquiry who had revoked the petition.

President Reagan had written an executive order making FDA powerless to do anything about aspartame until Hayes could get to FDA. Now the floundering Searle became profitable. The compromised FDA had full knowledge of aspartame's toxicity and the approval of this deadly carcinogen sentenced millions to disability and death.

An Atlanta Journal Constitution article dated 9/25/85 was titled "Reagan Says He Quit Using Sweeteners". It was in l985 there were two congressional hearings over the outrage of the public being poisoned. In this article it states: "President Reagan says he quit using artificial sweeteners because "we don't know what is in them,"... Unfortunately it was too late for the public as the U.S. Circuit Court of Appeals for the District of Columbia ruled in favor of NutraSweet, even though it was known that the FDA had asked for the indictment for Searle, and revoked the petition for approval.

Soffritti's new study "Lifespan Exposure to Low Doses of Aspartame Beginning During Prenatal Life Increases Cancer Effects in Rats" focused on damage from low-dosage aspartame consumption over the long term and clearly demonstrated a great danger to unborn babies and children. **Newly identified is risk of breast cancer as the aspartame-using child matures.**

Exposures were at low doses. A 20 kg (45 pounds) child drinking 2 cans of diet soda a day brings into his body 400 mg. of aspartame.

Food & Drink Weekly reports that soda is the most commonly consumed beverage among children and soft drink consumption is up 500% over the last 50 years. American consumption has skyrocketed to over 600 12-ounce servings per person per year, and climbing. Cokes goal is to jump sales 25% annually.

The aim of Soffritti's new study was to identify the cancer risk aspartame presents, starting with the mother's ingestion before the fetus is born. The study was conducted on groups of 70-95 male and female Sprague Dawley rats, administered aspartame with feed at concentrations of 2000, 400, or 0 ppm from the 12th day of fetal life until natural death.
The results of this carcinogenicity bioassay confirm and reinforce the earlier studys demonstration of aspartame's multipotential carcinogenicity. Further, the study demonstrates that when lifespan exposure to aspartame begins during fetal life, its carcinogenic effects magnify.

Dr. Philip Landrigan, Chairman of Community and Environmental Medicine at Mt. Sinai Medical Center, says Dr. Soffrittis study on rats strongly implies human risk. **He advises parents of young children to think very very carefully about giving drinks and other aspartame-contaminated foods to children.** He advocates federal action be taken to review regulation of aspartame and that the chemical be submitted for precise critical investigation.

The abstract and link to the full text to be published on EHP online, is on the homepage of the European Ramazzini Foundation: **www.ramazzini.it**
Il secondo studio Ramazzini sullaspartame in stampa sul giornale scientifico Environmental Health Perspectives Un secondo studio sul dolcificante artificiale aspartame, della Fondazione Europea Ramazzini, dal titolo LEsposizione ad Aspartame a Basse Dosi, dalla Vita Fetale e per Tutta la Vita, Aumenta gli Effetti Cancerogeni sui Ratti, tato accettato per essere pubblicato su Environmental Health Perspectives (EHP). Labstract e il pdf del testo completo di EHP online, isponibile nella homepage della Fondazione Europea Ramazzini: **www.ramazzini.it**

Aspartame carcinogenicity has been known for decades. In 1985 Dr Adrian Gross, FDA toxicologist warned Congress that aspartame violates the Delaney Amendment which prohibits from our foods any ingredient causing cancer in animals.

See two letters Dr. Gross addressed to Senator Howard Metzenbaum, and the memo that triggered a request for indictment of Searle for fraudulent submissions: **http://www.dorway.com/gross.txt** Both US Prosecutors hired on the defense team and the statute of limitations expired.

Atty. James Turner, author of the Chemical Feast and the Nader Report on Food Protection at FDA, explains: "Since l974 FDA and the Searle Drug Company have known that aspartame causes brain tumors in animals. In 1980 the public has known the Public Board of Inquiry affirmed the Searle studies showed cancer in animals and ruled that it should not be used in the food supply. It is past time that the FDA invoked the Delaney Clause and remove NutraSweet from the market. In July of 2005 further studies (Ramazzini) underscored the cancer causing capability of NutraSweet/ aspartame in animals."

See Mr. Turner in Sweet Misery: A Poisoned World explain how Rumsfeld's aspartame got approved after FDA said no. **http:// www.soundandfury.tv/pages/rumsfeld2.html**

Ralph G. Walton, M.D. of Safe Harbor Behavioral Health, Erie, PA, emphasizes:

"Dr. Soffritti's two outstanding studies on the multipotential carcinogenic effects of aspartame add significantly to the ever- **growing body of evidence on the hazards of this artificial sweetener.** The FDA's stubborn adherence to their original, and controversial, approval of aspartame is unconscionable. The public must be informed that the approvals, both in this country and Europe, are based on highly questionable industry-funded research, or, in the case of recently issued statements on aspartame's supposed safety, on a questionnaire which in no way represents legitimate research. Independent research, such as the recent Soffritti studies invariably demonstrates the extremely hazardous nature of this product." .safeharborbh.org

When the Ramazzini 2005 study was released the government and aspartame manufacturers were responsible for a coverup. They found an old AARP survey sent to a million American seniors: 16 pages with 56 questions. It asked: How high did you go in school? Had a hysterectomy? Do you eat brownies? Oatmeal? Margarine? Question #25 asked "Over the last 12 months when you drank coffee or tea, what kind of sweetener did you regularly add?" There were 6 multi-choice selections, one of which was: Equal or aspartame

The 10-year-old 3-word item instantaneously converted the questionnaire into the biggest aspartame study in history! It showed no problems at all! (None were asked for!) Thru saturation news releases this propaganda spread planet wide in major magazines and other media. **www.wnho.net/halt_the_spin_on_bogus_studies.htm**

Dr. Soffritti's work was presented to the European Food Safety Authority (EFSA). The EFSA risk assessment has been criticized from the beginning because of conflicts of interest among the panel members, particularly the chair Susan Barlow, who is an industry consultant. They rejected Dr. Soffritti's study with the lame excuse that cancers were caused by lung infections. Eminent Researchers and scientists know that respiratory infection is a factor in the dying process and never took their excuse as noteworthy.

Ramazzinis control rats had a much lower incidence of cancers than the aspartame-fed rats. Critics of EFSA's risk assessment said that if the cancers were caused by inflammatory lung disease than the control group would have had as many cancers as the rats fed aspartame.

EFSA didn't apologize so they were reported to the Universal Court of Justice. Then EFSA's Dr. Koeter admitted that industry pressured them to hijack science. **www.wnho.net/letter_to_efsa.htm**

Dr. H. J. Roberts, M.D., FACP, FCCP, author of several texts on aspartame disease commented: "The elegant studies by Dr. Morando Soffritti and his colleagues, coupled with other recent related publications, reinforce my longstanding opinion that aspartame products represent carcinogens -- or co-carcinogens -- for several major tumors in humans, especially involving the brain.

"The details appear in my text, Aspartame Disease: An Ignored Epidemic, and in my article, Does Aspartame Cause Human Brain Cancer? The latter indicated that aspartame or its metabolites "might activate one or more oncogenes that potentate or initiate cell mitosis, either by direct or indirect effects

"The failure of the FDA to acknowledge and act on the ongoing revelations over the last two decades about the hazards of aspartame products in a number of realms, especially when taken by children and pregnant women, remains a source of professional embarrassment.

This chemical constitutes an imminent public health hazard. I congratulate Dr. Soffritti for independently underscoring this warning."

Neurosurgeon Dr. Russell Blaylock remarked on Dr. Soffritti's first study: "The study released in the European Journal of oncology by Morando Soffritti and co-workers should terrify mothers and all those consuming aspartame sweetened products. This was a carefully done study, which clearly demonstrated a statistically significant increase in

several types of lymphomas and leukemias in rats. Both of these malignancies have increased significantly in this country since the widespread use of aspartame.

"The type of damage was a duplicate of that associated with cancers. **Along with this most recent study, this means that drinking a single diet cola sweetened with developing a lymphoma or leukemia.**

"They also found an increased incidence of malignant brain tumors, even though it was not statistically significant. This does not mean there is no association to brain tumors, since **only the animals exposed to aspartame developed the tumors. With children and pregnant women drinking the largest amount of diet colas, this puts their children at the greatest risk of developing one of these horrible diseases.** Their study found that even low doses of aspartame could cause these malignancies; yet, the higher the dose, the more cancers that were seen.

"Since aspartame can increase obesity and may even cause the metabolic syndrome that affects 48 million Americans, there is no reason to ever consume this product. At the least it should be immediately banned from all schools." Dr. Blaylock is author of Excitotoxins: The Taste That Kills about aspartame. In order to save the children this Report For Schools is repeatedly sent to schools and board of educations. **www.wnho.net/report_on_aspartame_and_children.htm**

Will FDA finally admit aspartame approval was illegal, violating Delaney and also adulteration statutes? Aspartame is sold as an additive, but it's a deadly excitoneurotoxic carcinogenic drug that interacts with all drugs and vaccines. Aspartame brain tumor cases from New York and New Jersey are being taken at this time.

Just when you think the FDA can't sink lower, they do. They issued a report the day before the release of the new study, denying its research, without any excuse. This was about the 2005 study but releasing this negative statement the day before the new one stands to confuse the public, as they intend. **http://www.wnho.net/open_letter_to_laura_tarantino_fda.htm**

After a study sponsored by Food Standards in the UK showed additives cause behavioral problems in children, large food chains there began the removal of aspartame from their products. But not the FDA in the US who have known this for a quarter of a century. **http://www.youtube.com/watch?v=7W-gba0GPwU** From Atavistik Pictures: **www.atavistik.com Dr. Louis Elsas, pediatric professor, genetics, testified to Congress in l985 that it's a teratogen and can trigger birth defects and mental retardation.** It's time for Congress to look into the matter that the FDA has allowed aspartame to remain on the market for a quarter of a century even though they know it causes cancer, and a host of neurodegenerative diseases. The stream of whoppers continues **www.wnho.net/whopper.htm** just like the millions of deaths from aspartame.

Dr. Betty Martini, D.Hum, Founder
Mission Possible International
9270 River Club Parkway
Duluth, Georgia 30097
770 242-2599
http://www.wnho.net
http://www.mpwhi.com/

Aspartame Toxicity Center
http://www.holisticmed.com/aspartame

To contact the Ramazzini Foundation:

Kathryn Knowles
Director of Resource Development
European Ramazzini Foundation
Tel. +39 051 6640460 (int.3)
skype: kathrynknowles
development@ramazzini.it
http://www.ramazzini.it/

Clinical study abstracts showing the potential benefits of Spirulina and Chlorella in their fight against cancer.

Here is some scientific support as to why I take feel that a Barley Grass Juice and Spirulina product would be a great addition to your daily diet. Lately I have been alternating between many different products like the Zrii and FrequenSea. When I do consume Barley Grass and Spirulina I take two teaspoons of the Barley Grass Juice and one teaspoon of the Spirulina per day mixed in with vanilla flavored rice milk or soymilk. Those who have or had cancer and or tumors tell me that by taking the above amounts two to four timed per day, they achieved their desired results. Results varied by individual.

Biochem Pharmacol. 2004 Aug 1;68(3):453-62.-Molecular mechanisms in C-Phycocyanin induced apoptosis in human chronic myeloid leukemia cell line-K562.

Subhashini J, Mahipal SV, Reddy MC, Mallikarjuna Reddy M, Rachamallu A, Reddanna P.-Department of Animal Sciences, School of Life Sciences, University of Hyderabad, Hyderabad 500046, India.

C-Phycocyanin (C-PC), the major light harvesting biliprotein from **Spirulina platensis is of greater importance because of its various biological and pharmacological properties. It is a water soluble, non-toxic fluorescent protein pigment with potent anti-oxidant, anti-inflammatory and anti-cancer properties.**

In the present study the effect of highly purified C-PC was tested on growth and multiplication of human chronic myeloid leukemia cell line (K562). The results indicate significant decrease (49%) in the proliferation of K562 cells treated with 50 microM C-PC up to 48 h. Further studies involving fluorescence and electron microscope revealed characteristic apoptotic features like cell shrinkage, membrane blebbing and nuclear condensation. Agarose electrophoresis of genomic DNA of cells treated with C-PC showed fragmentation pattern typical for apoptotic cells. Flow cytometric analysis of cells treated with 25 and 50 microM C-PC for 48 h showed 14.11 and 20.93% cells in sub-G0/G1 phase, respectively. C-PC treatment of K562 cells also resulted in release of cytochrome c into the cytosol and poly (ADP) ribose polymerase (PARP) cleavage. These studies also showed down regulation of anti-apoptotic Bcl-2 but without any changes in pro-apoptotic Bax and thereby tilting the Bcl-2/Bax ratio towards apoptosis. These effects of C-PC appear to be mediated through entry of C-PC into the cytosol by an unknown mechanism. The present study thus demonstrates that C-PC induces apoptosis in K562 cells by cytochrome c release from mitochondria into the cytosol, PARP cleavage and down regulation of Bcl-2.-PMID: 15242812 [PubMed - indexed for MEDLINE]

Arch Latinoam Nutr. 2002 Sep;52(3):232-40.- [Update on the pharmacology of Spirulina (Arthrospira), an unconventional food]

Chamorro G, Salazar M, Araujo KG, dos Santos CP, Ceballos G, Castillo LF.-Escuela Nacional de Ciencias Biologicas, Instituto Politecnico Nacional, M.A.D. Mexico Universidade Federal Fluminense, Niteroi, Brasil.

Spirulina (Arthrospira), a filamentous, unicellular alga, is a cyanobacterium grown in certain countries as food for human and animal consumption. It is also used to derive additives in pharmaceuticals and foods. **This alga is a rich source of proteins, vitamins, amino acids, minerals, and other nutrients. Its main use, therefore, is as a food supplement. Over the last few years, however, it has been found to have many additional pharmacological properties. Thus, it has been experimentally proven, in vivo and in vitro that it is effective to treat certain allergies, anemia, cancer, hepatotoxicity, viral and cardiovascular diseases, hyperglycemia, hyperlipidemia, immunodeficiency, and inflammatory processes, among others.** Several of these activities are attributed to Spirulina itself or to some of its components including fatty acids omega-3 or omega-6, beta-carotene, alpha-tocopherol, phycocyanin, phenol compounds, and a recently isolated complex, Ca-Spirulan (Ca-SP). This paper aims to update and critically review the results published over the last few years with regards to these properties. The conclusion is that even if this cyanobacterium has been one of the most extensively studied from the chemical, pharmacological and toxicological points of view, it is still necessary to expand the research in order to have more consistent data for its possible use in human beings.Publication Types: • Review, Tutorial PMID: 12448336 [PubMed - indexed for MEDLINE]

Int Immunopharmacol 2002 Mar;2(4):423-34

Activation of the human innate immune system by Spirulina: augmentation of interferon production and NK cytotoxicity by oral administration of hot water extract of Spirulina platensis.-Hirahashi T, Matsumoto M, Hazeki K, Saeki Y, Ui M, Seya T.- Department of Immunology, Osaka Medical Center for Cancer and Cardiovascular Diseases, Japan.

"Spirulina platensis is a cyanobacterial species that is surmised to potentiate the immune system leading to suppression of cancer development and viral infection. Here, we identified the molecular mechanism of the human immune potentiating capacity of Spirulina by analyzing blood cells of volunteers with pre and post oral administration of hot water extract of Spirulina. NK functions represented by IFN gamma production and cytolysis were enhanced after administration of Spirulina in >50% subjects. IFN gamma was produced in an IL-12/IL-18-dependent fashion. In vitro stimulation of blood cells with BCG cell wall skeleton (CWS) allowed more potent IL-12 p40 production in cells from volunteers given Spirulina than in cells without pre-exposure to Spirulina. As BCG-CWS serves as a ligand for Toll-like receptor (TLR) 2 and 4 to raise the maturation stage of monocytes/macrophages, Spirulina may be involved in the signaling responses through Toll in blood cells even when orally administered. These observations indicated that in humans Spirulina acts directly on myeloid lineages and either directly or indirectly on NK cells. The presence of co-operative IL-12 and IL-18 is critically important for NK-mediated IFN gamma production." -PMID: 11962722 [PubMed - in process]

Acta Pharmacol Sin 2001 Dec;22(12):1121-4-Chemo-and radio-protective effects of polysaccharide of Spirulina platensis on hemopoietic system of mice and dogs.

Zhang HQ, Lin AP, Sun Y, Deng YM. The Medical and Pharmaceutical Academe of Yangzhou University, Yangzhou 225001, China.
"AIM: To observe polysaccharide of Spirulina platensis (PSp) on the hematopoietic system of mouse and dogs which were damaged by injection of cyclophosphamide (CTX) and (60) Co-gamma irradiation. **METHODS**: CTX and (60) Co gamma ray were used to induce bone marrow damage, and the experimental animals were ig with different dose of PSp in vivo, after 12-d and 21-d administration, the whole blood cells and nucleated cells in bone marrow were measured, and the DNA in bone marrow were inspected by UV-spectrophotometer. **RESULTS**: CTX and (60) Co-gamma irradiation induced hemopoietic system damage in mice and dogs, respectively. PSp 30, 60 mg/kg increased the level of the white cells in blood and nucleated cells and DNA in bone marrow in mice but had no effects on red cells and hemoglobins. PSp 12 mg/kg increased the level of red cells, white cells, and hemoglobins in blood and nucleated cells in bone marrow in dogs ($P<0.01$), and the effects of PSp 60 mg/kg were better than that of berbamine hydrochloride 60 mg/kg. **CONCLUSION: PSp has chemo-protective and radio-protective capability, and may be a potential adjunct to cancer therapy."-**PMID: 11749812 [PubMed - in process]

Clin Exp Metastasis 1998 Aug;16(6):541-50-Inhibition of tumor invasion and metastasis by calcium spirulan (Ca-SP), a novel sulfated polysaccharide derived from a blue-green alga, Spirulina platensis.

Mishima T, Murata J, Toyoshima M, Fujii H, Nakajima M, Hayashi T, Kato T, Saiki I-Research Institute for Wakan-Yaku, Toyama Medical and Pharmaceutical University, Japan.

"We have investigated the effect of calcium spirulan (Ca-SP) isolated from a blue-green alga, Spirulina platensis, which is a sulfated polysaccharide chelating calcium and mainly composed of rhamnose, on invasion of B16-BL6 melanoma, Colon 26 M3.1 carcinoma, and HT-1080 fibrosarcoma cells through reconstituted basement membrane (Matrigel). Ca-SP significantly inhibited the invasion of these tumor cells through Matrigel/fibronectin-coated filters. Ca-SP also inhibited the haptotactic migration of tumor cells to laminin, but it had no effect on that to fibronectin. Ca-SP prevented the adhesion of B16-BL6 cells to Matrigel and laminin substrates but did not affect the adhesion to fibronectin. The pretreatment of tumor cells with Ca-SP inhibited the adhesion to laminin, while the pretreatment of laminin substrates did not. Ca-SP had no effect on the production and activation of type IV collagenase in gelatin zymography.

In contrast, Ca-SP significantly inhibited degradation of heparan sulfate by purified heparanase. The experimental lung metastasis was significantly reduced by co-injection of B16-BL6 cells with Ca-SP. Seven intermittent i.v. injections of 100 microg of Ca-SP caused a marked decrease of lung tumor colonization of B16-BL6 cells in a

spontaneous lung metastasis model. **These results suggest that Ca-SP, a novel sulfated polysaccharide, could reduce the lung metastasis of B16-BL6 melanoma cells, by inhibiting the tumor invasion of basement membrane probably through the prevention of the adhesion and migration of tumor cells to laminin substrate and of the heparanase activity."**-PMID: 9872601 [PubMed - indexed for MEDLINE]

Nutr Cancer 1995;24(2):197-202-Evaluation of chemoprevention of oral cancer with Spirulina fusiformis.-Mathew B, Sankaranarayanan R, Nair PP, Varghese C, Somanathan T, Amma BP, Amma NS, Nair MK.-Regional Cancer Centre, Medical College Campus, Kerala, India.

"The blue-green microalgae Spirulina, used in daily diets of natives in Africa and America, have been found to be a rich natural source of proteins, carotenoids, and other micronutrients. **Experimental studies in animal models have demonstrated an inhibitory effect of Spirulina algae on oral carcinogenesis.** Studies among preschool children in India have demonstrated Spirulina fusiformis (SF) to be an effective source of dietary vitamin A. We evaluated the chemopreventive activity of SF (1 g/day for 12 mos) in **reversing oral leukoplakia in pan tobacco chewers in Kerala, India.** Complete regression of lesions was observed in 20 of 44 (45%) evaluable subjects supplemented with SF, as opposed to 3 of 43 (7%) in the placebo arm ($p < 0.0001$). When stratified by type of leukoplakia, the response was more pronounced in homogeneous lesions: complete regression was seen in 16 of 28 (57%) subjects with homogeneous leukoplakia, 2 of 8 with erythroplakia, 2 of 4 with verrucous leukoplakia, and 0 of 4 with ulcerated and nodular lesions. Within one year of discontinuing supplements, 9 of 20 (45%) complete responders with SF developed recurrent lesions. Supplementation with SF did not result in increased serum concentration of retinol or beta-carotene, nor was it associated with toxicity. This is the first human study evaluating the chemopreventive potential of SF. More studies in different settings and different populations are needed for further evaluation."-Publication Types: Clinical Trial -Randomized Controlled Trial -PMID: 8584455 [PubMed - indexed for MEDLINE]

J Agric Food Chem. 2005 May 18;53(10):4207-12.

Antioxidant and antiproliferative activities of Spirulina and Chlorella water extracts.-Wu LC, Ho JA, Shieh MC, Lu IW.

Department of Applied Chemistry, National Chi-Nan University, Puli, Nantou, Taiwan. lw25@ncnu.edu.tw

Liver fibrosis is a chronic liver disease that will further develop to cirrhosis if severe damage continues to form. A potential treatment for liver fibrosis is to inhibit activated hepatic stellate cell (HSC) proliferation and, subsequently, to induce HSC apoptosis. It has been reported that antioxidants are able to inhibit the proliferation of HSCs. In this study, the aqueous extract of spirulina was chosen as the source of antioxidant to investigate the inhibitory effect on the proliferation of HSC. The growth inhibitory effects of aqueous spirulina and chlorella extract on human liver cancer cells, HepG2, were also studied and compared in pairs. **Results indicated that the total phenol content of spirulina was almost five times greater than that of chlorella (6.86 +/- 0.58 vs 1.44 +/- 0.04 mg tannic acid equivalent/g of algae powder, respectively).** The antioxidant activity of spirulina determined by the ABTS*+ method was higher than chlorella (EC50: 72.44 +/- 0.24 micromol of trolox equivalent/g of spirulina extract vs 56.09 +/- 1.99 micromol of trolox equivalent/g of chlorella extract). Results of DPPH* assay also showed a similar trend as the ABTS*+ assay (EC50: 19.39 +/- 0.65 micromol of ascorbic acid equivalent/g of spirulina extract vs 14.04 +/- 1.06 micromol of ascorbic acid equivalent/g of chlorella extract). **The aqueous extracts of these two algae both showed antiproliferative effects on HSC and HepG2, but spirulina was a stronger inhibitor than chlorella. Annexin-V staining showed that aqueous extract of spirulina induced apoptosis of HSC after 12 h of treatment. In addition, the aqueous extract of spirulina triggered a cell cycle arrest of HSC at the G2/M phase.** PMID: 15884862 [PubMed - indexed for MEDLINE]

J Med Food. 2004 Summer;7(2):146-52.

Effects of chlorella on activities of protein tyrosine phosphatases, matrix metalloproteinases, caspases, cytokine release, B and T cell proliferations, and phorbol ester receptor binding.-Cheng FC, Lin A, Feng JJ, Mizoguchi T, Takekoshi H, Kubota H, Kato Y, Naoki Y.

MDS Pharma Services Taiwan Ltd., 158 Li-Teh Road, Taipei 112, Taiwan, Republic of China. fong-chi.cheng@mdsps.com

A **Chlorella powder was screened using 52 in vitro assay systems for enzyme activity, receptor binding, cellular cytokine release, and B and T cell proliferation.** The screening revealed a very potent inhibition of human protein tyrosine phosphatase (PTP) activity of CD45 and PTP1C with 50% inhibitory concentration (IC(50)) values of 0.678 and 1.56 microg/mL, respectively. It also showed a moderate inhibition of other PTPs, including PTP1B (IC(50) = 65.3 microg/mL) and T-cell-PTP (114 microg/mL). Other inhibitory activities and their IC(50) values included inhibition of the human matrix metalloproteinases (MMPs) MMP-1 (127 microg/mL), MMP-3 (185 microg/mL), MMP-7 (18.1 microg/mL), and MMP-9 (237 microg/mL) and the human peptidase caspases caspase 1 (300 microg/mL), caspase 3 (203 microg/mL), caspase 6 (301 microg/mL), caspase 7 (291 microg/mL), and caspase 8 (261 microg/mL), as well as release of the cytokines interleukin (IL)-1 (44.9 microg/mL), IL-2 (14.8 microg/mL), IL-4 (49.2 microg/mL), IL-6 (34.7 microg/mL), interferon-gamma (31.6 microg/mL), and tumor necrosis factor-alpha (11 microg/mL) from human peripheral blood mononuclear cells. Chlorella also inhibited B cell proliferation (16.6 microg/mL) in mouse splenocytes and T cell proliferation (54.2 microg/mL) in mouse thymocytes. The binding of a phorbol ester, phorbol 12,13-dibutyrate, to its receptors was also inhibited by Chlorella with an IC(50) of 152 microg/mL. **These results reveal potential pharmacological activities that, if confirmed by in vivo studies, might be exploited for the prevention or treatment of several serious pathologies, including inflammatory disease and cancer.** -PMID: 15298760 [PubMed - indexed for MEDLINE]

Phytother Res. 2002 Sep;16(6):581-5.

Simple assay for antitumour immunoactive glycoprotein derived from Chlorella vulgaris strain CK22 using ELISA.-Noda K, **Tanaka K**, **Yamada A**, **Ogata J**, **Tanaka H**, **Shoyama Y**.

Department of Pharmacognosy, Graduate School of Pharmaceutical Sciences, Kyushu University, 3-1-1 Maidashi, Higashi-ku, Fukuoka 812-8582, Japan.
A quantitative ELISA system was developed using a monoclonal antibody (MAb) specific for an antitumour immunoactive glycoprotein (CVS) derived from C. vulgaris strain CK22. The full measuring range of the assay extends from 0.63 to 10.0 ng/mL of CVS. Although no cross-reaction was observed to proteins tested or other biological response modifiers (BRMs) derived from different sources, cross-reactions were found with culture supernatants from two other strains of C. vulgaris having a strong antitumour immunoactivity. Treatment of CVS with protease, acid, or alkali weakened or completely eliminated the reactivity against the MAb and also its antitumour immunoactivities. This ELISA system is suitable for the biologically active form of CVS derived from C. vulgaris strain CK22 and related immunoactive strains. Copyright 2002 John Wiley & Sons, Ltd.-PMID: 12237820 [PubMed - indexed for MEDLINE]

For those of you who are currently taking chemo you may want to also take a product that contains Chlorella. The Chlorella my help with the side effects as evidenced by the following study.

Cancer Immunol Immunother. 1996 Jun;42(5):268-74.

Protective effect of an acidic glycoprotein obtained from culture of Chlorella vulgaris against myelosuppression by 5-fluorouracil.-Konishi F, Mitsuyama M, Okuda M, Tanaka K, Hasegawa T, Nomoto K.

Research Laboratories, Chlorella Industries Co. Ltd., Fukuoka, Japan.

An acidic glycoprotein prepared from a culture of Chlorella vulgaris (CVS) was examined for its protective effect on 5-fluorouracil(5FU)-induced myelosuppression and indigenous infection in mice. Subcutaneous administration of CVS greatly reduced the mortality of non-tumor-bearing mice given a high dose of 5FU, and could increase the LD50 value of 5FU for these mice. After 5FU treatment, indigenous infection developed probably as a result of the impairment of the host defense system. CVS reduced the incidence of indigenous infections and this effect was attributable to the acceleration of recovery from 5FU-induced myelosuppression. Early recovery of hematopoietic stem cells, or cells responding to interleukin-3 or granulocyte/macrophage-colony-stimulating factor, was especially observed in the bone marrow of CVS-treated mice on days 4-9 after the injection of 5FU. **When tumor-bearing mice were given CVS during treatment with 5FU, CVS prolonged the survival of mice without affecting the antitumor activity of 5FU. In addition, CVS was itself shown to exert an antitumor effect. These results**

suggested that CVS may be beneficial for the alleviation of side-effects in cancer chemotherapy without affecting the antitumor activity of the chemotherapeutic agent. PMID: 8706047 [PubMed - indexed for MEDLINE]

Immunopharmacol Immunotoxicol. 2001 Feb;23(1):119-32.
Effects of the green algae Chlorella vulgaris on the response of the host hematopoietic system to intraperitoneal ehrlich ascites tumor transplantation in mice. Justo GZ, Silva MR, Queiroz ML.

Department of Pharmacology and Hemocentre, Faculdade de Ciencias Medicas, Universidade Estadual de Campinas (UNICAMP), SP, Brazil.

Chlorella vulgaris extract (CVE) was examined for its effects on the Ehrlich ascites tumor-induced suppression in the numbers of bone marrow and spleen granulocyte-macrophage progenitor cells (CFU-GM) in mice. No effects on bone marrow and spleen CFU-GM, as compared to controls, were observed in normal mice given 50, 100 and 200 mg/kg CVE orally for 5 days. In tumor-bearing mice, myelosuppression concomitant with increased number of spleen CFU-GM were observed. The number of CFU-GM in the bone marrow was restored to control levels after the administration of CVE (50, 100 and 200 mg/kg) to tumor-bearing mice, and a slight reduction in spleen colony formation was observed in these animals. In addition, CVE significantly prolonged the survival of mice inoculated with the Ehrlich ascites tumor. **These results suggest a protective antitumor effect of CVE which might be attributable, at least in part, to the stimulation of the production and, possibly, maturation of granulocytes and macrophages**.-PMID: 11322644 [PubMed - indexed for MEDLINE]

After reading the next two clinical studies I feel you should go out and get yourself a bottle of the Mangosteen or any anti-parasite product that you may be aware of.

Clinical study abstracts that show the potential link between parasites and cancer

Mutagenesis 2001 Nov; 16(6):495-7 Increased translocation frequency of chromosomes 7, 11 and 14 in lymphocytes from patients with neurocysticercosis. Herrera LA, Rodriguez U, Gebhart E, Ostrosky-Wegman P.Instituto de Investigaciones Biomedicas, Universidad Nacional Autonoma de Mexico, Mexico City, DF, Mexico. mailto:metil@hotmail.com

"Neurocysticercosis (NCC) has been associated with a high frequency of DNA damage in human circulating lymphocytes and more recently with the development of hematological malignancies. **(Background: Neurocysticercosis is the most common parasitic infection of the central nervous system. Tissue-invading larval forms of the pork tapeworm Taenia solium cause the disease. Historically, neurocysticercosis was endemic to only Latin America, Asia, and Africa, though it has become increasingly frequent in the US since the 1980s. Because of this epidemiological change, all general pediatricians should become familiar with its disease process.) Chronic inflammation, a common feature of helminthic infections, has been proposed to play a key role in carcinogenesis induced by parasites…**

These results suggest that persistent antigen stimulation can cause chromosome instability in lymphocytes from patients with NCC and should be considered as an additional mechanism whereby parasites may induce cancer." -PMID: 11682640 [PubMed - indexed for MEDLINE]

Trans R Soc Trop Med Hyg 2000 Jan-Feb;94(1):61-5-Possible association between Taenia solium cysticercosis and cancer: increased frequency of DNA damage in peripheral lymphocytes from neurocysticercosis patients.

Herrera LA, Ramirez T, Rodriguez U, Corona T, Sotelo J, Lorenzo M, Ramos F, Verdorfer I, Gebhart E, Ostrosky-Wegman P.-Instituto de Investigaciones Biomedicas, Universidad Nacional Autonoma de Mexico (UNAM), Mexico, D.F., Mexico. mailto:metil@hotmail.com

"Helminths, particularly some Schistosoma (blood flukes) species, have been associated with cancer in humans. Neurocysticercosis, produced by cysticerci of the helminth Taenia solium, **has been associated with the emergence**

of brain tumours and haematological malignancies..." PMID: 10748903 [PubMed - indexed for MEDLINE] Am J Surg Pathol 1984 Jan;8(1):73-7

Human ectopic fascioliasis in the cecum. Park CI, Kim H, Ro JY, Gutierrez Y.

"A case of ectopic human Fasciola spp. infection in the cecal wall is reported. The patient, a 27-year-old Korean woman, resident in Seoul, Korea, presented with nausea, vomiting, and epigastric tenderness. One week later a palpable mass was discovered in the right iliac fossa. A clinical diagnosis of a carcinoma of the colon was made and the patient underwent a cecal resection. **The mass proved to be an inflammatory reaction containing numerous tracts made by the migrating fluke, (parasite)." Fasciola sp.-**PMID: 6696167 [PubMed - indexed for MEDLINE]

Clinical study abstracts showing the potential benefits of Mangosteen, Coco (Dark Chocolate), Green Tea and Aloe Vera in their fight against cancer

I was first introduced to a Mangosteen juice product in June of 2004. I was very impressed with the benefits people were receiving so I decided to do some research. The amount of information was so extensive that I decided to include a few of the studies I found in this report. The medical profession may not tell you about all of the science behind many of the natural products that are on the market today. So, I decided to see what I could find. I learned that the Mangosteen fruit comes primarily from South East Asia. This fruit has many health enhancing properties primarily from powerful antioxidants called Xanthones.

Besides the Mangosteen I found other products that may be beneficial for just about everyone, especially cancer patients and those who would rather not have cancer. I was looking for scientific support for the following: **Trace Minerals, Aloe Vera, and Green Tea**. Well, I have included some of the studies that I found in this report. So I went to **www.pubmed.gov** and typed in the words: Mangosteen, Xanthones, Aloe Vera and Greet Tea and when I found out the names of some of the different Xanthones I also added their names to the search. I found that the Mangosteen Fruit also has anti-parasitic properties.

Anyway, here is the science that convinced me to include it in my report.

Back in 2004 when I started the research on Mangosteen I had given a bottle to my wife to see how it would help her with the two herniated discs that she has in her neck and back. After a week and half I asked my wife if she had any success. She told me that she did not have any need during that time to take her pain medicine unless she did something that she shouldn't have done.

For this report I primarily focused on two aspects of the many benefits that the Mangosteen fruit has been providing to its users. Since many cancer patients encounter pain along with the cancer I found some of the scientific evidence that would support its use for pain and cancer.

Mangosteen and Pain

Mol Pharmacol. 2004 Sep;66(3):667-74. gamma-Mangostin inhibits inhibitor-kappaB kinase activity and decreases lipopolysaccharide-induced cyclooxygenase-2 gene expression in C6 rat glioma cells.

Nakatani K, Yamakuni T, Kondo N, Arakawa T, Oosawa K, Shimura S, Inoue H, Ohizumi Y.-Department of Pharmaceutical Molecular Biology, Graduate School of Pharmaceutical Sciences, Tohoku University, Aoba, Aramaki, Aoba-ku, Sendai 980-8578, Japan.

We investigated the effect of gamma-mangostin purified from the fruit hull of the medicinal plant Garcinia mangostana on spontaneous prostaglandin E(2) (PGE(2)) genase release and inducible cyclooxy-2 (COX-2) gene expression in C6 rat glioma cells. An 18-h treatment with gamma-mangostin potently inhibited spontaneous PGE(2) release in a concentration-dependent manner with the IC(50) value of approximately 2 microM, without affecting the cell viability even at 30 microM. By immunoblotting and reverse-transcription polymerase chain reaction, we showed that gamma-mangostin concentration-dependently inhibited lipopolysaccharide (LPS)-induced expression of COX-2 protein and its mRNA, but not those of constitutive COX-1 cyclooxygenase. Because LPS is known to stimulate

inhibitor kappaB (IkappaB) kinase (IKK)-mediated phosphorylation of IkappaB followed by its degradation, which in turn induces nuclear factor (NF)-kappaB nuclear translocation leading to transcriptional activation of COX-2 gene, the effect of gamma-mangostin on the IKK/IkappaB cascade controlling the NF-kappaB activation was examined.
An in vitro IKK assay using IKK protein immunoprecipitated from C6 cell extract showed that this compound inhibited IKK activity in a concentration-dependent manner, with the IC(50) value of approximately 10 microM. Consistently gamma-mangostin was also observed to decrease the LPS-induced IkappaB degradation and phosphorylation in a concentration-dependent manner, as assayed by immunoblotting. Furthermore, luciferase reporter assays showed that gamma-mangostin reduced the LPS-inducible activation of NF-kappaB-and human COX-2 gene promoter region-dependent transcription. gamma-Mangostin also inhibited rat carrageenan-induced paw edema. **These results suggest that gamma-mangostin directly inhibits IKK activity and thereby prevents COX-2 gene transcription, an NF-kappaB target gene, probably to decrease the inflammatory agent-stimulated PGE(2) production in vivo, and is a new useful lead compound for anti-inflammatory drug development.** PMID: 15322259 [PubMed - in process]

Biochem Pharmacol. 2002 Jan 1;63(1):73-9. - Inhibition of cyclooxygenase and prostaglandin E2 synthesis by gamma-mangostin, a xanthone derivative in Mangosteen, in C6 rat glioma cells.
Nakatani K, Nakahata N, Arakawa T, Yasuda H, Ohizumi Y.- Department of Pharmaceutical Molecular Biology, Graduate School of Pharmaceutical Sciences, Tohoku University, Aoba, Aramaki, Aoba-ku, 980-8578, Sendai, Japan.

The fruit hull of Mangosteen, Garcinia mangostana L., has been used for many years as a medicine for treatment of skin infection, wounds, and diarrhea in Southeast Asia. In the present study, we examined the effect of gamma-mangostin, a tetraoxygenated diprenylated xanthone contained in Mangosteen, on arachidonic acid (AA) cascade in C6 rat glioma cells. gamma-Mangostin had a potent inhibitory activity of prostaglandin E2 (PGE2) release induced by A23187, a Ca2+ ionophore. The inhibition was concentration-dependent, with the IC50 value of about 5 microM. gamma-Mangostin had no inhibitory effect on A23187-induced phosphorylation of p42/p44 extracellular signal regulated kinase/mitogen-activated protein kinase or on the liberation of [14C]-AA from the cells labeled with [14C]-AA. However, gamma-mangostin concentration-dependently inhibited the conversion of AA to PGE2 in microsomal preparations, showing its possible inhibition of cyclooxygenase (COX). In enzyme assay in vitro, gamma-mangostin inhibited the activities of both constitutive COX (COX-1) and inducible COX (COX-2) in a concentration-dependent manner, with the IC50 values of about 0.8 and 2 microM, respectively. **Lineweaver-Burk plot analysis indicated that gamma-mangostin competitively inhibited the activities of both COX-1 and -2. This study is a first demonstration that gamma-mangostin, a xanthone derivative, directly inhibits COX activity.** PMID: 11754876 [PubMed - indexed for MEDLINE]

Shankaranarayan D, Gopalakrishnan C, Kameswaran L. Pharmacological profile of mangostin and its derivatives. Arch Int Pharmacodyn Ther. 1979 Jun;239(2):257-69.

Pharmacological profile of mangostin and its derivatives.-Shankaranarayan D, Gopalakrishnan C, Kameswaran L.-Mangostin (M), a naturally occurring xanthone in the rinds of the fruits of Garcinia mangostana Linn.

(Guttiferae) and its derivatives such as 3-0-methyl **mangostin** (MM), 3,6-di-O-methyl mangostin (DM), 1-isomangostin (IM), mangostin triacetate (MT), mangostin 3,6-di-O-(tetra acetyl) glucoside (MTG) and mangostin-6,6-di-O-glucoside (MOG) were screened for various pharmacological effects in experimental animals... M, IM and MT **produced pronounced anti-inflammatory activity** both by intraperitoneal and oral routes in rats as tested by carrageenininduced hind paw oedema, cotton pellet implantation and granuloma pouch techniques. Anti-inflammatory activity for M, IM and MT was observed even in bilaterally adrenalectomised rats. M, IM and MT did not produce any mast cell membrane stabilising effect and the degranulation effect of polymyxin B, diazoxide and Triton X-100 on rat peritoneal mast cells in vitro was not prevented. M, IM and MT did not alter the prothrombin time of albino rats. **M alone produced significant antiulcer activity** in rats.-PMID: 314790 [PubMed - indexed for MEDLINE]

Bioorg Med Chem. 2005 Aug 17; [Epub ahead of print]**Xanthones induce cell-cycle arrest and apoptosis in human colon cancer DLD-1 cells.**

Matsumoto K, Akao Y, Ohguchi K, Ito T, Tanaka T, Iinuma M, Nozawa Y.

Gifu International Institute of Biotechnology, 1-1 Naka-Fudogaoka, Kakamigahara, Gifu 504-0838, Japan; Gifu Prefectural Institute for Bio-industrial Technology, 3481-2 Kamihachiya, Hachiya, Minokamo, Gifu 505-0004, Japan.

We investigated the antiproliferative effects of four structurally similar prenylated xanthones, alpha-mangostin, beta-mangostin, gamma-mangostin, and methoxy-beta-mangostin, in human colon cancer DLD-1 cells. These xanthones differ in the number of hydroxyl and methoxy groups. Except for methoxy-beta-mangostin, the other three xanthones strongly inhibited cell growth at 20muM and their antitumor efficacy was correlated with the number of hydroxyl groups. Hoechst 33342 nuclear staining and nucleosomal DNA-gel electrophoresis revealed that the antiproliferative effects of alpha- and gamma-mangostin, but not that of beta-mangostin, were associated with apoptosis. It was also shown that their antiproliferative effects were associated with cell-cycle arrest by affecting the expression of cyclins, cdc2, and p27; G1 arrest was by alpha-mangostin and beta-mangostin, and S arrest by gamma-mangostin. **These findings provide a relevant basis for the development of xanthones as an agent for cancer prevention and combination therapy with anti-cancer drugs.** PMID: 16112579 [PubMed - as supplied by publisher]

Bioorg Med Chem. 2004 Nov 15;12(22):5799-806. -Preferential target is mitochondria in alpha-mangostin-induced apoptosis in human leukemia HL60 cells.

Matsumoto K, Akao Y, Yi H, Ohguchi K, Ito T, Tanaka T, Kobayashi E, Iinuma M, Nozawa Y.-Gifu International Institute of Biotechnology, 1-1 Naka-Fudogaoka, Kakamigahara, Gifu 504-0838, Japan. kmatsumo@giib.or.jp

Our previous study has shown that alpha-mangostin, a xanthone from the pericarps of Mangosteen, induces caspase-3-dependent apoptosis in HL60 cells. In the current study, we investigated the mechanism of apoptosis induced by alpha-mangostin in HL60 cells. Alpha-mangostin-treated HL60 cells demonstrated caspase-9 and -3 activation but not -8, which leads us to assume that alpha-mangostin may mediate the mitochondrial pathway in the apoptosis. Parameters of mitochondrial dysfunction including swelling, loss of membrane potential (deltapsim), decrease in intracellular ATP, ROS accumulation, and cytochrome c/AIF release, were observed within 1 or 2 h after the treatment. On the other hand, alpha-mangostin-treatment did not affect expression of bcl-2 family proteins and activation of MAP kinases. These findings indicate that alpha-mangostin preferentially targets mitochondria in the early phase, resulting in indication of apoptosis in HL60 cells. Furthermore, we examined the structure-activity relationship between xanthone derivatives including alpha-mangostin and the potency of deltapsim-loss in HL60 cells. Interestingly, replacement of hydroxyl group by methoxy group remarkably decreased its potency. It was also shown that the cytotoxicity substantially correlated with deltapsim decrease. **These results indicate that alpha-mangostin and its analogs would be candidates for preventive and therapeutic application for cancer treatment.**-PMID: 15498656 [PubMed - in process]

J Ethnopharmacol. 2004 Jan;90(1):161-6.-Antiproliferation, antioxidation and induction of apoptosis by Garcinia mangostana (mangosteen) on SKBR3 human breast cancer cell line.

Moongkarndi P, Kosem N, Kaslungka S, Luanratana O, Pongpan N, Neungton N.-Department of Microbiology, Faculty of Pharmacy, Mahidol University, Sri Ayudthaya Road, Rajdhevee, Bangkok 10400, Thailand. pypmk@mahidol.ac.th

This study was designed to determine the antiproliferative, apoptotic and antioxidative properties of crude methanolic extract (CME) from the pericarp of Garcinia mangostana (family Guttiferae) using human breast cancer (SKBR3) cell line as a model system. SKBR3 cells were cultured in the presence of CME at various concentrations (0-50 microg/ml) for 48 h and the percentage of cell viability was evaluated by 3-(4,5-dimethylthiazol-2-yl)-2,5-di phenyl

tetrazolium bromide (MTT) assay. CME showed a dose-dependent inhibition of cell proliferation with ED(50) of 9.25+/-0.64 microg/ml. We found that antiproliferative effect of CME was associated with apoptosis on breast cancer cell line by determinations of morphological changes and oligonucleosomal DNA fragments. In addition, CME at various concentrations and incubation times were also found to inhibit ROS production. **These investigations suggested that the methanolic extract from the pericarp of Garcinia mangostana had strong antiproliferation, potent antioxidation, and induction of apoptosis. Thus, it indicates that this substance can show different activities and has potential for cancer chemoprevention, which were dose dependent as well as exposure time dependent.** PMID: 14698525 [PubMed - indexed for MEDLINE]

Asian Pac J Cancer Prev. 2004 Oct-Dec;5(4):433-8.-Inhibitory effects of crude alpha-mangostin, a xanthone derivative, on two different categories of colon preneoplastic lesions induced by 1, 2-dimethylhydrazine in the rat.-Nabandith V, Suzui M, Morioka T, Kaneshiro T, Kinjo T, Matsumoto K, Akao Y, Iinuma M, Yoshimi N.- Tumor Pathology Division, Faculty of Medicine, University of the Ryukyus, Okinawa 903-0215, Japan.

The purpose of this study was to examine whether crude alpha-mangostin (a major xanthone derivative in Mangosteen pericarp (Garcinia mangostana)) has short-term chemopreventive effects on putative preneoplastic lesions involved in rat colon carcinogenesis. The crude preparation was obtained by simple recrystallization of an ethylacetate extract of Mangosteen pericarps. A total of 33 five-week-old male F344 rats were randomly divided into 5 experimental groups. Rats in groups 1-3 were given a subcutaneous injection of 1,2-dimethylhydrazine (DMH)(40 mg/kg body weight) once a week for 2 weeks. Starting one week before the first injection of DMH, rats in groups 2 and 3 were fed a diet containing 0.02% and 0.05% crude alpha-mangostin, respectively, for 5 weeks. Rats in group 4 also received the diet containing 0.05% crude alpha-mangostin, while rats in group 5 served as untreated controls.

The experiment was terminated 5 weeks after the start. Dietary administration of crude alpha-mangostin at both doses significantly inhibited the induction and/or development of aberrant crypt foci (ACF) ($P<0.05$ for 0.02% crude alpha-mangostin, $P<0.01$ for 0.05% crude alpha-mangostin), when compared to the DMH-treated group (group 1). Moreover, treatment of rats with 0.05% crude alpha-mangostin significantly decreased dysplastic foci (DF) ($P<0.05$) and beta-catenin accumulated crypts (BCAC) ($P<0.05$), to below the group 1 values. The proliferating cell nuclear antigen (PCNA) labeling indices of colon epithelium and focal lesions in groups 2 and 3 were also significantly lower than in group 1 and this effect occurred in a dose dependent manner of the crude alpha-mangostin. **This finding that crude alpha-mangostin has potent chemopreventive effects in our short-term colon carcinogenesis bioassay system suggests that longer exposure might result in suppression of tumor development.-**Publication Types: • Evaluation Studies-PMID: 15546251 [PubMed - indexed for MEDLINE]

J Nat Prod. 2003 Aug;66(8):1124-7.-Induction of apoptosis by xanthones from mangosteen in human leukemia cell lines. Matsumoto K, Akao Y, Kobayashi E, Ohguchi K, Ito T, Tanaka T, Iinuma M, Nozawa Y.

Gifu International Institute of Biotechnology, 1-1 Naka-Fudogaoka, Kakamigahara, Gifu 504-0838, Japan. kmatsumoto@giib.or.jp

We examined the effects of six xanthones from the pericarps of Mangosteen, Garcinia mangostana, on the cell growth inhibition of human leukemia cell line HL60. **All xanthones displayed growth inhibitory effects. Among them, alpha-mangostin showed complete inhibition at 10 microM through the induction of apoptosis.** PMID: 12932141 [PubMed - indexed for MEDLINE]

Fitoterapia. 2004 Jun;75 (3-4):375-7.-Antiproliferative activity of Thai medicinal plant extracts on human breast adenocarcinoma cell line.

Moongkarndi P, Kosem N, Luanratana O, Jongsomboonkusol S, Pongpan N.- Department of Microbiology, Faculty of Pharmacy, Mahidol University, Rajdhevee, Sri Ayudthaya Rd, Bangkok 10400, Thailand. pypmk@mahidol.ac.th

Ethanolic extracts of selected nine Thai medicinal plants were tested for antiproliferative activity against SKBR3 human breast adenocarcinoma cell line using MTT assay. **Garcinia mangostana showed the most potent activity.**

However, all plant extracts showed activity in potential range for further investigation on cancer cells. Copyright 2004 Elsevier B.V. PMID: 15158999 [PubMed - indexed for MEDLINE]

Planta Med. 2002 Nov;68 (11):975-9. -Garcinone E, a xanthone derivative, has potent cytotoxic effect against hepatocellular carcinoma cell lines.

Ho CK, Huang YL, Chen CC.- Department of Medical Research & Education, Veterans General Hospital, Taipei, ROC.

Treatment of hepatocellular carcinomas (HCCs) with chemotherapy has generally been disappointing and it is most desirable to have more effective new drugs. We extracted and purified 6 xanthone compounds from the rinds (peel) of the fruits of Garcinia mangostana L., using partitioned chromatography and then tested the cytotoxic effects of these compounds on a panel of 14 different human cancer cell lines including 6 hepatoma cell lines, based on the MTT method. Several commonly used chemotherapeutic agents were included in the assay to determine the relative potency of the potential new drugs**.**

Our results have shown that one of the xanthone derivatives, which could be identified as garcinone E has potent cytotoxic effect on all HCC cell lines as well as on the other gastric and lung cancer cell lines included in the screen. We suggest that garcinone E may be potentially useful for the treatment of certain types of cancer. PMID: 12451486 [PubMed - indexed for MEDLINE]

Lu ZX, Hasmeda M, Mahabusarakam W, Ternai B, Ternai PC, Polya GM.

Inhibition of eukaryote protein kinases and of a cyclic nucleotide-binding phosphatase by prenylated xanthones.-Chem Biol Interact. 1998 Jul 3;114(1-2):121-40.

Lu ZX, Hasmeda M, Mahabusarakam W, Ternai B, Ternai PC, Polya GM. School of Biochemistry, La Trobe University, Bundoora, Victoria, Australia.

A series of prenylated xanthones are variously potent inhibitors of the catalytic subunit (cAK) of rat liver cyclic AMP-dependent protein kinase (PKA), rat brain Ca2+ and phospholipid-dependent protein kinase C (PKC), chicken gizzard myosin light chain kinase (MLCK), wheat embryo Ca2+-dependent protein kinase (CDPK) and potato tuber cyclic nucleotide-binding phosphatase (Pase). The prenylated xanthones examined are mostly derivatives of alpha-mangostin in which the 3-hydroxyl and 6-hydroxyl are variously substituted with groups R or R', respectively, or derivatives of 3-isomangostin (mangostanol) in which the 9-hydroxyl is substituted with groups R' or the prenyl side chain is modified. The most potent inhibitors of cAK have non-protonatable and relatively small R' and R groups. Conversely, the most potent inhibitors of PKC and MLCK have bulkier and basic R' groups. Some prenylated xanthones are also potent inhibitors of CDPK. PKC and cAK are competitively inhibited by particular prenylated xanthones whereas the compounds that are the most potent inhibitors of MLCK and CDPK are non-competitive inhibitors. Prenylated xanthones having relatively small and non-protonatable R' and R groups inhibit a high-affinity cyclic nucleotide binding Pase in a non-competitive fashion. **(Protein kinases make up a veritable treasure trove of targets for a variety of indications, including diabetes, inflammatory disorders, and especially cancer.)** PMID: 9744560 [PubMed - indexed for MEDLINE]

Aloe Vera

Int Immunopharmacol. 2004 Dec 20;4(14):1775-84.-Aloe-emodin modulates PKC isozymes, inhibits proliferation, and induces apoptosis in U-373MG glioma cells, Acevedo-Duncan M, Russell C, Patel S, Patel R.

Department of Chemistry, University of South Florida, Tampa, FL, USA. macevedo@chuma.cas.usf.edu

Aloe-emodin (1,8-dihydroy-3-[hydroxymethyl]-anthraquione) purified from Aloe vera leaves has been reported to have antitumor activity. The objectives of our research were to determine how aloe-emodin regulates the cell cycle, cell proliferation, and protein kinase C (PKC) during glioma growth and development. To establish the cell cycle effects of aloe-emodin on brain cells [transformed glia cell line (SVG) and human glioma U-373MG cell line (U-373MG)], cells were treated with either dimethylsulfoxide (DMSO; control) or aloe-emodin (40 microM). Results

from flow cytometry demonstrated that aloe-emodin delayed the number of cells entering and exiting DNA synthesis (S) phase in both SVG and U-373MG cells indicating that aloe-emodin may inhibit S phase progression. Assessment of cell viability demonstrated that SVG and U-373MG glioma cell were highly sensitive to aloe-emodin. The aloe-emodin-induced decreased proliferation was sustained at 48-96 h. A PKC activity assay was quantified to establish the role of PKC in aloe-emodin's mode of action. Exposure of SVG and U-373MG glioma cells to aloe-emodin suppressed PKC activity and reduced the protein content of most of the PKC isozymes. **We determined that cancer growth inhibition by aloe-emodin was due to apoptosis (i.e., programmed cell death). Taken together, these results support the hypothesis that aloe-emodin represents a novel antitumor chemotherapeutic drug.** PMID: 15531293 [PubMed - indexed for MEDLINE]

Int Immunopharmacol. 2004 Mar;4(3):411-8.-Mannan from Aloe saponaria inhibits tumoral cell activation and proliferation.-Sampedro MC, Artola RL, Murature M, Murature D, Ditamo Y, Roth GA, Kivatinitz S.-Departamento de Quimica Biologica
CIQUIBIC, Facultad Ciencias Quimicas, Universidad Nacional de Cordoba, Cuidad Universitaria, C5000GYA-Cordoba 5016, Argentina.

In this study, we tested the antiproliferative effects of mannan from Aloe saponaria using normal murine (SpMC) and human cells (PBMC) and several tumoral cell lines. Employing flow cytometry, it could be determined that mannan inhibits the proliferative response in normal and tumoral cells. Mannan affects the expression of CD3 (+) SpMC indicating that mannan inhibits mainly T lymphocyte proliferative response. Also in SpMC cultured with or without mitogen mannan produces an increase of an activation marker (CD25). On C1498 cell line, mannan reduces CD3 expression and abolishes the CD25 expression. **In conclusion, mannan has a dual beneficial effect when applied to normal and tumoral cells at the same time by inhibiting the activation of cancer cells and improving that of normal ones.** PMID: 15037218 [PubMed - indexed for MEDLINE]

Life Sci. 2002 Sep 6;71(16):1879-92.-The antiproliferative activity of aloe-emodin is through p53-dependent and p21-dependent apoptotic pathway in human hepatoma cell lines. Kuo PL, Lin TC, Lin CC.

Graduate Institute of Natural Products, College of Pharmacy, Kaohsiung Medical University, Kaohsiung 807, Taiwan, ROC.

The aim of this study is to investigate the anticancer effect of aloe-emodin in two human liver cancer cell lines, Hep G2 and Hep 3B. We observed that aloe-emodin inhibited cell proliferation and induced apoptosis in both examined cell lines, but with different the antiproliferative mechanisms. In Hep G2 cells, aloe-emodin induced p53 expression and was accompanied by induction of p21 expression that was associated with a cell cycle arrest in G1 phase. In addition, aloe-emodin had a marked increase in Fas/APO1 receptor and Bax expression. In contrast, with p53-deficient Hep 3B cells, the inhibition of cell proliferation of aloe-emodin was mediated through a p21-dependent manner that did not cause cell cycle arrest or increase the level of Fas/APO1 receptor, but rather promoted aloe-emodin induced apoptosis by enhancing expression of Bax. **These findings suggest that aloe-emodin may be useful in liver cancer prevention.** PMID: 12175703 [PubMed - indexed for MEDLINE]

Am J Dermatopathol. 2002 Feb;24(1):17-22.-The effect of aloe emodin on the proliferation of a new merkel carcinoma cell line.- Wasserman L, Avigad S, Beery E, Nordenberg J, Fenig E.

Felsenstein Medical Research Center, Sackler Faculty of Medicine, Tel Aviv University, Rabin Medical Center Beilinson Campus, Petah Tikva 49100, Israel. yardenam@clalit.org.ie

A free-floating cell line has been established from a metastatic lesion of a Merkel cell carcinoma (MCC) patient. The cell line was characterized by immunocytochemical reactions with antibodies against the epithelial and neuroendocrine antigens: cytokeratin 20, neuron-specific enolase, chromogranin A, neurofilament protein, synaptophysin, and calcitonin. Karyotype analysis of the MCC cells showed deletion in chromosomes 3 and 7, loss of chromosome 10, and several translocations in other chromosomes. No mutation was detected in the TP53 gene, after analyzing the complete coding region. Growth factors such as basic fibroblast growth factor, transforming growth factor-beta, and nerve and epidermal growth factors had no effect on the proliferation of the cells. The differentiation-inducing agents sodium butyrate and dimethyl sulfoxide, especially the former, markedly inhibited the proliferation of the MCC cells. Aloe emodin, a natural constituent of aloe vera leaves, significantly inhibited the growth of MCC cells. **Aloe emodin**

has been reported to be nontoxic for normal cells but to possess specific toxicity for neuroectodermal tumor cells. Differentiation-inducing agents, and aloe emodin, merit further investigation as potential agents for treating MCC. PMID: 11803275 [PubMed - indexed for MEDLINE]

Carcinogenesis. 1999 Aug;20(8):1637-40.-In vitro chemopreventive effects of plant polysaccharides (Aloe barbadensis miller, Lentinus edodes, Ganoderma lucidum and Coriolus versicolor).- Kim HS, Kacew S, Lee BM.

Division of Toxicology, College of Pharmacy, Sungkyunkwan University, Changan-ku, Chunchun-dong, Kyunggi-do, Suwon 440-746, Korea.

A plant polysaccharide, **Aloe gel extract, was reported to have an inhibitory effect** on benzo[a]pyrene (B[a]P)-DNA adduct formation in vitro and in vivo. Hence, chemopreventive effects of plant polysaccharides [Aloe barbadensis Miller (APS), Lentinus edodes (LPS), **Ganoderma lucidum** (GPS) and Coriolus versicolor (CPS)] were compared using in vitro short-term screening methods associated with both initiation and promotion processes in carcinogenesis. In B[a]P-DNA adduct formation, APS (180 micrograms/ml) was the most effective in inhibition of B[a]P binding to DNA in mouse liver cells. Oxidative DNA damage (by 8-hydroxydeoxyguanosine) was significantly decreased by APS (180 micrograms/ml) and CPS (180 micrograms/ml). In induction of glutathione S-transferase activity, GPS was found to be the most effective among plant polysaccharides. In screening anti-tumor promoting effects, APS (180 micrograms/ml) significantly inhibited phorbol myristic acetate (PMA)-induced ornithine decarboxylase activity in Balb/3T3 cells. In addition, APS significantly inhibited PMA-induced tyrosine kinase activity in human leukemic cells. APS and CPS significantly inhibited superoxide anion formation. **These results suggest that some plant polysaccharides produced both anti-genotoxic and anti-tumor promoting activities in in vitro models and, therefore, might be considered as potential agents for cancer chemoprevention.**-PMID: 10426820 [PubMed - indexed for MEDLINE]

Int J Tissue React. 1998;20(4):115-8.-The therapeutic potential of Aloe Vera in tumor-bearing rats.- Corsi MM, Bertelli AA, Gaja G, Fulgenzi A, Ferrero ME.

Institute of General Pathology, Medical Faculty, University of Milan, Italy.
Aloe Vera has been claimed to contain several important therapeutic properties, including anticancer effects. The effect of Aloe Vera administration was studied on a pleural tumor in rat. Growth of Yoshida AH-130 ascite hepatoma cells injected (2 x 10(5) in 0.1 ml) into pleura of male inbred Fisher rats was evaluated at different times (7th and 14th days). **Data show that the use of Aloe Vera proved a therapeutic method, and that the present experimental model could be useful in the study of other therapeutics treatments in vivo**.-PMID: 10093794 [PubMed - indexed for MEDLINE]

Nutrition. 1998 Nov-Dec;14(11-12):846-52.-Vitamin C and aloe vera supplementation protects from chemical hepatocarcinogenesis in the rat.- Shamaan NA, Kadir KA, Rahmat A, Ngah WZ.

Department of Biochemistry and Microbiology, Universiti Putra Malaysia, Selangor, Malaysia.

The effects of vitamin C and aloe vera gel extract supplementation on induced hepatocarcinogenesis in male Sprague-Dawley rats (120-150 g) by diethylnitrosamine (DEN) and 2-acetylaminofluorene (AAF) was investigated. The severity of the carcinogenesis process was determined by measuring gamma-glutamyl transpeptidase (GGT) and the placental form of glutathione S-transferase (GSTP) histochemically in situ and in plasma and liver fractions. In addition, plasma alkaline phosphatase (ALP) and liver microsomal uridine diphosphate glucuronyl transferase (UDPGT) activity were also determined. Administration of DEN/AAF caused an increase in the surface area and number of enzyme-positive foci (both GGT and GSTP) compared with control. Supplementation of vitamin C or aloe vera gel extract to the cancer-induced rats suppressed this increase significantly ($P < 0.05$; $P < 0.001$). Increases in liver UDPGT, GGT, and GSTP activities were also observed with cancer induction that were again suppressed with either vitamin C or aloe vera gel supplementation. Plasma GGT in the DEN/AAF rats were determined monthly for the duration of the experiment and found to be reduced as early as 1 mo with aloe vera gel supplementation and 2 mo with vitamin C supplementation. **In conclusion, vitamin C and aloe vera gel extract supplementation were found to be able to reduce the severity of chemical hepatocarcinogenesis**.-PMID: 9834927 [PubMed - indexed for MEDLINE]

Vopr Onkol. 1986;32(12):38-40.-[Antimetastatic properties of aloe juice]-[Article in Russian]- Gribel' NV, Pashinskii VG.

An evaluation of antimetastatic properties of succus Aloes was carried out using three types of experimental tumors of mice and rats. It was found that succus Aloes treatment contributes to reduction of tumor mass, metastatic foci and metastasis frequency at different stages of tumor progress without affecting major tumor growth. Succus Aloes potentiates the antitumor effect of 5-fluorouracil and cyclophosphamide as components of combination chemotherapy.-PMID: 3798837 [PubMed - indexed for MEDLINE]

Cocoa (Dark Chocolate)

Since many people around the world enjoy the taste of cocoa (Dark Chocolate) I thought I would include some of the science that I found at **www.pubmed.gov** in this report.

Eur J Cancer Prev. 2006 Aug;15(4):353-61.

In-vitro effects of polyphenols from cocoa and beta-sitosterol on the growth of human prostate cancer and normal cells.-Jourdain C, **Tenca G**, Deguercy A, Troplin P, Poelman D.

BIOAlternatives, Gençay, France. **cjo@bioalternatives.com**

Cocoa contains many different types of physiologically active components. It was shown that cocoa beans are rich in specific antioxidants such as flavonoids, catechins, epicatechins and proanthocyanidins. Additionally, beta-sitosterol, the most common phytosterol, may play a protective role in the development of cancer. The aim of this in-vitro study was to evaluate the inhibitory effect of different cocoa polyphenols extracts, alone or combined with beta-sitosterol, on two human prostate cancer cell lines (nonmetastatic 22Rv1 cells and metastatic DU145 cells) and a normal human prostate cell line (RWEP-1). A synergy between beta-sitosterol and cocoa polyphenols extract was also researched. Cells were treated independently with five products from 1 to 72 h: (1/) synthetic beta-sitosterol, (2/) a cocoa polyphenols extract supplemented with beta-sitosterol, (3/) three different cocoa polyphenols extracts naturally containing beta-sitosterol. In the experiment, beta-sitosterol was tested from 10(-6) to 10(-3) %; cocoa polyphenols extract supplementation was with 0.72% beta-sitosterol; finally cocoa polyphenols extracts were added to the cells at very low concentrations ranging from 0.001 to 0.2%. The growth and viability of cells were measured using colorimetric assay at 1, 3, 6, 24, 48, and 72 h of treatment. IC50 and IC100 corresponding to the concentration leading to a decrease of 50% and 100% of cell growth were determined. At the highest tested concentration, cocoa polyphenols extracts induced a complete inhibition of growth of metastatic and nonmetastatic cancer cell lines. In addition, cocoa polyphenols extracts were more active against local cancer cells than against metastatic cells. Moreover, at the highest tested concentration, cocoa polyphenols extracts are not effective on a normal prostate cell lines. Beta-sitosterol induced low growth inhibition of both cancer cell line. Cocoa polyphenols extracts, however, were significantly more active and showed a strong and fast inhibition of cell growth than beta-sitosterol alone. No synergy or addition was observed when beta-sitosterol was tested together with the cocoa polyphenols extract. **Our results show that cocoa polyphenols extracts have an antiproliferative effect on prostate cancer cell growth but not on normal cells, at the highest tested concentration.**-PMID: 16835506 [PubMed - indexed for MEDLINE]

Free Radic Biol Med. 2006 Oct 15;41(8):1247-56. Epub 2006 Jul 11.

Procyanidins protect Caco-2 cells from bile acid- and oxidant-induced damage.-Erlejman AG, Fraga CG, Oteiza PI.

IQUIFIB-Department of Biological Chemistry (UBA-CONICET), School of Pharmacy and Biochemistry, University of Buenos Aires, Argentina.

Procyanidins can exert cytoprotective, anti-inflammatory, and anticarcinogenic actions in the gastrointestinal tract. Previous evidence has shown that procyanidins can interact with synthetic membranes and protect them from oxidation and disruption. Thus, in this study we investigated the capacity of a hexameric procyanidin fraction (Hex) isolated from cocoa to protect Caco-2 cells from deoxycholic (DOC)-induced cytotoxicity, cell oxidant increase, and loss of monolayer integrity. Hex interacted with the cell membranes without affecting their integrity, as evidenced by a Hex-mediated increase in the transepithelial electrical resistance, and inhibition of DOC-

induced cytotoxicity. DOC induced an increase in cell oxidants, alterations in the paracellular transport, and redistribution of the protein ZO-1 from cell-cell contacts into the cytoplasm. Hex partially inhibited all these events at concentrations ranging from 2.5 to 20 microM. Similarly, Hex (5-10 microM) inhibited the increase in cell oxidants, and the loss of integrity of polarized Caco-2 cell monolayers induced by a lipophilic oxidant (2,2'-azobis (2,4-dimethylvaleronitrile). Results show that the assayed procyanidin fraction can interact with cell membranes and protect Caco-2 cells from DOC-induced cytotoxicity, oxidant generation, and loss of monolayer integrity. **At the gastrointestinal tract, large procyanidins may exert beneficial effects in pathologies such us inflammatory diseases, alterations in intestinal barrier permeability, and cancer.**-PMID: 17015171 [PubMed - indexed for MEDLINE]

Biofactors. 2005;23(3):141-50.
Extraction and chromatographic separation of anticarcinogenic fractions from cacao bean husk.-Lee KW, Hwang ES, Kang NJ, Kim KH, Lee HJ.
Department of Food Science and Technology, School of Agricultural Biotechnology and Center for Agricultural Biomaterials, Seoul National University, Seoul 151-742, Republic of Korea.

The utilization of cacao bean husk (CBH), a by-product of chocolate manufacture, would be both environmentally and economically beneficial. For this purpose, a process for effectively separating and fractionating CBH fractions having cancer preventive potential was developed in this study. For screening the fractions with potent cancer preventive activity, we examined the effect of extracts and fractions of CBH on the inhibition of gap-junction intercellular communication (GJIC) and the DNA synthesis of cancer cells, both of which are characteristics of the promotion and progression stages in carcinogenesis. The extracts of CBH (especially, the 60% ethanol fraction after extraction with 50% acetone) containing 43 wt.% polyphenol exerted an excellent protective effect on H2O2-induced inhibition of GJIC in WB-F344 rat liver epithelial cells as determined by the scrape-loading/dye transfer assay. The enhancement of GJIC by the extracts of CBH was approximately 10-fold higher than that of a well-known dietary chemopreventive component, vitamin C. The extracts of CBH (especially, the 60% ethanol fraction) also suppressed DNA synthesis in all liver, stomach, and colon cancer cells as demonstrated by the ;3H-thymidine incorporation assay, by approximately four-fold higher than that of vitamin C. **These results imply that the polyphenol extracts and fractions of CBH are effective functional materials to be used in either preventing or inhibiting cancer.**-PMID: 16410636 [PubMed - indexed for MEDLINE]

Mol Cancer Ther. **2005 Apr;4(4):537-46.**

Pentameric procyanidin from Theobroma cacao selectively inhibits growth of human breast cancer cells.-Ramljak D, Romanczyk LJ, Metheny-Barlow LJ, Thompson N, Knezevic V, Galperin M, Ramesh A, Dickson RB.

Department of Oncology, The Research Building, Room W417, Lombardi Comprehensive Cancer Center, Georgetown University Medical Center, 3970 Reservoir Road, NW, Washington, District of Columbia 20057, USA.

A naturally occurring, cocoa-derived pentameric procyanidin (pentamer) was previously shown to cause G0/G1 cell cycle arrest in human breast cancer cells by an unknown molecular mechanism. Here, we show that pentamer selectively inhibits the proliferation of human breast cancer cells (MDA MB-231, MDA MB-436, MDA MB-468, SKBR-3, and MCF-7) and benzo(a)pyrene-immortalized 184A1N4 and 184B5 cells. In contrast, normal human mammary epithelial cells in primary culture and spontaneously immortalized MCF-10A cells were significantly resistant. We evaluated whether this differential response to pentamer may involve depolarization of the mitochondrial membrane. Pentamer caused significant depolarization of mitochondrial membrane in MDA MB231 cells but not the more normal MCF-10A cells, whereas other normal and tumor cell lines tested gave variable results. Further investigations, using a proteomics approach with pentamer-treated MDA MB-231, revealed a specific dephosphorylation, without changes in protein expression, of several G1-modulatory proteins: Cdc2 (at Tyr15), forkhead transcription factor (at Ser256, the Akt phosphorylation site) and p53 (Ser392). Dephosphorylation of p53 (at Ser392) by pentamer was confirmed in MDA MB-468 cells. However, both expression and phosphorylation of retinoblastoma protein were decreased after pentamer treatment. **Our results show that breast cancer cells are selectively susceptible to the cytotoxic effects of pentameric procyanidin, and suggest that inhibition of cellular proliferation by this compound is associated with the site-specific dephosphorylation or down-regulation of**

several cell cycle regulatory proteins.-PMID: 15827326 [PubMed - indexed for MEDLINE]

Green Tea

Biochem Biophys Res Commun. 2005 Sep 2;334(3):947-53.-EGCG inhibits activation of the insulin-like growth factor-1 receptor in human colon cancer cells.- Shimizu M, Deguchi A, Hara Y, Moriwaki H, Weinstein IB.

Herbert Irving Comprehensive Cancer Center and Department of Medicine, Columbia University Medical Center, New York, NY 10032, USA; Department of Medicine, Gifu University School of Medicine, Gifu 501-1194, Japan.
The IGF/IGF-1R system, which includes the IGF, IGF-1R, and IGFBPs proteins, plays an important role in the development and growth of colorectal cancer. We previously reported that in the HT29 human colon cancer cell line EGCG, the major biologically active component of green tea, inhibits activation of the RTKs EGFR, HER2, and HER3, and that this is associated with inhibition of multiple downstream signaling pathways. Since IGF-1R is also a RTK, in this study we examined the effects of EGCG on the activity of IGF/IGF-1R system in human colon cancer cells. We found that the colon cancer cell lines Caco2, HT29, SW837, and SW480 express high levels of the IGF-1R receptor, and that both SW837 and SW480 cells display constitutive activation of this receptor. Treatment of SW837 cells with 20mug/ml of EGCG (the IC(50) concentration for growth inhibition) caused within 6h a decrease in the phosphorylated (i.e., activated) form of the IGF-1R protein. At 12h, there was a decrease in the levels of both IGF-1 protein and mRNA and within 3-6h there was an increase in the levels of both IGFBP-3 protein and mRNA. The increased expression of the latter protein was sustained for at least 48h. When SW837 cells were treated with EGCG for a longer time, i.e., 96h, a very low concentration (1.0mug/ml) of EGCG also caused inhibition of activation of IGF-1R, a decrease in the IGF-1 protein, and an increase in the IGFBP-3 protein. EGCG also caused a decrease in the levels of mRNAs that encode MMPs-7 and -9, proteins that proteolyze IGFBP-3. In addition, treatment with EGCG caused a transient increase in the expression of TGF-beta2, an inducer of IGFBP-3 expression. These findings expand the roles of EGCG as an inhibitor of critical RTKs involved in cell proliferation, **providing further evidence that EGCG and related compounds may be useful in the chemoprevention or treatment of colorectal cancer.** PMID: 16053920 [PubMed - in process]

Mutat Res. 2005 Jun 28;-Modulation of signal transduction by tea catechins and related phytochemicals.- Shimizu M, Weinstein IB.

Herbert Irving Comprehensive Cancer Center and Department of Medicine, Columbia University Medical Center, HHSC-1509, 701 West 168 Street, NY 10032-2704, USA.

Epidemiologic studies in human populations and experimental studies in rodents provide evidence that green tea and its constituents can inhibit both the development and growth of tumors at a variety of tissue sites. In addition, EGCG, a major biologically active component of green tea, inhibits growth and induces apoptosis in a variety of cancer cell lines. The purpose of this paper is to review evidence that these effects are mediated, at least in part, through inhibition of the activity of specific receptor tyrosine kinases (RTKs) and related downstream pathways of signal transduction. We also review evidence indicating that the antitumor effects of the related polyphenolic phytochemicals resveratrol, genistein, curcumin, and capsaicin are exerted via similar mechanisms. Some of these agents (EGCG, genistein, and curcumin) appear to directly target specific RTKs, and all of these compounds cause inhibition of the activity of the transcription factors AP-1 and NF-kappaB, thus inhibiting cell proliferation and enhancing apoptosis. Critical areas of future investigation include: (1) identification of the direct molecular target(s) of EGCG and related polyphenolic compounds in cells; (2) the in vivo metabolism and bioavailability of these compounds; (3) the ancillary effects of these compounds on tumor-stromal interactions; (4) the development of synergistic combinations with other antitumor agents to enhance efficacy in cancer prevention and therapy, and also minimize potential toxicities.-PMID: 15992833 [PubMed - as supplied by publisher]

Chem Rec. 2005;5(3):119-32.-Green tea: Health benefits as cancer preventive for humans.-Fujiki H.
Faculty of Pharmaceutical Sciences, Tokushima Bunri University, Yamashiro-cho, Tokushima 770-8514, Japan.

Green tea is an acknowledged cancer preventive in Japan. The aim of this review article is to develop the concept of cancer prevention with green tea beverage for humans, which has largely been our exclusive research territory. This paper briefly reviews several topics, beginning with the introduction of our initial work on penta-O-galloyl-beta-D-

glucose and (-)-epigallocatechin gallate (EGCG), the main constituent of green tea extract. The mechanisms of EGCG action, particularly the reduction of TNF-alpha are discussed, and we show how use of (3) H-EGCG revealed a wide range of target organs for cancer prevention. The results of an epidemiological study in Saitama Prefecture allowed us to determine the cancer preventive amount of green tea-10 Japanese-size cups per day, about 2.5 g green tea extract-which made it possible for us to introduce the two-stage strategy of cancer prevention with green tea. The first stage is the delay of cancer onset for the general population. The second stage is the prevention of recurrence of cancer for patients following cancer treatment. **Combination cancer prevention with green tea and cancer preventive drugs is proving especially beneficial for Japanese, who drink green tea every day.** And finally, the stimulating comments of Prof. Jim Watson have encouraged green tea scientists. (c) 2005 The Japan Chemical Journal Forum and Wiley Periodicals, Inc. Chem Rec 5: 119-132; 2005: Published online in Wiley InterScience (www.interscience.wiley.com) DOI 10.1002/tcr.20039. PMID: 15889414 [PubMed - in process]

Cardiovasc Res. 2005 Aug 1;67(2):317-25.-Catechins prevent vascular smooth muscle cell invasion by inhibiting MT1-MMP activity and MMP-2 expression.-El Bedoui J, Oak MH, Anglard P, Schini-Kerth VB.

Pharmacologie et Physico-Chimie des Interactions Cellulaires et Moleculaires, UMR CNRS 7034, France.

OBJECTIVE: Regular consumption of green tea is associated with a reduced risk of mortality due to coronary diseases and cancer. The present study examined whether a green tea extract (GTE) inhibits activation of matrix metalloproteinase-2 (MMP-2), a major collagenase involved in vascular remodeling of atherosclerotic plaques, in vascular smooth muscle cells (VSMCs). METHODS AND RESULTS: The expression of MMP-2 was assessed by Northern and Western blot analyses in human aortic VSMCs. MMP-2 activity was evaluated by zymography, membrane-type1-MMP (MT1-MMP, MMP-14) activity by an enzymatic assay, and cell invasion by a modified Boyden chamber assay. The thrombin-induced activation of secreted MMP-2 was abolished by GTE and the green tea polyphenols (-)-epigallocatechin-3-gallate (EGCG) and (-)-epicatechin-3-gallate (ECG). GTE reduced the expression of MMP-2 mRNA and protein. GTE, EGCG and ECG directly inhibited cell-associated MT1-MMP activity, the physiological activator of MMP-2, in a reversible manner. Thrombin-stimulated VSMCs invasion was abolished by EGCG and ECG, and reduced by GTE. CONCLUSIONS: GTE inhibits thrombin-induced VSMCs invasion most likely by preventing MMP-2 expression and its activation by a direct inhibition of MT1-MMP. **The ability of green tea to prevent cell invasion and matrix degradation might contribute to its protective effect on atherosclerosis and cancer.**-PMID: 15885676 [PubMed - in process]

Anticancer Res. 2005 Jan-Feb;25(1A):397-402.-Novel D-ring analog of epigallocatechin-3-gallate inhibits tumor growth and VEGF expression in breast carcinoma cells.-Waleh NS, Chao WR, Bensari A, Zaveri NT.

Drug Discovery Program, Biosciences Division, SRI International, Menlo Park, CA 94025, USA.

The cancer chemopreventive activity of green tea and its major polyphenolic constituent, epigallocatechin-3-gallate (EGCG) have been attributed to its antioxidant, antiproliferative and antiangiogenic effects. Several new molecular targets for EGCG's anticarcinogenic activity have been proposed in the recent literature. However, the understanding of the molecular mechanisms of EGCG's activity in vivo have been confounded by its low oral bioavailability and low plasma levels. Studies of EGCG would be greatly aided by the availability of synthetic analogs of EGCG designed to understand the contributions of the A, B, and D-rings and the phenolic hydroxyl groups of EGCG to its molecular mechanisms of action. We recently reported the de novo synthesis of a D-ring analog of EGCG, with the objective of using such analogs to understand the molecular mechanisms of EGCG action. We report here the first studies with a synthetic D-ring analog of EGCG. We examined the ability of the synthetic D-ring analog to inhibit tumor cell proliferation in breast carcinoma cells. We also investigated the effect of the analog on stress-induced VEGF production in breast carcinoma cells using Northern analysis and quantitative RT-PCR. We report here that the synthetic D-ring analog inhibits breast cancer cell growth in vitro with potencies equivalent to those of EGCG. Our results also show that, like EGCG, the synthetic analog inhibits hypoxia- and serum starvation-induced production of VEGF mRNA in breast cancer cells. Such synthetic analogs are valuable for understanding the structure-function relationship of EGCG and identifying relevant mechanisms of the chemopreventive action of EGCG.-PMID: 15816564 [PubMed - indexed for MEDLINE]

Arch Latinoam Nutr. 2003 Jun;53(2):111-8.- [The chemo-protector effects of tea and its components]-[Article in Spanish]- Gonzalez de Mejia E.

Department of Food Science and Human Nutrition, University of Illinois, Urbana-Champaign, USA.

Tea has been consumed worldwide since ancient times to maintain and improve health. Its main active components are a type of polyphenols known as flavonoids, which include catechins and theaflavins. **Several epidemiological studies suggest that the consumption of green tea could prevent cancer development in humans. Likewise, animal studies have shown that green tea consumption may inhibit the development of prostate and breast cancer.** It has been shown that, through several mechanisms, tea polyphenols present antioxidant, and anticarcinogenic activities, thus affording several health benefits. It is important to better characterize tea components, to study their bio-availability and bio-transformation in vivo and to conduct clinical studies of its main active compounds.-PMID: 14528600 [PubMed - indexed for MEDLINE]

J Nutr. 2002 Aug;132(8):2307-11.-Green tea inhibits vascular endothelial growth factor (VEGF) induction in human breast cancer cells.-Sartippour MR, Shao ZM, Heber D, Beatty P, Zhang L, Liu C, Ellis L, Liu W, Go VL, Brooks MN.

Department of Surgery, Division of Oncology and. Center for Human Nutrition, University of California, Los Angeles 90095, USA.

Investigators have shown that green tea and its main catechin epigallocatechin-3 gallate (EGCG) may decrease the risk of cancer. Our previous study showed that green tea extract (GTE) as well as its individual catechin components inhibited MDA-MB231 breast cancer cell and human umbilical vein endothelial cell (HUVEC) proliferation. Further, GTE suppressed breast cancer xenograft size and decreased the tumor vessel density in vivo. In the current study, we investigated the effect of GTE on the major angiogenic factor vascular endothelial growth factor (VEGF) in an in vitro experiment. GTE or EGCG (40 mg/L) significantly decreased the levels of the VEGF peptide secreted into conditioned media. This occurred in both HUVEC and human breast cancer cells and the effect was dose dependent. Furthermore, GTE and EGCG decreased the RNA levels of VEGF in MDA-MB231 cells. This inhibition occurred at the transcriptional regulation level and was accompanied by a significant decrease in VEGF promoter activity. We also showed that GTE decreased c-fos and c-jun RNA transcripts, suggesting that activator protein (AP)-1-responsive regions present in the human VEGF promoter may be involved in the inhibitory effect of GTE. Furthermore, GTE suppressed the expression of protein kinase C, another VEGF transcription modulator, in breast cancer cells. **Inhibition of VEGF transcription appeared to be one of the molecular mechanism(s) involved in the antiangiogenic effects of green tea, which may contribute to its potential use for breast cancer treatment and/or prevention**.-PMID: 12163680 [PubMed - indexed for MEDLINE]

Cancer Lett. 2001 Jun 26;167(2):175-82.-Regular consumption of green tea and the risk of breast cancer recurrence: follow-up study from the Hospital-based Epidemiologic Research Program at Aichi Cancer Center (HERPACC), Japan.- Inoue M, Tajima K, Mizutani M, Iwata H, Iwase T, Miura S, Hirose K, Hamajima N, Tominaga S.

Division of Epidemiology and Prevention, Aichi Cancer Center Research Institute, 1-1 Kanokoden, Chikusa-ku, Nagoya 464-8681, Japan. minoue@aichi-cc.pref.aichi.jp

Experimental studies suggest various features of anticancer activity of green tea including inhibitory effect of tumor invasion and metastasis. This study was conducted to examine the association between regular green tea consumption prior to diagnosis and subsequent risk of breast cancer recurrence. The Hospital-based Epidemiologic Research Program at Aichi Cancer Center (HERPACC) was started in 1988, in which information on lifestyle has routinely been collected from all first-visit outpatients by questionnaire. A total of 1160 new surgical cases of female invasive breast cancers with HERPACC information diagnosed between June 1990 and August 1998 were followed up through December 1999, and the risk (hazard ratio: HR) of recurrence was assessed with reference to daily green tea consumption using a Cox proportional hazard model. During 5264 person-years of follow-up, 133 subjects (12%) were documented to suffer recurrence of breast cancer. A decreased HR for recurrence adjusted for stage was observed with consumption of three or more daily cups of green tea (HR=0.69, 95% confidence interval (95%CI)=0.47-1.00). Particularly in stage I, the HR was decreased statistically significantly (HR=0.43, 95%CI=0.22-0.84). A similar tendency was observed for stage II subjects, but was not present among more advanced stages.

Although careful interpretation is needed, these results suggest the possibility that regular green tea consumption may be preventive against recurrence of breast cancer in early stage cases.-PMID: 11369139 [PubMed - indexed for MEDLINE]

J Cancer Res Clin Oncol. 1999 Nov;125(11):589-97.-Two stages of cancer prevention with green tea.-Fujiki H.

Cancer chemoprevention is a new and important medical science in its own right. On the occasion of my presentation entitled "Natural agents and cancer chemoprevention" at the 90th AACR Meeting in 1999, I summarized our recent results on cancer prevention with **green tea.** In this article, the present status of clinical trials supported by the Chemoprevention Branch of the National Cancer Institute in the United States is first described by way of introduction. Although various natural products are now under investigation in phase I clinical trials, green tea has, perhaps, the greatest potential for further development. In order to expand our understanding of the effects of tea polyphenols and green tea, I review their ability to inhibit growth and cause apoptosis of cancer cells, their distribution into target organs and their other cancer-preventing properties. In addition, the paper focuses on the significance of reducing tumor necrosis factor alpha (TNFalpha) gene expression in cells and TNFalpha release from cells as essential activities for cancer prevention. As for the amounts of green tea effective in cancer prevention, **I present two results from our Research Institute: a prospective cohort study with over 8000 individuals in Saitama Prefecture revealed that the daily consumption of at least ten Japanese-size cups of green tea resulted in delayed cancer onset, and a follow-up study of breast cancer patients conducted at our Hospital found that stages I and II breast cancer patients consuming over five cups per day experienced a lower recurrence rate and longer disease-free period than those consuming fewer than four cups per day**. Thus, I propose here, for the first time, the two-stage approach to analyzing cancer prevention with green tea: cancer prevention before cancer onset and cancer prevention following cancer treatment. As an additional example of cancer prevention with natural agents, kava, a daily beverage in Fiji, is mentioned. **All the evidence reminds us of the significance of alternative medicine in practical cancer prevention.**-PMID: 10541965 [PubMed - indexed for MEDLINE]

Mutat Res. 1999 Jul 16;428(1-2):339-44.-Green tea and cancer chemoprevention.-Suganuma M, Okabe S, Sueoka N, Sueoka E, Matsuyama S, Imai K, Nakachi K, Fujiki H.-Saitama Cancer Center Research Institute, Ina, Kitaadachi-gun, Saitama 362-0806, Japan.

Worldwide interest in green tea as a cancer preventive agent for humans has increased, because it is non-toxic and it is effective in a wide range of organs. (-)-Epigallocatechin gallate (EGCG) is the main constituent of green tea; the others are (-)-epicatechin gallate, (-)-epigallocatechin and (-)-epicatechin (EC). This paper reports the results of our latest pharmacological and biochemical studies with 3H-EGCG, along with studies on human subjects. The study on bioavailability of 3H-EGCG in mice revealed the wide distribution of radioactivity in multiple organs. Specifically, radioactivity was found in all reported target organs of EGCG and green tea extract (digestive tract, liver, lung, pancreas, mammary gland and skin) as well as other organs (brain, kidney, uterus and ovary or testes) in mice. Recently, we demonstrated that EC enhanced incorporation of 3H-EGCG into human lung cancer cell line PC-9 cells. EC along with another cancer preventive agent sulindac also synergistically enhanced apoptosis in PC-9 cells induced by EGCG. **Moreover, a case-control study on breast cancer patients revealed that high daily consumption of green tea was associated with a lower recurrence rate among Stages I and II patients. All the results suggest that consumption of green tea is a practical and effective cancer preventive both before cancer onset and after cancer treatment.**-PMID: 10518005 [PubMed - indexed for MEDLINE]

Arabinogalactan

Introduction

Larch arabinogalactan is a polysaccharide powder derived from the wood of the larch tree (Larix species) and comprised of approximately 98 percent arabinogalactan. Arabinogalactans are found in a variety of plants but are more abundant in the Larix genus, primarily Larix occidentalis (Western Larch). The Western Larch is unique among pines in that it loses its needles in the fall. Western Larch is also known as Mountain Larch or Western Tamarack and is native to the Pacific and Inland Northwest United States as well as parts of British Columbia, Canada.1 Larch

arabinogalactan is approved by the U.S. Food and Drug Administration (FDA) as a source of dietary fiber, but also has potential therapeutic benefits as an immune stimulating agent and cancer protocol adjunct.

Description and Biochemistry

Pharmaceutical-grade larch **arabinogalactan** is a fine, dry, off-white powder with a slightly sweet taste and mild pine-like odor. It dissolves completely in water or juice, is low in viscosity and therefore easy to administer, even to children. It is composed of galactose and arabinose molecules in a 6:1 ratio, with a small amount of glucuronic acid. Arabinogalactans are long, densely branched polysaccharides of varying molecular weights (10,000-120,000). **Lower molecular weight polysaccharides typically exhibit an anti-inflammatory, anti-complement, antiallergy effect, while those of higher weights stimulate natural killer (NK) cell cytotoxicity and reticuloendothelial cells.** In the case of larch arabinogalactan, molecular weights of the two major fractions are 16,000 and 100,000, perhaps accounting for its wide range of therapeutic properties.2

Pharmacokinetics

Human studies on the pharmacokinetics of larch arabinogalactan are few and the amount absorbed following an oral dose remains unclear. Animal studies indicate that intravenous injection of purified larch arabinogalactan results in 52.5 percent of the dose being present in the liver and 30 percent in the urine 90 minutes after dosing. Hepatic clearance occurred with a half-life of 3.42 days.3 Non-absorbed larch arabinogalactan is actively fermented by intestinal microflora and is particularly effective at increasing beneficial anaerobes such as Bifidobacteria and Lactobacillus.4

Clinical Indications

Dietary Fiber: **Larch arabinogalactan is an excellent source of dietary fiber that is able to increase short-chain fatty acid production (primarily butyrate) via its vigorous fermentation by intestinal microflora.**2 It is well documented that butyrate is essential for proper colon health as it is the preferred substrate for energy generation by colonic epithelial cells.5 Butyrate also acts as a protectant for the intestinal mucosa against disease and cancer-promoting agents.6 Arabinogalactan added to human fecal homogenates has also been shown to decrease ammonia generation, and therefore may be of clinical value in the treatment of portal-systemic encephalopathy, a disease characterized by ammonia build-up in the liver.4 Larch arabinogalactan given to human subjects increased levels of beneficial intestinal anaerobes, particularly Bifidobacterium longum, via their fermentation specificity for arabinogalactan compared to other complex carbohydrates.7,8

Cancer Protocols: **Larch arabinogalactan may be an effective adjunct to cancer therapies due to its ability to stimulate NK cell cytotoxicity, stimulate the immune system, and block metastasis of tumor cells to the liver.**2 Tumor metastasis to the liver is more common than to other organ sites, probably due to tumor cell specificity for lectin-like receptor sites found in liver parenchyma. Animal studies have demonstrated arabinogalactan's ability to inhibit or block lectin receptor sites, thereby reducing tumor cell colonization of the liver and also increasing survival time of the subjects.9-11 Pretreatment with larch arabinogalactan was found to stimulate NK cell cytotoxicity via potentiation of the cytokine network, primarily via an increase in the release of gamma interferon.12

Pediatric Otitis: Media Recurrent otitis media is common in pediatric populations and it appears that improving immune system function might lead to a decrease in both frequency and severity of this condition. **Research has demonstrated larch and other arabinogalactans to be capable of enhancing the immune response to bacterial infection via stimulation of phagocytosis, competitive binding of bacterial fimbriae, or bacterial opsonization.** This was found to be particularly true for infection by gram negative organisms such as Escherichia coli and Klebsiella species.2,13 In addition, D'Adamo reports a decrease in occurrence and severity of otitis media in pediatric patients supplemented prophylactically with larch arabinogalactan.2 Larch arabinogalactan's mild taste and excellent solubility in water and juice make it a relatively easy therapeutic tool to employ in pediatric populations.

Chronic Disease: A number of chronic diseases are characterized by decreased NK cell activity, including chronic fatigue syndrome,14 viral hepatitis,15,16 HIV/AIDS,2 and autoimmune diseases such as multiple sclerosis.17 Stimulation of NK cell activity by larch arabinogalactan has been associated with recovery in certain cases of chronic

fatigue syndrome.18 Viral hepatitis (hepatitis B and C) is also characterized by a decrease in NK cell cytotoxicity15,16 and therefore these patients may benefit from its stimulation by larch arabinogalactan. In the case of multiple sclerosis, a small 2-year study of patients with the relapsing/remitting type concluded that disease severity was correlated with NK cell functional activity, supporting the hypothesis that NK cells play a role in the immunopathogenesis of this disease.17 Consequently, stimulation of NK cell cytotoxicity might be of clinical benefit to these patients. Patients with HIV/AIDS develop low CD4 cell counts and often are plagued by opportunistic infections. By virtue of its immune-stimulating properties, larch arabinogalactan has been shown to effect a slight increase in CD4 cell counts, in addition to decreasingsusceptibility to opportunistic pathogens.2

Hepatic Drug Delivery: Hepatic uptake of an injected dose of larch arabinogalactan resulted in 52.5 percent of the dose arriving in the liver. Due to its high hepatic concentration and its ability to increase vascular permeability,19 larch arabinogalactan has been suggested as a vehicle for administering diagnostic or therapeutic agents to the liver.3

Platelet Washing Medium: Larch arabinogalactan solution has been studied as a medium for use in platelet washing; a technique employed to separate platelets from platelet-rich plasma. The washed platelets can then be used in transfusions, bioassays, and research. Platelets washed with larch arabinogalactan solution were free of plasma proteins and retained both normal morphology and function.20

Side-Effects and Toxicity: **Larch arabinogalactan is a safe and effective immune-stimulating phytochemical. It is FDA-approved for use as a dietary fiber and in food applications.** Both acute and long-term toxicity studies in rats and mice reveal no evidence of toxicity.21 Human consumption is usually without side-effects; however, a small percentage of people (<3%) experienced bloating and flatulence, possibly due to the vigorous fermentation of the arabinogalactan by intestinal microflora.2 Because of its excellent safety profile and solubility in water and juice, larch arabinogalactan is considered a safe, effective immune-stimulating agent for pediatric use.

Dosage

Larch arabinogalactan in powder form is typically dosed in teaspoons or tablespoons at a concentration of approximately 4-5 grams per tablespoon. The typical adult dosage is one to three tablespoons per day in divided doses; the pediatric dose is one to three teaspoons per day. The powder is usually mixed with water or juice but can be added to food if desired.

References

1. Odonmazig P, Ebringerova A, Machova E, Alfoldi J. Structural and molecular properties of the arabinogalactan isolated from Mongolian larchwood (Larix dahurica L.). Carbohydr Res 1994;252:317-324.
2. D'Adamo P. Larch arabinogalactan. J Naturopath Med 1996;6:33-37.
3. Groman EV, Enriquez PM, Jung C, Josephson L. Arabinogalactan for hepatic drug delivery. Bioconjug Chem 1994;5:547-556.
4. Vince AJ, McNeil NI, Wager JD, Wrong OM. The effect of lactulose, pectin, arabinogalactan, and cellulose on the production of organic acids and metabolism of ammonia by intestinal bacteria in a faecal incubation system. Br J Nutr 1990;63:17-26.
5. Roediger WE. Utilization of nutrients by isolated epithelial cells of the rat colon. Gastroenterology 1989;83:424-429.
6. Tsao D, Shi Z, Wong A, Kim YS. Effect of sodium butyrate on carcinoembryonic antigen production by human colonic adenocarcinoma cells in culture. Cancer Res 1983;43:1217-1222.
7. Crociani F, Alessandrini A, Mucci MM, Biavati B. Degradation of complex carbohydrates by Bifidobacterium spp. Int J Food Microbiol 1994;24:199-210.
8. Slavin J, Feirtag J, Robinson R, Causey J. Physiological effects of arabinogalactan (AG) in human subjects. Unpublished research.
9. Hagmar B, Ryd W, Skomedal H. Arabinogalactan blockade of experimental metastases to liver by murine hepatoma. Invasion Metastasis 1991;11:348-355.
10. Beuth J, Ko HL, Oette K, et al. Inhibition of liver metastasis in mice by blocking hepatocyte lectins with arabinogalactan infusions and D-galactose. J Cancer Res Clin Oncol 1987;113:51-55.
11. Beuth J, Ko HL, Schirrmacher V, et al. Inhibition of liver tumor cell colonization in two animal tumor models by lectin blocking with D-galactose or arabinogalactan. Clin Exp Metastasis 1988;6:115-120.

12. Hauer J, Anderer FA. Mechanism of stimulation of human natural killer cytotoxicity by arabinogalactan from Larix occidentalis. Cancer Immunol Immunother 1993;36:237-244.
13. Reith FJ. Pharmaceuticals containing lactic acid derivatives and Echinacea. Bundesrepublik Deutsches Patentamt 27 21 014 11/16/78. [German Patent]
14. Levine PH, Whiteside TL, Friberg D, et al. Dysfunction of natural killer activity in a family with chronic fatigue syndrome. Clin Immunol Immunopathol 1998;88:96-104.
15. Corado J, Toro F, Rivera H, et al. Impairment of natural killer (NK) cytotoxicity activity in hepatitis C virus (HCV) infection. Clin Exp Immunol 1997;109:451-457.
16. Machado IV, Deibis L, Risquez E, et al. Immunoclinical, molecular and immunopathologic approach to chronic viral hepatitis. Therapeutic considerations. GEN 1994;48:124-132. [Article in Spanish]
17. Kastrukoff LF, Morgan NG, Zecchini D, et al. A role for natural killer cells in the immunopathogenesis of multiple sclerosis. J Neuroimmunol 1998;86:123-133.
18. Uchida A. Therapy of chronic fatigue syndrome. Nippon Rinsho 1992;50:2679-2683.
19. Kind LS, Macedo-Sobrinho B, Ako D. Enhanced vascular permeability induced in mice by larch arabinogalactan. Immunology 1970;19:799-807.
20. Hill RJ, Stenberg PE, Sullam PM, Levin J. Use of arabinogalactan to obtain washed murine platelets free of contaminating plasma proteins and appropriate for studies of function, morphology, and thrombopoiesis. J Lab Clin Med 1988;111:73-83.
21. Wagner H. Low molecular weight polysaccharides from composite plants containing arabinogalactan, arabinoglucan, and arabinoxylan. Bundesrepublik Deutsches Patentamt DE 3042491 7/15/82. [German Patent]

Trace Minerals

Vet Clin North Am Small Anim Pract. 2004 Jan;34(1):249-69, viii.-The use of nutraceuticals in cancer therapy.-Roudebush P, Davenport DJ, Novotny BJ.

Technical Information Services, Hill's Pet Nutrition, Inc. Hill's Science and Technology Center, PO Box 1658, Topeka, KS 66601, USA. phil_roudebush@hillspet.com

The high prevalence of nutraceutical use among human patients with cancer suggests that the use of nutraceuticals in pet animals with cancer is probably common. Dogs with a wide variety of malignant diseases have significant alterations in carbohydrate, protein, and fat metabolism. These metabolic alterations may be ameliorated by using functional foods relatively low in soluble carbohydrate, moderate amounts of protein that includes sources of arginine, and moderate amounts of fat supplemented with omega-3 long-chain polyunsaturated fatty acids. Well-controlled clinical studies in a variety of species with cancer, including rodents, people, and dogs, have documented that increased dietary and serum levels of omega-3 fatty acids are associated with a number of health benefits, including improved disease-free interval, survival time, and quality of life. **Other nutraceuticals of interest in patients with cancer include antioxidant vitamins, trace minerals, glutamine, protease inhibitors, garlic, tea polyphenols, vitamin A, and shark cartilage.** Publication Types: •Review• Review, Tutorial-PMID: 15032131 [PubMed - indexed for MEDLINE]

J Am Coll Nutr. 1998 Jun;17(3):244-9.-Effects of supplementation with a combination of antioxidant vitamins and trace elements, at nutritional doses, on biochemical indicators and markers of the antioxidant system in adult subjects.-Preziosi P, Galan P, Herbeth B, Valeix P, Roussel AM, Malvy D, Paul-Dauphin A, Arnaud J, Richard MJ, Briancon S, Favier A, Hercberg S.

Institut Scientifique et Technique de la Nutrition et l'Alimentation, Conservatoire National des Arts et Metiers, Paris, France.

OBJECTIVE: To test the impact of supplementation with nutritional doses of antioxidant nutrients on biochemical indicators of vitamin and trace element levels. DESIGN: A randomized double-blind trial was performed comparing two groups receiving daily either a combination of vitamins (beta-carotene, 6 mg; vitamin C, 120 mg; and vitamin E, 30 mg) and trace elements (zinc, 20 mg; and selenium, 100 micrograms); or a placebo. SUBJECTS: 401 subjects (166 males aged 45 to 60 years and 235 females aged to 35 to 60 years).
MEASURE OF OUTCOME: Biological markers of vitamin and trace element status and free radical parameters were

measured initially, 3 months, and 6 months after supplemention. RESULTS: Mean serum concentrations of alpha-tocopherol, vitamin C, beta-carotene, zinc and selenium increased significantly after 3 months of supplementation in the group receiving multivitamins associated with minerals. At baseline, 18.2% of the men and 5.1% of the women had low concentrations of serum vitamin C (< 20 mumol/l): 2.4% of the men and 17% of the women presented low concentrations of serum retinol (< 1.4 mumol/l): 18.7% of men and 10% of women had serum beta-carotene < 0.30 mumol/l. None of the study subjects had serum alpha-tocopherol concentrations below the limit cut-off point (< 9.3 mumol/l). Low serum zinc concentrations (< 10.7 mumol/l) were found in 15.1% of men and 23.8% of women. Low serum selenium concentrations (< 0.75 mumol/l) were found in 6% of men and 6.4% of women. A significant increase in plasma and red cell GPx activity was observed in groups receiving supplementation. No modifications were observed after 6 months of supplementation for malondyaldehyde. **CONCLUSION: This study demonstrates the efficacy of an intake of antioxidant vitamins and trace elements, given at nutritional doses, on biochemical indicators of vitamin and trace elements.**-Publication Types:• Clinical Trial• Randomized Controlled Trial-PMID: 9627910 [PubMed - indexed for MEDLINE]

Support Care Cancer. 1993 Nov;1(6):295-7.-Critical reappraisal of vitamins and trace minerals in nutritional support of cancer patients.- Stahelin HB.-Geriatric University Clinic, Kantonsspital, Basel, Switzerland.

The potential of a high intake of fresh fruits and vegetables in cancer prevention is well established. **Epidemiological studies support carotene, vitamins A, C, E and selenium as the active compounds.** Antioxidant properties and direct effects (e.g. inhibition of N-nitrosamine formation or cell-to-cell interactions) are invoked. **The role of other trace elements is less clear. The modulation of immune function by vitamins and trace elements remains important and affects survival.** In established cancers, the site-specific differences in the diet/cancer relation require appropriate dietary changes, e.g. low fat (20% by energy) in breast cancer, or high vegetable or fruit intake in lung cancer. Single high-dose supplements (e.g. Vitamin C) have proved to have no curative or life-prolonging effect. **Chemotherapy and radiation increase the requirements for antioxidant compounds. Supplementation can diminish the damage induced by peroxidation**. Carefully planned and monitored trials that establish the optimal intake of micronutrients as adjuvants in cancer patients are required.-Publication Types:• Review• Review, Tutorial-PMID: 8156246 [PubMed - indexed for MEDLINE]

Cancer. 1985 Jan 1;55(1 Suppl):295-300.-Micronutrient requirements of cancer patients.-Hoffman FA.

Several major factors may influence the micronutrient requirements of the patient with cancer. These factors include the metabolic state of the malignancy and its effects on host metabolism, the catabolic effects of antineoplastic therapy, and other physiologic stresses commonly associated with the treatment of cancer, i.e., surgery, fever and infection. Although the nutritional importance of vitamins, minerals, and trace elements is recognized, the optimal daily dose that will preserve lean body mass without enhancing tumor growth is not known. Recommended Dietary Allowances (RDAs), where established, are based on populations with nonmalignant diseases. **However, supplementation with vitamins, minerals, and certain trace elements is recommended for the cancer patient who requires prolonged parenteral support, since clinically relevant deficiency states have been described. The effect of malignancy on the metabolism of several of these micronutrients (iron, ascorbic acid, alpha tocopherol, selenium, zinc, copper) is discussed**.-PMID: 3917362 [PubMed - indexed for MEDLINE]

I use the Trace minerals from **Youngevity**. You can find them at **http://27672901.youngevityonline.com.**

After reading the following two articles I cannot stress enough the importance of eating anti-inflammatory foods. I know with today's lifestyles it may be difficult to eat the right foods all the time that is why I like the convenience of the **Ganoderma Lucidum and Excellium products by Gano Excel. I'll talk more about these later on.**

I included these next two articles, because I want to bring to your attention the potential dangers of inflammation.

Inflammation – The Root of All Illness?

Roman Bystrianyk, "Inflammation – The Root of All Illness?", Health Sentinel, July 27, 2005,

Inflammation is an integral part of the immune system. We're all familiar with inflammation. When you're cut it becomes red and swollen as a response by the immune system and as the cut heals the inflammation dies down. A

similar underlying, chronic, low-grade inflammation is now being considered by more and more scientists as a major cause of diseases not only for obvious diseases like arthritis and asthma, but also for heart disease, diabetes, Alzheimer's, and even cancer. A recent special edition of Newsweek examines this quiet hazard.

Years ago oxidation was being considered as the main culprit in many diseases. Now oxidation is grabbing more of the attention. According to neuroscientist James Joseph of Tufts University, **"Inflammation is the evil twin of oxidation.** Where you find one, you find the other." This discovery is solving "medical puzzles" such as people with high blood pressure have an increased risk for Alzheimer's or why people with rheumatoid arthritis have higher rates of sudden cardiac death. All these conditions are tied with a connecting thread of inflammation.
When your cut heals the inflammation recedes, but constant exposure to cigarette smoke, excess cholesterol, and low-grade infections can contribute to a low-grade, chronic inflammation. The inflammation simmers like, "a low flame on the back burner that we're unaware of until the pot burns."

Diabetes has emerged as a recent example. The connection between type II diabetes and obesity are so well known that some researchers consider the two combined into a single disease of "diabesity". According to the article, "When you gain weight, fat cells grow more biochemically active, churning out inflammatory compounds. As obesity ratchets up inflammation, inflammation in turn promotes insulin resistance, a central feature of diabetes and the so-called metabolic syndrome that precedes it."

Like diabetes, heart disease is linked with obesity. According to Dr. Peter Libby, chief of cardiovascular medicine at Brigham and Women's Hospital in Boston, **"Inflammation is the alpha and omega of atherosclerosis.** It's there at every step of the process." In the process plaque formation starts when cholesterol sticks to the artery walls and oxidizes. This triggers an immune response that attempts to clean up the problem. The inflammatory response is the body's attempt to heal, but encourages the formation of larger plaques that can eventually block the artery and result in a heart attack or stroke.

Certain cancers are also being linked to inflammation. According to Lisa Coussens, a cancer biologist at the University of California in San Francisco, **"people with chronic inflammatory bowel diseases have tremendously enhanced risk of colon cancer."** Some triggers of inflammation include, "cigarette smoke in the lungs, persistent infections like hepatitis C in the liver and chronic heartburn, which repeatedly irritates the lining of the esophagus with gastric acid." The result includes oxidative damage to the DNA which sometimes cripples the suicide mechanism of the cell that would often allow abnormal cells to self-destruct.

Although anti-inflammatory medications seem like an obvious answer they are fraught with problems. Inflammatory chemicals also serve important functions in the body and stopping their action may have a positive effect such as decreasing pain, but they can also have serious negative impacts. Vioxx is an example where inhibiting the COX-2 inflammatory enzyme relieved pain, but also impeded the process to prevent blood clots from forming in the arteries. Dr David Graham, an employee of the Food and Drugs Administration, estimated that up to 139,000 Americans have died or have been seriously injured as a result of taking Vioxx.

Even standard arthritis medications called NSAIDs have serious consequences. According to a June 1999 New England Journal of Medicine each year over 16,000 people die from gastrointestinal bleeding because of the unintended interference in the body's healing mechanism of the digestive tract. According the journal, **"It has been estimated conservatively that 16,500 NSAID-related deaths occur among patients with rheumatoid arthritis or osteoarthritis every year in the United States.** This figure is similar to the number of deaths from the acquired immunodeficiency syndrome and considerably greater than the number of deaths from multiple myeloma, asthma, cervical cancer, or Hodgkin's disease. If deaths from gastrointestinal toxic effects from NSAIDs were tabulated separately in the National Vital Statistics reports, these effects would constitute the 15th most common cause of death in the United States."

While drugs block a single target molecule greatly reducing its activity, natural anti-inflammatories have a wide-ranging, gentler action. According to Greg Cole a professor of medicine and neurology at UCLA, "you'll get a greater safety and efficacy reducing five inflammatory mediators by 30 percent than by reducing one by 100 percent."

Aside from avoiding the promoters of inflammation, such as cigarette smoke, there are approaches that can be used to turn down the heat on inflammation. Exercise and decreasing weight help reduce inflammation in the fat and liver

cells. A diet rich in vegetables, fruits, whole grains, and omega-3 fatty acids also turns down inflammation.

The omega-3 fatty acids have been shown in dozens of studies to help prevent heart attacks by "preventing arrhythmias, making blood less likely to clot in the arteries, improving the balance of good and bad cholesterol and limiting inflammation." **The omega-3s are found in coldwater fish such as salmon, sardines, and mackerel as well as walnuts, flaxseeds, and dark leafy greens.**

A diet rich in fruits and vegetables also helps. One anti-inflammatory compound that has been extensively studied is curcumin. Curcumin is the yellow pigment in the spice turmeric. Professor Cole has found that small doses of Curcumin reduce a number of inflammatory markers such as TNF-alpha (Tumor Necrosis Factor alpha) and IL-1 (Interlukin-1).

The article concludes, "The beauty of these lifestyle changes is that they're so low tech, affordable and effective. We may all have it within our grasp to reduce inflammation – if we can just muster the willpower." **SOURCE: Newsweek Special Edition, Summer 2005**

The following is an excerpt from **Oncologoy Vol 16, No 2 (February 2002) Chronic Inflammation and Cancer.** If you would like to read the entire article you can go to: http://www.cancernetwork.com/journals/oncology/o0202d.htm

Emily Shacter, PhD
Senior Investigator, Laboratory of Immunology, Center for Biologics Evaluation and Research, Food and Drug Administration, Bethesda, Maryland

Sigmund A. Weitzman, MD
Professor, Department of Medicine, Section of Hematology/Oncology, Northwestern University Medical School, Chicago, Illinois

Introduction
Inflammatory Conditions That Predispose to Cancer
General Mechanisms of Proneoplastic Activity
Proneoplastic Inflammatory Mediators
Treatment and Prevention
References

Reviewers' comments:
Krystyna Frenkel, PhD, New York University School of Medicine, New York, New York
Alan B. Weitberg, MD, Brown University Medical School, Providence, Rhode Island; Boston University School of Medicine, Boston, Massachusetts

A substantial body of evidence supports the conclusion that chronic inflammation can predispose an individual to cancer, as demonstrated by the association between chronic inflammatory bowel diseases and the increased risk of colon carcinoma. Chronic inflammation is caused by a variety of factors, including bacterial, viral, and parasitic infections, chemical irritants, and nondigestible particles. The longer the inflammation persists, the higher the risk of associated carcinogenesis. This review describes some of the underlying causes of the association between chronic inflammation and cancer. Inflammatory mediators contribute to neoplasia by inducing proneoplastic mutations, adaptive responses, resistance to apoptosis, and environmental changes such as stimulation of angiogenesis. All these changes confer a survival advantage to a susceptible cell. In this article, we discuss the contribution of reactive oxygen and nitrogen intermediates, prostaglandins, and inflammatory cytokines to carcinogenesis. A thorough understanding of the molecular basis of inflammation-associated neoplasia and progression can lead to novel approaches to the prevention and treatment of cancer. [ONCOLOGY 16:217-232, 2002]

Chronic inflammation may be a causative factor in a variety of cancers. In general, the longer the inflammation persists, the higher the risk of cancer. Hence, acute inflammation, such as occurs in response to a transient infection, is not regarded as a risk factor for the development of neoplasia, although many of the same molecular mediators are generated in both acute and chronic inflammation. In general, inflammatory

leukocytes such as neutrophils, monocytes, macrophages, and eosinophils provide the soluble factors that are thought to mediate the development of inflammation-associated cancer, although other cells, including the cancer cells themselves, also participate.

Inflammatory mediators include metabolites of arachidonic acid, cytokines, chemokines, and free radicals. Chronic exposure to these mediators leads to increased cell proliferation, mutagenesis, oncogene activation, and angiogenesis. The ultimate result is the proliferation of cells that have lost normal growth control. Animal models provide experimental evidence that chronic inflammation can promote cancer and further insights into possible mechanisms.
This review will summarize the clinical association between chronic inflammation and cancer and will describe the inflammatory factors and pathways that are thought to be proneoplastic. Emphasis will be placed on examining the role of the reactive oxygen and nitrogen intermediates, cytokines, and prostaglandins.

Now that you know how damaging inflammation can be I urge you to do your best to include natural anti-inflammatory foods, Trace Minerals, Mangosteen, and Aloe Vera in your diet.

Calcium D-Glucarate

I have been hearing a lot of good things about Calcium D-Glucarate so I decided to investigate. Here is some of the science that I found.

Integr Cancer Ther. 2003 Jun;2(2):139-44. -Detoxifying cancer causing agents to prevent cancer.- Hanausek M, Walaszek Z, Slaga TJ.

AMC Cancer Research Center, Denver, CO 80214, USA. hanausekm@amc.org

Different vitamins and other micronutrients in vegetables, fruits, and other natural plant products may prevent cancer development (carcinogenesis) by interfering with detrimental actions of mutagens, carcinogens, and tumor promoters. The goal of current studies in cancer prevention is to determine the mechanisms of synergistic action of the natural source compounds known to inhibit one or more stages of carcinogenesis, that is, initiation and promotion/progression. Many natural cancer preventive agents are effective inhibitors of tumor initiation, promotion, and/or progression. The mechanism of action is related to their abilities to prevent critical carcinogen metabolism and to increase detoxification of carcinogens and tumor promoters. The authors review here the potential role of the detoxification system and, in particular, the roles of D-glucaric acid, and the enzyme beta-glucuronidase in early detection and prevention of cancer. There is now growing evidence for the possible control of different stages of the cancer induction by inhibiting beta-glucuronidase with D-glucaric acid derivatives, especially with its salts (D-glucarates). D-Glucaric acid has been found in many vegetables and fruits. **Therefore, the consumption of fruits and vegetables naturally rich in D-glucaric acid or self-medication with D-glucaric acid derivatives such as calcium D-glucarate offers a promising cancer prevention approach.**-Publication Types:* Review* Review, Tutorial-PMID: 15035900 [PubMed - indexed for MEDLINE]

Cancer Detect Prev. 1997;21(2):178-90. - Metabolism, uptake, and excretion of a D-glucaric acid salt and its potential use in cancer prevention.-Walaszek Z, Szemraj J, Narog M, Adams AK, Kilgore J, Sherman U, Hanausek M.

University of Texas M. D. Anderson Cancer Center, Science Park-Research Division, Smithville, USA.

D-Glucaric acid (GA) is a nontoxic, natural compound. One of its derivatives is the potent beta-glucuronidase inhibitor D-glucaro-1,4-lactone (1,4-GL). The goal of this study was to demonstrate the in vivo formation of 1,4-GL from a D-glucarate salt and determine its metabolism, uptake by selected organs, and excretion following oral administration of potassium hydrogen D-[14C]glucarate to male and female Sprague-Dawley rats. 1,4-GL increases detoxification of carcinogens and tumor promoters/progressors by inhibiting beta-glucuronidase and preventing hydrolysis of their glucuronides. 1,4-GL and its precursors, such as potassium hydrogen D-glucarate and calcium D-glucarate, may exert their anticancer action, in part, through alterations in steroidogenesis accompanied by changes in the hormonal environment and the proliferative status of the target organ. Thus, GA derivatives may be useful as new

or adjuvant cancer preventive and therapeutic agents. In our study, 1,4-GL was found to be formed from the D-glucarate salt in the stomach of rats. It was apparently absorbed from the gastrointestinal tract, transported with the blood to different internal organs, and excreted in the urine and to a lesser extent in bile. There were no significant differences in the metabolism of PHG between male and female rats. **Thus, formation of 1,4-GL from D-glucaric acid derivatives may be prerequisite for their inhibition of chemical carcinogenesis in rodents and prevention of breast, prostate, and colon cancer in humans.**PMID: 9101079 [PubMed - indexed for MEDLINE]

Here is an article by Stewart A. Lonky, MD on Calcium D-Glucarate. After reviewing the science on Calcium D-Glucarate I decided it would be easier to include Dr. Lonky's article.

Calcium D-Glucarate

The following is a current review of information concerning D-Glucarate. By Stewart A. Lonky, MD, FACP

Much of the stimulus for this work came from the observation that populations who had diets very rich in fruits and vegetables had a lower incidence of cancer. The outcome of this work was the isolation of D-Glucarate from fruits and vegetables. This "purification" and use of D-Glucarate is patented.

In 1986 Walaszek and co-workers demonstrated that taking D-Glucarate orally, in animals and humans, leads to a slow release of a substance that inhibits glucuronidase. Glucuronidase is an enzyme that thwarts the body's efforts to rid itself of cancer causing substances known as carcinogens. Walaszek demonstrated that if you feed animals Glucarate, there is an increase in the level of a substance known as D-Glucaro-lactone, which inhibits glucuronidase. He looked at a model for breast cancer induction in rats, the animal used most frequently for breast cancer research. Rats given anthracene develop breast cancer, but if they were pre-treated with dietary Glucarate, tumor development was blocked in over 70% of the animals. It was shown that when D-Glucarate was fed to the animals, the levels of estradiol (the form of estrogen that causes breast cancer) were decreased in the blood. In summery, D-Glucarate lowers the level of glucuronidase, and in so doing allows the body to eliminate harmful carcinogens (cancer causing chemicals).

In 1986 these same researchers found that by giving the "active" agent, D-Glucaro-lactone by mouth favored the reduction of glucuronidase activity for one hour, using the Calcium D-Glucarate salt led to a 5-hour effect. These experiments were performed in animals fed various carcinogens and the level of free carcinogen or carcinogen bound to DNA was measured in the blood. With calcium D-Glucarate, these levels were drastically reduced over a sustained period of time. There was a direct correlation between the decrease in DNA binding of carcinogen and the ability to induce tumor formation. In essence, D-Glucarate administration favors the elimination of carcinogens in the stool, and the effect lasts for hours after a single low dose.

In 1990 Walaszek moved to the MD Anderson Carcinogenesis center at Houston. He published an article showing that Calcium D-Glucarate leads to a decrease in the "proliferation" of tumors themselves, in other words, once present this agent can decrease their growth.

In 1991, 1992, and 1993 there were a number of articles that tested D-Glucarate in human tumor cell cultures. In these studies Glucarate was added to derivatives of retinoic acid (a compound from vitamin A). Results demonstrated that the addition of D-Glucarate led to an increase in the anti-tumor activity of rentinoids.

In 1994 Walaszek and co-workers demonstrated that in certain human tumor cell culture lines, D-Glucarate was a potent anti-proliferative agent when used alone, without retinoic acid. There was an inability to stimulate tumor cell growth by the usual means when the tissue cultures were treated with D-Glucarate.

In 1995 Walaszek and co-workers demonstrated that feeding D-Glucarate to animals was always followed by conversion to the D-Glucaro-lactone product, and that this conversion led to an increase in the blood levels of this compound. In these studies a number of different carcinogens were used to try and induce breast cancer in rats. Although these carcinogens led to breast cancer in rats fed a placebo, those fed Glucarate did not develop breast cancer. The main carcinogen used in these studies was N-methyl-N-nitrosurea.

Work by other investigators, including Walaszek, in 1995 and 1996 showed that Calcium D-Glucarate and the Potassium hydrogen D-Glucarate were both excellent inhibitors of colon cancer in experimental animal models.

The most recent work on specific tumors has shown that the absorption, metabolism, and effectiveness of D-Glucarate was similar in both male and female animals. There is tumor inhibition shown for breast, prostate, lung, and colon cancer, and the mechanism of action is identical in each...there is a decrease in glucuronidase activity, a decrease in carcinogen level (because the body eliminates the carcinogen) and a decrease in tumorogenesis.

In summary, D-Glucarate is a naturally occurring substance that is not present in sufficient amounts to counteract natural and external carcinogens. By supplementing D-Glucarate as the calcium salt, we can get a long lasting effect of Glucarate, and this effect is to favor the body's natural defense mechanism for eliminating carcinogens. Without Glucarate, the body cannot efficiently eliminate these cancer-causing agents because of the interference from glucuronidase. By supplementing the diet with Glucarate, we can block glucuronidase activity and the body can rid itself of the carcinogens, thus preventing many forms of cancer, including lung, breast, prostate, and colon-Stewart A. Lonky, MD, FACP

References

1. Walaszek, Z., Hanausek, M., Szemraj, J., and Adams, A.K. 1998, D-Glucarate acid as a prospective tumor marker. Meth. Mol. Med., 14, 487-495.

2. Walaszek, Z., Szemraj, J., Adams, A.K., and Hanausek, M. 1992, Reduced levels of D-Glucaric acid in mammary tumor-bearing hosts and the effect of its supplementation during estrogen replacement and tamoxifin therapy. Proc. Am. Assoc. Cancer Res. 37: 183.

3. Heerdt, A.S., Young, C.W.., and Borgen, P.I., 1995, Calcium Glucarate as a chemopreventative agent in breast cancer., Isr. J. Med. Sci. 31: 101-105.

4. Walaszek, Z. Chemopreventative properties of D-Glucaric acid derivatives. Cancer Bull 1993; 45: 453-457.

5. Walaszek, Z., Szemraj, J., Adams, A.K., Kordari, P., and Hanausek, M. 1992, Reduced levels of D-Glucaric acid in mammary tumor-bearing Host. Breast Cancer Res. Treat., 375: 108.

6. Walaszek, Z., Hanausek, M., Adams, A.K. and Sherman, U. 1991, Cholesterol lowering effects of dietary D-Glucarate. Faseb J., 5: A930.

7. Walaszek, Z., Hanausek, M., Sherman, U. and Adams, A.K. 1990, Antiproliferative effect of dietary glucarate on the Sprague Dawley in rat mammary gland. Cancer Lett. 49: 51-57.

8. Walaszek, Z., Adams, A.K., Sherman, U., Viaje, A., Rotstein, J.B., Hanausek, M. and Slaga, T.J. 1990, Antiproliferate effects of Calcium D-Glucarate (CG) and D-glucaro-1,4-lactone (GL) on the rat mammary gland, colon and mouse skin. Proc. Am. Assoc. Cancer Res., 31: 124. p>

9. Walaszek, Z. 1990, Potential use of D-Glucarate acid derivatives in cancer prevention. Cancer Lett. 54: 1-8.

10. DiGiovanni, J., 1990, Inhibition of chemical carcinogenesis. In: Chemical Carcinogenesis and Mutagenesis II, Cooper, C.S. and Grover, P.L. (eds.), Springer Verlag, Berlin, pp. 159-224.

11. Walaszek, Z., Adams, A.K., and Flores, F., 1989, Inhibition of 7,12-dimethylbenz(a)-anthracene(DMBA)-induced rat mammary carcinogenesis by glucarate. Proc. Am. Assoc. Cancer Res., 30: 170.

12. Abbou-Issa, H., Koolemans-Beynen, A., Minton, J.P. and Webb, T.E., 1989, Synergistic interaction between 13-cis-retinoic acid and glucarate: activity against rat mammary tumor induction and MCF-7 cells. Biochem. Biophys. Res. Commun.,163: 1364-1369.

13. Dwivedi, C., Oredipe, O.A., Barth, R.F., Downie, A.A. and Webb, T.E., 1989, Effects of the experimental chemopreventative agent, glucarate on intestinal carcinogenesis in rats. Carcinogenesis, 10: 1539-1541.

14. Oredipe, O.A., Barth, R.F., Dwivedi, C. and Webb, T.E., 1989, Chemopreventative activity of dietary glucarate on azoxymethane-induced altered hepatic loci in rats. Res. Commun. Chem. Pathol. Pharmacol., 65: 345-359.

15. Dwivedi. C., Downie, A.A. and Webb, T.E., 1989, Modulation of chemically initiated and promoted skin tumorigenesis in CD-1 mice by dietary glucarate. J. Environ. Path. Toxicol. Oncol., 9: 253-259.

16. Walaszek, Z., Hanausek, M., Sherman, U., Del Rio, M. and Adams, A.K., 1989, Effects of (+) glucaric acid derivatives and tamoxifen on human breast cancer cells (MCF-7). Breast Cancer Res. Treat., 14: 175.

17. Walaszek, Z., Flores, F. and Adams, A.K., 1988, Effect of dietary glucarate on estrogen receptors and growth of 7,12-dimethylbenz[a] anthracene-induced rat mammary carcinomas. Breast Cancer Res. Treat., 12: 128.

18. Walaszek, Z., Hanausek-Walaszek, M. and Webb, T.E., 1988, Repression by sustained release or glucuronidase inhibitors of chemical carcinogen-mediated induction of a marker oncofetal protein in rodents. J. Toxicol. Environ. Health, 23: 15-27.

19. Abbou-Issa, H.M., Duruibe, V.A., Minton, J.P., Larroya, S., Dwivedi, C., and Webb, T.E., 1988, Putative metabolites derived from dietary combinations of calcium glucarate and N-(4hydroxypheny)retinamide act synergistically to inhibit the induction of rat mammary rumors by 7,12-dimethylbenz[a]-anthracene. Proc. Natl. Acad. Sci. USA. 85: 4181-4184.

20. Oredipe, O.A., Barth, R.F., Hanausek-Walaszek, M., Sautins, I., Walaszek, Z. and Webb, T.E. 1987, Effects of an inhibitor

of B-glucuronidase on hepatocarcinogenesis. Proc. Am. Assoc. Cancer Res., 28: 156.
21. Oredipe, O.A., Barth, R.F., Hanausek-Walaszek, M., Sautins, I. Walaszek, Z. and Webb, T.E. 1987, Effects of calcium glucarate on the promotion of diethylnitrosamine-initiated altered hepatic loci in rats. Cancer. Lett., 38, 95-99.
22. Walaszek, Z., Hanausek-Walaszek, M., Minton, J.P. and Webb, T.E. 1986, Dietary glucarate as antipromoter of 7,12-dimethylbenz[a]-anthra-cene-induced mammary tumorigenesis. Carcinogenesis, 7:1463-1466.
23. Minton, J.P., Walaszek, Z., Hanausek-Walaszek, M., and Webb, T.E. 1986, B-Glucuronidase levels in patients with fibrocystic breast disease. Breast Cancer Res. Treat., 8: 217-222.
24. Walaszek, Z., Hanausek-Walaszek, M., Webb, T.E., 1986, Dietary glucarate-mediated reduction of sensitivity of murine strains to chemical to chemical carcinogenesis. Cancer Lett., 33: 25-32.

You can eat foods high in glucaric acid (a form of calcium D-glucarate) such as **apples, brussels sprouts, broccoli, cabbage, and bean sprouts.** Or you could take a Calcium D-Glucarate supplement. You can go to your local health food store and ask for Calcium D-Glucarate or even search the web. Just type in the words Calcium D-Glucarate.

Fulvic Acid

While searching the Internet for products that you should know about regarding your health and cancer I came across this information regarding Fulvic Acid.

U.S. Senate Document 264, as well as the 1992 Earth Summit statistics, indicates that the mineral content of the world's farm and range soil has decreased dramatically. Over the last 100 years, the depletion of essential mineral nutrients in the soil in North America is estimated to average 85%, with some US farms depleted by 100%. This means that in most cases we are getting as little as 15% of the minerals and nutrients that were once readily available in our food sources -- sometimes less. These statistics show that the American soils are so depleted of their natural resources that they no longer provide plant foods with the mineral elements essential to human nourishment and health!

The natural extract from years of accumulated plant deposit and from water running through the humic deposit enters the plants through the root system. These humic substances affect the soil and consequently plant growth and quality. Also, as a result of pesticide and herbicide usage and the mineral and nutrient depletion, most of the microbial life in the soil is gone. Without the microbes necessary for plants to convert inorganic substances to organic, the plants become deficient in minerals and nutrients.

One of the organic minerals that has been greatly diminished through the years is fulvic acid (not to be confused with folic acid). With less fulvic acid produced, the plants themselves will take up less minerals and nutrients. The deficiency of fulvic acid may be the most critical factor missing in our diets today. Fulvic acids are vital in delivering substantial amounts of nutrients and minerals and their energies to the living cells.

One of the functions of fulvic acid is to balance and energize all cell life and biological properties it comes in contact with. In addition to the diminished nutritional value of our food, and because the cell walls get stiffer and thicker with age, they cannot get as much vital nutrients and oxygen as they once did. Fulvic acid is reported to relieve oxygen deficiency and increase the vital activity of cells. New energy will be gained just by providing your stressed out cells with the nutrients and oxygen needed.

Also, if you can restore individual cells to their normal chemical balance and electrical potential, then you have given those cells life and the potential to function at peak performance. Fulvic acid has been reported to "induce revitalization" to the cells. Flooding nutrients into your cells gives new vitality and energizes the entire body.

When fulvic acid acts upon a substance, its molecular size and weight are altered. This reportedly enables it to pass through cell membranes. Fulvic makes cell walls more permeable, so nutrients can more easily enter the cell, as well as allowing waste to leave the cells more readily.

One of the strongest advantages of fulvic minerals is the belief that the absorption greatly exceeds traditional tablet supplements. As with any nutrient or supplement, the only way your body can benefit is if it is absorbed. Fulvic is

believed to enhance this process. It also may intensify the metabolism of proteins and is thought to increase the body's ability to go after viruses, pathogens, and bacterial infections of all kinds. It therefore not only bolsters immunity, it stimulates and helps to regulate the immune system.

We know that certain diseases are not caused by outside invaders, but by the immune system's defense mechanisms attacking the body itself. Fulvic acid is thought to have the unique ability to selectively suppress or inhibit certain immune responses, while at the same time naturally increasing the body's immune response where necessary. This ability to selectively control, stimulate, and regulate the immune system is one of the reasons fulvic acid is being studied with such enthusiasm.

In addition to carrying essential nutrients to the cell, it has been shown that fulvic acid may be an excellent natural chelator of toxins and can reduce them to a harmless state. Fulvic acid is effective at neutralizing and detoxifying a wide range of toxic materials, heavy metals and other pollutants. It is essential to wash away the waste and toxins that cells produce.

Harmful free radicals are known to circulate throughout the body, injuring tissue, altering genes, disrupting crucial processes in the body, forming cell mutations and making cells susceptible to infections and diseases. Free radicals are a major contributing factor to nearly all situations of non-ideal health.

Fulvic acid is believed to bond to these free radicals, transforming them into organic, usable substances, or if the cell is too damaged, it is eliminated as waste.

All cells have electrical potential, when the electrical potential of a cell is reduced, progressive weakness and illness may occur. A person's electrical potential may be lowered by loss of blood or fluids, overwhelming emotional stress, accidents, lack of sleep, surgical shock, lingering infections, fatigue or an unbalanced diet. Cells disintegrate when their electrical potential is reduced to zero.

Scientists theorize that electrical and chemical balances at the cellular level can be created and controlled by electrolytes (substances that are soluble in water and are capable of conducting electrical current). Fulvic acid has been shown to be one of nature's most powerful organic electrolytes.>

Organic fulvic acid electrolytes charge, recharge, and restore the potential that is or once was normal to the cell, and in doing so, balances and supercharges cellular life. The fulvic acid electrolytes are thought to greatly increase the percentage rate of absorption through the digestive system of minerals, nutrients, vitamins, herbs and amino acids into the circulatory system.

In addition, fulvic acid is considered by many to be one of the safest and most powerful antiviral substances known. Although not an antibiotic in the technical sense, they provide an antibiotic-effect. A generally accepted benefit of fulvic acid supplementation is that it can be used indefinitely without fear of creating antibiotic resistant strains of disease.

When combining fulvic acid with nutritional supplements, you get the benefits of improved synergistic reactions that you cannot get by taking the ingredients individually.

As unfair as it may seem, you cannot expect to live a sedentary lifestyle, eat fast food, consume gallons of caffeinated drinks, go without exercise, and be in optimal health just by adding a dietary supplement. You must do all you can to help and support your natural body processes. Providing your system with necessary nutrients through supplementation is a wise step, but you must also eat healthy, balanced meals, get at least moderate exercise and plenty of rest, and drink beverages that hydrate your body.

Take control of your health today. A healthy lifestyle can transform your life in ways you and your family may really appreciate for years to come.

Fulvic Acid References

- Aiken, G. R., D. M. McKnight, and P. MacCarthy. Humic Substances of Soil, Sediment and Water. New York: Wiley-Interscience, 1985. Aluise, Rick. Health and Vitality. Grand Junction: Vital-Earth Minerals.
- Azo, S., and I. Sakai. Soil Science and Plant Nutrition. Tokyo
- Baker, W. E. "Vital Electrolytes." Geochimlen at Casmochtulan Acts 37 (1973): 267-281.
- Balch MD, James F., and Phyllis A. Balch. Prescription for Nutritional Healing. 2nd ed. New York.
- Bergh, J. J., et al. "Non Cataytic Oxidation of Water-Slurried Coal with Oxygen: Identification of Fulvic Acids and Acute Toxicity." Fuel 6.2 (1997): 149-154.
- Bhattacharya, Salil K.. "Activity of Shilajit of Alloxan-Induced Hyperglycemia in Rats."
- Bhattacharya, Salil K., et al.. "Mast Cell Protecting Effect of Shilajit and its Constituents." Phytotherapy Research 3.6 (1998).
- Chaboussou, F. "Les Planets Malades de Pesticides - Bases Nouvelles D'une Prevention Contre Maladies er Parasites (plants made sick by pesticides - new basis for the prevention of diseases and pests)." 1980
- Christman, R. F., and E. T. Gjessing. Aquatic and Terrestrial Humic Materials. Kent: The Butternut Grove, 1983.
- Davies, G. "Properties and Functions of Humic Acids." The Nucleus (1996).
- Davies, G., and E. A. Ghabbour, ed. Humic Substances: Structures, Properties and Uses. Cambridge: Royal Society of Chemistry, 1998.
- Davies, G., et al. . "Aerogels: New High Surface Area Forms of Solid Humic Acids." The Role of Humic Substances in the Ecosystems and in Environmental Protection. Ed. J. Drozd, S. Gonet, N. Senei, and J. Weber. Wroclaw: Polish Society of Humic Protection, 1998. 85-90.
- Davies, G., et al.. "Ash and Metal Fredd Humic Acids." The Role of Humic Substances in the Ecosystems and in Environmental Protection. Ed. J. Drozd, S. Gonet, N. Senei, and J. Weber. Wroclaw: Polish Society of Humic Protection, 1998. 717-722.
- Davies, G., et al.. "Isolation of Humic Acids." The Role of Humic Substances in the Ecosystems and in Environmental Protection. Ed. J. Drozd, S. Gonet, N. Senei, and J. Weber. Wroclaw: Polish Society of Humic Protection, 1998. 567-572.
- Davies, G., et al.. "Models for Adsorption on Humic acids." The Role of Humic Substances in the Ecosystems and in Environmental Protection. Ed. J. Drozd, S. Gonet, N. Senei, and J. Weber. Wroclaw: Polish Society of Humic Protection, 1998. 36-42.
- Davies, G., and E. A. Ghabbour. "Understanding Life after Death." Chem. & Industry June 1999: 426-430.
- Davies, G., et al.. "A Site Model for Adsorption of Aqueous Nucleobases, Nucleosides and Nucleotides on Compost-Derived Humic Acid." Journal of Physical Chemistry 101B (1997): 3228-3239.
- Davies, G., et al.. "Humic Acids are Versatile Natural Polymers." Advanced New Materials and Emerging technologies 1995: 677-685.
- Davies, G., et al.. "Isolation of Humic Acid from the Terrestrial Plant Brugmansia saguinea." Sci. Total Environ. 201 (1997): 79-87.
- Davies, G., et al.. "Tight Metal Binding by Peat and Soil Derived Humic Acids." Canadian Journal of Soil Science 81 (2001): 331-336.
- Davies, G., et al.. "Tight Metal Binding by Peat and Soil Derived Humic Acids." Humic Substances and Chemical Contaminants. Ed. C. E. Clapp, M. H. B. Hayes, N. Senesi, and P. R. Bloom. Soil Science Society of America, 2001. 371-395.
- Davies, G., et al.. "Tight Metal Binging by Humic Acids and its Role in Biomineralisation." J. Chem. Soc. (1997): 4047-4060.
- Davies, G., E. A. Ghabbour, and C. Steelink. "Humic Acids: Marvelous Products of Soil Chemistry." J. Chem. Edu. 78 (2001): 1609-1614.
- Ealey, P. A., L. D. Kohn, N. J. Marshall, and R. P. Ekins. "Forskolin Stimulation of Naphthylamidase in Guinea Pig Thyroid Sections Detected with a Cytochemical Bioassay." Acta Endocrinol 108.3 (1985): 367-371.
- Gamble, D. S., and M. Schnitzer. "Trace Metals and Metal-Organic Interactions in Natural Waters." Ann Arbor Science (1974).

- Ghabbour, A., and G. Davies, ed. Understanding Humic Substances: Advanced Methods, Properties and Applications. Cambridge: Royal Society of Chemistry, 1999.
- Ghabbour, E. A., et al.. "Adsorption of a Plant and a Soil Derived Humic Acid on the Common Clay Laolinite." Humic Substances: Structures, Properties and Uses. Ed. G. Davies, and E. A. Ghabbour. Cambridge: Royal Society of Chemistry, 1998. 185-194.
- Ghabbour, E. A., et al.. "Adsorption of Nucleic Acid Constituents on Metal-loaded Solid Peat and Soil Derived Humic Acids." Canadian Journal of Soil Science 81 (2001): 309-316.
- Ghabbour, E. A., et al.. "Isolation of Humic Acid from the Brown Alga Pilayella littoralis." Journal of Applied Pharmacology 6 (1994): 459-468.
- Ghabbour, E.A., and G. Davies, ed. Humic Substances: Versatile Components of Plants, Soil and Water. Cambridge: Royal Society of Chemistry, 2000.
- Hartwell, J. L. "Types of Anticancer Agents Isolated from Plants." Cancer Treatment 60: 1031-1067.
- Huang, W. H., and W. D. Delier. "Dissolution of Rock-Forming Silicate Minerals in Organic Acids; Simulated First Stage Weathering of Fresh Mineral Surfaces." America Mineralogical Journal 55 (): 2076-2097.
- Jackson, W. R. Humic, Fulvic and Microbial Balance: Organic Soil Conditioning. Evergreen: Jackson Research Center, 1993.
- Jansen, S. A., et al.. "Distribution of Polysaccharides in Humic Acids Derived from Plant Sources." The Role of Humic Substances in the Ecosystems and in Environmental Protection. Ed. J. Drozd, S. Gonet, N. Senei, and J. Weber. Wroclaw: Polish Society of Humic Protection, 1998. 157-162.
- Jansen, S. A., et al.. "Structural Modeling in Humic Acid." Material Science Engineer C 4 (1996): 175-179.
- Jansen, S., et al.. "The Role of Metal Complexation in the Solubility and Stability of Humic Acid." Material Science Engineer C 4 (1996): 181-187.
- Kanonova, M. M. Soil Organic Matter. Elmsford: Pergamon, 1966.
- Khairy, A. H., et al.. "Adsorption of Aqueous Nucleobases, Nucleosides and Nucleotides on Compost Derived Humic Acid. 1. Naturally Occurring Pyrimidines" Journal of Physical Chemistry 100 (1996): 2410-2416.
- Khairy, A. H., et al.. "Adsorption of Aqueous Nucleobases, Nucleosides and Nucleotides on Compost Derived Humic Acid. 2. Naturally Occurring Purines" Journal of Physical Chemistry 100 (1996): 2417-2423.
- Khristeva, L. A., and M. V. Luk Yaneko. "Role of Physiologically Active Substances in Soil-Humic Acids, Bitumens and Vitamins B, C, P-PA and D in the Life of Plants and Their Replenishment." Soviet Soil Sciences 1962: 1137-1141.
- Klocking, R.. "Humic Substances as Potential Therapeutics." 6th International Meeting of the International Humic Substances Society. , Monopoli, Italy. 20 September 1992.
- Kodmans, H., H. Schnitzer, and M. Jaakkimainen. "Chlorite and Biotite Weathering by Fulvic Acid Solutions in Closed and Open Systems." Canadian Journal of Soil Science 63 (1983): 619-629.
- Kowalska, M., A. Denys, and J. Bialek. Acta Pol. Pharm. 50.4-5 (1993): 393-395. Ling, W. H, and P. J. H. Jones. "Dietary Phytosteroils: A Review of Metabolism, Benefits and Side Effects." Life Science 57 (1995): 195-206.
- MacCarthy, P.. "An Introduction to Soil Humic Substances." International Humic Substances Society. , Chicago, Illinois. 2 December 1985.
- Madei, J. A., J. Kuryszko, and T. Garbulinski. Acta Pol. Pharm. 50.4-5 (1993): 397-404. Malcolm, R. L.. "Variations Between Humic Substances Isolated from Soils, Stream Waters and Groundwaters as Revealed by C-NMR Spectroscopy." International Humic Substances Society. , Chicago, Illinois. 2 December 1985.
- Mao, J., et al.. "Quantitative Characterization of Humic Acids Solid-State C13 NMR." Soil Sci. Amer. J. 64 (2000): 873-884.
- Mao, J., et al.. "Structure and Elemental Composition between Solid-State C13 NMR and Chemical Analysis." Humic Substances: Structures, Properties and Uses. Ed. G. Davies, and E. A. Ghabbour. Cambridge: Royal Society of Chemistry, 1998. 79-90.
- Paciolla, M. D., et al.. "Generation of Free Radicals by HA: Implications for Biological Activity." Humic Substances: Structures, Properties and Uses. Ed. G. Davies, and E. A. Ghabbour. Cambridge: Royal Society of Chemistry, 1998. 203-214.

- Paciolla, M. D., et al.. "Generation of Hydroxyl Radicals from Metal-Loaded Humic Acids." Environmental Science Technology 33 (1999): 1814-1818.
- Paciolla, M. D., et al. . "Generation of Hydroxyl Radicals from Metal-Loaded Humic Acids (correction)." Environmental Science Technology 34 (2000): 728.
- Padoe, H.L., Townshend, Al, Clerc, J.T., VenderLinden (Eds.) Analytica Chimica Acts, Special Issue, Humic and Fulvic Compounds, 232 (1), 1-235. Amsterdam, The Netherlands: Elsevier Science Publishers. (1990, May 1).
- Poapst, P. A., and M. Schnitzer. "Fulvic Acid and Adventitious Root Formation." Soil Biology and Biochemistry 3 (1971): 215-219.
- Radwan, A. , et al.. "Rapid Supercritical Fluid Extraction of Humic Acids." The Role of Humic Substances in the Ecosystems and in Environmental Protection. Ed. J. Drozd, S. Gonet, N. Senei, and J. Weber. Wroclaw: Polish Society of Humic Protection, 1998. 91-96.
- Radwan, A., et al.. "Isolation of Humic Acid from the Brown Algae Ascophyllum nodosum, Fucus versiculosus, Lamininaria and the marine angiosperm Zostera marina." Journal of Applied Phycology 8 (1997): 553-562.
- Radwan, A., et al.. "Supercritical Fluid CO2 Extraction Accelerates Isolation of Humic Acid from Live Alga Pilayella littoralis." Journal of Applied Phycology 8 (1997): 545-551.
- Rashid, M. A,. Geochemistry of Marine Humic Substances. New York: Springer-Verlag, 1985.
- Satyavati, G. V. Plants and Traditional Medicine. 5th ed. : Academic Press Limited, 1982.
- Schauss, Dr A. G. "Mineral and Trace Element Food Supply.".
- Schnitzer, M.. Symposium on Soil Organic Matter Studies. , Braunsweig.
- Schnitzer, M., and H. Dodama. "Reactions of Minerals with Soil Humic Substances." Minerals in Soil Environments. Ed. J. B. Dixon, and S. B. Weeds. Madison: Soil Society of America, 1977 .
- Schnitzer, M., and S. U. Khan. Humic Substances in the Environment. New York: Dekker, 1972.
- Senesi, N., Y. Chen, and M. Schnitzer. "The Role of Humic Acids in Extra-Cellular Transport and Chemical Determination of pH in Natural Waters." Soil Biology and Biochemistry 9 (1977): 397-403.
- Shibnath, Ghosal, et al.. "Antiulcerogenic Activity of Fulvic Acids and 4-methoxy-6-carbomethoxybiphenyl Isolated from Shilajit." Phytotherapy Research 2.2 (1988) .
- Sposito, G., et al.. "Trace Metal chemistry in Arid-Zone Filed soils Amended with Sewage Sludge: Comparative Study of the Fulvic Acid Fraction." Soil Science Society America Journal 45 (1982): 265-270.
- Swanson, H. L.. "Natural Organic Acids as Agents of Chemical Weathering." U. S. Geological Survey. , Washington, D. C.. 1970.
- Syltie, P. W. "Effects of Very Small Amounts of Highly Active Biological Substances on Plant Growth." Biological Agriculture and Horticulture 2 (1985): 245-269.
- Varnum, J. M., et al.. "Metal Uptake by "Metal Free" Humic Acid." The Role of Humic Substances in the Ecosystems and in Environmental Protection. Ed. J. Drozd, S. Gonet, N. Senei, and J. Weber. Wroclaw: Polish Society of Humic Protection, 1998. 741-746.
- Varnum, L. T., et al.. "Conformational Modeling of a Proposed Building Block of Humic Acid: Searching Chirally Undefined Conformational Space." The Role of Humic Substances in the Ecosystems and in Environmental Protection. Ed. J. Drozd, S. Gonet, N. Senei, and J. Weber. Wroclaw: Polish Society of Humic Protection, 1998. 73-77.
- Visser, S. A.. "Effects of Humic Substances on Higher Animals and Man: Possible Use of Humic Compounds in Medical Treatments." International Humic Substances Society. , Sevilla, Spain. 1988.
- Wiley, R. J., et al.. "Supercritical CO2 Drying of Peat and Soil Derived Humic Acid Gels." Understanding and Managing Organic Matter in Soil, Sediments and Water. Ed. R. S. Swift, and K. M. Sparks. Adelaide: International Substances Society, 2001. 47-52.
- Williams, Dr. Roger J. The Wonderful World within You. Wichita: Bio-communications Press, 1977.
- Williams, S. T. "Are Antibiotics Produced in Soil?." Pedobiologia 23 (1963): 426-435.
- Wilson MD, Denis E. Wilson's Syndrome: The Miracle of Feeling Well. : Cornerstone Publishing Company, 1988.
- Xing, B., et al.. "Evaluation of Different Solid-State C13 NMR Techniques for Characterizing Humic Acids" Understanding Humic Substances: Advanced Methods, Properties and Applications. Ed. G. Davies, and E. A. Ghabbour. Cambridge: Royal Society of Chemistry, 1998. 49. York: Avery Publishing Group, 1997.

Yuan, Shenyuan, et al.. Application of Fulvic Acid and its Derivatives in the Fields of Agriculture and Medicine. First ed., 1993.

If you are taking Fulvic Acid now, great if not I found a product that contains Fulvic Acid, Arabinogalactan and Aloe, it is called **Healose** by Nutriharmony. To learn more go to **www.NutriHarmony.com/healthy1**

Morinda citrifolia (Noni)

J Nat Prod. 2007 May 25;70(5):754-757. Epub 2007 May 5.

Anti-inflammatory and Potential Cancer Chemopreventive Constituents of the Fruits of Morinda citrifolia (Noni).-Akihisa T, Matsumoto K, Tokuda H, Yasukawa K, Seino KI, Nakamoto K, Kuninaga H, Suzuki T, Kimura Y.

College of Science and Technology, Nihon University, 1-8 Kanda Surugadai, Chiyoda-ku, Tokyo 101-8308, Japan, Department of Biochemistry and Molecular Biology, Kyoto Prefectural University of Medicine, Kamigyo-ku, Kyoto 602-0841, Japan, College of Pharmacy, Nihon University, 7-7-1 Narashinodai, Funabashi-shi, Chiba 274-8555, Japan, and Nakazen Company Ltd., 1190 Chinen, Shimajiri-gun, Okinawa 901-1513, Japan.

A new anthraquinone, 1,5,15-tri-O-methylmorindol (1), and two new saccharide fatty acid esters, 2-O-(beta-d-glucopyranosyl)-1-O-hexanoyl-beta-d-gluropyranose (4) and 2-O-(beta-d-glucopyranosyl)-1-O-octanoyl-beta-d-gluropyranose (5), have been isolated from a methanol extract of the fruits of Morinda citrifolia (noni) along with 10 known compounds, namely, two anthraquinones (2, 3), six saccharide fatty acid esters (6-11), an iridoid glycoside (12), and a flavanol glycoside (13). Upon evaluation of six compounds (5-7, 9, 10, and 13) for inhibitory activity against 12-O-tetradecanoylphorbol-13-acetate (TPA)-induced inflammation (1 mug/ear) in mice, four saccharide fatty acid esters, 5-7 and 9, exhibited potent anti-inflammatory activity, with ID50 values of 0.46-0.79 mg per ear. In addition, when compounds 1-13 were evaluated against the Epstein-Barr virus early antigen (EBV-EA) activation induced by TPA, all of the compounds exhibited moderate inhibitory effects (IC50 values of 386-578 mol ratio/32 pmol TPA).-PMID: 17480098 [PubMed - as supplied by publisher]

Hawaii Med J. 2004 Jun;63(6):182-4. Are immune responses pivotal to cancer patient's long term survival? Two clinical case-study reports on the effects of Morinda citrifolia (Noni).-Wong DK.-
kongwaiwong1@hotmail.com
In the State of Hawaii, there are abundant claims of benefit from cancer patients' use of the fruit juice of Morinda citrifolia (Noni). There is no well documented clinical report in peer review journals. The author here studiously examined 2 such claims through interview, review of the medical records and pathology slides. **The author concludes that these cases are valuable experiences and hope to stimulate interest in Noni research as an important part of adjuvant immunotherapy for cancer.-**PMID: 15298088 [PubMed - indexed for MEDLINE]

Ann N Y Acad Sci. 2001 Dec;952:161-8.

Cancer preventive effect of Morinda citrifolia (Noni).-Wang MY, Su C.

Department of Pathology, UIC College of Medicine, Rockford, Illinois 61107, USA. **mianwang@uic.edu**

Morinda citrifolia (Noni) has been extensively used in folk medicine by Polynesians for over 2,000 years. It has been reported to have broad therapeutic effects, including anticancer activity, in both clinical practice and laboratory animal models. The mechanism for these effects remains unknown. The hypothesis that Morinda citrifolia possesses a cancer preventive effect at the initiation stage of carcinogenesis was studied. Our preliminary data indicated that 10% Tahitian Noni Liquid Dietary Supplement or Tahitian Noni Juice (TNJ), made from Morinda citrifolia fruit by Morinda Inc, in drinking water for one week was able to prevent DMBA-DNA adduct formation. The levels of DMBA-DNA adducts were reduced by 30% in the heart, 41% in the lung, 42% in the liver, and 80% in the kidney of female SD rats. Even more dramatic results were obtained in male C57 BL-6 mice: 10% TNJ was able to reduce DMBA-DNA adduct formation by 60% in the heart, 50% in the lung, 70% in the liver, and 90% in the kidney. In order to explore the mechanism of this preventive effect, the antioxidant activity of TNJ was examined in vitro by lipid hydroperoxide (LPO) and tetrazolium nitroblue (TNB) assays. In the LPO assay, LPO oxidizes leucomethylene blue

to methylene blue in the presence of hemoglobin. The resultant blue color was quantified at 660 nm spectrophotometrically. In the TNB assay, superoxide anion radicals (SAR) reduce TNB into formazan blue that was also measured by absorption at 602 nm. TNJ showed a dose-dependent inhibition of both LPO and SAR in our system. The antioxidant activity of TNJ was compared to the effects of vitamin C, grape seed powder (GSP), and pycnogenol (PYC) at the daily dose per serving level recommended by U.S.RDAs or manufacturers. **The results suggest that prevention of carcinogen-DNA adduct formation and the antioxidant activity of TNJ may contribute to the cancer preventive effect of Morinda citrifolia.**-PMID: 11795436 [PubMed - indexed for MEDLINE]

Phytother Res. 1999 Aug;13(5):380-7.

An immunomodulatory polysaccharide-rich substance from the fruit juice of Morinda citrifolia (noni) with antitumour activity.-Hirazumi A, **Furusawa E**.

Department of Pharmacology, John A., Burns School of Medicine, 1960 East West Road, University of Hawaii, Honolulu, HI 96822, USA.

The fruit juice of Morinda citrifolia (noni) contains a polysaccharide-rich substance (noni-ppt) with antitumour activity in the Lewis lung (LLC) peritoneal carcinomatosis model. Therapeutic administration of noni-ppt significantly enhanced the duration of survival of inbred syngeneic LLC tumour bearing mice. It did not exert significant cytotoxic effects in an adapted culture of LLC cells, LLC1, but could activate peritoneal exudate cells (PEC) to impart profound toxicity when co-cultured with the tumour cells. This suggested the possibility that noni-ppt may suppress tumour growth through activation of the host immune system. Concomitant treatment with the immunosuppressive agent, 2-chloroadenosine (C1-Ade) or cyclosporin (cys-A) diminished its activity, thereby substantiating an immunomodulatory mechanism. Noni-ppt was also capable of stimulating the release of several mediators from murine effector cells, including tumour necrosis factor-alpha (TNF-alpha), interleukin-1beta (IL-1beta), IL-10, IL-12 p70, interferon-gamma (IFN-gamma) and nitric oxide (NO), but had no effect on IL-2 and suppressed IL-4 release. **Improved survival time and curative effects occurred when noni-ppt was combined with sub-optimal doses of the standard chemotherapeutic agents, adriamycin (Adria), cisplatin (CDDP), 5-fluorouracil (5-FU), and vincristine (VCR), suggesting important clinical applications of noni-ppt as a supplemental agent in cancer treatment**. Copyright 1999 John Wiley & Sons, Ltd.-PMID: 10441776 [PubMed - indexed for MEDLINE]

Ganoderma Lucidum (Reishi)

When I learned of the Ganoderma Lucidum mushroom I was intrigued. So I did my investigation, as I always do, on **www.pubmed.gov.** I was very impressed with all of the science that I found supporting its use. I was also very impressed with the results my daughter had with the Ganoderma. I have included a few of the hundreds of articles that I found for your education.

J Ethnopharmacol. 2007 May 4;111(2):219-26. Epub 2006 Nov 21.

Ganoderma lucidum polysaccharides enhance the function of immunological effector cells in immunosuppressed mice. Zhu XL, Chen AF, Lin ZB.

Department of Pharmacology, School of Basic Medical Science, Peking University Health Science Center, 38 Xueyuan Road, Beijing 100083, PR China.

The present study was designed to determine in vivo efficacy of Ganoderma lucidum polysaccharides (Gl-PS) for enhancing the activity of immunological effector cells in immunosuppressed mice. Mice were injected intraperitoneally (i.p.) once daily with low-dose (2.5mg/kg), intermediate-dose (25mg/kg), and high-dose (250mg/kg) of Gl-PS, respectively, for 7 consecutive days 24h after i.p. injection of a immunosuppressing anti-tumor agent cyclophosphamide (Cy, 300mg/kg). In Cy-treated mice, compared to vehicle, low-dose Gl-PS accelerated recovery of bone marrow cells, red blood cells and white blood cells, as well as splenic natural killer cells and natural killer T cells, and enhanced T and B cell proliferation responses on day 8, cytotoxic T lymphocyte activity on day 5, as well as NK cell and lymphokine activated killer cell activity on days 7-9. Furthermore, it promoted phagocytosis and cytotoxicity

of macrophages on day 12. The above beneficial effects induced by the low-dose Gl-PS treatment did not result in any side effects. **These results demonstrate the efficacious effects of low-dose Gl-PS treatment for enhancing the activity of immunological effector cells in immunosuppressed mice, and may provide a basis for applying this herb as an efficacious adjacent immunopotentiating therapy against cancer chemotherapy-induced immunosuppression**. PMID: 17182202 [PubMed - in process]
Zhongguo Zhong Yao Za Zhi. 2006 Oct;31(19):1618-22.

[Antitumor activity of extracts of Ganoderma lucidum and their protective effects on damaged HL-7702 cells induced by radiotherapy and chemotherapy]-[Article in Chinese] Wang DH, Weng XC.

School of Life Sciences, Shanghai University, Shanghai 200444, China.

OBJECTIVE: To study the inhibitory effect of Ganoderma lucidum, the extract of chloroform, the extract of ethyl acetate and the remains after two-time extraction on BEL-7402 and MGC-803 cells and their protective effects on HL-7702 cells pre-and post-exposed to cisplatin (DDP) and various doses of 60Co gamma irradiation. METHOD: The antitumor activity and protective effects on damaged HL-7702 cells induced by radiotherapy and chemotherapy of ganoderma lucidum were determined by MTT technique. RESULT: The anticancer activity of the extract of chloroform Ganoderma lucidum was the best: at the concentration of 0.125 mg x mL(-1), the inhibitory rate was over 50%. To the HL-7702 cells damaged by DDP, four kinds of extracts didn't exert restoring effect, but the pretreatment with the extract of chloroform reduced the damaged degree significantly. To the 60Co gamma irradiated HL-7702 cells, only the extract of chloroform exerted restoring effect to some extent when exposed to middle or high dose of irradiation. The pre-administration of four kinds of extracts reduced the damaged degree by radiation. **CONCLUSION: The extract of chloroform exerts notable antitumor effects on cancer cells and protective effects on damaged normal cells induced by radiotherapy and chemotherapy**. PMID: 17165589 [PubMed - in process]

Oncol Rep. 2006 Dec;16(6):1181-7.

Inhibitory effect of a water-soluble extract from the culture medium of Ganoderma lucidum (Rei-shi) mycelia on the development of pulmonary adenocarcinoma induced by N-nitrosobis (2-hydroxypropyl) amine in Wistar rats. Kashimoto N, Hayama M, Kamiya K, Watanabe H.

Department of Experimental Oncology, Research Institute for Radiation Biology and Medicine, Hiroshima University, Hiroshima 734-8553, Japan.
A water-soluble extract from the culture medium of **Ganoderma lucidum (Rei-shi) mycelia (MAK)** has been shown to exert a potent chemopreventive effect. The present study was designed to investigate the effects of dietary MAK supplementation on the development of lung tumors initiated by N-nitrosobis (2-hydroxypropyl) amine (BHP) in male Slc:Wistar rats. A total of 77 animals, 6 weeks of age, were divided into 5 groups and given BHP (2,000 ppm) in their drinking water for 10 weeks. The normal controls were not supplied with BHP. After treatment with the carcinogen, the rats were fed a normal control MF solid diet, or the same diet containing MAK (1.25%, 2.5% or 5%) for 12 weeks. Macroscopically, all the doses of MAK reduced the number of nodules, and the effect of 5% MAK was found to be especially significant. Microscopically, an increase in the number of proliferating cell nuclear antigen (PCNA)-negative tumors and a decrease in the number of tumors strongly positive for PCNA were observed in the tissue sections from the rats that had received all the doses of MAK. **The present results thus indicate that dietary supplementation with MAK inhibits the development of lung tumors, suggesting that MAK may be a potent chemopreventive agent against lung carcinogenesis.** PMID: 17089035 [PubMed - indexed for MEDLINE]

Int J Oncol. 2006 Sep;29(3):695-703.

Ganoderma lucidum inhibits proliferation of human breast cancer cells by down-regulation of estrogen receptor and NF-kappaB signaling.Jiang J, Slivova V, Sliva D.

Cancer Research Laboratory, Methodist Research Institute, Indianapolis, IN 46202, USA.

Ganoderma lucidum, an oriental medical mushroom, has been used in Asia for the prevention and treatment of a

variety of diseases, including cancer. We have previously demonstrated that G. lucidum inhibits growth and induces cell cycle arrest at G0/G1 phase through the inhibition of Akt/NF-kappaB signaling in estrogen-independent human breast cancer cells. However, the molecular mechanism(s) responsible for the inhibitory effects of G. lucidum on the proliferation of estrogen-dependent (MCF-7) and estrogen-independent (MDA-MB-231) breast cancer cells remain to be elucidated. Here, we show that G. lucidum inhibited the proliferation of breast cancer MCF-7 and MDA-MB-231 cells by the modulation of the estrogen receptor (ER) and NF-kappaB signaling. Thus, G. lucidum down-regulated the expression of ERalpha in MCF-7 cells but did not effect the expression of ERbeta in MCF-7 and MDA-MB-231 cells.
In addition, G. lucidum inhibited estrogen-dependent as well as constitutive transactivation activity of ER through estrogen response element (ERE) in a reporter gene assay. G. lucidum decreased TNF-alpha-induced (MCF-7) as well as constitutive (MDA-MB-231) activity of NF-kappaB. The inhibition of ER and NF-kappaB pathways resulted in the down-regulation of expression of c-myc, finally suppressing proliferation of estrogen-dependent as well as estrogen-independent cancer cells. **Collectively, these results suggest that G. lucidum inhibits proliferation of human breast cancer cells and contain biologically active compounds with specificity against estrogen receptor and NF-kappaB signaling, and implicate G. lucidum as a suitable herb for chemoprevention and chemotherapy of breast cancer.** PMID: 16865287 [PubMed - indexed for MEDLINE]

Biosci Biotechnol Biochem. 2006 Sep;70(9):2028-34. Epub 2006 Sep 7.

Anti-tumor activities of the antlered form of Ganoderma lucidum in allogeneic and syngeneic tumor-bearing mice. Nonaka Y, Shibata H, Nakai M, Kurihara H, Ishibashi H, Kiso Y,Tanaka T, Yamaguchi H, Abe S.
Institute for Health Care Science, Suntory Ltd., Osaka, Japan. Yuji_Nonaka@suntory.co.jp

We investigated the anti-tumor effects of a dry powder preparation of the antlered form of Ganoderma lucidum (G. lucidum AF, rokkaku-reishi in Japanese), a variant type of G. lucidum, not only in allogeneic Sarcoma 180-bearing ddY mice, but also in syngeneic MM 46-bearing C3H/He mice. G. lucidum AF inhibited tumor growth and elongated the life span when orally administered to mice by free-feeding of a 2.5% G. lucidum AF-containing diet. It also showed anti-tumor activity in spite of post-feeding after tumor inoculation. G. lucidum AF significantly countered the depression of splenic CD8+ cells and protected the decrease in interferon-gamma (IFN-gamma) production in regional lymph nodes of MM 46-bearing mice, indicating that the anti-tumor activity of G. lucidum AF might be caused by its immunostimulating action. **These results suggest that the ingestion of G. lucidum AF can be useful for the prevention and curing of cancer.** PMID: 16960396 [PubMed - indexed for MEDLINE]

Nutr Cancer. 2004;49(2):209-16.

Ganoderma lucidum suppresses growth of breast cancer cells through the inhibition of Akt/NF-kappaB signaling. Jiang J, Slivova V, Harvey K, Valachovicova T, Sliva D.

Cancer Research Laboratory, Methodist Research Institute, Indianapolis, IN 46202, USA.

Ganoderma lucidum (Reishi, Lingzhi) is a popular Asian mushroom that has been used for more than 2 millennia for the general promotion of health and was therefore called the "Mushroom of Immortality." Ganoderma lucidum was also used in traditional Chinese medicine to prevent or treat a variety of diseases, including cancer. We previously demonstrated that Ganoderma lucidum suppresses the invasive behavior of breast cancer cells by inhibiting the transcription factor NF-kappaB. However, the molecular mechanisms responsible for the inhibitory effects of Ganoderma lucidum on the growth of highly invasive and metastatic breast cancer cells, has not been fully elucidated. Here, we show that Ganoderma lucidum inhibits proliferation of breast cancer MDA-MB-231 cells by downregulating Akt/NF-kappaB signaling. Ganoderma lucidum suppresses phosphorylation of Akt on Ser473 and downregulates the expression of Akt, which results in the inhibition of NF-kappaB activity in MDA-MB-231 cells. The biological effect of Ganoderma lucidum was demonstrated by cell cycle arrest at G0/G1, which was the result of the downregulation of expression of NF-kappaB-regulated cyclin D1, followed by the inhibition of cdk4**. Our results suggest that Ganoderma lucidum inhibits the growth of MDA-MB-231 breast cancer cells by modulating Akt/NF-kappaB signaling and could have potential therapeutic use for the treatment of breast cancer**. PMID: 15489214 [PubMed - indexed for MEDLINE]

Int J Oncol. **2004 May;24(5):1093-9.**

Ganoderma lucidum inhibits proliferation and induces apoptosis in human prostate cancer cells PC-3. Jiang J, Slivova V, Valachovicova T, Harvey K, Sliva D.

Cancer Research Laboratory, Methodist Research Institute, E504, Indianapolis, IN 46202, USA.

Ganoderma lucidum (Reishi), an oriental medical mushroom, has been widely used in Asian countries for centuries to prevent or treat different diseases, including cancer. However, the mechanism(s) responsible for the effects of Ganoderma lucidum on cancer cells remain to be elucidated. We have previously demonstrated that Ganoderma lucidum down-regulated the expression of NF-kappaB-regulated urokinase plasminogen activator (uPA) and uPA receptor (uPAR), which resulted in suppression of cell migration of highly invasive human breast and prostate cancer cells. In this study, we investigated the effects of Ganoderma lucidum on cell proliferation, cell cycle, and apoptosis in human prostate cancer cells PC-3. Our data demonstrate that Ganoderma lucidum inhibits cell proliferation in a dose- and time-dependent manner by the down-regulation of expression of cyclin B and Cdc2 and by the up-regulation of p21 expression. The inhibition of cell growth was also demonstrated by cell cycle arrest at G2/M phase. Furthermore, Ganoderma lucidum induced apoptosis of PC-3 cells with a slight decrease in the expression of NF-kappaB-regulated Bcl-2 and Bcl-xl. However, the expression of proapoptotic Bax protein was markedly up-regulated, resulting in the enhancement of the ratio of Bax/Bcl-2 and Bax/Bcl-xl. **Thus, Ganoderma lucidum exerts its effect on cancer cells by multiple mechanisms and may have potential therapeutic use for the prevention and treatment of cancer.** PMID: 15067330 [PubMed - indexed for MEDLINE]
Integr Cancer Ther. 2003 Dec;2(4):358-64.

Ganoderma lucidum (Reishi) in cancer treatment.Sliva D.

Cancer Research Laboratory, Methodist Research Institute, Indianapolis, IN 46202, USA. **d-silva@clarian.org**

The popular edible mushroom Ganoderma lucidum (Reishi) has been widely used for the general promotion of health and longevity in Asian countries. The dried powder of Ganoderma lucidum was popular as a cancer chemotherapy agent in ancient China. The authors recently demonstrated that Ganoderma lucidum inhibits constitutively active transcription factors nuclear factor kappa B (NF-kappaB) and AP-1, which resulted in the inhibition of expression of urokinase-type plasminogen activator (uPA) and its receptor uPAR. **Ganoderma lucidum also suppressed cell adhesion and cell migration of highly invasive breast and prostate cancer cells, suggesting its potency to reduce tumor invasiveness. Thus, Ganoderma lucidum clearly demonstrates anticancer activity in experiments with cancer cells and has possible therapeutic potential as a dietary supplement for an alternative therapy for breast and prostate cancer.** However, because of the availability of Ganoderma lucidum from different sources, it is advisable to test its biologic activity. PMID: 14713328 [PubMed - indexed for MEDLINE]-**Author's Note: This is the reason I use the Ganoderma Lucidum from Gano Excel. To learn more about Ganoderma Lucidum go to www.BuyHealthyCoffee.us.**

Teratog Carcinog Mutagen. 2003;Suppl 1:85-97.

Antiperoxidative, anti-inflammatory, and antimutagenic activities of ethanol extract of the mycelium of Ganoderma lucidum occurring in South India.-Lakshmi B, **Ajith TA**, **Sheena N**, **Gunapalan N**, **Janardhanan KK**.

Amala Cancer Research Centre, Thrissur, Kerala, India.

Free radical mediated genetic instability is widely thought to be a major etiological factor for initiation of carcinogenesis. Mushrooms represent a largely untapped source of powerful new pharmaceutical products. In the present study, we examined the antiperoxidative, anti-inflammatory, and antimutagenic activities of the ethanol extract of the mycelium of a medicinal mushroom, Ganoderma lucidum, occurring in south India. Antiperoxidative activity was evaluated using Fe(2+)-ascorbate-induced lipid peroxidation in rat liver homogenate and a phorbol ester (croton oil)-induced lipid peroxidation in mouse skin. Antiinflammatory activity was evaluated against carrageenan-induced acute and formalin-induced chronic inflammatory paw edema in mouse and phorbol ester-induced mouse skin

inflammation. Antimutagenic activity was determined by the Ames mutagenicity assay using histidine mutant of Salmonella typhimurium strains TA 98, TA100, and TA102. Sodium azide (NaN(3)), N-methyl-N-nitro-N-nitrosoguanidine (MNNG), 4-nitro-o-phenylenediamine (NPD), and benzo[a]pyrene (B[a]P) were used as the mutagens. The extract showed significant inhibition of Fe(2+)-induced peroxidation of lipid in rat liver (IC(50) 510 +/- 22 microg/ml) and 37% inhibition of croton oil-induced peroxidation on the mouse skin at 20 mg/0.1 ml/skin. Carrageenan-induced acute and formalin-induced chronic inflammatory edema were inhibited by 56 and 60%, respectively, by the extract at 1,000 mg/kg body wt (i.p). The extract at a concentration of 5 mg/plate showed inhibition of mutagenicity elicited by direct acting mutagens, NaN(3) (55.5 and 75.7%) and MNNG (50.0 and 57.5%) for S. typhymurium strains TA100 and TA102, respectively. The extract at the same concentration also inhibited mutagenicity elicited by NPD (52.4 and 64.2%) and B[a]P (60.7 and 59.6%) for TA98 and TA100 strains, respectively. The B[a]P was activated in the presence of rat liver microsomal (S9) fraction. **The results of our study revealed that ethanol extract of Ganoderma lucidum mycelium possessed significant anti-peroxidative, anti-inflammatory, and anti-mutagenic activities. The findings suggest a medicinal use for the ethanol extract of the mycelium of G. lucidum occurring in South India**. Copyright 2003 Wiley-Liss, Inc. PMID: 12616600 [PubMed - indexed for MEDLINE]

Biochem Biophys Res Commun. 2002 Nov 8;298(4):603-12.

Ganoderma lucidum suppresses motility of highly invasive breast and prostate cancer cells.-Sliva D, Labarrere C, Slivova V, Sedlak M, Lloyd FP Jr, Ho NW.

Cancer Research Laboratory, Methodist Research Institute, 1800 N Capitol Avenue E504, Indianapolis, IN 46202, USA. dsliva@clarian.org

A dried powder from basidiomycetous fungi, Ganoderma lucidum, has been used in East Asia in therapies for several different diseases, including cancer. However, the molecular mechanisms involved in the biological actions of Ganoderma are not well understood. We have recently demonstrated that phosphatidylinositol 3-kinase (PI 3-kinase) and nuclear factor-kappaB (NF-kappaB) regulate motility of highly invasive human breast cancer cells by the secretion of urokinase-type plasminogen activator (uPA). In this study, we investigated the effect of G. lucidum on highly invasive breast and prostate cancer cells. Here we show that spores or dried fruiting body of G. lucidum inhibit constitutively active transcription factors AP-1 and NF-kappaB in breast MDA-MB-231 and prostate PC-3 cancer cells. Furthermore, Ganoderma inhibition of expression of uPA and uPA receptor (uPAR), as well secretion of uPA, resulted in the suppression of the migration of MDA-MB-231 and PC-3 cells. **Our data suggest that spores and unpurified fruiting body of G. lucidum inhibit invasion of breast and prostate cancer cells by a common mechanism and could have potential therapeutic use for cancer treatment.** PMID: 12408995 [PubMed - indexed for MEDLINE]

Phytother Res. 2001 May;15(3):245-9.

Inhibition of lipid peroxidation and oxidative DNA damage by Ganoderma lucidum.-Lee JM, Kwon H, Jeong H, Lee JW, Lee SY, Baek SJ, Surh YJ.

College of Pharmacy, Seoul National University, Seoul 151-742, South Korea.

Reactive oxygen species (ROS), such as superoxide anions and hydroxyl radicals, are associated with carcinogenesis and other pathophysiological conditions. Therefore, elimination or inactivation of ROS or inhibition of their excess generation may be beneficial in terms of reducing the risk for cancer and other diseases. Ganoderma lucidum has been used in traditional oriental medicine and has potential antiinflammatory and antioxidant activities. In the present study, we tested the amino-polysaccharide fraction (designated as 'G009') from Ganoderma lucidum for the ability to protect against oxidative damage induced by ROS. G009 significantly inhibited iron-induced lipid peroxidation in rat brain homogenates and showed a dose-dependent inactivation of hydroxyl radicals and superoxide anions. It also reduced strand breakage in phiX174 supercoiled DNA caused by UV-induced photolysis of hydrogen peroxide and attenuated phorbol ester-induced generation of superoxide anions in differentiated human promyelocytic

leukaemia (HL-60) cells. **These findings suggest that G009 from Ganoderma lucidum possesses chemopreventive potential.** Copyright 2001 John Wiley & Sons, Ltd. PMID: 11351361 [PubMed - indexed for MEDLINE]

Transplantation. 1995 Sep 15;60(5):438-43.

Ling Zhi-8: studies of a new immunomodulating agent.-van der Hem LG, van der Vliet JA, Bocken CF, Kino K, Hoitsma AJ, Tax WJ.
Department of Surgery, University Hospital Nijmegen, The Netherlands.

Ling Zhi-8 (LZ-8) is a protein derived from the fungus Ganoderma lucidum and has immunomodulatory capacities. It was shown to be mitogenic toward mouse splenocytes in vitro and immunosuppressive in vivo by reducing antigen-induced antibody formation and by preventing completely the incidence of autoimmune diabetes in nonobese diabetic mice. In this study, the mitogenic effects of LZ-8 on human mononuclear cells are reported. In accordance to its mitogenic effect on mouse splenocytes, LZ-8 proved to be mitogenic for human PBMC. This mitogenic effect of LZ-8 apparently required the presence of monocytes. We also demonstrated it to be immunosuppressive in vitro in a human MLC performed in the absence of monocytes, using purified T cells and EBV-transformed allogeneic B cells. Furthermore, we tested LZ-8 for its possible suppressive effects in 2 different models of allogeneic tissue transplantation. **LZ-8 proved to have a significant effect on cellular immunity, since its administration in an allografted mouse skin model resulted in an increased survival time. In a model of transplanted allogeneic pancreatic rat islets, LZ-8 was effective in delaying the rejection process of allografted islets. More frequent or continuous administration resulted in a further prolongation of survival time. No serious side effects of LZ-8 could be discerned in these experiments.** PMID: 7676490 [PubMed - indexed for MEDLINE]

Whey Protein

I know this may sound contradictory, but I thought you should read the two articles that I found and the scientific information on Whey Protein. The whole purpose of this report is to provide you with the information necessary for you to make an educated decision regarding your health.

08.19.05 -- Another Use for Whey Protein: Helping Diabetics Control Their Blood Sugar

By Greg Arnold, DC, CSCS, July 28, 2005, abstracted from "Effect of whey on blood glucose and insulin responses to composite breakfast and lunch meals in type 2 diabetic subjects" in the July 2005 issue of the American Journal of Clinical Nutrition

As a pure, natural, high quality protein from cow's milk, whey protein is an excellent protein to help build muscle. But whey protein has a myriad of other health benefits, having been found to 1) decrease blood pressure, 2) help prevent cancer 3) improve bone health, 4) and strengthen your immune system. 5) Finally, whey protein is even used to help treat HIV infections.

Now a new study 5 has found another use for whey protein: helping diabetics control their blood sugar. Inability to control blood sugar is the hallmark of Type 2 diabetes and is the primary culprit in the health complications associated with Type 2 diabetes, including increased risks for heart disease, blindness, nerve and kidney damage.6 As a result, controlling blood sugar is vital to managing (and of course preventing) Type 2 diabetes.

In the study, 14 subjects with Type 2 diabetes were served a high-Glycemic Index breakfast (white bread) followed by a high-Glycemic Index lunch (mashed potatoes with meatballs). These meals were supplemented with whey protein on one day and lean ham and lactose on another day. The researchers took blood samples before and during four hours after breakfast and three hours after lunch.

The researchers found that whey protein increased the release of insulin, the hormone responsible for controlling blood sugar, by 31 percent after breakfast and 57 percent after lunch compared to the control. As a result, blood glucose levels were "significantly reduced" after whey ingestion, but only after lunch. Researchers reasoned that since insulin

resistance is higher in the morning after the overnight fast, this explained whey's inability to reduce blood sugar levels after breakfast.

Nevertheless, "the addition of whey to meals with rapidly digested and absorbed carbohydrates stimulates insulin release and reduces postprandial blood glucose" and provides Type 2 diabetics with another way to help control their blood sugar and manage their condition.

Greg Arnold is a Chiropractic Physician practicing in Danville, CA. You can contact Dr. Arnold directly by emailing him at ***mailto:ChiroDocPSUalum@msn.com*** *or visiting his website* ***www.CompleteChiropracticHealthcare.com***

Reference:

1 FitzGerald, R. J., B. A. Murray, et al. (2004). "Hypotensive peptides from milk proteins." *J Nutr 134(4): 980S-8S*
2 Eason, R. R., M. C. Velarde, et al. (2004). "Dietary exposure to whey proteins alters rat mammary gland proliferation, apoptosis, and gene expression during postnatal development." *J Nutr 134(12): 3370-7*
3 Takada, Y., S. Aoe, et al. (1996). "Whey protein stimulated the proliferation and differentiation of osteoblastic MC3T3-E1 cells." *Biochem Biophys Res Commun 223(2): 445-9*
4 Wang, H., X. Ye, et al. (2000). "First demonstration of an inhibitory activity of milk proteins against human immunodeficiency virus-1 reverse transcriptase and the effect of succinylation." *Life Sci 67(22): 2745-52*
5 Frid AH. Effect of whey on blood glucose and insulin responses to composite breakfast and lunch meals in type 2 diabetic subjects. *Am J Clin Nutr. 2005 Jul; 82(1):69-75*
6 "Type 2 Diabetes" from the American Diabetes Association website: http://www.diabetes.org/type-2-diabetes.jsp

American Journal of Clinical Nutrition, Vol. 82, No. 1, 69-75, July 2005

ORIGINAL RESEARCH COMMUNICATION

Effect of whey on blood glucose and insulin responses to composite breakfast and lunch meals in type 2 diabetic subjects1,2,3 -Anders H Frid, Mikael Nilsson, Jens Juul Holst and Inger ME Björck

1 From the Clinic of Endocrinology, University Hospital MAS, Malmö, Sweden (AHF); the Department of Applied Nutrition and Food Chemistry, Lund University, Lund, Sweden (MN and IMEB); and the Department of Medical Physiology, The Panum Institute, University of Copenhagen, Copenhagen, Denmark (JJH)

Background: Whey proteins have insulinotropic effects and reduce the postprandial glycemia in healthy subjects. The mechanism is not known, but insulinogenic amino acids and the incretin hormones seem to be involved.

Objective: The aim was to evaluate whether supplementation of meals with a high glycemic index (GI) with whey proteins may increase insulin secretion and improve blood glucose control in type 2 diabetic subjects.

Design: Fourteen diet-treated subjects with type 2 diabetes were served a high-GI breakfast (white bread) and subsequent high-GI lunch (mashed potatoes with meatballs). The breakfast and lunch meals were supplemented with whey on one day; whey was exchanged for lean ham and lactose on another day. Venous blood samples were drawn before and during 4 h after breakfast and 3 h after lunch for the measurement of blood glucose, serum insulin, glucose-dependent insulinotropic polypeptide (GIP), and glucagon-like peptide 1 (GLP-1).

Results: The insulin responses were higher after both breakfast (31%) and lunch (57%) when whey was included in the meal than when whey was not included. After lunch, the blood glucose response was significantly reduced [–21%; 120 min area under the curve (AUC)] after whey ingestion. Postprandial GIP responses were higher after whey ingestion, whereas no differences were found in GLP-1 between the reference and test meals.

Conclusions: It can be concluded that the addition of whey to meals with rapidly digested and absorbed

carbohydrates stimulates insulin release and reduces postprandial blood glucose excursion after a lunch meal consisting of mashed potatoes and meatballs in type 2 diabetic subjects.

Key Words: Milk • whey • type 2 diabetes • blood glucose • serum insulin • incretin hormones

I found the following article on **www.LearningPlaceOnline.com**. When you come to the end of this article you will read that Ms. Place does agree **that milk has many substances that we should not be consuming**.

Supplement Savvy for Cancer: Whey Protein
By Jill Place, MA, RD

What It Is . . .

Remember Miss Muffet and her curds and whey? Whey is protein left over after cheese is made from cow's milk. This leftover whey protein can then be refined to a high quality with cross-flow microfiltration, ion exchange, and other processes that concentrate the protein and the immunoglobulins in it without inactivating them. The best way to keep whey protein stable and active without using preservatives is to make it in powder form. So, most supplements come in powders or capsules.

What It Does . . .

Whey protein has the highest biological value of any protein, so it's the protein best absorbed by your body. **Whey protein may be able to:**

- **show your immune system where unfriendly bacteria, yeasts, molds, and toxins are in your gut**, also known as Bad Gut Guys, **so that your immune system can destroy them** .
- bind iron with the **whey protein Lactoferrin**, which helps to **starve out the Bad Gut Guys**. Lactoferrin may also **keep the Bad Gut Guys from invading your intestinal walls**. Lactoferrin and lipoic acid may be a better bet to prevent anemia than iron in some cases.
- **damage the Bad Gut Guys** by Lactoperoxidase, a whey protein enzyme.
- **keep the Bad Gut Guys from sticking to intestinal walls** with its Globulin Proteins so that they're unable get a footfold to grow.
- upping Glutathione levels and therefore upping immune response. Glutathione is a protein that's important for maintaining cell and immune health.
- **increase immunity and intestinal health** with Immunoglobulins.
- **prevent diarrhea and other digestive problems**.

What To Do . . .

Since this type of protein comes in powdered form, it's perfect for shake recipes. Shakes are great for a quick, nutritious meal when you're on the go. **Shakes are also great if you just can't eat during chemotherapy or radiation and are losing weight.** Just drink a couple of these every day:

Blend in blender:

2 to 3 ounces fresh tofu or 1 cup soy, almond, or grain milk and/or 1 serving whey protein powder with any or all of the following:

- 4 to 8 ounces fresh juice (if you have mouth sores you may want to stick to low-acid types)

- 1 cup yogurt, frozen yogurt, or soy ice cream
- -1 cup frozen fruit (any type)
- 1 frozen banana (skin; freeze in zip lock bag)
- 1 large carrot or carrot or other vegetable juice
- 1 Tablespoon flax seeds or flax seed meal
- 1 Tablespoon wheat germ
- 1 Tablespoon wheat or rice bran
- 1 to 2 teaspoons flavor extracts, like almond, coconut, banana, and/or vanilla
- 2 to 4 drops Stevia, a natural non-caloric sweetener sold as a nutritional supplement
- 4 to 6 ice cubes, added one at a time to make a slushy

What To Watch Out For . . .

Because most milk has hormones and other undesirable substances, it's important that the milk used to produce this supplement is free from disease, hormones, immunizations, and contamination. Those who are sensitive to milk proteins and products may not be able to tolerate whey protein. The way it's made, however, may make it safe to use for those who are lactose intolerant. If you're lactose intolerant and interested in taking whey protein, proceed with caution with small amounts and build up to a full dose gradually over a few weeks. Discontinue use if you have bloating, abdominal pain, diarrhea, or other symptoms.

I included this article, because after learning more about whey protein I thought you would like to know the benefits and disadvantages of dairy products. It seems that the way whey is processed; the **cold processed ion exchange** form may be helpful. I found a company that has a cold processed ion exchange form of the whey that has a very low amount of lactose, which is very pure and bio-available. According to NutriHarmony their Ion-Exchange Whey Protein has less than 1% lactose and is approved for lactose intolerant individuals. NutriHarmony uses a proprietary process that uses magnets and energy to "clean" the whey and a special gravity fed micro filtration system and cold air-drying process that allows their whey to retain the maximum BCAA's. Their web site is **www.NutriHarmony.com/healthy1**.

Curr Pharm Des. 2007;13(8):813-28.

A Role for Milk Proteins and their Peptides in Cancer Prevention.
Parodi PW.-Dairy Australia, Human Nutrition and Health Research, Melbourne, Australia. **peterparodi@uq.net.au.**

A role for the amount and type of dietary protein in the etiology of cancer has not been studied extensively. **Nevertheless, there is no compelling evidence from epidemiological studies to indicate that protein, at levels usually consumed, is a risk factor for cancer**. **On the other hand, animal studies suggest that certain peptides and amino acids derived from dietary proteins may influence carcinogenesis.** The predominant protein in milk, casein, its peptides, but not liberated amino acids, have antimutagenic properties. Animal models, usually for colon and mammary tumorigenesis, nearly always show that whey protein is superior to other dietary proteins for suppression of tumour development. This benefit is attributed to its high content of cystine/cysteine and gamma-glutamylcyst(e)ine dipeptides, which are efficient substrates for the synthesis of glutathione. Glutathione is an ubiquitous cellular antioxidant that directly or through its associated enzymes destroys reactive oxygen species, detoxifies carcinogens, maintains proteins in a reduced state and ensures a competent immune system. Various experiments showed that tumour prevention by dietary whey protein was accompanied by increased glutathione levels in serum and tissues as well as enhanced splenic lymphocyte proliferation, phagocytosis and natural killer, T helper and cytotoxic T cell activity. **Whey protein components, beta-lactoglobulin, alpha-lactalbumin, and serum albumin were studied infrequently, but results suggest they have anticancer potential.** The minor component lactoferrin has received the most attention; it inhibits intestinal tumours and perhaps tumours at other sites. Lactoferrin acts by induction of apoptosis, inhibition of angiogenesis, modulation of carcinogen metabolising enzymes and perhaps acting as an iron scavenger. Supplementing cows with selenium increases the content of selenoproteins in milk, which on isolation inhibited colon tumorigenesis in rats.-PMID: 17430183 [PubMed - in process]

Anticancer Res. 2003 Mar-Apr;23(2B):1411-5.

The antioxidant system.-Bounous G, Molson JH.

Research and Development Department, Immunotec Research Ltd., 292 Adrien Patenaude, Vaudreuil-Dorion, Québec, Canada J7V 5V5.

The glutathione (GSH) antioxidant system is the principal protective mechanism of the cell and is a crucial factor in the development of the immune response by the immune cells. Experimental data demonstrate that a cysteine-rich whey protein concentrate represents an effective cysteine delivery system for GSH replenishment during the immune response. Animal experiments showed that the concentrates of whey protein also exhibit anticancer activity. They do this via the GSH pathway, the induction of p53 protein in transformed cells and inhibition of neoangiogenesis.-PMID: 12820403 [PubMed - indexed for MEDLINE]

J Nutr. 2001 Dec;131(12):3281-7.

Soy and whey proteins downregulate DMBA-induced liver and mammary gland CYP1 expression in female rats.-Rowlands JC, He L, Hakkak R, Ronis MJ, Badger TM.
Arkansas Children's Nutrition Center and Department of Pediatrics, University of Arkansas for Medical Sciences, Little Rock, AR 72202, USA. **jcrowlands@salvitas.com**

One possible mechanism by which diet may reduce cancer risk is through enhancement of metabolic systems that prevent activation of carcinogens or accelerate carcinogen inactivation. We studied the effects of diet and 7,12-dimethylbenz-(a)anthracene (DMBA) on hepatic and mammary gland CYP1A1, CYP1A2 and CYP1B1 enzymes in female Sprague-Dawley rats. Diets (AIN-93G) were fed from conception to adulthood, and DMBA was given by oral gavage at age 48-50 d. The protein sources of diets were casein (CAS), soy protein isolate (SPI) or whey protein hydrolysate (WPH). The DMBA-induced hepatic ethoxyresorufin-O-deethylase and methoxyresorufin-O-demethylase activities and CYP1A1 protein and mRNA expression were lower ($P < 0.05$) in SPI-fed rats compared with those fed casein. Differences in mammary gland CYP1 expression were also observed with decreased DMBA induction ($P < 0.05$) of all three CYP1 proteins and mRNAs in rats fed either SPI or WPH compared with those fed CAS. Most notable were the decreased constitutive and DMBA-induced mammary gland expression of CYP1B1 protein of 93 and 96%, respectively, in the SPI-fed rats relative to the CAS-fed controls. The diet-induced changes in CYP1 enzyme expression were consistent with changes in the AhR and ARNT transcription factors that regulate them. Decreased ($P < 0.05$) mammary constitutive AhR and ARNT proteins were measured in SPI-fed rats. There was also a 100% increase in constitutive AhR protein in the WPH-fed rats that paralleled a 100% increase in constitutive CYP1B1 protein in the mammary gland. **These results demonstrate the importance of diet in regulation of phase I metabolism in liver and mammary gland, and suggest a potential mechanism by which soy or whey proteins reduce DMBA-induced mammary tumor incidence.-**PMID: 11739881 [PubMed - indexed for MEDLINE]

Anticancer Res. 2000 Nov-Dec;20(6C):4785-92.

Whey protein concentrate (WPC) and glutathione modulation in cancer treatment.
Bounous G.-Research & Development Department, Immunotec Research Ltd., 292 Adrien-Patenaude, Vaudreuil-Dorion, Quebec, Canada, J7V 5V5.

The glutathione (GSH) antioxidant system is foremost among the cellular protective mechanisms. Depletion of this small molecule is a common consequence of increased formation of reactive oxygen species during increased cellular activities. This phenomenon can occur in the lymphocytes during the development of the immune response and in the muscular cells during strenuous exercise. It is not surprising that so much research has been done, and is still being done on this small tripeptide molecule**. Whey protein concentrate has been shown to represent an effective and safe cysteine donor for GSH replenishment during GSH depletion in immune deficiency states.** Cysteine is the crucial limiting amino acid for intracellular GSH synthesis. **Animal experiments showed that the concentrates of whey proteins also exhibit anti-carcinogenesis and anticancer activity.** They do this via their effect on increasing GSH concentration in relevant tissues, and may have anti-tumor effect on low volume of tumor via stimulation of

immunity through the GSH pathway. It is considered that oxygen radical generation is frequently a critical step in carcinogenesis, hence the effect of GSH on free radicals as well as carcinogen detoxification, could be important in inhibiting carcinogenesis induced by a number of different mechanisms. **Case reports are presented which strongly suggest an anti-tumor effect of a whey protein dietary supplement in some urogenital cancers.** This non toxic dietary intervention, which is not based on the principles of current cancer chemotherapy, will hopefully attract the attention of laboratory and clinical oncologists. -PMID: 11205219 [PubMed - indexed for MEDLINE

In my research I found some people who were very happy with a product called Essiac so I decided to find out more about it. For more information on Essiac you can also go to **www.essiacinfo.org/**

I found the following information while searching the net at **www.cancer-info.com/essiac.htm :**

Essiac: (ess-ee-ack):

Essiac is the most popular and favorite alternative medicine for cancer of all alternative remedies for cancer. If you are looking for an alternative medicine to compliment your cancer care, essiac would be a good first choice.

Essiac has been used for over 60 years to remedy the side effects of cancer treatments and to remedy cancer itself. Essiac is a time proven safe remedy for cancer.

History of Essiac and Rene Caisse. Canada's Cancer Nurse.

Rene Caisse spent her whole adult life treating cancer patients along with her life long friend, Mary McPherson, with this herb tea in her own clinic, until she died in 1978 at the age of 91. Many of the people she treated for cancer reported they were miraculously cured by taking it, while others claimed the tea relieved the pain and agony of cancer and made their lives living with cancer much more bearable.

When Rene presented her Essiac and its effectiveness to the medical society, some doctors were so impressed by the results that they petitioned the Canadian Government in 1938 to pass a Bill to "authorize Rene Caisse to practice medicine in the Province of Ontario in the treatment of Cancer and conditions resulting there from".

The Bill failed to pass by only 3 votes. Soon after, a Legislative Assembly passed "An Act For The Investigation Of Remedies For Cancer", by which Rene would have to reveal her formula. Rather than do this, Rene closed her clinic, later opening it again at the behest of the Minister of Health. Thereafter, she was allowed to treat patients certified as terminal by their physicians.

Rene Caisse kept the formula a secret all those years, fearing it would be exploited. Finally, 14 months before she died, she signed the properties (formula, trademark name Essiac®, notes, etc.) over to a Canadian company named Resperin, with hopes that it would clinically validated (which Resperin failed to do) and made available to all people.

Resperin failed miserably with the manufacturing of their "ORIGINAL RECIPE" Essiac. The ESSIAC manufactured by RESPERIN was of such poor quality that users felt that RESPERIN where not using the correct formula.

To date, RESPERIN no longer is the manufacturer of ESSIAC.

Essiac Information: Essiac has become a generic name for a herbal tea that is today's most popular alternative remedy for cancer. Essiac was originally an herbal tea attributed to Canadian nurse Rene Caisse (reen-case) of Bracebridge Ontario, Canada, who claimed that the formula came from a Native Ojibwa medicine man. She named it after the backward spelling of her own last name, Caisse.

Many users of essiac believe that essiac can and does improve the body's ability to fight cancer and that essiac is effective at reducing the side effects of chemotherapy and radiation treatments. User have reported that with the reduction in chemotherapy/radiation side effects, they are much better able to handle the full course of their treatments without interruption and delays in treatment.

Fresh is best.

The Ingredients of Essiac: The herbs in essiac are safe to use and are in some instances used individually as culinary herbs in food preparation and as additions to garden salads. Burdock root, an important ingredient in essiac was actually used as a prepared candy by the Ojibwa Natives of North America. Boiled in maple syrup, burdock root was eaten through the winter as a nutritious snack and candy.

J Altern Complement Med. 2004 Aug; 10(4):687-91.-Inhibition of prostate cancer-cell proliferation by Essiac.

Ottenweller J, Putt K, Blumenthal EJ, Dhawale S, Dhawale SW.-Department of Biology, Indiana University-Purdue University Fort Wayne, Fort Wayne, IN, USA.

OBJECTIVE: To assess the ability of Essiac tea extracts (Essiac Canada International, Ottawa, Canada) to modulate cancer cell proliferation and immune responsiveness. DESIGN: A noncancerous transformed cell line was compared to a cancerous cell line and spleen cells that had been isolated from mice to examine proliferation responses mediated by the addition of an Essiac preparation. RESULTS: We found in vitro evidence of decreased proliferation of both noncancerous transformed (CHO) and cancerous prostate cell line (LNCaP) when Essiac was present in the culture media. A dose response for inhibition was demonstrated by a linear regression performed on the data for both the CHO and LNCaP cells. The percent inhibition of the LNCaP cells was higher than the percent inhibition of the CHO cells suggesting that Essiac may have a more selective effect on cancer cells than transformed cells. In addition, the effects of Essiac were examined in an immune T-lymphocyte proliferation assay. At low doses of Essiac, augmentation of proliferation of these T cells was demonstrated, but at higher doses Essiac was inhibitory to T-cell proliferation. The same doses of Essiac that stimulated spleen cells were inhibitory for LNCaP cell proliferation. **CONCLUSIONS: Essiac preparations may be able to inhibit tumor cell growth while enhancing immune response to antigenic stimulation. This may be especially valuable in immune-suppressed individuals.** PMID: 15353028 [PubMed - in process]

Essiac can be found in your local health food store.

The following information came from The National Cancer Institute web site http://www.nci.nih.gov/cancertopics/pdq/cam/essiac

Overview

This complementary and alternative medicine (CAM) information summary provides an overview of the use of Essiac and Flor•Essence, which are proprietary herbal tea mixtures, as treatments for patients with cancer. The summary includes a brief history of the development of Essiac and Flor•Essence; a review of laboratory, animal, and human studies; and possible side effects associated with Essiac and Flor•Essence use.

This summary contains the following key information:

- Essiac and Flor•Essence are herbal tea mixtures originally developed in Canada.
- These products are marketed worldwide as dietary supplements.
- Proponents have claimed that Essiac and Flor•Essence can help detoxify the body and strengthen the immune system.
- Proponents of Essiac have claimed further that it can help relieve pain, improve quality of life, and reduce tumor size.
- Molecules with antioxidant, anti-inflammatory, anticancer, or immunostimulatory activity have been identified in the individual herbs in the Essiac and Flor•Essence formulas.
- No data are available from animal or human studies to suggest that Essiac or Flor•Essence can be effective in the treatment of patients with cancer.

Essiac and Flor•Essence are proprietary herbal tea mixtures produced by different manufacturers. Essiac is reported to contain 4 herbs: burdock root *(Arctium lappa),* Indian rhubarb root *(Rheum palmatum,* sometimes known as Turkish rhubarb), sheep sorrel *(Rumex acetosella),* and the inner bark of slippery elm *(Ulmus fulva* or *Ulmus rubra).*[1] Reviewed in [2-10] Flor•Essence is reported to contain the same 4 herbs as Essiac, plus 4 "potentiating" herbs: watercress *(Nasturtium officinale),* blessed thistle *(Cnicus benedictus),* red clover *(Trifolium pratense),* and kelp *(Laminaria digitata).*[11] Reviewed in [2-4,7]

The manufacturers of Essiac and Flor•Essence both claim they market the original herbal mixture promoted by the developer.[1,11] Although only 1 company manufactures Flor•Essence,[11] several companies produce and market Essiac-like products. Reviewed in [2,3,10] This summary contains information about the trademarked mixtures only and differentiates between the 2 products wherever possible.

Essiac and Flor•Essence are said to detoxify the body and strengthen the immune system.[1,11] Reviewed in [4,6,7,9] Proponents of Essiac claim further that it helps relieve pain, improves overall quality of life, may reduce tumor size, and may prolong the survival of patients with various types of cancer. Reviewed in [4,7,9] The individual herbs in the Essiac and Flor•Essence formulas have been shown to contain molecules that have anticancer, anti-inflammatory, antioxidant, or immunostimulatory activity (see Laboratory/Animal/Preclinical Studies). Reviewed in [2-4,9,12-15] It is said that the benefits of Essiac and Flor•Essence are dependent on the presence of the constituent herbs in the correct proportions. Reviewed in [2-4,9] A mixture of the Essiac herbs has shown a decreased proliferation of a prostate cancer cell line.[16]

Although the use of Essiac and Flor•Essence is generally associated with cancer, both products have been used to treat other health conditions. Essiac has reportedly been used to control diabetes and to treat acquired immunodeficiency syndrome (AIDS). Reviewed in [6] Flor•Essence has reportedly been studied in Russia as a treatment for chronic gastrointestinal diseases (i.e., esophagitis, gastritis, duodenitis, and colitis) and as a treatment for cirrhosis of the liver. Reviewed in [2] No data have been published in the peer-reviewed, scientific literature, however, to show the safety or the efficacy of Essiac or Flor•Essence in patients with cancer or these other health conditions (see also Human/Clinical Studies).

Essiac and Flor•Essence are sold worldwide as health tonics or herbal dietary supplements.[1,11] Reviewed in [2-4,10] In the United States, health tonics and dietary supplements are regulated as foods, not drugs. Therefore, premarket evaluation and approval by the Food and Drug Administration (FDA) are not required and specific disease treatment or prevention claims are not allowed. Because health tonics and dietary supplements are not formally inspected for manufacturing consistency, there may be considerable variation from lot to lot, and there is no guarantee that ingredients identified on product labels are present at all or are present in the specified amounts. It is important to note that the FDA has not approved the use of either Essiac or Flor•Essence for the treatment of patients with cancer or any other medical condition.

To conduct clinical drug research in the United States, researchers must file an Investigational New Drug (IND) application with the FDA. An IND application must also be made for clinical evaluation of dietary supplements as agents for the treatment or prevention of disease. The FDA's IND process is confidential, and the existence of an IND application can be disclosed only by the applicants. To date, no investigator has announced filing an IND application to study either Essiac or Flor•Essence in the treatment of patients with cancer.

Essiac and Flor•Essence are administered orally in the form of herbal teas.[1,11] Reviewed in [4,6,8,9,17] Originally, an extract of one of the herbs (not specified) was given to cancer patients by intramuscular injection at or near tumor sites, and the other herbs were given orally as a tea. Reviewed in [4,8,9,17]

Only minimal information about dose and schedule of administration is freely available from the manufacturer of Essiac.[1] According to the manufacturer, the dose will vary, depending on the reason for ingestion; the manufacturer's recommended schedules of administration assume a 12-week program of uninterrupted use.[1] Although Essiac is said to be safe for pets, no information is given about its safety in children.[1]

The manufacturer of Flor•Essence states that adults may consume from 30 to 360 mL (i.e., 1-12 fl oz) of Flor•Essence tea a day, depending on individual requirements, and that it may be used on an ongoing basis.[11] The manufacturer also suggests that Flor•Essence may be safely consumed by infants and children, but its use by pregnant women and nursing mothers is not recommended.[11]

The manufacturers of Essiac and Flor•Essence both state these products can be used in conjunction with other cancer treatments.[1,11] Nonetheless, some proponents of Essiac have recommended that no additional anticancer therapy (such as chemotherapy or radiation therapy) be undertaken while patients are being treated with the mixture. Reviewed in [8] The purported rationale for this recommendation is that conventional anticancer treatments may alter immune system function and prevent Essiac from working effectively. Reviewed in [8] as indicated previously,

however, no evidence has been reported in the peer-reviewed, scientific literature to show that Essiac is an effective treatment for patients with cancer.

References

1. Essiac. Kirkland, Canada: Altramed Health Products, 2002. Available online. Last accessed January 10, 2005.
2. Tamayo C, Richardson MA, Diamond S, et al.: The chemistry and biological activity of herbs used in Flor-Essence herbal tonic and Essiac. Phytother Res 14 (1): 1-14, 2000. [PUBMED Abstract]
3. Tamayo C: Essiac for cancer. Alternative Therapies in Women's Health 2 (3): 19-23, 2000.
4. Kaegi E: Unconventional therapies for cancer: 1. Essiac. The Task Force on Alternative Therapies of the Canadian Breast Cancer Research Initiative. CMAJ 158 (7): 897-902, 1998. [PUBMED Abstract]
5. Ernst E, Cassileth BR: How useful are unconventional cancer treatments? Eur J Cancer 35 (11): 1608-13, 1999. [PUBMED Abstract]
6. Locock RA: Essiac. Can Pharm J 130: 18-9, 1997.
7. Reviews of Therapies: Herbal/plant therapies: Essiac. Houston, Tex: M.D. Anderson Cancer Center, 2002. Available online. Last accessed January 10, 2005.
8. Herbal treatments. In: US Congress, Office of Technology Assessment.: Unconventional Cancer Treatments. Washington, DC: U.S. Government Printing Office, 1990. OTA-H-405, pp 71-5. Also available online. Last accessed January 10, 2005.
9. Essiac. Toronto, Canada: Canadian Breast Cancer Research Alliance, 1996. Available online. Last accessed January 10, 2005.
10. The History of Essiac & Rene Caisse, Canada's Cancer Nurse. Kirkland, Canada: Altramed Health Products, 2001. Available online. Last accessed January 10, 2005.
11. Flora Flor•Essence®. Burnaby, Canada: Flora Manufacturing & Distributing Ltd. Available online. Last accessed January 10, 2005.
12. Franke AA, Cooney RV, Custer LJ, et al.: Inhibition of neoplastic transformation and bioavailability of dietary flavonoid agents. In: Manthey JA, Buslig BS, eds.: Flavonoids in the Living System. New York, NY: Plenum Press, 1998. Advances in Experimental Medicine and Biology, 439, pp 237-48.
13. Waladkhani AR, Clemens MR: Effect of dietary phytochemicals on cancer development (review) Int J Mol Med 1 (4): 747-53, 1998. [PUBMED Abstract]
14. de Witte P: Metabolism and pharmacokinetics of anthranoids. Pharmacology 47 (Suppl 1): 86-97, 1993. [PUBMED Abstract]
15. Campbell MJ, Hamilton B, Shoemaker M, et al.: Antiproliferative activity of Chinese medicinal herbs on breast cancer cells in vitro. Anticancer Res 22 (6C): 3843-52, 2002 Nov-Dec. [PUBMED Abstract]
16. Ottenweller J, Putt K, Blumenthal EJ, et al.: Inhibition of prostate cancer-cell proliferation by Essiac. J Altern Complement Med 10 (4): 687-91, 2004. [PUBMED Abstract]
17. LeMoine L: Essiac: an historical perspective. Can Oncol Nurs J 7 (4): 216-21, 1997. [PUBMED Abstract]

While I searched for Mangosteen and Cancer I came across the following web site **http://www.cancertutor.com/.** I didn't gat a chance to explore the entire site, but from what I saw it was very informative for anyone who was interested in dealing with cancer by using alternative methods. Actually, anyone who is currently under a medical doctor's care and receiving treatment should also review alternative methods as an adjunct to their current program.

Sodium

Blood Purif. 2005;23(5):379-83. Epub 2005 May 27.

Effect of oral sodium bicarbonate supplementation on interdialytic weight gain, plasma sodium concentrations and predialysis blood pressure in hemodialysis patients.

Movilli E, Gaggia P, Camerini C, Brunori G, Vizzardi V, Cancarini G.

Chair and Division of Nephrology, Spedali Civili and University of Brescia, Italy. eziomov@libero.it

BACKGROUND: Correction of metabolic acidosis in dialysis patients should be considered of paramount importance. However, consuming sodium bicarbonate tablets during the interdialytic interval to reach predialysis bicarbonate levels of 23--24 mmol/l is not widespread due to the fear of greater interdialytic weight gain and fluid overload. For this reason we investigated in a cross-sectional and in an interventional study the effect of oral sodium bicarbonate supplementation on body weight gain, plasma sodium concentrations and predialysis blood pressure in a

group of stable uremic patients on regular hemodialysis (HD) treatment. Study DESIGN: 110 patients (67 men, 43 women), mean age 67+/-15 (range 22--89) years, on regular chronic HD treatment for 6--372 (median 48) months were studied. 70 patients were on regular oral bicarbonate supplementation for at least 4 weeks (group A), 40 patients were not on oral bicarbonate supplementation (group B). The following parameters were recorded: dry body weight (DBW), interdialytic weight gain (IWG), body mass index (BMI), plasma sodium (Na), serum pH, serum bicarbonate (sBic), K(t)/V, normalized protein catabolic rate (PCRn), predialysis systolic (SBP) and diastolic (DBP) blood pressure, and bicarbonate therapy (g/day). 18 patients not on oral bicarbonate supplementation with sBic levels</=20 mmol/l were started on oral bicarbonate therapy and were prospectively followed in the context of an interventional study of correction of chronic metabolic acidosis. The same parameters were recorded before (pre) and after (post) 4 months of oral bicarbonate supplementation. RESULTS:Serum pH and sBic concentrations were significantly higher in patients in group A compared to patients in group B (pH 7.37 +/- 0.02 group A vs. 7.33+/- 0.02 group B: p<0.001: sBic 23.8+/-1.4 group A vs. 20.9+/-1.4 group B: p<0.0001). Age, DBW, BMI, IWG, SBP, DBP, Na, K(t)/V and PCRn did not differ between groups. The mean daily dose of oral sodium bicarbonate administered to patients in group A was 1.9+/-0.9 (range 1--5, median 2) g/day. Also in the 18 patients who started bicarbonate treatment, a significant increase in serum pH and sBic concentrations and a significant reduction in PCRn were observed. No significant change in DBW, IWG, SBP, DBP and Na concentrations after 4 months of treatment was found. **CONCLUSIONS: Our data show that in stable uremic patients on regular HD treatment, oral daily administration of sodium bicarbonate is effective in correcting mild-moderate chronic metabolic acidosis, and does not cause increased interdialytic body weight gain, different plasma sodium concentrations and different systolic-diastolic blood pressure levels compared to patients not on oral sodium bicarbonate supplementation.**

PMID: 16088106 [PubMed - indexed for MEDLINE]

This information came from Wikipedia the online encyclopedia.

Medical uses

Sodium bicarbonate is used as an antacid to treat acid indigestion and heartburn [6] An aqueous solution is administered intravenously for cases of acidosis, or when there is insufficient sodium or bicarbonate ions in the blood. [7] This compound has also been used as for patients who have had a ureterosigmoidostomy.[citation needed] Dr. T. Simoncini, an oncologist in Rome, Italy, has controversially used sodium bicarbonate in the treatment for various types of cancer [8].

Adverse reactions to emergency administration include congestive heart failure, with edema secondary to sodium overload, and the metabolic complication of hyperosmolarity, metabolic alkalosis, and hypernatremia.[citation needed]

Sodium bicarbonate may also be used as an anti-fungal for dandruff caused by fungus.[9]

1 Company History". Church & Dwight Co..
6 "Sodium Bicarbonate". Jackson Siegelbaum Gastroenterology (1998).
7 "Sodium Bicarbonate Intravenous Infusion". Consumer Medicine Information. Better Health Channel (2004-07-13).
8 [1]
9 Use of Baking Soda as a Fungicide, By George Kuepper, Raeven Thomas, and Richard Earles, © NCAT November 2001

(Sodium and Hypertension)

Department of Chemical Pathology, Charing Cross & Westminster Medical School, London.

de Wardener HE.

Arch Mal Coeur Vaiss. 1996 Sep;89 Spec No 4:9-15

Over several million years the human race was programmed to eat a diet which contained about 15 mmol of sodium (1 g of **sodium chloride**) per day. It is only five to ten thousand years ago that we became addicted to salt. Today we eat about 150 mmol of sodium (9-12 g of salt) per day. It is now apparent that this sudden rise in sodium intake (in evolutionary terms) is the most likely cause for the rise in blood pressure with age that occurs in the majority of the world's population. Those which consume less than 60 mmol/day do not develop hypertension. The reason for the rise in sodium intake is not known but it is probable that an important stimulus was the discovery that meat could be preserved by immersion into a concentrated salt solution. This seemingly miraculous power endowed salt with such magical and medicinal qualities that it became a symbol of goodness and health. It was not until 1904 Ambard and Beaujard suggested that on the contrary dietary salt could be harmful and raise the blood pressure. At first the idea did not prosper and it continues to be opposed by a diminishing band. The accumulated evidence that sodium intake is related to the blood pressure in normal man and animals and in inherited forms of hypertension has been obtained from experimental manipulations and studies of human populations. The following observation links sodium and hypertension. An increase in sodium (chloride) intakes raises the blood pressure of the normal rat, dog, rabbit, baboon, chimpanzee and man. Population studies have demonstrated a significant correlation between sodium intake and the customary rise in blood pressure with age. The development of hypertensive strains of rats has revealed that the primary genetic lesion which gives rise to hypertension resides in the kidney where it impairs the urinary excretion of sodium. There is similar but less convincing evidence in essential hypertension. The kidney in both essential hypertension and hypertensive strains of rats share a number of functional abnormalities most of which are capable of impairing sodium excretion. Essential hypertension would appear to be as much a renal disturbance related to the intake of sodium as hypertension secondary to renal disease.

PMID: 8952809 [PubMed - indexed for MEDLINE]

To paraphrase what I had said earlier in this report, **any information obtained from this report is purely for educational and informational purposes only and should be discussed with your physician**.

If you have access or know someone with access to the Internet, explore and learn as much as you can. Put your life back in **your hands** and give **your body** the proper material to rebuild healthy cells with. **Your body** has the capability. So,

DO IT NOW.

Remember

Your Life Is In Your Hands

Learn As Much As You Can

and as the saying goes,

"Knowledge Is Power",

but

Knowledge Can Also Be Powerless,

Unless You Put That Knowledge Into Action.

As long as you and your doctor agree, a complementary/integrative medicine program can be coordinated to help with any treatment program that you are currently on.

If you or your doctor have any questions or would like to learn more about any of the topics mentioned in this report you can call **(516) 409-6978**, send me an e-mail at **healthcoach9@gmail.com,** or go to my web site **http://www.abcsofhealth.com/.** For those of you who may be interested, I do provide one on one and group nutrition/wellness programs and seminars and lymphatic exercise classes.

If you need help in deciding which products would be best for you, you can contact me and I will be glad to help you decide.

Important Note to Remember:

With Any New Diet Change Or Lifestyle Program You Decide To Follow, You Must Start Slowly. The Sicker You Are The Slower You Should Start, Because The Faster You Put Into Your Body Good Material, The Faster Your Body Gets Rid Of The Bad And If You Do This Too Quickly, You Actually May Feel Worse Than You Currently Do. Some Call This A Healing Crisis. If You Are Currently On Any Medication And/Or Chemotherapy, It Is Even More Important That You Start Slowly, Because The More Efficient Your Body Becomes The More Toxic Your Medication May Become. That Is Why You Should Always Be Under A Doctor's Care. As You Progress, You And Your Doctor May Feel That Your Medication Could Be Gradually Reduced And/Or Eventually Eliminated.

Once again, if you or your doctor need some clarification as how to coordinate a complementary health program with your current program or how a complementary health program may benefit you individually, I can be contacted by calling 516-409-6978 or by sending an e-mail to healthcoach9@gmail.com

I wish you all the best, and may **God Bless You, God Bless America and May God Bless Us All.**

Note: The information on lymphology was made possible with the help of Laura L. Hensley, RN, BSN, CL The medical clinical study references came from a combination of a Medline search at **www.PubMed.gov** and Robert Cohen and his **http://www.notmilk.com /** web site.