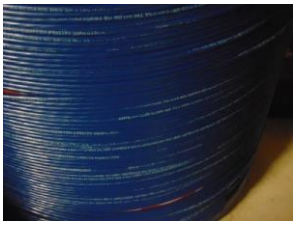
## Anti Nano

Here is the way to make your Anti Nano Chamber---what you will need for materials are wire ( insulated) a bucket ( pail) 5 gallon-some insulation tape
Take the wire and leave at least 12 inches or more so that the wire will be long enough to allow for contact with your power supply—then tape the wire to the pail to get it started so when you wrap the wire around the pail the wire won't slip-then proceed to wrap around til you cover the pail all he way—



when done then tape the finished wrapping –make sure you leave at least 12 inches to allow for contact for the power— Power supply can be a Laptop Power Supply

## How to Use

2 ways to do this and will give both formulas

**Formula 1** take 3 gallons of Vinegar( white) and add 1/8-1/4 cup of salt
And stir—connect power supply

**Formula 2** – 3 gallons of Distilled water and 1-2 caps of DMSO and Citric acid 1/8-1/4 cup and Salt 1/8- 1/4 cup mix and then connect power supply
Sit with either one leg or both in the pail for 20-30 minutes

**Formula 3**- take TSP -1/8 cup—Salt 1/8 cup Citric Acid 1/8 cup-Distilled water 3-3.5 gallons ( 12 litres for the metrically orientated) mix and set the bucket it up and place foot or feet into the bucket with the coils activated 20-30 minutes

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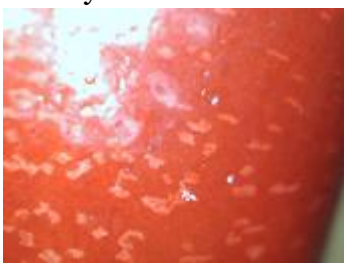
**Minimizing Reloading of NANO/Polymer/Biofilm—**with the current level of research and observations I am going to present here how you are being reloaded and offer sum Solutions as well to help you achieve optimal resistance and hopefully restoration

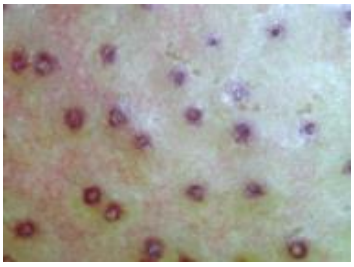
Here is how you are being reloaded—or I should say one of the ways you are being reloaded Daily

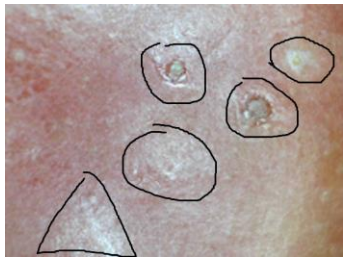

Cherry

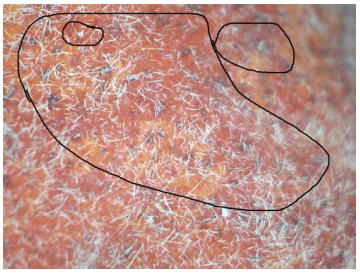

Bosh Pear

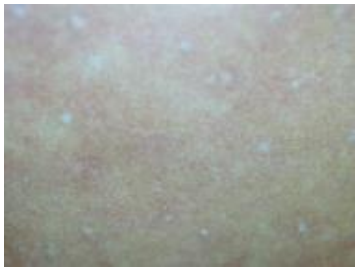

Peach stem

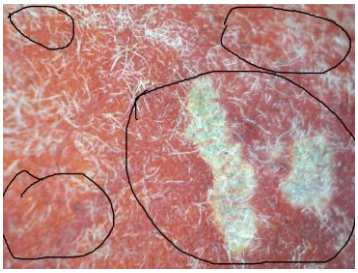

Cherries


Green Pears


Apple


Peaches


Apple


Peach


Banana


Banana

These are some of the fruits you are eating Daily that are reloading you with these nano materials and you are consuming which when they get inside of you will further spread and assimilate to your cells-dna-genetic code and chromosomes Do not use any supplements or foods that perpetrate anything NANO or **Monatomic (** nano a fancy way of saying this) especially anything metal there is more then enough Data to tell you that **Aluminum** ( chemtrail spraying nano particulates) **Titanium Dioxide** ( nano chemtrails and in cosmetics and supplements and pharmaceuticals) **Silver** ( nano-cause extreme damage especially to the male population)-

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**Key Things to utilize—Liposomes—Polymers and Colloidals ( non nano)-**

**Liposome**

easiest way to make a liposome is to use a powdered sunflower or egg lecithin ( do not use soy it will be contaminated with genetics /nano /glyphosates and glyposate adjuvants) and mix in them your powders or liquids and trap the content between the bi layer of fat and water and use this to get into the cells what is need to increase ATP and assist the mitochondria as well as remove the nano components out—or add herbals to repair the liver such as milk thistle –sage shizandra berry-dandelion—burdock- Vitamin C ( ascorbic acid) ALA ( alpha lipoic acid) NAC( n –acetyl cysteine) MSM-Methionine –copper –zinc-selenium- to restore and replenish the liver –lungs-immune system-endocrine system-brain etc.

**Polymer**

You can make a polymer deliver method by again fusing things in proteins such as smoothies or gelatin to get again the tissues and muscles and skeletal support ongoing---using gelatin –add to them either minerals—colustrum ( for the transfer factor and the other immune boosting properties) creatine—herbals such as goji-elderberry-green tea—rosemary—thyme—bay leaf--

**Colloidals –** can be anything in MICRON size for use not **nano for any metal since** they have a totally different effect then base metals—can be minerals as well as proteins-and sugars –such glycerine ( glycerol- you can call these glyconutrient when you add vitamins or nutrient) – aloe also can be utilize and even gelatin-
By adding again to them minerals-vitamins or antioxidants you can then see the suspension and utilze—making a solution as well will allow for better penetration on a cellular and skeletal and tissue levels—by either fusing or using electro suspension—or mechanical maceration-making a colloidal copper or gold or silver can be done with a boiling method—electro suspension( placing a power source on the materials and allowing a discharge of the materials in water or alcohol or other materials to suspend

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**Things to assist in energy restoring and repair**

## ATP Activator Formulas

[0028] In this example, an ATP synthesis activator was prepared by mixing herbs in

Thyme----10% by weight

Rosemary--10% by weight

Turmeric--10% by weight

Fennel--15% by weight

Grape seeds--15% by weight

Dandelion--15% by weight

Acanthopanax senticosus (Substitute Ginseng-Siberian or even Rhodiola)--30% by weight

All are powdered and then either made into a Pill form or encapsulated—could be fused as well in alcohol or other mixes to increase the usage and methods

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Formula 2 ATP Solution

An aqueous solution containing the following components was made:

10 mg/ml of adenosine 5′-triphosphate disodium salt (anhydrous) (18.15 mM)

1 mg/ml of glycine (13.32 mM) **–(Substitute Creatine)**

36 mg/ml of $Na_2HPO_4.7H_2O$ (134.3 mM) **( Substitute TSP)**

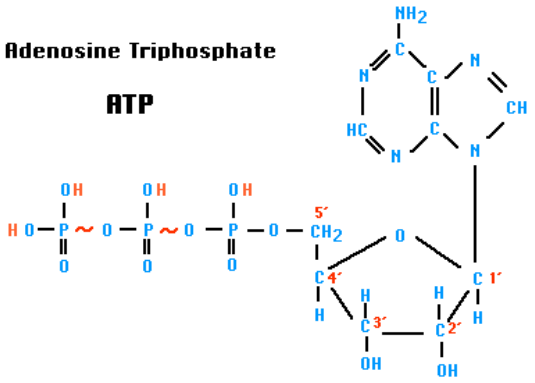
Sodium hydroxide (sufficient to adjust pH to 9.2-9.3)

Water sufficient to result in the above concentrations of adenosine 5′-triphosphate disodium salt (anhydrous), glycine, and 36 mg/ml of $Na_2HPO_4.7H_2O$.

Basically – 100 ml – of water

ATP- 1 gram---Creatine 100mgs—3.6 grams of TSP- ( also as addition add edta 1 gram and glycerol as well 10mls)

Repeat the formula and add edta 1gram—calcium carbonate ( or any other form) Potassium chloride or citrate-and about 100grams of sunflower lecithin ( you can add other ATP producers like ginger and rhodiola as we in either powder or extract) mix altogether and when it solidifies you have a liposomal ATP mix




ATP, ADP, NAD+5 6
Exogenous substances that increase endogenous production of ATP are:
ATP ATP 100 (ATP), Chromium Polynicotinate (Glucose Tolerance Factor) (HEED, Sustained Energy, Perpetuem, Premium Insurance Caps), Acetyl-L-Carnitine (Mito-r-Caps), LCarnitine (Sustained Energy & Perpetuem), Ginkgo Biloba (Super AO), Magnesium (Endurolytes & Premium Insurance Caps), Inosine (Race Caps Supreme), Coenzyme Q-10 (Race Caps Supreme), Vinpocetine (Super AO), Vitamin B3 (Premium Insurance Caps), Vitamin B5 (Premium Insurance Caps), and Vitamin C (Premium Insurance Caps).Undetermine
d7
**ENZYME ACTIVATORS TIMED DOSE FINAL PORTION OF WORKOUT**
☐
Coenzyme Q-108 9, Idebenome 10 (Race Caps Supreme) 30-60 mg GENE EXPRESSION INCREASE IN ATP-ENERGY ☐ INCREASE IN PERFORMANCE Coenzyme Vitamin B-211 12 13 (Premium Insurance Caps) 100-200 mg INCREASE IN PERFORMANCE FADH2 ☐
5 **Glucose, Chromium Polynicotinate (Glucose Tolerance Factor), Carnitine, Acetyl-L-Carnitine (ALC), LCarnitine,NADH, Ginkgo Biloba, Triiodothyronine (T3), Magnesium, Inosine, Coenzyme Q10, Hydergine,Piracetam, Vinpocetine, Vitamin B3, Vitamin B5, Vitamin C increase the endogenous production of ATP.**(Dean, Vitamin Research News. May 2000; Nicholson, Psychopharm. 101:147-59, 1985; Kisters et al. Magnes Res. 13:183-8, 2000; Regelson & Goodman, Thyroid Hormone. In: The Super-Hormone Promise: Nature's Antidote to Aging. Simon & Schuster, New York, NY, USA,

1996:169; Sinatra et al. L-Carnitine and the Heart. Keats Publishing. Los Angeles, USA. 1999:9; Seifulla et al. Exp Clin Pharmacol. 56(6):34-36, 1993).

**Adenosine Triphosphate**
is an endogenous compound that contains Adenine + Ribose (i.e. Adenosine) and three Phosphate groups. ADP is the precursor for the endogenous production of Adenosine Triphosphate (ATP). Adenosine Diphosphate is an endogenous substance manufactured within the body as part of the krebs cycle of energy production. Endogenous ATP is produced by oxidation of various chemicals the Krebs Cycle and is the body's primary "storehouse" of cellular energy. The human body is constantly manufacturing (and breaking down) ATP for
Energy production. Glucose is one of several precursors for the production of ATP - one molecule of Glucose can generate 38 molecules of ATP. Also the Electron Transport System (ETS) accepts hydrogen atoms via the Krebs cycle and passes them through a series of compounds until they combine with oxygen to form water. Each cycle of this process produces 11 molecules of ATP. The total amount of ATP recycled via metabolization by the average person for energy amounts to approximately 150-187 lbs of ATP each day. If deficits occur, fatigue results. Optimal peak production of ATP is required in order to avoid fatigue. One of the underlying causes of Chronic Fatigue Syndrome is impaired production of mitochondrial **Adenosine Triphosphate (ATP).** CFS patients have elevated blood levels of lactic acid, indicating suboptimal aerobic ATP production that can lead to fatigue and muscle aches. 7 “Undetermined” indicates the papers reviewed did not conclude the dosage range for affects..

**Coenzyme Q10** retards the aging process (including the aging process within the brain). Aging is associated with a decrease in coenzyme Q10 levels inside and outside mitochondria. (Lenaz et al.Biofactors. 8:195-204, 1998)
11 **Vitamin B2** protects the energy-producing mechanisms of the body from free radicals damage. · Haas, R. Smart antioxidants for your mind. In: Eat Smart Think Smart. Harper Collins Publishers. 1994:82.
**Coenzyme Vitamin B-3 100-200 mg** INCREASE IN PERFORMANCE NADH
**ACEYTL L-CARNITINE (OR LCARNITINE**- 750-1000 mg GENE EXPRESSION INCREASE IN ATP-ENERGY
**r-ALPHA-LIPOIC ACID** 500-750 mg GENE EXPRESSION INCREASE IN ATP-ENERGY □
**N-ACETYL CYSTEINE** 600-1200 mg INCREASE IN RECOVERYRATE & ATP-ENERGY Hendler, Sheldon Saul. The Doctor’s Vitamin and Mineral Encyclopedia. Arrow Books, London, England 1991:49. 9 Heart levels of **Coenzyme Q10** decline with the progression of the aging process. Coenzyme Q10 has been found to reverse accelerated apoptosis of cells involved in the aging process (this means that coenzyme Q10 has potential as a life extension agent by retarding the accelerated death of cells involved in the aging process). (Coles & Harris, Coenzyme Q-10 and life extension. In: Advances in Anti-Aging Medicine. Volume 1. Dr. Ronald M. Klatz (editor),1996)Kagan et al. Annals of the New York Academy of Sciences 887:31-47, 1999)

**10 Idebenone** is a smart drug that is similar in chemical structure to Coenzyme Q10 (a Quinone) and when present simultaneously enhances the potency of Coenzyme Q-10. Idebenone prevents Free Radicals from damaging the Deoxyribonucleic Acid (DNA) content of the Mitochondria (mitochondrial DNA). Idebenone is reported to enhance the function of and protect the mitochondria. (Wieland et al. Transplantation; Mason Anti-Aging Bulletin. 4(4):22-25, 1999)
12 **Vitamin B2** facilitates the metabolic production of energy from foods. Vitamin B2 plays a major role in the conversion of carbohydrates, proteins and fats into forms that the body can use for the production of energy. ☐☐Mervyn, L. Thorsons Complete Guide to Vitamins and Minerals (2nd Edition). Thorsons Publishing Group, Wellingborough, England. 1989:39. ☐☐Roberts, A. J., et al. Nutraceuticals: the Complete Encyclopedia of Supplements, Herbs, Vitamins and Healing Foods. Berkely Publishing Group. New York, USA. 2001:225.
13 **Vitamin B2** is a precursor for the production of Flavin Mononucleotide (FMN) and Flavin Adenine Dinucleotide (FAD), which are Hydrogen carriers for the production of Adenosine Triphosphate (ATP) within the body. Studies have confirmed that people who engage in regular exercise have a higher daily requirement for vitamin B-2. 14 Haas (1992) reports that decreased energy production is one of the first signs of vitamin B3 deficiency. ☐☐Haas, Elson M. Staying Healthy with Nutrition. Celestial Arts, Berkeley, California, USA. 1992:119.
15 **Vitamin B3** is involved in the metabolism of carbohydrates. Vitamin B3 is essential for the body's production ofEnergy (due to its role in the manufacture of Adenosine Triphosphate (ATP). Vitamin B3 reduces fatigue.
**Carnitine** (acetyl-l and l- forms) resides in the cytoplasm of cells where it combines with molecules of Acetyl Coenzyme A and molecules of long-chain saturated fatty acids to form a complex that can penetrate the wall of the mitochondria and facilitates the transportation of fatty acids across cell membranes into the mitochondria. Carnitine in the brain, blood, heart and muscles decreases in tandem with age, while carnitine levels in the liver increase with the aging, indicating that the transportation of carnitine from the liver to the blood declines as we age. Carnitine retards the progression of the aging by preventing cell death via the maintenance of energy supply to individual cells.
(Maccari et al. Exp Gerontol. 25:127-134, 1990; Paradies et al. Mech Ageing Dev. 84(2):103-112, 1995.)
17 **Carnitine (acetyl-l and l- forms)** increases the body's production of energy. Carnitine facilitates the transport of long-chain saturated fatty acids into the mitochondria and stimulates the oxidation of fatty acids to produce energy. A molecule of carnitine in the cytoplasm outside the mitochondria combines with a molecule of fatty acid and a molecule of Acetyl Coenzyme A to make a complex that can penetrate the wall of the mitochondria. Inside the mitochondria the complex liberates the carnitine, which can move outside to repeat its action of serving as a shuttle to carry more molecules into the mitochondria. (Arenas et al. Muscle & Nerve. 14(7):598-604, 1991.)
**18 Carnitine** improves cardiovascular function in people who engage in exercise, reduces the accumulation of lactic acid during exercise, prevents muscle pain in people who exercise, preserves muscle glycogen during exercise, increases muscle carnitine levels during exercise, prevents exercise-induced declines in muscle

carnitine levels and increases the rate of recovery following exercise. (Brass et al. J Am Coll Nutr. 17:207-215, 1998; Bremer, Physiol Rev. 63:1420-1480, 1983; Cerretelli et al. Int J Sports Med. 11(1):1-14, 1990.)

Citrate, cis-Aconitate,Isocitrate,Oxalosuccinate,alpha-Ketoglutarate,Succinyl-CO-A, Succinate,Fumarate,Malate.(Race Caps Supreme's d-AlphaTocopherol Succinates, Alpha-Ketoglutarate, proprietary kreb cycleintermediates)Undetermine d20

**KREBS CYCLE INTERMEDIATES ☐**
Alpha-Ketoglutarate, Glycine, Alanine.(Race Caps Supreme's Alpha-Ketoglutarate & proprietary kreb cycle intermediates; Glycine is formulated in HEED & Endurolytes powder.) Undetermined 21 SPECIFIC PROTEIN ACTIVES KREB CYCLE & OXIDATIVE PHOSPHORYLATION
Quercetin (3,5,7,30 ,40 -Pentahydroxyflavone) 22 23 24, 30-50 mg GENE EXPRESSION,INFLUENCE = INCREASE INRECOVERY-RATE & ATPENERGY
☐ Dimethylaminoethanol (DMAE) 25 26 27 28 -500-100 mg GENE EXPRESSION INFLUENCE = INCREASE IN RECOVERY-RATE & ATPENERGY
☐
Carnosine 29 30 (HEED, Sustained Energy, Perpetuem) 50 mg REDUCE AGE's ☐ ENERGY RECOVERY-RATE & ATP ENERGY
☐
BIOTIN 31

**MITOCHONDRIA/GENE EXPRESSION EFFECTS ENZYME PRECURSORS**

metabolism; gene expression VIA glucokinase synthesis
Hexokinase,
Glucose-Phosphate Isomerase,
Adolase,
Triosephosphate-Isomerase,
Glyceraldehyde 3-Phosphate-
Dehydrogenase, Phosphoglycermutase
Enolase,
Pyruvate Kinase.
(Race Caps Supreme's & Premium
Insurance Caps' Enzyme Enhancement
Undetermine d32

***Cysteine*** food sources include whey protein isolates, poultry, yogurt, egg yolks, red peppers, garlic, onions,. Whey proteins are rich in cysteine as well as in GSH precursor peptides. Recent clinical trials have demonstrated that intake of cysteine-rich whey protein formulas benefits patients with HIV/AIDS. In both short-term (2 weeks) and long-term (6 months) studies, supplementation with whey protein formulas increased plasma glutathione levels in patients with HIV infections. Further, the treatment was well tolerated. Also, intake of a cysteine-rich whey protein supplement for eight weeks increased weight gain, reduced the occurrence

of gastrointestinal side effects, and improved tolerance to highly active anti-retroviral therapy in HIV patients (Pacheco et al., J. Human Virology 5(1), 2002).
20 “Undetermined” indicates the papers reviewed did not conclude the dosage range for affects.
**Quercetin** was demonstrated to reduce (inflammation) the concentration of prostaglandin E2 in the pleural exudates of rats given carrageenan intrapleurally. (Mascolo, N., et al. J Pharm Pharmacol. 40:293-295, 1988.) 23
**Quercetin** has been demonstrated to inhibit (inflammation) mast cell degranulation and the subsequent release of histamine. (Miller, A. Alternative Medicine Review. 6(1):20-47, 2001.)
**Quercetin** protects the body’s endogenous Deoxyribonucleic Acid (DNA) from breakage from ferric iron-initiated hydrogen peroxide lipid peroxidation (Quercetin removes ferric iron out of the body). (Duthie et al. European Journal of Nutrition. 38(1):28-34, 1998.) 25 Pieralisi reports that athletes taking
**DMAE** for only 6 days significantly improved total workload capacity and maximal aerobic capacity (VO2 max). (Pieralisi et al. Clin Ther.13(3):373-382, 1991.) 26 Hochschild investigated the potential life-extending effect of
**DMAE** on old mice. DMAE administration in the drinking water resulted in a reduction in mortality and an increase in both mean and maximum survival times. This research found that DMAE extended the lifespan of mice by 27-49%. (Hochschild. Exp Geront. 8(4):177-183, 1973.)
27 **DMAE** increases energy and reduces fatigue. (Pfeiffer et al. Science. 126:610-611, 1957.)
28 **DMAE** inhibits (and reverses) the cross-linking of endogenous proteins. DMAE diminishes the extent of crosslinking in aged rats. (Zs-Nagy et al. Mech Aging Dev. 14:245-251, 1980.
29 **Carnosine (beta-alanyl-L-histidine)** rejuvenates cells that have reached their "*Hayflick Limit*" (causing the senescent phenotype of old cells that have reached their hayflick limit to revert to their juvenile phenotype state). The Hayflick Limit is the point at which a Cell reaches Cellular Senescence. Most Cells regenerate themselves by dividing to form two new Cells. Eventually Cells reach a limit beyond which they can no longer continue to divide. Senescent Cells that have reached their Hayflick Limit do not die, however they do become distorted in their form and function. For example, cultured human Fibroblasts have a Hayflick Limit of 60-80 divisions. Cells that have reached their Hayflick Limit have a grainy appearance. They assume odd sizes and shapes and no longer line up in parralel arrays. These changes are known as the senescent phenotype. (Hipkiss et al. Perspect Hum Biol. 1:59-70, 1995 & Yuneva et al. J Anti-Aging Medicine. 2:337-342, 1999.)
**Carnosine (beta-alanyl-L-histidine)** has protective functions additional to antioxidant and free radical scavenging roles. It extends cultured human fibroblast life span, kills transformed cells, protects cells against aldehydes and an amyloid peptide fragment and inhibits, in vitro, protein glycation (formation of cross-links, carbonyl groups and AGEs) and DNA/protein cross-linking. Carnosine is an aldehyde scavenger, a likely lipofuscin (age pigment) precursor and possible modulator of diabetic complications, atherosclerosis and Alzheimer's disease. (Boldyrev et al.

Biosci Rep. 19 (6), 581-587, 1999 & Hipkiss Int J Biochem Cell Biol. 30(8):863-868, 1998.)
31 **Biotin** deficiency is common. Estrogen excess, caffeine, egg whites, alcohol (ethanol) reduce or destroy the body's reserves of biotin. Pharmaceutical antibiotics destroy the beneficial intestinal bacteria that produce endogenous biotin and some forms of also directly destroy biotin itself. As deficiency occurs, reduced energy and premature fatigue are predicted. Biotin is closely involved (via its role as a cofactor for various enzymes) in the endogenous production of energy and the process of gluconeogenesis. Gluconeogenesis is a metabolic process involving in the formation of glucose by the liver from non- carbohydrate precursors such as glycerol, lactic acid or glucogenic amino acids. With Biotin deficiency fatigue will result. Four normal subjects were placed on an experimental diet that was deficient in biotin. After ten weeks of this diet, subjects experienced fatigue. Fatigue was relieved by subsequent biotin supplementation. (Sydenstricker et al. JAMA 118:1199-1200, 1940.)
32 “Undetermined” indicates the papers reviewed did not conclude the dosage range for affects.

SystemTM is a proprietary blend of enzymes: protease, amylase, glucoamylase, lipase, cellulase, phytase, maltase, and sucrase)

Inositol Hexaphosphate (IP6) 33 34 35 36-cDNA micrOanalysis downmodulatES gene transcription and cell cycle regulation & coherent upregulation of cell cycle inhibitors REDUCING RISK OF CARDIOVASCULAR DISEASE/CANCER ☐

Arginine-Lysine-Glucose-ALG 500-1000 mg each post workout ADDITIVE carbohydrates to prevent AGE's REDUCE AGE’s INCREASE RECOVERY-RATE & ATP ENERGY

HYDERGINE PRECURSORS 6-9 mg as Hydergine GENE EXPRESSION INCREASEIN ATP-ENERGY ☐

Urticaria Tomentosa (Cat’s claw) 38 39 20-60 mg Lipopolysaccharide-induced iNOS gene expression, nitrite formation, cell death and inhibited the activation of NF-kappaB ANTIOXIDANT & ANTIINFLAMMATORYAGENT

FORSKOLIN 40 41 250-500 mg unique diterpene activator of camp, specific stimulator of adenylyl cyclase, inhibits the release of histamine, GENE EXPRESSION INFLUENCE = circadian gene expression (rPer1, rPer2, dbp) INCREASE IN RECOVERYRATE & ATP-ENERGY

**Inositol Hexaphosphate** is an organic acid composed of one molecule of the myoinositol form of inositol joined with six phosphorus molecules (i.e. It is the hexaphosphate ester of inositol). IP6 up-regulates the expression of the

tumor suppressor gene p53 and p21WAF1/CIP1 gene and their modulation may be one of the mechanisms of the anti-neoplastic action of IP6. Since loss of p53 function enhances cancer cells' resistance to chemotherapeutic agents, the stimulating function of IP6 on p53 makes it an attractive (anti-cancer) adjuvant chemotherapeutic agent. (Saied et al. Anticancer Research. 18(3A):1479-1484, 1998)
34 **Inositol Hexaphosphate** prevents various types of cancer by repairing damaged double-strand DNA breaks Deoxyribonucleic Acid (DNA). (Hanakahi et al. Cell. 102:721-729, 2000.
35 **Inositol Hexaphosphate** inhibits the ability of iron to induce lipid peroxidation. (Porres et al. Proc Soc Exp Biol Med. 221(1):80-86, 1999)
36 **Inositol** is an antioxidant, scavenges hydroxyl free radicals, prevents fatty liver, and may be required for the metabolism of cholesterol and elevated serum cholesterol can occur as a result of inositol deficiency. (Ramakrishnan et al. Indian J Biochem Biophys. 36(2):129-33, 1999)
37 The structure of a synthetic **glucose-arginine-lysine** derived advanced glycation end product that is immunologically cross-reactive with naturally occurring counterparts. The properties of **arginine-lysine imidazole (ALI)**, immunoreactivity, acid-lability, nonfluorescence, and inhibition of formation by aminoguanidine, suggest that ALI is likely to typify an important class of the AGE cross-links that form in vivo. (Al-Abed & Bucala. Bioconjug Chem. 2000 Jan-Feb;11(1):39-45)
**Cat's claw (Uncaria tomentosa)** is a medicinal plant from the Amazon River basin, widely used for treating inflammatory disorders. It has previously been described as being an inhibitor of NF-kappaB. Cat's claw is an effective antioxidant and is a potent inhibitor of TNFalpha production. (Aquino et al. J Nat Prod. 54(2):453-459, 1991; Sandoval et al. Free Radical Biology & Medicine. 29(1):71-78, 2000)
**Uncaria tomentosa water extracts (C-Med-100)** was shown to enhance DNA repair, mitogenic response and leukocyte recovery after chemotherapy-induced DNA damage in vivo. (Sheng et al. Phytomedicine. 8(4):275-282, 2001)

GOAT'S RUE (G. Officinalis) 42 43 200-300 mg Guanidine-INDUCED hypoglycemic EFFECTS, positive effects on insulin receptorexpression & tyrosine kinase activity, pharmacological inhibitor of iNOS, NE, & ACh REDUCED PROSTGLANDIN PRODUCTION ☐
Butein (3,4,20 ,40 - Tetrahydroxychalcone)44 50 mg GENE EXPRESSION INFLUENCE = INCREASE IN RECOVERY-RATE & ATPENERGY ☐
Piceatannol (3,5,30 ,40 -tetrahdroxytrans- Stilbene)45 30-50 mg GENE EXPRESSION INFLUENCE = INCREASE IN RECOVERY-RATE & ATPENERGY
40 **Forskolin** enhances Energy production by every Cell in the body (due to it elevating the body's cAMP levels) and assists the treatment of hypothyroidism by stimulating the production and release of thyroid hormones. (Haye et al. Mol Cell Endocrinol. 43:41-50, 1990; Roger et al. Exp Cell Res. 172(282-290), 1990; Saunieret al. J Biol Chem.

265:19942-19946, 1990.)
41 **Forskolin** facilitates weight loss in persons afflicted with obesity by stimulating the production of camp, which in turn regulates the process of lipolysis. Forskolin inhibits the endogenous synthesis of fatty acids in adipocytes. Forskolin counteracts the decreased response by adipocytes to adrenaline, which occurs as a result of the aging process. (Allen et al. J Pharhacol Exp Ther. 238(2):659-664, 1986; Ho et al. Biochem Biophys Res Commun. 107(1):157-164, 1982; Hoffman et al. Horm Metab Res. 19:358-360, 1987; Okuda et al. J Lipid Res. 33:225-231, 1992.)
42 **Goat's Rue** lowers elevated blood sugar (glucose) levels (due to the **Galegine** content of goat's rue). Goat's Rue helps to prevent cross-Linking (it prevents the formation of toxic glycosylation end-products. Goat's Rue contains Galegine (2-methyl-2-butenyl-guanidine) a raw material used in the manufacture of pharmaceutical drugs such as
**Aminoguanidine, Metformin**, and **Phenformin**. Petricic et al found that Goat's rue lowered blood sugar in diabetic rats by 32%. Muller reported that Goat's Rue lowers blood sugar in diabetes mellitus patients and in normal subjects. (Petricic et al. Acta Pharm Jugosl. 32(3):219-223, 1982; Muller et al. Arch Expll Path Pharm. 125:212-228, 1927.)
43 Substances in **Goat's Rue** are used as precursors for the production of **Aminoguanidine**. However, both **Vitamin B-1 & Carnosine** are more potent substances for preventing and reversing cross-linking compared to **Aminoguanidine**.44 Research reports three classes of small molecules that activate sirtuins. They show that the potent activator
**resveratrol**, a polyphenol, lowers the michaelis constant of sirt1 for both the acetylated substrate and NAD 1, and increases cell survival by stimulating sirt1-dependent deacetylation of p53. In yeast, resveratrol mimics calorie restriction by stimulating sir2, by **increasing DNA stability and extending lifespan by 70%, and based on this research, I estimate that stimulation of sirti catalytic rate by plant polyphenols compound ratio to control Butein by 45%, Piceatannol by 41%, Isoliquiritigenin by 39%, Fisetin by 34%, Quercetin by 24%.** (Sinclair et al., Small molecule activators of Sirtuins extend saccharomyces cerevisiae lifespan. Nature. 2003 sep 11;425(6954):191-6. Epub 2003 aug 24.)
45 **Piceatannol** is a naturally occurring analog of Resveratrol. Piceatannol is a closely related stilbene that has antileukaemic activity and is also a tyrosine kinase inhibitor. Piceatannol differs from resveratrol by having an additional aromatic hydroxy group. The enzyme CYP1B1 is overexpressed in a wide variety of human tumours and catalyses aromatic hydroxylation reactions. We report here that the cancer preventative agent resveratrol undergoes metabolism by the cytochrome P450 enzyme CYP1B1 to give a metabolite, which has been identified as the known antileukaemic agent piceatannol. Piceatannol can be considered to be a promising chemopreventive or anticancer agent. (see footnote 11) Piceatannol increases cell lifespan by 41% compared to 70% for its parent metabolite Resveratrol.
RNA:DNA 10:1 50 1500-1800mg agE increase in cell rRNA INADEQUATE FOR cell volume
□

PROPIONYL L-CARNITINE51 Sublingual 200 mg x 1- INCREASE ATP STORE & ENERGY

49 **Resveratrol** is found in grape skins, seeds, stalks, vines and roots, but not the flesh of the fruit. The highest concentration of Resveratrol is in the grape skins. Resveratrol content of grape skins is 50-100 mcg per gram or 5,000-10,000 mcg per 100 grams. While I was excited to read of David Sinclair's research that reported that Resveratrol advanced lifespan, it also may produce mild estrogenic effects that oppose the androgenic value of testosterone particularly in males, partially in females. Until more research answers the safety and efficacy questions about this interesting substrate, I chose to reserve all testing applications. Genetic regulator of lifespan identified a gene that extends lifespan in yeast points to paradigm shift in longevity research may explain life extension via calorie restriction. Researchers at Harvard Medical School (HMS) discovered that a gene that may control lifespan. The gene, PNC1, determines lifespan according to its biochemical environment. A yeast strain with five copies of PNC1 lives 70 percent longer than other strains. The PNC1 gene protein a form of vitamin B3. Vitamin B-3 inhibits Sir2 gene expression and may either lengthen or shorten lifespan. Under normal conditions, Sir2 uses NAD as a cofactor to produce nicotinamide, which then inhibits Sir2 in a negative feedback loop. When the cell is exposed to environmental stresses like calorie restriction, heat shock, or osmotic stress (top), PNC1 is turned on. Then, Pnc1 protein converts nicotinamide to nicotinic acid, a molecule with no effect on Sir2. No longer inhibited by nicotinamide, Sir2 is inactivated resulting in increased cell lifespan. Sir2 extends lifespan by keeping ribosomal DNA stable. PNC1 converts nicotinamide into nicotinic acid, a molecule that does not affect lifespan. In doing so, it keeps nicotinamide from inhibiting Sir2, allowing the cells to live longer. The finding implies that lifespan is not simply dependent on accumulated wear and tear or metabolism, as some have suggested, but is at least partly controlled by an active genetic program in cells--one that could theoretically be boosted. Life extension from calorie restriction is a result of an active cellular defense involving turning on a specific gene. Severe calorie restriction extends the lives of many organisms like yeast, fruit flies, worms, and rats, and slows the aging process even preventing cancer in rats. While Sir2 is a necessary part of the equation, calorie restriction does not affect Sir2 levels, indicating that Sir2 must be regulated by another protein that does respond to calorie restriction. NAD, a cofactor of Sir2 and metabolite in the cell regulates metabolism. Because NAD level correlates to metabolism rate, this suggests that calorie restriction might lengthen lifespan by lowering metabolism. Dr. Sinclair's group showed that the effect of PNC1 was independent of NAD availability. They believe that the real regulator of Sir2 is nicotinamide, which is one of the products of the reaction between Sir2 and NAD. PNC1 levels are highly sensitive to environmental cues like calorie restriction, low salt, and heat that are known to make yeast live longer. Sinclair's team believes that the PNC1/nicotinamide pathway provides a genetic link between the environment of an organism and its lifespan, allowing an organism to actively change its survival strategies according to the stress sensed. Humans however have seven Sir genes, not just Sir2. The nicotinamide pathway inhibits human SIRT1, a homologue of Sir2. One of the immediate implications of the work is that it

emphasizes the functional difference between nicotinamide and nicotinic acid. Nicotinic acid (niacin) is a known anticholesterol treatment, while nicotinamide (or niacinimide) is sometimes touted for anti-aging abilities and is in clinical trials as a therapy for diabetes and cancer. Sinclair's study raises the concern of taking high doses of nicotinamide," Sinclair said, because nicotinamide puts a damper on Sir2's actions in the cell.50 Supplemental **RNA** or **DNA** is broken down (metabolized) within the body to its constituent Purines (Adenine and Guanine) and Pyrimidines (Cytosine and Uracil). These constituents are then reassembled within the body to form new Ribonucleic Acid (RNA) and Deoxyribonucleic Acid (DNA). Supplemental RNA possibly counteracts the progression of the aging. The Brain's levels of RNA decline in tandem with age. Supplemental RNA possesses possible life extension properties. Rats injected with supplemental RNA in conjunction with supplemental DNA lived for twice as long as controls and rats supplemented with oral RNA (25 mg per day) increased their lifespan by +16%. (Heyden, Nature. 184:433, 1959; Odens, Journal of the American Geriatrics Society. 21:450-451, 1973; Frank, Nucleic Acid and Antioxidant Therapy of Ageing and Degeneration. New York: Rainstone Publishing, 1977.)

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## Enhancing Energy Levels Through Mitochondrial Support

*By Nieske Zabriskie, ND* Surveys have shown that almost one-third of adults report having fatigue,[1] and 24 percent of patients report that their fatigue is a major health problem.[2] Many scientists believe that one cause of fatigue is mitochondrial dysfunction.[3] Mitochondria are structures within cells primarily responsible for energy production. The mitochondria are often referred to as the powerhouse of the cell, and are responsible for cellular respiration and the resulting generation of adenosine tri-phosphate (**ATP**). ATP is the chemical energy currency in the cell. The body produces an amazing 50 to 75 kg of ATP per day.[4] There are three main pathways used to generate energy: cellular respiration including glycolysis and the citric acid cycle, oxidative phosphorylation, and beta-oxidation. Mitochondrial dysfunction results in decreased ATP production and thus, may lead to fatigue.

### Aging, Fatigue, and Mitochondrial Function

Normal mitochondrial function is imperative for optimal energy production. Aging cells have a diminished ability to produce ATP due to changes in mitochondrial structure and function. Aging has been shown to decrease the efficiency of mitochondrial oxidative phosphorylation, which provides the majority of ATP production. Aging also increases the production of damaging free radicals such as reactive oxygen species (ROS) in the mitochondria.[5] Cells have several antioxidant enzymes to remove excess ROS from causing damage; however, these enzymes, as well as the enzymes required for oxidative phosphorylation, decrease with age.[6] Mitochondria have their own DNA, and research indicates that mitochondrial DNA mutations begin accumulating in cells in individuals after the mid-thirties,[7] which

contributes to the decreased ATP production and increased levels of ROS seen with increasing age.[8] Also, researchers have shown that the loss of muscle mass and function seen with aging is associated with mitochondrial damage in muscle cells.[9]

Numerous diseases are associated with mitochondrial dysfunction such as Parkinson's disease, Alzheimer's disease, coronary artery disease, chronic fatigue syndrome (CFS), fibromyalgia, and diabetes, among others.[10] Fatigue, in particular, is associated with mitochondrial dysfunction. One study found that muscle biopsies indicate that post-viral fatigue syndrome may be due to mitochondrial dysfunction precipitated by a virus infection.[11] Evidence also indicates that fatigue seen in other conditions such as metabolic syndrome is due to excess cellular oxidative stress caused by free radicals leading to oxidative damage to mitochondria, and resulting in reduced efficiency of mitochondrial energy production.[12] Studies have also shown that patients suffering with chronic fatigue have improved with supplementation of mitochondrial nutrients and **antioxidants**, showing a reduction in damage to mitochondrial membranes, restoring mitochondrial energy production, protecting cellular structures and enzymes from oxidative damage, and decreasing fatigue.[13]

## Nutrients to Support Mitochondrial Function

| TABLE 1. Fatigue-Fighting Nutrients | |
|---|---|
| L-Carnitine | Patients with chronic fatigue syndrome have significantly lower levels of serum acetyl l carnitine, total carnitine, and free carnitine. |
| Lipoic Acid | Protects and repairs age-induced mitochondrial DNA damage, thereby up-regulating mitochondrial function and improving energy production. |
| N-Acetyl Cysteine (NAC) | Directly improves mitochondrial energy production efficiency. |
| D-Ribose | In patients with fibromyalgia and/or chronic fatigue syndrome, supplementation with this five-carbon sugar has resulted in increased energy and overall well-being. |

While there are a number of nutrients shown to improve various aspects of mitochondrial function, there are seven nutrients that can be especially effective and act synergistically to improve mitochondrial function. These seven nutrients are **L-carnitine**, **lipoic acid**, **N-acetyl cysteine**, succinic acid, EDTA, plus **D-ribose**, and **Coenzyme Q10**.

| | |
|---|---|
| Coenzyme Q10 (CoQ10) | Decreases with age, which may contribute to age-related mitochondrial dysfunctions; Shown to decrease fatigue after physical activity and improve energy levels of chronic fatigue patients. |
| Succinate (succinic acid) | Helps support the health of patients with mitochondrial defects. |
| EDTA | Works with the above nutrients to stabilize mitochondrial membranes. |

Carnitine plays an important role in fatty acid metabolism and is essential for mitochondrial energy production. Acetyl-L-carnitine is a derivative of carnitine and is a precursor to the molecule acetyl coenzyme A, important in the citric acid cycle. N-acetyl-carnitine also assists in the transportation of long-chain fatty acids into the mitochondria for beta-oxidation. Beta-oxidation is the process in which fatty acids are broken down in mitochondria to generate Acetyl-CoA, the entry molecule for the citric acid cycle. The carnitines also have significant antioxidant activity, providing a protective effect against lipid peroxidation and oxidative stress.

Researchers have shown that patients with chronic fatigue syndrome have significantly lower levels of serum acylcarnitine, total carnitine, and free carnitine. Additionally, the study showed that serum levels of total and free carnitine correlated with the clinical presentation, as higher carnitine levels correlated with better functional capacity.[14] Similar studies also showed that the concentration of serum acylcarnitine in patients with chronic fatigue syndrome (CFS) tended to increase to normal levels with the recovery of general fatigue.[15] These studies suggest that mitochondrial dysfunction may contribute to or cause the symptoms of general fatigue, myalgia, muscle weakness, and post-exertional malaise in patients with CFS. In addition, numerous cardiovascular diseases exhibit similar energy metabolism dysfunction in that ATP synthesis is decreased due to inadequate fatty-acid fuels delivery to the mitochondria, and L-carnitine levels are decreased in these diseases.[16]

Lipoic acid is a potent antioxidant and has the ability to protect and repair age-induced mitochondrial DNA damage, thereby up-regulating mitochondrial function and improving energy production.[17] Animal studies have shown that supplementation with lipoic acid has dramatic effects on improving age-related declines in mitochondrial function. Lipoic acid reverses the decline in oxygen consumption, increases mitochondrial membrane potential, decreases levels of ROS and markers of lipid peroxidation, increases ambulatory activity and improves the age-associated decline of memory, increases the levels of antioxidants, and restores the activity of key enzymes.[18] Interestingly, numerous studies have shown

that acetyl-L-carnitine in combination with lipoic acid increases cellular metabolism and lowers oxidative stress better than either compound alone.[19]

N-acetyl cysteine (NAC) is a precursor for glutathione, a potent antioxidant, and stimulates the enzymes involved in glutathione regeneration. NAC also exhibits antioxidant properties of its own, counteracting the effects of reactive ROS and protecting mitochondrial proteins from damage. NAC has been shown to prevent programmed cell death (apoptosis) in cultured nerve cells and increases activity of mitochondrial complex proteins.[20-21] Additional studies have demonstrated that NAC supplementation decreased age-related memory loss, with decreased levels of oxidants in mice.[22] Research also indicates that NAC supplementation directly improves mitochondrial energy production efficiency.[23]

Ribose is a five-carbon sugar used by all living cells and is an essential component for energy production. Ribose provides the necessary substrate for synthesis of nucleotides, which form major cellular components such as ATP. The availability of ribose determines the rate at which these nucleotides can be made by the cells. In one study, D-ribose was supplemented to patients with fibromyalgia and/or chronic fatigue syndrome at a dose of 5 grams three times daily. Compared to baseline, patients reported significant improvement in all five categories measured including energy, pain intensity, sleep, mental clarity, and well-being with D-ribose supplementation. In fact, 66 percent of patients reported significant improvement, with an average increase in energy of 45 percent, and an average improvement in overall well-being of 30 percent.[24] Research also indicates that in muscle, ribose can accelerate ATP synthesis by up to 4.3-fold and increase energy salvage by up to 8-fold, which is important for muscle function and athletic performance.[25] In addition, pre- and post-exercise supplementation with D-ribose decreases free radical formation.[26]

Coenzyme Q10 (CoQ10) is a compound made by the body and primarily functions as an antioxidant, membrane stabilizer, and a cofactor in cellular respiration. CoQ10 is important in oxidative phosphorylation, and found in highest levels in the cells with the greatest energy demand, the heart and liver. CoQ10 decreases with age, which may contribute to age-related mitochondrial dysfunctions. In one study, researchers showed that in patients with chronic fatigue of unknown etiology for at least 6 months, 69 percent reported improvement with CoQ10 supplementation.[27] CoQ10 has also been shown to alleviate fatigue, improve physical performance, and decrease the recovery period with fatigue-inducing physical activity.[28]

Another substance important for mitochondrial health is succinate (succinic acid), a citric acid cycle intermediate, in which succinate is converted to fumarate. Supplementation with succinate has shown benefit in patients with mitochondrial defects.[29] Finally, EDTA can be used with all of the nutrients mentioned above to stabilize mitochondrial membranes.

**Conclusion**

Fatigue is a common complaint and presents with numerous medical conditions. However, optimizing mitochondrial function improves energy production, and may help alleviate fatigue. Nutrients such as L-carnitine, lipoic acid, N-acetyl-cysteine, succinate, and EDTA, combined with D-ribose and CoQ10 have all been shown to improve mitochondrial energy production.