Mentoring the Mentor

Mentor goals:
- To declare what is possible and establish a commitment to that possibility
- Address personal and professional barriers limiting the ability to serve
- Evolution of vision/mission/ethics that drive success
- Create immediate action steps to apply learning and growth
- Construct the round table of applied trophologists

Mentoring the mentor:
- Who are the mentors? – Practitioners
- Who are we mentoring? – Patients and GAP
- What’s the purpose? – Optimized life
- How does it work? – Whatever you learn you teach someone else (anyone else)
- Who’s is included? – Self selection, you pick yourself
Mentoring the mentor:

- Each participant attends monthly teleconferences (1 hour in duration, 4th Thursday of every 2nd month) creating a round table discussion/exploration of the dynamics and details of a nutrition-based holistic practice
- Each participant chooses how to convey the notes and information to their world and community – no information squandering

Review - Distinguish yourself

- It is more apparent why people are choosing alternative health care professionals who specialize in a functional approach
- No matter you specialty or technique you must distinguish yourself as an expert – people are just seeking to understand and they need you to do so
- Typically in the healthcare industry people are receiving shallow answers that leave them puzzled with the mystery of “Why is this happening to me?” and “What can I do about it?”
- Trends research over 10 years ago identified a number of factors essential to being successful in the nutritional field – one of those was establishing yourself as an expert

Review - Explanation as hope

- The practitioner’s ability to explain health issues and therapeutic outcomes creates an inflation of understanding in the patient which feels like hope
- Today in the professional world there is so much avoidance of ‘giving false hope’ that often we end up offering little hope at all
- I propose another model that bolsters hope and expectation and subsequently practices accountability as to whether the therapeutic endeavors are achieved or not
- As long as the hope that has been instilled is revisited and acknowledged as being accomplished or not the betrayal of false hope can be avoided
- So as an example, if a practitioner was describing the potential for nutritional intervention through supplements and diet modification to improve the lipid profile, then s/he would need to revisit to success or failure of the experiment within a reasonable period of time
- Our community is starving for legitimate hope, as a starting place, as empowerment to begin, as an idea to act upon
- There is genius in hope
Mentor Considerations
Basic inflammatory concepts as a primary health issue

Seven Pillars
Unified Mechanisms of Health
Promoting Physiology

7 Pillars of Healing
7 Unified Mechanisms of Health

- Endocrine/Hormonal
- Glycemic Management
- pH Bioterrain
- Immuno-Inflammatory
- Circulatory Status
- Digestive Potency
- Cellular Vitality
Choosing your own way
I didn’t choose to suffer
I chose to grow
The suffering was because of growth
It was worth it

So are we brave healers or timid professionals?
• Or both …
  • Do we try to prevent pain and discomfort or help people change into themselves as gracefully and directly as possible?
  • Do we believe that suffering does not lead to growth and purification?
  • So what are we avoiding with patients and personally? What are we afraid of? And btw who taught that to me/us?
What have I learned so far?

• That nutrients don’t hurt people …
• That herbs don’t hurt people …
• That the healing process may be trusted …
  (Indeed it is the only thing that can be trusted)

What do you want to learn

• How about …
• How to help another unlock the chronic pattern
• How to enter any process and create aliveness
• How to open the door wider so everything can be included
• How about the feeling that everything can be reached and blessed and healed and repaired
• How about getting sequentially to all of me

Principles: Inflammation as repair

The following statement comes from a well-known sports medicine book that has gone through five printings. “In spite of the widespread use of NSAIDs there is no convincing evidence as to their effectiveness in the treatment of acute soft tissue injuries.”

Say it clearly

This is a true statement, but definitely not strong enough.
More appropriate would be something like, –In spite of the widespread use of NSAIDs there is substantial evidence that they hamper soft tissue healing

Understanding shows itself

- NSAIDs have been shown to delay and hamper the healing in all the soft tissues, including muscles, ligaments, tendons, and cartilage
- Anti-inflammatories can delay healing and delay it significantly, even in muscles with their tremendous blood supply.
- In one study on muscle strains, Piroxicam essentially wiped out the entire inflammatory proliferative phase of healing (days 0-4). At day two there were essentially no macrophages (cells that clean up the area) in the area and by day four after the muscle strain, there was very little muscle regeneration compared to the normal healing process. The muscle strength at this time was only about 40 percent of normal.


Another study confirmed the above by showing that at day 28 after injury the muscle regenerative process was still delayed. The muscles of the group treated with Flurbiprofen (NSAID) were significantly weaker. The muscle fibers were shown under the microscope to have incomplete healing because of the medication.

Asking the right questions

- The key question regarding the healing of sports injury is, “What exactly does any therapy do to the fibroblastic cells that actually grow the ligament and tendon tissue?”

- Treatments that stimulate fibroblast proliferation will cause ligament and tendon repair and will help the athlete heal.

- Therapies that kill or hamper fibroblastic growth will be detrimental to the athlete.

Functional medicine

- In 1993 at the University of North Carolina School of Medicine, Division of Orthopaedic Surgery, Sports Medicine section, Dr. Louis Almekinders and associates studied human tendon fibroblasts to determine the effect of exercise and the NSAID Indomethacin on fibroblasts. Group I was the control in which no treatment was done; Group II—the tendons were exercised; Group III—the tendons were exercised and anti-inflamed with Indomethacin; and Group IV—the tendons were just anti-inflamed with the Indomethacin. All the tendons underwent injury through repetitive motion, similar to what would happen to an athlete in training. Seventy-two hours after the injury, it was noted that compared to controls the only group that showed increased levels of prostaglandins was the exercised group. The group that was exercised and received the NSAID, as well as the NSAID group, had statistically significant lower levels of prostaglandins (specifically Prostaglandin E2) in the tendons. This showed that the NSAID blocked the inflammatory healing of even the tendon injuries that were exercised or rehabilitated. The tendonitis that was treated with just the NSAID had almost no prostaglandins in the sample, signaling a complete inhibition of the inflammatory healing process. The effect was even more pronounced at 108 hours.

Why interrupt the repair

- The researchers also measured DNA synthesis in the fibroblasts. This showed which fibroblasts were proliferating. Again, the exercised group was the only group that exhibited elevated levels of DNA synthesis in the fibroblasts.

  Compared to the control group there was 100 percent more growth of fibroblasts in the exercise group. The tendons treated with Indomethacin had no DNA synthesis noted.
Counter culture

• This showed there was no fibroblastic growth occurring. The group that exercised and took the NSAID showed a little bit of growth. The authors concluded, "Motion and prostaglandin release in Group II were associated with increased DNA synthesis. Inhibition of prostaglandin by Indomethacin also coincided with a decrease in DNA synthesis... Inhibition of prostaglandin synthesis, and thereby DNA synthesis, may not be desirable during the proliferative stage of a soft tissue injury, when DNA synthesis for cell division of fibroblasts is needed to heal the injury to the tendon."

• The paper also stated a fact that many researchers in this field are wondering, "Despite the lack of scientific data, NSAIDs are widely used, often as the mainstay of treatment."


All making sense

• Another study was done on the use of perhaps the most popular anti-inflammatory medication used in sports medicine, ibuprofen, in the treatment of tendon injuries. It was found that only thing the ibuprofen doses used in the study caused the strength of the flexor tendons to decrease. A decrease in strength of the flexor tendons of 300 percent was observed at four weeks. The peak force of the flexor tendons of controls was 12.0 newtons, whereas in the Indomethacin group it was an average of 2.5 newtons. Extensor tendon analysis showed similar results, with controls having a breaking strength of 12.0 newtons and the tendons treated with the NSAID, 3.5 newtons. The authors noted, "Examination of the data reveals a marked decrease in the breaking strength of tendons at four and six weeks in the ibuprofen-treated animals... This difference was statistically significant."


Principles at work

From the above studies, it is clear that NSAIDs inhibit the fibroblastic growth process and thus diminish an athlete’s chance of healing. NSAIDs are used because they decrease pain, but they do so at the expense of hurting the healing of the injured soft tissue. A good example of this is a study on the use of Piroxicam (NSAID) in the treatment of acute ankle sprains in the Australian military.

Compared with the placebo group, the subjects treated with Piroxicam had less pain, were able to resume training more rapidly, were treated at lower cost, and were found to have increased exercise endurance on resumption of activity.

The conclusion of the study was that NSAIDs should form an integral part in the treatment of acute ankle sprains.


At first glance in reviewing this study, NSAIDs appear to be great, but the real question is did they help the ligament injury heal?
How we fool ourselves – for years

- In reviewing the study, the answer is a resounding NO! To test ligament healing the ankles were tested via the anterior drawer test. During this test the ankle was moved forward to determine the laxity in the ligaments. This study was published in 1997, and the author stated that this was the first time the clinical measurement of the anterior drawer sign had been used in a clinical trial. It meant that all the studies done prior to this one, in assessing whether anti-inflammatories helped with ankle sprains, did not test whether the ligaments healed. In this study at every date of testing after the initial injury, days three, seven, and fourteen, the Piroxicam-treated group demonstrated greater ligament instability. At the time of the initial injury the ligament instability in the Piroxicam group and the control group were exactly the same. This study showed that the NSAID stopped ligament healing, yet the person felt better. The authors noted: “This result is of concern in that it may reflect a paradoxically adverse effect of the NSAID-derived analgesia in allowing subjects to resume activity prematurely.”


This is true in all tissue repair

- Do you see the difference between pain relief and healing? The athlete needs healed tissue. Up until the present, too many studies were advocating NSAID use when it came to ligament injuries, because they were such great pain relievers, when in fact they were and are stopping the healing mechanisms of the body. Any technique or medication that stops the normal inflammatory process that helps heal the body must have a long-term detrimental effect on the body.

Cortisol as transactivation or repression -

- Unlike insulin, which stays outside the cell, cortisol is transported into the cytoplasm where it binds to a protein... receptor (GR) after which this complex is transported into the nucleus (HSP keep the complex from migrating prematurely)

- Once the GR moves into the nucleus there are 2 potential actions: activation (turning on) or repression (turning off) gene expression in the promoter region of certain genes

- The Glucocorticoid Response Element (GRE) binds with the GR and may then recruit other proteins required to activate gene expression and induce mRNA transcription
Getting the message out -

Hypothalamus
Paraventricular Nucleus
Releasing Hormone
Corticotrophin Releasing Hormone (CRH)
Release CRH
Paraventricular Nuclei
Median Eminence
Neurohypophysis
Adrenal Cortex
Anterior Pituitary
“Corticotrophs”
Adrenocorticotropic Hormone (ACTH)
Cortisol elevation provides negative feedback to paraventricular nuclei decreasing CRH
Cortisol Resistance
Adrenal Complex
Tyrosine
Reduce cortisol resistance
Androgenic hormones
Androstenedione, testosterone, DHT, progesterone

Modulating Cortisol
- Symplex, Hypothalmex/us – HPA general support
- Androgen up-regulation
- Adrenal Complex – 2-4/day licorice & rehmannia
- Allergen removal
- Drenamin – 6/day
- Dessicated Adrenal – 2-4/day for acute activation
- Eleuthero – 2-4/day
- Withania Complex – 2/day
- Vitanox 2-4/day
- Detoxification
- Change of thinking
- Neuro-emotional release
Modulating Cortisol

- Adrenal Complex (1-2) has exploded on the scene and represents another MediHerb homerun
- Introduced in 02/09 it has backordered multiple times as Americans have grasped its value as an idea whose time has come
- Licorice (250 mg of 7:1 extract) contains 25 mg of glycyrrhizin the active component that assists cortisone (a less active storage form of cortisol) to convert to cortisol (more active form)
- Rehmannia (150 mg of 5:1 extract) provides immune modulation
- Expect modulation in WHR, concentration, sleep quality, reduced muscle tension, relaxability, reduced anxiety
- Contraindicated when hypertension results

Pro-Inflammatory

4 – Immuno-Inflammatory

- Cumulative Repair Deficit – functional definition
- Cytokine driven inflammatory levels drive the adrenals
- All inflammation is perceived as a wild animal trying to eat you – fight or flight
- Cortisol increases, adrenals fatigue
Determining Food Allergies

- Blood type sensitivities
  -Eat For Your Blood Type, D’Amatto
- Most food allergies are delayed sensitivity reactions – difficult to objectively determine
- Elisa Act lymphocyte response assay
  - Dr. Russell Jaffe
- Elimination is the most accurate and labor intensive - 2 week elimination then reintroduce and watch for 4 days for reactions
- Histaminic Reactions (rash, red eyes, serous secretions) vs. Immune Activity (fever, catarrhal, lymphatic congestion, aching)
- Basic 4 allergies that most complicate healing process – wheat (gluten), corn, soy, milk (casein)
  - Additionally suspect chocolate, peanuts, tomatoes, beef

Food Allergies – Now & Later

<table>
<thead>
<tr>
<th>Immediate response within hours or next day</th>
<th>Delayed response onset 2-7 days later</th>
</tr>
</thead>
<tbody>
<tr>
<td>Histaminic</td>
<td>Immunological – viral, bacterial, parasitic</td>
</tr>
<tr>
<td>Red, burning eyes, serous secretions (clear)</td>
<td>Colds &amp; Flu – WBC mediated response</td>
</tr>
<tr>
<td>Tiredness, sleepiness</td>
<td>Achiness</td>
</tr>
<tr>
<td>Headaches</td>
<td>Catarrhal, phlegm (colored)</td>
</tr>
<tr>
<td>Mood changes, irritability</td>
<td>Fever</td>
</tr>
<tr>
<td>Rashes, hives</td>
<td>Eczema</td>
</tr>
<tr>
<td>Nausea, cramps, diarrhea</td>
<td>Emesis</td>
</tr>
<tr>
<td>Loss mental acuity</td>
<td>Elevated C-reactive protein, SED rate, AA:EA ratio</td>
</tr>
</tbody>
</table>

Stressors
- Tissue damage / cytokine release
- Activation 10 stages of inflammation
- Restorative functions unable to meet demand
- Cumulative Repair Deficit - inflammation
- Adrenal fatigue and hyper-reactivity
- Unburden inflammatory burdens

Symptoms - Chronic infection/toxic burdens
- Disease diagnosis – chronic progression
- Medical Intervention – anti-inflammatory

Liberated repair / renewal mechanisms
- Liberated repair / renewal mechanisms
- Graceful aging

Unburden
- Reduced inflammation / immune burden

#4 Core Physiologic Principal

Adrenal fatigue and hyper-reactivity
- Reduced Immunologic mechanisms
- Gut lining integrity – stop the leak
Generalization of allergen

- Milk allergy is primarily casein protein intolerance commonly seen in respiratory and atopic symptoms
- Wheat allergy is primarily a gluten protein intolerance commonly affecting GI symptoms and hyper tension & siderosis
- Corn allergy is primarily a zein protein intolerance commonly affecting neurological symptoms
- Soy allergy is more acquired and therefore can be unlearned commonly affecting acne rosacea and paranasal rashes
- Zypan or Betaine HCL (2-3/meal) will reduce food allergen effects

Neuro chemistry - Endorphins

- Food allergens can create morphine-like endorphins that may modulate vascular supply to regional brain areas – this has been observed on pet scans
- Caseinomorphins derived from milk protein allergy
- Glutenomorphins derive from gluten allergy
- This is the emerging biochemistry of how allergens can influence autism, ADHD, and neurological function
5 Stages of Inflammation

1 – Cytokine release from damaged cells
2 – Erythema increased blood flow
3 – Swelling plasma leak from capillaries into damaged area
4 – Leukocyte infiltration for clean up
5 – Fibrous tissue infusion creating repair

Inflammatory inhibition by cortisol

Cortisol has five effects on inflammation:
- Stabilization of membranes reducing rupture and cytokine release
- Decreases capillary permeability thus limiting swelling
- Decreases migration of WBC’s
- Suppresses immune system and T lymphocytes
- Lowers fever, interferon release and thus vasodilatation
Licorice can increase effects of Cortisol (1 tsp twice daily), thus is used a bridging protocol with prednisone

Genes – On or Off

We are in the midst of the nutrigenomics era, wherein it has been discovered that environmental factors, including diet, can turn on or turn off specific genes.

- It has been described as gene codes that may be up-regulated or down-regulated
- It is possible to do specific genomic studies that identify genetic predispositions in individual codes carried in the chromosomes
- This in turn may be predictive of certain cellular activities and metabolic tendencies an individual may have towards certain wellness or illness events
Unified Mechanisms

- As always there are some pathways that may be relevant not only to some people but to all, because of the high upstream nature of that genetic event
- The NF kappa beta gene activation has previously been observed as a gene code that may amplify inflammatory activity when engaged, and thus strategies have been developed to reduce and limit activation of this gene function
- It is well known that if the factors that reduce and limit NF kappa beta activation are employed downstream pro-inflammatory events may be effected

Free Radical Load and Antioxidant Relationship

- There are over 100,000,000,000 (100 Billion) free radicals created in the body per DAY.
- Previous medical logic was that of the stoichiometric model –

\[ 2H + 1O \rightarrow H_2O \]

1 Free Radical is offset by 1 Anti-oxidant
- ORAC – measurement – in vitro - of antioxidant capacities
- Lately, many people focused on the use of ORAC to quantify the power of their formula. There is no proof of this being valid in vivo. Also most diseased states are not dramatically altered by the use of antioxidants alone.

Many nutrients & botanicals inhibit the activation of NF-kappaB inflammatory gene activation:
- Omega 3 EPA's & GLA
- Green Tea
- Vitamin C Complex
- Curcumin/Turmeric
- Resveratrol
- Propolis
- Rosemary
- Propolis
- Vitamin D
Antioxidant Supply vs. Gene Activation

Oxygen Radical Absorbance Capacity (ORAC) is a method of measuring antioxidant capacities in biological samples in vitro.[1][2] A wide variety of foods has been tested using this methodology, with certain spices, berries and legumes rated highly.[3] Correlation between the high antioxidant capacity of fruits and vegetables, and the positive impact of diets high in fruits and vegetables, is believed to play a role in the free-radical theory of aging. However, there exists no physiological proof in vivo that this theory is valid. Consequently, the ORAC method, derived only in test tube experiments, cannot currently be applied to human biology.

- By activating Nrf2 you can multiply the body’s natural antioxidant response to combat inflammation, minimize free radical damage and transport detoxification to new levels.

Nrf2

Transcription activators that bind to antioxidant response elements (ARE) in the promoter regions of target genes. Important for the coordinated up-regulation of genes in response to oxidative stress.

Pro-inflammatory vs. Anti-inflammatory

- The goal biochemically is to promote inherent cell regulatory mechanism to complete repair activity without being exaggerated into inflammatory chaos
- So the interest turns to the foods and lifestyle events that assist the body to find its intelligent and innately directed repair activity
- Proper sleep (Phase 1-4) will promote Nrf2 gene activity and thus promote body balancing of free radical damage and toxicity
- Caloric restriction as in the Phase II diet will promote hormetic activity and bring about sirtuin and heat shock protein production and increase Nrf2 activity
Oxidative Stress

- Cancer
- Cardiovascular Disease
- Atherosclerosis
- Heart failure
- Myocardial Infarction
- Inflammation
- Renal Disease
- Neurological Disease
- Parkinsons
- Alzheimer's
- Cellular apoptosis/necrosis

Targeting Inflammation-Induced Obesity and Metabolic Diseases by Curcumin and Other Nutraceuticals

Extensive research within the past two decades has revealed that obesity, a major risk factor for type 2 diabetes, atherosclerosis, cancer, and other chronic diseases, is a pro-inflammatory disease. Several spices have been shown to exhibit activity against obesity through antioxidant and anti-inflammatory mechanisms. Among them, curcumin, a yellow pigment derived from the spice turmeric (an essential component of curry powder), has been investigated most extensively as a treatment for obesity and obesity-related metabolic diseases. Curcumin directly interacts with adipocytes, pancreatic cells, hepatic stellate cells, macrophages, and muscle cells. There, it suppresses the pro-inflammatory transcription factor nuclear factor-kappa B (NF-κB), signal transducer and activators of transcription (SIRT), and Wnt/beta-catenin, and it activates peroxisome proliferator-activated receptor-gamma and NFκB cell-signaling pathways, thus leading to the down regulation of adiponectin, including tumor necrosis factor, interleukin-6, resistin, leptin, and monocyte chemotactic protein-1, and the up regulation of adiponectin and other gene products. These curcumin-induced alterations reverse insulin resistance, hyperglycemia, hyperlipidemia, and other symptoms linked to obesity. Other structurally homologous nutraceuticals, derived from red chili, cinnamon, cloves, black pepper, and ginger, also exhibit effects against obesity and insulin resistance. The University of Texas M. D. Anderson Cancer Center, Houston, Texas
Curcumin and Nrf2 Activation

Fig. 1. Chemical structure of curcumin

Sulforaphane protects immature hippocampal neurons against death caused by exposure to hemin or to oxygen and glucose deprivation.

Sara L. Liu, W. Fei, D. Menon et al.
Department of Anesthesiology, Center for Shock, Trauma, and Anesthesiology Research (STAR), Uof M Sch/Medicine

Oxidative stress is a mediator of cell death following cerebral ischemia/reperfusion and hemoxotoxicity, which can be an important pathogenic factor in acute brain injury. Induced expression of phase II detoxification enzymes through activation of the antioxidant response element (ARE)/Nrf2 pathway has emerged as a promising approach for neuroprotection. Little is known, however, about the neuroprotective potential of this strategy against injury in immature brain cells. In this study, we tested the hypothesis that sulforaphane (SFP), a naturally occurring isothiocyanate that is also a known activator of the ARE/Nrf2 antioxidant pathway, can protect immature neurons from oxidative stress-induced death. The hypothesis was tested with primary mouse hippocampal neurons exposed to either O2/D and glucose deprivation (OGD) or hemin. Treatment of immature neurons with SFP immediately after the OGD or hemin exposure was effective in protecting immature neurons from delayed cell death. Exposure of immature hippocampal neurons to hemin induced significant cell death, and both pre- and cotreatment with SFP were remarkably effective in blocking cytotoxicity. RT-PCR analysis indicated that several Nrf2-dependent cytoprotective genes, including NAD(P)H quinone oxidoreductase 1 (NQO1), hemeoxygenase 1 (HO1), and glutamate-cysteine ligase modifier subunit (GCLM), which is involved in glutathione biosynthesis, were up-regulated following SFP treatment both in control neurons and following exposure to OGD and hemin. These results indicate that SFP activates the ARE/Nrf2 pathway of antioxidant defense and protects immature neurons from death caused by stress paradigms relevant to those associated with ischemic and traumatic injury to the immature brain.

Natural Antioxidant Activation from Supplementation of Sulforaphane

Phytoestrogen Sulforaphane

Le-yielding enzymes Antioxidants

Anti-inflammatory Nrf2 Signal transduction

Anti-inflammatory Nrf2 Anti-inflammatory
Resveratrol induces glutathione synthesis by activation of Nrf2 and protects against cigarette smoke-mediated oxidative stress in human lung epithelial cells. Kode A, Rajendraiah S, Cato B, Yang SR, Meason L, Rahman I.

19

Nuclear erythroid-related factor 2 (Nrf2), a redox-sensitive transcription factor, is involved in transcriptional regulation of many antioxidant genes, including glutathione S-transferase (GSTA1) in human normal keratinocytes HeLa cells. Resveratrol, a polyphenolic phytoalexin, has antioxidant signaling properties by inducing GSH biosynthesis, upregulating the expression of Nrf2 and protecting lung epithelial cells against CS-mediated oxidative stress. Treatment of human primary small airway epithelial and human alveolar epithelial (A549) cells with CS extract (CSE) resulted in dose-dependently decreased GSH levels and GCL activity, effects that were associated with enhanced production of reactive oxygen species. Resveratrol restored CSE-depleted GSH levels by up-regulation of UCL via activation of Nrf2 and also quenched CSE-induced release of reactive oxygen species. Interestingly, GSH failed to induce nuclear translocation of Nrf2 in A549 and small airway epithelial cells. On the other hand, resveratrol attenuated CSE-mediated Nrf2 modifications, thereby inducing its nuclear translocation associated with GCL expression. These data may have implications in dietary modulation of antioxidants in treatment of chronic obstructive pulmonary disease.

Dr. Stuart White
Mentoring the Mentors
June 3, 2015


Abstract: Astrocytes may modulate the survival of motor neurons in amyotrophic lateral sclerosis (ALS). We have previously shown that forskolin (FGF-1) activates astrocytes to increase secretion of nerve growth factor (NGF). NGF in turn induces apoptosis in co-cultured motor neurons expressing the p75 neurotrophin receptor (p75NTR) by a mechanism involving nitric oxide (NO) and peroxynitrite formation. We show here that FGF-1 increased the expression of inducible nitric oxide synthase and NO production in astrocytes, making adjacent motor neurons vulnerable to NGF-induced apoptosis. Sural astrocytes isolated from transgenic SOD1G93A rats did not display increased NO production and JNK-dependent apoptosis of co-cultured motor neurons. FGF-1 also activates the redox-sensitive transcription factor nuclear factor erythroid 2-related factor 2 (Nrf2) in astrocytes. Because Nrf2 increases glutathione (GSH) biosynthesis, we investigated the role of GSH production by astrocytes on p75NTR-dependent motor neuron apoptosis. The combined treatment of astrocytes with FGF-1 and poly(ADP-ribose) polymerase (PARP) increased GSH production and secretion, preventing motor neuron apoptosis. Moreover, Nrf2 activation in SOD1G93A astrocytes abolished their apoptotic activity. The protection exerted by increased Nrf2 activity was overcome by adding the NO donor DETA-NONOate to the co-cultures or by inhibiting GSH synthesis and release from astrocytes. These results suggest that activation of Nrf2 in astrocytes can reduce NO-dependent toxicity to motor neurons by increasing GSH biosynthesis.

Dr. Stuart White
Mentoring the Mentors
June 3, 2015

Naturally occurring phytochemicals for the prevention of Alzheimer's disease.

Alzheimer disease (AD) is an age-related neurodegenerative disease that is recognized as one of the most important medical problems affecting the elderly. Although a number of drugs, including several 

Green Tea catechins have been suggested to have the potential to prevent AD because of their anti-amylodigenic, anti-oxidative, and anti-inflammatory properties.

Dr. Stuart White
Mentoring the Mentors
June 3, 2015
New Product Alert – Read All About It!

HerbaVital released April, 2010 is a unique combination of factors to reduce the physiologic decline known as aging, but also acts as a hormetic influence to up-regulate stress responsibility and therefore survival status. This is a cocktail of daily herbal constituents that can universally support the declining stress response that is so essential to wellness and vitality. It is a strategy in a formula for daily minimizing of the underlying process of aging. This product takes the assessment out of the picture for the clinician and addresses the common background issues at work universally in the patient.

HerbaVital:
- Japanese Knot Weed root extract 100:1 80 mg providing 36 mg of natural resveretrol
- Milk Thistle seed 5:1 50 mg providing 48 mg of silybin
- Korean Ginseng root 5:1 50 mg
- Masson Pine bark 100:1 50 mg providing 37.5 mg proanthocyanidins
- Ginkgo Leaf 50:1 30 mg

Product Alert – Read All About It!

Vitanox is a unique combination of herbs to provide strong antioxidant protection, and how we discover also acts to up-regulate Nrf2 gene activity and subsequent survival compound status increase, including glutathione synthesis. This is a cocktail of daily herbal constituents that can universally support the overloaded detoxification and inflammatory mechanisms. It is a strategy in a formula for daily minimizing of the underlying process of aging and degeneration. This product was introduced by Kerry Bone based on widespread agreement about the merits of these herbs, before and correctly predicting the emerging research around Nrf2 gene activation.

Vitanox tablet:
- Rosemary leaf extract 5:1 200 mg providing carnosol and rosmarinic acid
- Green Tea leaf extract 25:1 166.7 mg providing 83.35 mg of catechins
- Turmeric rhizome extract 25:1 80 mg providing 70.4 mg curcumonoids
- Grape Seed extract 120:1 50 mg providing 42.5 mg procyanidins

Product Alert – Read All About It!

Cruciferous Complete is a combination of kale and brussel sprouts to protect against free radicals and now also is shown to up-regulate Nrf2 gene activity and subsequent survival compound status increase, including glutathione synthesis. This nutrient supports Phase I & II detoxification pathways promoting reduction of toxic load in the body and well as supports repair mechanisms involving the eye. It contains a myriad of nutrients including vitamins B6, C, K, calcium, copper, potassium, and dietary fiber. It also contains carotenoids, which include beta carotene and lutein which help quench free radical ROS effects and retinal repair activity.

Cruciferous Complete capsule:
- Vitamin K 4 mcg
- Potassium 10 mg
- Kale 300 mg
- Brussel Sprouts 300 mg
Who would benefit from Nrf2 Activator?

- Patients with Alzheimer's
- Patients with Parkinson's
- Exposure to physical stress
- Overresponse to oxidation
- Traumatic Brain Injury
- Fisher Syndrome
- Huntington's Disease
- Inflammatory Myopathy
- ALS

Principles at work

- Sufficient clinical observation allows mechanisms to be revealed that will remove the idiopathic mystery of hypertension and return it to a simple physiological modulation and resultant augmentation in function, balance, tissue fortification and promotes healthy genetic expression.
- This allows the symptom resolution to occur as a result of system ‘mosaic’ change, and then of course the downstream events occur.
- The longing in the public is for this sort of detective work to find the cause and make the correction – increasingly food is seen as medicine and people are asking more and more for what foods will change their health patterns.

Sequential Intervention

- By giving hope through discussion of therapeutic rationale and then accountably determine if the therapy had efficacy it is possible to initiate activity that may assist a person to make the changes that result in healing.
- Sequential intervention and accountably follow-up can show what has worked and what may still need to be employed.
- Promote an understanding of intervention that creates evolutions in individual physiology and show the effect of that intervention.
- See the concept of micro circulation dynamics as a unified mechanism of disease and a source to health.
- Allow every condition to become a strategic consideration of possible etiology and therapeutic rationale – people are in search of experts – reveal yourself.
- The comprehensive nature of nutritional therapy means there is always more physiology to optimize and support leaving an individual constantly refining as long as they wish to further improve their status.
- If the practitioner is accountable s/he will be allowed to experiment with reasonable ideas.
Change the world
It wants to