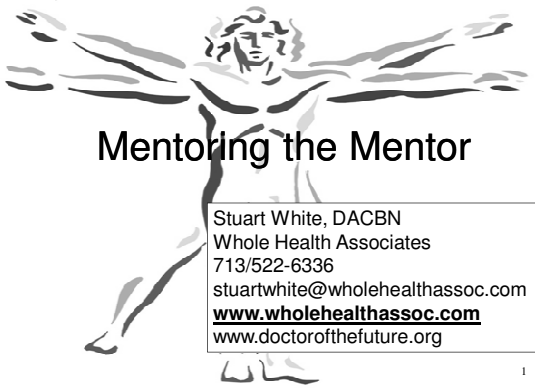


Mentoring the Mentor

Stuart White, DACBN
Whole Health Associates
713/522-6336
stuartwhite@wholehealthassoc.com
www.wholehealthassoc.com
www.doctorofthefuture.org

1

Eternal Truth

He who does not use his endeavors to heal himself is brother to him who commits suicide.

Proverbs 18:96

2

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

3

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

4

Mentoring the mentor:

- Each participant attends monthly teleconferences (1 hour in duration, 4th Wednesday 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

5

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

6

According to your DNA, from all recorded learning and adaptation, the number one cause of death is infection.

All effort shall be to survive that.

Research + Experience

You Will

Hypothesis -

The only reason why anything that ever was working, stops working is because of infection

Science

- Science is the description of the mechanized nature of reality. Why and how it works, and how to make it work – every time. As it studies the details of how things work it knows, all the rest it just hasn't discovered yet …
- Our practices are pure science – because they describe and document what works or not, and they never stop discovering new things …
- So we can make things work at will …

Precision Personalized Medicine

The cutting edge of medical development is based on genetic profile associated with pathogenic presentation

Goodheart said, "Be Precise" in 1970

Precisiion is accomplished through the practice of taking history and using the nutritional/physical examination, muscle testing coupled with tools like serum and urine analysis, genetic sampling, meridien stress assessment and heart sound recording, etc

- Musculoskeletal indicators can help show unique individual needs
- ROM
- Strength testing
- Nociception

Results

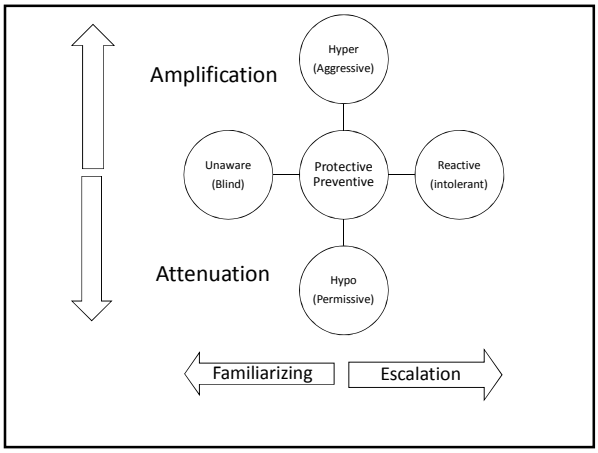
- Results described as "documented change" are the success of any practice.
- Results are:
  - Respected
  - Inspiring
  - Encouraging
  - Compelling
  - Foundational
- Success depends upon:
  - Creating change
  - Documenting change
  - Inspiring change

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)

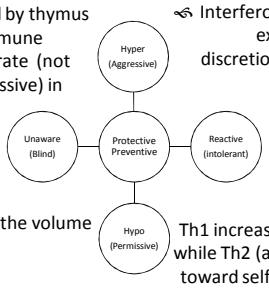
What do you want to learn

- 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/you/them



Immune Vigilance and Discretion

- Amplification / Attenuation –
- Escalation / Familiarizing –
- Thymosin produced by thymus cells mature the immune system to be moderate (not aggressive or permissive) in pattern recognition
- Interferon and cytokine expression shifts discretion/discernment



- It turns up or down the volume
- Th1 increases intolerance, while Th2 (and Th17) shifts toward self consciousness and absorption

Tools for Modulation -

Amplification	**Attenuation**
Thymex 5 bid	Lact Enz 2 qid
Golden Seal 2 bid	Prosynbiotic 2 qid
Andrographis Complex 2 bid	

Thymex

Cholesterol	5 mg
Vitamin C	5 mg

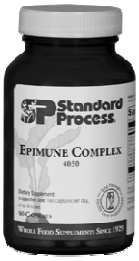
Proprietary Blend: 370 mg
Calcium lactate, bovine thymus Cytosol™ extract†, and magnesium citrate.

18

Tools for Modulation -

Escalation	Familiarization
Epimune 1 bid	Epimune 1 bid
Echinacea Premium 2 bid	Echinacea Premium 2 bid
Sesame Oil Perles 3 bid	Biost/Ostrophin PMG 3 bid
Spleen PMG/Spleen Dess	

Epimune

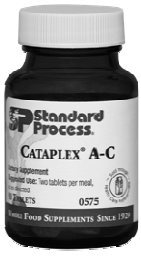


Vitamin C	20 mg
Calcium	30 mg
Zinc	10 mg

Proprietary blend amount: 706 mg
Dried yeast fermentate (EpiCor®)†, turkey tail mushroom powder†, maitake mushroom powder†, and maitake mushroom extract (Maitake Gold 404®)†.

20

Cataplex® AC



Vitamin A	1,500 IU
Vitamin C	11 mg

Proprietary Blend: 490 mg Echinacea (root)†, calcium lactate, sweet potato†, carrot (root)†, bovine adrenal†, bovine kidney†, nutritional yeast†, magnesium citrate, alfalfa flour†, dried alfalfa (whole plant) juice†, mushroom†, dried buckwheat (leaf) juice†, buckwheat (seed)†, bovine bone†, defatted wheat (germ)†, oat flour†, sunflower lecithin†, veal bone†, vitamin E (sunflower), rice (bran)†, and carrot oil†.

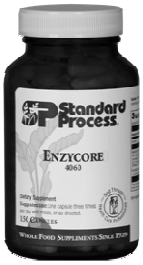
21

Digest Forte

Gentian root 2:1 extract from *Gentiana lutea* root 200 mg	100 mg
Tangerine fruit peel 5:1 extract from *Citrus reticulata* fruit peel 500 mg	100 mg
Feverfew leaf 3:1 extract from *Tanacetum parthenium* leaf 200 mg	66.7 mg
Ginger rhizome 10:1 extract from *Zingiber officinale* rhizome 250 mg	25 mg
Wormwood herb 4:1 extract from *Artemisia absinthium* herb 100 mg	25 mg
Tangerine (*Citrus reticulata*) fruit	

22

Enzycore



Proprietary Blend: 538 mg L-glutamine†, kale (aerial parts)†, beet (root)†, and vegetarian enzyme blend (acid maltase [1MaltU], alpha-galactosidase [45 GalU], amylase [1,800 DU], bromelain [32,880 FCCPU], glucoamylase [3 AGU], invertase [170 SU], lactase [325 ALU], lipase [230 FIP], peptidase [820 HUT], protease 3.0 [3 SAPU], protease 4.5 [4,930 HUT], protease 6.0 [1,640 HUT])†.

23

Inflammation: Response to Injury

Inflam = set on fire

Inflammation is triggered when tissue damage occurs

- The response:
  - 1 – Prevents the spread of damaging agents to nearby tissues
  - 2 – Disposes of cell debris and pathogens
  - 3 – Sets the stage for the repair process

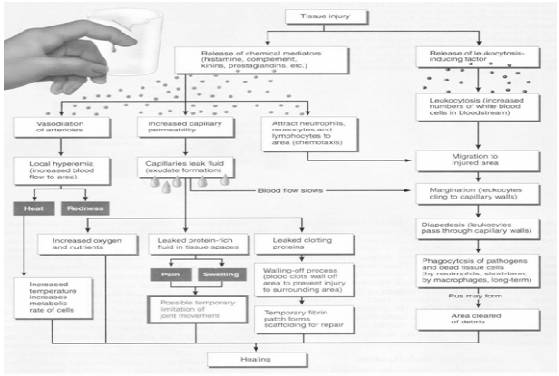
4 cardinal signs of inflammation:

1 – Rubor / redness due to hyperemia
2 – Heat
3 – Swelling
4 – Pain
5 – (Impairment of function – some experts consider this the fifth sign)

Chemical alarm sounds through histamine, kinins, prostaglandins, complement, cytokines

Tortora GJ and Derrickson B, Principles of Anatomy and Physiology, 2009, John Wiley & Sons, 12th Edition

From injury to repair



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.”

(Bruckner, P. Clinical Sports Medicine. New York City, NY: McGraw-Hill Book Company, 1995, pp. 105-109.)

26

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

27

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.

(Greene, J. Cost-conscious prescribing of nonsteroidal anti-inflammatory drugs for adults with arthritis. Archives of Internal Medicine. 1992; 152:1995-2002.)

28

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.

(Almekinders, L. An in vitro investigation into the effects of repetitive motion and nonsteroidal anti-inflammatory medication on human tendon fibroblasts. American Journal of Sports Medicine. 1995; 23:119-123.)

29

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.

30

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 IVûthe 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.

31

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.

32

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.”

(Almekinders, L. An in vitro investigation into the effects of repetitive motion and nonsteroidal anti-inflammatory medication on human tendon fibroblasts. American Journal of Sports Medicine. 1995; 23:119-123.)

33

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."

(Kulick, M. Oral ibuprofen: evaluation of its effect on peritendinous adhesions and the breaking strength of a tenorrhaphy. The Journal of Hand Surgery. 1986; 11A:100-119.)

34

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.

(Slatyer, M. A randomized controlled trial of Piroxicam in the management of acute ankle sprain in Australian regular army recruits. American Journal of Sports Medicine. 1997; 25:544-553.)

At first glance in reviewing this study, NSAIDs appear to be great, but the real question is did they help the ligament injury heal?

35

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."

(Slatyer, M. A randomized controlled trial of Piroxicam in the management of acute ankle sprain in Australian regular army recruits. American Journal of Sports Medicine. 1997; 25:544-553.)

36

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.

37

Cumulative Repair Deficit

- This is the functional term that defines the inflammatory status of the body
- Words describe ideas, and ideas are what direct us in our efforts – as clinicians when inflammation is named it is assumed that it should be reduced and blocked and that it is a bad thing – yet without the five stage inflammation process at work physiology falls into disrepair and the elaborate tissue based organism called the human body cannot be maintained and repaired
-
- In general inflammation should not be suppressed or reduced, but rather completed – when it is not completed by the end of the day it is called a cumulative repair deficit
- Most people over 40 carry a deficit of repair called silent or symptomatic inflammation

38

Inflammation – Eat(en) up

- It is not surprising that approximately 60 percent of the average diet's calories come from sugar, flour and refined oils.[3] The primary refined oils include corn, safflower, sunflower, cottonseed and soybean oils, which contain an excessive amount of linoleic acid, an omega-6 (n-6) fatty acid, which the body converts into arachidonic acid. Another 15 to 20 percent of calories come from over-fat animal products and 10 percent from dairy,[3] which contains preformed arachidonic acid.
- The issue of dietary arachidonic acid is important to understand. The arachidonic acid that we eat is converted by the body into prostaglandin E2 (PGE2), which causes pain and inflammation. In other words, we literally "eat" pain and inflammation for breakfast, lunch, dinner and snacks. Most patients have no idea that they over-eat pain and inflammation every day as dietary arachidonic acid. And most patients have no idea that the NSAIDs, aspirin and Tylenol® that many take every day then function to inhibit the conversion of dietary arachidonic acid into painful and inflaming PGE2.

Genes – On or Off

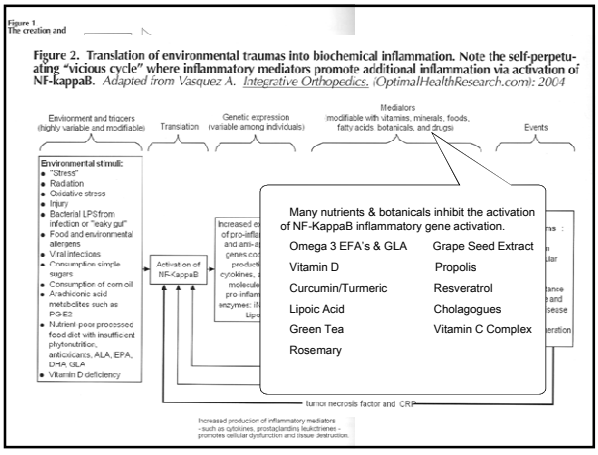
- We are in the midst of the nutrigenomics era, wherein it has been discovered that environmental factors, including diet, can turn on or turn off specific genes
- It has been described as gene codes that may be up-regulated or down-regulated
- It is possible to do specific genomic studies that identify 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

40

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

41



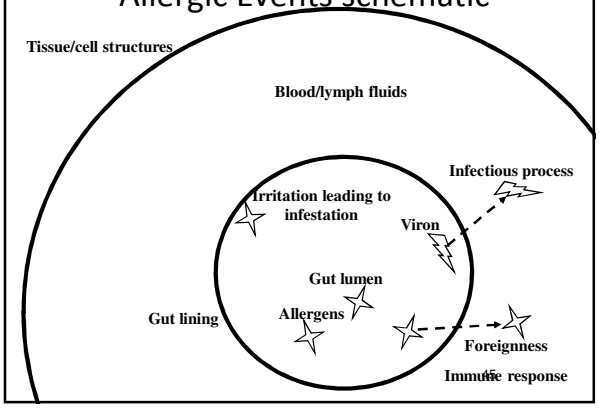
Determining Food Allergies

- 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 Serammune Labs, Virginia, 800/525-7372
- 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

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

Allergic Events schematic



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 effecting GI symptoms and hyper tension & siderosis
- Corn allergy is primarily a zein protein intolerance commonly effecting neurological symptoms
- Soy allergy is more acquired and therefore can be unlearned commonly effecting acne rosacea and paranasal rashes
-

46

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

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Just how much gluten is in Catalyn anyway?

Your average slice of whole wheat bread contains around 4.8 grams of gluten. Each tablet of Catalyn contains around .0125 mg of gluten - which means (drum roll) you would have to take around 384,000 Catalyn to equal the amount gluten in one slice of bread!

So, let's think about this . . . if you took 6 Catalyn tablets a day for a year – that's 365 days, that means it would take just over 175 years to get to the same amount of gluten as ONE slice of bread.

The smallest amount of gluten which has been shown by biopsy to cause damage to a celiac is 0.1 gram per day. This is approximately the amount of gluten contained in 1/48th of a slice of bread or 8000 Catalyn.

Catassi et al.http://www.celiac.com/articles/38/1/How-much-gluten-is-in-a-normal-diet-and-how-much-does-it-take-to-cause-damage-in-a-celiac)

48

Primary Inflammatory Foods

There are 14 primary inflammatory foods:

- To reduce these for five days is to dramatically reduce the inflammatory amplitude systemically – remarkable changes seen in 1-2 days – then re-introduction protocol can determine the actual offending food(s)
  - Milk
  - Corn
  - Gluten
  - Soy
  - Peanuts
  - Beef
  - Tomato
  - Chocolate
  - Rice
  - Nuts
  - Sesame
  - Peas
  - Eggs
  - Sugar

49

Cytokines – Immune Messages

- Immune response results in the release of cytokines meant to direct local and distant immune function
- These cytokine messenger molecules also drive HPA status and thus determine global brain status
- Cytokines subsequently cause the release of WBC inflammatory mediators to direct the inflammatory process of repair
- Therefore immune status and activity determine HPA/brain settings
- Hypervigilant or depressed immune states reflect in brain states

50

Cytokines – Immune Messages

- The HPA Axis is the conductor of homeostatic symphony
- This system additionally intertwines with virtually every aspect of physiology through the production of CRH and ACTH – indeed resistive and 'hard-to-treat" conditions all share the described cytokine cascade disturbance
- The take away is that any chronic perturbation to one element of the system will ripple through other components of the web
- Persistent stressors without relief continuously stimulate the CRH-ACTH-cortisol axis resulting in high levels of cortisol and some neuronal disruption and death in the negative feedback loop (hippocampus and hypothalamus)
- This may lead to depression and behavioral disturbance

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Cytokines – Immune Messages

- So the immune modulation and unburdening is required to achieve HPA and endocrine balance
- The concept of immune sparing and unburdening is essential to any long term concept of HPA integrity
- The sequential immune up-regulation is the avenue to HPA strength and health
- The HPA axis will not completely balance and limbic health will not be achieved without immune and cytokine support

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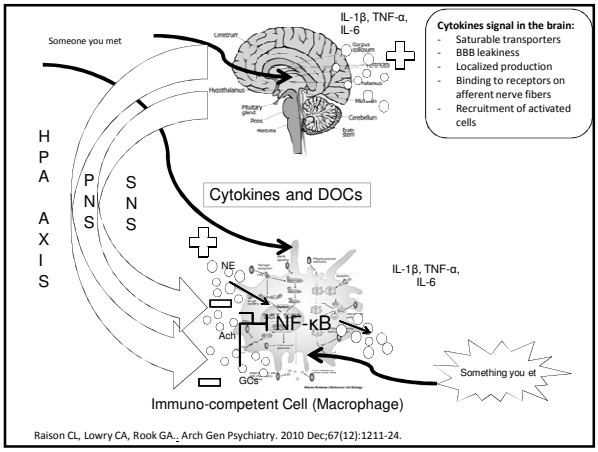


Raison CL, Lowry CA, Rook GA. Arch Gen Psychiatry. 2010 Dec;67(12):1211-24.

Pattern Recognition Receptors (PRRs)

- PRRs are involved in innate immune recognition and responses.
- PRRs recognize components of micro-organisms that are not found on the host → PAMPs (pathogen-associated molecular patterns)
- PRRs are found on the cell membrane and also within the cell
- Toll-like receptors (TLRs) are the most common class of PRRs and each recognizes a distinct bacterial, fungal or viral molecular pattern

PRR

Activation of the Toll –like receptors communicates the outside to the cytoplasm and then through the nuclear membrane in a similar fashion to the inner DNA.

These are like transformer step down conveyances of the outer world to the inner.

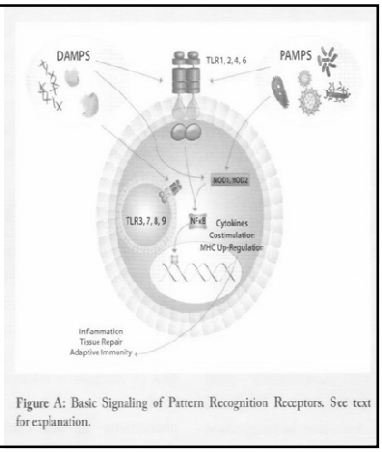
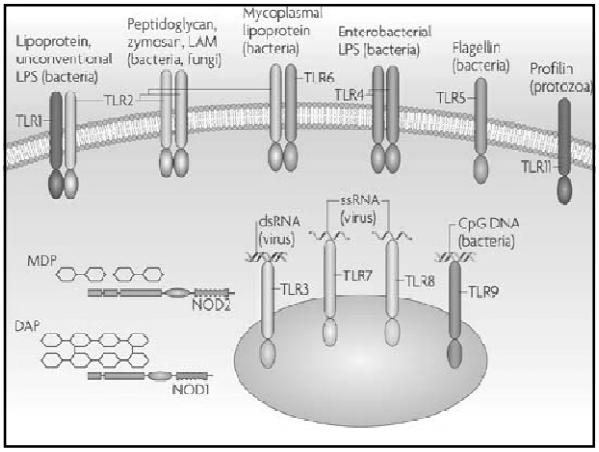


Figure A: Basic Signaling of Pattern Recognition Receptors. See text for explanation.



MHC Proteins – Self

Major Histocompatibility Complex (MHC)

The external surface of cells is dotted with variety of protein molecules, of which some are individually unique glycoproteins that mark the cell as 'self'.

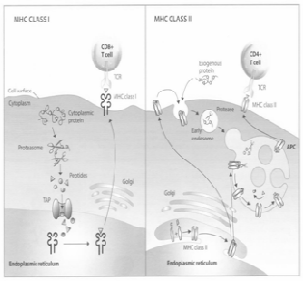
Every genetic expression of proteomics carries these 'self' MHC markers.

Class I MHC proteins are found on all cells of the body.

Class II MHC proteins are displayed only by certain immune acting cells.

Each MHC molecule has a deep groove which displays peptides from the breakdown of cellular proteins.

Infected cells have fragments of foreign antigens bound to the MHC resulting in immune activation.



Common TH1 & Th2 Cytokines

- TH1
  - Il-12
  - IFN – gamma
  - TNF – alpha
  - IL-2
  - GM – CSF
- TH2
  - IL-4
  - IL-5
  - IL-10
  - IL-13

IL-1 and IL-6 (and others) can show both TH1 and Th2 influences

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PRR's and Inflammation

Immune signaling is primarily activated through a host of receptors called pattern recognition receptors (PRR)

The first PRR discovered was the Toll-like receptor found in 1998 – since then there have been ten Toll-like receptors discovered.

C-type lectin receptors (CLR) are similar detecting helminths, fungi, mycobacterium –

Dectin-1 is a CLR that responds to pathogenic and immunomodulating fungi

Retinoic acid-inducible gene 1 (RIG-1) recognizes viral RNA and signals viral response.

Nucleotide-binding and oligomerization domain (NOD) can see PAMP and DAMP and regulate inflammatory response

When one or more PRRs recognize an antigen a cascade of intracellular signaling results incytokine up-regulation of the NF-kB inflammatory mechanism

PRR recognition directs the naïve Th0 maturation

A class of NOD-like receptors (NLR) produce complexes called inflammasomes

PPR – Toll – like Receptor (TLR)

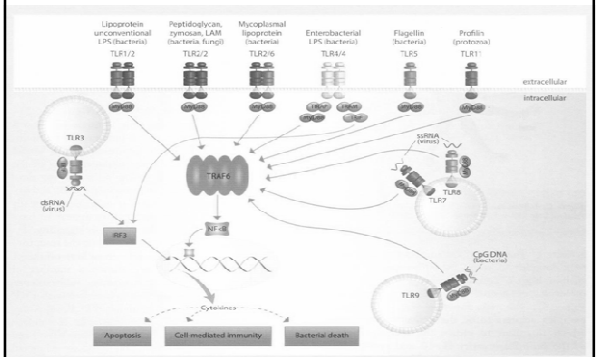


Figure B: Toll-like receptor (TLR) signaling. This diagram shows the different types of TLRs, their locations and the patterns they recognize. See text for more details about the signalling pathways. Image adapted from Minireview: Toll-like Receptors (TLR)-www.abdseronec.com.

Inflammasome – Master Sargent

Typical inflammasome is comprised of 7 molecules which facilitate pyroptosis and programmed cell death.

It is currently known to be triggered by LDL particles, high glucose, cholesterol crystals and certain fatty acids – this is the critical modulator of low-level inflammation of cardiac disease.

Target of low level inflammation

Bioactive plant compounds EGCG, luteolin, quercitin, chrysin have been shown to inhibit downstream signaling by PRR activation thus dampening inflammatory amplification.

Curcumin and Feverfew have been shown to inhibit signaling from NLR.

Prepare for a new class of herbal and nutrient tools driven by the now visible mechanisms and the confidence to support them.

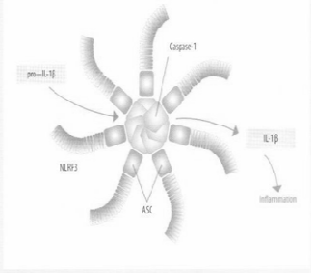


Figure C: Inflammasome (NLRP3) molecule. See text for details. Adapted from *Nature Medicine* 17, 790-791 (2011)

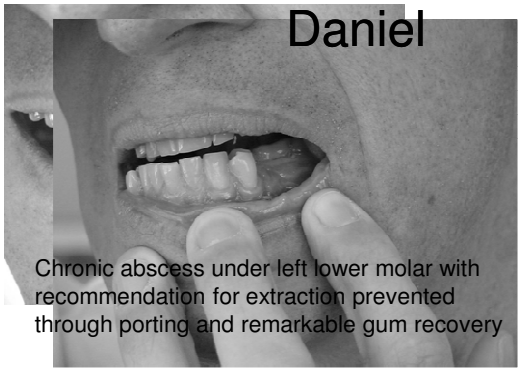
Modulating PRRs

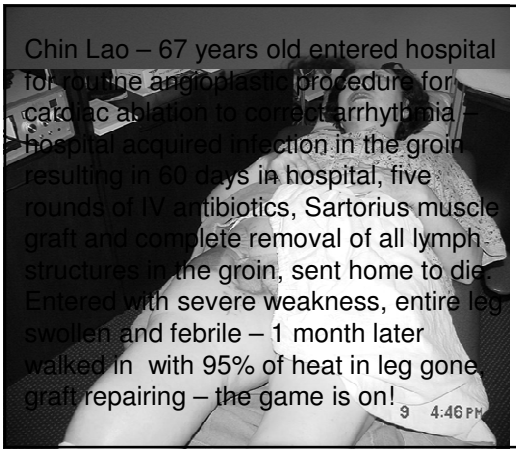
- A key to supporting immune function is modulation of the PRR.
- A number of phytochemicals have already been shown to affect PRR function and signaling.

PRR activation requires dimer formation (homodimers and heterodimers)

Recent studies show curcumin, sulforaphane and cinnamaldehyde are able to reduce PRR dimerization and reduce TLR signaling.

Anything's possible …





Protocol – MRSA

- Increase minerals to reduce acidosis:
  - Calcium Lactate (6) or Powder (2 tsp)
  - Magnesium Lactate (4)
  - Organic Minerals (6)
- Increase immune response with Sequential Immune Up-regulation:
  - Immuplex (6)
  - Sesame Seed Oil Perles (6)
  - Thymex (10)
  - Cyruta Plus (6)
  - Andrographis (6)
  - Congaplex (12)
  - Allerplex (12)
  - Gut Flora Complex (4)
  - Whey Pro Complete with Colostrum (IgG)
  - Chaparral
- Reduce inflammatory and Immune burden:
  - Remove food allergies
  - Reduce cortisol with low glycemic diet

Protocol – Shingles

- Increase serum calcium to reduce herpetic viral activity:
  - Calamo (6) – up to 16 daily for acute intervention
  - Calcium Lactate (6) or Powder (2 tsp)
  - Cataplex F tablets (6) – 16 daily for acute
- Increase immune response:
  - Immuplex (6) – 16 daily for acute
  - Sesame Seed Oil Perles (6) – 16 daily for acute
  - Thymex (10) for background immune bolstering
  - Cyruta Plus (6) -12 daily for acute
  - Andrographis (6)
- Promote amino acid availability and increase lysine:
  - Protefood (6)
  - Whey Pro Complete (2 Tbsp) includes colostrum &IgG
- Reduce anxiety and adrenal stress:
  - Minchex (6)
  - Drenamin (6)
- Neutralize acidosis:
  - SP Greenfood (6)
  - Organically Bound Minerals (6)

Immunosenescence

Immune aging – described as a loss of immune reserve and a loss of immune discretion at the same time.

Some immune features decline while others increase (antibody production and inflammatory upregulation).

These changes increase susceptibility to infectious disease, poor immunization response, cancers, autoimmune dysregulation.

Increased inflammation is called 'inflamaging'.

Factors determining rate of immune decline:

- High cortisol to DHEA ratio
- HGH reduction
- Thyroid decline
- Antioxidant depletion
- Immune burdening
- Stress
- Sleep loss of quality/quantity
- Diet (Elevated HgA1C)
- Exercise reduction

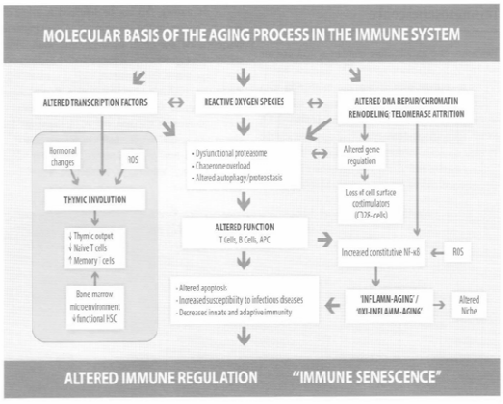


Figure 5: The Mechanisms of Immunosenescence. ROS- reactive oxygen species. HSC- hematopoietic stem cells. Adapted from *Antioxid Redox Signal.* Apr 15, 2011; 14(8): 1551–1585.

Factors affecting aging

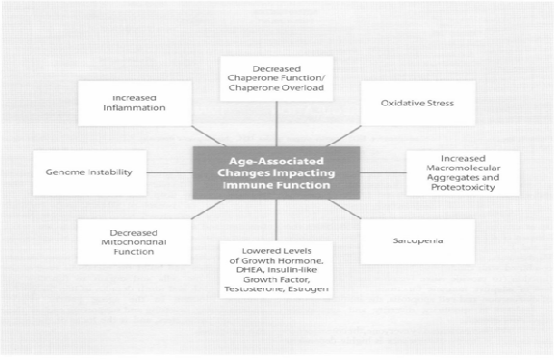
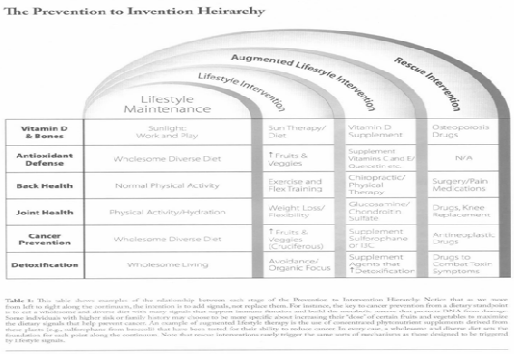


Figure 8: Age-Associated Factors that drive Immunosenescence. Adapted from *Antioxid Redox Signal.* Apr 15, 2011; 14(8): 1551–1585.

Guide to Longlife



Aging and thinking …

When you are 18 years old you worry about what others are thinking about you.

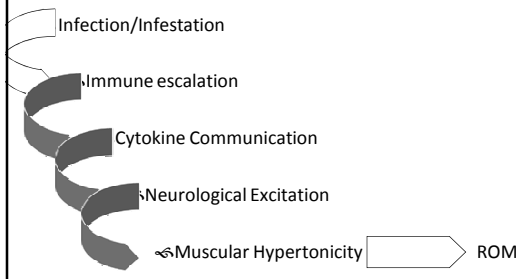
When you are 40 you don't care what others are thinking about you.

When you are 60 you realize that no one is thinking about you at all

Indicators – Measure, Measure

- Indicator testing can be important in the determination of what to do and documenting improvement.
- Common Indicators:
  - Range of motion –
  - Pain/Discomfort -
  - Isolated muscular weakness -
  - Postural assessment -

Indicators – Measure, Measure



Indicators – Range of Motion

- ROM –
- Touch the toes – bend at the waist (Hamstrings)
- Hip Rotation – Internal (Psoas tension)
- Shoulder Extension / Rotation – most mobile joint in the body is most revealing of neurological tone modulation
- Spinal Mobility – Lateral Flexion / Rotation
- Wrist – Extension / Flexion

Fungal / Yeast Overgrowth

- Fungus / Yeast / Mold – normally occurring in every person
  - - It is controlled by immune action and maintained in a diminished population
  - - Immune decline and environmental distortion allows for overgrowth
  - - Overgrowth results in competition for nutrients and gut wall invasion and degradation and subsequent immune burden resulting in degeneration and disease
  - - The gut health is directly tied to the degree of fungal and yeast overgrowth

Fungal and mold toxins

- Mycotoxins – all fungal and candida species express mycotoxins to defend themselves
- Mycotoxins are highly inflammatory to the immune system which results in cytokine expression and subsequent inflammation and neurological excitation
- Part of reducing the impact of Fungus must be the modulation of immune response - to learn to live with and not react to fungus
- Candida Albicans – known to release 79 mycotoxins including gliotoxins and acetaldehyde (alcohol) – it prefers to attack nervous tissue and muscle – as the colonization increases it depletes the body of B1 Thiamine essential for Acetylcholine synthesis and brain function and B3 Niacin essential to Krebs and energy

Experts say -

- "You can't smell it. You can't see it. It's mold poisoning … it's happening all over America and we are just beginning to figure out the detail"
- Dr. Mercola
- "Mold illness may be the most prominent health problem physicians are missing today – a hidden pandemic that's sweeping the nation."
- Dr. Mehmet Oz
- "Severe reactions may occur among workers exposed to large amounts of molds in occupational settings."
- Center for Disease Control CDC

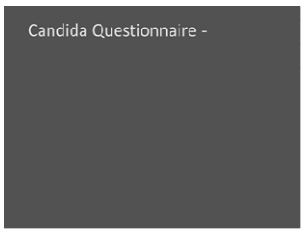
Mold toxin impact on tissue -

- Nerves – Rapid discharge resulting from cell membrane destabilization (depolarizes to easily) resulting in global hyper excitation – peripheral hyper reflexia, tingling/burning/tremor
- Brain – noise, brain fog, focus/memory/cognition/anxiety/dyslexia, seizures, vestibular balance, coordination, ADD, OCD, inflammation
- Muscles – low metabolic energy hence hypertonicity (it takes energy to maintain relaxation – eg rigor mortis)
- Immune System – Hyper-reactivity results in allergies, inflammaging, autoimmunity and depression over time – mast cells release more histamine resulting in allergies, welts, rash, rosacea, inflammation

Conditions related to mold

- Acne
- ADHD / Cognition
- ALS
- Dementia
- Anxiety
- Balance
- Carpal Tunnel
- Coordination
- Dandruff
- Dyskinesia / dystonia
- EMF Sensitivity
- Eczema
- Graves
- Loss of hearing
- Hives/welts
- Hyperacusis (sound)
- Inflammation
- Lyme
- Morning stiffness
- Neuropathy
- MS
- Pain increase
- Panic disorder
- Rosacea
- RA spectrum
- Insomnia
- Nightmares
- Spasticity
- RSD or CRPS
- TMJ
- Tremor
- Trigger points
- Vaccine injury
- Graying hair
- Hair loss/ thinning
- Friable skin
- Memory/dementia
- Plasticity
- Flexibility

Candida Questionnaire -



Candida Purification 90 Day Diet

Gut Flora Complex

Anise (*Pimpinella anisum*) fruit essential oil	125 mg
Andrographis ariel parts 10:1 extract from *Andrographis paniculata* ariel parts 1.0 g Containing andrographolide 10 mg	100 mg
Phellodendron stem bark 20:1 extract from *Phellodendron amurense* stem bark 1.6 g Containing berberine 36 mg	80 mg
Oregano (*Origanum vulgare*) leaf essential oil	75 mg

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Test for Mold Burden - using Gut Flora Complex

Puncture a perle of GFC and smear on the inside of the wrists and then rub them together	Threaten the mold
Give the body about 2-3 minutes for the mold to perceive the oregano oil and other antifungal compounds and begin to defend itself by releasing mold toxins	Immune reaction occurs
Observe that the ROM and muscular tonicity is reduced and tight with various actions	Limitation proves mold burden
Give orally 1-2 Turmeric Forte and immediately retest the ROM and muscular status	Proves immune modulation

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Important Herbs

Herbal Interventions

Rationale:

Herbal extracts and combinations are formulated to promote gastrointestinal health by fending off microbial challenges and burdens (such as parasites, fungal/yeast overgrowth, dysbiosis, stealth, etc.)

Formulations includes a blend of botanicals and plant compounds that have antimicrobial and anti-parasitic properties.

The standard American lifestyle that consists of poor diet, high stress and over consumption of refined carbs and sugars leads to an opportunistic environment for microbes and parasites.

Important Antiparasitic Herbs

- Artemisin
- Myrrh
- Stemona
- Wormwood
- Cloves
- Tannin herbs

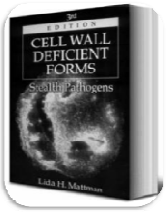
Defining Stealth Pathogens

Stealth Pathogens

Possible first reference to the term 'stealth pathogens' was by Radolf in 1994[1]

"...The syphilis and Lyme-disease spirochetes... persist in their hosts and have strategies for immune evasion..."

However, probably mainly popularized by the late Lida Mattman in her book of that subtitle, first published in 1974[2]



1. Radolf JD. *Trends Microbiol* 1994; **2**(9): 307-311
2. Mattman LH. *Cell Wall Deficient Forms: Stealth Pathogens* 3rd edn 2000; CRC Press, Boca Raton

What are Stealth Pathogens?

- Strict Mattman definition: cell-wall-deficient forms of bacteria (L forms or mollicutes) that can infect intracellularly
- Includes Borrelia and other spirochetes, Proteus, Rickettsia, Mycobacteria, Chlamydophila, Helicobacter, Streptococcus and Bartonella
- But now other organisms are (rightly) mentioned in this context

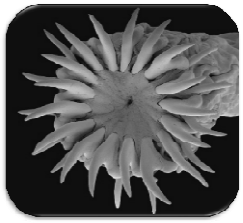
Stealth Pathogens and Chronic Diseases

- Includes autoimmune diseases, cancer, chronic fatigue syndrome (CFS), fibromyalgia syndrome (FMS), chronic Lyme and sarcoidosis
- Can be due to adverse consequences of immune camouflage (molecular mimicry)
- Or otherwise via secreted toxins, microbial waste products, host cell necrosis (danger signals) or by driving chronic inflammation

Stealth Pathogens: A Wider Landscape

Most pathogens employ stealth mechanisms

- Helminths
- Protozoa
- Mycoplasma
- Normal forms of bacteria
- Even viruses



The Original Stealth Pathogen

"*Treponema pallidum*, the syphilis spirochete: making a living as a stealth pathogen"

- Has a poorly antigenic and non-inflammatory surface
- New evidence that antigenic variation also contributes to its ability to evade host defenses
- Silently destroys internal organs in its tertiary phase

Radolf JD, Deka RK, Anand A et al. *Nat Rev Microbiol*. 2016; **14**(12): 744-759. PMID: 27721440

Radolf JD, Desrosiers DC. *Mol Microbiol*. 2009; **72**(5): 1081-1086. PMID: 19432802

Stealth Pathogens' Strategies

Viral Strategies

- Immune evasion: a critical strategy that many viruses have evolved to ensure survival in hosts
- *Herpesviridae* family: reduce cell surface MHC-I molecules[1]
- Epstein-Barr virus (EBV): also appears able to selectively methylate and silence regions of its genome to enable viral persistence[2]

1. Huang T, Osterrieder N. *Oncotarget.* 2015; **6**(26): 21761-21762. PMID: 26335280

2. Tao Q, Robertson KD. *Clin Immunol.* 2003; **109**(1): 53-63. PMID: 14585276.

Helminth Strategies

- Hookworms systematically suppress host immune response
- "Achieved via molecules that assist the parasite in stealthy evasion of the host immune response"
- Secrete factors that promote development of regulatory T cells that inhibit the host inflammatory response

Pearson MS, Tribolet L, Cantacessi C et al. *J Allergy Clin Immunol.* 2012; **130**(1): 13-21. PMID: 22742835

Stealth Pathogens: A New Definition

"Any pathogenic micro-organism employing strategies to persist in the body by hiding from, evading, misdirecting or even suppressing immune responses, leading to chronic disease or lack of well being."

Stealth Bioburden

- Studies have shown that more than one stealth organism might be driving chronic disease at any one time
- For example, a boy with an encephalopathy and autoantibodies: had antibodies against *Mycoplasma pneumoniae* and human herpesvirus 7 in cerebrospinal fluid[1]
- Suggests a role for a broad spectrum approach aimed across all types of pathogens, including viruses
- Highlights limitations of specific pathogen testing

1. Venâncio P, Brito MJ, Pereira G et al. *Pediatr Infect Dis J.* 2014; **33**(8): 882-883. PMID: 25222311

Artemisinin

- support normal flushing of natural toxins from the body
- encourage healthy function of organs of elimination
- cleanse the blood
- encourage healthy bowel function
- support healthy digestion
- support a healthy intestinal environment*

Qing Hao
Artemisia annua

A Brief History

- The 2015 Nobel Prize for Medicine was shared by Tu You You for her contribution to the discovery of artemisinin (qinghaosu)
- Use of *A. annua* dates back to 168 BCE
- Was used for periodic fevers
- Traditional methods of preparation probably optimised artemisinin extraction

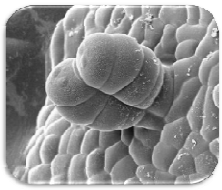
de Ridder S, van der Kooy F, Verpoorte R. *J Ethnopharmacol.* 2008; **120**(3): 302-314. PMID: 18977424

Basic Phytochemistry

Main artemisinin-related compounds in *A. annua* are artemisinin (ART), arteanniun B and artemisinic acid

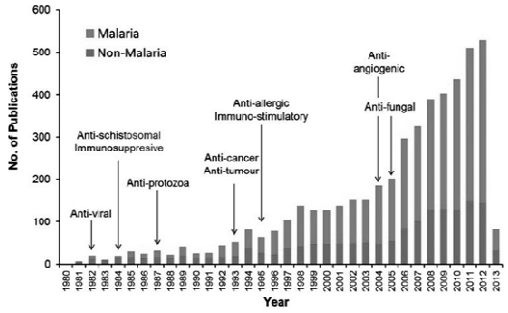
Up to 42% of ART found in the upper leaves, in the glandular trichomes

Also contains flavonoids and essential oil



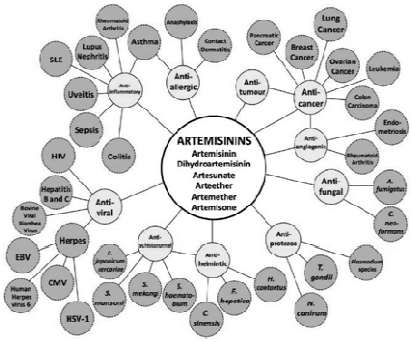
de Ridder S, van der Kooy F, Verpoorte R. *J Ethnopharmacol.* 2008; **120**(3): 302-314. PMID: 18977424

Not Just Malaria



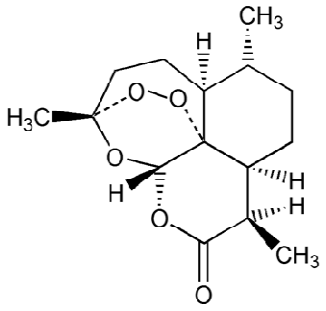
Ho WE, Peh HY, Chan TK et al. *Pharmacol Ther*. 2014; **142**(1): 126-139. PMID: 24316259

Not Just Malaria



Ho WE, Peh HY, Chan TK et al. *Pharmacol Ther*. 2014; **142**(1): 126-139. PMID: 24316259

Artemisinin Chemical Structure



Ho WE, Peh HY, Chan TK et al. *Pharmacol Ther*. 2014; **142**(1): 126-139. PMID: 24316259

Myrrh

- support healthy bowel function
- support healthy digestion
- support a healthy intestinal environment
- support gastrointestinal health
- support sinus and respiratory health*

Myrrh
Commiphora myrrha

The greatest use of your time

Think New Thoughts

Change the world

It wants to

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