
The Chemistry of Pain


After a two-year loan to the United States,
Michelangelo's David is being returned to Italy


Leucine
How to prevent sarcopenia

There are 5 main neurological sensory receptors.

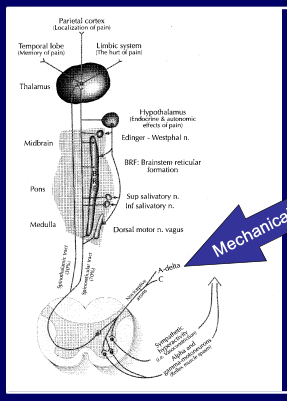
1. Mechanoreceptors
2. Nociceptors
3. Thermoreceptors
4. Chemoreceptors
5. Photoreceptors

Nociception refers to the reception of signals in the CNS evoked by activation of specialised sensory receptors that provide information about tissue damage.

Not all noxious stimuli that activate nociceptors are necessarily experienced as **pain.**

Noci comes from the Latin **"Nocere"** which means to injure. Injury is damage inflicted to the body by an external force.

Nociceptor pathways



Mechanical and Chemical irritation

From Chiropractic and Pain control. Drs systems

Mechanical pain varies with posture. Chemical pain is continuous.

Nociceptors are located in every tissue except

1. Articular cartilage
2. Inner two thirds of the annulus fibrosus
3. Nucleus pulposus
4. Brain

Nociceptor activity may result in

1. Sympathetic hyperactivity (vasoconstriction)
2. Reflex muscle spasm
3. Autonomic concommitants
4. Pain

Inflammation is the term given to describe the biological response that occurs as a result of tissue injury.

It is initiated by

1. Trauma
2. Allergic immunological reactions
3. Microbial infections
4. Chemical toxins, toxic metal and ionising radiation
5. Hypoxia

Acute inflammation is the healing process.

It serves to destroy, dilute and wall off the injurious agent but leads to healing by repair and remodelling of damaged tissue.

Chronic inflammation is unresolved acute inflammation. It is always destructive to tissues and is equated with disease.

Inflammation is divided into three stages

1. The acute inflammatory phase (first 72 hours)
2. The repair phase (48 hours to 6 weeks)
3. The remodelling phase.

The common causes of inflammation maybe

1. Trauma
2. Allergic immunological reactions
3. Microbial infections
4. Chemical toxins, toxic metal and ionising radiation
5. Hypoxia

Challenge for Inflammation

1. Strong muscle goes weak when challenged with
   C. Reactive Protein 6x.

1. A weak associated muscle strengthens when challenged with
   C. Reactive Protein 6x.

C-reactive protein is a protein found in the blood, the levels of which rise in response to inflammation. Iis an acute-phase protein. Its physiological role is to bind to phosphocholine expressed on the surface of dead or dying cells (and some types of bacteria) in order to activate the complement system via the C1Q complex.

CRP is synthesized by the liver in response to factors released by **macrophages and fat cells (adipocytes).** It is a member of the pentraxin family of proteins. C-reactive protein was the first pattern recognition receptor (PRR) to be identified.

Treatment

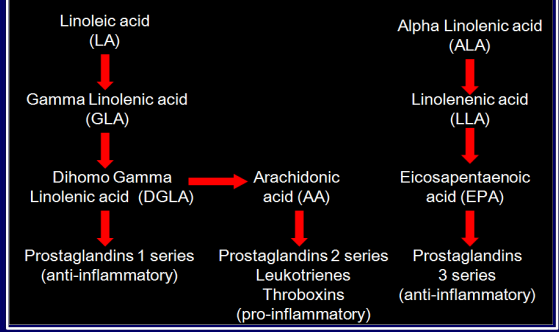
1. Challenge the patient with a positive Therapy Localisation from strength to weakness .

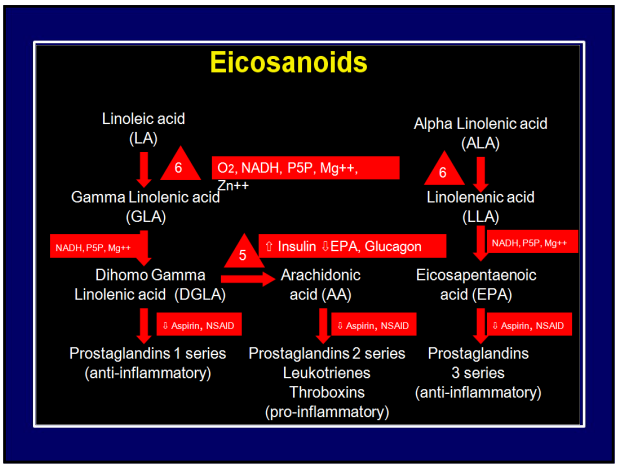
2. Cross challenge the weakness against Histamine 6x, Kinin 6x, Serotonin 6x, Prostaglandins PgE2 6x and Leukotriens B4 6x.

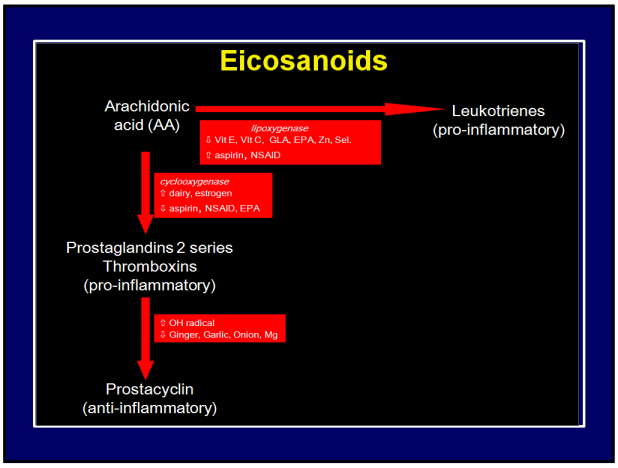
3. Follow the Chemical Mediators of Inflammation chart and identify all negating nutrients, which will aid in the metabolism of the inflammatory mediating chemicals.

CHEMICAL of INFLAMMATION	NUTRITIONAL SUPPORT
Histamine	Zn, Mg, Vit E, Bioflavonoids such as Quercitin, Hesperadin, Turmeric (Curcumin). Vit C
Serotonin (5HT)	Mg, Zn, Vit C, Vit E, EPA, other Antioxidants, Turmeric, Ginger and Bromelain.
(Brady) Kinin	Zn, Bromelain, Hesperadin, Naringen
Prostaglandins PgE2	GLA, EPA, Zn, Mg, B6, Folic Acid, B3, Vit C and Vit A.
Leukotrien B4	GLA, EPA, Vit E, Se, Glutathione, Ginger, Turmeric (Curcumin), Silymarin (Milk thistle) and Parthenium (Feverfew).

Eicosanoids




Eicosanoids
Linoleic acid (LA)
6
O2, NADH, P5P, Mg++, Zn++
Alpha Linolenic acid (ALA)
6
Gamma Linolenic acid (GLA)
Linolenenic acid (LLA)
NADH, P5P, Mg++
5
⇧ Insulin ⇩EPA, Glucagon
NADH, P5P, Mg++
Dihomo Gamma Linolenic acid (DGLA)
Arachidonic acid (AA)
Eicosapentaenoic acid (EPA)
⇩ Aspirin, NSAID
⇩ Aspirin, NSAID
⇩ Aspirin, NSAID
Prostaglandins 1 series (anti-inflammatory)
Prostaglandins 2 series Leukotrienes Throboxins (pro-inflammatory)
Prostaglandins 3 series (anti-inflammatory)


Eicosanoids
Arachidonic acid (AA)
lipoxygenase
⇩ Vit E, Vit C, GLA, EPA, Zn, Sel.
⇧ aspirin, NSAID
Leukotrienes (pro-inflammatory)
cyclooxygenase
⇧ dairy, estrogen
⇩ aspirin, NSAID, EPA
Prostaglandins 2 series Thromboxins (pro-inflammatory)
⇧ OH radical
⇩ Ginger, Garlic, Onion, Mg
Prostacyclin (anti-inflammatory)


Functional Testing

Functional Testing
Pain
Pulse rate (Kola test) / Pupil
Blood pressure
Vital capacity / Peak flow
O2 saturation
Body temperature
EAV / Vega
Range of motion
Leg / Arm length and rotation
Manual muscle testing

Manual muscle testing
is
a diagnostic tool
used to evaluate
the motor response
of the central nervous system to
a sensory challenge.

Check for strong muscles

Identify a strong indicator muscle such as

1. Rectus femoris (SI)
2. Deltoid (Lung)
3. Hamstrings (LI)
4. Subscapularis (Ht)
5. Popliteus (GB)
6. Pectoralis Major Stern (Liv)

7. Teres minor (TW)
8. Gluteus maximus (Cx)
9. Latissimus dorsi (Sp)
10. Psoas major (Kid)
11. Peroneals (BI)
12 Pectoralis major clav (St)

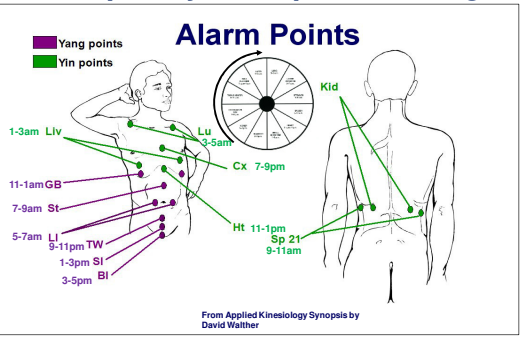
Dosing

From weakness a simulated copy of a nutrient may strengthen.

This will tell you that the nutrient is **effective.**

The exact dose has to be assessed for by the amount of capsules / liquid that exactly negates the weakness.

Frequency and optimal timing



With the remedy on the patient (from strength), cross challenge the **alarm points** for maintaining strength. This / these are the optimal times to prescribe the remedy.
Food supplements are generally St, SI, Cx

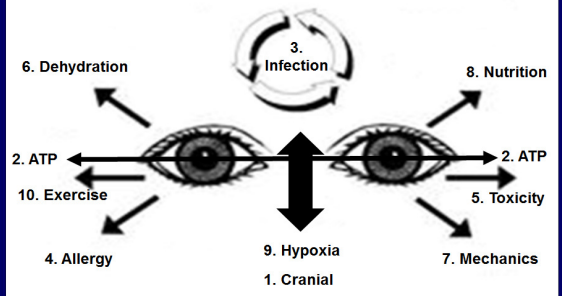
Basically all nutritional supplements should be given with meals.
Oils with the evening meal
Amino acids half an hour before breakfast.
Folic acid, CoQ10 and Probiotics last thing at night.
Herbs in between meals.

Finally always test any nutrient / remedy for tolerance.

Take a strong muscle and challenge the remedy for weakening.
If weakens then the remedy is intolerant and should not be prescribed.

Hypoxia

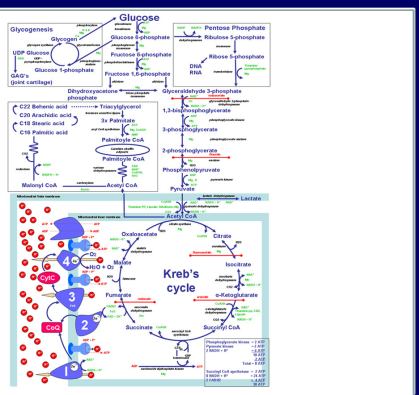
Eyes into Distortion (EID)



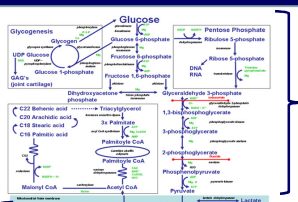
The chemical mediators of PAIN are

- Histamine
- Bradykinin
- Serotonin
- Prostaglandins E2
- Leukotriens B4
- *+ LACTIC ACID* ★

Energy pathway

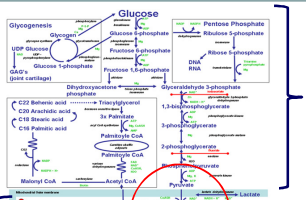


Energy pathway

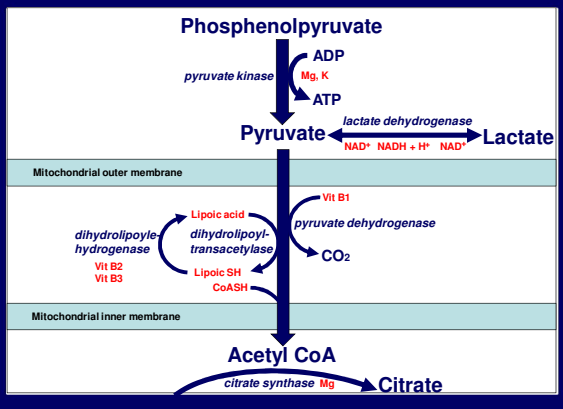


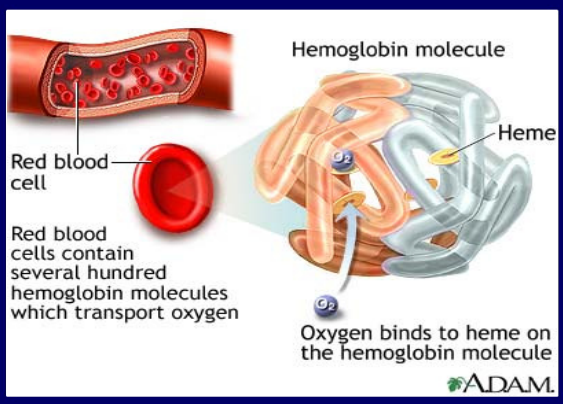
Electron transport or Oxidative phos-phorylation pathway

Energy pathway



Electron transport or Oxidative phos-phorylation pathway

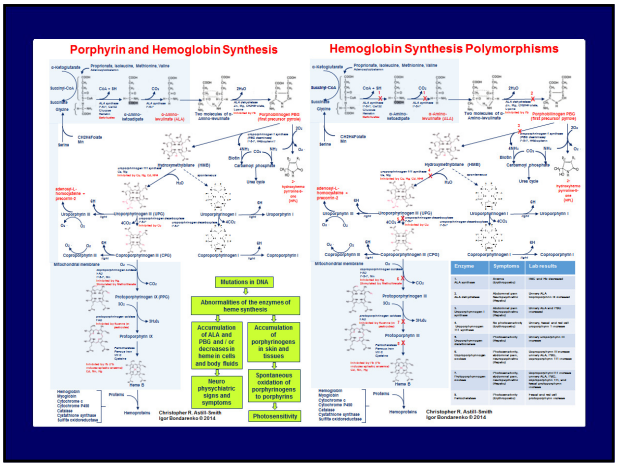




Getting **Oxygen** to the cells requires mature red blood cells containing adequate amounts of haemoglobin. Vital nutrients

Iron, Zinc,
Vit B12's, Vit B2
Folic acid (5MHTF)
Essential fatty acids
Vit A, Vit D and Vitamin B6
Vit C complex
Iodine, Selenium

HYPOXIA	BIOMARKERS O2	NUTRIENTS
OXYGEN	ALA	Glycine, Adenosylcobalamin, P-5-P
	PBG	Mg, Zn, CH2H4Folate
	UPG III 400nm acetate	H4Biopterin, Biotin, P-5-P
	CPG III 400nm acetate	P-5-P. Lutein
	PPG III	Vit B2 (FAD)
	PP IX	Vit B2 (FAD)
	Heme	Vit C, Cysteine, Fe
	Hemoglobin	
	Cell membranes	EFAs
		Vitamin D3
		Turmeric



Thus a **function challenge** for hypoxia would be

A weak muscle strengthens to being challenged with oxygen.

Mechanical assessment

Test Rectus femoris on one side. Should be strong. Tap contralateral knee flexor reflex and retest Rectus femoris. Should become inhibited.

Reverse procedure.

Test contralateral **Latissimus dorsi**. Should become inhibited.

Test ipsilateral **PMS**. Should become inhibited

The therapeutic adjustment involves therapy to

The cranio-sacral mechanism
The TMJ / occlusion
The occiput
The cervical spine
Sterno-clavicular
Acromio-clavicular
The manubrium or xiphi sternum

The thoracic spine
The lumbar spine
The pelvis
The lower extremity
The upper extremity
The rib cage
The sacrum
The coccyx
The pubic symphysis
The viscera

The optimal technique for treating this condition is

1. RESIST
2. Manipulation
3. Cranial
4. Meridian therapy
5. Colour (Miron light) therapy

1. RESIST Technique

(Reflex Extremity and Spinal Inhibitory and Stimulatory Technique)

Identify primary biomechanical dysfunction.

1. Two handed resistance to affected articulation.
2. In spinal lesions patient actively moves all extremity articulations for one minute.
3. In extremity lesions patient actively moves all spinal articulations for one minute.

INFLAMMATION

INFLAMMATION

Alteration to the microcirculation and accumulation of inflammatory cells are the hallmarks of inflammation.

PAIN, REDNESS, OEDEMA, HEAT, LOSS OF USE.

The processes are largely initiated by resident macrophages that secrete the cytokines

TNFα
IL-1
IL-6
IL-8
in response to tissue damage.

These cytokines in turn stimulate neighbouring stromal cells, such as the endothelial cells and fibroblasts, to release mediators that induce dilation of the local microvasculature, and cause permeabilization of capillaries.

The chemical mediators of inflammation are

Histamine

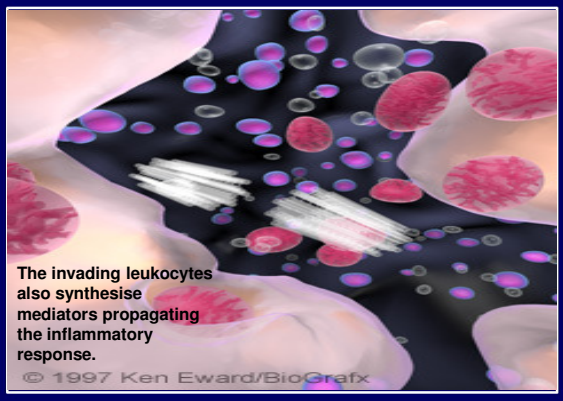
Bradykinin

Serotonin

Prostaglandins E2

Leukotriens B4

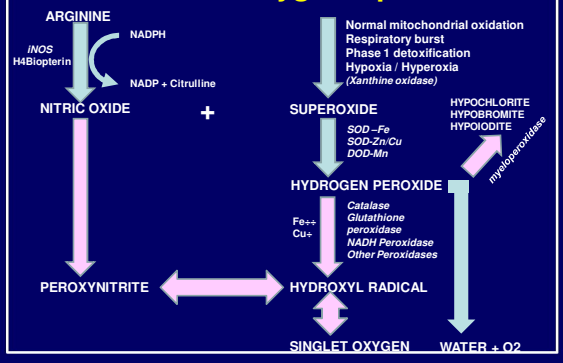
Activated endothelial cells also facilitate the egress of circulating leukocytes into the injured tissue by releasing chemotactic cytokines and lipid products as well as by expressing on their surface adhesion molecules such as ICAM.

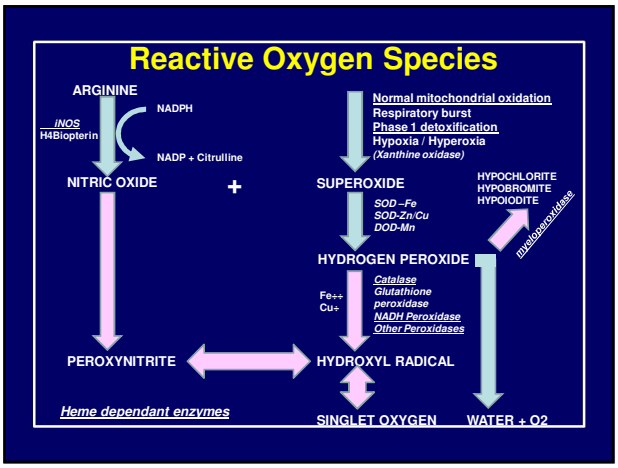


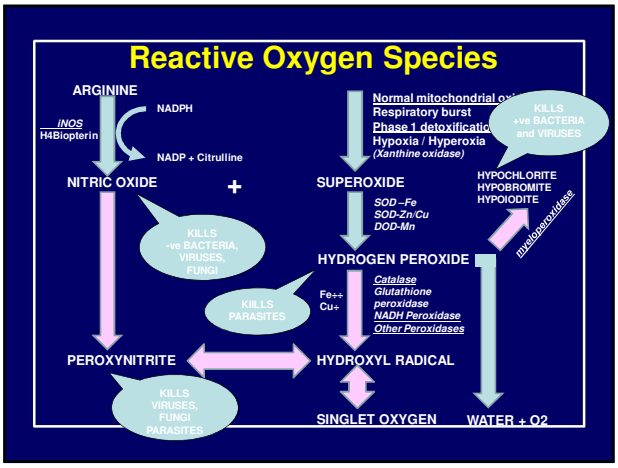
Macrophages as well as other leukocytes, recruited to the site of injury undergo a respiratory burst, producing ROS and proteolytic enzymes.

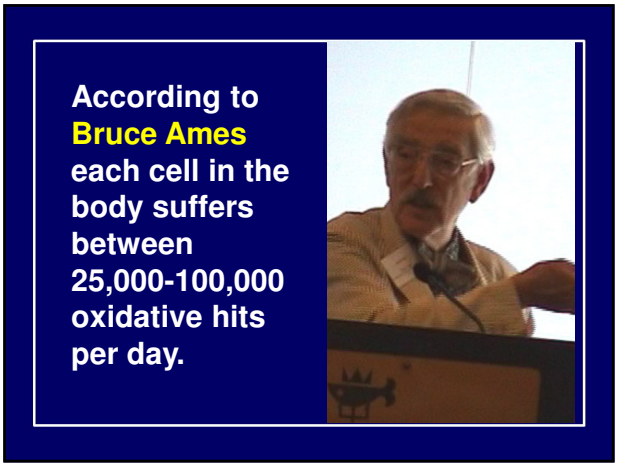
ROS are produced in three ways, each of which involves a specific enzyme.

Reactive Oxygen Species

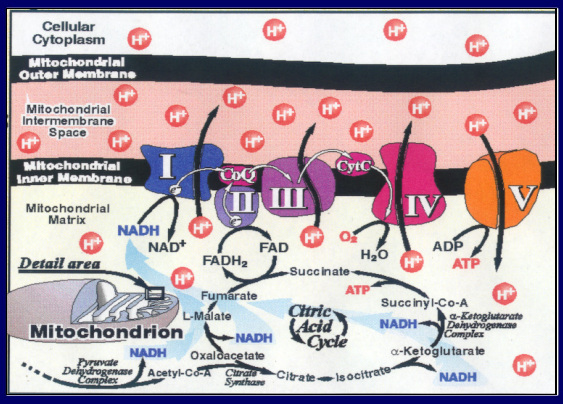


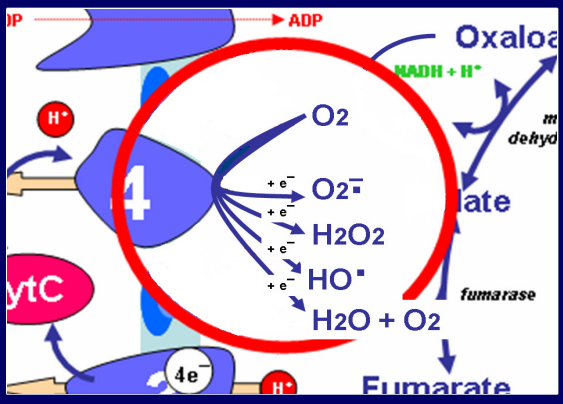

Reactive Oxygen Species
ARGININE
NADPH
iNOS
H4Biopterin
NADP + Citrulline
NITRIC OXIDE
+
Normal mitochondrial oxidation
Respiratory burst
Phase 1 detoxification
Hypoxia / Hyperoxia
(Xanthine oxidase)
SUPEROXIDE
SOD –Fe
SOD-Zn/Cu
DOD-Mn
HYDROGEN PEROXIDE
HYPOCHLORITE
HYPOBROMITE
HYPOIODITE
myeloperoxidase
Fe++
Cu+
Catalase
Glutathione peroxidase
NADH Peroxidase
Other Peroxidases
PEROXYNITRITE
HYDROXYL RADICAL
Heme dependant enzymes
SINGLET OXYGEN
WATER + O2


Reactive Oxygen Species
ARGININE
NADPH
iNOS
H4Biopterin
NADP + Citrulline
NITRIC OXIDE
+
Respiratory burst
Hypoxia / Hyperoxia
(Xanthine oxidase)
KILLS
+ve BACTERIA
and VIRUSES
SUPEROXIDE
HYPOCHLORITE
HYPOBROMITE
HYPOIODITE
SOD –Fe
SOD-Zn/Cu
DOD-Mn
myeloperoxidase
KILLS
-ve BACTERIA,
VIRUSES,
FUNGI
HYDROGEN PEROXIDE
KILLS
PARASITES
Fe++
Cu+
Catalase
Glutathione peroxidase
NADH Peroxidase
Other Peroxidases
PEROXYNITRITE
HYDROXYL RADICAL
KILLS
VIRUSES,
FUNGI
PARASITES
SINGLET OXYGEN
WATER + O2


According to
Bruce Ames
each cell in the body suffers between 25,000-100,000 oxidative hits per day.

This figure is obtained by measuring the quantity of **oxidised deoxyguanosine** in the urine per day and dividing by the number of cells in the body.





1. During the **respiratory burst**, membrane bound NAD(P)H oxidase is activated in both macrophages and granulocytes and produces superoxide anion radicals from molecular oxygen.

Superoxide in turn is reduced to H_2O_2 by SOD which can be further reduced to $OH\cdot$ by the Fenton reaction using Fe++.

2. Macrophages can also generate NO* via the conversion of arginine mediated by inducible nitric oxide synthase.

This reaction is stimulated by allergens, bacterial endotoxin and the cytokines TNFα, IL-1, IL-6, IL-8.

Subsequently nitric oxide and superoxide, both of which are products of activated macrophages, can react with each other, yielding **peroxynitrite anion**, which upon protonation decays to nitrogen dioxide and $OH\cdot$.

3. Granulocytes (large amounts in a single shot), and macrophages (small amounts but more constant), discharge the lysosomal enzyme myeloperoxidase into engulfed extracellular spaces, the phagocytic vacuoles.

Myeloperoxidase catalyses the formation of hypochlorite and other halogen derivatives from H_2O_2 and a halogen molecule.

Hypochlorite anion and the other halogen derivatives can further combine with H_2O_2 to form Singlet oxygen and $OH\cdot$.

Reactive Oxygen Species are necessary to

1. Kill invading bacteria, viruses, fungi and parasites
2. Promote cell growth
3. Promote cell division

Food allergy
and
Lectins

ALLERGY
Coombs and Gell immune inflammatory responses

Type 1
Allergic acute inflammation hypersensitivity is characterised by an allergic reaction that occurs immediately following contact with antigen, which is referred to as the allergen.
Activates on first time exposure to the antigen.
Mediated by IgE. Duration 2-3 days

Type 2
Acute inflammation mediated by cytotoxic antibodies or antibody-dependent cytotoxic hypersensitivity occurs when antibody binds to either self-antigen or foreign antigen on cells, and leads to phagocytosis, killer cell activity or complement-mediated lysis.
Activates on second time exposure.
Mediated by IgG and IgM
Duration 18-21 days

Type 3
Acute inflammation mediated by immune complexes. Hypersensitivity develops when immune complexes are formed in large quantities, or cannot be cleared adequately by the reticulo-endothelial system, leading to serum-sickness type reactions.
Activated on second time exposure.
Mediated by IgG and IgM
Duration 18-21 days

Type 4
Chronic inflammation delayed-type of hypersensitivity reaction (DTH) is most seriously manifested when antigens (for example those of tubercle bacilli) are trapped in a macrophage and cannot be cleared. T cells are then stimulated to elaborate lymphokines, which mediate a range of inflammatory responses.
Mediated by ?
Duration ?

Diagnosis

CHALLENGE from strength or weakness against

IgE for Type 1 (half life of 2-3 days)
IgG for Type 11 and 111 (half life of 21 days)
IgM for Type 11 and 111 (often Lectins show as IgM responses).
IgA (may indicate possible gut parasitic infestation)

Cross challenge against all foods in the FOOD and LECTIN KIT or best to check the patient's own food and drink samples.

Treatment approach

1. Challenge with the weakening food.
2. Cross therapy localise to each B&E point. Usually only one will negate the weakness.
3. Test for most optimal nutrient from the nutrients that synthesise or metabolise the associated neurotransmitter.

Maximising Human Performance

1. Scale of Health

"If 100 is the very best you have ever been in your health, and health being all encompassing i.e. Emotional, Biochemical and Structural, right now your Scale of Health currently calibrates at ……."

2. Epigenetic Scale of Health

"If 100 is the very best your health could possibly be, and health being all encompassing i.e. Emotional, Biochemical and Structural, right now your Epigenetic Scale of Health currently calibrates at ……."

Scale of Vitality

"On a Scale of 1-100 your Vitality as defined as your capacity to generate energy, right now calibrates at ……."

Scale of Fitness

"On a Scale of 1-1000 your degree of physical fitness – as defined by maximum aerobic performance – calibrates at ----"

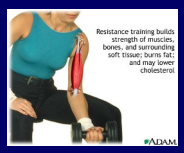
"There is an exercise program that will take your Scale of Fitness from ---- to 1000".

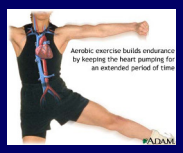
"The optimal form of exercise to accomplish this is -----"

Optimal Exercise

Sprinting
Jogging
Brisk walking
Gentle walking
Swimming
Bicycling
Yoga / Tai chi
Weight training

"The optimal time for performing this exercise per session is -----".





"This exercise needs to be performed ------- times a week"

Maximum Aerobic Pulse

should be

180 – age

10 points lower if less than 3 months regular exercise, recent illness.

20 points lower if any serious health disorders e.g. diabetes

MAP should only be for 1/3rd of exercise time.

1/3rd warming up

1/3rd MAP

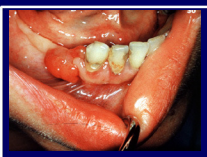
1/3rd warming down

Anaerobic exercise (i.e. pulse rate above 180 – age) should be only performed for a maximum of 3 months without a 3 month break before restarting.

Important to keep a firm aerobic base throughout the year.

Maximising optimal nutritional input in sport

1. Assess nutritional requirements in the clear
2. Assess requirements to complete the target distance.
3. Challenge for affirmation



Tissue repair

Tissue repair starts after the first initial phase of the inflammatory cascade which usually lasts 48-72 hours.

The initial phase is accompanied by **pain** as the same chemicals that drive the acute inflammatory process also sensitise the nociceptors.

The second phase of inflammation is called the **repair** phase and commences from the end of the first phase (48-72 hours) and lasts up to 6 weeks.

As the chemicals that drive the acute phase have now subsided so does pain.

So a **reduction in pain** indicates a change from the acute phase to the repair phase.

1. Repair versus Regeneration

All tissues are capable of repair except the teeth but not all are capable of regeneration.
Bone for instance can both repair and regenerate without a scar.

It is initiated by certain cytokines such as TNF-α, IL-1 and IL-6.

The process can be **inhibited by** glucocorticoids, cytotoxic agents, anti-neoplastic agents, anticoagulants, immuno-suppressive agents, penicillamine, female sex hormones and broad spectrum antibiotics.

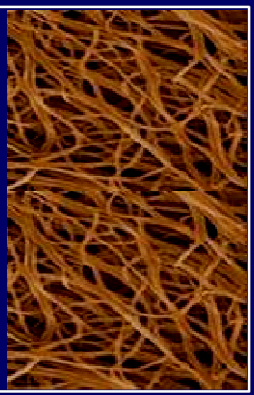
2. Granulation is stimulated by macrophages and covers the entire complex of inflammatory cells such as the fibroblasts which secrete glycoproteins and proteoglycans and endothelia which stimulate new capillary growth.
It is resistant to infection and pain and has a rich blood supply.

3. Angiogenesis is the production of new blood vessels from endothelial cell migration, proliferation and maturation. It is stimulated by hypoxia, the acute inflammatory cytokines and **Vitamin C.**
It is inhibited by shark cartilage, collagen, Irish Moss, genisteine and modified citrus pectins.

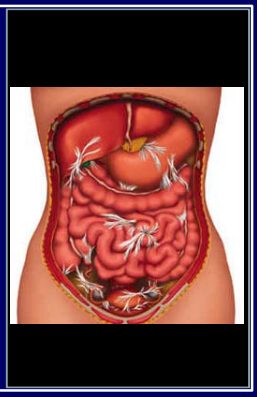
4. Fibroblast activity can only occur if the acute phase has resolved.

They arrive at the site of injury, adhere to the extracellular matrix and synthesise collagen, fibronectin and proteoglycans.

Fibrin is the end product of the coagulation cascade that is formed from fibrinogen. It acts as a scaffolding on which collagen can be deposited.



Excess and haphazard laying down of fibrin and collagen may lead to joint hypomobility, pain and to excess scar tissue (adhesion).



Tissue Remodeling

Dr. George Goodheart

Tissue Remodeling

Collagen

Elastin

Fibrin

The speed of full resolution of an injury depends upon the degree of vascularization of the injured tissue.

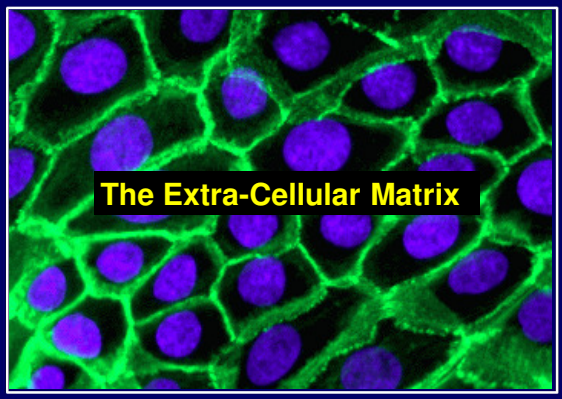
e.g. Ligaments have poor blood supply and may take 6 months to a year.

Muscle has a good blood supply and may take only 6 weeks.

Remodelling is a balance between collagen deposition versus degradation produced by *collagenase* released by fibroblast and macrophages.

Key factor is the restoration of normal range of motion.

Healing musculoskeletal tissues will realign themselves along the lines of stress imposed by movement.



Key Tissues in Degenerative Joint Diseases are

1. Collagen type 1 and 3
2. Elastin
3. Lubricin and Hyaluronic acid
4. Hyalin cartilage

The Steroid Hormones and Body Tissues

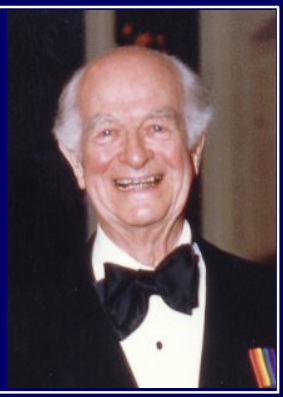
Fat – Estrogen
Muscle – Testosterone
Cartilage - Androstenedione
Bone – DHEA
Ligament – Aldosterone
Elastin - Progesterone

Extracellular matrix consists of

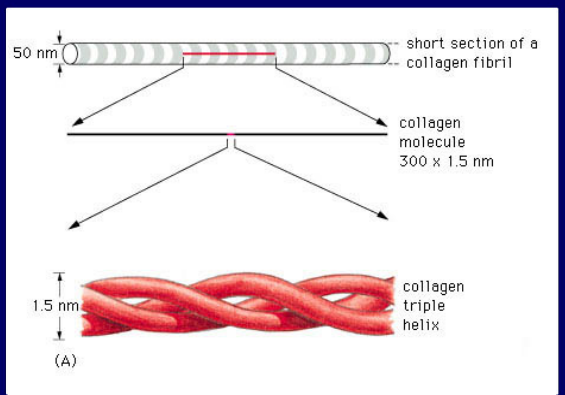
1. Fibrous tissue i.e. collagen (90% of connective tissue)
2. Elastin and Fibrin designed to withstand stretching tensions.
3. Fibrillin, Fibronectin and Laminin, which act as scaffolding for collagen and elastin.
4. Proteoglycans or Ground Substance designed to withstand compression forces.

Collagen Synthesis

Widespread protein: provides tensile strength.



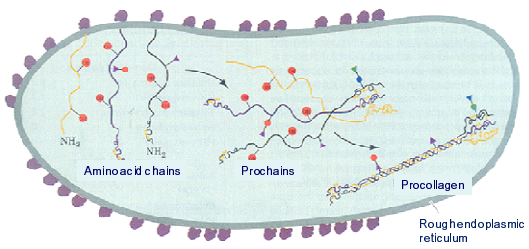
Dr Linus Pauling



COLLAGEN TYPE 1 Most connective tissues including skin, blood vessels, cornea, bone, ligaments and tendons. Thick fibres.
COLLAGEN TYPE 11 Cartilage (?O/A) intervertebral disc, vitreous humor and tendons. Thin fibres.
COLLAGEN TYPE 111 Extensible connective tissue such as skin, lung and the vascular system
COLLAGEN TYPE 1V Basement membranes. Very fine fibres.
COLLAGEN TYPE V Minor component of tissues containing type 1 especially tendons and bone.

COLLAGEN possesses a triple helix structure containing about a 1000 amino acids. Glycine residues occur at every third position (Gly-X-Y).

X and Y can be any other amino acids but 100 or so of the X positions are Proline and a 100 of the Y positions are Hydroxyproline. Most of the other amino acids are either Lysine or Hydroxylysine.



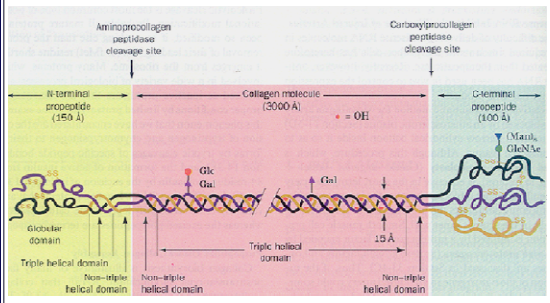
Collagen Production is as follows-

1. DNA transcription of various RNA molecules, which is **Zinc** dependant.
2. RNA instructs amino acids, mainly Glycine, Proline and Lysine to form collagen configurations in the ribosomes.

3. These are then attached to the Endoplamsic Reticulum where they produce single prochains. Proline and Lysine maybe hydroxylated here by prolyl hydroxylase and lysyl hydroxylase co-factored by Alpha keto glutarate, **Iron** and **Vitamin C** **Tetrahydrobiopterin?**

4. These are then glycosylated with glucose or galactose (cofactored by **Manganese** and **Vitamin A**).

5.Three pro-chains combine to form pro-collagen in the Endoplasmic Reticulum. And are then transported to the Golgi Apparatus and then to the plasma membrane. The pro-collagen intra and inter-chains are held together by disulfide bonds formed from PAPs or cysteine or sulfur.



6. Pro-collagen is then released into the extra-cellular space (co-factored by Zinc and Vitamin A)

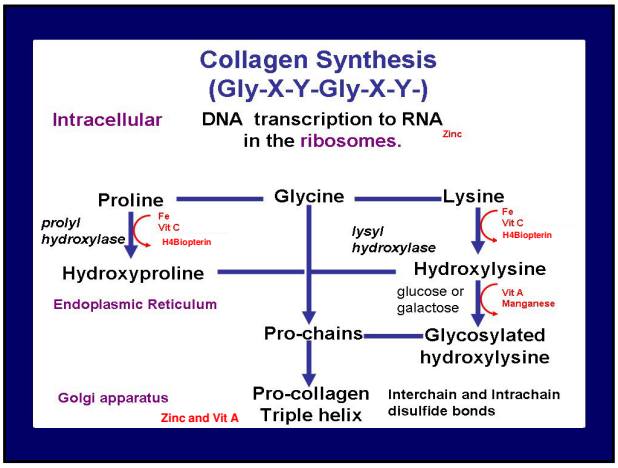
7. Here it is converted to collagen by peptidase enzymes and strengthened by cross-linking of the micro-fibrils by lysyl oxidase, a copper dependant enzyme which is inhibited by high levels of homocysteine (P-5-P and Vit C).

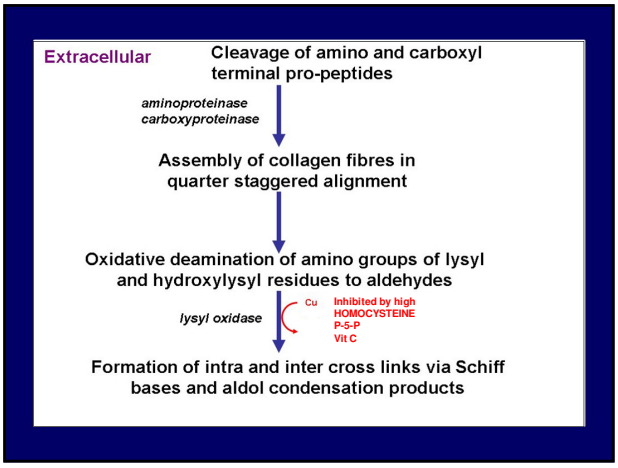
8. Thicker collagen fibres are formed by layers of microfibrils.

9. Joint immobilisation leads to excess cross linking causing adhesion formation.

The bioflavonoid catechin (from Green Tea) stimulates the normal collagen cross linking and so helps prevent adhesion formation especially after surgery.

Other polyphenolic compounds also aid normal collagen cross linking. Such as SMART Vitamin C and the Anthocyanidins such as Bilberry.





Epigenetic Testways - Collagen

Positive sustained challenge –skin tug

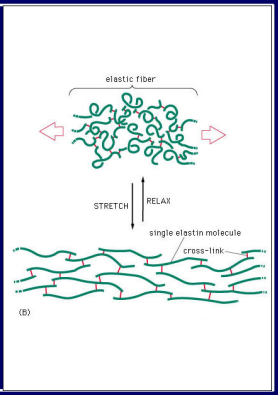
Negated by
Collagen Type 1
Collagen Type 2
Collagen Type 3
Collagen Type 4
Collagen Type 5

Challenge against
Zinc
Proline, Hydroxyproline
Lysine, Hydroxylysine
Glucose, Galactose
Fe, Vit C, Manganese, Vit A
PAP's, Sulfur, MSM
Zinc, Vit A
Cu, Vit B6

Bilberry, Si, (silicia for scarring), Vit E

Elastin

Fibrous protein forming elastic mesh and imparts yellow colour e.g. skin, aorta



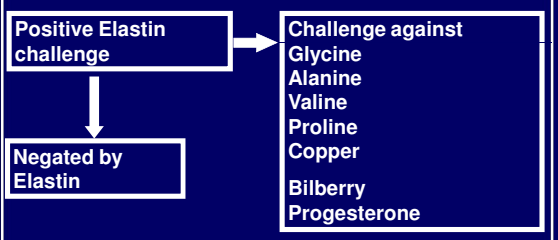
Elastin is a connective tissue protein that possesses elastic recoil properties.

Present in ligament, lung, arteries, skin, ear cartilage

It is 1/3rd Glycine, 1/3rd Alanine + some Valine and Proline.

It contains no hydroxyproline or hydroxylysine.
The covalent cross links are formed by a lysine aldol as in collagen and requires *lysyl oxidase,* the Cu+ dependant enzyme. (Inhibited by high Homocysteine levels).

Epigenetic Testways - Elastin



Skin revitalization
Main skin proteins are
Collagen – Pyridoxal-5-phosphate
Elastin - Methylcobalamin
Fibrin - Adenosylcobalamin
Fibronectin – Folinic acid

Skin revitalization

Main skin proteins are
Collagen – skin stretch challenge
Elastin – skin twang challenge
Fibrin – crease stretch challenge
Fibronectin – skin pressure challenge

Wonder cream

Organic cold pressed peanut oil
Organic cold pressed sesame seed oil
Cetyl alcohol from coconut
Shea butter
Hyaluronic acid
Natural preserver
Vitamin E
Lemongrass aromatherapy oil
Pyridoxal-5-phosp,
Methylcobalamin,
Adenosylcobalamin
Folinic acid

Hyaluronic Acid

Consists of an unbranched chain of repeating disaccharide units containing Glucuronic acid and N. Acetyl Glucosamine.
It is rich in synovial fluid, cartilage, loose connective tissue and the *vitreous body of the eye.*

Synovial fluid is a thick, stringy fluid found in the cavities of synovial joints.

With its egg-like consistency (*synovial* comes from Latin for "egg"), synovial fluid reduces friction between the articular cartilage and other tissues in joints to lubricate and cushion them during movement.

Normal synovial fluid contains 3-4 mg/ml hyaluronic acid). Synovial fluid also contains **lubricin** secreted by synovial cells.
It is chiefly responsible for so-called boundary-layer lubrication, which reduces friction between opposing surfaces of cartilage.

The role of lubricin is not to reduce friction, but rather to protect sliding surfaces from wear by forming a protective barrier between them.
The three constituents of joint fluid, lubricin, hyaluronic acid (HA) and lipids (45% **phosphatidylcholine**), are thought to play a role in mediating the friction incurred by joint motion.

Therapy localise joint

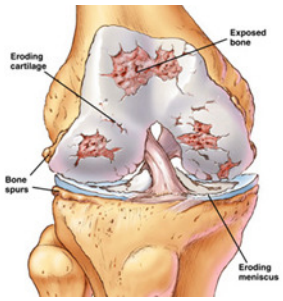
If positive challenge against Synovial fluid, then Hyaluronic acid. Challenge against

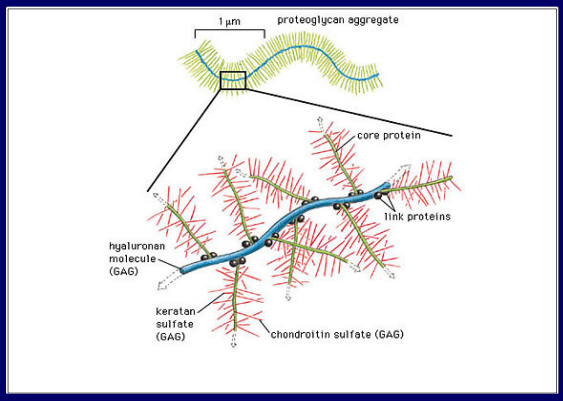
- Glucuronic acid
- N. Acetyl glucosamine
- Hyaluronic acid formulas
- Oils (PC, Omega3)

Crystals found in synovial fluid

1. Cholesterol
2. Monosodium urates
3. Calcium pyrophosphate dihydrate
4. Hydroxyapatite
5. Corticosteroid crystals
6. Calcium oxalate

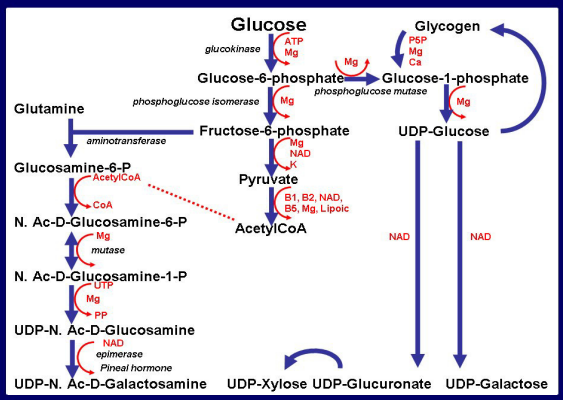
Glycosaminoglycans (GAGs)

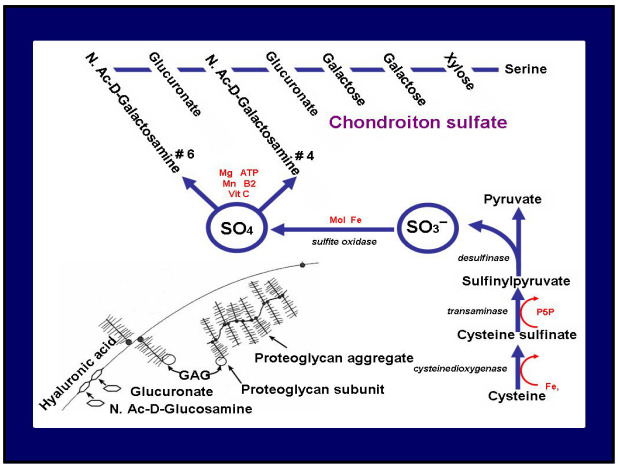


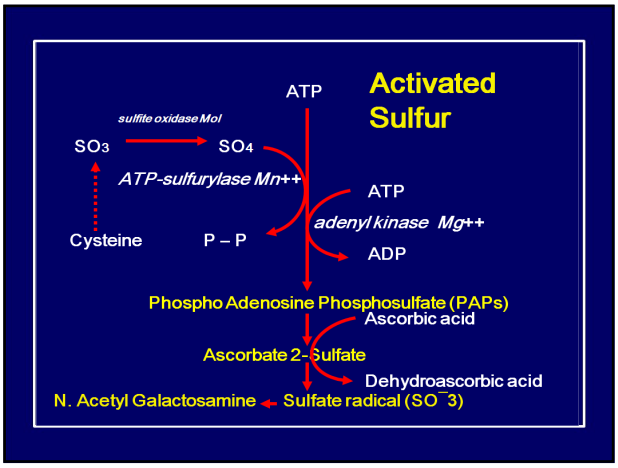


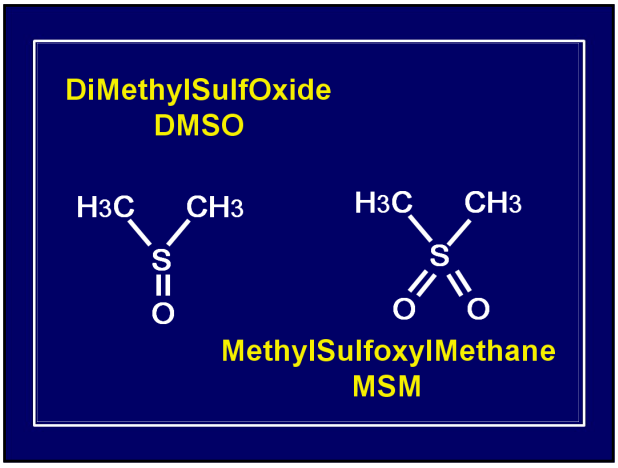
Glycosaminoglycans (GAGs) are various polysaccharides which are linked to a molecule of serine.

Each polysaccharide or sugar molecule is derived from the glycolytic pathway (from glucose) and connected together to make a GAG.




N. Ac-D-Galactosamine
Glucuronate
N. Ac-D-Galactosamine
Glucuronate
Galactose
Galactose
Xylose
Serine
Chondroiton sulfate
6
4
Mg ATP
Mn B2
Vit C
SO4
Mol Fe
sulfite oxidase
SO3−
Pyruvate
desulfinase
Sulfinylpyruvate
transaminase
P5P
Cysteine sulfinate
cysteinedioxygenase
Fe,
Cysteine
Hyaluronic acid
GAG
Glucuronate
N. Ac-D-Glucosamine
Proteoglycan aggregate
Proteoglycan subunit


Activated Sulfur
ATP
sulfite oxidase Mol
SO3
SO4
ATP-sulfurylase Mn++
ATP
adenyl kinase Mg++
Cysteine
P – P
ADP
Phospho Adenosine Phosphosulfate (PAPs)
Ascorbic acid
Ascorbate 2-Sulfate
Dehydroascorbic acid
N. Acetyl Galactosamine
Sulfate radical (SO¯3)


DiMethylSulfOxide
DMSO
H3C
CH3
S
O
H3C
CH3
S
O
O
MethylSulfoxylMethane
MSM

Glycosaminoglycans (GAGs) contain both carboxyl (amino acid) and sulfate radicals which are negatively charged.

They can thus attract cations such as sodium and are therefore designed to retain water up to 1000x their dry weight.

Epigenetic Testways - Arthrosis

Positive Therapy
Localisation to joint or "Grind Test"

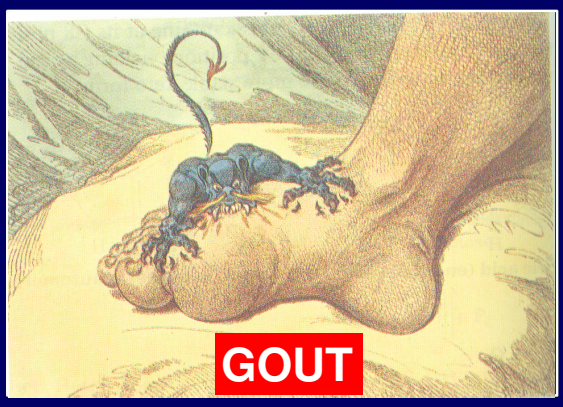
Negated by Chondroiton 4-sulfate or Chondroiton-6 sulfate

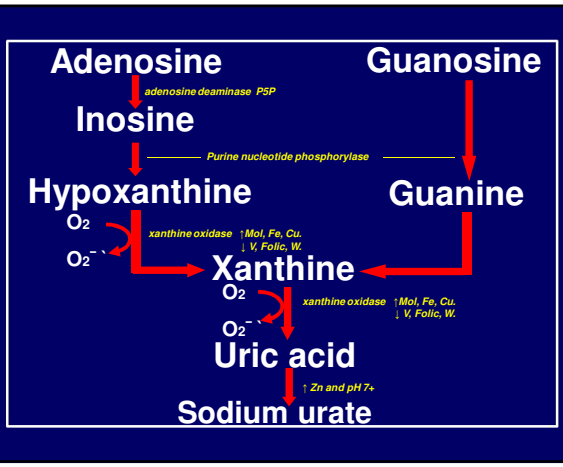
Challenge against
Magnesium
Glutamine
Glucosamine
Acetyl CoA
N.Acetyl-D-Glucosamine
NAD
N.Acetyl-D-Galactosamine
Galactose
Glucuronic acid
Xylose

Sulfur, MSM
Vit C

Manganese, Serine

GOUT





Constant aching, stress, and tenderness in the worst way. Inability to bend, loss of flexibility. Hardness and swelling at the big toe or fingers, wrists ankles and even the knees. Burning sensations and redness around the infected areas. Constant pain.

Purine high foods

Red meats which come from cows or sheep and include steak, chops, corned beef and larger pieces of meat usually roasted in the oven. Game. Meat extracts (e.g Oxo, Bovril). Gravy.
Brains, kidneys, liver & heart (offal), sweetbreads.
Shellfish such as , mussels, oysters and sea eggs.
Anchovies, herrings, mackerel, sardines.
Peas and beans.
Alcohol. especially beer and wine.

Pharmaceutical Treatment

Acute attack – NSAID.
No aspirin

Maintainence

Sulfinpyrazone *Anturan*
(promotes renal excretion)

Xanthine oxidase inhibitor Allopurinol *Zyloric*

Gout *Challenge against Uric acid*

Nutritional and Natural Medicines

- Zinc
- Sodium bicarbonate ★
- Glucosamine
- MSM

- Artichoke (cynara)
- Garlic
- Silymarin (milk thistle)
- Turmeric

Detoxify Cadmium

Gout diet

Grapes – Lowers acidity, Antioxidant

Bananas – Bromelain, Potassium

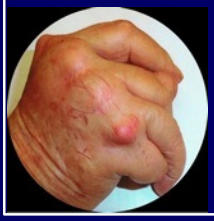
Cherries – Neutralizes uric acid, Anthocyanidins

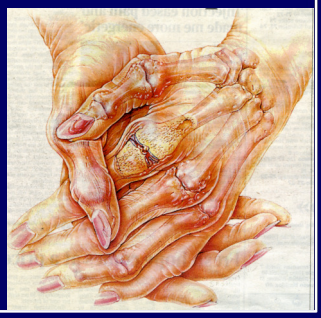
Pineapples
Rich in potassium uric acid - urates
Bromelain – anti-inflammatory
Vitamin C – antioxidant to purines
Folic acid – tissue repair

Blueberries
Potassium
Anthocyanidins
Vitamin C

Strawberries
Anthrocyanidins
Vitamin C
Quercitin inhibits xanthine oxidase

Rheumatoid Arthritis





Rheumatoid Arthritis

Rheumatoid Arthritis (RA) is a chronic, progressive and disabling auto-immune disease affecting 0.8% of the UK adult population. It is an incredibly painful condition, can cause severe disability (this varies between individuals and depends on how severe/aggressive the disease is) and ultimately affects a person's ability to carry out everyday tasks.

The disease can progress very rapidly (again the speed of progression varies widely between individuals), causing swelling and damaging cartilage and bone around the joints.
Any joint may be affected but it is commonly the hands, feet and wrists. It is a systemic, disease which means that it can affect the whole body and internal organs (although this is not the case for everyone with RA) such as the lungs, heart and eyes.

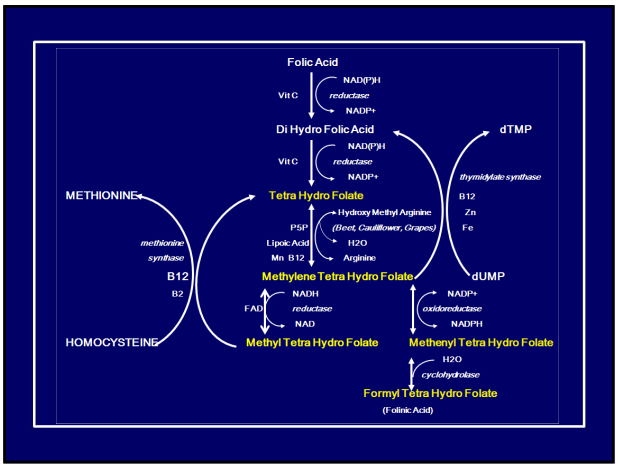
Researchers do know that many factors may contribute to the development of RA. Genetic, or hereditary, factors play a role. Scientists have shown certain genes that play a role in the immune system may be involved in determining whether or not a person may develop RA.

However, some people with RA do not have these particular genes, and other people who do have the genes never develop the disease. Environmental factors may also contribute to the cause of the disease.

Researchers have found that RA can be triggered by an infection, possibly a virus or bacterium, in people who have an inherited tendency for the disease.

Guanidine is the oxidised derivative of guanosine due to decomposition of nucleoproteins in a stagnant intestinal tract and is one of the most potent poisons. Guanidine has a powerful alkaline reaction and is a common finding in arthritis along with a stagnant bowel.

Guanidine stimulates acetylcholine and is a primary fatigue poison. It precipitates calcium from the blood serum Guanidine is eliminated by methylation (betaine or choline, methionine) and conjugation with acetic acid to form creatine.



First get over the alkalosis by neutralizing the guanindine.
Second detoxify the liver.
Third eliminate the constipation so more guanidine is not formed.

Natural treatments for RA

Vitamin A
Vitamin B5
Folic acid
Vitamin C
Vitamin E

Boron
Calcium
Iron
Manganese
Selenium
Silver

Natural treatments for RA

Plant oils

α-Lipoic acid
Turmeric
Cinnamon

Colon cleanse
Digestive enzymes
Prebiotics
Probiotics

Herbs

Ashwagandha (Withania somniferum)
Fennel (Foeniculum valgare)
Ginger (Zingiber officinale