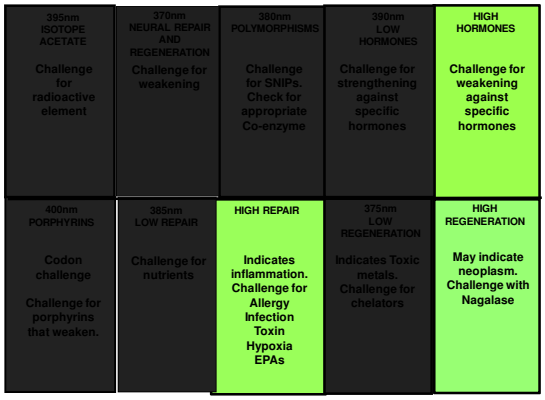
Epigenetic Management of Common Neurological Disorders

Diagnostic Entry Points
Body Type Acetates
Meridian points – Biophoton acetates
Hormones
Therapy localisation
Biomarkers
Weak muscle(s)
Eyes into Distortion
Food allergens
Phonocardiograph

1. **Remove – Allergens, Toxins, Infections**
2. **Replace – Nutrients, Digestive enzymes**
3. **Re-inoculate - Probiotics**
4. **Repair – High or Low**
5. **Regeneration – High or Low**



Causes of Aging

1. Degradation of "cellular timekeepers", known as telomeres.
2. Progressive death over time to the cellular main "power source", the mitochondria.
3. Free radical exposure and resulting oxidative damage (i.e. the "rusting" of the body's cells).

Identifying the Subconscious (Definitive) Meridian

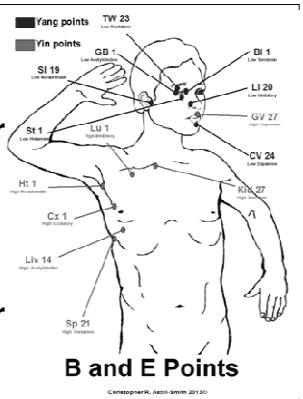
Identifying if there is a Subconscious emotion
Patient TLs Right to Left Amygdala areas and the Left to Right. If either weaken then there is a subconscious meridian.

Identifying the Subconscious meridian
With eyes looking down TL around al the B&E points.

The one that weakens is the subconscious meridian.

Yang points indicate neurotransmitter deficiencies.

Yin points indicate neurotransmitter excesses



B and E Points

Patient TLs positive B&E point(s) (or with subconscious meridian acetate on umbilicus) whilst looking down and practitioner taps Definitive Point 60x at 2 Hz.

Identify weak associated meridian muscle.
Usually just one on the associated meridian.
All other associated muscles will weaken when tested bilaterally.
Use this muscle to test for priority when using any nutritional supplement(s).

Vertigo

Vestibular challenge

1. ROD acetate to challenge for dim vision.

2. Bilateral TL to the temporal bones firstly eyes open and then eyes shut.

3. TL cervical spinous processes.

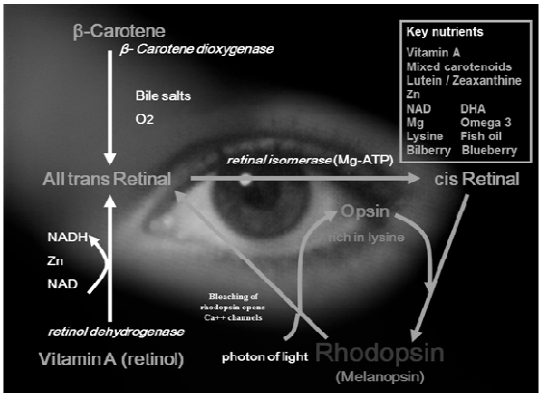
1. Peripheral vision

2. Labyrinthine

3. Cervical mechano-receptors

The ROD acetate is a dark grey acetate placed over the eyes making sure no other light enters through peripheral vision. Turn off any overhead lights. If a strong muscle then weakens indicates a problem with dim vision. The rods in the retina pick up shades of grey light between black and white and the outline of objects.

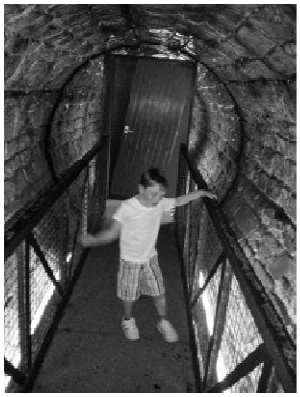
They are located all around the back of the retina and so pick up peripheral vision also. It is this peripheral vision that sends input to the vestibular centres in the brain to aid in maintaining balance.



Vertigo episodes where the world spins around are due to peripheral vision defects. When the person spins round the world it is due to labyrinthine disorders.

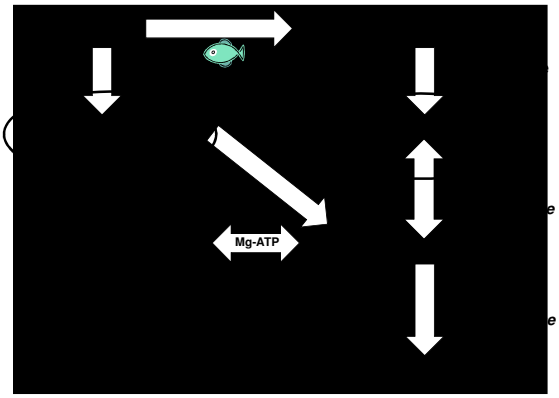
Flambard's Magic Tunnel

Peripheral vision challenge



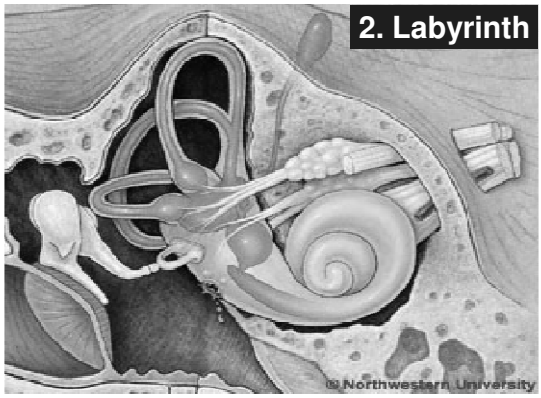
Labyrinthine challenge

Dim vision maybe due to a deficiency in the rod visual pigment rhodopsin.



SMART EYES (Lutein, Zeaxanthine and Bilberry extract) Vitamin A
Lutein / Zeaxanthine
Zinc
Niacin or NAD(H)
Magnesium

Bilberry
DHA
Omega 3 plant oils—Flax, Pumpkin and Walnut



Therapy Localise to both ears or temporal bones.
Test strong indicator muscle.
Should remain strong.
Patient closes eyes and if muscle goes weak then a labyrinthine dysfunction

Challenge for Zinc
Colloidal zinc
Zinc ascorbate
Zinc bisglycinate
Zinc citrate
Zinc picolinate
Triple zinc
Zinc chloride
Zinc sulfate

Challenge for excess Calcium against Calcium phosphate
If positive consider
Magnesium
EFAs
Vitamin K2
P-5-P

Research hub events

The National Council for Osteopathic Research's (NCOR) research hubs provide an informal environment where you can learn more about the evidence underpinning your practice. Their next meetings are:

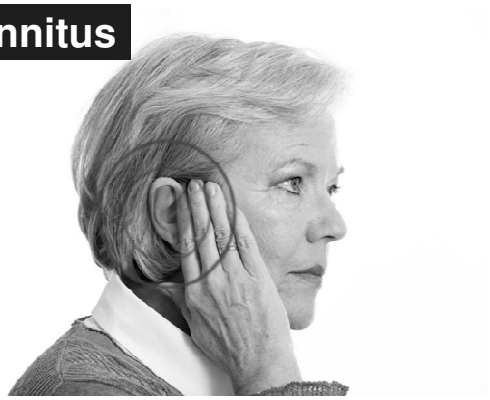
- ***Bristol*** **Thu 10 November**, 7-9pm (managing groin symptoms)
- ***Haywards Heath*** **Wed 7 December**, 7-9pm (vertigo and the Epley manoeuvre)
- ***Leeds*** **Mon 12 December**, 6.30-8.30pm (treatment and management of patients with breast cancer)
- ***Exeter*** **Sat 14 January**, 10am-12noon (intervertebral discs)

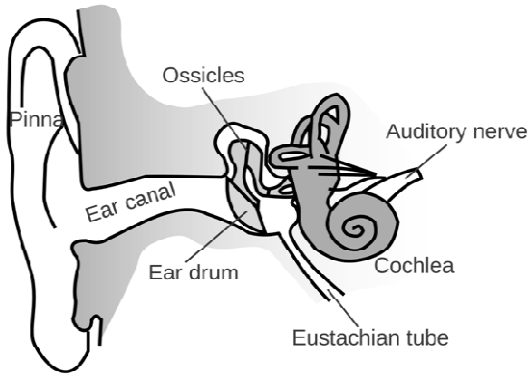
For details or to find out about setting up your own hub, email Carol Fawkes at NCOR. The research papers for discussion will soon be available on the NCOR website – if you cannot attend a hub meeting, why not use the papers as the basis of your own CPD event?

3. Cervical spine
Therapy localise each spinous process for weakening either in the clear or with movement.
If weakening treat accordingly.

Treatment Options
Manipulation / Adjustment
Therapy Localisation Technique
Miron torch
Respiratory adjust

Tinnitus



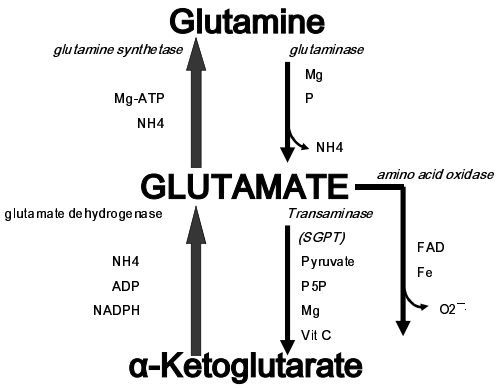


Tinnitus is the hearing of sound when no external sound is present. While often described as a ringing, it may also sound like a clicking, hiss or roaring. Rarely, unclear voices or music are heard.
It maybe continuous, fluctuating or pulsatile.

The sound may be soft or loud, low pitched or high pitched and appear to be coming from one ear or both. Most of the time, it comes on gradually.
In some people, the sound causes depression, anxiety or interferes with concentration.

One of the most common causes is noise-induced hearing loss. Other causes include: ear infections, disease of the heart or blood vessels, Ménière's disease, brain tumours, emotional stress, exposure to certain medications, a previous head injury, earwax and EM from amalgams.

Tinnitus maybe inflammation of the auditory nerve.
The peripheral axons of auditory nerve fibres form synaptic connections with the hair cells of the cochlea via ribbon synapses using the neurotransmitter glutamate.

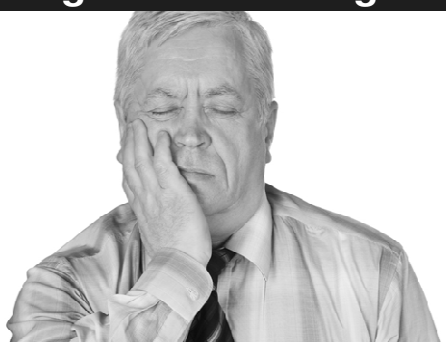


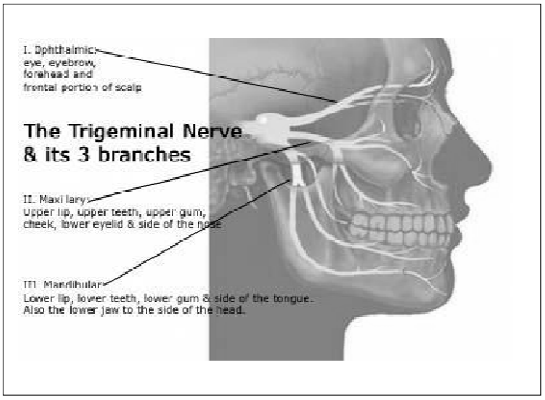
Treatment
Magnesium
EFAs
Vitamin K2

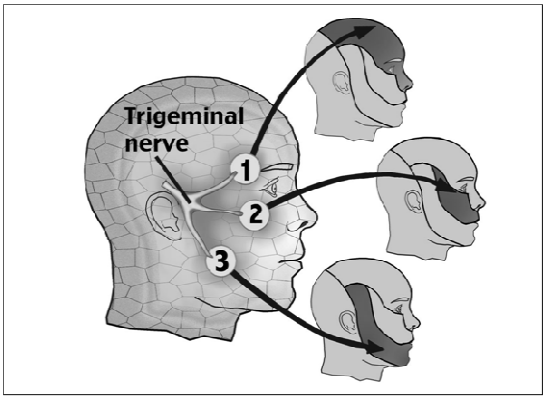
Vitamin B12 for pulsatile tinnitus

Consider amalgam extraction

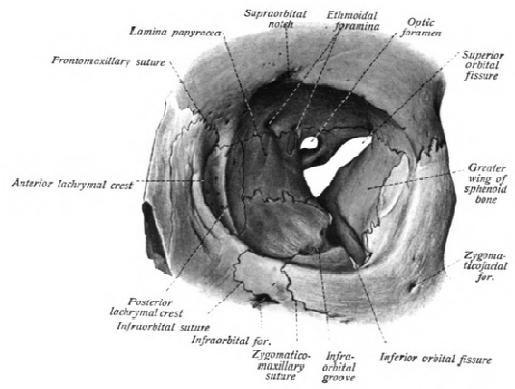
Trigeminal Neuralgia

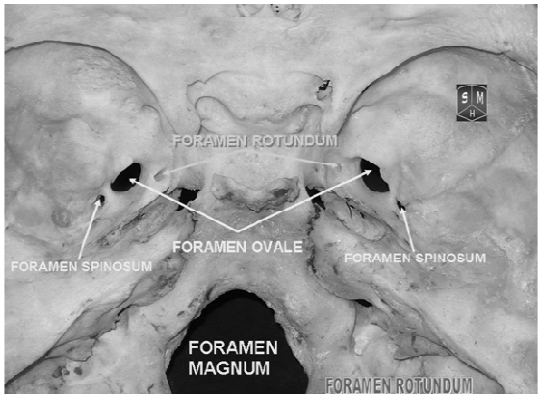






The ophthalmic, maxillary and mandibular branches leave the skull through three separate foramina: the superior orbital fissure, the foramen rotundum and the foramen ovale, respectively.





The sensory function of the trigeminal nerve is to provide tactile, proprioceptive, and nociceptive afference to the face and mouth. Its motor function activates the muscles of mastication, the tensor tympani, tensor veli palatini, mylohyoid and the anterior belly of the digastric.

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

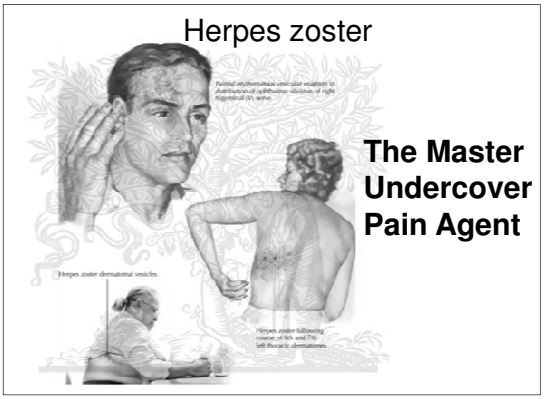
It is initiated by
1. **Trauma**
2. **Allergic immunological reactions**
3. **Microbial infections**
4. **Chemical toxins, toxic metal and ionising radiation**
5. **Hypoxia**

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.

Herpes zoster



The Master Undercover Pain Agent

Treatment for Herpes Zoster virus
Colloidal Silver
Detox 4 and 5
Virus
Herpes zoster 30x
Lymph

Alpha Lipoic acid
Calcium
Folates

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Review

Nature Reviews Immunology 7, 690-702 (September 2007) | doi:10.1038/nri2152

Calcium signalling in lymphocyte activation and disease

Stefan Feske

Calcium signals in cells of the immune system participate in the regulation of cell differentiation, gene transcription and effector functions. An increase in intracellular levels of calcium ions (Ca^{2+}) results from the engagement of immunoreceptors, such as the T-cell receptor, B-cell receptor and Fc receptors, as well as chemokine and co-stimulatory receptors. The major pathway that induces an increase in intracellular Ca^{2+} levels in lymphocytes is through store-operated calcium entry (SOCE) and calcium-release-activated calcium (CRAC) channels. This Review focuses on the role of Ca^{2+} signals in lymphocyte functions, the signalling pathways leading to Ca^{2+} influx, the function of the recently discovered regulators of Ca^{2+} influx (STIM and ORAI), and the relationship between Ca^{2+} signals and diseases of the immune system.

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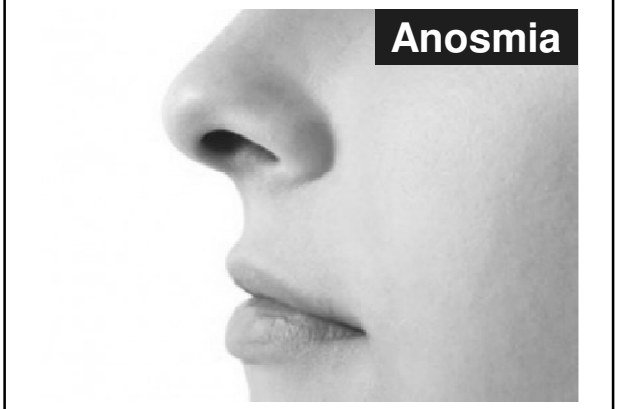
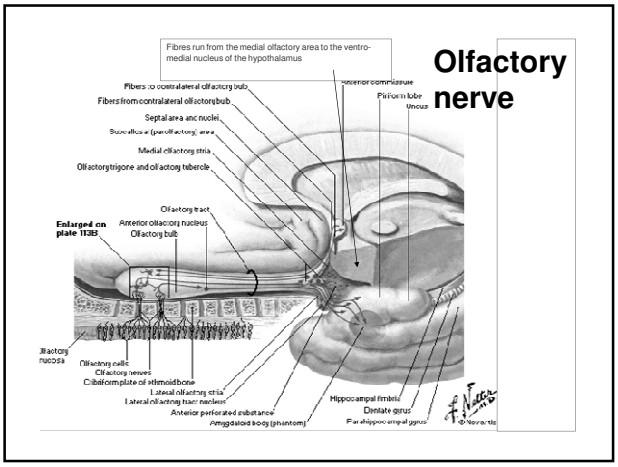




Anosmia is the inability to perceive odour or a lack of functioning olfaction—the loss of the sense of smell.
Anosmia may be temporary, but some anosmia (including traumatic anosmia) can be permanent.

Anosmia is due to a number of factors, including an inflammation of the nasal mucosa, blockage of nasal passages or a destruction of one temporal lobe. Inflammation is due to chronic mucosa changes in the paranasal sinus lining and the middle and superior turbinates.

Ageusia

Ageusia is the loss of taste functions of the tongue, particularly the inability to detect sweetness, sourness, bitterness, saltiness, and umami. It is sometimes confused with anosmia – a loss of the sense of smell..

Because the tongue can only indicate texture and differentiate between sweet, sour, bitter, salty, and umami, most of what is perceived as the sense of taste is actually derived from smell.

True ageusia is relatively rare compared to hypogeusia – a partial loss of taste – and dysgeusia – a distortion or alteration of taste.

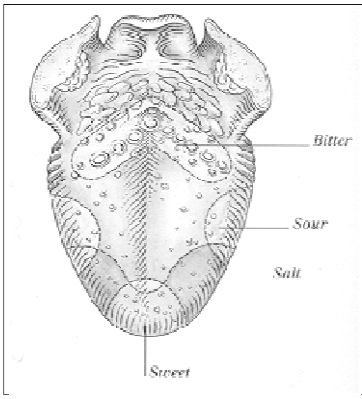
Treatment options
Zinc
Iodine
Vitamin A

Taste

Salt and Sweet at the tip of the tongue.

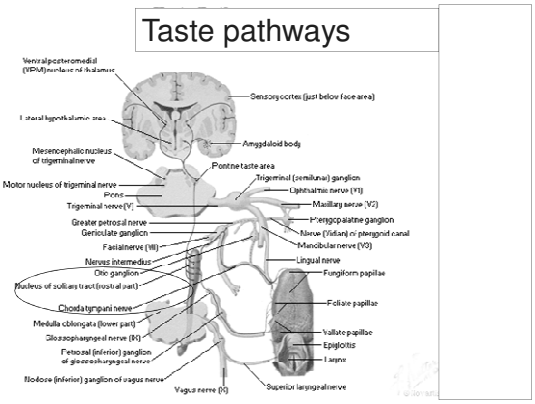
Bitter at the pharyngeal end.

Sour at the sides.



TASTE

Taste pathways



People with taste and smell dysfunction have a decreased level of zinc in parotid saliva and have a poor appetite.

Nasal mucus protein is also a zinc containing protein.

Traditional zinc taste test uses zinc sulfate 0.1% solution.

Best to use Colloidal Zinc for the Zinc Taste Challenge

Common Visual Disturbances

Dr. Mercola and Dr. Wunsch on the Dangers of LED

Near-infrared is important as it primes the cells in your retina for repair and regeneration, which explains why LEDs — which is devoid of infrared — are so harmful for your eyes and health

One-third of the energy your body consumes comes from the food you eat. The vast majority of the energy your body needs to maintain the systemic equilibrium comes from environmental infrared light exposure

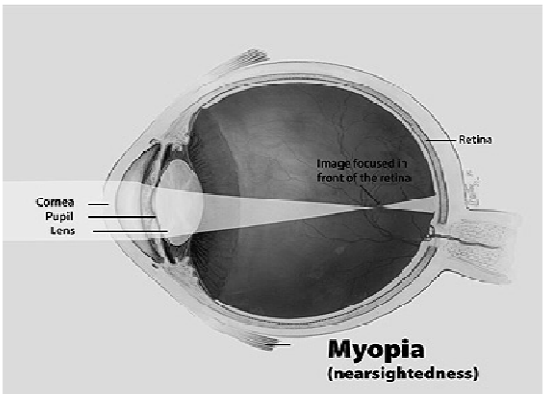
LEDs sabotage health and promote blindness. Limit your exposure to blue light during the daytime and at night. Swap out LEDs for incandescents or low-voltage incandescent halogen lights

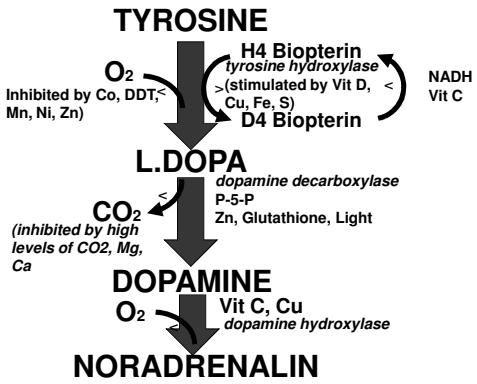
Dim Vision, Poor Peripheral vision, Vertigo –
ROD acetate

SMART EYES (Lutein, Zeaxanthine and Bilberry extract) Vitamin A
Lutein / Zeaxanthine
Zinc
Niacin or NAD(H)
Magnesium

Bilberry
DHA
Omega 3 plant oils—Flax, Pumpkin and Walnut

Myopia = distance challenge
Low Noradrenalin
or High Acetylcholine



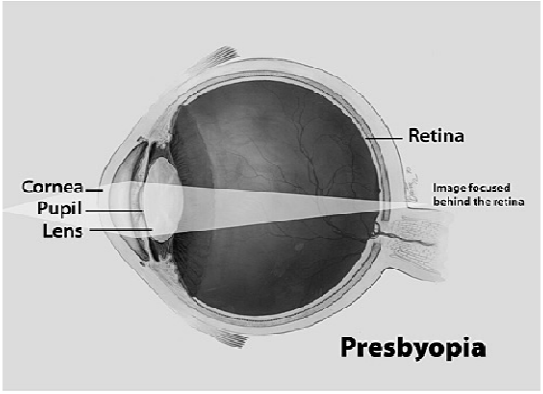


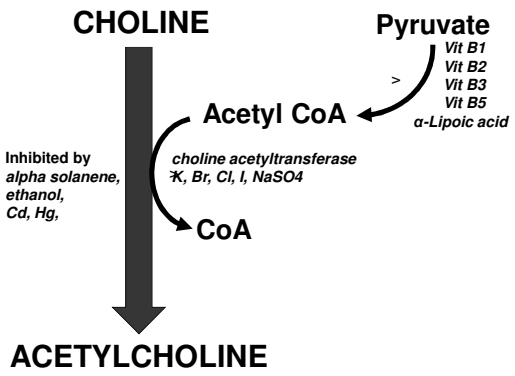
Key nutrients

Vit B12	(Adenosylcobalamin)
Fe++	(Iron citrate or Colloidal iron)
Folic	(Folinic acid or 5MTHF)
Vit B6	(Pyridoxal-5-phosphate)
Vit B3	(NADH)
Zn++	(Zinc picolinate, Colloidal zinc)
Mg++	(Magnesium citrate or Magnesium bisglycinate)
Cu++	(Copper bisglycinate)
Vit C	(Vitamin C or Potassium ascorbate)
DHA	(Omega 3 or DHA)

Presbyopia

Hyperopia = near challenge
Presbyopia = near challenge
negated by bright light
= Low Acetylcholine
or High Noradrenalin





Key nutrients

Choline (Choline bitartrate or Phosphatidylcholine)
Vit B5 (Pantothenic acid or CoA factors)
Vit B1 (Thiamine pyrophosphate)
Mn (Manganese citrate)
DHA (Omega 3 or DHA)

Cataract = weakness at any distance focused

Systemic supplementation

1. N. Acetylcysteine
2. Glutathione – low levels, increased risk due to increased lens membrane permeability
3. L. Lysine

Systemic supplementation

4. Vitamin A
5. Carotenoids
   lutein and zeaxanthine
6. Riboflavin-5-phosphate
7. Vitamin C high in aqueous humor
8. Vitamin E

9. Alpha Lipoic acid
10. Dimethylglycine
11. Copper – retards growth
12. Manganese – retards growth
13. Selenium
14. Zinc

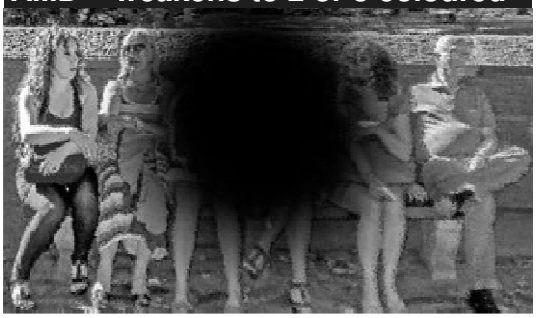
Fatty acids

1. DHA

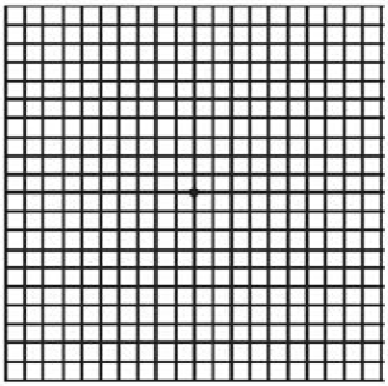
2. Omega 3

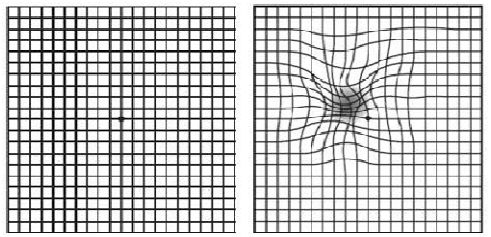
3. Phospholipids

Age related Macular Degeneration
AMD = weakens to 2 or 3 coloured

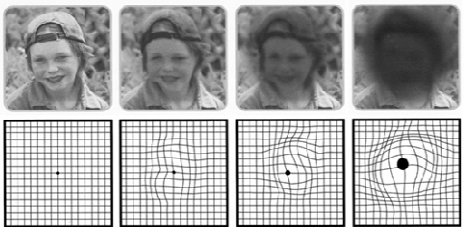


Amsler Grid





Amsler grid viewed with normal vision and with wet AMD.



Age related Macular Degeneration
AMD = weakens to 2 or 3 coloured acetates

Nutritional supplementation

1. Glutathione

Vitamin A

Lutein

Zeaxanthin

They are the only two carotenoid antioxidants to be found in the lens and the central area of the retina, called the macula.

Alpha lipoic acid

Pyridoxal-5-phosphate

Vitamin C - aqueous

Vitamin E – outer segment membranes of photoreceptors

Copper

Selenium – Jonathan Wright combination

Zinc – normally in high concentration in retinal pigment

Bilberry (Reduced capillary fragility, antioxidant and collagen stabilising effects)

Ginkgo biloba – increased visual acuity

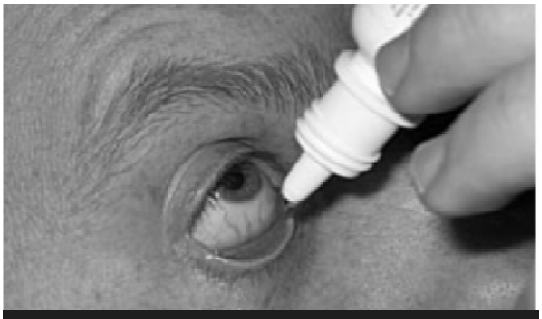
Omega 3 - DHA

Fatty acids

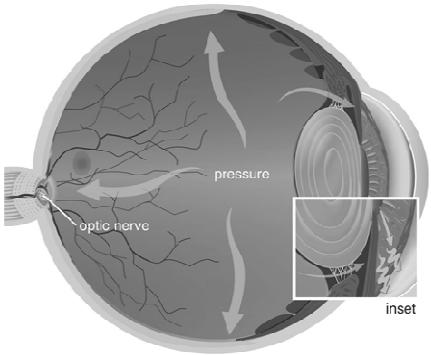
Omega 3

DHA

Phospholipids



Glaucoma = weakens on finger pressure on eyeball



Nutritional intervention

Glutathione – low in the anterior chamber.

Alpha Lipoic acid increased glutathione levels.

Taurine

Thiamine pyrophosphate

Adenosylcobalamin – lesions similar to end stage glaucoma (optic nerve atrophy)

Vitamin C – reduces intra-occular pressure

CoQ10

Chromium

Magnesium – reduces vasospasm

Melatonin – reduces intra-ocular pressure 500mcg in evening

West African Combination

Vitamin A 180,000 IU = 72 drops
Vitamin C 3 gm
Vitamin E 200 IU
Protein 29 gm

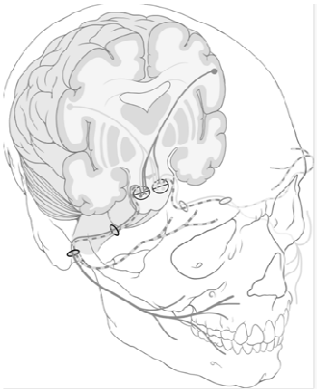
Ocular pressure returns to normal after one week.

Bell's palsy

Sir Charles Bell 1774 – 1842) was a Scottish surgeon, anatomist, neurologist, and philosophical theologian.

Bell's palsy is a type of facial paralysis that results in an inability to control the facial muscles on the affected side. Symptoms can vary from mild to severe. They may include muscle twitching, weakness, or total loss of the ability to move one or rarely both sides of the face.

Other symptoms include drooping of the eyelid, a change in taste, pain around the ear, and increased sensitivity to sound. Typically symptoms come on over 48 hours. It results from a dysfunction of cranial nerve VII (the facial nerve). Many believe that this is due to a viral infection that results in swelling.



The facial nerve's nuclei are in the brainstem (they are represented in the diagram as a „θ"). Orange: nerves coming from the left hemisphere of the brain. Yellow: nerves coming from the right hemisphere of the brain. Note that the forehead muscles receive innervation from both hemispheres of the brain (represented in yellow and orange).

Pathological Nervous System Disorders

All the common pathological neurological disorders have two things in common

1. **Genetics**
2. **Environment**

Genetics

1. **Damage to the DNA – DNA polymerase enzyme - Zn**
2. **Single nucleotide polymorphisms SNIPs – need for extra co-enzyme**
3. **Histones - (De)Methylation and (De)Acetylation**
4. **Malfunctioning codons – need for specific mineral**

Environment

1. **Infections – Bacteria, Viruses, Fungus, Parasites**
2. **Chemicals – Endogenous and Exogenous**
3. **Toxic metals**
4. **Radiation – Ionizing and Non-Ionizing**
5. **Allergy**
6. **Hypoxia**

All caused by Nutritional Deficiency?

Therapeutic considerations

1. **Energy production**
2. **Mitochondrial regeneration**
3. **Hypoxia**
4. **Antioxidant Support**
5. **Detoxification**
6. **Neurotransmitter Synthesis**

Parkinson's disease

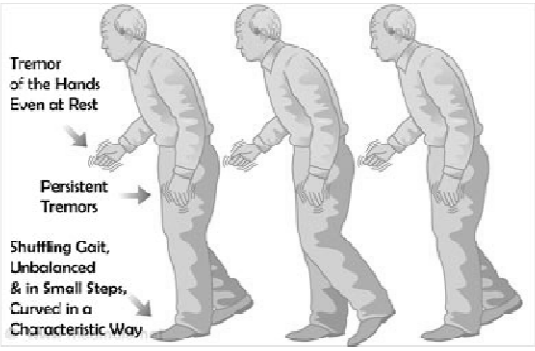
THE MAN WHO PARKINSON'S DISEASE IS NAMED AFTER WAS IMPLICATED IN A PLOT TO ASSASSINATE KING GEORGE III

March 19, 2014 | Emily Upton | Comment

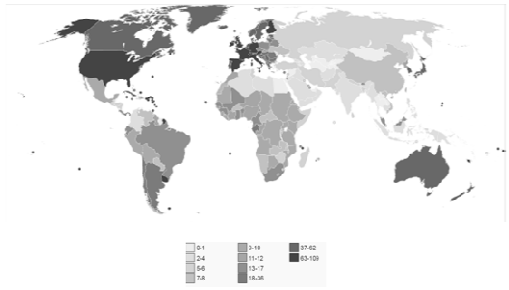
Today I found out they named Parkinson's disease after a man who was involved in an assassination attempt on King George III.

Parkinson's disease is a movement disorder characterized by tremors or shaking, with this particular symptom of the progressive disease resulting from copamine generating cell death in a part of the substantia nigra region of the brain. The disease was named after James Parkinson, a doctor, who wrote about it in 1817 in his 66-page paper, *An Essay on the Shaking Palsy*.

While his essay was important in the study of the disease, it had been around for a very long time, with recorded instances of it going back pretty much as long as humans have been writing things down. Parkinson had urged other doctors to get involved in researching the disease in order to find a cure, but his paper didn't really start to make its rounds until 1861. That was when Jean Martin Charcot, a French neurologist, separated the disease from other neurological disorders and named it "Parkinson's disease."



Parkinson's epidemiology



Parkinson's disease is a long-term disorder of the central nervous system that mainly affects the motor system. The symptoms generally come on slowly over time. Early in the disease, the most obvious are shaking, rigidity, slowness of movement, and difficulty with walking.

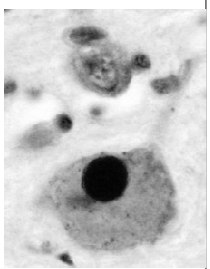
Thinking and behavioural problems may also occur. Dementia becomes common in the advanced stages of the disease. Depression and anxiety are also common occurring in more than a third of people. Other symptoms include sensory, sleep,and emotional problems.

The cause of Parkinson's disease is generally unknown, but believed to involve both genetic and environmental factors. Those with a family member affected are more likely to get the disease themselves.

There is also an increased risk in people exposed to certain pesticides and among those who have had prior head injuries while there is a reduced risk in tobacco smokers and those who drink coffee or tea.

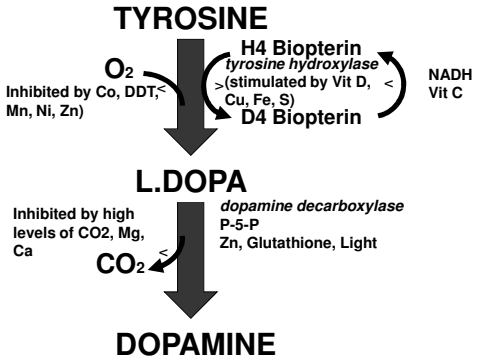
The motor symptoms of the disease result from the death of cells in the substantia nigra, a region of the midbrain. This results in not enough dopamine in these areas. The reason for this cell death is poorly understood but involves the build-upof proteins into Lewy bodies in the neurons.

Lewy bodies are abnormal aggregates of protein that develop inside nerve cells in Parkinson's disease, Lewy body dementia, and some other disorders.



Parkinson's disease may be partly due to oxidative stress. This oxidation may be relieved by neuromelanin. Patients with Parkinson's disease had 50% the amount of neuromelanin in the substantia nigra as compared to similar patients of their same age, but without Parkinson's.

Neuromelanin has been shown to bind neurotoxic and toxic metals that could promote neurodegeneration.

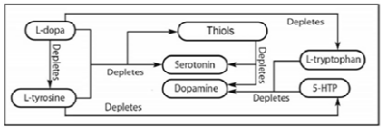


Drugs do nothing to increase the total number of serotonin and dopamine molecules in the system.

Drugs work be moving serotonin and dopamine concentrations from one place to another. In the process the serotonin and dopamine become depleted. Drugs that work with neurotransmitters do not work if there are not enough neurotransmitters. When the drugs quit working neurotransmitter levels are depleted below levels required for drug function.

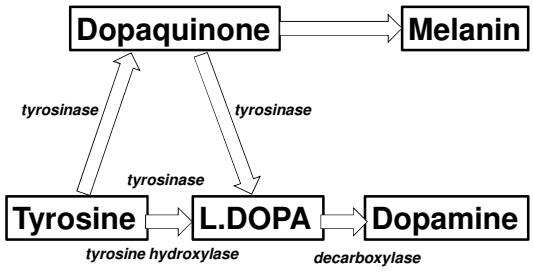
THE ONLY WAY to increase the neurotransmitter concentrations in the system is by giving the nutrients required by the body to make them.

Improper administration of nutrients can cause depletion of neurotransmitter and/or their precursors.



Learning to do it right takes time and dedication. Simply giving some amino acids and hoping for optimal results does not benefit the patient. When problems or side effects threaten treatment, get a consult

Melanin Steal

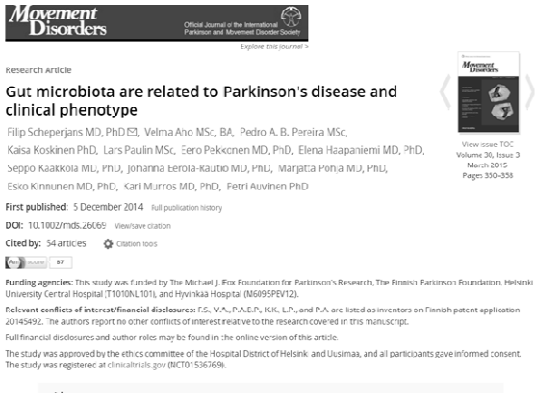


Applied Kinesiology findings
Low Dopamine CV
Low Serotonin BI
High Acetylcholine LIV

Aluminium toxicity and low boron which depletes serine and bromine.
Apple core is a good source of boron.

Don't forget the GUT which maybe where Parkinson's disease starts

Think
Digestive enzymes
Microbiota imbalance
Prebiotics
Probiotics



Movement Disorders
Official Journal of the International Parkinson and Movement Disorder Society

Explore this journal >

Research Article

Gut microbiota are related to Parkinson's disease and clinical phenotype

Filip Scheperjans MD, PhD ✉, Velma Aho MSc, BA, Pedro A. B. Pereira MSc, Kaisa Koskinen PhD, Lars Paulin MSc, Eero Pekkonen MD, PhD, Elena Haapaniemi MD, PhD, Seppo Kaakkola MD, PhD, Johanna Eerola-Rautio MD, PhD, Marjaana Pohja MD, PhD, Esko Kinnunen MD, PhD, Kari Murros MD, PhD, Petri Auvinen PhD

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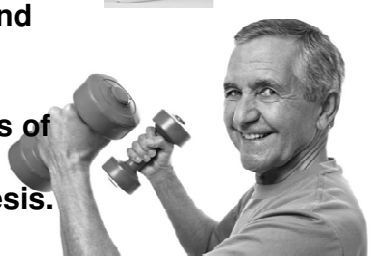
Full financial disclosures and author roles may be found in the online version of this article.

The study was approved by the ethics committee of the Hospital District of Helsinki and Uusimaa, and all participants gave informed consent. The study was registered at clinicaltrials.gov (NCT01536769).

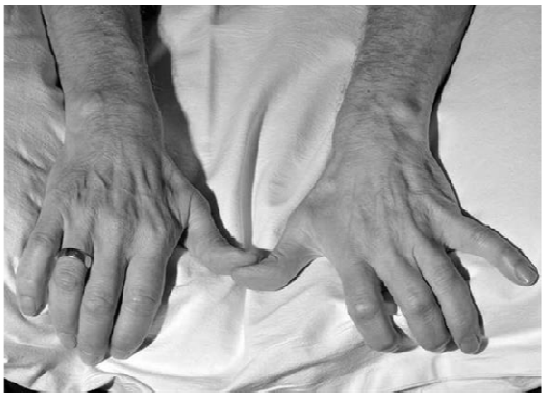
Aerobic but mainly resistance exercise and Spices are epigenetic modulators of brain neurogenesis.



Exercise and Spices stimulates the production of
Glial Derived Neurotrophic Factor (GDNF)
Brain Derived Neurotrophic Factor (BDNF)
Which stimulate new brain cells and connections.

Motor Neurone Disease

Lou Gehrig
1903-1941



Amyotrophic lateral sclerosis (ALS), also known as Lou Gehrig's disease and Motor Neurone Disease (MND), is a specific disease that causes the death of neurons which control voluntary muscles.

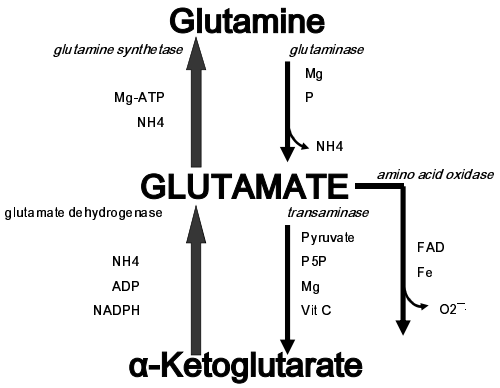
ALS is characterized by stiff muscles, muscle twitching, and gradually worsening weakness due to muscles decreasing in size. This results in difficulty in speaking, swallowing, and eventually breathing.

The cause is not known in 90% to 95% of cases. About 5–10% of cases are inherited from a person's parents. About half of these genetic cases are due to one of two specific genes. Before any muscular atrophy becomes apparent during ALS, roughly one-third of the motor neurons must be destroyed.

The disorder causes muscle weakness and atrophy throughout the body due to the degeneration of the upper and lower motor neurons.
Sensory nerves and the ANS are generally unaffected, meaning the majority of people maintain hearing, sight, touch, smell, and taste.

A defect on chromosome 21, which codes for Cu/Zn superoxide dismutase, is associated with about 20% of familial cases of ALS, or about 2% of ALS cases overall. This enzyme is a powerful antioxidant that protects the body from damage caused by superoxide, a toxic free radical generated in the mitochondria.

People with ALS have higher levels of glutamate in their serum and spinal fluid. There is a tentative association with exposure to a number of pesticides including the organochlorine insecticides aldrin, dieldrin, DDT, and toxaphene.



Prior to their destruction, motor neurons develop protein-rich inclusions in their cell bodies and axons. This may be partly due to defects in protein degradation.
These inclusions often contain ubiquitin, and generally incorporate one of the ALS-associated proteins.

Although many authors consider ALS to be caused by a combination of genetic and environmental risk factors, so far the latter have not been firmly identified, other than a higher risk with increasing age.

NMD maybe an aluminium toxicity – yttrium and boron deficiency.

CBS formula for detoxifying Chemicals

NAC, P-5-P, Mg, Vit C for high Glutamate and high Aluminium

Don't forget the GUT which maybe where MN disease starts

Think
Digestive enzymes
Microbiota imbalance
Prebiotics
Probiotics

Gut bacteria as a cause of Motor Neuron Disease

Scientists have started studying gut bacteria as a possible cause of a motor neuron toxin lying behind **Motor Neuron Disease** – they believe it is produced by a **clostridial** species, which then causes sporadic **amyotrophic lateral sclerosis** (ALS) in susceptible individuals.

Clostridial species are commonly present in humans but are normally held in check by beneficial gut bacteria. Research in the large scale **Human Microbiome** Project showed showed that almost all illness can be cause by a loss in diversity in the gut bacteria. When 'good' bacteria are reduced, 'bad' bacteria are not controlled and can cause increased toxin production, inflammation and even a negative immune response.

Lactic Acid Bacteria (LABs) can provide effective short-term controls (strains of *acidophilus*) and *bifidoacteria* too. Longer-term the diversity has to be re-introduced.

Some Clostridium bacteria toxins, like the tetanus and botulinum toxins, are known to target the motor system.

The study report talks of the following mechanism possibility, *"After gaining access to the lower motor neuron, the toxin would be transported back to the cell body, as occurs with the tetanus toxin, and destroy the lower motor neuron – the essential feature of ALS.*

Also like the tetanus toxin, some of the toxin would cross to neighboring cells and to the upper motor neuron and similarly destroy these motor neurons.

Weakness would relentlessly progress until not enough motor neurons remained to sustain life".

Damage to the diversity of gut bacteria normally occurs when people take drugs and especially antibiotics.

If this mechanism were proven correct solutions involving increasing the levels of bacteria in the gut, or neutralizing the toxins could prove effective.

Ref: http://www.ncbi.nlm.nih.gov/pubmed/15823706

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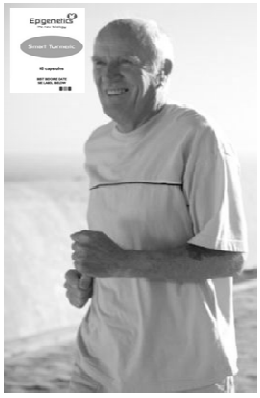
Aerobic and resistance exercise and Spices are epigenetic modulators of brain neurogenesis.



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Brain Derived Neurotrophic Factor (BDNF)
Which stimulate new brain cells and connections.

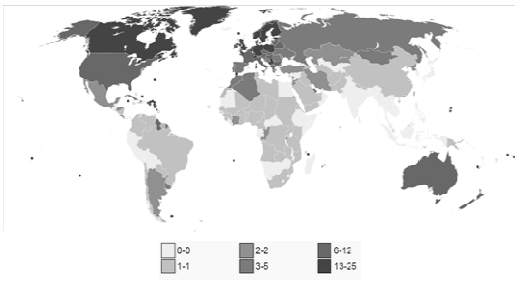
Multiple Sclerosis

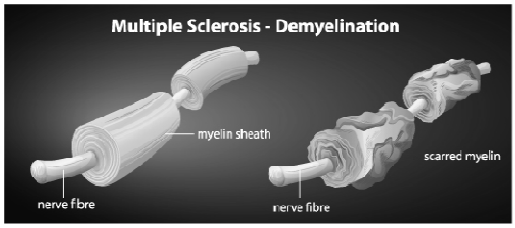
The French neurologist Jean-Martin Charcot (1825–1893) was the first person to recognize multiple sclerosis as a distinct disease in 1868.

Jean-Martin Charcot

Born 29 November 1825
Paris, France

Multiple Sclerosis epidemiology





Multiple sclerosis is a demyelinating disease in which the insulating covers of nerve cells in the brain and spinal cord are damaged. This damage disrupts the ability of parts of the nervous system to communicate, resulting in physical, mental, and sometimes psychiatric problems.

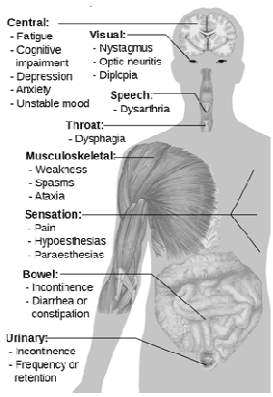
Specific symptoms can include double vision, blindness in one eye, muscle weakness, trouble with sensation, or trouble with coordination. MS takes several forms, with new symptoms either occurring in isolated attacks (relapsing forms) or building up over time (progressive forms).

Between attacks, symptoms may disappear completely; however, permanent neurological problems often remain, especially as the disease advances. While the cause is not clear, the underlying mechanism is thought to be either destruction by the immune system or failure of the myelin-producing cells.

Proposed causes for this include genetics and environmental factors such as being triggered by a viral infection such as herpes viruses, EBV, morbillinum, parotitis, rubella..
Multiple sclerosis is the most common autoimmune disorder affecting the CNS.

The name *multiple sclerosis* refers to the numerous scars (sclerae—better known as plaques or lesions) that develop on the white matter of the brain and spinal cord.

Main symptoms of Multiple Sclerosis



MS is more common in people who live farther from the equator, although exceptions exist. These exceptions include ethnic groups that are at low risk far from the equator such as the Samis, Amerindians, Canadian Hutterites, New Zealand Māori, and Canada's Inuit, -

As well as groups that have a relatively high risk close to the equator such as Sardinians, inland Sicilians, Palestinians and Parsis

MS is more common in regions with northern European populations and the geographic variation may simply reflect the global distribution of these high-risk populations. Decreased sunlight exposure resulting in decreased vitamin D production has also been put forward as an explanation.

STRUCTURE OF MYELIN
A kind of glial cell, the oligodendrocyte, has extensions from its cell body, which wrap around the axons (outgoing cell processes) of the neurons to protect and insulate the electric currents that travel through them.

The wrapping is called the "myelin sheath". Myelin is produced by these cells and is structured like rolls of concentric layering of cell membrane tissues around the myelinated nerves. There are some glial cell bodies visible between the layers.

Myelin is composed of 30% protein and 70% lipid
Basic protein and proteolipid
Phospholipids and Plasmalogens
Sphinglomyelins
Gylcospinglolipids i.e.
- **Cerebrosides and**
- **Gangliosides**

Cholesterol

GLIAL PROTEINS
1. Myelin Basic Protein
2. α2-Glycoprotein
3. Glial fibrillar acid protein
4. Other proteins of microglial cells:
NAD(P)H oxidase, peroxidase, lysosomal cationic proteins, lysozyme, lactoferrin etc.

GLIAL LIPIDS
Lipid content of the brain is as high as 50% dry weight, while myelin contains approximately 70% lipids. CNS is characterised by the most structural diversity of membrane lipids compared to other organs. In myelin, 18:1 (Oleic acid) and 18:0 (Stearic acid) are prevalent.

Myelin is rich in

1. **Phospholipids (especially Phosphatidylinositol 4,5-Diphosphate, Phosphatidic acid, Phosphatidyl choline, Phosphatidyl ethanolamine, Phosphatidyl serine, Phosphatidyl glycerol and Phosphatidyl inositol), Plasmalogens and Sphingomyelins (41-45%),**
2. **Glycosphingolipids and Ganglioside GM1 (27-30%)**
3. **Cholesterol (25-28%).**

Mature myelin has the least water content and highest lipid/protein ratio of all cell membranes. Phospholipids in myelin contain a variety of even numbered carbon saturated and unsaturated fatty acids at the C1 and C2 phospholipid attachments.

Nutrients for MS
Omega 3 or DHA
Phospholipids
CBS for lead – Vitamin D activity
Vitamin C
Iodine
Magnesium
Vitamin B12 (Adenosylcobalamin)
Vitamin K2 for decalcification

Don't forget the GUT which maybe where MS disease starts

Think
Digestive enzymes
Microbiota imbalance
Prebiotics
Probiotics

Link between gut bacteria, MS discovered

MS patients show lower levels of good bacteria

Date: June 27, 2016

Source: University of Iowa Health Care

Summary: Researchers are now saying bad gut bacteria -- or an insufficient amount of good bacteria -- may have a direct link to multiple sclerosis.

Researchers are now saying bad gut bacteria -- or an insufficient amount of good bacteria -- may have a direct link to multiple sclerosis as well.
"Every human carries trillions of bacteria in their gut (gut microbiome) and recent advances in research indicate that these tiny passengers play an important role in our overall health maintenance," says Ashutosh Mangalam, PhD, assistant professor of pathology at the University of Iowa Carver College of Medicine.
Since the bacteria are associated with contributing to good health, Mangalam and his colleagues wondered whether those with a chronic autoimmune disorder, such as multiple sclerosis, would then have a gut microbiome that is different than the microbiome found in healthy individuals.
In a study published online in the journal *Scientific Reports*, Mangalam and his team say that MS patients do, in fact, have a distinct microbiome from their healthy peers.

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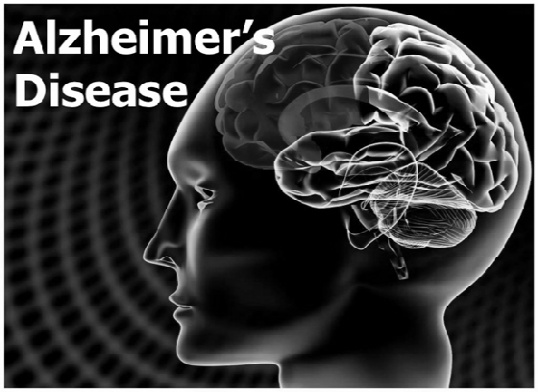
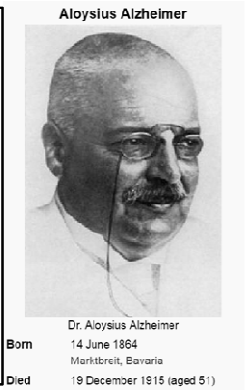


Alzheimer is credited with identifying the first published case of "pre-senile dementia", which Kraepelin would later identify as Alzheimer's disease.



In1991,the *amyloid hypothesis* po stulated that extracellular amyloid beta deposits are the fundamental cause of the disease. Support for this comes from the location of the gene for the amyloid precursor protein (APP) on chromosome 21, together with the fact that people with trisomy 21 (Down Syndrome) who have an extra gene copy -

almost universally exhibit AD by 40 years of age. Also, a specific isoform of apolipoprotein, APOE4, is a major genetic risk factor for AD. Whilst apolipoproteins enhance the breakdown of beta amyloid, some isoforms are not very effective at this task (such as APOE4), leading to excess amyloid build up in the brain.

Further evidence comes from the finding that transgenic mice that express a mutant form of the human APP gene develop fibrillar amyloid plaques and Alzheimer's-like brain pathology with spatial learning deficits.

The *tau hypothesis* proposes that tau protein abnormalities initiate the disease cascade. In this.model, hyperphosphorylated tau begins to pair with other threads of tau. Eventually, they form neurofibrillary tangles inside nerve cell bodies.

When this occurs, the microtubules disintegrate, destroying the structure of the cell's cytoskeleton which collapses the neuron's transport system (can also be the effect of mercury toxicity).This may result in malfunctions in biochemical communication between neurons and later in the death of the cells.

Approximately 50% of all Alzheimer's cases have APOE4 expression and have to careful with excess saturated fats in the diet.

The other 50% have Insulin resistance.

Memory loss leading to Alzheimer's degenerative changes begin in the hippocampus which starts to shrink, so short term memory is affected first.
The brain losses its plasticity and its ability to make connections between neurones, many of which will die.

Markers for examining people with Memory Loss
Amyloid beta protein fragment 1-42

ENERGY
Mg-ATP
DNA Polymerase
CoQ10 (Ubiquinone)
Complex III Cytochrome c reductase
Cytochrome C
Complex IV Cytochrome c oxidase
Cardiolipin
CO
CN
Malondialdehyde
Formic acid

HYPOXIA
O2
Hemoglobin
ALA
PBG
Uroporphyrin III
Coproporphyrin III
Protoporphyrin IX
Heme

Homocysteine
APOE4
Probiotics

Turmeric is a yttrium / boron base with three forms of boron in it. Alzheimer's maybe an aluminium toxicity – yttrium and boron deficiency.

Low iodine leads to senior moments.

Sulfur / Taurine

Don't forget the GUT which maybe where Alzheimer's disease starts

Think
Digestive enzymes
Microbiota imbalance
Prebiotics
Probiotics

Link between Alzheimer's and gut bacteria

9 June 2016

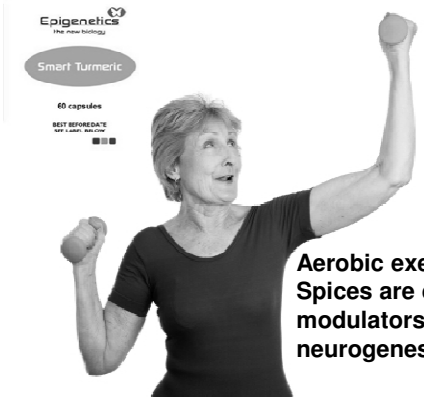
Recent research with mice points to a link between the composition of gut flora and the development of Alzheimer's disease. Researchers from KU Leuven and VIB have now joined a European research project that will investigate this link in greater detail and develop possible treatments based on their findings.

Scientists from the Ecole polytechnique fédérale de Lausanne in Switzerland found a link between the composition of gut flora and the development of Alzheimer's disease. Their findings are based on mice. The researchers from Lausanne have now teamed up with scientists from KU Leuven and VIB to examine which bacteria have an impact on Alzheimer's.

Professor Jeroen Raes, gut flora expert of the Laboratory of Molecular Bacteriology (KU Leuven / VIB) explains: "The discovery of a possible link between the composition of gut flora and the developmental mechanisms of Alzheimer's disease is of crucial importance. It opens up a whole new avenue in the battle against this incurable disease."

The new consortium, called AD-gut, will look for new methods to map the microbial composition of our gut flora quickly and with precision. The consortium will also try to find out which intestinal cultures speed up the development of Alzheimer's disease. This may help detect the disease at an earlier stage. Last but not least, the international team will examine whether it's possible to develop specific probiotic cocktails that can change the gut flora in such a way as to halt the development of Alzheimer's.

Acetylcholine Synthesis
Acetylcarnitine
CoA
Magnesium
B Complex
Choline or Phosphatidylcholine
Vitamin B1 (Thiamine PP)
Manganese
Smart Zinc



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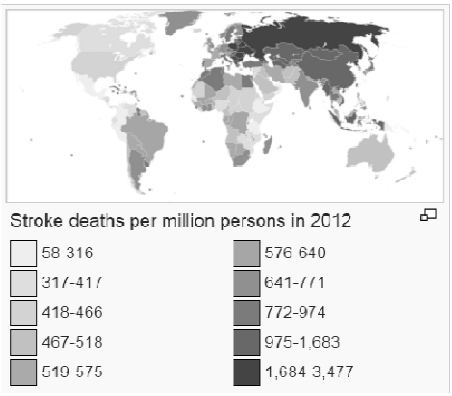
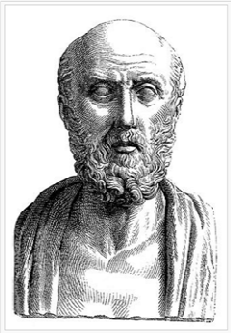
Which stimulate new brain cells and connections.

Stroke
CVA

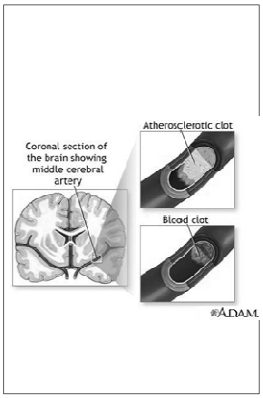


Hippocrates (460 to 370 BC) was first to describe the phenomenon of sudden paralysis that is often associated with ischemia. Apoplexy, from the Greek word meaning "struck down with violence", first appeared in Hippocratic writings to describe this phenomenon.

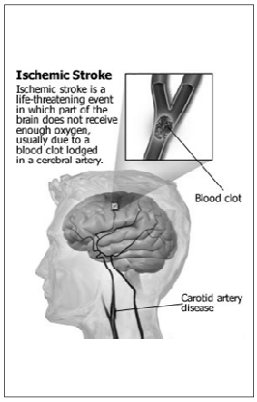


"First do no harm"

There are two kinds of strokes. An *ischemic stroke* occurs when the blood supply to the brain is interrupted, usually by a blood clot.

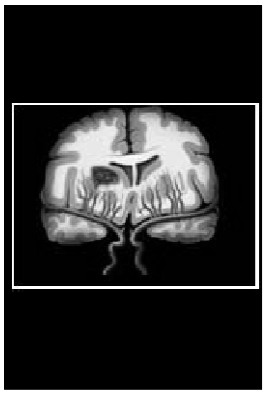


These clots may be caused by "hardening of the arteries" in the carotid arteries, which feed the head and brain with oxygen-rich blood.



Each year, 2.8 million older adults are treated in U.S. emergency rooms due to falls; more than 800,000 of them are hospitalized as a result
Falls are also the most common cause of traumatic brain injuries and account for $31 billion in direct U.S. medical costs annually
Balance and coordination exercises and strength training, along with optimizing your vitamin D levels, can reduce your risk of falls and fall-related injuries

The second kind of stroke is a *hemorrhagic stroke*, which occurs when there is bleeding into or around the brain.



Ischemic stroke

Probably due to oxidised cholesterol.

Omega 3 to prevent platelet aggregation.

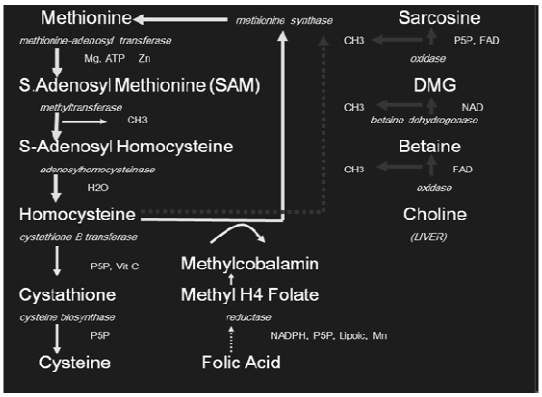
Magnesium, ginger, garlic to stimulate prostacyclins.

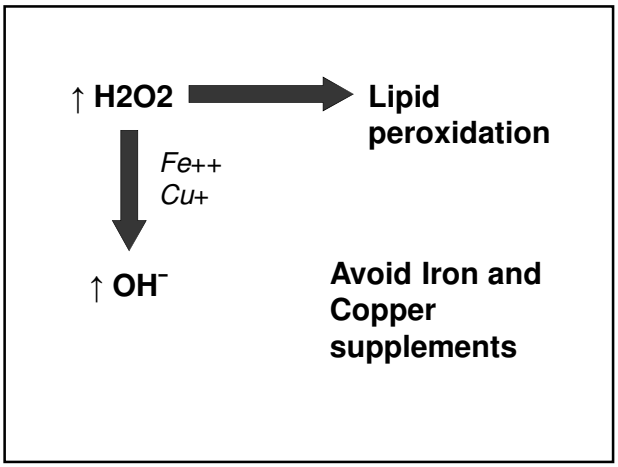
Hemorrhagic stroke

Due to weakened artery endothelial cells due to high homocysteine.

Consider homocysteine factors P5P, Vit C, Methylcobalamin, Methyl H4 folate, Betaine, DMG.

CBS Formula





Nutrients to consider with CVAs

Ischemic CVA	Hemorrhagic CVA
Omega 3	P5P
Phospholipid	Vitamin C
Magnesium	Methylcobalamin
Onion, Ginger, Garlic (GOG)	Methyl H4 folate
Selenium	CBS Formula
Vitamin K2	

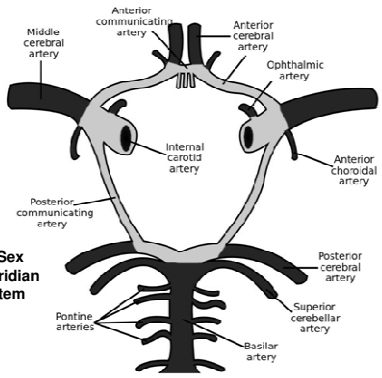
Tweaking our gut bacteria could help protect our brain from strokes

Experiments with animals saw 60% less brain damage.

PETER DOCKRILL 29 MAR 2016

Recent research has shown how fundamentally important the bacteria in our gut are to the rest of our mental and physical health, affecting everything from our appetite to our state of mind.

Now a new study suggests that our gut bacteria could even play a role in protecting us from brain damage, with an experiment involving mice showing that certain types of stomach microbes can actually help reduce the severity of strokes.



The Circulation / Sex (Pericardium) meridian is the arterial system

Aerobic exercise and Spices are epigenetic modulators of brain neurogenesis.

Exercise and Spices stimulates the production of
Glial Derived Neurotrophic Factor (GDNF)
Brain Derived Neurotrophic Factor (BDNF)
Which stimulate new brain cells and connections.

Summary of Nutrients to Consider for Managing Neurological disorders

1. **Energy production**
2. **Mitochondria regeneration**
3. **Hypoxia**
4. **Antioxidants**
5. **Detoxification**
6. **Neurotransmitter synthesis**

1. Energy production

B Complex
Magnesium
alpha Lipoic Acid
Smart Thinking Oil (DHA)
Selenium for T4>T3

2. Mitochondrial regeneration

Zinc for DNA polymerase
CoQ10
Smart Turmeric
Vitamin B12
Folates
Vitamin B6 (P-5-P)
Vitamin C
Smart Thinking Oil (DHA)
Coconut oil

Mitochondrial regeneration cont.
Smart Vitamin D3
Alpha Lipoic Acid + AcetylCarnitine
NADH for Complex 1
FADH2 for Complex 11

3. Hypoxia
Smart Thinking Oil (DHA)
Glycine, 5MTHFolate
Vitamin B12 (Adenosylcobalamin)
Magnesium, Zinc
Biotin, CH2H4Folate / H4Biopterin
Vitamin B6 (P-5-P)
Vitamin C
Iron

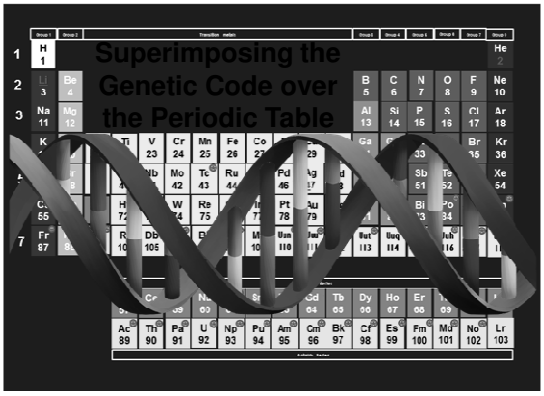
4. Antioxidant Support
Reduced Glutathione / Selenium
Smart Turmeric
Cloves / Cinnamon
Smart C Complex
Smart Vitamin E
Lutein, Ginkgo biloba
Lemon balm
Rosemary
Green tea

5. Detoxification
Glutathione – needs Vit C, a-Lipoic acid, Selenium, NAC
CBS formula, Taurine,
Potassium ascorbate
Smart Turmeric, Coriander
Yarrow
Lemon balm
Black walnut tincture
Probiotics

6. Acetylcholine Synthesis
Acetylcarnitine
CoA
Magnesium
B Complex
Choline or Phosphatidylcholine
Vitamin B1 (Thiamine PP)
Manganese
Smart Zinc

6. Other Neurotransmitters
Dopamine
Noradrenalin
Serotonin
Histamine
GABA / Glycine / Taurine
Aspartic acid / Glutamic acid

The single food most capable of repairing DNA is the Beetroot.
It can repair chromosome abnormalities.

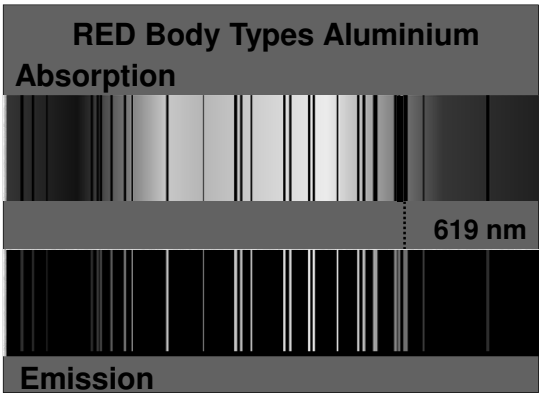


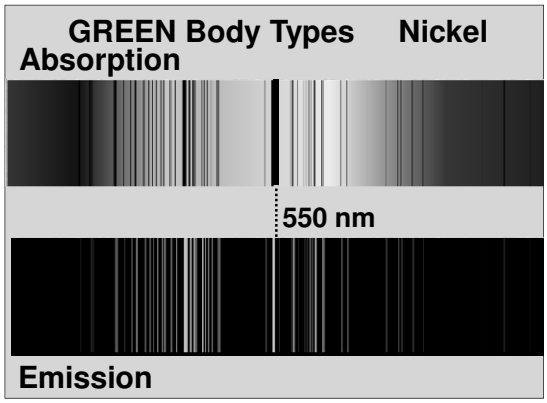
The tumour suppressing gene p53 found on chromosome 17 is a selenium based gene that suppresses the formation of tumours.

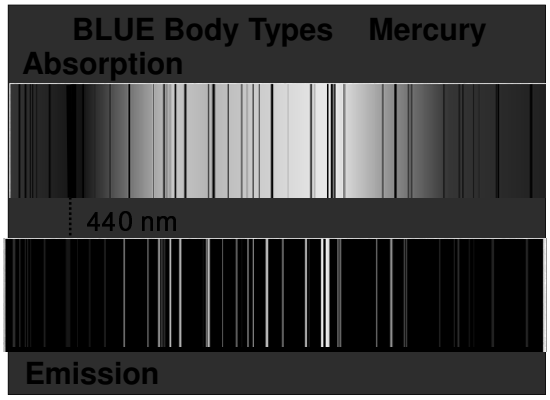
Molybdenum is a trace mineral found in unfiltered water, cheese, grains, leafy greens, nuts and liver
When plants fail to uptake molybdenum properly, potentially carcinogenic nitrosamines are produced because nitrate reductase, an enzyme, can't perform properly without molybdenum
Where there is little access to foods containing it, minimal dietary molybdenum is associated with esophageal and stomach cancers and other diseases.

Ovarian cysts – fibrocystic tumour = caffeine toxicity with iodine deficiency imbalances potassium.

Sleepies in the eye is iodine deficiency





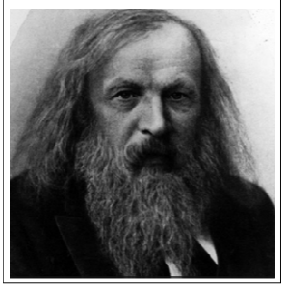


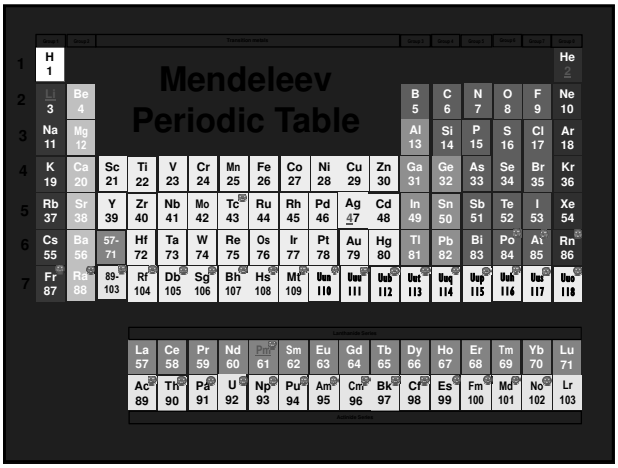
Essential minerals
Spectroscopic emission strengthens
Spectroscopic absorption weakens

Toxic minerals
Spectroscopic emission weakens
Spectroscopic absorption strengthens

1834 – 1907 was a Russian chemist and inventor. He formulated the Periodic Law, created a farsighted version of the periodic table of elements, and used it to correct the properties of some already discovered elements and also to predict the properties of eight elements yet to be discovered.

Dmitri Ivanovich Mendeleev



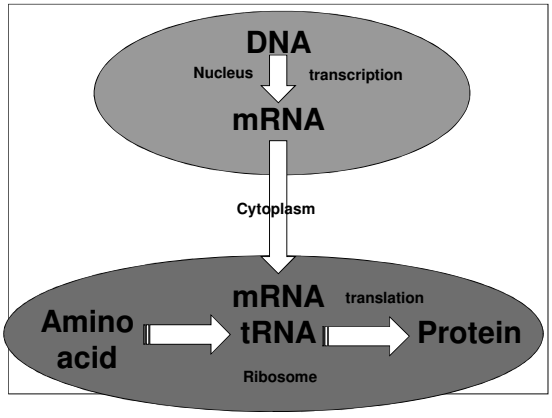


400nm inhibits Heme C in the Cytochrome oxidase pathway in the mitochondria.

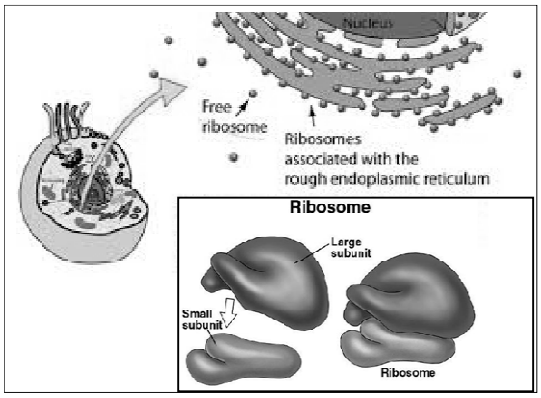
Complement to 400nm

Essential minerals	Toxic minerals	
Boron	Aluminium	Radium
Calcium	Antimony	Radon
Copper ↑↓	Arsenic*	Thallium
Chromium ↑↓	Beryllium*	Thorium
Indium	Bismuth	Uranium
Iodine	Bromine	
Iron ↑↓	Cadmium*	
Magnesium	Caesium	Known to be *
Manganese ↑↓	Chlorine	Carcinogenic
Molybdenum	Cobalt	
Platinum	Fluorine	
Potassium	Lead	
Selenium	Lithium	
Silica	Mercury	
Silver	Nickel*	
Sulphur	Palladium	
Zinc	Promethium*	

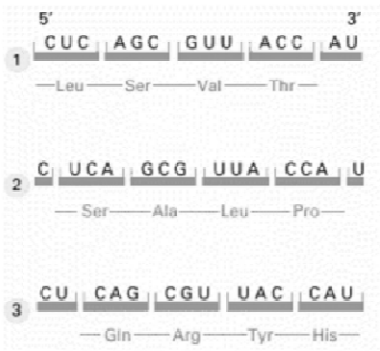
From RNA to Protein



The translation of the nucleotide sequence of an mRNA molecule into protein takes place in the cytoplasm on a large ribonucleoprotein assembly called a ribosome.



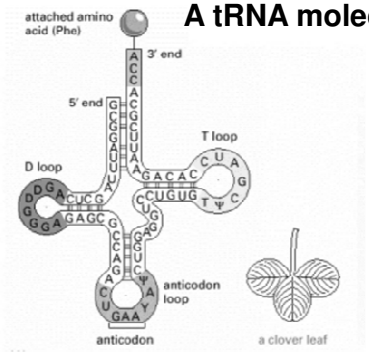
The amino acids used for protein synthesis are first attached to a family of tRNA molecules, each of which recognizes, by complementary base-pair interactions, particular sets of three nucleotides in the mRNA (codons).



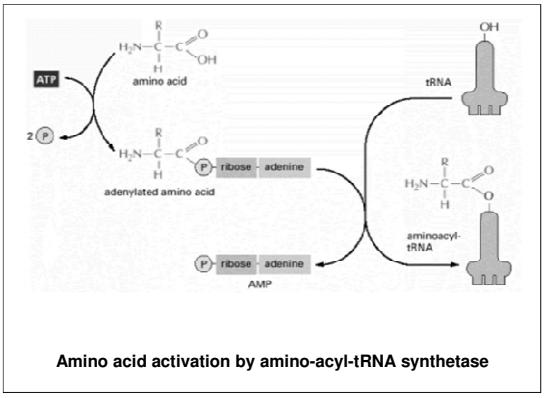
The three possible reading frames in protein synthesis

The sequence of nucleotides in the mRNA is then read from one end to the other in sets of three according to the genetic code. To initiate translation, a small ribosomal subunit binds to the mRNA molecule at a start codon (AUG) that is recognized by a unique initiator tRNA molecule.

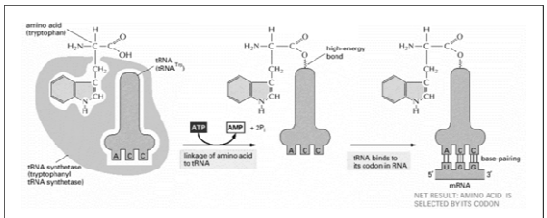
A tRNA molecule



During this phase, aminoacyl tRNAs—each bearing a specific amino acid bind sequentially to the appropriate codon in mRNA by forming complementary base pairs with the tRNA anticodon.

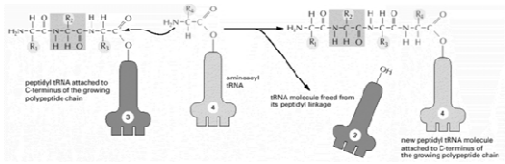


Amino acid activation by amino-acyl-tRNA synthetase

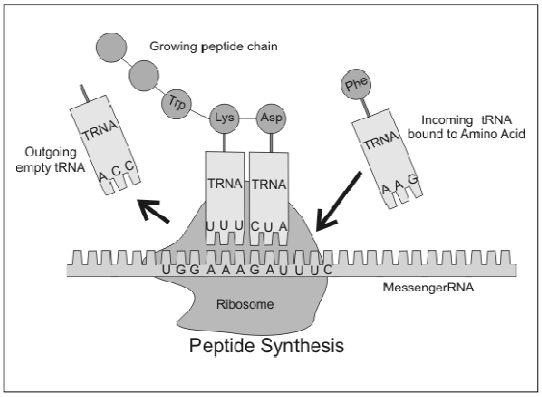


The genetic code is translated by means of two adaptors that act one after another.

Each amino acid is added to the C-terminal end of the growing polypeptide by means of a cycle of three sequential steps: aminoacyl-tRNA binding, followed by peptide bond formation, followed by ribosome translocation.



The incorporation of an amino acid into a protein.



The mRNA molecule progresses codon by codon through the ribosome in the 5′-to-3′ direction until one of three stop codons is reached. A release factor then binds to the ribosome, terminating translation and releasing the completed polypeptide.

Codon challenge

Codon Challenge

1. Using the 400nm acetate from weakness challenge with each nucleotide base for strengthening.
2. Leave positive nucleotide base on and wait 5 seconds for weakening.
3. Repeat for next nucleotide base to strengthen

4. Leave second positive nucleotide base on and wait 5 seconds for weakening.
5. Repeat for third nucleotide base to strengthen.
If double or treble codon use nucleotide from another packet.
e.g. AUU

This is the positive codon

The Codon Chart will tell you the
1. Associated Amino acid
2. Spinal level
3. Regulator (Co-ordinator) element
4. Valence
5. Codon meridian
6. Optimal nutrient(s)

CODON	AMINO ACID	SPINE	REGULATOR	VALENCE	MERIDIAN	NUTRIENTS
AAA	Lysine	C2		- 2	St	S, Cu
AAC	Asparagine	T5D		+ 3	Lu	
AAG	Lysine	T4		0	LIV	I
AAU	Asparagine	C6		- 4 +	SI	Si
ACA	Threonine	T2		- 4 +	TW	SI, Co, I
ACC	Threonine	T11D	Fluorine	- 1	BI	I
ACG	Threonine	C7		0	Kid	K
ACU	Threonine	L4D	Potassium	+1	LIV/Sp	Ca, Mg, Na, I, Se
AGA	Arginine	Co3	Barium	+ 2	Cx	Ca/Mg, Se
AGC	Serine	S5	Rhodium	- 4 +	SI	Si, K
AGG	Arginine	T10D	Nitrogen	- 3	SI	Se, Zn, Mg, Si
AGU	Serine	T9D	Boron	+ 3	Ht	K, Ca, Vit D, Vit k2, K,
AUA	Isoleucine	T1D		+ 3	Cx	B
AUC	Isoleucine	S3	Krypton	0	Sp	Se
AUG	Methionine (START)	S3D	Rubidium	+ 1	Sp	Se, Mg, B Complex,Mg, Vit A, I
AUU	Isoleucine	L1D	Aluminium	+ 3	Cx	Se, Ca, Mg, B, Si,P

CODON	AMINO ACID	SPINE	REGULATOR	VALENCE	MERIDIAN	NUTRIENTS
CAA	Glutamine	C4D		+3	Ht	
CAC	Histidine	C3		0	Sp	
CAG	Glutamine	Co 1D	Iodine	-1	Bl	I, Zn, K, Se, Ca, Si, P
CAU	Histidine	T8D	Lithium	+1	Sp	Ca, I, S, Mg
CCA	Proline	L2	Silicon	-4+	TW	S
CCC	Proline	Co 4	Lutetium	-4+	TW	Cc, Ca, B, K, I-3-C
CCG	Proline	C4		+2	Ht	Ca, Mg
CCU	Proline	L5 D	Scandium	+3	Lu	B, Ca
CGA	Arginine	T10	Carbon	-4+	SI	Se, Zn, Mg
CGC	Arginine	S2	Selenium	-2	St	
CGG	Arginine	L4	Argon	0	LIV	
CGU	Arginine	T8	Helium	0	Sp	Ca, Se
CUA	Leucine	L5	Calcium	+2	Lu/Sp	Si, Mg, Vit K, I
CUC	Leucine	T3	-	-2	GB	Se, S
CUG	Leucine (START)	S5 D	Antimony	-3	SI	Z, K, B, Cu, Mg
CUU	Leucine	S1	Cobalt	-4+	LI	Zn, Fe, Ge, V, Cr, Mn, Cu, K

CODON	AMINO ACID	SPINE	REGULATOR	VALENCE	MERIDIAN	NUTRIENTS
GAA	Glutamic acid	T11	Oxygen	-2	Bl	Si, Se, K
GAC	Aspartic acid	L2 D	Phosphorus	-3	TW	Ca, Se, Mg, Fe, K
GAG	Glutamic acid	Co 2 D	Cesium	+1	Kid	K, Zn
GAU	Aspartic acid	Co 2	Xenon	0	Kid	
GCA	Alanine	S4	Strontium	+2	Ht	Ca, B
GCC	Alanine	L1	Magnesium	+2	Cx	Ca, Na, K
GCG	Alanine	C6		-2	Bl	
GCU	Alanine	Co 1	Tellurium	-2	Bl	Se
GGA	Glycine	T11	Oxygen	-2	Bl/Kid	Si, Se
GGC	Glycine	T1		+2	Cx	Mg, Ca
GGG	Glycine	C1		-3	LI	S
GGU	Glycine	L3 D	Chlorine	-1	GB	
GUA	Valine	C6 D		-1	Bl	I, Se
GUC	Valine	T7		-2	St	Zn, Se, S, Mg, S
GUG	Valine	C5 D		-1	SI	
GUU	Valine	C3 D		+1	Sp	Na / K, I

CODON	AMINO ACID	SPINE	REGULATOR	VALENCE	MERIDIAN	NUTRIENTS
UAA	STOP 1	T6	Hydrogen	-4+	LI	K, Se
UAC	Tyrosine	T7 D		-1	St	Zn, I
UAG	STOP 2	L3	Sulfur	-2	GB	Se, S
UAU	Tyrosine	C2 D		-1	St	
UCA	Serine	S2 D	Bromine	-1	St	Ca, K, B
UCC	Phenylala-nine	T5		+2	Lu	Ca, Mg, Mn
UCG	Serine	T6 D		-3	LI	Zn, Mg
UCU	Serine	T2 D		-3	TW	
UGA	STOP 3	S5 D	Yttrium	+3	Ht	Se, Mg
UGC	Selenium cyst	T9	Beryllium	+2	Ht	Zn, Ca, I
UGG	Tryptophan	C7 D		+1	Kid	I
UGU	Cysteine	S1 D	Arsenic	-3	LI	S
UUA	Leucine	T12	Neon	0	Kid	
UUC	Phenylala-nine	T5		+2	Lu	Ca, Mg, Mn
UUG	Leucine (START)	T12 D	Sodium	+1	Kid	Ca, Se, I-3-C
UUU	Phenylala-nine	Co 3 D	Lanthanum	+3	Cx/Sp	Zn

The associated Amino acid (get from Product Test Kit) will display as making the test muscle go weak but may take a few moments to do so.

Similarly the regulating element if known will do this also.

Using the weakness obtained from the amino acid cross check against all the essential mineral Spectroscopic emission acetates for which one(s) strengthen.

This is the mineral the amino acid requires to activate the codon.

Challenge for the optimal form of the mineral from your Product Test Kit against the Subconscious Meridian's weak associated muscle. (e.g. Tensor fascia lata for the Large Intestine meridian)

Reset
TL the meridian B&E point, muscle test and the take finger off and retest muscle again

If now weakens means there is another nutrient so cross check for strengthening to amino acids to elicit any mono, di, tri, quad peptide required to activate the gene(s).
Prescribe positive amino acid(s) in organic apple juice at specific time as tested. Keep at least 15 minutes away from eating

And / Or any other nutrients by using the composites
Minerals
Vitamins
Co-enzymes
Unsaturated fatty acids
Probiotics
Herbs / Spices

You can next cross check in the clear for any toxic elements using the Toxic Metal Spectroscopic emission acetates. e.g.
Aluminium Lead
Antimony
Arsenic
Cadmium
Mercury

You may consider a metal chelator at this stage like
CBS formula
Taurine
NAC
Yarrow

If it strengthens the toxic metal acetate or weak associated muscle.

Alternative Meridian / Neurotransmitter challenge

1. **Identify Codon meridian that negates the Amino acid.**
2. **With YANG meridians challenge in the clear for weakening substrate. With YIN meridians challenge with neurotransmitter in the clear.**

3. Use this substance as the weakening marker.
4. Challenge different nutrients as above.
5. You can use the Codon Meridian's B&E point as a reset button. Remember to test both sides to ensure all nutrients have been identified.

Epithalamin

According the peptide theory of ageing, ageing is an evolutionary determined biological process of changes in gene expression resulting in impaired synthesis of regulatory and tissue-specific peptides in organs and tissues, which provokes their structural and functional changes and the development of diseases.

Correspondingly, correction of such disorders by means of stimulation of peptide production in the organism or through their delivery can promote the normalisation of disturbed body functions.

Based on the data about the amino acid compositions of peptide preparations, novel principles of the design of biologically active short peptides possessing tissue-specific activities has been developed.

Dipeptides specific for the thymus and tetrapeptides specific for the heart, liver, brain cortex, and pineal glands stimulate the in vitro outgrowth of explants of respective organs.

Interestingly, for eye retina and the pineal gland, a common tetrapeptide Ala-Glu-Asp-Gly (Epithalamin factors) has been designed, probably reflecting the common embryonal origin of these two organs. Epithalamin factors reproduces the effects of Epithalamin including those related to its geroprotector activity.

In particular, Epithalamin factors increases the lifespan of mice and fruit flies and restores the circadian rhythms of melatonin and cortisol production in old rhesus monkeys.

At the same time, Epithalamin prolongs the functional integrity of the eye retina in Campbell rats with hereditary Retinitis Pigmentosa and improves the visual functions in patients with pigmental retinal degeneration. Changes in gene expression were observed to be produced by the short peptide preparations.

Therefore, the effects of Epithalamin are suggested to be mediated by transcriptional machinery common for the pineal gland and the retina and, probably, for regulation of melatonin production

Melatonin, the hormone that regulates our daily cycle, is found to prolong life span in mice.
Melatonin in the blood is very sensitive to light exposure, and melatonin disappears with the dawn's early light

Anisimov found that sleeping in total darkness is better for longevity than exposure to light during the night.

(A recent study also suggests sleeping in the cold helps preserve insulin sensitivity.)

It was revealed that addition of tetrapeptide Ala-Glu-Asp-Gly to the cultural medium of human lung fibroblasts induces telomerase gene expression and contributes to a 2.4-fold lengthening of telomeres. Activation of gene expression is accompanied by a growing number of cellular divisions (by 42.5%), which is the evidence of Hayflick's limit being overcome.

The effect of di- and tetrapeptides Lys-Glu, Glu-Trp, Ala-Glu-Asp-Gly, Ala-Glu-Asp-Pro on the expression of 15 247 murine heart and brain genes before and after peptides administration was studied with the employment of DNA-microarray technology. In this experiment, there were used clones from the library of the National Institute on Ageing, USA.

This experiment provided unique data on alteration in the expression of different genes under the effect of peptide preparations. An important conclusion driven from the experiment was that every peptide specifically regulates particular genes.

Results of this experiment testify to the existing mechanism of peptide regulation of gene activity. It was also registered that dipeptide Lys-Glu, showing immunomodulating activity, regulates gene interleukin-2 expression in blood lymphocytes.

Telomere protectors and telomerase stimulants
Ashwagandha
Turmeric
Glutathione
CoQ10
Magnesium bisglycinate
Potassium ascorbate

Modified Therapy Localisation Technique

1. Practitioner identifies spinal or disc lesion
2. Practitioner performs spiral torsion
3. Practitioner put forefinger of one hand on lesion and of other hand on patient's chin
4. Pulse for 1 minute

Pulse together for 1 minute