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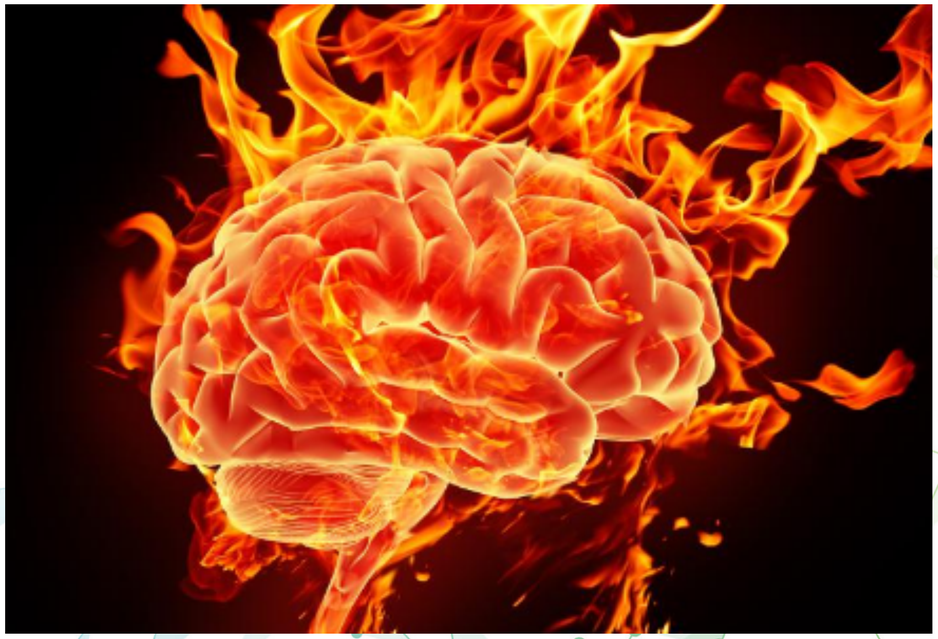
Migraine - Integrating nutrition & neuromechanical care



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100% Online nutrition and functional medicine course for Neuro-mechanical practitioners

- next enrolment November

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info@academyofchiropracticnutrition.com
Simon Billings to safe list/contacts

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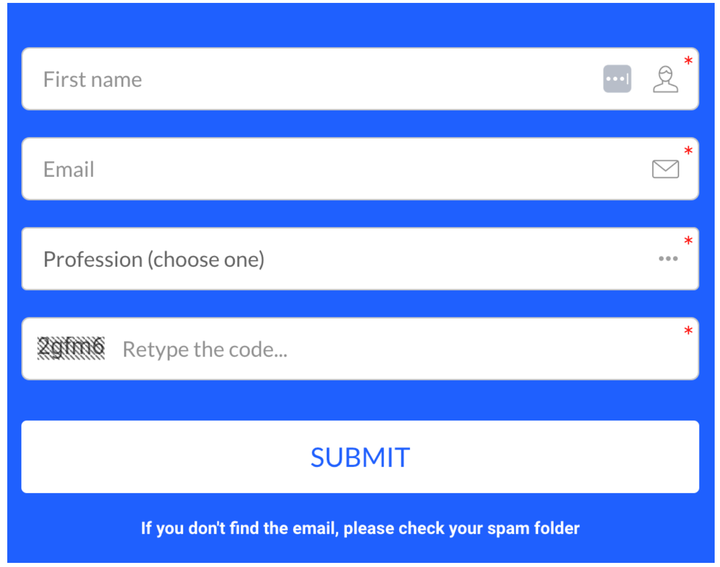

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Weekly newsletter Fri 5 pm

Plus vitamin D symptom & dosing chart

Vitamin D video on mechanisms behind vitamin D symptoms plus pregnancy & breastfeeding

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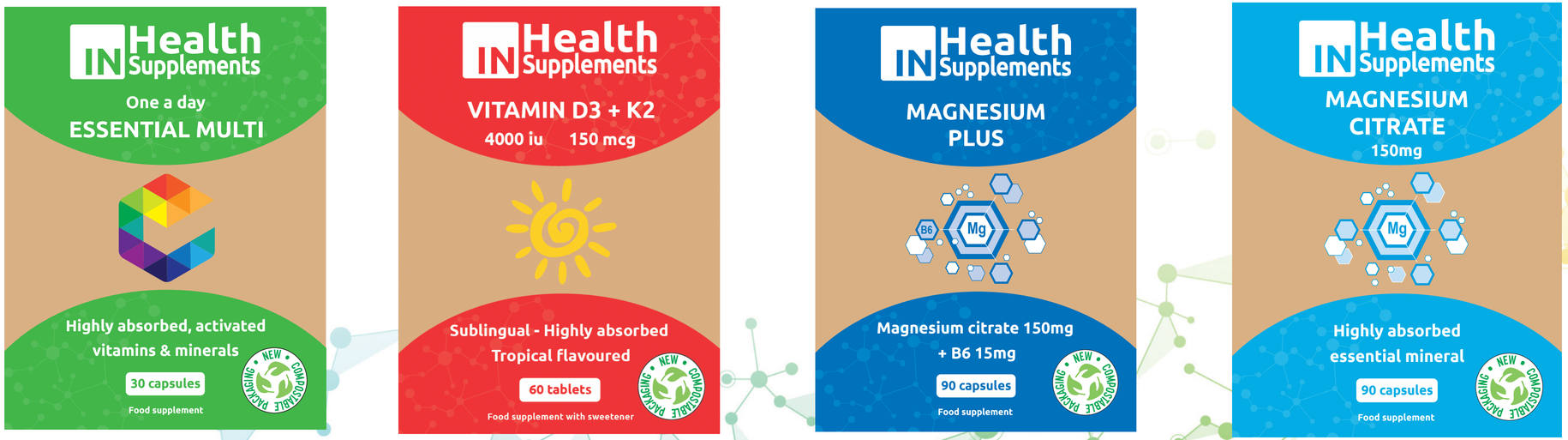
Health Supplements

w.inhealthsupplements.co.uk

Specialist in supporting results from Neuro-mechanical care

CORE 4



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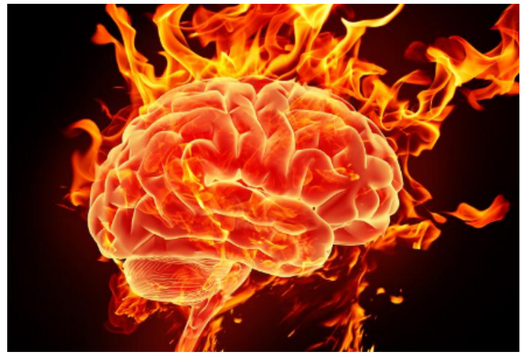
Primed for migraine?



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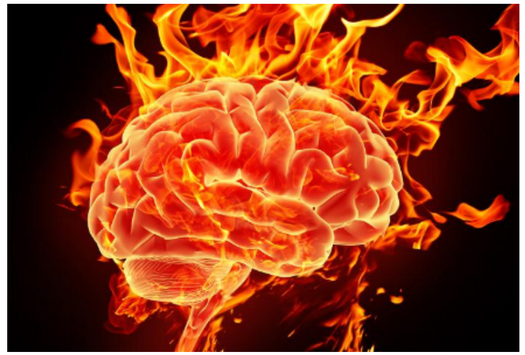
Primed for migraine?

(epi)genetics



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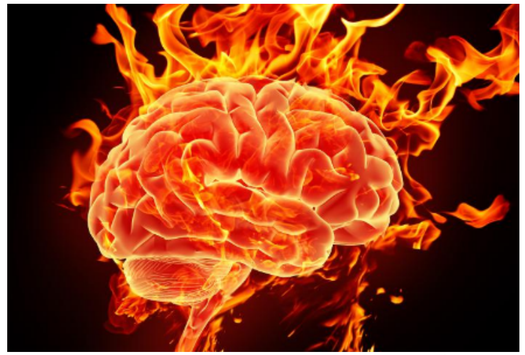
Primed for migraine?

(epi)genetics

Deficiencies - B2,6,12, Mg,Vit D, CoQ10, carnitine



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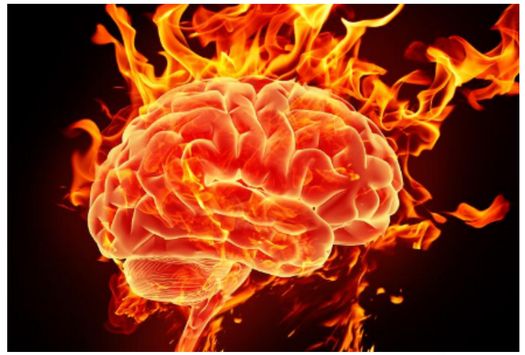
Primed for migraine?

(epi)genetics

Deficiencies - B2,6,12, Mg,Vit D, CoQ10, carnitine

Food reaction = inflammation



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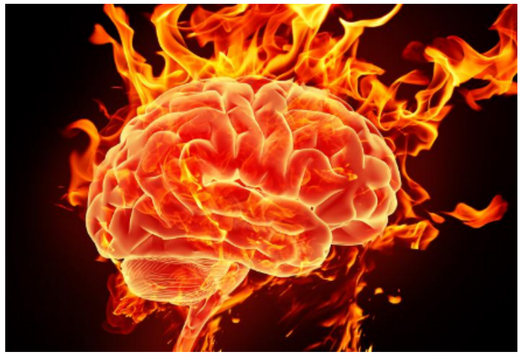
Primed for migraine?

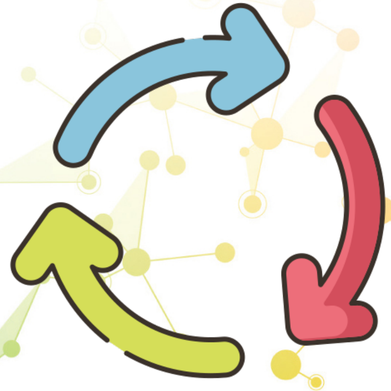
(epi)genetics

Deficiencies - B2,6,12, Mg,Vit D, CoQ10, carnitine

Food reaction = inflammation

No 1 source of free radicals





Vicious cycle

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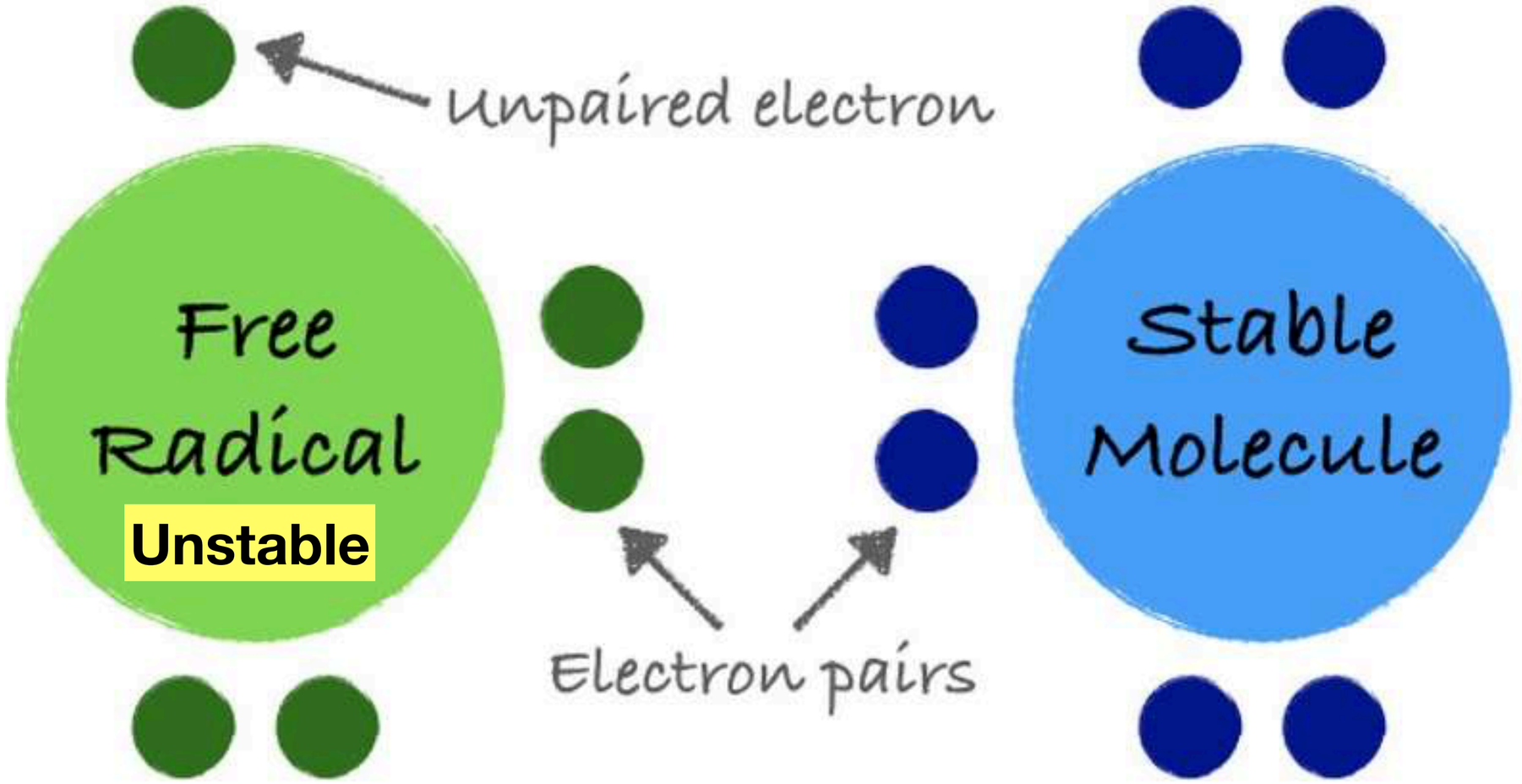

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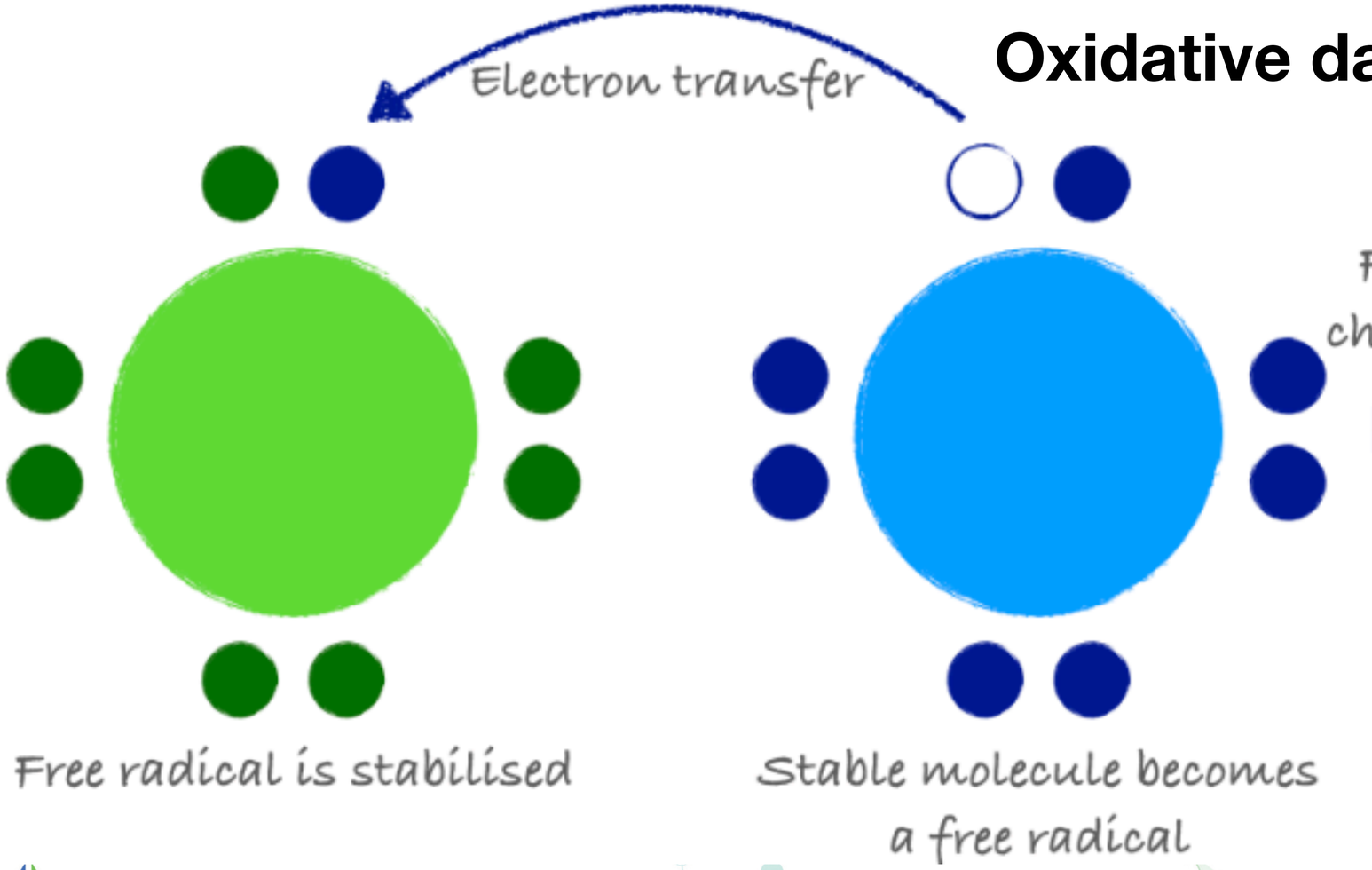
Oxidative damage



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“Co-morbidities”

AMERICAN MIGRAINE FOUNDATION

For many patients, migraine is associated with other illnesses such as:

- Depression
- Anxiety
- Stroke
- Irritable bowel syndrome
- Epilepsy
- Hypertension

Fibromyalgia
Endometriosis
Interstitial cystitis

- Chasing symptoms and labels not fundamental root causes

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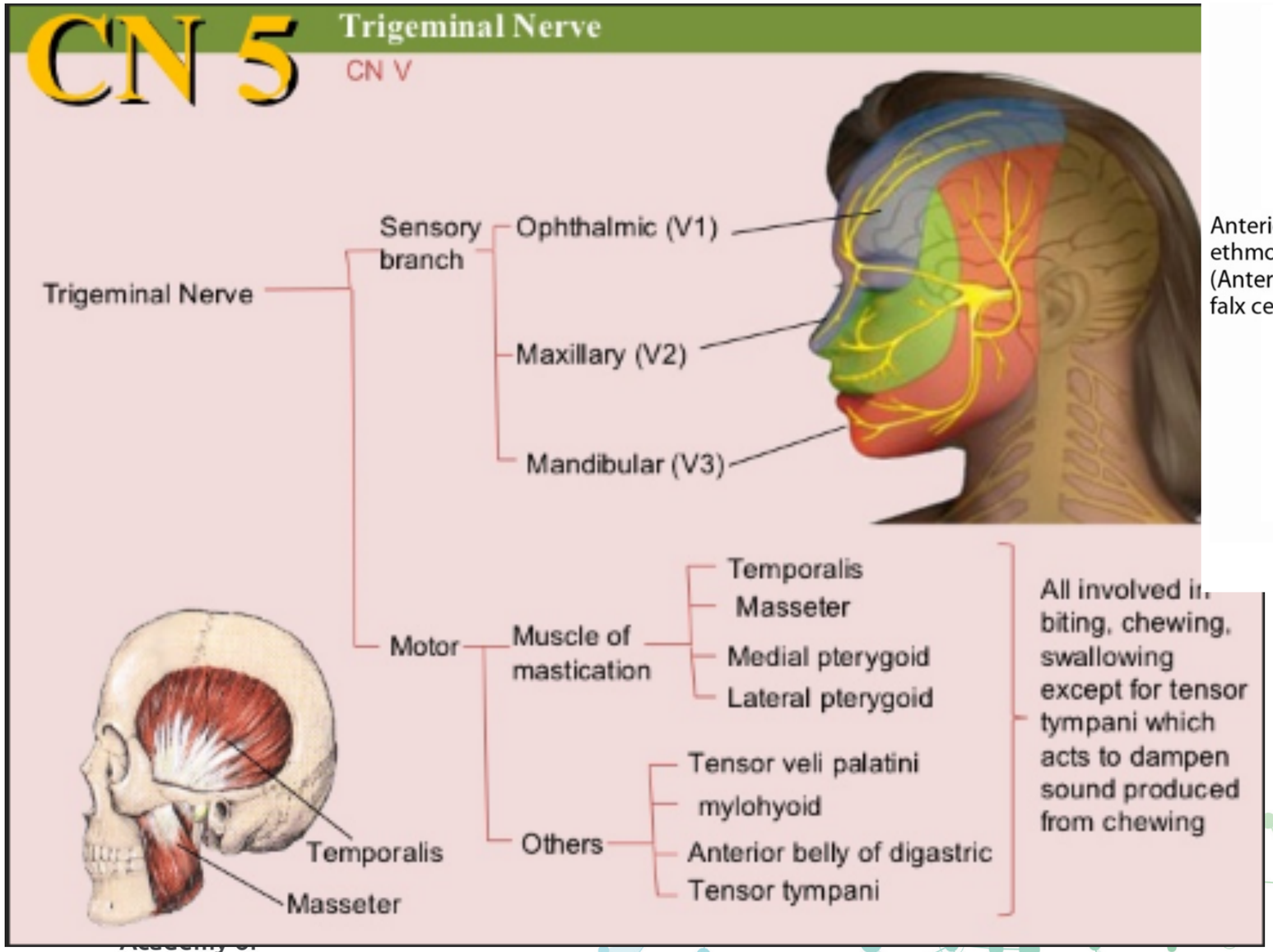
Migraine pain felt via trigeminal Nerve



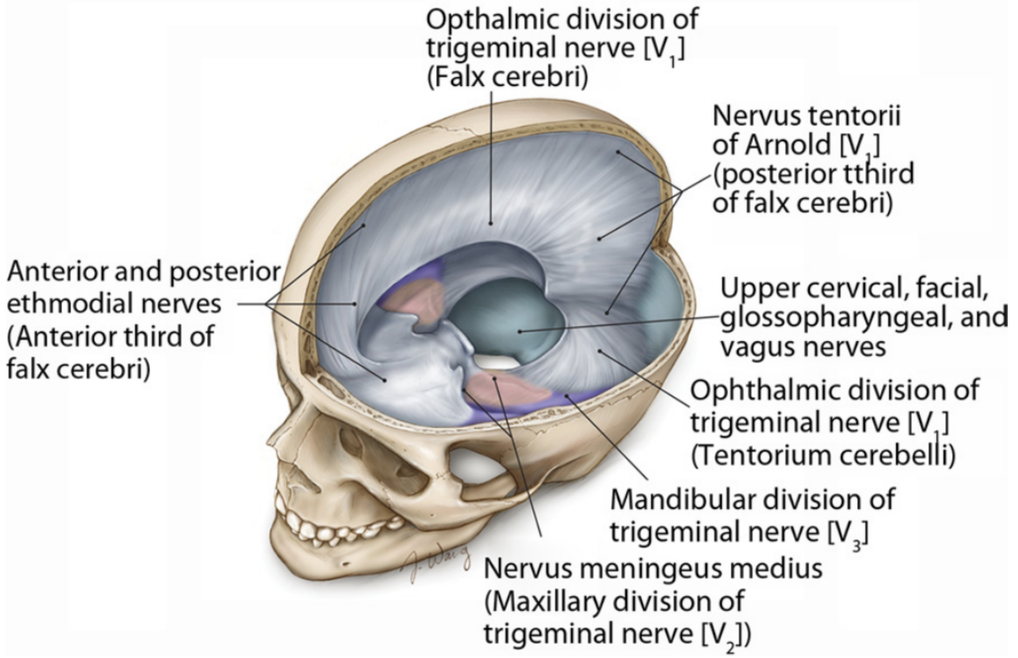


Figure 1. A summary of cranial dura innervation known to date.

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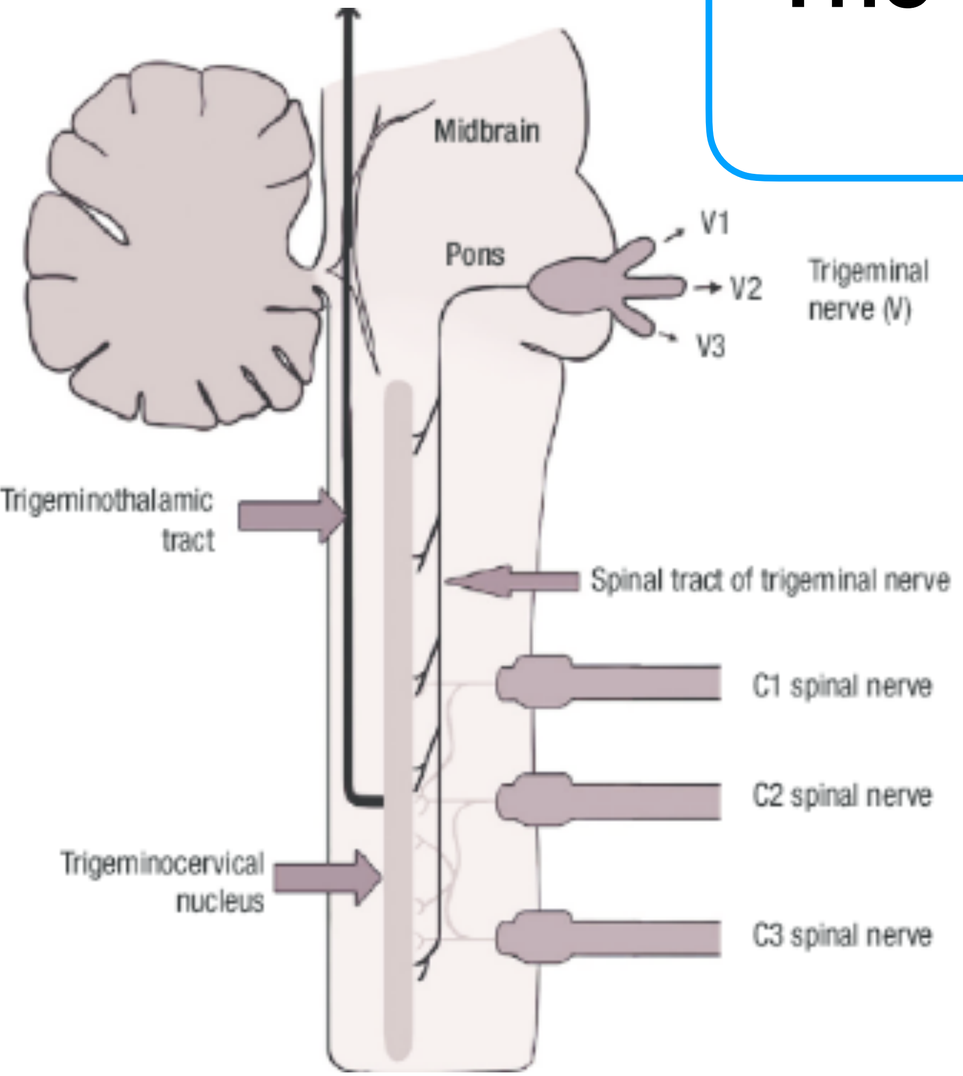
The pain of migraine felt via trigeminal nerve



<u>Trigemino-cervical nucleus</u>
Cervicogenic-Trigeminal nerve convergence

C1-3 nerves/structures can stimulate TGN, and thus contribute to migraine

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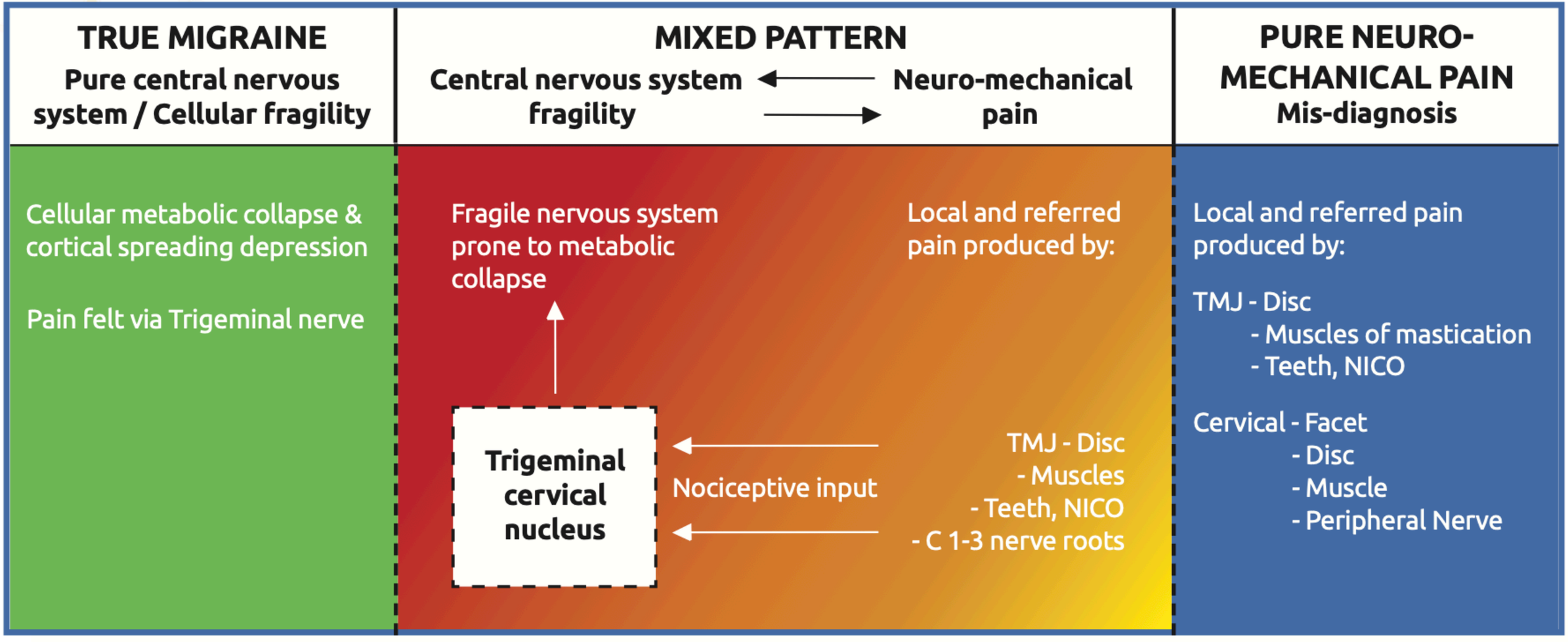
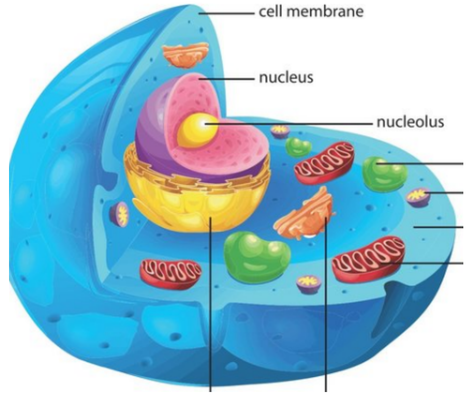
Migraine spectrum



Primary mechanism for Priming



Dysfunctional Mitochondria

Excess free radicals + inadequate anti-oxidant status
Overwhelming Oxidative damage

Sustained Inflammatory Response (SIR)
: Systemic & neurological

Metabolic collapse of cells leads to a massive self perpetuating wave of neuronal depolarisation

= Cortical spreading depression

Massive trigeminal nerve hypersensitivity/pain
Mild brain oedema

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Pure/true migraine is a cellular event

B2,6, folate
B12
Co Q10
Carnitine
Magnesium
Vitamin D

Good -
Missing / Add

- Fresh whole foods
- Fluids
- Protein & fats
- Vitamins & minerals
- Antioxidants
- Bacteria
- Hormones
- Sunlight rest & relaxation
- Sleep
- Love & connection

Bad -
Avoid / Remove, Reduce

- Bad food & Food sensitivities
- Obesity
- External Toxins
  - Heavy Metals
  - Allergens
  - Environmental
- Internal Toxins
  - Bad Bacteria / Infections
  - Homocysteine
- Drugs / Prescription Medications
- Bad Sleep
- Emotional Stress

Dysfunctional Mitochondria

Excess Free Radicals + Low Antioxidant Status = Oxidative Damage

Sustained Inflammatory Response (SIR) : Systemic & neurological

Neuromechanical dysfunction - Cervical spine (joints, nerves, muscles), TMJ, dental

CT failure - macro/micro

Neuronal fragility/instability

NMDAr

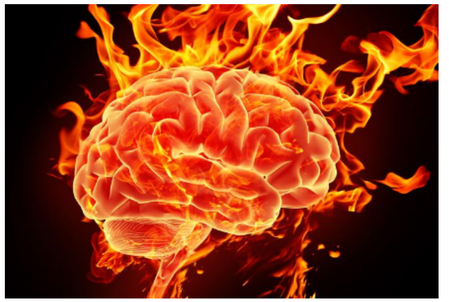
(PNS/CNS hypersensitivity)

Metabolic collapse = Cortical spreading depression

Pain via trigeminal nerve

"migraine"

Brain on fire



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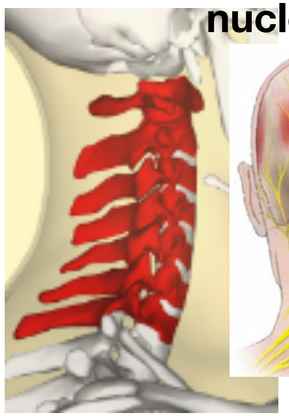
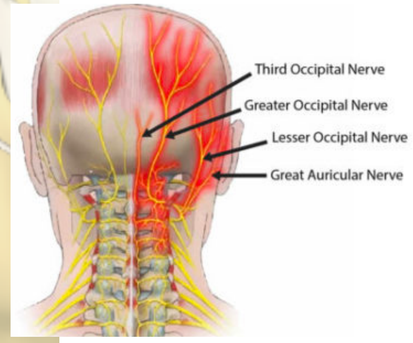
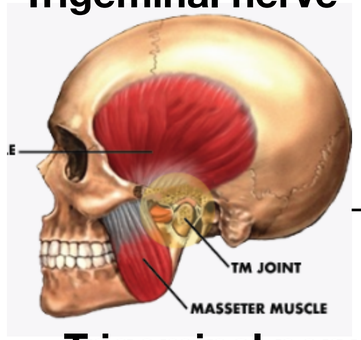
C1-3 > Trigemino-cervical nucleus





Trigeminal nerve



Trigeminal nerve

Neuromechanical Dysfunction

Connective tissue failure

Macro/micro

Diagnosed & undiagnosed (nervous system drivers)

Migraine

Metabolic collapse = cortical spreading /depression

Brain / neuronal fragility

PNS/CNS Hypersensitivity

Dysfunctional Mitochondria

Excess free radicals + inadequate anti-oxidant status
Overwhelming Oxidative damage

Sustained Inflammatory Response (SIR)
: Systemic & neurological

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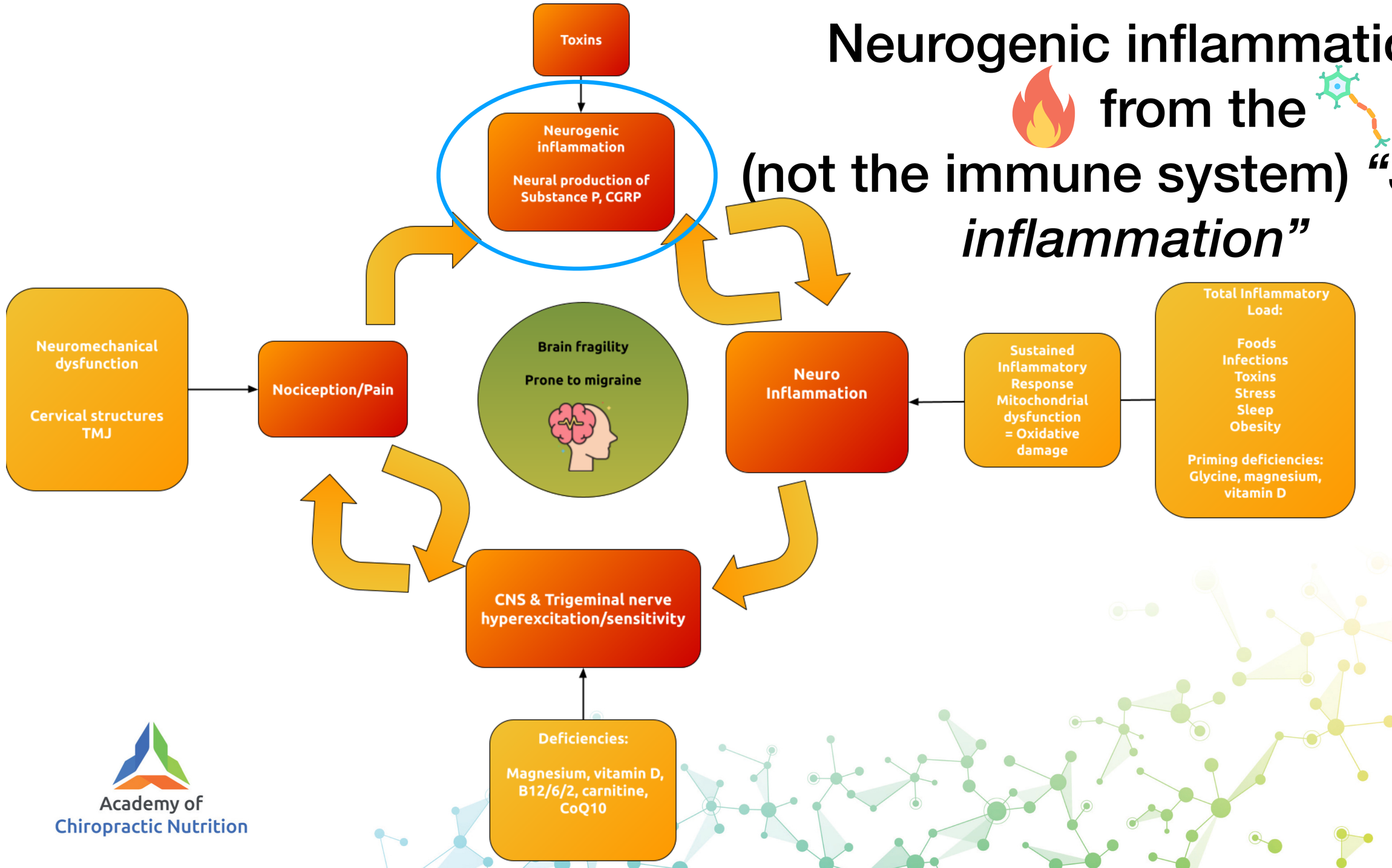
Neurogenic inflammation from the (not the immune system) *“Sterile inflammation”*



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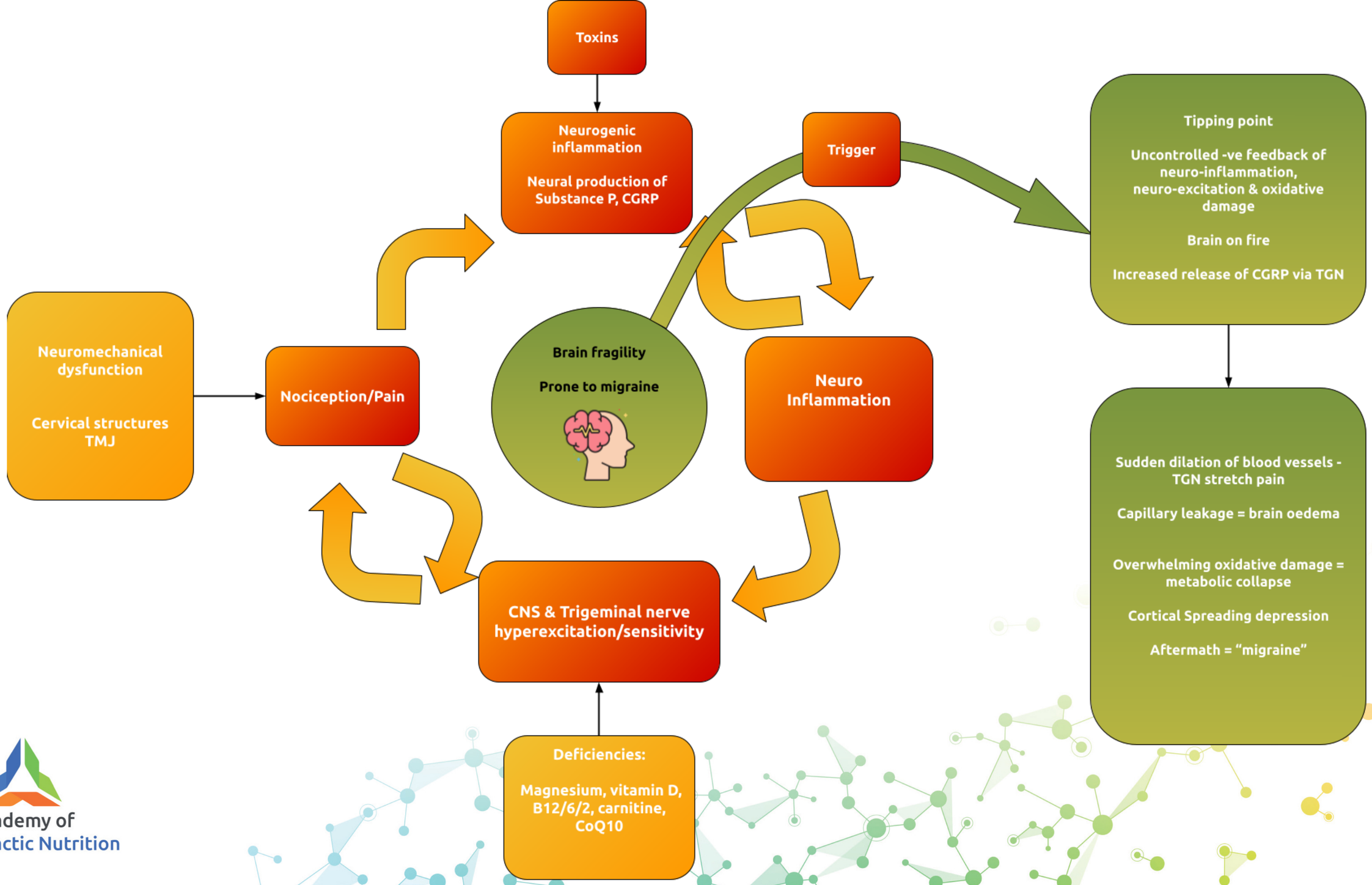

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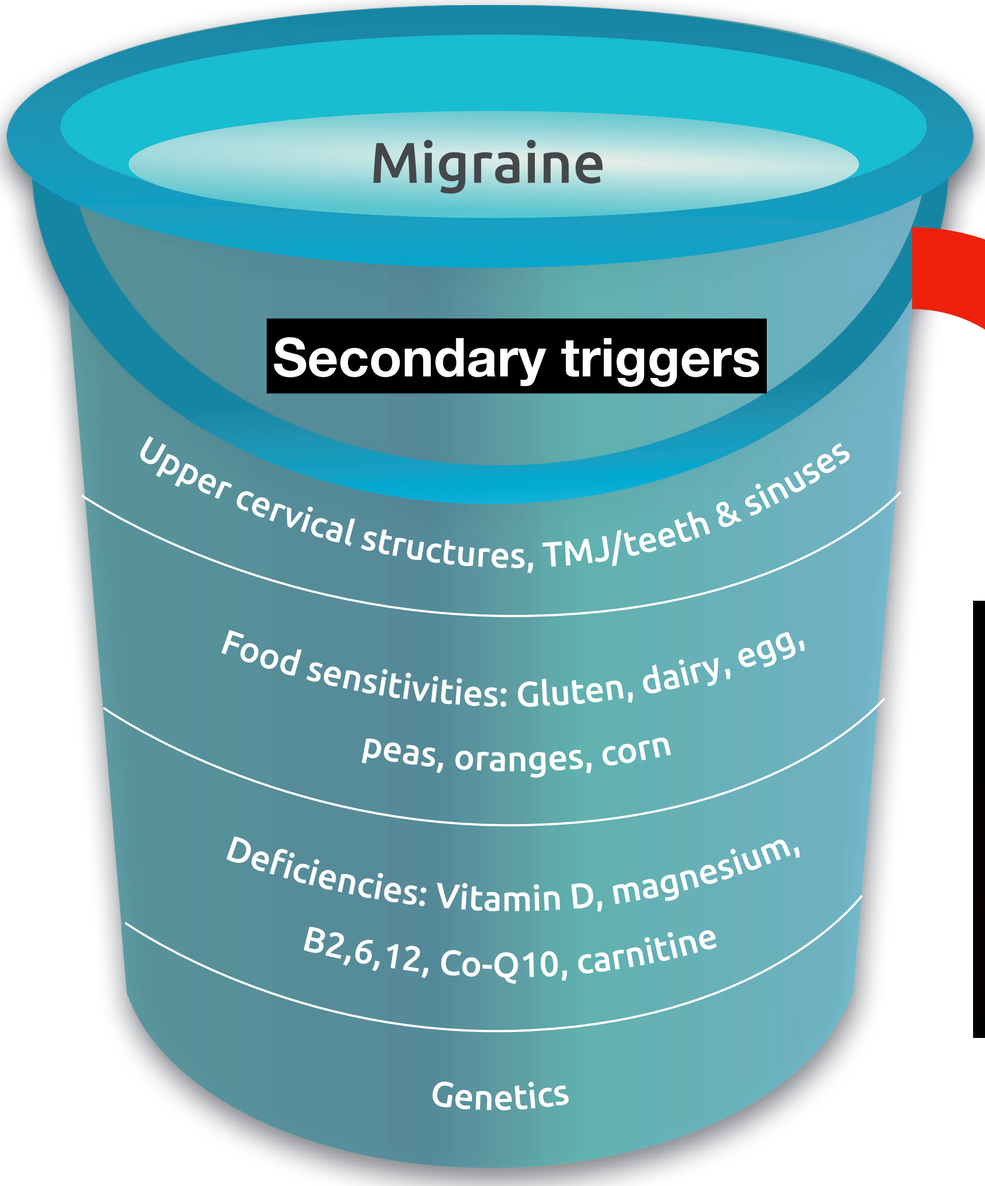
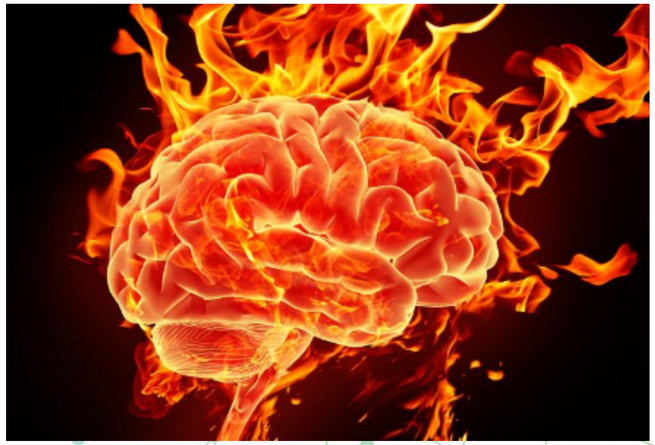
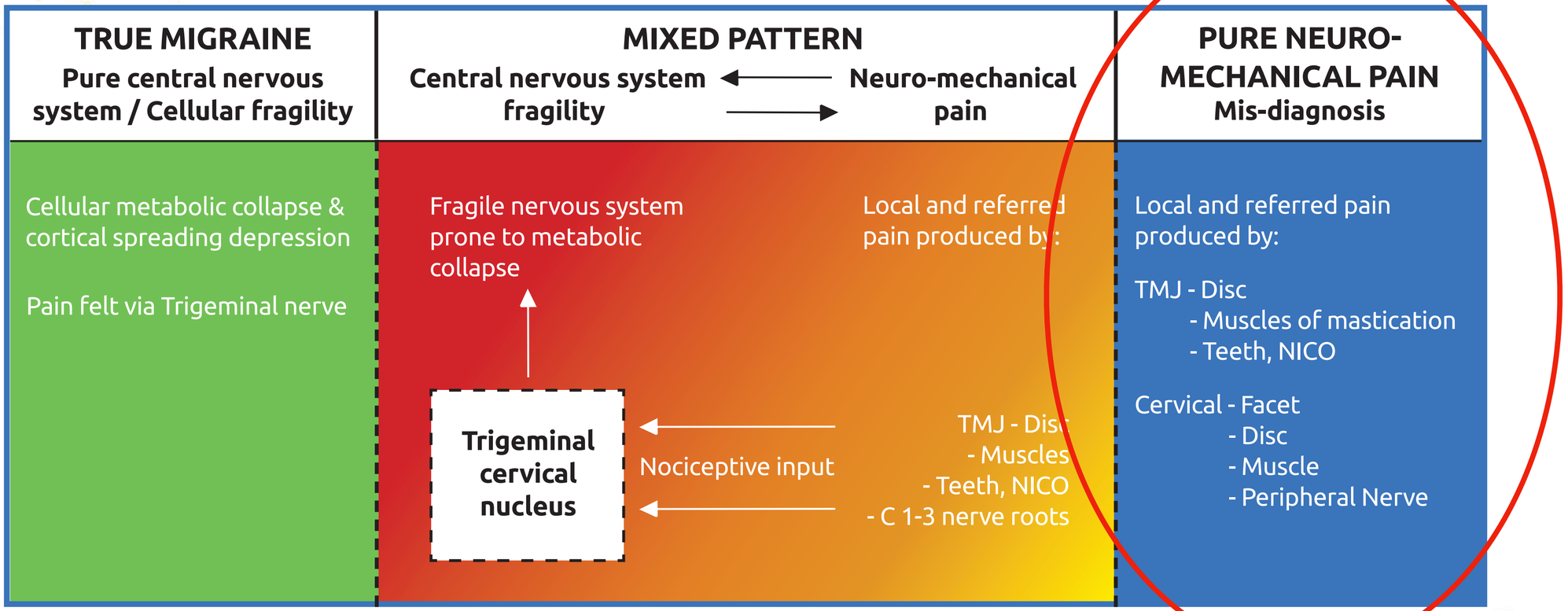


- Primary keystone issues for why patients are *“primed”* for migraine B2,6,12, mg, vitamin D, CoQ10, carnitine & foods - immune reaction
- Genetics tendency partly around methylation
- VS secondary downstream issues that *“trigger”* - stress, bright light, sleep changes, amines/nitrites/ MSG/sulphites (chocolate, wine) hormones

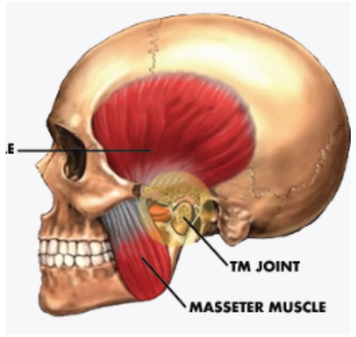


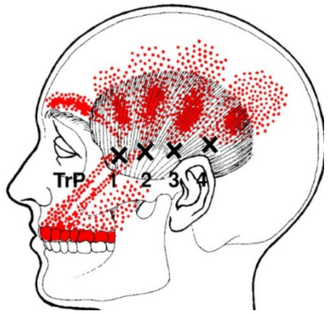
Migraine spectrum



Mis-diagnosis

- Cervicogenic - facet, peripheral nerve (GON), muscle TP referral
- TMD -





Temporalis TP = “Tension headache”

- Dental - Un-diagnosed tooth issues - filling (direct pain or affecting occlusion), infection (NICO lesion)

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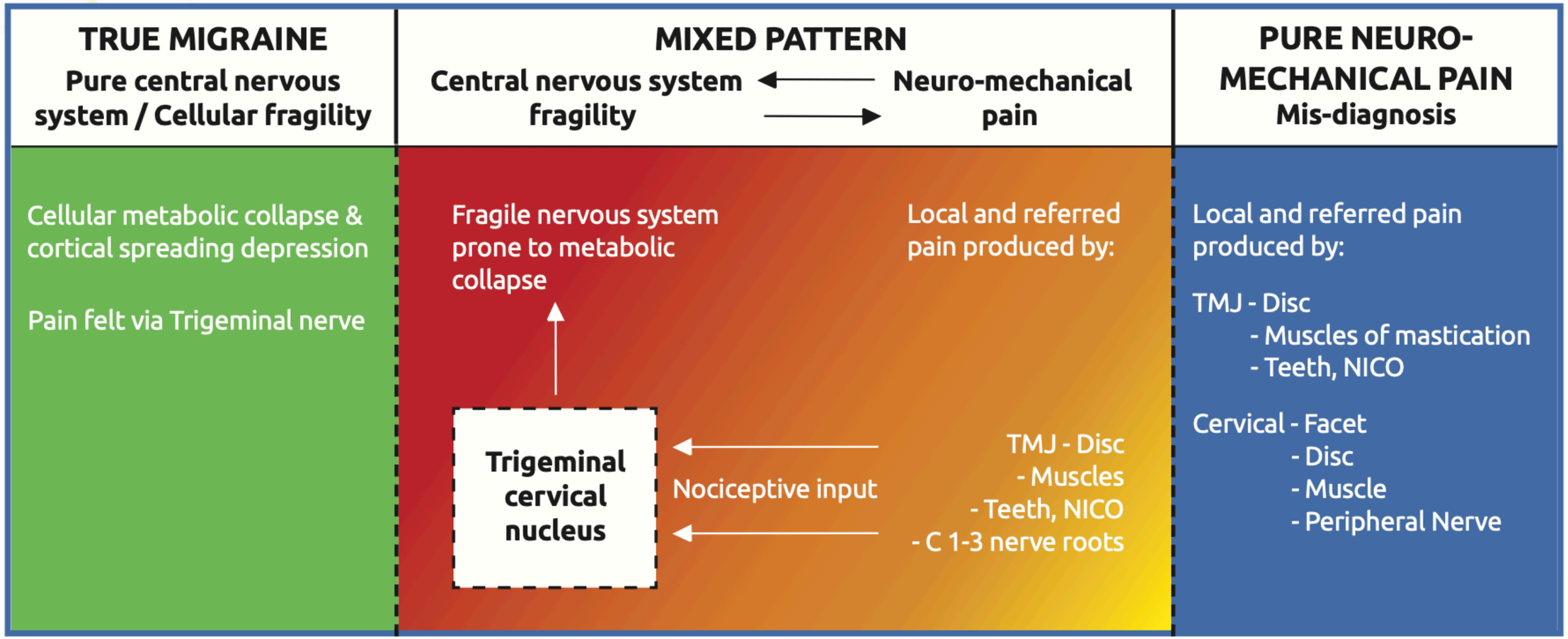
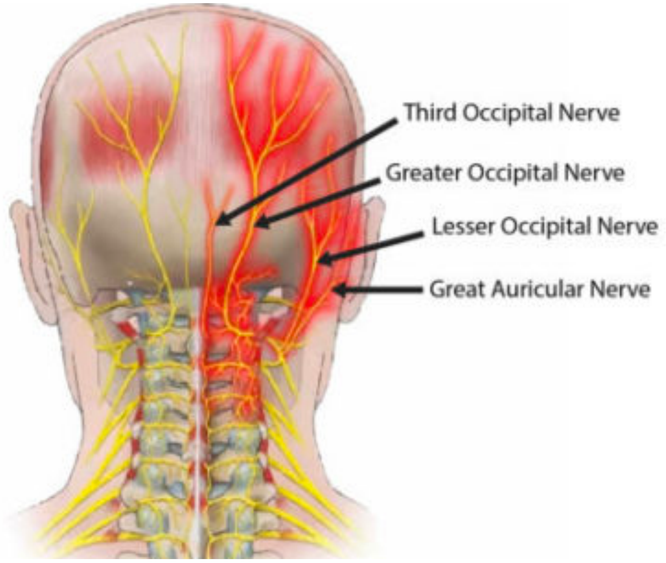
Mixed pattern
“normal” h/a
plus occasional true migraines

Migraine spectrum



Ch



Greater Occipital Nerve Block for the Treatment of Chronic Migraine Headaches: A Systematic Review and Meta-Analysis

Orr Shauly, B.S.
Daniel J. Gould, M.D., Ph.D.
Soma Sahai-Srivastava, M.D.
Ketan M. Patel, M.D.

Los Angeles, Calif.

2019

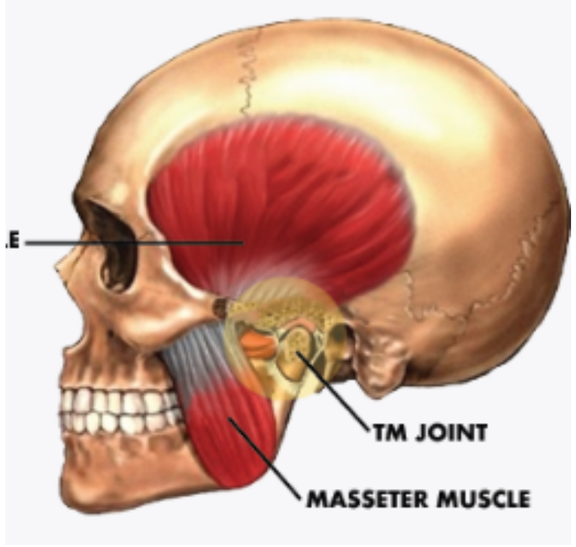
Mixed results - "migraine" is a spectrum thus some patients have no response other huge

Forest plot for the meta-analysis of headache days per month.

	Sample Size		No. Headaches / Month Mean (SD)				
Source	Intervention	Placebo	Intervention	Placebo	Weight	Mean Difference (95% CI)	
Gul et al., 2016	22	22	6.3 (1.9)	19.1 (6.3)	11%	-12.8	(-16.09 - -9.51)
Dilli et al., 2015	33	30	9.3 (4.8)	10.4 (6.8)	15%	-1.1	(-5.26 - 3.06)
Ashkenazi et al., 2018	19	18	5.5 (4.9)	14.3 (15.1)	9%	-8.8	(-16.74 - -0.86)
Cuadrado et al., 2017	18	18	13.6 (10.4)	18.0 (8.8)	9%	-4.4	(-11.21 - 2.41)
Inan et al., 2015	39	33	8.8 (4.8)	13.2 (6.7)	17%	-4.4	(-8.52 - -0.28)
Karadas et al., 2016	35	35	14.8 (4.7)	16.9 (4.3)	17%	-2.1	(-5.29 - 1.09)
Kashipazha et al., 2014	24	24	8.4 (3.5)	9.4 (3.8)	12%	-1	(-3.58 - 1.58)
Naja et al., 2006	24	23	11.0 (3.7)	14.1 (5.4)	11%	-3.1	(-6.37 - 0.17)
Total	**214**	**203**			**100%**	**-4.3**	**(-6.51 - -2.09)**

A new biofeedback approach for the control of awake bruxism and chronic migraine headache: utilization of an awake posterior interocclusal device

Uma nova abordagem via *biofeedback* para o controle do bruxismo de vigília e de enxaqueca crônica: utilização de um dispositivo interoclusal posterior em vigília



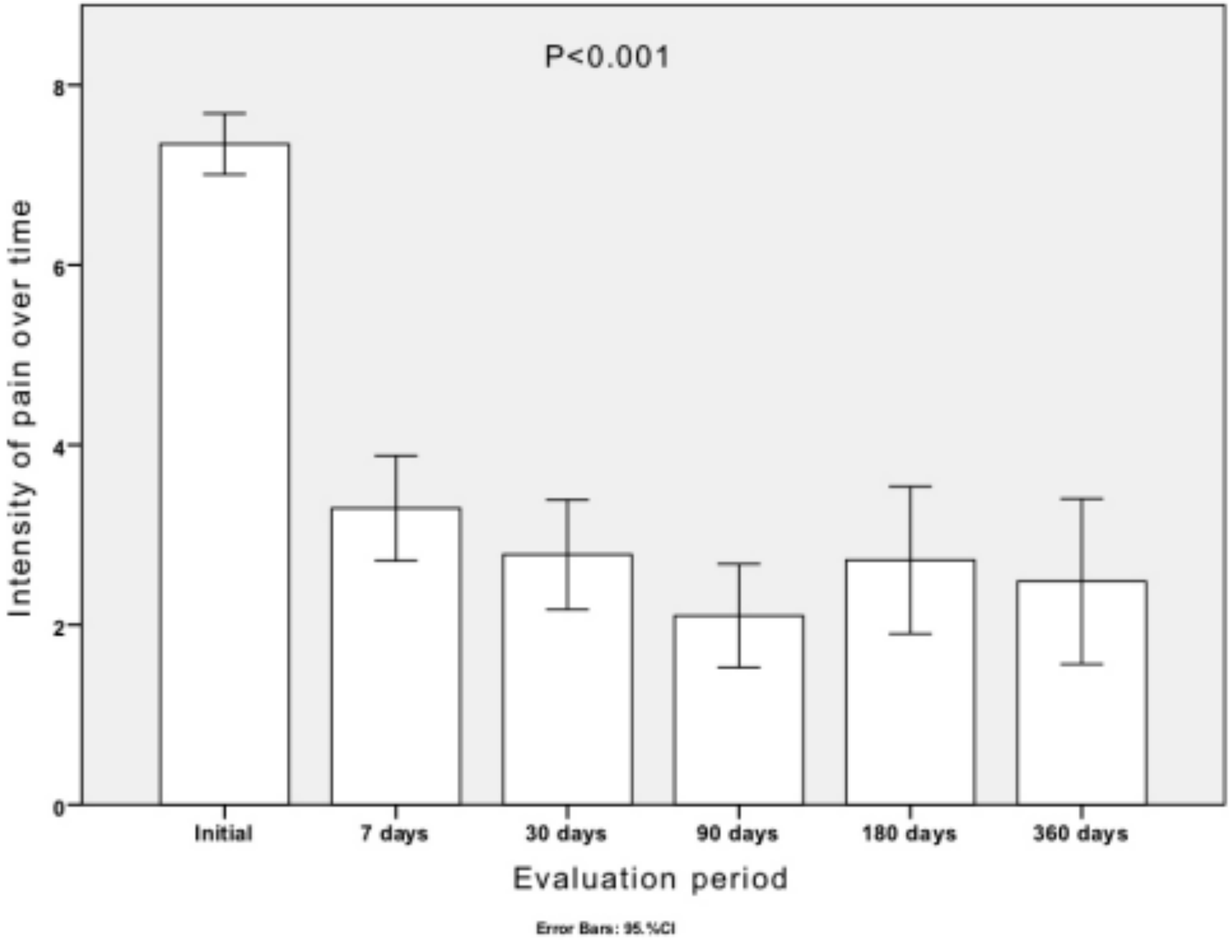


Figure 1. Average intensity of pain, including chronic migraine headache.

appliance removed at 90 days

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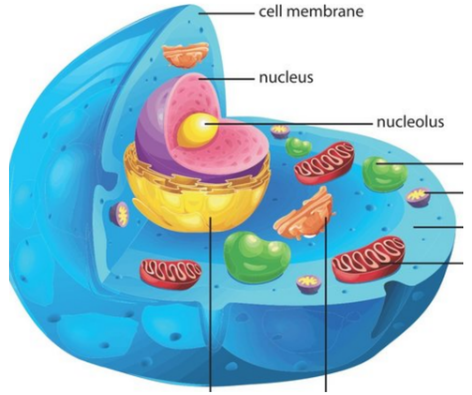
Pure/true migraine is a cellular event



Smouldering fire "PRIMED"

Dysfunctional Mitochondria

Excess free radicals + inadequate anti-oxidant status
Overwhelming Oxidative damage

Sustained Inflammatory Response (SIR)
: Systemic & neurological

Trigger

Metabolic collapse of cells leads to a massive self perpetuating wave of neuronal depolarisation

= Cortical spreading depression

Massive trigeminal nerve hypersensitivity/pain
Mild brain oedema

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ORIGINAL ARTICLE

Reactive oxygen species initiate a metabolic collapse in hippocampal slices: potential trigger of cortical spreading depression

Anton Malkov[1,2,5], Anton I Ivanov[1,5], Irina Popova[1,2], Marat Mukhtarov[1,3], Olena Gubkina[1], Tatsiana Waseem[1,4], Piotr Bregestovski[1] and Yuri Zilberter[1]

Reactive oxygen species = free radical

mechanisms of which are poorly understood. We suggest that ROS accumulation might also be the primary trigger of CSD. Indeed, we found that Tempol strongly reduced occurrence of CSD *in vivo*, suggesting that ROS accumulation may be a key mechanism of CSD initiation.

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PubMed.gov

migraine mitochondria

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10 Cite Share

A possible role for mitochondrial dysfunction in **migraine**.

Stuart S, Griffiths LR.

Mol Genet Genomics. 2012 Dec;287(11-12):837-44. doi: 10.1007/s00438-012-0723-7. Epu
Oct 7.

PMID: 23052833 Review.

Mitochondrial dysfunction in **migraine**.

Yorns WR Jr, Hardison HH.

Semin Pediatr Neurol. 2013 Sep;20(3):188-93. doi: 10.1016/j.spen.2013.09.002.

PMID: 24331360

Mitochondria in **migraine** pathophysiology - does epigenetics play a role?

Fila M, Pawłowska E, Blasiak J.

Arch Med Sci. 2019 Jul;15(4):944-956. doi: 10.5114/aoms.2019.86061. Epub 2019 Jun 20.

PMID: 31360189 Free PMC article.

Energy Metabolism Impairment in **Migraine**.

Cevoli S, Favoni V, Cortelli P.

Curr Med Chem. 2019;26(34):6253-6260. doi: 10.2174/0929867325666180622154411.

PMID: 29932030 Review.

CoEnzyme Q10 and riboflavin: the mitochondrial connection.

Markley HG.

Headache. 2012 Oct;52 Suppl 2:81-7. doi: 10.1111/j.1526-4610.2012.02233.x.

PMID: 23030537 Review.

The metabolic face of **migraine** - from pathophysiology to treatment.

Gross EC, Lisicki M, Fischer D, Sándor PS, Schoenen J.

Nat Rev Neurol. 2019 Nov;15(11):627-643. doi: 10.1038/s41582-019-0255-4. Epub 2019 Oct 4.

PMID: 31586135 Free article. Review.

Riboflavin and **migraine**: the bridge over troubled **mitochondria**.

Colombo B, Saraceno L, Comi G.

Neurol Sci. 2014 May;35 Suppl 1:141-4. doi: 10.1007/s10072-014-1755-z.

PMID: 24867851

Mitochondria, magnesium and **migraine**.

Welch KM, Ramadan NM.

J Neurol Sci. 1995 Dec;134(1-2):9-14. doi: 10.1016/0022-510x(95)00196-1.

PMID: 8747836 Review.

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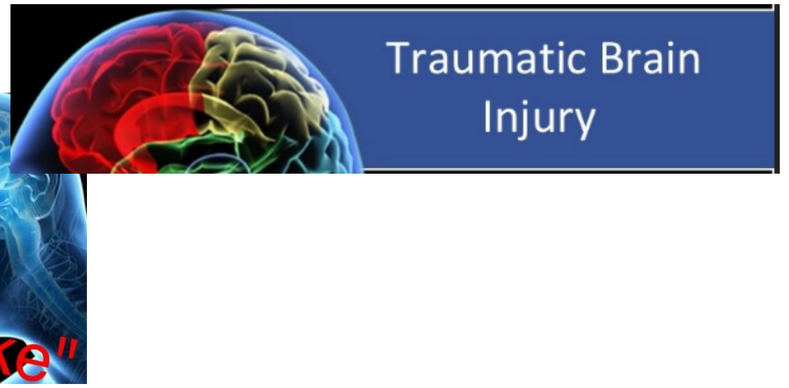
Cortical spreading depression



Why all migraine patients should be treated with magnesium

Alexander Mauskop · Jasmine Varughese

experienced during a migraine. Cortical spread depression (CSD) is a phenomenon that can explain the aura of migraines (Strong 2003). Aura consists of a variety of sensory warning signs or symptoms, such as blind spots, flashes of light, or tingling sensations in the hands or face. CSD occurs when the cerebral cortex is stimulated by chemical or electric signals. This stimulation leads to an excitation of the cerebral cortex, which is followed by extended depolarization of cortical neurons that gradually spreads through the cortex. This process is followed by a wave of oligemia. CSD has been documented by magnetic resonance imaging (Hadjikhani et al. 2001), epidural electrophysiological recordings (Fabricius et al. 2006; Strong et al. 2002), and intracortical multiparametric electrodes (Mayevsky et al. 2006)

Cytokines, SP, CGRP
Glutamate, Quinolinic acid
homocysteine

Dysafferentation/nociception

reduced blood volume
(vasoconstriction)

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Metabolic disturbances in patients suffering from migraine, stroke, epilepsy, subarachnoid hemorrhage, and traumatic brain injury may result in a spontaneous induction of deleterious changes in multiple neuronal parameters that cause a self-propagating wave of cellular depolarization in the cerebral cortex—cortical spreading depression (CSD).[6–9] As CSD is a pathological phenomena of great

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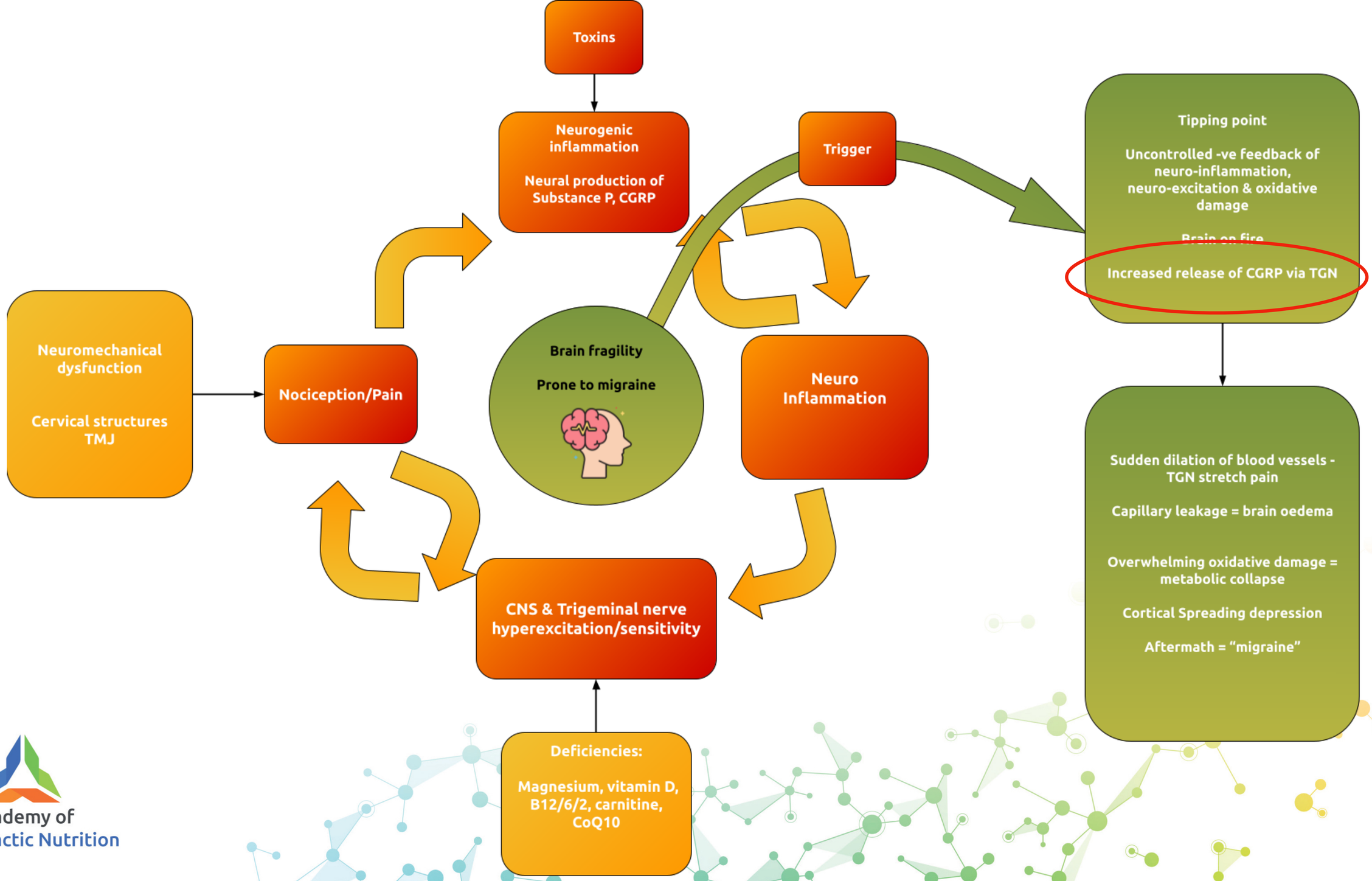

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Final common pathway

- Calcitonin gene related peptide CGRP - potent vasodilator, mast cell degranulation - histamine release & cytokines (part of inflammation & oxidation overwhelm - tipping point)
- Released by TGN response to   and nociception (TMJ/Cx1-3),
- **neurogenic inflammation**

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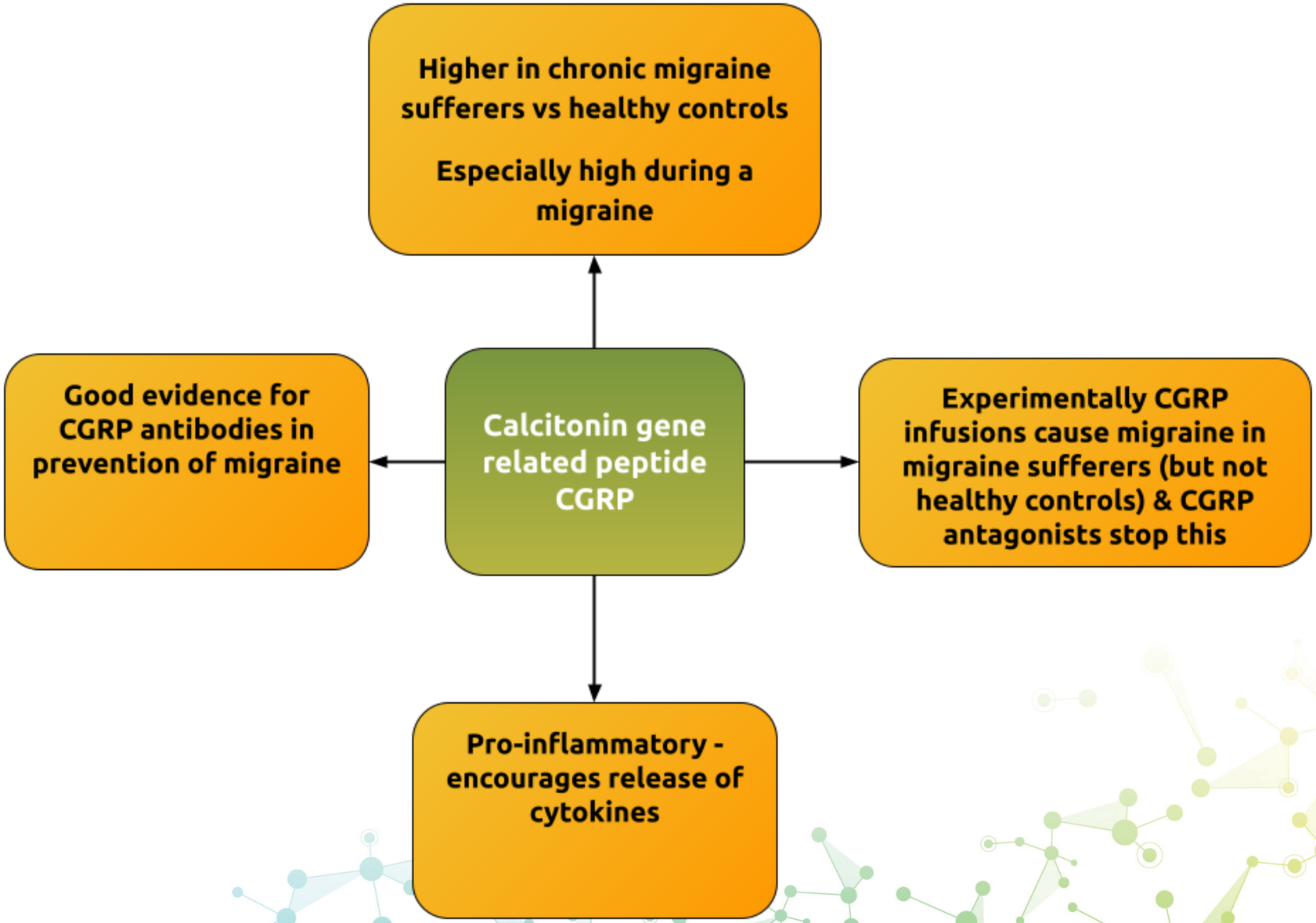

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PAIN

Erenumab: Is This Migraine Medication a Miracle Drug?

If your migraine symptoms are out of control, erenumab may offer the prospect of relief.

'Miracle' once-a-month drug to treat migraines approved for use on NHS

13 March 2020, 16:16

HEALTH & MEDICINE

New drugs that block a brain chemical are game changers for some migraine sufferers

Options to prevent and treat the severe headaches are becoming available

The cost for one kind of CGRP migraine treatment is about $6,900 per year or $575 per month. Other types may have slightly different costs. CGRP drugs are new and may cost more than other kinds of migraine treatment.

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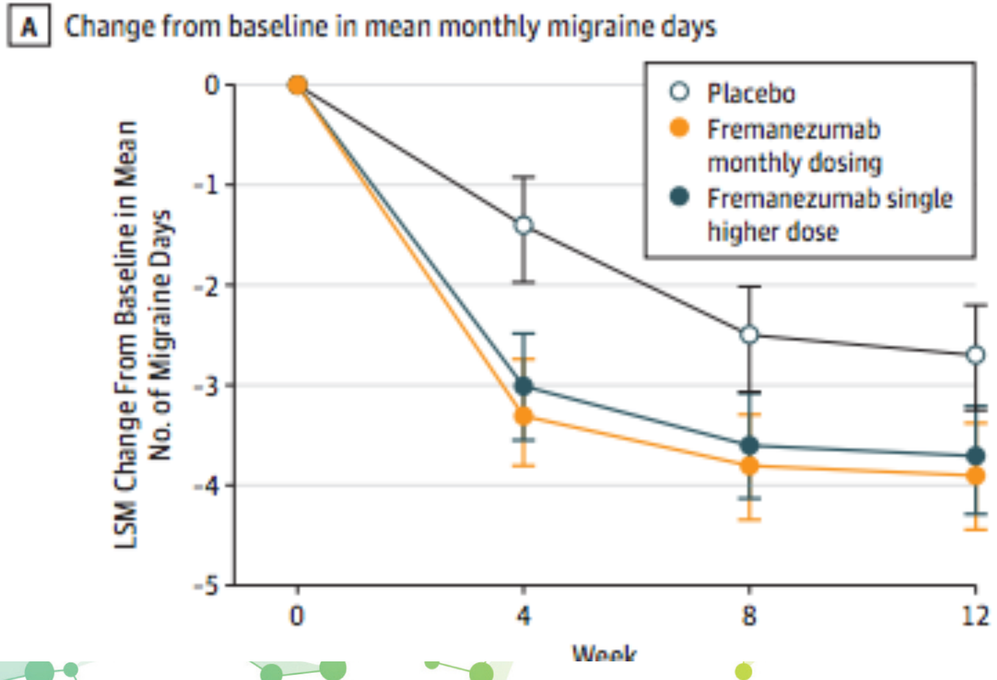
JAMA | Original Investigation

Effect of Fremanezumab Compared With Placebo for Prevention of Episodic Migraine
A Randomized Clinical Trial

INTERVENTIONS Patients were randomized 1:1:1 to receive subcutaneous monthly dosing of fremanezumab (n = 290; 225 mg at baseline, week 4, and week 8); a single higher dose of fremanezumab, as intended to support a quarterly dose regimen (n = 291; 675 mg of fremanezumab at baseline; placebo at weeks 4 and 8); or placebo (n = 294; at baseline, week 4, and week 8).

RESULTS Among 875 patients who were randomized (mean age, 41.8 [SD, 12.1] years; 742 women [85%]), 791 (90.4%) completed the trial. From baseline to 12 weeks, mean migraine days per month decreased from 8.9 days to 4.9 days in the fremanezumab monthly dosing group, from 9.2 days to 5.3 days in the fremanezumab single-higher-dose group, and from 9.1 days to 6.5 days in the placebo group. This resulted in a difference with monthly dosing vs placebo of -1.5 days (95% CI, -2.01 to -0.93 days; $P < .001$) and with single higher dosing vs placebo of -1.3 days (95% CI, -1.79 to -0.72 days; $P < .001$). The most common adverse events that led to discontinuation were injection site erythema (n = 3), injection site induration (n = 2), diarrhea (n = 2), anxiety (n = 2), and depression (n = 2).

45% reduction



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Migraine (and associated conditions) underpinned by mito dysfunction, inflammation and massive oxidative damage - can we ethically leave these in place & treat downstream consequences/symptoms?

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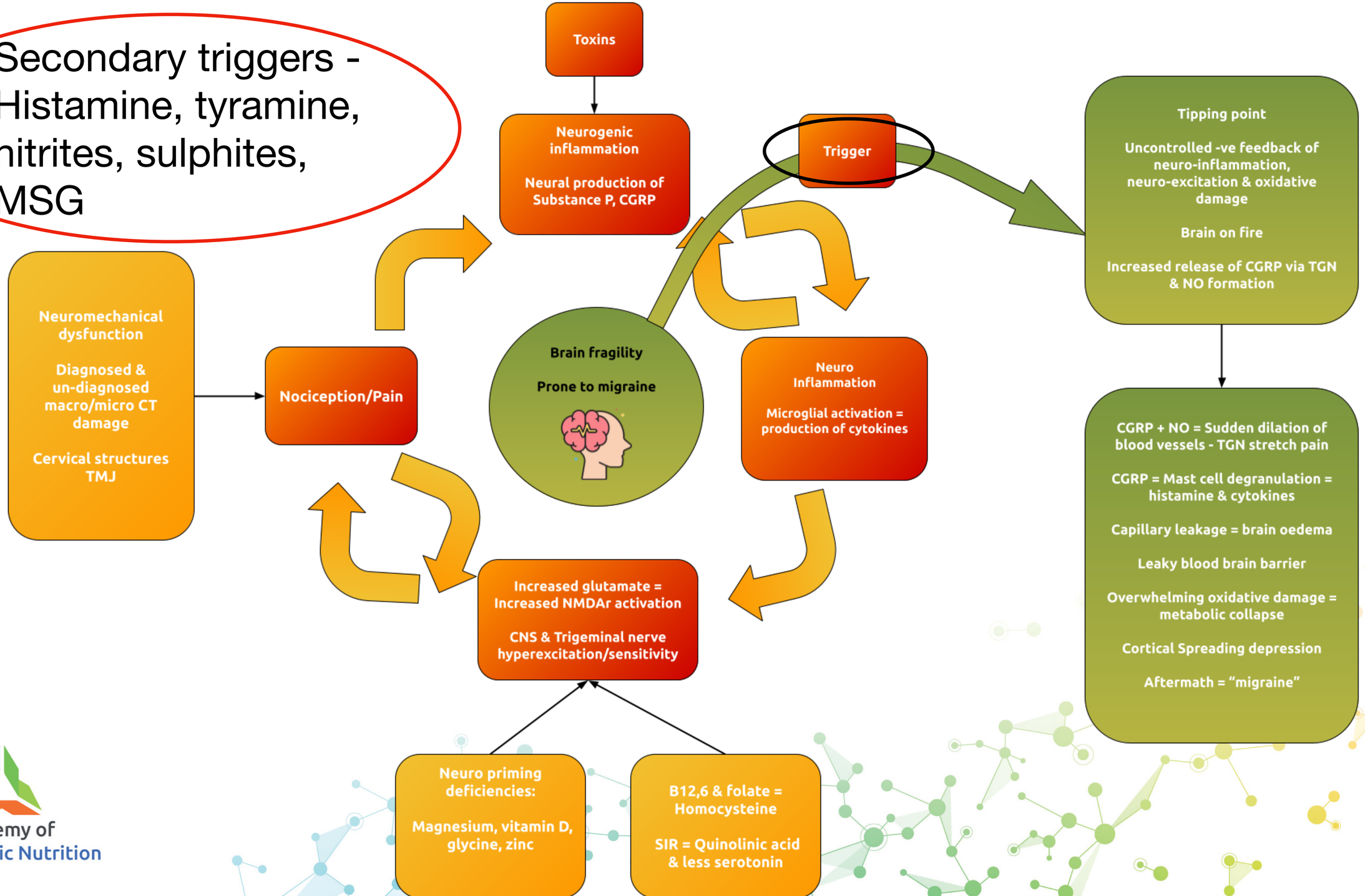
- Secondary triggers - Histamine, tyramine, nitrites, sulphites, MSG



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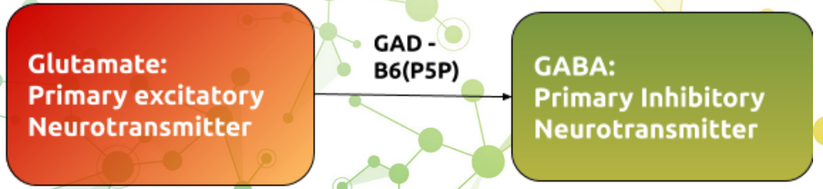
Secondary triggers

- Amines - tyramine

- Nitrites

- Sulfites

- MSG



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Food protein as a direct immune system trigger for inflammation - fills the bucket

Toxins

Neurogenic inflammation

Neural production of Substance P, CGRP

Neuromechanical dysfunction

Cervical structures TMJ

Nociception/Pain

Brain fragility

Prone to migraine

Neuro Inflammation

Sustained Inflammatory Response Mitochondrial dysfunction = Oxidative damage

Total Inflammatory Load:

Foods

Infections

Toxins

Stress

Sleep

Obesity

Priming deficiencies: Glycine, magnesium, vitamin D

CNS & Trigeminal nerve hyperexcitation/sensitivity

Deficiencies:

Magnesium, vitamin D, B12/6/2, carnitine, CoQ10

Big 5 foods

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Clinical point

- Chronic migraine patients are have almost always tried removing foods previously

- Usually based around classic amines or sulphites

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Clinical point

- Chronic migraine patients are have almost always tried removing foods previously
- Usually based around classic amines or sulphites

- Check if they did them individually or as a group? Response? and do they avoid them?
- Often have not removed foods based on immune sensitivity - Big 5 +

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THE LANCET
Volume 322, Issue 8355, 15 October 1983, Pages 865-869

IS MIGRAINE FOOD ALLERGY?: A Double-blind Controlled Trial of Oligoantigenic Diet Treatment

J Egger, J Wilson, C.M Carter, M.W Turner, J.F Soothill

3-4 weeks on one meat, one fruit, one veg, one carb

93% of 88 children with severe frequent migraine recovered on oligoantigenic diets; the causative foods were identified by sequential reintroduction, and the role of the foods provoking migraine was established by a double-blind controlled trial in 40 of the children. Most patients responded to several foods. Many foods were involved,

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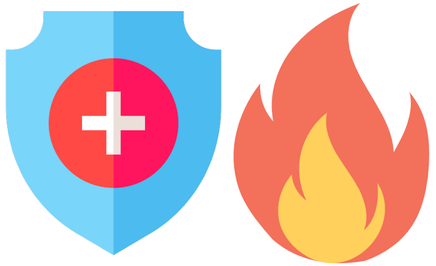
TABLE I—ASSOCIATED SYMPTOMS AND SIGNS

—	Patients completing oligoantigenic diet (88)		Patients completing trial (40)	
	Before diet	On diet	Group AP	Group PA
Abdominal pain, diarrhoea, flatulence	61	8	14	19
Behaviour disorder	41	5	12	16
Aches in limbs	41	7	12	17
Fits	14*	2	5	5
Permanent neurological signs	6	6	1	4
Rhinitis	34	15	5	9
Recurrent mouth ulcers	15	2	4	6
Vaginal discharge	11	1	3	5
Asthma	7	3	1	1
Eczema	6	3	3	4

*Sometimes coinciding with headaches in all 14: 9 had generalised or partial seizures, coinciding with headaches in all but 1.



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associated symptoms aka “co-morbidities”

TABLE III—NUMBER OF CHILDREN IN WHOM FOODS CAUSED SYMPTOMS

Food	n	Food	n	Food	n	Food	n
Cows' milk	27	Soya	7	White wheat flour	3	Vegetable oils	2
Egg	24	Tea	7	Artificial milk substitute	3	Lentils	2
Chocolate	22	Oats	6	Banana	3	Peas	2
Orange	21	Goats' milk	6	Strawberries	3	Ice cream	2
Wheat	21	Coffee	6	Melon	3	Rabbit	1
Benzoic acid	14	Peanuts	5	Carrots	3	Dates	1
Cheese	13	Bacon	4	Lamb	2	Avocado	1
Tomato	13	Potato	4	Rice	2	Rhubarb	1
Tartrazine	12	Yeast	4	Malt	2	Leek	1
Rye	12	Mixed nuts	4	Sugar	2	Lettuce	1
Fish	9	Apple	4	Ginger	2	Cucumber	1
Pork	9	Peaches	4	Honey	2	Cauliflower	1
Beef	8	Grapes	3	Pineapple	2	Mushrooms	1
Maize	8	Chicken				Runner beans	1

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TABLE IV—NON-SPECIFIC PROVOKERS OF MIGRAINE IN 38 PATIENTS

—	Before diet	On diet
Exercise	13	1
Trauma	11	1
Emotional	10	0
Perfumes and/or cigarette smoke	10	9
Travel	9	0
Bright light	5	0
Heat	2	1
Noise	2	0

- Primary keystone issues for why patients are *"primed"* for migraine
- vs secondary downstream issues that *"trigger"* - stress, bright light, sleep changes, chocolate, wine, hormones
- Genetics tendency partly around methylation and ion transport (membrane stability)

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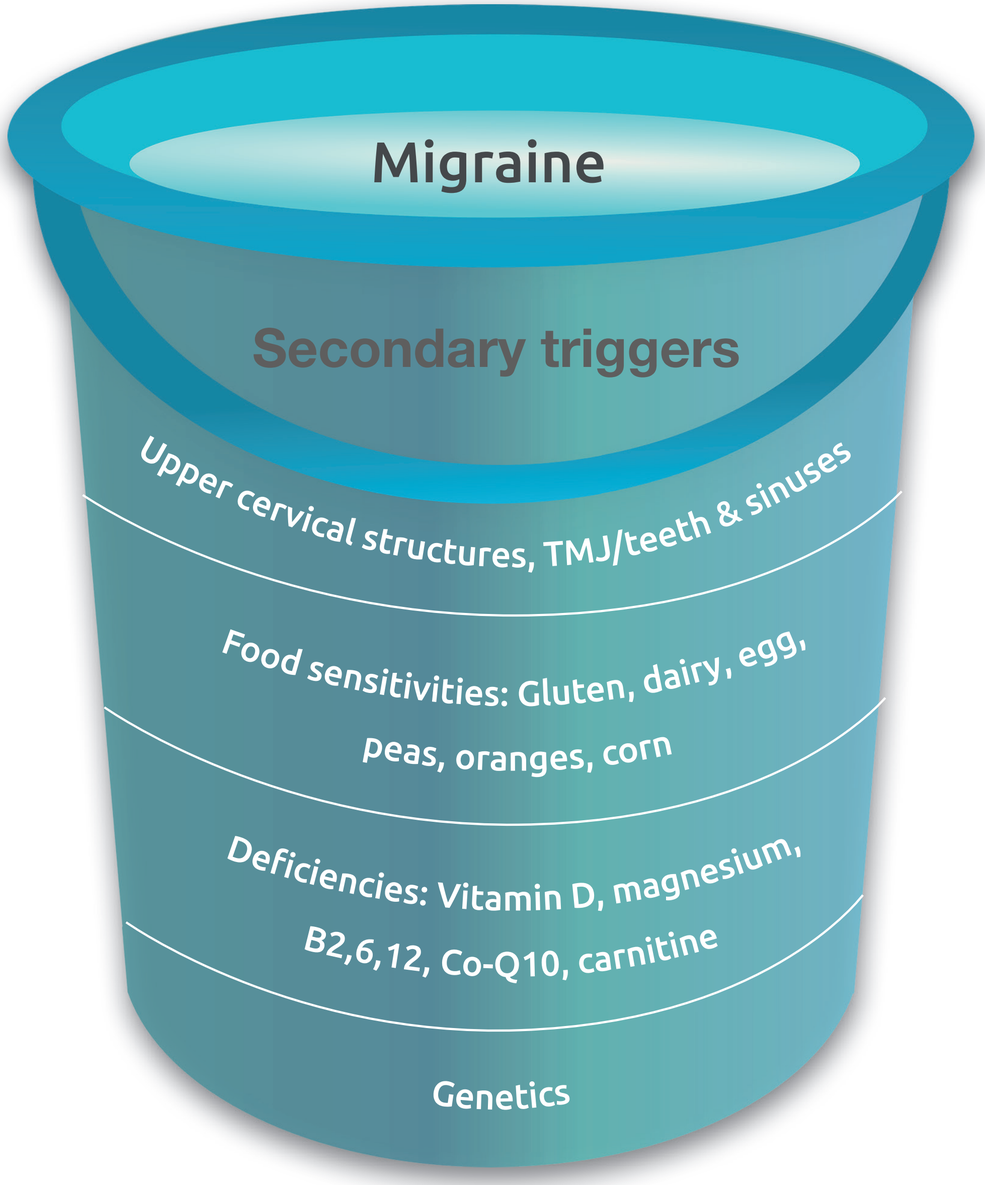

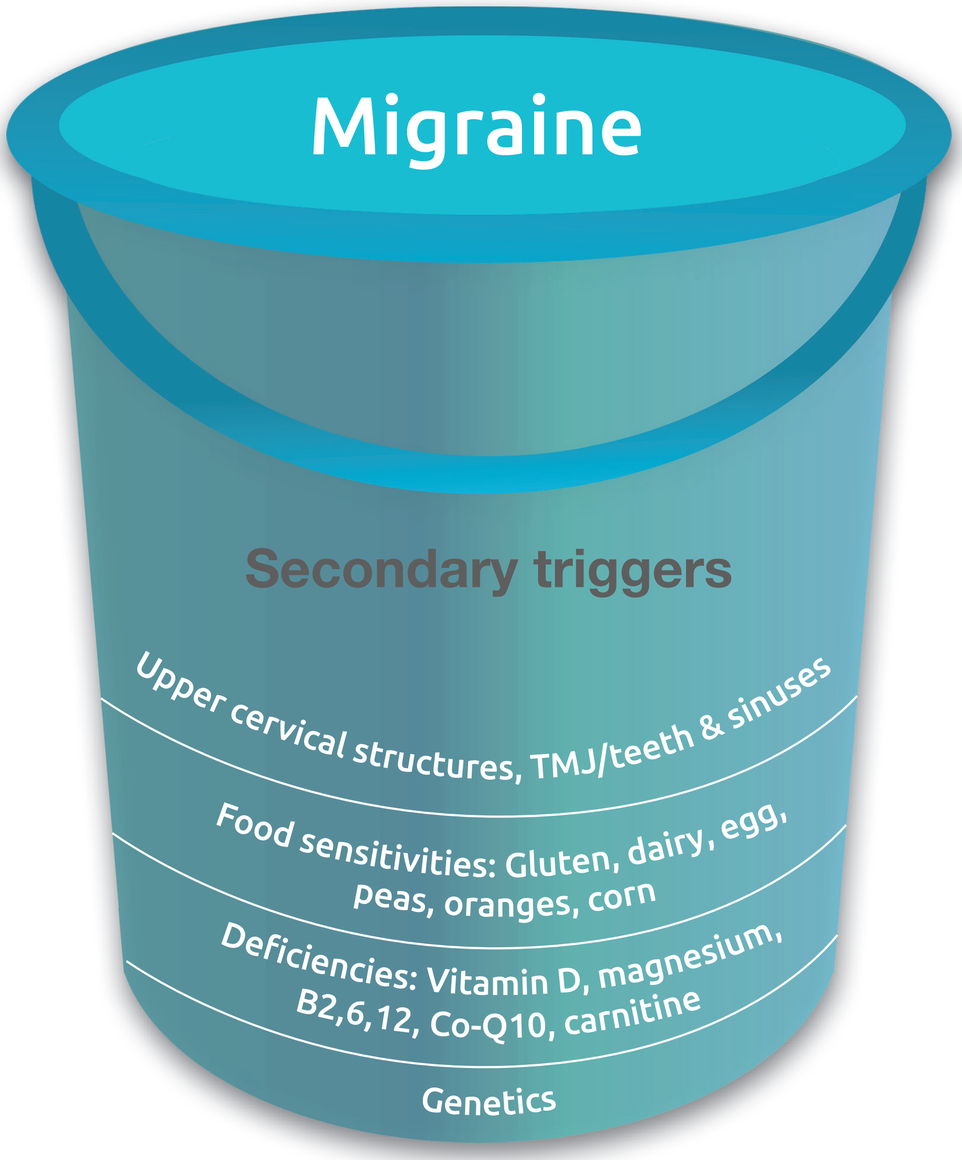

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Foods

- Known food *“triggers”* need to be avoided - usually are but double check it is 100% *(“I avoid it as much as I can”)*

- Corn, peas, citrus fruits, coffee/tea,
- chocolate, beans, yeast
- Or blood test

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Supplementation for migraine

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Level 1

Broad spec multi nutrient support - Foundational vits ADEK, BC, & supporting minerals (One a day Multi essential)

Magnesium 450-600 mg with extra B6 45-60 mg (Magnesium Plus x 3-4 daily)

Vit D 5000 iu daily total (Vit D/K2 sublingual)

B2 (riboflavin) 350 mg (combined daily total 400 mg)

Co Q 10 200mg

Acetyl - L - Carnitine 2000 mg

B12 - 1000 mcg daily Hydroxo or methyl (sublingual)

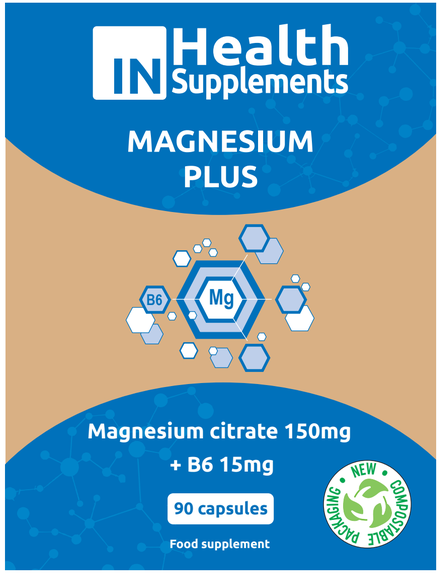
Foods - Complete avoidance of known triggers or trial of classic triggers

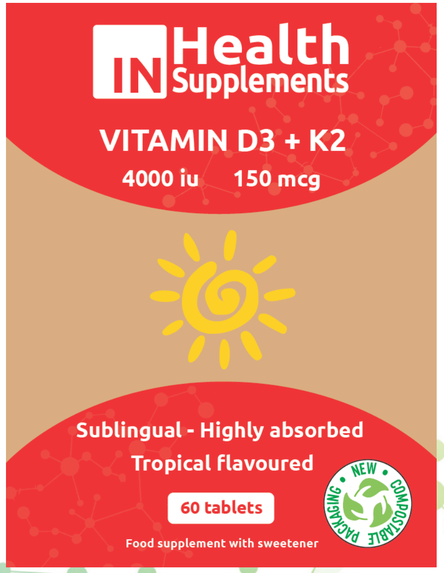
Plus Big 5 & potentially peas, citrus fruit, beans, corn, yeast, tea/coffee

IgG blood test

Intro 2-5 day modified fast (lemon & maple syrup)

Any individual consideration from Phase 1





+ 350 mg B2
+ 200 mg Coq10

www.inhealthsupplements.co.uk

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The effects of magnesium prophylaxis in migraine without aura

Magnesium Research 2008; **21** (2): 101-8

Emel Köseoglu[1], Abdullah Talaslıoglu[1], Ali Saffet Gönül[2], Mustafa Kula[1]

	Before treatment	After treatment	p
Mg treatment group attack frequency Median (min-max)	3.0 (2-5)	2.0 (0-3) 50%	< 0.001
Mg treatment group VAS score Mean ± SD	7.57 ± 0.86	4.00 ± 1.53 47%	< 0.001
Placebo treatment group attack frequency Median (min-max)	3.5 (2-5)	3.0 (2-5)	< 0.05
Placebo treatment group VAS score Median (min-max)	7.0 (6-8)	7.0 (5-8)	> 0.05

Mg treatment group n = 30 Placebo treatment group n = 10.
The average intensity of the attacks were recorded using a 10 cm Visual Analogue Scale (VAS).

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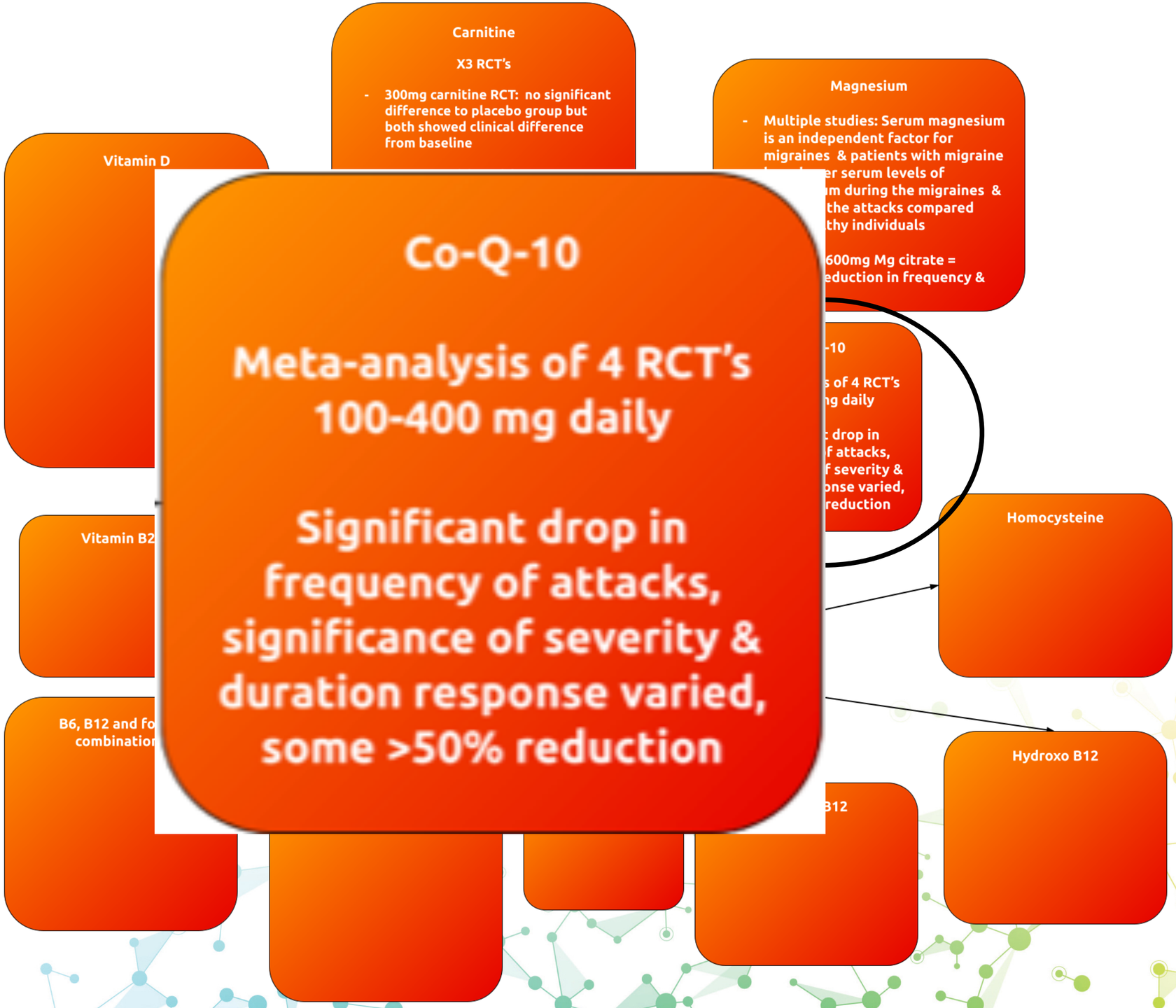

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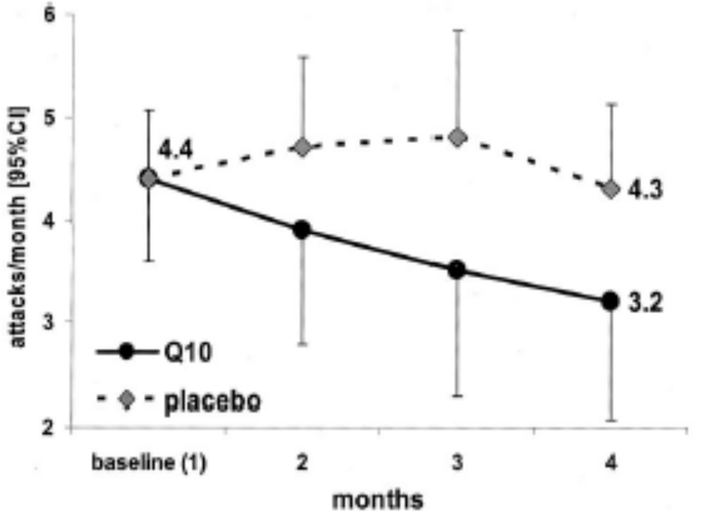
Efficacy of coenzyme Q10 in migraine prophylaxis: A randomized controlled trial

Abstract—Riboflavin, which improves energy metabolism similarly to coenzyme Q10 (CoQ10), is effective in migraine prophylaxis. We compared CoQ10 (3 × 100 mg/day) and placebo in 42 migraine patients in a double-blind, randomized, placebo-controlled trial. CoQ10 was superior to placebo for attack-frequency, headache-days and days-with-nausea in the third treatment month and well tolerated; 50%-responder-rate for attack frequency was 14.4% for placebo and 47.6% for CoQ10 (number-needed-to-treat: 3). CoQ10 is efficacious and well tolerated.

NEUROLOGY 2005;64:713–715

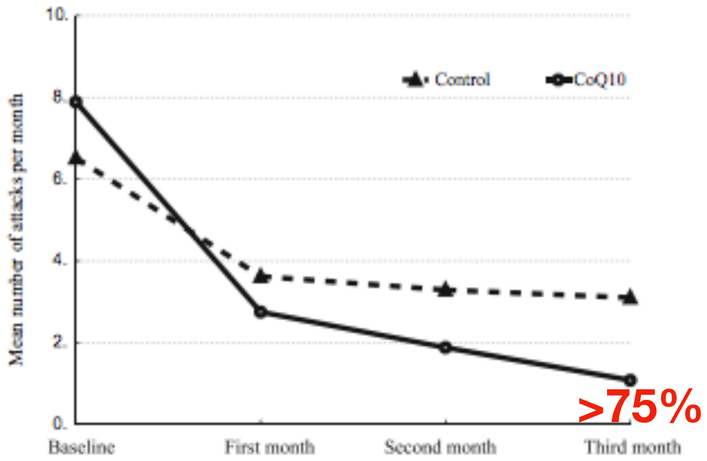
P.S. Sándor, MD; L. Di Clemente, MD; G. Coppola, MD; U. Saenger; A. Fumal, MD; D. Magis, MD; L. Seidel, MSc; R.M. Agosti, MD; and J. Schoenen, MD, PhD



27%

Effectiveness of coenzyme Q10 in prophylactic treatment of migraine headache: an open-label, add-on, controlled tr

Ali Shoeibi[1] · Nahid Olfati[1] · Mohsen Soltani Sabi[1] · Maryam Salehi[2] · Sara Mali[1] · Mahsa Akbari Oryani[3]



>75%

Fig. 2 Trend of reduction of mean number of attacks per month during trial in the CoQ10 and control groups

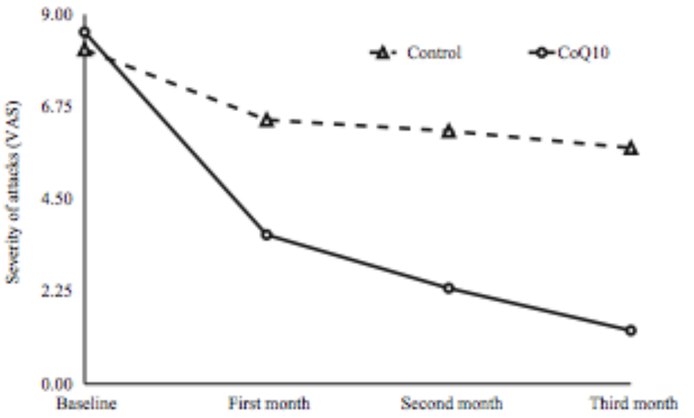


Fig. 3 Trend of reduction of attack severity during trial in the CoQ10 and control groups

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Carnitine

X3 RCT's

- 300mg carnitine RCT: no significant

Magnesium

e studies: Serum magnesium
lependent factor for
es & patients with migraine
wer serum levels of
ium during the migraines &
n the attacks compared
althy individuals

s 600mg Mg citrate =
reduction in frequency &

Vitam

Meta-analysis:
migraine suffer
cont

RCT with patien
nmol/L given 40
= >50% reductio
total numb

RCT 2
Approx 35% drop
disab
Reduce

Q-10

sis of 4 RCT's
mg daily

nt drop in
of attacks,
of severity &
ponse varied,
% reduction

Vitamin

Homocysteine

B6, B12 and
combinat

Hydroxo B12

B12

Vitamin D

Meta-analysis: Lowers vit D in migraine sufferers vs healthy controls

RCT with patients averaging 85 nmol/L given 4000 iu or placebo = >50% reduction in frequency & total number of days

RCT 2000 iu
Approx 35% drop in frequency & disability
Reduced CGRP

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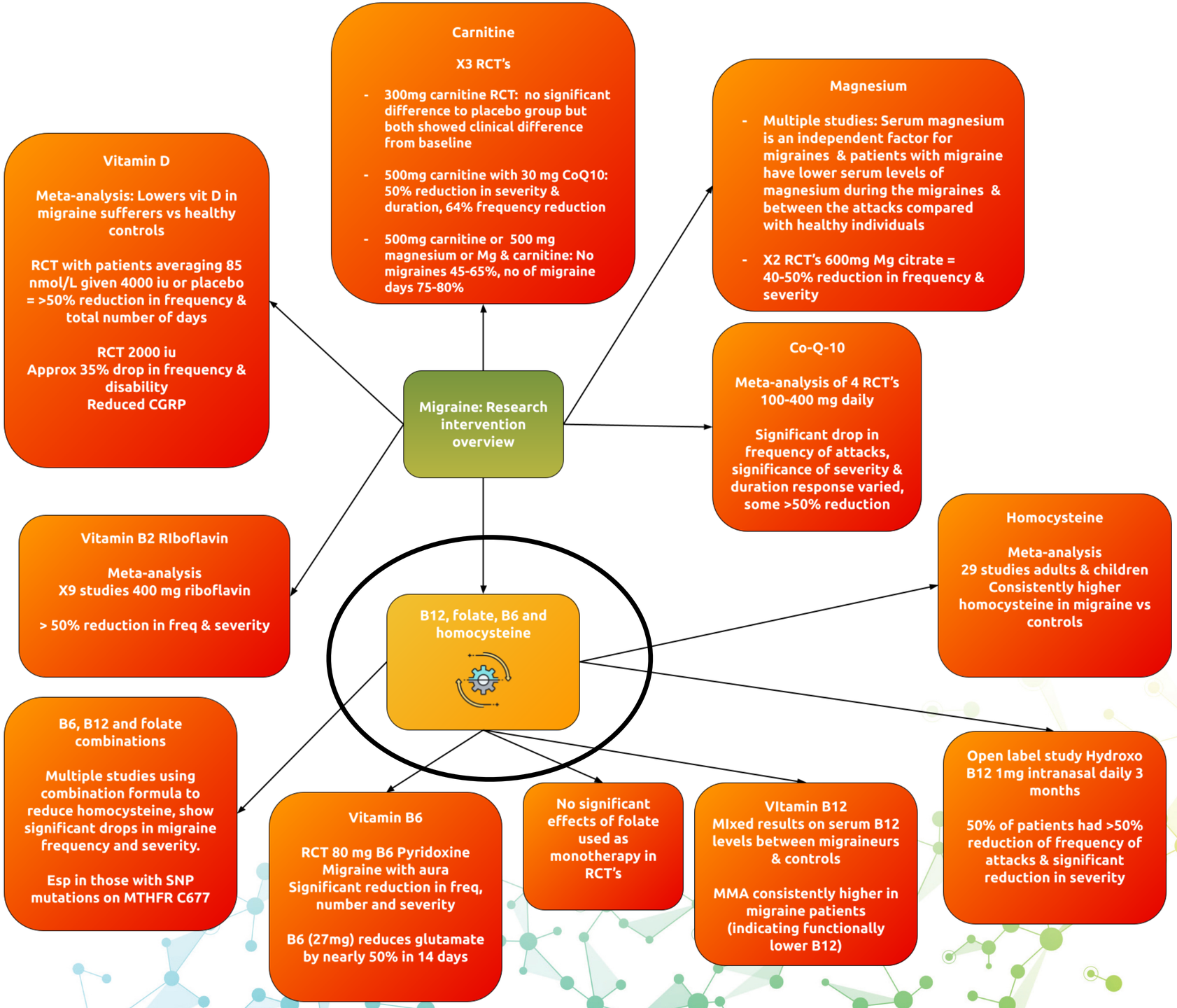

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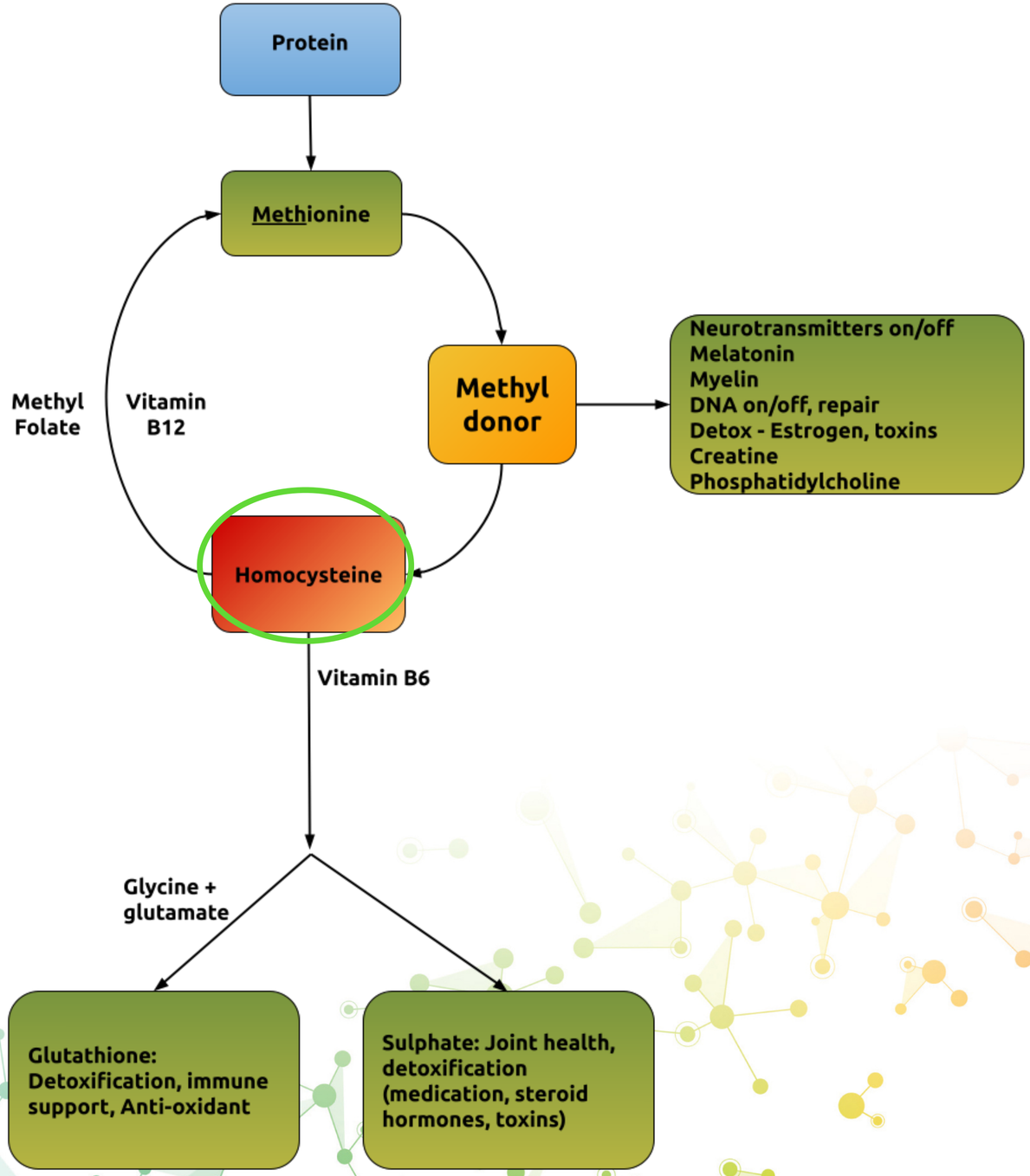
Methylation & Homocysteine

- 🔥 pro-inflammatory
- Trigger of hyper excitation of nervous system



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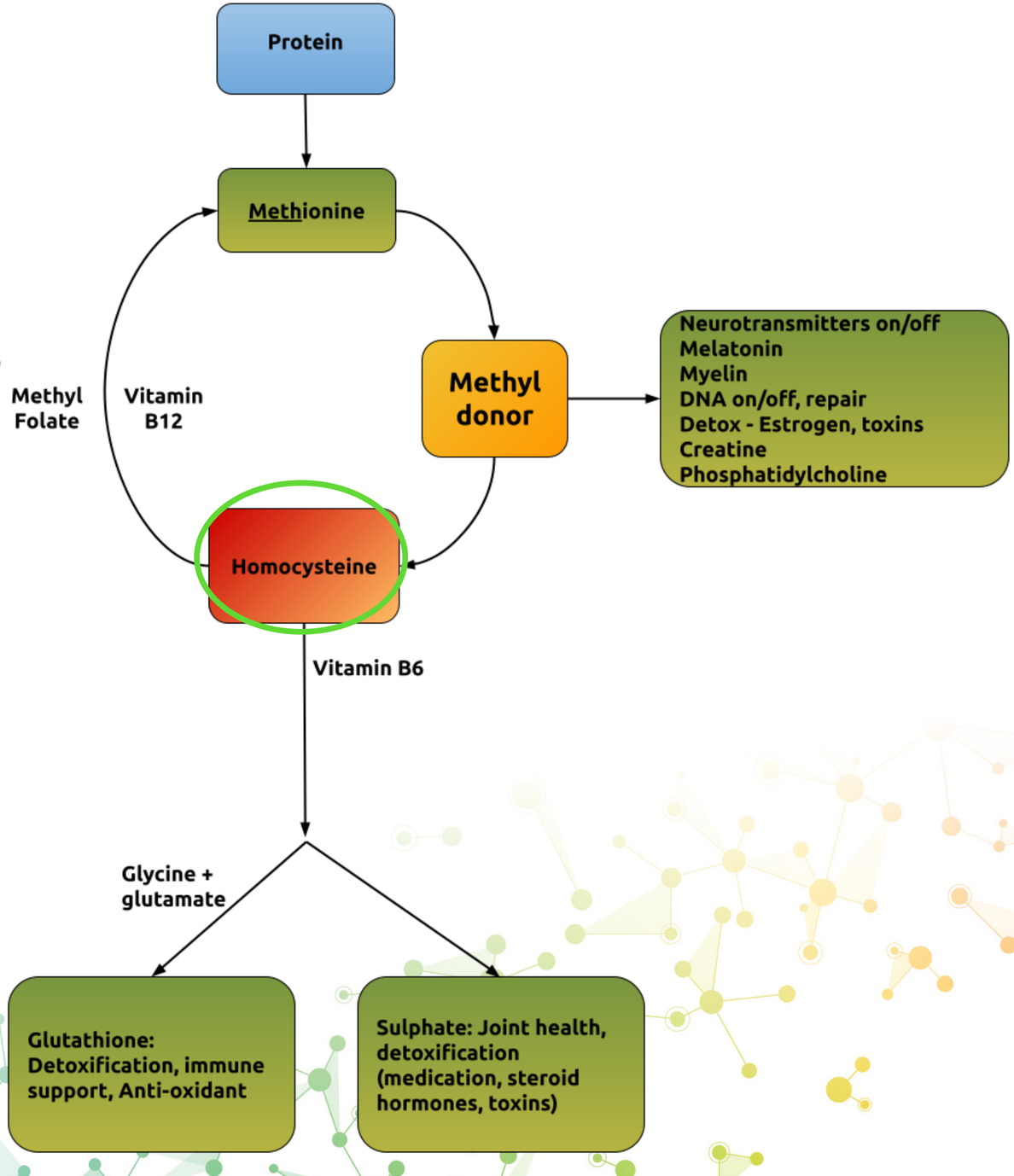
Methylation & Homocysteine

Genetic issues around production of active/methyl folate underpin a lot of genetics/ family hx of migraine

MTHFR



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B2
RDA 1.4 mg

Carnitine

X3 RCT's

- 300mg carnitine RCT: no significant difference to placebo group but both showed clinical difference from baseline
- 500mg carnitine with 30 mg CoQ10: 50% reduction in severity & duration, 64% frequency reduction
- 500mg carnitine or 500 mg

Magnesium

- Multiple studies: Serum magnesium is an independent factor for migraines & patients with migraine have lower serum levels of magnesium during the migraines & between the attacks compared with healthy individuals

Vitamin D

Meta-analysis: Lowers vit D in migraine sufferers vs healthy controls

Vitamin B2 RIboflavin

Meta-analysis
X9 studies 400 mg riboflavin

> 50% reduction in freq & severity

Esp in those with SNP mutations on MTHFR C677

Migraine with aura
Significant reduction in freq, number and severity

B6 (27mg) reduces glutamate by nearly 50% in 14 days

RCT's

& controls

MMA consistently higher in migraine patients (indicating functionally lower B12)

attacks & significant reduction in severity

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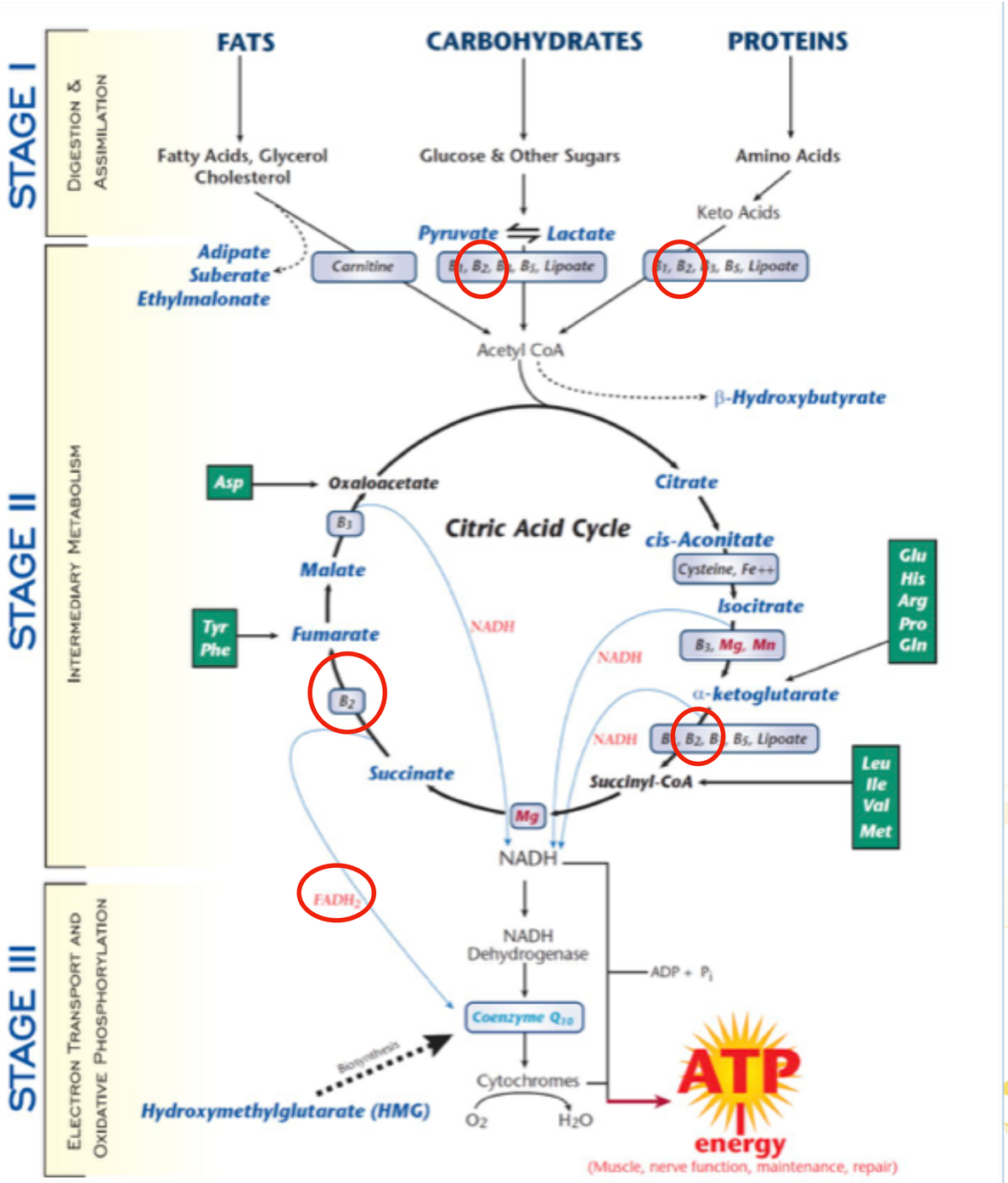
B2 mechs: Improved mito function



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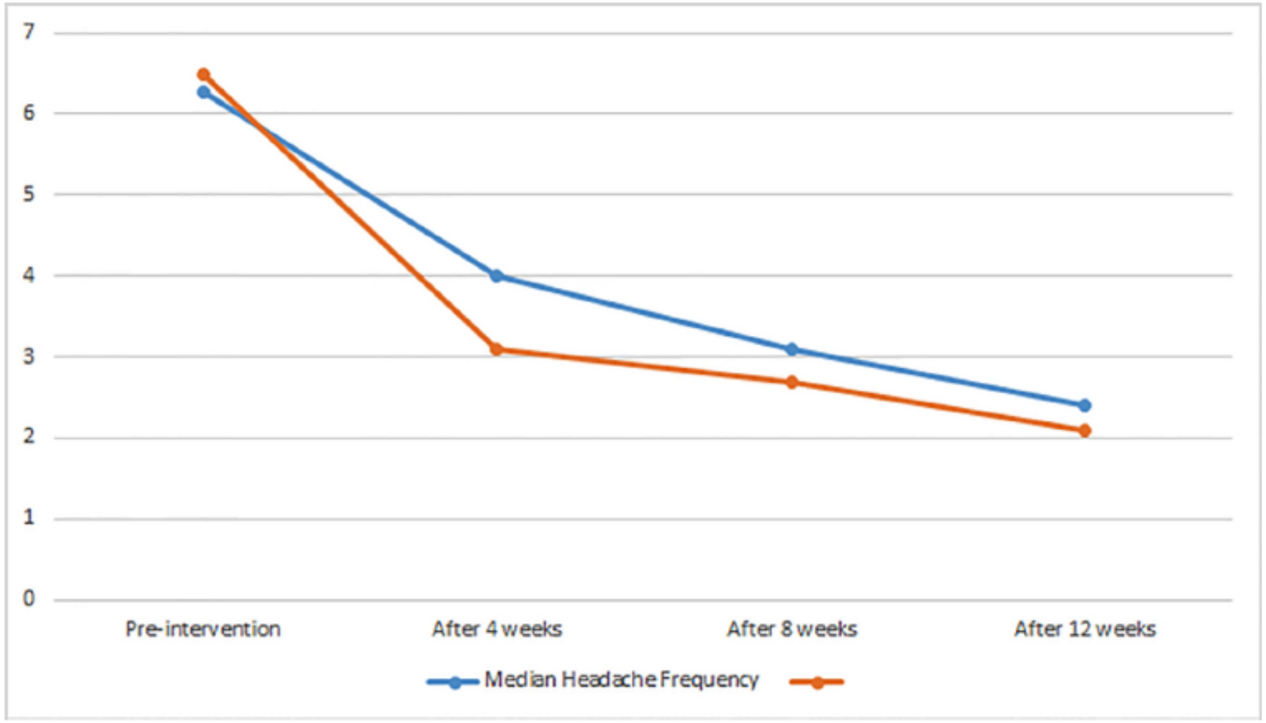
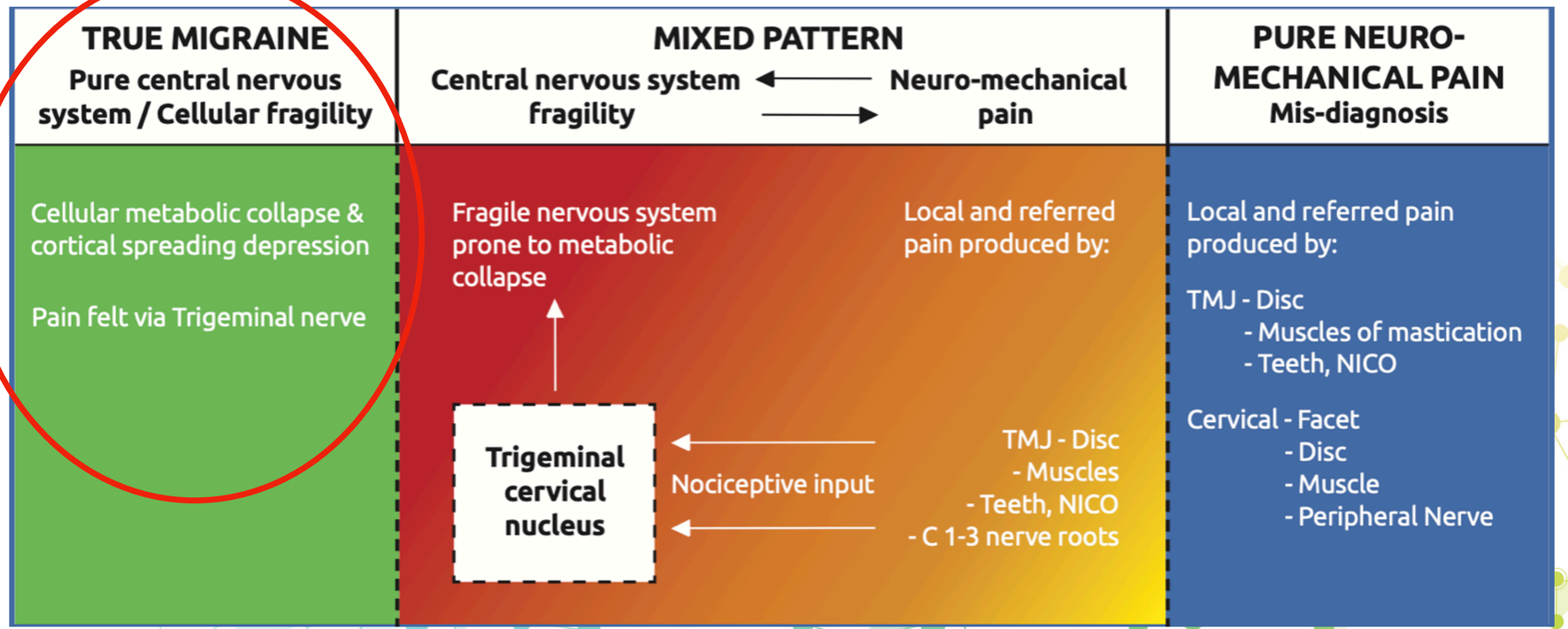


Figure 1. Changes in the frequency of headaches in the two groups, i.e., group 1 (vitamin B2 group) and group 2 (sodium valproate group)

Also, the duration of headaches decreased from about 15.1 ± 7.1 to 4.2 ± 2.6 hr/month in group 1 (vitamin B2 group) and from 16.2 ± 10.6 to 8.2 ± 4.7 hr/month in group 2 (sodium valproate group). Although there was a greater reduction in group 1 (vitamin B2 group), the difference was not statistically significant. Moreover, the

- Migraine **with aura** - typically are more likely to be pure “true” migraine with significant deficiencies, food sensitivities & genetic tendencies and will respond well to nutrition/supplements & may have less NM involvement



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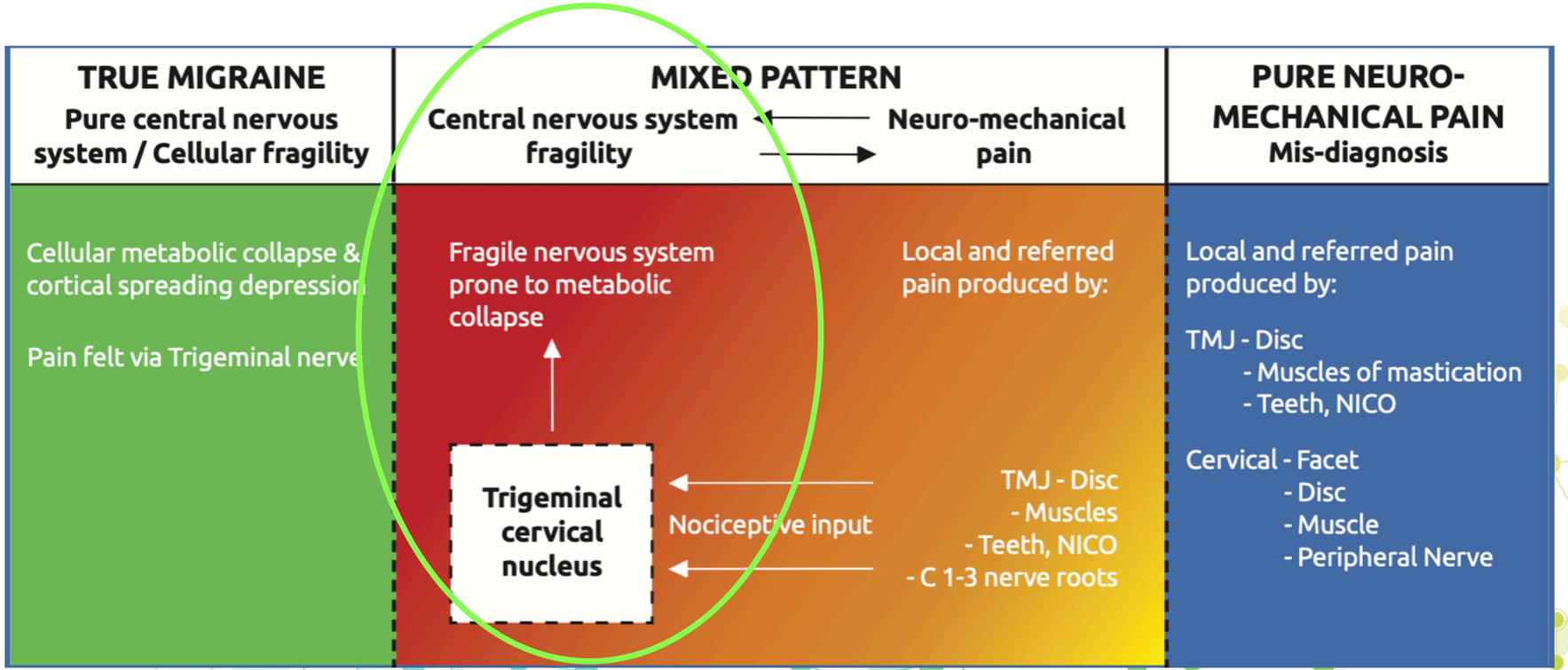
- Migraine **without aura**, higher chance of a mixed pattern with fragile CNS plus TMJ/Cx contribution to nervous system fragility



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The effects of vitamin D supplementation on interictal serum levels of calcitonin gene-related peptide (CGRP) in episodic migraine patients: post hoc analysis of a randomized double-blind placebo-controlled trial

Table 3 Changes in number of headache days, and migraine disability scores before and after supplementation with vitamin D or placebo in episodic migraine patients with/without aura

	Study sub-groups				*P* value*
	Patients with migraine with aura		Patients with migraine without aura		
	Vitamin D	Placebo	Vitamin D	Placebo	
Number of Headache Days per month					
Baseline	10.58 (3.67) [a,b]	7.75 (3.86)	6.81 (2.43) [a]	7.67 (3.29) [b]	0.008
After the trial	6.00 (3.45)	7.50 (3.32)	4.63 (2.40) [a]	7.17 (4.11) [a]	0.029
Changes	−4.58 (3.76) [a,b]	−0.25 (3.05) [a]	−2.09 (2.27)	−0.10 (2.91) [b]	0.000
P value#	0.001	0.801	< 0.001	0.868	
Migraine Related Disability (MIDAS score)					
Baseline	40.00 (26.38)	37.90 (18.53)	24.81 (10.80)	36.53 (22.75)	0.057
After the trial	23.08 (24.42)	42.90 (25.15) [a]	17.59 (10.94) [a,b]	32.46 (16.44) [b]	0.001
Changes	−16.92 (15.39) [a,b]	5.00 (20.18) [a]	−7.22 (9.25)	−1.46 (12.20) [b]	0.001
P value#	0.002	0.453	< 0.001	0.547	

Data are presented as mean (standard deviation)
Alphabets represent significant differences between each variable and two other variables, calculated by Bonferroni test (post-hoc)
*One-way analysis of variance (ANOVA)
Paired sample t-test

43%

32%

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- Late onset migraine >40’s with no childhood hx or family hx (esp without aura) - may lack genetic predisposition via methylation - thus less responsive to mitochondrial manipulation with B2/Coq10/carnitine/B12.

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- Late onset migraine >40's with no childhood hx or family hx (esp without aura) - may lack genetic predisposition via methylation - thus less responsive to mitochondrial manipulation with B2/B12.

- Increase suspicion of significant deficiency from medication or loss of hormones, or toxins (mercury - teeth, fish etc)
- and/or TMJ/dental - look for history of TMD, *"tension headaches"* (temporalis pain), significant dental work changing occlusion - mis-diagnosis and/or input to TGN-Cx nucleus
- and/or cervicogenic h/a misdiagnosed and/or cervical input to TGN

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Migraine screening protocol

Personal history

Colic as a baby
Migraine as a child
Abdominal migraine
Cyclical vomiting syndrome

Significant mental health issues previously (Inflammation & serotonin/quinolinic acid)
+ve use of SSRI
Insomnia

Vegan/vegetarian (reduced nutritional intake)

IBS, IBD, coeliac/gluten, reflux/heartburn (reduced absorption & foods sensitivities)

Fibromyalgia

TMD & significant dental intervention (TGN)

Head & neck trauma

Family history

Migraine

Methylation & inflammation genes:
Early onset or high frequency

- Dementia
- Stroke, MI
- Osteoporosis
- Cancer

Mental health - bipolar, psychosis, schizophrenic

B12 deficiency/Pernicious anaemia

Bowel disorders - IBD, coeliac/gluten, IBS

Medication

Statins - CoQ10

PPI - Minerals, B vits, protein/amino acids

Metformin - B12

Positive use of triptans - Low serotonin (5-HTP)

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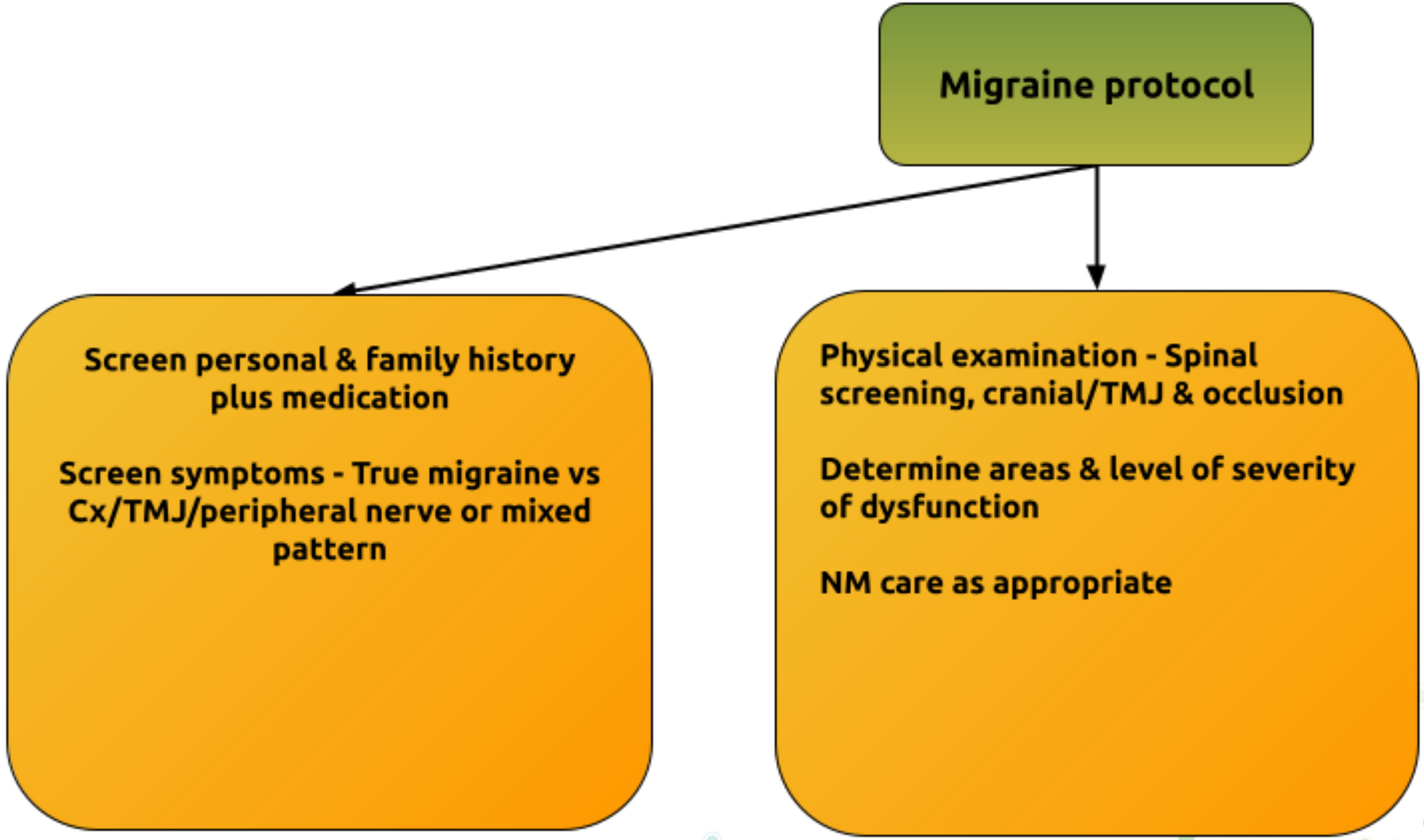

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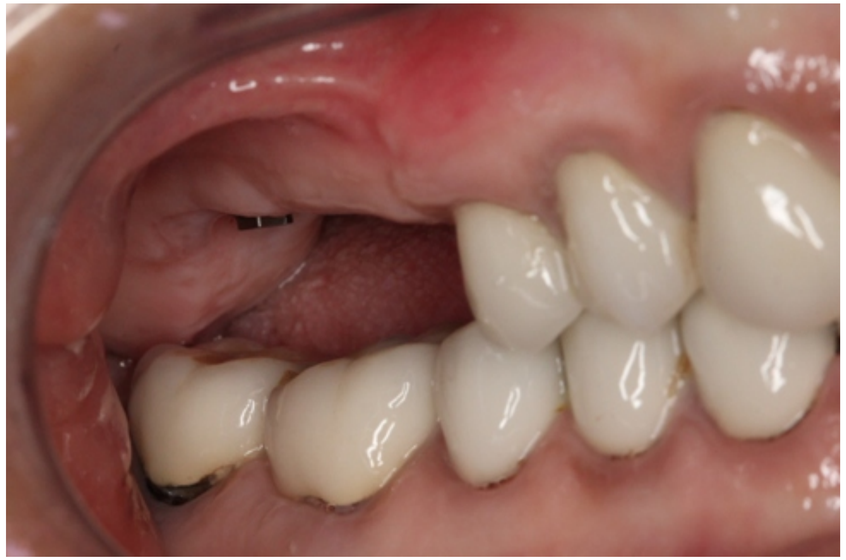
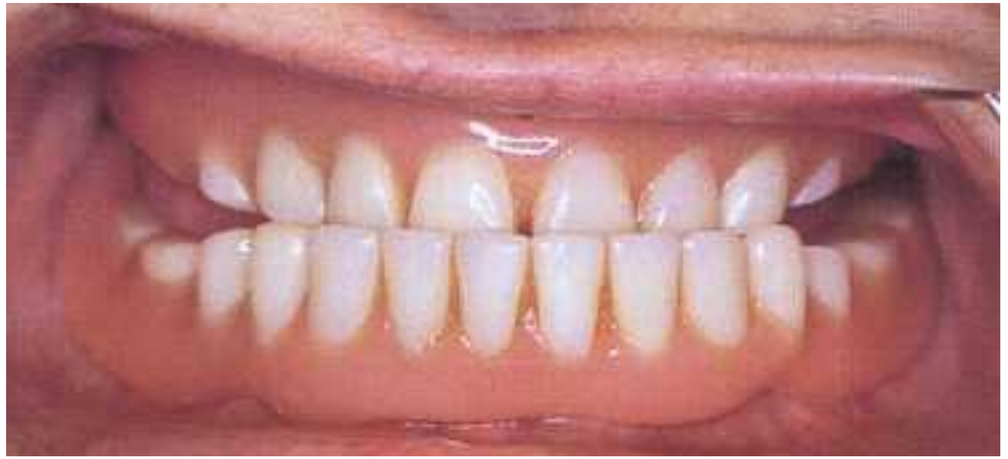
TMD - Loss of posterior molar height

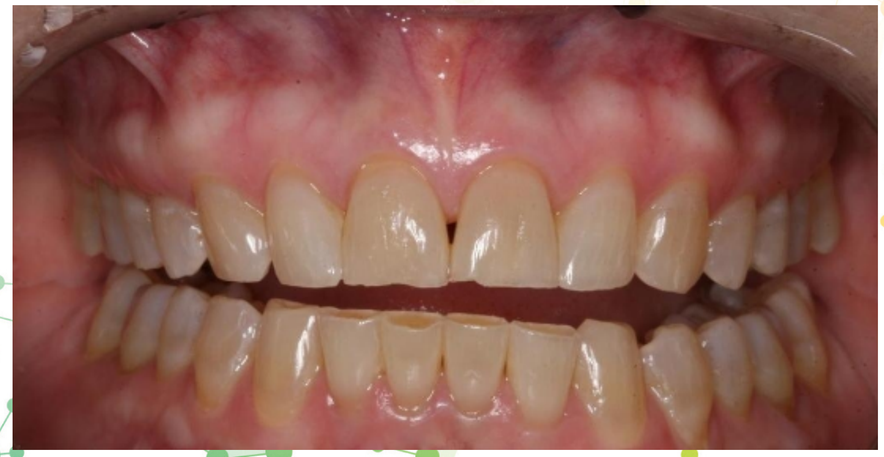
Loss of molars

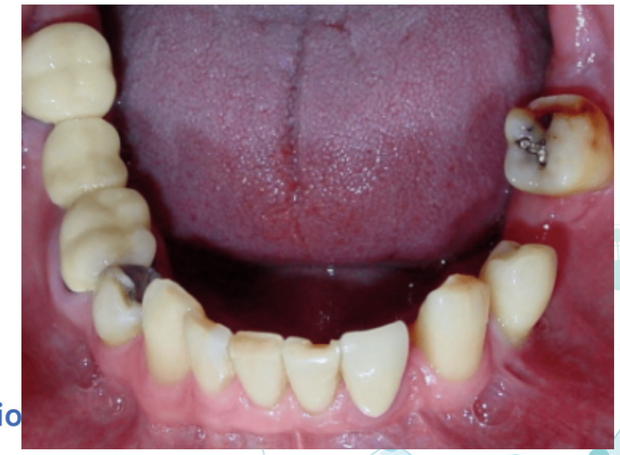


Very old and worn down denture



“flat” teeth line = anterior interference





falling in teeth

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Migraine protocol overview

Screen personal & family history plus medication

Screen symptoms - True migraine vs Cx/TMJ/peripheral nerve or mixed pattern

Physical examination - Spinal screening, cranial/TMJ & occlusion

Determine areas & level of severity of dysfunction

NM care as appropriate

Level 1 metabolic care
- Supplements
- Foods

- Sleep

Level 2

- Supplements
-

Any bespoke issues from phase 2 that may be relevant

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Level 1

Broad spec multi nutrient support - Foundational vits ADEK, BC, & supporting minerals
(One a day Multi essential)

Magnesium 450-600 mg with extra B6 45-60 mg
(Magnesium Plus x 3-4 daily)

Vit D 5000 iu daily total
(Vit D/K2 sublingual)

B2 (riboflavin)
350 mg (combined daily total 400 mg)

Co Q 10 200mg

Acetyl - L - Carnitine 2000 mg

B12 - 1000 mcg daily Hydroxo or methyl (sublingual)

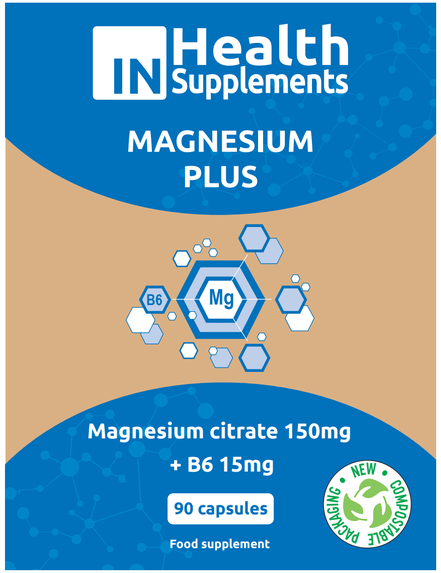
Foods - Complete avoidance of known triggers or trial of classic triggers

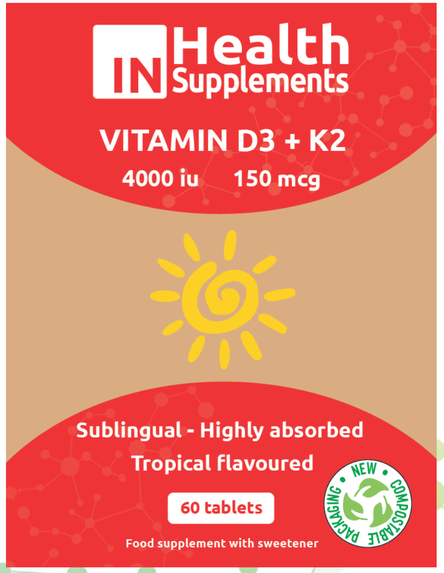
Plus Big 5 & potentially peas, citrus fruit, beans, corn, yeast, tea/coffee

IgG blood test

Intro 2-5 day modified fast (lemon & maple syrup)

Any individual consideration from Phase 1





+ 350 mg B2
+ 200 mg Coq10

Foods

- Known food *“triggers”* need to be avoided - usually are but double check it is 100% *(“I avoid it as much as I can”)*

- Corn, peas, citrus fruits, coffee/tea,
- chocolate, beans, yeast
- Or blood test

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1 month trial (ish)

- Most research trials run 3 months with full results then assessed
- 3 months of supplements can be off putting cost wise & migraine patients are quite sceptical & patients in general tend to be impatient (medical model of quick relief)

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1 month trial (ish)

- Most research trials run 3 months with full results then assessed
- 3 months of supplements can be off putting cost wise & migraine patients are quite sceptical & patients in general tend to be impatient (medical model of quick relief)

- Selling the higher levels of supplements & cost as a 1 month trial avoids subconscious concerns about cost in long term - *“it’s not forever”*
- A trend is all we want to see for  to continue

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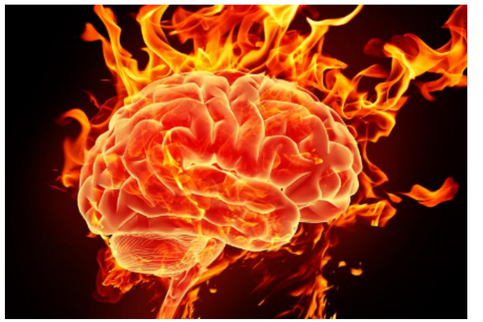
QUESTIONS?



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w.inhealthsupplements.co.uk

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Case study - March 2017

- 65 yo male, currently daily headache/migraine 7/10 av, since 1980's on and off, loc: R forehead
- Neck pain/stiffness
- AF - stress, driving, can wake with it, eating
- CT/MRI NAD
- Coeliac Dx 2 yrs ago - "mostly gluten free", B12 injections for 13 months now stopped
- Meds: Losartan, Family Hx: Sister MS 33 yo

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Exam

- Cx rom L rot 45, R 90
- TMJ - significant early protrusion with opening (abnormal), 40 mm max
- Anterior temporalis TP’s = h/a R forehead, upper cx = h/a
- No lower R molars (removed in Navy in 1980’s)
- Meersseman test +ve MAJOR

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Working Dx

- TMD w R temporalis myofascial pain referral mis-dx as migraine/ha, cx restriction likely driven by descending TMJ issues
- Likely SIR from non-compliance to GF, leaky gut/poor absorption? Low B12 (stopped injections), dysbiosis very likely
- Get GP notes, conservative care TMJ mob, Myofascial work to temporalis, cx, and SMT/IASMT to cx/tx, SOT/Cranial
- High chance he needs denture for TMD
- B12 SUBLINGUAL & multi, Vit D/K2/ Mag CITRATE 450MG

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Bloods 2015

Specimen: BLOOD **Taken: 16/04/2015 08:05 Recieved: 16/04/2015**

Reasons for Request?:
coeliacs

!Full blood count - FBC
(KHK) - Within Acceptable Limits

!	Haemoglobin estimation	131 g/L	(135 - 175)
	Total white cell count	5 10*9/L	(4.0 - 11.0)
	Platelet count	228 10*9/L	(150 - 400)
!	Red blood cell (RBC) count	4.18 10*12/L	(4.50 - 6.50)
	Haematocrit	0.398 L/L	(0.380 - 0.520)
	Mean corpuscular volume (MCV)	95.2 fL	(80 - 100)
	Mean corpusc. haemoglobin(MCH)	31.3 pg	(27.0 - 32.0)
	Mean corpusc. Hb. conc. (MCHC)	328 g/L	(300 - 358)
	Red blood cell distribut width	14.4	(11.0 - 14.5)
	Mean platelet volume	8.6 fL	(6.5 - 20.0)
	Neutrophil count	2.4 10*9/L	(2.0 - 7.5)
	Lymphocyte count	2.1 10*9/L	(1.5 - 4.0)
	Monocyte count	0.4 10*9/L	(0.2 - 0.8)
	Eosinophil count	0.1 10*9/L	

EMIS Report ID: 995055 Lab Report ID: 1-6365903344007
Issued: 17/04/2015 00:55 Received: 17/04/20

Bone profile
(KHK) - Tell Patient Normal

Serum calcium	2.21 mmol/L	(2.2 - 2.6)
Serum albumin	38 g/L	(35 - 50)
Serum inorganic phosphate	1.01 mmol/L	(0.8 - 1.5)
Serum alkaline phosphatase	38 u/L	(30 - 130)
Corrected serum calcium level	2.23 mmol/L	(2.20 - 2.60)

Liver function test

Blood haematinic levels
(KHK) - Tell Patient Normal

Serum vitamin B12	147 ng/L	(130 - 800)
Serum folate	6.6 ug/L	(4 - 20)

Please note change of reference range

Serum ferritin	89 ug/L	(20 - 330)

Specimen: SERUM **Taken: 16/04/2015 08:05 Recieved: 16/04/2015 11:19**

Reasons for Request?:
?? coeliacs

!Anti-tissue transglutnase lev	92.6 U/ml	< 15

(SJR) - positive result
If present, assay detects both IgG & IgA antibodies.

Feb 2016

Specimen Comments
Reasons for Request:
coeliacs

Anti-tissue transglutnase lev

Anti-tissue transglutnase lev	*17.9	U/ml	15

If present, assay detects both IgG & IgA antibodies.

Non-compliance to GF diet

- **Report Number: 2-63970237250001**
- **Report Date: 22/02/2016 at 06:35**
- **Specimen Type: Serum (Serum)**
- **Sample ID: BB578903K date 201602151129**
- **Collected: 15/02/2016 at 08:05**
- **Received: 15/02/2016 at 11:29**

Mal-absorbing B12 - folate low-ish
What else is he low in?

Blood haematinic levels

Serum vitamin B12	141	ng/L	130-800
Serum folate	8.2	ug/L	4-20

Please note change of reference range

Serum ferritin	101	ug/L	20-330

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Specimen Comments

Reasons for Request:
coeliacs

	Full blood count - FBC		
Haemoglobin estimation	140	g/L	135-175
Total white cell count	6.3	10*9/L	4.0-11.0
Platelet count	279	10*9/L	150-400
Red blood cell (RBC) count	*4.44	10*12/L	4.50-6.50
Haematocrit	0.424	L/L	0.380-0.520
Mean corpuscular volume (MCV)	95.5	fL	80-100
Mean corpusc. haemoglobin(MCH)	31.4	pg	27.0-32.0
Mean corpusc. Hb. conc. (MCHC)	329	g/L	300-358
Red blood cell distribut width	14.1		11.0-14.5
Mean platelet volume	8.7	fL	6.5-20.0
Neutrophil count	3.7	10*9/L	2.0-7.5
Lymphocyte count	2.0	10*9/L	1.5-4.0
Monocyte count	0.4	10*9/L	0.2-0.8
Eosinophil count	0.1	10*9/L	0.0-0.4

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2017

Specimen Comments
Reasons for Request:
widespread erythematous rash.

Full blood count - FBC

Haemoglobin estimation	*132	g/L	135-175
Total white cell count	8.5	10*9/L	4.0-11.0
Platelet count	313	10*9/L	150-400
Red blood cell (RBC) count	*4.24	10*12/L	4.50-6.50
Haematocrit	0.396	L/L	0.380-0.520
Mean corpuscular volume (MCV)	93.4	fL	80-100
Mean corpusc. haemoglobin(MCH)	31.1	pg	27.0-32.0
Mean corpusc. Hb. conc. (MCHC)	333	g/L	300-358
Red blood cell distribut width	14.4		11.0-14.5
Mean platelet volume	7.9	fL	6.5-20.0
Neutrophil count	4.8	10*9/L	2.0-7.5
Lymphocyte count	*1.3	10*9/L	1.5-4.0
Monocyte count	*0.9	10*9/L	0.2-0.8
Eosinophil count	*1.5	10*9/L	0.0-0.4

- **Report Number: 1-64316032600021**
- **Report Date: 02/02/2017 at 00:54**

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Thank you for referring this 65-year-old gentleman who has been troubled with headache for the last 30 years. He was seen in a Teaching Clinic with direct observation from Dr [redacted]

He reports that his headache started roughly 30 years ago whilst in the Navy and was initially attributed to stress and anxiety. These were a bifrontal band-like sensation which were fairly constant with periods of exacerbation and when severe, he would feel the need to go and sit quietly. At that time, he managed things fairly well with relaxation techniques.

Over the years, his headaches have changed and he now gets a strictly right sided headache lasting for months at a time, followed by periods of remission lasting up to 18 months. The last bout started 6 months ago. He describes a background headache which is constant and dull, localising to the right frontal region. He has these headaches all day every day, although when they first started they would tend to develop towards the end of the day on his long journey home from work . He also describes periods of exacerbation where he gets a dull throbbing pain and a tense feeling in the same region lasting from a couple of minutes up to 30 minutes at a time and he can get several of these exacerbations a day. He denies any other features accompanying these headaches except for watery eyes. He has not really identified any clear triggers or exacerbating factors for his headache and has tried acupuncture and been to the chiropractor with some benefit, but this was not sustained.

It is not fully clear what medications he has tried for his headache in the past. He was on Amitriptyline for years which helped with his sleep but did not relieve his pain. He did not get any benefit from Topiramate; Carbamazepine did seem to have some effect on the headache but he then went on to develop a rash so was forced to discontinue it. I think Pizotifen and Valproate have also been tried.

He denies any regular or frequent use of analgesics and only uses paracetamol and very rarely Co-codamol.

In terms of past medical history, he has been diagnosed with coeliac disease and is adhering to more or less a gluten free diet. He has also got hypertension, for which he is taking irbesartan. He lives with his wife and works as a production manager for a small company. He is an ex-smoker and admits to moderate alcohol consumption.

On examination, optic discs were was normal. Pupils were reactive and equal. Eye movements were full in all directions, there was no RAPD. Visual fields were full to confrontation and the remainder of the cranial nerve examination was normal. Tone, power and reflexes were normal with downgoing plantars. I could not find any neck stiffness or particular tenderness and temporal arteries were pulsatile and non tender.

It is unclear what the cause of Mr Chamberlain's headache is. They do not really fit into chronic migraine or chronic tension headache categories. Given their longstanding nature, it is highly unlikely that there is a sinister underlying cause. We have discussed that a trial of indomethacin is warranted in case we are dealing with the rare syndrome of hemicrania continua. He would require 25 mg bd for 3 days, then increasing to 50 mg bd for 3 days and then to 75 mg bd for 3 days. If there is no benefit after a week on this dose, to stop treatment. We have warned him that this can upset the stomach lining, and occasionally the kidneys and that as he thinks he may have had a peptic ulcer many years ago, coprescription with a PPI would be sensible. He must not take other NSAIDs e.g. diclofenac/ibuprofen whilst using it. If relief is obtained, I would suggest continuing treatment for 3 months of headache freedom before attempting withdrawal.

What is hemicrania continua?

Hemicrania continua describes a continuous, fluctuating, pain present on one side of the head.

The pain is usually mild to moderate in intensity. For many people with this condition they have episodes of severe pain on top of the continuous general pain.

During these painful episodes (or exacerbations), other symptoms also occur:

- cranial autonomic symptoms such as eye-watering, eye redness, nasal blocking or running and eye lid drooping. These will occur on the same side of the pain.
- migraine symptoms such as light, sound or smell sensitivity, nausea and vomiting.

It's not known what causes hemicrania continua. It's not clear whether there is a genetic element or triggering factors, as in migraine.

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Diagnosis

Hemicrania continua has a positive response when treated with the medication, indomethacin. This is an NSAID (non steroidal anti inflammatory drug). It is recommended that a trial of indomethacin is carried out while a strict headache diary is kept.

After a period of time (days to weeks) with an increasing dose of indomethacin, any change in the headache and symptom severity is assessed. This is compared to the headache pattern before the indomethacin was started.

Magnesium and riboflavin supplements can be helpful for chronic headache disorders and more information on their use can be found on the Migraine Trust website. Given the chronicity of his headaches, and failure to respond to medical measures, we will also ask for Dr Frankel to see him in the Headache Clinic. No follow up has been arranged in the General Clinic, as sadly I don't think there is anything further to offer.
With many thanks

Rx

- Adv STRICT GF diet - for healing & to avoid early death
- B12 sublingual, Multi Two per day, Vit D/mag
- C3 PL AR, C4 PR NAR, Cat 2 plus Bilat Inf sphenoid/max
- Digastric home isometrics
- Discussed dentures for lower R molar for TMJ

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2nd visit

- h/a lasting now only 10 mins, def comes with eating
- L cx rot 70
- IASTM cx/SS lig/scalenes/u/traps
- C5 bilat coupled Cat 2 B inf Sphenoid/max
- went over isometric digastric again

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6th visit

- More of same Rx plus temporalis TPT
- Cx rom 85 L, R 90
- 3 days no h/a
- Dentures to be fitted soon

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3 more Rx patient continuing to improve

- Cancels and then re-attends in 2018, been pain free, until last month x2 h/a a week
- x3 Rx, pain free
- x3 re attendances since

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