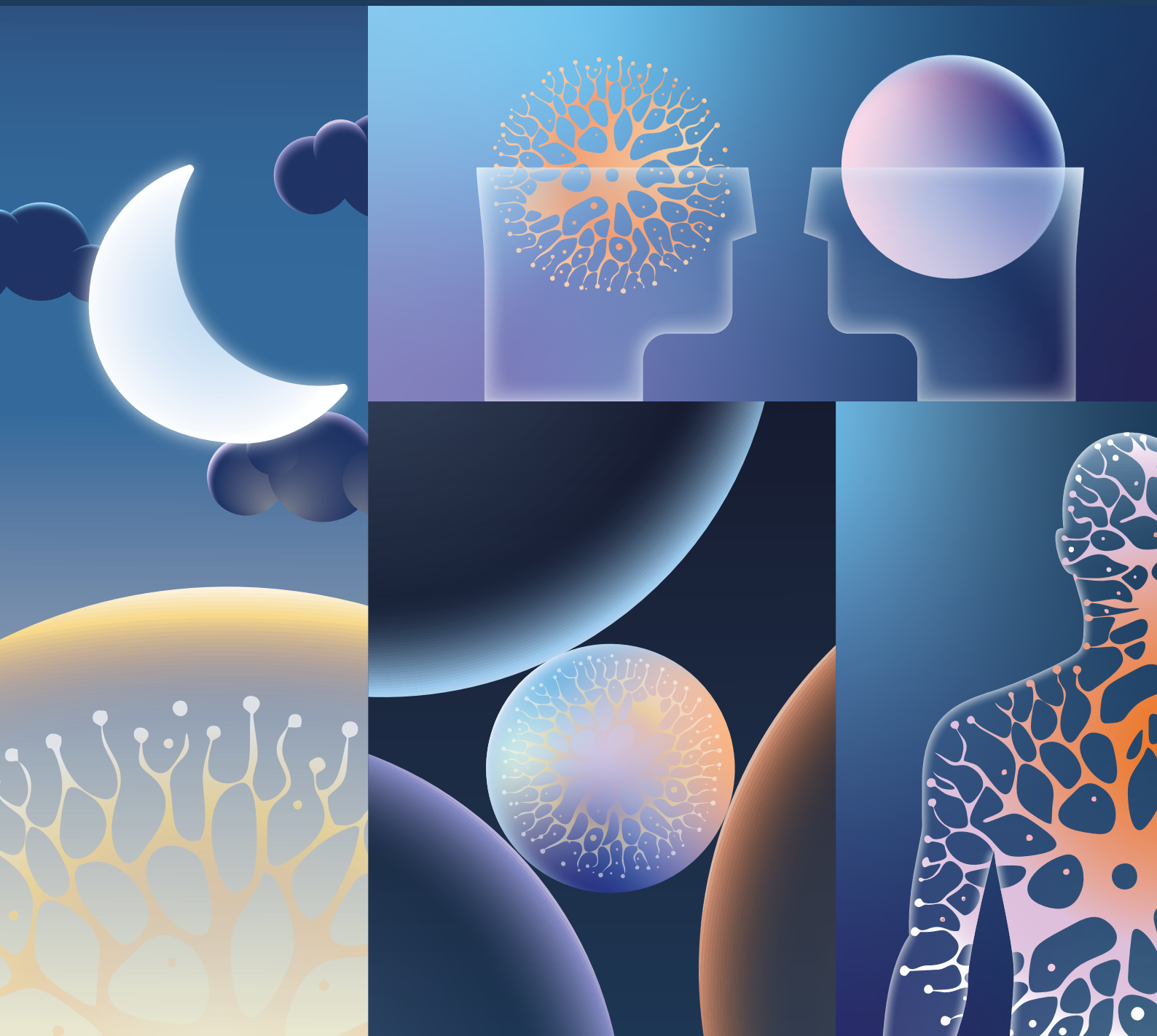
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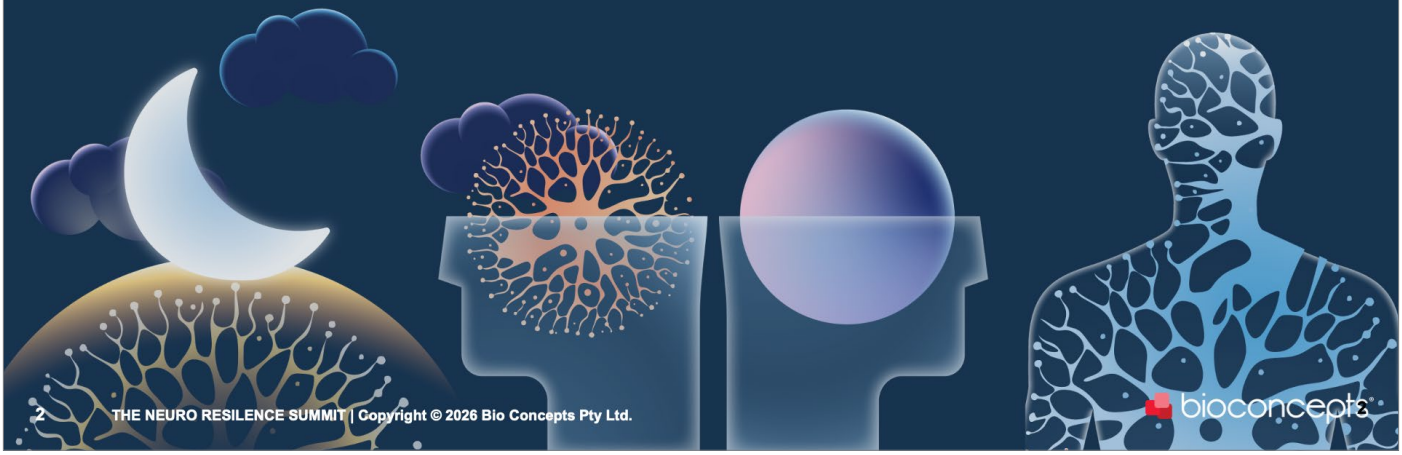
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PRESENTATION NOTES



Sleep is Medicine:

Harnessing Sleep Science for Longevity, Lifespan & Healthspan



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KEYNOTE SPEAKER

Olivia Arezzolo

Australia's Leading Sleep Expert
Bestselling Author in 10 countries
Keynote Speaker
Global Ambassador for Novotel



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Why am I here?



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OUTLINE

1. Why Can't Your Patients Sleep?
2. Stress & Cortisol
3. The 3 M's of Female Sleeplessness
4. Menstruation, Motherhood, Menopause
5. Clinical Toolkit + Tests
6. Supplements
7. Sleep Routines
8. 10-Day Sleep Reset

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Part 1

Why Can't Your Patients Sleep?

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Stress

Menstruation

Motherhood

Menopause

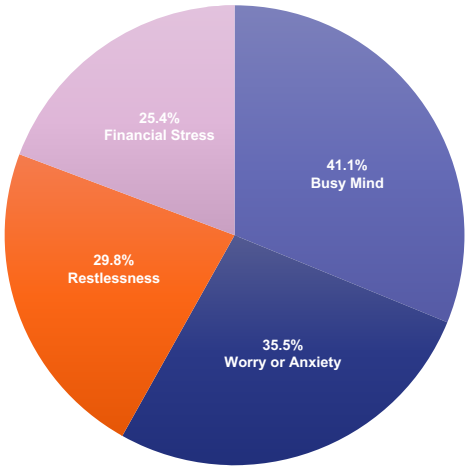
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Stress & Cortisol

- "I'm **waking at 3am"**
- "I fall asleep fine… but **I wake and can't return to sleep"**
- "My brain won't **switch off"**
- "Thoughts are on an endless loop"

Most Common Factors Affecting Sleep



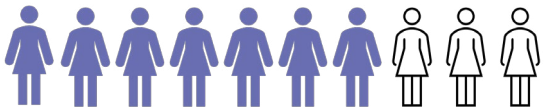
Source: https://www.sleephealthfoundation.org.au/news-and-articles/work-stress-fuelling-australians-poor-sleep-new-sleep-health-foundation-data-reveals

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Menstruation

- **"Every time I have my period, I'm I can't sleep properly"**
- **"I keep waking through the night"**
- **"My cramps keep me up"**



7 in 10 women experience disrupted sleep due to their period 3-6 days prior being the worst

Energy is low
Anxiety is high
Sleep is disrupted

Source: https://www.sleephealthfoundation.org.au/sleep-topics/menstrual-cycle-and-sleep

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Motherhood

64% of mums are exclusively caring for children overnight – compared to only 6% of dads.

It takes up to 6 years to fully recover after the birth of the first child

The nervous system remains hyperactive through the night long after infancy

- **I'm always tired... but that's what being a mum is, right?!"**
- **I'm too busy looking after everyone else to think about myself.**
- **"I ruminate in bed - I'm laying there thinking about everything I have to do"**

Source: https://pmc.ncbi.nlm.nih.gov/articles/PMC12025490/?utm_source=chatgpt.com

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Menopause

- **"I wake at 2am, 3am, 4am..."**
- **"I've tried everything – nothing works"**
- **"My sleep isn't what it used to be, but there's nothing I can do.“**

64% in perimenopause or menopause report sleep has become worse

85% of women experience hot flashes – they can last up to 20 years

Hormones = uncontrollable = uncontrollable sleep.

Sources: https://www.sleephealthfoundation.org.au/news-and-articles/work-stress-fuelling-australians-poor-sleep-new-sleep-health-foundation-data-reveals; https://www.resmed.com.au/blog/sleep-impact-menopause; https://www.sleepfoundation.org/women-sleep/menopause-and-sleep

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Part 2

How Can we Help your Patients Sleep?

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TOOLKIT

1. Test Biomarkers
2. Supplements
3. 10 Day Sleep Reset

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Step 1:

Test Biomarkers

Full Blood Count

- Illness?
- Infection?
- Anaemic?

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Test Biomarkers: Nutrients

- **Iron deficiency?**
- **B Vitamins: Folate & B12**
- **Vitamin D**
- **RBC Magnesium**

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Test Biomarkers: Hormones

- Thyroid?
- Fasting Glucose? HbA1c?
- Cortisol? Evening or Daytime?

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Sleep Study

- Sleep disorder?
- Sleep apnoea?

NOTE: test last, long wait times, high cost for private

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Test results can explain:

- **Extreme fatigue, low energy, high anxiety**
- **Chronic stress, 3am wakings, misaligned circadian rhythm (awake in the PM, tired in AM)**
- **Light, non-restorative sleep, nighttime wakings**
- **…and so much more**



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Step 2:

Supplements

Stack

For all: Mg + glycinate, L-theanine, Glycine

For anxiety/stress: L-theanine

For low mood: Saffron

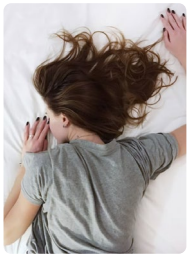
For fatigue: Creatine

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Magnesium Bisglycinate

- **28% ↓ insomnia severity over 4 weeks vs 18% placebo**
- **Dosage:** 250mg
- **Patient talk:** Magnesium = brakes of the nervous system
- **Who for?**
  All – especially women,
  1 in 3 are mg deficient



Source: Schuster J, Cycelskij I, Lopresti A, Hahn A. Magnesium Bisglycinate Supplementation in Healthy Adults Reporting Poor Sleep: A Randomized, Placebo-Controlled Trial. Nat Sci Sleep. 2025 Aug 30;17:2027-2040. doi: 10.2147/NSS.S524348. PMID: 40918053; PMCID: PMC12412596.

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L-Theanine

- **42% reduction in cortisol after a single 200 mg dose**
- **Dosage:** 200mg
- **Best results:** 30-60 minutes (does not require build up)
- **Patient talk:** Helps brain switch off
- **Who for?** Those struggling with 3am wakings, falling asleep, returning back to sleep, bedtime rumination, wired but tired

Source: Evans M, McDonald AC, Xiong L, Crowley DC, Guthrie N. A Randomized, Triple-Blind, Placebo-Controlled, Crossover Study to Investigate the Efficacy of a Single Dose of AlphaWave® L-Theanine on Stress in a Healthy Adult Population. Neurol Ther. 2021 Dec;10(2):1061-1078. doi: 10.1007/s40120-021-00284-x. Epub 2021 Sep 25. PMID: 34562208; PMCID: PMC8475422.

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Glycine

- **Regulates body temperature – critical for menopause, menstruation, overheating**
- **Dosage:** 3g
- **Best results:** 30-60 minutes (does not require build up)
- **Patient talk:** Helps lower core body temperature
- **Who for:** perimenopause, period, hot sleepers, in summer

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Saffron

- **24.6% increase in sleep quality after 4 weeks**
- **Dosage:** 14 mg
- **Best results:** after 4 weeks
- **Patient talk:** helps improve mood.
- **Who for:** Patients struggling with mood

Source: Lopresti AL, Smith SJ, Drummond PD. An investigation into an evening intake of a saffron extract (affron®) on sleep quality, cortisol, and melatonin concentrations in adults with poor sleep: a randomised, double-blind, placebo-controlled, multi-dose study. Sleep Med. 2021 Oct;86:7-18. doi: 10.1016/j.sleep.2021.08.001. Epub 2021 Aug 11. PMID: 34438361.

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Creatine

- **>29% improvement in problem solving when sleep deprived using creatine vs placebo**
- **Dosage:** 5g daily
- **When:** Take late morning – reduces need for 3/4pm coffee
- **Patient talk**: Backup generator for your brain – helps with clarity and concentration.

Source:Gordji-Nejad, A., Matusch, A., Kleedörfer, S. et al. Single dose creatine improves cognitive performance and induces changes in cerebral high energy phosphates during sleep deprivation. Sci Rep 14, 4937 (2024). https://doi.org/10.1038/s41598-024-54249-9

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Sleep Routine: 3-2-30-10

CHECKLIST

- **3 hours before sleep: finish eating**
- **2 hours before sleep: blue light glasses on OR dim lights/low screen brightness/night mode on**

CHECKLIST

- **30 minutes before bed:**
  - **Shower**
  - **Sleep supplements**
  - **Read / guided meditation**
- **10 minutes upon waking:**
  - **Direct morning light**

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Special Considerations

Menopause & 3am wakings

Menstruation

Motherhood

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Menopause

- **Goal 1: Create a cooling sleep sanctuary:**
  - **Frozen cooling gel pack under pillow. Hot? Flip it.**
  - **Use cooling bamboo bedding, 200-400 TC**
  - **Use cooling mattress topper**
  - **Air con = 18 degrees**
- **Goal 2: Supplements to rebalance nervous system**
  - **Magnesium, L-theanine, glycine**
  - **+ based on your symptoms**

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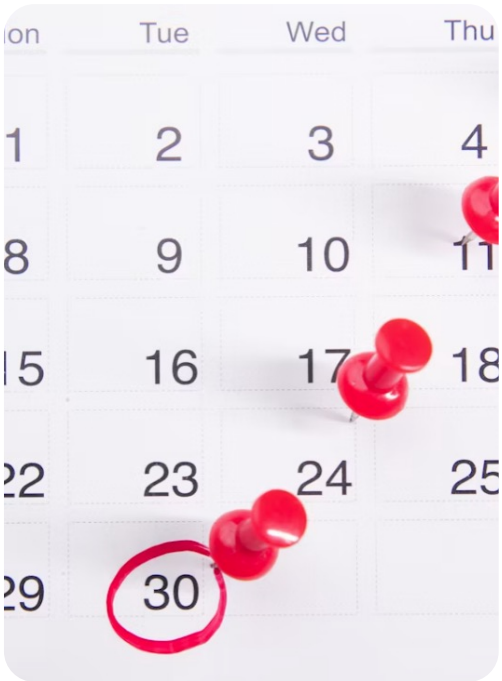
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3am wakings

- Step 1: Cool the body
  - use a cooling gel pack
- Step 2: Calm your breathing
  - 4-7-8 breathing
- Step 3: Calm the mind
  - Read
  - Meditate
  - Worry journal
- Step 3: Still can't sleep? Get up – repeat process
- Step 4: Return to sleep

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Menstruation

- Goal 1: Track 'Red Zone' = 3 days before period, first 2 days of period
- Goal 2: Sleep longer: 1 hour
- Goal 3: Extra care on sleep routine / supplements with Red Zone

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Motherhood

- Goal 1: sleep 3+ hours in single block
- Goal 2: Get support (actually ask)
- Goal 3: use red night lights for night feeds (helps mum return to sleep)
- No set sleep routines when kids are small – you're doing the best you can!

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10 Day Sleep Reset

Supplement support

Sleep routine

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10 Day Sleep Reset

With Olivia Arezzolo - your sleep girl :)

Your name: **Your #1 goal for Sleep Reset:**

When	What	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10	Progress
Supplements												
Before Sleep Reset	Get Sleep Supplements	☐										
Support												
Once a day	Check in with Olivia's Support Group - @oliviaarezzolo	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	0/10
Bedtime routine: 3-2-30-10												
3 hours before sleep	Last bite	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	0/10
2 hours before sleep	Last bright light - wear blue light glasses OR dim lights / lower screen brightness / turn night mode on	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	0/10
30 minutes before bed	Shower	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	0/10
30 minutes before bed	Take sleep supplement	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	0/10
30 minutes before bed	Charge phone in another room	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	0/10
30 minutes before bed	Meditate with me (linked HERE) or read	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	0/10
Before 11pm	Asleep before 11pm, with eye mask	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	0/10
10 minutes upon waking	Direct morning light	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	0/10

Medical disclaimer: This Sleep Reset provides general information and education only and is not medical advice. Supplements may not be suitable for everyone and may interact with medication. Please speak with your doctor or pharmacist before starting any supplement, especially if you are pregnant or breastfeeding, have a medical condition or take medication. Do not change or stop prescribed medication without medical advice.

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Connect!
Join my support group!
@oliviaarezzolo

Free eBooks (link in bio):

Hot, Wired, Awake?
The Menopause Sleep Plan
Ladies, 3am wakings?
..and more!

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Q&A
Q&A
Q&A
Q&A

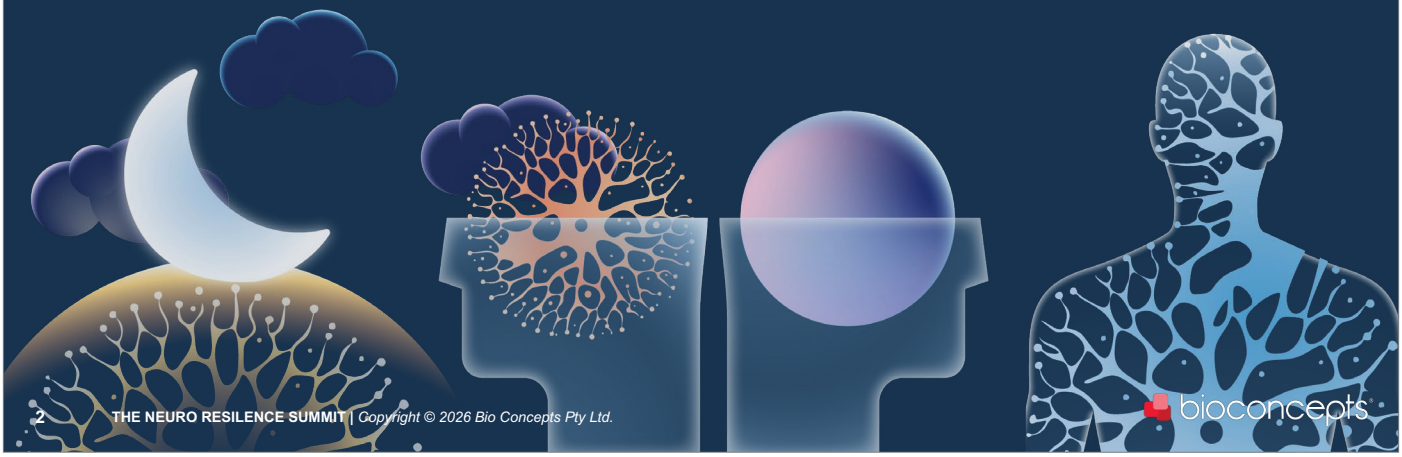
FEATURING

OLIVIA AREZZOLO

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Nourishing the Mind:

Practical Applications of Nutritional Psychiatry Research



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SPEAKER

Dr Tetyana Rocks



PhD, BNutrDiet, BSc (Hons), GCertHigherEd

Accredited Practising Dietitian

Director, Food & Mood Academy

Senior Research Fellow, Deakin University Food & Mood Centre

Honorary Research Fellow, NHS Foundation Trust

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OUTLINE

1. What is Nutritional Psychiatry?
2. Diet in the Treatment of Depression
3. Diet- and Gut-Related Treatments
4. Implementation

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The Food & Mood Centre

The **Food & Mood Centre** at Deakin University is a world-leading research centre that aims to understand the complex ways in which what we eat influences our brain, mood, and mental health.

The **Food & Mood Academy** is academic centre that aims to bring research to life through education and training.



FOOD & MOOD ACADEMY

DEAKIN UNIVERSITY | DEAKIN FOOD & MOOD CENTRE

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What is Nutritional Psychiatry?



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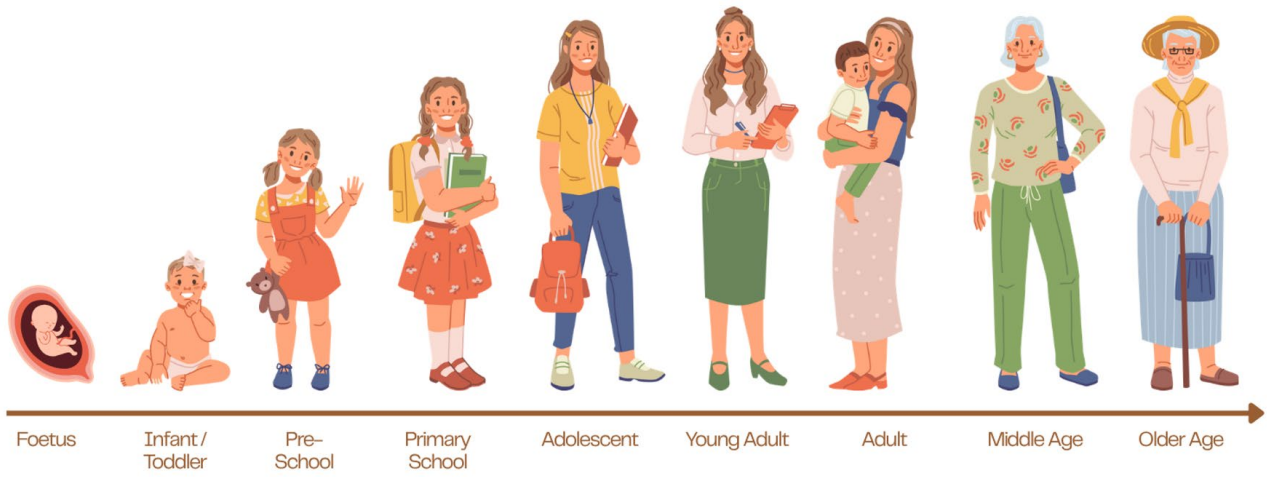
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What do we already know?

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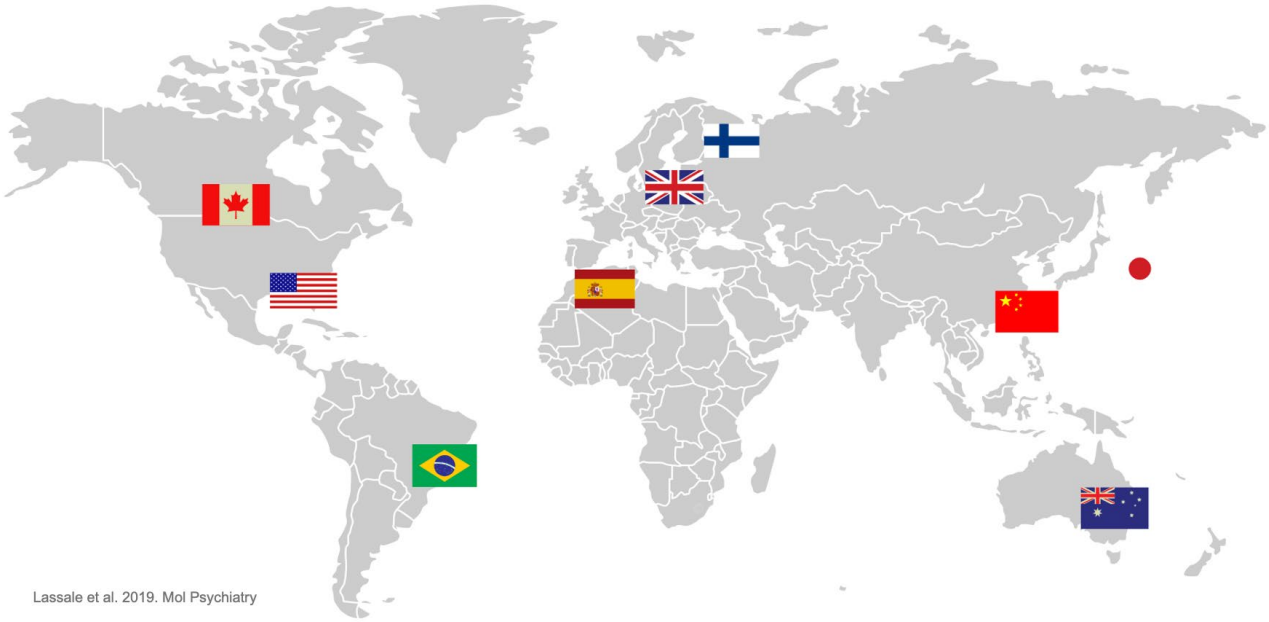
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Diet and Mental and Brain Health are Linked **Across Life Stages**



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Lassale et al. 2019. Mol Psychiatry

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Diet & Depression in **Adulthood**

41 longitudinal and cross-sectional studies

Decreased incidence of depression:

- Adherence to a Mediterranean diet: 0.67 (95% CI 0.55–0.82)
- Lower Dietary Inflammatory Index: 0.76 (95% CI 0.63–0.92).

Lassale et al. 2019. Mol Psychiatry

Review Article | Open access | Published: 26 September 2018

Healthy dietary indices and risk of depressive outcomes: a systematic review and meta-analysis of observational studies

Camille Lassale, G. David Batty, Amaria Baghdadli, Felice Jacka, Almudena Sánchez-Villegas, Mika Kivimäki & Tasnime Akbaraly

Molecular Psychiatry **24**, 965–986 (2019) | Cite this article

128k Accesses | **834** Citations | **1957** Altmetric | Metrics

Overall, diets that are higher in whole, plant-based foods are associated with decreased risk

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Dietary Interventions

Several studies with mental health as primary outcome (depression measures, quality of life)

All used Mediterranean diet.

The SMILES trial - individual, in-person, 12 weeks, MDD (Jacka et al. 2017)

The HELFIMED trial - group, diet + fish oil supplements (Parletta et al. 2019)

Uni students brief trial - young adults, 3 weeks (Francis et al. 2019)

The AMMEND study - young males, online (Bayes et al. 2022)

The CALM trial - group, online, diet + exercise (O'Neil et al. 2024)

Jacka et al. 2017 BMC Med; Parletta et al. 2019 Nutr Neuroscience; Francis et al. 2019 PLoS One; Bayes et al. 2022 AM J Clin Nutr; O'Neil et al. 2024 Lancet Reg Health Western Pacific

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Diet in the treatment of Depression: **The SMILES Trial**

First RCT to ask and answer the questions:
"If I improve my diet, will my mood improve?"

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Mood Boosting Diet

Adults living with depression

- Dietary Intervention
- Befriending Protocol

Dietary Intervention:

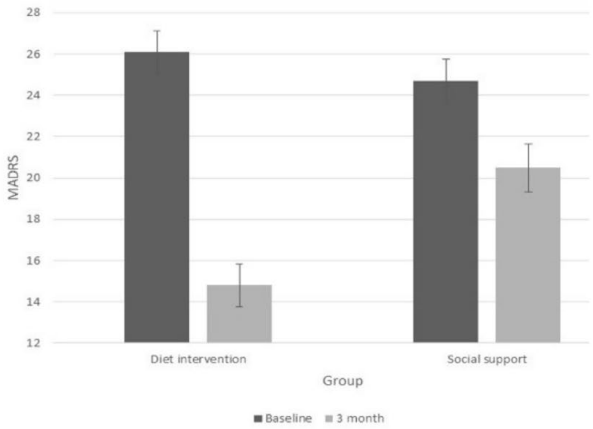
- Designed and delivered by dietitians
- Combination of Mediterranean diet and Australian Guide for Healthy eating

Jacka et al. 2017. BMC Med

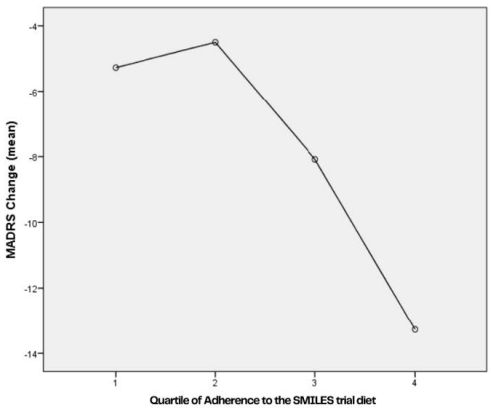
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MADRS Scores



MADRS Scores **Over Time**



Jacka et al. 2017. BMC Med

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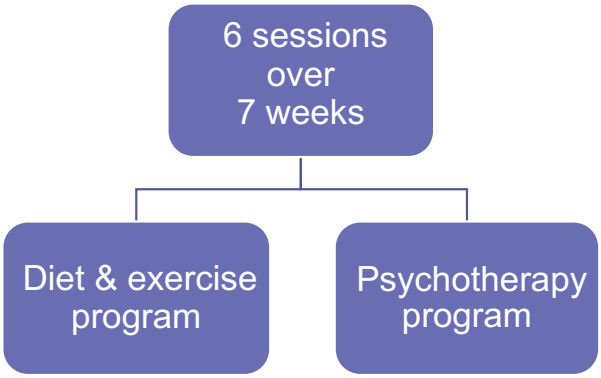
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The **CALM** Trial

Effectiveness of a group-based telehealth lifestyle program for Victorians experiencing COVID-19 related distress

Lifestyle intervention compared to psychotherapy (non-inferiority)

Online group intervention
n=182

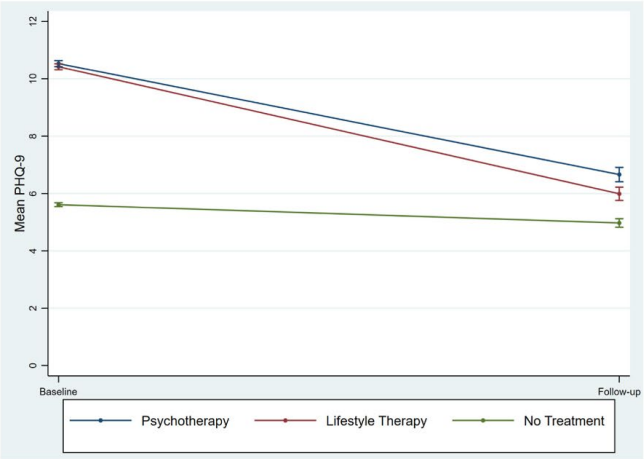


Young et al. 2022. BMC Psychiatry

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Two Paths, **Similar Results**



O'Neil et al. 2024. Lancet Regional Health

Lifestyle therapy is non-inferior to cognitive behavioural therapy for the reduction of depression symptoms

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Meta-Analysis > Nutr Rev. 2025 Jan 1;83(1):29-39. doi: 10.1093/nutrit/nuad176.

The impact of the Mediterranean diet on alleviating depressive symptoms in adults: a systematic review and meta-analysis of randomized controlled trials

Bruno Bizzozero-Peroni [1 2], Vicente Martínez-Vizcaíno [1 3], Rubén Fernández-Rodríguez [1], Estela Jiménez-López [1 4 5], Sergio Núñez de Arenas-Arroyo [1], Alicia Saz-Lara [1], Valentina Díaz-Goñi [1], Arthur Eumann Mesas [1]

Affiliations + expand

PMID: 38219230 DOI: 10.1093/nutrit/nuad176

Bizzozero-Peroni et al. 2024. Nutr Review

Synthesising the Evidence

N=5 RCTs with 1507 participants

Significant reduction among young and middle-aged adults with MD or mild to moderate depressive symptoms

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Diet in the Management of Mental Health

Adjunctive dietary treatments – successful strategy for significant symptom improvement and:

- Support physical health
- Support behavioural change
- Increase access to treatment
- Are highly cost-effective



Jacka et al. 2017. BMC Med; Parletta et al. 2019. Nutr Nurosci; Francis et al. 2019. PLoS ONE; Firth et al. 2019. Psychosom Med; Bayes et al. 2022. Am J Clin Nutr; O'Neil et al. 2024. Lancet Regional Health. (accepted).

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Professional Bodies Increasingly Include Diet As Part of Foundational Treatment

RANZCP Clinical Practice Guidelines



The Royal Australian & New Zealand College of Psychiatrists | CPD endorsed

CANMAT Guidelines

Line of treatment	Lifestyle intervention	Level of evidence
First line	Supervised exercise (low to moderate intensity, for 30 to 40 min at a time, 3 to 4 times a week, for a minimum of 9 weeks) **for MDE of mild severity**.	
	Light therapy (10,000 lux white light for 30 min daily) **for MDEs with seasonal (winter) pattern**.	
Second line	Light therapy **for mild severity nonseasonal MDE**.	
	Adjunctive exercise **for moderate severity MDE**.	
	Adjunctive light therapy **for moderate severity nonseasonal MDE**.	
	Adjunctive sleep hygiene and CBT-I.	
Third line	Adjunctive healthy diet (varied diet with high content of fruit, vegetables, and fibre, and low content of saturated fat and carbohydrates).	
	Adjunctive Mediterranean diet.	
	Adjunctive sleep deprivation (wake therapy).	
Insufficient evidence	Probiotics.	n/a

Level 1; Level 2; Level 3; Level 4.
Note. MDE = major depressive episode CBT-I = cognitive-behavioural therapy for insomnia.

CANMAT

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Malhi et al. 2021. ANZJP. Lam et al. 2024 Can J Psychiatry

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Lifestyle-Based Recommendations



Exercise & Physical Activity



Smoking cessation



Working & Volunteering



Mindfulness & Relaxation



Dietary Change



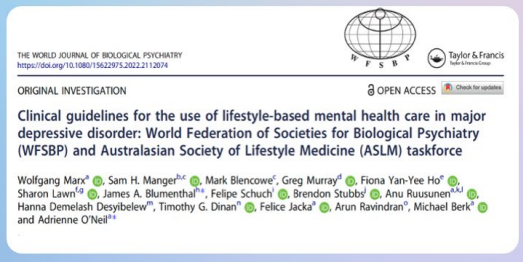
Sleep Hygiene



Social Connection



Green & Blue Spaces



THE WORLD JOURNAL OF BIOLOGICAL PSYCHIATRY
https://doi.org/10.1080/15622975.2022.2112074

ORIGINAL INVESTIGATION

OPEN ACCESS

Clinical guidelines for the use of lifestyle-based mental health care in major depressive disorder: World Federation of Societies for Biological Psychiatry (WFSBP) and Australasian Society of Lifestyle Medicine (ASLM) taskforce

Wolfgang Marx[a], Sam H. Manger[b,c], Mark Blencowe[c], Greg Murray[d], Fiona Yan-Yee Ho[e], Sharon Lawn[f,g], James A. Blumenthal[h,i], Felipe Schuch[j], Brendon Stubbs[j], Anu Ruusunen[k,k,l], Hanna Demelash Desyibelew[m], Timothy G. Dinan[n], Felice Jacka[a], Arun Ravindran[o], Michael Berk[a] and Adrienne O'Neil[b,g]

Marx et al. 2022. World J Biol Psychiatry

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Practice Resources for **Clinicians and Consumers**

ACCESS HERE

Australasian Society of Lifestyle Medicine





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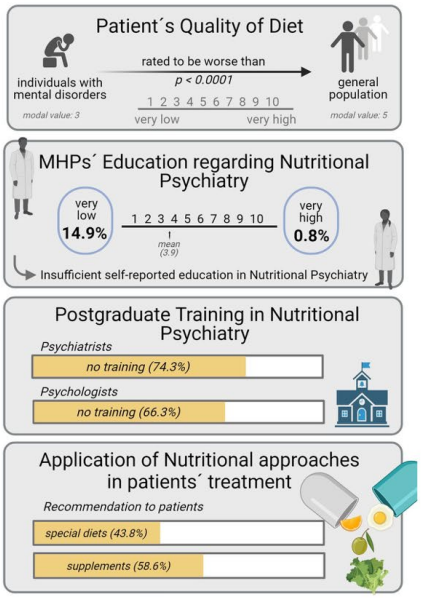
What about Clinical Practice

74% psychiatrist and 66% psychologists reported **no training in nutrition**

Only 0.8% rated their **nutrition education as 'very good'**

Yet, **59% were recommending supplements** and 44% recommending specific diet strategies

Morkl et al. 2021. Nutrients

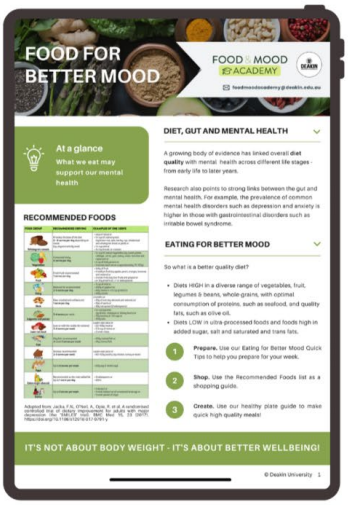


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Better Food for Better Mood
Consumer Resource

Download



Food & Mood Academy

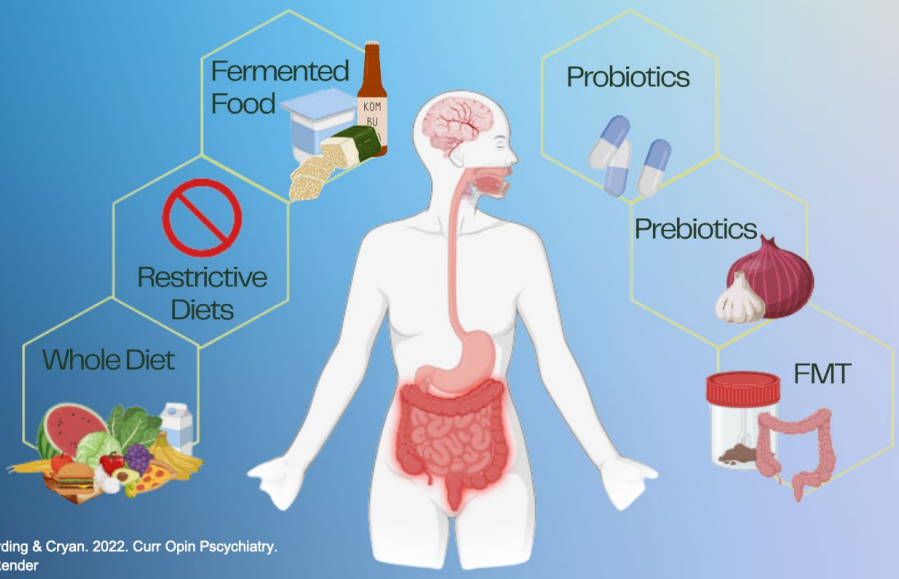
@foodandmoodacademy

foodmoodacademy@deakin.edu.au

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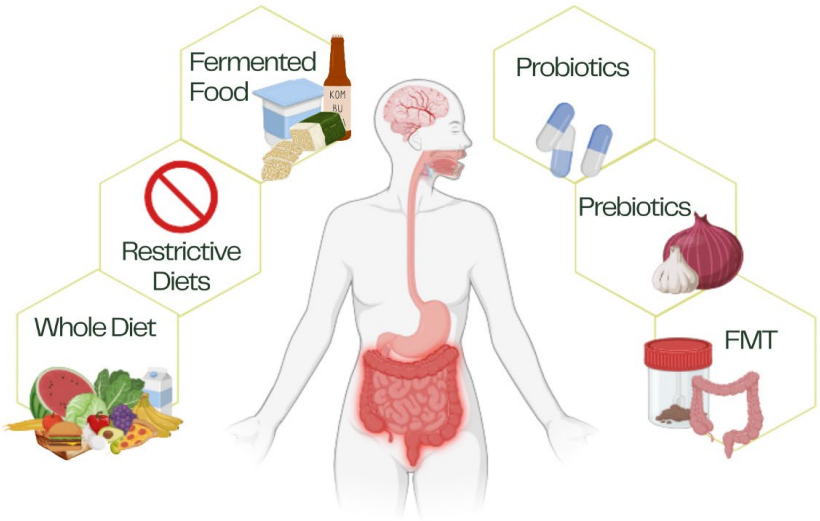
Diet - and Gut-related Treatments



Adopted from: Berding & Cryan. 2022. Curr Opin Pscychiatry.
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Whole Diet

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Mediterranean-Style Diet

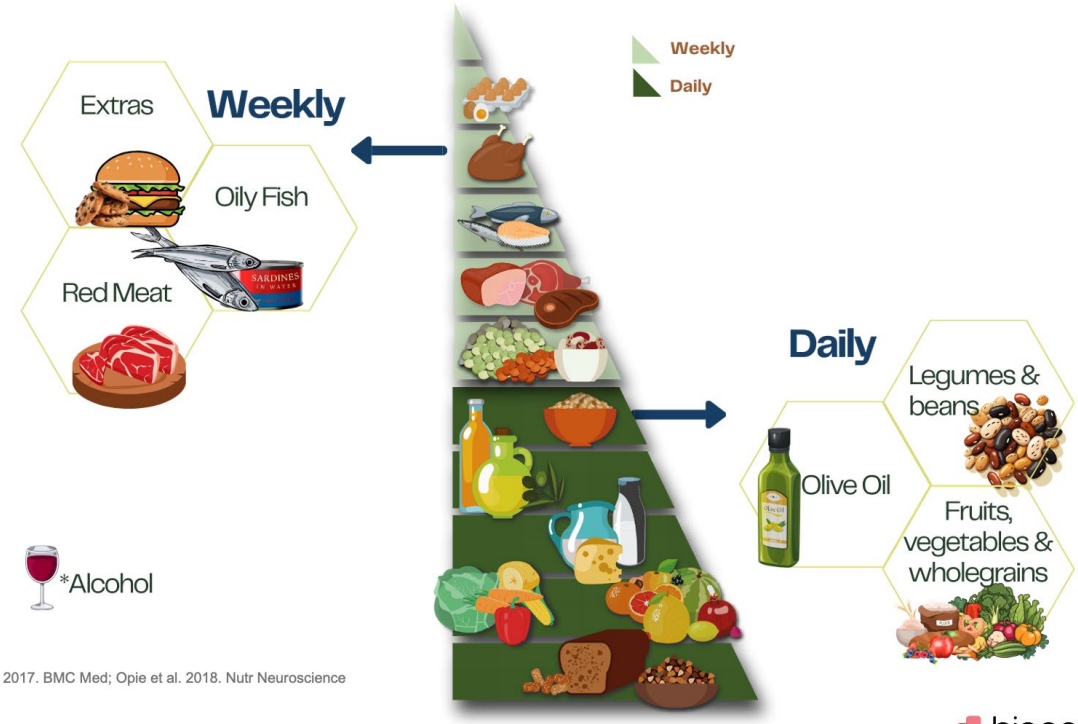


Simple Principles



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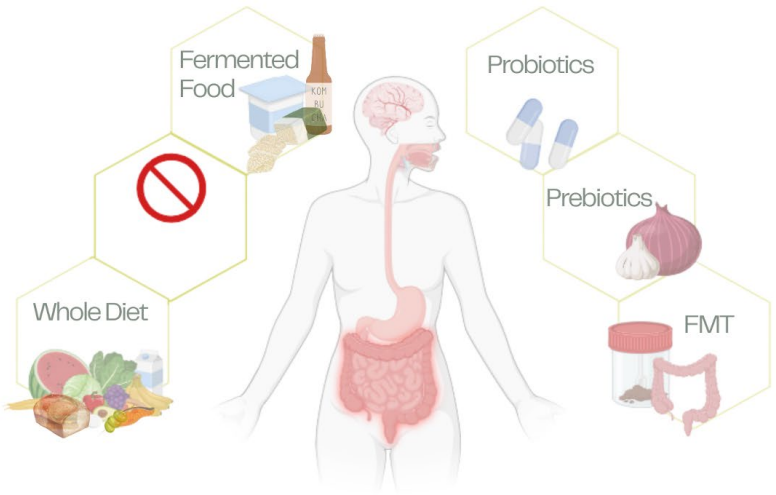
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Jacka et al. 2017. BMC Med; Opie et al. 2018. Nutr Neuroscience

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Restrictive Diets

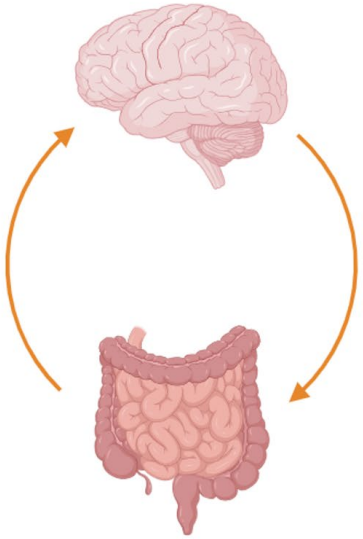
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Comorbid IBS and mental health

- **Irritable Bowel Syndrome (IBS)** affects up to 1 in 10 people
- One third of people with IBS experience **depression and/or anxiety**
- **Diet is a fundamental part of treatment.** The approach is dependent on severity of IBS:
  - **Mild:** education, support, and monitoring
  - **Impaired functioning:** psychological treatment, pharmacology



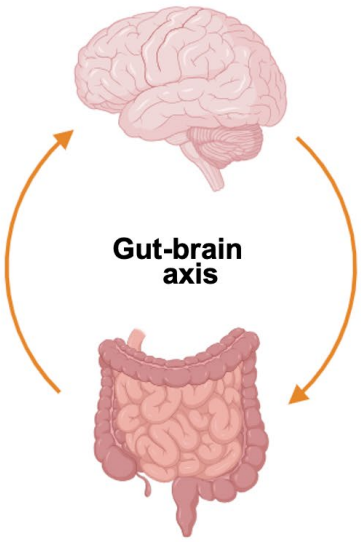
Simons et al. 2022. UE Gastroenterology; Staudacher et al. 2023. Nature Gastroenterol Hepatol
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Addressing comorbid IBS and mental concerns

Low FODMAP Diet	Gastrointestinal motor dysfunction & visceral hypersensitivity
Mediterranean diet	Gastrointestinal microbiome
Psychobiotics	Gastrointestinal microbiome



Adapted from Staudacher et al. 2023. Nature Gastroenterol Hepatol. Created with BioRender

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Disorders of gut-brain interaction

The TANDIM Study

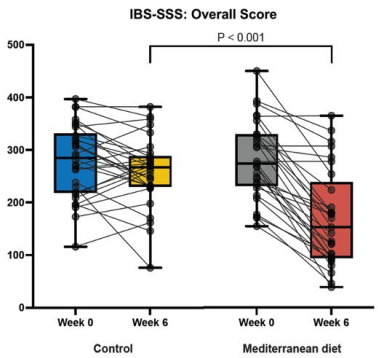
83% of the MD group reported improvement in **global GI symptoms** compared with 37% of controls

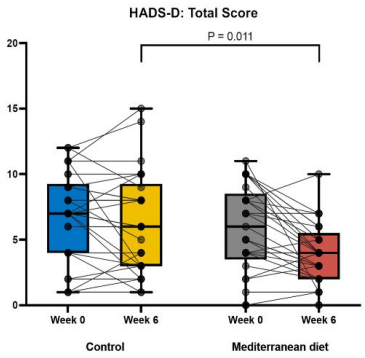
62% reported **improvement in depressive symptoms** compared with 23% of controls

59 individuals with IBS.

6 weeks of either:

- **Mediterranean diet (MD)**
- **Habitual diet**





Staudacher et al. 2023. Aliment Pharmacol Ther

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Ketogenic Diets & Depression & Anxiety: **A Systematic Review and Meta-Analysis**

- 50 studies (41,718 participants) assessed ketogenic diets (KDs) and mental health outcomes in adults.
- KDs showed modest improvements in depressive symptoms, especially when ketosis was biochemically verified.
- Evidence for anxiety benefits was mixed and not consistently significant in randomized trials.
- More high-quality, long-term studies are needed to confirm effectiveness and sustainability.



Research

JAMA Psychiatry | Original Investigation

Ketogenic Diets and Depression and Anxiety
A Systematic Review and Meta-Analysis

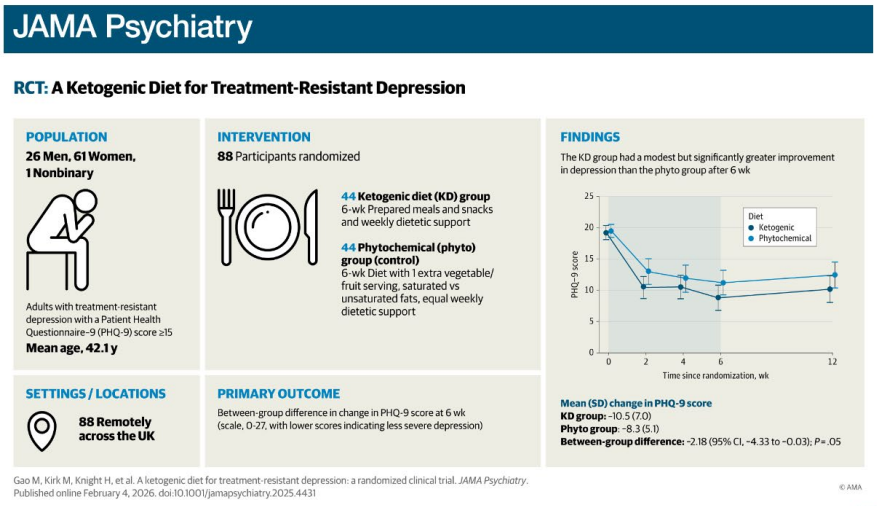
Reinhard Janssen-Aguilar, MD; Tulassi Vije, BHSc; Malika Peera, BHSc; Huda F. Al-Shamali, PhD; Shakila Meshkat. MD: Oiaowei Lin. MSc: Wendv Lou. PhD: Hugo Laviada-Molina. MD. MSc: Marv L. Phillips. MD: Venkat Bhat. MD. MSc

Janssen-Aguilar et al. 2026. JAMA Psychiatry

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Ketogenic Diet: A Randomised Controlled Trial



JAMA Psychiatry

RCT: A Ketogenic Diet for Treatment-Resistant Depression

POPULATION
26 Men, 61 Women, 1 Nonbinary

Adults with treatment-resistant depression with a Patient Health Questionnaire-9 (PHQ-9) score ≥15
Mean age, 42.1 y

INTERVENTION
88 Participants randomized

44 Ketogenic diet (KD) group
6-wk Prepared meals and snacks and weekly dietetic support

44 Phytochemical (phyto) group (control)
6-wk Diet with 1 extra vegetable/fruit serving, saturated vs unsaturated fats, equal weekly dietetic support

FINDINGS
The KD group had a modest but significantly greater improvement in depression than the phyto group after 6 wk

PHQ-9 score
Time since randomization, wk
Diet
Ketogenic
Phytochemical

Mean (SD) change in PHQ-9 score
KD group: −10.5 (7.0)
Phyto group: −8.3 (5.1)
Between-group difference: −2.18 (95% CI, −4.33 to −0.03); *P* = .05

SETTINGS / LOCATIONS
88 Remotely across the UK

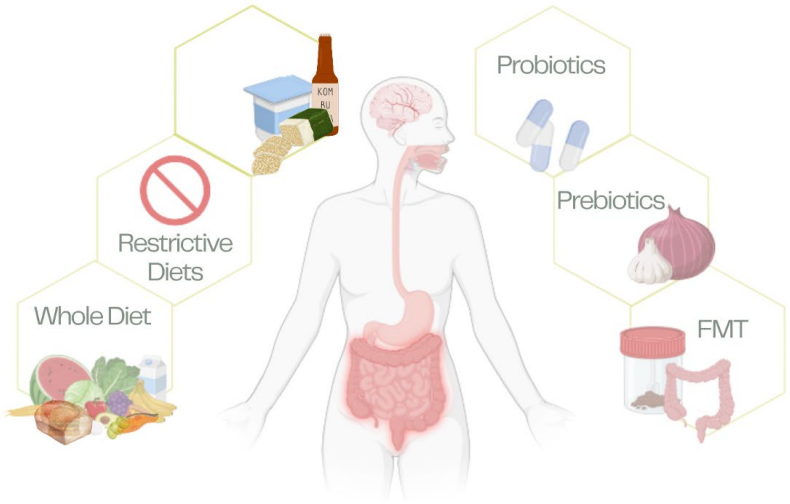
PRIMARY OUTCOME
Between-group difference in change in PHQ-9 score at 6 wk (scale, 0-27, with lower scores indicating less severe depression)

Gao M, Kirk M, Knight H, et al. A ketogenic diet for treatment-resistant depression: a randomized clinical trial. *JAMA Psychiatry*. Published online February 4, 2026. doi:10.1001/jamapsychiatry.2025.4431

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Fermented Food

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Fermentation Facts

Evidence of fermentation during the **Neolithic period** (~12,000 years ago)

Earliest fermentation methods were mostly wine making in the Middle East, Asia and Far East, spreading into the Mediterranean

Similar patterns have been seen for other ferments

Tamang et al. 2020. Compr Rev Food Sci Food Safe



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First links to Wellbeing

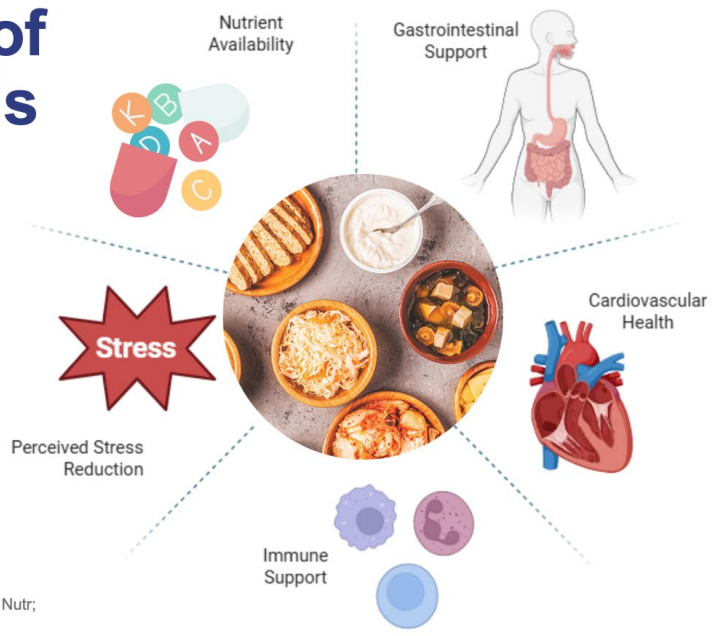


Petrova et al. 2021. Microorgansims

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Health Benefits of Fermented Foods



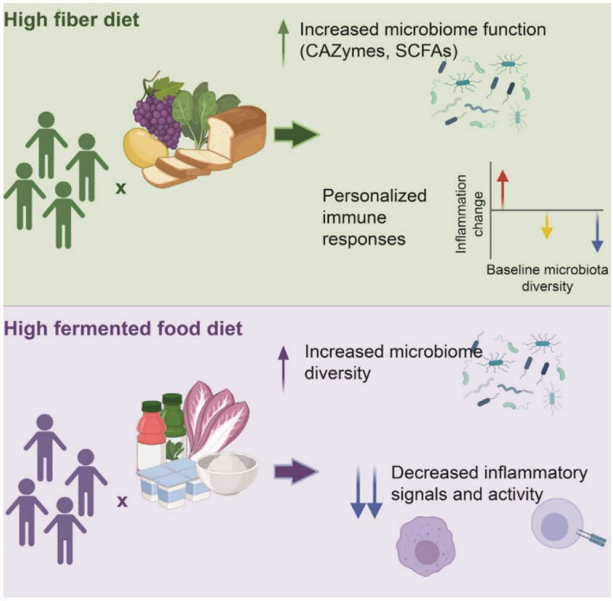
Şanlier et al. 2019. Crit Rev Food Sci Nutr; Santa et al. 2025, Frontiers Nutr; Wastyk et al. 2021. Cell; Berding et al. 2023. Molecular Psychiatry
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Fermented Foods

RCT comparing high fibre and high fermented food diet



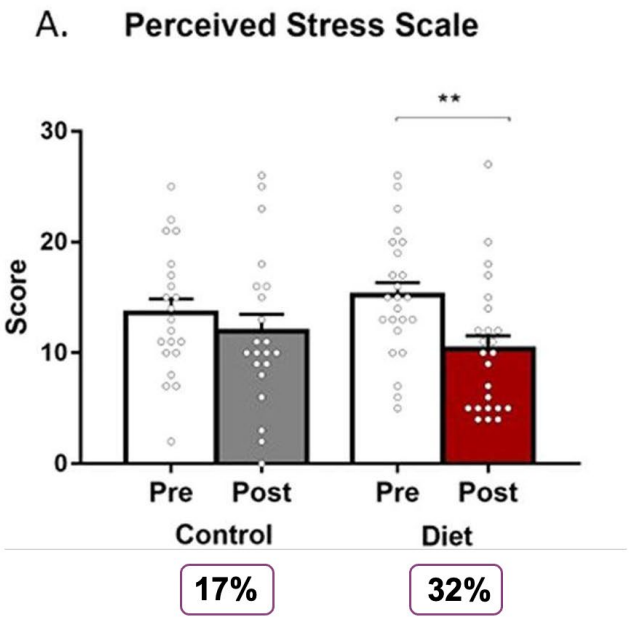
Wastyk et al. 2021. Cell

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Psychobiotic Diet

RCT comparing psychobiotic diet and control diet

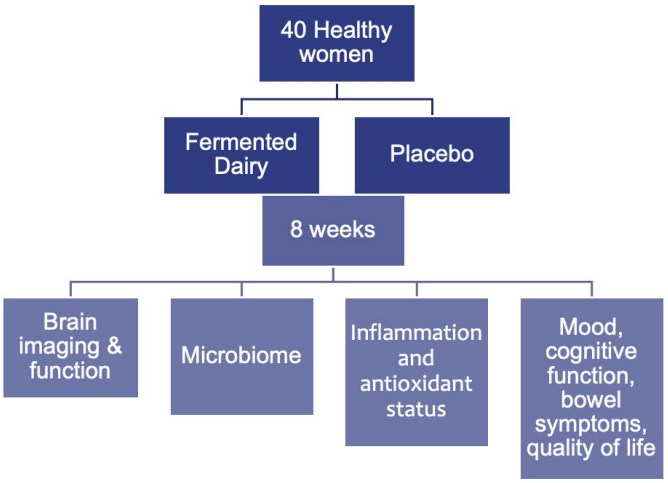


Berding et al. 2023. Molecular Psychiatry

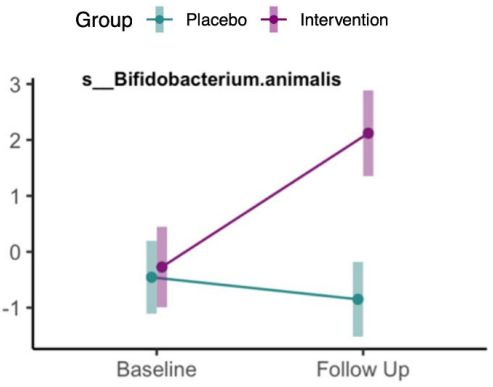
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Fermented Dairy Trial



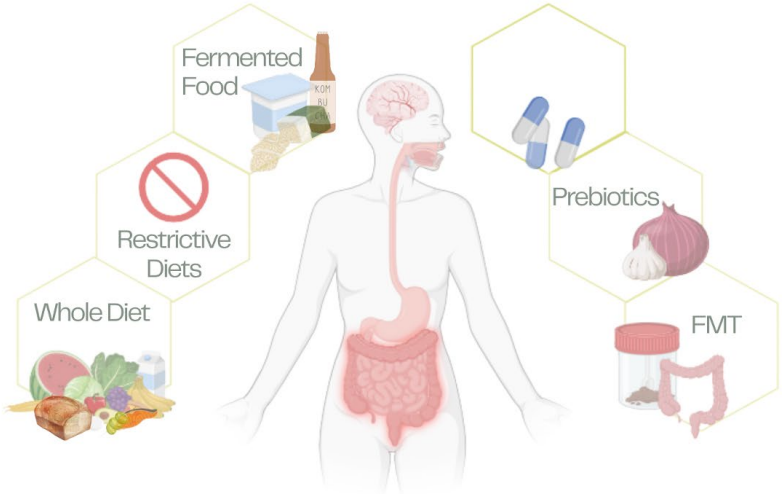
Changes in Bifidobacterium animalis are positively correlated with changes in brain functional connectivity



Marx et al. 2026. Gut

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Probiotics

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Probiotics for Mood Disorders

Some evidence that *Lactobacillus* and *Bifidobacterium* at doses 1-10B CFU might be helpful for symptoms of depression and anxiety.

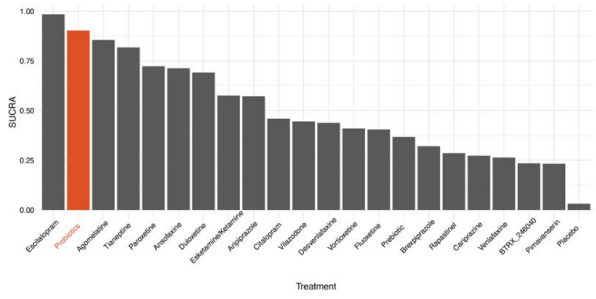
World Federation of Societies of Biological Psychiatry

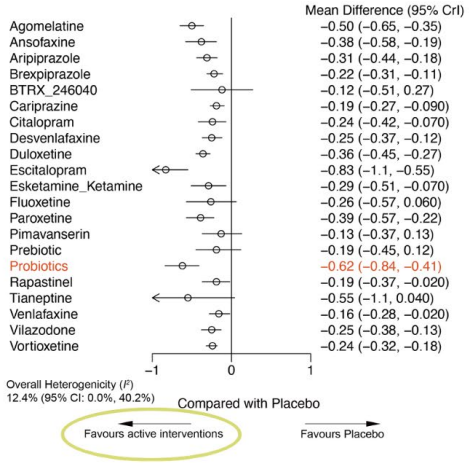
Sarris et al. 2022. World J Biol Psychiatry, Rahmannia et al. 2024. Gut Pathog

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Probiotics can be superior, and non-inferior, to antidepressants for depression



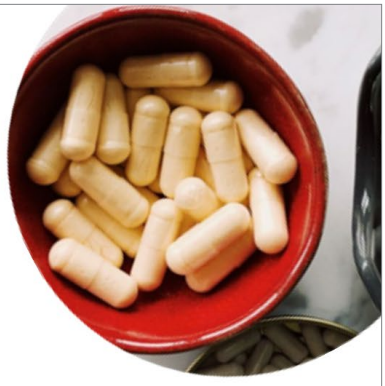


Zhao et al. 2025. Nutr Rev

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How to choose a probiotic



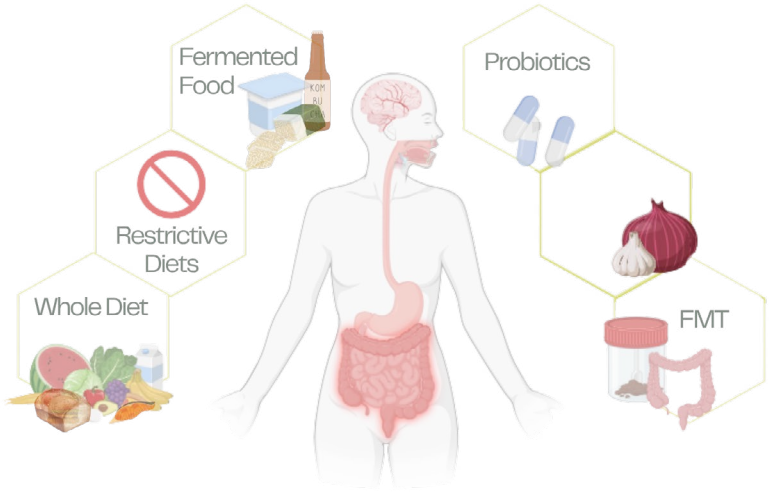
AEProbio

Clinical Guide to Probiotic Products Available in USA

Applications, Dosage Forms and Clinical Evidence to Date - 2023 Edition

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Prebiotics

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Prebiotics for Mood Disorders

- Limited mixed outcome studies
- Emerging evidence on pro-prebiotics - synbiotics

Probiotic, prebiotic, synbiotic and fermented food supplementation in psychiatric disorders: A systematic review of clinical trials

Carlos Ribera [1], Joan Vicent Sánchez-Ortí [2], Gerard Clarke [3], Wolfgang Marx [4], Sabrina Mörkl [5], Vicent Balanzá-Martínez [6]

Affiliations + expand

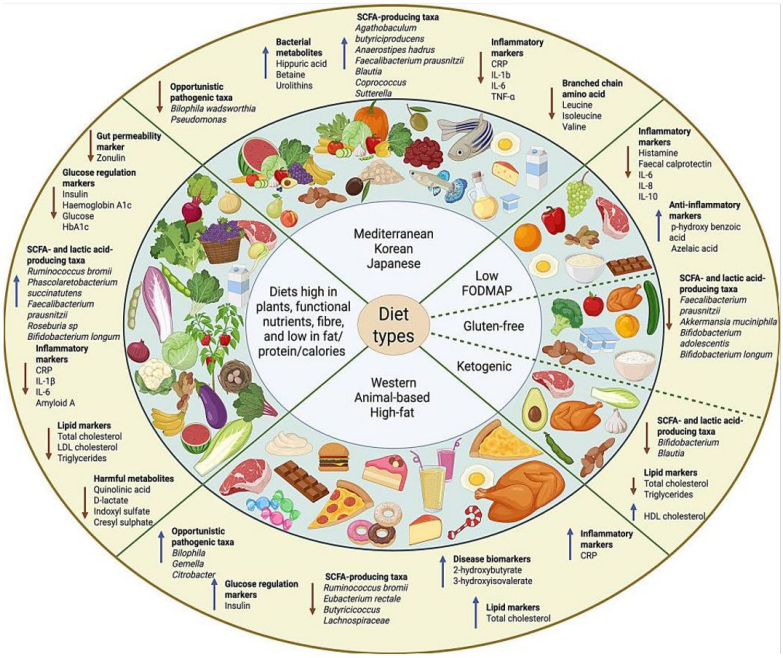
PMID: 38280441 DOI: 10.1016/j.neubiorev.2024.105561

Ribera at al. 2024. Neurosci Biobehav Rev

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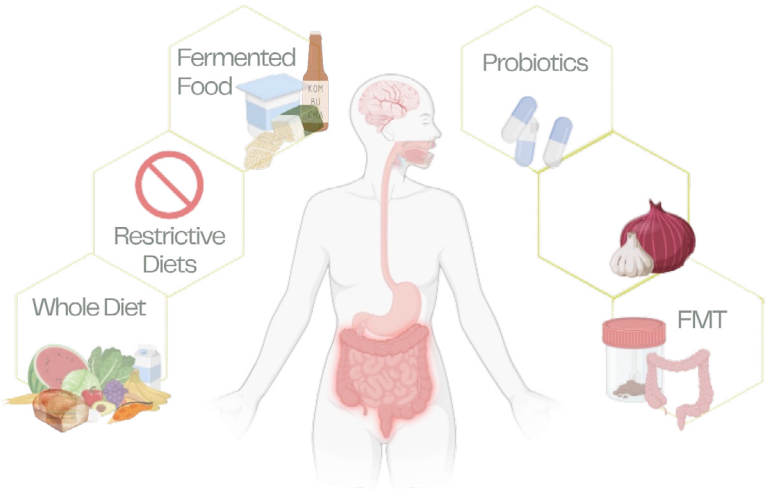
Fibre First: Where Prebiotics Fit In



Aslam et al. 2026. J Transl Med

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FMT

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FMT is a feasible treatment approach for major depression

World-first pilot trial of active FMT compared to placebo for moderate-to-severe depression (n=15)

FMT group showed greater improvements in **gastrointestinal symptoms and quality of life!**

Target area	Feasibility target	Outcome achieved	Target achieved (Yes/no)
Recruitment	Successful enrolment of *n* = 15 participants across a 6-month active recruitment period	15 participants were enrolled in a 2-month period	Yes
Retention	A minimum of *n* = 10 participants to complete study until the 8-week primary endpoint (i.e., a 33% attrition rate).	All 15 participants completed the study until the 8-week primary endpoint	Yes
Adherence to protocol	Participants should: 1. Receive 2 of the total 4 enemas 2. Attend their baseline appointments (in-person) and Week 2 and Week 8 appointments (via telehealth or in-person) 3. Provide baseline and Week 2 stool samples	1. All 15 participants received all 4 enemas 2. Attendance rate of 100% at key appointments. 3. All participants provided baseline stool samples. 14/15 Week-2 stool samples were returned.	1. Yes 2. Yes 3. Yes
Safety	Nil severe and/or serious AEs rated as likely due to study intervention in the active FMT group.	No serious or severe AEs were reported in either group during the 8-week study period.	Yes
Adequacy of blinding	The best achievable outcome for adequacy blinding is participants and researchers correctly guessing allocation at a rate of 50% (the rate due to chance). Whilst we are not likely to be statistically powered to measure this outcome, we aim to reach an outcome approaching 50%.	35.7% of participants correctly guessed treatment allocation (Fisher's Exact *P* = 0.45).	Yes

Green et al. 2023. Can J Psychiatry

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Implementation

Practical Steps for Implementation into Practice

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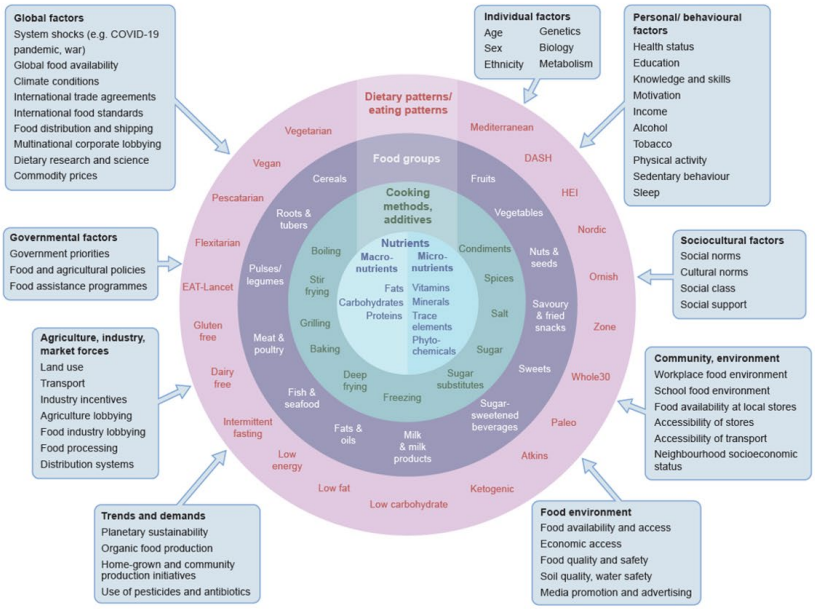
5 Questions to Ask Yourself

- HOW READY IS THE PERSON TO MAKE A CHANGE RIGHT NOW?
- DOES THE PERSON CURRENTLY HAVE THE CAPACITY TO FOLLOW ADVICE?
- IS THEIR MENTAL STATE SUFFICIENTLY STABLE TO ENGAGE IN BEHAVIOUR CHANGE?
- DO THEY HAVE ACCESS TO FOOD AND BASIC RESOURCES?
- CAN THEY REALISTICALLY IMPLEMENT EVEN A VERY SMALL CHANGE THIS WEEK?



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What do I need to consider?



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Assessing the Individual

Where to focus?

Psychosocial context
- Food insecurity
- Housing
- Cultural practices
- Cooking skills

Cognitive load
- Planning
- Motivation

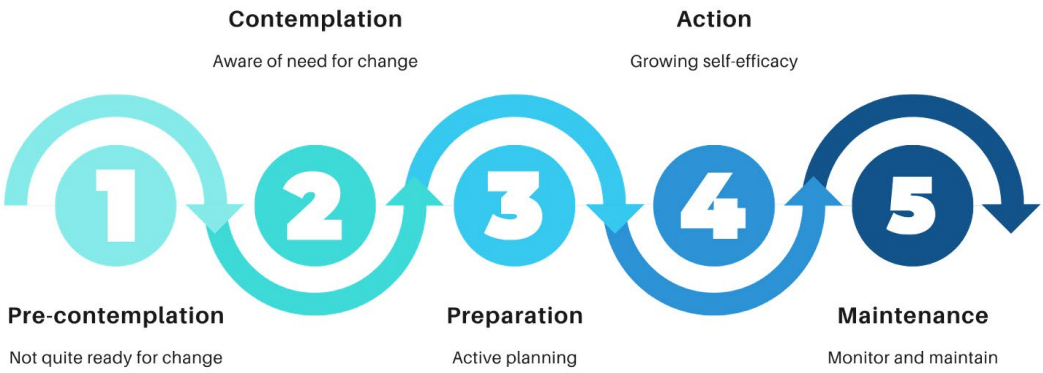
Support systems
- Family
- Peers
- System assistance

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The 5 Stages of Change

The Transtheoretical Model



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Assessing your client's stage of change



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HOW MUCH DO YOU KNOW ABOUT THE LINKS BETWEEN FOOD INTAKE AND MENTAL HEALTH?

WOULD YOU BE OPEN TO TRYING TO MAKE SOME CHANGES TO YOUR EATING?

HAVE YOU ATTEMPTED TO MAKE ANY CHANGES TO YOUR EATING IN THE PAST?

WHAT HAS WORKED FOR YOU WHEN MAKING CHANGES? WHAT GOT IN THE WAY?

WHAT HAVE YOU FOUND WORKS FOR YOU WHEN TRYING TO MAINTAIN GOOD HABITS?



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Communicating Diet-Related Messages

- Pick the right time
- Focus on the patient
- Take small steps
- Support capacity building
- Break through barriers
- Provide follow up

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Pick the right time

- **Work with symptoms** dynamics
- Identify **readiness** windows
- **Align** with treatment goals
- **Clarify:** start, focus, duration review
- **Communicate** with the team



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Focus on the patient

- Centre **their** goals
- Active + reflective **listening**
- Ask **what matters** most
- Attend to **non-verbal cues**
- **Share** decisions
- **Flexibility** is essential

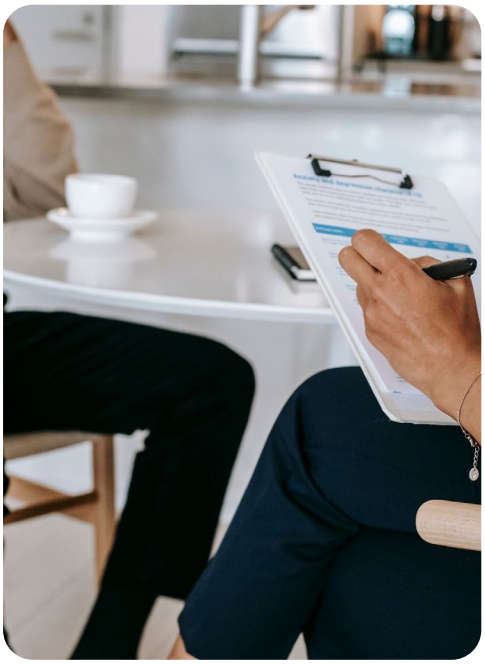


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Take small steps

- Use **Motivational** interviewing
- Help set 1-3 **SMART goals**
- **Link** goals to values
- Acknowledge immediate **needs**
- Help to plan short- and long-term actions



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Motivational Interviewing

Empathise: Active listening, normalising, affirming, non-judgement

Elicit: Open-ended questioning, summarise, mirror

Collaborate: Explain your process, elicit agreement and commitment

Connect: Eye contact, acknowledging, mirroring, affirm agency

Evoke: Reflect internal conflicts, overt, make concrete

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SMART Goals

I WILL SWAP MY USUAL SWEETS AT MID-MORNING FOR A HANDFUL OF NUTS ON THREE WORKDAYS NEXT WEEK

I WILL PLAN AND COOK 2 DINNERS AT HOME EACH WEEK FOR THE NEXT 4 WEEKS

BY THE END OF THIS MONTH, I WILL EAT AT LEAST ONE LENTIL-BASED DINNER PER WEEK

I WILL EAT 1 FRUIT (BANANA OR APPLE) EACH AFTERNOON FOR THE NEXT 2 WEEKS

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Support capacity building

- Build **self-care** skills
- Support **confidence** + competence
- Provide accountability tools
- Be authentic and **BELIEVE**
- Try alternative approaches



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Break through barriers

- **Change is challenging** – acknowledge it
- **Respect** concerns
- Consider **culture** + context
- Discourage ‘all or nothing’ approach
- **Challenge** unhelpful ideas
- Address **knowledge** gaps
- Use **real-life,** applicable examples



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Barrier: Lack of Knowledge or Skills

PROVIDE EVIDENCE-BASED FOUNDATIONAL KNOWLEDGE ON HEALTHY DIET (USING CULTURALLY-APPROPRIATE RESOURCES)

SUPPORT DEVELOPMENT OF BASIC SKILLS (E.G., PLANNING, SHOPPING STORAGE)

DISCUSS EASY FOOD OPTIONS (E.G., HEALTHY TAKEAWAY OPTIONS AND PRE-PREPARED MEALS) AND HELP IDENTIFY APPROPRIATE CHOICES

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Discuss **Foundational Concepts**

Protein	Cereals and Starchy Vegetables	Vegetables
Tinned Tuna / Salmon	Baked Potato / Sweet Potato	Pre-Cut Salad
Eggs	Wholemeal Pita	Frozen Veg
Beans	Multigrain bread	Roasted Veg
Tofu	Brown Rice	Stir-Fry Veg

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Discuss **Foundational Concepts**



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Provide follow-up

- Not everything fits one session
- **Flexible** approach but **consistent messaging**
- Normalise “start-stop” or “re-do” approach
- **Commit** to long-term support
- **Communicate** with consumers, carers, team



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Plant seeds

Elicit self-reflection through targeted open-ended questions



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Encourage a **growing mindset**

- Self-compassion
- Learning from lapses
- Avoid all or nothing thinking



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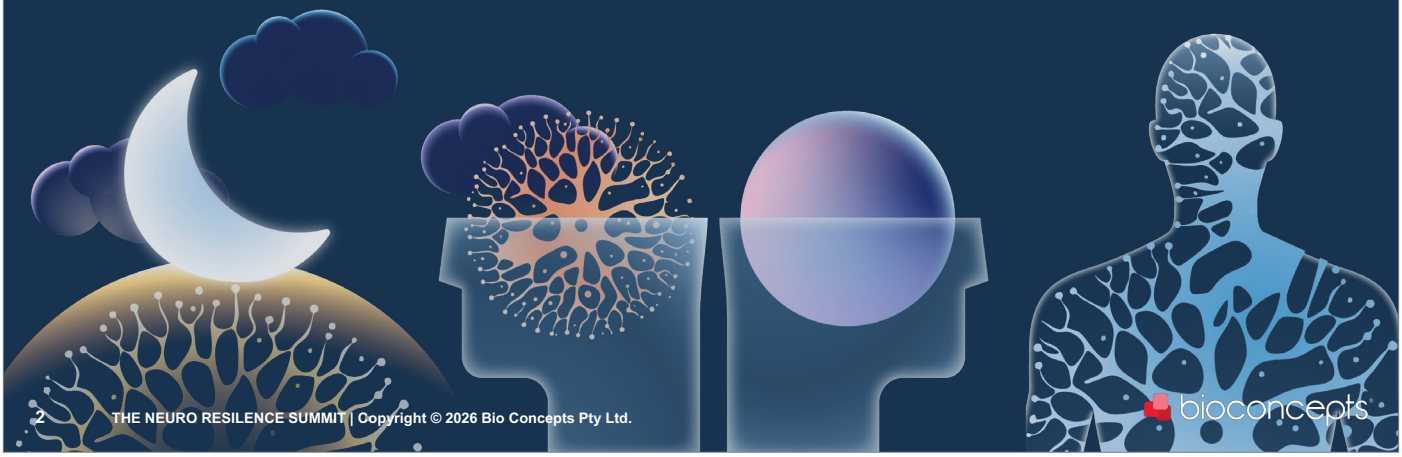
Q&A Q&A Q&A Q&A

FEATURING

DR TETYANA ROCKS

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Brain Health Fitness:
Neuroplasticity and the Future of Human Health



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KEYNOTE SPEAKER

Prof. Selena Bartlett



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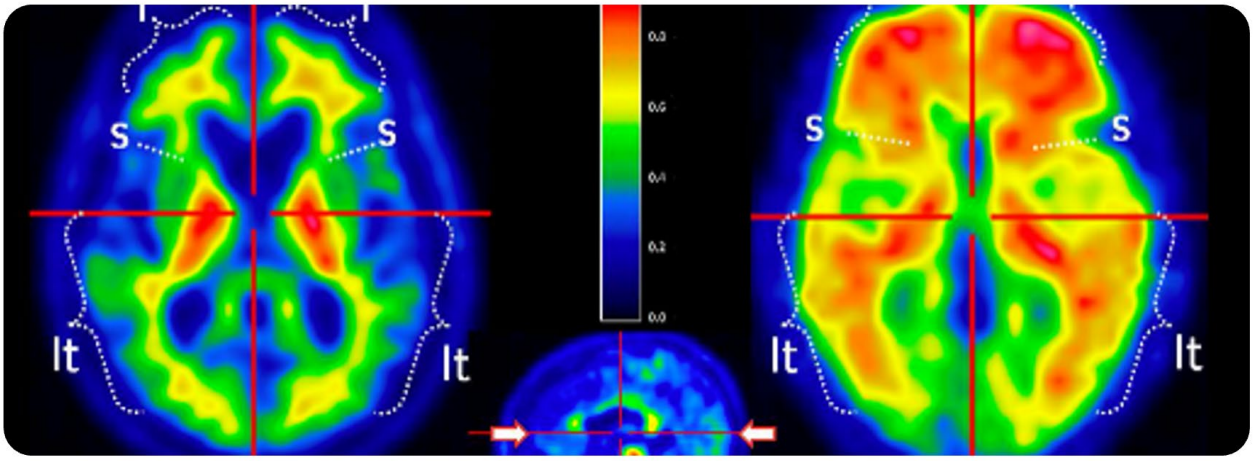
OUTLINE

1. Neuroscience has transformed how we understand the mind
2. The attention economy: screens, stress and connection
3. Early experience shapes the developing brain
4. Adverse Childhood Experiences and lifelong health
5. Neuroplasticity: change is possible
6. Thriving minds in the age of AI
7. The neuroscience of two: three tools for connection
8. Future of human health: brain, body and nature

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Neuroscience Transformed Our Understanding of Our Brains and Minds



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Working Memory Test

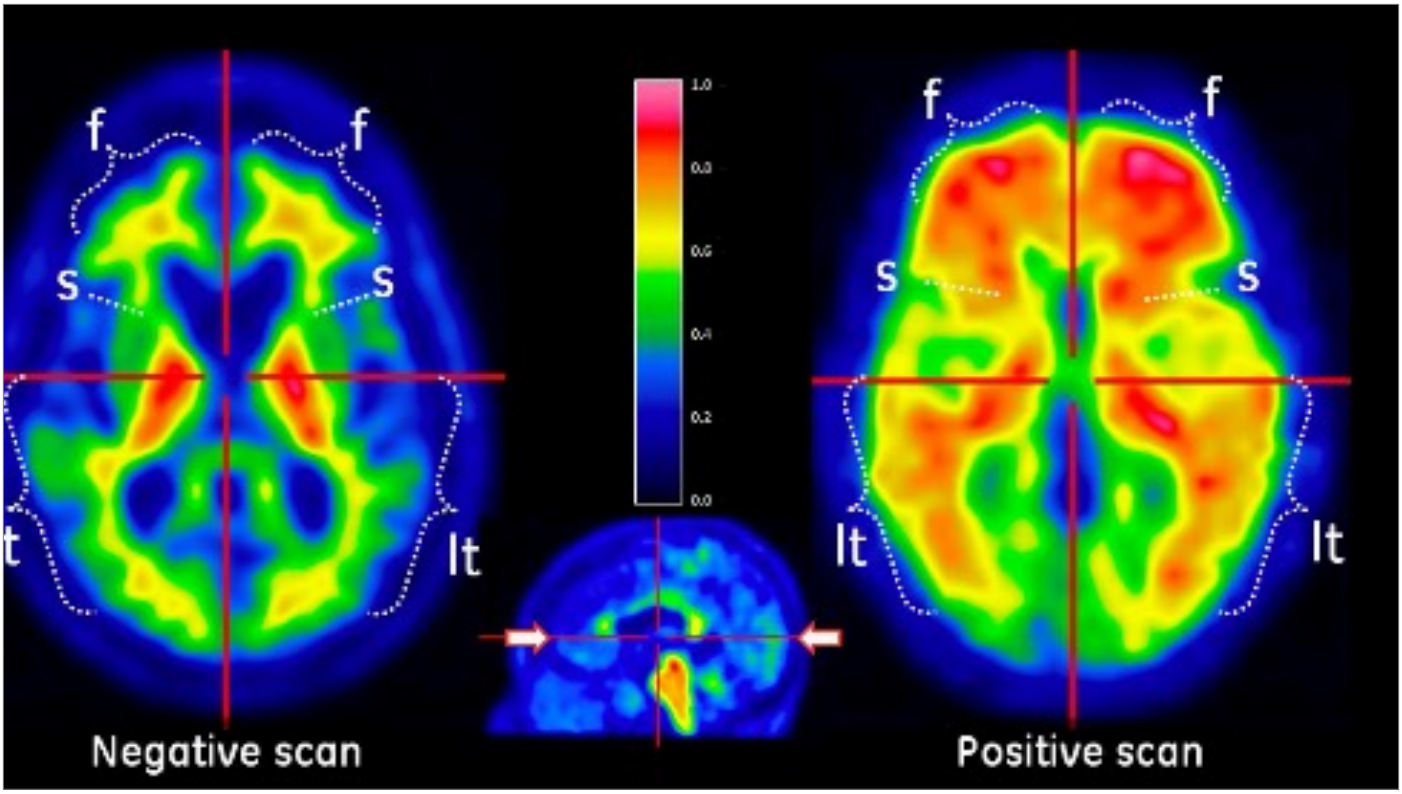
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Debrief and Reflection

Name the 3 things you are doing daily for your brain health and neuroplasticity?

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Excess Screen-Time Increases Stress



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Screens are designed to capture attention — all of our attention



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1975 “Still Face Experiment”
Dr. Edward Tronick



https://www.tracydennistiwary.com/

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frontiers | Frontiers in Public Health

TYPE Original Research
PUBLISHED 27 April 2023
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Mougharbel F, Chaput J-P, Sampasa-Kanyinga H, Colman I.

Longitudinal associations between different types of screen use and depression and anxiety symptoms in adolescents

Fatima Mougharbel[1,2], Jean-Philippe Chaput[2,3], Hugues Sampasa-Kanyinga[2], Ian Colman[3,4], Scott T. Leatherdale[5], Karen A. Patte[6] and Gary S. Goldfield[2]*

[1]School of Population Health, University of Ottawa, Ottawa, ON, Canada, [2]Healthy Active Living and Obesity Research Group, Children's Hospital of Eastern Ontario Research Institute, Ottawa, ON, Canada, [3]School of Epidemiology and Public Health, University of Ottawa, Ottawa, ON, Canada, [4]Centre for Fertility and Health, Norwegian Institute of Public Health, Oslo, Norway, [5]School of Public Health Sciences, University of Waterloo, Waterloo, ON, Canada, [6]Department of Health Sciences, Faculty of Applied Health Sciences, Brock University, St. Catharines, ON, Canada

Social media is part of everyday life, with 5.24 billion users worldwide.

Average time spent on social media is 2.7 hours, totalling 5 hours of screentime per day.

Simultaneously, there is a global rise in mental health issues, with a 25% increase in anxiety disorders and a 28% increase in depressive symptoms, primarily affecting young adults.

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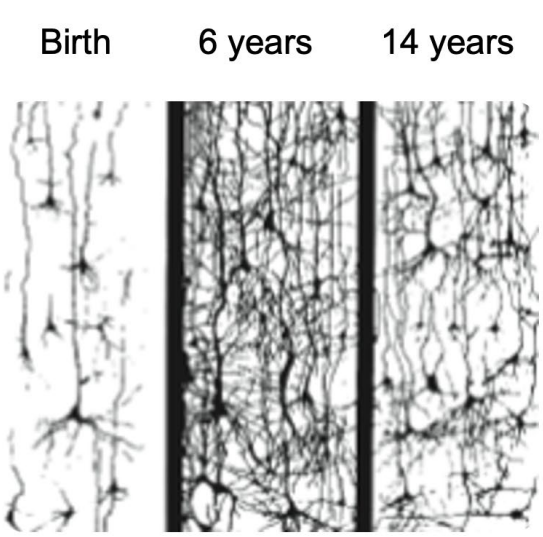


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Early Life Experiences Shape the Brain and Mental Health



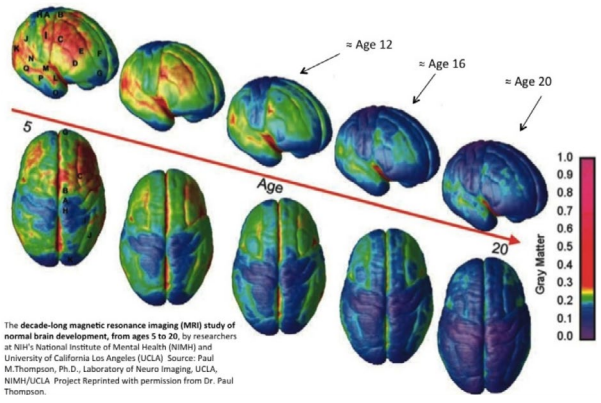
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Brains are Not Fully Developed Until 22-25 Years Old



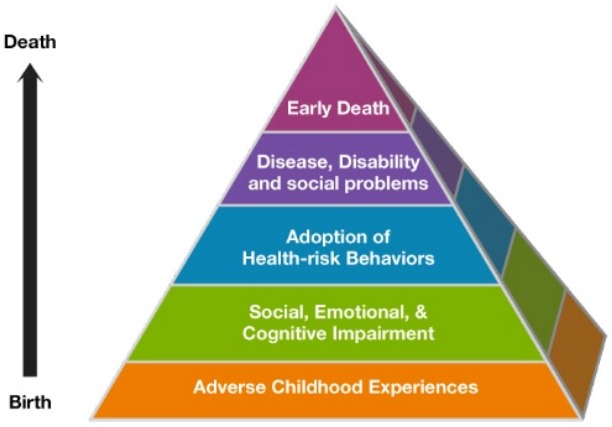
The above time-lapse study was conducted over 10 years. The darker colours represent brain maturity (brain development). It's now understood the brain continues developmental changes through age 22 on average for girls/women and 24 on average for boys/men).

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Before Screens

Adverse Childhood Experiences (ACES)



Kaiser Permanente; Study of 17K Californians

ACE Study:

- **17K** Kaiser HMO Members
- **75%** Caucasian
- **75%** Attended/graduated college
- **56%** 50 years old+

Fellitti and Anda (100 papers+)

Early life experience impacts the prefrontal cortex affecting impulse control, cognition. Foundational skills cannot be applied across the life span

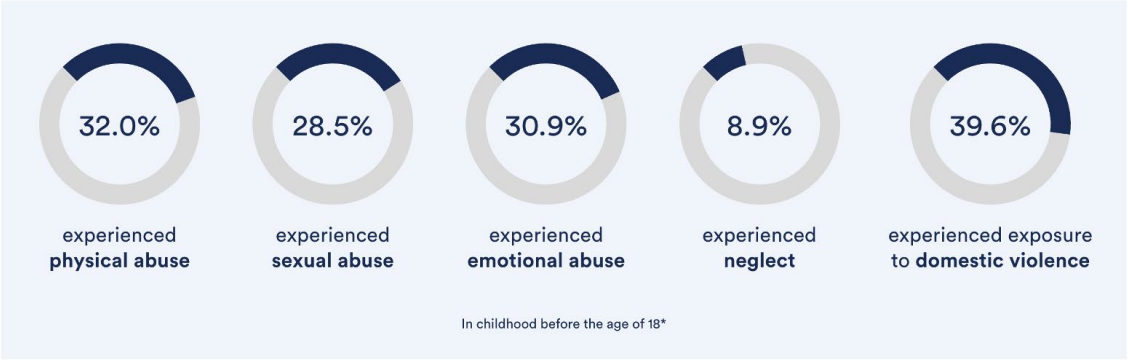
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Childhood Maltreatment is Common

VIEW MORE

Across the Australian population (ages 16-65+) we found:



Australian Child Maltreatment Study (ACMS) Findings
https://www.acms.au/

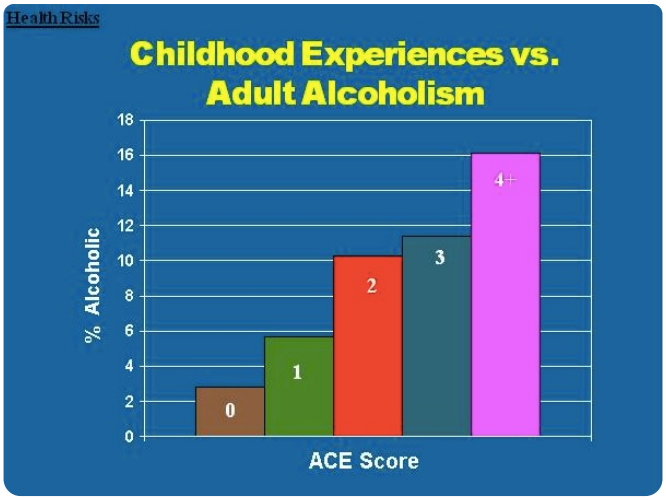
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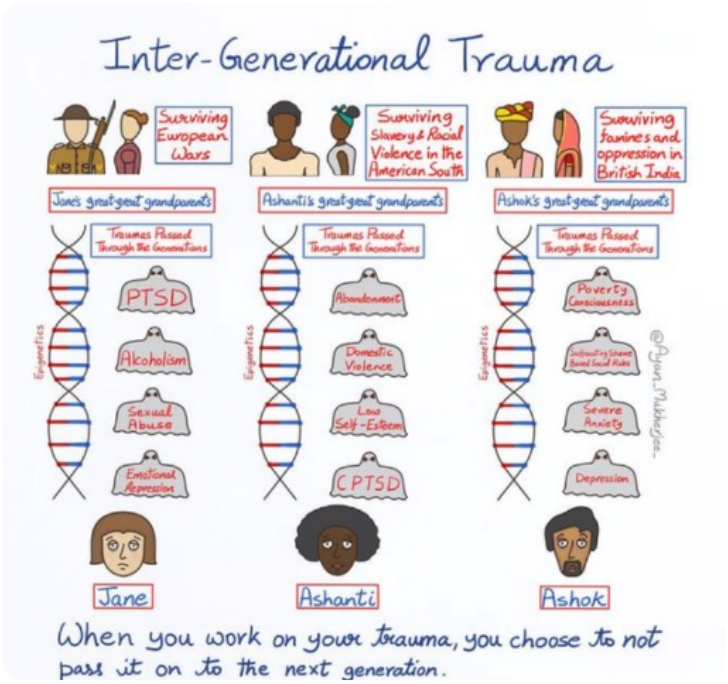
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Felitti, V. J., Anda, R. F., et al.
Title: Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study
Link: https://doi.org/10.1016/S0749-3797(98)00017-8

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Neuroplasticity Teaches us Change is Possible!



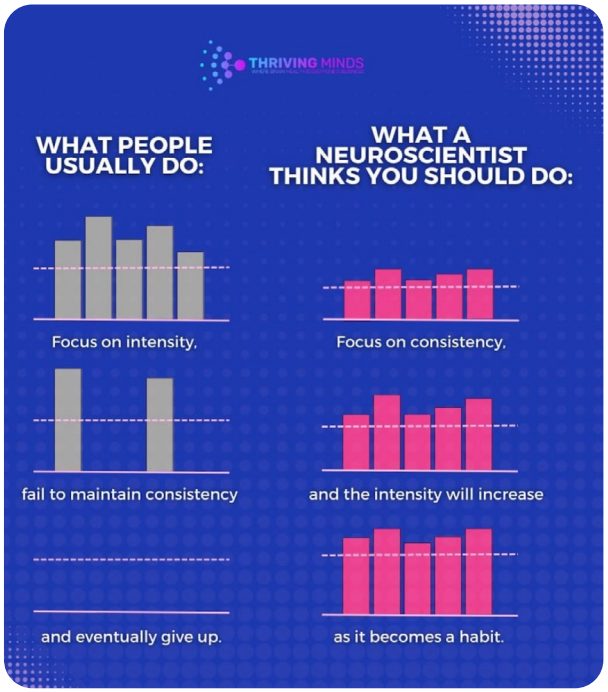
Pathways in the brain as revealed by brain imaging (DTI)

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It is Hard to Change

Activating New Synapses



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Thriving Minds in the Age of AI

AI is changing the conditions under which the brain pays attention, learns, works and recovers.

Neuroresilience

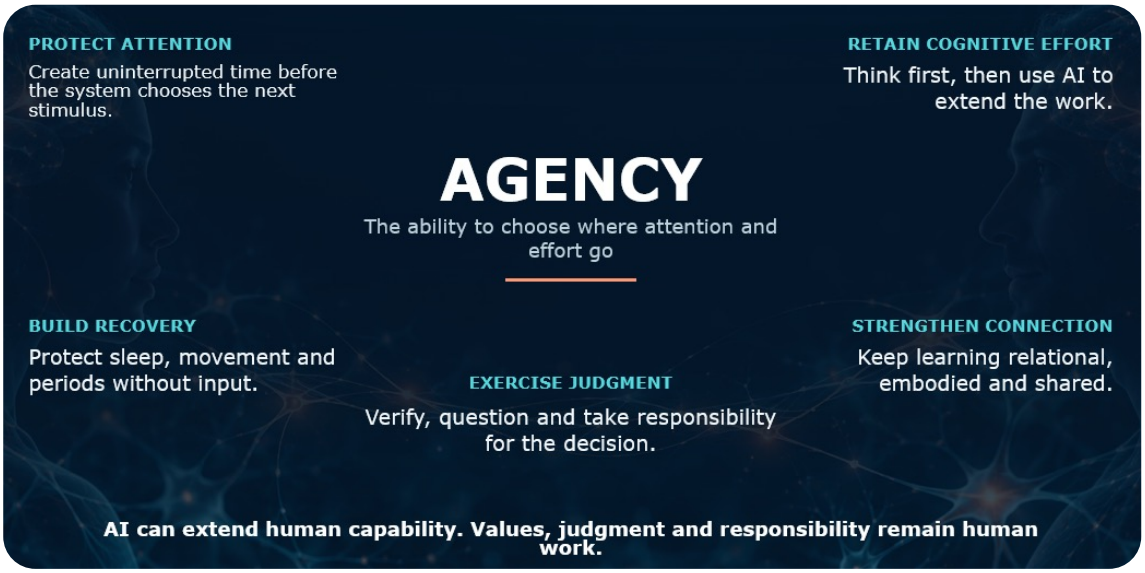
The capacity to adapt and recover while retaining agency over what we practice, strengthen and become.

The question is no longer whether we will use AI. It is what we choose to delegate and what must remain human.

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Conditions that protect human intelligence



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Building digital resilience

The five pillars of lasting professional protection online

Set Boundaries

Define which platforms you engage on, what topics you discuss, and who gets access to your personal life.

Audit Your Footprint

Regularly review your public profiles, Google your name, and remove unnecessary personal data online.

Build Your Support Network

Cultivate allies, mentors, and peers who can validate, advocate, and mobilise on your behalf when needed.

Prioritise Mental Health

Regular check-ins with a therapist, peer support groups, and digital detox routines are not optional — they're essential.

Know Your Legal Rights

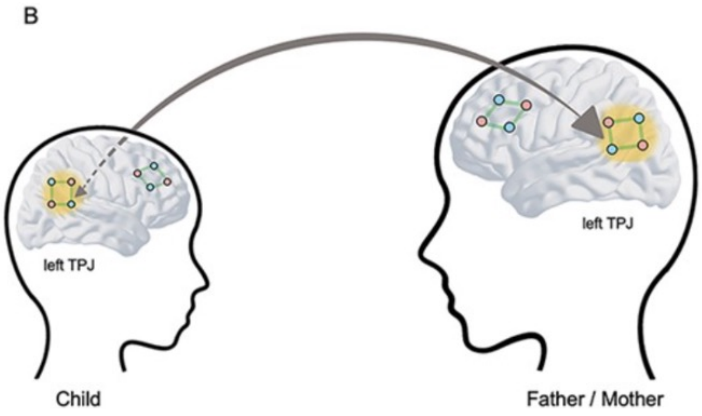
Understand defamation, harassment law, and workplace protections in your jurisdiction before you need them urgently.

You have the right to participate in professional life without fear.

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Our Brains Sync When We are Present and See the Person in Front of Us Inter-Brain Synchrony



PARENT-CHILD and TEACHER- STUDENT INTERBRAIN SYNCHRONY/MIRROR NEURONS Liu et al., 2024 Cerebral Cortex

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Inter-brain synchrony – neuroscience of 2 plus



Professor Joy Hirsch research shows that when people co-attend (look together, listen together), their brain activity can synchronise.



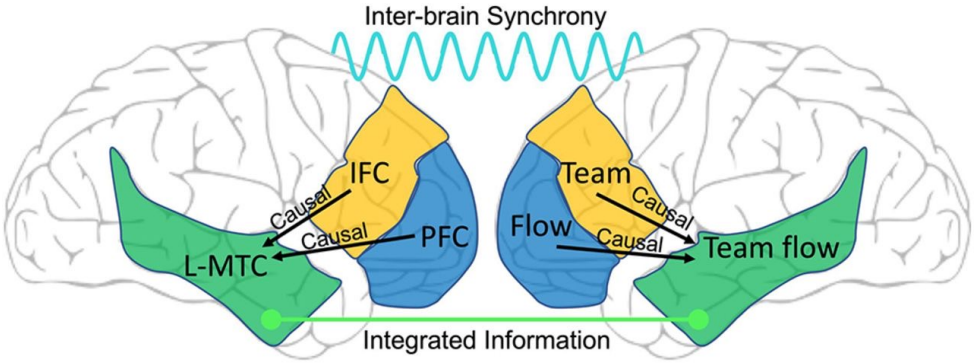


Episode #159. Maximise your Mind by Working with Others. Why TWO brains are better than one, the neuroscience of two! Dr Joy Hirsch,...
THRIVING MINDS PODCAST

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How Do We Leverage the Neuroscience of 2 for Healing and Building



"Team flow occurs when a group functions in a high task engagement to achieve a goal, commonly seen in performance and sports. Team flow can enable enhanced positive experiences, as compared with individual flow or regular socializing".

Mohammad Shehata et al. eNeuro 2021;8:ENEURO.0133-21.2021

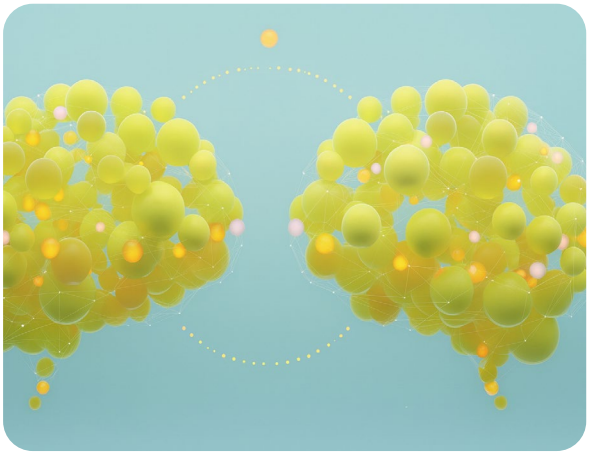
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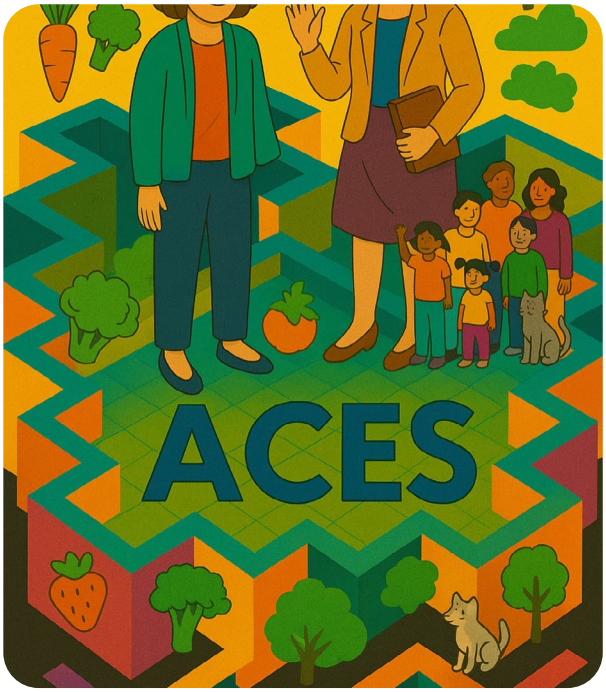
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Tool #1

Healing happens between us

– take a moment to connect





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Tool #2

Every person's journey is unique



Neuroplasticity takes a lot of courage and time!

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Tool #3

Being truly seen heals us



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Debrief and Reflection

When have you felt truly seen?

For 60 secs, simply write down
the people,
the places,
the times

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Future of Human Health

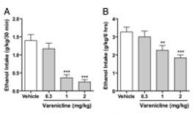
Brain health and wellbeing is a basic human right and not only someone else's problem

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Spent 20 years on the Biological Approach to Treating Addictions

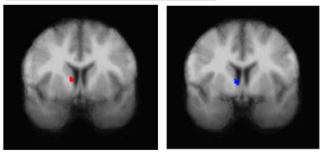




Steensland et al., 2007 PNAS.

Preliminary fMRI Analysis: (Ramachandi and Heilig)

BOLD activation during cue phase of Alcohol-MID task



PLACEBO (n=7)
Contrast: Alcohol vs. Neutral
p<0.005

VARENICLINE (n=8)
Contrast: Varenicline vs. Placebo for Alcohol vs. Neutral
p<0.01

Randomized, double-blind, placebo-controlled trial
50 subjects, smokers and non-smokers



South Burnett
Health News

Home Page

Nanango Pharmacy Graduate Develops New Anti-Addiction Drug



18th February 2008: University of Queensand pharmacy graduate **Dr. Selena Bartlett** - whose parents ran the **Nanango Pharmacy** until November last year - is starting clinical trials of a new drug that could curb addictions such as smoking, drinking, gambling and even depression.

The drug - marketed as *Chantix* or *Champix* by **Pfizer** - has reduced alcohol consumption in laboratory rats by **50 percent** and will be trialled in humans next month. Champix works by latching onto 'good feeling' receptors in the brain to block cravings for addictions such as **nicotine** or **alcohol**.

Dr Bartlett is currently the Director of the Preclinical Development Group at the Ernest Gallo Clinic and Research Centre at the University of California in San Francisco. The Champix trials are just one of 10 major projects for Dr Bartlett's lab team, which is also working on reducing ethanol consumption and new drug screening technologies.

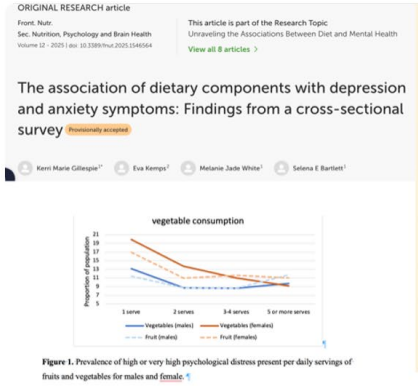
Dr. Bartett's interest in the area was inspired by a family tragedy. You can read the full story of her exciting new development **by clicking here**

***Photo:** Former Nanango-ite Dr. Selena Bartlett is a month away from human trials of a revolutionary new drug that could combat many forms of addiction (photo copyright to and courtesy of the University of Queensland)*

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Brain health can be improved through the mind and body


ORIGINAL RESEARCH article

Front. Nutr.
Sec. Nutrition, Psychology and Brain Health

This article is part of the Research Topic
Unraveling the Associations Between Diet and Mental Health
View all 8 articles

The association of dietary components with depression and anxiety symptoms: Findings from a cross-sectional survey

Provisionally accepted

Kerri Marie Gillespie, Eva Kemps, Melanie Jade White, Selena E Bartlett

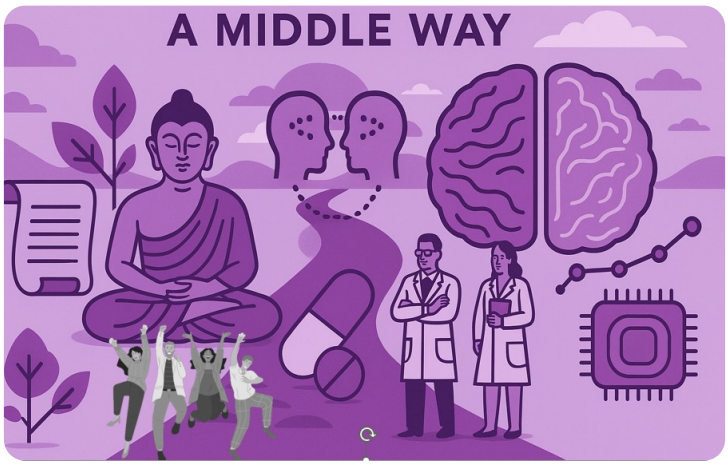
Figure 1. Prevalence of high or very high psychological distress present per daily servings of fruits and vegetables for males and female.



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Integrating Human Experiences with Modern Scientific Advances



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BRAIN VITALITY INDEX

	YES	NO
Morning ritual with stretch, breath, hydrate, intention	☐	☐
Stand tall and breathe deep	☐	☐
20+ minutes of joyful movement		
Scrolling and social media are reduced	☐	☐
7–9 hours of sleep each night	☐	☐
Kindness and gratitude for small things	☐	☐
Connect with someone or nature	☐	☐

With love and care,

Dr. Selena Bartlett

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Reset Your Brain in 90 seconds

Train Your Brain for Fitness Respond Rather than React to Stress



1. Panoramic view



2. Cold showers



3. Reduce sugar and processed food. Try raw nuts

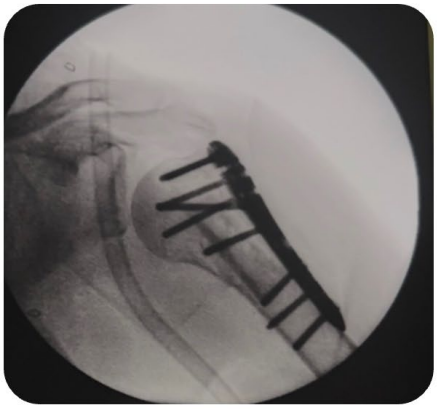


4. Reach out to one person. Train social connections to be happier

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Mind Your Bones



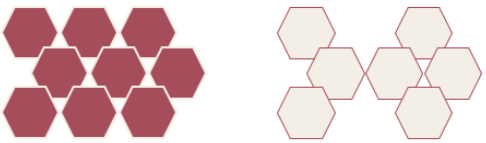
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Bones, Brain Health and Neuroplasticity

Osteopenia is the yellow light. Osteoporosis is the red.

INSIDE THE BONE



Healthy density

Thinner struts, wider gaps

854,000

Australians living with osteoporosis or osteopenia (self-reported, so likely an undercount)

1 in 4+

Women aged 75 and over with osteoporosis

105,000

Hospitalisations for minimal-trauma fracture, age 45+, in 2021–22

Source: Australian Institute of Health and Welfare, osteoporosis reporting (2022; hospitalisations 2021–22).

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Let Nature Do The Heavy Lifting



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Debrief and Reflection

What can we do together to build neuroresilient people using the knowledge of inter-brain synchrony and neuroplasticity?

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Summary

1. We don't need to perfect. **We need to be present.**
2. Accept where we come from, **and we can change inter-generational cycles for good.**
3. **Neuroresilience means we have the power to take daily actions because of neuroplasticity to build thriving minds and lives.**

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Awareness, Education and Practice





www.profselenabartlett.com

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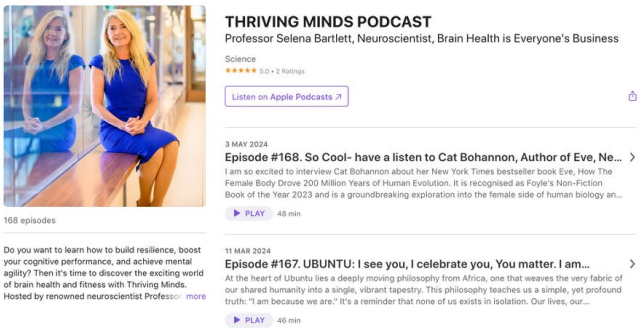
Access Free Resources

Thriving Minds Podcast
"Brain Health Is Everyone's Business"

Podcast

Website

LinkedIn



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Q&A
Q&A
Q&A
Q&A

FEATURING

PROF SELENA BARTLETT

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SPEAKER

Dr Frank R Golik

MBBS (Hons Qld) FRACGP
FACNEM

WIN HEALTH COACH CLINICS
Suite 2, North Park BC,
6 Babarra Street, Stafford, Q 4053

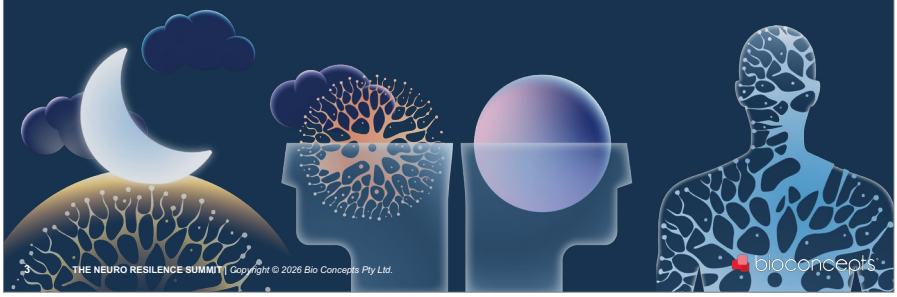
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Is it ADHD or something else?



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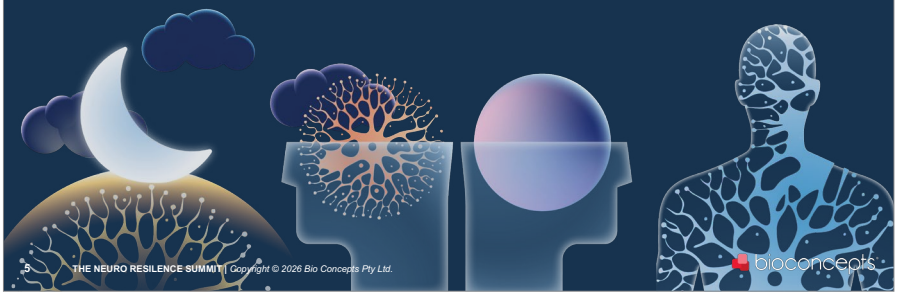
OUTLINE

1 Diagnosis and features of ADHD: 3 types
2 Possible causal factors
3 Relevant testing workup
4 Other conflicting features and symptoms
5 Management, treatments, and concerns
6 Is it truly ADHD or something else?
7 Possible alternative diagnoses
8 References

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Diagnosis and features of ADHD: 3 types



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ADHD as an issue of **Nutrition, Lifestyle, Genetics, and Environmental toxins**









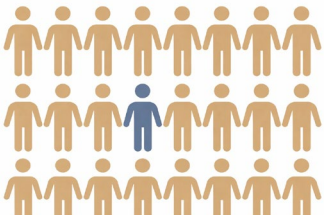


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Incidence
ADHD IN AUSTRALIA

Around 1 in 20 Australians have ADHD, or over 1 million people.



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ADHD

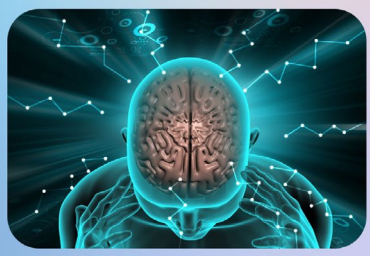
- ADHD is merely a *diagnosis of exclusion.* Incidence range: 9 to 20%
- ADHD is maturational DELAY and IMBALANCE in the brain
- ASD is only 10-20% of an issue; chronic (neuro) inflammation is a key factor
- ADHD shares 30 features of ASD
- ADHD folk often have low adrenal function, low dopamine, low testosterone, poor exercise in nature, and poor sleep
- ADHD is usually with HIND-BRAIN dominant + high sympathetic tone; low FORE-BRAIN; plus, often left-brain deficiency
- Poor sleep and screen-time/devices promote ADHD

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Brain Disharmony

- Hind brain/cerebellum/medulla: dominance in ADHD
- Fore-brain/frontal lobe: low in ADHD
- **Right brain**: creativity, music, art,
- intuition, imagination
- **Left brain:** logic, science, speech, cognitive function, sense of time
- Speech starts in cerebellum



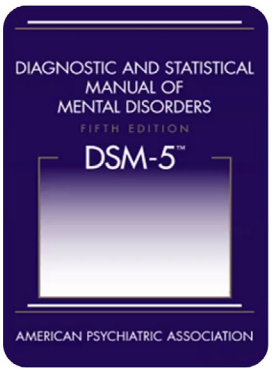
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Diagnosis of ADHD

3 Subtypes:

- Inattentive
- Hyperactive/Impulsive
- Combined



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ADHD

INATTENTIVE SYMPTOMS

- Fails to give close attention, makes careless mistakes
- Difficulty with sustained attention
- Not listening
- Not following instructions
- Difficulty organizing tasks and activities
- Avoids tasks requiring sustained attention
- Loses things
- Easily distracted
- Forgetful in daily activities

HYPERACTIVE / IMPULSIVE SYMPTOMS

- Talks excessively
- Fidgeting or moving
- Leaving seat in appropriately
- Running / climbing inappropriately
- Unable to take part in leisure activities quietly
- On the go / driven by motor
- Blurts out answers
- Difficulty waiting turns
- Interrupts others

CRITERIA

- < 17 – 6+ symptoms on either scale
- 18+ – 5+ symptoms on either scale
- Combined – meets criteria for both scales

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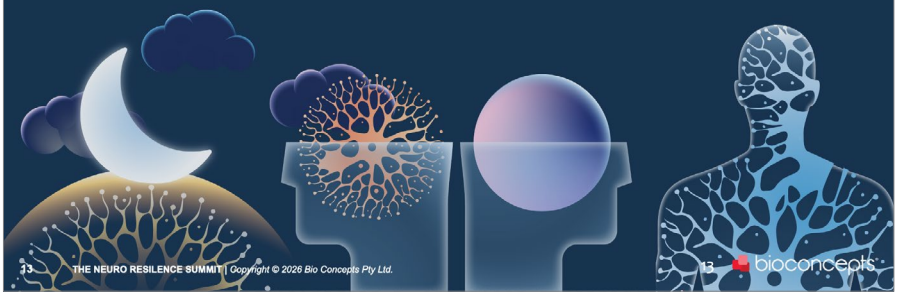
Additional Requirements

- Childhood Onset <12 yrs
- Must have symptoms in 2+ settings
- Symptoms present for at least 6 months
- Symptoms must cause impairment
- Symptoms assessed in context of age, developmental level and intellectual ability
- Differential Diagnoses
- Assessment of strengths/protective factors



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Possible Causal Factors



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Contributors to ADHD

- Genetics and family history/trauma
- Low Dopamine and Noradrenaline levels
- Diet: (opposite to Failsafe/Feingold) - Highly addictive, processed "foods" with food additives: preservatives, dyes, sugars, stabilisers, MSG/glutamates, salicylates, amines/histamine, etc
- Screen-time addiction; childhood trauma & maltreatment
- Lack of play and healthy exercise
- Nutrient deficiencies: iron, zinc, magnesium, lithium, copper
- Heavy metal toxicity: lead, aluminium, and mercury
- Infections (candida, viral, tick-borne), parasites, mould, mycotoxins



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Neuroinflammation in ADD/ADHD

Brian Hooker, Ph.D., chief scientific officer for Children's Health Defense and co-author of "Vax-Unvax: Let the Science Speak," cited a study he conducted with Neil Miller in 2021 that found fully vaccinated children were 20.8 times more likely to be diagnosed with ADD/ADHD compared with their unvaccinated counterparts.

The study used a survey-based instrument; but all cases of ADD/ADHD were verified using medical chart abstraction, Hooker said.

Hooker told The Defender:

"It is difficult to narrow down a single mechanism for the connection between ADHD and vaccine components — **although mercury and aluminum are both neurotoxins.**

"But the neuroinflammation that occurs especially from early childhood vaccines does **create a condition where brain immune cells [astrocytes and microglia] are in a constant state of immune activation, causing damage to neurons.**"

Lawrence B. Palevsky, MD

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7268563/
https://childrenshealthdefense.org/defender/1-in-9-kids-diagnosed-adhd-cdc-says/?utm_source=luminate&utm_medium=email&utm_campaign=defender&utm_id=20240530

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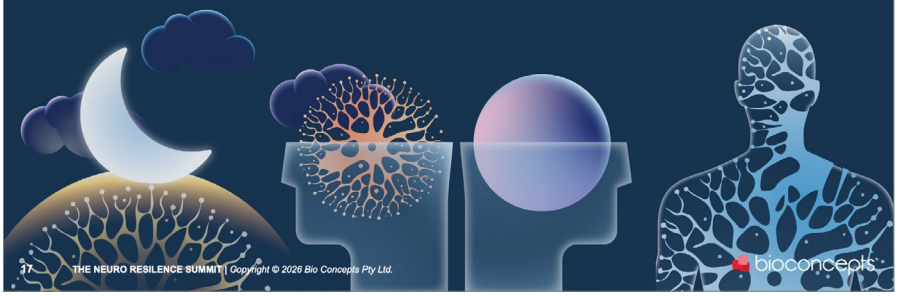
Neuroinflammation contributions

- Air Pollution
- Environmental Toxins
- Medicine
- Soil Depletion, farming
- Food allergies & sensitivities
- Vaccines
- Parasites
- Tick-borne Illnesses
- Mould
- Heavy metals: esp. aluminium
- Dyes, preservatives, food additives, food colourings
- Electromagnetic Radiation (EMR)
- Family Emotional Distress
- Dental Infections

Lawrence B Palevsky, MD

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Low Iron Levels



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Iron in ADHD

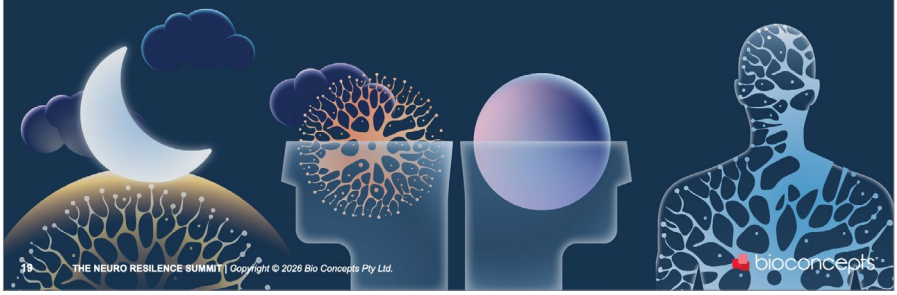
Iron plays a key role in dopamine metabolism

- Iron decreased Connor's ratings in ADHD[1]
- ADHD patients had signs of lower ferritin levels[2]
- 84% of children with ADHD appeared to have abnormally low ferritin levels, compared with 18% of children without ADHD
- Ferritin levels correlate with more hyperactivity in ADHD sample[3]

[1]Sever Y et al. Iron treatment in children with attention deficit hyperactivity disorder. A preliminary report. *Neuropsychobiology* 1997;35(4):178-80.
[2]Konofal E et al. Iron deficiency in children with attention-deficit/hyperactivity disorder. *Arch Pediatr Adolesc Med* 2004 Dec;158(12):1113-5.
[3]Oner O et al. Effects of Zinc and Ferritin Levels on Parent and Teacher Reported Symptom Scores in Attention Deficit Hyperactivity Disorder. *Child Psychiatry Hum Dev* 2010;41(4):441-447.

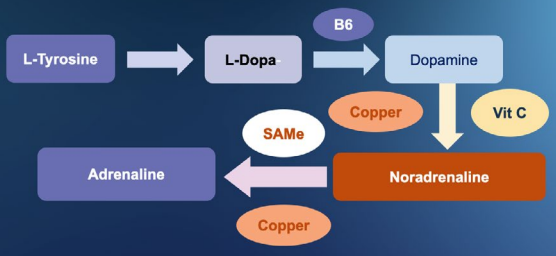
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Copper regulation



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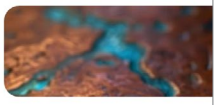
Copper and Neurotransmitter Synthesis



Copper plays an active role in neurotransmitter synthesis and affects the neurotransmitters dopamine, noradrenaline, and adrenaline.

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Copper Toxicity



Physical Symptoms

- Headaches
- Hypoglycaemia
- Increased heart rate
- Nausea/loss of desire to eat
- Copper deposits in the brain and liver
- Damage to the kidneys
- Anaemia
- Hair loss in women
- High copper interferes with zinc

Psychological Symptoms

- Depression
- Hallucinations
- Hyperactivity
- Insomnia
- Paranoia
- Personality changes
- Psychosis; detachment from reality
- Anxiety
- Hypomania

Eck, P., & Wilson, L. (1989). *Copper toxicity*. Phoenix, AZ: Eck Institute of Applied Nutrition and Bioenergetics.
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Excess Copper

- Excess oestrogen
- Birth control pill
- Use of prescription medications containing copper
- Smoking
- Copper in drinking water
- Zinc and manganese deficiency
- Chocolate and avocado (spray)



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Eck, P., & Wilson, L. (1989). *Copper toxicity*. Phoenix, AZ: Eck Institute of Applied Nutrition and Bioenergetics.

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Copper Deficiency

Physical Symptoms

- Not enough oxygen in the cells
- Lowered levels of HDL cholesterol
- Skin problems
- Swollen ankles
- Anaemia

Psychological Symptoms

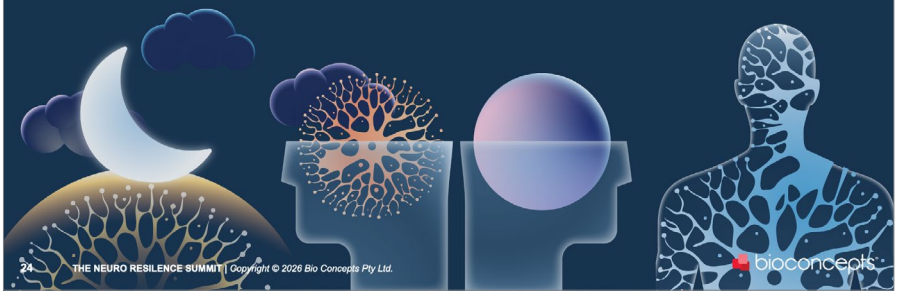
- Auditory hallucinations
- Depression

Note: Phytates hinder absorption. Copper accumulates in liver, kidneys, and brain

Eck, P., & Wilson, L. (1989). *Copper toxicity*. Phoenix, AZ: Eck Institute of Applied Nutrition and Bioenergetics.

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Low Magnesium Levels



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Magnesium in ADHD

One of the first minerals to **disappear from food** when processed and one of the first minerals to **leave the body when there is stress.**

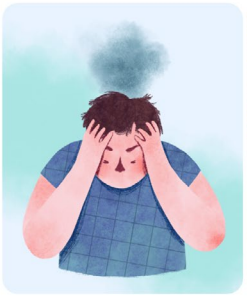


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Depletion of Magnesium

- Medications
- Alcohol
- Caffeine
- Soft drinks
- **Stress!**







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Symptoms of Magnesium Deficiency

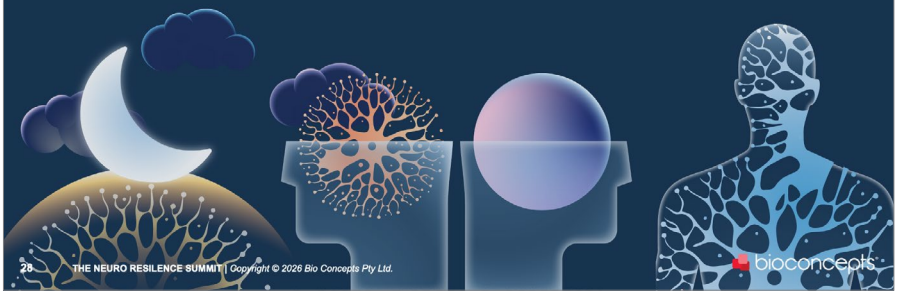
Health conditions associated with magnesium deficiency

- Difficulty with memory and concentration
- Depression
- Apathy
- Emotional lability
- Irritability
- Nervousness
- Anxiety
- Insomnia
- Constipation
- Migraine headaches
- PMS
- Dysmenorrhoea
- Fibromyalgia
- Fatigue
- Autism
- ADHD

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Low Zinc Levels



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Zinc in ADHD

- Zinc levels predict stimulant response[1]
- Serum zinc levels low in ADHD[2]
- Zinc effective as supplement to stimulant[3]
- Zinc effective in reducing hyperactive and impulsive behaviour[4]



Arnold LE et al. Does hair zinc predict amphetamine improvement of ADD/hyperactivity? *Int J Neurosci.* 1990;50(1-2):103-7.
Bekaroglu M et al. Relationships between serum-free fatty acids and zinc and ADHD. *J Child Psychol Psychiatry* 1996;37:225-227.
Akhondzadeh S et al. Zinc sulfate as an adjunct to methylphenidate for the treatment of attention deficit hyperactivity disorder in children: A double blind and randomized trial. *BMC Psychiatry* 2004;4:9.
Bilici M, Yildirim F, Kandil S, et al. Double-blind, placebo-controlled study of zinc sulfate in the treatment of attention deficit hyperactivity disorder. *Prog Neuropsychopharmacol Biol Psychiatry.* 2004;28:181-90.

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Zinc Deficiency



Image: ezracounseling.com

- Children with ADHD had significantly lower zinc levels than control children
- 30% of children with ADHD had severely deficient values
- Possible that low zinc values contribute to depressed production of melatonin and serotonin in the brain

Toren P. et al. Zinc deficiency in attention deficit hyperactivity disorder. *Biological Psychiatry* 1996: 40:1308-1310.

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Zinc and Plastics Chemicals



- Environmental toxins can heighten vulnerability to zinc deficiency
- **Bisphenol A (BPA) and phthalates** can bind to zinc and disrupt zinc pathways
- BPA and phthalate exposure is widespread
- Infants and children may be the most vulnerable to their effects

National Institute of Environmental Health Sciences - www.niehs.nih.gov/health/topics/agents/sya-bpa

Image: chemicalbull.com

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Zinc in the Nervous System

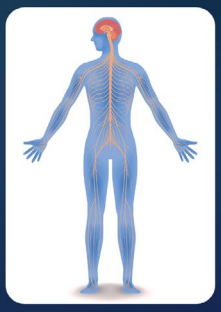


Image: qbi.uq.edu.au

- Maintains protein structure
- Promotes enzymatic activity
- Maintains neurotransmitter activity
- Structural function in the hippocampus

Prasad, A.S. (1995). Zinc: An overview. *Nutrition*, 11(1 Suppl), 93–99.

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Vitamin B6 Reverses ADHD

Research indicates that vitamin B6 at doses between 15–30 mg/kg body weight was as effective as Ritalin in treating attention deficit hyperactivity

Coleman M. et al. A preliminary study of the effect of pyridoxine administration in a subgroup of hyperkinetic children: a double-blind crossover comparison with methylphenidate. *Biological Psychiatry* 1979;14:741–751.

Vitamin B6

Food sources of vitamin B6 (pyridoxine) include beans, legumes, nuts, eggs, meats, fish, breads and cereals

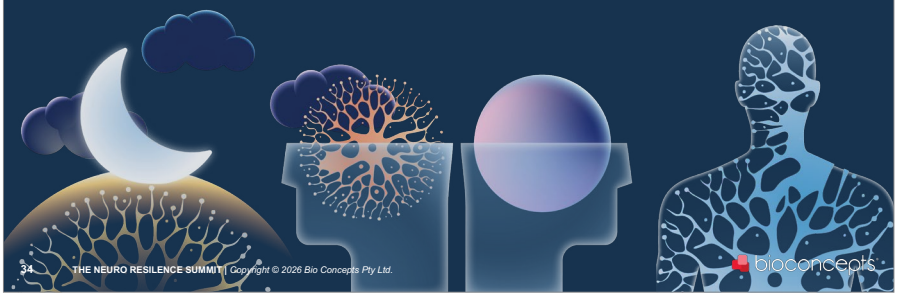
Ritalin® 10 mg Tablets

Each tablet contains 10 mg methylphenidate (as hydrochloride)

Contains lactose, maize starch, sucrose, acacia, talc and povidone

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Lead Toxicity



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Lead and ADHD

- 2,195 children aged 7–9 who did not have ADHD at baseline were followed for 2 years
- 5% (107 cases) developed suspected ADHD
- The risk of ADHD symptoms increased with increasing blood lead levels even in quite low concentrations

- 578 Mexican children aged 6–13 from the Early Life Exposure in Mexico to Environmental Toxicants (ELEMENT) birth cohort study
- Mothers completed Conners' Rating Scales-Revised
- Blood lead level ≤ 5 µg/dL was positively associated with hyperactive/impulsive behaviours

Children with blood lead levels in the highest quartile (>2.17 µg/dL) were **155% more likely to be diagnosed with ADHD**



Choi. *Psychiatry Res.* 2016 Feb 28;236:42–6.

Huang. *Environ Health Perspect.* 2016 Jun;124(6):868–74.

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Very low lead exposures and children's neurodevelopment

David C. Bellinger Children's Hospital Boston, Harvard Medical School, Harvard School of Public Health, Boston, Massachusetts, USA

Summary

No level of lead exposure appears to be 'safe' and even the current 'low' levels of exposure in children are associated with neurodevelopmental deficits. Primary prevention of exposure provides the best hope of mitigating the impact of this preventable disease.

Boston Children's Hospital
Until every child is well®

Bellinger. *Curr Opin Pediatr.* 2008 Apr;20(2):172–7.

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Can Childhood Lead Exposure Affect Cognition in Midlife?

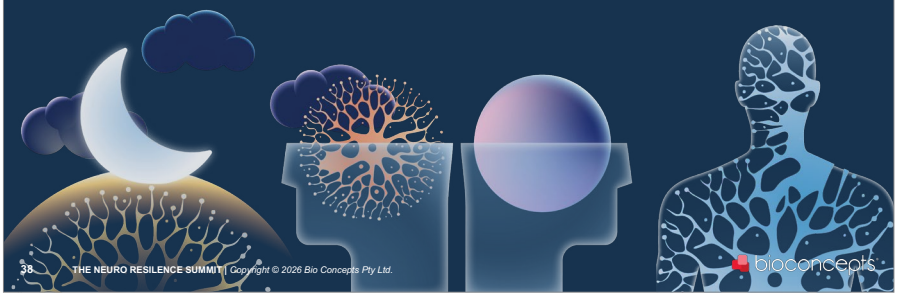
"The study, which prospectively observed 565 children from New Zealand for 4 decades, found that those with higher lead exposure in childhood had significantly lower IQ scores and socioeconomic status at age 38 years than those with lower lead levels."

"The total IQ loss in young children attributable to current lead exposures still exceeds that from brain tumours, congenital heart disease, and traumatic brain injury."

Authors: News Author: Sue Hughes; CME Author: Laurie Barclay, MD; Faculty and Disclosures

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Mercury Toxicity



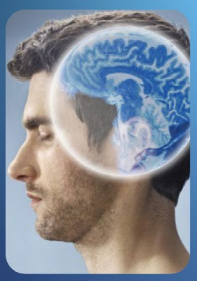
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Environmental Toxins and the Brain

The brain, especially the developing brain, is very vulnerable to contaminants

- Large size (relative to total body weight)
- Very metabolically active – toxin exposure further increases free radical production
- High concentration of fat serves as a reservoir for toxicants to build up
- High amount of sulphur makes it susceptible to binding a variety of toxic metals



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Methylmercury

- Very toxic and bioaccumulative.
  Crosses the blood-brain barrier and placenta.
  Stored in tissues – not readily eliminated.
- Humans are exposed via consumption of contaminated aquatic animals.
- Albacore (white) tuna
- Shark
- Mackerel
- Tilefish
- Swordfish



Jaishankar. *Interdiscip Toxicol.* 2014 Jun;7(2):60–72.

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Prenatal Mercury Exposure

- 421 8 year old children
- Behaviours assessed with Conners' Rating Scale – teachers and neuropsychological testing
- Mercury measured in maternal hair during pregnancy
- At >1 µg/g mercury, risk for inattention ↑ 40% and risk for impulsiveness/hyperactivity ↑ 70% for a 0.5 µg/g mercury increase



Prenatal mercury levels consistently associated with ADHD symptoms

Sagiv. *Arch Pediatr Adolesc Med.* 2012 Dec;166(12):1123–31.

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Childhood Fish Consumption

- 1,179 adults (aged 23–41 years) were asked about fish consumption in childhood (ages 7–12) and learning and behavioural problems
- They were also 2.1x more likely to have had difficulty paying attention or sitting still at school

Carwile. *Int J Environ Res Public Health*. 2016 Nov;13(11):1069.



Those who ate fish several times a week as children had a 5.2x higher odds of ADHD compared to non-fish consumers

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Heavy Metal Testing – Hair



- Toxic elements may be 200–300x more concentrated in hair than in blood or urine
- Hair mercury is an excellent indicator of total body mercury and of brain mercury exposure
- High levels in hair often appear before overt symptoms – can be a preliminary tool for predicting the development of physiological abnormalities
- Should be used in conjunction with symptoms and other laboratory tests

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Heavy Metal Testing – Hair

HAIR ELEMENTS

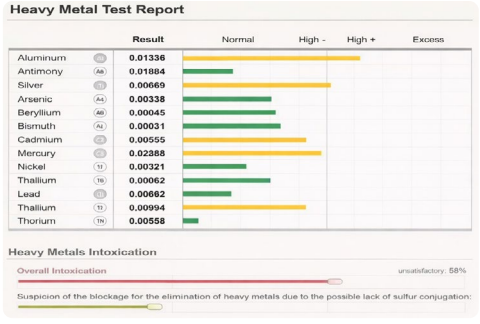
POTENTIALLY TOXIC ELEMENTS

TOXIC ELEMENTS	RESULT µg/g	REFERENCE RANGE	PERCENTILE 68th	PERCENTILE 95th
Aluminum	40	< 8.0		
Antimony	0.14	< 0.0 . (0.080		
Arsenic	0.067	< 0.080		
Beryllium	< 0.01	< 0.020		
Bismuth	0.034	< 2.0		
Cadmium	0.64	< 0.15		
Lead	1.9	< 0.40		
Mercury	< 0.93	< 0.93		
Platinum	0.003	< 0.005		
Thallium	0.005	< 0.005		
Thorium	< 0.001	< 0.005		
Uranium	0.011	< 0.060		
Nickel	0.66	< 0.40		
Silver	68	< 0.20		
Tin	1.0	< 0.30		
Titanium	1.0	< 1.0		
Total Toxic Representation				

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ECM Minerals & Metals



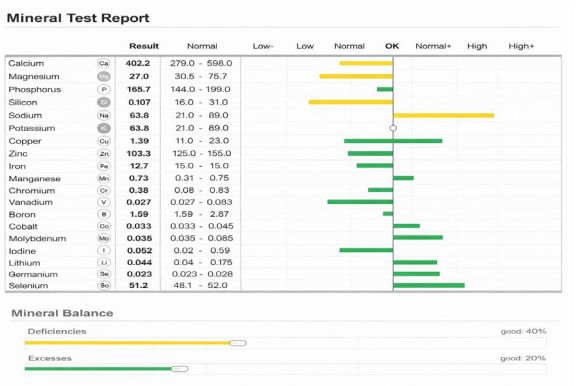


Dr Frank Golik - WIN Health Coach Clinics, Stafford, Brisbane.

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ECM Minerals & Metals

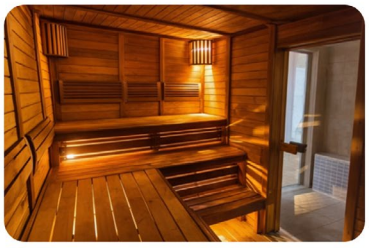


Dr Frank Golik - WIN Health Coach Clinics, Stafford, Brisbane.

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Detox Methods – Infrared Saunas

- The body removes toxic substances, including heavy metals, via sweat
- Infrared's penetrating heat stimulates metabolic activity, which triggers the release of stored toxins through sweat
- 160–180° for 15–30 minutes



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Chelation of Heavy Metals

1. **Counsel** the patient and/or parents for patience until preliminary requirements are correctly set
2. **Educate** the patients/parents about sources of these heavy metals and adopting avoidance and risk reduction
3. Ensuring that **the gut wall and blood-brain barriers** are sealed if possible
4. **Ensuring that the diet is corrected** by avoiding gluten and gluten-mimics such as maize, most grains, and pulses (lectins); + all sugars (esp. fructose). Dairy and soy are best avoided. All animal protein should be organic and wild/non-GMO. **Avoid glyphosate** when it is suspected. Soy, corn, phytates, lectins, and oxalates block absorption of minerals: zinc, magnesium, calcium, and iron
5. **Correct microbiomes** where possible. Be aware of biofilms.
6. Aim to keep **plasma zinc** well above 20-25 range; and serum copper below 14
7. Keep **vitamin D3** in 120-150 range; plus, vitamin K2 and sodium ascorbate
8. **Liver support** is vitally important

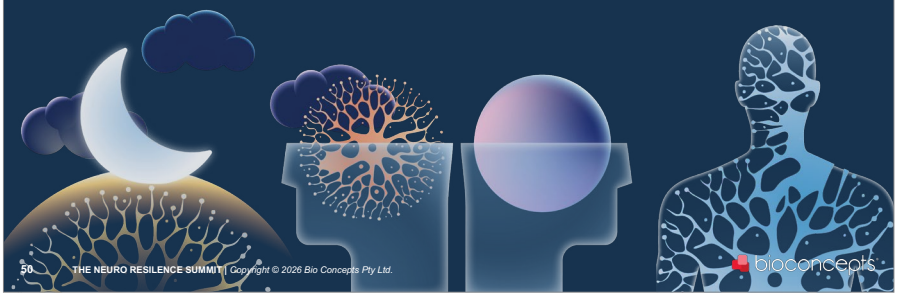
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Chelation of Heavy Metals

1. **Muscle-test everything repeatedly**. Used by **"start low and go slow"**. Warn about possible adverse reactions and how to manage them
2. **Aluminium** is, most times, the leading heavy metal. We can consider zinc, malic acid, and silica. Diatomaceous earth is rich in silica. Best at night
3. **Lead** is best extracted by IV disodium EDTA in glucose/saline; suppositories may have some action. NB that ALL chelation must be executed by fully trained medical doctors in their use
4. **Mercury, arsenic, and cadmium** are best extracted by IV DMPS. This no longer approved by the TGA. Weaker options are binders, homeopathic treatments, near-infrared saunas, and modified citrus pectin. Certain frequency devices can be helpful
5. **Mercury** is readily reabsorbed by the gut. Clean chlorella is important to take orally BD prior to taking oral chelator medication

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Pesticides, Plastics etc



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Pesticides/Insecticides

Insecticides destroy acetylcholinesterase thus reducing ACh.

In one study of 307 kids aged 4-9yrs, lower acetylcholinesterase activity was linked with a **4x greater risk of poor attention and executive function** and a **6x greater risk of memory and learning problems.**

Widespread insecticide exposure affects children. Studies link early life exposure to organophosphates with reductions in IQ and abnormal behaviour associated with ADHD and Autism.

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Suarez-Lopey. *Pediatrics*. 2013 Dec, 132(6): e1649-58
Roberts. *Pediatrics*. 2012 Dec; 130(6):e 1765-88

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Pesticides/Insecticides

- **Study:** 687 US children aged 8-15 yrs. Urinary levels of the pyrethroid metabolite 3-phenoxybenzoic acid (3-PBA) measured. Detectable urinary 3-PBA levels were more than 2x as likely to have ADHD compared with those who had non-detectable levels.
- Animal studies have suggested a strong link between pyrethroid exposure and hyperactivity, impulsivity and disruption of the system regulating the neurotransmitter dopamine, and human studies are starting to confirm those results. One study, published in the journal ***Environmental Health*** **in 2015**, found that children and young teenagers with higher levels of pyrethroid metabolites in their urine were significantly more likely to exhibit hyperactivity and impulsivity and to have an ADHD diagnosis. The effect was particularly strong in boys.
- Each 10-fold increase in urinary 3-PBA level was associated with 57% increase in the prevalence of ADHD.

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Wagner-Schuman. *Environ Health*. 2015 May 28: 14-44

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Pesticides: Fipronil & Pyrethroids

- Synthetic pyrethroids and fipronil are widely found in irrigation and rain-induced runoff and in urban streams (multiple studies, 2005–2013)
- Levels in urban sediment and water can exceed acute toxicity thresholds for sensitive species such as *Hyallela azteca* (Holmes et al., 2008)
- Pyrethroid exposure is linked to cognitive developmental disabilities in children

Distribution of pesticides in dust particles in urban environments. J. Richards, R. Reif, Y. Luo, J. Gan. ***Environmental Pollution***. Vol. 214, July 2016: 290-298
Viel et al. The PELAGIE mother–child cohort. Environment International, 2015; 82: 69.

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Prenatal BPA and ADHD

Prenatal BPA exposure is associated with behaviour problems, particularly in boys

- **Harley (2013):** 292 mother–child pairs — prenatal BPA linked to internalising problems (anxiety, depression) in boys; childhood BPA linked to conduct problems in girls and to inattention/hyperactivity in both sexes
- **Evans (2014):** BPA measured in urine at 27 weeks of pregnancy — higher prenatal BPA related to worse internalising and externalising behaviours (withdrawn/depressed, somatic, oppositional/defiant) in boys aged 6–10
    - **Casas (2015):** 438 mother–infant pairs in a Spanish cohort, urine sampled in the 1st and 3rd trimester — at age 4, BPA exposure associated with increased hyperactivity and inattention in boys

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Harley. *Environ Res.* 2013;126:43–50.
Evans. *Neurotoxicology.* 2014;45:91–9.
Casas. *Environ Res.* 2015;142:671–9.

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BPA and ADHD

A 2016 nationwide study of 460 children aged 8–15 found kids with higher urinary levels of BPA had **over 5x higher odds of being diagnosed with ADHD**



Tewar. *Environ Res.* 2016 Jun 6;150:112–118.

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Phthalates



Phthalates are **synthetic chemicals used to make plastics soft and flexible**

- Used in hundreds of consumer products such as tablecloths, floor tiles, shower curtains, garden hoses, swimming pool liners, raincoats, shoes, and car upholstery
- Humans are exposed to them daily through air, water, and food

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Phthalates

- Disrupt hormones
- Damage brain cells
- Affect fertility
- Potentially cause developmental disorders

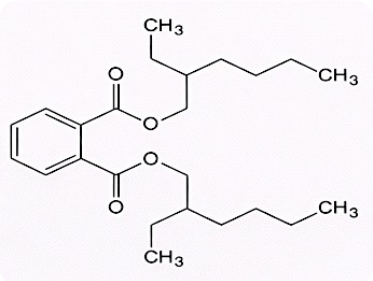


Verstraete. *Intensive Care Med.* 2016 Mar;42(3):379–92.

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DEHP

- Phthalates are banned in cosmetics in Europe but not in the US
- Di(2-ethylhexyl)phthalate (DEHP) is one of the most commonly used phthalates
- It has been banned from children's toys in Australia but is still used to soften medical device plastic



Verstraete. *Intensive Care Med.* 2016 Mar;42(3):379–92.

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Phthalates and ADHD

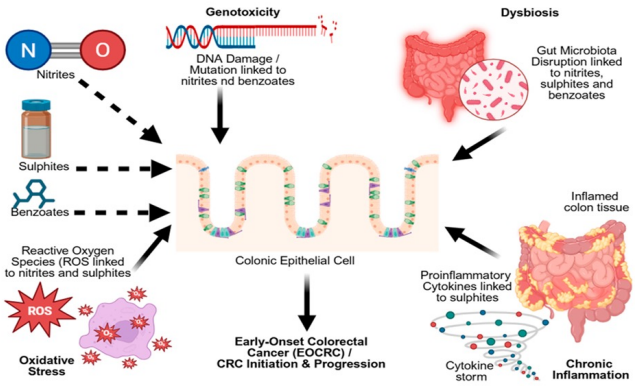
Phthalate exposure explained half of the attention deficit in post-PICU patients

- In a 2015 study, plasma DEHP metabolite concentrations were measured in 100 healthy children and 449 children treated in a PICU at age 0–16
- Upon PICU admission, children had **118–152x higher** DEHP plasma metabolites than healthy children
- Neurocognitive function was tested 4 years after PICU admission
- Exceeding the DEHP metabolite threshold was independently and highly significantly associated with attention deficit and impaired motor coordination



Verstraete. *Intensive Care Med.* 2016 Mar;42(3):379–92.

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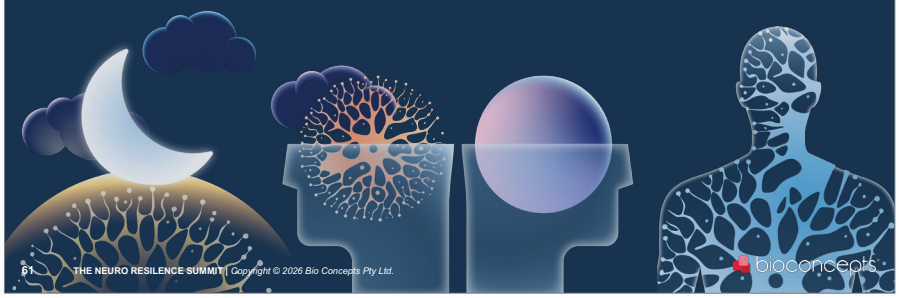


Food preservatives and the rising tide of early onset colorectal cancers

Foods 2025, 14(17), 3079. Mafe A, Busselberg, D

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Problematic Foods & Additives



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Top 8 Food Pitfalls



1. **Gluten/Casein:** form Gliadorphin and Casomorphin; **+ Soy**
2. **ALL Sugars** (esp. Fructose) and **Yeast**
3. **ALL Grains:** can mimic gluten (esp. corn); **and starches**
4. **Phenols/salicylates/amines/glutamates**
5. **Oxalates, lectins, and phytates**
6. **Histamine** (and -liberating) **foods; DAO-inhibiting foods**
7. **Latex foods** and **food allergies**
8. **Lack of good nutrition**

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Ultra-processed food consumption patterns among children with ADHD

Aim	Study	Findings	Conclusion
Assess whether ADHD in children is associated with ultra-processed food (UPF) intake	1,135 Israeli children aged 6–11 (111 with physician-diagnosed ADHD); UPF intake from 24-hour recall, classified by the NOVA system (2015–2016 national survey)	ADHD was linked to above-median UPF intake — by grams/day (OR 1.62, 95% CI 1.01–2.60) and by % of food weight (OR 1.65, 95% CI 1.06–2.58); no difference by stimulant treatment	ADHD is associated with higher UPF consumption, with potential in either direction

Namimi-Halevi et al. *Pediatric Research*. March 2026; DOI: 10.1038/s41390-026-04844-5

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Common Food Sensitives

Either Elimination or Rotation Diet



- Gluten
- Casein
- Soy
- Corn
- Eggs
- Citrus
- Peanuts and tree nuts (1. Cashew, 2. Macadamia)

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Digestive Enzymes

- Diminished brunch border enzymes in many children and adults (ASD etc)
- DPPIV – Breaks down gluten and casein opioid activity
- DPPIV – is damaged by organophosphates and certain heavy metals like mercury
- DPPIV: Dipeptidyl dipeptidase

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Opioid Peptides can cause…

- Addictions (foods and screens)
- Hallucinations
- High pain tolerance
- Inattention and spacey behaviour
- Aggression (self and others)
- Stimming
- Mood changes
- Poor eye contact

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Histamine Degradation Genes and Food Additive Effects on Children's ADHD

- **Food additives can worsen ADHD symptoms and trigger non-IgE histamine** release from circulating basophils
- **Children differ in how much food additives aggravate** their ADHD symptoms
- Histamine may mediate these effects, and variation in histamine-related genes may explain inconsistent findings across earlier studies

Stevenson J *et al. Am J Psychiatry.* 2010 Sep;167(9):1108–15.

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Scientific Rational for Diets

Research on gluten and casein for AUTISM

OPIOIDS

- Shimizu T, Yoshikawa M. (1991) **Enzymatic release of neocasomorphin and beta-casomorphin from bovine beta-casein. *Peptides.*** 20:957–962.
- Jinsook K., Krishnan KM., Laud C., Holland M. **Possible etiology and possible treatment of childhood autism. *Brain Dysfunction.*** 1998;17:70–91.
- Kamienski B, Kamienska A, Krotkyp P. (2007) 5(4):189–198.
- Shattock P, Whiteley P. (2002) Unidentified aspects in autism spectrum disorders, updating the opioid-excess theory and presentation for consideration in biomedical intervention. *Parent Train Ther*

DIGESTIVE PROBLEMS WITH GLUTEN & CASEIN

- Jyonouchi H, Geng L, Ruby A, Caden R, Zimmermen-Bier B. (2005) **Evaluation of an association between nutrient intake and peculiar urinary opioid excretion dietary profiles of children with autism spectrum disorder.** *J Pediatr.* 146(4):475–481.

RESEARCH ON FOOD SENSITIVITIES FOR ADHD

- Resm NM. **Diet and hyperactivity. Mandatory Intention Disorder Diet.** *Nutr Rev.* 2008 Oct;66(10):558–561.

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Scientific Rational for Diets

Research on gluten and casein for AUTISM

REDUCED AUTISTIC SYMPTOMS

- Knivsberg AM, Reichelt KL, Nodland M. (2001) **Reports on dietary intervention in autistic disorders. *Nutritional Neurosciences.*** 4:25–35
- Whiteley P, Haracopos D, Knivsberg AM, Reichelt KL, Hoien T, Nordvaad IL (2001) **A controlled, randomized study of dietary intervention in one hundred and twenty children.** *Pediatr.* 107.
- Knivsberg AM, Reichelt KL, Nordvaad IL, Hoien T. (2002) **Autistic syndromes and diet: A follow-up study.** Scand *Pediatr Suppl.* 1:28–37. *Int Rev. 2008* Oct;56(10):558–561.

RESEARCH ON FOOD SENSITIVITIES FOR ASTHMA

- McDonald KL, Miesiwki MJ, Graham P, Hodge Nr., Smith MW. (2000) **A diet and are closely linked with childhood asthma.** *Oir-Exp Allergy* 30:359.
- Ilicpoulos A., Konsiantinou L., et al. **Food allergens related closely with asthma in children.** *J Pediatr Pharmacol.* 2000: 98, Suppl. 9/69–83R.

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Gluten Grains & Ingredients to Avoid

GRAINS



- Wheat
- Rye
- Barley
- Spelt
- Kamut
- Triticale
- Oats (contaminated)
- Semolina

HIDDEN SOURCES

- Hydrolysed Vegetable Proteins
- MSG
- Dextrin
- Malt
- Citric acid
- Artificial flavours/colours/ spices
- Soy sauce
- Potato chips/fries

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Casein Containing Foods to Avoid

DAIRY

- Milk
- Cheese: all
- Butter
- Buttermilk
- Yoghurt (dairy)
- Ice cream
- Kefir
- Cream/sour cream



HIDDEN SOURCES

- Whey, casein
- Galactose
- Lactose, lactalbumin
- Lactic acid
- Sherbet
- Canned tuna
- Artificial butter flavour

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Avoid Soy



- Not a good substitute for dairy or protein
- Very difficult to digest
- Irritates the gastrointestinal tract
- **Blocks absorption** - calcium, magnesium, iron, copper and especially zinc – due to phytic acid and oxalates
- Blocks thyroid function
- Endocrine disruption in the reproductive hormones of both males and females

SOY SOURCES

- Tofu
- Soy protein
- Miso
- Tempeh
- Soy milk
- Soy cheese or ice cream
- Soy sauce
- Tamari
- Soy oil

HIDDEN SOY

- Lecithin
- Vitamin E

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Avoid Corn



- Top 5 allergens
- Contains high level of fungi
- Herbicide Atrazine delays puberty in boys, affecting endocrine function, fertility, and thyroid function
- Genetically modified corn - environmental experiment
- Round-Up-ready. Genes from bacterial toxin added - BT toxin

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Sugar evils

- **Feeds yeast, bacterial, viral, and parasitic over-growth**
- **Depresses the immune system**
- Contributes to **inflammation**
- **Cane sugar:** common food sensitivity. **It uses chemical processes**
- **Refined sugar = bleached sugar cane: devoid of nutrients**
- **Raw sugar = molasses added back to white sugar**
- **Fructose can promote inflammation, elevated urate, fatty liver, pre-diabetes, and insulin resistance.** In agave, maple syrup, honey
- **Better choice: Monk fruit (100%) or Stevia**

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Grains

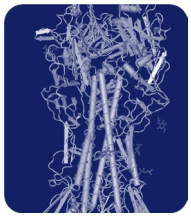
- Inflammatory
- Grains contain: **phytates, oxalates, and lectins**
- Refined grains in the form of flours impact blood glucose
- Inflammatory compounds that inhibit the absorption of minerals: **calcium, magnesium, zinc, and iron**
- Digestive discomfort
- Promote dysbiosis



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Lectins

- Found in: **grains, beans, soy, peanuts, almonds, pasteurised dairy** (raw milk contains sIgA to bind to lectins), **nightshades** (tomatoes, eggplant, capsicum, chilli, paprika, potatoes)
- **Proteins that do not break down easily. Resistant to stomach acid and digestive enzymes**
- Lectins **agglutinate red blood cells**
- Lectins **may bind to the gut wall and damage the gut lining.** If gut permeability is altered, other food proteins can pass through the gut into general circulation, they can create allergic/autoimmune responses. Lectins in circulation can bind to organs and cause damage.
- SCD/GAPS and Paleo/Primal diets are essentially reduced lectin diets



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Phenols/salicylates

Phenol/aromatic Ring Bound to –OH group

SYMPTOMS

- Red cheeks and ears
- Hyperactivity
- Sleep disturbances
- Aggression
- Irritability
- Diarrhoea
- Headache
- Head-bagging/self-injury
- Inappropriate laughter
- Hives/rashes
- Some forms of seizures

SOURCES

Artificial Phenols:

- Artificial colours, flavours
- Preservatives, additives
- Propionic acid



Salicylates:

- Red grapes, apples, berries, citrus, cucumbers, capsicums
- Aspirin

Diets: Feingold or Failsafe

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Food Additives

Unhealthy Ingredients to Avoid

Food additives can cause:
Hyperactivity*,
Inattentiveness,
Aggression,
Irritability,
Headaches/Pain,
Trigger asthma,
Can be addictive

Ingredients to Avoid	Sources
Artificial colors/flavors and preservatives	candy, cereal, "kids" foods
MSG (hydrolyzed protein, yeast extracts)	broth, bullion, soups, meat-flavored foods
Pesticides	non-organic produce and meat
Aspartame and other artificial sweeteners	sodas and other foods
High fructose corn syrup	sodas, jelly, candy
Trans fats	partially hydrogenated oil, commercial margarine, mayonnaise, peanut butter
Sodium nitrite	bacon, hotdogs, lunch meat

McCann D., Barrett A., Cooper A., Crumpline D., Dalen L., Grimshaw K., Kitchin P., Lok K., Plumstead L., Sonuga-Barke E., Warner JO, Stevenson J. "Food additives and hyperactive behaviour in 3-year-old and 8/9-year-old children in the community: a randomized, double-blinded, placebo-controlled trial." *Lancet.* 2007 Nov 22;370(9598):1560-7.

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Feingold Diet - Salicylates

- Almonds
- Apples
- Apricots
- Berries, raspberries, cherries
- Chili powder
- Cider and cider vinegar
- Cloves
- Coffee
- Cola drinks
- Cucumbers and pickles
- Curry powder
- Endive
- Grapes, raisins, currants
- Honey
- Nectarines and peaches
- Oranges and citrus
- Paprika
- Peppers (bell and chili)
- Pineapple
- Plums and prunes
- Radishes
- Tea
- Tomatoes
- Wine and wine vinegar
- Oil of wintergreen





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Failsafe/RPAH Elimination Diet

Low Salicylate, Amine and Glutamate

Additional salicylates to avoid

- Avocado
- Zucchini
- Broccoli
- Spinach
- Cantaloupe/rockmelon
- Watermelon
- Dates
- Cinnamon, thyme, oregano, rosemary, and most herbs/spices
- Watercress
- Artichoke
- Eggplant
- Peanuts




Diet: Failsafe

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Amines

Biogenic amines/derivatives of ammonia

SYMPTOMS

- Can be same as salicylates
- Often come on more slowly and last longer
- Aggressive/defiant
- Eczema
- Migraines
- Depression
- "Hungover"
- Irritable bowel symptoms

SOURCES



- Banana
- Cheese, yellow
- Aged or blue cheese
- Chocolate/cocoa
- Wine/beer
- Fermented foods: sauerkraut, yoghurt, tempeh
- Soy sauce
- Bone broths
- Meat and aged meat/fish

Diet: Failsafe

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Glutamates

Salts of glutamic acid/natural and MSG

SYMPTOMS

- Hyperactivity
- Headaches
- Hunger regulation imbalances, obesity
- Asthma symptoms
- Anxiety
- GI Complaints

SOURCES



- MSG
- Autolysed yeast
- Soy sauce
- Parmesan cheese
- Vegemite/Marmite
- Sauerkraut
- Bone broths
- Gelatin
- Peas
- Corn
- Tomatoes
- Grape juice

Diet: Failsafe

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Oxalates

Low Oxalate diet = 40–60 mg/day (exogenous)

Extremely High

Beans, Carob, Cocoa/Chocolate, Many fruits, Amaranth, Figs, Soy, Buckwheat, Quinoa, Teff, Nuts (almonds, pecans), Seeds, Spelt Potatoes, Spinach, Sweet potato, Yams, Beets, Eggplant, Kiwi fruit, Artichokes, Ginger, Turmeric, Curry, Olives, Tea, Cinnamon, Clove, Cumin, Allspice, Onion powder, Grain brans, Gluten-free flour, Peanuts, Swiss chard, Blackberries, Raspberries, Fennel, Rhubarb

High

Corn, Rice, Tapioca, citrus zest, soybeans, wheat, starfruit, tahini, yucca root

Low

Coconut, green bean, green pea, lupin, chickpea, yellow pea, black-eyed pea, green bean flours, sorghum, lemon, lime, blueberries, lettuce, kale, mustard greens, cauliflower, avocado

Zero

Cocoa butter, almond extract, cinnamon oil/extract



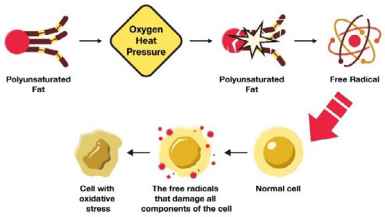
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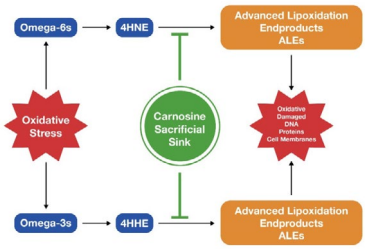
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Linoleic Acid

Increased intake in the Standard American Diet and its associations with chronic disease

Excess LA damages cellular tissue because this fragile PUFA is packed with double bonds that are easily altered by oxidation.





Carnosine is an endogenously produced dipeptide (beta-alanine + histidine). **This potent antioxidant limits excess-LA damage by binding to ALEs.**

Mercola J, D'Adamo CR. Nutrients 2023;15(14):3129.

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Microbiome and ADHD

- **Candida:** sleep problems, hyperactive, silly, giggly, brain-fog, drunk
- **Threadworms, other parasites:** sleep problems
- **Constipation:** sleep problems
- **Diarrhoea:** poor absorption of nutrients
- **Best way to improve microbiome:** consume variety of different vegetables, minimal fruit, nuts, seeds: they are prebiotics

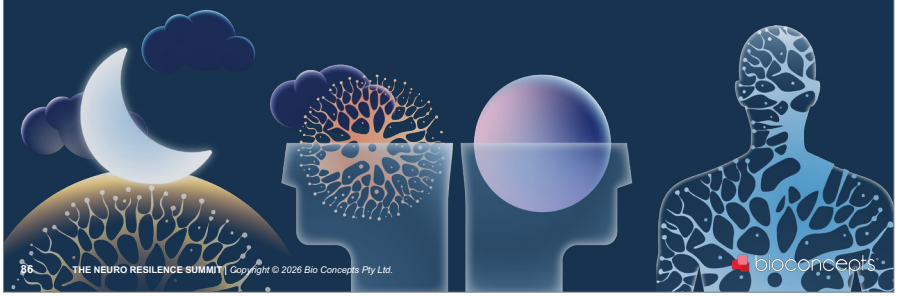


(Checa-Ros, 2021; Góralczyk-Bińkowska, 2022)

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Relevant Testing Workup



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Components of Assessment

Full clinical and psychosocial assessment

- Developmental History
- Mental Health History
- Medical History
- Mental State Examination
- Observer reports
- Screening tools
- Physical Examination



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Table 3: Categorising SDQ scores for the 4-17 year old version of the SDQ (not validated for 18+)

	Original 3-band categorisation			Newer 4-band categorisation			
	Normal	Borderline	Abnormal	Close to Average	Slightly Raised (/Slightly Lowered)	High (/Low)	Very High (/Very Low)
Parent completed SDQ							
Total difficulties score	0-13	14-16	17-40	0-13	14-16	17-19	20-40
Emotional difficulties score	0-3	4	5-10	0-3	4	5-6	7-10
Conduct difficulties score	0-2	3	4-10	0-2	3	4-5	6-10
Hyperactivity score	0-5	6	7-10	0-5	6-7	8	9-10
Peer difficulties score	0-2	3	4-10	0-2	3	4	5-10
Prosocial score	6-10	5	0-4	8-10	7	6	0-5
Impact score	0	1	2-10	0	1	2	3-10
Internalising difficulties score	-	-	-	0-5	6-7	8-9	10-20
Externalising difficulties score	-	-	-	0-8	9-10	11-12	13-20
Teacher completed SDQ							
Total difficulties score	0-11	12-15	16-40	0-11	12-15	16-18	19-40
Emotional difficulties score	0-4	5	6-10	0-3	4	5	6-10
Conduct difficulties score	0-2	3	4-10	0-2	3	4	5-10
Hyperactivity score	0-5	6	7-10	0-5	6-7	8	9-10
Peer difficulties score	0-3	4	5-10	0-2	3-4	5	6-10
Prosocial score	6-10	5	0-4	6-10	5	4	0-3
Impact score	0	1	2-6	0	1	2	3-6
Internalising difficulties score	-	-	-	0-5	6-7	8-9	10-20
Externalising difficulties score	-	-	-	0-6	7-9	10-12	13-20
Self-completed SDQ							
Total difficulties score	0-15	16-19	20-40	0-14	15-17	18-19	20-40
Emotional difficulties score	0-5	6	7-10	0-4	5	6	7-10
Conduct difficulties score	0-3	4	5-10	0-3	4	5	6-10
Hyperactivity score	0-5	6	7-10	0-5	6	7	8-10
Peer difficulties score	0-3	4-5	6-10	0-2	3	4	5-10
Prosocial score	6-10	5	0-4	7-10	6	5	0-4
Impact score	0	1	2-10	0	1	2	3-10
Internalising difficulties score	-	-	-	0-6	7-8	9-10	11-20
Externalising difficulties score	-	-	-	0-8	9-10	11-12	13-20

Note that both these systems only provide a rough-and-ready way of screening for disorders; combining information from SDQ symptom and impact scores from multiple informants is better, but still far from perfect.

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Screening Tools

- Children – Vanderbilt (6–12yr)
- Adolescents – SNAP IV (6–18yr)
- Adults – ASRS or ASRS-5 (Adult ADHD Self Report Scale)
- Psychologists – psychometric testing – WISC and Connors $$



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Baseline Assessment

- **Height and weight**
- **Cardiovascular Assessment** – HR, Blood Pressure (consider CV History)
- **Current sleep & apnoea**
- **Nutrition and appetite**
- **Blood tests:** FBC, hsCRP, E/LFTs, Iron studies, B12/ folate. Homocysteine, cortisol, Coeliac serology, plasma zinc/serum copper, lead, Hg, IgE & RAST, Faeces: PCR & Micro, Strep. Auto-Ab's (ASOT, AntiDNase B)



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Genetic factors/SNPs

- CNTNAP2 encodes CASPR2
- STYK1 (Serine/Threonine/Tyrosine Kinase 1)
- PEMT (Phosphatidylethanolamine N-Methyltransferase)
- SLC25A12 (Aralar)
- GABRB3 (GABA-A Receptor Beta 3 Subunit)
- HTR 2A (5-Hydroxytryptamine Receptor 2A)
- MBL 2 (Mannose-Binding Lectim-2): increase PANS/PANDAS
- TNF-α: AA (very rare) in 86% of PANS/PANDAS
- MTHFR
- COMT

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Genetic factors/SNPs

Potential vaccine reactivity:

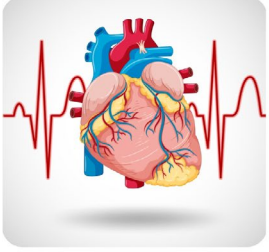
- NLRP3 (NLR Family Pyrin Domain Containing 3): increases inflammasome.
- CARD8 (Caspase Recruitment Domain Family Member 8; if normal, it slows inflammasome.
- CCL2 c-c Motif Chemokine Ligand 2- Monocyte Attractants: macrophages engulf aluminium particles. CCL2 increases AI macrophages and moves aluminium to the brain.
- SHANK3: SH3 and Multiple Ankyrin Repeat Domains 3. It helps brain plasticity.

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Cardiovascular Assessment

- History of congenital heart disease
- Sudden death in 1st degree relative <40
- Exertional dyspnoea
- Exertional syncope
- Palpitations
- Chest pain of possible cardiac origin
- Heart Murmur
- Hypertension



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Australian evidence-based Clinical Practice guidelines for ADHD

- **Diagnosis**
- **Rule out comorbidities**

Anxiety, learning problems, giftedness, hearing or vision impairment, thyroid disease, anemia, and other conditions

- **Treatment**

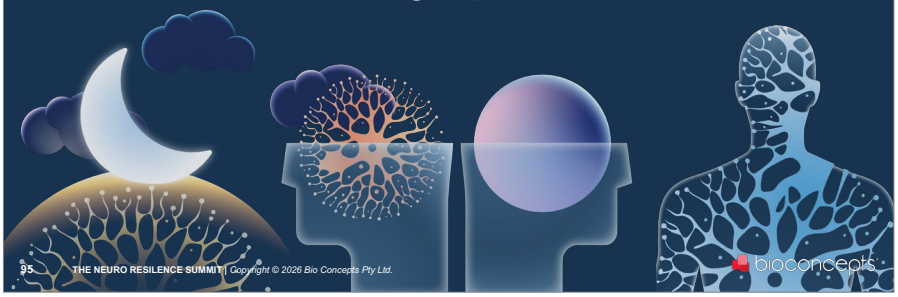
1.Non-pharmacological interventions have value beyond improving ADHD and can improve broader aspects of functioning for individuals and their families: active and healthy lifestyle, sleep…

2. Parent training
3. CBT
4. Support at school/university
5. Medications

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Other conflicting features and symptoms

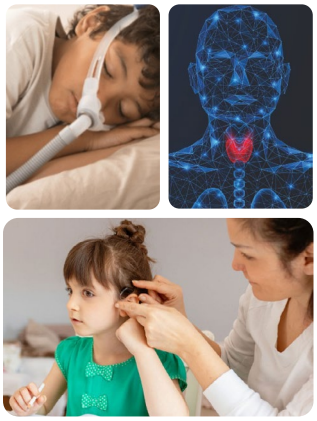


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Differential Diagnoses

- Thyroid disorders, hypothyroidism
- Iron, zinc, magnesium deficiency
- Hearing/vision impairment
- Epilepsy, absent seizures
- Autism Spectrum Disorder
- Anxiety and OCD; PTSD
- Trauma/abuse/family violence
- Sleep apnoea
- Speech and language disorders
- Other developmental delays
- Substance use
- High IQ and infections

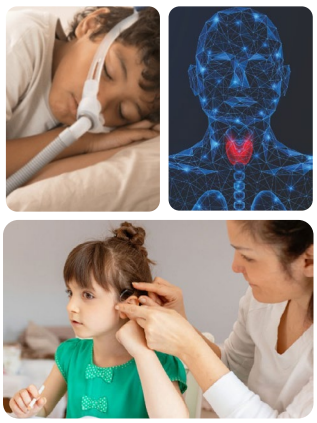


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Differential Diagnoses

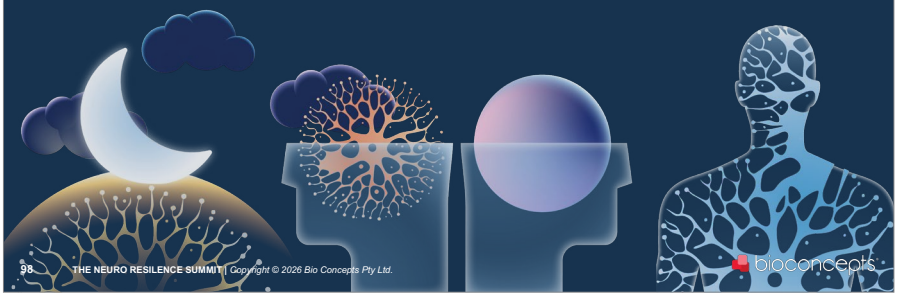
- Concussion and head injuries
- Hyperthyroidism/Grave's disease
- Lead, aluminium, and mercury toxicity
- Dyslexia, dyspraxia
- Coeliac disease and gluten intolerance/DPPIV deficiency
- Mould and EMF exposure: brain fog
- Vitamin D & A and Omega-3
- Mitochondrial dysfunction
- Allergies



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Management, Treatments and Concerns



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Overview: Lifestyle interventions for ADHD

- Sleep
- Exercise > 1-2 hours a day
- Time in nature versus screen time
- Stress reduction
- Nutrition and diet
- Supplements





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Non-Pharmacological Treatment

Lifestyle Changes
(Clinical Practice Point)

Parent/Family training
(Strong recommendation)

Parent/Family Training
(Conditional recommendation; strong recommendation is co-occurring oppositional defiant disorder/conduct disorder)

Cognitive-behavioural interventions
(conditional recommendation)

Cognitive-behavioural interventions
(strong recommendation)

ADHD coaching
(clinical consensus)

YOUNG CHILDREN → CHILDREN → ADOLESCENTS → ADULTS → OLDER ADULTS

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L- Theanine

L-Theanine in the Adjunctive Treatment of Generalised Anxiety Disorder: A Double-Blind, Randomised, Placebo-Controlled Trial

- **Study** 46 adults with DSM-5 GAD; adjunctive L-theanine 450–900 mg/day vs placebo added to stable antidepressants over 8 weeks (10-week protocol with pre/post observation); anxiety, sleep, and cognition assessed
- **Conclusion** Did not support L-theanine for anxiety in GAD, but its effect on sleep disturbance warrants further study

The one positive finding — sleep: greater self-reported sleep satisfaction than placebo (ISI item 4; p = 0.015), with a separation favouring L-theanine in non-clinical insomnia (ISI ≤ 14; p = 0.007)

Sarris J *et al. Journal of Psychiatric Research*, December 2018. DOI: 10.1016/j.jpsychires.2018.12.014

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Sleep Problems and ADHD

Up to 50% of children with ADHD have sleep problems:

- less REM sleep, shorter total sleep
- **Sleep-disordered breathing/apnoea:** 1/3 of children with ADHD (vs 3% of non-ADHD), mouth breathing (Knight, 2019)
- **Sleep onset insomnia** (50% of adolescents with ADHD), sleep maintenance-insomnia
- **Parasomnias, restless leg** (50% vs 2%), periodic limb movement disorder



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Sleep Problems and ADHD

- **Mouth breathing** (Kalaskar, 2021)
- **Insomnia** associated with more severe ADHD symptoms: deficits in sustained attention and processing speed (Li, 2022)
- **In OSA:** tonsillectomy improved ADHD symptoms more than Ritalin (Huang, 2006)
- Iron deficiency affects sleep: difficulty falling asleep, poor sleep quality, restless legs
- **Sleep makes dopamine more available in the brain, modulates attention**

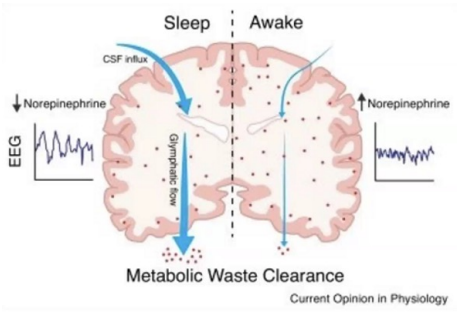


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Glymphatic System:

waves of CSF flush the brain and clear neurotoxic waste products during DEEP SLEEP



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Sleep Hygiene – The Basics

- Plenty of **exercise** during the day – at least 1 hour
- Exposure to **natural daylight** to help melatonin production
- Turn off all screens 2-3 hours before bedtime
- Use **NightShift** (macOS), **NightLight** (Windows) or **f.lux.com** to change screen colour from blue to orange
- **Chamomile tea** after dinner (apigenin binds to GABA receptors)
- Warm bath with **magnesium** SO4 salts + clean Bicarb
- Worry journal: problem-solving
- It is ok for a caregiver to sit or lie down with the child for comfort
- **Melatonin, L-Theanine, oral Magnesium & Zinc**



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Sleep in Children with ADHD

Sleep is dysregulated

- Meta-analysis: ADHD children vs typically developing peers
- Shorter total sleep time and lower sleep efficiency
- Longer sleep latency and higher awakening index
- Disturbances are pervasive — a key target in ADHD management

Xian P et al. Psychological Medicine 2025;55:e321

What helps children fall asleep

- **Behavioural** physical exercise, regulating digital device use, and a balance between activity and rest
- **Environmental** a calming sleeping environment and consistent evening routines
- Children stressed the importance of individualized approaches

Alvstrand M et al. BMC Pediatrics 2025;25:609

Evidence for interventions

- Systematic review of behavioural programs and melatonin
- 6 of 7 outcomes showed small-to-moderate effects (low certainty)
- **Sleep disturbances** moderate benefit from behavioural interventions (SMD −0.49; 95% CI −0.65 to −0.33; moderate certainty) in ages 5–13
- Behavioural and melatonin interventions can improve sleep quantity and quality

Sleep Medicine 2023;102:64–75

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Sleep Aids

- **Melatonin** (Esposito, 2019)
  - Considered safe, but there are no long term safety studies in children
  - Hormone – possible effects on growth?
  - Use smallest dose that works for shortest time needed: start with 1mg
- **Natural melatonin production improved with:**
  - Morning sunlight exposure
  - Dimmed/orange lights or candles in the evening
  - Tryptophan rich foods: turkey, chicken, eggs, nuts, seeds
  - Avoiding caffeine
  - Dark bedroom/eye mask



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The Hidden Dopamine Trap

Modern children's online content is engineered to maximise watch time through rapid cuts and **constant dopamine triggers, creating addictive patterns in toddlers that lead to overstimulation, irritability, and severe tantrums when screens are removed.**

This deliberate design exploits the brain's orienting response, rewiring developing nervous systems toward novelty-seeking while impairing sustained attention, executive function, and emotional regulation: **effects supported by studies linking fast-paced-media to ADHD-like symptoms and long term attentional defects.**

These same dopamine-hijacking tactics have permeated broader society, from addictive processed foods to social media and pornography, shifting us from sustained serotonin-based happiness and presence to **fleeting, hollow spikes that leave people feeling disconnected, depleted, and perpetually unsatisfied.**

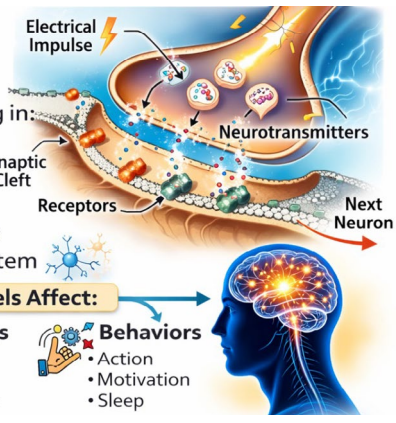
This engineered addiction is especially damaging in romantic relationships, where the cultural push for intense dopamine highs and excitement erodes the capacity for deep, stable, and truly nourishing intimate bonds on genuine connection and contentment.

A healthy, non-depleted nervous system naturally resists the pull of these artificial dopamine spikes, allowing us to appreciate more subtle and authentic sources of joy: **escaping this trap and reclaiming a vivid connection to life.**

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- At least 250 are known
- Release of these chemicals causes electrical impulses throughout the brain resulting in:
  - Thoughts
  - Feelings
  - Behaviors
- Neurotransmitter levels affect ***every facet*** of a biological system



Neurotransmitter Levels Affect:

Thoughts
- Learning
- Memory
- Focus

Feelings
- Mood
- Stress
- Anxiety

Behaviors
- Action
- Motivation
- Sleep

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Neurotherapy in ASD/ADHD

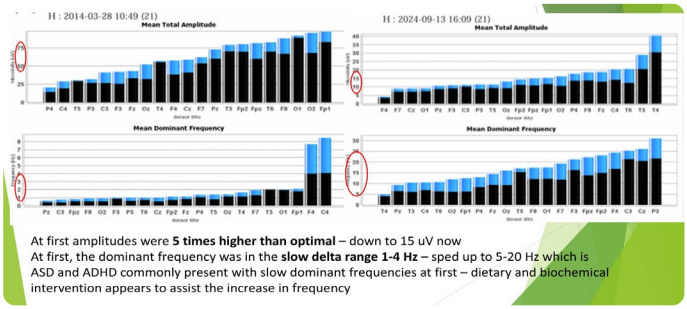
LENS - Low Energy Neurofeedback System

- Non-invasive, passive and medication free
- Sensors on the scalp record brainwaves & send back a personalised feedback signal that mirrors the brain's activity
- Encourages the brain to "reset" patterns to healthier functioning, enhancing neural connectivity
- Helps ASD by reducing rigid behaviour, improving sensory processing, regulating emotions, enhancing communication, attention and focus

Courtesy of Karin Oldham, GROW, The Grange

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Neurotherapy in ASD/ADHD

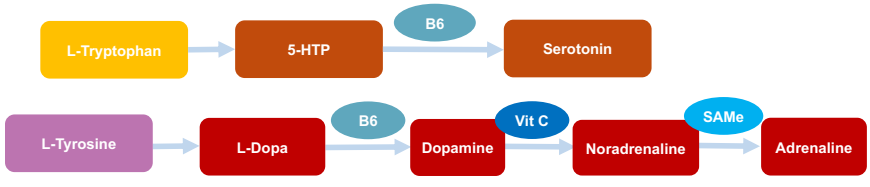


At first amplitudes were **5 times higher than optimal** – down to 15 uV now
At first, the dominant frequency was in the **slow delta range 1-4 Hz** – sped up to 5-20 Hz which is ASD and ADHD commonly present with slow dominant frequencies at first – dietary and biochemical intervention appears to assist the increase in frequency

Courtesy of Karin Oldham, GROW, The Grange

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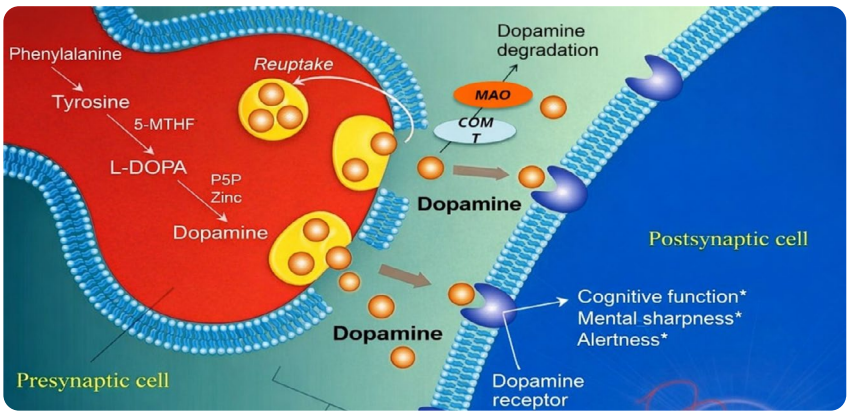
Neurotransmitter Synthesis



- Folate
- Vitamin B6
- Vitamin B12
- Vitamin C
- Vitamin D
- Vitamin B3
- Magnesium
- Zinc
- Iron
- Copper
- Manganese
- SAMe

Courtesy of Karin Oldham, GROW, The Grange

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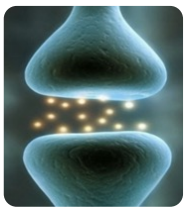
Courtesy of Karin Oldham, GROW, The Grange

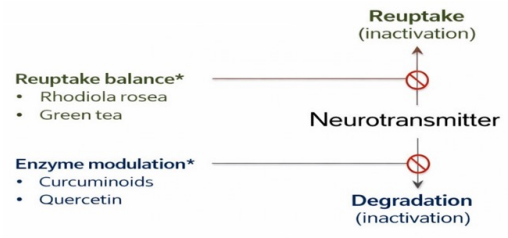
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Phytochemical Modulators

Enzyme modulation and reuptake balance

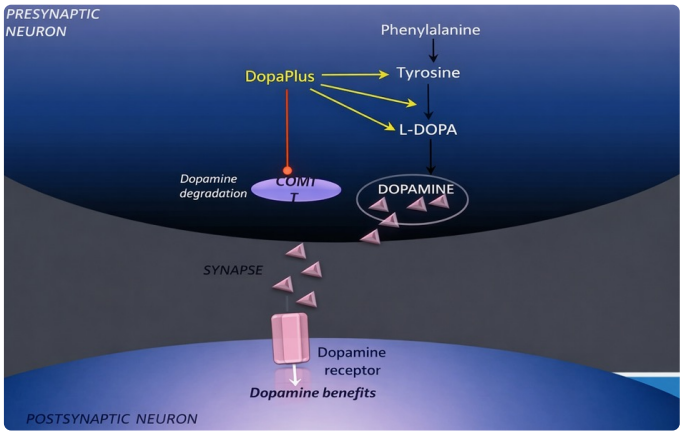
Once neurotransmitters are synthesised, their activity and stability is maintained by specific phytochemicals*





Courtesy of Karin Oldham, GROW, The Grange

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Courtesy of Karin Oldham, GROW, The Grange

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Important Tips

Melatonin
helps mitochondria along with sunlight; decreasing brain aluminium.
Increased by avoiding blue light from devices

Glycine
avoid in patients with oxalates; it stimulates AMP receptor

Methylene blue
helps mitochondria; breaks down biofilms

Lead
often in herbs and chocolate; very high in India, China, & Mexico; there is NO safe level; it kills attention and focus

MB12
methylates dopamine receptors (DRD4)

Glutamate
increases OCD

Aluminium
displaces magnesium

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Lifestyle Interventions for ADHD

Exercise

at least 1 hour/day

- **Moderate-to-intense aerobic exercise** beneficial and well-tolerated for children and adolescents with ADHD (Ng, 2017)
- Improves executive function (Montalva-Valenzuela, 2022; Christiansen, 2019)
- Reduces impulsivity; improves executive function, behaviour, attention (Den Heijer, 2017)
- **Immediate and long-term benefits** with only positive side effects
- Aerobic exercise raises serotonin, dopamine, BDNF and brain blood flow (Chan, 2021)
- Perceptual-motor exercises enhance neuroplasticity and synaptic connections (Chan, 2021)

Green play

Nature deficit disorder

- **Everyday green spaces** may ease ADHD: milder symptoms and better concentration with regular time in green areas; less hyperactivity after open-field play
- Children with attention deficits concentrate better after a walk in the park
  — effect size similar to Ritalin
- Play in green or natural settings reduces ADHD symptoms — the greener the better
- Improved cognitive development in children exposed to green spaces

Vitamin D

- Reduces inflammation
- Improves mood
- **Children with ADHD** are more likely to be vitamin D deficient
- **Deficiency** <50 nmol/L; **insufficiency** <120
- **One of the 3 most effective ADHD supplements** (Zhou, 2024)

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Screen Time and ADHD

Screen time raises ADHD risk

- Screen time before 3 years of age increases the risk of ADHD (Liu, 2023; Qu, 2023)
- AAP recommendation: no screens for <2 years
- <60 minutes per day for <5 year olds
- Reasonable for older children
- Affects concentration, focus, mood, and behaviour

The evidence

- 5-year-olds with >2 hours of screen-time/day had a 7·7-fold increased risk of meeting criteria for ADHD (Tamana, 2019)
- Increased ADHD behaviours with increased screen time (Beyens, 2018)
- **Smart phones:** millions of attentional windows and can induce ADHD in adolescents (Zheng, 2014)
- 25% of people with ADHD are addicted to screens
- Students addicted to their phones have lower self-regulated learning, low flow – feeling "in the zone" – and are constantly interrupted while studying (Lee, 2015)

Recommendations

- **Family Media Plan:** healthychildren.org/english/fmp/pages/mediaplan.aspx
- **Set limits and stick to them:** 1 hour of smartphone use/day; 1 hour of computer/day
- **Encourage and model other activities:** exercise, reading, board games, crafts, cooking, gardening
- Have media-free days/week
- **For studying:** turn off notifications/sound, schedule Do Not Disturb, put away phone, timed lockbox

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How long do parents spend fighting with their kids over screen time?

talker research

A research project through interviewing 2,000 parents

MAY 14, 2025

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Digital media, genetics, and ADHD risk in children Talker Research 2025

Implications and future directions

- Over four years, more social media use — unlike video games or TV — was linked to small but reliable increases in inattention in US children.
- The evidence favours a path from use to later symptoms, and genetic risk for ADHD (PGS-ADHD) did not change this link.
- As an observational study it can't prove causality, but the temporal pattern and robustness checks fit a likely causal relationship.
- Families, schools, and platforms can act now—through age limits, age verification, and notification controls.
- The findings may inform parental guidance and policy, with follow-up needed to see if the link persists into later adolescence.

Source: Nivins, S., Mooney, M. A., Nigg, J., & Klingberg, T. (2025). Digital Media Genetics and Risk for ADHD Symptoms in Children – A Longitudinal Study. Pediatrics Open Science. DOI: 10.1542/padsos.2025-000922

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How long do parents spend fighting with their kids over screen time? Talker Research 2025

Parents spend roughly 96 hours a year fighting with their kids over screen time — hundreds of technology-driven conflicts a year.

Behind the friction lies a deeper worry: 67% of parents fear screen addiction is costing them precious moments with their children.

Even more starkly, 41% fear they're losing their little one's childhood to technology altogether.

Their biggest concerns for kids online are exposure to predators (51%), accidental exposure to inappropriate content (46%), and screen addiction (45%).

Source: Talker Research for AngelQ (https://www.angelq.ai), survey of 2,000 parents of children in kindergarten through fifth grade.

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How long do parents spend fighting with their kids over screen time? Talker Research 2025

- Top worries: cyberbullying (44%), age-inappropriate content (40%), social media influence (37%), and mental health (37%).
- 61% say screen time is crowding out childhood development like social skills and empathy.
- 6 in 10 parents say their kids' time online is unsupervised.
- "Technology is aligned against families instead of allied with them. Our kids are losing years of childhood to exploitive products that addict them." – Tim Estes, CEO & Co-Founder, AngelQ
- Side effects reported: irritability (27%), mood swings (24%), and tantrums (22%).

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How long do parents spend fighting with their kids over screen time? Talker Research 2025

- Conflicts peak before dinner (64%), disrupting family bonding time.
- Top disagreements: length of screen time (43%), content or apps allowed (18%), and approval to watch (18%).
- Half of parents (52%) wish they had waited longer to give kids internet access – especially Gen Z (63%) vs. millennials (53%) and Gen X (50%).
- 34% say their own childhood tech experiences shaped how they limit their kids' access.
- Upsides parents value: exploring interests (60%), educational content (59%), digital skills (58%), cultural awareness (25%), and connecting with peers (24%).

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Stress and ADHD

Stress exacerbates ADHD symptoms

Common stressors that worsen symptoms:

- Bullying
- Harsh parenting
- Abuse
- Overscheduling
- Academic demands too high or too low

Stress reduction improves ADHD symptoms

- Mindfulness (Xue, 2019)
- Yoga (Chou, 2017)
- Smiling Mind App
- Fidgety toys (Aspiranti, 2021)
- Breathing exercises for calming (Zisopoulou, 2023)
- Positive parenting: Triple P

Recommendations

- Gentle, positive parenting to reduce stress
- Behaviour management (OT, psychologist)
- ADHD coaching
- Reduce school pressure, increase joy of learning
- Do not overschedule
- Mindfulness/breathing/yoga practice 10–15 minutes daily
- Fidget and sensory toys
- Sensory play area/quiet zone

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Anti-stress effect of theanine on students during pharmacy practice: Positive correlation among salivary α-amylase activity, trait anxiety, and subjective stress

Keiko Unno, Naoki Tanida, Naoto Ishii *et al . Pharm, Biochem, and Behaviour 111(2013) 128-135*

Results

- Placebo group had higher morning salivary α-amylase (sAA, a stress marker) than the theanine group during practice ($p = 0.032$)
- Subjective stress was significantly lower with theanine than placebo ($p = 0.020$)
- Higher pre-practice sAA linked to higher trait anxiety ($p = 0.015$) and shorter sleep ($p = 0.41 \times 10^{-3}$)

Conclusion

- Theanine intake suppressed students' initial stress response during long-term pharmacy practice

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Supplements as complementary treatments for ADHD

- **Bacopa monnieri** — consistently improved attention, hyperactivity, and memory.
- **Ginkgo biloba** — reduced symptoms, especially inattention, but less effective than standard medication.
- **L-theanine** — improved sleep efficiency in some cases; paired with caffeine may aid attention.
- **Caffeine** — minimal benefit on its own.

Bottom line: mild benefits as complementary treatments; more research is needed on safe dosages and long term use.

 Source: Nutrients. 2025 Apr 28;17(9):1482. doi: 10.3390/nu17091482 bioconcepts

Lithium and brain function

Courtesy of Mosaic Diagnostics:

- Interest in lithium for neurodegenerative disease began when bipolar patients on lithium showed lower rates of cognitive decline than peers on other medications. In 66 elderly bipolar patients on chronic lithium versus 48 who were not, Alzheimer's prevalence was just 5% compared with 33%. (Nunes et al., 2007)
- Two Danish studies using different designs reached strikingly similar results: records of over 21,000 lithium-treated patients showed decreased levels of both dementia and Alzheimer's. (Kessing et al., 2008, 2010)
- A prevention-focused trial randomized 45 participants with mild cognitive impairment (MCI) to sub-therapeutic lithium (150–600 mg/day) or placebo for 12 months. The lithium group showed reduced destructive tau proteins and improved cognitive performance, while placebo tau rose steadily; tolerability was good with 91% adherence. (Forlenza et al., 2011)
- Researchers concluded **lithium has a significant disease-modifying effect on preventing dementia and Alzheimer's** when initiated early in the disease progression.

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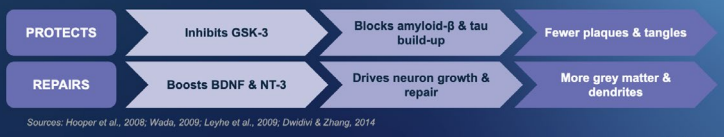
Lithium and brain function

Continued…

Courtesy of Mosaic Diagnostics:

- Low-dose evidence comes largely from epidemiology: eleven studies of lithium in drinking water across regions—including Texas counties, the 100 largest US cities, 99 Austrian districts, Greece and Japan — analysed outcomes in well over 10 million subjects. In 9 of the 11, higher lithium levels were linked to better behavioural, legal and medical outcomes. (Dawson, 1970; Schrauzer & Shrestha, 1990; Mauer et al., 2014)
- In a clinical trial, just 0.3 mg of lithium daily for 15 months kept Alzheimer's patients' cognitive scores stable while controls declined — and scores began rising three months in. (Nunes, 2013)

Key neuroprotective mechanisms of lithium



Sources: Hooper et al., 2008; Wada, 2009; Leyhe et al., 2009; Dwidivi & Zhang, 2014

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Lithium Orotate

Modern use of safer Lithium Orotate:

- **In ASD children:** maximum dose 20mg (elemental) evening dose
- **In Bipolar Affective Disorder or ADHD:** adults 60-80mg (elemental)
- **With orotate, more lithium enters the brain (rat studies);** and there are no toxic effects as happens with Lithium carbonate. Renal and thyroid functions are not at risk: especially in dehydration states. There is no need for blood levels testing to monitor toxicity
- **I prefer muscle testing to determine the dosage or usage in these patients**
- **These are available via compounding laboratories**

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Stimulant Medication

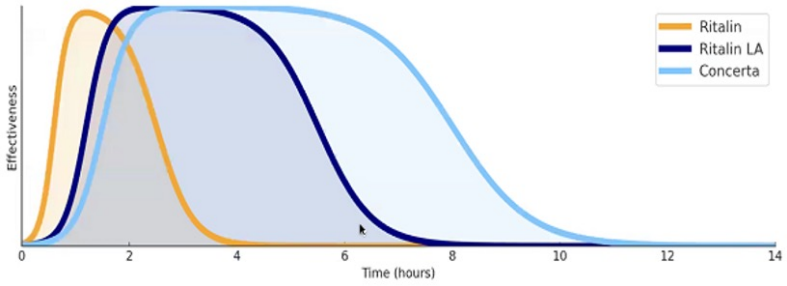


	Methylphenidate	Dexamfetamine
Immediate Release **4 hours**	Ritalin (10mg)	Dexamfetamine (5mg)
Long Acting **6-8 hours**	Ritalin LA (10,20,40,60mg)	
Extended Release **10-12 hours**	Concerta (18,27, 36,54mg) (Paed diagnosis for PBS)	Vyvanse (Lisdexamfetamine) (20,30,40,50,60,70mg)

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Methylphenidate preparations



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Second line medications





Atomoxetine (Strattera)

Guanfacine (Intuniv)

- Not tolerating Stimulants
- Inadequate control despite maximum doses of Stimulant medication
- Specialist input

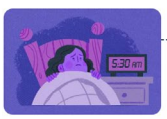
Clonidine: aids sleep

Melatonin: may help sleep

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Adverse Effects



Sleep Disruption

Appetite Suppression and weight loss





Mood changes

Wearing off effect



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ADHD methylphenidate treatment linked to adult obesity: JAMA study

Importance. **MPH is the top treatment for childhood ADHD, but its long-term effect on growth is unclear.**

Methods. Nationwide cohort of youths newly diagnosed with ADHD; 4-year MPH exposure linked to adult BMI and height.

Results. In 34,850 youths, children with ADHD had a higher adult BMI (24.3 vs 23.3); height was unaffected.

Conclusions and Relevance. MPH-treated childhood ADHD is linked to higher adult BMI and shorter stature than peers.

60%
higher adult BMI
vs. peers without ADHD

Jihun Song, Sun Jae Park, Jiwon Yu, *et al*
JAMA Network Open 12 January 2026; Vol 2. Number 1

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Monitoring

Baseline:

- Height, Weight
- CV assessment
- Sleep history
- Nutrition and Appetite

Review at **3 months after initiation** or **change in medication**

Review in person **6 monthly once stable**

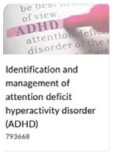


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Resources

- RACGP module – GP learning
- AADPA
- Health Pathways
- ADHD Australia
- ADHD Foundation of Australia



HealthPathways

ADHD Foundation

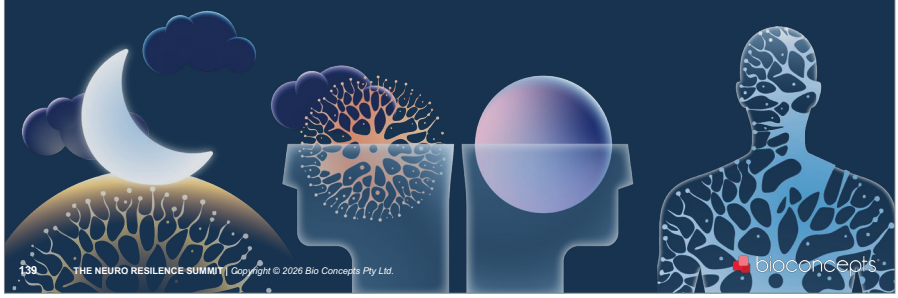


ADHD AUSTRALIA

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Is it truly ADHD or something else?



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Something else?

- The **"hyperactive" child** with a **very high IQ; and is gifted**.
- **The child with Autism level 3:** out of control for multiple reasons.
- **The intergenerational factors + maternal trauma** (first 2000 days of life).



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Brighter Beginnings: The First 2000 Days of Life

April 2021

Why do the First 2000 Days matter?

The first 2000 days of a child's life, from conception to the time they start school, is an important time for physical, social and emotional health. Experiences during these early years have deep and long-lasting effects, predicting outcomes like:

- **Success at school and in finding a job**
- **Lifelong health**
- **Risk of substance abuse and criminal behaviour**

Early experiences change the brain

- During the first 2000 days of life, the brain is rapidly changing and growing.
- A child's environment and experiences during these early years have a lasting impact on their health, development, learning and wellbeing. This is because the growing brain changes to respond to the conditions around it, and these changes stick.
- Exposure to toxic stress during the first 2000 days of life can disrupt development
- Exposure to risks like abuse and neglect, domestic violence, household mental illness and poverty lead to poorer health outcomes. The greater the number of stressors, the greater the risk to development.
- Protective factors can buffer the impact of stress and support healthy development. This includes things like parent support programs, substance abuse treatment, high quality childcare, and income support for low-income families.
- As seen on the left, these protective factors can counter risk factors to ensure better outcomes for

This Document - Understanding The First 2000 Days Of Life

1. Why does it matter?
2. Acting early
3. What can we do?
4. What works best?
5. Our plan for action

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Cardiovascular effects of prenatal stress
— Are there implications for cerebrovascular, cognitive and mental health outcome

Highlights

- Maternal stress but not synthetic GC exposure affects maternal-foetal circulation.
- Foetal GCs program blood pressure regulation and vascular tone in animal studies.
- Increase in peripheral resistance is predominantly programmed by endothelin-1.
- Increase in coronary resistance is programmed by reduced NO-mediated vasodilatation.
- Gestational age and sex are important moderators in cardiovascular programming.

Julia Müller, Antonow-Schlorke, Nasim Kroegel, *et al.* **Neuroscience & Biobehavioral Reviews Volume 117**, October 2020, Pages 78-97

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Something else?

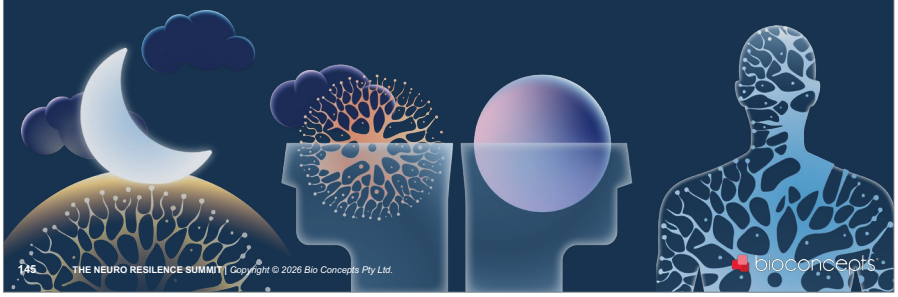
- **Climatic changes:** temperature, humidity, solar/lunar influences:
  - these can cause irritability, distractibility, poor focus, poor cognitive alertness, and disruptive behaviour.
- **Parasites:** commonly-theadworm/pinworm, Blastocystis, Dientamoeba, Flukes. They can be very disturbing: often at night & new/full moon.
- **Streptococcus/Enterococcus:** these and many other infections can be part of PANS/ PANDAS. Streptococcal infections can often produce autoimmune antibodies (ASOT, Ante-DNase Abs) adversely affecting the basal ganglia (Basal ganglia encephalopathy).

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Something else?

- **Lyme disease, Bartonella, Babesia, Rickettsia:** these conditions can be extremely debilitating - mentally & physically.
- **Mast cell activation syndrome:** can feature a plethora of irritating symptoms including clear rhinorrhoea, asthma, allergic rhinitis, abdominal pain, migraine, blushing, intense itching, and urticaria.
- **POTS:** can present as pre/or full syncope, weakness, vagueness, hypotension, and tachycardia. It can be related to thoracic outlet syndrome (vascular compression), Nutcracker syndrome, and ovarian or pelvic venous compression.

Possible alternative diagnoses

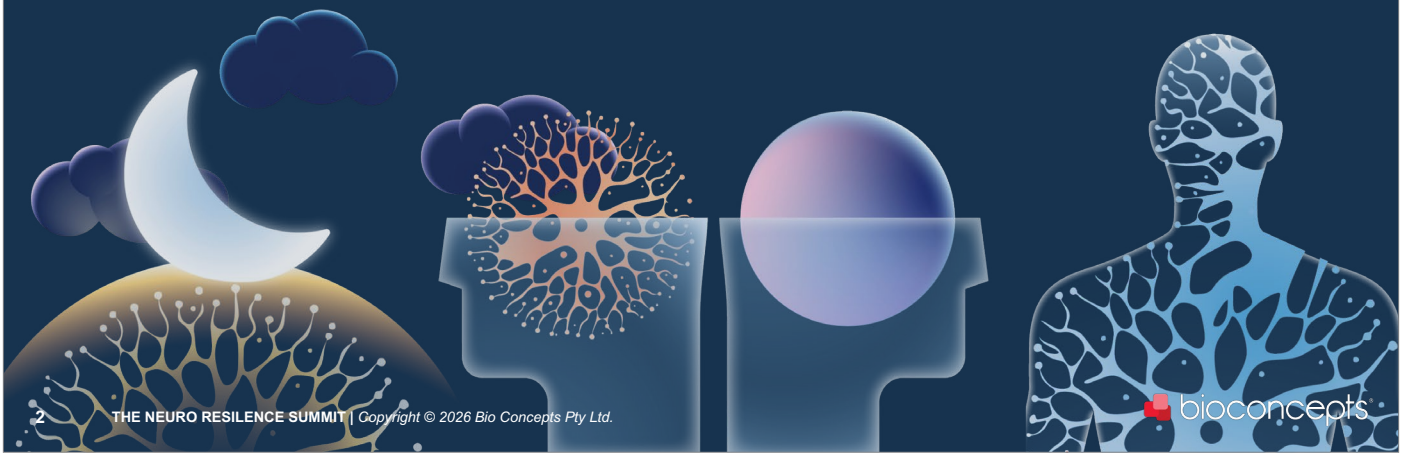


Something else?

- **Epilepsy:** esp. absent ones
- **Brain injury and multiple concussions**
- **Dyslexia**
- **Dyspraxia/apraxia**
- **Congenital conditions; hyperthyroidism**
- **ASD**
- **Brain fog from mould & vector infections**
- **Fraud/faking**

Beyond the Hazard Zone:

Decoding PMDD Through Genetics, Cannabinoids & Neurotransmitter Chaos



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SPEAKER

Tanya Borowski

Nutritional Therapist; IFMCP



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OUTLINE

1. The Landscape PMDD
2. The 5 Interwoven Pieces Framework
3. Hormones & Neurosteroids Impact on Neurotransmitters
4. The DASH-MC Model & Interplay
5. Nutrigenomics Interplay
6. HPA |EDC| Circadian Amplification

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The Landscape of PMDD

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The Scale of The Problem

- DSM-5 prevalence: 1.8–5.8% of menstruating women (PMDD)
- 3–8% prevalence cited in epidemiological literature
- PMDD affects as many women as generalised anxiety disorder or panic disorder
- PMDD is not 'bad PMS' — it is a depressive disorder with a neurobiological signature

Reilly et al. (2024) *Journal of Affective Disorders* 349: 534–540

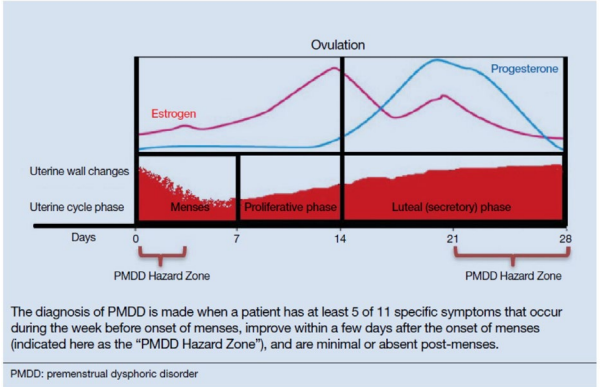
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The Hazard Zone

Figure 1

The menstrual cycle and the 'PMDD Hazard Zone'



The diagnosis of PMDD is made when a patient has at least 5 of 11 specific symptoms that occur during the week before onset of menses, improve within a few days after the onset of menses (indicated here as the "PMDD Hazard Zone"), and are minimal or absent post-menses.

PMDD: premenstrual dysphoric disorder

The Diagnosis

The presence of least **5 of 11**

- **Cognitive Affective**
- **Behavioural**

or

- **Somatic**

Present in the week before menses, and are minimal or absent post-menses

" PMDD Hazard Zone "

Diagnostic and statistical manual of mental disorders, 5th ed. Washington, DC: American Psychiatric Association; 2013.

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PMDD - Premenstrual Dysphoric Disorder

- Symptoms should be tracked prospectively for at least 2 menstrual cycles in order to confirm Dx
- One *must* be an ***affective*** *symptom* and another *must* be a ***behavioural/cognitive*** *symptom*

Affective symptoms:

- Lability of affect (e.g. sudden sadness, tearfulness, or sensitivity to rejection)
- Irritability, anger, or increased interpersonal conflicts
- Depressed mood, hopelessness, or self-deprecating thoughts
- Anxiety or tension, feeling "keyed up" or "on edge"

Behavioural/cognitive symptoms:

- Decreased interest in usual activities (e.g. work, hobbies, friends, school)
- Difficulty concentrating
- Lethargy, low energy, easy fatiguability
- Change in appetite, overeating, food cravings
- Hypersomnia or insomnia
- Feeling overwhelmed or out of control
- Physical symptoms (breast tenderness or swelling, headache, joint or muscle pain, bloating, weight gain)

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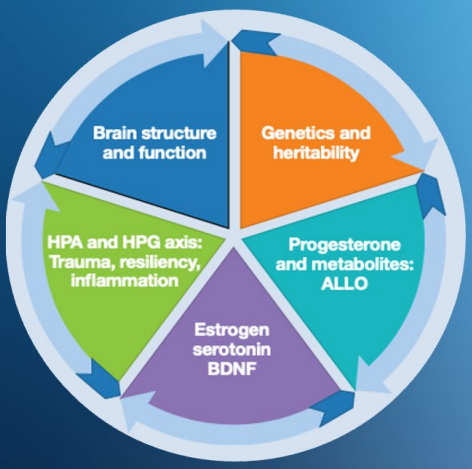
Beyond The Hazard Zone

The 5 Interwoven Pieces

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The 5 Interwoven Pieces Framework



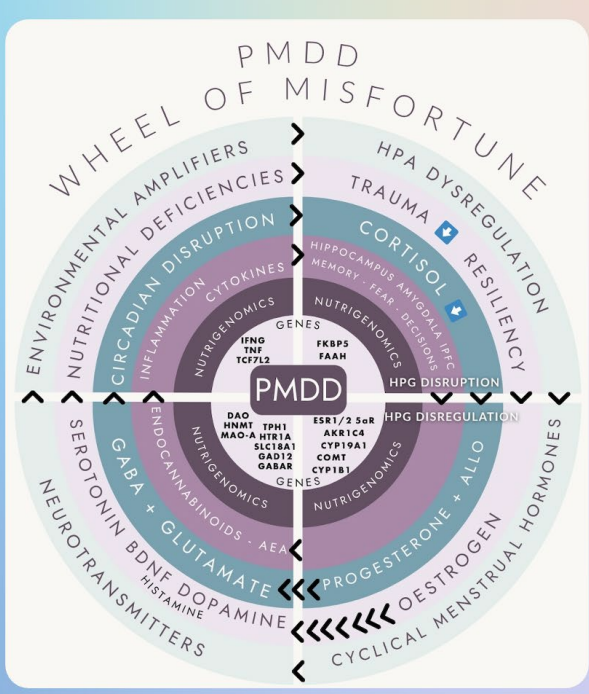
5 overlapping variables:

1. **Genetic susceptibility** (**ESR1** - oestrogen receptor, and **5-HT1A** - serotonin receptor, **5-HTTLPR** - serotonin transporter)
2. **Progesterone** and **allopregnanolone** (**AlloP**) and **GABA**
3. **Oestrogen, serotonin** & brain derived neurotrophic factor (**BDNF**)
4. **HPA/HPG axis** - trauma, resilience, sleep & inflammation. **Cortisol (FKBP5)**
5. Differences in brain structure & function (with hormonal variation)

All strongly influenced by genetics

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The Interwoven Pieces Expanded: A Systems Map of PMDD Aetiology

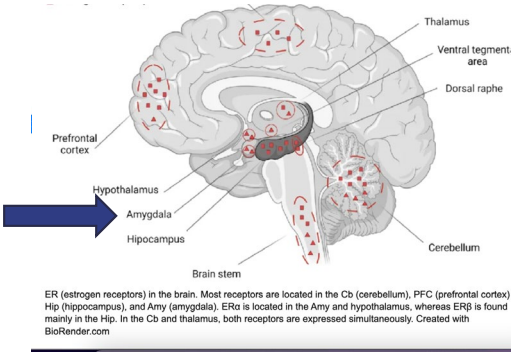


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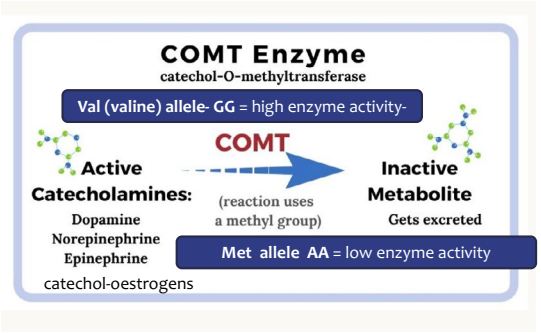
Genetic Susceptibility

ESR1 (ERα):

- Primary mechanism E2 action in brain
- ERα concentrated in the *amygdala* & hypothalamus
- **Amygdala** - threat detection, emotional reactivity, and arousal
- **Hypothalamus** – ANS function; HPA & somatic regulation
- ESR1 variants = first positive genetic finding in PMDD — but **only** in COMT Val/Val (fast clearance)



ER (estrogen receptors) in the brain. Most receptors are located in the Cb (cerebellum), PFC (prefrontal cortex) Hip (hippocampus), and Amy (amygdala). ERα is located in the Amy and hypothalamus, whereas ERβ is found mainly in the Hip. In the Cb and thalamus, both receptors are expressed simultaneously. Created with BioRender.com



Miller et al (2010) J Psychiatr Res. 44(12):788-794

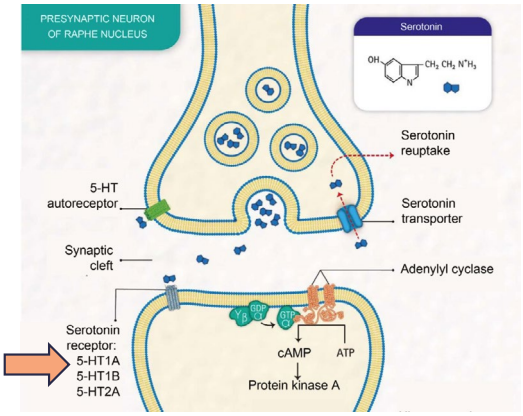
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Genetic Susceptibility

- **5-HT1A — C(-1019)G polymorphism**
- Increases auto-receptor expression

→ oversensitive feedback brake → excess inhibition of serotonin release → reduced baseline serotonergic tone

- 2.5× increased PMDD risk from a single allele — serotonergic vulnerability is hardwired, not hormone-driven
- SSRIs "flood" the autoreceptor → desensitisation → brake releases → serotonergic tone restored



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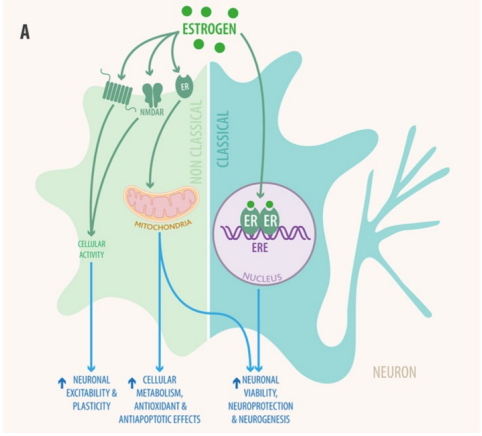
Hormones & Neurosteroids: Oestrogen in the Female Brain

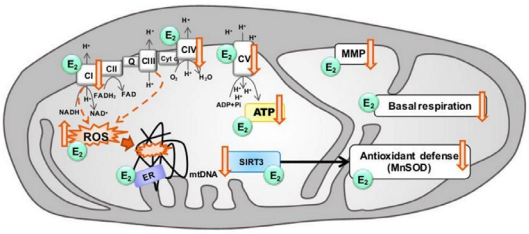
E2 exerts neuroprotective effects

- **Neurogenesis**: E2 ↑ BDNF → synaptic plasticity-neurogenisis→ learning & memory
- **Cell survival**: E2 → enhances the expression of antioxidant enzymes with mitochondria
- **Brain energy supply**: E2 ↑ glucose transporters, uptake & ATP-producing enzymes - fuels neurons
- **When E2 falls(cyclically & life-stage)**:

Neuroprotection*| *Bioenergetics* |*Neurogenesis all withdrawn

Not *just* a mood story, it is a brain maintenance story





Del Río et al. (2018). Frontiers in Public Health 6:141
Lejri et al. (2018) **Mitochondria, Estrogen and Female Brain Aging.Front. Aging Neurosci. 10:124.**

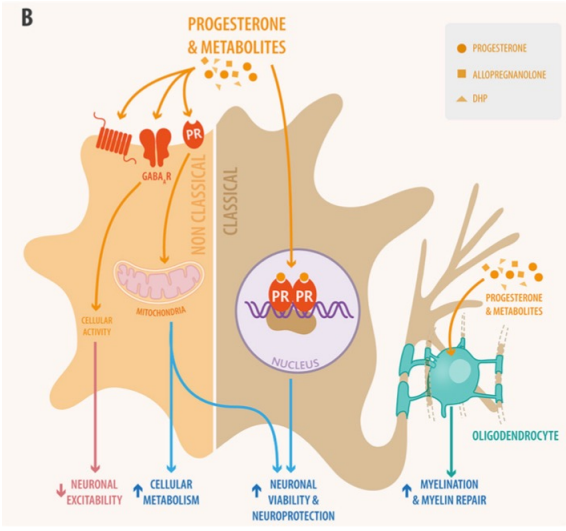
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Hormones & Neurosteroids; Progesterone in the Female Brain

- **P4 shares E2's neuroprotective roles:** ↑ cell survival, ↑ bioenergetic regulation, ↑ neural cell proliferation
- **Myelination**: P4 and its metabolites (DHP, ALLO) stimulate oligodendrocyte progenitor proliferation and myelin sheath synthesis in the CNS (GABA-A receptors)
- **When P4 and ALLO withdraw rapidly**:

myelination support, bioenergetic regulation and cell survival signalling are all simultaneously reduced



Del Río et al. (2018). Frontiers in Public Health 6:141

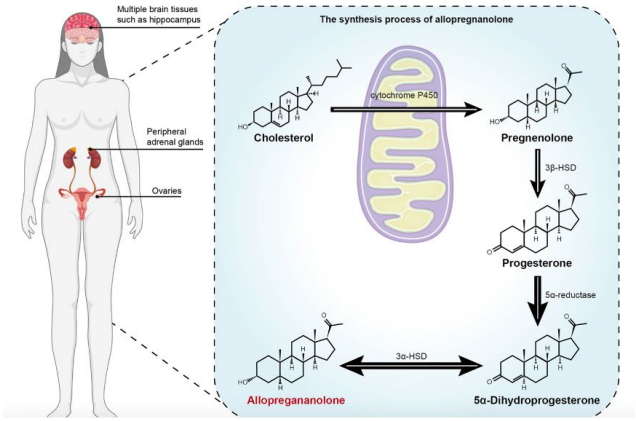
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Progesterone Metabolism

- Allopregnanolone levels rise in tandem with progesterone across the luteal phase
- Equal in PMDD and non – PMDD groups
- **Some** women with PMDD showed higher mid-luteal 5α-DHP, "suggesting" impaired conversion to ALLO

3a- HSD Inducers
PEA, EPO, gingko biloba, crocus sativus
&
SSRIs.



Martinez, P.E., et al., *5alpha-Reductase Inhibition Prevents the Luteal Phase Increase in Plasma Allopregnanolone Levels and Mitigates Symptoms in Women with Premenstrual Dysphoric Disorder.* Neuropsychopharmacology, 3/2016. **41**(4): p. 1093-1102.

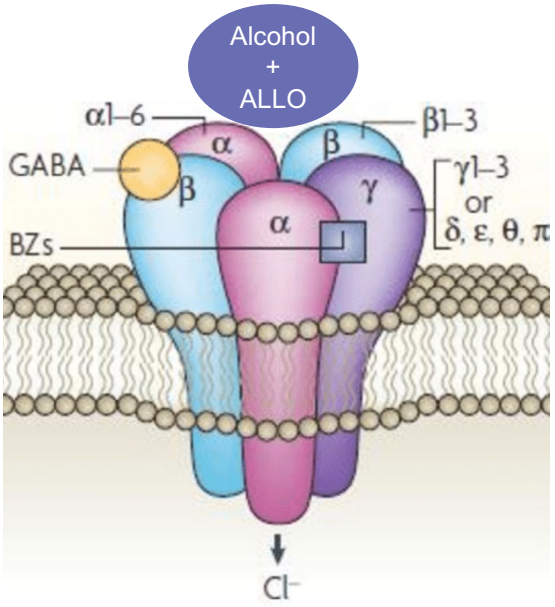
[Timby, E., et al., *Women with premenstrual dysphoric disorder have altered sensitivity to allopregnanolone over the menstrual cycle compared to controls-a pilotstudy.* Psychopharmacology. (Berl.), 6/2016. **233**(11): p. 2109-2117.

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GABA Receptor

- GABA-A receptor "shuffles" the 5 subunits throughout the menstrual cycle
- GABA binds →CL- into cell, reduces nerve activity
- *Benzo* – increase *frequency* channel opening
- *Alcohol* | *ALLO* – positive positive allosteric modulator enhance inhibitory effect
- PMDD types exhibit differences in GABA-A subunit structure and abnormal patterns of neural activation

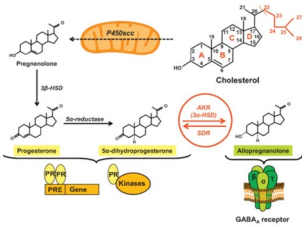


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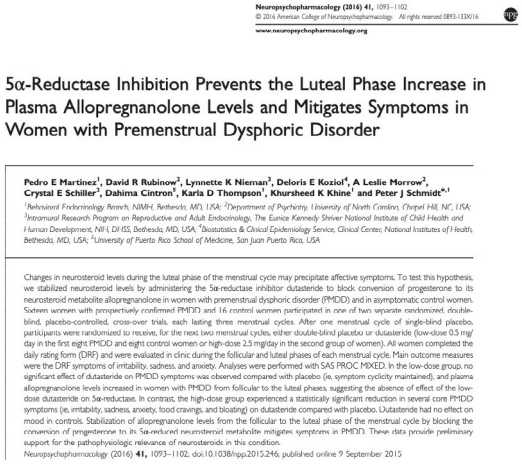
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Proof of Concept

- ***Dutasteride RCT*** (Martinez et al.): block 5α-reductase → stabilise ALLO → ↓ irritability, anxiety, low mood



- ***Sepranolone***: GABA-A steroid antagonist → blocks ALLO's adverse effects → **75% reduction** in mood symptoms
- ***SSRIs***: rapid effect (2–3 days) via ALLO biosynthesis enhancement and/or GABA-A sensitisation


Neuropsychopharmacology (2016) 41, 1093–1102
© 2016 American College of Neuropsychopharmacology. All rights reserved 0893-133X/16
www.neuropsychopharmacology.org

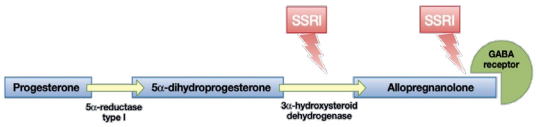
5α-Reductase Inhibition Prevents the Luteal Phase Increase in Plasma Allopregnanolone Levels and Mitigates Symptoms in Women with Premenstrual Dysphoric Disorder

Pedro E Martinez[1], David R Rubinow[2], Lynnette K Nieman[3], Deloris E Kozioł[4], A Leslie Morrow[2], Crystal E Schiller[2], Dahima Cintron[2], Karla D Thompson[1], Khursheed K Khine[1] and Peter J Schmidt*,[1]

[1]Behavioral Endocrinology Branch, NIMH, Bethesda, MD, USA; [2]Department of Psychiatry, University of North Carolina, Chapel Hill, NC, USA; [3]Intramural Research Program on Reproductive and Adult Endocrinology, The Eunice Kennedy Shriver National Institute of Child Health and Human Development, NIH, DHSS, Bethesda, MD, USA; [4]Biostatistics & Clinical Epidemiology Service, Clinical Center, National Institutes of Health, Bethesda, MD, USA; [5]University of Puerto Rico School of Medicine, San Juan Puerto Rico, USA

Changes in neurosteroid levels during the luteal phase of the menstrual cycle may precipitate affective symptoms. To test this hypothesis, we stabilized neurosteroid levels by administering the 5α-reductase inhibitor dutasteride to block conversion of progesterone to its neurosteroid metabolite allopregnanolone in women with premenstrual dysphoric disorder (PMDD) and in asymptomatic control women. Sixteen women with prospectively confirmed PMDD and 16 control women participated in one of two separate randomized, double-blind, placebo-controlled, cross-over trials, each lasting three menstrual cycles. After one menstrual cycle of single-blind placebo, participants were randomized to receive, for the next two menstrual cycles, either double-blind placebo or dutasteride (low-dose 0.5 mg/day in the first eight PMDD and eight control women or high-dose 2.5 mg/day in the second group of women). All women completed the daily rating form (DRF) and were evaluated in clinic during the follicular and luteal phases of each menstrual cycle. Main outcome measures were the DRF symptoms of irritability, sadness, and anxiety. Analyses were performed with SAS PROC MIXED. In the low-dose group, no significant effect of dutasteride on PMDD symptoms was observed compared with placebo (ie, symptom cyclicity maintained), and plasma allopregnanolone levels increased in women with PMDD from follicular to the luteal phases, suggesting the absence of effect of the low-dose dutasteride on 5α-reductase. In contrast, the high-dose group experienced a statistically significant reduction in several core PMDD symptoms (ie, irritability, sadness, anxiety, food cravings, and bloating) on dutasteride compared with placebo. Dutasteride had no effect on mood in controls. Stabilization of allopregnanolone levels from the follicular to the luteal phase of the menstrual cycle by blocking the conversion of progesterone to its 5α-reduced neurosteroid metabolite mitigates symptoms in PMDD. These data provide preliminary support for the pathophysiologic relevance of neurosteroids in this condition.

Neuropsychopharmacology (2016) **41**, 1093–1102; doi:10.1038/npp.2015.246; published online 9 September 2015



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Naturopathic Interventions

Saffron exhibits SSRIs-like actions


International Journal of Science and Research Archive
eISSN: 2582-8185
Cross Ref DOI: 10.30574/ijsra
Journal homepage: https://ijsra.net/
IJSRA
(Review Article)
Check for updates

Comparing the efficacy of saffron with fluoxetine for the effective management of premenstrual dysphoric disorder: A review

RAJKUMAR M *, VAISHNAVI S, SHRIBALAJI S and SHARU LATHA BINU AJ

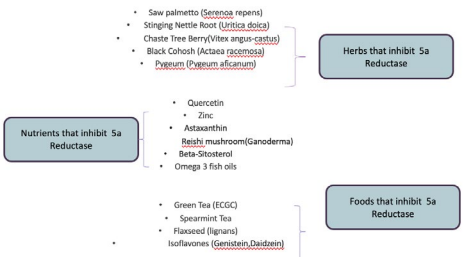
Department of Pharmacy Practice, Arulmigu Kalasalingam College of Pharmacy, Krishnankoil, Virudhunagar-626126, Tamil Nadu, India.

International Journal of Science and Research Archive, 2024, 12(02), 2001–2006

Publication history: Received on 04 July 2024; revised on 13 August 2024; accepted on 15 August 2024

Article DOI: https://doi.org/10.30574/ijsra.2024.12.2.1484

5a Reductase inhibitors



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Naturopathic Interventions

GABA Support

Valerian
Rosmarinic acid
(rosemary, lemon balm, sage, thyme)
L-theanine

3a HSD Inducers

PEA
EPO
gingko biloba
crocus sativus
B3 as co-factor

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The Four Neurotransmitters: As Hormonal Targets

Glutamate — brain's main *excitatory* neurotransmitter; drives memory, learning and cognitive processing

GABA — brain's main *inhibitory* neurotransmitter; balances neural excitability; when GABA function is disrupted → anxiety; when potentiated → anxiolysis (benzodiazepine mechanism)

Serotonin **(5-HT)** — wellbeing, mood, sleep-wake cycle, digestion, sexual behaviour, cognitive function

Dopamine — reward, motivation, pleasure, decision-making, learning

All four are directly regulated by oestrogen and progesterone — at the level of synthesis, release, degradation and receptor expression

All strongly influenced by genetics

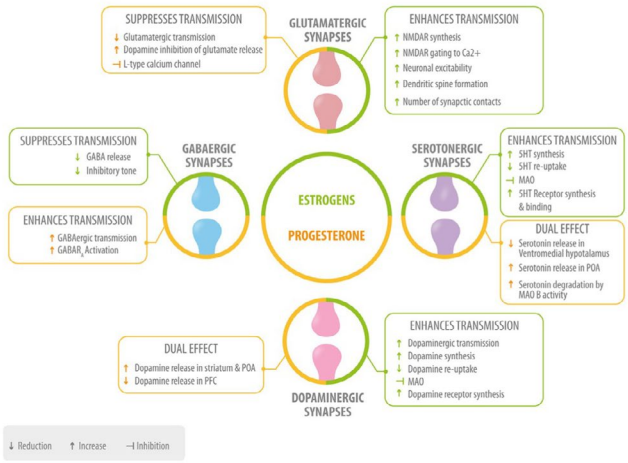
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Oestrogen & Neurotransmitters: The Brain's Amplifier

- **Glutamate:**
- E2 potentiates NMDA receptor → ↑ synaptic plasticity, learning & memory;
  ↑ spine density in hippocampus, amygdala & PFC

- **GABA:**
- E2 *suppresses* GABAergic transmission — creates a window of heightened neural excitability and sensitivity



*Int. J. Mol. Sci.*2025, *26*(6), 2532;https://doi.org/10.3390/ijms26062532
Del Río et al. (2018). Frontiers in Public Health 6:141

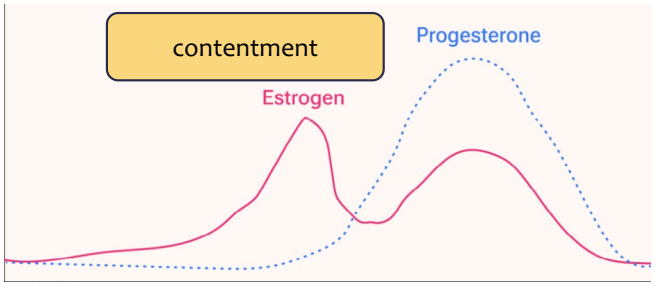
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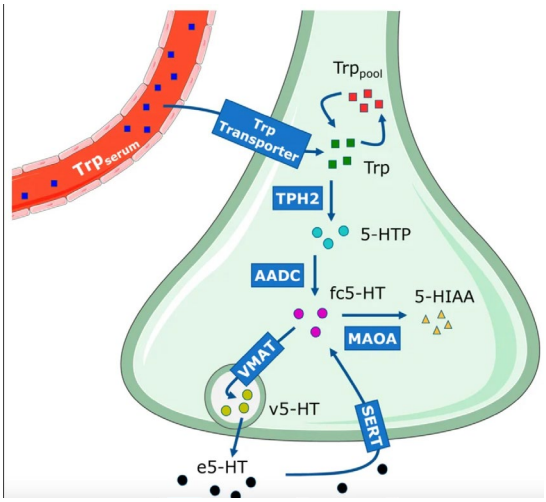
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Serotonin:

E2 → ↑ tryptophan hydroxylase (synthesis)
↓ MAO ↓ reuptake ↑ receptor expression
= more serotonin, available, longer acting

P4 → ↓ receptor expression ↑ MAO





- *Int. J. Mol. Sci.*2025, *26*(6), 2532;https://doi.org/10.3390/ijms26062532
- Del Río et al. (2018). Frontiers in Public Health 6:141

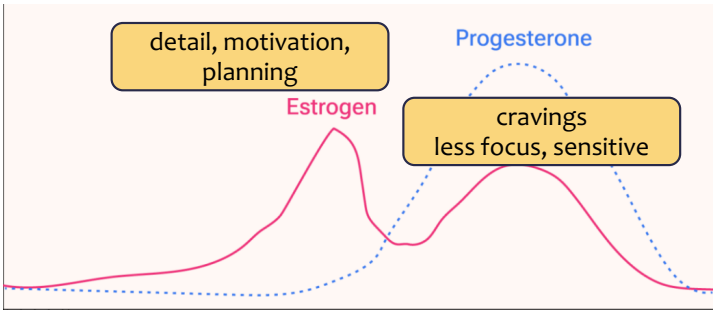
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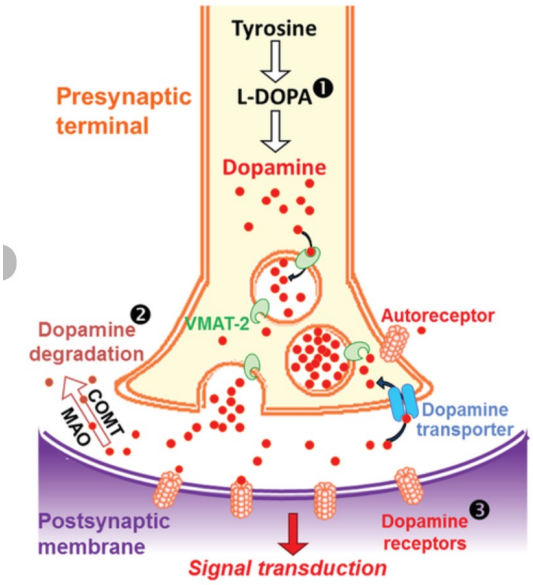
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Dopamine:

E2 → ↑ Tyrosine hydroxylase ↓ MAO
↑ dopamine receptors
protects working memory & motivation

ALLO → positive effect dopamine release = reward behaviour/ cravings – luteal phase
In pre-frontal cortex – inhibits dopamine





Int. J. Mol. Sci. 2025, 26(6), 2532;https://doi.org/10.3390/ijms26062532
Del Río et al. (2018). Frontiers in Public Health 6:141

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HPA/HPG axis - When Stress Hijacks the Hormonal System

- **HPA and HPG axes are inversely linked:** when HPA activates (stress → ↑ cortisol), HPG is suppressed → ↓ GnRH → ↓ FSH/LH
- **GABA is the central switch:** GABAergic transmission regulates both CRH and GnRH neurons — GABA-A receptor dysfunction disrupts both axes at once
- **Chronic stress /GABAergic HPA control:** early life stress shifts GABA currents in PVN neurons — the stress axis becomes permanently harder to switch off
- **PMDD-susceptible women:** altered ALLO-dependent GABA-A function at the PVN → heightened stress sensitivity during phases of hormonal variability

The vicious loop: hormonal flux → GABA-A instability → poor HPA regulation → ↑ cortisol → further HPG suppression → worsened hormonal dysregulation

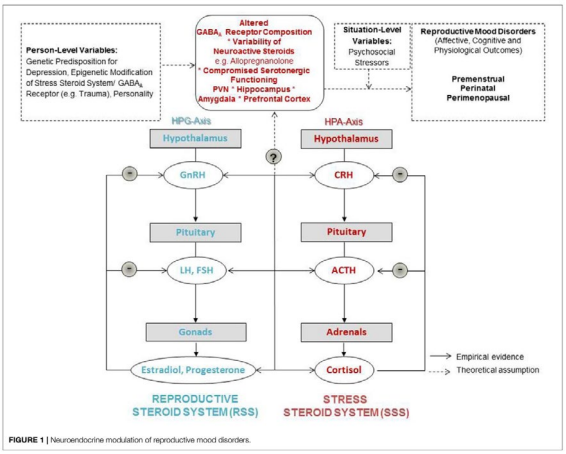


FIGURE 1 | Neuroendocrine modulation of reproductive mood disorders.

Sources: Schweizer-Schubert et al. (2021) Front. Med. 7:479646; Hantsoo & Epperson (2020) Neurobiol. Stress 12:100213

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Beyond The Hazard Zone

The DASH-MC Framework

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The DASH-MC Framework

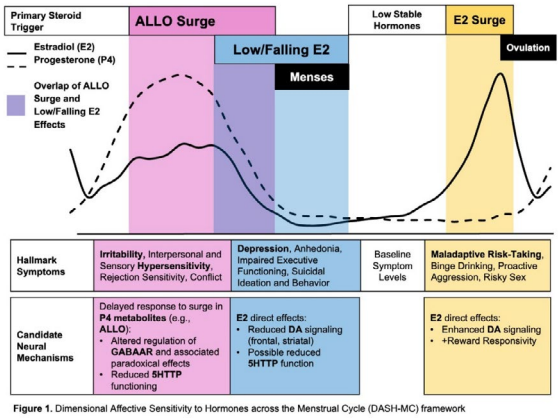


Figure 1. Dimensional Affective Sensitivity to Hormones across the Menstrual Cycle (DASH-MC) framework

Peters et al. DASH-MC (2024); Schweizer-Schubert et al. (2021)

The DASH-MC framework: Dimensional Affective Sensitivity to Hormones across the Menstrual Cycle — a transdiagnostic model of how hormonal change drives neurotransmitter dysregulation

The key insight: this is not about hormone levels being too high or too low — it is about how sensitively the brain's neurotransmitter systems respond to hormonal *change*

Three distinct patterns

Three distinct mechanisms

Three different timepoints

More nuanced interventions

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When: ~ cycle days approx. 17–24; onset after ovulation, peaks late luteal, **clears rapidly with menses**

What it looks like: irritability, reactive anger, rejection sensitivity, sensory hypersensitivity (including misophonia), emotional lability, somatic anxiety

Mechanism: P4 → ALLO surge → paradoxical GABA-A response (α4 subunit reconfiguration failure) + reduced luteal serotonin function

Treatment: SSRIs (luteal-phase dosing), dutasteride, sepranolone, GnRHa ± add-back

Naturopathic mimics >>> + personalised Nutrigenomics

Peters et al. DASH-MC (2024); Hantsoo & Epperson (2020)

Pattern 1: Midluteal — The Irritability & Hyperarousal Window

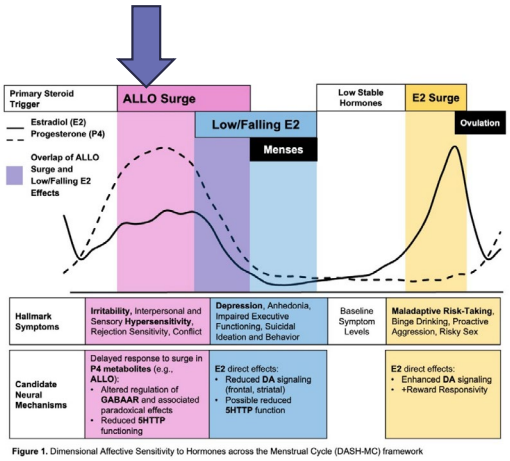


Figure 1. Dimensional Affective Sensitivity to Hormones across the Menstrual Cycle (DASH-MC) framework

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Naturopathic Interventions

Saffron exhibits SSRIs-like actions



International Journal of Science and Research Archive

eISSN: 2582-8185

Cross Ref DOI: 10.30574/ijsra

Journal homepage: https://ijsra.net/

IJSRA

(Review Article)

Check for updates

Comparing the efficacy of saffron with fluoxetine for the effective management of premenstrual dysphoric disorder: A review

RAJKUMAR M *, VAISHNAVI S, SHRIBALAJI S and SHARU LATHA BINU AJ

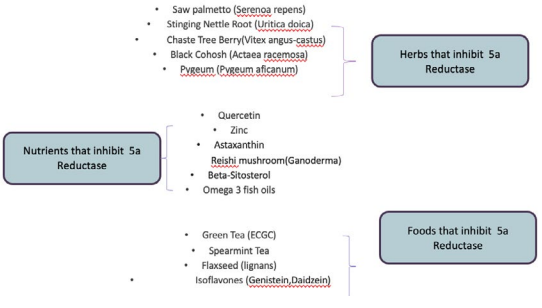
Department of Pharmacy Practice, Arulmigu Kalasalingam College of Pharmacy, Krishnankoil, Virudhunagar-626126, Tamil Nadu, India.

International Journal of Science and Research Archive, 2024, 12(02), 2001–2006

Publication history: Received on 04 July 2024; revised on 13 August 2024; accepted on 15 August 2024

Article DOI: https://doi.org/10.30574/ijsra.2024.12.2.1484

5a Reductase inhibitors



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When: ~ cycle days approx 26–5 (late luteal into early follicular); **does not resolve with the bleed — persists into follicular phase**

What it looks like: low mood, hopelessness, anhedonia, cognitive impairment (working memory, concentration, word retrieval), suicidal ideation

Critically missed: symptoms extend well past menses onset — this is why it is so consistently overlooked

Mechanism: falling E2 → ↓ dopamine + ↓ serotonin function → anhedonia, executive dysfunction, impaired fear extinction

Treatment distinction: GnRHa may NOT help this subtype — **perimenstrual E2 support** is the targeted intervention

Peters et al. DASH-MC (2024); Hantsoo & Epperson (2020)

Pattern 2: Perimenstrual — The Depression & Cognitive Dysfunction Window

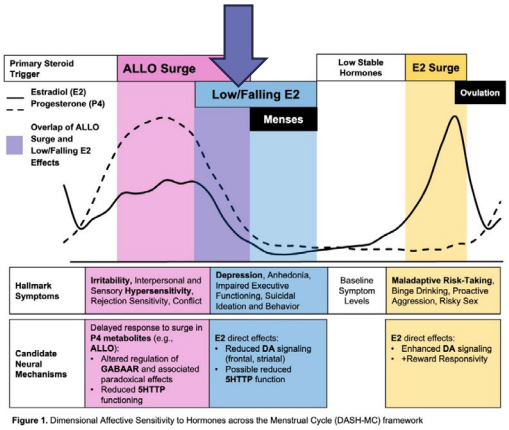


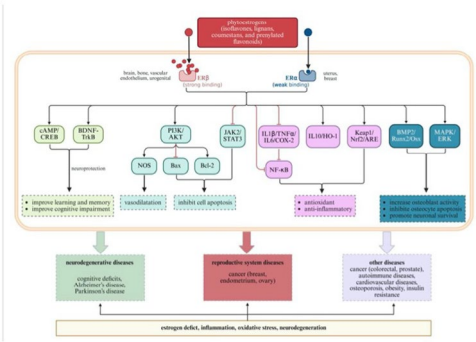
Figure 1. Dimensional Affective Sensitivity to Hormones across the Menstrual Cycle (DASH-MC) framework

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Naturopathic Interventions

Phytoestrogens



- Soy isoflavones (SIF)
- Selective oestrogen receptor modulators (SERMs)
- Interacts with ERs (ERβ-selective binding) to mitigate Sx associated with E2 decline

Liu et al, 2025

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When: ~ cycle days approx 11–14; mid-cycle periovulatory — **almost always missed because focus is only on premenstrual window**

What it looks like: increased alcohol use (social motives), impulsive decision-making, proactive aggression, elevated risk-taking, gambling

Mechanism: E2 surge → ↑ dopamine → heightened reward system responsivity — disinhibiting effect of alcohol nearly **doubled** during late follicular E2 elevations

Particularly relevant in: BPD, substance use disorders, eating disorders, bipolar disorder

Treatment considerations: Oestrogen metabolism pathways , dopaminergic regulation & impulse support

Pattern 3: Periovulatory — The Reward Hypersensitivity Window

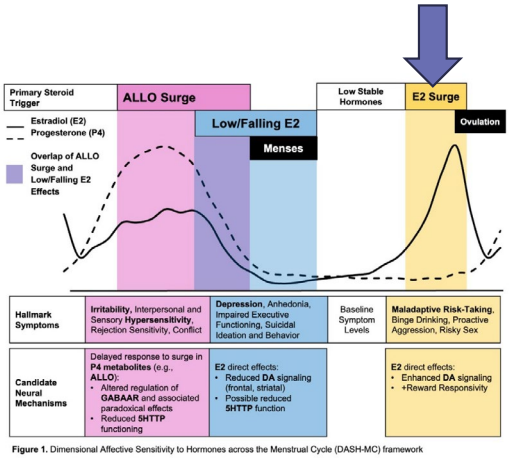


Figure 1. Dimensional Affective Sensitivity to Hormones across the Menstrual Cycle (DASH-MC) framework

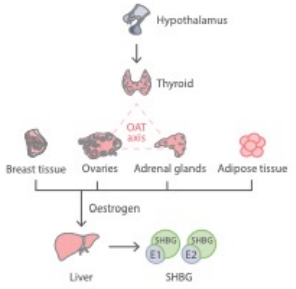
Peters et al. DASH-MC (2024); Hantsoo & Epperson (2020)

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Starting from the top down

After minimising exogenous endocrine disrupting influences and modifying diet and lifestyle factors, **balance begins upstream with the conductor - hypothalamic regulation of the OAT (ovarian, adrenal and thyroid).** Foundational interventions include Withania, Vitex, Peony, Black cohosh, iodine, zinc, magnesium, and vitamins A and B6.[13–20]

Protein, resveratrol, dietary flax lignans and vitamin D can support adequate **SHBG and albumin availability and function to reduce free circulating oestrogen.**[21–24]





Oestrogen receptor mediators including rosemary, resveratrol, curcumin, silymarin, black cohosh and dietary flax lignans can **selectively downregulate the effects of ERα** (inflammatory, proliferative and excitatory) **in favour of ERβ** (anti-inflammatory, antiproliferative, inhibitory).[16,21,25–28]

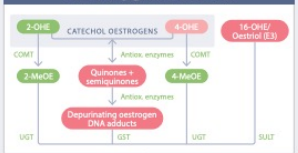
Restoring Oestrogen Balance[1,20,25,29–40]

1 Phase I: Hydroxylation (P450)



Clinical Target	Key modulators
↓ Aromatase (CYP- 19) activity to ↓ over production of oestrone (E1) & oestradiol (E2) = main oestrogen pool	**Inhibitors:** Rosemary, resveratrol, green tea, milk thistle, turmeric, DIM, zinc, dietary flax
↑ CYP-1A1/2 activity to ↑ 2-OHE which is antiproliferative, stable & quickly methylated	**Enhancers:** Resveratrol, rosemary, green tea, iodine, DIM, broccoli & radish sprouts, GSH, dietary flax
↓ CYP-1B1 activity to ↓ 4-OHE which is unstable & methylated slowly, but has less binding affinity for ERs	**Inhibitors:** Resveratrol, broccoli & radish sprouts, green tea, citrus, iodine, quercetin, rutin, luteolin
↓ CYP-3A4/5 activity to ↓ 16-OHE which is highly proliferative, inflammatory & has a high affinity for ERs	**Inhibitors:** DIM, broccoli & radish sprouts, green tea, resveratrol, rosemary, selenium

2 Phase II: Conjugation (Transferases)



Clinical Targets	Key modulators
Support optimal transferase function & gene modulation (COMT, UGT, GST, SULT) with cofactors & adequate antioxidants	**Methylation (COMT) enhancers:** SAMe, methionine, folate, B6, B12, broccoli & radish sprouts, rosemary, choline
2-OHE & 4-OHE (catechol oestrogens) are conjugated by all transferases but predominantly methylation into 2-MeOE & 4-MeOE	**Glucuronidation (UGT) enhancers:** resveratrol, silymarin, rosemary, green tea, calcium D-glucarate
Some are oxidised into quinones and semiquinones, & then neutralised by glutathionation	**Glutathionation (GST) enhancers:** resveratrol, silymarin, rosemary, turmeric, green tea, selenium, broccoli & radish sprouts, GSH, NAC
16-OHE/Oestriol (E3) is conjugated predominantly by sulfation and glucuronidation	**Sulphation (SULT) enhancers:** resveratrol, silymarin, molybdenum, NAC, broccoli & radish sprouts, sulfur aminos

3 Phase III: Elimination



Clinical Target	Key modulators
↓ dysbiosis & β-glucuronidase production to ↓ deconjugation of neutralised metabolites during enterohepatic circulation	**Inhibitors:** calcium D-glucarate, milk thistle, *L.rhamnosus*, broccoli & radish sprouts, selective antimicrobials, dietary flax, fibre
Support urinary and bile excretion of conjugated metabolites	**Enhancers:** Milk thistle, broccoli & radish sprouts, alkalising minerals, molybdenum, B3

Beyond The Hazard Zone

Nutrigenomics

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Why Nutrigenomics?

Heritability 30–80% — yet the same hormone levels produce wildly different responses between women

The answer is in the genome: SNPs in neurotransmitter synthesis, degradation and receptor sensitivity explain much of this individual variability

Key targets: ESR1, GABRA2, 5-HT1A, FKBP5, FAAH, CNR1+ NT's — the genetic levers that determine whether a woman tips into PMDD

Not deterministic — directional: variants shift thresholds; nutrition, sleep and lifestyle modulate expression

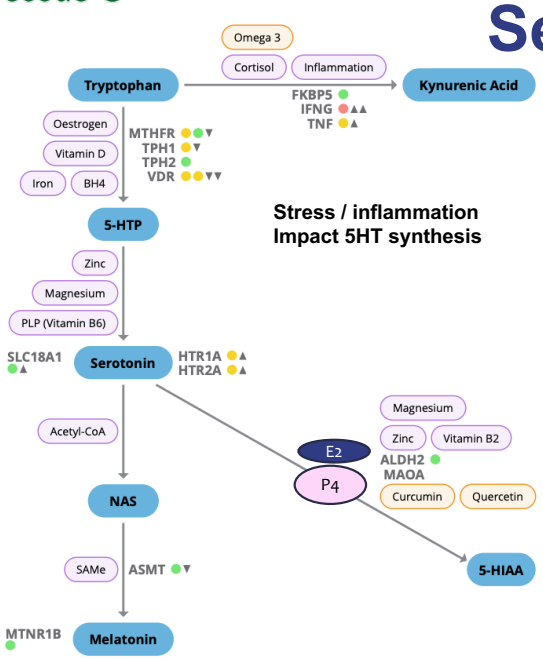
Same symptom, different mechanism, different intervention — nutrigenomic testing identifies which pathway to target first

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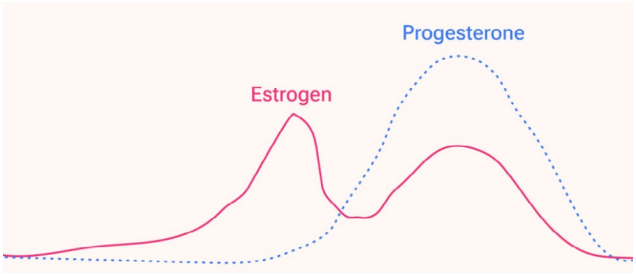
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Lifecode GX ®

Serotonin



Stress / inflammation
Impact 5HT synthesis



Drop E2 impact synthesis
Drop E2 +rise P4 faster metabolism

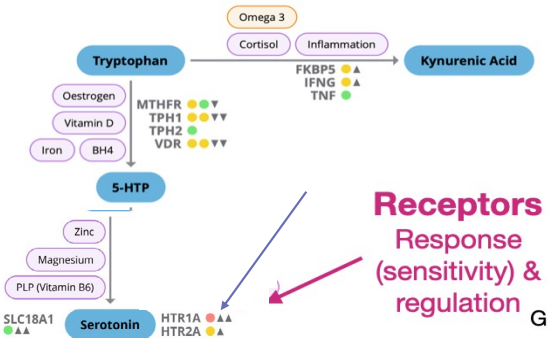
inducer inhibitor

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Serotonin

Lifecode GX ®



Receptors
Response (sensitivity) & regulation

HTR1A (5-HT1A)

Inhibits 5-HT release (presynaptic)
'Brake'/ 'protection' from excess

BUT

SNP rs6295 C(-1019)G
G allele (more/ **too much inhibition**).
'Low serotonin'
Anger. Aggression. Anxiety. Appetite. Neuroticism. OCD. **PMDD**

credit Emma Beswick and Lifecode Gx®

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Serotonin Support

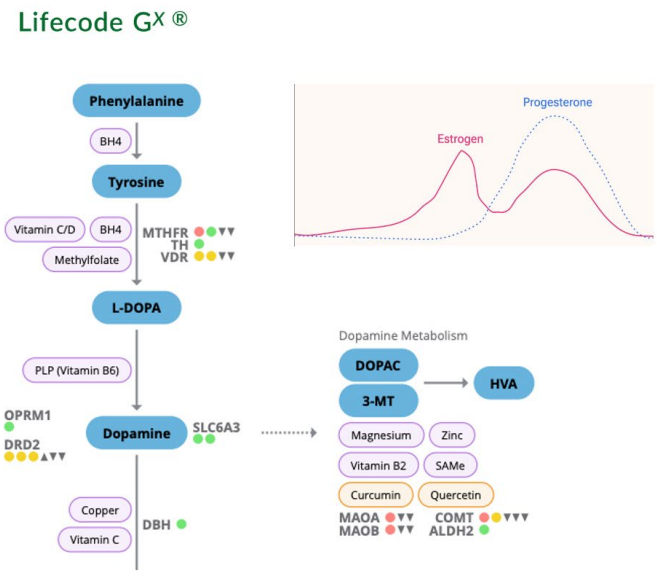
Nutrients	Herbs	Lifestyle
Tryptophan - white meat, almonds, dairy Iron, Zinc, B6 (PLP) Vitamin D Quercetin, Curcumin (MAO) Omega-3/EFAs (anti-inflammatory)	St John's Wort (*Hypericum perforatum*) - **5-HT Receptor** regulation Saffron - Similar to SSRI's inhibit the reuptake of 5HT	Manage cortisol/Inflammation (FKBP5) Sunlight (VDR)

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Dopaminergic regulation & impulse support

- Cofactors to synthesise - E2 induces tyrosine hydroxylase
- Receptor and transporter vulnerability – E2 downregulates DAT, reduced uptake
- Cofactors for optimal metabolism and biotransformation

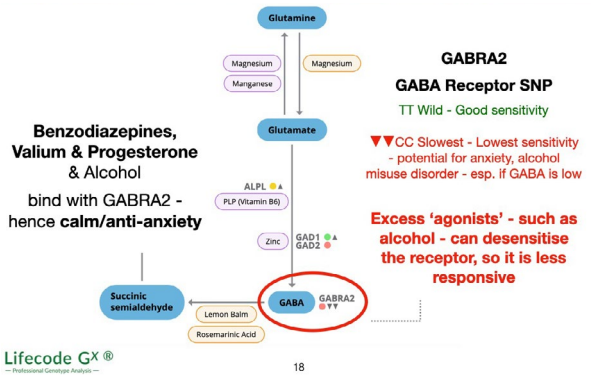


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GABA Pathway SNPs:

The Genetic Architecture of the ALLO Paradox



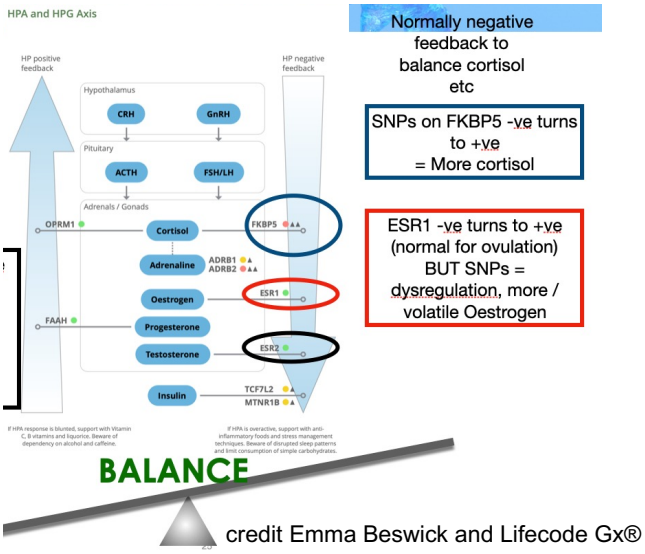
Nutritional targets:
B6/PLP, magnesium, zinc, glutamine, taurine, glycine, theanine, lemon balm, rosemarinic acid

- **GABRA2**:TT wild = good receptor sensitivity; **CC variant = lowest sensitivity** → anxiety, vulnereability to alcohol misuse
- **GAD1/GAD2** converts glutamate → GABA; variants ↓ conversion efficiency → **glutamate excess + GABA deficiency** simultaneously
- **SLC6A11** (GABA transporter): governs reuptake efficiency — variants alter how quickly GABA is cleared from the synapse
- **The PMDD bridge:** low baseline GABAergic tone + GABA-A subunit plasticity failure during ALLO surge = **paradoxical dysphoric response** — genetics explains who tips and why
- **Critical warning:** excess GABA-A agonists — alcohol, benzodiazepines — desensitise the receptor over time, making it less responsive to both therapeutic and endogenous signals including ALLO

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HPA Axis & Stress Resilience SNPs:

When the Stress System Can't Switch Off



Normally negative feedback to balance cortisol etc

SNPs on FKBP5 -ve turns to +ve = More cortisol

ESR1 -ve turns to +ve (normal for ovulation) BUT SNPs = dysregulation, more / volatile Oestrogen

credit Emma Beswick and Lifecode Gx®

- **FKBP5:** normally provides negative feedback to balance cortisol — **SNP variants flip this to positive feedback = more cortisol, prolonged stress response, trauma vulnerability**
- **ESR1 SNPs:** normally negative feedback on HPA → SNP = dysregulation, more volatile oestrogen signalling — directly amplifies HPA-HPG crosstalk
- **ADRB1/2:** ▲▲ greater fight-or-flight response to adrenaline → amplified physiological stress effects
- **TCF7L2 :** dysregulated blood sugar → **circadian disruption as a biological stressor** feeding directly back into HPA activation
- **Epigenetics:** trauma and ACEs alter FKBP5 methylation — creating an acquired vulnerability that mimics genetic risk

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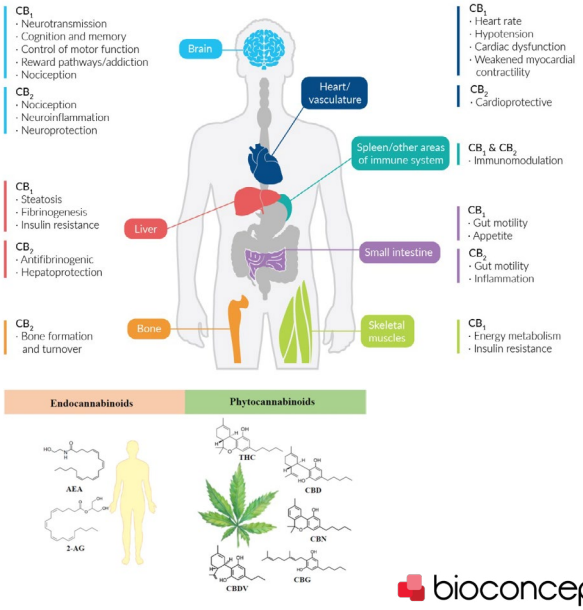
The Endocannabinoid System:
The Missing Piece in PMDD

AEA and 2-AG endogenous ligands

CB1 - **central nervous system**
CB2 - primarily expressed in periphery

also for **TRPV1** (transient receptor potential vanilloid type-1) channels

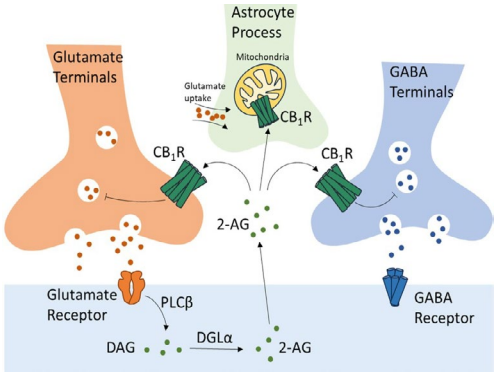
FAAH – break down AEA

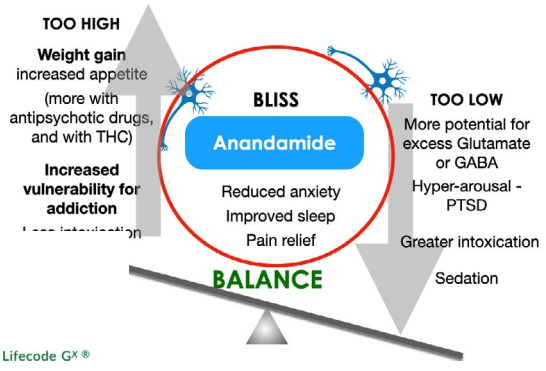


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The Endocannabinoid System

AEA (anandamide) = the bliss molecule:
reduces anxiety, improves sleep, relieves pain — when in balance via CB1 receptors





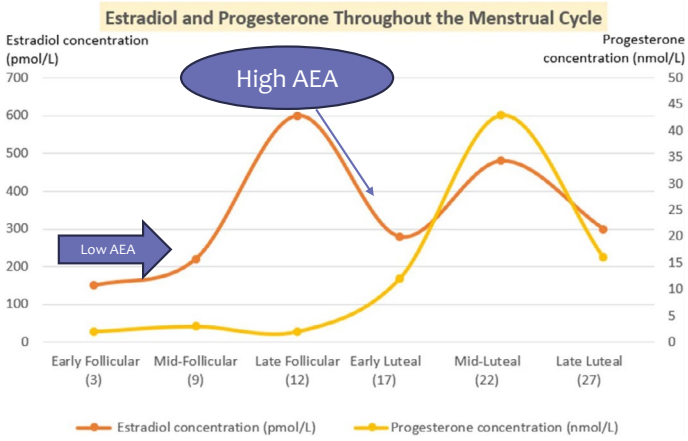
credit Emma Beswick and Lifecode Gx®

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The Endocannabinoid System – E2 & FAAH

Oestrogen inhibits FAAH — meaning E2 naturally raises AEA levels; when E2 falls perimenstrually, AEA falls with it

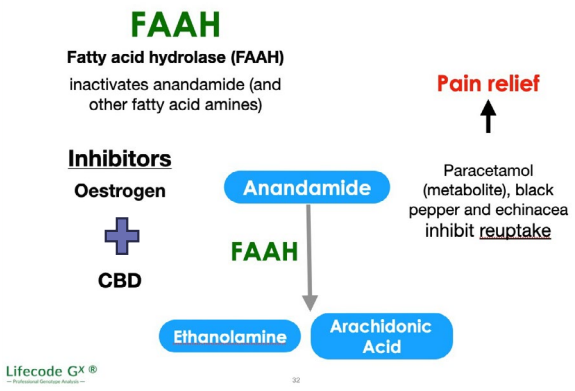
AEA peaks at ovulation then declines across the luteal phase — creating a cyclical ECS vulnerability that mirrors and compounds the ALLO/GABA vulnerability



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ECS SNPs: FAAH



FAAH (fatty acid amide hydrolase — degrades AEA):

CC normal: faster AEA clearance → more sensitive eCBS, but anxiety risk when AEA is low

CA slower clearance: excess eCB → desensitisation

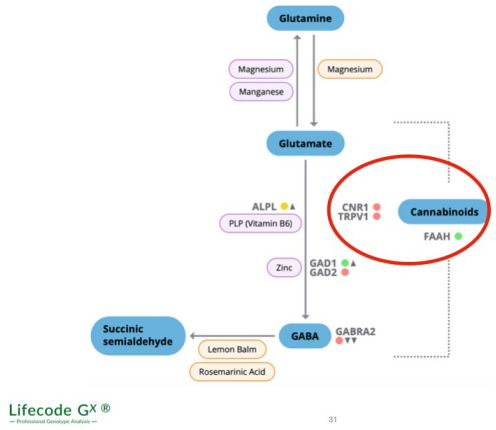
AA slowest clearance: excess eCB highest AEA but greatest desensitisation → addiction, obesity risk

FAAH inhibitors: oestrogen, CBD

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ECS SNPs: CNR1



CNR1 is the gene that codes for cannabinoid receptor 1

activation of CB1 **regulates glutamate and GABA by inhibiting their ongoing release**

rs1049353 (T/C)

C (wild) - relatively low activity ✗

↓

Less inhibition of GABA and Glutamate

T (variance) better regulation ✓

↓

reduced development of stress, of depression and anhedonia when exposed to stressful life events/abuse

credit Emma Beswick and Lifecode Gx®

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ECS SNPs: TRPV1 - pain, inflammation and mood

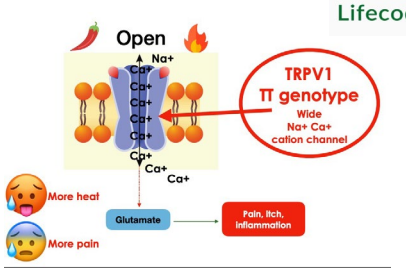
Transient Receptor Potential Cation Channel subfamily V member 1
Capsaicin Receptor
Vanniloid Receptor 1

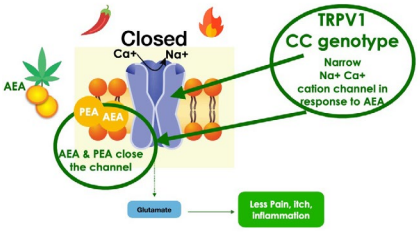
TRPV1 is involved in
Detection and regulation of **body temperature**

Transmission and modulation of **pain in response to noxious substances, inflammation** via nociceptor (nerve cell) activation

Sensitivity to capsaicin (in chilli peppers) and other **spicy foods** (pepper, garlic, mustard and wasabi) and to salt and sugar

Lifecode GX ®





credit Emma Beswick and Lifecode Gx®

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Sleep

Sleep: The Overlooked Mechanistic Driver

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Sleep Loss, Cortisol & the HPA Amplification Loop

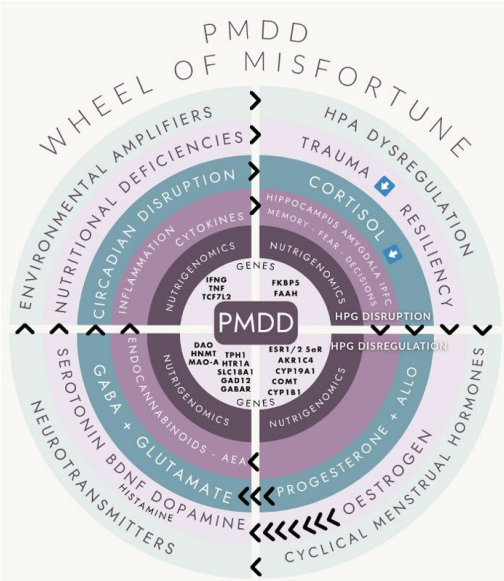
- **GABA-A and sleep are the same system:** the receptor governing ALLO sensitivity in PMDD is the same receptor governing sleep onset and slow-wave sleep architecture — they cannot be separated
- **FAAH high-activity variants → low AEA → poor sleep onset**, increased overnight pain sensitivity, reduced CB1-mediated sleep promotion — genetics determines the baseline
- **FKBP5:** prolonged cortisol response = a genetically sensitised sleep-cortisol interface that dysregulates HPA overnight
- **Sleep deprivation amplifies amygdala reactivity and reduces PFC top-down regulation** — recreating the exact neurobiological signature of PMDD in a healthy brain
- **Blunted nocturnal cortisol** (seen in high-ALLO PMDD women) → non-restorative sleep → elevated evening cortisol next day → worse sleep → repeat

Sleep is not a symptom — it is a mechanism. Treating sleep is treating PMDD at the level of it's neurobiology

Leproult & Van Cauter (2010). International Journal of Endocrinology; Hantsoo & Epperson (2020) Neurobiol. Stress 12:100213

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The Vicious Cycle: Everything Connects



Chronic stress + trauma (FKBP5, NR3C1) → prolonged cortisol → suppresses ALLO synthesis → GABA-A instability

GABA-A instability → paradoxical ALLO response → luteal hyperarousal → disrupted sleep architecture
Poor sleep (FAAH, CNR1) → ↓ AEA → ↑ HPA activation → neuroinflammation (↑ IL-6, CRP)

Neuroinflammation → luteal CRP surge (20–23% with 10-fold P4 rise) → amplified affective symptoms → worsened GABA-A receptor plasticity

Hormonal flux (ESR1, GABRA2, 5-HT1A) → neurotransmitter dysregulation → ↓ serotonin, ↓ dopamine, ↓ AEA simultaneously

Neurotransmitter depletion → amplified stress reactivity → poor sleep → HPA reactivation →

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Clinical Decision Framework: From Mechanism to Intervention

Step 1 — Establish the pattern: prospective daily tracking ≥2 cycles.

Step 2 — Map to DASH-MC dimension: midluteal (ALLO/GABA) → perimenstrual (E2/DA) →periovulatory (E2 surge/DA) → or mixed

Step 3 — Run targeted nutrigenomics

Step 4 — Assess the amplifiers: sleep quality, trauma history, inflammatory load, circadian regularity, cortisol pattern (CAR)

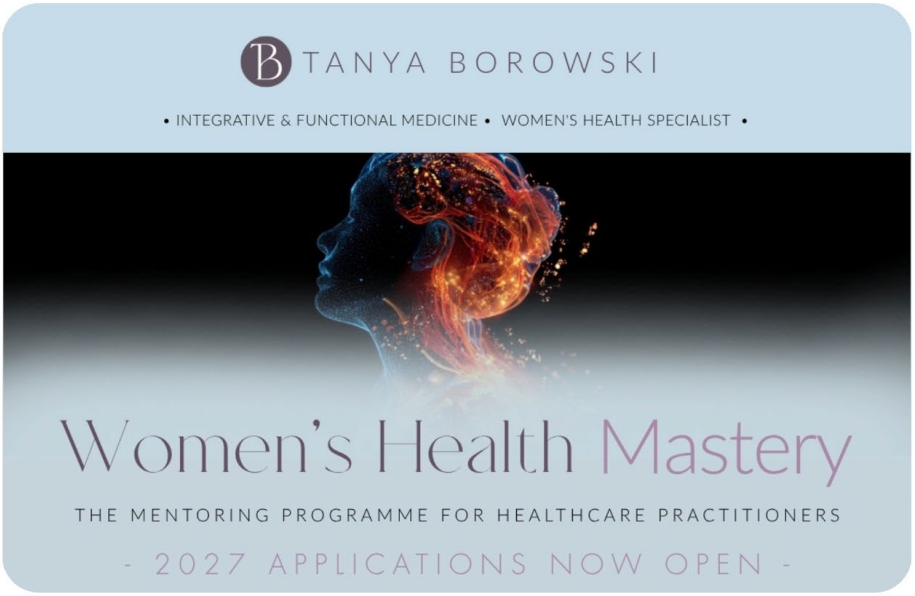
Pause – Consider – Reflect

Step 5 — Curate a personalized, adaptable, phased approach



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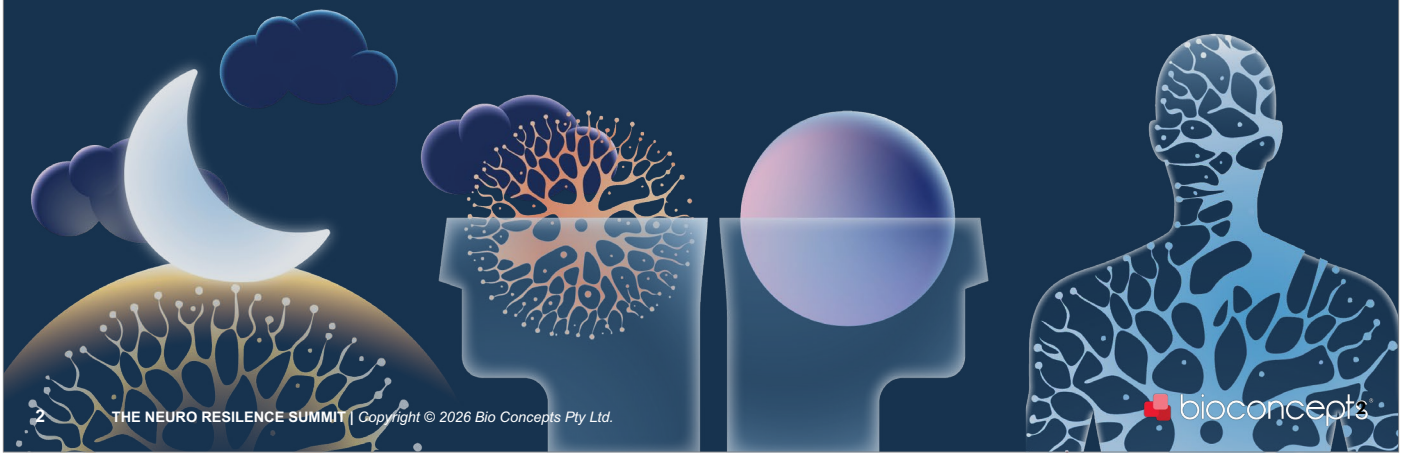
FEATURING

DR FRANK GOLIK
TANYA BOROWSKI

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Comfort is Overrated

Curiosity, Capacity, and the Courage to Be Better



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KEYNOTE SPEAKER

Pettina Stanghon

Bachelor of Applied Social Science,
NLP Master Practitioner, Clinical Hypnotherapy,
Marriage/Relational Counsellor and Mediator



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OUTLINE

1. The Comfort Crisis
2. Certainty as avoidance
3. Capacity, not comfort
4. Curiosity, not catastrophe
5. The Therapeutic Alliance
6. Prescribing hard things
7. Practical Questions
8. Psychological Adulthood

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Every person who walks into my clinic wants the same thing.

Not health.
Not change.
Comfort.

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"If I'm uncomfortable, something must be wrong."

The most dangerous belief encounter in a treatment room -

More dangerous than the diagnosis, the substance, or the story the client has told themselves for 20 years.

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And everyone in this room is seeing it

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What It Looks Like

Still waiting for someone to remove ***discomfort.***

- 6 Antidepressants
- 3 Psychologists
- 4 Podcasts
- 2 Podcasts

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Two Patients One Pattern

You know these clients.

They're in your clinics right now.

FOUR RELAPSES	FULLY COMPLIANT
Not because treatment failed - because the moment acute discomfort eased, he stopped.	Supplement compliant. Dietary compliant. Attends every appointment.
He felt better. He mistook that for being better.	Not moving - because compliance is comfortable. Change is not.

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The Uncomfortable Truth

What this talk is about:
Not what's wrong with your clients...
What's available when we stop making comfort the goal.



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The Certainty Pandemic



Addiction is a *mechanism* before it's a diagnosis.

The brains pursuit of relief from *discomfort* through a
repeatable, predictable behaviour.

The substance is just one version of that mechanism.

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The most widespread addiction in our culture has **nothing to do with substances.**

It's certainty.

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The Brain is a Prediction Machine

It functions to minimise surprise

– because to a nervous system shaped by evolution, surprise registers as a threat.

Confirms Belief

Dopaminergic reward.
Feels, briefly, safe.

Contradicts Belief

Same circuitry as physical danger activates.

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Friston, K. (2010). The Free Energy Principle: a unified brain theory? Nature Reviews Neuroscience, 11(2), 127 –138.

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Google didn't create certainty.
It industrialised it.

Social media isn't selling information.

It's selling **emotional confirmation.**

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The Clinical Question Changes

OLD QUESTION

How do I give my client better information?

NEW QUESTION

How do I make my client uncertain enough to be curious?

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Capacity. Not Comfort. Curiosity. Not Catastrophe.

The 4 C's Framework.

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Two Strategies. Two Outcomes.

COMFORT-SEEKING

Subtraction Strategy

- Remove the discomfort
- Reduce the symptoms
- Lower the pain threshold

⟶ **Fragility**

CAPACITY-BUILDING

Addition Strategy

- What can they tolerate?
- What can they move through?
- What can they recover from?

⟶ **Resilience**

McEwen, B.S. (1998). Stress, adaptation, and disease: Allostasis and allostatic load. Annals of the New York Academy of Sciences, 840(1), 33–44.

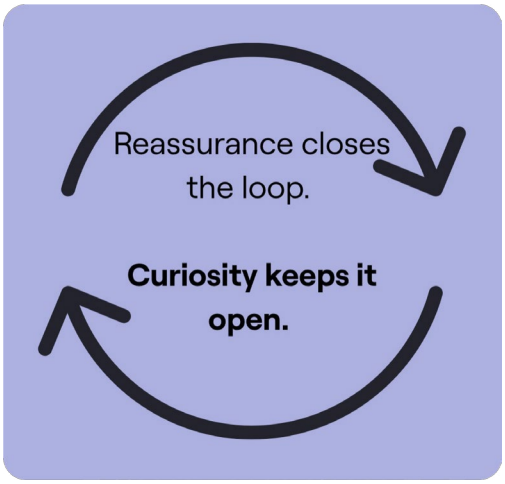
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The Antidote to Catastrophe

"Isn't that interesting?"

Three words I use more than anything in my clinic.



Gruber, M.J., Gelman, B.D., & Ranganath, C. (2014). States of curiosity modulate hippocampus-dependent learning. Neuron, 84(2), 486–496.

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The strongest predictor of change is not protocol.

It's the relationship.

The Therapeutic Alliance

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Fifty years of outcome research — across every modality — shows the **alliance accounts** for more variance in treatment outcomes than any specific technique.



Lettieri, G. (2026). Integrating neural, physiological, and interoceptive measures in social interaction. Frontiers in Neuroscience, 20, 1771470.

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Connection isn't a soft skill

It is the practice of regulation, curiosity, and accountability in real time.

Both people have skin in the game — that is when the dial moves.



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The therapeutic alliance has a physiology

Genuine connection is the meeting of two brain-body systems.

- Heart rate.
- Respiration.
- Autonomic arousal — measurably coupled, in real time.

*The **quality** of that synchrony predicts whether the interaction actually moves anything.*



Lettieri, G. (2026). Integrating neural, physiological, and interoceptive measures in social interaction. Frontiers in Neuroscience, 20, 1771470.

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Sarah

Third relapse. Highly functional.

She could explain her own patterns in extraordinary detail — every trigger, every root, every mechanism.

She was certain about why she drank.

That certainty was the thing keeping her stuck.

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"You know everything about why you drink.

And you're **still drinking."**

*"So, let's agree — **knowing isn't the problem**, and it **isn't the solution**."*

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The Comfort Transaction

Patient brings discomfort. → Practitioner removes it. → Client dependent on relief – nothing built.

The most important thing you can offer isn't relief.
***It's the experience of being capable** — and that only happens inside a relationship strong enough to ask hard things.*

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Feeling better is not the same as being better

Prescribing Hard Things

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Sleep beats supplements. Every time.

Chronic sleep deprivation elevates cortisol, accelerates neuroinflammation, and impairs prefrontal cortex function — the exact capacity you're asking your client to use.

How many of us prescribe sleep —
and then answer emails at 11:30 at night?



Walker, M. (2017). Why We Sleep: Unlocking the Power of Sleep and Dreams. Scribner.

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The quality of relationships

80 Years of data.

The Harvard Study of Adult Development – the longest-running study of adult health in history.

Waldinger, R.J., & Schulz, M.S. (2023). The Good Life. Simon & Schuster.

The single strongest predictor of late-life wellbeing isn't cholesterol, BMI, or exercise.

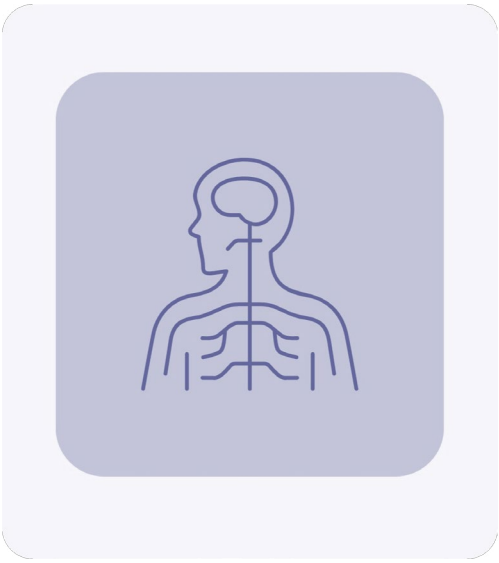
It was the quality of relationships.

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Consumption

We're asking people to regulate their nervous system

While carrying a device designed to dysregulate it.



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Am I prescribing hard things?

Or comfortable things?

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Practical Tools

Four questions that interrupt comfort seeking

1. "What would you have to stop believing for this to change?"
2. "What are you getting from staying where you are?"
3. "What does staying the same cost you that you haven't said out loud yet?"
4. "If you already knew you could handle this, what would you do differently?"

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Patterns to Notice

Explains it better than they manage it.
Sophisticated insight as avoidance.

Compliant, but not changing.
Compliance is comfortable.
Compliance is not growth.

Catastrophises new information.
Not dramatic – uncertain. Redirect to curiosity.

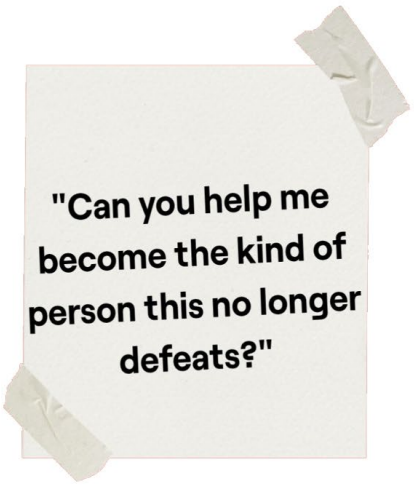
Returns unchanged, expects a new prescription.
The discomfort eased. They stopped.

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"Can you make this stop?"

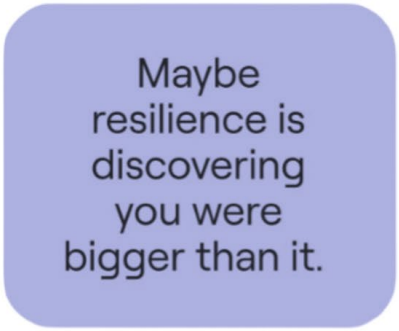
Or



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Maybe resilience isn't the absence of suffering.



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Capacity. Not Comfort.

Curiosity. Not Catastrophe.

Developing the person.
Not alleviating the symptom.

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Q&A
Q&A
Q&A
Q&A

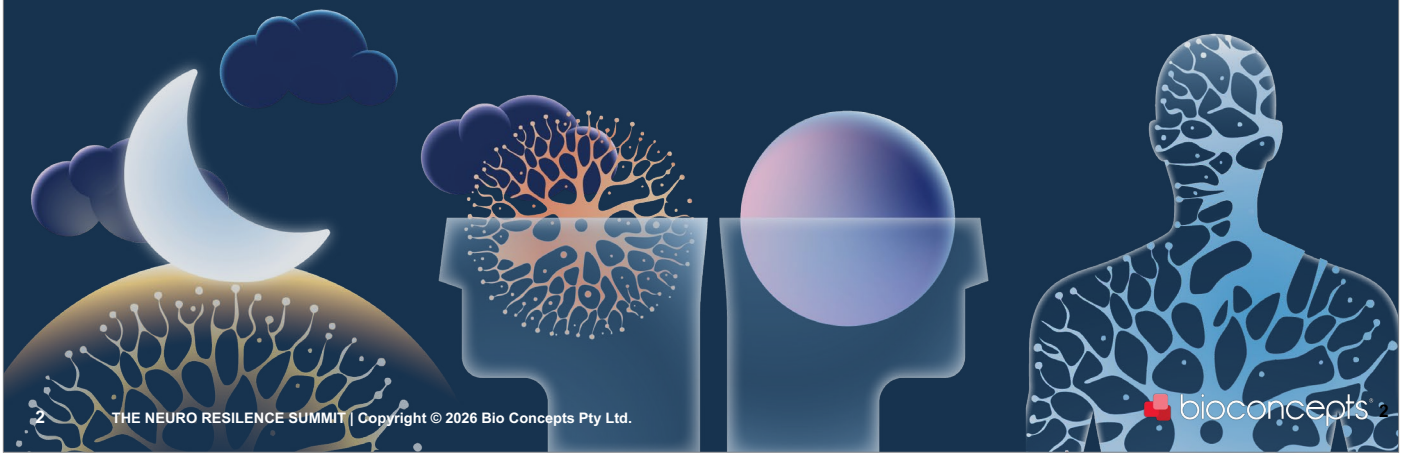
FEATURING

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The Socially Inflamed Brain

How Social Stress, Isolation and Defeat Drive Neuroinflammation - and how we can help to restore neuro-resilience



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SPEAKER

Sue Camp

MSC, B.SocSci (Hons), FMCP, mBANT,
rCNHC Registered Nutritional Therapist
Certified Functional Medicine Practitioner



- Practice in three London-based clinics
- Specialise in gut health, hormone health and optimising health and performance
- Corporate consultant
- Part of a Nutraceutical Clinical Education team
- Completing an International Masters in Clinical Psychoneuroimmunology (cPNI) with the Pruimboom Institute
- Avid Park runner and enthusiastic kayaker
- Enjoy the balance between hot yoga and dark chocolate!

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Two Patients

Which patient is at greater **risk of neuroinflammation, cognitive decline and chronic disease?**

Patient A

- 62 years old
- Similar diet
- Similar exercise
- Similar BMI
- Similar routine blood tests

Patient B

- 62 years old
- Similar diet
- Similar exercise
- Similar BMI
- Similar routine blood tests

Has lived with profound loneliness for the past five years

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Learning outcomes

- Explain how Social Associated Molecular Patterns (SAMPs), including loneliness, social isolation, chronic stress and social defeat, contribute to neuroinflammation through immune-brain communication
- Understand the key neuroimmune mechanisms linking social stress to impaired mood, poor sleep, cognition and resilience, including microglial activation, inflammatory signalling, mitochondrial dysfunction and altered glutamate/GABA balance
- Identify the patient history, clinical signs and functional biomarkers that may indicate socially driven neuroinflammation in practice
- Apply evidence-based functional medicine and cPNI strategies including nutrition, lifestyle medicine and targeted nutraceuticals to reduce neuroinflammation and improve neuro-resilience

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Today's Journey

Evolution
Why did the social brain evolve?

Threat
What happens when connection is lost?

Biology
How does isolation become neuroinflammation?

Assessment
How do we identify it in clinical practice?

Restoration
How do we rebuild resilience?

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Our Brains Evolved for Connection

Examining evolutionary traits across primate species they found four characteristics evolved together:

- **Larger brains**
- **Larger social groups**
- **Longer lifespan**
- **Greater reliance on social learning**

These traits did not evolve independently: they **co-evolved.**

Our brain requires other brains.

Street, S.E. Et al., *Coevolution of cultural intelligence, extended life history, sociality, and brain size in primates*, Proc. Natl. Acad. Sci. U.S.A. 114 (30) 7908-7914, https://doi.org/10.1073/pnas.1620734114 (2017).



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The importance of Social Learning

Intelligence is not only inside the individual.

It exists between people.

Every generation inherits **knowledge, skills, culture, language, technology, medicine.**

All through social learning.



Culture as a mechanism increases survival by acting as a ***"cognitive buffer"*** against environmental challenges.

Longer lifespans provide more opportunity to acquire, use and transmit socially learned knowledge.

Street, S.E. Et al., *Coevolution of cultural intelligence, extended life history, sociality, and brain size in primates*, Proc. Natl. Acad. Sci. U.S.A. 114 (30) 7908-7914, https://doi.org/10.1073/pnas.1620734114 (2017).

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What happens when the brain loses the very environment it evolved to expect?

Isolation removes an evolutionarily expected input

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Social Isolation

- Social connection is an evolutionarily conserved biological requirement
- When that input is lost, the consequences extend far beyond mood - they alter neuroimmune signaling, excitatory-inhibitory balance and inflammatory biology
- Social isolation is not simply a psychological stressor
- It represents a violation of the biological environment in which our neuroimmune system evolved



Image credit: Pexels: Adnan Kale

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Associated Molecular Patterns (AMPs)

Charles Janeway (1989)

- Demonstrated the immune system identifies pathogen-associated molecular patterns (PAMPs) via germline encoded pattern recognition receptors (PRRs)
- Triggers anti-infective defence mechanisms
- Classical paradigm founded on ‘non-self’ recognition

Polly Matzinger (1994)

- The fundamental impetus for immune activation is not solely the discrimination between self and non-self, but rather the detection of danger signals, encompassing bacterial toxins, viruses, rapidly proliferating tumour cells, and non-programmed (necrotic) cell death

Walter Land (2003)

- Concept of "damage-associated molecular patterns" (DAMPs) to differentiate endogenous danger signals from traditional pathogen-derived signals

Lin H, Xiong W, Fu L, Yi J, Yang J. *Damage-associated molecular patterns (DAMPs) in diseases: implications for therapy*. Mol Biomed. 2025 Aug 29;6(1):60. doi: 10.1186/s43556-025-00305-3. PMID: 40877572; PMCID: PMC12394712.

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Introducing the concept of SAMPs:

Social Associated Molecular Patterns

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What does the literature say?

Neuropsychopharmacology REVIEWS (2017) 42, 46–61
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46 www.neuropsychopharmacologyreviews.org

REVIEW

Repeated Social Defeat, Neuroinflammation, and Behavior: Monocytes Carry the Signal

Michael D Weber*[,1,2,3], Jonathan P Godbout[2,3,4] and John F Sheridan[1,2,3]

[1]Division of Biosciences, The Ohio State University, Columbus, OH, USA; [2]Center for Brain and Spinal Cord Repair, The Ohio State University, Columbus, OH, USA; [3]Institute for Behavioral Medicine Research, The Ohio State University, Columbus, OH, USA; [4]Department of Neuroscience, The Ohio State University, Columbus, OH, USA

Loneliness and Social Isolation as Risk Factors for Mortality: A Meta-Analytic Review

Julianne Holt-Lunstad[1], Timothy B. Smith[2], Mark Baker[1], Tyler Harris[1], and David Stephenson[1]

[1]Department of Psychology and [2]Department of Counseling Psychology, Brigham Young University

RESEARCH ARTICLE

Association of loneliness with all-cause mortality: A meta-analysis

Laura Alejandra Rico-Uribe[1,2,3], Francisco Félix Caballero[1,2,3], Natalia Martín-María[1,2,3], María Cabello[1,2,3], José Luis Ayuso-Mateos[1,2,3], Marta Miret[1,2,3]*

1 Department of Psychiatry, Universidad Autónoma de Madrid, Madrid, Spain, 2 Instituto de Salud Carlos III, Centro de Investigación Biomédica en Red de Salud Mental, CIBERSAM, Madrid, Spain, 3 Department of Psychiatry, Hospital Universitario de La Princesa, Instituto de Investigación Sanitaria Princesa (IIS-Princesa), Madrid, Spain

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Defining SAMPs

Social isolation

Lacking a sense of belonging socially; lack real or perceived social interactions either due to a physical lack of people with whom to engage or through a lack of fulfilling and quality relationships.

Loneliness

Defined as a distressing feeling that accompanies the perception that one's social needs are not being met by the quantity or especially the quality of one's social relationships.

De Puit JK, Challinor KL. *Critical Review of Neurobiological Evidence for Relationships Between Social Isolation, Loneliness and the Risk of Developing of Alzheimer's Disease: A New Model.* J Aging Res. 2025 Jul 2;2025:9924448.
doi: 10.1155/jare/9924448. PMID: 40636676; PMCID: PMC12240659.
Hawkley LC, Cacioppo JT. *Loneliness matters: a theoretical and empirical review of consequences and mechanisms.* Ann Behav Med. 2010 Oct;40(2):218-27. doi: 10.1007/s12160-010-9210-8. PMID: 20652462; PMCID: PMC3874845.

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Social Isolation = Biological Threat

From an evolutionary perspective, perceived social isolation signals increased vulnerability.

For social mammals, separation from the group historically meant:

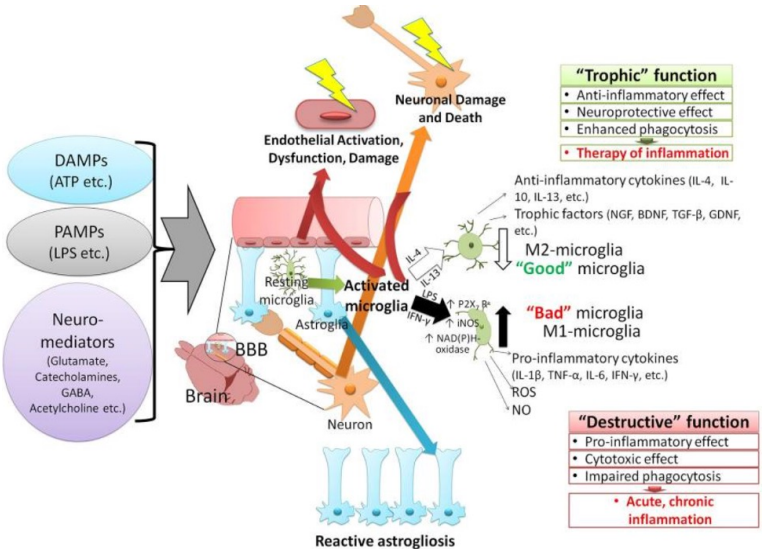
- Greater risk of predation
- Reduced protection
- Loss of thermoregulatory support
- Reduced access to food and shared resources
- Reduced reproductive success

The brain shifts into a state of heightened social threat vigilance, characterised by greater amygdala reactivity and activation of hypothalamic stress pathways.

Cacioppo JT, Cacioppo S, Capitanio JP, Cole SW. *The neuroendocrinology of social isolation.* Annu Rev Psychol. 2015 Jan 3;66:733-67. doi: 10.1146/annurev-psych-010814-015240. Epub 2014 Aug 22. PMID: 25148851; PMCID: PMC5130104.

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Tohidpour A, et al., *Neuroinflammation and Infection: Molecular Mechanisms Associated with Dysfunction of Neurovascular Unit.* Front Cell Infect Microbiol. 2017 Jun 20;7:276. doi: 10.3389/fcimb.2017.00276. PMID: 28676848

DAMPs/PAMPs & Neuroinflammation

PAMPs, DAMPs and neuromodulators drive neuroinflammation by:

- Activating glial cells
- Disrupting the blood–brain barrier
- And influencing whether the CNS undergoes repair or progresses to chronic neurodegeneration

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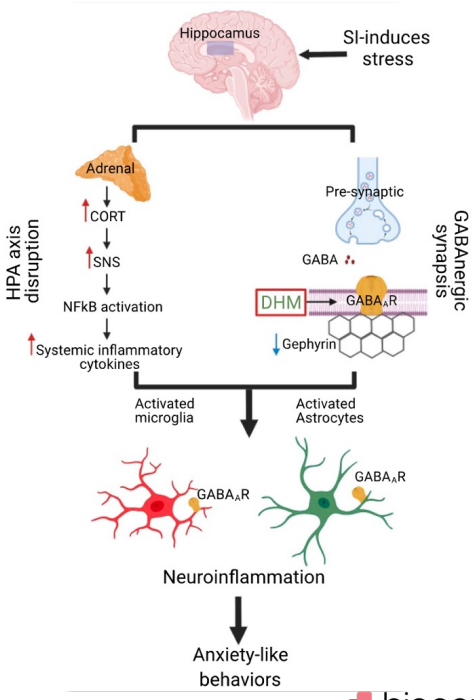
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Social-induced Neuroinflammation

Murine model

Chronic social stress produces
- Activated microglia
- Inflammatory cytokines
- Impaired neuroplasticity

Persistent glucocorticoid exposure leads to
- Glucocorticoid resistance
- Impaired negative feedback
- Persistent immune activation



Al Omran AJ, et al., *Social isolation induces neuroinflammation and microglia overactivation, while dihydromyricetin prevents and improves them.* J Neuroinflammation. 2022 Jan 4;19(1):2. doi: 10.1186/s12974-021-02368-9.

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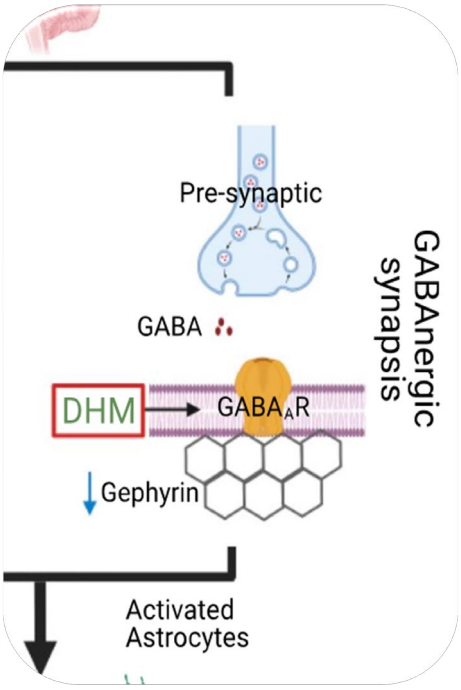
The role of GABA

Brain's principal inhibitory neurotransmitter

Gephyrin is a scaffold protein that anchors and clusters GABA-A receptors

Socially isolated mice → 40% reduction in hippocampal gephyrin expression, suggesting impaired GABAergic neurotransmission

Fewer functioning GABA-A receptors →
less inhibitory tone → greater neuronal firing → greater amygdala activity → **greater anxiety**



Al Omran AJ, et al., *Social isolation induces neuroinflammation and microglia overactivation, while dihydromyricetin prevents and improves them.* J Neuroinflammation. 2022 Jan 4;19(1):2. doi: 10.1186/s12974-021-02368-9.

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Pathways converge

Neuroinflammation itself further suppresses GABAergic transmission by:

- Reducing GAD67 (the enzyme that synthesises GABA)
- Downregulating GABA-A receptor expression
- Decreasing GABAergic neuronal density

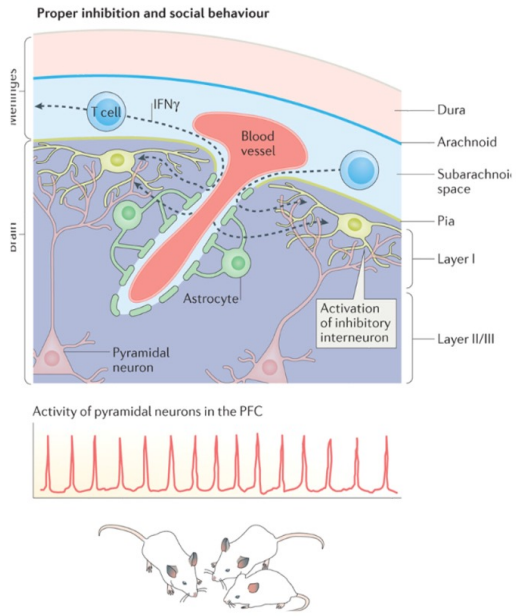
Loss of GABAergic inhibition on microglia and astrocytes removes a brake on their activation, amplifying neuroinflammation further.

The result is a self-reinforcing cycle driving anxiety-like behaviour.

Crowley T, Cryan JF, Downer EJ, O'Leary OF. Brain Behav Immun. 2016 May;54:260-277. doi: 10.1016/j.bbi.2016.02.001. Epub 2016 Feb 3. PMID: 26851553. *Inhibiting neuroinflammation: The role and therapeutic potential of GABA in neuro-immune interactions.*

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Filiano AJ, Gadani SP, Kipnis J. *How and why do T cells and their derived cytokines affect the injured and healthy brain?* Nat Rev Neurosci. 2017 Jun;18(6):375-384. doi: 10.1038/nrn.2017.39. Epub 2017 Apr 27. PMID: 28446786; PMCID: PMC5823005.

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The role of IFN-γ

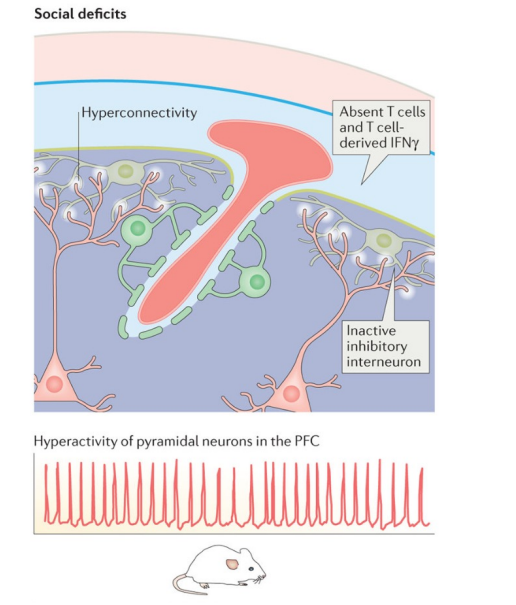
Meningeal T cells continuously release low levels of IFN-γ into the cerebrospinal fluid

This IFN-γ binds to receptors on GABAergic inhibitory interneurons within the prefrontal cortex

The result is:

- Increased tonic GABA currents
- Stronger inhibitory control
- Prevention of excessive neuronal firing
- Stable cortical connectivity
- Normal social interaction

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Filiano AJ, Gadani SP, Kipnis J. *How and why do T cells and their derived cytokines affect the injured and healthy brain?* Nat Rev Neurosci. 2017 Jun;18(6):375-384. doi: 10.1038/nrn.2017.39. Epub 2017 Apr 27. PMID: 28446786; PMCID: PMC5823005.

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The role of IFN-γ

In mice lacking IFN-γ or T cells:

- Less activation of inhibitory interneurons
- Reduced tonic GABA inhibition
- Pyramidal neurons become overactive
- Prefrontal cortex becomes hyperconnected

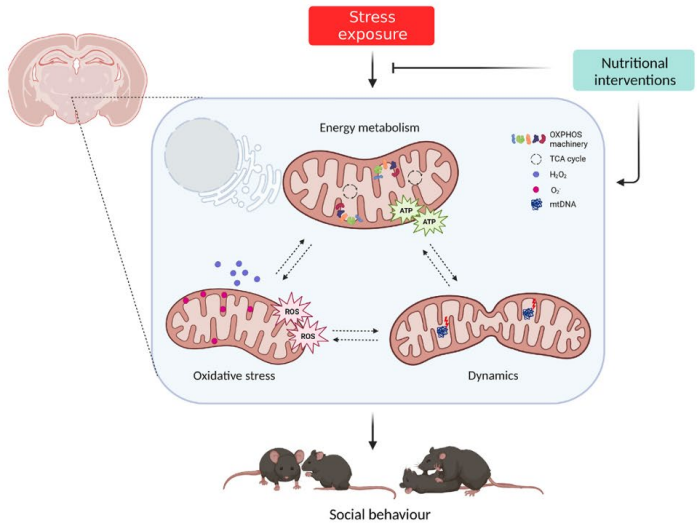
Social behaviour deteriorates

This is disinhibition - the brain loses its normal inhibitory restraint

IFN-γ maintains social behaviour by enhancing GABAergic inhibition

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Powering the social brain - mitochondria



Mitochondria are not simply ATP factories → they are signalling organelles

Mitochondrial function and dynamics are central in social behaviour and stress-induced social deficits.

Ülgen DH, Ruigrok SR, Sandi C. *Powering the social brain: Mitochondria in social behaviour.* Curr Opin Neurobiol. 2023 Apr;79:102675. doi: 10.1016/j.conb.2022.102675. Epub 2023 Jan 23. PMID: 36696841.

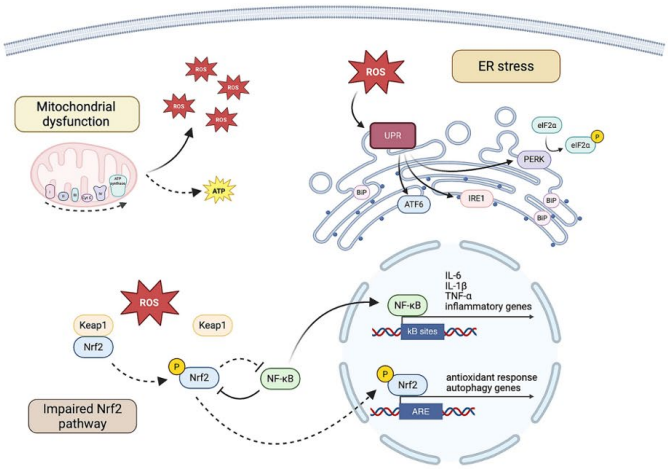
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Social Isolation & Mitochondrial function

Molecular pathways deregulated in the context of social isolation

Mitochondrial dysfunction and alterations in the respiratory chain, lead to decreased ATP production, increased Reactive Oxygen Species (ROS) generation and oxidative stress.



Qaisiya M, et al.,*Social isolation: an integrated molecular web that disrupts cellular homeostasis.* Front Neurosci. 2025 Dec 8;19:1693696. doi: 10.3389/fnins.2025.1693696.

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Social Isolation

- Social connection is an evolutionarily conserved biological requirement
- When that input is lost, the consequences extend far beyond mood - they alter neuroimmune signaling, excitatory-inhibitory balance and inflammatory biology
- Social isolation is not simply a psychological stressor
- It represents a violation of the biological environment in which our neuroimmune system evolved



Image credit: Pexels: Adnan Kale

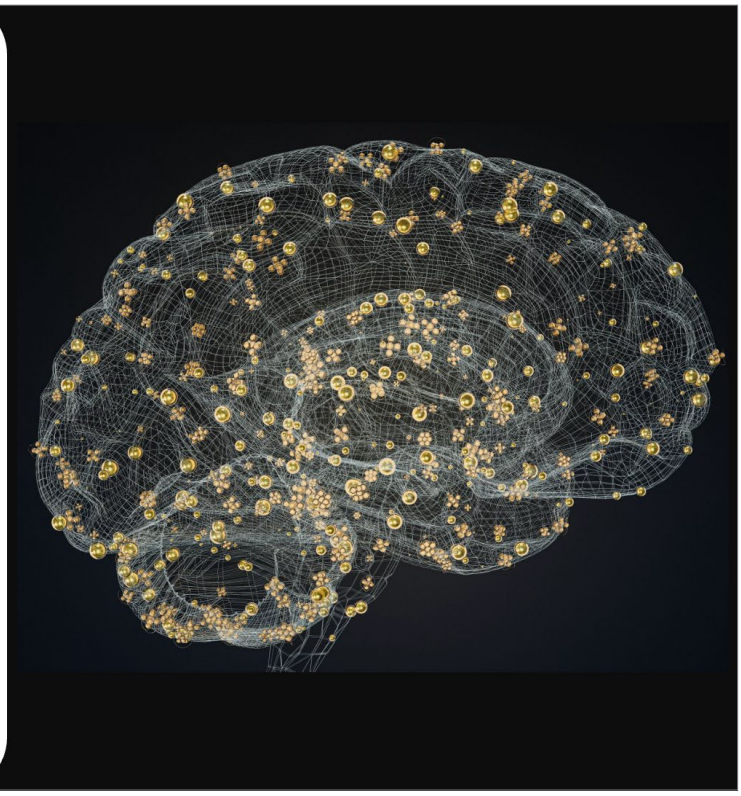
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Oxidative Stress

"The brain consumes about 20% of the bodily oxygen and yet, has relatively weak antioxidant mechanisms"

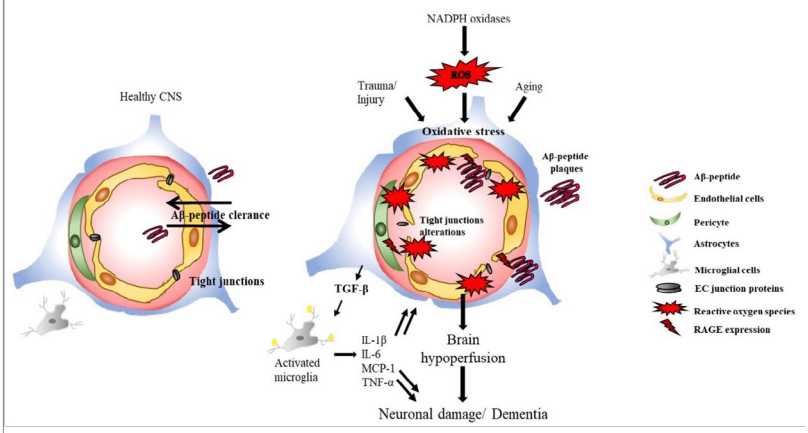
Mitochondria produce ROS as byproducts of ATP generation

Studies have reported that increased microglial activity could be an early event that leads to oxidative damage



Tarafdar A, Pula G. *The Role of NADPH Oxidases and Oxidative Stress in Neurodegenerative Disorders*. Int J Mol Sci. 2018 Nov 30;19(12):3824. doi: 10.3390/ijms19123824. PMID: 30513656; PMCID: PMC6321244.

Image credit: Unsplash Alex Shuper



NADPH Oxidases (NOX) activation

Another source of ROS, upstream of the mitochondria are the NOX enzymes – activated in the microglia and are also detected in the astrocytes and neurons.

Activated microglia and astrocytes produce high levels of ROS via NOXs, which have deleterious effects in the expression of important molecules involved in blood-brain barrier integrity.

Tarafdar A, Pula G. *The Role of NADPH Oxidases and Oxidative Stress in Neurodegenerative Disorders.* Int J Mol Sci. 2018 Nov 30;19(12):3824. doi: 10.3390/ijms19123824. PMID: 30513656; PMCID: PMC6321244.

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How does this affect GABA?

Reduced GABA → **Greater ROS** → **Increased neuroinflammation**

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Pulling it all together

- A social experience becomes a molecular signal
- That signal becomes oxidative biology through NOX
- That ultimately alters brain function and behaviour

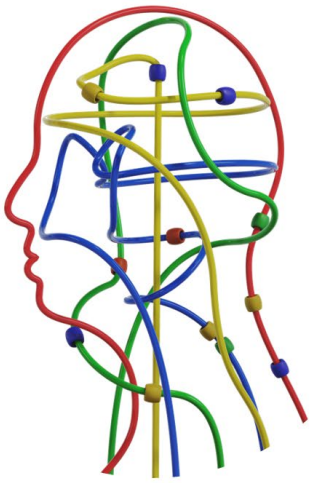


Photo by Luke Jones on Unsplash

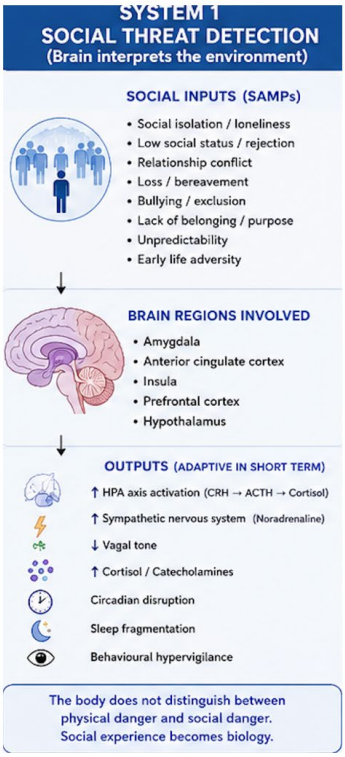
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Social signalling – the trigger

Social isolation, loneliness, rejection and low social status as Socially Associated Molecular Patterns (SAMPs).

The brain interprets the environment



Graphics generated by Chat GTP

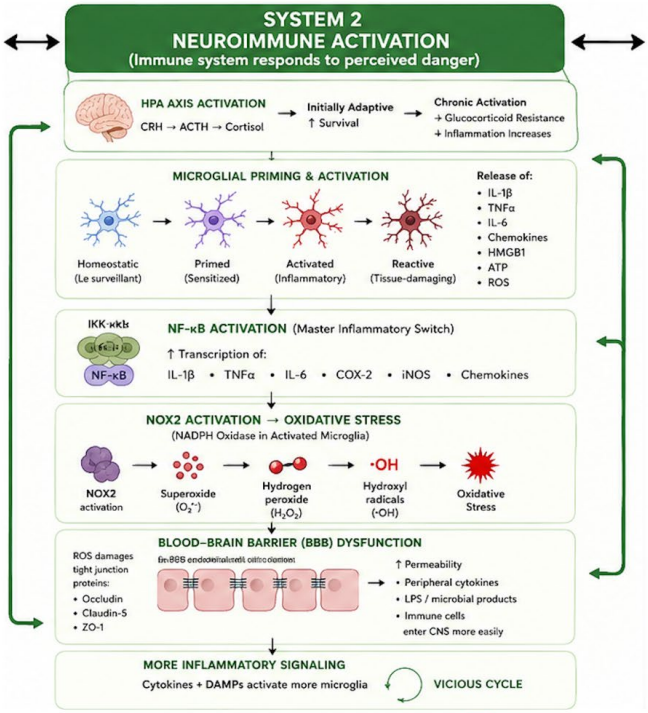
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Neuroimmune activation (the amplifier)

Immune system responds to perceived danger:

- HPA axis activation
- Microglial priming
- NF-κB signalling
- NOX-derived oxidative stress



Graphics generated by Chat GTP

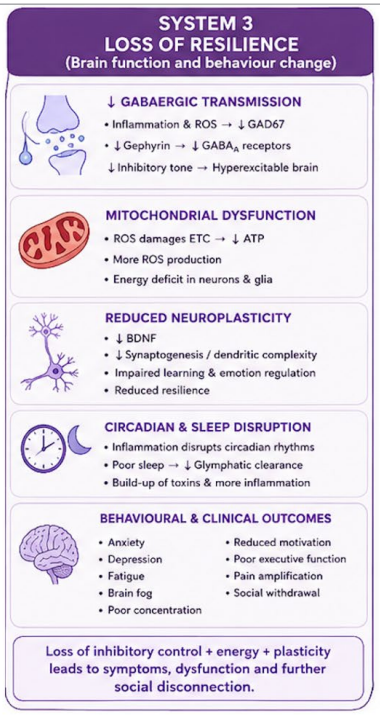
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Loss of resilience (the consequence)

- Impaired GABAergic inhibition
- Reduced synaptic plasticity and neurogenesis
- Blood-brain barrier dysfunction
- Mitochondrial impairment
- Anxiety, depression and cognitive dysfunction

Brain function changes



Graphics generated by Chat GTP)

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How do we assess SAMPs?

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Ask: UCLA Loneliness Scale

Scale:
INSTRUCTIONS: Indicate how often each of the statements below is descriptive of you.

C indicates "I often feel this way"
S indicates "I sometimes feel this way"
R indicates "I rarely feel this way"
N indicates "I never feel this way"

1.	I am unhappy doing so many things alone	O S R N
2.	I have nobody to talk to	O S R N
3.	I cannot tolerate being so alone	O S R N
4.	I lack companionship	O S R N
5.	I feel as if nobody really understands me	O S R N
6.	I find myself waiting for people to call or write	O S R N
7.	There is no one I can turn to	O S R N
8.	I am no longer close to anyone	O S R N
9.	My interests and ideas are not shared by those around me	O S R N
10.	I feel left out	O S R N
11.	I feel completely alone	O S R N
12.	I am unable to reach out and communicate with those around me	O S R N
13.	My social relationships are superficial	O S R N
14.	I feel starved for company	O S R N
15.	No one really knows me well	O S R N
16.	I feel isolated from others	O S R N
17.	I am unhappy being so withdrawn	O S R N
18.	It is difficult for me to make friends	O S R N
19.	I feel shut out and excluded by others	O S R N
20.	People are around me but not with me	O S R N

Russell, D , Peplau, L. A.. & Ferguson, M. L. (1978). *Developing a measure of loneliness.* Journal of Personality Assessment, 42, 290-294.

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Functional Assessments

There is no biomarker of loneliness

There are biological signatures of chronic social stress

Does this patient show evidence of a chronic biological response to perceived social threat?

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Social Resilience Panel

DOMAIN	FUNCTIONAL MARKERS	CLINICAL INTERPRETATION
Social Connection	UCLA Loneliness scale	Perceived social safety and connectedness
Autonomic Regulation	HRV, Resting heart rate, breathing rate	Vagal tone and adaptability
HPA Axis	DUTCH Hormone profile Salivary cortisol, DHEA	Threat detection and stress adaptation
Immune activity	hs-CRP, CBC, Lymphocyte subsets	Neuroimmune activation
Oxidative stress	GGT, Ox-LDL, 8-OHDG	Redox balance / NOX activity
Mitochondrial health	Organic acids, Co10, Mitochondrial Profiles	Cellular energy and resilience
Metabolic Resilience	Fasting insulin, HbA1c TG:HDL	Immunometabolic Reserve
Gut-Brain Axis	SCFAs, Calprotectin SIgA, LPS markers	Microbial regulation of immunity
Recovery capacity	Sleep quality	Allostatic recovery

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If social experiences can drive biology,
can biological interventions help restore resilience?

Clinical Interventions Restoring Neuro-resilience

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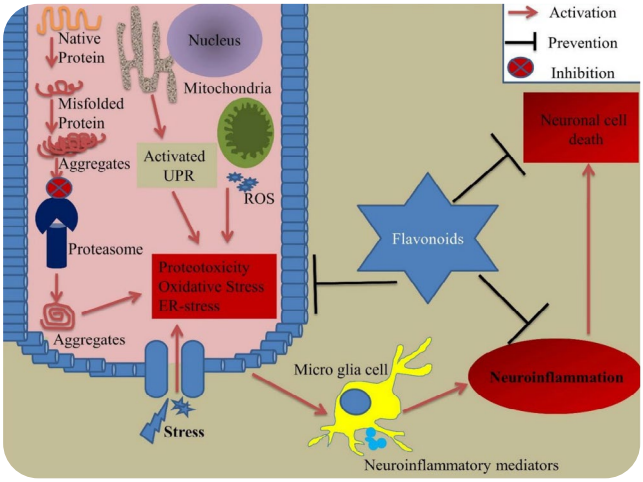
Interventions: **Restoring Neuro-resilience**

- Reduce inflammatory signalling
- Address oxidative stress
- Calm microglia
- Protect mitochondria
- Restore inhibitory tone
- Restore connection

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Flavonoids to the rescue



Devi S, Kumar V, Singh SK, Dubey AK, Kim JJ. *Flavonoids: Potential Candidates for the Treatment of Neurodegenerative Disorders. Biomedicines.* 2021 Jan 20;9(2):99. doi: 10.3390/biomedicines9020099. PMID: 33498503; PMCID: PMC7909525.

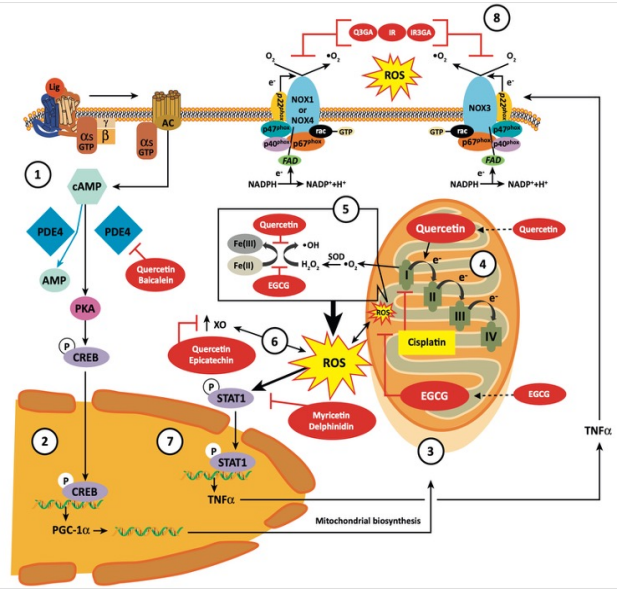
- Anti-inflammatory
- Neuroprotective
- Reduce oxidative stress
- Suppress microglial activation - reduce the neurotoxicity induced by neurotoxic species released by microglia

Luteolin
Apignenin
Rutin
Naringenin
Naringin
Morachalcone – Mulberry leaf
Baicalein
Quercetin
Morin

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More Flavonoids



Flavonoids exert cooperative effects by inhibiting distinct targets responsible for the generation of reactive oxygen species (ROS).

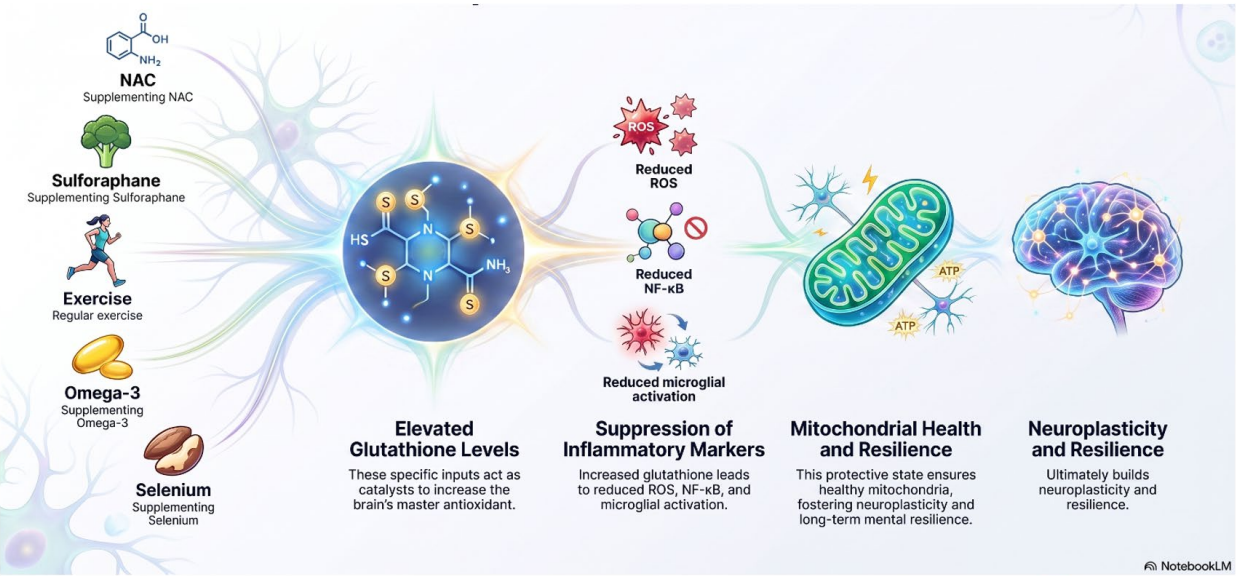
Quercetin
Baicalein
Epicatechin
EGCG
Myricetin
Delphinidin

Jones QRD, Warford J, Rupasinghe HPV, Robertson GS. *Target-based selection of flavonoids for neurodegenerative disorders.* Trends Pharmacol Sci. 2012;33(11):602-610. doi:10.1016/j.tips.2012.08.002onoids*: Potential Candidates for the Treatment of Neurodegenerative Disorders.* Biomedicines. 2021 Jan 20;9(2):99. doi: 10.3390/biomedicines9020099. PMID: 33498503; PMCID: PMC7909525.

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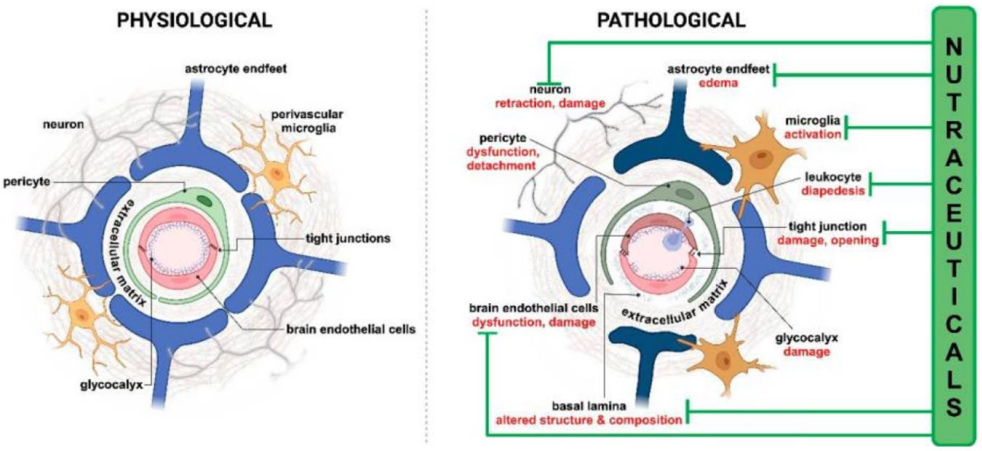
Antioxidants – Glutathione & more



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Protective role of nutraceuticals



Nutraceuticals in preclinical studies:

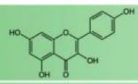
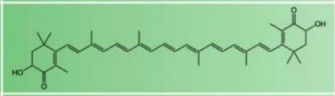
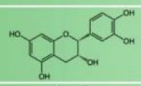
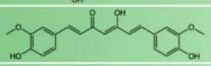
- Restore barrier integrity
- Decrease brain oedema
- Reduce oxidative stress and inflammation
- Restore healthy BBB phenotypes

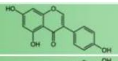
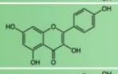
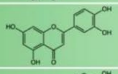
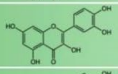
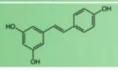
Kocsis AE, Kucsápszky N, Santa-Maria AR, Hunyadi A, Deli MA, Walter FR. *Much More than Nutrients: The Protective Effects of Nutraceuticals on the Blood-Brain Barrier in Diseases.* Nutrients. 2025 Feb 21;17(5):766.

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Protective role of nutraceuticals

COMPOUND	CHEMICAL COMPOSITION	FOOD SOURCE
Apigenin		
Astaxanthin		
Borneol/ Isoborneol		
Catechins		
Curcumin		
Docosa-hexaenoic acid		

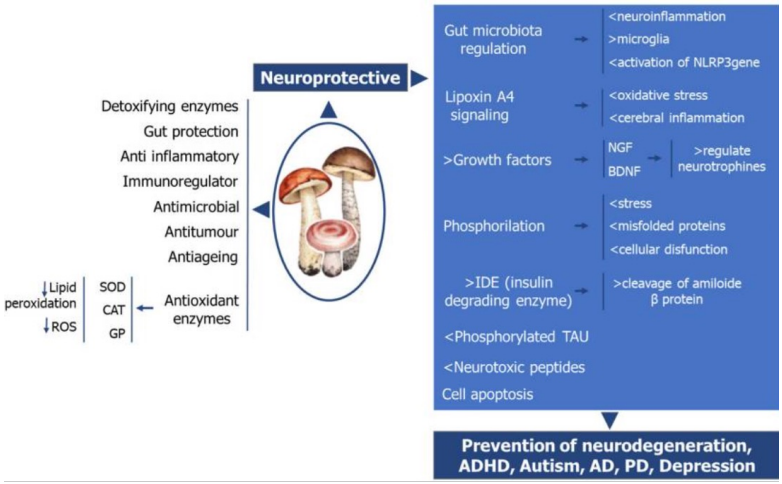
Genistein		
Kaempferol		
Luteolin		
Quercetin		
Resveratrol		

Kocsis AE, Kucsápszky N, Santa-Maria AR, Hunyadi A, Deli MA, Walter FR. *Much More than Nutrients: The Protective Effects of Nutraceuticals on the Blood-Brain Barrier in Diseases.* Nutrients. 2025 Feb 21;17(5):766.

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Medicinal Mushrooms



Bell V, Dimitrov P, Fernandes T. *Supporting Neurologic Health with Mushroom Nutrition.* Nutrients. 2025 May 2;17(9):1568. doi: 10.3390/nu17091568. PMID: 40362877; PMCID: PMC12073738.

Mushroom-derived bioactives may influence multiple interconnected pathological processes involved in neurodegeneration.

Resilience emerges not from targeting a single pathway, but from restoring multiple interconnected neuroimmune, mitochondrial and neuroplasticity networks.

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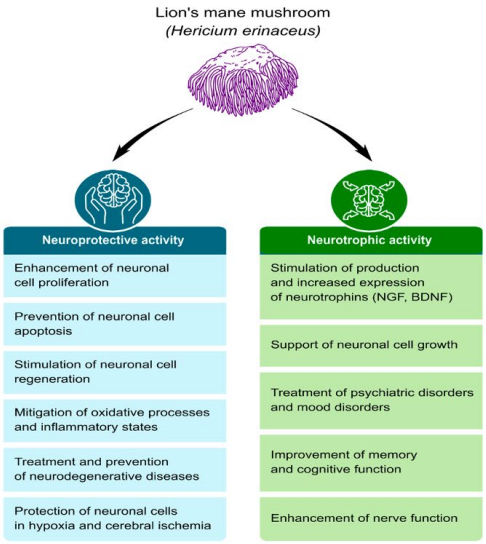
Lion's Mane – *Hericium ericanaceus*

Lion's Mane may support neuroimmune resilience by:

- Promoting neurotrophic signalling (NGF& BDNF)
- Reducing oxidative stress and inflammatory injury
- Protecting vulnerable neurons
- Enhancing neuroplasticity

Together, these mechanisms may improve the brain's capacity for repair and adaptation following chronic stress.

Evidence is derived primarily from preclinical studies.

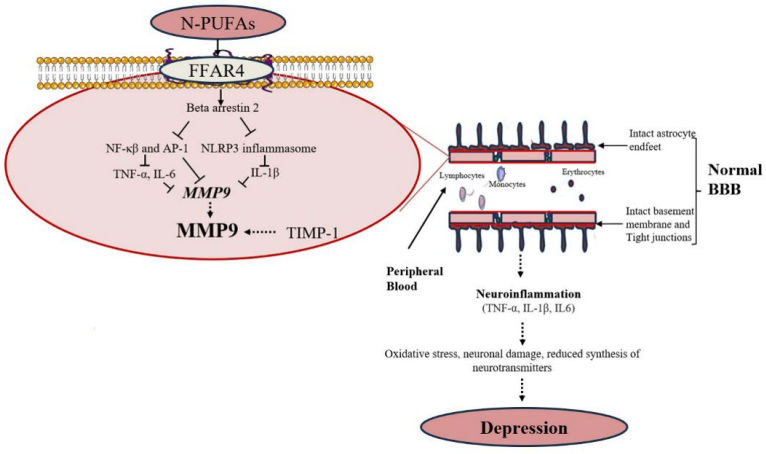


Szućko-Kociuba I, Trzeciak-Ryczek A, Kupnicka P, Chlubek D. *Neurotrophic and Neuroprotective Effects of Hericium erinaceus.* Int J Mol Sci. 2023 Nov 3;24(21):15960. doi: 10.3390/ijms242115960. PMID: 37958943; PMCID: PMC10650066.

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Omega-3 fatty acids



EPA and DHA act at multiple points within the neuroimmune network:

- Reducing inflammatory signaling
- Preserving blood–brain barrier integrity
- Supporting resolution pathways that protect the brain from chronic neuroinflammation

Malau IA, Chang JP, Lin YW, Chang CC, Chiu WC, Su KP. *Omega-3 Fatty Acids and Neuroinflammation in Depression: Targeting Damage-Associated Molecular Patterns and Neural Biomarkers.* Cells. 2024 Oct 29;13(21):1791. doi: 10.3390/cells13211791. PMID: 39513898; PMCID: PMC11544853.

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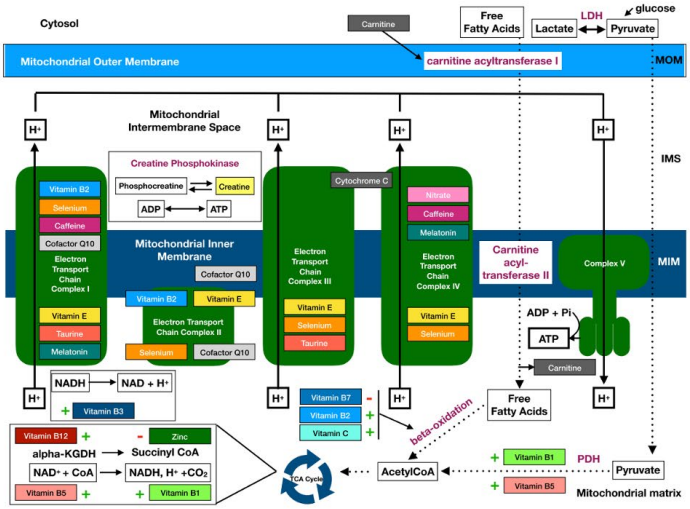
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Mitochondrial Nutrients

Every adaptive response requires energy.

Mitochondria rely on micronutrients to convert carbohydrates, fats and amino acids into ATP while maintaining antioxidant defences and redox homeostasis.

Wesselink E, Koekkoek WAC, Grefte S, Witkamp RF, van Zanten ARH. *Feeding mitochondria: Potential role of nutritional components to improve critical illness convalescence.* Clin Nutr. 2019 Jun;38(3):982-995. doi: 10.1016/j.clnu.2018.08.032. Epub 2018 Aug 31. PMID: 30201141.



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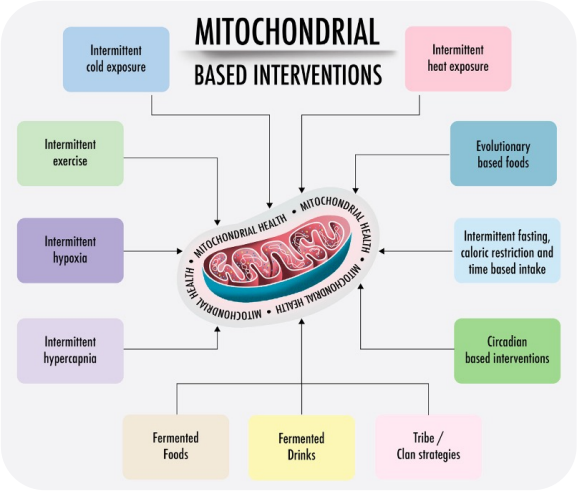
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Hormesis & the mitochondria

Hormesis - adaptive response to mild stress that builds resilience and increases resilience to future stress

Mitohormetic strategies can support mitochondrial resilience and health:

- Intermittent heat/cold exposure
- Intermittent fasting
- Intermittent hypercapnia/hypoxia
- Intermittent exercise
- Fermented foods



Casanova A, Wevers A, Navarro-Ledesma S, Pruimboom L. *Mitochondria: It is all about energy. Front Physiol.* 2023 Apr 25;14:1114231. doi: 10.3389/fphys.2023.1114231. PMID: 37179826; PMCID: PMC10167337.

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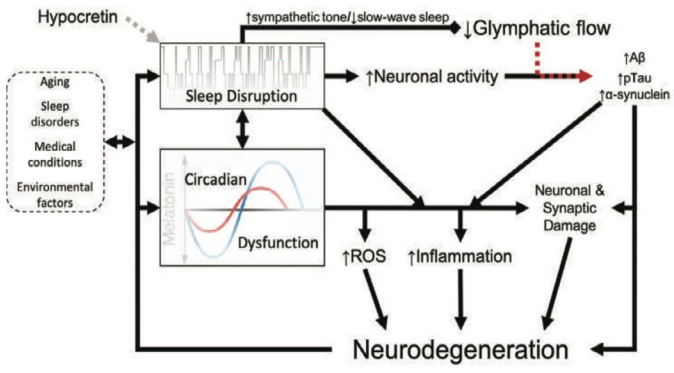
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Restoring the brain's protective state

Restoring inhibitory tone

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Sleep is underrated



Schneider L. *Neurobiology and Neuroprotective Benefits of Sleep. Continuum (Minneap Minn).* 2020 Aug;26(4):848-870. doi: 10.1212/CON.0000000000000878. PMID: 32756225.

Chronic sleep disruption promotes neuroinflammation, oxidative stress, impaired glymphatic clearance and synaptic dysfunction, accelerating biological processes associated with neurodegeneration.

Sleep is not simply a period of rest – it is an active neuroprotective state that supports metabolic restoration, glymphatic clearance, synaptic homeostasis and neuronal repair.

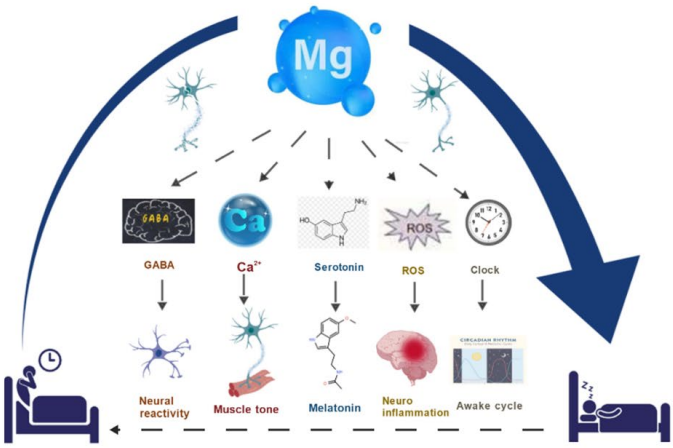
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Magnesium – not just for sleep

Magnesium helps shift the brain from a state of hypervigilance to restoration.

It enhances GABAergic neurotransmission, limits glutamate-mediated excitotoxicity through NMDA receptor modulation, supports melatonin production and circadian rhythms, reduces oxidative stress and inflammation, and optimises cellular energy production.

It supports the neurobiological conditions required for restorative sleep, neuroplasticity and resilience.

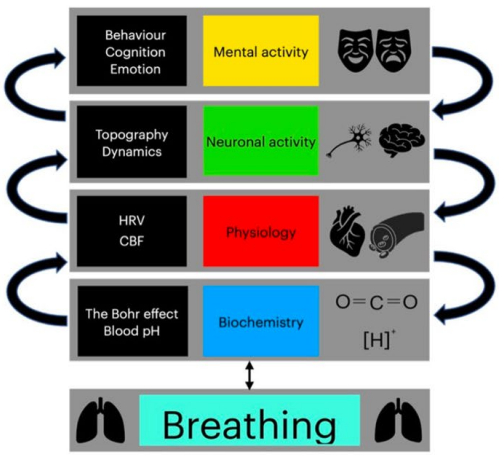


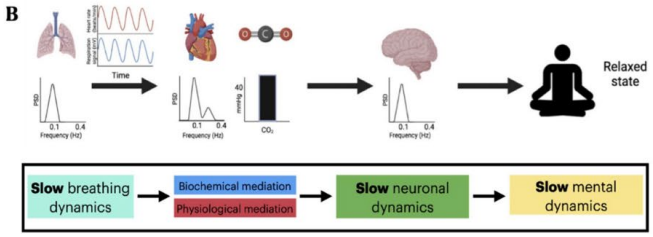
He C, Wang B, Chen X, Xu J, Yang Y, Yuan M. *The Mechanisms of Magnesium in Sleep Disorders*. Nat Sci Sleep. 2025 Oct 15;17:2639-2656. doi: 10.2147/NSS.S552646. PMID: 41116797; PMCID: PMC12535714.

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Breathing: Restoring Inhibitory Control





Breathing is both automatic and under voluntary control → **unique gateway for influencing brain function**

By modulating autonomic activity, neural synchrony and emotion-regulating networks, slow breathing helps shift the brain from a hypervigilant state towards regulation & restoration → **supporting the neuroimmune and metabolic foundations of resilience**

Goheen J, Anderson JAE, Zhang J, Northoff G. From Lung to Brain: *Respiration Modulates Neural and Mental Activity*. Neurosci Bull. 2023 Oct;39(10):1577-1590. doi: 10.1007/s12264-023-01070-5. Epub 2023 Jun 7. PMID: 37285017; PMCID: PMC10533478.

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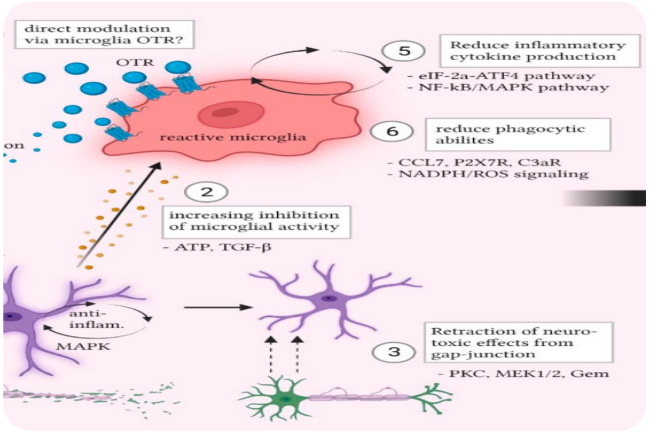
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Oxytocin – a neuroimmune regulator

Oxytocin shifts glial cells from pro-inflammatory to neuroprotective

Acting via astrocyte and microglial OT receptors, oxytocin helps to:

- Reduce cytokine production
- Attenuate NADPH-dependent oxidative activity
- Trigger astrocyte process retraction from the synapse - decreasing neurotoxic gliotransmitter release and restoring neuroimmune homeostasis



Knoop M, et al., *The Role of Oxytocin in Abnormal Brain Development: Effect on Glial Cells and Neuroinflammation. Cells.* 2022 Dec 2;11(23):3899. doi: 10.3390/cells11233899. PMID: 36497156; PMCID: PMC9740972.

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Oxytocin & Touch

Type of tactile stimulation	Primary Nerve Fibers/Receptors	Peptides/Transmitters released	Effects
Light (gentle) touch	CT-fibers Mechanoreceptors	xytocin Dopamine	Social interaction, wellbeing Anxiety↓
Firm touch	A-beta-fibers Mechanoreceptors	Oxytocin Dopamine Opioids Noradrenaline ↓	Calm, growth, reproduction, pain↓, blood pressure↓ Cortisol↓↓

Not all touch is biologically equivalent:

- Gentle affective touch - social approach through CT afferents and oxytocin
- Sustained touch - holding or hugging recruits additional oxytocinergic pathways → increases vagal activity, reduces stress and support restoration

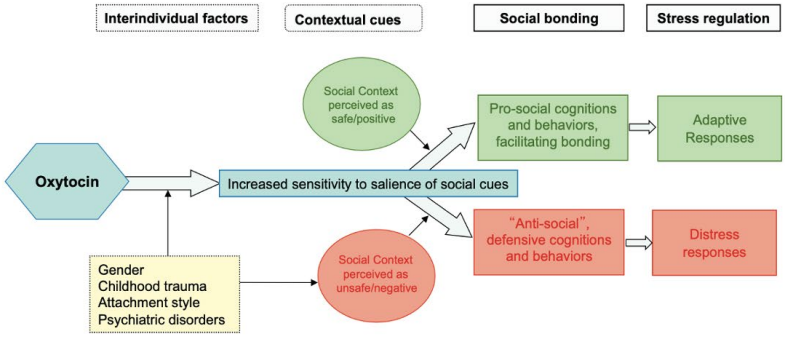
Provides the basis of the proposed "Calm and Connection System"

Uvnäs-Moberg K and Petersson M (2026) *Oxytocin release patterns and effects, with a focus on touch, social interaction, and closeness.* Front. Psychol. 17:1771799. doi: 10.3389/fpsyg.2026.1771799

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Oxytocin – A Social Safety Amplifier



Olff M, et al., *The role of oxytocin in social bonding, stress regulation and mental health: an update on the moderating effects of context and interindividual differences.* Psychoneuroendocrinology. 2013 Sep;38(9):1883-94. doi: 10.1016/j.psyneuen.2013.06.019. Epub 2013 Jul 12. PMID: 23856187

Oxytocin amplifies social salience - context and history determine the outcome.

When the social environment is perceived as safe, oxytocin promotes prosocial cognition, trust, and adaptive stress responses. When cues are perceived as threatening, oxytocin may amplify defensive, avoidant, or anti-social responses instead.

Restoring felt safety is a prerequisite for oxytocin's restorative effects.

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The Connecting Power of Nature

"A fundamental drive for humans is biophilia, a preference for natural environments that match the habitats in which humans evolved"

Research has shown time in nature improves wellbeing because it increases feelings of connection to nature and thus fulfils the biophilia drive.

- Green spaces increase feelings of community and thereby decrease loneliness
- They described these feelings of community as both emotional attachment to a neighbourhood and a sense of connection with place

Henderson L, Tipper L, Willicombe S, Gattis M. *Shared time in nature increases feelings of social connection amongst university students.* J Environ Psychol. 2024;96:102343. doi:10.1016/j.jenvp.2024.102343

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The Connecting Power of Nature

Cardiff University: 52 university students

- All completed the same collaborative 15-minute task either outdoors in a city park or indoors in a university office
- Most were strangers - researchers could assess whether the environment itself influenced interpersonal connection

The findings were clear:

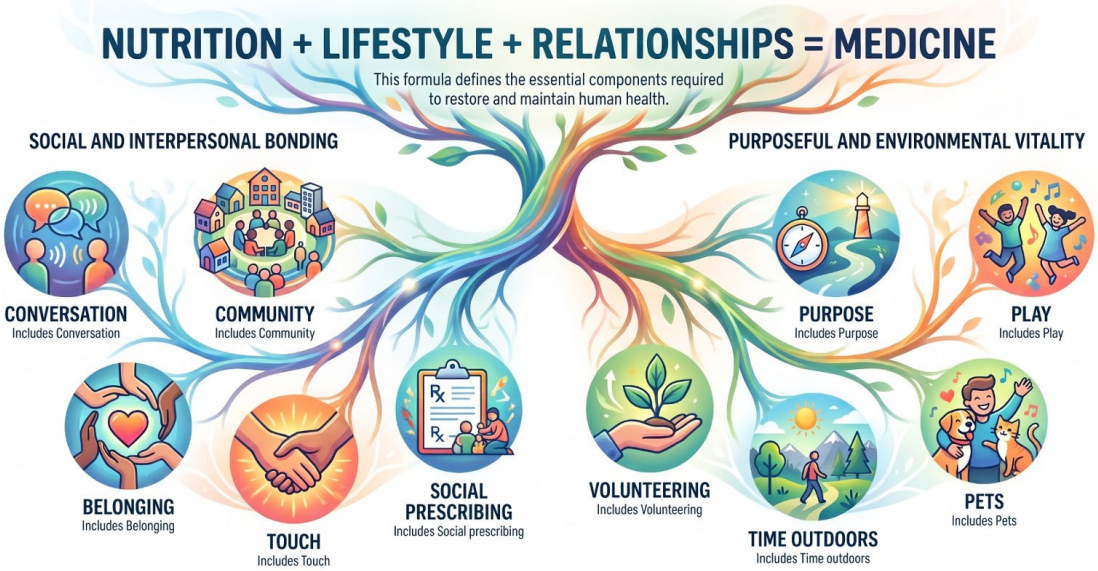
- Participants reported significantly greater feelings of social connection after completing the task in the park, with a large effect size
- There was no increase in social connection after completing the same task indoors
- Participants also experienced a significant increase in connection with nature following the outdoor activity

Henderson L, Tipper L, Willicombe S, Gattis M. *Shared time in nature increases feelings of social connection amongst university students.* J Environ Psychol. 2024;96:102343. doi:10.1016/j.jenvp.2024.102343

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Connection is Medicine: The New Equation for Health



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NotebookLM bioconcepts

Take home summary

- The brain evolved expecting social connection
- Social isolation is interpreted as biological threat
- Social experiences shape immune, mitochondrial and neural function
- Neuroinflammation reduces inhibitory tone and resilience
- Restoring resilience requires both biological and social interventions

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Reconnection and Resilience

The opposite of chronic stress is not simply relaxation.
It is reconnection.

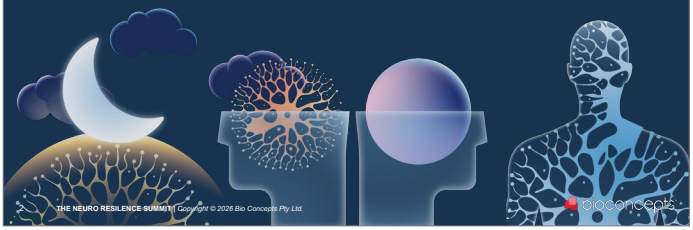
- **Reconnection** with ourselves through sleep and breath
- **Reconnection** with others through meaningful relationships
- **Reconnection** with the natural world that shaped our biology

Resilience emerges when the brain once again feels safe enough to reconnect.

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Sleep for Life:
How Good Sleep Supports Healthy Brain Ageing



SPEAKER

Stephanie R. Rainey-Smith
BSc (Hons), PhD



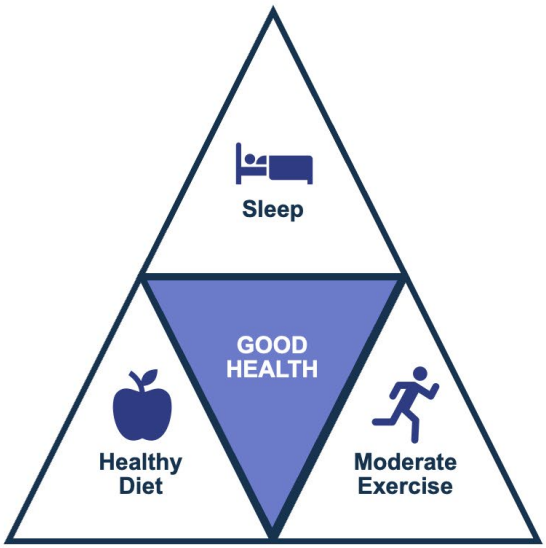
OUTLINE

1. Importance of sleep
2. Normal and suboptimal sleep
3. Health risks of poor sleep
4. Sleep and Alzheimer's disease
5. Modifying risk via improved sleep
6. Next steps
7. Summary of evidence
8. Key considerations

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Sleep – The Third Pillar of Good Health



Parliamentary Enquiry into Sleep Health Awareness in Australia, 2019

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Sleep – Importance

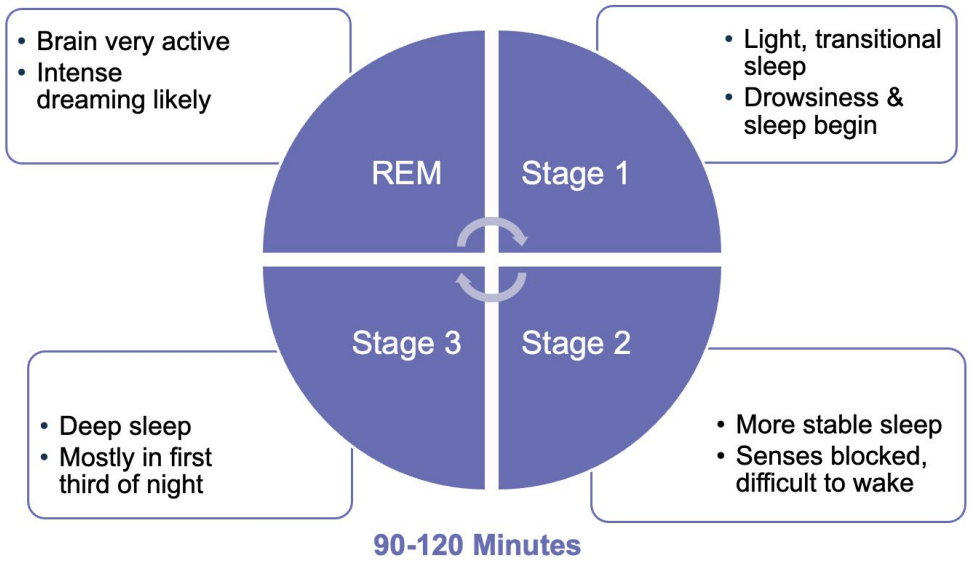
- Behaviour conserved across mammalian species
- Humans spend 1/3 of our lives sleeping
- Why do we sleep?
  - Prepare us for optimal functioning during the day
  - Conserve energy and regulate metabolism
  - Growth
  - Repair or replace damaged cells and tissues
  - Reinforce immune system
  - Restorative
  - Processing of information
  - Consolidation of memory
  - Clearance of toxins



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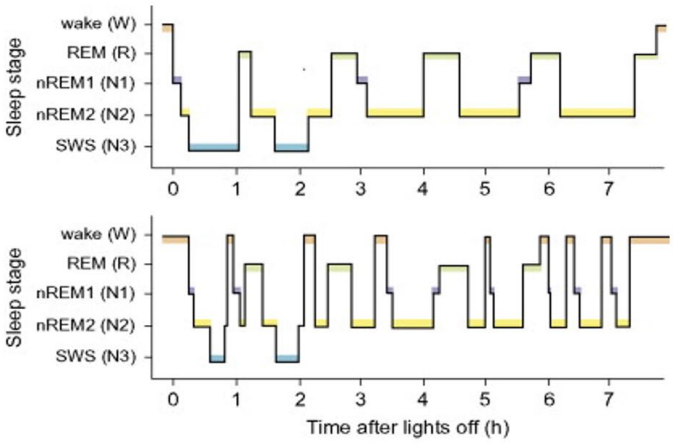
Sleep Architecture – Sleep Cycle



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Sleep Architecture – Changes with Age



Young Adults

Older Adults

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Common Types of Sleep Disorders

1. **Insomnia**
   Difficulty falling asleep or staying asleep
2. **Sleep Apnoea**
   Breathing repeatedly starts and stops
3. **Hypersomnia**
   Feeling exhausted after 8-10 hours of sleep
4. **Narcolepsy**
   Irregular sleep-wake cycles
5. **Parasomnia**
   Abnormal or unusual behaviour during sleep



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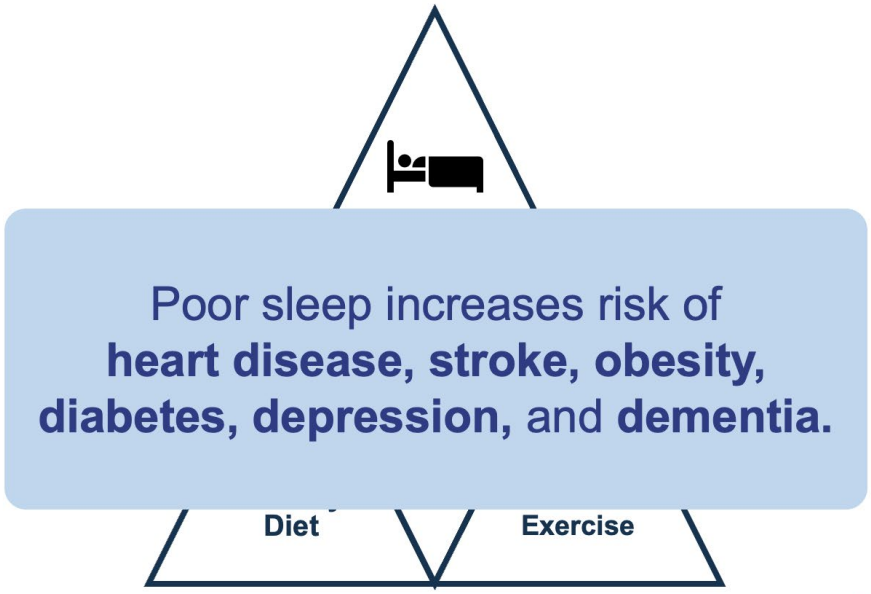
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Poor sleep is a growing and under recognised problem **impacting 6 in 10 adults over the age of 65**

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Sleep – The Third Pillar of Good Health



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Parliamentary Enquiry into Sleep Health Awareness in Australia, 2019

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Sleep and Cardiovascular Disease

Cardiovascular response to normal sleep:
- ↓ Sympathetic activity
- ↓ Heart rate
- ↓ Blood pressure
- ↓ Arrhythmogenicity
- ↑ Parasympathetic activity
- ↑ Cardiac refractory period



Adapted from Jean-Louis *et al.*, 2010

Physiologic effects
- Hypoxia
- Increased sympathetic activity
- Intrathoracic pressure changes
- Cortical arousal

Sleep Apnoea

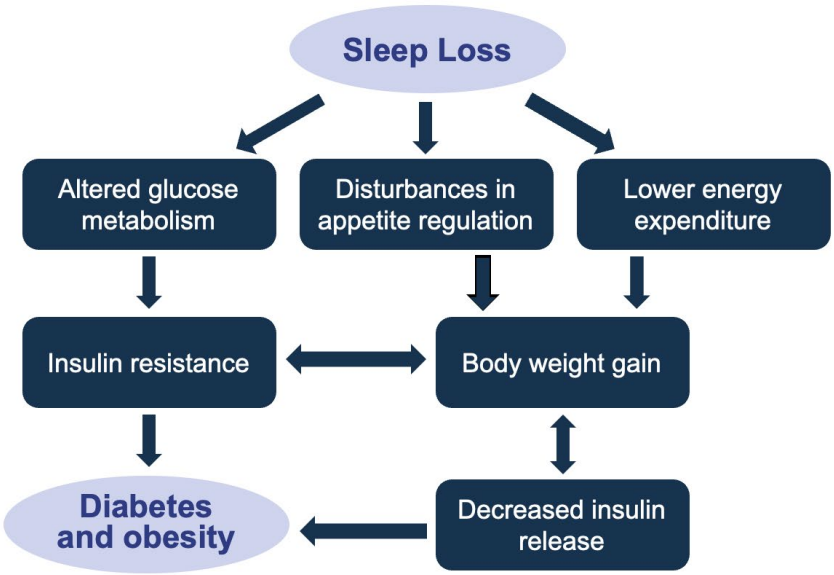
Cardiovascular Disease
- Hypertension
- Congestive heart failure
- Cardiac ischaemia
- Cardiac arrhythmia
- Cerebrovascular disease

Biologic effects
- Inflammation
- Oxidative stress
- Endothelial dysfunction
- Adipokines

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Sleep, Diabetes and Obesity

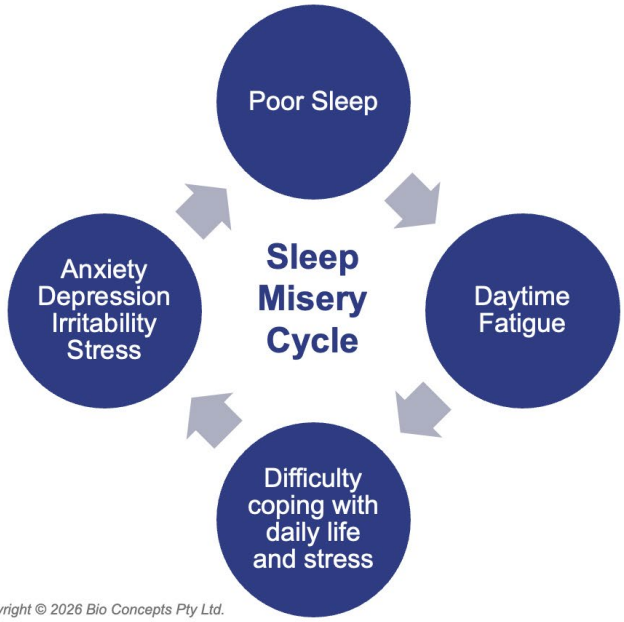


Adapted from Knutson *et al.*, 2007

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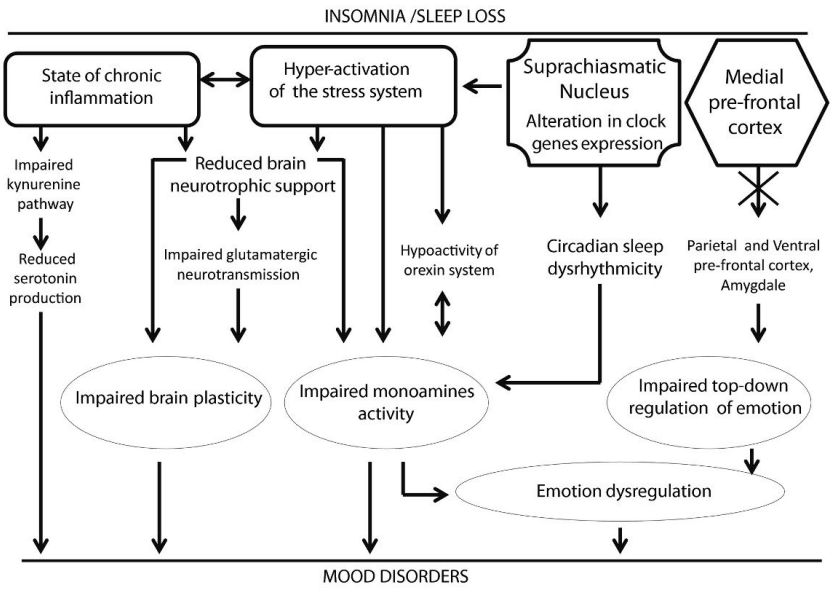
Sleep and Mood Regulation



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Sleep and Mood Regulation – Mechanisms



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Palagni *et al.*, 2019

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Sleep and Health Risks

- We know poor sleep increases risk of;
  - Heart disease
  - Obesity
  - Diabetes
  - Depression
- All increase dementia risk
- Poor sleep also independently increases dementia risk






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Sleep – Importance

- Behaviour conserved across mammalian species
- Humans spend 1/3 of our lives sleeping
- Why do we sleep?
  - Prepare us for optimal functioning during the day
  - Conserve energy and regulate metabolism
  - Growth
  - Repair or replace damaged cells and tissues
  - Reinforce immune system
  - Restorative
  - Processing of information
  - Consolidation of memory
  - Clearance of toxins



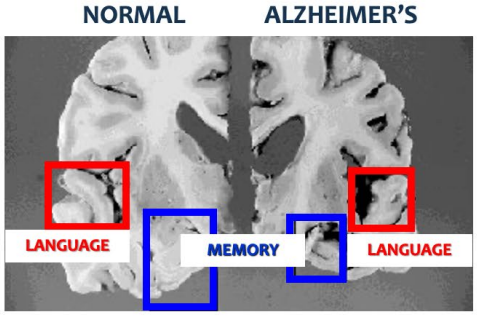
- **Relevance to Alzheimer's disease, the leading cause of dementia…**
  - Learning and memory impaired in cognitive decline and Alzheimer's
  - Mechanism of Aβ-amyloid (Aβ) clearance?

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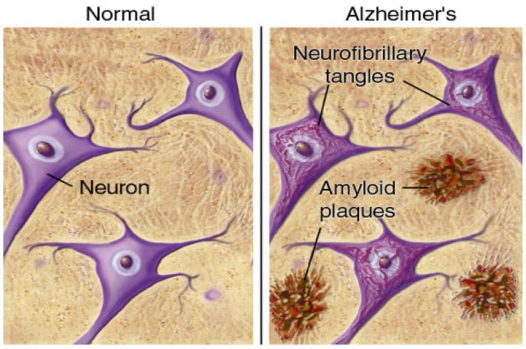
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Sleep and Alzheimer's Disease

Sleep disturbances (e.g. increased fragmentation) are one of first symptoms/complaints → due to neurodegeneration and neuropathology



Coronal section of normal and Alzheimer's affected brain



Amyloid plaques → Insoluble aggregates of Aβ-amyloid (Aβ)
Neurofibrillary tangles → Hyper phosphorylated tau

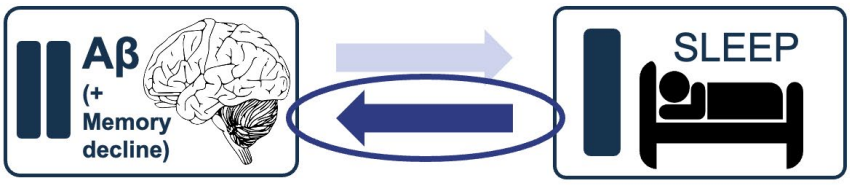
Images courtesy of Professor Ralph Martins, UWA

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Sleep and Alzheimer's Disease

- Sleep disturbances (e.g. ↑ fragmentation) are one of first symptoms – due to neurodegeneration and neuropathology
- However, suboptimal sleep also contributes to cognitive decline, and accumulation of neuropathology

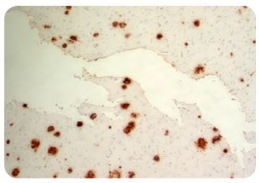


Bi-directional relationship

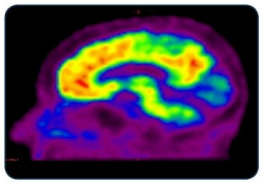
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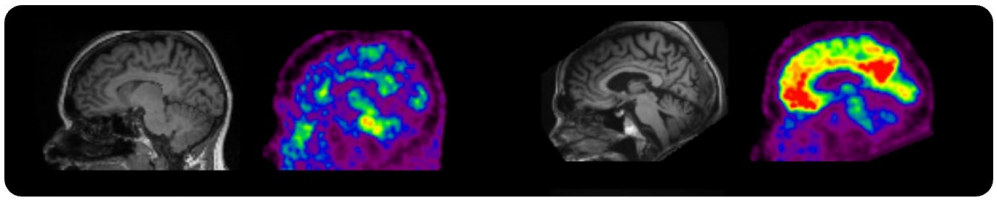
Brain Imaging - Amyloid PET



Amyloid beta (Aβ) plaques seen under a microscope in post-mortem brain tissue from a patient with AD



PET scan showing brain areas containing Aβ plaques (yellow and red areas) in a living person with early AD



Healthy Elderly

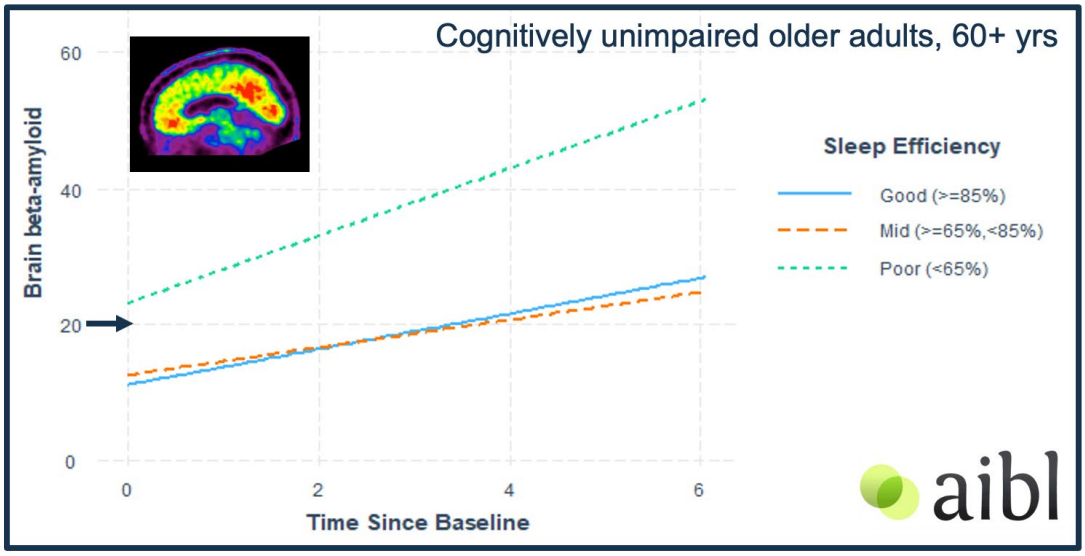
Alzheimer's Disease

PET images courtesy of Professor Chris Rowe, Austin Health

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Sleep and Brain Aβ Accumulation



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Pivac *et al.*, 2024

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How might suboptimal sleep lead to increased brain Aβ?

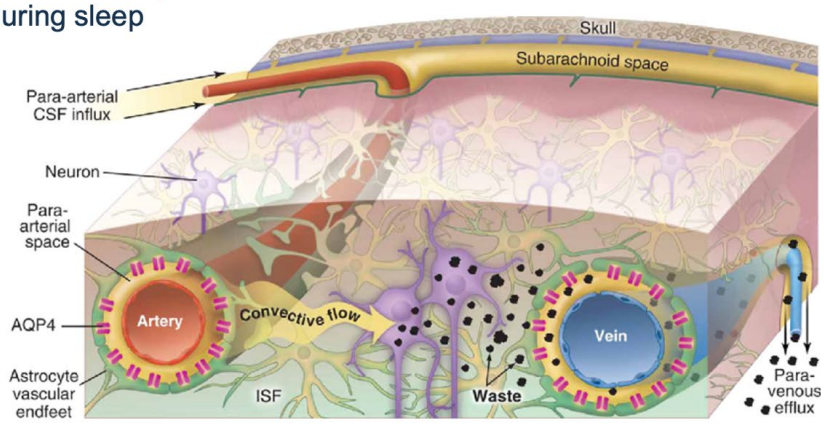
… poor clearance?

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Glymphatic System: 'glia' + lymphatic

- Metabolic waste propelled by convective flow of cerebrospinal fluid and interstitial fluid → paravenous space → lymphatic vessels → return to general circulation for clearance via kidneys & liver.
- Effective during sleep



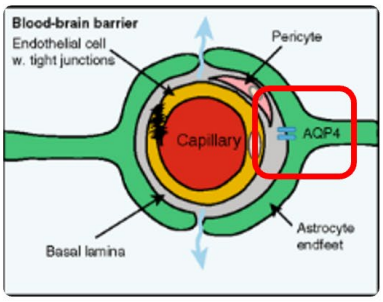
Nedergaard, Science, 2013

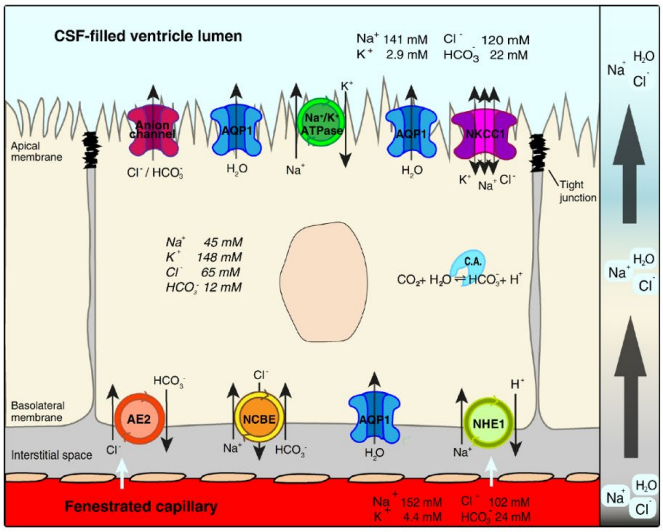
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Aquaporins (AQP) and the Glymphatic System

- Water channel proteins
- AQP4 - Astrocytic endfeet
- AQP1 - Choroid plexus





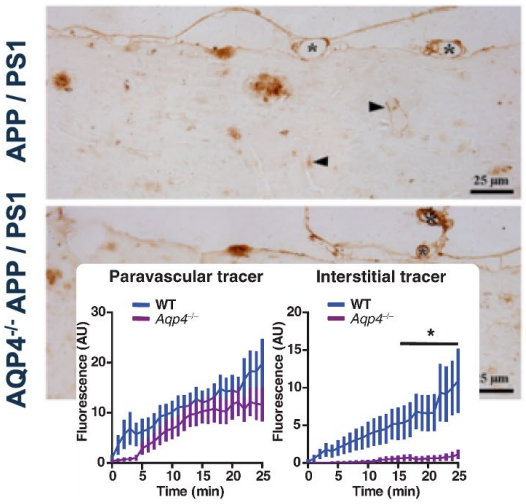
Benga and Huber, 2012; Potokar *et al.*, 2016

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Impact of AQP on brain Aβ burden

- Animal models:
  - *AQP4* knockout x AD transgenic mice
  - Increased Aβ deposition
  - Suggests less Aβ clearance via glymphatic system
- Clearance studies in *AQP4* knockout mice:
  - *AQP4* KO → decreased tracer clearance (Paravascular → Interstitial)
  - Poorer glymphatic function
- However, paucity of literature in humans on relationship between *AQPs* and brain Aβ burden



Yang *et al.*, 2012; Xu *et al.*, 2015; Igarashi *et al.*, 2014

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Sleep, AQPs and brain Aβ in humans

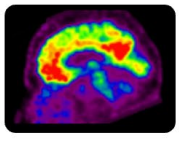
AIM: Determine whether genetic variants in *AQP1* and *AQP4* moderate the relationship between sleep parameters and brain Aβ burden in cognitively unimpaired older adults

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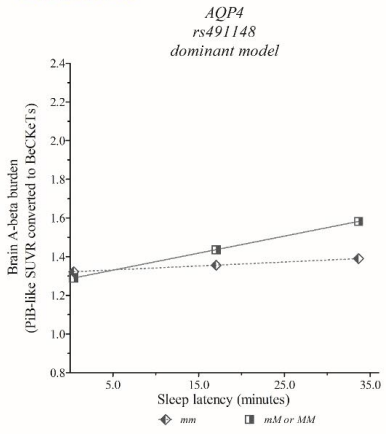
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AQP4: rs491148 moderates relationship between sleep onset latency and brain Aβ-burden in cognitively unimpaired older adults

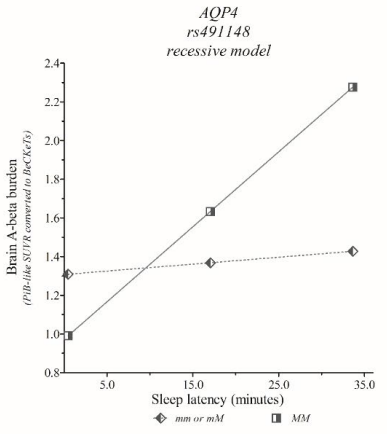
World First Study



Amyloid beta levels



Interaction model:
$\Delta R^2 = 0.017$, $p = 0.036$



Interaction model:
$\Delta R^2 = 0.020$, $p = 0.022$

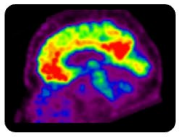
Rainey-Smith *et al.*, Transl. Psychiatry, 2018

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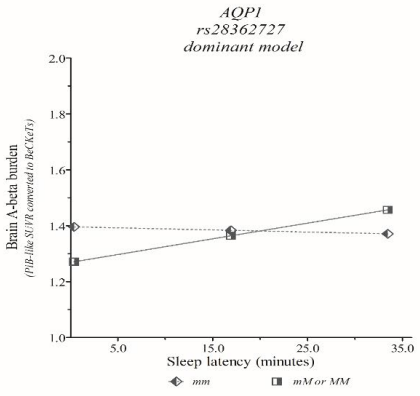
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AQP1: rs28362727 moderates relationship between sleep onset latency and brain Aβ-burden in cognitively unimpaired older adults

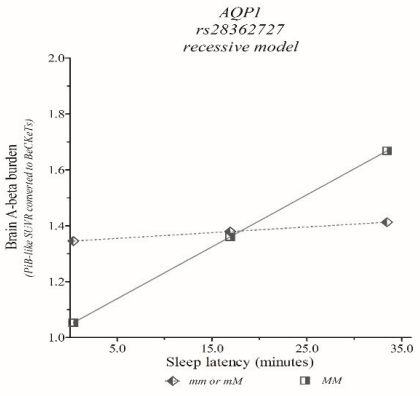
World First Study



Amyloid beta levels



Interaction model:
$\Delta R^2 = 0.018$, $p = 0.034$



Interaction model:
$\Delta R^2 = 0.035$, $p = 0.003$

Rainey-Smith *et al.*, Transl. Psychiatry, 2018

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Apolipoprotein E: Another important gene

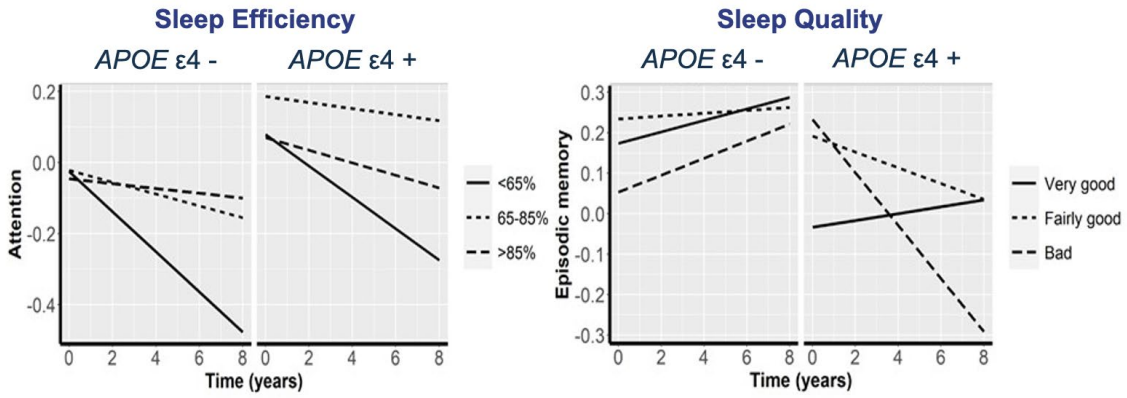
- *APOE* gene on Chromosome 19.
- Three major isoforms encoded by alleles ε2, ε3, ε4.
- Peripherally, ApoE protein is primarily produced by the liver and macrophages.
- In the central nervous system, ApoE is mainly produced by astrocytes.
- ApoE protein has many metabolic and neuronal functions including cholesterol processing and transport, axon guidance, microtubule stability and Amyloid processing.
- ***APOE* is the major genetic risk factor for Alzheimer's disease.**

***APOE* Allele**	**Alzheimer's Disease Risk**
ε2	Protective?
ε3	Normal risk?
ε4 Heterozygote	Increased risk
ε4 Homozygote	10-30 x risk by 75 years vs non ε4 carriers

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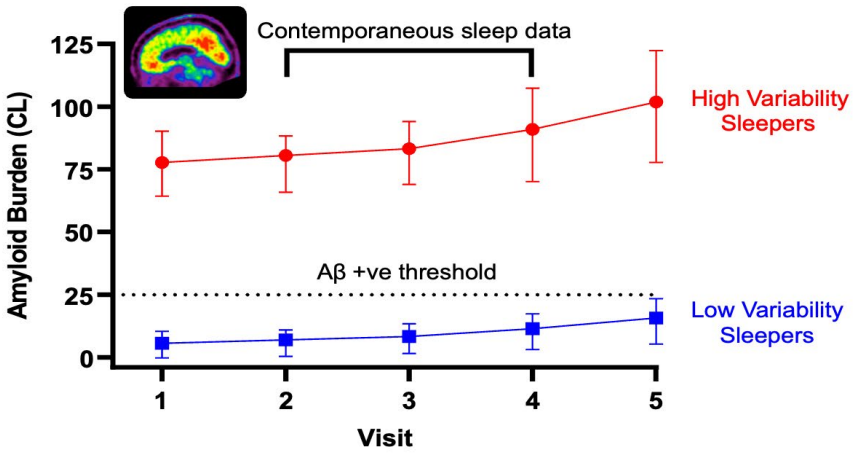
Sleep and cognitive trajectories



- *N* = 526, 60+ years, cognitively unimpaired
- Poorer self-report sleep efficiency, faster decline in attention, irrespective of *APOE* genotype ($p < 0.01$).
- Poorer sleep quality, faster decline in episodic memory in *APOE* ε4 carriers, and poorer performance in *APOE* ε4 non-carriers ($p < 0.05$), compared to good sleepers.

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Pivac *et al.*, Manuscript under review.

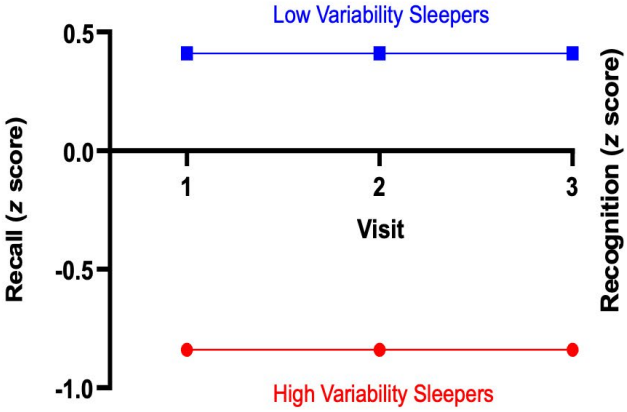
Sleep variability and brain Aβ

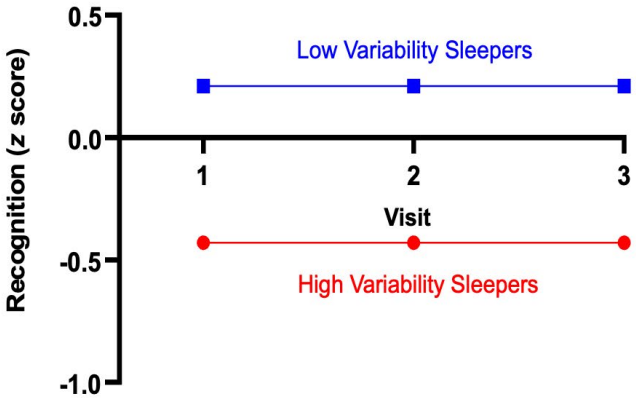


- Those with longer and more variable Time in Bed had substantially greater brain Aβ
- No class differences in rates of Aβ accumulation

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Mladenovic *et al.*, Manuscript in preparation.

Sleep variability and cognition



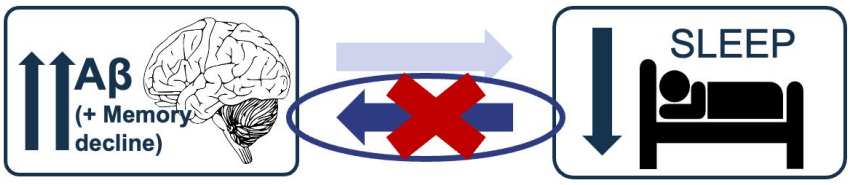


Recall *M*(*SE*)
Class 1: *z* = 0.41 (0.15)
Class 2: *z* = -0.84 (0.58)
Diff: 1.25 *SD* units

Recognition *M*(*SE*)
Class 1: *z* = 0.21 (0.09)
Class 2: *z* = -0.43 (0.30)
Diff: 0.64 *SD* units

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Mladenovic *et al.*, Manuscript in preparation.

If suboptimal sleep increases risk of cognitive decline and Alzheimer's disease, could improving sleep decrease risk?



Bi-directional relationship

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Sleep Improvement Study:
The Effect of Improved Sleep on Cognition and Biomarkers of Brain Health

Recruited individuals:

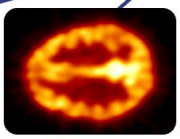
- Community-dwelling 60-80 years
- Poor sleep (PSQI > 5 or ISI > 10)
- Subjective memory complaint (MAC-Q > 25)
- No objective cognitive impairment
- No untreated severe obstructive sleep apnoea
- No severe current depression
- No severe chronic pain
- Willing to spend 2 nights in a sleep lab
- Willing to undertake brain scans

↑ risk of dementia due to Alzheimer's disease

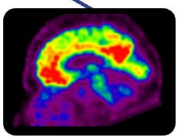




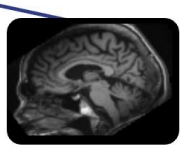
Measures of memory and thinking



Glucose metabolism



Amyloid beta levels



Brain structure volume

Rainey-Smith *et al.*, Manuscript in preparation.

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Sleep Improvement Study:
The Effect of Improved Sleep on Cognition and Biomarkers of Brain Health

BASELINE
Sleep assessment
Neuropsychologic al tests
Brain Imaging
Blood sample

INTERVENTION
To improve sleep

9 MONTHS
Sleep assessment

18 MONTHS
Sleep assessment
Neuropsychologic al tests
Brain Imaging
Blood sample

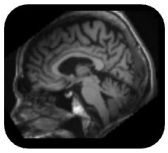
36 MONTHS
Sleep
Cognition
Imaging
Bloods



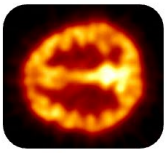
Sleep Assessments



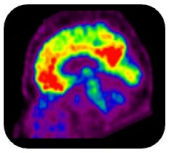
Measures of memory and thinking



Brain structure volume



Glucose metabolism



Amyloid beta levels



Blood-based biomarkers

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Sleep Improvement Study:
The Effect of Improved Sleep on Cognition and Biomarkers of Brain Health

INTERVENTION

Cognitive Behavioural Therapy for Insomnia (CBT-I)

First line treatment for insomnia globally

Proven efficacy for improving sleep

Durable effects and an excellent safety profile

Addresses underlying cause of poor sleep

Content delivery phase + reinforcement phase

Not previously investigated in target population, or in context of Alzheimer's disease biomarkers



Rainey-Smith *et al.*, Manuscript in preparation.

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Understanding Cognitive Behavioural Therapy for Insomnia

- Targets the behavioural and cognitive perpetuating factors of insomnia and avoids medication-related harms
- CBT-I treatment strategies include:
  - Restricting time spent in bed - to consolidate fragmented sleep
  - Stimulus control - to strengthen the association between bed and sleep
  - Sleep education - discussion of sleep cycles, effects of diet and exercise etc.
  - Cognitive therapy - to identify and modify dysfunctional beliefs and attitudes concerning sleep and insomnia

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Cognitive Behavioural Therapy for Insomnia: Cognition and brain changes

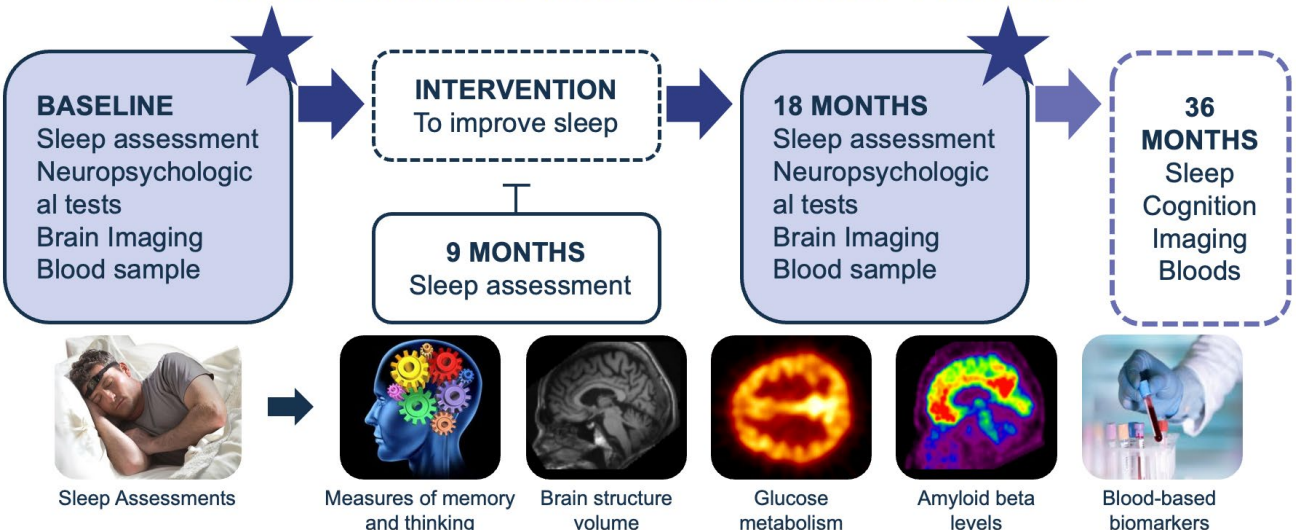
- Improved cognition post-CBT-I in dementia and mild cognitive impairment cohorts has been reported (Latella et al., 2025).
- Neural changes including increased brain volumes and glucose metabolism have been induced in clinical populations (chronic pain, PTSD, OCD) treated with non-sleep focused CBT (Seminowicz et al., 2013; Levy-Gigi et al., 2013; Apostolova et al., 2010).
- Following CBT-I, changes in subcortical functional connectivity, consistent with insomnia symptom reduction, have also been observed (Lee et al., 2018).
- **Stimulus for the current study investigating cognition and neuroimaging outcomes in older adults with subjective cognitive decline AND poor sleep, as a potentially preventive therapy for dementia due to Alzheimer's disease.**

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Sleep Improvement Study:

The Effect of Improved Sleep on Cognition and Biomarkers of Brain Health



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Sleep Improvement Study:
Demographics

Baseline Demographics (to date)	N = 62
Age, years	70.3 (5.1)
Female, N (%)	42 (67.8)
Education, years	14.9 (2.2)
APOE ε4 carriers, N (%)	19 (30.6)
High brain Aβ, N (%)	17 (27)
Body Mass Index	26.6 (5.5)
Cardiovascular disease score	0.3 (0.4)
Premorbid intelligence, CCRT	37.4 (6.6)
Cognitive to sleep assessment, weeks	7.2 (8.1)
Apnoea-Hypopnoea Index	11.2 (9.6)

Mean (SD) unless otherwise stated.

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Sleep and mood improvement following CBT-I program

	Baseline	18 months	t (df)	p; d
Self-reported sleep (N = 62)				
Sleep duration, hours	5.8 (1.3)	6.4 (1.1)	-4.59 (61)	<0.001; -0.58
Sleep efficiency, %	68.3 (0.1)	75.7 (0.1)	-5.10 (61)	<0.001; -0.65
Sleep onset latency, minutes	24.5 (17.7)	18.3 (13.7)	3.28 (61)	0.002; 0.46
Sleep disturbance score	10.0 (3.4)	9.1 (3.8)	2.05 (61)	0.044; n/a
Sleep quality	1.7 (0.6)	1.1 (0.5)	7.52 (61)	<0.001; 0.95
PSQI global score	9.8 (2.8)	7.1 (2.7)	8.61 (61)	<0.001; 1.09
Insomnia Severity Index score	13.7 (4.4)	7.3 (4.2)	10.41 (61)	<0.001; 1.32
Sleep medication use	0.7 (1.0)	0.5 (0.8)	1.96 (61)	0.055; n/a
DASS-21 outcomes				
Depression Scale score	2.8 (2.7)	2.3 (2.3)	1.83 (61)	0.073; n/a
Anxiety Scale score	1.3 (1.4)	1.3 (1.4)	0.17 (62)	0.864; n/a
Stress Scale score	5.2 (2.6)	4.0 (2.4)	3.59 (62)	0.001; 0.46

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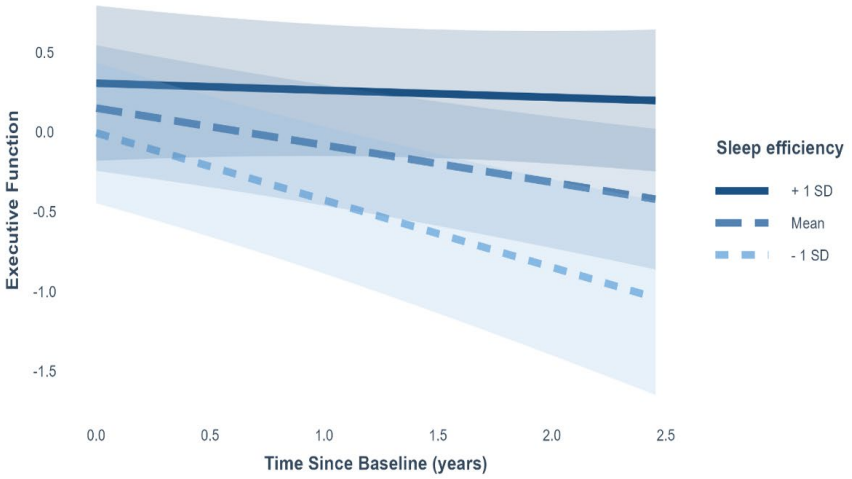
Associations of sleep improvement, following CBT-I program, with change in executive function, at 18-months

	Executive Function Whole Sample (N = 62)	
Models / Predictors	**β ± SE**	***p*-value**
Sleep duration (hr) * Time * Baseline brain Aβ	-0.19 ± 0.05	**0.001#**
Sleep duration (hr) * Time	0.14 ± 0.04	**0.001#**
Sleep duration (hr)	0.04 ± 0.10	0.648
Sleep efficiency (%) * Time * Baseline brain Aβ	-1.77 ± 0.60	**0.004#**
Sleep efficiency (%) * Time	1.58 ± 0.52	**0.003#**
Sleep efficiency (%)	0.95 ± 1.00	0.340
Sleep onset latency (min) * Time * Baseline brain Aβ	0.01 ± 0.00	0.071
Sleep onset latency (min) * Time	-0.01 ± 0.00	**0.013**
Sleep onset latency (min)	-0.01 ± 0.01	0.602
PSQI global score * Time * Baseline brain Aβ	0.07 ± 0.03	**0.004#**
PSQI global score * Time	-0.05 ± 0.02	**0.009**
PSQI global score	-0.02 ± 0.04	0.604

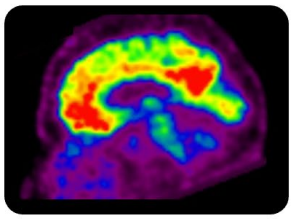
Linear Mixed Models include sleep measure, time, baseline brain Aβ (low/high), age, sex, *APOE* ε4 carrier status (-/+), DASS-21 anxiety score, premorbid intelligence as main effects, and three-way sleep * baseline brain Aβ * time between cognitive assessments interaction.
#significance following Bonferroni correction ($p < 0.008$).

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Greater sleep improvement, following the CBTi program, was associated with better or preserved executive function at 18 months, in preclinical AD



Cognitively unimpaired older adults with high brain Aβ (preclinical Alzheimer's)



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Sleep Improvement Study:
The Story So Far…

- Improved sleep quality at 18 months, following a CBT-I program, in cognitively unimpaired older adults with poor sleep and subjective memory complaint.
- Increased sleep duration and improved sleep efficiency, after the CBT-I program, were associated with better executive function at 18 months.
- Associations between increased sleep duration, improved sleep efficiency, and executive function at 18 months, observed in those with preclinical Alzheimer's disease.
- High brain Aβ burden + greater sleep improvements, after the CBT-I program, maintained or enhanced executive function over 18 months.

Rainey-Smith *et al.*, Manuscript in preparation.

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Sleep Improvement Study:
The Story So Far…

- Findings to date underscore important role for sleep in cognitive health, particularly among individuals at risk of dementia due to Alzheimer's disease (high brain Aβ).
- Findings also suggest implementation of targeted CBT-I programs for sleep improvement, in at risk individuals, may represent a viable strategy in the development of Alzheimer's prevention programs.

Rainey-Smith *et al.*, Manuscript in preparation.

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Sleep Improvement Study:
What next?

- More work to do – data collection ongoing (18 months finishing now), explore objective sleep data, blood-based biomarkers, change in neuroimaging biomarkers, etc.
- Are benefits maintained at 36 months?
- Are outcomes contingent on genotype, e.g. *APOE*, Aquaporin 4, etc?
- Leverage data to investigate related research questions:
  - Subset of SIS participants undergo Retinal Imaging – collaboration with Prof Ralph Martins, Dr Eugene Hone, Dr Purna Poudel, UWA
  - Subset of SIS participants undergo Pupillometry measurement in response to painful stimuli – collaboration with Prof Peter Drummond, MU
  - Microbiome study – collaboration with Dr Binosha Fernando, UWA

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When to Intervene?



- Understand sleep as a risk factor for suboptimal cognitive performance and detrimental brain changes **across the life course**.
- Investigate candidate mechanisms underlying the causal relationships between sleep and cognition.
- Provide insight around when to intervene and potential therapeutic targets.

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In Summary

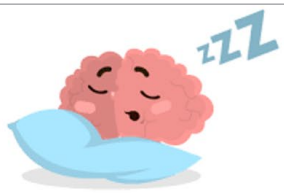


- Key biological processes occur during sleep
- Sleep is critical for both body and brain health
- However, poor sleep is common in older adults
- Poor sleep increases risk of heart disease, stroke, obesity, diabetes, depression, and dementia
- Some of this risk appears to be modifiable:
  - For example, evidence that intervention to improve sleep is likely beneficial for maintaining memory and thinking and brain health in older adults

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Key Considerations



- Screen for sleep quality in health clinics, and at home if possible.
- Understand the cause of poor sleep to determine most appropriate next steps.
- Try CBT-I before sleep medications, where suitable.
- Treat sleep apnoea to reduce risk of heart disease, stroke, and dementia.
- Improving education on good sleep habits is beneficial to all navigating the modern world.
- **Quality sleep is critical for both physical and brain health and addressing sleep problems should be prioritised.**

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Acknowledgements

Prof. Ralph Martins, UWA, MqU
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Dr Louise Pivac, Murdoch University
Dr Alex Mladenovic, UWA

Prof. Victor Villemagne, UPitt
Dr Vincent Doré, CSIRO
Dr Ying Xia, CSIRO

Dr James Doecke, CSIRO

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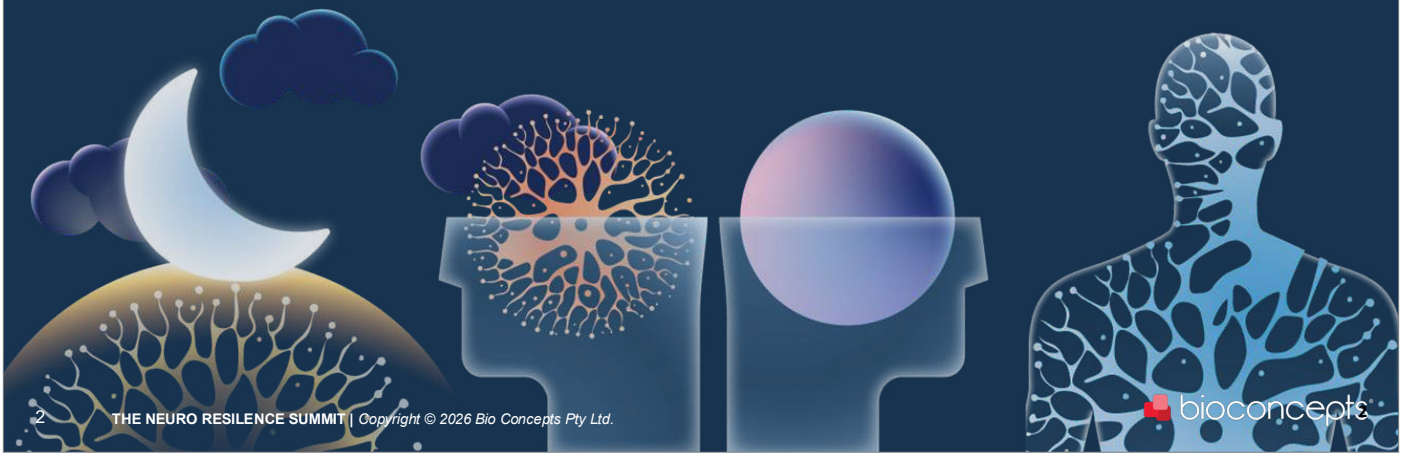
Q&A
Q&A
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Q&A

FEATURING

SUE CAMP

ASSOC PROF STEPHANIE RAINEY-SMITH

The Missing Link in Neuro-Resilience



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What is the most essential nutrient?

Mg

NAD+

CoQ

Saffron

Oxygen

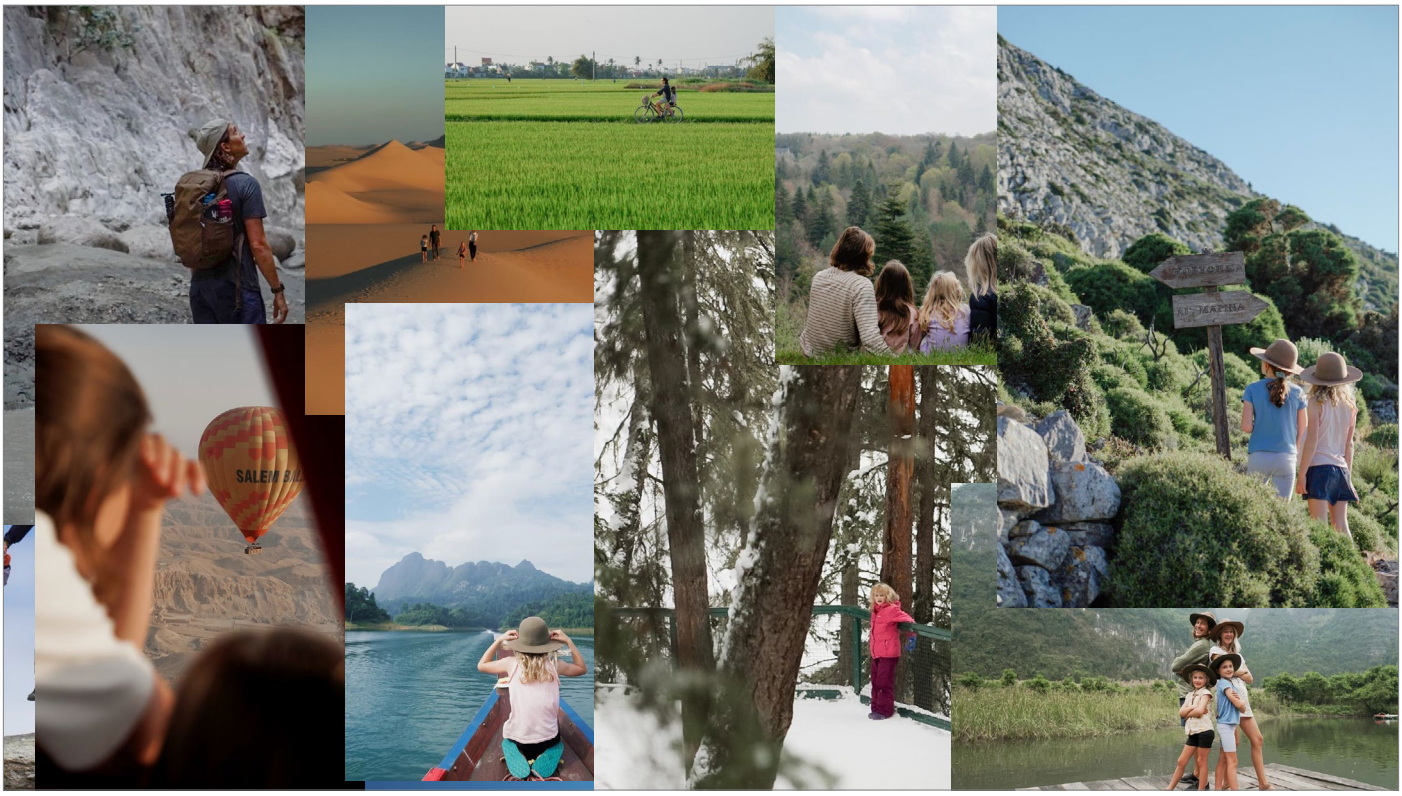
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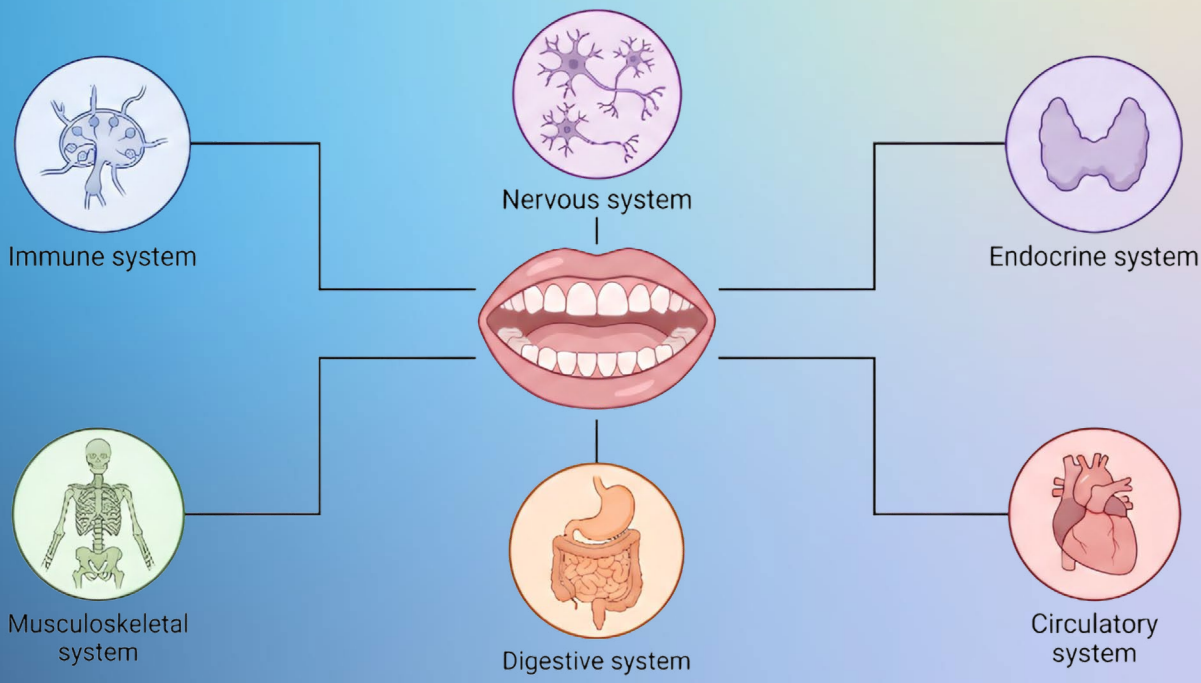


OUTLINE

1. What you're not seeing — the structural story
2. What it's costing them — the systemic consequence
3. What you can look for — the practitioner toolkit
4. How we work together — the interdisciplinary model
5. Why it matters — the smile

"Stress is the nonspecific response of the body to any demand."

— Hans Selye (1936)



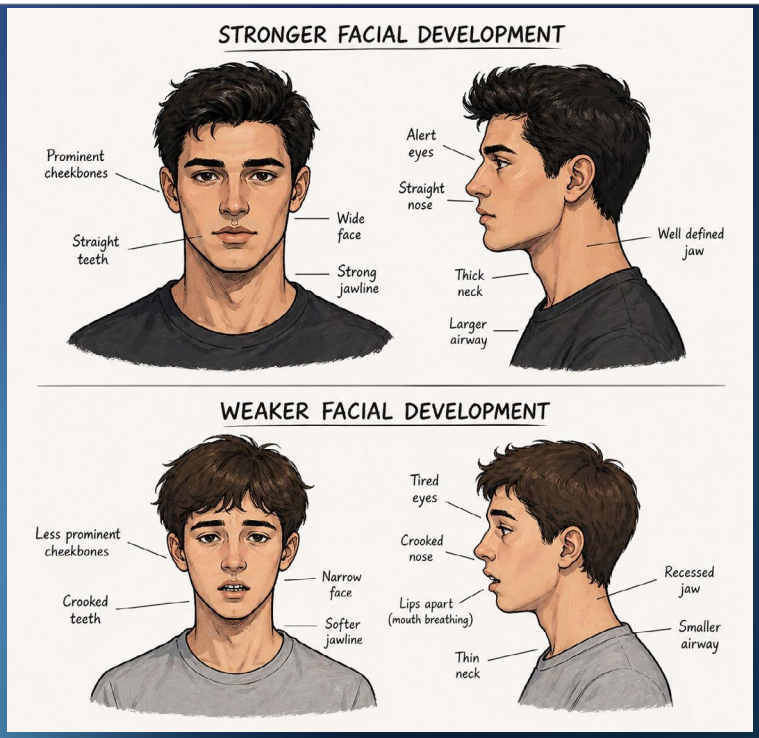
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What you're not seeing…

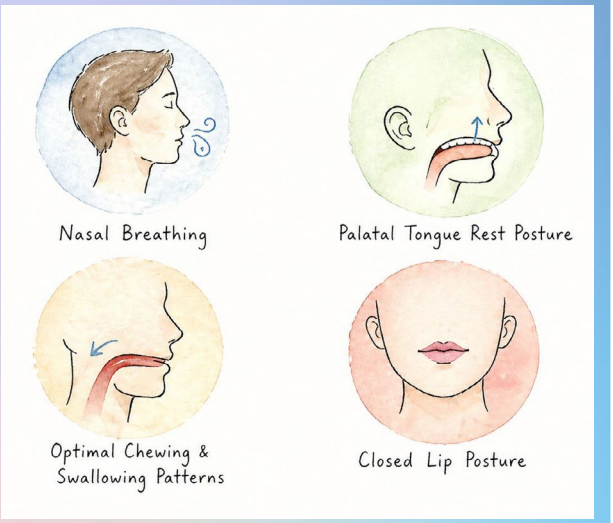
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Habits for Healthy Facial & Airway Development

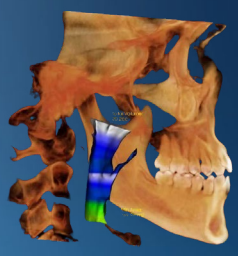
- Breath through the nose
- Tongue rests in the roof of the mouth
- Chew well
- Swallow without moving lips or cheeks
- Lips together at rest

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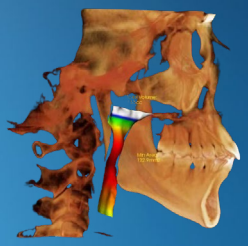
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Chewing

- Jaws need mechanical stimulus for growth
- Soft Food = narrow jaws in all dimensions
- Narrow jaw creates narrow airway
- Chewing promotes cognitive health
- Signals safety to nervous system through trigeminal-vagal pathways



Healthy Airway

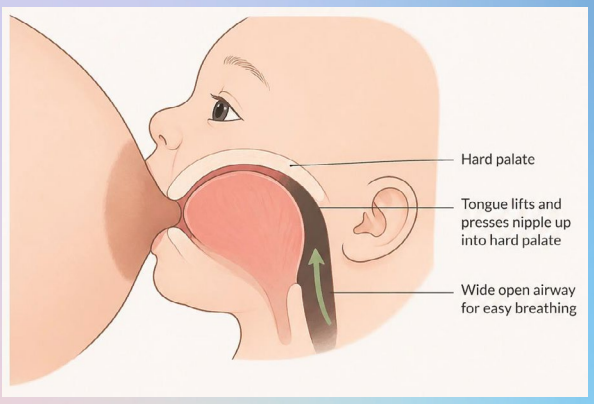


Compromised Airway

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Breastfeeding

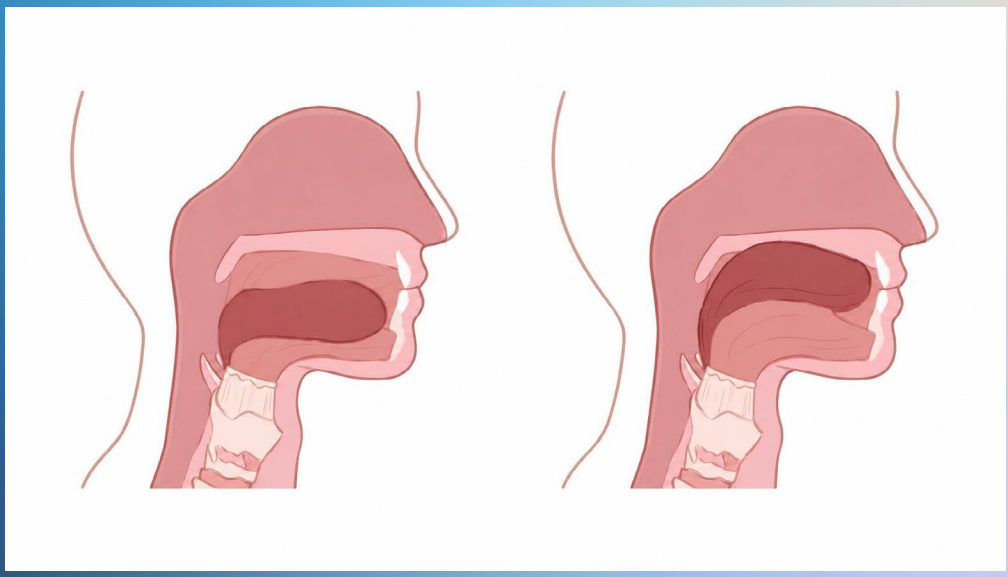


- Breastfeeding is early chewing and muscle training
- Pressure from sucking helps to form a broad flat palate
- Compromised by tongue ties, cranial strain from birth mechanics and other factors
- Bottle feeding doesn't require same muscle action.

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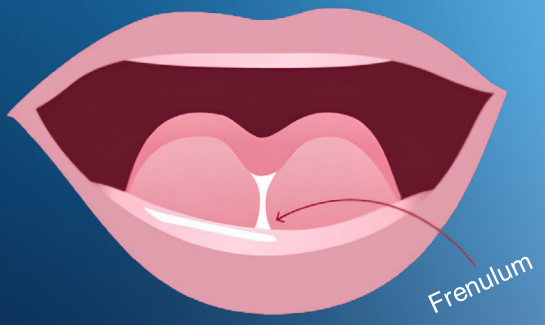
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The tongue shapes the palate… Or it doesn't



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Tongue Ties

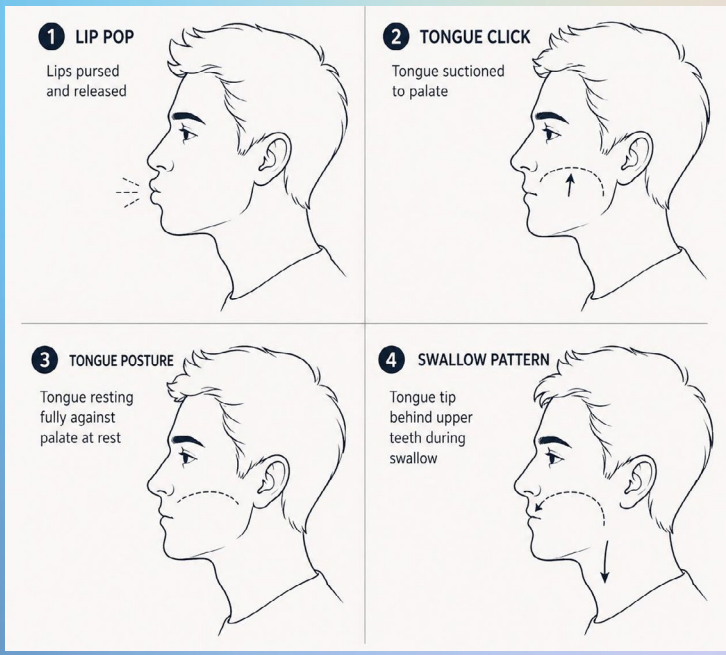


- Open wide, without closing, can your tongue tip touch the roof of your mouth?
- MTHFR variants → impaired folate metabolism
- Mass Folic acid (synthetic) supplementation vs Specific Methylfolate
- Emerging association: midline restrictions, including tongue ties, with excess circulating folate.
- Mid-line Fascial Strain Patterns create postural changes.

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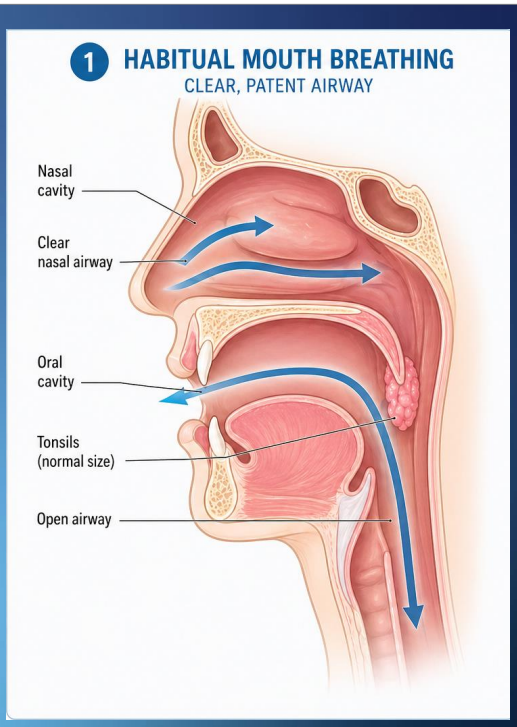
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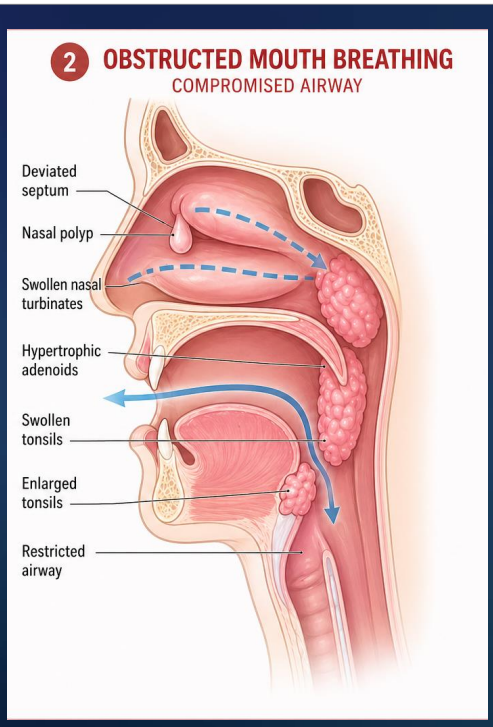
Oral Myofunctional Activities



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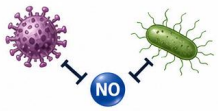
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1 **Nasal NO Production**

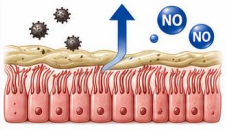
NO is generated in the paranasal sinuses and carried to the lungs with each breath.

Antimicrobial defence



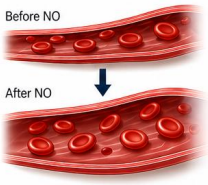
NO inhibits viral replication and bacterial growth in the upper airway.

Mucociliary clearance

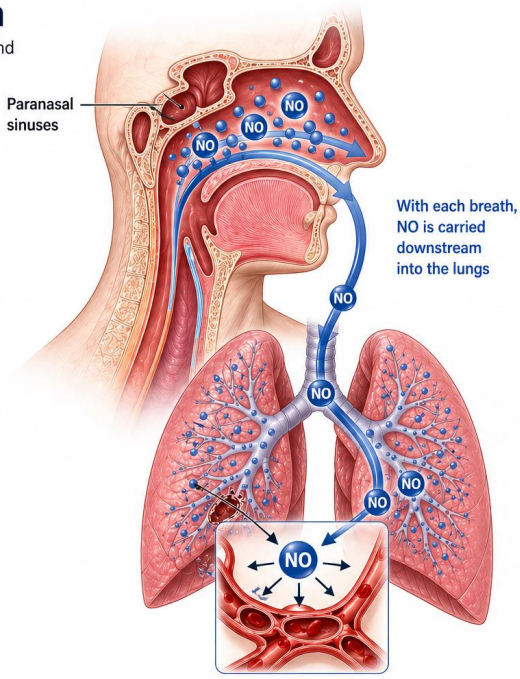


NO supports ciliary function, clearing pathogens and debris.

Pulmonary capillary vasodilation

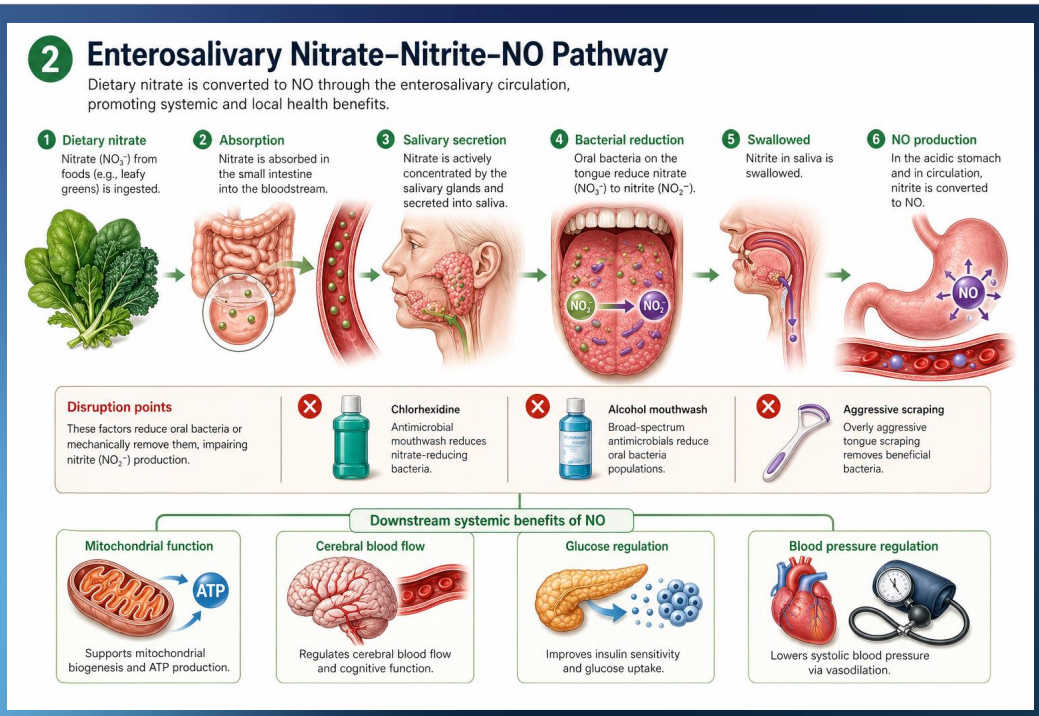


NO **promotes** smooth muscle relaxation and vasodilation, improving blood flow and oxygenation.



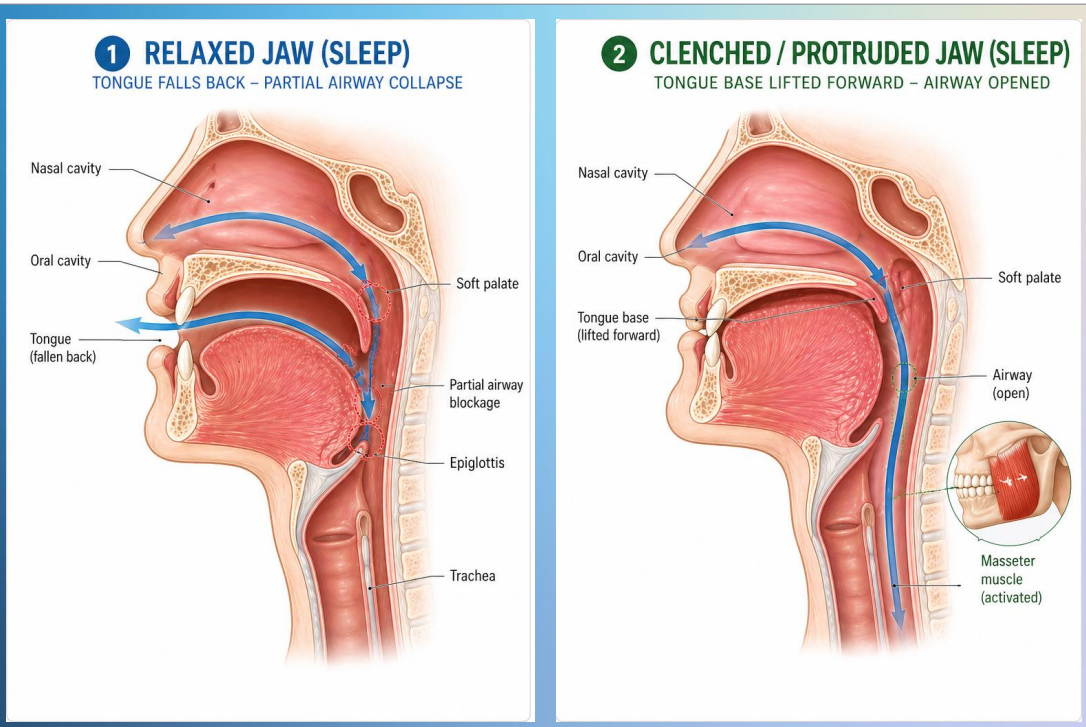
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What it's costing them…

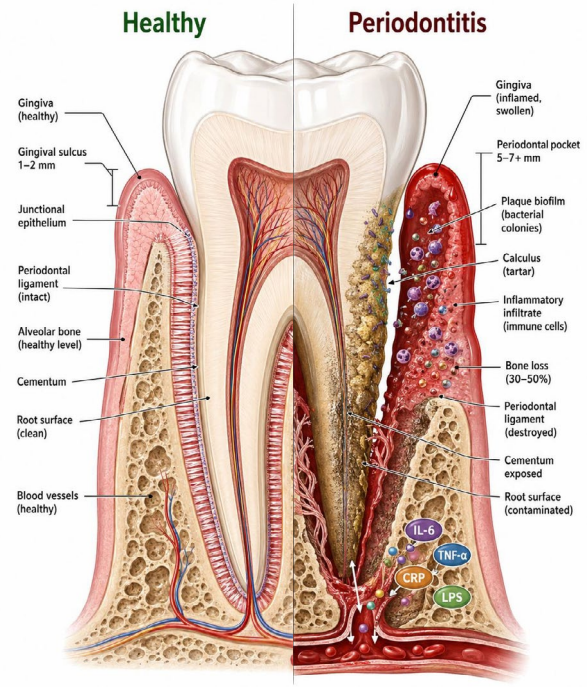
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Periodontal Disease

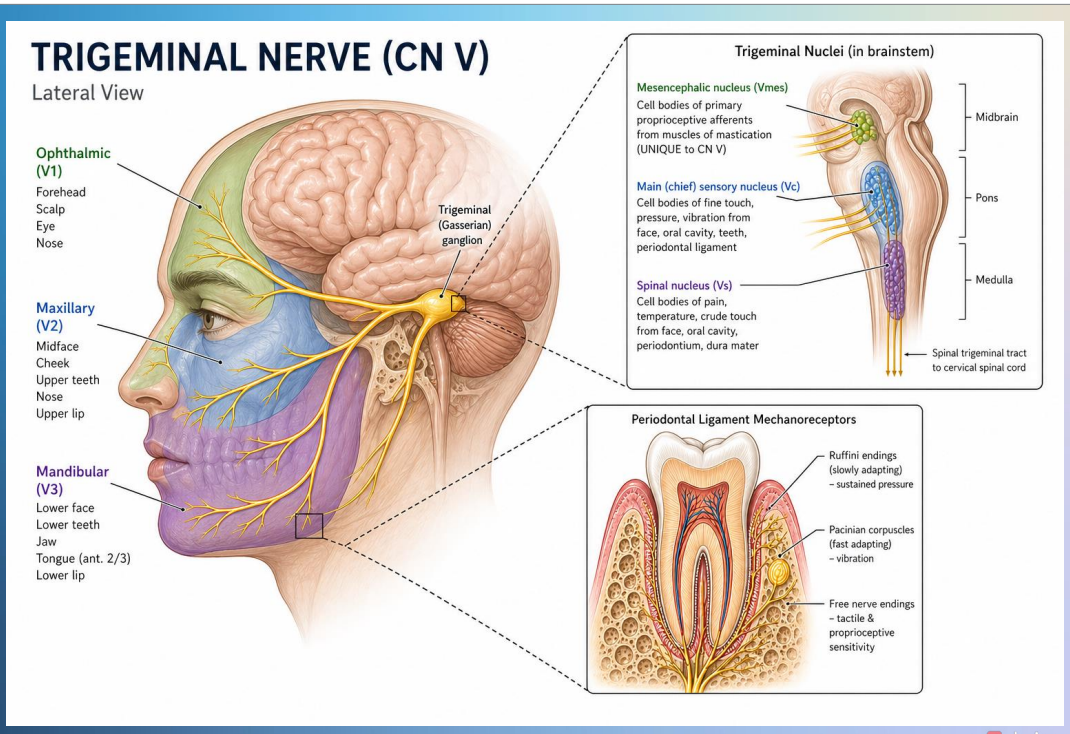
- From 2004-2018 Australia saw a 47% increase in moderate-severe disease

- All-cause mortality: ↑ 46%
- Cardiovascular mortality: ↑ 47%
- Cancer mortality: ↑ 38%
- Coronary heart disease mortality: 2x
- Cerebrovascular mortality: 3x
- Diabetes-related mortality: 3.2x



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Improving Vagal Tone

- Breathwork
- Cold exposure
- Meditation
- HRV training
- Supplements (omega-3, magnesium)
- Singing/humming

- Mouth stabilisation?

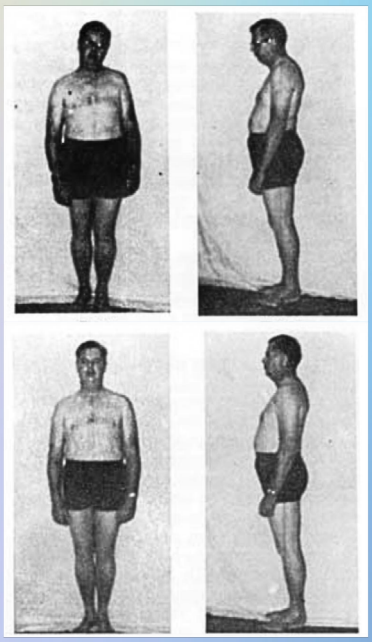
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Dental Distress Syndrome

Dr Alred Fonder (1916-2003) – “The Dental Physician”

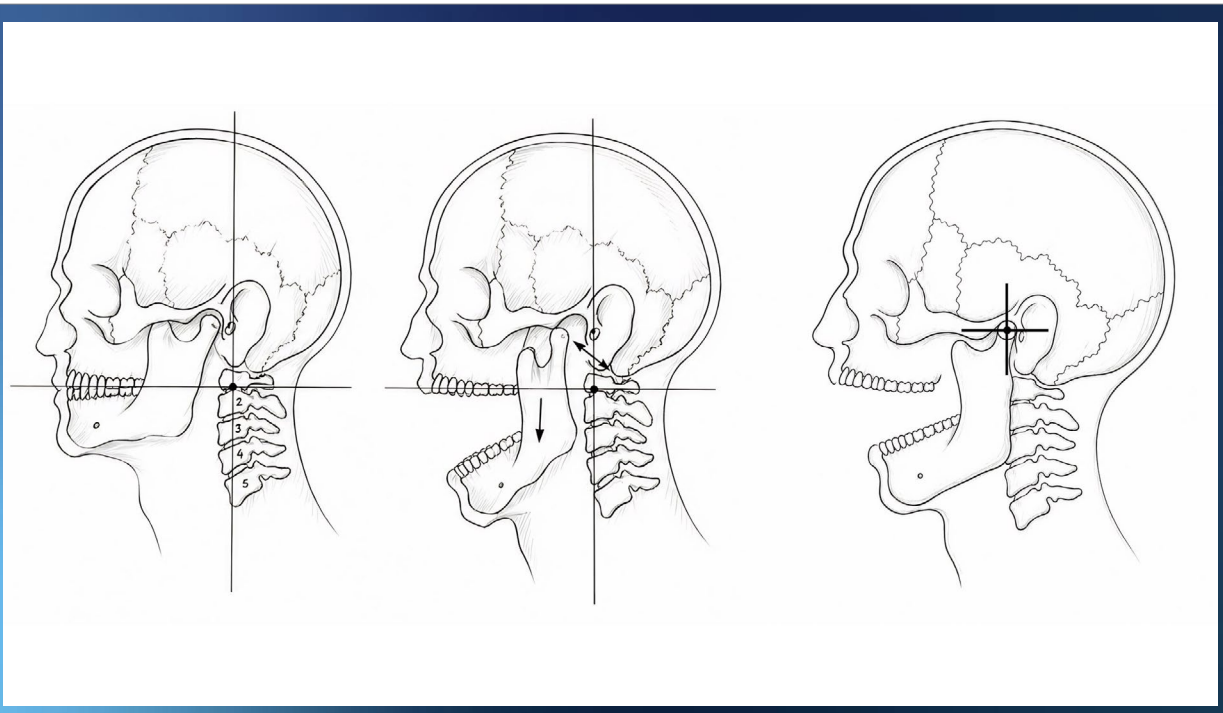
Worked with Dr Hans Selye to described the myriad of effects that stabilising the jaw had on the body

- Hormone regulation
- Gut function
- Circulation
- Epilepsy/seizure activity
- Hypothyroidism
- Chronic pain
- Dysautonomia
- Postural changes (incl. scoliosis)



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Cervicalvagopathy

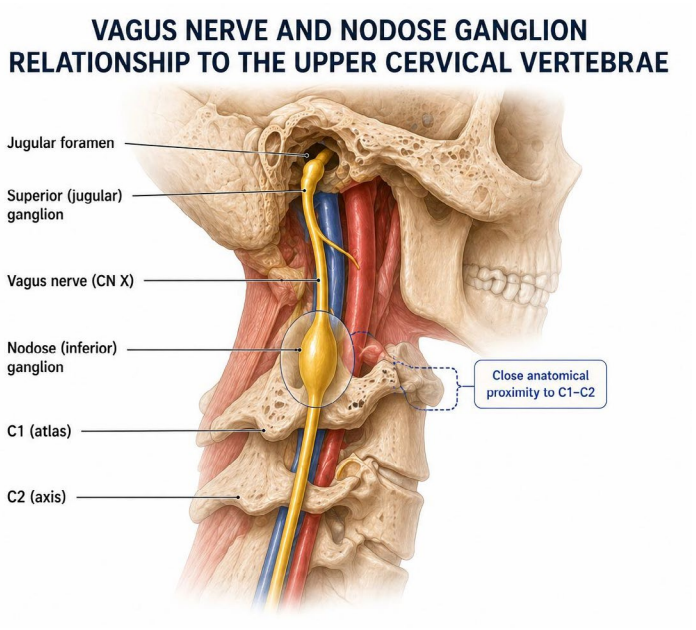
THE NODOSE GANGLION *Inferior vagal ganglion*

- Contains cell bodies of the vagus nerve's **sensory** neurons
- ~80% of vagal fibres are afferent (body → brain)
- Relay point for **interoception** — heart, lungs, gut, airway

C1/C2 COMPROMISE

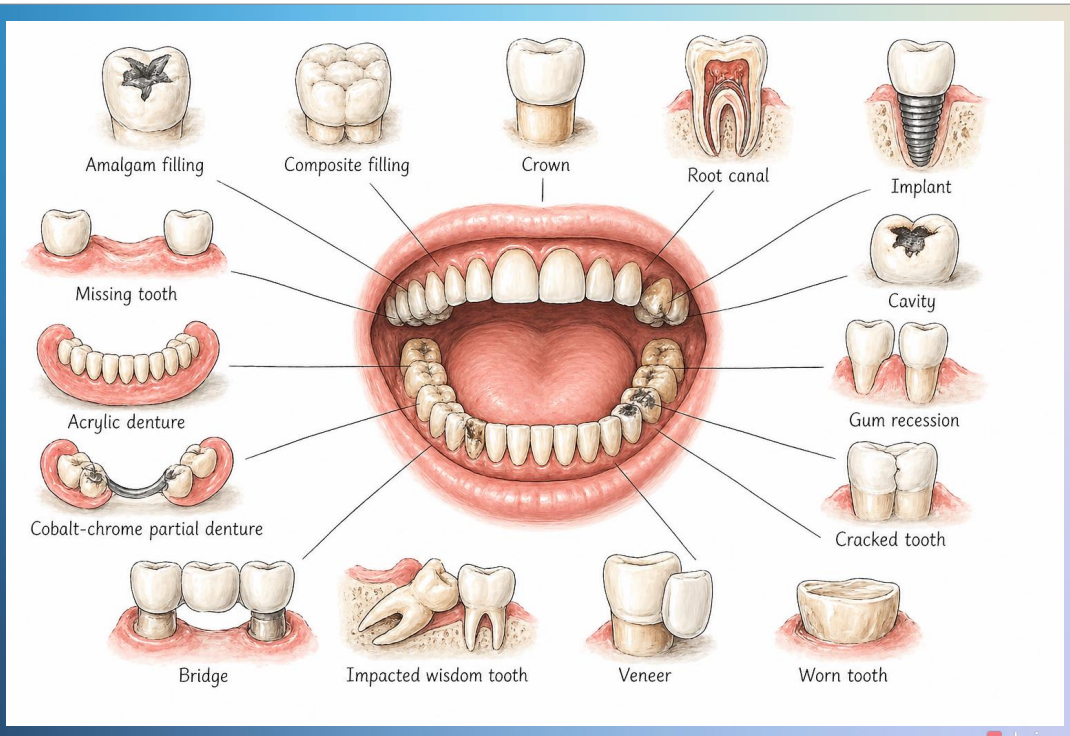
Upper cervical instability or restriction can mechanically impinge the nodose — degrading vagal afferent signal before it reaches the brainstem.

→ Impaired interoception → Disrupted autonomic regulation



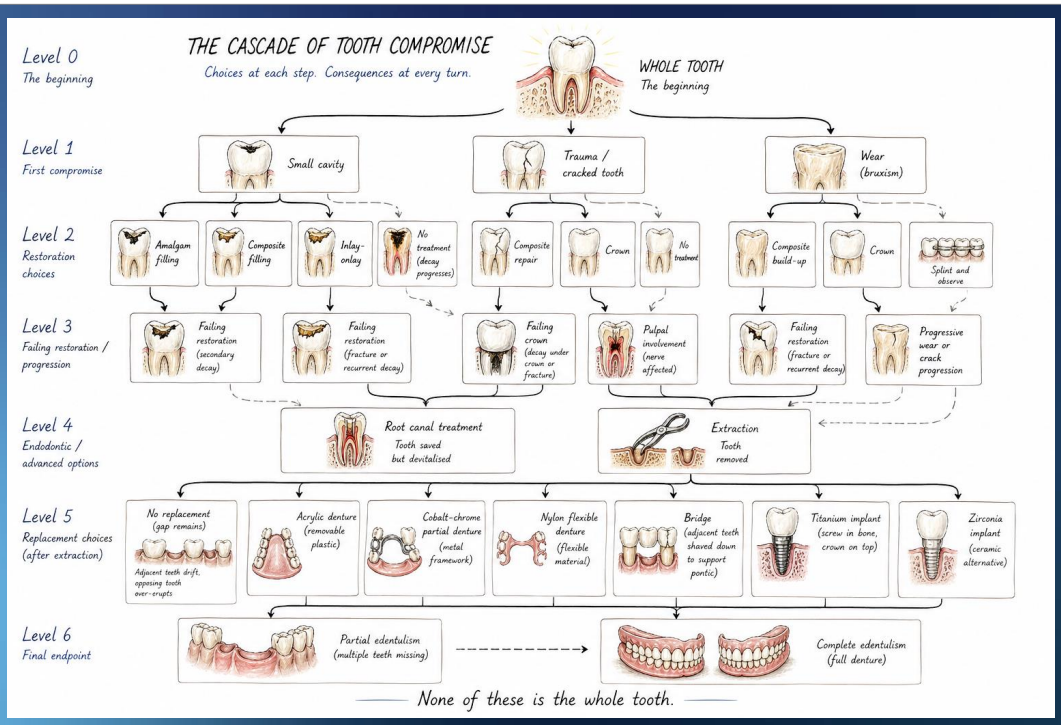
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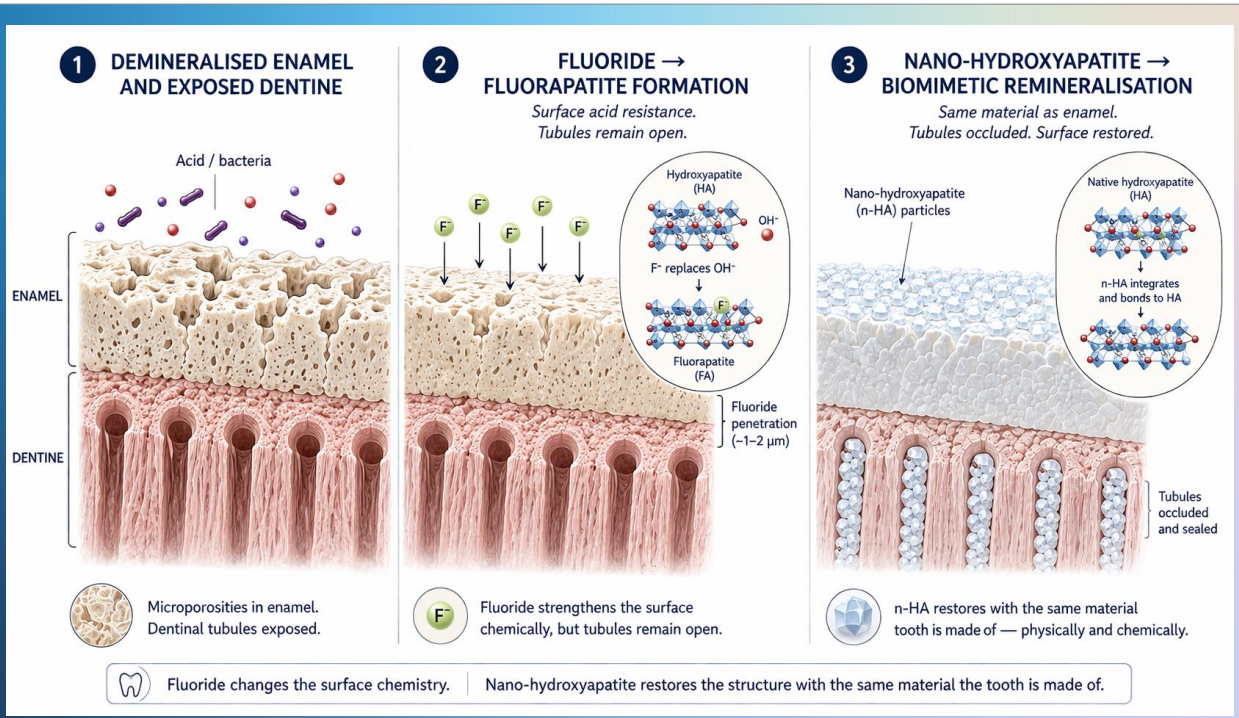
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THE CASCADE OF TOOTH COMPROMISE
Choices at each step. Consequences at every turn.
Level 0
The beginning
WHOLE TOOTH
The beginning
Level 1
First compromise
Small cavity
Trauma / cracked tooth
Wear (bruxism)
Level 2
Restoration choices
Amalgam filling
Composite filling
Inlay-onlay
No treatment (decay progresses)
Composite repair
Crown
No treatment
Composite build-up
Crown
Splint and observe
Level 3
Failing restoration / progression
Failing restoration (secondary decay)
Failing restoration (fracture or recurrent decay)
Failing crown (decay under crown or fracture)
Pulpal involvement (nerve affected)
Failing restoration (fracture or recurrent decay)
Progressive wear or crack progression
Level 4
Endodontic / advanced options
Root canal treatment
Tooth saved but devitalised
Extraction
Tooth removed
Level 5
Replacement choices (after extraction)
No replacement (gap remains)
Adjacent teeth drift, opposing tooth over-erupts
Acrylic denture (removable plastic)
Cobalt-chrome partial denture (metal framework)
Nylon flexible denture (flexible material)
Bridge (adjacent teeth shaved down to support pontic)
Titanium implant (screw in bone, crown on top)
Zirconia implant (ceramic alternative)
Level 6
Final endpoint
Partial edentulism (multiple teeth missing)
Complete edentulism (full denture)
None of these is the whole tooth.


1 DEMINERALISED ENAMEL AND EXPOSED DENTINE
Acid / bacteria
ENAMEL
DENTINE
Microporosities in enamel.
Dentinal tubules exposed.
2 FLUORIDE → FLUORAPATITE FORMATION
Surface acid resistance.
Tubules remain open.
Hydroxyapatite (HA)
OH⁻
F⁻ replaces OH⁻
Fluorapatite (FA)
Fluoride penetration (~1–2 µm)
Fluoride strengthens the surface chemically, but tubules remain open.
3 NANO-HYDROXYAPATITE → BIOMIMETIC REMINERALISATION
Same material as enamel.
Tubules occluded. Surface restored.
Nano-hydroxyapatite (n-HA) particles
Native hydroxyapatite (HA)
n-HA integrates and bonds to HA
Tubules occluded and sealed
n-HA restores with the same material tooth is made of — physically and chemically.
Fluoride changes the surface chemistry.
Nano-hydroxyapatite restores the structure with the same material the tooth is made of.

What you can look for…

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AIRWAY & ORAL FUNCTION SCREENING: QUICK VISUAL CHECKLIST

1 Mouth breathing at rest — lips apart



2 Smile width and arch shape

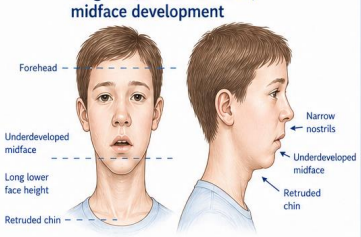


Broad smile / wider arch

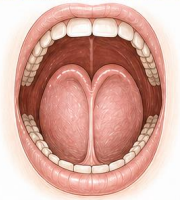


Narrow smile / narrow arch

3 Facial proportions — long face, narrow nostrils, midface development

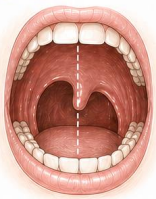


4 Tongue test — can it reach the palate? Heart-shape on lift?



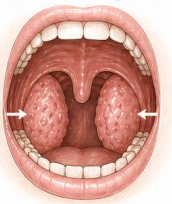
Tongue lifts but tip is restricted and pulls into a heart shape (positive for tongue tie).

5 Uvula symmetry on "Ahhh"



Uvula deviates to the left. Soft palate drops slightly on the right side.

6 Tonsil size — close to touching?



Tonsils enlarged, close to touching.

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What to Ask…

- Do teeth all fit? Were wisdom teeth removed or extractions for orthodontics?
- Snoring — patient, partner, children?
- Sleep quality? Nocturia?
- Fatigue despite adequate sleep hours?
- Bleeding gums?
- Jaw pain, clicking, locking? Headaches? Neck pain? Chronic tightness through head/neck/throat?
- Mouthwash, toothpaste, fluoride, tongue scraping?

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What to suggest…

If Mouth breathing:

- Bring awareness
- Anti-inflammatory diet
- Serrapeptase (mucolytic)
- Intranasal probiotics
- Xylitol or saline nasal flushes
- Homeopathic/grapefruit seed sprays

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Caring for the Mouth

- Antiseptic mouthwash →
  - Coconut oil pulling
- Fluoride toothpaste →
  - Nano-hydroxyapatite
- Manual brush →
  - Ultrasonic toothbrush
- String floss →
  - Water flosser

- Start chewing more!
- Breathe through your nose
- Tongue up in roof of mouth
- Lips together at rest
- Swallow without moving cheeks or lips

- See a hygienist occasionally
  - Air-polishing biofilm cleaning

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How we work together…

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A real conversation…

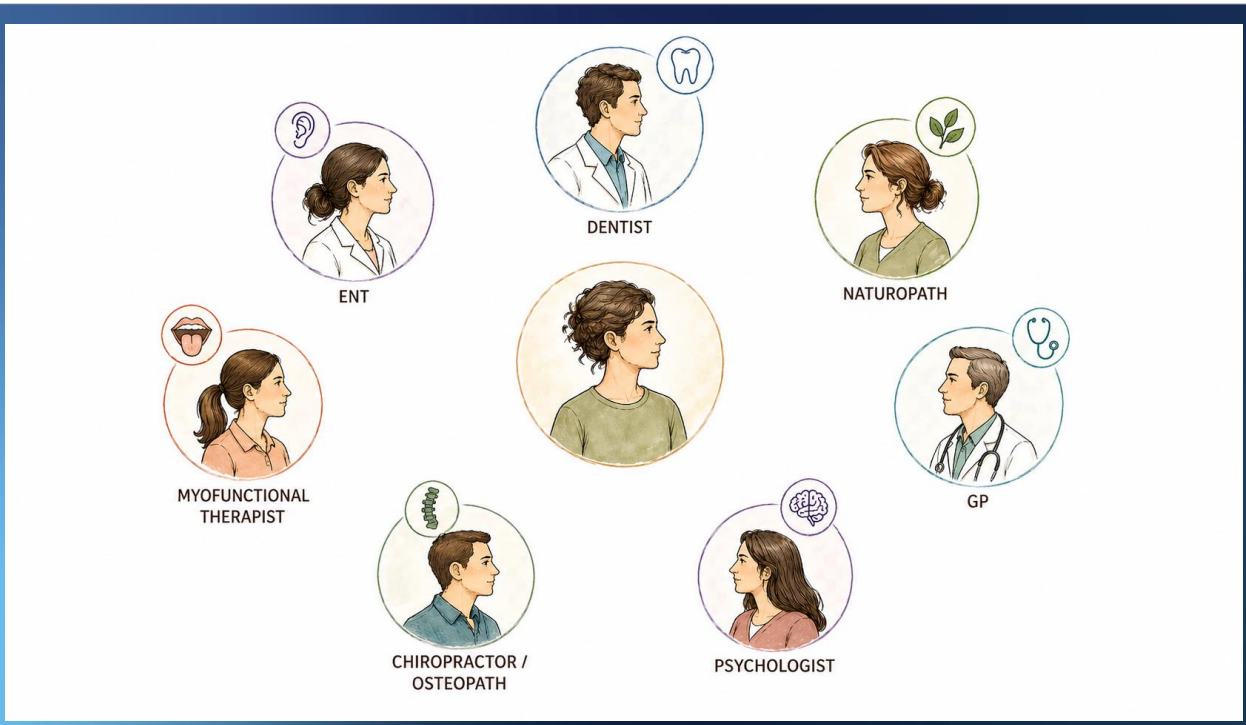
What she had

- Mum of three, recent ADHD dx
- Consistently poor sleep score
- Bad breathing at night
- Sore neck, tight jaw
- Headaches/migraines with aura
- Hormonal & gut issues
- Three root canals
- Concerned the root canals are killing her

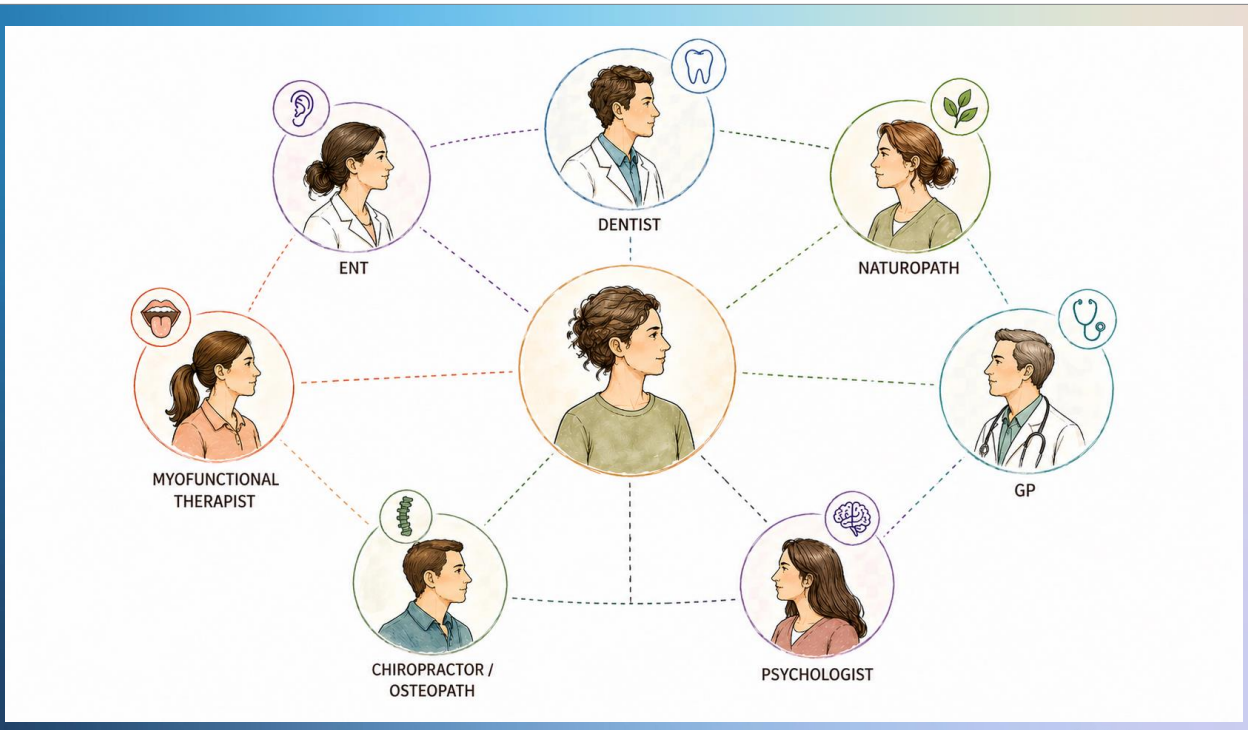
What she needed

- Capacity acknowledged first
- Myofunctional therapy before surgery
- Nutritional prep for septum repair
- Continued work with naturopath
- Tongue tie assessment
- Root canals as buying time, not crisis
- Sequenced plan, not panic

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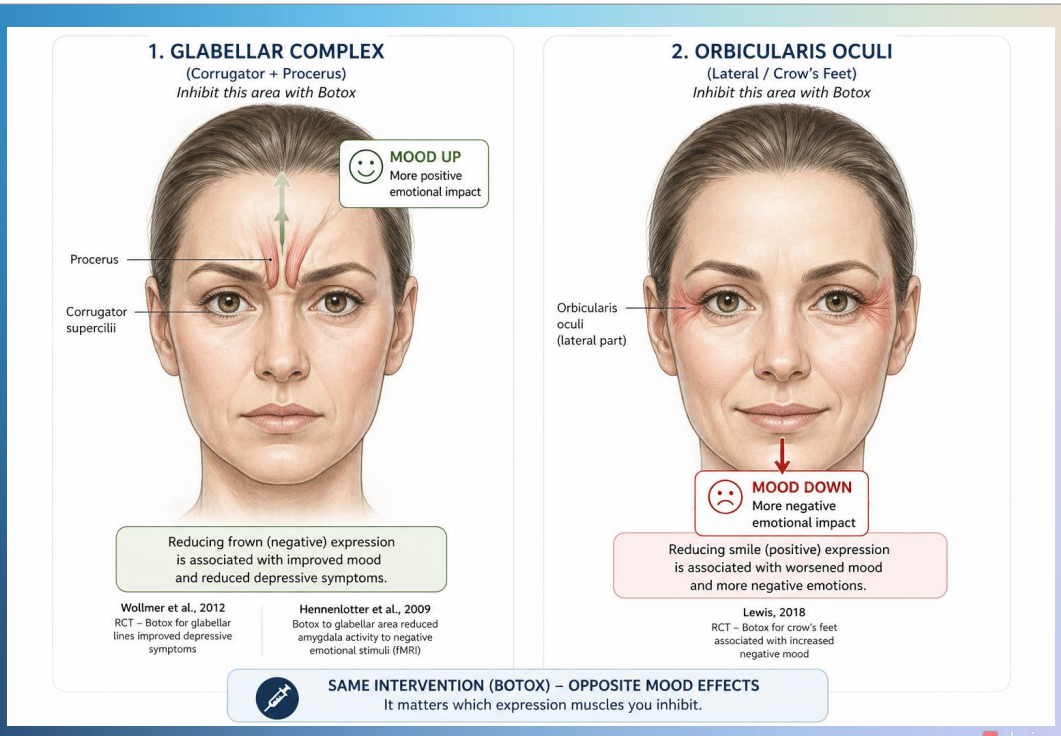


"You've got a lifetime to spend with your body. One body. One Jaw. One set of teeth."

Not urgent intervention — sustained orientation.

The Smile

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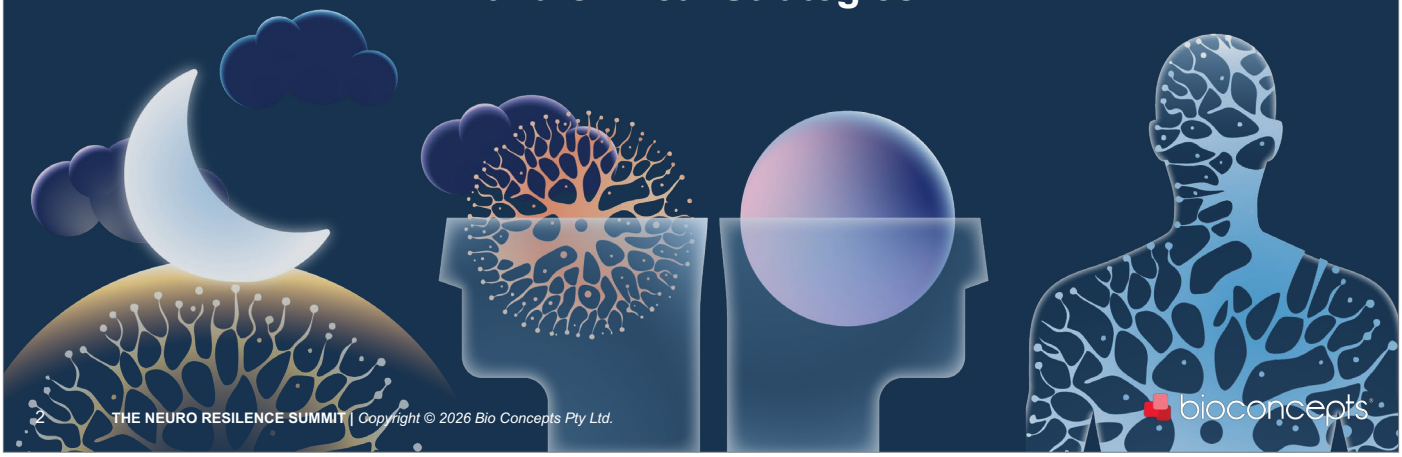
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Smiling isn't just expression.
It's regulation.

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A Functional Medicine Perspective of MS

Understanding **Root Causes**, **Neuroinflammation**, and **Clinical Strategies**



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SPEAKER

Jo Gamble

Trailblazing Functional Medicine Practitioner



The first UK graduate of the Institute for Functional Medicine (IFM), Jo is the founder of Embracing Nutrition.

She is an internationally recognised speaker, clinical mentor, and educator with over two decades of experience translating cutting-edge functional medicine science into practical clinical strategies.

Clinical focus areas:

- Root-cause resolution for chronic disease
- Autoimmunity and immune regulation
- Gut health and the gut-brain axis
- Hormonal balance and neuroinflammation
- Integrative oncology

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OUTLINE

1. **The MS burden**
   Why MS is a growing global challenge and why a functional medicine approach matters now more than ever
2. **Neuroinflammation**
   From gut to brain - the cascade of inflammation that drives MS and how peripheral triggers reach the CNS
3. **BBB & microglia**
   The blood-brain barrier as gatekeeper, and how microglia priming explains vulnerability to flares
4. **Molecular mimicry**
   Environmental triggers and how gut bacteria can cross-react with myelin to drive autoimmune attack
5. **Dietary evidence**
   Dr Terry Wahls' WAVES trial and the clinical evidence for nutritional intervention in MS
6. **Clinical strategies**
   Systematic 7-step functional medicine framework for MS: from gut repair to neuroprotection
7. **Nutraceutical support**
   Evidence-based application of key nutrients & Bio Concepts practitioner formulas in MS management
8. **Case study**
   Real-world clinical case demonstrating the functional medicine approach to MS in practice

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The MS Landscape

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MS is a Growing Global Challenge

Multiple sclerosis is a **chronic immune-mediated neuroinflammatory** condition affecting millions worldwide.

24%

Inc in Australians

Living with MS from 2017 to 2021 — and prevalence continues to rise globally.

80%

Affected by Fatigue

Fatigue is the most common and debilitating symptom in MS patients.

>50%

Modify Their Diet

Of MS patients modify their diet — often without evidence-based practitioner guidance.

 Copyright © 2026 Bio Concepts Pty Ltd. Reference: Irish et al., Mult Scler J Exp Transl Clin, 2021

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Fatigue: The Most Debilitating Symptom

An unmet clinical need

- **Fatigue affects up to 80% of MS patients** and is often reported as more disabling than mobility loss
- Pharmacological treatments for MS-related fatigue have **limited efficacy** and **significant side effect profiles**
- Fatigue drives **loss of employment, social withdrawal,** and **reduced quality of life**
- Functional medicine offers a **systems-based framework** to address **root causes — not just symptoms**

PATIENT IMPACT

80%

Of MS patients experience debilitating fatigue

"Fatigue is often the earliest and most persistent symptom, and the one that most limits daily functioning."

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MS is a Process, Not an Event

Like most autoimmune conditions, **MS develops progressively through stages — each with distinct clinical opportunities.**

STAGE 01

SILENT IMMUNE DYSREGULATION

Genetic susceptibility combines with environmental triggers to begin silently shifting immune balance long before clinical disease emerges.

Key features:

- Th17 dominance emerging
- Reduced Treg (regulatory T-cell) function
- Gut microbiome shifts begin
- Early nutrient depletion (vitamin D, omega-3)

DISEASE PROGRESSION



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MS Develops Through Stages

Stage 2 is where subclinical neuroinflammation begins — and where functional intervention has enormous impact.

STAGE 02

SUBCLINICAL NEUROINFLAMMATION

Peripheral inflammation begins to breach the CNS. Symptoms may still be vague — brain fog, fatigue, unexplained neurological complaints.

Key features:

- Increased intestinal permeability ("leaky gut")
- Systemic inflammation, elevated cytokines
- Early BBB compromise
- Microglia priming begins

DISEASE PROGRESSION



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The Functional Medicine Opportunity

STAGE 03

CLINICAL MS

Frank demyelination and relapsing-remitting or progressive disease manifest. However, **functional medicine intervention remains highly relevant:**

- **Reduce relapse frequency and severity through root-cause resolution**
- **Slow progression from relapsing-remitting to secondary progressive disease**

KEY MESSAGE

The functional medicine opportunity **exists at every stage — but is greatest in stages 1 and 2**, where intervention can prevent progression before irreversible neurological damage occurs.

DISEASE PROGRESSION



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From Gut to Brain

The Neuroinflammation Cascade

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The Gut Houses 70-80% of the Immune System

The gut is not peripheral to MS — it is central to it.

The gut-associated lymphoid tissue (GALT) is the largest immune organ in the body — training and regulating immune cells that circulate systemically.

In MS, the gut is not simply a target of inflammation. It is the **upstream driver** that connects environmental triggers to central nervous system autoimmunity.

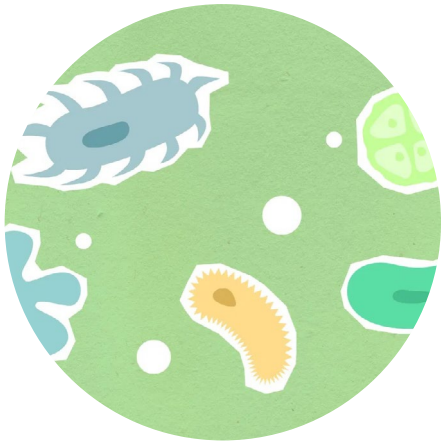


Image: Pexels (royalty-free). References: Parodi, Front Immunol, 2021; Ren, Front Immunol, 2025.

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Gut Dysbiosis is a Hallmark of MS

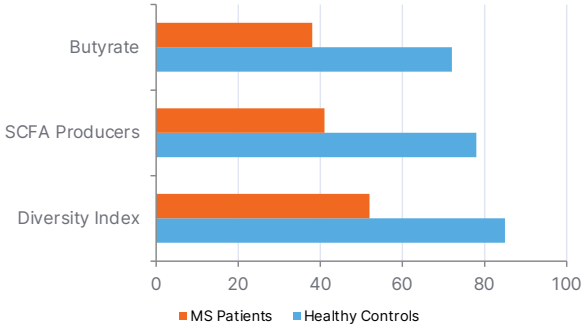
Reduced diversity, reduced protection

Multiple studies have consistently shown that people with MS have:

- **Reduced microbial diversity** compared to healthy controls
- **Reduced abundance** of beneficial SCFA-producing bacteria (Faecalibacterium, Prevotella)
- **Increased pro-inflammatory taxa** (Akkermansia, Acinetobacter in some cohorts)
- **Reduced butyrate** — critical for gut barrier AND BBB integrity

MICROBIAL DIVERSITY IN MS vs CONTROLS

Illustrative comparison (relative units)0



References: Ren et al., Front Immunol, 2025; Ghezzi et al., JCI, 2021; Parodi & Kerlero de Rosbo, Front Immunol, 2021.

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Leaky Gut Drives Systemic Inflammation

A **"leaky gut"** has been consistently reported in MS and in its animal models — **it is the mechanistic bridge.**

01
DYSBIOSIS & DIETARY TRIGGERS

Gluten, processed foods, and dysbiotic microbes damage the epithelium.

→

02
INCREASED INTESTINAL PERMEABILITY

Tight junctions loosen. Zonulin rises. LPS enters circulation.

→

03
SYSTEMIC INFLAMMATION

Cytokines, LPS, and immune activation reach every tissue.

→

04
CNS ACCESS & NEUROINFLAMMATION

BBB compromise allows immune cells and mediators to attack myelin.

> **Gut permeability is not a metaphor**
> **— it is a measurable, modifiable driver of every stage that follows.**

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Molecular Mimicry:
The Gut-Immune-Brain Link

How microbes trigger autoimmunity

- Microbial peptides from a permeable gut **can structurally resemble myelin antigens**
- The immune system, primed against these microbial fragments, **cross-reacts with myelin** in the CNS
- This "mistaken identity" drives the autoimmune attack on the myelin sheath
- Recent research (2025) confirms that gut bacterial mimicry can **accelerate MS progression**
- This explains why gut health is foundational to MS — not peripheral

THE MIMICRY CASCADE

MICROBIAL PEPTIDE → IMMUNE ACTIVATION → CROSS-REACTIVITY

MYELIN ATTACK

The immune response, targeting the microbial mimic, attacks the structurally similar myelin protein **— driving demyelination.**

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Key Clinical Insight

> **Addressing intestinal permeability and dysbiosis is not merely supportive care — it targets the initiating mechanism of autoimmune demyelination.**

This reframes gut work from adjunctive to **causal intervention** in MS.

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How Peripheral Inflammation Reaches The Brain

DIFFUSION PATHWAY

PATHWAY 01

Blood-derived cytokines passively cross the BBB at "leaky" brain regions.

Mechanism: Circumventricular organs lack a complete BBB, allowing peripheral inflammatory signals direct entry into brain tissue.

Clinical relevance: Explains how systemic inflammation can trigger central symptoms without requiring active BBB damage.

ACTIVE TRANSPORTATION

PATHWAY 02

Peripheral cytokines actively transported across the BBB by dedicated carrier systems.

Mechanism: Energy-dependent, saturable BBB transporters move cytokines into the CNS, even when the barrier is intact.

Clinical relevance: Peripheral inflammation reaches the brain by design — not just by BBB breakdown.

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Adapted from: Kharrazian D., Neuroinflammation Clinical Strategies, 2019.

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Four Pathways to Neuroinflammation

Continuing from Kharrazian — **pathways 3 & 4 of 4**

ENDOTHELIAL RECEPTOR BINDING

PATHWAY 03

Circulating cytokines bind to receptors on BBB endothelial cells, triggering release of inflammatory molecules directly into brain tissue.

Effect: BBB endothelium becomes a source of neuroinflammation itself.

VAGAL AFFERENT STIMULATION

PATHWAY 04

Peripherally produced cytokines stimulate vagal afferents (primarily from the liver), eliciting central inflammatory responses via neural signalling.

Effect: Inflammation reaches the brain neurally — even without blood-borne mediators.

CLINICAL IMPLICATION

Peripheral inflammation does not stay peripheral. Gut dysfunction, food sensitivities, and chronic infection have direct, multi-pathway access to the brain.

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The Blood-brain Barrier: Gatekeeper of The CNS

Increased BBB permeability is the core mechanism for MS.

INTACT BBB

Tight junctions between endothelial cells create a selective barrier. Only carefully regulated molecules pass into the CNS.

COMPROMISED BBB

Peripheral immune cells and inflammatory mediators gain access to the CNS. T-cells and antibodies attack myelin directly.

LPS ATTACK

Lipopolysaccharides (endotoxins) from gut permeability directly damages BBB tight junctions — creating a feed-forward inflammatory loop.

References: Ren et al., Front Immunol, 2025; Camara-Lemarroy et al., Brain, 2018.

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Leaky Gut = Leaky Brain

The parallel between intestinal and BBB permeability is not metaphorical — it is mechanistic.

BBB integrity depends on:

- Adequate SCFA production (especially butyrate)
- Low systemic inflammation
- Sufficient vitamin D (120-150 nmol/L)
- Healthy endothelial function (omega-3, glutathione)
- Intact tight junctions (protected from LPS)



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Microglia: The Brain's Immune Cells

SURPRISING FACT

The brain is **90% glial (immune) cells**

Microglia — the resident immune cells of the CNS — outnumber neurons 9 to 1.

STEADY STATE

Homeostatic Microglia

Support neuronal function. Prune synapses. Clear debris. Maintain a healthy CNS environment.

ACTIVATED STATE

Immune-Protective Microglia

Release inflammatory cytokines, ROS, and NO. Shift focus from neuronal support to defence — at the cost of neuron health.

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Microglia Priming Explains MS Flares

Once primed, microglia respond more aggressively to subsequent triggers — even minor ones.

THE PRIMING CONCEPT

An initial insult (LPS, infection, stress, food antigen) transforms microglia from steady to activated. They remain hyper-reactive — ready to overrespond to the next trigger.

Why MS patients experience flares from seemingly minor stressors:

POOR SLEEP

Sleep disruption triggers primed microglia to release inflammatory cytokines, worsening neurological symptoms.

MILD INFECTION

Even a common cold releases enough cytokines to activate primed microglia and precipitate a flare.

EMOTIONAL STRESS

Cortisol dysregulation directly influences microglial activation — connecting stress to relapse frequency.

Adapted from: Kharrazian D., Neuroinflammation Clinical Strategies, 2019.

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Recognising Neuroinflammation in your Patients

The clinical spectrum — from subtle brain symptoms to frank encephalopathy.

SUBTLE	MODERATE	SEVERE
Early/preclinical	Sickness behaviour	Encephalopathy
Brain fog, hazy recall	Depression	Delirium, confusion
Variations in mental speed	Inability to concentrate	Dementia, personality changes
Reduced brain endurance	Sleepiness, increased sleep demand	Seizures
Brain fatigue after chemical exposure	Lethargy, fatigue	Difficulty speaking
Brain fatigue after food proteins	Loss of motivation, malaise	Tremors, involuntary movements

Many MS patients present in the "moderate" category before diagnosis — creating a window for early intervention.

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Clinical Pearl: Early Recognition

PRACTICE INSIGHT

Many patients presenting with vague neurological symptoms — brain fog, unexplained fatigue, mood changes — are in the subtle-to-moderate neuroinflammation window. Recognition here creates the greatest opportunity for prevention.

Early recognition markers:

HISTORY CLUES

Post-viral brain fog, food-triggered fatigue, chemical sensitivity, exercise intolerance.

LAB PATTERNS

Low vitamin D, high hs-CRP, low B12, elevated homocysteine, gut markers.

EXAM FINDINGS

Reduced cognitive endurance testing, subtle balance changes, prolonged reaction times.

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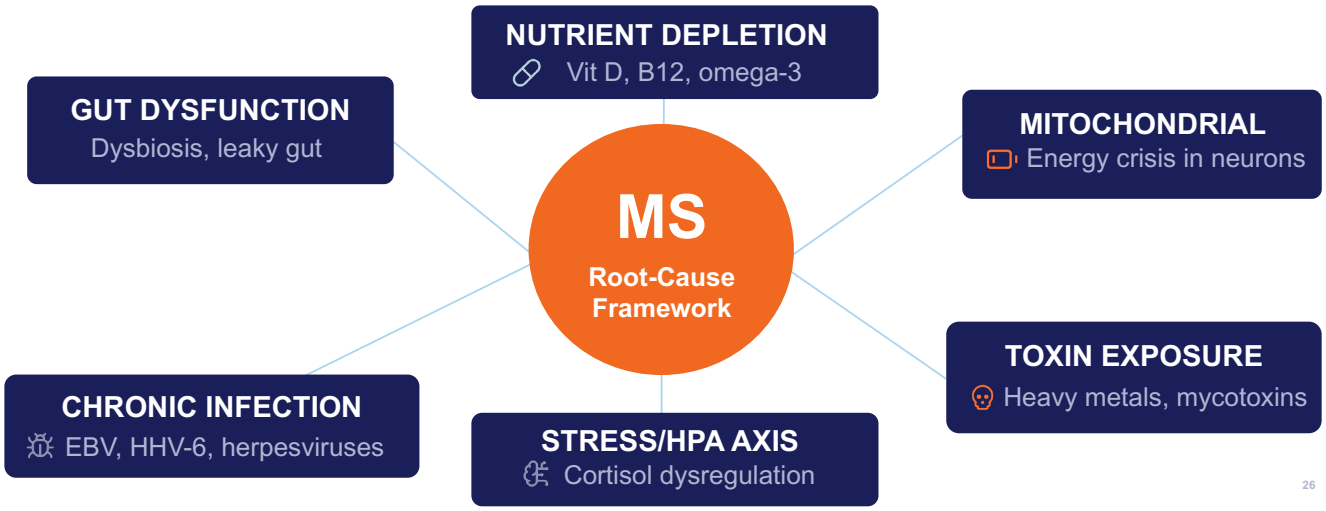
Environmental Triggers and Root Causes

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MS has Identifiable, Modifiable Root Causes

Six evidence-based root causes — each independently modifiable through functional medicine intervention.



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ROOT CAUSE 01 OF 06

Gut Dysfunction

What we see in MS patients

- **Reduced microbial diversity** and beneficial commensals
- **Depleted SCFA producers** (Faecalibacterium, Roseburia, Prevotella)
- **Increased intestinal permeability** with elevated zonulin
- **Elevated LPS/endotoxin** exposure driving systemic inflammation
- **Bacterial molecular mimicry** cross-reacting with myelin
- **Reduced tolerance** to food antigens (gluten, dairy)

CLINICAL ASSESSMENT

Investigations to consider

Stool testing
Comprehensive microbiome analysis, SCFA levels

Intestinal permeability markers
Zonulin, LPS antibodies, occludin

Food sensitivity testing
IgG panels, gluten reactivity, dairy antibodies

Systemic inflammatory markers
hs-CRP, calprotectin, cytokine panel

Ren et al., Front Immunol, 2025; Camara-Lemarroy et al., Brain, 2018.

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ROOT CAUSE 02 OF 06

Nutrient Deficiencies

Key nutrient depletions commonly identified in MS — and their clinical role.

VITAMIN D
Target: 120-150 nmol/L

Immunomodulation, T-reg support, BBB integrity, myelin protection.

OMEGA-3 EPA/DHA
Target: Omega-3 index >8%

Myelin composition, resolution of inflammation, membrane fluidity.

B12 & FOLATE
Target: Active B12 >70 pmol/L

Myelin synthesis, methylation, homocysteine control, neurological function.

MAGNESIUM
Target: RBC magnesium optimal

Nerve conduction, NMDA modulation, stress buffering, muscle function.

COQ10 & CARNITINE
Mitochondrial cofactors

Electron transport chain, ATP production, neuronal energy metabolism.

ANTIOXIDANTS
Glutathione, vitamins C & E

Neutralise oxidative stress driving demyelination and neuronal damage.

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ROOT CAUSE 03 OF 06

Mitochondrial Dysfunction

Energy crisis in the CNS

- Neurons are extraordinarily energy-demanding — 20% of body's ATP for 2% of body mass
- Demyelinated axons require 3x more ATP to maintain function
- Mitochondrial dysfunction is now recognised as a key driver of MS progression, not just a consequence
- Oxidative stress from failing mitochondria damages myelin lipids and axonal proteins
- Fatigue in MS may reflect underlying cellular energy failure — not just neurological damage

KEY MITOCHONDRIAL COFACTORS

Nutritional targets

CoQ10 (Ubiquinol)	Electron transport, complex I-III
L-Carnitine	Fatty acid transport into mitochondria
Alpha-Lipoic Acid	Universal antioxidant, PDH cofactor
Magnesium	ATP stabilisation, kinase activation
B-Complex	Krebs cycle, energy metabolism

References: Wahls TL, Curr Nutr Rep, 2019; Ghezzi et al., JCI, 2021.

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ROOT CAUSE 04 OF 06

Chronic Infections

LANDMARK EVIDENCE

32-fold

Increased MS risk following **Epstein-Barr virus infection** — establishing EBV as the leading candidate cause of MS.

Bjornevik et al., Science, 2022 — 10 million military personnel cohort

Chronic infections commonly involved:

- **Epstein-Barr virus (EBV) — molecular mimicry with GlialCAM**
- **Human herpes virus-6 (HHV-6) — active in MS lesions**
- **Chlamydia pneumoniae — vascular contribution**
- **Borrelia (Lyme) — differential + comorbid consideration**
- **Reactivation of dormant herpes viruses during stress**

CLINICAL IMPLICATIONS

- Support antiviral immunity (vitamin D, zinc, quercetin)
- Reduce EBV reactivation triggers (stress, sleep loss)
- Investigate reactivation markers: EBV EA-D, VCA IgG titres
- Support liver detoxification and immune resilience

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ROOT CAUSE 05 & 06 OF 06

Toxins & HPA Axis Dysregulation

ROOT CAUSE 05

TOXIN EXPOSURE

Common toxic burden in MS:

- Heavy metals: mercury, lead, aluminium
- Mycotoxins from water-damaged buildings
- Persistent organic pollutants (PCBs, PFAS)
- Endocrine disruptors: BPA, phthalates
- Pesticide/glyphosate exposure
- Air pollution and particulate matter

ROOT CAUSE 06

STRESS / HPA AXIS

Stress-relapse connection:

- Cortisol dysregulation drives microglial activation
- Poor sleep quality is a documented relapse trigger
- Trauma history increases MS risk (ACE studies)
- Vagal tone reduction impairs inflammation resolution
- Sympathetic dominance amplifies immune dysregulation
- HPA axis modulates BBB permeability

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Vitamin D: a Strong Modifiable Risk Factor

Serum 25(OH)D is one of the most consistently replicated modifiable risk factors in MS.

>120
nmol/L TARGET
Studies suggest 120-150 nmol/L optimal for MS risk reduction and disease modulation.

50%
REDUCED RELAPSE
Higher vitamin D levels associated with ~50% lower relapse risk in observational studies.

5000
IU/DAY TYPICAL
Common maintenance dose in MS practice — always monitor and individualise via serum testing.

MECHANISMS OF ACTION IN MS

- Immune tolerance — promotes T-regulatory cell function and suppresses Th17 pathogenic responses
- BBB support — maintains endothelial integrity and tight junction function
- Direct neuroprotection — supports oligodendrocyte differentiation and remyelination

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The Evidence for Dietary Intervention

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Dr Terry Wahls: From Wheelchair to Bicycle

A physician's own case

Diagnosed with **secondary progressive MS in 2003**, Dr Terry Wahls — an academic physician at the University of Iowa — deteriorated to needing a tilt-recline wheelchair.

Standard-of-care treatment was failing. She turned to **functional medicine principles** and designed a nutrient-dense diet targeting her mitochondria.

Within 12 months, she was **walking unaided**. Within 18 months, she was **cycling 29 kms**.

She became one of the most cited researchers in dietary intervention for MS — with multiple published RCTs.

"Food is the most powerful tool we have to change disease outcomes — and we have not been using it."

DR TERRY WAHLS

Clinical Professor, University of Iowa

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The Wahls Protocol

A modified Paleolithic elimination diet targeting mitochondrial support and immune modulation.

Daily "9 cups" targets (~2000 kcal reference)

3 CUPS DAILY
Leafy greens
Kale, spinach, chard, collards — folate, magnesium, chlorophyll.

3 CUPS DAILY
Sulphur-rich vegetables
Broccoli, cabbage, onions, garlic — glutathione and detoxification support.

3 CUPS DAILY
Deeply pigmented produce
Berries, beets, red cabbage — polyphenols and antioxidants.

ELIMINATE

Gluten, dairy, eggs, refined sugars, processed seed oils, all processed foods.

PRIORITISE

Wild-caught seafood, grass-fed meats, organ meats, seaweed, bone broths, healthy fats.

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The Waves Trial

Wahls versus Swank Study — a landmark randomised trial of dietary intervention in MS-related fatigue.

DESIGN
RCT
Randomised controlled trial with usual-care control arm

PARTICIPANTS
N=87
RRMS patients with elevated fatigue

DURATION
24 wks
With follow-up at 36 and 48 weeks

ARMS
3-ARM
Wahls diet vs Swank diet vs usual care control

PRIMARY QUESTION

Can a nutritionally-focused dietary intervention reduce MS-related fatigue?

Both diets emphasised whole foods and eliminated processed foods. The Wahls diet added a specific nutrient-density prescription (9 cups vegetables/fruits) plus mitochondrial support.

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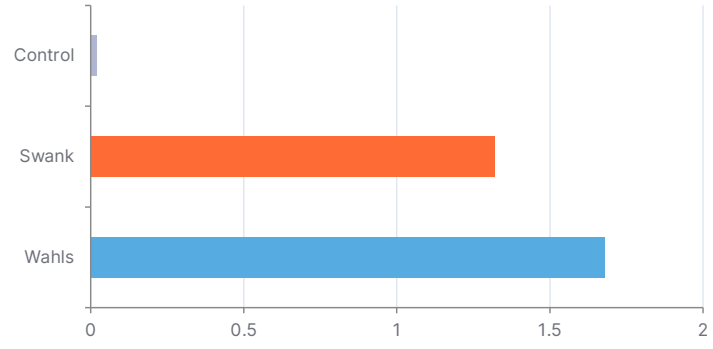
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Waves Results: Fatigue & Physical QoL

Both diet arms significantly outperformed control on primary outcomes.

FATIGUE REDUCTION (FSS at 12 weeks)

Change from baseline. Lower is better. All p<0.05 vs control.



WAHLS DIET
-1.68
FSS score reduction
Clinically meaningful; sustained across follow-up.

PHYSICAL QoL (MSQOL-54)
+6.6
Points improvement
Wahls arm; sustained at 24-week follow-up.

Reference: Wahls et al., Mult Scler J, 2021 (PMC8326636).

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Waves Results: Mental QoL & Mobility

Improvements extended beyond fatigue — into cognition, mood, and mobility.

MENTAL QoL (MSQOL-54)

+9.3

Points improvement

Sustained improvement in mood, cognitive function, and social function — beyond what fatigue reduction alone would predict.

6-MINUTE WALK

+45m

Additional distance walked

Significant improvement in mobility — a validated marker of MS progression and independence.

NUTRIENT STATUS

↑↑↑

Multi-nutrient improvement

Significant increases in serum folate, vitamin K, and multiple micronutrients — with objective biomarker improvement.

Reference: Wahls et al., Mult Scler J, 2021 (PMC8326636).

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Waves Patients Sustain the Protocol

Practitioner concern: will patients follow through? WAVES data answers unequivocally: yes.

ADHERENCE DATA

High compliance across both diet arms

Wahls diet adherence	89%
Swank diet adherence	86%
Retention at 24-week follow-up	>80%

Patients rated the diets as achievable and worthwhile — improvements in QoL sustained the commitment.

WHAT THIS MEANS CLINICALLY

Diet is a viable, sustainable therapy

- Given proper support and education, patients can and do sustain nutritional intervention
- Practitioners should not underestimate patient motivation — MS patients want tools they can actively use
- Combining diet with targeted nutraceutical support maximises outcomes
- Adherence is highest when clinical improvements are visible early

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Why the Wahls Protocol Works

Six mechanisms of action — each targeting a documented driver of MS pathology.

01
MITOCHONDRIAL SUPPORT
Delivers CoQ10, carnitine, B vitamins, and antioxidants critical for cellular energy.

02
ANTI-INFLAMMATORY
Eliminates inflammatory triggers (gluten, dairy, processed foods, seed oils).

03
MICROBIOME OPTIMISATION
Fibre-rich vegetables feed beneficial bacteria and boost SCFA production.

04
ANTIOXIDANT DENSITY
Deeply pigmented plants deliver polyphenols and flavonoids that neutralise oxidative stress.

05
OMEGA-3 & FATS
Wild seafood and grass-fed meats supply EPA/DHA for myelin support and inflammation resolution.

06
METABOLIC FLEXIBILITY
Balanced macronutrients and reduced glycaemic load improve insulin sensitivity and neural fuel use.

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Clinical Strategies & Nutraceutical Support

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The Functional Medicine MS Framework

A systematic 6-step clinical approach targeting root causes and supporting neuroresilience.

01 STEP ONE
REPAIR THE GUT
Remove triggers, restore microbial balance, heal intestinal barrier.

02 STEP TWO
RESOLVE INFLAMMATION
SPMs, curcumin, PEA — active resolution, not suppression.

03 STEP THREE
MITOCHONDRIAL SUPPORT
CoQ10, carnitine, ALA, B complex — restore cellular energy.

04 STEP FOUR
MYELIN & MEMBRANES
Omega-3s, phospholipids — supply myelin building blocks.

05 STEP FIVE
METHYLATION & ANTIOXIDANTS
B12, folate, glutathione — support detox and redox balance.

06 STEP SIX
STRESS & SLEEP
HPA regulation, adaptogens, magnesium — build resilience.

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STEP 01 OF 06 — GUT REPAIR (PART 2)

Remove & Replace

PHASE 1: REMOVE

Eliminate the drivers

- Inflammatory foods: gluten, dairy, refined sugar, seed oils
- Identified food sensitivities via IgG panels or elimination diet
- Pathogenic overgrowth: SIBO, candida, parasitic infections
- Environmental toxins: pesticides, endocrine disruptors, alcohol
- NSAIDs, PPIs, unnecessary antibiotics where clinically possible

PHASE 2: REPLACE

Restore digestive function

- Betaine HCl and digestive enzymes with meals if hypochlorhydria
- Bile support: ox bile, taurine, phosphatidylcholine
- Bitter herbs to stimulate digestive secretions (gentian, dandelion)
- Adequate hydration away from meals
- Mindful eating — vagal activation, chewing, meal spacing

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STEP 01 OF 06 — GUT REPAIR (PART 2)

Reinoculate, Repair & Rebalance

PHASE 3: REINOCULATE
Restore beneficial microbiota

- **Multi-strain probiotics** — Lactobacillus, Bifidobacterium species
- **Prebiotic fibres and resistant starches**
- **Fermented foods** — sauerkraut, kefir (if tolerated), kimchi

PHASE 4: REPAIR
Heal intestinal barrier

- **L-glutamine, zinc carnosine, N-acetylcysteine**
- **Slippery elm, marshmallow, aloe vera**
- **Colostrum and bone broth** — growth factors and glycine

SPECIFIC SUPPORT
Targeted gut repair formulas

Gut Support and Repair blend - Glutamine, zinc carnosine, quercetin, curcumin, Vitamins A and D.

Probiotics Supporting Immune Function - 20 billion CFU of *Lactobacillus rhamnosus* GG (ATCC 53103), *Lactobacillus paracasei* LP-33® and *Lactobacillus fermentum* GMNL-93.

Saccharomyces boulardii combined with B. Longum BB523
Yeast probiotic for pathogen resistance and antibiotic recovery.

Digestive enzymes
Pancreatic enzymes, combined with bromelain for additional proteolytic activity.

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STEP 02 OF 06 — RESOLVE INFLAMMATION (PART 1)

Resolution, not Suppression

THE PARADIGM SHIFT

Chronic inflammation is not just a failure of the inflammatory switch to turn off — it is a failure of the active resolution program. We must add resolution, not just remove suppression.

SUPPRESSION APPROACH
Blocking the fire

- NSAIDs, corticosteroids block key pathways
- Effective short term, but disrupts natural resolution
- Can impair healing and repair processes
- Does not address underlying drivers

RESOLUTION APPROACH
Activating clean-up

- **SPMs** (resolvins, protectins, maresins) actively terminate inflammation
- Derived from omega-3 EPA/DHA
- Promote macrophage clearance and tissue repair
- Enhance — rather than block — normal immune function

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STEP 02 OF 06 — RESOLVE INFLAMMATION (PART 2)

Key Nutraceuticals

SPECIALISED PRO-RESOLVING MEDIATORS

SPMs

Concentrated SPM mediators for active resolution.

MECHANISM

Resolvins and protectins actively signal immune cells to clear debris, restore homeostasis, and reduce microglial activation. Uniquely valuable when omega-3 alone is insufficient.

CURCUMIN

Highly bioavailable curcumin

Using the whole turmeric rhizome with improve bioavailability.

MECHANISM

Inhibits NF-κB, downregulates pro-inflammatory cytokines (TNF-α, IL-6, IL-1β), and crosses the BBB. Directly modulates microglial activation and reduces neuroinflammation.

PALMITOYLETHANOLAMIDE

PEA

Ultra-micronised PEA for endocannabinoid support.

MECHANISM

Downregulates mast cells and microglia via PPAR-α activation. Well-evidenced for neuropathic pain and neuroinflammation — with a strong safety profile.

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STEP 03 OF 06 — MITOCHONDRIAL SUPPORT (PART 1)

Restore Cellular Energy

WHY MITOCHONDRIA MATTER IN MS

Demyelinated axons need 3x more ATP to fire. When mitochondria fail, neurons enter energy crisis — driving both fatigue and progressive neurodegeneration.

ELECTRON TRANSPORT

ATP GENERATION

Support Complex I-IV with CoQ10, riboflavin, NADH — the workhorses of oxidative phosphorylation.

FUEL DELIVERY

SUBSTRATE TRANSPORT

L-carnitine shuttles fatty acids into mitochondria — the primary fuel for high-energy tissues.

MEMBRANE PROTECTION

ANTIOXIDANTS

Alpha-lipoic acid, glutathione, vitamin E — neutralise ROS produced by respiring mitochondria.

BIOGENESIS

NEW MITOCHONDRIA

Exercise, cold exposure, PQQ, and resveratrol activate PGC-1α — the master regulator of biogenesis.

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STEP 03 OF 06 — MITOCHONDRIAL SUPPORT (PART 2)

Mitochondrial Nutrients

Mitochondrial Support

Comprehensive mitochondrial support with CoQ10, alpha-lipoic acid, B-vitamins, and L-carnitine — designed for energy production and antioxidant defence.

CoQ10

Reduced form of CoQ10 for optimal bioavailability — critical for electron transport chain function and myocardial/neural energy.

Alkalising Magnesium

Multiple magnesium forms for cellular energy, muscle function, and nervous system support — essential mitochondrial cofactor.

Alpha Lipoic Acid + NAC

Universal antioxidants supporting glutathione recycling, oxidative stress mitigation, and BBB protection.

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STEP 04 OF 06 — MYELIN & MEMBRANES (PART 1)

Mitochondrial Nutrients

MYELIN COMPOSITION

70%

Myelin is 70% lipid

Cholesterol, phospholipids, sphingolipids, and long-chain fatty acids form the insulating sheath around axons.

Without adequate lipid raw materials — **particularly omega-3 EPA/DHA** — the CNS cannot repair or maintain myelin.

ROLE 01

Direct myelin substrate

DHA is the dominant omega-3 in myelin lipids and neuronal membranes.

ROLE 02

Precursor to SPMs

EPA/DHA convert to resolvins, protectins, maresins — active resolution signals.

ROLE 03

Membrane fluidity

Supports neurotransmission, synaptic plasticity, and BBB integrity.

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STEP 04 OF 06 — MYELIN & MEMBRANES (PART 2)

OMEGA-3 Evidence

THE EVIDENCE IN MS

- Lower omega-3 index in MS patients vs healthy controls
- EPA/DHA supplementation reduces MMP-9, a BBB-disrupting enzyme
- Higher fish intake correlated with reduced MS progression
- Omega-3s support oligodendrocyte differentiation and remyelination in animal models
- Target Omega-3 index >8% — with typical clinical dose 2-4g EPA+DHA/day
- Triglyceride form preferred for absorption vs ethyl esters

CONSIDERATIONS

Omega-3

High-strength EPA/DHA in triglyceride form in 2:1 ratio.

SPMs

Concentrated pro-resolving mediators from omega-3s.

Phosphatidylcholine

Membrane repair and myelin structural support.

Vitamin D3 + K2

Synergistic with omega-3s for immunomodulation.

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STEP 05 OF 06 — METHYLATION & ANTIOXIDANTS (PART 1)

Methylation & Redox Balance

Two intertwined systems supporting neurological function, detoxification, and cellular repair.

METHYLATION

The universal reaction

- Myelin synthesis — myelin basic protein requires methylation
- Neurotransmitter production and degradation (COMT, MAO)
- Homocysteine control — elevated levels are neurotoxic
- Detoxification — Phase II conjugation
- Epigenetic regulation — gene expression modulation

ANTIOXIDANT DEFENCE

Redox homeostasis

- Glutathione — master intracellular antioxidant, often depleted in MS
- Nrf2 pathway activation — upregulates cellular defence genes
- Oxidative stress is a major driver of demyelination
- Selenium and zinc — antioxidant enzyme cofactors
- Sulforaphane — potent Nrf2 activator from broccoli sprouts

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STEP 06 OF 06 — STRESS & SLEEP (PART 1)

HPA Axis & Neuroresilience

THE STRESS-SLEEP-INFLAMMATION AXIS

Poor sleep and sustained stress reactivate primed microglia, dysregulate cortisol rhythms, impair glymphatic clearance, and destabilise the BBB — a well-documented relapse pathway.

DOMAIN 01

SLEEP OPTIMISATION

Target 7-9 hours quality sleep. Address circadian disruption. Support glymphatic clearance — active during deep sleep. Screen for sleep apnoea.

DOMAIN 02

HPA REGULATION

Assess cortisol rhythm (DUTCH or salivary). Support adaptogenic herbs. Address ACE/trauma. Nervous system regulation practices.

DOMAIN 03

VAGAL TONE

Cold exposure, breathwork, humming, gargling, HRV training — activate the cholinergic anti-inflammatory reflex.

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STEP 06 OF 06 — STRESS & SLEEP (PART 2)

Nervous System Support

Practitioner-grade formulations supporting HPA regulation, sleep architecture, and neurological calm.

Alkalising minerals

Multi-form magnesium for GABAergic tone, muscle relaxation, and sleep quality. Deeply calming to the nervous system.

Adaptogens

Rhodiola, ashwagandha, and Panax ginseng blend for HPA-axis modulation and resilience to chronic stress.

Sleep Support

Passionflower, lemon balm, and glycine for sleep onset and maintenance without dependence. Supports deep-sleep architecture.

Sleep and Stress Support

L-theanine + taurine for daytime calm focus, GABA activity, and downregulation of sympathetic overdrive.

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Applied Clinical Practice

A 4-Year Clinical Journey

From Diagnosis and initial stabilisation to relapse, reassessment and restored agency

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Patient Presentation

CASE STUDY — SLIDE 1 OF 5

JESSICA - PSEUDONYM

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At initial presentation

Context
High-stress professional role

Migraine + visual disturbance prompted emergency assessment and brain MRI.

THE OPENING CHAPTER

1. Rapid transition into specialist care

Brain MRI -> neurology referral -> diagnosis of relapsing-remitting MS.

2. Acute treatment and a difficult decision

A corticosteroid course was given. Neurology recommended a DMT; Jessica initially deferred while considering her options.

3. Her immediate goal: regain a sense of control

Stress regulation, self-care and lifestyle were deliberately prioritised alongside neurological monitoring.

Source: de-identified clinical record supplied by presenter; Jessica is a pseudonym. Refs 39,40.

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BASELINE TESTS: BUILDING THE CLINICAL PICTURE

CASE STUDY — SLIDE 2 OF 5

COMPREHENSIVE STOOL PROFILE



DIGESTIVE OUTPUT

Faecal fat 75.1 mg/g

Above the report range; pancreatic elastase was >500.

MICROBIAL METABOLITES

Total SCFAs 11.6 µmol/g

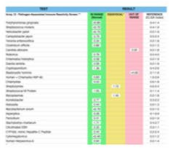
Below the report threshold; acetate was below detection.

INFLAMMATION CONTEXT

Calprotectin <17 µg/g

Within the reference range on the supplied report.

IMMUNE-REACTIVITY SCREEN



- Impaired fat handling appeared alongside low reported short-chain fatty-acid output.
- Selected immune-reactivity findings broadened the review of dietary and environmental exposures.
- The combined picture prioritised gut function, nutrient repletion and nervous-system resilience.

Source: de-identified laboratory reports supplied by presenter; values transcribed as reported. Refs 5–8,39,46.

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STRESS MOVED FROM BACKGROUND TO THE FOREGROUND

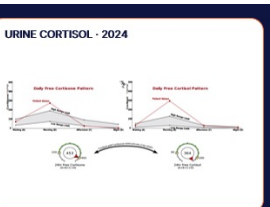
CASE STUDY — SLIDE 3 OF 5

Major family stress marked a pivotal change in the clinical course and prompted deeper metabolic and stress-physiology assessment.

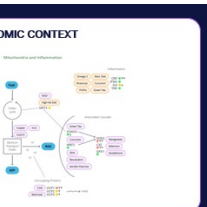
METABOLOMIC CONTEXT · 2024



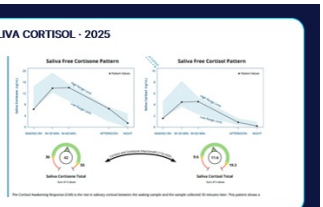
URINE CORTISOL · 2024



NUTRIGENOMIC CONTEXT



SALIVA CORTISOL · 2025



MARCH 2024

Major family stress became a defining context.

MAY–JULY 2024

Metabolic reassessment; urinary free cortisol 364 (report range 65–200).

JULY 2025

Salivary total in range; awakening rise still high at 187%.

2025 → MARCH 2026

Clinician-diagnosed relapse remained ongoing into March 2026.

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RESTORING AGENCY BECAME PART OF THE CARE PLAN

CASE STUDY — SLIDE 4 OF 5

Across the plans, emotional regulation was repeatedly translated into small, repeatable daily practices.

Emotional healing & regulation

- **Three daily 10-minute "credits"** for calming and self-directed time
- **4-4-4 breathing** and a deliberate pause before meals
- **5-4-3-2-1 grounding**, body scanning and progressive release
- **Safe-place imagery** and bilateral self-soothing for emotional overwhelm
- **Protecting "non-negotiables"** to rebuild boundaries and agency

Rest & pacing

Regular sleep and rest rhythm, morning light, gentle movement, recovery days and avoiding overexertion during fatigue.

Food foundations

Staged whole-food change, plant variety, adequate protein, hydration and food preparation adapted to digestive tolerance.

Monitored nutrients

Correction of iron and vitamin D deficiency, then individualised use of nutrient and fatty-acid classes with retesting.

Coordinated care

Generic supplements were introduced in stages, with interaction review, response monitoring and continued neurology follow-up.

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OUTCOMES: PROGRESS, RELAPSE & CLINICAL ADAPTATION

Objective gains and early MRI stability were followed by a prolonged relapse, prompting renewed investigation and adaptation.

CASE STUDY — SLIDE 5 OF 5

Iron Status

12 → 43.3

Ferritin µg/L; later value at 3 months

Vitamin D

28 → 123

nmol/L over 5 months; monitored case dose

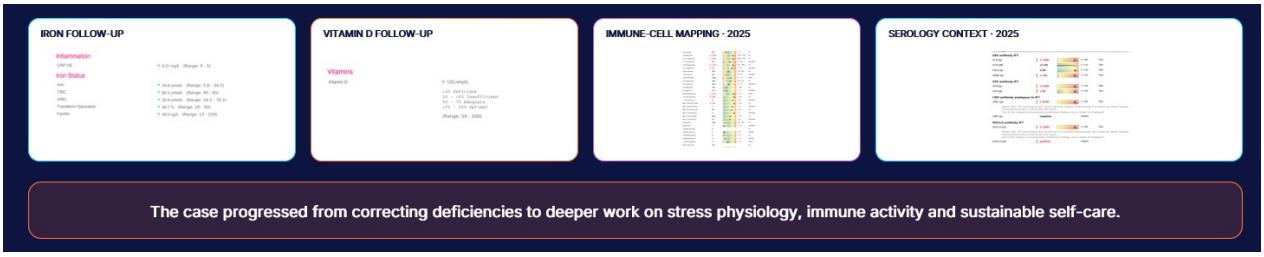
Early MRI

Stable

Six months after diagnosis

Clinician diagnosed relapse

2024 → 2026



Source: de-identified clinical and laboratory record supplied by presenter. Refs 39,40,46.

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KEY TAKEAWAYS · PART 1

Clinical Principles

Four foundational principles for the functional medicine practitioner working with MS.

01 MS is a systemic condition

Neurological expression of upstream systemic dysfunction — gut, inflammation, mitochondria, environment.

02 Resolve, do not just suppress

Add active resolution — SPMs, omega-3s, curcumin — alongside conventional care rather than only blocking pathways.

03 Individualise everything

Genetics, environment, history, and stage-of-disease all shape which pillars are prioritised and in what order.

04 Complement, don't compete

Functional medicine works alongside neurology and DMTs — supporting resilience and improving quality of life.

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KEY TAKEAWAYS · PART 2

Practitioner Action Plan

Six practical steps to translate this framework into your clinical practice from Monday morning

1. Begin with a comprehensive intake - timeline, ACE, infections, diet, environment, family history.
2. Order targeted testing - omega-3 index, hs-CRP, homocysteine, vitamin D, stool analysis.
3. Prioritise the foundation phase - gut, EFAs, vitamin D, magnesium, and sleep before layering complexity.
4. Add resolution - not just suppression - with SPMs, curcumin, and PEA when indicated.
5. Address stress, sleep, and vagal tone deliberately - they modulate every other pillar.
6. Track objective and subjective markers - retest at 3, 6, and 12 months.

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THE NEURO RESILIENCE SUMMIT