The Virome

How viral exposure interacts with our biology across the lifespan and what we can do about it

rn labs



What are viruses

- 100 times smaller than bacteria
- Lack the characteristics of a living organism on their own
- Have co- evolved with other organisms as intracellular parasites
- DNA (e.g. herpes, HPV) or RNA (e.g. HIV corona virus) surrounded by glycoproteins and lipids

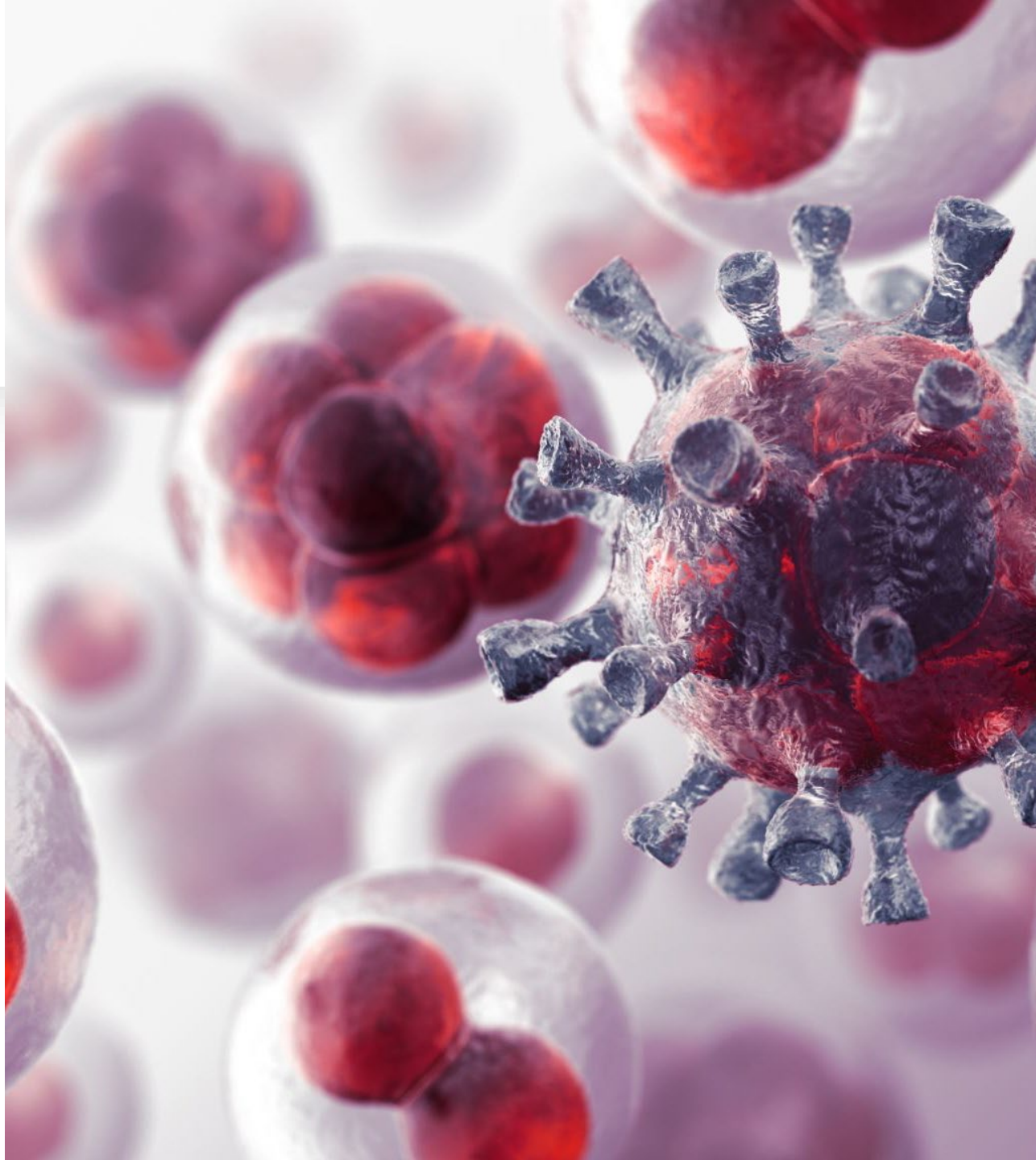


What We Will Cover

- The Prevalence of latent Viruses
- Their Relevance to health and Disease
- Effects on the Neuro Immune axis
- Nutraceutical and lifestyle options to mitigate their effects.

Chronic & Latent Viral load trends

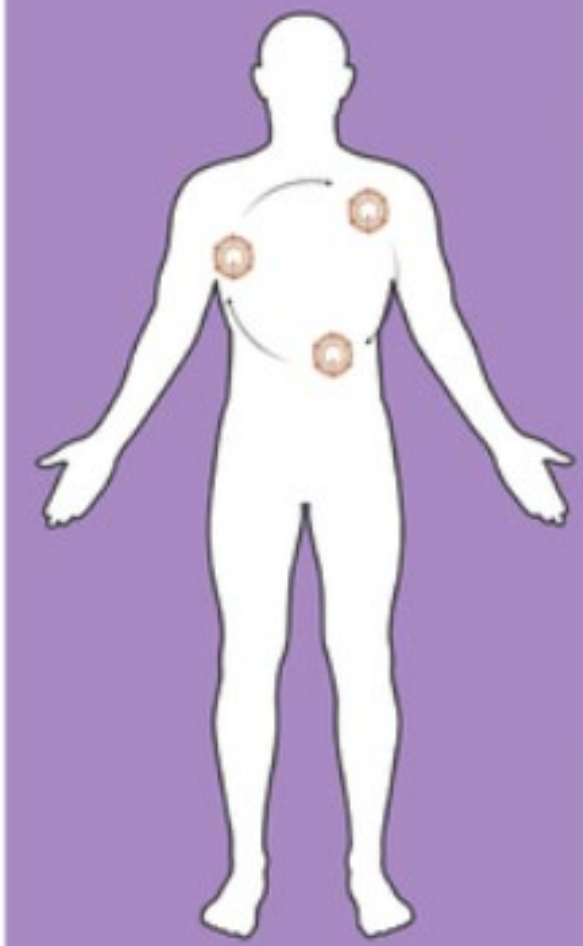
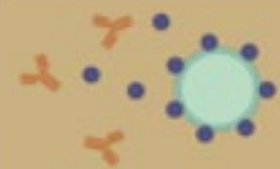
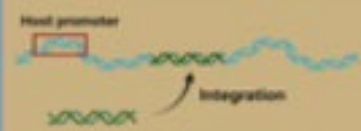
- Higher rate of latent viruses in modern humans
- Possibly due to increased identification and extended lifespan
- Modern living conditions and social interactions also play a role



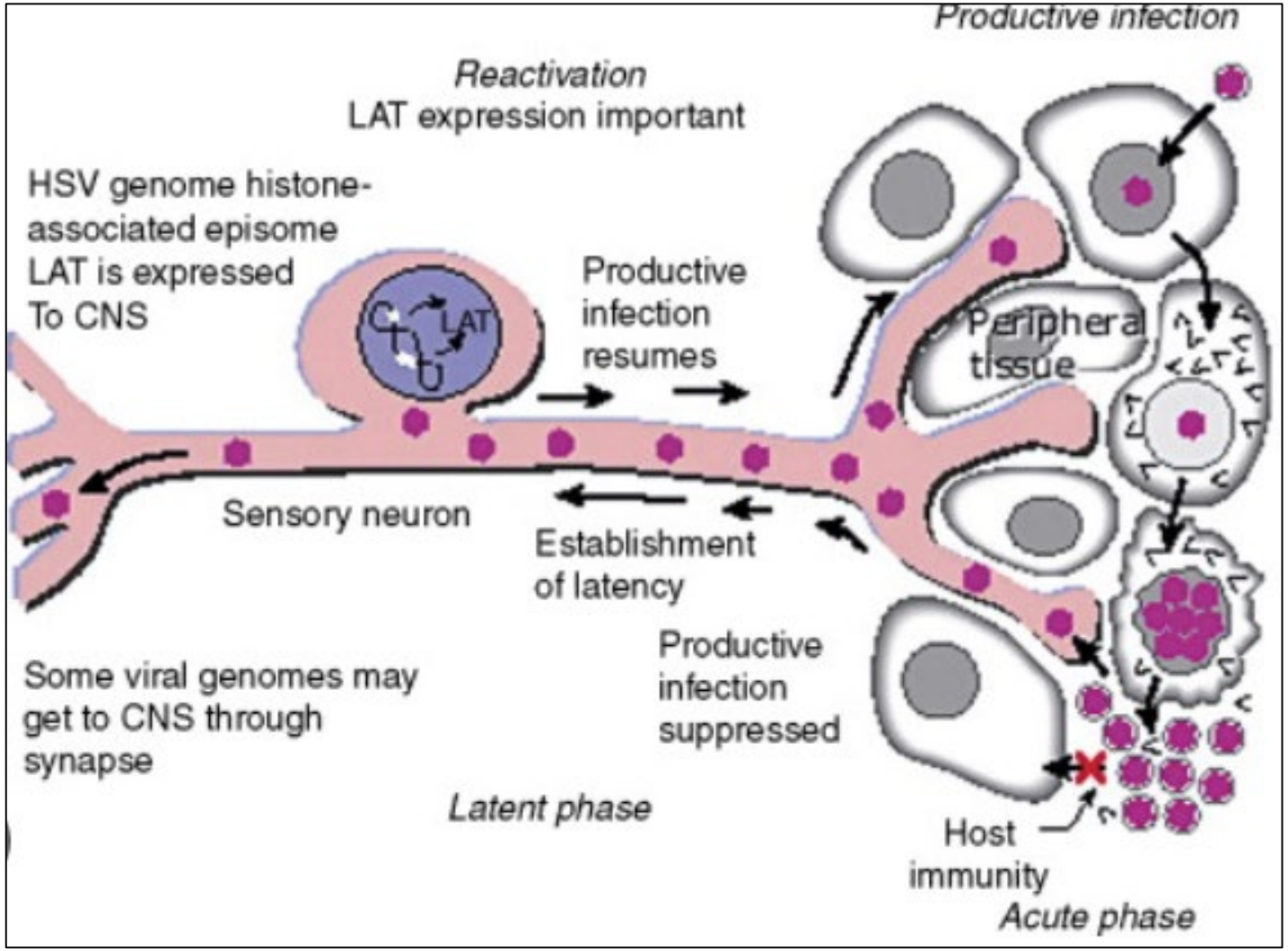
Viral load across a lifespan

- Childhood infections
- Adolesence- Glandular fever, Ross river etc.
- STDs
- Tick borne viruses- borrellia, Lyme
- Covid & other respiratory viruses (increased incidences of flu- include stats)
- Shingles

Pathogen	Pathogen Type	Primary Clinical Presentation / Latency Site	Prevalence in Australian Adults	Key Notes & Demographics
Epstein-Barr Virus (EBV)	Virus	Glandular fever / B lymphocytes	**90% – 95%**	Extremely common; most people acquire it during childhood or early adulthood via saliva (NHMRC).
Varicella-Zoster Virus (VZV)	Virus	Chickenpox (primary), Shingles (reactivation) / Sensory ganglia	**> 90%**	Most adults born prior to routine childhood vaccination carry the virus; reactivates as shingles in ~1 in 3 people over their lifetime (Australian Prescriber).
Herpes Simplex Virus 1 (HSV-1)	Virus	Oral herpes (cold sores), Genital herpes / Sensory ganglia	**70% – 80%**	Highly prevalent; often acquired in early childhood through non-sexual skin-to-skin contact or kissing (Queensland Health).
Cytomegalovirus (CMV)	Virus	Usually asymptomatic; mononucleosis-like illness / Myeloid progenitor cells	**50% – 57%**	National serosurveys show seropositivity increases with age (~50% in early 20s to ~79% by age 50–59) (NIH / PMC).
***Toxoplasma gondii* (Toxoplasmosis)**	Parasite (*protozoan*)	Usually asymptomatic; ocular disease, brain cysts / Neural and muscle tissues	**30% – 40%**	Acquired through undercooked meat, unwashed produce, or contact with cat faeces. Persists as latent tissue cysts; can reactivate in immunocompromised individuals (WEHI).
Herpes Simplex Virus 2 (HSV-2)	Virus	Genital herpes / Sacral sensory ganglia	**12% – 15%**	Predominantly transmitted via sexual contact; many carriers remain asymptomatic (Queensland Health).
Latent Tuberculosis (LTBI)	Bacterium	Asymptomatic lung infection / Macrophages	**~5%**	Lower in Australian-born residents (~0.4%) and significantly higher among overseas-born residents (~17%) (RACGP).

Viral infection cycle	Steps in latency induction	Mechanism of action
 Infected individual	**1. Direct infection of cells** Establishment of host entry	 Entry initiated by viral specific surface receptor interaction
	2. Silencing of initially productive infection Insufficient levels of transcriptional activators	 Reduction of transcription and initiation dormancy
	3. Epigenetic regulation DNA methylation, repressive histone modifications	 Modification Stabilize viral latency
	4. Transcriptional control Transcription factors like HFH-3, v-Myb, E2F, TCF11, Maf proteins, C-Rel, AP-1, regulate viral gene expression	 Disrupt autoregulatory circuit and favor latency establishment
	5. Immune evasion Immune evasion proteins m04, m06, and m152 enhance viral latency	 Suppressing immune responses
	6. Promoter occlusion Viral gene integrate upstream host gene promote occluding transcriptional factors	 Occludes transcriptional factors avoiding transcription

Effect of latent viruses on the Neuro-immune axis



The peripheral-to-central inflammatory axis

1

Peripheral reactivation

Latent pathogen reactivates or is chronically sensed by the immune system

2

Cytokine release

Innate immune cells release IL-6, TNF-α, IL-1β, IFN-γ

3

BBB signalling

Cytokines cross/signal across the blood-brain barrier via active transport and vagal afferents

4

Microglial priming

Resident brain immune cells shift to an activated, pro-inflammatory phenotype

5

Neurocircuit & neurotransmitter change

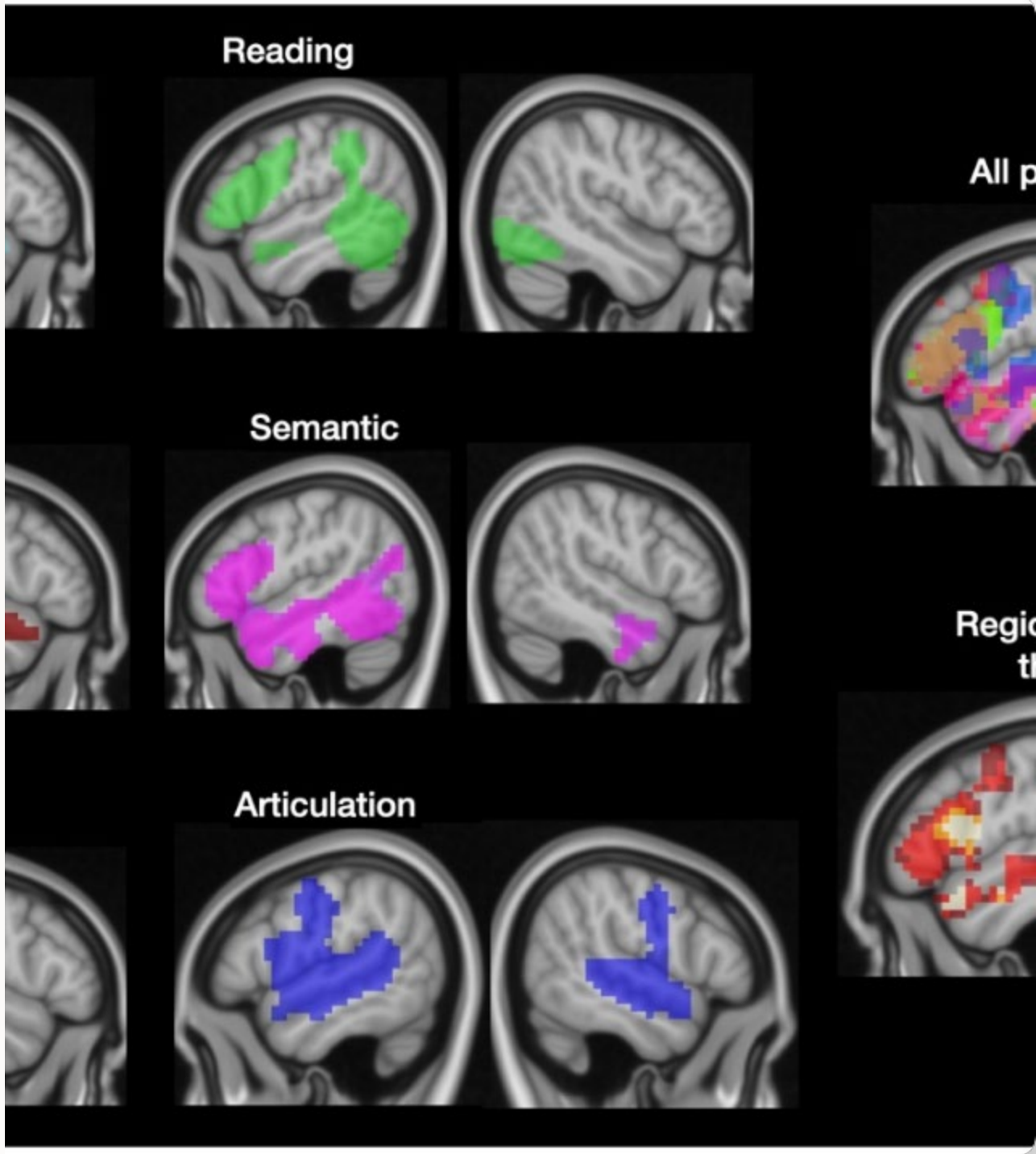
Altered monoamine synthesis, glutamate excitotoxicity, reduced neurogenesis

Miller AH & Raison CL, Nat Rev Immunol 2016; Dantzer et al., Nat Rev Neurosci 2008.

Microglial Activation

Priming & Inflammatory Stress

Chronically primed microglia release neurotoxic pro-inflammatory cytokines (IL-1β, TNF-α, IL-6), depleting ATP production, enhancing glutamate induced excitotoxicity and ultimately causing degeneration of neurons



Ref: PMC (2023). Microglia, Astrocytes and Oligodendrocytes in Neuroinflammatory Crosstalk.

Calm in the Storm: Microglia Shift

M1: Neurotoxic

Chronic activation of microglia leads to the release of TNF-α and IL-6, inhibiting a return to homeostasis.



M2: Neuroprotective

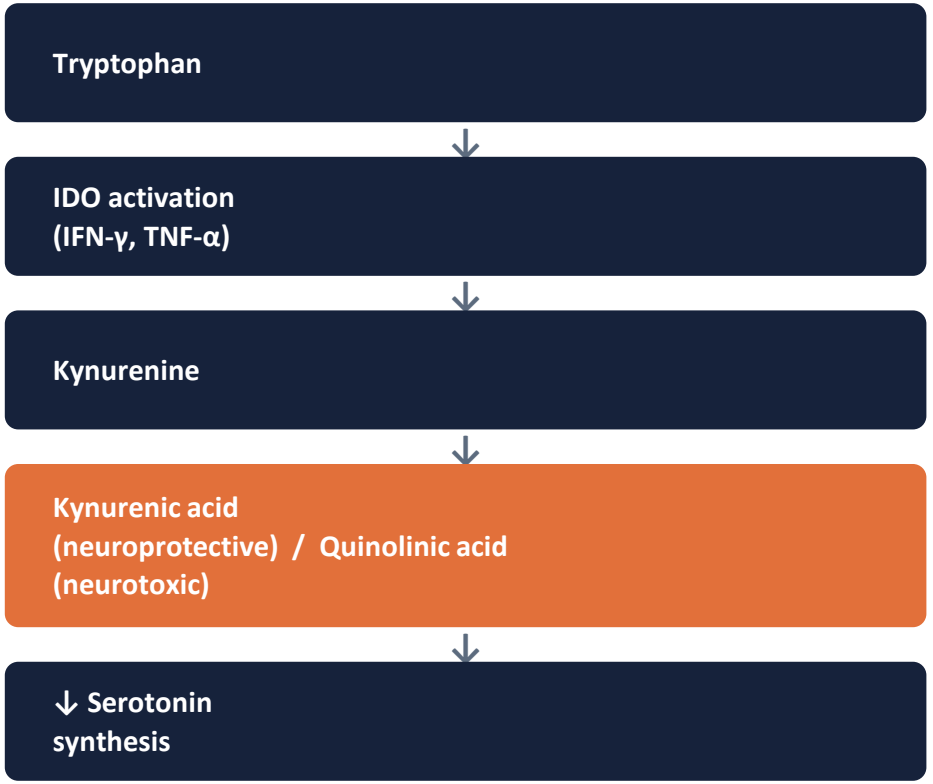
Butyrate, PEA & Omega 3 help to shift microglia to the M2 state, where they focus on debris clearance and neurotrophic support.

The kynurenine pathway

Pro-inflammatory cytokines (especially IFN-γ and TNF-α) induce the enzyme indoleamine 2,3-dioxygenase (IDO), which diverts tryptophan away from serotonin synthesis and toward the kynurenine pathway.

This produces neuroactive metabolites — some neuroprotective (kynurenic acid), others neurotoxic (quinolinic acid, an NMDA receptor agonist implicated in excitotoxicity).

This exact mechanism recurs across the pathogens— it is invoked in HSV/EBV-associated mood change, CMV-driven inflammaging, and is the leading proposed mechanism for Toxoplasma gondii's psychiatric associations.



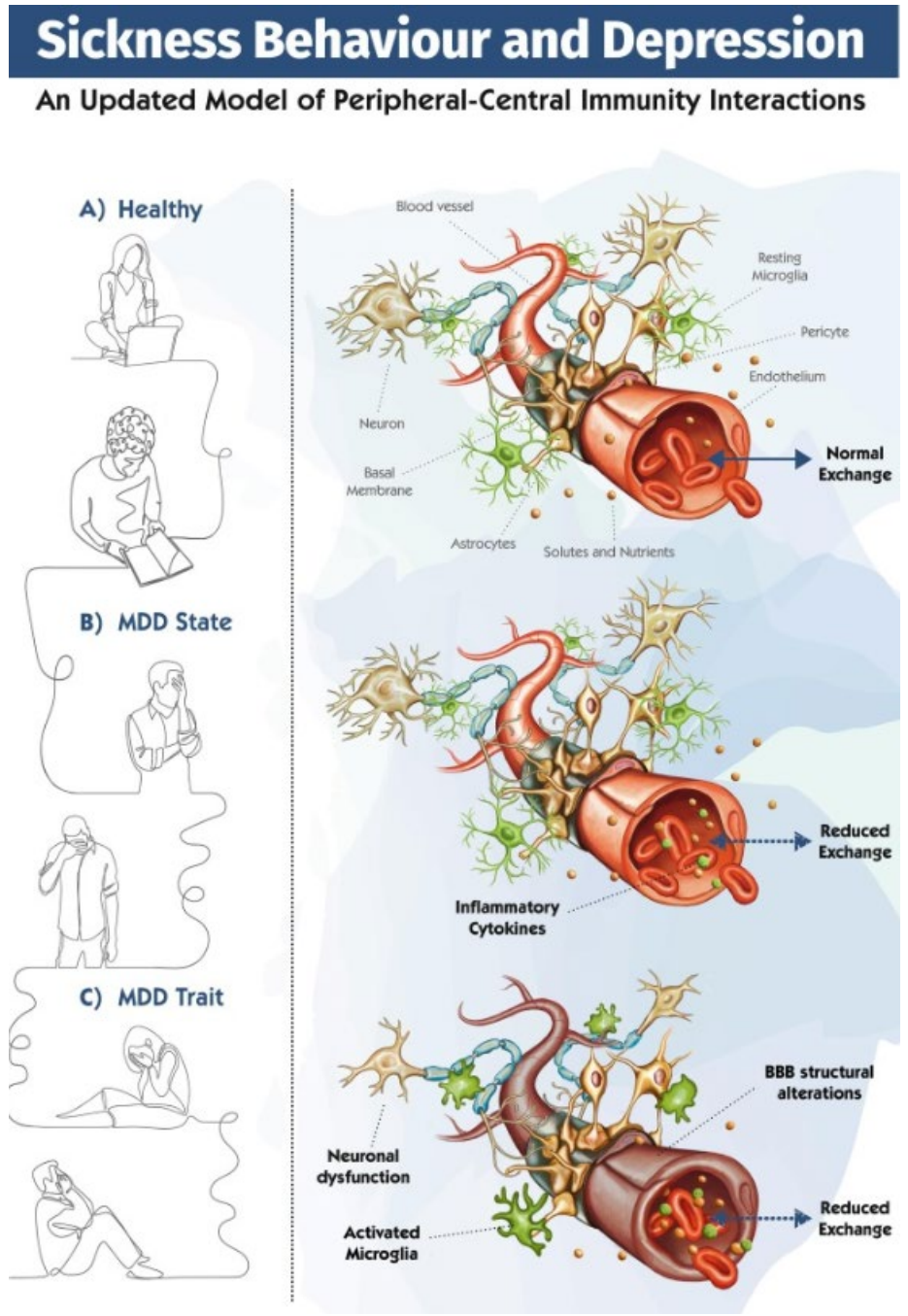
Notarangelo, F. M., et al. "Evaluation of kynurenine pathway metabolism in Toxoplasma gondii-infected mice: implications for schizophrenia." *Schizophrenia research* 152.1 (2014): 261-267.

Sickness behaviour and depression: An updated model of peripheral-central immunity interactions

Federico E. Turkheimer [a], Mattia Veronese [a b], Valeria Mondelli [c], Diana Cash [a], Carmine M. Pariante [c] Show more

Highlights

- Circulating immune mediators have potent influences on brain functions.
- Anti-inflammatory therapies are now used to improve treatment efficacy, particularly in subjects that do not respond to standard medication.
- These therapies require biomarkers to tailor these new therapies but also validated mechanisms of action.
- In this proposal paper we put forward a modified model of periphery-brain interactions that goes beyond the currently established view of microglia cells as the drivers of depression.
- We suggest that brain barriers may have a primary role in the pathophysiology of the disease and in treatment resistance.



Triple Latency as a Driver of Chronic Inflammation: An Integrative View of HSV, EBV, and CMV Persistence in Immunocompetent Hosts

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Clin. Pract. **2026**, *16*(4), 64; https://doi.org/10.3390/clinpract16040064

Submission received: 15 January 2026 / Revised: 12 March 2026 / Accepted: 22 March 2026 / Published: 24 March 2026

Mechanistic studies suggest cross-compartment immune priming, where CMV-driven T-cell exhaustion facilitates EBV reactivation, and viral cytokine signalling enhances HSV-associated neuroinflammation. Conclusions: HSV, EBV, and CMV triple latency may represent an underrecognized contributor to chronic inflammation in immunocompetent hosts.

The stress/immune link

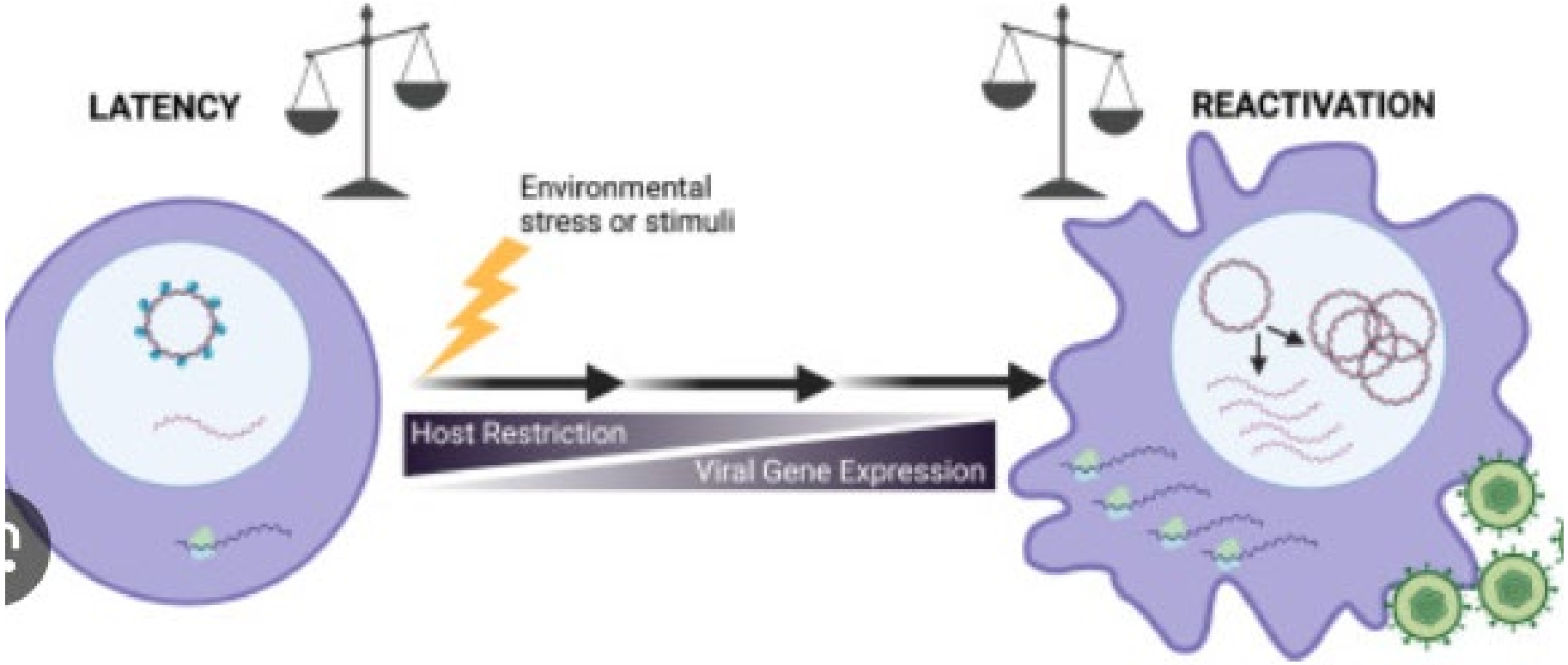
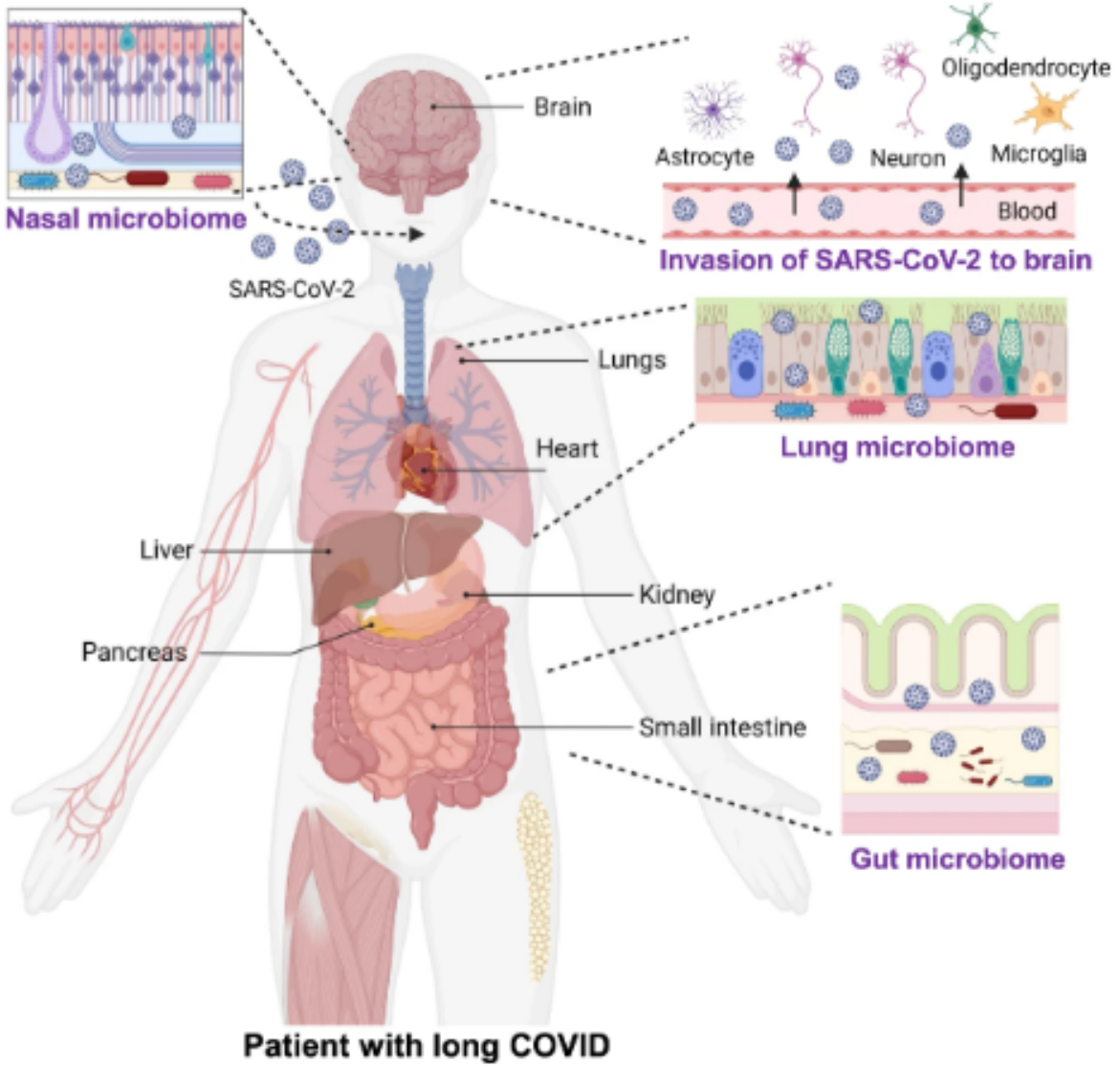


Fig. 1: Long-term sequelae in COVID-19 survivors.

From: Detrimental effects of COVID-19 in the brain and therapeutic options for long COVID: The role of Epstein–Barr virus and the gut–brain axis

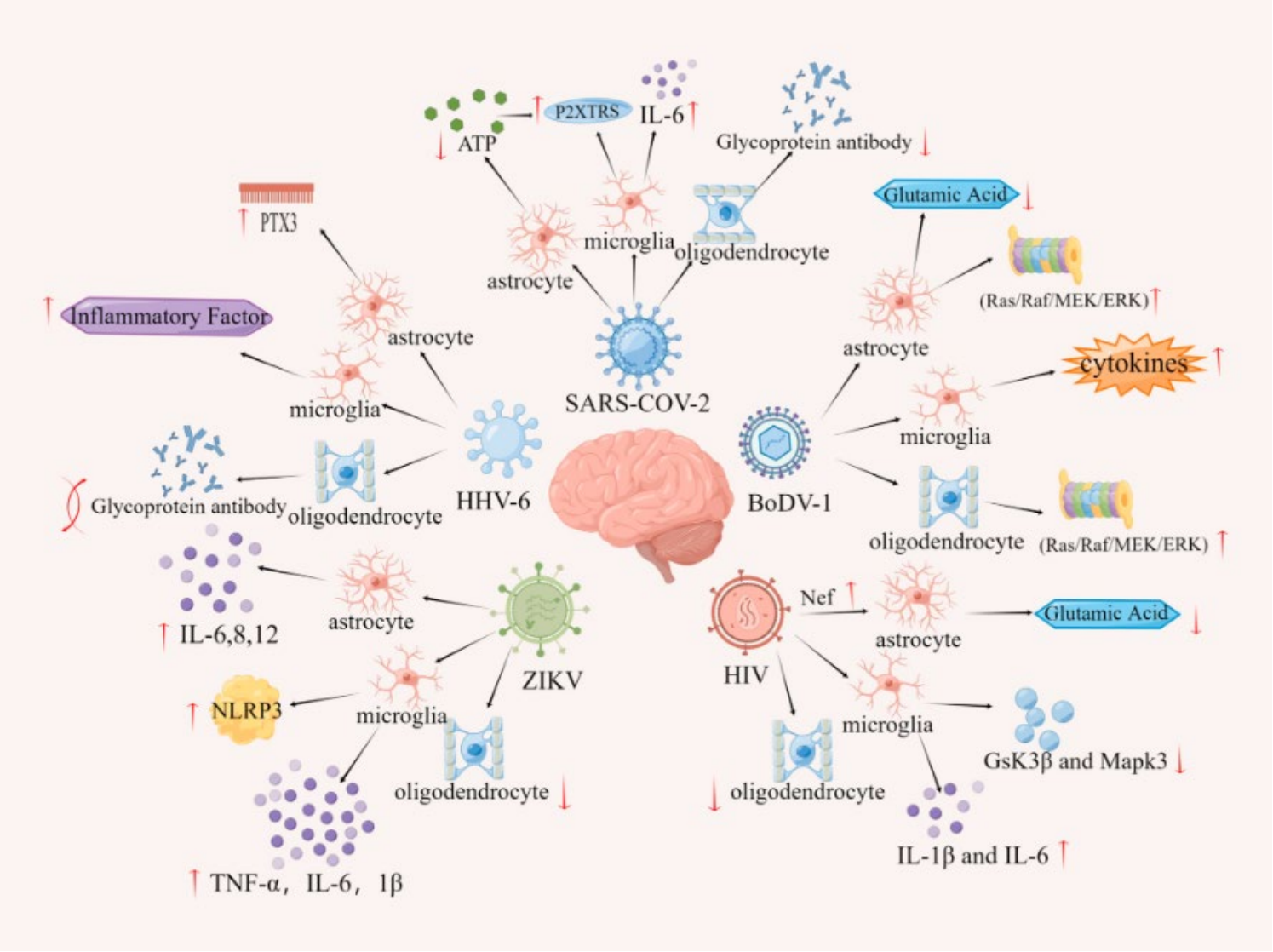


- Neurological symptoms (e.g., difficulty thinking or concentrating, headaches, dizziness, cognitive impairment)
- Psychiatric symptoms (e.g., depression, anxiety, sleep disturbance)

- Respiratory and heart symptoms (e.g., difficulty breathing or shortness of breath, cough, chest pain, fast-beating or pounding heart)

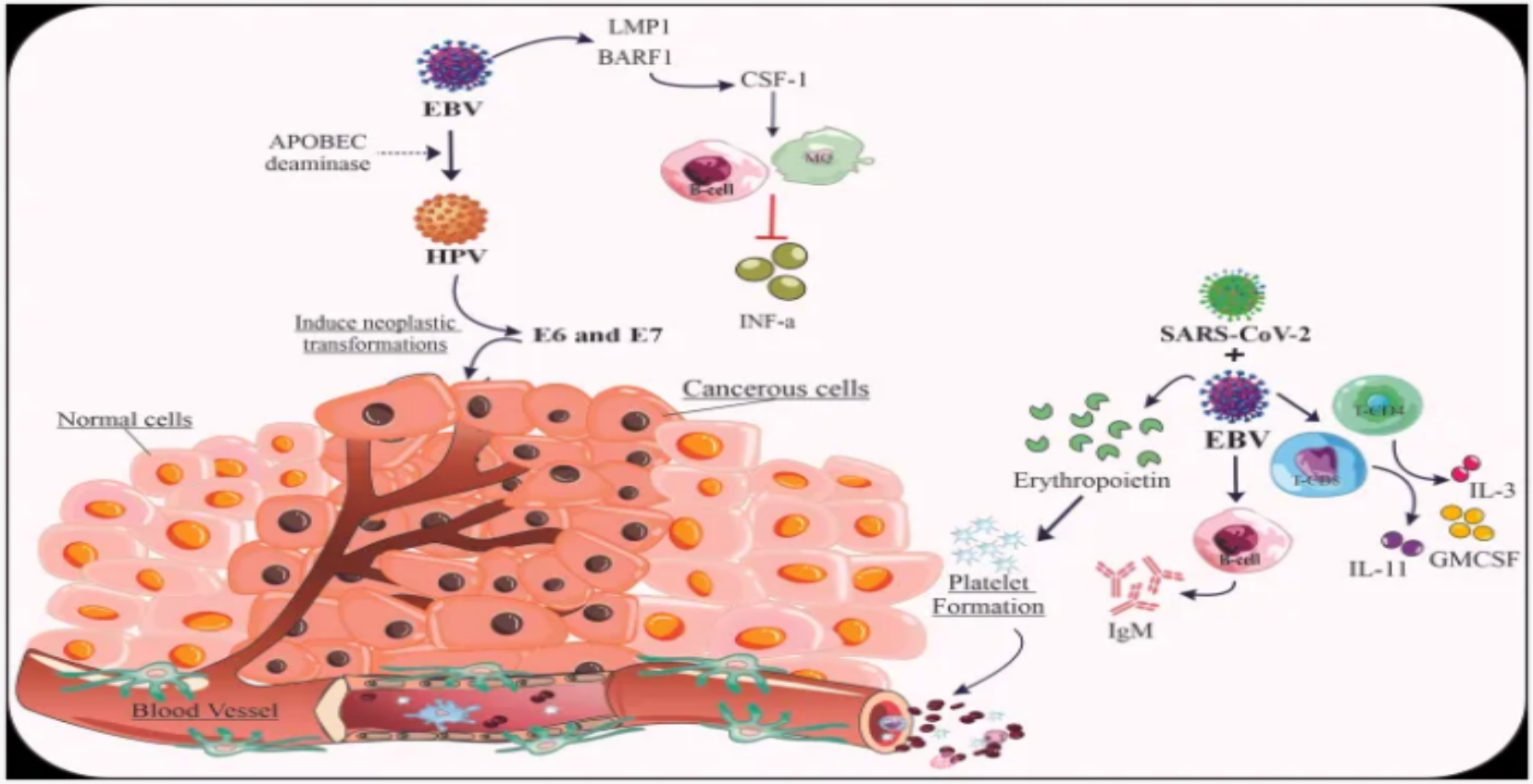
- Gastrointestinal symptoms (e.g., nausea, abdominal pain, loss of appetite, heartburn, constipation)

Effects of Latent viruses on Nerve Cells



Yu, Xinxin, et al. "Exploring new mechanism of depression from the effects of virus on nerve cells." *Cells* 12.13 (2023): 1767.

EBV+HPV+SARS-CoV2



The mechanisms related to the co-infection of EBV virus with HPV and SARS-CoV-2. The coinfection of HPV and SARS-CoV-2 with EBV can promote the severity of malignancy by inducing the expression of genes and cytokines related to specific pathways.

Viruses & Aging

Chronic and Latent Viral Infections and Leukocyte Telomere Length across the Lifespan of Female and Male Individuals Living with or without HIV

Nancy Yi Yang [1,2], Anthony Y Y Hsieh [1,2], Zhuo Chen [3], Amber R Campbell [1,4,5], Izabella Gadawska [1], Fatima Kakkar [6], Laura Sauve [4,5,7], Ari Bitnun [8], Jason Brophy [9], Melanie C M Murray [4,5,10], Neora Pick [4,5,10], Mel Krajden [1,11], Hélène C F Côté [1,2,4,*]; CIHR Team on Cellular Aging and HIV Comorbidities in Women and Children (CARMA)

Editors: Yasuko Tsunetsugu-Yokota, Mie Kobayashi-Ishihara

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PMCID: PMC11125719 PMID: 38793637

Depression and anxiety

- A 2014 meta-analysis of 28 case-control studies across 16 infectious agents found statistically significant associations between depression and HSV-1, varicella-zoster virus, and EBV specifically.
- A U.S. adult cohort study found HSV-1/2 and CMV exposure associated with depression, though cross-sectional design limits causal inference.
- Older serological studies (Amsterdam & Hernz, 1993) reported elevated HSV antibody titres in depressed patients relative to controls — an early and since-replicated observational signal.
- **Effect sizes for HSV and depression are generally small; HSV is best understood as one contributor within a broader "pathogen burden" framework rather than a dominant single cause.**

Coughlin, Steven S. "Anxiety and depression: linkages with viral diseases." *Public health reviews* 34.2 (2012): 7.

Biology and CNS latency

- T. gondii is an obligate intracellular protozoan parasite; definitive hosts are felids, but humans are commonly infected via undercooked meat, contaminated soil/water, or cat faeces exposure.
- After acute infection, the parasite converts to slow-growing bradyzoite cysts that persist lifelong, preferentially in neurons and skeletal/cardiac muscle — true CNS latency, comparable in principle to herpesviral latency.
- Global seroprevalence is estimated around 30%, with wide geographic variation (much higher in parts of Latin America and continental Europe than in North America or East Asia).
- Cyst reactivation and the ongoing host immune response to encysted parasites (rather than acute infection) is the presumed driver of any chronic neuropsychiatric effect.

Mechanism and psychiatric evidence

- T. gondii triggers a sustained Th1 response (IFN-γ, TNF-α), activating the kynurenine pathway and diverting tryptophan away from serotonin synthesis toward neurotoxic quinolinic acid — directly linking this parasite to the shared mechanism introduced earlier.
- T. gondii also directly modulates dopaminergic signalling in infected neurons, giving it a second, more parasite-specific mechanistic route distinct from pure cytokine signalling.
- Meta-analytic evidence for schizophrenia/psychosis is the most consistent (small-to-moderate association); depression and anxiety associations are more variable across studies, with some studies finding no association after full adjustment.
- **A 2026 path-analysis study found toxoplasmosis was directly associated with poorer physical health, which in turn predicted perceived stress, which in turn predicted anxiety — a stress-mediated indirect pathway, distinct from a direct neuroinflammatory effect, and notably absent in a comparison group with a different chronic infection (borreliosis).**

Fabiani S et al.; Xiao J & Yolken RH reviews; Frontiers in Psychiatry 2026 (fpsyt.2026.1764597).

Immunosenescence and inflammaging

- CMV establishes latency in myeloid progenitor cells and, unlike most latent viruses, provokes a massive, permanent expansion of CMV-specific memory T-cells over a lifetime (“memory inflation”) — in older seropositive adults, a large proportion of circulating CD8+ T-cells can be CMV-specific.
- This drives immunosenescence: loss of T-cell receptor diversity, accumulation of exhausted/senescent effector-memory T-cells, and loss of the co-stimulatory marker CD28 — a phenotype strongly associated with chronic, low-grade systemic inflammation (“inflammaging”).
- CMV seroprevalence rises with age (roughly 50% at age 40 to higher thereafter in most cohorts) and CMV is considered one of the principal drivers of the “Immune Risk Phenotype” linked to earlier mortality in older adults.
- **CMV's psychiatric relevance is largely mediated through systemic immune remodelling rather than neurotropism.**

Depression and cognitive evidence

- Cross-sectional cohort data have linked CMV seropositivity to depression, often in combination with HSV exposure, in U.S. adult samples.
- A community-dwelling older-adult study found cumulative “lifetime pathogen burden” (including CMV) correlated with inflammatory markers and depressive symptoms — supporting a cumulative rather than single-pathogen model.
- Because CMV's effect is exerted substantially through age-related immune remodelling, its depression association is expected to be more prominent in older adults than in younger cohorts — a useful clinical heuristic when interpreting individual patient data.
- **As with HSV, CMV likely acts as one contributor to cumulative inflammatory burden rather than a standalone diagnostic marker.**

Preprints.org 2023.11.1218 (PMC10815036); Bajaj et al., PMC5454039.

Ng TP et al., Brain Behav Immun 2022; Gale SD et al. 2018.

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The persistence and neuroinflammation hypotheses

- SARS-CoV-2 does not establish classical latency, but viral RNA and antigen have been detected in tissue reservoirs (gut, other organs) months after acute infection in some patients — a proposed “viral persistence” mechanism for long COVID.
- Long COVID affects an estimated ~36% of confirmed COVID-19 cases globally by pooled prevalence estimates, with anxiety reported in up to ~48% and depression in up to ~37% of affected patients in systematic reviews
- Proposed mechanisms include direct CNS viral invasion, blood-brain-barrier disruption, gut-brain axis dysregulation, and a vascular/endothelial dysfunction and hypoperfusion pathway, in addition to classical cytokine-driven neuroinflammation.
- Postmortem studies of severe acute COVID-19 have shown clear microglial activation and astrogliosis, and PET studies have shown microglial activation persisting up to two years post-infection in some symptomatic patients.

2026 update: a more nuanced picture

- **A 2026 TSPO-PET study (Journal of Neurology) directly compared long COVID patients to multiple sclerosis patients and healthy controls, using the same neuroinflammation-sensitive imaging technique.**
- Key finding: long COVID patients showed significantly lower white-matter neuroinflammatory signal than MS patients, and no statistically significant difference in whole-brain neuroinflammation or neurodegeneration markers versus healthy controls.
- However, within the long COVID group, higher depression, anxiety, and lower quality-of-life scores correlated strongly with elevated tracer signal specifically in the hippocampus, amygdala, and thalamus — limbic structures involved in mood and emotion regulation.

Pharmacy Times 2026 (prevalence review); Frontiers in Neurology 2024 (fneur.2024.1465787); AuntMinnie 2025 (2-year TSPO-PET data).
Tuomaala J, Saraste M, Smith E et al., J Neurol 2026 (10.1007/s00415-026-13842-w); cf. Braga J et al. pilot PET study, 2023 (Alzforum).

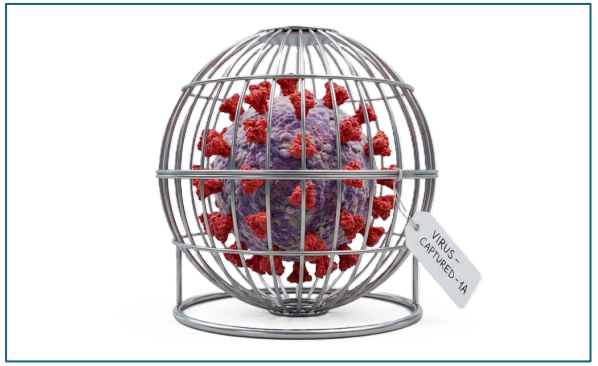
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St John' Wort: Pre-clinical evidence for anti viral activity, this is also a traditional use. Strong clinic evidence for Mild to moderate depression.

Astragalus: Exhibits adaptogenic, anti viral and immune modulating properties. Clinical evidence in relation to latent viruses suggests

Qing Hao (Sweet wormwood): Artemisia species have long been recognized and used as a source of powerful antiviral compounds (e.g malaria). Invitro and animal studies also show effects against certain latent viruses.

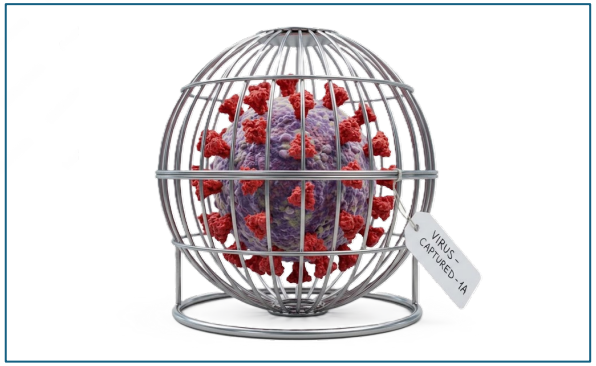
Mushroom Extracts: Extracts from various medicinal mushrooms have demonstrated potent anti viral and immune modulating actions. Clinical evidence show benefit in reducing the severity and reduction of respiratory viruses and in eliminating or inhibiting some latent viruses.

Thank You

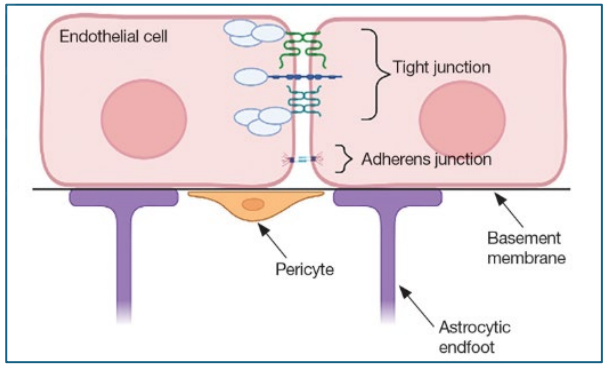
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LEVERS WE CAN PULL



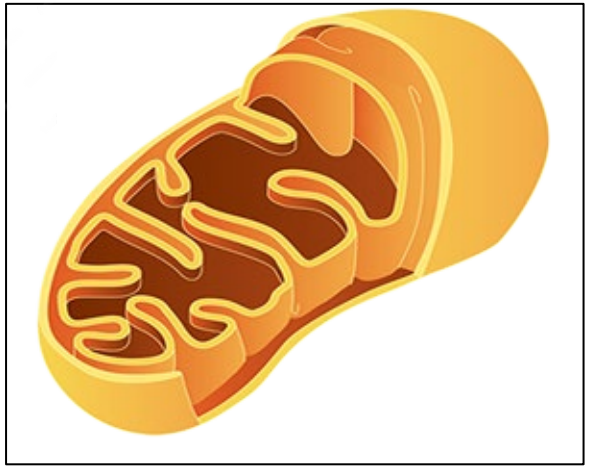
Reduce reactivation & support Immunity



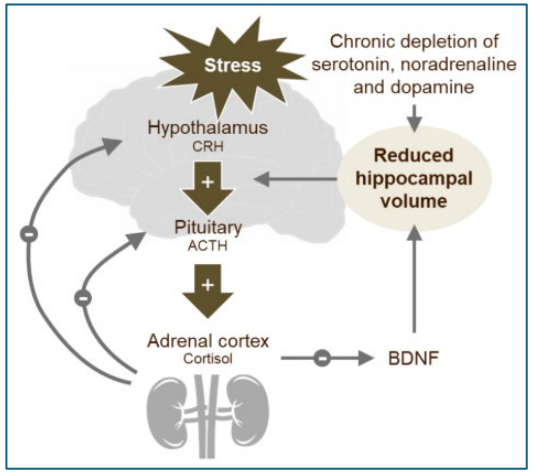
Support the integrity of Barriers



Reduce chronic Inflammation systemically & in the CNS



Support Mitochondrial Structure & Function



Regulate HPA axis & Support Parasympathetic Tone

https://neurotorium.org/image

AHCC

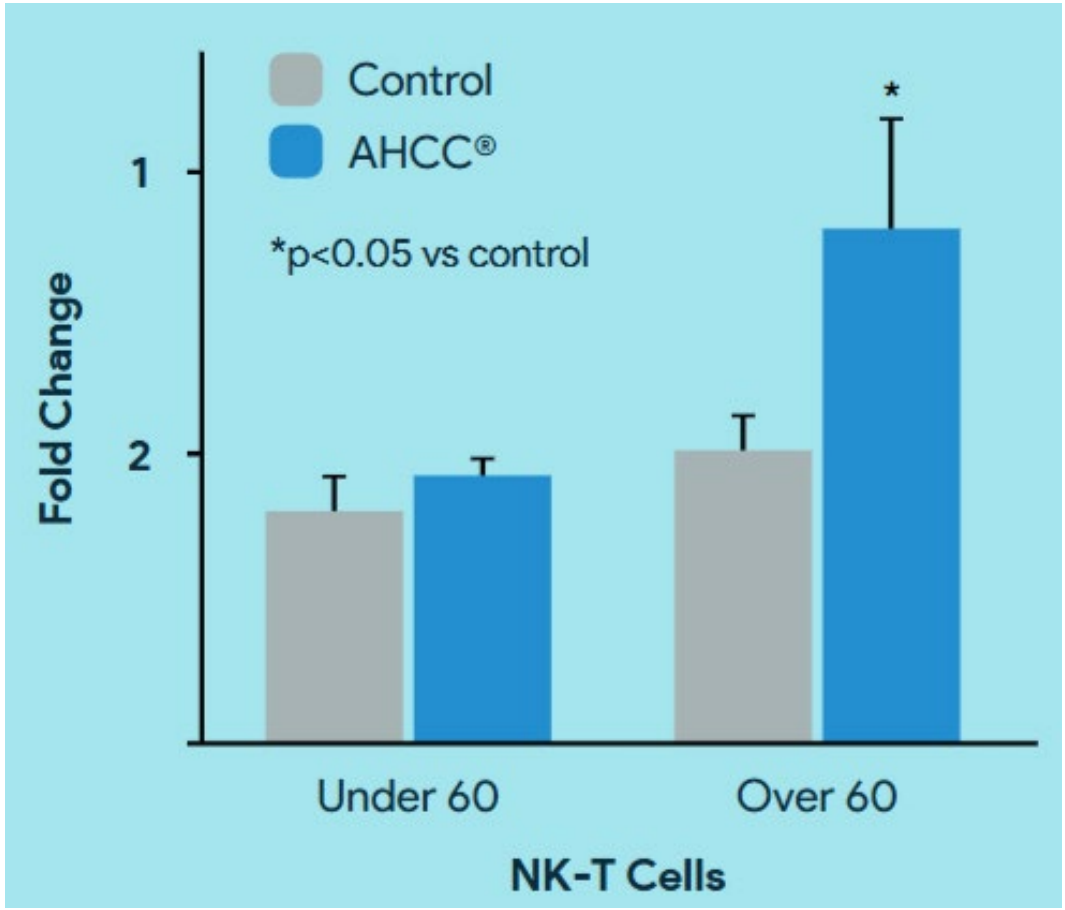
IFN-B suppression

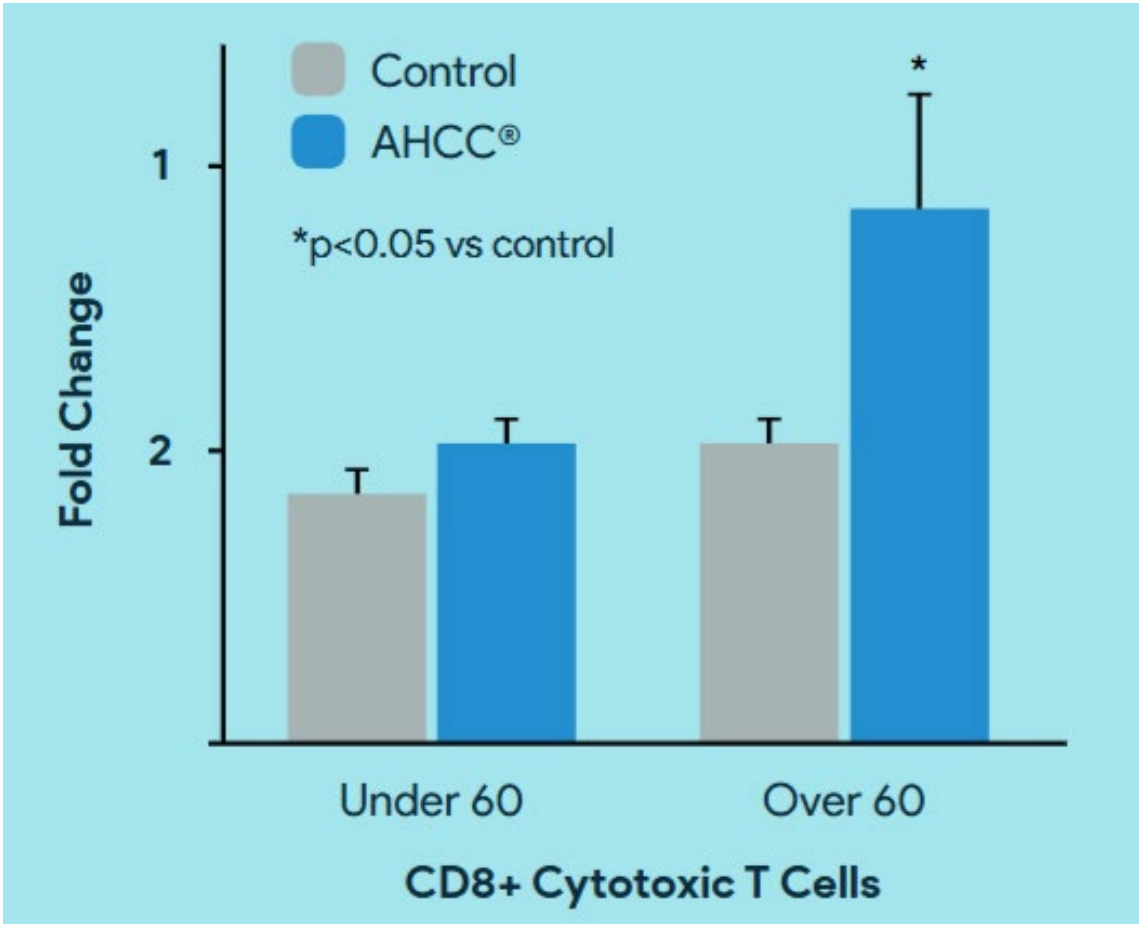
Enhancement of T cell, NK cell and dendritic cell function

Can facilitate ‘durable clearance’ of persistent viral infection

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AHCC Research





AHCC® Supplementation to Support Immune Function to Clear Persistent Human Papillomavirus Infections

Judith A. Smith[1,2], Anjali A. Gaikwad[1], Lata Mathew[1], Barbara Rech[3], Jonathan P. Faro[4], Joseph A. Lucci III[1,2], Yu Bai[5], Randall J. Olsen[6] and Teresa T. Byrd[1]*

[1] Department of Obstetrics, Gynecology and Reproductive Sciences, UT Health McGovern Medical School, Houston, TX, United States, [2] Department of Pharmacy, UT Heath-Memorial Hermann Cancer Center, Houston, TX, United States, [3] UT Physicians Women's Center, Houston, TX, United States, [4] Specialists in Obstetrics & Gynecology, Houston, TX, United States, [5] Department of Pathology, UT Health McGovern Medical School, Houston, TX, United States, [6] Department of Molecular Pathology, Houston Methodist Research Institute, Houston, TX, United States

Conclusion: Results from this phase II study demonstrated that AHCC 3 g once daily was effective to support the host immune system to eliminate persistent HPV infections and was well tolerated with no significant adverse side effects reported. The duration of AHCC supplementation required beyond the first negative result needs more evaluation to optimize success for durable outcomes. The suppression of the IFN-β level to less than 20 pg/ml correlated with clearance of HPV infections and merits further evaluation as a clinical tool for monitoring patients with HPV infections.

Nutritional support

- Lysine
- Vitamin C
- Vitamin D
- Zinc

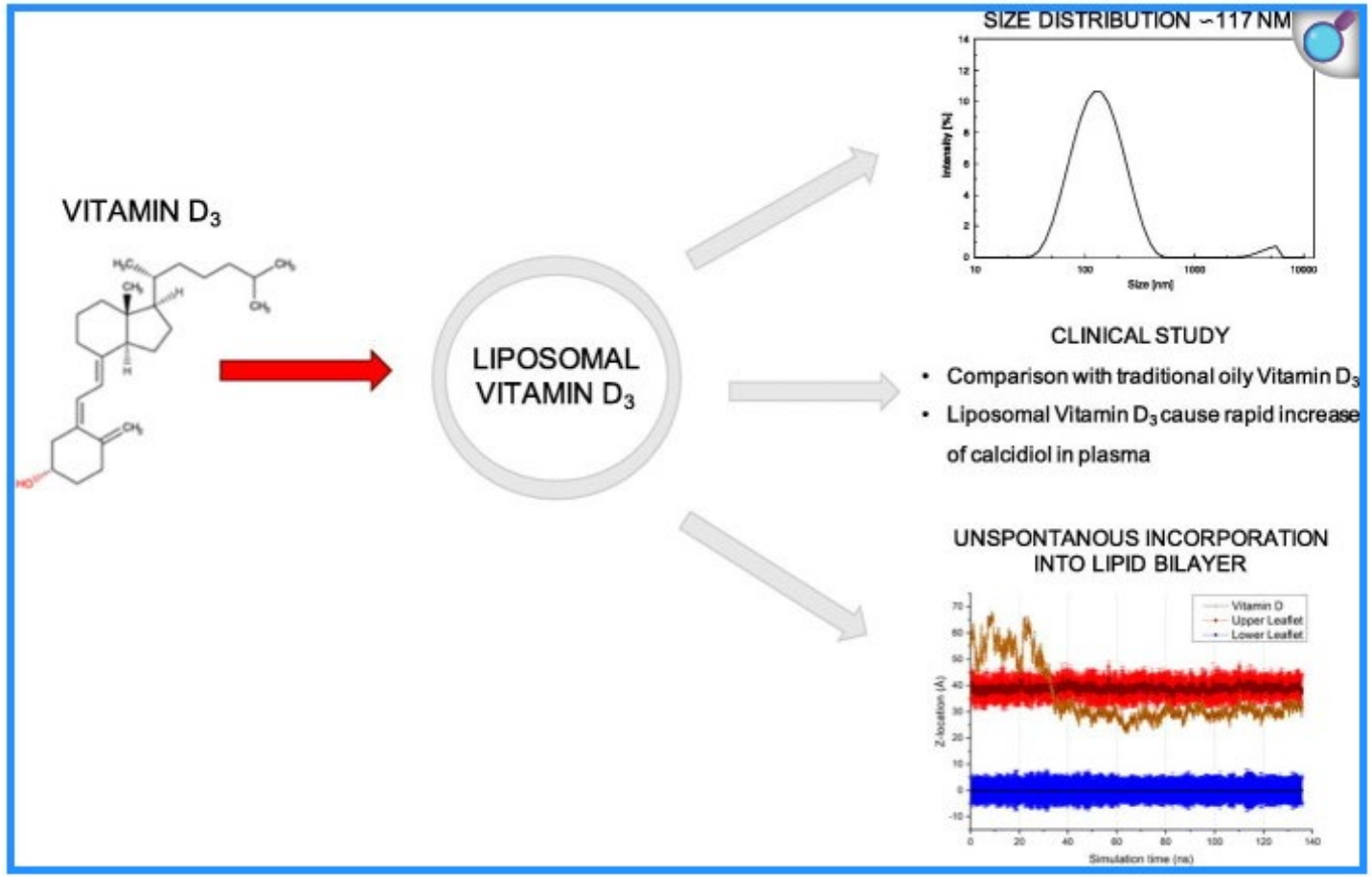


Liposomal D3

Absorption into plasma at around double the order of magnitude of standard oil based D3

Effects were most pronounced in deficient patients

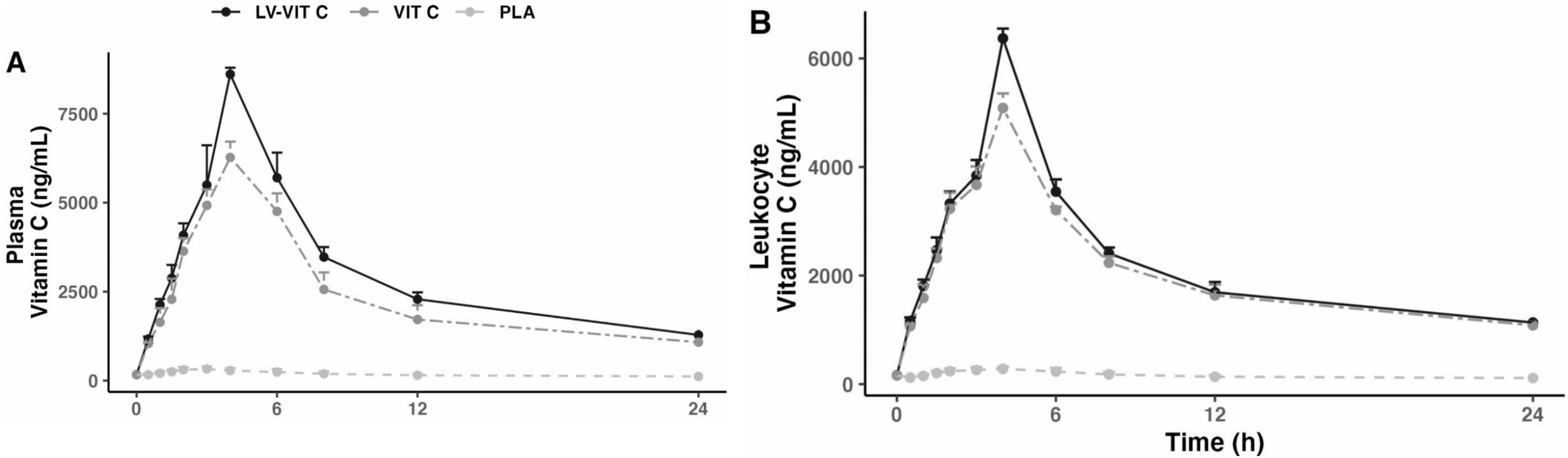
Liposomal formulations also seem to enhance incorporation into the lipid bi layer of cells, which supports activation of the VDR receptor



Dałek, P., Drabik, D., Wołczańska, H., Foryś, A., Jagas, M., Jędruchniewicz, N., ... & Langner, M. (2022). Bioavailability by design—Vitamin D3 liposomal delivery vehicles. *Nanomedicine*, *43*, 102552.

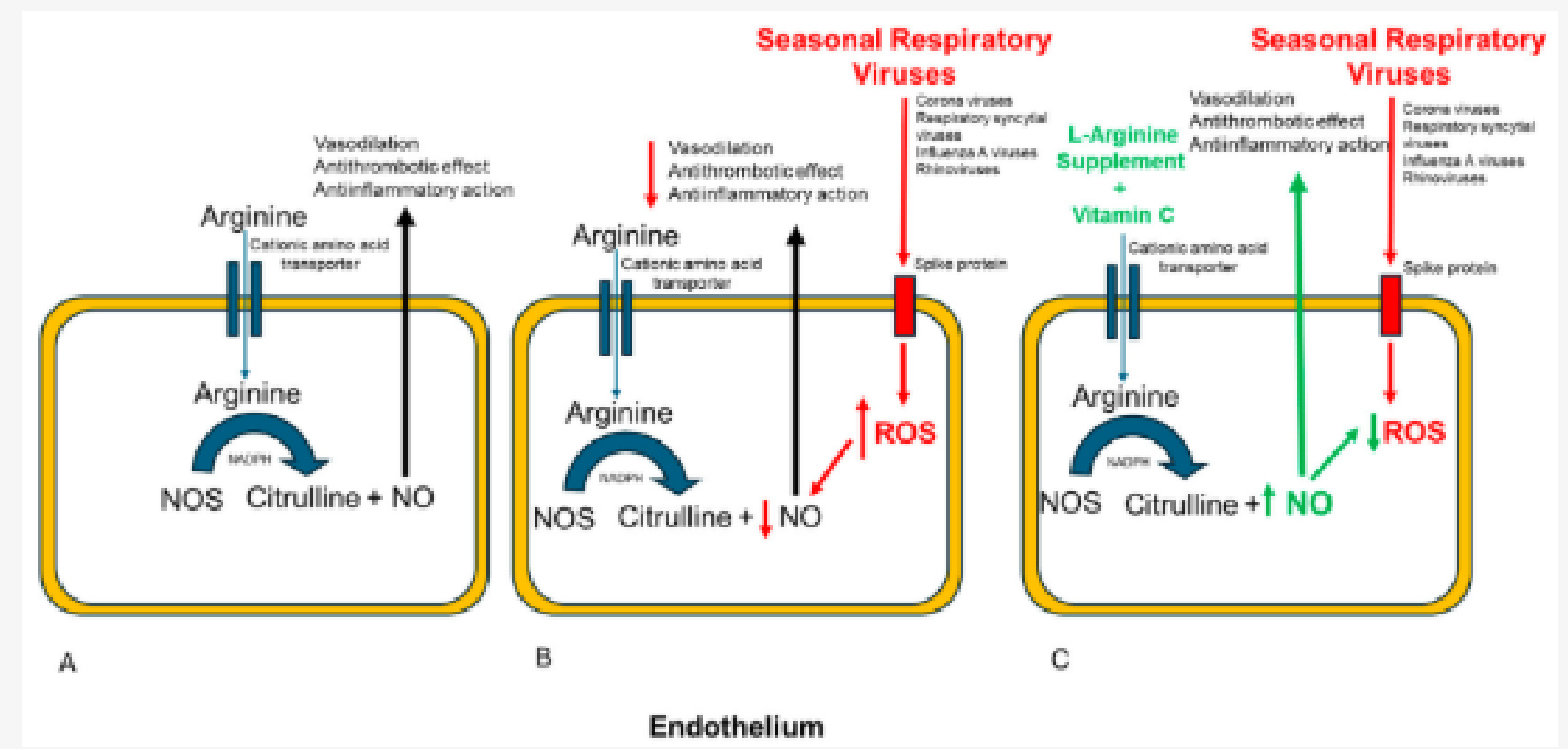
Liposomal Vitamin C

From: Liposomal delivery enhances absorption of vitamin C into plasma and leukocytes: a double-blind, placebo-controlled, randomized trial



Purpura, M., Jäger, R., Godavarthi, A., Bhaskarachar, D., & Tinsley, G. M. (2024). Liposomal delivery enhances absorption of vitamin C into plasma and leukocytes: a double-blind, placebo-controlled, randomized trial. *European journal of nutrition*, *63*(8), 3037-3046.

L-arginine & Vitamin C?



Lysine

- Lysine competes with arginine for uptake into virus

- Arginine is utilized by many viruses (especially Herpes viruses) to create the viral capsid. Deprivation of arginine has been shown to prevent viral replication.
- The severity and frequency of HSV flare ups is related to the amount of arginine in plasma
- The combination of lysine (1-3grams daily) and a low arginine diet has been shown to improve QOL for patients. Longer term studies (8 years) further support these results

Mailoo, V. J., & Rampes, S. (2017). Lysine for herpes simplex prophylaxis: a review of the evidence. *Integrative Medicine: A Clinician's Journal*, *16*(3), 42.

Mitochondrial support

- Antioxidant protection
- Provision of fuel
- Hormetic stimulation
- Cell membrane support
- Detoxification

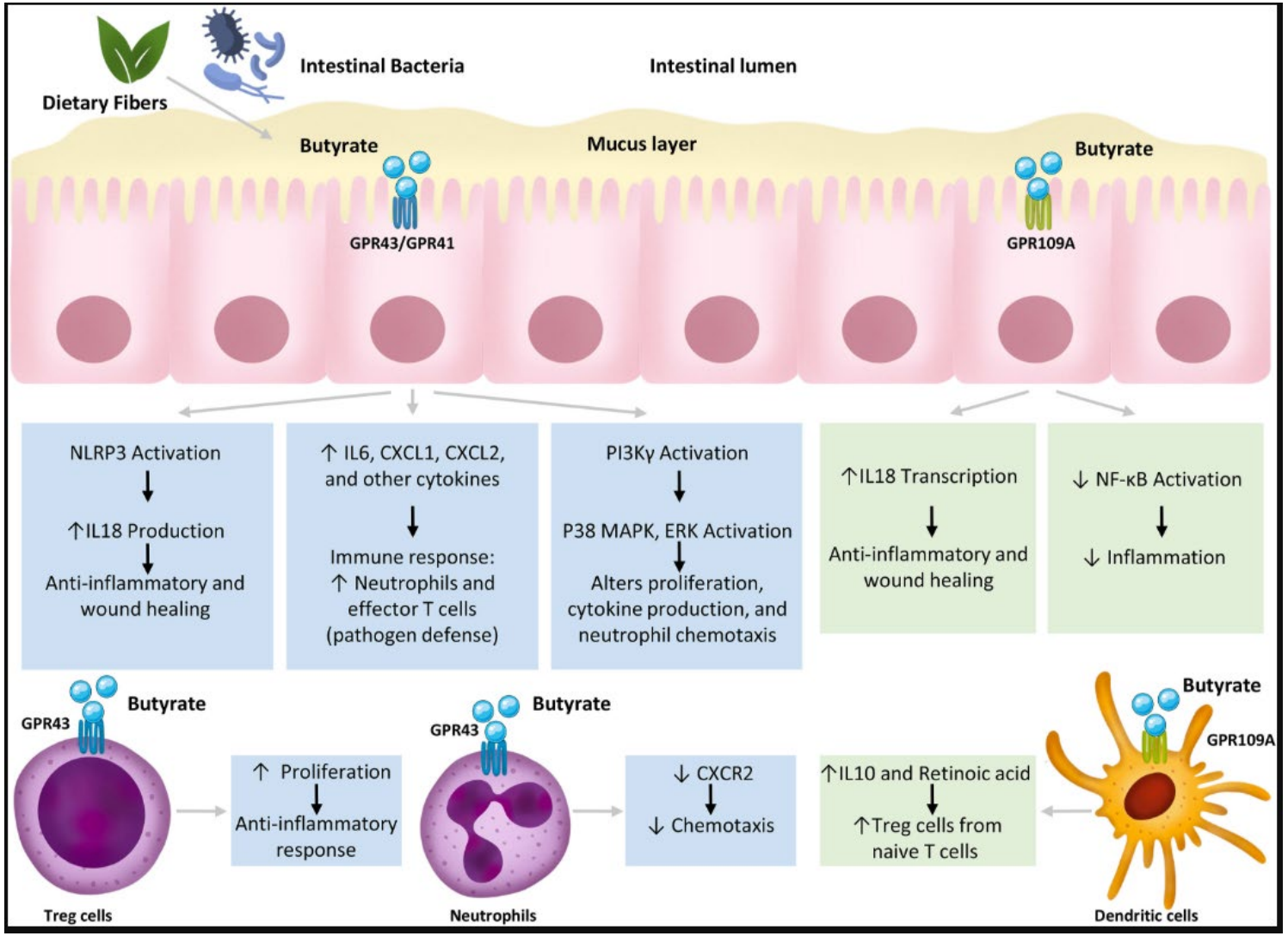


Nutritional support for Mitochondria

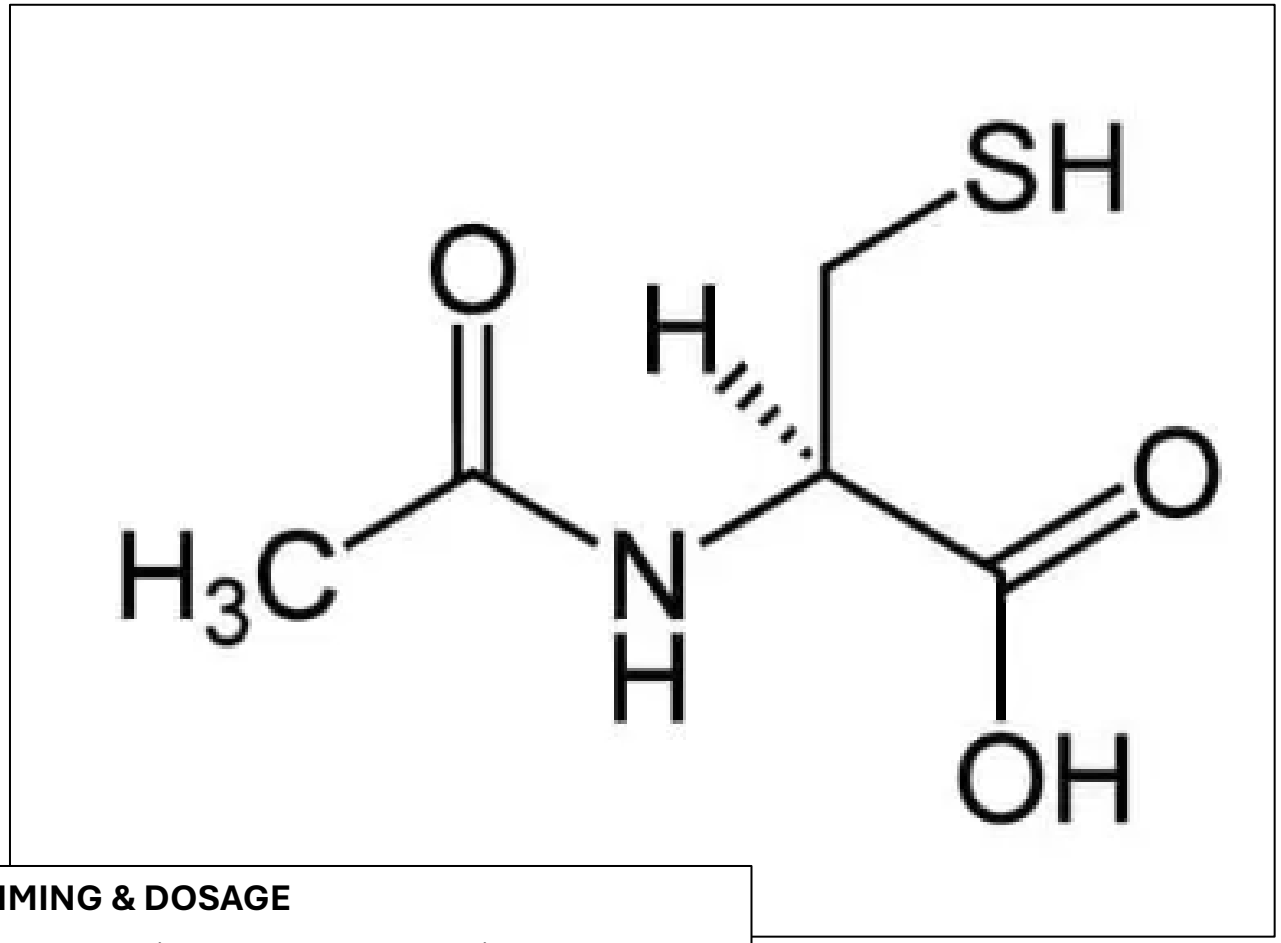
- Antioxidants- CO Q 10, Liposomal Glutathione, NAC
- NAD Precursors- NR, NMN
- Membrane Repair- Citicoline, Butyrate, Omega 3

Epithelial repair

- Butyrate is a major fuel source for colonocytes
- Exerts local immune regulatory and tissue healing effects through its' action on G coupled protein receptors, on the colonocytes and immune cells
- Reduces intestinal permeability, improves TJ integrity, increases mucin production (changes in genetic expression through HDAC inhibition, which supports goblet cell function) stimulates production of secretory IGA through t cell independent mechanism



N-Acetyl Cysteine: Therapeutic Actions



- Provides antioxidant protection, through mitigation of glutathione depletion and direct free radical scavenging
- Acts as a neuromodulator to protect against excitotoxicity
- Helps to restore resting state functional connectivity in the salience, default mode and executive control networks
- Has been shown to improve mitchondrial function in various disease states
- Mucolytic & Supports clearance of Heavy Metals at higher doses

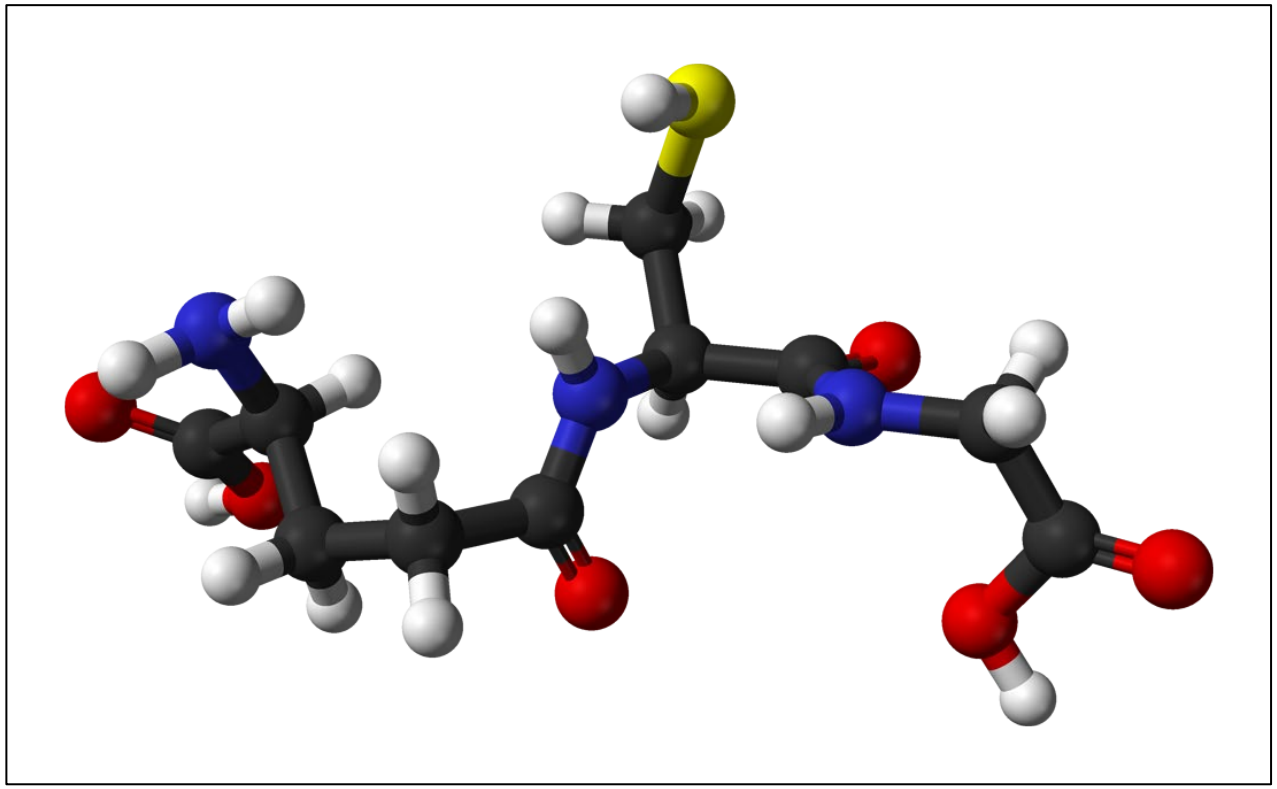
TIMING & DOSAGE

1-2 grams daily (approx. 150mg per kilo)

Divided dosage

Bhatti, J., Nascimento, B., Akhtar, U., Rhind, S. G., Tien, H., Nathens, A., & Da Luz, L. T. (2018). Systematic review of human and animal studies examining the efficacy and safety of N-acetylcysteine (NAC) and N-acetylcysteine amide (NACA) in traumatic brain injury: impact on neurofunctional outcome and biomarkers of oxidative stress and inflammation. *Frontiers in neurology*, *8*, 744.

Liposomal Glutathione



- Provides antioxidant protection, through mitigation of glutathione depletion
- Can be absorbed directly from the GIT an enter cells without processing
- Has been shown to increase blood and intracellular levels of glutathione in humans, with maximum increase being seen at the 4 week mark
- Increased glutathione levels have been shown to improve mitochondrial function in various disease states

TIMING & DOSAGE

250-500mg daily

2 weeks minimum

Bhatti, J., Nascimento, B., Akhtar, U., Rhind, S. G., Tien, H., Nathens, A., & Da Luz, L. T. (2018). Systematic review of human and animal studies examining the efficacy and safety of N-acetylcysteine (NAC) and N-acetylcysteine amide (NACA) in traumatic brain injury: impact on neurofunctional outcome and biomarkers of oxidative stress and inflammation. *Frontiers in neurology*, *8*, 744.

Clinical nutrition

Oral supplementation with liposomal glutathione elevates body stores of glutathione and markers of immune function

R Sinha, I Sinha, A Calcagnotto, N Trushin, J S Haley, T D Schell & J P Richie Jr

European Journal of Clinical Nutrition **72**, 105–111 (2018) | Cite this article

Subjects/Methods:

A 1-month pilot clinical study of oral liposomal GSH administration at two doses (500 and 1000 mg of GSH per day) was conducted in healthy adults. GSH levels in whole blood, erythrocytes, plasma and peripheral blood mononuclear cells (PBMCs) were assessed in 12 subjects at the baseline and after 1, 2 and 4 weeks of GSH administration.

Results:

GSH levels were elevated after 1 week with maximum increases of 40% in whole blood, 25% in erythrocytes, 28% in plasma and 100% in PBMCs occurring after 2 weeks ($P<0.05$). GSH increases were accompanied by reductions in oxidative stress biomarkers, including decreases of 35% in plasma 8-isoprostane and 20% in oxidized:reduced GSH ratios ($P<0.05$). Enhancements in immune function markers were observed with liposomal GSH administration including Natural killer (NK) cell cytotoxicity, which was elevated by up to 400% by 2 weeks ($P<0.05$), and lymphocyte proliferation, which was elevated by up to 60% after 2 weeks ($P<0.05$). Overall, there were no differences observed between dose groups, but statistical power was limited due to the small sample size in this study.

12 Healthy Adults
500mg or 1000mg of Liposomal Glutathione for 4 weeks

Increases observed after 2 weeks: 40% whole blood, 25% erythrocytes, 28% in plasma & 100% in PBMCs

Sinha, R., Sinha, I., Calcagnotto, A., Trushin, N., Haley, J. S., Schell, T. D., & Richie, J. P. (2018). Oral supplementation with liposomal glutathione elevates body stores of glutathione and markers of immune function. *European journal of clinical nutrition*, *72*(1), 105-111.



Nutritional Support for Barriers and Membranes

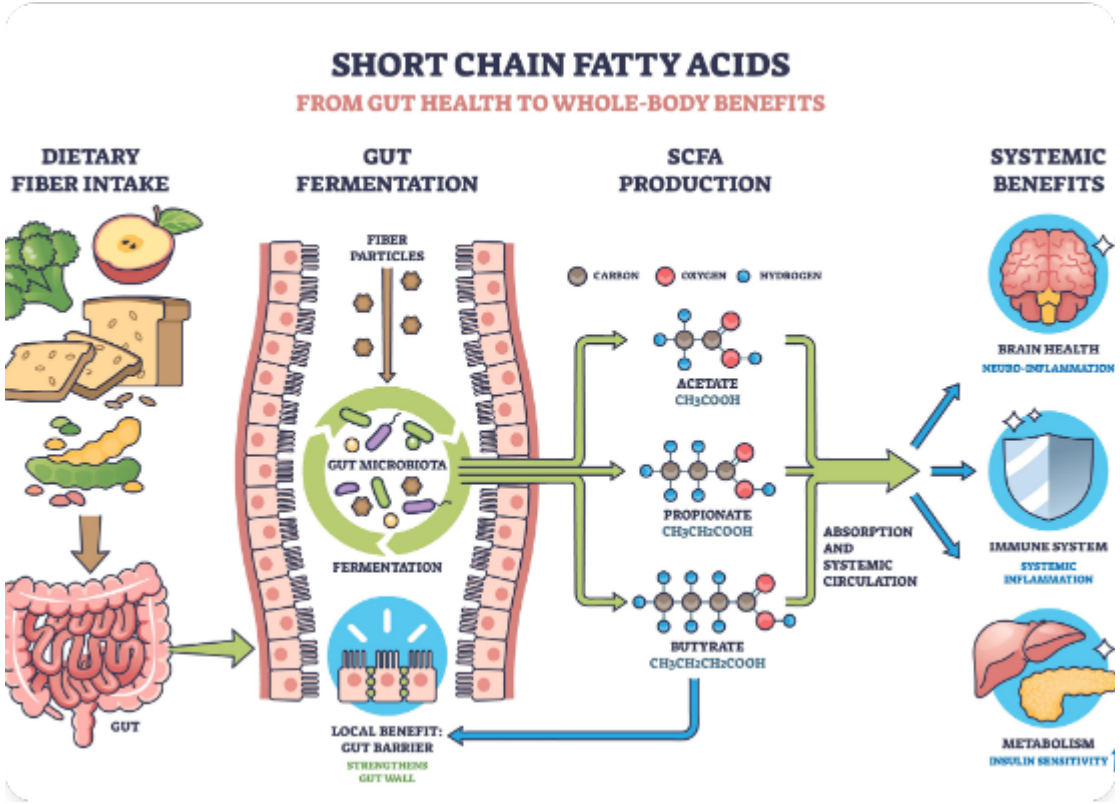
- Antioxidants- Liposomal Glutathione & Vitamin C, NAC
- BBB support- Citicoline & Butyrate, Zinc picolinate or glycinate
- Intestinal Barrier Support: Butyrate, Prebiotics, Zinc Carnosine
- Membrane Repair- Omega 3 & phospholipids,

Butyrate: Beyond the Gut

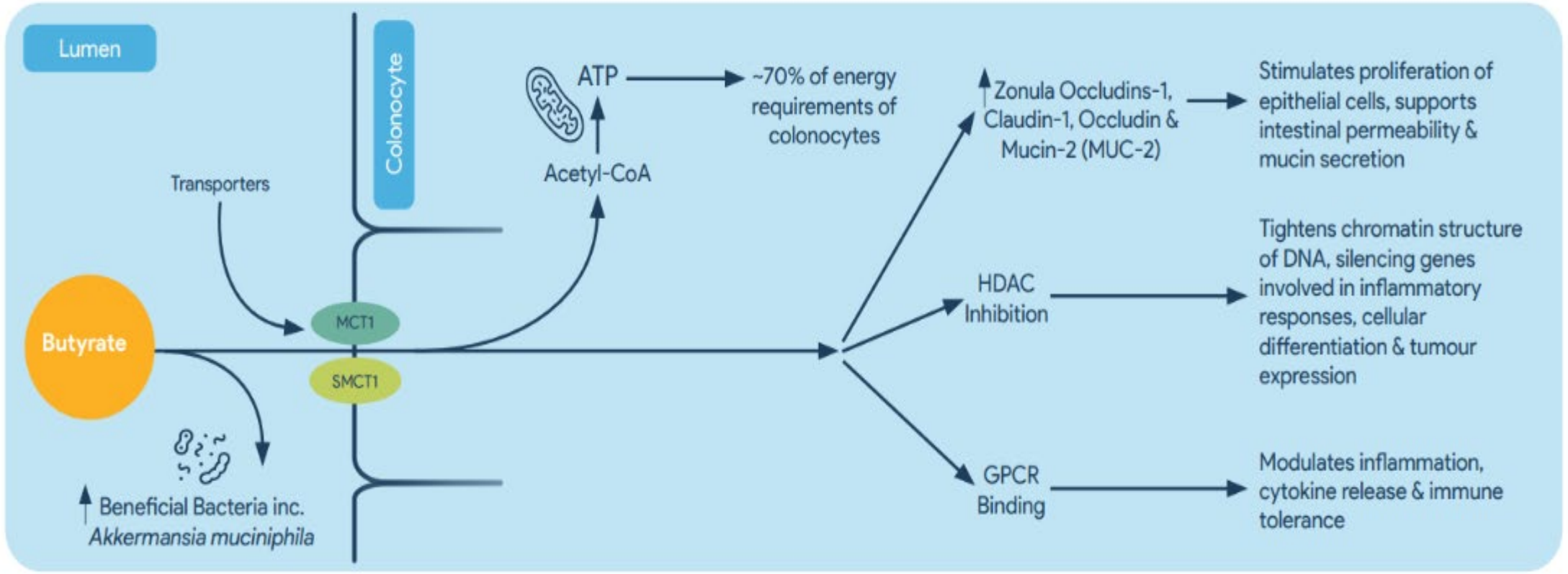
Short-Chain Fatty Acid (SCFA)

Butyrate is a postbiotic organic acid produced by the fermentation of dietary fiber by specific anaerobic bacteria in the colon.

While historically viewed as a simple fuel for colonocytes, modern neuroscience recognizes butyrate as a **systemic signaling molecule** that crosses the Blood-Brain Barrier to directly modulate neuronal health.

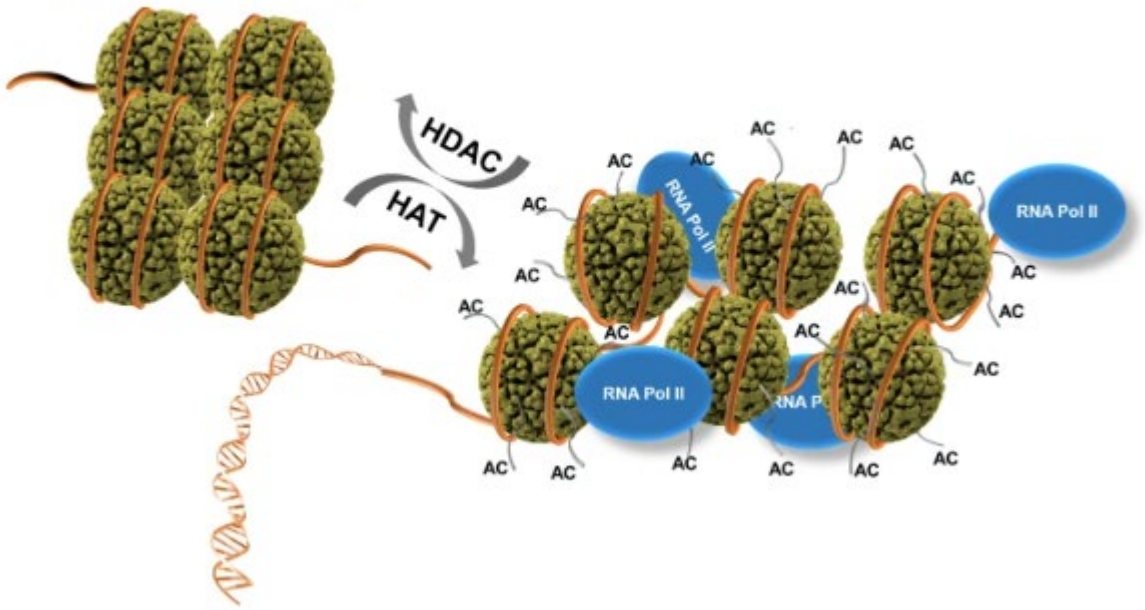


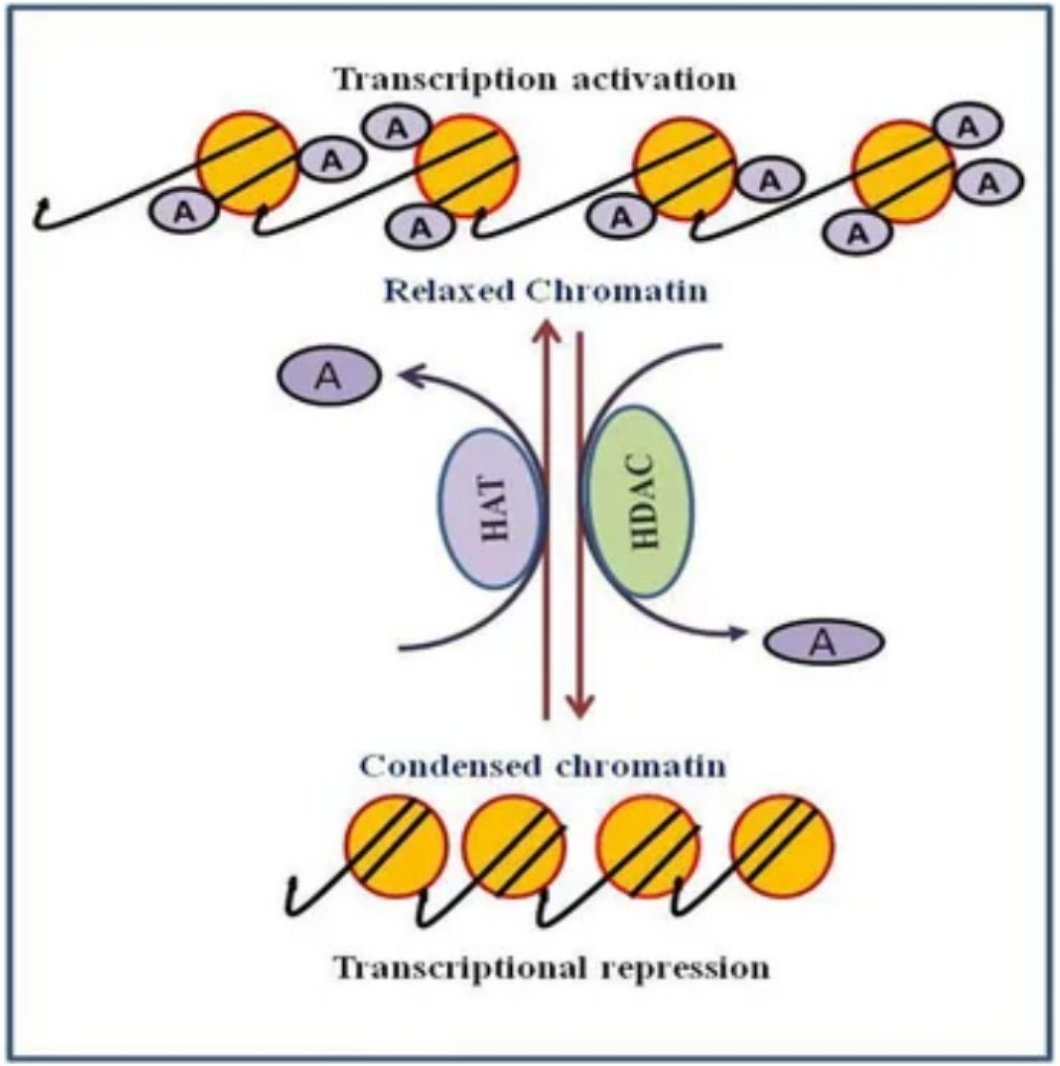
Butyrate Actions



Histone Deacetylase

Fig. 1: Regulation of gene expression and repression by histone acetylase (HAT) and histone deacetylase (HDAC).





Sources:



Prebiotics

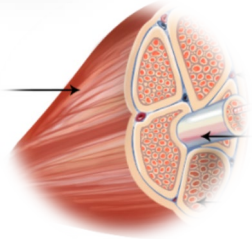


Dairy

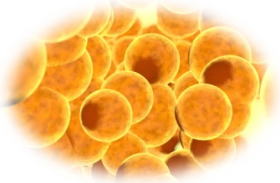


Direct Supplementation

Restores expression of PPARs to enhance insulin sensitivity

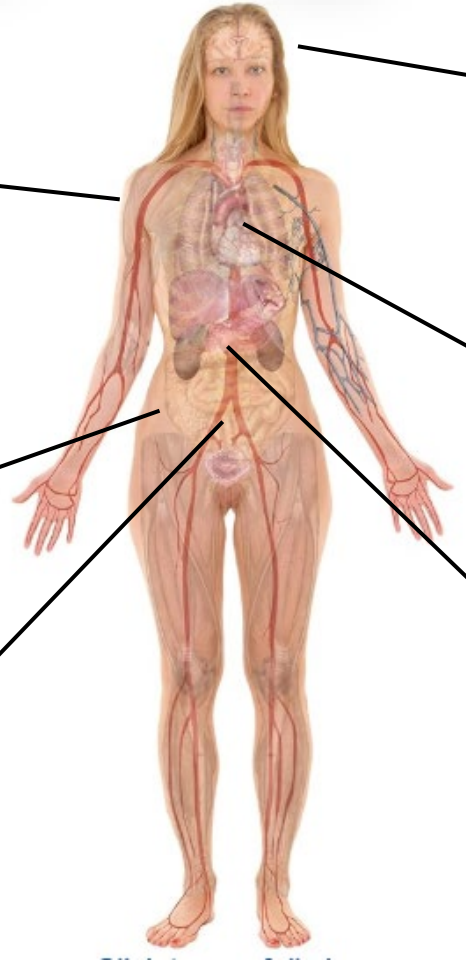


Enhances Beta oxidation and brown fat thermogenesis



Provides fuel for colonocytes, Supports mucin production, Enhances TJ integrity



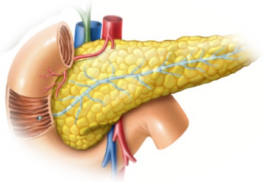




Supports BBB integrity
Reduces neuro-inflammation and supports neurogenesis

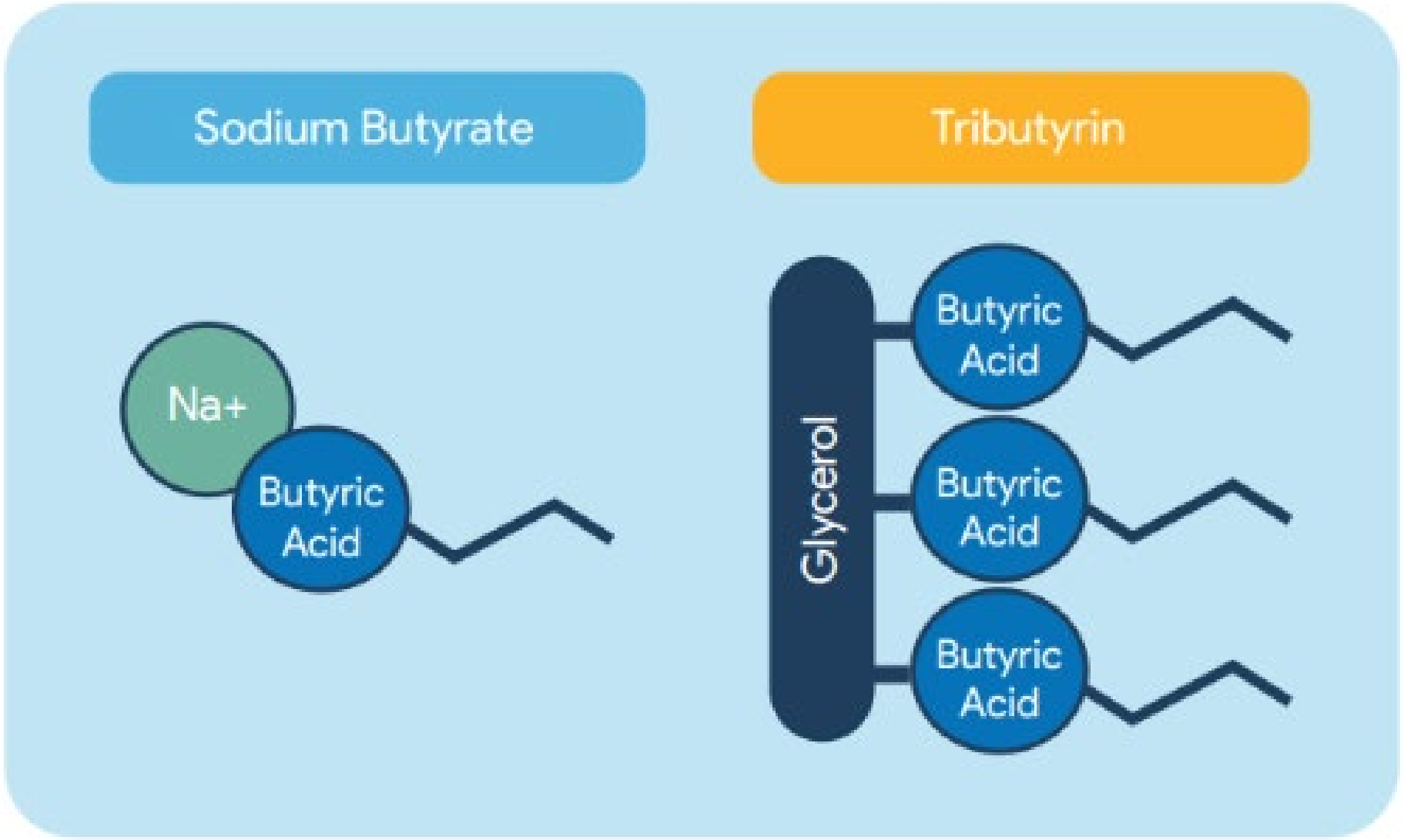


Activates Vagal Nerve, Supports CAP pathway, Regulates neuropeptide production



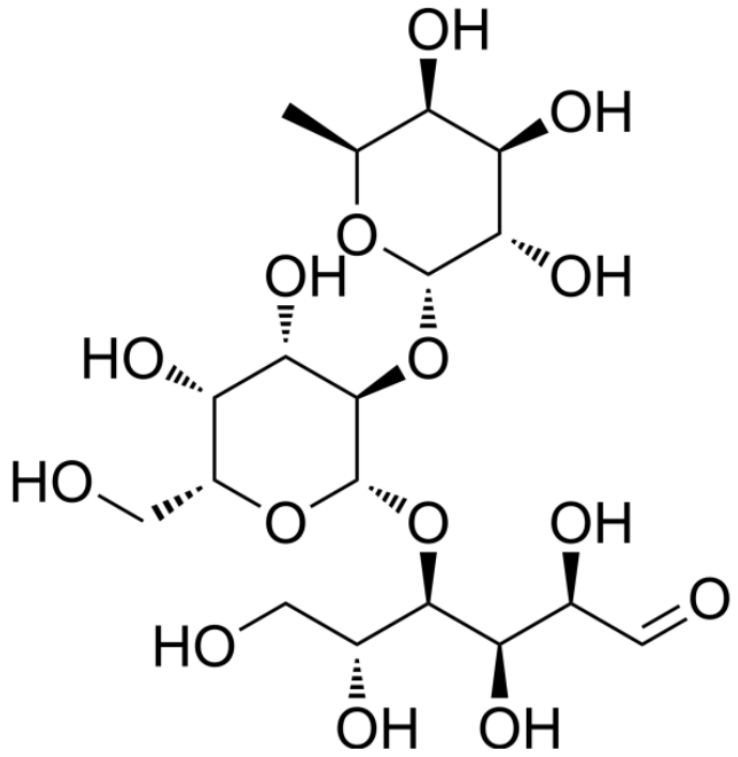
Protects beta cells from inflammation induced dysfunction

Systemic Effects of Butyrate


Sodium Butyrate
Tributyrin
Na+
Butyric Acid
Glycerol
Butyric Acid
Butyric Acid
Butyric Acid

2-FL

- Human Milk Oligosaccharide (HMO), abundant in human breastmilk
- Provides selective prebiotic activity to enhance bifidobacterium species and other SCFA producers
- Acts as a molecular decoy to reduce viral and pathogenic load



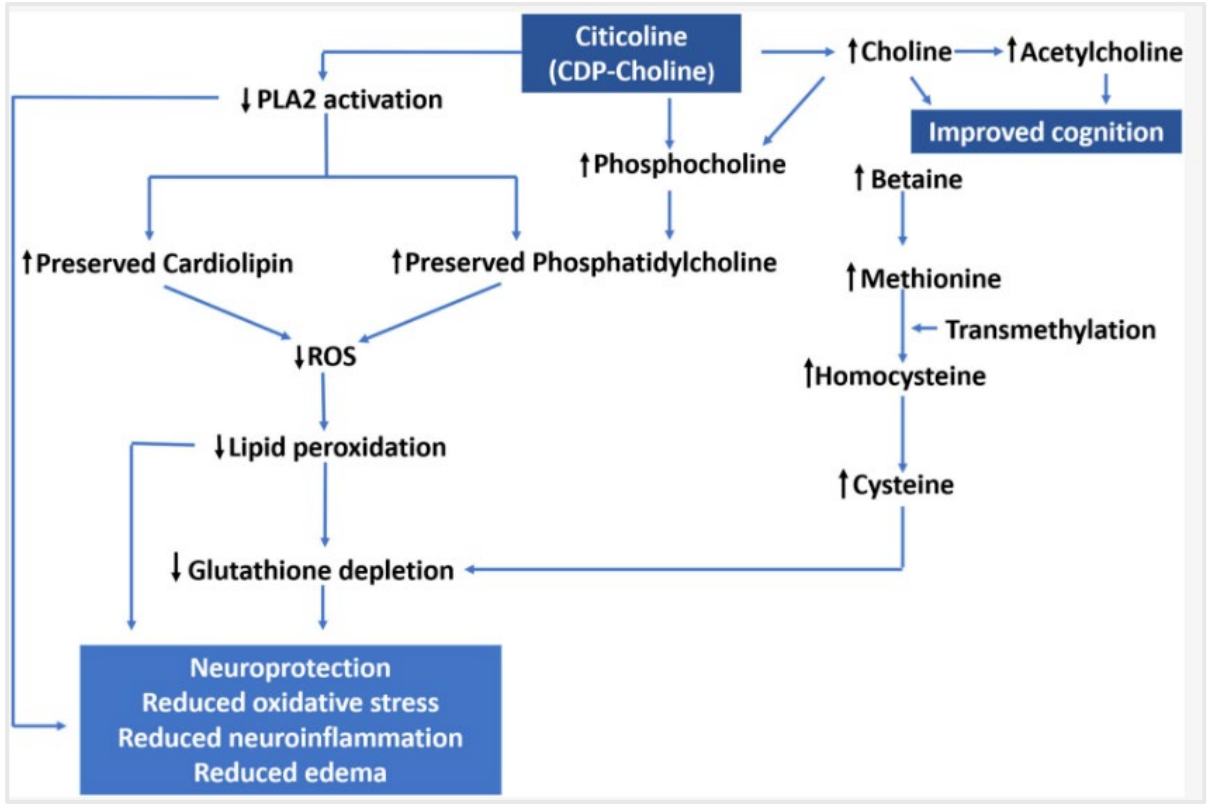
Pure Pharmaceutical-Grade 2-Fucosyllactose (2-FL)

Common dosage range: 5 - 20g per day
(It is recommended to start at lower doses such as 1.25 or 2.5g and work up to larger maintenance doses once tolerance is confirmed).

100g 2-Fucosyllactose (2-FL) Powder:

Density: 5ml = 2.5 g (0.5 g/ml)

Citicoline: Therapeutic Actions



- **NEUROPROTECTIVE**

  Supports neural cell membrane integrity and cell to cell communication

- **ACETYL CHOLINE PRECURSOR**

  Supports synaptogenesis and Cognitive recovery

- **SUPPORTS BBB integrity**

  Enhance TJ integrity with BBB

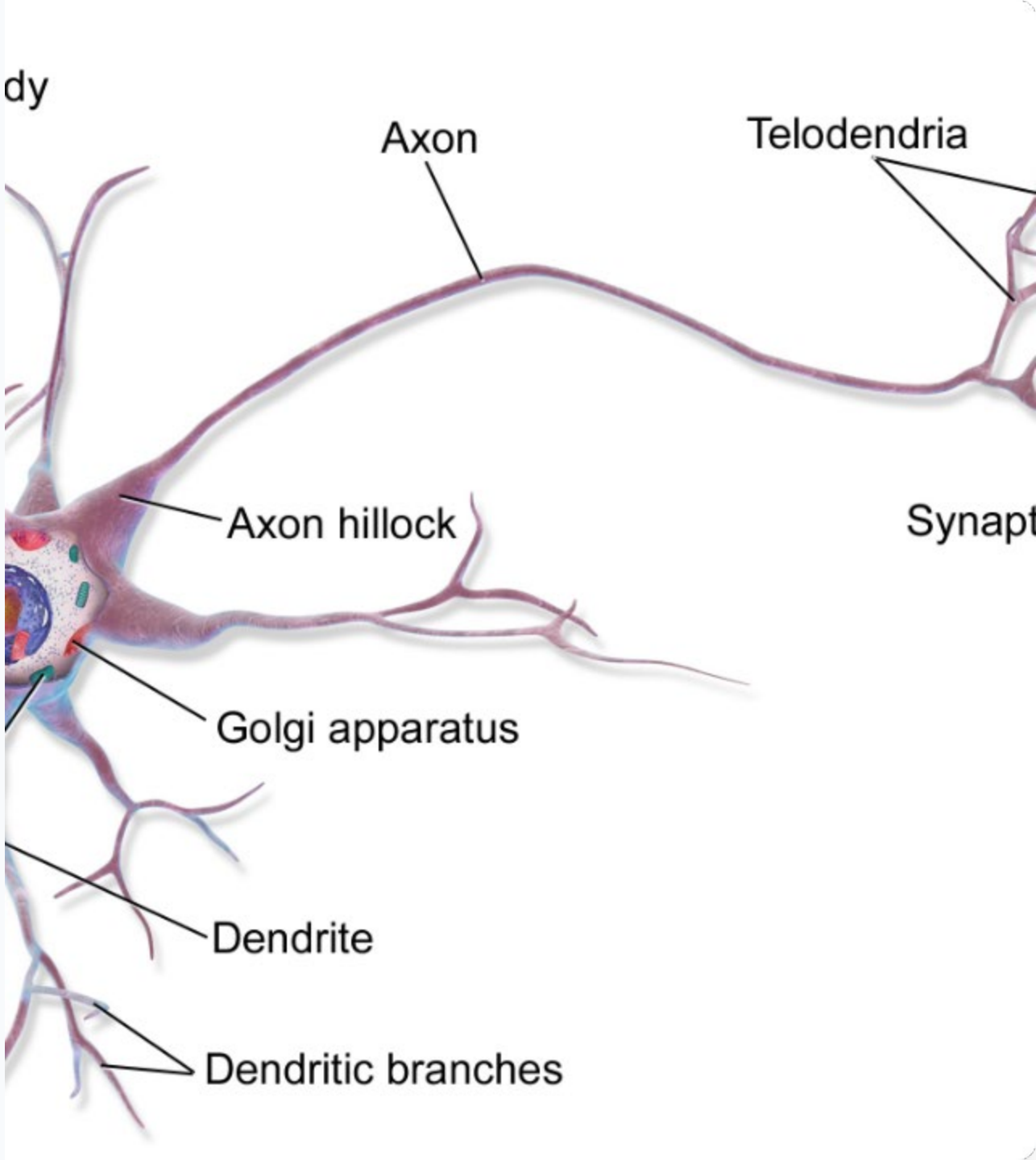
DOSAGE

- 300 mg daily to support BBB integrity
- 600mg- 2grams daily during cognitive decline or brain fog

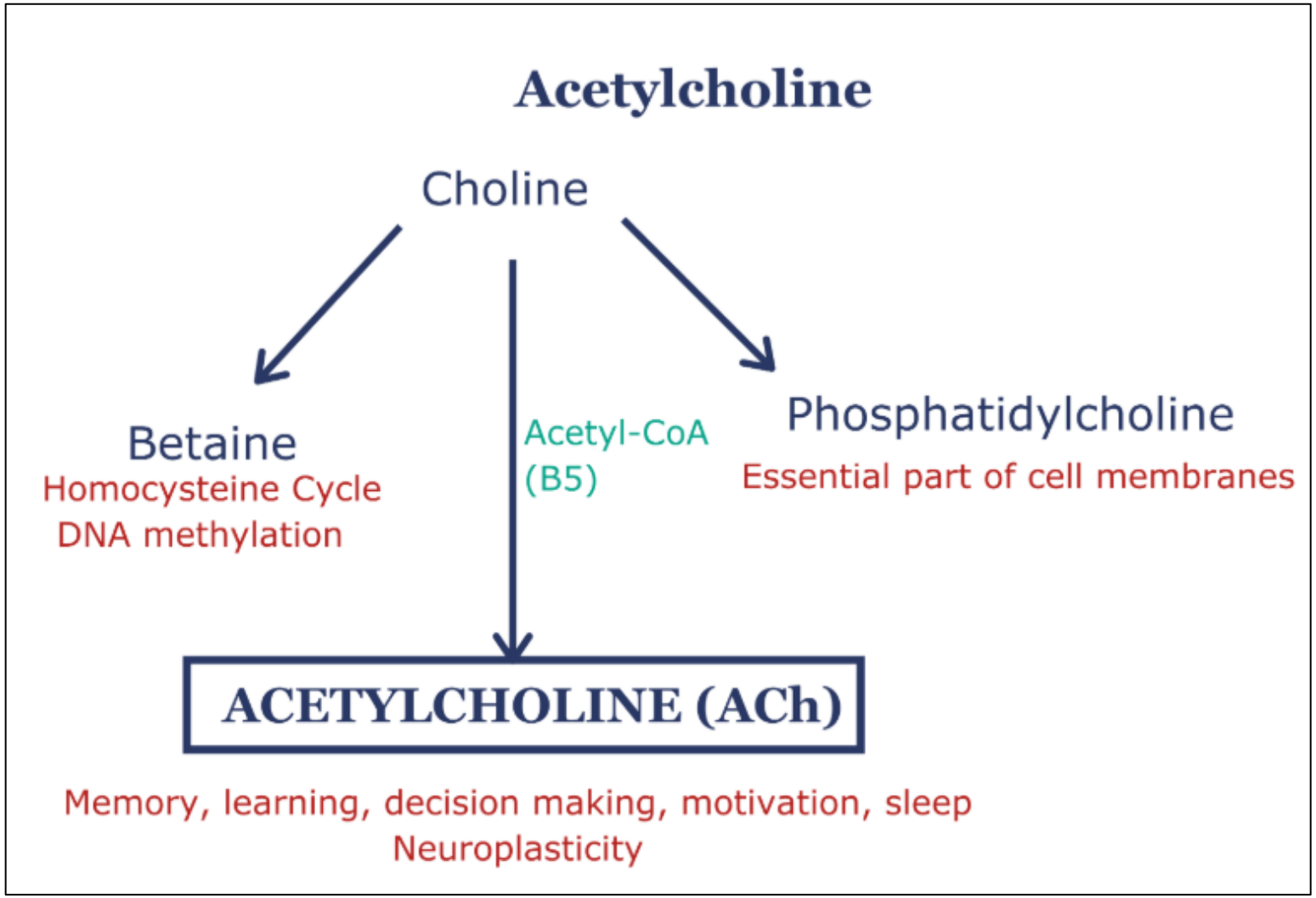
The Kennedy Pathway

Neuronal pools are depleted by age and stress. Citicoline reverses this trend by providing the raw materials for "The Kennedy Pathway."

By restoring membrane fluidity, it ensures that receptors and neurotransmitter transporters can function at peak capacity, preventing "neural static" and cognitive lag.



Choline Transport & Utilization



Sauna, Cold exposure, PEMF



REDUCES CHRONIC INFLAMMATION & OXIDATIVE STRESS



ENHANCEMENT OF WBC COUNTS AND ACTIVITY



CORTISOL AND VAGAL EFFECTS



ENHANCEMENT OF ATP PRODUCTION

Mindfulness, Sleep Hygiene & Vagal Nerve Stimulation



Reduces Chronic Inflammation



Encourages Parasympathetic activity

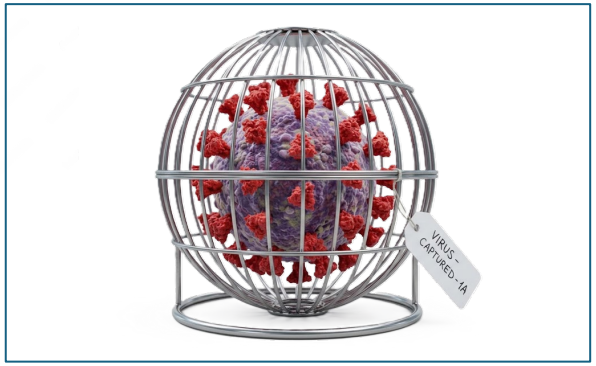


Helps to regulate cortisol levels

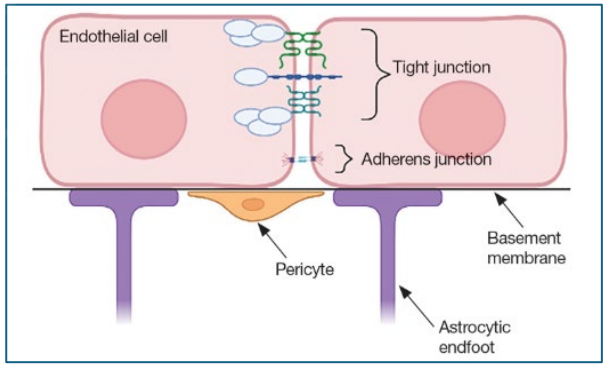


Reduces Neural Sensitivity & Provides Salutogenic Signaling

LEVERS WE CAN PULL



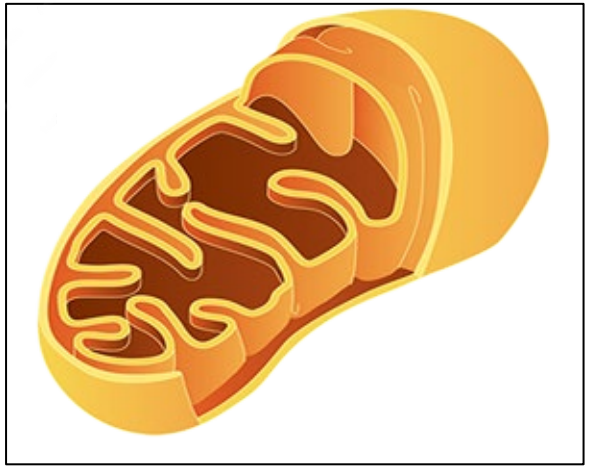
Reduce reactivation & support Immunity: AHCC, Astralgalus, St johns Wort, Wormwood, Liposomal C & D



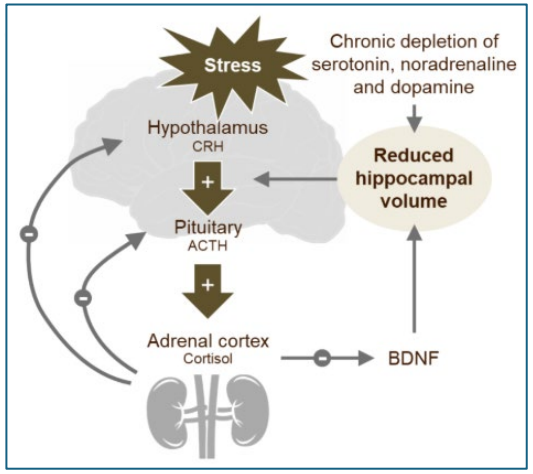
Support the integrity of Barriers: Butyrate, Zinc, Citicoline



Reduce chronic Inflammation systemically & in the CNS: Butyrate, PEA, Curcumin

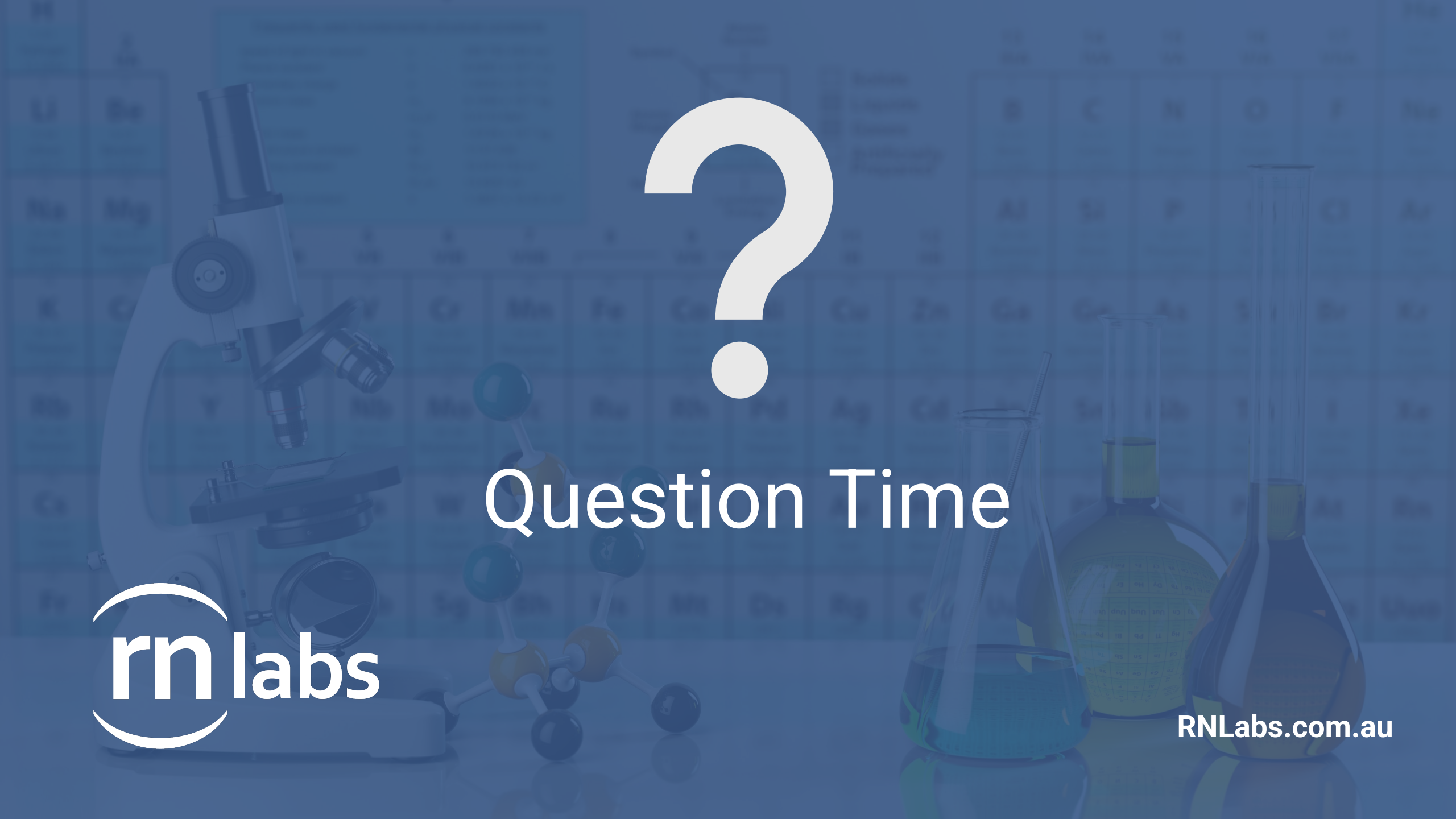


Support Mitochondrial Structure & Function: NAD Precursors, Omega 3 & phospholipids, Antioxidants, Hormetic therapies



Regulate HPA axis & Support Parasympathetic Tone: Mindfulness, VNS, Sleep hygiene, Counselling Adaptogenic and Anti depressant herbs

https://neurotorium.org/image



Question Time

rn labs

RNLabs.com.au

Thank You

rn labs

RNLabs.com.au