Casual Friday Presents

Back to School Focus

FM Strategies: Physiology, Pathology, Interventions.

A BIOGENETIX CLINICAL PRESENTATION
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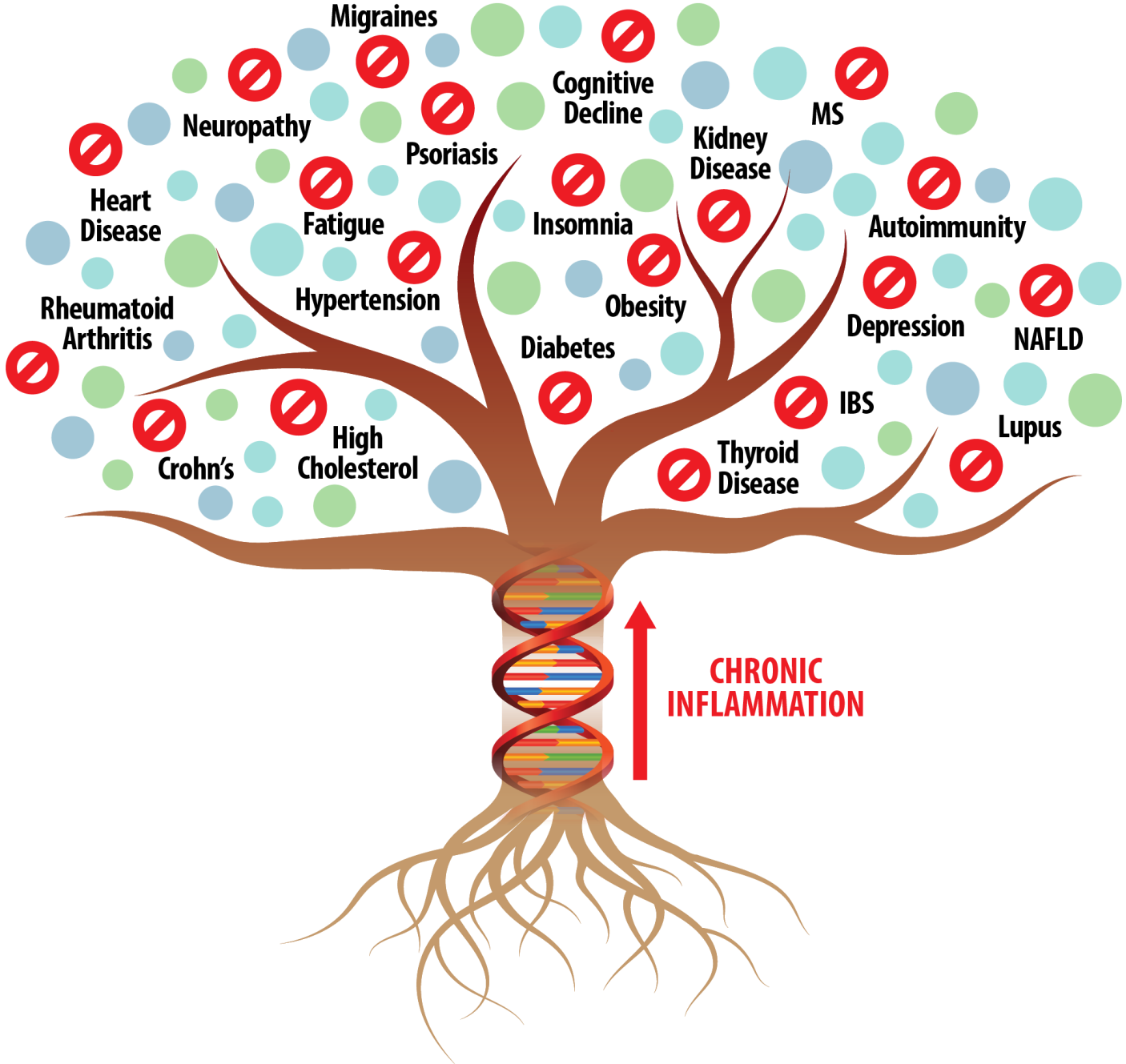
Migraines
Neuropathy
Psoriasis
Cognitive Decline
MS
Kidney Disease
Heart Disease
Fatigue
Insomnia
Autoimmunity
Hypertension
Obesity
Depression
NAFLD
Rheumatoid Arthritis
Diabetes
IBS
Lupus
Crohn's
High Cholesterol
Thyroid Disease
CHRONIC INFLAMMATION

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Neurotransmitters and Suggested Roles

Neurotransmitter	Role in focus and attention	Sub-optimal	Excessive
Acetylcholine	Sharpens sensory processing, sustained attention, learning and memory encoding	Poor concentration, forgetfulness, reduced mental clarity	Hypervigilance, sensory overload or difficulty disengaging attention
GABA	Filters irrelevant signals and limits distracting neuronal activity	Mental overstimulation, anxiety, distractibility and difficulty settling	Sedation, slowed processing, reduced alertness and impaired memory
Serotonin	Stabilizes mood and supports patience, impulse control and flexible attention	Irritability, impulsivity, anxious distraction and repetitive thinking	Apathy, reduced motivation, fatigue or cognitive dulling
Dopamine	Drives motivation, reward-based attention, working memory and task persistence	Low motivation, poor working memory, distractibility and difficulty initiating tasks	Overfocus, impulsivity, cognitive rigidity or attention drawn excessively toward rewards
Norepinephrine	Controls alertness, vigilance, signal detection and response to important information	Fatigue, low alertness, slow processing and poor sustained attention	Anxiety, hyperarousal, scanning for threats and fragmented attention

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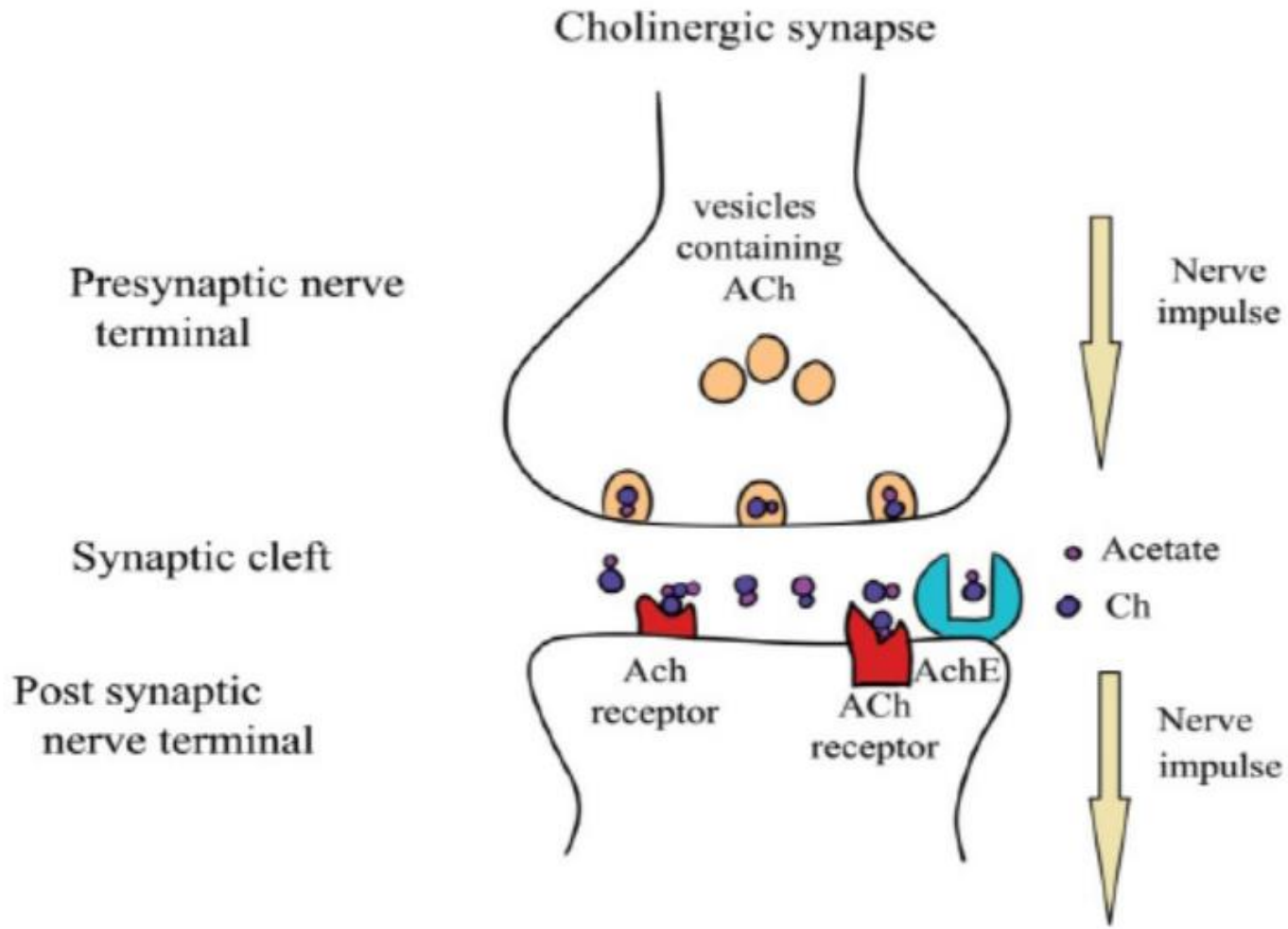

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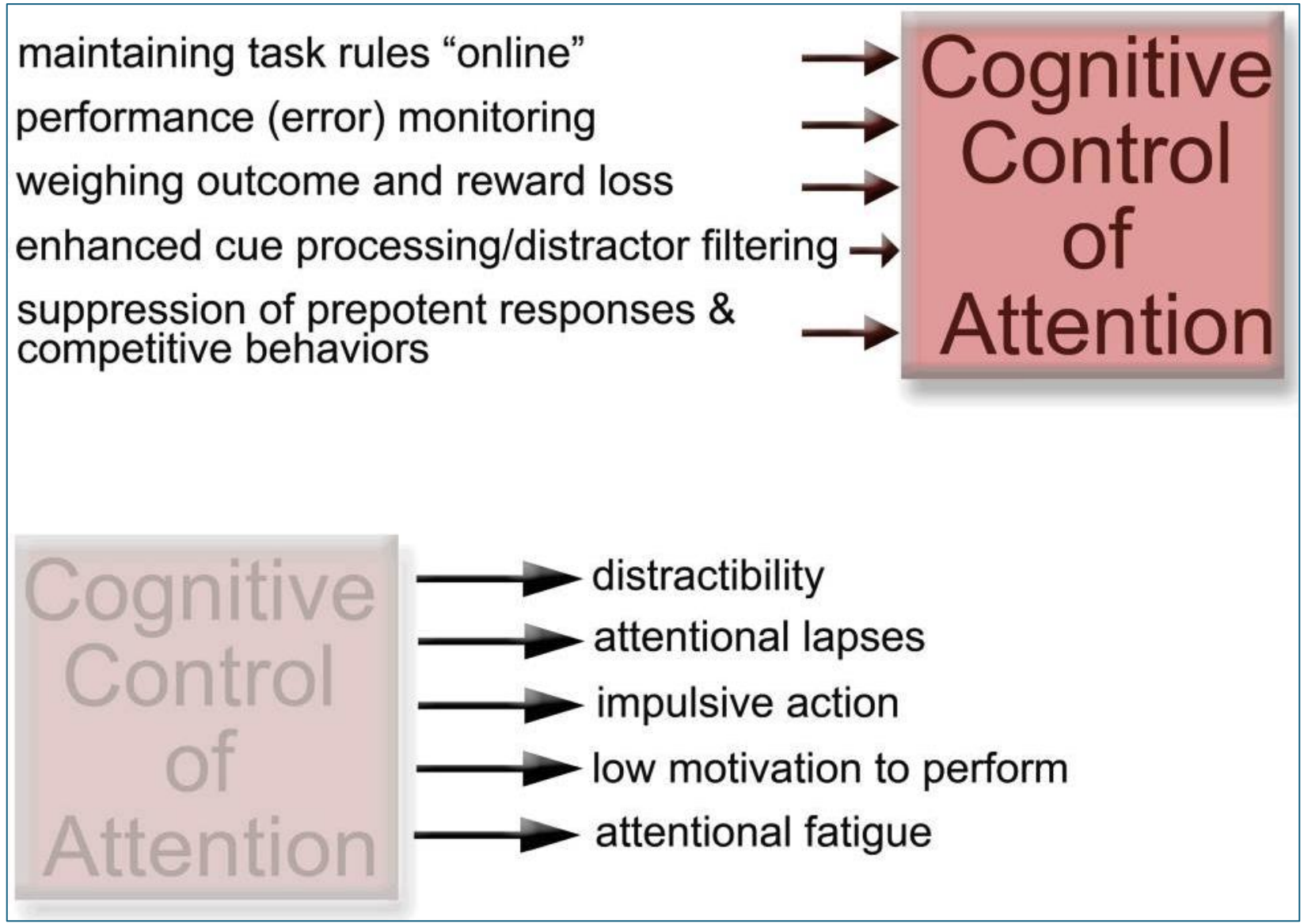
Deficits in Attentional Control: Cholinergic Mechanisms and Circuitry-Based Treatment Approaches

Martin Sarter [1], Giovanna Paolone [1]

The cognitive control of attention involves maintaining task rules in working memory (or "online"), monitoring reward and error rates, filtering distractors, and suppressing prepotent and competitive responses. Weak attentional control increases distractibility and causes attentional lapses, impulsivity and attentional fatigue. Levels of tonic cholinergic activity (changes over tens of seconds or minutes) modulate cortical circuitry as a function of the demands on cognitive control. Increased cholinergic modulation enhances the representation of cues, by augmenting cue-evoked activity in thalamic glutamatergic afferents, thereby increasing the rate of detection. Such cholinergic modulation is mediated primarily via α4β2* nicotinic acetylcholine receptors. Animal experiments and clinical trials in adult patients with ADHD indicate that attentional symptoms and disorders may benefit from drugs that stimulate this receptor. Tonic cholinergic modulation of cue-evoked glutamatergic transients in prefrontal regions is an essential component of the brain's executive circuitry. This circuitry model guides the development of treatments of deficits in attentional control.


maintaining task rules “online”
performance (error) monitoring
weighing outcome and reward loss
enhanced cue processing/distractor filtering
suppression of prepotent responses & competitive behaviors
Cognitive Control of Attention
Cognitive Control of Attention
distractibility
attentional lapses
impulsive action
low motivation to perform
attentional fatigue

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Distractibility is a cognitive construct that describes the increased probability for erroneous detection of (irrelevant) stimuli. Posner and colleagues (Posner, Snyder, & Davidson, 1980) defined detection as "*…the entry of information concerning the presence of a signal into a system that allows the subject to report the existence of the signal by an arbitrary response indicated by the experiment*". Erroneous detections can be triggered by salient stimuli or stimuli with unique features (often called "feature singletons"). Furthermore, weak top-down control of attention to the search field, object group or scene is a fundamental prerequisite for committing false detections (e.g., Theeuwes & Godijn, 2002).

Neurophysiological studies demonstrated that erroneous detection begins with the representation of a non-target cue in primary sensory regions (e.g., Roelfsema & Spekreijse, 2001). As will be discussed further below, such representation may not be sufficient for (false) detection. Likewise, weak signals may evoke an orienting response but they are not necessarily detected. The efficacy with which cues are amplified and distractors are filtered indicate the strength of attentional control processes. Cue amplification and distractor filtering are less effective if the demands on cognitive control are already high and if there is competition among stimuli (e.g., Lavie, 2005). fMRI studies have generated evidence in support of both cue amplification and distractor filtering as main mechanisms activated to combat distractibility (Polk, Drake, Jonides, Smith, & Smith, 2008). In the presence of explicitly defined distractors, as is the case with most laboratory tasks, evidence seems to favor neuronal mechanisms acting to enhance the processing of target cues (Nieuwenhuis & Yeung, 2005; Weissman, Warner, & Woldorff, 2004).

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Deficits in Attentional Control: Cholinergic Mechanisms and Circuitry-Ba

Mar

Au

PM

In the specific context of attentional task performance, impulsivity first concerns the frequent and compulsive disengagement from task performance (often termed, motor impulsivity). This form of impulsivity involves readily activated sets of complex yet fixed action patterns, such as grooming, exploration, locomotion, climbing, and fidgeting, all of which interfere with task performance. Deficits in the processing of time (Castellanos, et al., 2006) may foster the frequent execution of these motor programs.

Second, impulsivity concerns the failure to cancel or inhibit a specific behavioral response and, depending on task demands, to inhibit one response to execute a different, correct response. This more specific form of impulsivity often involves attentional reorientation and repositioning that is not controlled by task stimuli (impulsive action; Winstanley, Eagle, & Robbins, 2006; Winstanley, Olausson, Taylor, & Jentsch, 2010). A range of cognitive mechanisms may increase the propensity for such impulsive responses, including a weak "online" representation of the task rules. Both forms of impulsivity can be conceptualized as consequences of cognitive control deficits (e.g., Nigg, 2003). Weak cognitive control diminishes the capacity to suppress prepotent responding and competitive behaviors, and to sustain attention to the source of target cues and maintaining task-rules in working memory.

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Deficits in Attentional Control: Cholinergic Mechanisms and Circuitry-Based Treatment Approaches

Martin Sarter [1], Giovanna Paolone [1]

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PMCID: PMC3235713 NIHMSID: NIHMS339235 PMID: 22122146

To stabilize and recover performance in the presence of a distractor, subjects are required to enhance the processing cues, filter distractors, suppress the perhaps impulsive tendency to disengage from task, and to maintain motivation to perform (note that omission remained low in the experiment described above). Presumably, these mechanisms are activated by the onset of the distractor and the loss of reward early into the first distractor period (as the animals commit significantly more misses and false alarms). ***Augmented levels of tonic cholinergic activity are hypothesized to represent a main mechanism via which attentional control mechanisms are recruited and their efficacy is increased.***

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Cholinergic Modulation of Neuroinflammation: Focus on α7 Nicotinic Receptor

Roberta Piovesana [1,2], Michael Sebastian Salazar Intriago [3], Luciana Dini [3], Ada Maria Tata [3,4,*]

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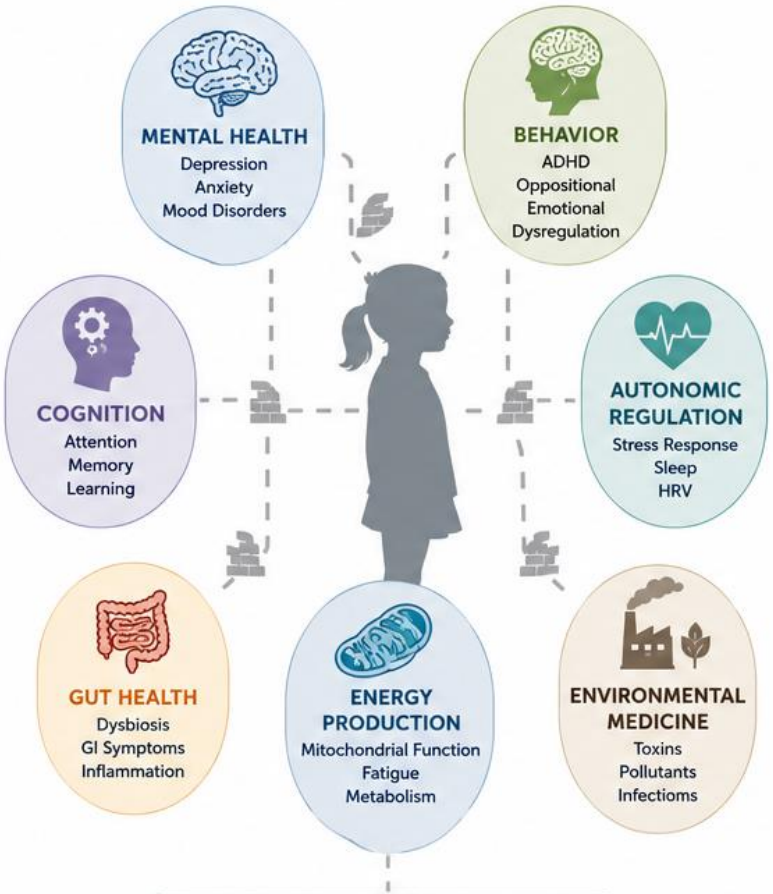
Abstract

All nervous system pathologies (e.g., neurodegenerative/demyelinating diseases and brain tumours) develop neuroinflammation, a beneficial process during pathological events, aimed at removing damaged cells, toxic agents, and/or pathogens. Unfortunately, excessive inflammation frequently occurs during nervous system disorders, becoming a detrimental event capable of enhancing neurons and myelinating glial cell impairment, rather than improving their survival and activity. Consequently, targeting the neuroinflammation could be relevant for reducing brain injury and rescuing neuronal and glial cell functions. Several studies have highlighted the role of acetylcholine and its receptors in the regulation of central and peripheral inflammation. In particular, α7 nicotinic receptor has been described as one of the main regulators of the "brain cholinergic anti-inflammatory pathway". Its expression in astrocytes and microglial cells and the ability to modulate anti-inflammatory

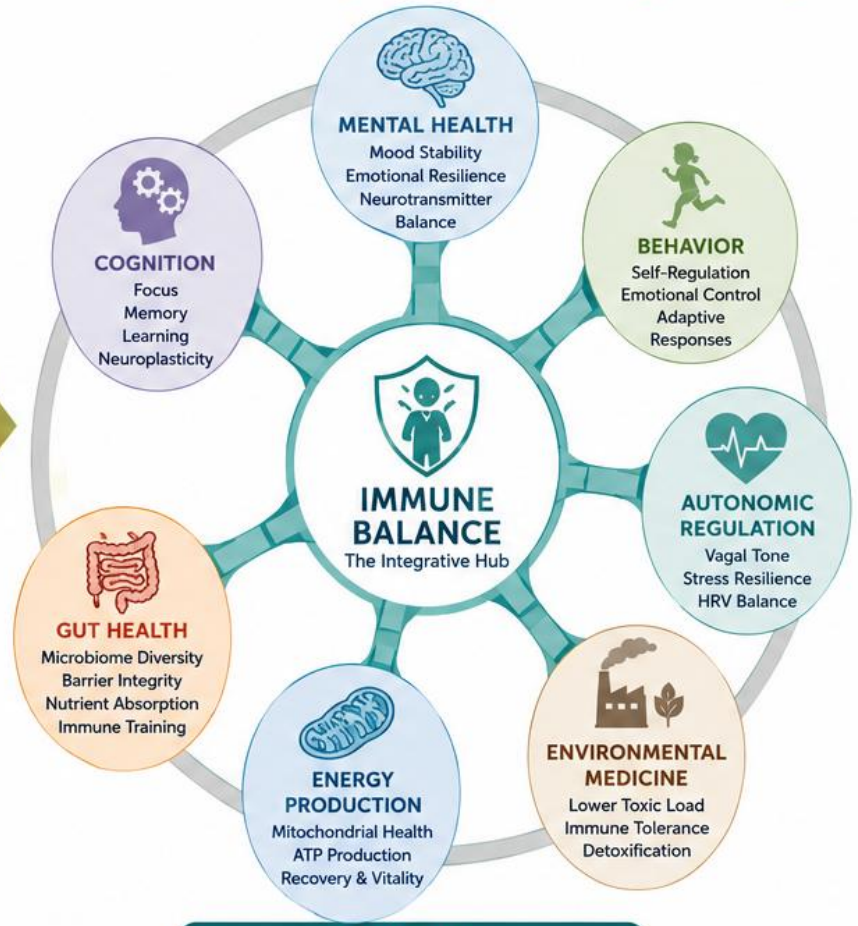
THE IMMUNE SYSTEM:
THE UNDERAPPRECIATED BRIDGE IN PEDIATRIC RESILIENCE

THE CONVENTIONAL MODEL
Systems Are Viewed in Isolation



OUTCOME
Fragmented Care, Missed Connections, Persistent Symptoms, Limited Resilience

THE FUNCTIONAL MEDICINE MODEL
The Immune System Bridges and Integrates All Systems



OUTCOME
Whole-Child Health, Systemic Resilience, Optimal Development, Lifelong Well-Being

HOW THE IMMUNE SYSTEM CONNECTS THE DOTS

- Communicates with the brain (cytokines, vagus nerve, microglia)
- Regulates inflammation and recovery
- Shapes the microbiome and gut barrier
- Influences neurotransmitters and mood
- Responds to environmental exposures
- Uses energy to defend, repair and adapt
- Teaches tolerance, calibrates threat, builds resilience

IMMUNE BALANCE IS NOT JUST ABOUT AVOIDING DISEASE— IT IS THE FOUNDATION OF RESILIENCE.

Energy Transfer Regulation

Mitochondria are not just “powerhouses.”
They are:

1. stress sensors
2. immune regulators
3. inflammation mediators
4. neurodevelopmental regulators
5. resilience organelles

A child with impaired mitochondrial function has reduced capacity to:

1. regulate emotions
2. recover from stress
3. sustain attention
4. tolerate frustration
5. maintain stable energy
6. mount healthy immune responses

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Basics, first.

1. Cut the distractor load.
2. Protect sleep and a stable wake time.
3. Practice attention in short, winnable doses.
4. Use motivation on purpose.
5. Daily movement, preferably outdoors.

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Unprocessed/ Minimally processed	Processed ingredients	Processed	Ultra - processed
 Fruit	 Oils	 Ham	 Supermarket bread
 Vegetables	 Sugar	 Cheese	 Ready meals
 Eggs	 Salt	 Bread	 Breakfast cereal
 Milk	 Honey	 Bacon	 Cakes
 Meat	 Butter	 Salted nuts	 Crisps

The Standard:

Drink water c/salt.
Eat meat and eggs.
Enjoy fruit.
Have nuts.
Use vegetables.
Experience sugars.

*Reward protein.

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NUTRITION & IMMUNE BALANCE

Key Nutrients That Regulate, Support & Strengthen the Immune System

VITAMIN D

- Innate immunity
- T-regulatory cells
- Inflammatory modulation

SUPPORTS
Immune tolerance & balanced responses

BEST FOOD SOURCES

Fatty fish, egg yolks, mushrooms, fortified foods

Sunlight is a key source

ZINC

- Barrier integrity
- Immune signaling

SUPPORTS
Mucosal barriers & immune defense

BEST FOOD SOURCES

Oysters, beef, chickpeas, pumpkin seeds, lentils, nuts

OMEGA-3s

- Inflammatory resolution
- Specialized pro-resolving mediators (SPMs)

SUPPORTS
Resolution of inflammation & immune balance

BEST FOOD SOURCES

Fatty fish, flaxseeds, chia seeds, walnuts, algae oil

MAGNESIUM

- Stress modulation
- Inflammatory control

SUPPORTS
Calm immune response & reduces overactivation

BEST FOOD SOURCES

Leafy greens, nuts, seeds, avocado, legumes, dark chocolate

PROTEIN

- Antibodies
- Immune cell production

SUPPORTS
Structural components of immune cells

BEST FOOD SOURCES

Lean meats, fish, eggs, dairy, beans, lentils, quinoa, tofu

A nutrient-dense diet provides the building blocks and regulators your immune system needs to detect threats, respond appropriately, and return to balance.

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Everyday Basics

In addition to a health-optimized diet:

1. B complex
2. Magnesium/Multimineral
3. Vit C
4. Multivitamin
5. Fish oil
6. Probiotic
7. Creatine

*PS support, Biog-max PC.

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We Want to Hear from You!

Give us your Casual Friday feedback
with this short 5-question survey.

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