NEUROMETABOLIC PROFILE - ATTENTION (NAP)

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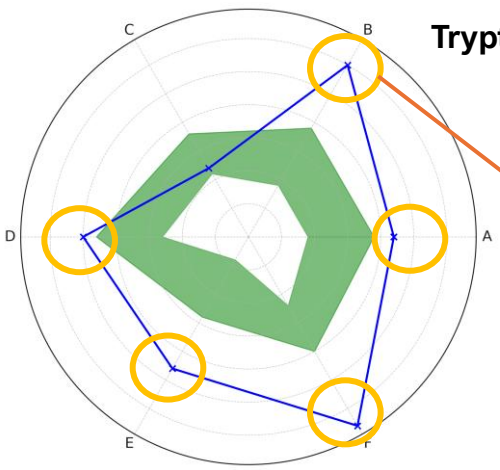
1. Introduction

Neurometabolism refers to the biochemical **processes that enable the nervous system to function – regulating it's signalling, energy production, and maintenance -** including the brain, spinal cord, and peripheral nerves. These processes depend on neurochemicals (e.g. tryptophan, serotonin, 5HIAA) and their metabolic pathways. Numerous human studies have shown that neurochemicals correlate with mental health, reinforcing the view that mental health reflects the condition of the entire nervous system, not just the brain in isolation. Exploratory human studies have particularly examined urinary biomarkers of dopaminergic and serotonergic metabolism (see References).

A **neurometabolic assessment** evaluates the functional state of the nervous system by analyzing its metabolic activity to reveal pathway level impairments that may influence mental health. A **neurometabolic profile** is a visualisation of the above.

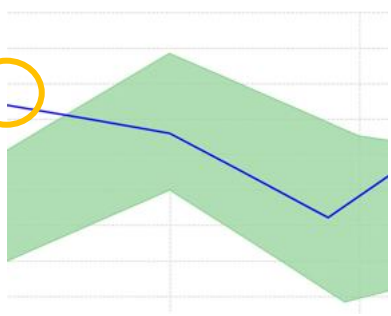
2. Neurometabolic Profile

The profile visualises an example of 1 of 6 measured values representing different neurometabolic pathways, which have all impact on the attention symptoms. 5 values are higher than optimal range. 1 of them, tryptophan is presented and described further as an example.



Tryptophan

Tryptophan is the first neurosubstance of the serotonergic pathway. The tryptophan value, in opposite to the rest of the pathway, is higher than optimal range.



Tryptophan Serotonergic pathway

3. Attention characteristics in profiles with high tryptophan

Table 3.1 presents the research on the impact of high tryptophan on the attention (references at the back of the document):

Effects of high tryptophan on attention	Explanation
Slower reaction time	Serotonin reduces prefrontal dopamine
Reduced sustained attention	Vigilance impairment in CPT tasks
Fatigue / sedation	Serotonin promotes sleepiness
Reduced working memory	Serotonin–dopamine interaction
Cognitive slowing	Observed in high-dose trials

4. Mechanisms of high tryptophan and nutrients strategies

Table 4.1 presents several contributing factors to high levels of tryptophan reflected in research (3 factors presented as examples, references at the back of the document):

Common contributing factors to high tryptophan	Mechanism	Relevant nutrients used in studies
Low activity of Tryptophan 2,3-dioxygenase (TDO)	TDO (in the liver) converts tryptophan to kynurenine. Low activity → reduced breakdown.	High amino acid intake, Vit B6, Iron, Zinc
Low activity of Indoleamine 2,3-dioxygenase (IDO)	IDO (extrahepatic) handles immune-mediated tryptophan catabolism. If inflammation is low or enzyme genetically less active, tryptophan can stay elevated.	Iron, Selenium, Omega 3 & 6, Zinc, Vit D
Vitamin B6 or B2 deficiency	Cofactors for enzymes downstream (kynurenine → niacin/NAD^+). Lack of these causes “bottleneck” and accumulation upstream.	Vit B6 and/or B2

5. Research on pharmacological options

Scientific literature describes several group of medications and mechanisms commonly explained in pharmacology references to address different neurochemical levels:

Main category	Subgroup	Example drugs	Main signalling substances	Core action in the brain
STIMULANTS	**Methylphenidate-based**	Methylphenidate, Ritalin, Concerta	↑ Dopamine, ↑ Noradrenaline	Blocks reuptake of dopamine & noradrenaline → increases signalling in prefrontal cortex and striatum → improves attention, motivation and executive function
STIMULANTS	**Amphetamine-based**	Lisdexamfetamine ,Dexamphetamine	↑↑ Dopamine, ↑ Noradrenaline	Increases release and blocks reuptake of dopamine & noradrenaline → stronger stimulation of attention and reward circuits
NON-STIMULANTS	**Noradrenaline reuptake inhibitor (NRI)**	Atomoxetine	↑ Noradrenaline (and indirectly dopamine in PFC)	Selectively blocks norepinephrine transporter → improves attention and impulse control without stimulant effect
NON-STIMULANTS	**Alpha-2 adrenergic agonists**	Guanfacine, Clonidine	Noradrenaline (α2A receptor)	Stimulates α2A receptors in prefrontal cortex → strengthens executive control networks, reduces hyperactivity, impulsivity and emotional dysregulation

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