# NEUROMETABOLIC PROFILE (NEP) - MOOD AND ENERGY
Jane Doe



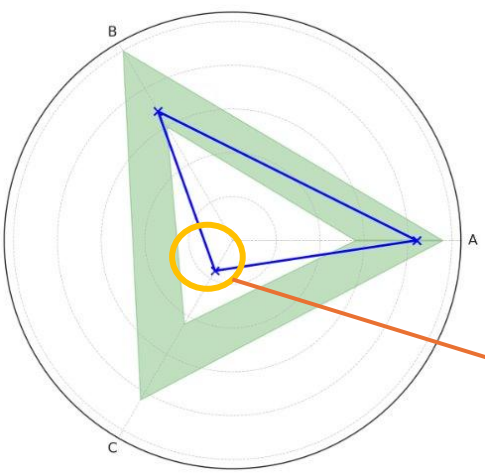
## 1. Introduction

**Neurometabolism** refers to the biochemical **processes that enable the nervous system to function – regulating its 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 for informational purposes only.

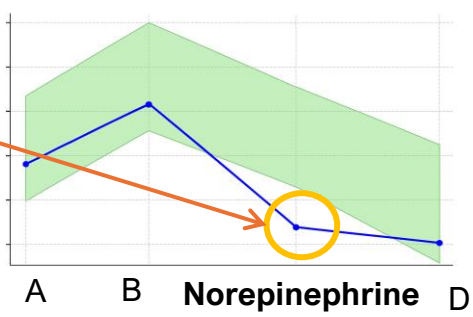
## 2. Neurometabolic Profile (NEP) of Jane Doe

NEP visualises 3 measured values representing different neurometabolic pathways, which have all may impact the mood and energy symptoms. Of those 3, one value, Norepinephrine, is lower than optimal range and described further as an example.




**Norepinephrine**

Several neurochemicals of the dopaminergic/norepinergic pathway are measured. The norepinephrine value, in opposite to the rest of the pathway, is lower than optimal range.




## 3. Impact of NP with low norepinephrine on mood and energy

Table 3.1 presents the research on the impact of low norepinephrine on the mood and attention (references at the back of the document):

| Effect of Low Norepinephrine (NE) | Explanation | Evidence Strength |
|---|---|---|
| Low mood | Reduced signaling in prefrontal cortex | Strong |
| Low energy & drive | Reduced activation of the locus-coeruleus arousal system | Strong |
| Cognitive slowing | Less efficient prefrontal processing | Strong |
| Excessive sleepiness | Reduced sympathetic tone | Moderate |
| Poor concentration & impaired attention | NE modulates attention networks in prefrontal cortex | Moderate |

## 4. Mechanisms of low norepinephrine and nutrients strategies

Table 4.1 presents several contributing factors to low levels of norepinephrine reflected in research and the applicable nutrients strategies (references at the back of the document):

| Common contributing factors to low norepinephrine (NE) | Mechanism | Relevant nutrients used in studies |
|---|---|---|
| Low availability of precursors (L-tyrosine) | Reduced dopamine → NE synthesis via the catecholamine pathway | L-Tyrosine – improves NE synthesis (multiple human trials) |
| Low availability of cofactors for NE synthesis | Vitamin C is required for dopamine β-hydroxylase, the enzyme converting dopamine → norepinephrine | Vitamin C – increases NE levels in adrenal tissue and circulation (human and animal studies) |
| Low availability of cofactors for NE synthesis | Copper is a cofactor for dopamine β-hydroxylase; deficiency decreases NE production | Copper – restores DBH activity and NE synthesis (clinical deficiency studies) |
| High metabolism of NE | COMT is the enzyme that breaks down norepinephrine | Rhodiola rosea contains rosavins and salidroside, which can partially inhibit COMT, improves uptake of tyrosine (NE precursor), and enhances NE release (multiple human and animal studies) |

## 5. Research on pharmacological options

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

| Groups | Mechanisms |
|---|---|
| SSRIs | selective serotonin reuptake inhibitors as medications that influence serotonergic pathways by reducing serotonin reuptake transporters |
| SNRIs | Serotonin–Norepinephrine Reuptake Inhibitors, influencing serotonin and norepinephrine by reducing their reuptake transporters |
| NDRIs | Norepinephrine–Dopamine Reuptake Inhibitors, influencing serotonin and norepinephrine by reducing their reuptake transporters |
| NaSSAs | Noradrenergic and Specific Serotonergic Antidepressants, increasing release of norepinephrine and serotonin by blocking inhibitory receptors (α2-adrenergic and 5-HT2 and 5-HT3 serotonin receptors) |

## 6. Jane Doe's values compared with optimal ranges (in g of Creatinine)

| Neurochemical | Optimal range (OR) | Lower than OR | In OR | Higher than OR |
|---|---|---|---|---|
| **Serotonergic pathway** | | | | |
| A | 3970-8450 mcg/g | | 5574mcg | |
| B | 61.0-103.2 mg/g | | 87.1 mcg | |
| C | 2988-5850 mcg/g | | 5215 mcg | |
| **Glutaminergic pathway** | | | | |
| A | 37-71 mg/g | | 48 mcg | |
| B | 1515-2710 mcg/g | | | 5665 mcg |
| C | 193-367 mcg/g | | | 546 mcg |
| **Dopaminergic pathway** | | | | |
| A | | | 10.5 mcg | |
| B | 4790-10278 mcg/g | | 6763 mcg | |
| C | 279-588 mcg/g | 237 mcg | | |
| D | 144-240 mcg/g | | 164 mcg | |
| E | 658-1449 mg/g | | | 1520 mcg |
| F | 3737-7048 mcg/g | | | 9614 mcg |
| Norepinephrine | 15.0-28.1 mcg/g | 13.0 mcg | | |
| H | 17.9-31.7 mcg/g | | 18.8 mcg | |
| I | 2580-4766 mcg/g | | 2635 mcg | |
| J | 1.4-4.2 mcg/g | | 1.7 mcg | |
| **Neuroprotection and neuroinflammation** | | | | |
| A | 7.1-293.1 mg/g | | 9.2 mg | |
| B | 19.7-58.4 mg/g | | 19.7 mg | |
| C | 5.2-15.3 mcg/g | | | 49.7 mcg |
| E | 79-140 mcg/g | 67 mcg | | |
| F | 257-960 mcg/g | | 262 mcg | |
| G | 639-1200 mcg/g | 439 mcg | | |
| H | 147-467 mcg/g | | 248 mcg | |
| I | 694-1510 mcg/g | | 905 mcg | |

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**Table 3.1**

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**Table 4.1**
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