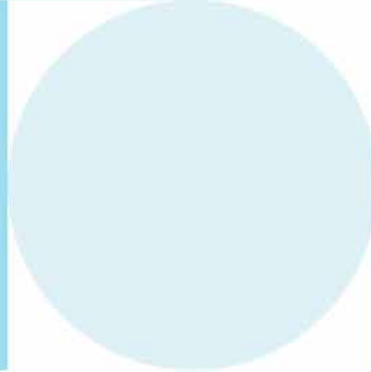
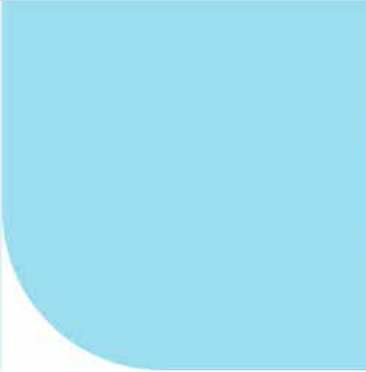
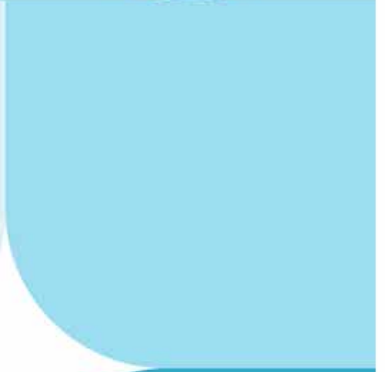
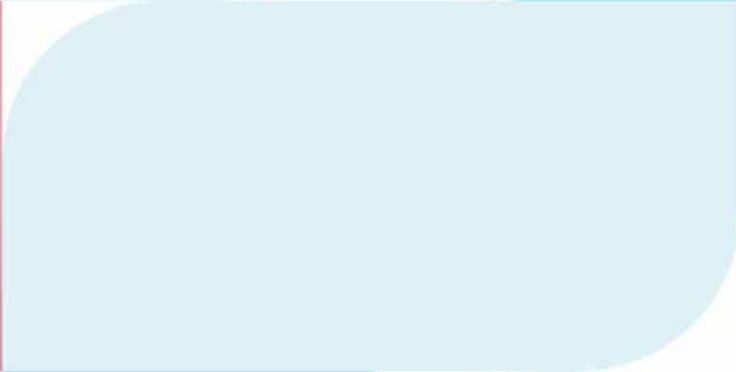




Test report



At-home test



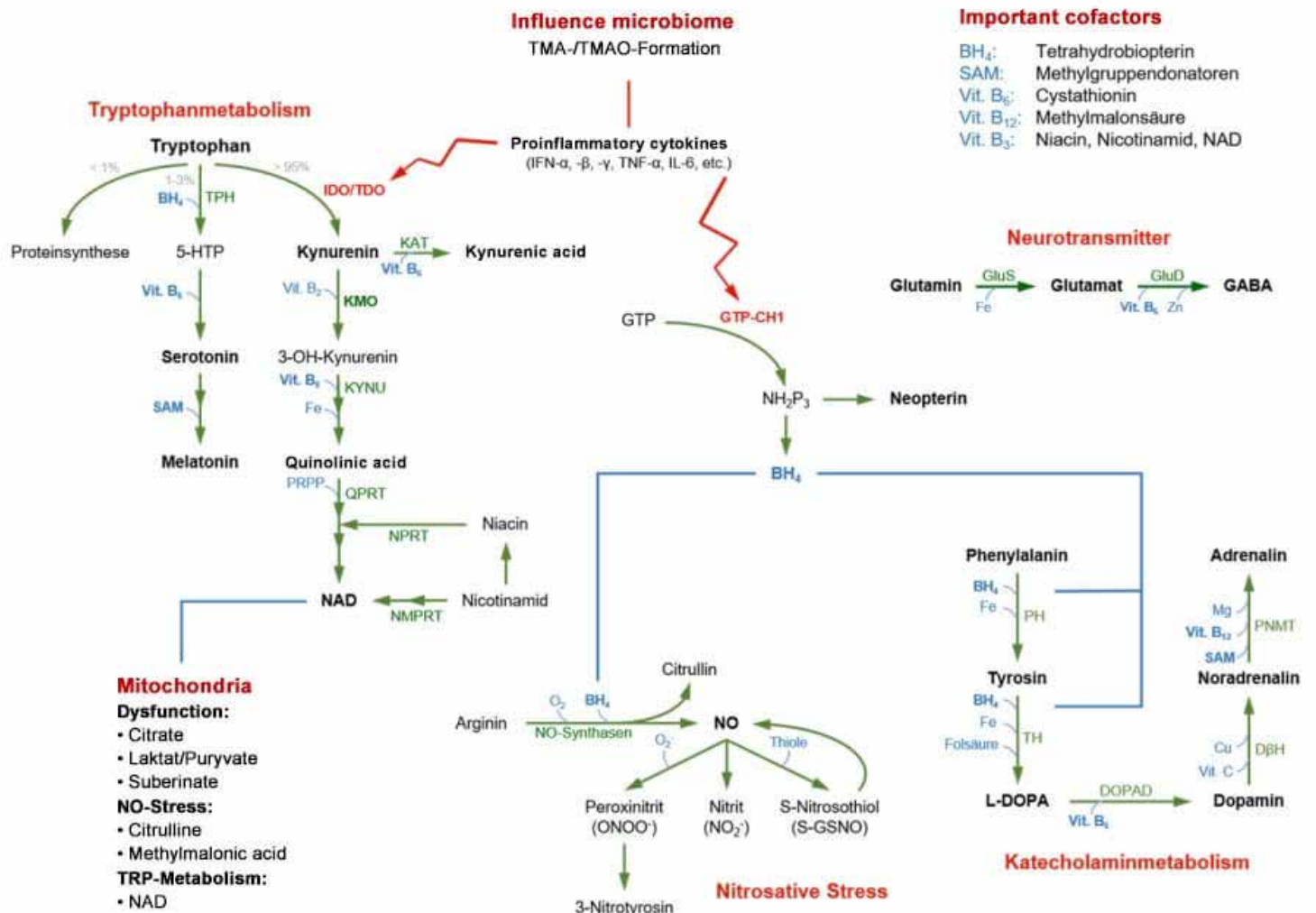
Neurotransmitters XL

 Lab test

 Urine

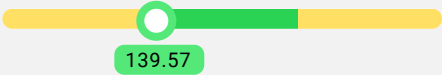
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Neurotransmitters - Tryptophan Metabolism

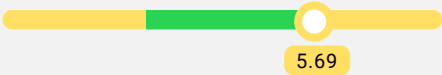


Neurotransmitters XL

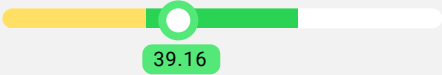
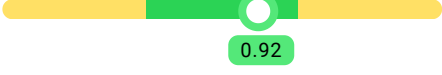
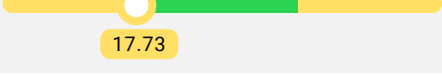
Neurotransmitters

Name	Your value	Reference value	Scale
Dopamine	 139.57 µg/g Crea	130 - 240 µg/g Crea	
Noradrenaline	 43.67 µg/g Crea	15 - 36 µg/g Crea	
Adrenaline	 5.82 µg/g Crea	2,0 - 5,5 µg/g Crea	
Serotonin	 145.84 µg/g Crea	80 - 190 µg/g Crea	

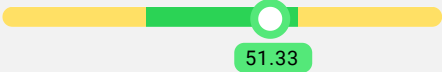
Additional Neurotransmitters

Name	Your value	Reference value	Scale
GABA	 5.69 µg/g Crea	1,5 - 5,0 µg/g Crea	
Glutamate	 21.83 µg/g Crea	8 - 25 µg/g Crea	

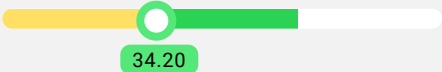
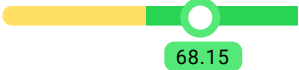
Kynurenine Pathway

Name	Your value	Reference value	Scale
Tryptophan	 39.16 µmol/g Crea	> 30 µmol/g Crea	
Kynurenine	 2.01 µmol/g Crea	1,0 - 2,7 µmol/g Crea	
Kynurenic acid	 4.46 µmol/g Crea	> 6,2 µmol/g Crea	
3-OH-Kynurenine	 0.92 µmol/g Crea	0,3 - 1,1 µmol/g Crea	
Quinolinic acid	 17.73 µmol/g Crea	18,5 - 32 µmol/g Crea	
NAD (Nicotinamide- Adenine- Dinucleotide)	 66.84 nmol/g Crea	> 42 nmol/g Crea	

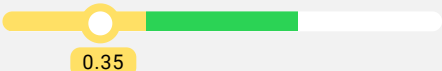
Enzyme activities

Name	Your value	Reference value	Scale
IDO-Activity	 51.33 Ratio	31 - 55 Ratio	
KMO-Activity	 3.97 Ratio	< 4,2 Ratio	

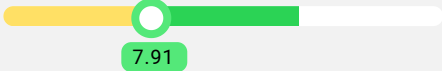
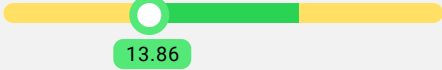
Catecholamine Metabolism

Name	Your value	Reference value	Scale
Phenylalanine	 34.20 µmol/g Crea	> 31 µmol/g Crea	
Tyrosine	 68.15 µmol/g Crea	> 42 µmol/g Crea	

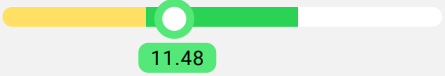
Important Cofactors

Name	Your value	Reference value	Scale
Cystathionine (Vitamin B6)	 12.45 µmol/l	< 25,0 µmol/l	
Methylmalonic acid (Vitamin B12)	 0.95 mg/g Crea	< 1,8 mg/g Crea	
Nicotinic acid	 0.35 µmol/g Crea	> 0,5 µmol/g Crea	
Nicotinamide	 3.50 µmol/g Crea	> 1,2 µmol/g Crea	
NAD (Nicotinamide-Adenine-Dinucleotide)	 66.84 nmol/g Crea	> 42 nmol/g Crea	

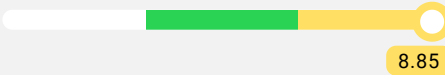
Methyl-Group Donors

Name	Your value	Reference value	Scale
S-Adenosylmethionine	 7.91 µmol/g Crea	> 7,5 µmol/g Crea	
Betaine	 45.69 µmol/g Crea	29 - 85 µmol/g Crea	
Choline	 13.86 µmol/g Crea	13 - 30 µmol/g Crea	

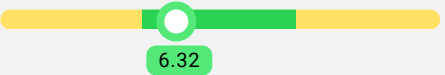
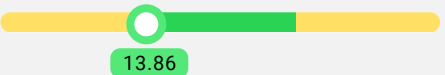
Methylation Capacity

Name	Your value	Reference value	Scale
SAM/SAH Ratio	 11.48 Ratio	> 9 Ratio	

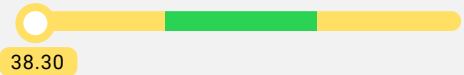
Nitrosative Stress

Name	Your value	Reference value	Scale
Citrulline	 8.85 $\mu\text{mol/g Crea}$	< 4 $\mu\text{mol/g Crea}$	
Citrate	 456.92 mg/g Crea	160 - 786 mg/g Crea	
Methylmalonic acid (Vitamin B12)	 0.95 mg/g Crea	< 1,8 mg/g Crea	

Mitochondrial Dysfunction

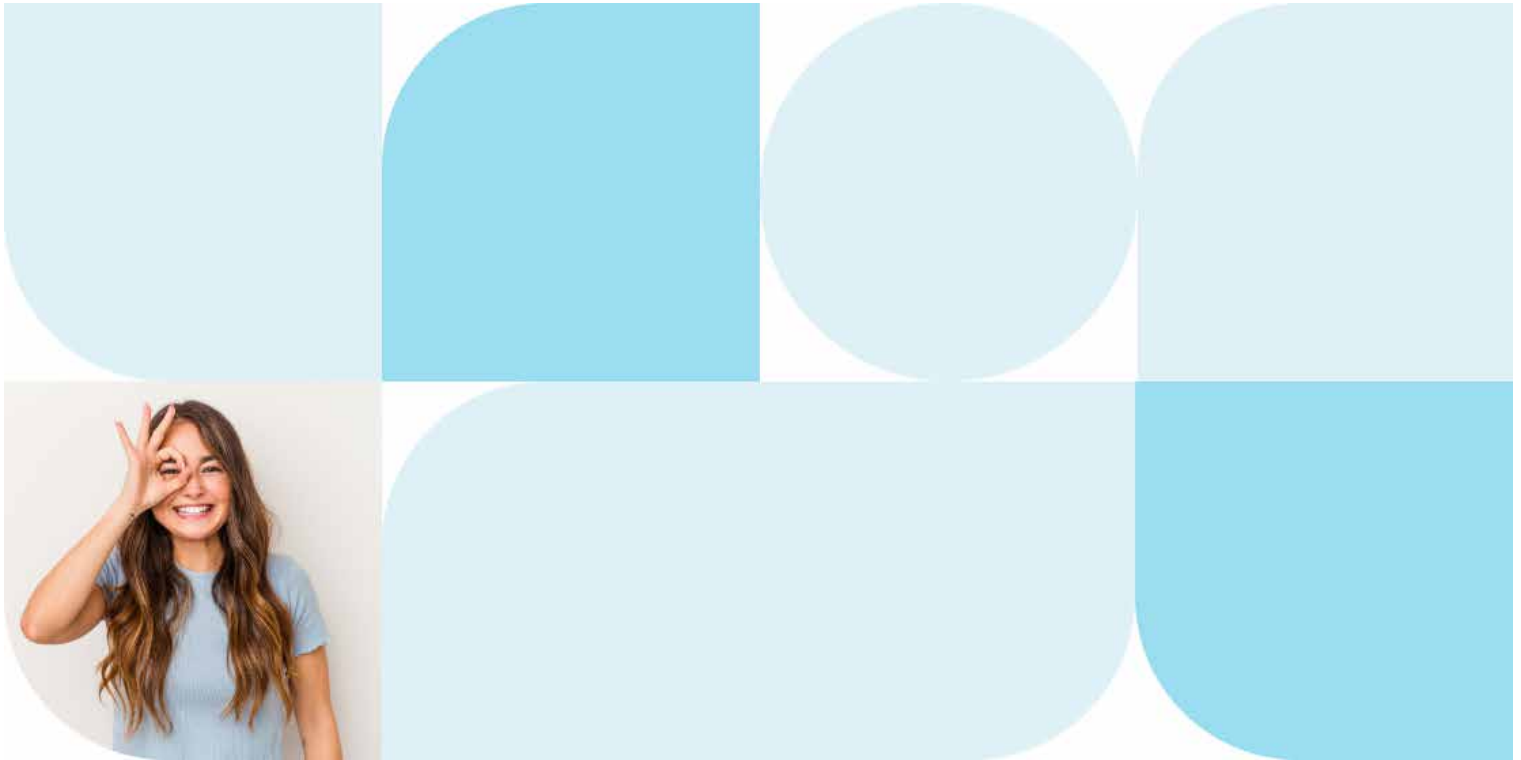
Name	Your value	Reference value	Scale
Lactate	 6.32 mg/g Crea	1,7 - 20,5 mg/g Crea	
Pyruvate	 5.69 mg/g Crea	< 5,4 mg/g Crea	
Suberic acid	 1.06 mg/g Crea	< 1,9 mg/g Crea	
Carnitine	 219.32 $\mu\text{mol/g Crea}$	11 - 90 $\mu\text{mol/g Crea}$	
Choline	 13.86 $\mu\text{mol/g Crea}$	13 - 30 $\mu\text{mol/g Crea}$	
Carnitine	 219.32 $\mu\text{mol/g Crea}$	11 - 90 $\mu\text{mol/g Crea}$	

Creatinine

Name	Your value	Reference value	Scale
Creatinine enzyme. (Urine)	 38.30 mg/dl	400 - 2780 mg/dl	

Serotonin Pathway

Name	Your value	Reference value	Scale
Serotonin	 145.84 µg/g Crea	80 - 190 µg/g Crea	 145.84



Extended Information

The requested analysis profile offers a comprehensive overview of the consequences of a variety of structural diseases. It contains more than 20 parameters. In addition to the complete mandible and complete analysis of the trigonum mandibulare, all important influencing factors that have a direct or indirect effect on the mandible process, including glenocranial movement.

What is being investigated?

GABA and glutamate are the most widely distributed neurotransmitters in the CNS. GABA is the primary inhibitory neurotransmitter and dampens the excitatory stress response. Glutamate is the primary excitatory neurotransmitter and should be regarded as an antagonist of GABA.

Also determined is a variety of other important cellular neurochemicals, which are made by neuroglia. Several neurotransmitters, stress and acute response important functions in the CNS and periphery. Last but not least, it is a precursor of the sleep hormone melatonin.

The ingestion of caffeine stress substances constitutes a healthy stress response in the body. In people who are under constant stress or already suffer from chronic affliction or burnout, this body's natural reaction is out of balance.

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Influencing factors such as chronic stress, severe pain, chronic inflammation or mitochondrial damage can also cause metabolic pathway shifts. This not only affects the formation of important stress substances, but also leads to the formation of metabolites that damage the organism, have a negative effect on mitochondria or promote oxidative stress. If the tryptophan metabolism is shifted due to chronic stress or other influencing factors mentioned above, serotonin synthesis drops significantly. Impairments of serotonin (5-HT) or even depression can result. Instead, tryptophan is increasingly converted into kynurenine and other neurotoxic, pro-inflammatory metabolites (e.g. quinoline acid). NAD formation decreases by 50 – 60 % and ATP formation in the mitochondria decreases.

If you really want to understand the influence of anxiety and stress, it is often not enough to determine neurotransmitters in the urine. You need to know about cofactor deficiencies and look at metabolite pathways as a whole. The requested profile offers this possibility. Based on the results, deficiencies can be compensated and metabolite shifts can be counteracted. Pure neurotransmitter examinations can at best only hint at causes or deficits.

Catecholamines

Dopamine, norepinephrine and epinephrine are physiologically active molecules known as catecholamines. Catecholamines act both as neurotransmitters and hormones due to the mechanisms of homeostasis through the autonomic nervous system. Elevated Adrenaline values can, for instance, occur with acute stress loads and often reflect a significant autonomic overload. When it comes to treatment, stress-reducing measures become an option, such as various stress-reduction techniques (e.g. autogenic training) or psychotherapy.

After being released, Adrenaline breaks down via catechol-O-methyltransferase (COMT) into norepinephrine, and then into vanillylmandelic acid (VMA) or metanephrine sulfate (MMS). To lower elevated Adrenaline values, attempts can be made to utilize cofactors of the enzymes that do the breaking down. These mainly include vitamin B12 (methylcobalamin) and magnesium (Mg), as well as B6 and vitamin B1. The question of whether there is nevertheless a deficiency of COMT or B6 and supplementation necessary, will be investigated in the following.

If Adrenaline values remain high in spite of the measures taken, and high blood pressure, physical stress, or hypoglycemia can be ruled out as potential causes, then there may also be a breakdown disorder from COMT gene variants, even if these mainly affect Dopamine and norepinephrine. COMT polymorphisms can be shown in the lab with gene tests.

In case of reduced norepinephrine levels, the cofactors vitamin C and copper can be supplemented to support the production of norepinephrine to dopamine. The cofactors can be supplemented, or a deficiency can be confirmed with a vitamin analysis and full blood metal analysis.

In case of elevated dopamine levels: Elevated dopamine levels can point to severe stress loads and often reflect a significant autonomic overload. When it comes to treatment, stress-reducing measures are an option, such as various stress-reduction techniques (e.g. autogenic training) or psychotherapy. Dopamine is metabolized by the enzymes COMT (catechol-O-methyltransferase) and MAO (monoamine oxidase), then after deamination to vanillylmandelic acid it is excreted in the urine. To lower elevated neurotransmitters, attempts can be made to utilize cofactors of the enzymes that do the breaking down. These mainly include vitamin B12 (methylcobalamin) and magnesium (Mg), as well as B6 and vitamin B1. If dopamine levels remain high in spite of measures taken, a breakdown disorder from existing gene polymorphisms should be ruled out. Primarily in the case of people with a COMT polymorphism MET/MET, the breakdown happens much more slowly than in those with the VAL, VAL, variant.

Serotonin

The levels of serotonin are, among other things, dependent on the presence of sufficient amounts of tryptophan in the diet. Many vitamins and minerals are also important for the formation of serotonin. Intake of too much alcohol, caffeine and amphetamine inhibit the production of serotonin in the body. Chronic stress, anxiety and other similar symptoms also prevent the brain from producing the right amount. Serotonin deficiency can affect physical and mental health and can sometimes be difficult to identify. When there is a great lack of serotonin, it can, for example, give rise to bad mood, stress and depression.

To increase the production of serotonin, one can increase the intake of tryptophan in the diet, in combination with foods rich in vitamin B6 and magnesium needed to convert tryptophan to serotonin. Keep in mind, however, that an increased intake of protein can inhibit the uptake of tryptophan. Exercise can also have a positive effect on serotonin production, especially endurance sports.

GABA

Gamma-aminobutyric acid (GABA, γ -aminobutyric acid) is the neurotransmitter with the largest physiological supply and the most important neurotransmitter in the central nervous system. Endogenous synthesis of GABA takes place with the help of the enzyme glutamate decarboxylase. It is synthesised from the excitatory neurotransmitter glutamate, with which GABA exists in equilibrium.

If there is a deficiency of gamma-aminobutyric acid, a therapeutic administration of GABA or a modulation of the GABA receptor becomes an option.

In case of therapeutic administration, usually 0.5 - 1 g of GABA is administered at night and if it is tolerated well the dose can be increased up to as much as 2 to 1 g daily. Phentermine and valerian preparations are suitable for receptor modulation. Valerian preparations should mainly be given in the evening, as they have a stronger sedative effect than phentermine preparations.

Glutamate

Glutamate (i.e. glutamic acid) is the most important excitatory neurotransmitter. The concentration of the neurotransmitters glutamate and GABA is approximately 1000 times higher than the concentrations of norepinephrine or dopamine. Among other things, glutamate is important for learning, memory and motor skills.

In case of low glutamate levels, glutamate can be given in a dose of 2 - 5 g per day, split up into multiple smaller doses. Intake should take place before mealtimes. If possible, drink to better absorption. Since elevated glutamate levels can have neurotoxic effects, it is important to monitor the levels.

If glutamate is elevated, excessive intake must not be ruled out (e.g. resulting from liver failure). Because GABA is the physiological antagonist of glutamate, it is necessary to strengthen the effect of GABA on the receptor. This is possible with herbal therapies such as phentermine or valerian preparations. Attention must be paid to adjustment of both herbal therapies.

Tryptophan metabolism

Tryptophan is precursor substance for serotonin synthesis. Pathological serotonin levels can usually be explained by changes in tryptophan metabolism. If the genetic inheritance not serotonin defective, understanding tryptophan metabolism becomes essential!

Tryptophan metabolism is responsible for much more than just serotonin synthesis. It has a major role in human health, regulating important neurochemical functions and broad stretches of the immune system. Stress-related changes in tryptophan metabolism can have significant consequences for the progression of diseases and for chances of recovery.

Due to the importance of tryptophan metabolism in the context of stress-related diseases, the precise state of relevant metabolites and enzymatic activities. This grants insights into the pathophysiology of subsequent damage and, often, measures for treatment.

If there is a deficiency of tryptophan, it is possible to administer tryptophan (500 - 1000 mg/day) if the kynurenine to tryptophan ratio is normal. In order to facilitate an effective conversion to 5-HTP and finally to serotonin, cofactors must be present in sufficient quantity. These mainly include folic acid and vitamin B6. Vitamin B2, too, should be available in sufficient quantity, as a deficiency can lead to activation of 5-kynurenine synthase. In the following, important cofactors will be investigated for their availability.

40% of KAT is formed with tryptophan from quinoline acid and 60% comes from the diet. Reduced KAT values in conjunction with normal quinoline acid levels could therefore suggest insufficient supply of KAT precursors (mainly or mainly from the diet). Not uncommonly, however, the enzymatic conversion (KAT) of quinoline acid into KAT is disrupted by environmental factors (e.g. alcoholism). In the following, we will attempt to clarify the effectiveness of the KAT deficiency by analyzing other main metabolites (see cofactors: vitamin B6).

In case of elevated enzymatic activity of IDO and KMO, it is then evidence that tryptophan is being converted at a higher rate to kynurenine and then into neuroactive metabolites (kynurenic acid). This happens at the expense of the neuroprotective and antioxidant kynurenine acid.

If the kynurenine to tryptophan ratio is elevated or kynurenine values are elevated, attempts should be made to reduce IDO activity with natural inhibitors. These include various secondary substances in therapeutically effective doses, such as curcuma, berberine, resveratrol or omega-3 fatty acids (primarily DHA). Curcuminoids increase the effect on IDO. For example, if curcuma is administered, it is always recommended to give DHA at the same time (at least 100 - 1000 mg/day).

More than anything else, curcuma has a significant effect on IDO activity. Curcuma preparations exhibit major differences with respect to their bioavailability, so doses must be adjusted accordingly. Studies show that berberine also has an inhibitory effect on IDO activity, in addition to the known modulatory effects on neurotransmitters in the brain, positive effects on glucose and lipid metabolism, and a strong protective effect on cancer cells.

Moderate endurance sports (> 2 mmol/lactate) too, have an impact on tryptophan metabolism. They cause increased enzymatic activity of kynurenine aminoglutarate transaminase (KAT). KAT converts L-kynurenine into kynurenine acid, which cannot pass the blood-brain barrier, but which does promote mitochondrial formation and has an antidepressant and neuroprotective effect. An increase in kynurenine acid can also be caused by a high diet.

Omega-3 fatty acids not only inhibit IDO activity, they also represent an important inhibitor of KMO activity. In this case, too, it is not as much EPA but rather DHA that effectively inhibits kynurenine monoamine. The recommended doses match the daily amounts discussed above. The riboflavin (B2) is also currently being discussed as a KMO inhibitor.

In case of tryptophan and serotonin deficiency: If the tryptophan deficiency comes from a long-term disorder, then causes must be addressed. The inflammatory activity that commonly accompanies such long-term can be reduced by administering probiotics and prebiotics. If acute stresses are present, they must be treated as well.

If there is a deficiency of tryptophan and serotonin, it is possible to administer tryptophan (200 – 1000 mg/day) if the kynurenine/tryptophan ratio is normal. In order to facilitate an effective conversion to 5-HTP and finally to serotonin, cofactors must be present in sufficient quantity. These mainly include folate acid and vitamin B6. Vitamin B6, too, should be available in sufficient quantity, as a deficiency can lead to activation of L-kynurenine synthase.

If serotonin is deficient, then 5-HTP can also be given. Treatment with L-tryptophan or 5-HTP is contraindicated if medications are being taken at the same time that affect the serotonin system (e.g. SSRIs, serotonin uptake inhibitors).

Ensure that tryptophan is not taken together with protein-rich meals, as branched chain amino acids in the blood-brain barrier can inhibit the absorption of tryptophan.

Phenylalanine and tyrosine

It is possible for Phenylethylamine to drop even if L-tyrosine is present in sufficient quantity. Brain conditions can induce significantly higher consumption of catecholamine. This causes an under-supply of the foundational building blocks phenylethylamine and tyrosine. Despite the phenylethylamine deficiency, catecholamine synthesis is not disrupted because tyrosine can also be used to form L-DOPA.

Availability of important cofactors in neurotransmitter and tryptophan metabolism

1. Vitamins

Vitamin B6 is an important cofactor in neurotransmitter synthesis and in tryptophan metabolism. If this vitamin is absent, L-DOPA cannot be converted into dopamine, and norepinephrine cannot be converted into Adrenaline. In tryptophan metabolism, the metabolic steps from 5-HTP to serotonin, from kynurenine to kynurenine acid and from 5-OH-kynurenine to quinoline acid all require vitamin B6 as a cofactor. The graph does not contain vitamin B6, but rather cystathionine – a functional marker for maintenance with B6-active vitamin B6.

Cystathionine: Even if there is no deficiency of B6-active vitamin B6, one should continuously ensure sufficient daily intake, which should not be below 3 mg.

Vitamin B12 is a cofactor for the methyltransferase responsible for turning norepinephrine into Adrenaline. Vitamin B12 is not measured directly either, but rather methylcobalamin acid, one of the most sensitive markers for a potential vitamin B12 deficiency.

Methylcobalamin acid shows if there is a deficiency of vitamin B12 as a cofactor.

Vitamin B6 affects the kynurenine metabolic pathway. Adequate supply of vitamin B6 leads to down-regulation of L-kynurenine synthase. However, the graph not only contains a direct measurement, but also one for serotonin and 5-HIAA. Niacin and niacinamide are released primarily through a peptic diet. The NAD formed from quinoline acid, on the other hand, comes from tryptophan metabolism and is crucial for sufficient 5-HT production in the mitochondria.

In case of normal glutathione acid, in combination with a depressed BH4 value and in conjunction with low levels for creatine acid and creatine itself. The low BH4 level can be explained by creatine and creatine itself, or possibly by a disrupted enzymatic function of glutathione S-transferase (GST), an enzyme which converts glutathione acid into BH4. The cause of these enzyme dysfunctions are often stresses from phthalates or parabens.

2. Tetrahydrobiopterin (BH4)

Tetrahydrobiopterin (BH4) is an important cofactor in human metabolism, even though formed by the body itself. Biopterin (BH1) in and of itself is not biologically active, but the tetrahydro form (tetrahydrobiopterin) can take on the function of cofactor in various reactions. The most important metabolic reactions where BH4 appears are cofactor and are: serotonin synthesis, and in the transformation of arginine into nitrogen monoxide and uric acid. If there are deficiencies of BH4, this will inhibit ongoing enzymatic processes.

Metabolic reactions where BH4 is involved:

- Conversion of phenylalanine to tyrosine (enzyme: phenylalanine hydroxylase)
- Transformation of tyrosine to L-DOPA (enzyme: tyrosine hydroxylase)
- Transformation of arginine to NO + uric acid (enzyme: NO synthase)
- Oxidation of norepinephrine to NE (enzyme: norepinephrine hydroxylase)

Caution: A lack of tetrahydrobiopterin can also accompany genetic diseases, most commonly an atypical phenylketonuria or an autosomal recessively inherited BH4 deficiency. These are severe neurological diseases, often with which manifest in early childhood and which get progressively worse if not treated. Such patients are extremely uncommon in day-to-day practice. Medical administration of tetrahydrobiopterin (sepiapterin) is required for this type of disease pattern with a genetic disorder of BH4 synthesis.

3. Methyl group donors

The BH4 / BH1 ratio shows the methylation capacity in the cells. Methylation capacities are very important for metabolism in the liver, genetic control, cell replication, as well as for the toxin removal system.

In case of lower values of BH4, betaine and choline suggest an inadequate supply of methyl group donors. A BH4 supplementation appears a reasonable (adult dose: 1000 – 1500 mg BH4, child dose 1 – 4 x 100 mg BH4).

If the BH4 / BH1 ratio is within normal levels, it suggests a sufficient methylation capacity in the cells. Methylation capacities are very important for metabolism in the liver, genetic control, cell replication, as well as for the toxin removal system.

If the BH4 / BH1 ratio is depressed, it can point to a reduced methylation capacity of the cells. 5-methyltetrahydrofolate and 5-methyltetrahydrohomocysteine are important elements in methyl group and homocysteine metabolism. Methylation capacities are very important for metabolism in the liver, genetic control, cell replication, and for the toxin removal system. Besides a deficiency of L-methionine, inadequate supply of vitamin B12, vitamin B9, folic acid, magnesium or zinc can negatively impact the methylation process (see: phenylalanine and methylmalonic acid).

The transfer of methyl groups from one molecule to another as part of a chemical reaction is known as methylation. Methylation reactions have a central role in many metabolic reactions. In this way, they affect epigenetic expression, immune reactivity, synthesis and control of neurotransmitters and hormones, the removal of toxins and protein metabolism. 5-methyltetrahydrofolate (BH4) represents one of the most important methyl group donors in detoxification and synthesis reactions. For example, the methyl groups of Adrenaline stem primarily from BH4. Other important methyl group donors are betaine and choline. If methyl donor groups are not present in sufficient quantity, this will affect Adrenaline synthesis. Levels will drop.

Influence of stress levels on mitochondria

NO stress / mitochondria

Chronic stress affects neuronal synthesis and neurochemical synthesis via a modification of cytoplasmic metabolism and DNA cycle, increasing degradation of neurochemicals from elevated consumption of neurochemicals under stress and a disrupted synthesis of new neurochemicals. A person will feel burnt out, tired and powerless.

But it is not only the neurotransmitter levels above which can cause these symptoms. Lack of energy can stem from mitochondrial issues. For example, lack of B12 causes activation or collapse of the tyrosine pathway. The result is an impairment of ATP synthesis. Inactive or inactive stress triggered by stress-induced chain inflammation can also cause mitochondrial dysfunction. This further causes mitochondrial ATP production to drop considerably as stress

Nitrosative stress

Citulline and citrulline make possible to draw information about nitrosative stress. A rise in citrulline is evidence for elevated NO synthesis from arginine, while rising citrulline levels point to mitochondrial dysfunction. Due to the effect of NO for uncoupling containing iron, iron in the citrate cycle is inhibited. This interferes with the subsequent conversion of citrate to isocitrate. Methylmalonic acid is a functional marker for a deficiency of vitamin B12, which, as an NO scavenger, is capable of breaking up the NO blockade on the paired enzyme.

If the levels of citrulline and citrate are within the normal, then it speaks against nitrosative stress.

Mitochondrial functional markers for orientation

The additional ergogenic aids lactate and pyruvate contained in the profile make it possible to detect mitochondrial disorders. If there are blockades, pyruvate from glucose can no longer enter the citrate cycle via decarboxylase. The result is an accumulation of pyruvate and possible increased conversion into lactate.

If dietary factors additionally result in a lack of L-carnitine, long-chain fatty acids from beta-oxidation can no longer be fed into mitochondrial energy production. L-carnitine provides the mitochondria with fuel by transporting fatty acids from the cytosol into the mitochondria. A deficiency of L-carnitine can therefore appear as an energy deficit. If beta-oxidation is disrupted, or if L-carnitine is lacking, fatty acids are alternatively broken down through beta-oxidation into medium-chain dicarboxylic acids, which are excreted in the urine. An example of one of these is suberic acid. Therefore, an increase in the marker in the profile points to the aforementioned disorders.

If the values for lactate and pyruvate are normal, then there is no evidence against stress-mediated disruptions in energy metabolism, as a normal suberic acid. Sufficient supply of L-carnitine is assumed.

In case of pathological lactate or pyruvate values, it points to stress-mediated disruptions in energy metabolism. Low L-carnitine levels cause the mitochondria to be undersupplied with long-chain fatty acids, which must then be converted to medium-chain dicarboxylic acids (e.g. suberic acid) via ω -oxidation as a compensatory measure.

Neopterin – Signs of immune activation

If Neopterin is within the normal range, then there are no signs of a Th1 activation of the immune system.

Elevated neopterin is evidence for an IFN- γ -mediated Th1 activation of the immune system. As a result of pro-inflammatory cytokines being released, the enzymes iNOS, IDO and IDO2/IDO can activate, thereby causing changes in numerous metabolic pathways.

Background: Elevated neopterin levels occur in diseases that accompany a cellular Th1 immune response. These include, for example, viral infections, infections by intracellular bacteria (e.g. tuberculosis), parasites, autoimmune diseases or COVID. Together with CRP a differential diagnosis is possible between bacterial and viral infections. While acute bacterial infections often only show slightly elevated neopterin levels, viral infections show extremely elevated values.

TMAO production as a cause of inflammations and arteriosclerosis

Regarding TMAO levels (trimethylamine N-oxide):

TMAO forms through oxidation of trimethylamine (TMA), which originates/bacteria make from choline, betaine or L-carnitine. Studies demonstrate that increased TMAO production promotes the occurrence of inflammations and can lead to an elevated risk of arteriosclerosis. However, betaine, choline and L-carnitine are not just precursor substances for the formation of TMAO, they also represent essential or semi-essential nutrients required to maintain important metabolic processes. Chronically low values can accompany nerve or muscle damage and should definitely be subject to further diagnostic investigation.

Low choline values can have negative effects. Many positive properties are attributed to choline and choline-containing compounds (e.g. lecithin). In the muscles they reinforce the muscles and lead to improved muscle membrane protection. They are an important component of cell membranes and are building block for the formation of acetylcholine. Additionally, they protect brain cells and possess an effect that calms and strengthens the nerves. Are there signs of body fat or elevated acute phase proteins in the blood (alpha-1-antitrypsin, CRP, procalcitonin)?

