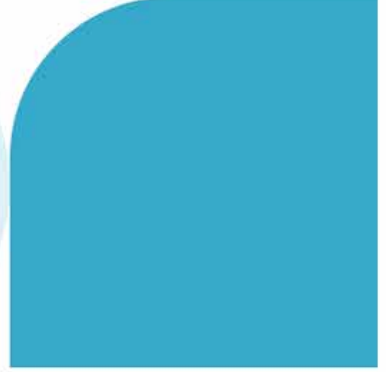
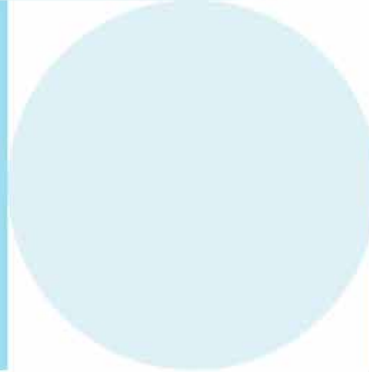
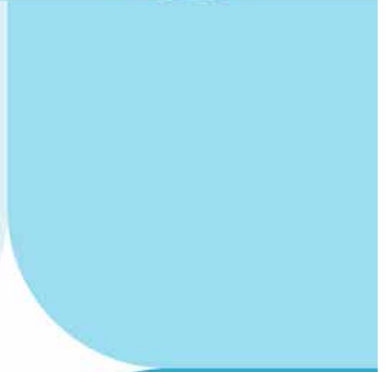
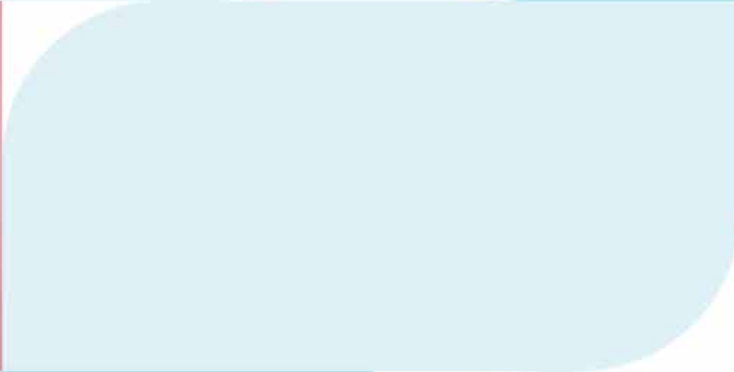




Test report



At-home test



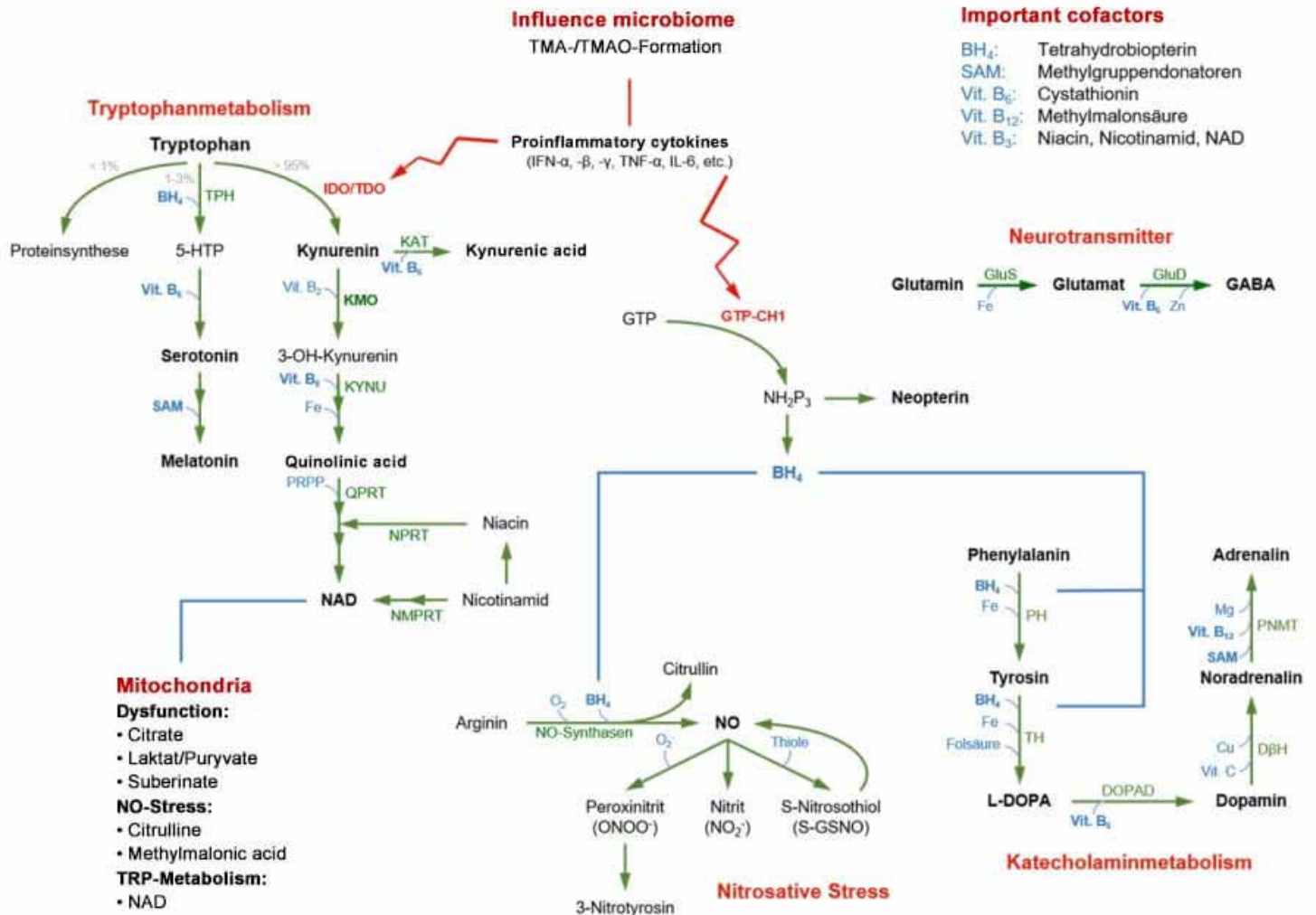
Neurotransmitters XL

Lab test

Urine

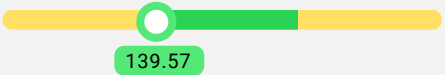


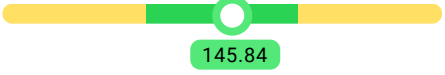
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Neurotransmitters - Tryptophan Metabolism

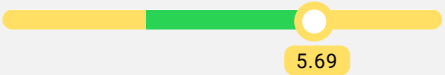



Neurotransmitters XL

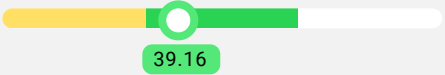



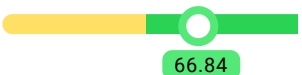
Neurotransmitters

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	


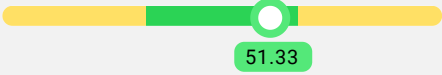


Additional Neurotransmitters

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	


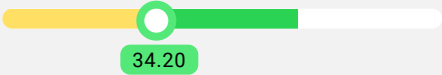

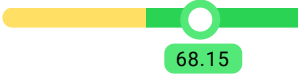
Kynurenine Pathway

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	




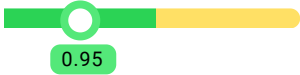

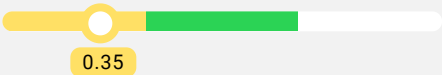




Enzyme activities

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


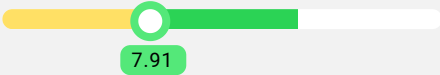



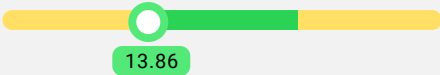
Catecholamine Metabolism

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


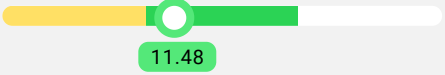
Important Cofactors

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


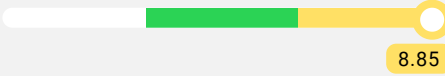




Methyl-Group Donors

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


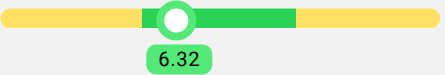










Methylation Capacity

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


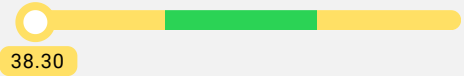
Nitrosative Stress

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	


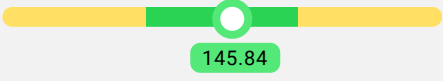
Mitochondrial Dysfunction

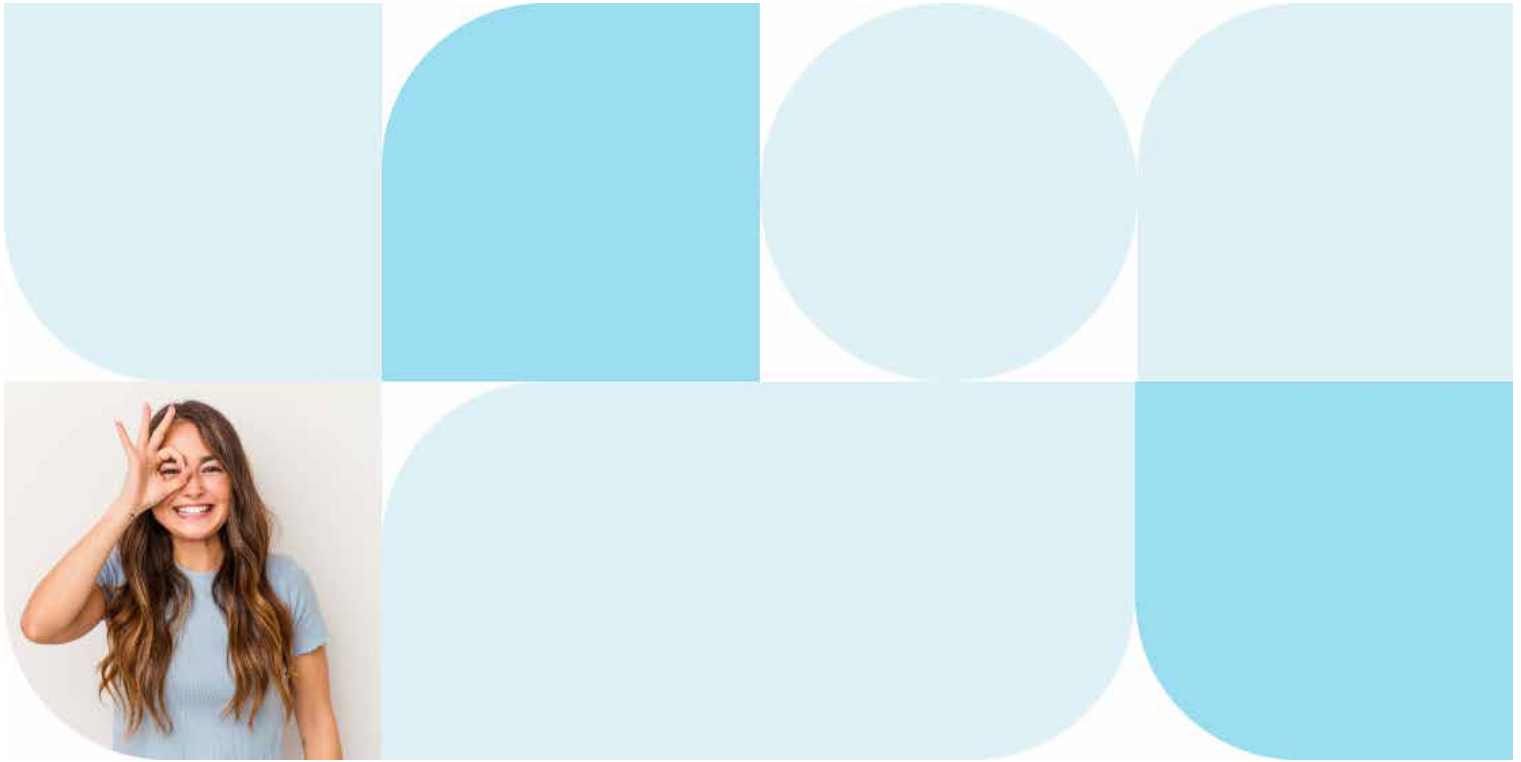
Name	Your value	Reference value	Scale
Lactate	 6.32 mg/g Crea	1,7 - 20,5 mg/g Crea	
Pyruvate	 5.69 mg/g Crea	< 5,4 mg/g Crea	
Suberic acid	 1.06 mg/g Crea	< 1,9 mg/g Crea	
Carnitine	 219.32 $\mu\text{mol/g Crea}$	11 - 90 $\mu\text{mol/g Crea}$	
Choline	 13.86 $\mu\text{mol/g Crea}$	13 - 30 $\mu\text{mol/g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



Extended Information

Neurotransmitters, tryptophan metabolism and relevant factors influencing acute or chronic stress

As we know, a wide variety of factors can cause stress. Acutely, pressures perform, performing or family problems as well as emotional factors transmute stress. During the length of job loss, loss of safety or negative changes in everyday working life. All the causes create and stress and activate a system's new metabolic functions to counteract the stress and increased release of cortisol and catecholamines.

Chronic stress leads to a sustained consumption of stress hormones and messenger substances (neurotransmitters) until the body has reached a condition that is often accompanied by pronounced psychological or physical symptoms of activation and requires extensive therapeutic measures. Depending on the situation, these include various relaxation techniques (e.g. autogenic training), psychotherapy and other molecular measures that help to regulate the stress of stress substances by administering various natural collectors. Therefore, these measurements when the doctor the results will be seen. The aim of the diagnostic carried out is to recognize stress-related changes at an early stage and to identify targeted countermeasures.

The requested analysis profile offers a comprehensive overview of the consequences of acute or chronic stress. It contains more than 20 parameters. In addition to the catecholamine and complete analysis of the tryptophan metabolism, all important influencing factors that have a direct or indirect effect on the metabolic processes being given are taken into account.

What is being investigated?

The neurotransmitters dopamine, noradrenaline and adrenaline, which have a stimulating effect and are formed in the nervous system from tyrosine and lysine, are investigated. First, L-DOPA is formed, which is further converted to dopamine. From this, noradrenaline and adrenaline are formed. Catecholamines have a stimulatory effect on the output and blood pressure. They affect parasympathetic and sympathetic nervous system, regulate effect on cellular enzyme activities.

5-HT and glutamate are also called 5-HTA (serotonin) and are important inhibitory neurotransmitters in the CNS. It counteracts the excitatory catecholamines and dampens the stress-related mediated stress response. 5-HT is formed and is formed from glutamate, which is a stimulatory neurotransmitter and should be kept below an optimum of 5-HTA.

Also determined is serotonin, another important inhibitory neurotransmitter, which is formed from tryptophan. Serotonin counteracts stress and exerts numerous important functions in the CNS and periphery. Last but not least, it is a precursor of the sleep hormone melatonin.

The regulation of all these stress substances constitutes a healthy stress response in the body. In people who are under constant stress or already suffer from chronic activation or burnout, the body's natural regulatory system of balance.

In order for neurotransmitters to be formed, a constant supply of amino acids is necessary. Numerous cofactors are required to ensure that enzymatic steps take place effectively. For an amino synthesis, for example, there are 8 vitamins (vitamin B1 and folic acid) and tetrahydropterin (BH4). In addition to BH4, B12, folic acid and vitamin C, methyl groups (from BH4) are needed for catecholamine synthesis. If important cofactors are missing, synthesis is disturbed, metabolic pathways are blocked and this has consequences.

...forming factors such as diet and stress, severe pain, chronic inflammation or mitochondrial dysfunction can also cause metabolic perturbations. This not only affects the formation of important stress substrates, but also leads to the formation of metabolites that damage the organism, have a negative effect on mitochondrial or promote oxidative stress. If the synthesis of melatonin is affected due to diet and stress or other influencing factors mentioned above, melatonin synthesis drops significantly. Impairments of serotonin/5-HT or even dopamine can result. Instead, norepinephrine is increasingly converted into L-homovaniline and other neurotransmitters, primary metabolites (e.g. serotonin and 5-HT) formation decreases by 50 - 80 % and 5-HT formation in the mitochondria decreases.

If you really want to understand the effects of anxiety and stress, it is often not enough to determine neurotransmitters in the urine. You need to know about cellular dysfunction and full or metabolic perturbations as a whole. The requested profile offers the possibility. Based on the results, dysfunction can be compensated and metabolites can be restructured. For neurotransmitter assessment, not just only but also stress or diet.

Catecholamines

Dopamine, norepinephrine and epinephrine are physiologically active molecules from catecholamine class. Catecholamines are both neurotransmitters and hormones due to the mechanism of homeostasis through the autonomic nervous system. Elevated adrenaline release for instance occur with acute stress levels and often a significant autonomic overload. When it comes to treatment, stress-reducing measures (breathwork, meditation, active stress-reduction techniques (e.g. yoga, Tai Chi) or psychotherapy).

After being released, adrenaline breaks down via catechol O-methyltransferase (COMT) into norepinephrine, and then into vanillylmandelic acid and its isomer normetanephrine (NMN). To lower elevated adrenaline values, attempts can be made to utilize cofactors of the enzymes that do the breaking down. These mainly include vitamin B6 (pyridoxine) and magnesium (Mg), as well as B12 and vitamin B1. The question of whether there is a metabolic dysfunction of COMT or MAO and supplementation necessary, will be investigated in the following.

If adrenaline values remain high in spite of the active stress and high blood pressure, physical strain or hyperglycemia can be ruled out as a potential cause, then there may also be a breakdown disorder from COMT gene variants, even if these mainly affect dopamine and norepinephrine. COMT polymorphism is broken in the 45 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 as a dietary or can be combined with a clinical analysis and full blood mineral analysis.

In case of elevated dopamine levels, elevated dopamine levels can point to severe stress levels and often a significant autonomic overload. When it comes to treatment, stress-reducing measures (breathwork, meditation, active stress-reduction techniques (e.g. yoga, Tai Chi) or psychotherapy). Dopamine is metabolized by the enzymes COMT (catechol O-methyltransferase) and MAO (monoamine oxidase), that after deamination to vanillylmandelic acid 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 B6 (pyridoxine) and magnesium (Mg), as well as B12 and vitamin B1. If dopamine levels remain high in spite of measures when a breakdown disorder from starting gene polymorphism should be ruled out. Primarily in the case of people with a COMT polymorphism MAO-A1, the breakdown happens much more slowly than in those with the MAO-A1A variant.

Serotonin

The levels of serotonin are, among other things, dependent on the presence of **tryptophan** in the diet. When **essential** minerals are also important for the formation of serotonin. Intake of too much alcohol, caffeine and amphetamines 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, there, for example, grows to feel mood, stress and depression.

To increase the production of serotonin, you 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. Sleep is also, however, that an increased intake of protein can inhibit the uptake of **tryptophan**. Serotonin also has a positive effect on serotonin production, especially under **exercise**.

GABA

Gamma-aminobutyric acid (GABA), **gamma** amino butyric acid is the neurotransmitter with the largest physiological supply and the most important one in the central nervous system. Endogenous synthesis of GABA takes place with the help of **transaminase** enzymes. GABA is synthesized from the excitatory neurotransmitter glutamate, with which GABA acts 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 stepped up to as much as 3 g daily. **Transaminase** and **vitamin** preparations are suitable for receptor modulation. **Vitamin** preparations should mostly be given in the evening, as they have a stronger sedative effect than **transaminase** preparations.

Glutamate

Excitatory glutamate 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 0 - 3 g per day split up into multiple smaller doses. Intake should **occur** before mealtimes. If possible, **transaminase** receptor. **Transaminase** glutamate levels can have neurotoxic effects. It is important to monitor the levels.

If glutamate-induced sensitization has **not** been ruled out (e.g. resulting from **over** exercise), because GABA is the physiological antagonist of glutamate, it is necessary to strengthen the effect of GABA at the receptors. This is possible with herbal therapies such as **transaminase** or **vitamin** preparations. Attention must be paid to **avoid** taking of both herbal therapies.

Tryptophan metabolism

Tryptophan is precursor substrate for serotonin synthesis. Psychologic/serotonin disorders usually be explained by changes in tryptophan metabolism. If the goal is to believe our serotonin levels, understanding tryptophan metabolism becomes essential.

Tryptophan metabolism is responsible for much more than just serotonin synthesis. It has a central role in human health regulating important neurochemical functions and broad aspects of the immune system. Stress-related changes in tryptophan metabolism can have significant consequences for the progression of disease and for outcomes of therapy.

Due to the importance of tryptophan metabolism in the context of stress-related illnesses, the pathologic alterations of metabolism and enzymatic activities. The genetic insights into the pathophysiology of consequent damage and effect, measure for treatment.

If there are deficiencies of tryptophan, it is possible to administer tryptophan (500 - 1000 mg/day) if the tryptophan tryptophanase is normal, in order to facilitate an effective concentration of 5-HTP and finally to serotonin. cofactors must be present in sufficient quantity. These include folic acid and vitamin B6. Vitamin B6 use, should be available in sufficient quantity, were deficiencies can lead to activation of α -hydroxytryptophan synthesis. In the following, important cofactors will be investigated for their availability.

60% of 5-HT is formed with tryptophan from guanine acid and 40% comes from the diet. Reduced 5-HT release in conjunction with normal guanine acid levels could therefore suggest insufficient supply of 5-HT precursor (mainly in mammals) from the diet. But unfortunately because the enzymatic conversion of 5-HTP to 5-HT is disrupted by environmental factors (e.g. alcoholism) in the following, we will attempt to clarify the circumstances of the 5-HT deficiency by analyzing other neurotransmitter metabolism (see cofactors vitamin B6).

In case of elevated enzymatic activity of 5-HT and 5-HT, it is then evidence that tryptophan is being converted at a higher rate to serotonin and then into neurotoxic pro-serotonin metabolites (serotonin acid). This happens in the presence of the neuroprotective and antioxidant tryptophanase.

If the tryptophan to tryptophan rate is elevated or tryptophan release is elevated, attempts should be made to reduce 5-HT activity with natural inhibitors. These include various secondary substrates in the particularly effective doses, such as curcumin, berberine, resveratrol or omega 3 fatty acids (primarily DHA). Combinations increase the effect on 5-HT. For example, if curcumin is administered, it is always recommended to give DHA at the same time (at least 1000 - 1000 mg/day).

More than anything else, curcumin has a significant effect on 5-HT activity. Curcumin preparations exhibit wide differences with respect to their bioavailability, so doses must be adjusted accordingly. Studies show that berberine has an inhibitory effect on 5-HT activity, in addition to the known inhibitory effects on neurotransmitters in the brain, positive effects on glucose and lipid metabolism, and a strong anti-inflammatory effect on cancer cells.

Medicinal herbs (magnolia) (2 mmol/day) can have an impact on tryptophan metabolism. The usual increased enzymatic activity of tryptophanase (mainly in mammals) 5-HT converts tryptophan into 5-HTP and, which cannot pass the blood-brain barrier, but which does promote neurochemical formation and has an antioxidant and neuroprotective effect. An increase in tryptophan activity can be caused by a high diet.

Omega 3 fatty acids not only inhibit 5-HT activity, they also represent an important inhibitor of 5-HT activity. In the case, too, it is recommended DHA but not the DHA that effectively inhibits tryptophan monoamine. The recommended dose must be 2-3g, amounts discussed above. Fructose (Sorbitol) is also currently being discussed as a 5-HT inhibitor.

In case of tryptophan and serotonin deficiency, if the tryptophan deficiency comes from absorption issues, then intake must be increased. The enzymatic activity that converts serotonin into tryptophan can be affected by administering probiotics and prebiotics. If mental issues are present, they must be treated as well.

If there are deficiencies of tryptophan and serotonin, it is possible to administer tryptophan (500 - 1000 mg/day) if the symptomatic tryptophan intake is normal. In order to facilitate an effective conversion to 5-HTP and finally to serotonin, cofactors must be present in a sufficient quantity. These mainly include folate and/or vitamin B12. Vitamin B12, too, should be available in a sufficient quantity as a deficiency can lead to reduction of L-tryptophan synthesis.

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

Enzymes that tryptophan is not taken together with protein-rich meals, as described above, can reduce the blood level. Some can affect the absorption of tryptophan.

Phenylalanine and tyrosine

It is possible for Phenylalanine to be used as L-tyrosine is present in a sufficient quantity. Stress conditions can induce significantly higher consumption of catecholamines. This occurs as under supply of the foundational building blocks phenylalanine and tyrosine. Despite the phenylalanine deficiency, catecholamine synthesis is not affected because tyrosine can also be used to form L-5-HTP.

Availability of important cofactors in neurotransmitter and tryptophan metabolism

1. Vitamins

Vitamin B12 is an important cofactor in neurotransmitter synthesis and in tryptophan metabolism. If this vitamin is absent, L-5-HTP cannot be converted into 5-HT, and norepinephrine cannot be transferred into Adrenaline, in tryptophan metabolism, the metabolic steps from 5-HTP to serotonin, from serotonin to norepinephrine and from D-5HT to norepinephrine to epinephrine and all require vitamin B12 as a cofactor. The profile does not contain vitamin B12, but rather cyanocobalamin - a functional marker for deficiencies with B12.

Cyanocobalamin: Even if there is no deficiency of B12, one should continuously ensure a sufficient daily intake which should not be below 2 mg.

Vitamin B11 is a cofactor for the methyltransferases responsible for turning norepinephrine into Adrenaline. Vitamin B11 is not measured directly either, but rather methylcobalamin and one of the most sensitive markers for a possible vitamin B11 deficiency.

Methylcobalamin will show if there is a deficiency of vitamin B11 as a cofactor.

Vitamin B6 affects the tyrosine metabolic pathway. Adequate supply of this leads to a downregulation of L-tyrosine synthesis. However, the profile not only contains such measurement, but also one for methylenetetrahydrofolate. Methyl and methylenetetrahydrofolate are released primarily through a genetic defect. The folate formed from quinine and, on the other hand, comes from tryptophan metabolism and is crucial for sufficient 5-HT production in the mitochondria.

In case of normal quinoline and in combination with a depressed total value and in conjunction with low levels for dopamine and/or norepinephrine. The low total level can be explained by: none and/or norepinephrine or possibly by a dysfunction of quinoline and phosphate transferase (TPH2), an enzyme which converts quinoline and into total. The cause of these enzyme dysfunction are often stresses from physical or psychiatric.

2. Tetrahydrobiopterin (BH4)

Tetrahydrobiopterin (BH4) is an important cofactor in human metabolism. It can be formed by the body itself. BH4 (BH4) is not of itself a neurotransmitter, 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 cofactor synthesis and in the re-synthesis of arginine, norepinephrine and dopamine. If there are deficiencies of BH4, there will be an ongoing metabolic process.

Metabolic reactions where BH4 is involved:

- Conversion of phenylalanine to tyrosine (enzyme: phenylalanine hydroxylase)
- Transformation of tryptophan to 5-HT (enzyme: tryptophan hydroxylase)
- Transformation of arginine to nitric oxide (enzyme: NO synthase)
- Oxidation of norepinephrine to NE (enzyme: norepinephrine hydroxylase)

Caution: A lack of tetrahydrobiopterin is an inherited autosomal recessive disease, most commonly an isolated phenylketonuria or an autosomal recessively inherited BH4 deficiency. These are serious neurological diseases which affect mostly childhood and which get progressively worse if not treated. Both patients are extremely dependent in day to day practice. Medical supervision of tetrahydrobiopterin (sepiapterin) is essential for the type of disease pattern with a genetic disorder of BH4 synthesis.

2.1 Methyl group donors

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

In case of lower values of BH4 / BH4 ratio and/or values suggest an inadequate supply of methyl group donors. A BH4 supplementer appears reasonable (adult dose: 500 - 1000 mg BH4, child dose: 2 - 4 x 250 mg BH4).

If the BH4 / BH4 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 of cell replication, as well as for the neurotransmitter system.

If the BH4 / BH4 ratio is decreased, it can point to a reduced methylation capacity of the cells. S-adenosylmethionine and S-adenosylhomocysteine are important elements in methyl group and homocysteine metabolism. Methylation capacities are very important for metabolism in the liver, genetic control of cell replication, and for the neurotransmitter system. Besides a deficiency of S-adenosylmethionine, inadequate supply of vitamin B12, vitamin B9, folic acid, magnesium or zinc can negatively impact the methylation process (see S-adenosylmethionine and methylation here and).

The transfer of methyl groups from one molecule to another as part of a chemical reaction is known as methylation. Methylation reactions have control points in many metabolic reactions. In this way, they affect gene expression, neurotransmitter synthesis and control of neurotransmitters and hormones, the removal of toxins and protein metabolism. S-adenosylmethionine (SAM) represents one of the most important methyl group donors. SAM synthesis and synthesis reactions. For example, the methyl groups of adrenaline stem primarily from SAM. Other important methyl group donors are betaine and choline. If methyl donor groups are not present in sufficient quantity, the synthesis of adrenaline synthesis levels will drop.

Influence of stress levels on mitochondria

NO stress / mitochondria

Research on the acute stress effect on protein synthesis and especially on synthesis via a modification of nitrogen metabolism and DNA code. Increasing levels of neurotransmitters lead to a decreased concentration of neurotransmitters under stress and a disrupted synthesis of new neurotransmitters. A person with high cortisol levels and anxiety.

But it is not only the neurotransmitters themselves which can cause these symptoms. Lack of energy can stem from mitochondrial issues. For example a lack of B12 due to activation or collapse of the folate cycle pathway. The result is an impairment of ATP synthesis. Indicators of oxidative stress, if paired to a stress-induced vitamin B12 deficiency, can also cause mitochondrial dysfunction. The further decreased mitochondrial ATP production is, the more severely it is affected.

Nitrosative stress

Clinical studies indicate possible direct influences about nitrosative stress. Some metabolic pathways for decreased ATP synthesis from arginine, while being shown to be related to mitochondrial dysfunction. During the synthesis of NO by nitric oxide synthase, arginine in the cytosol is oxidized. The reaction with the subsequent conversion of nitric oxide synthase. Methylenetetrahydrofolate is a functional marker for a deficiency of vitamin B12 which as an ATP source is capable of breaking up the NO blocks on the bound enzymes.

If the levels of nitric oxide and arginine within the normal, then it speaks against nitrosative stress.

Mitochondrial functional markers for orientation

The additional large amount of lactate and pyruvate contained in the plasma is not possible to detect mitochondrial disorders. If there are metabolic problems from glucose not so large under the stress cycle is likely. The result is an accumulation of pyruvate and possible metabolic conversion into lactate.

If there is a further additional 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 fatty acids by transporting fatty acids from the cytosol into the mitochondria. A deficiency of L-carnitine therefore causes fatty acids to be oxidized in the cytosol instead of in the mitochondria. If beta oxidation is targeted, or if L-carnitine is lacking, fatty acids are often oxidized to other down through beta oxidation into medium chain fatty acids, which are excreted in the urine. An example of one of these is acetoacetic acid. Therefore, an increase in the excretion of the plasma points to a mitochondrial disorder.

If the values for lactate and pyruvate are normal, then there is no direct evidence against stress-mediated disruption in energy metabolism as a metabolic disorder. In a normal supply of L-carnitine is normal.

In case of pathological behavior or genetic issues, it points to stress-mediated disruption in energy metabolism. Lack of L-carnitine leads to the mitochondria to be under-supplied with long chain fatty acids, which must then be converted to medium chain fatty acids (e.g. acetoacetic acid) via an oxidation via compensatory measures.

Neopterin – Signs of immune activation

If Neopterin is within the normal range, that there are no signs of a T_H1 activation of the immune system.

Elevated Neopterin is evidence for an IFN- γ mediated T_H1 activation of the immune system. As a result of pro-inflammatory cytokines being released, the enzymes iNOS, HMOX1 and 5-HT (CMT) are activated thereby causing changes in numerous metabolic pathways.

Background: Elevated Neopterin levels occur in diseases that accompany a cellular T_H1 immune response. These include, for example, viral infections, infections by intracellular bacteria (e.g. tuberculosis), genetic autoimmune diseases, or CVD. Together with IFN- γ 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 (3-methylamine N-oxide):

TMAO forms through oxidation of trimethylamine (TMA), which originates from the metabolism of choline, betaine and L-carnitine. Studies demonstrate that increased TMAO production promotes the occurrence of inflammations and can lead to an elevated risk of atherosclerosis. However, betaine, choline and L-carnitine are not just precursors, but also essential for the formation of TMA. They also represent essential or semi-essential nutrients required to maintain important metabolic processes. Especially for children, betaine, choline, carnitine, muscle damage and should definitely be subject to further diagnostic investigations.

Low choline values can have negative effects. When positive properties are attributed to choline and choline-containing compounds (e.g. lecithin) in the membrane they confer on the nucleus and lead to improved nuclear membrane protection. They are an important component of cell membranes and are building blocks for the formation of sphingolipids. Additionally, they protect from cell death and possess an effect that reduces and strengthens the nervous system (e.g. by acting on alpha-1 antitrypsin, acetylcholinesterase).

