THYMVITAL®
Dietary supplement with thymus extract, zinc and selenium

Thymus gland:
important organ of the immune system

Selenium and zinc:
contribute to protect cells from oxidative stress

Selenium and zinc:
contribute to normal function of the immune system

Zinc:
contributes to normal cognitive function
THYMVITAL®
Pharmaceutical production at the highest level

The biosyn® brand stands for top-tier production know-how and superlative quality

The pellet coating protects the sensitive thymus peptides from the stomach’s acidic environment

Only one capsule a day thanks to optimal pharmaceutical formulation

The capsules are very easy to open, so you can also sprinkle the pellets into your food
Why THYMVITAL®?

THYMVITAL® at a glance

- Unique galenic
- High-quality ingredients
- Only one capsule per day – and thus less expensive for the daily requirement than the products of the competition
- Optimal dosing

Unique Galenic

Providing the valuable ingredients where they need to be

The capsules contain pellets individually coated by a special process that makes them resistant to gastric juices but soluble in the small intestines. This ensures that the peptides in the thymus extract are not destroyed in the stomach, but absorbed unchanged in the small intestines and made available to the body.

Capsules for oral intake treated with this process show the same effect as a systemic application.[1] The manufacturing process simultaneously ensures that the capsules can be opened and the pellets ingested directly as well.
High quality ingredients: Sodium selenite Pentahydrate

Why the selenium form sodium selenite?

In contrast to other selenium forms, sodium selenite is completely absorbed by passive diffusion, reduced to the principal intermediate product, hydrogen selenide, and converted into the amino acid selenocysteine. However, sodium selenate is actively taken up by means of a transporter. A portion of this is directly eliminated with the urine. The rest must be converted by means of reduction to sodium selenite before it can be used by the body.[2]

The selenium form selenomethionine is still less efficient. Although selenomethionine is the selenium form that is primarily taken up with food, the absorption is considerably more complicated. Moreover, selenomethionine is not recognized by the body as a selenium form, and is instead confused with methionine and non-specifically incorporated into sulfurous amino acids. For this reason, only a portion of selenomethionine is available to the body for the production of important selenoproteins. In contrast to the remaining selenium metabolism, this process is not regulated.[2] The release of selenomethionine from sulfurous proteins does not proceed by demand, but rather depends on the methionine metabolism. Selenomethionine bound in proteins that are not recycled is not mobilized to fulfill the demand for selenium, but rather can simply accumulate.

Why sodium selenite in the form of sodium selenite-pentahydrate?

In contrast to sodium selenite, sodium selenite pentahydrate contains five water molecules. Sodium selenite pentahydrate is nevertheless solid matter. The therapeutic action of both molecules is identical. As the first and presumably still the only company in the world, biosyn Arzneimittel GmbH has been able to produce the active ingredient sodium selenite pentahydrate in the internationally prescribed GMP quality since 2009, thanks to a production process expressly developed and patented by biosyn. The quality of sodium selenite pentahydrate in THYMVITAL® corresponds to injectable liquid drug products.
High quality ingredients: Thymus extract

Thymus extract from calves consists of various constituents: thymus peptides, phospholipids, glycolipids and neutral peptides. The biological activity of the thymus extracts is based on the thymus peptides they contain.\[^3\] Only glands of calves from at least four-year BSE-free German stock younger than six months of age are employed in the manufacture of the thymus extract. The age of the calves plays a great role, since the amount of biologically-active thymus peptides is significantly reduced after six months.\[^3\]

Only one capsule a day means very little effort

Thanks to optimal capsule dosing and the special galenics, only one capsule a day is necessary to provide daily support of the immune system. Taken unchewed with some liquid approximately 30 minutes before breakfast provides the body with what it needs for the entire day.

Also under cost aspects, everything speaks for THYMVITAL®. Because the cheaper products of the competition require the consumption of several capsules per day.
A dose of 300 mg thymus extract per day is oriented on studies about applied dosages. These studies have demonstrated a positive effect of thymus extract at a dose of 100–300 mg thymus extract at a time.[1, 4]

The latest studies confirm that the supply of selenium for the population in Germany and Europe is suboptimal for good health. The selenium requirement rises under increased oxidative stress and high requirements on the immune system. A daily supplementation with 100 µg selenium ensures a sufficient supply to protect from oxidative stress and maintain the normal function of the immune system.

The activation of the thymus peptide – thymulin – is dependent on the availability of zinc. Zinc is bound one to one to thymulin. Already small changes in zinc intake or zinc availability influence the activity of thymulin. Zinc supplementation, apart from the direct influence of zinc via zinc proteins on the immune system, cognitive capabilities, and oxidative stress, therefore ensures that the thymus peptides are biologically activated and can have a positive effect on the body.

THYMVITAL® is an optimally tailored nutritional food supplement for the targeted protection from oxidative stress as well as to ensure the normal function of the immune system and thyroid gland along with cognitive functions.
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Thymus – an important component of the immune system

Function of the thymus

The thymus lies behind the sternum and assumes a central role in our immune system. This two-lobed organ consists of an exterior capsule, a densely populated cortex with unripe thymocytes, and an interior medulla (Fig. 1). The maturation and selection of the thymocytes takes place in the interior medulla. This process is called thymopoiesis and establishes an extensive repertory of T-lymphocytes that are directed against specific antigens.

What impairs the function of the thymus?

Limited thymus function leads to low T-cell production and consequently to greater susceptibility for infections. Two factors influence the function of the thymus: age and stress, whereby the term "stress" designates both physiological conditions as well as infections and treatments. A distinction is made between stress and age-induced loss of thymus function.

Stress reduces the thymus size through the acute loss of cortical thymocytes and reduces the production of naive T-cells. Once the stress factor has been removed, there is a spontaneous recovery phase. Old age leads to a gradual enlargement of the perivascular space and a reduction of tissue important for thymopoiesis.
Structure of the thymus

Fig. 1
Thymus in old age

The age-dependent regression of the thymus is called thymic involution. This already begins very early in life, between the 10th and 15th year of age, and progresses up to the development of the thymic fat tissue body (Fig. 2). The reduction of the thymus tissue mass as well as its cell content and the loss of the tissue organization together lead to a declining formation of naive T-cells. This loss of naive T-cells has a great influence on the peripheral T-cell pool.

With advancing age, T-cells show changed phenotype and function, diversity restrictions and replicative senescence. These age-dependent changes in T-cells contribute decisively to the development of immunosenescence. The extent of thymus functional capability is an independent factor for the determination of mortality in healthy elderly people.

**Fig. 2**
Stress interrupts the balance of the immune system and leads to acute thymic involution because of physiological conditions such as emotional stress, nutritional deficiencies, or pathological causes such as infections, diseases, and cancer treatment.[5]

Environmental stress factors such as long-lasting physical and emotional stress activate the HPA axis and induce the production of the stress hormone cortisol. This likewise results in an abrupt thymic involution and in its course to reduced thymopoiesis.[6]
Thymus peptides

The thymus produces a variety of thymus peptides that are also found in thymus extract. These include thymosin, thymulin, thymopoietin, thymopentine and thymostimulin. As various studies have shown, the individual thymus peptides have positive effects both in vitro as well as clinically (table 1). Natural thymus peptide mixtures in vivo show better effects than individual synthetic substances.
### Biochemical and clinical effect of thymus peptides

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<tr>
<th>Thymus peptide</th>
<th>Effect in vitro</th>
<th>Clinical effect</th>
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| Thymosin α1    | • Cytokine production  
• Expression of cytokine receptors  
• T-cell maturation  
• Activation of natural killer cells | • Antitumoral effects  
• Prophylaxis hepatitis B and C  
• Adjuvant for Immunizations |
| Thymosin β4    | • Involved in regulation of the cytoskeleton                                  | • Improves wound healing                                                      |
| Thymulin       | • Acts only on T-cells  
• T-lymphocyte maturation                                                      | • Treatment of primary immune deficiency (DiGeorge syndrome)                   |
| Thymopoietin   | • Maturation of prothymocyte to thymocytes  
• Stimulation of T-helper cells                                                 | • Improvement of rheumatoid arthritis                                          |
| Thymopentine   | • Inhibition of the T-suppressor cells                                         | • Prophylaxis against bacterial and viral infections                           |
| Thymostimulin  | • Differentiation and activation of T-cells                                    | • Antitumoral effect on malignant melanomas  
• Reduced infections in chemotherapies                                          |


*Table 1*
Selenium and zinc – antioxidants against oxidative stress

What are antioxidants?

Antioxidants are chemical compounds that specifically prevent undesired oxidation of other substances. Antioxidants therefore have great physiological significance as effective radical scavengers. In the organism, they inactivate free radicals, the so-called Reactive Oxygen Species (ROS), whose excessive presence in the body leads to oxidative stress. Oxidative stress is deemed partly responsible for aging and is presumed to be connected to the development of a series of medical conditions.
Oxidative stress increases the risk of a series of disorders

- Brain
  - Trauma
  - Stroke
  - Parkinson's
  - Alzheimer's

- Eye
  - Degenerative retinal damage
  - Cataract development

- Lungs
  - Asthma
  - ARDS
  - Hyperoxia

- Heart
  - Angioplastia
  - Keshan disease

- Blood vessels
  - Vasospasms
  - Atherosclerosis

- Organism
  - Aging
  - Cancer
  - Diabetes

- Kidney
  - Kidney transplant
  - Glomerulonephritis

- Gastrointestinal tract
  - Ischemic colitis

- Joints
  - Rheumatoids Arthritis

- Skin
  - Burns
  - Dermatitis
  - Psoriasis

Fig. 4
Reactive Oxygen Species (ROS)

ROS are highly reactive molecules that lack one electron. In order to complement this electron, ROS aggressively wrest the required electron from the next best molecule. These can be molecules of the cell membrane, proteins, or DNA. This electron robbery is called oxidation. Normally, there is an equilibrium between formation and decomposition of ROS in the body. If this equilibrium is disturbed, we talk about oxidative stress *(Fig. 5).*
Impact of oxidative stress on the body

If, for example, an electron was stolen by ROS from a molecule of the cell membrane, the cell membrane molecule now lacks one electron, and becomes itself a free radical with the same goal of robbing a radical from another molecule. A dangerous chain reaction is thereby set in motion, which can lead to massive damage in the body (Fig. 6).
Oxidative stress in the brain

Oxidative stress in the brain leads to cognitive limitations.\[^{10}\] Unfortunately, the brain is especially vulnerable for oxidative stress.\[^{11}\] There are a variety of causes for this (Fig. 7). First of all, the high oxygen consumption of the brain, which leads to excessive ROS production. The cell membrane of neurons is simultaneously rich in polyunsaturated fatty acids (PUFA) that are especially vulnerable to the attacks of free radicals.

An additional cause is the strong relationship of cell membrane surface to cytoplasm volume. Because of specialized neuronal forwarding, the stimulus and synaptic transfer activity of the brain cells are dependent on an efficient membrane function. The extended morphology of the axons is vulnerable to peripheral injuries, as is the neuronal anatomical network for interruptions.

The main cause for oxidative stress in the brain is glutamate

The main cause for oxidative stress in the brain is the important neurotransmitter glutamate. Simultaneously, the antioxidant defense mechanisms in the brain are low, particularly because of lower levels of glutathione peroxidase, catalase and vitamin E.

Cytochrome P450 also produces ROS, as well as activated microglia that in addition also form cytokines in a continuous process. The ROS thus generated can directly down-regulate tight-junction proteins and indirectly activate matrix metalloproteinases involved in opening the blood-brain barrier. In addition, the loss of trophic support when the brain tissue is no longer being securely provisioned with nutrients activates the NADPH oxidase. This in turn leads to an increase of ROS.

After brain damage, iron can form free radicals

The presence of hemoglobin in the neural tissue in the event of spontaneous iatrogenics or trauma is neurotoxic. Heme and iron are released and promote the formation of ROS. Moreover, iron is formed in the entire brain and after brain damage can release iron ions and form free radicals. Since neuronal cells cannot replicate, they are especially vulnerable to oxidative stress.

The interaction between nitrogen oxide and superoxides can furthermore lead to neuronal degeneration. Moreover, neuronal mitochondria produce oxygen. With increasing age, mitochondrial DNA damage therefore accumulate in the brain. Auto-oxidation of neurotransmitters also leads to the formation of oxygen and quinone which reduce glutathione.

Finally, high calcium traffic through neuronal cell membranes and the increase of intracellular calcium because of interference with iron transport plays a role. Both increased the oxidative stress in the brain.
Causes for the high susceptibility of the brain to oxidative stress


Fig. 7
A healthy body is able to avoid damaging oxidative stress.
Increase of oxidative stress due to external influences

A healthy body is able to gradually decrease the ROS formed by endogenous reactions and thus avoid damaging oxidative stress. However, people nowadays are exposed to a variety of factors that have increased oxidative stress in the human body by a multiple of the desired or useful amount (Fig. 8).
How can oxidative stress be reduced?

Oxidative stress can be reduced by different measures:[12]
1. Reducing environmentally harmful substances with oxidative characteristics
2. Increasing the level of endogenous and exogenous antioxidants
3. Reducing the development of oxidative stress

Endogenous oxidative stress can be influenced in two ways: by avoiding ROS formation or degrading ROS by antioxidants.

The intake of antioxidants through our daily diet should be the favored course, although nowadays difficult to achieve. In general the high nutrient depletion of the soils, acid rain, increased desert formation and environmental pollution, the extensive use of pesticides, the refinement of vegetables, the conversion, transport and storage of food, as well as cooking practices have all led to the decline of the antioxidant content in vegetables and fruit. This even goes so far that vegetables and fruit hardly contain essential micronutrients and antioxidants at all any more.[12]

In addition, the trace element selenium is a special case. The selenium content of fruit, vegetables and animal products depends completely on the selenium content of the soil. Because of geological developments, European soils are selenium-poor. In Germany the selenium content of the soil shows a North-South gradient, with the lowest values in Southern Germany. Cereals in Germany therefore have a 10- to 20-fold lower selenium content than American cereals.

Livestock are given European animal feed, which is why their selenium intake is also significantly reduced. Sometimes the insufficient selenium intake can cause primarily young animals to suffer from pathological selenium deficiency that can lead to the so-called white muscle disease. The young animals are too weak to stand up or suckle, and frequently die. For this reason, it is permitted to add selenium in the form of sodium selenite to the fodder in the EU.
Causes for the low content of antioxidants in vegetables and fruit

- Use of pesticides
- Plant refinement
- Post-harvest food processing
- Storage
- Transport
- Cooking practices
- Depletion of the soil

Low antioxidant content in vegetables and fruit

Strategies for Reducing or Preventing the Generation of Oxidative Stress

Fig. 9
Reduction of oxidative stress through the antioxidants selenium and zinc

Micronutrients such as selenium and zinc only have an antioxidative effect if they are incorporated in proteins. The antioxidative effect of zinc is divided into chronic and acute. Zinc unfolds a chronic effect by inducing other strong antioxidants such as metallothionein. A long-term zinc deficiency therefore leads to an increased susceptibility to oxidative stress.\(^{[13]}\) The acute effect is composed of two mechanisms, first the protection of protein sulfhydryl and second the reduction of ROS formation from hydrogen peroxide via the antagonism with redox-active transition metals such as iron and copper. In addition, zinc is part of the superoxide dismutase that degrades the superoxide radicals in hydrogen peroxide (Fig. 10).

As a component of selenoproteins, selenium is an important part of the oxidative protective mechanism in the body, both in the degradation of reactive oxygen as well as nitrogen species. Most of the selenoproteins are antioxidants, whereby the most important antioxidative selenoprotein is glutathione peroxidase, which degrades hydrogen peroxide to water. Zinc and selenium are hence both involved in the degrading of oxidative stress. A deficiency of both selenium as well as zinc makes the body vulnerable to oxidative stress.
Connection between antioxidant zinc or selenium-dependent proteins

Fig. 10
Selenium and zinc – essential for the immune system

An optimally functioning immune system has a major impact on health. Many external influences can have a negative impact on the immune system, among them a deficiency of various micronutrients. The micronutrients selenium and zinc are primarily emphasized because both micronutrients are essential for the immune system.

Selenium and the immune system

Selenium, in its function as a part of selenoproteins, plays a significant role in inflammation and immunity. An adequate selenium intake and thus an adequate selenium level in the blood is important to initiate immunity, regulate the immune response, hinder the body from an overshooting reaction, and combat chronic inflammation.\[14\]

The selenium concentration in the blood influences the so-called “oxidative burst” in both phagocytic as well as non-phagocytic cells. Various studies have shown that high selenium intake leads to an increased expression of antioxidant selenoproteins, which however does not reduce the level of ROS necessary to stimulate immune cells.\[14\] In general, selenoproteins influence the signal strength of T-cells. Especially glutathione peroxidase-1 regulates the half-life period of the ROS that arise on an “oxidative burst”.\[14\]

Human lymphocytes increase the mRNA expression of proteins involved in protein biosynthesis after a six-week intervention with 100 µg selenium per day in the form of sodium selenite.\[15\] This is probably the basis for an increased selenoprotein production and improved lymphocyte function.\[15\] A high selenium supplementation in the form of sodium selenite increases the expression of interleukin-2, a principal mediator of the immune system, and its receptors.\[16\] Because of autocrine and paracrine functions of interleukin-2, the proliferation capacity of immune cells is increased.

This study also showed that a high selenium intake displaces the TH1/TH2 balance of the T-helper cells towards TH1 (Fig. 11).\[16\] A selenium supplementation therefore promotes anti-viral immunity.
An adequate selenium intake is important for the TH1/TH2 balance of the T-cells


*Fig. 11*
One of the key mechanisms by which selenium improves the activation of T-cells is the activation of the transcription factor NFκB. The bonding of NFκB to specific genes is improved by the reduction of disulfide bonds of the NFκB-p50 subunit. The reduction of disulfide bonds is regulated by reduced thioredoxin.

This means that the more reduced thioredoxin is available in the cell, the better the immune system is activated. A high selenium intake increases the activity of the selenoprotein thioredoxin reductases-1. The greater the expression of thioredoxin reductases-1, the more reduced thioredoxin is in the cell.

Oxidative stress prior to the activation of the immune system is independent of ROS production at an “oxidative burst”. With low selenoprotein expression, the oxidative stress is increased. This oxidative stress inhibits the activation of the phagocytes. An adequate selenium supply is therefore not only necessary for the optimal activation and function of the phagocytes, but also for the expression of antioxidant selenoproteins which attenuate damage through ROS.

Eicosanoids are important modulators of inflammation and immune response. The selenium level influences the synthesis and effect of these mediators at several levels. An adequate selenium intake reduces pro-inflammatory eicosanoid biosynthesis. In addition, these feedback loops change within this signal cascade, and the selenium suppresses the pro-inflammatory eicosanoid biosynthesis and raises the anti-inflammatory eicosanoid production. Highly reactive hydroperoxide intermediate products arise within eicosanoid metabolism. Antioxidative selenoproteins protect the cells from oxidative damage by these ROS. Furthermore, selenoproteins are able to control the activity of cyclooxygenase and lipoxygenase.
Dual influence of selenium on inflammation


Fig. 12
Zinc and the immune system

Immune cells show a high proliferation rate and differentiation. They are therefore dependent on a constant supply of sufficient quantities of zinc. A zinc deficiency influences not only an individual component in the immune system, but the impact affects several levels as well as the expression of hundreds of genes.[19] Short-term effects include the regulation of the biological activity of thymulin, a thymus peptide. The long-term impacts include changes in the immune cell subpopulation.

An important mechanism of how zinc influences the immune system is its role as a signal molecule. Zinc homeostasis is controlled by three mechanisms: 1. transport through the plasma membrane by zinc transporters 2. intermediate storage by metallic ions 3. storage and release from vesicles, so-called zinzosomes[19]

Selenium and zinc preserve the balance of the immune system, among other things

Stress shifts the TH1/TH2 balance of the immune system in the TH2 direction. The cellular immune response important for the body’s defense against bacteria and viruses is thereby weakened. On the other hand, an imbalance in favor of TH2 helper cells can favor allergies, among other things. Both a selenium as well as a zinc deficiency have the same effect on the TH1/TH2 balance. A massive imbalance between TH1 and TH2 helper cells can develop. Selenium in the form of sodium selenite promotes the TH1 helper cells. The equilibrium of TH2 can also shift again in the direction of TH1.

In summary, a selenium and zinc supplementation can compensate a TH1/TH2 imbalance and consequently support the proper functioning of the immune system.
Zinc and the immune system

B-cells lymphopoiesis
T-cells function
TH1/TH2 balance
Production of antibodies

Acquired immunity
Congenital immunity

- Natural killer cell activity
- Oxidative burst
- Killing the bacteria of parasites
- Phagocytosis


Fig. 13

Zinc and selenium keep the immune system in balance, among other things

Stress ➔ TH1 ➔ TH2 ➔ Increased susceptibility for infections
+ Selenium + Zinc

Fig. 14
Selenium – essential for the thyroid gland

The thyroid gland is the organ with the highest selenium content per gram of tissue. Like iodine, selenium is essential for the thyroid gland. Selenoproteins are required for the creation of active thyroid gland hormones.

The majority of human thyroid gland hormones are in an inactive form (T4). For activation, an iodine atom must be split off from T4. To do this, the selenium-dependent deiodinase that form active T3 is required. Simultaneously, the formation of T4 from tyrosine produces hydrogen peroxide, that in turn must be decomposed in order to protect damage on thyroid gland tissue by the accumulation of hydrogen peroxide, hence oxidative stress. Glutathione peroxidase is responsible for doing this. The reaction uses NADPH that is regenerated by an additional selenoprotein, thioredoxin reductase (Fig. 15).
Effect of selenium supplementation on a hypofunctional thyroid

- Selenium protects the thyroid tissue
- Selenium reduces thyroid inflammations

**Fig. 15**
The symptoms of thyroid gland hypofunction overlap with symptoms for chronic stress.
Since the conversion of the inactive thyroid gland hormone T4 to active T3\(^{[21]}\) is reduced upon stress, a simultaneous selenium deficiency further reduces the concentration of T3 and greatly restricts the function of the thyroid gland. The symptoms of thyroid gland hypofunction are multifaceted and overlap with symptoms for chronic stress (Fig. 16).

<table>
<thead>
<tr>
<th>Symptoms of a hypofunctional thyroid</th>
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<tr>
<td><strong>Brain, Psyche</strong></td>
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<tr>
<td>• Depressive moods</td>
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<tr>
<td>• Irritability</td>
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<tr>
<td>• Anxiety</td>
</tr>
<tr>
<td>• Concentration disorders</td>
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<tr>
<td><strong>Heart</strong></td>
</tr>
<tr>
<td>• Dizziness</td>
</tr>
<tr>
<td>• Low blood pressure</td>
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<tr>
<td>• Slow heartbeat</td>
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<tr>
<td>• Slow pulse</td>
</tr>
<tr>
<td>→ Physical weakness</td>
</tr>
<tr>
<td><strong>Gastro-intestinal tract</strong></td>
</tr>
<tr>
<td>• Constipation</td>
</tr>
<tr>
<td>• Flatulence</td>
</tr>
<tr>
<td>• Sensation of bloating</td>
</tr>
<tr>
<td>• Nausea</td>
</tr>
<tr>
<td>• Stomach pain</td>
</tr>
<tr>
<td>• Disturbed metabolism</td>
</tr>
<tr>
<td>→ Weight gain, or temperature sensitivity marked by frequent and rapid sensation of freezing</td>
</tr>
<tr>
<td><strong>Muscles</strong></td>
</tr>
<tr>
<td>• Loss of muscular strength</td>
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<tr>
<td>• Muscle cramps</td>
</tr>
<tr>
<td>• Muscle pain</td>
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<tr>
<td><strong>Skin, Hair, Nails</strong></td>
</tr>
<tr>
<td>• Hard and brittle nails</td>
</tr>
<tr>
<td>• Dry and straw-like hair</td>
</tr>
<tr>
<td>• Dry and pale hair</td>
</tr>
<tr>
<td><strong>Bones</strong></td>
</tr>
<tr>
<td>• Bone loss</td>
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<tr>
<td>• Osteoporosis</td>
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Abb. 16
Zinc – For normal cognitive function

More than 200 proteins contain zinc as a co-factor, and zinc is a structural component in many additional proteins, hormones, hormone receptors and neuropeptides. In addition, zinc plays another role as neurosecretory product and co-factor in central nervous systems. Zinc occurs in high concentrations in the synaptic vesicles of the so-called “zinc-containing” neurons. These neurons are found almost exclusively in the forebrain. About 20% of the zinc content in the brain is located in these vesicles. The other 80% is part of the zinc metalloproteins. Previously, the precise role of zinc in the brain was not totally clear. But in the meantime, it has been established that a zinc deficit leads to cognitive impairment.
What are cognitive capabilities?

The cognitive capabilities of humans include learning, memory, perception, attention, orientation, planning, creativity, problem-solving and abstraction (Fig. 17).
When are cognitive functions impaired?

Burnout has previously not been recognized as a medical disorder itself, but rather considered to be a possible condition for causing other disorders. The term originally meant physical, emotional and mental exhaustion caused by work overloads. In the meantime, the term also includes conflicts between job and family, or the burdens involved in taking care of a needy relative. This overload is usually triggered by stress.

Stress-caused states of exhaustion result in restrictions of cognitive capabilities. Chronic stress influences several brain structures, among them the hippocampus, amygdala and prefrontal cortex. These structural changes lead to restricted memory functions. Several studies have examined the connection between burnout and cognitive restrictions. Those affected have problems with attention, concentration, memory and flexibility.

Zinc positively influences cognitive functions

Various studies have examined the influence of zinc supplementation on cognitive functions. Primarily, the quantification of individual cognitive capabilities have thereby proved to be problematical.

A pilot study in Japan investigated the effect of 7 mg zinc supplement per day for 10 weeks on young women. The result showed that zinc supplementation can reduce both the feeling of annoyance as well as depression (p = 0.009 or p = 0.011).

A study with school children who took a daily supplement of 5 mg zinc for three months showed that the zinc supplement improved specific cognitive capabilities (p = 0.009). The results of the study are also very interesting, because no zinc deficiency was found in the participating children.

A large study with older participants between 55 and 87 years without a zinc deficit were supplemented with 15 mg or 30 mg zinc per day for six months. However, after 3 months only one individual test showed a significant improvement (p = 0.03).
Causes for impaired cognitive functions

- Depression
- Neuro-degenerative diseases
- Exhaustion, tiredness
- Medications
- Permanent brain damage
- Zinc deficiency
- Burnout

Cognitive abilities

Fig. 18
Bibliography


THYMVITAL®

to support the immune system and the vital functions

THYMVITAL® at a glance

• Unique galenic

• High-quality ingredients

• Only one capsule per day – and thus less expensive for the daily requirement than the products of the competition

• Optimal dosing

Recommended intake

Take one capsule per day about 30 minutes before breakfast unchewed with some liquid.

THYMVITAL® should not be simultaneously consumed with high-dose, gastric juice-resistant, small intestine-active enzyme preparations (proteolytic enzymes), since the thymus extract might possibly be destroyed.

It has proven to be beneficial, for instance, to take a 4-week break after taking THYMVITAL® for three months.
Nutritional supplements

Ingredients: Thymus extract powder from calves (50%); filling material: microcrystalline cellulose; crystalline silica: shellac; beef gelatin (capsule); coloring agents: E 171, E 555; gelatin capsule; empty gelatin capsule; sodium selenite pentahydrate.

Package sizes: Package with 30 capsules (contents: 21 g)

Attention: The specified recommended daily dosage may not be exceeded. Food supplements should not be used as substitutes for a balanced and diversified diet. Use during pregnancy and lactation as well as by children below 12 years of age is not advised.

Store beyond the reach of small children.

08/2013

Contact and information

Further information is available at:
www.biosyn.de / www.thymvital.de

Please do not hesitate to get in touch with us for more information or feedback. We would also gladly send you information by post, upon request! You can request our Online Newsletter by sending an email to: information@biosyn.de, Keyword THYMVITAL®
THYMVITAL®
Dietary supplement with thymus extract, zinc and selenium

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