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What can biofactors do for dementia?

Many patients are affected by mental and cognitive disorders such as dementia. Scientific studies have shown that vitamins B1 and B12 in particular have a positive effect on dementia.

The term dementia describes chronic diseases of the brain that are accompanied by a gradual decline in cognitive, emotional and social abilities. The most common form of dementia is Alzheimer's disease, which accounts for around 60% of dementia cases worldwide. Other forms include vascular dementia, which accounts for around 15%, mixed forms between the two and, in 10% of cases, other forms of dementia such as Lewy body dementia, Parkinson's dementia, drug-induced dementia or dementia resulting from traumatic brain injury.
According to estimates by Alzheimer's Disease International, 46.8 million people worldwide are affected by dementia - and almost 8 million new cases are added every year. According to statistics, the number of dementia sufferers in Germany will also increase. While just under 1.6 million people were living with dementia in 2018 - which corresponds to 1.9% of the population - experts expect this figure to rise to 2.7 million in 2050 (3.4%).[1]

Note:
- A distinction must be made between primary and secondary dementia.
- In primary dementia - in contrast to the secondary form - the cause of the disease lies in cognitive areas of the brain itself and is not triggered by external factors.
- Primary forms of dementia are not yet reversible.
- The prevalence of causally treatable dementia is stated in the literature to be a maximum of 30%, with only some of the cognitive disorders being reversible at all despite adequate therapy.
- The most common causally treatable causes of cognitive impairment in secondary forms of dementia are depression, drug-induced cognitive impairment and vitamin B12 deficiency.
- In geriatric patients with suspected dementia, vitamin B12 deficiency even proved to be the second most common cause of the disease after depressive pseudodementia when the reversible causes were considered.
- In practice, this means that the disease as a whole cannot be cured, apart from the secondary forms of dementia mentioned above. Instead, the treatment of affected patients aims to alleviate the symptoms and slow down the progression of the disease.

Dementia and the biofactor vitamin B12 - what do the studies say?
In addition to the classic symptoms of vitamin B12 deficiency such as megaloblastic anemia or funicular myelosis, cognitive and memory disorders are also possible. A vitamin B12 deficiency can therefore be associated with significantly lower memory performance and, as already mentioned, is one of the most common treatable causes of secondary dementia.[2,3]

There is an increased risk of developing dementia with a vitamin B12 deficiency.[4] For example, a multicenter study was able to show that even mild cognitive deficits correlate with reduced vitamin B12 and increased homocysteine levels - a vascular-damaging amino acid. In addition, after three months of vitamin B12 supplementation, serum vitamin B12 levels had normalized in all participants, 84% of them reported an improvement in their cognitive symptoms and 78% achieved better scores in the Mini-Mental Status Test.[5]
Other studies have also confirmed the correlations described and also indicate associations between low vitamin B12 levels and elevated serum homocysteine levels with an increased risk of Alzheimer's dementia.[6,7]

Vitamin B12 deficiency: detect an treat early
The psycho-cognitive disorders of dementia observed in vitamin B12 deficiency can precede the hematological abnormalities of deficiency-related megaloblastic anemia by months to years or occur without any symptoms of anemia. The presence of a vitamin B12 deficiency can therefore be overlooked if only the blood count is used as an indicator of a vitamin B12 deficiency, especially as the onset of a deficiency can lead to rather unspecific symptoms such as tiredness and fatigue, concentration and sleep disorders, poor performance, low mood or inappetence. Detailed knowledge of the symptoms of vitamin B12 deficiency and targeted laboratory diagnostics are therefore particularly recommended for older patients with suspected cognitive disorders.

Dementia and diabetes mellitus: what role does vitamin B1 play?
Patients with type 2 diabetes have a significantly higher risk of developing Alzheimer's dementia (AD) compared to non-diabetics. There are now numerous indications that type 2 diabetes can contribute to Alzheimer's disease via impaired central glucose metabolism in the nerve cells and reduced glucose utilization in the brain.[8,9] The earlier diabetes-related changes occur, the stronger the link between type 2 diabetes and dementia. On the other hand, treatment with antidiabetic drugs over a longer period of time can attenuate the development of dementia.[10,11]

What effect can the biofactor vitamin B1 have on dementia?
A vitamin B1 deficiency could also play a role in Alzheimer's dementia, as reduced vitamin B1 concentrations have been detected in the brains of AD patients. Studies from previous years have already shown that compensating for a vitamin B1 deficiency could be a promising approach: For example, scientists demonstrated in animal experiments that eight weeks of treatment with the lipid-soluble vitamin B1 precursor benfotiamine in mice suffering from Alzheimer's disease can not only reduce pathological brain changes such as plaque formation, but also significantly improve performance deficits in learning behavior.
The scientists used the precursor benfotiamine in their studies because previous studies with water-soluble thiamine had only shown a slight positive effect. The main reason for this was considered to be the very low bioavailability of orally ingested water-soluble thiamine compounds. The fat-soluble precursor benfotiamine enters the body and tissues in much higher concentrations, which is obviously an important prerequisite for its effectiveness in the pathogenesis of Alzheimer's dementia.[12] The

significantly higher bioavailability of benfotiamine compared to water-soluble thiamine has also been demonstrated in other studies.[13]

Effect of benfotiamine also confirmed clinically[14]

A small but controlled human study on the benefits of oral benfotiamine in Alzheimer's dementia also showed promising results. According to the authors, benfotiamine already improved pathological factors that define Alzheimer's dementia in preclinical studies. Based on these results, the scientists now tested benfotiamine versus placebo for 12 months in people with mild cognitive impairment and mild Alzheimer's disease. The primary clinical endpoint was the "Alzheimer's Disease Assessment Scale-Cognitive Subscale" - abbreviated as ADAS-Cog. The other endpoints were the Clinical Dementia Rating (CDR) score and the measurement of blood AGEs as known risk factors for AD development. The results were quite positive:

- The increase in ADAS-Cog was 43% lower (almost statistically significant ($p = 0.125$)) in the benfotiamine group compared to placebo, indicating less cognitive decline.
- The worsening of CDR was 77% less in the benfotiamine group compared to placebo.
- Benfotiamine significantly reduced the increase in AGEs.

"Oral benfotiamine is safe and potentially effective in improving cognitive outcomes in individuals with mild cognitive impairment and mild Alzheimer's dementia," the authors summarized the results of their research.

Conclusion for practice

In summary, this article shows that - even if the study situation is not always consistent - attention should be paid to the adequate supply of selected biofactors in the treatment of psycho-cognitive disorders such as dementia in addition to classical methods.

Literature

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