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# For diabetic neuropathy compensate for vitamin B1 deficiency

**In order to intervene in the process of diabetic neuropathy, in addition to improving metabolic control and avoiding nerve-damaging risk factors such as alcohol abuse and smoking, a sufficient supply of the biofactor vitamin B1 should also be considered.**

The active metabolite of vitamin B1 (thiamine), thiamine diphosphate, is synthesized directly in the peripheral nerve cells, where it acts as a coenzyme in carbohydrate metabolism. Thiamine is particularly important as a cofactor for the enzyme transketolase, which plays a central role in the pentose phosphate pathway. This metabolic pathway produces NADPH, which is required for antioxidant defense against cell damage. A lack of thiamine reduces the activity of transketolase, which leads to a reduced NADPH level and increases oxidative stress. High oxidative stress in turn promotes the formation of AGEs (advanced glycation end-products).[1]
Hyperglycemia increases the need for vitamin B1. In addition, patients with diabetes mellitus may experience up to a fourfold increase in renal loss of the biofactor due to the disease itself. It must also be taken into account that the body can only store small amounts of the water-soluble vitamin and only for a short time. This can further increase the risk of neuropathies due to hyperglycemia.

**Benfotiamine versus thiamine**
Thiamine is transported in the body both actively and passively, depending on the concentration gradient. The uptake of thiamine from the small intestine into the enterocytes is mainly active, i.e. energy-intensive. Thiamine is transported into the cells against the concentration gradient by special transporters. These transporters use ATP to transport thiamine into the enterocytes.
If the concentration of thiamine in the cells is higher than in the intestine, thiamine can also be absorbed passively. This process does not require any energy, but takes place by diffusion via transport proteins, which introduce thiamine into the cells according to the concentration gradient. However, only a small amount is absorbed.
To counteract this, benfotiamine, a lipid-soluble precursor of thiamine, is often used. Benfotiamine is five times more bioavailable than thiamine and can be absorbed directly into the blood, where it then reaches the cells of the corresponding organs - without the need for a transporter. In comparison, equimolar amounts of thiamine increase the activity of transketolase by around 20%, while benfotiamine can increase activity by up to 400%.[2,3]
Targeted supplementation with benfotiamine can compensate for a thiamine deficiency, which can cause nerve damage. It also promotes the activation of thiamine-dependent transketolase, which inhibits pathogenic metabolic pathways. In this way, benfotiamine can counteract cell-toxic metabolic changes and alleviate symptoms of neuropathy.[4,5]

Recommendation for the practice

Laboratory diagnosis of vitamin B1 deficiency can be difficult. If a deficiency is suspected, a therapy trial with orally administered vitamin B1 in the form of benfotiamine is therefore recommended. Various randomized studies have tested different daily doses, and the optimal dose was found to be 300 mg benfotiamine twice daily. [6]

You can find more information here.

Do you suspect that you or your patients suffer from a deficiency of selected biofactors? Take the biofactor check and find out your personal risk.

***Literature:***


[1] Thornalley PJ et al.: High prevalence of low plasma thiamine concentration in diabetes linked to a marker of vascular disease. Diabetologia 2007 Oct; 50(10): 2164-2170

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[4] Stirban A: Therapie der diabetischen Neuropathie. 27. Kongress der Föderation der Internationalen Donau-Symposia über Diabetes mellitus. Diabetes-Congress-Report 2013; 2: 4-10

[5] Raj V et al.: Therapeutic potential of benfotiamine and its molecular targets. Eur Rev Med Pharmacol Sci 2018; 22: 3261-3273

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