BENFOTIAMIN

Peter Kempler
1st Dept. of Medicine, Semmelweis University, Faculty of General Medicine, Hungary

The absorption of water-soluble thiamine is limited and decreases further with increasing oral intake. According to comparative studies, the biological effectiveness of the thiamine prodrug benfotiamin (S-benzoylthiamine-O-monophosphate) is ten times higher than that of thiamine mononitrate. Benfotiamin has been shown to prevent three major pathways (the advanced glycation end – products pathway, the hexosamin pathway just as the protein kinase – C- diacilglycerol pathway) and may have significant utility in preventing complications. By boosting the activity of the enzyme transketolase, benfotiamin directs glucose substrates to the pentose phosphate pathway. These data has been outlined in the Banting Memorial Lecture, held by Professor Michael Brownley in 2004 at the annual meeting of the American Diabetes Association in Orlando.

It has been documented that benfotiamine accelerates the healing of ischaemic diabetic limbs in mice through protein kinase B/Akt-mediated potentiation of angiogenesis and inhibition of apoptosis. Benfotiamin has been shown to prevent endothelial dysfunction and oxidative stress following a meal rich in advanced glycation end products (AGE). In adult diabetic patients, benfotiamin has been reported to decrease hyperglycemia-induced damage and significantly reduce the neuropathic sensory symptoms in several studies. Particularly, an improvement in neuropathic pain and in the vibration perception threshold was documented. Moreover, significant improvements in nerve conduction velocity have been demonstrated.
In summary, nowadays benfotiamine is considered to have been established as a pathogenetic oriented therapeutic approach improving oxidative stress and metabolic changes caused by hyperglycemia. At the same time, it is a powerful drug that reduces symptoms of peripheral sensory neuropathy.