

**Benfotiamine Slowing and Blocking Diabetic
Complication and Retinopathy
Benfotiamine Blocks Three Major Pathways of
Hyperglycemic Damage and Prevents Experimental
Diabetic Retinopathy**

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Abstract

Three of the major biochemical pathways implicated in the pathogenesis of hyperglycemia induced vascular damage (the hexosamine pathway, the advanced glycation end product (AGE) formation pathway and the diacylglycerol (DAG)-protein kinase C (PKC) pathway) are activated by increased availability of the glycolytic metabolites glyceraldehyde-3-phosphate and fructose-6-phosphate. We have discovered that the lipid-soluble thiamine derivative benfotiamine can inhibit these three pathways, as well as hyperglycemia-associated NF-kappaB activation, by activating the pentose phosphate pathway enzyme transketolase, which converts glyceraldehyde-3-phosphate and fructose-6-phosphate into pentose-5-phosphates and other sugars. In retinas of diabetic animals, benfotiamine treatment inhibited these three pathways and NF-kappaB activation by activating transketolase, and also prevented experimental diabetic retinopathy. The ability of benfotiamine to inhibit three major pathways simultaneously might be clinically useful in preventing the development and progression of diabetic complications.