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Biochemical origins of diabetic complications; Contrasting influences of dietary components

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Biochemically, diabetic complications mostly arise as a consequence of excessive sugar catabolism via the glycolytic pathway. It is well recognised that generation of methylglyoxal (MG), a metabolic by-product formed following the spontaneous decomposition of the triose phosphates dihydroxyacetone phosphate (DHAP) and glyceraldehyde-3-phosphate (G3P), plays an important role in diabetic complications. MG is a highly reactive bicarbonyl which readily reacts with protein amino and guanidine groups via a process called non-enzymic glycosylation (glycation), eventually forming advanced glycation end-products (AGEs). DHAP and G3P are also glycating agents but less active than MG. It suggested that the glycolytic enzyme, triosephosphate isomerase (TPI) (which converts DHAP to G3P), is a metabolic Achilles' heel due to the enzyme's catalysis-induced loss of activity occurring as a result the deamidation of specific asparagine residues. Thus, excessive glycolysis may result in decreased TPI activity which will provoke DHAP accumulation and hence MG generation. This would be even more prevalent in cells in which synthesis of replacement TPI molecules is impossible, such as erythrocytes and in cells of eye lens core. Indeed it is possible that erythrocytes may be a major source of serum MG under conditions of excessive or continuous glycolysis. The possible role beneficial effects of the naturally-occurring, endogenous and dietary dipeptide, carnosine, in modulating glycolysis and scavenging MG will also be discussed.

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