

Glycation injury effects on aging and possible ways of prevention

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Abstract

Glycation is both a physiological and pathological process that mainly affects proteins, nucleic acids, and lipids. Advanced glycation end product (AGE) formation begins with the non-enzymatic glycation of free amino groups by sugars and aldehydes which leads to a succession of rearrangements of intermediate compounds and ultimately to irreversibly bound products known as AGEs. Recent studies have revealed the contributing roles of AGEs in the development of various aging-related conditions, such as diabetes, heart disease, and cancer. Controlling the blood sugar level is a natural method to inhibit glycation in diabetes. It is also seen from animal studies that exercise reduces the concentration of AGEs and highly reactive intermediates of AGE.

Methods of glycation

Glycation is both a physiological and pathological process that mainly alters proteins, nucleic acids, and lipids.

Exogenous and endogenous glycation produces harmful reactions that take place principally in the extracellular matrix or

within the cell cytosol and organelles. Advanced glycation end product (AGE) formation starts with the non-enzymatic glycation of amino groups by sugars and aldehydes which causes a succession of rearrangements of intermediate compounds and leads to irreversibly bound products known as AGEs. Nucleotides and lipids are mainly vulnerable targets that can in turn favor DNA mutation.

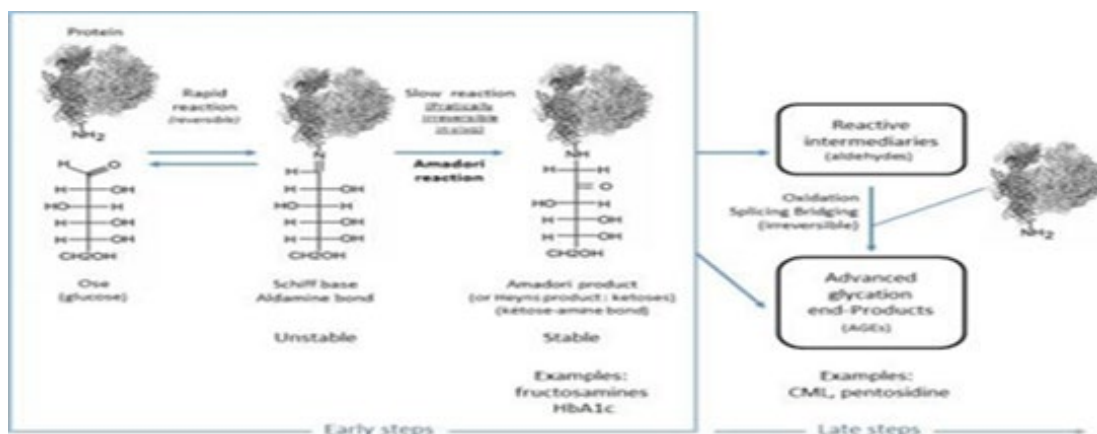


Figure 1: Glycation process

Results

Chronic hyperglycemia exhibits elevated AGE formation, and increased serum AGE levels. These changes lead to increased oxidative stress, insulin resistance, inflammation, pancreatic β-cell dysfunction, and eventually diabetic complications. AGEs can stimulate cardiovascular complications in the presence or absence of hyperglycemia. Accumulated AGEs also interact with mononuclear, endothelial, and smooth muscle cells, and cause cellular dysfunction, tissue damage, and atherosclerosis development. Dietary and endogenous AGEs can promote oxidative stress contributing to the accelerated progression of

obesity-related complications such as insulin resistance, AGE accumulation, and increased proinflammatory cytokines in adipose tissues. Accumulation of AGEs affects multiple systems are found to be associated with osteoporosis, gut microbiome-associated diseases, kidney diseases, cancer, neurodegenerative diseases, Parkinson’s disease, etc. The list goes on.

Discussion

When a carbohydrate attaches to a protein through fixation, the characteristics of that protein are altered. The protein can become resistant to enzymes for which it previously acted as the

substrate, resulting in an accumulation.

Glycation contributes to the mechanisms of insulin resistance. In addition, glycated insulin in pancreatic cells makes it harder to maintain homeostatic levels of glucose and stimulate lipogenesis. The glycation of enzymes induces conformational changes near the enzyme's active site. Paraonase, a plasma enzyme concerned with high-density lipoproteins that prevent the oxidation of LDL is inhibited by glycation. This can lead to hypertension or also thrombosis. Immunoglobulins are one of the families of proteins greatly affected by glycation. The glycation of immunoglobulin M is two times higher than that of immunoglobulin G, due to its different composition

Prevention and Conclusion

Controlling the blood sugar level is a very effective and natural method to inhibit glycation in diabetes.

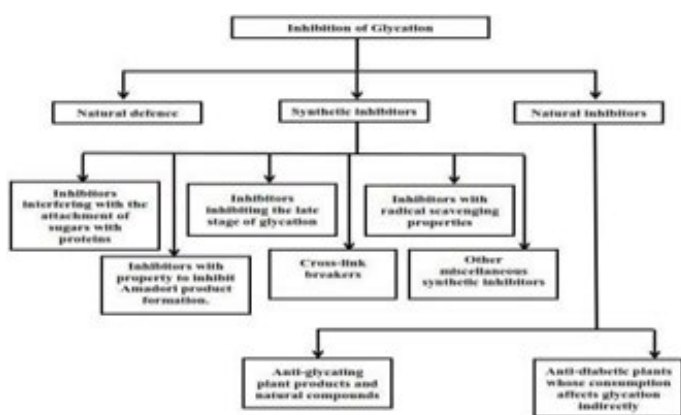


Figure 2: Inhibition of glycation: Possible strategies

Antioxidant compounds may be promising agents for prevention too. Several plant products have been proposed to be effective supplements against glycation. naturally occurring phytochemicals with anti-diabetic and antiglycation activities are relatively nontoxic, inexpensive, and available in an ingestible form. Exercise is also an effective way to prevent the aging effects of AGEs. Prevention of AGEs can help us live healthier life.

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