

SUMMARY

Non-enzymatic glycation is the reaction between aldehyde or ketone group of sugars and free amino group of protein. A complicated molecular process involving simple and more complex multistep reactions leads to the formation of heterogeneous advanced glycation end products (AGEs). Reactive intermediate reaction products, after undergoing numerous chemical modifications, form a series of derivatives that contribute to oxidative stress and structural protein changes, including the induction of protein aggregation or the reduction in binding of drugs to plasma proteins.

Glycation is associated with the generation of reactive oxygen species and when an oxidative step is involved, the reaction is called glycoxidation.

Generation of AGEs is severe in many health disorders, including hyperglycaemia, diabetic complications and neurodegenerative diseases. In recent years, oxidative stress, which is defined as an imbalance between the generation of reactive oxygen species and their elimination, as well as increased glycation were also included among the factors involved in the etiology of multiple sclerosis and cystic fibrosis.

This paper presents the results of studies showing whether glycoxidative damage of serum protein induced by oxidative stress is common in patients with multiple sclerosis. Moreover, this paper contains the comparison of the levels of glycoxidative damage of plasma protein in pediatric patients with cystic fibrosis with chronic bacterial infections. In addition, the paper presents an attempt to demonstrate the mechanism of the protective effects of antioxidants by determining the role of metal chelation in the glycation of albumin.

The data indicate that the determination of protein glycoxidative damage can be successfully used as a marker for the assessment of the progress of multiple sclerosis and cystic fibrosis. It has been shown that there is no correlation between the ability to chelate iron and antiglycation activity of the tested compounds.