# Inhibitory Effect of Some Acetyl Esters and Acetamides on Glycation of the Histone H1

Georgi Stoynev[a,*], Ljuba Srebreva[a], Roumyana Gugova[b], Evgeny Golovinsky[b], and Ivan Ivanov[a]

[a] Department of Gene Regulations, Institute of Molecular Biology, Bulgarian Academy of Sciences, 1113 Sofia, Bulgaria. Fax: +3 59 28 20 15 86. E-mail: stoynev@obzor.bio21.bas.bg
[b] Department of Molecular Design and Biochemical Pharmacology, Institute of Molecular Biology, Bulgarian Academy of Sciences, 1113 Sofia, Bulgaria

* Author for correspondence and reprint requests

Z. Naturforsch. **63 c**, 526–532 (2008); received January 10/March 5, 2008

Non-enzymatic glycosylation (glycation) is a spontaneous set of reactions between reducing sugars and free amino groups in proteins or other biomolecules leading to the formation of fluorescent and coloured compounds known as advanced glycation end products (AGEs). AGEs cause structural changes of key proteins in humans, and therefore they are related with a number of physiological processes and diseases such as aging, atherosclerosis, cataract, arthritis, Alzheimer's disease. Two main strategies have been employed to prevent the formation of AGEs: a) low carbohydrate diet and b) pharmacological intervention. The latter includes treatment with reactive compounds which might be either sugar competitors (type A), carbonyl traps (type B) or free radical trapping antioxidants (type C). Acetylsalicylic acid (ASA, aspirin) is a good example of sugar competitor capable of inhibiting glycation by acetylating $\varepsilon$-amino groups of lysine residues in proteins. Taking into consideration the inhibiting effect of ASA on glycation we designed to study the antiglycation activity of other acetyl group-containing compounds (acetamides and acetyl esters) using the lysine-rich protein histone H1 as a model. The glycation of the histone H1 was carried out by either fructose or a complex mixture of glycating agents obtained from *E. coli* and monitored by fluorescent spectroscopy, SDS-PAGE and measurement of the content of reactive carbonyl groups in the target protein. Our results showed that the inhibitory effect of phenyl acetate, acetanilide, 4-acetamidophenylacetic acid and isopropenyl acetate was comparable to that of ASA. Based on the obtained results we conclude that these compounds act as free radical scavengers protecting proteins from the damaging effect of reactive oxygen species produced during the formation of AGEs.

*Key words:* Glycation End Products, Glycation Inhibitors, Histone H1