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Effect of on 1,2,3-Triazole Derivatives the Activity of Liver Antioxidant Enzymes in Alloxan Diabetes

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A B S T R A C T

In this study, the effects of 1,2,3-triazo derivatives on the amount of lipid peroxidation product malondialdehyde (MDA) and superoxide dismutase (SOD), catalase activity in rat liver homogenate under the conditions of alloxan diabetes were studied.New derivatives of triazoles, TF-25, TS-27 and TB-31, were found to inhibit MDA content in rat liver homogenate and increase activity of antioxidant enzymes SOD and catalase in alloxan diabetes.

Introduction

The antioxidant defense system is represented by special enzyme systems SOD, catalase, glutathione peroxidase, glutathione transferase, peroxidases and non-enzymatic compounds (metal chelates - transferrin, ceruloplasmin, which inhibit the initial phase of LPO; neutralizing free radicals - ascorbate, vitamin E, reduced glutathione)., coenzyme Q, uric acid, bilirubin), which stops the acceleration of the LPO process (Memisoğullari *et al.*, 2003). The superoxide radical is usually eliminated by the antioxidant defense system. In mitochondria, free radicals are converted into H_2O_2 by the SOD enzyme, then split into H_2O and O_2 by glutathione peroxidase (in mitochondria) or diffuse into the cytoplasm, in that place neutralized by catalase in peroxisomes.

Elevated levels of MDA, the end product of LPO, and increased levels of antioxidant enzymes suggest that there is a link between increased oxidative stress due to LPO in diabetic conditions. Antioxidant enzymes play an important role in neutralizing ROS generated during oxidative stress.

In experimental diabetes, metabolites begin to form as a result of increased generation of free radicals in

the mitochondria of the heart, pancreas and liver (Sivitz, 2010). The toxic effect of metabolites is associated with the acceleration of the process of lipid peroxidation, which leads to a violation of the structural and functional unit of the cell membrane. (Ayala *et al.*, 2014). However, alloxan can reduce the amount of MDA in the liver homogenate in diabetic conditions.

To explain this mechanism, we determined the effect of new derivatives of 1,2,3-triazoles TF-25, TS-27 and TB-31 on the amount of MDA, the end product of the LPO process, in liver tissues in diabetic conditions.

Materials and Methods

To prepare a liver tissue homogenate, the livers of animals from the experimental groups were isolated. Homogenized in a Potter homogenizer in 120 mM KCl and 30 mM phosphate buffer (pH 7.4) at 0-4 ºC.

Fractions of nuclei and cells were centrifuged at 600 g for 10 min. The resulting supernatant was used to determine the activity of antioxidant enzymes in the form of a liver tissue homogenate.

MDA content, SOD and catalase activity were determined in the liver homogenate. The amount of MDA in the liver homogenate of experimental animals (Vladimirov, Archakov, 1972; Andreeva i dr., 1989) was determined by the proposed method.

The principle of this method was determined by the method based on the interaction of thiobarbituric acid with MDA formed from the peroxidation of unsaturated fatty acids with 2-3 diene bonds.

Superoxide dismutase activity (SOD) was determined based on the method of Chumakov (1977). Enzyme activity was expressed in units of Ed/mg acyl. Catalase activity was achieved using a method based on the formation of a yellow compound of hydrogen peroxide with molybdenum salts (Koralyuk, 1988)

Results and Discussion

In our experiment, it was found that the amount of MDA in the liver homogenate of group II animals with alloxan diabetes is increased compared to the concentration of MDA in the liver homogenate of control rats. The increase in the amount of MDA in the liver homogenate of animals with type II diabetes mellitus was 3.90 times higher than the control values and amounted to 4.3±0.25 nmol/mg protein.

New derivatives of 1,2,3- triazoles TF-25, TS-27 and TB-31 were administered orally to alloxan diabetic rats for 10 days. The level of glucose in the blood was monitored and experiments were carried out to determine the content of MDA in the homogenate of the liver of rats, which approached the control. It was found that the amount of MDA in the liver homogenate of rats receiving pharmacotherapy with new derivatives of triazoles TF-25, TC-27 and TB-31 decreased compared to the values of group II.

The amount of MDA in the liver homogenate of group III rats treated with TF-25 was 2.8 ± 0.17 nmol/mg protein, group IV treated with TS-27 was 1.9 ± 0.09 nmol/mg protein, and group V, who received TB-31. amounted to 2.3 ± 0.14 nmol/mg protein,The amount of MDA is 1.53, respectively, compared with the indicators of group II; Revealed a decrease of 2.26 and 1.72 times (Fig. 1). According to the literature, an increase in the amount of MDA in the liver occurs under the influence of activation of the process of lipid peroxidation in conditions of experimental diabetes (Miaffo *et al.*, 2019). Our experience also confirms the above. In our next experiments, as a result of pharmacotherapy of alloxan diabetic rats with new derivatives of 1,2,3-triazoles TF-25, TC-27 and TB-31, a change in the activity of the antioxidant enzyme SOD in the liver. (Fig. 2).According to the results, it was found that the amount of SOD in the liver homogenate of alloxan diabetic rats is 6.9±0.5 U/mg protein and decreases by 45.3% compared to the control (12.6±0.7 U/mg protein).

Fig.1 Effect of novel derivatives of 1,2,3-triazoles TF-25, TS-27 and TB-31 on MDA content in liver homogenate in alloxan diabetes. *R<0.05; **R<0.01; n=6.

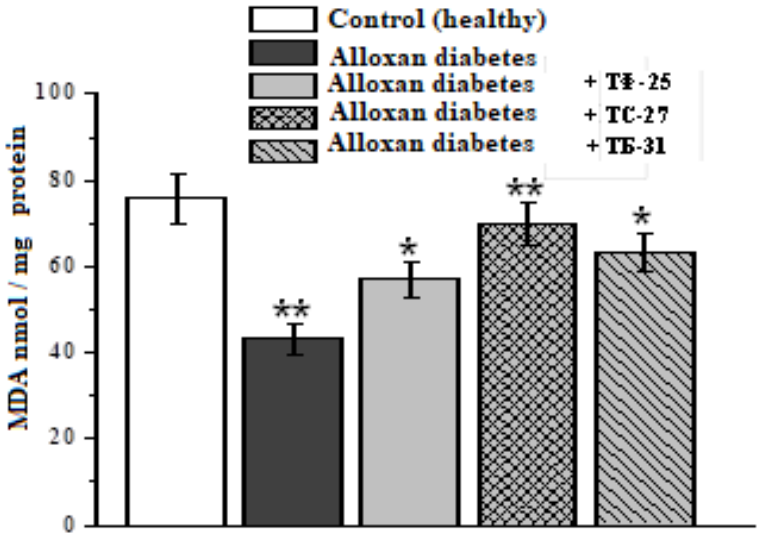


Fig.2 Effect of novel derivatives of 1,2,3-triazoles TF-25, TS-27 and TB-31 on SOD content in liver homogenate in alloxan diabetes. *R<0.05; **R<0.01; n=6.

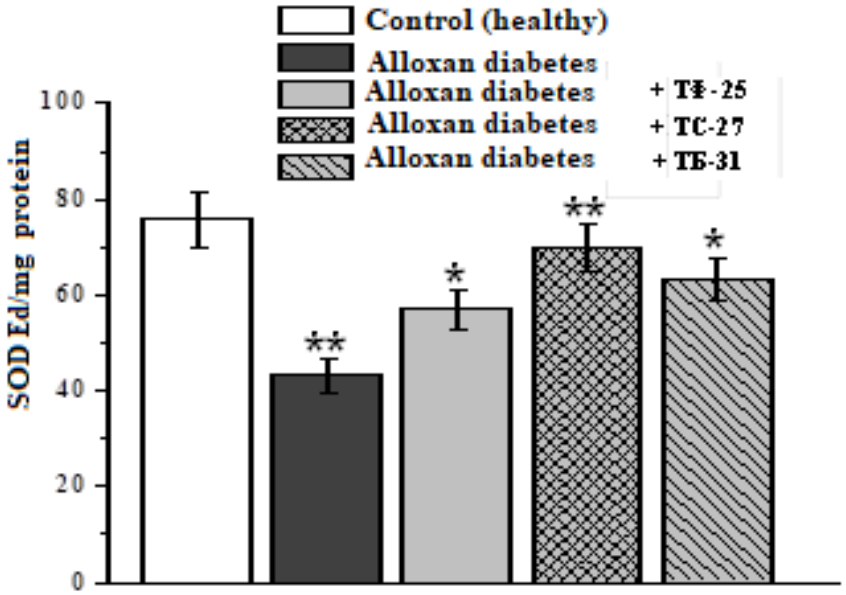
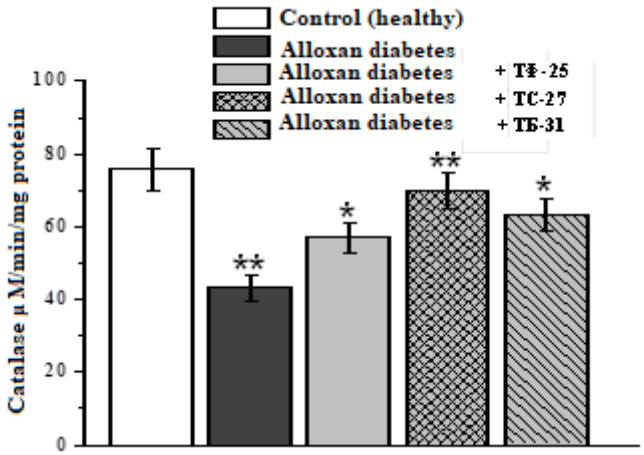


Fig.3 Effect of new derivatives of 1,2,3-triazoles TF-25, TS-27 and TB-31 on catalase activity in liver homogenate in alloxan diabetes. *P<0.05; **P<0.01; n=6.



Compared to group II, the amount of SOD in the liver homogenate of rats of experimental groups III, IV, and V treated with new triazole derivatives TF-25, TS-27, and TB-31 was 26.1%, 62.3%, and 39.1%, respectively. It was found that it increased by (Fig. 2).In conditions of diabetes, SOD enzyme activity in the liver decreases and antioxidant imbalance occurs. New triazole derivatives TF-25, TS-27 and TB-31 alloxan increases the activity of SOD enzyme, which is reduced in the liver under diabetic conditions.

In our experience, the development of an imbalance in the antioxidant system of liver cells has been proven in conditions of alloxan diabetes.

In our next experiment, we determined changes in the activity of another antioxidant enzyme, catalase, in the liver homogenate of a diabetes model and the effect of new 1,2,3-triazole derivatives on them (Fig. 3). It was found that the activity of catalase in the liver tissue of rats with alloxan-induced diabetes is 43.2±3.6 μM/min/mg of protein and decreases by 43% compared with the control.In group III, IV and V diabetic rats treated with alloxan, the enzyme activity decreased by 18.1%, 35.3% and 26.3%, respectively, compared to group II and was close to control values (Fig. 3). Catalase is an enzyme that acts as an antioxidant and plays an important role in the defense system against LPO products. Many studies have found a significant decrease in catalase activity in experimental diabetes (Miaffo *et al.*, 2019; Moodley *et al.*, 2015).

Changes in the activity of enzymes of the antioxidant system in patients with diabetes mellitus are due to the formation of lipid peroxidation and the formation of reactive oxygen species in excess.In particular, catalase is a strong inhibitor of superoxide radical. New triazole derivatives TF-25, TS-27, and TB-31 can restore plasma triglyceride, total cholesterol, and insulin concentrations in alloxan-diabetic rats. Regulators of the antioxidant system SOD and catalase and changes in the release of MDA LPO in diabetic conditions are reliably restored by new triazole derivatives, such as TF-25, TC-27 and TB-31. At the same time, the activity of the triazole derivatives TS-27 of these active substances was slightly higher than that of the derivatives TF-25 and TB-31.

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