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PROOXIDANT PROPERTIES OF LORATADINE AND DESLORATADINE IN THE CHEMICAL SYSTEM OF AUTO-OXIDATION OF ADRENALINE

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Neurodegenerative diseases (NDD) are a major health problem for the elderly. It is known that active forms of oxygen play a certain role in the pathogenesis of NDD.

According to the analysis of literary sources, it was found that some drugs, in particular antihistamines, show additional anti-inflammatory activity, which makes them promise in the treatment of neurodegenerative diseases. It is also known that antagonists of H₁-receptors show an antioxidant effect, inhibiting free radical reactions. Therefore, it is advisable to study the antioxidant activity of the most commonly used antihistamine drugs, such as loratadine and desloratadine. Loratadine is an effective, non-sedating antihistamine drug of the second generation of long-term action with a rapid and pronounced antiallergic effect and selective activity against peripheral H₁-receptors [1]. Desloratadine is a selective antagonist of H₁-histamine receptors of the third generation, which is an active metabolite of loratadine, and has anti-inflammatory and antioxidant properties [2].

The aim of the article: study of the effect of loratadine and desloratadine on the chemical system of autoxidation of adrenaline.

Materials and methods of research. Determination of the activity of loratadine and desloratadine in the chemical system of autoxidation of adrenaline was carried out *in vitro* spectrophotometrically [3]. The absorbance was measured for 7 minutes from the moment of adding low concentrations of adrenaline (230 μM) to the alkaline solution at intervals of 15 s. A 0.2 M carbonate buffer with a pH of 10.65 was used as an alkaline environment. The research was conducted at a temperature of 25.0°C. Kinetic studies were carried out on an OPTIZEN POP UV spectrophotometer (Mesasys, South Korea) with a built-in thermostat (thermostatic accuracy 25.0±0.1°C) in quartz glass cuvettes with an optical layer thickness of 1 cm at a wavelength of λ=347 nm.

Research results. The reaction rate of the formation of an intermediate product of autoxidation of adrenaline in the presence of loratadine (in the first sample) and desloratadine (in the second sample) in concentrations of 25, 50 and 100 μM in the system was investigated. The measurement was performed three times for each concentration. Quantitative expression of reaction rates was carried out by calculating the first-order rate constant (K_n¹) according to Eq. (1):

$$k_n^1 = \frac{1}{t} \cdot \ln \frac{D_\infty - D_0}{D_\infty - D_t} \quad (1)$$

where t – reaction time;

D_∞ – value of the absorbance after the end of the reaction;

D₀ – value of the absorbance at the beginning of the reaction;

D_t – the value of the absorbance at a certain point in time.

It was established that loratadine reliably accelerates the autoxidation reaction of adrenaline by 35%, 45% and 53% at concentrations of 25, 50 and 100 μM, respectively: k_n¹⁽⁰⁾=(1.018±0.590)·10⁻³ s⁻¹; k_n¹⁽²⁵⁾=(1.370±0.028)·10⁻³ s⁻¹; k_n¹⁽⁵⁰⁾=(1.478±0.037)·10⁻³ s⁻¹; k_n¹⁽¹⁰⁰⁾=(1.579±0.021)·10⁻³ s⁻¹ (p≤0,05).

In turn, desloratadine reliably accelerates the auto-oxidation reaction of adrenaline, but not so intensively - by 13%, 15% and 18% at concentrations of 25, 50 and 100 μM, respectively: k¹⁽⁰⁾=(1.018±0.590)·10⁻³ s⁻¹; k_n¹⁽²⁵⁾=(1.152±0.013)·10⁻³ s⁻¹; k_n¹⁽⁵⁰⁾=(1.117±0.013)·10⁻³ s⁻¹; k_n¹⁽¹⁰⁰⁾=(1.204±0.012)·10⁻³ s⁻¹ (p≤0,05).

Conclusions.

1. Loratadine and desloratadine stimulate the formation of superoxide radicals in the chemical system of autoxidation of adrenaline.
2. When the concentration of loratadine increases, the values of the rate constants of the first order of autoxidation of adrenaline increase up to 10%, and therefore this effect depends on the concentration of API.
3. Desloratadine does not so actively stimulate the formation of superoxide radicals in comparison with loratadine, its pro-oxidant effect is 23% less than that of loratadine.

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