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# STUDY OF THE ANTIHYPERTENSIVE ACTIVITY OF THE N-DEZ ALKALOID IN VIVO.

**[1,2]Sobirov S.B.**
**[1]Zaripov A.A.**
**[1]Zhumaev I.Z.**
**[2]Esimbetov A.T.**
**[1]Boboev S.N.**
**[3]Zhuraqulov Sh.N.**
[1]Institute of Biophysics and Biochemistry, under the Uzbekistan National University, Tashkent, Uzbekistan
[2]Karakalpak State University named after Berdakh, Nukus, Uzbekistan
[3]Institute of Chemistry of Plant Substances, Academy of Sciences of the Republic of Uzbekistan, Tashkent, Uzbekistan
**e-mail:** sadriddin-2022@mail.ru
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Arterial hypertension is a common chronic disease of the cardiovascular system; the sustained elevation of arterial blood pressure leads to the development of functional and structural changes in the heart, brain, kidneys, and blood vessels.[1] Various methods are used to model arterial hypertension in experimental studies; among them, inducing a rise in blood pressure via adrenaline administration allows for the short-term assessment of hemodynamic changes in the body. [2,3] The effect of adrenaline on the cardiovascular system is mediated through its interaction with α- and β-adrenoceptors. [4, 5] In particular, studying the antihypertensive properties of biologically active compounds derived from natural sources and plant alkaloids under in vivo conditions is of great importance for evaluating their pharmacological potential. [6]

In this regard, studying the effect of the alkaloid N-deacetyllappaconitine (N-DEZ) on arterial blood pressure in a living organism is of particular scientific interest. Against this background, evaluating the effect of the N-DEZ alkaloid on blood pressure under conditions of adrenaline-induced arterial hypertension using an in vivo approach is considered a significant scientific task. This enables the evaluation of the hypotensive efficacy of these alkaloids by recording changes in systolic and diastolic arterial blood pressure in live rats using the tonometry method.

In the initial stage of the experiments, arterial blood pressure values corresponding to the physiological norm were determined in the rats. In this process, the mean systolic arterial blood pressure (SABP) was.

In this case, systolic arterial blood pressure (SABP) averaged 122.3±12.5 mmHg, while diastolic arterial blood pressure (DABP) was 96.8±9.4 mmHg. To model experimental hypertension, adrenaline hydrochloride was administered to rats at a dose of 0.25 mg/kg. Thirty minutes after the administration of adrenaline into the rat's tail vein, it was observed that in the control group, systolic blood pressure increased from a baseline of 122.3±12.5 mmHg to 152.5±15.1 mmHg, while diastolic blood pressure rose from 96.8 ± 9.4 mmHg to 117.5±11.6 mmHg. High arterial blood pressure, triggered by the effect of adrenaline, persisted and continued to rise over subsequent intervals.

The N-DEZ alkaloid demonstrates an arterial blood pressure-lowering effect in a model of adrenaline-induced experimental hypertension. The effect of the drug was observed as a reduction in


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both systolic and diastolic blood pressure compared to the control group for a duration of 1–3 hours: specifically, to 121.5±12.5 mmHg at a dose of 10 mg/kg, and to 139.8±14.1 mmHg at a dose of 20 mg/kg.

At the same time, the fact that the effect at a dose of 20 mg/kg was not stronger than at 10 mg/kg indicates that the hypotensive effect of N-DEZ under these experimental conditions does not increase in direct proportion to the dose.

Based on the obtained experimental results and the analysis of available literature data, it was assumed that the hypotensive activity of N-DEZ alkaloid may be associated with the modulation of the β-AR–AS–sAMF–PKA reaction cascade in vascular smooth muscle cells, attenuation of adrenergic vasoconstrictor reactions, and reduction of vascular tone.

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