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Influence of Nonspecific Inhibitor of NO-Synthase L-NAME on Electric Characteristics of Premotor Interneurons of Terrestrial Snails

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Abstract
It has been found that the application of nonspecific inhibitor of NO-synthase L-NAME caused the depolarization shift of the membrane potential of premotor interneurons of defensive behavior of terrestrial snails. This effect is opposite to hyperpolarization shift of the membrane potential caused by the action of the nitric oxide donor—sodium nitroprusside.

Keywords Nitric oxide · L-NAME · Identified neurons · Membrane potentials · Snail

1 Introduction

The system of nitric oxide (NO) is one of the most studied systems of the body. However, despite today has accumulated a huge amount of data on the signaling targets of NO, a clear opinion on this matter is missing. NO is intra- and intercellular mediator that performs various signal functions; it is a molecule synthesized in response to physiological need in the cell from L-arginine with the participation of NO-synthase (NOS), activated by increased Ca^{2+} ions [1]. The effects of NO are associated with its action on ion channels, neurotransmitter secretion, calcium ion exchange, the cell's metabolism, and its genome. It is shown that NO plays a role as intercellular messenger and signaling molecule also in mollusks [2]. It is discovered that NO coordinates a number of behavioral programs in mollusks [3, 4]; it is found that NO is involved in the processes of learning and memory [5–8]. NO also controls the plastic properties of neurons: an inhibitor of NOS contributed to the development of habituation, and the NO donors caused the effect of sensitization [9]. It was shown the participation of NO in the plastic changes the synaptic transmission in various systems, including the nervous system of *Helix* [2, 10, 11]. In experiments on preparations of *Helix*, it was shown that NO donors increased the frequency of spikes and reduced the latency of spikes in the identified neurons [12] and that NO is released by two neurons, the cerebral giant cell (CGC) and the B2 buccal motor neuron in the isolated nervous system of the pond snail [13]. Thus, B2 has released more NO than the CGC neuron, but both cells were equally suppressed by the NOS inhibitor L-NAME. Earlier, we found that the NO donor caused the shift of membrane potential of premotor interneurons of defensive behavior of terrestrial snail [14]. Therefore, the aim of this work was to study effects of nonspecific NOS inhibitor L-NAME on the electrical characteristics of premotor interneurons of snail.

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2 Methods

The terrestrial snails *Helix lucorum*, the nervous system of which is well described, were used for the experiments. Before the experiments, the mollusks were in the active state for at least 2 weeks [15]. Analysis of electrical characteristics was carried out in the readily identifiable giant premotor interneurons LPa3 and RPa3 of the withdrawal reflex located in the rostral part of parietal ganglia (description and map in Balaban, 2002 [16]). The isolated nervous system was placed in a saline solution (SS) of the following composition: NaCl 80 mM, KCl 4 mM, $CaCl_2$ 10 mM, $MgCl_2$ 6 mM, $NaHCO_3$