

MEMBRANE DEPOLARIZATION AND THE EXCITATORY ACTION OF GABA AND MUSCIMOL IN SPIDER MECHANOSENSORY NEURONS
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Gamma-aminobutyric acid (GABA) is the main inhibitory neurotransmitter in a variety of animal species. Activation of ionotropic GABA receptors in the mechanosensory afferents entering the vertebrate spinal cord as well as the arthropod central ganglia induces primary afferent depolarization and an increase in membrane conductance (shunting), leading to inhibition. Mechanosensory neurons of the slit-sense organ (VS-3) in the patella of the tropical wandering spider *Cupiennius salei* are innervated by GABAergic efferents and they show similar responses (depolarization, shunting and inhibition) to application of GABA and muscimol, agonists of ionotropic GABA receptors. In the present study we show that when the VS-3 neurons were stimulated with a pseudorandom white-noise mechanical or electrical stimulation during muscimol application, the initial inhibitory response was followed by excitation, lasting for up to ten minutes. The VS-3 neuron spike rate, sensitivity and information capacity all increased during this period. Using intracellular current-clamp recordings, we investigated if membrane depolarization alone could induce similar excitatory effect as muscimol application. When VS-3 neurons were subjected to white noise stimulation while positive current was injected to produce 20-30 mV depolarization, a large increase in the neuron's spike rate, sensitivity and information capacity was observed. To learn if prevention of membrane depolarization during muscimol application would impede the excitatory effect, we used white-noise stimulation when the neurons were loosely voltage-clamped to their resting potential, thus suppressing slow changes in the membrane potential but allowing action potential transmission. Under these conditions muscimol elicited similar effects as under current-clamp conditions. These results suggest that membrane depolarization may contribute to the muscimol induced excitatory effect, but it is possible that another, yet unknown, signaling pathway is also involved.
Funded by: CIHR. NSHRF