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FOCUSED CONFERENCE GROUP: P12 - ION CHANNELOPATHIES: NEW WINDOWS ON COMPLEX DISEASE AND THERAPY

MINOCYCLINE INHIBITS D-AMPHETAMINE-ELICITED ACTION POTENTIAL BURSTS IN A CENTRAL SNAIL NEURON

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Minocycline is a second-generation tetracycline that has been reported to have powerful neuroprotective properties. In our previous studies, we found that d-amphetamine (AMPH) elicited action potential bursts in an identifiable RP4 neuron of the African snail, Achatina fulica Ferussac. This study sought to determine the effects of minocycline on the AMPH-elicited action potential pattern changes in the central snail neuron using the two-electrode voltage clamping method. Extracellular application of AMPH at 0.3 mM elicited action potential bursts in the RP4 neuron. Minocycline (0.3 mM-0.9 mM) inhibited the action potential bursts elicited by AMPH. Forskolin (0.05 mM), an adenylate cyclase activator, and dibutyryl cAMP, a membrane-permeable cAMP analog (1 mM), restored the inhibitory effects of minocycline on AMPH-elicited action potential bursts. Co-administration with forskolin (0.05 mM) plus tetraethylammonium chloride (TEA; 5 mM) or co-administration with TEA (5 mM) and dibutyryl cAMP (1 mM) also elicited action potential bursts, while these effects were inhibited by minocycline. In addition, minocycline prevented forskolin from eliciting action potential bursts at the high concentration of 0.1 mM. Notably, TEA (50 mM) and pentylenetetrazol (PTZ; 50 mM) elicited action potential bursts in the RP4 neuron, but these effects were not affected by minocycline. These results suggest that the cAMP-protein kinase A signalling pathway is involved in the inhibitory effects of minocycline against AMPH-elicited action potential bursts.