MECHANISMS OF LONG-TERM MODULATION OF SYNAPTIC TRANSMISSION IN CRAYFISH

by

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ABSTRACT

Synapses undergo remodeling in response to changes in electrical activity, sensory input and environmental conditions. This thesis focuses on two forms of synaptic plasticity, heat adaptation and long-term adaptation (LTA). Heat adaptation occurs in response to a brief exposure to sub-lethal heat which increases the temperature at which synaptic transmission fails during subsequent heat stress. Long-term adaptation occurs in response to chronic electrical stimulation, which increases the activity of less active (phasic) motoneurons and reduces neurotransmitter output so that the synaptic terminals resemble those of more active (tonic) motoneurons. Previous research has shown long-term adaptation and the synaptic effects of heat adaptation to be calcium dependent. The initial part of this thesis tests the hypothesis that heat adaptation and long-term adaptation share a common mechanism. The present study demonstrated that the induction of LTA through electrical stimulation (conditioning paradigm) does not, by itself, confer heat tolerance. This suggests that the mechanisms underlying these two forms of plasticity are not identical. The present study also examined the calcium channel subtypes which mediate the decreased transmitter output during LTA. Selective calcium channel blockers were used to determine the relative contributions of calcium channel subtypes to recorded postsynaptic potentials. The second part of this thesis tested the hypothesis that a change in the distribution of calcium channel types mediated the synaptic effects seen in long-term adaptation. The induction of LTA did not alter the responsiveness to inhibitors of P-type and L-type calcium channels (which are sub-types of high voltage activated calcium channels located in synaptic terminals). Thus, LTA does not involve a re-distribution of these two calcium channel sub-types.