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Firing pattern regulation in hypothalamic vasopressin neurons: roles of synaptic inputs and retrograde signaling

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Magnocellular neurosecretory cells (MNCs) of the hypothalamus release the hormones oxytocin (OT) and vasopressin (VP) into the blood. These cells demonstrate enhancement of hormone release with bursting patterns of electrical activity. OT neurons fire synchronized bursts at long intervals during parturition and milk ejection; VP neurons generate an asynchronous phasic bursting in response to osmotic and cardiovascular stimuli. The mechanisms of bursting activity in VP are not known completely and are believed to be different *in vitro* and *in vivo*. Whereas *in vitro,* phasic bursting in VP neurons appears to be governed by intrinsic deterministic mechanisms, *in vivo* burst generation and termination significantly depends on synaptic activity. Mounting evidences suggest that retrograde signaling *via* endocannabinoids (eCBs) plays a prominent role in modulating MNC synaptic activity [1]. Our recent experiments suggest that bursts of action potentials are capable of suppressing glutamatergic input in VP neurons. We also found that blocking eCB receptors increased burst duration and intra-burst action potential frequency, consistent with a potential role in burst termination.

To investigate theoretically the role of synaptic inputs in the phasic bursting activity in VP neurons, we used an updated multicompartmental model of the MNC [2]. The model takes into account MNC morphology and electrotonic properties and includes a set of realistic voltage-gated and Ca^{2+}-activated ion currents, compartmental Ca^{2+} dynamics and reproduces several of the hallmark characteristics of MNC electrophysiological properties. Phasic bursting in the model is controlled by both intrinsic and synaptic mechanisms: bursts of action potentials arise from the summation of slow depolarizing afterpotentials superimposed on a tonic background activation of glutamatergic synaptic inputs; activity-dependent release of a retrograde messenger (eCB) from the dendrites of VP neurons attenuates tonic glutamate release and leads to burst termination. Background synaptic activity was simulated as independent excitatory and inhibitory inputs mediated by AMPA and $GABA_A$ conductances. Our computational studies also suggest that $GABA_A$ receptor activation promotes burst firing patterns, and stochastic synaptic inputs play an important role in the modulation of phasic activity in VP neurons.

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