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Dopaminergic modulation of circuit excitability and plasticity in the lateral amygdala: A critical role for NPYergic interneurons?

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The amygdala, a brain structure located in the medial temporal lobe, plays an important role in fear memory acquisition and storage. The basolateral amygdala (BLA) constitutes the main entry site for sensory information from cortical and thalamic inputs to generate fear and anxiety-related behavioral outputs. Moreover, BLA plays a critical role in the response to stress. However, information processing within the amygdala is strongly dependent on inhibitory control, which provides an essential counterbalance to excitatory neurotransmission. Among several neuromodulators released in the amygdala, dopamine (DA) participates in mediating the stress response, modulation of neuronal activity and memory formation by targeting inhibitory circuits in the BLA. Although it has been shown that activation of DA receptors alters BLA neural activity and can gate the induction of plasticity, it is still unclear how DA modulates synaptic transmission and plasticity in the BLA in the presence of inhibition. Using extracellular field recordings in horizontal brain slices we demonstrate that DA at different concentrations (1-100 µM) is unable to enhance amygdala excitability in a significant manner in the presence of inhibition, in contrast to previous studies. Furthermore, we show that DA is indeed able to gate LTP induction in a concentration-dependent manner. These data demonstrate that the GABAergic inhibition present in the BLA exerts direct effects in the dopaminergic modulation of the circuit excitability and plasticity. One of the main inhibitory neuronal population in the BLA is NPYergic interneuronal population that plays a critical role in the amygdalar circuit function. Here, we further demonstrate that D1 receptors are abundantly expressed in NPYergic interneurons providing evidence for gating the dopaminergic modulation of inhibition in the BLA. We are currently generating state-of-the-art viral intervention tools to dissect the role of D1 receptor in the NPYergic intern.