Basal ganglia circuits, social context, and song plasticity

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Speech is a remarkable human skill achieved through vocal imitation and shaped by social interactions. Songbirds provide one of the few animal models for speech learning, and like humans, they possess a network of brain areas specialized for vocal production and plasticity. One of these brain regions is a basal ganglia-thalamocortical circuit critical for song plasticity. To investigate the contributions of this circuit to vocal plasticity, we recorded activity in the cortical output nucleus LMAN, and found that singing-related activity in this circuit is strongly modulated by social context. LMAN neurons switch between precise firing of single spikes when male birds sing to females and variable burst firing when males sing alone (undirected song). Moreover, variability in LMAN activity is associated with variability in song, and lesions of LMAN abolish context-dependent differences in song variability by reducing variability of undirected songs. These results demonstrate that this circuit can actively generate acute variability in song output, a critical component of trial-and-error learning. To further investigate what aspects of LMAN firing are crucial for song plasticity, we disrupted the circuit at the level of the basal ganglia. Lesions of the basal ganglia eliminated temporally patterned burst firing in LMAN during singing and prevented vocal plasticity. Together, these results indicate that patterned bursting in LMAN, which drives acute song variability, is also critical for song motor plasticity. The finding that lesions of this circuit eliminate both acute song variability and slower adaptive changes in song suggest that variability is necessary for plasticity. Variable burst firing in LMAN may enable small changes in song that are reinforced and accumulate, resulting in longer lasting changes in song. Because the basal ganglia-thalamocortical circuit for song is specialized for a single behavior, studies in songbirds are providing general insights into how such circuits function, both normally and in disease.