Ultrasonic vocalization in rats after unilateral dopamine depletion or mild doses of a dopamine receptor antagonist: Towards an animal model of vocalization in Parkinson disease.

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Speech and voice deficits associated with Parkinson Disease (PD) have been well-documented in humans. They are largely resistant to pharmacological and surgical treatment, but do respond to intensive speech treatments. The mechanism underlying this phenomenon is not well-understood and is difficult to systematically test in human models. Thus we turn to the rat as a model, which may be helpful especially if human and rat vocalizations share similar underlying neural events. The purpose of this study is to compare the ultrasonic vocalization (USV) of rats in three conditions: control, haloperidol-induced transient dopamine depletion, and unilateral 6-OHDA induced degeneration of dopamine neurons. A novel recording environment was developed that, for the first time, isolated individual rat USVs within a social environment. The detailed structures of different types of USVs within the-50 kHz range were analyzed. The data suggest that mild transient dopamine depletion with haloperidol or even unilateral degeneration of dopamine neurons are associated with changes in the USV acoustic signal, such as decreased frequency bandwidth. This study provides a foundation to examine the role of dopamine in sensorimotor processes underlying USV production and potentially to explore pharmacological, surgical, and behavioral treatments in dopamine deficient rats.