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Striatopallidal NMDA-receptors control locomotor and conditioning behaviours.

Laurie Lambot[1*], Elena Chaves Rodriguez[1], Yuqing Li[2], Serge N. Schiffmann[1], Alban De Kerchove D'Exaerde[1] and David Gall[1]

[1] Université Libre de Bruxelles, Faculté de Médecine, Belgium
[2] University of Florida, College of Medicine, Department of Neurology, United States

The basal ganglia (BG) are composed of several interconnected nuclei that have a critical role in goal-directed action selection, motor control, and habits. The striatum, the major input site of the BG, is predominantly composed of medium spiny neurons (MSN), which give rise to two projection circuits originating from two distinct populations of MSN: the striatonigral MSN (dMSN) give rise to the direct pathway and the striatopallidal MSN (iMSN) to the indirect pathway. Balanced activity between both pathways is crucial for harmonious functions of the BG. Imbalance of those two neuronal subpopulations is implicated in major neuropsychiatric disorders such as Parkinson and Huntington diseases as well as drug addiction. Despite the increasing knowledge concerning the key role of the striatal glutamate NMDA receptor (NMDA-R) for the BG physiology, it is still challenging to understand the specific functions of striatopallidal neuron NMDA-R. In this study, by a conditional deletion of the essential GluN1 subunit of NMDA-R specificaly in striatopallidal neurons (cKO), the functions of the NMDA-R in the indirect pathway were adressed. At the cellular level, the deletion of GluN1 in iMSN leads to the reduced number and strength of the excitatory corticostriatopalidal synapses and the intrinsic electro-responsiveness of the iMSN was higher, reflecting an homeostatic adaptation partially compensating the reduced synaptic inputs. The behavioral consequence of the NMDA-R absence in iMSN is an aberrant action selection in goal directed behaviour, without alteration of motor learning but changing of motor strategy, as well as alteration in habituation and amphetamine locomotor sensitization. Taken together, these data indicate that the loss of NMDA-R in the striatopallidal neurons disrupts operant and habituation behaviours.

Acknowledgements

L.L. was supported by a FRIA (Fonds pour la formation à la Recherche dans l'Industrie et dans l'Agriculture) PhD fellowship from the Fund for Scientific Research-Fonds de la Recherche Scientifique (FRS-FNRS) (Belgium) and Van Buuren grant from the fonds David et Alice Van Buuren. This study was supported by the Fondation Médicale Reine Elisabeth (Belgium). We would like to acknowledge Dr Frédéric Bollet-Quivogne of the Light Microscopy Facility (LiMiF).

Keywords: NMDA receptor, Neuronal morphology, Striatum, goal-directed behavior, Synaptic Transmission

Conference: 11th National Congress of the Belgian Society for Neuroscience, Mons, Belgium, 22 May - 22 May, 2015.

Presentation Type: Oral or Poster presentation **Topic:** Neuroscience

Citation: Lambot L, Chaves Rodriguez E, Li Y, Schiffmann SN, De Kerchove D'Exaerde A and Gall D (2015). Striatopallidal NMDA-receptors control locomotor and conditioning behaviours.. *Front. Neurosci. Conference Abstract: 11th National Congress of the Belgian Society for Neuroscience.* doi: 10.3389/conf.fnins.2015.89.00074

Received: 30 Apr 2015; **Published Online:** 05 May 2015.

* **Correspondence:** Miss. Laurie Lambot, Université Libre de Bruxelles, Faculté de Médecine, Bruxelles, 1070, Belgium, laurie.s.lambot@gmail.com