Early life stress and social hierarchies: The role of cortico-striatal circuits

ABSTRACT:

Background
Social behaviors are critical to the survival of most animal species. Gregarious behaviors strengthen social groups, reduce stress and increase fitness. However, the genetic and circuit basis of the regulation of social behaviors are not well understood.

Aims
We aimed to dissect the influence of specific genes in social behaviors and their influence in neuronal circuits. One part of our project aimed at discovering, in an unbiased manner, which genetic elements could regulate social subordinance when animals experience early life stress. A second project investigated the role of GPRASP2, a gene known to influence sociability and autism in humans, in order to best understand its role in the brain.

Method
To dissect which genetic elements are responsible to induce social subordination following early life stress, we used a maternal deprivation protocol in C57BL6 mice. We also used a genetically engineered mouse model to understand the role of GPRASP2 in the regulation of social behaviors. For both approaches we employed a range of behavioral, histological and electrophysiological characterization.

Results
This work sheds light on ethologically relevant processes with impact on biomedical sciences. Specifically, we found that the NPYergic system is important in the regulation of cortical circuits and the expression of social subordinate behavior. We also found that loss of GPRASP2 leads to altered social behaviors, deficits in social recognition and increased dominate behavior.

Conclusions
Our work identified novel genetic elements that play a significant role in the regulation of social behaviors.

Keywords
Social behaviour, Social hierarchy, Animal model, Electrophysiology, Neuronal circuits

Published Work:

Franco, L. O., Carvalho, M. J., Costa, J., Ferreira, P. A., Guedes, J. R., Sousa, R., ... Peça, J. (2020). Social subordination induced by early life adversity rewires inhibitory control of the prefrontal cortex via enhanced Npy1r signaling. *Neuropsychopharmacology, 45*, 1438-1447. doi: 10.1038/s41386-020-0727-7

Edfawy, M., Guedes, J. R., Pereira, M. I., Laranjo, M., Carvalho, M. J., Caldeira, G., Gao, X., Ferreira, P. A., Wang, D., Cardoso, A. L., Feng, G., Carvalho, A. L., Peça, J. (2019). Abnormal mGluR-mediated synaptic plasticity and autism-like behaviors in Gprasp2 mutant mice. *Nature Communications, 10*, Article number: 1431. doi: 10.1038/s41467-019-09382-9

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