ABSTRACT

Alzheimer's disease is a notable burden to the contemporary society concerning not only healthcare but also economy. Its aetiology remains unknown, but we know the mechanisms of pathogenesis, which allow us to create animal models (especially mice). In this thesis, we investigate the behavioral phenotype of the frequently used APPswe/PSEN1 murine model. We find that neither six-month-old nor ten-month-old animals show impairment in anxiety (open field test and elevated plus maze) or social behavior tests (three chambre test and social interaction test) compared to controls. However, the prepulse inhibition test revealed that PPI completely disappear in Alzheimer mice, indicating a significant aberration in sensorimotoric gating.

The second part of this thesis focuses on a chemogenetic study of the role of parvalbumin interneurons in Alzheimer's disease and the effect of their excitation on sociability, social memory and sensorimotor gating. Here we use the aforementioned model crossed with Pv-Cre mice, which allowed us to introduce Cre-dependent DREADDs and in this group of animals we investigated social behaviour and the presence of disturbances in sensorimotor gating. However, the results obtained so far do not provide any evidence for an effect of parvalbumin interneuron excitation on animal behaviour, and further experiments with larger numbers of animals will be needed.

Keywords: Alzheimer's disease, behavior, parvalbumin interneurons