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Abstract

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GABAergic system deficit in the prefrontal cortex of Df(h15q13)/+ genetic mouse model of schizophrenia

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Introduction: The 15q13.3 microdeletion is a 1.5 mega-base copy number variation that affects six genes from the human chromosome 15, causing higher risk to develop schizophrenia among other neuropsychiatric disorders. Human carriers of this microdeletion syndrome experience different levels of cognitive dysfunctions; including attention, learning and memory deficits. The GABAergic system is a key regulator of the cognitive functions, therefor GABA deficit might be correlated to the cognitive symptoms of schizophrenia.

Methods: A Df(h15q13)/+ model was recently generated to mimic the 15q13.3 microdeletion. Using this model, we investigate the possible GABA deficit in the prefrontal cortex using behavioral, molecular and structural approaches.

Results: Df(h15q13)/+ mice displayed visuospatial memory deficit in the object location test. Furthermore, a decrease in the protein levels of GAD65 and VGAT in the PFC of Df(h15q13)/+ mice was detected. However, GABA neurotransmitter levels did not change between genotypes.

Conclusion: Results suggest a deficit in the GABA system at the presynaptic level in the prefrontal cortex of Df(h15q13)/+ mice. Further investigations are necessary to correlate these deficits to the other observations from this microdeletion syndrome.