“The role of parvalbumin within the Pvalb interneurons: from firing properties of individual neurons to complex behaviors altered in autism spectrum disorders (ASD)”

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In the last 20 years an increasing number of genetic and chromosomal mutations have been pointed out in association to Autism Spectrum Disorders, and the respective animal models have been created. While none of these models can recapitulate the syndrome, they are helpful in disentangling the endophenotypes of the disorder, and for clarifying its neural bases. Prof Schwaller pointed out the excitatory/inhibitory balance as a major function affected in ASD and demonstrated how congenitally reduced functionality of parvalbumine neurons eventually results in behavioral peculiarities that relate to ASD.

Selected Publications:

1) Filice F¹, Vörckel KJ², Sungur AO³, Wöhr M⁴, Schwaller B⁵. Reduction in parvalbumin expression not loss of the parvalbumin-expressing GABA interneuron subpopulation in genetic parvalbumin and shank mouse models of autism. Mol Brain. 2016

2) Wöhr M¹, Orduz D⁵, Gregory P³, Moreno H⁴, Khan U⁴, Vörckel KJ¹, Wolfer DP⁵, Welzl H⁶, Gall D⁵, Schiffmann SN⁶, Schwaller B⁵. Lack of parvalbumin in mice leads to behavioral deficits relevant to all human autism core symptoms and related neural morphofunctional abnormalities. Transl Psychiatry. 2015