A specific GABAergic synapse onto oligodendrocyte precursors does not regulate cortical oligodendrogenesis

Authors : Maddalena Balia ; Najate Benamer, María Cecilia Angulo ; First published: 10 August 2017Full publication history DOI: 10.1002/glia.23197 View/save citation Cited by (CrossRef): 0 articles

Funding Information

Funding information IDEX-Paris Cité Sorbonne, Grant number: (TransGABA); Agence Nationale de la Recherche (ANR), Grant number: ANR-14-CE13-0023; Fondation pour la Recherche Médicale, Grant number: («Equipe FRM DEQ20150331681»); Fondation pour l'aide à la recherche sur la Sclérose en Plaques (ARSEP); M.B. received fellowships from Université Paris Descartes and Fondation pour la Recherche Médicale (FRM).

Abstract

In the brain, neurons establish bona fide synapses onto oligodendrocyte precursor cells (OPCs), but the function of these neuron-glia synapses remains unresolved. A leading hypothesis suggests that these synapses regulate OPC proliferation and differentiation. However, a causal link between synaptic activity and OPC cellular dynamics is still missing. In the developing somatosensory cortex, OPCs receive a major type of synapse from GABAergic interneurons that is mediated by postsynaptic γ2-containing GABAA receptors. Here we genetically silenced these receptors in OPCs during the critical period of cortical oligodendrogenesis. We found that the inactivation of γ2-mediated synapses does not impact OPC proliferation and differentiation or the propensity of OPCs to myelinate their presynaptic interneurons. However, this inactivation causes a progressive and specific depletion of the OPC pool that lacks γ2-mediated synaptic activity without affecting the oligodendrocyte production. Our results show that, during cortical development, the γ2-mediated interneuron-to-OPC synapses do not play a role in oligodendrogenesis and suggest that these synapses finely tune OPC self-maintenance capacity. They also open the interesting possibility that a particular synaptic signaling onto OPCs plays a specific role in OPC function according to the neurotransmitter released, the identity of presynaptic neurons or the postsynaptic receptors involved.

Lien vers l'article