Analysis of Vti1a and Vti1b double deficiency in neuronal cells

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During embryogenesis crucial steps of the neuronal development are strictly regulated by intra and extracellular signals therefore endosomal transport has emerged as a modifier of signaling. SNARE proteins play a central role in these trafficking processes. Vti proteins belong to the Qb-SNARE family and are involved in the neuronal development during the embryonal stage. They are conserved from yeast to humans. A double deficiency of the mouse homologues Vti1a and Vti1b was created in previous work. A single *knock-out* of one of the *Vti* genes is not lethal whereas the lack of both proteins leads to a perinatal death due to neuronal cell death and defects in neurite outgrowth. In this work, we present investigations into neuronal cells of double *knock-out* (DKO) mouse neurons via immunocytochemistry and other techniques to show differences in neurite morphology and postsynaptic densities. Recently, we were able to present that the Golgi morphology in DKO neurons is changed compared to double heterozygous (DHET) littermates. Previously published in: Emperador-Melero J et al. (2018) Nat Commun 9, 3421. In this work we could go further into details of these differences. To deepen the understanding of the molecular mechanisms that underlie the genetic defects we analyzed a variety of signaling pathways.