

Poster presentation

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Maintenance of entorhinal projection in the dentate gyrus despite massive loss of virus infected major target neurons

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from Infectious diseases of the nervous system: pathogenesis and worldwide impact
Paris, France. 10–13 September 2008

Published: 23 September 2008

BMC Proceedings 2008, 2(Suppl 1):P71

This abstract is available from: <http://www.biomedcentral.com/1753-6561/2/S1/P71>

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Axons undergo structural changes after depletion of their target neurons. Infection of newborn Lewis rats with Borna disease virus (BDV) results in a progressive selective loss of dentate granule cells which are the major targets of axons arising from entorhinal cortex. Here we examined whether other neuronal cell types in the dentate gyrus are affected by BDV infection and whether the loss of granule cells affects the distribution pattern of entorhinal projection. Newborn Lewis rats were infected with BDV at birth. At various time points post infection (21, 42, 63 days p.i.) the entorhinal projection was anterogradely DiI traced. At the same time points, immunocytochemistry to detect calbindin (granule cells) or parvalbumin (PARV), a marker for GABAergic interneurons in the dentate gyrus, was performed. At 21 days p. i. a typical v-shaped granule cell layer had formed containing numerous PARV-immunopositive neurons. An entorhinal projection had developed similar to non-infected hippocampi. At 42 days p.i. the distribution of DiI-labelled entorhinal axons was not altered, although dendritic alterations were observed both in PARV-labelled neurons as well as in granule cells. In addition, the granule cell layer was less compact, indicating the onset of neuronal loss. Nine weeks p. i. the density of PARV-immunostained dendrites appeared to be reduced and only few granule cells were found. Despite this loss of typical target dendrites, the entorhinal projection persisted as in control hippocampi. This maintenance of entorhinal axons in their correct termination

layer was also observed in BDV-infected organotypic entorhino-hippocampal co-cultures, which are a model to mimic BDV-mediated pathogenesis. Thus, neonatal BDV infection does not exclusively affect granule cell viability but also results in dendritic changes of interneurons in the dentate gyrus. Our data suggest that, although presynaptic entorhinal afferents are maintained, postsynaptic changes of excitatory and inhibitory target neurons may alter the balance of neuronal activity, which may be related to granule cell death.

Supported by the DFG: Sch 632, He 1520.