

Sensory deprivation in one modality can enhance the development of the remaining modalities via mechanisms of synaptic plasticity. Mice of C3H strain suffers from RD1 retinal degeneration that leads to visual impairment at weaning age. Independently on the retinal degeneration there is also present olivocerebellar degeneration caused by Lurcher mutation. This neurodegenerative disorder causes motor deficits, increased CNS excitability as well as changes in synaptic plasticity.

The aim of this study was to evaluate a role of whiskers in compensation of the visual deficit and to assess the influence of the olivocerebellar degeneration on this process. To differentiate contribution of the whiskers from other mechanisms that can take part in the compensation, we investigated the effect of both chronic and acute tactile deprivation. We focused on motor skills (rotarod, beam walking test), gait control (CatWalk system), spontaneous motor activity (open field) and the CNS excitability (audiogenic epilepsy).

In the seeing mice without olivocerebellar degeneration, the removal of the whiskers had no effect. In the blind animals without olivocerebellar degeneration, chronic tactile deprivation caused changes in gait and impaired the performance in motor tests. Some other compensatory mechanisms were involved but the whiskers are essential because the acute removal of the whiskers revealed more marked change of gait and the worsening of the motor performance. Both chronic and acute tactile deprivation induced anxiety-like behaviour. Only a combination of blindness and chronic tactile deprivation led to an increased sense of hearing.

In both seeing and blind mice suffering from olivocerebellar degeneration, chronic as well as acute tactile deprivation influenced performance in the motor tests and spontaneous motor activity. This finding suggests that neurodegenerative process impairs not only cross-modal plasticity but also sensorimotor integration. Method of audiogenic epilepsy revealed increased sensitivity of hearing in the blind chronically tactile deprived mice. This finding could be explained with increased CNS excitability in the Lurcher mice.