Mice with targeted mutations have been studied in order to understand mechanisms of complex behaviours. Molecular dissection of behavioural phenotypes, such as brain function and learning and memory, is made possible by this approach. By general behavioural test patterns, information about possible behavioural changes caused by modified genes is achieved.

In this pro gradu work I studied the effects of N-syndecan deficiency on the behaviour of mice. N-syndecan is a transmembrane heparan sulphate proteoglycan that is expressed in the central nervous system of vertebrates. The function of N-syndecan is not yet fully understood. N-syndecan is a co-receptor for HB-GAM (Heparin Binding Growth Associated Molecule) suggesting involvement in the regulation of neurite outgrowth in neuronal cells. N-syndecan is mainly expressed in newly born animal brains and therefore it is suggested that N-syndecan is involved in the regulation of axonal outgrowth in developing brain tissue. Recent studies also show that N-syndecan is a factor in the stabilisation of long term potentiation in adult rat hippocampus. The major opinion is that long term potentiation is an important factor in the formation of memories.

The aim of this study was to define the behavioural phenotype of N-syndecan deficient mice. The behaviour of the mice was measured in a general test pattern. The tests were performed on 12 N-syndecan knockout mice of which 6 were female and 6 male. 8 wildtype mice, 4 female and 4 male, served as the control group. Both wildtype and knockout mice were developed from 129 Sv mice bred in C57Bl/6 mice.

The tests started with a health control. The gross neurological function of the mice was tested in a general reflex control, containing control of the visual cliff behaviour, the approach reflex and the ear twitch reflex. Additionally the whisker orienting reflex was controlled on mice with long enough whiskers. The anxiety and exploration rates of the mice were measured in the light-dark box, the elevated plus-maze and the open field. Locomotor activity was measured in a spontaneous locomotor activity test and in the open field. Balance and co-ordination were measured in the rota-rod test and in the string test. In the forced swimming test the behavioural despair of the mice was measured. The ability to feel pain was controlled in the hot plate test and the vision abilities of the mice were measured in the visible platform training blocks of the water maze test. The learning and memory abilities of the mice were measured in the “Morris” water maze test.

We found no statistically significant effect of N-syndecan deficiency on the behaviour of mice. All mice were healthy and showed no signs of gross neurological impairment. The locomotor activity of the mice groups were similar and also the balance and co-ordination seemed to be unaffected by the lack of N-syndecan. The sense functions of the mice groups were similar. The behavioural despair test showed no effect of the N-syndecan deficiency on despair behaviour. Some parameters in the elevated plus-maze showed a trend of lesser anxiety in wildtype mice; however, this difference was not statistically significant. In the “Morris” water maze the N-syndecan knockout mice showed a trend of slower learning but this difference was not statistically significant either.

The amount of mice available to a study like this is limited due to both technical and ethical reasons. Due to the small amount of animals in this study the statistical analyses were aggravated. Additional extensive studies are required to get reliable results of the effect of N-syndecan deficiency on behaviour of mice. Linkage of genotype to behaviour is difficult and the results of this study should be considered as guidelines for future studies in the behaviour of mice with modified expression of N-syndecan.