Natural learning in mouse populations: food place reversal in mice overexpresssing the cell adhesion molecule L1

Vyssotski,A.L. (1), Dell'Omo,G. (1), Vyssotski,D.L. (1),
Wolfer,D.P. (1), Schachner,M. (2), Poletaeva,I. (3), Lipp,H.P. (1)

(1) University of Zürich, Institute of Anatomy, CH-8057 Zürich, Switzerland

(2) Zentrum für Molekulare Neurobiologie, University of Hamburg

(3) Laboratory of Physiology & Genetics of Behavior, Moscow State University

Transgenic mice overexpressing the neural cell adhesion molecule L1 are known to adapt quicker to new situations such as platform reversal in the water maze. In order to test mouse learning abilities under more naturalistic conditions, we have developed computerized set-ups that permit to monitor simultaneously the spatial behavior of many transponder-tagged mice living in large outdoor pens. In this study, we tested whether L1 transgenic mice would also show superior place reversal learning following relocations of distant food sources. 77 mice were tagged wi th transponders and then released into an outdoor pen (20 x 20 m) for 67 days Eight computer-controlled antennas recorded every visit of a microchip-tagged mouse during the entire observation period. Mice lived in two central shelters of 2x2m, each one containing one antenna. The other six antennas were placed in the open area of the pen at variably distant locations. Food was delivered for 19 days inside the shelters. After this, the location of food delivery was changed several times and we observed the adaptation of the mice by recording the order and the number of visits at rewarded and empty sites. After stopping food delivery inside the shelters, the L1-overexpressing mice increased their searching activity significantly more than wildtype mice ($p < 0.01$), at least for some of the antennas. In addition, transgenic mutants returned to the previous food locations less frequently ($p<0.02$). Thus, testing reversal learning under naturalistic conditions confirms the predictions based on water maze learning.

Supported by: NCCR on Neural Plasticity and Repair, SNF