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Englische Zusammenfassung der Dissertation mit dem

Thema: **Die Konsequenzen temporärer Inhibitionen der *Nuclei amygdalae medialis* auf das unkonditionierte, soziale Langzeit-Wiedererkennungsgedächtnis von Mäusen**

Individual recognition of conspecifics is a prerequisite for complex behaviours in socially living animals. Rodents use “olfactory signatures” to recognize each other individually. Under laboratory conditions, non-conditioned social recognition memory is investigated with the social discrimination test. This task consists of two sessions. During the first session (sampling), the experimental subject is allowed to acquire the “olfactory signature”. 24 hours later, long-term social recognition memory is tested in a second session (choice). Mice of different laboratory strains are able to form a long-term recognition memory for conspecifics. As described in a number of studies, processing of “olfactory signatures” seems to involve several brain areas linked to both the main and the accessory olfactory system – but in particular the *Nucleus amygdalae medialis* (NAMe). The present study was designed to investigate how (propagation and/or processing of signals/information) and when (during acquisition, consolidation, recall) the NAMe is involved in social recognition memory. This was intended to be achieved by bilateral injections of lidocaine, muscimol and anisomycin into the NAMe at different defined points in time during the social discrimination test.

An initial series of tests proved that important preconditions are given. At first, both male and female juveniles are similarly suitable as stimulus conspecifics. Furthermore, experimental subjects fully recovered from the surgery for the implantation of guide cannulaes at postoperative day seven and thus could be used for behavioural testing. Additionally, neither the placement of the guide cannulaes nor the injection procedure influenced the recognition memory of the experimental subjects. Finally, the substances injected during the main experiments reached the cells of the NAMe.

Moreover, an immunohistochemical analysis revealed an increase of c-Fos synthesis in both the NAMe and the *Cortex piriformis* (Pir) after the presentation of the volatile fraction of the “olfactory signature” indicating a role of both brain areas for the processing of olfactory stimuli. This does not rule out the possibility that the c-Fos synthesis is a neuronal correlate for an emotional response upon the social stimulus having no relevance for long-term recognition memory.

Already the injection of the control substance NaCl-solution into the NAMe under defined conditions impaired long-term recognition memory. NaCl-solution might have influenced the extracellular ion composition and thus disturbed the adequate propagation of information. The injection of muscimol three hours before sampling impaired long-term recognition memory. Muscimol might have had a modulatory effect upon the propagation of information, however, the remaining neuronal activity seemed to be sufficient for memory recall after the injection three hours before choice. Anisomycin injection into the NAMe both three hours before sampling or three hours before choice impaired long-term recognition memory. One can not exclude the relevance of protein synthesis in the affected brain area, especially as the injection of anisomycin into the NAMe also inhibited the protein synthesis in the Pir. Nevertheless, it is more likely that anisomycin pharmacologically influenced local neurochemical mechanisms. Thus, anisomycin, similar to NaCl-solution and muscimol respectively, generated changes in the local propagation of information thereby probably affecting the function of other brain areas relevant for recognition memory. Due to experimental results and theoretical considerations indicating experimental limitations, the data obtained with injections of lidocaine seemed to be the most reliable: Long-term recognition memory was impaired only when lidocaine was injected directly before choice. Thus, the affected area, in particular the NAMe, is seemingly not involved in the propagation of signals during the acquisition or consolidation of olfactory information important for recognition memory, but likely to act as an essential relay station for information during recall of long-term social recognition memory.