Acknolegment

I would like to thank my supervisors, Professor Matthias Laska and Postdoctoral fellow David Willhite, for their assistance and support during this study. Thanks also to Professor Gordon Shepherd and the other members of the Shepherd Laboratory at Yale.

Contact info:

Email:
eribo088@student.liu.se

Olfactory and cognitive abilities in two strains of Alzheimer's disease model

Linköpings universitet

Yale school of medicine

Final Thesis Masters Programme Applied Ethology and Animal Biology

By: Erik Boman

Background

Alzheimer's disease (AD) is a progressive and irreversible neurodegenerative disorder. It affects about 10% of people at the age of 65 and about 50% of people at the age of 85. It is the most common form of dementia and causes abnormal changes in the brain which worsen over time and interfere with many aspects of the brain.

As the disease progresses persons affected with AD suffer from cognitive and sensory impairments, widespread loss of mental abilities and ultimately death. One of the earliest symptoms of AD in humans is an olfactory impairment which is currently used, among other criteria, to diagnose human AD.

In this study I used two strains of AD model mice which overexpress proteins that are implicated in the neuroanatomical changes which characterize AD.

The Aim

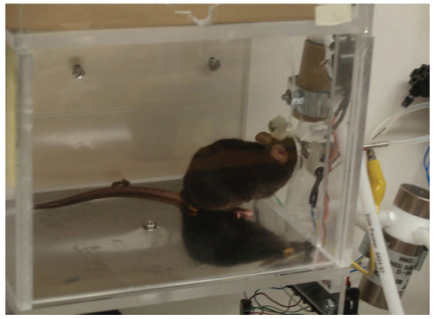
To assess olfactory and cognitive abilities in two strains of Alzheimer's disease model mice and in healthy control mice over a time period.

To learn more about how the neuroanatomical changes in the brain caused by AD and the observed cognitive and olfactory impairments are linked.

Results

There were no systematic differences in olfactory performance of AD model mice and the control mice across the testing period.

There was no indication of an age-related decline in performance in any of the mouse strains across the testing period.



When the mouse inserst its snout into an opening of the chamber it is presented with an odor.

Discussion

The lack of difference in performance between the mouse strains tested in the present study might be due to the mice not having been tested long enough as the AD-related impairments have to develop over time.

It was expected that the AD model mice would display an impairment in olfactory performance at least during the end of the study. Several hypothetical reasons may account for the findings that this did not occur:

1. The mice do not develop the proteins typical of AD at all.
2. The mice will develop the proteins typical of AD later on.
3. The mice may have already developed these proteins but these neuroanatomical changes do not, or not yet, affect their olfactory performance.
4. Animal models of a human neurodegenerative disease do not necessarily show the same pattern of symptoms as the humans themselves.
5. The tests used in the present study might not be sensitive enough to detect changes in olfactory performance and/or spatial learning and memory.