Microglia in the olfactory bulb of Parkinson's and Alzheimer's patients.

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(see also overlap with projects from Van Dam, vdBerg, Swaab)

The olfactory bulb (OB) is affected early in both Parkinson's (PD) and Alzheimer's disease (AD), evidenced by the presence of disease-specific protein aggregates and an early loss of olfaction. Whereas previous studies showed amoeboid microglia in the classically affected brain regions of PD and AD patients, little was known about such changes in the OB. Using a morphometric approach, a significant increase in amoeboid microglia density within the anterior olfactory nucleus (AON) of AD and PD patients was observed. These amoeboid microglia cells were in close apposition to β-amyloid, hyperphosphorylated tau or α-synuclein deposits, but no uptake of pathological proteins by microglia could be visualized. Subsequent analysis showed (i) no correlation between microglia and α-synuclein (PD), (ii) a positive correlation with β-amyloid (AD), and (iii) a negative correlation with hyperphosphorylated tau (AD). Furthermore, despite the observed pathological alterations in neurite morphology, neuronal loss was not apparent in the AON of both patient groups. Thus, we hypothesize that, in contrast to the classically affected brain regions of AD and PD patients, within the AON rather than neuronal loss, the increased density in amoeboid microglial cells, possibly in combination with neurite pathology, may contribute to functional deficits.