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Oral presentation

Cognitive impairment in Parkinson's disease: limbic dementia?

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Objective
To examine the neuroanatomical background of cognitive decline in long-lasting Parkinson's disease (PD).

Background
Previous studies reported an association between cortical Lewy body (LB) formation and dementia in PD. However, it is unclear whether cognitive decline in this disorder is related to specific patterns of LB distribution within the cerebral cortex. Moreover, the prediction of cognitive status based on concomitant assessment of LB and Alzheimer disease lesions has led to conflicting results.

Design
Clinicopathologic study in 22 elderly cases with PD patients in whom parkinsonism preceded cognitive decline by at least 3 years. Cognitive status assessed prospectively using the Clinical Dementia Rating scale (CDR); quantitative assessment of LB, neurofibrillary tangles (NFT), and senile plaques (SP) was performed in Brodmann areas 9, 21, 24, 41 and the entorhinal cortex. Statistical analysis was performed using both correlation coefficients and logistic regression models.

Results
There was a highly significant correlation between CDR scores and regional LB scores in the entorhinal and area 24. LB and SP densities in the entorhinal cortex accounted for 36.2% and 19.3% of the variability in CDR scores. LB densities in area 24 could explain 25.2% of this variability. NFT densities did not predict cognitive status. In multivariate models only LB densities in the entorhinal cortex and anterior cingulate cortex were significantly associated with CDR scores and explained 36.8% and 25.7% of its variability.

Conclusions
These results imply that an assessment of LB pathology limited to the entorhinal cortex and area 24 may be sufficient to predict cognition in PD. They also suggest that LB formation in limbic areas may be crucial for the development of PD dementia.