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The pathology of Locus Coeruleus nuerons in parkinson's disease and parkinson's disease with dementia

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Parkinson's disease (PD) is the second most common neurodegenerative disorder, characterised by motor symptoms in sufferers. There is a well-established link between motor symptoms and the reduced supply of the neurotransmitter dopamine (DA) due to the loss of midbrain dopaminergic (DA-) neurons alongside increased Lewy pathology. PD patients may also develop non-symptoms with dementia highly prevalent in individuals (~80% of PD sufferers). PDD is not explained by a sole deficit in the neurotransmitter dopamine and the pathology changes associated with non-motor symptoms remain elusive in PD. Emerging evidence suggests that another neurotransmitter, noradrenaline (NA) which the locus coeruleus (LC) is a chief supplier in human brains, may be affected in PDD. The aim of this study is to examine the severity of LC pathology in PDD, PD and healthy age-matched controls. Immunohistochemistry with antibodies against tyrosine hydroxylase (NA-neuronal marker), alpha-synucleinopathy and phosphor-tau was applied in the rostrocaudal planes of the LC using post-mortem human brain tissue. We hypothesize that specific regional and cell type degeneration in the LC will be more severe in PDD due to the projections from these LC regions to prefrontal cortex and hippocampus. Our study has confirmed that the pathological deterioration of the LC in PD compared to controls is obvious, revealed by; reduced NA-neuronal number and increased α-synuclein deposition. We further reveal significant differences between PD and PDD with even greater amounts of α-synuclein pathology and a more substantial reduction in the number of NA-neurons at caudal levels of the LC in PDD.

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