

Anhedonia as an early clinical feature of frontotemporal dementia

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State of the art: Mounting evidence indicates severe motivational disturbances in behavioural variant frontotemporal dementia (bvFTD) leading to a constellation of poor patient and carer outcomes. Here we present new findings demonstrating the importance of anhedonia (loss of pleasure) as an early clinical feature of the bvFTD syndrome. We show that anhedonia is dissociable from apathy and depression, and is highly predictive of functional decline in bvFTD.

Methodology: We recruited 172 participants, including 87 FTD, 34 Alzheimer's disease, and 51 healthy older control participants, who completed measures of anhedonia (SHAPS), motivation (CBI-Motivation) and mood (DASS-Depression). Voxel-based morphometry explored associations between whole-brain grey matter atrophy and outcomes on the three measures using a transdiagnostic approach.

Results: Relative to Controls, FTD patients displayed severe anhedonia (SHAPS; $p < .001$) and apathy (CBI-R; $p < .001$), while Alzheimer's patients showed apathy (CBI-R; $p < .001$) in the absence of anhedonia (SHAPS; $p = .99$). Anhedonia was associated with degeneration of frontostriatal regions including bilateral medial/OFC, insula and putamen. This anhedonia circuit was distinct from that underpinning apathy, with only a small region of overlap in the right OFC. Finally, regression analyses in a subset of patients revealed that anhedonia predicted functional impairment exclusively in bvFTD, whereas apathy emerged as the sole predictor of functional impairment in Alzheimer's disease.

Conclusion: Our studies are the first to demonstrate profound anhedonia in FTD mediated by distinct neural correlates to that of apathy or depression. These findings highlight an overlooked behavioural feature of the bvFTD syndrome with crucial implications for patient and carer wellbeing.

Conflicts of interest

No conflict of interest.