Rapporti tra disturbi di personalità, psicopatie e demenza frontotemporale

REGIONE CALABRIA
DSM REGGIO CALABRIA - ASP 5
SERVIZIO SEMIRESIDENZIALE
(Day Hospital – Centro Diurno - Centro UVA)
Dirigente Responsabile: A. Nucera

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Interpersonal traits change as a function of disease type and severity in degenerative brain diseases
Marc Sollberger,1,2,3,4 John Neuhaus,5 Robin Ketelle,1,2 Christine M Stanley,1,2
Victoria Beckman,1,2 Matthew Growdon,1,2 Jung Jang,1,2 Bruce L Miller,1,2
Katherine P Rankin1,2

ABSTRACT

Background Different degenerative brain diseases result in distinct personality changes as a result of divergent patterns of brain damage; however, little is known about the natural history of these personality changes throughout the course of each disease.

Objective To investigate how interpersonal traits change as a function of degenerative brain disease type and severity.

Methods Using the Interpersonal Adjective Scales, informant ratings of retrospective premorbid and current scores for dominance, extraversion, warmth and ingenuousness were collected annually for 1 to 4 years on 188 patients (67 behavioural variant frontotemporal dementia (bvFTD), 40 semantic dementia (SemD), 81 Alzheimer’s disease (AD)) and 65 older healthy controls. Using random coefficient models, interpersonal behaviour scores at very mild, mild or moderate-to-severe disease stages were compared within and between patient groups.

Results Group-level changes from premorbid personality occurred as a function of disease type and severity, and were apparent even at a very mild disease stage (Clinical Dementia Rating¼0.5) for all three diseases. Decreases in interpersonal traits were associated with emotional affiliation (ie, extraversion, warmth and ingenuousness) and more rigid interpersonal behaviour differentiated bvFTD and SemD patients from AD patients.

Conclusions Specific changes in affiliative interpersonal traits differentiate degenerative brain diseases even at a very mild disease stage, and patterns of personality change differ across bvFTD, SemD and AD with advancing disease. This study describes the typical progression of change of interpersonal traits in each disease, improving the ability of clinicians and caregivers
Abstract
Premorbid personality characteristics could have a pathoplastic effect on behavioral symptoms and personality changes related to neurodegenerative diseases. Patients with personality disorders, in particular of the dramatic cluster, may present functional frontolimbic abnormalities. May these neurobiological vulnerabilities linked to a premorbid personality disorder predispose or represent a risk factor to subsequently develop a neurodegenerative disorder? Are subjects with personality disorders more at risk to develop a dementia than mentally healthy subjects? This topic is discussed presenting the clinical case of a patient who suffered of a probable Narcissistic Personality Disorder and subsequently developed a clinically diagnosed Frontotemporal Dementia.
Abstract
Modern developmental psychology tends to draw a positive, resource-based picture of human aging. We will however focus on more difficult aspects of personality in old age which are of psychiatric relevance: the persistence of cluster A and C personality disorders, antisocial personality in the elderly; the interaction of personality and a detection of mild cognitive impairment (MCI); personality features as risk or protective factors or early signs of Alzheimer's dementia; changes of personality in Parkinson's disease and frontotemporal dementia. We will briefly mention recent neuroimaging studies which appear to suggest a functional neuroanatomy of personality. A quote from Cicero's cato major, de senectute indicates that some of his perceptions regarding classic personality characteristics of the elderly can be recognized in our patients and can be prevented or treated with modern interventions.
Background
The neurobiological basis of personality is poorly understood. Frontotemporal lobar degeneration (FTLD) frequently presents with complex behavioural changes, and therefore potentially provides a disease model in which to investigate brain substrates of personality.

Aims
To assess neuroanatomical correlates of personality change in a cohort of individuals with FTLD using voxel-based morphometry (VBM).

Method
Thirty consecutive individuals fulfilling consensus criteria for FTLD were assessed. Each participant’s carer completed a Big Five Inventory (BFI) questionnaire on five key personality traits; for each trait, a change score was derived based on current compared with estimated premorbid characteristics. All participants underwent volumetric brain magnetic resonance imaging. A VBM analysis was implemented regressing change score for each trait against regional grey matter volume across the FTLD group.

Results
The FTLD group showed a significant decline in extraversion, agreeableness, conscientiousness and openness and an increase in neuroticism. Change in particular personality traits was associated with overlapping profiles of grey matter loss in more anterior cortical areas and relative preservation of grey matter in more posterior areas; the most robust neuroanatomical correlate was identified for reduced conscientiousness in the region of the posterior superior temporal gyrus.

Conclusions
Quantitative measures of personality change in FTLD can be correlated with changes in regional grey matter. The neuroanatomical profiles for particular personality traits overlap brain circuits previously implicated in aspects of social cognition and suggest that dysfunction at the level of distributed cortical networks underpins personality change in FTLD.
Background: Behavioural disturbances are common features in neurodegenerative disease but their neural correlates have only recently explored. We investigated the grey and white matter neural correlates of disinhibition via neuropsychological and carer information in a sample of behavioural variant frontotemporal dementia (bv-FTD) and Alzheimer’s disease (AD) patients, to establish neuroanatomical markers of this behavioural diagnostic feature.

Methods: We employed the Hayling test of inhibitory functioning and a carer questionnaire (CBI - Cambridge Behavioural Inventory) as measures of disinhibition. Mean and overlap-based statistical analyses on selected test variables were conducted to investigate profiles of performance in bvFTD, AD patients and controls. Hayling and CBI scores were entered as covariates in a grey matter voxel-based morphometry (VBM), as well as in a white matter diffusion tensory imaging (DTI) analysis to determine the grey and white matter neural correlates of disinhibition.

Results: Not surprisingly, bvFTD patients showed more disinhibition on both behavioural measures in comparison to AD patients and controls. VBM results revealed that atrophy in orbitofrontal/subcallosal, medial prefrontal cortex and anterior temporal lobe areas covaried with both neuropsychological and carer disinhibition measures. In addition, DTI analysis revealed that white matter integrity fractional anisotrophy (FA) values of the white matter tracts connecting the identified grey matter regions, namely uncinate fasciculus, forceps minor and genu of the corpus callosum, correlated with the disinhibition measures of the Hayling test.

Conclusions: To our knowledge, this is the first study identifying the grey and white matter structures related to disinhibition in bvFTD and AD. Further, we find converging evidence across neuropsychological and carer information that the orbitofrontal/subcallosal brain region is critical for inhibiting prepotent responses. Identification of atrophy in this region may allow better clinical identification of disinhibition in neurodegenerative conditions.
Fig. 2  Personality change data in participants with frontotemporal lobar degeneration and controls.

BFI, Big Five Inventory; E, extraversion; A, agreeableness; C, conscientiousness; N, neuroticism; O, openness.
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<th>Trait</th>
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<th>Relatively preserved grey matter</th>
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<td>Neuroticism</td>
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<td>Extraversion</td>
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a. Empty cells indicate no grey matter correlates were identified at the voxel-wise (P < 0.001 uncorrected) and cluster extent (60 voxel) thresholds used.
Patients with neurodegenerative disease show distinct patterns of personality change, some of which may be traced to focal neurologic damage, while others may be mediated by cultural reactions to functional impairment. While such changes are early and pervasive in behavioral variant frontotemporal dementia (bvFTD), and milder changes are seen in Alzheimer’s (AD), no study has examined all Big 5 factors of personality in mild cognitive impairment (MCI) patients. Also, the influence of culture and ethnicity on disease-related personality changes has seldom been examined. Premorbid and current personality were measured in 47 Greek patients with bvFTD, AD, and MCI according to informant reports using the TPQue5, a 5-factor inventory in the Greek language and accounting for Greek cultural factors. bvFTDs showed greater decreases in conscientiousness than ADs and MCIs. ADs and MCIs showed increased neuroticism, while the bvFTD patients were rated as having become much less neurotic in the course of their disease. The pattern of personality change in MCIs was very similar to that of ADs, supporting recent evidence that personality changes occur as early as the MCI disease stage. In all groups, personality changes were similar to those previously described in non-Mediterranean cultures, supporting the hypothesis that they may result directly from disease-specific neurologic processes.
Beyond cognitive reserve: Behavioural reserve hypothesis in Frontotemporal Dementia
Enrico Premia, Valentina Garibotto, Stefano Gazzina, Mario Grassi, Maura Cosseddu, Barbara Paghera, Marinella Turla, Alessandro Padovani, Barbara Borroni

Background: The brain reserve hypothesis posits that there are individual differences in the ability to cope with brain pathology, and that brain damage extent and clinical symptoms are not tightly linked. If cognitive reserve hypothesis has been demonstrated in Alzheimer Disease and Frontotemporal Dementia (FTD), no evidence of reserve mechanisms on behavioural disturbances has been corroborated yet. In FTD, distinct behavioural phenotypes may be identified.

Objective: To test the behavioural reserve hypothesis in behavioural variant FTD (bvFTD).

Methods: As previously demonstrated, bvFTD patients were grouped into four behavioural phenotypes, i.e. “disinhibited”, “apathetic”, “language”, and “aggressive”, by means of Confirmatory Factor Analysis on behavioural assessment. Educational achievement was considered as proxy measure of reserve on behavioural disturbances, and cerebral SPECT as an indirect expression of brain pathology. On each group, the effect of education on brain damage was assessed by slope analysis.

Results: A specific effect of education attainment on “disinhibited” phenotype was observed, the higher the education, the greater the hypoperfusion in the right inferior frontal gyrus and the left medial frontal gyrus and right caudate (P < 0.001). On the other behavioural phenotypes, no effect of education was reported in modulating brain damage.

Conclusions: We suggest that in neurodegenerative diseases the concept of brain reserve might be extended, as compensatory mechanisms are in action not only for cognitive deficits but for behavioural disturbances as well.
In the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5), the behavioral variant of major frontotemporal neurocognitive disorder (bvFT-NCD) is subclassified into “probable bvFT-NCD” or “possible bvFTNCD.” When genetic evidence is unavailable, cases without clinical neuroimaging are subclassified into “possible bvFT-NCD,” whereas cases whose clinical images show the typical characteristics are subclassified into “probable bvFT-NCD.” Thus, the cases that meet the diagnostic criteria of bvFT-NCD based on their symptoms, but lack the neuroimaging characteristics, fall between the two categories of probable and possible bvFT-NCD. These cases herein are defined as “unclassified bvFT-NCD,” and the present study aims at considering an appropriate diagnostic approach to such cases, that is, whether unclassified bvFT-NCD should be included in bvFT-NCD as a third subcategory, or whether it should be classified into diseases other than bvFT-NCD.

**Conclusions:** To establish clinical diagnostic criteria for unclassified bvFT-NCD, accumulation of cases and evidence will be required along with longitudinal observation using various diagnostic technologies and post-mortem examination.

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