BRAIN COMMUNICATIONS

LETTER TO THE EDITOR

The multifactorial nature of social cognition in neurodegenerative disorders—Response to: The interplay of social cognition subdomains in frontotemporal dementia

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We read with interest the letter to the editor by Van den Stock et al.[1] regarding our recent study exploring the neural substrates of moral reasoning in the behavioural variant of frontotemporal dementia (bvFTD).[2] Our findings highlight the interplay between emotional and conceptual processes in responding to highly conflictual personal moral dilemmas, likely supported by the uncinate fasciculus. Van den Stock et al.[1] build on our findings by providing a re-analysis of previously published work, offering further insights into the relationships between different domains of social cognition at the psychological and neural level.

In their letter, Van den Stock et al.[1] uncovered associations between utilitarian decisions on personal high-conflict moral dilemmas and performance on a theory of mind task in bvFTD. No associations were found, however, between the theory of mind performance and 'emotional conflict' towards the dilemmas, as measured by reaction time. The authors propose that impairment in a single 'G-like' common factor may underlie deficits across social cognition subdomains in bvFTD. This proposal is intriguing and could potentially account for the pervasive and cross-modal socioemotional disturbances typically seen in bvFTD.[3]

Despite its broad appeal, we note a number of inconsistencies in the literature that seem difficult to reconcile under such a hypothesis. First, not all social cognitive subdomains are uniformly impaired in bvFTD. In both our moral reasoning study and the original 2017 study by Van den Stock et al.,[4] bvFTD patients did not differ from healthy controls in terms of their propensity to make utilitarian decisions on personal high-conflict moral dilemmas.[2,4] Indeed, we demonstrated that despite no differences in the actual decisions made on these dilemmas, bvFTD patients displayed an abnormal affective response toward their decisions. Second, if a common factor was driving social cognitive impairments in bvFTD, consistent and robust correlations between different measures of social cognition would be expected in these patients. This is not always the case, as while some studies show associations between performance on social cognitive tests,[2,5] others do not.[6] This, in fact, is also true of the 'emotional conflict' index employed by Van den Stock et al.[1] in their letter, which does not appear to correlate with theory of mind performance in their bvFTD sample.

Finally, evidence from other neurodegenerative disorders poses a challenge to the 'G-like' social cognition factor hypothesis. For example, theory of mind and moral reasoning performance do not appear to be correlated in Parkinson's disease,[7] while variable profiles of loss and sparing are observed on tests of empathy, moral reasoning, and theory of mind in Alzheimer's disease.[2,8,9] These findings reflect the difficulty in distinguishing between general cognitive or executive difficulties versus a pure sociocognitive deficit and speak to the complexity of attempting to distil multidimensional constructs into one common underlying factor.

These complexities are further borne out on the neural level. Van den Stock et al. include a novel re-analysis of resting-state functional imaging data, revealing associations between weaker medial prefrontal cortex and anterior temporal lobe functional connectivity and increased

utilitarian decisions on personal high-conflict dilemmas in bvFTD. Both the medial prefrontal cortex and anterior temporal lobes play well-established roles in supporting distinct facets of social cognition, including emotional processing, mental state representation, and social conceptual knowledge.[10] How such processes could be captured by a single sociocognitive factor remains an open question, though we agree that complex network dynamics are likely at play. We would argue, however, that such neural complexity further supports a multidimensional account of socioemotional cognition.

Inconsistencies in the inter-relationships between social cognitive domains, at both the behavioural and neurobiological level, suggest that a single underlying factor is unlikely to fully account for the complexity of sociocognitive disturbances in bvFTD. We agree with the authors[1] that future studies employing large-scale consortia, with pooled data across centres, will be essential to further explore the underlying dimensions of social, emotional, and conceptual processes and their corresponding neural mechanisms, eventually permitting a unified account of these uniquely human functions.

Competing interests

The authors report no competing interests.

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