COMMENTARY

Bridging psychiatry and neurology through social neuroscience

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Social neuroscience launched a novel multilevel (neural, hormonal, molecular and genetic) explanation of social cognition in psychiatry. In particular, the use of different levels of scientific inquiry assessing a) behavioral social cognition sensitivity to psychiatric impairment, b) neural networks engaged in social behaviors, c) the genetic underpinning of social phenomena, and d) the influence of the social environment on biological processes, have been outstandingly addressed by Cacioppo et al's paper (1).

Neuroscientific progress suggests that the separation between psychiatry and neurology is counterproductive. Classical neurological conditions present a range of social cognition impairments that are often underrecognized and frequently undertreated. Social neuroscience has made important progress in elucidating the neurobiology of the social brain, but has not focused sufficiently on neurological disorders. Here we consider the implications of social neuroscience research for a specific neuropsychiatric condition, the behavioral variant of frontotemporal dementia (bvFTD). Moreover, we highlight the importance of social neuroscience for the cross-talk among psychiatry and neurology.

BvFTD is a neurodegenerative disease whose initial symptoms are often confused with several psychiatric conditions. It is characterized by early decline in social interpersonal behavior, personality changes, and progressive deterioration in social functioning (2). Conventional neuropsychological assessment as well as clinical routine neuroimaging have been not been very useful for early diagnosis (2). The social neuroscience approach has raised new opportunities for research and translational applications in bvFTD.

First, social cognition assessment in bvFTD has allowed the detection of early and subtle behavioral impairments, appearing even before imaging signatures of brain atrophy, or a clear decline in formal cognitive status (3). In particular, social cognition tasks that resemble everyday behavior seem to be a far more adequate assessment for this purpose (4). Social cognition assessment may soon become part of the clinical screening for bvFTD.

Second, it has been proposed that models of social cognition associated with a degeneration of the fronto-insulo-temporal (social context network model) or fronto-insular (salience network) regions may explain the myriad of bvFTD social cognition impairments (2). For instance, Von Economo neurons are large spindle-shaped cells, abundant in the insular and anterior cingulate cortex. Among primates, these neurons have evolved only in hominids, and seem to be particularly vulnerable in neuropsychiatric conditions resulting in social cognition impairments. In bvFTD, a specific loss of these neurons within fronto-temporo-insular atrophy, at early stages, has been associated clinically with changes in empathy, social awareness, and other social cognition domains (5).

Third, an important genetic component of bvFTD has been related with social cognition impairment. There are three main genes for bvFTD: MAPT, GRN, and C9ORF72. Patients with C9ORF72 mutations exhibit widespread frontotemporal atrophy, associated with psychiatric presentations as well as with social neglect (6). In a similar way, animal models and clinical studies of GRN have shown early social and emotional changes, without gross impairment in overall health (6).

Fourth, the potential role of the social world, and its interaction with brain changes in bvFTD, deserves consideration. For instance, feeling lonely is associated with increased risk for dementia (7) and with a wish to hasten death in FTD (8).

An inter-level social neuroscience approach combining the study of social behavior, neural networks, genetic influences, and the interactions between social behaviors and social cognition would help to provide a more in-depth understanding of bvFTD, as well as of the overlaps of this disorder with the symptomatology and social cognition impairments of several psychiatric conditions (9). A new form of cross-talk between psychiatry and neurology may thus be developed in the social neuroscience arena, spearheaded by work on bvFTD as perhaps the clearest example of the bridges between the two disciplines.

Stimulating this cross-talk between neurological and psychiatric research seems to be one of the most promising roles for social neuroscience. Several neurological conditions with mental health manifestations (e.g., neurodegenerative conditions, prosopagnosia, tuberous sclerosis, and Angelman, Heller, Prader-Willi, Williams, Turner and Klinefelter syndromes) present impaired social functioning (10). Here, we have highlighted the multilevel social neuroscience approach to bvFTD, but the understanding of several other neurological conditions could benefit from this approach.

Many social cognition domains (social emotions, decision making, theory of mind, empathy, moral

cognition, and social norms) may be impacted differentially in various psychiatric and neurological conditions, and the differences in such parameters could be built into technologies for diagnosis and measurement of treatment efficacy.

Psychiatrists and neurologists within this novel social neuroscience approach may be able to contribute a powerful multidisciplinary and transdisciplinary approach (11), that would be both clinically and theoretically relevant to major advances in contemporary neuropsychiatry.

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