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How is quantification of social deficits useful for studying autism and schizophrenia?

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Imagine going to see your M.D., describing how you feel unwell, and being told that you have 'deficits in your immune system'. You would presumably inquire about the nature of the deficits, and whether they involve, say, autoimmune disorder or reduced levels of leukocytes. Now imagine a trip to the psychiatrist, whereupon, after a battery of tests, you are told that you have 'deficits in social cognition'. What have you learned?

Pinkham *et al.* (2019) compared individuals with autism to those with schizophrenia, for a large set of psychological tests designed to assess social-cognitive performance. Their comprehensive study was the largest to date in terms of sample sizes and numbers of tests, for such comparisons. They found that performance levels did not differ significantly between these two clinical groups for most of the tests. Their primary inference, couched in well-justified caveats, was an indication of 'the potential benefit of applying treatments transdiagnostically'.

An alternative interpretation of this and similar studies is that poor performance on standard psychological tests of social cognition provides little information about the diagnosis, causes, or treatments of psychiatric conditions. This interpretation is based on three main points.

First, similar levels of deficits in social cognition, as indexed by self-report and task-based psychological tests, can result from similar, partially overlapping, independent, or opposite biological causes. Table 1 lists studies in which patients with autism and schizophrenia were compared for both deficit-associated psychological traits (measured, or based on previous work) and their neurological mechanisms assessed by EEG and/or MRI. In each case, similar psychological deficits showed evidence of opposite underlying causes. Such results suggest that information on levels of psychological deficit is of limited usefulness, for understanding or treatment, without information on neurological causes or correlates. Apparently, there are too many ways for complex systems, like human social-cognitive development, to undergo alterations in function, for processes to be inferred using levels of deviation from optimality. Pinkham *et al.* (2019) acknowledge that shared deficits need not indicate similarity, but this point is not applied to their inferences, nor to the more general question of what information quantitative levels of deficits actually provides. A broader issue, as regards interpretation of social deficits found in both autism and schizophrenia, is that they are typically considered as evidence of etiologic overlap (e.g. Couture *et al.*, 2010). This belief is belied by the apparent complete lack of evidence in the literature for shared social deficits in autism and schizophrenia (or psychosis) being demonstrated to be underlain by shared neurological mechanisms.

Can disorder-specific tests salient to social-cognitive abilities help to alleviate such limitations? The Autism Quotient (AQ) test, one of the most commonly used metrics of autistic traits, shows elevated scores among individuals with anorexia (Westwood *et al.*, 2016), borderline personality (Dudas *et al.*, 2017), suicide attempts (Richards *et al.*, 2019), and schizophrenia spectrum disorders (De Crescenzo *et al.*, 2019). These results can be construed either as evidence of psychological overlap between autism and each of these conditions, or as indicating that the AQ broadly quantifies social problems (among other traits) with limited regard to their diverse underlying causes. Is there neurological overlap that grounds such psychometric data in biology?

Psychological tests that are based not on deficits *per se*, but on patterns of qualitative psychological differences, should, by contrast, provide information that can link more readily to potential causes. For example, the Movie for Analysis of Social Cognition permits differentiation of social deficits as due to under- *v.* over-mentalizing (Dziobek *et al.*, 2006; Fretland *et al.*, 2015), which can in turn be connected with different patterns of neural activation (e.g. Backasch *et al.*, 2013).

Second, quantification of psychological deficits, in the context of psychiatric categories, departs from Research Domain Criteria (RDoC) core recommendations to focus on adaptive biological phenotypes and how they malfunction, rather than on diagnoses. Approaches based on RdoC indeed fit with the standard medical model for the disease, which involves determining what biological system has become maladaptive, and how (Nesse and Stein, 2012). Application of the standard medical model of disease to psychiatry requires recognition that diagnoses are typically reified (falsely considered as 'real'), as opposed to conceptualized as what they are: societal constructs that are useful for scientific communication. Pinkham *et al.* (2019) contend that 'shared dimensions of observable behaviors may help to pinpoint

Table 1. Studies that compare subjects with autism and schizophrenia (or schizophrenia spectrum disorders) using the same protocol, and including neurological information from EEG and/or MRI

Study	Test(s), data collected	Pattern of results in autism compared to schizophrenia	Comments
Martinez *et al.* (2019*a*)	Face emotion recognition and motion sensitivity; EEG, fMRI, and rsMRI	Similar behavioural deficits in autism and schizophrenia at both tasks, underlain by distinct EEG and MRI patterns, some of which (EEG Theta power, Alpha ERD, and Alpha ssVEP, and MRI Early Visual and Dorsal) showed opposite patterns	See cogent discussion by Foss-Feig (2019)
Eack *et al.* (2017)	Visual perspective taking; MRI functional connectivity analysis	Similar behavioural impairments in autism and schizophrenia subjects; distinct differences in functional connectivity in social brain regions	Authors note that their results, in conjunction with previous studies, suggest that fronto-temporal connectivity is lower in schizophrenia and higher in autism, compared to controls
Stanfield *et al.* (2017)	Test of social judgment; fMRI data	Similar patterns of deviation from controls for judgments in subjects with autism and schizotypal personality disorder; fMRI activation patterns of controls described as intermediate between the two	Region with diametric pattern in autism (higher) *v.* schizotypal personality disorder (lower) includes temporoparietal junction, a region important in social cognition
Ciaramidaro *et al.* (2014)	Social intentionality test; fMRI data	Behavioural deficits in subjects with autism (lower accuracy) and schizophrenia (slower responses); fMRI shows evidence of 'opposite neural signatures' for social brain regions in the two groups	Interpreted in terms of hypo-intentionality in autism and hyper-intentionality in schizophrenia; see also Martinez *et al.* (2019*b*)
McCormick *et al.* (2012)	EEG mirror neuron paradigm, with actively psychotic subjects with schizophrenia spectrum disorders (SSD); replicated the protocol used by Oberman *et al.* (2005) in the study of autism subjects	Higher mu suppression among subjects with SSD compared to controls, with degree of suppression positively correlated with psychotic symptom severity; lower mu suppression in subjects with autism than in controls	Deficits in empathy considered as symptomatic in both autism and schizophrenia, from other studies

More-extensive comparisons of autism with schizophrenia, for diverse phenotypes, are provided in Crespi and Go (2015, Table 2).

common mechanisms' in the RdoC framework, but such pinpointing may be more likely through analysis of how biological adaptations connect to psychological maladaptations, rather than through computation of psychological impairments.

Third, the quantification of deficits in psychiatry, in conjunction with the application of reified DSM diagnoses, encourages treatments based on categories rather than individuals. Current diagnostic categories can instead be considered as starting points for differential diagnosis of the biological causes of each individual's psychological difficulties. Such individual-based, differential-diagnostic procedures are, of course, the norm in non-psychiatric medicine, such as immunology, but they are extremely challenging in psychiatry given the complexities of how the brain develops and works. However, remarkably, even given sets of well-validated causes and correlates of, for example, autism, procedures for differential diagnoses of its biological etiology have yet to be developed, aside from genetic testing. Self-report and task-based psychological tests for social cognitive domains will certainly have their place in individualized diagnostic protocols, but only to the degree that they indicate differences rather than just quantifying deficits, and thereby connect relatively directly with biological causation.

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