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Common social cognitive impairments do not mean common causes: A commentary on Cotter et al. (2018)

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ABSTRACT

Many clinical conditions, ranging from psychiatric to neurodegenerative illnesses, are associated with impairments in the processes by which we perceive, interpret, and respond to social information; a suite of abilities known as social cognition. Through a systematic review of meta-analyses, Cotter et al. (2018) present a compelling view of social cognitive deficits as a core phenotype of many clinical conditions. However, we caution against one potential interpretation of their findings, namely, that similar social cognitive outcomes are produced by similar causes. Specifically, we argue that while the outcome may look similar across clinical conditions (i.e., global social cognitive deficits), the cause and nature of those impairments are likely to differ, and, as a consequence, so too will its remediation. We advocate for the development of better methods for assessing social cognition, which may speak to the varying nature of social cognitive impairment across conditions. Ultimately, a better understanding of how social cognition is impaired will facilitate the development of more targeted, more effective treatments, that will improve patient care.

Now decades of research have shown that social cognition, or the cognitive processes that underlie our ability to perceive, interpret, and respond to social information, is impaired across clinical conditions, ranging from psychiatric illness to neurodegenerative disorders. Indeed, there is growing consensus among clinical researchers that social information processing deficits are a core feature of clinical conditions that robustly impacts day-to-day functioning. This growing recognition is embodied in NIMH's RDoC initiative, which includes *social processes* as a set of elemental processes that, when impaired, may form the building blocks of psychopathology.

Cotter et al. (2018) make these points in a thorough and thoughtful way. They present a review of meta-analyses demonstrating that social cognitive impairment is a transdiagnostic phenomenon that may index neurological health and serve as a linchpin for the real-world dysfunction we see in psychiatric and neurological illness. Cotter et al.'s efforts are a compelling call to action for researchers to prioritize the study of social cognitive deficits and their treatment.

However, we are wary about one possible interpretation of their findings. Someone may see these data and reasonably think that because the outcome is similar across conditions—a generalized social cognitive deficit as demonstrated by low “global” (Cotter et al., 2018, p. 96) scores on social cognitive tasks—so too is the cause and nature of

the impairment. Said otherwise, that poor performance across conditions is related to disruption to the same processes. And, given these similar disordered processes and outcomes, the same method of intervention will work across clinical conditions. This “common cause” inference is not an unreasonable inference to make. In fact, similar lines of thinking characterize the rationale behind areas of inquiry in psychiatry (e.g., attempts to find a “gene for” a given illness despite massive phenotypic heterogeneity that likely maps onto different genetic bases) and clinical psychology (e.g., arguments that treatment outcome equivalence between different psychotherapies means that the therapies *necessarily* work through the same non-specific, common mechanisms despite different theoretical bases and intervention procedures).

In the case of social cognitive impairment, Cotter et al.'s characterization of social cognitive deficits as transdiagnostic phenotype is well supported by the data. However, we caution readers against making a common cause inference. Similar social cognitive outcomes on social cognitive tasks do not necessarily mean similar causes of the dysfunction. To be clear, Cotter et al. reference this issue when they discuss the limited utility of studying “global scores” on ToM tasks and the need to evaluate “condition-specific emotion recognition profiles,” for example, as a way of “providing further insight into underlying

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disease pathology” (pp. 96–97). Indeed, we agree with Cotter et al. in that it is profoundly unclear what a “global score” represents, how comparable these are across measures, and ultimately, how useful this insight is without knowing more about the nature of the impairment.

For example, in the case of theory-of-mind (ToM), does poor performance reflect a lack of spontaneous and implicit mental state understanding or difficulty with conscious, deliberate reasoning about mental states? Does it reflect a failure of self-inhibition versus belief inference per se? Does it reflect a tendency to over- versus under-attribute belief states or the intensity of emotional experiences? These subtle differences, which are obscured by the omnibus effect sizes presented in Cotter et al., have been borne out in the literature in many of the clinical conditions reviewed in their paper. Importantly, interventions for social cognitive deficits would vary substantially based on the answers to these questions. Thus, if we want to predict illness course, reliably and sensitively screen for illness, and effectively intervene, we need to address these and related questions; that is, we need to better understand the nature of the deficits. Evaluating the overall magnitude of impairment across clinical conditions will only get us so far.

What might help to address the common cause inference, and in turn provide a more nuanced picture of social cognitive impairment across clinical conditions in a way that might facilitate early detection and treatment? We believe that part of the solution may lie in better assessment. As mentioned by Cotter et al., many extant social cognitive tasks are ill-equipped to address nuanced differences in social cognitive processes. There are notable exceptions of tasks that attempt to dissociate sub-processes involved in ToM (e.g., Biervoye et al., 2018), among other social cognitive processes, and, as mentioned by Cotter et al., initiatives to create neuropsychological batteries that assess a range of social cognitive domains (e.g., Bland et al., 2016). However, these tasks/batteries are few and far between, and their reliability is often either poor or unknown (National Advisory Mental Health Council Workgroup on Tasks and Measures for RDoC, 2016), making their use in clinical trials, for example, dubious. Even worse, some of the most widely used tasks lack ecological validity, face validity, and are confounded by variables that covary with psychopathology such as social class and culture (Dodell-Feder et al., 2018). More research into

better social cognitive assessment methods can change that. And given the current interest in this, which is likely to increase with Cotter et al.’s cogent review, there is no better time than now.

In summary, Cotter et al. (2018) make a strong argument for the transdiagnostic nature of social cognitive impairment, and we applaud their synthesis of the literature, which we hope will increase awareness around the importance of social cognitive deficits in clinical disorders. However, we caution readers against making the common cause inference. While the outcome may look similar across clinical conditions (i.e., global social cognitive deficits), the cause and nature of those impairments are likely to differ, and, as a consequence, so too will its remediation. We advocate for the development of better methods for assessing social cognition, which may speak to the varying nature of social cognitive impairment across conditions. Ultimately, a better understanding of how social cognition is impaired, through better assessment, will facilitate the development of more targeted, more effective treatments, that will improve patient care and protect people from the damaging effects of social disconnection.

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