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Discussion

Response to "Tandon et al. Psychiatry is a clinical neuroscience, but how do we move the field"

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In our perspective article (Torous et al., 2015), we suggest use of a neural systems-level, symptom-focused formulation approach to guide biological aspects of clinical case discussions during psychiatry residency training. These efforts are framed as one of several useful tools capable of catalyzing the integration of cognitive-affective neuroscience and neuropsychiatry into psychiatric practice. Tandon and co-authors in their commentary (Tandon et al., 2015) suggest unequivocally that our psychiatric neuroscience informed approach to clinical case formulations will not advance the neuroscience knowledge of the psychiatric practioner. While the authors make several important discussion points including highlighting the widening "language" gap between neuroscientists and clinicians, emphasizing that many mental health symptoms manifest prominently in an interpersonal, social context, and that the practice of psychiatry also draws from other disciplines outside of the brain sciences, we nonetheless disagree with several of their concerns.

(1) "*The anatomical localization model put forth by Torous and co-workers represents only a small part of our current understanding of neuro-circuitry.*" To be clear, we do not propose a traditional "lesion" model, but rather a network model; this approach emphasizes interactions between distributed brain regions that function as organized units mediating aspects of complex affective, cognitive, perceptual, behavioral and social functions. Within a group of brain regions that comprise a network, "hubs" represent sites of particularly robust connectivity with other brain regions both within a given network and across other networks (Buckner et al., 2009). Regional neuroanatomy represents the basic building blocks of network neuroscience. Individuals training in clinical brain science must develop proficiency in localizing well-researched symptom domains to specified brain networks and associated critical regions within a given network. For example, amygdalar based corticolimbic networks have been shown to subserve aspects of social life, including social network size (Bickart et al., 2012), and atrophy in these corticolimbic networks may correlate with deficits in social behaviors in patients with frontotemporal dementia (Bickart et al., 2014). Furthermore, a systems-level, brain-symptom relationship approach also considers that psychiatric symptoms seen in neuropsychiatric populations with focal lesions may be partially the product of functional and/or structural alterations occurring in distantly connected brain regions (a phenomena termed diaschisis) (Carrera and Tononi, 2014). Symptom-specific, brain-symptom relationships have been similarly identified and synthesized in idiopathic psychiatric disorders; examples include associations between the severity of experienced persecutory delusions and corticolimbic neural activations in response to social and linguistic threat in patients with schizophrenia (Perez et al., 2015). Thus, rather than representing only a small and static part of our understanding of neural circuitry, we believe that our model empowers trainees to explore the full, evolving clinical potential of brain research. Advances in the science of functional connectivity have only bolstered the need for psychiatrists to understand and communicate in the entwined languages of neuroanatomy and neural networks.

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(2) "*One needs to avoid oversimplification, particularly if it superficially appears to be clinically relevant*." We agree that brain-symptom relationships should be discussed with equal emphasis on anatomic specificity and a healthy recognition that clinical psychiatric neuroscience concepts require continuous updating and refinement. However, robust, clinically relevant network-symptom discussions do not oversimplify complex phenomena; we argue that such discussions provide a versatile scaffold of knowledge and are critical for our efforts to educate psychiatric residents in the emerging science of mind. For example, while the neurobiology of language has advanced greatly beyond the basic tenants that Broca's area mediates expressive language and Wernicke's area subserves receptive language functions (Mesulam et al., 2014), these brain-symptom relationships continue to allow generations of neurology residents to conceptualize aspects of left hemisphere lateralized stroke syndromes in clinical settings. This has set the stage for such clinical notions to be challenged and refined with the advent of new research technologies (Sepulcre, 2015). The time may now be ripe for making neuroscience-based predictions of disordered circuitries when dealing with idiopathic psychiatric syndromes; predictions such as aberrant amygdala or cingulate activity in our patient can be confirmed or refuted by the tools of modern imaging and clinical electrophysiology (contrast this with how to verify a formulation of our patient's depression being related to unresolved grief or aggression turned inward). Formulating testable hypotheses and then refuting them is the way knowledge in the rest of medicine and all of science has accrued; it is time for psychiatry to walk this path. To argue that our model is an oversimplification overlooks its didactic purpose and challenges the basic tenants of medical education in the 21st century.

(3) "*There are real dangers associated with propagating poorly substantiated theories of neurobiological underpinnings of mental illness*." Vibrant, dynamic discussions of brain-symptom relationships firmly defined as "work-in-progress" models and not framed as absolute truths allows academic psychiatrists to engage in real-time brain science discussions that have the potential to facilitate translational research efforts and expedite the bench-to-bedside delivery of much-needed, novel biological therapies. Work-in-progress models absolutely require high-quality, rigorous investigations to test and update models on an ongoing basis. Discussions of brain-symptom relationships at the level of case formulations are also no substitute for well conducted clinical trials investigating the utility of a biological informed treatment. Our educational approach emphasizes a conceptual understanding of circuit level brain function (and dysfunction) by learning the method of "mapping" particular psychiatric symptoms onto discrete neural circuits. This translational exercise will facilitate the modern day psychiatrist's adoption and successful implementation of future validated, rigorously tested biologically informed treatments (Etkin, 2014). One might actually argue that the real danger here is in not adequately teaching residents the clinical neurosciences, not exposing trainees to new models of understanding the brain and mind, and not giving future psychiatrists the best possible toolkit with which to conceptualize and treat patients.

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