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Using Advances from Cognitive Science to Understand the Etiology and Maintenance of Psychopathology

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Far too often, psychologists stay isolated in their particular area—we identify as a cognitive psychologist or a clinical psychologist, but rarely as both; we attend clinical psychology conferences or cognitive ones, but there is little overlap in attendance; we recruit graduate students in one area or the other, but seldom advise across areas. Yet, so much of the exciting work happens at the intersection between areas and disciplines, as we integrate (and beg, borrow, and steal!) theories, paradigms, and methods from across fields to create a novel synthesis. This has been especially true in the marriage between clinical and cognitive science, and the results have taught us a great deal about how thinking changes along the continuum from mental health to mental illness.

The aim of this special issue is to bring together reviews of cutting-edge research that illustrate how advances in cognitive science have improved our understanding of the etiology and maintenance of psychopathology, while also highlighting the many questions that remain unanswered. Authors were asked to consider how the application of cognitive science theories, methods, and paradigms has increased understanding of the etiology and maintenance of a particular clinical domain in light of the evidence related to distal and proximate risk and vulnerability. They were also asked to consider gaps in our understanding, such as the limited evidence for causal relationships between cognitive changes and onset of psychopathology. Beyond etiology, authors were encouraged to discuss how cognitive science has altered the ways we prevent, assess, diagnose, and treat each clinical domain, and they were asked to envision how these advances might alter clinical practice over the next 5, 10, and 20 years. Recognizing that we have far more questions than answers at this stage, we also asked authors to consider how to remove the barriers to making more rapid progress; that is, what methodological, theoretical, and infrastructure advances are needed to optimize the contributions that cognitive science can make to reducing the burden of mental illness? Along these lines, to make salient one obvious example of a gap in our knowledge that is hindering progress, all authors were asked to discuss what we currently know (and more importantly, don't know) about the ways individual differences and identities, such as demographic characteristics tied to race and ethnicity, age, sex, and so forth, interact with cognitive processes to alter the expression of and outcomes for a given clinical domain.

The authors rose to the challenge. They wrote about a wide variety of cognitive processes and clinical problem areas, and their papers make clear not only the tremendous progress that has occurred in this subfield, but also the long way that the marriage between clinical and cognitive science has to go to fulfill its promise.

McNally (2019) outlines emerging conceptual and measurement issues that fundamentally challenge the conclusions drawn from research on attentional bias for threat, focusing in particular on variants of the dot probe task, the dominant experimental attentional bias paradigm in anxiety research. He first reviews theory and evidence that had suggested selective attention toward threat cues across numerous anxiety disorders, and tracks the steady refinement of procedures for isolating the cognitive mechanisms underlying this bias. He then outlines the theory and origin of attentional bias modification, a set of procedures that typically train attention toward nonthreatening cues. McNally highlights the early promise of attentional bias modification scalability, but also some of the later null trials. As he explains, issues of the reliability of attentional bias measures (i.e., difference scores between mean reaction times for trial types that are individually reliable yet highly correlated, resulting in an unreliable linear composite) have emerged and now threaten the foundation of research on attentional bias. The ostensibly robust relationship between this bias and anxiety may result from greater attentional variability in people with anxiety than in those without, or from publication bias, among other possibilities. McNally notes that some researchers no longer view attentional bias as a trait, but as a process that fluctuates over time, and suggests that this bias in anxious people may be driven by an interpretation bias that lowers their threshold for attending to threat. He further points out that not all people with anxiety have an attentional bias for threat and that this may account for inconsistent findings. McNally concludes that attentional bias modification may be a useful component of stepped-care models, but the field clearly needs new, more reliable indices of attentional bias for threat.

Whereas McNally reviews research from a single cognitive domain (attentional bias for threat) across several anxiety disorders, Ouimet, Ashbaugh, and Radomsky (2019) review research from several cognitive domains—perception, attention, memory, executive functioning, dual systems—that pertain to the development, maintenance, and treatment of a single disorder: obsessive-compulsive disorder (OCD). Ouimet et al. conclude that evidence for *deficits* (i.e., dysfunctional processes) in these areas is mixed, but that people with OCD may have *biases* (i.e., dysfunctional content) that moderate the observed deficits and maintain symptoms. For example, the finding that people with OCD

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overestimate the size of disorder-relevant, but not disorder-irrelevant, stimuli suggests their perception is biased, yet intact. Notably, Ouimet et al. conclude that evidence for attentional bias in OCD (specifically a difficulty disengaging from threatening stimuli, which attentional control moderates) is mixed. Moreover, Ouimet et al. point out that attentional bias modification may be a promising adjunctive treatment, but a paucity of studies precludes attributing OCD symptom change to bias change. Paralleling McNally's discussion of attentional bias measures, Ouimet et al. observe that cognitive measures in OCD research often conflate processes. Further, most of the research, which is correlational, obfuscates whether deficits and biases cause or are caused by symptoms. The authors also highlight the role of cognitive distrust (e.g., doubting one's perception, memory, or decision making) in OCD and suggest that beliefs about cognition are amenable treatment targets. However, the authors lament that, aside from exposure and response prevention, cognitive science has yielded few interventions for OCD, and call for a shift from phenomenological to mechanism research with an eye toward identifying underlying processes most readily targetable in clinical practice.

Knowles and colleagues (Knowles, Cox, Armstrong, & Olatunji, 2019) discuss the transdiagnostic effects of disgust-related biases, presenting evidence that experiencing disgust more frequently or perceiving disgusting cues as more harmful is associated with an increased likelihood to experience anxiety-related psychopathology, along with borderline personality disorder, schizophrenia, eating disorders, and sexual dysfunction. In their review, Knowles et al. provide a detailed account for the ways in which disgust-related biases—at varying levels of conscious awareness—have been observed in memory, interpretation, judgement of expectancies, and attention. In particular, they posit that attentional avoidance may help disgust-prone individuals down-regulate the discomfort associated with being exposed to something disgusting, but attentional avoidance likely also accounts at least in part for findings that disgust-related biases are slower to habituate than those related to fear. While interventions that target maladaptive attentional avoidance will necessarily take on a different structure than typical attentional bias modification for threat (as the goal is to get someone to attend to a disgusting cue for a *longer* duration rather than to attend *less* to the cue), effectively intervening on attentional avoidance also has the potential to reduce symptoms across a variety of disorders. However, like McNally and Ouimet et al. discuss within their respective reviews, Knowles et al. state that before these advances in cognitive science can be translated into consistently effective interventions, future research must focus on individual differences that may moderate the effect of disgust-related biases on psychopathology.

LeMoult and Gotlib (2019) highlight a different process by which depression is thought to develop and persist. Specifically, they provide compelling evidence for the central role that difficulties inhibiting, and disengaging from, negative material in working memory play in the negative mood states that are typical of depression. LeMoult and Gotlib summarize the vast efforts that have been put towards better understanding deficits in this underlying cognitive control mechanism, as well as in downstream regulatory tendencies, and negative biases in attention and long-term memory. Importantly, their review emphasizes the consistent finding that deficits in cognitive control mechanisms are fundamental to the experience of depression, perhaps pointing to an underlying mechanism of change that could have cascading effects on biases and processes shown to exacerbate depressive symptoms. Additionally, they show that intervention approaches borne from this expansive literature can effectively target emotion regulation strategy use and negative cognitive biases to reduce depressive symptoms (e.g., rumination-focused cognitive behavior therapy: Watkins et al., 2007; cognitive bias modification: see Hallion & Ruscio, 2011, for review), though this work is still in early phases. They conclude by encouraging the field to extend these aggregate-level findings to the individual in order to develop more personalized and contextualized intervention strategies that can be effective for more people.

In contrast to other mental disorders, trauma-related disorders are linked to a specific onset event. Iyadurai and colleagues (Iyadurai et al., 2019) discuss the promise of intervening early following traumatic events through targeting intrusive thoughts in order to help prevent the eventual development of posttraumatic stress disorder (PTSD). Yet, while early interventions following real-world traumas have shown some promise (e.g., Horsch et al., 2017; Iyadurai et al., 2017), it is still largely unclear for whom and in what situations early intrusive thoughts will persist and develop into PTSD. Considering that intrusive thoughts may be normative in the hours and days following a traumatic event and recede naturally for some individuals, Iyadurai et al. raise important questions surrounding the decision of whether and when to intervene post-trauma. Given this challenge, their review underscores the importance of studying the temporal sequence of trauma in controlled laboratory experiments—including peri-trauma processes, hormonal differences, memory encoding, and cognitive processing style—to better understand the processes by which a traumatic event does or does not lead to sustained levels of clinical impairment. Though difficulties in measuring the occurrence of intrusive thoughts are notable, Iyadurai et al. suggest that intrusive thoughts may represent a promising treatment target for PTSD, perhaps allowing for greater intervention specificity than approaches attending to overall symptoms.

The study of alcohol use disorder (AUD) provides a valuable context to study cognitive biases that tend to be appetitive, rather than avoidant. Lindgren and colleagues (Lindgren et al., 2019) present recent advances in the study of AUD, spanning the areas of implicit cognitive biases, executive function, and functional connectivity neuroimaging. They also describe behavioral economic approaches to understanding alcohol decision making, and call for integrating situational context into models that emphasize the functional interplay between more impulsive and more controlled cognitive systems on addiction progression and maintenance. For example, they note that greater attentional biases towards alcohol-related cues and impairments in working memory have both been shown to predict alcohol use (Janssen, Larsen, Vollebergh, & Wiers, 2015; Khurana *et al.* 2013), suggesting strengthened impulsive systems and weakened control systems, respectively, are associated with increased drinking behavior. While much has been learned about the nosology of AUD, Lindgren et al. emphasize the importance of conceptualizing the interactions between cognitive processes and external factors (e.g., access to alcohol) to more fully account for the course of hazardous drinking. Further, given the effects of alcohol on brain development, Lindgren et al. highlight a relative gap in the literature concerning individual and situational differences in how these factors change and interact with each other differently across development and in response to sustained alcohol use. Despite substantial evidence that a broad range of cognitive biases and processes play a role in AUD treatment and relapse, work in these areas has yet to show sufficiently reliable effects to serve as stand-alone treatments. Although there is promise that some of these interventions (e.g., cognitive bias modification) may act as useful adjuncts to treatment-as-usual, future work is clearly needed to establish causal links between the posited cognitive mechanisms and alcohol use.

Finally, Cha, Wilson, Tezanos, DiVasto, and Tolchin (2019) systematically review the effects of a range of cognitive risk factors on self-injurious thoughts and behaviors (SITBs), finding that negatively valenced cognitions and biases are generally more predictive than content-neutral cognitive deficits. Regarding the former, SITB-themed cognitive risk factors (especially implicit associations between the self and death, and attentional bias toward suicide cues) robustly predict future suicidal ideation and attempts, whereas more general negative thought content and negative interpretations of oneself in relation to others only modestly predict suicidal ideation. With respect to cognitive deficits, cognitive inflexibility predicts future suicidal ideation, and rumination, a negatively valenced risk factor, mediates this relationship. Additionally, although low IQ modestly predicts future suicide attempts and death, this may be due to low socioeconomic status

following psychiatric diagnosis. Cha et al. also review trials of interventions informed by cognitive psychology (e.g., cognitive behavioral therapy) and observe that, although suicidal ideation generally declines across conditions, few trials assess the mediating or moderating roles of cognitive risk factors. In addition, studies most commonly assess suicidal ideation, leading the authors to call for research examining multiple SITBs to understand the pathway to suicide.

Together, these articles make clear not only the considerable progress that has been made in identifying cognitive biases and process differences that characterize a broad range of psychopathologies, but also the challenges that exist before these advances in cognitive science can actually improve clinical care. At this point, sufficient evidence has amassed to make clear that biases in cognitive content and processes are not simply epiphenomena of various clinical states. There are too many examples of them preceding and predicting future psychopathology, showing causal relationships with symptom change, and demonstrating cognitive mediation for an epiphenomenal argument to be widely applicable (though this may of course still be the case for a given instance). Moreover, it is clear that biased cognition describes many clinical problems, and there is evidence for a number of transdiagnostic markers, such as preferential processing of negatively valenced cues. Further, finding evidence for psychopathology-linked cognitive biases tied to attention, interpretation, memory, and automatic associations is the norm, rather than the exception, for the disorders studied to date.

At the same time, we still have far more evidence for cognitive biases as correlates of a disordered state than rigorous demonstrations of their role in etiology and mechanisms of change. While there are certainly null findings that raise important challenges to cognitive models of psychopathology, the more common case is simply a lack of data from which to draw causal conclusions. Future research is clearly needed to resolve open questions about when a cognitive difference tied to a clinical phenomenon reflects a difference in content (e.g., difficulties disengaging from ambiguously threatening material), process (e.g., differences in working memory), or both. In addition, further tests of the causal role of cognitive biases in changing a variety of clinical outcomes (beyond just symptom change) are needed, as are longitudinal evaluations of change in cognitive biases and associated clinical fluctuations over time. Also, there is a need to study biases in interaction with one another and across disorders, and a parallel need to isolate key change processes (e.g., using terms like *biased attention* is very broad and leaves too much ambiguity about the nature of biases—are the key differences tied to orienting, engagement, disengagement, or some other process?). Notably, the field has moved away from models that assume linear, sequential, isolated processing; instead, more recent models assume dynamic, iterative processing that involve complex interactions between different aspects of automatic and strategic cognitive processing (see Jones, Kirkland, & Cunningham, 2014). Yet, our empirical work often uses designs that provide only a static snapshot of a process in isolation, stripped of critical contextual information.

Given the many mixed findings highlighted in almost all of the articles in this special issue, it is clear that more work is needed on well-powered tests of moderation and mediation. The signal is getting lost to the noise as we use measures that conflate processes and samples that are highly heterogeneous without considering contextual influences and subgroups. Along these lines, every paper noted the absence of sufficient work examining the impact of individual differences; we still know little about how cognitive biases and their functional links to psychopathology differ as a function of demographic characteristics and identities, such as age, race, and sex. Another common concern noted across many of the articles is the need for methodological advances that can improve on the low reliability of many paradigms and the tendency for measures to lack precision in assessing temporal dynamics. In parallel with these paradigm design challenges, more precision in theory development is also needed. For instance, there is a need to be more exact in our conceptual terminology in order to improve the predictive value of our models; as just one example, executive function is a very broad, multifaceted concept, and pointing to differences at that level makes it difficult to isolate the relevant processes that need to be targeted to advance models of etiology and maintenance. The broader field is also evolving in its expectations for rigorous open science practices, including preregistering hypotheses, sharing data, and conducting replications. It is critical that we move toward open science best practices in our work so we can make more rapid progress and better understand which findings are likely reproducible.

With methodological progress, we will be better positioned to properly evaluate the clinical implications and applications of advances in cognitive science for prevention, identification, and treatment. As the papers comprising this special issue make clear, there are good reasons to be hopeful that interventions that target cognitive markers may one day have dramatic effects on symptom reduction and improve quality of life; however, advances in methodology are desperately needed to make this hope a reality. The papers in this special issue highlight exciting steps along this path.

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