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Theories of Psychopathology: Introduction to a Special Section

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Abstract

This special section on theories of psychopathology provides an opportunity to collect the emergent, cross-cutting scholarship that is challenging traditional approaches to understanding mental illness. Here, we appraise the state of theory in the field, and emphasize the pitfalls of working in the context of overly flexible, unchallenged, and essentially unchallengeable theoretic models, such as the biopsychosocial model, which we argue has become the de facto theoretic model for our field. We further posit that theoretic shortcomings are contributing to the often-referenced pessimism regarding our progress in understanding and treating mental illness, and introduce the charge of the authors of the papers in this section to articulate novel, falsifiable theories of psychopathology. We briefly touch on the intertwined issue of how to define psychopathology, and discuss a key issue raised by the array of papers comprising the section, namely how to conceptualize the spatiotemporal boundaries of complex causal systems. We then use this schematic for understanding how these theories relate to each other and to the vanilla biopsychosocial model they are vying to replace. Ultimately, it is our belief and hope that progress in theoretic thinking will catalyze faster progress in research and improvements to and novel developments in clinical prevention and intervention efforts.

General Scientific Summary:

This paper introduces a special section of the Journal of Psychopathology and Clinical Science on “Theories of Psychopathology.” In this piece, we discuss the state and importance of theory in the field, and outline the goal of and introduce the papers included in the special section.

Keywords

psychopathology; biopsychosocial model; causal systems; meta-theory

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The Perennial Challenges of the Psychopathology Researcher

The pitfalls and sometimes glacial rate of advancement in psychopathology are well known to any who have embarked on and even more so those who have persisted in this endeavor. Yet, the urgency and profound importance of scientific progress and breakthroughs, their translation into actionable clinical prevention and intervention efforts, and subsequent dissemination and implementation of these efforts are felt viscerally by those of us affected by psychopathology, either by our own lived experience or through that of someone close to us (a list that includes practically everyone; see the provocative findings reported in Caspi et al., 2020).

In one sense, we have made significant progress in our study of psychopathology. This *Journal* and many others have volumes filled with research findings, and in many respects our understanding of psychopathology evolved dramatically over the course of the 20th and so far in the 21st century. And yet, if one were to "take the temperature" of the field, we hazard enthusiasm would be lukewarm. A common perspective is that we have made disproportionate investments and efforts relative to the knowledge gained regarding what exactly psychopathology is, what causes it, how to prevent and how to treat it (Insel, 2022).

This is certainly due in part to the difficulties inherent to this line of work, such as the staggering complexity of psychological phenomena and mechanisms. The combination of this complexity with the idiographic nature of psychological development and transactional processes, both of which impede generalization, suggests low expectations may be appropriate (Lykken, 1987). More fundamentally, there is inherent vagueness to the constructs of interest in psychopathology research, and the many levels of analysis at which one can focus one's lens when viewing these phenomena adds an additional layer of complexity to research in this field (Markon, 2013; see also Cronbach & Meehl, 1955). Furthermore, the frequent infeasibility of conducting true experiments severely limits the causal inferences that can be drawn in psychopathology research (Vaidyanathan et al., 2015). Meehl (1978) famously outlined further difficulties in both the application of statistical testing to and the interpretation of null findings in the context of psychopathology research. These challenges interact with a number of more specific challenges to further complicate interdisciplinary research efforts, for example in cognitive neuroscience (see Miller's classic 1996 discussion) and psychiatric genetics (see Kendler, 2005). While there are certainly difficulties inherent to our science, we pose the question of whether the problem is broader and arguably more pervasive than these issues. We are concerned about the state of theory in the field -- something we may have control over -- and the effect weak theories have on our science, our thinking, and our progress.

Epistemological Concerns

The field lacks a unifying theoretic framework other than a vague notion that psychopathology is caused by a combination of biological, psychological, and social (all broadly defined) factors. We further argue that psychopathology research in much of the world operates using this overarching biopsychosocial model as a sort of de facto theoretic framework. Despite being vague, this model has morphed somewhat over time,

incorporating biological, psychological, and social components at multiple levels of analysis as well as their interactions. However, for the most part the biopsychosocial model has received little to no intellectual attention in over 50 years. Instead of theoretic progress, we have seen the biopsychosocial model expand to accommodate the advances we have made in psychopathology research.

Despite all this, the biopsychosocial model is still so broad and vague it is essentially irrefutable, a quality that renders this de facto theoretic model unscientific (Popper, 1968), in its current form. In practice, this breadth and vagueness render the biopsychosocial model largely invisible to us, its presence looming high overhead, many steps removed from the science that we do. When considered in combination with universal deference to this model, the approach bears all the hallmarks of Kuhn's (e.g., 1970) notion of "normal science", wherein a pervasive theoretic framework is essentially taken for granted and studies serve to flesh out the details with only relatively minor adjustments ever made to the theory. As Popper (1970) warns us, this sort of scientific orientation is characterized by a limited set of pathways to meaningfully challenge a prevailing theory.

And yet we know that theoretic frameworks influence every aspect of the scientific process, from our identification and framing of research questions to the methods we use to solve these problems and our interpretation of research findings (Popper, 1968). Thus, it should concern us that our de facto theory is so disembodied from our science, and that it is essentially irrefutable. Also per Popper (1968), there is a higher yield in terms of information gained when we have falsified a theory rather than supported it, and Popper's prescription for us as we attempt to acquire knowledge is to iterate towards the truth by way of proposing strong theories followed by enthusiastic attempts to falsify them, then again proposing new theories and so forth.

With these issues at top of mind, we posit that the *lack of innovation in overarching theoretic perspective is an appreciable part of what slows our progress and makes our science difficult*, contributing to a gloomy sense of being stuck in a non-cumulative scientific endeavor. Perhaps this would improve if we devoted as much effort to the development of cutting-edge and comprehensive theoretic frameworks as we do to our day-to-day Kuhnian (1970) puzzle solving. In addition to catalyzing innovative research, such theoretic advances could serve to organize subtheories at more and more specific levels of inquiry and analysis, and stitch together our work in a conceptually coherent and more cumulative way. For this special section, we have challenged clinical scientists to revisit this default theoretic paradigm in psychopathology -- to make the invisible visible, and to transform the useful aspects of the current model–the idea that there are multiple and interacting causal factors at multiple "levels" of analysis contributing to the development of psychopathology– into fully articulated scientific theories amenable to testing and refutation which inspire innovative empirical work and put forth novel conceptualizations of psychopathology.

The Stakes and the Stakeholders

While certainly ambitious, the charge to generate novel, unifying theories of psychopathology may at first blush also seem esoteric. However, in the context of

psychopathology, Popper's (1968) points regarding theory serving as the lens for all of our interpretations and problem solving translate into a profound impact of our theoretic framework on clinical care. For example, Braslow and colleagues (2021) have argued that our piecemeal biopsychosocial conceptualization of psychotic disorders justifies treatments that target specific components of the disorder at the expense of emergent, holistic outcomes. They suggest that by solely focusing our tools on specific symptoms or symptom dimensions we serve our patients poorly when they suffer more pervasive and often graver challenges, such as high rates of chronic houselessness and incarceration. These direct implications of our theories for contemporaneous patient care may seem counterintuitive, but it should come as no surprise that the way we think about our patients impacts the way we treat them.

Despite some of our arguments above, we can understand a potential counterargument that efforts at formulating a unifying theory of psychopathology are premature given how much is still unknown in this field. It could even be said that such efforts are misguided; indeed, some readers may recall Santiago Ramón y Cajal's 1897 *Advice for a Young Investigator* to focus on data collection because, "Hypotheses pass, but facts remain. Theories desert us, facts defend us." To address these issues, we turn back to Sir Karl Popper. Popper's (1968) points regarding the absence of any true authority of knowledge and our lack of access to any objective truth are especially resonant in the field of psychopathology research (e.g., see MacCorquodale & Meehl, 1948 and Cronbach & Meehl, 1955; see also the history of difficulties surrounding nosology). Similarly, we believe that Popper's (1968) prescription of conjectures and refutations as the most reasonable means of navigating such an opaque situation applies in this specific case too. Analogous to the point about the effect of our theoretic thinking on clinical care, we believe that theoretic framework has profound effects on our science, and that in considering our research, making progress on our theories will facilitate scientific discoveries about mechanisms of and treatments for psychopathology. This might occur by enhancing the cumulative and coherent nature of our efforts while also providing guidance toward the most impactful research questions.

Ontological Questions: What Is Meant by Psychopathology?

A core issue underlying this special section is how to define psychopathology and mental illness. On one level, this question again has the potential to speak to our academic distance from the problem; people suffering from mental illness and those who care for them are faced with the challenge of *how* to treat mental illness rather than *what* is mental illness. However, in addition to our related points outlined above, an answer to *what* provides leverage as to the *why*s of psychopathology. In many other domains then, clarity about *what* and *why* has provided leverage for the *how*s of addressing suffering.

A common means for defining psychopathology has been to identify it as a statistical deviation, a notion that undergirded the former title of this journal, the *Journal of Abnormal Psychology. Abnormality* marks something outside the norm. As noted elsewhere (Patalay & MacDonald, 2022), this approach has several shortcomings, the foremost being that psychopathology is common (Schaefer et al., 2017). Furthermore, as DeYoung and Krueger (this issue) observe, "some people with high scores on almost any symptom dimension

do not have the suffering or impairment that is expected to accompany mental illness." Other definitions have focused on *pathology*, the Greek root of which means passion or suffering. The term was adopted by medicine in the 1600s to mean disease or disorder, before being backformed (ironically) in the 1800s to become *psychopathology* (Oxford English Dictionary, n.d.). While suffering is common in mental illness, many theorists would wish to include ego syntonic conditions such as psychopathy and some eating disorders that are destructive or dangerous without disturbing the individual per se.

One of us has suggested examining psychopathology from an engineering perspective and adopting the tools developed for the quality control of metal fatigue and failed rocketry to open new approaches for thinking about mental illness (MacDonald et al., 2016). This branch of engineering science, known as reliability engineering, distinguishes between faults and failures. A fault occurs when an individual component does not perform its required function. When a neurotransmitter receptor impairs synaptic communication, an item in memory is lost, or a facial expression is misapprehended, a fault has occurred. Depending on how noisy and complicated the system -- and the brain is both -- many faults might occur without a failure. A failure occurs when there is a loss of some fundamental function of the brain that impairs cognition, emotion, or behavior. Thus a failure impedes the functionality of a particular subsystem, but not the whole system. In contrast, a *general* failure mode prevents the system from performing its designed function at all. This is the kind of failure that sends the rocket crashing into the hillside. A general failure mode in a human might be similarly disastrous. From this perspective, the goal of psychopathology research is to understand how faults cascade into the failures of the central nervous system that substantially increase the risks of a general failure mode which could include any number of synthetic criteria, from individual suffering to causing suffering in others, to other holistic criteria as one sees fit.

By emphasizing continued functioning, reliability engineering explicitly recognizes the importance of homeostasis in the face of a changing environment. This theme also emerges in several of the articles in this section. Nigg (this issue) notes that psychopathology can be viewed as emerging from dynamic perturbation and adaptation processes, and that therefore a theory of mental illness must address the "complexity, development, environmental/social context, and self-regulating mental evaluations of experience." Jin, Jonas and Mohanty (this issue) also highlight the role of adaptation, noting that "psychopathology is the observable consequences of [the function of internal and external forces, such as genetic and temperamental predispositions, biochemical fluctuations, as well as familial, social, and cultural factors] that cause harmful dysfunction or, more broadly, maladaptive functioning" in a given environment (Del Giudice, 2016; Wakefield, 1992). By adopting an evolutionary perspective -- which is to say adaptation over a much longer period -- these authors further broaden the timescale of adaptation. DeYoung and Krueger (this issue) note the importance of the individual's perspective, defining "psychopathology as *persistent failure to move toward one's goals, due to failure to generate effective new goals, interpretations, or strategies when existing ones prove unsuccessful"*. What these definitions of psychopathology highlight are the need for progress toward one's own and appropriate contextual goals, while acknowledging the dangers of rigidity in the face of environmental

changes. If this is to be our explicandum, or thing to be explained, how shall we understand what constitutes a threat to progress or a cause of rigidity?

Boundaries of Causal Systems

By design, the papers in this special section illustrate the challenge of defining complex causal systems. Delineating the components of a causal system as well as how their dynamics progress over time is critical to understanding any causal network. This is especially critical when studying behavior, where the phenomenon of interest comprises an individual's response to the immediate environment in conjunction with various aspects of that organism's past. It becomes more critical when different levels of analysis are considered, and individuals, their environments, and their responses become part of a broader socioecological system. Thus one major question suggested by the papers as a whole is how do we define and isolate a causal system? That is, is it best to focus consideration on the person per se, that person and what has been their immediate surroundings, or some broader system? And how do we define such a system with regard to its time frame?

The question of how narrowly or broadly to define the explanatory system surrounding an individual — or indeed, what constitutes an individual — has become an important theme not only in psychology but in other fields of science as well. Within the behavioral sciences, for example, there has been increased interest in idiographic and intensively intraindividual accounts of behavioral and psychological processes (e.g., Wright & Woods, 2020). Simultaneously, however, other fields have seen increased interest in how to define individuals vis-a-vis broader sociobiological contexts, due to observations that dynamics over time are often understood in terms that transcend traditional conceptions of a specific individual organism (Krakauer et al., 2020). The salience of these issues points to challenges in how we measure and define the scope of predictive and causal accounts.

This issue of causal system boundaries extends to time frames as well. One perspective focuses on proximal events associated with adjustment, such as in the case of grief or trauma-related psychopathology. On the other end of the spectrum are perspectives in which psychopathology is seen as part of a dynamic evolution over time, beginning as early as birth or even beforehand, and continuing through development in a cumulative cascade of mutual causes. Approaches to causality are diverse in perspective, from how to define individuals and their constituent systems, to the temporal scale over which those explanations are considered.

The Current Offerings

Each of the papers in this special section adopts a position with regard to the definition of psychopathology and these spatial and temporal boundaries of causation. As a set, however, they illustrate the landscape of potential processes acting within and among individuals in their respective contexts, with regard to a specific moment in time as well as over longer periods of time.

DeYoung and Krueger (this issue) present an innovative self-regulatory definition of psychopathology focused on goal adaptation, reframing adjustment in terms of intraindividual phenomena and allowing for highly idiographic characterizations of mental illness. In a complementary idiographic treatment, Lazarus and Rafaeli (this issue) discuss modes — distinct patterns of coactivated affects, cognitions, motives, and behaviors that constitute the manifestations and causal dynamics of psychopathology. Their treatment includes explicit discussion of development and time, how individuals might navigate multiple discrete states at different times, and how these states might develop. Both of these papers, although not focused on intraindividual processes exclusively, provide formal means for understanding individuals per se as unique systems, and frameworks for describing and understanding unique features of psychopathology, as well as idiography as a ubiquitous feature of psychopathology.

Other papers in the special section shift focus away from the individual in certain respects, either by moving away from intraindividual processes per se, toward extraindividual variables, or by shifting focus toward more nomothetic, interindividually generalizable patterns. Jin, Jonas, and Mohanty (this issue) discuss the implications of predictive processing models for psychopathology. In these models, the brain can be seen as operating to reduce surprise from environmental stimuli, thereby reducing free energy required by the individual to navigate the world. Predictive processing models provide a framework for linking the past — ontogenetic as well as phylogenetic —to the present, and a novel paradigm for understanding how psychopathology might develop and persist. Although nomothetic in focus, it provides a broader framework for understanding how idiographic patterns might come to be, and integrates the intra- and extraindividual seamlessly in one account.

Wright, Pincus, and Hopwood (this issue) provide an overview of contemporary integrative interpersonal theory, an integrative account of the “emergence, expression, and maintenance of socioaffective functioning and dysfunction across levels and timescales of analysis.” The authors and their colleagues have argued that some forms of psychopathology are best understood as interpersonal disorders (Wright, Ringwald, Hopwood, & Pincus, in press), and here provide an expanded account of interpersonal theory and its critical relevance to the etiology and maintenance of psychopathology. Their account dovetails with predictive processing models in focusing on expected environment-response patterns, but with a focus on the form of those expectations for a critical domain. It also encompasses the intraindividual and interindividual levels of analysis, outlining how recurring patterns in the external world can come to shape the structure of internal processes.

Hostinar and colleagues (this issue) underscore the central role of stressors and stress response in the development and persistence of psychopathology. In their paper, they emphasize the importance of characterizing pathological behaviors in the context of stress, and of the role of early stress in shaping later psychopathology patterns. As in the papers by Jin et al. (this issue) and Wright et al. (this issue), intraindividual processes are a focus, but in a more nomothetic framework, aimed at characterizing common psychological patterns, and with a broader timescale in mind.

Other papers in this special section expand the scope of causal dynamics even further, with a focus on individuals as parts of a dynamic system that unfolds over the course of development. In these accounts, the focus is often on how individuals shape their environments as much as how the environment shapes behavior, and how these mutual processes unfold over time. Elam, Lemery-Chalfant, and Chassin (this issue) discuss gene-environment cascades during development, and how, with gene-environment correlation, the environment can be seen partially as an extension of genotype in many cases, either in an individual or in their family, and how these environmental influences can be buffered by appropriate interventions. Nigg (this issue), in a complementary paper, outlines mechanisms by which the environment affects genotypic expression via epigenetic effects, and the role of environment-neurodevelopment dynamics more broadly. The paper of Olthof and colleagues (this issue), similarly, treats these issues very broadly, outlining fundamental theoretic issues about psychopathology being a property of individuals versus the system of which individuals are a part. Each of these papers insightfully raises questions about the boundary of the individual versus environment, especially as these relate to one another dynamically over the course of development.

This special section grew from a dissatisfaction with the theoretic foundations of the scientific model our field has adopted. Many of us have, in effect, whittled at our branch of the biopsychosocial model of psychopathology without much regard to the overall tree. The theories in this resulting section add new value. By highlighting common processes and *leitmotifs* across problems that might appear to be quite distinct, they suggest a way of thinking that goes beyond a narrowly defined diagnosis. These shared processes can be thought of as core failure modes, emphasizing their relative importance and the need to test, and in some cases we hope, falsify the theories contained in these pages. Finally, we hope that by reexamining the conceptual foundations of our science we might hasten the pace of moving psychopathological discoveries into actionable treatments.

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