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STANDARD PAPER

The Classification and Explanation of Depression

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Abstract

In the field of psychopathology there is still a lack of consensus on how mental disorders, such as depression, should be classified and explained. Many of our current classifications suffer from disorder heterogeneity and are conceptually vague. While some researchers have argued that mental disorders are better explained from a biological perspective, others have made the case for pluralistic and integrative explanations. Using depression as an extended example, we explore the challenges in classifying and explaining psychopathology. We begin by evaluating the current approaches to classification, including frameworks for what we consider a mental disorder. This is followed by a detailed summary of current explanatory perspectives in psychiatry. The relationship between classification and explanation presents unique theoretical challenges in understanding mental disorders. We suggest that by adjusting our focus from understanding syndromes to clinical phenomena we can advance our understanding of mental disorders.

Keywords: classification; explanation; depression; phenomena; psychopathology

The term *depression* is frequently used to capture experiences of prolonged sadness and debilitating low mood. In the context of mental health, depression is considered a mental disorder that is marked by significant disturbances in mood, cognition, physiology, and social functioning. A deep sadness and its invariants, such as hopelessness, sorrow, emptiness, and despair, have formed the core features of depression that, over time, have expanded to include an inability to experience pleasure, psychomotor dysfunction, changes in sleep and eating behaviours, difficulty concentrating, and suicidal thoughts (Horwitz, Wakefield, & Lorenzo-Luaces, 2017). Since the introduction of diagnostic criteria, we now have an expanded set of concepts for describing depression, including mild, moderate, severe, major depressive disorder (MDD), dysthymia (chronic depression), and seasonal affective disorder (American Psychiatric Association [APA], 2013).

Part of the problem with our nosology in psychopathology is that there is no *clear* consensus on how mental disorders such as depression should be classified (Kincaid & Sullivan, 2014). This has important implications in our psychopathology research as, more often than not, the targets of explanation are diagnostic categories (e.g., MDD). If we get our clinical nosology wrong, then our psychiatric research will suffer as a consequence.

Ideally, the concept of depression should not only serve to describe an individual's clinical presentation but also to explain why their thoughts and behaviours are abnormal; describing people as suffering from depression should provide a deeper explanation of why they experience prolonged sadness along with other typical features of the disorder (Thagard, 2016). Over the last 50 years there have been significant advancements in our understanding of depression. However, despite the wealth of research within the field, it is unclear how best to build explanations of mental disorders such as depression.

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Using depression as an extended example, this article aims to explore the current issues in the classification and explanation of psychopathology. We begin by evaluating the current approaches to classification. An overarching challenge in this area is the lack of a framework for what we consider mental disorders; therefore, we explore alternative frameworks in the context of depression. Next, we provide a detailed evaluation of the current explanatory perspectives in psychopathology. Finally, we explore the unique relationship between classification and explanation and make some suggestions for future development.

Approaches to Classification

Classification is the construction of categories or groups to which entities (disorders or persons) are assigned on the basis of their shared attributes or relations (Millon, 1991). In medicine and psychiatry, diagnosis and classification are interrelated concepts: a diagnosis is the allocation of signs and symptoms into a classification category. Classifying entities that are similar in theoretically important ways is a valuable part of scientific practice (Cooper, 2012). When correctly achieved, we can expect to predict how these entities will behave, based on the groups they are assigned to. However, our classifications in psychiatry have been fraught with conceptual challenges. Namely, there is contention over whether current classifications represent *real* and *valid* entities.

The most prominent model of classification in psychiatry is the categorical model, which represents mental disorders as discrete categories that are defined by non-arbitrary boundaries. For example, people who suffer depression are thought to be qualitatively distinct from the normal population. Categorical models are common in medical research and are easy to use under conditions of incomplete clinical information (Jablenksy, 2012). However, the dimensional nature of mental disorders presents some challenges when trying to construct a categorical classificatory system. While the categorical approach requires setting *thresholds* that qualitatively distinguish a disordered from a non-disordered state, the multidimensional nature of mental illness requires that thresholds be set for each component dimension (Clark, Cuthbert, Lewis-Fernández, Narrow, & Reed, 2017). For example, MDD has emotional (e.g., depressed mood), behavioural (e.g., psychomotor changes), cognitive (e.g., difficulty concentrating), and physical (e.g., fatigue) dimensions, each requiring a 'threshold' determination to meet diagnostic criteria and to be considered sufficiently disordered (Clark et al., 2017). These distinctions are fairly arbitrary, based largely on an individual's self-report of signs and symptoms and a clinician's own expertise.

Many researchers have suggested that disorders such as depression should be seen as dimensional; for example, people who suffer from *severe* depression occupy a higher position on a continuous latent variable rather than being qualitatively different from the normal population (Borsboom, 2008). The advantage of this view is that it allows for greater heterogeneity and discrimination between different levels of severity of a disorder. This is particularly useful for patients who meet the diagnostic criteria for multiple psychiatric disorders as it allows for the diagnosis of 'subthreshold' conditions (Jablensky, 2012). However, it may not be enough to say that depression is a latent continuum as we may actually be referring to a set of latent continua, intertwined in various ways, or even varied sets of continua for different population groups (Borsboom, 2008).

In order to highlight these issues, we explore the advantages and limitations of three alternative taxonomies for classifying mental disorders: the *Diagnostic and Statistical Manual of Mental Disorders*, the Hierarchical Taxonomy of Psychopathology, and the Research Domain Criteria project.

Diagnostic and Statistical Manual of Mental Disorders

The majority of psychopathology research revolves around mental disorders as defined and classified by the *Diagnostic and Statistical Manual of Mental Disorders* (5th edition; *DSM-5*; APA, 2013). For example, a diagnosis of MDD requires five (or more) symptoms from a list including: weight loss or weight gain, insomnia or hypersomnia, psychomotor retardation or agitation, fatigue, feelings of

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worthlessness or excessive guilt, diminished concentration, and suicidal ideation. These symptoms must have been present for a period of 2 weeks, represent a *change* in functioning, and be causing significant impairment or distress (APA, 2013). At least one of the symptoms must be either depressed mood or a loss of interest/pleasure (APA, 2013).

For the most part, the *DSM-5* represents a categorical classificatory system in which mental disorders are classified as distinct entities. Since the publication of the *DSM-III*, the manual has been largely descriptive, excluding specific theories of the causes of mental disorders in order to maintain reliability across its use (Cuthbert & Kozak, 2013; Pincus, 2012). The *DSM*'s utilisation of diagnostic criteria and its emphasis on empirical data has improved reliability and facilitated communication among clinical practitioners and researchers (Pincus, 2012). However, there are several conceptual disadvantages with the current categorical approach that pose significant limitations.

The criteria for classifying mental disorders does not always bear a close relationship to the intended theoretical construct they aim to represent. For disorders such as MDD, there are a vast number of symptom combinations that meet criteria for diagnosis, and different disorders may significantly overlap. For example, symptoms of restlessness, fatigue, difficulty concentrating, irritability, and sleep disturbance overlap between generalised anxiety disorder (GAD) and MDD (APA, 2013). Depression is associated with a wide range of features, many of which are central to the disorder, such as depressed mood, whereas others are not specific to depression (Allen & Badcock, 2003). This makes it difficult to confirm whether statistical associations are directly linked to the whole disorder or only one component, weakening our ability to make valid explanations of these categories.

Hierarchical Taxonomy of Psychopathology

In response to the limitations of traditional taxonomies, the Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2017) was established with the aim of developing an empirically driven classification system based on advances in research on the organisation of psychopathology. The HiTOP model incorporates 'dimensions', or psychopathologic continua, that reflect individual differences in maladaptive characteristics across the entire population (Kotov et al., 2017). These dimensions are organised hierarchically across *six* levels: *homogeneous components* (constellations of closely related symptom manifestations; e.g., low mood and loss of pleasure); *maladaptive traits* (specific pathological personality characteristics; e.g., anxiousness); *syndromes* (composite of related components/traits; e.g., MDD); *subfactors* (groups of closely related syndromes; e.g., distress subfactor); *spectra* (larger constellations of syndromes; e.g., internalising spectrum); and *superspectra* (broad dimensions comprised of multiple spectra; e.g., general factor of psychopathology).

The model focuses on the level of 'spectra', which is argued to provide a more detailed and specific picture of psychopathology. It is also able to better account for comorbidity as co-occurring syndromes (e.g., MDD, GAD, dysthymia, post-traumatic stress disorder [PTSD], and borderline personality disorder [PD]) are grouped under the same spectrum (e.g., internalising).

The advantage of HiTOP is that it provides a *parsimonious* alternative to traditional taxonomies that better captures the dimensional nature of mental disorders. The HiTOP model also provides a broad account of psychopathology that includes nearly all common conditions, is empirically supported, and has cross-over with current concepts and measures already being used.

A significant limitation with the model is that much of the research into the HiTOP dimensions has relied on traditional diagnostic categories. This problem is exacerbated by the direct incorporation of *DSM* diagnoses into the model (e.g., MDD and borderline PD). The problem is that categorical diagnoses may distort findings as they lack construct validity; the criteria for diagnosing disorders do not always bear a tight relationship to the theoretical construct that they were originally intended to represent. The HiTOP model also uses a number of existing instruments to assess component/trait, syndrome, subfactor, and spectrum levels of the model; however, these measures are largely self-report, with 13 out of the 15 measures having a complete or partial self-report format.

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Research Domain Criteria

Due to the lack of progress in psychopathology research, primarily because of the absence of knowledge of the causal processes underpinning mental disorders, the US National Institute of Mental Health (NIMH) initiated the Research Domain Criteria project (RDoC; Insel et al., 2010). The RDoC framework is *not* a classification system; it is conceptualised as a long-term program of research that may ultimately lead to the development of such a system (Lilienfeld & Treadway, 2016).

The RDoC framework exists as a research matrix with five domain constructs and their associated systems on the y-axis and units of analysis on the x-axis. The matrix is designed to discover the components and mechanisms that constitute the core psychological systems using data gathered at the different units of analysis. The *five* functional psychological systems that have been identified so far are: *negative valance systems* (enable response to aversive stimuli or contexts; e.g., fear, anxiety, loss); *positive valance systems* (enable response to positive stimuli or contexts; e.g., reward seeking, reward/habit learning); *cognitive systems* (enable cognitive processes; e.g., attention, perception, memory); *systems for social processes* (enable responses in interpersonal settings; e.g., affiliation, attachment, social communication); and, *arousal and regulatory systems* (enable arousal systems and regulate homeostatic systems; e.g., circadian rhythms, sleep-wakefulness, brain-stem activation). Data is gathered across *seven* units of analysis: genes, molecules, cells, circuits, physiology, behaviour, and self-report. Ultimately, the aim is to work out how normal psychological systems function and what occurs if they are faulty in some way.

The RDoC framework is novel in that it shifts the focus from utilising traditional diagnostic categories in research towards dysregulated neurobiological systems (First, 2012). As a consequence, it lays out the potential for a classificatory framework that moves away from purely descriptive categories to introduce relevant causal processes. However, the framework does face some conceptual issues based on some of its core assumptions. In the RDoC framework, mental disorders are conceptualised as disorders of brain circuitry, and it is assumed that dysfunctions in these circuits can be identified using clinical neuroscience tools, including electrophysiology and functional neuroimaging (First, 2012). In its current form, five out of the seven units of analysis exist at the level of *biology* (i.e., cells, genes, molecules, physiology, and circuits). Additionally, psychological, social/cultural, and phenomenological variables are largely omitted from the research matrix. Although the matrix does include a 'self-report' unit of analysis, self-report is not limited to any level of analysis and could include reported changes in social functioning, behaviour, cognitive processing, and physiology. The overemphasis on the biological and neural *levels of analysis* presents a challenge to the framework. Namely, the framework runs the risk of losing important causal and contextual information for understanding the development of mental disorders such as depression.

Summary

In its current form, the *DSM* suffers from several conceptual challenges that limit its validity as a classification system. These include problems with heterogeneity, complexity, and disorder overlap. An overarching issue is its inability to accommodate the dimensional nature of mental disorders and its clear omission of causal information. Both HiTOP and RDoC present novel and useful opportunities to revolutionise and improve our current nosology; however, they are currently in the early stages of development and face several conceptual limitations. These include the HiTOP's reliance on current diagnoses, as defined and operationalised in the *DSM*, and the RDoC's neuro-centrism.

It is important to note that categorical and dimensional classifications are not necessarily at odds with each other. Recently, the two models have been combined to create mixed models that have qualitative categories with quantitative traits (Jablenksy, 2012). For example, the categorical description of schizophrenia can be refined with quantitative traits, such as measurements of memory dysfunction, attention, and changes in event-related potentials (Jablensky, 2009). This approach allows us to refine our current categorical classifications while retaining their usability.

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What is a Mental Disorder?

An overarching challenge in the area of classification is the lack of a coherent framework or model for what we consider a mental disorder (Kendler, 2005, 2008). Without a cohesive perspective on what 'mental disorders' actually are, it is difficult to create classification systems that reflect their nature. In the current section, we evaluate five conceptualisations of mental disorders: the biopsychosocial model, mechanistic property cluster kinds, the disease model, the symptom network model, and the embodied-enactive perspective.

The Biopsychosocial Model

According to the *biopsychosocial model* (BPSM; Engel, 1977), mental disorders are caused by a combination of social, psychological, and biological factors. Rather than focusing on the causal factors from a single domain, the model considers a plurality of factors, including those external to the organism (Zachar & Kendler, 2007). Although this model has provided a heuristic to remind researchers to give attention to the three aspects of an illness, it has not provided any guidance on how to prioritise; there is no indication on how these three elements interact with each other (Ghaemi, 2009). Furthermore, the model offers no account of any mechanisms underlying the interactions between the three sets of variables. In this sense, the BPSM is inadequate as a model of mental disorders, and is best viewed as a framework for undertaking psychological research.

Mechanistic Property Cluster Kinds

Kendler, Zachar, and Craver (2011) argue that *mechanistic property cluster* (MPC) kinds can provide a useful framework for conceptualising mental disorders. MPCs do not have simple, deterministic essences; rather, they are defined in terms of complex, mutually reinforcing networks of causal mechanisms, at multiple levels, that act and interact to produce the key features of the kind. One possibility for a property cluster kind is that the clinical features of a disorder are causally interrelated to one another; for example, suicidal ideation might be caused by both depressed mood and feelings of guilt (Kendler et al., 2011). Another possibility for a property cluster kind is when a series of causal processes (psychological or biological) interact with each other to produce an underlying state that, in turn, leads to the disorders clinical features (Kendler et al., 2011).

There are some limitations to the MPC approach; the model does not provide insight into which mechanisms and causal processes should be emphasised in our nosology. This may make it difficult to decide which disorders to 'lump' together and which to 'split' (Kendler et al., 2011). However, by appealing to causal mechanisms, the MPC model presents a useful framework to improve our classifications by incorporating causal structures into our descriptions of mental disorders.

Disease Model

The disease model posits that mental disorders represent diseases and are the result of pathological processes in specific parts or systems of the brain (Zachar & Kendler, 2007). At the core of the model is the notion of *disease entities*: natural kinds of diseases that medical research is dedicated to discovering, defining, and characterising (Hucklenbroich, 2014). The typical pattern of discovery in medical science begins with observing isolated symptoms or symptom clusters, that are then lumped together into constellations called syndromes, followed by identification of the disease entity by discovering the unifying causal basis of that syndrome (Hucklenbroich, 2017). It follows that identification and definition of disease entities depends on the identification and recognition of its primary cause.

Although the concept of disease entities have featured prominently in medical science, psychiatry is the exception. Currently, most mental disorders do not represent disease entities that have *clear* causal

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(etiopathogenetic) explanations (Hucklenbroich, 2014). While disorders such as depression and schizophrenia *could* be disease entities or clusters of disease entities in the *clinical* sense, many other disorders are at best clinical syndromes with ambiguous boundaries and arbitrary criteria for identification (Hucklenbroich, 2014).

It has been suggested that we should impose the medical model on psychiatry in which we view diseases as *abnormalities* of the body that lead to a stereotypic syndrome presentation; or, alternatively, non-diseases as problems of human experience that are expressed as bodily symptoms (Ghaemi, 2012). While it is important that we align our nosological assumptions in order to better identify those disorders that better represent disease entities, imposing a medical model on psychiatry will be challenging due to the constitutive nature of mental disorders. For disorders like depression, the core symptoms of distress, such as depressed mood and self-reproach, are constitutive of the illness. This makes it difficult to identify a unifying etiopathogenetic explanation of the syndrome.

In addition, a consistent critique of the application of the disease model in psychiatry is the prioritisation of neurological abnormalities and subsequent neglect of non-biological factors such as behavioural processes. However, Murphy (2016) suggests that while intentional phenomena (i.e., mental states like beliefs and desires) *do* produce an additional level of explanation in psychopathology, we can extend our traditional thinking of pathology (i.e., in terms of tissue, such as lesions or disrupted synaptic connections) to also include properties of cognitive systems.

Symptom Network Model

An alternative conceptualisation is the *symptom network model* (SNWM; Borsboom, 2017; Borsboom, Cramer, & Kalis, 2018), in which mental disorders are not considered to be disease entities; rather, they are hypothesised to arise from the causal interaction between symptoms in a network. This view of mental disorders is mereological: the symptoms are parts of a larger system of symptoms and causal connections that we refer to when we use the word 'depression'. In the example of depression, an adverse life event, such as the loss of a partner, may activate symptoms in the network, such as depressed mood, which, in turn, may cause neighbouring symptoms, such as insomnia and fatigue, to be activated (Borsboom, 2008).

The SNWM is a novel departure from the emphasis on syndromes and presents a useful opportunity to better identify patterns of symptoms and understand their relationship with each other. The SNWM also presents a view of *comorbidity* as an intrinsic feature of mental disorders. Although symptom-symptom interactions will be most active within symptom sets commonly associated with a given mental disorder, the presence of *bridge symptoms* that belong to more than one disorder network (e.g., fatigue in MDD and GAD) means that comorbid patterns of symptom interactions may occur (Borsboom et al., 2018).

While the SNWM provides a description of the *relationships* among symptoms it is *agnostic* as to how the causal relations between the symptoms are actually instantiated. This is where the SNWM differs significantly from the MPC approach. Unlike the MPC model, the SNWM is not committed to understanding the particular mechanisms that generate the network structure and the symptoms themselves (Borsboom, 2017). While the MPC approach is best understood as an explanatory model that is set out to represent the mechanisms that produce symptoms, the SNWM may be more usefully construed as a *phenomenal model*, suited to detecting patterns among symptoms (Craver & Kaplan, 2018; Hochstein, 2016b).

Embodied-Enactive Perspective

Traditionally, cognitive science has utilised a software-hardware distinction, in which the brain is viewed as the hardware and the mind/cognitive processes as the software. This has led to the notion that mental disorders are the result of functional 'bugs' in the software and that the mind can be studied in abstraction from the brain (Drayson, 2009). Alternatively, mental disorders may be better

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represented by an *embodied* approach to the mind (Colombetti, 2014; Fuchs, 2009; Gallagher, 2017; Maiese, 2016). In contrast to the traditional orthodox of cognitive science, the *embodied* perspective argues against the computational stance in favour of studying the mind in the broader context of the embodied and situated nature of the person. Understanding the mind will require understanding the brain, embedded in the body, within its wider environment. From this viewpoint, mental disorders are conceptualised as disorders of embodied brains that are embedded in their natural and social environment (Drayson, 2009).

The embodied perspective may better capture the nature of psychopathology, as the causal factors relevant to mental disorders frequently extend beyond the brain to include the body and the environment. For example, depression includes dysfunctions in higher level cognitive processes, such as abstract thought and memory, but also lower level bodily symptoms, such as psychomotor retardation and changes in sleep (Drayson, 2009; Fuchs, 2009). One potential difficulty in adopting an embodiment perspective is that the level of complexity it assumes makes it hard to study specific psychopathological processes in isolation from the whole in which they are embedded.

Summary

Although the BPSM reminds us to pay attention to biological, psychological, and social factors, it is inadequate as a model of mental disorder. The SNWM faces similar challenges; although it better identifies patterns of symptoms, the model ignores the casual processes that underpin them. MPC kinds presents a useful alternative that appeals to the relationship between causal mechanisms and the phenomena they produce; however, the model fails to specify which mechanisms to prioritise. While the disease model does prioritise neurological mechanisms, its suitability varies across the range of mental disorders. The embodied-enactive perspective, in its current state, functions less as a model of mental disorder but does provide a novel perspective on studying the mind.

Explanatory Perspectives

Ultimately the concept of a mental disorder should not only serve to describe but also to explain why peoples' thoughts and behaviours are abnormal (Thagard, 2016). For example, across the history of psychiatry, there have been various explanations of depressive conditions. The ancient Greeks introduced the term 'melancholia' to suggest that depression is caused by an excess of black bile. In the late 19th century, Kraepelin focused on the melancholic type of depression, linking it with mania under the general category of manic-depressive conditions. Kraepelin believed psychiatric disorders are disease entities with a specific etiology and pathology. Freud, on the other hand, considered depression to be a psychological problem resulting from a reaction to loss (Horwitz et al., 2017; Paykel, 2008). During the cognitive revolution of the 1950s and 1960s, greater research in psychology began to focus on the role of thought processes in human experience. This led to the development of cognitive theories of psychopathology that focused on the mediating role of maladaptive cognitions in the development of disorders such as depression (Alloy, Salk, Stange, & Abramson. 2017).

Over the last 50 years there have been significant advancements in our understanding of depression, particularly in the field of neurobiology. Genetic research has pointed to specific genotypes that may place certain individuals at risk of developing the disorder (Berrettini & Lohoff, 2017). Imbalances in neurotransmitters, such as serotonin and dopamine, have played prominent roles in the treatment of depressive symptoms (Cowen, 2017; Pringle & Harmer, 2017). Endocrine abnormalities, including dysfunction in the production of cortisol and increases in the levels of inflammation, have also been implicated in the development of depression (Cowen, 2017; Maletic & Raison, 2017). While structural abnormalities, such as neural atrophy in the hippocampus, and functional abnormalities in key brain regions, such as the amygdala, have also been associated with depression, with specific implications regarding their role in cognitive processes such as memory and attention (Newman, Bauer, Soares, & Sheline, 2017). And most recently, advancements in the understanding of neural

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networks have also been applied to the field (Newman et al., 2017). Despite the wealth of research into the causal factors and processes that may cause or constitute depression, there is still a *lack* of consensus on how best to *build explanations* of the disorder. We explore a range of explanatory strategies/perspectives that aim to offer the best account of mental disorders such as depression.

Biological Reductionism

Over the last few decades in psychiatry, there has been a rise in the biological reductionist perspective (Bergner, 2004; Borsboom et al., 2018). From this perspective, mental disorders or, more broadly, psychological functioning, is best explained in terms of basic neurobiological processes (Bickle, 2003; Pennington, 2014). As a result, multilevel models, especially those including psychological and social explanatory perspectives, are often rejected or are only accepted with the caveat that the 'real' causal effects occur at the level of biology (Kendler, 2005).

However, mental disorders are unlikely to be amenable to purely reductionist explanations of psychopathology (Mitchell, 2009). Understanding some mental disorders will require consideration of factors at psychological and social levels of analysis. For example, in depression, attempting to understand first-person experiences, such as humiliation and loss, at the level of neurobiology is unlikely to be the most efficient level at which to characterise these experiences (Kendler, 2005). Although these experiences are ultimately expressed in the brain, they carry important causal information about human behaviour that cannot simply be reduced to neurobiology (Kendler, 2005). For example, high-threat events that combine elements of both loss and humiliation (e.g., a separation initiated by another individual) better predict depression onset than high-threat events that feature only one element (Kendler, Hettema, Butera, Gardner, & Prescott, 2003). Additionally, those events that involve a loss of status (e.g., a separation initiated by the respondent) are more depressogenic than those involving solely loss (e.g., a death). Mental disorders are frequently characterised by intentional information, such as the descriptions of mental states like beliefs, desires, and emotions, which cannot successfully be characterised as neurobiological phenomena (Bergner, 2004; Ward & Clack, 2019).

Unification

Many theorists have sought to integrate research findings across varying domains and build *unified* theories of mental disorders. In the field of depression research, the most recent example is Beck and Bredemeier's (2016) unified model of depression, which attempts to integrate the clinical features of depression, advances in neurobiology, and evolutionary perspectives of the disorder within an overarching cognitive framework. The model is unique in its ability to provide a systematic, detailed account of the full range of symptomology of depression (including atypical symptoms and adaptive functions) and the natural progression of depression from predisposition to recovery. Despite these strengths, a comprehensive appraisal of the model's coherence and empirical adequacy has revealed some important challenges that need to be addressed (see Clack, Wilshire, & Ward, 2019). These include clarification of the adaptive nature of depression, understanding the role of information processing, and privileging of the cognitive level of analysis. Many of these challenges are built on core assumptions that arise by presenting the model from a cognitive-evolutionary framework. Furthermore, an overarching challenge for a *unified* account of psychopathology is how to integrate the different scientific domains (e.g., psychology, neuroscience, evolutionary biology, genetics, sociology), as each identifies a different dimension of causal influence (Hochstein, 2016a). We discuss one way of linking different explanatory models in the next section.

Explanatory Pluralism

Mental disorders are complex psychological phenomenon and involve multiple causal processes impacting on different levels, both inside and outside of the individual, and are often further

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complicated by interactions between levels (Kendler, 2008; Maletic & Raison, 2017). The evidence that mental disorders are multifactorial has led many researchers to opt for a form of explanatory pluralism; that is, psychiatric disorders are viewed as the result of causal processes at the biological, psychological, and social level (Hochstein, 2016b).

An example of such an approach is Kendler, Gardner, and Prescott's (2006) developmental model of MDD, in which a number of major causal factors, from different levels or domains interacting over time, have been identified. These include factors such as *developmental adversity* (e.g., early loss, childhood sexual abuse, and low parental warmth), *genetic vulnerability* (e.g., family history of depression) and *psychological variables* (e.g., low self-esteem and early-onset anxiety).

A specific form of explanatory pluralism that may be particularly useful in the field of psychopathology is *integrative pluralism* (Mitchell, 2003; 2009). Integrative pluralism suggests that theories at different levels of organisation, levels of analysis, and domains of inquiry (e.g., psychological, developmental, cultural, biological) can neither be reduced nor stand in isolation if we are to advance our explanatory understanding. As a methodological process, integrative pluralism is the process of establishing links between local theories across multiple levels of explanation (Mitchell, 2003). An important example in the field of depression is the research carried out by Caspi et al. (2003) demonstrating the interaction between specific genotypes and environmental adversity in the development of depression.

Carrying out integrative work in psychopathology is not an easy task; it requires incorporating research from different fields and relies on sufficient knowledge across multiple levels of analysis to explain a range of diverse phenomena. Integrative work faces ideological obstacles; many psychologists resist incursion of neuroscience and biology into their preferred level of analysis (Lilienfeld, 2007). There are also cognitive obstacles to integrative work (Lilienfeld, 2007); thinking in terms of *vertical integrations* across levels of analysis (e.g., understanding depression at the level of neural circuits) is more difficult than *horizontal integration* of etiological variables at the same level (e.g., understanding depression at the level of negative schemas).

Summary

The biological reductionist perspective has been heavily criticised for omitting important causal and constitutional information, particularly from psychological and social levels. In response, researchers have argued for more pluralistic or unified explanations that incorporate information from multiple levels. Although the idea of a unified model is attractive, the idealisation of our current theories makes this difficult to achieve — altering our current models and theories to provide a unified account of depression means we run this risk of losing value in these explanations. Pluralistic and integrative explanations are difficult to develop. However, integrative work is important for scientific progress, particularly when attempting to provide coherent explanations of complex constructs such as depression (Hochstein, 2016b).

Improving our Classifications and Explanations

The classification and explanation of mental disorders, and the relationship between these two scientific tasks, has been a contentious issue in psychiatry. For example, Bolton (2012) argues that while classification is an important task in psychiatry, what is essential is to have common concepts and language that ensure reliability and generalisability of our research. Additionally, he argues that psychiatry has been over-occupied with classification, which is less important than other tasks in science such as predication and explanation.

Ghaemi (2012), on the other hand, argues that the focus on *pragmatism* in our current nosology prevents the discrimination between those conditions that are diseases and those that are social constructs. He argues that the main goal of our nosology should be to aid the discovery of the diseases that are causing the symptoms of psychopathology. He adds that research based on current classifications is

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limiting our biological research; if our phenotypes are inaccurate then our explanations will suffer as a consequence (Ghaemi, 2012).

Classification and explanation are not completely independent scientific tasks; the way we *classify* mental disorders directly impacts how we *explain* them, which in turn impacts our classifications. The problem is that psychiatry has become stuck in a circular trap; *DSM* categories form the explanatory targets for our research and clinical practice, which is then used to explain mental disorders.

The way in which we classify mental disorders may also restrict what explanatory strategies can be successfully utilised. For example, a failure to provide reductionist explanations of mental disorders may be the direct consequence of our classification systems. Our ability to detect highly specific and sensitive biomarkers of mental disorders may not be because biological reductionism is a poor strategy, but that our explanatory targets (i.e., diagnoses) are imprecise (Lilienfeld & Treadway, 2016).

Improving our current classifications in psychiatry is not an easy task. A significant challenge is how can we increase our understanding of mental disorders and advance our classification systems without abandoning the descriptive value of our current classifications and the decades of research that has been a product of it.

Traditionally, research into mental disorders has focused on understanding psychiatric syndromes as illustrated in diagnostic manuals. However, there has been little discussion about the nature of the symptoms and signs themselves that constitute mental disorders. While the SNWM offers a novel departure from the emphasis on syndromes, the model is only *descriptive* and offers no *explanatory* account of the processes that constitute symptoms.

We suggest that *one* way of advancing our understanding of mental disorders is to move our focus from syndromes and symptom clusters to *clinical phenomena*. Phenomena are the relatively stable, recurrent general features of the world that we seek to explain (Haig, 2014). On account of their generality and stability, phenomena are useful targets of scientific explanation. Examples of general phenomena in clinical psychology include low self-esteem, aggression, low mood, and ruminative thoughts. These phenomena are usefully construed as empirical regularities and are inferred from data sources such as behavioural observation, self-report, and psychometric test scores.

Phenomena Specification

Developing our understanding of mental disorders will require careful consideration of the way in which we describe and classify them. The majority of psychopathology research currently revolves around mental disorders as defined and operationalised in the *DSM-5* (Berenbaum, 2013). However, due to the uncertainty and heterogeneity surrounding our current classifications, simply interpreting associations at the level of diagnoses can be misleading.

For example, research demonstrating an increase in depressive symptoms following IFN-α treatment in humans has been used to support the hypothesis that inflammation can *cause* depression (Capuron & Miller, 2004; Harrison et al., 2009). However, in studies that have examined response to IFN-α treatment, nearly all patients who receive treatment appear to experience a sudden onset of *neuro-vegetative* symptoms, while those who developed *cognitive and affective* symptoms of the depression were more likely to have experienced a prior depressive episode (Capuron & Miller, 2011; Harrison et al., 2009). Therefore, the evidence *only* suggests that inflammation plays a role in the onset of neuro-vegetative symptoms that are *also observed* in depressed states such as fatigue. Working only at the level of diagnostic categories would run the risk of falsely concluding inflammation *causes* depression. A more accurate conclusion would be that somatic 'depressive'-like symptoms associated with inflammation are simply part of an inflammation induced response.

In order to improve our explanations of depression, and other mental disorders, we need greater specification of the clinical phenomena (i.e., symptoms, signs, or problems) our models and research seek to explain. It is not enough to conclude that finding X is associated with depression, as the features of depression may vary significantly between individuals. Rather, we need to *specify* the depressive phenomena under investigation in subjects. This also applies when developing theoretical models;

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it is necessary to clarify the specific depressive phenomena our models attempt to explain otherwise we may be offering different explanations of varying constructs.

Towards Multimodel Explanation of Clinical Phenomena

Rather than constructing a 'theory' of depression and limiting ourselves to a specific level of analysis or altering models or theories to provide a single unified account of depression, we should aim to develop a multilevel explanation of depression that incorporates mutually supportive models.

In addition, we suggest that rather than attempting to build explanations of disorders as currently defined in the *DSM-5*, we should seek to construct detailed explanations of the *key* phenomena that constitute mental disorders. Conceptualising the symptoms of mental disorders, such as depressed mood, as manifestations of a real pathological condition (i.e., as *clinical phenomena*) can assist us in directing research into their structure and relationships. In addition, by adopting model pluralism we can build a comprehensive and multifaceted explanation of the processes that constitute phenomena.

This can be achieved using the Phenomena Detection Method (PDM; see Ward & Clack, 2019). The PDM provides a way to 'bootstrap' the explanation of the symptoms of mental disorders, moving from thin descriptions of client concerns to rich representations of clinical phenomena. Its purpose is to aid our understanding of the structures and processes constituting mental disorders by analysing their central symptoms (and signs). A full outline of the method is beyond the scope of the current article, but for a full description see Ward and Clack (2019).

An adequate conceptualisation of *depressive phenomena* should involve the gathering of information at all relevant levels, including biomolecular components, neural networks, psychological functions, and social processes. This would include both *etiological explanations,* which aim to depict the mechanisms that *cause* the phenomenon of interest, and compositional explanations, which describe the components of a mechanism and their interactions that constitute a phenomenon. For example, an etiological explanation of depressed mood would illustrate the series of processes, such as low parental warmth, early loss, and low self-esteem, that over time lead to its onset and progression. On the other hand, a compositional explanation would describe the different processes and structures, such as negative cognitions or activations in the amygdala-hippocampal area, that compose depressed mood. Achieving this level of description allows for the gradual assembly of a coalition of local models that are linked to collectively provide a comprehensive way of understanding the phenomenon (Potochick, 2017).

Because of the complex nature of phenomena, we suggest representing their different levels or aspects using multiple models (Potochnik, 2017). For example, a *family* of models are needed to explain the phenomenon of depressed mood, including behavioural models (e.g., embodiment theories), physiological models (e.g., sympathetic withdrawal), neural models (e.g., affective networks, cortical-subcortical imbalance), psychological models (e.g., mood-dependent memory, cognitive theory), phenomenological models (e.g., Heidegger's moods), and so on. Each idealised model highlights central processes at a particular scale (level) and ignores others. It is unlikely that a unified theory will be produced at this point; rather, the output is a coalition of 'friendly' models each focusing on a specific set of processes and structures at varying spatial scales and levels of abstraction (e.g., negative self-appraisal vs. amygdala-hippocampal activation). There is a variety of different representations and explanations of a phenomenon, each best suited for a different goal or purpose (Potochnik, 2017). The *type* of explanation sought is compositional; the aim is to understand how the clinical phenomenon under investigation is constituted. However, etiological concerns can be linked in to the existing multimodel explanation in order to illustrate the series of processes that over time lead to the onset and progression of this phenomenon.

Developing explanations of multiple depressive phenomenon (e.g., depressed mood, anhedonia, self-reproach) in this way would improve our overall understanding of depression and clarify the way different depressive phenomena are related to each other within the construct. It is a bottom-up

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approach of identifying and explaining mental disorders that side-steps problems associated with traditional diagnostic categories.

Phenomena-Based Classification

Developing explanations of psychopathology phenomena may also positively impact on how we classify mental disorders such as depression. Introducing causal information into our classifications may help alleviate some of the uncertainty around our current categories. It has been suggested that by distinguishing syndromes on the basis of etiology and pathology, causal explanation may be able to help deal with the arbitrary distinctions seen in categorical classification and allow us to discriminate more finely among conditions that are currently grouped together (Murphy, 2016). Unambiguously classifying mental disorders as a function of their cause is difficult considering their multifactorial nature (Zachar & Kendler, 2007). However, by developing explanatory models for a set of interrelated phenomena, we can begin to introduce relevant causal information for the *specific* phenomena that make up our categories. Additionally, those phenomena that share similar or related compositional and causal processes are more likely to form reliable clusters.

A classification system built around models of clinical phenomena would likely vary significantly from our current categories; many symptoms could disappear if research evidence fails to demonstrate they represent stable features of individuals in distress, while others may be absorbed into different clinical phenomena. For example, depressed mood and anhedonia could on further description collapse into one phenomenon or be even further subdivided into three or four distinct phenomenon (e.g., loss of meaning, diminished experience of pleasure, despair, and sadness).

Understanding the mechanisms that comprise phenomena means that categories based on clusters of phenomena would be more likely to warrant reliable inferences concerning the additional properties (e.g., other symptoms) and the course of a disorder. In this sense, diagnosis would be more of an explanatory task (as seen in medicine), reducing the dangers of relying on syndrome allocation to determine treatment.

Conclusion

The classification and explanation of mental disorders has been fraught with challenges and controversy. While the categorical approach has been the most prominent model of classification in psychiatry, many researchers have argued that disorders such as depression should be seen as dimensional. Current manuals, namely the *DSM-5*, have been heavily criticised because of their problems of disorder heterogeneity and overlapping diagnostic criteria. While novel approaches such as the HiTOP and RDoC better accommodate the dimensional nature of mental disorders and incorporate causal information, they face conceptual limitations (i.e., HiTOP's reliance on current diagnostic categories and the RDoC's neuro-centrism).

An ongoing challenge is the lack of a coherent framework for what we consider a mental disorder. While the BPSM may provide a useful a framework for undertaking psychological research, its failure to specify the mechanisms underlying the interactions between the three domains (i.e., biological, social, psychological) makes it inadequate as a model of mental disorder. MPC kinds appeal to the relationship between causal mechanisms and phenomena but fails to specify which mechanism to prioritise. In contrast, the disease model prioritises neurological mechanisms, but its suitability across the range of mental disorders has been questioned. The SNWM of mental disorders provides a useful descriptive approach to better identify patterns of symptoms. However, it is agnostic to understanding the casual processes that underpin symptoms and suffers as an explanatory model.

Despite the rise in reductionist explanations of mental disorders, their omission of important causal and constitutional information from differing levels of analysis limits their use as a comprehensive explanatory strategy. This has created space for more pluralistic and integrative explanations of mental disorders that incorporate information from multiple levels of analysis. Although the idea of a unified model is attractive, it is unlikely to be achieved due to the idealisation of our current theories.

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The relationship between classification and explanation presents a unique challenge to explaining depression and other mental disorders. Namely, our current classifications are frequently taken for granted in research and theory; if our categories of depression are invalid then our explanations will suffer as a consequence. In turn, our reliance on current categories forming the explanatory targets of our research makes it difficult to modify and improve our classifications.

By moving our focus from describing and explaining syndromes and symptom clusters to describing and building explanations of clinical phenomena, we can improve our understanding of mental disorders and sidestep many of the current challenges described. This will require us to specify the phenomena our research and theories seek to explain; develop multilevel explanations of clinical phenomena that incorporate mutually supportive models; and build classifications systems around models of clinical phenomena that would make diagnosis more of an explanatory task.

There is an apparent need to revolutionise our current approaches to classification and to utilise better explanatory strategies. However, achieving this is no easy task. On one hand, our psychopathology research has relied on disorders as defined and operationalised in the *DSM*; but it this reliance on these poorly defined categories that has limited our ability to build coherent explanations. The advantage of building descriptions and explanations of *clinical phenomena* is that as our knowledge of the central processes that underpin the relevant phenomena increases, we can begin to *modify* our current classifications without the need to completely *abandon* their descriptive value and the existing research that has been a product of them. In short, we can provide a more *secure* basis for the relationship between signs, symptoms, and pathological conditions.

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