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The Role of Causal Knowledge in Reasoning About Mental Disorders

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Abstract and Keywords

Despite the lack of scientific consensus about the etiologies of mental disorders, practicing clinicians and laypeople alike hold beliefs about the causes of mental disorders, and about the causal relations among symptoms and associated characteristics of mental disorders. This chapter summarizes research on how such causal knowledge systematically affects judgments about the diagnosis, prognosis, and treatment of mental disorders. During diagnosis, causal knowledge affects weighting of symptoms, perception of normality of behaviors, ascriptions of blame, and adherence to the DSM-based diagnostic categories. Regarding prognosis, attributing mental disorders to genetic or neurobiological abnormalities in particular engenders prognostic pessimism. Finally, both clinicians and laypeople endorse medication more strongly as an effective treatment if they believe mental disorders are biologically caused rather than psychologically caused. They also do so when considering disorders in the abstract versus equivalent concrete cases. The chapter discusses the rationality, potential mechanisms, and universality of these phenomena.

Keywords: causal, diagnosis, prognosis, treatment, mental disorder

Lifetime prevalence rates for mental disorders are surprisingly high—in the double digits in every country examined by the World Health Organization (WHO; Kessler et al., 2007). In the United States, the country with the highest lifetime prevalence studied by the WHO, about half of the population has had a diagnosable mental disorder at least once throughout the life span. In any given year, approximately one-quarter of adults in the United States meet diagnostic criteria for one or more disorders (Kessler et al., 2005). In addition, mental disorders have been identified as one of the three most costly categories of medical conditions (Keyes, 2007); Insel (2008) estimated the direct and indirect

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economic burden of serious mental illness at around $317 billion annually in the United States alone.

Unfortunately, the causes of mental disorders are still unclear and controversial, and consensus about the etiologies of mental disorders has eluded researchers. Thus, the Introduction of the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*; American Psychiatric Association, 2013), which serves as the official nosology of mental disorders in the United States and a number of other countries, states that "a complete description of the underlying pathological processes is not possible for most mental disorders" (p. xi). Despite rapid gains made by research in clinical neuroscience, "scientists have not identified a biological cause of, or even a reliable biomarker for, any mental disorder" (Deacon, 2013, p. 847). Although many mental disorders have a heritable component, few specific genes have been associated with any mental disorder; namely, "we do not have and are not likely to ever discover 'genes for' psychiatric illness" (Kendler, 2005, p. 1250).

Given this lack of scientific consensus, an important question is how practicing mental health clinicians and laypeople conceptualize the etiologies of (p. 604) disorders, and how such conceptualizations subsequently affect their diagnostic and clinical reasoning. We begin by discussing causality in clinicians' and laypeople's concepts of mental disorders. After reviewing evidence suggesting an abundance of different causal theories about mental disorders, the bulk of this chapter is devoted to reviewing the consequences of such causal explanations. These reviews are organized based on three critical aspects of reasoning when dealing with mental disorders: diagnosis, judgments of prognosis, and decisions about treatment.[1] We review both clinicians' and laypeople's reasoning whenever possible. In addition, most of the effects of causal knowledge demonstrated in this domain may not be restricted to reasoning involving mental disorders. Instead, they may stem from more domain-general cognitive biases, which we include in our discussion.

Causality in Mental Disorder Concepts

Despite the lack of scientific consensus about the etiologies of mental disorders, practicing clinicians as well as laypeople appear to have beliefs or hunches about the causes of mental disorders, and about the causal relations among symptoms of mental disorders. For example, when Kim and Ahn (2002b) asked practicing clinicians to specify any relations at all among the symptoms within mental disorders, 97% of all relations that the clinicians drew and labeled were causal relations or relations that imply causality (Carey, 1985; Wellman, 1990). This finding suggests that causality is important in mental-health clinicians' concepts of mental disorders. For familiar disorders such as major depression, anorexia nervosa, and borderline personality disorder, clinicians were also reasonably in agreement with each other regarding the causal structure of the symptom-

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to-symptom relations in the disorder. Laypeople also agreed with the general causal structure of clinicians' theories, suggesting that these theories (at a broad level) are understandable in common-sense terms (Ahn & Kim, 2005; Kim & Ahn, 2002a).

The causality inherent in people's concepts of mental disorders suggests that people may essentialize them (Medin & Ortony, 1989). Namely, do people believe that mental disorder categories have underlying, fundamental essences that cause the surface symptoms, such that within each mental disorder, a single essence shared by all instances of the disorder serves as a common cause for the surface symptoms (e.g., Ahn et al., 2001; Waldmann & Hagmayer, 2006; see top panel of Figure 30.1 for an illustration)? Borsboom and Cramer (2013) argued that, unlike medical diseases, mental disorders do not have common-cause structures in reality. To understand this contrast, consider a brain tumor that causes headaches, forgetfulness, and foggy eyesight. Here, the tumor is the root cause of the symptoms, and is separate from the symptoms occurring as a consequence of that cause. For instance, one can have headaches without a brain tumor, and a brain tumor can conversely exist without headaches. However, consider a mental disorder, such as major depression (MD), and its core symptoms (e.g., feeling sad or disinterested). It is highly unlikely that one can be depressed without feeling sad or disinterested; rather, depression has been defined by its symptoms (Borsboom & Cramer, 2013; but see Bhugra & Mastrogianni, 2004, for a consideration of cross-cultural differences). Similarly, in the case of a substance use disorder, the presence of the symptoms (e.g., using a substance) is necessary to say that the disorder is present. So, Borsboom and Cramer (2013) argued that, rather than being represented in a common-cause structure wherein symptoms are only directly causally connected to the essence, (p. 605) mental disorders have symptom-to-symptom causal relations in reality (as can be seen in many Bayesian network representations discussed elsewhere in this volume: Griffiths, Chapter 7; Rehder, Chapter 20; Rottman, Chapter 6; see the bottom panel of Figure 30.1 for an illustration).

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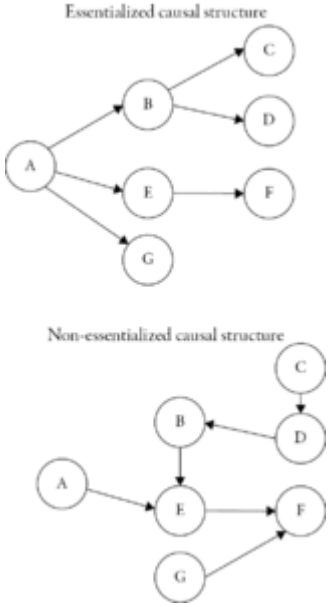

Figure 30.1 Illustration of essentialized and non-essentialized causal structures.

Note that Borsboom and Cramer's (2013) discussion is concerned with the metaphysics of mental disorders, rather than with people's beliefs about mental disorders per se. Do people represent mental disorders as causal networks of symptoms or separate sets of common-cause structures? Ahn, Flanagan, Marsh, and Sanislow (2006) found that laypeople (i.e., undergraduate students) generally essentialize mental disorders, seemingly adhering to the notion of underlying common-cause structures. For example, laypeople in their study believed that for a given mental disorder, there must be an underlying cause that is necessary and sufficient that also causes surface symptoms. Practicing clinicians, however, were more ambivalent about such statements, neither agreeing nor disagreeing with them. The reasons for such ambivalence are unclear; Ahn et al. (2006) used *DSM-IV* disorders that were judged to be familiar to clinicians and laypeople, but it is possible that the *DSM-IV* taxonomy itself does not correspond to clinicians' own taxonomy. For instance, rather than being construed as a single essentialized disorder, major depression may be construed by clinicians as having several distinct subtypes, each of which may be essentialized as a relatively distinct "disorder."

Effects of Causal Knowledge on Diagnosis

How does causal knowledge affect the diagnosis of mental disorders? In the United States, formal diagnoses of mental disorders are made based on the *DSM*. As mentioned earlier, etiologies in psychopathology are controversial. As a result, the modern editions of the *DSM* (i.e., *DSM-III*, APA, 1980; *DSM-III-R*, APA, 1988; *DSM-IV*, APA, 1994; *DSM-IV-TR*, APA, 2000; *DSM-5*, APA, 2013) have all adopted a descriptive approach intended "to be neutral with respect to theories of etiology" (APA, 1994; pp. xvii–xviii). Thus, most mental disorders in the *DSM-5* (APA, 2013) are currently defined in terms of a set of surface symptoms or conditions the patient must meet for diagnosis (in addition to functional impairment).

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For example, schizophrenia is defined as having two or more of the following five symptoms (along with an impaired level of functioning): hallucinations, delusions, disorganized speech, grossly disorganized or catatonic behavior, and negative symptoms. Thus, if clinicians follow the prescribed diagnostic approach of the *DSM*, they will search for symptoms in their patients that match the *DSM* diagnostic criteria and make diagnoses accordingly, without incorporating any additional notions they may have of how these symptoms may affect each other and, in many disorders, what caused these symptoms in the first place. Furthermore, in most cases, all symptoms are weighted equally in the *DSM*.

Nevertheless, causal knowledge affects various aspects of diagnoses of mental disorders. In this section, we review how causal knowledge determines which symptoms are seen as especially important, which can eventually affect how mental disorders are diagnosed. In addition, we review how causal knowledge affects judgments of how abnormal a person's behaviors are. While the aforementioned topics concern effects of existing causal knowledge, we also review recent studies on factors affecting the way that laypeople and clinicians infer biological or psychological bases of mental disorders in the first place.

Effects of Causal Knowledge on Feature Weighting: Causal Status Effect

Kim and Ahn (2002a, 2002b) found from both clinicians and laypeople that when symptoms are causally related, the ones that are seen as causing other symptoms are regarded as more important and essential to the disorder concept than their effects. We have called this general phenomenon the *causal status effect* (Ahn, 1998). Namely, a feature serving as a cause in a causal relationship is considered more central than a feature serving as an effect, when all else is held equal (see Ahn & Kim, 2000; Sloman, Love, & Ahn, 1988, for a discussion of additional factors that should be expected to interact with the causal status effect in determining feature centrality). For instance, according to *DSM-IV*, "distorted body image" and "absence of period (in women) for more than 3 menstrual cycles" were both required in order to warrant a diagnosis of anorexia nervosa, making these two symptoms equally important for classification.[2] However, according to data collected from clinicians by Kim and Ahn (2002a), "distorted body image" was most causally central in the clinicians' theories, whereas "absence of the period (in women) for more than 3 menstrual cycles" was rated the most causally peripheral. Furthermore, "distorted body image" was considered to be the most diagnostically important of the criteria, and (p. 606) "absence of the period (in women) for more than 3 menstrual cycles," though also a *DSM-IV* diagnostic criterion for anorexia nervosa, was considered to be the least diagnostically important.

The causal status hypothesis can readily be intuitively understood, and its real-life examples are abundant. We tend to form an illness concept based on the virus (e.g., Ebola; influenza) that causes the symptoms (e.g., fever; coughing) rather than by the symptoms per se. DNA structure causes many other properties of plants and animals

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(e.g., appearance; mechanism of reproduction), and hence is considered the most important feature in their classification. Indeed, after DNA sequencing became available, some species were reclassified in the Linnaean taxonomy. For example, the domestic dog was once considered its own separate species, but in 1993 the Smithsonian Institution and the Society of Mammalogists reclassified it as a subspecies of the gray wolf, largely based on genetic information that had become newly available. In law, the severity of crimes often depends more on the suspects' intentions rather than their surface behaviors (e.g., conspiracy to commit murder is typically a much more serious offense than involuntary manslaughter, even if the victim died only in the second case). In judging whether people are nice or not, we tend to place more weight on their intentions (i.e., what motivated or caused their actions) rather than on what they did.

Indeed, the causal status effect has been demonstrated not only in the domain of mental disorders, but also with controlled artificial stimuli (e.g., Ahn, Kim, Lassaline, & Dennis, 2000) and across various types of categories (see Ahn & Kim, 2000, for a more detailed overview of the causal status effect). For instance, different features are central for natural kinds and artifacts: in natural kinds, internal or molecular features are more conceptually central than functional features, but in artifacts, functional features are more conceptually central than internal or molecular features (e.g., Barton & Komatsu, 1989; Gelman, 1988; Keil, 1989; Rips, 1989). Ahn (1998) showed that the causal status effect can explain this phenomenon. In natural kinds, internal/molecular features tend to cause functional features (e.g., cow DNA determines whether or not cows give milk) but for artifacts, functional features determine compositional structure (e.g., chairs are used for sitting, and for that reason, they are made of a hard substance).

The causal status effect appears to be fairly cognitively primitive, as shown by two sets of studies. First, the effect has been demonstrated with young children (Ahn, Gelman, Amsterlaw, Hohenstein, & Kalish, 2000), although it might not be innate, as Meunier and Cordier (2008) found the causal status effect with 5-year-olds but not with 4-year-olds in biological categorization. Second, although a number of studies had supported the notion that theory-based categorization may be a fundamentally slower, more deliberately executed process than similarity-based categorization (e.g., Smith & Sloman, 1994), Luhmann, Ahn, and Palmeri (2006) found the causal status effect even under speeded conditions for categorization. They showed that the time required to make judgments using causal knowledge or theory was equivalent to that using base-rate information of features, which is traditionally considered a more rapid, associative reasoning process.

In the domain of mental disorder diagnosis, Flores, Cobos, López, Godoy, and González-Martín (2014) examined whether the use of causal theories is the result of fast and automatic processes that take place very early on in clinical reasoning to comprehend cases, or slow, deliberate processes triggered only when clinicians are asked to make a diagnostic judgment. To test these possibilities, they presented clinicians with information that was either consistent or inconsistent with widely accepted causal theories and measured clinicians' reading times. For instance, according to a relatively well-established causal theory in clinical science regarding eating disorders, a strong fear

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of gaining weight (X) causes a refusal to maintain a minimal body weight (Y), which in turn causes overt behaviors such as a strict diet, vomiting, and laxative abuse (Z). Therefore, a clinical report stating that X is followed by Y, which is followed by Z, would be consistent with this causal theory, whereas Z followed by Y, which is followed by X, would be inconsistent. The idea is that inconsistent ordering, as in the latter case, would slow down the clinicians' reading times if their causal theories are spontaneously activated while reading the report. The authors found that temporal order manipulated based on the causal theories affected clinicians' reading times but not the reading times of students (who presumably do not have pre-existing causal theories).

Rationale for the Causal Status Effect

Why would causally central features be conceptually central as well, as repeatedly shown in demonstrations of the causal status effect within and outside of the mental disorder domain? One of the most (p. 607) important functions of concepts is to allow us to make inductive inferences about unknown features (e.g., Anderson, 1990). People appear to believe that given X causing Y, hypothetical features associated with the cause (X) (e.g., other effects besides Y that might be caused by or associated with X) are more likely to be present than unknown features associated with the effect (Y), even when the associative strengths are equal (Proctor & Ahn, 2007).

Suppose a clinician learned that his patient has chronic feelings of emptiness (feature X), and she devotes herself to work to the exclusion of friendships and leisure (feature Y). The clinician might spontaneously infer that this patient has chronic feelings of emptiness because she is excessively devoted to work (Y→X). The idea is that given this causal knowledge, the clinician would believe a potential symptom associated with the cause feature (e.g., paying extraordinary attention to checking for possible mistakes, which might be associated with excessive devotion to work) is more likely to be true than an unknown symptom associated with the effect feature (e.g., impulsive harmful actions, which might be associated with chronic feelings of emptiness). A different clinician might infer a different causal relation, for example, that this patient is excessively devoted to work because she chronically feels emptiness (X→Y). Given the opposite causal direction, the opposite inferences would be made; the clinician would believe that impulsive harmful actions would be more likely to be true than paying extraordinary attention to checking for possible mistakes.

Proctor and Ahn (2007) tested this idea by experimentally manipulating the causal relations of identical features. Participants—students and practicing clinicians—were first presented with a pair of mental disorder symptoms for a patient. In one condition, they were told that symptom X causes symptom Y. In the other condition, participants learned that Y causes X. Then participants were asked about the likelihood that the target person would have other symptoms. One of the questions asked about the likelihood of a feature (X' henceforth) that is judged to be more strongly associated with X than with Y. Similarly, Y' was judged to be more strongly associated with Y than with X. When they were told

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that X causes Y, participants judged that X' would be more likely to be present, whereas when told that Y causes X, they judged that Y' would be more likely to be present. That is, their inductive judgments of an unknown feature were guided by the causal status of features with which the unknown features were associated. Note that these results are difficult to explain by a purely associationist account, because the associative strength between X and X' and between Y and Y' were equated in the study (see Le Pelley, Griffiths, & Beesley, Chapter 2 in this volume, for a similar discussion). Because people appear to believe that cause features allow for more inductive inferences (as demonstrated in the preceding study), they may weigh cause features more heavily than effect features.

Another possible reason for weighing causes more strongly than effects is based on psychological essentialism (Medin & Ortony, 1989; see Ahn, 1998; Ahn & Kim, 2000, for discussion of psychological essentialism with respect to the causal status effect). If a mental disorder does indeed have a common-cause structure, then the more fundamental a cause is, the more useful it would be as an indicator of the presence/absence of the disorder’s common cause or causal essence. That is, if the causal relations among symptoms are probabilistic rather than deterministic, and features can be caused by multiple causes, features that are closer to the common cause in a causal network (in the top panel of Figure 30.1, feature B) would be more indicative of the common cause (feature A in the top panel of Figure 30.1) than those that are further away from the common cause (feature C in the top panel of Figure 30.1). Thus, cause features should be weighted more than effect features if a mental disorder has a common-cause structure.

If a mental disorder is believed to be essentialized with a common-cause structure with multiple levels of causal relations, as illustrated in the top panel of Figure 30.1, then the closer a feature is to the disorder’s fundamental common cause, the more useful it tends to be as an indicator of the presence/absence of this common cause or causal essence. For instance, feature B would be more indicative of feature A than feature C would be in the top panel of Figure 30.1. To explain, note that in most causal relations, an event can be caused by more than one possible cause, such that if one believes that A causes B, knowing that B is present does not guarantee that A must have been present because B could have been caused by an event other than A. Furthermore, if one believes that A causes B, which in turn causes C, knowing that C is present is even less likely to guarantee that A was present; C could have been caused by an event other than B, which, in turn, could have been caused by an event other than A—that is, the further away an event is from the root cause, the worse the event is (p. 608) as an indicator of the root cause’s presence. Thus, the deeper cause a feature is in a causal chain, the more informative it can be about the root cause of the chain. For that reason, people may place more weight on cause features than their effect features.

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Effects of Causal Knowledge on Judgments of Abnormality

Causal knowledge also influences the degree to which clinicians perceive people's behaviors to be psychologically abnormal in a global sense (e.g., abnormal; psychologically unhealthy; in need of treatment). In an informal observation of clinicians' reasoning tendencies, Meehl (1973) noted that when clinicians felt that they understood a patient, the patient seemed less abnormal to them; that is, they seemed to apply an "understanding it makes it normal" heuristic in gauging abnormality. Ahn, Novick, and Kim (2003) empirically examined whether causal explanations indeed influence clinicians' overall perceptions of how abnormal a person is. We developed descriptions of hypothetical patients with artificial disorders in which three behavioral symptoms were described as being linked in a causal chain (e.g., "because Penny frequently suffers from insomnia and is in a habitual state of sleep deprivation, she has trouble remembering the names of objects. This memory problem, in turn, leads her to suffer from episodes of extreme anxiety, because she fears that it will cause her to embarrass herself in front of others"; Ahn et al., 2003, p. 747). In one experiment, one group of practicing clinical psychologists and clinical graduate students received these descriptions. A second group also received deeper causal explanations for the root symptom in each of these causal chains. For instance, the phrase "because she is very stressed out due to her workload" was added as a causal explanation for why "Penny frequently suffers from insomnia." As predicted by Meehl (1973), clinicians who received the additional life-event root cause explanations judged that these people were less abnormal (i.e., more psychologically healthy) than those who did not receive such explanations.

A parallel phenomenon in judgments of the need for psychological treatment—a concrete practical measure of judgments of abnormality—has also been documented (Kim & LoSavio, 2009). Vignettes of artificial disorder cases similar to those described in the preceding were judged less in need of psychological treatment when externally controlled precipitating events (e.g., "ever since he was drafted into the army") were described as launching the causal chain of symptoms than when internally controlled events (e.g., "ever since he enlisted in the army") were described, despite the fact that both explanations were rated as equally satisfactory—that is, "understanding makes it normal" when deeper causal explanations for the root symptoms are precipitated by factors outside the person's control. Thus, not only do reasoners seem to adhere to the notion that "understanding it makes it normal," but their judgments also align with the more radical inference that "understanding it solves the problem." Yet in reality, the person would be no less in need of subsequent support or intervention than if a causal explanation had not presented itself to the reasoner.

Kim, Paulus, Gonzalez, and Khalife (2012) reported evidence for a proportionate-response effect, such that judgments of abnormality are predicted by the proportionality (in terms of valence and magnitude) of the behavioral response to the precipitating event. Proportionality between cause and effect has long been observed to serve as a cue to causality (Einhorn & Hogarth, 1986); when an effect is disproportionate to its cause,

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people are less likely to perceive the causal connection. For example, people found it difficult to accept the germ theory of disease when it was first proposed, in part because germs were said to be microscopically small, whereas the effects of these germs could be staggeringly large, wiping out huge swaths of the human population (Medin, 1989).

Thus, Kim, Paulus, Gonzalez, et al. (2012) hypothesized that the proportionality between precipitating events and subsequent behaviors would predict clinicians' judgments of abnormality. They presented practicing clinical psychologists with descriptions of hypothetical people who were either exhibiting behaviors characteristic of post-traumatic stress disorder (PTSD), behaviors characteristic of depression, or mildly distressed (sub-clinical) behaviors. These behaviors were prefaced by descriptions of either severely negative (traumatic) events or mildly negative (everyday) events. Clinicians' judgments of the psychological abnormality of these hypothetical behaviors were strongly influenced by the proportionality between the severity of the event relative to the behaviors. In fact, exhibiting only mildly distressed behaviors after a traumatic event was judged to be just as abnormal as exhibiting full-blown PTSD or depression symptoms after the same event, despite the fact that only the presence of actual disorder symptoms is supposed to be treated as evidence of disorder in the *DSM*. (p. 609) In keeping with Meehl's (1973) assertion that clinicians behave as though "understanding it makes it normal," clinicians' judgments of the ease of understanding the behaviors also correlated with their abnormality judgments (Kim, Paulus, Gonzalez, et al., 2012). That is, the more proportionate the event was to the behaviors, the easier clinicians found it to understand the behaviors, and the less clinicians rated the behaviors to be psychologically abnormal.

Judgments of Psychological Versus Biological Bases of Mental Disorders

Another broad classification of mental illnesses that people may make is whether they are biologically or psychologically based. That is, how do people decide to categorize a given illness as a brain disease or as a disorder of mind? Entertaining this judgment involves some degree of acceptance, even temporarily, of mind–body dualism, given that all disorders of mind must be brain diseases. Yet this judgment will have strong down-stream influences on choices of treatments, ascriptions of blame, and judgments about prognosis, as will be discussed later in this chapter.

For physicalists, biological causes are lower level explanations for psychosocial explanations of behavior. For instance, an overactive amygdala is experienced as anxiety; in reality, either could be invoked as an explanation for poor performance in a swim meet. Instead of treating these two as different levels of explanation, however, laypeople as well as clinicians appear to treat these two causes as being complementary; that is, laypeople tend to treat the relationship between biological and psychosocial explanations similarly to that between internal and external attributions. For instance, upon learning that an exam was easy, one may automatically discount the possibility that a student who got an A on the exam worked hard. Similarly, people's endorsement of biologically construed

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bases of behaviors (e.g., genes, brain structures, neurotransmitters) appears to be inversely related to their endorsement of psychologically construed bases of behaviors (e.g., intentionality, desire, motivations).

Some evidence for this inverse relationship comes from Miresco and Kirmayer's study (2006) with clinicians. They found that clinicians judged psychological factors to be important for explaining dysfunctional behavior exhibited by a person with narcissistic personality disorder. In contrast, biological factors were seen as important for explaining that same dysfunctional behavior when exhibited by a person having a manic episode. That is, narcissistic personality disorder was seen as psychologically based, whereas manic episodes were seen as biologically based, even when explaining the same symptom across the two disorders. Clinicians' ascriptions of blame for the symptoms also varied depending on the underlying mental disorders; the person was blamed more for the same behavior if the person was described to have narcissistic personality disorder rather than a manic episode, presumably because the latter was assumed to be biologically caused (Miresco & Kirmayer, 2006).

Ahn, Proctor, and Flanagan (2009) found a similar relationship. They examined the entire set of mental disorders appearing in the *DSM-IV-TR* (APA, 2000), and measured mental health clinicians' beliefs about their biological, psychological, and environmental bases. Clinicians' attributions of disorders to environmental and to psychological factors were highly positively correlated, but there was a large negative correlation between clinicians' attributions to biological factors and to environmental factors. Clinicians conceptualized mental disorders along a single continuum spanning from highly biological disorders (e.g., autistic disorder) to highly non-biological disorders (e.g., adjustment disorders).

Miresco and Kirmayer (2006) concluded that these kinds of results indicate that "mental health professionals continue to employ a mind-brain dichotomy when reasoning about clinical cases" (p. 913). Yet, rather than believing that mind and body are separate entities, clinicians' conceptualizations may reflect *explanatory dualism* (or functionalism; Putnam, 1975), wherein biological and psychological explanations are viewed as complementary ways of describing human behaviors (Davidson, 1970; Fodor, 1974; Kendler, 2001). Explanatory dualism does not make any ontological assumptions about mind and brain; it simply states that any given mental activities may sometimes be better explained using biological constructs and may at other times be better explained using psychological constructs. Explanatory dualism is therefore not necessarily irrational (Dennett, 1995; Putnam, 1975). For instance, although a person's feelings of depression can be explained in terms of the activity of neurons, neurotransmitters, neuromodulators, and hormones, the same feelings can also be explained in terms of experiencing interpersonal conflict and stress. This latter explanation can (p. 610) be undertaken without necessarily denying depression's biological basis. Thus, the formal disciplines of biology and psychology can coexist without denying each other's validity.

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However, recent studies have demonstrated that the tension between biological and psychological explanations is strong enough to lead to an irrational bias in both clinicians and laypeople (Kim, Ahn, Johnson, & Knobe, 2016; Kim, Johnson, Ahn, & Knobe, 2016). People's judgments appeared to reflect the belief that psychological causes and biological causes have an inverse relationship, such that when psychological causes become more salient or plausible, biological causes are automatically discounted (and vice versa). Thus, psychological and biological causes share a common effect (e.g., surface symptoms), where the two types of causes compete with each other (e.g., Waldmann, 2000) in a manner inconsistent with the notion of explanatory dualism.

Specifically, Kim, Ahn, et al. (2016) and Kim, Johnson, et al. (2016) asked clinicians and laypeople to judge the psychological and biological bases of everyday and disordered behaviors. Behaviors were either described in the context of a person (e.g., Sarah's repetitive behaviors, in which she checks her window locks three times upon leaving the house) or in the abstract (e.g., repetitive behaviors, which are generally characteristic of a particular disorder). As shown in past work, concretely described behaviors (i.e., in the context of a specific person) increased endorsement of psychological bases of those behaviors. More importantly, in line with an inverse dualism account, people correspondingly reduced their endorsement of the biological bases of the same behavior, relative to their judgments when behaviors were described abstractly. Thus, inverse dualism could lead to irrational judgments; mere changes in the framing of behavior descriptions changed judgments of the psychological and biological bases of behaviors.

The preceding results are consistent with well-documented findings in causal reasoning. Causal model theory predicts that there will be competition among causes in common-effect structures, but not among effects in common-cause structures in causal learning (Waldmann, 2000; see Le Pelley et al., Chapter 2 in this volume). In the case of inverse dualism, biological and psychological causes potentially give rise to the same behavior (in a common-effect structure), and people accordingly behave as though accepting one cause automatically denies the other.

Effects of Causal Knowledge on *DSM*-Based Diagnosis

As we have discussed, the modern versions of the *DSM* (i.e., from 1980 to the present) have deliberately excluded information about causal etiology from the diagnostic criteria for nearly all disorders. One of the few exceptions to this general rule was, for many years, the bereavement exclusion in the diagnosis of major depression (*DSM-III*, 1980; *DSM-III-R*, 1988; *DSM-IV*, 1994; *DSM-IV-TR*, 2000). Specifically, even if the requisite behavioral symptoms for major depression were clearly present, a diagnosis of depression was not to be made if the person was in a period of bereavement due to the loss of a loved one. A number of observers argued that the bereavement exclusions should be expanded to include all major loss events (e.g., divorce, job loss, sudden loss of all possessions, as in the case of a natural disaster; Horwitz & Wakefield, 2007; Wakefield, Schmitz, First, & Horwitz, 2007), to avoid falsely pathologizing normal coping responses

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to loss. The *DSM-5* (2013) Task Force, however, opted instead to remove the bereavement exclusion criterion from the list of formal criteria in the *DSM-5* (2013); one justification given for this decision was to avoid missing opportunities to provide mental health care to those in distress.

The case of depression therefore provided a particularly strong test of the "understanding it makes it normal" hypothesis as applied to *DSM*-based diagnosis. Judgments of abnormality do not necessarily translate to diagnostic judgments that would be made while considering whether to follow the recommendations of the *DSM*. Notably, the "understanding it makes it normal" hypothesis, as applied to diagnosis, would be that a bereavement or other loss life event—anything severely negative—would lead to reduced endorsement of a diagnosis of depression. Kim, Paulus, Nguyen, and Gonzalez (2012) presented practicing clinical psychologists with vignette case descriptions of people exhibiting severe depression symptoms. These were prefaced either by a bereavement-related loss event (e.g., the death of the person's spouse), non-bereavement loss event (e.g., the end of the person's marriage), everyday event (e.g., going about everyday life with one's spouse), or no event. Clinical psychologists reduced their endorsement of a diagnosis of depression when the case was prefaced by either type of loss event, compared to being prefaced by the everyday or no event—that is, they did not strictly adhere to the diagnostic criteria listed in any version of the (p. 611) modern *DSM* system in making their judgments. Rather, they appeared to subscribe to the notion that "understanding it makes it normal" and less worthy of depression diagnosis, in concert with Horwitz and Wakefield's (2007) arguments.

Interestingly, while some may argue that *DSM-5* (APA, 2013) unnecessarily pathologized normal reactions to loss by removing the bereavement exclusion criteria for major depression, in other disorders such as PTSD, *DSM-5* (APA, 2013) may conversely have overlooked some abnormal cases by explicitly specifying the causes required for a diagnosis. In order for a diagnosis of PTSD to be made, the patient must have experienced a traumatic event involving exposure to actual or threatened death, injury, or sexual violence, not just anything the person finds traumatizing. According to the "understanding makes it normal" effect, however, a person who has PTSD symptoms after experiencing the type of trauma designated by *DSM-5* (APA, 2013) would be judged less abnormal than someone who has PTSD symptoms without that trauma. Yet, the former would be the one eligible for the diagnosis, and therefore more abnormal in an official sense. Indeed, Weine and Kim (2014) found that practicing clinicians, clinical psychology graduate students, and laypeople alike judged people with PTSD symptoms following a *DSM-5*-qualifying traumatic event to be less psychologically abnormal, yet simultaneously more likely to warrant a PTSD diagnosis than people with the identical symptoms following an upsetting event that does not meet *DSM-5* criteria.

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Prognosis

Psychosocial explanations for mental disorders (e.g., those based on such factors as childhood trauma and current or past stress) are increasingly being replaced by biological explanations (e.g., genes or brain abnormalities; Pescosolido, Martin, Long, Medina, Phelan, & Link, 2010). This shift has important consequences in that the two types of explanations yield differences in inductive inferences. One type of clinical judgment that has been consistently shown to differ, given biological versus psychosocial explanations for psychopathology, is that regarding prognosis. In particular, biological explanations, such as those that attribute mental disorders to genetic or neurobiological abnormalities, have been shown to engender prognostic pessimism—a belief that the disorders are relatively unlikely to remit or be treated successfully (Kvaale, Haslam, & Gottdiener, 2013). A possible mechanism underlying this effect is people's adherence to neuro- and genetic essentialism, in which the brain and DNA, respectively, are seen as the fundamental "essences" underlying mental disorders (Dar-Nimrod & Heine, 2011; Haslam, 2011). Because these essences are perceived as deeply rooted and relatively immutable causes of symptoms, neuro- and genetic essentialism can lead to the belief that mental disorders are difficult to overcome or to treat effectively.

For example, Phelan (2005) showed that attributing an individual's mental disorder to genetic causes increased laypeople's tendency to view the disorder as likely to persist throughout the individual's life. Bennett, Thirlaway, and Murray (2008) similarly found that when participants read a vignette describing a person with schizophrenia, they rated the individual as less likely to recover from the disorder when they were told it was genetically caused. Furthermore, Deacon and Baird (2009) found that when undergraduates were asked to imagine themselves as suffering from depression, they were more pessimistic about "their" prognoses when they were told that depression was caused by a chemical imbalance than when they were told that it was caused by a combination of biological, psychosocial, and environmental causes. Lebowitz, Rosenthal, and Ahn (2016) also found that a case of attention-deficit/hyperactivity disorder (ADHD) was rated as less treatable when symptoms were explained biologically than when they were explained psychosocially.

More recently, such findings documenting an association between biological explanations for psychopathology and prognostic pessimism have been shown to extend to people considering their own real psychiatric symptoms. In the first such study, Lebowitz, Ahn, and Nolen-Hoeksema (2013) found that the more people attributed their own depressive symptoms to genetic and biochemical causes, the longer they expected to remain depressed. Other work has shown that people with and without symptoms of generalized anxiety disorder predict that the disorder will have a longer duration when they are presented with a biological explanation (versus no explanation) of its etiology (Lebowitz, Pyun, & Ahn, 2014). Kemp, Lickel, and Deacon (2014) provided the "results" of a fake biochemical test to individuals who screened positive for a past or current depressive

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episode. They found that participants who were told that they had a “serotonin deficiency” (suggesting their depression had been caused by a “chemical imbalance”) experienced more prognostic pessimism than participants (p. 612) in a control condition who were told their neurotransmitter levels fell within normal limits.

Related to the issue of prognostic pessimism and essentialism, biological explanations for psychopathology can also engender the belief that affected individuals lack agency or control over their psyches, and that this is why they are unlikely to overcome their disorders. For example, in one study, undergraduates given a “chemical imbalance” explanation of depression rated themselves as less able to effectively control depression on their own if they were to suffer from it, compared to participants given a multifactorial explanation (Deacon & Baird, 2009). In another experiment, participants randomly assigned to be told that they carried a gene that increased their risk of alcoholism rated themselves as having less control over their drinking than participants who were told they did not have such a gene (Dar-Nimrod, Zuckerman, & Duberstein, 2013). Kemp, Lickel, and Deacon (2014) found that participants who were led to believe that their depression stemmed from a serotonin imbalance scored lower on a measure of confidence in their ability to regulate their own negative moods than did individuals in a control condition.

Studies documenting how people perceive the downsides of recent advances linking psychopathology to biological factors reveal that some do foresee risks associated with prognostic pessimism and decreased feelings of agency. For example, while people affected by mental disorders appear largely receptive to the use of psychiatric genetics and neuroscience in mental health, a subset of those people also perceive the possibility that associated fatalistic beliefs could lead to discrimination (e.g., in employment or insurance coverage), psychological distress, and even suicide (Illes, Lombera, Rosenberg, & Arnow, 2008; Laegsgaard, Kristensen, & Mors, 2009; Lebowitz, 2014; Meiser et al., 2008).

Notably, the association between biological causal explanations and prognostic pessimism does not appear to be limited to mental disorders. For example, among overweight and obese Americans, attributing one’s own weight status to biological causes appears to be associated with the belief that body weight is unchangeable (Pearl & Lebowitz, 2014). In line with the aforementioned findings regarding reduced feelings of agency among people with essentialist views of their symptoms, Burnette (2010) found that (mostly overweight or obese) dieters who viewed body weight as fixed reported being less persistent after weight-loss setbacks. Genetic explanations for inactivity have also been shown to decrease self-efficacy and intentions to exercise (Beauchamp, Rhodes, Kreutzer, & Rupert, 2011) and to increase consumption of unhealthy food (Dar-Nimrod, Cheung, Ruby, & Heine, 2014). Related research has found that information about the American Medical Association’s recent decision to classify obesity as a disease reduced obese individuals’ concerns about weight and the importance they placed on health-focused dieting, ultimately predicting higher-calorie food choices (Hoyt, Burnette, & Auster-

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Gussman, 2014). In general, it has been predicted that the increasing emphasis on understanding the genetic bases of health and illness could increase fatalism about health outcomes (Dar-Nimrod & Heine, 2011).

Treatment

Among both clinicians and laypeople, different causal attributions for mental disorders also appear to consistently affect judgments about the likely effectiveness of treatment, and what types of treatment may be seen as appropriate. In light of the ongoing shift toward increased emphasis on the biological factors involved in psychopathology (e.g., Insel & Wang, 2010), it is particularly important to understand how biological conceptualizations of mental disorders affect beliefs about treatment.

Data from nationally representative US samples indicate that attributing mental disorders to biological causes is associated with an increased perception that treatment is advisable (Pescosolido et al., 2010; Phelan, Yang, & Cruz-Rojas, 2006). However, there is also evidence that views about different types of treatment for mental disorders can be differentially affected by biological explanations. For example, as Americans' belief that depression stems from biological causes increased significantly from 1996 to 2006, so did their preferences for biologically focused treatment (Blumner & Marcus, 2009). Indeed, among laypeople, biological explanations for mental disorders appear to be associated with increased belief in the effectiveness of medication but decreased belief in the effectiveness of psychotherapy (Deacon & Baird, 2009; Iselin & Addis, 2003; Lebowitz et al., 2012). This effect has also been documented among individuals affected by depression (Kemp et al., 2014).

These findings suggest that mind–body dualism is prevalent in people's thinking about mental disorders—that is, people may conceive of the mind and the body/brain as separate entities, with the former construed as having psychological properties while the latter is construed as having biological (p. 613) properties. Thus, "non-biological" treatments that are seen as acting through psychological or psychosocial mechanisms (e.g., psychotherapy) would be viewed as less likely to be effective when psychopathology is attributed to biological causes (and thus perceived as residing in the brain, genes, or body), while biomedical treatments (such as pharmacotherapy) are seen as more likely to be effective in such cases.

Notably, despite their psychopathology-related expertise, mental-health clinicians do not appear to be immune to such dualist beliefs. Ahn et al. (2009) asked practicing mental health clinicians to consider a variety of mental disorders, list their cause(s), and rate each cause with respect to the degree to which it was psychological, environmental, and biological in nature. At least 30 days later, a subset of these clinicians was also asked to judge the degree to which they felt that psychotherapy versus medication would be effective in treating each mental disorder. In general, they found that clinicians rated

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medication as more effective for disorders that had been perceived as more biologically and less psychologically environmentally based, while they rated psychotherapy as more effective for disorders that were seen as more psychologically/environmentally and less biologically based.

Causal beliefs have been shown to influence clinicians' treatment decisions, even at the level of the individual patient. A study by de Kwaadsteniet, Hagmayer, Krol, and Witteman (2010) presented practicing clinicians with two realistic, complex case descriptions of mental health patients, along with information about each patient's environment, recent events, and results of psychometric tests. Clinicians were asked to create a "causal map" for each patient by diagramming the respective roles of the various psychosocial factors in causing each patient's symptoms. Results indicated that although the resulting individual causal maps differed between clinicians, each map was predictive of which interventions the clinician rated as most effective for each case. This finding underscores the notion that clinicians' causal beliefs play an important role in their clinical reasoning.

Issues for Future Research

This section considers three issues for future research: (1) Are the effects of causal background knowledge on reasoning about mental disorders rational? (2) What are mechanisms underlying competition between psychological and biological explanations for mental disorders? (3) Are the effects of causal background knowledge on reasoning about mental disorders universal or culture-specific?

Rationality

While the official nosology of mental disorders (namely, *DSM-5*, 2013) avoids causal interpretations and differential weightings of symptoms in most cases, both clinicians' and laypeople's causal knowledge affect how symptoms of mental disorders are weighed, as shown by the findings reviewed in this chapter. We also showed that clinicians' diagnoses of mental disorders and judgments of normality are also affected by their causal knowledge. Is being affected by causal knowledge when reasoning about mental disorders rational? Next, we discuss this issue in terms of the effects of causal knowledge on diagnosis and treatment judgments, followed by the effects on normality judgments.

Needless to say, if the available causal background knowledge is valid, it would be rational to use it for diagnoses or treatment decisions. However, because the etiologies of mental disorders are still controversial, it may seem problematic that clinicians and laypeople use their idiosyncratic causal beliefs in clinical reasoning. Thus, one might argue that both clinicians and laypeople should be encouraged or reminded to utilize official diagnostic norms (i.e., *DSM*) without relying on their own causal theories, in order to avoid idiosyncratic weighting of features or subjective judgments on treatment efficacy.

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After all, one obvious and well-documented benefit of using the *DSM* in clinical practice is improved reliability in diagnoses, potentially providing a rational basis for discouraging the use of personal causal theories.

Yet, there are reasons to take causal knowledge seriously. Even though there is little agreement about the root causes of mental disorders, certain kinds of causal relations among symptoms are well accepted, and the use of such causal knowledge can be beneficial. For instance, if the causal status effect stems from a rational reasoning bias (e.g., giving more weight to features that are more indicative of an essence or a core feature), perhaps making diagnoses based on causal reasoning or suggesting treatment options targeted to controlling deeper causes would be rational, and doing so might facilitate more accurate diagnoses or the discovery of better treatment plans.

Furthermore, enforcing nosology based only or mostly on surface symptoms can hinder significant progress in psychopathology research (p. 614) by preventing discoveries of better classification schemes based on hidden, underlying causes. For instance, major depressive disorder, which is considered a single disorder in the *DSM-5*, may consist of several subtypes, each involving different causal mechanisms and requiring different kinds of treatments. Furthermore, some of these subtypes may be closely tied to generalized anxiety disorder. For these reasons, the field has been moving away from symptom-based descriptions of mental disorders, and many researchers have begun focusing on understanding biological mechanisms underlying mental disorders. In that sense, returning to the prototype approach to mental disorder classifications may not be rational.

The issue of rationality is also controversial when considering judgments of normality based on causal history. Meehl (1973) presented the “understanding makes it normal” phenomenon as a reasoning fallacy: just because one now sees how the symptoms had developed, it should not make the symptoms less abnormal. This is one of the reasons that the *DSM-5* (2013) removed the bereavement exclusion criteria; severe depression should receive clinicians’ attention and treatment, regardless of causes. On the other hand, it does appear rational for judgments of normality and diagnoses of mental disorders to be affected by information on precipitating conditions, as they can be informative for prognosis and treatments. For example, clinicians clearly need to be aware of a patient’s trauma history in order to determine whether or not a diagnosis of PTSD is appropriate and to plan a course of treatment in such a case.

Mechanisms Underlying the Effects of Biological Explanations

Another important research area, which recently has been receiving a great deal of attention, is uncovering the effects of biological explanations on reasoning about mental disorders. In general, biological and psychosocial explanations appear to have an inverse relationship such that if one type of explanation is dominant, the other is discounted. The two types of explanations also bring about different kinds of inductive inferences.

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Biological explanations tend to result in pessimistic prognostic assumptions about mental disorders compared to psychosocial explanations, although biological explanations also cast symptomatic individuals as less responsible for their symptoms than psychosocial explanations (Kvaale, Haslam, & Gottdiener, 2013).

From cognitive psychologists' perspectives, one intriguing research agenda is to identify the mechanism underlying this inverse relationship. As we have discussed, one possibility is dualism. It is possible that people can see a person either as a psychological agent or a biological mechanism, and it is difficult to conceive of a person as both because people have difficulty envisioning how mental activities cause or are caused by material processes. Thus, they might believe that biological dysfunctions should be treated with medications, whereas psychological impairments should be treated with psychotherapy. Another related, though not necessarily alternative, possibility is the perception of free will. Biological explanations may decrease the extent to which free will is seen as relevant. In the absence of free will, prognostic pessimism may increase because there is not much one can do about the symptoms, and treatment based on psychotherapy that would require agency on the part of the patient may appear not as promising. Future research should attempt to identify the degree to which each of these two specific mechanisms can, separately or conjointly, account for the negative consequences of biological explanations.

Understanding the mechanism underlying the effects of biological explanations on mental disorder concepts would also be crucial in devising any intervention programs. It is generally accepted that the best line of treatment for mental disorders is often a combination of medication and psychotherapy. To make such a combined approach more acceptable for clinicians as well as patients, an educational intervention explaining how psychotherapy can cause changes in patients' brains may be effective if reluctance to receive psychotherapy for biologically construed mental disorders stems from dualism. Alternatively, perhaps one reason that laypeople are reluctant to take medications for mental disorders is the fear that doing so will decrease their free will. Thus, future research in these areas could have beneficial impacts on public health.

Universality of the Phenomena

Another important future research question is whether the processing biases discussed in this chapter are universal phenomena or are restricted to Western cultures, in which all of the previously reviewed studies were conducted. Different cultures do have somewhat different causal beliefs about mental disorders. For instance, Hagmayer and Engelmann (2014) provide a systematic review of causal beliefs about depression among both (p. 615) non-Western and Western cultural groups, documenting some cultural differences (e.g., magic, evil spirits, and the devil being listed as the top causes of depression among African Yoruba; Lavender et al., 2006). The question relevant to the current chapter would be whether such differences in causal beliefs also lead to differences in the way this causal knowledge affects reasoning about mental disorders. For instance, causal

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theories generated by clinicians in Kim and Ahn (2002b) are only moderately consistent across the clinicians, but nonetheless, clinicians consistently demonstrated the causal status effect based on their own causal theories. Likewise, even though different cultural groups exhibit different causal beliefs, they may still exhibit the same causal status effect, as well as similar effects of causal explanation on judgments of normality and treatment efficacy, and so on. For instance, if the Yoruba group believes that counteracting evil magic should be the best treatment for depression, then they would be exhibiting the same kind of processing biases as the Western group, even though they have different causal beliefs. Such a possibility remains to be more fully documented. For instance, Lavender et al. (2006) merely note, “In the Yoruba and Bangladeshi groups people believed that the root causes of depression should be addressed,” without any further details, such as quantitative estimates of how strong this association between root cause and treatment efficacy judgments is.

In general, few studies have directly examined the universality of processing biases in usage of causal beliefs about mental disorders. Hagmayer and Engelmann (2014), for instance, note that none of the studies they reviewed about depression examined the use of causal beliefs for diagnoses. Furthermore, tracking whether choices of treatment correlate with beliefs about causes of mental disorders is further muddled by practical issues such as availability of treatments in a given culture. For instance, even though a patient believes in psychosocial causes for depression, she may indicate a preference for biomedical treatments because counseling or psychotherapy are not available in her area (Hagmayer & Engelmann, 2014).

We speculate that because the processing biases discussed in the current chapter are rooted in deeper beliefs and biases, such as essentialism (e.g., Sousa, Atran, & Medin, 2002) and dualism (e.g., Bering, 2006), which have been demonstrated to appear in various cultures, these processing biases would be present across a wide variety of cultures. Thus, future investigations into the universality of these phenomena can also further shed light onto the originality of these phenomena.

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Notes:

(1.) This chapter does not cover the effect of causal explanations on social stigma associated with mental disorders (e.g., Haslam, 2011; Kvalle, Gottdiener, & Haslam, 2013; Lincoln, Arens, Berger, & Rief, 2008; Phelan, 2005).

(2.) Interestingly, the amenorrhea criterion (absence of the period) for anorexia nervosa was removed from *DSM-5* (APA, 2013) because there were no clinically relevant differences between women who did and did not meet that criterion, as long as they met all the other criteria. This recent change seems to lend some credence to the conceptualizations of the clinician participants from Kim and Ahn (2002a), who also judged that amenorrhea is not an important part of anorexia nervosa.

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