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Understanding Our Own Biology: The Relevance of Auto-Biological Attributions for Mental Health

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As knowledge of the neurobiological basis of psychopathology has advanced, public perceptions have shifted toward conceptualizing mental disorders as disorders of biology. However, little is known about how patients respond to biological information about their own disorders. We refer to such information as auto-biological—describing our own biological systems as a component of our identity. Drawing on research from attribution theory, we explore the potential for auto-biological information to shape how patients view themselves in relation to their disorders. We propose an attributional framework for presenting auto-biological information in a way that encourages agency, rather than destiny. We argue that this framework has the potential to change expectations and improve outcomes in the treatment of psychiatric disorders.

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In the 25 years since the United States Congress declared the 1990s the "Decade of the Brain," mental health researchers have increasingly accepted the notion that psychiatric disorders are fundamentally diseases of the brain. Funding sources such as the National Institute of Mental Health have incentivized the search for brain-based indicators and predictors of psychiatric disorders as a way to revolutionize how mental disorders are conceptualized, diagnosed, and treated (Weinberger & Goldberg, 2014). This shift in research priorities has been reflected in public opinion, with a majority of U.S. adults now believing that psychiatric disorders like depression (67%) and schizophrenia (86%) are caused by biological factors (Pescosolido et al., 2010). Moreover, consumer-focused messages, such as "depression is a chemical imbalance," originating from pharmaceutical companies are now commonplace and have shaped public attitudes toward mental illness as well as the self-perceptions of individuals affected by it.

Although originally expected to reduce social stigma and blame, many biological messages about psychiatric disorders have had the opposite effect. For example, when individuals endorse a brain cause for a psychiatric disorder like schizophrenia, they rate an affected individual as more likely to be dangerous and less likely to achieve symptom remission (Kvaale, Gottdiener, & Haslam, 2013). Similar biases have been observed when a genetic cause for a mental disorder has been offered (Angermeyer, Holzinger, Carta, & Schomerus, 2011; Haslam & Kvaale, 2015; Kvaale et al., 2013). Of course, inadvertently worsening the stigma associated with these disorders is directly counter to the intentions of the funding agencies and advocacy groups that have promoted biological messages. Those agencies and groups operated under the assumption that providing information about the biological causes of mental illness would reduce the blame placed on those affected (Jones & Mendell, 1999). In one sense, such messages have had the intended effect: It does appear that public

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perceptions of blame for mental illness are reduced by biological messages (Crisafulli, Von Holle, & Bulik, 2008; Lincoln, Arens, Berger, & Rief, 2008). However, other aspects of mental illness stigma—such as perceptions of dangerousness, desire for social distance, and pessimism about change—are increased when members of the public endorse biological causes for mental illness (Angermeyer et al., 2011; Haslam & Kvaale, 2015; Phelan, Yang, & Cruz-Rojas, 2006).

The majority of the research on the impact of biological messages on public perceptions of mental disorders has focused on lay beliefs about mental illness—or "folk psychiatry" (Haslam, 2005). Studies have been conducted using national survey samples, undergraduate students, or online convenience samples, and have assessed attitudes toward a fictional character with a particular disorder. The attitudes of those who are actually suffering from mental illness have received far less research attention (see Lebowitz, 2014, for a review). This is a notable omission considering the proportion of the population affected, with one source estimating that 46% of U.S. adults will experience at least one psychiatric disorder during their lifetime (Kessler, Berglund, et al., 2005). Consequently, when we measure "public beliefs" about mental illness, what we get is (almost) equally likely to reflect beliefs about ourselves as beliefs about others.

The term *auto-biological*, as we use it here, refers to one's knowledge, attitudes, or beliefs about one's own biological systems, whether measured at the level of the gene, the neuron, the brain, or the entire organism. We hold that such information has the potential to shape our personal identity by increasing our understanding of our own nature and history. After all, the scientific reality that we are biological beings is separate from the degree to which we think of ourselves as such. Individual differences in how people think about themselves in relation to their cells, genes, or organs—their auto-biology—may powerfully affect other aspects of self-directed thought and behavior, potentially even cycling back to influence the biology itself.

In this review, we consider the assertion that auto-biological beliefs may be particularly salient for individuals facing a disease state that is characterized by disordered subjective experience rather than simply disordered biology. How is one to understand, for example, that something as ethereal as mood can be tied to something as mechanistic as the firing of neurons? A conventional medical diagnosis like diabetes is linked to auto-biological beliefs, but in a less ambiguous way. We have a clearly biological definition of the disorder (dysfunctional insulin metabolism), and we can be taught how our own behavior and treatment decisions interact with this biological signature. An individual diagnosed with depression, on the other hand, is faced with a much more complex problem of attempting to understand how dysfunctional biology contributes to the subjective experience of emotional pain (Kendler, 2005). This is a difficult problem for the scientist, clinician, and patient alike, and it deserves more attention from the perspective of the individual who must make treatment decisions based on his or her understanding of the disorder. From this auto-biological perspective, we examine the impact of the recent shift toward biological conceptualizations of mental disorders on those who are affected by it most: individuals with a psychiatric diagnosis.

BIOLOGICAL BELIEFS AND SCIENTIFIC REALITIES

We begin our discussion with a brief review of the experimental consequences of introducing biological information about mental disorders. A critical look at the existing studies reveals that many of the biological messages that have been investigated are at best reductionistic and at worst frankly inaccurate. When examined alongside the scientific knowledge they attempt to convey, the biological messages provided in many of these studies appear overly simplistic (and inadvertently misleading).

Depression Is a Chemical Imbalance

The chemical imbalance explanation for depression resulted from the discovery that drugs affecting levels of monoamines such as serotonin and norepinephrine were effective in treating depression (Lacasse & Leo, 2005). Direct-to-consumer advertising by companies marketing antidepressants that can "correct" the alleged chemical imbalance has helped spread the message, along with articles in the popular press that have adopted the chemical imbalance terminology (Leo & Lacasse, 2008). The result is that the chemical imbalance explanation for depression is widely endorsed and

yet scientifically distorted. For example, in a survey of undergraduate students, 24% erroneously believed that doctors can directly measure amounts of different brain chemicals to diagnose depression, and 35% believed that doctors treat depression by adding or subtracting brain chemicals to find the right balance (France, Lysaker, & Robinson, 2007).

The impact of the chemical imbalance message has been investigated experimentally. Undergraduates who reported previous or current depressive episodes were given a fictional "Rapid Depression Test," which purported to measure neurotransmitter levels via cheek swab (Kemp, Lickel, & Deacon, 2014). Half of the participants were given test results that indicated their depression was caused by a serotonin imbalance, and they were shown a bar graph depicting lower serotonin levels compared to other neurotransmitters. Control participants were given test results depicting all neurotransmitters in the "normative range." Participants in the chemical imbalance group reported increased prognostic pessimism and lower perceived ability to regulate negative mood states compared to the control group. They also rated pharmacotherapy as more credible than psychotherapy, confirming the finding that medication is considered the best option for treating a "chemical" disorder (Deacon & Baird, 2009).

This specific investigation is a clear example of the harm that can be caused by presenting incorrect, reductionistic biological messages about mental disorders. Despite the ubiquity of the "chemical imbalance" explanation in popular culture, we know from decades of scientific research that depression is not simply a chemical imbalance. Research on the mechanisms of action of antidepressant medications has revealed that these drugs affect brain plasticity and neural network dynamics and that medication efficacy (or lack thereof) cannot be explained by simple local changes in brain chemistry (Krishnan & Nestler, 2010). Recent work suggests that even the initially proposed mechanism of action of antidepressant medications was substantially incorrect: These drugs do not work via acute increases in monoamine transmission but rather operate over a longer timescale, affecting gene transcription factors in specific regions of the brain but not others (Pittenger & Duman, 2008). And they do not work for everyone: In most clinical trials, around 50% of patients achieve total symptom remission (and note that efficacy rates for most psychotherapies are comparable; Amick et al., 2015; DeRubeis et al., 2005; Elkin et al., 1989; Hollon et al., 1992).

The current state of knowledge about the etiology of depression suggests a model of causality that is far more complex, involving a dynamic interaction between biological and psychological processes. The behavioral link between stressful life events and depression (Hammen, Marks, Mayol, & DeMayo, 1985) can now be explored mechanistically, and neuroscience research has revealed the similarities between the stressed brain and the depressed brain. Inflammatory pathways that are triggered by chronic stress are also overactive in depression (Miller, Maletic, & Raison, 2009), and chronic stress can lead to reduced neurogenesis and cellular atrophy in the hippocampus—changes that have been associated with depression in human postmortem as well as animal studies (Saveanu & Nemeroff, 2012). Antidepressant drugs may reverse some of these stress-induced changes (Pittenger & Duman, 2008); for example, the downstream targets of antidepressant drugs can increase the activity of transcription factors to induce neuroplastic changes at the level of the synapse. Thus, it appears that stress may lead to depression through mechanisms of altered molecular neuroplasticity, and antidepressant medications may work to reverse those changes. From the perspective of auto-biology, we imagine this conclusion may in fact hold more comfort for individuals than the previously held notion that such drugs temporarily adjust relative levels of neurotransmitters.

Mental Illness Is Genetic

The Human Genome Project completed the first full sequence of the human genome in the early 2000s, coinciding with substantial growth of the field of behavioral genetics and an expanded search for the genes that underlie medical illnesses as well as psychiatric disorders. Unfortunately, one-to-one mapping of genes and mental disorders has proved elusive and may be overly simplistic (Kendler, 2006). While the complexity of genetic contributions to psychopathology has been accepted by the scientific community, it has not been well communicated to the general public, with the result that the public conception of psychiatric

genetics is often frankly inaccurate. For example, 24% of individuals surveyed in one study believed that the brain was the main location of genes in the body (Lanie et al., 2004).

Genetic information is particularly challenging to communicate, as it is both scientifically complex and ethically sensitive. Genetic explanations of behavior have been said to appeal to the natural tendency to "essentialize," or to categorize people and other living things based on what are perceived to be fundamental, underlying natural characteristics (Haslam & Ernst, 2002; Rothbart & Taylor, 1992). When a genetic explanation for a particular behavior or outcome is evoked, it sets in motion a pattern of thinking called *genetic essentialism* (Dar-Nimrod & Heine, 2011) through which a number of characteristics are automatically assigned: (a) *immutability*, that genetically caused outcomes are inescapable and predetermined; (b) *etiological specificity*, that genetic causes supersede all other causes for behavior (e.g., environmental or psychological causal factors); and (c) *discreteness*, that individuals in a genetic group are homogeneous and uniquely defined by a shared characteristic.

The rigid categorization that results from genetic essentialism serves to exacerbate stigma against diagnosed or marginalized groups (Dar-Nimrod & Heine, 2011) and has been implicated in the frequently observed association between genetic explanations of mental illness and stigma (Haslam, 2011). For example, undergraduate participants tend to rate vignette characters as more dangerous and less likely to recover when given a biogenetic explanation of their mental disorders, compared to environmental or psychosocial explanations (Bennett, Thirlaway, & Murray, 2008; Boysen & Gabreski, 2012; Lincoln et al., 2008; Walker & Read, 2002).

Inarguably, there is a genetic component to disorders like schizophrenia and depression. However, the statement "mental illness is genetic" obscures the critical observation that research in psychiatric genetics has revealed no simple gene–disorder associations for major mental illnesses. Instead, a complex interplay of developmental, environmental, genetic, and epigenetic factors confers relative risk or resilience for disease. In one well-publicized example of a gene–environment interaction, the "short" allele for the promoter region of the serotonin transporter gene was shown to confer significantly greater vulnerability for depression only in combination with exposure to stressful life events (Caspi et al., 2003).

A statistical gene–environment interaction says nothing about the mechanism by which psychological processes or environmental factors affect the biological systems of some individuals but not others. One possible pathway by which such an interaction could occur is through *epigenetics*, which refers to modifications that can alter gene function without changing the underlying DNA sequence (Dudley, Li, Kobor, Kippin, & Bredy, 2011; Tsankova, Renthal, Kumar, & Nestler, 2007). Modification of histones—the structural proteins that support strands of DNA—can affect whether or not a gene is ultimately transcribed into its end product (Sun, Kennedy, & Nestler, 2013). Similarly, the addition of a methyl group to a cytosine residue in the DNA sequence can either enhance or silence gene expression (Tsankova et al., 2007). Interestingly, these epigenetic "tags" (e.g., histone modification, methylation) will differ for DNA in different cells in different regions and can be induced or reversed in response to environmental events. For example, rat pups that receive frequent licking and grooming from their mothers in the first week of life show changes in the DNA methylation of genes that regulate the stress response, making them less reactive to stress in adulthood (Weaver et al., 2004). Thus, through epigenetics, a stable, fixed DNA sequence can become a flexible, adaptive system that is responsive to the environment (Sweatt, 2009).

How exactly all these pieces fit together—stress-induced changes in neuroplasticity, individual differences in stress sensitivity, and experience-dependent epigenetic modification—is not yet clear. Nonetheless, it is evident from this cursory review that no single conclusion can be drawn about *what is* the biological nature of a disorder like depression. Clearly, biological processes are involved in the onset and maintenance of the disorder, but no single biological explanation can entirely account for what it means to be clinically depressed. Instead, from the convergence of genetics, neuroscience, and psychology emerges a more complete picture of the biology of depression, one that stands in stark contrast to the most widespread beliefs

about the nature of the disorder. This discrepancy is unfortunate because a more dynamic view of the biology of depression is arguably more optimistic—we know that depression is responsive to various treatments, subject to environmental influences, and not genetically inevitable.

At least one research group recently attempted to measure the influence of a more nuanced biological message on attitudes about depression. Lebowitz and colleagues (2013) recruited a sample of individuals via Amazon's Mechanical Turk who scored in the clinically significant symptom range on the Beck Depression Inventory (BDI $\geq$ 16). Half of the participants watched a short video explaining environmental effects on gene expression (i.e., epigenetics) and emphasized that brain chemistry is "malleable," whereas the other half learned that depression is a "biological illness" that is genetically inherited and associated with structural brain differences. Encouragingly, participants in the malleable condition reported a greater sense of agency with regard to dealing with their symptoms than those in the biological illness condition. In a subsequent replication sample, similar effects were shown both immediately and at six-week follow-up (Lebowitz & Ahn, 2015). In contrast to the studies previously reviewed (which compared the effects of biological versus nonbiological messages), this study was the first to manipulate the content of the biological message, in an attempt to move from simplistic biological descriptors (i.e., "genetic disease" or "chemical imbalance") to a more nuanced explanation of the dynamic interplay between environmental influences and psychopathology. It may provide a useful starting point for interventions that aim to introduce more realistic, complex, and arguably more adaptive messages about the biology of mental illness.

ATTRIBUTION AS A CANDIDATE PSYCHOLOGICAL MECHANISM

Having reviewed the existing literature on biological beliefs about mental illness, we now move to a discussion of the psychological processes that could mediate the impact of beliefs about the biology of mental illness on individuals with a disorder. As stated, research on the consequences of such beliefs for affected individuals is limited, and results from existing studies are mostly discouraging (Lebowitz, 2014). Furthermore, the existing research has been largely atheoretical, leaving the reader with the conclusion that biological messages are often harmful, but with little sense of why this might occur or what could be done to modify the effects. At this point, the question of whether or not biological information should be communicated to patients is immaterial. Biological conceptualizations are already part of our national dialogue about mental health and have influenced beliefs in a majority of the U.S. population (Pescosolido et al., 2010; and note that these figures do not reflect any further changes occurring in the past six years). Thus, given that dissemination has already occurred, there is an urgent need to clarify what meaning an individual might subscribe to auto-biological information—especially within the context of a psychiatric diagnosis—and what psychological mechanisms might be harnessed to improve the way biological information is delivered to patients to mitigate any potential negative impacts.

The focus of this review will now shift toward considering depression in particular, although we are confident that much of the following discussion could be applied trans-diagnostically. Depression is the most common mood disorder, affecting 16.6% of U.S. adults at some point in their lifetime, and is the leading cause of disability for individuals aged 16–45 (Kessler, Chiu, et al., 2005; World Health Organization, 2008). Beyond its prevalence in the population, depression is a suitable target disorder for the current discussion for two reasons. First, in contrast to disorders that are characterized by psychotic symptoms (e.g., schizophrenia), public opinion of the etiology of depression is less exclusively biological and tends instead to incorporate a number of different causal factors including stress, relational difficulties, and personality variables (Schomerus et al., 2012). Second, patient beliefs and expectations play a powerful role in the treatment of depression, with placebo effects in therapy and drug trials accounting for as much as 30% of treatment response (Rutherford, Wager, & Roose, 2010). Given that expectations for improvement appear to be an active ingredient in the treatment of depression, meta-cognitive beliefs about the disorder and its biology may be particularly influential.

Indeed, beliefs about the etiology of depression may affect an individual's tendency to both seek and engage

in treatment. Endorsing a biological cause for depression has been associated with greater intent to accept a diagnosis among individuals who were experiencing depressive symptoms (Van Voorhees et al., 2005). Patients with a diagnosis are more likely to choose a treatment that matches their etiological beliefs (Schweizer et al., 2010), and assigned treatments that are congruent with a patient's etiology beliefs are perceived as more likely to be helpful (Iselin & Addis, 2003). And while two studies have shown that preexisting etiological beliefs do not influence treatment outcomes (Dunlop et al., 2012; Leykin, DeRubeis, Shelton, & Amsterdam, 2007), one study suggested that patients who endorsed biological beliefs at baseline were more likely to respond to antidepressant medication than those who did not (Sullivan et al., 2003). Etiology beliefs also appear to be shaped by treatment experiences: After successfully completing a treatment, patients are more likely to endorse beliefs that are in-line with the treatment modality they received (Leykin et al., 2007). Thus, while it appears that beliefs about depression etiology may be influential, we still have very little understanding of the mechanism by which such beliefs might affect an individual's engagement or success in treatment.

Our search for candidate psychological mechanisms drew us to the classic literature on attribution. Attribution is the process by which we infer cause-and-effect relationships in an attempt to explain behavior. Whether applied to our own behavior or the behavior of others, attributions are considered an aspect of "naïve psychology" (Heider, 1958) because they exist naturally in the minds of laypeople and are distinct from philosophical or academic explanations of behavior. The attribution literature provides an appropriate theoretical background for the present discussion because it directly addresses the question: What is the impact of beliefs about causality on behavior? In pursuit of this question, we drew relevant examples from the attribution literature of attempts to alter individuals' attributions about their own behavior along three primary dimensions: internal/external, stable/unstable, and controllable/uncontrollable. While examples in this section are drawn from disparate psychological literatures, we illustrate how the same principles can be meaningfully applied to understanding how individuals account for the causes of their own mental disorders.

Internal Versus External

Early attribution research focused on the basic distinction between attributing one's own behavior to internal or external causes (Heider, 1958). Experimental paradigms using pill placebos provided an opportunity to study the effects of attributing one's behavior (e.g., arousal level) to an internal cause (anxiety) or an external cause (a pill). Several studies found benefits of attributing arousal to external sources: For example, Nisbett and Schachter (1966) found that participants who attributed a physiological fear reaction to at placebo, rather than the upcoming threat of shock, were able to tolerate higher levels of shock and reported less pain than participants who attributed their reaction to an internal fear state. In a study with chronic insomniacs, Storms and Nisbett (1970) found that participants who expected to be aroused by a placebo taken a bedtime had an easier time falling asleep compared to participants who expected to be relaxed by the pill (and thus attributed their continued wakefulness to their own internal anxiety).

These early misattribution studies concluded that attributing a physiological state (arousal) to an external source (drug) frees the individual from making internal attributions that could be distressing, such as "I am afraid of this shock" or "My thoughts make it impossible to fall asleep." However, if generalization of a behavior to a broader context is desired, then it is better to attribute that behavior to an internal as opposed to an external factor. For example, Davison and Valins (1969) led participants to believe that they had taken a "vitamin" that conferred skin insensitivity prior to receiving electric shocks; half of the participants were then debriefed and told the truth about the vitamin, leading them to think that any increase in their shock tolerance was attributable to internal factors. These internal attributions allowed the debriefed group to tolerate higher levels of shock in a delayed posttest compared to the group who attributed their earlier tolerance to the vitamin. This study makes the simple yet crucial point that attributing a positive outcome to an external source creates a reliance on that source (in the same way that a patient on medication may become reliant on that medication for continued symptom management). Internal attributions, in contrast, can persist beyond any single environmental context and have more potential for generalizability.

Extending the clinical implications of these early attribution studies to the diagnosis and treatment of depression, the extent to which an individual attributes his or her depression to internal versus external factors may matter a great deal. For example, attributing depression to a genetic risk could be viewed as external ("it was handed down to me by my parents"), whereas attributing it to a personality factor could be viewed as internal ("there is something wrong with me"). Patients may take comfort in an external attribution, in this case, as it could remove a sense of responsibility or self-blame for the depression etiology. In turn, the internal versus external distinction could be relevant to a patient's expectations for improvement both during and after a course of treatment. Such expectations would likely differ depending on whether symptom improvement is believed to be caused by factors within an individual—particularly changes in depressogenic processes or characteristics—or by something external, such as medication.

Stable Versus Unstable

A second attributional dimension, stable/unstable, has been investigated most thoroughly in the educational and social psychology literatures. In the domain of academic achievement, a student's belief in the stability of his or her performance across time has clear implications for sustaining motivation following success or failure. For example, Dweck (1975) concluded that exposing students to failure and attributing it to lack of effort—an unstable factor that can change across time —improved their performance compared to a group of students who were exposed to only success feedback. Blackwell, Trzesniewski, and Dweck (2007) later investigated beliefs about the stability of intelligence as an individual difference: *Entity* theorists believe that intelligence is an unchangeable, fixed quantity that individuals possess to various degrees, whereas *incremental* theorists believe that intelligence is malleable and capable of being developed over time. In a large-scale, school-based intervention, the authors gave one group of students an incremental message ("that learning changes the brain by forming new connections and that students are in charge of this process"; Blackwell et al., 2007, p. 254) and followed them, along with a control group, through the acadmically crucial transition years of junior high school. The incremental group reported increased motivation in math class and showed a reversal of the normative decline in math grades that tends to occur during that developmental period. The same effect appears to hold for students in higher education: Wilson and Linville found that college freshmen who were told that grades tend to improve over the four years in college improved their academic performance and had lower dropout rates compared to a group that was not given any information about GPA trends (Wilson & Linville, 1982, 1985).

The positive academic outcomes associated with these brief attributional interventions speak to the power of the stable/unstable dimension for affecting beliefs as well as future behavior. The two types of implicit theories discussed in Dweck's work—incremental versus entity—can be readily applied to biological beliefs about depression. An entity perspective might emphasize the immutability of genetic risk, the structural changes that are thought to occur in the depressed brain, and the "chemical imbalance" that underlies depressed mood. An incremental perspective, in contrast, might emphasize gene–environment interactions, functional brain changes that occur with depression, and the potential for psychological as well as pharmacological treatments to reverse those changes. These are initial hypotheses at the moment, as to our knowledge implicit beliefs about the biology of depression have yet to be measured systematically. Nevertheless, the recent research investigating the effects of a "malleable" message about the biology of depression (Lebowitz & Ahn, 2015; Lebowitz et al., 2013) suggests that this perspective has promise, and provides important evidence that shifting from an entity to an incremental perspective on the biology of depression could have an impact on a patient's expectations for improvement and intention to seek treatment.

Controllable Versus Uncontrollable

The final attributional dimension—controllable/uncontrollable—has been explored in the clinical as well as the social psychological literatures. Evidence from both fields suggests that attributing our behavior to factors within versus outside our control has dramatically different implications for future behavior. The learned helplessness model of depression (Abramson, Seligman,

& Teasdale, 1978), based on the original learned helplessness work with animals, asserted that "helplessness"—the belief that reinforcement is independent of behavior—is a core factor in the etiology and maintenance of depression. In an early experimental investigation, Klein and Seligman (1976) induced learned helplessness in a group of healthy college students by subjecting them to inescapable aversive noise. Half of these participants (along with a group of depressed participants who were presumed to have already learned helplessness) then underwent a "therapy" experience—completing a set of solvable discrimination problems—that was expected to correct the belief that responses are not tied to outcomes. They found that completing solvable therapy problems reduced the escape deficits associated with helplessness in depressed and nondepressed subjects alike. Thus, learned helplessness was reversed through a therapeutic experience in which responses were reliably tied to reinforcement, thus conferring controllability in a situation previously deemed uncontrollable. Subsequent work has confirmed that learned helplessness (or hopelessness) appears to play a causal role in the etiology of depression and suicidality (Nock & Kazdin, 2002; Waszczuk, Coulson, Gregory, & Eley, 2016), but is amenable to intervention (Handley et al., 2013).

In contrast to the other two attributional dimensions, for which information-only interventions are effective in shifting people's beliefs (e.g., from stable to unstable), the controllable/uncontrollable dimension may require direct experience of controllability to change beliefs and behavior. Evidence in support of this assertion can be found in the literature on self-efficacy. In outlining his theory, Bandura (1977) made a distinction between two types of expectancies: An outcome expectancy describes the belief that an outcome is controllable, whereas an efficacy expectancy is the belief that one is capable of controlling the outcome. Bandura's theory, in effect, suggests that two conditions are required for the controllability dimension to influence behavior: (a) The process must be controllable, and (b) the person must believe in his or her ability to control it. If condition B is not met, condition A is no longer useful and could even result in additional shame about one's personal inability to affect the outcome (see Abramson et al., 1978, for a related discussion of personal helplessness).

According to Bandura (1977), the most persuasive source of efficacy information is performance accomplishments—multiple success experiences accumulated over time. This point will likely resonate with behavioral therapists, who tend to build in success experiences early in treatment to boost patient confidence and encourage engagement (Maddux, 2009). Verbally persuading someone of their own efficacy, in contrast, is easier to implement but much less effective (Bandura, 1977). Building self-efficacy one way or another is particularly important in the treatment of depression: Low levels of self-efficacy have been shown to be related to anxiety and depressive symptoms (Muris, 2002), and depressed patients who report greater efficacy in their ability to control negative cognitions at the end of treatment are less likely to relapse (Kavanagh & Wilson, 1989).

From the reattribution studies reviewed here, it can be seen that modifying dysfunctional attributions has the potential to bring about lasting change. We propose that this same psychological mechanism could be used to shape auto-biological attributions in such a way as to encourage a more adaptive stance toward the diagnosis and treatment of mental disorders. Indeed, the studies reviewed in this section have attempted to correct maladaptive beliefs (e.g., "intelligence is fixed"; Blackwell et al., 2007) and demonstrated that a change in beliefs led to a change in behavior. The challenge going forward is to use these same basic strategies and techniques to correct the reductionist perspective that dominates public beliefs about depression and introduce a more accurate and hopeful picture of the disorder (Kendler, 2005). For patients suffering from depression, encouraging a perspective that validates the subjective as well as the biological nature of depression is essential. Equally important are the beliefs and behaviors that may be impacted by changing a patient's perspective: treatment seeking, motivation, and expectations for recovery. The remainder of this article directly addresses the challenge of designing and disseminating more accurate biological messages and suggests an attributional framework for considering the auto-biology of depression.

TOWARD A FRAMEWORK FOR INTERVENTION

Imagine a conversation between a therapist and patient in which the patient seeks to understand the biological aspects of his or her depression. A variety of approaches could be taken by the therapist—a focus could be placed on etiology (the biological origins of the disorder), on the patient's current experience (the biological factors that influence mood), or on the potential for treatment (the biological basis of symptom reduction). To better define these approaches, we suggest that to focus on etiology is to apply a *retrospective* frame, whereas to focus on symptom reduction is to apply a *prospective* frame. A review of the biological messages provided in existing experimental studies reveals that most of those messages would fit into the retrospective category, by focusing on the presumed neurochemical or genetic causes of a disorder (that, in any individual case, cannot be determined after the fact). We identified only two recent experiments (Lebowitz & Ahn, 2015; Lebowitz et al., 2013) that included examples of prospective framing—a mention of the role of epigenetics and neuroplasticity in depression.

Framing auto-biological information in retrospective versus prospective ways may have strikingly different consequences for beliefs about a disorder. Evidence for the affective consequences of a retrospective frame comes from the stigma literature (Weiner, Perry, & Magnusson, 1988). Stigma related to physical ailments (like cancer) is considered onset-uncontrollable and is associated with altruistic emotional reactions—increased pity, liking, and help-giving. Stigma related to mental–behavioral problems that are often, justifiably or not, considered onset-controllable (like addiction) is associated with lack of pity, dislike, and anger. Introducing biological information about depression in a retrospective frame may help shift the disorder in the eyes of the patient and/or the perceiver from the onset-controllable category to the onset-uncontrollable category, thus reducing blame. Indeed, the only aspect of stigma that is reliably reduced by biological information is blame (Haslam & Kvaale, 2015; Kvaale et al., 2013). This finding has been confirmed in qualitative interviews, where patients report experiencing a sense of relief once they are able to attribute the cause of their disorder to biology rather than their own failings: "If it is a genetic disease—well—then it is not your fault, it's just the way it is" (Laegsgaard, Stamp, Hall, & Mors, 2010, p. 475). Taken together, applying a retrospective biological frame to depression—one that emphasizes the biological/genetic aspects of etiology and the uncontrollable nature of the onset—could be helpful for reducing blame by shifting the attribution from an internal cause to an external one.

However, while retrospective messages may reduce blame, they may be less adaptive for coping with current symptoms or motivating engagement in treatment. The results of the existing experimental work on biological explanations clearly implicate the genetic essentialism perspective, and particularly that biological information framed retrospectively leads to assumptions of immutability or, to use the attribution theory term, stability. The work of Blackwell and colleagues (2007) has shown the considerable psychological costs of such stable, or "entity," theories for multiple behavioral outcomes. On this point, the extant work on biological messages, with its essentialist implications, reveals its most fundamental weakness: By portraying biology as a fixed entity, which confers immutable illness status on some but not others, these messages actually violate a key principle of systems biology, that is, the dynamic and nondeterministic nature of biological systems over time.

Biology at the level of the organism is characterized by both stability and mutability. That is, biological systems are dynamic, responsive to the environment, and in many cases highly plastic over time (Bateson et al., 2004; Turrigiano & Nelson, 2004; West-Eberhard, 1989). There are few, if any, identified biological systems—from the level of molecules all the way up to ecosystems—that do not have some mechanism for adaptation to changes in the environment. Therefore, paradoxically, the inclusion of a systems-based, dynamic understanding of biology in framing mental illness may have the salutary effect of shifting beliefs away from stability and immutability, and toward the unstable and changeable end of the attributional spectrum. A prospective biological frame, which emphasizes the possibility of change, has potential as an interventional tool to encourage adaptive coping and treatment seeking in those affected by mental illness. Such a frame may be especially useful when applied to depression, a disorder in which hopelessness is a symptom and for

which treatment expectations can significantly enhance symptom reduction (Rutherford et al., 2010).

Framing the biology of depression retrospectively versus prospectively leads to distinct predicted patterns of attributions. The attributions that are predicted to follow from each type of frame are depicted in Figure 1. According to this model, a retrospective frame leads to external attributions of depression, in which the disorder is due to "my biology" instead of "me." A retrospective frame also leads to attributions of stability and uncontrollability, both of which lessen personal responsibility but increase pessimism (and in the case of depression, hopelessness) regarding prognosis. In contrast, a prospective frame encourages internal attributions through a rejection of static brain–mind dualism (i.e., "my biology is me"; Kendler, 2005). The affective consequences of an internal biological attribution are unclear; personal responsibility may increase, which can be accompanied by guilt (but note that guilt, unlike shame, tends to motivate behavior, and so may actually be helpful in the context of treatment seeking; Weiner, 1994).

A prospective frame also leads to the attribution of instability, which is critical for increasing effort and motivation in response to the possibility of change (Dweck, 1975), and controllability, or the assertion that an individual can exercise at least some degree of control over auto-biological systems. This framing should reduce feelings of hopelessness and encourage the active engagement of strategies to reduce discomfort (Abramson et al., 1978). Importantly, however, according to self-efficacy theory, merely knowing that a process is controllable is not sufficient; an individual must also be convinced of his or her capacity to exert control (Bandura, 1977). This insight requires an interventional strategy that is both persuasive and convincing—one that provides accurate and comprehensible information along with compelling evidence of its credibility.

Changing the Message

Thus far, we have identified the gap between what we know about the biology of mental illness and we have what is communicated to those who are affected, and reviewed the existing literature, which suggests that the biological explanations provided to date have been mostly harmful. We explored attribution theory as a putative psychological mechanism for these effects and proposed a framework for presenting messages about biology that more accurately communicate scientific reality and have the potential to shape healthier auto-biological attributions. But what might such a message actually look like? How can science communicators and clinicians distill the essential biological truths into a message that conveys complexity, while at the same time being simple enough for a nonscientist to understand and benefit from?

Although the scientific literature on neuroplasticity and epigenetics is rapidly growing, the technical nature of these literatures can be intimidating to nonexperts. Luckily, a number of institutes and organizations exist with the explicit goal of relaying scientific information to general audiences. These groups have created communication resources such as videos and interactive web content that are broadly understandable and yet maintain their scientific integrity (a selection of such resources can be found in Appendix S1). These

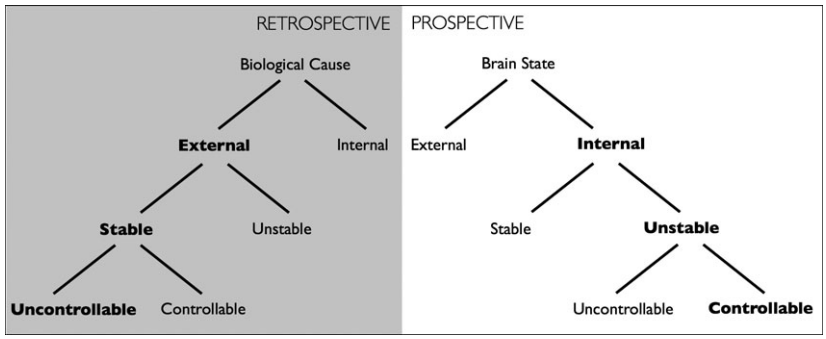

Figure 1. Predictive model depicting the attributional consequences of framing the biology of depression with a retrospective or prospective frame.

resources are ready and available for dissemination, but can also serve as a model for investigators who want to develop their own materials for communicating their science to a broader audience. A motivated consumer in search of readily digestible scientific content need look no further than the work of these groups to find comprehensible, accurate descriptions of epigenetics and neuroplasticity.

The overarching message that should be communicated in a prospective biological frame is that experience can change the brain. Indeed, one could go even further and argue that brains are "designed" to be responsive to experience (Belsky & Pluess, 2009; Kolb & Whishaw, 1998). In clinical practice, this message can be individually tailored to the relevant experiences or concerns of an individual. For example, a patient who is hesitant to practice emotion regulation strategies outside of a session could be provided with readily understandable information about long-term potentiation and the importance of repetition to achieve lasting neurobiological change (e.g., Bliss & Collingridge, 1993; Carr, Jadhav, & Frank, 2011).

A clinician or communicator need not start from scratch in designing an auto-biological message. An excellent example of messaging research can be found in the work of the Frameworks Institute, a nonprofit organization that focuses on reframing social and scientific scholarship for public consumption. While the Frameworks Institute has not (yet) focused their efforts directly on framing biological information about mental disorders for patients, projects they have conducted on early childhood development and child mental health are a rich source of examples and ideas for translating complex science into simple, comprehensible messages for public audiences (Davey, 2010).

The first step in the Frameworks Institute's strategy is to collect information about the currently dominant thought patterns on a topic (e.g., child mental health) by surveying the general public and comparing the results to data collected from scientific experts on the topic (Kendall-Taylor, 2012). Having identified the "gaps in knowledge" between the two groups, they then design a "simplifying model" or metaphor that can be used to convey a concept to the public in a simple, easily comprehensible format. For example, the simplifying model "Levelness" emerged from testing as the best metaphor for explaining the concept of child mental health (Erard, Kendall-Taylor, Davey, et al., 2010). Here, the term *levelness* is used as it is applied to furniture, like a table. A table that is not level is not optimally functional, in the same way that a child with poor mental health cannot function optimally. A child's brain developing on a "level floor," with supportive relationships, good nutrition, and health care, will function better than a brain that develops on a "sloped or slanted floor," characterized by abuse or violence, unreliable relationships, and poor resources. The strengths of this metaphor are that it can be used to advocate for multiple pathways of intervention (fix the floor, fix the table, or both), as well as the importance of intervening early ("little wobbles" can become "bigger wobbles" later if not fixed during childhood).

Three principles of effective messaging can be gleaned from the Frameworks approach. First, an effective message need not contain biological language to explain a biological concept. Frameworks projects use nonbiological metaphors to ensure comprehension from the broadest possible audience (Erard, Kendall-Taylor, Davey, et al., 2010). Second, the approach to background research and the criteria for selecting an effective metaphor will depend on whether the goal is to introduce a new concept (e.g., epigenetics; Erard, Kendall-Taylor, Simon, et al., 2010) or rework an entrenched, counterproductive set of ideas (e.g., depression is a chemical imbalance). Third, an effective metaphor must be generative, meaning that it can be built upon and expanded by its users without losing its conceptual utility (Erard, Kendall-Taylor, Davey, et al., 2010). We strongly advocate for the use of a Frameworks-type approach for designing effective metaphors to communicate complex biological concepts in a way that can be readily understood and used by patients. The institute's website (listed in Appendix S1) is an excellent source of research reports, metaphor examples, and training modules for prospective "messengers."

Message Dissemination

Once an effective biological message has been designed, the next challenge lies in its dissemination. Messages about mental health tend to receive the broadest dissemination through public service announcements by national organizations (e.g.,

National Institute of Mental Health [NIMH], National Alliance on Mental Illness [NAMI]). As evidence accumulates for the importance of thoughtful communication of biological information about mental disorders, it is our sincere hope that such organizations will refine the messages that they publicly disseminate. Such efforts should be based on empirical testing of prospective messages (similar to the Frameworks approach) with both patient and nonpatient samples.

For researchers seeking to communicate their findings to the general public, we recommend consulting the educational resources provided in Appendix S1 for examples. In addition, care should be taken in communicating with the media and general audiences to avoid reductionist explanations of behavior (Miller, 2010). Investigations of media portrayals of neuroscientific research find that reductionistic, "essentialist" messages predominate, along with enthusiastic endorsements of the potential of novel therapeutic techniques that, in reality, require years of further testing before they can be incorporated into the treatment of psychiatric disorders (Racine, Waldman, Rosenberg, & Illes, 2010). Portraying such techniques as biological "silver bullets" diminishes the role of existing psychological treatments and reinforces the notion of mental illness as a "brain disease" that can only be repaired through biological intervention. Researchers can help reverse this trend by carefully attending to media portrayals of their work and guarding against exaggerated claims that could alter public expectations for the future of biological interventions.

For clinicians, we recommend incorporating more accurate biological messages directly into the existing structure of clinical interactions. Indeed, providing patients with information about their symptoms and linking that information to practical management skills is not a new approach; rather, these two components are the basis of *psychoeducation*—a "catch-all" term that describes didactic exchanges between therapist and patient (Goldman, 1988). Psychoeducation can be delivered as a stand-alone intervention or in conjunction with a course of individual or group psychotherapy, and recent approaches have extended beyond the traditional informational model to include more active skill instruction and problem solving (McFarlane, Dixon, Lukens, & Lucksted, 2003). As such, the existing psychoeducational framework provides an ideal structure for delivering information about the biology of a disorder to patients in the early stages of treatment. Through biologically informed psychoeducation, a dynamic, systems-oriented biological "theory" of dysfunction could be integrated into treatment from the beginning and could play an integral role in identifying symptoms, setting improvement goals, and establishing a treatment rationale.

An example of an approach that already integrates biological information is the psychoeducational sessions that are incorporated into cognitive behavioral therapy (CBT) for children with obsessive-compulsive disorder. Using kid-friendly metaphors, symptoms are described as "brain hiccups" and the brain as a "worry computer" that sometimes sends the wrong signals at the wrong times (March & Mulle, 1998). For parents, a discussion of the treatment rationale is accompanied by positron emission tomography (PET) images showing a reduction in abnormal neural activation following a course of CBT. Other psychoeducational approaches may not reference biology directly but incorporate some of the illness attributions discussed here. For example, in interpersonal therapy for depression (IPT; Weissman & Markowitz, 1994), patients are given a diagnosis and assigned a "sick role" early in treatment, which functions to reduce self-blame, externalize the disorder, and reinforce both its episodic nature and the likelihood that treatment will be beneficial. Consistent with the present model, patients in IPT also take responsibility to overcome the sick role throughout the course of treatment, in effect shifting themselves from a "retrospective" frame to a "prospective" one.

The environment of trust fostered in an effective therapy relationship may be an ideal setting for exploring the meaning of auto-biological information for identity. In the same way that clinicians across approaches (e.g., acceptance and commitment therapy, Hayes, Strosahl, & Wilson, 2012; dialectical behavior therapy, Linehan, 1993) seek to find a balance between encouraging acceptance and promoting change, a clinician can help a patient find a balance between accepting auto-biological truths (e.g., depression runs in my family) and considering the potential for auto-biological change (e.g., therapy can change the brain). Metaphor is already considered a useful tool in psychotherapy (Martin, Cummings, & Hallberg, 1992), and effective

biological metaphors could be employed early in treatment and tailored to individual patients to maximize their utility. In attributional terms, a skilled clinician armed with an effective biological metaphor could employ both retrospective and prospective framing based on the current needs of a patient, with the goal of shifting the patient's attributions away from guilt or self-blame and toward agency and motivation for change.

Some therapists already integrate biological information into their practice and do so according to their own clinical experience and intuition rather than via a structured, systematized approach. This type of communication may have great value, but we suggest it be approached with caution. Indeed, clinicians in a survey study reported less empathy toward fictional patients whose disorders were attributed to biology compared to psychosocial factors (Lebowitz & Ahn, 2014). This worrying replication of the stigma findings in a sample of clinicians suggests that biological essentialism may bias interactions even within the context of mental health treatment. Keeping this in mind, therapists should approach discussions of biology with the same empathy and compassion that they bring to other clinically sensitive issues and carefully assess each patient's auto-biological beliefs for signs of self-blame or guilt that could undermine treatment progress.

The conclusion we hope the reader will draw from this article is that biological information has the potential to powerfully impact an individual and thus should be communicated with careful consideration and forethought. Systematic research is needed to guide the information that clinicians and other providers give patients about the biology of depression, and the faster the science changes, the more urgent this need becomes. Different types of messages—retrospective or prospective—serve different purposes and ultimately can be shaped to the needs of the patient. The idiosyncratic nature of mental illness beliefs does not preclude research on the topic or discourage the development of a set of guidelines for psychoeducation and clinical communication more broadly.

Techniques for Reattribution

We encourage clinicians and communicators to draw from current examples (Appendix S1) to design and disseminate better biological messages. However, for many patients, simply providing a specific message may not be enough. As we have learned from self-efficacy theory, information-only interventions are less effective than those that provide information along with demonstration or practical skill instruction (Bandura, 1977; Schindler et al., 2015). To achieve true attributional change, patients may need to learn through active experience that biological processes are unstable and controllable. How might we provide such a demonstration? Demonstrating neuroplasticity or changes in gene expression within the current mental health treatment or research context is technically unfeasible, but other proxy measures can be used to help people link behavior and biology. Biofeedback, whether it be physiological via electrocardiogram (Lehrer et al., 2003) or neural via EEG (Sterman, 1996) or real-time fMRI (deCharms, 2008), offers a more direct way to show an individual how a thought or behavioral strategy can impact a biological system. Real-time fMRI, while still expensive and not widely available, has the advantage of anatomical localization compared to other biofeedback approaches and has been successfully applied to reduce discomfort in chronic pain patients (deCharms et al., 2005). Another technique with a controversial but growing evidence base is neurocognitive training, which purports to enhance general intelligence or working memory through daily practice of a cognitive task (e.g., Jaeggi, Buschkuehl, Jonides, & Perrig, 2008; though see Melby-Lervag & Hulme, 2013, for a critical review of such studies). Self-directed "brain training" programs have grown in popularity, and customers pay to access cognitive tasks designed to train specific domains through repeated exposure. How any purported neural benefits are demonstrated to consumers, however, is unclear (above and beyond the observable improvement in behavioral performance).

A promising technique that could be harnessed as a intervention tool for attributional change is the use of mobile devices and smartphones to deliver attribution-relevant information in real time (Boschen & Casey, 2008; Luxton, McCann, Bush, Mishkind, & Reger, 2011). One could imagine an application that integrates instruction and strategy use with some type of feedback from physiological data collected by a sensor. For example, an existing app called "Stress Check" uses the

iPhone camera to detect heart rate—the developers of the application have marketed it as a tool to help people monitor and learn to control their reactions to everyday stressors (Dickinson, 2011). A potential app that could be relevant for depression would be one that tracked mood and provided feedback about the efficacy of cognitive regulation strategies using heart rate or some other proxy physiological marker that is linked to emotional distress.

A number of questions deserve to be considered when designing and testing such interventions. For example, what level of biological detail is most useful? Does demonstrating a direct link to the brain (e.g., real-time fMRI neurofeedback) confer more credibility than can be achieved with peripheral physiology alone (e.g., heart-rate biofeedback)? How should metaphor and demonstration be used together to communicate auto-biological information, and what are the qualities of such a metaphor? Considering the unintentional harm wrought by the "chemical imbalance" explanation, which concepts should a new biological metaphor emphasize and which should it avoid (e.g., dynamic rather than static, growth-oriented rather than disease-oriented)? While preliminary answers to these questions can be gleaned from existing work on message framing and biofeedback interventions, they deserve to be explored in the context of theoretically driven interventions that attempt to link the biology of depression to the subjective experience and beliefs of individuals with the disorder.

SUMMARY AND CONCLUSION

In this review, we have sought to articulate a challenge that is as yet unmet by current mental health research. What emerges from a review of the neuroscience literature is a growing set of biological mechanisms that are dynamically involved in initiating and maintaining the symptoms of mental disorders such as depression. No single biological cause, and no one level of analysis, can adequately define such inherently subjective, brain-based disorders (Kendler, 2005). The complexity of defining the biological nature of disorders like depression poses substantial difficulty for researchers and the general public alike. Perhaps unsurprisingly, reductionist explanations such as "depression is a chemical imbalance" have persisted despite their limited scientific value. From an individual patient's perspective, however, such reductionist explanations can be harmful—increasing stigma against those with a diagnosis and leaving them with little sense of agency or hope for improvement.

We are not proposing that biological explanations should be dismissed entirely—nor would such a dismissal be desirable or even possible, given the degree of dissemination of biological information that has already occurred. Rather, we propose that efforts be made at the level of national agencies and organizations (i.e., NIMH, NAMI) to design and disseminate simple and yet accurate biological messages about disorders such as depression, which could also counteract reductionist messages from the pharmaceutical industry. Simultaneously, we encourage clinicians and researchers to present auto-biological information to patients and participants in a way that empowers recovery and encourages optimism. Drawing on research from attribution theory, we explored the potential for auto-biological information to shape how patients view themselves in relation to their disorders. In doing so, we built on previous work (Lebowitz, 2014) and proposed a theory-based framework for presenting biological information in a way that encourages agency, rather than destiny. By focusing on the active future (prospective) rather than the causal past (retrospective), we suggest that a systems-based, dynamic understanding of the biology of mental illness can shift beliefs away from stability, entity, and immutability, and toward the unstable, incremental, and changeable end of the attributional spectrum.

The initial model and predictions presented here await testing but hopefully have established a starting point for interventions that aim to change beliefs about the biology of psychological disorders. Whether delivered as laboratory-based microinterventions (Strauman et al., 2012) or integrated into a broader psychoeducational approach with patients, auto-biological messages have the potential to powerfully shape the attitudes and behaviors of individuals struggling with mental illness. Such an individual is unlikely to seek or engage in treatment if he or she believes that the treatment is incapable of correcting underlying biology. Therefore, dissemination of evidence-based psychological treatments should be accompanied by a parallel effort to educate the public

that experience—including psychotherapy—can change the brain. By portraying biological systems as responsive to experience rather than fixed, we can achieve scientific accuracy as well as convey optimism. We argue that a realistic understanding of the role biology plays in mood and behavior can be harnessed to correct dysfunctional attributions and improve mental health outcomes for the patients we serve.

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SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article:

Appendix S1. Resources for communicating neuroscience information to general audiences.