IJNRD.ORG ISSN : 2456-4184
INTERNATIONAL JOURNAL OF NOVEL RESEARCH AND DEVELOPMENT (IJNRD) | IJNRD.ORG
An International Open Access, Peer-reviewed, Refereed Journal

Etiological Underpinnings of Mental Illnesses: A Critique of Reductive Approaches

Ruheen Sawhney
Student
The British School

Abstract

Language matters when it comes to describing illnesses, their symptomatology, and their diagnostic and prognostic trajectories. While language around describing physical illnesses is straightforward, there is a considerable amount of variability, confusion, and misconceptions in defining or describing mental illnesses. Such confusions and misconceptions often dilute the understanding of mental illnesses, thereby encouraging reductionism. A part of the reason for reductionist approaches to mental illnesses might be attributable to the heterogeneity of experiences surrounding mental illnesses. This means similar diagnoses of mental illnesses can be accompanied by radically different symptom profiles. The current review aims to identify and critique the reductionism inherent in the narrative of four common mental illnesses (schizophrenia, bipolar disorder, generalized anxiety disorder, and major depressive disorder). In doing so, the paper will review the etiological sources of these illnesses and will bridge structural factors with individual factors to establish a more holistic understanding.

Keywords: Mental illness, Misconceptions, Etiology, Symptomatology.

Introduction

One of the starkest distinctions between mental and physical illness is that the former is never amenable to being explained through a single unifying and homogeneous lens. While cutting-edge technological aid has enabled sophisticated ways of detecting the underlying causes of physical illness, predicting statistically accurate

prognostic trajectories, and making way for advanced treatment plans based on addressing the underlying cause, the same can hardly be said for mental illnesses. When it comes to mental illness, it is imperative to understand the underlying causes through multiple perspectives, ethnocultural factors, systemic discrimination, oppression, and so forth. This renders the narrative discourse around mental illness to be one steeped in non-linearity, complexity, and an inherent heterogeneity in determining categorical root causes. While commonly used models of mental illness like the bio-psycho-social model (Engel, 1977) have gained prominence in explaining the diversity of causes in the manifestation of mental illnesses, it has drawn heavy criticism for its component referents (the biological, the psychological, the social) not always being in concordance with each other (e.g., Alvarez et al., 2012). In other words, as a multilevel model, the bio-psycho-social paradigm obscures which level of the system is the most clinically important at any given point in time (Sadler & Hulgus, 1990). While there are contemporary revisions to the bio-psycho-social model and the existence of other competing models, categorical etiological underpinnings remain far from accessible when it comes to mental illnesses. The current paper will attempt to bridge this gap by holistically integrating the etiologies of four of the most etiologically heterogeneous mental disorders. In doing so, the paper will identify gaps in knowledge that pertain to the current global mental health crisis. Just as terminologies relevant to physical illnesses are a part of everyday discourse practices, the present paper aims to add to the recent advances in enriching mental health literacy by building an accessible vocabulary for identifying the root causes of mental illnesses.

Differences in etiological explanations

The roots of mental illness are attributable to a combination of risk factors like genetic predisposition, personality traits, attachment styles, parenting styles, drug abuse, and so on and so forth. In a meta-analysis to recognize the root cause of mental illnesses, Furber et al. (2016) identified 10 primary and 23 secondary risk factors associated with impaired mental health (see Furber et al., 2016, for review). The meta-analysis also revealed an interesting pattern in the literature where a few disorders were overstudied (e.g., schizophrenia) with a focus on biological and psychological risks over and above other potentially important factors like environmental stress or occupational hazards. Broadly, this paper will focus on three schools of etiological explanations: cognitive-behavioral, biological, and social. The rationale for elaborating on broadly distinct schools of etiological understanding is directly proportional to the aim of the paper in making mental health

language more accessible. By breaking the language of mental health into actionable component units, it might be more comprehensible for more people to grasp the expansivity of the concepts.

Cognitive-behavioral school of thought

Under a cognitive lens, mental illnesses are a result of individuals' perception of events over and above the event itself (Beck, 1964). This view is also rooted in the modern-day understanding of emotions- we feel and emote differently to different stimuli or situations because we appraise each stimulus or situation differently. Even simply, there exists a great deal of individual variability in appraising the significance or insignificance of any event. The cognitive model of mental illness proposes that how people feel is correlational with how they interpret the situation- stronger feelings will entail deeper psychological reaction than mild feelings. Beck (1978) outlined three levels of the cognitive model: core beliefs, dysfunctional assumptions, and negative automatic thoughts.

Core beliefs are deep-seated beliefs about the self, others, and the world. Core beliefs are highly malleable to childhood experiences, are strongly internalized, and are often thought of in absolute ways (e.g., I am a bad person, the world is an unjust place, things are forever doomed).

Dysfunctional beliefs are rigid, conditional rules that people adopt, and over time internalize. They are also internalized and are expressed as either "if-then" or "should" statements. For example, "If I do not get an A+, then I should not attempt the test at all."

Negative automatic thoughts are thoughts that are involuntarily activated in certain situations. They typically center around themes of extreme negativity, hopelessness, and worthlessness. For example, the constant feeling of being a failure and having intrusive thoughts like "I am a failure".

Together, the cognitive model accounts for the dysfunctional thoughts that implicitly underlie mental illnesses. In a cognitive model, an individual's idiosyncratic experiences are understood within their own individualized context. A formulation of mental illness as conceptualized through this model involves having "a hypothesis about the causes, precipitants, and maintaining influences of a person's problems" (Eels, 1997).

The biological school of thought

The biological model assumes that mental disorders like schizophrenia, major depressive disorder, attention-deficit/hyperactivity disorder (ADHD), and substance use disorders are biologically-based brain

diseases and that certain aspects of our biology are responsible for our thoughts and behaviors. In this paper, the three major biological causes of mental disorders will be explored: genetics, the dopamine hypothesis, and neural correlates. There is substantial evidence that genetic inheritance tends to play a significant role in the development of mental abnormalities such as schizophrenia. If one has a parent with schizophrenia, for example, the risk of developing schizophrenia is nearly six times higher (Goldstein, Buka, Seidman, & Tsuang, 2010) than if one does not. Furthermore, as one's genetic relatedness to family members diagnosed with schizophrenia increases, so does one's risk of developing schizophrenia or other similar mental disorders (Gottesman, 2001). However, an excessive focus on genetics is not sufficient to capture the complexities of mental illnesses which manifest in different forms and capacities. A more balanced view of framing genes as a potential contributor to mental illness is the stress diathesis model. The stress diathesis model informs how both genes and environmental factors coincide in giving rise to a mental illness. In order words, in the grand debate between nature versus nurture's role in mental illness, the stress diathesis model calls for a more balanced approach between the two. Prior research indicates that while a biological explanation for mental illness reduces blame and limits stigma associated with mental illness, it may unintentionally exacerbate other components of stigma, particularly the benevolence and dangerous stigmas. The other side of the argument is that psychosocial explanations of mental illnesses are promising, but they obscure the growing evidence regarding genetic and biological factors (Corrigan & Watson, 2004). Overall, more than a strictly biological or social approach, the recommendation on language use in describing mental illnesses tilts the scale toward a balanced and holistic approach that does not obfuscate important details, yet does not over-pathologize mental illnesses that ultimately lead to discrimination and otherization.

The psychosocial school of thought

Mental health does not exist in isolation from social and societal factors, therefore, mental illness, too, has strong roots in the inequitable distribution of resources, power, and money, vicious cycles of inter-generational poverty, and ill-health (Hart, 1997; Jacob, 2013). Public health reformers who long advocated social reform on political, economic, humanitarian, and scientific grounds had long acknowledged the reciprocal relationship between poverty and disease (Porter, 1997). These social determinants of health significantly affect mental health. Poverty, gender, violence, cultural tensions, social discrimination, political oppression, ethnic

cleansing, armed conflicts, and forced migration are associated with depression, anxiety, and other common mental disorders. Poverty works through the experience of insecurity, helplessness, hopelessness, rapid social change (like inflation), risk of violence, and physical illness- all of which end up affecting one's mental health. Overall, the psychosocial lens conceptualizes mental illness as a complex product of social and systemic level constructs. In doing so, the psychosocial school of thought shifts the conversation from the individual to the social and systemic, thereby instigating thoughts on the role of the society in the inception and maintenance of mental illness. Indeed, a study by Lam et al. (2009) found that psychological accounts of mental illness were relatively more de-stigmatizing than purely biological accounts.

The role of integrative reviews

Integrative reviews synthesize the findings from a large corpus of literature to answer pertinent questions and make knowledge more accessible to audiences beyond academia. When it comes to mental health, such initiatives are the need of the hour given the climate of stigmatization, discrimination, and otherization based on mental illnesses. Oftentimes, such stigmatizing attitudes stem from a position of lack of awareness. To that end, integrative reviews serve the bigger purpose of demystifying certain unknowns to make way for more awareness. Such endeavors also hold the potential of influencing larger systemic level changes like policy change and legislative change to better serve marginalized identities. Kinderman (2009) has remarked that "psychological models of mental disorder" or the "mediating psychological processes model" have the potential to assist mental health service policy development and implementation. The following sections of the paper will focus on four different etiologically complex and commonly misunderstood mental illnesses. The overarching rationale for highlighting these mental illnesses is to shed light on the heterogeneity of issues underlying these illnesses thereby advocating for a more expansive and holistic mindset when it comes to understanding psychological illnesses.

Schizophrenia

Schizophrenia consists of a mental condition that modifies the perception of reality of individuals impairing memory and the ease of doing basic activities that require attention, concentration, and memorization. In a profile on cognitive impairments in schizophrenia, cognitive deficits have been regarded as a central feature. Evidence suggests that there are discrete domains of cognitive impairment in schizophrenia For example, Bilder

et al,. (2002) found mild to moderate deficits in attention, verbal fluency, working memory, and processing speed. Such cognitive deteriorations fuel the discourse around the neuroscientific causes of schizophrenia. While there are many biological explanations for schizophrenia, one of the most commonly discussed is the dopamine hypothesis. The dopamine hypothesis states that schizophrenic symptoms are a cause of hyperactive dopamine transmission in the brain (Carlsson, 1963). However, the dopamine hypothesis has been challenged. First, the symptoms of schizophrenia include both positive (e.g., hallucinations and delusions) and negative (e.g., anhedonia, poverty of speech) symptoms. The initial dopamine hypothesis of schizophrenia accounted exclusively for positive symptoms and did little to explain the negative ones. Furthermore, the symptomatology of schizophrenia displays great variability in its manifestation. There might be different combinations of positive and negative symptoms cooccurring. In that situation, the dopamine hypothesis, while an important scientific breakthrough, does little justice to understanding the illness from a holistic perspective. The recent development in the dopamine hypothesis proposes hyperactive dopamine transmission in the mesolimbic areas of the brain and hypoactive dopamine transmission in the prefrontal cortex. This recent extension in the dopamine hypothesis is not common knowledge in the general population (e.g., in psychology textbooks), and this goes on to show the potentially dangerous ramifications of reductive science.

A largely understudied area in the etiology of schizophrenia is the psychosocial aspect leading up to the illness. A meta-analysis of the psychosocial underpinnings of schizophrenia revealed numerous social factors that contribute to the illness- like major life events, migration-related stress of minority groups, childhood trauma, and so on. Yet, in the mainstream discourse, we hardly discuss theories like minority stress theory and social disadvantage in light of serious mental health considerations like schizophrenia.

Bipolar Disorder

Bipolar disorder is characterized by extreme mood swings that include emotional highs (mania) and lows (depression). Cognitive profiling of bipolar disorder reveals that the most affected domains are attention, verbal learning, memory, and executive functions (Sole et al., 2017). However, similar to schizophrenia, understanding bipolar disorder as just characterized by deficits in these domains can elude the role of stressors that play a role in the development and maintenance of bipolar symptoms. Research has documented that social and environmental factors predict the prognosis of bipolar disorder and that childhood abuse is associated with a

more severe illness course (Johnson et al., 2016). Similarly, poor family functioning, low social support, and exposure to chronic stress work in various combinations to lead to bipolar disorder. It is important to consider that negative social stressors like poor family functioning, low social support, and chronic stress are outcomes of systemic poverty. Therefore, examining and scrutinizing the symptomatology of bipolar disorder without factoring in structural issues at play does a disservice to understanding the etiological heterogeneity of the disorder.

Major Depressive Disorder

Commonly known as depression, it is a buzzword to represent feelings of persistent sadness, and the trio of hopelessness, helplessness, and worthlessness. Depression manifests in numerous ways, some more insidious (e.g., difficulty getting out of bed, canceling plans) than others (e.g., visible persistent sadness). The Covid-19 pandemic has fuelled a mental health crisis in addition to a physical health catastrophe where social isolation, lack of perceived support, loneliness, fear of infection, suffering and death, grief and bereavement have worked in concert leading to record levels of mental health concerns across the world. These collective mental health crises during the time of Covid-19 accurately capture the futility of one-size-fits-all reductive approaches when it comes to the etiological underpinnings of complex illnesses. Cognitive theories of depression have been hypothesized as a way to understand the developmental trajectory and maintenance of depression. Cognitive theories like Beck's theory (Beck, 1987), hopelessness theory (Abramson et al., 1989), and the response-styles theory (Nolen-Hoeksema, 1991) identify distinct cognitive vulnerability factors (like dysfunctional attitudes, negative cognitive styles, ruminative response styles) that are hypothesized to contribute to the onset and maintenance of schizophrenia. On the other hand, neuroimaging studies have revealed brain abnormality in individuals with depression, thereby fuelling the discourse around depression being the result of altered neural structures, most notably the amygdala and the hippocampus. However, once again, an examination of these etiological explanations in isolation, without connecting them to larger social and structural contexts like systemic oppression, discrimination, and multidimensional poverty renders the picture incomplete.

Generalized anxiety disorder

Generalized anxiety disorder is marked by excessive anxiety and worry about everyday life without any apparent reason. This disorder is prominently characterized by the persistence of free-floating worry that invades

every aspect of life. The cognitive theory of generalized anxiety disorder regards worry, the core component of the disorder as a function of maladaptive information processing, biased in the direction of perceived threat (Aikins & Craske, 2001). In order words, according to the cognitive school, we experience worry due to a persistent preoccupation with a perceived threat in everyday life and that leads to an information processing style that is maladaptive and dysfunctional. Neuroscientific advances have corroborated these hypotheses with patterns of brain waves between afflicted and healthy controls. However, to understand the roots and the ramifications of the illness, we once again, need to examine the etiology in the context of structural level issues like chronic stress as a result of poverty that might heighten one's sensitivity to perceiving threat in the environment, systemic discrimination, hate crimes, and so on.

The bio-psycho-social model: it is enough to counter reductive discourses?

The bio-psycho-social model (Engel, 1977) examines the complex interconnections between biology, psychology, and social factors in the manifestation of mental illnesses. First conceptualized as a counter to strictly medical models, the biopsychosocial model has come a long way to spark discourses on the interactions between individual etiological factors in the maintenance of mental illnesses. However, despite the existence of such a model, common debates and discussions on mental illnesses still continue to center the biomedical and genetic etiologies over and above psychosocial factors. It is a reductive practice. The significant downside of such conventions is that they lead to the assumption that mental disorders are unlikely to improve or abate (Lebowitz & Applebaum, 2019). Genetic and biomedical explanations also contribute to a greater harm by increasing people's confidence in the effectiveness of biomedical treatments (like pharmacotherapy) but decreasing their confidence in the effectiveness of non-biomedical treatments (like psychotherapy). Thus, clearly, the existence of the bio-psycho-social model is not a sufficient counter to the problem of stigmatizing mental illnesses by conceptualizing them from a biomedical perspective. This paper proposes that this problem could potentially be mitigated by raising awareness and creating an accessible vocabulary for the general population to understand the role of etiological heterogeneity in any mental disorder.

The concerns with White psychology

The Diagnostic and Statistical Manual (DSM) of mental illnesses is based exclusively on a sample that is White. In fact, Whiteness in psychology is a concern for the field as a whole as such an approach overly

represents the Western population while underrepresenting the diversity of the Global South (Barrett, 2022). Therefore, the DSM is not an accurate representation of mental illness etiologies as they unfold in diverse contexts, like India, for instance. The structural and systemic issues in India (e.g., the interaction of class and caste) are naturally qualitatively different from any country in the Global North, and therefore any bio-psycho-social conceptualization based on guidelines in the DSM would be fundamentally incomplete in capturing the richness of contexts in India. If anything, the current paper is a clarion call to develop etiological models based on indigenous psychology and to conceptualize a shift from Western, White psychology when it comes to representing mental illnesses.

Conclusion

The current paper is an attempt to bridge the gap between the holistic etiological conceptualization of mental illnesses and public awareness of the same. In sum, the paper has made several recommendations by recognizing the gaps in the literature. One, an accessible vocabulary to discuss mental illnesses is largely absent. Two, there is a general preoccupation with biomedical models to understand mental illnesses. This leads to a reductive approach and does not factor in pernicious systemic forces in the manifestation of mental illness symptoms. Third, to make any real-time progress, indigenous models of mental health need to be developed to capture the richness of contextual drivers of mental illness. Overall, the paper aims to contribute to the literature by recognizing the potential of indigenous psychology to better understand mental health.

References

Abramson, L. Y., Metalsky, G. I., & Alloy, L. B. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological review*, *96*(2), 358.

Aikins, D. E., & Craske, M. G. (2001). Cognitive theories of generalized anxiety disorder. *Psychiatric Clinics of North America*, *24*(1), 57-74.

Alvarez, A. S., Pagani, M., & Meucci, P. (2012). The clinical application of the biopsychosocial model in mental health: a research critique. *American journal of physical medicine & rehabilitation*, *91*(13), S173-S180.

Barrett, H. C. (2022). Psychology within and without the state. *Annual review of psychology*, *73*, 461-487.

Beck, A. T. (1964). Thinking and depression: II. Theory and therapy. *Archives of general psychiatry*, *10*(6), 561-571.

Bilder, R. M., Volavka, J., ál Czobor, P., Malhotra, A. K., Kennedy, J. L., Ni, X., ... & Lieberman, J. A. (2002). Neurocognitive correlates of the COMT Val158Met polymorphism in chronic schizophrenia. *Biological psychiatry*, *52*(7), 701-707.

Carlsson A, Lindqvist M. Effect of chlorpromazine or haloperidol on formation of 3methoxytyramine and normetanephrine in mouse brain. *Acta Pharmacol Toxicol (Copenh)* (1963) **20**:140–4. doi:10.1111/j.1600-0773.1963.tb01730.x

Corrigan, P. W., & Watson, A. C. (2004). At issue: Stop the stigma: Call mental illness a brain disease. *Schizophrenia bulletin*, *30*(3), 477-479.

Engel, G. L. (1977). The need for a new medical model: a challenge for biomedicine. *Science*, *196*(4286), 129-136.

Furber, G., Leach, M., Guy, S., & Segal, L. (2017). Developing a broad categorisation scheme to describe risk factors for mental illness, for use in prevention policy and planning. *Australian & New Zealand Journal of Psychiatry*, *51*(3), 230-240.

Goldstein, J. M., Buka, S. L., Seidman, L. J., & Tsuang, M. T. (2010). Specificity of familial transmission of schizophrenia psychosis spectrum and affective psychoses in the New England family study's high-risk design. *Archives of General Psychiatry*, *67*(5), 458-467.

Gottesman, I. I., & Erlenmeyer-Kimling, L. (2001). Family and twin strategies as a head start in defining prodromes and endophenotypes for hypothetical early-interventions in schizophrenia. *Schizophrenia research*, *51*(1), 93-102.

Hart N. The social and economic environment and human health. In: Detels R, Holland WW, McEwen J, Omenn GS, editors. *Oxford Textbook of Public Health.* 3rd ed. Vol. 1. Oxford: Oxford University Press; 1997. pp. 95–123.

Jacob, K. S. (2013). Psychosocial adversity and mental illness: Differentiating distress, contextualizing diagnosis. *Indian journal of psychiatry*, *55*(2), 106.

Johnson, S. L., Tharp, J. A., Peckham, A. D., & McMaster, K. J. (2016). Emotion in bipolar I disorder: Implications for functional and symptom outcomes. *Journal of abnormal psychology*, *125*(1), 40.

Kinderman, P. (2009). Understanding and addressing psychological and social problems: The mediating psychological processes model. *International Journal of Social Psychiatry*, *55*(5), 464-470.

Lam, D. C., Salkovskis, P. M., & Warwick, H. M. (2005). An experimental investigation of the impact of biological versus psychological explanations of the cause of "mental illness". *Journal of Mental Health*, *14*(5), 453-464.

Lebowitz, M. S., & Appelbaum, P. S. (2019). Biomedical explanations of psychopathology and their implications for attitudes and beliefs about mental disorders. *Annual Review of Clinical Psychology*, *15*, 555-577.

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of abnormal psychology*, *100*(4), 569.

Porter D. Public health and centralization: the Victorian British State. In: Detels R, Holland WW, McEwen J, Omenn GS, eds. *Oxford textbook of public health*. 3rd edn. Vol 1. Oxford: Oxford University Press, 199719–34.

Sadler, J. Z., & Hulgus, Y. F. (1990). Knowing, valuing, acting: Clues to revising the biopsychosocial model. *Comprehensive Psychiatry*, *31*(3), 185-195.

Solé, B., Jiménez, E., Torrent, C., Reinares, M., Bonnin, C. D. M., Torres, I., ... & Vieta, E. (2017). Cognitive impairment in bipolar disorder: treatment and prevention strategies. *International Journal of Neuropsychopharmacology*, *20*(8), 670-680.

Weissman, A. N., & Beck, A. T. (1978). Development and validation of the Dysfunctional Attitude Scale: a preliminary investigation.