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Uncovering the Cause of Depression—A Medical or a Humanistic Approach?

Reflections on Nassir Ghaemi's *On Depression*

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Abstract: S. Nassir Ghaemi takes a broad clinical and philosophical perspective on depression, by challenging its current diagnostic system, the lack of clarity regarding its causality and the controversial treatment efficacy of antidepressants. This essay discusses some of Ghaemi's insightful observations and analyses. Ghaemi rightfully points to the fact that current diagnostic categories for depression ignore any consideration of its causality. The author highlights Aristotle's discourse on causality and its significance for understanding depression. A second valuable observation is that multiple causality legitimises the recognition of the biological existential duality of this condition. From this position, the argumentation for the use of antidepressants to treat those depressions which have an existential determination, becomes disjunctive; Ghaemi seems to favour that the existential pain is formative, even if it leads to depression, and that antidepressants bring little benefit to alleviating this pain.

Keywords: Jaspers, Karl; Ghaemi, S. Nassir; *General Psychopathology*; depression; despair; psychiatry; neuroscience; causality; ontological; existential.

When we look at the advancements made in the medical sciences over the past century, we see a steady accumulation of concrete ideas and practices, as well as decisive breakthroughs in understanding the nature and etiology of disease. Unfortunately, this is not the case when we examine the history of psychiatry. Psychiatry, as we know it today, came to be in a sporadic, conflicting manner, as Roy Porter and Mark Micale claim, "with long periods of advance, stagnation, and even regression," and there continue to be opposing thoughts on the nature and etiology of psychiatric conditions, particularly clinical depression.[1] They argue that, "both empirically and interpretively, extant histories of psychiatry reveal a vastly greater degree of difference among themselves than historical accounts of any other discipline" (*RPH* 5). This claim is supported by Dr. Paul McHugh, a distinguished University Professor of Psychiatry at the Johns Hopkins University School of Medicine, who believes that probably the most distracting issue that is blocking any significant advancements in psychiatry is the "long-standing factionalism" that has been dividing psychiatrists into two camps according to their orientation towards

[1] Roy Porter and Mark S. Micale, "Introduction: Reflections on Psychiatry and its Histories," in *Discovering the History of Psychiatry*, eds. Mark S. Micale and Roy Porter, New York, NY: Oxford University Press 1994, pp. 3-36, here p. 5. [Henceforth cited as *RPH*]

biological or dynamic psychiatry.[2] The initial collision between empirical and interpretative psychiatry occurred in the early twentieth century, beginning with Emil Kraepelin and Sigmund Freud, two of the great Germanic psychiatrists. Despite its efforts, organized American psychiatry was, until now, unable to unify Kraepelin's categorical presentations of psychiatric disorders with Adolph Meyer's view of mental disorders as "reactions of an individual's personality to a combination of psychological, social, and biological factors."[3]

Ever since the deep-rooted establishment of a DSM culture, perspectives on the diagnosis of depression have persisted to ignore its causality. This is due to the fact that the DSM focuses mainly on the use of symptoms to diagnose disorders, and fails to capture the diversity and complexity within disorders. According to McHugh,

> DSM regularly fails to distinguish between conditions with similar symptomatic appearances such as between ordinary sadness and clinical depression… this failure derives directly from the inattention of DSM-III/IV to distinguishing the generative causes of either normal or abnormal mental states.[4]

Unfortunately, there is a reluctance, and even a resistance, by many in the field of psychiatry to change the DSM symptom-based diagnostic system, as "many psychiatrists fear that any classificatory differentiation based on views about the generation of psychiatric disorders will restart the war between the dynamic and biological schools that DSM-III settled" (*PS* online). With regard to clinical depression, we are still debating how much in its spectrum can be attributed to purely biological factors (genetics), purely environmental factors, or to the coalescence of both.

In his book *On Depression*, Nassir Ghaemi approaches the discussion of depression by exploring the complexity of its causality from both a medical and philosophical perspective, as he revisits whether depression is simply a medical disease, whether it is a reflection of the existential despair that we normally feel about various aspects of life and death as part of the normal human experience, and how we can possibly grasp these differences.[5] By raising these topics, Ghaemi refreshingly brings to our attention a much-needed systemic view on depression, as well as a meaningful understanding of "the mix of biology and environment" (*OD* 15), which needs to be re-examined.

The Complex Causality of Depression

In his discussion regarding the causality of depression, Ghaemi takes us back to Aristotle's four different modes of causation, comprising of the material cause, which consists of the immanent material or the substratum that sets everything else in motion, the essence, which constitutes form and pattern, the moving cause (or efficient cause), from which comes the beginning of the change, and lastly, the final cause, for whose sake all the changes take place (*OD* 15-6).

In the context of clinical depression, there is an interplay between the material (first) cause, which is the individual's unmodifiable biological susceptibility to depression (genetics and early life development), and the efficient causes, which are the immediate life events that trigger a clinical depression, such as the death of a loved one, a divorce, or some other form of loss. Ghaemi cautions that "one of the greatest errors in understanding depression is to mistake first and efficient causes, or susceptibility and triggers" (*OD* 15). The first cause must exist in order to result in depression later in life, however, it may not be sufficient in and of itself to lead to clinical depression later in life. However, the efficient causes, the so-called triggers, are not necessary, as depression can occur without them, and, in turn, these events can occur without necessarily triggering depressive episodes; nonetheless, in some individuals, these events lead to depression.

> So first causes are necessary but usually not sufficient; efficient causes are often sufficient, but not necessary. One usually needs both, and neither alone is the cause of depression. [*OD* 16]

The overlap of different modes of causation in psychiatry has also been previously highlighted by the

[2] Paul R. McHugh & Phillip R. Slavney, *The Perspectives of Psychiatry*, 2nd edition, Baltimore, MD: The Johns Hopkins University Press 1998, p. 18.

[3] Jonathan M. Metzl, *The Protest Psychosis: How Schizophrenia Became a Black Disease*, Boston, MA: Beacon Press, 2009, p. 82.

[4] Paul R. McHugh, "Psychiatry at Stalemate," *Cerebrum* (October 13, 2009). Last accessed 8-10-2017, http://www.dana.org/Cerebrum/2009/Updating_the_Diagnostic_and_Statistical_Manual_of_Mental_Disorders/#McHugh_jump. [Henceforth cited as *PS*]

[5] S. Nassir Ghaemi, *On Depression: Drugs, Diagnosis, and Despair in the Modern World*, Baltimore, MD: The Johns Hopkins University Press, 2013. [Henceforth cited as *OD*]

German-Swiss psychiatrist and philosopher Karl Jaspers. Like Aristotle, who sees multiple possible ontological developments in any natural process, Jaspers discusses the philosophical concept of *Existenz*, as infinitely open possibility of becoming that "exists empirically as freedom only," or "open possibility."[6] To Jaspers, *Existenz* can find itself only "in the constant peril of endless reflection, which may end with despair" (*P2* 41).

Hence, Ghaemi poses a critical question: "Is depression then inherent to the lively effort of ontological becoming?" Alternatively, we can ask: Could depression be just the road to exhaustion due to the lack of capacity to tolerate the inherent pain resulting from our search for the final form? The answer has to take into account the understanding of causality. When the first cause contains an inevitable development toward depression, efficient may be just circumstantial.

Jaspers makes a clear distinction between causal and meaningful connections.[7] In order to better grasp the concept of causality, Jaspers advises us to replace the mechanistic model of a one-way causal relationship with the model "of an infinite living network" or "a vast reciprocity" (*GP* 453). Living events are, therefore, seen as "infinite interplay of cycles of events" (*GP* 452).

An existential struggle remains forgotten in our modern diagnostic classification system, which, as already discussed, focuses on symptomatology. One can ascertain from Ghaemi's words an urging for us to wake up from this neglect—a warning that is reminiscent of Jaspers' request for existential understanding (*GP* 308). Jaspers advises that, in our attempt to know rather than talk about causes, we should look for intermediary causes in order to "pass from the first-noticed, external and remote cause of the phenomenon" to "the more immediate and direct cause" (*GP* 451). To Jaspers,

> psychic events "emerge" out of each other in a way which we understand….Thus we understand psychic reactions to experience, we understand the development of passion, the growth of an error, the content of delusion and dream; we understand the effects of suggestion, an abnormal personality in its own context or the inner necessities of someone's life. Finally, we understand how the patient sees himself and how this mode of self-understanding becomes a factor in his psychic development. [*GP* 302-3]

Rightfully, Jaspers acknowledges that psychiatry has to go beyond the experimentally reproducible causal connections explored by natural sciences, and search in addition to comprehend psychic phenomena by using empathic understanding which "always leads directly into the psychic connection itself" (*GP* 304).

Ghaemi emphasizes that in the therapeutic environment the goal of empathy is to help create a "safe place" where patients "can begin to find out the painful truth" about their deepest thoughts and fears and wishes (*OD* 117). This consideration unfolds the way empathy can contribute to improved existential understanding of possible causality. Empathy is meant to help clinicians understand the patient's choice for one of the multiple possibilities of becoming. The way the clinician applies it facilitates the patient to acknowledge, bear, and accept this choice.

Along all these considerations, I profoundly agree with Ghaemi; we just cannot highlight enough the importance of properly disentangling causality in depression and the role empathy would play in this process. By mistaking material and efficient causes, we are facing a fundamental error in understanding depression (*OD* 15), an error that may conduce to wrong treatment choices and make us ultimately fail in our professional mission.

Ghaemi's exploration of the causality of depression prompts us to meditate about how existential pain relates to depression and to one's fear about life, especially when depression is seen as the unresolved struggle for one's acceptable finality. Such continuum between healthy growth, neurosis, and depression is clearly underrepresented in the training of mental health professionals.

Existential Struggle and Its Influence on the Brain

Recent advances in neuroscience, as well as epidemiological and clinical data, all suggest the fact that "psychiatric diagnoses are not neatly compartmentalized entities with clearly defined boundaries."[8] The authors argue that neurobiology,

[6] Karl Jaspers, *Philosophy, Vol. 2*, transl. E. B. Ashton, Chicago and London: The University of Chicago Press 1970, p. 4. [Henceforth cited as *P2*]

[7] Karl Jaspers, *General Psychopathology, Vols. 1-2*, transl. J. Hoenig and Marian W. Hamilton, Baltimore, MD: The Johns Hopkins University Press 1997, pp. 301-13. [Henceforth cited as *GP*]

[8] David J. Kupfer, Emily A. Kuhl, William E. Narrow and Darrel A. Regier, "On the Road to DSM-V," *Cerebrum* (October 13, 2009). Last accessed 8-10-2017,

imaging, and genetics are expanding the scope of our understanding of psychiatric disease by providing more tangible, quantifiable information. As is known for some time in neuroscience, the existential struggle actually influences the brain's internal connectivity, its plasticity, and its development.[9] Existential struggle becomes one of possible efficient causes in the Aristotelian sense, and the therapeutic interventions, pharmaceutical or psychotherapeutic, may equally become efficient causes as well. When this struggle is related to depression, it can certainly become formative. Ghaemi supports this assumption by reflecting on the lessons passed on by the psychiatrist Viktor Frankl (*OD* 93-7). He embraces Frankl's argument that the only realistic way to understand a reaction is to truly understand its circumstances (*OD* 96). Since sufferance may be sometimes the only way of experiencing existence, our therapeutic endeavor should mainly focus on managing the changes that come along with suffering. Psychotherapy should help make the existential struggle bearable, but not annihilate the struggle. Ghaemi further observes that "this basic misery of human life is an existential, not medical fact" (*OD* 73). There is indeed merit to this interpretation. However, this basic misery may become the effective cause able to catalyze depression. When existential struggle comes with too much uncertainty, it may overwhelm one's ability to tolerate new insights, or to tolerate the pain inherent to an unfortunate becoming-process.

Functional neuroimaging studies have demonstrated that uncertainty and ambiguity may change the connectivity between regions of the brain that exert regulatory influences over emotional processes.[10] Such operations are experience-dependent over the course of development. The emotional salience of the environment affects the person's ability to learn about safety signals, thus impacting on individual's potential for becoming. It is possible that meaningful events, which become efficient causes in the Aristotelian sense, may influence an individual's potential for attaining happiness by interfering with the brain's response of "disambiguating the threat stimulus,"[11] and favor positive or negative valence calculations of emotional experiences. Inherently, this begs the question whether anxiety, along with depression, is existential or medical, as Ghaemi explores by addressing the notion of biological existentialism (*OD* 65). Ghaemi's position on this, once again, brings to mind Karl Jaspers' discourse on the coalescence of innate potentialities of the organism and of the psyche that are subsequently conditioned by all the events that occur thereafter. Jaspers defines the concept of Anlage as the "sum-total of all the endogenous preconditions of psychic life" (*GP* 455). Our constitution (*Anlage*) contains innate potentialities, which may be modeled by the environment in order to become manifest (*GP* 455-6). Essentially, one's life history as a whole may have the capacity continually to modify individual dispositions.

This perspective brings us to one of Ghaemi's book main provocations, namely the role of antidepressants in the becoming-process. The verdict is still out whether antidepressants attenuate the struggle, or increase the tolerance of the incertitude related to the struggle. Since the neurotic depression was removed from the diagnostic lexicon, the last "two decades of research" can hardly prove that "antidepressants have little efficacy in neurotic depression" (*OD* 73). Ghaemi examines Peter Kramer's stance on antidepressant therapy.[12] I agree with the author on Kramer's viewpoint that antidepressants may pave one's tolerance for the need of ontological becoming, and thus "provide new insights into life and free people up from inhibitions and limitations" (*OD* 35). Considering the previous existential perspective on depression, and the dynamic interdependence between the predisposition and the experiential struggle, antidepressants might simply allow someone who could be happy, and could better tolerate the incertitude of existential struggle, to

http://www.dana.org/Cerebrum/2009/Updating_the_Diagnostic_and_Statistical_Manual_of_Mental_Disorders/ [Henceforth cited as *OTR*]

[9] Norman Doidge, *The Brain that Changes Itself: Stories of Personal Triumph from the Frontiers of Brain Science*, New York, NY: Viking Penguin, 2007.

[10] Paul J. Whalen, F. Caroline Davis, Jonathan A. Oler, Hackjin Kim, M. Justin Kim, and Maital Neta, "Human Amygdala Responses to Facial Expression of Emotion," in *The Human Amygdala*, eds. Paul J. Whalen and Elizabeth A. Phelps, New York, NY: The Guilford Press 2009, pp. 265-288, here pp. 272-5.

[11] Tony W. Buchanan, Daniel Tranel, and Ralph Adolphs, "The Human Amygdala in Social Function," in *The Human Amygdala*, eds. Paul J. Whalen and Elizabeth A. Phelps, New York, NY: The Guilford Press 2009, pp. 289-318, here p. 290.

[12] Peter D. Kramer, *Listening to Prozac: A Psychiatrist Explores Antidepressant Drugs and the Remaking of the Self*, New York, NY: Viking Penguin, 1993.

become so. Ghaemi states eloquently, "a life without despair would be a life without hope, for hope cannot exist except as an antidote to despair" (*OD* 172). Despair may be the "emotional source of spirituality" (*OD* 148), but it may sometimes "block one from seeing any exit" (*OD* 165). When the response to despair fails to reach acceptance of the self, antidepressants may interact with the Anlage, and facilitate the first cause move towards an alternative possible becoming. Since the Major Depression diagnostic category disappoints, for both clinical and philosophical obvious reasons, we just need to create a new dimensional diagnostic system, and seek convincing evidence for the success or failure of the antidepressants.