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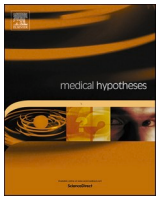

Short Communication

Depression as reversible biopsychosocial break up after schismogenesis

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ABSTRACT

In recent decades, an important amount of biomedical and psychosocial data regarding major depression etiology and treatment has emerged. Despite this wealth of information, a global hypothesis seeking to integrate all available data into a unified explanatory model of depression is still missing. The biopsychosocial model is a precedent in this endeavor. This model proposes a systemic conceptual framework, suggesting that the dynamic interactions of biological, psychological, and social factors constantly condition individuals' health status. However, it has been criticized for its lack of practical utility when it comes to understanding the onset and potential reversion of depression. Gregory Bateson proposed a systemic, transdisciplinary construct that may be useful for addressing these criticalities: schismogenesis. While this concept has found increasing application across different areas such as politics, ecology, or even psychotherapy, to the best of our knowledge, its use as a biopsychosocial etiology hypothesis for depression remains unexplored. The schismogenesis hypothesis suggests that depression can be considered as a switching process mediated by positive feedback curves, leading to a functional dissociation due to insufficient inhibitory control. Nonetheless, this process can be either reversed spontaneously or through external help, resulting in a rebalancing of the system.

Introduction

Depression usually arises from the interplay of genetic vulnerability and stressful life experiences [1,2]. Beyond genetic predisposition, biological stressors (immunological, hormonal, etc.) can also play a role in increasing depression risk [1]. Moreover, the likelihood of experiencing depression increases not only in stressful experiences, but also in circumstances where the biopsychosocial balance can be more easily disrupted [3,4]. In fact, evidence suggests that the increased vulnerability to depression in developed countries may be related to increasing unhealthy lifestyle factors such as sedentarism, bad diets, loss of social support network, excessive work demands, sleep deprivation, etc. [2,5].

This vulnerability-stress is accepted as an empirically proven model in the depression's etiology. In a simplified way, it proposes that the greater the vulnerability to depression, the less stress is required to trigger the disorder. However, this model does not provide an explanatory mechanism for how depression is developed.

The biopsychosocial model was proposed by Engel in 1997 as a response to the limitation of the traditional medical model and its biological reductionism [6]. This model assumes that mind, brain, and social network are interdependent complex systems. The optimal function of these systems occurs when there is balance between connection and differentiation among its parts, facilitating the flow of energy and information without blockages [7]. Recent research has suggested that mental disorders, including depression, may be explained by persistent overactivation of certain domains of the mind and brain, leading to a rigid and intrusive emergence of cognition, emotions, and behaviors. Conversely, other "negative" symptoms may correlate with associated compensatory underactivated domains [8–11]. This phenomenon entails a biopsychosocial blockage as well as a functional brain, mental and social disintegration in individuals suffering from depression.

Furthermore, when it comes to depression, the biopsychosocial model suggests that negative emotionality, negative beliefs, and social isolation feed one another, worsening the patients' condition. However, again, it is unfortunate that there are very few comprehensive and commonly accepted explanations regarding how individuals reach such state and how they can overcome it [2,7]. Therefore, we believe that the transdisciplinary systemic construct of schismogenesis, introduced by Bateson, may be useful to update and improve the biopsychosocial hypothesis on depression already proposed [7,8,12].

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Schismogenesis hypothesis about depression

Schismogenesis appears when the stimulating interactions arising in a biological or psychosocial system disproportionately exceed inhibitory control. This results in positive feedback curves that cannot be adequately modulated by the system itself [8,13–15]. When this happens, the psychosocial and brain system is functionally fractured into two different domains. On one hand, one domain is overactivated and revolted against the rest of the system and its regulatory inputs; on the other hand, the other domain is compensatorily underactivated in an effort to prevent the progress of the revolted domain [8,13–15].

Patients with depression frequently perceive a sense of being fragmented and disconnected from the world and from themselves [16]. Besides, they often use the divided mind metaphor (one part is healthy and the other one is sick) to explain their experience; referring to the depressive part as a source of unavoidable suffering [17]. Similarly, recent research on depression points out four essential elements that may explain depressive patients' narratives about their emotional and physical experiences: patients feel overwhelmed by negative emotions, unable to experience positive emotions, trapped in a forceless and painful body, and disconnected from their mind, body and the world around [18]. These subjective experiences may be related and seem to be coherent with the blockage and functional disintegration that underly the schismogenesis process. The schismogenesis hypothesis could also explain the inhibitory sequelae of depression ("negative" symptoms) as the functional insufficiency of rigidly hypoactivated areas [15,19]. Such "negative" symptoms related to the inhibitory sequelae of depression could include cognitive impairment, a limitation in the ability to experience positive emotions, and a limitation in affiliation behaviors that are usually associated with social withdrawal [8,15].

In order to improve the theoretical and practical usefulness of biopsychosocial models, new perspectives have been suggested [6]. For instance, from the evolutionary point of view, natural selection promotes efforts that aim at adapting and surviving, but not at psychological well-being. Thus, depression could be adaptive despite the suffering and disability burden that entails. However, how can depression counterbalance the apparent disadvantages in survival and reproduction that it entails? Possibly, by easing the disconnection from potential life-threatening situations [8]. This is in line with the schismogenesis hypothesis: if the individual is put under more stress and tension than it can bare, it crashes and disconnects to prevent irreversible damage.

Discussion

The schismogenesis biopsychosocial hypothesis about depression entails some empirical influences. Regarding the etiology of schismogenesis, this hypothesis refuses the existence of a specific, sufficient, or necessary causal factor shared by all cases of depression. Instead, it supports that there are multiple factors capable of unbalancing the stress-vulnerability relationship, whether they are predisposing, precipitating, or maintainers of the disorder. The challenge is to understand which of these factors are more relevant in each patient and address them to reverse the blockage and disintegration.

Talking about pathogenesis, this hypothesis discards the possibility of explaining the development of depression through linear causality. Instead, it suggests a circular causality mechanism that integrates all factors involved in depression onset and maintenance (Fig. 1).

Regarding the clinical implications of this hypothesis, it should be highlighted that brain, psychological, and social systems instantly influence each other reciprocally. Any dynamic dysfunction, whether it starts in the brain, the mind, or the social network will have an impact on the functioning of the other systems and can significantly differ from person to person [7]. Thus, a great interindividual variability in depression symptomatic expression should be expected.

The schismogenesis hypothesis suggests that looking for biological and psychosocial blockage and functional disintegration related markers may help in validating a depression diagnosis and monitoring its evolution. For example, remarkably interesting progress is being made in the study of the functional brain dysconnectivity in depression [11].

Depression prognosis depends on bearable stress levels of the system and vulnerability maintenance factors, but not necessarily on precipitating factors. In addition, each evolution is unpredictable and discontinuous. The resulting brain and psychosocial disintegration lead to a loss of the ability to interact and adapt to the environment. This loss can progressively consolidate over time on a functional and structural level [4]. Thus, prolonged depressive episodes lead to a worse prognosis, require more recovery time after the episode, and increase the risk of new episodes in the future.

The therapeutic approach should be multimodal and based on any strategy that contributes to unlocking and reintegrating the system. While, at same time, adjusted to the patient's characteristics and preferences. The faster and more multicomponent the treatment, the more effective it will be.

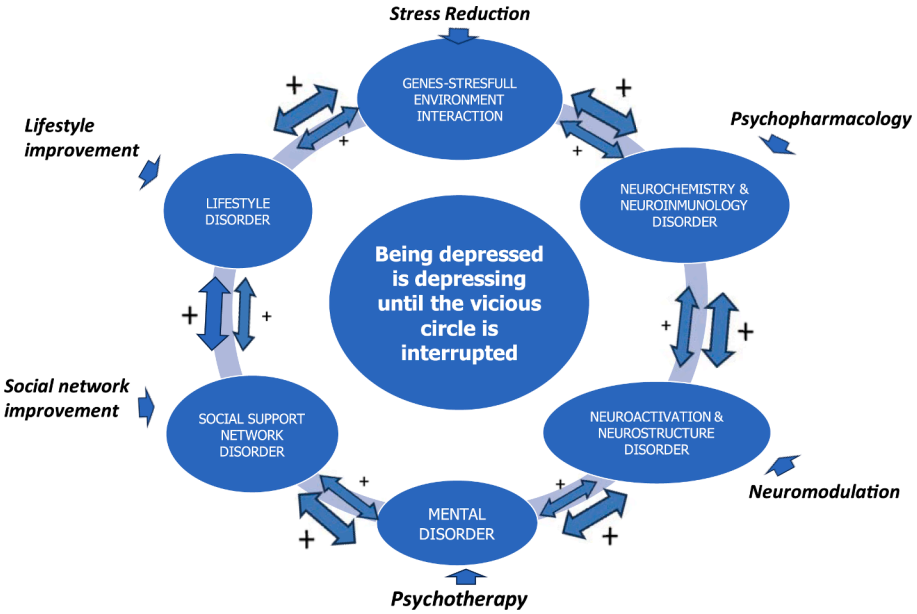

Fig. 1. Basic diagram of depression as reversible biopsychosocial schismogenesis.

The first therapeutic objective should therefore be the reduction of brain and psychosocial stress levels. The second should be to decrease vulnerability, restoring the balance between stimulatory and inhibitory interactions to enable the reintegration of the system. However, how could this be done in practice? By global or selective strategies.

Global strategies are based on the generation of enough global activation and/or inhibition in the system. They aim to reset the dynamic dysfunction and thus facilitate the restart of the system in a more integrated way. Examples of this strategy would be the use of electroconvulsive therapy, esketamine, and other psychedelics [20].

Selective strategies are based on the production of enough activation (in location and intensity) in the rigidly underactivated domains, or inhibition in the rigidly overactivated domains [8,13–15]. In previous articles, we suggested in detail how each antidepressant treatment selectively works [8,14,15]. Some examples are: the mindfulness-based cognitive therapy, which may be considered as a very selective disruption of the dynamic dysfunction, but scarce in intensity [21]; the neuromodulation via Transcranial Magnetic Stimulation, which would be a less selective but more intense approach [15]; and lifestyle modification interventions, which would have comparatively intermediate selectivity and intensity [2,21].

It should be remarked that the biopsychosocial perspective assumes that any therapy directed to the biological level will simultaneously have an impact on the psychological and social levels and vice versa [7,22]. Any experience that promotes a more flexible and adaptive flow of information in the brain and psychosocial network will help to improve mental health [7]. For example, on a social level, integration based on significant relationships contributes in a positive way to emotional regulation, while social relationship loss tends to generate stress and social isolation. According to this, it has been proposed that the greater the social support network, the lower the risk of depression [23].

As we have already suggested, depression could be seen as a schismogenesis in the biopsychosocial network that worsens over time. To stop and reverse schismogenesis dynamics and improve the prognosis of the disorder, therapeutic changes should be implemented as quickly as possible (Fig. 1). Explaining to patients that depression could be considered a protective functional state of the mind and brain, but that acting upon it should be done rapidly, may help them to better adhere to treatments.

Conclusion

The schismogenesis hypothesis proposes that depression and other mental disorders appear when self-reinforcing feedback curves in the brain, mind and social network cannot be modulated [13]. This generates a functional dissociation (disintegration) with two rigidly opposed domains, one of them overactivated, and the other one compensatory underactivated. Depression is therefore better defined as the difficulty of getting out of a negative emotional state, than by the presence of the emotional state itself [19]. However, it is important to recognize that this hypothesis has the disadvantage of being based on systemic and complexity approaches that are generally unfamiliar to mental health professionals, so its understanding may not be easy [24].

The schismogenesis hypothesis suggests that the faster and multicomponent the antidepressant treatment the more effective it will be, but this is far from proven [25]. Future research may help to validate or discard this and other aspects of this hypothesis.

Authorship contribution statement

RGJ, MGT, and LRE conducted the literature searches and analyses. RGJ, MGT, and LRE wrote the first draft of the manuscript. All authors contributed to and approved the final manuscript.

Consent statement/Ethical approval

Not required.

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CRediT authorship contribution statement

Rocío Gómez-Juanes: Writing – review & editing, Writing – original dra, ft, Conceptualization. **Lorenzo Roldán-Espínola:** Writing – review & editing, Writing – original draft, Conceptualization. **Mauro García-Toro:** Writing – review & editing, Writing – original draft, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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