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Stress as a model for neurobiology of depression

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Depression is a common and global mental health disorder, currently ranked as the leading cause of disability worldwide, and at worst, can lead to suicide [1]. A research approach that describes reliable neurobiological findings on Depression based on psychopathological syndrome will be more solid contrasted to a nonetiological system of classification.

The relationship between stress and illness is a strong example of a field of study that can be more fully understood from an integrative perspective. Throughout the literature on Depression, the condition has been consistently associated with the dysregulation of the stress response system, which is comprised of the endocrine, nervous, and immune systems, and is activated upon detection of physical or emotional stressors [2]. It is now broadly accepted that psychological stress may change the internal homeostatic state of an individual. During acute stress, adaptive physiological responses occur, whenever there is an acute interruption of this balance, an illness may result. Of particular interest are the psychological stress (stress in mind) and the interactions of the nervous, endocrine, and immune systems [3,4].

The social and physical environments have an enormous impact on our physiology and behavior, and they influence the process of adaptation or "allostasis" [5]. It is correct to state that at the same time that our experiences change our brain and thoughts, namely, changing our mind, we are changing our neurobiology.

Chronic stress has been accepted as a significant risk factor for the pathogenesis and exacerbation of numerous physical and psychiatric diseases [6-7]. It is widely accepted that chronic stress may cause an allostatic shift in the diurnal rhythm of cortisol and stress-induced cortisol levels, increasing the sensitivity of the Hypothalamic-Pituitary-Adrenal (HPA) axis [4-5]. More specifically, it is postulated that chronic stress may cause dysregulation of the receptors (GRs and/or MRs), directly resulting in increased vulnerability to physical and psychiatric diseases, such as Depression [4,6,8].

The concept of early life stress (ELS) encapsulates all traumatic experiences occurring before the age of 18, or a woman's first menstrual cycle [9]. In the sum of the literature as mentioned earlier, both ELS and Depression are associated with abnormalities within the HPA axis. However, the abnormalities observed in response to ELS may increase susceptibility to adulthood depression [4,6].

It is widely accepted that the heterogeneity of Depression hinders the progress and in both further treatments and research, and therefore, we may need to take a more comprehensive approach to identify predictive markers. For this to happen, we would need to explore predictive patterns of biological markers from multiple areas such as HPA axis, inflammatory markers, neurodevelopmental markers. Moreover, in addition to accounting for other psychological, social and economic risk factors [10]; although it should be stressed that not all individuals whom experience ELS will go on to develop Depression in adulthood, and therefore we need also to identify bio-psycho-social protective factors that could lead to resilience and be used in early intervention strategies.

We suggest a future focus on clinical characteristics, especially neurogenerative symptoms, might provide a deeper understanding of the biological mechanisms involved. The integrated analysis of psychopathology, ELS and neuroendocrine function may provide useful indicators to improve diagnosis and treatment outcome.

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