THE STRESS-DIATHESIS MODEL AND SUICIDAL BEHAVIOR

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Abstract: *Suicide is a complex behavior, a multi-causal phenomenon with a large number of risk factors which can be categorized in explanatory models and facilitate the assessment of suicide risk. Suicide is an important subject for its worldwide prevalence, and the severity for its social impact. The stress-diathesis model supports that suicide is the result of an interaction between environmental stressors and a trait-like diathesis or susceptibility to suicidal behavior. A stress-diathesis model was proposed based on the integration of the neurobiology and the psychopathology which still forms the basis for much of the current research in suicidology. The application of stress-diathesis models to suicidal behaviour has substantial implications for the identification of suicide risk and the prevention of suicidal behavior. The identification of trait-dependent vulnerability factors can be expected to facilitate early recognition of suicide risk*.
Key-words: *suicide, stress, vulnerability factors*

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Suicide is a behavioral outcome with a large number of contributing factors. Research has focused on a wide array of neurobiological and psychological topics in an attempt to better understand the pathophysiology of suicide. Neurobiological inquiries have included neurotransmitter analyses, genetic studies, neuroendocrine studies, biological markers, and imaging studies (1).

The relationship on serotonin to suicidality has been most widely studied (1). An association between reduced serotoninergic activity, as indicated by lower levels of cerebrospinal fluid (CSF) 5-hydroxyindoleacetic acid (5-HIAA), and serotonin dysfunction and suicidality has emerged since the 1970s (2), (3), (5). (30). Changes in norepinephrine transmission in suicide have also been investigated, but although there may be some association between a decreased number of noradrenergic neurons and suicidality, (16) overall, CSF studies have shown no significant difference in norepinephrine metabolites in those with suicidal behavior (3), (18). Data regarding the role of dopamine in suicide are inconclusive. (3), (15), (18), (19). Heightened hypothalamic-pituitary-adrenal (HPA) axis activity has been implicated in the pathophysiology of suicide, although not all studies of the relationship between the HPA axis abnormalities and suicidal behavior have reached the same conclusions (1), (2), (3), (6), (15).

Suicide can be conceptualized as anger turned on oneself or anger toward others directed at the self (20). Suicide has also been seen as being motivated by three driving forces: the wish to die, the wish to kill, and the wish to be killed (20). Deficits in ego function have also been postulated to predispose to suicide, (19) as have poor object relations (20). Hopelessness is a central psychological correlate of suicide, and extensive study on it has suggested that hopelessness may be the best overall predictor of suicide (3). Shame, worthlessness, poor self-esteem, early traumatic relationships, and intense psychological pain are also key concepts in the understanding of suicide (20), (21). In addition, poor coping skills, antisocial traits, hostility, dependency or over-dependency, self- consciousness, and high intropunitiveness have also been associated with suicide (20), (22). Finally, research has postulated correlations between observed neuroanatomical,

neurotransmitter, and neuroendocrine findings in suicide and attendant cognitive traits of loser status, no escape, and no rescue as central to understanding suicidal behavior (6).

Early descriptions of the roles of stress and a diathesis in the development of suicidal behavior were grounded in sociobiology (23). The term "stress" was first used in the 1930s by the endocrinologist Hans Selye to describe responses of laboratory animals to various stimuli. Originally Selye meant "stress" to be the response of an organism to a perceived threat or "stressor", but the term is now used to mean the stimulus rather than the response.

The diathesis concept has a long history in medical terminology. The word diathesis stems from the Greek idea of predisposition, which is related to the humoral theory of temperament and disease (24). The term has been used in a psychiatric context since the 1800s. Theories of schizophrenia brought the stress and diathesis concepts together and the particular terminology of diathesis–stress interaction was developed by Meehl, Bleuler, and Rosenthal in the 1960s (25).

In the modern sense, the biological traits produced by the genetic disposition are the diathesis. The term "diathesis'' has, however, been broadened to include cognitive and social predispositions that may make a person vulnerable to a disorder such as depression. In this broader sense, the diathesis is the necessary antecedent condition for the development of a disorder or problem, whether biological or psychological. The "cry of pain" model is a clear example of such a psychological approach to the study of the diathesis to suicidal behavior. In most models, whether biological or psychological, the diathesis alone is not sufficient to produce the disorder but requires other potentiating or releasing factors to become pathogenic. The diathesis, in this case, includes the vulnerability to stress (24).

Rubinstein (1986) developed a stress-diathesis theory of suicide, in which the effects of specific situational stressors and the categories or predisposing factors of vulnerable individuals in a given culture were integrated in a biocultural model of suicidal behavior. Mann and Arango (1992) then proposed a stress-diathesis model based on the integration of the neurobiology and psychopathology, which still forms the basis for much of the current research in suicidology.

Psychosocial crisis and psychiatric disorders may constitute the stress component of stress-diathesis model and it is difficult to separate the impact of psychosocial adversity from that of psychiatric illness (26).

Early-life adversity and epigenetic mechanisms might explain some of the link between suicide risk and brain circuitry and neurochemistry abnormalities (27). Clinical studies have shown that reported childhood adversity such as deprivation and physical or sexual abuse, is a risk factor for psychopathological phenomena in later childhood and adulthood. Also, there are findings from studies using post-mortem and in-vivo techniques that show impairment of the serotonin neurotransmitter system and the HPA axis stress response system in the diathesis for suicidal behavior.

Neuroanatomical, physiological and genomic alterations may contribute to the long-lasting detrimental effects of exposure to childhood adversity on the risk of psychopathology. (28). Postmortem and neuroimaging studies have clearly demonstrated structural and functional changes in the brains of individuals with a history of suicidal behavior, which may correlate with components of the diathesis. Postmortem findings include fewer cortical serotonin neurons in key brain regions such as the dorsal and ventral prefrontal cortex, which also appear to correlate with components of the diathesis (29). These components may include aggression and/or impulsivity, pessimism and hopelessness, and problem-solving or cognitive rigidity (30), (31), (32). Increasing evidence points at a role of increasing neuropsychological deficits in the medial temporal cortex-hippocampal system, perhaps due to the detrimental effects of stress hormones on serotoninergic

neurons. Studies of levels of the serotonin metabolite 5-HIAA in the cerebrospinal fluid of suicide attempters have shown that depressed suicide attempters have lower levels than depressed non-attempters, repeating attempters have lower levels than so-called first-evers, the use of violent methods is associated with lower levels than the use of non-violent methods, and attempted suicide patients with lower levels show a poorer survival in terms of death from suicide (33).

Cognitive stress diathesis model of suicidal behavior. Williams and Pollock (2001) have described a diathesis for suicidal behavior in cognitive psychological terms, that is the, is the "cry of pain" model, which was elaborated in the "differential activation model". According to this model, suicidal behavior represents the response to a situation that has three components: sensitivity to signals of defeat, perceived "no escape": limited problem-solving abilities may indicate to persons that there is no escape from problems or life events, perceived "no rescue". The occurrence of suicidal behavior is associated with a limited fluency in coming up with positive events that might happen in the future. This limited fluency is reflected not only by the perception that there is no escape from an aversive situation but also by the judgement that no rescue is possible in the future. The fluency of generating positive future events correlates negatively with levels of hopelessness, a core clinical predictor of suicidal behavior. This suggests that hopelessness does not consist of the anticipation of an excess of negative events, but indicates that hopelessness reflects the failure to generate sufficient rescue factors.

Mann et al proposed a stress-diathesis model based on the findings from a clinical study of a large sample of patients admitted to a university psychiatric hospital. When compared to patients without a history of suicide attempts, patients who had attempted suicide show higher scores on subjective depression and suicidal ideation, and reported fewer reasons for living. In addition, suicide attempters show higher rates of lifetime aggression and impulsivity, comorbid borderline personality disorder, substance use disorder or alcoholism, family history of suicidal acts, head injury, smoking, and childhood abuse history. The risk for suicidal acts thus is determined not only by a psychiatric illness (the stressor) but also by a diathesis as reflected by tendencies to experience more suicidal ideation and to be more impulsive and, therefore, more likely to act on suicidal feelings. It describes a predisposition to suicidal acts that appears to be part of a more fundamental predisposition to both externally and self-directed aggression. Aggression, impulsivity, and borderline personality disorder are the key characteristics, which may be the result of genetic factors or early life experiences, including a history of physical or sexual abuse. A common underlying genetic or familial factor may therefore explain the association between suicidal behavior with the aggression/impulsivity factor and/or borderline personality disorder, independent of transmission of major depression or psychosis. The serotonin neurotransmission system may also play a role. Given the evidence linking low serotoninergic activity to suicidal behavior, it is conceivable that such low activity mediates genetic and developmental effects on suicide, aggression, and alcoholism (34).

Neurobiological model of suicidal behavior. Jollant et al. (2008) provide an example, using a functional neuroimaging technique, that is neurobiological approach to stress-diathesis models of suicidal behavior. This model was investigated by exposing young males with a history of depression to angry, happy, and neutral faces while being euthymic. Findings in young males with a history of attempted suicide were compared to those in young males without such a history. Relative to affective comparison subjects, suicide attempters showed greater activity in the right lateral orbitofrontal cortex (Brodmann area 47), and decreased activity in the right superior frontal gyrus (area 6), in response to prototypical angry versus neutral faces, and greater activity in the right cerebellum to mild angry versus neutral faces. Suicide attempters were distinguished from

non-suicidal patients by responses to angry and happy faces that may suggest increased sensitivity to others' disapproval, higher propensity to act on negative emotions, and reduced attention to mildly positive stimuli. It is concluded that these patterns of neural activity and cognitive processes may represent vulnerability markers of suicidal behavior in men with a history of depression.

Studies in the domains of neuropsychology, cognitive psychology, neurobiology and clinical psychiatry have provided increasing evidence in support of a stress –diathesis model of suicidal behavior.

The application of stress-diathesis models to suicidal behavior has substantial implications for the identification of suicide risk and the prevention of suicidal behavior. The identification of trait-dependent vulnerability factors can be expected to facilitate early recognition of suicide risk. Vulnerability traits are open to modification early in life, and interventions during sensitive periods of development may have durable effects on personality and thereby affect vulnerability to suicide (35). The interdependence of stress and diathesis components would however also mean that interventions targeting the diathesis may also decrease exposure to stressors and suggest that relief of stress effects would enhance the efficacy of therapeutic interventions.

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