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Constitutional and Biological Predictors of the Risk of Suicidal Behavior in Mental Disorders

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ABSTRACT

The prevention of suicidal behavior is an extremely important issue in modern psychiatry and is of high social importance due to high prevalence of this phenomenon. Despite the availability of a number of psychometric scales for assessing suicide risk, their use may be limited, since due to the mental state of patients, it is not always possible to assess the risk of suicide.

Thus, the search for potential structural and peripheral biomarkers of a suicidal behavior risk is a pressing issue in psychiatry. Existing studies are usually limited to searching for one or several markers or factors and do not take into account the integrity of the human body with its inherent complementarity of both pathogenic and sanogenic factors, including social and environmental, compensatory mechanisms, adaptation threshold, and reversible and irreversible decompensation.

To date, there is no single point of view that fully explains the genesis of suicidal behavior, and the potential biological factors vary greatly depending on the methods used. Based on data collected from recent studies examining a variety of biological markers associated with suicide, it can be confirmed that suicidal behavior in individuals with mental disorders is a complex, multifactorial, and polygenic mental state.

Keywords: suicidal risk, suicide, parasuicide, predictors, risk factors, mental disorders

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Конституционально-биологические предикторы риска суицидального поведения при психических расстройствах

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РЕЗЮМЕ

Профилактика суицидального поведения является чрезвычайно важным вопросом современной психиатрии и имеет высокую социальную значимость в связи с большой распространенностью данного феномена.

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Несмотря на наличие ряда психометрических шкал для оценки суицидального риска, их применение может быть ограничено, поскольку в силу психического состояния пациентов далеко не всегда удается провести оценку риска самоубийства.

Таким образом, поиск потенциальных структурных и периферических биомаркеров риска суицидального поведения является актуальным направлением в психиатрии. Имеющиеся исследования, как правило, ограничены поисками одного или нескольких маркеров или факторов и не учитывают целостности человеческого организма со свойственными ей комплементарностью как патогенных, так и саногенных факторов, включая социально-средовые, компенсаторными механизмами, порогом адаптации, обратимой и необратимой декомпенсацией.

На сегодняшний день не существует единой точки зрения, полностью объясняющей генез суицидального поведения, а потенциальные биологические факторы сильно различаются в зависимости от используемых методов. На основе данных, собранных в ходе недавних исследований, изучающих разнообразные биологические маркеры, связанные с суицидом, можно подтвердить, что суицидальное поведение у лиц с психическими расстройствами является сложным, многофакторным и полигенным психическим состоянием.

Ключевые слова: суицидальный риск, суицид, парасуициды, предикторы, факторы риска, психические расстройства

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INTRODUCTION

Suicidal behavior remains one of the most pressing issues in clinical psychiatry due to its high prevalence among patients [1, 2]. According to numerous studies, approximately 90% of individuals who have ever attempted suicide have a mental disorder [2]. The risk of suicide is 20 times higher in patients with depressive and bipolar disorder and 10–13 times higher in patients with schizophrenia compared to the general population [2, 3].

Suicidal behavior is a complex and multifactorial process encompassing various forms and manifestations of mental activity aimed at taking one's own life intentionally. The study of suicidal phenotypes within psychopathology is crucial for elucidating the nature of risk factors, predicting, and preventing suicide.

Previously, suicide risk factors in patients with mental disorders were proposed, including previous suicide attempts, early age of disease onset, substance abuse, pronounced delusions and hallucinations, severe neurocognitive deficits, patient's illness perception, etc. [4–7]. However, predicting suicidal behavior based on these factors remains unreliable, since demographic factors are not universal, and clinical factors are subject to change, depending on both the course of the pathological process itself and the implemented treatment and rehabilitation measures.

In this regard, the study of the constitutional and morphological type (morphological phenotype, morphophenotype, constitutional type) of patients, which is a structural biomarker of reactivity that is not subject to significant changes in adults, is a promising direction in the search of predictors of the suicidal behavior risk. The most comprehensive studies in this area have been conducted on a schizophrenia model. Based on the results of these studies, N.A. Kornetov [8–10] formulated an anthropological paradigm that showed the role of constitution in the course and outcome of the

disease, opening up possibilities for predicting pathokinesis.

The pathogenesis of many mental disorders is believed to begin long before the onset of the main symptoms, during critical periods of brain development [11]. Adverse conditions during prenatal development can lead to changes in the brain structures responsible for perception and emotional regulation. In addition, abnormalities in the development of the nervous system, including dysfunctions of the neurotransmitter systems (dopaminergic, serotonergic, glutamatergic, etc.), have been associated with both the risk of developing many mental disorders and suicidal behavior [11, 12]. It is assumed that peripheral biomarkers characterizing brain neuroplasticity, damage to neuronal structures and nervous tissue, neurotoxicity, immune inflammation, breakdown of monoamines and catecholamines, and carriage of certain polymorphisms of the genes encoding these biomarkers also contribute to the genesis of suicidal behavior to some extent [13, 14]. Thus, ignoring the biological mechanisms underlying suicidal behavior in patients with mental disorders may decrease the quality of this prognosis.

In light of the above, we have summarized and systematized data on the constitutional and biological factors involved in the genesis of suicidal behavior in individuals with mental disorders. The data presented will form the basis for the development of predictive models of suicide risk, which will ultimately contribute to reducing the burden of suicide among patients.

We classified constitutional and biological predictors of the risk of suicidal behavior as constitutional and morphological, structural and functional, genetic, and molecular.

THE ROLE OF CONSTITUTIONAL AND MORPHOLOGICAL PREDICTORS IN THE SUICIDAL BEHAVIOR GENESIS

The theories of body types proposed by E. Kretschmer [15] and W.H. Sheldon [16] are historical concepts that attempted to correlate somatotypes and mental disorders. Accordingly, E. Kretschmer distinguished four constitutional and morphological types of people: 1) the asthenic type, which has a slender build and is more prone to developing schizophrenia; 2) the pyknic type, which has a round and soft physical build and is probably more prone to bipolar disorder; 3) the athletic type which is more prone to epilepsy; and 4) the dysplastic type, which cannot be classified as any of the other three types [15].

W.H. Sheldon had similar criteria, classifying body types into ectomorphic (asthenic type), endomorphic (pyknic type), and mesomorphic (athletic type). He also believed that there is a deeper, genetically determined association between the somatotype and personality traits [16]. However, these classifications were based only on observational studies at the beginning of the XX century.

Later, the concept of anthropometry and the role of constitution in the development and course of mental disorders was continued by Russian authors [8, 17, 18]. The accumulated research and theoretical experience has proven the feasibility of identifying the main constitutional and morphological types in the clinical analysis of somatic-symptom and mental disorders [8]. With regard to the role of anthropometric characteristics of suicidal individuals, it was established that the asthenic type is associated with unfavorable clinical dynamics of schizophrenia and suicidal behavior [17].

The predominance of the asthenic body type in patients with pronounced hypochondriacal symptoms also supports the constitutional and morphological predisposition of suicidal behavior in schizophrenia [18]. In the study by A.A. Zalivin [19], the role of constitutional features of suicidal individuals with mental disorders in the post-suicide period was established.

The analysis of the distribution of somatic sexual differentiation of suicidal individuals by cohorts of the post-suicide period showed that suicide ideation in the post-suicide period was associated with the dysplastic body type. The remaining cohorts were primarily characterized by the normosthenic body type, secondarily – by the hypersthenic body type in the critical period, and by the hypersthenic and asthenic body types in the manipulative period.

A number of studies on individual anthropometric parameters have also been conducted in this context. E. Laakso et al. [20] examined excess weight as a risk factor for suicidal behavior. The authors found

that girls with suicide attempts were more likely to be overweight and to have affective and eating disorders and anxiety. A similar conclusion was reached by M.Z. Zhang et al. [21] – overweight and obesity were associated with an increased risk of suicidal ideation (for overweight, odds ratio (OR) = 1.10; 95% confidence interval (95% CI) 1.01–1.20; for obesity: OR = 1.17; 95% CI: 1.01–1.35) and suicide attempts (for overweight: OR = 1.12; 95% CI = 1.02–1.23; for obesity: OR = 1.12; 95% CI: 1.00–1.25). Subgroup analysis showed that the associations between overweight/obesity and suicide attempts were significant only for girls.

The study including an older age group with adjustments for covariates revealed that overweight and obese young adults (19–44 years) (OR = 1.18, $p < 0.01$), underweight and thin middle-aged adults (45–64 years) (OR = 1.32, $p < 0.05$), and obese elderly people (65 years and older) (OR = 1.19, $p < 0.05$) were more likely to have suicidal ideation compared to age-matched healthy-weight individuals [22].

The results of this study show that the association between body weight and suicidal ideation varies by age group. An increase in the body roundness index (BRI), proposed by D.M. Thomas et al. [23], led to an increased likelihood of suicidal ideation; individuals with the highest BRI had suicidal ideation 1.52 times more often, regardless of sociodemographic features [24].

In an analytical review, J. Zhang et al. [25] indicated an inverse relationship between body mass index (BMI) and the risk of completed suicide, regardless of the region of residence and gender of the study participants. In general, among men, high BMI was associated with a low risk of suicidal behavior, whereas among women, high BMI was associated with an increased risk of unsuccessful suicide attempts.

Therefore, the discrepancies in the results obtained from studies of individual anthropometric parameters indicate the need for a comprehensive approach to identifying structural biomarkers of suicidal behavior, especially since body weight fluctuates over a lifetime both up and down. The use of integrated body type parameters based on skeletal muscle measurements (constitutional and morphological type and somatic sexual differentiation) is a more promising and reliable tool for assessing the risk of suicidal behavior, as they are relatively stable during ontogenesis and are genetically determined.

STRUCTURAL AND FUNCTIONAL CHANGES IN THE BRAIN IN PATIENTS WITH SUICIDAL BEHAVIOR

The study of neurophysiological changes underlying suicidal behavior has some limitations due to difficulties in analyzing brain structure and function. Nevertheless, such studies are widely represented in the literature. Most neuroimaging studies in suicidal individuals focus on the prefrontal cortex. This region is involved in stress response, executive functions, and psychomotor skills [26].

Studies of the prefrontal cortex in patients with suicide attempts have shown changes in activation patterns, leading to social maladjustment and impaired decision-making related to reward [3, 27]. Structural magnetic resonance imaging (MRI) also clearly demonstrates a decrease in the volume (thickness) of gray matter in the ventromedial prefrontal cortex in patients with suicide attempts compared to healthy controls [3, 28, 29], which confirms the role of prefrontal cortex dysfunction in the genesis of suicidal behavior. In addition, multimodal neuroimaging studies combining structural and functional imaging methods (MRI and positron emission tomography (PET)) showed a significantly smaller volume of gray matter in the cerebellum, right orbitofrontal cortex, and hippocampus in young patients with bipolar disorder with suicide attempts compared to similar patients without suicide attempts. What is more, in the uncinate fasciculus and ventral and right cerebellar areas, a decrease in white matter integrity and a decline in functional connectivity between the amygdala, right rostral prefrontal cortex, and left ventral prefrontal area were observed [30].

Impaired functioning of neural networks responsible for behavior and emotion regulation may be associated with alterations in thalamocortical pathways, potentially increasing the risk of suicidal behavior in patients [31]. The analysis of brain activity patterns using electroencephalography (EEG) indicated associations between the degree

of suicidal intent and decreased cortical function in patients with depression [32, 33].

Similar results were obtained in one of our studies on patients with affective disorders, where lower alpha and theta power and pronounced interhemispheric asymmetry with a predominance of the right hemisphere were found in patients with a history of suicide attempts [34]. An increased risk of suicide in patients with depression may be associated with relatively low mean (0.5–5 Hz) EEG coherence in the frontal and occipital regions, as well as a decrease in the amplitude of changes in the mean coherence (0.5–45 Hz) in the prefrontal cortex in response to emotional stress [35, 36]. These data also indicate a decline in brain resources in suicidal individuals and are consistent with the results of evoked potential tests [37, 38].

In relation to patients with schizophrenia, who also have a relatively high risk of suicidal behavior, we previously found lower baseline beta power and a weak activation response (Berger effect) in those with a history of suicide attempts [39]. These parameters were significant factors in a model for predicting suicide attempts in patients with schizophrenia. Lower amplitude values and increased latency of evoked potentials were also observed in patients with schizophrenia and suicide attempts [40].

Thus, the results of the studies reviewed above suggest that patients at high risk for suicidal behavior exhibit reduced activity in brain structures, which may be responsible for reduced adaptive responses to stress. However, the structural and functional characteristics identified in suicidal individuals do not serve as screening tools and merely complement the clinician's assessment of a suicide risk.

GENETIC PREDICTORS OF THE SUICIDE RISK

The constitutional and morphological as well as structural and functional factors discussed above are determined by the genotype. In terms of the biological basis of suicidal behavior, there is currently a growing body of research examining the role of genetic factors. These studies demonstrate that suicidal behavior is determined both by genetic and hereditary factors, with the heritability of suicidal behavior accounting for approximately 43% [41]. Furthermore, genetic studies demonstrate a high association between mental disorders and suicidal behavior [42–44].

To date, more than 2,500 genes associated with suicidal behavior have been identified, 40 of which are linked to the cell cycle and DNA repair [45]. In a large study of the European population, two significant loci were found, including six single-nucleotide polymorphisms (rs34399104, rs35518298, rs34053895, rs66828456, rs35502061, and rs35256367), associated with the risk of suicide [42]. According to the results of another study, including 122,935 participants, three more polymorphisms were found to increase the risk of suicide (rs62535711, rs598046, rs7989250) [43]. The role of other polymorphisms in the genesis of suicidal behavior in mental disorders is also suggested, for example, rs4809706, rs4810824, and rs6019297 [44].

Associations of genes involved in various signaling pathways with suicidal behavior in patients have also been established [46–48]. A 20% increase in CD68 mRNA levels was found in the prefrontal and anterior cingulate cortex of individuals with completed suicide, which in turn explains the increase in cerebral cytokines, including tumor necrosis factor α and interleukin-1β (IL-1β) [49]. A potential role in the genesis of suicidal behavior is also attributed to changes in the expression of genes involved in the biosynthesis of gamma-aminobutyric acid (GABA) and adenosine triphosphate (ATP) [50]. Additionally, increased DNA-dependent ATPase activity has been found in suicidal individuals, and changes in the expression of polyamines (involved in immunity, oxidative stress, cell proliferation / apoptosis) and their metabolic enzymes suggest their potential role in suicidal behavior in patients [51]. Changes in the expression of genes encoding catechol-O-methyltransferase (COMT), brain-derived neurotrophic factor (BDNF), monoamine oxidase A (MAOA), serotonin (5-HTTLPR, HTR2A), and adrenergic receptors (ADRA2A, ADRA2B) are associated with an increased risk of suicide in patients with mental disorders [52].

Modern data confirm the heritability and genetic predisposition of the suicide risk. At the same time, accumulated data suggest a polygenic nature of

suicidal behavior. Thus, the heritability of suicide attempts may be caused by the accumulation of relevant genes, an example of pleiotropic interaction and/or epistasis. Therefore, the underlying causes of suicide can be diagnosed early for preventive treatment.

MOLECULAR MARKERS OF A SUICIDE RISK

Attempts to identify peripheral biomarkers that can predict suicidal behavior have long been made by assessing serotonin metabolism disorders [53–55]. Low serotonin activity has been associated with suicide in the general population [53]. One early study demonstrated significantly lower concentrations of the serotonin metabolite 5-hydroxyindoleacetic acid (5-HIAA) in the cerebrospinal fluid of individuals with major depression who had committed suicide [55].

Clearly, the pathophysiological changes in suicidal behavior are not limited to the functioning of monoamine metabolism. Using high-throughput technologies, in particular microarray-based gene expression profiling, made it possible to identify molecular pathways that were previously not suspected of involvement in suicidal behavior: GABAergic, glutamatergic, and polyamine neurotransmission [56].

The role of inflammation in suicidal behavior was proposed as early as in 1993, when elevated levels of IL-2 were found in individuals who had attempted suicide [57], and this was later confirmed in other studies, backing the links between immune system imbalance and the pathophysiology of suicide [58, 59]. Some authors point to a potential role of IL-6 in the genesis of suicidal behavior [59–61]. The presence of IL-6 receptors on brain cells confirms its effect on neurons, thereby causing aggressive / helpless behavior by regulating monoaminergic neurotransmitters and their metabolites in the central nervous system, in addition to synaptic transmission and regulation of neuroplasticity [59, 60]. Furthermore, studies have shown that patients with suicide attempts have elevated concentrations of tumor necrosis factor α, transforming growth factor β1, vascular endothelial growth factor, kynurenic acid, IL-1β, and IL-6 and lower levels of interferon γ, IL-2, and IL-4 [61].

Apparently, suicidal behavior is associated with dysfunction of the hypothalamic – pituitary – adrenal and hypothalamic – pituitary – thyroid axes [62–67]. Elevated cortisol levels activate microglia and cause neuroinflammation, disrupting BDNF function, and then induce neurotoxicity, leading to neuronal death [62]. Suicidal individuals were found to have higher levels of cortisol in saliva, cerebrospinal fluid, and plasma than healthy volunteers [63, 64]. Patients with depression and suicide attempts were found to have elevated levels of corticotropin-releasing hormone in the paraventricular nucleus of the hypothalamus, forebrain, and locus coeruleus [65]. Suicide attempts are also significantly more common in patients with hypothyroidism [66, 67]. There is a suggestion that low thyroid-stimulating hormone levels may be associated with a predisposition to depression and suicidal behavior [67].

Some studies suggest that cholesterol levels may be a potential marker of depression and suicide risk [68]. A meta-analysis of primary prevention trials of statins (cholesterol-lowering drugs) showed that they reduce the risk of cardiovascular mortality but increase the risk of suicide [69]. On the one hand, studies show that patients at high risk of suicidal behavior had lower total cholesterol levels than the control group [68, 69]. On the other hand, Y. Molero et al. [70] did not find any significant associations between cholesterol levels and suicidal behavior.

CONCLUSION

To date, there is no unified perspective that fully explains the genesis of suicidal behavior, and potential biological factors in its development vary significantly across studies, depending on the methods and approaches used. Existing studies are typically limited to searching for one or a few markers or factors and do not consider the integrity of the human body, with its inherent complementarity of both pathogenic and sanogenic factors, including socio-environmental factors, compensatory mechanisms, adaptation thresholds, and reversible and irreversible decompensation.

Based on data collected in recent studies examining a variety of biological markers associated with suicide, it can be confirmed that suicidal

behavior in individuals with mental disorders is a complex, multifactorial, and polygenic mental state, and a relevant area of research.

Understanding the genesis of suicidal behavior provides the basis for its prevention. Further improvement of molecular genetic methods, neuroimaging technologies, and brain function is necessary to uncover neural networks and their molecular and biochemical associations with the risk of developing suicidal behavior in patients in order to develop new pathogenetically based prediction models of suicidal tendencies.

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