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Posttraumatic stress disorder: a narrative review

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Post-traumatic stress disorder (PTSD) is a syndrome and mental disorder that may develop either immediately or after a delay in adults, adolescents, and children (regardless of ethnicity, nationality, culture, or age) who have experienced or witnessed one or more traumatic events, a series of events, or a set of circumstances.

The aim of this review is to contextualize PTSD within the semantic framework of syndrome description, based on an integrated body of reliable and interrelated knowledge on the subject.

PTSD in children, adolescents, and women is examined through the following dimensions: statistics and epidemiology; clinical symptomatology; diagnostic methods; elements of research methodology; brain growth chart databases and age-normed MRI templates of the brain; target anatomy; genetics; key elements of PTSD pathogenesis; the microbiome; PTSD and comorbidity; PTSD risk prediction; stress resilience; PTSD treatment strategies; pharmacological agents for the prevention and treatment of PTSD; probiotics; evidence-based classical pharmacotherapies; non-recommended pharmacological agents and treatment approaches; and promising directions for future research.

Conclusions. The relevance of research on PTSD is driven by the high prevalence of traumatic events in human life and their consequences, including the development of PTSD. PTSD can adversely affect various aspects of an individual's life, including mental and physical health, social relationships, and overall functioning. The need for continued research in this field is associated with the search for effective methods of diagnosis, treatment, and rehabilitation of individuals suffering from PTSD, as well as with the development of preventive measures aimed at reducing the likelihood of this disorder.

The authors declare no conflict of interest.

Keywords: post-traumatic stress disorder, statistics, epidemiology, clinical symptoms, brain, magnetic resonance imaging, drugs, microbiota, comorbidity.

Посттравматичний стресовий розлад: наративний огляд

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Посттравматичний стресовий розлад (ПТСР) – це синдром, психічний розлад, що може виникати як одразу, так і з відтермінуванням у дорослих, підлітків і дітей (незалежно від етнічної приналежності, національності, культури чи віку), які пережили або стали свідками однієї чи кількох травматичних подій, серії подій або низки обставин.

Мета – контекстуалізація ПТСР у межах семантичного поля опису синдрому на основі інтегрального комплексу взаємопов'язаних достовірних знань із цієї теми.

ПТСР у дітей, підлітків та жінок розглянуто в наступних аспектах: статистика та епідеміологія; клінічна симптоматика; методи обстеження; елементи методології досліджень; бази карт росту головного мозку та нормовані за віком магнітно-резонансно-томографічні шаблони головного мозку; анатомія мішені; генетика; елементи патогенезу ПТСР; мікробіота; ПТСР і коморбідність; прогнозування ПТСР; стресостійкість; стратегії лікування ПТСР; лікарські засоби (ЛЗ) для профілактики та лікування ПТСР; пробіотики; доказово рекомендовані класичні ЛЗ; нерекомендовані ЛЗ та методи лікування; перспективні напрями майбутніх досліджень.

Висновки. Актуальність досліджень ПТСР зумовлена високою поширеністю травматичних подій у житті людей та їхніми наслідками, зокрема розвиток ПТСР. ПТСР може негативно впливати на різні аспекти життя людини: психічне та фізичне здоров'я, соціальні відносини та функціонування в цілому. Необхідність досліджень у цій галузі пов'язана з пошуком ефективних методів діагностики, лікування та реабілітації осіб, які страждають на ПТСР, а також із розробкою профілактичних заходів для зниження ймовірності розвитку цього розладу.

Автори заявляють про відсутність конфлікту інтересів.

Ключові слова: посттравматичний стресовий розлад, статистика, епідеміологія, клінічна симптоматика, головний мозок, магнітно-резонансна томографія, лікарські засоби, мікробіота, коморбідність.

Stress is a universal human phenomenon; however, individual responses to it can vary significantly depending on personal characteristics. **Posttraumatic stress disorder (PTSD)** is a syndrome and mental disorder that may develop either immediately or after a delay in adults, adolescents, and children (regardless of ethnicity, nationality, culture, or age) who have experienced or witnessed one or more traumatic events, a series of events, or a set of circumstances. These may include physical, sexual, psychological, or other forms of violence (e.g., war, societal aggression, domestic violence, abduction, school shootings), crimes; natural disasters (such as earthquakes, hurricanes, or floods); man-made disasters, motor vehicle or aviation accidents, fires, and historical trauma. PTSD is characterized by intrusive memories, avoidance of trauma-related stimuli, heightened arousal (including hypervigilance), and negative beliefs and mood [25,49]. Individuals may perceive such events or circumstances as emotionally or physically harmful or life-threatening, potentially affecting their mental, physical, social, and/or spiritual well-being [2,3].

Any event that poses a threat to life or results in physical harm may trigger the development of PTSD. Emotional trauma can initiate a cascade of neurobiological processes with long-term consequences, including alterations in gene expression. Early-life maltreatment and neglect of a child's basic needs can disrupt the regulation of the developing neurobiological system, diminishing its stress resilience and, in turn, leading to later difficulties in emotional regulation during adulthood [17].

Terminological Aspects. The pathological condition currently referred to as PTSD has been known to humanity since ancient times – dating back as far as the phenomenon of war itself. Throughout different historical periods, a variety of terms were used to describe it: «nostalgia» (the Seven Years' War) [36], «soldier's heart» (the American Civil War), «traumatic neurosis,» «shell shock» (World War I), «combat fatigue» (World War II), and «war neuroses."

In the context of pediatrics, such concepts as «developmental trauma disorder» and «pediatric medical traumatic stress» are of particular interest, from which PTSD can be distinguished based on the specificity of its clinical symptomatology.

The aim of this review is to contextualize PTSD within the semantic framework of syndrome description, based on an integrated body of reliable and interrelated knowledge on the subject.

Statistics and Epidemiology. Approximately 70.4% of people worldwide report having experienced at least one traumatic event during their lifetime. In the United States, 83% of individuals report having encountered a traumatic event at least once in their life, and among this group, the lifetime prevalence of PTSD is 6.8% [13].

PTSD is more frequently diagnosed in women than in men [13]. A high prevalence of PTSD has been observed among women with the following social profile: young, unemployed, socially vulnerable, unmarried at the time of assessment, with a low level of education, and working in high-risk professional sectors (e.g., military service, police, firefighting) [25].

The prevalence of PTSD in the general population ranges from 5% to 10%. Among direct victims of disasters and natural catastrophes, prevalence rates are significantly higher – reaching 30% to 40%. The proportion of suicide attempts among individuals with PTSD is approximately 19% [49].

According to the data [7], the lifetime prevalence of PTSD ranges from 2.3% to 9.1% in the civilian population and from 6.7% to 50.2% among military personnel; from 10% to 20% in women and from 6% to 8% in men. These rates vary depending on the type and severity of the traumatic event, as well as on economic, cultural, and social factors, and the research methodology employed. During the COVID-19 pandemic, PTSD prevalence fluctuated depending on the population category and observation period, ranging from 12% to 27% in the general population, up to 30% in high-risk groups (pregnant women, individuals with cancer, HIV, or other chronic illnesses), 17% to 29% among healthcare workers, and 6.5% to 61% among individuals infected with the virus.

Among children, the prevalence of traumatic experiences is estimated at 31%, and the incidence of PTSD by the age of 18 reaches 7.8%. One-year prevalence rates range from 3.5% to 4.7% [44].

Clinical Symptomatology. PTSD is characterized by three clusters of symptoms: re-experiencing the traumatic event, avoidance of trauma-related memories, and a persistent sense of current threat, manifested as exaggerated startle responses and hypervigilance. Symptom duration varies widely – from full recovery within three months to symptom persistence for more than 50 years. A comprehensive and detailed description of symptoms is provided in publications [26]. In 2013, PTSD was revised in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) [2], having been reclassified from the

category of anxiety disorders to a newly defined group of trauma-related disorders. This reclassification aimed to better differentiate PTSD from anxiety and depressive disorders, with which it shares partial symptom overlap. According to the updated criteria, a diagnosis of PTSD requires significant exposure to a traumatic event that results in substantial impairment in occupational and social functioning for more than one month.

However, in an effort to encompass the diversity of post-traumatic symptoms, the DSM-5 classification has become increasingly amorphous. It has been demonstrated that the diagnostic criteria for PTSD in DSM-5 (Diagnostic and Statistical Manual of mental disorders) exhibit a high degree of heterogeneity and allow for 636,120 possible symptom combinations [16].

Early Research. The first article dedicated to combat-related PTSD, based on MRI findings of hippocampal volume in chronic PTSD, was published in 1996 [18]. The first study utilizing functional MRI (fMRI) in PTSD was published in 2000 [33]. Initial studies on heritability and genetic correlations in PTSD were conducted in 2017 [13].

Methods of Examination. To investigate brain function, structure, and connectivity, as well as to identify biomarkers of PTSD, neuroimaging techniques are most commonly used: functional MRI (fMRI), structural MRI (sMRI), and diffusion-weighted MRI (dMRI). The standard method for assessing white matter integrity is diffusion tensor imaging (DTI), which is based on measuring the directional movement of water molecules during diffusion. Fractional anisotropy, a parameter measured by DTI, is considered a quantitative indicator of the microstructural integrity of white matter, reflecting fiber density, axonal diameter, and degree of myelination. Fiber tractography and DTI-derived metrics are used to study structural connectivity between specific brain regions.

Resting-state fMRI (rs-fMRI) is a method for studying the macrostructure of spontaneous neural activity based on the analysis of low-frequency oscillations (0.01–0.08 Hz) of the blood oxygenation level-dependent (BOLD) signal in the absence of tasks or external stimuli (at rest). The BOLD signal fluctuates with high temporal coherence between spatially distinct brain regions that are functionally connected, forming the basis of specific sensory, motor, and cognitive networks [47]. Therefore, rsfMRI analyses often rely on temporal correlations between the signal time series of two brain regions, which are thought to reflect the synchronization of spontaneous neural activity between them; hence the term «(functional) network connectivity."

Elements of the research methodology are described to some extent in article [41]. Most studies focus mainly on brain structures; however, it is reasonable to anticipate that interest in the chronoarchitecture of nearly all organs and body systems will increase over time. Trauma is also an imprint left by painful experiences on the mind, brain, and the entire body [45].

Currently, databases are being developed based on parallel time series of structural and functional images, functional connectivity indices, topography of brain organ metabolites, as well as physical, mental, and cognitive health metrics, biochemical, microbiological, and immunological characteristics of biospecimens from infants, children, adolescents, and adults of various ethnic backgrounds, taking into account environmental characteristics and social context. In essence, this constitutes a high-throughput resource for constructing informative models of organ and system development, simulating the whole organism, mapping associations (for example, between brain indices and behavior), establishing a foundation for prospective studies of health and disease trajectories, and identifying bifurcation points along these trajectories.

In essence, this constitutes a science-intensive resource for building informative models of organ and system development, simulating the whole organism, constructing association maps (e.g., between brain metrics and behavior), creating a foundation for prospective studies of health and disease trajectories, and identifying bifurcation points along these trajectories.

In particular, in the context of studying PTSD as well as other conditions, the following areas should be noted: the creation of models of neurostructural and neurofunctional development and growth of the human brain; normalization of MRI images (e.g., voxel-based morphometry); solving functional tasks involving functional connectivity (BOLD); constructing 3D head models for the analysis of electroencephalography/magnetoencephalography source substrates; performing accurate alignment and reliable identification of normal, variant, atypical, and pathological brain development trajectories; preventive prediction of the manifestation of orphan, psychiatric, and somatic diseases. Examples of available databases are provided below.

The brain growth chart database and age-normalized brain MRI templates include: 494 subjects aged from 4.5 to 19.5 years (with 6-month intervals) [35]; subjects aged from 8 days to 4.3 years (13 age groups: 2 weeks, 3, 4.5, 6, 7.5, 9, 12, 15, 18 months, 2, 2.5, 3, and 4 years); as well as subjects aged from 2 weeks to 89 years (intervals: 3 months – up to 1 year; 6 months – up to 19.5 years; 5 years – from 20 to 89 years) [34]. The basic component of the dataset is an averaged MRI template for each age category.

Baby Connectome Project [20]. The primary goal is to characterize brain development and behavior in typically developing infants during the first 5 years of life. High-resolution sMRI (T1- and T2-weighted images), dMRI, and resting-state fMRI were used to assess functional connectivity. Subjects were children aged 0 to 5 years with typical development, without the use of sedation.

The ABCD Project (*Adolescent Brain Cognitive Development)* [10] is an ongoing 10-year prospective cohort study involving more than 11,500 children, starting at the age of 9–10 years and continuing into early adulthood. The project is conducted by a consortium of 21 data collection sites across the United States (established in 2015; data collection began in 2017). Data are collected in seven domains: physical health, mental health, brain imaging, biospecimens (hair, blood, saliva, and deciduous teeth), neurocognition, substance use, and culture and environment. The ABCD imaging protocol includes 3D T1- and 3D T2-weighted images, diffusion-weighted imaging (DWI), resting-state fMRI, and three functional fMRI tasks.

The database, compiled from materials of 18 international datasets (including the ABCD study) containing dMRI images and covering almost the entire human lifespan (total N=51,830 individuals; age range: 3–80 years), was created to build a model of brain white matter (WM) microstructure [46].

The database for constructing reference charts, created on the basis of the largest and most comprehensive dataset [11] (with acknowledgment and consideration of the limitations associated with known biases in MRI studies regarding the representativeness of the global population), was compiled from 123,984 MRI scans obtained from more than 100 primary studies involving 101,457 participants, ranging in age from 115 days post-conception to 100 years.

An MRI database of typically developing school-aged children from the USA and China was compiled at 1.25-year intervals [12]. Analysis of these data revealed volumetric growth differences between these two ethnic groups – primarily in the lateral frontal and parietal regions, which show the greatest interindividual variability in structure and function. For infants aged 0-9 months, imaging sessions were conducted during natural sleep without the use of sedation, following established MRI protocols [11].

Target anatomy. Structural and functional imaging in PTSD has primarily focused on five key brain regions: the hippocampus and parahippocampal areas (known as the medial temporal lobe), the ventromedial prefrontal cortex, the amygdala, the anterior cingulate cortex, and the insular cortex/insula [32].

In a meta-analysis of regions of interest in patients with PTSD compared with all control subjects, reductions were observed in total brain volume, intracranial volume, and the volumes of the hippocampus, insular cortex (insula), and anterior cingulate cortex. Similar changes were observed in individuals with PTSD compared with both non-traumatized and traumatized control subjects. In traumatized control participants compared with non-traumatized controls, bilateral hippocampal volume reduction was noted [6].

Genetics. Twin studies have demonstrated that approximately 30% of the variance associated with PTSD is attributable solely to genetic factors. To date, the potential role of 16 genes in conferring susceptibility to PTSD has been identified. PTSD, by its nature, is similar to other complex genetic phenotypes such as schizophrenia, depression, or human height in that it represents a highly polygenic phenotype, likely influenced by thousands of loci across the genome, many or even all of which are located in genomic regions not traditionally considered of greatest significance [13]. In women, PTSD may increase the predisposition to post-traumatic symptoms in their future offspring, including at the level of gene expression.

An analysis of pleiotropic mechanisms linking PTSD with brain imaging phenotypes has been performed [29]. In a study involving 66 PTSD patients and 91 PTSD-free volunteers, pleiotropic associations were identified between caudate nucleus volume and childhood trauma, as well as between right lateral ventricle volume and lifetime alcohol use disorder [31].

In a study involving 216 participants (Republic of Korea; 133 healthy volunteers and 83 patients with PTSD) [22], the interaction between childhood

trauma and the Val66Met polymorphism of the brain-derived neurotrophic factor (*BDNF*) in relation to PTSD symptoms and cortical thickness was examined. Structural MRI (*sMRI*) with T1-weighted images, genotyping of the *BDNF* rs6265 variant from blood samples, and clinical assessment were performed. The thickness of both brain regions showed significant correlations with psychological symptoms, including depression, anxiety, rumination, and cognitive emotion regulation strategies; however, this was predominantly observed in individuals with the Val/Val genotype. The interaction between childhood trauma and the *BDNF* polymorphism significantly affects PTSD symptoms and cortical thickness, and the Val/Val genotype may increase the risk of PTSD in the Korea's population.

Elements of PTSD pathogenesis. To date, no generally accepted theory of the mechanisms underlying PTSD pathogenesis exists. Based on the analysis of hormonal, biochemical, genetic, and morphofunctional changes occurring in PTSD in peripheral organs and in the central nervous system, it has been concluded that PTSD pathogenesis should be considered as an integrative inflammatory process involving both peripheral and central systems [26].

Microbiota. Within the framework of systems biology, the human being is considered as an organism in which the microbiota (a non-pathogenic microbial community comprising more than 10,000 species of microorganisms, including bacteria, archaea, fungi, and protozoa) functions as a kind of invisible «extracorporeal organ,» consisting of approximately 10^{14} microbial cells (comparable to the number of the body's own cells), weighing about 2–3 kg, and performing a wide range of vital local and systemic functions [24,51].

The «microbiota-gut-brain» axis provides a bidirectional link between gut microbes and the brain. The potential role of the gut microbiome in mental health and in a number of chronic diseases has been recognized [30,48]. The severity of PTSD symptoms in women may differ from that in men. Sex is one of the important host factors influencing the human microbiome.

It has been established that dysbiosis of the human gut microbiome is associated with various inflammatory conditions also linked to PTSD, including inflammatory bowel disease (IBD), cardiometabolic diseases, and diabetes mellitus [24]. It was found [48] that certain taxa correlate with the severity of PTSD symptoms: the genera *Mitsuokella*, *Odoribacter*, *Catenibacterium*, and *Olsenella* (the abundance of this consortium was higher in the South African cohort of patients with PTSD, and based on this combination, PTSD was identified with an accuracy of 66.4% [28]), as well as the phyla *Actinobacteria*, *Lentisphaerae*, and *Verrucomicrobia*.

Differences in the taxonomic composition of gut microorganisms have been identified between groups of patients with PTSD and a trauma-exposed control group. Individuals with PTSD are characterized by reduced microbiome diversity. The practical significance of potential risk and resilience factors for PTSD has been substantiated, including a specific composition of the gut microbiome that may potentially protect against or increase susceptibility to the development of this disorder [48]. The role of potential risk and stress resilience factors for PTSD, including specific gut microbiome compositions that may either confer protection or increase susceptibility to the disorder, has been practically substantiated [48].

A certain composition of the gut microbiome provides a degree of stress resilience, likely through the production of anti-inflammatory short-chain fatty acids (SCFAs). Administration of *Mycobacterium vaccae* reduces stressor-induced hippocampal microglial sensitivity to immune challenge *ex vivo* and has been shown to exert other local anti-inflammatory effects [1].

PTSD reduces microbial synchrony between mother and child. The impact of war on the development of PTSD in children in Israel was examined in article [51]. Observations and five assessment rounds were conducted starting from 2004–2005 among children who had experienced war-related traumatic events and among control group participants – from early childhood (mean age – 2.76 years, n=232) to adolescence (mean age – 16.13 years, n=84). Fecal samples were collected from mothers and adolescents, and the composition, diversity, and microbial synchrony of the microbiome between mother and child were assessed.

Adolescents with PTSD had a lower level of microbial diversity (Shannon index) compared with stress-resilient peers, as well as lower microbial synchrony with their mothers. This suggests that reduced microbial concordance between mother and child may indicate increased susceptibility to PTSD. Low microbiome diversity correlated with a higher number of post-traumatic symptoms in early childhood, a greater number of emotional and behavioral

problems in adolescence, and poor maternal care. In germ-free mice that received a microbiome transplant from adolescents with PTSD, increased anxiety-like behavior was observed. The microbiome profile associated with traumatic experience at least partially determines the anxiety component of the PTSD phenotype and highlights the involvement of microbial factors in stress resilience mechanisms. The microbiome serves as an additional biological memory of early childhood stress and represents a promising target for pharmacotherapy [51].

According to a meta-analysis (5,824 articles) [30], two out of six studies reported a decrease in alpha diversity of the gut microbiota in patients with PTSD (standardized mean difference – SMD – for the Shannon diversity index was 0.27; 95% confidence interval: -0.62 to 0.609; p=0.110). Two studies observed a significant reduction in the abundance of bacteria from the *Lachnospiraceae* family. In one of these studies, bacteria of this taxon were positively correlated with PTSD symptom severity scores, whereas in two others, *Lachnospiraceae* (which ferment dietary fibers producing short-chain fatty acids (SCFAs) that exert anti-inflammatory and modulatory effects on the intestinal mucosa) were associated with higher levels of cognitive functioning. Other results did not demonstrate consistent patterns and appeared to be unique to each individual study [24,30].

PTSD and Comorbidity. In most cases, PTSD is accompanied by major depressive disorder, panic attacks, dysthymia, alcoholism, substance abuse, social phobia, antisocial behavior, aggressiveness, and psychosomatic disorders. The identification of patients with pronounced comorbid symptoms may be of substantial clinical importance, as these individuals are at the highest risk for long-term chronic posttraumatic dysfunctions [19]. Depression occurs as frequently as PTSD following a traumatic event, and these two disorders are highly comorbid. PTSD is often accompanied by major depressive disorder, and empirical evidence indicates that effective treatment of PTSD simultaneously contributes to a reduction in depressive symptoms [8].

PTSD is associated with an increased risk of developing IBD. Studies have shown that patients with IBD are more susceptible to PTSD, and the presence of PTSD, in turn, exacerbates the course of IBD symptoms [24].

PTSD is a risk factor for chronic diseases, including coronary heart disease, stroke, diabetes mellitus, as well as premature mortality.

Prognosis. PTSD develops in only a small proportion of individuals exposed to stressogenic factors. For example, 60% of men and 50% of women in developed, prosperous countries have encountered at least once in their lifetime situations of a psychotraumatic nature that could potentially lead to the development of PTSD. However, only 20–30% of individuals from this at-risk group (i.e., according to various estimates, 1–10% of the general population) actually develop PTSD.

Additional risk factors for PTSD development, not directly related to the traumatic event, have also been identified: younger age at the time of trauma exposure, female sex, lower socioeconomic status, lack of social support, premorbid personality traits, and the presence of anxiety or depressive disorders, which increase the likelihood of PTSD development [3].

Prediction of PTSD symptoms was described in a sample of 87 women (mean age – 34.22 years, range – 18-65 years) at 1, 6, and 14 months after a traumatic event, using an artificial intelligence (AI) deep learning model based on fMRI scan results (in the resting state, during an emotional reactivity task, and the «Safe or Risky Domino Choice» test) obtained immediately after the trauma. For each participant, activation levels in 117 brain regions were analyzed [39]. The predictive ability for PTSD chronicity was achieved with Area Under the ROC Curve (AUC): AUC = 0.84±0.02 and accuracy = 81.33%±5.37.

Stress Resilience. Parameters of stress exposure that enhance stress resistance in adult rats have been identified. Stress during critical developmental periods forms a phenotype with increased resilience to inflammatory pain exposure, which was observed in a response organized at the supraspinal level in adult animals [9].

It is assumed that resilience (the ability to experience trauma without developing PTSD symptoms) was formed at the genetic level in the course of evolution. Resilience to a given type of danger is higher the earlier in evolutionary history that danger emerged. In particular, the following patterns are observed.

MRI markers of human stress resilience have been identified: stress-resilient individuals exhibit increased gray matter volume in the prefrontal cortex and hippocampus; increased activation of the prefrontal cortex, anterior cingulate cortex, and anterior spinal region; and decreased activation of the amygdala [15]. In stress-resilient adolescents, a larger gray matter volume in the frontal regions and hippocampus has

been noted. fMRI studies have revealed the role of the amygdala and ventral striatum in shaping stress resilience. Stress resilience is associated with greater structural connectivity of the corpus callosum. Neural circuits involved in emotion regulation and the reward system are linked to stress resilience in youth [14].

Veterans seeking treatment for combat-related PTSD have higher rates of childhood physical abuse compared to combat veterans without PTSD. Childhood physical abuse preceded the development of combat-related PTSD in Vietnam War veterans [5].

Treatment strategies for PTSD primarily focus on psychotherapy (talk therapy) and pharmacotherapy, including the use of selective serotonin reuptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs).

In a meta-analysis of 19 randomized controlled trials of cognitive behavioral therapy (CBT) in children or adolescents with PTSD (published before July 25, 2021, and retrieved from seven databases), outcomes were compared with those of a control group that included treatment as usual or other types of therapy. Compared to the control, CBT was effective in reducing PTSD symptoms in children and adolescents; however, CBT was not effective in reducing avoidance symptoms. CBT may decrease the severity of PTSD in children and adolescents and alleviate symptoms of depression and anxiety, as has been proven in the treatment of PTSD in victims of sexual abuse and war, as well as in patients over the age of seven years [36,50].

In 56 studies conducted across 30 countries (1,370 adults and children with PTSD who underwent narrative exposure therapy and 1,055 individuals in control groups), significant between-group effects were identified in favor of narrative exposure therapy in reducing PTSD symptoms in the long term [36,40].

From the perspective of PTSD symptom reduction, eye movement desensitization and reprocessing and CBT demonstrated no statistically significant differences [21,36].

Some hope is placed on digital technologies for the treatment of PTSD through the use of the Internet for CBT and written exposure therapy [36]. These interventions have advantages such as improved accessibility, effectiveness, and reduced stigmatization compared to traditional face-to-face therapy. The treatment involves the use of an online program containing self-learning modules that patients can access from their computer, tablet, or smartphone. The program also includes six face-to-face therapy sessions [4].

Pharmacotherapy for the prevention and treatment of PTSD. In PTSD, the efficacy of SSRIs is the most extensively studied; these agents reduce anxiety and facilitate engagement in psychotherapy [3,5,27,44,52]. The therapeutic aims of antidepressants extend beyond alleviating depression to include obsessive-compulsive symptoms, intrusive thoughts, anxiety-phobic states, anger outbursts, and alcohol craving.

The search for **medicinal products (MPs)** is also carried out within the framework of drug repurposing – a field that now has a clearly defined name, although it has existed previously (the use of known MPs for new indications). The rationale for searching for an effective MP within the scope of repurposing lies in the fact that intracellular signaling pathways are characterized by numerous cross-interactions. Based on the consideration of PTSD primarily as an inflammatory integrative process of the peripheral and central systems, the use of a multifunctional MP from the heparin group – capable of crossing the blood-brain barrier (BBB) and possessing adaptogenic properties – has been justified [26].

In this context, the pleiotropic effects of oxytocin (dose-dependent anti-phobic, stress-reducing, sedative, anxiolytic, cardioprotective, regenerative, and antiapoptotic actions; intranasal administration has demonstrated potential in reducing PTSD symptoms) [43]; dalargin (wound-healing, reparative, anti-inflammatory, antioxidant, lymphokinetic, hypotensive, antiarrhythmic, cardioprotective, pulmonary-protective, hepatoprotective, pancreatic-protective, hypocholesterolemic, antiatherosclerotic, antihypoxic, anti-ischemic, analgesic, antidepressant, antistressor, immunomodulatory, anticarcinogenic, antitumor, and antimetastatic actions) [37]; phenytoin (anticonvulsant, analgesic, anti-inflammatory, sedative, tonic, antihypoxic, and antitoxic actions) [5]; berberine (anti-inflammatory, antifungal, antiarrhythmic, geroprotective, antiapoptotic, antiviral, antisclerotic, neuroprotective, antidiabetic, anxiolytic – manifested in reduced anxiety, fear, and emotional tension – antioxidant, and antitumor actions) [27,38]; curcumin (anti-inflammatory, antidepressant, hepatoprotective, antibacterial, and antitumor actions) [27]; tetramethylpyrazine (antioxidant, anti-inflammatory, antiapoptotic, angioprotective, cardioprotective, neuroprotective, and geroprotective actions) [27]; citalopram (antidepressant effect) [5]; propranolol (hypotensive, cardioprotective, antianginal, antiarrhythmic, membrane-stabilizing, antitumor, actoprotective, and antiatheroge-

nic actions) [3]; as well as N-acetylcysteine (NAC) [23] (mucolytic, antitoxic, neurotropic, antioxidant, anti-inflammatory, nephroprotective, anti-infective, radioprotective, pulmonary-protective, and anticarcinogenic actions) are under investigation.

A multicenter, randomized, double-blind, placebo-controlled study involving adults with treatment-resistant PTSD has been described. Patients received oral NAC at a dose of 2.7 g/day or placebo for 12 weeks. Significant between-group differences were observed at week 64 in the duration of craving for psychoactive substances (Cohen's d=1.61) and resistance to this craving (Cohen's d=1.03) – both outcomes in favor of NAC. The therapeutic action of NAC was investigated in a mouse model of PTSD, which significantly improved cognitive function and reduced hippocampal neuronal apoptosis [52].

Pharmacological treatment of PTSD should always be combined with psychotherapy. For example, NAC shows certain potential as a treatment for PTSD in combination with CBT.

Probiotics. It has been suggested that greater alpha diversity of the gut microbiome protects the body against pathogenic influences of any nature [42]. The microbiome represents an additional biological memory of early-life stress and a target for MPs [51].

A certain composition of the gut microbiome provides a degree of stress resilience, presumably through the production of anti-inflammatory SCFAs. Administration of *Mycobacterium vaccae* reduces stressor-induced sensitivity of hippocampal microglia to immune challenge ex vivo and has also been shown to exert other local anti-inflammatory effects [1].

The influence of the gut microbiota on stress response leads to assumptions about the antidepressant effect of probiotics such as *Bifidobacterium*. Overall, the regulation of microbiota diversity through probiotics and prebiotics may represent a potential therapeutic approach for depression in PTSD.

Evidence-based recommended classical MPs [36,44]: fluoxetine (for monotherapy), paroxetine (same), venlafaxine XR (same), sertraline.

Non-recommended MPs and treatment methods [36,44]. MPs: risperidone, quetiapine, olanzapine and other atypical antipsychotics; divalproex, tiagabine, guanfacine, ketamine, hydrocortisone, D-cycloserine; benzodiazepines; cannabis; cannabinoids.

There is insufficient evidence to support the use of the following methods: electroconvulsive therapy; repetitive transcranial magnetic stimulation; hyperbaric oxygen therapy; stellate ganglion block; vagus nerve stimulation.

Conclusions

The relevance of research on posttraumatic stress disorder (PTSD) is determined by the high prevalence of traumatic events in people's lives and their consequences, including the development of PTSD. PTSD can negatively affect various aspects of human life, including mental and physical health, social relationships, and overall functioning. The need for research in this field is associated with the search for effective methods of diagnosis, treatment, and rehabilitation of individuals suffering from PTSD, as well as with the development of preventive measures to reduce the likelihood of this disorder.

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Promising directions for future research: the formation of large national cohorts (women, children, adolescents) with data on the gut microbiome, PTSD, as well as potential host factors (demographic, socioeconomic, and medical indicators); prospective assessment of trauma/PTSD prior to microbiome sampling in cohort participants to study the association of PTSD with the microbiome independently of trauma exposure; integration of multi-omics data (metagenomics, metatranscriptomics, and metabolomics) for systematic identification of mechanisms linking the gut and PTSD (previous studies have shown that an abnormal gut environment, such as intestinal barrier dysfunction, concentrations of SCFAs and various microbial metabolites, is associated with PTSD); identification of causal relationships to determine predictive species/pathways contributing to PTSD, which would facilitate the rational development of synbiotics for the prevention and treatment of PTSD.

References/Література

1. Admon R, Lubin G, Stern O, Rosenberg K, Sela L et al. (2009). Human vulnerability to stress depends on amygdala's predisposition and hippocampal plasticity. Proceedings of the National Academy of Sciences of the United States of America. 106(33): 14120-14125. https://doi.org/10.1073/pnas.0903183106.
2. American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders. 5th ed. American Psychiatric Publishing.
3. Auxéméry Y. (2012). Posttraumatic stress disorder (PTSD) as a consequence of the interaction between an individual genetic susceptibility, a traumatogenic event and a social context. Encephale. 38(5): 373-380. https://doi.org/10.1016/j.encep.2011.12.003.

4. Blackie M, De Boer K, Seabrook L, Bates G, Nedeljkovic M. (2024). Digital-based interventions for complex post-traumatic stress disorder: A systematic literature review. Trauma, Violence, & Abuse. 25(4): 3115-3130. https://doi.org/10.1177/15248380241238760.
5. Bremner JD, Southwick SM, Johnson DR, Yehuda R, Charney DS. (1993). Childhood physical abuse and combat-related posttraumatic stress disorder in Vietnam veterans. American Journal of Psychiatry. 150(2): 235-239. https://doi.org/10.1176/ajp.150.2.235.
6. Bromis K, Calem M, Reinders AATS, Williams SCR, Kempton MJ. (2018). Meta-analysis of 89 structural MRI studies in posttraumatic stress disorder and comparison with major depressive disorder. American Journal of Psychiatry. 175(10): 989-998. https://doi.org/10.1176/appi.ajp.2018.17111199.
7. Burback L, Brémault-Phillips S, Nijdam MJ, McFarlane A, Vermetten E. (2024). Treatment of posttraumatic stress disorder: A state-of-the-art review. Current Neuropharmacology. 22(4): 557-635. https://doi.org/10.2174/1570159X21666230428091433.
8. Burton MS, Cooper AA, Mello PG, Feeny NC, Zoellner LA. (2021). Latent profiles of comorbid depression as predictors of PTSD treatment outcome. Behavior Therapy. 52(4): 970-981. https://doi.org/10.1016/j.beth.2020.12.005.
9. Butkevich IP, Mikhailenko VA, Vershinina EA. (2020). Combination of stressful influences during critical periods of development increases resistance to stress of inflammatory pain in adult rats. Russian Journal of Physiology. 106(3): 267-282. https://doi.org/10.31857/S0869813920030024.
10. Casey BJ, Cannonier T, Conley MI, Cohen AO, Barch DM, Heitzeg MM et al. (2018). The Adolescent Brain Cognitive Development (ABCD) study: Imaging acquisition across 21 sites. Developmental Cognitive Neuroscience. 32: 43-54. https://doi.org/10.1016/j.dcn.2018.03.001.
11. Dean DC, Tisdall MD, Wisnowski JL, Feczko E, Gagoski B, Alexander AL et al. (2024). Quantifying brain development in the HEALthy Brain and Child Development (HBCD) Study: The magnetic resonance imaging and spectroscopy protocol. Developmental Cognitive Neuroscience. 70: 101452. https://doi.org/10.1016/j.dcn.2024.101452.
12. Dong HM, Castellanos FX, Yang N, Zhang Z, Zhou Q, He Y et al. (2020). Charting brain growth in tandem with brain templates at school age. Science Bulletin. 65(22): 1924-1934. https://doi.org/10.1016/j.scib.2020.07.027.
13. Duncan LE, Cooper BN, Shen H. (2018). Robust findings from 25 years of PTSD genetics research. Current Psychiatry Reports. 20(12): 115. https://doi.org/10.1007/s11920-018-0980-1. Erratum: Duncan LE, Cooper BN, Shen H. (2018). Erratum to: Robust findings from 25 years of PTSD genetics research. Current Psychiatry Reports. 20(12): 119. https://doi.org/10.1007/s11920-018-0984-x.
14. Eaton S, Cornwell H, Hamilton-Giachritsis C, Fairchild G. (2022). Resilience and young people's brain structure, function and connectivity: A systematic review. Neuroscience & Biobehavioral Reviews. 132: 936-956. https://doi.org/10.1016/j.neubiorev.2021.11.001.
15. Egan LA, Park HRP, Gatt JM. (2024). Resilience to stress and trauma: A narrative review of neuroimaging research. Current Opinion in Behavioral Sciences. 58: 101408. https://doi.org/10.1016/j.cobeha.2024.101408.
16. Galatzer-Levy IR, Bryant RA. (2013). 636,120 ways to have posttraumatic stress disorder. Perspectives on Psychological Science. 8(6): 651-662. https://doi.org/10.1177/1745691613504115.
17. Giotakos O. (2020). Neurobiology of emotional trauma. Psychiatriki. 31(2): 162-171. https://doi.org/10.22365/jpsych.2020.312.162.
18. Gurvits TV, Shenton ME, Hokama H, Ohta H, Lasko NB, Gilbertson MW et al. (1996). Magnetic resonance imaging study of hippocampal volume in chronic, combat-related posttraumatic stress disorder. Biological Psychiatry. 40(11): 1091-1099. https://doi.org/10.1016/S0006-3223(96)00229-6.
19. Harnett NG, van Rooij SJH, Ely TD, Lebois LAM, Murty VP, Jovanovic T et al. (2021). Prognostic neuroimaging biomarkers of trauma-related psychopathology: Resting-state fMRI shortly after trauma predicts future PTSD and depression symptoms in the AURORA study. Neuropsychopharmacology. 46(7): 1263-1271. https://doi.org/10.1038/s41386-020-00946-8.
20. Howell BR, Styner MA, Gao W, Yap PT, Wang L, Baluyot K et al. (2019). The UNC/UMN Baby Connectome Project (BCP): An overview of the study design and protocol development. NeuroImage. 185: 891-905. https://doi.org/10.1016/j.neuroimage.2018.03.049.
21. Hudays A, Gallagher R, Hazazi A, Arishi A, Bahari G. (2022). Eye movement desensitization and reprocessing versus cognitive behavior therapy for treating post-traumatic stress disorder: A systematic review and meta-analysis. International Journal of Environmental Research and Public Health. 19(24): 16836. https://doi.org/10.3390/ijerph192416836.
22. Jin MJ, Jeon H, Hyun MH, Lee SH. (2019). Influence of childhood trauma and brain-derived neurotrophic factor Val66Met polymorphism on posttraumatic stress symptoms and cortical thickness. Scientific Reports. 9(1): 6028. https://doi.org/10.1038/s41598-019-42563-6.
23. Kanaan RA, Oliver G, Dharan A, Sendi S, Maier A, Mohebbi M et al. (2023). A multi-centre, double-blind, 12-week, randomized, placebo-controlled trial of adjunctive N-acetylcysteine for treatment-resistant PTSD. Psychiatry Research. 327: 115398. https://doi.org/10.1016/j.psychres.2023.115398.
24. Ke S, Hartmann J, Ressler KJ, Liu YY, Koenen KC. (2023). The emerging role of the gut microbiome in posttraumatic stress disorder. Brain, Behavior, and Immunity. 114: 360-370. https://doi.org/10.1016/j.bbi.2023.09.005.
25. Kunimatsu A, Yasaka K, Akai H, Kunimatsu N, Abe O. (2020). MRI findings in posttraumatic stress disorder. Journal of Magnetic Resonance Imaging. 52(2): 380-396. https://doi.org/10.1002/jmri.26929.
26. Lapshin MS, Kondashevskaya MV, Epishev VV, Patochkina NA. (2023). Pathogenesis of post-traumatic stress disorder, therapeutic targets. Advances in Physiological Sciences. 54(1): 55-69. https://doi.org/10.31857/S0301179823010058.
27. Lee B, Shim I, Lee H, Hahm DH. (2018). Berberine alleviates symptoms of anxiety by enhancing dopamine expression in rats with post-traumatic stress disorder. Korean Journal of Physiology & Pharmacology. 22(2): 183-192. https://doi.org/10.4196/kjpp.2018.22.2.183.
28. Malan-Muller S, Valles-Colomer M, Foxx CL, Vieira-Silva S, van den Heuvel LL, Raes J et al. (2022). Exploring the relationship between the gut microbiome and mental health outcomes in a posttraumatic stress disorder cohort relative to trauma-exposed controls. European Neuropsychopharmacology. 56: 24-38. https://doi.org/10.1016/j.euroneuro.2021.11.009.
29. Nievergelt CM, Maihofer AX, Klengel T, Atkinson EG, Chen CY, Choi KW et al. (2019). International meta-analysis of PTSD genome-wide association studies identifies sex- and ancestry-specific genetic risk loci. Nature Communications. 10(1): 4558. https://doi.org/10.1038/s41467-019-12576-w.
30. Petakh P, Oksenych V, Kamyshna I, Boisak I, Lyubomirskaya K, Kamyshnyi O. (2024). Exploring the interplay between posttraumatic stress disorder, gut microbiota, and inflammatory biomarkers: A comprehensive meta-analysis. Frontiers in Immunology. 15: 1349883. https://doi.org/10.3389/fimmu.2024.1349883.

31. Polimanti R, Wendt FR. (2021). Posttraumatic stress disorder: From gene discovery to disease biology. Psychological Medicine. 51(13): 2178-2188. https://doi.org/10.1017/S0033291721000210.
32. Prasad A, Chaichi A, Kelley DP, Francis J, Gartia MR. (2019). Current and future functional imaging techniques for post-traumatic stress disorder. RSC Advances. 9(42): 24568-24594. https://doi.org/10.1039/c9ra03562a.
33. Rauch SL, Whalen PJ, Shin LM, McInerney SC, Macklin ML, Lasko NB et al. (2000). Exaggerated amygdala response to masked facial stimuli in posttraumatic stress disorder: A functional MRI study. Biological Psychiatry. 47(9): 769-776. https://doi.org/10.1016/s0006-3223(00)00828-3.
34. Richards JE, Sanchez C, Phillips-Meek M, Xie W. (2016). A database of age-appropriate average MRI templates. NeuroImage. 124; Pt B: 1254-1259. https://doi.org/10.1016/j.neuroimage.2015.04.055.
35. Sanchez CE, Richards JE, Almli CR. (2012). Neurodevelopmental MRI brain templates for children from 2 weeks to 4 years of age. Developmental Psychobiology. 54(1): 77-91. https://doi.org/10.1002/dev.20579.
36. Schrader C, Ross A. (2021). A review of PTSD and current treatment strategies. Missouri Medicine. 118(6): 546-551. URL: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8672952/.
37. Semenova OG, Vyushina AV, Pritvorova AV, Rakitskaya VV, Ordyan NE. (2024). Morphological adrenal glands changes in rats with different individual-typological behavior features in the PTSD model after dalargin injections. Russian Journal of Physiology. 110(1): 58-78. https://doi.org/10.31857/S0869813924010048.
38. Shayganfard M. (2023). Berberine: Is it a promising agent for mental disorders treatment? Current Molecular Pharmacology. 16(3): 307-320. https://doi.org/10.2174/1874467215666220509213122.
39. Sheynin S, Wolf L, Ben-Zion Z, Sheynin J, Reznik S, Keynan JN et al. (2021). Deep learning model of fMRI connectivity predicts PTSD symptom trajectories in recent trauma survivors. NeuroImage. 238: 118242. https://doi.org/10.1016/j.neuroimage.2021.118242.
40. Siehl S, Robjant K, Crombach A. (2021). Systematic review and meta-analyses of the long-term efficacy of narrative exposure therapy for adults, children and perpetrators. Psychotherapy Research. 31(6): 695-710. https://doi.org/10.1080/10503307.2020.1847345.
41. Soares JM, Magalhães R, Moreira PS, Sousa A, Ganz E, Sampaio A et al. (2016). A hitchhiker's guide to functional magnetic resonance imaging. Frontiers in Neuroscience. 10: 515. https://doi.org/10.3389/fnins.2016.00515.
42. Spragge F, Bakkeren E, Jahn MT, B N Araujo E, Pearson CF, Wang X et al. (2023). Microbiome diversity protects against pathogens by nutrient blocking. Science. 382(6676): eadj3502. https://doi.org/10.1126/science.adj3502.
43. Stauffer CS, Morrison TE, Meinzer NK, Leung D, Buffington J, Sheh EG et al. (2022). Effects of oxytocin administration on fear-potentiated acoustic startle in co-occurring PTSD and alcohol use disorder: A randomized clinical trial. Psychiatry Research. 308: 114340. https://doi.org/10.1016/j.psychres.2021.114340.
44. Torrico TJ, Mikes BA. (2024). Posttraumatic stress disorder in children. In StatPearls [Internet]. StatPearls Publishing. URL: https://www.ncbi.nlm.nih.gov/books/NBK559140/.
45. Van der Kolk BA. (2014). The body keeps the score: Brain, mind, and body in the healing of trauma. New York, NY: Viking.
46. Villalón-Reina JE, Zhu AH, Nir TM, Thomopoulos SI, Laltoo E, Kushan L et al. (2023, Nov). Large-scale normative modeling of brain microstructure. 2023 19th International Symposium on Medical Information Processing and Analysis (SIPAIM). https://doi.org/10.1109/SIPAIM56729.2023.10373451.
47. Wei W, Zhang K, Chang J, Zhang S, Ma L, Wang H et al. (2024). Analyzing 20 years of resting-state fMRI research: Trends and collaborative networks revealed. Brain Research. 1822: 148634. https://doi.org/10.1016/j.brainres.2023.148634.
48. Winder C, Lodhia A, Basso M, Cohen Kadosh K. (2025). Gut microbiome differences in individuals with PTSD compared to trauma-exposed controls: A systematic review. Frontiers in Neuroscience. 19: 1540180. https://doi.org/10.3389/fnins.2025.1540180.
49. Xiao S, Yang Z, Su T, Gong J, Huang L, Wang Y. (2022). Functional and structural brain abnormalities in posttraumatic stress disorder: A multimodal meta-analysis of neuroimaging studies. Journal of Psychiatric Research. 155: 153-162. https://doi.org/10.1016/j.jpsychires.2022.08.010.
50. Xian-Yu CY, Deng NJ, Zhang J, Li HY, Gao TY et al. (2022). Cognitive behavioral therapy for children and adolescents with posttraumatic stress disorder: Meta-analysis. Journal of Affective Disorders. 308: 502-511. https://doi.org/10.1016/j.jad.2022.04.111.
51. Yirmiya K, Turjeman S, Shtossel O, Zagoory-Sharon O, Moadi L, Rubin E et al. (2024). Microbiome signature of posttraumatic stress disorder and resilience in youth. Psychological Trauma: Theory, Research, Practice, and Policy. 17(7): 1490-1504. https://doi.org/10.1037/tra0001727.
52. Zhou Y, Yuan X, Guo M. (2025). Unlocking NAC's potential ATF4 and m6A dynamics in rescuing cognitive impairments in PTSD. Metabolic Brain Disease. 40(2): 129. https://doi.org/10.1007/s11011-024-01485-7.

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