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Transplanted Cells, Transferred Minds: Can Transplanted Cells Influence Mental Illness? (A Review)

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Abstract—Mental disorders such as schizophrenia, major depressive disorder, autism spectrum disorders, and various psychoses exhibit incredibly diverse clinical presentations, with symptoms varying significantly among patients. Numerous studies have provided substantial evidence that genetic factors play a major role in the development of such conditions, particularly schizophrenia. However, the genetics of schizophrenia and other mental disorders are highly complex. The development of these diseases is influenced not only by genetic factors, but also by other, often unidentified, contributors. For a long time, this led to the belief that the pathogenesis of major psychiatric disorders—primarily schizophrenia—could not be directly linked to transmissible processes. In other words, it was considered impossible for a mental illness to be transmitted from one person to another. In recent years, however, some findings have challenged this assumption. There have been reports of schizophrenia-like symptoms emerging in recipients of bone marrow transplants from donors diagnosed with schizophrenia, as well as cases of sustained remission in patients with schizophrenia following transplantation from donors without any psychiatric history. Experimental animal models have also provided evidence supporting the plausibility of such a mechanism. While these mechanisms cannot alter the recipient's genotype, they likely have the potential to initiate or suppress pathological processes. At present, there is no widely accepted biological explanation for how these effects occur. Nonetheless, ongoing research in this area appears crucial for understanding the pathogenesis of mental disorders and for developing innovative therapeutic strategies. The aim of this review is to summarize current research on the potential role of transmissible mechanisms in schizophrenia and other neuropsychiatric disorders, and to explore how these findings may inform the development of novel therapeutic approaches.

Keywords: schizophrenia, psychosis, mental disorders, cell transplantation, bone marrow, stem cells

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INTRODUCTION

The term psychosis refers to a pathological condition in which a person loses touch with reality. Psychoses can be caused by various reasons is also one of the main signs of schizophrenia—a mental illness of a person (Lasalvia and Ruggeri, 2018). Schizophrenia is a complex disorder of unknown etiology associated with disorders of neurotransmitter systems (Striebel, 2024). A schizophrenic may experience periods of psychosis or be in such a period for a long time. Schizophrenia is a chronic mental disorder that affects about one percent of the population (Charlson et al., 2018; Nakazawa, 2022). Currently, a significant proportion of patients have a high chance of long-term remission, but many patients become disabled (Charlson et al., 2018). Symptoms of schizophrenia are varied and typically include delusions, hallucinations, disorganized speech, cognitive impairment, loss of motivation, and associated desocialization (Striebel, 2024). Symptoms of schizophrenia are usually divided into three main categories: Positive symptoms are the presence of signs that are absent normally: Auditory and visual hallucinations, paranoia, distortion of perception, and behavior (Jauhar et al., 2022). Negative symptoms, that is, the absence of signs that are present normally: impaired emotional interaction, decreased speech

production (alogia), asociality, decreased desire and motivation to initiate purposeful activities, anhedonia (Jauhar et al., 2022). Cognitive symptoms: confused and disordered thinking and speech, impaired logical thinking, problems with attention, concentration and memory (Jauhar et al., 2022; Striebel, 2024). In many, but not all cases, treatment with antipsychotics in conjunction with psychotherapy leads to more or less prolonged remission. The development of the disease is influenced by many genetic and non-genetic factors, the latter likely to play a trigger role in the disease process. It is currently not possible to determine the exact cause of the disease in each individual case (García-Cabrerizo and Cryan, 2024).

The problem of the biological mechanism of schizophrenia has always been at the forefront of neurobiological research. One of the most developed areas is the dopamine hypothesis of schizophrenia (Brisch et al., 2014; Striebel, 2024). Most of researchers believe that hyperactivation of the dopamine system in the mesolimbic pathway contributes to the positive symptoms of schizophrenia, whereas hypodopaminergia in the mesocortical pathway is responsible for negative symptoms (Brisch et al., 2014). The dopamine hypothesis is supported by the results of many clinical and preclinical studies, but the development of schizophrenia is not limited to dopamine disorders alone (Winterer, 2006; Roeske et al., 2021). In addition, while quite successfully explaining the symptoms of the disease, the dopamine hypothesis does not explain its mechanism. Of course, the lack of understanding of this mechanism complicates therapy. To complicate matters, schizophrenia is actually a general term for a group of mental disorders that affect an individual's perception of reality. In other words, it is a spectrum disorder that describes a range of conditions that have similar symptoms but may have different underlying biological mechanisms.

However, while the biological mechanisms of schizophrenia may be varied and not fully understood, scientists can focus on understanding at least some of them. It is more than likely that at least some of them are associated with neuroinflammation (Chaves et al., 2024). Many studies suggest that inflammation in the central nervous system (CNS), as well as some other immune dysfunctions, may play a significant role in the pathogenesis of schizophrenia (Tao et al., 2025). The term neuroinflammation usually refers to an inflammatory response in the brain or spinal cord. From a biological point of view, it is a complex response of the immune system to injury, intoxication, disease or infection of the central nervous system (Bernier et al., 2019).

It has been repeatedly shown that neuroinflammation can cause tissue damage, cell death, and contribute to the progression of some neurodegenerative diseases of the brain (Calcia et al., 2016; Wu et al., 2025). There is a large amount of data explaining the role of neuroinflammation in various mental and neurological disorders. Most theories agree that stress of various etiologies can initiate deregulation of the immune system (Ermakov et al., 2022). Along with genetic and epigenetic factors, this can cause the development of a number of mental pathologies, including schizophrenia (Bennett and Molofsky, 2019). The dopamine hypothesis does not contradict the results on the role of neuroinflammation in the development of schizophrenia. Dopamine itself can have an immunomodulatory effect (Arreola et al., 2016; Channer et al., 2023).

It has been shown that microglia play a very important role in the pathogenesis of schizophrenia (Uranova et al., 2023). Microglia originate from mesodermal cells, particularly erythromyeloid cells (EMCs). These cells arise early in embryogenesis, before the nervous system is formed. They then migrate to the CNS and differentiate into microglia, the key cells responsible for brain homeostasis and immune responses in the brain throughout adulthood. It is possible that dopamine receptors in microglial cells allow direct interaction between dopamine and the immune system. Thus, inflammation can affect dopamine function, and dopamine can affect inflammatory processes. Neuroinflammation is recognized as a potential factor in the development and progression of schizophrenia (Murphy et al., 2021). Patients with schizophrenia also showed elevated C-reactive protein, cytokine dysregulation, elevated neutrophils and autoantibodies, and dysregulated microbiota (Gobshtis et al., 2019; Ermakov et al., 2022). All these factors are not observed in all patients with SZ and are not present simultaneously in all cases, but with schizophrenia the probability of these factors increases significantly.

People with schizophrenia have elevated inflammatory markers and also have changes in the immune system (Miller and Goldsmith, 2019). The first hit occurs due to maternal immune activation (MIA) during the fetal and perinatal stages (Talukdar et al., 2020). This initial hit primes the immune system, particularly affecting microglia (Handunnetthi et al., 2021). When

activated, microglia can respond to stressors by initiating excessive pro-inflammatory responses, which negatively impacts brain development (Ozaki et al., 1995). It was demonstrated that microglia, especially in the gray matter of the brain, are activated in schizophrenia. Upon activation, microglia release proinflammatory cytokines and free radicals. Being neurotoxic factors these chemicals contributing to cognitive decline. The second hit typically occurs in late adolescence or adulthood and is associated with dopaminergic imbalance and increased synaptic pruning during the onset of psychosis (Chafee and Averbeck, 2022). The two-hit hypothesis highlights the critical role of both prenatal and postnatal factors in neuroinflammation in schizophrenia, but does not fully explain the pathogenesis of the disease.

Can bone marrow transplantation be a cause of the development of mental disorders? Cell transplantation is a therapeutic method in which cells that do not originally belong to the tissue at the site of administration, and often obtained from another individual, are transplanted into a specific tissue of the recipient (Mansourabadi et al., 2021). Various cell transplantation protocols are widely used in modern medicine. Most widespread bone marrow transplant (BMT) is a therapy for patients with certain types of cancer or certain other diseases (Simpson and Dazzi, 2019). BMT involves taking cells that are normally found in the bone marrow, filtering those cells, and transplanting them into either the same patient or another person (Simpson and Dazzi, 2019). BMT for cancer is the most widely known, but far from the only form of therapy based on cell transplantation (Castello et al., 2004). There are a large number of BMT protocols aimed at different clinical tasks, and transplantation of bone marrow cells directly into the bone marrow is more effective in at least some cases (Castello et al., 2004). Preclinical studies show that intravenous bone marrow cell transplantation can enhance angiogenesis and neurogenesis (Castello et al., 2004). This strategy is being used more and more widely, and not only in hematology oncology (Simpson and Dazzi, 2019). It is natural that the question of the possibility of using cell transplantation in the treatment of mental disorders is increasingly being discussed (Villanueva, 2025). A number of animal experiments have shown that stem cells may offer treatments for disorders such as depression and schizophrenia (Donegan et al., 2016).

Most researchers agree that immune dysregulation, characterized by altered cytokine levels, immune-related gene expression, and neuroinflammation, plays an important role in the pathogenesis of schizophrenia (Jayakumar et al., 2024).

It has been shown that immune dysfunction occurs at both the systemic and brain levels and is manifested by changes in cytokine profiles, phagocytic activity and immunogenic potential (Jayakumar et al., 2024). Immune dysfunction is also accompanied by the generation of microparticles—vesicles, which are released by cells of the immune system (Ermakov et al, 2022). These microparticles carry out the transfer of biologically active molecules between cells, that is, they play an important role in intercellular communication and activation of immune system cells (Ermakov et al, 2022). The role of immune-related genes CD19 and CD20 in schizophrenia has been shown. Immune dysregulation and changes in neuroinflammatory pathways in schizophrenia are likely related to dopamine-induced activation of autoimmune T-cells (de la Fontaine et al., 2006; Schwieler et al., 2015). This causes cytokine release and may lead to microglial activation, contributing to the progression of inflammatory processes and neurodegeneration in schizophrenia (Schwieler et al., 2015; Uranova et al., 2023; Jayakumar et al., 2024). It is known from clinical practice that pharmacological treatment aimed at reducing the level of inflammatory cytokines to some extent reduces the severity of psychotic symptoms (Malavia et al., 2017). Numerous studies have shown a significantly higher incidence of bone marrow diseases, such as leukemia, in patients with schizophrenia (Miyaoka et al., 2017; Jayakumar et al., 2024). The pathogenesis of schizophrenia has been linked to bone marrow by a study demonstrating an increase in reticular cells, abnormal lymphocytes, and multinucleated giant cells in patients with schizophrenia (Ekström et al., 2022). However, the cause-and-effect relationship between these phenomena remains unclear.

The purpose of this review is to present evidence that the bone marrow transplantation can transfer (or eliminate) symptoms of psychiatric diseases, especially schizophrenia, both in clinical practice and in animal models.

BONE MARROW TRANSPLANTATION AND MENTAL DISORDERS

Although a life-saving procedure for patients with a number of serious diseases, BMT can lead to unexpected pathologies. A rare phenomenon is the transmission

of genetic diseases through hematopoietic progenitor cells (HPCs). Transmissive transmission through donor transplants of cyclic neutropenia, Gaucher disease and some other diseases is possible (Morgan et al., 2017; Somaraju and Tadepalli, 2017). This possibility requires careful genetic screening of the donor before transplantation.

A major impetus for the development of research in this direction occurred as a result of a recent clinical case that received great resonance in the biomedical community. A 23-year-old male patient was diagnosed with treatment-resistant paranoid schizophrenia according to DSM-IV-TR (Miyaoka et al., 2017). The patient's physical and neurological parameters were normal, and no pathologies were detected using computed tomography and magnetic resonance imaging of the brain. Treatment with antipsychotics was unsuccessful (Miyaoka et al., 2017). At the age of 24 years, the patient was diagnosed with acute myeloid leukemia, while auditory hallucinations and delusions continued. The patient underwent BMT. To prevent graft-versus-host disease, the patient received the immunosuppressants methotrexate and cyclosporine A, following a standard protocol. (Miyaoka et al., 2017). After thirty days, the patient's psychotic symptoms had almost disappeared. After eight years, the improvement in somatic and psychiatric symptoms was maintained, and the patient has no residual psychiatric symptoms (Miyaoka et al., 2017).

Using of immunosuppressant medications is a routine practice during bone marrow transplants, particularly allogeneic transplants where donor cells are used. Immunosuppressants prevent the transplanted stem cells (graft) from attacking the recipient's body (host), so-called as graft-versus-host disease (GVHD). There is a very wide range of immunosuppressants, all of which have different side effects (Yoshida et al., 2008). Among these side effects are neuropsychiatric ones, but our knowledge about them is limited. This is because it is difficult to separate whether and to what extent the neuropsychiatric disturbances observed are a direct result of the patient's disease or the immunosuppressants (Bourgeois and Hategan, 2014; Bösche et al., 2015). Neurological and mental side effects of immunosuppressants are considered to be non-specific and therefore difficult to predict.

Apparently, this case is not unique. A 21-year-old male patient with schizophrenia diagnosed at age 15 reportedly received multiple stem cell transplants (SCTs) for Hodgkin lymphoma (González-Llano et al., 2018). Within eight months after the second transplantation, the patient's symptoms of schizophrenia decreased, hallucinations decreased by 90%, and cognitive symptoms improved (González-Llano et al., 2018). The authors of this publication, however, note that the recovery was not complete, and the severity of the disease before transplantation remained unknown (González-Llano et al., 2018).

Around the same years, that is, in the last decade, an almost mirror case was described and published. A male patient with a clear psychiatric history was diagnosed with chronic lymphocytic leukemia (CLL) and bone marrow aplasia at the age of 67 years (Sommer et al., 2014). In connection with this, he received a BMT from one of his brothers. This brother, 12 years younger than the patient, suffered from schizophrenia and was regularly treated with antipsychotic drugs. He had completed hematologic recovery without complications 4 weeks after SCT. However, several weeks later he developed acute psychotic symptoms: frequent hallucinations and delusions. The patient was hospitalized in a psychiatric clinic (Sommer et al., 2014; Gobshtis et al., 2019). Treatment with antipsychotics was ineffective, remission did not occur, and the patient died several years later for an unknown reason (Sommer et al., 2014).

These reports gave rise to the hypothesis that the risk of mental disorders may be transmitted through bone marrow cell transplantation. A study was conducted to evaluate the possibility that the development of mental disorders in donors could be transmitted to the recipient (Ekström et al., 2022). The authors concluded that, based on a statistical analysis of 1363 donor-recipient pairs, transmission of risk for psychosis, bipolar disorder or depression could not be confirmed (Ekström et al., 2022). The study tested the hypothesis that the risk of mental disorders could be transmitted through hematopoietic stem cell transplantation (HSCT). The approach used to study diseases transmitted through blood transfusions was used. All cases of receiving stem cells from a donor who had been diagnosed with psychosis, bipolar disorder or depression were checked (Ekström et al., 2022). However, as reported by the authors themselves, the main limitation of this study is limited power due to the low incidence of psychiatric disorders in the sample. There has also been no analysis of improvement or recovery in psychiatric patients after transplantation (Ekström et al., 2022). Post-transplant depression is a common complication of cancer treatment, but in some cases, it may be related

to the donor's psychiatric history. Some controversial cases include a report of the development of depression and anxiety in a 33-year-old patient after HSCT from his brother, who had a history of recurrent depressive disorder (Weydt et al., 2011; Rømer et al., 2024).

However, although a few cases of transmission of mental disorders through cell transplantation are considered proven, such cases are quite rare (Ekström et al., 2022). The study specifically found no statistically significant risk of transmission of psychosis or bipolar disorder (Ekström et al., 2022). On the other hand, it has been found that about half of patients who undergo a traditional stem cell transplant show signs of delirium, but these symptoms may be mild, go away without treatment, and do not attract the attention of medical personnel (Fann et al., 2011, 2005).

Schizophrenia, like many other mental and neurological disorders, is associated with neuroinflammation. The clinical picture of schizophrenia and Parkinson's disease (PD) are very different, but there are similarities in their pathogenesis. Neuroinflammation plays an important role in the development and progression of PD, contributing to the damage of neurons producing dopamine (Castillo-Rangel et al., 2023). Like schizophrenia, Parkinson's disease affects the brain's dopamine system. It has been shown that people with schizophrenia spectrum disorders may have an increased risk of developing PD. Parkinson's disease is characterized by decreased levels of dopamine in the brain, which leads to movement disorders and can also cause mental problems, primarily depression (Leal et al., 2013; Castillo-Rangel et al., 2023). In PD, microglia are activated, resulting in the release of proinflammatory cytokines (IL-1β and TNF) that can damage neurons. Astrocytes also contribute to neuroinflammation by releasing proinflammatory cytokines. Proinflammatory cytokines increase damage to dopamine neurons (Leal et al., 2013). A large statistical study has led to some unexpected results: as it turns out, kidney transplantation significantly reduces the risk of developing PD in a patient (Baek et al., 2021). This correlation is not limited to kidney transplants—patients who have received kidney, heart, lung and BMTs have a 37% lower risk of developing PD compared to the general population (Fan et al., 2019; Baek et al., 2021).

ANIMAL MODELS AND PRECLINICAL RESEARCH

So, a number of clinical cases have shown that most likely, in some cases, the development of a mental disorder may be associated with transmissible factors. This gave impetus to animal studies aimed at identifying mechanisms linking mental disorders to cell transplants.

Some animal studies examining the transmission of mental and neurological diseases through cell transplantation have yielded very revealing results. The mouse model can't perfectly replicate Alzheimer's in animals, but mice model develops some of the symptoms associated with the condition. They also develop beta-amyloid clumps in their brains, but lack tangles of the protein tau. One of the main mouse models of Alzheimer's disease is the 5xFAD (five Familial Alzheimer's Disease—FAD—mutations) animal strain, a model of familial Alzheimer's disease (Plachez et al., 2023). Bone marrow from 5xFAD mice was transplanted into healthy control mice (Singh et al., 2024). Recipient mice developed accelerated cognitive decline, amyloid plaques and blood-brain barrier (BBB) dysfunction. This study challenges the neurocentric view of Alzheimer's disease (Inyushin et al., 2017). It is logical to assume that similar mechanisms can work in the case of PD (Singh et al., 2024).

Various studies have demonstrated a link between blood-brain barrier (BBB) dysfunction and mental disorders (Zhang et al., 2025). It is likely that increased BBB permeability may facilitate the infiltration of immune cells and proinflammatory molecules into the brain, leading to pathological changes (Najjar et al., 2017). Molecular mechanisms of neuronal dysfunction in schizophrenia and a number of other mental pathologies may be associated with a violation of the BBB, but may also play a separate role. In many cases, they are associated with a violation of glutamate neurotransmission. Analysis of genes associated with SCZ, as well as postmortem transcriptomic and proteomic studies of SCZ, consistently point to synaptic dysfunction, in many cases accompanied by immunological abnormalities (Farsi and Sheng, 2023). However, it is unclear how these abnormalities may lead to the diverse clinical syndromes.

AUTISM SPECTRUM DISORDER (ASD)

Autism spectrum disorder (ASD) is a mental problem that can cause significant social, communication and

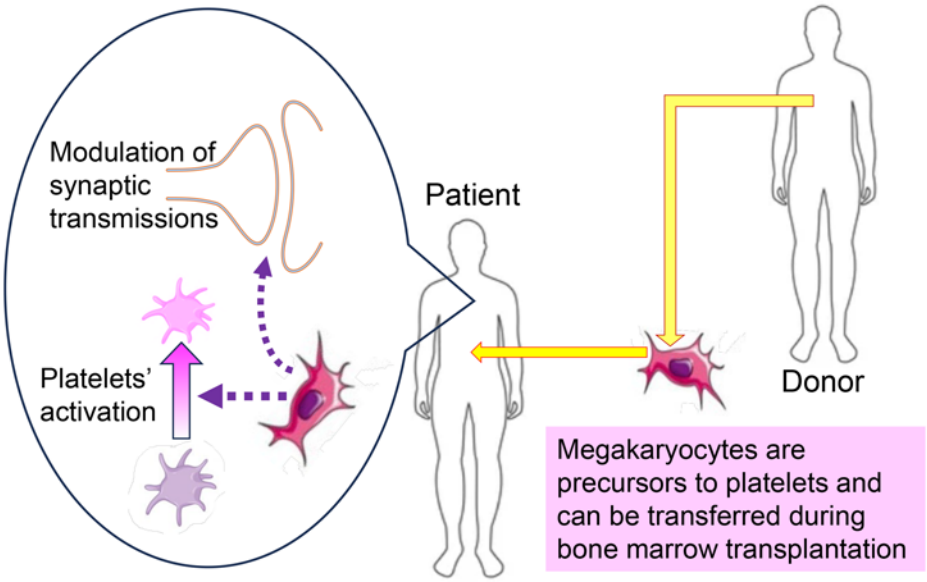

Fig. 1. Megakaryocytes in the bone marrow enter the patient's body during therapeutic transplantation. Being precursors of platelets, through neuroimmune interactions they can influence neuroinflammation and modulate synaptic transmissions to a wide extent.

behavioral problems. Although the exact causes of autism spectrum disorders (ASD) are complex and not fully understood, genetic factors play a key role (Zhang et al., 2024). There appear to be a number of biological mechanisms that may act independently, ultimately directing an individual's development toward what is eventually called ASD (Yasuda et al., 2023). Rodent models of ASD include various genetically modified or inbred mouse strains that exhibit certain traits characteristic of humans with ASD, as well as models with targeted mutations in genes involved in information processing (Kazdoba et al., 2016).

The BTBR (Bipolar and Trem2-mutated) mouse model is a commonly used inbred strain that exhibits behavioral and neuroanatomical features similar to those observed in human with autism spectrum disorder (ASD). These animals demonstrate reduced social interaction, low exploratory behavior, along with unusual vocalizations and high anxiety. The *BTBR* T^{+} $Itpr^{3tf/J}$ (BTBR) mouse line is characterized not only by disturbances in social behavior, but also by disturbances in the immune system, which makes it possible to study the relationship between behavior and immunity (O'Connor et al., 2021). These animals demonstrate reduced social interaction, low exploratory behavior, along with unusual vocalizations and high anxiety. Unlike autistic animals, C57BL/6J mice are highly social animals without immune system abnormalities. In an experimental study, BTBR mice received BMTs from wild type mice (Schwartzer et al., 2017). According to the results of behavioral testing after transplantation, BTBR recipient mice that received allogeneic bone marrow from C57 donor mice, but not syngeneic BTBR bone marrow, showed increased sociability and grooming behavior (Schwartzer et al., 2017). These very impressive data provide compelling evidence for a causal relationship between peripheral immune phenotype and social behavior (Schwartzer et al., 2017).

POSSIBLE MECHANISMS EXPLAINING THE EFFECTS AFTER BONE MARROW TRANSPLANTATION

Bone marrow transplantation can exert neuromodulatory effects through several cell types. These include hematopoietic stem cells, neural stem cells, glial cells—primarily oligodendrocytes, astrocytes and microglia. It is possible that neuromodulatory effects are exerted by endothelial cells that form the blood-brain barrier, and cells of the peripheral immune system. Platelet abnormalities have been demonstrated to be associated with ASD (Padmakumar et al., 2019). As is known, platelets are formed and released into

the bloodstream by precursor cells—megakaryocytes located in the bone marrow (Patel et al., 2005; Ehrlich et al., 2012). Megakaryocytes, as precursors to platelets, are transferred during bone marrow transplantation and could influence neuroimmune interactions through platelet-derived factors including serotonin and amyloid precursor protein (Cunin and Nigrovic, 2019; Koupenova et al., 2022). During a BMT, megakaryocytes from the donor enter the recipient's body (Fig. 1). These are platelet precursor cells, which can influence neuroinflammation and neurotransmission (Ehrlich et al., 2012). It would be logical to assume that bone marrow cell transplantation changes platelet activation signaling, which may also affect certain cellular elements of the central nervous system (Inyushin et al., 2020, 2017).

An indirect argument in favor of this assumption may be the fact that platelets can apparently damage the permeability of the BBB, increasing its permeability (Lv et al., 2024). Platelet-released platelet-activating factors (PAFs), P-selectin, ADP, PDGF-AA, and PDGF-CC are candidate permeability-enhancing agents (Lv et al., 2024). Platelets also secrete amyloid-β (Aβ), which triggers neuroinflammation and downregulates tight junction protein expression, which further damages the BBB. Finally, platelets can promote the release of reactive oxygen species (ROS), which damage DNA, proteins, and lipids in BBB cells (Lv et al., 2024). Platelets are highly likely to play a role in maintaining brain plasticity by modulating the generation of new neurons from progenitor cells. They can also increase the number of dendritic spines and synapses, as well as facilitate proinflammatory activation of microglia during brain damage (Dukhinova et al., 2018). Platelets carry molecules that influence brain function, including transforming growth factor-β, β-2 microglobulin, and gelsolin (Kronenberg et al., 2010; Smith et al., 2015). A mouse study showed that platelet CD40 L causes neuroinflammation and neuronal death in the hippocampus and cortex, which may be directly related to the development of neuropsychiatric pathologies in humans (Leiter and Walker, 2019).

Although platelets are not part of the nervous system, platelets from patients with schizophrenia have been shown to have specific changes, including enlarged vacuoles and increased glycogen levels (Asor and Dorit, 2012). Among many secreted substances, platelets secrete amyloid precursor protein, which is broken down into amyloid-β (Aβ) peptides, the accumulation of which in brain plaques is a major feature of AD (Roldán-Kalil et al., 2025). Platelets contain large amounts of serotonin, and the serotonergic system plays a key role in the development of behavioral disorders including depression and anxiety disorders. Moreover, platelets are able to absorb dopamine and express dopamine receptors, which suggests their involvement in the pathogenesis of schizophrenia (Ehrlich et al., 2012; Peitl et al., 2020). Platelets isolated from the blood of patients with schizophrenia carry surface antibodies at a significantly higher titer than platelets from healthy people of the same age. These observations likely support the possibility that platelet-associated antibodies (PAA) may be involved in the etiology of some psychiatric dysfunctions associated with dementia and schizophrenia (Asor and Dorit, 2012; Ehrlich et al., 2012).

CELL TRANSPLANTATION AS A THERAPEUTIC APPROACH

For now, a variety of pharmacological treatments are available to patients with mental disorders, sometimes in combination with psychotherapy. In rare cases, deep brain stimulation, electroconvulsive therapy, and transcranial magnetic stimulation can be added to the arsenal of therapeutic agents. However, the search for new therapeutic tools continues, and the inclusion of methods based on cell transplantation in the therapeutic arsenal seems very likely. Currently, many antipsychotic drugs, mainly acting as dopamine D2 receptor antagonists, are able to reduce the positive symptoms of schizophrenia (Donegan et al., 2016). Unfortunately, they are much less effective against negative symptoms, and at the same time they are not without side effects. Thus, there is there is a need for more effective treatments (Donegan et al., 2016). It would not be a mistake to say that cell transplantation is a new and quite promising direction in the field of treatment of neuropsychiatric disorders, including depression and schizophrenia. Cell transplantation is being tried to treat not only schizophrenia, but also autism spectrum disorders. But ASD is an even more heterogeneous entity than schizophrenia, and research is primarily empirical in nature, with very modest success (Shamim et al., 2023). Combining cell-based therapies with pharmacological approaches targeting neuroinflammation and platelet-mediated signaling could lead to more effective and personalized treatments for mental disorders. Bone marrow stem cells may play a role in treating neurological deficits or neurotransmitter imbalances, thereby improving psychiatric disorders

(Barzilay et al., 2011). Cellular signaling in processes associated with mental disorders is extremely complex, but to date we have only a few data.

CONCLUSION

Bone marrow transplantation is a very common treatment strategy nowadays, successfully used in clinical practice in the treatment of various diseases. This procedure is not directly related to the brain or the nervous system in general. However, as it turned out, in some cases the BMT procedure can lead to the transmission of symptoms of mental disorders. The opposite situation is also possible, when a BMT leads to remission of a patient with mental pathology. Both of these situations are rare, but the presence of such clinical cases is beyond doubt. The mechanism of such transmission action is unclear and certainly requires serious study, but it probably includes complex chains of intercellular molecular interactions, involving both the transplanted cells themselves and the cells of the central nervous system. Further studies of the transmissible factor in mental pathologies should be focused on the following areas: 1) Analysis of donor material in bone marrow transplantation. 2) Development of new pharmacological agents aimed at regulating the interaction of donor and recipient cells. Development of new pharmacological agents aimed at regulating the interaction of donor and recipient cells.

ABBREVIATIONS AND NOTATION

BMT—bone marrow transplant;

CNS—central nervous system;

PD—Parkinson's disease;

ASD—autism spectrum disorder.

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This work does not contain any studies involving human and animal subjects

CONFLICT OF INTEREST

The authors of this work declare that they have no conflicts of interest.

AUTHOR CONTRIBUTION

Conceptualization: V.Yu.T., A.B.V., M.Y.I.; project administration: A.B.V.; funding acquisition: M.Y.I., A.B.V., writing: V.Yu.T., M.Y.I.; editing: V.Yu.T., A.B.V.

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