REVIEW

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Microbiological bases of obsessive-compulsive disorder – the role of viruses, bacteria, and parasites in the onset and progression of OCD

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Obsessive-compulsive disorder (OCD) is a current topic of discussion nowadays. OCD presents a variety of different etiologies including environmental, viral, cognitive, or genetic aspects. In this article, we focused on the possible correlation between various infectious diseases as well as generally the relationship between viruses, bacteria, and parasites, and an increased OCD risk. In this narrative review, we analyzed different types of articles found on PubMed, Google Scholar, and Scopus, as well as the articles of the National Institute of Mental Health. Searching criteria included articles from 1991 till the end of November 2023, research involving human and animal patients (including monkeys and rats), and research published in English. Research showed a relationship between *Herpes simplex virus*, *Rubella virus, Human immunodeficiency virus*, *Borna disease virus*, *Mycoplasma pneumoniae*, *Toxoplasma gondii*, streptococcal infections, as well as gut microbiota and increased OCD risk. The possible mechanisms of this relation include neuroinflammation, brain tissue damage, autoimmune processes, and impairments in neurotransmitter levels. Infections caused by *Varicella zoster virus*, *Measles virus*, *Mumps virus*, *Epstein-Barr virus*, *Cytomegalovirus,* or *Borrelia Burgdorferi* may also contribute to the increased risk of OCD. Reports showed an increased frequency of OCD occurrence in a group of infected people compared to a healthy group. However, there is no evidence of the influence of *Influenza virus*, *Coxsackie virus*, *Poliovirus*, *Parvovirus B19*, *Enterovirus 71*, *West Nile virus*, *Treponema Pallidum*, or *Toxocara* infections on the OCD risk. There is a significant relationship between various infectious diseases and an increased OCD risk. However, further studies are crucial to discover the exact pathomechanisms of these correlations and the potential influence of other pathogens on the onset of OCD.

Key words: obsessive-compulsive disorder, infectious diseases, infection, psychiatric disorders

INTRODUCTION

Obsessive-compulsive disorder (OCD) is a current topic of several discussions. OCD is a chronic mental illness that is characterized by uncontrollable, recurrent thoughts as well as the same repetitive actions that significantly impair functioning (Lack, 2012; Olatunji et al., 2019). People who suffer from OCD present actions consisting of various thoughts or urges to do upsetting, time-consuming, or anxiety-inducing things. OCD also presents as repetitive motor behaviors that are performed solely to reduce anxiety ultimately leading to the obsession that something needs to be done a certain way (Drubach, 2015; Spencer et al., 2023).

However, OCD is one of the most frequent mental disorders nowadays. It is estimated that 1-3% of the population suffers from OCD. What is also crucial, while the estimates regarding the OCD prevalence remain mostly stable across cultures, the estimates of subclinical obsessive-compulsive symptoms have fluctuated from 2 to 19% of the population. There is a correlation between gender and age, and the occurrence rate of OCD. Generally, while females are affected at an amount greater rate compared to males in adulthood,

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boys are more typically impacted in childhood, with over 25% of males experiencing symptoms before the age of 10; in girls, the onset of OCD usually appears during puberty. Age is the strongest sociodemographic determinant of lifelong OCD, consistent with the early age of beginning, with the probability of onset being the highest for individuals by 18 years of age. However, there are also cases of OCD onset in individuals over the age of 18 (Drubach, 2015; Stein et al., 2019; Mathes et al., 2019; Nazeer et al., 2020; Cervin, 2023). Importantly, OCD shows high correlations with other mental illnesses. Not only depression and anxiety have been shown to significantly comorbid with OCD, but also schizophrenia, bipolar disorder (BD), body dysmorphic disorder, post-traumatic stress disorder (PTSD), phobias, or substance use disorders (Stein et al., 2019; Sharma & Reddy, 2019; Szalisznyó et al., 2019; Pampaloni et al., 2022).

The etiology of OCD is considered to be multifactorial including genetics, neuropsychological, infectious, as well as environmental factors (Krebs & Heyman, 2015; Bozorgmehr, 2017; Jalal et al., 2023). Genetic determinants of OCD occurrence may be associated with single nucleotide polymorphism (SNP) and genes. For instance, it was shown that SNPs belonging to the CAPRIN2 and IPO8 genomic regions correlate with the increased risk of OCD onset. Also, some genes including CPE and SETD3 significantly expressed in brain cells may play an important role in OCD occurrence (Alemany-Navarro, 2020; Mahjani et al., 2022). People with a family history of OCD episodes, especially involving first-degree relatives, have the predicted recurrence risk for the illness ranging from 6% to even 55%, while the general population's overall prevalence of OCD is thought to be no more than 3% (Mahjani et al., 2021; International Obsessive Compulsive Disorder Foundation Genetics Collaborative (IOCDF-GC), OCD Collaborative Genetics Association Studies (OCGAS), 2018). Furthermore, there is an increasing interest in the investigation of the association between the microbiological impact and the onset of OCD. There have been reports about a steadily increasing quantity of research about the relationship between OCD and viral and bacterial infections along with the parasites. Further, the abovementioned factors could not only be a reason for OCD onset but also lead to such mental disorders as depression, BP, schizophrenia, paediatric acute-onset neuropsychiatric syndrome (PANS), childhood acute neuropsychiatric syndrome (CANS), paediatric autoimmune neuropsychiatric disorders associated with streptococcal infections (PANDAS), or paediatric infection-triggered neuropsychiatric disorder (PITAND) (Alam et al., 2017; Calaprice et al., 2017; Belz et al., 2018; Marazziti et al., 2018; Wilbur et al., 2019; Maisarah et al., 2022; Marazziti et al., 2023).

Aim and search strategy

The aim of this narrative review was to find a correlation between different types of microbiota and an increased risk of OCD development. The literature review was based on different types of articles including reviews, meta-analyses, systemic reviews, letters to the editor, case reports, and randomized controlled trials found on PubMed, Google Scholar, Scopus, Web of Science, and articles from the National Institute of Mental Health. Searching criteria included articles from 1991 till the end of November 2023, and research involving human and animal patients (including monkeys, mice, and rats); the language of all the reviewed articles was English. The search string included the following keywords: OCD, OCD etiology, OCD and bacteria, OCD and viruses, OCD and parasites, OCD infections, OCD microbiota.

Viruses and OCD

Herpes Simplex Virus

More than 400 million people all over the world are infected with *Herpes simplex virus 1* (HSV-1) and *Herpes simplex virus 2* (HSV-2). HSV infection might appear due to contact with mucosal surfaces, genital fluids, lesions, or other bodily secretions. In most cases, the infection spreads *via* sexual transmission and this primarily concerns HSV-2 infection. HSV-1 infection, on the other hand, is a chronic infection primarily contracted orally during childhood. First manifestations such as fever, headache, tiredness, lymphadenopathy, or genital ulcers may appear during primary infection. Infections can also show up as completely asymptomatic, moderate, or subclinical (Crimi et al., 2019; Sirka, 2020; Cole, 2020; AlMukdad et al., 2023). Besides common symptoms, there might be a relationship between HSV infection and the onset of OCD. There are some studies that suggest an increased risk of OCD occurrence in patients with chronic HSV infection. The potential role of HSV infection and its effects on the central nervous system and further OCD onset and progression may be various. Firstly, inflammation processes occurring within the brain that might appear due to the infection such as encephalitis could potentially lead to neuronal tissue damage. Studies showed temporary oscillating abnormalities in the frontotemporal lobes of patients with HSV-acquired central nervous system infections (Khanna et al., 1997). Some HSV-infected patients also presented pathological EEG results or symptoms of medial temporal lobes involvement as well as abnormalities in the orbitofrontal area in magnetic resonance

imaging (MRI) (Gourie-Devi, 2006). There are two case reports available, showing OCD symptoms development related to the HSV-associated encephalitis. MRI done in one of the patients showed alterations in the cingulate gyrus, hypothalamus, and right insula, as well as the medial temporal lobe. The second patient presented hypodense lesions in the right frontal and temporoparietal areas (Więdłocha et al., 2015). Moreover, also EEG presented some alterations, revealing left centro-temporal slow and sharp waves. Further, clinical examination of the HSV-infected patients showed gliosis in temporo-frontal areas of the brain, which is also considered to play an important role in OCD development (Więdłocha et al., 2015).

The encephalitis caused by HSV infection may affect regions of the brain that are crucial in behavioural control. Activity abnormalities in the caudate nucleus, thalamus, cingulate gyrus, and cortex or in their anatomical connections may result in an increased risk of OCD because of the HSV infection (Gourie-Devi, 2006; Więdłocha et al., 2015).

Furthermore, it is possible that chronic and latent HSV infection can be periodically reactivated and lead to abnormalities in the brain structure similar to those presented above eventually leading to OCD development. Another theory postulates that changes in the body such as stress, endocrine turmoil, or other infections may provoke latent HSV infection to reappearance leading to neuronal dysfunctions being considered one of the factors associated with an increased OCD risk (Maia et al., 2022).

Varicella Zoster Virus

Varicella zoster virus (VZV) also known as *Human herpes virus 3* is a DNA α-herpes pathogen that affects human nervous tissue. It is 150–200 nm in size and presents several sequence similarities with the genome of the HSV. VZV incubation lasts from 10 to 21 days; from one to four days before the onset of the cutaneous rash until all lesions are healed, and the infected patients remain infectious. Before crusting develops, fresh lesion crops last for four to five days. Even though the illness is usually associated with mild symptoms, it can be more severe in a group of adults and elder people with other organ dysfunctions or comorbidities (Sauerbrei, 2016; Levin et al., 2016; Kennedy & Gershon, 2018; Gagliardi et al., 2019; Patil et al., 2022). VZV remains latent in adrenal glands and ganglionic neurons where it can reactivate and lead to the infection of the central nervous system (Nagel et al., 2020). Lately, several reports regarding the relationship between VZV infection and OCD development, have been described in the literature. VZV infection complications affecting the nervous system may include encephalitis, meningitis, cerebellitis, myelopathy, vasculopathy, or even neuralgia (Budman & Sarcevic, 2002). Studies suggest that auto-immune activity is set off by a molecular mimicry between VZV and the cerebrum (Budman & Sarcevic, 2002). Several case reports showed a correlation between VZV infection and several psychiatric disorders including symptoms of obsession and compulsivity, especially in a group of young people. Two cases of 6 and 10-year-old children presented similar ways of infection evolution, symptoms, neuropsychiatric complications, and recovery after immunomodulatory treatment (Dahiya et al., 2023). Both patients presented psychiatric symptoms which appeared 3-6 weeks after VZV infection. Both of the patients had no previous psychiatric medical history. Expand clinical diagnosis revealed intrathecal secretion of IgG with positive oligoclonal bands in both patients and abnormalities in neuroimaging in a second patient. PET-CT showed lesions in the occipital and parietal lobes suggesting encephalitis. The decision of immunosuppressive treatment implementation resulted in significant clinical improvement, while typical antiviral and psychiatric treatment was ineffective. The pulse of intravenous steroids given to those patients caused regression of neuropsychiatric symptoms (Dahiya et al., 2023). The possible pathomechanism of an increased OCD development risk due to the VZV infection may be related to the autoimmune processes affecting neurotransmitters and their receptors. It was discovered that anti-NMDA receptor encephalitis may appear as a result of auto-reactive anti-NMDA receptor antibody generation after VZV infection (Dale et al., 2003). Moreover, there have been reports about a VZV patient who experienced progressive encephalomyelitis that was positive for anti-Gly receptor antibodies. All the pathologies including brain tissue damage, neurotransmitter abnormalities, and autoimmunological processes may lead to elevated OCD occurrence risk (Yaramiş et al., 2009; Więdłocha et al., 2015).

Rubella Virus

A single-stranded, positive-sense RNA *Rubella virus* causes the acute disease rubella, with an estimated one hundred thousand cases of congenital rubella syndrome occurring annually. The virus is the only belonging of the *Rubivirus* family and a member of the *Togaviridae* family. Despite the fact that rubella is a clinically mild sickness with rush and fever, congenital rubella syndrome, which has major medical and public health implications, can arise from a primary *Rubella virus* infection in the early stages of pregnancy (Bouthry et al., 2014; Lambert et al., 2015; Winter & Moss, 2022). Rubel-

la infection in early life can cause serious damage to the central nervous system leading to neurological dysfunctions including psychiatric disorders. Neuroimaging of people with the congenital occurrence of rubella presents basal ganglia lesions, white matter abnormalities, and calcifications, along with a smaller intracranial volume (Yamashita et al., 1991; Lim et al., 1995; Ostrander & Bale, 2019). All the pathologies related to the congenital rubella syndrome could occur as one of the developmental factors of OCD. Furthermore, rubella infection acquired in childhood may lead to serious central nervous system inflammation resulting, for instance in encephalitis (Lane et al., 1996). In a considerable group of patients with serological confirmation of rubella infection, further examinations including neuroimaging and EEG presented significant brain abnormalities. CT scans revealed diffuse cerebral edema and ischemic areas in some young patients (Chaari et al., 2014). There have also been reports of a child with cerebral hernia where the primary anomalies revealed in MRI included diffusion sequences, fluid-attenuated inversion recovery, cerebral edema, as well as cortical hypersignal in T2. The EEG showed epileptic discharges without any motor convulsive activities. Knowing that the outcomes of rubella infection including basal ganglia abnormalities or central nervous system damage caused by inflammatory processes play an important role in OCD development, and corresponding to the reports about higher frequency of OCD occurrence in a group with rubella antibodies detected in blood than in a group of healthy people, the potential role of rubella on the increased risk of OCD should be considered (Lane et al., 1996; Chaari et al., 2014).

Measles Virus

Measles is a highly infectious acute disease that affects over 10 million people annually resulting in over 100,000 fatalities globally. The major route of the *Measles virus* transmission is *via* respiratory droplets. Before measles vaccinations were developed in the 1960s, the disease was the main cause of sickness and mortality among children worldwide, resulting in almost 2 million fatalities (Moss, 2017). In developing nations, measles is a major cause of blindness, particularly in people who are struggling with vitamin A deficiency (Crecelius & Burnett, 2020). Cough, fever, rash, runny nose, and conjunctivitis are typical measles symptoms (Berche, 2022). Moreover, measles could lead to some serious clinical complications including encephalitis, acute pneumonia, or otitis media (Walter & Malani, 2022). Further, besides the typical symptoms and complications of measles, this viral infection is considered to be a possible cause of an elevated risk of OCD onset. Some serious central nervous system complications associated with the measles infection such as primary measles encephalitis, measles inclusion body encephalitis (MIBE), acute post-infectious measles encephalitis (APME), or subacute sclerosing panencephalitis (SSPE) might also occur because of the *Measles virus* infection (Khanna et al., 1997; Ferren et al., 2019). Although primary measles encephalitis is a result of the currently ongoing illness, the etiology of acute post-measles encephalitis may be more complicated. Research suggests that molecular mimicry plays a role in the development of this illness. When myelin proteins typically expressed by oligodendrocytes and circulating antibodies interact, several major impairments within the central nervous system occur (Buchanan & Bonthius, 2012). Moreover, the inflammatory immune response may also be related to the adherence of infected leukocytes to the brain microvascular endothelial cells (Ajdacic-Gross et al., 2016). Another serious complication of measles infection is subacute sclerosing panencephalitis. It was discovered that there is increased intrathecal production of specific anti-measles antibodies during SSPE which can cause destabilization in usual brain functioning. MRI scans performed due to panencephalitis showed abnormalities in the grey and white matter of the brain. There have been reports revealing neurons, microglia, astrocytes, and oligodendrocytes seizure due to the measles infection (Fisher et al., 2015). Moreover, it was suggested that disturbances within the functioning of the oligodendrocytes may lead to demyelination processes. It is proposed that inflammation, nervous tissue damage, and immunological processes caused by measles infection might be associated with an increased OCD risk, especially considering the similar influence of infectious pathogens mentioned above on the OCD onset (Buchanan & Bonthius, 2012). Another aspect showing the possible influence of the *Measles virus* on OCD onset is that in patients with OCD, the anti-measles antibodies are detected more frequently than in a population of healthy people (Khanna et al., 1997).

Mumps Virus

The *Mumps virus* belongs to the negative-sense RNA, non-segmented *Paramyxoviridae* family (Rubin et al., 2015). The infection transpires when a sick person's droplets or items come into touch with contaminated secretions. Swelling and discomfort of the parotid glands are characteristic features of *Mumps virus* infection, however, it can also affect other tissues and organs (Magurano et al., 2018). Furthermore, serious negative consequences including pancreatitis, nephritis, myocarditis, orchitis, encephalitis, and meningi-

tis might result from mumps (Su et al., 2020). Fortunately, since the 1960s, when a live attenuated mumps vaccine became available, the incidence of the disease has substantially dropped in nations that have implemented mumps vaccination programs (Bogusz & Paradowska-Stankiewicz, 2022). Several research showed a relationship between mumps infection and the risk of OCD development at the same time showing a significant impact of the *Mumps virus* on the central nervous system. Although MMR vaccines are common and effective precautions against being affected, there are reports about encephalitis, meningitis, myelitis, cranial neuritis, and polyradiculitis in Mumps-infected patients (Unal et al., 2005). In some cases, mumps-acquired nervous system infection may lead to cortical blindness, focal neurological deficiency, intracranial hypertension, cerebral edema, or brainstem disorders. Moreover, approximately 10 years after primary mumps infection, there is a possibility of subacute sclerosing panencephalitis which may lead to serious nervous damage or even death (Unal et al., 2005). All the pathologies affecting the central nervous system being a consequence of a mumps infection can be visualized during neuroimaging. MRI examinations commissioned in mumps-infected patients often reveal the lesions in hippocampus or brainstem, cerebral or focal cord edema, and demyelinating regions of the brain. It was shown that all the inflammatory processes and neurological deficits caused by severe mumps infection may lead to an increased risk of OCD (Venkatesan & Murphy, 2018).

Human Immunodeficiency Virus

Human immunodeficiency virus (HIV) which causes acquired immunodeficiency syndrome (AIDS) is a global issue nowadays. HIV belongs to the *Retroviridae* family, which is made up of the genus Lentivirus and can be recognized by a significant amount of genetic variation (Fanales-Belasio et al., 2010). Currently, there are two types of HIV isolates namely HIV-type 1 (HIV-1) and HIV-type 2 (HIV-2). HIV-1 is the primary cause of AIDS worldwide, whereas HIV-2 is only found in certain parts of Central and Western Africa (Moir et al., 2011). Although the virus that triggers AIDS was finally identified in 1983, the scientific community quickly concluded that HIV had been spreading throughout the world long before AIDS was first recognized (Melikyan, 2014). The most frequent way of HIV transmission across the globe is sexual transmission, which occurs after exposure to the virus in semen or mucosal surfaces. Less common forms of transmission include transfusions of blood and its components, drug injection, or HIV infection of a fetus or a newborn through exposure to an infected mother (Beloukas et al., 2016; Nikolopoulos et al., 2016). There are several serious consequences resulting from HIV infection including some mental disorders (Lampe, 2022). Research showed a relationship between HIV infection and the onset of OCD. HIV-related stress is considered to be a major contributing factor in the development of OCD in certain patients (Bruce & Stevens, 1992; McDaniel & Johnson, 1995). HIV infection also plays an important role in neuroinflammation which might also be associated with the onset and progression of OCD symptoms. There are reports about the elevated levels of anti-myelin oligodendrocyte glycoprotein antibodies in some HIV-infected patients; furthermore, studies showed an increased rate of neurotoxins in their central nervous system. HIV is latent primarily within the microglia but may also affect neurons or astrocytes leading to chronic neuroinflammation. As a consequence, chronic inflammation associated with the activity of inflammatory cytokines such as IL-1β or TNF-α might cause further brain damage and demyelination. Research on HIV-infected patients showed white matter gliosis, astrogliosis, axonal, and myelin abnormalities. MRI scans also presented the lesions in white matter and focal hyperactivity in different areas of the brain. Summing up chronic neuroinflammation, abnormalities in the central nervous system functions, and brain damage are common features of HIV infection (Everall et al., 2005; Hlebowicz et al., 2019; Sreeram et al., 2022). Current evidence suggests a noteworthy association between HIV infection and OCD onset but further research on this matter is needed (Jin et al., 2010).

Borna Disease Virus

The *Borna disease virus* (BDV) is an example of the negative-strand RNA non-segmented viral family *Bornaviridae*, genus bornavirus (Ludwig & Bode, 2000). Its molecular structure and ability to generate noncytolytic, sustained central nervous system infection in a broad range of host species set it apart from other animal RNA viruses (Carbone et al., 2001). Considering the biological characteristics, molecular biology, and morphology of the virus were clarified, there was a sharp rise in interest in the disease in the early nineties (Taieb et al., 2001). Initial indications suggested that BDV infection could result in mild behavioral disorders due to its influence on the structure of the central nervous system, its functions, as well as development (Jordan & Lipkin, 2001; Hornig et al., 2003). It was suggested that modifications in the neural correlates of neural processing of information that are linked to BDV infection may induce the onset and progression of OCD (Teixeira et al., 2014). It was observed that BDV

antigen can activate T lymphocytes, which in response increases the synthesis of inflammatory cytokines. Studies show the striato-thalamo-cortical connections hyperactivity and suggest that BDV infection might affect it by interfering with neurotransmitter function (including functions of GABA, aspartate, and glutamate) of the viral elements (Rauch et al., 1997; 2001; Dietrich et al., 2005). By raising glutamate levels, antigens may stimulate activity in such brain regions as the anterior cingulate cortex through the thalamocortical pathways. This could lead to increased activity and potentially excitotoxic neurodegeneration. This process might also affect the ventro-anterior thalamic nucleus which due to the connections to the crucial structures can determine the symptoms of OCD; also, the anterior cingulate cortex and orbitofrontal cortex might be affected (Rotge et al., 2010). Moreover, the influence of BDV on the hippocampus and synaptic conduction can result in memory dysfunctions that play a significant role in cognitive dysfunctions in OCD (Robbins et al., 2019; Zhang et al., 2022).

Bacteria and OCD

Mycoplasma pneumoniae

Humans may encounter numerous struggles due to the intracellular bacteria *Mycoplasma pneumoniae* (Atkinson et al., 2008). The genus Mycoplasma, which is a member of the class Mollicutes, belongs to the Gram-positive bacterial group (Chaudhry et al., 2016). It was first identified in the course of the 1940s that atypical pneumonia could be triggered by *Mycoplasma pneumoniae*. Community-acquired pneumonia (CAP) is the illness caused by *M. pneumoniae* in a group of people of all ages (de Groot et al., 2017; Naghib et al., 2018). The fact that *M. pneumoniae* can affect the nervous system besides being the respiratory tract pathogen is crucial in its impact on OCD development. The possible pathomechanisms of the elevated risk of OCD in a patient with *M. pneumoniae* infection are considered to be multifactorial (Ercan et al., 2008). The inflammatory processes due to encephalitis or encephalomyelitis could lead to serious nervous tissue damage including thalamic necrosis. MRI examinations commissioned in *Mycoplasma pneumoniae*-infected demonstrated significant cortical swelling, hyperactivity in temporal and frontal lobes, and subcortical white matter (Krzyściak et al., 2013; Gerentes et al., 2019). All impairments in the nervous system may lead to the onset of various psychiatric disorders including OCD. Another theory takes into consideration the autoimmune processes. Some case reports showed that specific antibodies against neurons found in the blood samples of *M. pneumoniae*-infected patients included anti-dopamine D1 receptor, anti-dopamine D2 receptor, anti-tubulin, and anti-lysoganglioside antibodies. These antibodies might lead to various basal ganglia dysfunctions, which play a significant role in OCD development (Kim et al., 2016). Moreover, some research showed that the *Mycoplasma pneumoniae* cell membrane consists of certain lipoproteins that are thought to be powerful cytokine inducers. These lipoproteins might trigger autoimmunity due to their molecularly mimicking different structures of human cells, especially in the brain (Maness et al,. 2021; Meyer, 2021). T helper lymphocytes generate cytokines such as IFN-γ or IL-17A, that stimulate microglia and impair the blood-brain barrier, allowing migration of antibodies into the brain and causing, for instance, neurotransmitter dysfunctions. Considering all the neurological dysfunctions caused by *M. pneumoniae*, this pathogen might be considered one of the important trigger factors of OCD (Lannes-Costa et al., 2021; Endres et al., 2022).

The genus Streptococcus

The genus Streptococcus which is an element of many species' microbiota, includes Gram-positive cocci, part of the *Streptococcaceae* family (Kim et al., 2004). Over one hundred species of streptococcus can be distinguished; *Streptococcus pneumoniae*, *Streptococcus pyogenes*, *Streptococcus agalactiae*, or *Streptococcus canis* are some of the best known of the genus (Martino et al., 2009). Although these pathogens are usually associated with pyogenic or pulmonary infections, they might also play a worth-mentioning role in the onset of OCD (Mehta et al., 2020). It is generally known that autoimmune processes play an important role in OCD development. Abnormalities related to streptococcal infections including PANDAS result in neuronal damage and irreversible changes in the functioning of the central nervous system. An autoimmune reaction may be triggered by ß-haemolytic streptococcus infections and the resulting generation of anti-streptococcal antibodies such as anti-DNase B antibodies or anti-streptolysin O antibodies. Molecular mimicry is a process where human antibodies against Streptococcus pyogenes cross-react with distinct basal ganglia receptors. PANDAS patients also present the overactivation of calcium calmodulin-dependent protein kinase II (CaMKII) which affects dopamine secretion levels and may interrupt neurotransmitter levels (Yiş et al., 2008; Kurlan et al., 200). Moreover, anti-D1/D2R antibodies that are often detected in groups of streptococcus-infected patients cause dopamine receptor internalization and lead to further neurotransmitter dysregulation (Bitnun

et al., 2003). There are also reports of acetylcholine secretion abnormalities, anti-lysoganglioside, anti-pyruvate kinase, and anti-tubulin antibodies elevated levels due to streptococcus infections (Okumura et al., 2018). Furthermore, recent research showed an increasing number in the subpopulation of B lymphocytes that are D8/17 positive and may be associated with hereditary variables in streptococcus-infected groups of people (Freeman et al., 2021). This factor might correlate with an increased risk of PANDAS occurrence. The mimicry, increased levels of proinflammatory cytokines, and dysregulation in enzymes and neurotransmitter function may result in serious central nervous system complications including encephalitis. Neuroimaging of patients revealed extended basal ganglia and striatal volumes and their hyperactivity, lentiform and caudate nucleus lesions, expanded putamen, caudate and *globus pallidus* dimensions, and gliosis (Baj et al., 2020; Piras et al., 2020).

All the impairments in the central nervous system including basal ganglia disorders, neurotransmitter level abnormalities, or influence on cortico-striato-thalamo-cortical circuits are crucial for the onset and progression of OCD (Hsu et al., 2022).

Gut Microbiota

The gut microbiota is an essential component of the human body that adapts over time to adapt to its hosts (Milani et al., 2017). It interacts with the host in a variety of ways that impact functioning throughout the host's lifetime. It affects the host's immunity, metabolism, and well-being in general (Adak & Khan, 2019; Ma et al., 2022). An interesting fact may be that the microbiota of humans' digestive systems corresponds with the potential possibility of OCD occurrence. It has been suggested that the gut microbiota plays an essential part in the development of the brain, myelination processes, and complex nervous functions due to the influence on neuronal and oligodendrocyte development (Rees, 2014). What is crucial considering the relationship between the gut microbiota and OCD is the gut-brain axis. The gut-brain axis is a relationship that exists between the gut microbiota and the brain including immunological, hormonal, metabolic, and neurological processes (Marazziti et al., 2021). The modulate features of gut microbiota play an important role in inflammatory processes which are one of the factors of increased OCD risk. Dysregulations in gut microbiota may lead to the increased concentration of short-chain fatty acids (SCFA) such as propionate, acetate, and butyrate which may stimulate the microglia, resulting in a cytokine burst. In some cases, such inflammatory factors ejection could disrupt the blood-brain barrier causing brain damage (Turna et al., 2016; Troyer et al., 2021). Moreover, there have also been reports about the possible regulatory influence of the gut microbiota on brain function discovered in mice *via* neuronal transcription and gene expression. Such activity might impact the neurotransmission processes, intensity of inflammation, or hormonal homeostasis. The gut microbiota also regulates the function of the hypothalamic-pituitary-adrenal axis affects the stress level and plays a potential role in OCD development (Kamble et al., 2023). Moreover, it modifies neurotransmitters like dopamine, GABA, serotonin, and glutamate. Neurotransmitter abnormalities are considered one of the pathological attributes of OCD. Another interesting fact may be the potential therapeutic effect of probiotic supplementation on mental and neurological state improvement (Zang et al., 2023). Recent studies revealed that *Lactobacillus* and *Bifidobacterium* intake can improve cognition processes in experimental groups of different animals (Bendriss et al., 2023).

Parasites and OCD

Toxoplasma gondii

An intracellular parasite of worldwide significance, *Toxoplasma gondii* can remarkably multiply, prosper, and attack a significant number of cells (Liu et al., 2015). Its special ability is that *T. gondii* can survive for a long time in the central nervous system of different hosts. The majority of *T. gondii* infections in humans are relatively untroubled or even asymptomatic, but congenital infections can cause a severe condition in foetuses or immunocompromised people (Mendez & Koshy, 2017). The disease primarily spreads by drinking tainted water or eating raw or undercooked meat that contains living cysts (Lima & Lodoen, 2019). Due to its extraordinary affinity to brain tissues, *Toxoplasma gondii* induces peripheral immune cell activation, inflammatory mediators, and stimulation of microglial cells (Miman et al., 2010). The presence of *T. gondii* or its cysts appears to be dispersed throughout a remarkably broad range of brain areas causing immunological activation and promoting inflammation, which may be the cause of neurodegeneration (Flegr & Horáček, 2017). There are some cases revealing lesions found in basal ganglia in neuroimaging of patients with toxoplasmosis. Considering that basal ganglia abnormalities play a significant role in OCD development, more attention should be paid to neurotoxoplasmosis (Miman et al., 2018; Nayeri Chegeni et al., 2019). Furthermore, reports indicate the possibility of dopamine level modification and increased cytokines production caused

by *T. gondii*. The presence and function of tyrosine hydroxylase enzymes (AHH 1 and 2) and the ability to secrete interleukins (IL-2) in the *T. gondii* genetic material may lead to considerable neurological disturbances by encoding proteins that generate L-DOPA. L-DOPA may induce fluctuations in the central nervous system by the interference with synaptic transmission of dopamine (Baker et al., 2020). Other neurotransmitters which secretion may be affected by *T. gondii* infection are noradrenaline, glutamate, GABA, kynurenic acid, serotonin, or nitric oxide. All the processes overlap on the central nervous system dysfunctions, being an important factor in OCD development (Nayeri Chegeni et al., 2019). Moreover, OCD may be caused by anomalies in the hypothalamic-pituitary-adrenal gland axis brought on by immunological responses or hormonal imbalances due to the *T. gondii* infection (Virus et al., 2021). A subsequent argument for the correlation between the parasite infection and an increased OCD risk is that the presence of *T. gondii* IgG antibodies was significantly more often observed in a group of OCD-suffering patients than in a healthy population (Akaltun et al., 2018).

Other pathogens

Some research showed several cases concerning *Epstein-Barr virus* (EBV) infection or *Cytomegalovirus* infection as possible risk factors for OCD but the current knowledge does not allow for formulating strict relationships between the abovementioned pathogens and OCD (Caruso et al., 2000; Hultman et al., 2023). Another correlation may appear in the case of Borrelia Burgdorferi infection and OCD occurrence. However, similar to the abovementioned viruses, the possible mechanism of this interaction has not yet been found (Fallon & Nields, 1994). Even though the literature search indicated some information regarding the possible relationship between OCD and *Influenza virus*, *Coxsackie virus*, *Poliovirus*, *Parvovirus B19*, *Enterovirus 71*, *West Nile virus*, *Treponema pallidum*, or *Toxocara*, there is still a significant lack of data to provide any assumptions and conclusions in this matter (Fallon & Nields, 1994).

DISCUSSION

All the information gathered in this narrative review presents an interesting correlation between some infectious diseases and an increased risk of OCD. According to the research devoted to HSV infections, all the inflammatory processes during illness might significantly affect the central nervous system leading to the alterations that might be associated with the onset and/or progression of the OCD symptoms. As it was proven in different studies, pathological reactions that concern the nervous system and lead to irreversible lesions in its structure and functions, play an important role in OCD development. One of many possible pathomechanisms of OCD onset is inflammatory activities in the central nervous system. Encephalitis associated with the HSV infection may result in neuronal tissue injury. What is also crucial, periodic reactivation of the latent chronic HSV infection and organism changes caused by stress, endocrine disruption, or any other abnormality that could contribute to latent HSV infection, may also result in neuronal damage. All the above mentioned aspects can be a trigger of OCD. Rubella infection may also be a trigger factor for OCD occurrence. Central nervous system changes such as white matter abnormalities, calcifications and smaller intracranial volume presented in neuroimaging examinations suggest serious complications and brain damage in a group of Rubella-infected people. As it was mentioned above, inflammatory processes and neuronal abnormalities are important components of OCD onset and progression.

In relation to HIV infection and an increased risk of OCD, the possible correlations might be various. First of all, HIV-related stress may potentially be a major contributing factor in the development of OCD in certain patients. Secondly, chronic inflammation related to the activity of inflammatory cytokines might cause brain damage and demyelination. Moreover, recent studies revealed elevated levels of anti-myelin oligodendrocyte glycoprotein antibodies in this group of patients which may eventually lead to neurodegeneration processes.

One of the characteristic features associated with the infection by *Borna disease virus* is modifications in the neural processing of information. Research highlights the hyperactivity of the striato-thalamo-cortical connections and implies that BDV infection may impact it by disrupting the neurotransmitter function (including aspartate and glutamate functions) of the viral components. Furthermore, the alterations in the hippocampus and synaptic conduction may induce memory problems, which are a major contributor to the cognitive problems associated with OCD. Regarding bacterial infections, *Mycoplasma pneumoniae* can cause encephalitis which results in nervous tissue damage. Besides inflammatory factors, *M. pneumoniae* infections could contribute to autoimmune processes including pathological changes in the brainstem, cerebellum, and basal ganglia. All the lesions may be a significant factor in OCD onset and progression.

Another important group of infections that might be associated with an increased risk of OCD include streptococcal infections. Streptococcal infections may result in neuronal damage and irreversible changes in the central nervous system functions such as gliosis which is a fibrous glial formation in the CNS regions of injury. Moreover, studies concerning the correlation between OCD and streptococcal infections pay attention to the increased levels of anti-basal ganglia antibodies in the blood samples of patients with streptococcal infections. Defects of the basal ganglia possess significance for the onset of OCD symptoms.

Research also showed a significant impact of gut microbiota on OCD development. Gut microbiota plays an essential part in the development of the brain and complex nervous processes and the formation of the brain due to the influence on neuronal and oligodendrocyte development. Other important arguments, that may prove such a correlation are modification abilities on neuroinflammatory processes, neurotransmitter levels, and the hypothalamic-pituitary-adrenal axis. All the elements are important features in OCD development. *Toxoplasma gondii* being a parasite is considered to be one of the important risk factors for possible OCD occurrence. Due to its significant affinity to the neuronal tissue, *Toxoplasma gondii* induces peripheral immune cell activation, inflammatory mediators, and stimulation of microglial cells.

Infections caused by *Varicella zoster virus*, *Measles virus,* or *Mumps virus* are also considered to contribute to the increased risk of OCD development. Research revealed a remarkable relationship between more frequent OCD occurrence and patients infected by the aforementioned viruses. It was shown that OCD develops more often in a group of people with increased levels of antibodies against *VZV*, as well as the *Measles* and *Mumps viruses* in the blood rather than in a group of healthy people. The possible correlation may be associated with neuroinflammatory processes but in order to prove this hypothesis further studies are needed.

With regards to other pathogens that also were included in the research, there are some case reports about *Epstein-Barr virus*, *Cytomegalovirus,* or *Borrelia Burgdorferi* infections and their possible influence on an increased OCD risk. However, there is not enough available information to formulate such a statement and discover the exact processes involved in this correlation. Further research is needed to elaborate on this matter. Similarly, there is no evidence of the possible influence of *Influenza virus*, *Coxsackie virus*, *Poliovirus*, *Parvovirus B19*, *Enterovirus 71*, *West Nile virus*, *Treponema pallidum*, or *Toxocara* infections on OCD development.

CONCLUSIONS

Different types of infection including viral, bacterial, or parasite infections may be an important factor in OCD onset or progression. Abnormalities that have been observed during infections mentioned above such as neuroinflammation, neuronal tissue damage and demyelination, autoimmunological processes, endocrine disruption, or impairment in neuronal connections and neurotransmitter functions are important factors in OCD increased development risk. Research showed that there is a correlation between different types of infections and elevated number of OCD onset. However, the exact pathomechanisms have not been proven yet. Further research and studies are indispensable for a better understanding of an infectious aspect of OCD.

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