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Review Article

Viral Fingerprint Effect on Human Behavior

Noura B. Abdulrahman[1*]

[1]Biology Department, College of Science, Tikrit University, Tikrit, Saladin, Iraq

***Corresponding Author:** Noura B. Abdulrahman
Biology Department, College of Science, Tikrit University, Tikrit, Saladin, Iraq

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Abstract: Viral infections can have long-lasting immunological and neurological fingerprints, or so-called virial fingerprints. These effects are seen in behavioral changes, cognitive impairments and neurological disorders. This review provides a synthesis of the present state of the art focused on current evidence for how viruses interfere with the central nervous system (CNS) in terms of blood-brain barrier (BBB) integrity, neuro-inflammation, neurotransmitter imbalances and vascular dysfunction. It also covers the clinical manifestations of brain fog, anxiety, and migraine and focus on management strategies for post-viral syndromes.

Keywords: Neuro-virology, Central Nervous System (CNS), Blood-Brain Barrier (BBB), Neuro-inflammation, Viral Fingerprints, Post-Viral Syndromes.

INTRODUCTION

Patients who suffered from viral infections may show persistent symptoms who comprise spasm disorders, behavioral hitches, sleeping disorders, regular headache, immune troubles and motor incapacities (Michaeli *et al*., 2014). Numerous signs of neurocognitive impairment had also been informed as sequelae of viral encephalitis, as attention-deficit/hyperactivity disorder (ADHD), memory and learning disorders and finally speech disorder which completely known as “Brain Fog” (Michaeli *et al*., 2014; Huang *et al*., 2006; Pöyhönen *et al*., 2021). In this object, it will be debated most of the mechanisms that stated up to the present time which would be employed by such viruses to stretch brain and in what way they influence the probity of the blood-brain barrier (BBB). Besides that, it would be described the universal immune activity in general and the immune response that happened at CNS by infections with those viruses. They’re the understanding keys to realize that most of these viral pathogens primarily, encourage a general infection; consequently, they would stretch the CNS. These article information will present emphasis on the viruses influence on CNS beside systemic response. At the end, the long-term sequel of infection by which viruses would be described.

Post Viral Effect on Human Immunity

It is charming and a bit worrying how a virus can act as the "starting gun" for the immune system to turn against itself. Since we are looking at examples beyond the most common ones like HIV, we can see how everyday viruses like the common cold or the stomach flu are linked to long term autoimmune conditions (Jane E. Libbey M.S., Robert 2002).

The development of these diseases is usually a "multi-hit" process. A virus alone rarely causes autoimmunity, and it is usually a combination of the genetic susceptibility (in this case, specific HLA genes), environmental factors (such as smoking or vitamin D deficiency), and the severity of the initial viral infection. For instance, children with severe cases of Covid-19 had been showed to have an expressively greater possibility of developing new-onset autoimmune issues compared to people with mild; an example: Multiple Sclerosis Virus (MSV) and Epstein-Barr Virus (EBV). The two mentioned viruses can cause mononucleosis ("Mono"), is now thought to be the leading trigger for Multiple Sclerosis.

It was found that “of 10 million military personnel found that the risk of MS increased 32 fold after infection with EBV”. This result by attacking the immune system a protein in the virus, that looks almost identical to myelin, which is the protective coating around your nerves (Bjornevik, K., *et al*., 2022; Lanz, T. V., *et al*., 2022; Roe 2025).

Another case is Coxsackie-virus B and Type 1 Diabetes, the Coxsackievirus is a common "stomach flu" or hand foot and mouth disease virus. Research suggests that certain enteroviruses can infect the beta cells in the pancreas and instead of just getting rid of the virus. The insulin-producing beta cells became as a target of immune system so they could be destroyed, producing Type 1 Diabetes initiation, frequently appears in childhood (Yeung, W. C., *et al*., 2011).

The third example is the case of the virus known as the Coronavirus-2 (covid-19) and Rheumatoid Arthritis / Lupus, there is recent data that shows that there is a drastic increase in "new-onset" systemic autoimmune diseases after covid-19. The infected people are being detected as “Rheumatoid Arthritis and Systemic Lupus Erythematosus (SLE) months after recovering”; intense of "cytokine storm" that takes place during a severe infection can break the body's "immune tolerance" causing the body system to stop recognizing its own tissues (Chang., *et al*., 2023; Liu, Y., *et al*., 2021)

Some Viruses Can Pass BBB

As it known, encephalitis that’s caused by viral infection is the most public foundation of neuropathology (Chen *et al*., 2020). Numerous viruses were labelled as causes resulting in encephalitis. They are including: rhabdoviruses, arbovirus, enterovirus, herpesvirus, retroviruses, orthopneumovirus, orthomyxoviruses and coronavirus (SARS-CoV-2 together) (Rozenberg, 2013; Stahl and Mailles, 2019; Yasmeen J. Al-bayaa *et al*., 2022). These infections are resulting in inducing irritation and damage to the brain causing encephalitis, whom could be achieved by recognizing viral elements or their antigens in the CNS (Beattie *et al.,* 2013; Al-Obaidi *et al*., 2018; Chen and Li, 2021). The common way to reach the brain by those viruses and so, infect the brain is by disrupting the blood-brain barrier BBB (Spindler and Hsu, 2012; Bohmwald *et al*., 2021b; Thomsen *et al*., 2021; Yasmeen J. Al-bayaa et al., 2022). This barrier is a physical obstruction in the interface amongst the circulatory system, CNS, immune system and the relaxation of the organism. It is found in the whole of vertebrates, owns a very controlled permeability, and is responsible for keeping the microenvironment of the brain a part from microorganisms (*Jayaraman et al., 2018*). The normal healthy BBB can protect the brain and neurons from several agents that are circulating in the blood which may cause damage to them, like microorganisms, toxins, antibodies and finally, immune cells (Bohmwald *et al*., Chen and Li, 2021). “This barrier is mainly composed of a monolayer of brain microvascular endothelial cells found along the vascular tree [mostly kept together by tight junctions (TJ)], pericytes, and astrocytes (giving structural support to this structure) (Nikolakopoulou *et al*., 2019; Chen and Li, 2021)”. Inside the BBB, TJs function is as extend that controls transmembrane activity, it’s the most part of the barrier that has a selective permeability, so it protect the brain from the foreign and hurter organism (Nikolakopoulou *et al*., 2019).

Brain Fog and Anxiety Are Common Components of Post-Viral Syndrome

A condition in which symptoms persist for weeks or months after an initial infection has subsided. Brain fog is not an actual medical diagnosis but rather a descriptive term for cognitive impairments such as poor concentration, lapsing memory and mental "cloudiness" (Loizidou *et al*., 2026).

Biological Mechanisms

Viruses can cause these symptoms in several ways which affect the central nervous system:

1. **Neuro-inflammation:** The immune system is in a response to tie the virus by production of proteins termed cytokines. When this response still hyperactive after the virus is away of the body, it will lead to persistent inflammation in the brain, that interfering with the communication between neurons. Microglial motivation and releasing of cytokines (e.g., IL-6, TNF-alpha) have synergistic effect on synaptic dysfunction, neuronal harm and activation of inflammasomes (such as NLRP3), whom then enhances irritation in the CNS. The initiation of the inflammation elements as: NLRP3 (NOD-like receptor family pyrin domain containing 3) inflammasome besides caspase-1 prime releasing of proinflammatory cytokines, like: interleukin-1 beta (IL-1v), interleukin-6 (IL-6) and tumor necrosis factor alpha (TNFv). Such cytokines resulting in vascular permeability and proangiogenic factors for example: vascular endothelial growth factor A (VEGF-A) which upset the blood-brain barrier (BBB) and modify blood vessel homeostasis. This cascade helps microglial activation and involves in neurovascular dysfunction and in the end leads to (CNS) compromise in case of infection by Covid virus (Kanberg, 2020; Freeman T.L., Swartz T.H., 2020; DeOre *et al*., 2021; Wei Z.D., *et al*., 2023; *et al*., 2022).
2. **Blood-Brain Barrier (BBB) Disturbance:** The inflammatory responses can result in leaking blood-brain barrier and so, make it more permeable, letting immune cells and\or toxins to enter the brain and that gives extra damage the brain (Chen and Li, 2021).
3. **Neurotransmitter Imbalance:** Viral infections may disrupt chemicals which regulate mood and thinking, such as serotonin and dopamine. Low serotonin levels are associated specifically with both the mood disturbances (anxiety/depression) and cognitive slowing suffered under Long Covid (Wong, 2023). Long Covid has been highly associated to serotonin decline. Research has shown that the occurrence of the virus in the gut can cause inflammation, which reduces the absorption of tryptophan (the precursor molecule that is essential to produce serotonin). This led to reduce the circulating of serotonin levels, which compromises memory, learning and mood control. Low serotonin is directly linked to depression, anxiety and cognitive slowing "brain fog" in Long Covid patients (Harris, 2023).

In case of dopamine disruption, Sarco virus 2 can interfere with the dopamine signaling that is a special case compared to other respiratory viruses like influenza. Dopamine is crucial in reward processing, motivation and executive function. Damage to dopamine pathways plays a role in lack of energy, decreased motivation and poor cognitive function in Long Covid patients (Wong, 2023).

Automatic Pathways (Jun Zhue al 2026 and Currey *et al*., 2026):

1. **Gut-Brain Axis:** Gut viruses modify gut tryptophan metabolism and decreased serotonin production.
2. **Inflammatory Signaling:** Viral RNA-induced Type I interferons suppress serotonin uptake and lead to hypercoagulability, which additionally disrupts neurotransmitter homeostasis.
3. **Vascular Injury:** COVID-19 induces microvascular brain damage that interferes with the serotonin and dopamine transmission, and this fact supports the alleged presence of chronic cognitive and mood symptoms. Only an imbalance between serotonin and dopamine can explain the hallmark symptoms of Long COVID (Wong, 2023; Jun Zhue al 2026 and Currey *et al*., 2026):
   - **Mood changes:** Anxiety and depression because of serotonin loss.
   - **Cognitive slowing:** Disrupted executive, serotonin, and dopamine signaling lead to impaired memory, attention and executive functioning.
   - **Weakness and loss of motivation:** The dysfunction of the dopamine pathways decreases energy and motivation.
   - **Vascular Problems and Hypoxia:** There are viruses that result in micro-clots or damage to blood vessel linings (endothelial dysfunction), which limits oxygen and blood flow to parts of the brain that regulate memory and executive functions (Prasad *et al*., 2021).

The Mechanism of Virus-Induced Migraine

Clinically, numerous of viral infections are associated in migraine by triggering it or generating eternal alterations in the frequency and intensity of migraine. Although most diseases trigger general "secondary" headaches, there are several viruses that directly interact with the same pathways of neurological imitation that triggers primary migraine disorders (Yamanaka *et al*., 2023). Viruses affect the pain systems of the brain in a number of biological ways:

- **Neuro-inflammation:** Many of pro-inflammatory cytokines (such as TNF-α and IL-6) that can enter into the immune system have a direct effect of sensitizing the nerve edges in the brain (related with pain) (Kursun *et al*., 2021; Yamanaka *et al*., 2023).
- **The Trigeminal System Activation:** Migraine pain is transmitted mostly by effecting on the trigeminal nerve. A direct target of viruses like SARS-CoV-2 is often nasal cavity, subsequently ongoing irritation of these nerve edges (Murphy *et al*., 2025; ProBiologists 2025 Huang *et al*., 2026).
- **Vascular Effects:** The vascular inflammation which is lining of blood vessels (endothelial cells) that occurs thru a viral infection can be a reason to release CGRP (calcitonin gene-related peptide), whom a major component that dilates blood vessels and roots pain during a migraine (Al-Mahdi *et al*., 2023; Boldig, K., and Butala, N. 2023; Practical Neurology 2025).
- **Secondary Stressors:** Effects that are in the establishment of a viral disease are high fever, sleep deprivation, dehydration, and physical stress, all of which are well-known migraine triggers (Doherty 2025).

Viruses Commonly Associated with Brain Fog and Migraine

- **SARS-CoV-2 (COVID-19):** This is the virus that is narrowly linked with the symptoms of the brain fog and anxiety (Kverno K. 2024). It also aggravates migraines already existing. It has been found that about 60% of migraine patients had more frequency and intensity of attack post infection. It is also capable of causing a phenomenon of a migraine in individuals who have not previously had the disorder Huang *et al*., 2026; Murphy *et al*., 2025).
- **Epstein - Barr virus (EBV):** EBV is well-known in producing mononucleosis mono; it is also recognized to be a famous reason of chronic and incurable headache. Research has exposed that chronic primary migraine is strongly associated to consuming gone by EBV infection in the past. It is the famed cause of the long-term post-viral fatigue and brain malfunction (Kverno, 2024).
- **Herpes Simplex Virus (HSV-1):** This is a virus that is believed to be dormant in nerve cells and can trigger inflammations when reinforced. There is some evidence that HSV-1 has a potential of being dormant in the trigeminal ganglion, one of the critical nerve clusters of migraine pain, and its reactivation is possibly a potential trigger of attacks (Chen *et al*., 2020; Michaeli *et al.,* 2014).
- **Viruses of Influenza and Common Cold:** Systemic inflammation caused by severe strains of the flu may result in persistent neurological symptoms (Freeman & Swartz, 2020). These systemic infections usually lessen the migraine threshold, it means that a person converts more vulnerable to its usual triggers during and after the disease.

Management and Recovery Strategies

Post-viral brain fog cannot be treated only in one way, but the following are the methods that may assist:

1. **Pacing:** Planned rest and task organization and eliminating overwork by dividing the task into smaller steps and taking regular rests (Michaeli *et al*., 2014).
2. **Cognitive Rehabilitation:** The exercises that focus and memorize (Poyhonen *et al*., 2021).
3. **Lifestyle Modifications:** Anti-inflammatory diet, healthy sleeping, and light exercise (Freeman and Swartz, 2020).
4. **Psychological Resources:** CBT and mindfulness to reduction anxiety and enhance clarity (Kverno, 2024).
5. **Migraine Therapies:** The use of Triptans and anti-CGRP as post-viral migraine treatment (Chen *et al*., 2020; Huang *et al*., 2006; Poyhonen *et al*., 2021).

CONCLUSION

Viruses cause permanent fingerprints on the human body and behavior by interfering with the CNS with neuroinflammation, BBB damages, and neurotransmitter imbalance. These effects are expressed in brain fog, anxiety and migraine which consume a major effect on the quality of life. In spite of the fact that there is no clear cure, pacing, lifestyle interventions, and specific therapies present good prospects of recovery. Further studies in the viral neuropathology of the virus are necessary to come up with effective treatment. COVID, SARS-CoV-2, Epstein -Bar virus Herpes Simplex Virus, and Influenza virus are the most contributing viruses in the elucidated symptoms.

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