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Mechanism Of Action Of Covid 19 And Its Manifestations

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Abstract

Various Neurological Manifestation associated with severe acute respiratory syndrome (SARS- CoV-2) has been recorded ranging from mild to severe symptoms and syndromes. An intense review of all the current published literature was conducted and found that headache, psychomotor agitation, taste and smell abnormalities, generalized muscle weakness, and Myalgia were the common neurological manifestation of SARS CoV-2. However, Seizures, stroke, flaccid paralysis, and Guillain-Barre syndrome were some of the rarely found cases among COVID 19 patients. the SARS CoV-2 virus has been found in over a hundred countries across the world. In March 2020, the World Health Organization declared a pandemic after receiving reports of more than 700,000 deaths worldwide (WHO). While first believed to be limited in its effects, the virus soon progressed to other organs, including the central nervous system (CNS), leading to symptoms such as paralysis and death. For a long time, it was thought that viral entrance into host cells through the receptor ACE2 was the key determinant of the S protein of CoV-2 linked to the SARS membrane.

Keywords: COVID-19, Alzheimer's disease, Guillain-Barre.

Introduction

In late December 2019, the SARS CoV-2 virus has been found in over a hundred countries across the world. In March 2020, the World Health Organization declared a pandemic after receiving reports of more than 700,000 deaths worldwide (WHO). While first believed to be limited in its effects, the virus soon progressed to other organs, including the central nervous system (CNS), leading to symptoms such as paralysis and death (Boukhris et al., 2020**)**. Neurological symptoms, including severe cerebrovascular illness and decreased awareness, were seen in 36.4 percent of the 214 COVID-19 patients . More recent studies are also looking at the neurological effects of COVID-19.

A cold or the flu pales in comparison to what's going on here. To arrive at a diagnosis, many pathophysiological and hematological issues are being studied. Here If a virus can latch to a spike protein, it can penetrate cells. Enzyme Natriuretic Enzyme 2 [ACE2] is crucial in this procedure ACE-2 expression has an impact on both the pathogenesis of COVID-19 and the tropism of the virus (Crews et al., 2017). Cells in the brain that express the ACE-2 gene include neurons as well as glia, endothelial cells, and arterial smooth muscle cells. ACE-2 is also detected in the temporal lobe and the hippocampus, which are linked to Alzheimer's disease (AD) (Hassan et al., 2020). SARS-CoV-2 may cause demyelination of the CNS, neurodegeneration, and cellular necrosis as a consequence of the host's immune response to the virus. These conditions may worsen Alzheimer's disease and other kinds of dementia, as well (Hassan et al., 2020). Long-term consequences of premature aging and neurological diseases after SARS-CoV-2 infections are unknown. In those with Alzheimer's illness, SARS-CoV-2 may aggravate their symptoms.

Pathophysiology of COVID 19 infection

For a long time, it was thought that viral entrance into host cells through the receptor ACE2 was the key determinant of the S protein of CoV-2 linked to the SARS membrane. In research, TMPRSS2 proteolytic enzymes release viral RNA at S20 of SARS-CoV-2 protein, which causes membrane fusion and viral infectivity, according to the findings. Viral RNA endocytosis, whether clathrin-dependent or clathrin-independent, has been proven to play a role in viral RNA entrance into the host cell. By creating two structural proteins and polyproteins upon cytoplasmic release, this enzyme helps to speed up viral

replication in the body (Eswaran et al., 2020). It is the quick and synchronized innate and adaptive immune responses that initiate antiviral immunity in host cells after SARS-CoV-2 genome entry. Viral RNA-associated molecular patterns are recognized by Toll-like receptors (TLRs), RIG-I/MDA5, and CRP-type PRRs, including Mannose-binding lectin (MBL) and CRP (PAMPs). Type I interferon activates the body's innate and adaptive immune systems (IFN).

Three antiviral signaling molecules are involved in the activation of interferon genes when a virus is recognized: MAVS, IFN- (TRIF), and the activator of interferon genes (STING). MyD88 and other adaptor molecules like it play a critical role in the downstream cascade. Through this interaction, NFB and IRF3 are activated, allowing for the transport of DNA into the nucleus more easily. IFN-/ and other pro-inflammatory cytokines, including IL-6, are produced in the nucleus by these transcription factors (Ebate et al., 2020). A variety of first-line defenses are triggered by contact between the virus and the host cell. As a result of type I IFNs, the JAK/STAT pathway is activated and IFN-stimulated genes (ISGs) are transcribed (ISRE). This may inhibit viral multiplication and phagocytosis by macrophages and NK cell-mediated limitation of infected cells by increasing type I IFN concentrations. To ensure that the virus can survive in the host cell, the JAK-STAT signaling pathway must be disrupted, as well as macrophage expression.

Adaptive immunity to viral infections is largely believed to rely heavily on the Th1-mediated immune response. Antigen-presenting cells (APCs) play a key role in T-cell responses helped T cells (CD4+) produce a molecular cluster that comprises granzymes, perforin, and IFN- to aid cytotoxic T cells in eliminating virus-infected cells. Infection-fighting antibodies are produced by humoral immune responses triggered by B cells. COVID-19 individuals had high levels of interleukins (IL-1, IL-2, and other ILs, IL-4, and others), IP-10, and macrophage colony-stimulating factor (MCSF) MCP-1 and GCSF hepatocyte growth factor (HGF), IFN-, and mRNA and TNF-, which signals an escalation in the illness. "Lympopenia" and "cytokine storms" may have an impact on COVID-19's pathogenesis (Zimmerman and Martina, 2017). Cytokine storms may cause necrosis or death of T cells in cancer and other persistent infections.

stem's capacity to combat the illness. Viral sepsis and inflammation-induced lung damage may cause further consequences, such as acute respiratory distress syndrome (ARDS), necrotizing fasciitis (NF), and pneumonia. There is a "cytokine storm" to blame for these symptoms. Eosinophil, monocyte, and basophil counts are significantly decreased in those with severe IgE deficiency. The COVID (19th in the series) (Fig 1 and 2) Coughing and sneezing are the primary routes through which the SARS CoV-2 virus enters the body of an infected person. When the S1 and S2 spike proteins fuse with the alveolar cell's ACE 2 receptor, the enzyme Transmembrane Serine Protease is required to break down them (2) (Ebate et al., 2020). (TMPRSS2). Starting with ribonucleic acid, two ORFs (1a and 1b) are synthesized, and they are then used to make structural and nonstructural proteins in the cell. It is the Golgi complex that transports viral proteins to the endoplasmic reticulum intermediate complex (ERGIC) (ERGIC). Following exocytosis and ejection, virus cells are expelled from the host body. T cells are infected by macrophages, which activate B and T helper cells. As a consequence, the virus affects the whole immune system.

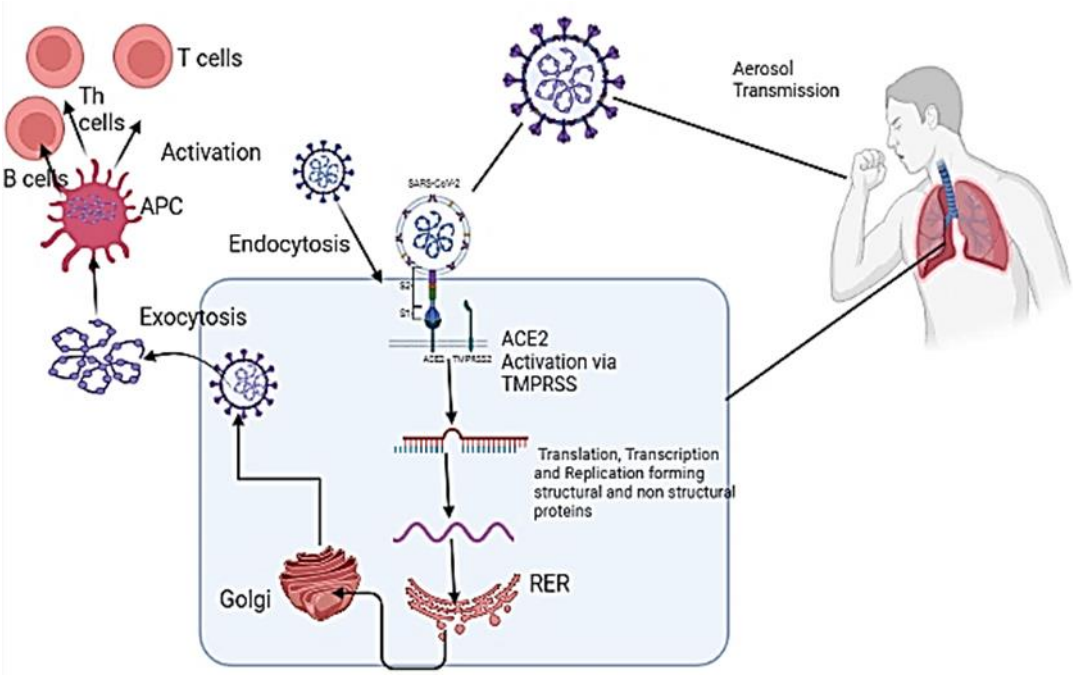

Figure 1.

Figure 1: Upon the entry of the virus the cell undergoes endocytosis and thereby leads to an activation of ACE2 via Transmembrane serine protease After this t, he series of events take place such as transcription, translation, and replication forming structural and nonstructural proteins and further processed to Rough Endoplasmic reticulum and Golgi complex-forming complex called Endoplasmic reticulum Golgi intermediate complex, finally through

the process of exocytosis, the viral cells are released and presented to antigen-presenting cells(APC) which in turn present to T cells B cells.

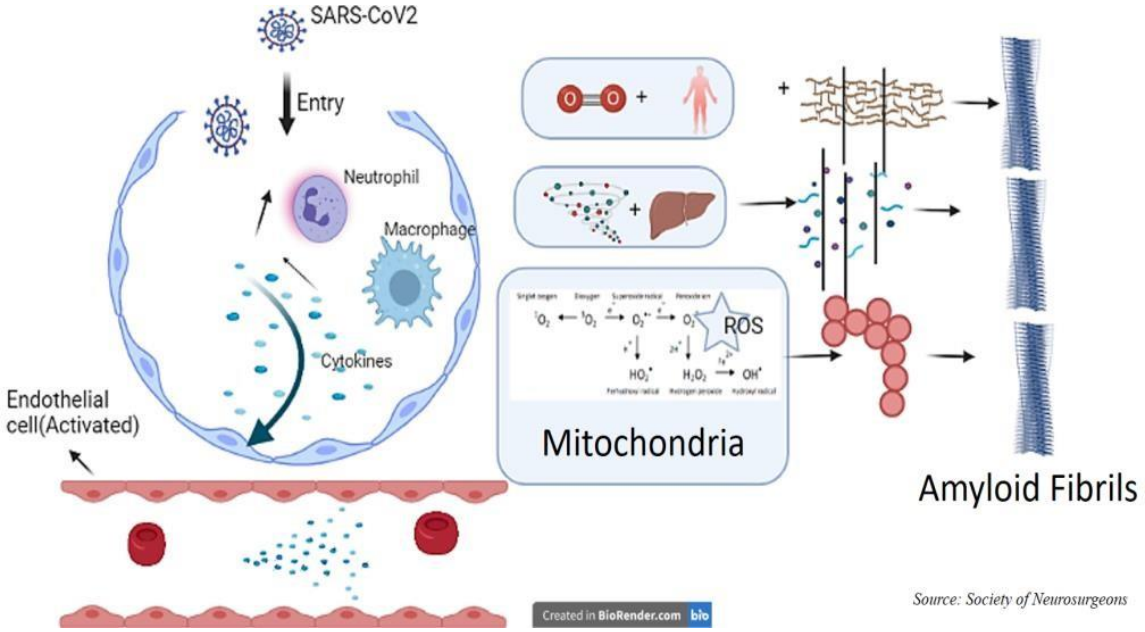

Figure 1a

Figure 1a: **To battle Alzheimer's disease (AD), many funding and research programs have been implemented, to discover the illness's underlying causes and devise effective treatments for it.** Research into Alzheimer's disease (AD) is difficult since the molecular changes that cause the disease to develop in the brain years before symptoms increase. Before any symptoms occur, the brain has already been affected by

neurodegeneration, inflammation, and oxidative stress. This indicates that a brain assault, in particular, might worsen an already-decreasing state within. SARS-CoV2 should not be ignored as a possible cause of an increased risk of Alzheimer's disease in those who have been infected, and scientists should encourage research into ACE-2 and A in those who have been exposed to SARS-CoV2 (Ebate et al., 2020). This will be addressed with the development of a prevention strategy that makes use of pharmaceutical and nonpharmacological treatment alternatives.

4. Possible Impact of SARS-CoV-2 Infection on Alzheimer's Disease Exacerbation

Are there any indications that ApoE4 might cause neurological symptoms? As a significant component of the blood's circulating lipoproteins, ApoE (ApoE4) plays a key role in the clearance of lipoproteins from the circulation. Three allelic genetic variations of human ApoE, ApoE2, ApoE3, and ApoE4, each with their receptor-binding characteristics, are expressed in the body. In some situations, ApoE2 binds less strongly than ApoE3 and ApoE4 more strongly than ApoE3, at least for ApoE receptors (Boukhris et al., 2020). For reasons that aren't clear, ApoE2 and E4 homozygosity both increase the risk of dyslipidemias, albeit to different degrees. However, active astrocytes and microglia in the brain release ApoE, which is present in the blood as well. Since ApoE4 is the most prominent hereditary risk factor for Alzheimer's disease (AD), and ApoE2 protects against AD, brain ApoE production is likely physiologically relevant.

Addiction to the Alzheimer's Virus Brain Infectious Theory

When microorganisms including Chlamydia pneumonia, Borrelia, and Helicobacter pylori were found in postmortem brains, the infectious theory of illness was linked to Alzheimer's [12] (Kumar et al., 2016). There was higher incidences of CNS herpes simplex virus (HSV1) infection in those with the apoE -4 type 4 allele, a well-known genetic predictor for Alzheimer's disease (CNS).

When immune suppression, peripheral infection, or inflammation took place, apoE-4 carriers' latent HSV1 infection became active, according to the research. Alzheimer's disease was the result of this reactivation. HHV-6A and HHV-7 levels in postmortem brain tissue in Alzheimer's disease patients are higher, compared to healthy aging individuals or those with another neurodegenerative condition than in Alzheimer's disease patients. According to the virus hypothesis of Alzheimer's disease, there is insufficient evidence to support the relationship between viral infection and long-term neurological damage (Moore et al., 2020). Viruses may have a role in the development of a broad spectrum of less evident neurological disorders, according to an expanding body of research. This may lead to dementia when the immune system assaults infected cells and causes them to die or lyse. CMV and VSV both have this potential.

SARS-CoV-2 can enter cells with the help of transmembrane proteases like ACE2. Angiotensin-Converting Enzyme 2 (ACE2) is one of these transmembrane proteases (1-7). A variety of tissues and organs, including the lungs, kidney, gut, and brain, have been reported to have ACE2 (Inyushin et al., 2017). In addition, not all haplotypes of apoE have the same concentration in blood, CSF, and tissues. This means that people with the APOE4 haplotype have lower levels of apoE in their blood and brains than people with the other isoforms, and the quality of some of the app's effects may be dependent on the apoE concentration. Inhibiting apoE4 activity, if it is damaging to the brain, might delay or prevent Alzheimer's disease. Targeting and inhibiting the apoE4 action may be accomplished by genetic, biochemical, and immunological means, all of which exist (Duggan et al., 2020). In AD patients with apoE4 (40–60 percent), this technique might help, but stopping all apoE activity in the adult brain would be better if this could be done without ramifications if all apoE forms are harmful (albeit to different degrees).

Aside from its role in Alzheimer's disease, the liver's production of the apoE protein has several other functions in the brain that may be relevant. Like the rest of the body, adipose tissue is dependent on apoE for lipid transport and homeostasis. ApoE4's aberrant health effects may be connected to its faulty lipid metabolism

since apoE4 is hypolipidated and less effective than apoE3 in promoting cholesterol efflux. If you want to understand AD as a complex illness, none of the models currently in use can do it justice (Yong et al., 2021). As a result, it is difficult to evaluate the downstream signaling ramifications of AD models' A and tau expression levels. Endogenous rat chemicals may also interact differently with human AD molecules than human AD molecules do with rodent molecules, suggesting a possible mechanism for this interaction. The TOMM40 gene on chromosome 19, which has many isoforms that are closely associated with specific forms of the APOE gene, is one example of a gene associated with apoE4 and AD that has not yet been tested using animals as models (for example, the TOMM40 gene).

Alzheimer's disease-like neurodegenerative processes may be immediately revoked by the thought that SARS-CoV-2 infection may initiate or accelerate a long-term neurodegenerative process. According to the currently known studies, there is no link between COVID-19 and the start of Alzheimer's disease. As soon as possible, draw judgments that may later come under scrutiny. Alzheimer's disease (AD) pathogenesis is heavily influenced by beta-amyloid (A) (Illenberger et al., 2020). A transmembrane protein called an amyloid precursor protein is cleaved to make it (APP). Alzheimer's disease has previously been related to many genes and proteins, including the amyloid precursor protein (APP), presenilin 1 and 2, and amyloid 2. (AD). The beta-amyloid explanation for Alzheimer's disease has been backed by several animal studies and human treatment trials. At the moment, pharmaceutical research is mostly devoted to identifying methods of lowering the A load in drugs.

Alzheimer's disease-related genetic modifications are already taking place 20 years before the beginning of symptoms, according to new research. First molecular changes are loads. There is a substantial link between viral infection and beta-amyloid. Colleagues found evidence of an in-vivo effect for the antibacterial peptide A. (AMP). In comparison to age-matched non-AD samples, brain homogenates from people with Alzheimer's disease (AD) have increased antibacterial activity (Ojeda-Juárez et al., 2020). AMP activity was shown to be correlated with the concentrations. After removing anti-A antibodies from AD brain homogenates, the enhanced antibacterial activity associated with A-mediated activity was significantly reduced or eliminated. These results suggest that Alzheimer's disease may be triggered by a short-lived viral infection. Using this approach has several advantages as well.

Inflammation in the CNS may become unusually self-reinforcing when cerebral A accumulates over time owing to an inflammatory response triggered by a short-lived viral infection. Neuroinflammation may be a precursor to Alzheimer's disease, as well (Greenwood, 2014). Therefore, viruses prefer ACE2-expressing tissue, therefore any scenario that raises ACE2 expression, particularly in the brain is likely to enhance viral invasion risk and may trigger molecular pathways that contribute to neurodegeneration.

One mechanism by which antihypertensive drugs affect the central nervous system is stimulation of the ACE2/Ang(1-7)/Mas) axis, which is well-known (RAS). The ACE2 pathway may be activated to increase heart baroreflex sensitivity. It also increases bradykinin levels, which has a hypotensive effect on hypertension rats, while also promoting depression and increasing blood pressure (Inyushin et al., 2019). Vasopressin and nitric oxide are also released more quickly. It has been found that the ACE2/Ang-(1-7)/Mas axis provides neuroprotection in a variety of experimental brain damage. Reduced RAS signaling, including the expression of ACE1, Ang-II, and AT1R, as well as lower RAS activity have been linked to ACE2 neuroprotection, which reduces oxidative stress and neuroinflammation (Dominys et al., 2017). Mice with increased long-term potency and synaptic plasticity showed enhanced cognition after activating the Mas receptor. On the other hand, mice lacking the Mas gene had no defense. As a consequence of treatment with anti-hypertensive RAS-targeting drugs, there was a reduction in the incidence of Alzheimer's disease (Torniainen-Holm et al., 2019). Mitigated hippocampal A degeneration, neuroinflammation, and cognitive decline in AD Tg mice aged 13–14 months after the administration of diminazene aceturate

(DIZE). Data from this study reveal that ACE2 overexpression is beneficial in the CNS, even in the setting of molecular disease.

To put it another way, if a patient is infected with a virus that causes neuroinflammation and A burden, then RAS-targeting anti-hypertensive drugs may cause Alzheimer's disease pathological processes. ACE2 activation has been associated with enhanced neuromodulation and immunological modulation at physiological levels, but these increases may be neurotoxic at relatively high concentrations, a factor in the development of Alzheimer's disease (Cliford et al., 2013). NO is also produced by astrocytes, microglia, and blood macrophages in response to long-term infection or the deposition of inflammation-inducing mediators, such as A. (both the soluble and the fibrillar forms). These medicines have not yet been linked to an increase in the incidence or exacerbation of acute respiratory distress syndrome in patients. Even yet, we can't rule out the long-term effects of our CNS damage.

If SARS-CoV-2′ can infect macrophages, microglia, astrocytes, and other CNS cells, such as dendritic cells, we need to know how the CNS might be damaged. For example, the activation of glial cells by a neurotropic virus might lead to an inflammatory reaction. Patients with healthy levels of interleukin-6, a fellow cytokine storm member, also have symptoms of covid-19 (Friedman et al., 2018). It has long been known that the brain's innate immune cells, known as microglia, play a role in neurodegenerative disorders. Neurotoxic factors, such as pro-inflammatory mediators and reactive oxygen/nitrogen species, have been linked to chronically activated microglia (Wakim et al., 2017). Neurons may die over time if microglia are activated by a single stimulation, such as an infection with a pathogen. Activation of brain microglia by SARS-CoV-2 infection may lead to chronic inflammation and neurodegeneration.

Table1: Possible pathways and Mechanism of COVID-19

Pathway	Mechanism
Beta-amyloid	Aβ is produced from APP by consecutive cleavages by BACE-1 and the γ-secretase complex. Scientists have worked on this pathway and these enzymes for a long time in an attempt to produce an AD medication with a hypothesis that limiting the activity of these enzymes would diminish the synthesis of Aβand hence Aβ-mediated cellular toxicity
E (ApoE)4	Amyloid-beta (A) clearance mechanisms are controlled by ApoE and ATP-binding cassette A1 (ABC A1) (ABCA1). The expression of ApoE and ABCA1 is induced by the activation of nuclear hormone receptors (LXR, PPARgamma, and RXR), which are all nuclear hormone receptors.
Neuroinflammation	Several acute and chronic neurological conditions are linked to neuroinflammation. Various disease paradigms have comparable processes when acute lesions in the brain parenchyma cause severe and extremely complicated neuroinflammatory responses. Inflammatory responses are triggered when CNS microglial cells detect tissue injury.
Microglia Activation	Following exposure to pathogen-associated molecular patterns (PAMPs) and/or endogenous damage-associated molecular patterns (DAMPs), microglia are activated. Activated microglia may take on a variety of phenotypes, each of which is

	influenced by the environment in which it grows.

Implications for Research

To battle Alzheimer's disease (AD), many funding and research programs have been implemented, to discover the illness's underlying causes and devise effective treatments for it. Research into Alzheimer's disease (AD) is difficult since the molecular changes that cause the disease to develop in the brain years before symptoms increase. Before any symptoms occur, the brain has already been affected by neurodegeneration, inflammation, and oxidative stress. This means that an attack that is harmful to the brain, in particular, might compound its already-deteriorating condition. SARS-CoV2 should not be ignored as a probable cause of an elevated risk of Alzheimer's disease in individuals who have been infected, and scientists should stimulate research into ACE-2 and in those who have been exposed to SARS-CoV2. This will be addressed with the development of a prevention strategy that makes use of pharmaceutical and nonpharmacological treatment alternatives.

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