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Narrative Review

CRISPR-Cas9: The Association between SARS-CoV-2 and Neurodegenerative Disorders (NDDs) Occurrence

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Abstract

COVID-19 is a respiratory infection caused by SARS-CoV-2 that can also have neurological manifestations and complications. There is growing evidence that COVID-19 may be associated with an increased risk of neurodegenerative disorders, such as Alzheimer's disease, Parkinson's disease, and Amyotrophic lateral sclerosis. The mechanisms underlying this association are not fully understood but may involve direct viral invasion of the brain, systemic inflammation, endothelial dysfunction, and amyloid formation. These factors may trigger or accelerate the neuronal damage and debilitation that characterize neurodegenerative disorders. Hence, this study aims to review the highlighted connections between SARS-COV-2 and NDDs, considering new technologies in treating challenging diseases. To put it better, CRISPR-Cas9 is a genome editing tool that targets and modifies specific DNA sequences in living cells. It has been used to create animal models, study gene function, and develop gene therapies for various diseases. CRISPR-Cas9 may also be a promising tool to combat COVID-19 and prevent or treat its neurological complications by targeting either the viral genome or the host factors essential for viral infection. It has been concluded that there are still some questions and limitations to the clinical application of CRISPR-Cas9, such as delivery efficiency, specificity, safety, and immunogenicity, which require further investigation and biological and medical observation to be accepted as a stable alternative treatment in this field.

Keywords: SARS-CoV-2, COVID-19, Viral Infection, Neurodegenerative Disorders, CRISPR-Cas9, Genome Editing

Introduction

The Role of COVID-19 in Neurodegenerative Disease (NDDs)

Amyloid proteins are abnormal protein structures that can accumulate in various tissues and organs, causing damage and dysfunction.[1] Amyloid proteins are associated with several diseases, such as Alzheimer's, diabetes, rheumatoid arthritis, and atherosclerosis.[2] Recently, some studies have suggested that the pathogenesis and complications of COVID-19, the disease caused by the novel coronavirus SARS-CoV-2, can lead to neurological dysfunction.[3,4] COVID-19 is a respiratory infection that can cause symptoms from mild to severe.[5] Some of the intense and long-term symptoms of COVID-19 include blood clots, neurological problems, inflammation, and organ damage.[6] The mechanisms underlying these symptoms still need to be fully understood,[7] but some evidence points to the involvement of this disease in cognitive deterioration.[7,8]

Spike Protein and Immune Response May Induce Amyloid Protein Formation

One hypothesis is that the spike protein of SARS-CoV-2, which is responsible for binding to and entering human cells,[9] can induce the formation of amyloid proteins in the body.[10] The spike protein has been shown to interact with several amyloidogenic proteins, such as beta-amyloid, alpha-synuclein, tau, prion, and TAR DNA-binding protein 43 (TDP-43).[11,12] These interactions may trigger the aggregation of these proteins and lead to neurodegeneration and other complications.

Another hypothesis is that the immune response to SARS-CoV-2 infection can produce amyloid proteins as part of the acute-phase reaction.[13,14] The cytokine interleukin-6 (IL-6), elevated in COVID-19 patients,[15] can stimulate the synthesis and release of serum amyloid A (SAA) from the liver.[16] SAA is an

inflammatory protein that can form amyloid deposits in various tissues and organs, especially in diabetic patients.[17] The potential link between COVID-19 and amyloid proteins has important implications for this disease's diagnosis, treatment, and prevention. Further research is needed to elucidate the molecular mechanisms and clinical consequences of amyloid formation in COVID-19 patients.

Overall Structure of the Information Presentation

This review aims to provide an updated overview of the connection between SARS-CoV-2 and neurodegenerative diseases and illustrate the CRISPR-CAS9 tools as a potential treatment for both complications. In this process, a comprehensive literature search was conducted in major scientific databases, including Google Scholar, PubMed, Web of Science, and Scopus, to gather relevant information. The search terms used were divided into various related topics, the most significant ones stated in the abstract keyword section. Results were considered only among the peer-reviewed articles published in English between 2020 and 2023; however, several pieces were *unrestricted* due to their importance. Ultimately, the discussion of this review will provide insights into the potential mechanisms and clinical implications of CRISPR techniques in COVID-19, NDD treatment, and its ongoing or upcoming challenges. Moreover, it sheds light on the highlighted aspect of this topic that needs additional research and experience.

Discussion

Recognition of Neurodegenerative Disease (Figure 1)

Defenition of NDDs

Neurodegenerative disorders are a group of diseases that affect the structure and function of neurons in the central nervous system, leading to cognitive impairment, motor dysfunction, and dementia. Some examples of neurodegenerative disorders are Alzheimer's disease, Parkinson's disease, Amyotrophic lateral sclerosis, and Huntington's disease.[18] The neurodegenerative disorders recognition mechanism refers to identifying and diagnosing

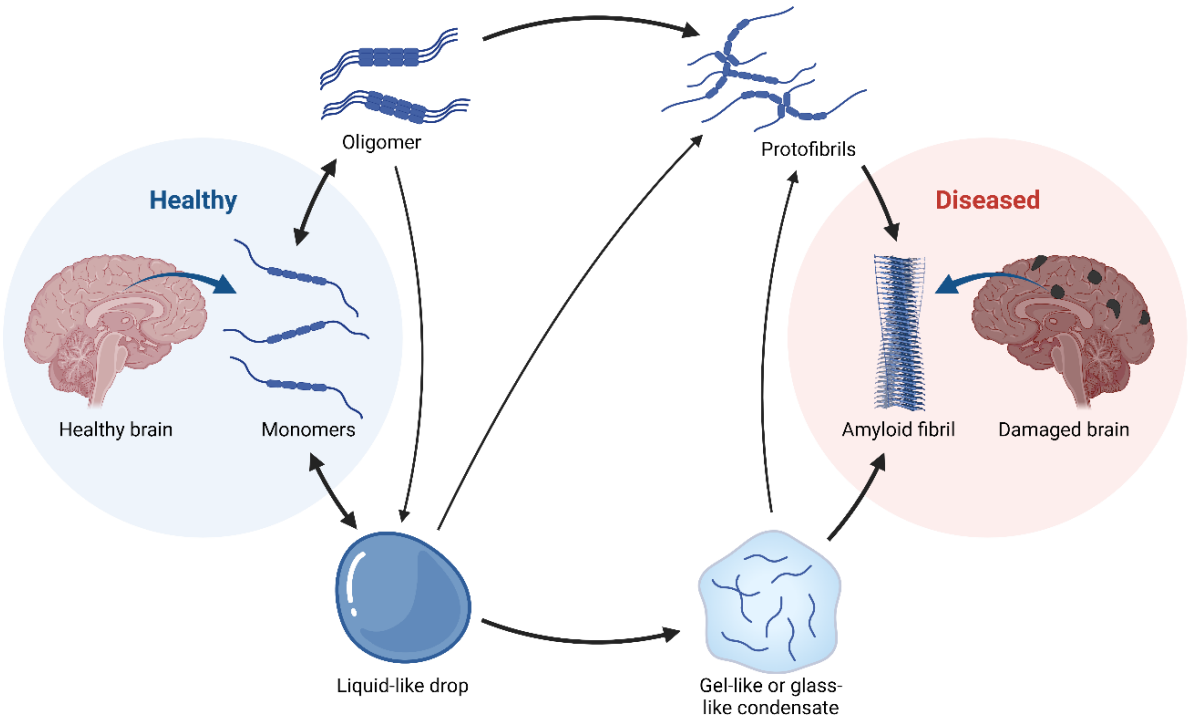

Figure 1. Amyloid fibrillation is a complex process involving various intermediates forming, including liquid-like drops, oligomers, protofibrils, and amyloid fibrils. In a healthy brain, amyloid monomers are maintained in a soluble and non-toxic state. However, in a damaged brain, these monomers can self-assemble into various intermediates, leading to the formation of amyloid fibrils and, ultimately, the appearance of amyloid plaques. Liquid-like drops are considered essential intermediates in the aggregation process, allowing for the rapid assembly of amyloid fibrils. Similarly, gel-like or glass-like condensates may also play a role in amyloid fibrillation, as they can promote the formation of stable oligomers and protofibrils. Understanding the role of these intermediates in amyloid fibrillation could provide valuable insights into the development of therapeutic strategies for neurodegenerative diseases associated with amyloid plaques. Created with Biorender.com.

these disorders based on their clinical features, biomarkers, and pathological findings;[19] also, it is challenging because of their heterogeneity, complexity, and overlap.[20] However, recent advances in molecular pathology, neuroimaging, and genetics have improved the recognition mechanism of neurodegenerative disorders by providing more specific and sensitive criteria and tools.[21,22]

Molecular Studies of NDDs

Molecular pathology studies the molecular alterations underlying pathological changes in tissues and cells.[21] The molecular pathology of neurodegenerative disorders is based on identifying abnormal protein aggregates characteristic of each condition.[23] These protein aggregates are also known as proteinopathies or proteopathies.[24] For example, Alzheimer's disease is associated with amyloid-beta plaques and tau tangles;[25,26] Parkinson's disease is related to alpha-synuclein Lewy bodies;[27] Amyotrophic lateral sclerosis is associated with TDP-43 or Superoxide dismutase 1 (SOD1) inclusions;[28] and Huntington's disease is associated with mutant huntingtin aggregates.[29]

Neuroimaging of NDDs

Neuroimaging uses various techniques to visualize the structure and function of the brain.[30] Neuroimaging of neurodegenerative disorders can help detect the patterns of brain atrophy, hypometabolism, perfusion, connectivity, and neurotransmission specific to each condition.[31] Neuroimaging can also help measure the biomarkers that reflect the pathological processes in the brain.[32,33] For example, positron emission tomography (PET) can measure the levels of amyloid-beta or tau in Alzheimer's disease;[34] magnetic resonance imaging (MRI) can measure the iron accumulation or dopaminergic loss in Parkinson's disease;[35] diffusion tensor imaging (DTI) can measure the axonal degeneration or white matter integrity in Amyotrophic lateral sclerosis; and functional MRI (fMRI) can measure the neural activity or network dysfunction in Huntington's disease.[36]

Genetic Studies of NDDs

Genetics studies the genes and their variations that influence traits and diseases. Genetics of neurodegenerative disorders can help identify the genetic factors that contribute to these disorders' risk, onset, progression, and phenotype.[37,38] Genetics can also help understand the molecular mechanisms and pathways involved in neurodegeneration.[39] For example, genome-wide association studies (GWAS) can identify common genetic variants that are associated with sporadic forms of neurodegenerative disorders;[40] whole-exome sequencing (WES) or whole-genome sequencing (WGS) can identify rare genetic variants that cause familial forms of neurodegenerative diseases;[41,42] transcriptomics or epigenomics can identify gene expression or regulation changes that affect neurodegeneration.[43]

Recognition Mechanism of NDDs

The recognition mechanism of neurodegenerative disorders is essential for improving the diagnosis, prognosis, treatment, and prevention of these disorders.[25,44,45] However, there are still some limitations and challenges for the recognition mechanism of neurodegenerative diseases, such as lack of specificity, sensitivity, validity, reliability, accessibility, affordability, etc.[46,47] Therefore, enhanced research and development are needed to improve the recognition mechanism of neurodegenerative disorders by integrating multiple modalities and sources of information.

Viral Pandemic Diagnosis (Figure 2)

SARS-CoV-2 Defenition

SARS-CoV-2 outbreak and recognition mechanism refers to identifying and understanding the origin, transmission, and pathogenesis of the novel coronavirus that causes COVID-19.[48] The recognition mechanism of the SARS-CoV-2 outbreak is essential for developing effective diagnostic, preventive, and therapeutic strategies to control the pandemic.[49] The SARS-CoV-2 outbreak is a global public health emergency that started in late 2019 in Wuhan, China. Based on the World Health Organization (WHO) report, It has since spread to more than 231 countries and territories, infecting over 760 million people and causing nearly 7 million deaths as of July 2023.[50] SARS-CoV-2 belongs to the genus Betacoronavirus, including SARS-CoV and MERS-CoV, two other highly pathogenic coronaviruses that emerged in humans in 2003 and 2012, respectively.[51]

Recognition Mechanism of SARS-CoV-2

The SARS-CoV-2 recognition mechanism involves the identification of its origin, transmission, and pathogenesis; the source of SARS-CoV-2 is still under

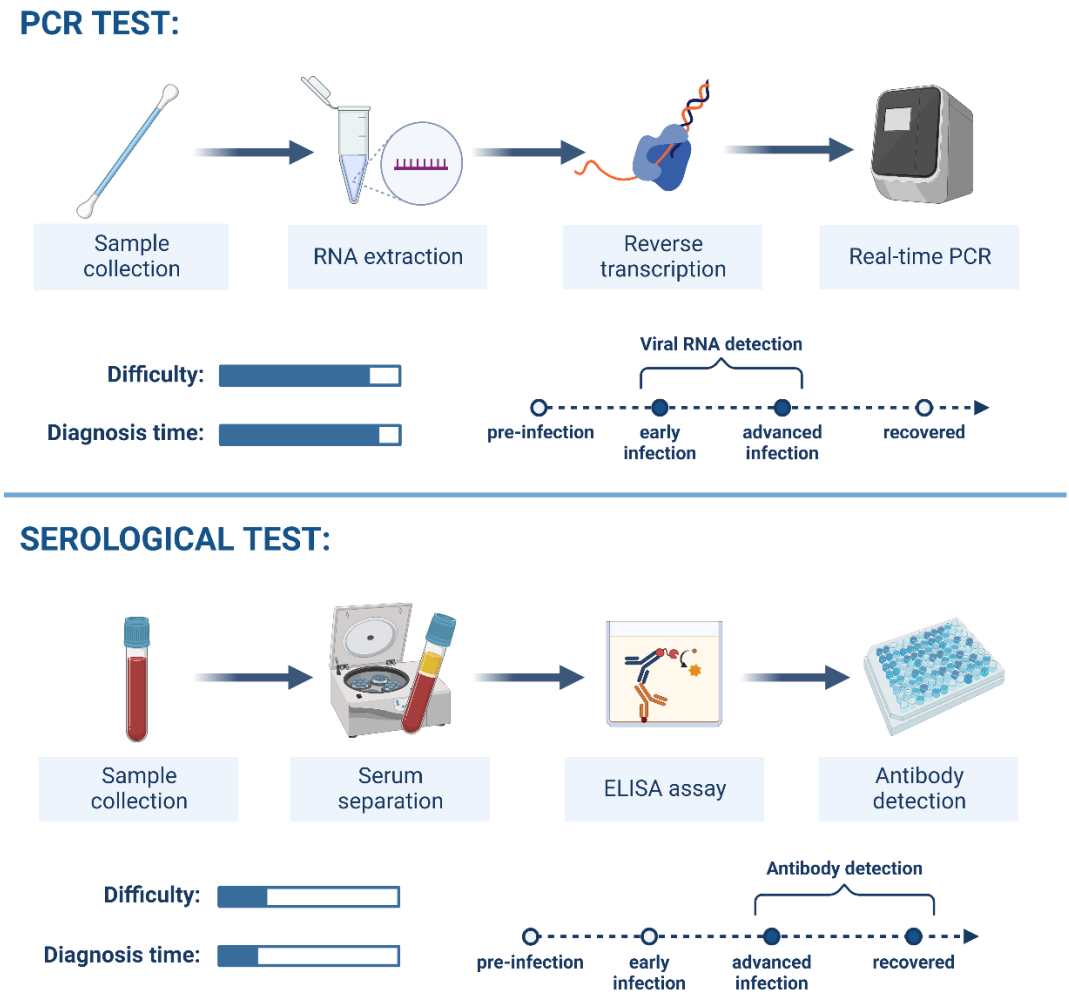

Figure 2. Different laboratory methods have been introduced to detect infections like SARS-CoV-2 (COVID-19); this illustration, it is displayed two types of determination methods that have been used in the laboratories, such as A) PCR test is widely used for detecting viral RNA in various sample types. Sample collection is a critical step in the PCR process, affecting the quality and quantity of RNA extracted from the sample. Reverse transcription, which converts RNA to cDNA, is another crucial step. Real-time PCR, which measures the amount of amplified cDNA in real-time, allows for quantifying viral RNA. The difficulty rate and diagnosis time of PCR tests can vary depending on several factors, including the type of sample collected, the PCR assay's performance, and the sample's viral load. PCR tests can diagnose individuals at different stages of infection, including pre-infection, early infection, advanced infection, and recovered individuals. The viral RNA detection time in these individuals can vary, with pre-infection and early infection having lower viral RNA detection rates than advanced and recovered individuals. B) Serological tests commonly detect antibodies against a particular pathogen in serum samples. Sample collection is a critical step in the serological testing process, as it affects the quality and quantity of antibodies in the sample. Serum separation is an essential step in obtaining high-quality serum samples. The ELISA assay is a commonly used serological test that involves the detection of specific antibodies using an enzyme-linked immunosorbent assay. Serological tests can diagnose individuals at different stages of infection, including pre-infection, early infection, advanced infection, and recovered individuals. The difficulty rate and diagnosis time of serological tests can vary depending on several factors, including the type of sample collected, the performance of the ELISA assay, and the antibody detection stage. The antibody detection time can also vary depending on the stage of infection, with early infection having lower antibody detection rates compared to advanced infection and recovered individuals. Created with Biorender.com.

investigation.[52] Still, genomic and phylogenetic analyses suggest that it is a zoonotic virus originating from bats and may have involved an intermediate animal host before jumping to humans.[53-55] The transmission of SARS-CoV-2 occurs mainly through respiratory droplets and aerosols generated by infected individuals when they cough, sneeze, or talk.[56,57] The virus can also be transmitted through contact with contaminated surfaces or objects. The pathogenesis of SARS-CoV-2 involves the interaction of its spike protein with the human receptor angiotensin-converting enzyme 2 (ACE2), which mediates its entry into host cells.[58] The

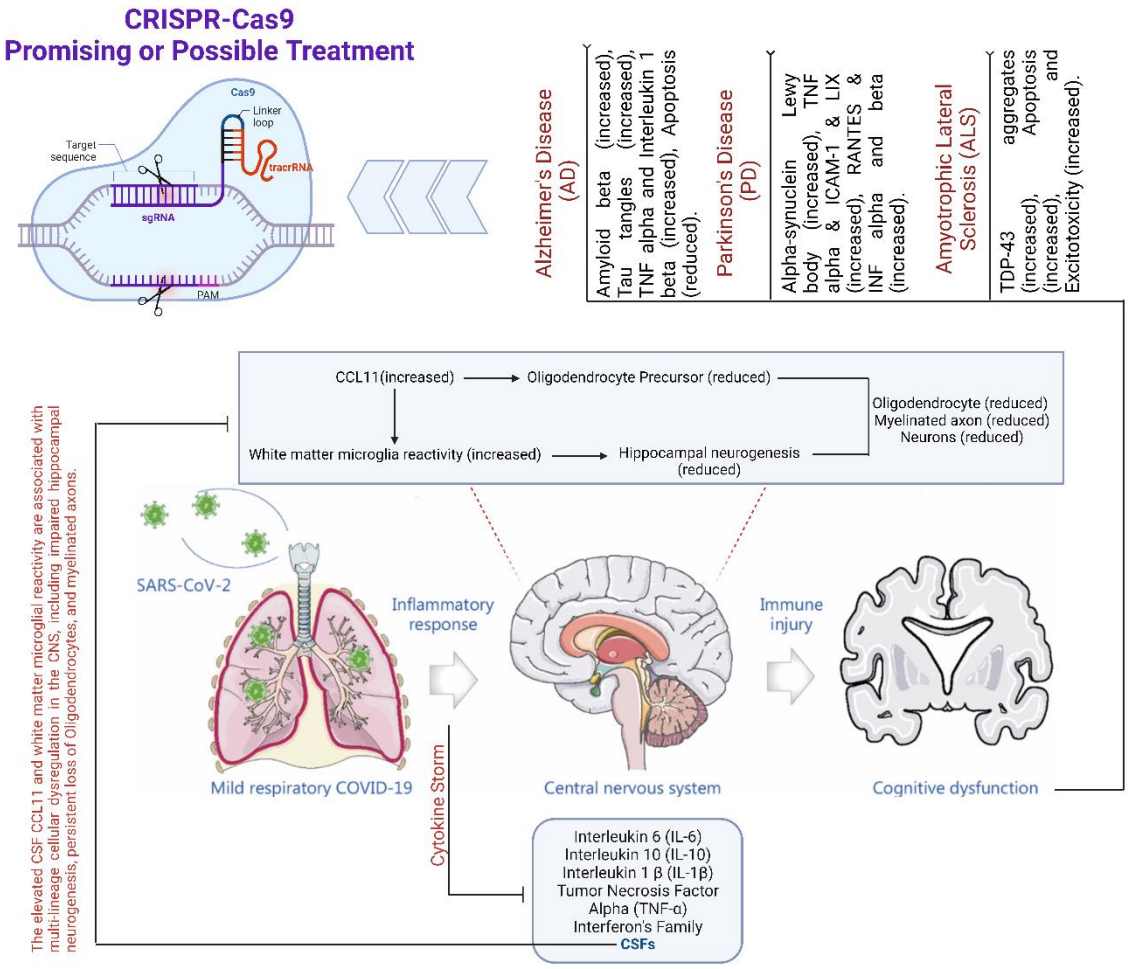

Figure 3. Infectious agents, including viral and bacterial pathogens, have been shown to contribute to neurodegenerative diseases through direct or immune-mediated mechanisms. Infection can lead to the pro-inflammatory activation of CNS resident immune cells, including astrocytes and microglia, resulting in neuronal death. The resulting inflammatory state is thought to play a role in the pathogenesis of Alzheimer's disease (AD) and Parkinson's disease (PD), characterized by the accumulation of neurotoxic protein aggregates and high levels of pro-inflammatory cytokines. Some pathogens can also directly infect neurons, leading to alterations in metabolism, enhanced neuronal excitotoxicity, and enhanced apoptosis, as seen in ALS. Neuroinflammation is a central pathophysiology that links mild respiratory COVID-19 to cognitive impairment. SARS-CoV-2 infection can cause an excessive peripheral inflammatory response, resulting in immune injury in the CNS. The elevated CSF CCL11 and white matter microglial reactivity are associated with multi-lineage cellular dysregulation in the CNS, including impaired hippocampal neurogenesis, persistent loss of oligodendrocytes, and myelinated axons. In less severe cases of COVID-19, microglial cells act as the innate immune response in the CNS, essential for proper viral clearance. However, the overactivation of microglial cells in more severe COVID-19 cases can promote detrimental effects indirectly by activating astrocytes or T lymphocyte-mediated neurotoxicity and directly by inducing synapse loss, further contributing to neuronal degeneration in response to viral infection. The cytokine storm, primarily produced by microglia, leads to increased BBB permeability and may be responsible for several of the neurological symptoms of COVID-19. Created with Biorender.com.

virus then replicates and triggers an immune response that can cause inflammation, tissue damage, and organ dysfunction.[59,60] The severity and outcome of COVID-19 depend on various factors, such as viral load, host genetics, age, comorbidities, and immune status.[61]

The Importance of Viral Detection

The recognition mechanism of the SARS-CoV-2 outbreak is crucial for improving the diagnosis, prognosis, treatment, and prevention of COVID-19.[62] Various methods have been developed to detect SARS-CoV-2 infection, such as reverse transcription polymerase chain reaction (RT-PCR), antigen tests, antibody tests, and genomic sequencing.[63-65] Several vaccines have been authorized or approved for preventing COVID-19, such as mRNA, viral vector, inactivated, and protein subunit.[66-68] Various drugs have been tested or repurposed for treating COVID-19,

such as antivirals, monoclonal antibodies, corticosteroids, and immunomodulators.[69] The recognition mechanism of the SARS-CoV-2 outbreak is still evolving as new virus variants emerge and new data become available. Therefore, further research and surveillance are needed to monitor the pandemic's dynamics and develop more effective interventions to combat SARS-CoV-2.

Neurodegenerative Disease and SARS-COV-2 Association (Figures 3 & 4)

Different Likely Pathways between NDs and SARS-CoV-2

One possible linkage between neurodegenerative diseases and SARS-CoV-2 is the direct virus invasion into the brain through the olfactory nerve or the blood-brain barrier.[70] The virus may infect neurons and glial cells, causing inflammation, oxidative stress, apoptosis, and neuronal dysfunction. The virus may also interact with proteins involved in neurodegeneration, such as amyloid-beta, tau, alpha-synuclein, TDP-43, and prion proteins.[71,72] These interactions may trigger or accelerate the aggregation and deposition of these proteins in the brain, leading to neurodegeneration.[4,73]

Another possible linkage between neurodegenerative diseases and SARS-CoV-2 is the indirect effect of the systemic immune response to the viral infection. The cytokine storm in severe COVID-19 cases may cause systemic inflammation and endothelial dysfunction,[74] impairing the blood supply and oxygen delivery to the brain.[75,76] The cytokines may also cross the blood-brain

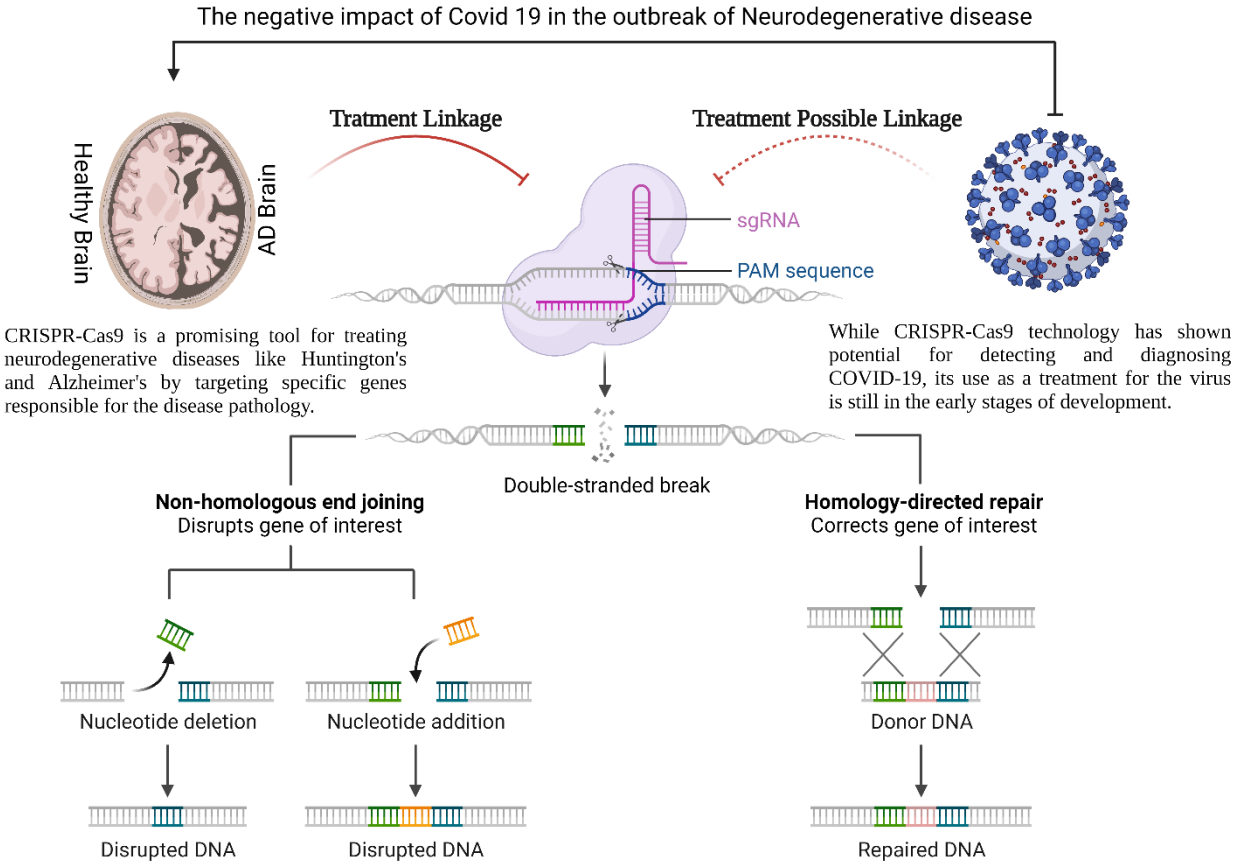

Figure 4. Neurodegenerative diseases, such as Alzheimer's disease (AD), are characterized by the loss of neurons and synapses in the brain, resulting in cognitive and functional decline. COVID-19, caused by SARS-CoV-2, has also been associated with neurological symptoms and cognitive impairment. While there is currently no known treatment linkage between COVID-19 and neurodegenerative diseases, research has shown that the CRISPR-Cas9 system has the potential to be used in treating these diseases. Specifically, the CRISPR-Cas9 system can introduce site-specific double-strand breaks in the genome using single guide RNAs (sgRNAs) that target a specific PAM sequence, allowing for the disruption of genes of interest. The resulting double-strand break can be repaired through non-homologous end joining (NHEJ), which may result in nucleotide deletions or additions, or through homology-directed repair (HDR), which uses a donor DNA template to repair the break with the correct gene of interest. This technology can potentially repair or replace disrupted DNA in the AD brain and potentially in the brains of individuals with neurological symptoms resulting from COVID-19. Created with Biorender.com.

barrier and activate microglia and astrocytes, causing neuroinflammation and neurotoxicity.[77] Moreover, some cytokines may induce the production of serum amyloid A (SAA), an acute-phase protein that can form amyloid deposits in various tissues and organs.[78] SAA may also interact with other amyloidogenic proteins and contribute to neurodegeneration.[79] The linkage between neurodegenerative diseases and SARS-CoV-2 has important implications for both conditions' diagnosis, prognosis, treatment, and prevention. Further research is needed to elucidate this linkage's molecular mechanisms and clinical outcomes

Treatment of Neurodegenerative Disorders through CRISPR-Cas9 (Figures 3 & 4)

Proposed Treatment by CRISPR-Cas9 Technology

The linkage between CRISPR-Cas9 and neuro-degenerative disease treatment is based on this genome editing tool's potential to modify the genes involved in the pathogenesis of these disorders.[80] Neurodegenerative diseases are characterized by the progressive loss of neurons and their functions, leading to cognitive impairment, motor dysfunction, and dementia. Some examples of neurodegenerative diseases are Alzheimer's disease, Parkinson's disease, Amyotrophic lateral sclerosis, and Huntington's disease.[81] CRISPR-Cas9 is a system that can precisely target and edit specific DNA sequences in living cells. It consists of two components: a guide RNA (gRNA) that recognizes and binds to the target DNA sequence and a Cas9 enzyme that cuts the DNA at the target site.[82,83] The cut DNA can then be repaired by the cell's mechanisms, resulting in either an insertion or deletion of nucleotides (indels) or a replacement of nucleotides with a desired sequence (knock-in).[84]

Animal Models and CRISPR-Cas9

CRISPR-Cas9 has been used to create animal models of neurodegenerative diseases by introducing mutations or deletions in the genes that are associated with these disorders, such as Amyloid Precursor Protein (APP), Presenilin 1 (PSEN1), Presenilin 2 (PSEN2), Synuclein Alpha (SNCA), Leucine-rich repeat kinase 2 (LRRK2), (SOD1), Chromosome 9 open reading frame 72 (C9orf72), Huntingtin (HTT), etc.[85-88] These models can help understand these genetic alterations' molecular mechanisms and phenotypic consequences. CRISPR-Cas9 has also been used to explore the therapeutic potential of gene editing for neurodegenerative diseases by correcting or silencing the disease-causing genes in vitro or in vivo. For example, CRISPR-Cas9 has been used to reduce the expression of Beta-secretase 1 (BACE1), an enzyme that cleaves amyloid precursor protein (APP) into amyloid-beta peptides that form plaques in Alzheimer's disease.[80,89,90] This decreased amyloid-beta levels and improved cognitive function in mouse models of Alzheimer's disease. Similarly, CRISPR-Cas9 has been used to lower the expression of mutant huntingtin (HTT), a protein that forms aggregates in Huntington's disease.[89,91] This reduced HTT aggregation and improved motor function in mouse Huntington's disease 98 mouse models.

Challenge of Delivery

The delivery of CRISPR-Cas9 to the brain is one of the significant challenges for its clinical application in neurodegenerative diseases.[92] Various strategies have been developed to overcome this challenge, such as viral vectors, nanoparticles, exosomes, etc. However, these methods still need to be improved, such as low efficiency, immunogenicity, toxicity, off-target effects, etc.[93] Therefore, further research is required to optimize the safety and efficacy of CRISPR-Cas9 for neurodegenerative disease treatment.

Recognition and Medication of Viral Infection via CRISPR-Cas9 (Figure 4)

CRISPR-Cas9 as a Promising Tool

CRISPR-Cas9 is a genome editing tool that targets and modifies specific DNA sequences in living cells. It has been used for various purposes, such as creating animal models, studying gene function, and developing gene therapies.[94] CRISPR-Cas9 may also be a promising tool to combat viral infections, such as COVID-19, by targeting either the viral genome or the host factors essential for viral infection.[95] Targeting the viral genome directly by CRISPR-Cas9 can limit virus replication and prevent disease. However, this strategy may also induce the formation of viruses to escape variants that can evade CRISPR-Cas9 recognition and cleavage.[96,97] To overcome this problem, multiplexed CRISPR-Cas9 systems that target multiple sites of the viral genome can inhibit the formation of escape mutants and increase the efficiency of viral clearance.[98]

Table 1. An overview of the presented data, a comparison between NDs (in this case, Alzhemer's and Parkinson's diseases) and SARS-CoV-2 infection supported by risk factors, CRISPR-Cas9 treatment, proposed diagnostic ways, clinical observation, and pioneered countries information.

Example of complications	NDs vs. SARS-CoV-2 infection	Risk factors	CRISPR Cas9 treatment	Newly proposed diagnostic ways	Clinical observations	Pioneered countries
Alzheimer's disease (AD)	SARS-CoV-2 can infect the CNS via different routes and cause neurological symptoms . SARS-CoV-2 may interact with TLR2, an innate immune receptor that plays a role in AD pathogenesis. SARS-CoV-2 infection may induce or accelerate AD pathology in patients.	Age, genetic factors (e.g., APOE4), cardiovascular diseases, diabetes, hypertension, etc.	CRISPR/Cas9 can be used to edit genes related to AD, such as APP, PSEN1, PSEN2, APOE, etc. CRISPR/Cas9 can also be used to create animal models of AD for research purposes	Biomarkers such as amyloid-beta, tau, neurofilament light chain, etc., can be detected in blood or cerebrospinal fluid samples for early diagnosis of AD. Neuroimaging techniques such as PET and MRI can also assess brain structure and function in AD.	A case of rapidly progressive AD was reported in a 75-year-old woman who had COVID-19 with mild respiratory symptoms. She developed cognitive impairment, memory loss, disorientation, and behavioral changes within three months after COVID-19.	USA, China, UK, Germany, Japan, etc
Parkinson's disease (PD)	SARS-CoV-2 can affect dopaminergic neurons that are involved in PD pathogenesis. SARS-CoV-2 may also interact with TLR2, which is implicated in PD pathology. SARS-CoV-2 infection may worsen PD symptoms or increase the risk of developing PD	Age, genetic factors (e.g., SNCA, LRRK2), environmental toxins (e.g., pesticides), oxidative stress, inflammation, etc	CRISPR/Cas9 can edit PD-related genes, such as SNCA, LRRK2, GBA, PARKIN, etc. CRISPR/Cas9 can also be used to create animal models of PD for research purposes.	Biomarkers such as alpha-synuclein, DJ-1, urate, etc., can be detected in blood, saliva, or cerebrospinal fluid samples to diagnose PD. Neuroimaging techniques such as PET and MRI can also assess brain structure and function in PD.	No specific cases of PD after COVID-19 were found in the web search results. However, some studies have reported that COVID-19 may exacerbate motor and non-motor symptoms of PD or trigger parkinsonism-like features in some patients	USA, China, UK, Germany, France, etc
COVID-19	COVID-19 is caused by SARS-CoV-2 infection that affects the respiratory system and other organs. COVID-19 can cause symptoms such as fever, cough, shortness of breath, loss of taste or smell, etc. COVID-19 can also cause complications such as pneumonia, acute respiratory distress syndrome (ARDS), septic shock, etc..	Age, comorbidities (e.g., diabetes, cardiovascular diseases), immunosuppression (e.g., cancer), obesity, smoking, etc.	CRISPR/Cas9 can target viral genes or host genes involved in a viral entry or replication. CRISPR/Cas9 can also be used to create animal models of COVID-19 for research purposes.	Diagnostic tests such as PCR, antigen, or antibody tests can detect SARS-CoV-2 infection or immune response. Saliva-based or rapid tests can also be used for screening.	A case of Creutzfeldt-Jakob disease (CJD) was reported in a 67-year-old man who had COVID-19 with severe respiratory failure. He developed rapidly progressive dementia, myoclonus, ataxia, and akinetic mutism within four weeks after COVID-19.	USA, China, UK, Germany, India etc

Reduction of Viral Infection

Targeting the host factors essential for viral infection by CRISPR-Cas9 can interfere with the virus entry, replication, assembly, or release.[99] For example, CRISPR -Cas9 can knock out or knock down the expression of host receptors, such as ACE2 for SARS-CoV-2, or host enzymes, such as Transmembrane serine protease 2 (TMPRSS2) for SARS-CoV-2. This can reduce the susceptibility of host cells to viral infection and prevent virus spread.[95,100] CRISPR-Cas9 is a versatile and powerful tool to combat viral infections, but it also faces some challenges and limitations, such as delivery efficiency, specificity, safety, immunogenicity, etc. Therefore, further research and optimization are needed to improve the performance and applicability of CRISPR-Cas9 for antiviral purposes.

Conclusion

In conclusion, neurological disorders are characterized by chronic dysfunction and degeneration of neuronal cells, which various factors, including viral infections, may cause. Viruses can affect the central and peripheral nervous system, causing direct or indirect damage to the neurons or triggering immune responses that may lead to pathological signs. Some neurological disorders associated with viral infections are Alzheimer's disease, Parkinson's disease, Guillain-Barré syndrome, multiple sclerosis, and epilepsy. However, the exact mechanisms and causal relationships between viral infections and neurological disorders are not fully understood and require further investigation. Genomic solutions may offer new perspectives for diagnosing, preventing, and treating these conditions by identifying viral and host genetic factors that influence susceptibility, pathogenesis, and outcome of the infections. This study tried to cover different aspects of these complications in parallel with introducing new method tools that can be counted as a stable and permanent treatment. Although the significance of this treatment method has been proved multiple times, its critical risk factor was not revealed comprehensively, which displays the treatment consequences over the years and generations.

Conflict of Interest

The author declares no conflicts of interest.

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