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Case Report

The Brain Damage of COVID-19

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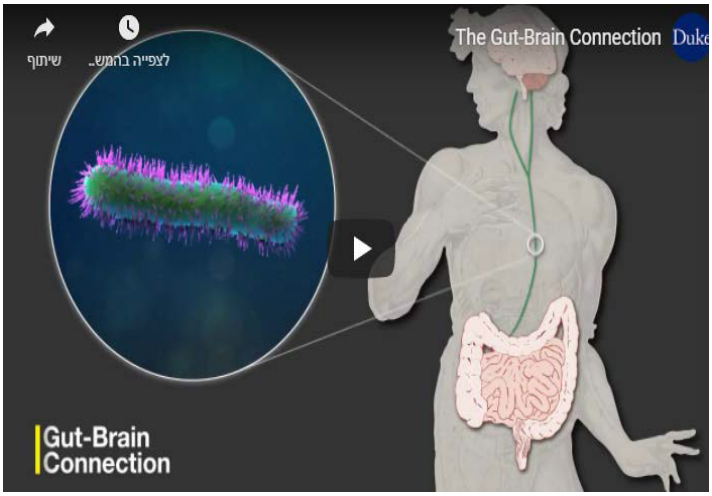
The coronavirus SARS-COV-2 virus is dwelling in the intestins and could travel into the brain by use of VAGUS nerve gut-brain avenue, causing its brain damages [1-3].

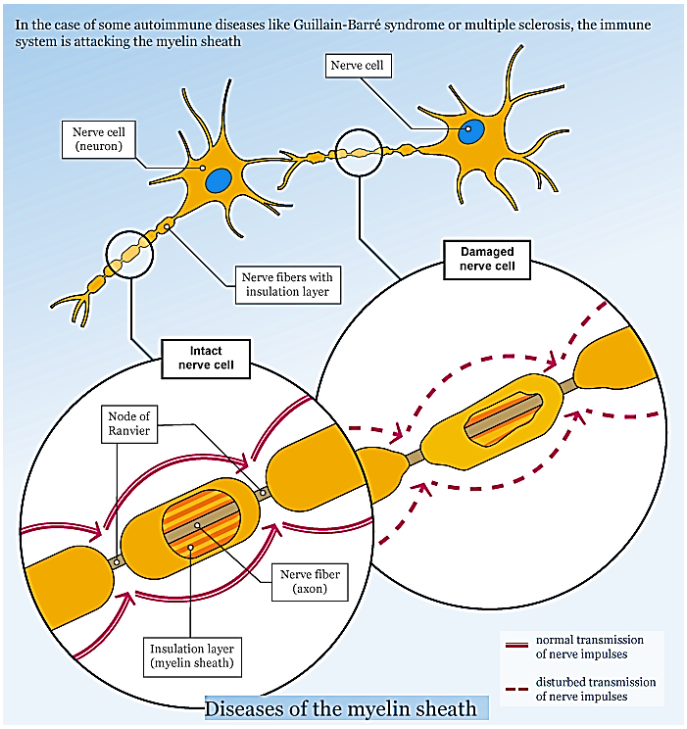
Introduction and Background

It is now clear that many patients with COVID-19 show neurological symptoms from loss of odor, to hallucinations, to an increased risk of stroke. There are also longer-term consequences for the brain, including oral encephalitis / chronic fatigue syndrome and Guillain-Barre Syndrome [4]. Guilin-Barre syndrome, an autoimmune disease that causes abnormal sensation and weakness because of delay in sending signals through the nerves. Most often reversible, in severe cases it can cause prolonged paralysis involving the respiratory muscles, require ventilator support and sometimes leave permanent neurological deficits. Early recognition by specialist neurologists is the key to proper treatment [5].



In addition, a mysterious neurological syndrome known as encephalitis lethargica appeared around the end of World War I and continued to affect more than a million people worldwide. There is limited evidence for its causes, and whether the trigger was influenza or an autoimmune disorder after infection [6].

There are numerous signs that the novel coronavirus SARS-CoV-2 not only attacks the lungs and respiratory tract, but also other organs on a massive scale [7]. It can severely affect the heart, vessels, nerves, kidneys, and skin. Acute disseminated encephalitis (ADEM) is a condition of immune-mediated inflammatory demilitarization that mainly affects the white matter of the brain and spinal cord [8]. The disorder manifests as acute encephalopathy that is activated associated with a polypological neurological deficiency and is on its way.



Thousands of cases of brain damage, even with Spanish flu

British neurologists fear that COVID-19 may leave mild brain damage in some patients, which will only become clear in the coming years. According to the study, there were similar long-term side effects found in those who recovered from the devastating Spanish flu in 1918, in which apparently up to a million people suffered brain damage [9].

COVID 19 and Alzheimer's terrible diseases look like having a similar effect on the brain [4]. It might be that the course of death by the stop of the interoception might be of the same reasons—the degradation and degeneration of the cerebellum. More and more knowledge about the cerebral part of the corona disease revealed. The idea to attack both with the same remedy or medicine might become a reality, so is the Harvard effort towards such a solution justified [10].

How does inflammation affect memory over time?

If neuronal cell activation is limited for the duration of the disease, then how can inflammation cause long-term memory loss or increase the risk of cognitive decline? Both the brain and the immune system have explicitly evolved to change as a result of experience, to neutralize the danger, and to maximize survival [11]. In the brain, changes in the connections between neurons allow us to store memories and change behavior quickly to escape a threat, or look for food or social opportunities. The immune system developed to regulate the inflammatory response and antibody production against pathogens previously encountered. However, long-term changes in the brain after illness are also closely related to an increased risk of age-related cognitive decline and Alzheimer's disease. The disruptive and destructive actions of nerve cells and inflammatory signaling can permanently impair memory. This can occur by permanent damage to the neural connections or to the neurons themselves and also through more subtle changes in the way nerve cells function. The potential association between COVID-19 and lasting effects on memory is based on observations of other diseases. For example, many patients recovering from a heart attack or bypass surgery report cognitive deficits that persist excessively during aging. Another major disease that suffers from similar cognitive complications is sepsis - a multi-organ function caused by inflammation. In animal models of these diseases, we also see memory impairments and changes in neuro-immune and neuronal function that last for weeks and months after the disease]. You are too busy to read everything. We understand. That is why we have a weekly newsletter. Sign up for a good read on Sunday Even mild inflammation, including chronic stress, are now recognized as risk factors for dementia and cognitive decline during aging In my lab I and my friends also saw that even without a bacterial or viral infection, activating an inflammatory signal over a short period of time leads to long-term changes in neuronal function in brain areas associated with memory and memory impairments.

The immune system and the brain

Many of the symptoms we attribute to infection actually stem from the immune system's protective responses. Rhinitis during a cold is not a direct effect of the virus, but a result of the immune system's response to the cold virus. This is also true when it comes to feeling sick. General illness, fatigue, fever, and social withdrawal are caused by the activation of these changes in brain and behavior, although they irritate our daily lives, are very adaptive and incredibly beneficial. By resting, you allow the immune response that requires energy to do its thing. Fever makes the body less welcoming to viruses and increases the efficiency of the immune system. Social withdrawal may help reduce the spread of the virus. In addition to changing behavior and regulating physiological responses during illness, the immune system that specializes in the brain also plays a number of other roles. It has recently been shown that the neuroimmune cells that sit in the connections between brain cells (synapses), which provide energy and thin amounts of inflammatory signals, are essential for the formation of proper memory.

Unfortunately, it also provides a way in which diseases like COVID-19 can cause acute neurological symptoms and long-term problems in the brain. Microglia are immune cells that specialize in the brain. In healthy situations they use their arms to check the environment. During an immune response, microglia change its shape to confuse pathogens. But they can also damage their neurons and connections that store memory. Inflammation, the immune cells that specialize in the brain are activated, spreading enormous amounts of inflammatory signals and changing the way they attach to neurons. For one type of cell, microglia, it means transforming, pulling the modest arms and turning them into screwedup mobile cells, enveloping potential pathogens, or cell debris in their path. But in doing so, they also destroy and consume away at the neural connections that are so important for memory storage. Another type of neuro-immune cell, called astrocyte, usually envelops the connection between neurons during disease-causing activation and imposes inflammatory signals on these nodes, thus effectively preventing the changes in relationships between neurons that store memories. Because COVID-19 involves the massive release of inflammatory signals, the effect of this disease on mind is of particular interest to me. This is because of both short-term effects both on cognition (hallucination) and the potential for long-term changes in memory, attention, and cognition. There is also an increased risk of cognitive decline and dementia, including Alzheimer's disease, during aging [12].

Neurological Complications of COVID 19

The current coronavirus is hitting the world. Like other outbreaks of epidemics, it hits waves. Today the world, after the experience of a brief recovery from the first wave, is experiencing the second wave. The past teaches us that the second wave can be more destructive than the first. But we are in the midst of this, East and West all over the world infected with the virus.

Modern medicine knows very well and very effectively how to treat and even cure the lesion as it passes through the body systems. From infection running through the body. The eyes and nose to the

intestines to the lungs, kidneys, heart, and more. But its penetration into the human brain is a challenge that is difficult to deal with. This penetration to rest and the overresponsive "cytokine storm" is what causes the human immune system to destroy the nerve cells of the brain and cause the activity of all life systems in the body to stop [13]. Circulating cytokines released during a severe systemic inflammatory stress could lead to atherosclerotic plaque instability and rupture [14]. This is a stage that brings with it the bitter death of the person infected with the virus.

And the exaltation of God in the throats of the experts will not help, because like Alzheimer's disease, there is no cure for it yet. The long-awaited vaccine will probably be able to treat mainly during the infection phase. But preventing brain damage and cytokines still poses a huge challenge to science. And right now there is no savior. Hence probably the great anxiety.

The world is trying by closure and cessation of activity, a measure taken back in the times of the Black Death or the Black Death was a plague that struck Asia and Europe in several waves for about five years (1347–1351) and killed, according to various estimates, between 75 and 200 million people in Europe and Asia. Estimates range from 60% of Europe's population, to about 35 million people in China alone. Outbreaks of the disease, on a smaller scale, continued to strike in various places in Europe until the 17th century.

The public health crisis COVID-19 has led to general recommendations from officials to contain its spread. Anxiety about "contracting" the virus is prominent among the public. Specific pathways for anxiety due to contractile disease examined. Expected that a contributing characteristic of fear of being infected with COVID-19 involve a tendency to tenderness and sensitivity, emotional responses that are part of a broader behavioural immune system. The results support a moderate relationship between a tendency to disgust and sensitivity in the relationship between physical concerns related to anxiety sensitivity and fear of infection in COVID-19. These results provide support for individual differences in BIS activation. Mental health consequences of epidemics.

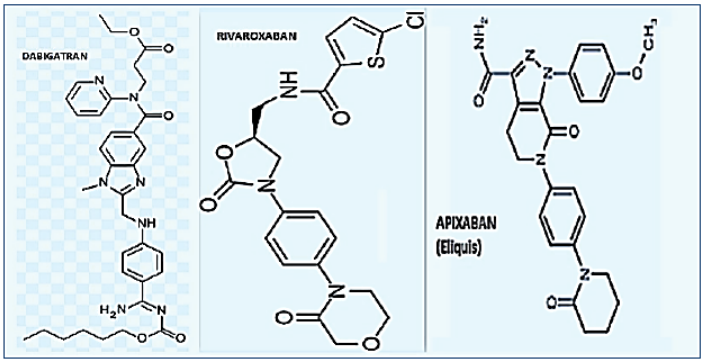
Since December 2019 almost 10 million cases and 500,000 deaths as a result of the coronavirus virus. Severe acute respiratory distress syndrome (SARS-CoV-2) has been reported worldwide. Although coronary artery disease (COVID-19) respiratory complications have been the most common and life threatening, there are more and more reports of central and peripheral nervous system (PNS) involvement.

In people with pre-existing cardiovascular obstruction, infection, fever and inflammation can impair the stability of the asymptomatic lipid layer that previously existed in the cardiovascular system. Fever and inflammation also make the blood more prone to blood clotting, interfering with the body's ability to dissolve blood clots - a punch of two similar to injecting gasoline into the coals [15].

Doctors report on a mysterious neurological syndrome known as encephalitis for tragedy appeared around the end of World War I and continued to affect more than a million people worldwide. There is limited evidence for its causes, and whether the trigger was influenza or an autoimmune disorder after infection [4].

In addition to a sleepy coma, some patients suffered from movement disorders that is renenissant to the Parkinson's disease, which affected them for the rest of their lives. In people with pre-existing cardiovascular obstruction, infection, fever and inflammation can impair the stability of the asymptomatic lipid layer that previously existed in the cardiovascular system. Fever and inflammation also make the blood more prone to blood clotting, interfering with the body's ability to dissolve blood clots - a punch of two similar to injecting gasoline into the coals [16].

These neurological complications included encephalopathy, ischemic stroke, acute nodular encephalopathy, and Guilin-Barra syndrome (GBS). Coronavirus 2019 (COVID-19) is an epidemic. Neurological complications of COVID-19 Not reported. Encephalopathy has not yet been described as a presenting symptom, either complication of COVID-19. There is a report, a case of a 74-year-old patient, traveling from Europe to the United States and served with encephalopathy and COVID-19. Categories [17].



Blood Thinners

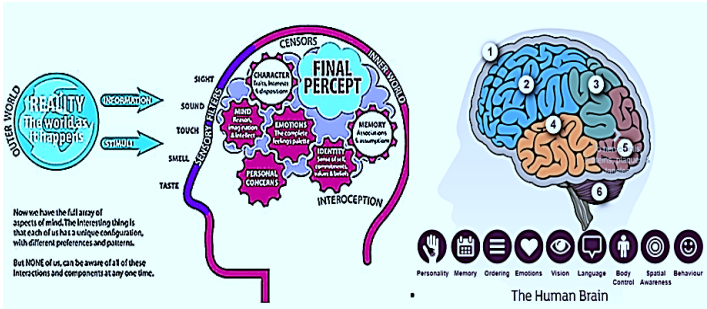
Brain damage could also take place in such circumstances. The doctors thought they thought the blood thinners were causing bleeding in the brain, but what was happening was weird and different. Some people suffer from an acute stroke as a result of a clot, and exclude vital areas of the brain from the blood supply. Tests show that there are remarkably high levels of a marker for the amount of clotting in the blood known as D-dimer. It is usually less than 300 and in stroke, patients can rise to 1,000. Patient's levels were over 80,000. This high level of coagulation shows something about his body's response to the infection that caused the blood to become incredibly sticky. The body's response to infection causes the blood to become so sticky. During the lock, there was a decrease in the number of surgeries for emergency stroke. But within two weeks, neurologists had treated six 19 Cubid patients who had suffered a stroke. These were not related to the common

risk factors for stroke, such as high blood pressure or diabetes. In both cases, they saw very high coagulation levels.

Part of the trigger for the beatings was a massive overreaction of the immune system, causing inflammation in the body and brain. Doctors examined the patient's brain images projected on a wall. They can highlight the vast areas of damage, presented as white blur, that affect his vision, memory, coordination, and speech. The stroke was so severe that doctors thought he was unlikely to survive or be severely disabled [2]. The sudden odor loss in COVID-19 may help us understand how SARS-CoV-2 works. However, several early studies show that the anosmia seen with COVID-19 exists in 30-98 % of infected people seen in hospitals, far more than it does in other respiratory infections. (Examining odor function is standardly difficult to standardize across locations, cultures, and scenarios, so this leads to high variability in the estimated prevalence of hyposemia). Another international study, presented in large numbers, asked people to check themselves with household items and report odor intensity. There is something special about the coronavirus that attacks the sense of smell in particular, which may help us understand how the virus works.

Losing yhe sense of smell

Lost of senses like sight, smell, taste, and more are reminiscent of neurodegeneration like in Alzheimer's disease.

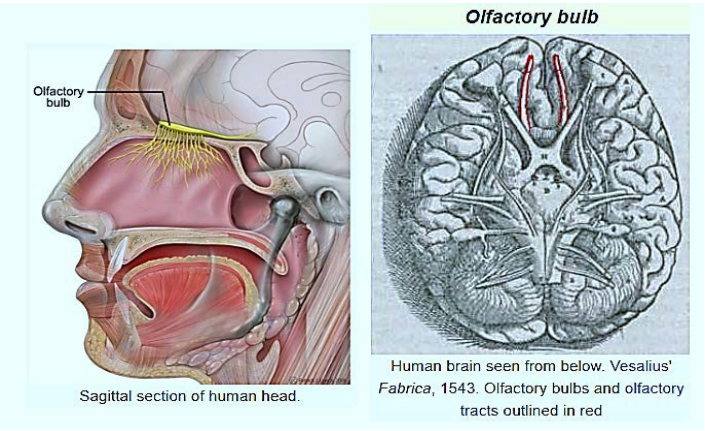


Progression of neurodegeneration brain destruction in Alzheimer's

How does the coronavirus attack the sense of smell? The research comes out quickly, and we have access to early reports, usually using pre-print servers that have not yet gone through the peer review process and should be interpreted carefully (as happens in many of the studies discussed above). Mechanical studies indicate which factors are relevant to hypoxemia and anosmia. Most scientists agree that SARS-CoV-2, like the previously known SARS-CoV, uses the angiotensin-converting enzyme 2 receptors (ACE2) to achieve cell entry through contact with a spike protein. SARS-CoV-2 appears to need PRSS2TM, a protease, to help deliver a prickly protein in the process of entering cells and some other proteins. This shows that cells must express all of these proteins in order for the virus to penetrate them and hijack their machines in order to replicate [18].

ACE2 and TMPRSS2 are expressed in many cell types, and are abundant in the nose, throat, and upper respiratory tract. In the nose, the expression is seen in both the respiratory epithelium (RE) and the sensory epithelium (OSE) but at much higher levels in OSE. In OSE, the proteins are expressed in the supporting stem cells (suppositories) and the smell of pregnancy, as well as in the cell glands (mucus) and the stem cells at lower levels.

There is accumulating evidence that the virus can migrate to the central nervous system through the nose and olfactory bulbs and as by other ways without invading the sensory neurons.



The olfactory bulbs (credit Wikipedia)

All of these tissues help maintain the health of the sensory nerve cells and the mucous layer so that odors can activate the sensory neurons. To date, olfactory sensory neurons themselves do not appear to have the correct expression patterns for SARS-CoV-2 binding, which means that the virus cannot invade directly into these neurons that infiltrate the olfactory cortex directly into the cortex.

There is proof that the virus can migrate to the central nervous system through the bulbs and the odor of life as well as in other ways without invading the sensory neurons. However, the expression patterns of ACE2 and PRSS2TM and the sudden onset and relatively rapid recovery suggest that COVID-19 anemia is not caused by damage to the central nervous system but by loss of olfactory information before it reaches the brain. If the symptoms were by the inner brain driven, we would expect a slower recovery process and more complex range of symptoms, including perhaps prosemia or pantosemia (distorted odors or hallucinations) that have not been reported.

Sensory epithelial inflammation may restrict airflow to the relatively small, high-up nasal odor cleft without causing a stuffy nose or breathing disorder, as demonstrated in a published report.

Damage to the pale cells in SE can also affect the function of sensory nerve cells in many ways (like Metabolic, structural, inflammatory), so that even if odors can reach neurons, they may not be able to transmit signals.

There are still many questions open to this mystery, but unprecedented international collaborations and early data sharing will undoubtedly propel research forward faster than usual. The odor relationship is an important clue to the virus mechanisms and leverage many decades of basic research by chemosensory scientists that took place in relative obscurity and put a spotlight on anosmia as a sensory disability.

Final Remarks

Acute Respiratory Syndrome (SARS-CoV-2) causes COVID-19, a pandemic respiratory disease that presents with fever, cough, and often pneumonia. Furthermore, thromboembolic events throughout the body including the central nervous system (CNS). Given the first indication of the presence of viral RNA in the brain and cerebrospinal fluid and in light of the neurological symptoms in a large majority of COVID-19 patients, it is likely that SARS-CoV-2 infiltration of the central nervous system. By accurately examining and mapping the aerobic and pharyngeal regions and brain of patients dying from COVID-19 accurately, we not only describe a primary nervous system infarction as a result of the cerebral blood drug but also demonstrate SARS-CoV-2 neurotropism. SARS-CoV-2 enters the nervous system by trampling the border of the olfactory-mucosal interface by utilizing the immediate vicinity of the olfactory mucosa and healing the nerves, including sensitive and sensitive nerve endings. Researchers found that SARS-CoV-2 monitors defined neuroanatomical structures. It penetrats defined neuroanatomical regions, including the primary respiratory control and cardiovascular control center in the medulla oberonga [7].

COVID-19 proves to be a more serious disease than seasonal flu in everyone, including people with diabetes. All of the standard infection prevention measures that have been extensively reported are even more important when dealing with this virus. Recommended precautions are the same as for the flu, such as frequent hand washing and covering coughs and sneezing in the tissue or elbow. We encourage people with diabetes to follow the guidelines of the CDC and review how you manage sick days - preparing for a sick day can make it easier.

President Obama warned the United States to prepare for a return to agenda in 2014, Bill Gates, and many others warned the world of the impending global pandemic caused by viruses. They demanded that the scientific, industrial, and economic should prepare; some work was done but in low gear, too slowly as we experience today [19-22].

Among adults, the risk for severe illness from COVID-19 increases with age, with older adults at highest risk. Severe illness means that the person with COVID-19 may require hospitalization, intensive care, or a ventilator to help them breathe, or they may even die.

COVID 19 in elderly Patients is a very risky matter:
Men in these groups appear to be at a higher risk than females. Chronic obstructive pulmonary disease (COPD). cardiovascular diseases and hypertension have been identified as strong predictors for intensive care units (ICU) admission.

(The prevalence of background medical conditions was 7% for ischemic heart disease, 30% for hypertension, and 19% for diabetes. Disease severity tended to be higher older individuals (mean age 47 vs. 57 vs. 61, = 0.001) and patients were more likely to be male (80% vs. 84% vs. 52%). They were more likely to present with hypertension (50% vs. 41% vs. 20%, = 0.002) and diabetes (31% vs. 25% vs. 12% = 0.001).

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