

Coronavirus, Immunity and Use of Pulsed Electromagnetic Fields (PEMF's)

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ABSTRACT

The coronavirus pandemic COVID-19 has created havoc globally. At this point there is no known cure and the death rate is high. This paper reviews the risks of spread of the virus, symptoms, incubation, pathophysiology, process of infection and the immune effects of the virus. By better understanding of the COVID-19 tissue targets, immune effects and process of infection, some targeted therapeutic strategies are discussed, as well as a proposed use of pulsed electromagnetic fields (PEMFs) to help at various points through the full scope of the infection, from incubation to replication, viremia, pneumonia and through recovery.

Keywords

Coronavirus, Immunity, Pulsed Electromagnetic Fields, Infectious diseases.

Introduction

This paper is intended to discuss the immune responses of the body to the COVID-19 virus, as we know them now. Also, I will also present how pulsed electric magnetic fields (PEMFs) affect inflammation, a hallmark of infection with this virus.

It will take months to years to have a more complete picture of the COVID-19 virus and the host immune responses. As with any other viral infection, the body mounts an immune reaction to combat the invasion. Most of the symptoms in acute viral respiratory infections are due to the physical responses caused by the body's immune reactions to the virus [1]. So, understanding the immune responses can guide us in developing treatment strategies to decrease the potentially overwhelming immune reactions and subsequent damage from the infection.

As with all infections, there are 3 components: the infectious agent/s, the immune reactions and the tissue damage. Addressing the infectious agent/s and the immune reaction is not enough in terms of managing the situation. Recovering from the tissue damage is an important part of more rapidly returning to, hopefully, whole health.

The risk of spread of a virus is important to consider. The basic reproductive number, R_0 , which is the average number of people that one infected individual will pass the virus on to. If R_0 is higher than 1, continued transmission can occur. R_0 of SARS-CoV-2 ranged from 2.2-2.6, as of March 10, 2020, with an epidemic doubling time of 6.4 days. This implies that, in order to reduce R_0 below 1, more than half of the current infections must be prevented or controlled. For a relative perspective, a typical seasonal flu strain has an R_0 of around 1.2, which means for every five infected people, the disease will spread to six new people on average, who pass it along to others. It's worth considering the relative R_0 of Measles from a spread perspective. Measles infection R_0 is cited between 12 and 18, meaning each person with measles infects between 12 and 18 new people in an unvaccinated population. For an expanded discussion of this issue and the importance of considering mortality in any discussion of R_0 , see [2].

The COVID-19 virus

Because the makeup of the COVID-19 virus is similar to SARS-CoV (SARS) and MERS-CoV (MERS), it may be reasonable to assume that the immune system may deal with this virus mostly similarly and it may likewise try to evade the body's immune responses. Coronavirus disease 2019 (COVID-19) is caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), so, the two terms are synonymous. The fatality rate from SARS-CoV-2, so far, appears lower than that of SARS (9.14%) and MERS (34.4%). The cumulative confirmed SARS-CoV-2 cases,

occurring within approximately only 2 months after the outbreak, markedly exceeded SARS, (8,096 cases, since 2002) and MERS (2,494 cases, since 2012). The highly contagious nature of SARS-CoV-2 is probably due to the virus spreading via asymptomatic-infected individuals.

The most common symptoms of COVID-19 are fever, fatigue, and respiratory symptoms, including cough, sore throat and shortness of breath. Although diarrhea was present in about 20-25% of patients with SARS and MERS, intestinal symptoms are rarely reported in people with COVID-19. Muscle aches were the third most common reported symptom after fever and cough [3].

The mean incubation period has been reported to be 6.4 days, ranging from 2.1 to 11.1 days (2.5th to 97.5th percentile). However, from a larger sample for estimation, the median incubation period was only 3 days, but could be as long as 24 days [4]. The median time from symptom onset to acute respiratory distress syndrome (ARDS) can be about 8 days. Then the virus begins a second attack, causing further aggravation of symptoms around 7 to 14 days after onset [5].

In a new study [6], with respect to those dying of COVID-19, until Feb 25, 2020, estimates were made of the time between onset of symptoms of infected individuals and death or discharge from hospital by age-strata to give the infection: fatality ratio. The mean duration from onset of symptoms to death was 17.8 days (16.9–19.2 days) and to hospital discharge 24.7 days (22.9–28.1). Of all laboratory confirmed and clinically diagnosed cases from mainland China (for 70,117 individuals), the case fatality ratio in China overall was 1.38%. It was 0.32% in those aged <60 years, 6.4% ≥ 60 years and up to 13.4% (11.2–15.9) in those ≥ 80 years. From non-China cases, these rates were 1.4% for <60 years, 4.5% [1.8–11.1] for ≥ 60 years. The estimated overall infection: fatality ratio for China was 0.66%, with an increasing profile with age. Proportion of infected individuals likely to be hospitalized increased with age up to a maximum of 18.4% in those aged ≥ 80 years [6].

Process of infection

During infection the virus passes through the mucous membranes, especially nasal and larynx, then enters the lungs through the trachea, bronchi and bronchioles. The most common early symptoms of infection are fever and cough. The virus may enter the peripheral blood from the lungs, causing viremia. So, the virus can create havoc not only by direct action on the airways but also later during the infection spreading from within, from the blood.

The virus attacks the organs that have ACE-2 receptors, such as the lungs, heart, kidneys and gastrointestinal tract. The SARS-CoV-2 has been detected in fecal samples, because of entry into the circulation from the lungs and then travels in the blood to the intestines.

On entry into the pulmonary system, the SARS-CoV-2 virus attaches itself onto ACE-2 receptors. These receptors are mainly

found in a small subset of cells in the lung called type 2 alveolar cells. ACE-2 is a receptor similar to ACE, angiotensin-converting enzyme, an enzyme in the Renin-Angiotensin system (RAS). ACE-2 receptors are also found in the kidney and gastrointestinal tract, which can also harbor SARS-CoV. See the graphic (Figure 1) below of the ACE-2 receptor [7].

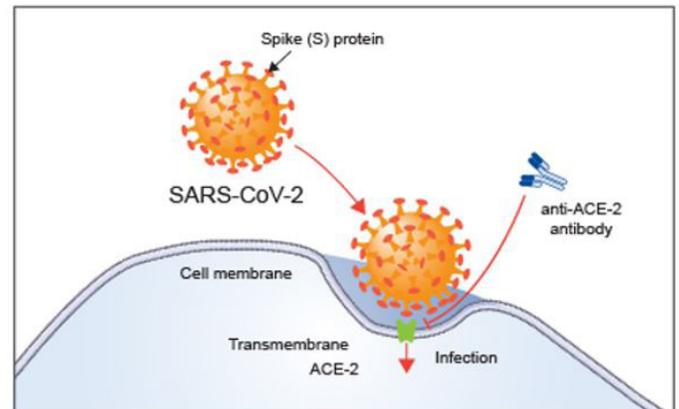


Figure 1: ACE-2 is the host cell receptor responsible for mediating infection by SARS-CoV-2, the novel coronavirus responsible for COVID-19.

The scourge of COVID-19 is respiratory failure. This virus can cause diffuse alveolar damage that results in acute respiratory distress syndrome (ARDS), severe inflammatory disease of the lung, that can lead to death. The mortality from ARDS depends on the severity of the disease, being 27%, 32%, and 45% of deaths for mild, moderate, and severe disease, respectively [8]. The primary cells affected are the type II alveolar cells.

The list of those who are most vulnerable would include those with significant underlying lung disease or in those who are significantly immunocompromised including, but not limited to, organ transplants, active chemotherapy with low white blood counts, immunosuppressive medications, and generally low immune function, such as seen in the very elderly.

Even though the virus starts by entry into the nose, and if not controlled by the body at this point of entry, replication then proceeds down the respiratory tract ending in the alveoli of the lung. From there infection can lead to pneumonia and progress to ARDS. Because the alveoli are the most important target tissue of COVID-19 infection, it may be helpful to describe them.

Alveoli

The trachea descends into the chest from the larynx and divides into bronchi, then into bronchioles. At the end of the bronchioles are the alveoli, which are often described as grape-like clusters. These clusters are alveolar sacs. Each bronchiole gives rise to between two and eleven alveolar ducts. Each duct opens into five or six alveolar sacs into which clusters of alveoli open. New alveoli continue to form until the age of eight years. A typical pair of human lungs contain about 300 million alveoli, producing 70 m² (750 sq ft) of surface area. The diameter of an alveolus is between

200 μm (0.008 inches) and 500 μm (0.02 inches).

The alveoli are where the oxygen exchange in the lung happens (Figure 2). [9] Carbon dioxide [CO_2] diffuses from the blood into the alveolus and out of the lungs on exhalation. Oxygen (O_2) coming from the air we breathe after inhalation diffuses into the blood and then into the general circulation of the body.

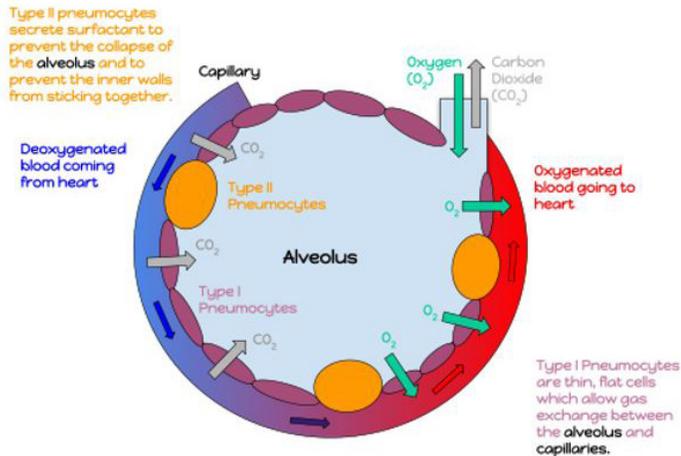


Figure 2: Alveolus, types of pneumocytes and gas exchange.

There are three major types of cells making up an alveolus. Two types are pneumocytes or pneumonocytes known as type I and type II cells, found in the alveolar wall, and a large scavenging (phagocytic) cell known as an alveolar macrophage that moves about in the cavities of the alveoli, and in the connective tissue between them. These alveolar macrophages have the task of cleaning up microbial, viral and particulate material entering the lungs through the airways.

Type I cells are thin and flat and form the structure of the alveoli. Type II cells, also called type II pneumocytes or type II alveolar cells, release a type of “soap,” pulmonary surfactant, to lower alveolar surface tension, and can also differentiate to replace damaged type I cells. Type II cells are the most numerous cells in the alveoli. Surfactant is a film of fatty phospholipids that reduces alveolar surface tension. Re-inflation of alveoli after each exhalation is made easier by the surfactant. Without the surfactant the alveoli would collapse, leading to fluid filling the lungs. Type II cells are typically found at the blood-air barrier, where oxygen exchange happens (Figure 2).

The significance of SARS-CoV-2 attacking the type II alveolar receptors of the lungs is that rapidly progressive infection leads to loss of surfactant producing cells, causing collapse of alveolar clusters, thus leading to pneumonia and shortness of breath. If not stopped quickly, this process can progress to potentially fatal ARDS.

Pneumonia is an inflammatory condition of the lung tissue, which can be caused by both viruses and bacteria. Pneumonia is the most common cause of death, as well, from influenza viruses. In

response to the lung inflammation caused by infection, cytokines and fluids are released into the alveolar cavity, walls of the alveoli, or both, reducing the surface area of gas exchange. In severe cases where cellular respiration cannot be independently maintained, supplemental oxygen or respiratory support may be required.

The immune response

In most infections, the body mounts a brisk defensive immune response. White blood cell (WBC) counts usually go up, mostly represented by lymphocytes. Lymphocytes engaged in battle with a virus will produce alarm sounding molecules, called cytokines. There are both pro-inflammatory and anti-inflammatory cytokines. The cytokines tell other circulating WBCs that help is needed for the battle. As more and more WBCs enter the fight, progressively greater amounts of cytokines are produced. When the amounts become disproportionately large, the so-called “cytokine storm” happens.

Unlike other types of viral infections, most patients hospitalized with SARS-CoV-2 develop low white blood cell lymphocyte counts (lymphopenia) and pneumonia with characteristic “ground glass opacity” lung changes on chest x-ray or CT. In a study of 41 hospitalized patients, there were high-levels of pro-inflammatory cytokines including IL-2, IL-7, IL-10, G-CSF, IP-10, MCP-1, MIP-1A, and $\text{TNF}\alpha$, indicative of the “cytokine storm.” These findings are similar to that seen with the other coronavirus infections, SARS and MERS. The presence of lymphopenia and pro-inflammatory “cytokine storm” have a major role in the damage or pathology of COVID-19. This “cytokine storm” can precipitate overwhelming viral sepsis and inflammation-induced lung injury which lead to the complications of severe pneumonia, acute respiratory distress syndrome (ARDS), respiratory failure, shock, organ failure and potentially death. Death is more likely to occur by multiorgan failure, most often in elderly and those with underlying health conditions, such as, seen in smokers, uncontrolled hypertension, cardiovascular disease, diabetes [10].

Those more severely affected, shift to increased neutrophil type WBCs, continuing reductions in lymphocytes, increased cytokine IL-6, and evidence of increased inflammation with an elevated C-reactive protein (CRP). Increased neutrophils and decreased lymphocytes correlate with disease severity and likelihood of death. Those needing ICU care had higher levels of many cytokines indicative of significant activation of the innate or intracellular immune system.

Increased capillary permeability, due to inflammation, is a hallmark of ARDS. In ARDS, damage of capillaries and alveoli cause impaired fluid removal from the alveolar space and accumulation of protein-rich fluid inside the alveoli, producing even more diffuse alveolar damage and more release of pro-inflammatory cytokines (TNF, IL-1 and IL-6). Neutrophils are recruited to the lungs by the cytokines, become activated and release toxic mediators, such as reactive oxygen species and proteases. Extensive free radical production overwhelms endogenous anti-oxidants and causes oxidative cell damage.

Inflammation due to dramatic neutrophil activation is key in the development of ARDS. NF-kappa B activation also contributes to the pro-inflammatory mediators in the lungs of ARDS patients. In addition, other factors increase blood vessel permeability and destroy micro-vascular structure, enhancing inflammation and lung damage. So, several different pathways are involved in ARDS development. There is no single biomarker that can predict outcome in ARDS patients [11].

Immune changes and phases of infection

Beyond the initial phases of infection, including implantation, incubation and initial viral replication, if the infection progresses, and becomes established as a more severe infection, particularly for hospitalized individuals, the clinical infection can be divided into three phases: viremia, acute phase (pneumonia) and the recovery phase [9]. Viremia means that the virus has expanded into the circulation. If immune function of patients in the acute phase (pneumonia phase) is effective, and there are no other underlying diseases, the virus can be controlled or suppressed and can enter the recovery phase. If the patient is older, which normally means a naturally less effective immune system, or in an immune impaired state, combined with other underlying diseases, such as hypertension, diabetes and cancer, the immune system cannot effectively control the virus in the acute phase (pneumonia phase). In this case, the person can quickly become severely ill or critical. Significant reductions in lymphocytes, especially T cells and B cells, increases in inflammatory cytokines and D-Dimer lead to more severe disease (Figure 3).

Types of immune response

There are basically 2 types of immune response in the body to infection: innate and adaptive [12]. Effective innate immune response against viral infection relies heavily on interferon (IFN) type I (IFN-1) responses and its downstream effects. IFN-1 helps control viral reproduction and also induction of effective adaptive immune response.

Interferons are very important for helping the body to fight viruses. They are a group of proteins made and released by the body's cells in response to the presence of several viruses. Virus-infected cells release IFNs that cause nearby cells to activate their anti-viral defenses, by stimulating production of proteins that prevent a virus from producing and replicating its RNA and DNA [13]. IFNs also activate immune cells, such as virus-fighting, natural killer cells and macrophages; they increase host defenses by getting T-cells to recognize viral antigens (antigen presentation).

There are two types of adaptive responses: the cell-mediated immune response, done by T cells, and the humoral (circulatory) immune response, which is by B cells and antibodies that are activated by the infection.

Innate immune response and progression of infection in SARS-CoV-2 infection

During the SARS-CoV-2 infection process, there is a progression of changes in the immune response and effects on the body. The

white blood cell count (WBC) in peripheral blood in the early stage of the disease is normal or slightly low, and a low lymphocyte count (lymphopenia) is seen. Reduction in B lymphocytes may happen early, which may reduce protective antibody production. In more severely ill patients, lymphocytes are more significantly reduced. Lymphocytes appear to gradually decrease with COVID-19 infection as the disease progresses [5].

Patients with higher levels of IL-6, a pro-inflammatory cytokine, show higher levels of signs of inflammation. This situation likely contributes to the aggravation of the disease around 7-14 days after onset. Higher levels of neutrophil WBCs (possibly indicative of secondary infections), D-Dimer (a reflection of increased clotting), blood urea nitrogen and creatinine (indicative of kidney damage) were seen in those who didn't survive.

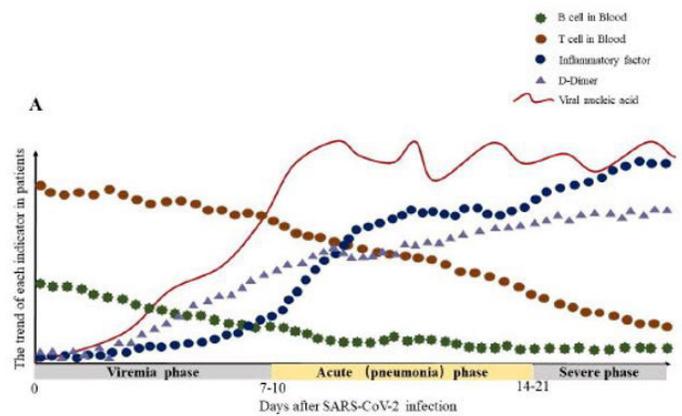


Figure 3: Hypothetical pathogenesis of COVID-19.

The X-axis is the number of days after SARS-CoV-2 infection, and it is divided into three phases – viremia, acute pulmonary phase, and severe phase. The Y-axis is the trend of T cells, B cells, inflammatory factors, D-Dimer and viral load in patients. The trend of each indicator in COVID-19 patients ending with severe disease. From Lin [5].

The virus appears to suppress anti-viral interferon (IFN) responses early on, resulting in uncontrolled viral replication. The degree of dampening of IFN-1 is related to disease severity. This happens through several mechanisms induced by the virus. The IL-10 cytokine, increased by the virus, also further suppresses IFN-1 production.

Decreased IFN levels lead to increased viral replication, which leads to increased influx of hyper-inflammatory neutrophils and monocytes/macrophages. The increases in these innate immune cells results in progressive deterioration and lung immunopathology changes, hence, pneumonia and ARDS.

This early suppression of IFN-1 is a reason why asymptomatic infected individuals can transmit the virus [14]. Interestingly, there are very few COVID-19 cases in young children. They have highly effective innate immunity and may suppress the virus at the very beginning of exposure more effectively.

Once the virus really gains reproductive steam, there appears to be a biphasic hyperproduction of IFN. This may be because cells start to become hyper-reactive to the increased viral load. At this stage, there is also an increased influx of neutrophils and macrophages, the major sources of pro-inflammatory cytokines, contributing to “cytokine storm”.

In the T cell system, specific Th1/Th17 may be activated and contributes to exacerbate inflammatory responses. In the B cell system, B cells/plasma cells produce SARS-CoV-2 specific antibodies that help neutralize viruses.

SARS-Co-V2 not only affects alveolar cells but also also directly infects macrophages and T lymphocyte cells, another key feature in the damage and spread caused by SARS-CoV2. Since macrophages and T cells are recruited to help fight the infection, these infected macrophages and T cells, migrate to the site of infection/inflammation and contribute to the escalating damage caused by the infection.

Adaptive immune response

T lymphocytes are a major source of cytokines. T lymphocytes have specific receptors on their cell surface to allow recognition of foreign pathogen antigens. There are two main subsets of T lymphocytes, known as CD4 and CD8. CD4 type T lymphocytes are helper T cells, and are the most prolific cytokine producers. These CD4 cells are subdivided into Th1 and Th2, and the cytokines they produce are known as Th1-type cytokines and Th2-type cytokines [14]. The Th1 response is key for successful control of SARS and MERS and is probably true for SARS-CoV-2.

The cytokine environment dictates the direction of T cell responses. Helper T cells control the overall adaptive response, while cytotoxic T cells are essential in disposing of viral infected cells. The humoral (circulatory) immune response, by producing neutralizing antibody, protects by limiting infection at later phases and prevents reinfection in the future.

Antibody protection

Antibody development varies with the type of coronavirus infection. SARS-CoV infection induces identifiable antibodies as early as day 4 after onset of disease and in most patients by 14 days [14]. Long lasting virus specific immunoglobulin G (IgG) and neutralizing antibody have been seen as long as 2 years after infection. For MERS-CoV infection, seroconversion is seen at the second or third week of disease onset. For both of these coronavirus infections, delayed and weak antibody response are associated with severe outcome. IgM provides acute, short-term immune protection, while the body develops the long-term protection of IgG. For SARS-CoV-2, one patient showed peak specific IgM at day 9 after onset and IgG by week 2. Five patients with confirmed COVID-19 had some cross-reactivity with SARS-CoV, indicating that there may be some cross-virus species immune protection. In other words, previous exposure to SARS-CoV may infer some protection for COVID-19.

Inflammation due to neutrophil activation is key in the pathogenesis of ARDS. Fundamental transcription abnormalities, involving NF-kappa B, that is required for transcription of genes for many pro-inflammatory mediators, are present in the lungs of ARDS patients. In addition, other factors such as endothelin-1, angiotensin-2 and phospholipase A-2 increase vascular permeability and destroy micro-vascular structure, thereby, enhancing inflammation and lung damage. In conclusion, as several different pathways are involved in ARDS development, there is no single biomarker that can predict outcome in ARDS patients [11].

Immune evasion

Coronaviruses are particularly adapted to evade immune detection and dampen human immune responses. This partly explains why they tend to have a longer incubation period, 2-11 days on average compared to influenza, 1-4 days [14]. The longer incubation period is probably due to their immune evasion properties, efficiently escaping host immune detection at the early stage of infection. They do this mostly by inhibiting innate immune responses, especially by reducing IFN-1 responses. Also, recognition of the virus is lessened because macrophage or immune dendritic tissue cells are infected, leading to markedly diminished helpful T cell activation.

Long term consequences of pulmonary coronavirus infections

There are long-term health consequences of coronavirus infections [15]. One is primarily from lung involvement and the other is from the longer-term use of steroids during infections. It is unknown yet what the long-term consequences of COVID-19 infections will be. There are significant differences between the various coronaviruses and their disease processes. However, ARDS is common to all of them.

In one study [15], reporting on outcomes 15 years after SARS infections, the researchers describe the recovery curve of lung injury. The percent of SARS lung lesions showed improvement from 2003 to 2004, around the time of the infection, and plateaued thereafter from 2004 to 2018. SARS-induced pulmonary lesions originally seen on imaging recovered most within 1 year after rehabilitation. There was a correlation between pulmonary CT images and functional changes. Pulmonary function in 2018 was basically the same as that in 2006, with mildly impaired oxygen/carbon dioxide diffusion function. There was no substantial recovery over that time, despite natural “aging-related” loss of pulmonary function over the ensuing period. This indicates stability of whatever loss occurred early in the recovery period. The pulmonary function of those with normal CT findings after recovering from SARS in 2003 was better than those with abnormalities. The result is that better management of pulmonary damage and therefore function early after recovery should improve long-term pulmonary function.

For those who needed higher dose and longer-term steroids, subchondral osteonecrosis happened in 5%-10% of patients with SARS according to MRI scans after systemic steroid treatment. Higher amounts of cumulative steroid dose increased the risk of

osteonecrosis and therefore the need for hip replacements.

Treatment of COVID-19 infections

There is no known or established successful therapy or preventive strategy yet, specific for COVID-19 disease. Immunization is still projected to be at least a year away. Much research is still in progress.

Chloroquine/hydroxychloroquine has been found to be a potent inhibitor of most coronaviruses, including SARS-CoV1 [16]. Chloroquine has been used since 1934 for the treatment and prophylaxis of malaria. It has been found to have broad-spectrum activity against a range of bacterial, fungal and RNA viral infections. It is also used routinely as an anti-inflammatory agent for the treatment of rheumatoid arthritis and lupus erythematosus.

Following the initial Wuhan outbreaks in 2019, about 100 infected patients have been treated with chloroquine. They had a more rapid decline in fever, faster promotion of becoming virus negative, improvement of lung CT images and clinical recovery compared with control groups. There were no obvious serious adverse effects [17]. Numerous chloroquine for COVID-19 clinical trials are being conducted in China, the USA and elsewhere (www.clinicaltrials.gov).

For COVID-19 disease chloroquine/hydroxychloroquine may not only have antiviral activity but could also act indirectly to reduce pro-inflammatory cytokines and/or by activating anti-SARS-CoV-2 CD8+ T-cells. This is why it is used for autoimmune disorders.

Besides chloroquine, several other drugs such as arbidol, remdesivir, and favipiravir are currently undergoing clinical studies [18]. Both chloroquine and the antiviral drug remdesivir have been reported to inhibit SARS-CoV-2 in vitro. It is likely that a cocktail of drugs may be required to fully impact COVID-19 infections, as is done in the treatment of hepatitis C and HIV. Whether chloroquine may be found to have preventive value is still unknown and is being investigated.

The role of Pulsed Electromagnetic Fields (PEMFs) in helping with COVID-19 infections

Some of the effects of PEMFs on helping the body to fight and prevent viral infections is reviewed in Pawluk [19]. These effects tend to be nonspecific. At this point, there is little research to show that PEMFs can kill viruses, in general, or the coronavirus specifically. However, one study [20] investigated mice with intranasal injection of H1N1 influenza virus and treated with a 2 kHz PEMF at 1 uT, 10 uT, 100 uT, and 1000 uT for 7 days and 30 minutes per day, and compared to a control group. In the control group influenza virus titers in the lungs increased from 800 [days 1 – 2] to 3200 [days 3 – 5], then fell to 1600. In the PEMF treatment groups, the influenza virus titers did not exceed 800 in the 1 uT group, and only 400 and the 10 and 100 uT groups.

Influenza virus antibody levels were 20 in the control group

and up to 320 in the 1 uT group at 640 in the the 10 and 100 uT groups. All these changes were significant at the $p < 0.01$ level. Interestingly, mice in the 1000 uT group had much more severe disease succumbing by the 3rd day after infection. While infected mice seemed to have increased resistance to H1N1 infection with this PEMF treatment system, the results may not be able to be extrapolated either to coronavirus infections or to humans.

For COVID-19 infection, it's worth considering the role of PEMFs in the various phases of infection: as prevention, in the incubation period, in the 3 phases of active COVID-19 infection (viremia, acute phase and recovery), in convalescence and for healing of the infection-damaged tissue.

For use in the incubation phase, PEMFs stimulate phagocytosis [19]. So, once the virus begins to incubate in the nose, routine use of PEMFs before or during the time of invasion may be more likely to impact the proliferation of the virus by lessening its production.

It is unknown how effective PEMFs might be if only initiated during the viremia and acute phase of infection. During any acute inflammatory process, there is an explosion of production of and demand for ATP. PEMFs are known to increase the production of ATP [21]. So, the only possible mechanism I can think of to assist this process, might be the ability of PEMFs to help the body produce more ATP molecules that are required during the acute phase of infection to control inflammation.

PEMFs, by virtue of their action on adenosine receptors during acute inflammation [22], may conceivably enhance the degree of acute inflammation as part of the process of resolution of the infection. That's why in some of the older PEMF literature there is a recommendation for limiting the use of PEMFs in acute infections. So, while it's possible that PEMFs may enhance the acute inflammatory process to get those infected through to resolution faster, there is likely to be some degree of uncomfortable or unacceptable aggravation first. In the setting of sepsis, this accentuation of inflammation may be very undesirable. This is why I generally do not recommend PEMFs during actively developing significant acute infection. From this perspective, PEMFs would be best used by "bracketing" the infection, preventively ahead of it, to decrease the likelihood of it happening, and after some degree of resolution of the acute infection, to speed recovery and repair.

I previously reported that PEMFs have a significant role in decreasing inflammation, and cytokine burden, throughout the body by actions on the adenosine receptor [23]. There is also increased inflammation and production of significant amounts of pro-inflammatory cytokines in the setting of overweight and obesity, leading to a pre-existing cytokine burden [24]. Overweight and obesity are especially common in diabetes and diabetes is a risk factor for increased severity of COVID-19 infection [10]. PEMFs can reduce this cytokine burden.

Because of the general effect of PEMFs on inflammation, regardless of the source and cause, PEMFs should significantly

reduce inflammation associated with coronavirus infections. A recent review [25], discussed the value of PEMFs (or extremely low-frequency magnetic fields – ELF-MFs) for reducing chronic cellular danger signals that lead to inflammation and immune cell activation and for promoting cellular and tissue healing caused by infection.

The adenosine blog makes it clear that an appropriate intensity PEMF is needed to produce optimal reduction of inflammation. The optimal PEMF intensity is 1.5 mT/15 Gauss at the target tissue [26]. The peak intensity of the magnetic field will depend on the amount of lung tissue that needs to be treated. If the problem is primarily in the bronchial passages, the magnetic field intensity, at about 3 – 4 inches into the body needs to be at a minimum of about 2000 Gauss, and preferably 4000 Gauss. If major areas of the lungs are involved, a wider field PEMF system delivering about 5000 Gauss would be needed to be used to both the front and back of the chest, and over both lungs.

PEMFs should be used early and aggressively in the initial, less symptomatic, stages of any viral infection. Once an infection gets to the point of requiring hospitalization, PEMFs are not likely to be allowed into the hospital setting. So, PEMFs should be considered in the rehabilitation or recovery phase at home after hospitalization, even if ARDS happened. The goal will be to reduce the amount of inflammatory damage and scarring of pulmonary, alveolar and bronchial, tissue as the body goes through the recovery and healing process.

For infection preventive purposes, in the light of the coronavirus pandemic, a lower intensity PEMF system of about 200-1000 Gauss, whether portable (DC) or needing current (AC), applied over the thymus would help to stimulate T cells. This is especially important since one of the key aspects of aggressive COVID-19 infection is the initial and progressive reduction in lymphocytes. The thymus is located under the upper part of the sternum inside the chest. It may be worthwhile, although hypothetical, to stimulate the thymus with a PEMF to “stoke” and optimize the immune system, especially during this time of the pandemic.

In addition, routine, daily, systemic/whole body use of PEMFs, prior to any infections, will tend to reduce the inflammatory burden throughout the body and optimize cellular function, through various mechanisms. A healthy body is more likely to resist any viral infections in the first place and, once infected, the body is less likely to have infection progress and produce significant symptoms.

Even long after recovery from COVID-19 infections, PEMFs may still be very important to heal the affected lung tissues to the extent possible to improve air exchange [19]. This is especially true in those with clear scarring on CT or reduced pulmonary function testing. In this situation, it is likely that PEMFs will be needed long-term to maintain lost or damaged function and facilitate longer-term lung healing [27].

Conclusion

The recent and current pandemic of COVID-19 infection has had a major global impact. Since this is a novel viral infection, there are as yet no approved and proven therapeutic strategies. The virus is very infectious and when established is very aggressive in implanting and spreading in the host. The virus reduces host antiviral responses particularly by suppressing interferon (IFN-1) production, facilitating more rapid and invasive infection. Because it impacts the ACE 2 receptor in the Type II cells of the alveoli spread in the lungs is rapid, initiating a huge inflammatory response resulting in the so-called “cytokine storm.” Suppression of the body’s response to invasion leads to rapid multiplication of the virus resulting in circulatory viremia. This viremia can then spread to other ACE 2 receptors in the body resulting in damage to other organs in addition to the lungs. In the lungs this rapidly progressive cascade of events results in acute respiratory distress syndrome (ARDS), which is lethal in about 50% of cases. With viremic sepsis, multiorgan failure can also occur.

Besides supporting the body’s immune system to be as strong as possible with lifestyle and supplement strategies as a background, prior to exposure to the COVID-19 virus, two potential therapeutic approaches may be taken early in the course of infection. One is the controversial use of hydroxychloroquine (HCQ) and the other is the concomitant use of pulsed electromagnetic fields (PEMFs). HCQ is thought to have some antiviral actions and has been used extensively for decades in autoimmune and rheumatologic conditions for its anti-inflammatory properties. So, HCQ could well have dual actions in helping to combat COVID-19 infections.

PEMFs have been shown to have multiple supportive anti-infective and immune supportive actions in the body and may be used as an adjunctive therapy pre-infection, during early infection and facilitating recovery after infection.

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