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Evidence for a Connection between COVID-19 and Exposure to Radiofrequency Radiation from Wireless Telecommunications Including Microwaves and Millimeter Waves

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Abstract: COVID-19 public health policy has focused on the SARS-CoV-2 virus and its effects on human health while environmental factors have been largely ignored. In considering the epidemiological triad (agent-host-environment) applicable to all disease, we investigated a possible environmental factor in the COVID-19 pandemic: ambient radiofrequency radiation from wireless communication systems including microwaves and millimeter waves. COVID-19 surfaced in Wuhan, China shortly after the implementation of city-wide 5G (fifth generation of wireless radiation), and spread globally, demonstrating a statistical correlation to international communities with 5G antennas installed. In this study, we examined the peer-reviewed scientific literature on the detrimental bioeffects of radiofrequency radiation (RFR) and identified several ways in which RFR may be contributing to COVID-19 as a toxic environmental cofactor. We conclude that RFR and, in particular, 5G, which involves 4G infrastructure densification, has exacerbated COVID-19 prevalence and severity by weakening host immunity and increasing SARS-CoV-2 virulence by (1) causing morphologic changes in erythrocytes including echinocyte and rouleaux formation that may be contributing to hypercoagulation; (2) impairing microcirculation and reducing erythrocyte and hemoglobin levels exacerbating hypoxia; (3) amplifying immune system dysfunction, including immunosuppression, autoimmunity, and hyperinflammation; (4) increasing cellular oxidative stress and the production of free radicals exacerbating vascular injury and organ damage; (5) augmenting intracellular Ca^{2+} essential for viral entry, replication, and release, in addition to promoting pro-inflammatory pathways; and (6) worsening heart arrhythmias and cardiac disorders. In short, RFR is a ubiquitous environmental stressor that contributes to adverse health outcomes of COVID-19. We invoke the Precautionary Principle and strongly recommend a moratorium on 5G wireless infrastructure at this crucial time to help mitigate the pandemic, and to preserve public health until governmental safety standards for RFR exposure based on current and future research are defined and employed.

Keywords: coronavirus; COVID-19; electromagnetic stress; EMF; environmental factor; microwave; millimeter wave; pandemic; public health; radiofrequency

Science Publications

Introduction

Background

COVID-19 has been the focus of international public health policy throughout 2020. Despite unprecedented public health protocols to quell the pandemic, the number of COVID-19 cases continues to rise. We propose a reassessment of our public health strategies.

According to the Center for Disease Control and Prevention (CDC, 2020), the simplest model of disease causation is the epidemiological triad consisting of three interactive factors: the agent (pathogen), the environment, and the health status of the host. Extensive research is being done on the agent (SARS-CoV-2). Risk factors that make a host more likely to succumb to the disease have been elucidated. However, environmental factors have not been sufficiently explored. Are there environmental factors involved and actions that we can take to mitigate COVID-19? In this paper, we investigate the role of wireless communication radiation as a widespread environmental stressor.

This is the first comprehensive paper examining scientific evidence that radio-frequency radiation including 5G (fifth generation of wireless communication technology and its necessary earlier generations), henceforth referred to as RFR, may be a contributing factor in COVID-19. RFR from wireless technology has already been recognized as a form of environmental pollution and physiological stressor (Balmori, 2009). Assessing the detrimental health effects of RFR is crucial to develop an effective, rational public health policy that will expedite eradication of the COVID-19 pandemic. In addition, because we are on the verge of worldwide 5G deployment, it is critical to consider the damaging health effects of RFR before it is too late.

5G is a protocol that will use high frequency bands of the electromagnetic spectrum in the vast radiofrequency range from 600 MHz to 90 GHz, which includes millimeter waves, in addition to the currently used 3G and 4G LTE microwave bands. Focused pulsed beams of radiation will emit from new base stations and antennas placed close to homes and schools whenever persons access the 5G network. The system requires significant densification of 4G as well as new 5G antennas that will dramatically increase the population's wireless radiation exposure both inside structures and outdoors. In addition, up to 100,000 satellites are planned to be launched into low earth orbit to achieve a global wireless network. This infrastructure will significantly alter the world's electromagnetic environment to unprecedented levels and may cause unknown consequences to the entire biosphere, including humans.

COVID-19 began in Wuhan, China in December 2019, shortly after city-wide 5G had "gone live" on October 31, 2019. COVID-19 outbreaks soon followed in other areas where 5G had also been at least partially implemented, including South Korea, Northern Italy, New York City, Seattle, and Southern California. In April 2020, Payeras and Cifre (2020) reported a statistically significant correlation between the density of 5G antennas and the location of COVID-19 cases in specific regions throughout the world. During the first wave in the United States, COVID-19 attributed cases and deaths were higher in states with 5G infrastructure compared with states that did not yet have this technology (Tsiang and Havas, manuscript submitted).
There is a large body of peer reviewed literature, since before World War II, on the biological effects of wireless radiation that impact many aspects of our health. In examining this literature, we found intersections between the pathophysiology of COVID-19 and detrimental bioeffects of wireless radiation exposure. Here we present the evidence that RFR is a contributing factor exacerbating COVID-19.

Overview on COVID-19

The clinical presentation of COVID-19 has proven to be highly variable, with a wide range of symptoms and variability from case to case. According to the CDC, early disease symptoms may include sore throat, headache, fever, cough, chills, among others. More severe symptoms including shortness of breath, high fever, and severe fatigue may occur in a later stage. The neurological sequela of taste and smell loss has also been described.

Ing *et al.,* (2020) determined 80% of those affected have mild symptoms or none, but older populations and those with comorbidities, such as hypertension, diabetes, and obesity, have a greater risk for severe disease (Garg *et al.,* 2020). Acute respiratory distress syndrome (ARDS) can rapidly occur (Wu *et al.,* 2020) and cause severe shortness of breath as endothelial cells lining blood vessels and epithelial cells lining airways lose their integrity, and protein rich fluid leaks into adjacent air sacs. COVID-19 can cause insufficient oxygen levels (hypoxia) that has been seen in up to 80% of intensive care unit (ICU) patients (Gattinoni, 2020) exhibiting respiratory distress. Decreased oxygenation and elevated carbon dioxide levels in patients' blood have been observed, although the etiology for these findings remains unclear.

Massive oxidative damage to the lungs has been observed in areas of consolidation documented on lung radiographs and CT scans in patients with COVID-19

pneumonia (Cecchini and Cecchini, 2020). This cellular stress may indicate a biochemical rather than a viral etiology (Cavezzi *et al.*, 2020).

Because disseminated virus can attach itself to cells containing an ACE-2 (angiotensin-converting enzyme 2) receptor, the disease can spread and damage organs and soft tissues throughout the body, including the lungs, heart, intestines, kidneys, blood vessels, fat, testes, and ovaries, among others. The disease can increase systemic inflammation and induce a hypercoagulable state. Without anticoagulation, intravascular blood clots can be devastating (Bikdeli *et al.*, 2020).

In COVID-19 patients referred to as "long-haulers," symptoms can wax and wane for months (Carfi *et al.*, 2020). Shortness of breath, fatigue, joint pain and chest pain can become persistent symptoms. Post-infectious brain fog, cardiac arrhythmia, and new onset hypertension have also been described. Long term chronic complications of COVID-19 are being defined as epidemiological data are collected over time.

As our understanding of COVID-19 continues to evolve, environmental factors, particularly those of wireless electromagnetic fields, remain unexplored variables that may be contributing to the disease including its severity in some patients. Next, we summarize the bioeffects of RFR exposure from the peer reviewed scientific literature published over decades.

Overview on Bioeffects of Radiofrequency Radiation (RFR) Exposure

Organisms are electrochemical beings, and low-level RFR from wireless communication devices, including cell phone antennas, base stations, Wi-Fi, and cell phones, among others, may disrupt regulation of numerous physiological functions. Non-thermal bioeffects (below the power density that causes tissue heating) from very low-level RFR exposure have been reported in thousands of peer-reviewed scientific publications at power densities below the ICNIRP (International Commission on Non-Ionizing Radiation Protection) exposure guidelines (ICNIRP, 2009). Low-level RFR has been found to impact the organism at all levels of organization, from the molecular to the cellular, physiological, behavioral, and psychological levels. Moreover, it has been shown to cause systemic detrimental health effects including increased cancer risk (Bortkiewicz *et al.*, 2017), endocrine changes (Sangun *et al.*, 2016), increased free radical production (Yakymenko *et al.*, 2016), DNA damage (Ruediger, 2009), changes to the reproductive system (Asghari et al., 2016), learning and memory defects (Zhang *et al.*, 2017), and neurological disorders (Pall, 2016). Having evolved within Earth's extremely low-level natural radiofrequency background, organisms lack the ability to adapt to heightened levels of unnatural radiation of wireless technology with digital pulse modulation.

The peer-reviewed world scientific literature has documented evidence for detrimental bioeffects from RFR exposure including 5G frequencies over several decades. The Soviet and Eastern European literature from 1960-70s demonstrates significant biological effects, even at exposure levels 100 times below 1 mW/cm^2, the current guideline for maximum public exposure in the US. Eastern studies on animal and human subjects were performed at low exposure levels (<1 mW/cm^2) for long durations (typically months). By contrast, most US research has been performed over short durations of weeks or less. There have been few long-term studies on animals or humans.

Illness from RFR exposure has been documented since the early use of radar. Prolonged exposure to microwaves and millimeter waves from radar was associated with various disorders termed "radio-wave sickness" decades ago by Russian scientists. A bibliography of over 3,700 references on the reported biological effects in the world scientific literature was published in 1972 (revised 1976) by the US Naval Medical Research Institute report (Glaser, 1972; 1976). The BioInitiative Report (Sage and Carpenter, 2012), authored by 29 experts from 10 countries, and updated in 2020, provides a scholarly contemporary summary of the literature on the bioeffects and health consequences from RFR exposure, including a compendium of supporting research. Recent reviews have been published (Belpomme *et al*., 2018; Di Ciaula, 2018; Russell, 2018; Miller *et al.*, 2019). A comprehensive review on the bioeffects of millimeter waves reports that even short-term exposures produce marked bioeffects (Pakhomov *et al.*, 1998).

Methods

An ongoing literature study of the unfolding pathophysiology of COVID-19 was performed throughout 2020. To investigate a possible connection to bioeffects from RFR exposure, we examined over 250 peer-reviewed research reports from 1969 – 2020, including reviews and studies on cells, animals, and humans. This included the world literature in English and Russian reports translated to English, on RFR from 600 MHz – 90 GHz, the spectrum of wireless communication radiation (2G – 5G inclusive), at non-thermal power densities ($<$ 5 mW/cm^2) and with particular emphasis on low power densities ($<1mW/cm^2$) and long term exposures. The following search terms were used in queries in MEDLINE® and the Defense Technical Information Center (https://discover.dtic.mil) to find relevant study reports: radiofrequency radiation, microwave, millimeter wave, radar, MHz, GHz, blood,

red blood cell, erythrocyte, hemoglobin, hemodynamic, oxygen, hypoxia, vascular, inflammation, pro-inflammatory, immune, lymphocyte, T cell, cytokine, intracellular calcium, sympathetic function, arrhythmia, heart, cardiovascular, oxidative stress, glutathione, ROS (reactive oxygen species), COVID-19, virus, and SARS-CoV-2. Occupational studies on RFR exposed workers were included. From analysis of these studies in comparison with new information unfolding on the pathophysiology of COVID-19, we identified several ways in which adverse bioeffects of RFR exposure intersect with COVID-19 manifestations and organized our findings into five categories.

Results

Table 1 lists the manifestations common to Covid-19 including disease progression and the corresponding adverse bioeffects from RFR exposure. Although these effects are delineated into categories—blood and vascular changes, oxidative stress, immune system disruption, increased Ca^{2+} levels, and cardiac arrhythmias—it must be emphasized that these effects are not independent of each other. For example, blood clotting and inflammation have overlapping mechanisms, and oxidative stress is implicated in erythrocyte morphological changes as well as in hypercoagulation, inflammation, and organ damage.

Table 1. Bioeffects of RFR exposure in relation to COVID-19 manifestations and their progression

RFR Exposure Bioeffects	COVID-19 Manifestations
Blood Changes	Blood and Vascular Changes
Short-term: rouleaux, echinocytes Long-term: reduced blood clotting time, reduced hemoglobin, hemodynamic disorders	Rouleaux, echinocytes Hemoglobin effects; vascular effects → Reduced hemoglobin in severe disease; autoimmune hemolytic anemia; hypoxemia and hypoxia → Endothelial injury; impaired microcirculation; hypercoagulation; disseminated intravascular coagulopathy (DIC); pulmonary embolism; stroke
Oxidative Stress	Oxidative Stress
Glutathione level decrease; free radicals and lipid peroxide increase; superoxide dismutase activity decrease Oxidative injury in tissues and organs	Glutathione level decrease; free radical increase and damage; apoptosis → Oxidative injury; organ damage in severe disease
Immune System Disruption and Activation	Immune System Disruption and Activation
Immune suppression in some studies; immune hyperactivation in other studies Long-term: suppression of T-lymphocytes; inflammatory biomarkers increased; autoimmunity; organ injury	Decreased production of T-lymphocytes; elevated inflammatory biomarkers. → Immune hyperactivation and inflammation; cytokine storm in severe disease; cytokine-induced hypo-perfusion with resulting hypoxia; organ injury; organ failure
Increased Intracellular Calcium	Effects of Increased Intracellular Calcium
From activation of voltage-gated calcium channels on cell membranes, with numerous secondary effects	→ Increased virus entry, replication, and release → Increased NF-kB, pro-inflammatory processes, coagulation, and thrombosis
Cardiac Effects	Cardiac Effects
Up-regulation of sympathetic nervous system; palpitations and arrhythmias	Arrhythmias → Myocarditis; myocardial ischemia; cardiac injury; cardiac failure

Effects on the Blood

RFR exposure can cause morphologic changes in blood readily seen via microscopic examination of peripheral blood samples. Two recent studies documented the formation of erythrocyte aggregates (Havas, 2013) and erythrocyte aggregates and echinocyte formation upon human exposure to 4G-LTE smart phone radiation (microwaves) (Rubik, 2014). One study investigated the effect of cell phone radiation on the peripheral blood of ten human subjects (Rubik, 2014). Exposure to radiation from a cell phone for two consecutive 45-minute periods caused two types of effects: initially increased stickiness of peripheral red blood cells and rouleaux formation (rolls of stacked red blood cells) and subsequently formation of echinocytes (spiky red blood cells).

Similar red blood cell changes have been described in peripheral blood of COVID-19 patients (Lakhdari *et al*., 2020). Rouleaux formation is observed in 1/3 of COVID-19 patients, whereas spherocytes and echinocytes have been observed at variable levels. Rouleaux formation impedes the microcirculation. These blood changes may also impede oxygen transport, contributing to hypoxia, and increase the risk of thrombosis (Wagner *et al.*, 2013) and therefore stroke, which can manifest in COVID-19.

Additional blood effects have been observed in both humans and animals exposed to RFR. In 1977, a Russian study reported that rodents irradiated with 5 - 8 millimeter waves (60 - 37 GHz) at 1mW/cm^2 for 15 minutes/day over 60 days developed hemodynamic disorders, suppressed red blood cell formation, reduced hemoglobin, and an inhibition of oxygen utilization (oxidative phosphorylation by the mitochondria) (Zalyubovskaya, 1977). In 1978, a 3-year Russian study on 72 engineers exposed to millimeter wave (5G RFR) generators emitting at 1 mW/cm^2 or less showed a decrease in their hemoglobin levels and numbers of red blood cells, and a tendency toward hypercoagulation,

whereas a control group showed no changes (Zalyubovskaya and Kiselev, 1978). Such deleterious hematologic effects from RFR exposure may also contribute to the development of hypoxia and blood clotting observed in COVID-19 patients.

It has been proposed that the SARS-CoV-2 virus attacks erythrocytes and causes degradation of hemoglobin (Cavezzi et al., 2020). Viral proteins may attack the 1-beta chain of hemoglobin and capture the porphyrin, along with other proteins from the virus catalyzing the dissociation of iron from heme (Wenzhong and Li, 2020). In principal this would reduce the number of functional erythrocytes and cause the release of free iron ions that could cause oxidative stress, tissue damage, and hypoxia. With hemoglobin partially destroyed and lung tissue damaged by inflammation, patients would be less able to exchange CO_2 and O_2, and would become oxygen depleted. In fact some COVID-19 patients show reduced hemoglobin levels, measuring 7.1 g/L and even as low as 5.9 g/L in severe cases (Lippi and Mattiuzzi, 2020). Clinical studies of almost 100 patients from Wuhan revealed that the hemoglobin levels in the blood of most patients infected with SARS-CoV-2 are significantly lowered resulting in compromised delivery of oxygen to tissues and organs (Chen *et al*., 2020). In a meta-analysis of 4 studies with a total of 1210 patients and 224 with severe disease, hemoglobin values were reduced in COVID-19 patients with severe disease compared to those with milder forms (Lippi and Mattiiuzzi, 2020). In another study on 601 COVID-19 patients, 14.7% of anemic COVID-19 ICU patients and 9% of non-ICU COVID-19 patients had autoimmune hemolytic anemia (Algassin et al., 2020). In patients with severe COVID-19 disease, decreased hemoglobin along with elevated erythrocyte sedimentation rate, C-reactive protein, lactate dehydrogenase, albumin (Ghahramani *et al.,* 2020) , serum ferritin (Cheng *et al*., 2020), and low oxygen saturation (Tobin *et al*., 2020) provide additional support for this hypothesis. In addition, packed red blood cell transfusion may promote recovery of COVID-19 patients with acute respiratory failure (Ejigu *et al*., 2020).

In short, both RFR exposure and COVID-19 can cause deleterious effects on red blood cells and reduced hemoglobin levels contributing to hypoxia in COVID-19. Endothelial injury may also contribute to hypoxia and many of the vascular complications seen in COVID-19 (Varga *et al*., 2020), discussed in the next section.

Oxidative Stress

Oxidative stress is a nonspecific pathological condition reflecting an imbalance between an increased production of ROS and an inability of the organism to detoxify the ROS or to repair the damage they cause to biomolecules and tissues (Betteridge, 2000). Oxidative stress can disrupt cell signaling, cause the formation of stress proteins, and generate free radicals, which are highly reactive and can cause DNA damage.

SARS-CoV-2 inhibits intrinsic pathways designed to reduce ROS levels, thereby increasing morbidity. Immune dysregulation, i.e. the upregulation of interleukin (IL)-6 and tumor necrosis factor α (TNF-α) (Giamarellos-Bourboulis *et al.,* 2020) and suppression of interferon α and interferon β (IFN-α, IFN-β) (Hadjadj *et al.,* 2020) has been identified in the cytokine storm accompanying severe COVID-19 infections and generates oxidative stress (Cecchini and Cecchini, 2020). Oxidative stress and mitochondrial dysfunction may further perpetuate the cytokine storm, worsening tissue damage, and increasing the risk of severe illness and death.

Similarly low-level RFR generates ROS in cells that cause oxidative damage. In fact, oxidative stress is considered as one of the primary mechanisms in which RFR exposure causes cellular damage. Among 100 currently available peer-reviewed studies investigating oxidative effects of low-intensity RFR, 93 studies confirmed that RFR induces oxidative effects in biological systems (Yakymenko *et al*., 2015). RFR is an oxidative agent with a high pathogenic potential especially when exposure is continuous (Dasdag and Akdag, 2016).

Oxidative stress is also an accepted mechanism causing endothelial damage (Higashi *et al.,* 2009). This may manifest in patients with severe COVID-19 in addition to increasing the risk for blood clot formation and worsening hypoxemia (Cecchini and Cecchini, 2020). Low levels of glutathione, the master antioxidant, have been observed in a small group of COVID-19 patients, with the lowest level found in the most severe case (Polonikov, 2020). The finding of low glutathione levels in these patients further supports oxidative stress as a component of this disease (Polonikov, 2020). In fact, glutathione, the major source of sulfhydryl-based antioxidant activity in the human body may be pivotal in COVID-19 (Guloyan et al., 2020). Glutathione deficiency has been proposed as the most likely cause of serious manifestations in COVID-19 (Polonikov, 2020). The most common co-morbidities, hypertension (Marushchak *et al*., 2019); obesity (Choromanska *et al*., 2020); diabetes (Gordon-Strachan *et al.,* 2018); and COPD (Marushchak *et al*., 2019)) support the concept that pre-existing conditions causing low levels of glutathione may work synergistically to create the "perfect storm" for both the respiratory and vascular complications of severe infection. Another paper citing two cases of COVID-19 pneumonia treated successfully with intravenous glutathione also supports this hypothesis (Horowitz *et al.* 2020).

Many studies report oxidative stress in humans exposed to RFR. Peraica *et al.* (2008) found diminished blood levels of glutathione in workers exposed to RFR from radar equipment (10 microW/cm^2 to 10 mW/cm^2; 1.5 –10.9 GHz). Garaj-Vrhovac *et al.* (2011) studied bioeffects following exposure to non-thermal pulsed microwaves from marine radar (3 GHz, 5.5 GHz and 9.4 GHz) and reported reduced glutathione levels and increased malondialdehyde (marker for oxidative stress) in an occupationally exposed group (Garaj-Vrhovac *et al*., 2011). Blood plasma of individuals residing near mobile phone base stations showed significantly reduced glutathione, catalase, and superoxide dismutase levels over unexposed controls (Zothansiama *et al.,* 2017). In a study on human exposure to RFR from cell phones, increased blood levels of lipid peroxide were reported while enzymatic activities of superoxide dismutase and glutathione peroxidase in the red blood cells decreased, indicating oxidative stress from cell phone exposure (Moustafa *et al*., 2001).

In a study on rats exposed to 2450 MHz (wireless router frequency), oxidative stress was implicated in causing red blood cell lysis (hemolysis) (Hassan *et al.*, 2010). In another study, rats exposed to 945 MHz (base station frequency) at 3.67 W/m^2 for 7 hours/day, over 8 days, demonstrated low glutathione levels and increased malondialdehyde and superoxide dismutase enzyme activity, hallmarks for oxidative stress (Yurekli *et al.*, 2006).

There is a correlation between oxidative stress and thrombogenesis (Loscalzo, 2003). ROS can cause endothelial dysfunction and cellular damage. The endothelial lining of the vascular system contains ACE-2 receptors that are targeted by SARS-CoV-2. The resulting endotheliitis can cause luminal narrowing and result in diminished blood flow to downstream structures. Thrombi in arterial structures can further obstruct blood flow causing ischemia and/or infarcts in involved organs, including pulmonary emboli and strokes. Abnormal blood coagulation leading to micro-emboli was a recognized complication early in the history of COVID-19 (Tang *et al*., 2020). Out of 184 ICU COVID-19 patients, 31% showed thrombotic complications (Klok *et al*., 2020). Cardiovascular clotting events are a common cause of COVID-19 deaths (Bikdeli *et al*., 2020). Pulmonary embolism, disseminated intravascular coagulation (DIC), liver, cardiac and renal failure have all been observed in COVID-19 patients (Zaim *et al*., 2020).

Patients with the highest cardiovascular risk factors in COVID-19 include males, the elderly, diabetics, and obese and hypertensive patients. However, increased incidence of strokes in younger patients with COVID-19 has also been described (Yaghi *et al.,* 2020).

Oxidative stress is caused by RFR exposure and is known to be implicated in cardiovascular disease. Ubiquitous environmental exposure to RFR may contribute to cardiovascular disease by creating a chronic state of oxidative stress (Bandara and Weller, 2017). This would lead to oxidative damage to cellular constituents and alter signal transduction pathways. In addition, pulse-modulated RFR can cause oxidative injury in liver, lung, testis, and heart tissues mediated by lipid peroxidation, increased levels of NOx, and suppression of the antioxidant defense mechanism (Esmekaya *et al.*, 2011).

In summary, oxidative stress is a major component in the pathophysiology of COVID-19 as well as in cellular damage caused by RFR exposure. Similar effects are observed in both that are caused by increased free radical formation and glutathione deficiency.

The Immune Response

When SARS-CoV-2 first infects the human body, it attacks cells lining the nose, throat, and upper airway harboring an ACE-2 receptor. Once the virus gains access to a cell via its spike protein, it converts the cell into a self-replicating machine. In response to COVID-19 infection, both an immediate systemic innate immune response as well as a delayed adaptive response has been shown to occur (Cao, 2020). The virus can also cause a dysregulation of the immune response, particularly in the decreased production of T- lymphocytes (Qin *et al.*, 2020). Severe cases tend to have lower lymphocyte counts, higher leukocyte counts and neutrophil-lymphocyte ratios, as well as lower percentages of monocytes, eosinophils, and basophils (Qin *et al.*, 2020). Severe cases of COVID-19 show the greatest impairment in T- lymphocytes.

In comparison, low-level RFR studies on laboratory animals also show impaired immune function (McCree, 1980). Findings include physical alterations in immune cells, a degradation of immunological responses, inflammation, and tissue damage. Baranski (1971) exposed guinea pigs and rabbits to continuous or pulse-modulated 3,000 MHz microwaves at an average power density of 3.5 mW/cm^2 for 3 hours/day over 3 months and found nonthermal changes in lymphocyte counts, abnormalities in nuclear structure, and mitosis in the erythroblastic cell series in the bone marrow and in lymphoid cells in lymph nodes and spleen. Other investigators have shown diminished T-lymphocytes or suppressed immune function in animals exposed to RFR. Rabbits exposed to 2.1GHz at 5mW/cm^2 for 3 hours/day, 6 days/week, for 3 months, showed suppression of T-lymphocytes (Nageswari *et al*., 1991). Rats exposed to 2.45 GHz and 9.7 GHz for 2 hours/day, 7 days/week, for 21 months showed a significant decrease in the levels of

lymphocytes and an increase in mortality at 25 months in the irradiated group (Adang *et al.*, 2009). Lymphocytes harvested from rabbits irradiated with 2.45 GHz for 23 hours/day for 6 months show a significant suppression in immune response to a mitogen (McRee *et al.*, 1980).

In the acute phase of COVID-19 infection, blood tests demonstrate elevated erythrocyte sedimentation rate (ESR), C-reactive protein, and other elevated inflammatory markers (Zhou *et al.*, 2020), typical for an innate immune response. Rapid viral replication can cause death of epithelial and endothelial cells and result in leaky blood vessels and pro-inflammatory cytokine release (Yang, 2020). Cytokines, proteins, peptides and proteoglycans that modulate the body's immune response, are modestly elevated in patients with mild-to-moderate disease severity (Upadhyay *et al*, 2020). In those with severe disease, an uncontrolled release of pro-inflammatory cytokines--a cytokine storm--can occur. Cytokine storms originate from an imbalance in T-cell activation with dysregulated release of interleukin (IL)-6, IL-17, and other cytokines. Programmed cell death (apoptosis), ARDS, disseminated intravascular coagulopathy (DIC), and multi-organ system failure can all result from a cytokine storm and increase the risk of mortality.

By comparison, Soviet researchers found in the 1970s that RFR irradiation can damage the immune system of animals. Shandala (1977) exposed rats to 500 microW/cm^2 microwaves for 1 month, 7 hours per day, and found impaired immune competence and induction of autoimmune disease. Rats irradiated with 2.45 GHz at 500 microwatts/cm^2 for 7 hours daily for 30 days produced autoimmune reactions, and 100-500 microwatts/cm^2 produced persistent pathological immune reactions (Grigoriev *et al.*, 2010). Exposure to microwave radiation, even at low levels (100-500 microwatts/cm^2), can impair immune function, causing physical alterations in the essential cells of the immune system and a degradation of immunologic responses (Grigoriev, 2012). Szabo *et al.* (2001) examined the effects of 61.2 GHz exposure on epidermal keratinocytes and found an increase in IL-1b, a pro-inflammatory cytokine. Makar *et al.* (2003) found that immunosuppressed mice irradiated 30 minutes/day for 3 days by 42.2 GHz showed increased levels of tumor necrosis factor-alpha, a cytokine produced by macrophages.

In short, COVID-19 can lead to immune dysregulation as well as cytokine storm. By comparison, exposure to low-level RFR as observed in animal studies can also compromise the immune system, with chronic daily exposure producing immunosuppression or immune dysregulation including hyperactivation.

Intracellular Calcium Levels

Low-level nonthermal RFR exposure leads to increased intracellular Ca^{2+} via the activation of voltage-gated calcium channels (Pall, 2013), which is considered to be one of the primary mechanisms of action of RFR on organisms. Intracellular Ca^{2+} is also essential for virus entry, replication, and release, and it has been reported that viruses hijack calcium channels and increase intracellular Ca^{2+} (Chen *et al.*, 2019). Even though direct evidence has not been reported, there is indirect evidence that increased intracellular Ca^{2+} may be involved in COVID-19. In a recent study, elderly hospitalized COVID-19 patients treated with calcium channel blockers (CCBs amlodipine or nifedipine) were more likely to survive and less likely to require intubation or mechanical ventilation than controls (Solaimanzadeh, 2020). Furthermore, CCB drugs strongly limit SARS-CoV-2 entry and infection in cultured epithelial lung cells (Straus *et al.*, 2020). CCBs also block the increase of intracellular Ca^{2+} caused by RFR exposure (Pall, 2013).

Intracellular Ca^{2+} is a ubiquitous second messenger involved in numerous biochemical processes. Increased intracellular Ca^{2+} is a significant factor in up-regulation of transcription nuclear factor kB (NF-kB) (Sen *et al.*, 1996), an important regulator of pro-inflammatory cytokine production as well as coagulation and thrombic cascades. NF-kB is hypothesized to be a key factor underlying severe clinical manifestations of COVID-19 (Do *et al.*, 2020).

In short, RFR exposure may enhance the infectivity of the virus by increasing intracellular Ca^{2+} that may also indirectly contribute to inflammatory processes and thrombosis.

Heart Disease and Arrhythmias

Cardiac arrhythmias are more commonly encountered in critically ill patients with COVID-19 (Atri *et al.*, 2020). The cause for arrhythmia in COVID-19 patients is multifactorial and includes cardiac and extra-cardiac processes (Dherange et al, 2020). Direct infection of the heart muscle by SARS-CoV-19 causing myocarditis, myocardial ischemia caused by a variety of etiologies, and heart strain secondary to pulmonary or systemic hypertension can result in cardiac arrhythmia. Hypoxemia caused by diffuse pneumonia, ARDS, or extensive pulmonary emboli represent extra-cardiac causes of arrhythmia. Electrolyte imbalances, intravascular fluid imbalance, and side effects from pharmacologic regimens can also result in arrhythmias in COVID-19 patients. Patients admitted to the ICU have been shown to have a higher increase in cardiac arrhythmias, 16.5% in one study (Colon *et al.*, 2020).

Although no correlation between electromagnetic fields (EMFs) and arrhythmia in COVID-19 patients has been described in the literature, many ICUs are equipped with wireless patient monitoring equipment and communication devices producing a wide range of EMF pollution (Gokmen *et al*., 2016).

COVID-19 patients commonly show increased levels of cardiac troponin, indicating damage to the heart muscle (Sandoval *et al.*, 2020). Cardiac damage has been associated with arrhythmias and increased mortality. Cardiac injury is thought to be more often secondary to pulmonary emboli and viral sepsis, but direct infection of the heart, i.e. myocarditis, can occur through direct viral binding to ACE-2 receptors on cardiac pericytes, affecting local and regional cardiac blood flow (Chen *et al*., 2020).

Immune system activation along with alterations in the immune system may result in plaque instability, contributing to development of acute coronary events and cardiovascular disease in COVID-19.

Regarding RFR exposure bioeffects, in 1969 Christopher Dodge of the Biosciences Division, U.S. Naval Observatory in Washington DC, reviewed 54 papers and reported that RFR can adversely affects all major systems of the body, including impeding blood circulation; altering blood pressure and heart rate; affecting electrocardiograph readings; and causing chest pain and heart palpitations (Dodge, 1969). In the 1970s Glaser reviewed more than 2000 publications on RFR exposure bioeffects and concluded that microwave radiation can alter the ECG (electrocardiogram), cause chest pain, hypercoagulation, thrombosis, and hypertension in addition to myocardial infarction (Glaser, 1971; 1976). Seizures, convulsions, and alteration of the autonomic nervous system response (increased sympathetic stress response) have also been observed.

Since then many other researchers have concluded that RFR exposure can affect the cardiovascular system. Potekhina *et al*. (1992) found that certain frequencies (55 GHz; 73 GHz) caused pronounced arrhythmia. In 1997, a review reported that some investigators discovered cardiovascular changes including arrhythmias in humans from long-term low-level exposure to RFR including microwaves (Jauchem, 1997). However, the literature also shows some unconfirmed findings as well as some contradictory findings (Black and Heynick, 2003). Havas *et al.* (2010) reported that human subjects in a controlled, double-blinded study were hyper-reactive when exposed to 2.45 GHz, developing either an arrhythmia or tachycardia and up-regulation of the sympathetic nervous system, which is associated with the stress response. Saili *et al.*, (2015) found that exposure to Wi-Fi affects heart rhythm, blood pressure, and the efficacy of catecholamines on the cardiovascular system, indicating that RFR can act directly and/or indirectly on the cardiovascular system. Most recently, Bandara and Weller (2017) present evidence that people who live near radar installations (millimeter waves: 5G frequencies) have a greater risk of developing cancer and experiencing heart attacks. Similarly, those occupationally exposed have a greater risk of coronary heart disease. Microwave radiation affects the heart, and some people are more vulnerable if they have an underlying heart abnormality (Cleary, 1969).

In short, both COVID-19 and RFR exposure can affect the heart and cardiovascular system, directly and/or indirectly.

Discussion

According to the CDC and epidemiology, there are multiple causal factors that underlie disease, including environmental factors and the health status of the host. Evidence from the literature summarized here suggests a connection between several adverse health effects of RFR exposure and the clinical course of COVID-19. The evidence indicates that RFR may weaken the host, exacerbate COVID-19 disease, and thereby worsen the pandemic.

This evidence presented here does not claim causation. Clearly COVID-19 occurs in regions with little wireless communication. In addition, the relative morbidity caused by RFR exposure in COVID-19 is unknown. The question of causation could be investigated in controlled laboratory experiments.

Human exposure to ambient RFR has significantly increased in 2020 as a side effect to the pandemic. Stay-at-home measures designed to reduce the spread of COVID-19 have inadvertently resulted in greater public exposure to RFR, as people carry out work and school related activities via wireless communication. Telemedicine creates another source of RFR exposure. Even hospital inpatients, particular ICU patients, experience increased RFR exposure as new monitoring devices utilize wireless communication systems that may exacerbate COVID-19 and other health disorders.

The bioeffects of RFR exposure are typically nonlinear rather than exhibiting the familiar linear dose-response effects from biochemicals. RFR bioeffects depend upon specific values of wave parameters including frequency, power density, exposure time, and modulation, as well as the history of exposure. Importantly, RFR bioeffects can involve “response windows” of specific parameters whereby extremely low level fields can have disproportionally detrimental effects (Blackman *et al.*, 1989). This nonlinearity of RFR bioeffects can result in biphasic responses such as immune suppression from one set of parameters, and

immune hyperactivation from another set of parameters, as described herein.

RFR is a widespread, yet often neglected, environmental stressor that can produce a wide range of adverse bioeffects. For decades, independent international research scientists have emphasized the health risks and cumulative damage caused by wireless radiation (Sage and Carpenter, 2012; Russell, 2018). Our findings here are consistent with a large body of established research.

Some of the reported results on bioeffects from RFR exposure seem inconsistent but they are not always true replications. There may be small differences in methods, including unreported details such as the history of exposure of the organisms, or non-uniform body exposure. Additionally, industry-sponsored studies tend to show less adverse bioeffects than studies conducted by independent researchers, suggesting industry bias (Huss *et al.*, 2007). Yet studies employing real-life exposures from commercially available devices display high consistency in showing adverse effects (Panagopoulos, 2019). In gathering papers and examining existing data in this study, we looked for outcomes providing evidence to support a connection between the bioeffects of RFR exposure and COVID-19. We did not make an exhaustive search or attempt to weigh the evidence. The RFR exposure literature is extensive and currently contains over 30,000 research reports dating back several decades.

The telecommunication industry claims that 5G is safe because it complies with current RFR exposure guidelines. However, these guidelines were established in 1996, are antiquated, and are not safety standards. No scientific analyses of the health effects of 5G have been done, and none are currently planned by the industry. 5G networks would expose the public to RFR at an unprecedented scale. With the planned "internet of things," the entire environment of our planet will be drastically changed by unnatural, digitally pulse-modulated microwaves and millimeter waves with power densities trillions of times above the natural background. The long-term effects of this global experiment on humans and the biosphere are unknown.

When a course of action raises threats of harm to human health, precautionary measures should be taken, even if clear causal relationships are not yet fully established. Therefore, we must apply the Precautionary Principle (Kriebel *et al.*, 2001) regarding wireless 5G. The authors urge policymakers to execute an immediate worldwide moratorium on wireless 5G infrastructure until its safety can be assured.

Several unresolved safety issues must be addressed before wireless 5G is further implemented. Questions have been raised about 60 GHz, a key 5G frequency planned for extensive use that is a resonant frequency of the oxygen molecule (Tretyakov *et al.*, 2005). It is possible that adverse bioeffects might ensue from oxygen absorption of 60 GHz. Bioeffects from prolonged RFR exposure of the whole body need to be investigated in animal and human studies, and long-term exposure guidelines need to be considered. Independent scientists need to conduct concerted research to determine, once and for all, the biological effects of real-world exposure to RFR frequencies carrying digitally pulsed signals. Testing could also include real-life exposures to multiple toxins (chemical and biological) (Kostoff *et al.*, 2020). Environmental impact assessments are also needed. Once we understand the long-term biological effects of wireless 5G, we can set clear safety standards of public exposure limits and design an appropriate strategy for safe deployment.

Conclusion

We conclude that RFR and, in particular, 5G, which involves densification of 4G, has exacerbated the COVID-19 pandemic by weakening host immunity and increasing SARS-CoV-2 virulence by (1) causing morphologic changes in erythrocytes including echinocyte and rouleaux formation that may be contributing to hypercoagulation; (2) impairing microcirculation and reducing erythrocyte and hemoglobin levels exacerbating hypoxia; (3) amplifying immune dysfunction, including immunosuppression, autoimmunity, and hyperinflammation; (4) increasing cellular oxidative stress and the production of free radicals exacerbating vascular injury and organ damage; (5) augmenting intracellular Ca^{2+} essential for viral entry, replication, and release, in addition to promoting pro-inflammatory pathways; and (6) worsening heart arrhythmias and cardiac disorders. In short, wireless communication radiation is a ubiquitous environmental stressor, and evidence presented here suggests that it is a contributing factor in the COVID-19 pandemic.

This is the first scientific paper documenting a link between RFR emitted by wireless communication devices and COVID-19. Healthcare workers and policymakers should consider RFR as an environmental cofactor exacerbating the COVID-19 pandemic. Methods for reducing exposure to RFR should be provided to all patients and the general population.

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Author Contribution

Both authors made a substantial intellectual contribution to the work. Beverly Rubik searched and examined the RFR exposure literature and wrote most of the initial draft. Robert Brown contributed much of the material on COVID-19 and contributed to writing and editing the manuscript.

Author Disclosure

The authors declare that they have no conflicts of interest in preparing and publishing this manuscript. No competing financial interests exist.

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