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COVID-19 and the Unraveling of Experimental Medicine - Part III

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Abstract

In the first two segments of our COVID-19 trilogy we examined the failure of the scientists and policy-makers to favorably alter dynamics of the SARS-CoV-2 pandemic. Containment policies such as lockdowns and closure of businesses, which came with great social and economic costs, had no meaningful impact on morbidity or mortality. The mRNA vaccines were an unqualified disaster: they neither halted viral spread nor conferred herd immunity and, in their wake, spawned unacceptably high morbidity and mortality rates: to data there have been approximately 1,183,493 COVID-19 vaccine-related adverse event reports in the US-based Vaccine Adverse Event Reporting System (VAERS) including 25,641 deaths. Globally, this translates to about 23.67 million adverse events and about 512,820 deaths. Medical science has unleashed yet another mass casualty event which will likely surpass any of the pharmacologically-induced tragedies of the 20th century.

In this third part we examine the path not taken: a handful of cheap, widely available, home-based therapies—ozone preconditioning, hydroxychloroquine, and light/vitamin treatment—which, had they been implemented early in the pandemic could have reduced morbidity and mortality by 80% or more. We estimate these interventions could have prevented about 4.8 million deaths globally and 768,000 in the US and in the process put an early end to the pandemic. Contrary to claims made by COVID-19 czar Anthony Fauci, there is an abundance of evidence in the medical literature in support of the very treatments he rejected out-of-hand. Moreover, the evidence was present well before the pandemic but was ignored by medical scientists. We conclude by discussing implications of the fraudulent mRNA vaccine scheme and the dark web of manipulation and disinformation promulgated by those who sponsored this dangerous and ill-conceived experiment. The pandemic sounds a clarion call mandating widescale reform of the healthcare system, medical-industrial complex, and their incestuous relationship with governmental and academic oversight bodies.

Introduction

In the first two segments of our COVID-19 trilogy we examined the abysmal failure of the science community and policy-makers to curtail the dynamics of the SARS-CoV-2 pandemic. Mitigation or containment strategies, which came with great social and economic costs, had no meaningful impact on morbidity or mortality. The mRNA vaccines were an unqualified disaster: they neither halted viral spread nor conferred herd immunity and, in their wake, spawned a laundry list of disabling side effects. In the process, medical science has unleashed yet another mass casualty event which, in all likelihood, will surpass any of the pharmacologically-induced tragedies of the 20th century.

One of the profoundly disturbing aspects of the pandemic was suppression of views that ran contrary to the science narrative. Social media outlets such as YouTube, Facebook and Twitter censored alternative content. News networks like CNN incessantly reported biased pro-vaccine accounts while ignoring the accumulating mass of counterfactual evidence that began to surface in the summer of 2021. As a consequence, they disseminated a fog of disinformation.

*Hyperlink to 1,366 references for COVID-19 vaccine associated complications:
https://www.thegms.co/publichealth/pubheal-rw-22042302-references.pdf

Not only did such tactics undermine basic scientific and democratic principles but, as we shall see, greatly amplified the carnage of the pandemic and cost many more lives.

In this third part of the series we examine the path not taken: a handful of cheap, widely available, home-based therapies which, had they been implemented in a timely manner, especially in the early months of the pandemic before vaccines were even available, could have drastically reduced morbidity and mortality—by up to 80% and probably more. Contrary to claims made by authoritative voices, there was and is an abundance of evidence in the medical literature in support of the very treatments that mainstream medicine rejected.

We conclude by discussing implications of the fraudulent mRNA vaccine scheme and the dark web of manipulation and disinformation promulgated by those who sponsored this dangerous and poorly conceived experiment. The hidden subtext revolves around betrayal of public trust. The pandemic sounds a clarion call mandating widescale reform of the healthcare system, medical-industrial complex, and their incestuous relationship with governmental and academic oversight bodies.

Before the Storm

We have defined the tendency to become infected with SARS-CoV-2 and to express symptoms as a state of susceptibility or, conversely, lack of resistance. Such susceptibility takes origin in the deficient functions of the immune system as a result of its inability to contain and incapacitate the virus. For most of the 20th century immune protective functions were regarded to be secondary to the synthesis and release of neutralizing antibodies. As we have seen, however, the antibody response is far downstream from the primary locus of function which resides in the phagocytic activity of cells like macrophages and neutrophils. For this reason, early immunologists like Metchnikoff and Bordet conceived immune function as part of an organized internal digestive system.

The ability of the phagocytic system to contain SARS-CoV-2 in the interstitial fluid space is the crucial determinant that distinguishes asymptomatic viral invasion from full-blown infection. Once phagocytic barrier functions have been breached systemic activation of the immune response by the cytokine system ensues along with symptoms like fever, fatigue, weakness, cough, shortness of breath, body aches and more. One observes varying states of susceptibility related to such cellular functions across the age spectrum in the population.

According to a 2021 Centers for Disease Control (CDC) report there is marked age-related risk stratification for death secondary to COVID-19 infection. Data indicate that the mortality rate in the 0-17 year range is only about 0.002% or 20 per million. When infected, children usually have mild symptoms and are more likely to be asymptomatic. This same population typically has lower antibody responses [1-7]. Mortality risk jumps by nearly 25-fold in the 18-49 year range to about 0.05% or 500/million; by 300-fold in the 50-64 year group to 0.6% or 6,000/million; and, astonishingly, by 4500-fold in the 65+ year range to about 9% or 90,000/million [8]. Clearly, risk is not spread evenly across the population.

By the same token across all age groups we observe a markedly heightened risk for severe COVID-19 disease and death in those with pre-existing chronic conditions like diabetes, hypertension, obesity, heart disease, renal disease, cirrhosis, COPD, cancer, and frailty [9-21]. Individuals with such conditions are more likely to require hospitalization, have longer hospital stays, be admitted to the ICU, require mechanical ventilation, and experience various organ failure syndromes. Having a single co-morbidity like diabetes or hypertension raises the risk for adverse COVID-19 outcomes by up to 2-3-fold depending on the study. And as we have seen, those with severe disease tend to express higher antibody levels [22-28].

In part I of the series we provided evidence linking such adverse outcomes to impaired intracellular digestion, i.e., autophagy, which, in reality, represents the basis for what scientists call immunity. Intracellular digestion, a lysosomal function, is a highly conserved activity shared by all cells which has evolved into a specialized function in phagocytic immune cells. Autophagy consists of a series of acid-dependent, energy-driven molecular pathways that evolved for the purpose of degradation and recycling of aged and dysfunctional cellular structures. Impairment of autophagy precedes a plethora of pathologic states like inflammation, aging, metabolic disorders, neurodegenerative, cardiovascular and renal diseases as well as cancers [29-34].
Autophagic function is highly dependent on the continued

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availability of aerobic energy substrates like NADPH and ATP and when mitochondrial dysfunction occurs, as during periods of oxidative stress or inflammation, degradative and recycling functions are impaired. Oxidative stress diminishes mitochondrial function leading to buildup of reactive oxygen species and acids, NLRP3 inflammasome formation, and initiation of the so-called cytokine storm [35-45]. All such intracellular stress-related disturbances are present in full-blown COVID-19 infection [46-63].

In part I and in multiple earlier papers, we demonstrated the presence of a blood-borne energy field generated by the systolic and diastolic motions of the heart. Energy substance flows through the blood and interstitial fluid compartment and, ultimately, is transported by ion channel mechanisms across cell membranes into the cytoplasm. Interruption of energy flow triggers mitochondrial dysfunction, oxidative stress, and the inflammatory response. The critical phase of the cardiac cycle is diastole during which magnetic energy is drawn into the blood and produces the outward motion of the ventricles and arterial walls. It is not at all coincidental that diastolic dysfunction is associated with virtually all the same chronic disorders as seen with disturbances in cellular autophagy [64-99].

Clinical phenomena associated with COVID-19 infection are mediated by widespread inflammation originating in the vascular endothelium resulting in impaired diastolic function and energy generation which, in turn, diminishes intracellular digestion by phagocytes. The energy deficit also results in formation of autoantibodies that cross-react with the body's own components to promote cell death with spillage of contents like nucleic acids into the interstitial fluid space. As a consequence, neutrophil extracellular traps, large agglomerates of cellular debris, accumulate which further amplify the spiral of deterioration. Autoimmune mechanisms induce platelets to release clot-promoting substances that induce widespread intravascular thrombosis further compromising organ function. All these events are downstream from a primary energy deficit.

In an earlier series of papers, we established the existence of a complex body-wide energy field driven by aether, the all-encompassing energetic precursor substance first described by Aristotle nearly 2400 years ago [100-103]. The aether concept was rejected by physicists at the turn of the 20th century but in recent decades has been recognized to be indispensable in explaining a multitude of energy-related phenomena. We described three intertwined and interconvertible primary energy forms in living bodies: the magnetic, taking origin in the vascular system; the radiant, deriving from external sunlight, generated in the interstitial fluid space beneath the skin; and the dielectric, in play at the cellular and molecular level and mediated primarily by electro-ionic mechanisms.

Given the primacy of energy metabolism in the economy of living bodies and the inescapable relation between energy deficiency and the pathologic alterations of COVID-19, i.e., diastolic dysfunction, mitochondrial dysfunction, inflammation, impaired autophagy, immune dysfunction, and hypercoagulability, it is obvious that in all cases the primary therapeutic strategy must be replenishment of the energy debt.

By the same token, the earliest manifestation of the syndrome, constitutional symptoms like fatigue, weakness or fever, are premonitory signs of energy depletion. At this juncture the gap between susceptibility and resistance must be bridged: all subsequent developments of COVID-19 infection are only expressions of a progressive and mounting energy debt.

By the time hospital-based treatments are initiated, often 10-12 days after symptom onset, individuals are in more advanced energy depletion. Customary treatments like anti-coagulants, steroids, immune-suppressives or anti-virals block downstream effects but fail to address the primary energy shortage. Based on such considerations, it is axiomatic that all therapeutic efforts should be initiated as early as possible while individuals still have adequate energy reserves to mount a restorative response. Repletion of energy flow corrects the cellular disturbances, augments autophagy and phagocytosis and, as a consequence, enhances resistance.

Ozone Preconditioning

Of the therapeutic approaches intended to augment energy flow and boost resistance ozone has perhaps the most impressive résumé. It ties into a fascinating episode in 20th century molecular biology that highlights yet another failure of the science community to integrate and synthesize its own experimental evidence into a coherent body of knowledge.

In the mid-1980s Charles Murry and colleagues,

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seeking to unravel pathologic mechanisms of heart attack, conducted an experiment to determine whether intermittently reopening the coronary arteries to allow for brief return of blood flow altered the course of cellular injury [104]. In a control group of dogs, a coronary artery was clamped for 40 minutes to assess the extent of infarct damage. Another group underwent a series of four 5-minute arterial occlusions interrupted by 5-minute intervals of reperfusion. Afterward the artery was clamped for 40 minutes. To their astonishment, animals that received preconditioning (PC) pulses had only about 25% of damage as the control group.

Preconditioning is now regarded as the most powerful innate form of protection ever discovered. It has game-changing potential and points to novel ways of addressing diseases science has been attempting to solve for decades with little or no success. In over 35 years since its discovery tens of thousands of reports have appeared in the research literature detailing its various aspects and yet, to date, scientists are unable to explain its basis.

The protection afforded by the PC phenomenon has been widely substantiated. When the PC sequence is applied prior to a prolonged ischemic episode a 2-3h period of protection ensues during which ischemia-mediated damage is markedly diminished. Biochemical analysis indicates that PC enhances mitochondrial function and reduces acid formation. Surprisingly, cardiovascular functions like endothelial-dependent vasodilation are preserved and the myocardium becomes resistant to potentially lethal cardiac arrhythmias [105-114].

Some scientists argued that ATP-sensitive potassium channels mediate the effect but studies were inconclusive. Others argued that nitric oxide is a key player while still others pointed at oxygen-derived free radicals. Various substances such as neurotransmitters, erythropoietin, and heat shock proteins have all been proposed but to date no convincing molecular explanation for the PC phenomenon has come to light [115-125].

A 1993 study found that PC pulses applied in one vascular territory of the heart protected the rest of the heart from prolonged arterial occlusion [126]. Researchers hypothesized that protection was induced by factor(s) 'activated, produced, or transported throughout the heart' by brief periods of ischemia. Several years later another study found reduction in myocardial infarct size in rabbits after administration of PC pulses to skeletal muscle [127]. Now called remote preconditioning, it implicates a body-wide causal nexus that, to date, has resisted all explanatory attempts based on molecular and cellular mechanisms.

Reports soon followed describing protection after PC pulses in multiple organs besides the heart, including the brain, liver, intestines, kidneys, stomach and lungs [128-141]. Preconditioning pulses applied to any vascular bed confer body-wide resistance to prolonged ischemia. The PC response, quite clearly, originates in the cardiovascular system and blood and diffuses throughout the body. Reports suggest beneficial effects are transferable from one animal to another by transfusion of blood or bodily fluids [142-144].

In 1996 a study reporting a complex temporal signature to the PC phenomenon confounded matters even more [145]. The initial period of heightened resistance to ischemic injury disappears after about 2-3h but then protective effects recur in echo-like fashion about 24h later and persist for up to 48-72h. Called the second window of protection, effects are associated with the appearance of various mediator substances in the blood and thus is believed to reflect enhanced gene transcription [146, 147]. Such gene activation is driven by an influx of energy into the cell.

Over the years it became recognized that the PC response could be induced by physiologic means such as hyperthermia, exercise, cardiac pacing or, conversely, by pharmacologic substances like ethanol, volatile anesthetics, and various toxins [148-166]. This is where ozone, possibly the most powerful PC agent yet discovered, comes into play. Through the studied effects of ozone, the enigma of the PC phenomenon has finally been resolved. We described the ozone-mediated PC response in greater detail in an earlier piece [167].

Ozone, tri-atomic oxygen (O_3), a toxic environmental gas, was recognized nearly 200 years ago in areas surrounding lightning strikes and referred to as 'the smell of lightning.' When in excess in atmospheric air ozone produces difficulty in breathing, cough, nasal congestion, tear formation, chest discomfort and, in susceptible individuals, predisposes to asthma attacks, chest pain and occasional heart attack. A powerful oxidant, ozone diverts energy intended for cellular use resulting in impaired

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mitochondrial function, diminished ATP synthesis, production of reactive oxygen species and a host of toxic intermediary compounds.

In the 1970s sporadic reports attributed paradoxical beneficial effects to ozone in various diseases. In the late 1980s studies described beneficial results in HIV patients [168-171]. Later reports described enhanced immune function [172-181]. The list of disorders that responded favorably to ozone treatment grew dramatically: autoimmune conditions, heart disease, peripheral vascular disease, fibromyalgia, neurodegenerative diseases, renal and gastrointestinal disorders, various cancers, healing of wounds chronic pain and more [182-236]. Reports describe beneficial effects in COVID-19 infection [237-254].

PC comprises two opposing aspects: the immediate consequences of the toxic insult followed by the protective response initiated to counteract its noxious influence. Once in contact with body fluids, ozone, 10-15X more soluble in water than diatomic oxygen (O_2), immediately solubilizes. Dissolved ozone is an *über* energy sink that draws electron-equivalents from biomolecules leaving them in a depleted (oxidized) state. This results in conversion of lipids in plasma and cell membranes into various oxidation products which, in turn, lead to formation of reactive oxygen species and intracellular injury [255-263]. Such brief insults are then counterpoised by a striking release of energy into the blood that results in the initial 2-3h window of PC protection.

Blood cells, in particular erythrocytes (RBCs), are among the first to experience ozone's oxidative effects and mount a response. Highly metabolically active, RBCs form a large part of the blood with an estimated mass of up to 5 pounds (2.3kg) in an average adult. Upon contact with ozonated fluid, RBCs undergo a decrease in energy production, in the 5-25% range over about 15-20 minutes, and then respond with a dramatic rebound surge in energy release along with an outpouring of antioxidant compounds. Ozone induces up-regulation of enzymes in RBCs resulting in enhanced production of NADPH and ATP with energy infusion into the blood and neutralization of the detrimental oxidizing effects of ozone [264-268].

Heightened energy output by the RBC mass translates directly into increased blood flow and energy delivery to peripheral tissues. RBCs release large amounts of nitric oxide (NO) in response to oxidative stress that not only increases RBC hardiness and deformability but interacts with the vascular endothelium to maintain active arterial dilation which, as we know, is a reliable proxy for blood energy content [269-280].

It is apparent that the first phase of the PC response, aimed at augmenting blood energy levels, is responsible for orchestrating subsequent cellular events. Energy currents, carried in the interstitial fluid space, enter cells via ion channels, enhance mitochondrial function and intracellular energy metabolism and thereby induce a plethora of genes that actively counteract oxidative stress. The second window of protection is clearly driven by gene induction: critical response pathways include nuclear factor erythroid 2-related factor 2 (Nrf2) and the heme oxygenase-1 enzyme (HO-1) system [281].

The powerful antioxidant and anti-inflammatory effects unleashed throughout the body by low dose ozone administration are mediated through activation of the transcription factor Nrf2. Nrf2, master regulator of redox balance, binds to over 200 different genes, known as the antioxidant response element (ARE), and effects transcription of cytoprotective substances like heat shock proteins, antioxidant and detoxification molecules, enzymes involved in synthesis of glutathione, a host of growth factors like vascular endothelial growth factor (VEGF), erythropoietin (EPO) and more. The Nrf2-driven battery of gene products also effects breakdown and/or refolding of misfolded proteins, DNA repair, mitochondrial rebuilding, autophagy regulation, as well as intracellular metabolism. Impaired Nrf2 function is a hallmark of many chronic disease conditions [282-288].

Ozone is cheap and easily generated by passing oxygen across a voltage gradient simulating a lightning strike. Various routes of administration are employed: autohemotherapy, which involves removing a small aliquot of venous blood, exposing it to ozone, and reinjecting it into the vein; rectal or vaginal insufflation of ozone gas; direct intravenous (IV) injection of ozone gas; IV infusion of ozonated saline solution; topical administration of ozonated oil preparations.

All of the various approaches are safe and side effects virtually non-existent. Water-based approaches are hampered by instability and volatility and must be administered straight away. We developed an infusion method for a drinkable preparation in which stability is preserved for at least 4 weeks. We have used the oral

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route exclusively since June, 2021 and have observed no significant differences compared with the intravenous infusion route.

We have treated several hundred COVID-19 patients with ozone in the outpatient setting. Many individuals had multiple comorbidities and presented with moderate-to-advanced disease including respiratory difficulty and low oxygen saturations; some had radiologic evidence of COVID-19 pneumonia. Ozone combined with other adjuvant therapies relieved symptoms, shortened the course of disease, and appeared to decrease morbidity especially in those who sought treatment early in the course of the infection.

Enhancing Dielectric Capacitance

The second strategy for augmenting the body's energy economy involves boosting cellular metabolism and mitigating inflammation. The archetypal substances for this are the aminoquinolines, i.e., chloroquine (CQ) and hydroxychloroquine (HCQ), derivatives of quinine, which have been used for centuries to interrupt the inflammatory cycle [289-291].

Anthony Fauci claimed there was no evidence to support HCQ in COVID-19 infection but he was flat out wrong. Fauci overlooked a nearly 400-year history of quinine-related compounds and their striking capacity to modulate all kinds of inflammation. Well before the COVID-19 pandemic multiple studies showed efficacy of CQ against the original SARS coronavirus infections and later against Middle East Respiratory Syndrome virus which is also a coronavirus [292-294]. Based on the mediocre clinical track record of remdesivir our COVID-19 czar would have been well-served sticking with this time-honored strategy.

The aminoquinoline issue has split the science community and, ultimately, begs the question, 'what is science?' [295]. Multiple meta-analyses from across the globe failed to find beneficial effects from these substances in COVID-19-infected individuals [296-301]. End of story? No. The majority of studies were in hospitalized subjects with or without multiple co-morbidities, who may or may not have been on mechanical ventilation, who may or may not have received other treatments and in whom dosages varied across the spectrum. But medicine is a science of individuals and not an amorphous mass of statistical data. Trends and outcomes do not always align.

We failed to identify a single pooled-data study that methodically examined the interactions between timely HCQ administration, dosing considerations and pre-existing co-morbidities on clinical outcomes. COVID-19 infection is an energy deficient state the severity of which is directly proportional to the energy debt. It goes without saying that outcomes of all individuals in more advanced states of disease will be worse regardless of the therapy employed. Studies point to lack of beneficial outcomes with all conventional therapies including antiviral agents [302].

More troubling is the bias of the academic science community [303]. According to one meta-analysis, there was stark variation in CQ/HCQ outcome studies between the US and the rest of the world. Of 68 studies originating in the US, 39 (57.4%) were unfavorable while only 7 (10.3%) reported favorable results. Of 199 studies originating elsewhere, 66 (33.2%) were unfavorable, 69 (34.7%) favorable and 64 (32.2%) indeterminate. Studies with at least one US main author were 20.4% ($P < 0.05$) more likely to report unfavorable results than non-US studies. Study authors concluded that such bias contributed to dissemination of unfavorable results, i.e., misinformation, regarding CQ/HCQ. Science, it seems, is not immune to the dramatic cultural polarization which has taken place during the pandemic.

Then there is the notorious meta-analysis of 96,000 hospitalized COVID-19 patients from 671 hospitals across the globe by Harvard cardiologist Mandeep Mehra published in *Lancet* in May, 2020 which found no benefits from CQ/HQ therapy and was supposedly associated with increased risk of cardiac arrhythmias and death. The article was cited by Fauci to support his claim that these substances were ineffective. Mehra's study was later found to be fraudulent and subsequently retracted by *Lancet* [304].

Multiple studies conducted during the pandemic both in COVID-19 hospitalized subjects or outpatients found benefits with HCQ either alone or in combination with other agents such as zinc and azithromycin [305-313]. Hospital-based studies found shorter length of stay, decreased likelihood of ICU transfer and death, as well as shortened period of viral shedding. One large Italian study found a 30% lower death rate in hospitalized patients

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given HCQ [314]. In a study of 2541 hospitalized patients, HCQ alone had a 13.5% death rate, HCQ + azithromycin 20%, azithromycin alone 22% versus 26.4% with standard treatments [315]. Another hospital-based study of 8075 subjects found death rates of 17.7% in HCQ-treated subjects versus 27.1% in the no-HCQ limb [316]. These are impressive results by any measure.

In a 2020 American outpatient study, 144 COVID-19-infected subjects treated with HCQ + zinc + azithromycin were compared with 377 untreated controls. Of the treated subjects only 4 (2.8%) required hospitalization versus 58 (15.4%) of the untreated group. There was one death in the treated group (0.7%) versus 13 (3.4%) of untreated subjects [317]. In an intriguing 2020 Brazilian epidemiologic study data was tracked weekly for six months on COVID-19 caseloads, hospitalizations, deaths, social isolation practices and sales of CQ/HCQ in the state of Santa Catarina. Sales of CQ/HCQ were significant predictors of all outcomes while social isolation indices had no bearing. Some have suggested that lower caseloads and mortality rates in Africa are related to widespread HCQ use for malarial prophylaxis. These correlations suggest that CQ/HCQ affect transmissibility of SARS-CoV-2 which is more than can be said for the mRNA vaccines [318].

In the early 17th century Jesuit missionaries, while traveling in the Andean forest region, observed that natives used a substance to control shivering during cold temperatures. The bark of the 'fever tree,' when dried and made into a powder, was also highly effective in ameliorating fevers. In following centuries 'cinchona' became widely used throughout Europe as the first effective anti-malarial agent, reducing fevers, as a tonic for gastrointestinal ailments, soothing muscle cramps, and calming nerves.

In the 1890s English physician Joseph Payne reported the benefits of quinine in patients with systemic lupus erythematosus (SLE). During World War II incidental beneficial effects with skin rashes and joint pain were observed in soldiers placed on anti-malarial prophylaxis. Later studies confirmed the efficacy of these compounds in SLE and rheumatoid arthritis. General effects include anti-inflammatory, anti-infective, immunomodulatory, anti-thrombotic as well as metabolic. But how such widespread effects are mediated remains unclear.

CQ/HCQ are beneficial in autoimmune conditions like Sjögren's syndrome, the iron-related disorder porphyria cutanea tarda, and the curious entity known as polymorphic light eruptions. In addition, they are effective in a variety of bacterial, viral and parasitic infections, and have shown efficacy as adjuvants in various cancers. The broad range of applications suggests effects are mediated not solely by turning on or off specific molecular pathways but rather as a non-specific amplifier of cellular metabolism and cellular digestive functions. Downstream effects are seen in the blood, arterial wall, interstitial fluid space, and the intracellular compartment [319-321].

In 1984 blood glucose-lowering effects of CQ/HCQ were discovered in type II diabetics. These agents improve insulin resistance implicating a shift in glucose metabolism at the cellular level [322-324]. They improve lipid profiles by decreasing serum triglyceride and cholesterol levels which are also likely related to metabolic alterations at the cellular level [325-327]. Treatment with HCQ is protective against thrombosis in SLE patients with anti-phospholipid autoantibodies [328-332]. But how to understand such a broad range of effects?

The term 'dielectric' was coined in the mid-19th century by physicists to designate a set of properties observed in relation to externally applied electrical currents. While substances like copper or silver conduct electricity, and insulators like glass, oils or rubber repel it, dielectric substances, instead, undergo internal polarization, i.e., separation of positive and negative charges, which amounts to creation of an internal field, i.e., the dielectric field. Cells are tiny dielectric capacitors.

When a strong electrical potential is applied to ferrous objects like iron their nuclei resonate, realign internally, and expel intra-atomic magnetism giving rise to an external magnetic field. The same dynamics are at play in the cardiovascular system when a magnetic field is generated in the blood to produce the outward diastolic motion of the heart and arterial walls.

With dielectricity, on the other hand, force lines are directed inwardly and radially as seen during contraction of the ventricles. The dielectric, the primary energy field, lies at the aether boundary, i.e., the inertial plane, and, when activated, produces torque thereby inducing energy flow. In reality, magnetism arises from the dielectric and the two always coexist in a single conjoined and inseparable field.

Dielectric materials possess high polarizability, expressed numerically as the dielectric constant, an indicator of

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energy storing ability or capacitance. But capacitance doesn't simply indicate passive energy storage. In capacitance the dielectric field draws conductors into tighter spatial apposition thus increasing the counter spatial torque and activating aether flux. In living bodies the dielectric force originates in water, which has one of the highest dielectric constants, i.e., polarizability, of all substances indicating its ability to generate energy through the conjoined magneto-dielectric field and aether.

The intracellular dielectric field can be conceived as possessing two opposing poles, the cathode, the source of magnetic-dominant aerobic energy which maintains the surrounding fluid space in the alkaline pH range, and the anode through which energy is drawn out and around which the cell water is energy depleted and acidic. Inflammation, the result of mitochondrial dysfunction and deficient energy flux, employs less efficient acid-producing pathways and, as such, represents a shift toward the anodic pole and loss of intracellular capacitance. This is where the aminoquinolines come into play.

CQ/HCQ actively accumulate in cells and many researchers believe their effects are mediated intracellularly. In both immune and non-immune cells, the primary effect of CQ/HCQ is to shift cytoplasmic pH into the alkaline range, in other words, from the anode towards the cathodic pole of the dielectric field. Alkalinization causes mitochondrial metabolism to shift from catabolic to anabolic processes thereby inducing protein synthesis, repair of membranes, and stabilization of DNA [333]. Such effects impair entry of viral pathogens into the cell as well as inhibiting their replication. It goes without saying that these effects are energy-dependent.

Researchers claim CQ/HCQ induce alkalinization by their accumulation in cells but this doesn't make sense. These compounds are weak bases but the magnitude of the effect is much greater than can be explained by their physical presence alone. They more likely act on the basis of field effects by enhancing dielectric capacitance in cells thereby inducing aether flux and flow of negatively-charged ion currents.

CQ/HCQ decrease pro-inflammatory cytokine release by macrophages while at the same time promoting phagocytosis; they block pro-inflammatory T-cell proliferation and shift immune cell balance toward anti-inflammatory subsets. This is directly related to alterations in WBC metabolism toward a more efficient energy-generating mode [334-343].

During the pandemic we employed HCQ in two ways. Due to its accumulation in cells HCQ has an extremely long half-life, by most estimations in the 3-4 week range. In older individuals and in those with comorbidities we used 200-400 mg per week for prophylaxis, about the same as for malaria prevention. During active infection we increased the amount to 200-400 mg/day over 5-7 days. In both cases, evidence suggests that HCQ boosts intracellular energy flow thereby enhancing resistance and producing less severe infections while still allowing for development of natural immunity. We had no side effects with such low doses. At a cost of about $0.25 per tablet it is mind-boggling why this simple and effective strategy was not employed in all high-risk individuals during the pandemic.

Catching the Light

Having recognized all inflammatory disorders including COVID-19 as primary energy deficiency states, we examine a third strategy to boost energy flow and enhance resistance: modulation of radiant light energy and vitamin D. Radiant light, the third primary energy form, functions as an intermediary between the blood-borne magnetic field generated by the heart and the cell-based dielectric field. Radiant light interacts with water in the subdermal fluid space to generate current flows that, through the vitamin D-related system of enzymes and membrane receptors, amplify cell function. A flood of evidence has surfaced during the pandemic affirming the import of this alternate energy pathway.

Multiple studies link vitamin D deficiency (VDD) with severity of COVID-19 infection, hospitalization, length of stay, ICU admission, pulmonary complications, need for mechanical ventilation, and death [344-358]. In one study 82% of COVID-19 cases had VDD compared with 47% of control subjects. Severely symptomatic COVID-19 patients have lower vitamin D levels than mildly-symptomatic or non-infected subjects. In another study, death rate in VDD patients was 46.5%, 29.5% in vitamin D insufficient subjects, and only 5.5% in those with normal vitamin D levels. Yet another study found that patients with vitamin D levels less than 30 ng/ml had a 25% mortality while those greater than 30 ng/ml were only 9%. There is solid science behind such clinical trends.

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Vitamin D is a surrogate for available light energy. Both autoimmune and infectious disorders tend to cluster in seasonal and geographic patterns related to the quantity and quality of ambient sunlight reaching the earth's surface. At the 45th parallels, for example, available light energy gradually decreases from about 12 hours per day near the autumnal and vernal equinoxes to about 9 hours at the winter solstice. During this period the quality of light is markedly attenuated due to the incident angle of solar rays.

The energy deficiency syndromes tend to express themselves more commonly at high latitudes or in winter and spring months after sustained periods of light deprivation. Chronological patterns have been reported in various autoimmune disorders, MS being the most well documented, as well as infectious diseases like tuberculosis [359-363]. Since the early 20th century there have been five pandemics including the recent COVID-19 outbreak which have all shown similar temporal predispositions [364]. Neoplastic conditions like breast cancer also show seasonal behaviors that appear to influence survival patterns [365-367].

Studies affirm that blood vitamin D levels correlate with ambient light exposure and serve as a reliable proxy by which to gauge its effects. Many reports chronicle the relationship between VDD and the various autoimmune diseases [368-401]. At the north 45th parallel there is a null period between November and February during which sunlight is insufficient to trigger vitamin D synthesis. Such fluctuations roughly correlate with seasonal variations in disease incidence and activity [402-407].

A host of factors including ethnicity and cultural factors like mode of dress play into the picture. Dark-skinned peoples require up 6-fold greater sunlight exposure to get the same blood levels of vitamin D as light-skinned people. In the northern US dark-skinned people are predisposed year-round to VDD but especially in winter [408-412]. This must certainly play a permissive role in the worse outcomes seen in such individuals during the COVID-19 pandemic. Anything that affects transmission of radiant energy affects vitamin D synthesis, the so-called sunshine hormone.

Through photosynthesis-like mechanisms, light rays induce formation of cholecalciferol which is subsequently transformed to 25(OH)D_3 and, finally, to the highly-active 1,25(OH)$_2D_3$ form. The latter energy-requiring step is affected by the cytochrome p450 enzyme system. The final product is said to be about 500-fold more biologically active than its precursor suggesting light-induced energy flow through vitamin D pathways. Vitamin D receptors at the cell membrane mediate the effects of highly-active vitamin D. Such receptors are present throughout the body including immune cells. Activated vitamin D influences at least 500 different gene activities through receptor-mediated epigenetic mechanisms [413].

Given such biological potency one would expect vitamin D to be a panacea for COVID-19-related inflammation but clinical studies are inconsistent. A small number of studies showed no relation between vitamin D levels and outcomes in COVID-19 infection [414-418]. Others found no benefit with administration of vitamin D in hospitalized COVID-19 patients [419]. Why are effects so mixed? It would appear that the rate-limiting step lies not only in the synthesis of the vitamin D precursor but in its energy-dependent conversion into the biologically potent form. Studies in individuals with autoimmune disease suggest that light plays the active role in conferral of benefits.

Norwegian subjects with psoriasis had significant clinical improvement in skin lesions after 16 days of sub-tropical sun exposure which was preceded by changes in immune function. Pro-inflammatory cytokine and T-cell levels in both skin and blood decreased and were replaced by resistant anti-inflammatory T-cell subsets [420, 421]. A Scottish study using narrowband ultraviolet phototherapy in subjects with various immune-mediated skin disorders found similar shifts in immune function along with increased vitamin D levels after 4 weeks of treatment [422]. Such effects occur on the basis of energy transfer between light and water which, in turn, provides energy equivalents for proteins and various enzyme systems to carry out their designated functions.

Studies indicate that water is sensitive to the effects of light. Researcher Gerald Pollack discovered that radiant light energy induces water to undergo spontaneous structural reorganization into two different forms, one, which he calls exclusion zone (EZ) water based upon its tendency to exclude particles, and the other, called bulk water, which forms adjacent to EZ water [423]. EZ water contains a surplus of negative charge and has higher pH and density. Bulk water, containing an excess of protons, is acidic with lower pH and specific gravity. The two different physical states of water, like the space between the cathode and anode, create a charge separation with

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resultant current flow between them. Water is unique among substances in possessing both high dielectric capacitance as well as an ability to conduct alternating electrical currents.

By the same token it is recognized that all physiological functions in the body are affected by conformational changes in proteins (which represent states of polarization and depolarization). All proteins are surrounded by a mantle of water, called the hydration layer, which corresponds to Pollack's EZ water and tends to form around hydrophilic surfaces. Technologies such as NMR spectroscopy and x-ray crystallography affirm the primary role played by tissue water in protein-related dynamics [424-428]. The hydration shell appears to be instrumental in determining not only 3D protein structure but the folding process itself. Changes in water state have been recognized to be at play in a host of protein misfolding disorders [429-433]. It seems likely that light-induced charge separation in the interstitial water compartment provides energy to transform vitamin D into its active form.

Light-induced effects are mediated by the ubiquitous superfamily of enzymes known as the cytochrome p450 system which, evidence shows, is also activated by externally applied light pulses [434-437]. Skin keratinocytes, which are capable of effecting the whole vitamin D synthesis sequence, contain α-1-hydroxylase, the enzyme that transforms vitamin D into its highly potent form [438-441]. It appears this enzyme system plays a key role in protecting the lungs from COVID-19-related complications as well.

The primary mode of entry of SARS-CoV-2 into the body is through the lungs. Multiple studies confirm VDD is associated with a higher incidence of upper airway infections secondary to rhinoviruses, respiratory syncytial virus as well as coronavirus. Epithelial cells throughout the lung and upper airway express high levels of α-1-hydroxylase and continuously generate highly-active $1,25(OH)_2D_3$ which, through vitamin D receptors, turns on genes that release antimicrobial substances like defensins and cathelicidins as well as enhancing phagocytic activity [442-445].

Studies found VDD was associated with elevated cytokine storm markers like TNF-α, IL-1, IL-6, IL-10, and IL-21 [446-450]. In one study the prevalence of VDD in severely ill COVID-19 patients was 97.8% compared with 32.9% in asymptomatic COVID-19-positive subjects. Mortality rate was higher (21%) in VDD subjects versus those with normal vitamin D levels (3.1%). Not surprisingly inflammatory markers such as IL-6 and TNF-α were elevated. Macrophages play a key role in the evolution of COVID-19 associated respiratory distress syndrome and possess vitamin D receptors [451-454]. Multiple articles thus recommend widescale vitamin D supplementation to modulate the inflammatory response in COVID-19 infections [455-458].

We employed high-dose vitamin D, light treatments and HCQ prophylactically and in early COVID-19 infections in dozens of individuals without encountering a single serious COVID-19 infection or hospitalization. Academic pundits would argue our numbers were far too small to draw substantive conclusions but treatment on the basis of established principles trumps random (and flawed) experimentation as in the case of the mRNA vaccines.

License to Kill

In previous sections we established the efficacy of three cheap and widely available therapeutic approaches—ozone PC, HCQ, and light/vitamin D—which, had they been implemented in either a preventive role or early in the course of COVID-19 infection, could have markedly improved outcomes and decreased morbidity and mortality. Evidence in support of their effectiveness had been in the medical literature years before the pandemic but was roundly ignored by the science community and policy makers at the public's expense.

We saw that when PC pulses are applied prior to a sub-lethal period of ischemia release of energy into the blood by RBCs results in about 75% reduction in tissue damage. While this degree of protection would not be expected in the elderly or in those with comorbidities it nonetheless remains quite substantial. There were significant reductions in disease severity and death rate in subjects treated with HCQ alone or in combination with other agents. Mortality rates in hospitalized patients with VDD were at least 2-3X higher than in subjects with normal vitamin D levels. Based on such considerations we conclude that the three modalities in combination, either before or shortly after symptom onset, could have reduced pandemic-related morbidity and mortality by *at least* 80%.

*Hyperlink to 1,366 references for COVID-19 vaccine associated complications:
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At the time of this writing, March 17, 2022, there had been 462,758,117 total COVID-19 cases globally, 6,056,725 deaths, with an overall mortality rate of 1.3% [459]. In the US there were 78,891,488 reported cases cumulatively with 960,194 deaths equating to a 1.2% mortality rate. It is generally accepted that 80-85% of confirmed cases are mild, 10-15% moderate, and about 5% severe. Clinical criteria are established for each of these categories [460-465]. For convenience we will use 85%, 10%, and 5% to approximate this distribution. It goes without saying that most deaths are associated with severe cases.

Based on such figures the global burden of severe cases during the pandemic so far approximates 23 million with about 7.9 million in the US. Had ozone PC, HCQ, and light/vitamin D treatment been implemented either preventively or at the time of symptom onset we estimate that about 4.8 million deaths globally and 768,000 in the US could have been prevented. By the same token, had these modalities been utilized broadly on a populational scale instead of the failed vaccines there would have been a generalized downgrading of severity across the spectrum resulting in fewer morbid complications and hospitalizations. Given the superiority of natural immunity compared with the vaccines, this would have been a far safer and quicker path to herd immunity than that afforded by vaccines.

To date during the pandemic around 879,000 individuals in the US have required hospitalization with average length of stay about 5-6 days; complicated cases involving ICU admission can extend for weeks [466]. According to one study, costs for uncomplicated admissions averaged about $50,000 while complicated admissions were in the $200-300,000 range [467]. Hospitalization costs in the US alone will run into the tens of billions of dollars if not more. Despite improvements in survival rates during the pandemic ICU mortality remains in the 20-30% range [468-472]. Much of this carnage could have been averted had COVID-19 been treated properly.

In what will likely go down as the greatest public health disaster in medical history, Fauci and policy-makers failed to establish home-based therapeutic protocols. Instead, COVID-19-positive individuals were sent home to manage for themselves while continuing to spread the virus among close contacts. Many eventually developed severe symptoms requiring hospitalization. Treating an early, mild condition has a markedly greater likelihood of success than the far-advanced, energy-depleted state. 'The strategy from the outset,' claims cardiologist Peter McCullough, 'should have been implementing protocols to stop hospitalizations through early treatment of Americans who tested positive for COVID but were still asymptomatic' [473].

Early in the pandemic McCullough reflected on the absurdity of doing nothing for a year or more while vaccines were still in the pipeline. Chinese physicians had already published an early treatment regimen in March, 2020 resulting in a dramatic decrease in caseload by May, 2020 [474, 475]. Searching the medical literature McCullough compiled the first US COVID-19 protocol which was published in July, 2020 in the *American Journal of Medicine* [476, 477]. The regimen was administered to over 800 people in the Dallas area with a resultant 85% decrease in hospitalization and mortality. 'We could have dramatically reduced COVID fatalities and hospitalizations,' McCullough argues, 'using early treatment protocols and repurposed drugs including ivermectin and hydroxychloroquine and many, many others.' He claims the COVID-19 pandemic in the US could have been ended as early as May, 2020 [478].

Pierre Kory, pulmonary medicine and critical care specialist St Luke's Medical Center, Milwaukee, and president of Front Line COVID-19 Critical Care Alliance agrees: 'the efficacy of some of these drugs as prophylaxis is almost miraculous'. Early intervention after exposure, he added, stops viral replication and prevents development of the cytokine storm and pulmonary complications. 'Dr. Fauci's suppression of early treatments,' Kory claimed, 'will go down in history as having caused the death of a half a million Americans in the ICU' [479]. Our estimates suggest the numbers will be quite a bit larger. Like McCullough, Kory argues that early treatment could have stopped the pandemic in the spring of 2020. Fauci's deliberate and premeditated policies represent the highest form of malpractice.

Ryan Cole, clinical pathologist and medical director of Cole Diagnostics, the largest independent medical lab in Idaho, also became a strong proponent of early intervention after observing numerous striking turnarounds in COVID-19-infected persons: 'Early treatment of COVID-19, plain and simple, saves lives'. He argues that if the medical community had been pro-active, the early multi-drug approach would have saved hundreds of thousands of lives in the US. 'Never in the history of medicine has early treatment . . . been so overtly neglected by the medical profession on such a massive scale'. In the case

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of COVID-19, 'not to treat is to do harm'. And Cole adds: 'The sacred doctor-patient relationship needs to be wrenched away from Anthony Fauci and the government/pharmaceutical industrial complex . . . Doctors need to return to their oaths'. The pandemic has laid bare the irremediable flaws of a healthcare system that 'has lost its direction and soul' [480].

Medical internist Deborah Viglione is more blunt: 'The pandemic was not managed by real science but by political science'. Real science, she argues, showed that masks and lockdowns were useless. These measures were implemented, says Viglione, to manipulate the population through fear to submit to the vaccine agenda. After diagnosis patients were sent home to quarantine and return only if they couldn't breathe, 'but what kind of strategy is that?' The focus, she continued, should have been on early treatment to reduce viral loads, boost the immune response, and reduce oxidative stress.

Physicians who spoke out against Fauci's catastrophic policy, says Viglione, were labelled as spreading misinformation and threatened with revocation of licensure or board certification. Meanwhile, she continued, authorities attempted to suppress life-saving medications by pressuring pharmacists not to fill prescriptions or insurance companies not to reimburse claims. This assertion has been echoed by numerous frontline physicians in the battle against COVID-19.

Viglione has treated over 500 patients from her office using ozone PC and a variety of other modalities including HCQ, quercetin, ivermectin, vitamins B, C, D, and zinc depending on severity of symptoms or existing comorbidities. 'The vast majority of our patients did extremely well with reduction of their symptoms and duration of illness. We had a high severity of illness. Most of our patients presented a week or more into their illness and were already in cytokine storm. They simply refused to go into the hospital. In spite of this we still had an extremely low death rate'. In fact, Viglione added, 'nurses in the local EDs were telling patients to come to us instead of staying there'. This is quite a different narrative from that which health care systems are trumpeting.

In his 2021 book *The Real Anthony Fauci*, Robert F. Kennedy Jr traces the web of distortion to Fauci, whose obsession with the mRNA vaccines and remdesivir led him to ignore and suppress other effective treatments. He engaged in 'blatant and relentless manipulation of data to serve the vaccine agenda'. Fauci's policies, says Kennedy, 'were so grotesquely ill-conceived, so unfounded in science, so tethered to financial interests, that they caused hundreds of thousands of wholly unnecessary deaths'. Instead of adhering to science-based data Fauci relied on arbitrary dictates from the CDC and WHO while urging the public to 'trust the experts' even though the experts were often wrong. Throughout the pandemic there was a 'shockingly low quality of virtually all data pertinent to COVID-19' [481].

Instead of supporting the work of McCullough, Kory and others, federal agencies and public media began actively censoring information on effective remedies. 'Dr. Fauci refused to promote any of these interventions,' Kory claimed. 'It's not just that he made no effort to find effective off-the-shelf cures—he aggressively suppressed them'. McCullough agrees: 'It shocks the conscience that there is still no official protocol'. Anyone who tries to publish a protocol, he claims, 'will find themselves airtight blocked by the journals that are all under Fauci's control'. McCullough was fired from the staff of Baylor Medical Center. Cole's laboratory was dropped by one Idaho's largest healthcare networks [482]. Numerous other physicians paid a steep price for breaking line against Fauci's ruinous policy.

Not Ready for Prime Time

The human cost in terms of morbidity and mortality also includes adverse effects related to the mRNA vaccines. To date there have been approximately 1,183,493 COVID-19 vaccine-related adverse event reports, including 25,641 deaths, reported to VAERS since the beginning of the pandemic. As we discussed in part II of the paper VAERS data is difficult to quantify the true magnitude due to under-reporting however when we compared available data to the number of administered vaccines, we found an adverse event rate about 40-fold higher compared with 2010 influenza vaccine event rates.

To date about 10,783,650,787 COVID-19 vaccine doses had been administered globally and 537,567,013 vaccine doses in the US with US representing 4.98% of total vaccines administered or a 20:1 ratio [483]. If we apply this ratio to the VAERS data we estimate the number of adverse events globally to be 23.67 million with about 512,820 deaths over about a 15-month period. The total number of influenza-related vaccine deaths reported in VAERS over the past 30 years was only 9,357.

*Hyperlink to 1,366 references for COVID-19 vaccine associated complications:
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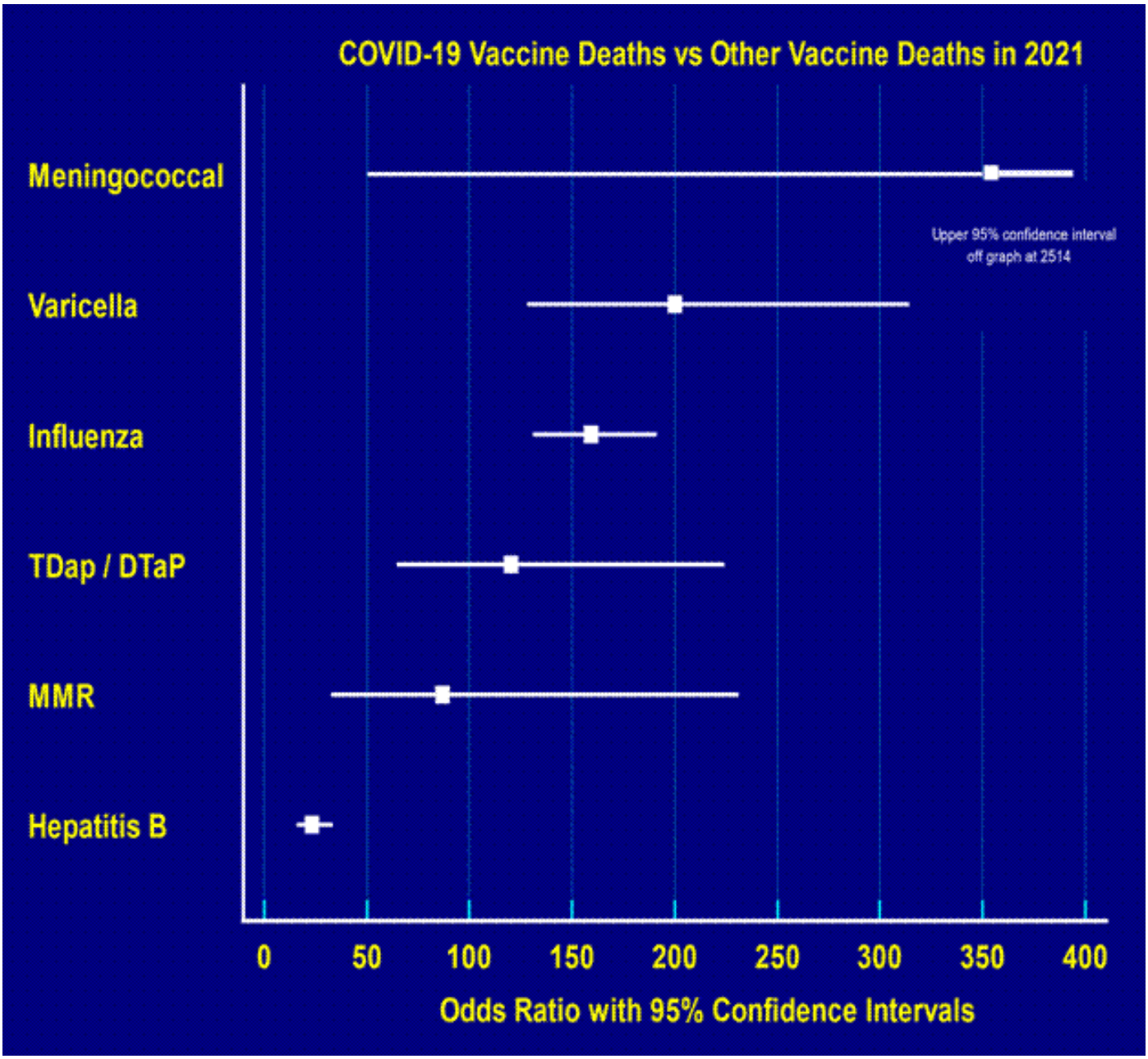
In part II of this series, we showed that when 2021 VAERS COVID-19 deaths were compared to that of 2010 influenza vaccines, there was a 40-fold higher risk with the mRNA vaccines. When odds ratios were calculated from COVID-19 vaccine deaths relative to other vaccine-related deaths in 2021 we obtained even more striking inequalities: 354-fold higher than meningococcal vaccine; 200-fold higher than varicella vaccine; 157-fold higher than influenza vaccines; 120-fold greater than that of the Tdap/DTaP vaccines; 86-fold higher than the MMR vaccines and; 23-fold greater than the hepatitis B vaccine as depicted in **Figure 1.** Such disparities are mind-boggling.

Figure 1. Odds ratios for COVID-19 vaccine deaths compared to that of six other vaccines and the year 2021. Odds ratios and 95% confidence intervals are depicted. All odds ratios are statistically significant (P<0.0001).



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We recently compared peer-reviewed medical journal publication of adverse events (AE's) for COVID-19 vaccines since rollout (only 16 months) to that of six other vaccines ranging from 436 – 784 months **(Appendix 1).**

Appendix 1. All 1,366 references for COVID-19 vaccine associated complications are listed by subject matter. All 1,366 references are hyperlinked here and are all published in peer-reviewed medical journals from the onset of the COVID-19 vaccinations on December 15, 2020 to March 15, 2022 (16 months) [495].

Appendix 1. Subject-wise segregation of 1,366 references

COVID-19 Vaccine Published Complications Subject of Article(s)	Number of Publication(s)	Reference Numbers in the Hyperlink
Anaphylaxis	47	1 - 47
Antiphospholipid Antibodies	3	48 - 50
Arterial & Venous Thromboembolism	160	51 - 210
Arthritis	2	211 - 212
Auto-Immune Disorders	21	213 - 233
Autopsy Findings	11	234 - 244
Blood Disorders	10	245 - 254
Cancer	7	255 - 261
Cardiac Disease (Myocarditis / Pericarditis)	336	262 - 597
Cardiac Disease (other)	15	598 - 612
Dementia / Alzheimer's / Delirium	2	613 - 614
Encephalopathy & Neurological Injury	46	615 - 660
Eye Diseases	11	661 - 671
Facial Nerve Palsy	28	672 – 699
Gastroparesis	1	700
Guillain Barre Syndrome	51	701 – 751
Hearing Loss / Tinnitus	13	752 – 764
Hemolytic Uremic Syndrome	1	765
Hemorrhage	38	766 – 803
Hepatitis	19	804 – 822
Immune and DNA Impacts	7	823 - 829
Kidney / Urinary Disorders	23	830 - 852
Lung Disease	3	853 - 855
Lymphadenopathy	60	856 - 915
Multiple Sclerosis	1	916
Muscle Disorders	5	917 - 921
Prion Disease	1	922
Radiation Recall Syndrome	5	923 - 927
Rhabdomyolysis	12	928 - 939
Seizure Disorder	6	940 - 945
Shoulder / Musculoskeletal / Bursitis	7	946 - 952
Skin Reactions	41	953 - 993
Thyroid Disease	33	994 - 1026
Vaccine-Induced Thrombotic Thrombocytopenia	209	1027 - 1235
Varicella Zoster (Shingles) / Herpes	27	1236 - 1262
Vasculitis	48	1263 - 1310
Miscellaneous	56	1311 – 1366
TOTAL	1366	1 - 1366

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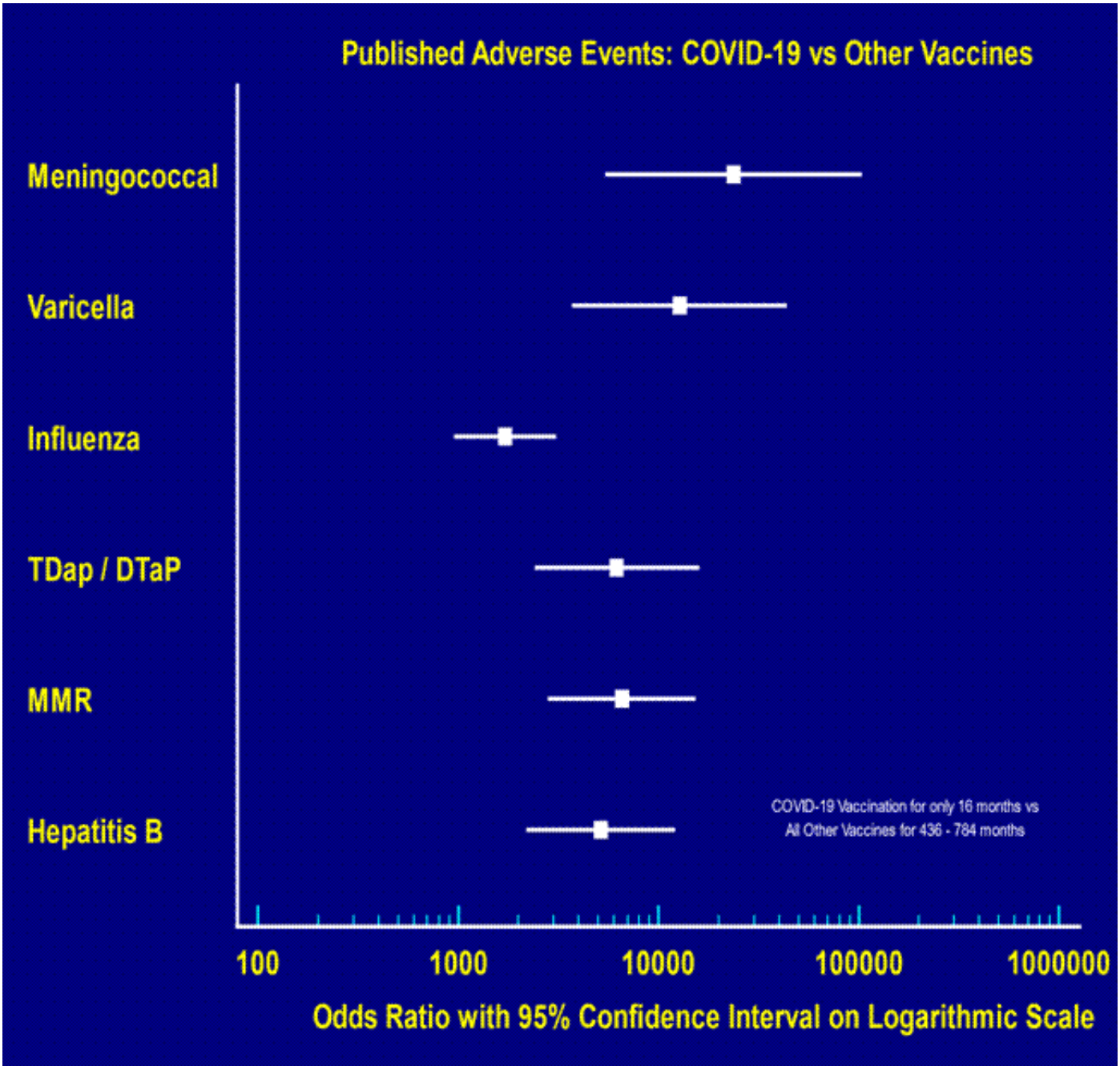
When odds ratios for published AE's were calculated (COVID-19 vaccine vs six other vaccines) there were again stunning inequalities. COVID-19 vaccinations had a 23,682-fold greater number of peer-reviewed publications of AE's than that of meningococcal vaccine; 12,721-fold higher than varicella vaccine; 1,712-fold higher than influenza vaccine; 6,190-fold greater than that of the Tdap/DTaP vaccines; 6,559-fold greater than MMR vaccines; and, 5,154-fold greater than Hepatitis B vaccine as depicted (Figure 2). Such irregularities are troubling given that COVID-19 vaccine AE's are less likely to be reported due the healthcare system and social media influences.

Figure 2. Odds ratios for COVID-19 vaccine published adverse events (AE's) compared to that of other vaccines. The duration of time was only 16 months for the COVID-19 vaccine while that of the other vaccines ranged from 436 to 784 months. The duration of time was not controlled in this graph. All odds ratios are statistically significant (P<0.0001).



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Add to these irregularities that swirled around the clinical trials and rollout of the mRNA vaccines and one's skepticism of this science-based initiative is further piqued. In October 2020 the FDA urged Pfizer and Moderna to use a clinical trial design that would preserve the integrity of data collection. To this end Fauci endorsed a blinded crossover study to enable ongoing assessment of efficacy and safety. The companies argued the crossover design was 'onerous' and overly complicated and that it would be unethical to withhold the vaccines from study participants. Both companies granted individuals access to their study data [484, 485].

Why did the FDA permit this breach of protocol? Diana Zuckerman, president of the National Center for Health Research, argues that the FDA could have demanded that the companies adhere to the guidelines to receive approval. Failure to implement the recommended study design resulted in loss of valuable data. She was also concerned about inadequate numbers of elderly subjects in the trial which, she claimed, makes it impossible to determine how effective the vaccine is for frail, elderly subjects.

Consumer representative Sheldon Toubman, lawyer and member of the FDA advisory panel, cited a paucity of evidence as to whether the vaccine is effective in preventing severe COVID-19 infections. And based on other vaccine trials, he raised concerns as to whether the six-week follow-up period was sufficient to reliably assess the safety of these novel and untested preparations. Such flaws in the planning and design of the vaccine trials, beyond constituting a beach of established experimental protocol, raise ethical concerns.

If the planning stage of the vaccine trials was shaky then the clinical phase should have raised even more eyebrows. As we describe in part II, a shocking whistleblower exposé in *BMJ* in November, 2021 alleged improprieties involving Pfizer vaccine trials including not only unblinding of subjects but falsification of data, using inadequately trained personnel, and unacceptable delays in follow-up of adverse event reports [486].

Data obtained through a Freedom of Information Act (FOIA) indicate AE's far exceeded original estimates. The Pfizer data of patients vaccinated from December 15, 2020 to February 28, 2021 had 1,223 deaths noted. A total of 274 pregnant women received the vaccine and 75 (27.4%) suffered 'serious' AE's while another 49 (17.9%) had "non-serious" AE's (page 12 on Pfizer document) [487, 488]. See these published court-ordered documents entitled '5.3.6 post marketing experience' on Public Health and Medical Professionals for Transparency website, phmpt.org. In late April 2021 this data was sent to both the FDA and CDC which, nonetheless, continued to issue glowing safety reports. This leads us to question the propriety, if not legality, of conferring Emergency Use Authorization (EUA) to the vaccine manufacturers.

Under federal law new medicines and vaccines do not qualify for EUA if there are existing FDA-approved substances that are also effective against the disease: 'there must be no adequate, approved and available alternatives to the candidate product for diagnosing, preventing, or treating the disease or condition . . .' Before the pandemic, as we have seen, there was abundant evidence in the research literature supporting the efficacy of modalities like ozone PC, HCQ, and vitamin D and others for the treatment of inflammatory conditions. Add to this the self-serving agenda of the vaccine manufacturers in both setting up the trials and carrying them out and one begins to suspect a trail of collusion leading back to the very powers that issued the EUA. This brings to mind the age-old quandary *quis custodiet ipsos custodes*: who guards the guards?

And beyond granting EUA under potentially fraudulent circumstances one is confronted by lapses in oversight. By the time the Delta variant surfaced in the summer of 2021 and spread across the globe there was ample data to suggest primary vaccine failure. We cited multiple studies showing a high percentage of Delta-variant infections in previously vaccinated persons. By late July the CDC was aware of these trends and formulated a policy to downplay the magnitude of breakthrough infections and focus on the role of the unvaccinated population in the Delta surge [489].

By mid-August the Whitehouse Coronavirus Task Force had reams of data from across the US and the world showing no relationship between emergent Delta spread patterns and vaccination rates [490]. And yet President Biden continued for the rest of 2021 and into 2022 to promote vaccination and booster jabs while at the same time publicly wearing a mask as if upholding an established, time-honored principle. Even presidents bow down to science. The gap between spin and reality has never been more obvious.

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Why were the COVID-19 vaccines approved and recommended by the FDA and CDC when there were so many unanswered questions regarding safety and propriety? Why did the American Board of Medical Specialties (ABMS), the American Board of Obstetrics & Gynecology (ABOG) and the Federation of State Medical Boards (FSMB) threaten all physicians in the United States with loss of licensures and board certifications if "COVID-19 misinformation" was spread? [488]. ABOG, ABMS and FSMB documents show that "COVID-19 misinformation" is used as a euphemism purposed to gaslight and eliminate vaccine hesitancy [488]. As of this publication ABOG is still pushing the vaccine in pregnant women and women of reproductive age despite the grave concerns expressed.

Why wasn't the mRNA vaccine experiment terminated in the summer of 2021? And, as if in defiance of this lapse, the very same trends repeated themselves in the autumn with the Omicron variant. Why wasn't the vaccine program stopped in late fall? Certainly, the evidence was compelling. And by January 1, 2022, daily COVID-19 caseloads across the globe were over double as compared to January 1, 2021 as the vaccines were being rolled out. And yet governments and healthcare systems continued to hawk the mRNA vaccines while blaming the unvaccinated for soaring hospitalization costs [491].

Why wasn't there more public and scientific debate about spiraling caseloads in late 2021 and early 2022 or the escalating number of adverse event reports? Why were the 1,366 peer-reviewed publications of AE's over 16 months since the vaccine rollout summarily ignored? [495]. And why were early treatment protocols never established? Instead the number of infections along with their attendant morbidity and mortality continued to surge throughout the winter of 2021-22. If one seeks to make a case for failure of oversight look no further.

As the saying goes, 'denial ain't just a river in Egypt'. No matter how the academic science community attempts to twist the narrative to its own ends, the fact remains that herd immunity can and will be reached *only* through the unvaccinated and breakthrough infections in the vaccinated. The mRna vaccines weren't the free ticket into the Promised Land as scientists claimed. And in their wake, they left a wide swath of devastation.

Science in Crisis

The problems we cite concerning suppression of early treatment protocols and corruption of vaccine trials is not peculiar to the US. Multiple instances of such aberrations came to light in the UK: internal documents withheld from the public, officials ordered not to discuss certain matters, publication of fraudulent research; other research smothered or censored. In a November 2020 editorial in *BMJ* entitled 'COVID-19: politicization, 'corruption,' and suppression of science,' executive editor and physician Kamran Abbasi argues that science was manipulated for political and financial gain [492].

During the COVID-19 pandemic era the concept of evidence-based medicine has become an anachronism and the more pertinent concern now is 'who's evidence?' and 'to what end?' In the US, Operation Warp Speed was used to justify hasty shoddily-designed vaccine trials while suppressing widely available alternative medicines. At the same time the culture was inundated by a flood of misinformation and disinformation by those who held the reins of power. The pandemic unleashed scales of opportunism by politicians, industry, academic scientists and healthcare systems while, at the same time, revealing just how easily the common perception of reality can be purposefully distorted during times of crisis.

'Politicization of science,' writes Abbasi, 'was enthusiastically deployed by some of history's worst autocrats and dictators, and it is now regrettably commonplace in democracies. The medical-political complex tends towards suppression of science to aggrandize and enrich those in power. And, as the powerful become more successful, richer, and further intoxicated with power, the inconvenient truths of science are suppressed. When good science is suppressed, people die'. This precise tactic was employed by the CDC, FDA, ABMS, ABOG and FSMB. This echoes the timeless axiom 'power corrupts; absolute power corrupts absolutely.'

In a March 2022 Opinion piece published in the *BMJ,* Jon Jureidini and Leemon McHenry, authors of *The Illusion of Evidence-Based Medicine: Exposing the Crisis of Credibility in Clinical Research* (2020), up the ante and argue that evidence based medicine has been thoroughly corrupted by corporate interests, lack of oversight, and rife with special interest among influential academic scientists [493].

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Evidence based medicine seeks to provide a solid fact-based foundation for the practice of medicine. Its effectiveness depends on reliable data gathered from well-conducted clinical trials but how can this occur when the trials are controlled by the industry itself? *Cui bono?* The whistleblower article and data obtained through the FOIA request reveal the degree to which industry sponsored trials were manipulated and how vulnerable the evidence-based process truly is. 'Until this problem is corrected,' write Jureidini and McHenry, 'evidence-based medicine will remain an illusion.'

The scientific ideal of impartiality and strict adherence to evidence, i.e., objectivity, allows perpetuation of a legitimate fact-based science; absent this and science becomes little more than a polyglot jumble of unsubstantiated claims. Never in recent centuries has the gap between science and religion been so razor-thin. The scientific ideal is threatened by corporations in which financial interests prevail over objectivity. 'Patients die', Jureidini and McHenry argue, 'because of the adverse impact of commercial interests on the research agenda, universities, and regulators'.

Corporations are not accountable to the public but to shareholders. Fierce corporate tribalism, brand loyalty and public perception inevitably triumph over scientific truth. Universities have become pawns of corporations which exercise undue influence over research agendas, journal content and medical education. In recent decades the corporate culture has insinuated itself throughout all layers of the university.

Academic deans have been replaced by profit-driven 'managers' who cultivate affiliations with the industrial sector to enhance revenues. Based on their affiliations with prestigious universities, published academics are actively courted by corporate 'sponsors' to influence practice patterns and enhance brand identity. Such 'key opinion leaders' become paid members of corporate advisory boards and 'product champions' at medical conferences and continuing medical education programs. And, in the process, they compromise their impartial point-of-view while continuing to reap all the benefits as university faculty members.

This dynamic, wholly pervasive during the pandemic, created an uneven playing field: corporations and universities enacted their one-sided vaccine agenda while critics faced consequences ranging from rejection of intellectual work by journals, ostracization in their professional community, legal threats, and loss of licensure, accreditation or ability to earn a livelihood. For this reason, we have increasingly come to regard the medical-industrial complex as little more than a cartel. We define a cartel as a group of independent producers who band together to control the production, distribution, and pricing of a commonly shared commodity.

Peter McCullough confronted this organized wall of resistance when attempting to establish early treatment protocols for COVID-19 infected individuals. The universities that rely on hundreds of millions of dollars in annual funding from Fauci and the NIH were unmoved: 'We didn't have a single academic institution come up with a single protocol', he claims. 'They didn't even try. Harvard, Johns Hopkins, Duke, you name it. Not a single medical center set up even a tent to try to treat patients and prevent hospitalization and death. There wasn't one ounce of original research coming out of America to fight COVID—other than the vaccines' [494]. Is this at all surprising?

Finally, we harken back to the very *raison d'etre* of experimental science which, ostensibly, is to cultivate understanding and insight. Experiments are performed to establish the factuality of phenomena and to aid in making critical discriminations; at some point, however, facts must be incorporated into an overarching intellectual framework, i.e., a causal theory. It is at this juncture that molecular and cellular scientists have completely missed the boat. The pandemic has laid bare the limitations of the experimental method not to mention the failure of academic scientists to integrate valid facts into a coherent theoretical framework.

The pandemic revealed grave flaws in current immune theory which, as we showed in part I, can be traced back to the turn of the 20th century, *over 120 years ago*, involving questions that were never resolved by academics despite the fact that the correct interpretation had been advanced in the 1880s. This hardly instills confidence in either science or the experimental method. In the majority of instances one can draw the same conclusions by careful observation. The experimental method has been used as a shibboleth by academics to control public opinion and the arbitrary knowledge content of their science. One might argue that it has far outlived its purpose and intent.

In part III we encountered the PC phenomenon, now

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regarded as the most powerful innate form of protection in living bodies, discovered accidentally in 1986 by molecular biologists, which has been the focus of *tens of thousands of experiments* over the past 35 years and yet, remains a complete enigma. Molecular science is long on description and detail but woefully short on explanation. In earlier publications we showed that the PC phenomenon is generated by a blood-borne energy field. To date no one has refuted our claim.

In part I we showed *on the basis of credible experimental evidence* that all pathophysiologic phenomena associated with COVID-19 infection are referable to diffuse endothelial inflammation and, in turn, that such inflammation is secondary to diastolic dysfunction and deficient energy generation in the vascular system. The primacy of the diastolic phase of the cardiac cycle has been recognized by medical science since the mid-1980s and, like the PC phenomenon, has been the subject of innumerable clinical studies. And yet, to our knowledge, not a single academic publication has pointed out the inescapable link between diastolic function and energy generation. Apparently, academics believe they can cherry-pick evidence that suits their own purposes.

Had timely adjudications been made on these critical issues by the academic community it would have been readily apparent to all that early treatment protocols advanced by McCullough, Kory, Cole, Viglione, and numerous others were not only fitting and proper but, in fact, *the most important* therapeutic approach to COVID-19 infection. Had this path been taken many, many deaths would have been prevented. The conclusion is unavoidable that the self-serving academic community played a major role in the pandemic tragedy.

Such developments not only sing the swan song of molecular and cellular medicine but point to the failure of academics to perform their designated function, i.e., to maintain a vibrant and evolving science. If the academics can't keep their own house in order who will step in to fill the void? The pandemic has shown quite clearly that intelligent and motivated physicians are fully capable of making necessary clinical adjudications and choosing effective treatments and, in fact, do not need the kind of patronistic babysitting afforded by the academic cartel. East is East, West is West, and never the twain shall meet. There must be a parting of ways between the two, a reconciliation through separation so to speak, that will permit the emergence and evolution of a vital new integrated and functionally based system of medicine. We develop this theme further in future articles.

References

1. Convalescent Plasma Antibody Levels and the Risk of Death from Covid-19. Joyner MJ, Carter RE, Senefeld JW, et al. *NEJM* 2021; 384(11):1–13

2. Differences in Antibody Kinetics and Functionality Between Severe and Mild Severe Acute Respiratory Syndrome Coronavirus 2 Infections. Rijkers G, Murk JL, Wintermans B, van Looy B, et al. *J Infect Dis*2020;222(8):1265–9.

3. Antibody responses to SARS-CoV-2 in patients with differing severities of coronavirus disease 2019. Kowitdamrong E, Puthanakit T, Jantarabenjakul W, et al. *PLoS One*2020; 15(10): e0240502

4. Kinetics of Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) Antibody Avidity Maturation and Association with Disease Severity. Luo YR, Chakraborty I, Yun C, et al. *Clin Infect Dis* 2021;73(9): e3095-97

5. The kinetics of humoral response and its relationship with the disease severity in COVID-19. Ren L, Zhang L, Chang D, et al. *Commun Biol*2020; 3(1):1–7

6. A longitudinal study of SARS-CoV-2 infected patients reveals a high correlation between neutralizing antibodies and COVID-19 severity. Legros V, Denolly S, Vogrig M, et al. *Cell Mol Immunol*2021; 18(2): 318-27

7. The dynamics of immune response in COVID-19 patients with different illness severity. Zhang B, Yue D, Wang Y, et al. *J Med Virol 2020* July:1–8.

8. COVID-19 Pandemic Planning Scenarios.*Centers for Disease Control and Prevention*March 19, 2021. https://www.cdc.gov/coronavirus/2019-ncov/hcp/planning-scenarios.html

9. Age and frailty are independently associated with increased COVID-19 mortality and increased care needs in survivors: results of an international multi-centre study. Geriatric Medicine Research Collaborative; Covid Collaborative, Welch C.*AgeAgeing*2021;50(3):617-30

10. Detrimental effect of diabetes and hypertension

*Hyperlink to 1,366 references for COVID-19 vaccine associated complications:
https://www.thegms.co/publichealth/pubheal-rw-22042302-references.pdf

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

on the severity and mortality of COVID-19 infection: a multi-center case-control study from India. Jayaswal SK, Singh S, Malik PS, et al. *Diabetes MetabSyndr*2021;15(5):102248

11. Prevalence of co-morbidities and their association with mortality in patients with COVID-19: a systematic review and meta-analysis.Singh AK, Gillies CL, Singh R, et al. *Diabetes ObesMetab*2020;22(10):1915-24

12. Diabetes mellitus and hypertension increase risk of death in novel coronavirus patients irrespective of age: a prospective observational study of co-morbidities and COVID-19 from India.Gupta A, Nayan N, Nair R, et al. *Compr Clin Med* 2021;3(4):937-44

13. Frailty is associated with in-hospital mortality in older hospitalized COVID-19 patients in the Netherlands: the COVID-OLD study. Blomaard LC, van der Linden CMJ, van der Bol JM, et al. *Age Ageing*2021; 50(3):631-40

14. Age, frailty, and comorbidity as prognostic factors for short-term outcomes in patients with coronavirus disease 2019 in geriatric care.Hägg S, Jylhävä J, Wang Y, et al. *J Am Med Dir Assoc*2020;21(11):1555-1559.e2

15. Clinical frailty scale (CFS) indicated frailty is associated with increased in-hospital and 30-day mortality in COVID-19 patients: a systematic review and meta-analysis.Rottler M, Ocskay K, Sipos Z, et al. *Ann Intensive Care*2022:12(1):17

16. Characteristics and outcomes of frailty admitted to ICU with coronavirus disease 2019: an individual patient data meta-analysis.Subramaniam A, Anstey C, Curtis JR, et al. *Crit Care Explor* 2022;4(1):e0616

17. Conditions favoring increased COVID-19 morbidity and mortality: their common denominator and its early treatment.Shevel E. *Mo Med*2021;118(2):113-15

18. Is it all in the heart?Myocardial injury as a major predictor of mortality among hospitalized COVID-19 patients.Harmouch F, Shah K, Hippen JT, et al. *J Med Virol*2021;93(2);973

19. Pre-existing health conditions and severe COVID-19 outcomes: an umbrella review approach and meta-analysis of global evidence.Treskova-Schwarzbach M, Haas L, Reda S, et al. *BMC Med* 2021;19(1):212

20. Diabetes, hypertension, body mass index, smoking and COVID-19-related mortality: a systematic review and meta-analysis of observational studies.Mahamat-Saleh Y, Fiolet T, Rebeaud ME, et al. *BMJ Open*2021; 11(10):e052777

21. Clinical characteristics and morbidity associated with coronavirus disease 2019 in a series of patients in metropolitan Detroit.Suleyman G, Fadel RA, Malette KM, et al. *JAMA Netw Open*3(6):e2012270

22. Disease severity dictates SARS-CoV-2-specific neutralizing antibody responses in COVID-19. Chen X, Pan Z, Yue S, et al.*Signal Transduct Target Ther*2020; 5(1):1–6.

23. Antibody Responses in COVID-19: A Review. Chvatal-Medina M,Mendez-Cortina Y, Pablo J. Patiño PJ, et al. *Front Immunol*2021; 12:633184216

24. Immune responses to SARS-CoV-2 infection in hospitalized pediatric and adult patients.Pierce CA, Preston-Hurlburt P, Dai Y, et al. *Sci Transl Med*2020; 12(564): eabd5487

25. High neutralizing antibody titer in intensive care unit patients with COVID-19. Liu L, To KKW, Chan KH, et al. *Emerg Microbes Infect*2020; 9(1):1–30

26. Anti–SARS-CoV-2 Antibody Responses in Convalescent Plasma Donors Are Increased in Hospitalized Patients; Subanalyses of a Phase 2 Clinical Study. Microorganisms. Terpos E, Politou M, Sergentanis TN, et al. *Microorg*2020; 8(12): 1885

27. Sex, age, and hospitalization drive antibody responses in a COVID-19 convalescent plasma donor population.Klein SL, Pekosz A, Park HS, et al. *J Clin Invest*2020; 130(11):6141–50

28. Postconvalescent SARS-CoV-2 IgG and neutralizing antibodies are elevated in individuals with poor metabolic health.Racine-Brzostek SE, Yang HS, Jack GA, et al. *J Clin Endocrinol Metab* 2021; 106(5): e2025--e2034

29. Autophagy in major human diseases. Klionsky DJ, Petroni G, Amaravadi RK, et al. *EMBO*2021;40(19):e108863

30. Autophagy and inflammation.Matsuzawa-Ishimoto Y, Hwang S, Cadwell K. *Annu RevImmunol*2018; 36:73-101

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

31. Dual role of autophagy in hallmarks of cancer.Singh SS, Vats S, Chia AY, et al. *Oncogene*37(9):1142-58

32. Targeting autophagy to overcome human diseases. Condello M, Pellegrini E, Caraglia M, etal. *Int J Mol Sci*2019;20(3):725

33. Autophagy, Warburg, and Warburg reverse effects in human cancer. Gonzalez CD, Alvarez S, Ropolo A, et al. *Biomed Res Int*2012;1820(5):595-600

34. Autophagy in chronic kidney disease.Lin TA, Wu VC, Wang CY. *Cells*2019;8(1):61

35. Mitochondrial uncoupling: a key controller of biological processes in physiology and diseases.Demine S, Renard P, Arnould T. *Cells*2019;8(8):795

36. Mitochondrial dysfunction and autophagy in hepatic ischemia-reperfusion injury. Go KL, Lee S, Zendejas I, et al. *Biomed Res Int*2015;2015:183469

37. Mitochondria and the autophagy-inflammation-cell death axis in organismal aging. Green DR, Galluzzi L, Kroemer G2011;333(6046):1109-12

38. Autophagy, organelles and ageing.Terman A, Gustafsson B, Brunk UT. *J Pathol*211(2):134-43

39. Mitochondria, autophagy and age-associated neurodegenerative diseases: new insights into a complex interplay.Lionaki E, Markaki M, Palikaras K, et al. *BiochimBiophys Acta*2015;1847(11): 1412-23

40. Role of mitochondrial inner membrane permeabilization in necrotic cell death, apoptosis, and autophagy.Lemasters JJ, Qian T, He L, et al. *Antioxid Redox Signal 2002*;4(5):769-81

41. Mitochondrial dysfunction in the pathogenesis of necrotic and apoptotic cell death. Lemasters JJ, Qian T, Bradham CA, et al. *BioenergBiomembr*1999;31(4):305-19

42. Critical role of mitochondrial dysfunction and impaired mitophagy in diabetic nephropathy.Saxena S, Mathur A, Kakkar P. *J Cell Physiol*2019;234(11):19223-236

43. Mitochondria and autophagy: critical interplay between the two homeostats.Okamoto K, Kondo-Okamoto N. *BiochimBiophys Acta*2012;1820(5):595-600

44. Age-associated mitochondrial dysfunction accelerates atherogenesis.Tyrrell DJ, Blin MG, Song J, et al. *Circ Res*2020;126(3):298-314

45. Aging impairs mitochondrial function and mitophagy and elevated interleukin 6 within the cerebral vasculature. Tyrrell DJ, Blin MG, Song J, et al. *J Am Heart Assoc.* 9(23):e17820

46. COVID-19 and ROS storm: what is the forecast for hypertension? de Oliveira AA, Priviero F, Lima VV, et al. *Am J Hypertens*2021;34(8):779-82

47. Elucidating of oxidative distress in COVID-19 and methods of its prevention. BarciszewskaAM. Chem *Biol Interact* 2021; 344:109501

48. Oxidative stress as key player in Severe Acute Respiratory Coronavirus (SARS-CoV) infection. Delgado-Roche L, Mesta F. *Arch Med Res*2020;51(5):384-87

49. Neurological implications of COVID-19: role of redox imbalance and mitochondrial dysfunction. Kaundal RK, Kalvala AK, Kumar A. *Molecular Neurobiol*2021; 58(9):4575-87

50. Reactive oxygen species, proinflammatory and immunosppressive mediators induced in COVID-19: overlapping biology with cancer.Kalyanaraman B. *RSC Chem Biol*2021;2(5):1402-14

51. Tissue damage from neutrophil-induced oxidative stress in COVID-19. Schönrich G, Raftery MJ, Samstag Y, et al. *Nat Rev Immunol* 2020; 20(9):515-16

52. The longitudinal immune response to coronavirus disease 2019: chasing the cytokine storm. Chau AS, Weber AG, Maria NI, et al. *Arthritis Rheumatol*2021; 73(1):23-35

53. Cytokine storm in COVID-19: pathogenesis and overview of anti-inflammatory agents used in treatment.Soy M, Keser G, Atagündüz P, et al. *Clin Rheumatol*2020;39(7):2085-94

54. Controlling the cytokine storm is vital in COVID-19.Tang L, Yin Z, Hu Y, et al. *Front Immunol*2020;11:570993

55. Inflammatory response in COVID-19 patients resulting from the interaction of the inflammasome and SARS-CoV-2.Cheon SY, Koo BN. *Int J Mol Sci*2021;22(15):7914

56. Targeting the NLRP3 inflammasome in severe COVID-19.Freeman TL, Swartz TH. *Front Immunol* 2020;11:1518

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

57. Inflammation and pyroptosis as therapeutic targets for COVID-19. Yap JKY, Moriyama M, Iwasaki A.*J Immunol* 2020;205(2):307-12

58. Controlling the cytokine storm is vital in COVID-19. Tang L, Yin Z, Hu Y, et al. *Front Immunol*2020;11:570993

59. The cytokine storm and COVID-19.Hu B, Huang S, Yin L. *J Med Virol*2021;93(1):250-56

60. Cytokine storm in COVID-19: pathogenesis and overview of anti-inflammatory agents used in treatment.Soy M, Keser G, Atagündüz P, et al. *Clin Rheumatol*2020;39(7):2085-94

61. COVID-19: consider cytokine storm syndromes and immunosuppression.Mehta P, McAuley DF, Brown M, et al. *Lancet*2020;395(10229):1033-34

62. Immunopathology of Hyperinflammation in COVID-19. Gustine JN, Jones D. *Am J Pathol* 2021;191(1):4-17

63. Hyperinflammation and immune response generation in COVID-19.Mishra KP, Singh AK, Singh SB. *Neuroimmunomodulation* 2020;27(2):80-86

64. Diastolic dysfunction in the elderly.Genesis and diagnostic and therapeutic implications.Kitzman DW. *Cardiol Clin*2000;18(3):597-617

65. Ventricular diastolic abnormalities in the critically ill.Vignon P. *CurrOpinCrit Care*2013;19(3):242-49

66. The vicious combination of left ventricular diastolic dysfunction and frailty.Ishuzu T. *Circ J*2018;82(8):1994-95

67. Prevalence and progress of left ventricular diastolic dysfunction in the elderly: the PROTEGER study.Pan B, Xu ZW, Xu Y, et al. *Am Heart J*2010;160(3):471-78

68. Left ventricular diastolic function in the elderly. Tokushima T, Reid CL, GardinJM *Am J GeriatrCardiol* 2001;10(1):20-29

69. Factors contributing to energy loss in left ventricle during diastolic and systolic phases in elderly patients. Futami S, Ishikawa J, Maeda T,et al. *Echocardiography*2021;38(1):72-80

70. Diastolic dysfunction and cardiac troponin I decrease in aging hearts.Pan B, Xu ZW, Xu Y, et al. *Arch BiochemBiophys*2016; 603:20-28

71. Pathophysiology of diastolic dysfunction in chronic heart failure.Segers VM, De Keulenaer GW. *Future Cardiol*2013;9(5):711-20

72. Evolving focus on diastolic dysfunction in patients with coronary artery disease.Ohara T,Little WC. *CurrOpinCardiol*2010;25(6):613-21

73. Diastolic dysfunction in arterial hypertension. De Simone G, Palmieri V. *J Clin Hypertens(Greenwich)*2001;3(1):22-27

74. Diastolic dysfunction precedes myocardial hypertrophy in the development of hypertension.Aeschbacher BC, Hutter D, Fuhrer J, et al. *Hypertens*2001; 14(2): 106-13

75. Left ventricular diastolic dysfunction in newly diagnosed untreated hypertensive patients. Fici F, Ural D, Tayfun S, Kozdag G, et al. *Blood Press*2012; 21(6): 331-37

76. Relation of arterial stiffness to diastolic dysfunction in hypertensive heart disease.Mottram PM, Haluska BA, Leano R, et al. *Heart*2005;91(12):1551-56

77. Diastolic dysfunction and hypertension.Nadruz W, Shah AM, Solomon SD. *Med Clin NorthAm* 2017;101(1):7-17

78. Effects of obesity on cardiovascular hemodynamics, cardiac morphology, and ventricular function.Alpert MA, Omran J, Bostick BP. *CurrObes Rep* 2016;5(4):424-34

79. Obesity and metabolic features associated with long-term development of diastolic dysfunction in an initially healthy population-based cohort.Chau K, Girard N, Magnusson M, Lamiral Z, et al. *Clin Res Cardiol* 2018;107(10):887-896

80. Left ventricular diastolic dysfunction in morbidly obese patients in the preoperative for bariatric surgery. Tavares IS, Sousa AC, Menezes Filho RS, et al. *Arq Bras Cardiol* 2012;98(4):300-06

81. Pre-clinical diastolic dysfunction. Wan SH, Vogel MW, Chen HH. *J Am Coll Cardiol*2014;63(5):407-16

82. Multimarker assessment of diastolic dysfunction in metabolic syndrome patients.Mocan M, An-

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

ton F, Suciu S, Rahaian R, et al. *MetabSyndrRelatDisord*2017;15(10):507-514

83. Metabolic syndrome and cardiovascular diseases in Korea.Suh S, Lee MK. *J AtherosclerThromb* 2014;21(Suppl 1):S31-36

84. The metabolic syndrome in early pregnancy and risk of gestational diabetes mellitus. Chatzi L, Plana E, Pappas A, Alegkakis D, et al. *Diabetes Metab*2009;35(6):490-94

85. Metabolic syndrome is associated with electrical and mechanical dysfunction.Yilmaz H,Özcan KS, Sayar N, Kemaloglu T, et al.*Med PrincPract*2015;24(2):147-52

86. Left ventricular dysfunction in diabetes mellitus: An update.Freire CM, Moura AL, Barbosa Mde M, Machado LJ, et al. *Arq Bras Endocrinol Metab*2007;51(2):168-75

87. Diastolic dysfunction in asymptomatic hemodialysis patients in the light of current echocardiographic guidelines. Malik J, Kudlicka J, Valerianova A, et al. *J Cardiovasc Imaging*2019;35(2):313-17

88. Left ventricular diastolic dysfunction in early stage chronic kidney disease. Otsuka T, Suzuki M, Yoshikawa H, Sugi K. *J Cardiol*2009;54(2):199-204

89. Left ventricular diastolic dysfunction by tissue Doppler echocardiography in pediatric chronic kidney disease.Lindblad YT, Axelsson J, Balzano R, Vavilis G, et al. *PediatNephrol*2013;28(10):2003-13

90. Impaired systolic and diastolic left ventricular function in children with chronic kidney disease— results from the 4C study.Doyon A, Haas P, Erdem S, Ranchin B, et al. *Sci Rep*2019;9(1):11462

91. Left ventricular diastolic dysfunction in normotensive postmenopausal women with type 2 diabetes mellitus.Maiello M, Zito A, Cecere A, et al. *Cardiol J*2017;241:51-56

92. Left ventricular diastolic dysfunction in diabetic patients: pathophysiology and therapeutic implications.Tsujino T, Kawasaki D, Masuyama T. *Am J Cardiovasc Drugs*2006;6(4):219-30

93. Association between non-alcoholic fatty liver disease and left ventricular diastolicdysfunction in patients of type 2 diabetes.Saluja M, Kumar K, Swami YK, Meena SR. *J AssocPhysicians India* 2019;67(8):20-24

94. Nonalcoholic fatty liver disease and advanced fibrosis are associated with left ventricular diastolic dysfunction.Chung GE, Lee J-H, Lee H, Kim MK, et al. *Atherosclerosis*2018May;272:137-144.

95. Non-alcoholic fatty liver disease is associated with left ventricular diastolic dysfunction in essential hypertension.Fallo F, Dalla Pozza A, Sonino N Lupia M, et al. *NutrMetab CardiovascDis*2009 Nov;19(9):646-53

96. Association between diastolic cardiac dysfunction and nonalcoholic fatty liver disease: A systematic review and meta-analysis.Wijarnpreecha K, Lou S, Panjawatanan P, et al. *Dig LiverDis*2018 Nov;50(11):1166-1175.

97. Diastolic function as an early marker for systolic dysfunction and all-cause mortality among cancer patients.Arnold JH, Rozenbaum Z, Hochstadt A, et al.*Echocardiography*2021;38(4):540-48

98. Systolic and diastolic dysfunction in long-term adult survivors of childhood cancer. Brouwer CA, Postma A, Vonk JM, et al. *Eur J Cancer*2011;47(16):2453-62

99. Diastolic myocardial dysfunction by tissue Doppler Imaging predicts mortality in patients with cerebral infarction.Olsen FJ, Jørgensen PG, Møgelvang R, et al. *Int J Cardiovasc Imaging*2015;31(7):1413-22

100. Unexpected magnetic attraction: Evidence for an organized energy field in the human body.Thorp JA, Thorp KE, Lile EK, Viglione J. *G Med Sci*2021;2(4):001-015.

101. Aether, fields & energy dynamics in living bodies - Part I.Thorp KE, Thorp JA, Walker PR.*G Med Sci*2021;2(5):014-025.

102. Aether, fields & energy dynamics in living bodies - Part II.Thorp KE, Thorp JA, Walker PR. *GMed Sci*2021;2(6):001-020.

103. Aether, fields & energy dynamics in living bodies - Part III.Thorp KE, Thorp JA, Walker PR *GMed Sci*2021;2(6):021-047.

104. Preconditioning with ischemia: a delay of lethal cell injury in ischemic myocardium.MurryCE, Jennings RB, Reimer KA.Circulation. 1986 Nov;74(5):1124-36.

105. Ischemic preconditioning reduces infarct size in swine myocardium.Schott RJ, RohmannS, Braun ER, Schaper W.*Circ Res*1990 Apr;66(4):1133-42.

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

106. Effect of preconditioning ischemia on reperfusion arrhythmias after coronary arteryocclusion and reperfusion in the rat.Hagar JM, Hale SL, Kloner RA.*Circ Res*1991 Jan;68(1):61-8.

107. Preconditioning myocardium with ischemia.Jennings RB, Murry CE, Reimer KA.*CardiovascDrugs Ther*1991 Oct;5(5):933-8.

108. Blockade of ATP-sensitive potassium channels prevents myocardial preconditioning indogs.Gross GJ, Auchampach JA.*Circ Res*1992 Feb;70(2):223-33.

109. Ischemic preconditioning attenuates acidosis and postischemic dysfunction in isolated rat heart.Asimakis GK, Inners-McBride K, Medellin G, Conti VR. *Am J Physiol*1992 Sep;263(3 Pt 7):H887-94.

110. Ischemic preconditioning protects against coronary endothelial dysfunction induced by ischemia and reperfusion.Richard V, Kaeffer N, Tron C, Thuillez C.*Circulation* 1994Mar;89(3):1254-61.

111. Ischemic Preconditioning Slows Ischemic Metabolism and Limits Myocardial Infarct Size. Reimer KA, Heide RSV, Jennings RB. *Ann NY Acad Sci*1994 Jun;723(1):99-115

112. Pronounced antiarrhythmic effects of ischemic preconditioning. Parratt J, Vegh A.*Cardiosci* 1994 Mar;5(1):9-18.

113. Ischemic preconditioning improves postischemic function, but not energy metabolism of skeletal muscles.Gürke L, Marx A, Sutter PM, et al. *Swiss Surg*1995;(2):107-9.

114. Ischemic preconditioning inhibits glycolysis and proton production in isolated working rat hearts.Finegan BA, Lopaschuk GD, Gandhi M, Clanachan AS. *Am J Physiol*1995 Nov;269(5 Pt 2):H1767-75

115. Adenosine slows ischaemic metabolism in canine myocardium in vitro: Relationship to ischaemic preconditioning.Vander Heide RS, Reimer KA, Jennings RB. *Cardiovasc Res*1993 Apr;27(4):669-73

116.The cardioprotective effects of ischemic 'preconditioning' are not mediated by adenosine receptors in rat hearts.Li Y, Kloner RA.*Circulation*1993 May;87(5):1642-8.

117. Oxygen radicals can induce preconditioning in rabbit hearts.Tritto I, D'Andrea D, Eramo N, et al. *Circ Res*1997 May;80(5):743-8.

118. Ischaemic preconditioning is not mediated by oxygen derived free radicals in rats.RichardV, Tron C, Thuillez C.*Cardiovasc Res*1993 Nov;27(11):2016-21

119. The protective effect of late preconditioning against myocardial stunning in conscious rabbits is mediated by nitric oxide synthase. Evidence that nitric oxide acts both as a trigger and as a mediator of the late phase of ischemic preconditioning. Bolli R, Manchikalapudi S, Tang XL, et al. *Circ Res*1997 Dec;81(6):1094-107.

120. Blockade of ATP-sensitive potassium channels increases infarct size but does not prevent preconditioning in rabbit hearts.Thornton JD, Thornton CS, Sterling DL, Downey JM.*Circ Res*1993 Jan;72(1):44-9.

121. Mitochondrial ATP-dependent potassium channels: novel effectors of cardioprotection?Liu Y, Sato T, O'Rourke B, Marban E.*Circulation*1998 Jun 23;97(24):2463-9

122. Nitric oxide mediates cerebral ischemic tolerance in a neonatal rat model ofhypoxic preconditioning. Gidday JM, Shah AR, Maceren RG, et al. *J Cereb Blood Flow Metab*1999 Mar;19(3):331-40

123. Ischemic preconditioning and the beta-adrenergic signal transduction pathway. Lochner A, Genade S, Tromp E, et al. Circulation1999 Aug 31;100(9):958-66

124. Preconditioning-mediated neuroprotection through erythropoietin?Dawson TM.Lancet.2002 Jan 12;359(9301):96-7

125. Expression of heat shock protein after ischemic preconditioning in rabbit hearts.TanakaM, Fujiwara H, Yamasaki K,et al. *Jpn Circ J*1998 Jul;62(7):512-6.

126. Regional ischemic 'preconditioning' protects remote virgin myocardium from subsequent sustained coronary occlusion.Przyklenk K, Bauer B, Ovize M, et al. *Circulation*1993Mar;87(3):893-9

127. Ischemic preconditioning at a distance: reduction of myocardial infarct size by partial reduction of blood supply combined with rapid stimulation of the gastrocnemius muscle in the rabbit.Birnbaum Y, Hale SL, Kloner RA.*Circulation*1997 Sep 2;96(5):1641-6

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

128. Renal ischemia/reperfusion remotely improves myocardial energy metabolism during myocardial ischemia via adenosine receptors in rabbits: effects of "remote preconditioning". Takaoka A, Nakae I, Mitsunami K, et al. *J Am Coll Cardiol*1999 Feb;33(2):556-64

129. Ischemic preconditioning attenuates functional, metabolic, and morphologic injury from ischemic acute renal failure in the rat. Cochrane J, Williams BT, Banerjee A, et al. *Ren Fail*1999 Mar;21(2):135-45

130. Acute ischaemic preconditioning protects against skeletal muscle infarction in the pig. Pang CY, Yang RZ, Zhong A, et al. *Cardiovasc Res.*1995 Jun;29(6):782-8

131. Role of ischemic preconditioning on ischemia-reperfusion injury of the lung.Soncul H, OzE, KalaycioguS.*Chest*1999 Jun;115(6):1672-7

132. Focal ischemic preconditioning induces rapid tolerance to middle cerebral artery occlusion in mice.Stagliano NE, Pérez-Pinzón MA, Moskowitz MA, Huang PL. *J Cereb Blood Flow Metab*1999 Jul;19(7):757-61

133. Ischemic preconditioning improves postischemic acute renal failure.Riera M, HerreroI, Torras J, et al. *Transplant Proc*1999;31(6):2346-7.

134. Induction of tolerance against traumatic brain injury by ischemic preconditioning. Pérez-Pinzón MA, Alonso O, Kraydieh S, Dietrich WD. *Neuroreport.* 1999 Sep 29;10(14):2951-4

135. Preconditioning protects against ischemia/reperfusion injury of the liver.Nilsson B, Friman S, Gustafsson BI, Delbro DS.*J Gastrointest Surg*2000 Jan-Feb;4(1):44-9

136. Hepatic preconditioning preserves energy metabolism during sustained ischemia.PeraltaC, Bartrons R, Riera L, et al.*Am J PhysiolGastrointest Liver Physiol.* 2000 Jul;279(1):G163-71

137. Intestinal ischemia induces late preconditioning against myocardial infarction: a role for inducible nitric oxide synthase.Wang Y, Xu H, Mizoguchi K, et al. *Cardiovasc Res*2001Feb1;49(2):391-8

138. Organ preconditioning.Raeburn CD, Cleveland JC Jr, Zimmerman MA, Harken AH. *ArchSurg*2001 Nov;136(11):1263-6.

139. Kidney ischemic preconditioning.Bonventre JV.*CurrOpinNephrolHypertens.* 2002Jan;11(1):43-8.

140. Brain tolerance and preconditioning.Rejdak R, Rejdak K, Sieklucka-Dziuba M, StelmasiakZ, Grieb P.*Pol J Pharmacol*2001 Jan-Feb;53(1):73-9.

141. Intestinal ischemic preconditioning protects the intestine and reduces bacterial translocation.Aksöyek S, Cinel I, Avlan D, et al. *Shock*2002 Nov;18(5):476-80.

142. Ischemic preconditioning may be transferable via whole blood transfusion: Preliminary evidence.Dickson EW, Reinhardt CP, Renzi FP, et al.*J Thromb Thrombolysis*1999 Aug;8(2):123-9.

143. Rabbit heart can be "preconditioned" via transfer of coronary effluent.Dickson EW, Lorbar M, Porcaro WA, et al.*Am J Physiol* 1999 Dec;277(6):H2451-7

144. "Preconditioning at a distance" in the isolated rabbit heart.Dickson EW, Porcaro WA, et al. *AcadEmerg Med.*2000 Apr;7(4):311-7

145. Infarct limitation of the second window of protection in a conscious rabbit model.YangXM, Baxter GF, Heads RJ, et al. *Cardiovasc Res*1996 May;31(5):777-83

146. A "second window of protection" occurs 24 h after ischemic preconditioning in the rat heart. Yamashita N, Hoshida S, Taniguchi N, et al. *J Mol Cell Cardiol*1998;30(6):1181-9

147. Ischemic preconditioning: from the first to the second window of protection.PagliaroP, Gattullo D, Rastaldo R, Losano G.*Life Sci*2001 May 25;69(1):1-15.

148. Heat shock. A new approach for myocardial preservation in cardiac surgery.LiuX, Engelman RM, Moraru II. *Circulation*1992 Nov;86(5 Suppl):II358-63.

149. Non-ischemic myocardial preconditioning.Domenech R, Macho P.*Mol Cell Biochem*-1998Sep;186(1-2):201-3

150. Ischemic preconditioning during successive exercise testing.Zdrenghea D, Ilea M, Predescu D, Potâng E.*Rom J Intern Med*1998 Jul-Dec;36(3-4):161-5.

151. Regular alcohol consumption mimics cardiac preconditioning by protecting against ischemia-reperfusion injury.Miyamae M, Diamond I, Weiner MW, et al. *Proc Natl Acad Sci USA*1997 Apr 1;94(7):3235-9

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

152. Pharmacological preconditioning of ischaemia. LaghiPasini F, Capecchi PL, et al. *ClinHemorheolMicrocirc*1997 Jan-Feb;17(1):73-84

153. Oxygen radicals can induce preconditioning in rabbit hearts.Tritto I, D'Andrea D, Eramo N, et al. *Circ Res*1997 May;80(5):743-8.

154. Myocardial preconditioning using adenosine: review and clinical experience.ZarroDL, Palanzo DA, Sadr FS.*Perfusion*1998 Mar;13(2):145-50

155. Thermal preconditioning protects rat cardiac muscle cells from doxorubicin-induced apoptosis.Ito H, Shimojo T, Fujisaki H, et al. *Life Sci*1999;64(9):755-61

156. Hyperthermic preconditioning prevents blood-brain barrier disruption produced by hypoxia-ischemia in newborn rat.Ikeda T, Xia XY, Xia YX, Ikenoue T. *Brain Res Dev Brain Res.* 1999 Oct 20;117(1):53-8.

157. The effect of hyperthermic preconditioning on the immune system in rat peritonitis. Ozveri ES, Bekraki A, Cingi A, et al. *Intensive Care Med*1999 Oct;25(10):1155-9.

158. Preconditioning with erythropoietin protects against subsequent ischemia-reperfusion injury in rat kidney.Yang CW, Li C, Jung JY, et al. *FASEB*2003 Sep;17(12):1754-5

159. Cardioprotection by volatile anesthetics.Bienengraeber MW, Weihrauch D, Kersten JR, et al. *Vascular Pharmacol*2005 Apr-May;42(5-6):243-52

160. Exercise preconditioning upregulates cerebral integrins and enhances cerebrovascular integrity in ischemic rats.Ding YH, Li J, Yao WX, et al. *Acta Neuropathol*2006 Jul;112(1):74-84.

161. Exercise preconditioning reduces brain damage and inhibits TNF-alpha receptor expression after hypoxia/reoxygenation: an in vivo and in vitro study. Ding YH, Mrizek M, Lai Q, et al. *CurrNeurovasc Res.*2006 Nov;3(4):263-71.

162. Exercise preconditioning of the myocardium. KavazisAN.*Sports Med.*2009;39(11):923-35

163) Ginkgolide B preconditioning protects neurons against ischaemia-induced apoptosis.WuX, Qian Z, Ke Y, Du F, Zhu L. *J Cell Mol Med.* 2009;13(11-12):4474-83

164. Helium preconditioning attenuates hypoxia/ischemia-induced injury in the developing brain.Liu Y, Xue F, Liu G, et al. *Brain Res.* 2011 Feb;1376:122-9

165. Resveratrol Preconditioning Induces a Novel Extended Window of Ischemic Tolerance in the Mouse Brain.Koronowski KB, Dave KR, Saul I, et al. *Stroke*2015 Aug;46(8):2293-8

166. Pharmacologic preconditioning: translating the promise.Gidday JM. *Transl StrokeRes.* 2010 Jan 3;1(1):19-30

167. Ozone preconditioning: Waking up the dragon. Thorp KE, Thorp JA. *G Med Sci*2021;2(3):10-39

168. The use of ozone-treated blood in the therapy of HIV infection and immune disease: A pilot study of safety and efficacy.Garber GE, Cameron DW, Hawley-Foss N, et al. *AIDS*1991;5(8): 981-84

169. A reasonable approach for the treatment of HIV infection in the early phase with ozone therapy (autohaemotherapy). How 'inflammatory' cytokines may have a therapeutic role.Bocci V. *Mediators Inflamm*1994;3(5):315-21

170. Inactivation of human immunodeficiency virus type 1 by ozone in vitro.Wells KH, Latino J,Gavalchin J, et al. *Blood* 1991;78(7):1882-90

171. Ozonation of blood for the therapy of viral diseases and immunodeficiencies.A hypothesis.Bocci V. *Med Hypotheses*1992;39(1):30-34

172. Beneficial effects of nontoxic ozone on H_2O_2-induced stress and inflammation.Kucukgul A,Erdogan S, Gonenci R, Ozan G. *Biochem Cell Biol*2016 Dec. 94(6);577-583.

173. Ameliorative effects of ozone on cytokine production in mice injected with humanrheumatoid arthritis synovial fibroblast cells.Chang JD, Lu HS, Chang YF, et al. *Rheumatol Int*2005;26(2):142-51.

174. The effect of intra-articular injection of different concentrations of ozone on the level of TNF-alpha, TNF-R1, and TNF-R2 in rats with rheumatoid arthritis. Chen H, Yu B, Lu C, et al. *Rheumatol Int*2013;33(5):1223-27.

175. Changes in Th17 cells frequency and function after ozone therapy used to treat multiplesclerosis patients. Dadashpour M, Yousefi M, Ahmadi M. *MultSclerRelatDisord*2020;46:102466.

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

176. Anti-inflammatory effect of ozone therapy in an experimental model of rheumatoid arthritis.Tartari APS, Moreira FF, Pereira MCDS, et al. *Inflammation*2020;43(3):985-993.

177. Ozone therapy attenuates NF-kappab-mediated local inflammatory response and activation of Th17 cells in treatment of psoriasis. Zeng J, Lei L, Zeng Q, et al. *Int J Biol Sci*2020;16(11):1833-1845.

178. Ozone therapy ameliorates inflammation and endometrial injury in rats with pelvicinflammatory disease.Wei A, Feng H, Jia X-M. *Biomed Pharmacother*2018;107:1418-25

179. Effect of ozone oxidative preconditioning on inflammation and oxidative stress injury in rat model of renal transplantation.Wang Z, Han Q, Guo YL, et al. *Acta Cir Bras*2018; 33(3);238-49.

180. Medical ozone treatment ameliorates the acute distal colitis in rat.Aslaner A, Çakır T,Tekeli SÖ, Avcı S, et al. *Acta Cir Bras*2016 Apr;31(4):256-63.

181. The effects of ozone therapy on caspase pathways, TNF-alpha, and HIF-1alpha in diabetic nephropathy. Güçlü A, Erken HA, Erken G, Dodurga Y et al.*Int Urol-Nephrol.* 2016; 48(3):441-50

182. Ozone therapy prevents renal inflammation and fibrosis in a rat model of acutepyelonephritis.Caliskan B, Guven A, Ozler M, et al. *Scand J Clin Lab Invest* 2011;71(6);473-80.

183. Assessment of effect of ozonated water irrigation on gingival inflammation in patients undergoing fixed orthodontic treatment.Jose P, Ramabhadran BK, Emmatty R, Paul TP. *J IndianSoc Periodontol*2017;21(6);484-488.

184. Therapeutic efficacy of ozone in patients with diabetic foot.Martınez-Sanchez G, Al-Dalain SM, Menendez S, Re L, et al. *Eur J Pharmacol* 2005;523:151-161

185. Ozone therapy ameliorates inflammation and endothelial injury in rats with pelvic inflammatory disease.Wei A, Feng H, Jia XM, et al.*Biomed Pharmacother*2018;107;1418-1425.

186. Ozone Therapy in Patients with Viral Hepatitis C: Ten Years' Experience. Mawsouf MN,Tanbouli TT, Viebahn-Hänsler R. *J Int Ozone Ass*2012;34(6):451-458

187. Ozone therapy prevents the onset of dysplasia in HPV16-transgenic mice—a pre-clinical efficacy and safety analysis.Peirone C, Mestre VF, Medeiros-Fonseca B, et al. *BiomedPharmacother*2018;104:275-279

188. In Vitro Inactivation of Herpes Virus by Ozone.Petry G, Rosato LG, Nespoto J. *J Int Ozone-Ass*36(3):249-252

189. Ozone and oxidation therapies as a solution to the emerging crisis in infectious disease management: A review of current knowledge and experience.Rowen RJ. *Med Gas Res*2019; 9(4):232-37

190. Assessment of the effect of ozonated water irrigation of gingival inflammation in patients undergoing fixed orthodontic treatment. Jose P, Ramabhadran BK, Emmatty R, et al. *J IndianSoc Periodontol*2017;21(6);484-488.

191. Effect of ozone oxidative preconditioning on inflammation and oxidative stress injury in rat model of renal transplantation.Wang Z, Han Q, Guo YL, et al. *Acta Cir Bras*2018; 33(3);238-249.

192. Medical ozone increases methotrexate clinical response and improves cellular redoxbalance in patients with rheumatoid arthritis.León Fernández OS, Viebahn-Haensler R, CabrejaGL, Espinosa IS, et al. *Eur J Pharmacol*2016 Oct 15;789:313-318.

193. Validity of Oxygen-Ozone Therapy as Integrated Medication Form in Chronic Inflammatory Diseases. Bocci V, Zanardia I, Valacchi G, et al. *Cardiovasc HematolDisord Drug Targets*2015;15(2):127-38

194. Autohemotherapy with ozone as a possible effective treatment for Fibromyalgia.Moreno-Fernández A, Macías-García L, Valverde-Moreno R, et al. *Acta Reumatol Port*2019 44;244–249

195. Ozone therapy as add-on treatment in fibromyalgia management by rectal insufflation: An open-label pilot study. Hidalgo-Tallón J, Menéndez-Cepero S, Vilchez JS, et al. *J AlternComplementMed*2013;19:238–242.

196. Ozone therapy in 65 patients with fibromyalgia: An effective therapy.Tirelli U, Cirrito C,Pavanello M, et al. *Eur Rev Med Pharmacol Sci*2019;23:1786–1788.

197. Protective Effects of Ozone Oxidative Postconditioning on Long-term Injury After Renal Ischemia/Reperfusion in Rat. Jiang B, Su Y, Chen Q, et al. *Transplant Proc*2020; 52(1):365-372.

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

198. Ozone therapy prevents renal inflammation and fibrosis in a rat model of acute pyelonephritis. Caliskan B, Guven A, Ozler M, et al. Scand J Clin Lab Invest. 2011 Oct. 71(6);473-80.

199. Ozone oxidative postconditioning inhibits oxidative stress and apoptosis in renal ischemia and reperfusion injury through inhibition of MAPK signaling pathway. Wang L, Chen Z, Liu Y, etal. *Drug Des Devel-Ther*2018 May 21;12:1293-1301

200. Ozone therapy ameliorates tubulointerstitial inflammation by regulating TLR4 in adenine-induced CKD rats.Chen Z, Liu X, Yu G, et al. *Ren Fail*2016;38(5):822-30

201. Medical ozone therapy reduces shock wave therapy-induced renal injury.Uğuz S, Demirer Z, Uysal B, et al. *Ren Fail*2016;38(6):974-81

202. Ozone preconditioning attenuates contrast-induced nephropathy in rats.Kurtoglu T, Durmaz S, Akgullu C, et al. *J Surg Res*2015 May 15;195(2):604-11

203. Targeting and inflammation in chronic kidney disease.Ruiz S, Pergola PE, Zager RA, Vaziri ND. *Kidney Int*2013;83:1029-1041.

204. Effect of medical ozone therapy on renal blood flow and renal function of patients with chronic severe hepatitis.Gu XB, Yang XJ, Zhu HY, et al. *Chin Med J (Engl)* 2010;123(18):2510-13

205. Effects of medical ozone therapy on acetaminophen-induced nephrotoxicity in rats.Demirbag S, Uysal B, Guven A, et al. *Ren Fail*2010 May;32(4):493-7

206. Ozone oxidative post-conditioning in acute renal failure.Calunga JL, Trujillo Y, Menéndez S, et al. *J Pharm Pharmacol*2009 Feb;61(2):221-7

207. Reversion by ozone treatment of acute nephrotoxicity induced by cisplatin in rats.Gonzalez R, Borrego A, Zamora Z, et al. *Mediators Inflamm* 200413;307-312

208. Improvement of renal oxidative stress markers after ozone administration in diabetic nephropathy in rats. Morsy MD, Hassan WN, Zalat SI. DiabetolMetab Synd. 2010; 2: 29–35.

209. Preliminary results of ozone therapy as a possible treatment for patients with chronichepatitis C.Zaky S, Kamel SE, Hassan MS, et al. *J Altern Complement Med*2011 Mar;17(3):259-63.

210. Effects of ozone treatment on the infectivity of hepatitis A virus.Vaughn JM, Chen YS, Novotny JF, et al. Can J Microbiol1990;36(8):557-60

211. Clinical study of medical ozone therapy in chronic hepatitis B of 20 patients.Jiao XJ, Peng X.*Zhonghua Shi Yan He Lin Chuang Bing Du Xue Za Zhi*2008;22(6):484-5

212. The effect of rectal ozone on the portal vein oxygenation and pharmacokinetics of propranolol in liver cirrhosis (a preliminary human study.Zaky S, Fouad EA, Kotb HI. *Br J Clin Pharmacol*2011;71(3):411-5

213. The Role of Ozone Therapy in Hepatic Fibrosis due to Biliary Tract Obstruction.Kocaman H, Erginel B, Onder SY, et al. Eur J Pediatr Surg2016 Feb;26(1):133-7

214. Oxygen/ozone protects the heart from acute myocardial infarction through local increase of eNOS activity and endothelial progenitor cells recruitment. Di Filippo C, Luongo M, Marfella R, Ferraraccio F, et al. NaunynSchmiedebergs Arch Pharmacol2010 Sep;382(3):287-91.

215. Beneficial Effects of Ozone Therapy on Oxidative Stress, Cardiac Functions and Clinical Findings in Patients with Heart Failure Reduced Ejection Fraction. Buyuklu M, Kandemir FM, Set T, et al. *Cardiovasc Toxicol* 2017;17, 426–33

216. Antiarrhythmic effect of acute oxygen-ozone administration to rats.Di Filippo C, Cervone C, Rossi C et al. *Eur J Pharmacol*2010 Mar 10. 629(1-3);89-95

217. A single subcutaneous injection of ozone prevents allodynia and decreases the over-expression of pro-inflammatory caspases in the orbito-frontal cortex of neuropathic mice.Fuccio C, Luongo C, Capodanno P et al. *Eur J Pharm*2009,603,42–49.

218. AMPK activation by peri-sciatic nerve administration of ozone attenuates CCI-induced neuropathic pain in rats.Lu L, Pan C, Chen L, Hu L et al.*Mol Cell Biol*2017 Apr;9(2):132-143.

219. Intervertebral foramen injection of ozone relieves mechanical allodynia and enhances analgesic effect of gabapentin in animal models of neuropathic pain.Luo WJ, Yang F, Yang F, Sun W et al. *Pain Physician*2017 Jul;20(5):E673-E685.

220. Low back pain related to a sacral insufficiency fracture: role of paravertebral oxygen-ozone thera-

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

py in a paradigmatic case of nociplastic pain.de Sire A, Baricich A, Minetto MAet al. *Funct Neurol* 2019 Apr/Jun;34(2):119-122.

221. The effect and safety of ozone autohemotherapy combined withpharmacological therapy in postherpetic neuralgia.Hu B, Zheng J, Liu Q, Yang Y, Zhang Y. J PainRes2018 Aug 27;11:1637-1643.

222. Effects of ozone on the pain and disability in patients with failed back surgery syndrome.Barbosa DC, Ângelos JSD, Macena GMJ, et al. *Rev Assoc Med Bras*2017 Apr;63(4):355-360.

223. Efficacy and safety of percutaneous ozone injection around Gasserian ganglion for the treatment of trigeminal neuralgia: A multicentre retrospective study.Gao L, Chen RW, WilliamsJP et al. *J Pain Res*2020 May 4;13:927-936.

224. Effects of ozone applied by spinal endoscopy in patients with chronic pain related to failed back surgery syndrome: a pilot study.Magalhães FN, Soares SC, Torres JMet al. *NeuropsychiatrDis Treat*2013;9:1759-66.

225. Ozone-augmented percutaneous discectomy: a novel treatment option for refractory discogenic sciatica.Crockett MT, Moynagh M, Long N, et al. *Clin Radiol*2014 Dec;69(12):1280-86.

226. CT-guided oxygen-ozone treatment for first degree spondylolisthesis and spondylolysis.Bonetti M, Fontana A, Albertini F. *Acta Neurochir Suppl*2005;92:87-92.

227. Ischemic stroke penumbra and extracorporeal ozone treatment.Wasser G. *Neuroradiol J*2013;26(3):243-51.

228. Selective ozone concentrations may reduce the ischemic damage after a stroke.Frosini M, Contartese A, Zanardi I, et al. *Free Rad Res*2012 May;46(5):612-8

229. Brain ischemia and hypometabolism treated by ozone therapy.Clavo B, Suarez G, Aguilar Y et al. *ForschKomplementmed* 2011;18(5):283-7.

230. Effects of major ozonated autoheamotherapy on functional recovery, ischemic brain tissue apoptosis and oxygen free radical damage in the rat model of cerebral ischemia.Wu X, Liu X, Huang H, Li Z et al. *J Cell Biochem*2019 Apr;120(4):6772-6780.

231. Changes in Th17 cells frequency and function after ozone therapy used to treat multiple sclerosis patients. Izadi M, Tahmasebi S, Pustokhina I, et al. *MultSclerRelatDisord* 2020Nov;46:102466.

232. Mechanisms of pathophysiology of blood vessels in patients with multiple sclerosis treated with ozone therapy: A systematic review.Ameli J, Banki A, Khorvash F, et al. *Acta Biomed* 2019 Sep;90(3):213-217

233. The right method of ozone therapy used to treat multiple sclerosis patients.Travagli V. *MultSclerRelatDisord*2020 Nov;46:102545.

234. The effects of oxygen-ozone therapy on regulatory T-cell responses in multiple sclerosis patients. Tahmasebi S, Qasim MT, Krivenkova MV, et al. *Cell Biol Int* 2021; 45(7):1498-1509

235. Medical ozone promotes Nrf2 phosphorylation reducing oxidative stress and pro-inflammatory cytokines in multiple sclerosis patients.Delgado-Roche L, Riera-Romo M, Mesta F, et al. *Eur J Pharmacol*2017 Sep 15;811:148-154

236. Ozone: a natural bioactive molecule with antioxidant property as potential new strategy in aging and in neurodegenerative disorders.Cassellati C, Galoforo AC, Bonvicini C, et al. *AgeingRes Rev*2020;63:101138

237. Ozone therapy in COVID-19: a narrative review.Cattel F, Giordano S, Bertiond C, et al. *Virus Res* 2021;291:198207

238. Ozone therapy for the treatment of COVID-19 pneumonia: A scoping review.Izadi M,Cegolon L, Javanbakht M, et al. *Int Immunopharmacol*2021. Mar;92:107307.

239. Potential role of oxygen-ozone therapy in treatment of COVID-19 pneumonia.HernándezA, Viñals M, Isidoro T, et al. *Am J Case Rep*2020;21:e925849.

240. Ozone therapy for patients with COVID-19 pneumonia: preliminary report of a prospective case-control study.Hernández A, Viñals M, Pablos A, et al. *Int Immunopharmacol*2021;90:107261

241. Two known therapies could be useful as adjuvant therapy in critical patients infected by COVID-19. Hernández A, Papadakos PJ, Torres A. 2020; 67(5):245-52

242. Safety and efficacy of ozone therapy in mild to moderate COVID-19 patients: a phase I/II random-

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

ized control trial (SEOT study).Shah M, Captain J, Vaidya V, et al. *Int Immunopharmacol*2021; 91:107301

243. A preliminary evaluation on the efficacy of ozone therapy in the treatment of COVID-19. Zheng Z, Dong M, Hu K. *J Med Virol*2020; 92(11):2348-50

244. Ozone as adjuvant support in the treatment of COVID-19: A preliminary report ofprobiozovid trial. Araimo F, Imperiale C, Tordiglione P, et al. *Med Virol*2021;93(4):2210-20

245. Effectiveness of ozone therapy in addition to conventional treatment on mortality in patients with COVID-19.Çolak Ş, Genç Yavuz B, et al. *J Clin Pract*2021;75(8):e14321

246. Novel therapy for COVID-19. Does intravenous ozonated-saline affect blood and tissue oxygenation? Thorp JA, Hollonbeck SA, Viglione DD, et al. *J Gyn Res Obstet* ISSN:2581-5288

247. Deborah D. Viglione MD, Presented at the Frontier's in Ozone Conference in Miami, Florida November 5th,2021

248. A preliminary evaluation on the efficacy of ozone therapy in the treatment of COVID-19.Zheng Z, Dong M, Hu K. *J Med Virol*2020 Nov;92(11):2348-2350

249. Safety and efficacy of ozone therapy in mild to moderate COVID-19 patients: A phase 1/11 randomized control trial (SEOT study).Shah M, Captain J, Vaidya V, et al. *Int Immunopharmacol*2021.91:107301

250. Oxygen-ozone (O_2-O_3) immunoceutical therapy for patients with COVID-19. Preliminary evidence reported.Franzini M, L. Valdenassi, G. Ricevuti, S, et al. *Int Immunopharmacol.*2020; 88:106879

251. Potential Cytoprotective Activity of Ozone Therapy in SARS-CoV-2/COVID-19.Martínez-Sanchez G, Schwartz A, Di Donna V. *Antioxidants (Basel)*2020; 9:389

252. Ozone Therapy as a Possible Option in COVID-19 Management.Gavazza A, Marchegiani A,Rossi G, et al. *Front Pub Health*2020;8:417.

253. Ozone (O3) and SARS-CoV-2: Physiological Bases and Their Therapeutic Possibilities According to COVID-19 Evolutionary Stage.Fernandez-Cuadros ME, Albaladejo-Florín MJ, Pena-Lora D et al. *SN Compr. Clin. Med*2020;2(8):1094-1102

254. Potential mechanisms by which the oxygen-ozone (O2–O3) therapy could contribute to the treatment against the coronavirus COVID-19.Valdenassi UTL, Franzini M, Ricevuti G, et al. *EurRev Med Pharmacol Sci*2020; 24:4059–4061

255. The Biochemical and Pharmacological Properties of Ozone: The Smell of Protection in Acute and Chronic Diseases.Di Mauro R, Cantarella G, Bernardini R, Di Rosa M.*Int J Mol Sci*2019 20(3):634.

256. Mechanisms of Action Involved in Ozone Therapy: Is healing induced via a mild oxidative stress?Sagai M, Bocci V *Med Gas Res*2011Dec 20;1:29.

257. Ozone Therapy: A clinical review.Elvis AM, Ekta JS. *J Nat Sci Biol Med 2011*;2(1):66-70

258. Ozone therapy: An overview of pharmacodynamics, current research, and clinical utility. Smith NL, Wilson AL, et al. *Med Gas Res*2017 Oct 17.7(3);212-219.

259. A multifaceted molecule with unexpected therapeutic activity.Zanardi I, Borrelli E, Valacchi G et al.*Ozone Curr Med Chem*2016;23(4):304-14

260. Oxidant-specific biomarkers of oxidative stress. Association with atherosclerosis and implication for antioxidant effects.Niki E. Free Rad Biol Med. 2018 May 20;120:425-440.

261. Pisoschi AM, Pop A. The role of antioxidants in the chemistry of oxidative stress: A review. 2015 Jun 5. Eur J Med Chem97:55-74.

262. Kattoor AJ, Pothenini NVK, Palagiri D, Mehta JL. Oxidative Stress in Atherosclerosis. CurrAtheroscler Rep. 2017 Sep 18. 9(11);42.

263. Strohmaier H, Hinghofer-Szalkay H, Schaur RJ. Detection of 4-hydroxynonenal (HNE) as a physiological component in human plasma. J Lipid Mediat Cell Signal. 199511:51-61.

264. A physicochemical investigation on the effects of ozone on blood.Travagli V, Zanardi I, Silvietti A, Bocci V.*Int J Biol Macromol*2007 Dec 1. 41(5);504-11.

265. Erythrocyte ascorbate recycling: Antioxidant effects in blood.Mendiratta S, Qu ZC, May JM *Free Rad*

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

*Biol Med*199824:789-797.

266. Enzyme-dependent ascorbate recycling in human erythrocytes: Role of thioredoxin reductase.Mendiratta S, Qu ZC, May JM. *Free Rad Biol Med* 1998. 25;221-228.

267. The emerging role of reactive oxygen and nitrogen species in redox biology and some implications for plasma applications to medicine and biology. Graves DB.*J Phys D Appl Phys*2012;45;263001.

268. Total antioxidant status in plasma and body fluids.Rice-Evans C, Miller NJ. *MethodsEnzymol* 1994234:279-293.

269. Regulation of red blood cell deformability is independent of red blood cell-nitric oxide synthase under hypoxia. Grau M, Lauten A, Hoeppener S, et al. *Clin HemorheolMicrocirc*2016 Sep 12. 63(3);199-215.

270. Remote ischemia preconditioning increases red blood cell deformability through red blood cell-nitric oxide synthase activation.Grau M, Kollikowski A, Bloch W. *Clin HemorheolMicrocirc* 2016; 63(3):185-97.

271. Nitric oxide, vasodilation and the red blood cell.Simmonds MJ, Detterich JA, Connes P*Biorheology*2014; 51(2-3);121-34.

272. Circulating nitrite contributes to cardioprotection by remote ischemic preconditioning.Rassaf T, Totzeck M, Hendgen-Cotta UB, et al. *Circ Res*2014;114(10):1601-10.

273. Ischemic Preconditioning enhances performance and erythrocyte deformability of responders.Tomschi F, Niemann D, Bloch W, et al. *Int J Sports Med*2018 Jul. 39(8);596-603.

274. Is red blood cell a mediator of remote ischaemic preconditioning?Gopalakrishnan M,Saurabh S. *Med Hypotheses*2014 Dec. 83(6);816-8.

275. Red blood cell-derived nitric oxide bioactivity and hypoxic vasodilation.Schmidt H, FeelischM. *Circulation*2019 Jun 4;139(23):2664-2667.

276. Nitrite reduction to nitric oxide by deoxyhemoglobin vasodilated the human circulation.Cosby K, Partovi KS, Crawford JH, et al. *Nat Med*2003; 9(12):1498-505.

277. Active nitric oxide produced in the red cell under hypoxic conditions by deoxyhemoglobin-mediated nitrite reduction.Nagababu E, Ramasamy S, Abernethy DR, et al. *J Biol Chem*2003;278(47);46349-56.

278. Erythrocytes are major intravascular storage sites of nitrite in human blood.Dejam A,Hunter CJ, Pelletier MM, et al. *Blood*2005;106(2);734-9.

279. Erythrocytes: Oxygen sensors and modulators of vascular tone.Ellsworth ML, Ellis CG, Goldman D, et al. *Physiol (Bethesda)*2009; 24;107-16.

280. Inhibition of suicidal erythrocyte death by nitric oxide.Nicolay JP, Liebig G, Niemoeller OM, et al. *Pflugers Arch*2008;456(2):293-305.

281. Advances in hypoxia-inducible factor biology. Choudhry H, Harris AL. *CellMetab*2018;27(2):281-298.

282. The Nrf-2/HO-1 Signaling Axis: A Ray of Hope in Cardiovascular Diseases.Zhang X, Yu Y, LeiH, et al. *Cardiol Res Pract*2020Jan 30;2020:5695723.

283. An Overview of Nrf2 Signaling Pathway and Its Role in Inflammation.Saha S, Buttari B,Panieri E, et al. *Molecules*2020;25(22):5474.

284. Role of Nrf2/HO-1 system in development, oxidative stress response and diseases: an evolutionarily conserved mechanism.Loboda A, Damulewicz M, Pyza E, et al. *Cell Mol Life Sci*2016;73(17):3221-47.

285. Nrf2 activation is involved in ozonated human serum upregulation of HO-1 in endothelial cells.Pecorelli A, Bocci V, Acquaviva A, et al. *Toxicol Appl Pharmacol*2013; 267;30-40.

286. Is ozone pre-conditioning effect linked to Nrf2/EpRE activation pathway in vivo? A preliminary result. Re L, Martinez-Sanchez G, Bordicchia M, et al. *Eur J Pharmacol*2014;742:158-162

287. Nrf2/ARE regulated antioxidant gene expression in endothelial and smooth muscle cells in oxidative stress: implications for atherosclerosis and preeclampsia.Mann GE, Niehueser-Saran J, Watson A, Gao L, et al. *Sheng Li Xue Bao*2007 Apr;59(2):117-27

288. Nrf2 as a master redox switch in turning on the cellular signaling involved in the induction of cytoprotective genes by some chemopreventive phytochemicals.Surh YJ, Kundu JK, Na HK*Planta Med*2008 Oct.74(13);1526-39.

289. Hydroychloroquine: From malaria to autoimmunity.Ben-Zvi I, Kivity S, Langevitz P,Shoenfeld Y. *Clin*

*Rev Allergy Immunol*2012;42(2):145-53

290. Chloroquine analogues in drug discovery: New directions of uses, mechanisms of actions and toxic manifestations from malaria to multifarious diseases. Al-Bari MA. *J AntimicrobChemother* 2015;70(6):1608-21

291. In vitro inhibition of severe acute respiratory syndrome coronavirus by chloroquine.Keyaerts P, Vijgen L, Maes P. *BiochemBiophys Res Commun*2004;323(1):264-68

292. Chloroquine is a potent inhibitor of SARS coronavirus infection and spread.Vincent MJ,Bergeron E, Benjannet S, et al. *Virol J*2005;2:69

293. Repurposing of clinically developed drugs for treatment of Middle East respiratory syndrome coronavirus infection.Dyall J, Coleman CM, Hart BJ. *Antimicrob Agents Chemother*2014;58(8):4885-93

294. Current and future use of chloroquine and hydroxychloroquine in infectious, immune, neoplastic, and neurological diseases: A mini-review.Plantone D, Koudriavtseva T. *Clin DrugInvest*2018;38(8):653-71

295. Regulators split on antimalarials for COVID-19. Jaffe S. *Lancet*2020;395(10231):1179

296. COVID-19 pandemic: A narrative review of the potential roles of chloroquine and hydroxychloroquine.de Barros CM, Almeida CAF, Pereira B, et al. *Pain Physician*2020;23(4S):S351-S366

297. Hydroxychloroquine and chloroquine for COVID-19: no evidence for effectiveness. de Barros CM, Almeida CAF, Pereira B, et al. *Ned TijdschrGeneeskd*2020;164:D5141

298. Chloroquine or hydroxychloroquine for prevention and treatment of COVID-19.Singh B,Ryan H, Kredo T, et al. *Cochrane Database Syst Rev*2021;2(2):CD03587

299. A systematic review of the prophylactic role of chloroquine and hydroxychloroquine in coronavirus-19 disease (COVID-19).Shah S, Das S, Jain A, et al. *Int J Rheum Dis*2020; 23(5):613-19

300. An updated systematic review of the therapeutic role of hydroxychloroquine in coronavirus disease-19 (COVID-19).Das S, Bhowmick S, Tiwari S, et al. *Clin Drug Invest*2020;40(7):591-601

301. Effect of hydroxychloroquine with or without azithromycin on the mortality of coronavirus disease 2019(COVID-19 patients: a systematic review and meta-analysis.Fiolet T, Guihur A,Rebeaud ME, et al. *Clin Microbiol Infect*2021;27(1):19-27

302. Efficacy and safety of antiviral treatment for COVID-19 from evidence in studies of SARS-CoV-2 and other acute viral infections: A systematic review and meta-analysis.Liu W, Zhou P, Chen K, et al. *CMAJ*2020;192(27):E734-E744

303. Unfavorable hydroxychloroquine COVID-19 research associated with authors having a history of political donations.Berry AC, Gonnering RS, Rodriguez I, et al. *Cardiovasc Med*2021;22(1):191-98

304. Two elite medical journals retract coronavirus papers over data integrity questions.CharledPiller& Kelly Servick. *Science*June 4, 2020 https://www.science.org/content/article/two-elite-medical-journals-retract-coronavirus-papers-over-data-integrity-questions

305. Efficacy of early hydroxychloroquine treatment in preventing COVID-19 pneumonia aggravation, the experience from Shanghai, China.Su Y, Ling Y, Ma Y, et al. *Biosci Trends*2021;14(6):408-14

306. Outcomes of 3,737 COVID-19 patients treated with hydroxychloroquine/azithromycin and other regimens in Marseille, France: a retrospective analysis. Lagier JC, Million M, Gautret P, etal. *Travel Med Infect Dis*2020;36:101791

307. Comparing the impact of hydroxychloroquine-based regimens and standard treatment on COVID-19 patient outcomes: a retrospective cohort study. Almazrou SH, Almalki ZS, Alanazi ASet al. *Saudi Pharm J* 2020;28(12):1877-82

308. Early hydroxychloroquine but not chloroquine use reduces ICU admission in COVID-19 patients. Lammers AJJ, Brohet RM, Theunissen REP, et al. *Int J Infect Dis*2020;101:283-89

309. Early combination therapy with hydroxychloroquine and azithromycin reduces mortality in 10,429 COVID-19 outpatients.Million M, Lagier JC, Tissot-Dupont H, et al. *Cardiovasc Med*2021;22(3):1063-72

310. Role of hydroxychloroquine in multidrug treatment of COVID-19.McCullough PA, StrickerRB, Risch HA. *Rev Cardiovasc Med*2021;22(3):545-46

311. Randomized Controlled Trials of Early Ambulatory

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

Hydroxychloroquine in the Prevention of COVID-19 Infection, Hospitalization, and Death: Meta-Analysis.Ladapo JA, McKinnon JE, McCullough PA, Risch HA. *medRxiv*doi: https://doi.org/10.1101/2020.09.30.20204693

312. Early outpatient treatment of symptomatic, high-risk COVID-19 patients that should be ramped up immediately as key to the pandemic crisis. Risch HA. *Am J Epidemiol*2020;189(11):1218-26

313. Chloroquine and hydroxychloroquine in the treatment of COVID-19 with or without diabetes: A systematic search and a narrative review with a special reference to India and other developing countries. Singh AK, Singh A, Shaikh A. *DiabetMetabSynd*2020;14(3):241-46

314. Use of hydroxychloroquine in hospitalized COVID-19 patients is associated with reduced mortality: Findings from the observational multicenter Italian CORIST study.COVID-19 RISK and Treatments (CORIST) Collaboration. *Eur J Intern Med*2020;82:38-47

315. Treatment with hydroxychloroquine, azithromycin, and combination in patients hospitalized with COVID-19.Arshad S, Kilgore P, Chaudhry ZS, et al. *Int J Infect Dis*2020;97:396-403

316. Low-dose hydroxychloroquine therapy and mortality in hospitalised patients with COVID-19: a nationwide observational study of 8075 participants.Catteau L, Dauby N, Montourcy M, et al. *Int J Antimicrob Agents*2020;56(4):106144

317. COVID-19 outpatients: Early risk-stratified treatment with zinc plus low-dosehydroxychloroquine and azithromycin: A retrospective case series study. Derwand R, Scholz M,Zelenko V. *Int J Antimicrob Agents*2020;56(6):106214

318. Correlation of the rise and fall in COVID-19 cases with the social isolation index and early outpatient treatment with hydroxychloroquine and chloroquine in the state of Santa Catarina, southern Brazil: A retrospective analysis.Neves FS. *Travel Med Infect Dis*2021;41:102005

319. Current and Future use of chloroquine and hydrochloroquine in infectious, immune, neoplastic and neurological diseases: A mini-review. Plantane D, Koudriavtseva. *Clin Drug Invest*2018;38(8):653-71

320. Hydroxychloroquine in rheumatic autoimmune disorders and beyond.Nirk EL, Reggiori F,Mauthe M. *EMBO Mol Med* 2020;12(8):e12476

321.Therapy and pharmacological properties of hydroxychloroquine and chloroquine in treatment of systemic lupus erythematosus, rheumatoid arthritis and related diseases.Rainsford KD, Parke AL, Clifford-Rashotte M, Kean WF. *Inflammopharmacology.* 2015Oct;23(5):231-69

322. In vivo chloroquine-induced inhibition of insulin degradation in a diabetic patient with severe insulin resistance.Blazar BR, Whitley CB, Kitabchi AE, Tsai MY, et al. *Diabetes*1984Dec;33(12):1133-7

323. Hydroxychloroquine in decompensated, treatment-refractory noninsulin-dependent diabetes mellitus. A new job for an old drug?Quatraro A, Consoli G, Magno M, Caretta F. *AnnIntern Med*1990;112(9):678-81

324. Potential Effect of Hydroxychloroquine in Diabetes Mellitus: A Systematic Review on Preclinical and Clinical Trial Studies.Wondafrash DZ, Desalegn TZ, Yimer EM, et al. *J DiabetesRes*2020 Feb 27;2020:5214751.

325. Cholesterol-lowering effect of hydroxychloroquine in patients with rheumatic disease: Reversal of deleterious effects of steroids on lipids.Wallace DJ, Metzger AL, Stecher VJ, et al. *Am J Med*1990 Sep;89(3):322-6.

326. Hydroxychloroquine reduces low-density lipoprotein cholesterol levels in systemic lupus erythematosus: A longitudinal evaluation of the lipid-lowering effect.Cairoli E, Rebella M,Danese N, Garra V, et al. *Lupus*2012 Oct;21(11):1178-82.

327. Hydroxychloroquine use associated with improvement in lipid profiles in rheumatoid arthritis patients.Morris SJ, Wasko MC, Antohe JL, Sartorius JA. *Arthritis Care Res (Hoboken)*2011 Apr;63(4):530-4

328. The pharmacologic mechanisms and therapeutic activities of hydroxychloroquine in rheumatic and related diseases.Hu C, Lu L, Wan JP, et al. *Curr Med Chem* 2017;24(20):2241-49

329. Chloroquine inhibits stimulated platelets at the arachidonic acid pathway.Nosál R,Jancinová V, Petriková M. *Thromb Res.* 1995 Mar 15;77(6):531-42.

330. Thrombosis and systemic lupus erythematosus: The Hopkins Lupus Cohort perspective.Petri M. *Scand*

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

*J Rheumatol*1996;25(4):191-3

331. Hydroxychloroquine reverses platelet activation induced by human IgG antiphospholipid antibodies. Espinola RG, Pierangeli SS, Gharavi AE, Harris EN. *ThrombHaemostas*2002;87(3):518-22

332. The protective effect of antimalarial drugs on thrombovascular events in systemic lupus erythematosus.Jung H, Raja B, Su J, Shariati-Sarabi Z, et al. *Arthritis Rheum*2010 Mar;62(3):863-8.

333. Antimalarials.Koranda FC. *J Am Acad Dermatol*1981 Jun;4(6):650-5.

334. Mode of action of hydroxychloroquinone in rheumatoid arthritis: Evidence of an inhibitory effect on toll-like receptor signaling.Kyburz D, Brentano F, Gay S.*Nat Clin PractRheumatol*2006

335. Chloroquine autophagic inhibition rebalances Th17/Treg-mediated immunity and ameliorates systemic lupus erythematosis.An N, Chen Y, Wang C, Wu ZH, Xue J, et al. *CellPhysiolBiochem* 2017

336. Chloroquine & Hydroxychloroquine equally affect tumor necrosis factor-a, interleukin 6 & interferon-g by peripheral blood mononuclear cells.Van den Borne BEEM, Dijkmans BAC, DeRooij HH. *J Rheumatol*1997

337. Chloroquine inhibits processing of tumor necrosis factor in lipopolysaccharide-stimulated RAW 264.7 macrophages.Jeong JY, Jue DM. *J Immunol*1997 May 15;158(10):4901-7.

338. Chloroquine inhibits production of TNF-alpha, IL-1beta and IL-6 from lipopolysaccharide-stimulated human monocytes/macrophages by different modes. Jang C-H, Choi J-H, Byun M-S, Jue D-M. *Rheumatology (Oxford)*2006 Jun;45(6):703-10.

339. Killing of Escherichia coli by Crohn's Disease Monocyte-derived Macrophages and Its Enhancement by Hydroxychloroquine and Vitamin D.Flanagan PK, Chiewchengchol D, Wright H, Edwards SW. *Inflamm Bowel Dis*2015 Jul;21(7):1499-510.

340. Chloroquine inhibits tumor necrosis factor production by human macrophages in vitro. Picot S, Peyron F, Vuillez JP, Polack B, Ambroise-Thomas P. *J Infect Dis*1991 164: 830

341. Chloroquine and hydroxychloroquine equally affect tumor necrosisfactor-a, interleukin 6, and interferon-c production by peripheral bloodmononuclear cells.Van Den Borne BEEM, Dijkmans BAC, De Rooij HH, Le Cessie S, Verweij CL. *J Rheumatol*1997; 24: 55 – 60

342. Mechanism of action of antimalarial drugs: inhibition of antigen processing and presentation.Fox RI, Kang HI. *Lupus*1993 Feb;2 Suppl 1:S9-12.

343. Mechanisms of action of hydroxychloroquine and chloroquine: implications for rheumatology. Schrezenmeier E, Dörner T. *Nat Rev Rheumatol*2020 Mar;16(3):155-166.

344. Vitamin D status is associated with in-hospital mortality and mechanical ventilation: a cohort of COVID-19 hospitalized patients.Mahamat-Saleh Y, Fiolet T, Rebeaud ME, et al. *Mayo Clin Proc* 2021; 96(4):875-86

345. The relationship between the severity and mortality of SARS-CoV-2 infection and 25-hydroxyvitamin D concentration—a metaanalysis. Oscanoa TJ, Amado J, Vidal X, et al. *Adv Respir Med* 2021;89(2):145-57

346. Vitamin D status in hospitalized patients with SARS-CoV-2 infection.Hernández JL, Nan D, Fernandez-Ayala M, et al. *J Clin Endocrinol Metab*2021;106(3):e1343-e1353

347. Evaluation of vitamin-D status and its association with clinical outcomes among COVID-19 patients in Pakistan.Asghar MS, Yasmin F, Dapke K, et al. *Am J Trop Med*2021;106(1):150-55

348. Prevalence of clinical outcomes of vitamin D deficiency in COVID-19 hospitalized patients: A retrospective single-center analysis.Afaghi S, EsmaeiliTarki F, et al. *Tohoku J Exp Med 2021*;255(2):127-34

349. Vitamin D and COVID-19 severity and related mortality: a prospective study in Italy.Campi I, Gennari L, Merlotti D, et al. *BMC Infect Dis*2021;21(1):566

350. Vitamin D deficiency is associated with higher hospitalization risk from COVID-19: A retrospective case-control study.Jude EB, Ling SF, Allcock R, et al. *J Clin Endocrinol Metab*2021;106(11):e4708-e4715

351. The relationship between 25(OH) vitamin D levels and COVID-19 onset and disease course in Spanish patients.Diaz-Curiel M, Cabello A, Arboiro-Pinel R, et

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

al. *J Steroid Biochem Mol Biol*2021;212:105928

352. Association of vitamin D status with SARS-CoV-2 infection or COVID-19 severity: A systematic review and meta-analysis.Kazemi A, Mohammadi V, Aghababaee SK, et al. *Adv Nutr*2021;12(5):1636-58

353. Potential benefit of vitamin D supplementation in people with respiratory illnesses, during the COVID-19 pandemic.Chetty VV, Chetty M. *Clin Transl Sci*2021;14(6):2111-16

354. Serum level of Vitamin D is associated with COVID-19 mortality rate in hospitalized patients.Ranjbar M, KarbalaieNiya MH, et al. *J Res Med Sci*2021;26:112

355. Vitamin D status and SARS-CoV-2 infection and COVID-19 clinical outcomes.Chiodini I, Gatti D, Soranna D, et al.*Front Pub Health*2021;9:736665

356. Vitamin D supplementation and COVID-19 risk: a population-based, cohort study.Oristrell J, Oliva JC, Casado, et al. *Endocrinol Invest*2022;45(1):167-79

357. Calcifediol treatment and COVID-19-related outcomes.Nog X, Ovejero D, Pineda-Moncusí M, et al. *J Clin Endocrinol Metab*2021;106(10):e4107-e4027

358. COVID-19 and vitamin D (Co-VIVID study): A systemic review and meta-analysis of randomized controlled trials.Varikasuvu SR, Thangappazham B, Vykunta A, et al. *Expert Rev Anti Infect Ther*2022 Feb 3:1-7

359. Effect of calcifediol treatment and best available therapy versus best available therapy on intensive care unit admission and mortality among patients hospitalized for COVID-19: A pilot randomized clinical study. Entrenas Castillo M, Entrenas Costa LM, Vaquero Barrios JM, et al. *J Steroid BiochemBiol 2020*;203:105751

360. Seasonality and autoimmune diseases: The contribution of the four seasons to the mosaic of autoimmunity.Watad A, Azrielant S, Bragazzi NL, Sharif K, et al. *J Autoimmun*2017Aug;82:13-30.

361. Association between seasonal factors and multiple sclerosis.Watad A, Azrielant S, SorianoA, Bracco D, et al. *Eur J Epidemiol*2016 Nov;31(11):1081-1089.

362. Sun exposure over the life course and associations with multiple sclerosis.Tremlett H, Zhu F, Ascherio A, Munger KL. *Neurology*2018 Apr 3;90(14):e1191-e1199.

363. Seasonality of tuberculosis in the United States, 1993-2008.Willis MD, Winston CA, Heilig CM, Cain KP, Walter ND, Mac Kenzie WR.*Clin Infect Dis*2012 Jun;54(11):1553-60

364. Seasonality of tuberculosis in Israel, 2001-2011. Margalit I, Block C, MorZ.*Int J Tuberc Lung Dis*2016 Dec 1;20(12):1588-1593

365. *Influenza: The Last Great Plague.* W.I.B. Beveridge publ.Prodist, NY1978

366. Global breast cancer seasonality.Oh E-Y, Ansell C, Nawaz H, Yang C-H, Wood P, Hrushesky. WJM*Breast Cancer Res Treat*2010 Aug;123(1):233-43.

367. Impact of season of diagnosis on mortality among breast cancer survivors.Kuzmickiene I, Atkocius V, Aleknavicius E, Ostapenko V. *J Cancer Res Ther*2018 Dec;14(Supplement):S1091-S1097.

368. Changes in risk of death from breast cancer with season and latitude: sun exposure and breast cancer survival in Norway.Poroinicu AC, Lagunova Z, Robsahm TE, Berg JP, Dahlback A, Moan J. *Breast Cancer Res Treat*2007 May;102(3):323-8.

369. Vitamin D and autoimmune diseases.Illescas-Montes R, Melguizo-Rodríguez L,Ruiz C, Costela-Ruiz VJ. *Life Sci*2019 Sep 15;233:116744.

370. Seasonal variations of 25-OH vitamin D serum levels are associated with clinical disease activity in multiple sclerosis patients.Hartl C, Obermeier V, Gerdes LA, Brügel M, et al. J Neurol Sci2017 Apr 15;375:160-164.

371. Association of seasonal serum 25-hydroxyvitamin D levels with disability and relapses in relapsing-remitting multiple sclerosis.Brola W, Sobolewski P, Szczuchniak W, Góral A, et al. *Eur J Clin Nutr*2016 Sep;70(9):995-9.

372. Hypovitaminosis D association with disease activity in relapsing remitting multiple sclerosis in Brazil. Becker J, Callegaro D, Lana-Peixoto MA, Talim N, et al. *J Neurol Sci*2016 Apr15;363:236-9.

373. 25-Hydroxyvitamin D levels in serum at the onset of multiple sclerosis.Soilu-Hänninen M, Mononen AI, Heikilä A, Viljanen M, Hänninen A. *MultScler*2005 Jun;11(3):266-71.

374. A low vitamin D status at diagnosis is associated with an early conversion to secondary progressive multiple sclerosis.Muris A-H, Rolf L, Broen K, Hupperts R,

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

et al. *J Steroid BiochemMol Biol*2016 Nov;164:254-257.

375. Environmental risk factors for multiple sclerosis: a review with a focus on molecular mechanisms.O'Gorman C, Lucas R, Taylor B. *Int J Mol Sci*2012;13(9):11718-52.

376. Low 25 (OH) vitamin D levels are associated with autoimmune thyroid disease in polycystic ovary syndrome.Muscogiuri G, Palomba S, Caggiono M*Endocrine*2016 Aug;53(2):538-42.

377. 25 Hydroxyvitamin D Deficiency and Its Relationship to Autoimmune Thyroid Disease in the Elderly.Muscogiuri G, Mari D, Prolo S, Fatti LM, et al. *Int J Environ Res Public Health*2016 Aug 26;13(9):850.

378. Vitamin D and Autoimmune Thyroid Disease-Cause, Consequence, or a Vicious Cycle?Vieira IH, Rodrigues D, Paiva I. *Nutrients*2020 Sep 11;12(9):2791.

379. Meta-analysis of the association between vitamin D and autoimmune thyroid disease.Wang J, Ly S, Chen G, Gao C, et al. *Nutrients*2015 Apr 3;7(4):2485-98.

380. Vitamin D and systemic lupus erythematous: A review.*Clin Exp Rheumatol*Jan-Feb 2018;36(1):153-162.

381. Vitamin D in lupus - new kid on the block? Kammen DL. *Bull NYU Hosp Jt Dis*2010;68(3):218-22.

382. Vitamin D and systemic lupus erythematosus: State of the art.Schneider L, Dos Santos ASP, Santos M, da Silva RM, et al. *Clin Rheumatol*2014 Aug;33(8):1033-8.

383. Vitamin D level in rheumatoid arthritis and its correlation with the disease activity: A meta-analysis.Lee Y-H, Bae S-C. *Clin Exp Rheumatol*Sep-Oct 2016;34(5):827-833.

384. Prevalence of vitamin D deficiency in rheumatoid arthritis and association with disease activity and cardiovascular risk factors: data from the COMEDRA study. Cechitti S, Tatar Z, Galan P, Pereira B, et al. *Clin Exp Rheumatol*Nov-Dec 2016;34(6):984-990.

385. European multicentre pilot survey to assess vitamin D status in rheumatoid arthritispatients and early development of a new Patient Reported Outcome questionnaire (D-PRO).Vojinovic J, Tincani A, Sulli A, Soldano S, et al. *Autoimmun Rev*2017 May;16(5):548-554.

386. Vitamin D and Sjögren syndrome.Garcia-Carrasco M, Jiménez-Herrera EA, Gálvez-RomeroJL, et al. *Autoimmun Rev*2017 Jun;16(6):587-593.

387. Vitamin D treatment for connective tissue diseases: Hope beyond the hype?Reynolds JA,Bruce IN. *Rheumatology (Oxford)*2017 Feb;56(2):178-186

388. Vitamin D and systemic lupus erythematosus - The hype and the hope. Shoenfeld Y, Giacomelli R, Azrielant S, Berardicurti O, et al. *Autoimmun Rev*2018 Jan;17(1):19-23.

389. Severe vitamin D deficiency is a prognostic biomarker in autoimmune hepatitis.Ebadi M,Bhanji RA, Mazurak VC, Lytvyak E, et al. *Aliment PharmacolTher*2019 Jan;49(2):173-182.

390. Vitamin D deficiency and diabetes.Berridge M. *Biochem J*2017 Mar 24;474(8):1321-1332.

391. Vitamin D and diabetes.Takiishi T, Gysemans C, Bouillon R, Mathieu C. *Endocrinol Metab Clin North Am*2010 Jun;39(2):419-46

392. The relationship between vitamin D level and organspecific autoimmune disorders in newly diagnosed type I diabetes mellitus.Akdere G, Efe B, Sisman P, Yorulmaz G. *BratislLekListy*2018;119(9):544-549.

393. Low levels of 25-hydroxyvitamin D in children and adolescents with type 1 diabetesmellitus: A single center experience.Bae KN, Nam H-K, Rhie Y-J, Song DJ, et al. *Ann Pediatr Endocrinol Metab*2018 Mar;23(1):21-27.

394. Vitamin D and inflammatory bowel disease.Ardesia M, Ferlazzo G, Fries W. *Biomed Res Int*2015;2015:470805.

395. Vitamin D and Crohn's disease in the adult patient: A review. Basson A. *JPEN J ParenterEnteral Nutr*2014 May;38(4):438-58.

396. Vitamin D deficiency and the pathogenesis of Crohn's disease.White JH. *J Steroid BiochemMol Biol*2018 Jan;175:23-28.

397. Does vitamin D play a role in autoimmune endocrine disorders? A proof of concept.AltieriB, Muscgiuri G, Barrea L, Mathieu C, et al. *Rev EndocrMetabDisord*2017 Sep;18(3):335-346.

398. Vitamin D endocrine system involvement in autoimmune rheumatic diseases.Cutolo M,Pizzorni C,

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

Sulli A. *Autoimmun Rev*2011 Dec;11(2):84-7.

399. Vitamin D and inflammation.CannellJJ, Grant WB, Holick MF. *Dermatoendocrinol*2015Jan 29;6(1):e983401.

400. Vitamin D for the management of multiple sclerosis.Jagannath VA, Filippini G, DiPietrantoni C, Asokan GV, et al. *Cochrane Database Syst Rev*2018 Sep 24;9(9):CD008422.

401. Vitamin D for the management of multiple sclerosis.Jagannath VA, Fedorowicz Z, Asokan GV Robak EW, Whamond L. *Cochrane Database Syst Rev*2010 Dec 8;(12):CD008422

402. Seasonal variation of serum vitamin D levels in Romania.Niculescu DA, CapatinaCAM, Dusceac R, Caragheorgheopol A, Ghemigian A, Poiana C.*Arch Osteoporos* 2017 Dec 11;12(1):113.

403. Circannual versus seasonal variations of longitudinally sampled 25-hydroxycholecalciferol serum levels.Cugini P, Coen G, Scavo D, Lucia P, Mazzaferro S, BianchiniG, MassimettiC, Donato G.*Biochem Med* 1984 Aug;32(1):22-9.

404. Biological Effects of Sunlight, Ultraviolet Radiation, Visible Light, Infrared Radiation andVitamin D for Health.Holick MF. *Anticancer Res*2016 Mar;36(3):1345-56.

405. Ultraviolet B Radiation: The Vitamin D Connection.Holick MF. *Adv Exp Med Biol*2017;996:137-154.

406. Influence of season and latitude on the cutaneous synthesis of vitamin D3: exposure to winter sunlight in Boston and Edmonton will not promote vitamin D3 synthesis in human skin.Webb AR, Kline L, Holick MF.*J Clin Endocrinol Metab*1988 Aug;67(2):373-8.

407. Environmental factors that influence the cutaneous production of vitamin D.Holick MF*Am J Clin Nutr.* 1995 Mar;61(3 Suppl):638S-645S.

408. Global Overview of Vitamin D Status.van Schoor N, Lips P.*Endocrinol Metab Clin North Am* Dec;46(4):845-870

409. Vitamin D deficiency and insufficiency among US adults: Prevalence, predictors and clinical implications.Liu X, Baylin A, Levy PD. *Br J Nutr* 2018 Apr;119(8):928-936.

410. Vitamin D deficiency in urban Massachusetts newborns and their mothers.MerewoodA, Mehta SD, Grossman X, Chen TC, Mathieu JS, Holick MF, Bauchner H.*Pediatrics*2010Apr;125(4):640-7

411. Extremely High Prevalence of Maternal and Neonatal Vitamin D Deficiency in the Arab Population. Fouda MA, Turkestani IZ, Almusharraf S, Al-Ajlan A, Angkaya-Bagayawa FF, Sabico S, Mohammed AG, Hassanato R, Al-Serehi A, Alshingetti NM, Al-Daghri NM.2017;112(3):225-230

412. Sunlight and Vitamin D: A global perspective for health.Wacker M, Holick MF*Dermatoendocrinol*2013 Jan 1;5(1):51-108.

413. Vitamin D Receptor: New Assignments for an Already Busy Receptor.Norman AW.*Endocrinology*2006; vol 147: pp. 5542-5548

414. Vitamin D Status and severe COVID-19 disease outcomes in hospitalized patients.Pecina JL, Merry SP, Park JG, et al. *Prim Care CommunHealth* 2021;12:21501327211041206

415. Impact of vitamin D status on COVID-19 severity among hospitalized patients in the western region of Saudi Arabia: a retrospective cross-sectional study. Bushnaq T, Algethami F, Qadhi A, et al. *Int J Environ Res Pub Health 2022*;19(3):1901

416. Vitamin D supplementation for the treatment of COVID-19: A living systematic review. Stroehlein JK, Wallqvist J, Iannizzi C, et al. *Cochrane Database Syst Rev 2021*;5(5):CD015043

417. Exploring the link between vitamin D and clinical outcomes in COVID-19.Lohia P, Nguyen P, Patel N, Kapur S. *Am J Physiol Endocrinol Metab*2021;320(3):E520--E526

418. Vitamin-D levels and intensive care unit outcomes in a cohort of critically ill COVID-19 patients.Orchard L, Baldry M, Nasim-Mohi M, et al. *Clin Chem Lab Med* 2021;59(6):1155-63

419. A single-oral bolus of 100,000 IU of cholecalciferol at hospital admission did not improve outcomes in the COVID-19 disease: The COVID-VIT-D randomized multicenter international clinical trial.Cannata-Andía JB, Díaz-Sottolano A, Fernández P, et al. *BMC Med*2022;20(1):83

420. Sun exposure induces rapid immunological changes in skin and peripheral blood in patients

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

with psoriasis. Søyland E, Heier I, Rodríguez-Gallego C, Mollnes TE, Johansen FE, Holven KB, Halvorsen B, Aukrust P, Jahnsen FL, de la Rosa Carrillo D, Krogstad AL, Nenseter MS.*Br JDermatol*Feb;164(2):344-55

421. Sun exposure rapidly reduces plasmacytoid dendritic cells and inflammatory dermal dendritic cells in psoriatic skin. Heier I, Søyland E, Krogstad AL, Rodríguez-Gallego C, Nenseter MS, Jahnsen FL.*Br J Dermatol*Oct;165(4):792-801

422. Effects of ultraviolet light on human serum 25-hydroxyvitamin D and systemic immune function.Milliken SV, Wassall H, Lewis BJ, Logie J, Barker RN, MacdonaldH, VickersMA, Ormerod AD.*J Allergy Clin Immunol*2012 Jun;129(6):1554-61

423. *The Fourth Phase of Water: Beyond Solid, Liquid and Vapor.* Gerald H. Pollackpubl Ebner & Sons, Seattle2013

424. Water structure and interactions with protein surfaces.Raschke TM. CurrOpin Struct Biol 2006 Apr;16(2):152-9

425. Water Determines the Structure and Dynamics of Proteins.Bellissent-Funel MC, Hassanali A, Havenith M, Henchman R, Pohl P, Sterpone F, van der Spoel D, Xu Y, Garcia AE.Chem Rev2016 Jul 13;116(13):7673-97

426. Water mediation in protein folding and molecular recognition.Levy Y, Onuchic JN.Annu Rev Biophys-Biomol Struct2006;35:389-415.

427. Dynamics of hydration water in proteins.Teixera J. *Gen PhysiolBiophys*2009;28(2):168-73.

428. Sub-terahertz spectroscopy reveals that proteins influence the properties of water at greater distances than previously detected.Sushko O, Dubrovka R, Donnan RS.J Chem Phys2015 Feb 7;142(5):05510

429. The role of water in amyloid aggregation kinetics. Stephens AD, Kaminski Schierle GS.CurrOpin Struct Biol. 2019 Oct;58:115-123

430. Local Structure and Dynamics of Hydration Water in Intrinsically Disordered Proteins. Rani P, Biswas P.J Phys Chem B2015 Aug 27;119(34):10858-67

431. Interaction with the surrounding water plays a key role in determining the aggregation propensity of proteins.Chong SH, Ham S.Angew Chem Int Ed Engl.2014 Apr 7;53(15):3961-4

432. Protein structural and surface water rearrangement constitute major events in the earliest aggregation stages of tau.Pavlova A, Cheng CY, Kinnebrew M, Lew J, Dahlquist FW, Han S.*ProcNatl Sci USA*2016 Jan 12;113(2):E127-36

433. Role of water in protein aggregation and amyloid polymorphism.Thirumalai D, ReddyG, Straub JE. *Acc Chem Res*2012 Jan 17;45(1):83-92

434. The activation of the cytochrome P-450 dependent monooxygenase system by light.Müller-Enoch D, Gruler H.*Z Naturforsch C*1986 May-Jun;41(5-6):604-12

435. Light-induced activation and synchronization of the cytochrome P-450 dependent monooxygenase system. Häberle W, Gruler H, Dutkowski P, Müller-Enoch D. *Z Naturforsch C*1990 Mar-Apr;45(3-4):273-9

436. Light-driven biocatalysis with cytochrome P450 peroxygenases.Girhard M, KunigkE, Tihovsky S, Shumyantseva VV, Urlacher VB. *Biotechnol Appl Biochem*2013 Jan-Feb; 60(1): 111-18

437. Slaving the cytochrome P-450 dependent monooxygenase system by periodically applied light pulses.Gruler H, Müller-Enoch D.*Eur Biophys J*1991; 19(4): 217-19

438. Extrarenal expression of 25-hydroxyvitamin d(3)-1 alpha-hydroxylase.Zehnder D, Bland R, Williams MC, McNinch RW, et al. *J Clin Endocrinol Metab*2001 Feb;86(2):888-94.

439. Extrarenal expression of the 25-hydroxyvitamin D-1-hydroxylase.Adams JS, Hewison M *Arch BiochemBiophys*2012 Jul 1;523(1):95-102.

440. Expression of 25-hydroxyvitamin D-1alpha-hydroxylase (1alphaOHase, CYP27B1) splice variants in HaCaT keratinocytes and other skin cells: Modulation by culture conditions and UV-B treatment in vitro. Seifert M, Tilgen W, Reichrath J. *Anticancer Res*2009 Sep;29(9):3659-67.

441. Vitamin D regulation of immune function during COVID-19.Bikle DD. *Rev EndocrMetabDisord*2022 Jan 29:1-7

442. Respiratory epithelial cells convert inactive vitamin D to its active form: Potential effects on host defense.Hansdottir S, Monick MM, Hinde SL, et al. *J Immunol* 2008;181(10):7090-99

443. Vitamin D increases the antiviral activity of bronchial epithelial cells in vitro.Telcian AG,Zdrenghea MT, Edwards MR, et al. *Antiviral Res*2017;137:93-101

444. Sinonasal epithelial cells synthesize active vitamin D, augmenting host innate immune function.Sultan B, Ramanathan M Jr, Lee J, et al. *Int Forum Allergy Rhinol*2013;3(1):26-30

445. New insights into upper airway innate immunity.Hariri BM, Cohen NA. *Am J Rhinol Allergy*2016;30(5):319-23

446. Modulation of the immune response to respiratory viruses by vitamin D.Greiller CL,Martineau AR. *Nutrients* 2015;7(6):4240-70

447. Vitamin D effects on lung immunity and respiratory diseases.Hansdottir S, Monick MM. *Vitamin Horm*2011;86:217-37

448. Emerging roles of vitamin D-induced antimicrobial peptides in antiviral innate immunity.White JH. *Nutrients*2022;14(2):284

449. Vitamin D and COVID-19 severity and related mortality: A prospective study in Italy.CampiI, Gennari L, Merlotti D, et al. *BMC Infect Dis*2021;21(1):566

450. Analysis of serum cytokine and protective vitamin D levels in severe cases of COVID-19. Bayraktar N, Turan H, Bayraktar M, et al. *J Med Virol*2022;94(1):154-60

451. The association between TNF-a, IL-6, and vitamin D levels and COVID-19 severity and mortality: a systematic review and meta-analysis.Halim C, Mirza AF, Sari MI. *Pathogens* 2022;11(2):195

452. Cholecalciferol level and its impact on COVID-19 patients.Saeed MAM, Mohamed AH,Owaynat AH. *Egypt J Intern Med*2022;34(1):23

453. Effects of vitamin D on macrophages and myeloid-derived suppressor cells (MDSCs)hyperinflammatory response in the lungs of COVID-19 patients. Kloc M, Ghobrial RM, Lipińska-Opałka A, et al. *Cell Immunol*2021;360:104259

454. The role of macrophages in the pathogenesis of SARS-CoV-2-associated acute respiratory distress syndrome.Kosyreva A, Dzhalilova D, Lokhonina A, et al. *Front Immunol*2021;12:682871

455. Vitamin D receptor stimulation to reduce acute respiratory distress syndrome (ARDS) in patients with coronavirus SARS-CoV-2 infections: Revised Ms SBMB 2020_166.Quesada-Gomez JM, Entrenas-Castillo M, Bouillon R. *J Steroid Biochem Mol Biol*2020;202:105719

456. Rays of immunity: Role of sunshine vitamin in management of COVID-19 infection and associated comorbidities.Udaya Kumar V, Pavan G, Murti K, et al. *Clin NutrESPEN*2021;46:21-32

457. Cytokine storm modulation in COVID-19: A proposed role for vitamin D and DPP-4 inhibitorcombination therapy (VIDPP-4).Pinheiro MM, Fabbri A, Infante M. *Immunotherapy*2021;13(9):753-65

458. Vitamin D supplementation for the prevention and treatment of COVID-19: a position statement from the Spanish Society of Geriatrics and Gerontology.Tarazona-Santabalbina FJ,Cuadra L, CancioJM, et al.*Rev EspGeriatrGenomtol*2021; 56(3):177-82

459. World Health Organization Coronavirus (COVID-19) Dashboardhttps://covid19.who.int/

460. COVID-19: Clinical features. McIntosh K. *UpToDate* https://www.uptodate.com/contents/covid-19-clinical-features

461. Estimates of severity of coronavirus disease 2019: A model-based analysis.Verity R, Okell LC, DorigattiI. *Lancet Infect Dis*2020;20(6):669-77

462. Epidemiological and clinical characteristics of coronavirus disease (COVID-19) cases at a screening clinic during the early outbreak period: A single centre study.Khan M, Khan H, Khan S, et al. *J Med Microbiol*2020;69(8):1114-1123

463. Global percentage of asymptomatic SARS-CoV-2 infections among the tested population of individuals with confirmed COVID-19 diagnosis: a systematic review and meta-analysis.Ma Q,Liu J, Liu Q, et al. *JAMA Netw Open*2021;4(12):e2137257

464. COVID-19 Treatment Guidelines: Clinical Spectrum of SARS-CoV-2 Infection.https://www.covid19treatmentguidelines.nih.gov/overview/clinical-spectrum/

465. Global, regional, and national estimates of the population at increased risk of severe COVID-19 due

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

to underlying health conditions in 2020: A modelling study. Clark A, Jit M,Warren-Gash C, et al. *Lancet Glob Health*2020;8(8):e1003-e1017

466. COVID-19 Hospitalizations.State Health Facts.

https://www.kff.org/other/state-indicator/covid-19 hospitalizations/?currentTimeframe=0&sortModel=%7B%22colId%22:%22Location%22,%22sort%22:%22asc%22%7D

467. Average charge for COVID-19 hospitalization, by state.Paavola A *Hospital CFO Report*October 20, 2021. https://www.beckershospitalreview.com/finance/average-charge-for-covid-19-hospitalization-by-state.html

468. Trends in ICU Mortality from Coronavirus Disease 2019: A Tale of Three Surges.Auld SC,Harrington KRV, Adelman MW, et al. *Crit Care Med* 2022;50(2):245-55

469. ICU outcomes and survival in patients with severe COVID-19 in the largest health caresystem in central Florida.Oliveira E, Parikh A, Lopez-Ruiz A, et al. *PLoS*2021;16(3):e0249038

470. Mortality comparison between the first and second/third waves among 3,795 critical COVID-19 patients with pneumonia admitted to the ICU: A multicentre retrospective cohort study.Carbonell R, Urgelés S, Rodriguez A, et al. *Lancet Reg Health Eur*2021;11:100243

471. Mortality in patients admitted to intensive care with COVID-19: An updated systematic review and meta-analysis of observational studies.Armstrong RA, Kane AD, Kursumovic E, et al. *Anesthesia*2021;76(4):537-48

472. Global impact of coronavirus disease 2019 infection requiring admission to the ICU: A systematic review and meta-analysis.Tan E, Song J, Deane AM, et al. *Chest*2021;159(2):524-36

473. *The Real Anthony Fauci.* Robert F. Kennedy Jr.Skyhorse Publishing2021.

474. Breakthrough: Chloroquine phosphate has shown apparent efficacy in treatment ofCOVID-19 associated pneumonia.Gao J, Tian Z, Yang X. *Biosci Trends*2020;14(1):72-73

475. Diagnosis and Treatment Protocol for Novel Coronavirus Pneumonia (Trial Version 7).National Health Commission & National Administration of Traditional Chinese Medicine.Ed.Wei P-F. *Chin Med J (Engl)*2020;133(9):1087-95

476. The pathophysiologic basis and clinical rationale for early ambulatory treatment of COVID-19. McCullough PA, Kelly RJ, Ruocco G, et al. Am *J Med 134*(1):16-22

477. Multifaceted highly targeted sequential multidrug treatment of early ambulatory high-risk SARS-CoV-2 infection (COVID-19).McCullough PA, Alexander PE, Armstrong R, et al. *Rev Cardiovasc Med 2020*; 21(4):517-530

478. *The Real Anthony Fauci* Robert F. Kennedy Jr.Skyhorse Publishing 2021, pg. 8

479. Ibid, pp. 8-9

480. Ibid, pg. 17

481. Ibid, pp. 1-29

482. Controversial Doc No Longer with One of Idaho's Largest Health Networks.AmandaD'Ambrosio. *MedPage Today* December 10, 2021

https://www.medpagetoday.com/special-reports/exclusives/96143

483. World Health Organization Coronavirus (COVID-19) Dashboard

484. COVID-19: Should vaccine trials be unblinded?Lenzer J. BMJ 2020;371 https://www.bmj.com/content/371/bmj.m4956

485. The Price of Success—How to Evaluate COVID-19 Vaccines When They're Available Outside Clinical Trials.Rita Rubin. *JAMA Medical News and Perspectives.* February 18, 2021 https://jamanetwork.com/journals/jama/fullarticle/2776787

486. Covid-19: Researcher blows the whistle on data integrity issues in Pfizer's vaccine trial. Thacker PD. B*MJ*November 2, 2021; 375 doi: https://doi.org/10.1136/bmj.n2635

487. Dr. Nagase Talks on FOIA of Pfizer's COVID-19 Vaccine Documents, Adverse Events, and More. Download the documents at this website.

https://centipedenation.com/first-column/dr-nagase-

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302

talks-on-foia-of-pfizers-covid-19-vaccine-adverse-events-and-more/

488. Thorp JA, Renz T, Northrup C, Lively C, Breggin P, Bartlett R, et al. Patient Betrayal: The Corruption of Healthcare, Informed Consent and the Physician-Patient Relationship. The Gazette of Medical Sciences.2022;3(1):046-069. https://www.doi.org/10.46766/thegms.medethics.22021403

489. Internal CDC document on breakthrough infections.July 29,2021

https://www.washingtonpost.com/context/cdc-breakthrough-infections/94390e3a-5e45-44a5-ac40-2744e4e25f2e/

490. Increases in COVID-19 are unrelated to levels of vaccination across 68 countries and 2947 counties in the United States.https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8481107/

491. Unvaccinated COVID-19 hospitalizations cost billions of dollars.Amin K. Cox. *Health System Tracker.* December 22, 2021 https://www.healthsystemtracker.org/brief/unvaccinated-covid-patients-cost-the-u-s-health-system-billions-of-dollars/

492. Covid-19: politicisation, "corruption," and suppression of science. Editorial AbbasiK. BMJ2020 November 13; 371 https://www.bmj.com/content/371/bmj.m4425

493. The illusion of evidence-based medicine.Jureidini J, McHenry LB. *BMJ* 2022 March16;376. https://www.bmj.com/content/376/bmj.o702

494. *The Real Anthony Fauci.* Robert F. Kennedy Jr.Skyhorse Publishing 2021, pp. 8-9

495. Hyperlink to 1,366 peer-reviewed medical journal publications documenting severe adverse effects of the COVID-19 vaccinations. https://www.thegms.co/publichealth/pubheal-rw-22042302-references.pdf

Thorp KE, Thorp JA, Thorp EM. COVID-19 and the Unraveling of Experimental Medicine - Part III. G Med Sci. 2022; 3(1):118-158.
https://www.doi.org/10.46766/thegms.pubheal.22042302