The Covid-Szenario as the Coronation of the Transhumanist Agenda

by

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Abstract

The events that occurred around the covid-19 pandemic were accompanied by a huge amount of apparently irrational behaviour of mainstream media, health authorities and politics. To achieve a coherent understanding of our latest history, it is unavoidable to reconsider those events as parts of a running transhumanist plot to turn humanity into a cyborg-race. This includes the expulsion of the soul and the emotional body, the mentalization of all remaining consciousness structures and - as a capstone - the genetic transformation of the human genome itself. The article delivers a first understanding how this task is accomplished by biochemical, genetic, epigenetic and bioenergetic measures, utilizing both covid-19 and the covid vaccines as bioweapons. The establishment of the core read/write interface utilizing Morgellons and Neuronal Nanobots has been discussed in previous articles. The biochemical integration of covid related changes into this preexisting structure is understood.

<covid-19> <long covid> <transhumanism> <thyroid> <niacin sink> <bioweapon> <Go Syndrome>

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1. Introduction

Both in Klaus Schwabs Locksteps to the Great Reset promoted by the WEF and the patent scripts of mRNA vaccines, Covid-19 and covid-19 measures appear to be the stage for the introduction of a transhumanist agenda which includes the genetic transformation and ownership of the human race. In the publication series ENVIRONMENTAL MEDICINE'S APPROACH TO GEOENGINEERING-INDUCED DISEASE, published by Harald Kautz, two transhumanist technologies have been described; the quantum-dot-dye-filled Morgellons, and neuronal nano-bots, addressing the biophoton based light-body and the type of consciousness generated by the central nervous system, both forming an artificial, bidirectional interface to read out and rewrite human consciousness. By the end of the year 2021, many properties of the covid-19 RNA, the mRNA of the vaccines and the spike protein, which the two produce alike, have been scientifically examined and understood. This article will outline, how the long-term consequences of a covid-19 infection as well as of vaccine administration bind into the transhumanist agenda.

In a brief overview, the article will show, how both the disease and the vaccine manage to put the human body into a state of chronic disease, disrupt self-repair mechanisms of the DNA to enable gene editing by mRNA-technology – risking cancer – detach the "human spirit", i.e. the perspectivized consciousness-aspect that would be able to reedit the human genome by creative expression, pulling its information from the natural "life-force field" carrying the blueprints of life. The article will show how the emotional body is detached and its anchor-crystals are replaced by a crystalline grid processing binary, i.e. mental fields only, opening the body to be possessed by administrative "coopted insects", aka archons. To be able to hypnotize humanity into this transformation, hunting constructs borrowed from a toxic cone snail and three different snakes have been introduced as a measure of mind control, creating the desire to be stung, as well as to create fear and division between the vaccinated and non-vaccinated.

To be able to understand the complexity of this insult, we need to revisit some of the knowledge we presume to have about the nature of our body. Science tends to handle reality with monocausal explanations, which change as often and as abrupt as the fashion. It just needs a short visit of the paradigms-shifts in medical science, to see and understand that though highlighting every single aspect has added to the medical understanding, following those paradigms as monocausal sources of disease has caused more damage to human health than it brought benefits.

2. Method - revisiting old concepts and cutting-edge research

A brief research on paradigm shifts in medical science brought up a list of 10 paradigm shifts, displayed in the following list as the numbers 1-10. However, before the discovery of germs and viruses, different observations served as explanations for disease, and being short in scientific measuring equipment, doctors relied on their senses – including perceptions that today would be classified as clairvoyance. Looking at the newest developments, epigenetics and the Dr.Jekyll-and-Mister-Hide-like twinflame of biophoton-research and optogenetics are highly qualified attendees for the next paradigm shifts to come.

But let's have a look at the full list.

0. miasmatic theory (western medicine) and theories of correlative correspondence (TCM)

- 1. infectious diseases
- 2. genetic diseases
- 3. diseases caused by malnutrition
- 4. diseases caused by hormonal dysfunctions
- 5. allergic diseases
- 6. autoimmune diseases
- 7. somatic mutation/selection diseases
- 8. ischemic cardiovascular diseases
- 9. amyloid deposits (including prions)
- 10. the NO/ONOO⁻ cycle
- 11. epigenetics
- 12. biophoton research and optogenetics

Just to refresh and update: miasmatic theory and the theory of correlative correspondence base on the assumption, that the physical plane correlates with an invisible, spiritual realm, that can directly be addressed in healing measures. Epigenetics acknowledge the fact that genes are either methylated or demethylated, switched on or off, and that this mechanism is as essential to genetic expression as the genetic code itself. Interestingly, epigenetics are sensitive to conscious decisions. Biophoton research and optogenetics base their reasoning on the discovery that a good share of genetic sequences produces single photon emissions, that add up to standing wave based light communication between DNAs. This leads into the field of quantum entanglement and phase conjugate technologies, that can interfere and interact with the light-based human consciousness – a variety of which can be found in transhumanist technologies.

It is easy to see, that good research covers the complex interaction of the different mechanisms, and tries to avoid monocausal explanations. Also, it is quite obvious, that the paradigm shift No 12 100% matches the lost knowledge of miasmatic theory, finally describing the matter the invisible is composed of. The author kindly suggests to the reader to re-visit this short excursion into meta-science if he should experience cognitive dissonance when it comes to the discussion of the invisible.

3. Data - Aspects of Long Covid

The following chapters down to 3.6.2 are a straight forward quote of the research done and published by the Butterfly Method, please visit updated versions at <u>butterfly-method.com</u>. I removed the treatment theory, practitioners and those who take responsibility for their own health can look it up at his website.

3.1. Microbiome Viral Persistence

Viral persistence is one question that is the easiest association to make with Long Covid and the most complicated. There is emerging research indicating COVID19 persistence challenges our understanding of normal viral behavior, including papers speculating it could integrate into the DNA and be expressed later. Dr. Bruce Patterson of InceIDX has developed an inflammatory marker panel for Long Covid which can show patterns of inflammation correlated to Long Covid and has

developed a theory of viral persistence. <u>His theory is that monocytes are harboring S1 viral proteins,</u> causing them to create an auto-immune response which he believes causes all of the symptoms of Long Covid. Monocyte persistence can't explain the long term evolving and complex symptoms of Long Covid and are an oversimplification of the body as a living system but it is evidence of persistence nonetheless. The most interesting research is from a small biotech in Italy who <u>observed</u> patients with COVID19 pathology who were testing negative and found that their fecal samples were positive for the viral RNA, this led them to do some very interesting work. Using their existing biotech capabilities, they saw covid <u>patient samples had peptide toxins similar to venoms present</u>, and <u>attracted a lot of attention</u>. They had different shuffling of amino acids - indicating they came from bacteria not a virus acting on a human body. They were able to culture sars-cov-2 genetic material on human microbiome cultures, showing the virus seems to grow and express SARS-CoV-2 genetic material. This is a fundamental discovery which connects many dots but is not accepted by the general scientific consensus system.

The authors then used a number of antibiotics and showed that they could drop the viral presentation to almost 0, the best antibiotic being azithromycin which might be familiar to many as it was promoted early in the pandemic. While it challenges our understanding of human viruses, research has found that the S1 protein can bind to and engage bacteria present in our microbiome and validates their hypothesis. This hypothesis could also contribute to the dramatic microbial remodeling that happens during COVID19.

This microbiome viral persistence theory could explain the observations that SARS-Cov-2 fecal tests stay positive much longer than other methods and those with diarrhea have much worse outcomes. It could also explain the front end of the viral persistence theory, S1 units are showing up in monocytes because they are being produced in the microbiome and transported through the body. This could also explain the fact that 50% of those hospitalized with COVID19 on a ventilator don't even have detectable viral load in the blood but it is detectable in the nasopharyngeal canal. In fact there is no correlation to circulating viral load and poor outcomes, but with nasal swabs which represent the microbiome it does correlate.

There is an enormous body of literature around the rapid and lasting alterations of the microbiome triggered by COVID-19. A Google Scholar search of "COVID-19 + microbiome" leads to over 36,000 research papers on the subject. A systematic review of the subject is an excellent jumping off point to navigate this enormous collection of research. Not only is there intestinal microbiome remodeling, but it is also represented in the microbiome of the lung, mouth and nasal canal in a number of papers linked Here, Here, Here, Here, Here, Here, Here and Here. Translocation of microbiome bacteria is found throughout the body in COVID-19 patients, with one paper considering this dynamic a key player in the acute disease. These studies indicate the complex relationship between the virus, bacteria and the body. It's interesting to note that breakdown of tryptophan metabolism is seen in dysbiosis and is highly involved in COVID-19. Papers discussing tryptophan metabolism and COVID-19 are linked Here, Here, Here, Here and Here. One studying observing MIS-C inflammatory syndrome in children after COVID-19 linked it to disruption of GI barrier and a persistent presence of viral material in the gut. By treating it with a peptide which closed the tight junctions, there was a rapid lowering of viral load and inflammatory markers. This indicates that "leaky gut" is a risk factor from inflammatory diseases resulting from COVID-19.

There is an exotic interplay with pathogenic challenge and the thyroid/parathyroid that is yet to be discovered and the microbial persistence of viral material could be constantly stressing the thyroid. The thyroid is very often strongly challenged immediately upon viral or bacterial infection, possibly being the cause of the "sore throat" which is very common early in infections. The mechanisms is not understood but there is <u>incredibly strong evidence of thyroid dysfunction and a huge cross section of viral diseases</u>, even when the viral material is not found in the thyroid. Detectable viral or bacterial infections preceded thyroid diseases in more than 40% of patients in one study. The mechanisms have not been found but there is some link between pathogenic presence in the body and thyroid challenge and this could be a source of consistent thyroid stress due to microbial viral persistence.

The known tryptophan disruption and production of indole containing tryptophan breakdown products in the gut can also directly challenge the endocrine system as they interact with the Aryl hydrocarbon receptor which can lead to systematic disruption of the endocrine system.

3.2. Thyroid/Parathyroid Dysfunction

3.2.1. The Thyroid

A review of the overlap between thyroid disorders and COVID19 discusses the many common morbidities and symptoms. A detailed review of COVID19 and the thyroid looks at a number of potential mechanisms as well as reviews the clinical literature. Thyroid dysfunction was linked to long term anosmia or loss of sense of smell, after COVID19 with more than 50% of patients having long term anosmia also having hypothyroidism. This is very relevant as a risk factor as 10% of the population has some type of thyroid dysfunction and more likely much more due to the complexities of testing thyroid function. Another review looks at clinical data and mechanisms around SARS-CoV-2 and the thyroid.

One study found higher SARS-CoV-2 viral load correlated to smaller thyroid volume. In one study, more than half of hospitalized COVID-19 patients had abnormal thyroid biomarkers during hospitalization that normalized during recovery. Another study in China found that around 62% of COVID-19 patients had thyroid abnormalities.

Mechanisms of loss of smell such as oxidative damage and direct viral invasion of the olfactory bulb could explain anosmia, but it cannot explain the phantom smells and occasional increases in sense of smell after COVID19 infection. These things can only be explained easily by thyroid dysfunction which often manifests as complex changes in taste and smell perception as well as changes in hearing such as tinnitus. Studies indicate that thyroid triggered tinnitus is caused by disruption of cochlear outer hair cells and a review of animal studies indicate mechanisms of disruption and show reversibility with normalization of thyroid function in most cases. A study of 50 patients who had their thyroid function tested during the course of their infection, 64% had abnormal thyroid function visible on laboratory tests of TSH, TT3 and TT4. A review paper found many studies that linked thyroid hormone dysfunction and COVID-19.

A recent paper looking at the metabolic changes after COVID-19 found <u>long term dysregulation of</u> <u>key metabolic ratios including lower glutamine/glutamate ratios, higher kynurenine/tryptophan ratios</u> <u>and higher LDL/HDL ratios</u>. The LDL/HDL ratio changes line up with <u>thyroid dysfunction as it is</u> <u>highly involved with lipid metabolism</u>, and <u>a recent study on LDL/HDL ratios found its an excellent</u>. <u>marker for detecting what is called "asymptomatic subclinical hypothyroidism"</u>. While microbial dysfunction could also explain it, the <u>tryptophan pathway and kynurenine metabolism is known to be</u> <u>affected by a dysfunctional thyroid</u>. Finally, <u>the glutamine-glutamate cycle is known to be</u> <u>compromised in hypothyroidism</u>.

All of the long term metabolic marker shifts detected in these studies are changes expected when the thyroid is dysregulated. The studies show it could be caused by "asymptomatic subclinical hypothyroidism" which challenges our modern medical testing paradigm which relies on biomarkers and symptoms to determine problems in the body.

A patient advocate group led survey of 1200 Long Covid patients early and the pandemic painted a picture of the age and gender prevalence of Long Covid. Women are represented significantly more than men and there is a peak in middle age which is surprising as it does not correlate to immune strength. This same representation of age and gender prevalence is seen in <u>literature reviewing cases</u> presenting to a hospital with thyroid conditions over time.



Figure 3. Long Covid survey compared to age distribution of thyroid cases in a hospital survey

A website which reviews and catalogues all available research on compounds which are studied for <u>COVID19 treatment</u>, assigns an index number according to the benefit of the compound averaged across the studies. In the context of mortality results, Povidone-iodine ranks 88% while trendy and controversial treatments such as Ivermectin rank 55%, Hydroxychloroquine ranks 22%, Vitamin C ranking 19% and the standard of care Remdesivir ranking 19%. Most interesting, the protocol for this iodine based treatment is not ingestion but nasal irrigation. The general thought process is that this drops the viral load in the nose by directly destroying the viral material. Most likely the mechanism is actually the stimulation and supplementation of the thyroid gland due to iodine being absorbed into the nasal mucosa in close proximity to the thyroid gland. This is validated by the <u>study of nitric oxide</u> nasal sprays which dramatically dropped the viral load but only caused improvements in 50% of the patients.

Deficiencies in thyroid cofactors like selenium, copper and vitamin A are all correlated with worse COVID-19 outcomes. <u>Selenium is involved in iodine processing as well as the production of critical seleniocystein proteins such as glutathione reductase and a lack of selenium links to higher rates of morbidity. Low Vitamin A is also highly correlated to poor COVID-19 outcomes and one paper speculates that high dose vitamin A could be a relevant therapeutic approach for COVID-19.</u>

Studies of patients with thyroid dysfunction also show lower levels of zinc and copper which are important cofactors for thyroid functioning. Copper is critical for iron transport and homeostasis and is chronically missing from our modern society. Not only does its deficiency trigger iron deposition leading to endocrine disruption <u>but it is directly involved in supporting thyroid function. Serum</u> copper levels are strongly correlated to thyroid dysfunction and are considered an acceptable biomarker for thyroid hormone resistance. One study even looked at copper/iron dynamics in thyroid dysfunction and found interesting correlations. Dysfunction of the thyroid is linked to altered iron homeostasis, although the mechanism is not understood. There seem to be positive feedback mechanisms and cascading effects between thyroid and iron dysfunction, strongly linked to deficient cofactors such as copper.

Symptoms of <u>thyrotoxicosis include heat intolerance</u>, <u>palpitations</u>, <u>anxiety</u>, <u>fatigue</u>, <u>weight loss</u>, <u>irregular menses in women</u>, <u>and tremor</u>. Those noticing changes in symptoms during sleep-wake cycles or menstruation cycles should consider the role of thyroid in their disease state. <u>Studies have</u> <u>shown that hypothyroidism is linked to low ferritin</u> while <u>high ferritin is correlated to</u> <u>hyperthyroidism and normalizes during therapy</u>. In subacute thyroiditis, which is a known condition of thyroid dysfunction caused by infections, there is a correlation to disruption in iron homeostasis due to increases in hepcidin, an iron transporter. Other studies on auto-immune hyperthyroidism. found there was a disruption of iron homeostasis that could not be explained by changes in hepcidin levels. There is a dangerous feedback loop when it comes to the thyroid and tissue iron overload, as iron preferentially accumulates in endocrine tissues which lead to dysfunction of the thyroid and further iron homeostasis disruption, even trigger thyroid autoimmune disorders.

The thyroid hormone T3 was found to tightly correlate with serum ferritin levels in a number of papers linked: <u>here</u>, <u>here</u>, <u>here</u> and <u>here</u>. Most interestingly, in all of the linked studies, the ferritin normalized upon normalizing of the T3/T4 hormone ratio. Experiments administering T3 to hyperthyroid patients dramatically increase ferritin. <u>Patients with even sub-acute thyroid issues had</u>

altered ferritin levels. The mechanisms are not understood but recent studies have found that T3 regulates Iron Regulating Protein.

One study reviewing many iron markers in hypothyroidism found that it lowered levels of iron and ferritin while dramatically increasing total iron binding capacity. While this seems like a positive thing, these tests do not measure tissue iron deposition which is the real driver of morbidity and and swings in iron transport markers indicate a potential breakdown of iron homeostasis and iron deposition. A good insight into this dynamic is seen in experiments with rats having induced hyperthyroidism and hypothyroidism, both sets had increased levels of liver iron deposition while the hyperthyroid rats had 36% lower ferritin and the hypothyroid had 38% higher ferritin.

Thyroid dysregulation often causes visual disturbances and the mechanism has been found to be visual evoked potential slowing, this can be tested for non-invasively by flashing a series of images while connected to an EEG. In fact many of the sensory disturbances caused by thyroid issues are due to compromised evoked potentials and this may be the core mechanism of the loss of smell. Those with visual disturbances may experience "Pattern Glare" and another self diagnostic test is the Visual Contrast Sensitivity Test which can be taken for free online, this test is often used for neurotoxin related illnesses to monitor recovery and those recovering from COVID-19 may see dramatic increases in their VCS scores. In general issues with the thyroid can lead to a number of strange and evolving eye symptoms.

Thyroid dysfunction is known to affect carbohydrate metabolism and both hypothyroidism and hyperthyroidism are known to lead to diebetic conditions and glucose insensitivity through different mechanisms. Thyroid dysfunction also effects brain energy metabolism and could be the cause of the strange brain fogs and other brain abnormalities.

"Thyroid Storm" is an inflammatory breakdown of the thyroid gland that may be highly relevant in COVID-10, with case studies already identifying it linked <u>Here</u>, <u>Here</u>, <u>Here</u>, <u>Here</u> and <u>Here</u>. Interestingly, <u>the use of corticosteroids is very helpful for thyroid storm</u> and are one of the more beneficial advances in widely used front-line therapies for COVID-19. Interestingly, research studying "cytokine storm" in COVID-19 found that the concept of cytokine storm may be irrelevant and its link to COVID-19 may just be a result of media pop-science driving academic researchers. It seems there are instead positive feedback loops of oxidative stress, "thyroid storm" and other mechanisms may be contributing additional cytokine markers.

The cardiac manifestations of COVID-19 are observed via biomarkers such as troponin, but in terms of tissue studies and autopsies, a viral attack based cardiac damage is never confirmed. It's very interesting to note that thyroid dysfunction can trigger cardiac symptoms as well as an increase in the troponin biomarkers used to determine cardiac damage, without any organic heart damage. In fact, there are many case studies showing thyroid dysfunction mimicking or driving cardiac disease including cardiac damage biomarkers linked Here, Here, Here and Here. These mechanisms combined with the electrical decoherence in the heart caused by iron and calcium mineralization could cause all of the observed cardiac abnormalities associated with vaccination and COVID-19 and should be quickly reversible if this is the case. This is an excellent review paper on the cardiac manifestations of thyroid dysfunction.

While there are many studies on the levels of thyroid stimulating hormone (TSH), Triiodothyronine (T3), Thyroxine (T4) as well as auto-antibodies against thyroid related molecules, there are no studies on thyroglobulin levels or more granular thyroid biomarkers during or after COVID-19. <u>There is an ongoing clinical trial</u> which reviews thyroid biomarkers and correlates them to inflammatory and other markers that correlate with disease progression.

In the general medical practice, TSH, TT3 and TT4 are the common tests ordered with TSH out of range often being the main deciding point of the hyperthyroidism or hypothyroidism function. This is a flawed understanding and oversimplification and should never be used on its own to start thyroid hormone treatment. TT3 is a marker dependent on the liver and gut and represents the measurement of an axis of organs and not a direct thyroid marker. TT4 may be the more relevant biomarker in a standard thyroid panel although most doctors make decisions looking at TSH in isolation. A proper analysis of thyroid function will compare many markers of thyroid function to determine transport, binding, conversion and other issues.

A proper thyroid analysis should include: TSH, Total T3, Free T3, Total T4, Free T4, T3 Uptake, Reverse T3, TBG Antibody, TPO Antibody and TGB Antibody for a full picture of what is going on in the thyroid. <u>While Tg is traditionally used as a cancer biomarker, its levels indicate iodine status</u> and should also be added. It's also important to note that the thyroid is highly dynamic particularly in situations of recent infection or immune challenge and any measurements should take that into

account. <u>The levels of TSH are known to fluctuate on a daily cycle and can double over the course of a single cycle</u>. In fact, normal thyroid panels of TSH, TT3 and TT4 taken at a single time point may not represent the status of the thyroid at all. The fact that most labs use "adapted ranges" of what thyroid levels should be, which creates a moving target when comparing results from one lab to another.

In general, thyroid testing should be done with an endocrinologist who is properly trained to administer and analyze a full thyroid panel. Thyroid ultrasounds are a much more sensitive measurement of thyroid function as issues often show up on ultrasound but not on bloodwork. More than half of the population has thyroid nodules so ultrasound could be a source of stress as nodules are analyzed to determine potential for cancer activity, which is why most practitioners wont do an ultrasound of the thyroid until after the blood tests have been done.

Studies have shown that <u>thyroid dysfunction causing the same functional changes seen in Long Covid</u> <u>can come from thyroid dysfunction which does not cause acute symptoms and is not detectable via</u> <u>clinical biomarkers</u>. This challenges our normal medical paradigm of relying only on blood tests to determine dysfunction and diagnosis as it will simply not be visible in many cases. In fact, when it comes to thyroid dysfunction, <u>papers have stated that lab analysis is not sufficient to rule out thyroid</u> <u>issues and symptoms</u>, patient history, ultrasound and other methods should be used together and intuitively.

3.2.2 The Parathyroid

The parathyroid glands are locally very close to the thyroid and regulate calcium metabolism, a recent paper investigates the role of the parathyroid in COVID-19. <u>A review called "Managing</u> Parathyroid Disease in COVID19" reviews the available evidence of involvement of the parathyroid in COVID19, possible mechanisms, case studies and treatment theory. Parathyroid dysfunction is associated with calcifications in the brain and nervous system, which can manifest as complex. neurological symptoms. Using advanced imaging techniques, microcalcifications were detected in one case study, linked to a dysfunction of the parathyroid gland.

The thyroid and parathyroid glands are known to responsively change morphology during dysfunction, ultrasound tests of the thyroid and parathyroid may be a simple non-invasive method to indicate if they are involved in Long Covid. While the general medical understanding is that the thyroid and parathyroid serve different functions and are unrelated, the general medical understanding is simply wrong and as usual does not see the human body as an interconnected system.

While these two glands serve different regulatory functions, they are located in the same physical location and both are a part of the endocrine system. <u>A study of patients with parathyroid</u> <u>dysfunction found that 71% of them also had thyroid dysfunction</u>. Just like with the thyroid, hypoparathyroidism perfectly correlates with low ferritin levels. There are documented cases of thyroid issues triggering parathyroid issues. There are feedback loops where thyroid dysfunction leads to iron dysregulation, which causes deposition of iron in endocrine tissues, creating a positive feedback loop. There are also feedback loops where dysfunction, in fact, <u>calcifications are found in 38.6% of thyroid nodules</u>.

The parathyroid is responsible for converting 25-hydroxyvitamin D to its active form, which could indicate why vitamin D status was so strongly correlated to fatality but supplementing vitamin D did not have such strong correlations to survival, it is because the lack of vitamin D represents a metabolic breakdown not a deficiency. In fact, vitamin D supplementation will negatively affect the important vitamin A/vitamin D ratio which strongly affects calcium and iron transport. There is a clinical trial which will be fascinating to follow which studies parathyroid hormone in the progression of COVID19.

A study of hyperparathyroidism indicates that the spontaneous ground glass opacities can form due to diffuse calcification. This finding is rare and it may only be possible due to nucleation of iron deposition in the lungs combined with dysfunction of the parathyroid specific to COVID-19. The study states, "On X-ray examination, the disease manifests as scattered patchy shadows with restricted distribution, whereas on chest CT, it manifests as disperse patchy shadows or ground-glass opacity". These findings are identical to COVID19 lung pathology. A study of those having long

term, non resolving lung pathology found that 77% had dysregulation of iron transport. This may indicate that iron deposition in the lungs triggers spontaneous calcium crystallization due to the combined dysfunctions of the thyroid and parathyroid glands and iron and calcium homeostasis respectively.

An emerging and experimental approach is the compound methylene blue which has traditionally been used as an industrial dye, it has become a favorite of biohackers for its positive effects on bioenergetic functioning. Methylene blue is also known to be an endocrine modulator and stimulates the thyroid gland, its commonly used before parathyroid surgery as it preferentially is absorbed by endocrine tissue and stains it blue. In clinical research for hospitalized COVID-19 patients, methylene blue rapidly increased the blood oxygen and was almost twice as effective than the standard of care at reducing death.

While there are not many common biomarkers for parathyroid dysfunction, parathyroid hormone is one that can be commonly tested for as well as ionized calcium and blood calcium. In fact graphing blood calcium vs parathroid hormone can indicate disease states or a move toward disease states in a very elegant manner. While blood calcium vs PTH is an excellent way to triangulate disease states, ionized calcium is also a very important metric to compare to blood calcium.

Anecdotally, it seems that parathyroid dysfunction is much more common during peaks of illness such as the March 2020 peak. This explains why Long Haulers who got sick around this time had much worse outcomes and longer time to recovery, potentially due to tissue mineralization. This seems to be caused by the high viral load, or most likely some yet understood environmental mechanisms which amplify COVID-19 morbidity.



Figure 4. Calcium graphed against Parathyroid Hormone can indicate disease state according to the Norman Parathyroid Clinic.

3.3. Psychobiology of COVID-19 Theory

As the thyroid seems to be the only organ specific dysregulation it's interesting to think about what the thyroid represents in terms of psychobiology. In the study of psychobiology, it is speculated that consciousness constructs or emotional conflicts can cause stress and dysfunction in specific organs. While psychobiology is not well understood and does not have a place in our current public health

paradigm, it is rapidly emerging and may be a game changing modality in the future. <u>Psychobiology</u> is already a thread that runs through every aspect of medical research due to the fascinating placebo and nocebo effects.

In psychobiological modalities, the thyroid gland is associated with emotional conflict archetypes of "not being fast enough" which comes from the archetypal conflict of "not being fast enough to take or offload a morsel of food". The general theme of "running out of time" associated with the thyroid is interesting as the thyroid regulates metabolism and <u>dysfunctions in the thyroid are known to alter</u> <u>time perception</u>. In the study of psychobiology, the consciousness conflict of "running out of time" stresses the thyroid gland and triggers a psychobiological coping mechanism to speed up or slow down the organisms metabolism.

Interestingly studies found a 60% higher rate of death in some occupations which is an enormous statistical signal that needs to be understood. Those occupations include cooks, agricultural packers, food packers, transporters and other jobs that are constantly under time pressure to "get rid of a food morsel" and could have compromised thyroids in the lens of psychobiology. Many of the other jobs highly represented are jobs with time pressure unrelated to food. Jobs involving work on computers are represented which is interesting as the <u>cumulative negative effects of blue light radiated from</u> computer screens is known to cause thyroid dysfunction in itself. There is a strong representation of jobs such as security services, military and groundskeepers which is interesting as the psychobiology conflict associated specifically with the ducts of the thyroid are "moving face forward into a dangerous situation knowingly". If these are conflicts you see causing strong emotional responses in multiple places in your life, and you are interested in exploring psychobiology please <u>book a</u> consultation as it is a main research interest.

3.4. Disruption of Iron Homeostasis

There are strong indications of disruption of iron homeostasis in COVID19 — the paper "<u>Iron:</u> <u>Innocent bystander or vicious culprit in COVID-19 pathogenesis?</u>" reviews mechanisms and findings connected to iron in COVID19. <u>Multiple papers</u> have found that large amounts of free iron in the body are correlated to fatality, blood types with higher iron binding capacities correlate to fatality and iron dynamics are significantly interrupted in COVID19 patients.

A paper titled, "<u>COVID-19 gone bad: A new character in the spectrum of the hyper-ferritinemic</u> <u>syndrome?</u>" goes into detailed and compares COVID19 biomarkers to hyper-ferritinimia biomarkers in the acute phase, but does not discuss the long term issues. <u>Another paper</u> links issues with the blood to ferritin biomarkers and states, "Strongly associated with the COVID-19 coagulopathies is the presence of hyperferritinemia". <u>Another paper</u> discusses the role of iron chelators and COVID19 being a hyperferritinemic syndrome. <u>A paper discussing iron nano-particle pollution and COVID19</u> <u>mechanisms</u> states, "altered iron balance favoring excess reactive or catalytic iron may be the single most important underlying pathological process predisposing to severe COVID".

<u>A study of patients 60 days after COVID19 stated</u>, "60 days after disease onset, 30% of subjects still presented with iron deficiency and 9% had anemia, mostly categorized as anemia of inflammation. Anemic patients had increased levels of inflammation markers such as interleukin-6 and C-reactive protein and survived a more severe course of COVID-19. Hyperferritinemia was still present in 38% of all individuals and was more frequent in subjects with preceding severe or critical COVID-19."

A paper has recently been published which shows that there is a very interesting <u>sequence similarity</u> <u>between hepcidin, the iron transport enzyme and the SARS-CoV-2 genetic sequence</u>. This is relevant to research because as <u>one paper states</u>, genetic malfunctions affecting the hepcidin-ferroportin axis are a main cause of iron overload disorder. Another relevant paper shows that transferrin receptors, an iron transport system, are an entry receptor for SARS-CoV-2 with binding affinity almost as strong as ACE-2.

Iron release into the microbiome can disrupt biofilms and cause the mobilization of bacteria, it can also convert these bacteria to "pathobionts" or potentially harmful bacteria. In general, most pathogenic bacteria possess more efficient pathways to acquire free iron than beneficial bacteria as it is essential for virulence expression and replication. Iron overload is known to alter the microbiome.

particularly the tryptophan metabolism, which is already known to be causing issues in the niacin sink trap. This is another example of multiple mechanisms creating feedback loops and catalyzing each-other in Long Covid. Many papers linked breakdown of tryptophan metabolism and COVID-19 are linked Here, Here, Here, Here and Here.

Interestingly, this is the exact mechanism proposed to contribute to Alzheimer's Disease, known as the "Iron Dysregulation and Dormant Microbes (IDDM) hypothesis". <u>A recent paper states</u>, "The simultaneous iron dysregulation and microbial aberrations affect the hematological system, promoting fibrin amylodiogenesis, and pathological clotting. Systemic inflammation and oxidative stress can contribute to blood brain barrier permeability and the ensuing neuro-inflammation, characteristic of Alzheimer's type dementia." Iron is also a critical mechanism to mediate bacterial infections and <u>forward looking research shows anomalous presence of bacterial DNA reads and biomarkers in COVID19 clinical data indicating attack of iron utilizing anaerobic Prevotella sp. bacteria on the blood.</u>

COVID19 may most similarly mimic cadmium poisoning, where a catalytically active metal is causing systemic oxidative stress. It is relevant to note that in cadmium poisoning, the symptoms overlap perfectly with COVID19, including "cytokine storm", anosmia and ground glass lung opacities. The cadmium poisoning mechanisms have been well studied and an analysis will show its close relationship. Combined with the many papers linking COVID19 and systemic oxidative stress, it paints a picture which looks more like a metal catalyzed oxidative disease than a respiratory illness. In fact, the whole "cytokine storm" concept in COVID19 seems to be pop-science, a recent paper indicates that the cytokine storm component of COVID19 is minimal, it appears to be an "oxidative storm".

Iron causes rampant oxidation in the body through energetic oxygen radical production, including via the extremely powerful Fenton reaction. Iron will oxidize any materials it comes into contact with, specifically cellular membrane lipids (such as the mitochondrial membrane) and DNA. Recently, <u>a</u> paper was published which reviewed biomarkers of oxidative DNA damage and found a strong correlation to COVID19 severity. Another paper which looked at Ascorbate levels which would be reduced during oxidative stress, states: "Our study revealed that vitamin C levels are undetectable in more than 90% of the patients included. The mechanisms of this significant reduction in vitamin C are uncertain." A study of antioxidants as well as biomarkers of oxidative stress in COVID19 patients shows dramatic patterns of low antioxidants and high oxidative stress. Thiol levels, a common biological group very sensitive to oxidation is highly correlated to COVID19 disease progression in a recent paper. The serum thiol biomarker levels tested are considered a direct measure of redox status because of their rapid reaction with oxygen radicals.

Excess iron in the brain can cause a number of issues, including the micro-hemorrhaging seen on COVID19 MRI studies. This disease state is known as neuroferritinopathy or the general term, Neurodegeneration with Brain Iron Accumulation (NBIA). This could explain some of the strange symptoms of "long haulers" such as neurological changes on just one side of the body. Research around iron dysfunction in the brain and nervous system is one of the most exciting areas of medicine, with a recent paper stating: "A major feature of virtually all neurodegenerative diseases is the accumulation of excess iron." In general, excess iron levels are associated with Alzheimer's disease, Parkinson's disease, Huntington's disease, Friedreich's ataxia and other neurological disorders, cancer, Fanconi anemia, stroke, heart disease, diabetes and ageing. A fascinating video discussions how iron accumulation and dysregulation may be a core mechanism of aging and age related diseases.

Ferritin deposited in the brain will show up on MRI according to a review, "On brain MRI in neuroferritinopathy, iron deposits are observed as low-intensity areas on T2WI and as signal loss on T2()WI. On T2WI, hyperintense abnormalities reflecting tissue edema and gliosis are also seen. Another characteristic finding is the presence of symmetrical cystic changes in the basal ganglia, which are seen in the advanced stages of this disorder. Atrophy is sometimes noted in the cerebellar and cerebral cortices.". T2 hyperintensities of white matter is one of the most common reported MRI findings in those that are suffering from Long Covid and a paper on iron storage disease states, "T2 hyperintensities in white matter have been reported in most NBIA (Neurodegeneration with Brain Iron Accumulation) subtypes".

<u>A recent paper discusses</u> the unique aspects of iron induced fibrogen clots, they are not only remarkably resistant to degradation but they have an ability to "capture" red blood cells and cause further damage. The reason they are resistant to healthy degradation is due to the iron causing active

oxidation to the enzymes trying to break them down. This strange decomposition resistant fibirinamyloid type microclotting is the <u>exact type seen persistent after the acute COVID19 phase</u>. The author points out that this mechanism looks identical to iron triggered clotting . <u>Another study shows</u> the connection between iron overload diseases and the strange blood clotting and red blood cell shape changes seen in COVID19.

Long haulers often suffer from "Sticky Blood Syndrome" where it is incredibly difficult to draw blood from them, this is a known effect of anti-phospholipid syndrome, an autoimmune disease commonly seen after COVDI19. While multiple studies show that COVID19 causes auto-antibodies targeting the blood, the root cause has not been identified. <u>These exact same autoimmune diseases are seen in hyperferritinemia</u>, triggered by free iron releasing in the blood. In fact, iron effecting the blood in this way can lead to "Catastrophic Anti-phospholipid Syndrome", which one paper theorizes could be a mechanism of COVID19 fatality. <u>A meta-analysis found almost half of patients</u> <u>hospitalized with COVID19 developed the same auto-antibodies that are known to be induced by iron</u>. This same paper discusses iron causing macrophage activation syndrome. Its interesting to point out that in ferritin triggered macrophage activation syndrome, the general treatment is corticosteroids, the same treatment which is used in acute COVID19 cases and seems to prevent the manifestation of some long term side effects, but coming with its own issues.

This macrophage activation comes from their inability to process iron properly and get "fat with iron", which some researcher believe is due to issues with copper/ceruloplasmin enzyme and a driver of auto-immune disease. A study correlates the lung damage seen in COVID19 with iron levels, indicating a mechanism of lung damage similar to the ground glass opacities seen in cadmium toxicity- metal catalyzed auto-oxidation. <u>Tissue analysis shows iron filled macrophages in lung tissue from COVID19 patients</u>. Another tissue study shows a number of iron abnormalities, including iron laden macrophages and iron dysregulation in the bone marrow.

Dysfunction of the thyroid is linked to altered iron homeostasis, although the mechanism is not understood. This is complex because the endocrine system is one of the main targets of iron deposition, indicating that there are positive feedback loops of iron dysregulation and endocrine dysfunction. The cardiac manifestations of COVID-19 are observed via biomarkers such as troponin, but in terms of tissue studies and autopsies, a viral attack based cardiac damage is never confirmed. Cardiac tissue is one of the preferential points of tissue iron accumulation and can lead to all of the same effects observed in COVID-19. Combined with calcium mineralization on top of the accumulated iron and vascular/cardiac calcification, the mechanisms of iron and calcium deposition can explain all of the observations of COVID-19 cardiac damage and are reversible. Some researchers specializing in iron indicate that all iron lab tests do not represent iron deposited in the tissues and low iron is almost never due to lack of iron in the diet. In fact, there are many researchers who believe that the laws regulating that iron needs to be added to grains and other staple foods, are causing much more harm than good. They state there is evidence that iron is correlated to a number of modern disease states and combined with the environmental toxins like glyphosate disrupting iron homeostasis. This may be a case of pop-science driving public policy, as anemia seems to be caused by disruption of iron transport and not unavailability of iron in the diet. In fact, Morley Robbins states that the majority of people he sees in his nutritional practice with iron overload are actually vegetarians who eat more grains and other food artificially supplemented with iron. Groups like the Gates Foundation heavily promote iron supplementation in the developing

world in an attempt to stamp out anemia, but on the population level they may be doing much more harm than good, particularly in the context of a disease like COVID19 which mobilizes and redistributes iron. It does seem that anemia is an issue with iron transport, not dietary iron, in fact the role of copper in anemia should be promoted instead of iron according to a study cited by researchers in this area.

While MRI can be used with a skilled technician to analyze the iron present in the brain, not all types of iron can be properly visualized. <u>According to research on brain iron oxides</u>, "An alternative clinical technique for the detection of brain iron is trans-cranial ultrasound or trans-cranial sonography (TCS). TCS has been developed as a cost-effective and portable method to detect changes in certain brain regions exhibiting iron accumulation".

3.5. Disruption of Calcium Homeostasis

A study of tissues in those that died of COVID-19 found overt calcification in more than 10% of cases in the lung tissueAnother clinical research paper finds calcium and vitamin D are highly correlated to poor outcomes. A paper titled, "Intramuscular deposit of calcium is a potential reason for hypocalcaemia in COVID-19" points out that low calcium could be due to calcium crystalizing and depositing in the tissues and shows examples of dramatic and rapid calcifications in the muscle tissues during COVID-19. Multiple studies have found that low calcium and low phosphate are excellent predictors of disease severity. This makes sense in the context that one of the most common forms of calcium crystallization is calcium phosphate based crystals.

One virologist found his humanized rats were spontaneously sick with COVID-19 like pathology and their tissues tested positive for spike and capsid proteins using immunological staining. He also found deposits of iron, colocalized with bi-flourescent crystals, suspected to be calcium phosphate crystals due to the dual optical emission depending on their structure. The images below are from unpublished literature currently in peer review. Fluorescent microscopy of biological calcifications caused by calcium phosphate crystals shows very similar fluorescent properties.



Figure 5. Unpublished research from Bitily et al showing iron deposition as well as fluorescent mineralizations suspected calcium phosphate in the tissues of humanized rats which stained positive for SARS-CoV-2 spike and capsid proteins.

There are a number of case studies of rapid onset arthritis, pseudogout and muscle calcifications during COVID-19 or after vaccination linked to this potential mechanism linked <u>Here</u>, <u>Here</u>, <u>Here</u>, <u>Here</u> and <u>Here</u>. A review of the case studies and literature around rapid onset arthritis due to COVID-19 is a good jumping off point. One study even found large increases in search terms on Google. Trends related to calcification symptoms and treatment during the pandemic.

There is a huge body of work on vascular calcifications during COVID-19 as they are easy to image with normal methods and strongly linked to poor outcomes. A number of representative papers are linked <u>Here</u>, <u>Here</u>, <u>Here</u>, <u>Here</u> and <u>Here</u>. <u>Cardiac calcifications are a known cause of myocarditis</u> and the mechanisms of cardiac calcification should be a top research priority due to the existing literature

linking it with COVID-10. Tissue iron deposition is known to trigger co-mineralization with calcium which is also known to cause myocarditis.

It's important to note that calcifications do not have to be this dramatic to cause long term issues and that calcification is a general process in the body, diffuse calcifications may fall under the limits of detection of many imaging methods. Tissue biopsies could be used with very specific microscopy methods to detect calcifications even at a low level, but no studies of this type have been published. In calcification diseases, iron deposition in the tissues acts as a nucleation point to start calcium crystal growth from calcium rich biofluid in the location. Therefore any disease state which causes iron dysregulation as well as calcium dysregulation, will cause feedback loops of co-crystalization. Tissue calcification, similar to tissue iron overload, seems to be a core mechanism of health problems during aging.



*Figure 6. Spontaneous muscle calcifications found in the paper, "<u>Intramuscular deposit of calcium</u> is a potential reason for hypocalcaemia in COVID-19".

A study called, "<u>COVID-19 Unveiling Brain Calcifications</u>", goes into detail about the brain and nervous system calcifications that could be leading to neurological issues in COVID-19 and being not easily detectable on standard imaging.

A record of a calcification event can be seen in the eyes in some cases after COVID19, this would be relevant to look at if there are continuing eye pains and vision changes. These rings are called the "Limbus Sign" and represent calcifications which are stabilized by the fatty tissues around the iris and are persistent even when calcium homeostasis normalizes. Because calcium phosphate crystals are UV fluorescent, you can observe these rings in the eyes by shining a blacklight at an angle. While UV is dangerous for the eyes, commercial blacklights are longer wavelengths that are generally safe and used in nightclubs around the world.

Those with the Limbus Sign should have their parathyroid function tested, either through testing the blood for the levels of parathyroid hormones or through ultrasound imaging of the parathyroid glands. Low calcium is intimately linked to the dysfunction of the parathyroid gland, but its root cause has not been identified in studies of COVID-19 which focus on receptor binding chemistry and not the body as a living system.

3.6. Metabolic Traps

3.6.1. Niacin Sink Metabolic Trap

The Niacin Sink Trap has been documented and well researched, <u>initially during the study of HIV by</u> <u>Dr. Ethan Taylor</u> and recently in the context of COVID19, the downstream effects on tryptophan and other systems has been researched in a groundbreaking paper regarding Long Covid. Dr. Taylor studied COVID made the <u>observation that low selenium levels correlated to poor outcomes in China</u>, most likely due to a triggering of the niacin sink trap although it is also known as an important thyroid cofactor. This mechanism of the breakdown involving tryptophan metabolism discussed in many papers, linked <u>Here, Here, Here</u>, and <u>Here</u>. There is a lot of evidence of this metabolic trap and NAD+ depletion during COVID-19 which could be treated by niacin and cofactors. The paper, <u>COVID-19: NAD+ deficiency may predispose the aged, obese and type2 diabetics to mortality through its effect on SIRT1 activity</u>, has received an excellent response and <u>seems to be validated in mice</u>, relevant abnormalities found in ferrets and the mechanism echoed by other researchers. Dysfunction of relevant systems are found in COVID19 and correlated with inflammatory markers. Clinical trials are under way to treat the same mechanism using more exotic and commercial compounds than the simple ones used with success in their case. The theory is that oxidative stress causes NAD+ depletion, due to DNA repair enzymes (PARP) overactivating and some people with vitamin deficiencies, existing NAD+ depletion or genetic deficits in energy metabolism have long term NAD+ dysfunction. The body tries to make up for low NAD+ by feeding in tryptophan, which is the precursor for serotonin and melatonin, causing poor mood and sleep. Serotonin is also a master regulator in circulation and having low levels causes a number of issues including with the gut/brain axis. Every time tryptophan is fed into the cycle, large amounts of neurotoxic Kynurenine metabolites are produced. Furthermore, energy disruptions are specific to high metabolically active tissues such as the heart and brain.

The Open Medicine Foundation, a leader in Chronic Fatigue Research, supports a number of research initiatives into this mechanism which they generally call the "<u>Metabolic Trap Theory of CFS</u>" which indicates a breakdown of the tryptophan homeostasis and a metabolic trapping of the IDO pathway. A quality blog post goes into detail in the various aspects of OMF promoted metabolic trap theory. Observations of high blood sugar being a predictor of death in COVID19 patients, regardless of diebetic status validate metabolic changes being a major risk factor of COVID19 according to NAD+ theory. Activation of the same kynurenine pathways predicted in NAD+ theory of COVID19 correlate to blood sugar deregulation. A number of recent metabolomics papers have found massive disruptions in tryptophan and kynurenine markers and a paper reviewed disrupted tryptophan metabolism in the lens of microbial dysbiosis and other systematic changes.



Figure 7. Biochemical cascades in a vicious feedback cycle based on the original work of Dr. Taylor, "The oxidative stress-induced niacin sink (OSINS) model for HIV pathogenesis"

Unrelated to the core mechanism, a recent publication in the journal Nature, stated: "vitamin B3 (niacin or nicotinamide) is highly effective in preventing lung tissue damage. It might be a wise approach to supply this food supplement to the COVID-19 patients." A recent bioinformatics analysis also indicated niacin should be studied as a treatment for COVID19. A forward looking pre-print indicates nicotinic acid may act as a one of a kind bioenergetic "pump" of inflammatory molecules out of cells, critical for COVID19. Niacin has been seen to easily cure systematic NAD+ deficiency in clinical research.

<u>Researchers have found that melatonin helps express GPR109A</u>, the receptor that many of niacins therapeutic effects seem to come from and anecdotally the use of melatonin before niacin seems to significantly increase the beneficial effects. <u>Melatonin is also an iron binder specific to the nervous system and is neuroprotective against metal auto-oxidative damage.</u>

This method of using melatonin to express the GPR109A receptors followed by niacin is called the "Niatonin Method" and has developed an almost cult following due to its ability to rapidly restore energy homeostasis in some cases and rapidly alleviate some inflammatory diseases.

3.6.2. Methyl Metabolic Trap

While it has been understood since the 1980's, the methyl metabolic trap is very well explained in the context of COVID19 in the very important paper, "<u>COVID-19: A methyl-group assault?</u>". <u>One group of researchers points out the genetic complexities in this methyl trap in the context of COVID19</u>, as well as the resulting homocystein overload that can be present.

<u>One piece of clinical research</u> points out that there is a link between biomarkers of NAD+, oxidative stress, methylation, folate and B12 — this is interesting as it bridges the gap between the two metabolic traps. In a pro-oxidative environment, the unstable cob(I)alamin gets oxidized to cob(II)alamin. The paper states, "MS inactivation occurs when free radicals oxidise cob(I)alamin to a cob(II)alamin species. Re-activation requires <u>methyl group</u> donation by SAM" Other than oxidative stress, another mechanistic bridge seems to be the enzyme "Cob(II)alamin_reductase", which is an NAD+ dependent enzyme that converts cob(II)alamin back to cob(I)alamin. A third possibility is the direct disruption of methionone synthase or <u>catalysis of homocystein production by iron</u>.

Proponents of copper/iron bio-energetic balancing point out that <u>methionone synthase may very well</u> <u>be copper dependent</u> and copper deficiency is driving the unbalancing of iron, oxidative stress and metabolic traps. <u>Recent studies on rats being fed a copper deficient diet seem to validate this concept</u>. There is a known "homocysteine paradox" where high homocystein correlates to cardiac and vascular disease yet interventions that aim to lower homocystein are not effective at lowering morbidity. <u>Some researchers believe the homocysteine paradox is due to a lack of copper</u>, (and iron homeostasis disruption) therefore homocystein is not the driver of disease but a biomarker. <u>Multiple papers</u> show <u>xcess dietary iron is known to disrupt copper homeostasis</u>, which is required for proper regulation of iron transport. Whatever the mechanism, its clear the methylation/folate cycle is disrupted at the B12 dependant methionine synthase step.



Figure 8. Disruption of methionine synthase due to oxidative stress destroying cob(I)alamin, potentially by iron auto-oxidation, schematic based on the paper, "<u>COVID-19: A methyl-group</u> <u>assault?</u>".

The Methyl Metabolic Trap will create symptoms depending on the genetic makeup of the persons methylation system. Most common would be the symptoms of vitamin B12 deficiency including: anemia, "COVID tongue", shortness of breath, feeling cold, neurological damage dizziness, irregular heartbeats, weight loss, numbness or tingling in your hands and feet, muscle weakness, personality changes, unsteady movements, mental confusion or forgetfulness.

Many people will have symptoms of hypohomocysteinemia as homocystein builds up due to the broken methyl cycle, these include: seizures, psychiatric issues, eye abnormalities, vascular issues. Many people will also have symptoms of glutathione deficiency including slowing down of physical reactions, speech (psychomotor retardation), intellectual disability and a loss of coordination (ataxia). The methyl trap will also cause issues with gene expression, epigenetic changes and demyelination of nerves.

The broken methylation cycle can present with histadelia symptoms (high histamine) such as: muscle pain, headaches, fatigue, insomnia, irritability, anxiety, paranoia, being suspicious, and hallucinations. This can also cause issues with sulfur metabolism, leading to the "COVID smell".

There are reports of dramatically high B12 measurements without B12 supplementation and the above paper states, "There may also be evidence of inactive B12 'analogues' in these patients, which may represent endogenous inactive oxidation products". Its possible that B12 analouges are cross reacting with the assay and building up due to oxidative destruction of B12 intermediates. Its also possible that elevated B12 measurements are because the labs use methylmalonic acid as a proxy indicator for B12, and elevated methylmalonic acid would be expected. The <u>use of methyl donors</u> such as trimethylglycine has been known to help in situations of methionine synthase deficiency but addressing the core issues will help the body leave this metabolic trap.

A urinary organic acids panel may be very relevant to run as changes in homocysteine, histamine, kyneurien metabolites and other markers out of range would be expected due to both of the metabolic traps.

3.7. Vaccine efficiency versus general acquired immune deficiency

There is a second article I would like to quote in its entirety. For two reasons: it mostly displays official data and only a little discussion of the subject, and it is possible that the platform this article was published on will vanish from the internet soon. The data themselves will be still available, most of them come from the UK Health Security Agency 'Vaccine Surveillance' reports on Covid-19 cases. It is named: *UK Government reports suggest the Fully Vaccinated are rapidly developing Acquired Immunodeficiency Syndrome, and the Immune System decline has now begun in Children*, and it was published by The Expose on October the 30th 2021.

The eight previous Public Health England / UK Health Security Agency 'Vaccine Surveillance' reports on Covid-19 cases show that double vaccinated 40-79 year olds have now lost lost 50% of their immune system capability and are consistently losing a further 4-5% every week (between 3.7% and 7.9%).

Projections also now show that 30-49 year olds will have zero Covid / viral defence at best, or a form of vaccine mediated acquired immunodeficiency syndrome at worst, by the first week in January and all double vaccinated people over 30 will have completely lost that part of their immune system which deals with Covid-19 in the next 18 weeks.



The Vaccine Surveillance reports published by the UK Health Security Agency (previously Public Health England) of all fully genome sequenced UK Delta Covid-19 cases (*mainly using a genome identifying PCR test*), clearly show the progressive damage that the vaccines are doing to the immune response of the fully vaccinated.

Here is the weekly decline in double vaccinated immune system performance compared to unvaccinated people.

Vaccine efficacy is measured using Pfizer's vaccine effectiveness formula...

Unvaccinated case rate per 100k - Fully Vaccinated case rate per 100k / the Larger of Unvaccinated or Vaccinated case rate

We are using the normalised absolute ratio of vaccinated to unvaccinated case numbers to determine vaccine effectiveness just as Pfizer itself does.

	Week35 Vaccine Efficacy		Week37 Decline	Week38 Decline	Week39 Decline	Week40 Decline	Week41 Decline		Week42 Vaccine Efficacy		Weeks from week42 (October18-24) before total immune system failure (100% degradation)
CHILDREN	+60.1%	+6.4%	+11.6%	+5.7%	+4.3%	+1.6%	0%	- 8.2 %	+81.4%	-4.1% from peak	45 weeks (181.4/4.1)
18-29	+53.2%	-2.5%	-1.9%	-4.0%	-4.3%	-7.0%	-8.6%	-4.0%	+20.9%	-4.6%	27 weeks (120.9/4.6)
30-39	+31.6%	-6.0%	-7.0%	-10.5%	-11.4%	-10.5%	-7.7%	-2.2%	-23.7%	- 7.9 %	10 weeks (76.3/7.9)
40-49	-21.1%	-5.2%	-5.3%	-8.1%	-6.9%	-5.6%	-3.2%	-1.6%	-56.9%	-5.1%	9 weeks (43.1/5.1)
50-59	-24.1%	-4.0%	-2.4%	-3.9%	-5.7%	-5.7%	-5.0%	-2.3%	-53.1%	-4.1%	12 weeks (46.9/4.1)
60-69	-27.5%	-4.2%	-2.9%	-4.2%	-2.1%	-5.8%	-4.0%	-2.5%	-53.2%	-3.7%	13 weeks (46.8/3.7)
70-79	-23.5%	-4.1%	+0.7%	-3.9%	-3.6%	-9.6%	-6.3%	-0.6%	-50.9%	-3.9%	13 weeks (49.1/3.9)
80+	+8.5%	-5.6%	-7.1%	-3.1%	-2.5%	-8.3%	-7.1%	-2.4%	-22.8%	-4.5%	18 weeks (77.2/4.5)
18+	-0.4%	-4.5%	-3.7%	-5.4%	-5.2%	-7.5%	-6.0%	-1.5%	-34.2%	-4.8%	14.6 weeks

A vaccine effectiveness of +50% means that double vaccinated people are 50% more protected from Covid than unvaccinated people. It means that the Delta case rate in the vaccinated is half the delta case rate in the unvaccinated.

A Vaccine efficacy of -50% means that unvaccinated people are 50% more protected from Covid than doubly vaccinated people. It means that the delta case rate in the vaccinated is double the Delta case rate in the unvaccinated.

A Vaccine efficacy of 0% means that doubly vaccinated people are 0% more protected from Covid than unvaccinated people. It means that the delta case rate in the vaccinated equals the Delta case rate in the unvaccinated. It means the vaccines have lost all their effectiveness.

Age group	Week35 Vax Efficacy	Week36 Vax Efficacy	Week37 Vax Efficacy	Week38 Vax Efficacy	Week39 Vax Efficacy	Week40 Vax Efficacy	Week41 Vax Efficacy	Week42 Vax Efficacy
	+60.1%		+78.0%	+83.7%	+88.0%	+89.6%	+89.6%	+81.4%
18-29	+53.2%	+50.7%	+48.8%	+44.8%	+40.5%	+33.5%	+24.9%	+20.9
30-39	+31.6%	+25.6%	+18.6%	+8.1%	-3.3%	-13.8%	-21.5%	-23.7%
40-49	-21.1%	-26.3%	-31.6%	-39.7%	-46.6%	-52.2%	-55.4%	-56.9%
50-59	-24.1%	-28.1%	-30.5%	-34.4%	-40.1%	-45.8%	-50.8%	-53.1%
60-69	-27.5%	-31.7%	-34.6%	-38.8%	-40.9%	-46.7%	-50.7%	-53.2%
70-79	-23.5%	-27.6%	-26.9%	-30.8%	-34.4%	-44.0%	-50.3%	-50.9%
80+	+8.5%	+2.9%	-4.2%	-7.3%	-9.8%	-18.1%	-25.2%	-22.8%
18+	-0.4%	-4.9%	-8.6%	-14.0%	-19.2%	-26.7%	-32.7%	-34.2%



These new figures show a slowing down in degradation last week which is good news. But they also show children are beginning to suffer immune system degradation which is despicable news. Their figures went up due to more and more 12-15 year olds being included in the cohort since Chris Whitty overruled the Joint Committee on Vaccination and Immunization. They have now just about finished being included. So, the slow degradation can now begin to be seen.

At the other end of the age scale, the 80+ group saw a major improvement, due to the boosters which will buy them a couple of months of improved Covid immunity which will hide the continuing gradual immune system degradation. These boosters are the same as the original vaccinations (because no other shot has yet been approved).

So the worry is that whilst Covid-19 immunity may be improved for a couple of months, their general immunity will begin to degrade even faster than it would have done had the booster not been taken. We shall see if that turns out to be the case from future data.

The 70-79 year olds are also seeing a slowdown in apparent degradation due to the boosters improving their Covid response. It may also be the case that other age group are already taking boosters. There is no shortage of places who will vaccinate people on demand without offering the

necessary information for giving an informed consent. The latest clinical information from Dr Richard Fleming detailed below is terminal for vaccines and boosters.

The risk benefit analysis for these vaccines has now become a risk detriment analysis for everyone over 30.

Latest Projections

Everybody over 30 will have lost 100% of their entire immune capability (certainly for Covid and most likely for viruses and certain cancers – following the evidence from Cole Diagnostics in Idaho and Dr Nathan Thompson and Dr Ralph Baric) within 18 weeks.

Fully vaccinated 30-49 year olds will have lost it by the 1st week in January. These people will then have no immune defense against Covid-19 at all. The question then becomes how much of the immune system is involved in defending against Covid-19? The worst-case scenario is that they effectively develop full blown acquired immunodeficiency syndrome and destroy the NHS.

"In individuals aged greater than 30, the rate of positive COVID-19 test is higher in vaccinated individuals compared to unvaccinated". – <u>PHE Vaccine Surveillance Report for week 41.</u>

"There is the potential for ADE, but the bigger problem is probably Th2 immunopathology," says Ralph Baric, an epidemiologist and expert in coronaviruses—named for the crown-shaped spike they use to enter human cells—at the University of North Carolina at Chapel Hill.

In previous studies of SARS, aged mice were found to have particularly high risks of <u>life-threatening</u> <u>Th2 immunopathology in which a faulty T cell response triggers allergic inflammation, and poorly</u> <u>functional antibodies that form immune complexes, activating the complement system and potentially</u> <u>damaging the airways.</u>

Baric expresses his concern about what that might mean for use of a COVID-19 vaccine in elderly people. "Of course, the elderly are our most vulnerable population," he adds. – <u>https://www.pnas.org/content/117/15/8218</u> (the Proceedings of the National Academy of Sciences of the USA)

The underlined passage (which has been redacted from the online PNAS report – but is in many other online copies – <u>https://principia-scientific.com/study-covid-19-vaccine-can-destroy-your-immune-system/</u>) is critical as it relates to an immune deficiency in killer T cells.

This was seen by Dr Ryan Cole who has done over 100,000 pathology lab examinations from Covid patients. He identified it as a form of AIDS (reverse HIV he called it – where you lose CD8 killer T cells rather than CD4 Helper T cells). Ralph Baric should know. In 2002 on April 19, the University of North Carolina filed <u>US patent 7279327</u> for an infectious replication defective coronavirus (to be used as a virus vector for an HIV vaccine), claiming priority from US28531801P. Inventors were: Kristopher M. Curtis, Boyd Yount, Ralph S. Baric.

These immune system degradations could be caused by ADE (Antibody Dependent Enhancement – where the vaccine induced antibodies start working in reverse) and be specific to Covid, or could be more general and result in a form of vaccine mediated AIDS (Acquired Immunodeficiency Syndrome). Baric suspects the latter above.

The fact that the 3rd Jabs worked in Israel (for a short period of time before the 4th jabs were proposed) means that vaccine antibodies do still have a protective effect immediately after vaccination. So that may rule out ADE. The latest figures suggest that boosters are working, in the short term for 70-79-year-olds and 8the 80+.

The falling efficacy of the vaccines does not asymptotically approach zero (which would mean that vaccines merely lose effectiveness over time). It goes straight through zero and then goes dangerously negative (which means the vaccines become toxic to the immune system). Then it goes more negative in a linear manner week on week. If this continues then the vaccines will completely destroy the part of your immune system which deals with Covid by the end of January.

This may well result in more cases of Shingles, HPV, Herpes, Epstein Barr, Endometriosis and other viral infections – <u>https://www.nbc12.com/2021/10/15/reports-shingles-outbreaks-not-directly-linked-covid-19-vaccine/</u>

(...)

The immune system boost or degradation column, which is the vaccine efficiency/inefficiency column, column10, is calculated from Pfizer's vaccine efficiency formula of -

U-V/U for U>V and U-V/V for V>U

This is the formula they used to claim 95% vaccine efficiency against Wuhan alpha.

For previous versions of Table 2 from Week 32 onwards see here.

Cases reported by specimen date between week 37 & 40 2021 – https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/10 25358/Vaccine-surveillance-report-week-41.pdf

Age group	Total Cases	Vax status unknown	1 dose 1-20 days before specimen date	1 dose ≥21 days before specimen date	2nd dose ≥14 days before specimen date: double vaxxed	Unvaccinated cases	Rates per 100k in double vaxxed (V)	Rates per 100k in unvaxxed (U)	Immune system boost or degradation % (U-V)/U when positive (pfizer's formula) (U-V)/V when negative	Weekly Decline
Under 18	348,514	22,301	6,396	7,964	654	311,199	276.5	2,670.7	+89.6% (includes 180k 12-15 cases)	+1.6%
18-29	60,057	7,683	837	8,937	22,053	20,547	402.6	605.0	+33.5%	-7.0%
30-39	83,007	7,138	626	6,479	48,232	20,532	823.9	709.8	-13.8%	-10.5%
40-49	111,896	6,778	292	3,551	89,546	11,729	1,455.8	696.2	-52.2%	-5.6%
50-59	74,981	4,506	85	1,463	63,929	4,998	903.1	489.3	-45.8%	-5.7%
60-69	38,184	2,455	24	525	33,486	1,694	589.0	314.1	-46.7%	-5.8%
70-79	23,109	1,363	7	201	20,916	622	451.5	253.0	-44.0%	-9.6%
80+	10,770	839	7	184	9,365	375	364.6	298.5	-18.1%	-8.3%

Cases reported by specimen date between week 38 & 41 2021 – https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/10 27511/Vaccine-surveillance-report-week-42.pdf

Age group	Total Cases	Vax status unknown	1 dose 1-20 days before specimen date	1 dose ≥21 days before specimen date	2nd dose ≥14 days before specimen date: double vaxxed	Unvaccinated cases	Rates per 100k in double vaxxed (V)	Rates per 100k in unvaxxed (U)	Immune system boost or degradation % (U-V)/U when positive (pfizer's formula) (U-V)/V when negative	Weekly Decline
Under 18	397,882	24,292	10,698	11,001	743	351,148	314.1	3,013.6	+89.6% (includes 230k 12-15 cases)	0%
18-29	62,885	7,512	758	8,404	25,309	20,902	462.1	615.4	+24.9% (615.4-462.1)/615.4 as %	-8.6%
30-39	92,257	7,346	636	6,545	56,004	21,726	956.7	751.1	-21.5% (956.7-751.1)/956.7 as %	-7.7%

Cases reported by specimen date between week 39 & 42 2021 – https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/10 29606/Vaccine-surveillance-report-week-43.pdf

Age group	Total Cases	Vax status unknown	1 dose 1-20 days before specimen date	1 dose ≥21 days before specimen date	2nd dose ≥14 days before specimen date: double vaxxed	Unvaccinated cases	Rates per 100k in double vaxxed (V)	Rates per 100k in unvaxxed (U)	Immune system boost or degradation % (U-V)/U when positive (pfizer's formula) (U-V)/V when negative	Weekly Decline
Under 18	411,079	24,798	16,640	13,812	821	355,008	586.2	3,149.6	+81.4% (includes 240k 12-15 cases)	-8.2%
18-29	68,780	7,713	686	8,532	29,413	22,436	532.9	674.0	+20.9% (674.0-532.9)/674.0 as %	-4.0%
30-39	102,344	7,858	645	6,856	63,237	23,748	1,071.8	817.7	-23.7% (1071.8-817.7)/1071.8 as %	-2.2%
40-49	145,641	7,989	291	3,962	119,063	14,336	1,936.2	834.9	-56.9% (1936.2-834.9)/1936.2 as %	-1.5%
50-59	102,009	5,330	81	1,767	88,740	6,091	1,248.7	586.1	-53.1% (1248.7-586.1)/1248.7 as %	-2.3%
60-69	54,020	2,968	22	702	48,161	2,167	836.6	391.2	-53.2% (836.6-391.2)/836.6 as %	-2.5%
70-79	32,909	1,822	14	254	30,025	794	635.4	312.2	-50.9% (635.4-312.2)/635.4 as %	-0.6%
80+	13,231	936	7	219	11,635	434	432.5	333.8	-22.8% (432.5-333.8)/432.5 as %	+2.4%

Choosing your formula...

Age group	Rates per 100k in double vaxxed (V)	Rates per 100k in unvaxxed (U)	Immune system boost or degradation % (U-V)/U when positive (pfizer's formula) (U-V)/V when negative	Vaccine efficacy % for double vaxxed compared to unvaxxed (U-V)/U (Pfizer's formula)
Under 18	586.2	3,149.6	+81.4% (includes 240k 12-15 cases)	+81.4% (includes 240k 12-15 cases)
18-29	532.9	674.0	+20.9% (674.0-532.9)/674.0 as %	+20.9% (674.0-532.9)/674.0 as %
30-39	1,071.8	817.7	-23.7% (1071.8-817.7)/1071.8 as %	-31.1% (1071.8-817.7)/817.7 as %
40-49	1,936.2	834.9	-56.9% (1936.2-834.9)/1936.2 as %	-131.9% (1936.2-834.9)/834.9 as %
50-59	1,248.7	586.1	-53.1% (1248.7-586.1)/1248.7 as %	-113.1% (1248.7-586.1)/586.1 as %
60-69	836.6	391.2	-53.2% (836.6-391.2)/836.6 as %	-113.9% (836.6-391.2)/391.2 as %
70-79	635.4	312.2	-50.9% (635.4-312.2)/635.4 as %	-103.5% (635.4-312.2)/312.2 as %
80+	432.5	333.8	-22.8% (432.5-333.8)/432.5 as %	-29.6% (432.5-333.8)/333.8 as %

The Immune System boost/degradation column is a measure of the boost or damage to your immune system – see report

The Vaccine Effectiveness % for double vaccinated column shows how much more or less resistant to Covid the double vaccinated are than the unvaccinated - see report

So if you are 40 years old and double vaxxed then your immune response is now degraded by 56.9%. This means that unvaccinated 40 year olds are 56.9% less likely to catch covid than the fully vaccinated, and the fully vaccinated 40 year olds are 131.9% more likely to catch covid than the unvaccinated.

You can look at it either way. It just depends whether your chosen parameter is the double vaccinated or the unvaccinated. But whichever one you choose, the outlook this winter for those who have been fully vaccinated with the experimental Covid-19 injections looks terrible.

PHE death rates verses ONS death rates

There is a massive immunological contradiction between the PHE case rates which crudely speaking now show that the vaccinated are twice as likely to be infected as unvaccinated whereas the PHE death rates show that the unvaccinated are 3 to 6 times more likely to die than the vaccinated.

The contradiction arises because the immunological functionality of the vaccines is merely to increase or decrease the viral load, but we now know that the viral loads are the same in the vaccinated as in the unvaccinated (*see here*).

This was also confirmed by Dr Fauci earlier this year -

"What we learned that's new ... is that when you look at the level of virus in the nasopharynx of people who are vaccinated who get breakthrough infections, it's really quite high and equivalent to the level of virus in the nasopharynx of unvaccinated people who get infected," Fauci said in an interview with CBS News' "Face the Nation" on Sunday. The nasopharynx is part of the nasal cavity near the back of the throat https://www.theepochtimes.com/fauci-amount-of-covid-19-in-breakthrough-delta-cases-almost-identical-to-unvaccinated 3929532.html

Furthermore it is known that the vaccines have been becoming progressively less effective/more damaging since then. So what is happening with case numbers should also be occurring with deaths unless the vaccines only kick in at near lethal viral loads. But that is nonsense because they merely train the immune system to recognise a new antigen. They should be altering the manner in which it responds upon recognition. Vaccines do not do that.

Although of course these gene therapies are a lot more than vaccines. So from Fauci's own mouth the death should be following the case numbers not going in the other direction. Fortunately the ONS (Office of National Statistics) produce overall mortality figures and Profs Norman Fenton and Martin Neil of Queen Mary College University of London have analysed these as follows –



Week 26 was 2021 June 28th -July 4th. Below is the mortality rate for those who had received two doses of a Covid-19 vaccine for the first half of 2021 -



Since 19th March the double dose vaccination mortality rate has increased week-on-week more or less consistently. – Prof Norman Fenton – <u>https://www.normanfenton.com/post/comparing-age-adjusted-all-cause-mortality-rates-in-england-between-vaccinated-and-unvaccinated</u>

So the ONS death rates as analysed by Prof Fenton are consistent with the PHE case rates as analysed in the article. The PHE death rates are not credible. So the ONS death rates show the same linear increase as the PHE case numbers are showing. That makes a lot more sense immunologically.

The effect of the Pfizer Vaccine on the blood

Dr Richard Fleming teamed up with Prof Luc Montagnier, who won the Nobel prize for discovering HIV, and filed a case in the Hague along with Holocaust survivors on breaches of the Nuremberg Code by Government worldwide forcing vaccines on their citizens <u>(see here)</u>.

Then he had the simple idea (which no one else appears to have thought of) to look at blood samples under the microscope and then add the Pfizer Vaccine.

The vaccine destroys all the Haemoglobin in the blood and makes the red cells stick together and form clots. It is amazing – the blood just stops being red. That is why people cannot breathe. Watch minute 9 to minute 20 of this video and you will see precisely what the vaccines do to human blood.

When you are jabbed, the vaccine goes into your deltoid muscle hopefully (they are supposed to withdraw the plunger slightly to make sure they have not hit a blood vessel – but they are not generally doing that now). If it hits a blood vessel then what you see in the video happens immediately

If the jab does hit the muscle then the above happens more slowly.



The lighter areas on the left and right pictures are where a drop of the Pfizer vaccine has hit the blood on the 40x microscope slide. You can see that the red colour is missing from the red cells in the vaccine droplet areas. This is the destruction of your haemoglobin by the vaccine. It occurs within seconds according to the video.

If you have no haemoglobin, then your blood cannot carry any oxygen to your tissues and your breathing fails. The video also shows that the haemoglobin depleted red blood cells start to stick together. They start to clot within minutes of the vaccine mixing with the blood, destroying the blood's ability to carry oxygen.¹

¹ UK Government reports suggest the Fully Vaccinated are rapidly developing Acquired Immunodeficiency Syndrome, and the Immune System decline has now begun in Children. The Expose, online October 30, 2021 at https://www.jbc.org/article/S0021-9258(18)82653-9/pdf.

3.8. **DNA Repair inhibition by the spike protein**

Slowly, there are symptoms becoming visible that demand a proper explanation. Cancer clinics report that fully healed patients relapse after being vaccinated, with accelerated tumor growth. Covid patients, even after full recovery, seem to age quicker.

There is one single study out there, that might deliver an explanation to these observations. Even though the observations are still "soft", the study itself delivers some undeniable results that should be looked at. I would like to cite the most important passages and figures out of this study:

Abstract: Severe acute respiratory syndrome coronavirus 2 (SARS–CoV–2) has led to the coronavirus disease 2019 (COVID–19) pandemic, severely affecting public health and the global economy.

Adaptive immunity plays a crucial role in fighting against SARS–CoV–2 infection and directly influences the clinical outcomes of patients. Clinical studies have indicated that patients with severe

COVID-19 exhibit delayed and weak adaptive immune responses; however, the mechanism by which SARS-CoV-2 impedes adaptive immunity remains unclear. Here, by using an in vitro cell line,

we report that the SARS–CoV–2 spike protein significantly inhibits DNA damage repair, which is required for effective V(D)J recombination in adaptive immunity. Mechanistically, we found that the spike protein localizes in the nucleus and inhibits DNA damage repair by impeding key DNA repair protein BRCA1 and 53BP1 recruitment to the damage site. Our findings reveal a potential molecular mechanism by which the spike protein might impede adaptive immunity and underscore the potential side effects of full-length spike-based vaccines.

(...)

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(...)



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Figure 1. Effect of severe acute respiratory syndrome coronavirus 2 (SARS–CoV–2) nuclear-localized proteins on DNA damage repair. (**A**) Subcellular distribution of the SARS–CoV–2 proteins. Immunofluorescence was performed at 24 h after transfection of the plasmid expressing the viral proteins into HEK293T cells. Scale bar: 10 µm. (**B**) Schematic of the EJ5-GFP reporter used to monitor non-homologous end joining (NHEJ). (**C**) Effect of empty vector (E.V) and SARS–CoV–2 proteins on NHEJ DNA repair. The values represent the mean \pm standard deviation (SD) from three independent experiments (see representative FACS plots in Figure S2A). (**D**) Schematic of the DR-GFP reporter used to monitor homologous recombination (HR). (**E**) Effect of E.V and SARS–CoV–2 proteins on HR DNA repair. The values represent the mean \pm SD from three independent experiments (see representative FACS plots in Figure S2B). The values represent the mean \pm SD, n = 3. Statistical significance was determined using one-way analysis of variance (ANOVA) in (**C**,**E**). ** p < 0.01, *** p < 0.001.



Figure 2. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) spike protein inhibits DNA damage repair. (A) Schematic of the primary structure of the SARS-CoV-2 spike protein. The S1 subunit includes an N-terminal domain (NTD, 14-305 residues) and a receptor-binding domain (RBD, 319-541 residues). The S2 subunit consists of the fusion peptide (FP, 788-806 residues), heptapeptide repeat sequence 1 (HR1, 912-984 residues), HR2 (1163-1213 residues), TM domain (TM, 1213–1237 residues), and cytoplasm domain (CT,1237–1273 residues). (B,C) Effect of titrated expression of the spike protein on DNA repair in HEK-293T cells. (D,E) Only full-length spike protein inhibits non-homologous end joining (NHEJ) and homologous recombination (HR) DNA repair. The values represent the mean \pm SD from three independent experiments (see representative FACS plots in Figure S4A,B). (F) Full-length spike (S-FL) protein-transfected HEK293T cells exhibited more DNA damage than empty vector-, S1-, and S2-transfected cells under different DNA damage conditions. For doxorubicin: $4 \mu g/mL$, 2 h. For γ -irradiation: 10 Gy, 30 min. For H₂O₂: 100 μ M, 1 h. Scale bar: 50 µm. (G) Corresponding quantification of the comet tail moments from 20 different fields with n > 200 comets of three independent experiments. Statistical significance was assessed using a two-way analysis of variance (ANOVA). NS (Not Significant): * p > 0.05, ** p < 0.01, *** p < 0.001, **** *p* < 0.0001.

4. Discussion of the data - Miasma Theory of Disease, Lock N Load Theory of Bioenergetic Disease Transfer: Trace Amine Receptors (lock) and Thyroid (load)

Recently discovered bio-photonic mechanisms of the thyroid, the trace amine chemoreceptors in the nose and metabolic changes due to COVID-19 have indicated a core mechanism for disease transfer which is highly leveraged by COVID-19 disease. It may be possible to stop pathogenic disease transfer from human to human by addressing it. The mechanism appears to be an energetic "lock on" utilising Trace Amine Receptors and a "loading" of bio-photonic information using the thyroid. The thyroid seems to be a bio-photonic antenna which synchronises bio-photonic transfer on vocal sound waves among other functions. Newly discovered chemoreceptors (Trace Amine Receptors) in the nose may be the "targeting" mechanism of this bio-photonic antenna, allowing it to "lock on" to particularly energetic targets and "download" information, including disease information which may be the core mechanism of disease transfer.

This theory offers insights, for example, the first symptom of most viral infections is often a sore throat, potentially a bioenergetic stress on the thyroid. This may explain the behaviours of dogs smelling each-others anuses on greeting as the skatole compound in the anus will trigger the trace amine receptors allowing energetic "download". This may also explain the strong drive for humans to consume pepper with meals, as the piperine in the pepper would activate the same receptors, allowing the smell and taste of the food to allow the perception of the energetic component associated with the smell. This could also indicate the mechanism of "Thieves Oil" an essential oil mix which was rumoured to be coated on rags so thieves could rob the grades of those dead with bubonic plague without contracting the disease, the strong notes for the cinnamon and citrus flavours may have overwhelmed the trace amine receptors, preventing disease energetic download. It could also validate the approach of the bird beak style "plague doctor" masks used, which were filled with fragrant herbs such as garlic and others to prevent disease.

4.1. Thyroid, Biophysics of Disease Transfer (Load)

The thyroid has many small compartments full of "colloid" which is made up of proteins containing iodine, more than 95% are cross linked into a giant polymer dispersed into a colloid². This area has a huge amount of enzymes and nobody knows what they are there for. It seems like the enzymes can carefully tune this giant morphing polymer and studies have shown it dynamically changing colours and optical characteristics depending on the enzyme's activities³. This polymer is also highly optically absorbent across a lot of frequency range including visual frequencies.

The thyroid polymer shifts in thickness dynamically due to enzyme activities and during

² The thyroid has many small compartments full of "colloid" which is made up of proteins containing iodine, more than 95% are cross linked into a giant polymer dispersed into a colloid.

³ EDUARDO DE ROBERTIS, W. W. NOWINSKI, THE PROTEOLYTIC ACTIVITY OF NORMAL AND PATHOLOGICAL HUMAN THYROID TISSUE, *The Journal of Clinical Endocrinology & Metabolism*, Volume 6, Issue 3, 1 March 1946, Pages 235–246, https://doi.org/10.1210/jcem-6-3-235

activation or deactivation⁴, indicating the polymer dynamics are a critical control system. This compartment full of morphing polymer is surrounded by cilia or fibers that move and transmit information⁵, this puzzles scientists because they should only exist in organelles that secrete materials through them, they are clearly transferring information.

In this follicle polymer it is very common to have bifringent calcium crystals that interact uniquely with polarised light. Scientists can't understand how this type of crystal forms and believe it's somehow stabilised by the polymer. These types of crystals have strange bio-photonic properties and are involved in exotic physics. The polymer also contains a unique form of⁶ calcium oxalate crystals which are not found in any other human tissues⁷. Unlike most biological calcifications which happen in unhealthy tissue these crystals are way more present in healthy tissue. Crystals also form in rosette and other formations inside of this colloid. When they are looked at under ultrasound they start reverberating and causing "comet tail" artifices which show they have parallel fine gap structures and are resonant cavities⁸.

Iodine already has a huge nuclear cross section which gives it unique properties for absorbing radiation and exotic physics. Large surface area interfaces like colloids have unique biophysics properties and are highly tuneable, the fact that it's made from giant cross-linked proteins full of iodine and tuned by many enzymes is just mind blowing. This living polymer is full of many different types of crystals and interfaces to the nerves by cilia. Bifringent crystals also participate in exotic biophysics.

4.1.1. Thyroid and environmental stress

Studies on geomagnetic radiation found that it was a risk factor for thyroid cancers and other thyroid dysfunction⁹. Low frequency RF radiation was found to strongly disrupt the thyroid and induce hypothyroidism¹⁰. Birth month is strongly correlated to thyroid disease state later in life, with all relationships having a biyearly structure, indicating geomagnetic conditions during thyroid develop affect function¹¹. Similar yearly oscillations have been found

^{4 &}lt;sup>®</sup>M. D. LEVINE: MUCOLYTIC ACTIVITY OF THE THYROID GLAND in Journal of Endocrinology. DOI: https://doi.org/10.1677/joe.0.0060288 Volume 6: Issue 3

⁵ This compartment full of morphing polymer is surrounded by cilia or fibers that move and transmit information

^{6 &}lt;sup>®</sup>Katoh R, Kawaoi A, Muramatsu A, Hemmi A, Suzuki K. Birefringent (calcium oxalate) crystals in thyroid diseases. A clinicopathological study with possible implications for differential diagnosis. The American Journal of Surgical Pathology. 1993 Jul;17(7):698-705. DOI: 10.1097/00000478-199307000-00007. PMID: 8317610.

^{7 &}lt;sup>a</sup>calcium oxalate crystals which are not found in any other human tissues.

⁸ Men they are looked at under ultrasound they start reverberating and causing "comet tail" artifices which show they have parallel fine gap structures and are resonant cavities.

^{9 &}lt;sup>®</sup>Grigoryev P.Ye., Vaiserman A.M., Mekhova L.V., Bolgov M.Y., Bezrukov O.F. GEOMAGNETIC ACTIVITY AS RISK FACTOR OF THYROID CANCER (HYPOTHESES, ESTIMATION, SUBSTANTIATION). Original research №12, 2 2010.

¹⁰ Esmekaya, Meric & Seyhan, Nesrin & Ömeroğlu, Suna. (2010). Pulse modulated 900 MHz radiation induces hypothyroidism and apoptosis in thyroid cells: A light, electron microscopy and immunohistochemical study. International journal of radiation biology. 86. 1106-16. 10.3109/09553002.2010.502960.

^{11 &}lt;sup>®</sup>Thvilum M, Brandt F, Brix TH, Hegedüs L. Month of birth is associated with the subsequent diagnosis of autoimmune hypothyroidism. A nationwide Danish register-based study. Clin Endocrinol (Oxf). 2017

correlated with COVID-19 cases, indicating a potential geophysical driver¹². The dynamic pattern could be modelled by using 3 simple sine waves – one per each region. Each sine wave peaks twice a year. The thyroid seems to be highly involved in the early stage of most viral infections¹³, yet only small amounts of viral material are found at best and no direct viral engagement mechanisms of the thyroid are understood.

4.1.2. Thyroid and COVID-19

According to the Butterfly-Method, a functional medicine framework for treating long covid, "A review of the overlap between thyroid disorders and COVID19 discusses the many common morbidities and symptoms¹⁴. A detailed review of COVID19 and the thyroid looks at a number of potential mechanisms as well as reviews the clinical literature¹⁵. Thyroid dysfunction was linked to long term anosmia or loss of sense of smell, after COVID19 with more than 50% of patients having long term anosmia also having hypothyroidism¹⁶. This is very relevant as a risk factor as 10% of the population has some type of thyroid dysfunction¹⁷ and more likely much more due to the complexities of testing thyroid function. Another review looks at clinical data and mechanisms around SARS-CoV-2 and the thyroid¹⁸.

One study found higher SARS-CoV-2 viral load correlated to smaller thyroid volume. In one study, more than half of hospitalized COVID-19 patients had <u>abnormal thyroid biomarkers¹⁹</u> during hospitalization that normalized during recovery. Another study in China found that around 62% of COVID-19 patients had thyroid abnormalities²⁰.

Mechanisms of loss of smell such as oxidative damage and direct viral invasion of the olfactory bulb could explain anosmia, but it cannot explain the phantom smells and occasional increases in sense of smell after COVID19 infection. These things can only be explained easily by thyroid dysfunction which often manifests as complex changes in taste and smell perception as well as changes in hearing such as tinnitus²¹. Studies indicate that thyroid

Dec;87(6):832-837. doi: 10.1111/cen.13425. Epub 2017 Aug 22. PMID: 28727153.

¹² Similar yearly oscillations have been found correlated with COVID-19 cases, indicating a potential geophysical driver.

¹³ The thyroid seems to be highly involved in the early stage of most viral infections.

^{14 &}lt;sup>®</sup>Kanchan Kumari, Gagan B.N. Chainy, Umakanta Subudhi: Prospective role of thyroid disorders in monitoring COVID-19 pandemic, Heliyon, Volume 6, Issue 12, 2020, e05712, ISSN 2405-8440.

¹⁵ Scappaticcio, L., Pitoia, F., Esposito, K. *et al.* Impact of COVID-19 on the thyroid gland: an update. *Rev Endocr Metab Disord* (2020). https://doi.org/10.1007/s11154-020-09615-z.

^{16 &}lt;sup>®</sup>Tsivgoulis G, Fragkou PC, Karofylakis E, et al: Hypothyroidism is associated with prolonged COVID-19induced anosmia: a case–control study. Journal of Neurology, Neurosurgery & Psychiatry 2021;92:911-912.

¹⁷ Soh SB, Aw TC. Laboratory Testing in Thyroid Conditions - Pitfalls and Clinical Utility. Ann Lab Med. 2019;39(1):3-14. doi:10.3343/alm.2019.39.1.3

^{18 &}lt;sup>®</sup>Wenjie Chen, Yuang Tian, Zhihui Li, Jingqiang Zhu, Tao Wei, Jianyong Lei, Potential Interaction Between SARS-CoV-2 and Thyroid: A Review, *Endocrinology*, Volume 162, Issue 3, March 2021, bqab004, https://doi.org/10.1210/endocr/bqab004

¹⁹ Min Chen, Weibin Zhou, and Weiwei Xu: Thyroid Function Analysis in 50 Patients with COVID-19: A Retrospective Study. Thyroid 2021 31:1, 8-11.

^{20 &}lt;sup>®</sup>Wang W, Su X, Ding Y, et al. Thyroid Function Abnormalities in COVID-19 Patients. *Front Endocrinol (Lausanne)*. 2021;11:623792. Published 2021 Feb 19. doi:10.3389/fendo.2020.623792

triggered tinnitus is caused by disruption of cochlear outer hair cells²² and a review of animal studies indicate mechanisms of disruption²³ and show reversibility with normalization of thyroid function in most cases. A study of 50 patients who had their thyroid function tested during the course of their infection²⁴, 64% had abnormal thyroid function visible on laboratory tests of TSH, TT3 and TT4. A review paper found many studies that linked thyroid hormone dysfunction and COVID-19²⁵.

While the mechanism is most likely not direct viral attack, it is interesting to note that mRNA coding for ACE-2 receptors has been found in the thyroid²⁶. Thyroid dysfunction is common in a huge cross section of viral infections²⁷ and there have been case studies published of COVID-19 causing thyroid dysfunction as well as vaccination for COVID-19²⁸. The number of case studies already published of thyroid dysfunction after vaccination is a strong safety signal, due to the number of cases and the lag time between cases and publication. Additional

^{21 &}lt;sup>®</sup>Irmgard D. Dietzel, Sivaraj Mohanasundaram, Vanessa Niederkinkhaus, Gerd Hoffmann, Jens W. Meyer, Christoph Reiners, Christiana Blasl and Katharina Bohr: Thyroid Hormone Effects on Sensory Perception, Mental Speed, Neuronal Excitability and Ion Channel Regulation. Submitted: November 21st 2011Reviewed: May 13th 2012. Published: July 18th 2012. DOI: 10.5772/48310

²² Ashuma Sachdeva, Veena Singh, Isha Malik, Prasanta Saha Roy, Himanshu Madaan, Rajesh Nair: Association between serum ferritin and thyroid hormone profile in hypothyroidism. International Journal of Medical Science and Public Health | 2015 | Vol 4 | Issue 6 Page 863ff.

²³ Meyerhoff, William L.: Hypothyroidism and the ear: Electrophysiological, morphological, and chemical considerations. The Laryngoscope, VL 89, S19, 0023-852X, online on the 1st of January 2022 at https://doi.org/10.1002/lary.5540891501

²⁴ Min Chen, Weibin Zhou, Weiwei Xu: Thyroid Function Analysis in 50 Patients with COVID-19: A Retrospective Study. 2021 Jan;31(1):8-11. doi: 10.1089/thy.2020.0363. Epub 2020 Jul 10.

^{25 &}lt;sup>®</sup>L. Croce' D. Gangemi, G. Ancona, F. Liboaà, G. Bendotti, L. Minelli, L. Chiovato: The cytokine storm and thyroid hormone changes in COVID- 19. Journal of Endocrinological Investigation https://doi.org/10.1007/s40618-021-01506-7, November 27, 2020 / Accepted: 9 January 2021.

^{26 &}lt;sup>a</sup>Rotondi, M., Coperchini, F., Ricci, G. *et al.* Detection of SARS-COV-2 receptor ACE-2 mRNA in thyroid cells: a clue for COVID-19-related subacute thyroiditis. *J Endocrinol Invest* 44, 1085–1090 (2021). https://doi.org/10.1007/s40618-020-01436-w

²⁷ Desailloud R, Hober D. Viruses and thyroiditis: an update. *Virol J.* 2009;6:5. Published 2009 Jan 12. doi:10.1186/1743-422X-6-5.

²⁸ Oyibo S O (June 29, 2021) Subacute Thyroiditis After Receiving the Adenovirus-Vectored Vaccine for Coronavirus Disease (COVID-19). Cureus 13(6): e16045. doi:10.7759/cureus.16045

case studies linking COVID-19 vaccination and thyroid dysfunction^{29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53}. While analysis of patients hospitalized with COVID-19 did not correlate with active thyroid treatment, studies simply looking at untreated thyroid dysfunction and COVID-19 morbidity found a strong connection⁵⁴. The thyroid function and size are known to correlate with the menstrual cycle, which could indicate why women with long covid tend to have flare ups around their cycle⁵⁵.

A recent paper looking at the metabolic changes after COVID-19 found long term dysregulation of key metabolic ratios including lower glutamine/glutamate ratios, higher kynurenine/tryptophan ratios and higher LDL/HDL ratios⁵⁶. The LDL/HDL ratio changes line

²⁹ Franquemont S, Galvez J. Subacute Thyroiditis After mRNA Vaccine for Covid-19. J Endocr Soc. 2021;5(Suppl 1):A956-A957. Published 2021 May 3. doi:10.1210/jendso/bvab048.1954

^{30 &}lt;sup>a</sup>Das, L., Bhadada, S.K. & Sood, A. Post-COVID-vaccine autoimmune/inflammatory syndrome in response to adjuvants (ASIA syndrome) manifesting as subacute thyroiditis. *J Endocrinol Invest* (2021). https://doi.org/10.1007/s40618-021-01681-7

³¹ Zettinig, G., Krebs, M. Two further cases of Graves' disease following SARS-Cov-2 vaccination. J Endocrinol Invest (2021). https://doi.org/10.1007/s40618-021-01650-0

^{32 &}lt;sup>a</sup>Gowri M. Ratnayake, Dorota Dworakowska, Ashley B. Grossman: Can COVID-19 immunisation cause subacute thyroiditis? CLINICAL ENOCRINOLOGY. First published: 17 July 2021 https://doi.org/10.1111/cen.14555

 ³³ Mungmunpuntipantip, Rujittika; Wiwanitkit, Viroj: Abnormal Thyroid Function Following COVID-19
Vaccination, Indian Journal of Endocrinology and Metabolism: March–April 2021 - Volume 25 - Issue 2 - p
169 doi: 10.4103/ijem.ijem_286_21

^{34 &}lt;sup>®</sup>Vera-Lastra O, Ordinola Navarro A, Cruz Domiguez MP, Medina G, Sánchez Valadez TI, Jara LJ. Two Cases of Graves' Disease Following SARS-CoV-2 Vaccination: An Autoimmune/Inflammatory Syndrome Induced by Adjuvants. Thyroid. 2021 Sep;31(9):1436-1439. doi: 10.1089/thy.2021.0142. Epub 2021 May 3. PMID: 33858208.

^{35 &}lt;sup>®</sup>Olga Vera-Lastra, Alberto Ordinola Navarro, Maria Pilar Cruz Domiguez, Gabriela Medina, Tania Ivonne Sánchez Valadez, and Luis J. Jara: Two Cases of Graves' Disease Following SARS-CoV-2 Vaccination: An Autoimmune/Inflammatory Syndrome Induced by Adjuvants. Thyroid. Sep 2021.1436-1439. http://doi.org/10.1089/thy.2021.0142

^{36 &}lt;sup>®</sup>Sriphrapradang, C. Aggravation of hyperthyroidism after heterologous prime-boost immunization with inactivated and adenovirus-vectored SARS-CoV-2 vaccine in a patient with Graves' disease. *Endocrine* **74**, 226–227 (2021). https://doi.org/10.1007/s12020-021-02879-8

³⁷ Ratnayake GM, Dworakowska D, Grossman AB. Can COVID-19 immunisation cause subacute thyroiditis? [published online ahead of print, 2021 Jul 17]. *Clin Endocrinol (Oxf)*. 2021;10.1111/cen.14555. doi:10.1111/cen.14555

^{38 &}lt;sup>®</sup>Sriphrapradang, C., Shantavasinkul, P.C. Graves' disease following SARS-CoV-2 vaccination. *Endocrine* **74**, 473–474 (2021). https://doi.org/10.1007/s12020-021-02902-y

³⁹ Oyibo SO. Subacute Thyroiditis After Receiving the Adenovirus-Vectored Vaccine for Coronavirus Disease (COVID-19). Cureus. 2021;13(6):e16045. Published 2021 Jun 29. doi:10.7759/cureus.16045

^{40 &}lt;sup>®</sup>Armando Patrizio, Silvia Martina Ferrari, Alessandro Antonelli, Poupak Fallahi: A case of Graves' disease and type 1 diabetes mellitus following SARS-CoV-2 vaccination, Journal of Autoimmunity, Volume 125, 2021, 102738, ISSN 0896-8411, https://doi.org/10.1016/j.jaut.2021.102738.

⁴¹ Melisa Şahin Tekin, Suzan Şaylısoy & Göknur Yorulmaz (2021) Subacute thyroiditis following COVID-19 vaccination in a 67-year-old male patient: a case report, Human Vaccines & Immunotherapeutics, DOI: 10.1080/21645515.2021.1947102

^{42 &}lt;sup>®</sup>di Filippo, L., Castellino, L. & Giustina, A. Occurrence and response to treatment of Graves' disease after COVID vaccination in two male patients. *Endocrine* (2021). https://doi.org/10.1007/s12020-021-02919-3

⁴³ Burçin Gönül İremli, Süleyman Nahit Şendur, Uğur Ünlütürk, Three Cases of Subacute Thyroiditis Following SARS-CoV-2 Vaccine: Postvaccination ASIA Syndrome, *The Journal of Clinical Endocrinology* & *Metabolism*, Volume 106, Issue 9, September 2021, Pages 2600–2605, https://doi.org/10.1210/clinem/dgab373

up with thyroid dysfunction as it is highly involved with lipid metabolism⁵⁷, and a recent study on LDL/HDL ratios found it's an excellent marker for detecting what is called "asymptomatic subclinical hypothyroidism"⁵⁸. While microbial dysfunction could also explain it, the tryptophan pathway and kynurenine metabolism is known to be affected by a dysfunctional thyroid⁵⁹. Finally, the glutamine-glutamate cycle is known to be compromised in hypothyroidism⁶⁰.

All of the long-term metabolic marker shifts detected in these studies are changes expected when the thyroid is dysregulated. The studies show it could be caused by "asymptomatic subclinical hypothyroidism" which challenges our modern medical testing paradigm which relies on biomarkers and symptoms to determine problems in the body.

- 55 Riisfeldt O.: Influence of Thyrotoxicosis on Menstruation. From the Surgical Clinic C of the University, Rigshospitalet, Copenhagen Chief Surgeon: Professor E. Dahl-Iversen M. D. Gynaecologia 1949;128:237– 247 (DOI:10.1159/000312309).
- 56 Elaine Holmes et al: Incomplete Systemic Recovery and Metabolic Phenoreversion in Post-Acute-Phase Nonhospitalized COVID-19 Patients: Implications for Assessment of Post-Acute COVID-19 Syndrome. From the Surgical Clinic C of the University, Rigshospitalet, Copenhagen Chief Surgeon: Professor *E. Dahl-Iversen* M. D.. *J. Proteome Res.* 2021, 20, 6, 3315–3329, May 19, 2021 https://doi.org/10.1021/acs.jproteome.1c00224
- 57 Rizos CV, Elisaf MS, Liberopoulos EN. Effects of thyroid dysfunction on lipid profile. Open Cardiovasc Med J. 2011;5:76-84. doi:10.2174/1874192401105010076

⁴⁴ Chatzi, S., Karampela, A., Spiliopoulou, C. *et al.* Subacute thyroiditis after SARS-CoV-2 vaccination: a report of two sisters and summary of the literature. *Hormones* (2021). https://doi.org/10.1007/s42000-021-00332-z

⁴⁵ Athanasios Siolos, Konstantina Gartzonika, Stelios Tigas: Thyroiditis following vaccination against COVID-19: Report of two cases and review of the literature, Metabolism Open, Volume 12, 2021, 100136, ISSN 2589-9368, https://doi.org/10.1016/j.metop.2021.100136.

⁴⁶ Rubinstein, Tal J.: Thyroid Eye Disease Following COVID-19 Vaccine in a Patient With a History Graves' Disease: A Case Report. Ophthalmic Plastic and Reconstructive Surgery, Volume 37, Number 6, 15 July 2021, pp. e221-e223(3) DOI: https://doi.org/10.1097/IOP.00000000002059

⁴⁷ Patel KR, Cunnane ME, Deschler DG. SARS-CoV-2 vaccine-induced subacute thyroiditis. Am J Otolaryngol. 2022;43(1):103211. doi:10.1016/j.amjoto.2021.103211

⁴⁸ Pierman G, Delgrange E, Jonas C. Recurrence of Graves' Disease (a Th1-type Cytokine Disease) Following SARS-CoV-2 mRNA Vaccine Administration: A Simple Coincidence?. *Eur J Case Rep Intern Med*. 2021;8(9):002807. Published 2021 Sep 2. doi:10.12890/2021_002807

^{49 &}lt;sup>®</sup>Saygılı ES, Karakilic E, Subacute thyroiditis after inactive SARS-CoV-2 vaccine BMJ Case Reports CP 2021;14:e244711.

^{50 &}lt;sup>®</sup>Henrique M. Leber, Leticia Sant'Ana, Nina R. Konichi da Silva, Mariana C. Raio, Thiago Jose Muniz Machado Mazzeo, Camila Matsuura Endo, Heloisa Nascimento & Carlos E. de Souza (2021) Acute Thyroiditis and Bilateral Optic Neuritis following SARS-CoV-2 Vaccination with CoronaVac: A Case Report, Ocular Immunology and Inflammation, DOI: 10.1080/09273948.2021.1961815

^{51 &}lt;sup>®</sup>Yamamoto, K.; Mashiba, T.; Takano, K.; Suzuki, T.; Kami, M.; Takita, M.; Kusumi, E.; Mizuno, Y.; Hamaki, T. A Case of Exacerbation of Subclinical Hyperthyroidism after First Administration of BNT162b2 mRNA COVID-19 Vaccine. *Vaccines* 2021, *9*, 1108. https://doi.org/10.3390/vaccines9101108

⁵² Bornemann C, Woyk K, Bouter C. Case Report: Two Cases of Subacute Thyroiditis Following SARS-CoV-2 Vaccination. *Front Med (Lausanne)*. 2021;8:737142. Published 2021 Aug 24. doi:10.3389/fmed.2021.737142

⁵³ Patel M, Shahid M, Khawaja A, Ejike C, Vemuri K. Subacute Thyroiditis Secondary to Moderna COVID-19. Vaccine: A Case Report of a Rare Manifestation. Advances in Clinical Medical Research and Healthcare Delivery. 2021; 1(2). doi: 10.53785/2769-2779.1019.

^{54 &}lt;sup>®</sup>Abobaker A, Darrat M. The association between biochemically confirmed thyroid gland disorder and morbidity and mortality in patients with COVID-19. *J Med Virol*. 2021;93(12):6449-6450. doi:10.1002/jmv.27213
4.2. Trace Amine Receptors, Biophysics of Disease Transfer (Lock)

The trace amine receptors in the nose are a new class of receptors that have recently been discovered which are specific to indole and amine molecules, which are present associated with feces, putrification, death, etc. These receptors are activated when the smell of feces, deaths, rot, etc are detected by a person, these then seem transfer the energetic blueprint of the source of the smell to "lock on" to the thyroid gland. The thyroid gland shifts its iodine-based polymer/colloid to tune into this source of energetics and download the information.

These trace amine receptors are a different system than normal smell processing, they are not odour receptors, they are chemoreceptors and linked to different parts of the body and brain⁶¹. This newly found chemical sensing system is cross linked into the brain, immune cells, thyroid, GI tract and other critical tissues⁶². Some of these receptors are highly specific to the compounds emitted from decaying corpses and may be chemoreceptors for detecting the smell of death⁶³. Most interestingly, these receptors are found in the thyroid colloid, associated with the cilia, which seem to transfer information to the thyroid polymer⁶⁴. This strengthens the theory that the trace amine receptors hand off bioenergetic targeting information to the thyroid.

⁵⁸ Mahto M, Chakraborthy B, Gowda SH, Kaur H, Vishnoi G, Lali P. Are hsCRP Levels and LDL/HDL Ratio Better and Early Markers to Unmask Onset of Dyslipidemia and Inflammation in Asymptomatic Subclinical Hypothyroidism?. *Indian J Clin Biochem*. 2012;27(3):284-289. doi:10.1007/s12291-012-0206-y

⁵⁹ Hiroshi Okamoto, Fumiko Okada and Osamu Hiyashi: Kynurenine Metan'bolism in Hyperthyrodoism. The Journal of Biological Chemistry. Vo. 246, N0 24, Issue of Dec. 25, pp7759-7763, 1971.

^{60 &}lt;sup>®</sup>Daiane Cattani, Paola Bez Goulart, Vera Lúcia de Liz Oliveira Cavalli, Elisa Winkelmann-Duarte, André Quincozes dos Santos, Paula Pierozan, Daniela Fraga de Souza, Viviane Mara Woehl, Marilda C. Fernandes, Fátima Regina Mena Barreto Silva, Carlos Alberto Gonçalves, Regina Pessoa-Pureur, Ariane Zamoner, Congenital hypothyroidism alters the oxidative status, enzyme activities and morphological parameters in the hippocampus of developing rats, Molecular and Cellular Endocrinology, Volume 375, Issues 1–2, 2013, Pages 14-26, ISSN 0303-7207, https://doi.org/10.1016/j.mce.2013.05.001.

^{61 &}lt;sup>®</sup>Liberles SD, Buck LB. A second class of chemosensory receptors in the olfactory epithelium. Nature. 2006 Aug 10;442(7103):645-50. doi: 10.1038/nature05066. Epub 2006 Jul 30. PMID: 16878137.

^{62 &}lt;sup>a</sup>AUTHOR=Espinoza Stefano, Sukhanov Ilya, Efimova Evgeniya V., Kozlova Alena, Antonova Kristina A., Illiano Placido, Leo Damiana, Merkulyeva Natalia, Kalinina Daria, Musienko Pavel, Rocchi Anna, Mus Liudmila, Sotnikova Tatiana D., Gainetdinov Raul R.: Trace Amine-Associated Receptor 5 Provides Olfactory Input Into Limbic Brain Areas and Modulates Emotional Behaviors and Serotonin Transmission. Frontiers in Molecular Neuroscience. VOL.13, 2020, P18, https://www.frontiersin.org/article/10.3389/fnmol.2020.00018, DOI=10.3389/fnmol.2020.00018, ISSN=1662-5099.

^{63 &}lt;sup>®</sup>Espinoza Stefano, Sukhanov Ilya, Efimova Evgeniya V., Kozlova Alena, Antonova Kristina A., Illiano Placido, Leo Damiana, Merkulyeva Natalia, Kalinina Daria, Musienko Pavel, Rocchi Anna, Mus Liudmila, Sotnikova Tatiana D., Gainetdinov Raul R.: Trace Amine-Associated Receptor 5 Provides Olfactory Input Into Limbic Brain Areas and Modulates Emotional Behaviors and Serotonin Transmission. JOURNAL=Frontiers in Molecular Neuroscience, VOL. 13, 2020, P18, URL=https://www.frontiersin.org/article/10.3389/fnmol.2020.00018 DOI=10.3389/fnmol.2020.00018.

^{64 &}lt;sup>a</sup>Szumska J, Qatato M, Rehders M, Führer D, Biebermann H, Grandy D, K, Köhrle J, Brix K: Trace Amine-Associated Receptor 1 Localization at the Apical Plasma Membrane Domain of Fisher Rat Thyroid Epithelial Cells Is Confined to Cilia. Eur Thyroid J 2015;4(suppl 1):30-41. doi: 10.1159/000434717

4.3. Indole "Hormones" and COVID-19

The indole-based compounds required to trigger the trace amine receptors are highly present due to COVID-19 infection, in fact, metabolomics studies indicate it is one of the most correlated metabolic dysfunctions. Disruption of thryptophan metabolism is one of the clearest signatures of COVID-19. Univariate and multivariate analyses have identified two metabolites that are central in the tryptophan-nicotina⁶⁵ mide pathway. The 3-indole acetic acid is a breakdown product of tryptophan metabolism.

Importantly, pronounced dysregulation was observed in the serum levels of products of tryptophan metabolism, including Indole, 2-hydroxypyridine and Indole-3-acrylic acid⁶⁶. <u>These compounds produced in the body at high levels are disruptive</u> and can contribute to the long term mechanisms of long covid and vaccine damage.

4.4. Blocking Trace Amine Receptors to stop information download

A patent application was filed for antagonizing the TAA receptors to block perception of bad odors⁶⁷. They screened natural products libraries against TAA receptors and found a list of hits, geriniol is the strongest safe natural product and is a major component of citrus, palmarosa, rose oil and citronella oil. palmarosa essential oil has the highest composition of geraniol at 67.6–83.6%.

TAA agonists can be used as the TAA antagonist geriniol only antagnoized the receptor at a rate of 85% or so, this can bind the receptor to prevent indole binding. In the composition thieves oil, the lemon oil component contains Methyl Anthranilate which would bind TAA receptors and could have potentially been the most relevant active ingredient. Indole is present in jasmine oil at high rates (1%) and would bind most directly the TAA receptors.

methyl-N-methyl anthranilate, found in high quantities in Citrus reticulata mandarin essential oil, (around 50% of the total composition) is a secondary amine and will bind the trace amine receptors efficiently.

A combination of palmarosa essential oil, jasmine essential oil and mandarin essential oil might potentially block activation of the trace amine receptors enough to prevent energetic disease transfer.

⁶⁵ Blasco, H., Bessy, C., Plantier, L. *et al.* The specific metabolome profiling of patients infected by SARS-COV-2 supports the key role of tryptophan-nicotinamide pathway and cytosine metabolism. *Sci Rep* 10, 16824 (2020). https://doi.org/10.1038/s41598-020-73966-5

^{66 &}lt;sup>®</sup>Kaur,G.; Ji,X.;Rahman, I. SARS-CoV2 Infection Alters Tryptophan Catabolism and Phospholipid Metabolism. *Metabolites* 2021,*11*,659. https://doi.org/ 10.3390/metabo11100659

⁶⁷ Phttps://patents.google.com/patent/WO2016126903A1/en

5. Gu Syndrome and The Three Corpses, PIEZO1/2 Bioenergetic Field Structure Theory of Pathogen Superinfection

Recently discovered mechanisms of PIEZO1/2 PIEZO ion channel receptors indicate they are a critical aspect of fine-tuning systematic homeostasis, represented in all of the critical machinery of the human body. It is theorized that the PIEZO1/2 ion channel receptors act as antennas, coordinating an energetic field structure of consciousness around the human body, acting as a higher-level control system of body operations and lower level control system of consciousness. The ancient concepts of "Gu Syndrome" and "The Three Corpses" could indicate the observation of this field structure, and its disruption leading to pathogenic superinfection and chronic disease. This may indicate a critical new bioenergetic mechanism explaining Post Viral Syndrome, Long Covid, EDS, Fibromvalgia and the many chronic disease cluster classifications are caused by pathogenic disease, biotoxin exposure or severe trauma. These acute events which would modify the PIEZO ion channel energetic field structure may cause the structure to become decoherent, preventing centralised control and coherence, leading to the semi-permanent takeover of this field structure by energetic consciousness constructs attached to dormant pathogens. This concept of the PIEZO energetic field structure may be represented in Chinese medicine as Qi, and the takeover and decoherence of this field structure resulting in Gu Syndrome/3 corpses may be represented as "pestilent Qi".

Gu syndrome is a systematic disease from ancient Chinese medicine which was removed in the newer iterations due to its magic connections. One of the main modern researchers of Gu Syndrome is Heiner Fruehauf, a leading scholar and Chinese medicine practitioner. His essay, "Driving Out Demons and Snakes: Gu Syndrome, A Forgotten Clinical Approach to Chronic Parasitism"⁶⁸ gives important analysis of this ancient disease. Most interestingly are the symptom clusters of "Gu Syndrome", which overlap perfectly with post-viral diseases and diseases of chronic infection such as Lyme's disease.

Heiner Fruehauf quotes Gu syndrome symptoms as⁶⁹: "Digestive symptoms: Chronic diarrhoea, loose stools or alternating diarrhoea and constipation; explosive bowel movements; abdominal bloating or ascites; abdominal cramping and/or pain; nausea; intestinal bleeding and/or pus; poor appetite or ravenous appetite, peculiar food cravings.

Neuromuscular symptoms: Muscle soreness, muscle heaviness, muscle weakness; wandering body pains; physical heat sensations; cold night sweats; aversion to bright light.

Mental symptoms: Depression, frequent suicidal thoughts; flaring anger, fits of rage; unpredictable onset of strong yet volatile emotions; inner restlessness, insomnia; general sense of muddledness and confusion, chaotic thought patterns; visual and/or auditory hallucinations; epileptic seizures; sensation of "feeling possessed."

Constitutional signs: Progressing state of mental and physical exhaustion, indications of source qi damage; dark circles underneath the eyes; mystery symptoms that evade clear diagnosis; history of acute protozoan infection; history of travel to tropical regions; floating

 ^{68 &}quot;https://classicalchinesemedicine.org/driving-out-demons-snakes-gu-syndrome-chronic-parasitism/
69 "Ebd.

and big pulse or congested (choppy) pulse; stagnation in sublingual veins; rooted damp tongue coating; red tongue tip or red 'parasite dots'* on top of the tongue."

Gu syndrome has important clues to the connection between energetic consciousness field structures including the definition of patients feeling "hollowed out", and the magic connection to the disease state which led to it being removed from modern TCM practices. Even the symbology of the Chinese pictograms representing Gu indicate the stagnation of a hollow cavity. "Since the beginning of Chinese writing approximately 3,500 years ago, it has portrayed either two or three worms squirming in a vessel. In the words of a traditional commentator "Gu is if a cooking vessel remains unused for a long time and worms start to grow in it". This is another hinting indicating the loss or decoherence of an energetic field structure surrounding the human body leading to the overgrowth of pathogens.

Gu syndrome was thought to be triggered by demons who took over the body and caused disease as well as overgrowth of pathogens, specifically parasites. Gu syndrome was thought to be caused by black magic attacks, due to the alterations in consciousness, dark invasive thoughts and other hard to explain manifestations.

Similar to Gu syndrome, in the Daoist traditions there is a similar concept of pathogen superinfection called "The Three Corpses. This describes three parasites which exists at the physical as well as supernatural level, in the head, chest and stomach.

Like Gu syndrome, <u>The Three Corpses</u>, can be treated and expelled using approaches common to most ancient modalities of treating possession and demons. One of the most common threads is Cinnabar, a mineral of mercury and sulphur, which is also known as an elixir of eternal life. Cinnabar in combination with 4 other minerals is known as "Five Stones Powder", a medicine with magical properties. <u>The chapter "Alluring Stimulant" in the Book</u> <u>"Healing with Poisons"</u> goes into detail about the effects and use of this composition, indicating it may be a unique type of bioenergetic treatment which could possibly reignite the coherence of the PIEZO energetic field. A composition of equal parts of finely ground Cinnabar, Realgar, Malachite, Potash Alum and Magnetite seems to be the most likely composition. Most ancient texts describe this composition being rolled into pellets and a daily amount taken between the side of hemp seeds and small beans, indicating a daily dose of around 150mg of the mixed composition.

While modern scholars blame the toxic effects of mercury on the active properties of cinnabar and five stones powder, modern toxicology studies indicate that it is generally inert and safe in the context of mercury toxicity, due to the element being bound in a mineral/crystal structure⁷⁰. Interestingly, while toxicity due to the metallic mercury is not a concern, the compound does seem to cross the blood brain barrier and have effects on the brain after long term use⁷¹. The other component which may be toxic in the five stones powder is Realgar, a mineral made of arsenic. <u>One study states</u>, "for safe use of Realgar, it is recommended that the daily doses of Realgar (with soluble arsenic < or = 1.7 mg x g(-1)) for an adult of the body

⁷⁰ Liu, Jie and Shi, Jing-Zheng and Yu, Li-Mei and Goyer, Robert and Waalkes, Michael: Experimental biology and medicine (Maywood, N.J.), Vol. 233, P. 810–7, 2008.

^{71 &}lt;sup>®</sup>Huang CF, Liu SH, Lin-Shiau SY. Neurotoxicological effects of cinnabar (a Chinese mineral medicine, HgS) in mice. Toxicol Appl Pharmacol. 2007 Oct 15;224(2):192-201. doi: 10.1016/j.taap.2007.07.003. Epub 2007 Jul 14. PMID: 17707451.

weight about 60 kg could be 10-160 mg depending on the variation of the treatment duration.

Based on the toxicity studies, the dose of Five Stones Powder required for short term treatment (maximum 150mg) would not cause acute toxicity due to the suspected toxic mineral components. Both Cinnabar and Realgar daily doses (maximum 30mg) would be within range of the safe threshold, particular if used as a short-term treatment to trigger bioenergetic changes.

The Five Stones Treatment is not without risk as the treatment process must be actively managed by the patient. The traditional use is to take the Five Stones Powder and then exercise or induce high temperatures with thick clothes, most likely to help circulate the inorganic material which is not water soluble. The later stage is to cool the body during the energetic changes, where the name "Cool Food Powder" came from. In most cases, the patient cools his body in cold water baths to sink the internal heat produced in the treatment process. There are many anecdotes of patients suffering negative long-term effects due to improperly managing body temperature through the treatment process. While the acute toxicity may not be a realistic concern, the potential bioenergetic effects improperly managed may be a concern. Compounds such as ginger are often added to these preperations to "circulate Qi" and "cold" foods like mango and cucumber are taken to offset bioenergetic changes.

The structure of possession medicine in most contexts follows a very interesting pattern: There seem to be 3 main therapeutic mechanisms. The first is bioenergetic medicine using minerals such as cinnabar and realgar, components of "five stones powder" which is known to energize the human body and reignite Qi, although are not traditionally bioavailable or relevant in the receptor binding understanding of therapeutics, they are highly active and effective in practice. The second is the use of low doses of toxic plant materials which are usually ion channel blockers with a purgative action, indicating involvement in the enteric nervous system and a modification of the ion channels which are believed to coordinate the bioenergetic field structure. The third is the use of medicines which address the conceptual plane, such as the DNA of centipedes as well as the sap of "powerful trees", which are believed to act as a "predator" of parasitic constructs (demon/worm/parasite constructs natural predator is the centipede) on the conceptual plane once the DNA is ingested and its consciousness field structure integrates with the human body.

The plants used in these possession medicines include aconite and false hellbores, two of the few plants containing sodium channel openers. The formulations also include chinese blistering beetle, which is speculated to contain toxins similar to Grayanotoxins like related species, another of the few natural sodium channel openers. The "Demon Vessel" plant, mayapple, contains compounds known to stun microtubules, which act as biophotonic antennas of conciousness. Croton seed, another highly represented plant, contains compounds which are known to have antinociceptive effects, indicating its action on mechanosensitive ion channels like PIEZO1/2. It is clear that the choices of plants in possession medicine of ancient china is intellegently driven to target modification the biophotonic interface of the human body.

6. Venom as a Vehicle for Implementing Consciousness Constructs, Spike Protein as a Self Replicating Mind Control Envenomation

Recently discovered behavioural pharmacology of venoms has led to more questions than answers. Recently studies on the snake bite and release mechanisms has found that prey envenomated with snake venom are trackable over long distances by the snake, while studies of the disintigrin venom component leading to the tracking ability indicate this compound cannot be shedding from the prey in quantities which would allow for chemical tracking, nor could the bodies breakdown products. The historical use of Tetrodatoxin as a "zombie poison", the "Doom Jellyfish", the miraculous healing properties of Venom medicines like Kambo and the many anomalies in the study of venom hunting and prey behaviours indicate that there is a higher dimensional component to envenomation. Venom appears to be a tool of implementing a consciousness construct on a prey, affecting the PIEZO1/2 energetic field structure and therefore directly affecting the nervous system and consciousness itself and implementing bioenergetic tools and techniques which assist in the survival and hunting behaviours of the source organism. The presence of the unique classes of venom in the blood of COVID-19 patients and the resulting behaviour changes indicate the spike protein is leveraging a number of consciousness constructs borrowed from the animal kingdom to create population level behavioural changes which will lead to its increased fitness, integration with the human population in the long term and control over host behaviour.

6.1. Microbial Integration

According to the Butterfly-Method, a functional medicine framework for treating long covid, "The most interesting research is from a small biotech in Italy who observed patients with COVID19 pathology who were testing negative and found that their fecal samples were positive for the viral RNA⁷², this led them to do some very interesting work. Using their existing biotech capabilities, they saw covid patient samples had peptide toxins similar to venoms present⁷³, and attracted a lot of attention⁷⁴. They had different shuffling of amino acids - indicating they came from bacteria not a virus acting on a human body. They were able to culture sars-cov-2 genetic material on human microbiome cultures, showing the virus seems to grow and express SARS-CoV-2 genetic material⁷⁵. This is a fundamental discovery which connects many dots but is not accepted by the general scientific consensus system. The authors then used a number of antibiotics and showed that they could drop the viral presentation to almost 0, the best antibiotic being azithromycin which might be familiar to many as it was promoted early in the pandemic. While it challenges our understanding of human viruses, research has found that the S1 protein can bind to and engage bacteria present

^{72 &}lt;sup>a</sup>Brogna B, Brogna C, Petrillo M, et al. SARS-CoV-2 Detection in Fecal Sample from a Patient with Typical Findings of COVID-19 Pneumonia on CT but Negative to Multiple SARS-CoV-2 RT-PCR Tests on Oropharyngeal and Nasopharyngeal Swab Samples. *Medicina (Kaunas)*. 2021;57(3):290. Published 2021 Mar 20. doi:10.3390/medicina57030290

 ⁷³ Brogna C, Cristoni S, Petrillo M *et al.* Toxin-like peptides in plasma, urine and faecal samples from COVID-19 patients [version 2; peer review: 2 approved]. *F1000Research* 2021, 10:550 (https://doi.org/10.12688/f1000research.54306.2)

^{74 ®}Edb.

⁷⁵ Petrillo M, Brogna C, Cristoni S, Querci M, Piazza O, Van den Eede G. Increase of SARS-CoV-2 RNA load in faecal samples prompts for rethinking of SARS-CoV-2 biology and COVID-19 epidemiology. *F1000Res*. 2021;10:370. Published 2021 Jul 1. doi:10.12688/f1000research.52540.3

in our microbiome⁷⁶ and validates their hypothesis. This hypothesis could also contribute to the dramatic microbial remodelling that happens during COVID19⁷⁷.

This microbiome viral persistence theory could explain the observations that SARS-Cov-2 fecal tests stay positive much longer than other methods⁷⁸ and those with diarrhea have much worse outcomes⁷⁹. It could also explain the front end of the viral persistence theory, S1 units are showing up in monocytes because they are being produced in the microbiome and transported through the body. This could also explain the fact that 50% of those hospitalized with COVID19 on a ventilator don't even have detectable viral load in the blood but it is detectable in the nasopharyngeal canal. In fact, there is no correlation to circulating viral load and poor outcomes, but with nasal swabs which represent the microbiome it does correlate.

There is an enormous body of literature around the rapid and lasting alterations of the microbiome triggered by COVID-19. A Google Scholar search of "COVID-19 + microbiome" leads to over 36,000 research papers on the subject. A systematic review of the subject is an excellent jumping off point to navigate this enormous collection of research⁸⁰. Not only is there intestinal microbiome re-modelling, but it is also represented in the

^{76 &}lt;sup>®</sup>Guillaume Carissimo, Lisa F P Ng, A promiscuous interaction of SARS-CoV-2 with bacterial products, *Journal of Molecular Cell Biology*, Volume 12, Issue 12, December 2020, Pages 914–915, https://doi.org/10.1093/jmcb/mjaa068

^{77 &}lt;sup>®</sup>Yamamoto S, Saito M, Tamura A, Prawisuda D, Mizutani T, Yotsuyanagi H (2021) The human microbiome and COVID-19: A systematic review. PLoS ONE 16(6): e0253293. https://doi.org/10.1371/journal.pone.0253293

⁷⁸ Kipkorir V, Cheruiyot I, Ngure B, Misiani M, Munguti J. Prolonged SARS-CoV-2 RNA detection in anal/rectal swabs and stool specimens in COVID-19 patients after negative conversion in nasopharyngeal RT-PCR test. J Med Virol. 2020 Nov;92(11):2328-2331. doi: 10.1002/jmv.26007. Epub 2020 Aug 2. PMID: 32401374; PMCID: PMC7272912.

^{79 &}lt;sup>a</sup>Shang H, Bai T, Chen Y, Huang C, Zhang S, Yang P, Zhang L, Hou X. Outcomes and implications of diarrhea in patients with SARS-CoV-2 infection. Scand J Gastroenterol. 2020 Sep;55(9):1049-1056. doi: 10.1080/00365521.2020.1800078. Epub 2020 Aug 4. PMID: 32749177.

^{80 &}lt;sup>a</sup>Shinya Yamamoto, Makoto Saito, Azumi Tamura, Diki Prawisuda, Taketoshi Mizu. Otani, Hiroshi Yotsuyanagi: The human microbiome and COVID-19: A systematic review. Published: June 23, 202. 1https://doi.org/10.1371/journal.pone.0253293.

microbiome of the lung, mouth and nasal canal in a number of papers linked^{81, 82, 83, 84, 85, 86, 87}. Translocation of microbiome bacteria is found throughout the body in COVID-19 patients, with one paper considering this dynamic a key player in the acute disease⁸⁸. These studies indicate the complex relationship between the virus, bacteria and the body. It's interesting to note that breakdown of tryptophan metabolism is seen in dysbiosis⁸⁹ and is highly involved in COVID-19. Papers discussing tryptophan metabolism and COVID-19 are linked^{90, 91, 92, 93}. One studying observing MIS-C inflammatory syndrome in children after COVID-19 linked it to disruption of GI barrier and a persistent presence of viral material in the gut⁹⁴. By treating it with a peptide which closed the tight junctions, there was a rapid lowering of viral load and inflammatory markers. This indicates that "leaky gut" is a risk factor from inflammatory diseases resulting from COVID-19.

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- 82 ^aJohn P. Haran, ..., Beth A. McCormick, Vanni Bucci: Inflammation-type dysbiosis of the oral microbiome associates with the duration of COVID-19 symptoms and long COVID. Published August 17, 2021. JCI Insight. 2021;6(20):e152346. https://doi.org/10.1172/jci.insight.152346.

83 [®]Aktas, Busra; Aslim, Belma: Gut-lung axis and dysbiosis in COVID-19. *Turk J Biol*; 44(3): 265-272, 2020. MEDLINE | ID: covidwho-618514

- 84 Zhong, H., Wang, Y., Shi, Z. *et al.* Characterization of respiratory microbial dysbiosis in hospitalized COVID-19 patients. *Cell Discov* 7, 23 (2021). https://doi.org/10.1038/s41421-021-00257-2
- 85 [®]Xu, R., Lu, R., Zhang, T. *et al.* Temporal association between human upper respiratory and gut bacterial microbiomes during the course of COVID-19 in adults. *Commun Biol* 4, 240 (2021). https://doi.org/10.1038/s42003-021-01796-w
- 86 [®]Signatures of COVID-19 severity and immune response in the respiratory tract microbiom. Carter Merenstein, Guanxiang Liang, Samantha A. Whiteside, Ana G. Cobián-Güemes, Madeline S. Merlino, Louis J. Taylor, Abigail Glascock, Kyle Bittinger, Ceylan Tanes, Jevon Graham-Wooten, Layla A. Khatib, Ayannah S. Fitzgerald, Shantan Reddy, Amy E. Baxter, Josephine R. Giles, Derek A. Oldridge, Nuala J. Meyer, E. John Wherry, John E. McGinniss, Frederic D. Bushman, Ronald G. Collman. medRxiv 2021.04.02.21254514; doi: https://doi.org/10.1101/2021.04.02.21254514 Now published in *mBio* doi: 10.1128/mBio.01777-21
- 87 ^aSaroj Khatiwada, Astha Subedi: Lung microbiome and coronavirus disease 2019 (COVID-19): Possible link and implications, Human Microbiome Journal, Volume 17, 2020, 100073, ISSN 2452-2317, https://doi.org/10.1016/j.humic.2020.100073.
- 88 [®]Vincenzo Cardinale, Gabriele Capurso, Gianluca Ianiro, Antonio Gasbarrini, Paolo Giorgio Arcidiacono, Domenico Alvaro, Intestinal permeability changes with bacterial translocation as key events modulating systemic host immune response to SARS-CoV-2: A working hypothesis, Digestive and Liver Disease, Volume 52, Issue 12, 2020, Pages 1383-1389, ISSN 1590-8658, https://doi.org/10.1016/j.dld.2020.09.009.
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- 90 Eroğlu İ, Eroğlu BÇ, Güven GS. Altered tryptophan absorption and metabolism could underlie long-term symptoms in survivors of coronavirus disease 2019 (COVID-19). Nutrition. 2021;90:111308. doi:10.1016/j.nut.2021.111308
- 91 [®]Zanella LGFABD, Galvão LL (2021) The COVID-19 Burden or Tryptophan Syndrome: Autoimmunity, Immunoparalysis and Tolerance in a Tumorigenic Environment. J Infect Dis Epidemiol 7:195.
- 92 Tiffany Thomas, ..., Steven L. Spitalnik, Angelo D'Alessandro: COVID-19 infection alters kynurenine and fatty acid metabolism, correlating with IL-6 levels and renal status. Published June 19, 2020, JCI Insight. 2020;5(14):e140327. https://doi.org/10.1172/jci.insight.140327.
- 93 [®]Sagar Vyavahare, Sandeep Kumar, Nicholas Cantu, Ravindra Kolhe, Wendy B. Bollag, Meghan E. McGee-Lawrence, William D. Hill, Mark W. Hamrick, Carlos M. Isales, Sadanand Fulzele, "Tryptophan-Kynurenine Pathway in COVID-19-Dependent Musculoskeletal Pathology: A Minireview", *Mediators of Inflammation*, vol. 2021, Article ID 2911578, 6 pages, 2021. https://doi.org/10.1155/2021/2911578
- 94 [®]Lael M. Yonker, Tal Gilboa, Alana F. Ogata, Yasmeen Senussi, Roey Lazarovits, Brittany P. Boribong, Yannic C. Bartsch, Maggie Loiselle, Magali Noval Rivas, Rebecca A. Porritt, Rosiane Lima and Jameson P.

6.2. SARS-CoV-2 Venom Production

The most important research done to date on the spike protein/SARS-CoV-2 was done by a small Italian biotechnology firm. They had developed a peptide analysis system for detecting complex peptides in blood and studies the blood of patients infected with COVID19. They found a complex mix of peptides which mimic venoms, specifically in three main classes representing two types of organisms: conotoxins from the toxic cone snail and a number of classes from snakes. Most interestingly, these peptide venoms were similar across all patients, but not the exact same, with shuffled amino acid sequences. This indicates they did not come from direct production from a viral blueprint, but expressed from bacteria. This led to the detection of SARS-CoV-2 integrating with bacteria in the lower microbiome and further analysis and therapeutic concepts. They were able to culture sars-cov-2 genetic material on human microbiome cultures, showing the virus seems to grow and express SARS-CoV-2 genetic material⁹⁵. The three main classes of venoms were Conotoxin, Phospholipase A2 and Disintigrin/metalloproteases.

The Phospholipase A2 venom type peptides are easily detectable and are found highly correlated to COVID19 morbidity. One paper state's⁹⁶, "A decision tree generated by machine learning identified (Phospholipase A2) levels as a central node in the stratification of patients who died from COVID-19." Another paper states⁹⁷, "Strikingly, sPLA2-IIA and BUN also stood out as the two unique and essential predictors of the mortality in severe COVID-19 patients, with their feature importance rankings significantly higher (p < 0.0001) than other clinical indices in our random forest analysis." A paper looking at mortality data states⁹⁸, "PLA2 activity was identified as sPLA2-IIA28, and deceased COVID-19 patients averaged 18.7-fold higher than normal (<10 ng/ml)". This could even indicate the mechanism of comorbidity, "According to the WHO reports, most COVID-19 deaths seen are in people who suffered from other chronic diseases characterized by phospholipidosis and phosphatidylglycerol deficiency, including hypertension, liver, heart, and lung diseases and diabetes."

Davis, Eva J. Farkas, Madeleine D. Burns, Nicola Young, Vinay S. Mahajan, Soroush Hajizadeh, Xcanda I. Herrera Lopez, Johannes Kreuzer, Robert Morris, Enid E. Martinez, Isaac Han, Kettner Griswold Jr. AND Nicholas C. Barry, David B. Thompson, George Church, Andrea G. Edlo, Wilhelm Haas, Shiv Pillai, Moshe Arditi, Galit Alter, David R. Walt, Alessio Fasano: Multisystem inflammatory syndrome in children is driven by zonulin-dependent loss of gut mucosal barrier. The Journal of Clinical Investigation. The American Society for Clinical Investigation, 2021,7, vol. 131, https://www.jci.org/articles/view/149633

⁹⁵ Petrillo M, Brogna C, Cristoni S, Querci M, Piazza O, Van den Eede G. Increase of SARS-CoV-2 RNA load in faecal samples prompts for rethinking of SARS-CoV-2 biology and COVID-19 epidemiology. *F1000Res*. 2021;10:370. Published 2021 Jul 1. doi:10.12688/f1000research.52540.3

^{96 &}lt;sup>®</sup>Snider JM, You JK, Wang X, Snider AJ, Hallmark B, Zec MM, Seeds MC, Sergeant S, Johnstone L, Wang Q, Sprissler R, Carr TF, Lutrick K, Parthasarathy S, Bime C, Zhang HH, Luberto C, Kew RR, Hannun YA, Guerra S, McCall CE, Yao G, Del Poeta M, Chilton FH. Group IIA secreted phospholipase A2 is associated with the pathobiology leading to COVID-19 mortality. J Clin Invest. 2021 Oct 1;131(19):e149236. doi: 10.1172/JCI149236. PMID: 34428181; PMCID: PMC8483752.

⁹⁷ Popov, Dmitri. (2020). COVID-19 Toxicity, Role of phospholipases A2 in the development of Acute Severe Respiratory Distress Syndrome (ASRDS). 10.13140/RG.2.2.12006.65600.

^{98 &}lt;sup>a</sup>Justin M. Snider, Jeehyun Karen You, Xia Wang, Ashley J Snider, Brian Hallmark, Michael C. Seeds, Susan Sergeant, Laurel Johnstone, Qiuming Wang, Ryan Sprissler, Hao Helen Zhang, Chiara Luberto, Richard R. Kew, Yusuf A Hannun, Charles E. McCall, Guang Yao, Maurizio Del Poeta, Floyd H. Chilton: Group IIA Secreted Phospholipase A₂ Plays a Central Role in the Pathobiology of COVID-19. medRxiv preprint doi:

https://doi.org/10.1101/2021.02.22.21252237.

6.3. PIEZO1/2 Ion Channel and Consciousness Field Structures

The connection between venoms, COVID-19 and consciousness as well as the mysterious "magical" aspects of Gu syndrome seem to appear in the ion channel effects of COVID-19. There is a mysterious cluster of symptoms appearing in Long Covid which are also present most predominantly in vaccine damage. Anecdotally, these symptoms are: "Burning skin, tingling, brain fog, palpitations, internal jerking, adrenaline rushes, headache, sensitivity to light, weakness in both legs, internal vibrations, tremors, intrusive thoughts, feelings of impending doom, feeling something moving around the body".

There was a research paper written on the subject titled, "Internal Tremors and Vibration Symptoms Among People with Post-Acute Sequelae of SARS-CoV-2: A narrative review of patient reports"⁹⁹. There are hints that this cluster of symptoms is due to an energetic field structure with a consciousness component. Anecdotally, spouses can feel the electrical vibrations by sitting next to them as well as see the pulses caused by its integration with muscle ion channels. The above paper contains the following quote, "Few months later, unrelenting Neuro issues vibrations, ripples, tremors, became intense foot cramps, painful ankle, foot drop, leg spasms started mostly r foot. I had to wear ankle brace use cane. Very difficult to sleep. Husband could see ripples under skin and feel the vibrations at times."

Venoms are known to affect the ion channels and another hint toward a unifying mechanism comes in the genetic disposition studies of COVID-19 and a unique new class of ion receptor mutations. One study finds a mutation in the PIEZO1 receptor correlates to SARS-CoV-2 PCR positivity and another paper finds a correlation between these receptor mutations and COVID-19 fatality¹⁰⁰. The morbidity associated with PIEZO ion channel mutations as well as the cluster of exotic symptoms due to COVID-19 indicate this could be the biological structure which is responsible for coordinating a bioenergetic field structure, and COVID-19 may leverage this field structure. Those with buzzing, tingling and other ion channel like symptoms report dramatic changes in consciousness, feeling "possessed" and feeling something floating around their body, interacting with different organs and systems including systematic shocks throughout their blood stream.

The structure of the PIEZO ion channels indicates a unique role, it is formed by three "fingers" appearing in a propeller shape, tapering upwards from a common location in the cell membrane. While all other ion channels appear geometrically as an amorphous shape, the PIEZO ion channel has a strong shape and structure following Phi ratios, indicating it could have a higher dimensional bioenergetic function, acting as an antenna. Looking at the distribution of the PIEZO ion channel and its function its role as a synchronisation field for

⁹⁹ Daisy, Massey, BA; Anna D Baker, MPH; Diana Zicklin Berrent, JD; Nick Gu the; Suzanne Pincus,

Shidlovsky; Liza Fisher; Connor B Grady, MPH; César Caraballo, MD; Richa Sharma, MD, MPH; Harlan M Krumholz, MD, SM: Internal Tremors and Vibration Symptoms Among People with Post-Acute Sequelae of SARS- CoV-2: A narrative review of patient reports. medRxiv preprint doi: https://doi.org/10.1101/2021.12.03.21267146

^{100&}lt;sup>®</sup>Cheng, C.W., Deivasikamani, V., Ludlow, M.J., De Vecchis, D., Kalli, A.C., Beech, D.J.: Genetic variants of *PIEZO1* associate with COVID-19 fatality. School of Medicine, University of Leeds, Leeds, LS2 9JT, UK. medRxiv preprint doi: https://doi.org/10.1101/2020.06.01.20119651

the human body becomes clearer, as well as its role in post pathogenic diseases.

The PIEZO ion channel appears in all tissues which are involved in movement, either macro movement like connective tissues and cartilage, or micro movement like sheer flow in blood vessels and red blood cell surfaces as well as the valves which control lymph fluid. Its highly represented in the GI tract and particularly involved in the EC cells of the gut, which are the motion detecting cells responsible for producing serotonin. Studies have found PIEZO receptors are a critical aspect of this EC cell-based serotonin production¹⁰¹ and dysfunction of a PIEZO field structure indicates why many chronic diseases have disruption of serotonin production. The PIEZO receptors are also known to have a critical role in innate immunity¹⁰². These receptors are also involved in platelet activation and thrombosis¹⁰³, potentially connecting the energetic aspects of this field structure and the clotting side effects of COVID-19.

There are many clusters of symptoms associated which chronic disease, often cause by a post pathogenic illness. Orthostatic intolerance, joint hypermobility (Ehlers-Danlos syndrome), Fibromyalgia, Chronic Fatigue Syndrome, myalgic encephalomyelitis, microbial dysbiosis/SIBO are often highly correlated in fatiguing chronic diseases¹⁰⁴. All of these chronic and mysterious disease states are often triggered by a pathogen or toxic exposure and are connected by the PIEZO receptor. The PIEZO receptors are highly involved in the growth of cartilage and involved in bone growth and metabolism¹⁰⁵, indicating why both fibromyalgia and joint pains as well as joint hypermobility are involved in pathogen triggered chronic disease states often involve gut dysbiosis and depression/anxiety cycles. And the orthostatic intolerance is most easily explained by the loss of ability of the PIEZO receptors to judge changes in blood flow and blood pressure/baroreception, with one paper hypothesising that they are the driving mechanism of the baroreflex sensing system¹⁰⁶.

This PIEZO field structure seems to be responsible for the fine tuning of macro biological function, including the intelligent circulation of lymph fluid, the dynamic changes of the GI

103[®]Zhao W, Wei Z, Xin G, Li Y, Yuan J, Ming Y, Ji C, Sun Q, Li S, Chen X, Fu W, Zhu Y, Niu H, Huang W. Piezo1 initiates platelet hyperreactivity and accelerates thrombosis in hypertension. J Thromb Haemost. 2021 Dec;19(12):3113-3125. doi: 10.1111/jth.15504. Epub 2021 Oct 8. PMID: 34411418.

104[®]Peter C. Rowe, Diana F. Barron, Hugh Calkins, Irene H. Maumenee, Patrick Y. Tong, Michael T. Geraghty, Orthostatic intolerance and chronic fatigue syndrome associated with Ehlers-Danlos syndrome, The Journal of Pediatrics, Volume 135, Issue 4, 1999, Pages 494-499, ISSN 0022-3476, https://doi.org/10.1016/S0022-3476(99)70173-3.

^{101&}lt;sup>®</sup>Constanza Alcaino, Kaitlyn R. Knutson, Anthony J. Treichel, Gulcan Yildiz, Peter R. Strege, David R. Linden, Joyce H. Li, Andrew B. Leiter, Joseph H. Szurszewski, Gianrico Farrugia, Arthur Beyder: A population of gut epithelial enterochromaffin cells is mechanosensitive and requires Piezo2 to convert force into serotonin release. Proceedings of the National Academy of Sciences Aug 2018, 115 (32) E7632-E7641; DOI: 10.1073/pnas.1804938115.

^{102&}lt;sup>®</sup>Solis, A.G., Bielecki, P., Steach, H.R. *et al.* Mechanosensation of cyclical force by PIEZO1 is essential for innate immunity. *Nature* 573, 69–74 (2019). https://doi.org/10.1038/s41586-019-1485-8

^{105&}lt;sup>®</sup>Wang, L., You, X., Lotinun, S. *et al.* Mechanical sensing protein PIEZO1 regulates bone homeostasis via osteoblast-osteoclast crosstalk. *Nat Commun* **11**, 282 (2020). https://doi.org/10.1038/s41467-019-14146-6

^{106&}lt;sup>®</sup>Stocker SD, Sved AF, Andresen MC. Missing pieces of the Piezo1/Piezo2 baroreceptor hypothesis: an autonomic perspective. J Neurophysiol. 2019 Sep 1;122(3):1207-1212. doi: 10.1152/jn.00315.2019. Epub 2019 Jul 17. PMID: 31314636; PMCID: PMC6766733.

tract and microbiome as well as serotonin production, the change in fine tuning of blood flow and blood cell volume and is highly represented both in the brain and in the enteric nervous system of the GI tract. This bioenergetic field structure seems to exists as an electrical intelligence of the body and during pathogenic disease, seems to be taken over by the electrical intelligence of the pathogen for purposes of controlling the body. In some cases, when the pathogenic disease has run its course, the body's natural PIEZO field structure does not return to coherence, leading to dormant pathogens taking this field structure by default and implementing their own constructs, as well as a feedback loop of their physical multiplication and growth. Pathogen triggered disease may not be the only cause, as any venom should implement a conciseness construct on this field and has the potential to disrupt it. Even biotoxins and venoms removed from organisms like shell fish poison in food can cause these illnesses, indicating it is due to the venom construct and not the organism itself. Other systematic causes should be able to disrupt this field such as an acute emotional/physical trauma like a car accident, or a dramatic environmental stress like hypothermia.

The natural mechanism of pathogens taking this energetic field may indicate why all ancient medicine teaching discussed demons and possessions as the source of disease, as they were perceiving the consciousness effects of the pathogens constructs acting on the PIEZO field structure, which is highly represented in the enteric nervous system and does effect to some extent higher consciousness. The "Three Corpses" view of Doaist medicine seems to recognise the division of this bioenergetic field, they state that there is a worm of each of the three corpses in the gut, the chest and the head. These three worms may represent nodes of this bioenergetic field structure which can independently become docoherent. This makes sense in the context of pathogen triggered disease as many people find after recovery from illness, they have a long-term cough, respiratory illnesses more often and other issues associated only with the chest, while others recover from pathogen illness in the GI tract and find it never feels right again. Some people with "Neuro AIDS" or "Neuro COVID" find that their symptoms are clustered around their head and brain and persist in the long term.

6.4. Venom Constructs

In terms of pathogens and venoms implementing constructs on the PIEZO field structure of the human body, there seem to be different mechanisms. Pathogens must be existing in the human body and their consciousness constructs influence is correlated by the amount of the pathogen present. Venoms on the other hand, can be introduced externally and have a rapid, intense effect on the PIEZO system which wears off quickly. Pathogens seem to have a more complex consciousness construct whereas venoms induce a construct which is very simple, but very intense.

One excellent example is the ritual use of Kambo, the venom secretion of the *Phyllomedusa bicolor* tree frog. Kambo is used to both physically and energetically purge the body, causing a short but intense sickness with projectile vomiting. Combined with the lymph fluid dumping into the stomach beforehand, this is a highly effective physical detoxification method. But this purging construct seems to exist at higher levels, acting on the PIEZO energetic structure to control lymph coordination and lymph fluid dumping as well as fully overtaking the PIEZO field structure and ejecting disease constructs and then smoothly returning it to the bodies control afterwards. There is an effect on higher consciousness, where emotional constructs are

brought to the surface and seem to purge at the exact same moment of the physical purge. This fractal induction of a "purging" construct across the physical plane, bioenergetic plane and consciousness plane is an excellent example of the true power of venoms and their associated constructs. This "purging" construct comes from the evolutionary purpose of the venom, when the frog is eaten by a predator, the venom is excreted from its skin, causing the predator to purge the frog from its stomach and mouth.

The same fractal induction of venom constructs across multiple layers of existence (physical, bioenergetic, consciousness) seems to be leveraged highly by SARS-CoV-2 as well as the spike protein. While cone toxin and snake venoms have been found in COVID-19 patients and are strongly associated with physical symptoms, there is indication the higher higher dimensional activity. This higher dimensional mechanism can be observed by comparing the hunting behaviours of the animal which produced the venoms found in COVID-19, compared to the changes in higher consciousness associated with COVID-19 or spike protein exposure.

The cone snail, or *Conus Sp.* is a sea snail that hunts fish by using a spike full of incredibly potent venom, one of the most potent. This symbolism of a toxic needle/spike is highly relevant in the conceptual realm. The Cone snail releases toxins into the water around it which stun and hypnotise the fish, the fish then swims into the cone snail and is then hit by a toxic spike. This same construct is observable at the population level, one member of a household gets vaccinated, they shed spike proteins into the environment of their home, and then the rest of the family, previously vaccine resistant decides they must immediately get the vaccine and go to the vaccination center to be penetrated with a syringe (spike) full of toxins (venom). This construct is so powerful that there are many reports of vaccine hesitant people walking into vaccination centers in a daze, realising their mistake and returning home, only for the process to repeat itself many times over.

There are multiple classes of venoms in COVID-19 patients represented from the snake and seem to be multiple constructs also represented. The first construct seems to be energetic discrimination, this is associated with the disintegring component of the venom which is utilized by snakes to track the prey they had previously bitten, in an attempt to avoid injury¹⁰⁷. This venom construct seems to involve an energetic discrimination ability between the vaccinated and unvaccinated. You can imagine that in evolutionary terms, a mouse struck by a snake will be tracked energetically, but it will also have consciousness changes, being pushed to avoid their tribe/den so the snake does not have to burrow or encounter opposition in retrieving its prey. You can imagine that this discrimination causes prey that are envenomated to stick together to make collection easier, while they avoid their tribe/den as "they are not like them". You can also imagine that the mice in the tribe and den avoid the envenomated prey because "they are not like them" and are a risk to the tribe/den as they are compromised and may allow a predator to destroy their den. This dynamic seems to be playing out on the population level with the unvaccinated and vaccinated avoiding each other for different reasons, following the same dynamics as snake/prey strike and release behaviour.

Another class of venoms present are the Phospholipase A2 type peptides. These are venoms which mimic biological enzymes in the human body and have unique abilities to target specific organs, they are highly specific and targeted. These venoms seem to be organ-paralysis-agents and contribute to the breakdown of the prey's body and escape mechanisms.

^{107&}lt;sup>a</sup>Saviola AJ, Chiszar D, Busch C, Mackessy SP. Molecular basis for prey relocation in viperid snakes. *BMC Biol.* 2013;11:20. Published 2013 Mar 1. doi:10.1186/1741-7007-11-20.

At the higher levels, it seems these class of venoms may break down the prey's ego and consciousness in the face of an oncoming predator to prevent defensive action. This is a common construct found in venom toxicity and venom medicine, the feeling of impending doom and health failure. This construct seems to be affecting consciousness at the population level with much of the population mentally paralysed in the face of an oncoming enemy (health tyranny) and yet highly fearful of their health and impending health issues (COVID-19 infection).

7. Summary of findings

The data gathered and the mechanisms postulated are quite complex. To give a better understanding of the issue, it makes sense to observe things from the perspective of possible intent.

From there, we can see a number of goals someone possibly tries to achieve:

• Detaching the emotional body from the physical body as well as from the dayconsciousness attached to the body, that would be by definition: the sum of identifications of the "I", to open the body up for demonic entities, for the entities to become included into the sum of these identifications. In plain text: this is about demonic possession.

The first mechanism, the detaching of the emotional body, is mainly achieved by disbalancing the haemoglobin/ferritin ration. With the thyroid malfunctioning, the Ca supply is know to collapse, while with the destruction of red blood cells, the ferritin deep storage is increased. This will lead to a decay of the hexagonal Calcium Phosphates, that are then replaced by orthorhombic Iron Phosphates. The change in crystal symmetry defines the type of field structure that can attach to the crystalline cluster and by that, to the perception of the nervous system.

• Braking the epigenetic switch BRCA1/53BP1 that controls the state of disease/health, to detach the spiritual self, i.e. the aspect of the divine self, attached to the current incarnation, also referred to as the "soul aspect" to kill any inner dialogue that could question and dissolve these identifications. The souls the is replaced by the consciousness associated with the Covid RNA and/or mRNA.

This idea or better observation is coming from the anecdotal, subjective experience of methylating/demethylating the two genes by affirmation. The mechanism of disease control and its subjective perception was evident to quite a number of test persons.

• Braking the epigenetic switch BRCA1/53BP1 that controls the state of disease/health, to disable genetic self-repair, to be able to run a multiple step genetic reprogramming by mRNA vaccines without having nature resetting itself to default inbetween the single shots.

This idea is coming from the research of the role of these genes in the development of breast cancer.

• Poisoning humans with venoms borrowed from nature to manoeuvre humanity into victimhood and compliance, making people wanting to get jabbed.

This is becoming evident just by considering the likelihood of coincidences. The number of genetic sequences producing snake and cone-snail venoms cannot be combined in COVID RNA by coincidence. Once I acknowledge intend, the correlation of predatory behaviour of snakes and cone-snails to the psychology observed in the population during the pandemic, also cannot be regarded as coincidental.

8. Outlook for possible therapeutic innovations

The understanding of these findings can be used in various therapeutic approaches, like blocking infection/shedding with essential oils, resetting health by affirming the epigenetics back into place, or using alternative methods like radionics, that work with encoded affirmations to switch epigenetics by administering globules or other carrier substances informed with radionic signatures. Venoms can be neutralized with antidotes. Haemoglobin/ferritin ratio can be addressed with nutritional measures, while giving the magnetization of the body observed in many vaccinated people as well as with Long Covid a diagnostic value to determine how bad the disbalance is. Manipulated genetics can be restored by strengthening the entanglement of the DNA to the natural background field that holds the natural blueprints of life.

Being a member of the timeloopsolution Consortium, based in Germany/Switzerland, the author took part in the development of such solutions. You might consider visiting the general info page timeloopssolution.com. Solutions are traded by sirisana.com.