Covid-19 : What if there is a cure for the Covid-19 infected and vaccinated?

Exposing the cause and mechanism of illness behind Covid-19, paving the way to full health restoration

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Introduction

People with comorbidities such as high blood pressure, diabetes, cancer, Alzheimer's Disease, Heart Disease, COPD, chronic kidney disease, chronic liver disease and AIDS are more likely to suffer severe symptoms if exposed to SARS-CoV-2 through infection or vaccination. So, why then, does exposure to SARS-CoV-2 lead to the development of these very same conditions? The connection is the kidneys.

This document aims to draw attention to the mechanism behind much of Covid-19 illness, "long covid" and the symptoms on the horizon for the Covid-19 vaccinated population, with a particular interest in AIDS.

Resume

Transmembrane TNF-alpha, a potent pro-inflammatory cytokine, facilitates SARS-CoV-2 entry into macrophages, lymphocytes, monocytes and dendritic cells via CD4 receptors, stimulating the release of Interferon 1 (IFN 1). IFN 1, is essential in controlling the growth and activity of the above listed immune system cells, activating the antiviral defences of these cells. SARS-CoV-2 infiltrates CD4 cells via the GP120 gene insertion on the binding site of SARS-CoV-2, which allows SARS-CoV-2 to enter the kidney distal convoluted tubules, which have no ACE2 receptors, explaining the symptoms, markers and autopsy results we know today as Covid-19.

Background

Why are some people asymptomatic when exposed to SARS-CoV-2 whilst others develop moderate to severe conditions with wide-ranging symptoms, including death? Why do children rarely develop Covid-19 symptoms? These questions and the understanding of the majority of the seemingly unrelated symptoms of Covid-19 can be traced back to SARS-CoV-2 induced damage of the kidney's proximal and distal convoluted tubules. Pre-existing kidney tubule damage explains who is at risk of Covid-19 illness and who is not, as SARS-CoV-2 further impairs kidney function in the already kidney impaired.

In order to understand the mechanism behind Covid-19, we must first recognize that ACE2 is not the only pathway SARS-CoV-2 uses to infect human cells. GP120 facilitates the infiltration of SARS-CoV-2 into macrophages, lymphocytes, monocytes and dendritic cells, via CD4 cells, triggering an immune-mediated inflammatory state as explained in a rare

paper published in the *American Society of Nephrology*. Importantly, the mechanism behind GP120 also exposes the pathway towards an effective protocol available to restore the health of the SARS-CoV-2 exposed population.

GP120; the missing link behind Covid-19 illness (via infection or vaccination)

Why some people develop symptoms after Covid-19 infection or vaccination and others don't, comes down to **the capacity of a person's kidney tubules before infection/vaccination**. SARS-CoV-2 induces acute kidney injury, via ACE2 and CD4 receptors, to both the proximal and the distal kidney tubules, further impairing kidney function, in the already kidney impaired.

Kidney tubule injury symptoms are diverse and multi-system. The vast array of Covid-19 symptoms are, unsurprisingly, the same as kidney tubule damage symptoms, as SARS-CoV-2 "targets" the kidney tubules.

Covid-19 symptoms	**Kidney tubule damage symptoms**
- Chronic fatigue	- Chronic fatigue
- Chronic pain	- Chronic pain
- Chronic inflammation	- Chronic inflammation
- Fever	- Fever
- Headaches	- Headaches
- Brain fog	- Brain fog
- Dizziness	- Dizziness
- Chronic itching	- Chronic itching
- Drawn or gaunt face	- Drawn or gaunt face
- Skin rashes and blisters	- Skin rashes and blisters
- Water retention	- Cellulite and water retention
- Swelling	- Swelling
- Congestion	- Congestion
- Reduction of smell and vision	- Reduction of smell and vision
- Heart arrythmias	- Heart arrythmias
- High blood pressure/hypertension…	- High blood pressure/hypertension…

Acute Kidney Injury (AKI) is widely recognized in hospitals treating Covid-19 symptoms, *"As we cared for patients with COVID-19, we noticed an alarming number of patients who developed acute kidney injury (AKI)"* [(1)]

In April, 2020, a group of Chinese scientists, mostly working in Wuhan, published a paper titled, *Human Kidney is a Target for Novel Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) Infection*. [(2)] The paper states *"We found that 27.06% of patients with COVID-19 had abnormal eGFR, and patients who are aged or have comorbidities more commonly developed ARF, suggesting that ARF is relatively common following SARS-CoV-2 infection."* [(3)] **SARS-CoV-2 induces acute kidney injury that can lead to Acute Renal Failure (ARF), in people with pre-existing kidney tubules damage**, often demonstrated by kidney related comorbidities such as, but not limited to: Diabetes [(4), (5), (6)], high blood pressure [(7), (8), (9)], skin

conditions [10], [11], [12], cancer [13], [14], [15], heart disease [16], [17], [18], AIDS [19], [20], [21], autism [22], [23], [24], and dementia [25], [26], [27].

Whereas people with no, or minimal pre-existing kidney damage, before being exposed to SARS-CoV-2 may not notice a slight decrease in kidney tubule function, other than for example early signs of kidney tubule damage which include chronic fatigue, chronic inflammation, chronic pain or skin rashes. The Chinese scientists went further stating; *"SARS-CoV-2 mediated ARF may be one of the major causes of multiorgan failure and eventual death in COVID-19* patients" [28] Concluding; *"SARS-CoV-2 induces ARF in COVID-19 patients."* [29]

The lower the kidney function prior to SARS-CoV-2 infection the more severe the Covid-19 symptoms, *"highlighting the importance of pre-renal ARF in this pandemic."* [30] *"The presence of AKF is a factor of a worse prognosis and higher mortality in patients admitted with SARS-CoV-2 infection."* [31] Pre-existing kidney damage prior to exposure to SARS-CoV-2 suggests why children, who usually have better functioning kidneys, were rarely affected by Covid-19. However, the kidney damage witnessed in the population who suffered severe Covid-19 symptoms, does not just occur in the elderly and chronically ill. The Covid-19 infected and vaccinated population have been exposed to the same kidney tubule injury. "Long Covid" is quite literally induced kidney insufficiency, that if untreated will lead to the rise of kidney-related chronic diseases.

The elephant in the room being; SARS-CoV-2 injures the kidneys, but it is pre-existing kidney tubule damage that determines who develops Covid-19 symptoms and the severity of those symptoms, as SARS-CoV-2 induces further acute kidney injury, in the already kidney impaired.

SARS-CoV-2: The mechanics of Covid-19 illness

Cell entry: ACE2 receptor & CD4 receptor

Disease origin:

ACE2 receptors are abundant in the kidney's proximal convoluted tubules, allowing SARS-CoV-2 to enter proximal tubule cells. *"Renal biopsy and autopsy records present significant acute tubule injury, revealing that interstitial injury is more common and severe than glomerular damage. The kidney autopsy results showed diffuse acute proximal tubular injury with loss of brush border and nonisometric vacuolation."* [32] SARS-CoV-2 induces proximal tubule damage, yet *"The glomerular lesion was minor".* [33] *"ACE2 staining revealed that ACE2 expression was predominant in proximal tubular cells, particularly in areas with severe acute tubule injury."* [34]. Deficiency of ACE2 and local tissue damage caused by SARS-CoV-2 infection disrupts RAAS (renin-angiotensin-aldosterone-system), which has a systemic effect on the body's water volume (both extra and intracellular), reducing blood and lymph fluidity, impairing cellular respiration, leading to thickened blood/clotting, lymph congestion, impaired waste elimination… Proximal tubule damage can be partially counter-balanced in the distal convoluted tubules which regulate RAAS, in order to maintain vital fluid homeostasis.

In the case of Covid-19, **the importance of a second pathway used to enter cells that do not have ACE2 receptors, illuminates the mechanics of Covid-19 illness and also the pathway to health restoration. Autopsies show *"The renal distal tubule was highly damaged in severe COVID-19 patients."*** [35] *"These results indicate that the kidney is a direct target for SARS-CoV-2 infection; however, the way how SARS-CoV-2 enters the renal tubular cells was still unknown ".* [36] This confusion arises because focus is still being placed on ACE2 as the only SARS-CoV-2 portal into human cells, yet ACE2 is not the only pathway SARS-CoV-2 uses to enter cells:

- **SARS-CoV-2 targets and damages the proximal convoluted tubules via ACE2 receptors, creating an ACE2 deficiency.** *"Destruction of ACE2 by SARS-CoV-2 results in loss of control of the pro-inflammatory actions of Ang II and tissue destruction."* [37]
- **SARS-CoV-2 targets and damages the distal convoluted tubules via CD4 receptors.** The GP120 gene, located on the SARS-CoV-2 spike protein [38], allows SARS-CoV-2 to enter the distal tubules, via macrophages, lymphocytes, monocytes and dendritic cell infiltration.

Nobel Prize laurate, and discoverer of HIV, Professor Luc Montagnier detected the GP120 gene (the "AIDS gene"), in the SARS-CoV-2 genome, implying in early 2020 that SARS-CoV-2 would create AIDS symptoms in some of the SARS-CoV-2 infected/vaccinated population. In order to understand the mechanism behind Covid-19 symptoms, including induced immunopathology, we need to study the mechanism of action of GP120 in HIV.

Covid-19 and the AIDS connection

A paper published in the Journal of the *American Society of Nephrology* in 2016, led by nephrologist Patricio Ray, MD explains *"No one understood how HIV could affect kidney cells that lack the receptors expressed in T cells and white cells"* yet nephrologists were seeing HIV infection damaging kidney tubule epithelial cells that lacked CD4, a major receptor where HIV attaches. The nephrologists discovered **Transmembrane TNF-alpha facilitates GP120 entry into certain cell types, enabling HIV, (and therefore SARS-CoV-2, as they both carry the GP120 gene), to enter cells and replicate, *"like a Trojan horse, the macrophage sits atop the epithelial cell with HIV hidden inside, opening a doorway into the kidney cell for high levels of HIV-1 to enter."*** [39]

Transmembrane TNF-alpha, a potent pro-inflammatory cytokine, facilitates SARS-CoV-2 entry into macrophages, lymphocytes, monocytes and dendritic cells via GP120 infiltration into CD4 cells located on the epithelium of these immune cells, stimulating the release of Interferon 1 (IFN 1). IFN 1, being a type of cytokine essential in controlling the growth and activity of other immune system cells, activating the antiviral defences of these cells that are nearby the infected cell to limit the spread of the virus. The *"Release of type I IFNs is one of the most potent mechanisms of innate immunity against viruses and other intracellular pathogens."* [40] Increased IFN 1 levels in SARS-CoV-2 infection, occurs in direct response to increased TNF-alpha facilitating GP120 entry into macrophages, monocytes, lymphocytes and dendritic cells.

SARS-CoV-2 is able to enter the kidney's distal tubules as GP120 infiltrates CD4 receptors of the immune cells that are abundant in the interstitium of the proximal and distal tubules, exactly where the tubule damage has been identified in Covid-19 autopsy results. It should be noted that SARS-CoV-2 induces the same proximal tubule damage as HIV; "*The kidney autopsy results showed diffuse acute proximal tubular injury with loss of brush border and nonisometric vacuolation."* [41] *"However, in autopsy kidney samples, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) nucleoprotein was detected in ACE2+ or TMPRSS2+ renal tubular cells, whereas the RNAscope® Assay targeting the* ***SARS-CoV-2 Spike gene was positive mainly in the distal tubular cells and seldom in the proximal tubular cells.*** *In addition, the TMPRSS2 and* ***kidney injury marker protein levels were significantly higher in the SARS-CoV-2-infected renal distal tubular cells, indicating that SARS-CoV-2-mediated AKI mainly occurred in the renal distal tubular cells.*** *"* [42] **GP120 drives SARS-CoV-2 spike protein into kidney distal tubule cells, where no ACE2 receptors are found, inducing AKI. AKI in the distal tubules and the severe disruption of RAAS seen in Covid-19, arrive from GP120 induced damage in the kidney's distal convoluted tubules.**

The kidney's proximal and distal tubules regulate vital water, sodium and electrolyte reabsorption in the body, maintaining blood and lymph fluidity and function, cell structure and function, cell signalling, MiRNA transport, cellular energy production…, through sodium and water reabsorption and via the renin-angiotensin-aldosterone-system (RAAS), in order to maintain fluid homeostasis required to maintain fluid volume and cellular respiration.

SARS-CoV-2 induces distal tubule damage of the *macular densa* cells along the distal tubule epithelium, leading to a loss of water volume in the body, resulting in hypovolemia, ischemia, hypoxia, hematuria, proteinuria, elevated D'dimer levels, as *"D-dimer levels are often elevated in renal insufficiency"* [43], congestion within the lymphatic system backing up into the interstitium, triggering interstitial acidosis and the irritation of the outer membrane of vital organs causing inflammation, necrosis and fibrosis of organ tissue seen in the tubulointerstitium. GP120 also initiates immune-mediated tubule pathogenesis in Covid-19, in the same manner it does during infection with HIV.

IMPORTANT !!! Future health consequences for the Covid-19 vaccinated population

"*Renal tubule injury: a driving force toward chronic kidney disease"* [44] The kidney tubule injury created by SARS-CoV-2 vaccine exposure will lead to a massive increase in chronic kidney disease, if the kidney connection to Covid-19 goes undetected. Added to this, in 2012 chronic kidney disease already occurred in 1/10 people throughout the world (1/7 in America) and 1/200 children are born in America with a form of kidney insufficiency [45] due to a combination of chemicals such as, but not limited to: certain medications including over the counter pain killers, chemotherapy, the weedkiller RoundUp, glyphosate [46], [47] and sodium fluoride [48], [49] found in toothpaste, drinking water in many countries and non-stick cooking pans.

Many chemicals in our environment today chronically damage the kidney tubules, creating a silent chronic kidney disease epidemic, as "*Renal tubule injury"* is *"a driving force toward chronic kidney disease"* [50]

Why is the chronic kidney epidemic going unnoticed?

75% of countries have declared a shortage of nephrologists, mostly through a lack of interest and funding in the field. Blood tests are being offered as a solution to diagnose kidney insufficiency, yet these tests are inefficient as they do not detect early kidney tubule damage. Standard kidney diagnostics mostly focus on glomerular filtration rates, yet it is well accepted that damage to the kidney tubules occurs prior to any glomerular dysfunction.

Through a lack of attention to the problem, a lack of medical training focused on the role of the kidney tubules in disease development, health maintenance and health restoration and a lack of effective testing and diagnostic tools, kidney tubule damage is ravaging world health.

Kidney tubule injury drives chronic kidney disease, which leads to the development of many chronic diseases. In the face of 90 years of scientific data, including multiple Nobel Prize winning discoveries and experienced-based medicine showing the opposite [(51), (52)], allopathic medicine still favors the notion that kidney insufficiency is caused by chronic disease, simply because early diagnostics and detection of kidney tubule damage is going under the radar. As such no chronic diseases are being reversed in the allopathic medical system, as no chronic disease treatments target kidney tubule restoration. However, when the kidneys are restored through a specific diet-supplementation regime or a kidney transplant, extraordinary results occur, for example; the prestigious peer review journal *Front Cell Neuroscience* published a paper in 2018, titled *'A Novel Perspective Linkage Between Kidney Function and Alzheimer's Diseases*', stating *"It has long been believed that kidney function is linked to brain activity. Clinical studies demonstrate that patients with chronic kidney disease (CKD) are more prone to cognitive impairment and Alzheimer's disease (AD),* ***and the degree of cognitive impairment is closely related to CKD progression and renal failure. Moreover, the fact that cognitive function in CKD patients is significantly improved after successful kidney transplantation reveals a linkage between CKD and AD****."* *(53)*

If kidney tubule damage leads to the development of chronic disease and Covid-19 infection and vaccination induce kidney tubule damage, shouldn't we be seeing an unexplained rise of all kidneys-related chronic diseases? Yes, and we are.

Below are CDC "provisional" statistics from 2020 (estimated to be much higher), confirming the unexplained rise in chronic disease was already occurring in the USA, PRIOR to Covid-19 vaccination:

The increase in **heart disease** deaths in 2018 compared to 2017 was **7,924**
The increase in **heart disease** deaths in 2019 compared to 2018 was **3,660**
The increase in **heart disease** deaths in 2020 compared to 2019 was **31,841**

The increase in **stroke** deaths in 2018 compared to 2017 was **1,427**
The increase in **strokes** deaths in 2019 compared to 2018 was **2,195**
The increase in **strokes** deaths in 2020 compared to 2019 was **9,045**

The increase in **Alzheimer's** deaths in 2018 compared to 2017 was **615**

The increase in **Alzheimer's** deaths in 2019 compared to 2018 was **2,701**
The increase in **Alzheimer's** deaths in 2020 compared to 2019 was **11,883**

The increase in **Diabetes** deaths in 2018 compared to 2017 was **1,382**
The increase in **Diabetes** deaths in 2019 compared to 2018 was **2,701**
The increase in **Diabetes** deaths in 2020 compared to 2019 was **13,459**

Table. Number of Deaths for Leading Causes of Death, US, 2015-2020[a]

	No. of deaths by year					
Cause of death	2015	2016	2017	2018	2019	2020
Total deaths	2 712 630	2 744 248	2 813 503	2 839 205	2 854 838	3 358 814
Heart disease	633 842	635 260	647 457	655 381	659 041	690 882
Cancer	595 930	598 038	599 108	599 274	599 601	598 932
COVID-19[b]						345 323
Unintentional injuries	146 571	161 374	169 936	167 127	173 040	192 176
Stroke	140 323	142 142	146 383	147 810	150 005	159 050
Chronic lower respiratory diseases	155 041	154 596	160 201	159 486	156 979	151 637
Alzheimer disease	110 561	116 103	121 404	122 019	121 499	133 382
Diabetes	79 535	80 058	83 564	84 946	87 647	101 106
Influenza and pneumonia	57 062	51 537	55 672	59 120	49 783	53 495
Kidney disease	49 959	50 046	50 633	51 386	51 565	52 260
Suicide	44 193	44 965	47 173	48 344	47 511	44 834

[a] Leading causes are classified according to underlying cause and presented according to the number of deaths among US residents. For more information, see the article by Heron.[4] Source: National Center for Health Statistics. National Vital Statistics System: mortality statistics (http://www.cdc.gov/nchs/deaths.htm). Data for 2015-2019 are final; data for 2020 are provisional.

[b] Deaths with confirmed or presumed COVID-19, coded to *International Statistical Classification of Diseases and Related Health Problems, Tenth Revision* code U07.1 as the underlying cause of death.

(54)

WARNING: the next wave of symptoms created by SARS-CoV-2 induced kidney tubule damage; skin rashes and wounds, cancer, AIDS, chronic fatigue and neurological conditions will not be diagnosed as symptoms of such, but rather blamed on new variants, or new epidemics if the kidney connection is not made public.

However, ***"SARS-CoV-2 Spike gene was positive mainly in the distal tubular cells and seldom in the proximal tubular cells.*** *In addition, the TMPRSS2 and* ***kidney injury marker protein levels were significantly higher in the SARS-CoV-2-infected renal distal tubular cells, indicating that SARS-CoV-2-mediated AKI mainly occurred in the renal distal tubular cells."*** (42)

GP120 drives SARS-CoV-2 spike protein into kidney distal tubule cells, where no ACE2 receptors are found, inducing AKI. AKI in the distal tubules and the severe disruption of RAAS seen in Covid-19, come from GP120 induced damage in the kidney's distal convoluted tubules, not ACE2.

An opportunity for new medical practices

Covid-19 will allow the elephant in the room to be exposed; chronic kidney disease, caused by chronic kidney tubule damage is driving the world's chronic disease epidemic, and in a beautiful twist of fate, **chronic tubule damage is easily reversible**, opening a pathway to

disease prevention and disease reversal, based on 90 years of scientific discoveries and evidenced based medicine, a better way forward can now take place.

Better diagnostics required

"Kidney function estimation was commonly made using SCr concentration, blood urea nitrogen (BUN) level and urine analysis. However accumulating evidence has demonstrated that these biomarkers are not optimal to detect kidney disease in early stages." [55] Kidney tubule *"Damage usually precedes alterations in functions. For instance it is known that albuminuria precedes the decrease in eGFR"* [56] Making albuminuria a better indication of tubule damage, however symptomology remains the best measure of tubule damage, as *"Although albuminuria is a powerful biomarker, it may occur after the damage has occurred or may not be present in other types of kidney damage such as tubulointerstitial disease and hypertensive kidney disease."* [57]

Solutions:

Any successful treatment for the SARS-CoV-2 infected or vaccinated must cover; ACE2 receptor regeneration, blockage of the GP120 pathway, kidney and other tissue restoration, lymphatic drainage (targeted metabolic waste removal) and RAAS (fluid) restoration. All of which are accessible, with little cost, to every person in need.

Suramin

"Suramin is a drug that has been used to treat African sleeping sickness and river blindness for over a hundred years. Suramin has also been shown to be an inhibitor against a wide variety of viruses, including Zika and Chikungunya viruses, indicating its diverse mechanisms of action against multiple viruses." [58]

"For instance, it can prevent viral attachment, entry, and release from host cells by its interactions with viral capsid proteins. Recent research has shown it to be an in vitro inhibitor of SARS-CoV-2 infection in cell culture by preventing viral entry. The current study shows this to be due to its potent inhibition of SARS-CoV-2 RNA-dependent RNA polymerase (RdRp), an essential viral lifecycle enzyme. Suramin is 20 or more times as powerful as remdesivir, which is the only currently approved anti-COVID-19 drug." [59]

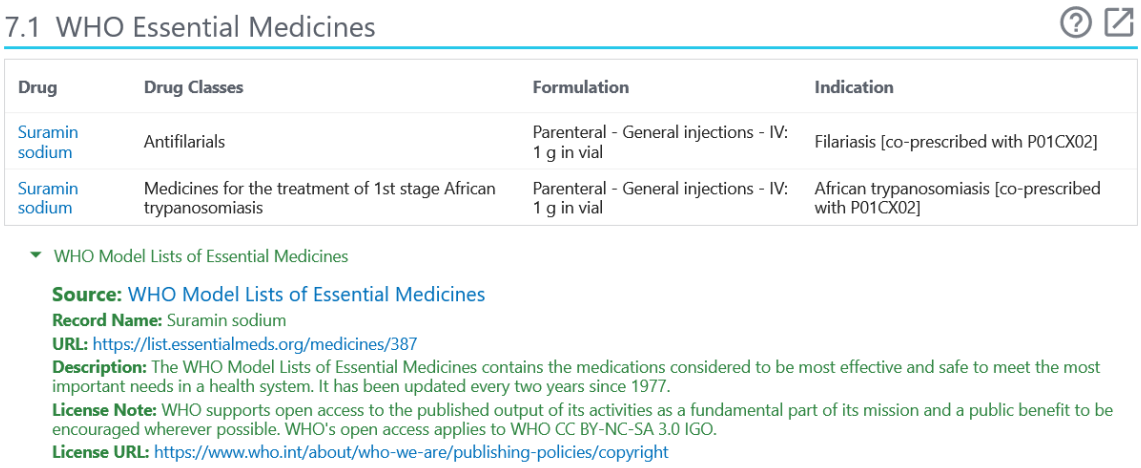
"The antiparasitic drug suramin inhibits SARS-CoV-2 replication… suramin also inhibited the progression of infection." [60], [61], [62], [63], [64], [65]

For Suramin to be a real cure against Covid-19 illness, it needs to stop entry, replication and inhibit the progression of SARS-CoV-2 infection in human cells, which it does. However, as the pathological process behind Covid-19 stems greatly from kidney tubule injury impairing RAAS, a Covid-19 "cure" would also need to protect and even repair kidney tubule damage, which Suramin does. *"In this study, we determined the effects of suramin on renal ischemia/reperfusion-induced acute kidney injury in mice, in particular its effect when administered after renal injury has been established. Increasing concentrations of suramin*

were given 24 hours following reperfusion, a time when serum creatinine levels were at their highest level. This treatment improved renal function, as evidenced by decreased blood urea nitrogen and serum creatinine to control values and diminished histopathologic tubular damage. Suramin-treated animals had a significant reduction in apoptotic tubular cells and infiltrating leukocytes. There was also an increase of proliferating tubular cells following reperfusion compared to the number found in untreated animals. ***Our study shows that suramin promotes the recovery of renal function and has effective therapeutic applications when given after the occurrence of renal injury.*** *"* (66), (67), (68), (69), (70)

On the PubChem website, Suramin has listed therapeutic effects on: *"Autistic disorders, carcinomas, hypothermia, kidney neoplasms, multiple myelomas, pain, Urinary Bladder Neoplasms."* Unsurprisingly, as all the listed conditions stem from kidney tubule damage *(I can back this point up upon request).*

Suramin is *"an FDA approved drug"* (71) and listed by The WHO as an *"Essential Medicine"*. (72)



7.1 WHO Essential Medicines

Drug	Drug Classes	Formulation	Indication
Suramin sodium	Antifilarials	Parenteral - General injections - IV: 1 g in vial	Filariasis [co-prescribed with P01CX02]
Suramin sodium	Medicines for the treatment of 1st stage African trypanosomiasis	Parenteral - General injections - IV: 1 g in vial	African trypanosomiasis [co-prescribed with P01CX02]

WHO Model Lists of Essential Medicines

Source: WHO Model Lists of Essential Medicines
Record Name: Suramin sodium
URL: https://list.essentialmeds.org/medicines/387
Description: The WHO Model Lists of Essential Medicines contains the medications considered to be most effective and safe to meet the most important needs in a health system. It has been updated every two years since 1977.
License Note: WHO supports open access to the published output of its activities as a fundamental part of its mission and a public benefit to be encouraged wherever possible. WHO's open access applies to WHO CC BY-NC-SA 3.0 IGO.
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(73)

If GP120 was the pathogen behind both AIDS and Covid-19, creating the same pathway to disease, the same "cure" would have the same positive affect on both diseases, which Suramin does, and who better to explain the efficiency of Suramin on HIV infection than Dr. Anthony Fauci?

An article titled *"Researchers Report Progress in Fight Against AIDS Virus"* published in 1985, stated *"Dr. Anthony Fauci, director of the National Institute of Allergy and Infectious Diseases, said that leading researchers met here Monday and concluded that one promising drug, suramin, if administered intravenously for six or more weeks, can effectively block the ability of the AIDS virus to reproduce itself in the body."* Continuing *"Dr. Fauci said there was 'no question' that the early data 'Very strongly suggests' that suramin suppresses the virus, at least while the patient is taking the drug."* (74)

Note from the author

If there is one thing that Covid-19 has taught us, it is that when information is violently censored, you can be sure it is directly over the target. So, I guess now we know why the Indian paper, published in January, 2020, *"Uncanny similarity of unique inserts in the 2019 - nCoV spike protein to HIV-1 gp120 and Gag"* [(75)] which identified GP120 and Gag inserts in the SARS-CoV-2 spike protein was so swiftly censored. Their paper reads: *"We found 4 insertions in the spike glycoprotein (S) which are unique to the 2019-nCoV and are not present in other coronaviruses. Importantly, amino acid residues in all the 4 inserts have identity or similarity to those in the HIV-1 gp120 or HIV-1 Gag. Interestingly, despite the inserts being discontinuous on the primary amino acid sequence,* ***3D-modelling of the 2019-nCoV suggests that they converge to constitute the receptor binding site****. The finding of 4 unique inserts in the 2019-nCoV, all of which have identity /similarity to amino acid residues in key structural proteins of HIV-1 is unlikely to be fortuitous in nature. This work provides yet unknown insights on 2019-nCoV and sheds light on the evolution and pathogenicity of this virus with important implications for diagnosis of this virus."*

I guess now we know why their paper was so swiftly censored. **There is no doubt that GP120 is the main pathogen in SARS-CoV-2, and just as in AIDS, the GP120 damage is reversible if the correct action is taken.**

Conclusion

In order to restore health in the SARS-CoV-2 exposed population, a multi-facet, yet simple and cost-effective protocol needs to be established.

Further research into the use of Suramin is needed in the SARS-CoV-2 exposed population. Suramin blocks SARS-CoV-2 entry, replication and inhibits disease progression, however, the development of Covid-19 and AIDS goes further than just HIV or SARS-CoV-2 infiltration of GP120 into CD4 cells damaging kidney tubules. In order to restore all damaged tissue, not just the kidney tubules, stimulation of collagen and elastin synthesis is essential, along with increased cellular respiration activity, the correction of RAAS to restore fluid volume, lymphatic drainage and mediated MiRNA recovery.

A successful Covid-19 protocol must focus on the three principles of health restoration:

1. Cleaning/detox: detoxing the cells and storage of metabolic and inflammatory waste, including Covid-19 spike protein.
2. Hydration: of the extra and intracellular fluid and stimulation of ATP production.
3. Repair and restoration of the kidney tubules and other damaged tissue.

All of which is simple, cost-effective and can be made available to every person.

The day medicine focuses on health is the day the people take back their authority. The direct contrast of the World Council for Health's *"Oath of a Medicus"* which puts the people first, with the WHO's IHR which requires surrendering the freedoms and sovereign rights of the people *"to protect them",* should be the ultimate wake-up call for humanity.

As health restoration comes back into medicine, so will freedom and prosperity for the people.

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