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Protocol

Issues which Influence the Etiology of CoVid-19 infection: A Proposed Treatment Protocol Based upon Optimising the Autonomic and Immune Response

Graham Wilfred Ewing*

Chief Executive, Mimex Montague Healthcare Limited, Mulberry House, 6 Vine Farm Close, Cotgrave, Nottingham NG12 3TU, USA

*Corresponding author: Chief Executive, Mimex Montague Healthcare Limited, Mulberry House, 6 Vine Farm Close, Cotgrave, Nottingham NG12 3TU, USA, Tel: (+44) 115-9899618; mob: 07885-755847; E-mail: graham.ewing@mmhcl.co.uk

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Abstract

In general the criteria used to diagnose a medical condition is based upon the known etiology of the condition however in the case of CoVid-19 the pathophysiology of the infection, in particular how it leads to the death of patients infected with the virus, has yet to be clearly defined. This paper considers the range of known observations which accompany the onset of the CoVid-19 viral infection and looks at phenomena which could be deployed as the basis of a diagnostic test, and whether this could lead to ways of treating the condition by stimulating the immune response in the CoVid-19 patient. Neutrophils, the white blood cells which fight infection, have polar characteristics. Most proteins are polar entities e.g. insulin, cytokines, immunoglobulins, etc. These proteins, in particular, have a unique chemical structure which can adopt different spatial orientations and/or structures. Many of these cells and proteins depend for their structure, energetics and reactivity, upon the prevailing intercellular pH and the availability of essential minerals in particular the essential minerals magnesium and zinc. It outlines a possible treatment regimen based upon enhancing the activity of cytokines and other immune proteins by modulating intercellular/blood pH, optimising levels of magnesium and zinc, and perhaps also by neuromodulating the autonomic nervous system.

Keywords: CoVid-19, neuromodulation

Introduction

Whilst the CoVid-19 virus has been circulating in the world economy since the period late Q3/2019-early Q4/2019 there is not yet an accepted understanding of its mode of action. It is an entirely new viral infection, of uncertain and/or unknown origin, therefore knowledge of how it functions and, in particular how it infects patients and leads to their death has not yet been identified although information is steadily being accumulated by reporters in the tabloid press and in published research in medical journals i.e. by researchers, clinicians, and doctors through their work with patients.

The genome for the virus was first decoded by BGI in early Q1/2020 however at least three strains, and over 33 mutations, of the virus are now known to be circulating [1,2]. It is therefore inevitable that the virulence of each of these strains will differ and that each strain will influence how the body functions in subtly different ways.

The mode of action of the CoVid-19 virus appears to differ between population groups i.e. between the young and old, between different racial groups, and between those who are healthy and those who are not healthy, in particular those who have chronic medical conditions e.g. diabetes, obesity, heart disease(s), lung disease(s), etc. The mode of action of the virus may be quite different to previous viral outbreaks. If so, this presents a quite different set of challenges to the healthcare professions and specialisms as they seek to develop their coping strategies. Moreover if the CoVid-19 virus does not act on all people, irrespective of their demographic status and/or characteristics e.g. age, race, gender, health; it may mean that the production of a vaccine may be a very much more difficult if not impossible task.

Changes of genetic profile as a result of exposure to viruses/viral contaminants and virus-like particles (mainly vaccines) increase predisposition to and occurrence of type 1 diabetes [3-5], heart disease[6], and presumably also a range of other medical indications. The early indications are that CoVid-19 infects the patient through the ACE pathways [7] and damages and/or alters heart function [8-11].

CoVid-19 patients have high levels of Ferritin, low levels of haemoglobin, erythrocytes and neutrophils, and elevated levels of the inflammatory markers ALT, CRP, etc. Despite there being adequate levels of oxygen supplied to the lungs there are indications that destruction of haemoglobin – evidenced by the observation that the blood has a very dark appearance - prevents

delivery of oxygen to the alveoli and hence to the blood, and its absorption by the blood, and hence its delivery to the brain, heart and other organs which require oxygen in order to perform their function [12]. This is given increased validity when noting the beneficial effect of artificially enriching blood with oxygen using extracorporeal membrane oxygenation (ECMO) [13]. It becomes even more intriguing when noting that some patients 'who are not struggling and don't show signs of oxygen deprivation in their blood have such low levels in their blood that a lab report would suggest they're already dead' [14].

It is generally considered that exposure to viruses tends to infect all who are exposed to the virus although clearly there are many in the populations of the world who have a greater level of resistance and/or immunity to infection e.g. through diet, being less stressed, being physically fit, and having a more resilient genetic profile.

The CoVid-19 coronavirus poses a number of issues which the scientific community is struggling to address. The virus affects a broad range of people in particular those who have lower levels of immunity. Although the statistics surrounding this viral infection are at the earliest stages of compilation it is reported in the tabloid press and in medical journals that the risk of death is greater for those who are immunosuppressed e.g. those with pre-existing chronic conditions who are being treated by drugs, waiting for organ replacement procedures, are overweight and physically unfit, are being treated with immunosuppressive drugs perhaps following organ replacement surgical procedures, being treated for cancer, the elderly, etc.

In the case of CoVid-19 patients die as a result of a perceived 'cytokine storm' in which components in the immune response overwhelm the body's autonomic response and results in the bronchospasm/cough response which attempts to physically remove the accumulation of water and sputum/phlegm and/or via the mechanism which inhibits and/or impedes the ability of the lungs to exchange/replace CO2 with O2.

A 'Cytokine storm' is an often noted expression of the immune response to a viral infection. It comprises the release of more than 150 inflammatory mediators such as various interleukins, interferons, tumour necrosis factor alpha, Monocyte chemoattractant protein 1, etc; and is considered to be indicative of acute respiratory distress syndrome and multiple organ failure similar to that experienced by patients who have been infected by the SARS-COV-2 virus (CoVid-19).

Diagnosis

It is rare for diagnostic tests to be precise and accurate. Biomarker tests are invariably based upon a set of assumptions which limit the accuracy of the tests. Bearing this in mind, there are currently two mechanisms for diagnosing whether the patient is infected by CoVid-19: the PCR test to determine exposure to the virus i.e. whether the patient is currently infected with the virus; and antibody tests which determine whether the patient has recovered from the CoVid-19 virus.

Ideally a test which determines CoVid-19 would be the most ideal solution to this problem however this is not yet possible. Perhaps the most promising technique in terms of its potential specificity and sensitivity is binding the virus to a substrate which can be measured [15] e.g. nucleocapsid protein. In order to best resolve such problem(s) the research community is developing tests which break the virus into components which can then be measured however this introduces a range of possible errors into the test e.g. the component being measured may not be the only component being measured by the test. It could measure similar components which have alternative origins and which respond to the particular diagnostic substrate. The test will also be influenced to some degree by factors which influence the test outcomes e.g. the sampling process using a swab may not be infallible, the accuracy of the PCR and/or antibody tests may differ between different manufacturers [16].

This exposes a number of issues, deficiencies, in the test regime e.g. (i) the sampling process may have distinct limitations i.e. throat swabs are only reliable in the first week of the disease; thereafter the virus can disappear from the throat whilst it multiplies in the lungs [17]. (ii) The patient may only recently have been infected by the CoVid-19 virus and the infection may not yet be sufficiently severe, or of such magnitude, that it is identified by the PCR test i.e. the sensitivity of the test may be inadequate. As CoVid-19 is a highly infectious virus it highlights that this could be a very significant limitation of the test. (iii) A positive test result may just be confirming that the patient is infected however negative tests result may not be able to confirm that the patient is free from infection. Specificity may be inadequate. (iv) The antibody tests are not yet able to reliably determine whether the patient has had the infection. Initial indications are the antibody tests which are available from Chinese suppliers may only be 30-50% reliable. So far UK-based suppliers have been unable to develop a satisfactory test i.e. of sufficient specificity and sensitivity which satisfies the authorities [17,18] although there are initial indications that new second or third generation tests have 80+% sensitivity.

This does not yet address fundamental requirements regarding the treatment regime, in particular, what is happening in the patient when they are infected and when chronic conditions are developing. To try and understand how the CoVid-19 virus influences the patient's health biomedicine uses a broad range of histopathology and radiological tests to identify how the patient is responding to the infection [19,20] e.g. to have a x-ray of the lungs by MRI/CTS, check heart function, blood oxygen levels, white blood cell count, liver and kidney function.

Pathophysiology of the CoVid-19 Viral Infection

In general, modern medicine reacts to the symptoms i.e. it uses drugs which treat the symptoms rather than addressing the fundamental causal mechanism which, in this case, is the CoVid-19 viral infection. It uses drugs to kill secondary infections but, with few possible exceptions, it cannot yet kill or otherwise eradicate the primary viral CoVid-19 infection. It uses drugs and devices to tackle the symptoms e.g. to open the breathing pathways and hence ease breathing and the supply of oxygen [21], to suppress or enhance the cough stimulus, to ease congestion, to reduce body temperature - this is based upon the assumption that lack of oxygen supply is the fundamental problem - but it does not yet address the fundamental reasons why the young and healthy are less vulnerable to infection and to develop secondary complications, whilst up to 15% of the population are likely to develop secondary infections and complications e.g. in

particular pneumonia. It is early in the research into this topic. Nevertheless there are interesting pieces of information which are becoming evident e.g. some doctors, recognising the poor outcomes being obtained using ventilators (up to 90% of patients on ventilators do not recover), are starting to question the wisdom of using ventilators so extensively [21,22].

In general those who are younger are much less liable to develop complications or die from the infection. CoVid-19 affects patients with a compromised immune and/or autonomic response. This includes those who are diabetic, obese and have chronic problems with the heart or lungs, and who are immunocompromised e.g. the elderly, those being treated for cancer, kidney/dialysis patients [23], etc; are considered to be high-risk patients i.e. highly at risk of death from CoVid-19. Chinese researchers have reported that the majority of patients infected with CoVid-19 and who go on to develop more serious complications and/or die of the condition tend to be those who are much heavier than normal [24]. So what is the difference between these different sub-groups?

Younger patients without congenital conditions tend to have lower body weight and to be more active. This means that their immune function is better able to respond to viral infections at the point of contact i.e. via the body interfaces including, but not limited to, the eyes and lungs. In general the elderly or those with congenital issues influencing the function of the heart and lungs tend to have a heavier predisposition which influences (i) how much effective lung capacity they have available to respond and (ii) how much blood is able to circulate throughout the body, including the lungs, and its immune response. Moreover if the patient is heavier, perhaps diabetic and/or obese, their intercellular pH will decline (intercellular acidity increases), blood pH will decline (acidity increases), and levels of magnesium and zinc will decline [25-29] and hence the prevailing immune response will be reduced [25-31]. It conceivably indicates elevated levels of acidity (low intercellular pH) and nothing to transport oxygen which would be exascerbated the ability of an already weakened heart to pump blood. Similarly, the immune response of patients declines as they age and have lower functional capabilities [31,32]. This is illustrated by data, assembled by the CDC, which records the age-related risk of death in South Korean CoVid-19 patient and in the UK the latest data from the Office of National Statistics (ONS) reports: 36% were aged 85 or over; 33% were aged 75 to 84; 19% were aged 65 to 74;11% were aged 45 to 64; just 1% were aged 15 to 44. No-one can make up their mind about the reliability of the diagnostic tests [33,34] (Table 1).

Table 1. Korean CoVid-19 patient and in the UK the latest data from the Office of National Statistics (ONS) reports.

Age years and above	% deaths
80+	10.4%
70-80	5.35%
60-70	1.51%
50-60	0.37%
<30	0%

What needs to be tested?

The obvious indications to be tested [35] include: 2019-nCoV pneumonia, body temperature for signs of fever, lungs for signs of shortness of breath and/or breathing difficulties. So what do we know about patient susceptibility to CoVid-19? Those who are young and/or physically fit are not predisposed to this infection whilst those who have diabetes, hypertension, obesity, senility, and pre-existing and/or congenital conditions of the heart, lungs and kidneys have significant risk of death [36]. In the UK, 38% of patients admitted to intensive care are obese and 55% of these patients subsequently die. It affects different age and racial groups [37] in different ways. As the disease progresses it affects patients in different ways e.g. ca 80% of critically ill patients develop kidney failure whilst an estimated 80-90% of patients with kidney failure die [38].

There is evidence that drinking alcohol increases the risk of contracting this virus [39] which illustrates that the conversion of ethyl alcohol to acetic acid i.e. which elevates levels of intercellular acidity, may be significant. Elevated levels of acidity have the effect of reducing levels of essential minerals including magnesium and zinc, and elevating levels of transition metals including iron (as ferritin). This is significant because immune cells and proteins - neutrophils, cytokines, immunoglobulins, etc - are being expressed in excess because such patients are magnesium deficient and hence that the cytokines cannot function due to this magnesium deficiency. The body is producing cytokines to combat the effect of the virus but the cytokines are ineffective. The cytokines require magnesium to function. It has no other options other than to continue to produce a steady stream of cytokines - the so called 'cytokine storm' - which steadily overwhelm the metabolic capabilities and/or processes in the lungs [40] and allow growth of 2019-nCoV pneumonia.

"It might be that we're seeing a cytokine storm because of a failure of interferon to restrict the virus to begin with, so the lungs start calling for more help. That's exactly what we're trying to understand right now." Ordovas-Montanes J.

Magnesium is a recognised cofactor in a wide range of immune processes [41] e.g. it is a co-factor which supports the activity of neutrophils (WBC), immunoglobulin synthesis, C′3 convertase, immune cell adherence, antibody-dependent cytolysis, IgM lymphocyte binding, macrophage response to lymphokines, T helper–B cell adherence [42] and cytokine function [43,44]. In general, increased levels of essential minerals stimulate immune function. This is not new to medicine. Such knowledge has been applied in other circumstances e.g. to treat HELP syndrome in a subset of patients with pre-eclampsia [45].

Zinc must also be significant because the enzyme carbonic anhydrase is Zinc dependent i.e. Zinc activates carbonic anhydrase which catalyses the conversion of CO2 to HCO3 (bicarbonate) in the lungs, in particular in the red blood cells (RBC) [46-48]. Accordingly a shortage of Zinc, will suppress the conversion of CO2 to HCO3, increase blood acidity, suppress the levels of oxygen in the lungs, etc (Figure 1).

A new AI tool [48] found, by examining patterns in the data, that three particular features were predictive of the severity of onset of the condition with up to 80% accuracy. The tool is based upon measuring the liver enzyme alanine aminotransferase (ALT), myalgia, and haemoglobin levels i.e. that these three

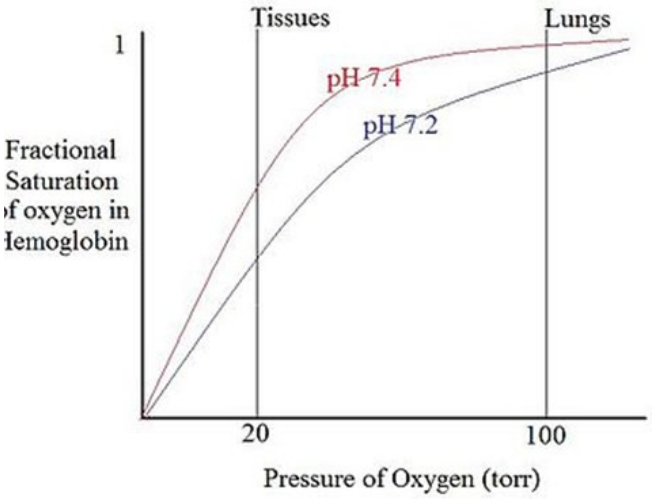

Figure 1. Oxygen levels vs Saturation of Oxygen in Haemoglobin at different pH (Wikipedia).

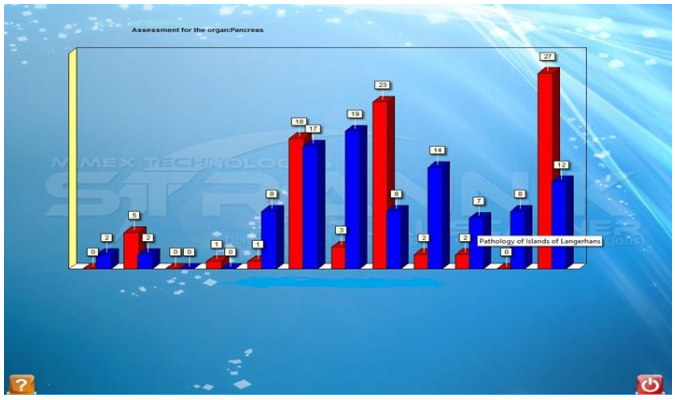

Figure 2. Strannik Virtual Scanning Test Report: Pancreas.
Report: This particular patient has reduced expression of insulin (7 units(blue)) and low phenotype (2units(red)) which are at presymptomatic level (below 10 units) for both type 1 and type 2 diabetes i.e. the patient has pre-diabetes.
Note 1: The SVS test determines determine ca 15 pathological indications in each of the 30 main organs.

prognostic indicators were considered to be predictive of the onset of severe disease.

Accordingly it is reasonable to conclude that death arising from CoVid-19 is due to a combination of acidosis, the hypoxia arising therefrom, and pneumonia.

Stimulating the Immune Response

We can see what can be measured using contemporary biomedical indices however what measures can be introduced to treat CoVid-19 patients? In general there are two fundamental mechanisms which can be used to treat CoVid-19: (i) by acting upon the symptoms using drugs and (ii) by acting upon the fundamental regulatory mechanisms. So far drugs are proving to be relatively ineffective although there is hope that combinations of several existing drugs e.g. remdisivir, hydroxychloroquine, and perhaps also the antibiotic azithromycin to act upon pneumonia (which develops as a result of the progressive inability of the immune response to suppress such infections); may have a beneficial effect.

Use broad-spectrum screening technologies which have the potential to improve the understanding of the patient's overall health and to do so at significantly lower cost than contemporary diagnostic methods/tests.

The Strannik Virtual Scanning test would, in principle, be able to identify the pathologies of concern in at risk patients however it is not, at this stage, known whether the test could be satisfactorily undertaken by patients who are in the advanced stages of infection (Figure 2).

In earlier reports the author reported how the Strannik Virtual Scanning (SVS) test could be used to screen for the onset of pathologies which were associated with the onset or progression of diabetes, cardiac pathologies, and many other medical indications, and that the test is 2-23% more accurate than the entire range of biomarker-type diagnostic and scanning tests against which it was compared [49].

In the SVS test each pathology is described by two parameters: The first is the phenotype - the number is a mathematical expression or correlate of the number of pathological reactions which occur per unit of time. The second is the genotype. The greater the number, the greater is the pathological indication.

Note 2: In general, numbers below 10 units are indicative of the pre-symptomatic state whilst the numbers above 10 units are at the symptomatic level e.g. pathology of the islets of Langerhans is the diabetic indication. A patient with a 14/4 result would be a type 2 diabetic (at the symptomatic level) but would also be experiencing reduced levels of insulin expression although at a minor presymptomatic level. A patient with a 3/21 result would be a type 1 diabetic whilst a patient with a 15/20 result would have both type 1 and type 2 diabetes.

Each report also provides a numerical indication of the most destabilized physiological system and organs. In the case of CoVid-19 SVS would determine pathological onset and levels of pathological progression - diabetic indications would likely be accompanied by cardiovascular indications, perhaps cardiosclerosis, cardiac myopathy and/or cardiac insufficiency, and a range of pathological indications in other organs and physiological systems e.g. blood pressure; and related organs e.g. lungs (e.g. respiratory insufficiency, bronchial asthma, pneumonia, etc), kidneys (e.g. renal insufficiency), liver (e.g. liver insufficiency), peripheral blood vessels, musculoskeletal system (e.g. myopathy), etc. (iii) Use neuromodulation interventions to enhance the immune response as outlined. Whereas natural sunlight is commonly associated with a decline in viral infections during the summer, conceivably that the UV radiation has a sterilising effect upon viruses [50-52]. The first studies report [53] in locations where there is strong sunlight the virus has a half-life of 2 minutes and does not survive i.e. in many non-CoVid cases sunlight acts as an immune stimulant and/or photoactivator however such phenomena has been extensively studied in past research [54]. It is not a new phenomena.

The Strannik Neuromodulation Therapy (SLT) modulates the coherent function of the autonomic nervous system and/or immune response i.e. it uses knowledge of how light has photoactivating effect upon the body's function; however it requires that the patient is treated using a non-invasive light therapy. This may present practical and/or logistical issues re the CoVid-19 patient which, although not insurmountable, would add to the inconvenience of such an approach. Nevertheless the prospect

of being able to achieve therapeutic outcomes of 75-96% effectiveness [49] should ensure that, at some stage, such an approach is considered.

Discussion/ Conclusions

The author suggests that levels of blood pH, magnesium and zinc should be measured and that a therapeutic regime based upon regulating these parameters - pH, and levels of magnesium and zinc - should be beneficial re the treatment of CoVid-19 patients.

Therapeutic regimes have been introduced by doctors which focus upon disrupting the virus e.g. using chloroquine and/or hydroxychloroquine; acting upon the developing pneumococcal infection using an antibiotic e.g. azithromycin; and using zinc as an immune stimulant. It is suggested that by implementing the proposed treatment regime that the need for drugs e.g. hydroxychloroquine, azithromycin; would be much reduced.

The most susceptible patients appear to be those with elevated levels of intercellular acidity arising from their lifestyles i.e. being inactive, consuming acidified and/or alcoholic beverages, being diabetic and obese, who subsequently develop heart, kidney and liver problems. These are medical indications which are accompanied by low levels of essential minerals and which adversely influence their immune response. The proposed regimen can easily be adopted because many of the ingredients are freely available as a result of treating patients with pre-eclampsia.

Regulating blood pH would enhance the absorption and bioavailability of magnesium and zinc. This would, in turn, optimise the immune response, in particular the cytokines, alleviate the 'cytokine storm' often observed in such patients, and optimise the elimination of CO2 which accumulates due to lack of zinc in such patients.

Note 3: the use of Magnesium via an IV drip would elevate the patient's body temperature so extreme care should be taken when administering this form of therapy. Pre-eclampsia patients who have been administered such forms of therapy have described 'being boiled from the inside' [55].

Abbreviations

BGI: Beijing Genetics Institute; ACE: Angiotensin-converting enzyme

References

1. Forster P, Forster L, Renfrew C, et al. Phylogenetic network analysis of SARS-CoV-2 genomes. Proc Natl Acad Sci U S A. 2020;117(17):9241-9243.
2. https://www.dailymail.co.uk/health/article-8237849/Coronavirus-mutated-Strains-evolved-far-deadlier-spread-Europe-New-York.html?ito=push-notification&ci=13484&si=82324
3. Bergamin CS, Dib SA. Enterovirus and type 1 diabetes: What is the matter?. World J Diabetes. 2015; 6(6):828-39.
4. Harrison LC, Perrett KP, Jachno K, et al.Does rotavirus turn on type 1 diabetes?.PLoS Pathog. 2019; 15(10):e1007965.
5. Ewing GW. The Interpretation of Genetic Data - Considering the Effect of Changes to Gene Conformation -- If the facts don't support the theory, change the theory – how does this contribute to understanding Diabetes? J Genet Disor Genet Rep. 2017; 6(4):1-4.
6. Kathiresan S, Srivastava D. Genetics of Human Cardiovascular Disease. Cell. 2012; 148(6):1242-1257.
7. https://www.sciencealert.com/why-is-this-coronavirus-so-much-more-dangerous-a-coronavirus-expert-explains
8. Kwong JC, Schwartz KL, Campitelli MA, et al. Acute Myocardial Infarction after Laboratory-Confirmed Influenza Infection. N Engl J Med. 2018; 378:345-353.
9. Madjid M, Safavi-Naeini P, Solomon SD, et al. Potential Effects of Coronaviruses on the Cardiovascular System - A Review. JAMA Cardiol. 2020.
10. Bonow RO, Fonarow GC, O'Gara PT, et al. Association of Coronavirus Disease 2019 (COVID-19) With Myocardial Injury and Mortality. JAMA Cardiol. 2020.
11. Jeyanathan T, Overgaard C, McGeer A. Wuhan Data Link COVID-19 With Myocardial Damage.
12. Wenzhong L, Hualan L. COVID-19: Attacks the 1-Beta Chain of Hemoglobin and Captures the Porphyrin to Inhibit Human Heme Metabolism. ChemRxiv. 2020. Preprint.
13. https://www.dailymail.co.uk/health/article-8208571/NYC-hospital-removes-blood-enriches-oxygen-returns-Covid-patients.html
14. https://www.statnews.com/2020/04/08/doctors-say-ventilators-overused-for-covid-19/
15. https://www.england.nhs.uk/coronavirus/wp-content/uploads/sites/52/2020/03/guidance-and-sop-covid-19-virus-testing-in-nhs-laboratories-v1.pdf
16. https://en.wikipedia.org/wiki/COVID-19_testing
17. https://www.npr.org/sections/goatsandsoda/2020/03/27/822407626/mystery-in-wuhan-recovered-coronavirus-patients-test-negative-then-positive?t=1586179064566
18. https://www.bbc.co.uk/news/uk-52177125
19. https://fortune.com/2020/04/04/italy-coronavirus-symptoms-test-ai-china-covid-19/
20. https://www.timesofisrael.com/some-doctors-fear-ventilators-could-actually-be-harming-virus-patients/
21. https://www.livescience.com/coronavirus-ventilator-deaths-new-york.html?utm_source=notification
22. Bushinsky DA, Coe FL, Katzenberg C, et al. Arterial pCO2 in chronic metabolic acidosis. Kidney Int. 1982;22(3):311-4.
23. Preliminary Estimates of the Prevalence of Selected Underlying Health Conditions Among Patients with Coronavirus 2019-United States, February 12-March 2019, 2020. MMWR Morb Mortal Wkly Rep. 2020;69:382-386.
24. Schmitz C, Perraud A-L. Molecular, Genetic, and Nutritional Aspects of Major and Trace Minerals. 2017;319-331.
25. Tam M, Gómez S, González-Gross M, et al. Possible roles of magnesium on the immune system. Eur J Clin Nutr. 2003;57(10):1193-7.
26. Galland L. Magnesium and immune function: an overview. Magnesium. 1988;7(5-6):290-9.
27. Shankar AH, Prasad AS. Zinc and immune function: the biological basis of altered resistance to infection. Am.J.Clin.Nutr. 1998;68(2 Suppl):447S-463S.
28. Anzilotti C, Swan DJ, Boisson B, et al. An essential role for the Zn2+ transporter ZIP7 in B cell development. Nat Immunol.2019;20:350-361.
29. Aspinall R, Lang PO. Interventions to restore appropriate immune function in the elderly. Immun Ageing. 2018;15:5.
30. Cairns CB, Krafi M. Magnesium Attenuates the Neutrophil Respiratory Burst in Adult Asthmatic Patients Charles B. Cairns, MD,

Monica Krafi. Acad Emerg Med. 1996;3:1093-1097.
31. Weyand CM, Goronzy JJ. Aging of the Immune System. Mechanisms and Therapeutic Targets. Ann Am Thoracic Soc. 2016;13(5).
32. https://doi.org/10.1513/AnnalsATS.201602-095AW
33. https://www.npr.org/sections/goatsandsoda/2020/03/27/822407626/mystery-in-wuhan-recovered-coronavirus-patients-test-negative-then-positive?t=1586179064566
34. Kanne JP, Little BP, Chung JH, et al. Essentials for Radiologists on COVID-19: An Update. Radiology Scientific Expert Panel, 2020.
35. Chen N, Zhou M, Dong X, et al. Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. The Lancet. 2020; 395(10223):507-513.
36. Cai Q, Chen F, Luo F. Obesity and COVID-19 Severity in a Designated Hospital in Shenzhen, China (3/13/2020). Available at http://dx.doi.org/10.2139/ssrn.3556658
37. https://www.bbc.co.uk/news/world-us-canada-52245690
38. https://nymag.com/intelligencer/2020/04/coronavirus-nyc-hospital-icu.html
39. https://www.thesun.co.uk/news/11416568/drinking-alcohol-increase-coronavirus-who-warns/?utm_medium=browser_notifications&utm_source=pushly
40. Ziegler CGK, Allon SJ, Nyquist SK, et al. SARS-CoV-2 receptor ACE2 is an interferon-stimulated gene in human airway epithelial cells and is detected in specific cell subsets across tissues Cell. 2020.
41. Tam, M., Gómez, S., González-Gross, M. et al. Possible roles of magnesium on the immune system. Eur. J. Clin. Nutr.2003; 57, 1193–1197.
42. Galland L. Magnesium and immune function: an overview. Magnesium 1988;7:290-299.
43. Sugimoto J, Romani AM, Valentin-Torres AM, et al. Magnesium Decreases Inflammatory Cytokine Production: A Novel Innate Immunomodulatory Mechanism. J Immunol. 2012;188(12).
44. [43] Bernstein H, Sugimoto J, Suzuki-Kakisaka H, et al. Magnesium decreases inflammatory cytokine production: a novel innate immunomodulatory mechanism. Am JObstetrics Gynecol. 2012; 206(1):S361.
45. Duley L, Neilson JP. Magnesium sulphate and pre-eclampsia. Trial needed to see whether it's as valuable in pre-eclampsia as in eclampsia. Brit Med J 1999;3;319(7201): 3-4.
46. Fegler J. Function of Carbonic Anhydrase in Blood. Nature 1944;153:137-138.
47. Supuran CT. Carbonic anhydrases--an overview. Curr Pharm Des. 2008;14(7):603-14.
48. Jakubowski M, Szahidewicz-Krupska E, Doroszko A. The Human Carbonic Anhydrase II in Platelets: An Underestimated Field of Its Activity. BioMed Res Int. 2018; 4548353
49. Jiang X, Coffee M, Bari A, et al. Towards an Artificial Intelligence Framework for Data-Driven Prediction of Coronavirus Clinical Severity. Computers, Materials Continua 2020; 63(1): 537-551.
50. Grakov IG, Ewing GW, Mohanlall R, et al. A summary or meta-analysis of data regarding the use of Strannik Virtual Scanning as a screening modality for healthcare. Asian Journal of Pharmacy. Nurs Med Sc. 2017;5(3):55-71.
51. Larcom LL, Thaker NH. The effects of Temperature and Ultra-Violet Radiation on Multiplication of Bacteriophage 029. Bioph J. 1977;19:299-306.
52. Norval M, el-Ghorr A, Garssen J, et al. The effects of ultraviolet light irradiation on viral infections. Br J Dermatol. 1994;130(6):693-700.
53. Norval M. The Effect of Ultraviolet Radiation on Human Viral Infections. Photochem Photobiol. 2006;82:1495-1504.
54. https://www.thesun.co.uk/news/11468665/coronavirus-dies-sunlight-us-homeland-security-study/?utm_medium=browser_notifications&utm_source=pushly
55. Lytle CD, Sagripanti J-L. Predicted Inactivation of Viruses of Relevance to Biodefense by Solar Radiation. J Virol. 2005;79(22):14244–14252.
56. Management of Severe Pre-eclampsia and EclampsiaA: Guidelines
57. https://www.rqia.org.uk/RQIA/files/84/8425a24b-5ceb-448d-b214-3be15e19bd32.pdf

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