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SARS-CoV-2 Miscellany (Spike Proteins, Replication Rate, Infectivity, Viability, Eradication Methods, Therapeutic Vitamins, and Susceptivity to Specific UV Light)

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Abstract

The *Cambridge Dictionary*, in one sense, defines the word, miscellany, as a "collection of pieces of writing...." The foundation of this particular miscellany is a doctoral dissertation (see Supplementary Note) that was adapted into four distinct peer-reviewed articles (also published by E-Cronicon). However, several interesting and informative sections of the dissertation remained unused after the adaption that resulted in those four published articles. Nevertheless, the authors considered some research "remnants" of practical value and possibly engaging to a reader. Thus, the following dissertation extracts were selected from the remaining research, comprising the following SARS-CoV-2 miscellany.

Keywords: *Ascorbic Acid; Coronaviridae; Detergent; Disinfectant; Heat Treatment; Radiation; Replication Rate; Survivability; Temperature; UV Light; Vitamin C; Vitamin D*

Abbreviations

AA: Ascorbic Acid; BSA: Bovine Serum Albumin; CAP: Community-Acquired Pneumonia; IQR: Interquartile Range; LOD: Limit of Detection; R_0: Replication Rate; ROS: Reactive Oxygen Species; QAC: Quaternary Ammonium Compound

Salient points

What is SARS-CoV-2?

SARS-CoV-2 belongs to the family of coronaviridae and the genus betacoronavirus. SARS-CoV-2 is formed of spherical structures with spike proteins on its surface, resembling the appearance of a crown on electron microscopic images, hence, the name coronavirus [1]. The isoelectric points (pI) of the various proteins—occupying the surface of the virus—ranges between pI = 6.24 for the spike protein S, pI = 8.57 for a small envelope protein, and pI = 9.51 for another membrane protein [2].

Replication rate (R_0) and infectivity of SARS-CoV-2

As described by Delamater., *et al.* (2019): "The basic reproduction number (R_0), also called the basic reproduction ratio or rate or the basic reproductive rate, is an epidemiologic metric used to describe the contagiousness or transmissibility of infectious agents" [3]. The

R_0 of a virus describes how many individuals will be infected by one infected carrier (with the virus) in a population susceptible to infection. For example, with $R_0 = 2$, one person will spawn, on average, two new cases of the infection or disease. The R_0 is derived by taking the number of infected cases over time and modeling the spread of the disease as one parameter to determine the shape of the curve [4]. Regarding SARS-CoV-2, the R_0 has been declining. However, in some cases, during the initial growth phase of the COVID-19 pandemic (such as occurred in Italy), the R_0 was between 2.43 and 3.10 [5].

SARS-CoV-2 viability on various surfaces

Researchers van Doremalen., *et al.* (2020) compared the viability of SARS-CoV-1 and SARS-CoV-2 in distinct environments: aerosol, cardboard, plastic, stainless steel, and copper. Viable SARS-CoV-2 titers decreased most strongly on copper and cardboard within and 8–12h and 24h until reaching the undetectable threshold, respectively [6].

Environmental conditions considered were a temperature between 21–23°C and relative humidity of 40%. The virus remained detectable on stainless steel for 48h, while it remained measureable up to 96h on a plastic surface [6]. The study did not precisely determine the virus' viablility in an aerosol complex. In aerosol form, the virus remained evident beyond 3h; however, the experiment did not continue beyond that point.

Future studies regarding the infectivity of SARS-CoV-2 aerosols for longer than three hours should prove consequential. SARS-CoV-2 demonstrates many stability similarities compared to SARS-CoV-1, suggesting that some of the treatments against the earlier coronavirus species might be effective against SARS-CoV-2 [6]. The half-life of SARS-CoV-2 on copper and as an aerosol is similar to that of SARS-CoV-1, further suggesting that the virus has similarly diminished viability in such circumstances.

The effect of climate temperature on SARS-CoV-2 inhibition and proliferation

Climate and weather conditions appear to influence the spread of SARS-CoV-2. The incidence of viral infections is higher in the colder winter months than in the warmer summer months. However, the specific causes of this connection have not been conclusively investigated.

Weather conditions directly influence viral stability, inhibition, proliferation, contamination vectors, and transmission. A less humid environment inhibits aerosol formation, diminishing viral transmission. Higher temperatures inactivate virus particles, resulting in less viral contamination of surfaces and virus transmission.

It has been hypothesized that SARS-CoV-2 thrives in colder climates compared to warmer climates. Araujo and Naimi (2020) used existing data and models to predict SARS-CoV-2 pandemic profiles across various climate zones and humidity variations. They found, with a 99% confidence interval, that the average interquartile range (IQR) of positive SARS-CoV-2 cases was associated with temperatures between -4.02°C and 15.58°C. (With a 95% confidence interval, the IQR stretches from -2.04°C to 9.49°C, regarding precipitation; the 99% confidence interval stretches from 4.68 mm to 116.06 mm; the 95% confidence interval lays between 19.75 mm and 94.43 mm [7]).

These data suggest that hot and humid climates are unlikely to provide areas and means of viral proliferation through the population. Instead, cooler and drier climates are more conducive to the development of such pandemics.

Cold temperate climates have the highest risk of SARS-CoV-2 pandemic spread, followed by warm temperate climates; arid, equatorial and polar climates have a much lower probability of developing a significant SARS-CoV-2 infection outbreak [7]. These findings have been confirmed by other studies, using original measurements (Sajadi., *et al.*, 2020) or modeling (Wang., *et al.*, 2020) [8,9]. Additional studies

by O'Reilly., *et al.* (2020) have pointed to the lower transmission rate of SARS-CoV-1 in warmer and humid climates [10]. However, Lai., *et al.* (2020) noted that those observational findings could have been confounded by delays from travel patterns [11].

For a pandemic to arise, there must be infected carriers. Using travel-network-based modeling with mobile phone data and airline itineraries, the researchers determined an approximate delay of up to two weeks before significant symptoms in infected people became noticeable due to travels to the original epicenter [11]. More accurate models may benefit from incorporating travel-patterns and symptom-delay metrics regarding SARS-CoV-2.

The effect of detergents on SARS-CoV-2

The determination of the degree a virus can be deactivated using detergent depends on the lipophilicity of its envelope. If that envelope is lipophilic, detergent molecules will insert into the viral envelope—disrupting the envelope and destroying the virus. SARS-CoV-2 and other members of the genus coronavirus, HPV, HIV, and influenza viruses have lipophilic envelopes. Thus, such viruses can be destroyed by detergents and other antimicrobial molecules, such as quaternary ammonium compounds (QACs), alcohols, proteases, and peroxides [12]. As CQ, HCQ, and other anti-malarial compounds have a lipophilic structure; theoretically, they could adversely impact SARS-CoV-2 (compromising or destroying the virus) [13].

The effect of disinfectants on SARS-CoV-2

Several experiments suggested that SARS-CoV-1 can be rapidly deactivated using common and less well-known disinfectants, such as isopropyl alcohol (70% or 100%), Desderman® N (a mixture of ethanol and biphenyl), and Sterillium® (a mixture of 1- and 2-propanol)—all were shown to deactivate viral particles in less than one minute (when mixed in a virus suspension). Formaldehyde and glutaraldehyde required two minutes to deactivate the virus [14].

Similar results were obtained using commercial antimicrobial disinfectants, such as Virugard®, Mikrobac®, Kohrsolin®, Dismozon®, and Korsolex®. Wine vinegar inhibited SARS-CoV-1 infectivity, suggesting its potential antimicrobial application for SARS-CoV-2 [15]. Fixatives, like methanol, acetone, formaldehyde, and glutaraldehyde, are viricidal. Hence, numerous options are available to effectively decontaminate surfaces and clean viruses from surfaces [16].

The effect of heat treatment on SARS-CoV-2

SARS-CoV-1 is resistant to heat treatment to some extent. While the titer will sink below the limit of detection (LOD) after 20 minutes at 56°C and after 4–10 minutes at 65°C. However, one hour or more is needed to inactivate the virus completely. At 75°C, extinction is achieved after 45 minutes. To date, a similar study on SARS-CoV-2 has not been undertaken. However, from the surface and aerosol experiments, the physicochemical properties of SARS-CoV-1 and SARS-CoV-2 are similar [17].

The effect of specific vitamins on SARS-CoV-2 infection

Vitamin C

Vitamin C, also referred to as ascorbic acid (AA), has been proposed as a treatment and prophylaxis for SARS-CoV-2 infections [18]. Vitamin C promotes and improves immune function. Manning., *et al.* (2013) found that AA advanced the maturation of T cells *in vitro* and *in vivo* [19]. Moreover, AA positively impacted epithelial barriers, serving as a first line of defense against germs, including viruses. According to Carr and Maggini (2017), AA bolstered the production of collagen fibers, stabilizing epithelial barriers in the skin and other body surfaces [20].

AA, as an antioxidant, protects against reactive oxygen species (ROS), stabilizing and enhancing tissue structures, and preventing long-term damage to specific cells. Moreover, AA boosts keratinocyte differentiation and augments the wound-healing process. Thus, AA may aid in the prevention, treatment, or recovery from SARS-CoV-2 or lessen damaging effects of a SARS-CoV-2 infection or COVID-19 [21].

Besides the maturation of T cells, AA supports phagocyte function, B cell differentiation, and antigen presentation, and regulates the inflammatory response by modulating cytokines and reducing histamine concentration. Thus, while AA does not directly interact with SARS-CoV-2, it enhances the immune system and reduces inflammatory propensity—indirectly lessening the adverse effects of SARS-CoV-2 infections [20,21].

A recent meta-analysis by Hemilä and Chalker (2020) confirmed that vitamin C administration significantly reduced the length of mechanical ventilation [22].

Vitamin D

Vitamin D has been described as preventing or mitigating SAR-CoV-2 infections [23]. SARS-CoV-2 transmission and infection appear greater during colder winter months—typically when sunlight is reduced and vitamin D levels are naturally lower—than in warmer summer months. Moreover, the virus spread appears less in the equatorial or near-equatorial regions due to a sustained warmer climate. Specific studies have shown the significant effect of latitude on vitamin D deficiencies: individuals living in regions far removed from the equator are more at risk for such a deficiency than equatorial inhabitants [24]. Moreover, advanced age and obesity have been established as comorbidities for SARS-CoV-2 infections, and both comorbidities are associated with vitamin D deficiency. According to Grant., *et al.* (2020), the lack of adequate vitamin D was linked to a higher risk for ARDS presentation—a severe symptom of a SARS-CoV-2 infection [23].

Grant., *et al.* (2020) also reported an inverse correlation between inadequate vitamin D levels and an increase in community-acquired pneumonia (CAP) [23]. This finding adds further credence that vitamin D could reduce symptoms of a SARS-CoV-2 infection or enhance the chances for recovery from such [23,25].

An inverse correlation also exists between diminished vitamin D levels and increases in CRP levels, risk of heart failure, risk of sepsis, and production of pro-inflammatory cytokines, such as IL-6. However, several clinical trials using vitamin D supplementation for several disease states have yielded mixed results. Vitamin D supplementation did not significantly resolve CAP, yet other trials showed that vitamin D supplementation suppressed the upregulation of IL-6 during inflammation reactions. Similar observations have been made for the reduction of CRP after vitamin D supplementation [23].

The effect of directed UV light on SARS-CoV-2

Current research is investigating UV light's role in reducing SARS-CoV-2 infections through the commercial installation of UV light-emitting devices into buildings accessed regularly by people. One company is developing the Headlight device, emitting UV light directly into susceptible or SARS-CoV-2-infected lung tissue [26].

Although high doses of focused UV light can kill coronaviruses in less than one minute, it is not entirely clear how an ambient level of UV will impact the spread of SARS-CoV-2 infections [27]. For example, researchers found no evidence between infection rate and cumulative incidence when correlating the number of SARS-CoV-2 infections in specific geographic areas in China [28]. Thus, it remains plausible that the wavelength, dosage, and duration of UV light exposure have a combined effect against a SARS-CoV-2 infections.

UV-irradiation reduces SARS-CoV-1 to the LOD in one minute; complete inactivation takes fifteen minutes. However, bovine serum albumin (BSA) led to a complete inactivation time of over sixty minutes [29]. Studies in which SARS-CoV-1 was irradiated with gamma-rays showed that the virus did not lose infectivity [29]— suggesting that the virus is remarkably stable in high-energy exposure.

Summary

Medical research is striving for the effective prevention and treatment of SARS-CoV-2 and COVID-19. However, will ongoing efforts be sufficient to contain and eliminate this pandemic—or will SARS-CoV-2 and its current and future variants inevitably prove too formidable of a foe for humankind to overcome? Only time will tell. However, the pace of research and control measures are accelerating, resulting in more focused and effective containment, treatment, and preventive measures.

Conflict of Interest Statement

The authors declare that this paper was written in the absence of any commercial or financial relationship that could be construed as a potential conflict of interest.

Supplementary Note

This paper is based on prior doctoral research: Chen M.H. (2019). "SARS-CoV-2: Dynamic Stimulation and Control of the Immune System by Integrated Therapies" (unpublished doctoral dissertation).

References

1. Cascella M., *et al*. "Features, evaluation and treatment coronavirus (COVID-19) (2020). https://www.ncbi.nlm.nih.gov/books/NBK554776/

2. Scheller C., *et al*. "Physicochemical properties of SARS-CoV-2 for drug targeting, virus inactivation and attenuation, vaccine formulation and quality control". *Electrophoresis* (2020). https://analyticalsciencejournals.onlinelibrary.wiley.com/doi/10.1002/elps.202000121

3. Delamater PL., *et al*. "Complexity of the Basic Reproduction Number (R0)". *Emerging Infectious Diseases* 25.1 (2019): 1-4. https://wwwnc.cdc.gov/eid/article/25/1/17-1901_article

4. Ma J. "Estimating epidemic exponential growth rate and basic reproduction number". *Infectious Disease Modelling* 5 (2020): 129-141. https://www.sciencedirect.com/science/article/pii/S2468042719300491

5. D'Arienzo M and Coniglio A. "Assessment of the SARS-CoV-2 basic reproduction number, R0, based on the early phase of COVID-19 outbreak in Italy". *Biosafety and Health* (2020). https://www.sciencedirect.com/science/article/pii/S2590053620300410

6. Van Doremalen N., *et al*. "Aerosol and surface stability of SARS-CoV-2 as compared with SARS-CoV-1". *New England Journal of Medicine* 382.16 (2020): 1564-1567. https://www.nejm.org/doi/full/10.1056/nejmc2004973

7. Araujo MB and Naimi B. "Spread of SARS-CoV-2 Coronavirus likely to be constrained by climate". *Med Rxiv* (2020). https://www.medrxiv.org/content/10.1101/2020.03.12.20034728v3

8. Sajadi MM., *et al*. "Temperature and latitude analysis to predict potential spread and seasonality for COVID-19 (2020). https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7290414/

9. Wang J., *et al*. "High temperature and high humidity reduce the transmission of COVID-19 (2020). https://www.cebm.net/study/covid-19-high-temperature-and-high-humidity-reduce-the-transmission-of-covid-19/

10. O'Reilly KM., *et al*. "Effective transmission across the globe: the role of climate in COVID-19 mitigation strategies". *The Lancet Planetary Health* (2020). https://www.thelancet.com/journals/lanplh/article/PIIS2542-5196(20)30106-6/fulltext

11. Lai S., *et al*. "Assessing spread risk of Wuhan novel coronavirus within and beyond China, January-April 2020: a travel network-based modelling study". *Med Rxiv* (2020). https://www.medrxiv.org/content/10.1101/2020.02.04.20020479v2

12. Kapil S., *et al*. "Rapid inactivation of SARS-like coronaviruses (No. SAND2004-1120). Sandia National Laboratories.fection". *Frontiers in Immunology* 9 (2004): 754. https://www.osti.gov/biblio/918739

13. Srivastava AK., *et al*. "In silico investigations on the potential inhibitors for COVID-19 protease". *arXiv Preprint arXiv* 3.11 (2003): 722-727. https://arxiv.org/abs/2003.10642

14. Scheller C., *et al*. "Physicochemical properties of SARS-CoV-2 for drug targeting, virus inactivation and attenuation, vaccine formulation and quality control". *Electrophoresis* (2020). https://analyticalsciencejournals.onlinelibrary.wiley.com/doi/10.1002/elps.202000121

15. Cascella M., *et al*. "Features, evaluation and treatment coronavirus (COVID-19) (2020). https://www.ncbi.nlm.nih.gov/books/NBK554776/

16. Kariwa H., *et al*. "Inactivation of SARS coronavirus by means of povidone-iodine, physical conditions and chemical reagents". *Dermatology* 212.1 (2006): 119-123. https://www.karger.com/Article/FullText/89211

17. Scheller C., *et al*. "Physicochemical properties of SARS-CoV-2 for drug targeting, virus inactivation and attenuation, vaccine formulation and quality control". *Electrophoresis* (2020). https://analyticalsciencejournals.onlinelibrary.wiley.com/doi/10.1002/elps.202000121

18. Hemilä H and Chalker E. "Vitamin C as a possible therapy for COVID-19". *Infection and Chemotherapy* (2020a): 52. https://pubmed.ncbi.nlm.nih.gov/32410417/

19. Manning J., *et al*. "Vitamin C promotes maturation of T-cells". *Antioxidants and Redox Signaling* 19.17 (2013): 2054-2067. https://app.dimensions.ai/details/publication/pub.1032704959

20. Carr AC and Maggini S. "Vitamin C and immune function". *Nutrients* 9.11 (2017): 1211. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5707683/

21. Desai M. "Coronavirus 2019 (COVID-19) - using ascorbic acid and zinc supplementation (COVIDAtoZ) (2020). https://clinicaltrials.gov/ct2/show/NCT04342728

22. Hemilä H and Chalker E. "Vitamin C may reduce the duration of mechanical ventilation in critically ill patients: a meta-regression analysis". *Journal of Intensive Care* 8.1 (2020): 15. https://jintensivecare.biomedcentral.com/articles/10.1186/s40560-020-0432-y

23. Grant WB., *et al*. "Evidence that vitamin D supplementation could reduce risk of influenza and COVID-19 infections and deaths". *Nutrients* 12.4 (2020): 988. https://pubmed.ncbi.nlm.nih.gov/32252338/

24. Leary PF., *et al*. "Effect of latitude on vitamin D levels". *Journal of the American Osteopathy Association* 117.7 (2017): 433-439. https://www.degruyter.com/document/doi/10.7556/jaoa.2017.089/html

25. Aranow C. "Vitamin D and the immune system". *Journal of Investigative Medicine* 59.6 (2011): 881-886. https://pubmed.ncbi.nlm.nih.gov/21527855/

26. Osborne H. "Russia developing coronavirus treatment that disinfects the body with UV light from inside (2020). https://www.newsweek.com/russia-developing-coronavirus-treatment-that-disinfects-body-uv-light-inside-1508350

27. Scheller C., *et al*. "Physicochemical properties of SARS-CoV-2 for drug targeting, virus inactivation and attenuation, vaccine formulation and quality control". *Electrophoresis* (2020). https://analyticalsciencejournals.onlinelibrary.wiley.com/doi/10.1002/elps.202000121

28. Yao Y., *et al*. "No association of COVID-19 transmission with temperature or UV radiation in Chinese cities". *European Respiratory Journal* 55.5 (2020c). https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7144256/

29. Darnell ME and Taylor DR. "Evaluation of inactivation methods for severe acute respiratory syndrome coronavirus in noncellular blood products". *Transfusion* 46.10 (2006): 1770-1777. https://pubmed.ncbi.nlm.nih.gov/17002634/

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