

On the Anti-Correlation Between COVID-19 Infection rate and Natural Ultra Violet Index

Arnon Blum ¹, Constantina Nicolaou ², Ben Henghes ², Ofer Lahav ^{2*}

¹Ariel University, Adelson School of Medicine, Laniado Hospital, Israel.

²Department of Physics & Astronomy, University College London, Gower Street, London, WC1E 6BT, UK.

***Corresponding Author:** Ofer Lahav, Department of Physics & Astronomy, University College London, Gower Street, London, WC1E 6BT, UK.

Received date: June 16, 2026; **Accepted date:** July 02, 2026; **Published date:** July 13, 2026

Citation: Arnon Blum, Constantina Nicolaou, Ben Henghes, Ofer Lahav, (2026), On the Anti-Correlation Between COVID-19 Infection rate and Natural Ultra Violet Index, *Clinical Research and Clinical Trials*, 15(5); DOI:10.31579/2693-4779/330

Copyright: © 2026, Ofer Lahav. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Abstract:

Background: Although social distancing and public health interventions are known to reduce the transmission of COVID-19, environmental factors may also influence infection rates. We investigated the hypothesis that natural ultraviolet (UV) radiation decreases COVID-19 infections by enhancing host immunity through increased vitamin D synthesis and nitric oxide production, as well as by exerting direct antiviral effects.

Methods: Daily COVID-19 infection data and ultraviolet index (UVI) measurements were analyzed for the United Kingdom from 23 March 2020 to 10 March 2021. The relationship between COVID-19 infections (F) and UVI was assessed using logarithmic transformation, and the correlation between $\log_{10}(F)$ and $\log_{10}(UVI)$ was calculated, with particular emphasis on the period following the first national lockdown (11 May 2020 to 10 March 2021).

Results: A strong inverse association was observed between COVID-19 infection rates and UVI. During the post-lockdown period, $\log_{10}(F)$ and $\log_{10}(UVI)$ demonstrated a marked anti-correlation, with a correlation coefficient of -0.934 , indicating that higher ambient UV exposure was associated with lower reported infection rates.

Conclusions: The strong inverse correlation between ambient UV radiation and COVID-19 infection rates suggests that environmental UV exposure may contribute to reducing viral transmission. Although this relationship does not establish causality and may reflect the influence of additional confounding factors associated with UVI, the findings support the inclusion of UVI as a variable in epidemiological models of COVID-19 infection and mortality. Further studies in additional countries and geographical regions are warranted to determine the generalizability of these observations and to clarify the underlying biological mechanisms.

Keywords: COVID-19; inflammatory cytokine storms; immunity; biological mechanisms; l-arginine

Introduction

Our hypothesis is that natural UV light suppresses the spread of the COVID-19 virus in at least two ways: by affecting the virus itself and by affecting human skin. We note that although natural UV radiation may cause skin cancer, it also generates vitamin D, which supports the immune system. There are three types of solar UV radiation, classified according to their wavelength. UVC is short-wavelength radiation (100–280 nm) and is the most damaging to the human body.

However, it is completely filtered by the atmosphere and does not reach the Earth's surface. It is well known that laboratory-produced UVC is used to inactivate viruses. UVB is medium wavelength radiation (280–315 nm), and most of it is filtered by the atmosphere. UVA is long wave length radiation (315–400 nm) and accounts for approximately 95% of the UV radiation reaching the Earth's surface. The UV Index (UVI) is a measure of the intensity of sun burn producing UV radiation at a particular place and time. Typical UVI values in the UK range from 0 to 8.

The possible correlation between UV light and COVID-19 has been discussed in the literature, with conflicting conclusions. One study [1] found no association between COVID-19 transmission and UV radiation in Chinese cities. Another study [2] reported only a modest impact of UV light and other environmental factors on reducing COVID-19 transmission. A further study [3] suggested that UV radiation may be less effective in areas with high levels of air pollution, where UV light is converted into heat. In contrast, several other studies [4–6] have reported that UV light is associated with a reduced COVID-19 growth rate. Given these conflicting findings regarding the impact of UV light on COVID-19 transmission, we take a fresh look at data from the UK. Our study was also motivated by the second wave of COVID-19 observed in many countries in the Northern Hemisphere during the winter.

Most respiratory viral infections exhibit a seasonal pattern that may be related to climatic changes, humidity, solar UV irradiation, latitude, air pollution, altitude, and human factors, including genetic, epigenetic, and

behavioral characteristics. Enveloped viruses, such as influenza A and B, generally exhibit greater stability at lower temperatures [7].

One study examined the climate of 50 cities affected by COVID-19 and found that eight cities had particularly high morbidity and mortality rates. All eight cities were located between latitudes 30°N and 50°N, with average temperatures ranging from 5°C to 11°C and low humidity. Countries located below latitude 35°N had lower COVID-19 mortality rates. In contrast, countries located above 35°N receive insufficient sunlight for adequate vitamin D synthesis. Vitamin D deficiency has been associated with hypertension, diabetes mellitus, obesity, and increased mortality rates [8–11].

Countries with the highest COVID-19 mortality rates are also known to have a high prevalence of vitamin D deficiency, including Italy, Spain, the UK, and France. In contrast, in Nordic countries, where sunlight exposure is limited, vitamin D food fortification is mandatory, and COVID-19 mortality rates were lower during the recent pandemic [12]. Milan is located at latitude 45°N, whereas Naples is located at latitude 40°N. Naples receives approximately 58 more sunny days per year than Milan [13]. During the study period, the COVID-19 death toll was 403 per million in Naples compared with 15,729 per million in Milan, representing more than a 39-fold difference. A study conducted in Scotland found that UVA exposure was inversely associated with the incidence of myocardial infarction, independent of temperature and UVB irradiation [14]. UVA penetrates the epidermis, reaching blood vessels, keratinocytes, and endothelial cells [15].

Sunlight activates nitric oxide (NO) in the skin. Nitric oxide is a potent modulator of the cardiovascular system, reducing blood pressure and peripheral vascular resistance [16–18]. It is also an important signaling molecule involved in maintaining vascular homeostasis, regulating cellular proliferation and inflammatory processes, and exerting antibacterial and antiviral effects [19–21]. Under normal physiological conditions, NO is produced in endothelial cells through the oxidation of L-arginine to nitric oxide and citrulline. However, inducible nitric oxide synthase (iNOS) is calcium-independent and is activated during conditions of acute or chronic inflammation and infection [22–25]. Nitric oxide has been shown to inhibit SARS-CoV replication by interfering with fusion between the viral spike (S) protein and its receptor, angiotensin converting enzyme, and by inhibiting viral RNA replication [26]. Vitamin D stimulates the production of nitric oxide (NO) in the vascular endothelium, which helps prevent severe COVID-19 by protecting blood vessels, blocking viral replication through cellular autophagy, and suppressing dangerous inflammatory cytokine storms [27].

UVA penetrates the dermal layer, reaching keratinocytes, fibroblasts, microvascular endothelial cells, keratinocytes, Langerhans cells, dermal fibroblasts, and melanocytes- all have the capacity to express inducible nitric oxide synthase (iNOS) following activation by cytokines [27, 28, 29].

Methods

The data used in this study are publicly available. COVID-19 data were obtained from the Johns Hopkins University (JHU) COVID-19 database, and ultraviolet index (UVI) data were obtained from TEMIS. The data was free for the public and we didn't need to recruit patients or humans to explore our questions.

Institutional Review Board Statement: the study is on public free data, and there was no need for an Institutional Review Board. The study was on published data free for all, and did not recruit patients or any subjects.

Informed Consent Statement: there was no need for an informed consent in a study that is based on historical data, published free on line, so that researchers could use to their analysis.

In our study, we focused on the UK, using COVID-19 data [29] and UVI data for London [30]. Although the UVI data were obtained for London, the variation in UVI across different locations in the UK is within 0.8 UVI units. The stringency index represents the lockdown measures implemented in the UK and is defined in [31].

Results

Figure 1 shows the daily numbers of infections and deaths in the UK, together with the UVI and stringency index, for the period from 22 January 2020 to 10 March 2021. Visual inspection of the data reveals a strong anti-correlation between daily infections and the UVI.

As expected, the UVI increased from January 2020 to July 2020, when it reached its peak, and then declined. From January to April 2020, while the UVI was increasing, the number of infections also increased. The UK government imposed its first national lockdown on 23 March 2020, resulting in a decrease in infections due to social distancing measures. The lockdown was relaxed on 11 May 2020. However, the increase in the UVI between 23 March and 1 July 2020 may also have contributed to the decline in the number of infections. From 2 July to October 2020, the increase in infections was strongly anti-correlated with the UVI, as quantified in Table 1 and illustrated in Figure 1. In November 2020, the number of cases declined following the implementation of a second national lockdown but increased again after the restrictions were lifted. During this period, the UVI remained low and therefore may not have contributed to reducing the number of cases.

To quantify the correlation observed in Figure 1, we calculated the correlation coefficient between X and Y , as defined in Eq. (1), where μ represents the mean and σ the standard deviation. We applied this analysis to $X = \log_{10}(\text{UVI})$ and $Y = \log_{10}(F)$. For the period from 23 March 2020 (the date on which the first UK lockdown was imposed) to 10 March 2021, the correlation coefficient was $\rho = -0.917$, with a p-value of 1.80×10^{-14} , indicating a strong inverse correlation and providing strong evidence against the null hypothesis of no correlation. In addition, for the period from 11 May 2020 (when the initial relaxation of lockdown measures began) to 10 March 2021, the correlation coefficient was $\rho = -0.934$, with a p-value of 2.24×10^{-136} . During this period, the stringency index remained relatively stable, varying by approximately 13%; therefore, the effect of changes in lockdown measures was likely to be limited.

The correlation coefficients (ρ) for different time intervals and time lags of 7 and 14 days are presented in Table 1. Applying a time lag produced only minor changes in the correlation coefficient, probably because multiple competing effects obscured any specific lag effect. Figure 2 shows the daily numbers of infections and the UVI in the upper panel, while the lower panel presents the rolling correlation coefficient between $\log_{10}(\text{UVI})$ and $\log_{10}(F)$ using a 50-day moving window. A negative correlation between COVID-19 infections and the UVI is evident from mid-April onwards.

Study period	No lag	7-day lag	14-day lag
23 Mar 2020 – 10 Mar 2021	−0.917	−0.910	−0.886
11 May 2020 – 10 Mar 2021	−0.934	−0.922	−0.896
2 Jul 2020 – 10 Mar 2021	−0.927	−0.910	−0.879
28 Oct 2020 – 10 Mar 2021	−0.751	−0.738	−0.611

Table 1: Pearson correlation coefficients (ρ) between $\log_{10}(\text{UVI})$ and $\log_{10}(F)$ for different study periods and time lags. The selected study periods correspond to key phases of the COVID-19 pandemic in the UK: the first national lockdown (23 March–11 May 2020), the subsequent relaxation of restrictions, the period following the minimum number of reported infections (approximately 1 July 2020), and the onset of the second national lockdown after 28 October 2020. Bootstrap analysis showed that the standard error of all correlation coefficients was < 0.05 .

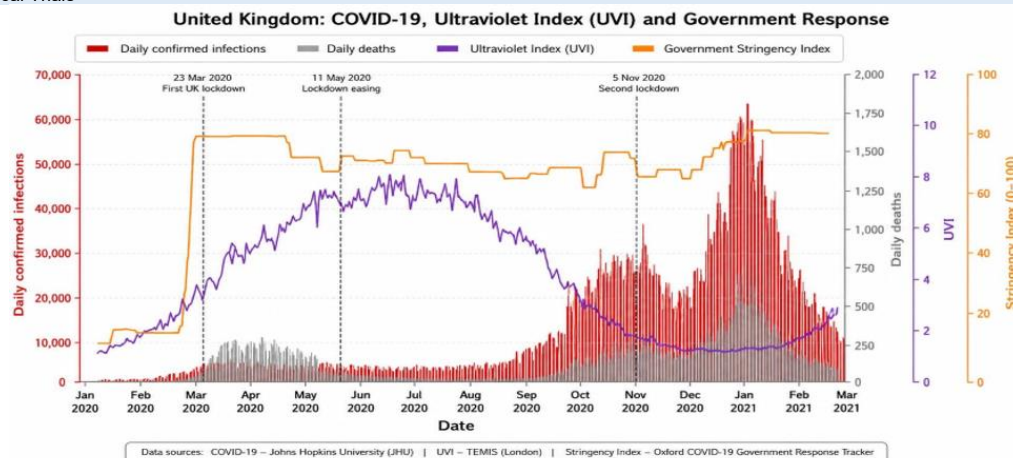


Figure 1: Daily confirmed COVID-19 infections (dark blue) and deaths (light blue) in the United Kingdom, together with the Oxford COVID-19 Government Response Stringency Index (orange) and the ultraviolet index (UVI; red), from 22 January 2020 to 10 March 2021. The figure illustrates the temporal relationship between the progression of the COVID-19 epidemic, seasonal variation in UVI, and the implementation of government restrictions. A marked inverse relationship between COVID-19 infection rates and UVI is apparent following the relaxation of the first national lockdown in May 2020.

Discussion

In conclusion, we found an intriguing empirical anti-correlation between the daily UVI and COVID-19 infections in the UK, with a correlation coefficient of $\rho = -0.934$ between 11 May 2020 and 10 March 2021. We are currently extending our analysis to other countries and regions. At present, we highlight the example of Chile as a Southern Hemisphere case. During the periods of increasing (25 March to 6 June 2020) and decreasing (7 June to 28 October 2020) infection rates, we found anti-correlations of $\rho = -0.907$ and $\rho = -0.730$, respectively, consistent with the anti-correlation observed in the UK. Another important comparison would be between countries with similar environmental conditions but different lockdown policies, such as Norway and Sweden.

We emphasize that a correlation between two variables does not necessarily imply causation. If UV light directly influences infection rates, it may do so by reducing viral survival, enhancing the immune response through increased vitamin D production, or through a combination of both mechanisms. Alternatively, the UVI may serve as a surrogate marker for other factors. For example, low UVI values may reflect increased time spent indoors (e.g., at home or in shops), thereby increasing opportunities for viral transmission, or may coincide with other winter-related illnesses that increase susceptibility to infection. Additional factors that may contribute include population density, temperature, humidity, air pollution, and other geographical or environmental variables.

Conclusions

We emphasize that COVID-19 transmission is influenced by multiple factors. There is strong empirical evidence that social distancing and mask wearing are important measures for reducing viral transmission. It is also clear that vaccination represents the most effective strategy for controlling COVID-19. Nevertheless, we advocate including UV exposure as a parameter in models of COVID-19 transmission and considering its potential role in future public health and medical strategies.

References

1. Yao, Y. et al. No association of COVID-19 transmission with temperature or UV radiation in Chinese cities. *European Respiratory Journal* 55. issn: 0903-1936.
2. Xu, R. et al. The Modest Impact of Weather and Air Pollution on COVID-19 Transmission.
3. Yudistira, N., Sumitro, S. B., Nahas, A. & Riama, N. F. UV light influences covid-19 activity through big data: tradeoffs between

northern subtropical, tropical, and southern subtropical countries.

4. Merow, C. & Urban, M. C. (2020). Seasonality and uncertainty in global COVID-19 growth rates. *Proceedings of the National Academy of Sciences*.
5. Nicastro, F. et al. Modulation of COVID-19 Epidemiology by UV-B and -A Photons from the Sun.
6. Cherrie, M. et al. Ultraviolet a Radiation and COVID-19 Deaths: A Multi Country Study.
7. Price, R. H. M., Graham, C. & Ramalingam, S. (2019). Association between viral seasonality and meteorological factors. English. *Scientific Reports* 9.
8. Kunutsor, S. K., Apekey, T. A. & Steur, M. (2013). Vitamin D and risk of future hypertension: meta-analysis of 283,537 participants. *European Journal of Epidemiology*; 28, 205–221.
9. Mauss, D., Jarczok, M. N., Hoffmann, K., Thomas, G. N. & Fischer, J. E. (2015). Association of Vitamin D Levels with Type 2 Diabetes in Older Working Adults. *International Journal of Medical Sciences*; 12, 362–368.
10. Yao, Y. et al. (2015). A meta-analysis of the relationship between vitamin D deficiency and obesity. *International Journal of Clinical and Experimental Medicine*; 8, 14977–14984.
11. Herrick, K. A. et al. (2019). Vitamin D status in the United States, 2011-2014. *American Journal of Clinical Nutrition* 110, 150–157.
12. Lips, P. et al. (2019). Current vitamin D status in European and Middle East countries and strategies to prevent vitamin D deficiency: a position statement of the European Calcified Tissue Society. *European Journal of Endocrinology*; 180, P23–P54.
13. Whittemore, P. B. (2020). COVID-19 fatalities, latitude, sunlight, and vitamin D. *American Journal of Infection Control* 48, 1042–1044.
14. Mackay, D. F. et al. (2019). UVA and Seasonal Patterning of 56 370 Myocardial Infarctions Across Scotland, 2000-2011. *Journal of The American Heart Association* 8.
15. Holick, M. F. (2016). Biological Effects of Sunlight, Ultraviolet Radiation, Visible Light, Infrared Radiation and Vitamin D for Health. English. *Anticancer Research* 36, 1345–1356.
16. Weller, R. B. (2017). The health benefits of UV radiation exposure through vitamin D production or non-vitamin D pathways. Blood pressure and cardiovascular disease. *Photochemical & Photobiological Sciences* 16, 374–380.

17. Liu, D. et al. (2014). UVA Irradiation of Human Skin Vasodilates Arterial Vasculature and Lowers Blood Pressure Independently of Nitric Oxide Synthase. *Journal of Investigative Dermatology* 134, 1839–1846.
18. Oplaender, C. et al. (2009). Whole-Body UVA Irradiation Lowers Systemic Blood Pressure by Release of Nitric Oxide from Intracutaneous Photolabile Nitric Oxide Derivates. *Circulation Research* 105, 1031–U215.
19. Pope, M. et al. (1998). Resistance to murine hepatitis virus strain 3 is dependent on production of nitric oxide. *Journal of Virology* 72, 7084–7090.
20. Adler, H. et al. (1997). Suppression of Herpes simplex virus type 1 (HSV-1) induced pneumonia in mice by inhibition of inducible nitric oxide synthase (iNOS, NOS2). *Journal of Experimental Medicine* 185. AAAAI/AAI/CIS Joint Meeting, San Francisco, CA, FEB 21-26, 1997, 1533–1540.
21. Lane, T., Paoletti, A. & Buchmeier, M. (1997). Disassociation between the in vitro and in vivo effects of nitric oxide on a neurotropic murine coronavirus. *Journal of Virology* 71, 2202–2210.
22. Akaïke, T. et al. (1996). Pathogenesis of influenza virus-induced pneumonia: Involvement of both nitric oxide and oxygen radicals. *Proceedings of The National Academy of Sciences of The United States of America* 93, 2448–2453.
23. Coleman, J. (2001). Nitric oxide in immunity and inflammation. *International Immunopharmacology* 1, 1397–1406.
24. Poljakovic, M., Karpman, D., Svanborg, C. & Persson, K. (2002). Human renal epithelial cells express iNOS in response to cytokines but not bacteria. *Kidney International* 61, 444–455.
25. Saxena, S., Mathur, A. & Srivastava, R. Induction of nitric oxide synthase during Japanese encephalitis virus infection: Evidence of protective role. *Archives of Biochemistry and Biophysics* 391, 1–7.
26. Akerstrom, S., Gunalan, V., Keng, C. T., Tan, Y.-J. & Mirazimi, A. (2009). Dual effect of nitric oxide on SARS-CoV replication: Viral RNA production and palmitoylation of the S protein are affected. *Virology* 395, 1–9.
27. Holliman, G., Lowe, D., Cohen, H., Felton, S. & Raj, K. (2017). Ultraviolet Radiation-Induced Production of Nitric Oxide: A multi-cell and multi-donor analysis. *Scientific Reports* 7.
28. Kuchel, J., Barnetson, R. & Halliday, G. (2003). Nitric oxide appears to be a mediator of solar simulated ultraviolet radiation-induced immunosuppression in humans. *Journal of Investigative Dermatology* 121, 587–593.
29. Dong, E., Du, H. & Gardner, L. (2020). An interactive web-based dashboard to track COVID-19 in real time. *Lancet Inf Dis.* 20, 533–534.
30. Van Geffen, J., Van Weele, M., Allaart, M. & Van der A, R. (2017). TEMIS UV index and UV dose operational data products, version 2. Dataset.
31. Hale, T., Webster, S., Petherick, A., Phillips, T. & Kira, B. (2020). Oxford COVID-19 Government Response Tracker.
32. Efron, B. (1979). Bootstrap Methods: Another Look at the Jackknife. *Ann. Statist.* 7, 1–26.



This work is licensed under Creative Commons Attribution 4.0 License

To Submit Your Article Click Here:

Submit Manuscript

DOI:10.31579/2693-4779/329

Ready to submit your research? Choose Auctores and benefit from:

- fast, convenient online submission
- rigorous peer review by experienced research in your field
- rapid publication on acceptance
- authors retain copyrights
- unique DOI for all articles
- immediate, unrestricted online access

At Auctores, research is always in progress.

Learn more <https://auctoresonline.org/journals/clinical-research-and-clinical-trials>