UVB radiation alone may not explain sunlight inactivation of SARS-CoV-2

Paolo Luzzatto-Fegiz*[1], Fernando Temprano-Coleto[1], François J. Peaudecerf[2], Julien R. Landel[3], Yangying Zhu[1], Julie A. McMurry[4]

1 Department of Mechanical Engineering, University of California, Santa Barbara, CA, USA ;

2 Institute for Environmental Engineering, ETH Zürich, Switzerland;

3 Department of Mathematics, University of Manchester, UK;

4 Translational and Integrative Sciences Center, Department of Molecular Toxicology, Oregon State University, OR, USA

* Corresponding author. Email: pfegiz@ucsb.edu , Phone: +1-805-455-9094

Key words

SARS-CoV-2, COVID-19, environmental persistence, sunlight, UVA, UVB, modeling, inactivation methods, photobiology

Recently, Ratnesar-Shumate et al. [1] reported rapid sunlight inactivation of SARS-CoV-2 in simulated saliva and in complete growth medium (gMEM). Independently and essentially simultaneously, Sagripanti & Lytle [2] introduced a theory for sunlight inactivation of SARS-CoV-2, building on their earlier work for similar viruses [3]. To the best of our knowledge, these data and theory have yet to be compared; when establishing this comparison, the experimentally reported sunlight inactivation in [1] is several times faster than predicted by theory, suggesting that additional experiments and hypotheses may be needed to fully elucidate the mechanism of SARS-CoV-2 sunlight inactivation.

Briefly, the theory of Sagripanti & Lytle [2,3] considers direct photochemical damage to viral RNA, which is maximal for UVC (wavelengths between 200-280 nm). The effectiveness of UVC is expressed as the exposure that produces one e-fold reduction in infectious virion concentration (i.e. to 37% of the initial value) at a wavelength of 254 nm, which is written as D_{37} [3]. Since larger D_{37} implies slower inactivation, D_{37} is effectively an inverse sensitivity. Based on genome size, for *Coronaviridae*, Lytle & Sagripanti estimated D_{37} between 2.5-3.9 J/m^2, and D_{37} = 3.0 J/m^2 for SARS-CoV-2 [2]; this value is used in the calculations presented here. Although no UVC reaches the Earth's surface, longer UV wavelengths can still affect viral RNA, albeit with decreased sensitivity. To account for this, Lytle & Sagripanti [3] introduced an action spectrum, expressed as the ratio between sensitivity at a given wavelength λ and the UVC sensitivity at 254 nm [3]. Writing this relative sensitivity as $r(\lambda)$, and expressing the spectral irradiance at a given wavelength as $E_{e,\lambda}(\lambda)$, one can evaluate an "equivalent UVC" irradiance (in W/m^2) as

$$E_{\text{equiv}} = \int r(\lambda)\, E_{e,\lambda}(\lambda)\, \mathrm{d}\lambda \,. \qquad (1)$$

Since $r(\lambda)$ drops to around 10^{-4} by a wavelength of 320 nm, this integral is performed only over the UVB spectrum (280 to 315 nm). In the calculations reported here, the $r(\lambda)$ is the one

compiled in [3], the irradiance spectra of [1] are used for $E_{e,\lambda}(\lambda)$, and the integral is performed numerically. The infectious virion concentration V would decay with time t as

$$V(t) = V(0) \exp[-(k_0 + E_{equiv} / D_{37}) t] , \quad (2)$$

where k_0 is the inactivation rate in the dark, which is negligible in the experiments of [1].

As shown in figure 1, the experimentally observed inactivation rates from [1] are significantly faster than the theoretical ones from equation (2). Furthermore, achieving a good fit to the data would require a UVB sensitivity that is beyond the largest values reported for any virus, to the best of our knowledge [3]. As a matter of fact, the experimentally-observed inactivation in simulated saliva is over eight times faster than would have been expected from the theory. Even in gMEM, inactivation is over three times faster than expected from theory. Although one might attempt to explain this significant difference in inactivation rates by considering the difference in light attenuation within each medium, this effect alone would still lead to slower inactivation relative to theory, contrary to what has been reported by the experiments of Ratnesar-Shumate et al. [1], and therefore is not sufficient to explain the disagreement between theory and experiments.

This discrepancy suggests that additional hypotheses should be tested for the sunlight inactivation mechanism. Other mechanisms of sunlight inactivation are known to exist for other viruses, beside direct nucleic acid damage, as reviewed by Nelson et al. [4]. For example, sunlight in the UVA wavelength range may interact with sensitizer molecules in the medium, yielding photo-produced reactive intermediates that can damage the virus [5]. If sensitivity to wavelengths other than UVB were to be found, sunlight could mitigate outdoor transmission over a broader range of latitudes and daytimes than previously expected. Furthermore, inexpensive and energy-efficient wavelength-specific light sources might be used to augment air filtration systems at relatively low risk for human health, especially in high-risk settings such as hospitals and public transportation.

Overall, these results point to the need for additional experiments, in order to separately test the effects of specific illumination wavelengths and of medium composition.

Figure 1. Comparison for SARS-CoV-2 on stainless steel surfaces for different media, showing $TCID_{50}$ versus time for different suspension media and simulated sunlight intensities. Symbols: data of Ratnesar-Shumate et al. [1] plotted with grey dots; for clarity, means at each time are also plotted with open circles, with error bars showing standard deviation. Purple solid line: UVB-inactivation theory of [3] with SARS-CoV-2 inverse sensitivity D_{37} = 3.0 J/m^2, from [2]. Green dotted line: UVB-inactivation theory of [3] with D_{37} from a fit to all data for a given medium. (*A, B, C*) show simulated saliva; (*D, E, F*), gMEM (complete growth medium). (*A, D*) show high simulated sunlight; (*B, E*) medium simulated sunlight; (*C, F*) low simulated sunlight.

References

1. Ratnesar-Shumate S, Williams G, Green B, et al. Simulated Sunlight Rapidly Inactivates SARS-CoV-2 on Surfaces. J Infect Dis. **2020**; 222(2):214–222.

2. Sagripanti J-L, Lytle CD. Estimated Inactivation of Coronaviruses by Solar Radiation With Special Reference to COVID-19. Photochem Photobiol. **2020**; 96(4):731–737.

3. Lytle CD, Sagripanti J-L. Predicted inactivation of viruses of relevance to biodefense by solar radiation. J Virol. **2005**; 79(22):14244–14252.

4. Nelson KL, Boehm AB, Davies-Colley RJ, et al. Sunlight-mediated inactivation of health-relevant microorganisms in water: a review of mechanisms and modeling approaches. Environ Sci Process Impacts. **2018**; 20(8):1089–1122.

5. Kohn T, Nelson KL. Sunlight-mediated inactivation of MS2 coliphage via exogenous singlet oxygen produced by sensitizers in natural waters. Environ Sci Technol. **2007**; 41(1):192–197.

Conflict of interest statement:

J McMurry is a cofounder of Pryzm Health, a bioinformatics company focusing on rare Mendelian diseases.

The other authors report no conflict of interest.

Funding statement:

This work was supported by the University of California, Santa Barbara [Vice Chancellor for Research COVID-19 Seed Grant] and by the Army Research Office Multi University Research Initiative [W911NF-17-1-0306 to P.L.-F.].

Meetings where the information has previously been presented:

Part of this work appeared as a poster presentation in the meeting "Climatological, Meteorological, and Environmental factors in the COVID-19 pandemic", 4-6 August 2020. https://public.wmo.int/en/events/meetings/covid-19-symposium

Corresponding author contact information:

Paolo Luzzatto-Fegiz

Department of Mechanical Engineering

University of California, Santa Barbara

CA 93106

pfegiz@ucsb.edu

805-455-9094

Figure 1