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CORRESPONDENCE

UVB Radiation Alone May Not Explain Sunlight Inactivation of SARS-CoV-2

To the Editor—Recently, Ratnesar-Shumate et al [1] reported rapid sunlight inactivation of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) in simulated saliva and in complete growth medium (gMEM). Independently and essentially simultaneously, Sagripanti and Lytle [2] introduced a theory for sunlight inactivation of SARS-CoV-2, building on their earlier work with similar viruses [3]. To the best of our knowledge, these data and theory had not been compared. When establishing this comparison, the experimentally reported sunlight inactivation in Ratnesar-Shumate et al [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 and Lytle [2, 3] considers direct photochemical damage to viral RNA, which is maximal for UVC (wavelengths between 200 and 280 nm). The effectiveness of UVC is expressed as the exposure that produces one e-fold reduction in infectious virion concentration (ie, to 37% of the initial value) at a wavelength of 254 nm, which is written as D_{37} [3]. Because larger D_{37} implies slower inactivation, D_{37} is effectively an inverse sensitivity. Based on genome size, for Coronaviridae, Lytle and Sagripanti estimated D_{37} between 2.5 and 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 and Sagripanti [3] introduced an action spectrum, expressed as the ratio between sensitivity at a given wavelength λ and the UVC sensitivity at 254 nm. 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

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

Because $r(\lambda)$ 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 by Lytle and Sagripanti [3], the irradiance spectra of Ratnesar-Shumate et al [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

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

where k_0 is the inactivation rate in the dark, which is negligible in the experiments of Ratnesar-Shumate et al [1].

As shown in Figure 1, the experimentally observed inactivation rates from Ratnesar-Shumate et al [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 8 times faster than would have been expected from the theory. Even in gMEM, inactivation is over 3 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 photoproduced 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 to separately test the effects of specific illumination wavelengths and of medium composition.

Notes

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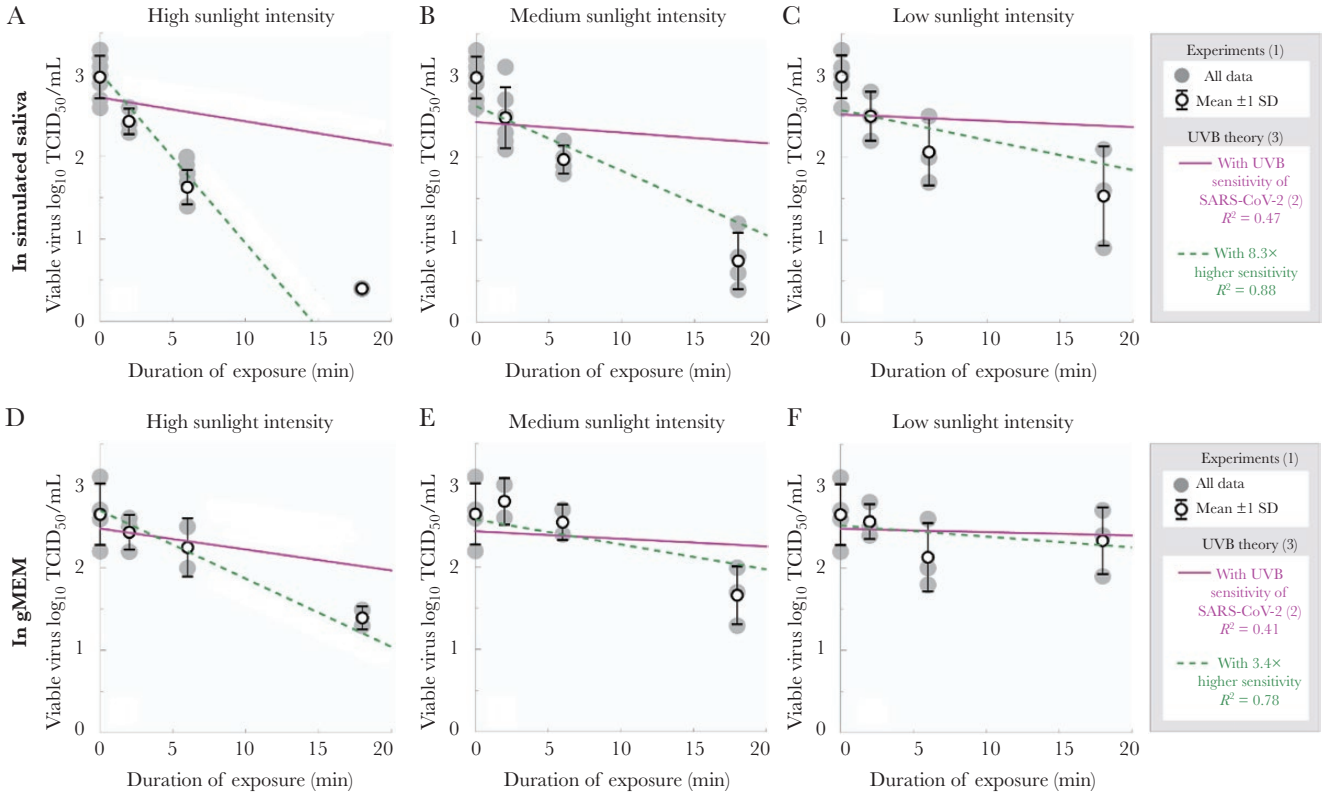

Figure 1. SARS-CoV-2 $TCID_{50}$ versus length of exposure to different simulated sunlight intensities and in different suspension media: (*A–C*) in simulated saliva; (*D–F*) in gMEM; (*A* and *D*) high simulated sunlight intensity; (*B* and *E*) medium simulated sunlight intensity; and (*C* and *F*) low simulated sunlight intensity. Data of Ratnesar-Shumate et al [1] are plotted with grey dots and means at each time point with open circles; error bars are standard deviations. Solid lines indicate the UVB-inactivation theory of Lytle and Sagripanti [3] with SARS-CoV-2 inverse sensitivity D_{37} = 3.0 J/m^2, from Sagripanti and Lytle [2]. Dotted lines indicate the UVB-inactivation theory of Lytle and Sagripanti [3] with D_{37} from a fit to all data for a given medium. Abbreviations: gMEM, complete growth medium; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; $TCID_{50}$, 50% tissue culture infectious dose.

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