Rapid Communication

Estimated Inactivation of Coronaviruses by Solar Radiation With Special Reference to COVID-19

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Received 19 May 2020, accepted 1 June 2020, DOI: 10.1111/php.13293

ABSTRACT

Using a model developed for estimating solar inactivation of viruses of biodefense concerns, we calculated the expected inactivation of SARS-CoV-2 virus, cause of COVID-19 pandemic, by artificial UVC and by solar ultraviolet radiation in several cities of the world during different times of the year. The UV sensitivity estimated here for SARS-CoV-2 is compared with those reported for other ssRNA viruses, including influenza A virus. The results indicate that SARS-CoV-2 aerosolized from infected patients and deposited on surfaces could remain infectious outdoors for considerable time during the winter in many temperate-zone cities, with continued risk for re-aerosolization and human infection. Conversely, the presented data indicate that SARS-CoV-2 should be inactivated relatively fast (faster than influenza A) during summer in many populous cities of the world, indicating that sunlight should have a role in the occurrence, spread rate and duration of coronavirus pandemics.

INTRODUCTION

The current (2019-2020) COVID-19 world pandemic is caused by a member of the Coronavirusidae family [Reviewed in (1)]. Coronaviruses have a lipid-containing envelope with the genome consisting of a single-stranded, positive-sense RNA genome that is not segmented (2–5). Coronavirus have the largest genomes of all ssRNA viruses which will become of relevance latter in the work. In the absence of pandemics, coronaviruses cause about 15–20% of all upper respiratory infections in humans (6). Previous pandemics like Severe Acute Respiratory Syndrome (caused by SARS-CoV during 2002–2003), and Middle East Respiratory Syndrome (caused by MERS-CoV during 2012) indicate that pandemics caused by coronaviruses should be expected to occur with frequency (7,8). Additional coronaviruses are known to cause disease in animals closely associated to humans like cat and dog, rat and mouse, cow, swine, chicken and turkey (6).

Although clusters of infected family members and medical workers have confirmed direct, person-to-person transmission (9), the rapid expansion of COVID-19, that progressed unquenched even after quarantine of nearly one-third of the world population and major social distancing measures, suggests that an environmental component (with the virus remaining infectious outside the host) plays a role in disease transmission. Of relevance here is the amount of infectious virus present in the aerosolized droplets produced by COVID-19 symptomatic patients or nonsymptomatic carriers. This amount is not well established for coronaviruses, but it has been reported that nasal secretions contain up to 107 infectious influenza viral particles per ml (10), from which aerosolized droplets generated by coughing, sneezing and talking can contain several hundred infectious virions (11). These micro droplets can reach distances of 12.5 meters (over 40 feet, (12)). SARS-CoV has been reported to persist on contaminated surfaces with risk of disease transmission for up to 96 h (13) and other coronaviruses for up to 9 days (14). SARS-CoV-2 persisted viable from 3 h to 3 days depending on the type of surface on which it was deposited (15). Influenza virus was readily re-aerosolized by sweeping floors without much loss in infectivity (16). It must be assumed that SARS-CoV-2 will be re-aerosolized in a similar manner.

Three main physical factors generally considered with a potential effect on virus persistence outdoors, include temperature, humidity and the contribution of sunlight. The survival of influenza virus, a member of the Orthomyxoviridae family, also with ssRNA and a lipid-containing envelope, only varied up to 9% when the relative humidity changed between 50% and 70% (17). Rather extreme changes in relative humidity between 15% and 90% varied survival of influenza 12.5–fold [1.1 Log10, (18)]. In these studies, virus survival was even less influenced by changes in temperature. A recent study where virus infectivity was corrected by aerosol losses and natural decay, demonstrated that aerosolized influenza A virus remained equally infectious at all relative humidity tested, ranging from 23% to 98% (19). In agreement with the relatively small effect of humidity and temperature on influenza virus inactivation, epidemiological studies concluded that the mortality increase in winter was largely independent of temperature and humidity (20,21).

If the limited role of relative humidity and temperature (within the range encountered in the environment) reported for influenza A parallels that for SARS-CoV-2 then, the effect of artificial and natural UV radiation on SARS-CoV-2 inactivation should be pre-eminent. The pre-eminent effect indoors of germicidal UV (UVC, 254 nm) radiation is clearly confirmed by a report
whereby inactivation of air-borne virions by UV radiation virtu-
ally prevented the spread of influenza among patients in a vet-
erans hospital, during the same time that an epidemic of influenza 
ravaged similar patients in nearby nonirradiated rooms (22).

There are published reports indicating that very high doses of 
UVC are effective for inactivating SARS-CoV-2 or SARS-CoV 
that had been added to different blood products or remaining in 

virus culture medium (23–28) but there is no data on the viral 
sensitivity to UVC in UV-transparent liquids or in absence of 
protective substances, as needed to estimate UVC sensitivity. 
Nor is there information for UVC inactivation of the virus sus-
pended in aerosols or deposited on surfaces as needed for envi-
ronmental risk assessment.

Ultraviolet radiation in sunlight is the primary virucidal agent 
in the environment (29–31). This notion is supported by the cor-
relation found in Brazil between increased influenza incidence in 

hospital admission records and solar UV-blocking by smoke dur-
ing the burning season (32). The reports on influenza A warrant 
the present study to estimate UV sensitivity of SARS-CoV-2 and 
its possible role in the COVID-19 pandemic.

The purpose of this study was twofold, (1) to estimate the 
sensitivity of SARS-CoV-2 to inactivation by germicidal UV 
(UVC) and (2) to predict the inactivation of the virus by the 

UVB in sunlight for various populous cities of the world at dif-
ferent times of the year. These goals were achieved by utilizing 
a model developed for biodefense purposes for estimating solar 
UVB inactivation of dangerous viruses (30). This methodology 
has been validated with Ebola and Lassa viruses (33). The model 
has also been used to estimate inactivation of influenza viruses at 
various times in numerous locations in the U.S. and globally 
(34).

Estimation of the time required for inactivation of 90% and 
99% of infectious virus reported here should be useful in evalu-
ating the persistence of SARS-CoV-2 in environments exposed 
to solar radiation.

MATERIALS AND METHODS

We estimated SARS-CoV-2 virus UV (254 nm) sensitivity and inactivation 
at different U.S. and global locations by an approach originally developed 
to predict the survival of viruses of interest in biodefense (30) and later 
employed to estimate persistence of influenza A virus (34).

**SARS-COV 2 virus UV254 sensitivity.** The UVC sensitivity is reported 
here as D37 which corresponds to the UV fluence that produces, on 
average, one lethal hit to the virus, resulting in 37% survival. D37 equals 
the reciprocal of the slope on the semi-logarithmic graph of viral survival 
versus dose and can be calculated by dividing the fluence that results in 
1 Log10 reduction of virus load by 2.3 (the natural logarithmic base). A 
lower value of D37 indicates a higher sensitivity to inactivation by UV 
radiation. Comparison of a virus of unknown UVC sensitivity to that of 
other viruses of similar genomic structure allows an estimate to be 
determined (30). An important part of the method is the fact that UVC 
sensitivities of viruses depends proportionally on genome size, especially 
with single-stranded RNA or DNA, that is, the larger the genome “target”, 
the more sensitive (and lower D37). This results in the product 
the genome size and the D37, defined as size normalized sensitivity 
(SnS), being relatively constant for a given type of viral genome (30) 
and it is used in this study to compare viruses with ssRNA genomes. 
This approach has been used successfully to estimate the UVC 
sensitivities of Ebola and Lassa viruses, later confirmed experimentally 
in the laboratory (33), thus validating the method.

**Solar intensity at different locations and times of year.** Solar UVB 
flux measured by the USDA UVB Monitoring and Research Program 
(35) have been used in the development and testing of the method (30). 
Maximum daily solar UVB fluence values for the selected locations at 
specific times of year have been presented in a previous article predicting 
the inactivation of influenza A by solar UVB (34). Those daily solar flux 
values were normalized using a virucidal action spectrum to 254 nm 
equivalent levels (30). Whereas the total UV254 equivalent fluence per 
full day was previously used in the influenza A inactivation study (34), 
the fluence values at solar noon are preferable and are used here because 
they are essentially constant during two hours (36,37). It has been 
previously determined that 35% of the total daily UVB occurs in the 
two-hour period (120 min) around solar noon (37). Thus 35% of the total 
daily UVB fluence divided by 120 min yields the noontime UVB flu 
(in J m⁻² min⁻¹) at the locations and times of the year presented in 
Tables 2 and 3. It should be noted that the solar UVB fluence used in 
the present study assumed no atmospheric influence, whether by haze, clouds 
or air pollution. Also, there was no correction for an increase in the solar 
virucidal effect due to the elevation of the urban sites (38).

RESULTS

UVC sensitivity of SARS-CoV-2

In Table 1 we compare the genomic and UV254 characteristics of 
SARS-CoV-2 (causing COVID-19) with those of other coron-
aviruses and viruses that have similar nucleic acid composition. 
The first three coronaviruses cause disease in humans. Studies 
with MHV and EtoV have found similar values for D37,8 (36,39). 
Therefore, a reasonable estimate for the D37,8 for the SARSs and 
MERS-CoV viruses would be 3.0 J m⁻². Comparison with other 
ssRNA viruses yields a similar D37 value. Since the influenza A 
 genomes are 2.2 times shorter than those of the coronaviruses, it 
is further reasonable that the coronaviruses (larger UV targets) 
would be at least twice as sensitive to UVC; the reciprocal ratio 
the genome sizes times the D37 for the influenza viruses yields 
an estimated D37 for SARS-CoV-2 of 4.7 J m⁻². When a similar 
comparison is done with the viruses of the other ssRNA families 
in Table 1, the median value for the SARS-CoV-2 D37 was 
5.0 J m⁻². The D37 value of 3.0 J m⁻² was used in the following 
calculations because it follows from values derived directly 
from members of the same Coronaviridae family; D10 
(6.9 J m⁻²) was used as it represents 10% survival (90% inactivation).

It may be useful to estimate the solar exposure for 99% virus 
inactivation (1% survival) or for even lower levels of survival. 
Because the material in aerosols created by COVID-19 patients 
and carriers may shield the virus from the UV as has been 
shown in laboratory experiments with viruses in culture medium, 
the virus survival curves indicate that the virus apparently 
becomes less UV sensitive (33,36,40–42). This resulted in a 
change of slope of approximately 4-fold in experiments with 
Ebola, Lassa and influenza A viruses and affected several percent 
of the virus population (33,42). Therefore, for survival beyond 
10%, a UV fluence of 4 times the chosen D10 (28 J m⁻²) was 
assumed. This value was used to estimate the solar exposure 
needed for 99% inactivation. Assuming that the survival curve 
maintains that 4-fold greater UV resistance at lower survival 
levels, 99.9% inactivation (disinfection level) would require 
56 J m⁻²; sterilization level inactivation (10⁻⁶ survival) would 
require 140 J m⁻².

Estimated time for inactivation of SARS-CoV-2 virus

Table 2 shows reported solar virucidal flux at solar noon together 
with the estimated minutes of sunlight exposure needed at vari-
ous populous North American metropolitan areas to inactivate 
90% of SARS-CoV-2. The (+) sign in Table 2 indicates that 
99% of SARS-CoV-2 may be inactivated within the two hours
period around solar noon during summer in most US cities located south of Latitude 43°N. Also 99% of the virus will be inactivated during two hours midday in several cities south of latitude 35°N in Fall, but only Miami and Houston will receive enough solar radiation to inactivate 99% of the virus in spring. During winter, most cities will not receive enough solar radiation to produce 90% viral inactivation during 2-hour midday exposure (underlined values in Table 2).

Table 3 presents germicidal solar flux values and resulting inactivation of SARS-CoV-2 for populous metropolitan areas on other continents. The values in Tables 2 and 3 clearly illustrate that SARS-CoV-2 in environments exposed to sunlight will be differentially inactivated in different cities and at different times of the year. For example, at winter solstice (December, in the northern hemisphere), just at the beginning of the COVID-19 pandemic, virus exposed to full midday sunlight would be reduced by at least 90% (1 Log10) during 22 min in Mexico City, and will be receiving enough germicidal solar flux to inactivate 99% of virus as indicated by (+) in Table 3. A 90% inactivation of SARS-CoV-2 in December should have taken considerably longer time in Shanghai (99 min), and Cairo (86 min). Nearly full virus persistence should occur in winter (December) in the European cities listed in Table 3 (where COVID-19 was severe). Of course, the same trend applies to the Southern Hemisphere where winter begins in June and 90% of SARS-CoV-2 should be inactivated in 41 min in Sao Pablo (Brazil), but not within the 2 hours solar noon period in Buenos Aires (Argentina) or Sydney (Australia) in the incoming winter season.

**DISCUSSION**

The transmission of viral infections and evolution of pandemics is a multi-factorial process involving, among others, properties of the viral agent, health condition of the host and available health care, viral inactivation in the environment, social dynamics and political decisions. It is well known that there is direct transmission of infectious virions by inhalation of contaminated aerosols exhaled, coughed or sneezed from infected persons, allowing for little time and opportunity for environmental viral inactivation, unless the virions settle on some surface. Although direct (person-to-person) transmission is important between nearby individuals (9), it is remarkable that the COVID-19 pandemic progressed at a sustained rate even after one-third of the world population was in quarantine or in-house lock-down (50). The rapid progression of the COVID-19 pandemic, in spite of greatly hindered direct transmission, supports elucidating the relevance of indirect infection through aerosolized virus, contact with contaminated surfaces and other fomites, and the inactivation thereof.

Changes in relative humidity and ambient temperature have been reported as having a rather limited effect on environmental virus survival and disease transmission (17–21). In contrast, UVC radiation has considerable virucidal effect (22). The methodology employed in the present study has been used previously to estimate the UVC sensitivity of Lassa virus and other viruses of relevance in biodefense (30). A close agreement was obtained between UVC D37 values predicted for Lassa virus and other viruses of relevance in biodefense (30). A close agreement was obtained between UVC D37 values predicted for Lassa virus (member of the Arenavirus family) (13 J m−2, table 4 in Ref 30) and measured years later in the laboratory (16 J m−2) (33). These results suggest that the accuracy of the methodology used here to estimate the UV sensitivity of the SARS-CoV-2 virus from data obtained for members of the same family may be within 20%.

The relevance of sunlight in viral inactivation contrasts with and is supported by the (1) long-term persistence in darkness of smallpox (an Orthopoxivirus) in scabs and surfaces (51), (2) with laboratory results where pathogenic viruses in the dark survived

Table 2. Calculated maximum* virucidal (254-nm equivalent) UV flux during two-hour period around solar noon for populous metropolitan areas in North America at specified times of year. Effectiveness estimated for inactivation of SARS-CoV-2 virus

<table>
<thead>
<tr>
<th>Metropolitan area</th>
<th>Latitude</th>
<th>Summer Solstice</th>
<th>Spring</th>
<th>Fall</th>
<th>Winter Solstice</th>
</tr>
</thead>
<tbody>
<tr>
<td>Miami, FL</td>
<td>25.8°N</td>
<td>0.51/14</td>
<td>0.34/20</td>
<td>0.41/17</td>
<td>0.13/53</td>
</tr>
<tr>
<td>Houston, TX</td>
<td>29.8°N</td>
<td>0.44/9</td>
<td>0.25/8</td>
<td>0.33/21</td>
<td>0.08/66</td>
</tr>
<tr>
<td>Dallas, TX</td>
<td>32.8°N</td>
<td>0.39/18</td>
<td>0.20/34</td>
<td>0.28/25</td>
<td>0.06/115</td>
</tr>
<tr>
<td>Phoenix, AZ</td>
<td>33.4°N</td>
<td>0.39/18</td>
<td>0.19/36</td>
<td>0.26/27</td>
<td>0.05/138†</td>
</tr>
<tr>
<td>Atlanta, GA</td>
<td>33.7°N</td>
<td>0.39/18</td>
<td>0.18/38</td>
<td>0.26/27</td>
<td>0.05/138</td>
</tr>
<tr>
<td>Los Angeles, CA</td>
<td>34.1°N</td>
<td>0.38/18</td>
<td>0.18/38</td>
<td>0.26/27</td>
<td>0.05/138</td>
</tr>
<tr>
<td>San Francisco, CA</td>
<td>37.7°N</td>
<td>0.34/20</td>
<td>0.13/53</td>
<td>0.20/34</td>
<td>0.03/230</td>
</tr>
<tr>
<td>Washington, D.C.</td>
<td>38.9°N</td>
<td>0.33/21</td>
<td>0.12/57</td>
<td>0.19/36</td>
<td>0.02/300</td>
</tr>
<tr>
<td>Philadelphia, PA</td>
<td>39.9°N</td>
<td>0.32/22</td>
<td>0.11/63</td>
<td>0.18/38</td>
<td>0.02/300</td>
</tr>
<tr>
<td>New York City, NY</td>
<td>40.7°N</td>
<td>0.32/22</td>
<td>0.10/69</td>
<td>0.17/41</td>
<td>0.02/300</td>
</tr>
<tr>
<td>Chicago, IL</td>
<td>41.9°N</td>
<td>0.31/22</td>
<td>0.09/77</td>
<td>0.15/46</td>
<td>0.01/300</td>
</tr>
<tr>
<td>Boston, MA</td>
<td>42.3°N</td>
<td>0.30/23</td>
<td>0.09/77</td>
<td>0.15/46</td>
<td>0.01/300</td>
</tr>
<tr>
<td>Detroit, MI</td>
<td>42.4°N</td>
<td>0.30/23</td>
<td>0.09/77</td>
<td>0.15/46</td>
<td>0.01/300</td>
</tr>
<tr>
<td>Toronto, Ontario</td>
<td>43.6°N</td>
<td>0.29/24</td>
<td>0.08/86</td>
<td>0.14/49</td>
<td>0.01/300</td>
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<tr>
<td>Minneapolis, MN</td>
<td>45.0°N</td>
<td>0.28/25</td>
<td>0.07/99</td>
<td>0.13/53</td>
<td>0.01/300</td>
</tr>
<tr>
<td>Seattle, WA</td>
<td>47.6°N</td>
<td>0.26/27</td>
<td>0.06/115</td>
<td>0.11/63</td>
<td>0.01/300</td>
</tr>
</tbody>
</table>

*Maximum solar exposure with no clouds, haze, air pollution or shadows to reduce exposure, independent of site elevation. Obtained using the virus inactivation action spectrum normalized to unity at 254 nm (30). Methodology: Maximum daily solar UVB fluence values for the selected locations at specific times of year have been reported in Tables 1 and 2 in the previous article on predicted influenza inactivation by solar UVB (34). 35% of the total daily UVB fluence divided by 120 min yields the noontime UVB flux in J m⁻² min⁻¹ at the locations and times in Tables 2 and 3. The UVB fluence Dₜ₀ to inactivate SARS-CoV-2 90% (10% survival) was estimated as 6.9 J m⁻². "°" denotes that under ideal conditions, solar UV could inactivate SARS-CoV-2 99% (1% survival) during 2-hour period around solar noon. Four times the Dₜ₀ under ideal conditions, solar UV could inactivate SARS-CoV-2 99% (1% survival) during 2-hour period around solar noon.

...for much longer times (Tₗₗ [time to 37% survival]) between 15 and 43 h for the different viruses studied (52), and (3) with the rapid inactivation of vaccinia virus exposed to direct sunlight or simulated solar UVB (42).

The solar germicidal flux shown in Tables 2 and 3 allows estimating SARS-CoV-2 inactivation outdoors for the cities presented, as well as for almost any other location for which latitude is known, from sun exposure under clear skies. Modeling of viruses suspended in the atmosphere indicates that the diffuse (scatter) component of sunlight may still have approximately 50% of the virucidal efficacy exerted by direct solar radiation (38,53). These findings demonstrate that viral inactivation by sunlight continues outdoors (albeit at half the rate or less) even in the shade or in polluted air or partially cloudy days.

Although the solar zenith angle at a given location is the same at the spring and fall equinoxes, the solar UV radiation received in the northern hemisphere was generally greater in the fall than in the spring, except for the location furthest south, Hawaii (latitude 19.5°N). Data for Alexandria, New Zealand, in the southern hemisphere where the seasons are reversed, demonstrated the same trend with spring UVB radiation being lower than fall UV radiation (data not shown). This differential solar germicidal fluence between spring and summer has been previously discussed (30).

Data for the COVID-19 pandemics from the World Health Organization and from Johns Hopkins’ Center for Systems Science and Engineering (as of May 7, 2020) indicates that of the 30 countries with highest infections per million inhabitants, 28 were north of the Tropic of Cancer (the two exceptions being Qatar and Mayotte) (54). Any correlation between solar flux during December-March 2019/20, (when COVID-19 was in expansion) and infection rate is limited by inaccuracy and availability of testing, different numbers of infected travelers, as well as vast differences on each country demographics and response. However, the statistical data (as of May 7 2020 (54)) suggest that COVID-19 may have progressed differently in countries at northern latitudes where it was winter and sun exposure was limited at the onset of the pandemic, than in countries in the southern latitudes where summer sunlight was abundant.

Considering that SARS-CoV-2 is three times more sensitive to UV than influenza A (as presented in Table 1 and discussed in RESULTs) it should be inferred that sunlight should have an effect on coronaviruses transmission at least similar to that previously established for the evolution of influenza epidemics (22,32). If we accept a possible virucidal role of sunlight during coronavirus pandemics, then forcing people to remain indoors may have increased (or assured) contagion of COVID-19 among same house-hold dwellers and among patients and personnel inside the same hospital or geriatric facilities. In contrast, healthy people outdoors receiving sunlight could have been exposed to lower viral dose with more chances for mounting an efficient immune response. This argument supports considering the results of two opposed containment approaches to deal with the COVID-19 crisis.
rather implemented large-scale social distancing, face mask wearing measures and/or instituted quarantine mainly for travelers and infected patients (57).

Analyzing the value (if any) of whole-population quarantine or in-house lock-down of healthy individuals is beyond the scope of the present work. However, the freely available epidemiological data (as of May 29, 2020 (54)) demonstrates that lock-down measures preventing healthy individuals from remaining outdoors have not resulted in an obvious and statistically significant difference on infections per million inhabitants when compared to countries where healthy individuals were free to stay outdoors, with potential exposure to sunlight radiation. If lock-down of healthy citizens may not be determinant as these statistics suggest, then the potential role of being outside exposed to direct or scattered sunlight in COVID-19 pandemic should not be underestimated.

**CONCLUSION**

The data presented estimates the sensitivity to UVC (254 nm) of the SARS-CoV-2 virus with a $D_{37}$ of 3.0 J m$^{-2}$, corresponding to 90% inactivation ($D_{10}$) after a dose of 7 J m$^{-2}$. Inactivation of 99% viral load ($D_{1}$) was estimated to be 28 J m$^{-2}$ ($4 \times D_{10}$) due to the biphasic nature of the virus inactivation curve found for other viruses shielded by culture media and other components that accompany virus infections.

90% or more of SARS-CoV-2 virus will be inactivated after being exposed for 11-34 min of midday sunlight in most US and world cities during summer. In contrast, the virus will persist for 1-3 days in winter (December–March), with risk of re-aerosolization and transmission in most of these cities.

Although latitude, population size, public health and control measures vastly vary among countries, the viral persistence estimated here for cities at northern latitudes where COVID-19 expanded rapidly during winter 2019–2020 and relatively higher viral inactivation in more southern latitudes receiving high solar radiation during the same period, suggests an environmental role for sunlight in the COVID-19 pandemic.

**Acknowledgements**—The authors appreciate the encouragement to initiate this study received from Ms. Jessica Seigel (journalist, New York University).

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### Table 3. Calculated maximum* virucidal (254-nm equivalent)$^3$ UV for two-hour period around solar noon for selected major world cities at specified times of year: Effectiveness estimated for inactivation of SARS-CoV-2 virus

<table>
<thead>
<tr>
<th>City</th>
<th>Latitude</th>
<th>Summer Solstice</th>
<th>Spring</th>
<th>Equinox</th>
<th>Fall</th>
<th>Winter Solstice</th>
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<tbody>
<tr>
<td>Central and South America</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Bogota, Colombia</td>
<td>4.6 °N</td>
<td>0.64$/11^+$</td>
<td>0.64$/11^+$</td>
<td>0.64$/11^+$</td>
<td>0.64$/11^+$</td>
<td>0.64$/11^+$</td>
</tr>
<tr>
<td>Mexico City, Mexico</td>
<td>19.5 °N</td>
<td>0.64$/11^+$</td>
<td>0.62$/11^+$</td>
<td>0.62$/11^+$</td>
<td>0.31$/22^+$</td>
<td></td>
</tr>
<tr>
<td>Sao Paulo, Brazil</td>
<td>23.3 °S</td>
<td>0.55$/13^+$</td>
<td>0.40$/17^+$</td>
<td>0.48$/14^+$</td>
<td>0.17$/41^+$</td>
<td></td>
</tr>
<tr>
<td>Buenos Aires, Argentina</td>
<td>34.6 °S</td>
<td>0.37$/19^+$</td>
<td>0.17$/41</td>
<td>0.24$/29</td>
<td>0.04$/172^+$</td>
<td></td>
</tr>
<tr>
<td>Europe</td>
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<tr>
<td>Barcelona, Spain</td>
<td>41.4 °N</td>
<td>0.31$/22^+$</td>
<td>0.10$/69</td>
<td>0.16$/43</td>
<td>0.01$/300</td>
<td>0.05$/300</td>
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<td>48.9 °N</td>
<td>0.25$/28^+$</td>
<td>0.05$/138</td>
<td>0.10$/69</td>
<td>0.00$/300</td>
<td>0.00$/300</td>
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<td>London, UK</td>
<td>51.5 °N</td>
<td>0.23$/30</td>
<td>0.04$/173</td>
<td>0.09$/77</td>
<td>0.00$/300</td>
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<td>Moscow, Russia</td>
<td>55.7 °N</td>
<td>0.20$/34</td>
<td>0.03$/230</td>
<td>0.07$/99</td>
<td>0.00$/300</td>
<td>0.00$/300</td>
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<td>Middle East</td>
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<td></td>
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<tr>
<td>Baghdad, Iraq</td>
<td>33.3 °N</td>
<td>0.39$/18+</td>
<td>0.19$/36</td>
<td>0.26$/27+</td>
<td>0.05$/138</td>
<td>0.05$/138</td>
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<tr>
<td>Tehran, Iran</td>
<td>35.7 °N</td>
<td>0.36$/19+</td>
<td>0.16$/43</td>
<td>0.23$/30+</td>
<td>0.04$/172</td>
<td>0.04$/172</td>
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<tr>
<td>Istanbul, Turkey</td>
<td>41.0 °N</td>
<td>0.31$/22+</td>
<td>0.10$/69</td>
<td>0.16$/43</td>
<td>0.02$/300</td>
<td>0.02$/300</td>
</tr>
<tr>
<td>Africa</td>
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<td></td>
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</tr>
<tr>
<td>Kinshasa, Congo</td>
<td>4.3 °S</td>
<td>0.64$/11+</td>
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</tr>
<tr>
<td>Lagos, Nigeria</td>
<td>6.4 °N</td>
<td>0.64$/11+</td>
<td>0.64$/11+</td>
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<td>0.64$/11+</td>
<td>0.64$/11+</td>
</tr>
<tr>
<td>Khartoum, Sudan</td>
<td>15.6 °N</td>
<td>0.64$/11+</td>
<td>0.64$/11+</td>
<td>0.64$/11+</td>
<td>0.32$/22+</td>
<td>0.08$/86</td>
</tr>
<tr>
<td>Cairo, Egypt</td>
<td>30.0 °N</td>
<td>0.43$/16+</td>
<td>0.25$/28+</td>
<td>0.32$/22+</td>
<td>0.08$/86</td>
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<tr>
<td>Mumbai (Bombay), India</td>
<td>19.0 °N</td>
<td>0.64$/11+</td>
<td>0.62$/11+</td>
<td>0.62$/11+</td>
<td>0.32$/22+</td>
<td>0.08$/86</td>
</tr>
<tr>
<td>Shanghai, China</td>
<td>31.2 °N</td>
<td>0.42$/16+</td>
<td>0.22$/31</td>
<td>0.31$/22+</td>
<td>0.07$/99</td>
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</tr>
<tr>
<td>Seoul, Republic of Korea</td>
<td>33.5 °N</td>
<td>0.38$/18+</td>
<td>0.19$/36</td>
<td>0.26$/27+</td>
<td>0.05$/138</td>
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<tr>
<td>Tokyo, Japan</td>
<td>35.7 °N</td>
<td>0.36$/20+</td>
<td>0.16$/43</td>
<td>0.23$/30+</td>
<td>0.04$/172</td>
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<tr>
<td>Australia</td>
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<td></td>
</tr>
<tr>
<td>Sydney, Australia</td>
<td>33.9 °S</td>
<td>0.38$/18+</td>
<td>0.18$/38</td>
<td>0.26$/27+</td>
<td>0.05$/138</td>
<td></td>
</tr>
</tbody>
</table>

*Maximum solar exposure with no clouds, haze, air pollution or shadows to reduce exposure, independent of site elevation. Obtained using the virus inactivation action spectrum normalized to unity at 254 nm (30). Methodology: Maximum daily solar UVB fluence values for the selected locations at specific times of year have been represented in Tables 1 and 2 in the previous article on predicted Influenza inactivation by solar UVB (34). 35% of the total daily UVB fluence divided by 120 min yields the noontime UVB flux in J m$^{-2}$ min$^{-1}$ at the locations and times in Tables 2 and 3. The UVB fluence $D_{10}$ to inactivate SARS-CoV-2 90% (10% survival) was estimated as 6.9 J m$^{-2}$. Under ideal conditions, solar UV could inactivate SARS-CoV-2 99% (1% survival) during 2-h period around solar noon. Four times the $D_{10}$ was chosen to account for the likely bi-phasic inactivation due to protective elements surrounding the virus. Underlined values indicate solar UVB is likely not enough to inactivate SARS-CoV-2 90% (10% survival) during two-hour period around solar noon. Flux values above 0.62 are likely underestimates. Therefore, the time for 90% and 99% inactivation are possibly overestimates.