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Rapid Communication

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

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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 *Coronaviridae* 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). Coronaviruses have the largest genomes of all ssRNA viruses which will become of relevance latter in this 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 10^7 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 Log_{10}, (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 preeminent. The pre-eminent effect indoors of germicidal UV (UVC, 254 nm) radiation is clearly confirmed by a report

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whereby inactivation of air-borne virions by UV radiation virtually prevented the spread of influenza among patients in a veterans 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 suspended in aerosols or deposited on surfaces as needed for environmental risk assessment.

Ultraviolet radiation in sunlight is the primary virucidal agent in the environment (29–31). This notion is supported by the correlation found in Brazil between increased influenza incidence in hospital admission records and solar UV-blocking by smoke during 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 different 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 evaluating 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-CO V2 virus UV_{254} sensitivity. The UVC sensitivity is reported here as D_{37} which corresponds to the UV fluence that produces, on average, one lethal hit to the virus, resulting in 37% survival. D_{37} 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 Log_{10} reduction of virus load by 2.3 (the natural logarithmic base). A lower value of D_{37} 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 D_{37}). This results in the product of the genome size and the D_{37}, 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 UV_{254} equivalent fluence per full day was previously used in the influenza A inactivation study (34), the flux 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 flux (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 flux 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 UV_{254} characteristics of SARS-CoV-2 (causing COVID-19) with those of other coronaviruses 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 D_{37}s (36,39). Therefore, a reasonable estimate for the D_{37}s for the SARSs and MERS-CoV viruses would be 3.0 J m^{-2}. Comparison with other ssRNA viruses yields a similar D_{37} 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 of the genome sizes times the D_{37} for the influenza viruses yields an estimated D_{37} 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 D_{37} was 5.0 J m^{-2}. The D_{37} 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; D_{10} (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 D_{10} (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-Co V-2 virus

Table 2 shows reported solar virucidal flux at solar noon together with the estimated minutes of sunlight exposure needed at various 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

Table 1. UVC sensitivity of SARS-CoV-2 and selected viruses.*

Virus family	Genome	Size† (Knt)	Measured‡ D_{37} (J m^{-2})	SNS§ (J m^{-2} Knt)	Predicted D_{37} (J m^{-2})	References
Coronaviridae						
SARS-CoV-2	ssRNA+	29.8		89	3.0	
SARS-CoV	ssRNA+	29.7		89	3.0	
MERS	ssRNA+	30.1		89	3.0	
MHV	ssRNA+	31.6	2.9	91		(36)
EToV	ssRNA+	28.5	3.1	88		(39)
Togaviridae						
SINV	ssRNA+	11.7	19	220		(43)
VEEV	ssRNA+	11.4	23	260		(44)
SFV	ssRNA+	13.0	7.2	94		(39)
Paramyxiviridae						
NDV	ssRNA-	15.2	11-13.5	170-210		(45,46)
MeV	ssRNA-	15.9	8.8-10.9	140-170		(47)
Orthomyxoviridae						
FLUAV	ssRNA-	13.6				
Melbourne H1N1			10.2	139		(48)
NIB-4 H3N2-3			11	150		(40)
NIB-6 H1N1			9.6	131		(40)
ISAV	ssRNA-	14.5	4.8	70		(49)
Rhabdoviridae						
RABV	ssRNA-	11.9	4.3	51		(39)

*Selected viruses of different genetic Families having ssRNA as the genome. †Size of the genome expressed as thousands of nucleotide bases (Knt). ‡UVC fluence that causes one lethal event per virus on average, resulting in 37% survival. §Size-normalized sensitivity defined as the product of the D_{37} and the genome size in thousands of bases is relatively constant for a given genome type, but can be vastly different for different genomic types. If the size and genome type is known for an untested virus, the D_{37} can be predicted from the SNS.

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 Log_{10}) 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 D_{37} 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

		Solar virucidal UV flux (J/m^2_{254} ^{2}/min)‡/Time for 90% Infectivity reduction (min)§			
			Equinox		
Metropolitan area	Latitude	Summer Solstice	Spring	Fall	Winter Solstice
Miami, FL	25.8 °N	0.51/***14*** +‖	0.34/***20*** +	0.41/***17*** +	0.13/***53***
Houston, TX	29.8 °N	0.44/***16*** +	0.25/***28*** +	0.33/***21*** +	0.08/***86***
Dallas, TX	32.8 °N	0.39/***18*** +	0.20/***34***	0.28/***25*** +	0.06/***115***
Phoenix, AZ	33.4 °N	0.39/***18*** +	0.19/***36***	0.26/***27*** +	0.05/***138***¶
Atlanta, GA	33.7 °N	0.39/***18*** +	0.18/***38***	0.26/***27*** +	0.05/***138***
Los Angeles, CA	34.1 °N	0.38/***18*** +	0.18/***38***	0.26/***27*** +	0.05/***138***
San Francisco, CA	37.7 °N	0.34/***20*** +	0.13/***53***	0.20/***34***	0.03/***230***
Washington, D.C.	38.9 °N	0.33/***21*** +	0.12/***57***	0.19/***36***	0.02/>***300***
Philadelphia, PA	39.9 °N	0.32/***22*** +	0.11/***63***	0.18/***38***	0.02/>***300***
New York City, NY	40.7 °N	0.32/***22*** +	0.10/***69***	0.17/***41***	0.02/>***300***
Chicago, IL	41.9 °N	0.31/***22*** +	0.10/***69***	0.16/***43***	0.01/***>300***
Boston, MA	42.3 °N	0.30/***23*** +	0.09/***77***	0.15/***46***	0.01/***>300***
Detroit, MI	42.3 °N	0.30/***23*** +	0.09/***77***	0.15/***46***	0.01/***>300***
Toronto, Ontario	43.6 °N	0.29/***24***	0.08/***86***	0.14/***49***	0.01/***>300***
Minneapolis, MN	45.0 °N	0.28/***25***	0.07/***99***	0.13/***53***	0.01/***>300***
Seattle, WA	47.6 °N	0.26/***27***	0.06/***115***	0.11/***63***	0.01/***>300***

*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}. ‖"+" 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 D_{10} was chosen to account for the likely biphasic 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.

for much longer times (T_{37} [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 Alexandra, 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.

Almost all countries and territories affected with COVID-19 have closed their borders, mandated the use of masks and promoted social distancing. By 26 March, 2020, 1.7 billion people worldwide were under some form of lock-down, which increased to 3.9 billion people by the first week of April, amounting to more than half of the world's population (55). Schools, universities and colleges have closed either on a nationwide or local basis in 177 countries, affecting approximately 98.6 percent of the world's student population (56). In addition to these measures, some countries (for example: Italy, Spain, the UK, Peru, Chile, Argentina and Rep South Africa) implemented nationwide strict quarantine and in-house lock-down measures, often enforced by police, that decreased the time individuals could spent outdoors thus preventing potential exposure to sunlight. Most countries (like USA, Finland and Brazil) implemented regional less stringent lock-down measures at varying degrees. A third group of countries (for example: Sweden, Belorussia, Nicaragua, Uruguay, Indonesia, South Korea and Namibia) did not mandate lock-downs that prevented healthy individuals to remain outdoors with potential exposure to sunlight (57). These "unlock" countries have not enforced any strict lock-downs but have

Table 3. Calculated maximum* virucidal (254-nm equivalent†) UV flux 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

		Solar virucidal UV flux (J/m^2_{254} ^{2}/min)‡/Time for 90% Infectivity reduction (min)§			
			Equinox		
City	Latitude	Summer Solstice	Spring	Fall	Winter Solstice
Central and South America					
Bogota, Colombia	4.6 °N	0.64#/***11***+‖	0.64/***11***+	0.64/***11***+	0.64/***11***+
Mexico City, Mexico	19.5 °N	0.64/***11***+	0.62/***11***+	0.62/***11***+	0.31/***22***+
São Paulo, Brazil	23.3 °S	0.55/***13***+	0.40/***17***+	0.48/***14***+	0.17/***41***
Buenos Aires, Argentina	34.6 °S	0.37/***19***+	0.17/***41***	0.24/***29***	0.04/***172***¶
Europe					
Barcelona, Spain	41.4 °N	0.31/***22***+	0.10/**69**	0.16/**43**	0.01/**>300**
Paris, France	48.9 °N	0.25/***28***+	0.05/**138**¶	0.10/**69**	0.00/**>300**
London, UK	51.5 °N	0.23/***30***	0.04/**173**	0.09/**77**	0.00/**>300**
Moscow, Russia	55.7 °N	0.20/***34***	0.03/**230**	0.07/**99**	0.00/**>300**
Middle East					
Baghdad, Iraq	33.3 °N	0.39/**18**+	0.19/**36**	0.26/**27**+	0.05/**138**
Tehran, Iran	35.7 °N	0.36/**19**+	0.16/**43**	0.23/**30**	0.04/**172**
Istanbul, Turkey	41.0 °N	0.31/**22**+	0.10/**69**	0.16/**43**	0.02/**>300**
Africa					
Kinshasa, Congo	4.3 °S	0.64/**11**+	0.64/**11**+	0.64/**11**+	0.64/**11**+
Lagos, Nigeria	6.4 °N	0.64/**11**+	0.64/**11**+	0.64/**11**+	0.64/**11**+
Khartoum, Sudan	15.6 °N	0.64/**11**+	0.64/**11**+	0.64/**11**+	0.32/**22**+
Cairo, Egypt	30.0 °N	0.43/**16**+	0.25/**28**+	0.32/**22**+	0.08/**86**
Asia					
Mumbai (Bombay), India	19.0 °N	0.64/**11**+	0.62/**11**+	0.62/**11**+	0.32/**22**+
Shanghai, China	31.2 °N	0.42/**16**+	0.22/**31**	0.31/**22**+	0.07/**99**
Seoul, Republic of Korea	33.5 °N	0.38/**18**+	0.19/**36**	0.26/**27**+	0.05/***138***
Tokyo, Japan	35.7 °N	0.36/**20**+	0.16/**43**	0.23/**30**	0.04/***172***
Australia					
Sydney, Australia	33.9 °S	0.38/**18**+	0.18/**38**	0.26/**27**+	0.05/***138***

*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 biphasic 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.

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× 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 infectious for a day or more 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.

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