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Environmental Susceptibility and Resistance to Coronavirus Disease-19 (COVID-19): A Review

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ABSTRACT: It is believed that certain environmental factors modulate coronavirus disease-19 (COVID-19). This review outlines the role of environmental factors in COVID-19 infectivity, spread, and severity. Relevant articles were retrieved from Google Scholar, SpringerLink, and Scopus, then pooled and duplicates removed with EndNote software. Available information reveals that temperature, relative humidity (RH), sunlight, pollutants, and population density modulate COVID-19 infectivity and pathogenicity. COVID-19 spread is promoted by low temperature (< 25 °C) and RH (< 40%), whereas it is inhibited by high temperature (> 25 °C) and RH (> 40%). Sunlight exposure alters the virus's genetic material and boosts the host's immune function by raising serum vitamin D (25-hydroxyvitanim D), reducing the virus's viability and replication. Prolonged indoor stays with poor ventilation cause re-breathing of the air and increase carbon dioxide concentration, particularly in crowded rooms, predisposing to COVID-19. Pollutants, including particulate matter, nitrogen dioxide, ozone, and sulphur dioxide, may overexpress the virus's receptor called angiotensin-converting enzyme 2 (*ACE2*), thereby increasing the virus's infectivity. Pollutants may also induce inflammation of the respiratory tract, weakening the immune function and thereby increasing susceptibility to COVID-19. High population density increases body contact and thus susceptibility to the virus. To stem the incidence and mortality of COVID-19, the mentioned environmental factors must be kept at healthy levels.

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Coronaviruses were first identified in the 1960s among humans and some other animals (Alsahafi and Cheng, 2016). Of the coronaviruses, the subgroup that infects humans, called human coronaviruses (HCoVs), is highly susceptible to mutations, resulting in the group's high evolutionary potential (Alsmo and Alsmo, 2014). The recent outbreak of severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) brings to seven the number of HCoVs. The six previous strains are human coronavirus-229E (HCoV-229E), human coronavirus-NL63 (HCoV-NL63), human coronavirus-OC43 (HCoV-OC43), human coronavirus-HKU1 (HKU1), the Middle East respiratory coronavirus (MERS-CoV), and severe acute respiratory coronavirus (SARS-CoV) (American Lung Association, 2020). The 229E, NL63, OC43, and HKU1 strains cause mainly self-limiting upper airway infections, characterized by cough, fever, nasal discharges, and sore throats (Assiri *et al*., 2016). Human coronaviruses became a global burden after the outbreak of SARS-CoV in 2003 in Guangdong, China (Atkinson *et al*., 2009; Atkinson *et al*., 2018). SARS-CoV spread to 29 countries and infected over 8000 humans, with a mortality rate of about 10% (Atkinson *et al*., 2018; Backer, 2008). Human coronaviruses became more dreadful with the outbreak of MERS-CoV in Jordan in April 2012 (Bashir, 2020), and later in Saudi Arabia (Bergman, 2013), and much later in some regions of the world (Brown *et al*., 2003; Chan *et al*., 2020). Moreover, on

December 29, 2019, SARS-CoV-2 broke out in Wuhan, China (Chan *et al*., 2021) and infected over 200 countries (Chin *et al*., 2020). Shortly after, the world health organization (WHO) renamed the new virus as coronavirus disease-2019 (COVID-19) (Coccia, 2020) to reflect the year it emerged. SARS-CoV-2 is genetically similar to SARS-CoV and MER-CoV (Comunian, 2020). HCoVs' primary targets are epithelial cells in the lungs and gut, where they multiply and spread to other parts of the body (Alsahafi and Cheng, 2016; Condair Group, 2020). Human to human transmission can occur via several routes, including respiratory droplets, airborne particles, fomites, or fecal matter (Alsahafi and Cheng, 2016). HCoVs are highly infectious, among which SARS-CoV-2 is the most infectious (Conticini, 2020). COVID-19 could be symptomatic or asymptomatic and affected individuals may express mild or severe respiratory conditions (Corman *et al*., 2018). The mortality and socioeconomic burden of COVID-19 are enormous and increasingly threaten human wellbeing, necessitating a global search for a cure and preventive measures. According to some researchers, this can be leveraged on the differential susceptibility of people to the disease, which is suggestive of some environmental factors among other factors. Thus, sufficient knowledge of the relationship between environmental factors and the spread of the virus may provide a clue to curtailing the spread and mortality of the disease. This review outlined environmental factors that modulate the transmission and mortality of COVID-19 to give an insight into strategies that may reduce the spread and mortality of the disease.

MATERIALS AND METHODS

Searched Academic Repositories and Search Strategy: Relevant articles were retrieved from PubMed, SpringerLink, Google Scholar, and Scopus using search terms including coronavirus, COVID-19, HCoVs, SARS-CoV-2, and environmental influence on SARS-CoV-2. The academic repositories were searched separately, after which the articles were pooled and duplicates removed using EndNote software.

Inclusion and Exclusion Criteria: Articles that were written in English and focused on COVID-19 and its environmental aspects were included. Articles published prior to 2003 were excluded because little was known about the disease prior to that year. Studies with only abstracts available were also excluded. One hundred and twenty (120) articles were retrieved from the academic repositories searched and 92 were retained after removing duplicates. Eighty-three (83) of the 92 articles passed the eligibility criteria, with 79 of them meeting the study's objectives and so being chosen.

Environmental Susceptibility and Resistance to COVID-19: Available information shows that environmental factors such as temperature, humidity, sunlight, air pollutants, and population density can influence the spread and resistance to COVID-19. The links between these factors and COVID-19's spread and mortality are outlined below.

Temperature and Relative Humidity: When a human breathes, sneezes, talks, or coughs, some microscopic water droplets called aerosols are emitted, which contain mainly water and some solutes, such as salts, proteins, and other substances (Constantini, 2020). In healthy individuals, aerosols are harmless, but in individuals expressing respiratory tract infections, they may contain harmful and infectious microbes such as bacteria and viruses (Constantini, 2020). The transmissibility of the microbes in aerosols is influenced by certain factors, among which humidity is an important one. Relative humidity (RH) > 40 and < 70% limits the survival of viruses and bacteria (Dominici, 2020). Above 40% RH, an aerosol retains its moisture content and becomes heavier and less able to remain airborne (European Center for Disease Prevention and Control, 2020). Furthermore, the dissolved salts in an aerosol can create a toxic environment for any suspended microbe, decreasing its transmissibility and health risk (European Center for Disease Prevention and Control, 2020). Thus, RH above 40% reduces airborne infection by reducing the air's microbial loads and inhibiting their infectivity (European Center for Disease Prevention and Control, 2020). However, below 40% RH, an aerosol rapidly loses its moisture content through evaporation, which makes it smaller and remain airborne longer (European Center for Disease Prevention and Control, 2020). Loss of water also changes an aerosol's structure to a crystallized solid state, preserving the viruses and bacteria in it and boosting their viability and infectivity (European Center for Disease Prevention and Control, 2020). A study conducted on COVID-19 in Sydney, Australia discovered that a 1% decrease in RH increased COVID-19 incidence by 6% (Filippidou and Koukouliata, 2011). Similarly, in a study that monitored the occurrence of COVID- 19 in 30 Chinese provinces (average RH = 17.93% to 86.20%), a 1% increase in average RH decreased daily incidence between 11 and 22% (Frieman and Baric, 2008).

The ambient temperature is also important in the spread and survival of microbes in aerosols. Low temperatures similar to those obtained during winter

cause vasoconstriction of the respiratory tract mucosa, reducing immune cell recruitment against invading microbes (Frontera *et al*., 2020). On the other hand, high temperatures, like those obtained during the summer, break down the lipid layers of viruses and inactivate them (Gorbalenya *et al*., 2020; Grant *et al*., 2020). In a study that monitored the effects of temperature on COVID-19 in 166 countries (average temperature = -5.28 to 34.30 °C), every 1 °C rise in temperature caused a 3.08% and 0.51% decrease in daily incidence and mortality, respectively (Gruber-Bzura, 2018). Similarly, in a study that monitored the spread of COVID-19 in 11 notable cities in China (average temperature = -1.0 to 14.9 ˚C), the reproduction rate of the virus decreased as the temperature increased (Guo *et al*., 2020). In addition, heating at 56 ˚C for 30 minutes and 60 ˚C for 60 minutes in an experiment reduced the population of SARS-CoV-2, while total inactivation was achieved at 92 ˚C for 15 minutes (Hamidi *et al*., 2020). In another study, researchers found that SARS-CoV-2 was highly stable at 4 ˚C but was deactivated in 5 minutes at 70 ˚C (Hedberg *et al*., 1989). Generally, the higher the viral load, the higher the temperature that will be required to inactivate SARS-CoV-2 and vice versa (Hamidi *et al*., 2020).

Furthermore, temperature and RH can synergistically affect the transmission of COVID-19 and could be partly responsible for the worldwide varied susceptibility to the disease. The combined effects of low temperature and RH as found in temperate climates such as East Asia, Europe, and North America (RH < 50%; temperature < 25 ˚C) could explain the disease's relatively high infectivity in the regions (Hijawi *et al*., 2013). Conversely, the high temperature and RH of the tropical climates, including Southeast Asia, Africa, and South America (RH > 50%; temperature > 25 ˚C), could be the cause of the relatively low infectivity of the disease in the regions (Hijawi *et al*., 2013). This means that, regardless of the season or climate, air-conditioned environments with typical temperatures of 22–25 ˚C and RH of 40–50% will promote the viability and transmission of COVID-19 (Gorbalenya *et al*., 2020). This could partly be responsible for the transmission of the disease in the summer when there is an increased use of air-conditioners due to hot weather. In a fully air-conditioned eatery in China, of the 91 people who entered the restaurant on a particular day, 10 became infected, in which airflow from air conditioners was blamed for the transmission (Hobday *et al*., 1997).

Sunlight Exposure: The combination of sunlight exposure and ventilation was the main antimicrobial strategy in use before the discovery of antibiotics (Hobday and Cason, 2009). Sunlight exposure boosts resistance to infection by decontaminating surfaces (Hobday and Dancer, 2013) and synchronizing the body's biological rhythms (Ilie *et al*., 2020). During the 1918 influenza outbreak, infected people who were treated outdoors responded better and recorded less mortality than those treated indoors (Jiang *et al*., 2016). Moreover, the synergistic effects of sunlight exposure and ventilation prevented exposure of medical staff during the influenza pandemic (Jiang *et al*., 2016). In a hospital in Boston, Massachusetts, outdoor treatment reduced mortality among influenza patients from 40% to about 13% (Kan *et al*., 2010). The application of sunlight exposure as an antimicrobial strategy was so famous then that military doctors used it to treat infected wounds during the First World War (Kesic *et al*., 2012). Indoor stays in poorly ventilated rooms with many occupants will cause re-breathing of the air in the room and raise the concentration of carbon dioxide (CO_2), increasing the vulnerability of the occupants (Loon and Lun, 2013). In a study that looked at ethnic and racial differences in susceptibility to COVID-19 in Georgia, Latino/Hispanics had lower COVID-19 morbidity than other ethnics or races, with the exception of African Americans (Lu *et al*., 2020). This is because Latinos spend more time outdoors, which again highlights the importance of sunlight exposure in the risk reduction and treatment of COVID-19 (Lu *et al*., 2020).

Sunlight stimulates antimicrobial activity by emitting ultraviolet radiation (UVR) and increasing plasma concentrations of 25-hydroxyvitamin D (25(OH) D), the serum active form of vitamin D. Vitamin D deficiency (25(OH)D < 30 nmol/L) raises the risk of respiratory infections by compromising the immune system (Lytle and Sagripanti, 2005; Ma *et al*., 2020), whereas adequate vitamin D levels improve immune function and reduce infections (Malta *et al*., 2020). A healthy vitamin D level can stimulate antimicrobial peptides that reduce viral replication and pro-inflammatory cytokines in the lungs (Martineau *et al*., 2017). This suggests that normal vitamin D levels or vitamin D supplementation may ameliorate diseases associated with an excess of pro-inflammatory cytokines such as COVID-19. In a study that investigated the importance of vitamin D in COVID-19's spread and fatalities among 20 European nations, the most vulnerable individuals, mainly the elderly, had the lowest vitamin D levels (Ma *et al*., 2020). While the mean vitamin D level of the participants was 56 mmol/L, the vulnerable group was between 26 and 45 nmol/L (Ma *et al*., 2020). In a systematic review involving 11321 participants (aged zero to 95), vitamin D administration decreased the risk of acute

respiratory tract infection among the participants (Mourtzoukou and Falagas, 2007). In the mentioned study, protective effects were noticed in a subgroup that took daily or weekly vitamin D without additional bolus doses, but not in those that took one or more bolus doses (Mourtzoukou and Falagas, 2007). The protective effects in the aforementioned subgroup were stronger in those with baseline 25(OH)D < 25 nmol/L than in those with baseline 25(OH)D ≥ 25 nmol/L (Mourtzoukou and Falagas, 2007). In a related study involving 5660 patients, similar results were reported in which dosing with vitamin D once a day seemed most effective (Neiderud, 2015). For COVID-19 specifically, individuals at risk might benefit from taking 10,000 IU/d of vitamin D for a few weeks, followed by 5000 IU/d (Martineau *et al*., 2017). The dosages mentioned are intended to raise plasma 25(OH)D levels to a healthy level (> 100 nmol/L) (Martineau *et al*., 2017). For the treatment of COVID-19 patients, higher vitamin D doses might be useful (Martineau *et al*., 2017).

Regarding UVR, it kills viruses by altering genetic materials or generating free radicals (Nightingale, 1863). Based on the wavelength, the UVR is classified into three components, which are UVC (260 nm), UVB (290 to 320 nm) and UVA (320 to 380 nm) (Ogen, 2020). The most effective wavelength for inactivating viruses is the UVC, but is not present in the sunlight that reaches the earth's surface (Ogen, 2020). Both UVB and UVA reach the earth's surface and have been shown to damage the nucleic acids of the viruses, though not as efficiently as UVC (Ogen, 2020). In a study, sunlight simulation similar to the summer solstice at 40 °N latitude at sea level on a clear day inactivated 90% of infectious SARS-CoV-2 every 6.8 minutes in simulated saliva and every 14.3 minutes in culture media (Pastorino *et al*., 2020). Significant inactivation also occurred at lower simulated sunlight levels, but at a slower rate (Pastorino *et al*., 2020). In simulated sunlight similar to that of the winter solstice at 40 °N latitude, 90% of infectious SARS-CoV-2 was inactivated every 14.3 minutes in simulated saliva dried on a surface (Pastorino *et al*., 2020). However, when SARS-CoV-2 in simulated saliva was exposed to darkness, no significant decay was observed in 60 minutes (Pastorino *et al*., 2020). These findings suggest that SARS-CoV-2 infectivity may be greatly decreased in outdoor environments exposed to direct sunlight compared to indoor environments (Pastorino *et al*., 2020). The findings further revealed that the sunlight inactivation rate of SARS-CoV-2 depends on both the intensity of sunlight and the surface on which the virus is suspended (Pastorino *et al*., 2020). In another study that estimated solar inactivation of SARS-CoV-2, the results indicate that SARS- CoV-2 aerosolized by infected individuals and deposited on surfaces could remain infectious outdoors for a long time during the winter in many temperate climates, with continued risk for reaerosolization and human infection (Peiris *et al*., 2004). In contrast, SARS-CoV- 2 could be inactivated quickly in the summer, which suggests that sunlight influences the occurrence, spread rate, and duration of coronavirus pandemics (Peiris *et al*., 2004).

Pollutant Exposure: Air pollution is the most important environmental cause of disease, with seven million deaths attributed to the combined effects of indoor and outdoor air pollution (Qi *et al*., 2020). According to Ratnesar *et al*. (2020), air pollution accounts for 17% of all deaths and diseases caused by acute lower respiratory infections. Pollutants with the greatest public health concern include particulate matter (PM), ground-level ozone (O_3), nitrogen dioxide (NO_2), and sulphur dioxide (SO_2) (Ratnesar *et al*., 2020). In a study that investigated the link between environmental pollutants and COVID-19 incidence in California, USA, among other pollutants, PM10, PM2.5, SO_2, NO_2, and CO showed a strong association (Rocklov and Sjodin, 2020). Prolonged exposure to PM can influence a pathogen's infectivity by weakening the immune system, especially in the elderly (Rondanelli *et al*., 2018). Microorganisms, including SARS-CoV-2, can cling to and be transported by PM, so detection of SARS-CoV-2 RNA or any other infectious microbe on PM from outdoor air samples in any city could predict disease onset (Rose *et al*., 1989a). Particulate matter can also induce inflammation in the lung cells, thereby increasing the susceptibility and severity of diseases associated with inflammation, such as COVID-19 (Rose *et al*., 1989b). Moreover, PM can penetrate deep into lung passageways and enter the bloodstream, causing cardiovascular, cerebrovascular, and respiratory impacts (Ratnesar *et al*., 2020), which are some of the pathologies of COVID-19. These suggest that PM exposure may predispose to COVID-19 or worsen its outcome. In a study that examined the link between PM2.5 exposure and COVID-19 across 3,080 counties in the USA, a 1 μ g/m^3 increase in long-term exposure to PM2.5 increased the risk of COVID-19 mortality by 8% (Sagripanti and Lytle, 2020). In a related study, the fast and widespread spread of COVID-19 in Northern Italy was linked with prolonged exposure to non-permissible levels of PM10 (Saygin *et al*., 2017). In another study carried out in Bergamo, Italy, SARS-CoV-2 RNA was detected in particulate matter, which again proved air pollutants can transmit microbes (Rose *et al*., 1989a). Prolonged exposure to PM2.5 overexpresses SARS-CoV-2's receptor called angiotensin-converting enzyme 2 (*ACE2*), increasing

the viral load and impairing host immune function (Schoeman and Fielding, 2019).

Nitrogen dioxide exposure is also associated with some pathologies of COVID-19, such as respiratory and cardiovascular diseases (Seah and Agarwal, 2020). Nitrogen dioxide is a free radical and so can induce an inflammatory response and cellular damage (Schoeman and Fielding, 2019). In February 1987, NO_2 diffusion caused breathing problems among players and spectators of indoor hockey games in Minnesota, USA (Setti *et al*., 2020). The affected experienced cough, hemoptysis, and/or dyspnea shortly after attending a hockey game (Setti *et al*., 2020). This incident showed that NO_2 may directly cause respiratory illness or worsen it. In particular, some studies have shown that NO_2 exposure may aggravate some symptoms and raise the fatality of COVID-19. A study conducted in Europe showed that 78% of deaths happened in five regions in Italy and Spain that are seriously polluted with NO_2 (Su *et al*., 2019). The researchers reasoned that chronic exposure to NO_2 could be responsible for the high COVID-19 mortality in the mentioned regions and possibly elsewhere (Su *et al*., 2019). In an experiment, mice exposed to NO_2 before cytomegalovirus infection required a 100-fold lower viral load and showed a higher re-infection rate than control mice (Sunyer *et al*., 2003). In a related experiment, mice exposed to 5 ppm of NO_2 before a viral infection rapidly developed lower respiratory tract injury (Szeto *et al*., 2020). Exposure to the same concentration of NO_2 without viral exposure did not cause tissue damage (Szeto *et al*., 2020). Re-inoculation of the virus after 30 days caused re-infection only in mice previously exposed to NO_2 (Szeto *et al*., 2020).

Sulphur dioxide is a gas produced by fuel combustion, and its major source in most cities is traffic (The Havard Gazette, 2020). Hence, SO_2 might be a surrogate for the traffic pollution mixture (The Havard Gazette, 2020). Short-term exposure to SO_2 may cause bronchoconstriction in normal and asthmatic subjects (The Havard Gazette, 2020). Continued exposure to SO_2 at high levels increases respiratory symptoms and reduces lung function (Travaglio, 2021). In 1998, SO_2 pollution occurred in Nottingham and Leicestershire, UK, which increased hospitalization for respiratory and cardiovascular diseases (Vijgen *et al*., 2005). This showed that SO_2 may directly cause respiratory illness or worsen it. There is a dearth of studies on the association between SO_2 exposure and COVID-19. However, findings on previous viral outbreaks suggest an association might exist. In a study that monitored the effects of air pollutants on the spread and severity of influenza-like illnesses in China, elevated concentrations of SO_2 were strongly linked (Ward *et al*., 2020). In another study that probed the link between outdoor air pollution and daily deaths, also in China, SO_2 and NO_2 were significantly associated with daily mortality from all causes, including cardiopulmonary diseases (Weapon Against Influenza, 1918). This association appeared to be more pronounced in the cool season than in the warm (Weapon Against Influenza, 1918), which are some of the factors that determine the spread and pathogenicity of COVID-19. Short-term exposures to ozone irritate the respiratory system and may damage lung tissues, reducing lung function and increasing airway inflammation, as well as making the lungs more sensitive to other irritants (World Economic Forum, 2020). Chronic exposure to ozone may cause lung cancer and chronic respiratory diseases (World Economic Forum, 2020). Ozone exposure affects both healthy individuals and individuals with existing breathing problems (World Economic Forum, 2020). The most sensitive people to ozone exposure are the elderly and children, as well as people with existing diseases such as respiratory diseases, diabetes mellitus, asthma, lung cancer, cardiovascular disease, and immune system deficiency (World Economic Forum, 2020). Some of these effects are related to some pathologies of COVID-19, suggesting that the pollutant may worsen COVID-19. In a study that investigated the correlation between air pollutants and SARS-CoV-2 in England, elevated levels of NO_2 and ozone were associated with COVID-19 lethality (WHO, 2020a). In a related study carried out in Milan, Italy, ozone was again positively correlated with COVID-19 (WHO, 2020b). Ozone was found to be positively correlated with high air temperature, low RH, and precipitation in the same study, implying that ozone may act as a COVID-19 virus incubator, enhancing virus transmission in hot weather (WHO, 2020b). In an in vitro study that used human nasal epithelial cells, pre-exposure to ozone was demonstrated to disrupt the protease/antiprotease balance, increasing influenza susceptibility (WHO, 2005). Generally, pollutant levels and types in the air vary across countries, seasons, and times (Qi *et al*., 2020), which could explain differences in COVID-19 susceptibility around the world. The observed high transmission and vulnerability to COVID-19 in polluted environments underscore the importance of pollution control in maintaining optimum health (Su *et al*., 2019). To minimize the adverse effects of air pollution, particularly on the transmission of microbial infections such as COVID-19, governments at all levels should come up with effective pollution reduction policies. Individuals can also limit their outdoor exposure by wearing masks when necessary (Qi *et al*., 2020). Exposure to indoor air pollutants can

be reduced by limiting the usage of solid and liquid fuels and, when necessary, clean fuels and stoves that burn fuel efficiently should be used (Qi *et al*., 2020; WHO, 2020c). Indoor air cleaners can also help to improve the quality of indoor air (Qi *et al*., 2020).

Population Density: The spread of COVID-19 can be up-regulated by high population density and mobility. Wuhan, the city in which the virus originated, is a notable transport center in China with over 11 million inhabitants (Wu *et al*., 2020a). The high population and mobility could be responsible for the explosion of the disease in the city. Population density is a factor in the COVID-19 pandemic, as it was in previous disease outbreaks (Wu *et al*., 2020b). The close contact of people in cities is an important factor in the transmission of infectious diseases such as SARS and the avian flu (Zaki *et al*., 2012; Zhu *et al*., 2020). A major strategy for reducing the transmission of COVID-19 is the avoidance of body contact (Zoran *et al*., 2020), particularly from someone sneezing and coughing. However, staying at least one meter away from people coughing and sneezing, as recommended by the WHO, becomes increasingly difficult with higher population densities (Zoran *et al*., 2020). High population density is also synonymous with increased anthropogenic activities accompanied by high environmental pollution, which is a modifier of COVID-19 spread. Though there is a dearth of studies on the association between population density and COVID-19, the high incidence of the disease in some populated cities suggests an association. For instance, based on the data from the Italian Civil Protection on March 21st, 2020, the densely populated cities of Lombardy and Emilia Romagna recorded 12% mortality compared to the Italian national average of 4.5% (Wu *et al*., 2020b). In Nigeria, the three most populated cities, Lagos, Kano, and Abuja, recorded the highest cases and mortality of COVID-19. Lagos, in particular, has a small landmass and accounted for most of the cases, possibly due to overcrowding and pollution from industries, homes, and vehicles. The city is a major economic hub in Africa with high mobility of people. Put together, avoiding overcrowding or close contact will be necessary to limit the spread of COVID-19.

Conclusion: Environmental factors, such as temperature, humidity, sunlight, population density, and pollutant exposure, influence the viability, replication, and cell-binding ability of SARS-CoV-2, the causative agent of COVID-19. Generally, high temperature, humidity, and sunlight exposure reduce the virus's infectivity and pathogenicity, while high pollutant exposure and population density promote it. To stem the spread and fatalities of COVID-19, environmental factors must be kept within healthy limits.

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