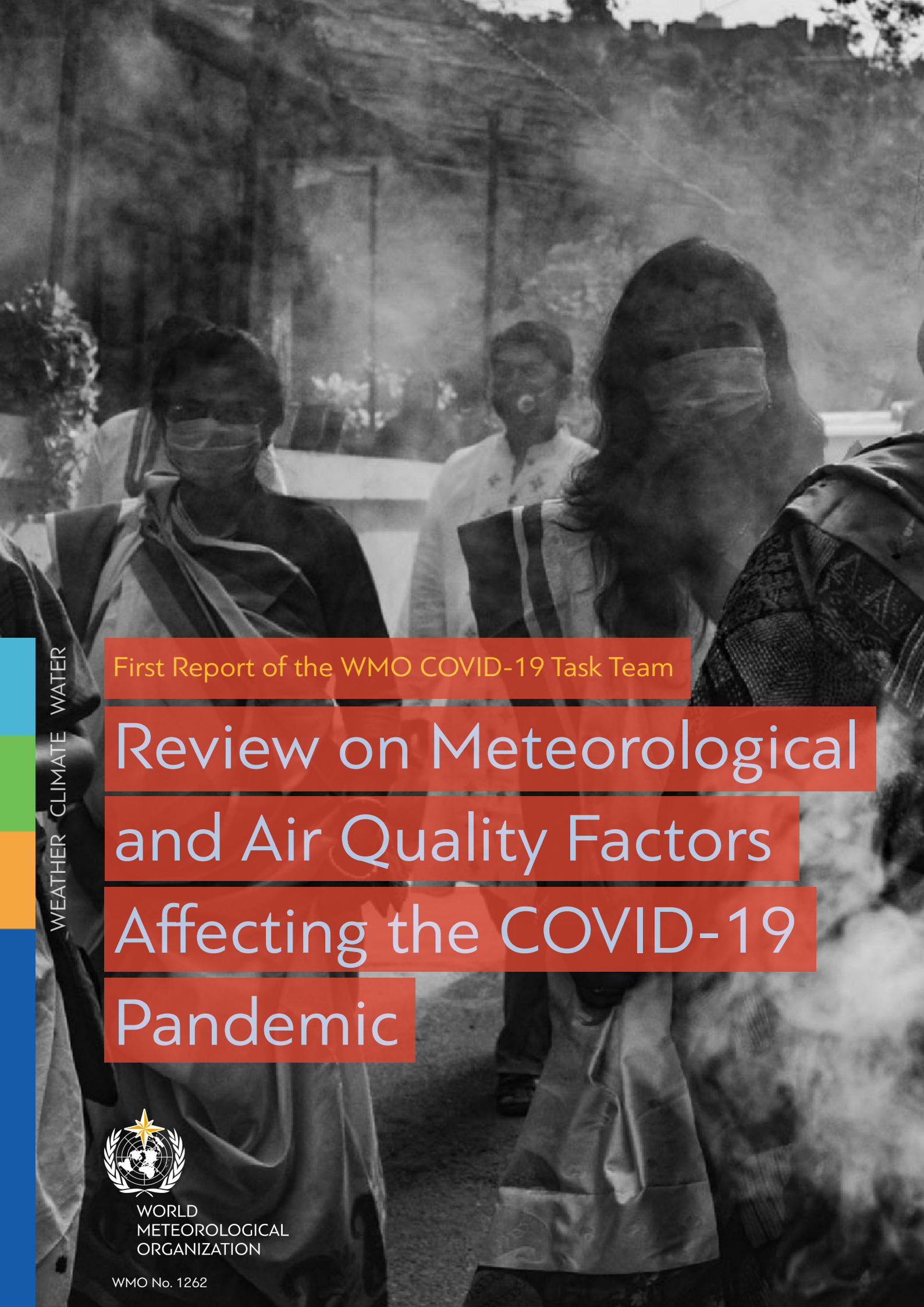

WEATHER CLIMATE WATER

First Report of the WMO COVID-19 Task Team

Review on Meteorological and Air Quality Factors Affecting the COVID-19 Pandemic

WORLD METEOROLOGICAL ORGANIZATION

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EXECUTIVE SUMMARY

In September 2020, the World Meteorological Organization (WMO) Research Board set up an interdisciplinary and international Task Team to respond to the challenge of providing timely decision support and relevant knowledge on meteorological and air quality (MAQ) factors affecting the sever acute respiratory syndrome coronavirus 2 (SARS-CoV-2) (the virus that causes COVID-19) pandemic. The aims of the Task Team are: to provide decision makers and the public with a rapid summary of the state of knowledge regarding potential MAQ influences on SARS-CoV-2/COVID-19; to offer general technical guidance for researchers and service providers who wish to consider MAQ data in their analyses, estimates, predictions and projections of COVID-19 risks.

This report presents a summary of key findings to date, as informed by peer-reviewed literature. It is motivated both by the global relevance of the subject and by the staggering number of papers and preprints currently available, which emphasizes the need for careful review and communication of the state of the science.

We note that the report adopts a number of constraints. First, it considers only peer-reviewed papers (not preprints) that were published by the first week of January 2021. Second, the report focuses on outdoor MAQ conditions. Links between outdoor and indoor environments are considered, but the report does not address details of indoor air circulation or climate control. Third, this report was written just as the existence of new strains of SARS-CoV-2 were recognized. As such, there was no peer-reviewed literature to consider regarding the influence of MAQ on transmission of the new strains, or on severity of infections caused by these new strains. We also emphasize that the report is not a formal systematic review; it is a summary of the overall state of the literature.

Main findings:

- **Epidemiological studies of COVID-19 have, to date, offered mixed results regarding the meteorological sensitivity of the virus and the disease.**
- **COVID-19 transmission dynamics in 2020 appear to have been controlled primarily by government interventions rather than meteorological factors.** Other relevant drivers include changes in human behaviour and demographics of affected populations, and more recently, virus mutations.
- **Respiratory viral infections frequently exhibit some form of seasonality,** particularly in temperate climates. The seasonality in respiratory viral diseases – in particular the autumn–winter peak for influenza and cold-causing coronaviruses in temperate climates – has fuelled expectations that COVID-19 will prove to be a strongly seasonal disease should it persist for multiple years.
- The underlying mechanisms that drive seasonality of respiratory viral infections are not yet well understood. A combination of direct impacts on virus survival, impacts on human resistance to infection, and indirect influence of weather and season via changes in human behaviour may be at work (Figure 1).
- Laboratory studies of SARS-CoV-2, the virus that causes COVID-19, have yielded **some evidence that the virus survives longer under cold, dry and low ultraviolet radiation conditions.** However, these studies have not yet indicated if direct meteorological influences on the virus have a meaningful influence on transmission rates under real world conditions.
- There is **evidence that chronic and short-term exposure to air pollution exacerbates symptoms and increases mortality rates for some respiratory diseases.** This is consistent with early studies of COVID-19 mortality rates, but these results need to be confirmed and consolidated by controlling for individual-level risk factors. There is no direct, peer-reviewed evidence of pollution impacts on the airborne viability of SARS-CoV-2 at this time.

- Process-based modelling studies anticipate that **COVID-19 transmission may become seasonal over time, suggesting MAQ factors may support monitoring and forecasting of COVID-19 in the coming months and years.**
- At this stage, however, **evidence does not support the use of MAQ factors as a basis for governments to relax their interventions aimed at reducing transmission.**
- **Research quantifying links between MAQ factors and COVID-19 is needed.** It is critical that modelling studies properly account for confounding factors, consider both direct and indirect MAQ effects, address limitations in the COVID-19 data record, report uncertainty ranges, evaluate predictive skill, and apply appropriate statistical or process-based modelling techniques.
- The availability of open, timely and quality-controlled data on COVID-19 and associated risk factors is critical for studies of MAQ influence and for many other studies of COVID-19 risk. Efforts to provide these data have been disjointed, and point to **the need for a reporting infrastructure that supports data management and dissemination for analysis of epidemic diseases.**
- Peer-reviewed studies have the potential to influence public health decisions and public perceptions of disease risk. For this reason, it is critical that researchers, publishers and information providers **maintain high standards for analysis and evaluation of emerging studies.**
- Just as importantly, **clear and active communication between researchers, the media, and decision makers is required** to ensure that scientific findings are applied to policy in an appropriate, objective, transparent and responsible manner.

These conclusions are informed by the Task Team's evaluation of studies that had cleared peer review at the time this report was written. They will be updated as appropriate in subsequent Task Team reports and communications.

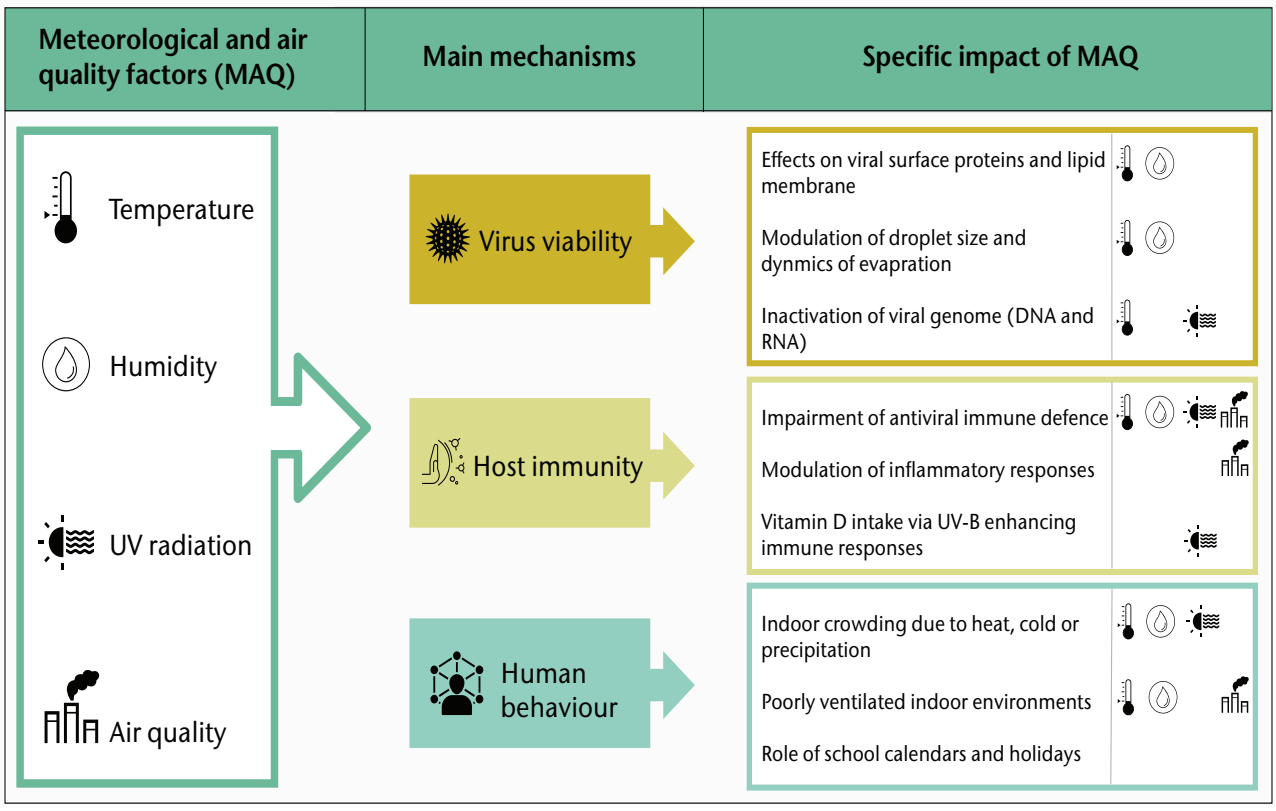

Figure 1. Examples of proposed mechanisms through which MAQ factors influence respiratory viral infections

BACKGROUND

After the first full year of the COVID-19 pandemic, the influence of MAQ conditions on SARS-CoV-2 transmission and severity of COVID-19 continues to be an area of active research. From the beginning of the pandemic there was scientific and popular speculation that COVID-19, like other respiratory viral infections, would exhibit some form of seasonality and vary according to weather conditions and air quality. In particular, previous experience with influenza and other seasonal coronaviruses pointed to the potential for a wintertime peak in temperate climate zones. Early efforts to quantify these effects, however, yielded uncertain and contradictory results (Briz-Redón and Serrano-Aroca, 2020; Mecenas et al., 2020; Shakil et al., 2020; Smit et al., 2020). The contradictions in these results, combined with methodological and data-related limitations in some rapidly released studies, have led to some confusion regarding the importance of MAQ factors in determining COVID-19 risk (Zaitchik et al., 2020). The large proportion of immunologically naïve individuals across the globe is expected to outweigh environmental effects during the pandemic phase of the infection (Baker et al., 2020). As COVID-19 becomes a more endemic disease, seasonality may become more evident (Kissler et al., 2020), although the extent to which seasonality may be driven by MAQ conditions or other factors, such as simple annual waning of immunity in the human population (Callow et al., 1990) is yet to be understood.

The purpose of this report, elaborated since September 2020 by the Task Team, is twofold: first, to offer a summary of the evidence and current understanding of the potential links or connections between MAQ factors and COVID-19; second, to offer general technical guidance for researchers and service providers who wish to incorporate MAQ data in their estimates, predictions and projections of COVID-19 risk. The report is motivated both by the global relevance of the subject and by the fact that the flood of preprints of varying quality have highlighted the need for careful review and communication of the state of the science. The WMO Task Team will update and expand on this report regularly over the coming year, considering recent findings and comments from different scientific communities, the World Health Organization (WHO) and collaborating centres, climate service providers, governments and stakeholders.

THE STATE OF UNDERSTANDING

Proposed mechanisms

There are a number of mechanisms through which meteorological conditions can influence the transmission and severity of respiratory viral infections (Figure 1). While several of these mechanisms are plausible for SARS-CoV-2 transmission, and some have been demonstrated under laboratory conditions, the relative importance of the proposed mechanisms in real world conditions is a matter of continued investigation (Moriyama et al., 2020). We consider here the three variables – ultraviolet (UV) radiation, air temperature and humidity (either separately or combined) – for which mechanisms have been more extensively investigated for respiratory viral infections other than COVID-19, since mechanistic understanding of SARS-CoV-2 and COVID-19 is not yet mature (Audi et al., 2020; Moriyama et al., 2020).

Increasing doses of UV radiation of all wavelengths, and high-energy UV-C radiation in particular, have been demonstrated to reduce the survival of DNA and RNA viruses (Darnell et al., 2004; Lytle and Sagripanti, 2005; Bedell et al., 2016; Buonanno et al., 2020; Schuit et al., 2020a), for example, in darkness, the half-life of influenza was 31.6 minutes compared to a half-life of approximately 2.4 minutes when exposed to full-intensity simulated sunlight (Schuit et al., 2020a). While the impact of UV radiation on virus survival is clear in laboratory settings, its importance for SARS-CoV-2 transmission has not yet been proven (European Centre for Disease Prevention and Control (ECDC), 2020; Ratnesar-Shumate et al., 2020; Schuit et al., 2020b). Some studies have identified negative correlations or non-linear associations between UV radiation and indicators of SARS-CoV-2 transmission (Runkle et al., 2020), including the COVID-19 case growth rate (Merow and Urban, 2020). However, the size of effect is small, the statistical form of the relationship is unclear, and not all studies have confirmed statistical associations (Pequeno et al., 2020; Yao et al., 2020a). Additionally, epidemiological studies of UV radiation influence have not distinguished between direct impacts on the virus and indirect impacts on disease spread, perhaps mediated by UV-B influence on levels of vitamin D (Cannell et al., 2006; Shaman et al., 2011; Tamerius et al., 2011; Grant et al., 2020). A key question is whether the confirmed direct mechanism through which UV radiation can reduce SARS-CoV-2 survival in aerosols and on surfaces is a meaningful modifier of SARS-CoV-2 transmission risk. Such a UV radiation influence could have implications for how risk varies with season, latitude, and likely altitude, but it has yet to be established with certainty and robustness.

For temperature and humidity, a broader suite of mechanisms has been proposed. The first relates directly to virus stability and transmission. Laboratory studies of several enveloped viruses such as influenza and other human coronaviruses have indicated a sensitivity to relative or absolute humidity and temperature (Harper, 1961; Schaffer et al., 1976; Ijaz et al., 1985; Chan et al., 2011), which is assumed to be due to environmental effects on viral surface proteins and lipid membrane (Tang, 2009). Lower temperature was found to enhance the structural order of lipids of the influenza virus envelope and improve stability (Polozov et al., 2008). Laboratory results of environmental sensitivities vary by virus, but point to the fact that low-humidity environments favour the rapid evaporation of water from exhaled aerosols, forming droplet nuclei (<5 μm in diameter) that might allow for prolonged viability and transmissibility of influenza virus (Weinstein et al., 2003; Tellier, 2006; Lowen et al., 2007). This raises the possibility that the seasonality of these viruses may be related to higher survival rates in cooler and drier environments. Laboratory studies of viral transmission in animals, which have focused on influenza, also show that low relative humidity (20%–35%) and cool temperatures (5 °C) favour enhanced transmission in experiments that span a wide range of humidity and temperature conditions (Lowen et al., 2007, 2008). There is evidence that SARS-CoV-2 can be transmitted by aerosols (droplets ≤5 μm) (Lednicky et al., 2020; Liu Y. et al., 2020;

Tang et al., 2020). However, the relative importance of airborne transmission (infection through virus-containing respiratory droplets ≤5 μm that remain suspended in the air over longer distances) in relation to contact transmission (infection through direct contact with an infectious person or contaminated surface) or droplet transmission (infection through virus-containing respiratory droplets within shorter distances) remains to be further investigated (Klompas et al., 2020). It has been hypothesized that cool and dry conditions are particularly suitable for aerosol transmission of viruses in mid-latitude winter settings, attributing higher importance to aerosol transmission in temperate climates (Lowen et al., 2008; Tamerius et al., 2011; Huang Z. et al., 2020). Other sensitivities might be observed in warm and humid climates where small respiratory droplets absorb water, increase in size and then settle more readily on surfaces, placing a greater focus on contact transmission over airborne transmission in tropical climates (Lowen et al., 2008; Tamerius et al., 2011).

A second, complementary set of mechanisms proposed for temperature and humidity influence on respiratory viruses are associated with the human antiviral defence. Studies have demonstrated that nasal and bronchial mucociliary clearance (MCC) can be impaired under cold and dry conditions (Soane et al., 2001; Eccles, 2002), reducing the body's ability to clear virus-containing particles once they are inhaled. Dry air can also inhibit epithelial cell repair in the trachea and lungs, as shown in animal models (Kudo et al., 2019), further reducing the body's ability to fend off infection. The immune system itself may also be sensitive to humidity and temperature. This applies to the innate immune system, as the expression of interferon-stimulated genes critical to induce an antiviral state (Ivashkiv and Donlin, 2014) may be reduced under low-humidity conditions such as those found indoors when outdoor temperatures are low (Foxman et al., 2015, 2016). The adaptive immune system may be compromised as high temperature was found to hinder virus-specific CD8+ T cell responses and antibody production after influenza infection (Moriyama and Ichinohe, 2019). The lethality of influenza in mice was found to increase in low-humidity conditions, a pattern that has been attributed to impaired immune system responses, including impaired MCC, innate antiviral defence and tissue repair function (Kudo et al., 2019).

While most studies have identified immune system sensitivities to low-temperature and low-humidity conditions, there is also evidence that high ambient temperatures can impair antibody production after influenza infection in mice (Moriyama and Ichinohe, 2019), pointing to the potential for multiple meteorological sensitivities. It has been proposed that sudden large changes in temperature can impair the human immune system and trigger immune evasion (Togias et al., 1985; Group, 1997; Graudenz et al., 2006; Guo et al., 2011, 2016; Loh et al., 2013), suggesting that the human thermoregulation of immune defence is potentially less adjustable to sudden large changes in temperature (Guo et al., 2011, 2016) and less resistant to various diseases (Group, 1997; Graudenz et al., 2006). Studies have demonstrated that the sudden large change in temperature tends to cause high respiratory mortality (Group, 1997; Graudenz et al., 2006; Guo et al., 2011, 2016; Loh et al., 2013; Liu Q. et al., 2020) and increased influenza risk (Liu Q. et al., 2020). In addition, studies have shown that exposure to high ambient temperature (for example, a heatwave) may increase the risk of respiratory and cardiovascular disease, in particular for vulnerable population groups such as the elderly, children or individuals with pre-existing conditions (Åström et al., 2011; Witt et al., 2015; Bunker et al., 2016; Xu et al., 2016; Song et al., 2017). Heat-vulnerable population groups might experience a higher risk of respiratory virus infection relative to the general population.

A third, indirect but potentially highly relevant mechanism through which temperature might influence transmission risk is via its influence on human behaviour. It is now well-established that COVID-19 transmission commonly occurs in closed spaces (ECDC, 2020). As is often noted, people tend to spend more time congregating in poorly ventilated environments when it is cold, hot and/or precipitating outside, and this behavioural change might partly explain seasonal patterns in the risk of respiratory viral infections like those producing COVID-19 (Audi et al., 2020). In hot climates,

people tend to escape the heat, spending increased time in air-conditioned indoor spaces. The importance of these behavioural factors relative to direct environmental influence on virus survival or host response is an active area of research, and no firm conclusions can be drawn for COVID-19 at this time. However, as evidence builds for the importance of aerosolized transmission of COVID-19 (Doremalen et al., 2020; Lednicky et al., 2020; Liu Y. et al., 2020), the role of weather in pushing people towards closed indoor environments requires attention. Extremely warm weather might also have a negative impact on efforts to reduce transmission rates. It has been shown that mask wearing increases facial temperature and discomfort (Scarano et al., 2020), which could reduce the appropriate use of masks in hot or humid conditions. Warm conditions also lead to increased use of air conditioning, which can change the direction of airflow and has been hypothesized to increase risk of indoor transmission (ECDC, 2020; Lu et al., 2020).

The hypothesis that ventilation and airflow might influence SARS-CoV-2 transmission in indoor environments builds on studies that have shown ventilation to be a predictor of transmission rates for other viruses, including influenza and SARS (Li et al., 2007; Knibbs et al., 2011). For SARS-CoV-2, quantitative empirical evidence of these relationships is beginning to emerge, though the relationships are complex and beyond the scope of this report. The expectation that airflow and ventilation might influence SARS-CoV-2 transmission, combined with this emerging evidence, is strong enough for health authorities to have issued recommendations. For example, ECDC recommends that direct airflow should be diverted away from individuals, and that the use of indoor air recirculation should be avoided to reduce the dispersion of SARS-CoV-2. Also, the minimum number of indoor–outdoor air exchanges per hour, depending on the applicable building regulations, should be ensured given that an increase in the number of air exchanges per hour can reduce the likelihood of indoor transmission (ECDC, 2020). From a meteorological perspective, it is known that outdoor meteorological conditions have a significant influence on indoor air-exchange rates, and that outdoor temperature is a particularly strong predictor (Wallace et al., 2002). Given this association and the general understanding that air exchange is a factor in indoor transmission, the potential for meteorology to influence indoor SARS-CoV-2 transmission via its impact on air-exchange rates warrants further investigation.

In the case of air quality, research on COVID-19 and other respiratory illnesses can be divided into: studies that are concerned with the transmission of the pathogen, primarily related to aerosol dynamics; studies related to the effects of certain pollutants on the immune response function, influencing the severity of disease symptoms and probability of death. There is substantial evidence for the second case, drawn from a number of respiratory diseases. In addition, concerns exist that long-term conditions resulting from COVID-19 infections can be worsened by chronic air pollution exposure, increasing hospital admissions for respiratory and cardiovascular conditions (Brunekreef et al., 2021). It is well known that chronic exposure to fine particulate matter (particles with a diameter of ≤2.5 μm (PM2.5)) (Kloog et al., 2012; Hassan et al., 2019), ozone (O_3) (Purvis et al., 1961; Yazdi et al., 2019) and nitrogen dioxide (NO_2) (Kulle and Clements, 1988) can lead to immune system dysregulation such as overexpression of inflammatory cytokines and chemokines (Bernstein et al., 2004; Fattorini and Regoli, 2020). This may have a direct impact on the severity of COVID-19 symptoms, since the body's ability to respond to the infection is compromised, and it also means that individuals with a history of exposure to air pollution are more likely to have comorbidities that make them more susceptible to severe health impacts when faced with a viral infection (Ciencewicki and Jaspers, 2007; Kim et al., 2018), including cardiovascular diseases, respiratory diseases, diabetes and hypertension (Yang et al., 2020).

Exposure to episodic acute air pollution, from both fine particulate matter and gases (O_3, NO_2 and sulfur dioxide (SO_2)), can also lead to intensified respiratory infection symptoms due to exacerbation of asthma and other inflammatory responses that can lead to acute respiratory distress (Guarnieri and Balmes, 2014). Ozone was found to disrupt the protease/antiprotease balance in the human airway,

leading to an increased risk of influenza infection (Kesic et al., 2012). Laboratory findings showed that O_3 can effectively inactivate influenza virus (Hiroshi and Jp, 2009; Sooryanarain and Elankumaran, 2015). Other evidence suggests that O_3 might enhance pulmonary innate immunity, which promotes allergic responses in susceptible individuals (Ali et al., 2018), indicating that the various mechanisms by which inhaled oxidants might modulate viral pathogenesis are highly complex and still remain to be fully characterized (Kesic et al., 2012).

There is substantially less evidence regarding impacts of air quality on virus transmission. Aerosol transmission has been identified in other coronaviruses (Doremalen et al., 2020; Hou et al., 2020), which offers reason to expect that it could be important for SARS-CoV-2 as well. Insomuch as particulate matter might serve as a transport medium for virus agents, enhancing aerosol stability or virus survival on aerosols, it might enhance transmission potential both directly and indirectly through deposition of viable virus on fomites, defined as inanimate objects that, when contaminated, can transmit pathogens to new hosts. The interaction between MAQ should also be considered (Chen et al., 2020; Le et al., 2020; Wang P. et al., 2020; Wang and Zhang, 2020). In addition to its direct impact on virus stability, meteorology may also influence virus transmission via its affects on air quality (Liu Y-S. et al., 2020), for example, O3 is formed by photochemical reactions between sunlight and precursor pollutants, which are favoured in warm conditions (Guarnieri and Balmes, 2014).

Epidemiological evidence from other respiratory viral diseases

While direct epidemiological evidence of MAQ influence on COVID-19 is only beginning to emerge, there is more mature evidence from other respiratory viruses (Audi et al., 2020; Moriyama et al., 2020). This evidence contributes to the expectation that COVID-19 may be sensitive to variations in MAQ factors.

The first conclusion that can be drawn from these potential analogue epidemics is that many respiratory viruses exhibit some form of seasonality, particularly but not only in temperate climates (Tamerius et al., 2011; Deyle et al., 2016). Notably, influenza, human respiratory syncytial virus and multiple human coronavirus strains exhibit clear wintertime peaks in mid-latitude regions (Monto, 2002; Tamerius et al., 2011; Midgley et al., 2017; Killerby et al., 2018). In subtropical climates, influenza has been shown to peak both in winter and summer (Zheng et al., 2017). Seasonality is also observed in some cases in tropical climates – for example, tropical influenza epidemics tend to occur during the rainy season although year-round transmission has also been documented in these climates (Tamerius et al., 2011; Hirve et al., 2016).

Attributing this seasonality to variability in specific or combined MAQ variables, however, has been more challenging. Studies on meteorological influence have generally focused on temperature or humidity – either relative humidity (degree of saturation of the air) or absolute or specific humidity (measures of the total amount of water vapour in the air). Quantification of these associations, and application to risk forecasts, is most advanced for influenza (Shaman et al., 2010; Dugas et al., 2013; Pei et al., 2018; Liu Q. et al., 2020). These studies have shown predictive skill for both temperature and humidity in mid-latitude regions (Shaman et al., 2010; Dugas et al., 2013; Pei et al., 2018). Absolute humidity, in particular, has been shown to be important in forecast systems (Shaman et al., 2017). One reason that absolute humidity might be a good indicator is that it is conserved between outdoor environments and heated indoor environments. This means that a meteorological estimate of absolute humidity is representative of conditions in indoor environments where most transmission occurs (Nguyen et al., 2014; Marr et al., 2019). The same is not true for temperature or relative humidity,

which are significantly different between outdoor and heated indoor environments (Nguyen and Dockery, 2016; Marr et al., 2019).

The mechanisms through which proposed meteorological drivers influence influenza, however, are still debated (Shaman et al., 2010; Lowen and Steel, 2014; Marr et al., 2019), reflecting general uncertainties regarding the dominant pathways of influenza transmission (Killingley and Nguyen-Van-Tam, 2013; Milton et al., 2013; Asadi et al., 2020). There is laboratory evidence that the influenza virus is sensitive to humidity and temperature (Shaman and Kohn, 2009; Irwin et al., 2011; Marr et al., 2019), but it is not clear whether this sensitivity is primarily responsible for observed epidemiological associations. Absolute humidity and temperature are often strongly correlated (Lipsitch and Viboud, 2009; Shaman et al., 2010), so it is possible that they serve as proxies for each other in epidemiological studies, or that dominant mechanisms flow through human behaviour patterns or human immune system responses (Kudo et al., 2019; Moriyama et al., 2020; Tay et al., 2020).

Seasonality of respiratory viruses, including influenza, may also be influenced by replication competition between competing viruses (Linde et al., 2009; Casalegno et al., 2010; Zheng et al., 2017; Nickbakhsh et al., 2019), further complicating epidemiological studies of MAQ sensitivity. Seasonality may also be influenced by societal behaviour, such as school holiday calendars (Figure 1) (Cauchemez et al., 2008, 2009; Huang et al., 2014). It should also be noted that not all viruses exhibit a wintertime peak, that the findings of the relative importance of humidity and temperature in driving influenza transmission vary from study to study (Martinez, 2018), and that MAQ-based influenza prediction was found to be less accurate in the tropics and subtropics (Kramer and Shaman, 2019). Nevertheless, the observed seasonality of these other respiratory viruses in temperate zones does imply that COVID-19, should it persist for multiple seasonal cycles, is likely to exhibit some form of cold season peak.

With respect to air quality, there is strong evidence for influenza and other respiratory viruses that poor air quality contributes to an increase in lower respiratory infection mortality (Troeger et al., 2018). Long-term studies of air pollution have presented convincing evidence of the effects of fine particles (PM2.5) on all-cause mortality, morbidity and mortality from cardiovascular and respiratory disease as well as diabetes and lung cancer. In addition, NO_2 and O_3 are associated with respiratory disease and mortality. There also exists strong evidence from short-term studies demonstrating effects on mortality and hospital admissions from very low exposure levels. Chronic diseases such as asthma, chronic obstructive pulmonary disease, lung cancer, heart disease and diabetes can predispose to and worsen the outcomes of respiratory infections such as COVID-19 (Brunekreef et al., 2021). Exposure to polluted air can lead to oxidative stress and damage to the respiratory system, which reduces resistance to infection (Ciencewicki and Jaspers, 2007). This applies to both acute and chronic exposure. For example, NO_2 levels have been found to correlate with respiratory infection hospitalizations on a timescale of days (Fusco et al., 2001) and with the prevalence of respiratory infections between different cities (Jaakkola et al., 1991). The influence of NO_2 on infection rates has been supported in laboratory studies (Rose et al., 1988).

Similarly, elevated particulate matter levels in outdoor air have been found to correlate with respiratory infection rates and severity for both acute (Schwartz et al., 1991; Wordley et al., 1997; Lin et al, 2005; Dominici et al., 2006; Chen et al., 2017) and chronic (Dockery et al., 1989; Pope III et al., 2004) particulate matter exposures. Similar evidence exists for carbon monoxide and SO_2 exposures, while studies of O_3 have yielded mixed results (Wolcott et al., 1982; Ali et al., 2018; Domingo and Rovira, 2020). A seven-year study documented a positive relationship between O_3 and paediatric influenza incidence. This study further demonstrated a significant interaction between particulate matter and temperature, whereas findings for O_3 were found to be temperature independent (Xu et al., 2016). Other modelling analyses showed that O_3 was negatively associated with influenza risk (Ali et al., 2018; Su et al., 2019). Specific to coronaviruses, air pollution was found to increase the case fatality rate for

SARS in China (Cui et al., 2003). Wind speed has been shown to interact with air quality factors (Cox et al., 1975; Kurita et al., 1985), that is, low wind speed can favour the stagnation of particulate matter mixed with viral agents (Coccia, 2020, 2021). A negative association was found between wind speed and the secondary attack rate of SARS, indicating that high wind speed potentially contributes to the dilution and removal of droplets, decreasing the suspension time of SARS in the air (Cai et al., 2007).

Air pollutants might also play a role in airborne transmission of viruses. It has been demonstrated that particulate matter can be a carrier of airborne pathogens (Cambra-López et al., 2010; Cao et al., 2014), and that aerosols can enhance the stability of some viruses, including Middle East respiratory syndrome CoV (MERS-CoV) (Doremalen et al., 2013). The importance of these pathways to infection risk relative to the influence that air pollution has on host resistance to infection is not well characterized.

Empirical studies of COVID-19 and SARS-CoV-2

As described above, the expectation that the transmission of SARS-CoV-2 and/or the severity of COVID-19 symptoms may be sensitive to MAQ factors stems from at least two lines of reasoning: first, a number of other respiratory viral infections exhibit geographical distributions, seasonal patterns, or interannual variability that can be associated with MAQ conditions (Audi et al., 2020; Moriyama et al., 2020); second, laboratory studies have shown that the ability of many viruses to survive in the air or on surfaces is sensitive to MAQ factors (Tang, 2009; Marr et al., 2019; Asadi et al., 2020). Many studies on COVID-19 have focused on the first line of reasoning, and have sought to quantify association between variations in MAQ factors and COVID-19 using epidemiological records (Briz-Redón and Serrano-Aroca, 2020; Mecenas et al., 2020; Shakil et al., 2020; Smit et al., 2020; McClymont and Hu, 2021). These studies have applied COVID-19 case data in a variety of ways, including analysis of raw case numbers through time, cumulative case numbers, case growth rates, and estimates of the basic reproduction number (the mean number of new infections caused by an infected individual in a completely susceptible population, in the absence of interventions) or effective reproduction number (reproductive number accounting for acquired immunity and interventions) derived from case data. Methodological approaches have included pure statistical analysis, including correlation analysis, multivariate regression, and machine learning techniques; the use of mechanistic transmission models; and combinations of statistical analyses and mechanistic transmission models (Smit et al., 2020). A smaller number of studies have attempted controlled experiments of SARS-CoV-2 sensitivities to MAQ conditions (second line of evidence) (Biryukov et al., 2020; Chan et al., 2020; Dabisch et al., 2020; Riddell et al., 2020; Smither et al., 2020).

The results of early epidemiological studies on the meteorological influence on COVID-19 rates yielded inconclusive and sometimes controversial results. A number of these studies reported potential associations between one or a combination of meteorological variables and metrics of COVID-19 spread, growth rate, or prevalence (Briz-Redón and Serrano-Aroca, 2020; Mecenas et al., 2020; Shakil et al., 2020; Smit et al., 2020; Carleton et al., 2021; McClymont and Hu, 2021; Pan et al., 2021; To et al., 2021). However, there is no consensus across studies on the relative importance of various meteorological variables (for example, UV radiation, temperature, humidity) and at this stage no firm or generalizable conclusions can be drawn on the statistical associations between COVID-19 and these leading MAQ candidate variables.

Inconsistent results from early studies do not imply MAQ factors will not affect SARS-CoV-2 transmission or COVID-19 severity; indeed, patterns in the northern hemisphere in recent months have suggested that COVID-19 might be exacerbated in colder climates. To date, epidemiological studies have been limited by the short and often inconsistent and inaccurate COVID-19 data record, by the fact that they have been performed with less than one full seasonal cycle of data, and by the dominant

influence of behavioural change and public health interventions during the first year of the pandemic (Jüni et al., 2020; Meyer et al., 2020; Rubin et al., 2020; Smit et al., 2020). Additional challenges have included correlation of meteorological variables, exclusion of other modulating factors, residual confounding, data heterogeneities across countries, high proportion of asymptomatic cases, and the difficulty of obtaining data on COVID-19 cases and on potential modulating factors at subnational levels. It would also be expected that during the pandemic phase of COVID-19 non-MAQ factors such as low levels of immunity in the population and vulnerability to infection and disease and impacts of non-pharmaceutical interventions (NPIs) likely predominate in determining epidemiological patterns (Baker et al., 2020; Jüni et al., 2020; Meyer et al., 2020), while seasonality and effects of MAQ may emerge during the post-pandemic phase (Kissler et al., 2020) – though other modelling work has suggested that seasonality does have a potential influence even early in epidemics (López and Rodó, 2020). For this reason, it is important to emphasize the uncertainty in our current understanding of the potential impact of MAQ factors on COVID-19, while continuing to investigate potential for weather and climate information to support public health policy and the COVID-19 response.

Peer-reviewed laboratory studies of SARS-CoV-2 sensitivity to meteorological variables are still rare. Results of available studies do include some of the evidence of meteorological sensitivity. This includes findings that the survival of SARS-CoV-2 in aerosols as well as on surfaces declines in the presence of increased UV-A and UV-B radiation (Ratnesar-Shumate et al., 2020; Schuit et al., 2020b). This pattern has been found for SARS-CoV-2 in aerosols as well as on surfaces, which was found to be effectively inactivated by UV-A and UV-B intensities representing simulated levels of natural sunlight. Exposure to simulated sunlight of SARS-CoV-2, suspended in either simulated saliva or culture media, resulted in a 90% inactivation every 6.8 minutes and 14.3 minutes, respectively. Laboratory studies also suggest that the virus survives longer under colder conditions (Biryukov et al., 2020; Chan et al., 2020; Dabisch et al., 2020); for example, in the presence of high-intensity simulated sunlight and 20% relative humidity, mean decay rates of SARS-CoV-2 were 18.9 ± 4.8% per minute at 10 °C compared to 38.1 ± 8.9% per minute at 40 °C (Dabisch et al., 2020). Results for humidity are less clear, with some indication that there is a slight increase in virus survival at lower relative humidity (20%) (Biryukov et al., 2020; Dabisch et al., 2020) and other experiments showing greater survival at both medium (40%–60%) and high (68%–88%) relative humidity, depending on different suspension matrices (Smither et al., 2020). Results of these types of laboratory studies are critical for understanding the mechanisms that might link meteorology to COVID-19 transmission, and they can have important implications for assessing risk or informing climate control (for example, air conditioning) in indoor environments. These studies are, however, fundamentally different from epidemiological studies in that they focus on the specific issue of virus survival under controlled conditions rather than on meteorological influences on disease risk at population scale. Their transferability to real-life settings is not always straightforward.

Studies of the influence of air pollution on COVID-19 have addressed both the acute impacts that compromised air quality might have on transmission or severity of COVID-19 symptoms and the influence that chronic exposure to air pollution can have on human susceptibility to COVID-19. Consistent with studies of other respiratory infections, early studies have indicated that there is a positive relationship between prevailing air quality conditions (Wang B. et al., 2020; Wang R. et al., 2020; Yao et al., 2021), especially fine particulate matter (PM2.5), and COVID-19 mortality rates (Wu et al., 2020; Yao et al., 2020b; Jiang and Xu, 2021), as well as with total case counts (Borro et al., 2020). One global analysis concluded that long-term exposure to ambient fine particulate air pollution contributed to approximately 15% (95% confidence interval 7%–33%) of COVID-19 mortality worldwide, through the third week of April 2020, with higher rates in more polluted environments (Pozzer et al., 2020).

While a number of studies have found associations between air pollution and COVID-19 rates (Conticini et al., 2020; Dettori et al., 2020; Ogen, 2020; Petroni et al., 2020; Wu et al., 2020; Zhu et al., 2020;

Yao et al., 2021), concerns have arisen about methodological issues in some studies (Heederik et al., 2020; Villeneuve and Goldberg, 2020). This includes studies of the impact of O3 on COVID-19 dynamics, which have yielded inconsistent results, demonstrating either positive or negative associations between O3 and COVID-19 risk (Adhikari and Yin, 2020; Ran et al., 2020; To et al., 2021). In addition, several studies have investigated the reduction in air pollution levels as a result of COVID-19 restrictions (Kumari and Toshniwal, 2020; Menut et al., 2020; Ordóñez et al., 2020; Venter et al., 2020; Saxena and Raj, 2021), which might result in beneficial health effects, for example, short-term and long-term avoided mortality from PM2.5 exposure (Giani et al., 2020). However, the totality of lockdown-induced changes such as delayed treatments for disease, mental health impacts and changes in physical activity should be considered, which might offset or surpass the observed reductions in burden of disease due to improved air quality during COVID-19 lockdown periods (Brunekreef et al., 2021).

In general, disentangling any independent effects of air pollution from effects of other causes of COVID-19 outbreaks has been challenging, potentially resulting in an overestimation of the effects of air pollution on COVID-19 occurrence and severity in some studies (Brunekreef et al., 2021). Results therefore need to be confirmed and consolidated by controlling for individual-level risk factors (Wu et al., 2020). To date, there has been no peer-reviewed evidence of the direct influence that various air pollutants have on SARS-CoV-2 transmission. Indoor air pollution, caused by sources such as smoking, woodstoves and fireplaces, is another potentially important and understudied factor in SARS-CoV-2 transmission, given that people spend most of their time indoors (Brunekreef et al., 2021). Future large cohorts and administrative databases, enabling the inclusion of individual-level data and high-resolution spatiotemporal data, will provide opportunities to thoroughly investigate effects of air pollution on COVID-19 dynamics (Brunekreef et al., 2021).

Influence of MAQ relative to other factors

The trajectory of the COVID-19 pandemic depends on many factors, such that potential MAQ influences must be evaluated in the context of and in combination with other drivers of disease risk. Susceptible supply (Baker et al., 2020), NPIs (Kissler et al., 2020; Rubin et al., 2020), access to vaccines, health system capacity (Armocida et al., 2020; Legido-Quigley et al., 2020) and population demographics (for example, population density and age structure) (Dowd et al., 2020; Kashnitsky and Aburto, 2020; Rocklöv and Sjödin, 2020) or underlying health burdens (Richardson et al., 2020; Yang et al., 2020) have all been identified as determinants of risk that could dominate MAQ influences, particularly in early stages of the pandemic. Understanding and, where possible, quantifying the relative impacts of these compound factors – for example, the influence of temperature versus the influence of an NPI, such as mask mandates or limits on gatherings, can be critical for optimizing control measures (Rubin et al., 2020). A frequently noted case of NPIs affecting observed MAQ COVID-19 associations is the fact that many northern hemisphere countries began to implement masking requirements and movement restrictions in the spring of 2020, as temperatures began to warm and specific humidity increased. In the absence of careful analysis and adequate data on NPIs, this could lead to unjustified conclusions regarding the protective influence of warm temperatures or higher humidity conditions. At this stage, quantitative estimates of the influence that MAQ and non-MAQ factors have on COVID-19 transmission are too widely dispersed to support definitive and robust statements on the conditions under which MAQ influence may significantly alter the trajectory of the pandemic (Briz-Redón and Serrano-Aroca, 2020; Mecenas et al., 2020; Shakil et al., 2020; Smit et al., 2020). Nevertheless, the fact that COVID-19 transmission continued, often at high rates, in northern mid-latitudes during the boreal summer and autumn of 2020, and the fact that transmission has been severe in some tropical environments, suggests that hypothesized MAQ sensitivities have not dominated transmission patterns to date, despite the need for more detailed investigation. The potential for certain behavioural interventions to have a strong influence on pandemic trajectory, in contrast, has been clearly established

(Flaxman et al., 2020; Haug et al., 2020; Pan et al., 2020). A recent study in Manaus, Brazil, for example, estimated that the cumulative COVID-19 incidence reached approximately 76% under conditions of poor control (Buss et al., 2020). Reduced mobility in response to public health warnings or mandated lockdowns, for example, has had a strong influence on transmission rates in some contexts (Badr et al., 2020; Kraemer et al., 2020; Wang B. et al., 2020), though this influence has been shown to vary over time (Badr and Gardner, 2020).

MAQ-informed risk forecasts: status and prospects

A MAQ-informed disease forecast is useful insomuch as it can predict future changes in disease risk with actionable accuracy and precision. While several groups have proposed MAQ-informed approaches to forecasting COVID-19 risk (da Silva et al., 2020; Gupta et al., 2020; Huang J. et al., 2020; Sajadi et al., 2020), at present these proposed approaches have not been evaluated in the context of experimental or operational forecast systems. A number of institutions and research groups provide scientifically robust information about the potential drivers discussed in the previous sections: key MAQ variables, social and demographic variables, and sometimes tools to cross link these with COVID-19 metrics and policies (for example, the European Centre for Medium-Range Weather Forecasts Copernicus Monthly climate explorer for COVID-19, and the National Aeronautics and Space Administration/Columbia University Socioeconomic Data Analysis Center Global COVID-19 Viewer). But these platforms are more "toolboxes" than integrated decision-making "operational" systems. Many meteorological services, health services, and complementary institutions do have the capacity to provide climate-informed risk estimates rapidly if and when robust exposure–response relationships are established. Growing indications that COVID-19 does have some degree of MAQ sensitivity point to the potential to implement such forecast systems in the future, perhaps as a component of a package of seasonal health hazards. But it is critical that these relationships and their importance relative to other factors be established robustly, based on experience from existing actionable forecast systems, prior to their use in decision-making.

Should evidence consolidate in favour of moving towards actionable MAQ-informed forecasts, it will be instructive to consider experience with other infectious disease forecasts. For temperate zones, influenza again provides a potential analogue as an MAQ-sensitive viral respiratory infection (Lowen et al., 2007; Moriyama et al., 2020). Several seasonal influenza forecast models have been implemented and show skill in predicting the timing and magnitude of the seasonal peaks several weeks in advance (Dugas et al., 2013; Pei et al., 2018; Reich et al., 2019). Although seasonality is incorporated into these models through several non-MAQ predictor variables, inclusion of humidity as a predictor has been found to improve the prediction skill. Notably, the use of average seasonal humidity conditions can be just as or more effective than real-time monitoring of humidity variability when generating influenza forecasts (Shaman et al., 2017; Kramer and Shaman, 2019), pointing to the fact that analysis of seasonal average MAQ conditions can be a primary source of actionable information relative to forecasts. It is also recognized that active communication and system co-development between modellers and public health practitioners is necessary for influenza forecasts to be used to their full potential (Doms et al., 2018).

Meningococcal meningitis offers an example of actionable MAQ-informed forecasts in the tropics (Sultan et al., 2005; Cuevas et al., 2007; Ayanlade et al., 2020). Work performed under a WHO initiative on meningitis forecasts for sub-Saharan Africa has demonstrated that absolute humidity is a meaningful predictor of disease risk, with applications to climatological risk mapping and real-time forecast (Thomson et al., 2013; García-Pando et al., 2014). MAQ-informed forecasts have also shown potential for water-borne diseases (Baracchini et al., 2017), vector-borne diseases, including dengue (Hii et al., 2012; Lowe et al., 2014; Petrova et al., 2020), Rift Valley fever (Linthicum et al., 1999;

Anyamba et al., 2009), malaria (Hoshen and Morse, 2004; Thomson et al., 2006), and, in temperate zones, west Nile virus (Manore et al., 2014; DeFelice et al., 2017), among others. While the ecological basis that underlies MAQ sensitivity in vector-borne diseases is quite different from the potential MAQ-influences on COVID-19, a number of key lessons can be learned. These include the critical importance of high-quality data (Yamana and Shaman, 2020) and stakeholder engagement (Lowe et al., 2020) when attempting an actionable forecast.

A key lesson from experimental and operational MAQ-informed disease-risk monitoring and forecast systems is that monitoring and forecast can be particularly useful when vaccines or other effective treatments are available, as the prediction can inform effective deployment of supplies and personnel (García-Pando et al., 2014). In the case of COVID-19, this emphasizes the potential value of integrating MAQ-informed risk analyses with the analysis of NPIs and vaccination strategies. Rather than obviating the need for forecasts, the presence of effective prevention and treatment strategies often elevates their utility to decision makers (López and Rodó, 2020).

BEST PRACTICES FOR RESEARCH AND FORECASTS

Open collaboration and communication

The pandemic has shown how vital openness, interdisciplinarity and international collaboration are in underpinning factual, data-driven decision-making and in providing actionable information. This is particularly true for developing countries that might lack comprehensive research structures. The increase in openness and collaboration across countries and institutions during the first year of the COVID-19 pandemic has been a remarkably positive development, and the same level of openness must be maintained in the future. Good governance and sustainable funding are required to establish interdisciplinary and equitable cooperation, enabling systemic change at international, national, regional and local levels. This proposition is not new but brings multiple challenges, including establishing

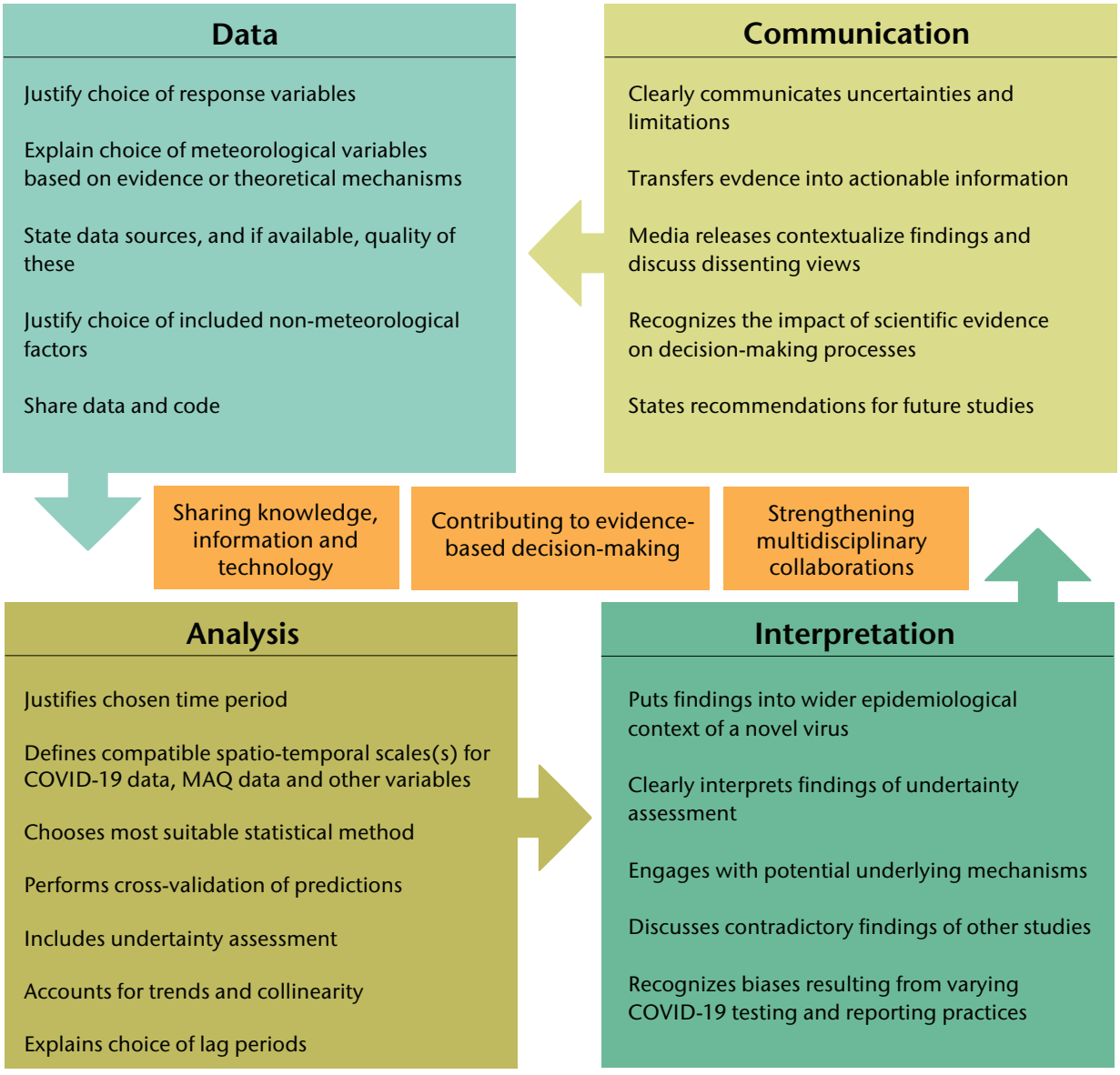

Figure 2. Schematic of best practices for research on MAQ influences on COVID-19, modified after Zaitchik et al. 2020.

principles of cooperation, for example in terms of clarity of parameter definitions, format standards for data interoperability, statistical methods, data resolution, data aggregation methods, frequency, updates, standardized protocols (for reporting, for example), metadata requirements, code sharing and privacy considerations where applicable (Figure 2). In some cases, open collaboration can run into conflict with national data policies. This is a persistent challenge that calls for diplomacy and appropriate enabling environments to encourage open data exchange.

The WMO Global Framework for Climate Services helps to ensure MAQ information, such as that produced by National Meteorological and Hydrological Services, to be more effectively used in different applications, including public health (WMO, 2014). This is achieved by tailoring MAQ information to the specific needs of the application, through the co-design of the information services between data providers and users. This joint development process strengthens the collaboration between National Meteorological and Hydrological Services and health agencies, and the products can produce diverse potential benefits. These include providing a more comprehensive understanding of health risks, enhancing integrated disease surveillance and monitoring systems, and providing tactical information to improve health service design and delivery. Such collaborations are also critical for implementing effective health early warning systems; supporting community risk awareness; and informing mid- and long-term health sector planning and the evaluation of climate-sensitive health services and interventions (WHO/WMO, 2016).

Effective communication between researchers, data providers, disseminators of information and end users is a critical component of successful climate services. In the case of MAQ-informed COVID-19 risk analysis and forecasting, this need for strong communication is particularly critical. Public health policies and personal behavioural decisions are often made quickly with imperfect information, and a miscommunicated or ineffectively communicated MAQ-informed risk analysis has the potential to do substantial harm (Carlson et al., 2020). For this reason the research and forecast communities must assume extra responsibility when disseminating results (Figure 2) (Zaitchik et al., 2020). This includes clear, plain-language summaries of key findings, limitations of the analysis, generalizability, and relationship to other relevant studies. It also includes engaging with media and social media in a measured and balanced manner. It is not uncommon, in general applications, for a research result or an experimental forecast to be promoted without full context. In the case of COVID-19, researchers and data providers must engage carefully to pre-empt misinterpretation or overinterpretation of results (Carlson et al., 2020).

Methodological considerations

Researchers and users of research products should recognize the intended use of various analysis methodologies employed in COVID-19 studies. As noted earlier in this report, that includes clarity on the difference between laboratory studies performed under controlled conditions – which are critical for informing mechanistic understanding of MAQ sensitivities – and epidemiological studies that attempt to infer MAQ influence on the basis of observed infection rates and health outcomes. The former can offer direct mechanistic evidence for potential MAQ influence on virus dynamics, and can be critical for informing risk mitigation strategies, but it can be difficult to apply sensitivities observed in a controlled laboratory setting to inform quantitative transmission risk estimates outside the laboratory. The latter can be directly informed by "real-world" information on the dynamics of the pandemic, but statistical coefficients (or model parameters) in these analyses that quantify MAQ sensitivities are, in general, derived from imperfect data and without any strong constraints to pinpoint mechanisms that underlie estimated sensitivities. As such, a laboratory-reported sensitivity to an MAQ variable – for example, reduced SARS-CoV-2 survival at higher temperature – should not be interpreted as a quantitative estimate of temperature influence on transmission rates. At the same

time, a statistical association between temperature and COVID-19 cases – even one that is found to be robust across multiple studies – does not necessarily tell us why we see the association (for example, human behavioural factors versus virus survival rates), which may place limits on the kinds of health policy decisions it can inform.

Within the world of epidemiological analysis, a distinction should be made between process-based models, such as compartmental models that simulate interactions between susceptible, infectious and recovered individuals within a population (Baker et al., 2020; Shi et al., 2020); fully empirical models, which fit statistical relationships to observed data without a priori hypotheses (Merow and Urban, 2020; Meyer et al., 2020; Rubin et al., 2020); and hybrid approaches that include characteristics of both. This distinction is relevant when interpreting a projection based on modelled MAQ sensitivities. For example, a fully empirical model that reveals statistically significant associations between COVID-19 and temperature does so solely on the basis of the data used to train the model. The generalizability of the model, then, depends on the nature of the input data and the anticipated stability of statistical associations as the COVID-19 pandemic evolves. The demonstrated predictive capability of such models also depends on predictive performance in out-of-sample cross validation – that is, testing a model's prediction against data that were not used to train the model. Good statistical fit to training data does not, in itself, constitute evidence of predictive skill.

In contrast, a process-based model includes embedded hypotheses that inform the model's projection of transmission risk. That can be a strength, in that it means the model can be used to understand potential MAQ sensitivities in the context of broader disease dynamics (Baker et al., 2020), yielding insights on how MAQ influence might be expected to evolve with pandemic stage or in the context of public health interventions. Such models, however, can be difficult to parameterize, and they are sometimes applied to produce detailed projections that may give the impression of precision even in the absence of robust data for the disease in question. Thus, the results of projected MAQ influence on COVID-19 risk derived from process-based models should be communicated and interpreted with understanding of whether key conclusions are grounded in COVID-19 data. For example, many valuable studies use process-based models to explore hypothetical scenarios that are not constrained by data, or that are based on analogy to other respiratory viral infections. These hypothetical and analogue studies can be very useful for exploring transmission dynamics and projecting potential epidemic trajectories, but their purpose needs to be stated clearly so that they are not mistaken for specific predictions.

Data considerations

Data quality, usability and interoperability pose significant challenges for studies of MAQ influence on COVID-19 risks. These challenges apply to at least three types of data: the COVID-19-related outcome variables, MAQ data, and data on non-MAQ influences on COVID-19 risk, including space- and time-varying influence of NPIs and other behavioural factors.

With respect to the outcome variable, COVID-19 analysis has, in general, struggled with inconsistent and predominantly unreliable data on COVID-19 case numbers, COVID-19-attributed deaths, excess mortality during the pandemic and other COVID-19-related health indicators. These challenges have stemmed from uneven policies on and access to COVID-19 testing, reporting delays, limited health system resources and the lack of existing standards and mechanisms to create and maintain databases for pandemic monitoring and response (Gardner et al., 2020). COVID-19 has recently completed the first annual cycle, indicating the limited time frame of available COVID-19 data but also future prospects of increased data availability, which could enhance the robustness of modelling studies. Given these limitations, researchers working on MAQ-informed COVID-19 risk must be aware of potential biases in

the health data used in their analyses. The use of quality-controlled centralized COVID-19 databases such as the Johns Hopkins University Coronavirus Dashboard (Dong and Gardner, 2020), the New York Times Case Inventory (*The New York Times, 2020*), Our World in Data COVID-19 database (Roser et al., 2020) the COVID Tracking Project (*The Atlantic, 2020*), the Financial Times excess mortality database (Burn-Murdoch et al., 2020), among others, offers some confidence in the raw data used. Nevertheless, even these data aggregators can inherit data artifacts from contributing sources, such that additional quality control is required when the data are applied to large, automated analyses (Badr et al., 2020). Once clean data are obtained, researchers must consider carefully the meaning of their selected response variable. For different applications, it may be important to look at total case counts, incidence, growth rates, or reproductive numbers (R0 or Rt) (Smit et al., 2020). There is no single right answer for this choice of response variable, though there are known advantages and disadvantages of each (Smit et al., 2020). It is critical that MAQ-informed COVID-19 risk analyses justify the choice of response variable, ensure that the estimates used for the selected response variables are robust, and interpret study results in the context of what that response variable means for risk assessment. Regardless of the form of the response variable, it is also critical to acknowledge and, when possible, adjust for data biases resulting from uneven access to COVID-19 testing, reporting delays and inconsistency in reporting of results (Smit et al., 2020).

For the MAQ variables themselves, studies have made use of in situ station-based data, remotely sensed data, meteorological reanalysis, and combinations of the three, such as the Copernicus Climate Data Store and National Oceanic and Atmospheric Administration (NOAA) datasets for meteorology, and the Copernicus Atmospheric Data Store for main air pollutants (Copernicus Climate Change Service, 2020; NOAA Climate.gov, 2014). The quality of these data sources should be verified with data producers or directly with the information provided in the respective peer-reviewed publications. Just as importantly, MAQ data must be applied at a spatial scale appropriate to COVID-19 analysis. The use of single stations or national averages, for example, can be problematic when analysing COVID-19 patterns in large or climatically diverse countries (including strong land–sea differences, orography, urban–rural contrasts, and the like). MAQ data should, in general, be reconciled with COVID-19 data at the finest spatial scale for which both forms of data are considered to be reliable. In cases where this is not possible at a resolution meaningful for MAQ analysis, researchers should be cautious in pursuing and interpreting MAQ-informed studies. Finally, while empirical studies that attempt to make MAQ-informed COVID-19 predictions using multiple MAQ variables can reveal new and meaningful associations, interpretation of such studies should include consideration of the potential mechanisms through which MAQ conditions influence COVID-19 risk. This can contribute to broader understanding and can also clarify instances where a statistical association might be the result of one variable serving as a proxy for another, more process-relevant environmental condition.

To quantify associations between MAQ factors and variations in COVID-19, models should also consider other intrinsic and extrinsic factors that influence the trajectory of the pandemic. As described in the following section, these factors include quasi-static health-relevant factors, such as demographic data, prevalence of comorbidities at the scale of analysis, healthcare-system capacity, metrics of economic development, and transportation infrastructure (Briz-Redón and Serrano-Aroca, 2020; Smit et al., 2020). They also include time-varying non-MAQ factors that have evolved over the course of the pandemic, such as mobility, adoption of protective behaviours, and the implementation and enforcement of NPIs. There is no clear consensus regarding the best sources of information on these diverse epidemiological factors, and in many cases there is simply an absence of reliable data. Given these limitations, it is critical that empirical studies of MAQ influences clearly state a rationale and methodology for the treatment of non-MAQ factors, both to ensure that the best effort is made to control for relevant confounders and to indicate relevant limitations, assumptions and uncertainties. There is strong potential for correlated processes to lead to false MAQ associations with COVID-19 data records, if studies are not properly conducted. This risk must be acknowledged, communicated

and guarded against. Collaboration across fields, including by climate data providers, epidemiologists, health data scientists and experts in risk communication is particularly important in this regard.

Covariates, confounders and interactions

Both longitudinal and cross-sectional studies of MAQ influence on COVID-19 risk must, at a minimum, account for behavioural change in response to COVID-19. While an argument can be made that COVID-19 awareness and interventions were low in the very earliest stages of the pandemic, it is evident that by the spring of 2020 there was widespread change in behaviours and policies that influence disease transmission. It is difficult to account for these diverse behavioural changes and interventions when assessing the impact of MAQ factors on COVID-19. Measures of personal mobility (Google LLC, 2020), inventories of COVID-19 response policies (Hale et al., 2020), pollutant emissions changes (Guevara et al., 2020), and dates of cessation of in-person schooling or other public activities (Auger et al., 2020) have all been employed as controls alongside MAQ variables. Cross-sectional studies – or studies of any design that include analysis of COVID-19 across different locations – must additionally account for demographic differences between locations. Information on differences in underlying comorbidities, connectedness to other populations, and health-system capacity can also provide useful controls, where such data are available (Badr et al., 2020; Legido-Quigley et al., 2020; Richardson et al., 2020; Yang et al., 2020). In addition, studies need to adjust for factors that might considerably influence the estimate of the response variable, including COVID-19 testing rates and reporting methodologies/delays. Emerging research should also consider the launch of large-scale COVID-19 vaccination on a global scale, including vaccine production capacities and population coverage. The future trend of the COVID-19 pandemic will most likely depend on the coverage and effectiveness of approved COVID-19 vaccines, which may also interact with changes in human behaviour and weather.

Forecasts and communication of uncertainty

The skill of a disease forecast is its capacity to usefully project future changes in disease risk. Such forecasts are motivated by the potential to support early implementation of measures that minimize the consequences for the public. Those measures may be public health actions such as awareness campaigns, vaccination campaigns or preparedness of healthcare systems. The capacity to predict COVID-19 patterns using environmental and epidemiological data will determine the feasibility of establishing a meaningful forecast system.

If established, such forecasts will inevitably come with substantial uncertainty. Indeed, forecast uncertainty is a crucial communication challenge across meteorology, epidemiology, and many other prediction-oriented fields. As the challenge is well recognized, there are extensive resources on communication strategies and best practices (National Research Council, 2006; WMO, 2008; Carr et al., 2016; Lutz et al., 2019). MAQ-informed COVID-19 forecasts could potentially influence policy decisions relevant to the trajectory of the pandemic and its broader societal impacts. This makes adherence to best practices critical, even for “experimental” forecasts that may be disseminated without any claim to be operational; once released to the public, a forecast can be treated as authoritative even if it is intended only as a research exploration. All forecast products, therefore, should include uncertainty estimates and statements on the purpose of the forecast and its limitations, as well as detailed and acquainted sensitivity tests. If quantitative uncertainty estimates are not possible, indicative measures of confidence should be provided in a prominent way. Forecasts must be supported with accessible and relevant performance metrics, including out-of-sample performance statistics for any model that is presented as a basis for actionable predictions.

Report methodology

This report was drafted by a WMO Research Board Task Team of experts, selected on the basis of their contribution to studies of environmental influences on SARS-CoV-2/COVID-19 and other infectious diseases. Since September 2020, the Task Team has performed a thorough review of available peer-reviewed literature on COVID-19 and, as relevant, other MAQ-sensitive respiratory viruses. The text of the report represents an expert evaluation of this rapidly evolving literature. Only peer-reviewed articles accepted for final publication prior to January 8, 2021 are cited in the report. These articles were additionally subjected to a Task Team evaluation of quality and reliability prior to being included. We note that the Task Team's approach to the literature was to focus on those studies that were deemed to be most relevant to report goals. The report is not a systematic review, and does not attempt to be comprehensive in its treatment of available literature. We also acknowledge that the fast pace of COVID-19 publication and the tendency for authors to disseminate their reports on preprint servers prior to review has meant that the peer-reviewed literature has lagged behind publicly discussed research findings. The Task Team has accepted this lag to avoid including findings that have not yet passed peer review in any formal way. Nevertheless, the availability of potentially important studies on preprint servers does inform scientific discourse, and where relevant we have noted emerging trends in pre-review literature. Statements in the report relating to methodological recommendations and to the potential of MAQ-informed COVID-19 forecasts represent the consensus of the Task Team, as supported by peer-reviewed citations where appropriate.

LIST OF SCIENTIFIC TERMS

Absolute humidity	Total mass of water vapour contained in a unit volume of air, irrespective of temperature, expressed as grams per cubic metre
Adaptive immune system	Part of the immune system that provides an antigen-specific response following exposure to pathogens or foreign substances, primarily including B cells, T cells and circulating antibodies
Airborne or aerosol transmission	Infection through exposure to smaller virus-containing respiratory droplets and particles (usually ≤5 μm) that remain suspended in the air over longer distances
Basic reproduction number	Average number of secondary infections caused by an infected individual in a completely susceptible population, in the absence of interventions
Case fatality rate	Proportion of deaths from a certain disease compared to the total number of individuals diagnosed with the disease for a particular time period
Case growth rate	Measure to capture changes in number of infections per unit time, relevant to indicate the speed of a disease spread
Confounding factors	Factors associated with the exposure and response variables that may mask or modify an actual association or falsely demonstrate a non-existing association between study variables
Contact transmission	Infection through direct contact with an infectious person or indirect human-to-human contact via contaminated surfaces or objects
COVID-19	Coronavirus disease 2019, caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)
Cross-sectional studies	Observational studies that analyse data from a population or a representative subset at a specific point in time
Droplet nuclei	Respiratory droplets ≤5 μm in diameter, remaining suspended in the air for longer distances and time periods
Droplet transmission	Infection through exposure to virus-containing respiratory droplets within shorter distances between the infected and exposed person
Effective reproduction number	Average number of secondary infections per infectious case, accounting for acquired immunity and interventions
Empirical models	Models based on observable phenomena that fit statistical relationships to observed data without a priori hypotheses
Excess mortality	Difference between the observed number of deaths in a specific time period and expected numbers of deaths, based on average deaths over the same period in previous years
Fomite	Inanimate objects that, when contaminated or exposed to infectious agents, can transmit pathogens to new hosts
Human coronaviruses	Besides SARS-CoV-2, there exist six other human coronaviruses: SARS, MERS, HKU1, NL63, OC43 and 229E

Human thermoregulation	Essential human ability of regulating and exchanging body heat, including complex physiological self-regulatory mechanisms and processes required to achieve a stable internal environment
Human respiratory syncytial virus	Common human respiratory virus that usually causes respiratory infections with mild, cold-like symptoms, but that can be serious for infants and elderly people
Immune evasion	Strategy used by pathogenic organisms to evade a host's immune response to enhance pathogen survival and maximize the probability of being transmitted to a new host
Incidence	Number of individuals who develop a specific disease in a population during a specified time period
Inflammatory chemokines	Extended family of small cytokines produced during infection or injury that determine the migration of cells to sites of infection or damaged areas
Inflammatory cytokines	Type of signalling molecules that are secreted from immune cells and play an important role in regulating the host defence against pathogens, mediating the innate immune response
Innate immune system	Non-specific first line of defence against pathogens, comprising physical barriers (for example, skin) and both cellular (for example, granulocytes, natural killer cells) and humoral (complement system) defence mechanisms
Interferon-stimulated genes	Genes primarily involved in the innate immune response, whose expression is stimulated by interferons (group of signalling proteins), inducing an antiviral state effective against viruses, bacteria and parasites
Middle East respiratory syndrome	A viral respiratory disease, first identified in Saudi Arabia in 2012, transmitted between animals and humans, with most cases linked to the Arabian Peninsula
Mucociliary clearance	Defence mechanism of the lungs in which mucus and potentially harmful foreign substances contained in it are moved out of the lungs
Out-of-sample cross validation	Model validation technique for assessing how the results of a statistical analysis generalize to an independent data set, used for predictive models to test the model's ability to predict new data that was not used in estimating it
Oxidative stress	Disturbance in the balance between the production of reactive oxygen species and the ability of the biological system to detoxify these reactive products
Prevalence	Total number of individuals who have a disease at a specific period of time, regardless of when they first developed the disease
Process-based modelling	Models that represent disease processes with specific hypotheses about the mechanisms that drive infection dynamics, for example, compartment models simulating interactions between susceptible, infectious and recovered individuals

Protease/antiprotease balance	Delicate interaction of enzymes and proteins that are involved in the respiratory function, critical for cellular regeneration, cellular repair, tissue homeostasis and host defence
Relative humidity	Fraction of water vapour in the air, relative to the maximum amount of vapour in the air at a given temperature and pressure, expressed as a percentage
Residual confounding	Distortion that remains after controlling for confounding in the design and/or analysis of a study
Respiratory droplet	Droplets produced by exhalation, which span a wide spectrum of sizes and can be divided into the categories of larger droplets and smaller droplets (or droplet nuclei ≤5 μm)
Secondary attack rate	Probability that an infection occurs among susceptible people within a specific group, which can provide an indication of how interactions relate to transmission risk
Severe acute respiratory syndrome	A viral respiratory illness, SARS was first reported in China in 2003 and spread to many other countries before it was contained within one year
Severe acute respiratory syndrome coronavirus 2	First identified in December 2019, SARS-CoV-2 is the virus that causes COVID-19 disease
Specific humidity	Mass of water vapour in a unit mass of moist air, expressed as grams per kilogram
UV radiation	UV radiation is classified into three types with different wavelength ranges (the shorter the wavelength, the more harmful the UV radiation), differing in their biological activity and the extent to which they can penetrate the skin
UV-A	UV radiation with a wavelength range of 315–400 nm, accounting for approximately 95% of the UV radiation reaching the Earth's surface, and which is able to penetrate into deeper layers of the skin
UV-B	UV radiation with a wavelength range of 280–315 nm is mostly filtered by the atmosphere and cannot penetrate beyond the superficial skin layers
UV-C	UV radiation with a wavelength range of 100–280 nm is completely filtered by the atmosphere and represents the most damaging type of UV radiation
Virus envelope	Outermost layer of enveloped viruses (compared to non-enveloped viruses), consisting of a lipid bilayer that protects the genetic material and interacts with cell receptors
Virus-specific CD8+ T cells	Mediators of adaptive immunity, critical for mediating clearance following acute viral infections in the lung and capable of providing protection against secondary infections

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