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**The ventilation of buildings and other mitigating measures  
for COVID-19: a focus on winter 2020**

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# The ventilation of buildings and other mitigating measures for COVID-19: a focus on winter 2020

The Royal Society Rapid Assistance for Modelling the Pandemic (RAMP) Project  
Task 7: Environmental and Aerosol transmission

October 27, 2020

**This document constitutes draft guidance which has been published for consultation purposes only.**

**The intended audience include: advisors to UK Government (e.g. SAGE), Public Health England, relevant government departments (e.g. the Department for Education), ventilation practitioners (e.g. manufacturers and designers), skilled building service managers, and interested scientists.**

## 1 Executive summary

Winter 2020 presents significant risks in managing the ventilation of buildings and maintaining a healthy indoor environment. The extent of the COVID-19 pandemic, both in terms of its stage of development and the controlling measures in-place, varies widely across the globe both inter- and intra-country. Within the United Kingdom of Great Britain and Northern Ireland (UK) the disease seems to be at a dangerous juncture with the reproductive number (i.e. the average number of infections arising from a single infectious case) currently above one for COVID-19 (e.g. the R-number is being reported, as of September 18, as 1.1–1.4, UK Government, 2020). This is at a time when cooler weather is approaching where people typically spend longer indoors in the company of others and the supply of outdoor ventilation air is reduced. Simultaneously, the UK and other governments are trying to re-open their economies, which is accompanied by increased social interactions (e.g. schools reopened in August and September 2020 throughout the UK, with universities soon to follow). This report is intended to review much of the knowledge surrounding the indoor spread of COVID-19, and present new results which can inform guidance for mitigating the impact of the disease — our focus has been the UK but our findings will be more widely useful.

It is the premise of this report that COVID-19 may be spread via three main routes (droplet, contact and airborne) all of which we assume to be potentially significant. By consideration of the indoor environment and our behaviour within it, we discuss potential mitigating strategies for all three routes. However, our primary focus is the airborne/aerosol route, since mitigating the spread via this route is the most challenging. The evidence suggests that adequate supply of outside (or at least uncontaminated) air is crucial in helping ensure the reproductive number of a particular indoor space is minimised and ideally remains below one. Findings suggest that for many indoor spaces which are regularly attended by the same/similar group of people (e.g. an open plan office or school classroom) adhering to existing design guidance for adequate ventilation (e.g. 10 litres per second per person, or 10l/s/p, for offices (Chartered Institution of Building Services Engineers, 2015) and 5–10l/s/p for school classrooms (see UK Government, 2018a)) and occupancy densities, results in airborne infection risk that remains relatively low for most reasonable scenarios. Modelling estimates suggest that in these cases the reproductive number is less than one, suggesting each infection is expected to give rise to less than one new infection. However, for any indoor space where respiratory activity levels of a potential infector are expected to increase to anything above those corresponding to sedentary talking, increased caution is required.

Appropriate social distancing (§2.2) and/or the wearing of face coverings appear to be effective measures to help mitigate the risk of transmission via the droplet route. Increased hand hygiene and the cleaning of surfaces, particularly high-touch public surfaces (i.e., those frequently touched by more than one individual), will reduce transmission via the contact route. Cleaning of surfaces using disinfectants

1  
2  
3 based on alcohols and reduced contact with these surfaces appear to be effective measures to reduce  
4 transmission (§2.1.2). Indoor spaces that bring individuals together over long periods, e.g. open-plan  
5 offices, school classrooms and the like, or those that lead to increased respiratory activity, e.g. gyms,  
6 choral halls, etc..., are expected to make the airborne spread of the virus an important consideration.

7  
8 Assessment of the ventilation provision or, where practical, the monitoring of CO<sub>2</sub> levels to indicate  
9 ventilation provision (§3), can help manage the risk of COVID-19 transmission via the airborne route in  
10 winter 2020. Most documented cases of transmission which are believed to have arisen from the airborne  
11 route have been in environments where the outdoor air supply would not have complied with current UK  
12 design guidance. It is inferred from this, and the documented modelling, that provision of outdoor air in-  
13 line with existing design guidance will help reduce the risk of transmission by the airborne route. The rate  
14 of provision of outdoor air can be inferred by monitoring CO<sub>2</sub> levels in occupied spaces, maintaining these  
15 below about 1 000 ppm being indicative of adequate ventilation in many indoor environments, including  
16 offices (with design guidance for some indoor spaces permitting 1 500 ppm see UK Government, 2018*a*,  
17 and §3.2 for a fuller discussion). However, higher ventilation rates may be needed wherever activity levels  
18 increase beyond desk-based work. Risks can also be reduced by reducing occupancy (whilst ensuring  
19 full outdoor air supply rates are maintained), staggering occupancy (via appropriate timetabling) and  
20 by the purging of indoor spaces between events (§2.4). Numerous additional engineering strategies are  
21 available to help further reduce the risk of transmission and we review these in detail (see §4).

22 The review of current knowledge has identified the following key research questions.

- 23 • What are the SARS-CoV-2 viral load distributions and respiratory droplet size distributions emitted  
24 by an individual carrying out activities such as sitting, walking, talking, singing, sneezing,  
25 coughing? How might these vary with an individual's size, age, etc...?
- 26 • What is the trajectory of droplets and aerosols containing viral particles from exhalation to re-  
27 moval under different ventilation modes, occupancy levels, occupant behaviour/movement, and  
28 environmental conditions (e.g. temperature, humidity, etc...)?
- 29 • How can risk and severity of infection of an individual be determined from the nature of viral  
30 exposure (i.e. how and where droplets or viral particles are deposited in the respiratory system of  
31 an individual, their frequency, the peak/cumulative dose, etc...)?
- 32 • What processes determine the timescales for purging a space or determining the frequency of  
33 cleaning and which environmental conditions affect these?
- 34 • What are the quantitative impacts of wearing face coverings?
- 35 • How effective are localised outdoor air supplies and/or purification methods, and what factors  
36 affect their results?
- 37 • How can existing knowledge, and perhaps answers to the above questions, be deployed to better  
38 understand and predict the spread of COVID-19, for which high-spreading statistical outlier events  
39 (so called 'superspreaders') appear to be significant?
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For Review Only



## 2 The indoor spread of COVID-19 and winter 2020

Florence Nightingale is credited with having promoted the idea that the indoor environment plays a critical role in determining health outcomes. Her pioneering work on hospital ward design is still highly relevant with the guiding principles of high ceilings, adequate natural lighting, and sufficient ventilation proving sound design for any indoor environment. However, modern architectural approaches, which often rely on mechanical means to condition the environment, may not follow these principles. In this document we focus on two indoor spaces, namely, open-plan offices and school classrooms, because these constitute spaces which are recurrently attended by the same group of people, are occupied for significant portions of each weekday, are frequently populated at moderate to high occupancy density and do not often adhere to Nightingale's design principles. In short, these settings contain very common spaces in which a significant proportion of the population may potentially be at moderate to high risk of exposure to the COVID-19 coronavirus. However, the findings and guidance reported within this document apply generically to almost all indoor spaces and we urge that the guiding principles be widely applied.

### 2.1 COVID-19 transmission indoors

The novel coronavirus disease (COVID-19), which causes respiratory and other symptoms, was declared a pandemic by the World Health Organization (WHO) in March 2020. Transmission of such respiratory infections occurs via virions encapsulated in particles of respiratory secretions (in this case the virus SARS-CoV-2) formed in the respiratory tract of an infected person and spread to other humans via three routes: the droplet route, the contact route and the airborne route (Mittal *et al.*, 2020).

The droplet route involves the transfer of respiratory droplets from an infected person to the mucous membranes of a subject, i.e. respiratory droplets land in the mouth, nose or eyes of others. The contact route takes place when respiratory droplets are deposited onto surfaces that are then touched by other people who go on to touch their mouth, nose or eyes before washing their hands. The airborne route (also called aerosol transmission) occurs when exhaled respiratory droplets are small enough to remain suspended in air such that they can be inhaled into the respiratory system of other people.

At the beginning of the COVID-19 pandemic, a lack of direct empirical evidence on airborne transmission of SARS-CoV-2 highly influenced health policy decisions which were intended to control the pandemic and the public response to it. However, an increasing body of evidence (particularly from poorly ventilated indoor environments), a better understanding of the disease progression, and information on the asymptomatic and pre-symptomatic transmission of the virus strongly support the case for airborne transmission of SARS-CoV-2 virus (see Morawska *et al.*, 2020; Morawska & Milton, 2020, for discussion and the references therein).

In an indoor environment the ventilating flow modulates the transport and advection of any aerosols (including bio-aerosols), pollutants, and CO<sub>2</sub> produced by indoor-sources/occupants and further determines their subsequent removal from within the indoor environment. Traditionally, building ventilation has been studied in the context of thermal comfort and in the last few decades energy efficiency. However, there has been a timely shift of focus and, in addition to energy efficiency and thermal comfort, indoor air quality (and implicitly the removal of any indoor airborne pollutants produced by the occupants) has become a core focus (Sloan Brittain *et al.*, 2020). Within our guidance we exploit this cutting-edge knowledge to offer advice to mitigate COVID-19 spread via the airborne route. However, it is essential not to do so at the expense of considering droplet transmission and contact transmission, and measures to minimise the risk of transmission via these routes must be given as much priority as consideration of the airborne route. As such, within this section we review transmission via the droplet and aerosols route (§2.1.1) and contact routes (§2.1.2), we then consider the role that social distancing (§2.2), face coverings (§2.3), and occupancy behaviour (§2.5) can play in affecting the various modes of transmission. In §3 we address the role of ventilation in influencing the airborne transmission route. We go on to discuss the suitability of other measures in mitigating the spread (§4) and we present three appendices covering: factors affecting, and modelling considerations for, surface transmission (appendix B), details of the potential for ultraviolet germicidal irradiation (UVGI) air disinfection to mitigate COVID-19 transmission (appendix C), and details of current governmental guidance for face coverings (appendix ??).



### 2.1.1 Transmission via droplets and aerosols

Respiratory diseases are transmitted by exposure to pathogen-laden droplets produced by expiratory events such as breathing, coughing, sneezing, speaking, singing and laughing (Stelzer-Braid *et al.*, 2009; Yan *et al.*, 2018). The expiratory droplets range between 0.01–1000  $\mu\text{m}$  (Bake *et al.*, 2019), and conventionally they are classified in two categories; droplets smaller than 5  $\mu\text{m}$  are referred to as droplet nuclei or aerosols, whereas droplets larger than 5–10  $\mu\text{m}$  in diameter are classified as respiratory droplets (World Health Organization, 2014; Milton, 2020). This somewhat arbitrary size classification implicitly refers to the transmission modes/mechanisms, namely droplet, and airborne transmission. However, the distinction between droplet transmission and airborne transmission determined by a simple cut-off in droplet size neglects a multitude of physical processes crucial to the droplet evolution within an indoor environment. For example, droplets that are larger than a selected cut-off size at the source may shrink due to evaporation, becoming sufficiently small (before any impact occurs) that they then contribute to airborne transmission. The distinction between droplet transmission and airborne transmission is better explained by the route of infection. Droplet transmission occurs when a subject is exposed to large ( $> 5 - 10 \mu\text{m}$ ) pathogen-laden droplets expelled by an infected person that come into contact with their mucous membranes. Droplet transmission usually occurs in close proximity (see §2.2). While droplets may fall quickly onto a surface close to the source, aerosols are expected to remain airborne for longer periods and can be advected away from the source with ventilation flows leading to what we term ‘the airborne transmission route’. Therefore, an important aspect of understanding droplet and airborne transmission is the size distribution of the expiratory droplets containing the virus also include water, salts and organic material (Kumar & Morawska, 2019). Droplets and aerosols produced by violent expiratory events such as coughing and sneezing have been investigated and reviewed by several authors, including Yang *et al.* (2007); Bourouiba *et al.* (2014), Bourouiba (2020) and Mittal *et al.* (2020). However, under normal circumstances, the cumulative amount of expiratory fluid and consequently the droplets and aerosols produced by low-frequency intermittent events such as coughing and sneezing are less than that of high-frequency events such as breathing and talking (Gupta *et al.*, 2010).

The studies conducted on disease progression suggest that infectivity of COVID-19 peaks before the onset of symptoms and consequently, preventing pre-symptomatic and asymptomatic transmission is key to containing the spread of the disease (Matheson & Lehner, 2020). At the early stage of SARS-CoV-2 infection, upper respiratory tract symptoms and the presence of high concentrations of SARS-CoV-2 virus in oral fluids are common (Wölfel *et al.*, 2020) which support the recent findings identifying speech droplets to be a potential cause of transmission (Stelzer-Braid *et al.*, 2009; Anfinrud *et al.*, 2020; Stadnytskyi *et al.*, 2020).

Conversational speech produces a wide range of droplet sizes (sub-micron up to the order of 100  $\mu\text{m}$ ) which are exhaled at speeds of of the order of  $3.5 - 4 \text{m s}^{-1}$ . The reported size distributions of speech droplets show a large variation, due to different measurement techniques, evaporation of droplets prior to measurement, and natural variation amongst different people (Xie *et al.*, 2009). Aerosol measurements capable of measuring particles in the range 0.5–20  $\mu\text{m}$  indicate that speech droplets form across the measurement range, with geometric mean diameter of  $\sim 1 \mu\text{m}$ , droplet number concentrations in exhaled breath of the order of  $0.1 - 1 \text{cm}^{-3}$ , and exhaled particle emissions rates of the order of  $1 - 10 \text{s}^{-1}$  (Asadi *et al.*, 2019; Johnson *et al.*, 2011). Asadi *et al.* (2019) showed that speaking louder is correlated with higher particle emissions and found that a small fraction of people are ‘super-emitters’, who consistently release an order of magnitude more particles than others. Chao *et al.* (2009) measured the droplet size distribution of cough and speech droplets at mouth opening and found the geometric mean diameter of cough droplets was 13.5  $\mu\text{m}$ . In contrast, speech droplets were 16  $\mu\text{m}$ , but had a reported maximum diameter of up to 1000  $\mu\text{m}$ . Xie *et al.* (2009) reported the average speech droplet diameter to be between 50–100  $\mu\text{m}$ . Interestingly, this study also showed that both the number and the droplet size increased significantly when the subjects swallowed food dye solution (with or without sugar) before the experiment, indicating that eating may promote the release of higher numbers and larger sizes of expiratory droplets. Although light scattering measures only larger droplets and consequently provides a conservative estimate of total droplet count, Stadnytskyi *et al.* (2020) measured high droplet release rates relative to other studies when using this technique. Both Anfinrud *et al.* (2020) and Stadnytskyi *et al.* (2020) showed that speech droplets of size 10–100  $\mu\text{m}$  can remain suspended for up to 30 s. Therefore, it is imperative to appreciate that speech droplets can potentially transmit respiratory diseases by both the droplet and airborne transmission routes. In contrast, it has consistently been shown that the majority of aerosol particles in exhaled breath are  $< 5 \mu\text{m}$  (Fennelly, 2020), which diminishes the possibility of droplet transmission when breathing; however, airborne transmission cannot be ruled out.

1  
2  
3 When droplets are exhaled, they evaporate at a rate that depends on droplet size and composition,  
4 and the relative humidity and temperature of the air. Redrow *et al.* (2011) compared the evaporation  
5 time and resulting nuclei sizes of model sputum, saline solution, and water droplets. They showed  
6 that sputum droplets containing protein, lipid, carbohydrate, salt and water leave larger nuclei than  
7 salt solution. They calculated the time scales of evaporation of water droplets at room temperature,  
8 for relative humidities between 0 to 80%, to be 0.1 – 1 s for droplets less than 10  $\mu\text{m}$  and 7 – 40 s for  
9 100  $\mu\text{m}$  droplets. Therefore, it is expected that droplets larger than 100  $\mu\text{m}$  settle on the floor or other  
10 nearby surfaces (Liu *et al.*, 2017), while droplets smaller than about 10  $\mu\text{m}$  tend to form nuclei and are  
11 transported as passive scalars, i.e. they were transported by airflows without the dynamics of the airflow  
12 being significantly affected (Xie *et al.*, 2007).

13  
14 The final size of exhalation droplets depends upon many factors including the initial size, non-volatile  
15 content, relative humidity, temperature, ventilation flow, and the residence time of the droplet. Marr  
16 *et al.* (2019) gave the equilibrium size for 10  $\mu\text{m}$  sized model respiratory droplets containing 9  $\text{mg ml}^{-1}$   
17 NaCl, 3  $\text{mg ml}^{-1}$  protein, and 0.5  $\text{mg ml}^{-1}$  surfactant to be 2.8  $\mu\text{m}$  and 1.9  $\mu\text{m}$  at relative humidities of  
18 90% and < 64%, respectively.

19 A significant uncertainty in our ability to quantify the relative importance of airborne transmission is  
20 the viral load associated with different aerosol sizes for different expiratory events, at different stages of  
21 infection and the potential for natural variation amongst people. This information is currently unknown,  
22 leading to large uncertainty bounds.

### 2.1.2 Transmission via surface contacts and fomites

23  
24 Fomites are inanimate objects that have become carriers of virus particles and these have been shown to  
25 play a role in the spread of viruses. Little is known of the true risk of becoming infected by SARS-CoV-2  
26 through this pathway as it is difficult to isolate from the droplet and airborne transmission routes, the  
27 dose required to become infected has not been determined and the majority of studies that have looked  
28 at its survival on surfaces have used far greater viral loads than would be deposited naturally (Vasickova  
29 *et al.*, 2010). Knowledge of SARS-CoV-2 survival on surfaces and interactions between the fomite and  
30 droplet/airborne routes through deposition and re-suspension of viral particles is nevertheless important  
31 in minimising the risk of infection.

32  
33 Fomite transmission occurs predominantly from human behaviour through non-infected individuals mak-  
34 ing contact with or handling infected objects, which may be infected via deposition of large droplets from  
35 infected individuals or via direct contact by individuals with viral particles on their hands. The non-  
36 infected individual then transfers viral particles from their hands to mucous membranes by touching  
37 their eyes, nose or mouth. Advice given by various groups to avoid unnecessary contact between hands  
38 and objects in public environments, as well as advice to avoid touching one's face, is sound and should be  
39 encouraged. In China it was found that the majority of surfaces within hospital wards that had infected  
40 individuals, were found to have traces of the virus (Ye *et al.*, 2020), and therefore cleaning of surfaces is  
41 an important mitigation strategy to avoid infection.

42  
43 This section briefly summarises what is known about SARS-CoV-2 and its survival and transmission  
44 via surfaces. Note that a more thorough and detailed literature review is found in Appendix B of this  
45 document, which details the sources upon which this advice is based.

46  
47 **Factors affecting the survival of SARS-CoV-2 on surfaces.** A number of environmental factors  
48 affect the survival of SARS-CoV-2 on surfaces.

- 49 • **Temperature** effects have been reported to be significant, with higher temperatures decreasing  
50 survival times (Dietz *et al.*, 2020). Comfortable indoor temperatures should be maintained and  
51 the use of air conditioning should be minimised wherever practical with the appropriate supply of  
52 outdoor air remaining a priority.
- 53 • **Humidity** has been shown to also have an effect on the virus, with drier conditions being more  
54 suitable for virus survival (Biryukov *et al.*, 2020). While higher humidity is preferable to reduce  
55 viral infection, there are numerous health issues related to high humidity and promotion of mould  
56 growth. We therefore advise that in cold weather the relative humidity should be maintained at  
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58  
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3 between 40–50 %, rather than below 30 %, which is typical of many indoor environments in winter  
4 (Dietz *et al.*, 2020).  
5

- 6 • **Light** is also demonstrated as an effective method for SARS-CoV-2 deactivation with 90% of the  
7 virus inactivated every 6.8 to 14.3 minutes depending on the intensity of simulated natural light  
8 (Schuit *et al.*, 2020). UV-C light has been shown to deactivate other strains of coronavirus Bedell  
9 *et al.* (2016). While the use of artificial light cleaning technologies is not suggested as a replacement  
10 for disinfectant cleaning practices, well-lit rooms, particularly via natural lighting is preferred based  
11 on evidence from SARS-CoV-2 and other viruses (Dietz *et al.*, 2020; Schuit *et al.*, 2020).  
12
- 13 • It is now well-known that SARS-CoV-2 has different survival times on different surfaces, with  
14 laboratory inoculations of SARS-CoV-2 survival rates varying from 3 hours for paper and tissue  
15 to up to 72 hours (3 days) on hard, smooth surfaces such as plastic and stainless steel (and also  
16 on surgical masks) (van Doremalen *et al.*, 2020). Glass and bank notes have survival times in  
17 the region of 3 days, with cloth and wood reported at 2 days. More recent results suggest even  
18 longer survival times such as at least 28 days at 20°C and 50% relative humidity when dried onto  
19 non-porous surfaces at a starting viral load typically excreted by infected patients (Riddell *et al.*,  
20 2020). While likely viral loads on contaminated objects are highly uncertain, and hence the risk  
21 associated with the fomite pathway relative to that for droplet and aerosol transmission cannot  
22 currently be quantified, we believe it is important to regularly clean often-handled objects and  
23 surfaces in public spaces.

24 **Cleaning recommendations.** The above leads to the recommendation that it is important to fre-  
25 quently clean high-touch surfaces such as door handles, classroom and meeting room desks, tap handles,  
26 swing door handles, ticket machines, pin code keypads, communal office kitchens, etc. Providing point-  
27 of-contact public alcohol-based disinfectant, as is now common on university campuses, shops and public  
28 transport, is an effective mitigation strategy to ensure that public spaces are less likely to become con-  
29 taminated. We conjecture that an effective mitigation strategy for certain public spaces that involve  
30 fomites, such as public computer laboratories found in universities or libraries, would be to encourage  
31 users to clean the workspace (keyboards, mouse, desk and hands) before and after use. Further research  
32 is needed to establish the efficacy of such interventions depending on the frequency of cleaning and in  
33 comparison (or in combination) with other mitigation approaches such as handwashing. The wearing of  
34 face coverings will also reduce droplet deposition on the workspace and should therefore be encouraged.

35 Disinfectants based on alcohols (ethanol, 2-propanol, 2-propanol with 1-propanol) as well as other com-  
36 mon disinfectants (glutaraldehyde, formaldehyde, and povidone iodine (0.23%–7.5%)) are very effective  
37 against SARS-CoV-2 and come highly recommended (Kampf *et al.*, 2020). UK Government advice to use  
38 soap and water to clean surfaces may not be the most effective since soap and water alone was not shown  
39 to deactivate the virus after 5 minutes, however if accompanied by scrubbing this may be more effective  
40 at its physical removal (Kampf *et al.*, 2020). Sodium hypochlorite requires a concentration of at least  
41 0.2%, whilst hydrogen peroxide requires a concentration of at least 0.5% and must be left incubating for  
42 at least 1 minute.  
43

## 44 2.2 Social distancing indoors

45 Social distancing describes the effort to ensure that individuals remain separated by a particular distance  
46 and is often recommended primarily to reduce the transmission of disease via the droplet route. The  
47 quantification of an appropriate social separation/distance to avoid droplet transmission is often based on  
48 research by Wells (1934). His model for disease transmission considered droplets produced by sneezing or  
49 coughing to behave ballistically, with no interaction between them; i.e. droplets would fall from the height  
50 they were produced to the floor ( $\sim 2$  m vertically), while simultaneously evaporating. In spite of the  
51 evaporation process, so called ‘large’ droplets would reach the floor; meanwhile so-called ‘small’ droplets  
52 would evaporate quickly leaving relatively ‘dry’ aerosol particles known as droplet nuclei. Wells proposed  
53 two mechanisms for infection: droplet transmission due to large droplets and airborne transmission due to  
54 small droplets that evaporate sufficiently to become suspended in the air for long times. Wells calculated  
55 a cut-off between small and large droplets as  $100 \mu\text{m}$  (not  $5 \mu\text{m}$ , as is often cited).  
56

57 Wells’ falling-evaporation curve has been used to propose a social distancing rule by considering how far  
58 large droplets travel horizontally as they fall. The total distance travelled by a droplet is determined by  
59 its initial horizontal velocity, and also whether it is contained in a coherent flow structure caused, for  
60 example, by coughing or sneezing (Bourouiba, 2020). Small droplets fall more slowly than large droplets,

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3 so travel further, but they take less time to evaporate. The size of the largest droplet that totally  
4 evaporates before falling 2 m is identified, then the horizontal distance this droplet travels is calculated  
5 and used to define a social distancing rule. For example, for droplets expelled at 10 m/s (typical for  
6 coughing) this distance is 2 m, while for droplets expelled at 1 m/s (breathing) this distance is less than  
7 1 m (Xie *et al.*, 2007).  
8

9 In addition to the work of Wells, the experiments by Jennison (1942) have also been used as evidence  
10 for social distancing of 1-2 m. Jennison used high-speed photography to examine the fall of droplets  
11 produced by talking, coughing and sneezing, concluding that the majority of the droplets fell to the  
12 ground within 1 m (the field of view for the experiments). However, no details were provided about how  
13 this conclusion was reached and it was acknowledged that the experimental method was not sensitive  
14 enough to capture all the droplets, tending to select for larger droplets (Bahl *et al.*, 2020).

15 The Wells model and many of its extensions assume that droplets behave independently of each other,  
16 travelling ballistically. However, more recent research has shown that this is often not a good assumption.  
17 Experimental studies of coughing and sneezing show that exhalation results in a puff of warm, humid  
18 air that influences the distance travelled by groups of droplets (Bahl *et al.*, 2020). Experimental images  
19 show that the turbulent gas cloud emitted from a human sneeze can travel 8 m, carrying particles along  
20 with it (Bourouiba, 2020). These results suggest that for coughing and sneezing, the exhalation or puff  
21 needs to be taken into account in calculating the maximum distance travelled by droplets. Moreover,  
22 consideration of these images highlights that violent respiratory events have significant directionality and  
23 in contexts where the direction of these events can be inferred then this should influence the layout of  
24 desks, etc., within classrooms and offices.

25 Current social distancing advice aims to reduce droplet transmission, however social distancing can  
26 also reduce transmission by small droplets, as aerosols are diluted with distance from the source (Chen  
27 *et al.*, 2020). The suggested distance of 2 m is based on a model that assumes ballistic droplets, rather  
28 than particles that travel within an exhaled puff that dilutes with distance. Further research is needed to  
29 identify an appropriate distance at which sufficient dilution has occurred. This distance will be influenced  
30 many factors, including the nature of the exhalation (breathing, talking, coughing, etc.), the properties  
31 of the air (humidity, temperature), the droplet size distribution, the virus concentration with droplet  
32 size, and the size of the infectious dose.  
33

34 There is increasing evidence that airborne transmission of SARS-CoV-2 is significant (e.g. Morawska  
35 *et al.*, 2020; Fennelly, 2020). Outdoors, in well-ventilated indoor spaces, and for short interaction times,  
36 social distancing will reduce airborne transmission. However, for longer exposure times and/or in poorly  
37 ventilated spaces, social distancing is unlikely to be sufficient as the dilution at room scale will not  
38 reduce the aerosol concentration enough to avoid an infectious dose. This emphasises the importance of  
39 additional measures, such as good ventilation and face coverings.  
40

### 41 **2.3 Guidance on face masks and coverings**

42 There is substantial evidence that face masks and coverings can lessen the spread of COVID-19 by  
43 reducing the emissions of virus-laden particles, both droplets and aerosols (Leung *et al.*, 2020) from  
44 the wearer. Moreover, it has also been shown that face masks and coverings may also be protective  
45 by reducing the dose of SARS-CoV-2 received by the wearer. Face shields might prevent the escape of  
46 droplets from the user, as well as similarly protecting the user and providing some limited protection to  
47 the eyes, but they are unlikely to stop the transmission of aerosols (the airborne route) unless used in  
48 conjunction with a face mask or covering. The collated evidence presented within this section has been  
49 gathered from studies across the world and represents the most recent understanding of the subject, as  
50 presented by a cross-section of peer-reviewed papers and scholarly works.  
51

52 In short, the use of face masks and coverings can reduce the spread of COVID-19 indoors; this is especially  
53 pertinent in settings where there is decreased physical distancing: such as shops, public transport or in  
54 work environments. However, the impact of wearing face masks or coverings on the intended interactions  
55 and activities should be carefully considered with members of disadvantaged or vulnerable groups given  
56 due attention.  
57  
58  
59  
60



### 2.3.1 A summary of the evidence base for the use of face masks and coverings

A systematic review and meta-analysis of distancing, face masks and eye wear in Lancet from June 2020 concluded that “face mask use could result in a large reduction in risk of infection, with stronger associations with N95 or similar respirators compared with disposable surgical masks or similar (Chu *et al.*, 2020)”. They also noted that “transmission of viruses was lower with physical distancing of 1 m or more” and “eye protection also was associated with less infection”. These reviews were in both health-care and non health-care settings. A more recent meta-analysis from August presented significant results, “that face masks protect populations from infections and do not pose a significant risk to users” (Ollila *et al.*, 2020).

Masks often refer to surgical or respiratory masks (respirators) that medical staff use, whereas face coverings encompass broader types and materials such as homemade cloth masks, but may include just a simple scarf (The Royal Society, 2020).

Leung *et al.* (2020) “Demonstrated the efficacy of surgical masks to reduce coronavirus detection and viral copies in large respiratory droplets and in aerosols”. Their results suggest that these masks could prevent transmission of viruses from symptomatic (and for COVID-19 pre-symptomatic/asymptomatic) individuals.

There is evidence that face masks and coverings may be effective at reducing COVID-19 cases across the world. Mitze *et al.* (2020) stated “after face masks were introduced on 6 April 2020, the number of new infections fell almost to zero” in the city of Jena, Germany; concluding “that the daily growth rate of COVID-19 cases in the synthetic control group falls by around 40 percent due to mandatory mask-wearing relative to the control group”. Similarly, “from epidemiological data, places that have been most effective in reducing the spread of COVID-19 have implemented universal masking, including Taiwan, Japan, Hong Kong, Singapore, and South Korea” (Prather *et al.*, 2020).

“Our analysis reveals that the difference with and without mandated face covering represents the determinant in shaping the trends of the pandemic worldwide. Zhang *et al.* (2020) conclude that wearing of face masks (or coverings) in public corresponds to the most effective means to prevent inter-human transmission, and this inexpensive practice, in conjunction with extensive testing, quarantine, and contact tracking, poses the most probable fighting opportunity to stop the COVID-19 pandemic, prior to the development of a vaccine.”

“The benefits face masks could offer as a non pharmaceutical intervention were investigated using mathematical models and show that face mask use by the public could make a major contribution to reducing the impact of the COVID-19 pandemic” Stutt *et al.* (2020). They demonstrated that mask (and face covering) wearing can reduce transmission, with high rates of adoption likely to reduce the  $R_0$  value to below one.

As of the 5<sup>th</sup> June 2020 the World Health Organization (2020a) has recommended the wearing of face masks and coverings for communities and circumstances, where there is risk of transmission and in areas where physical distancing cannot be achieved, for the “general public should be encouraged to use medical and non-medical masks in areas with known or suspected community transmission” of COVID-19 virus. More recently (in August 2020) the WHO have released a video to advise the wearing of face masks or coverings.

The Royal Society DELVE Initiative (2020) DELVE Initiative reported that “asymptomatic (including pre-symptomatic) infected individuals are infectious” and “respiratory droplets from infected individuals are a major mode of transmission”. Reporting that masks reduced droplet dispersal and “cloth-based face masks reduce emission of particles by variable amounts”, similar to the percentage reductions reported (of viral, bacterial and dust particles) by surgical masks. In a more recent study, key findings were that cloth face coverings are effective in protecting the wearer and those around them and that face masks and coverings are part of ‘policy packages’ that need to be seen together with other measures such as social distancing and hand hygiene (The Royal Society, 2020).

The physics of particle capture by the materials of face masks is complex, it also recognised these mechanisms will be at play in face coverings. However, coverings (and face masks), as well as reducing the airflow which can distribute the virus, may simply prevent transmission by stopping the evaporation of droplets their escape to become droplet nuclei (Leung *et al.*, 2020).

Face masks and coverings may also protect the wearer, to different degrees of effectiveness. The material

1  
2  
3 and the make-up of the face mask or covering is important for the filtration efficacy. Wilson *et al.* (2020a)  
4 modelled the risk of transmission based on published data on the effectiveness of various material against  
5 a viral challenge; these included FFP2 (N95) respirator material (at 95% efficiency), with surgical mask  
6 material slightly less efficient. A vacuum cleaner bag (83%) was found to be the most efficient household  
7 material and a scarf (44%) the least efficient. In between these two materials were a tea towel, cotton  
8 mix, linen, a pillowcase, silk and 100% cotton T-shirt.

9  
10 User protection is discussed by Gandhi *et al.* (2020) who hypothesise that “universal masking reduces  
11 the ‘inoculum’ or dose of the virus for the mask-wearer, leading to more mild and asymptomatic infec-  
12 tion manifestations.” They state numerous cases where universal masking led to fewer cases, or more  
13 asymptomatic cases as opposed to comparative examples, whether this was in animals, on cruise ships,  
14 meat factories, or regions of universal mask wearing. “Countries accustomed to masking since the 2003  
15 SARS-CoV pandemic, including Japan, Hong Kong, Taiwan, Thailand, South Korea, and Singapore, and  
16 those who newly embraced masking early on in the COVID-19 pandemic, such as the Czech Republic,  
17 have fared well in terms of rates of severe illness and death.”

18  
19 The use of face shields as an alternative to face masks and coverings within the service industry in the  
20 UK is popular, it is also of great benefit to those of hard of hearing (HoH). “Face shields can substantially  
21 reduce the short-term exposure of health care workers to large infectious aerosol particles, but smaller  
22 particles can remain airborne longer and flow around the face shield more easily to be inhaled.” Lindsley  
23 *et al.* (2014). Thus their use might prevent the escape of droplets from the user, as well as similarly  
24 protecting the user and providing some limited protection to the eyes. They are however, unlikely to stop  
25 the transmission of aerosols (the airborne route), without being used in conjunction with a face mask or  
26 covering. Verma *et al.* (2020) visualised the flow around a face shield (and respirators with valves) and  
27 noted “that although face shields block the initial forward motion of the jet, the expelled droplets can  
28 move around the visor with relative ease and spread out over a large area.” Similar results were found  
29 for respirators with exhale valves, with aerosols escaping through the valve.

30  
31 Any covering of the face negatively impacts the hard of hearing (HoH), with close to 11 million people in  
32 the UK who are HoH (around 1 in 6 people). Where possible, it would be prudent to adapt the guidance  
33 on face masks to accommodate them, as the usage of face masks may hinder the ability to listen and  
34 lip-read. An effective strategy to accommodate those HoH is to use clear face masks (Grote & Izagaren,  
35 2020), or the use of novel technologies such as captioning apps.

36  
37 Finally, the application of the precautionary principle suggests that people should be encouraged to  
38 wear face masks and coverings on the grounds that, in many circumstances, we have little to lose and  
39 potentially something to gain from this measure. Greenhalgh *et al.* (2020) said that “masks are simple,  
40 cheap, and potentially effective . . . and outside the home in situations where meeting others is likely  
41 (for example, shopping, public transport), they could have a substantial impact on transmission with a  
42 relatively small impact on social and economic life.”

## 43 2.4 Source reduction through timetabling and purging between events

44  
45 Airborne infection risk is reduced when the ventilation provision of outdoor air is maximised. Operating  
46 the existing indoor environment conditioning and controlling equipment in a manner that fixes the  
47 outdoor air supply rate to be maximal (with due consideration to the practical limits for a comfortable  
48 indoor environment), the airborne risk can be greatly reduced by lowering occupancy in a given indoor  
49 space. For example, should the ventilation plant be kept running at the same level (i.e. unchanged  
50 absolute outdoor air supply rate) and the occupancy halved (e.g. through week in – week out working)  
51 in an indoor space then the chances that infection occurs within is approximately halved.

52  
53 Where reductions to the absolute levels of occupancy are impractical then some change of timetabling  
54 should be considered. Where possible, attendance should be extended by some occupants arriving  
55 and leaving earlier than usual with other occupants arriving and leaving later than usual. Moreover,  
56 consideration should be given to purging rooms between meetings, classes and events. This would require  
57 the room to be unoccupied between consecutive events during which period all possible efforts are made  
58 to increase the outdoor air supply rate (whether by opening windows, doors and ventilation systems).  
59 At the end of the purging period it is best if the room is cleaned in the manner detailed in §2.1.2  
60 to minimise the chance of spread to the next occupants. It is highly likely that the greatest rates of  
decay in the concentration of virus-laden particles will occur at the start of the purging periods. So  
any purging duration is better than none as long as the increased ventilation flows are given time to

1  
2  
3 establish themselves. That said, the longer the purging duration then the lower will be the resulting  
4 concentration levels. To establish how effective these purging strategies may be, a simple ‘room-change’  
5 time scale can be calculated by dividing the volume of the space by an estimate of the rate of outdoor  
6 air supply during purging; note that the room-change time scale, in hours, is simply the inverse of the  
7 air changes per hour (AC/hr). Melikov *et al.* (2020) report the intake fraction (the proportion of air  
8 exhaled by the infected person that is then inhaled by another occupant) for various purge scenarios.  
9 They quantified the reduction in intake fraction for ventilated cases with purging periods (of between  
10 15 min and 30 min) which were in the range 0.2–1.8 room-change time scales. They went to consider cases  
11 of increased ventilation rates which led to purging times (15 min) being around 5.5 room-change time  
12 scales. Their conclusions included the suggestion that periods of constant occupation should be short  
13 with appropriately long breaks being recommended; break durations of 10–20 min were recommended  
14 for occupancy durations of 30–45 min for classrooms, meeting rooms, conference rooms, etc....

15  
16 In summary, wherever possible occupancy should be reduced (by remote working and/or reduced occu-  
17 pancy), additionally unoccupied periods should be introduced at regular intervals throughout the day  
18 during which the space should be purged and after which the space should be cleaned. Risk-based  
19 calculations are needed to determine the optimal purging times.

## 20 21 **2.5 The influence of occupancy behaviour on indoor air movement: impli-** 22 **cations for the spread of COVID-19**

23  
24 The movement of people within enclosed spaces leads to considerable disturbance of the air and any  
25 airborne aerosols. Although aerosols greater than about 10 micron might, in some settings, settle from  
26 the space relatively rapidly and so they may be dispersed by people movement, smaller aerosols (e.g.  
27  $< 5 \mu\text{m}$ ) which can remain airborne for several 10’s of minutes may be much more strongly affected  
28 by such dispersal. This dispersal may in fact dominate other dispersal processes if there is a sufficient  
29 frequency of people passing through a space. People have widths typically in the range 0.3 – 0.5 m, and  
30 move at speeds of 1 – 2 m/s even when walking, and this leads to a highly turbulent wake with Reynolds  
31 number of about 100,000. The mixing associated with this wake in corridors, supermarket aisles, meeting  
32 rooms, school classrooms or other spaces with relatively high people density and movement (even with  
33 2 m spacing) may be key for quantifying the aerosol dispersion prior to it being ventilated or settling out  
34 from the space. Indeed, with ventilation timescales of 10 – 20 minutes in typical buildings, with 5 – 10  
35 air changes per hour, and the settling time of small aerosols ( $< 10 \mu\text{m}$ ) being of comparable duration, the  
36 aerosols may be mixed by the wakes of many people. For example, in a supermarket, with one person  
37 moving down an aisle every 10 – 30 s, a cloud of infected aerosol may be mixed by the wakes of between  
38 20 – 120 people. This mixing leads to a more uniform, but smaller concentration of aerosol in space,  
39 thereby increasing the risk of exposure to some aerosol for the subsequent people that pass by the aisle,  
40 although the amount of aerosol may be smaller; the associated risk of infection from any virus in these  
41 aerosols depends on dose and hence the amount of aerosol to which they are exposed (Bhamidipati &  
42 Woods, 2020).

43  
44 Laboratory simulations of the dispersal of both clouds of dye and suspended particles have been carried  
45 out in a fluid-filled channel of size 1.04 m  $\times$  0.10 m  $\times$  0.20 m, as a model of a corridor. To model the  
46 movement of people, cylinders of radius 0.015 – 0.050 m were moved back and forth along the channel,  
47 with speeds of 0.1 – 0.2 m/s, thereby providing a dynamically similar flow regime for the full-scale flow  
48 of people’s wakes. This models the mixing of the dye or cloud of particles along the channel. Data  
49 shows that the motion of the cylinder leads to an effective dispersion coefficient for the along-channel  
50 mixing, which provides the basis for a theoretical model (Mingotti *et al.*, 2020b). In experiments with  
51 a background ventilation along the corridor, in addition to the people-driven mixing, a dilution wave  
52 migrates along the corridor after the release of infected aerosol along the corridor, but the dispersion  
53 associated with people movement causes the aerosol to mix back upstream into the dilution wave, delaying  
54 the effectiveness of the ventilation (Mingotti & Woods, 2020). In figure 1(a, b), images at successive  
55 times from an experiment illustrate the dispersal of a cloud of dye along the channel. In figure 1(c), the  
56 concentration data from a number of experiments collapse to a simple model for the dispersion.

57  
58 Scaling up to a building, we find that the typical mixing rates associated with people in a corri-  
59 dor/supermarket aisle depend on the frequency of passage of people. With a person walking along  
60 the corridor/aisle every 10 – 40 s (Choudhary *et al.*, 2010; de Wijk *et al.*, 2018), this corresponds to  
a dispersion coefficient in the range 0.05 – 0.2 m<sup>2</sup>/s (Mingotti *et al.*, 2020b), so that over a period of  
600 – 1200 s, airborne aerosol may spread about 5 – 15 m along the corridor.



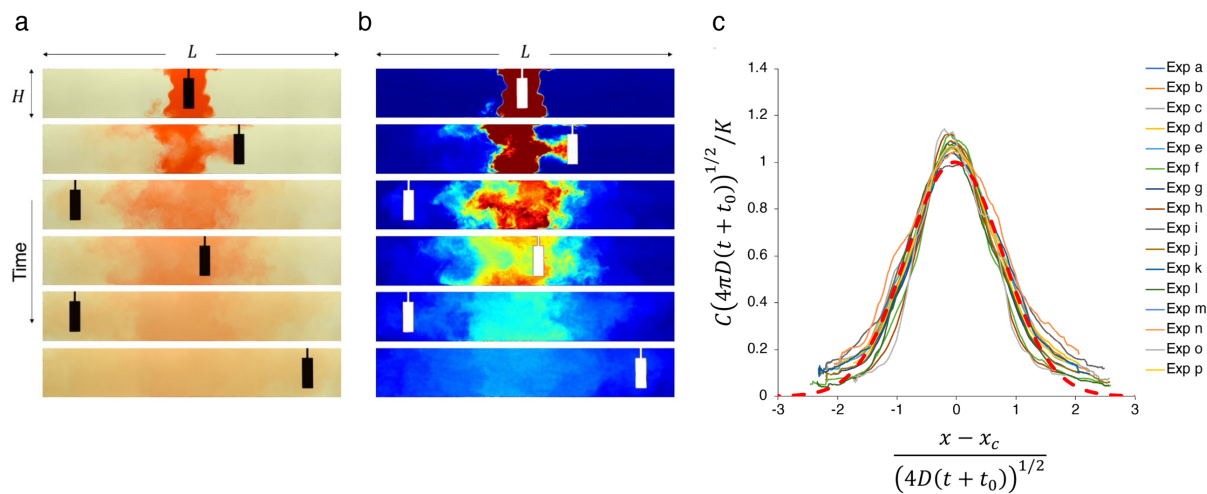


Figure 1: (a, b) Images show the depth-averaged concentration of a cloud of dye in a laboratory tank. This evolves in time owing to the mixing produced by the repeated motion of a cylinder, representing movement of people in a corridor. In (a), pictures of the tank are shown as captured during an experiment. In (b), false colours are used to represent the dye concentration field in each of these pictures, with red being the maximum concentration and blue showing absence of dye. The white rectangle on each image in (b) illustrates the position of the cylinder at that time. (c) Collapse of the experimental data of the depth-averaged dye concentration to a continuum model of the concentration of the dye along the channel. The  $y$  axis shows the dimensionless concentration, at each time scaled relative to the theoretical value in the centre of the channel at that time, and the  $x$  axis shows the dimensionless distance along the channel, scaled with the predicted diffusive spreading along the channel at each time (after Mingotti *et al.*, 2020*b*).

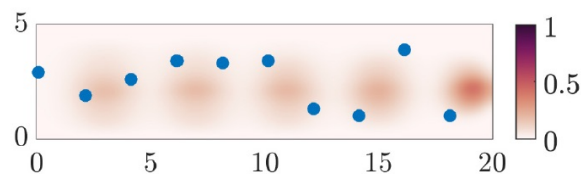


Figure 2: Image showing the mixing of individual clouds of infected aerosol, dyed different shades of red, and normalised relative to the initial concentration as seen on the legend. The corridor is 5 m wide ( $y$  axis) and 20 m long ( $x$  axis), with people (blue dots) moving from left to right along the corridor. In this simulation, the along-corridor people spacing is 2 m and they move with speed 1.5 m/s, while the clouds of aerosol are produced by one person moving down the corridor, so the older cloud at the left hand end of the corridor is more dispersed than that on the right (after Bhamidipati & Woods (2020)).

1  
2  
3 Bhamidipati & Woods (2020) developed a simple computational model for the dispersal of aerosols in  
4 a building by a stream of individual people moving through a building, modelling the detailed mixing  
5 produced by each person. This has led to a series of simulations of the dispersal of clouds of aerosol as  
6 people pass along a corridor. Figure 2 shows the concentration of aerosol with time, over a period of 15 s  
7 as an infected person walks down a corridor breathing out. A series of local clouds of aerosol-laden air  
8 are breathed out by the infected person, and these are then dispersed owing to the mixing produced by  
9 the continuing stream of people following in the wake of the infected person.

10 Models of different building types are under continued development, but the key result is the efficacy  
11 of mixing produced by the movement of people, which can lead to widespread dispersal of small aerosol  
12 produced by an infected person. This has leading order implications for the occupancy levels in shops  
13 and in the corridors of classrooms and offices in terms of the risks of exposure to the small aerosols due  
14 to the repeated passage of people. The combination of aerosol settling and ventilation of the air from the  
15 space typically leads to a residence time of the small aerosols of several tens of minutes; if these small  
16 aerosols are present in sufficient numbers to play a role in infection transmission, which depends on the  
17 source strength (i.e. the number of infectious people present), and the residence time (i.e. as regulated  
18 by the ventilation rate), then the continued presence of the infectious and healthy people may provide a  
19 pathway for transmission.

20  
21 Further experiments have been carried out in several operational buildings in which localised dilute  
22 clouds of CO<sub>2</sub> are released at a point in a room or corridor, and the subsequent spreading of this cloud  
23 is then measured over time; the CO<sub>2</sub> acts as an analogue for the small aerosols in that it moves with  
24 the air flow in the space (Mingotti *et al.*, 2020a; Woods *et al.*, 2020). Comparisons have been made  
25 between the rate of dilution and flushing of the CO<sub>2</sub> from the space in the case with people moving and  
26 with no people moving in the space. In the example of a ventilated corridor, for example, the impact  
27 of the people moving along the corridor is to drive additional mixing of the cloud of CO<sub>2</sub> into the new  
28 ventilation air, thus delaying the flushing of the CO<sub>2</sub> from the space. This illustrates how the internal  
29 mixing processes of the air in buildings, and especially people movement, can lead to greater residence  
30 times for small aerosols (Mingotti *et al.*, 2020a; Woods *et al.*, 2020). To mitigate the risks of these small  
31 aerosols, potential solutions include increasing ventilation rates, reducing the duration of exposure, and  
32 reducing the source of the aerosols by reducing occupancy levels and through the use of face masks.

### 34 35 **3 Ventilation and the airborne transmission route**

36 The adequate ventilation of a building space should be regarded as the primary mitigating measure  
37 against the spread of airborne diseases. In temperate climates this leads to the simple advice that all  
38 ventilation (by which we refer exclusively to the supply of outdoor, or suitably sterilised or filtered, air)  
39 systems be operated to maximise supply and ventilation openings (e.g. windows, vents, louvres, doors,  
40 etc.) be opened to the extent permitted by design. However, in colder periods (like the British heating  
41 season) there is a conflict between reducing airborne infection risk, by increasing the outdoor air supply,  
42 and maintaining occupants thermal comfort and reducing energy consumption and the associated costs.  
43 In this section we seek to provide guidance to resolve this conflict.

44  
45 In order to precisely quantify the risk of airborne infection occurring it is necessary to determine oc-  
46 cupants' exposure to airborne virus particles. To do so rigorously is challenging (often to the point of  
47 impracticality see §3.1) and thus it is wise to focus efforts on estimating the likelihood, perhaps better  
48 the change in likelihood, that airborne infection may occur within a space under a number of scenarios  
49 under the assumptions made.

#### 50 51 **3.1 Towards an understanding of ventilation and COVID-19**

52  
53 Ventilation, i.e. the supply of outdoor air, dilutes any pollutants produced indoors — including viable  
54 airborne virus particles. This dilution is dominated either: by the incoming outdoor air mixing with the  
55 existing indoor air, with then further mixing occurring as the air is transported through the building space  
56 before the ultimate evacuation outdoors; or by seeking to introduce the outdoor air into the occupied  
57 zone of the building space in a relatively unmixed state and then 'displace' any air polluted by airborne  
58 virus particles back outdoors (e.g. Bolster & Linden, 2008a,b; Mingotti & Woods, 2015a,b). Irrespective  
59 of the intended strategy, mixing will occur inhomogeneously within the building space which results in  
60 unpredictable distributions of virus particles. Moreover, differences in temperature between indoor and

1  
2  
3 outdoor air and the production of heat within the building space (every occupant outputting  $\sim 100$  W of  
4 heat) exacerbate the complexity of indoor air flows and increase the unpredictability of the distribution  
5 of airborne virus particles. Finally, in all indoor spaces (perhaps excluding COVID-19 hospital wards) it  
6 is unknown if there are any infectious occupants (the sources) and where they might be located. As such,  
7 trying to predict the distribution of viral contamination, for example using normal exhaust ventilation  
8 design techniques (which require knowledge of the source location and rate of contamination) is likely to  
9 be futile. Therefore estimating the risk of infection via modelled data typically requires the assumption  
10 that the distribution of airborne virus laden particles is relatively uniform irrespective of reality. We  
11 note that in the case that 'dead' or 'stagnant' zones can be expected or evidenced within an indoor space  
12 then further considerations are required see, for example §4 for further discussion.

13  
14 As such, the critical input pertaining to the control of the environmental quality for any model estimating  
15 airborne infection risk within an indoor space is the bulk supply of outdoor air, or ventilation rate per  
16 person which we denote  $Q$ . However, outdoor air may enter an indoor space via a ventilation system,  
17 windows, doors, vents, cracks in the building fabric or, indeed, though the very fabric itself (i.e. many  
18 building materials, e.g. bricks, are porous). As such, there is significant scope for both intentional  
19 and unintentional supply of outdoor air. Directly measuring the air flow through all of the potential  
20 pathways for any given indoor space is challenging. Pressure testing can be used to measure infiltration  
21 rates but cannot assess the ventilation rates in operational settings and does not help with individual  
22 spaces or zones. For purely mechanically ventilated, well-sealed, buildings it may be tempting to assume  
23 the intended outdoor air supply is actually achieved; however, such an assumption is not without risk.  
24 Measurements of the actual air flows could be made in the duct work but these are not without their  
25 own challenges (the velocity profiles of air flows in the these typically tortuous ducts is a challenging  
26 area of fluid mechanics in its own right).

27  
28 In most indoor environments the dominant source of  $\text{CO}_2$  is human activity and the level of  $\text{CO}_2$  in  
29 outdoor air remains broadly constant (at about 400 ppm). As such, it may be tempting to try to infer  
30 outdoor air supply (ventilation) rates by monitoring  $\text{CO}_2$ . However, whilst  $\text{CO}_2$  is an excellent proxy by  
31 which to determine indicative levels of ventilation, rigorously determining the precise (typically transient)  
32 outdoor air supply rate is non-trivial (e.g. see the appendices to Burrige *et al.*, 2020).

33  
34 Crucially, Rudnick & Milton (2003) established a methodology, based on the Wells-Riley model (Wells,  
35 1955; Riley *et al.*, 1978), which takes monitored  $\text{CO}_2$  data (the equipment to do so costing less than a  
36 couple of hundred pounds per sensor) and directly infers the risk of airborne infection without the need  
37 to assess/assume the ventilation rate (nor does it require the space to be in steady-state). Their model,  
38 assuming the presence of an infector, can be parameterised for any airborne disease to provide both the  
39 likelihood of infection and the reproductive number (or R-number, i.e. the average number of infections  
40 arising from a single infectious case) for indoor spaces over time periods selected such that the space  
41 remains constantly occupied.

42  
43 In addition to the monitored  $\text{CO}_2$ , the risk reported by Rudnick & Milton (2003) depends strongly on  
44 the time period of assessment, the occupancy level, the nature of the virus, and occupancy activity —  
45 the latter two aspects being parameterised via the rate of generation of infectious quanta  $q$  per person  
46 (usually expressed in quanta per hour). Wells (1955) conceived the idea of a quantum (or infectious dose)  
47 in an effort to describe the stochastic behavior of airborne infection, and values for the quanta generation  
48 rate have been derived for SARS-CoV-2 (e.g. Buonanno *et al.*, 2020a). However, just as for most other  
49 airborne diseases, there is wide variation in the values relevant for an infectious individual depending on  
50 a) their activity level, b) the viral load in their sputum and c) the ratio between one infectious quantum  
51 and the amount of infectious RNA/ml.

52  
53 Buonanno *et al.* (2020a) report that in typical scenarios with low activity levels,  $q \approx 1/\text{hr}$  may be  
54 appropriate for COVID-19. Buonanno *et al.* (2020a) further suggest that should an individual be vocal-  
55 ising (in a manner not dissimilar to talking) whilst carrying out light exercise (e.g. walking) then values  
56 as high as  $q \approx 100/\text{hr}$  can be inferred — for offices occupied and ventilated at appropriate levels (e.g.  
57  $\sim 10\text{ m}^2$  per occupant and the outdoor air supply per person is  $Q \approx 101/\text{s/p}$  Chartered Institution of  
58 Building Services Engineers, 2015) then for occupancy periods of constant occupancy of, say, four hours  
59 the R-number for the office (assuming a floor-to-ceiling height of between 3–4 m) would be around 4,  
60 i.e. from a single infector then four new COVID-19 infections would result. For more typical behaviour  
61 in an office, i.e. most occupants carrying out desk work with a few talking relatively quietly for which  
62  $q \approx 1/\text{hr}$ , then even assuming the office remains constantly occupied for a full nine hour day, R-numbers  
63 of around 0.1 are obtained (these rise to around 0.2 if the office is poorly ventilated, i.e.  $Q = 41/\text{s/p}$ ,

R-numbers, $R_A$	$Q = 41/s/p$	$Q = 101/s/p$	$Q = 201/s/p$
$q = 0.3/hr$	0.25	0.13	0.07
$q = 1/hr$	0.84	0.42	0.24
$q = 5/hr$	4.0	2.1	1.2
$q = 20/hr$	14	7.6	4.4
$q = 100/hr$	35	26	18

Table 1: COVID-19 R-numbers,  $R_A$ , calculated over the period that a pre/asymptomatic person remains attending work in an open-place office (floor plan of 400 m<sup>2</sup> and floor-to-ceiling height of 3.5 m) occupied by 40 people for 8 hrs each day (see Burrige *et al.*, 2020).

and drop to around 0.06 if the ventilation is doubled).

The results of Buonanno *et al.* (2020a) take a value of  $q = 142/hr$  and show the airborne infection risk for various public indoor spaces (namely, shops and restaurants), reporting R-numbers for differing exposure scenarios (changing outdoor air supply and occupancy scenarios) which they describe as before and after ‘lockdown’ (N.B. they do not report values for restaurants after lockdown). They select modelled durations of  $\sim 3$  hrs. For poorly ventilated spaces Buonanno *et al.* (2020a) report R-numbers ranging from  $2 \leq R \leq 50$  before lockdown ( $1.71/s/p \geq Q \geq 0.21/s/p$ ) and after lockdown  $0.1 \leq R \leq 0.8$  after ( $10.51/s/p \geq Q \geq 5.21/s/p$ ). For better ventilated spaces then  $1 \leq R \leq 6$  ( $101/s/p \geq Q \geq 4.51/s/p$ ) before lockdown and afterwards  $0.1 \leq R \leq 0.4$  ( $551/s/p \geq Q \geq 221/s/p$ ).

Since an occupant can become infected at any point on any given day then taking any particular duration seems an arbitrary choice which, for the most part, is hard to justify. Burrige *et al.* (2020) point out that for indoor spaces which are regularly/consistently attended by the same/similar group of people (e.g. open-plan offices or some school classrooms, and herein a ‘regularly attended space’) one should consider the likelihood of infection over a period during which an infectious person may remain pre/asymptomatic. For COVID-19 this period is currently estimated to be between five and seven days. They developed simple extensions to the work of Rudnick & Milton (2003) which enabled variations in occupancy behaviour and activity to be accounted for and the likelihood of infection to be assessed from monitored CO<sub>2</sub>. In doing so, Burrige *et al.* (2020) calculate the likelihood that an indoor space contributes to the spread of COVID-19 by assuming that a single pre/asymptomatic infector regularly attends the space and ceases to do so once they show symptoms reporting the absolute R-number,  $R_A$ , for modelled and monitored spaces.

As shown in table 1 for typical behaviour in a typical office Burrige *et al.* (2020) report  $R_A \approx 0.5$  which rises to  $R_A \approx 0.8$  for a poorly ventilated space ( $Q = 0.41/s/p$ ) and falls to  $R_A \approx 0.2$  if the ventilation is doubled — which are reassuringly below one, thereby suggesting that the return to desk-based office work is unlikely to contribute significantly to the COVID-19 spread. However, should most of the office remain sedentary but start vocalising (akin to a call-center, or similar) then  $R_A \approx 2$  — highlighting the importance of occupancy behaviour in determining whether or not a particular indoor space contributes to the spread of COVID-19 or not. Burrige *et al.* (2020) also present results for the airborne infection risk from CO<sub>2</sub> data monitored within an office with uncontrolled natural ventilation. The particular office is not of a modern well-sealed design. The risk levels within that monitored naturally ventilated office remain comparable with the modelled office, intended as being ‘typical’, therein. However, the airborne infection risk was calculated for periods when the windows were opened and for those when the windows remained shut — the risk of infection was approximately doubled when the windows remained shut. This provides quantitative support for the guidance that where practical ventilation openings (like windows, doors, etc.) should be opened, but doing so is not without conflict, see appendix A.

Such findings beg the question: how might the infection risk vary between seasons as weather changes from being typically temperate and what might this mean for the spread of COVID-19 in winter 2020? Vouriot *et al.* (2020) examined the R-number within various spaces inside schools. Their most meaningful results are for classrooms, which can be regarded as regularly attended spaces, where they reassuringly find R-numbers below one if pupils are assumed to be carrying out desk based learning in a relatively calm/quiet environment and quanta generation of  $q \approx 1$  are appropriate. The R-numbers rise to  $1.5 \leq R_A \leq 3.8$  if one assumes values of  $q \approx 5$  which are more appropriate if the class is carrying out more vocal activities and they rise further still if one assume pupils are actively moving around the classroom. Crucially, in all cases, the greatest R-numbers are obtained during colder winter months (for example,

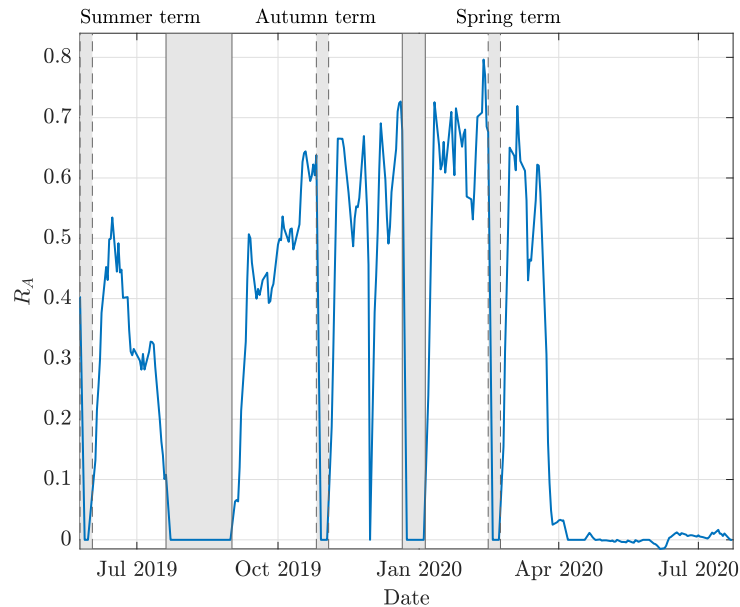


Figure 3: The variation in the absolute R-number,  $R_A$ , during the period 25<sup>th</sup> May 2019 to 20<sup>th</sup> July 2020 determined from monitored CO<sub>2</sub> and the school's occupancy timetable for a typical classroom within a relatively modern build school (rebuilt in September 2016). The shaded regions correspond to holiday periods. For data recorded during the summer term of 2019 the average was  $R_A = 0.35$ , for the autumn term of 2020 the average was  $R_A = 0.53$ , and for data recorded during the spring term of 2020 the average was  $R_A = 0.63$  until the lockdown of 20<sup>th</sup> March.

November to February) — being typically around 80% greater than those estimated for more temperate months (i.e. May to September). We see no reason not to expect similar trends in the reproductive number of indoor spaces beyond schools and such convictions underpin our guidance to assess and ideally monitor ventilation provision in order to understand the needs for modification and the implementation of other measures.

### 3.2 Ventilation guidance for winter 2020

A concern for the northern hemisphere winter is that buildings will become less well ventilated, with a lower supply of outside air in order to maintain warm conditions indoors. Ventilation systems that recirculate a proportion of the indoor air, primarily to temper the temperature of the outdoor air without increasing energy consumption, are common but it is crucial to regard only the flow of outdoor air as contributing to the ventilation rate (see appendix C.5 for more detailed discussion). We recommend evaluating the benefits of increased monitoring of the indoor environment, especially indicative outdoor air supply rates via CO<sub>2</sub> levels. CO<sub>2</sub> levels not exceeding 1000 ppm within an indoor space broadly indicating that outdoor air supply is likely to be adequate for offices and mechanically ventilated classrooms (i.e.  $Q \approx 10\text{l/s/p}$ ), with the equivalent level being 1500 ppm in naturally ventilated school classrooms (i.e.  $Q \approx 5\text{l/s/p}$ , see Annex A of UK Government, 2018*a*, for a full discussion). Doing so will assist in: a) highlighting high-risk spaces for which mitigation measures need to be considered (e.g. enhanced ventilation provision to manage certain activities or some of those measures detailed in §4), and b) helping obtain the quantitative evidence to reassure occupants as to their relative safety. Where monitoring of CO<sub>2</sub> is undertaken, consideration of the build-up and decay of CO<sub>2</sub>, and the variation in CO<sub>2</sub> levels within the space can provide additional insights; where experience or skills are lacking, engagement with a professional building services engineer would be beneficial. We note that the design of school classrooms will, in some cases, have been carried out allowing CO<sub>2</sub> levels to reach up to 2000 ppm for brief periods; as Vouriot *et al.* (2020) show, this does not necessarily indicate unacceptable risk levels for COVID-19. Moreover, active management of a classroom's windows and other ventilation openings may enable these high peaks to be avoided or minimised, appendix A provides discussion of strategies to do so in winter without excessively impacting occupant thermal comfort.



Where people are brought together in moderate to high densities for significant portions of the day and no monitoring is intended, we recommend that the design provision for ventilation of the indoor space be investigated to determine whether adequate ventilation is likely being achieved. Consideration should be given to installation and the subsequent maintenance to estimate whether or not the design provision might realistically be attained. Where the design provision is unavailable, efforts should be made to ‘reverse engineer’ an understanding of the ventilation. In the absence of knowledge as to the number of infected occupants, ventilation provision should always be considered per capita based on the design occupancy — we ventilate buildings for the sake of the occupants. Decreasing occupancy density should be considered both to minimise transmission via the droplet route, the fomite route (see 2.2) and, crucially, the airborne route as the per capita ventilation provision from fixed systems/plant can be drastically increased without new investment (see 2.4). Where ventilation rates meet existing design guidance the expected risk of airborne infection might be regarded as low. In the absence of monitoring, consideration should be given to potential stagnant zones within indoor space (e.g. a sheltered reading corners or break-out spaces); where suspected these should be addressed (see 4).

Where CO<sub>2</sub> monitoring is carried out, sensors should be placed at heights and locations within the space representative of the breathing zone. Where practical, occupancy and excess CO<sub>2</sub> (i.e. CO<sub>2</sub> above outdoor levels) should be recorded and simple calculations undertaken to estimate the level of risk to occupants (see, e.g. BurrIDGE *et al.*, 2020) — we expect that, in the most part, these calculations will prove simply reassuring. Where no calculations are desired, then for given activity within a space, it is worth noting that the airborne infection risk depends directly on the excess CO<sub>2</sub> relative to the number of persons responsible for producing it — this latter quantity being impossible to evaluate without monitoring (in which case the simple calculations of BurrIDGE *et al.* (2020) may prove more useful). In all cases, we suggest that consideration not only of risk but also the rate at which the risk is increasing with time is prudent. The relative rate of increase in airborne infection risk is directly proportional to the ratio of the instantaneous values excess CO<sub>2</sub> and current occupancy (see BurrIDGE *et al.*, 2020). These values are easily obtained and indicate the level of risk that will be realised should all else remain equal.

We note, however, that ventilation flows are complex and may involve air flow from one space in a building to another. They are also strongly influenced by the locations of openings and the strength of internal heat gains. Further work on the implications of these effects is needed.

Finally, we conclude that the risk of COVID-19 being spread by the airborne route is not insignificant, varies widely with activity level and environmental conditions (which are predominantly determined by the bulk supply of outdoor air), and are expected to increase in winter 2020 relative to summer.

## 4 Other mitigating measures

This section explores some of the additional measures available to mitigate risk of airborne transmission of COVID-19 in indoor environments. Here by ‘airborne’ transmission we mean transmission via smaller particles which can be suspended in the air for a considerable time. By ‘droplet’ transmission we mean short-range transmission via larger droplets which fall to a surface within seconds and within a few meters of the infected person.

The risk of airborne transmission indoors can be mitigated through dilution of the indoor air by clean outdoor air, as discussed in §3. This requires a ventilation system for which the air intake can be increased, or installing secondary ventilation systems. Substantially increasing the ventilation in a space is often impractical. Further, in order to reduce the risk of infection by a factor, the ventilation rate must be increased by the equivalent factor. Increasing the volume of outdoor air becomes particularly challenging in winter without compromising the thermal comfort of occupants or energy use (due to an increased heating load). These factors limit the viability of reducing risk by increasing ventilation.

The mechanisms for the transmission of COVID-19 are not yet well understood. Reducing the risk of infection to zero is not possible (short of global eradication), therefore, where practical to do so, all available measures should be taken in order to gain the biggest reduction in risk. In the absence of or to complement increased ventilation, alternative engineering control measures can be used. Some of the measures available are discussed here.

#### 4.1 Air filters or cleaners

Filters and air cleaners, such as UV cleaners or photocatalytic oxidation, can either be installed directly within the existing ventilation system, removing virus-laden particles from recirculated air, or as independent units within a room to supplement the existing ventilation.

In their latest guidance document in response to the pandemic, The Federation of European Heating, Ventilation and Air Conditioning associations (REHVA) recommend avoiding the use of centralised recirculation as typical local air filters within these systems are not effective at filtering out viral material which tends to be too small for the filter (REHVA, 2020). Installing high-efficiency particulate air (HEPA) filters would allow virus-laden particles to be removed, however these are not easily installed in existing systems and further system modifications are required in order to provide a higher pressure drop to maintain the same airflow rate.

Standalone air filters can be effective at removing virus-laden particles provided they target the appropriate range of particle size. However, regardless of the efficacy of the filter itself, the supply of clean air is limited by the flow rate of air passing through the filter. Despite this, the supply of clean air in a space can be up to  $1000\text{ m}^3/\text{hr}$  (Zuraimi *et al.*, 2011). However, filters require regular servicing to maintain their effectiveness - clogged filters lead to a build up of viral particles and can act as a source of viral matter rather than a sink (Eames *et al.*, 2009).

Air cleaners such as UV air cleaners are also limited by the volume of air that can be passed through the device. UV light has been shown to be effective in deactivating various viruses under laboratory conditions, including coronaviruses (Morawska *et al.*, 2020; Beggs & Avital, 2020). While the evidence that UV is effective against SARS-CoV-2 specifically is currently limited, it seems highly likely to be the case (Beggs & Avital, 2020).

The efficacy of air filters and cleaners is likely to be highly sensitive to their location within a room. Depending on the size, shape and airflow patterns within a room, the air within some areas of the room may never reach the device. In the worst case scenario, the device simply recirculates the same small volume of air within a much larger room. Therefore while these devices tend to promise certain air changes per hour (AC/hr) equivalent of clean air, in reality this is only true if the air pulled in by the device has not already been cleaned. In smaller, poorly ventilated rooms such devices are likely to provide significant benefit, however for larger spaces an understanding of the airflow patterns within the room is required to ensure that the device is effective.

Care should be taken when considering which air cleaner/filter to use as many devices have been found to have a limited effect (Siegel, 2016; Zuraimi *et al.*, 2011). HEPA filters are often recommended as the most effective technology currently available (e.g. Zuraimi *et al.*, 2011).

#### 4.2 Personalised ventilation

Personalised ventilation (PV) supplies clean air directly to the breathing zone of an occupant of a room via a device installed at their workstation. A certain minimum velocity is required for the supplied air in order to penetrate the convective flow driven by body heat (Melikov, 2004). Further, a large target area is desired in order to account for occupants' movement. The required rate of clean air supply can therefore be high. While studies have shown that PV can be effective at reducing risk for occupants while at their workstations (Melikov *et al.*, 2002; Melikov, 2004), protection is not provided to occupants when away from their workstations. PV may in fact facilitate the transport of exhaled pathogens to other occupants (Bolashikov & Melikov, 2009). Installing a personalised device for all occupants in an office is also likely to be expensive and impractical.

PV may be most viable in scenarios where the occupant is required to remain at their workstation for the duration of their shift.

#### 4.3 Desk and ceiling fans

While desk or ceiling fans do not enhance the bulk supply of outdoor air, they can be used to increase air mixing within a room, which may lead to a more homogeneous distribution of virus-laden particles. Where areas of stagnant air are identified (or are suspected), a fan can be used to increase mixing with the wider space, therefore potentially reducing the risk of the accumulation of virus-laden particles within the stagnant zone.



1  
2  
3 The localised (relatively) high velocity air flows produced by desk fans may result in increased re-  
4 suspension of virus-laden particles and this risk should be considered. However, studies have found  
5 that using either desk or ceiling fans to increase air mixing within a room can lead to increased rates of  
6 deposition (Mosley *et al.*, 2001; Thatcher *et al.*, 2002; Xu *et al.*, 1994). These experiments use oil droplets  
7 (Thatcher *et al.*, 2002), cigarette smoke particles (Xu *et al.*, 1994) and a combination of oil droplets,  
8 salt and incense (Mosley *et al.*, 2001) under controlled laboratory conditions. However, deposition rates  
9 can vary by orders of magnitude depending on the particle size, room surface-to-volume ratio (which  
10 was varied predominantly via the inclusion of furniture) and airflow speeds and turbulence within the  
11 space. Generally the removal of airborne particles through deposition is likely to be much lower than that  
12 through ventilation, however for poorly ventilated rooms these rates can be comparable, particularly for  
13 larger particles ( $> 1\mu m$ ). It is unclear whether the results of these studies would translate to increased  
14 deposition rates of COVID-19-laden particles under real-world conditions. The impact of changing  
15 deposition rates on the risk of surface transmission via fomites is also unclear.

16  
17 Due to this uncertainty and the potential risk of increasing the re-suspension of virus-laden particles  
18 within the space, the use of desk fans is generally not recommended. However, when a stagnant zone is  
19 evident, either via intuition (e.g. sheltered spaces like reading corners within classrooms and breakout  
20 spaces within open-plan offices) or through monitoring CO<sub>2</sub> concentrations, the benefit of using a desk  
21 fan to increase mixing with the wider space may outweigh the potential drawbacks. Where fans are used  
22 in this capacity they should be orientated such that increased mixing is achieved between the stagnant  
23 zone and the surrounding space (into which the outdoor air provision should be checked).

24  
25 Ceiling fans promote vertical mixing of air within rooms and, in addition to increased bulk mixing, help  
26 reduce temperature stratifications from forming within the room. As such where the ventilation strategy  
27 relies on stratification within the room, e.g. where a displacement ventilation strategy can be successfully  
28 evidenced, the use of ceiling fans is not recommended. Otherwise, temperature stratification within a  
29 room could significantly inhibit the vertical mixing, and dilution, of virus-laden particles from within  
30 the breathing zone and therefore the use of a ceiling fan is likely to be beneficial. Moreover, in the  
31 heating season the downwards mixing of warmer air from the ceiling can enable the increased supply of  
32 outdoor air without compromising thermal comfort (nor increasing the energy consumption associated  
33 with heating). Further, their use in conjunction with upper room UV (see §4.6) has been found to greatly  
34 increase the exposure of virus-laden particles to the upper region of the room (McDevitt *et al.*, 2008;  
35 Morawska *et al.*, 2020).

#### 36 4.4 Air ionisation

37  
38 Air ionisation is a relatively new technology and involves the production of ions such as the hydroxyl  
39 radical ( $OH^-$ ) from a corona discharge between two high potential electrodes. These ions have been  
40 shown to have germicidal properties as they react with the surface structure of the pathogen (Bolashikov  
41 & Melikov, 2009). The technology was shown to be effective against certain bacterial pathogens when  
42 installed in a hospital intensive care unit, but with no effect on others (Kerr *et al.*, 2005). Ionisers are  
43 easy to deploy and have high energy efficiency but have not been evidenced as effective devices against  
44 viruses including SARS-CoV-2. Some devices are known to produce ozone so their effect on air quality  
45 should also be considered (Siegel, 2016).

#### 46 4.5 The use of screens

47  
48 Using screens to provide a physical barrier between the occupants of a space is a simple and easily  
49 applied measure to mitigate the risk of transmission. The use of screens is widespread in hospitals and  
50 the commercial sector; in supermarkets they are used to provide a barrier between the shop assistant  
51 and the customer.

52  
53 However, this application is targeted at reducing the risk of infection via larger droplets. There are  
54 very few examples of the use of screens to mitigate airborne transmission (i.e. smaller droplets or  
55 droplet nuclei), either by providing a barrier directly between occupants, or in an attempt to favourably  
56 manipulate the airflow patterns within a space. Noakes *et al.* (2006) use CFD simulations to investigate  
57 the effect of partitioning a hospital ward room on the risk of patient-to-patient and patient-to-visitor  
58 transmission. They conclude that, combined with carefully considered changes to the positioning and  
59 number of the ventilation inlets and outlets, the risk of infection can be reduced significantly. However,  
60

1  
2  
3 in some instances the risk of transmission from patient to visitor was increased by the presence of the  
4 partition due to reduced airflow in certain areas of the room.

5  
6 This highlights the main problem with the use of screens indoors; the impact of the screen on the airflow  
7 patterns within a space is very difficult to predict. While the exchange of air between two areas of a  
8 room may be reduced, the presence of a screen can lead to areas of stagnant or recirculating flow where  
9 the virus could accumulate. Further to this, installing screens which affect airflow would require an  
10 evaluation of the impact on any environmental monitoring undertaken, and crucially on fire safety (e.g.  
11 as they may prevent smoke from reaching the smoke detector or have other fire safety implications).

#### 12 13 **4.6 Upper room Ultra-Violet Germicidal Irradiation**

14  
15 Upper room UV provides a way to utilise the deactivating properties of UV light without the limitation  
16 of the airflow rate through a cleaning unit. While real world studies of its application are limited (e.g.  
17 McLean, 1961), laboratory and modelling studies suggest that, provided sufficient efficacy of UV in  
18 deactivating the virus, the method can be effective at mitigating airborne transmission (e.g. Escombe  
19 *et al.*, 2009; Noakes *et al.*, 2015). Upper room UV is likely to be at its most effective in poorly ventilated  
20 spaces (Noakes *et al.*, 2015) and can be installed reasonably easily at a low cost. However sizing a system  
21 is not always straightforward, and it is essential that systems are installed by professionals who also  
22 ensure that occupied zone UV-C irradiation levels are safe.

23  
24 The method depends on the virus reaching the upper section of the room which is exposed to UV light.  
25 Therefore, appropriate internal flow patterns are required in order to ensure that the virus is transported  
26 through the unit and deactivated. The process is complicated by the large range in particle sizes (see  
27 §2.1.1) produced by an infected person, where larger particles require higher vertical velocities to be  
28 carried to the upper section. Therefore, while a large range of particle sizes may well be deactivated,  
29 there is a danger that a certain fraction remains. Studies such as Nardell *et al.* (2008) have demonstrated  
30 that the method can be implemented with minimal risk to occupants due to UV exposure. Further,  
31 “ozone-free” UV lamps can be used to avoid adverse effects on indoor air quality due to the production  
32 of ozone. Only real world trials will prove the efficacy of the method (see §C) and costs (of purchase,  
33 installation and maintenance) should always be compared to the equivalent costs of appropriate upgrades  
34 to the ventilation provision to the indoor space.

#### 35 36 **4.7 Summary**

37  
38 Each of the engineering control measures considered here have their advantages and limitations, and  
39 none are able to entirely eradicate the risk of transmission. Upper room UV holds promise as a method  
40 to significantly reduce the risk of transmission, particularly in poorly ventilated spaces. However further  
41 effort is required to demonstrate its effectiveness against SARS-CoV-2 in real world scenarios. Air  
42 ionisation is an emerging technology, but there are unresolved concerns regarding its impact on indoor  
43 air quality, primarily due to the production of ozone, and there is little evidence that it is effective  
44 against viruses. The addition of air filters that are effective against viral transmission to the existing  
45 ventilation system are likely to require complementary modifications to the ventilation system to, at the  
46 very least, account for the changes in pressure drops across the system. Independent filter units can be  
47 used, however in order to maximise their impact in larger spaces an understanding of the airflow patterns  
48 within the space is useful. Filters are an established technology which are likely to reduce risk without  
49 any detrimental impact to occupants if appropriate maintenance can be ascertained and achieved. Using  
50 desk fans to increase the mixing of air within a room is easily applied and may be a very affordable  
51 measure, however their merits are questionable in the absence of evidence of stagnant zones. Ceiling  
52 fans provide the benefit of increased vertical mixing within the room. However in the case of both desk  
53 and ceiling fans there are uncertainties as to their impact on re-suspension and deposition of virus-laden  
54 particles and the associated impact on transmission risk. The use of screens can be effective to mitigate  
55 spread via the droplet route, but their use is problematic as a measure against airborne transmission since  
56 impacts on the circulation of air and the local ventilation provision is unpredictable without detailed  
57 bespoke study. Moreover, a full assessment of a screen’s impact on fire safety would be required ahead  
58 of any installation.

59  
60 The primary control measure for airborne transmission indoors is ventilation and achieving adequate  
outdoor air supply rates to ensure an acceptable level of risk should be the priority. However, where  
additional risk reduction is required or desired other measures can be implemented to help mitigate

the spread of COVID-19. Where the intention of these measures is to reduce the risk of airborne transmission their costs should always be compared to the cost of upgrading the existing ventilation provision. Moreover, these measures should always be implemented in addition to any existing measures to reduce the spread of infection via all routes of transmission, e.g. adequate hand hygiene & cleaning, the wearing of face coverings or other personal protection equipment (PPE) and social distancing.

## 5 Author contributions

This document results from the work and discussions led by Professors Paul Linden (pfl4@cam.ac.uk) and Christopher Pain (c.pain@imperial.ac.uk) under Task 7 (Environmental and aerosols transmission) within the Royal Society's 'Rapid Modelling of the Pandemic project' (RAMP).

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## Supplementary material

### A Natural inputs to ventilation provision

By design well-sealed buildings have ventilation provision that has been specified and installed, as per our guidance (§1 and §3.2) we recommend this provision is reviewed in light of COVID-19 prior to winter 2020. Many more buildings are, by design or otherwise, not well-sealed and in such cases the increased supply of outdoor air by natural means (e.g. through opening windows, doors, vents, louvres, etc. . . ) may offer potential to mitigate risk of airborne transmission. However, these naturally driven flows rely on driving winds (which in winter are typically colder than comfortable indoor temperatures) or buoyancy (arising from temperature differences between indoor and outdoor air) and so if not managed carefully will lead to either significant increases in heating bills and/or zones where cold draughts are uncomfortable. In this appendix, we seek to discuss practical means by which these natural flows can be exploited to mitigate risk but in a manner which attempts to balance the potentially negative consequences for indoor experience and energy consumption.

#### A.1 Natural Ventilation in winter

The amount of outdoor air that can be reasonably provided during winter may be less than in the summer due to impacts on indoor air temperature and occupant comfort. As there is a high confidence that, compared to adequately ventilated spaces, poorly ventilated spaces increase the risk of SARS-CoV-2 transmission via the far field (>2m) airborne route, it is important to ensure that poorly ventilated spaces are avoided.

Approved Document Part F (ADF) sets out what, in ordinary circumstances, may be accepted as reasonable provision for compliance to the Building regulations: *There shall be adequate means of ventilation provided for people in a building* (HM Government, 2013). At the least, it is important to ensure that adequate ventilation is provided year round (poor indoor air quality also negatively impacts health, wellbeing and productivity Kukadia & Upton, 2019). Wherever possible outdoor air flow rates should be maximised (i.e. increased more than the ADF provision rates for adequate airflow) where it is reasonable to do so. For low occupancy indoor spaces it may be that an airflow rate measured in litres per second per person (l/s/p) only provides a low overall flow rate and in these cases

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2  
3 a minimum flow rate should be considered (Jones *et al.*, 2020).  
4

5 However increased ventilation in the winter may lead to unwanted oc-  
6 occupant behavioural responses that result in ventilation provision being  
7 deactivated or minimised. For example, increased ventilation could re-  
8 sult in colder indoor environments or cold draughts resulting in occupants  
9 closing or turning off ventilation provision. Thus, the goal of increasing  
10 ventilation provision results in no, or little, ventilation provision if such  
11 behavioural responses are elicited. Ventilation plays a key role in dilution  
12 of airborne SARS-CoV-2 encapsulated in respiratory aerosol — poorly  
13 ventilated spaces increase the risk, although there is a diminishing law of  
14 returns in increasing the ventilation rate. The potential benefits of increas-  
15 ing ventilation to a poorly ventilated space are greater than increasing a  
16 well-ventilated space by the same amount (Jones *et al.*, 2020).  
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23 In winter, the driving forces for natural ventilation are usually greater  
24 (pressure differences caused by wind and indoor/outdoor temperature dif-  
25 ferences Chartered Institution of Building Services Engineers, 2015, 2005)  
26 and therefore, to deliver the same flow rate, openings do not need to be as  
27 wide in the winter as they would need to be in the summer. What follows  
28 provides guidance for modulating various ventilation openings to deliver  
29 outdoor airflows whilst minimising occupant discomfort.  
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#### 35 **A.1.1 Adjusting airflow through natural ventilation openings**

36  
37 *A single set of high- and low-level openings:* In this configuration it is  
38 preferable to open the high level vents first to provide outdoor air, and to  
39 open the low level windows to further maximise airflow when reasonable.  
40 The turbulent plume of cooler outdoor air entering through high level  
41 vents will entrain warm room air as it falls under gravity, tempering the  
42 air before it enters the occupied zone (Turner, 1973). A helpful draught  
43 plume calculator is available in the BB101 calculation tools, which enables  
44 this effect to be measured (UK Government, 2018b). A safe means of  
45 opening and closing high level vents should be supplied in workplaces (HM  
46 Government, 1992b, , regulation 15).  
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53 *Multiple openable windows and/or vents:* Where a room has multiple open-  
54 able vents, it may be possible to deliver the adequate ventilation provision  
55 through opening of just one vent. However, it is usually possible to cre-  
56 ate a more comfortable indoor environment, with respect to draughts, if  
57 the airflow is achieved through opening all the vents by a smaller amount  
58 than that necessary in the scenario of a single set of openings as described  
59  
60

1  
2  
3 above. If there are openable vents at both high and low level, then the  
4 principle of opening as many high level vents should initially be considered  
5 (see above).  
6  
7

8 *Sash windows:* As with high and low level windows, it is better to open  
9 the high level sash to provide openings at the top of the vent to encourage  
10 entrainment of outdoor air with the warm indoor air in the first instance.  
11 To further increase outdoor airflow the bottom sash can also be opened.  
12  
13

14 *Other vents:* In addition to windows, there are other vents and louvre  
15 systems that can be modulated and similar to windows, the principles  
16 of opening high level vents and multiple vents a small amount should be  
17 considered in the first instance.  
18  
19

20  
21 *CO<sub>2</sub> sensors:* High levels of CO<sub>2</sub> (i.e. values greater than 1 500 ppm)  
22 are indicative of a poorly ventilated space, and therefore CO<sub>2</sub> can be a  
23 useful indicator of a space that is lacking adequate ventilation. However,  
24 low CO<sub>2</sub> concentrations are not necessarily indicative of a well ventilated  
25 space. If CO<sub>2</sub> sensors are to be deployed they should be Non-dispersive  
26 Infra-red (NDIR) CO<sub>2</sub> sensors, which actually detect CO<sub>2</sub> in the space,  
27 rather than the less expensive eCO<sub>2</sub> sensors that do not detect CO<sub>2</sub> and  
28 infer a CO<sub>2</sub> concentration by measuring room volatile organic compound  
29 (VOC) concentrations instead.  
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#### 36 **A.1.2 Occupant Comfort**

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38 A person's sensation of warmth is influenced by the following main physical  
39 parameters, which constitute the thermal environment:  
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- 41 ● air temperature,
- 42
- 43 ● mean radiant temperature,
- 44
- 45 ● relative air speed, and
- 46
- 47 ● humidity.  
48

49 Besides these environmental factors there are personal factors that affect  
50 thermal comfort:  
51

- 52 ● metabolic heat production, and
- 53
- 54 ● clothing.  
55

56  
57 It is possible to adjust the personal environmental factors to improve oc-  
58 cupant comfort, particularly where outdoor air supply may decrease occu-  
59 pant comfort. CIBSE Guide A describes the predictive mean vote (PMV)  
60

1  
2  
3 method of measuring occupant thermal comfort, which can be used to see  
4 how changes to apparel and metabolic activity can improve occupant ther-  
5 mal comfort (Chartered Institution of Building Services Engineers, 2015).  
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8 *Clothing:* Clothing provides a layer of insulation that contributes to the  
9 occupant's perception of comfort and is dependent upon the material and  
10 fit. Typically winter wear would have a value of 0.8 to 1.0 clo, although  
11 studies in recent decades in Europe and the UK have found values generally  
12 at the lower end. This may be a result of occupants acclimatising to,  
13 and expecting, warmer indoor environments in the winter. For example  
14 compare typical winter office clothing of the 21<sup>st</sup> Century, with that in  
15 vogue at the turn of the 20<sup>th</sup> Century. To improve occupant comfort,  
16 particularly in naturally ventilated indoor spaces, occupants should be  
17 encouraged to dress appropriately and relaxation of dress codes should be  
18 considered if necessary to allow warmer clothes to be worn.  
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25 *Metabolic rate:* Sedentary activities have a relatively low metabolic ac-  
26 tivity which can contribute to occupant thermal discomfort. Encouraging  
27 periods of activity, to move around the room, or partake in some light ex-  
28 ercise, will help to improve thermal comfort – as well as aiding in meeting  
29 display screen equipment (DSE) regulations which require regular breaks  
30 or changes in activity when using DSE (HM Government, 1992a) and rec-  
31 ommendations for regular movement to improve health.  
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36 *Position of occupants in relation to openable vents:* Where it is possible,  
37 increasing the distance of occupants from openable vents provides more  
38 time for incoming cool air plumes to entrain with warm room air prior to  
39 entering the occupied zone.  
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## 43 **A.2 Summary**

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45 We summarise by first suggesting that when attempting to reduce the risk  
46 of airborne spread of COVID-19 by increasing ventilation through natural  
47 means (e.g. opening windows wider) occupants comfort be given due con-  
48 sideration; otherwise, the chances that occupants act to intervene and un-  
49 intentionally increase infection risk (e.g. by closing windows) is significant.  
50 To that end, the principles that high-level ventilation openings should be  
51 preferentially used, and that multiple smaller openings are preferable to a  
52 lesser number of larger openings, should be employed. Given that the max-  
53 imum capacity of heating systems are broadly fixed and should be designed  
54 to accommodate the ventilation air required to deliver adequate indoor air  
55 quality, we do not encourage large increases in energy consumption, to  
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ventilate an indoor space, beyond that which is absolutely necessary.

## B SARS-CoV-2 on hard surfaces

Respiratory viruses can be transmitted from an infected individual to a susceptible host through three main pathways illustrated in Figure 4. In this report, we focus on pathogen transmission mediated by surfaces of inanimate objects known as *fomites* that, when contaminated with an infectious agent, can transfer disease to a new host. Fomite contamination can occur through: (i) direct contact with infected individuals and their bodily secretions/fluids; (ii) bioaerosols generated via breathing, talking, sneezing, or coughing; (iii) by an airborne virus that settles after a contaminated fomite is disturbed. A susceptible individual coming into contact with a contaminated fomite surface may pick up the pathogen and self-inoculate leading to infection.

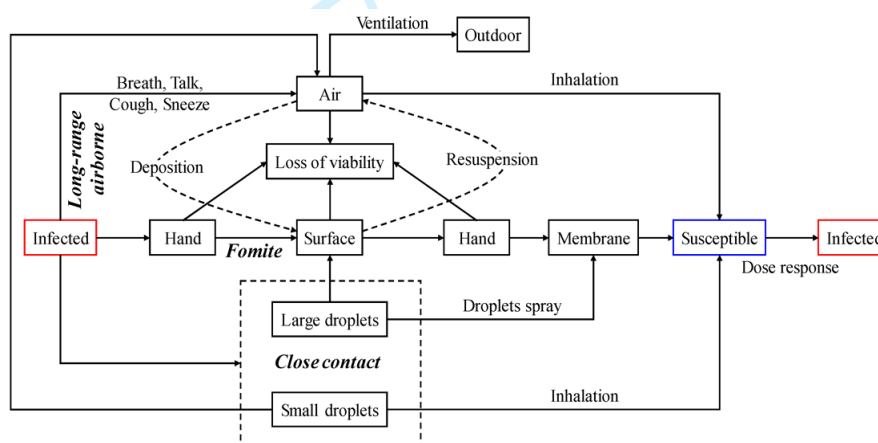


Figure 4: Three potential transmission pathways for respiratory viruses. [obtained from Zhang *et al.* (2018)]

Risk of viral infection through fomites can be assessed on the basis of three main considerations which determine the quantity of pathogen picked up upon touching the surface:

1. The concentration of virus on the surface, which is affected by
  - (a) source of virus (*e.g.* sneezing, coughing or touching the surface by infected individuals)
  - (b) time elapsed since surface contamination
  - (c) surface characteristics (*e.g.* material, smoothness, roughness, porosity) which affect the survival of the virus

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2  
3 (d) environmental and skin conditions (*e.g.* temperature, humidity,  
4 perspiration)  
5

6 (e) cleaning and/or disinfection activities since contamination  
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8  
9 2. The probability/frequency of contact with a contaminated fomite (*e.g.*  
10 through touch by hand); probability of virus transfer from hands to  
11 the mucous membranes of mouth or nose  
12

13  
14 3. The individual susceptibility to the virus (dose response curve)  
15

16 Many of the above factors are hard to quantify or control, and hence there  
17 is limited existing knowledge on transmission via fomites. In most settings  
18 the exact pathway of respiratory virus transmission is not known because  
19 of continuous interplay between the surface and droplet/airborne trans-  
20 mission pathways through droplet deposition and possible re-suspension  
21 of the pathogen. Although most of the evidence of fomite transmission  
22 is indirect, and no evidence has yet been reported for SARS-CoV-2, it is  
23 generally accepted that viruses can survive on hard surfaces for prolonged  
24 periods of time, that they can be transferred between fomites and hands,  
25 and that hands come into contact with mucous membranes, thus enabling  
26 this transmission pathway (Boone & Gerba, 2007; Kutter *et al.*, 2018).  
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32 The report will not cover aspects related to aerosol deposition of the virus  
33 (including the effects of ventilation on the rate thereof) which, although  
34 crucial for the quantification of risk of infection, is not within the remit of  
35 Subgroup 4. The topic of virus re-suspension will also not be covered due  
36 to lack of evidence in the literature.  
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#### 41 **B.1 Surface deposition and contact frequency**

42

43 The risk of infection through fomites depends on the frequency of contact  
44 with contaminated objects. It is therefore important to have quantita-  
45 tive information about the level of contamination of different surfaces as  
46 well as the likelihood of susceptible individuals coming into contact with  
47 them. Figure 5, from Chia *et al.* (2020) shows that SARS-CoV-2 is easily  
48 transported and deposited onto surfaces in a hospital setting (airborne in-  
49 fection isolation rooms), with certain surfaces that are commonly handled  
50 showing traces of the virus. Note that the figure indicates the likelihood  
51 of finding the virus on different surfaces but does not reflect actual viral  
52 loads found on them.  
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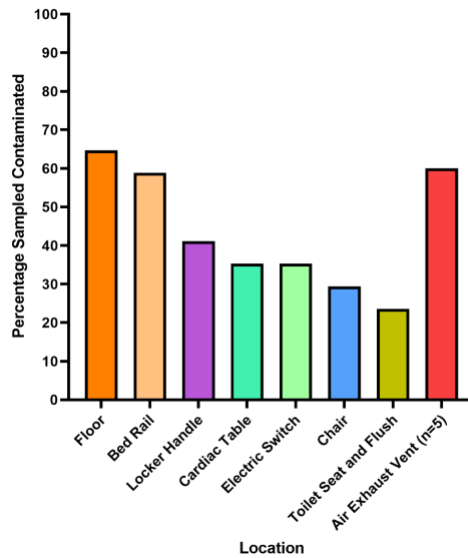


Figure 5: Percentage of contaminated swabs from surface samples in a hospital room housing a SARS-CoV-2 patient. [obtained from Chia *et al.* (2020)]

Zhang *et al.* (2018) have reported a large study of surface touch in a student office, in which they collected over 120,000 touch events. They introduce a classification of surfaces into high-touch (*e.g.* those touched multiple times by the same person) and high-risk surfaces (touched by multiple individuals) as well as private (*e.g.*, a mobile phone) and public (*e.g.*, door knob, light switch, etc.). The study revealed that 96.7% of touches in an office setting involved private surfaces and only 1.2% of the touches were of public surfaces which play the most important role in fomite transmission. The authors use a network approach to assess ‘distances’ between surfaces in the surface touch network and the dynamics of spread throughout the office. The paper also reports frequencies and average durations of contact with various surfaces in a student office which provides rich data for modelling transmission.

## B.2 Factors affecting survival of virus on surfaces

The survival of viruses on surfaces is strongly impacted by the type of surface and surface material upon which it is deposited as well as by a number of environmental factors such as ambient temperature, light, humidity, and pH (if in droplet form on surface). Additionally, factors such as type of contamination (fingertip, droplet, etc.) and the concentration of the virus inocula (relevant to droplets) are important (Warnes *et al.*, 2015). For SARS-CoV-2, the virus was found to be active across a wide range of pH, from 3 to 10 (Chin *et al.*, 2020) and so changing the pH to deactivate the virus may not be a feasible option.

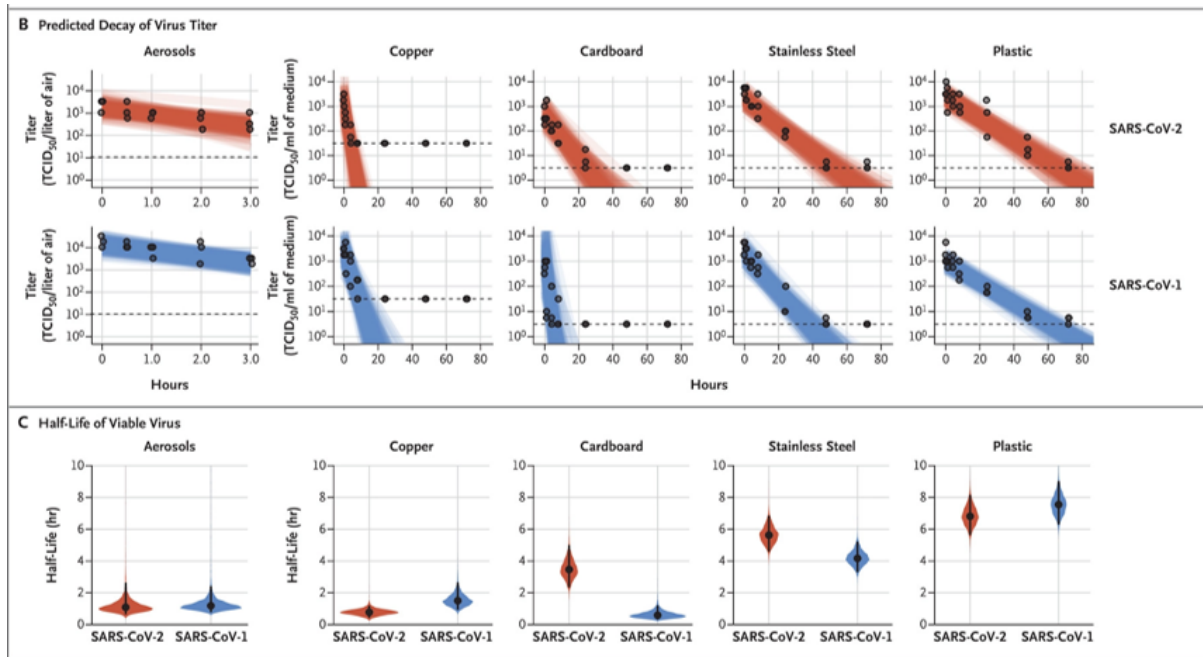


Figure 6: Exponential decay and half-lives of SARS-CoV-2 virus (red) and SARS-CoV-1 virus (blue) on different surfaces. [obtained from van Doremalen *et al.* (2020)]

### B.2.1 Surface material

The type of surface can affect the stability of the SARS-CoV-2 virus. A study has investigated the stability of both SARS-CoV-1 and SARS-CoV-2 viruses on plastic, stainless steel, copper and cardboard surfaces under conditions of 21–23°C and 40% relative humidity (van Doremalen *et al.*, 2020). Note that the initial concentration of the virus used on the surface is  $10^{3.7}$  TCID<sub>50</sub> per ml of medium. The virus decayed exponentially with time on each of these surfaces, as seen in Figure 1. The half-lives of the virus on plastic and stainless steel have been reported as 6.8 hours and 4.6 hours, respectively. From Figure 6, the half-lives of the virus on cardboard and copper are approximately 4 hours and 1 hour, respectively. This study also reports that the amount of infectious virus greatly reduced within 72 hours and 48 hours for plastic and stainless steel, respectively, and that no infectious virus was found on cardboard and copper after 24 hours and 4 hours, respectively. Another, more recent study (Riddell *et al.*, 2020) reports significantly longer survival times for SARS-CoV-2 on surfaces. In particular, at 20°C and 50% relative humidity the half-life of the virus is reported to be 43 hours for stainless steel, 49 hours for a polymer bank note, 46 hours for glass and 66 hours for a paper bank note. This study also demonstrates a substantial decrease in the survival times with increasing ambient temperature.

Table 2: Time taken (approximate) for no infectious virus to be found on each surface (Chin *et al.*, 2020).

<i>Surface</i>	<i>Time taken (hours)</i>
Stainless Steel	168
Plastic	168
Wood	48
Cloth	48
Paper	3
Tissue	3
Glass	96
Bank note	96
Mask (inner layer)	168
Mask (outer layer)	>168

The stability of the SARS-CoV-2 virus on stainless steel, plastic, wood, cloth, paper, tissue, banknote and inner and outer layers of a surgical mask has been evaluated by Chin *et al.* (2020). Each surface was pipetted with a virus culture of approximately  $10^{7.8}$  TCID<sub>50</sub> per ml of medium (higher than the concentration used in van Doremalen *et al.* (2020)), maintaining conditions of 22 °C and 65% relative humidity. This study has not reported the half-lives of the virus on each of these surfaces; however, the authors have reported the time it took for no infectious virus to be detected on each surface. Table 1 summarises their findings.

Results also show that both inner and outer layers of the mask retain infectious virus for a significantly long time (Chin *et al.*, 2020). However, it is possible that their experimental approach does not truly reflect a real scenario, as the virus culture has simply been pipetted onto the inner layer of the mask. It is not clear from this study whether it is possible for the virus medium (especially in droplet form) to seep through from the outer layer to the inner layer and retain a high concentration of the virus within the inner layer. Furthermore, it is unlikely that the inner layer of the mask would remain at 22 °C in reality, as exhaled air from the nose would increase the temperature.

The study also concludes that the virus remains stable for longer on smooth surfaces (Chin *et al.*, 2020); however, note that roughness/smoothness has not been quantified. Exceptions to this have been seen in a study performed on the Coronavirus 229E strain (Warnes *et al.*, 2015). The authors of this paper report that an increase in the copper content of a surface (such as in brass) can significantly reduce the time taken for the virus to be inactivated. They claim that this inactivation is due to the release of copper ions and reactive oxygen species. It is possible that this phenomenon is the cause of the low half-life reported for SARS-CoV-2 on



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3 copper, as seen in van Doremalen *et al.* (2020).  
4

5 Biryukov *et al.* (2020) has studied the half-life of SARS-CoV-2 on stainless  
6 steel, plastic, and nitrile gloves (all nonporous surfaces) under various tem-  
7 peratures and relative humidities (see section on Effects of Temperature  
8 and Humidity). Interestingly, their results do not show significant varia-  
9 tions in viral decay rates on these surfaces when environmental conditions  
10 are kept the same, and conclude that surface type does not impact viral  
11 stability. It is unknown why these results are different from those of van  
12 Doremalen *et al.* (2020) and Chin *et al.* (2020), highlighting the need for  
13 further experiments in this area.  
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### 20 **B.2.2 Effects of temperature and humidity**

21  
22 Experiments on SARS-CoV-2 under varying temperature have showed that  
23 the virus is less stable under increasing temperature (Chin *et al.*, 2020;  
24 Dietz *et al.*, 2020). Chin *et al.* (2020) show that the inactivation of the virus  
25 can be reduced to 5 minutes at a temperature of 70 °C, whilst remaining  
26 stable for over 14 days at 4°C.  
27  
28  
29

30 Biryukov *et al.* (2020) have analysed the effects of different temperatures  
31 (24°C and 35°C) and relative humidities (20%, 40%, and 60%) on SARS-  
32 CoV-2, diluted in saliva and deposited on surfaces in droplet form. They  
33 confirm that the half-life of the virus is reduced when temperature and  
34 humidity are increased, either independently or in combination (the lowest  
35 half-life of approximately 2 hours has been reported for the temperature-  
36 humidity combination of 35°C and 60%). While they also analyse the  
37 effects of different droplet sizes and deposition on three different nonporous  
38 surfaces (stainless steel, plastic and a nitrile glove), they conclude that the  
39 size of the droplet and surface type do not significantly impact the half-life.  
40  
41  
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45 Riddell *et al.* (2020) report SARS-CoV-2 survival times at 50% relative  
46 humidity and three temperature levels (20°C, 30°C and 40°C) showing  
47 that the half-life of the virus reduces drastically with temperature from 43  
48 – 66 hours at 20°C to 10 – 33 hours at 30°C to 1.5 – 3 hours at 40°C for  
49 most surfaces.  
50  
51  
52

53 Studies on other strains of coronaviruses show similar trends (Kampf *et al.*,  
54 2020; Ren *et al.*, 2020). One study reports that temperatures of 30 – 40°C  
55 is sufficient to reduce time taken to inactivate viruses (Kampf *et al.*, 2020).  
56 Casanova *et al.* (2010) also concludes that increasing both temperature and  
57 humidity have contributed to faster inactivation/decay of coronaviruses. A  
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3 review on viruses on surfaces has reported that the viruses are more stable  
4 at relative humidity levels below 50% (Vasickova *et al.*, 2010). Dietz *et al.*  
5 (2020) predicts that higher relative humidity facilitates larger droplets con-  
6 taining viruses to settle on surfaces more quickly, thus reducing airborne  
7 transmission (Dietz *et al.*, 2020).  
8  
9

10  
11 Based on these results, we can conclude with a high level of certainty  
12 that virus stability on surfaces decreases with increasing temperature and  
13 humidity. In reality, relative humidity levels should be increased cautiously  
14 as levels of 80 % or greater can lead to mould growth (Dietz *et al.*, 2020).  
15  
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### 18 **B.2.3 Effects of surface finish, texture or roughness**

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20  
21 All surfaces are inherently rough at some length-scale, and the particular  
22 quality of the surface is determined by the method of manufacture and any  
23 finishing processes, such as polishing. Even with the most sophisticated  
24 finishing processes, it has to be accepted that some roughness remains,  
25 and that the length-scale of the roughness features might be of a similar  
26 size to that of some droplets falling onto that surface. This then prompts  
27 questions as to differences in evaporation, adhesion, surface tension, and  
28 touch transferability of viral material contained in droplets that land proud  
29 on a smooth (or relatively smooth) surface, to those that land or flow down  
30 into roughness grooves in the surface.  
31  
32  
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35  
36 The science of surface characterisation, rubbing friction and surface lu-  
37 brication is called “Tribology”, and was established by Bowden & Tabor  
38 (1950). Surface texture (deliberate or arising from roughness) is charac-  
39 terised in a number of ways, but a common metric is  $R_a$ , which is defined  
40 as the arithmetical mean deviation of the surface profile being assessed.  
41 Other metrics consider peak to trough distance, root-mean-square, statis-  
42 tical aspects (skew and kurtosis), or reflect more directly the method of  
43 measurement (sizes and numbers of peaks and troughs over a given mea-  
44 surement area). Another approach recognises a fractal-like distribution  
45 in roughness and characterises roughness based on Box-Fractal dimension  
46 (Mandelbrot *et al.*, 1984; Mandelbrot, 2006). For the present purposes, a  
47 suitable characterisation should express the fraction of surface area which  
48 would be likely to catch droplets of a given size within a trough-like re-  
49 gion. On the basis of simple geometric considerations, one could imagine  
50 that for some combination of surface roughness types and droplet size and  
51 number, the contamination would mainly fall beneath the level at which  
52 a finger could press into the surface to touch, as illustrated in Figure 7.  
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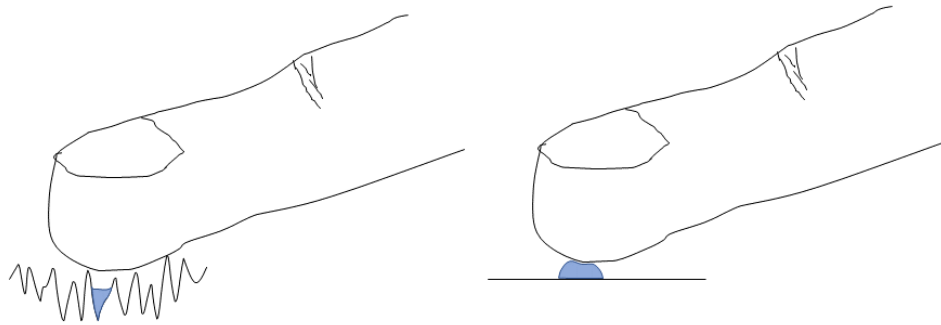


Figure 7: Illustration of how a droplet in a surface roughness groove (left) can be inaccessible to finger touch compared with a droplet on a smooth surface (right).

Another aspect that would influence the survival of infected droplets on surfaces is the adhesion of the droplet to the surface. The authoritative text on the subject of adhesion and adhesives is that by Kinloch (1987). The geometric interlocking of interfaces is not thought to be a strong factor in surface adhesion, but in considering the removal of droplets or dried droplet residue from a surface, there is a question to be asked of whether such material can be wiped off cleanly, or whether some material is forced more deeply into grooves on the surface. Understanding adhesion mechanisms at the micro-scale could provide some valuable insight into the mechanical aspects of the cleaning process.

### B.3 Modeling approaches

#### B.3.1 A model to predict viral transfer from surfaces

Transfer of microorganisms to hands from contaminated surfaces can be modelled mechanistically King *et al.* (2020):

$$C_H = \lambda_{S \rightarrow H} C_S \quad (1)$$

where  $C_H$  is the concentration of virus on fingers,  $\lambda_{S \rightarrow H}$  is the percentage transfer during a single contact which is pathogen and surface dependent and  $C_S$  is the concentration on the surface. This can be extended to multiple contacts:

$$C_n = C_{n-1} + \lambda_{S \rightarrow H} (C_S - C_{n-1}) \quad (2)$$

where  $n$  is the  $n^{\text{th}}$  surface contact.  $\lambda_{S \rightarrow H}$  is difficult to measure accurately and while experiments are underway to quantify this for SARS-CoV-2, surrogates can be used. Kraay *et al.* (2018) suggest  $\lambda_{S \rightarrow H}$  for influenza may

lie between 4% and 16%, whilst rhinovirus may range up to 40%. Data of finger to mucosa transfer is much more scarce but Rusin *et al.* (2002) estimates a viral surrogate at 33.9% (unpublished standard deviation was later found to be 16%).

### B.3.2 A model to predict viral decay rate

A simple thermodynamic model has been developed by Yap *et al.* (2020) which reasonably predicts the decay rate of different coronaviruses including SARS-CoV-2 at different temperatures. By assuming an exponential decay for the quantity of pathogens,  $C$ , with

$$C = C_0 e^{-kt},$$

where  $C_0$  is the initial quantity,  $t$  is time (seconds) and  $k$  is the decay rate. The decay rate is assumed to satisfy a relation (the *Arrhenius equation*) that is typical for the thermal denaturation of protein, namely

$$k = A e^{-E_a/RT},$$

where  $T$  is the ambient temperature (Kelvin),  $R$  is the Gas constant,  $E_a$  is the activation energy (J/mol) and  $A$  is an arbitrary frequency in the limit of high temperature. In particular, they found a universal relation (a *Meyer-Neldel relation*) between  $E_a$  and  $A$  for different coronaviruses, namely

$$\log(A) = 0.394E_a - 5.63,$$

based on experimental data from previous work. Although it has been assumed that this relation holds for all temperatures, the authors claimed that this is a reasonable assumption for proteins and, most importantly, this relation also has good agreement with previous findings. For SARS-CoV-2, we can readily predict the decay rate using the experimentally fitted value  $E_a = 135,692$  J/mol.

The authors have also suggested that this model can be extended to incorporate other catalytic effects *e.g.* humidity and surface properties by establishing further relations with the activation energy.

### B.3.3 Droplet evaporation from a surface

Numerous studies exist for the size of saliva droplet upon expulsion from the mouth (via breathing and coughing), with results between studies showing considerable variance. However, by assuming the mechanical

properties of pure water, the mathematics is well-established for predicting the time scale for such droplets to evaporate, namely

$$t_{\text{evap}} \sim \frac{\rho\theta R_0^2}{D(1-H)c_{\text{sat}}},$$

where  $\rho$  is the density of water,  $\theta$  is the contact angle,  $D$  is the diffusion coefficient,  $H$  is the relative humidity,  $c_{\text{sat}}$  is the saturation concentration of vapour in air, and  $R_0$  is the initial droplet radius (Stauber *et al.*, 2014, 2015) [see Dunn *et al.* (2009), Schofield *et al.* (2018) and Bhardwaj & Agrawal (2020) for further details on how these parameters depend on temperature and humidity].

There is evidence of a slower drying time of saliva as compared to pure water; In Liu *et al.* (2017), saliva droplets released onto a Teflon-printed slide showed a 20% longer evaporation time than those consisting of pure water. These results were especially pronounced at lower humidities and over longer time periods. Additionally, the same study notes evidence that droplets produced by coughing may be more likely to contain a higher solute concentration, due to the presence of pulmonary mucus. Droplets produced by coughing are also larger in size (Chao *et al.*, 2009), indicating a greater likelihood to be deposited on surfaces.

Under the assumption of pure water properties, such physical modeling indicates total droplet evaporation within seconds, which is multiple orders of magnitude less than the typical lifetime of SARS-CoV-2 on various surfaces. The results suggest that SARS-CoV-2 do not require a saliva droplet to survive although we have not found any evidence corroborating this. However, this time scale can be of interest for frequently touched surfaces, *e.g.* door handles. In such settings, a droplet with a high viral load can transfer from surface to skin by contact.

#### **B.3.4 Modelling of droplet residue and how its mechanical performance and attachment to surfaces could influence its survival**

At the present time, there has been no micro-mechanical characterisation of the material properties of partially-evaporated saliva droplets; however, if such properties were known then computational models of droplets undergoing evaporation could be made. At some point during this process, the droplet would first show higher viscosity, then become increasingly non-Newtonian as it transitions from being fluid-like to becoming a gel, or solid-like. The morphology of the droplet during that process would be



1  
2  
3 interesting to study, particularly in regard to how the evaporation process  
4 drives the droplet shrinkage.  
5

6 It is fairly obvious that a rough surface provides more surface area than  
7 a smooth one, and so a fluid material spread over a rough surface would  
8 have more contact, and therefore be more greatly affected by heat conduc-  
9 tion between the surface and the fluid. Depending on the surface tension  
10 characteristics, and how well the fluid wetted the surface, then there could  
11 also be greater fluid spread, and more area available for evaporation.  
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16 The following is mere supposition, but illustrates how having a better  
17 understanding the material properties would yield a better understanding  
18 of viral survival and the effect of surface roughness in that survival. Let us  
19 suppose that an outer layer of the droplet forms a skin or crust: an outer  
20 layer, exposed to the air, and therefore more dried out by the evaporation  
21 process. Let us suppose that one action of the skin is to protect or reduce  
22 evaporation of the inner material. Should that skin rupture, then the  
23 inner material would become exposed, and that would then form a skin,  
24 and the volume of inner material would be reduced. Encouraging the  
25 skin to rupture frequently could lead to faster evaporation and thus to  
26 complete drying out. Depending on the adhesion strength of the droplet  
27 to the walls of roughness grooves in the material, it is possible that the  
28 evaporation could lead to the skin becoming stretched and tearing. The  
29 sort of modelling result that such analysis could provide are given in Figure  
30 8, but these are to be understood to be illustrative only, since the materials  
31 property data they are based on is fabricated. If the virus requires some  
32 moisture to survive then devising ways of obtaining greater degrees of  
33 drying would be beneficial to us.  
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43 Alternatively, the more important aspect might be the ease of which a part  
44 of the droplet can be adhered to a finger and plucked from the surface.  
45 What are the relative adhesion strengths of the droplet to hard surface,  
46 and droplet to finger? Does finger dampness play a role in this? When  
47 a droplet is picked up on a finger in this way, is it transferred whole, or  
48 does a part remain stuck to the surface? If the latter is the case, then  
49 presumably the moister inner material is then exposed, and perhaps the  
50 virus is more readily transferred from the finger to other parts of the body,  
51 such as hand-to-face contact.  
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57 Given the potential value that credible geometrical, fluid and mechanical  
58 modelling could provide, micro-scale materials characterisation would en-  
59 able a much greater understanding of the physical processes within the  
60

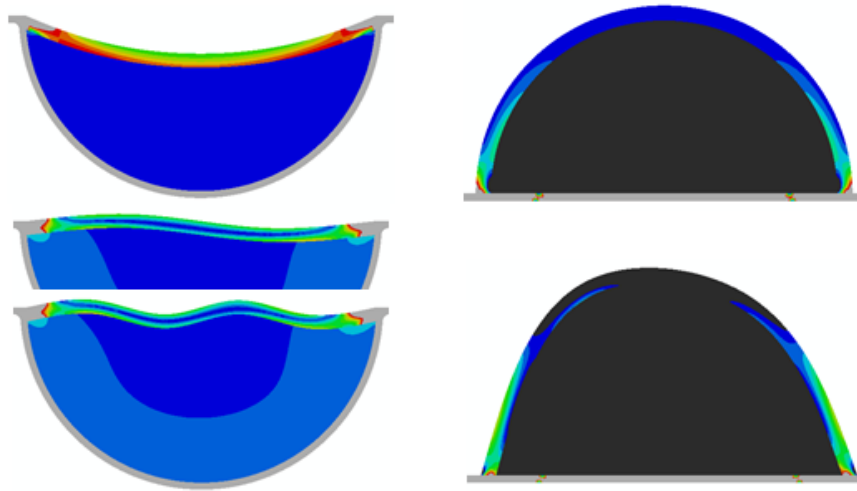


Figure 8: Illustration of skin stresses in shrinking droplets: for droplets in surface roughness grooves (left) and droplets on a smooth surface (right). Notice that skin undulation or asymmetry potentially mitigates against the surface tearing mechanism.

droplet, how the droplet is transferred, and how this influences the survivability of the virus within the droplet.

#### B.4 Self-inoculation via hand-to-face contacts

For an individual to get infected after contact with a contaminated surface, self-inoculation with a sufficient dose of the virus must occur. Typical hand-to-nose contact frequency occur about  $2.5 \pm 2.2$ /hour (Wilson *et al.*, 2020b). A study by Nicas & Best (2008) reports observed frequency of contact between individuals' hands and mucous membranes (eyes, lips and nostrils) and a model to quantify the risk of infection taking into account pathogen pick-up, inactivation on the hands with time and frequency of contact between hands and mucous membranes. The data reported are based on seasonal Influenza A virus.

Zhang & Li (2018) report a detailed study of transmission of Influenza A in a student office through person-to-person contact and surface touch behaviour. The authors consider airborne and fomite-mediated transmission pathways and report detailed data about surfaces and individuals involved including close contact (enabling transmission through large droplets) and surface touch observations. Based on a mathematical model describing both the aerosol and surface transmission pathways, the authors conclude that 4.2% of risk of infection by Influenza A is due to transmission via fomites. On the other hand, in the office 95.1% of viruses were transmitted via private surfaces and only 4.9% via public surfaces with desks being the most contaminated due to virus-laden droplets generated while talk-

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3 ing, sneezing and coughing being deposited on desks (including computer  
4 keyboards, mice, etc. located on them).  
5

6 It has recently been suggested that transmission through contact between  
7 a contaminated hand and the eye may not be the preferred way of infection  
8 by SARS-CoV-2 (Liu & Sun, 2020).  
9  
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## 11 12 **B.5 Cleaning and disinfection**

### 13 **B.5.1 What is an effective detergent?**

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16 Soap and water has been widely recommended to remove the SARS-CoV-  
17 2 virus from surfaces and skin. Chaudhary *et al.* (2020) investigates the  
18 mechanisms employed by the surfactants found in soap to disrupt the virus  
19 and conclude that soap and water is effective against the virus. However,  
20 Chin *et al.* (2020) implies that hand soap solution cannot inactivate the  
21 SARS-CoV-2 virus very well; that infectious virus can be found after 5  
22 minutes of incubating in hand soap solution. However, they have not  
23 simulated hand washing and have simply added the virus culture to hand  
24 soap solution. The physical removal of the virus trapped in soap micelle  
25 while rinsing hands has thus not been simulated in this study.  
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### 32 **B.5.2 What is an effective disinfectant?**

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35 Kampf *et al.* (2020) reports that the following substances can readily  
36 inactivate the infectivity of coronaviruses (by  $4\log_{10}$  or more): Ethanol  
37 (78%–95%), 2-propanol (70%–100%), 2-propanol (45%) with 1-propanol  
38 (30%), glutardialdehyde (0.5%–2.5%), formaldehyde (0.7%–1%) and povi-  
39 done iodine (0.23%–7.5%).  
40  
41

42  
43 Quaternary ammonium compounds (QAC) are known to be effective against  
44 both enveloped and non-enveloped viruses and as such are a common addi-  
45 tive to disinfectants. Although they exhibit a residual biocidal effect over  
46 2 to 4 hours after application which has been shown to be effective against  
47 norovirus, it is unknown whether enveloped coronaviruses are susceptible  
48 at these lower concentrations. Sodium hypochlorite requires a concentra-  
49 tion of at least 0.2%, whilst hydrogen peroxide requires a concentration of  
50 at least 0.5% and left incubating for at least 1 minute. Chlorhexidine diglu-  
51 conate has been found to be ineffective against coronaviruses (Kampf *et al.*,  
52 2020). It has been highlighted that SARS-CoV-2 is an enveloped virus and  
53 therefore can be less resistant to disinfection than non-enveloped viruses  
54 (Glasbey & Whiteley, 2020). The United States Environmental Protection  
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3 Agency has listed substances [see List N in United States Environmental  
4 Protection Agency (2020)] that are potentially effective against SARS-  
5 CoV-2 (Glasbey & Whiteley, 2020). Neither the substances in this list nor  
6 the substances in Kampf *et al.* (2020) have been experimentally proven  
7 to be effective against SARS-CoV-2, although they have been proven for  
8 other strains of coronaviruses. Appropriate procedure should begin with  
9 cleaning with soap and warm water followed by application of a disinfectant  
10 in the above category.  
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### 16 **B.5.3 Light deactivation**

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18 Dietz *et al.* (2020) reports that the impact of sunlight and UV light on the  
19 stability and decay of SARS-CoV-2 has not yet been investigated. UV-C  
20 light has been proven to inactivate other strains of coronaviruses within 10  
21 minutes or less (Bedell *et al.*, 2016). However, authors of this study state  
22 that UV-C light is best employed alongside regular cleaning of surfaces,  
23 and use of light cannot simply substitute effective cleaning practices. These  
24 measures also require thorough risk assessment and mitigation to ensure  
25 that occupants' health is not compromised and exposure is minimised.  
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## 32 **B.6 Science behind current UK government advice**

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34 The UK government guidelines for cleaning of surfaces that are currently  
35 available do cite few scientific articles regarding fomite transmission and  
36 we can therefore conclude that this pathway is thought to be less significant  
37 than other transmission pathways.  
38  
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40 The current government advice on cleaning in non-healthcare settings is to  
41 wipe hard surfaces with warm soapy water before applying detergent. The  
42 guidelines lack definitions for both substances and only suggest chlorine-  
43 based disinfectants. In this document, we have provided a list of poten-  
44 tially effective detergents and disinfectants (mostly alcohol-based) that  
45 have been verified by experiments on other strains of coronaviruses.  
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50 Waste that is suspected to be contaminated by COVID-19 should be stored  
51 for 72 hours before disposal. This quarantine period also applies to re-  
52 turned goods in shops and clothes that have been tried on. We assume  
53 that this duration is based on the results by van Doremalen *et al.* (2020)  
54 which suggest that SARS-CoV-2 can remain active for 72 hours (see §B.2  
55 on Factors affecting survival of virus on surfaces). We are unable to find  
56 any guidelines on parcels.  
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3 Working safely during coronavirus (COVID-19) (link) contains sector-  
4 specific guidelines, including those for transport, schools and childcare set-  
5 tings. These sectors have all been asked to clean ‘regularly and frequently’—  
6 frequencies have not been quantified.  
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## 10 B.7 Conclusions and key questions

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12 While there is still a lack of detailed and complete information regard-  
13 ing the behaviour of SARS-CoV-2 virus on surfaces, experimental studies  
14 have confirmed that certain environmental conditions (such as high tem-  
15 perature and humidity) can reduce the stability of the virus, thus reducing  
16 infectivity. Furthermore, some studies have shown which disinfectants are  
17 potentially more effective against the virus than others, what surfaces are  
18 typically infected, and for how long the virus remains on potentially infec-  
19 tious on surfaces.  
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25 There is a lack of clarity in the UK government’s advice on what cleaning  
26 products and methods are effective against the virus and which surfaces  
27 should be cleaned more frequently. In order to accurately advise the pub-  
28 lic, as well as operators of key infrastructure, such as public transport and  
29 hospitals, it is important to combine these experimental results with mod-  
30 els that can be used to inform decision-making. In order to build such  
31 models, information regarding the types of deposits on surfaces, the con-  
32 centration of the virus in these deposits, which surfaces are handled more  
33 frequently, as well as dose response curves are vital.  
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## 40 C UVGI and COVID-19

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43 With the emergence of COVID-19 and the subsequent global pandemic  
44 there has been considerable interest in the use of UV light to disinfect  
45 blood plasma (Ragan *et al.*, 2020; Keil *et al.*, 2020; Eickmann *et al.*,  
46 2020), medical equipment (Heimbuch & Harnish, 2019; Hamzavi *et al.*,  
47 2020; Card *et al.*, 2020; Derraik *et al.*, 2020) and air (Morawska *et al.*,  
48 2020), in the hope that this might reduce transmission of the disease. In  
49 particular, UVGI, a long established technology which utilizes UV-C light  
50 at wavelengths close to 254 nm (Reed, 2010), appears to have considerable  
51 potential as an intervention for inactivating SARS-CoV-2 (Inagaki *et al.*,  
52 2020; Beggs & Avital, 2020; Bianco *et al.*, 2020) and other pathogenic  
53 coronaviruses (Eickmann *et al.*, 2020; Heimbuch & Harnish, 2019; Duan  
54 *et al.*, 2003; Darnell *et al.*, 2004; Darnell & Taylor, 2006; Kariwa *et al.*,  
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3 2006; Bedell *et al.*, 2016).

4  
5 The biological impact of UV radiation is primarily due to the absorption  
6 of energetic photons by nucleic acids. DNA has an absorption spectrum  
7 which has a maximum in the 260 – 265 nm region, which rapidly declines  
8 thereafter as the wavelength increases (Harm, 1980). At these wavelengths  
9 photons of light are absorbed by nucleic acids, both DNA and RNA, to  
10 form dimers (fused base pairs) (Beggs, 2002) that impair the replication of  
11 viruses, inhibiting their ability to cause infection. Dimers involving both  
12 pyrimidines (cytosine and thymine) and purines (adenine and guanine) are  
13 the principal photoproducts formed, with pyrimidine dimers, particularly  
14 those involving thymine, generally predominating (Jagger, 1967). The for-  
15 mation of these dimers inhibits transcription of the viral genome, thus  
16 preventing synthesis of the necessary proteins required for the viral repli-  
17 cation. The number of bases in the viral genome is an important factor  
18 for determining sensitivity to UV damage, with longer genomes present-  
19 ing more target molecules, increasing the likelihood that the virus will be  
20 inactivated for a given level of UV exposure (Sagripanti & Lytle, 2020).  
21 As such, the SARS-CoV-2 virus, having a genome that is almost twice as  
22 long as the influenza viral genome, should in theory be more susceptible  
23 to UV damage than influenza. Having said this, because both are RNA  
24 viruses and do not have thymine in their genome, they will both tend to be  
25 less sensitive to UV damage compared to DNA viruses of similar genomic  
26 length.  
27  
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29 While most work that has been undertaken on UVGI has involved low-  
30 pressure UV lamps with a strong UV-C spectral emission at 253.7 nm, in  
31 recent years devices that produce light at other wavelengths have also been  
32 investigated. Specifically, deep-UV (DUV) light emitting diodes (LEDs),  
33 which emit light at around 280 nm on the boundary between UV-B and  
34 UV-C light, have been evaluated. With specific reference to COVID-19,  
35 UV light from DUV-LEDs has been shown to inactivate the SARS-CoV-2  
36 virus in petri dishes (Inagaki *et al.*, 2020). Others have also demonstrated  
37 that UV-C light at 222 nm can be used effectively to inactivate coron-  
38 aviruses in aerosols (Buonanno *et al.*, 2020b).  
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### C.1 UVGI inactivation Kinetics

At any point in time the amount of viral inactivation (disinfection) achieved for a given UV radiant flux (irradiance) can be described using the following first order decay equation (McDevitt *et al.*, 2012)

$$N_t = N_0 \times e^{-ZEt}, \quad (3)$$

where  $N_0$  and  $N_t$  are the number of viable viral particles (virions) at time zero and  $t$  seconds respectively;  $Z$  is the UV susceptibility constant for the virus ( $\text{m}^2/\text{J}$ );  $E$  is the irradiation flux ( $\text{W}/\text{m}^2$ ); and  $t$  is time in seconds.

The UV irradiation dose received by the virus is given simply by

$$H = E \times t, \quad (4)$$

where  $H$  is the observed UV irradiation dose ( $\text{J}/\text{m}^2$ ).

By combining equations 3 and 4, and rearranging we can obtain an equation for  $Z$ , namely

$$Z = -\frac{1}{H} \times \ln\left(\frac{N_t}{N_0}\right) = -\frac{1}{H} \times \ln(f), \quad (5)$$

where:  $f$  is the survival fraction.

Because the relationship between the UV dose and the natural logarithm of the survival fraction is broadly linear for most viral species, it means that the behaviour of any given virus exposed to UV-C light can be succinctly described by the value of  $Z$ , irrespective of the actual UV dose applied. As such, for any given viral species, if the value of  $Z$  is known, then it should be possible to predict with reasonable accuracy how the virus will behave when exposed to a given UV-C dose in any context. Microbes that exhibit larger  $Z$  values are more susceptible to UV damage, whereas those with small  $Z$  values are more difficult to inactivate.

UV inactivation plots for most viral species tend to be straight lines, although some might exhibit a curve (Kariwa *et al.*, 2006). Notwithstanding this, the model described in equation 3 is still a good approximation for most viral species (McDevitt *et al.*, 2012) up until the point where the ‘target’ becomes saturated with UV photons. At this point, because all the virions have already been inactivated, increasing the UV dose further has no effect and so the linear relationship between UV dose and the log reduction becomes decoupled, with the result that the  $Z$  value no longer applies.

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3 Instead of quantifying UV inactivation in terms of survival fraction, many  
4 researchers, particularly those working in biology, describe the reduction  
5 in the microbial count in terms of log reduction, which can be converted  
6 to survival fraction as follows  
7  
8

$$9 \quad f = \frac{1}{10^A}, \quad (6)$$

10  
11  
12 where  $A$  is the  $\log_{10}$  reduction in the number of virions.  
13  
14

## 15 C.2 Susceptibility of SARS-CoV-2 to UVGI

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17 While a substantial amount of work has been undertaken on the UV irra-  
18 diation of coronaviruses in various contexts (Inagaki *et al.*, 2020; Bianco  
19 *et al.*, 2020; Buonanno *et al.*, 2020b; Walker & Ko, 2007), relatively little  
20 work has been done specifically on the SARS-CoV-2 virus, and that which  
21 has been done has focused solely on the irradiation of the virus on surfaces  
22 (Signify, 2020), in blood (Ragan *et al.*, 2020; Keil *et al.*, 2020) or in liquids  
23 (Inagaki *et al.*, 2020; Bianco *et al.*, 2020). However, from the work that  
24 has been done to date, a clear and consistent picture emerges, namely that  
25 in comparison with SARS-CoV-1 and MERS-CoV, the SARS-CoV-2 virus  
26 appears to be relatively easy to inactivate with UV-C light (Beggs & Avi-  
27 tal, 2020). This is clearly illustrated in table 3, which shows the calculated  
28  $Z$  values for various UV-C (and deep-UV at 280 nm) irradiation experi-  
29 ments involving SARS-CoV-1, MERS-CoV and SARS-CoV-2 suspended  
30 in liquids. From this, it can be seen that the adjusted mean  $Z$  for SARS-  
31 CoV-1 was 0.00489 (SD = 0.00611)  $\text{m}^2/\text{J}$ , similar to that for MERS-CoV  
32 ( $Z$  value = 0.00104  $\text{m}^2/\text{J}$ ), whereas by comparison SARS-CoV-2 appears  
33 much more susceptible to UV damage at 254 nm (adjusted mean  $Z$  =  
34 0.14141 (SD = 0.09045)  $\text{m}^2/\text{J}$ ) and 280 nm (mean  $Z$  = 0.03684  $\text{m}^2/\text{J}$ ).  
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48 While no irradiation experiments have yet been undertaken on SARS-  
49 CoV-2 virions in air, a few aerosol experiments have been done using  
50 other related coronaviruses. The results of these experiments, together  
51 with other selected viruses for comparison purposes, are listed in table 4.  
52 These reveal that compared to influenza A (mean  $Z$  = 0.19435  $\text{m}^2/\text{J}$ ), the  
53 coronaviruses ( $Z$  = 0.377  $\text{m}^2/\text{J}$  for murine (mouse) hepatitis virus (MHV)  
54 coronavirus (Walker & Ko, 2007); 0.410  $\text{m}^2/\text{J}$  for human coronavirus 229E  
55 (Buonanno *et al.*, 2020b); and 0.590  $\text{m}^2/\text{J}$  for human coronavirus OC43  
56 (Buonanno *et al.*, 2020b)) are much more susceptible to UV damage, which  
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Virus	UV-C dose (mJ/cm <sup>2</sup> )	Inactivation (log reduction)	UV susceptibility constant , Z (m <sup>2</sup> /J)	Reference
SARS-CoV-1	>81	> log 0.602	0.00171	Duan et al. (Duan <i>et al.</i> , 2003)
SARS-CoV-1	241	log 1.4*	0.00134*	Darnell et al. (Darnell <i>et al.</i> , 2004)
SARS-CoV-1	1446	log 4.5*	0.00072*	Darnell et al. (Darnell <i>et al.</i> , 2004)
SARS-CoV-1	4819	log 4.1*	0.00020*	Darnell & Taylor (Darnell & Taylor, 2006)
SARS-CoV-1	40	log 3.2*	0.01833*	Kariwa et al. (Kariwa <i>et al.</i> , 2006)
SARS-CoV-1	121	log 5.325	0.01017	Kariwa et al. (Kariwa <i>et al.</i> , 2006)
SARS-CoV-1	1000	≥ log 4.81	0.00111	Heimbuch & Harnish (Heimbuch & Harnish, 2019)
SARS-CoV-1	50	log 3.05	0.01405	Eickmann et al. (Eickmann <i>et al.</i> , 2020)
SARS-CoV-1	100	≥ log 3.5	0.00806	Eickmann et al. (Eickmann <i>et al.</i> , 2020)
MERS-CoV	1000	≥ log 4.5	0.00104	Heimbuch & Harnish (Heimbuch & Harnish, 2019)
SARS-CoV-2	3.7	log 3.3	0.20536	Bianco et al. (Bianco <i>et al.</i> , 2020)
SARS-CoV-2	5	log 2.0	0.09210	Signify (Signify, 2020)
SARS-CoV-2	22	log 6.0	0.06280	Signify (Signify, 2020)
SARS-CoV-2	3.75**	log 0.9	0.05526	Inagaki et al. (Inagaki <i>et al.</i> , 2020)
SARS-CoV-2	37.5**	log 3.0	0.01842	Inagaki et al. (Inagaki <i>et al.</i> , 2020)

Table 3: Calculated  $Z$  values for the UV-C (254 nm) and deep-UV (280 nm) irradiation experiments involving coronaviruses suspended in liquids (Beggs & Avital, 2020).

\* Estimated from plots and data presented in source material.

\*\* Using deep-UV light at 280 nm (all other experiments performed using UV-C light at 254 nm).

Researchers	Virus	UV-C wavelength	Effective $Z$ value ( $\text{m}^2/\text{J}$ )	Reporter
Jensen (Jensen, 1964)	Adenovirus	254 nm	0.0546	Kowalski et al. (Kowalski <i>et al.</i> , 2000)
Jensen (Jensen, 1964)	Coxsackie B-1	254 nm	0.1108	Kowalski et al. (Kowalski <i>et al.</i> , 2000)
Jensen (Jensen, 1964)	Influenza A	254 nm	0.1187	Kowalski et al. (Kowalski <i>et al.</i> , 2000)
Jensen (Jensen, 1964)	Sindbis virus	254 nm	0.1040	Kowalski (Kowalski, 2009)
(Jensen, 1964)	Vaccinia virus	254 nm	0.1528	Kowalski et al. (Kowalski <i>et al.</i> , 2000)
Walker & Ko (Walker & Ko, 2007)	Adenovirus	254 nm	0.0390	Walker & Ko (Walker & Ko, 2007)
Walker & Ko (Walker & Ko, 2007)	MHV coronavirus	254 nm	0.3770	Walker & Ko (Walker & Ko, 2007)
McDevitt et al. (McDevitt <i>et al.</i> , 2012)	Influenza A	254 nm	0.2700	McDevitt et al. (McDevitt <i>et al.</i> , 2012)
McDevitt et al. (McDevitt <i>et al.</i> , 2007)	Vaccinia virus	254 nm	2.5400	McDevitt et al. (McDevitt <i>et al.</i> , 2007)
Buonanno et al. (Buonanno <i>et al.</i> , 2020b)	Human coronavirus 229E	222 nm	0.4100	Buonanno et al. (Buonanno <i>et al.</i> , 2020b)
Buonanno et al. (Buonanno <i>et al.</i> , 2020b)	Human coronavirus OC43	222 nm	0.5900	Buonanno et al. (Buonanno <i>et al.</i> , 2020b)

Table 4: Summary of reported effective  $Z$  values for single-pass UV-C irradiation experiments performed on selected aerosolised viruses in air (Beggs & Avital, 2020)

is perhaps only to be expected given that they have a genome that is approximately twice the length of the influenza virus (Sagripanti & Lytle, 2020). Importantly, the coronavirus  $Z$  values are an order of magnitude greater than those obtained for SARS-CoV-2 in liquid, implying that when aerosolised, coronaviruses in general and SARS-CoV-2 in particular, are much easier to disinfect compared with when they are presented in liquids or on surfaces. This however is to be expected as the medium in which microbes are irradiated greatly influences the magnitude of the  $Z$  value achieved. This is because UV-C light is attenuated, due to scattering and absorption (Gregory, 2005), as it passes through liquids (Mamane, 2008). Consequently, viruses are generally easier to disinfect in the air compared with when they are on surfaces or in liquids.

Collectively, the results presented above strongly suggest that the SARS-



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3 CoV-2 virus is relatively easily inactivated by UV-C light and that when  
4 aerosolised the virus is likely to exhibit a UV susceptibility constant,  $Z$ ,  
5 that is similar in magnitude to other coronaviruses in air. As such, this  
6 indicates that SARS-CoV-2, when suspended in air, should be reasonably  
7 easy to inactivate using UV light at 254 nm. As such, UVGI air disinfection  
8 applications appear to have potential as an intervention to inhibit the  
9 transmission of COVID-19 in buildings and other enclosed spaces.  
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### 15 C.3 UVGI air disinfection applications

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17 While it is clear from the discussion above that the SARS-CoV-2 virus  
18 can be relatively easily inactivated using UV-C light, this does not nec-  
19 essarily mean that installing UVGI air disinfection in buildings or other  
20 enclosed spaces (e.g. passenger vehicle, trains, buses, etc.) will prevent  
21 the transmission of COVID-19 in these facilities. This is because, in order  
22 to break the chain of COVID-19 transmission, UVGI needs to be applied  
23 in a manner that: (i) is appropriate to the situation; (ii) targets the route  
24 of transmission; and (iii) disinfects enough air to be effective. Too often do  
25 building owners and occupiers, seduced by adverts claiming a '99.9% kill'  
26 against an infectious pathogen, install expensive UVGI room air cleaners  
27 in their facilities, only to find that they offer little or no protection at all.  
28 This occurs because the impressive claims made by manufacturers often  
29 relate to single-pass microbiological tests (which may or may not involve  
30 relevant microbial species) rather than addressing how the device will ac-  
31 tually perform in a given room space. So for example, a UVGI room air  
32 cleaner may inactivate (kill) every microbe that passes through it, but if  
33 the air flow rate through the device is small, then it will have little im-  
34 pact on a large, well ventilated, room space. Therefore, when considering  
35 UVGI air disinfection it is important to evaluate its likely performance  
36 in the context of the room space, something which will inevitably involve  
37 evaluation of room occupancy levels and the geometry of the room space,  
38 as well as consideration of the ventilation system.  
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50 UVGI air disinfection devices can be broadly classified into:

- 51 • **Upper-room UVGI systems** in which an open UV-C irradiation  
52 field above the heads of room occupants is used to disinfect aerosolised  
53 bacteria and viruses suspended in the air. Because UV-C light is harm-  
54 ful to humans, such systems utilize louvres and baffles that obscure  
55 the UV lamps from eyesight so that the room occupants are kept safe.  
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57 • **In-duct UVGI systems** which utilise UV-C lamps mounted in the  
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3 return or supply air ducts of mechanical HVAC systems to disinfect  
4 the air either to or from the room space.  
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- 6 • **UVGI room air cleaners** which are located within the room space  
7 and employ UV-C lamps mounted in a container with a fan. These  
8 can vary in size, but only disinfect the air that passes through the  
9 device.  
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13 A detailed discussion of each of the above UVGI variants follows in the  
14 sections below, so here we will restrict ourselves to a broad discussion  
15 of some general principles that are applicable to all types of UVGI air  
16 disinfection system.  
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### 20 C.3.1 Target microbes

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22 Unlike air filters, which capture all particulates of a given size irrespective  
23 of their biological status, UVGI air disinfection systems target specific mi-  
24 crobes. They do not clean the air; rather they use biophysical mechanisms  
25 to inactivate target viral and bacterial species, which once inactivated re-  
26 main in the air stream. Target microbes are generally viral or bacterial  
27 species that cause infectious disease. So in the case of COVID-19, the  
28 target microbe is the SARS-CoV-2 virus. So when sizing a UVGI air dis-  
29 infection system to inhibit the transmission of COVID-19, it is necessary  
30 to ensure that any SARS-CoV-2 virions in the air will receive a lethal dose  
31 of UV-C light, since a sub-lethal dose may leave some virions infectious.  
32 In order to calculate the necessary lethal dose to be administered, it is  
33 first necessary to use the appropriate  $Z$  value for the target microbe in an  
34 aerosolised state, and then, if possible, add a factor of safety in order to  
35 ensure that the installation will adequately protect the room occupants.  
36 In the case of COVID-19, although the precise  $Z$  value for SARS-CoV-2  
37 in air is not known,  $Z$  values for three other closely related coronaviruses  
38 in air have been determined (Buonanno *et al.*, 2020b; Walker & Ko, 2007).  
39 Of these, the  $Z$  value (0.377 m<sup>2</sup>/J) for MHV coronavirus (Walker & Ko,  
40 2007) is the lowest value, implying that this is the most hardy of the three.  
41 It is therefore suggested that this is probably a good candidate for the tar-  
42 get  $Z$  value of SARS-CoV-2 in air (Beggs & Avital, 2020) as it is likely to  
43 be a conservative value.  
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### 56 C.3.2 Safety

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58 UV-C light is highly biologically active and as such can cause damage to  
59 humans. Furthermore, although UV light is far more energetic than the  
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3 visible portion of the electromagnetic spectrum, it is invisible to humans,  
4 and so damage can occur without the individuals concerned noticing. Ex-  
5 posure to UV light can result in transient corneal inflammation (*photoker-*  
6 *atitis*) in the eye (Cullen, 2002), which may go unnoticed, but may progress  
7 to include inflammation of the conjunctiva (*photoconjunctivitis*) (Grifoni  
8 *et al.*, 2005). Acute exposure to UV-C radiation can cause more severe  
9 corneal damage (Cullen, 2002), which generally abates after several days,  
10 leaving no permanent damage (Cullen, 2002), although there have been  
11 reports of clinical symptoms persisting for as much as two years after an  
12 acute UV injury (Zaffina *et al.*, 2012). For this reason, it is important  
13 to ensure that UV-C lamps are completely hidden from the view of room  
14 occupants, either through total enclosure or the use of louvres or shields.  
15 In addition, in order to protect the eyes, UV-C safety goggles should be  
16 worn when undertaking maintenance work on UV systems.  
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24 UV-C light can also cause cutaneous damage (erythema) (Harrison &  
25 Young, 2002), resulting in reddening of the skin akin to sunburn. In this  
26 respect, UV-C light is actually less effective than UV-B at causing ery-  
27 thema because it has a lower penetration capability. More importantly,  
28 because prolonged exposure to UV light is associated with cancer, doubts  
29 have been raised about the safety of upper-room UVGI air disinfection  
30 systems due to the risk of exposure to reflected UV-C light. However,  
31 the International Commission on Illumination (CIE) review of the sub-  
32 ject found that upper-room UVGI air disinfection could be safely used  
33 without significant risk for long-term delayed effects such as skin cancer  
34 (Commission Internationale de L'Eclairage, 2010).  
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### 42 C.3.3 What UVGI can and cannot do

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44 Irrespective of the specific type of UVGI air disinfection used, it is im-  
45 portant to be aware of the limitations of the technology. While UVGI air  
46 disinfection can help to reduce the airborne (aerosol) transmission of infec-  
47 tious diseases, it cannot protect against close range droplet transmission.  
48 So in the context of COVID-19, this means that UVGI air disinfection  
49 cannot protect an individual if, for example, an infected person coughs di-  
50 rectly in their face. However, if upper-room UVGI is applied, it can protect  
51 individuals from the many hundreds of smaller aerosol particles (generally  
52  $<50 \mu\text{m}$  in diameter) that are exhaled by room occupants, which can be  
53 readily transported on room air currents (Beggs, 2020). In addition, when  
54 mounted in return air ductwork, UVGI lamps can also protect against  
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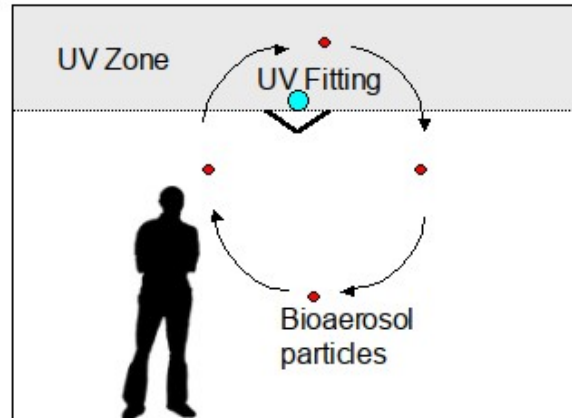


Figure 9: An upper-room UVGI air disinfection installation

infectious aerosol particles being recirculated back into the room spaces through the mechanical ventilation system, if the system recirculates a portion of the air in order to save energy.

#### C.4 Upper-room UVGI air disinfection

Upper-room UVGI, as the name suggests, involves the creation of an open field UV-C irradiation field within the upper portion of a room as shown in figure 9. This can be done using either wall or pendant fittings mounted at high level similar to those shown in figure 10. Since exposure to UV-C light can be harmful, such devices are fitted with louvres or baffles, in order to prevent: (i) the occupants below from seeing the lamps; and (ii) the UV light from penetrating into the lower room space. The goal of upper room UVGI is to inactivate any infectious microbes (e.g. viruses and bacteria) that may pass through the UV zone and thus disinfect the air inhaled in the lower room space. As such, upper-room installations rely on natural convection currents, rather than fans, to carry aerosol particles through the UV field. By using the convection currents that occur naturally in room spaces it is possible to disinfect very large volumes of air relatively quickly (Miller *et al.*, 1999). As such, upper-room UVGI air disinfection is a well-established technology (First *et al.*, 1999*a,b*) that has proven effective as a public health intervention to prevent the spread of airborne diseases such as measles (Nardell & Nathavitharana, 2019) and tuberculosis (TB) in buildings (Escombe *et al.*, 2009; Noakes *et al.*, 2006).



Figure 10: Typical wall mounted upper-room UVGI air disinfection fitting.

	Wall-Mounted Fixtures	Wall-Mounted Fixtures	Ceiling-Mounted Fixtures	Ceiling-Mounted Fixtures
	Corner Mount	Wall Mount	Pendent	Pendent with Fan
Beam pattern	90°	180°	360°	360°
Minimum ceiling height	2.44 m	2.44 m	2.89 m	2.89 m
Fixture mounted height	2.1 m	2.1 m	2.4 m	2.4 m
Ideal UV-C intensity (flux) for effective disinfection	> 10 $\mu\text{W}/\text{cm}^2$	> 10 $\mu\text{W}/\text{cm}^2$	> 10 $\mu\text{W}/\text{cm}^2$	> 10 $\mu\text{W}/\text{cm}^2$

Table 5: Suggested installation summary for upper-room UVGI air disinfection (ASHRAE, 2019; Coker *et al.*, 2001)

#### C.4.1 Calculating the required UV flux

Despite first being used in the 1930s (Reed, 2010), the guidelines for designing upper-room UVGI systems are surprisingly sparse. This is primarily because the required UV-C radiant flux,  $E$ , is wholly dependent on the target microbe and the air movement characteristics of the room space, both of which may vary greatly depending on the application being considered. Also, because most UVGI air disinfection activity has focused on the eradication of TB (Coker *et al.*, 2001), the guidelines which exist tend to be written in the context of that disease and generally take a ‘rule-of-thumb’ approach. With respect to this, Table 5, which is taken from the ASHRAE UVGI air disinfection guidelines (ASHRAE, 2019), is typical of the genre.

While to date no guidelines exist regarding upper-room UVGI and COVID-19, Beggs and Avital recently produced a feasibility study (Beggs & Avital, 2020) to evaluate the potential efficacy of the technology in this context. This study included a methodology (presented below) for estimating the upper-room UV flux required to disinfect the SARS-CoV-2 virus in ventilated room spaces. This approach utilized the methodology described in Beggs and Sleight (Beggs & Sleight, 2002) and assumed that the room air is well mixed, which is a reasonable approximation for many applications (Beggs & Sleight, 2002). If the space is well mixed, then the average



particle residence time,  $t_{res}$ , (in seconds) in the room will be

$$t_{res} = \frac{1}{n} \times 3600, \quad (7)$$

where  $n$  is the room ventilation rate in air changes per hour (AC/hr).

From equation 7 it can be approximated that the average particle residence time in the upper-room UV field,  $t_{uw}$ , (in seconds) will be

$$t_{uw} = t_{res} \times \frac{h_{uw}}{h_r}, \quad (8)$$

where  $h_r$  is the floor-to-ceiling height (m), and  $h_{uw}$  is the depth of the upper-room UV zone (m).

Because  $Z$  values are often determined experimentally using microbes suspended in liquids or on surfaces, it may be necessary to adjust the  $Z$  value for use with upper-room UVGI systems (Beggs *et al.*, 2006; Yang *et al.*, 2017), as follows

$$Z_{ur} = Z \times c_{ur}, \quad (9)$$

where  $Z_{ur}$  is the effective upper-room  $Z$  value ( $\text{m}^2/\text{J}$ ), and  $c_{ur}$  is a correction coefficient.

$Z_{ur}$  can be assumed to be the same as the aerosol  $Z$  value for the target microbe, examples of which are presented in table 4.

So if we assume that the air in a room is well mixed, by combining equations 4, 5 and 8 it is possible to compute the average irradiation flux ( $\text{W}/\text{m}^2$ ),  $E_r$ , that is required to achieve a desired survival fraction,  $f_r$ .

$$E_r = \frac{1}{(Z_{ur} \times t_{uw})} \times \ln(f_r). \quad (10)$$

In which case, the average UV dose received ( $\text{J}/\text{m}^2$ ),  $H_r$ , is

$$H_r = E_r \times t_{uw}. \quad (11)$$

Alternatively, the disinfection achieved by an upper-room UVGI system can be thought of as being equivalent to additional air changes in the room space (McDevitt *et al.*, 2008). In this scenario, the UV rate constant,  $k_{uw}$ , which can be thought of as the equivalent air change rate per second, can be determined using (Beggs *et al.*, 2006), i.e.

$$k_{uw} = Z_{ur} \times E \times \frac{h_{uw}}{h_r}. \quad (12)$$

So in a ventilated room in which contamination ceases at time zero, we can utilize both the UV rate constant,  $k_{uv}$ , and a rate constant,  $k_v$ , for the ventilation (i.e.  $n \div 3600$ ), to produce a decay model for the room space

$$C_t = C_0 \times e^{(k_v + k_{uv} + k_d)t}, \quad (13)$$

where  $C_0$  and  $C_t$  are the concentrations of viable viral particles in the room space (virions/m<sup>3</sup>) at time zero and  $t$  seconds respectively;  $k_v$  is the ventilation rate constant;  $k_d$  is the particulate deposition rate constant (e.g. 0.0014 s<sup>-1</sup> (Stadnytskyi *et al.*, 2020)); and  $t$  is time in seconds.

Similarly, the following continuous contamination model represents the contaminant concentration in the room space,  $C_{uv}$ , under steady-state conditions

$$C_{uv} = \frac{q}{(k_v + k_{uv} + k_d) \times V}, \quad (14)$$

where  $V$  is the room volume (m<sup>3</sup>), and  $q$  is the steady-state room contamination rate (virions/s).

Using this simple approach, it is possible to estimate:

- The average UV-C flux ( $\mu\text{W}/\text{cm}^2$ ),  $E_r$ , required to achieve the desired level of inactivation in the target microbe.
- The average UV-C dose ( $\text{mJ}/\text{cm}^2$ ),  $H_r$ , required to achieve the desired level of inactivation in the target microbe.
- The equivalent air change rate per second,  $k_{uv}$ , that can be achieved by the UVGI installation with regard to the target microbe.

#### C.4.2 Upper-room UVGI feasibility study

Recently Beggs and Avital (Beggs & Avital, 2020) undertook a feasibility study to evaluate the potential efficacy of the upper-room UVGI air disinfection as a measure to prevent the transmission of COVID-19 in a ventilated room space. In this study equations 8 and 10 were used to estimate the average upper-room irradiation flux that would be required to achieve a 50 - 90% reduction in aerosolised SARS-CoV-2 virions (through the action of the UV-C alone) in a 4.2 × 4.2 × 2.5 m high room space for a range of ventilation rates. These dimensions were chosen because they are typical for an upper-room UVGI installation in which the lamp height is 2.1 m above the floor (First *et al.*, 1999b). In the model it was assumed that the air was completely mixed, which meant that according to equation 8, aerosol particles would spend on average 16% of their room residency time in the UV zone.

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3 In addition to computing the required UV flux, the performance of a  
4 standard upper-room UVGI fitting was evaluated against the challenge  
5 of SARS-CoV-2. This was done in accordance with the guidelines stated  
6 by First (First *et al.*, 1999b), in which it was assumed that the room con-  
7 tained a single 30 W (input) UV-C fitting capable of delivering an average  
8 upper-room flux of  $50 \mu\text{W}/\text{cm}^2$  (ASHRAE, 2019), with performance mod-  
9 elled in terms of equivalent ventilation rate using equation 12.  
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14 Because to date no UV irradiation experiments have been performed on  
15 aerosols containing the SARS-CoV-2 virus, in the feasibility study the  
16 value of  $Z_{ur}$  was taken to be  $0.377 \text{ m}^2/\text{J}$ , which was the value that Walker  
17 and Ko obtained for the MHV coronavirus in air (Walker & Ko, 2007).  
18 Because this was considered a conservative value, it was selected as a suit-  
19 able surrogate for SARS-CoV-2. In addition, because of the uncertainty  
20 associated with this assumed value, a 10-fold ‘factor of safety’ was intro-  
21 duced into the analysis by also modelling a worst-case scenario in which  
22  $Z_{ur}$  was  $0.0377 \text{ m}^2/\text{J}$ .  
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28 Table 6 presents the results of the room analysis using these two values for  
29  $Z_{ur}$ , for a range of ventilation rates. From this it can be seen that there  
30 is a direct inverse relationship between particle residence time in the UV  
31 field,  $t_{uv}$ , and the required irradiation flux,  $E_r$ , as predicted by equation  
32 10. This means that for any given  $Z$  value, the value of  $E_r$  will double  
33 as the room ventilation rate doubles. The table also reveals that there  
34 is a direct inverse relationship between  $Z_{ur}$  and  $E_r$ . From the calculated  
35 values in this table it can be seen that if  $Z_{ur} = 0.377 \text{ m}^2/\text{J}$ , then with  
36 an average UV flux of just  $10 \mu\text{W}/\text{cm}^2$  it should be possible to achieve  
37  $>90\%$  inactivation of SARS-CoV-2, even at a ventilation rate of 8 AC/h.  
38 However, if in reality,  $Z_{ur}$ , is  $0.0377 \text{ m}^2/\text{J}$ , then all the calculated fluxes  
39 would have to increase by a factor of ten to achieve the same results. Given  
40 that accepted guidelines (First *et al.*, 1999b) recommend for a room 2.5  
41 m high, one 30 W (input) UV lamp per  $18.58 \text{ m}^2$  of floor area, which  
42 will produce an average flux in the region  $50 \mu\text{W}/\text{cm}^2$ , this means that  
43 even under this worst-case scenario it should still be possible to achieve  
44 disinfection rates  $>90\%$  for all but the highest ventilation rates.  
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54 When the UV flux was fixed at an average of  $50 \mu\text{W}/\text{cm}^2$ , it was found  
55 that for  $Z_{ur} = 0.377 \text{ m}^2/\text{J}$  the upper-room UVGI installation produced an  
56 equivalent air change rate of 108.6 AC/h, whereas if  $Z_{ur} = 0.0377 \text{ m}^2/\text{J}$   
57 this fell to 10.9 AC/h. These values were constant and unaffected by the  
58 actual room ventilation rate.  
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Ventilation rate (AC/h)	Average particle residence time in UV field. (mins.)	UV susceptibility constant, $Z_{ur}$ ( $m^2/J$ )	Average irradiance required for 50% inactivation ( $\mu W/cm^2$ )	Average irradiance required for 70% inactivation ( $\mu W/cm^2$ )	Average irradiance required for 90% inactivation ( $\mu W/cm^2$ )
1	9.6	0.3770	0.319	0.554	1.060
2	4.8	0.3770	0.638	1.109	2.121
4	2.4	0.3770	1.277	2.218	4.241
6	1.6	0.3770	1.915	3.327	6.362
8	1.2	0.3770	2.554	4.436	8.482
1	9.6	0.0377	3.192	5.544	10.604
2	4.8	0.0377	6.384	11.088	21.207
4	2.4	0.0377	12.768	22.177	42.414
6	1.6	0.0377	19.152	33.266	63.621
8	1.2	0.0377	25.536	44.355	84.829

Table 6: Predicted average upper-room UV irradiance fluxes required to achieve 50%, 70% and 90% inactivation for SARS-CoV-2 assuming a range of  $Z_{ur}$  values and ventilation rates. (Assuming  $Z_{ur} = 0.377$  or  $0.0377 m^2/J$ ) (Beggs & Avital, 2020)

Collectively, the results from the feasibility study suggest that upper-room UVGI may have considerable potential as an intervention against the transmission of COVID-19 in buildings, especially in situations where achieving high ventilation rates might otherwise be impractical.

#### C.4.3 Room air movement and particle decoupling

While the results of the feasibility study are encouraging, it is important to remember that, unlike TB which is spread via the inhalation of droplet nuclei  $<5-10 \mu m$  in diameter, COVID-19 can be transmitted through the exhalation of larger respiratory droplets  $<100 \mu m$ , which rapidly reduce in size due to evaporation (Beggs, 2020; Xie *et al.*, 2007; Liu *et al.*, 2017) to become aerosols say  $<50 \mu m$  in diameter (Nicas *et al.*, 2005). These aerosol particles have settling velocities  $<0.1 m/s$  and as such can readily be transported on convective room air currents, with the result that they can remain suspended in room air for many minutes. However, if the velocities of the convection currents drop, then some of the larger aerosol particles may decouple from the air stream and settle out due to gravitational deposition, potentially passing through the breathing zone where they can be inhaled by the room occupants. This is particularly the case if the air is poorly mixed, and stagnant regions exist within the room space. Under such circumstances larger aerosol particles may be inhaled without being fully irradiated by the upper-room UV field, undermining the effectiveness of the whole UVGI installation. Consequently, if upper-room UVGI is to be effective against COVID-19, it is important both to promote

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3 good room air mixing and also to ensure that larger aerosol particles (e.g.  
4 10-50  $\mu\text{m}$  in diameter) receive a lethal UV irradiation dose. As such, this  
5 may require upper-room UVGI systems to be supplemented with ceiling  
6 mounted fans (Zhu *et al.*, 2013) or other devices to promote the neces-  
7 sary air movement to ensure that larger aerosol particles are adequately  
8 irradiated.  
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12 Upper-room UVGI air disinfection is highly dependent on good air mix-  
13 ing occurring between the upper and lower portions of the room space  
14 (Beggs & Sleigh, 2002; Noakes *et al.*, 2004a; Nicas & Miller, 1999). In  
15 the feasibility study it was assumed that complete mixing occurred, which  
16 although a reasonable approximation in many instances, is not always the  
17 case because short circuiting can occur (Beggs & Sleigh, 2002). If the  
18 room air mixing factor, which describes the inter-zonal air flow rate rela-  
19 tive to the absolute room ventilation rate, is low, say for example due to  
20 stratification in a poorly ventilated space, then this can greatly impair the  
21 disinfection performance of an upper-room UVGI system (Beggs & Sleigh,  
22 2002; Noakes *et al.*, 2004a). It is therefore important when designing such  
23 systems to carefully consider the air movement in the room space, in order  
24 to eliminate stagnant regions and maximise air movement through the UV  
25 field. With this in mind analysis can be performed using either zonal mod-  
26 els (Noakes *et al.*, 2004a,b) or computational fluid dynamics (CFD) (Zhu  
27 *et al.*, 2013; Gilkeson & Noakes, 2013). Zonal models are relatively simple  
28 and easy to use, but limited in scope (Noakes *et al.*, 2004b), whereas CFD  
29 is much more flexible and comprehensive, but is computationally expen-  
30 sive, requiring specialist skills and software to execute any models that are  
31 constructed.  
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#### 44 C.5 In-duct UVGI air disinfection

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46 Many older commercial HVAC and mechanical ventilation systems recircu-  
47 late a portion (in the region 50 – 80%) of the room air in order to save en-  
48 ergy. In the context of the COVID-19 pandemic this strategy is potentially  
49 extremely hazardous because it means that aerosol particles containing the  
50 SARS-CoV-2 virus extracted from one location in a building may become  
51 widely distributed throughout the whole building by the mechanical (re-  
52 circulating) ventilation system. If this happens, then the HVAC system  
53 could in effect become a pathogen distribution system. However, although  
54 in theory infectious aerosol particles can be dispersed around buildings by  
55 this route, the clinical evidence in support of this opinion is somewhat  
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3 lacking, with for example, no evidence found to link the outbreak that  
4 occurred on the Diamond Princess cruise ship in January 2020 with the  
5 ship's central air conditioning system (Xu *et al.*, 2020). Nevertheless, the  
6 UK *Health and Safety Executive* (HSE) recommends: “*You can continue*  
7 *using most types of air conditioning system as normal. But, if you use a*  
8 *centralised ventilation system that removes and circulates air to different*  
9 *rooms it is recommended that you turn off recirculation and use a fresh air*  
10 *supply.*” (Health and Safety Executive, 2020) and the *World Health Organization* (WHO) states: “*For mechanical systems, increase the percentage of*  
11 *outdoor air, using economizer modes of HVAC operations and potentially*  
12 *as high as 100%.*” (World Health Organization, 2020b) While there may be  
13 controversy as to whether or not recirculating HVAC systems contribute  
14 to the spread of COVID-19, there is strong clinical evidence implicating  
15 recirculated air in the transmission of TB, with an outbreak on a US naval  
16 vessel attributed to a recirculating mechanical ventilation system, in which  
17 a single undiagnosed index case managed to infect 140 out of 308 crew  
18 members (Houk, 1980). Given the evidence from this case and the general  
19 lack of knowledge surrounding COVID-19 transmission in buildings, it is  
20 therefore entirely understandable that the public health authorities might  
21 wish to adopt a precautionary approach in relation to the recirculation of  
22 return air.  
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35 Notwithstanding the above discussion, while it is possible in the UK to op-  
36 erate HVAC systems using 100% outside air during the summer months,  
37 the situation is very different in the winter months when temperatures  
38 drop. This is because the heating coils in the air handling units (AHUs)  
39 in many installations do not have enough power to enable the supply air  
40 to be heated from, for example,  $-1^{\circ}\text{C}$  to say  $28^{\circ}\text{C}$ . Consequently, if 100%  
41 outside air is supplied, then during periods in which the weather is cold,  
42 many buildings might become uncomfortable and potentially uninhabit-  
43 able. Consequently, making such buildings both COVID-19 ‘safe’ and  
44 also comfortable is a major challenge. However, it is this precise chal-  
45 lenge to which in-duct UVGI air disinfection is particularly well suited.  
46 By installing UVGI lamps in the return air duct-work it is possible to  
47 irradiate the target microbes and inactivate them before they are recir-  
48 culated back into the room space. In so doing, the UVGI lamps can  
49 in theory protect both the room occupants and the AHU from cross-  
50 infection/contamination, while still enabling the system to recirculate re-  
51 turn air. So in the context of the COVID-19 pandemic, in-duct UVGI  
52 might be technology that could be retrofitted into commercial HVAC sys-  
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3 tems in order to enable buildings to become fully operational during the  
4 winter months.  
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6 In-duct UVGI air disinfection is also applicable to HVAC systems where  
7 thermal wheels are installed to recover waste heat. While these systems  
8 negate the need for the recirculation of return air, there is still the risk  
9 of cross-contamination with the SARS-CoV-2 virus because the wheel re-  
10 volves between the two air streams. Employing UVGI lamps in the return  
11 air stream before the wheel potentially might prevent this from happening.  
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#### 16 17 **C.5.1 Where should in-duct UVGI lamps be placed?**

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19 In the context of the transmission of airborne disease (e.g. TB), while  
20 UVGI lamps can be placed in either the return or supply air ducts, it is  
21 generally preferable to install the lamps on the return air side of the AHU.  
22 This is because in this position, the UV field not only protects against  
23 pathogens being recirculated, but also prevents the up-stream duct-work,  
24 filters and coils from becoming contaminated. This is particularly impor-  
25 tant in the context of COVID-19 because coronaviruses can remain viable  
26 on surfaces for several days (van Doremalen *et al.*, 2020) and therefore may  
27 pose a threat to staff performing maintenance duties. In addition, mount-  
28 ing the lamps on the return air duct before the AHU will also protect  
29 anyone in the vicinity of the system exhaust air outlet.  
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#### 36 37 **C.5.2 Failing safe**

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39 With all UVGI systems it is important that they ‘fail safe’, so that building  
40 occupants are protected should the lamps fail. For this reason ASHRAE  
41 recommend that in-duct UVGI should “*always be used in combination with*  
42 *proper filtration*” (ASHRAE, 2019). In the context of COVID-19, this  
43 presents a tricky problem. Because COVID-19 is caused by a virus, this  
44 would necessitate the installation of HEPA filters, which would completely  
45 undermine the reason for retrofitting UVGI, namely to protect the building  
46 occupants while keeping fan energy consumption low. Therefore, workable  
47 solutions need to be developed to overcome this problem.  
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#### 53 54 **C.5.3 Computation of the UV flux**

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56 While in theory the UV-C dose received by a microbe passing through a  
57 UV field can be easily computed using equation 4, in practice with in-duct  
58 systems, computation of the dose received can be complex. Because the air  
59 velocities in mechanical ventilation ducts are relatively high, generally in  
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3 the region 3-6 m/s, it means that residence times in the UV field are very  
4 short, generally  $<1$  s. This means that in order to administer a lethal dose  
5 the UV flux has to be very large, often requiring multiple high-powered  
6 lamps. Consequently, without knowing the precise arrangement of the  
7 lamps and access to numerical modelling techniques it is difficult to com-  
8 pute the dose that will be received by a target microbe. The situation is  
9 further complicated by the fact that if the UV flux is not evenly distributed  
10 across the duct, microbes passing through the periphery of the field may  
11 not receive a lethal dose. If this occurs, the microbe may remain infectious,  
12 undermining the efficacy of the whole installation. As such, in order to  
13 properly evaluate how a given in-duct UVGI air disinfection installation  
14 will perform, it is necessary to employ CFD analysis (Capetillo *et al.*, 2015,  
15 2017; Yang *et al.*, 2018). Although a rough estimate can be obtained of  
16 how a proposed in-duct UVGI installation might perform, using standard  
17 mathematical techniques (Beggs *et al.*, 2000), without CFD or some other  
18 advanced numerical modelling technique, it is not possible to be fully con-  
19 fident about any predictions made, unless of course experimental work is  
20 carried out.

21  
22 Given the complexity associated with designing in-duct UVGI air disin-  
23 fection systems, it is perhaps not surprising that few guidelines exist re-  
24 garding this type of installation (ASHRAE, 2019; Kowalski & Bahnfleth,  
25 2004), with those that do exist being somewhat vague. For example, re-  
26 garding in-duct UVGI the ASHRAE guidelines simply state: “*The required*  
27 *average irradiance for a typical in-duct system is on the order of 1000 to*  
28 *10,000  $\mu\text{W}/\text{cm}^2$ , but it could be higher or lower depending on the appli-*  
29 *cation requirements.*” (ASHRAE, 2019) Because it is difficult for building  
30 owners and consultant engineers to perform the necessary in-duct UVGI  
31 calculations, they are forced to rely on the claims made by manufacturers  
32 regarding the performance of their equipment. Consequently, there is need  
33 for the manufacturers of UVGI air disinfection equipment to undertake ro-  
34 bust microbiological testing, as well as fully characterising the UV fields  
35 produced by their devices.

## 36 C.6 UVGI room air cleaners

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38 Many manufacturers make ‘UV-C in a box’ style air disinfection devices  
39 for use in room spaces. These devices essentially comprise UV-C lamps or  
40 LEDs mounted in a container with a fan. They are generally free-standing  
41 units designed to disinfect all the air that passes through the device. As  
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3 such, manufacturers frequently make claims akin to ‘the unit achieves a  
4 99.9% disinfection rate’ based on a ‘single-pass’ microbiological test. These  
5 claims can be however extremely misleading, because they relate purely to  
6 the air that passes through the UV device and not to the effect that the  
7 unit will have on the room space, which can be calculated for steady-state  
8 using

$$C = \frac{q}{\dot{v}_r + (\dot{v}_{uv} \times \eta)}, \quad (15)$$

14 where  $C$  is the contaminant concentration in the room space under steady-  
15 state conditions (e.g. (virions/m<sup>3</sup>),  $q$  is the steady-state room contami-  
16 nation rate (virions/s),  $\dot{v}_r$  is the room ventilation rate (m<sup>3</sup>/s),  $\dot{v}_{uv}$  is the  
17 air flow rate through the UV air cleaner (m<sup>3</sup>/s), and  $\eta$  is the single-pass  
18 efficiency of the air disinfection device expressed as a fraction.

22 From equation 15 it can be seen that if the room ventilation rate is large in  
23 comparison to the amount of air flowing through the UV air cleaner, then  
24 even if all the air passing through the unit is 100% disinfected, the overall  
25 impact of the device on the room space will be minimal. So unless the  
26 air cleaner delivers a high air flow rate, or alternatively the room is very  
27 poorly ventilated, devices like this are unlikely to have a major impact on  
28 the transmission of COVID-19 when employed in large spaces.

33 Notwithstanding the discussion above, if used in small poorly ventilated  
34 spaces, or in conjunction with partitions, then small UVGI room air clean-  
35 ers may be effective in protecting individuals against COVID-19. Consider  
36 for example, a communal office desk that has been partitioned with screens  
37 (i.e. creating booths) in order to minimise spread of COVID-19. If local  
38 air cleaning devices could be developed that disinfected the air within the  
39 individual spaces created by the desk partitions, then this might provide  
40 additional protection to individual workers. However, the feasibility of  
41 this would need to be verified either through CFD analysis or experimen-  
42 tal work.

## 49 C.7 Discussion

51 The principal findings of this Appendix are that:

- 54 • The SARS-CoV-2 virus is susceptible to damage from UV-C light and  
55 as such can be relatively easily inactivated using UVGI.
- 57 • The SARS-CoV-2 virus appears to be particularly vulnerable to UV-C  
58 light when aerosolised and as such, may (in theory) be readily disin-  
59 fected using upper-room UVGI air disinfection.

- If retrofitted to recirculating HVAC installations, in-duct UVGI air disinfection may potentially be a useful technology for enabling commercial buildings to function during the winter months of the COVID-19 pandemic.
- With respect to COVID-19 transmission, no suitable guidelines exist regarding UVGI air disinfection.

When considering the above findings it is important to note the caveats “in theory” and “potentially”. These have been inserted because although there is strong evidence that the SARS-CoV-2 virus is susceptible to UV-C light, suggesting that UVGI air disinfection has considerable potential, there still is much doubt about how the technology should be applied in order to protect the occupants of buildings. While this doubt stems, in part, from an incomplete understanding of the mechanisms by which COVID-19 is transmitted, it is also due to inherent issues associated with the technology itself, which make its application difficult, as evidenced by the lack of detailed guidelines on the design of UVGI air disinfection systems. These inherent drawbacks relate specifically to: (i) the fact that the design of UVGI air disinfection systems is strongly influenced by the choice of the target microbe; and (ii) difficulty in predicting how any given installation will behave without the use of complex simulation tools like CFD. This makes it difficult to produce robust guidelines that are applicable to all situations. For example, the guidelines applicable to, say, the transmission of TB in hospitals, might not be suitable for protecting against the spread of COVID-19 in bars and music venues. Furthermore, once an air disinfection system is installed it is difficult, without extensive microbiological testing or a clinical trial, to tell whether or not the air disinfection system is actually protecting the occupants against infection. Collectively, these factors have greatly inhibited the uptake of UVGI air disinfection, with the result that many are suspicious of the technology.

Notwithstanding the above discussion, the results of the feasibility study by Beggs and Avital (Beggs & Avital, 2020) suggest that upper-room UVGI air disinfection may well have a role to play in preventing the transmission of COVID-19 in some settings. However, much remains unknown about the limits of this technology with regard to COVID-19. Upper-room UVGI was primarily developed as an intervention against ‘true airborne’ diseases such as TB, where the infectious particles are  $<5-10 \mu\text{m}$  in diameter. However, COVID-19 is not a ‘true airborne’ disease in the classical sense, inasmuch as it can also be transmitted by inhalation of aerosol



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3 droplets in the range 10-100  $\mu\text{m}$ . Depending on their size and room air  
4 velocities, these larger aerosol particles can readily become suspended in  
5 the air and be dispersed around the room space. While many of these air-  
6 borne particles are transported by convection currents to the upper part  
7 of the room space, some particles, due to their mass, may decouple from  
8 convective air streams and settle out due to gravitational deposition. In  
9 doing so they may fall through the breathing zone, where if they have not  
10 been fully inactivated by the UV field, they will be a potential hazard. So  
11 while it is undoubtedly the case that upper-room UVGI can help to inhibit  
12 the spread of some COVID-19, the extent to which it can be effective is  
13 not known and there is urgent need for CFD analysis work to evaluate  
14 how the technology can be adapted to be effective against COVID-19. In  
15 particular, there is a need to explore the use of room mounted fans in  
16 conjunction with UVGI air disinfection to promote better air movement  
17 between upper and lower room zones.

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26 Given the urgent need for a solution, which will make commercial proper-  
27 ties (offices, shops, pubs, etc.) and public buildings (schools, universities,  
28 etc.) COVID-19 safe as well as habitable during the winter months, per-  
29 haps the most promising potential application of UVGI air disinfection is  
30 as a retrofit for recirculating HVAC systems. By installing in-duct UVGI  
31 in the return air duct-work it should be possible to permit the recirculation  
32 of air while ensuring that SARS-CoV-2 virions are not recirculated. How-  
33 ever, while this retrofit solution should not increase fan power consump-  
34 tion, the installation of UV-C lamps might considerably increase electrical  
35 power consumption, something which may make UVGI unfeasible. In ad-  
36 dition, any requirement for 'fail safe' filters might also make in-duct UVGI  
37 untenable.

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44 Because the performance of UVGI air disinfection installations is difficult  
45 to validate, architects, engineers and building owners are generally forced  
46 to rely on claims made by equipment manufacturers. However, the ev-  
47 idence base on which these claims are made, particularly those relating  
48 COVID-19, is often very weak, with little or no microbiological testing in-  
49 volving viruses undertaken. Consequently, commissioning bodies such as  
50 the NHS and government departments are reluctant to accept these claims  
51 without substantial clinical (biological) evidence, thus inhibiting the gen-  
52 eral uptake of UVGI air disinfection. As a result, there is urgent need  
53 for manufacturers of UVGI systems to undertake appropriate microbiologi-  
54 cal testing of their products so that they can demonstrate their efficacy  
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3 against the SARS-CoV-2 virus.  
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5 Regarding UVGI air disinfection, the guidelines that exist (ASHRAE,  
6 2019; First *et al.*, 1999*a,b*; Coker *et al.*, 2001), which were largely de-  
7 veloped to control the spread of TB (Coker *et al.*, 2001), tend to be rather  
8 vague on technical issues, preferring instead to take a ‘rule-of-thumb’ ap-  
9 proach. In particular, the guidelines take little or no account of room air  
10 flow patterns, which (as discussed above) are crucial to the performance of  
11 upper-room UVGI with respect to COVID-19 transmission. Consequently,  
12 these guidelines cannot be relied upon in the context of COVID-19 and as  
13 a result there is urgent need to develop new guidelines that are focused on  
14 this disease.  
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## 21 C.8 Conclusions

22 The conclusions regarding UVGI are:  
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- 24 • There is strong evidence that the SARS-CoV-2 virus can be inacti-  
25 vated by irradiation with UV-C light and that the virus is particularly  
26 vulnerable to UV damage when aerosolised.  
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- 28 • There is good theoretical evidence to suggest that upper-room UVGI  
29 might be effective at disinfecting the SARS-CoV-2 virus in room air.  
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- 31 • While in theory upper-room UVGI can militate against COVID-19  
32 transmission in enclosed spaces, because larger aerosol particles (i.e.  
33 10-100  $\mu\text{m}$  in diameter) can decouple from room air convection cur-  
34 rents, it may be that additional supplementary air movement devices  
35 (e.g. room mounted fans, etc.) will be needed to ensure good air  
36 disinfection.  
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- 38 • In-duct UVGI air disinfection could potentially be a useful technol-  
39 ogy for enabling commercial buildings to function during the winter  
40 months of the COVID-19 pandemic.  
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- 42 • There is urgent need for a robust evidence base regarding UVGI air  
43 disinfection. In particular, because it is difficult to predict how UVGI  
44 equipment will perform in any given context, there is heavy reliance on  
45 the claims made by manufacturers. As such, there is urgent need for  
46 manufacturers of UVGI equipment to undertake appropriate microbi-  
47 ological testing so that end users can be confident that their products  
48 will work effectively against the SARS-CoV-2 virus.  
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- 50 • There is urgent need to develop robust methodologies for validating the  
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3 performance of UVGI air disinfection systems, in order to demonstrate  
4 that they work (i.e. prevent the spread of COVID-19 in buildings).

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7 • There is a need for suitable guidelines regarding the design and use of  
8 UVGI air disinfection systems in the context of COVID-19.  
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## 10 11 **D Aerosols in the context of singing, and woodwind & brass** 12 **musical instruments** 13 14

15 Music is an important part of our cultural heritage as well as providing  
16 or supporting much of the entertainment industry. While recognising the  
17 importance of the activities of professional musicians and singers and the  
18 organisations which employ them, it is important to note that the student  
19 and amateur musical scene represents a much larger number of people.  
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22 For professionals, students and amateurs alike, the mechanics of playing  
23 or singing are similar, and so measurement of the aerosol risks can be  
24 considered in a similar way. There are some significant differences in regard  
25 to the impact to the community and environment in which the aerosol is  
26 likely to be produced, namely:  
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31 • Professionals and conservatoire level students are likely to be spending  
32 greater periods of time playing in both rehearsal and performance  
33 settings and be sharing a performance venue with a very much larger  
34 and more mixed audience than is the case for amateurs.  
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36 • Outdoor performance by amateur woodwind and brass groups is com-  
37 mon, particularly during the summer months. Some amateur groups  
38 are choosing to rehearse outdoors at the present time.  
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40 • Shared use or short-term loan of an instrument is common for early  
41 level students, and in some amateur groups. This is also an issue for  
42 pre-sale trial of instruments.  
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44 • Amateur musical groups include early level students, people of ad-  
45 vanced age, and people with pre-existing health conditions.  
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51 The need for improving the safety of indoor rehearsal and performance  
52 is well understood, and research to measure the level of aerosol created  
53 during performance is being undertaken, so that risks from musicians and  
54 singers can be compared with the risks of other performers such as orators,  
55 actors, dancers and sports competitors. Currently, the only published  
56 and peer-reviewed report is that on the vuvuzela by Lai *et al.* (2011). A  
57 substantially more thorough study carried out by Gregson *et al.* (2020)  
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3 comparing aerosols produced in singing, speaking and breathing is now  
4 available on the pre-print server ChemRxiv. A further piece of work by  
5 the same authors investigating aerosol production in musical instruments  
6 is expected to be released in the near future (Front Row, BBC Radio 4,  
7 2020). Another major study is in preparation at Colorado State University  
8 (Volckens *et al.*, 2020). More informal studies have been carried out by  
9 a number of organisations (Bamberger Symphoniker, 2020; Brandt, 2020;  
10 The Vienna Philharmonic, 2020; Kähler & Hain, 2020; Performing Arts  
11 Aerosol Study, 2020).

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17 When such aerosol production is better understood, appropriate ventila-  
18 tion and protection measures for concert halls, theatres and arenas can  
19 then be designed. Organisations which represent professionals and am-  
20 amateurs, and also those which represent performance venues, are already  
21 producing extensive guidelines on the operational logistics of rehearsal and  
22 performance (Orchester.ch, 2020; Brass Bands England, 2020; Musicians'  
23 Union, 2020).

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28 Outdoor performance presents less risk than indoor, particularly where  
29 mitigations, such as socially distanced seating of the players and separa-  
30 tion from the audience or passers-by, are upheld. Mitigations for indoor  
31 playing consider issues of ventilation, and numbers of players in a room  
32 of a given volume. Other mitigations that have been suggested, but for  
33 which the efficacy has not yet been established, involve the wearing of  
34 a mask while playing, with a hole cut for the mouthpiece; and the use  
35 of a covering to trap aerosols exiting from the end of the instrument. All  
36 such mitigations are undesirable: outdoor performance feels unsatisfactory  
37 without the venue reverberation and musicians would usually choose to sit  
38 close to each other to hear each other better; wearing a mask while playing  
39 will impact on the player's ability to perform; and covers on instruments  
40 reduce the sound level and can also impact on tuning and sound quality.

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48 It should also be noted that a person moving through a room can create a  
49 wake or cause settled particulates to be resuspended in the air. Musicians  
50 who make large body movements may contribute to this effect, so whilst  
51 percussion, string players and conductors do not blow their instruments  
52 their bodily movements may contribute to the redistribution of aerosol  
53 around a rehearsal or performance space. This is illustrated in the string  
54 player photographs in The Vienna Philharmonic (2020) where the bow  
55 movement entrains the flow.

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60 For woodwind and brass instruments, the player's breath drives the sound

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3 production, and breath generated aerosols will be carried through the in-  
4 strument. Some of that aerosol load would be captured within the instru-  
5 ment as condensation. In brass instruments, which are not perforated with  
6 finger holes, and have lengthy and convoluted pipework, such condensa-  
7 tion might be expected to be more complete than in shorter woodwind  
8 instruments. Notwithstanding this, condensation cannot be relied upon as  
9 a means to reduce aerosol when the instrument is warm, or being played  
10 in a warm environment.  
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15 Since the interior of an instrument can become contaminated if played by  
16 an infected player, the issue of effective cleaning of loan or trial instru-  
17 ments is important. An approach might be to place each player on a track  
18 and trace register after each loan or trial, or apply a quarantine period to  
19 the instrument. If the instrument is needed sooner, or if a player devel-  
20 ops COVID-19, then, where feasible, it could be put through a chemical  
21 cleaning cycle. Metallic instruments and student grade plastic instruments  
22 might tolerate such treatment, but quality wooden instruments would not.  
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28 There is considerable ongoing research in this area at the present time, and  
29 it may be some time before complete peer-reviewed reports are available.  
30 Some reports cited here are media press reports.  
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