**Evidence of environmental dispersion for different mechanisms, and the risks and potential mitigations/measures of control within different environments from what we know about COVID-19:**

**A brief evidence summary for SAGE, 14 Apr 2020**

*Redactions have been made to protect author identities and remove reference to individuals and organisations. No scientific content has been redacted.*

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**Key conclusions from this review**

- The dispersion of virus is due to a complex interaction between people either generating or interacting with virus particles and conditions defined by the local environment. This includes the layout of the space and conditions including air movements and ventilation rates, temperature and humidity. It is more nuanced than a simple distinction between airborne and contact driven, and there is a pressing need to understand potential risks with a finer degree of granularity than in current population epidemic models.

- Evidence from studies evaluating the physics of aerosol dispersion suggest that particles released from a cough or similar could travel much further than 2m, potentially up to 7-8m. Aerosol from environmental sources released in large quantities can travel further still under certain wind conditions, as shown in the Amoy Gardens SARS outbreak. In many environments dilution by the airflow will mean that the risk of significant transfer of virus over large distances will be limited, however this risk has not been clearly quantified. The small amount of evidence reported from sampling studies to date in hospitals suggests that virus has been detected up to 4m from the source in air and up to 3m as deposition. Virus has also been detected in corridor air adjoining patient rooms. It is not clear whether this virus is viable or is at a sufficient concentration to cause infection.

- There is limited conclusive evidence as to where transmission takes place, however one study from China indicates that it is very likely that the majority of transmission is in indoor environments. Cases reported where large outbreaks are associated with a single index patient appear to also happen in indoor environments.

- Models that consider the mechanistic aspects of transmission may be useful to understand dispersion of virus in different environments; however it is critical that models are developed with a view to quantifying exposure/risk. It is feasible to model dispersion of aerosol considering the different sizes of aerosol particles, the environmental conditions and the occupancy scenario, and indeed there are many such models. However the majority make no attempt to quantitatively relate the model outcomes to exposure, considering both spatial distance and time of exposure.

- There is a challenge to relate physical models to actual infection risk, as the data on viral load released by people and remaining in the environment is very limited, and knowledge of infectious dose for different exposure routes is unknown. Probabilistic approaches using ranges based on the limited current data on SARS-COV-2 and other related human coronaviruses may be a feasible approach. Alternatively relative effects of different control strategies can be modelled without specific viral load or dose-response data.

- Modelling the effect of strategies such as enhancing building ventilation rates, or the use of facemasks on exposure to virus in the air in some typical settings could potentially be carried out fairly easily. Models could also be developed for contact transmission (e.g. exposure in a supermarket) as well as aerosol deposition in indoor and outdoor environments to improve surface cleaning and hand hygiene recommendations – these would likely be more difficult. Validation of all of these models would be very challenging.

- There are some existing models that may be able to be adapted for analysis, however some of the modelling approaches are computationally and time intensive to set-up and run. National high performance computational resource and support may be required to address the demands of this.

- A key next step would be to identify specific scenarios of priority interest and convene an appropriate expert group to develop a strategy for modelling each one, to ensure models produce outputs that are relevant. This will most likely be engineers/mathematical modellers working with clinical, virology, public health and statistical experts, as well as the people who manage the particular environments of interest. Priority interest could be established by examining person density and frequency of visits for different environments, together with an initial assessment of local conditions (e.g. ventilation) to give a risk score that could be used to warrant deeper investigation.

- There are environmental control strategies such as building ventilation where it may be prudent already to give stronger recommendations as a precautionary measure, particularly in higher risk enclosed environments. Providing clearer guidance on how people could do this in different environments would be useful.

**Scope of this paper**

1. This paper focuses on aerosol dispersal and environmental spread of pathogens, identifying evidence of relevance to the SARS-COV-2 virus, particularly in relation to the current measures on social distancing and evidence on transmission risk in different indoor and outdoor environments.

2. We consider the research evidence on: mechanisms for environmental dispersal; exposure to pathogens; quantification of infection risk in different environments; approaches to control risk; areas for further research including potential for modelling environment specific transmission.

3. SAGE should note that this is not a comprehensive literature review. We summarise some of the key knowledge and papers in this area based on our expertise and a time-limited review of the relevant literature. We also highlight aspects of our own work and ongoing work of some other groups where models/methods could be expanded and applied to the current situation.

4. We include emerging evidence for COVID-19 including papers that are on pre-print servers and have yet to have full academic peer review. The evidence base for COVID-19 changes on a daily basis and hence this is correct to the best of our knowledge at the time of writing.

**Aerosol release, dispersion and deposition**

*A note on aerosol particle size and terminology*

5. Critical to understanding aerosol dispersion is particle size. Particles are referred to by their diameter. The SARS-COV-2 virus is around 0.06-0.14 μm (60-140nm) in diameter [1]. Respirable particles are those below 10μm in diameter which have a probability of being inhaled into deep lung. Particles up to 20μm can reach the thorax, and up to 100μm can be inhaled and impact in the nose and mouth. A human hair is approximately 60μm and below 40μm is unlikely to be visible to the naked eye. The majority of particles of respiratory interest are not visible.

6. In medical literature airborne infection normally refers to infection via very small pathogen carrying particles that are 5μm or less in diameter; these are sometimes referred to as droplet nuclei. Those greater than 5- 10μm are normally referred to as respiratory droplets. The term aerosol is used by some to only refer to droplet nuclei, while others will use it to describe a wider range of particles. In this document we use the term aerosol as an overarching term to describe respiratory particles across the whole size range of interest.

*Aerosol generation*

7. It is well recognised that normal respiratory tract activities such as breathing, talking and singing, as well as coughing and sneezing, generate aerosol particles in a range of sizes from 0.01 to more than 500 μm in diameter[2]. It is not clear which is the dominant size range, however the majority of recent studies using optical methods for particle counting suggest most particles are below 10μm.

8. Measurement of pathogen carrying aerosols in infected people is more challenging than simply measuring respiratory aerosols and requires careful set up of appropriate sampling equipment. However, several studies have demonstrated pathogens in the respirable range (below 10μm) in the exhaled breath of people with infections including for TB [3][4], influenza [5][6] and *Pseudomonas* in cystic fibrosis [7]. These studies conduct direct measurements of the exhaled breath shortly after exit from the mouth/nose and so provide the best evidence for quantitative microbial load at the point of human release into the air.

9. No conclusive expired breath data exists yet for COVID-19. A study conducted in China [8] (pre-print paper) took 4 exhaled breath samples of COVID-19 patients during an environmental sampling study which were negative, however these were not carried out using appropriate specialist sampling equipment. However high viral titres (concentrations) have been demonstrated in the nose and throat of $4.9 \times 10^4$ and $9.9 \times 10^3$ copies/ml respectively [9]. The potential for viral aerosol formation due to breathing and coughing airflows therefore remains. The most relevant data for any estimates will most likely be that for influenza. A body of work from Milton's group at the University of Maryland has detected influenza virus in exhaled breath [5][6][10], with 8.8 fold more in fine aerosol fractions (<5μm) than coarse [6]. The geometric mean copy number of culturable influenza virus over 30 min was $3.8 \times 10^4$ for the fine fraction, $1.2 \times 10^4$ for the coarse fraction and $8.2 \times 10^8$ per nasopharyngeal swab[10].

10. Data on aerosol from healthy people is useful to understand the size range and number of particles during different activities but should be used cautiously when calculating viral shedding

rate. The quantity of virus carried by an aerosol will depend on diameter and the viral titre in the respiratory fluids; respiratory aerosols will be comprised of the viral particles surrounded by complex respiratory fluids containing salts, proteins, surfactants as well as water. These components have a mass larger than the virus and hence are significant in determining aerosol size [11].

11. Determining the number of viruses carried by an aerosol is not straightforward; data from influenza shows that the virus may be preferentially in a particular size fraction[10] and hence a simple volume fraction calculation is not appropriate. The process of aerosolisation is complex and poorly understood, but it is an effect where respiratory airflows and fluids coating (e.g. the nose and throat) interact. It may also be possible that hydrophobicity of some microorganisms causes preferential aerosolisation. There is no evidence either way as to whether this is the case for SARS-COV-2.

12. Aerosol generation has also been demonstrated from environmental sources, particularly toilets and building sewer systems. Toilets are well known to produce aerosols that contaminate bathroom environments [12] although a conclusive link between this source and infection is hard to show. Poorly maintained drain traps (u-bends) were suspected as a transmission mechanism for the 2003 SARS outbreak [13], [14] and has since been demonstrated as a mechanism for biological aerosol production in laboratory studies [15]. Recent studies have shown that hand dryers can disperse microorganisms into the air in bathroom environments [16].

*Aerosol dispersion and deposition*

13. The fate of a respiratory aerosol depends on a number of factors including the initial size of the aerosol and the microorganism(s) contained within it, the velocity with which it is ejected from the mouth, the interactions between particles, and the temperature, humidity and airflow in the environment that it is released into.

14. Very large droplets (greater than 800μm), behave ballistically and can travel more than 2m when released forcefully (e.g. in a cough) simply due to their large size and momentum[17]. In still air settling time for small individual particles is governed by fluid dynamics principles (Stokes law) and depends on their size and physical properties; a 2μm particle would typically take 4.5 hours to fall 2m while a 10μm particle would fall the same distance in 11min. While this is widely used in evaluating aerosol risk [18] and suggests larger particles fall quickly, reality is much more complex.

15. Studies show that initial aerosols rapidly evaporate as they leave the 100% humidity of the human respiratory tract and enter the lower humidity external environment. The evaporation to a stable size is rapid – less than 1s is suggested [19] - and will depend on initial size [20] and composition[11].

16. Air, even in indoor environments, is not still - the movement of air in indoor environments is easily enough to maintain a 10μm particle airborne and transport it with air movements caused by ventilation flows or other movements (e.g. people walking, doors opening). Imaging of human cough aerosols [21] and a controlled laboratory study [22][23] show complex behaviour where aerosol particles travel in a turbulent cloud or plume and are influenced by buoyancy in the environment. This enables the aerosol to travel a bigger distance together in air than may be expected, with distances of up to 7-8m predicted in indoor conditions [16].

17. Under certain flow conditions there is evidence that infectious aerosols can travel in plumes for very large distances. A study of the Amoy Gardens outbreak during the 2003 SARS outbreak

suggested that virus aerosolised from the sewer system in an apartment building infected others in the same building and was carried on the wind to infect several people in neighbouring apartments 10's of meters away [14]. There are also well documented cases of transmission of legionnaire's disease through aerosol released from cooling towers. In both cases the amount of pathogenic material released in the aerosol is considerably higher than from a respiratory source.

18. While it is not clear how well the SARS-COV-2 virus survives in the air in real environments, a controlled laboratory study has shown it to be stable in aerosol for more than 3 hours, with a half-life of over 1 hour [24]. This appears to be more stable than influenza under similar conditions. Similar studies of the MERS-COV virus showed 63% survival after 1 hour at high humidity, but much lower survival in hot dry climate conditions [25].

19. Evidence of air contamination comes from a small number of studies conducted in hospital environments. We have identified 7 studies to date that have carried out air sampling for SARS-COV-2, one published paper [26], one early-release paper [27] and 5 papers still in pre-print that should be treated with some care [8][28]–[31]. Four of these studies found several positive samples for virus in air, two found a very small number of positive samples and one found no positive samples. Three studies give some quantitative data on viral loads.

20. A study in Singapore [28] found positive air samples in 2 out of 3 patient rooms, with samples in the 1-4μm and >4μm size ranges ranging from 1.84-3.38 viral copies/litre air. A typical volume of air breathed in 1 minute is ~10L.

21. A study in Nebraska, USA [29] found 63% of air samples were positive with a mean 2.86 copies/litre, including in patient rooms and the hallway air. In one case they sampled close to the patient (4.07 copies/l) and at >6ft (2.48 copies/l) suggesting some dilution with distance. Highest concentrations were found in personal samplers worn by the sampling team when in the presence of a patient receiving oxygen (19.17 and 48.21 copies/l).

22. A study in Wuhan [31] provides quantitative data for their small number of positive air samples, finding 0.019 copies/l in a toilet area and 0.018-0.042 copies/l in a room used to remove PPE. They identified positive samples in the 0.25-1μm and >2.5μm size ranges.

23. While these studies give an initial indication it should be noted that measurements are from a small number of samples and are all in well-ventilated hospital rooms. With all microorganisms that are present in small quantities in air it can be challenging to sample as it can be difficult to collect sufficient microbial mass to reliably detect. While TB has been known to be airborne since the 1950's, it was only in the 2000's that it was reliably sampled from exhaled breath and in rooms. Currently, there is insufficient data on SARS-COV-2 to confidently confirm how widespread air contamination is, and at what level. It is also not clear how likely this is beyond patient rooms, although there is a small amount of evidence showing corridor areas outside patient rooms may be contaminated.

24. Aerosol deposition is known to be a route to surface contamination for many pathogens. Studies in controlled chamber [32] and healthcare [33] environments demonstrate bacterial deposition onto surfaces, but data on particle size is not measurable. There is limited data for virus deposition and a study of influenza showed very little deposition [34]. In most environments it is hard to show which proportion of surface contamination is due to deposition from air vs contact from hands.

25. The small number of studies to date suggests that aerosol deposition of the SARS-COV-2 virus onto surfaces is happening. Data from hospital sampling studies have found virus on numerous room surfaces including the floor, high-touch sites and low touch sites such as ventilation grilles. Bathroom sites are noted in several of the studies as highly contaminated [8][26][28][29][31], which may warrant further exploration. Moreover, there is some evidence that even patients suffering from COVID-19 but showing mild symptoms, such as in Ong et al.[26], can release virus laden droplets which subsequently deposit onto surfaces [26][36] creating environmental reservoirs and clear opportunity for contamination of hands during contacts [36][37]. The study conducted by Liu [31] also measured deposition through passive aerosol sampling and indicated deposition rates of 31 and 113 copies/$m^2$surface/hr at sampling locations approximately 2m and 3m from the patients respectively.

*Research needs relating to aerosol generation and dispersal*

26. The most significant missing data on aerosol generation is on the viral load in exhaled breath, the aerosol sizes that carry this virus and how this varies between people. This type of data would enable a much better understanding of the significance of different sized aerosols in transmission and enable risk type calculations from computational models that already exist for aerosol dispersion in multiple environments. The most accurate data would most likely come from Prof Don Milton at the University of Maryland who has an instrument specifically designed for collecting viral aerosol in exhaled breath [6]. Cruder sampling may be possible with the CASS device [3][4]; [redacted]. A mask sampling approach for TB could also potentially be adapted for SARS-COV-2 [38]. Sampling would have to be carried under virology leadership.

27. Studies to understand whether deposition of viral particles occurs would be valuable to support understanding of whether environmental surface contamination is through aerosol or physical contact. This would be measured using settle plates or similar which would sample the deposited virus fraction from the air, but not the touch contacts. This is particularly relevant in high risk locations including hospitals and care homes, as well as where people are looking after sick relatives at home. Such data would support understanding of whether for example high contamination seen in bathroom areas of infected patients is due to aerosol release or high touch sites, as this subsequently informs the likely exposure and most appropriate control strategies.

**Exposure to aerosols in air, and potential for control**

*Mechanisms and evidence for exposure*

28. Exposure to aerosol particles depends on physical location with respect to the source, its speed of release (cough higher than breathing) and the size of the particles, as well as the environmental conditions. Conventional thinking is that people less than 1-2m away will be exposed through deposition of larger particles onto mucous membranes, while those further away could inhale the fine aerosol particles. The importance of these two mechanisms is dependent on the disease. Current scientific opinion from world experts in environmental transmission of disease suggests that this distinction may be too simple and could be overlooking routes for exposure.

29. Although close range exposure is widely thought to be dominated by droplets, laboratory and modelling studies [17][39] examining exposure (1-2m) to different sized particles suggests that

inhalation exposure to fine aerosols (airborne risk) could be a more significant part of transmission than the direct deposition of droplets onto mucous membranes. This may be significant for the PPE requirements of those in close proximity to infected people and for Aerosol Generating Procedures in clinical environments. The mathematical model in [17] while not validated with humans (would be very hard to do) enables a method for estimating the relative importance of the droplet deposition and inhalation routes for different distances between people.

30. Modelling and laboratory experiments [17][39] also suggest that aerosols with an initial size between 75μm and 400μm are likely to deposit onto surfaces quickly within 2m and in many cases within 1m. The exposure to these aerosols is therefore through direct droplet deposition onto the face or via contact transmission from contaminated surfaces. The amount of exposure will be determined by the virus concentration within the droplets and the duration of contamination. A person in a hospital room coughing for hours will create significantly more contamination than a person in a supermarket who coughs once at a particular location.

31. While very large ballistic droplets (>800 μm) can potentially travel further than 2m, they will only expose someone directly if they land on mucous membranes on the face. This would normally require being face to face with the source and because of the distance involved has a very low chance of the droplet hitting the target. Exposure to these droplets is much more likely through subsequent contact with contaminated surfaces as discussed below in paragraphs (61-75).

32. Exposure to aerosols below 10μm diameter for those greater than 2m away will be dominated by the airflow conditions in the environment. While there will be some deposition of particles resulting in contact transmission risk, exposure through inhalation is likely to be the dominate risk, and their dispersion can be predicted with airflow models. The exposure will be determined by the rate at which aerosols are generated together with the rate at which they are diluted or removed by ventilation indoors or wind outdoors.

33. Aerosols with an initial size between 10 and 75μm are the most complex group. These sizes can potentially travel beyond 2m as they can evaporate to form smaller aerosols which are then carried by the air [20]. Airflow models can predict such particle behaviour but need to carefully take account of evaporation conditions.

34. There is good evidence from infection data with other diseases that some people can be “super spreaders” producing a significantly higher infectious aerosol load than others and resulting in high transmission rates. A controlled study of TB transmission (human to guinea pig) identified a small number of individuals who caused significantly higher infections than others [40]. Outbreaks which give “clear evidence” for airborne transmission of disease tend to be those with a large number of cases from a single individual [41], and may be the result of a super spreader. In the majority of such cases those infected spend a measurable amount of time (>1 hr) with the index case.

35. There is emerging evidence for super spreaders in the COVID-19 pandemic (e.g. Boston conference outbreak, Skagit Valley Chorale outbreak, outbreaks associated with religious meetings in France and South Korea) which potentially point to airborne transmission or significant aerosol deposition. However to date these do not appear to have had investigation of the environment in any detail.

36. While data on SARS-COV-2 transmission is still emerging there is growing evidence that transmission is predominantly indoors. An analysis of cases in China [42] (pre-print paper)

resulting in more than three secondary cases identified homes and transport as the dominant locations, although food and shopping venues led to more cases. Only one of 7324 cases was associated with an outdoor environment – a conversation between two men. Reported super-spreader outbreaks are all indoors or in semi-enclosed and crowded outdoor venues.

*Potential for controlling exposure to respiratory aerosols*

37. The primary route to controlling exposure to fine aerosol particles in an indoor environment is ventilation. Increasing ventilation rates ensures better dilution of aerosol particles, and adjusting mechanical systems to prevent recirculation of air is a prudent measure. Professional bodies in several countries have strongly recommended such measures including ASHRAE in the US and REHVA in the EU. CIBSE in the UK have promoted REHVA guidance and supported such recommendations.

38. Exposure to aerosol is related to occupancy density, and hence minimising the number of people in indoor environments is a prudent measure. In many places this has already been implemented through the 2m social distancing, but for environments where this is challenging (e.g. public transport) maximising the ventilation rate and potentially controlling person density within such spaces would be appropriate.

39. Air cleaning technologies including ultraviolet disinfection may be a viable approach for controlling fine aerosol particles, particularly in rooms with poorer ventilation. UVGI disinfection is shown to be effective against coronaviruses in air [43], and our modelling study suggests that upper-room UV installation could be equivalent to doubling the ventilation rate [44].

40. Outdoor environments normally dilute contaminants in the air very well compared to indoor spaces. It is feasible that people in semi-enclosed spaces or immediately downwind and within a few metres of an infected person could experience a higher than expected viral load in aerosol under certain flow conditions, although this hasn't been calculated to our knowledge. This is unlikely to be a significant route for infection unless people are in this position for a long period of time. This is backed up in the outbreak investigation data to date, suggesting indoor spaces are the highest risk. However this risk could potentially be estimated through airflow calculations similar to those used to estimate urban pollution concentrations.

41. There is evidence to suggest that use of surgical type face masks by members of the public may help to prevent transmission of pathogens carried in aerosols. When worn by someone who is coughing, masks are shown through flow visualisations to considerably limit the distance reached by a cough [21]. A recent rapid review indicates there are some studies that show a small reduction in transmission when masks are used, although others do not show an effect [45]. A PHE study showed that homemade masks, while not as effective as surgical masks significantly reduced the number of microorganisms expelled by volunteers [46].

*Modelling exposure risk in air and through direct droplet deposition*

42. Modelling aerosol exposure risk requires a consideration of the spatial variation in the aerosol and how this changes with time. The physics of this process is a complex multiphase flow problem, which then needs to be coupled with an understanding of the viral load, virus survival, exposure route and infectious dose.

43. Evaluating the spatial and transient concentration of an aerosol is the first step in this process and can be carried out using computational or experimental flow based analysis methods as detailed below.

44. Computational Fluid Dynamics (CFD) is a tool that can enable detailed simulation of the airflow in indoor and outdoor environments and how contaminants in the air are dispersed. The model provides the ability to characterise flow patterns, identify areas of high contamination risk and assess the fate of different sizes of particles. It can also include the influences of aspects such as convective flows from heat sources and different ventilation settings (indoors) as well as wind speeds (outdoors).

45. CFD simulation has been widely used to explore disease transmission risk and the influence of hospital ventilation. Studies carried out during the 2003 SARS outbreak showed that nosocomial transmission [47] and the Amoy gardens [14] matched the airflow paths predicted using the CFD software. The method has also been used to explore ventilation in isolation and operating rooms as well as in chamber based studies to evaluate exposure risks [39].

46. While CFD is a powerful tool, it is a physics based model and produces deterministic simulations, and hence requires multiple cases/parametric study to understand variation in risk. The majority of simulation results are case specific, so care needs to be taken with generalising findings. Studies must be set up and run under the guidance of experts who understand the limitations of the tool and the context of the transmission scenario. The tool is computationally expensive, often requiring significant model run time (sometimes days) to produce accurate results from complex problems.

47. Several CFD models for cases of interest already exist, however the majority are not produced with the view to conducting a quantitative assessment of exposure. For example over the past week studies have appeared that suggest cough particles in supermarkets [48], and those released by runners [49] both pose a risk. Yet neither of these studies has considered the amount of aerosol that people would be exposed to (and by what route), and made any attempt to link this quantitatively to infectious dose or to clearly consider relative risk under different exposure scenarios. A small number of studies have included this distinction including the analytical model [17] and preceding CFD study [39] from Prof Yuguo Li's group in Hong Kong discussed in paragraph 29.

[redacted]Ventilation network or zonal models such as the NIST CONTAM tool provide another approach that can be used to model dispersion of aerosol under certain circumstances. These tools are not capable of capturing the detailed spatial distribution close to an aerosol source, but can be useful to model quantitative dispersion of the fine aerosol particles that remain airborne for long periods, and particularly to examine relative concentration risks in neighbouring room spaces throughout a building and the influence of the ventilation system. Such models are deterministic, but are very fast to run (seconds compared to hours) relative to CFD. The CONTAM tool offers a simple method for modelling exposure. [redacted]

49. Experimental analogue models use water as a surrogate for air to assess the behaviour of a contaminant released into different ventilated environments. Such models have been widely used to assess ventilation flows [50] as well as cough aerosols [22] and hospital flows [51]. A University of Cambridge team is currently using this approach to explore ventilation and layout in field hospitals for COVID-19. While such models are visually very powerful and can

demonstrate the influence of different scenarios with both space and time varying behaviour, it can be difficult to extract quantitative data on exposure for risk assessments.

50. Full scale surrogate measurements within the actual environment are potentially powerful in being able to assess dispersion and effectiveness of ventilation/airflows in the real-world. This can include tracer gas tests or release of inert and harmless particulates. Studies in well controlled mechanically ventilated indoor environments can often be carried out quickly, but those in outdoor environments or naturally ventilated buildings [52][53] often need considerable data to give clear conclusions. Studies also need to measure the source generation in order to understand the relative exposure at a distance. We have not yet investigated the urban pollution literature, but it is possible that there are studies that provide some quantitative insights into exposures in outdoor environments, for example for cigarette smoking.

51. Models linking airborne infection to indoor environmental conditions go back to the 1950's where the Wells-Riley equation was developed to predict cases of infection in ventilated environments based on the time of exposure, breathing rate of occupants, ventilation of the building and a parameter termed the "quantum of infection" which is related to the infectious dose. While this model has its limitations, it has been used widely to evaluate airborne disease outbreaks including TB, Measles and Influenza. We have recently developed an approach to relate the value for quanta to viral load in exhaled breath for influenza [54].

52. There is precedent for coupling outcomes from zonal ventilation models to the Wells-Riley and epidemic models [55], including considering stochastic effects [56]. Our group has applied the approach in theoretical analysis to hospital ventilation design [57] and application of UVGI disinfection devices [44]. Studies have also linked CFD model outcomes with the Wells-Riley model in the SARS outbreak [58].

53. Time of exposure is an important factor that must be taken into account in all assessments. Most exposure between strangers in the outdoor environment will be of the order of seconds. Those working together, shopping or interacting with friends in a distancing conversation may involve longer periods of time from a few minutes up to hours. Those in indoor environments where social distancing is hardest will have the greatest exposure time. Estimating such values will be important in modelling risks with any degree of accuracy.

54. Infectious dose is also a major consideration and there is very little data to support this for COVID-19 (and indeed for many diseases), and whether this changes with the route of exposure (inhalation vs direct deposition on mucous membranes vs hands touching faces). There is some data available for SARS/MERS [59] which could be used cautiously as a starting point.

*Research needs relating to aerosol exposure and control*

55. While there are numerous studies that have or are currently modelling aerosol dispersion in the context of disease transmission, the critical step that is missing is an evaluation of exposure. Ideally models would relate the aerosol concentration in air to actual data on viral loads in exhaled breath, but as highlighted in the first section of this paper, this data is missing. As a result the most appropriate approach would be comparative studies between scenarios but designed with the potential to feed in additional data as it becomes available.

56. There is a gap in understanding of outdoor exposure. In most cases risk is probably small due to rapid dilution of aerosol, however it could matter in some specific circumstances where people are in closer proximity or the wind conditions are such that the aerosol doesn't disperse. There

could be some benefit in conducting simulation of a small number of cases to quantify relative influences of parameters. This could provide some public reassurance, although the low risk in most circumstances would mean this modelling is probably not the highest priority.

57. Studies of viral aerosol exposure risk in indoor environments and the influence of mitigation strategies such as masks, ventilation or hygiene controls would benefit from further attention. There is a challenge with identifying which environments and scenarios to model as all buildings/cases will be different. However some environments where there may be benefits in developing more detailed models include hospital environments (public, wards, circulation, critical care, Nightingales), common public spaces such as supermarkets, generic office environments and public transport environments. Bathroom environments may also be worth exploring in more detail given the high contamination seen in hospital sampling studies.

58. Modelling the influence of facemasks on dispersion and inhalation is likely to be feasible. There are existing studies on masks (both CFD and imaging) which could be used to define parameters such as proportion of aerosol particles released, as well as direction and velocity of release. Similarly for exposure there is data on the efficacy of masks in limiting inhalation. Combining a simple breathing exposure model with an airflow dispersion model could enable an estimate of the benefits of masks in different settings

59. Modelling influence of ventilation on airborne risk in different settings would be feasible. We have previously developed simple zonal models coupled with infection risk models that could be adapted. Developing such models would need a clear understanding of the environment of interest and its occupancy to make sure that the scenarios were realistic.

60. Modelling deposition is the most challenging and uncertain area to assess. Measurement of deposition rates in indoor environments is challenging and depends on the aerosol size as well as the local flow conditions. CFD modelling approaches give some indication of likelihood of deposition location and could be used to estimate the build-up over time, however there is a high level of uncertainty associated with modelling deposition processes. This would then link into contact transmission modelling as detailed below.

**Contact transmission**

*Routes and mechanisms for transmission*

61. Contacts with contaminated surfaces called fomites were intrinsically linked to the transmission of SARS-COV-1 during the outbreak of 2003 [37][60] and evidence suggests they are also implicated in the spread of SARS-COV-2 [61].

62. SARS-COV-1 was shown to survive well on common surfaces [24] and in some circumstances to last months on dry surfaces [60], while coronavirus 229E is less hardy. However, under laboratory conditions (40% relative humidity), SARS-COV-2's half-life showed that it can persist in viable form for at least 10 hours on plastic, 6 hours on cardboard, and at least 6 hours on stainless steel [24][28][61][62], whereas it is less stable on copper: 2 hours. Moreover, viable virus was detected on plastic and steel surfaces up to 72 hours under the same conditions [24]. However, both increased temperature and humidity has been seen to have a positive effect in Middle East respiratory syndrome coronavirus (MERS-COV) inactivation [63]. MERS-COV could still be recovered after 48 hours at the 20°C – 40% RH condition, whereas the virus remained only viable for eight hours for 30°C – 80% RH and for 24 hours at 30°C – 30% RH respectively

[63].

63. The potential for transfer to and from hands during contacts with fomites has been demonstrated, for many bacterial and viral microorganisms, via transfer efficiency studies [64]. When contact between fomites and hands occurs, a portion of the virus is transferred, which for SARS-COV-1 ranged up to 24% with bare hands but is reduced to 3% when wearing latex gloves [65]. This proportion is described quantitatively by the *transfer efficiency*, or the fraction of a contaminant on an object that is transferred to another upon contact [66][67]. The ability to predict this transfer is then tantamount to being able to assess effectiveness of public health interventions, including cleaning procedures and hand hygiene recommendations.

*Control strategies*

64. Surface cleaning is one of the key strategies for controlling contact transmission. In environments where there is potential for aerosol deposition as well as transport of infectious material through contact with surfaces it will be important to consider this route to environmental contamination when planning cleaning strategies.

65. On stainless-steel surfaces ethanol at concentrations between 62% and 71% reduced coronavirus infectivity within 1 min exposure time by a factor of 100 to 10,000 (2.0-4.0 $\log_{10}$). Concentrations of 0.1-0.5% sodium hypochlorite and 2% glutardialdehyde reduced coronavirus by a factor of > 1000 (3.0 $\log_{10}$) in viral titre. In contrast, 0.04% benzalkonium chloride, 0.06% sodium hypochlorite and 0.55% ortho-phtalaldehyde were less effective [61]. It must be noted that these were under laboratory conditions and is suggestive that infectious virus will remain for at 1h after cleaning.

*66.* Hand hygiene is well recognised as a primary control strategy. CoV229E can remain on hands for up to 1h without disinfection [68]. Efficacy (or how effective a product is at removing the virus) is not 100% for any method even after one minute, and efficacy is influenced negatively by the degree of soiling on the hands. Use of 62% ethanol on CoV229E and SARS-COV-1 was 3$\log_{10}$ over 5 minutes meaning that risk is not insignificant after hand disinfection [61][68].

*67.* Donning of gloves for touching contaminated surfaces and doffing afterwards may be the optimal way of reducing hand contamination. However it should be noted that SARS-COV-1 can remain viable on latex gloves for 8 hours, highlighting the need for regular doffing.

*68.* Hand drying with air dryers should be avoided to reduce the spread of airborne pathogen carrying particles [69]. Preferably use paper towels.

*Modelling of contact transmission*

69. A *Pathogen Accretion Model* (PAM) has been developed at Leeds for predicting microbial concentration on hands following one (or multiple sequential) hand-surface contact(s) [66][67][70]. A key parameter in this model is the *transfer efficiency* between hands and surfaces, which is influenced by environmental factors, the microorganisms, and other characteristics of the transfer event such as contact pressure or humidity of the hand [71]. Transfer efficiencies have also been shown to be organism- and surface-dependent, where nonporous surfaces (e.g. plastic) are associated with greater transfer efficiencies than porous surfaces (e.g. fabric) [72].

70. The PAM could be linked to agent-based models so that stochastic sequences of hand-surface contacts in real scenarios can be realistically represented; for example, through discrete-time or

continuous-time Markov chains [67][73][74], which have been shown to be representative of defined procedures, e.g. healthcare episodes [67].

71. Studies of facial contacts can be leveraged to establish inoculated doses for the different agents. For example, some studies have shown that we touch our heads 19 times per hour on average whilst we touch our mouth 3 (sd=3) times per hour during non-eating activities [75][76]. Once specific doses have been established, dose-response curves [59] can be exploited to evaluate individual infection risk.

72. These models are already being applied in a hospital context but could also be used in other (e.g. community) settings. The final aim is to quantify individual infection risk, and to evaluate the efficacy of different control strategies. For example, a parametric *what-if* scenario type setup can be run to understand the effectiveness both spatially and temporally of cleaning regimes in supermarkets. This would include analyzing typical usage of trolleys, the transfer efficiency for SARS-COV-2 from the handles and coupling this with a variety of contact patterns of customers. The movement of the virus could then be tracked throughout the shop to identify high-risk contacts and how virus can be transferred from the initially contaminated trolley to products and then to other customers or staff. This would then be the base for analyzing cleaning regimes of trolleys, effectiveness of providing gloves for customers or hand hygiene using alcohol gel at regular intervals around the supermarket to mitigate buildup of virus. Another example would be public transport systems, where people are interacting physically with vehicles (e.g. holding surfaces), have proximity to each other and are aerosolizing and breathing – together with any airflows.

*Research needs relating to contact transmission modelling*

73. Transfer efficiencies for SARS-COV-2 need to be experimentally obtained, for different surface types, viral loads and a variety of environmental conditions (e.g., humidity and temperature). The team at Leeds has implemented Bayesian approaches in the past in order to estimate these from experimental data.

74. In order to link hand-surface transmission models to infection risk, dose-response curves for SARS-COV-2 would need to be in place. Alternatively, dose-response information for similar pathogens could be leveraged as a first step [59].

75. Behavioral data is needed for each scenario under analysis in order to calibrate these agent-based models. For example, for customers in supermarkets, information such as typical surface contact sequences and rates, number of products purchased/interacted with during a single shopping episode, average duration of a shopping episode, usage rate of baskets vs trolleys, disinfection rates of baskets/trolleys, etc. could be leveraged.

76. If we were to incorporate aerosol deposition through cough/sneeze, typical ventilation settings in different environments would need to be available and linked to models.

77. There is a need to consider how data from mechanistic models discussed in this paper would relate to population level epidemic models, and how this affects how both types of models are defined.

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