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COVID-19 and Beyond

A Brief Introduction to Passenger Aircraft Cabin Indoor Air Quality

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The realization during the COVID-19 pandemic that exhaled breath contained aerosols leading to airborne infection from infectious persons even those unaware that they had been infected has generated interest in the role that HVAC systems might play. The fact that HVAC systems serve two purposes, both to heat and cool the space and to provide clean air to breathe (in the case of aircraft there is a third purpose which is to pressurize the cabin so occupants can breathe normally), has led to some confusion among those addressing this subject. Many thought that spatial air change rate, a factor in thermally conditioning the space, was also a factor in controlling airborne virion concentration from the breath of an ill person. The higher the better. But that is not the case. Many also thought that the presence of HEPA filters means the air will be free of virions. But that also is not true if these filters are in the spatial recirculation system and do not intercept the breathe of an infectious person before it can travel to others I the space. Further, building ventilation standards ignore ceiling height, yet all other factors being equal, the higher the ceiling height the better the air quality. This paper provides the equations that govern group exposure and inhalation of occupant generated aerosols and gases following occupancy. They model both the 'lag' time after occupancy during which infectious aerosol concentration rises to the equilibrium (maximum) virion aerosol concentration when virion shedding rate equals virion

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removal rate, as well as the ongoing dose accumulation with time afterwards. The 'lag' time duration increases as air change rate decreases, while the equilibrium concentration is independent of air change rate. If air change rates are high due to a low spatial volume per occupant rather than to a high virus-free air supply per occupant, exposures are higher due to the shorter lag time. High air change rates also reduce the age of air, potentially increasing the viability of virions being inhaled and lowering the number of virions plating out in the space rather than being inhaled. Dose predictions compare favorably with recent industry measurements in wide body aircraft.

Preface

When this article was first published[1], ASHRAE received some very critical comments on it, so the article was peer reviewed once again and these concerns were all addressed in direct responses to each peer reviewer without any changes to the article required. However, additional supporting references were provided to this second set of peer reviewers, including a corroborative comparison with a coincidently published industry aerosol dispersion experiment in two wide body aircraft cabins.[2] This updated version of the original article was also peer reviewed and includes the points made in the post publication responses to the second set of peer reviewers including 64 additional references.

Article Scope

This article will be of interest to bodies protecting the public from infectious disease transmission, to HVAC and ECS engineers and to researchers in this field.

Introduction

Air travel is the dominant mode of public travel internationally. In the US, intercity air travel dominates by an order of magnitude over intercity bus and train combined.[3] While the material provided in this paper applies to both building and vehicular settings, the claims of the air travel industry provide a useful structure for examining some important ideas in transportation and building air quality design and analysis.

Since human breath generated infectious aerosols inhaled by other exposed persons as an infectious respiratory virion and bacterial dose is cumulative with time numerically for all settings a person passes through, until the respiratory cilia have cleared them, it is important to design all public settings with HVAC/ECS systems for control of human generated infectious aerosols with their typical sequence of passage in mind. The

predictions of the equations provided in this article have been corroborated with the findings of an in-depth investigation in wide body aircraft. Corroboration with other spaces remains, noting that one major difference between aircraft spaces and buildings that could play an ameliorating exposure role is typically much lower air change rates in buildings but similar ventilation rates per person, as a result of higher ceiling heights which not only increases spatial volume per person thereby diluting virion concentration in mixed spaces, but in poorly mixed spaces, allowing initially warm human breath to rise out of the breathing zone of other occupants. Further, while building spaces have much lower air change rates than public transportation settings due in part to their higher ceilings, the equations that set maximum bioeffluent concentration are a function of ventilation rate per person and ventilation effectiveness rather than air change rate, and these apply to all indoor spaces. The misconception that they do not stems in part from the fact that floor occupancy density is quoted as a factor in current building ventilation standards while volume occupancy density is not. This is misleading and results in ceiling height being ignored by some users of these standards and the impact of increasing this dimension to reduce air change rate and thus increase virion aerosol age, lessen virion viability at low relative humidity[56] and increase the time for possible virion plating out and thus lessen airborne concentration. Thus, for example, school classrooms, gyms and auditoria with higher ceiling heights than those spaces in aircraft cabins that have lower ceiling heights, but similar floor occupancies, with their lower spatial air change rates may be thought to have worse air quality relative to human virion aerosol exposures when in fact the reverse is true. Their virion exposures with be no higher and may be lower depending on ventilation effectiveness and age of air related plating out, while the age of the virion aerosols will be older and thus potentially less virulent.

One other important point to note is that threshold dose limits vary with occupants so keeping exposure doses below HID_{50} values or any other such criterion, does not mean no one will get sick at values far below that criterion if there even is one established. Nevertheless, similar criterion should be applied to all such settings.

Turning to the airline passenger industry, it has been claimed, for example, that passenger cabin air quality is exceptionally good compared with many other settings. Some airlines claim the air in aircraft cabins is cleaner than that in offices and on a par with the air in hospitals because it has HEPA filters in the cabin recirculation system. But the HEPA filters in the cabin recirculation system cannot intercept the dispersion of virion aerosols between the occupants within the cabin. One airline said the air is particularly good because it is very dry, creating a sterile cabin environment. Some say virus particles will only travel one or two rows. Nearly all say the air change rate is high compared to building settings and recirculated air is passed through HEPA filters that remove nearly 100% of airborne viruses. [2,4,5,6,7,8,9,10] This ‘higher the air change rate the better the air quality’ when it comes to the COVID-19 concern is not limited to aircraft. A Harvard document guidance for schools also makes this claim.[11] More recently, some industry publications have observed that the number of virion-bearing aerosols likely to be inhaled in aircraft cabins is very small relative to the number emitted.[12,13]

This article will review these claims and their relevance. It will provide equations that predict total viral dose predictions for groups of persons exposed to the breath of a virion exhaling ill occupant(s) in indoor settings, along with examples and a comparison with the findings of an industry experiment in a wide body aircraft.

The importance of airborne infection transmission

For decades it was thought that tuberculosis was transmitted through droplets and fomites, because it occurred most often after close contact. We now know that *tuberculosis* bacterial infections can be transmitted only via the air from speaking, coughing or singing, and not by fomites, kissing or sharing a drink or a toothbrush.[14,15]

The importance of aerosol inhalation as a COVID-19 infection route, i.e., and not primarily fomites, was not initially recognized during the COVID-19 pandemic. However, that perception changed with time, and public mask wearing became an important measure, especially in public transportation vehicles and hubs such as stations and airports.[16,17,18]

A recent review consistently found that humans produce pathogens predominately as aerosols or small respirable particles (< 5 microns) with PCR studies identifying infectious aerosols in the air of rooms with persons ill with COVID-19, the common cold, influenza A and B, TB, measles, herpes, and chicken pox. Low humidity exacerbates the risk.[19]

Virion shedding can be substantial. For example, in influenza-infected subject virion shedding testing, Yan *et al* measured the geometric mean RNA copy numbers as 76,000 copies/hour fine (particles < 5 micron) aerosol and 24,000 copies/hr coarse aerosol and found that sneezing was rare, and that sneezing, and coughing were not necessary for influenza infectious aerosol generation.[20] Coleman *et al* discovered that fine aerosols (≤5μm) generated during talking and singing contain more SARS-CoV-2 copies than coarse aerosols (>5μm) and may play a significant role in the transmission of SARS-CoV-2.[21]

A mathematical study found that, in exposure to exhaled droplets during close contact (< 2 m) via both short-range airborne and large droplet sub-routes, the large droplet route only dominates when the droplets are larger than 100 μm and when the subjects are within 0.2 m while talking or 0.5 m while coughing. The smaller the exhaled droplets, the more important the airborne route. The large droplet route contributes less than 10% of exposure when the droplets are smaller than 50 μm and when the subjects are more than 0.3 m apart, even while coughing.[22]

While aerosols will disperse much further than droplets, the concentration of infectious aerosol particles generally falls with distance, even when those infectious particles are carried by aerosols. [23] This would not be true in perfectly mixed systems where everyone has equal risk regardless of distance. However, it generally is true for airplanes in the longitudinal direction and for many other environments because of the reduced ventilation

effectiveness near the source. However, in the same row in aircraft cabins, the concentrations can increase with distance due to the air flow pattern and the source location.[2]

Figure 1 Aircraft cabins are high occupancy density with low ceilings as well as crowded floor seating

Commercial passenger aircraft cabins are high occupancy density with crowded floors and low ceilings (Figure 1), with air currents moving aerosols in all directions, making 2-meter social distancing impracticable if all seats are occupied and infectious aerosol exposures nearby to infected persons before any central recirculation filtration can take place. Other factors that could affect the passengers are the low cabin humidity (~10%) that can weaken our immune system's defense against lower respiratory tract infections and a high air change rate that causes several exposure issues. These include a rapid rise in human generated aerosol concentration, the potential for stratification and decreased ventilation effectiveness and due to the lower 'age of air,' more virulent virion exposures and higher exposures due to the aerosol cabin air concentrations having less time to be reduced via plating out. These issues will be discussed further.

Six input parameters govern occupant airborne occupant-generated virus average occupant exposure concentration in any portion of any uniformly over the portion of the floor area where the infectious aerosol spreads, mechanically ventilated setting at some ventilation effectiveness, except near boundaries such as walls where concentrations will be lower. The equation works for one point source or many distributed sources if the total particles shed are identical (i.e., infectious aerosol mass is neither gained nor lost). In other words, it is not necessary that there be more than one infectious aerosol shedding occupant or, if there are several shedding, that they be dispersed uniformly over the

portion of the floor where other occupants are exposed to obtain the equilibrium bioeffluent concentration to which they are exposed. [24, 25, 27, 35,36] (Equation 1). The six input parameters needed are:

- p, the inverse of the number of occupants in the setting that are exposed to the virions of one or more actively shedding ill persons (this number is known for wide body aircraft thanks to the Transcom experiments[2]). If the number exposed is not known, set p=1 to obtain the total amount inhaled.
- N, the average rate of infectious aerosol shedding (or any other bioeffluent emission being assessed) by any continuously virion shedding (or any other bioeffluent generating) occupants in the space.
- V, the virion (or any other bioeffluent being assessed) free air supply per occupant to the space.
- Ve, the ventilation effectiveness.
- v, the average occupant specific volume, the inverse of conventionally defined occupancy density, and
- t, the time after all the occupants have enter the space.

The first four parameters predict the maximum (equilibrium) average airborne virion concentration, C, for the number of persons typically exposed to a virion shedding ill person in the setting. The last four parameters set lag time to reach equilibrium. The smaller the occupant specific volume, the faster the airborne virus concentration reaches its maximum (equilibrium) concentration.

The average emitted aerosol concentration in the air of exposed persons is given by [25, 26, 35, 36]

$$C = p[N/VVe][1-\exp\{-VVet/v\}] \quad (1a)$$

where

C = average virion or any other bioeffluent concentration at time t in the space containing the exposed persons whether all or only one of the exposed persons is emitting that bioeffluent,

p = 1/number of persons exposed

N = rate of bioeffluent infectious virion aerosol generation/ill person shedding in the space

t = occupancy time

v = occupant specific volume

V = infectious aerosol-free ventilation rate per person (HVAC outdoor air + virus-filtered recirculation air + envelope infiltration air)

Ve = ventilation effectiveness. Ve can be greater than, equal to or less than 1

Ve = 1 in an on average uniformly mixed system

Equation 1 also works with non-uniformly ventilated spaces. Simply plug in the seat by seat (local) Ve.[72]

Equation 1 can also be written:

$C = p[N/VV_e][1-\exp(-ACH.t)]$ (1b)

where
ACH = actual (not nominal) air changes per hour, and
and
ACH = VVe/v = VVeOD, and
OD = occupancy density, p/L

Equation 1 can also be written:

$C = p[N.OD/ACH][1-\exp(-ACH.t)]$ (1c)

Thus, the higher the air change rate, the faster the airborne virion concentration (or any other occupant-generated bioeffluent such as human breath aerosols and carbon dioxide, perspiration, perfume, clothing and skin oil volatile organic compound emissions), rises to its maximum value after which “air change rate is not in the equation.”[27]

The infectious aerosol average dose, D, for an exposed group is the time-integrated function of virion concentration times inhalation rate, I, and is given by[1,26,35, 36]

$D = \int ICdt = p\{NI/VV_e\}\{t+[v/(VV_e)][\exp(-VV_et/v)-1]\}$ (2a)

Or

$D = p\{NI/VV_e\}\{t+[1/(ACHV_e)][\exp(-ACH.t)-1]\}$ (2b)

Or

$D = p\{NIOD/ACH\}\{t+[1/(ACHV_e)][\exp(-ACH.t)-1]\}$ (2c)

where

D = Virions inhaled by the group or group dose, virions

I = Exposed group average inhalation rate, L/s

Equation 2 predictions match those measured in the Transcom wide body aircraft cabin setting experiments as will be illustrated later.[2] The local Ve tells where infections are most likely to occur. Social distance (6 ft) is less effective in well mixed spaces, so it is for example more effective in a single aisle than a wide body airplane.[72]

The longer the exposure time, t, the higher the group dose.[26, 36] The higher the virus-free airflow per person, V, and the greater Ve, the lower the exposed group total dose. Alternatively, the higher the air change rate and the lower the occupancy density, the lower the exposed group total dose.

Note that Equations 1 and 2 do not predict the dose distribution needed to calculate individual infection probabilities. Scenarios can be proposed using the total dose but its distribution to adjacent occupants comes from CFD analysis or better still, given the Transcom findings, from careful in-depth experiments such as was done by the Transcom investigators.

Even without these distributions known, the ability to calculate total dose for known setting design exposure periods will enable ECS/HVAC engineers to design V, Ve, filtration/purification devices to minimize infection risk for exposed occupants in each setting of concern, without know exactly what they are, and can do so with or without masks of known efficiency being worn by either, both or none of the ill person and the exposed persons.

Once the distribution of the total dose in each setting is known, assuming it differs by setting, engineers can easily plug in the Equation 2 calculated total dose to each known distribution and compare individual infection risks, one setting to another. Coincidentally, health scientists will need to establish HID 50s, HID10s etc. for each pathogen of concern to make individual dose calculations useful.

Air change rate and lag time

High air change rates are positive indicators of ventilation performance when it comes to mitigating surface area related contaminants such as material offgasing emissions and one-time emission events, and to controlling spatial air temperatures. However, when it comes to the abatement of occupant bioeffluent emissions such as occupant body odor or breath aerosols, a high air change rate that is the result of a high occupant density rather than a high ventilation rate per person does not necessarily indicate good ventilation performance; and there are associated issues.

Let us review the governing equation for bioeffluent emissions (Eq. 1a, 1b and 1c). Air change rate (ACH) is not in the equilibrium portion of Eq. 1a and 1b as noted by Chen.[27] It is, however, present in the exponential component of these equations. Here the ACH component reduces the equilibrium component by an amount that diminishes more

rapidly with time the higher the air change rate. The time between occupancy and negligible equilibrium concentration reduction is termed the ‘lag’ time. In the case of passenger aircraft and subway cars with their high air change rates, for example, the lag time is about 10 minutes while for building spaces with their lower air change rates the lag time could be 30 minutes or more.

Thus, high air change rates (ACH) in high occupancy density (i.e., crowded) settings that result from meeting but not exceeding minimum ventilation standards) increase airborne virion infection risks for short exposure times when compared to less crowded settings that meet the standards at a lower air change rate. Equilibrium (maximum) infectious aerosol concentration may never be reached in offices for example, with their low occupancy densities and thus long lag times, and with people leaving for lunch, coffee breaks, etc.

The time to decay to zero concentration after the occupants leave a space is the same time length in reverse. Here there is an advantage to having a high air change rate after a group leaves a space and a second group enters. If the second group entering a passenger cabin or a classroom entering has no infected person(s) shedding virion aerosols, but the first group does, the time of clearing will be important for the second group, and the shorter the lag in clearing the space, the lesser the second group’s dose. If on the other hand the second group also has infected person(s) shedding virion aerosols, the time of clearing may not significantly change the dose inhaled by the exposed group in the second class. In any case, the greater the time between one group departing a space, and the next group entering it, assuming the ventilation rate remains unchanged, the lesser any dose inhaled by the second group.

Air change rate and age of air

High air change rates reduce the age of virions in the air, so any virions inhaled are likely to be more virulent than in spaces with lower exchange rates. They also reduce the likelihood that virions will plate out or have settled before exiting the space. These factors increase the infection risk of exposed occupants.

Air change rate and stratification

High air change rates can result in stratification of ventilation air flows and possible lowering of the portion of ventilation air reaching occupants or increasing exposures of occupants downstream of an ill person thereby increasing the infection risk of exposed occupants.

Filtration

While aircraft HEPA filtration removes almost 100% of the 0.3 micron and larger particles circulating through them (and supposedly, therefore, all viruses), the recirculation filters do not intercept the exposure to an ill person's aerosols of nearby neighbours.[13, 13, 28]

Furthermore, the amount of air recirculated through these filters and supplied to the passengers is one-eighth the amount circulated through MERV 13 office air filters, which remove at least 30% of 0.3 micron particles and larger. Thus, with their eight times larger airflows through less efficient filters, building filters can remove twice the number of viruses from the air supplied to each office occupant than aircraft HEPA filters remove from the air they supply to aircraft cabin occupants.[25,26] Offices HVAC generally supply a higher minimum V than aircraft and they can use free cool, i.e., bring in extra outdoor air rather than run their ACs to save energy while aircraft can't do this.

ASHRAE Standard 52.2 indicates that MERV13 filters actually have a 50% or greater 0.3 micron and larger removal efficiency.[29] Thus, in the hypothetical situation where a MERV 13 filter with twice the air flow through it per person as flowing through a HEPA filter, the MERV 13 filter will supply the same quantity of virus-free air to each person as would the HEPA filter. Decreasing filter face velocity by inceasing filter surface area also increases filter efficiency.

It is not that aircraft HEPA filters should be replaced by MERV13 filters any more than building MERV13 filters should be replaced with HEPA filters. Each system has been designed to perform spatial heating and cooling properly as well as meet ventilation requirements. The aircraft ECS blower system is designed to put a cabin air certain CFM through HEPA filters and will remove more 0.3 micron particles than any lessor filter with the same airflow through it.

What is true is that the greater the CFM/p passing through a HEPA filter or any other filter that removes some or all of a target particle size, the lower the equilbrium airborne concentation of that particle size in the space being served, all other parameters being the same.

Regarding the performance of lesser than HEPA filters for sub 0.3 micron particle size, the physics involved changes . For these particle sizes filter thickness as well as mesh hole area and duct diameters and lengths are all important factors. That is because the motion of particles smaller than 0.5 µm MMD is like that of gas molecules in the air. That is, they exhibit Brownian motion, act more like a gas and move according to molecular diffusion and the resistance to such movement.[31] Awareness of the physics involved for ultra-fine particles is well known. See for example, the Canadian Center for Occupational Health and Safety web site.[30]

The equation governing gas molecule mass transfer through a filter or duct via molecular diffusion from an area of high to low gas concentration is predicted by:

$$M_d = D * (\rho_2 - \rho_1) * A/L \quad (3)$$

where

M_d = mass transfer
D = mass diffusivity
$\rho_2 - \rho_1$ = partial density gradient
A = cross sectional area through which diffusion takes place
L = filter thickness or length over which diffusion takes place

So as L increases and/or A decreases mass transfer of fine particles < 5 microns MMD decreases.

In one published experiment, only 1% of particles from 3 to 20 nanometers in MMD (roughly 5 to 30 times smaller than corona virus) passed through 0.029" thick fiberglass furnace filters at face velocities of 10 fpm (0.05 m/s) like face masks, and only 0.1% through near HEPA filters.[31] Moroto measured 50% removal of 0.1 micron particles increasing up 75% removal of 0.8 and 0.03 micron particles by EN779 F7 (ASHRAE MERV13-14) filters for bag filter face velocities up to 43 fpm (0.219 m/s).[32] HEPA filters remove 100% of particles in the 0.03 to 0.8 microns size (MIL Std -282).[33]

Of course, a virus doesn't normally travel alone. Influenza virion bearing particle size distributions have been measured in airplanes indicating filter efficiency based on 0.3-micron diameter will underpredict actual efficiency.[34]

So, while HEPA filtration of the recirculated supply air does preclude re-distribution of infectious aerosols, the cabin airflows initially distribute unfiltered infectious aerosols to several rows fore and aft of an infector.[2,13,13,28] Movement of service carts and people through the aisle(s) might also carry suspended particles farther from their source. Further, since infectious aerosol concentrations generally (but not in cabin rows) increase with decreasing distance, in-cabin filtration near each occupants breathing zone would be a better approach, such as installing supplementary or even primary re-circulating filter systems overhead under the stowage bins or in the side panel where the slot diffusers are located. [35,36], under the seat[27] or in the head rest.[37]

Example calculations

Some examples input data are provided in Table 1 for eight settings. Occupant specific volume, v, and infectious virus-free air supply, VVe, to exposed persons and any ill person were developed based on 2010 HVAC code data, typical filters (HEPA removing all and

MERV13 removing 30% of virion aerosols in the air passing through them), and estimated infiltration rates through envelope leaks and openings (zero in aircraft but not in buildings or subway cars). These same data were used in SAE and ASHRAE publications a decade ago.[36,26] Minimum ventilation standards for newer buildings and aircraft have since changed.[38,39]

Table 1 Example eight setting V, Ve, v and ACH in eight setting types

Setting	v, M3/p	Group design exposure, hrs.	V, L/s/p	Ve	V*Ve, L/s/p	ACH= V/v
Subway Car	0.7	0.5	13.7	0.65	8.9	70.4
Narrow Body Aircraft	1	6	9.4	0.65	6.1	33.8
Wide Body Aircraft	1.6	14	11.8	1	11.8	26.6
Classroom, Grades 9+	8.1	6	10.9	1	10.9	4.8
Auditorium, Theater	10.2	4	10.6	1	10.6	3.7
Classroom, Grades 3-8	11.3	6	12.1	1	12.1	3.9
Lucas Oil Stadium Texas	26.6	4	12.6	0.9	11.3	1.7
Office	28.3	8	23.1	1	23.1	2.9

Fabian *et al* measured exhaled breath (without coughing, sneezing etc.) influenza A and B generation rates ranging from 3.2 to 20 influenza virus ribonucleic acid (RNA) copies per minute.[40] For the eight settings comparative dose virion concentration calculations, an influenza virus generation rate, N, of 11 virions per minute from an ill person was used.

A group of 19 exposed individuals surrounding an infected person (p = 0.05), have been used for these eight settings to calculate airborne virus concentration and inhalation dose scenarios. The predicted airborne virus concentrations in eight settings with the same percentage of ill persons versus time for the first hour for these scenarios are shown in Figure 2.

Comparing these setting concentrations with the setting crowdedness and outdoor air change rates for these settings, shows that the time to virion equilibrium concentration in the air (the mixed state) correlates directly with setting crowdedness and inversely with setting outdoor air change rate. Thus, for settings with the same equilibrium concentration and exposure time, the higher the crowdedness (1/OD), the higher the risk of a viral infection.

The slight trend variation for theaters is a result of their low outdoor air supply/person but high virus-free filtered recirculation air. Offices, on the other hand, have both higher supplies of outdoor air and virus-free filtered recirculation air, while the sports stadium analyzed had no filtered recirculation air. In practice, occupants of offices may never breathe air at its maximum virus concentration, since work hours can be staggered and people continually come and go for meetings, lunch, etc.

The equilibrium concentrations are plotted in Figure 3 versus the ACH = VVe/v. This figure shows the wide variability in air change rates and their lack of correlation with equilibrium virion concentrations, with building settings having ACH rates of 5 or lower while the transportation vehicles have air changes per hour greater than 20. Clearly there is no relationship between the two parameters for high OD settings meeting ventilation standards.

There are settings where the exposure risk is greater than those in long duration aircraft flights. For example, occupants in private homes, nursing homes and hospitals with OVID patients, with ongoing and thus high dose exposures to the aerosols generated by an infected occupant have the higher risks of being infected.

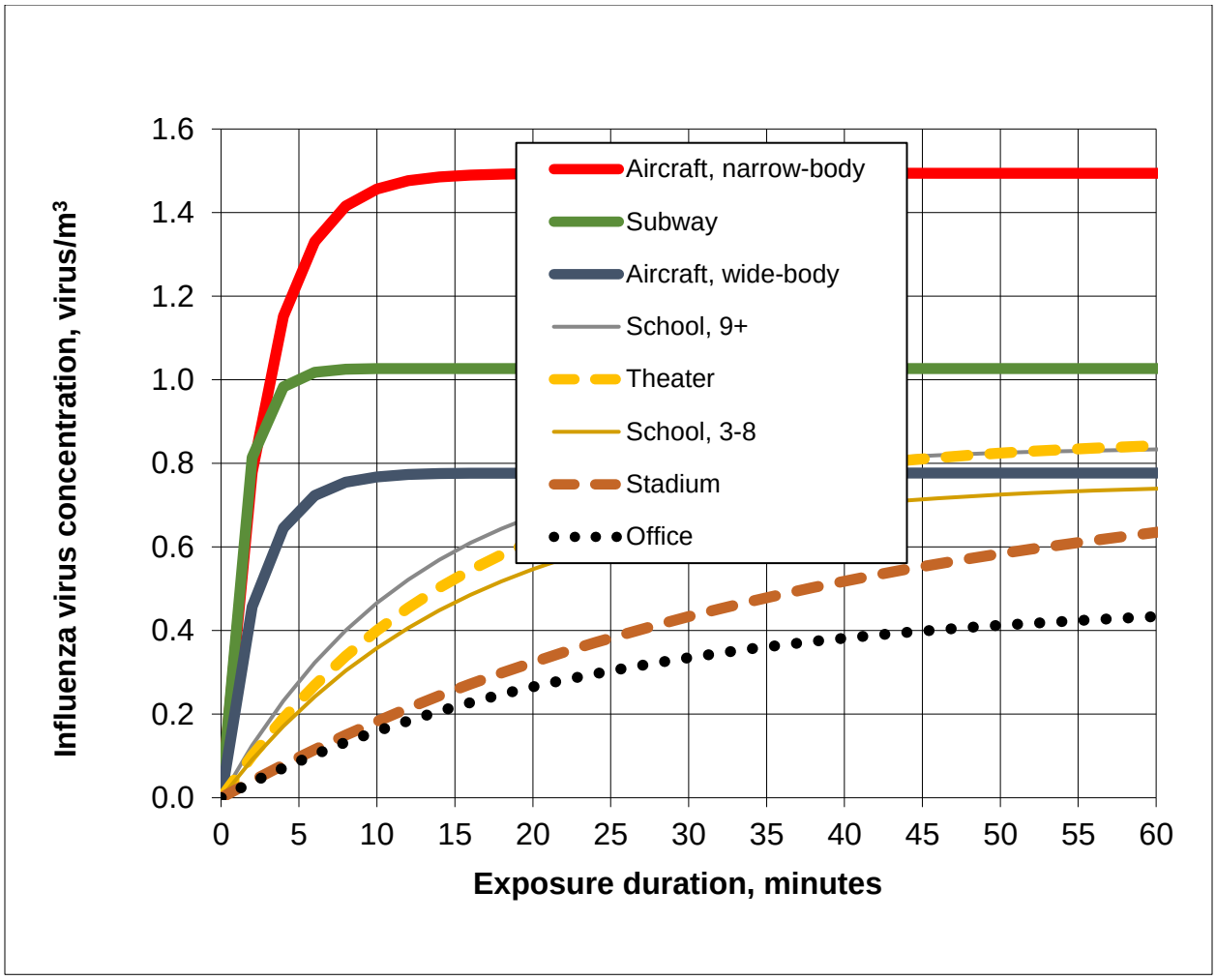

Figure 2 Average infectious aerosol concentrations in the air breathed by the exposed groups in the eight settings during the first hour of occupancy.

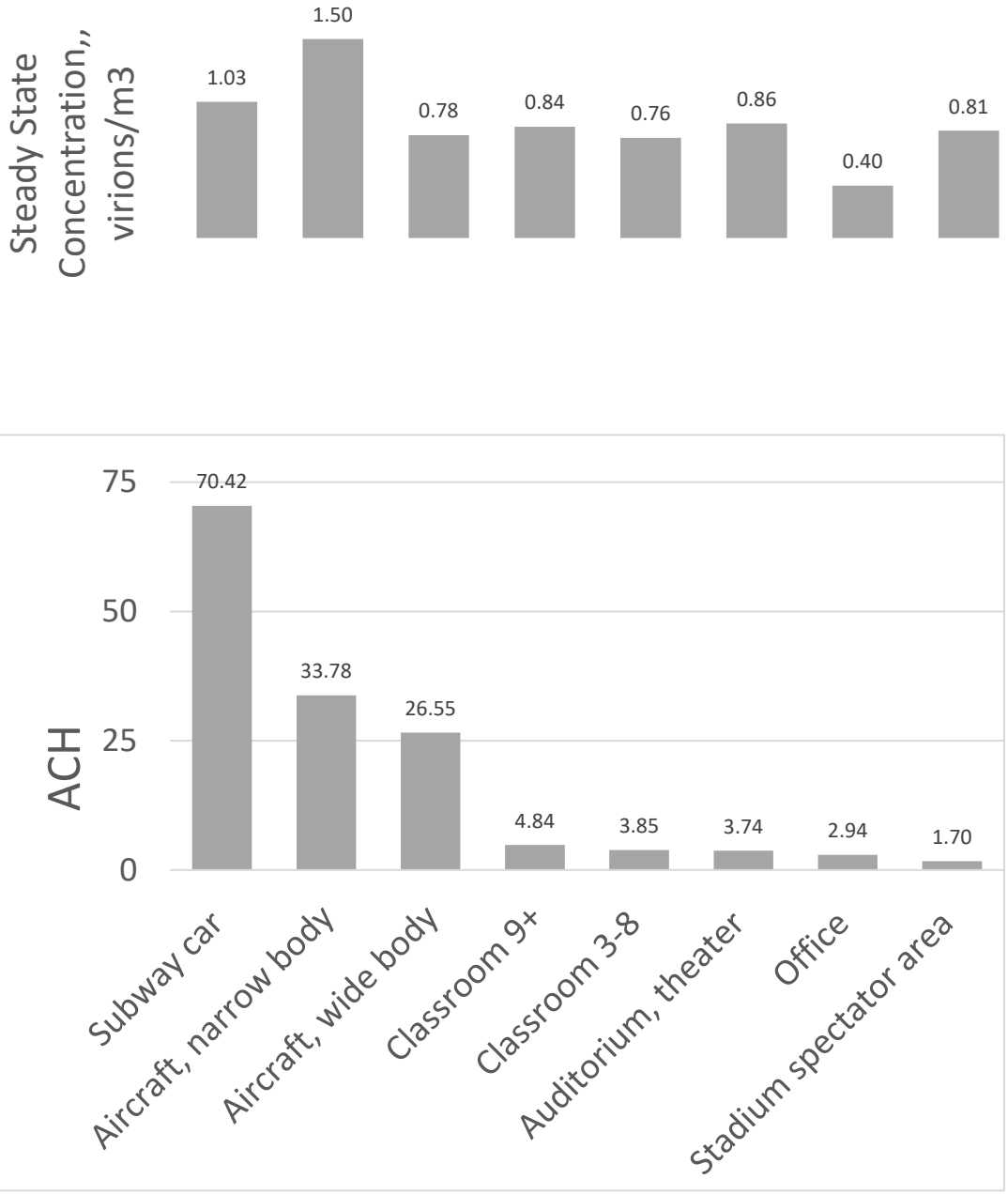

Figure 3 Steady state virion concentration versus air change ACH rate for the eight settings

Example inhalation dose calculations using Equation 3 are shown in Figure 4 for an 'at rest' occupant inhalation rate, I, of 0.132 L/s (0.28 cfm) (between sleep and light breathing rate and closer to the latter, for both sexes and most age groups[41]) and some design (longest) exposure durations. As flights can be long duration in comparison with the time spent in the other settings, even though airborne concentrations may be like other settings, dose will be higher.

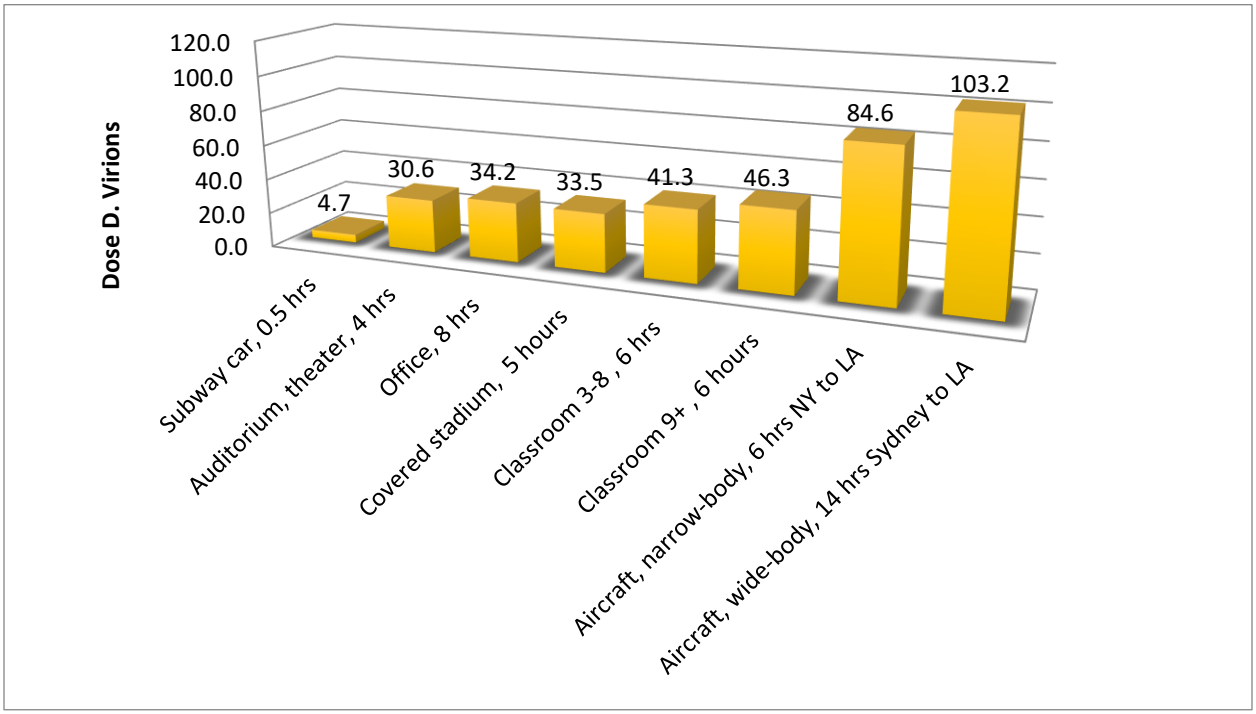

Figure 4 Predicted relative number of virions inhaled by a group of exposed persons (group total) during normal at rest inhalation from the breath of one Influenza A infected person for the design exposure periods in the eight settings.

Industry Aerosol Dispersion and Inhalation Measurements

A series of glass bead dispersion and deposition (inhalation) experiments were made in two wide body aircraft cabins by an industry group led by Transcom.[2] In one of these, a B777-200, several aft seat glass bead release locations from which four were selected to compare with Equation 2 predictions, were reported for 46 seats for two rows on each side of the glass bead (infection) source. The same input data were used for Equation 2 calculations as were used in the Transcom experiments, i.e., a 14 hour breath aerosol release and inhalation period, a bead deposition 'inhalation' rate, I = 3.5 L/min, a spacial 32 ACH rate comprising a virus-free air supply, V, of 9.44 L/s/seat, a Ve=1, and an occupancy density $v=OD^{-1}$ of 1061 L/p.

The locations of the glass bead release seats selected for comparison with Equation 2 predictions (i.e., seats 47D, 47F, 47J and 47L) are shown in Figure 4.

For a two class B777-200 passenger cabin with 375 passengers, these Equation 2 group dose predictions are an average of the Transcom measurements for various ill person locations. For a one class cabin with 418 passengers the group dose is near the higher group dose ill person seat locations and for a three class cabin nearer the lower group dose ill person seat locations.

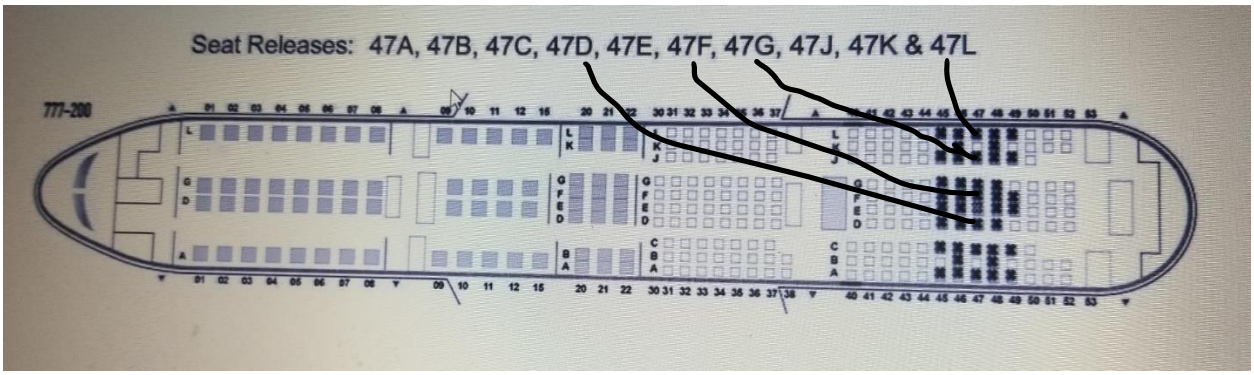

Figure 4 The B777 glass bead release seats chosen for comparison with Equation 2 predictions

The Equation 2 predicted percentage inhaled of those released versus the Transcom percentages are provided in Figure 5 for a two class cabin (business and economy). In this figure, the B stands for 'breath dispersion,' and the NM for 'no mask.'

Figure 5 shows the Transcom measured versus the Equation 2 predicted exposed passenger dose from virions shed from the breath of an ill person seated at four different locations in a two class (business and economy) cabin with 375 occupants using the same 3.5 L/min/p occupant inhalation rate, 15,075 cu. ft. cabin size and cabin 35 ACH. [2] The predicted dose percent inhalation was calculated for a 14 hour duration exposure, a virus-fee ventilation rate, VVe, of 11.1 L/s/p, and a specific occupancy density $v=OD^{-1}$ of 138 L/p.

As can be seen in the figure, the Transcom experimental group dose measurements straddle the predicted dose in this 375 occupancy example. For a one class 418 economy seating arrangement and the same air change rate, cabin size and inhalation rate, the predicted group dose is nearer the ill person seat locations 47F and 47D associated with a higher group dose, while for a three class 305 seating arrangement and the same air change rate the predicted group dose is nearer the ill person seat location 47J associated with a lower group dose

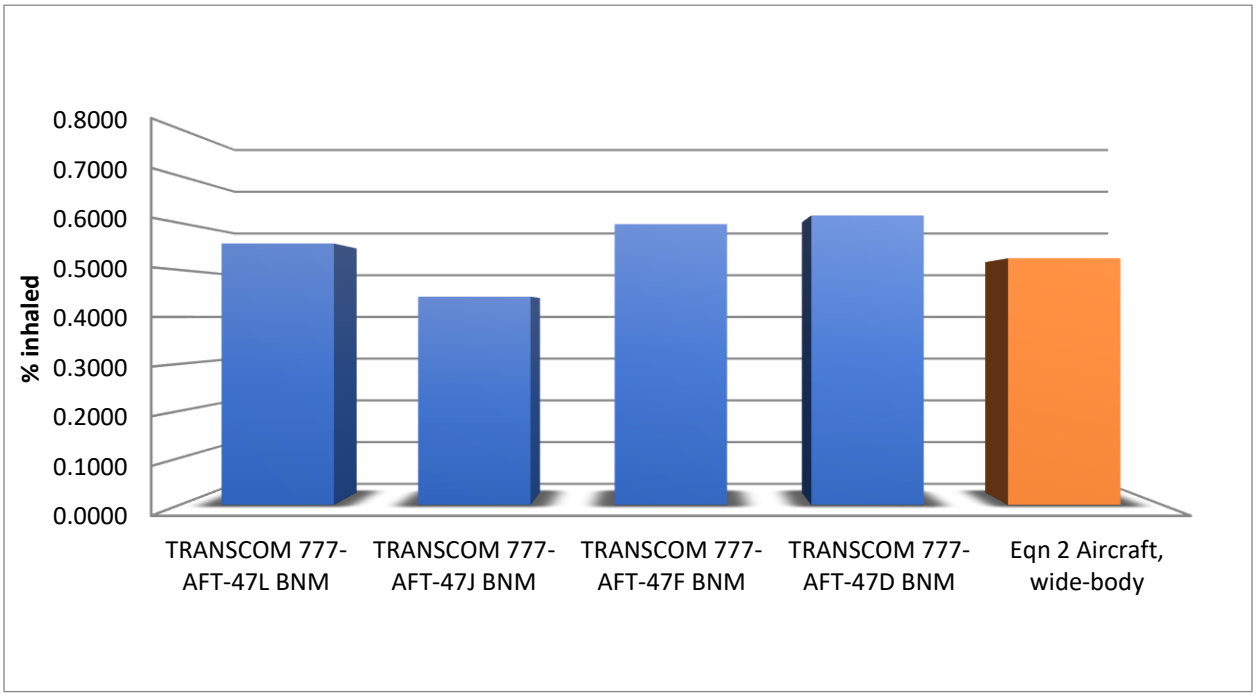

Figure 5. A two class Transcom 777-200 cabin group percent inhaled of shed virions measured versus Equation 2 predicted

Case studies

Viral infections such as from SARS-CoV-2 are spread from an infectious ill person to others via three very different mechanisms:

1. Sporadic cough and sneeze droplets that supposdly travel 6 feet or less;
2. Surface contamination by fomites; and
3. Continuously exhaled breath aerosols (≤ 5 micron) that various researchers have found can stay aloft 30 minutes or even longer and move around with air currents.[2]

Aerosols stay aloft more than 30 minutes in larger buildings, and this assures there is very little settling in the cabin because the residence time is lower. Since infections take several days to manifest, case studies have difficulty in identifying the infecting mechanism(s) and the setting(s) of origin. This situation has improved for COVID-19 infection setting/origin/follow-up, and aerosols and mask wearing are thought to be very important, given the screening, testing, surface disinfection, social distancing, and mask wearing measures in place.

One case study, for example, concluded there was a 9.8 to 17.8% COVID-19 attack rate on persons spread throughout an aircraft passenger cabin the cabin during a 17 percent full, 7-hour international flight to Ireland.[42]

Aerosols are more likely to inoculate the respiratory system, where the minimum dose requirement to inoculate is lower and the symptoms more severe than if the inoculation occurs in the nasal system where the larger droplets are more likely to rest.[43] In the past more limited longitudinal transport has been postulated.[44] However, more recent research on a wide body airplane indicates that a 10% concentration of droplet nuclei remains after traveling 4.39 m (14.4 ft) or five rows.[13]

Equation 2 predicts the total number of virion aerosols inhaled by persons in the same space but not their dispersion or individual inhalation. It does this prediction by accounting for the per occupant outdoor air supply rate and filtration rate of particles from the recirculation air supply and the ventilation effectiveness as it relates to diluting aerosols in the occupant breathing zone. It will also accommodate an estimation of the filtration benefit of mask wearing on aerosol exposure reduction, which should eliminate viral infection risks from surfaces contaminated with fomites (except for eye touching) and reduce risk from airborne droplets, both emitted and inhaled. Eye exposure might be important only in hospital-related viral transmission during ophthalmic practice.[17]

An example calculation has been made for an August 3, 2020, flight from Delhi to Hong Kong which post flight found 11 passengers were infected.[45] In the calculations that follow it is assumed that masks removed 50% of the virions from both exhaled and inhaled air. As influenza virions are similar in size to those of COVID-19 i.e. 0.08 to 0.1 microns vs 0.06 to 0.16 microns17,[46] in place of COVID-19 for which there are no shedding and infectiousness criteria, Influenza A copies virion shedding rate is used.[20] Also, for conservatism the lowest HID50 infectiousness criteria is used.[47] Incidentally, if the dose received is less than the HID50 criteria it means that less than 50% of exposed persons will be infected. It does not mean there will be no infections. In this regard it should also be noted that that the relationship between dose and infection rate is probabilistic and likely non-linear.

Equation 2 HID50 predictions and all parameters used are provided in Table 2.

If Influenza A is a valid surrogate and no one was wearing masks, it is possible that up to 7 COVID-19 infections occurred during that flight from exposure to one shedding ill person with a false negative pre-flight test.[48] If on the other hand the virions from this one infector were shared equally with the number of persons exposed in the Transcom wide body cabin tests (e.g., 46), there would be likely be no infections created during flight. It is likely also that if masks were being worn during flight no COVID-19 infections occurred during the flight or, if there were infections that these persons had not yet begun shedding at the time of the post flight tests. To these predictions must be added any virions inhaled prior to the flight and those of the 24,000 v/hr coarse aerosols (>5 microns) particles shed by ill persons that were aerosolized and inhaled before settling or exiting the cabin.

Table 2

Narrow body and wide body passenger aircraft influenza A virions inhaled and infections per index infector (not all ill persons are infectious), I = 9 L/min/p, t= 5 hr. N=76,000 v/hr. (<5 micron)[20], HID50 = 900 v.47 Narrow body OD= 1 m3/p, V=9.4 L/s/p, Ve=0.65. Wide body: OD=1.6 m3/p, V=11.8 L/s/p, Ve=1.

	Narrow body virions inhaled	Narrow body predicted infections	Wide Body Virions inhaled	Wide body predicted infections
No mask	9,244	≤7	4,794	≤4
Mask 50%	2,311	≤2	1,199	≤1

Davis *et al* reported a peak individual dose of 0.3% and an average dose of 0.05% of the nonvolatile mass of particles launched by coughing in a CFD simulation, with the launch meant to represent virions shed, the latter being in line with recent experimental testing reported in literature.[12] Kinahan *et al* measured exposures at surrounding infected persons and measured average dose as ~0.01% of particles shed in the Transcom experiment.[13] This individual inhalation percentage of virions shed is 1/50th of the percentage inhaled by the exposed group of persons measured in the Transcom experiment.[2] In other words, the larger the group of exposed persons the lower average exposure. Does that mean fewer people will get ill that greater the virion dispersion? Not necessarily as infectious dose can vary widely between individuals.[47]

Other factors: Dry Air in Aircraft Passenger Cabins

The air in passenger cabins is dry with a relative humidity (RH) normally less than 20%[49] and typically 10% after about 15 minutes as the flight progresses.[50] The air in buildings and residences in cold climaates can also be low. For example, an RH of 20% is not uncommon indoors during sub-zero Fahrenheit weather and such outdoor temperatures are not uncommon in winter in Canda and the northern US.

Cold and low RH conditions favor survival and transmission for some influenza virus, which also include viruses like respiratory syncytial virus, human rhinovirus, and avian influenza virus.[51,52] In addition to air at 10% RH being uncomfortable, it has been shown to impair nasal mucociliary clearance, innate antiviral defense and tissue repair function in mice and is, therefore, postulated to do so in humans.[53] Additionally, the lower the RH the faster droplets become aerosols. For example, at 27% RH droplets turns into aerosols within a few seconds.[54]

In terms of the quantifiable increased severe infection risk from COVID-19 and other coronaviruses due to cabin humidity this low, all we know for sure is that influenza in the United States and Canada occurs primarily in the fall and winter.[55] This is when people spend more time indoors which may account for most of the rise. But it is also when relative humidity in buildings with a heating system operating is perhaps 20% – 35% as opposed to being 50% – 65% indoors in summer air-conditioning weather. January-February mice tests in a Cornell University lab produced a significantly higher incidence of influenza transmission between infected and susceptible exposed mice (48 were infected out of the 216 exposed) than was the case during summer tests when only 1 out of 120 exposed were infected.[56] Weather would typically have ranged between 17F and 37F. This influenza seasonality is relevant for COVID-19 as well, unless a definitive understanding of the seasonality of SARS2 is developed that says otherwise, given that each are caused by a corona virus.

Quoting a recent virology journal article:

"The human-to-human transmission of the SARS-CoV-2 in Wuhan, China, began in December 2019. SARS-CoV-2 is a close relative of SARS-CoV, which spread during the winter of 2002–2003. Given that the expression of the receptor for both SARS-CoV-2 and SARS-CoV, angiotensin-converting enzyme 2, appears to be concentrated in a small population of type II alveolar cells, we speculate that the low humidity and temperature environment would promote the viability of SARS-CoV-2 in the droplets and impaired ciliary clearance and innate immune defense, for robust access to the deep lung tissue and rapid transmission between infected individuals. Since the respiratory airways, where the type I and II alveolar cells are located, are not reachable by respiratory droplets with a diameter of more than 5 micrometers, it appears likely that at least the severe cases of COVID-19 with viral pneumonia are the result of airborne transmission events. A recent study that examined province-level variability of the basic reproductive numbers of COVID-19 across China found that not only dry and cold locations experience high viral spread, but certain locations with high AH (absolute humidity) also have higher viral transmission within the population.

Seasonal changes in the environmental factors can affect not only local defense mechanisms but also systemic physiological changes. Thermoneutral temperature housing potentiates antitumor immunity and GVHD (graft-versus-host disease) onset in mice. In addition, a short daylight period and consequent deficiency of vitamin D impair nonspecific immune responses. In conclusion, the combination of low humidity, temperature, and sunlight may trigger an impairment of the local and systemic antiviral defense mechanisms, leading to the increased host susceptibility to the respiratory viruses in winter." [57]

Since the source of aircraft cabin ventilation air at 36,000 ft or so during cruising flight is very dry, the humidity that is present in the cabin is primarly from the occupants. Most of that is from metabolic (perspiration and breath) but a cup of coffee or a washcloth could

be secondary sources of water vapor. Part of the low humidity problem is that a portion of the cabin air with its ventilation and humidity components passes from the cabin to behind the cabin insulation through liner leaks into the envelope (the space between the cabin surfaces and the airplane exterior) where air humidity condenses on cold surfaces. The cabin air is drawn there around insulation blankets through liner leaks and openings by stack pressures which are highest near the top of the wall. These stack pressures are less than 10 Pascals at the top of the wall at cold soak.[61] For aircraft engineers, this cabin liner pressure differentials are miniscule compared with the 8.2 psi (56,500 Pascals) cabin pressurization exerted on the fuselage that is required when cruising at 40,000 ft to provide a cabin air density equivalent to that at an 8000 ft altitude.

Some of this liner leakage air is not lost as useful ventilation air and none of it is lost to the recirculation filtration system. However, the leakage air water vapor condenses on the cold surfaces on the structure of the airplane behind the insulation leading to ice accumlation and wet materials. When ice melts during ground stops, the water runs to the belly of the plane where it can be drained while on the ground.[58]

Passenger aircraft cabins at 8,000 ft equivalent air pressure that are ventilated with 10 CFM/p of outside air (i.e. meet the FAA requirement of 0.55 lb/min/p of outside air) will reach an equilibrium 10% relative humidity (RH) value after initial ground-based RH has dissipated. This is the RH value reported by Giaconia *et al.*[50] It is predicted using a passenger metabolic latent heat of evaporation of 0.1 lb/hr/p, a cabin liner leakge of 25% of humid cabin ventilation air where it loses all of its moisture, the recirculation air loses its moisture due to condensation in the belly of the plane, and there is negligble moisture addition from other sources including the ambient make up air.[59] This 25% cabin envelope leakage rate fits with unpublished cabin envelope leakage measurements made by the author and others.

Equilbrium RH will be higher if the ventilation rate and/or envelop leakage is lower, and lower if ventilation rate and/or envelope leakage rate is higher. For example, if there were no envelope leakage and 10 CFM/p outdoor air, the equilibrium RH is 14%. While with 25% leakage it is 10%. If there were no envelope leakage and 7.5 CFM/p outdoor air the equilibrium RH is 18% and with 25% leakage it is 14%. If there were no envelope leakage and 15 CFM/p outdoor air the equilibrium RH is 9% and with 25% leakage it is 7%.

To achieve humidity target levels of 30-50%, humidifiers with water tanks perhaps supplemented by exhaust air water vapor receovery are required. However, healthful humidities of 30-50% RH[60] or even wintertime building lows of 20-25% would increase the condensation on the structure and dead weight accumulation so envelope protection such as proposed by the Horstman, Preston and Walkinshaw would be warranted, [61,62] assuming mask wearing on aircraft to raise inhaled air humidity will not be an ongoing habit or requirement.

As noted earlier, increasing a very low relative humidity can also reduce virus viability depending on the time spent in the air.[63] For example, increasing relative humidity from 10% to 40% for virions in the air for 30 minutes decreases their viability by 16.5%, the equivalent to the effectiveness of the least effective face mask in eliminating 1-2.5 micron diameter particles.[64] Clearly further consideration needs to be given to increasing cabin humidity to at least 20-25% and preferably 30%, and investigating the role this might play in decreasing the potential for respiratory illness transmission and its severity.

Other factors: Lack of Mobility on Aircraft

Infection risk based on Wells-Riley type models describing airborne transmission of infectious diseases, define a threshold of infection based on the dose of quanta where a percentage of infection is expected.[65,66]

An airplane cabin with a single infector will show higher risk in the seat rows near the infector and lessor risk beyond that. The infector might move about the cabin distributing the virus more evenly, resulting in a lower dose for those in nearby seats and higher dose for the others. Surprisingly, if the infector could spread the viruses evenly, the individual doses would drop to that of a perfectly mixed Ve = 1 system. But the number of those exposed would increase proportionately and the Wells-Riley model would predict roughly the same number of infections. That is why a uniform concentration Wells-Riley model often shows the same number of infections as a distributed risk Wells-Riley model.

If the quanta generation is high enough to where a stationary infector would saturate the nearby passengers to the non-linear risk plateau of the Wells-Riley equation, then the movement of the infector is sufficient for additional infections farther away.

If the infector is stationary but other passengers pass nearby or stand in the aisle waiting for the lavatory, more people are exposed to the higher doses. As infection risk increases with inhaled dose, for any quanta of infectious aerosols inhaled by a group of exposed persons, the larger the group, the lower the dose to each member on average. But risk is based on a lower threshold based on the number in the group. In other words, stationary sitting maintains a virion concentration gradient with respect to an infector and potential ‘infectees.’ Putting one of these in motion flattens the exposure gradient and exposes a larger number of people but reduces the highest exposures. As another example of potential exposure flattening, occupants in ground-based public transit vehicles often can move around more freely, whereas in aircraft occupants may have to remain in one place for hours with a potentially ill person nearby. Also, in buildings there often is a greater possibility of occupants ‘social distancing.’

Other Factors: Cabin pressure effects

Turning to another issue with flying, most aircraft passengers face the problem of being in a lower air pressure environment than that to which they are accustomed. As the cabin altitude increases, the resting pulse rate increases to compensate for the reduced O_2 content of the blood. However, with acclimatization, heart rate and cardiac output tend to fall again.[67]

The maximum aircraft cabin pressure allowed is the equivalent of an 8000 ft altitude (¾ atmosphere or 75.3 kPa).[68,69] A14 flight study measured a cabin mean altitude equivalent of 6500 ft.[70] At these altitudes most persons will have no noticeable hypoxic problem, although for some there might be a headache and feeling of fatigue.[71]

The initial increase in respiratory rate was not accounted for in the dose calculations of Figures 4 or 5.

Personal Measure: Wearing Masks on Aircraft

Turning to measures with the current fear of COVID-19 exposures in cruise ships and aircraft, at the time of writing the US Department of Transportation was requiring that masks be worn in public transportation systems including on airlines. This is a necessary and effective step during this pandemic.[72] But will wearing masks be a publicly acceptable long-term solution?

Mask effectiveness varies. N95 masks can be very effective, while lower grade masks (i.e., surgical masks, bandanas, single ply cloth coverings) are not as effective.[73] Mask fit is an important factor. For example, the number of particles penetrating through the face seal leakage of tested respirator/masks far exceeded the number of those penetrating through the filter medium. For the N95 respirator, the excess was (on average) by an order of magnitude and significantly increased with an increase in particle size.[74]

Furthermore, mask wearing is a sociological phenomenon, and we should not assume ECS effectiveness based on generalizations about this behavior and how it might change under different settings and circumstances.

Will concerns about infectious aerosol risks end with this COVID-19 pandemic? Quoting from Scheuch[17]

"*Already in 2008, the group led by Patricia Fabian and Donald Milton was able to detect influenza viruses in exhaled aerosol particles on the suggestion of David Edwards. The authors found that 87% of the exhaled aerosol particles were < 1 micron in size*.[75] "

Yang measured a much lower ratio of <1 micron in the air rather than the breath. The airplane day-care and health centre had similar size distributions.34 Perhaps the size

distribution changes in room with agglomeration Brownian etc. but does not appear to be affected by the differing humidity.

Milton *et al* again detected influenza viruses in the exhaled air of infected patients.[76] They distinguished between larger aerosol particles >5 µm generated by coughing and smaller aerosol particles 5 µm. In 35 of 37 patients with influenza, they found significant amounts of influenza viruses in the small aerosol range, which were caused by normal breathing, whereas they could only detect virus RNA when coughing in 16 out of 37 patients, and the amounts of virus material collected were also much lower than those found in the small aerosol particles during normal breathing. The group also tested whether breathing masks used by the patients could effectively hold back these particles to protect health care workers. This worked quite well for the coarse aerosol particle fraction because virus material was only found in 4 out of 37 patients when the patient wore surgical masks. This was not the case for the fine aerosol particle fraction. Viruses were found in 29 of the 37 patients even with a breathing mask. The number of exhaled viruses was reduced by 55% by wearing a surgical mask.

Leung *et al* also found viruses in the exhaled aerosol particles.[77] They concluded, “Our findings indicate that surgical masks can efficaciously reduce the emission of influenza virus particles into the environment in respiratory droplets, but not in aerosols.”

Masks also will raise the relative humidity of the air being breathed by trapping the wearer’s humidity from his or her exhaled breath and that could be another reason masks lower the severity of COVID-19.[78] However, that moisture could create microbial growth exposure in a reused mask if it is not kept clean and dry between uses.

Masks will help protect others nearby, but not perfectly, so given the close quarters and airflow velocities, aerosols that escape can still move around the cabin perhaps five rows either way. Masks with ventilators should not be allowed because they allow viruses to be exhaled directly into the cabin.

Future Work

Beyond the wearing of masks, using virion-removing filters in HVAC recirculation systems and increasing recirculation rates offers the most promise. In passenger aircraft with their highly pressurized air supplies available to each occupant, installation of Venturi filtration near each passenger which will intercept and remove airborne pathogens shed by any ill persons before they can be inhaled by others seems practically possible.[27,35]

Raising aircraft cabin humidity also needs to be investigated as a means of reducing infection severity rather than relying on mask wearing to do this. One solution which would make humidification without condensation possible is to pressurize the insulation envelope.[61]

Conclusions

1. Equations 1 and 2 provide a means to estimate group exposure to and inhalation of airborne infectious aerosols shed by one or more ill persons in any indoor space.

2. The group inhalation predictions of Equation 2 compare with the findings of an in-depth aircraft industry led experiment in a wide body aircraft cabin.

3. A low percentage of virions inhaled by all receivers versus those shed by a potential infector is the norm for any setting.

4. The wider the dispersion of an infectious aerosol, the lower the average individual inhalation, but not necessarily the lower of the number of persons who become infected as susceptibility varies widely.

5. Experimental measurement, or CFD modelling using *inter alia* knowledge of the HVAC created air currents, the spatial volume and shape, and the exposed occupant spacing, is required to predict the extent of aerosol dispersion and individual virion dose within the dispersion.

6. High aircraft cabin nozzle supply velocities generate turbulent fluctuations which disperse airborne contaminant in all directions before their removal. The US TRANSCOM experimental findings confirm this dispersion for wide body aircraft with aerosols from a single cough spreading about 26 ft, which is equivalent in distance to about 11 rows in an aircraft passenger cabin if the row pitch is 29 inches. Previously, some cabin CFD models suggested breath aerosols can spread only eight cabin rows from an infector.

7. The predictions of Equation 2 indicate that commercial air travel on long duration flights with current aircraft ECS system design pose a relatively high risk of a person acquiring a virus infection compared with the other public spaces investigated for their design exposure durations. However, there are many settings not modelled that pose higher aerosol risks than air travel either because of long exposure durations, their low per person ventilation rates or other factors. These include homes and senior care/nursing facilities where exposures could be ongoing.

8. The presence of HEPA filters in a ducted recirculating HVAC system located outside the conditioned space does not necessarily create a low concentration of infectious aerosols in the space. That requires a high air recirculation rate on a per person basis. HEPA filters capturing the breath of each person within the space before their breath aerosols can be dispersed to others would be the best approach where this is practical if masks are not worn.

9. A high air change rate (ACR) in a densely occupied space is helpful in removing contaminants generated by single, transient emission events, or by materials. However, a high ACR does not necessarily create low occupant-generated infectious virion exposure. Rather, a high ACR coupled with high occupant density can result in substantially increased exposures to human generated aerosols and gases by reducing the lag time to reach maximum (equilibrium) airborne aerosol concentrations, while the corresponding reduced age of air may increase the potential for more serious infections by increasing the number of virions remaining aloft and viable, by decreasing the chance of virions plating out and not being inhaled, and by increasing the potential for aerosol stratification and reduced ventilation effectiveness.

10. Low humidity levels such as occur in winter in buildings and houses in cold climates and in aircraft on every flight, could reduce the rate of respiratory system mucociliary clearance of inhaled virions and thereby increase the extent and seriousness of respiratory infections. Wearing a mask not only reduces both the spread of infectious aerosols and inhaled aerosol inhaled dose by filtration, it could also aid in mucociliary clearance of inhaled virions by increasing inhaled air humidity in the winter heating season in cold climates, and in aircraft passenger cabins.

11. There are many factors at play in setting ventilation standards, some of which may not be noted. However, the quoting of floor occupancy density in building ventilation standards without mention of ceiling height is misleading. Spatial height plays an important role in determining the concentration of occupant shed aerosols and gases. For example, the high ceilings in churches or auditoria can result in reduced occupant virion exposures due to an increased lag time to reach equilibrium concentration after the occupants have entered the space, due to the large dilution volume per occupant if the space is mixed, or conversely if the space is stratified due to the aerosols in warm human breath rising out of the breathing zone due to buoyancy. As another example, when “occupant density” is defined as the number of persons per unit floor area, then an aircraft is the same as an auditorium. But when “occupant density” is defined in terms of how much “dilution volume” of air is assigned to each person, then aircraft have a higher occupant density than auditoria. In part because of this high occupant density, modeling data illustrate that the concentration of bioeffluent in aircraft cabin air gets higher (and does so faster) when compared to other indoor spaces like offices or auditoria. A delay or lag time before a ventilation system need be started in low occupancy density spaces such as an auditorium if the only inside air contaminants were associated with human occupancy was allowed over three decades ago by ASHRAE 62-1989 “due to the capacity of the air in the space to dilute (these) contaminants …before they reached steady state concentration.”[79]

12. ASHRAE ventilation standard setting committees should recognize the role their standards can play in human virion aerosol infection mitigation and set ventilation (outdoor air plus aerosol filtered recirculation air supplied to each occupant) rate

standards for each setting addressed based on design exposure times, and occupancy densities based on spatial volumes including ceiling height and not simply floor occupancy density, bearing in mind that virion inhalation is cumulative with time and is not restricted to virion inhalation in one setting. For example, an individual might spend two hours in a transportation terminal prior to travelling in the vehicle plus another half hour collecting baggage in the disembarking terminal plus the time to travel in the vehicle to their destination, and virion exposure could occur in each of these settings.

13. When estimating infections using Equations 1 and 2, note that threshold dose limits vary with individual occupants, so keeping exposure doses below HID50 values or any other such criterion does not mean no one will get sick at values far below that criterion. Further, the wide variation between measured HID50 values for influenza virus in the two studies referenced, depends not only on the species involved but also on the great variation that is possible. Much more research needs to be done here, and on the breath virion shedding from ill persons.

Several of these points were made months after the first edition of this article in a testimony to the U.S. House of representatives:[80]

"Like buildings, our means of transportation have not been designed to protect us from the risk of infection. Aircraft with their well-maintained systems that provide good ventilation and filtration of air still have proved vulnerable to infection transmission because of passenger density…"

"Aircraft cabins present an especially challenging environment because of the very high occupant density in a fully loaded aircraft"

"Data have shown the relative humidity in the range of 40-60% results in the most rapid decline in airborne virus viability.

- *In flight, relative humidity in aircraft cabins is typically below 30%.*
- *Most airline aircraft do not have humidification capability.*
- *If it is available increasing the humidity may provide a benefit.*
- *Any humidification should observe aircraft manufacturer guidance as elevated humidity in aircraft cabins has the potential to create safety concerns unrelated to disease transmission"*

"HEPA filters have demonstrated good virus removal efficiency, however the ability of the HEPA filters to remove viruses and other particles is directly related to the airflow through the filter"

"Airlines and manufacturers consistently claim that the high air exchange rate onboard aircraft protects passengers and crew from airborne exposure to viruses like COVID-19. While air exchange rate onboard is high, so too is the production of 'bioeffluent'…"

“ In the small space of the cabin, the rate of dilution of bioeffluent is consistently and considerably lower than in ground-based environments.”

“ While it is difficult to predict the effect for a given seat on a given aircraft, laboratory studies show that on the average, PAOs (gaspers) do provide some modest reduction of exposure”

I wrote similar peer reviewed papers on this same subject absent the humidity concern, that also investigated the roles of ACH and filtration in the spread of disease and with many of the same conclusions a decade ago that are being made in this article.[26,26,36,36]

The level of precaution to prevent respiratory system infections occurring during airplane travel during the COVID-19 period is higher than taken in many of the other setting examples but not as high as in hospitals. Currently masks are required in airports and aircraft as is screening, and accessibility (e.g., a sick person is more likely to go to the store than fly on an airplane). Before these measures were taken, aircraft travel on longer flights posed a higher risk of a person acquiring a virus infection compared with many other public spaces and their worst case scenarios. However, other scenarios could exceed the risk on the airplane. Obviously, the risk is related to prevalence and exposure duration where the infectious population is a large fraction of the total population.

So, what can you do personally when flying until major improvements are made in cabin ventilation and air filtration? Wear a mask. Get vaccinated. If there is an overhead gasper outlet, turn it on and point it between you and the passenger next to you. Finally, as respiratory rates are higher during boarding and disembarking than when seated, and lower while sleeping versus seated awake, relax whenever possible! [81,82]

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