Virus Transmission Modes and Mitigation Strategies, Part 2

Airborne Transmission And Distribution

BY JONATHAN BURKETT, P.E., HFDP, MEMBER ASHRAE

Part 1 of this article in last month's *ASHRAE Journal* looked at what makes up a virus, how it can be released into a space through droplets and aerosols, and the effect of certain environmental conditions like temperature and humidity on the droplets. Part 2 will look at airborne transmission and the effect of common air distribution methods on virus propagation.

Airborne Transmission

As mentioned in Part 1, as droplets evaporate the residual material contained in the droplet (mucus, dust, solids, virions, etc.) form droplet nuclei. These droplet nuclei can then remain suspended for long periods of time and be transmitted long distances from their origin. Airborne transmission can be further broken up into short-range and long-range transmission. Short-range airborne transmission occurs when two people are in close proximity (typically <2 m [6.6 ft]) and the aerosols are directly inhaled.

Long-range airborne transmission occurs when droplets evaporate to a size where they can remain suspended in the air for prolonged periods of time. These droplets can then be carried by air currents, thermal plumes, electrostatic charges and pressure differentials throughout the room or to connected rooms through the HVAC system.

A study by Chen, et al., found that when comparing large droplet transmission and short-range airborne transmission, the short-range airborne mode dominated the exposure risk for both talking and coughing.[1,2] The large droplets only beat out the short-range airborne mode when the droplets were larger than 100 µm and the subjects were close (≤0.2 m [7.9 in.] for talking and ≤0.5 m [20 in.] for coughing).[1]

The proximity to the infected person greatly affects the exposure risk.[2] Research has shown that at distances within 1 m to 1.5 m (3 ft to 5 ft) a substantial increase in exposure to droplet nuclei exhaled by the source patient exists.[2] General dilution ventilation does not directly affect these large droplets or short-range airborne modes of transmission when a susceptible person is in close proximity to the source.[2]

The importance of long-range airborne transmission in the spread of pathogens has been debated over the years, but viruses such as measles and chicken pox are thought to be capable of infections by long-range airborne transmission.[3] Other viruses, like coronaviruses

Jonathan Burkett, P.E., is a mechanical engineer at Buford Goff & Associates in Columbia, S.C.

and influenza, appear to have the capability for long-range airborne transmission under certain conditions.[3]

Viral Load and Infectious Dose

In 1955 Wells suggested the concept of quantal infection as a unit of measure of the infectious dose.[4] A quantum of infection is the minimum number of infectious airborne particles required to produce infection in a susceptible host.[5,6] Infectious quanta shouldn't be confused with the number of infectious particles released from the source (viral shedding). Quantum of infection is a measurement of the inhaled particles able to cause infection.[7] For some diseases, such as tuberculosis or smallpox, the infectious dose required to produce infection is low, even down to one organism.[8]

The idea of quantum of infection was used in the Wells-Riley equation, which assumes a well-mixed room (i.e., droplet nuclei are instantaneously and evenly distributed in a space). Wells defined the quantum of infection as being 63.2% of occupants infected when each occupant breathed one infective particle.[7] The major limitation for the Wells-Riley equation is in the estimation process of the quanta generation rate. This rate must be estimated from an outbreak case in which the attack rate is substituted back into the equation. This backward estimation assumes all infection cases are caused by airborne infections; influencing factors, such as survival rate, deposition rate, etc., can cause the rate to vary widely across different cases.[9]

A dose-response type model is a toxicological approach to assess infection risk that addresses some of the shortcomings of the Wells-Riley equation.[9] In the dose-response model, infectious dose data is required to construct the dose-response relationship.[10] This infectious dose data is sometimes only available for animals and requires extrapolation to adapt to humans. The dose-response model is more flexible than the Wells-Riley equation in that it can be used to model transport methods other than the airborne route, but it requires the dose data, which may not be known early on in an outbreak.

The basic reproduction number (R_0) is another popular metric (Table 1). R_0 is an indicator of the contagiousness or transmissibility of an infectious agent and is defined as the mean number of infections caused by an infected individual in a susceptible population.11 Values of R_0 greater than 1 indicate the infectious agent can start spreading in a population. In general, the higher the R_0 the harder it is to control the spread of disease if an epidemic breaks out.[12] While R_0 can be a useful metric, care must be taken to use the most recent data available to calculate it. Many reported R_0 still use obsolete data from outbreaks in the early 20th century, which may not be valid today.[12]

TABLE 1 Basic reproduction number (R_0) for various diseases.

DISEASE	REPRODUCTION NUMBER
Measles (Pre-Vaccination: 1912 – 1928)[12,32]	12 - 18
Mumps[32]	4 - 7
Ebola (2014 Outbreak)[33]	1.51 - 2.53
SARS-CoV-2 (COVID-19)[34]	1 - 2.8
Seasonal Influenza[35]	0.9 - 2.1

While not fully understood, some people can act as superspreaders; these people infect a disproportionately large number of susceptible contacts.[13] It is believed superspreading is a normal feature of disease spread,[11] and it has been linked to several outbreaks, such as the 2003 SARS-CoV outbreak in Hong Kong and the 2015 MERS-CoV outbreak in South Korea.[14] Not taking into account these superspreaders can skew an R_0 value based on population estimates.[11]

Additional Transmission and Transport Modes

An occupied space is a dynamic environment full of complex interactions between occupants, thermal plumes, vortices created by movement of people within the space, the ventilation equipment, and other environmental conditions. As a person moves around a space, the layer of air closest to the body is comparable to their walking speed.[5] This pushing of the front layer creates a volume flux of air and a wake bubble behind the person. At walking speeds above 0.2 m/s (0.45 mph), the thermal plume gives way to the created wake, which mixes strongly with the surrounding air, entraining room air and transporting it by the wake.[15] A person walking forward at 1 m/s (2.2 mph) would create a volume flux of about 255 L/s (540 cfm) with an attached wake of 76 L to 230 L (2.7 ft^3 to 8.1 ft^3) behind them.[5] Walking on carpet can also resuspend pathogens attached to dust from the surface.[10] These floor-level contaminants would now be smaller after evaporation and could then be transported by the thermal plume of someone standing nearby to the breathing zone.[15]

Sometimes equipment located in the space can be a source of particle generation, which can act as a vehicle to increase the carriage and intake of aerosolized droplets. Laser printers have been found to be the main source of particulate matter in office buildings (where tobacco smoking is not allowed).[16] When a laser printer makes copies, only about 75% of the toner material is effectively transferred to the drum. The remaining material can be released to the air as electrostatically charged fine particles that can remain airborne for a long time.[16] These charged particles can attract virus-laden aerosols, droplets and droplet nuclei that when inhaled are more likely to be deposited in the lungs due to their charge.[16]

Many preschool and kindergarten classrooms contain toilet rooms. These toilets can also be a source of droplet generation. Gravity flow, pressure-assisted gravity flow, and pressure valve systems have been compared, and each of these systems have been found to produce both droplets and droplet nuclei.[17] As water enters the bowl after flushing, the turbulence creates large droplets that can contaminate the toilet seat, surrounding floors and nearby surfaces. A toilet plume of aerosol particles (<3 µm) traveling at 5 m/s (16 fps) or greater is also produced that can entrain viruses and carry them away from the toilet and remain airborne for an extended period of time.[17,18] The vomit and feces of an infected person can contain high pathogen concentrations[19] and can continue to produce virus-laden aerosols even after several flushes.[17] In general, studies have shown greater aerosol production occurs with higher flush energy.[17] Pathogens like coronaviruses, norovirus and rotavirus have all been shown to be spread by the fecal-oral route.[18]

A recent study has also shown urinals may pose a similar issue to that of a toilet.[20] Urinals have long been discounted as a source of exposure, but researchers have recently extracted a SARS-CoV-2 virus particle from the urine of an infected patient.[20] When the urinal was flushed, more than 57% of the particles produced traveled away from the urinal, and the particles reached a height of 0.84 m (33 in) in only 5.5 seconds.[20] While the viral load of urine may require more research, saliva is commonly deposited in urinals and could be dispersed by the flush.

FIGURE 1 Zone air distribution effectiveness.[21]

$$E_z = \frac{C_e - C_s}{C - C_s}$$

where

E_z = Zone air distribution effectiveness
C = Average contaminant concentration at the breathing zone
C_e = Average contaminant concentration at the exhaust
C_s = Average contaminant concentration at the supply

Effect of Air Distribution and Pressure Differential

Air distribution can play an important role in the movement of droplet nuclei in the space. Many classrooms use mixing type ventilation in which high velocity cool air is supplied at the ceiling level and returned either at the ceiling level or the floor. Displacement ventilation and underfloor ventilation are two other types of ventilation methods often used in high performance schools.

Displacement ventilation is a stratified air system that supplies low speed, cool air near the floor level that slowly moves across the room. As this air reaches a heat source (person, equipment, etc.), the cold, dense air is warmed, creating a thermal plume that draws the clean air up the source, forcing polluted air up above the breathing zone where it can be returned. Underfloor ventilation introduces air from diffusers at the floor level, but unlike displacement ventilation, which relies on buoyancy, the air is typically supplied using high velocity jets, and mixing occurs in the occupied zone. Above the occupied zone, stratification can still occur and, similar to displacement ventilation, pollutants can be carried by the upward flow to a return in the ceiling.

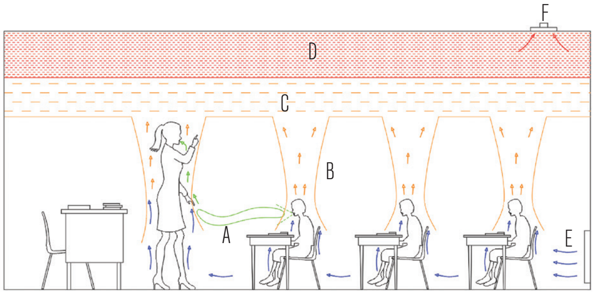
ASHRAE Standard 62.1-2019 defines the zone air distribution effectiveness (E_z) (*Figure 1*) as "the ratio of the change of contaminant concentration between the air supply and air exhaust to the change of contaminated concentration between the air supply and the breathing zone."[21] Table 6-4 in ASHRAE Standard 62.1-2019 lists the zone air distribution effectiveness of several systems. For an ideal mixed air system, the E_z equals 1.0. For a displacement ventilation system, Xie, et al., calculated an E_z of 1.3 for a classroom and 1.25 for an underfloor ventilation system.[22] Table 6-4 also shows that when the location of the return diffusers is above 5.5 m (18 ft), a higher E_z can be obtained. CFD modeling of these systems also shows indirect exposure under displacement ventilation is generally lower than the exposure under mixing ventilation.[23] In actual environments though, mixing ventilation isn't always fully mixed and stratification can still occur in parts of the room.

Displacement ventilation is also more complex. The lock-up effect can occur in which a secondary layer is formed directly below the upper, warm stratified layer, trapping exhaled breath below at an intermediate height.[24] This air eventually gets carried into the upper layer where it can be removed through the return (*Figure 2*). Studies have shown this secondary layer can even drop down to the inhalation height (although as mentioned in Part 1, most of the intake air comes from a lower height and is carried up by the body thermal plume).[25] When a person is laying on their back, displacement ventilation can offer very good protection from cross-infection.[26] However, when a person is sitting up or lying on their side, the stratification can cause exhalation to travel further horizontally.[23,27] As mentioned above, for displacement ventilation, locating the return higher can increase the air distribution effectiveness, but locating the return low can decrease the ventilation effectiveness to be much less than mixing ventilation.[28]

Displacement ventilation is also sensitive to the use of ceiling fans, personal fans and the movement of the occupants in the space. These can affect the stratification of the space.[24] The presence of a radiant wall (poorly insulated wall or window wall) has been shown to significantly affect the airflow pattern and contaminant dispersion under displacement ventilation. Locating a person between the radiant wall and contaminant source has a greater impact on the risk of infection than the return diffuser location or number of air changes.[25] The position of the infected person relative to the susceptible person also plays a role. When a seated infected person is located near a standing susceptible person, the chance of cross-infection is greater.[26]

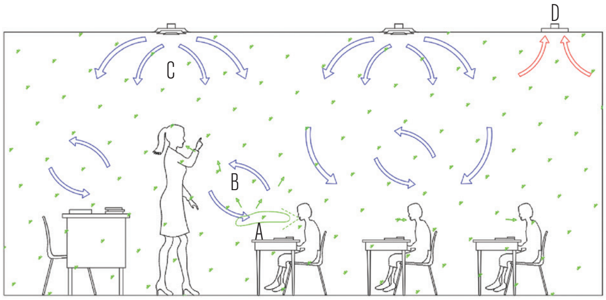
Mixing ventilation tends to create a uniform concentration of infected air in the room (*Figure 3*). For mixing ventilation, the exhaled breath's path can be interrupted by the supply airstream, which displaces these contaminants to the rest of the room.[29] Since the room is well mixed, the location of the return diffuser doesn't have a large impact on the contaminant removal.[28] Mixing ventilation can also supply more air changes to a space without drafts vs. displacement ventilation, which can affect the dilution of the particles in the air.[26]

FIGURE 2 Displacement ventilation.



A. Cough event directed forward.
B. Thermal plume upward.
C. Lock-up, secondary thermal layer.
D. Primary thermal layer.
E. Displacement ventilation supply diffuser.
F. Return diffuser in the ceiling.

FIGURE 3 Mixing ventilation.



A. Cough event directed forward.
B. Expiratory jet collides with airflow.
C. Mixing ventilation distributes the particles throughout the space.
D. Ceiling return.

Underfloor air distribution can provide local mixing of infected air, but still prevent contaminants from spreading throughout the space. These local diffusers mix the air upward to a stratified layer, similar to displacement ventilation, where they are then removed from the space by a return diffuser in the ceiling. Underfloor air distribution can provide better aerosol removal performance than mixing ventilation.[10,30]

Chen, et al., reviewed 10 scientific papers to create a database for the 191 cases of personal exposure to exhaled contaminants contained in the 10 scientific papers.[23] The papers were then used to validate a method for differentiating direct and indirect exposure to these contaminants. After normalizing this exposure data for mixing ventilation and displacement ventilation, the median exposure to exhaled contaminants for mixing ventilation was found to be 0.99 (close to a well-mixed condition, i.e., 1.0). Though displacement ventilation has a theoretical exposure value less than mixing ventilation, the researchers calculated a median exposure value of 1.23 (23% higher than a well-mixed condition). Chen, et al., attributed this higher-than-expected value to the inclusion of many cases with a short inter-person distance that negated the benefits of the displacement ventilation.[23]

One last concept used to control the spread of pathogens is pressure differential. ASHRAE Standard 170-2017 calls for a minimum pressure differential of 2.5 Pa (0.01 in. w.c.) across the envelope and between adjacent rooms for both airborne infectious isolation (AII) rooms and protective environment (PE) rooms.[31] While schools may not have the same requirements as an AII or PE room, areas such as the nurse's office and toilet rooms may benefit from the use of a negative pressure differential between these spaces and adjoining spaces to prevent virus migration.

Conclusions

This article looked at the airborne transmission of viruses and their propagation by the air distribution system. As discussed, displacement ventilation can provide a very high theoretical ventilation efficiency, but the presence of a lock-up layer and poor protection from expiratory jets may negate some of this ventilation efficiency in regard to virus exposure near an infected person. As discussed in Part 1, if the infected person is wearing a face mask, the ventilation efficiency could be much higher.[24] Mixing ventilation, in general, provides uniform exposure to contaminants in a space and the potential for greater spread at diluted levels.[24]

References

1. Chen, W., N. Zhang, J. Wei, H.-L. Yen, et al. 2020. "Short-range airborne route dominates exposure of respiratory infection during close contact." *Building and Environment* 176:106859. https://doi.org/10.1016/j.buildenv.2020.106859

2. Liu, L, Y. Li, P.V. Nielsen, J. Wei, et al. 2017. "Short-range airborne transmission of expiratory droplets between two people." *Indoor Air* 27(2):452–462. https://doi.org/10.1111/ina.12314

3. Tellier, R., Y. Li, B.J. Cowling, et al. 2019. "Recognition of aerosol transmission of infectious agents: a commentary." *BMC Infect Dis* 19:101. https://doi.org/10.1186/s12879-019-3707-y

4. Wells, W. 1955. *Airborne Contagion and Air Hygiene; An Ecological Study of Droplet Infections*. Cambridge: Harvard University Press.

5. Li, Y., G.M. Leung, J.W. Tang, X. Yang, et al. 2007. "Role of ventilation in airborne transmission of infectious agents in the built environment—a multidisciplinary systematic review." *Indoor Air* 17(1):2–18. https://doi.org/10.1111/j.1600-0668.2006.00445.x

6. Tang, J.W., Y. Li, I. Eames, P.K. Chan, et al. 2006. "Factors involved in the aerosol transmission of infection and control of ventilation in healthcare premises." *J Hosp Infect*. 64(2):100–14. https://doi.org/10.1016/j.jhin.2006.05.022

7. Nardell, E. 2016. "Wells revisited: infectious particles vs. quanta of *Mycobacterium tuberculosis* infection–don't get them confused." *Mycobacterial Diseases* 6(5). https://doi.org/10.4172/2161-1068.1000231

8. Nicas, M., W.W. Nazaroff, A. Hubbard. 2005. "Toward understanding the risk of secondary airborne infection: emission of respirable pathogens." *Journal of Occupational and Environmental Hygiene* 2(3):143–154. https://doi.org/10.1080/15459620590918466

9. Yu, H.C. 2016. "An empirical drag coefficient model for simulating the dispersion and deposition of bioaerosol particles in ventilated environments." Doctoral Dissertation. Hong Kong Polytechnic University. http://hdl.handle.net/10397/85573

10. Aliabadi, A.A., S.N. Rogak, K.H. Bartlett, S.I. Green. 2011. "Preventing airborne disease transmission: review of methods for ventilation design in health care facilities." *Adv Prev Med*. 2011:124064. https://doi.org/10.4061/2011/1240642

11. Lloyd-Smith, J.O., S.J. Schreiber, P.E. Kopp, W.M. Getz. 2005. "Superspreading and the effect of individual variation on disease emergence." *Nature* 438:355–359. https://doi.org/10.1038/nature04153

12. Delamater, P.L., E.J. Street, T.F. Leslie, Y. Yang, et al. 2019. "Complexity of the basic reproduction number (R_0)." *Emerging Infectious Diseases* 25(1):1–4. https://dx.doi.org/10.3201/eid2501.171901

13. Asadi, S., A.S. Wexler, C.D Cappa, et al. 2019. "Aerosol emission and superemission during human speech increase with voice loudness." *Sci Rep* 9:2348. https://doi.org/10.1038/s41598-019-38808-z

14. Wong, G., W. Liu, Y. Liu, B. Zhou, et al. 2015. "MERS, SARS, and Ebola: the role of super-spreaders in infectious disease." *Cell Host Microbe* 18(4):398–401. https://doi.org/10.1016/j.chom.2015.09.013

15. Tang, J. W., T.J. Liebner, B.A. Craven, G.S. Settles. 2009. "A schlieren optical study of the human cough with and without wearing masks for aerosol infection control." *Journal of the Royal Society, Interface* 6(Issue Suppl 6):S727–S736. https://doi.org/10.1098/rsif.2009.0295.focus

16. He, S., J. Han. 2020. "Electrostatic fine particles emitted from laser printers as potential vectors for airborne transmission of COVID-19." *Environmental Chemistry Letters* 1–8. https://doi.org/10.1007/s10311-020-01069-8

17. Johnson, D., R. Lynch, C. Marshall, K. Mead, et al. 2013. "Aerosol generation by modern flush toilets." *Aerosol Science and Technology* 47(9)1047–1057. https://doi.org/10.1080/02786826.2013.814911

18. Li, Y.Y., J.X. Wang, X. Chen. 2020. "Can a toilet promote virus transmission? From a fluid dynamics perspective." *Physics of Fluids* 32(6):065107. https://doi.org/10.1063/5.0013318

19. Johnson, D.L., K.R. Mead, R.A. Lynch, D.V. Hirst. 2013. "Lifting the lid on toilet plume aerosol: a literature review with suggestions for future research." *Am J Infect Control* 41(3):254–258. https://doi.org/10.1016/j.ajic.2012.04.330

20. Wang, J.X., Y.Y. Li, X.D. Liu, X. Cao. 2020. "Virus transmission from urinals." *Physics of Fluids* 32(8):081703. https://doi.org/10.1063/5.0021450

21. ANSI/ASHRAE Standard 62.1-2019, *Ventilation for Acceptable Indoor Air Quality*.

22. Lee, K.S., Z. Jiang, Q. Chen. 2009. "Air distribution effectiveness with stratified air distribution systems." *ASHRAE Transactions* 115(2).

23. Chen, C., B. Zhao, D. Lai, W. Liu. 2018. "A simple method for differentiating direct and indirect exposure to exhaled contaminants in mechanically ventilated rooms." *Building Simulation* 11(5):1039–1051. https://doi.org/10.1007/s12273-018-0441-0

24. Bhagat, R.K., M.S. Davies Wykes, S.B. Dalziel, P.F. Linden. 2020. "Effects of ventilation on the indoor spread of COVID-19." *Journal of Fluid Mechanics* 903:F1. https://doi.org/10.1017/jfm.2020.720

25. Villafruela, J.M., I. Olmedo, F.A. Berlanga, M. Ruiz de Adana. 2019. "Assessment of displacement ventilation systems in airborne infection risk in hospital rooms." *PloS One* 14(1):e0211390. https://doi.org/10.1371/journal.pone.0211390

26. Nielsen, P.V. 2012. "Air distribution systems and cross-infection risk in the hospital sector." *Ventilation 2012: The 10th International Conference on Industrial Ventilation*.

27. Nielsen, P.V. 2009. "Control of airborne infectious diseases in ventilated spaces." *Journal of the Royal Society, Interface* 6(Issue Suppl_6):S747–S755. https://doi.org/10.1098/rsif.2009.0228.focus

28. Yi, Y., W. Xu, J.K. Gupta, A. Guity, et al. 2009. "Experimental study on displacement and mixing ventilation systems for a patient ward." *HVAC&R Research* 15(6):1175–1191. https://doi.org/10.1080/10789669.2009.10390885

29. Memarzadeh, F., W. Xu. 2012. "Role of air changes per hour (ACH) in possible transmission of airborne infections." *Build. Simul*. 5:15–28. https://doi.org/10.1007/s12273-011-0053-4

30. Bolashikov, Z.D., A.K. Melikov. 2009. "Methods for air cleaning and protection of building occupants from airborne pathogens." *Build. Environ.* 44(7):1378–1385. https://doi.org/10.1016/j.buildenv.2008.09.001

31. ANSI/ASHRAE/ASHE Standard 170-2017, *Ventilation of Health Care Facilities*.

32. Paul, E., M. Fine. 1993. "Herd immunity: history, theory, practice." *Epidemiologic Reviews* 15(2):265–302. https://doi.org/10.1093/oxfordjournals.epirev.a036121

33. Althaus, C.L. 2014. "Estimating the reproduction number of Ebola virus (EBOV) during the 2014 outbreak in West Africa." *PLoS Currents Outbreaks* 1. https://doi.org/10.1371/currents.outbreaks.91afb5e0f279e7f29e7056095255b288

34. Al-Raeei, M. 2021. "The basic reproduction number of the new coronavirus pandemic with mortality for India, the Syrian Arab Republic, the United States, Yemen, China, France, Nigeria and Russia with different rate of cases." *Clinical Epidemiology and Global Health* 9:147–149. https://doi.org/10.1016/j.cegh.2020.08.005

35. Coburn, B.J., B.G. Wagner, S. Blower. 2009. "Modeling influenza epidemics and pandemics: insights into the future of swine flu (H1N1)." *BMC Med* 7:30. https://doi.org/10.1186/1741-7015-7-30 ■

Advertisement formerly in this space.