HVAC filtration and the Wells-Riley approach to assessing risks of infectious airborne diseases

Final Report

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Table of Contents

Introduction

The airborne transmission of respiratory pathogens such as measles, tuberculosis, severe acute respiratory syndrome (SARS), influenza, common colds, and others in indoor environments and the associated risk of infection presented to uninfected individuals are governed by several complex physical and biological processes. Communicable respiratory illnesses lead to large excesses in expenses associated with health care, absence from work, and lost worker productivity (Fisk, 2000), but the control of airborne infectious disease transmission in indoor environments is not yet entirely understood. Several studies have shown that building-related factors such as increased outdoor air ventilation rates, lower occupant density, and use of UV germicidal irradiation can indeed reduce the risk of infectious disease transmission inside buildings (Langmuir et al., 1948; Milton et al., 2000; Sun et al., 2011). However, because these mechanisms all work to fundamentally control airborne infectious particle concentrations indoors, might commonly available particle filters in recirculating heating, ventilating, and air-conditioning (HVAC) systems also reduce the risk of infectious disease while potentially requiring less energy than some other alternatives?

To help address this question, this report (i) reviews existing literature on the physical, biological, and epidemiological factors that influence the transmission of airborne infectious diseases; (ii) connects relevant findings from the review and a standard Wells-Riley risk model to existing knowledge of HVAC particle filtration standards (i.e., MERV from ASHRAE Standard 52.2); and (iii) describes and applies a modified Wells-Riley methodology for estimating the impact of commonly available HVAC filtration on the spread of infectious disease in buildings using individual case studies of airborne influenza, rhinovirus, and tuberculosis transmission in hypothetical office, school classroom, and hospital waiting room environments. The overall intent is to develop a method to predict the impacts of commonly available HVAC filters on the risks associated with airborne infectious diseases. Several recommendations for future research are also presented.

Modes of disease transmission

There has been a long-standing debate about the dominant methods of spreading certain infectious diseases, which can be transmitted in at least three separate ways:

1. Inhalation of airborne infectious particles (i.e., “infectious aerosols”);
2. Direct contact with pathogen sources; and
3. Contact with contaminated object surfaces (i.e., “fomites”).

Both the inhalation and surface-contamination routes are impacted in large part by the size of infectious particles emitted from an infected person. Larger particles (e.g., those greater than 10 μm in aerodynamic diameter) will settle out of the air rapidly, decreasing the airborne concentration but contaminating the surface on which they land. The surface on which particles deposit may include inanimate objects or parts of a receptor’s body, such as the nose, mouth, or eyelids.

Smaller particles (e.g., those smaller than 10 μm) will tend to remain airborne for longer periods of time, which increases the importance of airborne transmission and inhalation. For the

purposes of this work involving indoor air filtration, we will consider disease transmission only through airborne infectious particles (i.e., infectious aerosols). Airborne aerosol transmission has been shown to be a predominant route of transmission for a number of infectious diseases that affect a large fraction of the population, including rhinovirus (Dick et al., 1987; Sun et al., 2011), influenza (Moser et al., 1979; Tellier, 2009), tuberculosis (Escombe et al., 2007a), and SARS (Wong et al., 2004). There is also growing evidence that increased outdoor air ventilation rates can reduce the transmission of these same diseases (Li et al., 2007), which further confirms the importance of airborne transmission.

When an individual coughs, sneezes, speaks, or even breathes, particles consisting of liquid water, proteins, salts, and various other organic and inorganic matter (or "*droplets*") are expelled into the air. If the person is infected with a particular respiratory tract infection, those droplets are also composed of smaller infectious particles themselves, which may include viruses or bacteria depending on the type of infection. Note that viruses are typically an order of magnitude smaller in size than bacteria: ~30-200 nm for viruses vs. ~200-1000+ nm for bacteria (Kowalski and Bahnfleth, 1998). In exploring the likely impacts of particle filtration on virus and bacteria disease transmission, some researchers have assumed that the individual virus or bacteria particles are aerosolized and exist suspended as individual organisms (Kowalski and Bahnfleth, 2002; RTI, 2004); however, it is likely more appropriate to consider the particles as larger expelled droplets that contain aggregates of the smaller infectious particles within (Nardell, 2001; Nicas et al., 2005; Verreault et al., 2008). This report will thus focus primarily on the latter: expelled droplets containing infectious fragments within.

Once expelled from a very high relative humidity environment (the human body) to a relatively less humid environment (most indoor environments), droplets rapidly decrease in size as the surrounding liquid evaporates. Figure 1 describes this process visually (Verreault et al., 2008). Several studies have shown that this liquid evaporation typically occurs within less than one second of emission, depending on the original particle size and ambient thermodynamic conditions (Nicas et al., 2005; Chen and Zhao, 2010). In general, particles smaller than ~50 μm in diameter are predicted to reach this rapid (< 1 sec) equilibrium state. After rapid evaporation, a "*droplet nucleus*" containing the mix of solid particles (including any infectious particles) remains. Droplet nuclei typically have particle diameters that are 40-50% of the original droplet size (Nicas et al., 2005; Yang and Marr, 2011).

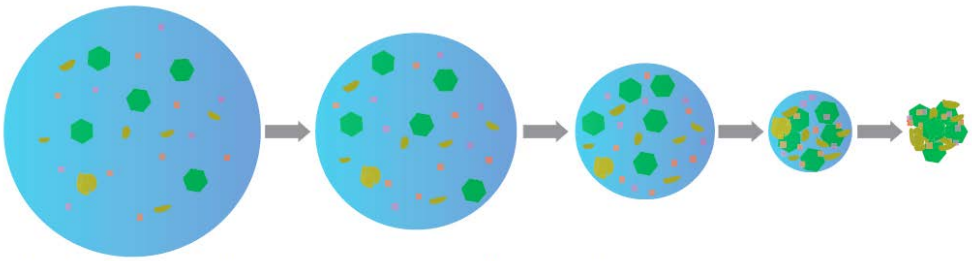

Figure 1. Evaporation of a liquid expelled *droplet* to a *droplet nucleus* (Image source: Verreault et al., 2008)

Because of these phenomena of emission followed by rapid evaporation, it is generally appropriate to consider the control of airborne infectious particles in terms of the control of *droplet nuclei*. Because the transport and control mechanisms of any aerosol are primarily functions of particle size (Hinds, 1999), it is important to first consider the particle size

distributions of droplet nuclei. These particle size impacts are also important for direct surface- and body-contamination cases; however, the receptor mechanisms (i.e., dermal or mucosal uptake) are different from the mechanism considered in this report (inhalation) and thus these modes of transmission are outside of the scope of this report.

Particle size distributions of droplet nuclei

It is commonly believed that droplet nuclei particles average 1 to 3 μm in diameter (Nardell, 2001), although several recent studies have shown considerable variation in the size distribution of expelled droplets and droplet nuclei. The size and number of expelled droplets can vary greatly according to the person and their activity (Edwards et al., 2004), as well as with the type of infection they have (recall that bacteria are larger than viruses, so it is reasonable that droplet nuclei containing bacteria particles are also larger). Even the measurement technique used to explore the emission of droplet nuclei affects the resulting size distribution, which is important to consider because many of these previous studies occurred in the 1950s or earlier and may have been limited by instrumentation.

Nicas et al. (2005) provide a detailed summary of nearly 50 years of measurements of particle size distributions of droplets (and droplet nuclei) emitted by humans during coughing and sneezing. These studies, with their wide range of methodologies, show a wide range in the mean particle diameter emitted during coughing and sneezing activities. Nicas et al. (2005) summarize these studies and conclude that cough-generated aerosols could be divided into two lognormally distributed modes: (i) a small particle distribution with a geometric mean diameter of ~10 μm and geometric standard deviation (GSD) of 9.0 and (ii) a large particle distribution with a geometric mean diameter of ~160 μm and GSD of 1.7. However, the smaller particle distribution was found to contain the majority of cough particles (~70%). Given that the same authors described some evidence of smaller particles being more infectious than larger particles, it appears that smaller (i.e., < 10 μm) particles are likely of greatest concern for droplet nuclei transmission of infectious diseases.

Several more recent studies utilizing more advanced measurement techniques have revealed a general consensus that the majority (often 80-90%) of particles expelled during various human activities are actually smaller than 1-2 μm in diameter, although questions remains about the relative importance of particle number, surface area, volume, and overall infectivity. The next sections begin by reviewing previous studies on aerosol size distributions expelled during human activities.

Recent studies on aerosols expelled during *coughing*

Papineni and Rosenthal (1997) measured particle size distributions in the exhaled breath of 5 human subjects using an optical particle counter, which allows for characterization of particles from 0.3-10 μm (note that Nicas et al. 2005 also included this study in their review). Measurements were made during four activities: mouth breathing, nose breathing, coughing, and talking. During each of the four activities, the average fraction of aerosolized particles less than 1 μm was 84-90%. The size distribution did not change significantly between activities, although coughing emitted a larger number of particles than the other three activities, on average. There was also considerable variation in the absolute numbers of particles aerosolized between the five

subjects (the difference in <1 μm concentrations from the highest emitter to the lowest emitter was a factor of ~8). These results suggest that (i) most expelled airborne droplets may be reasonably assumed to be smaller than 1 μm and that (ii) some people may act as "super-spreaders" not only because of the chemical and biological composition of their exhaled breath droplets but also because of the large absolute number concentrations of droplets emitted.

In a similar study, Yang et al. (2007) measured the size distribution of droplets and droplet nuclei during coughing by 54 human test subjects using an aerodynamic particle sizer (APS) and a scanning mobility particle sizer (SMPS), which allows for characterization of particles from ~20 nm to ~30 μm in diameter. The total average size distribution of the droplet nuclei was contained within 0.58 to 5.42 μm particles, with 82% of particles existing in the 0.74-2.2 μm size range. Variations in size between age groups and gender groups were not significant, although concentrations from male subjects (and from 30-50 year olds) were higher.

Morawska et al. (2009) measured size-resolved particle concentrations with an aerodynamic particle sizer during several activities performed by human subjects ranging from breathing to coughing, which allowed for characterization of ~0.5 to 20 μm particles. For most activities, the vast majority of particles expelled were associated with a lognormal distribution with a count median diameter of ~0.8 μm. This mode accounted for ~84% of the total number of particles generated on average for both breathing and coughing. Unfortunately, other distribution statistics were not reported in their work, which makes it difficult to interpret their findings in the same manner as the two previously mentioned studies.

Most recently, Lindsley et al. (2012) measured aerosol size distributions emitted by nine individuals during coughing using a laser particle counter, which allowed for characterization of ~0.3 to 10 μm particles. Measurements were performed both while the patients were infected with influenza and again after they recovered. The number of particles produced per cough was higher when subjects had influenza than when subjects had recovered, although the difference was not statistically significant. The authors reported a count median diameter (CMD) of approximately 0.63 μm with a GSD of 1.54 to 1.83, which is generally in agreement with Yang et al. (2007), albeit with a somewhat smaller mean diameter.

Relative size fractions of particle distributions from three of the four aforementioned studies are compared in Table 1.

Table 1. Summary statistics for the coughing distributions in Papineni and Rosenthal (1997), Yang et al. (2007), and Lindsley et al. (2012)

	% of total number concentration during coughing		
Particle size	Yang et al. (2007)	Lindsley et al. (2012)	Papineni and Rosenthal (1997)
0.3 to 0.5 μm	1%	2%	
0.5 to 0.7 μm	7%	23%	88%
0.7 to 1.0 μm	33%	56%	
1 to 2 μm	57%	19%	12%
2 to 3 μm	2%	0%	
Total	100%	100%	100%

Table 1 shows that approximately 41% of particles emitted during coughing in Yang et al. (2007) were in the 0.3-1 μm size range; approximately 57% were in the 1-2 μm size range (and 59% in the 1-3 μm size range). Conversely, Lindsley et al. (2012) differed in that ~81% of particles were detected in the 0.3-1 μm size range while only ~19% were detected in the 1-2 μm size range (and none in the 2-3 size range). Finally, Papineni and Rosenthal (1997) detected ~88% of expelled particles in the 0.3-1.0 μm size range, while only ~12% were detected in the 1-3 μm size range. Differences between these three studies may be attributed to a combination of differences in study design, test subjects, and instrumentation. Regardless, these studies generally align in that they all show that the majority of particles emitted during coughing are smaller than 2 μm, and often smaller than 1 μm in diameter.

Recent studies on aerosols expelled during *normal breathing*

Aerosols are also expelled during activities other than coughing, albeit typically in lower absolute number concentrations. For example, Fabian et al. (2008) measured airborne particle concentrations in the exhaled breath of patients with the flu. Nearly 90% of particles exhaled during normal tidal breathing were smaller than 1 μm. On average, ~70% of particles in exhaled breath were 0.3-0.5 μm; ~20% were 0.5-1.0 μm; and ~15% were 1-5 μm, as shown in Figure 2. The total particle number concentrations during these breathing activities were also quite low relative to previous cough studies. Additionally, particle sizes were smaller than those emitted during previous coughing studies, which suggests that coughing indeed generates larger particles in larger number concentrations than normal breathing. This is general in agreement with Papineni and Rosenthal (1997).

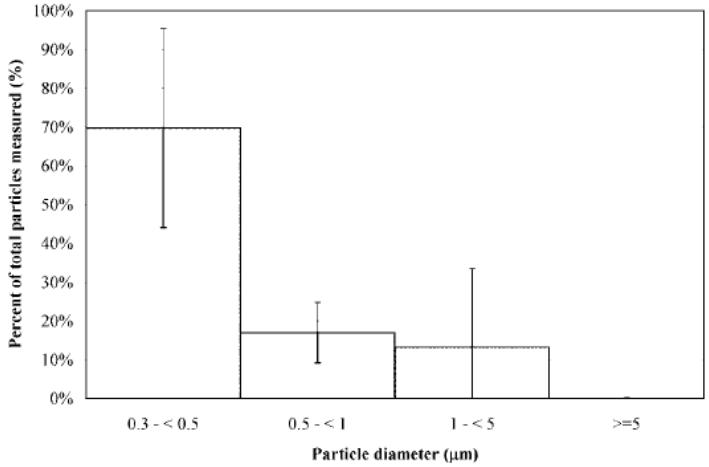

Figure 2. Exhaled breath size distribution from influenza infected subjects (Image source: Fabian et al., 2008)

Similar tidal breathing measurements were later made using subjects infected with human rhinovirus (the cause of the common cold). Across all infected subjects, approximately 80% of the total particles emitted were associated with sizes 0.3 to 0.5 μm (Fabian et al., 2011). Results again showed the importance of "super-spreaders," in that approximately 24% of the patients were responsible for exhaling approximately 81% of the total particles emitted. However, total number concentrations during normal breathing remained much lower than those generated during coughing studies.

More controlled experiments using ferrets (which are reasonable surrogates for human studies) generally confirm these previous experiments. For example, Gustin et al. (2011) reported a count

median aerodynamic diameter (CMAD) of ~1 μm (GSD = 1.98) for normal breathing and a CMAD of ~0.9 μm (GSD = 1.75) for sneezing in ferrets. Higher peak aerosol concentrations were observed during sneezing. The total number of particles smaller than 1 μm was approximately 53% for breathing and 58% for sneezing, again suggesting that the majority of expelled particles (droplet nuclei, post-evaporation) are smaller than 1 μm, albeit with some variation.

Several other researchers have utilized SMPS measurements to characterize droplet nuclei distributions expelled during different forms of breathing. Some of these studies suggest that the majority of particles expelled are actually smaller in diameter than previously observed. For example, Haslbeck et al. (2010) measured particle sizes expelled (and subsequently aged) by 16 healthy human subjects predominantly in the submicron range with a count-median diameter of ~0.4 μm. In a similar study of 16 human subjects, Holmgren et al. (2010) observed peaks during tidal breathing as low as 70 nm (0.07 μm with GSD = 2.0), with an additional broad peak between 0.2-0.5 μm during airway closure. Particle concentrations were generally higher during the latter airway closure period.

Infectious particles within droplet nuclei

While previous studies have been helpful for identifying the size of particles expelled during human activities, several more recent studies have utilized more sophisticated techniques to also identify the actual presence of viruses and bacteria in expelled droplets and droplet nuclei. These studies offer insight not only into what size aerosols exist after expulsion from the human body, but in what size-fractions are viruses or bacteria actually present.

For example, Fabian et al. (2008) measured influenza virus and airborne particle concentrations in the exhaled breath of patients with the flu. They found influenza virus RNA in the exhaled breath of 60% of those infected with influenza A. Similar results later confirmed mere breathing as a source of airborne viruses from infected patients (Stelzer-Braid et al., 2009). Blachere et al. (2009) measured size-fractioned aerosol particles in a hospital to test for airborne influenza virus using real-time polymerase chain reaction (PCR: a method to amplify and quantify targeted DNA molecules). They found that ~53% of the detectable influenza virus particles they found were within the respirable aerosol fraction (i.e., less than 4 μm). More specifically, 46% were in the >4 μm stage; 49% were found on the 1-4 μm stage; and 4% were collected on the back-up filter (<1 μm). Similarly, Lindsley et al. (2010) collected size-fractioned airborne particles in different locations within an urgent care clinic and determined the amount of airborne virus RNA in different size regimes using real-time PCR. Their results are summarized in Table 2 below.

Table 2. Size distribution of influenza RNA measured in Lindsley et al. (2010)

Sampling Location	Distribution of viral RNA		
Personal samplers	< 1.7 μm	1.7-4.9 μm	> 4.9 μm
	32%	16%	52%
Lower stationary samplers	< 1 μm	1-4.1 μm	> 4.1 μm
	13%	37%	50%
Upper stationary samplers	< 1 μm	1-4.1 μm	> 4.1 μm
	9%	27%	64%

Although the size-fractions between personal samplers and stationary samplers differed slightly in Lindsley et al. (2010), ~10-30% of influenza RNA was found in particles < 2 μm; ~20-40% was found in particles ~1 to ~4 μm; and ~50-60% was found in particles > 4 μm, on average, in the health care facility.

Lindsley et al. (2010a) measured influenza viral RNA expelled during coughing by infected human test subjects with similar aerosol monitoring equipment as Lindsley et al. (2010). Influenza RNA was detected in particles expelled by 81% of the infected subjects. Approximately 35% of the influenza RNA was contained in particles > 4 μm; 23% in particles 1-4 μm; and 42% in particles < 1 μm.

Most recently, Yang et al. (2011) measured the size distribution of airborne influenza A viruses in a healthcare center, a day-care facility, and onboard airplanes. They found that 64% of the detected viral genome copies were associated with particles smaller than 2.5 μm in diameter, on average. The mean relative amounts of virus found in the <0.25 μm, 0.25-0.5 μm, 0.5-1.0 μm, 1.0-2.5 μm, and >2.5 μm size fractions were 15%, 10%, 11%, 28%, and 36%, respectively, although no trend in size distribution was observed across the different sampling locations.

These previous studies all confirm that aerosols generated during coughing by influenza patients and subsequently remaining suspended in indoor environments indeed contain the influenza virus and that much of that viral RNA is contained within particles in the respirable size range (i.e., <4 μm). However, whereas ~100% of the number of particles emitted during the aforementioned coughing and breathing studies were smaller than 4 μm in size, only 40-70% of the influenza virus RNA is typically detected on particles in this size range (Blachere et al., 2009; Lindsley et al., 2010; Lindsley et al., 2010a), suggesting that the virus content of aerosols may actually be skewed toward larger particles. A bias toward the presence of virus particles contained in larger particle size fractions also suggests that particle surface area or volume may be more appropriate for characterizing infectious aerosols, which is reasonable considering that a 5 μm particle would have a volume approximately 100 times greater than a 1 μm particle and thus likely store a greater amount of virus particles inside droplet nuclei. Although there is considerable uncertainty in all of these measurements and sample sizes remain limited, these studies provide important insight into what size of expelled and suspended particles actually contain virus particles.

At this point, we have enough knowledge of the nature of particle sizes and numbers emitted during various human activities, and the likelihood of those aerosols containing virus content, to inform a preliminary mechanistic study of droplet nuclei transport and control based on particle characteristics alone. However, to continue exploring the overall impact that HVAC filtration may have on infectious disease transmission, the next section reviews existing literature related to methods of quantifying actual *risks* of acquiring airborne infectious diseases.

Risks of airborne infectious diseases

One often-used approach to quantifying the risks associated with airborne transmission of respiratory diseases is the Wells-Riley model (Riley et al., 1978). The Wells-Riley model is based on a concept of "quantum of infection," whereby the rate of generation of infectious airborne particles (or *quanta*) can be used to model the likelihood of an individual in a steady-state well-mixed indoor environment being exposed to the infectious particles and subsequently succumbing to infection. The Wells-Riley equation was originally derived according to Equation 1. Note that other researchers have also expanded this model to include dynamic, or time-varying, exposures (Gammaitoni and Nucci, 1997).

$$P_{\text{infection}} = \frac{cases}{susceptiles} = 1 - e^{-\frac{Iqpt}{Q_{\text{oa}}}} \quad (1)$$

where
$P_{\text{infection}}$ = the probability of infection
cases = the number of infection cases
susceptibles = number of susceptible individuals
I = number of infector individuals
p = pulmonary ventilation rate of a person (m^3/hour)
q = quanta generation rate (1/hr)
t = exposure time (hr)
Q_{oa} = room ventilation rate with clean air (m^3/hour)

It is important to note that the unit "*quantum of infection*" in this risk model is not an actual physical unit; it is a hypothetical infectious dose unit that is typically back calculated from epidemiological studies. It describes the number of infectious particles/pathogens in a way that implicitly includes both the amount of particles generated in time and the infectivity of particles (which also inherently captures particle size effects and probability of deposition in appropriate regions of the respiratory system).

The existing literature on quanta generation rates (q) is quite limited, and varies according to the type of disease and the original epidemiological case study. Published values of q for several infectious aerosols include:

- Influenza: ~15 to ~500 per hour (67 and 100 per hour are both commonly used)
  (Rudnick and Milton, 2003; Liao et al., 2005; Beggs et al., 2010; Sze To and Chao, 2010)
- Rhinovirus: ~1 to ~10 per hour
  (Rudnick and Milton, 2003)
- Tuberculosis: ~1 to ~50 per hour (~13 per hour has been commonly used)
  (Nardell et al., 1991; Escombe et al., 2007; Beggs et al., 2010; Chen et al., 2011)
- SARS: ~10 to ~300 per hour
  (Liao et al., 2005; Qian et al., 2009)
- Measles: ~570 to ~5600 per hour
  (Riley et al., 1978)

These values suggest that the combination of the number of expelled particles and their infectivity, size-resolution, and deposition in human lungs all combine to characterize the potency of these diseases approximately as: measles > influenza and SARS > tuberculosis > rhinovirus. Values for *q* for four of these infectious aerosols are also summarized in Figure 3.

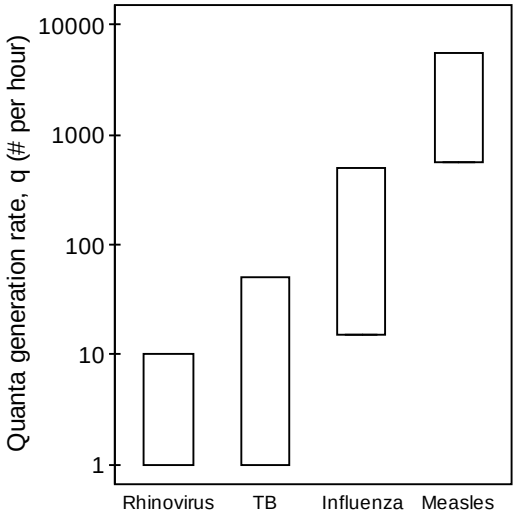

Figure 3. Ranges of quanta generation rates (*q*) for four infectious aerosols observed in the existing literature

It should be noted that there are also more physically and biologically grounded models for assessing risk of infectious respiratory diseases, such as dose-response (D-R) models that incorporate mass balances on the actual infectious particle doses (i.e., not just droplet nuclei), the "infectivity" of the particles, and the organism's susceptibility to disease (Sze To and Chao, 2010). D-R models have been used in a few recent studies to model infectious disease transmission using more mechanistic properties of both tuberculosis (Jones et al., 2009) and influenza (Yang and Marr, 2011). However, dose-response models also have their own inherent limitations, including often requiring interspecies extrapolation to estimate susceptibility of a human subject. In accordance with a long history of existing studies, this report will continue to rely on a modification of the Wells-Riley model. Further work should also incorporate similar analyses using dose-response methodologies.

It is also important to note the assumption of well-mixed indoor environments in this model and in previous derivations of quanta generation rates. This assumption ignores the importance of close-contact airborne spread of infectious diseases that occurs when expelled infectious aerosols deposit directly on susceptible parts of the human body. However, quanta generation rates are usually back calculated from scenarios where long-range transport was obviously a major factor. For example, Rudnick and Milton (2003) estimated quanta generation rates of 15-128 per hour (depending on steady-state or dynamic assumptions) using data from a 56-seat passenger airplane that was grounded for 4.5 hours. One known infector and 29 other passengers remained on the plane for the duration of the delay and 25 (86%) ultimately contracted influenza. While the quanta generation rates in this case may have accounted for some close-range disease transport, it is reasonable to assume that many passengers would have been infected by longer-range aerosol transport.

Despite some of these limitations, the Wells-Riley model has been used previously to show that some building factors, particularly outdoor air ventilation rates, can be an important removal mechanism for airborne infectious agents (Chen et al., 2006; Escombe et al., 2007). Because the risk model in Equation 1 is not normalized for indoor air volumes, the equation is limited to use only during specific case studies. As an example, Figure 4 shows the relationship between probability of infection, outdoor air ventilation rate, and quanta generation rate using the Wells-Riley model for a hypothetical 500 m^2 (5,300 ft^2) office building where adults with a pulmonary ventilation rate of 0.48 m^3/hr (approximately the average adult inhalation rate in U.S. EPA, 2011) are continuously in the presence of one infected individual for 8 hours. One can see that no amount of outdoor air ventilation could stop the most infectious disease from spreading (hypothetical q = 10,000/hr) while the risk of acquiring the common cold (rhinovirus, with q = 10/hr) could be reduced from ~10% in a tight building (AER = 0.3/hr) to only ~0.5% by increasing ventilation rates to ~6/hr.

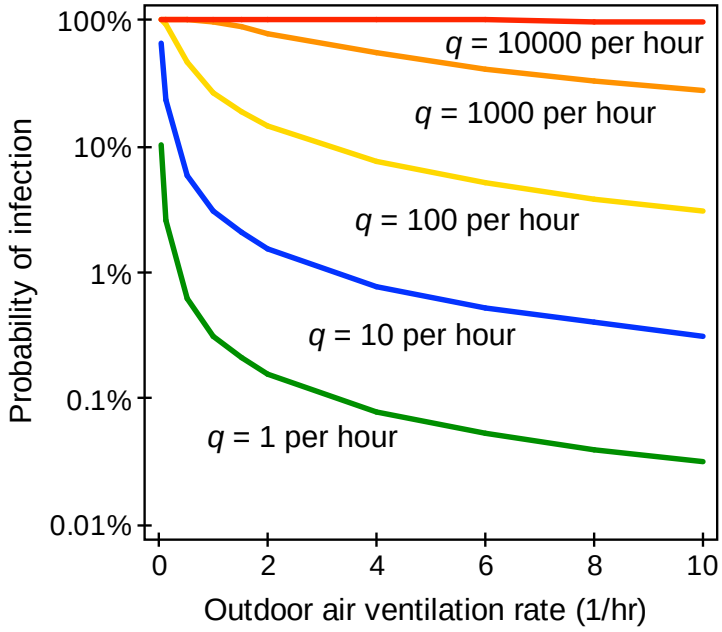

Figure 4. Probability of infection vs. outdoor air ventilation rates for a wide range of quanta generation rates if an average adult (p = 0.48 m^3/hr) spends 8 hours in a hypothetical 500 m^2 (5,400 ft^2) office building (V = 1,200 m^3 = 42,000 ft^3) with one infected occupant present the entire day

Note that if this hypothetical office building had a typical occupant density of 25 workers, ASHRAE Standard 62.1 would require the minimum outdoor air ventilation rate to be ~450 cfm (~750 m^3/hr), which translates to ~0.6 air changes per hour. At 0.6 air changes per hour and under the aforementioned conditions, the probability of an individual acquiring the influenza virus from an infected individual would be ~40% (assuming q = 100/hr). In this case, 9-10 workers would acquire the flu virus (40% of 24 susceptible workers). Increasing the outdoor air ventilation to 4 per hour would reduce the number of infected workers to ~2 individuals (~8% of 24 susceptible workers).

Incorporating other loss terms into the Wells-Riley equation

From a physical mass balance perspective, HVAC filtration is easily equated with outdoor air ventilation because they both can be used to reduce indoor concentration of airborne particles. In fact, the steady-state Wells-Riley equation has previously been modified in other investigations to include additional removal terms other than outdoor air ventilation, including filtration by personal respirators, UV degradation, particle deposition, and HVAC filtration (Fennelly and Nardell, 1998; Nazaroff et al., 1998; Fisk et al., 2005), as shown in Equation 2.

$$P_{\text{infection}} = 1 - \exp\left[-\frac{Iqpt}{V} \Big/ \left(\lambda_{\text{ventilation}} + k_{\text{filtration}} + k_{\text{deposition}}\right)\right] \quad (2)$$

where

V = indoor air volume (m^3)

$\lambda_{\text{ventilation}}$ = clean air ventilation rate (Q_{oa}/V, 1/hr)

$k_{\text{filtration}}$ = infectious particle removal rate due to filtration (1/hr)

$k_{\text{deposition}}$ = infectious particle deposition rate (1/hr)

Deposition removal rates ($k_{\text{deposition}}$) depend primarily on particle size, density, and room characteristics such as airspeeds and surface areas (Lai and Nazaroff, 2000; Nicas et al., 2005). Filtration removal rates ($k_{\text{filtration}}$) depend on the rate of airflow through the HVAC filter (Q_{filter}), the system operational time (f_{HVAC}), and the removal efficiency of the filter or air-cleaning device installed (η_{filter}), which also depends on particle size. These parameters are shown in Equation 3. Note that the airflow rate through the filter divided by the volume of the indoor air space served and multiplied by fractional operation time is also called the recirculation rate ($\lambda_{\text{recirculated}}$).

$$k_{\text{filtration}} = f_{\text{HVAC}} \frac{Q_{\text{filter}} \eta_{\text{filter}}}{V} = \lambda_{\text{recirculated}} \eta_{\text{filter}} \quad (3)$$

where

f_{HVAC} = fractional HVAC operation time (-)

Q_{filter} = airflow rate through filter (m^3/hr)

η_{filter} = particle removal efficiency of the filter (-)

$\lambda_{\text{recirculated}}$ = recirculation rate through the HVAC filter (1/hr)

Although Equations 2 and 3 are shown without explicitly considering particle size effects, both the filtration and deposition loss parameters ($k_{\text{deposition}}$ and $k_{\text{filtration}}$) are size-dependent. Therefore, size-resolved estimates of $k_{\text{deposition}}$ and $k_{\text{filtration}}$ should be utilized. We explore $k_{\text{filtration}}$ in more detail in the next section, but we rely on an approximate estimate of gravitational settling (i.e., $k_{\text{deposition}}$) from Nicas et al. (2005), as shown in Equation 4.

$$k_{\text{deposition}} = \frac{0.108 d_p^2 \left(1 + \frac{0.166}{d_p}\right)}{H} \quad (4)$$

where
d_p = aerodynamic diameter of particle (μm)
H = height of room (m)

We should also note that depending on the nature of the virus or bacteria inside of droplets or droplet nuclei, some rate of inactivation may also occur as aerosols are exposed to indoor air ($k_{inactivation}$); this rate has been explored for some viruses (Benbough, 1971; Yang and Marr, 2011) and is dependent in part in relative humidity. However, we exclude this loss rate in part because of a lack of existing data on size-resolved inactivation rates for multiple infectious aerosols of concern and in part because quanta generation rates, when back calculated using Equation 1, will inherently account for any inactivation that occurred during the case study period.

Finally, Gammaitoni and Nucci (1997) adapted the steady-state Wells-Riley risk model with additional control strategies (e.g., ventilation, HEPA filtration, and UVGI air cleaning) to also include time-varying (i.e., unsteady or dynamic) exposures. They evaluated tuberculosis infection control measures during a hypothetical infection outbreak in a single room of a hospital using their modified Wells-Riley model. Assuming that quanta generation rates were constant and that a certain number of infective persons (I) are present at an initial time t = 0, the probability of infection ($P_{infection}$) can be expressed as a function of time t, the quanta generation rate q, the volume V, and the number of "equivalent air changes" (i.e., the total loss/disinfection rate, C) as shown in Equation 5. Note that C is in the same form as the denominator terms in Equation 2.

$$P_{infection} = 1 - e^{-\frac{pIq}{V} \times \frac{Ct + e^{-Ct} - 1}{C^2}} \quad (5)$$

where
C = the total loss/disinfection rate (e.g., $\lambda_{ventilation} + k_{filtration} + k_{deposition +} k_{inactivation}$, 1/hr)

This method has recently been used to model the risk of airborne transmission of several diseases (e.g., influenza, TB, rhinovirus, and measles) in hospital rooms and patient waiting rooms under different outdoor air ventilation rates alone (Beggs et al., 2010; Knibbs et al., 2011). An advantage of the G-N model is that it allows for exploration of the importance of the amount of time of occupation by both infectious and susceptible individuals.

Given these advancements in incorporating other infectious aerosol loss mechanisms into traditional risk models, it is worth exploring what impacts traditional fibrous HVAC particle filtration may have on the spread of infectious airborne diseases. Exploring HVAC filtration is of interest in part because there may be ways to reduce the energy costs associated with outdoor air ventilation by recirculating more indoor air through higher efficiency HVAC filters. We will first consider incorporation of filtration into risk models and then will explore energy costs in a subsequent section of this report.

Linking MERV and risk of infectious disease transmission

As Equations 2-5 describe, the impact that recirculating HVAC filtration can have on the transmission of infectious airborne diseases is a function of (i) size-resolved particle removal efficiency of the HVAC filter in the size range of the infectious aerosols of interest and (ii) recirculation rates through the HVAC system (which are also a function of fractional operation time, HVAC airflow rate, and indoor air volume of the space served by the HVAC system).

Size-resolved filtration efficiency

Size-resolved particle removal efficiency for most filters is tested in laboratory settings according to ASHRAE Standard 52.2 (or its European equivalent EN 779). The standard involves measurements of upstream and downstream particle concentrations over a range of airflow rates and dust loading conditions to establish size-resolved particle removal efficiencies for 0.3 to 10 μm particles. Those values are then summarized into three distinct particle size bins: 0.3-1 μm, 1-3 μm, and 3-10 μm. Filters are assigned a Minimum Efficiency Reporting Value (MERV) according to their mean removal efficiencies in these bins. In general, the higher the MERV, the higher the minimum removal efficiency is for most particle sizes (although only the highest MERV filters are actually rated for the smallest particle size range). A summary of the MERV table from Standard 52.2 is shown in Table 3.

Table 3. Minimum Efficiency Reporting Values (MERV) and associated particle removal efficiencies (%)

	Composite particle removal efficiency (%)		
MERV	**0.3-1 μm**	**1-3 μm**	**3-10 μm**
1			<20
2			<20
3			<20
4			<20
5			20-35
6			35-50
7			50-70
8			70+
9		<50	85+
10		50-65	85+
11		65-80	85+
12		80+	90+
13	<75	90+	90+
14	75-85	90+	90+
15	85-95	90+	90+
16	95+	95+	95+

While Table 3 shows that HVAC filters are rated on their mean minimum removal efficiencies over just three size ranges, individual filter performance actually varies quite drastically even within each of the relatively narrow size bins. Additionally, some airborne particles of concern (i.e., those emitted via breathing, talking, coughing, or sneezing) may exist outside of the 0.3 to 10 μm size range, as described in a previous section. To illustrate the variations that occur across narrower ranges of particle sizes, Figure 5 shows polynomial curve fits that were made using actual measured data from a series of off-the-shelf filtration products in a laboratory setting

(Hecker and Hofacre, 2008). Note that the particle size range was extended by more sophisticated aerosol monitoring equipment, down to approximately 10 nm (0.01 µm).

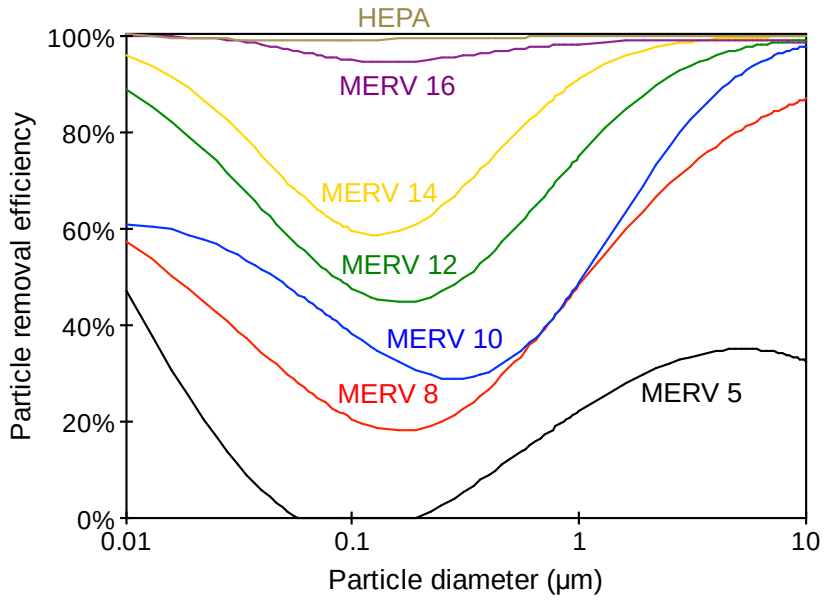

Figure 5. Fitted size-resolved particle removal efficiency curves for various actual MERV filtration products tested in a laboratory setting in Hecker and Hofacre (2008)

It should be noted that the removal efficiency values in Table 3 are entirely universal and generalizable; that is, a MERV 12 filter, for example, will always have 80% or higher removal efficiency for 1-3 µm particles and 90% or higher removal efficiency for 3-10 µm particles (when evaluated according to the test conditions). However, the removal efficiency values in Figure 5 are *not* generalizable, in that they each represent a particular filter available on the market (or in some cases, an average across multiple commercially available filters). Therefore, it is entirely appropriate that one MERV 12 filter (to use the same example from above) could have 10% removal efficiency for 0.3-1 µm particles while another could have 70% removal efficiency for the same size range (Hecker and Hofacre, 2008). The standard only dictates minimum mean efficiencies for particular size bins.

In using this information on test standards to link HVAC filtration ratings to infectious airborne disease risk, it is important to consider the advantages and disadvantages of using more detailed but specific data (i.e., Figure 5) versus using more generalizable but less specific data (i.e., Table 3). For the sake of making the connection between filtration and infectious disease risk as generalizable and universal as possible, this analysis will utilize only the minimum removal efficiencies for 0.3-10 µm particles as outlined by ASHRAE Standard 52.2 and shown in Table 3 unless otherwise noted.

Size-resolved quanta generation rates

In addition to selecting representative size-resolved HVAC filtration efficiencies, there are other challenges to linking HVAC filtration ratings to infectious airborne disease risk models. First, the quanta generation rate (q) that drives the risk calculations is not an entirely physical or intrinsic property. It is back calculated from epidemiological studies and is thus a function of the

assumptions for all of the other parameters utilized in its estimation from, for example, the Wells-Riley model in Equation 1 or the adjusted models in Equation 2 or Equation 5. Therefore, it is difficult to establish how *q* may actually vary by particle size. Some recent studies have explored size-resolved quanta generation rates by linking size-resolved particle measurements of actual viable bacteria to emission rates during human activity studies and assuming that, for example, 1 colony forming unit of bacteria (CFU) is equivalent to 1 "quanta" (Jones et al., 2009; Chen et al., 2011; Liao et al., 2013). Therefore, the quanta generation rate would simply be equal to the bacterial CFU emission rate. However, work in this area remains limited to date in the number of infectious diseases studied, particularly for viruses.

Another complicating issue is that because the control/loss mechanisms described in Equations 2-5 rely on accurate size-resolution of particles, it is difficult to establish exactly what is the particle size distribution of interest. The previous review on particle size distributions expelled during human activities (i.e., breathing, talking, coughing, and sneezing) revealed a lack of consensus and consistency in the particle sizes that are emitted. So not only is it difficult to map quanta generation rates to different particle sizes, it is difficult to know exactly what particle sizes are being expelled during a particular activity (not to mention the difficulty in knowing what particle sizes were originally emitted during the epidemiological studies from which values for *q* were derived).

It is reasonable to assume that the droplet nuclei particles (which contain other infectious particles) that are emitted from an individual during simple breathing will be smaller in size than if that same virus particles are emitted during coughing, as described in the previous review. In this case, aerosols with the same "infectivity" (according to bulk quanta generation rates) would actually have different removal mechanisms based on their different particle sizes. For example, a MERV 10 filter would be expected to capture ~50% of the larger droplet nuclei (in the 1-3 μm size range) but the same filter would have no expectations of removal of only slightly smaller 0.3-1 μm droplet nuclei particles. Therefore, coughed particles would likely be controlled with greater efficiency than breathed particles, all else being equal. Complicating this is the recent evidence (Blachere et al., 2009; Lindsley et al., 2010; Lindsley et al., 2010a; Yang et al., 2011) that infectious particles themselves (measured by more sophisticated DNA based techniques) may actually exist in more abundance in the slightly larger particle size range and may scale with particle surface area or volume more so than particle number. It may be reasonable to assume that the majority of the quanta generation may actually be associated with those larger particles, although smaller particles tend to exist in much greater abundance in terms of number concentration.

Given the large uncertainties and number of assumptions associated with each of these parameters, we will rely on some simplifying assumptions of our own in an attempt to link MERV to infectious disease risk models. Although there is considerable uncertainty both across the small number of previous studies and within individual studies for the existence of infectious particles in different size-fractions of aerosols, we will rely on an approximate average across those recent studies to determine best estimates for assumptions of the particle-size-resolution of *quanta*. These assumptions should be tested in the future as more data are revealed, but relying on these assumptions should be sufficient to make first-order approximations at this time.

Size-resolved aerosol emission from humans to use in risk models

Let us first assume that quanta generation rates are not evenly distributed across all particle sizes that are expelled during human activities but that they align more closely with the recent measurements of virus content in size-fractioned aerosols (Blachere et al., 2009; Lindsley et al., 2010; Lindsley et al., 2010a; Yang et al., 2011). We will also focus on measurements conducted in indoor environments rather than directly on exhaled breath of coughing subjects because we assume that those indoor measurements account for a combination of all human respiratory activities that occur indoors (i.e., breathing, coughing, and sneezing) in a manner that realistically describes timing patterns for these activities. Therefore, we will primarily utilize the size distributions containing influenza virus RNA reported in Lindsley et al. (2010) and shown in Table 2. In that investigation, personal and stationary measurements varied slightly in particle size cut-off ranges and neither sampling device aligned exactly with the ASHRAE 52.2 filtration standard classification system, so we have adjusted their measured values using engineering judgment to a context more appropriate for use with MERV.

Therefore, we assume the following particle size distribution of infectious aerosols and quanta generation rates using data from Table 2:

- 15% of infectious droplet nuclei are in the 0.3-1 μm size range;
- 25% exist in the 1-3 μm size range; and
- 60% exist in the 3-10 μm size range.

Linking to MERV

At this point, we understand most of the parameters that are required for the previously described risk models. Let us first consider filtration efficiency (η_{filter}) for expelled droplet nuclei from the assumed combination of human activities in an indoor environment. We will estimate a particle-size-weighted average filtration efficiency according to our assumptions of 15% 0.3-1 μm, 25% 1-3 μm, and 60% 3-10 μm particles for each level of MERV. Table 4 shows removal efficiencies for droplet nuclei to be utilized in this work for infectious disease transmission modeling.

Table 4. Assumed filter removal efficiency for 0.3-1 μm, 1-3 μm, 3-10 μm and total weighted-average droplet nuclei particles

MERV	0.3-1 μm	1-3 μm	3-10 μm	Droplet nuclei-weighted[b] η_{filter}
4[a]	1%	9%	15%	11%
7[a]	17%	46%	50%	44%
11[a]	30%	65%	85%	72%
13	70%	90%	90%	87%
14	80%	90%	90%	89%
15	90%	90%	90%	90%
16	95%	95%	95%	95%
HEPA	99.9%	99.9%	99.9%	99.9%

[a] Values for 0.3-1 and 1-3 μm are taken from Stephens and Siegel (2012)
[b] Assuming 15% are 0.3-1 μm, 25% are 1-3 μm, and 60% are 3-10 μm

The droplet nuclei weighted average values for minimum removal efficiency are estimated using the aforementioned weighted-average. Unfortunately, only MERV 13 filters and above are actually characterized for removal efficiency of 0.3-1 μm particles according to ASHRAE

Standard 52.2 (Table 3). For MERV < 13, removal efficiency values were taken from a previous experimental case study using off-the-shelf filtration products in residences (Stephens and Siegel, 2012), which although limited to the particular filtration products used within, can still provide some cursory information about lower MERV filters. As one can see, a range of MERV filters have been chosen to represent filtration efficiencies for droplet nuclei aerosols from as low as ~11% (MERV 4) to as high as ~99.9% (HEPA).

While Table 4 provides estimates for mean removal efficiencies for various MERV filters across the size ranges of concern for infectious droplet nuclei transmission, the other important parameter in determining the impact of filtration on infectious disease risk is the recirculation rate, or the rate of airflow through the filter divided by the space volume and multiplied by the fraction of time the system operates.

The recirculation rate is a function of building properties that exist outside of the type of filtration utilized. That is, a building's HVAC system airflow rate, runtime, and indoor air volume served are all unique and defined regardless of whether a MERV 4 or MERV 16 filter is installed. This limits us to considering only case studies of particular buildings instead of developing a wholly generalizable link between MERV and infectious disease risk. However, the following section relies on case studies of three particular indoor environments (an office, an elementary school classroom environment, and a hospital waiting room) to demonstrate the likely impacts that HVAC filtration can have on the risk of spreading three particularly airborne infectious diseases (influenza, rhinovirus, and tuberculosis). The *methods* used herein are generalizable and repeatable for other environments, and the 9 individual case studies (three diseases in three environments) are later summarized to make the *results* more generalizable.

Case studies: office, school, and hospital environments

Assuming the aforementioned droplet nuclei particle size distributions emitted during human activities containing infectious particles and the associated filtration removal efficiencies that we have assumed, let us consider a case study in each of three hypothetical environments to demonstrate the potential impacts of HVAC particle filtration on transmission of airborne infectious diseases. We will utilize the modified steady-state Wells-Riley risk model, as described in Equation 2. We will consider the influenza A virus (the common flu), rhinovirus (the common cold), and tuberculosis (TB, a bacterial infection). These have been chosen for their combination of data availability, evidence of aerosol transmission routes, and implications for impacts across a large portion of society.

In each environment, we will make assumptions about the number of susceptible occupants and their pulmonary ventilation rates. The number of infector individuals (I) will be 1 in all cases. Reasonable occupant densities, outdoor airflow rates, recirculation rates, and space volumes will be used assuming that ASHRAE Standard 62.1 or other relevant ventilation standard is adequately met. Note that particle deposition rates ($k_{\text{deposition}}$) are important for removal but are highly variable depending on the nature of each environment. For simplicity, we assume constant values of $k_{\text{deposition}}$ in each environment and estimate them using a procedure described in Equation 4 (and in Nicas et al. 2005). Using a geometric mean diameter between 0.3 and 1 μm (0.55 μm), between 1 and 3 μm (1.7 μm), and between 3 and 10 μm (5.5 μm) in conjunction

with an assumed infectious particle size distribution of 15% for 0.3-1 μm, 25% for 1-3 μm, and 60% for 3-10 μm, deposition rates in a 3 m height room are estimated as ~0.01 per hour, ~0.12 per hour, and ~1.1 per hour, respectively. These lead to a size-weighted-average estimate of ~0.7 per hour for $k_{deposition}$, for infectious droplet nuclei. For the three infectious diseases considered, we use central estimates of previously published values of quanta generation rates (q) as follows:

- Rhinovirus: q = 5 per hour
- Tuberculosis: q = 13 per hour
- Influenza: q = 100 per hour

Office Environment

Let us first consider a case study on a hypothetical 500 m^2 office building with 3 m ceilings (V = 1500 m^3). The office space has 25 occupants, one of which is infected with the influenza virus, rhinovirus, or TB (I = 1; *susceptibles* = 24).

Per ASHRAE Standard 62.1, the minimum outdoor air ventilation rate should be 5 cfm per person + 0.06 cfm/ft^2, which is equivalent to ~450 cfm (~760 m^3/hr) in the assumed space. Assuming the outdoor air supply fraction of total airflow is approximately 25% (Persily and Gorfain, 2004), the total supply airflow rate would be ~1800 cfm (~3000 m^3/hr), with ~1350 cfm (~2300 m^3/hr) provided as recirculated air. Therefore, the removal rate due to outdoor air supply can be estimated as ~0.51 per hour (450 cfm or ~760 m^3/hr divided by 1500 m^3 = 0.51 per hour). The removal rate due to recirculated air filtration is thus ~1.52×η_{filter} per hour (where 1350 cfm, or ~2300 m^3/hr, divided by 1500 m^3 = 1.52 per hour; multiply by particle removal efficiency to get $k_{filtration}$). The estimated removal rates of droplet nuclei particles due to recirculated air filtration through various MERV filters are thus shown in Table 5.

Table 5. Droplet nuclei particle removal rates ($k_{filtration}$) for a range of MERV filters for use in modeling risk inside the office building with a recirculation rate of 1.52 per hour

MERV	Droplet nuclei removal efficiency (η_{filter})	Assumed filter removal rate ($\lambda_{recirculated}\times\eta_{filter}$, 1/hr)
4	11%	0.17
7	44%	0.67
11	72%	1.09
13	87%	1.32
14	89%	1.35
15	90%	1.37
16	95%	1.45
HEPA	99.9%	1.52

If we assume that adult occupants work 8-hour days and that all occupants have an average breathing rate while working of 0.67 m^3/hr (U.S. EPA, 2011), we can estimate the probability of infection of each individual being present for an entire day with each filtration case as shown in Figure 6.

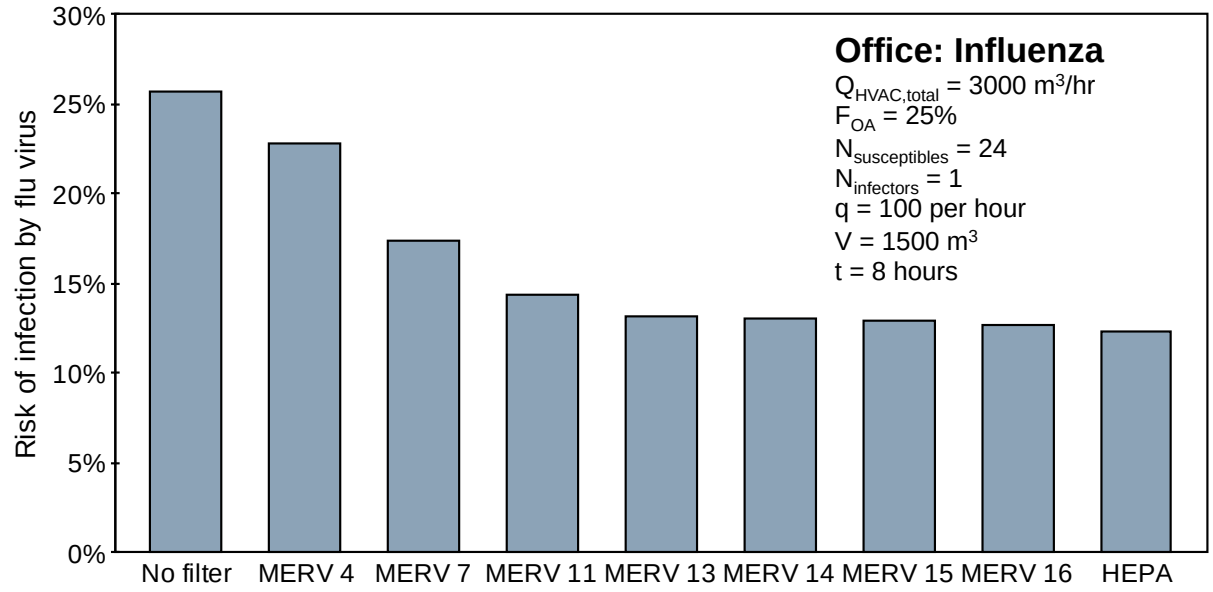

Figure 6. Projected risk of infection by influenza virus during an 8-hour workday in a hypothetical office building with 25 occupants and 25% outdoor air supply using a range of HVAC filters installed in a system with a recirculation rate of 1.5 per hour

The individual risks can also be multiplied by the number of susceptible occupants (24) to estimate the likely number of infections attributable to the one infectious individual under each filtration scenario. For example, in the no filter case, 6 of the 24 susceptible occupants (25%) are estimated to get the flu from this one infectious individual. Even a relatively low efficiency filter (MERV 7) is predicted to reduce the number of infected individuals to 4 (to 17% risk). Increasing to MERV 11 likely prevents another individual from airborne influenza infection. Finally, increasing to MERV 14-16 or even HEPA filtration all have the same effect of lowering the likely number of infected individuals to 3 (a ~50% reduction relative to no or low filtration efficiency).

Individual risks for tuberculosis (q = 13 per hour) and rhinovirus (q = 5 per hour) are estimated in a similar fashion for the hypothetical office environment and shown in Figure 7 and Figure 8, respectively.

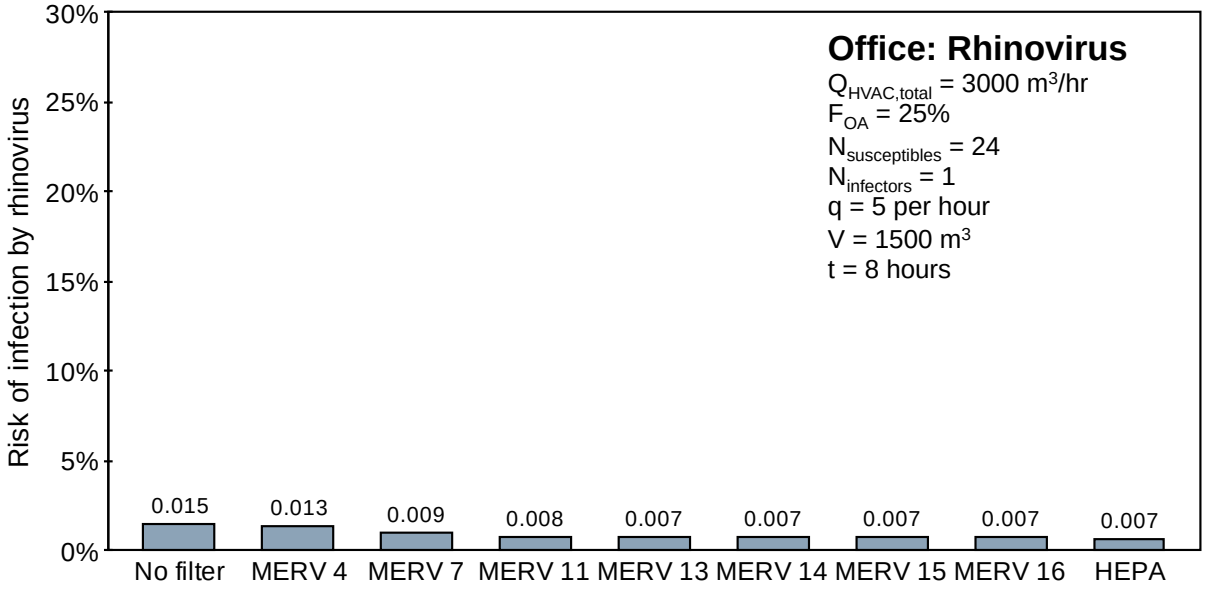

Figure 7. Predicted risk of infection by rhinovirus (common cold) in the hypothetical office building. Note that the y-axis values are in percentages for this and subsequent figures; the values noted above the bars are fractional (i.e., 0.015 = 1.5%).

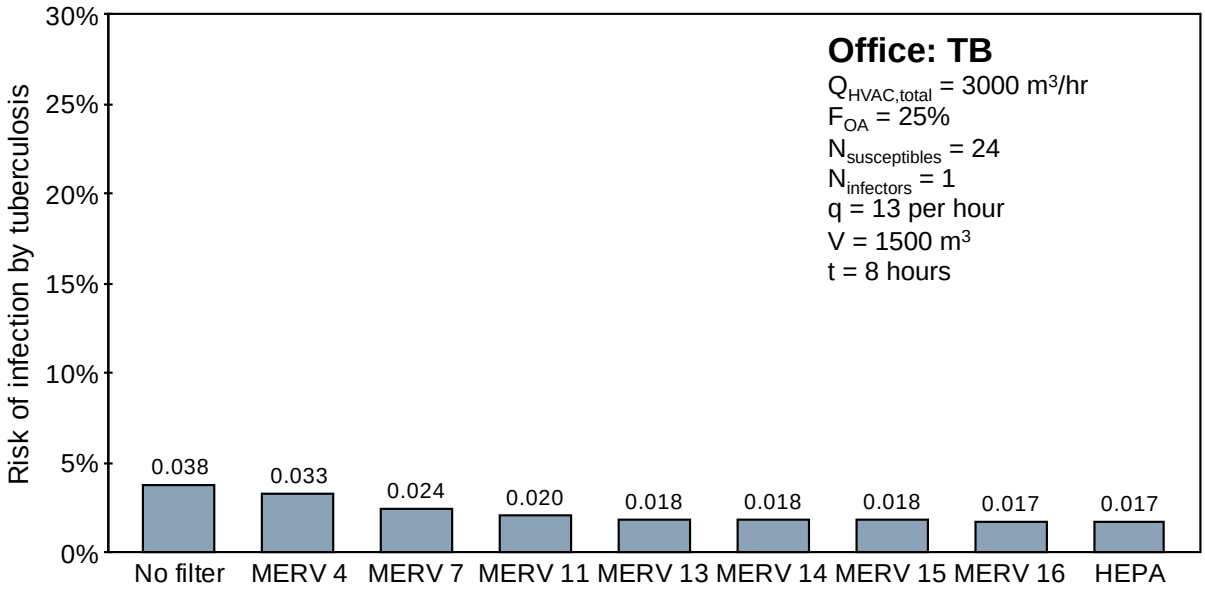

Figure 8. Predicted risk of infection by tuberculosis (TB) in the hypothetical office building

Note that the relative risk reductions for both rhinovirus and TB are the same as influenza for each filter. However, because the quanta generation rates are much lower for rhinovirus and TB, the absolute impact is lower. For example, zero infectious are predicted for rhinovirus even without a filter installed in this office environment. For TB, even a modest amount of filtration (e.g., MERV 11) would likely reduce the number of TB-infected individuals to zero in this hypothetical office building (a decrease from only 1 without a filter or with a lower efficiency

filter). These results suggest that (i) filtration is more important for more highly infectious diseases, and (ii) even HEPA filtration cannot reduce risks to zero because of limitations in recirculation rates (or the absolute amount of air that comes in contact with filtration).

School Environment

Let us now consider a case study on a hypothetical 100 m^2 classroom environment with 3 m ceilings (V = 300 m^3). The classroom space has 35 children aged 10-11 years old (this is the default occupancy value per ASHRAE Standard 62.1). One student is infected with the influenza virus (I = 1; *susceptibles* = 34). All occupants have lower pulmonary ventilation rates than adults: p = 0.5 m^3/hr (U.S. EPA, 2011).

Per ASHRAE Standard 62.1, the minimum outdoor air ventilation rate should be 10 cfm per person + 0.12 cfm per ft^2 of foor area. In this room, the total outdoor air supply would be ~480 cfm (~815 m^3/hr). Assuming the outdoor air supply fraction of total airflow is approximately 25% (Persily and Gorfain, 2004), the total supply airflow rate would be ~1880 cfm (~3200 m^3/hr), with ~1400 cfm (~2380 m^3/hr) provided as recirculated air. Therefore, the removal rate due to outdoor air supply can be estimated as ~2.7 per hour (480 cfm or ~815 m^3/hr divided by 300 m^3 = 2.71 per hour). The removal rate due to recirculated air filtration is thus 8.14$\times\eta_{filter}$ per hour (where 1400 cfm, or ~2380 m^3/hr, divided by 300 m^3 = 8.14 per hour; multiply by particle removal efficiency to get $k_{filtration}$). The estimated removal rates of droplet nuclei particles due to recirculated air filtration through various MERV filters in this hypothetical school environment are thus shown in Table 6.

Table 6. Droplet nuclei particle removal rates ($k_{filtration}$) for a range of MERV filters for use in modeling risk inside the school classroom with a recirculation rate of 8.14 per hour

MERV	Droplet nuclei removal efficiency (η_{filter})	Assumed filter removal rate ($\lambda_{recirculated}\times\eta_{filter}$, 1/hr)
4	11%	0.93
7	44%	3.59
11	72%	5.84
13	87%	7.08
14	89%	7.20
15	90%	7.33
16	95%	7.73
HEPA	99.9%	8.13

If we assume that children are present 8 hours per day, we can estimate the probability of infection of each individual student with each filtration case as shown in Figure 9.

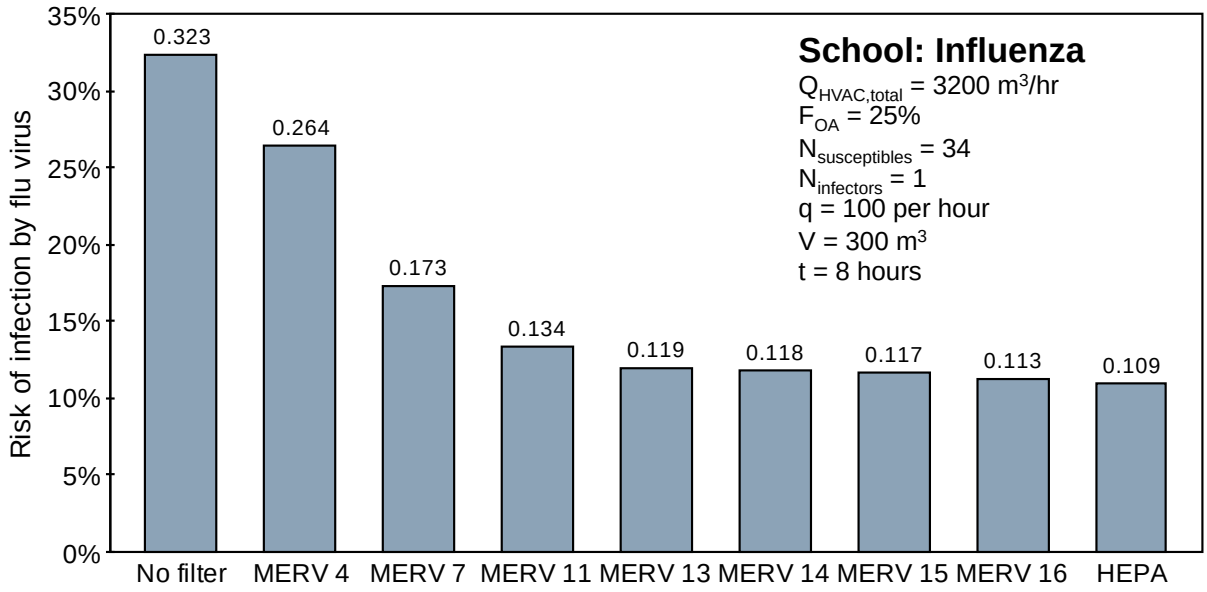

Figure 9. Projected risk of infection by influenza virus during an 8-hour day in a hypothetical classroom environment with 35 child occupants and 25% outdoor air supply using a range of HVAC filters installed in a system with a recirculation rate of 8.14 per hour

Without any filtration, 11 children are expected to be infected with the influenza virus after spending the entire school day in the presence of one infector student. MERV 7 filtration is expected to reduce that to 6 children; MERV 11 to 7 children; MERV 13 to 5 children; and MERV 14 through HEPA to 4 children. The probability of infection is thus reduced by approximately 60-70% for filtration levels of MERV 13 or higher.

Similarly, the risks of infection from rhinovirus and TB in the classroom environment are shown in Figure 10 and Figure 11.

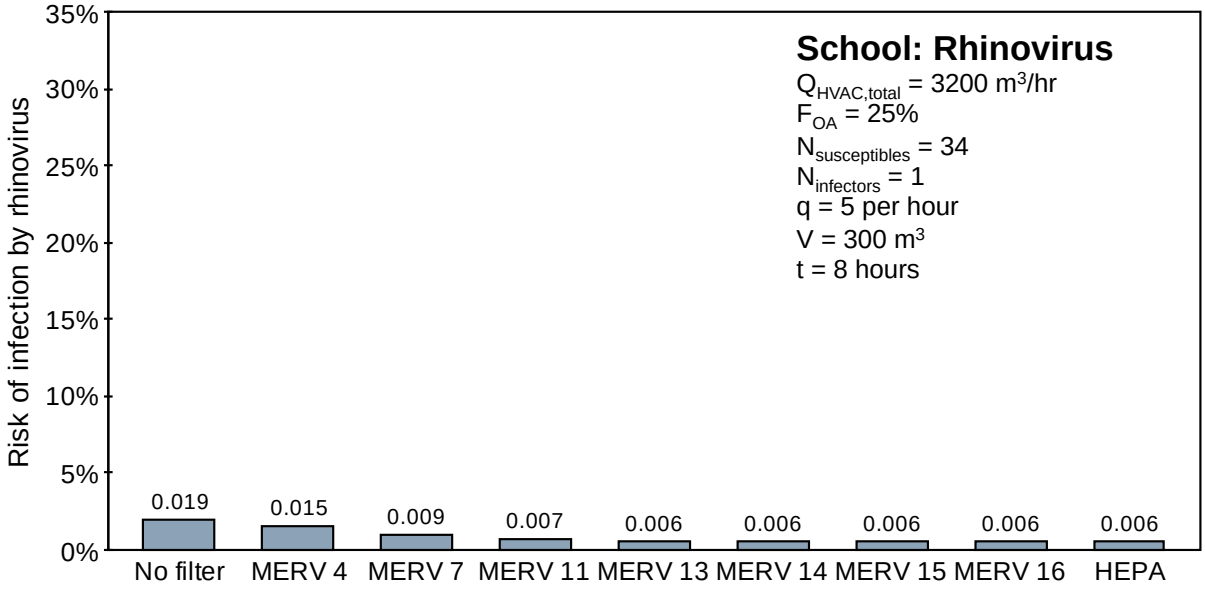

Figure 10. Predicted risk of infection by rhinovirus (common cold) in the hypothetical classroom environment

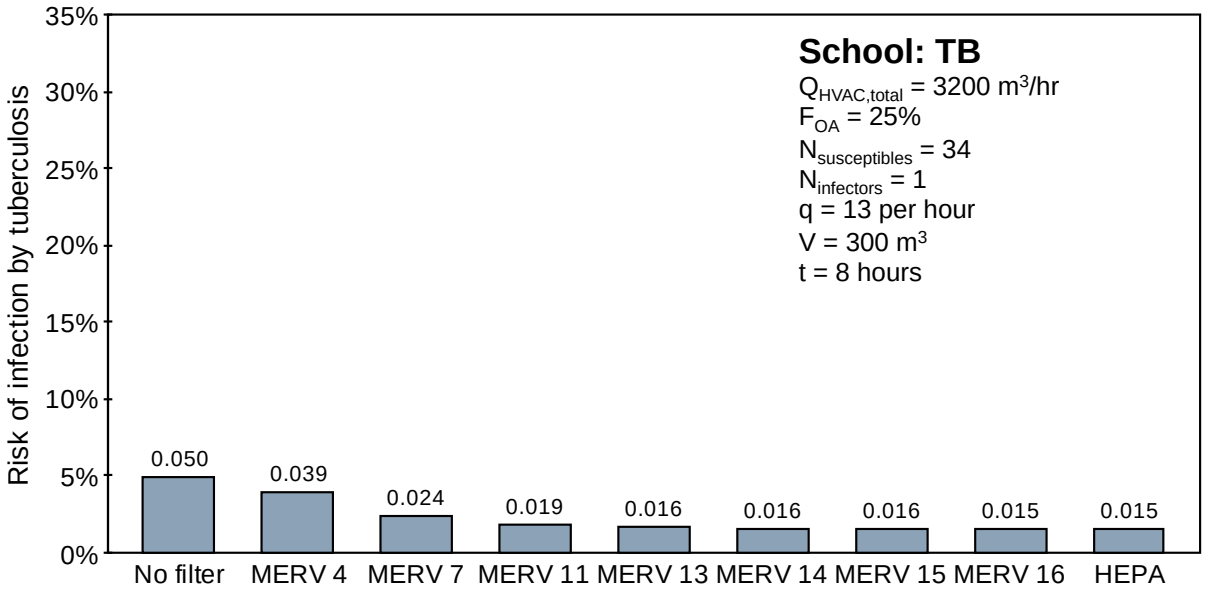

Figure 11. Predicted risk of infection by tuberculosis (TB) in the hypothetical classroom environment

Similar relative reductions in risk are predicted to occur, but again because quanta generation rates are lower for rhinovirus and TB, the absolute number of infected students is much lower than for influenza. MERV 7 filtration or higher is expected to reduce the number of students infected with rhinovirus from 1 to 0; similarly, MERV 4 through HEPA would all have about the same impact of a reduction from 2 TB-infected students to 1.

Hospital Environment

Let us now consider a case study on a hypothetical 200 m^2 hospital waiting room environment with 3 m ceilings (V = 600 m^3). The waiting room has 50 adults present; one person is infected (I = 1; *susceptibles* = 49). All occupants have a pulmonary ventilation rate p = 0.67 m^3/hr (U.S. EPA, 2011). Occupants are assumed to spend a much shorter amount of time in the waiting room: only 2 hours.

Minimum ventilation requirements in hospital environments are not provided in ASHRAE Standard 62.1 but in ASHRAE Standard 170, which states that the minimum outdoor air ventilation rate in ER waiting rooms should be 2 air changes per hour. The minimum recirculation rate through an HVAC system should be 10 per hour (for a total of 12 total air changes per hour, ventilated + recirculated). Therefore, the removal rate due to outdoor air supply can be estimated as 2 per hour and the removal rate due to recirculated air filtration is $10 \times \eta_{\text{filter}}$ per hour. The estimated removal rates of droplet nuclei particles due to recirculated air filtration through various MERV filters in this hypothetical hospital waiting room environment are thus shown in Table 7. Note that a recirculation rate of 10 per hour in this environment with V = 600 m^3 leads to a recirculated airflow rate of 6000 m^3/hr and a total airflow rate of 7200 m^3/hr.

Table 7. Droplet nuclei particle removal rates ($k_{\text{filtration}}$) for a range of MERV filters for use in modeling risk inside the hospital waiting room environment

MERV	Droplet nuclei removal efficiency (η_{filter})	Assumed filter removal rate ($\lambda_{\text{recirculated}} \times \eta_{\text{filter}}$, 1/hr)
4	11%	1.14
7	44%	4.41
11	72%	7.18
13	87%	8.70
14	89%	8.85
15	90%	9.00
16	95%	9.50
HEPA	99.9%	9.99

The probability of influenza infection of each individual with each filtration case is shown in Figure 12.

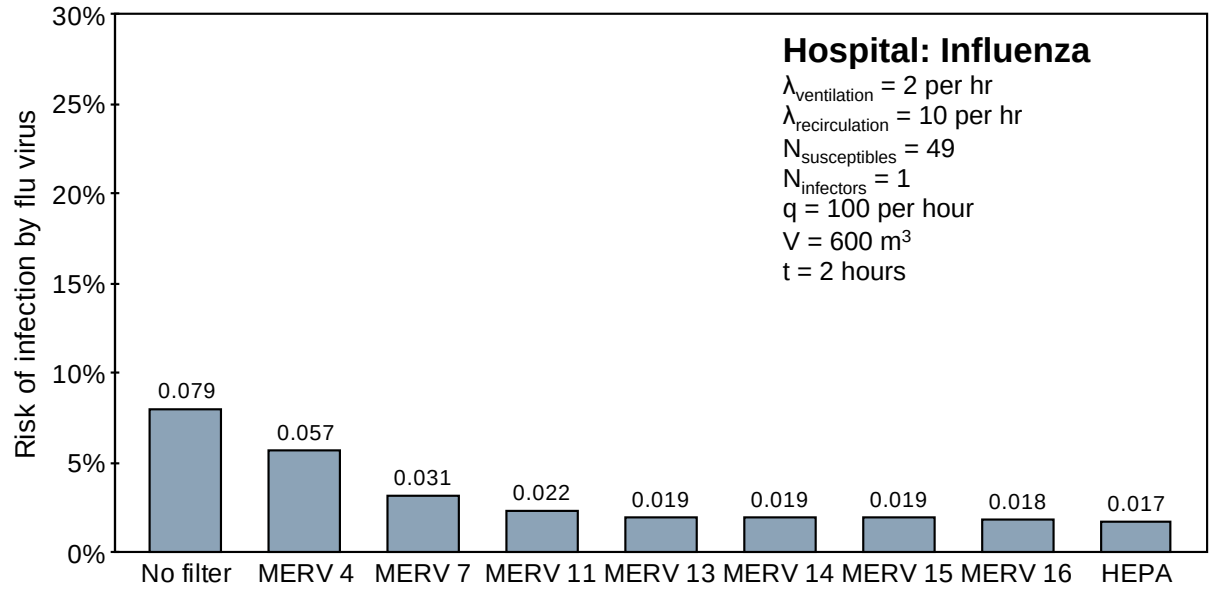

Figure 12. Projected risk of infection by influenza virus during a 2-hour stay in a hypothetical hospital waiting room environment with 50 adult occupants and ASHRAE 170 minimum ventilation rates

Because of the initially relatively large outdoor air exchange rate of 2 per hour combined with a shorter time of occupancy (2 hours), initial risks in this unfiltered hospital waiting room environment are expected to be lower than the previous two environments. At baseline without a filter, 4 out of 49 people are predicted to be infected with the influenza virus after 2 hours (~8%). Increasing to MERV 4 could likely reduce this to 3 people; MERV 7 to 2 people; and MERV 11 and above to 1 person. Again, even HEPA filtration cannot reduce this value to zero because of limitations associated with even this high recirculation rate of 10 per hour.

Rhinovirus and TB infection risks are similarly shown in Figure 13 and Figure 14.

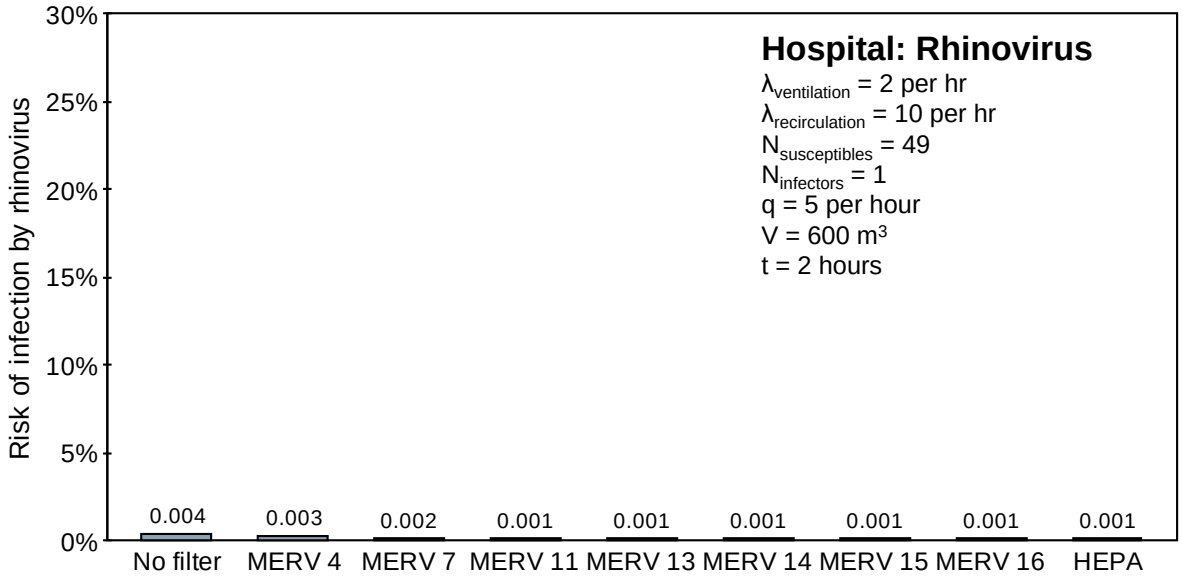

Figure 13. Predicted risk of infection by rhinovirus (common cold) in the hypothetical hospital environment

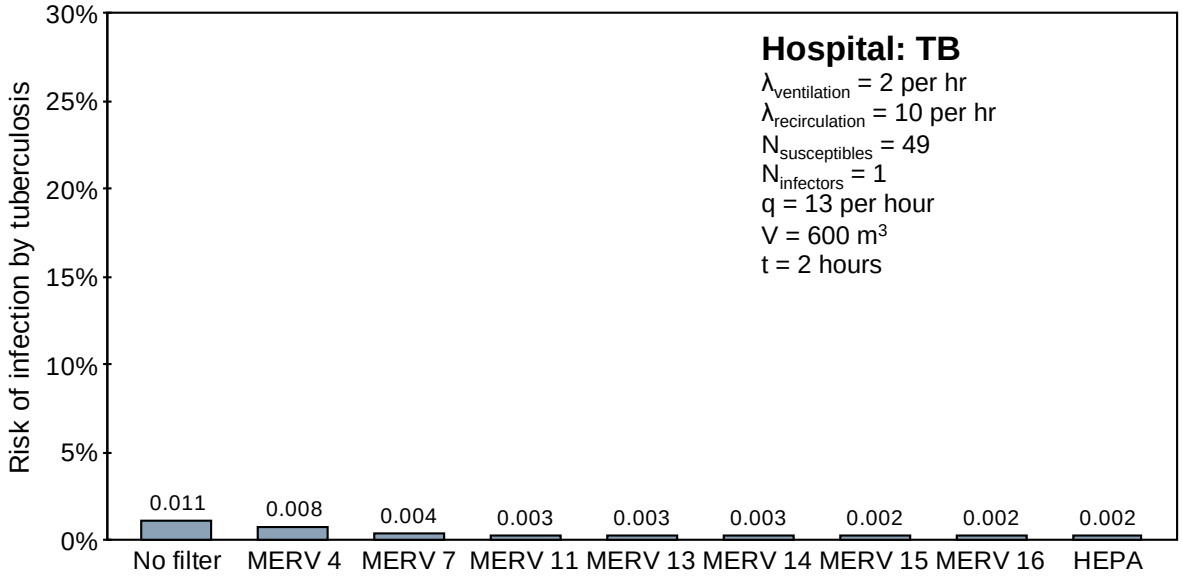

Figure 14. Predicted risk of infection by tuberculosis (TB) in the hypothetical hospital environment

Again, relative risk reductions follow a similar pattern, but absolute numbers of infected people are lower. Rhinovirus is not expected to spread via infectious aerosol inhalation in this waiting room environment under the assumptions of only 2 hours of exposure time. The risk of infection by TB is expected to be reduced from 1 person to zero with MERV 4 filters or greater.

These individual case studies highlight some of the potential impacts of, and limitations to, control of infectious aerosols by common HVAC filtration products. While not an exhaustive demonstration of a large number of indoor environments, we can see that the trends in risk

reductions associated with increasing HVAC filtration efficiency are generally similar in relative terms. The next section will summarize these 9 case studies in an attempt to make results herein more generalizable.

Average risk reductions across the case studies

While the infection risk reductions shown in the previous section are unique to each building environment and their own set of assumptions, we can use the 9 case studies to explore some preliminary trends in risk reductions. Figure 15 shows the predicted relative risk (RR) of each HVAC filtration selection for each of the 9 case studies. RR values use the "no filter" as a baseline; thus, each RR is calculated as the probability of infection with a filter divided by the probability of infection without a filter. Individual cases are shown in gray and the average across the 9 cases is shown in red. Values on the x-axis should be interpreted as categorical MERV values, not discrete numeric values, although they appear numeric. These same values are shown numerically in Table 8.

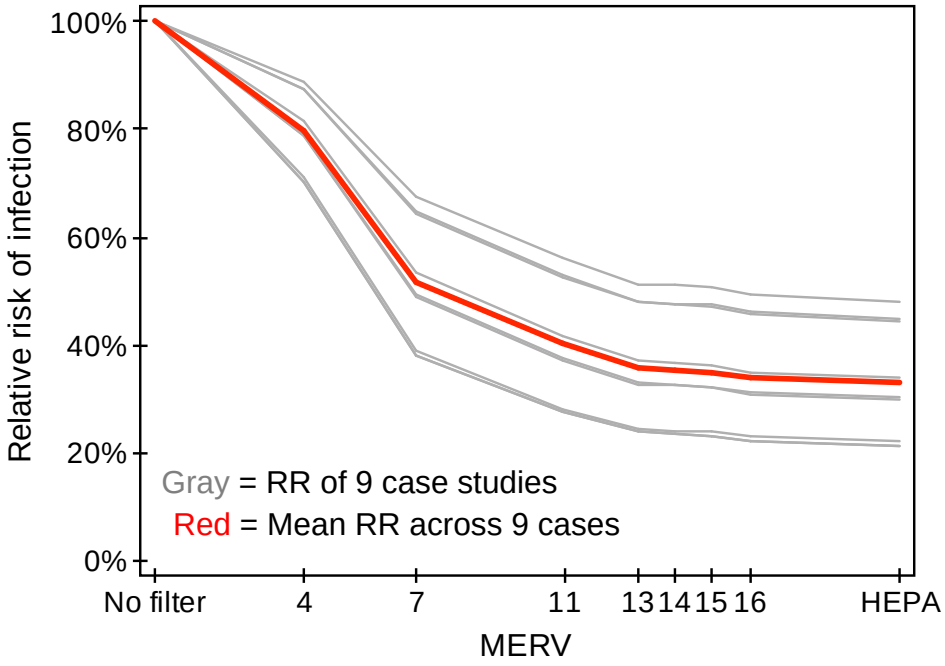

Figure 15. Relative risk of infection across the 9 case studies and 9 levels of HVAC filtration

Table 8. Mean relative risk (RR) of infection across the 9 case studies in this report

Filter condition	Mean RR across 9 case studies
No filter	1.00
MERV 4	0.80
MERV 7	0.53
MERV 11	0.40
MERV 13	0.36
MERV 14	0.35
MERV 15	0.35
MERV 16	0.34
HEPA	0.33

On average across the 9 case studies herein, MERV 4 filters are expected to provide a ~20% reduction in risk of infection (RR of ~0.8). MERV 7 would provide a reduction of ~50% (down to RR of 0.5). MERV 11 would provide a reduction of ~60% (down to RR of 0.4). Finally, MERV 13-16 and even HEPA filters are all predicted to provide ~64-67% reductions in RR (down to 0.33-0.36). Thus, it appears that enhanced HVAC filtration may have diminishing returns as even a 100% efficient filter is limited by the recirculation rate (i.e., the amount of recirculating airflow through the HVAC filter divided by the space volume).

Given the long list of assumptions provided to model these infectious disease risks, further research should be performed to quantify the sensitivity of the models herein to some of the basic assumptions about infectious particle sizes, quanta generation rates, occupancy, and exposure times. However, these results do suggest that recirculating HVAC filtration can likely play an important role in reducing the risk of transmitting airborne infectious diseases in indoor environments. In order to explore the initial hypothesis that HVAC filtration may achieve risk reductions at lower costs than outdoor air ventilation rates, the next section attempts to estimate the costs of controlling infectious diseases first by outdoor air ventilation and then by HVAC filtration.

Costs of controlling infectious disease: outdoor air vs. HVAC filtration

Although outdoor air ventilation rates have been shown to decrease the risk of spreading some infectious airborne diseases, introducing more ventilation air also comes with an energy penalty. The amount of energy required to condition excess ventilation air varies according to the magnitude of outdoor airflow rates, climate conditions, and system equipment and efficiency. However, approximate estimates of the amount of energy required to condition the sensible load from outdoor air ventilation can be made using metrics of heating-degree-days (HDD) and cooling-degree-days (CDD) and by making assumptions about equipment efficiency and system operational times.

Cost of outdoor air delivery

An approximation of the amount of energy required for heating on an annual basis can be made using Equation 6. Note that this equation estimates HDDs for each hour of assumed occupied

times based on a 65°F (18.3°C) basis. Assumed operational schedules are described in a subsequent section for each environment. Also, this equation assumes that outdoor air ventilation rates do not vary during operational times.

$$E_{\text{heating}} = \lambda_{\text{ventilation}} V \rho_{\text{air}} C_{\text{p,air}} HDD \frac{1}{\eta_{\text{heating}}} \alpha \quad (6)$$

where
E_{heating} = energy required for heating (MJ, assume fuel is natural gas)
ρ_{air} = density of air (1.2 kg/m^3)
$C_{\text{p,air}}$ = specific heat capacity of air (1000 J/(kg-K))
HDD = heating degree days during times of building operation (K-days)
η_{heating} = conversion efficiency of heating equipment (-)
α = units conversion factor (24 hours/day × 10^{-6} MJ/J)

Similarly, the amount of energy required for cooling on an annual basis can be approximated using Equation 7 (and utilizing the same assumptions as Equation 6):

$$E_{\text{cooling}} = \lambda_{\text{ventilation}} V \rho_{\text{air}} C_{\text{p,air}} CDD \frac{1}{\eta_{\text{cooling}}} \beta \quad (7)$$

where
E_{cooling} = electricity required for cooling (kWh)
η_{cooling} = electric efficiency of cooling equipment (-)
CDD = cooling degree days during times of building operation (K-days)
β = units conversion factor (24 hours/day × 0.277 kWh/MJ × 10^{-6} MJ/J)

Finally, annual energy costs can be estimated by multiplying average utility rates (assuming these are constant for simplicity) by the amount of delivered energy required (electric + gas).

Cost of outdoor air delivery in the three case study environments

Here we estimate the cost of outdoor air delivery in the three case study environments, assuming four different locations across the United States: Chicago, IL, Charlotte, NC, Houston, TX, and Phoenix, AZ. Let us assume that a 90% efficient natural gas boiler or furnace is used for heating and the air-conditioning equipment has a constant electric cooling efficiency of 300% (COP = 3.0). Let us assume the office building and classroom does not operate continuously year-round, but that it is occupied with workers/students ~31% of the time (i.e., 11 hours per day from 7 am to 6 pm, excluding holidays and weekends). HDDs and CDDs were estimated for each hour of the assumed schedule over the course of a year using TMY3 weather data. Let us assume the hospital waiting room does have to operate 100% of the time (i.e., 8760 hours per year).

Let us assume electricity rates are constant at $0.12/kWh and that natural gas rates are $4/MMBTU (1 MMBTU = 1055 MJ). These are generally in line with natural average rates (and we do not explore geographic variations, for simplicity). Climate data (HDD and CDD) utilized in the models are described in Table 9.

Table 9. Climate conditions (HDD and CDD) used in modeling the energy costs of outdoor air ventilation for (i) the office and classroom environments and (ii) the hospital waiting room under assumed operational schedules

	Chicago	Charlotte	Houston	Phoenix
	Office and Classroom			
HDD (°F-day)	1607	831	367	286
HDD (K-day)	893	461	204	159
CDD (°F-day)	540	747	1284	1820
CDD (K-day)	300	415	713	1011
	Hospital Waiting Room			
HDD (°F-day)	5810	3295	1678	1246
HDD (K-day)	3228	1831	932	692
CDD (°F-day)	1267	1775	3031	4862
CDD (K-day)	704	986	1684	2701

Given all of these assumptions, approximate energy costs will increase linearly with the level of outdoor air ventilation, as shown in Figure 16 for the three building environments. These costs should be considered approximations, as several simplifying assumptions have been made for clarity.

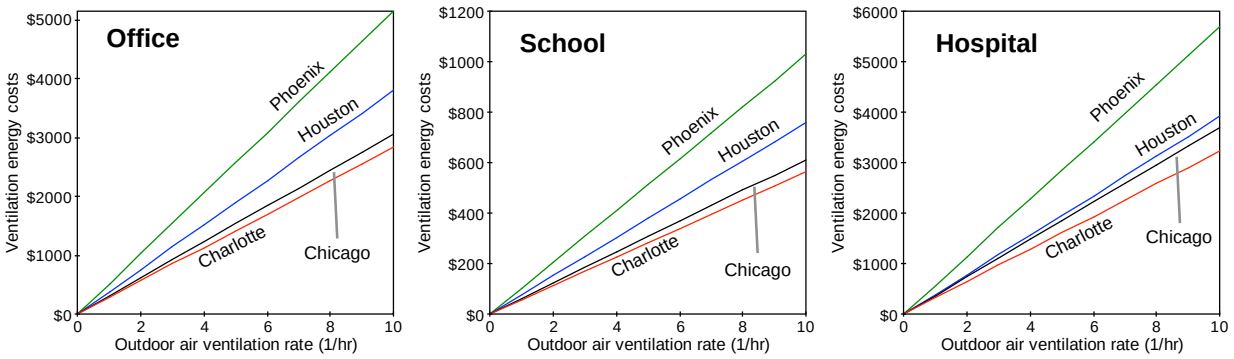

Figure 16. Approximate energy costs for a range of outdoor air ventilation rates in the three types of case study environments. Note the differences in scales on the y-axes. The energy required is specific to the assumptions of indoor air volumes for each space (not entire buildings), and no additional fan energy is assumed

Because natural gas costs are currently low and because of the greater requirements for electricity for cooling in the warmer climates of Phoenix and Houston, Chicago and Charlotte are expected to deliver the same outdoor air ventilation rates at somewhat lower costs. Phoenix is the most expensive because of the large number of cooling degree days required. These values provide an approximate baseline for us to evaluate HVAC filtration costs against in each environment in each geographic location. The slope between energy costs and outdoor ventilation rate is utilized in a later section to determine equivalent filtration costs of outdoor air.

Cost of HVAC filtration

In commercial environments with variable speed HVAC systems, any additional cost of HVAC filtration can be expressed first in terms of additional fan power required to overcome the additional pressure drop associated with a higher efficiency (typically higher pressure drop)

filter. This is not necessarily the case in many smaller commercial environments (Stephens et al., 2010), but is a reasonable assumption for this analysis. Additional costs for filter replacement and disposal must also be considered (Bekö et al., 2008).

First, the additional fan power required to deliver a particular airflow rate through an HVAC system can be estimated in Equation 8.

$$W_{\text{filtration}} = \frac{Q_{\text{recirculated}} \Delta P_{\text{avg}}}{\eta_{\text{fan}} \eta_{\text{motor}}} \tag{8}$$

where
$W_{\text{filtration}}$ = instantaneous power draw required for filtration (W)
Q_{HVAC} = airflow rate through the HVAC filter (m^3/s)
ΔP_{avg} = average pressure drop across filter (Pa)
η_{fan} = fan efficiency (assume 70% constant as in Bekö et al. 2008)
η_{motor} = motor efficiency (assume 65% constant as in Bekö et al. 2008)

The total fan energy cost can be estimated using Equation 9.

$$C_{\text{filtration}} = W_{\text{filtration}} t_{\text{operating}} P_{\text{electric}} \tag{9}$$

where
$C_{\text{filtration}}$ = cost required to overcome filter pressure drop ($)
$t_{\text{operating}}$ = absolute amount of time that the building is occupied (hours)
P_{electric} = electric price ($/Wh)

To estimate the total cost of filtration, one must also consider the initial cost of the filtration product itself, the number of filters required for each installation, the typical lifespan of filtration products, and the labor for filter installation and subsequent disposal at the end of its life span. These labor costs were estimated directly from Bekö et al. (2008) as $12 per filter installation and $5 per filter disposal. Assuming each filter is 24” × 24” (0.6 × 0.6 m), the number of filters required for each environment was estimated by (i) dividing the airflow rate through the HVAC filter by an assumed constant 2 m/s face velocity, which yields the approximate area of filtration required, and (ii) determining the number of 24” × 24” (0.6 × 0.6 m) filters to achieve that approximate area, rounding up to the nearest whole number. This yielded one filter required in the office and school environments and two filters in the hospital waiting room environment.

The initial costs of filtration products and the typical life spans were taken from an anonymous contact in the commercial filtration industry. These cost estimates are provided in Table 10. The office and school environments were assumed to operate 2717 hours per year while the hospital environment operated 100% of the time (8760 hours per year).

Table 10. Assumptions used in estimating the cost of HVAC filtration

Filter	Size	Purchase cost	Initial pressure drop (Pa)	Final pressure drop (pa)	Average pressure drop (Pa)	Expected filter life
MERV 4	24x24x2	$2	22	125	73	3 months
MERV 7	24x24x2	$4	72	149	111	3 months
MERV 11	24x24x2	$7	95	187	141	4 months
MERV 13	24x24x2	$11	102	187	144	4 months
MERV 14	24x24x12	$50	127	249	188	12 months
MERV 15	24x24x12	$90	70	249	159	12 months
MERV 16	24x24x12	$125	65	249	157	12 months
HEPA	24x24x12	$150	249	498	374	12 months

Using these assumed values and the equations outlined above, estimates of the annual costs of filtration in each of the three environments are shown in Figures 17, 18, and 19. Costs are divided into fan energy costs, filter purchase costs, labor costs, and disposal costs.

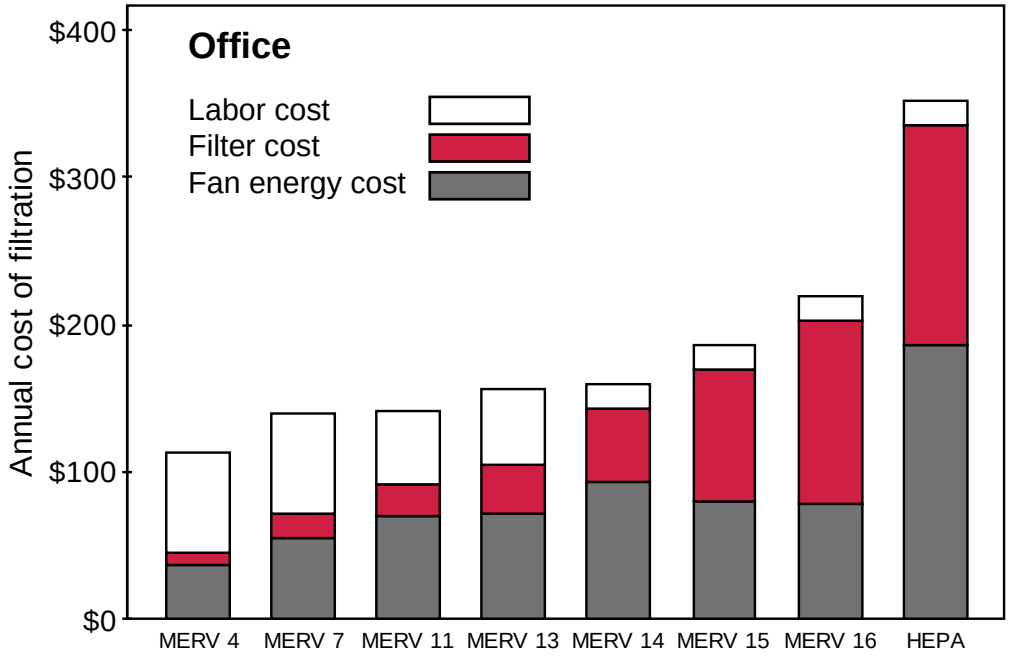

Figure 17. Estimated annual cost of filtration in the hypothetical office environment

In the office environment, the total annual costs of filtration are expected to be less than $200 for all filters below MERV 16. A HEPA filter is expected to cost over $350 per year.

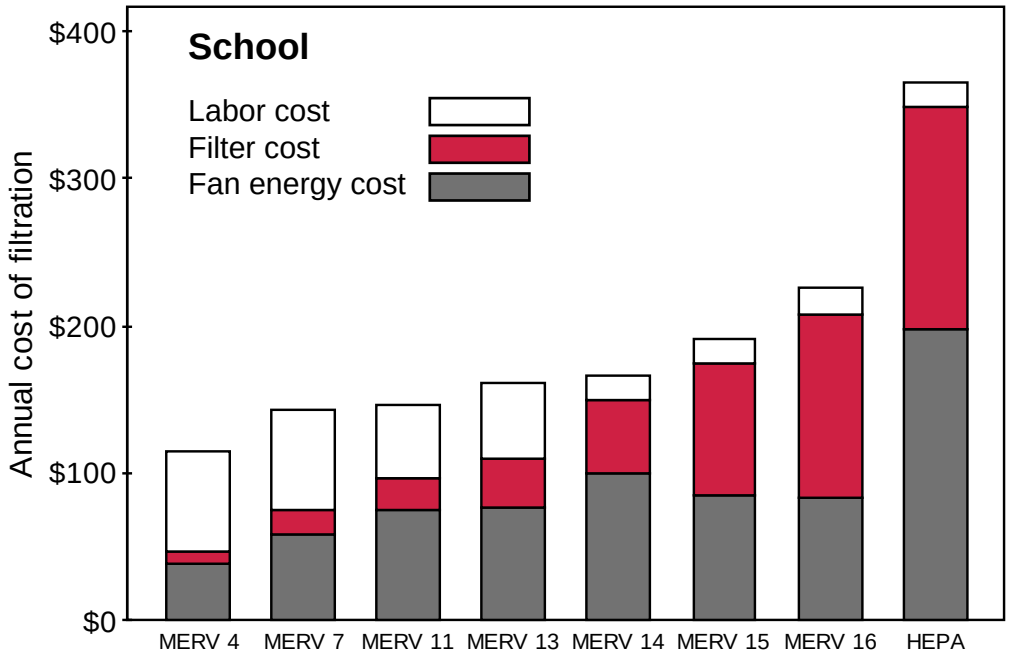

Figure 18. Estimated annual cost of filtration in the hypothetical school classroom environment

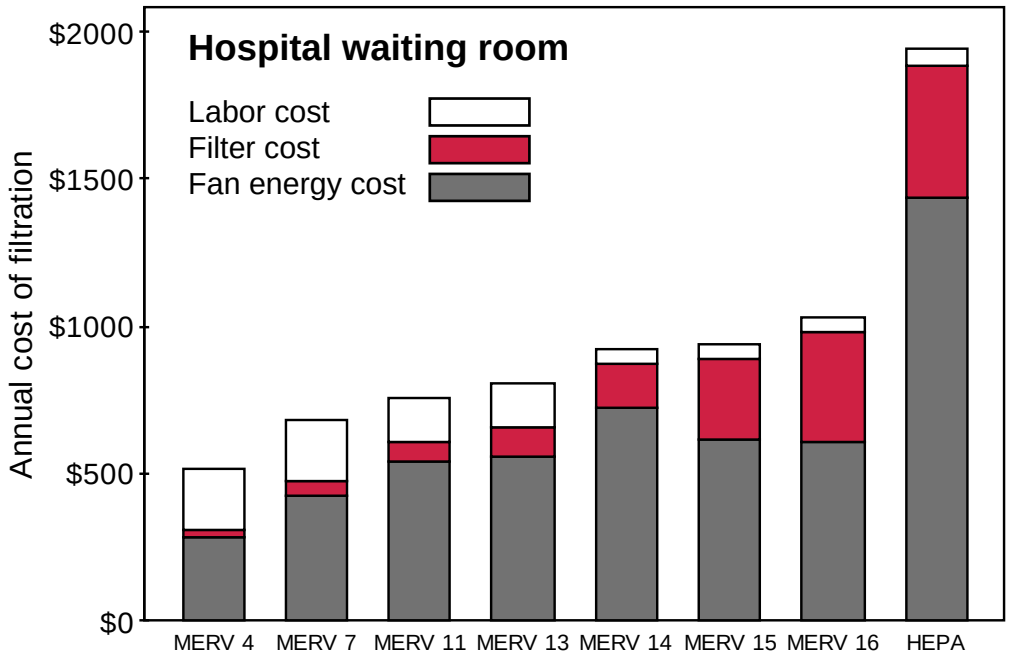

Figure 19. Estimated annual cost of filtration in the hypothetical hospital waiting room environment

Filtration costs for the school classroom environment are expected to be similar to the office environment because of the similar rates of airflow through the HVAC filter and operation time. Filtration costs for the hypothetical hospital waiting room environment are expected to be much higher because of the longer operation times combined with greater airflow rates. MERV 11-16

filters are expected to cost between $750 and $1050 annually. HEPA filtration is expected to cost nearly $2000 per year.

Note that considerable uncertainty exists in not only the cost estimates but in the typical life spans and replacement schedules. Future research should explore the sensitivity to these assumptions, but these values are considered appropriate for now.

Comparing the cost of HVAC filtration to outdoor air ventilation

One way to compare these estimates of total annual filtration costs and total annual outdoor air ventilation costs is to normalize them by their ability to remove infectious droplet nuclei from indoor air. Therefore, Figure 20 shows the total cost *per unit removal rate* of both outdoor air ventilation (OA) in each of the four cities explored previously and equivalent HVAC filtration (i.e., these values are total annual costs divided by the assumed rate of droplet nuclei removal in each environment). Thus the units are in $ per 1/hour of removal. This approach allows for direct comparison between OA ventilation and HVAC filtration, although it does not provide information about the magnitude of achievable removal rates in particular environments.

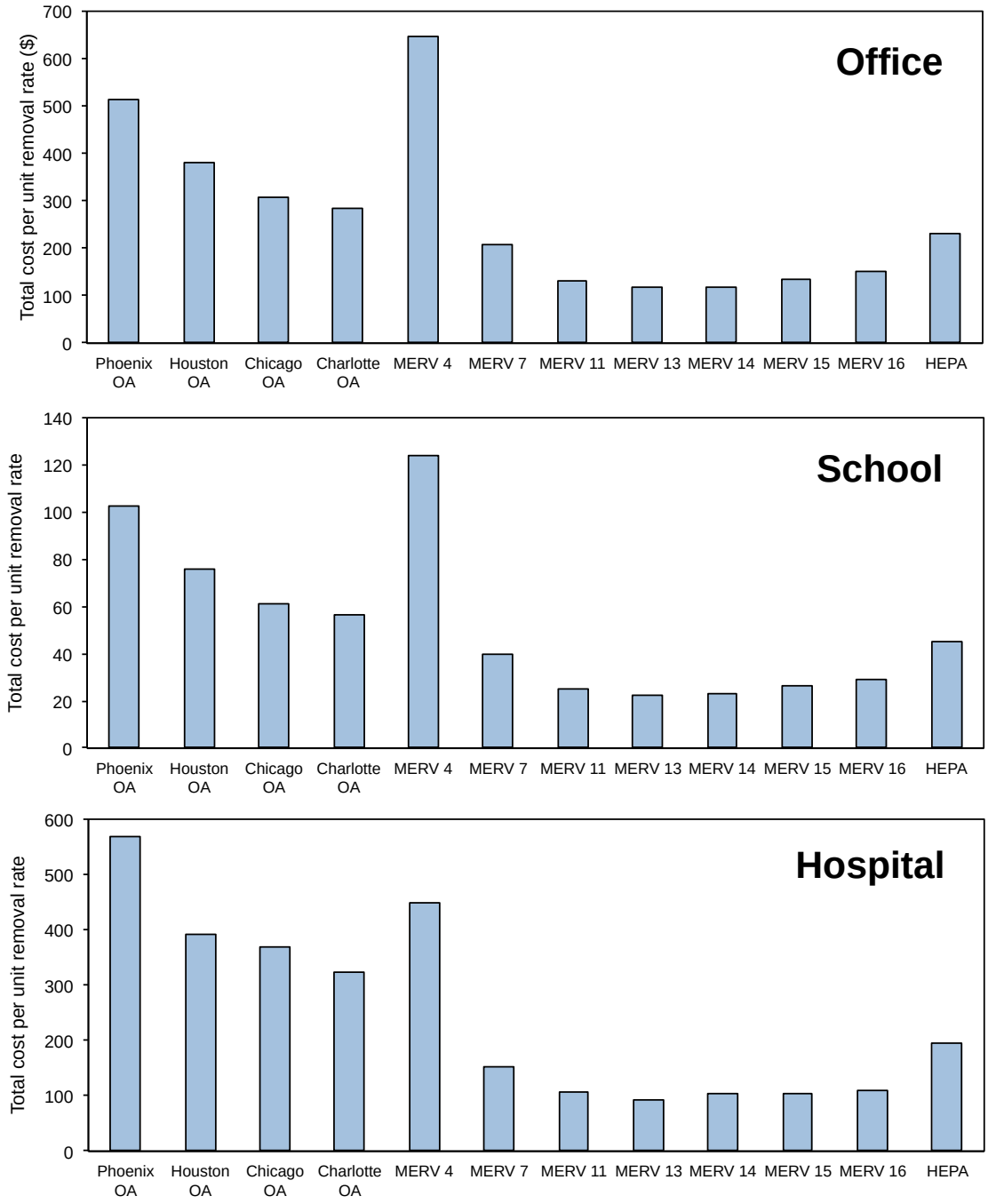

Figure 20. Total annual cost per unit removal rate ($ per 1/hr) of outdoor air ventilation and HVAC filtration products in each of the three hypothetical case study environments

Note that in each environment, MERV 13, 14, and 15 filtration products generally provided the least expensive removal mechanism (in terms of $ per 1/hour of infectious droplet nuclei removal rate). This is attributed to both relatively high effectiveness (as described in the risk modeling) and relatively low costs. Even HEPA filtration is expected to cost less on an annual basis than outdoor air ventilation in all environments. MERV 4 is the only filtration product that is more expensive than outdoor air ventilation, primarily because of its very low effectiveness.

While Figure 20 is helpful for comparing normalized costs of particle removal, Figures 21-23 plot the mean predicted relative risk (RR) values from the Wells-Riley modeling in each environment (average across each of the three diseases, as RR values were similar) versus the total cost of providing that risk reduction with HVAC filtration. To be able to compare outdoor air (OA) ventilation in each location directly to HVAC filtration, OA ventilation rates were adjusted to achieve the equivalent removal rate of filtration and are also shown in Figures 21-23. For example, a MERV 15 filter is expected to achieve 1.37 per hour in infectious droplet nuclei removal in the office environment (as described earlier). For outdoor air ventilation to achieve the same risk reduction as filtration, the office environment would need to provide 1.37 air changes per hour of outdoor air ventilation. Thus, OA ventilation and HVAC filtration can be thought of as equivalent in terms of risk reduction (y-axis), but will differ in their cost estimates (x-axis). For each of the four geographic locations, the slope between ventilation energy costs and outdoor air ventilation rates from Figure 16 was used to estimate the cost of providing this equivalent reduction. Each case study is reported separately: Figure 21 describes the office environment; Figure 22 describes the school classroom; and Figure 23 describes the hospital waiting room. Full data table are listed in the Appendix.

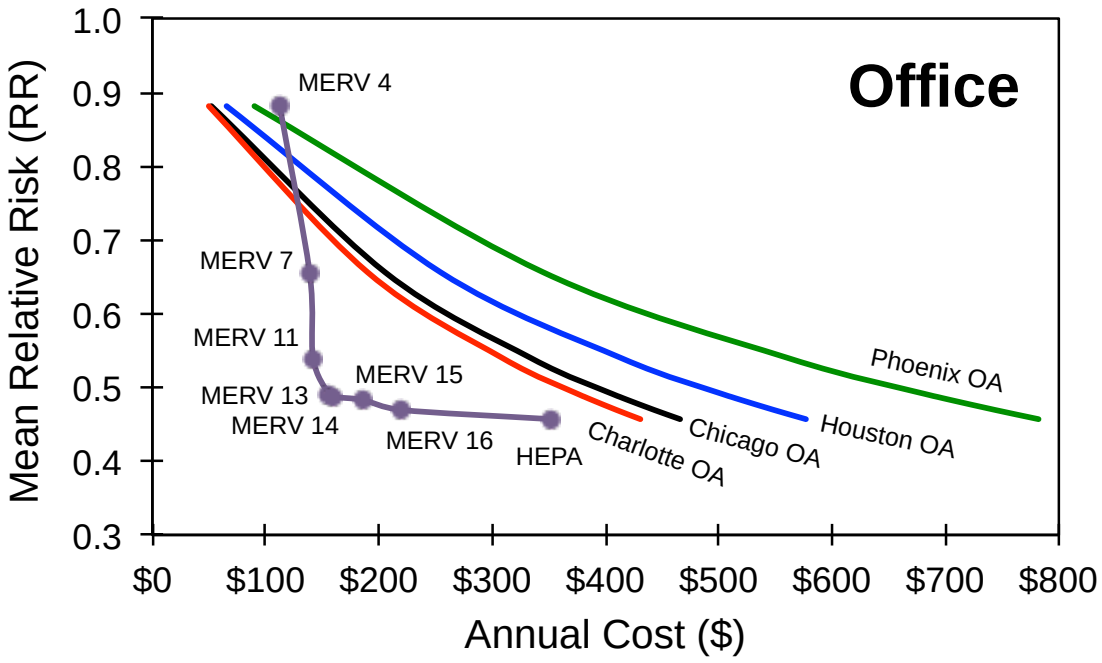

Figure 21. Relative risk (RR) of infectious disease transmission across the three diseases in the office environment with both HVAC filtration and "equivalent" outdoor air ventilation rates. Note that lower costs and lower risks (both beneficial) lie in the bottom left corner of the graph.

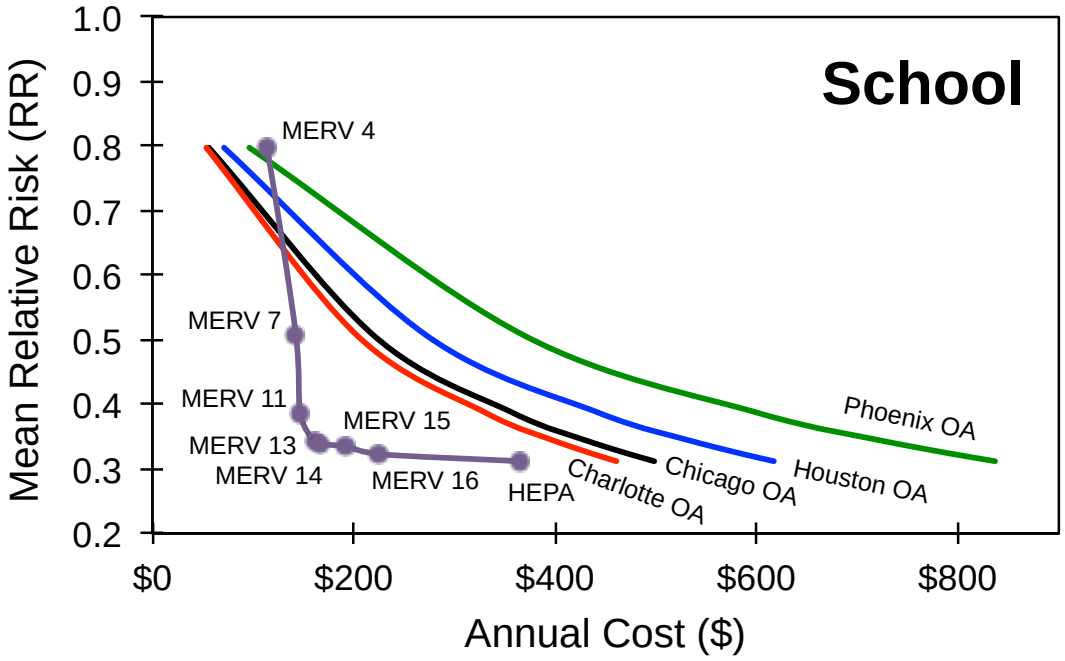

Figure 22. Relative risk (RR) of infectious disease transmission across the three diseases in the classroom environment with both HVAC filtration and "equivalent" outdoor air ventilation rates. Note that lower costs and lower risks (both beneficial) lie in the bottom left corner of the graph.

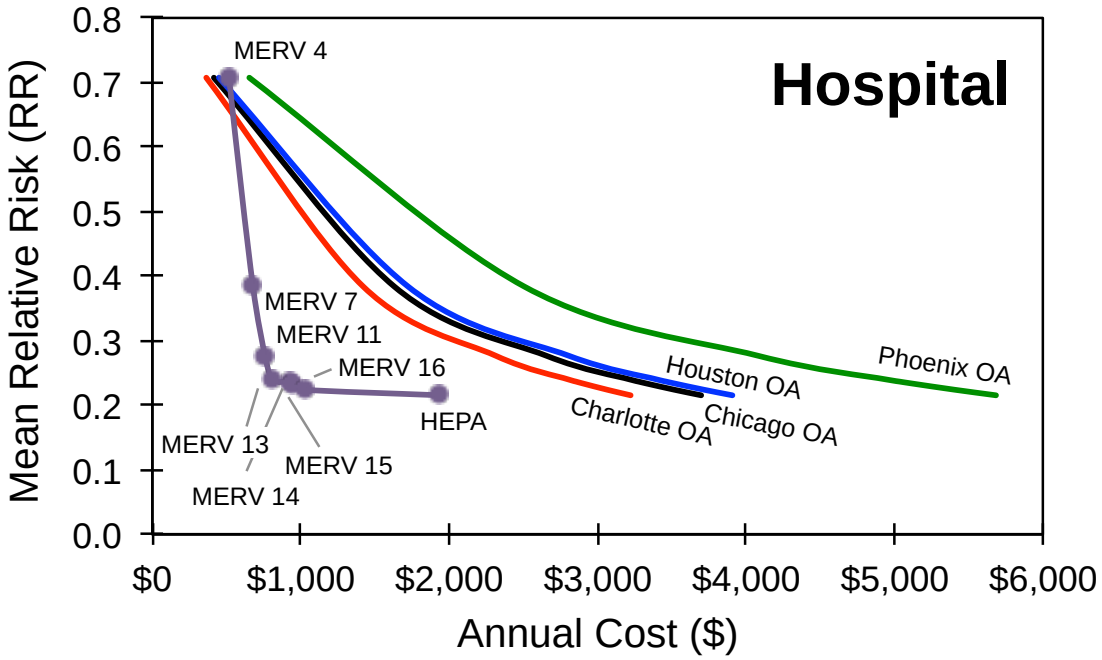

Figure 23. Relative risk (RR) of infectious disease transmission across the three diseases in the hospital waiting room environment with both HVAC filtration and "equivalent" outdoor air ventilation rates. Note that lower costs and lower risks (both beneficial) lie in the bottom left corner of the graph.

In Figures 21-23, the lowest cost and highest risk reduction cases appear closest to the bottom left corner. In the office and school environments, we predict that relative risks of 30-50% can be achieved by MERV 11-16 filtration products for the three infectious aerosols studied at a total annual cost including fan energy, replacement, and disposal of approximately $140-220. Equivalent amounts of outdoor air ventilation would cost approximately $300-800, depending on climate and level of risk reduction specified. In the hospital waiting room, MERV 13-16 appear to all achieve similar relative risks of only ~25% compared to no filtration at a cost of ~$800-1000 per year compared to ~$2000-5000 for equivalent risk reduction by outdoor air delivery. Medium efficiency filtration products (MERV 4-11) are also relatively inexpensive but are less effective in reducing infectious disease risks. Overall all, across the three environments, MERV 14-16 filtration products appear to be able to achieve substantial risk reductions for influenza, rhinovirus, and tuberculosis at much lower costs than an equivalent level of outdoor air ventilation.

In each of these three hypothetical environments it is clear that HVAC filtration products are predicted to achieve the greatest risk reductions at much lower costs of operation relative to providing an equivalent amount of outdoor air ventilation. The difference in costs between filtration and ventilation varies widely by climate and is highest in the warm climates of Houston and Phoenix.

Limitations and future research needs

It is clear that filtration of recirculated air may be able to reduce the transmission of airborne infectious diseases, according to the calculations herein. However, there is a tremendous amount of uncertainty involved in each step of this modeling effort, so a logical next step is to explore the sensitivity of the model to input parameters in much greater detail. While this case study of three hypothetical buildings and three types of infectious aerosol has been presented for instruction, more robust statistical techniques can be used to simulate a wide range of buildings, occupants, and infectious aerosol properties to provide a more generalizable estimate of the likely impacts of filtration across the building stock. Additionally, these risk models should be used in conjunction with more detailed hourly building energy balances to explore the energy impacts of infectious disease filtration relative to control by outdoor air ventilation, which will necessarily vary by climate, human occupancy and activity, and building operational characteristics. Additionally, it is clear that particle size distributions of expelled droplet nuclei should be measured in more standardized ways with much greater numbers of individuals. The discrepancies in reported size distributions lead us make broad assumptions about the average particle sizes of infectious aerosols, upon which every calculation herein is subsequently based. Future epidemiological work should also validate the predictions herein.

Summary and conclusion

In this work, an existing airborne infectious disease risk model (i.e., the Wells-Riley equation) was modified to include removal by recirculating HVAC filters and linked directly to the primary rating metric of ASHRAE Standard 52.2 for filtration products: MERV. Based on a

series of assumptions about infectious particle emissions from human activities, the risk of acquiring influenza, rhinovirus, and tuberculosis from a single infector in a range of three different environments was modeled with a particular focus on what different levels of HVAC filtration (i.e., MERV) could do to reduce those risks. Additionally, the costs of providing HVAC filtered air were compared to the costs of providing equivalent outdoor air ventilation.

Overall, HVAC filtration products are predicted to achieve the greatest risk reductions at the lowest cost of operation out of all these cases. MERV 13-16 appear to all achieve similar relative risks in these environments (compared to no filtration) at a cost much lower than equivalent outdoor air delivery, depending somewhat on climate and building type. Medium efficiency filtration products (MERV 7-11) are also inexpensive but are less effective in reducing infectious disease risks.

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Appendix: Data Tables

Table A1. Estimated costs of HVAC filtration in each location

Location	MERV 4	MERV 7	MERV 11	MERV 13	MERV 14	MERV 15	MERV 16	HEPA
Office	$112	$139	$142	$156	$160	$186	$220	$352
School	$115	$143	$147	$161	$167	$192	$225	$365
Hospital	$511	$677	$759	$808	$924	$935	$1031	$1939

Table A2. Estimated relative risks (RR) of infection in each location for each filter condition in each location

	Office			School classroom			Hospital waiting room			
Filter condition	*Flu*	*Rhinovirus*	*TB*	*Flu*	*Rhinovirus*	*TB*	*Flu*	*Rhinovirus*	*TB*	Mean
No filter	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
MERV 4	0.89	0.88	0.88	0.82	0.79	0.79	0.71	0.70	0.70	0.80
MERV 7	0.68	0.64	0.65	0.54	0.49	0.49	0.39	0.38	0.38	0.52
MERV 11	0.56	0.53	0.53	0.41	0.37	0.37	0.28	0.27	0.27	0.40
MERV 13	0.51	0.48	0.48	0.37	0.33	0.33	0.24	0.24	0.24	0.36
MERV 14	0.51	0.47	0.48	0.36	0.32	0.33	0.24	0.23	0.23	0.35
MERV 15	0.51	0.47	0.47	0.36	0.32	0.32	0.24	0.23	0.23	0.35
MERV 16	0.49	0.46	0.46	0.35	0.31	0.31	0.23	0.22	0.22	0.34
HEPA	0.48	0.44	0.45	0.34	0.30	0.30	0.22	0.21	0.21	0.33