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Effects of Surface Material, Ventilation, and Human Behavior on Indirect Contact Transmission Risk of Respiratory Infection

Gin Nam Sze-To,[1] Yang Yang,[2] Joseph K. C. Kwan,[3,4] Samuel C. T. Yu,[3,4] and Christopher Y. H. Chao[1,2,*]

Infectious particles can be deposited on surfaces. Susceptible persons who contacted these contaminated surfaces may transfer the pathogens to their mucous membranes via hands, leading to a risk of respiratory infection. The exposure and infection risk contributed by this transmission route depend on indoor surface material, ventilation, and human behavior. In this study, quantitative infection risk assessments were used to compare the significances of these factors. The risks of three pathogens, influenza A virus, respiratory syncytial virus (RSV), and rhinovirus, in an aircraft cabin and in a hospital ward were assessed. Results showed that reducing the contact rate is relatively more effective than increasing the ventilation rate to lower the infection risk. Nonfabric surface materials were found to be much more favorable in the indirect contact transmission for RSV and rhinovirus than fabric surface materials. In the cases considered in this study, halving the ventilation rate and doubling the hand contact rate to surfaces and the hand contact rate to mucous membranes would increase the risk by 3.7–16.2%, 34.4–94.2%, and 24.1–117.7%, respectively. Contacting contaminated nonfabric surfaces may pose an indirect contact risk up to three orders of magnitude higher than that of contacting contaminated fabric surfaces. These findings provide more consideration for infection control and building environmental design.

KEY WORDS: Hand contact; indirect contact transmission; indoor building material; quantitative risk assessment; respiratory infection

1. INTRODUCTION

There are three major modes of transmission of respiratory diseases: airborne transmission, droplet transmission, and indirect contact transmission.(1) Infectious particles can be deposited on solid surfaces. When people contact these contaminated surfaces and then contact their eyes, mouths, or noses, there could be a potential infection of respiratory diseases. This is referred to as the indirect contact transmission (or fomite transmission). Conjunctiva and nasal mucous membrane can also be a portal of entry for many respiratory pathogens.(2) Table I shows some examples. In buildings, infectious particles can be

[1]Building Energy Research Center, Fok Ying Tung Graduate School, The Hong Kong University of Science and Technology, Clear Water Bay, Hong Kong.
[2]Department of Mechanical and Aerospace Engineering, The Hong Kong University of Science and Technology, Clear Water Bay, Hong Kong.
[3]Health, Safety and Environment Office, The Hong Kong University of Science and Technology, Clear Water Bay, Hong Kong.
[4]Division of Environment, The Hong Kong University of Science and Technology, Clear Water Bay, Hong Kong.
*Address correspondence to Christopher Y. H. Chao, Department of Mechanical and Aerospace Engineering, The Hong Kong University of Science and Technology, Clear Water Bay, Hong Kong; meyhchao@ust.hk.

Table I. Portals of Entry of Some Pathogens Causing Respiratory Infection

Pathogen	Conjunctiva and nasal mucous membrane as portals of entry?
Measles virus	Yes
Varicella-zoster virus (chickenpox)	Yes
Influenza virus	Yes
SARS coronavirus	Yes
Variola virus (smallpox)	Yes
Respiratory syncytial virus	Yes
Rhinovirus	Yes
Adenovirus	Yes
M. Tuberculosis	No

deposited on indoor surface materials and clothing of occupants.

Although some studies suggested that indirect contact may not be the most dominant mode of transmission for some respiratory infectious diseases such as influenza, this mode still has a significant contribution to the transmission of respiratory infectious diseases.[2–6] In a risk assessment study, Nicas and Jones suggested that virus-contaminated hand contact with facial membranes contributed to 31% of the infection risk.[7] In an experimental infection study, 1 of 8 test animals acquired infection through indirect contact mode.[4] In addition, indirect contact transmission can pose a much longer duration for potential exposure than the other two modes of transmission.[8] This is because infectious particles only have the life times from a few seconds to hours in the indoor air, depending on their sizes, ventilation rate, and the airflow pattern.[9,10] However, viable pathogens contained in deposited particles can survive on inanimate surfaces from hours to weeks.[8,11] These surfaces become fomites, which can pose potential risk to susceptible persons for a long period of time, even after the sources of the infectious particles are removed.[8]

Factors affecting the risk of indirect contact transmission of a respiratory disease include (i) infectious source strength—the quantity of infectious agents introduced into the air during the exposure time interval: this influences the risk of other modes of transmission as well; (ii) human behavior—how often would the susceptible person contact the contaminated surfaces and his/her mucous membrane; (iii) ventilation—this affects the dispersion of the infectious particles in the space and the subsequent deposition of infectious particles on various surfaces; and (iv) surface material—this affects the amount of infectious particles that can be deposited on the material surface, the survivability of the pathogenic microorganisms on the surface, and the transfer rate of pathogenic microorganisms from the contaminated surface to the hand after a contact.

Although understanding has been established on the indirect contact transmission process of respiratory infection, little research has been done to evaluate the relative significance of these factors. Quantitative infection risk assessment provides a scientific way for such evaluation. The classical Wells-Riley equation used for infection risk assessment of respiratory disease only considers airborne transmission mode; dose-response model, on the other hand, is a more general exposure and risk assessment approach that can assess infection risk from multiple transmission modes.[12] To assess the risk of indirect contact transmission of respiratory diseases, the processes starting from the deposition of aerosolized pathogens onto surfaces and the transfer of pathogens from contaminated surfaces to hands and from hands to mucous membranes have to be considered. After each expiration action, more pathogens would be deposited on the contaminated surfaces. More pathogens would accumulate on the hand after each contact. The viability of the pathogen decays exponentially with time. Therefore, a nonlinear process should be considered. Nicas and Sun[13] have developed a Markov chain risk assessment model in which the indirect contact transmission is one of the exposure pathways. Their work has provided a foundation for this kind of research. Their model assumes a steady-state pathogen load on the contaminated surfaces for simplicity. Nicas and Best[14] have developed a model assessing the indirect contact transmission risk by considering an average pathogen load on hand. Atkinson and Wein[2] have developed an indirect contact transmission model with a simplification of assuming all the viruses on the susceptible hand to die off before each new hand contact of the contaminated surface. Wan *et al.*[5] used mathematical sequences to describe the indirect contact transmission process and to assess the exposure level. Wan *et al.*[15] used some integrals to replace some components in the mathematical sequences for simplification.

In this study, risk assessments were performed to evaluate the relative significance of human behavior, ventilation, and surface material on indirect contact transmission of respiratory infection. Three pathogens capable to be transmitted via indirect contact were considered: influenza A virus, respiratory syncytial virus (RSV), and rhinovirus. Influenza can spread rapidly to susceptible people by both direct and indirect routes. Although droplet transmission is thought to be the primary mode of influenza

transmission, there is also evidence to support indirect contact transmission of influenza.[16] RSV is the most important cause of respiratory infection in young children worldwide.[17] Although almost all children are vulnerable toward RSV, it can also infect adults. Rhinovirus is the most frequent cause of the common cold in both adults and children, which may cause serious respiratory tract disease and exacerbations of asthma.[18,19] In this work, the indirect contact risks of these three diseases were assessed in two indoor environments, a hospital ward and an aircraft cabin. Nosocomial infections among patients and health-care workers frequently occur in hospital environments. Due to high occupant density, the aircraft cabin is another typical environment where infection of respiratory diseases easily spread. Given the focus on the relative magnitude of risk, the model of Wan *et al.*[5] was simplified. The results revealed and quantified the relative importance of different factors in indirect contact transmission and provided additional information for consideration on infection control and building environmental design.

2. METHODS

2.1. Exposure and Risk Assessment Model

A simplified model based on the mathematical sequences developed by Wan *et al.*[5] was used. We consider a steady-state pathogen load on the contaminated surface to simplify the original model. Necessary parameters for the model are listed and explained in Table II. Assuming a surface without any pathogen, the index patient coughs f_s times per hour and an amount of N_x pathogen is deposited on a surface after each cough. A fraction c_h of the pathogen on that surface would be transferred to the hand after a contact. The decay rate of the pathogen on the surface is a. The frequency of hand-to-contaminated-surfaces contact is f_h. The relationship between the dose of pathogens on the contaminated surface E_s and time t is:

$$\frac{dE_s}{dt} = f_s N_x - aE_s. \tag{1}$$

Because there are continuous source and sink of pathogen on the contaminated surfaces, it is plausible to assume a steady-state pathogen load on the contaminated surface. When the pathogen load on the surface reaches the steady-state, the equation is:

$$0 = f_s N_x - a E_s. \tag{2}$$

Then, the pathogen load on the surface is:

$$E_s = \frac{f_s N_x}{a}. \tag{3}$$

Knowing the amount of pathogens on the contaminated surface, the dose of pathogens transferred to the hand surface after the first hand-to-contaminated-surface contact is:

$$E_h(1) = c_h \frac{f_s N_x}{a}, \tag{4}$$

where E_h is the dose of pathogens on the hand. The total dose of pathogens transferred to the hand after the second hand contaminated surface is:

$$E_h(2) = c_h \frac{f_s N_x}{a}\left(e^{-\frac{b}{f_h}} + 1\right), \tag{5}$$

where b is the exponential decay constant of the pathogen on the hand. The total dose of pathogens transferred to the hand after the n^{th} hand-to-contaminated-surface contact is therefore,

$$E_h(n) = c_h \frac{f_s N_x}{a}\left(1 + e^{-\frac{b}{f_h}} + \cdots + e^{-(n-1)\frac{b}{f_h}}\right). \tag{6}$$

The expansion is a finite geometric series and can be simplified as follows:

$$E_h(n) = c_h \frac{f_s N_x}{a} \cdot \frac{1 - e^{-n\frac{b}{f_h}}}{1 - e^{-\frac{b}{f_h}}}. \tag{7}$$

The dose of pathogens delivered to the mucous membrane of the susceptible person from the hand can also be estimated based on Equations (3) and (7). The dose of pathogens delivered to the mucous membrane after the first hand-to-mucous-membrane contact is:

$$E_m(1) = 1 \times E_h\left(\frac{f_h}{f_m}\right), \quad \text{for} \quad f_h > f_m, \tag{8}$$

where E_m is the dose of pathogens on the mucous membrane and f_m is the frequency of the hand-to-mucous-membrane contact. The total dose of pathogens delivered to the mucous membrane after the second hand-to-mucous-membrane contact is:

$$E_m(2) = 2 \times E_h\left(\frac{f_h}{f_m}\right), \quad \text{for} \quad f_h > f_m. \tag{9}$$

The total amount of pathogens delivered to the mucous membrane after the pth hand-to-mucous-membrane contact is:

$$E_m(p) = p \times E_h\left(\frac{f_h}{f_m}\right) = p \times \frac{c_h f_s N_x}{a} \times \frac{1 - e^{-\frac{b}{f_m}}}{1 - e^{-\frac{b}{f_h}}}, \quad \text{for} \quad f_h > f_m. \tag{10}$$

Table II. Necessary Parameters for Indirect Contact Exposure and Risk Assessments

Parameters	Description
Pathogen load on the contaminated surface	To obtain this parameter, the amount of expiratory aerosols deposited on the contaminated surface should be estimated first. This can be achieved by numerical modeling: construct a numerical model of the premises in the study, the fates of the expiratory aerosols are then simulated numerically and the number of aerosols deposited on the susceptible surface could be counted. A validated numerical modeling scheme should be used. The amount of deposited expiratory aerosols can also be obtained by conducting experiments: tracer particles are injected into the premises in study to simulate the expiratory aerosols and the particles deposited on the susceptible surface are then extracted and measured for number counts.[18,19] The experimental method would provide more accurate results but it is more time consuming than the numerical modeling. Knowing the amount of expiratory aerosols deposited on the contaminated surface, the pathogen load on the surface can be calculated by multiplying the volume of the expiratory droplets by the pathogen concentration in the respiratory fluid and factoring with the loss of viability of the pathogen due to the desiccation effect during aerosolization.[20]
Transfer rate of pathogen between surfaces	The transfer rate depends on the species of the pathogen and the material type of the inanimate surface. Many transfer rate data are available in the literature. Fabric/porous material has a higher transfer rate than nonfabric/nonporous material in general.[21]
Viability of pathogen on the contaminated surface	Viability of pathogen on solid surface is also dependent on the species of the pathogen and the material type of the inanimate surface as well as other environmental factors such as sunlight and temperature. Pathogens decay more rapidly on skin than on inanimate surfaces. Pathogens usually survive on skin for less than an hour or up to a few hours only.[8]
Contact rates	The hand-to-contaminated-surface- and hand-to-mucous-membrane contact rates depend on the individuals' personal and working behaviors. These parameters can be obtained by conducting observational studies. Currently, there are only limited studies available.[22,23]

In the case of $f_h \leq f_m$, the total amount of pathogens delivered to the mucous membrane will be:

$$E_m(n) = n \times \frac{c_h f_s N_x}{a} \times e^{-\frac{b}{f_m}}. \quad (11)$$

Equations (10) and (11) estimate the dose of pathogens delivered to the mucous membrane if all pathogens on the hand are delivered to the mucous membrane. No study reporting the transfer efficiency of pathogens from the hand to the mucous membrane was found except one, which reported that about 34% of viruses on the finger pad will transfer to the lip after a contact.[24] The transfer efficiency of microorganisms between surfaces with moisture was 1–2 orders higher than that without moisture.[25] Mucous membranes in the nostrils and eyes are generally much moister than lips. Based on these considerations, the transfer efficiency from the finger pad to the mucous membrane is assumed to be 1 for simplicity, in which all the pathogens on the finger pad are transferred to the mucous membrane system after each contact.[5]

Based on the above equation, we can calculate the dose of pathogens delivered to the mucous membrane. Then, the dose-response equation can be used to calculate the infection risk via indirect contact transmission:

$$P_I = 1 - \exp[-rE_m], \quad (12)$$

where P_I is the probability of infection and r is a fitting parameter evaluating the infectivity of the pathogen,[12] which can be calculated from infectious dose data. Even if the virus gets to the mucous membrane, if the person has protective immunoglobulin A (IgA), viral attachment will not occur and thus infection cannot occur. The susceptibility of receptor is also reflected in r.

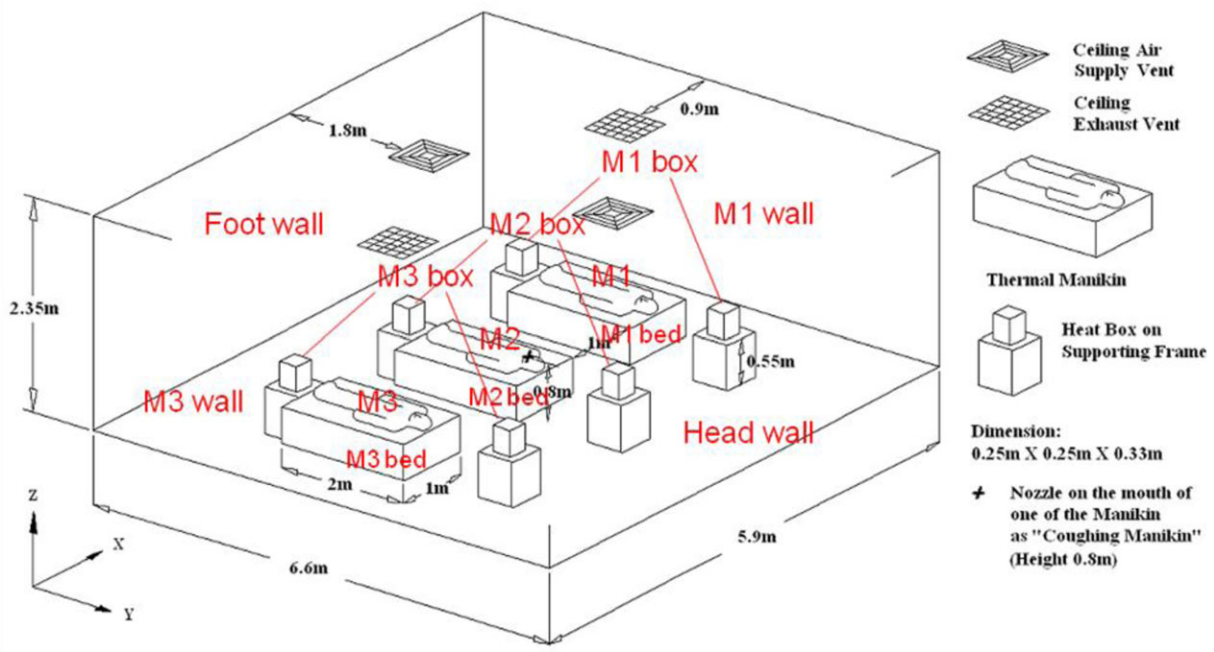

Fig. 1. The hospital ward.

2.2. The Scenarios

Infection risks of three respiratory diseases via indirect contact transmission were assessed in two environments, a hospital ward and an aircraft cabin. The infectious particle deposition data were obtained from two previous studies.[5,26] In the two studies, the infectors generated infectious particles via coughing. The polydispersed size profile of cough droplets and the cough air jet velocity were considered in the two studies. The dispersion of the infectious particles and their deposition on different surfaces were modeled.

A hospital ward that contains three beds and three patients was considered. Fig. 1 shows the configuration of the scenario. The ward had dimensions of 5.9 m × 6.6 m × 2.35 m ($W \times L \times H$). Two four-way-spread type ceiling supply diffusers, 0.6 m × 0.6 m, were installed to supply ventilation air and two ceiling exhaust vents, also of dimensions 0.6 m × 0.6 m, were installed. Three beds with dimensions of 1 m × 2 m × 0.6 m ($W \times L \times H$) were placed in the ward with a 1 m separation distance in between. A manikin was placed on each bed to represent a lying patient. The middle one was the index patient, which generated infectious particles by coughing. A numerical model was constructed based on the ward configurations, and the amount of the deposited infectious particles on each surface was calculated based on the numerical particle deposition data. The numerical modeling result was validated experimentally using polydispersed droplets mimicking human cough droplets size profile and nonvolatile content. The hospital ward and the modeling on the infectious particle deposition as well as the experimental validation of the results are available in Chao *et al.*[26] with greater details.

Two total air changes per hour (ACH) were considered in the hospital ward, 11.6 and 6 ACH. Two coughing directions of the index patient on bed, both upward and laterally toward patient M3 were also considered. A health-care worker was assumed to work in the ward for 8 hours. The worker's contact frequency of hand contaminated surfaces was assumed to be 3 times per hour, on the blanket on patient M1 (surface area: 1.93 m^2). The patients stayed in the ward for 24 hours but 8 hours of sleeping time of the patients was not considered. The contact frequency of hand contaminated surfaces was assumed to be 3 times per hour for patients. The patients were assumed to touch the blankets on them.

In the aircraft cabin scenario, an aircraft cabin section consists of three rows of seven seats and two

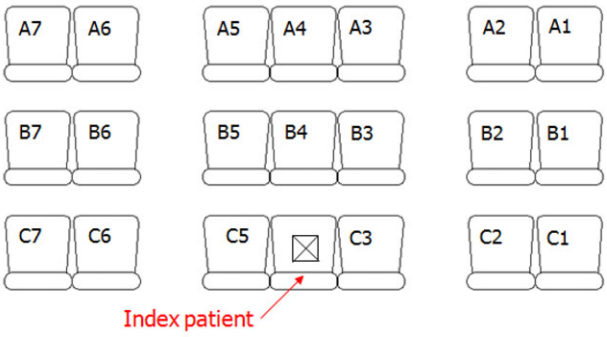

Fig. 2. The aircraft cabin.

aisles were considered. Fig. 2 shows the configuration of this scenario. The internal geometry of this cabin mockup had dimension of 4.9 m × 3.2 m × 2.1 m ($W \times L \times H$). Two longitudinal overhead ventilation air supply slots with dimensions of 12 mm W × 3,200 mm L were equipped at the middle part of the ceiling. The return air was extracted through two perforated ducts installed along the lower longitudinal edge of the floor, one on each side. Numerical geometry was constructed based on this aircraft cabin mockup. Airflow, transient particle dispersion, and deposition were then simulated. The numerical modeling result was validated experimentally using polydispersed droplets mimicking human cough droplets size profile and nonvolatile content. Information on the experimental validation and the particle deposition modeling results are available in Wan *et al.*[5] with greater details. A 4-hour flight was considered. C4 was the index passenger who generated infectious particles by coughing. The amount of the pathogens that was generated from the index passenger's cough-generated infectious particles and then deposited onto each surface was calculated based on the particle deposition data.

Two total ACH were considered in the aircraft cabin mock-up, 9.8 and 19.5 ACH. The passengers were assumed to have a contact frequency of hand to contaminated surfaces of 3 times per hour. The passengers were assumed to contact the back of the seat (surface area: 0.336 m^2) right in front of them during the flight.

Using the risk assessment model, Equation (12), and the parameters of the three viruses obtained from literature (Table III), risk assessments of the three diseases were conducted for the two environments. To evaluate the significance of contact frequencies on the risk of indirect contact transmission, the infection risks of the health-care worker were also assessed under a doubled hand-to-contaminated-surfaces contact frequency and hand-to-mucous-membranes contact frequency for comparison. Indirect contact transmission risks of the health-care worker were also compared between contacting fabric and nonfabric contaminated surfaces to evaluate the significance of indoor surface material on this transmission mode. In Table III, the transfer efficiencies of pathogens from the contaminant surface to the finger pad and the decay rate of the pathogens on the surface are further classified into data for fabric materials (or porous materials) and nonfabric materials (or nonporous materials). Effect of the material type on the quantity of infectious particle deposition will be discussed in the next section. The pathogen transfer efficiency from the contaminated surface to the hand, c_h in Table III, was further factored by the ratio of the finger pad area to the contaminated surface area. In this study, the finger pad area was taken as 6.5 cm^2.

3. RESULTS AND DISCUSSIONS

3.1. Effect of Ventilation Rate

Table IV shows the infection risks of the patients and the health-care worker in the hospital ward scenario under 6 and 11.6 ACH. It was expected that the infection risks to the diseases should generally increase with the reduction of the ACH in the ward because a higher ventilation rate should more efficiently dilute the infectious particles and remove them from the indoor environment before they can deposit on the surfaces. However, this was not always observed. In the upward cough case, when the ventilation rate was nearly doubled, the infection risks of patient M1 and the health-care worker to the three diseases were reduced by 9.5%, but the infection risks of patient M3 to the three diseases were increased by 7.9%. In the lateral cough case, when the ventilation rate was nearly doubled, the infection risks of patient M1 and the health-care worker to the three diseases were also increased by 31.1%. Changing the air exchange rate changes advection and turbulence, thus changing the deposition patterns of infectious particles. Under the higher ventilation rate, more infectious particles were dispersed to some positions by the stronger air flow. Therefore, more pathogens were deposited on the surfaces around these positions in the higher ACH case,

Table III. Parameters of Influenza A Virus, RSV, and Rhinovirus Used in the Infection Risk Assessment

Pathogen	Influenza A Virus		RSV		Rhinovirus	
Parameter	Value	Remarks	Value	Remarks	Value	Remarks
c	5×10^5 $TCID_{50}$	Median concentration from 7 patients[27]	3.63×10^5 $TCID_{50}$/mL	Average of three volunteers[32]	1600 $TCID_{50}$/mL	Average of 4 volunteers[20]
Human infectious dose	$ID_{50} = 223.5$ $TCID_{50}$ $r = 0.0031$	Nasal infectious dose, for large aerosols and mucous membrane (mean value of the range: 127–320)[23]	$ID_{50} = 1.0\times10^4$ $TCID_{50}$ $r = 6.93\times10^{-5}$	The 50% human infectious dose when given by nose, mouth, or eye[32]	$ID_{50} = 0.032$ $TCID_{50}$ $r = 21.66$	The nasal ID_{50}[22]
f_s	18/hr	Median cough frequency of 60 patients[28]	18/hr	Median cough frequency of 60 patients[28]	2.9/hr	Average from 5 people[3]
f_m	0.7/hour	Frequency of eye-rubbing and nose-picking of 124 adults[29]	0.7/hour	Frequency of eye-rubbing and nose-picking of 124 adults[29]	0.7/hour	Frequency of eye-rubbing and nose-picking of 124 adults[29]
f_h	3/hour	Assumption	3/hour	Assumption	3/hour	Assumption
c_h	0.00251	Transfer efficiency of influenza virus to finger pad from handkerchiefs and paper facial tissue[9]	0.000126	Transfer efficiency of RSV to finger pad from paper[33]	0.0001	Transfer efficiency of rhinovirus to finger pad from fabrics and paper[35]
	0.0794	Transfer efficiency of influenza virus to finger pad from stainless steel and plastic[9]	0.0000997	Transfer efficiency of RSV to finger pad from countertop[33]	0.21	Transfer efficiency of rhinovirus to finger pad from steel, table, pen[35]
a	0.027	On bed sheet[30]	5.756	On paper[34]	0.554	On fabric and paper tissue[3]
	0.75	Influenza A virus survival on glass[30]	0.0025	RSV virus survival on glass[34]	0.28	Rhinovirus survival on glass[3]
b	6.4	Influenza virus survived on skin[31]	0.36	RSV virus survival on skin[34]	2.04	Rhinovirus virus survival on skin[3]

a and b were obtained using exponential fitting. For data only reported the maximum duration, it is assumed that 99.9% of viruses were inviable at the reported maximum duration.

Table IV. Infection Risk via Indirect Contact Transmission in the Hospital Ward Case (Numbers Inside the Parentheses Indicate the Percentage Differences)

	Susceptible	Infection risk (cough upward)	Infection risk (cough laterally)
Influenza A virus			
11.6 ACH	Patient M1	1.20×10^{-5}	7.99×10^{-8}
	Patient M3	1.20×10^{-5}	2.63×10^{-4}
	Health-care worker	5.43×10^{-6}	3.63×10^{-8}
6 ACH	Patient M1	1.32×10^{-5} (+10%)	6.10×10^{-8} (–23.7%)
	Patient M3	1.11×10^{-5} (–7.5%)	3.05×10^{-4} (+16%)
	Health-care worker	6.00×10^{-6} (+10.5%)	2.77×10^{-8} (–23.7%)
RSV			
11.6 ACH	Patient M1	5.35×10^{-11}	3.57×10^{-13}
	Patient M3	5.35×10^{-11}	1.18×10^{-9}
	Health-care worker	2.43×10^{-11}	1.62×10^{-13}
6 ACH	Patient M1	5.91×10^{-11} (+10.5%)	2.73×10^{-13} (–23.5%)
	Patient M3	4.95×10^{-11} (–7.5%)	1.36×10^{-9} (+15.3%)
	Health-care worker	2.69×10^{-11} (+10.7%)	1.24×10^{-13} (–23.5%)
Rhinovirus			
11.6 ACH	Patient M1	4.71×10^{-10}	3.15×10^{-12}
	Patient M3	4.71×10^{-10}	1.04×10^{-08}
	Health-care worker	2.14×10^{-10}	1.43×10^{-12}
6 ACH	Patient M1	5.20×10^{-10} (+10.4%)	2.40×10^{-12} (-23.8%)
	Patient M3	4.36×10^{-10} (–7.4%)	1.20×10^{-8} (+15.4%)
	Health-care worker	2.37×10^{-10} (+10.7%)	1.09×10^{-12} (–23.8%)

resulting in higher exposure to some patients and the health-care worker. When the ventilation rate was nearly doubled, the average infection risk among the patients and health-care worker to the three diseases in the upward cough case was reduced by 3.7%, but was increased by 16.2% in the lateral cough case.

Table V shows the infection risks of the passengers in the aircraft cabin scenario after a 4-hour flight under 9.8 and 19.5 ACH. Out of the 20 susceptible passengers, 12 passengers had reduced infection risks to the three diseases when the ventilation rate in the cabin was nearly doubled. In contrast, the other eight passengers, seated at A6, B6, C7, A3, B3, C3, B1, and C1, had increased infection risks to the three diseases when the ventilation rate in the cabin was nearly doubled. This was also due to the air flow under the higher ACH bringing more infectious particles to some regions in the cabin. Therefore, the particle deposition on some surfaces in these regions increased, resulting in higher pathogen loads on these contaminated surfaces. Doubling the ventilation rate reduced the average infection risk of the susceptible passengers to the three diseases by about 5.6%.

The relation between ventilation rate and infection risk of respiratory diseases has been studied systematically both by numerical work and by experimental work. It has been conclusively recognized that higher ventilation rate has an advantage in reducing the infection risk of respiratory diseases by airborne transmission. However, from the results of the current study, the indirect contact transmission risks were not always reduced when ventilation rate is increased in some cases. Doubling the ventilation rate only decreases the average risk by less than 20%. In a previous study, the infection risk through airborne transmission was decreased by 41% on average when the ventilation rate was nearly doubled,[5] indicating that ventilation rate has a lower significance on indirect contact transmission than it does on airborne transmission. In both environments, there were some susceptibles who had higher infection risks under a higher ventilation rate. As the air flow pattern under different ventilation rates are different, increase in ventilation rate may disperse more infectious particles to some regions in the premises and outweigh the dilution effect in these regions. One experimental study also found that higher ventilation rate might increase the exposure level of respiratory pathogens for some susceptibles located further away from the sources due to the same reason.[36]

Table V. Infection Risks via Indirect Contact Transmission in the Aircraft Cabin

ACH	A7	A6	A5	A4	A3	A2	A1
Influenza							
9.8	9.02×10^{-7}	6.38×10^{-7}	1.26×10^{-5}	1.46×10^{-5}	1.11×10^{-5}	9.23×10^{-7}	1.13×10^{-6}
19.5	7.45×10^{-7}	6.69×10^{-7}	1.22×10^{-5}	1.19×10^{-5}	1.37×10^{-5}	8.01×10^{-7}	9.12×10^{-7}
	B7	B6	B5	B4	B3	B2	B1
9.8	7.01×10^{-6}	6.08×10^{-6}	3.37×10^{-4}	8.64×10^{-4}	2.99×10^{-4}	5.94×10^{-6}	5.83×10^{-6}
19.5	6.75×10^{-6}	6.09×10^{-6}	3.18×10^{-4}	7.79×10^{-4}	3.13×10^{-4}	5.48×10^{-6}	5.88×10^{-6}
	C7	C6	C5	C4	C3	C2	C1
9.8	6.47×10^{-7}	7.52×10^{-7}	1.77×10^{-5}	Index passenger	1.43×10^{-5}	1.38×10^{-6}	1.03×10^{-6}
19.5	6.96×10^{-7}	6.03×10^{-7}	1.44×10^{-5}		1.62×10^{-5}	9.15×10^{-7}	1.08×10^{-6}
RSV							
9.8	4.03×10^{-12}	2.86×10^{-12}	5.6×10^{-11}	6.52×10^{-11}	4.97×10^{-11}	4.13×10^{-12}	5.06×10^{-12}
19.5	$3.33 \times 10^{-1}2$	2.99×10^{-12}	5.45×10^{-11}	5.31×10^{-11}	6.11×10^{-11}	3.58×10^{-12}	4.08×10^{-12}
	B7	B6	B5	B4	B3	B2	B1
9.8	3.14×10^{-11}	2.72×10^{-11}	1.51×10^{-9}	3.87×10^{-9}	1.34×10^{-9}	2.66×10^{-11}	2.61×10^{-11}
19.5	3.02×10^{-11}	2.72×10^{-11}	1.43×10^{-9}	3.49×10^{-9}	1.40×10^{-9}	2.45×10^{-11}	2.63×10^{-11}
	C7	C6	C5	C4	C3	C2	C1
9.8	2.90×10^{-12}	3.36×10^{-12}	7.90×10^{-11}	Index passenger	6.42×10^{-11}	6.16×10^{-12}	4.59×10^{-12}
19.5	3.11×10^{-12}	2.70×10^{-12}	6.43×10^{-11}		7.23×10^{-11}	4.09×10^{-12}	4.85×10^{-12}
Rhinovirus							
9.8	3.55×10^{-11}	2.52×10^{-11}	4.96×10^{-10}	5.74×10^{-10}	4.38×10^{-10}	3.64×10^{-11}	4.46×10^{-11}
19.5	2.93×10^{-11}	2.64×10^{-11}	4.80×10^{-10}	4.68×10^{-10}	5.38×10^{-10}	3.16×10^{-11}	3.59×10^{-11}
	B7	B6	B5	B4	B3	B2	B1
9.8	2.76×10^{-10}	2.40×10^{-10}	1.33×10^{-8}	3.40×10^{-8}	1.18×10^{-8}	2.34×10^{-10}	2.30×10^{-10}
19.5	2.66×10^{-10}	2.40×10^{-10}	1.26×10^{-8}	3.07×10^{-8}	1.24×10^{-8}	2.16×10^{-10}	2.32×10^{-10}
	C7	C6	C5	C4	C3	C2	C1
9.8	2.55×10^{-11}	2.96×10^{-11}	6.96×10^{-10}	Index passenger	5.65×10^{-10}	5.42×10^{-11}	4.05×10^{-11}
19.5	2.74×10^{-11}	2.38×10^{-11}	5.66×10^{-10}		6.37×10^{-10}	3.60×10^{-11}	4.27×10^{-11}

Table VI. Infection Risks of Health-Care Worker Under Two Hand-to-Contaminated-Surface Contact Frequencies and Two Hand-to-Mucous-Membrane Contact Frequencies (Numbers Inside the Parentheses Indicate the Percentage Differences)

	Influenza A virus	RSV	Rhinovirus
f_h			
3/hour	5.43×10^{-6}	2.43×10^{-11}	2.14×10^{-10}
6/hour	7.3×10^{-6} (+34.4%)	4.72×10^{-11} (+94.2%)	3.66×10^{-10} (+71%)
f_m			
0.7/hour	5.43×10^{-6}	2.43×10^{-11}	2.14×10^{-10}
1.4/hour	1.18×10^{-5} (+117.3%)	3.01×10^{-11} (+23.9%)	3.82×10^{-10} (+78.5)

3.2. Effect of Contact Frequencies

Table VI shows the infection risks of the health-care worker in the hospital ward scenario under two hand-to-contaminated-surface contact frequencies. The health-care worker was considered to contact the blanket on patient M1 in the 11.6 ACH, index patient coughing upward case. By doubling the hand-to-contaminated-surface contact frequency, the infection risk of the health-care worker increased by 34.4% for influenza A virus, 94.2% for RSV, and 71.2% for rhinovirus.

Table VI also shows the infection risks of the health-care worker in the hospital ward scenario under two hand-to-mucous-membranes contact frequencies. The health-care worker was also considered to contact the blanket on patient M1 in the 11.6 ACH, index patient coughing upward case. By doubling the hand-to-mucous-membranes contact frequency, the infection risk of the health-care worker increased by 117.7% for influenza A virus, 24.1% for RSV, and 78.4% for rhinovirus.

The mean frequency of eye-rubbing and nose-picking used in this study was 0.7 per hour, obtained

from Hendley *et al*.,[29] in which 124 adult subjects were observed for around 5 hours. During the observation, very diverse personal behaviors were found. Some participants kept repeating eye-rubbing and nose-picking activities; others rarely touched their eyes, mouths, or noses. For those participants who tend to touch their mucous frequently, their infection risk would obviously be higher. For comparison purpose, every susceptible person was considered to have the same contact frequency. The three pathogens showed different sensitivities of indirect contact transmission risk to contact frequency. As the contact frequencies are nonlinearly related to the exposure, the variation of risk among the three diseases was larger when the contact frequencies were nearly doubled than when the ventilation rates were nearly doubled. The contact frequencies were found to have more significant effect on the risk of indirect contact transmission than the ventilation rate. Therefore, it is more effective to lower the infection risk via indirect contact transmission by reducing the hand contacts than controlling the ventilation rate.

3.3. Effect of Surface Material

Table VII shows the infection risk comparison between different fabric and nonfabric surface materials. The health-care worker was considered to contact the blanket on patient M1 in the 11.6 ACH, index patient coughing upward case. The pathogen transfer fraction and survival on nonfabric material surface data are shown in Table III. If the surface contacted by the health-care worker was a nonfabric instead of a fabric surface, his/her infection risks would be increased by 13.9% for influenza A virus, 1,820.8% for RSV, and 4,154.0% for rhinovirus. The huge differences for RSV and rhinovirus were due to the large differences in survival on surface and transfer fraction, respectively, between fabric and nonfabric surface materials. From the literature data we found, RSV decay rate on nonfabric material is three orders of magnitude lower than that on fabric material and rhinovirus transfer fraction from nonfabric surface is three orders of magnitude higher than that from fabric material. These properties made nonfabric material much more effective in the indirect contact transmission of RSV and rhinovirus.

The infection risk values reported in Table VII have not included the effect of surface material on the amount of particle deposition. Fabric and nonfabric materials have different deposition rates of particles. The deposition rate also depends on air flow conditions. A study by Wu *et al*.[37] found that the particle deposition rate on fabric material (wallpaper) was higher than some nonfabric materials, such as wood and stainless steel, but lower than other nonfabric materials, such as cement paint and PVC. Another study by Thatcher *et al*.[38] showed that the particle deposition rate in a carpeted room was about double of the rate in a room with bare surfaces. If a doubled pathogen load on a fabric surface was considered to cater for the particle deposition rate difference, the infection risk for contacting fabric material would be approximately doubled. Contacting fabric materials would result in a higher indirect contact transmission risk toward influenza A virus than contacting nonfabric materials. However, due to the much higher pathogen transfer efficiencies and better survival rates of RSV and rhinovirus on nonfabric materials, infection risks of these two diseases of contacting nonfabric materials were still a few orders of magnitude higher than the infection risk of contacting fabric materials. Therefore, contacting nonfabric materials still resulted in a much higher indirect contact transmission risk in the cases of RSV and rhinovirus.

There are various types and grades of fabric material as well as nonfabric material. Due to limited researches on the topic, complete sets of these influencing parameters for a specific fabric/nonfabric material for all three microorganisms are not available. As found in literature, the microorganism survival on the surface, the transfer rate of microorganism onto hand, and the deposition rate of particle onto the surface have obvious differences between fabric material surface and nonfabric material surface. The role of material surface on microorganism survival, transfer rate of microorganism onto hand, and the deposition rate of particle onto the surface involve complicated mechanisms that are not well understood. In this study, the aim is to point out that fabric material surface and nonfabric material surface, in general, can lead to very different indirect contact transmission risks.

4. LIMITATIONS

The risk assessment model employed in the current study has made several assumptions and simplifications. The assessed risks can show the relative significances between different risk factors as for comparison purpose in the current study, but readers should be aware of the assumptions and

Table VII. Infection Risks of Health-Care Worker Contacting Fabric and Nonfabric Materials (Numbers Inside the Brackets Indicate the Percentage Differences)

Material	Influenza A virus	RSV	Rhinovirus
Fabric material	5.43×10^{-6}	2.43×10^{-11}	2.14×10^{-10}
Nonfabric material	6.19×10^{-6} (+13.9%)	4.43×10^{-8} (+1820.8%)	8.89×10^{-7} (+4154.0%)

simplifications of the current risk assessment model if they want to use it for purposes other than relative comparison. In addition, the cases considered had not covered every possible surface that the occupants might contact. For comparison purpose, the current study also has not considered the complicated stochastic factors in the risk assessment, for example, the distribution of hand contact frequency among the susceptible individuals.

For simplicity and comparison purpose, in the two environments, the occupants were considered touching certain surfaces with a set of assumed contact frequencies. It is possible that the settings did not cover every possible surface that the occupants might contact. When a different surface is considered, it can be considered as a different pathogen load being input into the risk assessment model. Thus, the choice of surfaces contacted by occupants will not affect the percentage differences of the assessed risks when varying the contact frequencies and the surface material, hence the outcome of comparison, in any significant way. The choice of surfaces contacted by occupants, however, may affect the percentage difference of the risks when varying the air change rate, since increase in air exchange rate does not uniformly decrease the pathogen load on all surfaces. The current choice of surfaces contacted by occupants was able to show the relative significances between different risk factors, and to show that increase in air exchange rate does not uniformly decrease exposure and risk.

The current study only considered three common respiratory pathogens that are capable to be transmitted through indirect contact. Other respiratory pathogens that are also capable to be transmitted through indirect contact might have different properties and may show different results.

5. CONCLUSIONS

Quantitative infection risk assessments were conducted on three pathogens and in two different environments to evaluate the significance of different factors in indirect contact transmission of respiratory infection in indoor environment. Generally, the indoor surface material was found to be the most significant influencing factor in the indirect contact transmission risk for at least two pathogens out of the three; human behavior—the frequencies of hand contacts—could also affect the indirect contact transmission risks of the three pathogens significantly; ventilation rate was found to be least significant in indirect contact transmission among these three factors, whereas in some situations increase in ventilation rate was also found to increase the risks of some susceptible persons. Although the air pollutant concentration in a building will decrease when ventilation rate is increased, the air pollutant is not perfectly mixed and the change in ventilation rate in a room will change the airflow pattern as well. Thus, airflow under an increased ventilation rate may transport more pollutant to some regions in a room. If there are occupants in these regions, their exposure and risk to the air pollutant will be higher than that in the lower ventilation rate case. In case of particulate matter, more particles may be transported to some regions and deposited onto surfaces around these regions under a different ventilation rate. This was observed in the experimental and numerical results from the previous studies.[5,26] Therefore, increase in ventilation rate does not necessarily reduce the risks of all routes of transmission. Reducing the hand contact rates on contaminant surfaces and mucous membranes could reduce the infection risk significantly. Therefore, reducing unnecessary hand contacts on surface and on eyes and nose are important personal hygiene practices. Fabric material contributes much lower risk than nonfabric material for RSV and rhinovirus. The risk of indirect contact transmission could be up to three orders of magnitude higher when contacting contaminated nonfabric surface than contacting contaminated fabric surface. However, choice of indoor surface material also affects the indoor concentrations of other pollutants. Therefore, the current results alone do not lead to the conclusion that fabric material should always be used as indoor

surfaces. Also, it should be noted that the findings on the ventilation effect are for indirect contact transmission risk only in the current study. For the overall disease transmission risk, where airborne transmission and other transmission routes shall also be considered, ventilation is definitely playing a more important role as indicated from many studies. Further studies can be focused on evaluating the effect of ventilation rate on the combined infection risk of indirect contact transmissions, airborne transmission, and droplet spray transmission; and the effectiveness of disinfection of inner surfaces[39,40] and the use of antimicrobial coating[41] on the indirect contact transmission of respiratory infection.

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