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Original Article

Respiratory viruses in the patient environment

Linh T. Phan PhD[1], Dagmar M. Sweeney MS[2], Dayana Maita MD[3], Donna C. Moritz MD[3], Susan C. Bleasdale MD[3], Rachael M. Jones PhD[1] and for the CDC Prevention Epicenters Program

[1]Division of Environmental and Occupational Health Sciences, School of Public Health, University of Illinois at Chicago, Chicago, Illinois, [2]Sequencing Core, University of Illinois at Chicago, Chicago, Ilinois and [3]Department of Medicine, College of Medicine, University of Illinois at Chicago, Chicago, Illinois

Abstract

Objective: To characterize the presence and magnitude of viruses in the air and on surfaces in the rooms of hospitalized patients with respiratory viral infections, and to explore the association between care activities and viral contamination.

Design: Prospective observational study.

Setting: Acute-care academic hospital.

Participants: In total, 52 adult patients with a positive respiratory viral infection test within 3 days of observation participated. Healthcare workers (HCWs) were recruited in staff meetings and at the time of patient care, and 23 wore personal air-sampling devices.

Methods: Viruses were measured in the air at a fixed location and in the personal breathing zone of HCWs. Predetermined environmental surfaces were sampled using premoistened Copan swabs at the beginning and at the end of the 3-hour observation period. Preamplification and quantitative real-time PCR methods were used to quantify viral pathogens.

Results: Overall, 43% of stationary and 22% of personal air samples were positive for virus. Positive stationary air samples were associated with ≥5 HCW encounters during the observation period (odds ratio [OR], 5.3; 95% confidence interval [CI], 1.2–37.8). Viruses were frequently detected on all of the surfaces sampled. Virus concentrations on the IV pole hanger and telephone were positively correlated with the number of contacts made by HCWs on those surfaces. The distributions of influenza, rhinoviruses, and other viruses in the environment were similar.

Conclusions: Healthcare workers are at risk of contracting respiratory virus infections when delivering routine care for patients infected with the viruses, and they are at risk of disseminating virus because they touch virus-contaminated fomites.

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Healthcare workers (HCWs) are at risk for contracting viral respiratory infections while providing care to patients with these infectious diseases.[1–3] Environmental surfaces in the patient room frequently touched by both HCWs and patients have been contaminated with respiratory viruses,[4,5] suggesting surfaces could facilitate the transmission of respiratory viruses via the contact route. However, contact patterns have not been observed in conjunction with viral contamination. Respirable influenza virus particles have been detected in the rooms of patients infected with influenza virus and in the breathing zones of HCWs, but the factors that determine the magnitude or presence of these viruses, other than the presence of an infected patient, remain unclear.[6–8] Viral contamination, in conjunction with the behaviors of HCWs and patients, needs to be characterized because these factors influence the frequency, pathways, and magnitude of occupational exposure.

Author for correspondence: Rachael M. Jones, Email: rachael.jones@utah.edu

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The primary objective of this study was to characterize the presence and magnitude of respiratory viruses in the air and on environmental surfaces associated with care for hospitalized patients having acute respiratory viral infections. The secondary objective was to explore the association between observed care activities and viral contamination in the environment. Relative to other studies of HCW exposure to respiratory viruses in healthcare settings, this work is unique because it focuses on the association between viral contamination of the environment and the behaviors of HCWs and patients.

Methods

Study setting and participants

The study was conducted over 2 years from March 2017 to June 2017 and from September 2017 to April 2018 in an acute-care hospital in Chicago, Illinois. Patient and HCW participant recruitment and consent have been described elsewhere.[9] This study was approved by the University of Illinois at Chicago Institutional Review Board (protocol no. 2015-0990). All patient participants had a positive test for a respiratory viral infection within 3 days prior to the day of observation. Researchers performed

observations in patient rooms during a 3-hour period, typically from 8 A.M. to 12 P.M. Patient rooms were cleaned daily with Virex Plus (Diversey, Fort Mill, SC), a quartenary ammonium-chloride–based disinfectant. As a negative control, 5 clean, empty patient rooms in 4 different hospital units were selected at random and environmental surfaces were sampled for viruses on 2 different days (Supplemental Material 1 online).

Subsequent to observation, the medical records of participating patients were reviewed to extract data regarding signs, symptoms, and comorbidities. To get a general sense of illness severity, a disease severity score was tabulated by modifying a method used for community-acquired pneumonia.[10]

Air sampling

Viruses were measured in the air at a fixed location in the patient room, 1 m above the floor within 1.6 m of the patient's head for the duration of the experiment, or until patients requested the sampler be turned off. Viruses were also measured in the breathing zone of HCWs during patient encounters: 1 personal sample was collected per experiment. All air sampling used the National Institute for Occupational Safety and Health (NIOSH) 2-stage cyclone air sampler, which separately collects particles in 3 size ranges:[11] aerodynamic diameter $d_a < 1$ μm; $1 < d_a < 4$ μm; and $d_a > 4$ μm (Supplemental Material 2 online).

Surface sampling

Environmental surfaces were sampled using premoistened Copan swabs (Copan Diagnostics, Murrieta, CA). At the beginning of the experiment, the following surfaces were sampled (if present in the patient room) to reflect baseline contamination: tray table, IV monitor, bed rail, computer keyboard, and computer mouse. At the end of the experiment, the surfaces sampled at baseline were sampled again and the following additional surfaces were sampled (if present): exterior bed control panel, IV pole hanger, telephone, call button/TV remote control, and isolation stethoscope. For surfaces sampled at the baseline and at the end of the experiment, different locations with the same area on that surface were swabbed (Supplemental Material 3 online). Computer stations were only sampled if they were permanently located in the patient room; mobile computer stations used by HCWs during the observations were not sampled. The surfaces were selected for sampling because previous work has identified them to be frequently touched surfaces by HCWs, and 3 of them (eg, computer keyboard and mouse and IV monitor) were only used by HCWs.[12,13] All surfaces sampled were swabbed horizontally and vertically following a protocol (Supplemental Material 3 online).

Sample processing and analysis

Sample processing and analysis methodology is described in detail in Supplemental Material 4 (online). Briefly, after extraction of genetic material from the sampling media, preamplification and quantitative polymerase chain reaction (qPCR) were performed using assays for targeted viruses on triplicates of each sample to obtain the cycle threshold values. A sample was considered positive when, having undergone preamplification, a qPCR Ct value of ≤35 was obtained for at least 2 of the triplicate results. For samples determined to not be positive, the value Ct = 40 was substituted as the limit of detection. Quantitation of each sample was based on a linear regression derived from the 5-point preamplified standard curve for each target, with correction for dilution, preamplification, volume of RNA extraction eluate, volume of reverse transcription, and double-stranded nature of the DNA standard. Gene copies were equated with the gene copy per square centimeter sampled and gene copies per cubic meter of air sampled.

Statistical methods

Data management for this study was described elsewhere.[14] All data analysis was performed with R (The R Project for Statistical Computing, Vienna, Austria). Preliminary analysis indicated that virus concentrations (gene copy per cm^2 and per m^3) were approximately log-normally distributed. Left-censored values were imputed as follows. Log-normal distribution parameters were estimated using the *cenmle* function of the 'NADA' library,[15,16] and values were then selected from the fitted lognormal distribution at randomly selected percentiles below the percentage of censored values.[17] Because the limit of detection varied between samples, randomly selected values were verified to be below the limit of detection, and a value was selected again if it was greater than the limit of detection.

Differences in virus concentrations among ≥3 groups were tested with the Kruskal–Wallis (KW) test, followed by pairwise Wilcoxon (W) tests with *P* values adjusted using the Bonferroni method, while differences between 2 groups were tested with the Mann-Whitney (MW) test. Differences in proportions among groups were tested with the χ^2 test where expected values were determined using the overall mean proportion, followed by pairwise Wilcoxon test for pairwise comparisons. Spearman's method was used for correlation testing. The χ^2 test was used to test for the associations between the presence of virus and categorical predictor variables.

Results

Participant characteristics

We observed healthcare activities for 52 patients with viral respiratory infections: 30 patients were in droplet and contact isolation, 21 in droplet isolation, and 1 was in contact isolation. In total, 11 patients were in intensive care units (ICUs); 18 were in the clinical decision unit; 9 were in specialty units; and 14 were in other general medical surgical units or non-ICUs.[9] Patients were grouped by infection: (1) influenza, including influenza A (n = 23) and influenza B (n = 8); (2) rhinovirus (n = 15); and (3) other (1 parainfluenza, 1 coronavirus, 3 respiratory syncytial virus, and 1 adenovirus). This grouping was selected because the viruses have characteristic environmental persistence.[18] Because some patients had coinfections, patients with the target respiratory infections could be in a variety of isolation categories. All participants with influenza or rhinovirus infections were in droplet and contact or droplet isolation. Adenovirus samples were excluded from sample processing due to the complexity of multiple virus strains.

Moreover, 3 patient participants were on ventilators (2 with rhinovirus and 1 with parainfluenza). In general, patients were not severely ill, with 4 (8%) and 8 (15%) of participants having high and moderate disease severity, respectively. Participants with high disease severity were immunocompromised, and 3 of the 4 had malignancies. Overall, 35% of patient participants were immunocompromised. Cough was recorded in the charts of 50 (77%) of patient participants.

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Airborne virus

Stationary air samplers operated for 55–197 minutes, with a mean duration of 161 minutes. Of the 47 stationary air samples, 43% were positive for virus in any of the 3 size fractions, and the mean virus concentration was 7,300 virus copies/m^3 (Table 1). Virus was present in all 3 size fractions, though virus was present in different size-fractions in different samples. Differences in the proportion of samples positive for virus nor in the virus concentrations among the 3 size fractions were not statistically significant (χ^2 $P = .17$ and Kruskal Wallace [KW] $P = .47$).

When stationary air samples were considered by pathogen group, 11 samples (38%) from the room of a patient with influenza were positive for influenza, 6 samples (43%) were positive for rhinovirus from the rooms of patients with rhinovirus, and 3 samples (75%) were positive from the rooms of patients with other viruses (Table 1). Neither the proportion of positive samples nor the virus concentrations differed among the 3 virus groups (χ^2 $P = .37$; KW $P = .43$). When considered by size fraction, the only difference in the proportion of positive samples was observed in the largest size fraction ($d_a > 4$ μm; χ^2 $P = .01$).

Personal air samplers were operated for 2–41 minutes, with a mean duration of 13 minutes. Of the 23 personal air samples, 22% were positive for virus and had mean concentration of 3×10^6 copies/m^3 (Table 1). Virus was detected only in the 2 smaller size fractions ($d_a < 1$ μm, and $1 < d_a < 4$ μm), but inference about the size distribution of virus in the breathing zone is limited by the small sample size. Virus was recovered from 1–2 samples for each virus group. Virus concentrations of the positive personal air samples ranged from 1.7×10^4 to 6.8×10^7 copies/m^3 and were more variable than concentrations measured by the stationary air sampler.

Personal air samples were more likely to be positive for virus when the stationary air sample was positive than when that stationary air sample was negative, but the odds ratio was not statistically significant (OR, 2.5; 95% CI, 0.3–23.5) (Table 2). Also, the virus concentrations in stationary air samples were not significantly correlated with those in personal air samples ($\rho = -0.13$; $P = .56$). Stationary air samples were more likely to be positive when >5 HCW encounters occurred during the observation period than when ≤5 encounters occurred (OR, 5.3; 95% CI, 1.2–37.8), but this factor could not be tested for personal air samples (Table 2). Cough reported on the day of observation was not associated with the stationary or personal viral concentrations (Table 2). Additional results are in Supplementary Material 5 online.

Surface contamination

Virus presence and concentrations were measured on surfaces at the beginning of the experiment (baseline) and after the experiment (Tables 3 and 4). At baseline, virus was present on the computer keyboard (67%) and bedrail (52%) in most experiments. The proportion of baseline surface samples positive for virus and the distribution of virus concentrations were statistically significantly different among the surfaces sampled (χ^2 $P = .00$ and KW $P = .001$); but the proportion of positive surface samples and in the distribution of virus concentrations at baseline did not differ among the virus groups (Table 3). Every surface swabbed at the end of the experiments tested positive for virus at least once for each virus group, and >50% of the sampled surfaces were positive for virus in 14 of 51 (27%) of the experiments (Table 4). Virus was present on the computer keyboard (68%), the call button (57%), and the computer mouse (50%) in most experiments, and the proportions of positive samples differed significantly among surfaces at the end of the experiments for all viruses (χ^2 $P < .01$), influenza (χ^2 $P < .01$), and rhinovirus (χ^2 $P = .03$). As at baseline, the virus concentrations were statistically significantly different among the surfaces (KW $P < .01$), with very low virus concentrations measured on the IV monitor relative to the other surfaces, particularly for rhinovirus (Table 4).

Contacts with surfaces may deposit or remove virus from the surface, and we found moderate positive correlations ($0.3 \leq \rho \leq 0.6$) between the numbers of contacts by HCWs and virus concentrations for some surfaces (Table 5). Patient contacts were not associated with virus concentrations (Supplemental Material 6 online).

The odds that ≥50% of surfaces sampled at the end of an experiment were positive for virus was significantly higher when the stationary air sample was positive for virus (OR, 15.3; 95% CI, 3.3–112.2) and was higher, but not significantly, when ≥ 10 coughs were observed (OR, 1.45; 95% CI, 0.3–5.7) (Table 2). In 53%–76% of the experiments, depending upon the surface, surfaces sampled at the beginning and end of the experiment were both positive or both negative, and the proportion changing from negative to positive was similar to the proportion changing from positive to negative (Table 6). Virus concentrations measured at baseline and at the end of the experiment were only statistically positively correlated on the computer keyboard ($\rho = 0.99$; $P < .05$) (Supplemental Material 7 online).

Of the 42 surface samples collected across 5 clean, empty patient rooms, only 1 sample was found positive for influenza A, influenza B, or rhinovirus: a telephone swab sample for influenza A. Thus, the virus was not generally present in the hospital environment absent an infected patient.

Discussion

We found respiratory viruses in air in the room near the patient's head in 43% of the experiments, and in 22% of the personal air samples (Table 1). The relatively low detection of virus in the personal air samples is likely due, at least in part, to the short duration of sampling relative to the stationary air samples (10 minutes versus 3 hours). It is typical that virus is not detected in all air samples collected near patients with viral respiratory infections. Leung et al,[6] for example, placed the NIOSH 2-stage cyclone aerosol sampler 1.0 or 1.5 m from the patient in a hospital ward; they detected virus in 50% of study periods with patients having laboratory-confirmed influenza. Using the same device, Lindsley et al[19] reported that 57% of personal samplers worn by HCWs for 4–5 hours in an urgent care clinic and 50% of stationary samplers were positive for influenza or RSV during influenza season. These data affirm that HCWs may inhale respiratory viruses while in the room of infected patients and that virus may disperse through the environment to deposit on surfaces.

Cough is thought to be a primary emission mechanism for respiratory viruses[20] and has been associated with virus concentration measured by stationary air samplers when patients had an increased nasopharyngeal viral load.[8] In this study, cough reported on the day of observation was not associated with virus concentrations measured in stationary or personal air samples (Table 2). However, this finding may be an artifact of the study design because coughs were only recorded during observation periods, which are a subset of the 3-hour sampling duration. Future work could explore use of the automated sound-based cough monitor to measure cough with less disruption of patient privacy.[21]

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Table 1. Virus Measured in the Air at a Stationary Location and in the Personal Breathing Zone of Healthcare Workers in the Rooms of Patients With Acute Respiratory Infection

	All Viruses		Influenza Viruses		Rhinoviruses		Other Viruses		P Value Comparing proportions Among Virus Groups[a]	P Value Comparing virus Concentration Among Virus Groups
Air Samples	Positive Proportion (%)	Mean (75^{th}P, Max) (copies/m^3)	Positive Proportion (%)	Mean (75^{th}P, Max) (copies/m^3)	Positive Proportion (%)	Mean (75^{th}P, Max) (copies/m^3)	Positive Proportion (%)	Mean (75^{th}P, Max) (copies/m^3)		
Stationary location										
$d_a < 1\ \mu m$	6/47 (13)	1.4×10^3 ($0.0; 4.1 \times 10^4$)	3/29 (10)	130 ($0.0; 2.3 \times 10^3$)	2/14 (14)	4.3×10^3 ($0.0; 4.1 \times 10^4$)	1/4(25)	380 ($380; 1.5 \times 10^3$)	.70	.89
$1 < d_a < 4\ \mu m$	12/47 (26)	1.1×10^3 ($9.1; 3.2 \times 10^4$)	9/29 (31)	1.4×10^3 ($33; 3.2 \times 10^4$)	2/14 (14)	2.8×10^3 ($0.0; 2.2 \times 10^4$)	1/4 (25)	73 (73; 290)	.50	.15
$d_a > 4\ \mu m$	13/47 (27.7)	4.2×10^3 ($260; 1.4 \times 10^5$)	5/29 (17)	99.6 ($0; 1.3 \times 10^3$)	6/14 (43)	1.3×10^4 ($5.2 \times 10^3; 1.4 \times 10^5$)	2/4 (50)	560 (1.1×10^3; 1.2×10^3)	**.01**	.45
All stages	20/47 (42.6)	7.3×10^3 ($1.2 \times 10^3; 1.4 \times 10^5$)	11/29 (38)	1.7×10^3 ($500; 3.2 \times 10^4$)	6/14 (43)	2.1×10^4 ($1.8 \times 10^4; 1.4 \times 10^5$)	3/4 (75)	1.0×10^3 ($1.5 \times 10^3, 2.7 \times 10^3$)	.37	.43
P Value for differences among stages[a,b]	.17	.47	.13	.46	.12	.45	.15	.39		
Personal breathing zone										
$d_a < 1\ \mu m$	3/23 (13)	3.0×10^6 ($0; 6.8 \times 10^7$)	1/13 (7)	1.3×10^3 ($0; 1.7 \times 10^4$)	0/6 (0)	2.4×10^{-7} ($4.1 \times 10^{-7}; 9.1 \times 10^{-7}$)	2/4 (50)	1.7×10^7 ($1.7 \times 10^7; 6.8 \times 10^7$)	**.05**	.81
$1 < d_a < 4\ \mu m$	2/23 (8)	4.0×10^3 ($0; 6.5 \times 10^4$)	1/13 (7)	5.0×10^3 ($0; 6.5 \times 10^4$)	1/6 (17)	4.3×10^3 ($0; 2.6 \times 10^4$)	0/4 (0)	1×10^{-4} ($10^{-4}; 4 \times 10^{-4}$)	.64	.42
$d_a > 4\ \mu m$	0/22 (0)	0.1 (0.0; 1.8)	0/13 (0)	0.15 (0.00; 1.83)	0/7 (0)	7.7×10^{-8} ($2.7 \times 10^{-8}; 4.1 \times 10^{-7}$)	0/4 (0)	2.1×10^{-6} ($2.6 \times 10^{-6}; 7.5 \times 10^{-6}$)	. . .[c]	.63
All stages	5/23 (22)	3.0×10^6 ($1; 6.8 \times 10^7$)	2/13 (15)	6.3×10^3 ($0.13; 6.5 \times 10^4$)	1/6 (17)	4.3×10^3 ($0; 2.6 \times 10^4$)	2/4 (50)	1.7×10^7 ($1.7 \times 10^7; 6.8 \times 10^7$)	.32	.30
P Value for differences among stages[a,b]	.22	.45	.59	.44	.34	.42	.09	.39		

Note. d_a, aerodynamic diameter. Bold indicates statistical significance.
[a]χ^2 test was used to compare differences in proportions among groups.
[b]Kruskal-Wallis test was used to compare virus concentration differences among groups.
[c]No proportion comparison test was conducted as all samples of stage 3 were negative across pathogen groups.

Table 2. Possible Determinants of the Presence of Virus in Air and Surfaces

	Stationary Air Sample Positive for Virus			Personal Air Sample Positive for Virus			≥50% of Sampled Surfaces Positive for Virus		
Characteristics	Yes	No	OR (95% CI)[a]	Yes	No	OR (95% CI)[a]	Yes	No	OR (95% CI)[a]
Cough on the day of observation									
Yes	13	22	0.42 (0.1–1.6)	3	14	0.4 (0.1–4.1)			
No	7	5		2	1				
Total observed coughs									
≥10 coughs	4	7	0.7 (0.2–2.8)	2	4	2.3 (0.2–19.8)	4	8	1.45 (0.3–5.7)
<10 coughs	16	20		3	14		10	29	
Respiratory treatment									
Yes	4	7	0.7 (0.2–2.8)	3	4	5.3 (0.7–52.5)			
No	16	20		2	14				
Distance from stationary air sampling location to patient's head									
<80 cm	9	11	1.2 (0.4–3.9)						
≥80 cm	11	16							
Total HCW visits, including observed encounters and short interactions									
>5 encounters	18	17	**5.3 (1.2–37.8)**	5	16	NA	14	24	NA
≤5 encounters	2	10		0	2		0	13	
Personal air sample positive for virus									
Yes	3	2	2.5 (0.3–23.5)						
No	6	10							
Stationary Sample positive for virus									
Yes							11	9	15.3 (3.3–112.2)
No							2	25	

Note. OR, odd ratio; CI confidence interval; HCW, healthcare worker.
[a]The χ^2 test was used to test for the significance of the odds ratio

Table 3. Virus on Environmental Surfaces Measured in the Room of Patients with Acute Respiratory Infection at Baseline

	All Viruses		Influenza Viruses		Rhinovirus		Other Viruses		*P* Value Comparing Proportions Among Virus Groups[a]	*P* Value Comparing virus Concentration Among Virus Groups[b]
Surfaces	Positive Proportion (%)	Mean (75^{th}, Max) (copies/m^2)	Positive Proportion (%)	Mean (75^{th}, Max) (copies/m^2)	Positive Proportion (%)	Mean (75^{th}, Max) (copies/m^2)	Positive Proportion (%)	Mean (75^{th}, Max) (copies/m^2)		
Bedrail	25/48 (52)	2.4×10^3 (64.1; 1×10^5)	14/29 (48)	140 (6.4; 1.2×10^3)	8/15 (53)	7.1×10^3 (610; 1×10^5)	1/4 (25)	1.1×10^3 (1.2×10^3; 4.4×10^3)	.60	.26
IV monitor	5/37[a] (18)	1.8 (0.0; 36)	2/23 (8)	1.1 (0.0; 25)	3/11 (27)	3.8 (0.0; 36)	0/3 (0)	5.2×10^{-7} (7.9×10^{-7}; 1.6×10^{-6})	.26	.22
Keyboard	12/18[a] (67)	7.1×10^3 (17; 1.1×10^5)	7/12 (58)	160 (4.6; 1.3×10^3)	3/4 (75)	2.9×10^4 (2.9×10^4; 1.1×10^5)	2/2 (100)	3.9×10^3 (5.8×10^3; 7.7×10^3)	.47	.17
Mouse	6/17 (35)	2.1×10^3 (0.6; 3.1×10^4)	3/11 (27)	2.8×10^3 (0.3; 3.1×10^4)	3/4 (75)	1.3×10^3 (1.3×10^3; 5.1×10^3)	0/2 (0)	3.6×10^{-3} (2.4×10^{-3}; 3.1×10^{-3})	.12	.27
Tray table	19/48 (39)	620 (3.5; 2.4×10^4)	8/29 (27)	850 (0.1; 2.4×10^4)	9/15 (60)	360 (140; 4.0×10^3)	2/4 (50)	3.3 (5.0; 10)	.10	.21
P value for differences among surfaces[a,b]	**.00**	**.001**	**.00**	**.03**	.31	.10	.13	.13		

Note. Bold indicates statistical significance.
[a]χ^2 test was used to compare differences in proportions among groups;
[b]Kruskal-Wallis test was used to compare virus concentration differences among groups;

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Table 4. Virus on Environmental Surfaces Measured in the Room of Patients with Acute Respiratory Infection After the Observation Period

Surfaces	All Viruses		Influenza Viruses		Rhinoviruses		Other Viruses		*P* Value Comparing Proportions Among Virus Groups[a]	*P* Value Comparing Virus Concentration Among Virus Groups[b]
	Positive Proportion (%)	Mean (75^{th}, Max) (copies/m^2)	Positive Proportion (%)	Mean (75^{th}, Max) (copies/m^2)	Positive Proportion (%)	Mean (75^{th}, Max) (copies/m^2)	Positive Proportion (%)	Mean (75^{th}, Max) (copies/m^2)		
Bedrail	24/51 (47)	2.0×10^3 (170; 6.7×10^4)	12/29 (41)	760 (6.1; 1.5×10^4)	9/14 (64)	5.2×10^3 (410; 6.7×10^4)	3/5 (60)	690 (180; 3.2×10^3)	.33	.31
IV monitor	9/39(23)	0.9[b] (0.0; 16)	7/25 (28)	1.1 (0.03; 15.7)	1/12 (8)	0.7 (0.0; 8.5)	1/4 (25)	0.5 (0.5; 1.9)	.39	.36
Keyboard	15/22 (68)	2.6×10^{3b} (6.8; 5.6×10^4)	10/15 (67)	51 (1.3; 560)	3/5 (60)	1.1×10^4 (13; 5.6×10^4)	2/2 (100)	130 (190; 240)	.58	.26
Mouse	11/22 (50)	6.6×10^3 (64.5; 1.4×10^5)	7/15 (47)	30 (32; 170)	2/5 (40)	290 (36; 1.4×10^3)	2/2 (100)	7.1×10^4 (1.0×10^5; 1.4×10^5)	.32	.15
Tray table	23/49 (47)	4.7×10^3 (3.0; 1.7×10^5)	14/30 (47)	41 (0.9; 1.0×10^3)	6/15 (40)	1.5×10^4 (12; 1.7×10^5)	3/4 (75)	720 (780; 2.7×10^3)	.45	.11
Call button	26/46 (57)	9.0×10^3 (99; 2.9×10^5)	17/30 (57)	2.2×10^3 (4.5; 6.4×10^4)	6/13 (46)	2.5×10^4 (160; 2.9×10^5)	3/3 (100)	6.9×10^3 (1.0×10^4; 1.9×10^4)	.24	.09
Telephone	19/44 (43)	750 (9.3; 1.3×10^5)	9/29 (31)	62 (1.8; 1.5×10^3)	8/13 (61)	1.3×10^3 (440; 1.1×10^4)	2/2 (100)	6.9×10^3 (1.0×10^4; 1.3×10^4)	**.05**	.07
IV pole hanger	12/48 (25)	5.1×10^5 (0.0; 2.5×10^7)	7/30 (23)	8.2×10^5 (0; 2.5×10^7)	1/13 (7)	1.9×10^{-4a} (6.8×10^{-5}; 1.7×10^{-3})	4/5 (80)	430[a] (13; 2.0×10^3)	**.00**	**.01**
Isolation stethoscope	9/33 (27)	10 (0.1; 220)	5/20 (25)	0.9 (0.0; 8.2)	3/11 (27)	25 (4.8; 220)	1/2 (50)	21 (32; 43)	.75	.46
Exterior bed control	12/51 (23)	27 (0.00; 810)	4/31 (13)	0.6 (0.0; 18)	5/15 (33)	33 (5.5; 270)	3/5 (60)	170 (29; 810)	**.04**	.05
P value for surface differences	**.00**	**.00**	**.00**	**.01**	**.03**	**.04**	.44	.17		

Note. Bold indicates statistical significance.
[a]Significant pairwise comparison at the significant level of $\alpha = 0.05/3 = 0.017$;
[b]Significant pairwise comparison at the significant level of $\alpha = 0.05/45 = 0.001$

Table 5. Correlation Between Virus Concentration on Surfaces After the Observation Period and Contact Frequency of Healthcare Workers During Observed Care Activities

No. of Contacts by HCWs on Surface	Spearman's Correlation Coefficient (*P* Value)									
	Virus Concentration on Surface									
	Tray Table	IV Monitor	Mouse	Keyboard	Bed Rail	Call Button	Telephone	IV Pole Hanger	Isolation Stetho-scope	Exterior Bed Control
Tray table	−0.1 (.46)									
IV pole		0.1 (.68)						**0.5 (.00)**		
Computer station			−0.1 (.56)	−0.2 (.35)						
Bed rail					0.1 (.60)					
Call button						−0.1 (.50)				
Telephone							**0.6 (.00)**			
Isolation stethoscope									0.3 (.15)	
Other bed surface										**0.3 (.02)**
Patient	−0.1 (.54)	0.3 (.09)	−0.2 (.39)	−0.1 (.51)	−0.1 (.64)	0.0 (.91)	**0.4 (.00)**	**0.3 (.05)**	0.2 (.40)	**0.4 (.00)**
All fomites	−0.1 (.58)	0.1 (.07)	−0.2 (.37)	−0.2 (.28)	−0.1 (.58)	−0.1 (.67)	0.2 (.12)	**0.5 (.00)**	−0.1 (.57)	0.2 (.12)

Note. Bold indicates statistical significance.

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Table 6. Change in Virus Presence Status From Baseline to After the Experiment Observation Period

	Surface, Proportion (%)				
Virus Presence Baseline:After	Tray Table	IV Monitor	Mouse	Keyboard	Bed Rail
No change[a]	27/48 (56)	28/37 (76)	9/17 (53)	12/18 (67)	32/48 (67)
Absent:present	12/48 (25)	6/37 (16)	5/17 (29)	4/18 (22)	7/48 (15)
Present:absent	9/48 (19)	3/37 (8)	3/17 (18)	2/18 (11)	9/48 (18)

[a]Virus was present-present or absent-absent

Aerosol-generating procedures (AGPs) are medical procedures thought to generate respiratory aerosols and that have been associated with increased risk of occupationally acquired infection among healthcare personnel,[22] but some have argued that infectious aerosols are generated by routine care.[23] Respiratory treatment is thought to be an AGP, and when performed with delivery of nebulized medication, respiratory treatment has been found to generate aerosols.[24] Although the sample numbers were limited, in this study respiratory treatment was not associated with increased odds of virus detection in stationary air samples (OR, 0.7; 95% CI, 0.2–2.8). However, >5 visits by HCWs during the 3-hour observation period was associated with virus detection in stationary air samples (OR, 5.3; 95% CI, 1.2–37.8), though most observed care activities were physical exam and other routine care activities.[9] Numerous HCW visits may indicate that the patient is relatively ill and, thus, shedding a lot of virus in the environment, but the association of numerous HCW visits with airborne virus could also reflect the fact that routine care activities introduce virus into the air.

Influenza virus, rhinovirus, and other viruses were detected on all of the environmental surfaces sampled (Tables 3 and 5) at frequencies similar to those reported in the rooms of patients with Middle East respiratory syndrome.[25] Surfaces contacted primarily by HCWs, rather than patients, may become contaminated with viruses through deposition from air or transfer of virus on the hands or gloves of HCW or other contacts, which is consistent with the association between positive stationary air samples and the detection of virus on most surfaces (OR, 15.3; 95% CI, 3.3–112.2). Consistent with the role of HCW contacts, we found positive correlations between virus concentration on some surfaces frequently touched by HCWs and the number of contacts, including the IV pole hanger, telephone, and exterior bed control (Table 6). We did not find any statistically significant associations between patient contacts and virus concentrations on surfaces frequently contacted by patients, but this could be due to the fact that patient contacts were only observed during HCW encounters, not for the 3-hour duration of the observation. This study is the first in which the association between contact behaviors of HCWs, patients, and the magnitude of viral contamination on environmental surfaces in the patient rooms have been explored.

This study has several limitations. Our analytical methods quantified the amount of virus target genes and did not assay viable viruses, and the average ratio between virus target genes and viable viruses for the assays used is unknown, limiting inference from these data for infection risk. Also, the efficiency of the sampling devices has not been characterized and is expected to be less than unity.[26] Thus, virus may be present more often and at higher concentrations than reported in this study. Furthermore, we only observed patient activities during HCW encounters, which likely resulted in an underestimation of the numbers of coughs and surface contacts. We tested only for crude associations, rather than using regression modeling, owing in part due to limited sample sizes, which means that we may have missed some complex relationship among predictor variables that influence virus contamination in the environment. Finally, we did not follow HCWs for outcomes related to virus exposure; therefore, the risk of respiratory infection among the HCWs can only be estimated.

The data obtained in this study suggest that influenza, rhinovirus, and other viruses disperse similarly into the air and onto surfaces of the patient room and that HCWs encounter these viruses in the air and on surfaces during patient care activities.

Supplementary material. To view supplementary material for this article, please visit https://doi.org/10.1017/ice.2019.299

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