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Major Article

Estimating the effect of hand hygiene compliance and surface cleaning timing on infection risk reductions with a mathematical modeling approach

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Key Words:
QMRA
Intervention efficacy
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Background: Quantitative tools are needed to relate infection control interventions to infection risk reductions.

Methods: A model for predicting virus concentrations on hands was used to predict rotavirus, rhinovirus, and influenza A virus doses. Variability in behaviors, transfer efficiencies for various contact types, and surface areas of contact were included. Dose-response curves were used to relate estimated doses to infection risks. Percent reductions from baseline in average rotavirus, rhinovirus, and influenza A virus dose and infection risk were calculated for interventions.

Results: Baseline average infection risks for rotavirus, rhinovirus, and influenza A virus were 0.43, 0.20, and 5.51×10^{-6}, respectively. One and 2 cleaning events decreased average viral infection risks by 6.98%-17.06% and 13.95%-34.66%, respectively. A 15% increase in hand compliance decreased average infection risks by 6.98%-20.51%. A 15% increase in hand hygiene compliance paired with 2 cleaning events decreased average infection risks by 20.93%-47.55%.

Discussion: This study demonstrates the infection risk benefits of combined interventions.

Conclusions: Models such as the one in this study could be used to optimize timing and frequency of cleaning events and to create hand hygiene compliance goals to achieve infection risk targets.

It is well known that fomites play a large role in nosocomial disease transmission, and once contaminated, survival of pathogens on fomites may be on the order of months.[1–6] Influenza has been shown to remain viable on stainless steel surfaces for as long as 2 weeks, whereas remaining viable on porous surfaces for 1 week.[7] Understanding how contacts with contaminated surfaces influence spread and infection risk for health care workers and patients has been explored with bacteriophage tracer studies and several quantitative microbial risk assessment (QMRA) studies.[8–13] However, the influence of timing on cleaning and hand hygiene behavior efficacy is relatively unexplored within the context of reducing viral exposures in health care settings. Some models that have explored interventions in health care settings included parameters related to health care worker behavior and compared efficacy in exposure reduction between hand hygiene compliance and surface cleaning.[14,15] However, of the mathematical models that exist within a health care context, few compare predicted health outcomes to independent data.[16]

Previous studies modeling viral exposures and infection risk have assumed a single viral concentration value on hands for the whole simulated exposure period. This originates from the assumption that the microbial concentration on hands reaches steady-state concentration, because exposure durations are often long in comparison to the rates of viral losses from the hands.[13,17] However, these steady-state models do not address the second-by-second peaks in exposure that occur as a result of contacts with highly contaminated surfaces or contacts that result in a larger quantity of virus transferred from the surface or object to the hand. We developed and validated a model to incorporate individual behavioral events and second-by-second viral exposures in health care settings.[18] A limit to this previous model is the overestimation of predicted viral doses and risk due to seed concentrations of bacteriophage tracers at levels higher than routinely expected in a health care environment.[18] High concentrations of bacteriophage are typically used so that the bacteriophage are easily detected on surfaces, avoiding data below limits of detection. The fraction of samples below a detection limit is therefore not well

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reflected by bacteriophage tracer studies in comparison to experimental data. For example, in a study conducted by Ganime et al,[19] 14% (73 of 504) of samples in intensive care units had detectable rotavirus, and of those samples, 61.6% (45 of 73) had concentrations ranging from 3.43-2.94 $\times 10^3$ genome copies/mL. In a bacteriophage tracer study conducted in an urgent care setting, 56.5% (13 of 23) surface samples were positive for the bacteriophage, and positive samples had concentrations ranging from 2.70-3.445 $\times 10^3$ plaque forming units (PFU)/cm^2.[12] Few data exist characterizing the distribution of viral concentrations in health care environments. Survey studies conducted in health care facilities often report low proportions of virus detection in the environment, making the variability of exposure intensities uncertain.[19–21] However, a mixed distribution approach, or the use of multiple distributions, has been used to estimate virus concentrations in the environment, and has been recognized as a method to account for "left-censored" microbial data, or data below detection limits.[17,22] Applying a realistic distribution of virus concentrations to existing models based on bacteriophage tracer data would yield more meaningful exposure and associated risk predictions.

In addition to lacking information regarding viral surface concentrations in health care environments, few studies have addressed the timing of recontamination of surfaces postcleaning, especially specific to viruses.[23] Understanding how patterns of recontamination affect infection risk and intervention efficacy will inform future efforts to define and maintain cleanliness standards. Modeling is a useful method to explore how recontamination may occur and affect risk and to inform future environmental microbiological studies that can validate, disprove, or inform these assumptions.

The purpose of this study was to use a viral exposure model to account for a distribution of expected virus concentrations in health care environments and the timing of surface cleaning events. This model was then used to predict rotavirus, rhinovirus, and influenza A virus infection risks and to evaluate the effect of increased hand hygiene compliance, single and multiple cleaning events, and combined interventions on risk reductions.

METHODS

Model framework

A simulation model estimating second-by-second changes in viral concentration on hands was used in this study. More information regarding model structure and parameters are provided by Wilson et al.[18] Adjustments to the model made in this study included incorporating a distribution to reflect the variability of virus concentrations on health care surfaces,[17] modifications for timed cleaning events, calculating dose and infection risk with dose-response curves, and changing hand hygiene compliance rates to predict infection risk reductions.

Distribution for virus concentrations on surfaces

Distributions for virus concentrations on surfaces were simulated with methods used in a previous QMRA study.[17] A mixed distribution was used, in which surface concentrations assumed to be below a limit of detection were represented using a uniform distribution, and surface concentrations above the limit of detection were represented using an exponential distribution.[17] Assuming a uniform distribution for samples below a limit of detection has been used in microbial and environmental data contexts.[22,24] The limit of detection and the parameters for these distributions were informed by rotavirus surface concentrations reported by Ganime et al.[19] This distribution was used to simulate rotavirus, rhinovirus, and influenza A virus concentrations on surfaces and was randomly sampled for each second of simulated time. An acknowledged limitation to this approach is that only rotavirus concentrations were used to inform simulated viral concentrations on surfaces. Using the same viral surface concentrations for all 3 viruses meant that observed differences in estimated doses and infection risks were functions of simulated human behaviors and dose-response relationships.

Modeling hand hygiene and surface cleaning interventions

Modeled interventions included a single cleaning event modeled 1 hour after simulation start time, 2 cleaning events modeled 1 and 3 hours after simulation start time, and a change in hand hygiene compliance (from 36%-51%), a compliance change observed during an intervention conducted in US nonintensive care units.[25] This hand hygiene compliance change was modeled by increasing a baseline hand hygiene compliance (36%) to 51% for each simulated individual.

During a simulated cleaning event, the sampled surface concentration for that simulated moment was reduced by 99.99%. It is acknowledged that reductions of this magnitude are often not achieved in real-world settings, especially for porous surfaces. However, it was estimated that at least a 99.99% reduction on surfaces would be needed to meet risk targets for rotavirus and rhinovirus for a 6-hour exposure time, and in other QMRA studies, reductions on surfaces have been treated the same for porous and nonporous surfaces.[17] It was then assumed that surfaces became recontaminated linearly over time. To our knowledge, data to inform expected surface recontamination patterns specific to our model scenarios were unavailable. Surfaces were recontaminated at 2 hours postcleaning in the simulation. This recontamination time was chosen based on a bacteriophage study conducted in an urgent care facility in which surfaces reached a contamination level fairly constant throughout the duration of the study within 2 hours after seeding.[12,18] In the model, viral concentrations on surfaces were randomly sampled for each simulated moment. For surface cleaning event times, viral concentrations were altered to reflect initial viral reduction and subsequent recontamination. A term named "$Contamination_t$" was used to describe the fraction of the baseline virus concentration removed for time t after a cleaning event. The following equation describes the function of recontamination over time after simulated cleaning events:

$$Contamination_t = \frac{-reduction}{T_r} t + reduction$$

$$C_t = B_t(1 - Contamination_t)$$

where $Contamination_t$ = fraction of baseline concentration missing since cleaning event, reduction = initial viral reduction on surface from cleaning event, T_r = recontamination time (seconds), t = time postcleaning event (seconds), B_t = baseline viral concentration on surface at time t postcleaning event (PFU/cm^2), C_t = current viral concentration on surface at time t postcleaning event (PFU/cm^2)

Calculating cumulative doses and infection risk

Doses were summed for the 6 hours of simulated activity. Doses for rotavirus occurred as a result of hand-to-mouth exposures.[26] Doses for rhinovirus were a result of hand-to-nose and hand-to-eyes contacts.[27] Doses for influenza A virus were a result of hand-to-nose, hand-to-eyes, and hand-to-mouth contacts.[28] Infection risks for each simulated dose were estimated using beta-Poisson dose-response curves.[29] This dose-response curve form was used because it is the recommended dose-response curve for rotavirus, rhinovirus, and influenza A virus.[29–33]

Calculating percent reductions in average infection risk and cumulative dose estimates

For each intervention, the following equation was used to calculate percent infection risk reductions for average estimated doses and infection risks:

$$\%\,reduction = \frac{intervention - baseline}{baseline} \cdot 100\%$$

Sensitivity analysis

Spearman correlation coefficients calculated with primary model stochastic parameters that assumed baseline hand hygiene compliance and no cleaning events were used to evaluate the influence of model inputs on estimated cumulative dose. All stochastic parameters included in this portion of the sensitivity analysis were included in all primary and sensitivity analysis models. The absolute value of Spearman correlation coefficients were ranked, in which a lower rank corresponded to a larger absolute value Spearman correlation coefficient, indicating a larger effect on estimated cumulative dose. This sensitivity analysis method has been used in other QMRA studies.[18,34]

The effect of other modeling decisions, recontamination time, and baseline hand hygiene compliance on estimated cumulative dose were evaluated. However, their impacts on infection risk were not evaluated because of the effect of the dose-response curve on any differences between sensitivity and primary models. Aside from the 2-hour recontamination time assumed in the primary model, secondary sensitivity analysis models 1 and 2 assumed recontamination times of 4 hours and 6 hours, respectively. Secondary sensitivity analysis models 3 and 4 assumed baseline compliance rates of 50% (intervention compliance: 65%) and 75% (intervention compliance: 90%), respectively. Estimated infection risk reductions were then compared to evaluate the effect of assumed baseline hand hygiene compliance and assumed recontamination times.

RESULTS

Primary model results

On average, estimated rotavirus, rhinovirus, and influenza A virus cumulative doses were 3.74, 0.17, and 3.90 viral particles, respectively, in which no intervention was simulated (baseline) (Table 1). The largest estimated doses were observed for the baseline scenario, whereas the smallest estimated doses were observed for the scenario involving a 15% increase in hand hygiene compliance combined with 2 cleaning events. The average estimated rotavirus, rhinovirus, and influenza A virus cumulative doses for this scenario were 1.96, 0.089, and 2.05 viral particles, respectively.

Baseline average rotavirus, rhinovirus, and influenza A infection risks were 0.43, 0.20, and 5.51×10^{-6}, respectively (Table 1). Increasing hand hygiene from 36%-51% reduced average rotavirus, rhinovirus, and influenza A virus infection risks by 6.98%-20.51% (Table 2). The greatest reduction as a result of increased hand hygiene compliance was observed for influenza A virus, with a 20.51% risk reduction. A single cleaning event resulted in a similar range of risk reductions, with average rotavirus, rhinovirus, and influenza A virus infection risks being reduced by 6.98%-17.06% (Table 2). Once again, the greatest infection risk reduction, 17.06%, was observed for influenza A virus. Two surface cleanings resulted in reductions in viral risk ranging from 13.95%-34.66% (Table 2). A 15% increase in hand hygiene compliance paired with a single cleaning and with 2 cleanings reduced average infection risks by 13.95%-34.12% and 20.93%-47.55%, respectively.

Table 1
Primary model estimated cumulative doses (viral particles) and infection risks

	Cumulative dose									
	Baseline		15% HH increase		1 Cleaning		2 Cleanings		15% HH increase + 1 cleaning		15% HH increase + 2 cleanings	
	Mean (SD)	**(Min, Max)**	Mean (SD)	(Min, Max)	Mean (SD)	(Min, Max)	Mean (SD)	(Min, Max)	Mean (SD)	(Min, Max)	Mean (SD)	(Min, Max)
Rotavirus	**3.74 (1.38)**	**(0.82, 8.94)**	2.96 (1.15)	(0.69, 8.10)	3.10 (1.22)	(0.63, 8.46)	2.44 (0.99)	(0.42, 6.82)	2.46 (1.02)	(0.43, 7.13)	1.96 (0.84)	(0.40 5.71)
Rhinovirus	**0.17 (0.10)**	**(0.01, 0.71)**	0.14 (0.090)	(0.0028, 0.72)	0.14 (0.090)	(0.0068, 0.67)	0.11 (0.075)	(0.0044, 0.65)	0.11 (0.077)	(0.0014, 0.55)	0.089 (0.063)	(0.0011, 0.46)
Influenza A virus	**3.90 (1.41)**	**(0.88, 9.16)**	3.10 (1.17)	(0.75, 8.22)	3.24 (1.24)	(0.70, 8.62)	2.55 (1.014)	(0.46, 6.94)	2.57 (1.04)	(0.45, 7.16)	2.05 (0.86)	(0.42, 5.91)
	Infection risk											
	Baseline		15% HH increase		1 Cleaning		2 Cleanings		15% HH increase + 1 cleaning		15% HH increase + 2 cleanings	
Rotavirus	**0.43 (0.050)**	**(0.24, 0.54)**	0.40 (0.052)	(0.22, 0.53)	0.40 (0.053)	(0.20, 0.54)	0.37 (0.056)	(0.16, 0.51)	0.37 (0.056)	(0.16, 0.52)	0.34 (0.058)	(0.15, 0.49)
Rhinovirus	**0.20 (0.067)**	**(0.027, 0.39)**	0.18 (0.066)	(0.0073, 0.39)	0.18 (0.066)	(0.017, 0.39)	0.16 (0.063)	(0.011, 0.38)	0.16 (0.064)	(0.0036, 0.36)	0.14 (0.061)	(0.0031, 0.34)
Influenza A virus	$\mathbf{5.51 \times 10^{-6}}$ $\mathbf{(2.00 \times 10^{-6})}$	$\mathbf{(1.25 \times 10^{-6}}$, $\mathbf{1.29 \times 10^{-5})}$	4.38×10^{-6} (1.65×10^{-6})	$(1.06 \times 10^{-6}$, $1.16 \times 10^{-5})$	4.57×10^{-6} (1.75×10^{-6})	$(9.84 \times 10^{-7}$, 1.22×10^{-5}	3.60×10^{-6} (1.43×10^{-6})	$(6.48 \times 10^{-7}$, $9.80 \times 10^{-6})$	3.63×10^{-6} (1.47×10^{-6})	$(6.38 \times 10^{-7}$, $1.012 \times 10^{-5})$	2.89×10^{-6} (1.21×10^{-6})	$(5.90 \times 10^{-7}$, $8.34 \times 10^{-6})$

HH, hand hygiene; *Max*, maximum; *Min*, minimum; *SD*, standard deviation.
Bolded values indicate baseline values to which intervention values can be compared.

Table 2
Percent reduction in average estimated cumulative doses and infection risk

	% Reduction in average estimated cumulative dose				
	15% Increase in HH	1 Cleaning	2 Cleanings	15% Increase in HH + 1 cleaning	15% Increase in HH + 2 cleanings
Rotavirus	20.86	17.11	34.76	34.22	47.59
Rhinovirus	17.65	17.65	35.29	35.29	47.65
Influenza	20.51	16.92	34.62	34.10	47.44
	% Reduction in average estimated infection risk				
	15% Increase in HH	1 Cleaning	2 Cleanings	15% Increase in HH + 1 cleaning	15% Increase in HH + 2 cleanings
Rotavirus	6.98	6.98	13.95	13.95	20.93
Rhinovirus	10.00	10.00	20.00	20.00	30.00
Influenza	20.51	17.06	34.66	34.12	47.55

HH, hand hygiene.

Stochastic parameter Spearman correlation coefficients and ranking

The 5 most influential parameters on estimated rotavirus dose, in order of most to least influential, were number of mouth contacts, number of handwashes, mouth transfer efficiency, surface concentration, and number of nonporous surface contacts (Table 3). Estimated rotavirus dose had a negative linear relationship with number of handwashes, indicating that an increase in handwashing would relate to a decrease in dose. Estimated rotavirus dose had positive linear relationships with the number of mouth contacts, mouth transfer efficiency, surface concentration, and number of nonporous surface contacts, indicating that these contact types would relate to an increase in dose. These 5 parameters were also the most influential for estimated influenza A virus doses (Table 3). For estimated rhinovirus doses, the 5 most influential parameters, in order of most to least influential, were the number of nose contacts, number of eye contacts, number of handwashes, fractional surface area of eye contact, and number of porous surface contacts (Table 3).

Hand-to-nose contacts were infrequent in simulated behaviors, and not all simulated persons had hand-to-nose contacts. Although the number of hand-to-nose contacts could be included in the Spearman correlation coefficient calculations as a zero value, a fractional surface area of zero would be a meaningless placeholder that would capture the effect of frequency of hand-to-nose contacts in addition to differences in transfer efficiency. For this reason, fractional surface areas for hand-to-nose contacts were not included.

Comparison of primary model and sensitivity analyses model estimated percent reductions in average cumulative doses

Although the primary model that assumed a baseline hand hygiene compliance rate of 36% resulted in estimated percent reductions in average cumulative dose by 17.65%-20.86%, assuming a baseline hand hygiene compliance rate of 51% resulted in percent reductions in average estimated cumulative doses ranging from 14.29%-16.22%. A 15% increase in hand hygiene compliance with a baseline of 51% was slightly less effective at reducing overall doses than from a baseline of 36% hand hygiene compliance. This trend was consistent when baseline hand hygiene compliance rate was 75%, in which percent reductions in average estimated cumulative dose were even smaller (9.00%-15.25%) (Table 4).

As the assumed recontamination time of surfaces increased, the estimated reductions in cumulative dose increased (Table 4). Assuming a 4-hour recontamination time in comparison to a 2-hour recontamination time only very slightly increased reductions in estimated cumulative dose. However, the model with an assumed 6-hour recontamination time estimated percent reductions in average cumulative dose notably larger than those estimated with an assumed 2- or 4-hour recontamination time.

DISCUSSION

This study demonstrates that a 15% increase in hand hygiene compliance for each individual may have a slightly greater effect on

Table 3
Stochastic parameter Spearman correlation coefficients for rotavirus, rhinovirus, and influenza A virus doses and sensitivity analysis ranking

		Rotavirus		Rhinovirus		Influenza A virus	
Parameter	Units	Spearman correlation coefficient	Rank	Spearman correlation coefficient	Rank	Spearman correlation coefficient	Rank
Surface concentration	Viral particles/cm^2	0.13	4	0.057	6	0.13	4
Nonporous transfer efficiency	Fraction	0.0035	12	0.035	10	0.0036	14
Porous transfer efficiency	Fraction	0.062	8	0.033	12	0.064	9
Mouth transfer efficiency	Fraction	0.15	3	0.020	15	0.15	3
Total hand surface area	cm^2	0.071	7	0.056	7	0.074	7
Fractional surface area of mouth contact	Fraction	−0.016	11	−0.027	13	−0.017	12
Fractional surface area of surface contact	Fraction	0.048	10	0.039	9	0.050	10
Fractional surface area of eye contact	Fraction	−0.00076	14	0.13	4	0.0061	13
Handwashing efficacy	Log_{10}	0.00070	15	−0.054	8	−0.0010	15
Number of mouth contacts	Number of contacts/6 hours	0.40	1	0.021	14	0.39	1
Number of eyes contacts	Number of contacts/6 hours	0.058	9	0.29	2	0.072	8
Number of nose contacts	Number of contacts/6 hours	−0.0029	13	0.46	1	0.029	11
Number of handwashes	Number of handwashes/6 hours	−0.34	2	−0.27	3	−0.35	2
Number of nonporous surface contacts	Number of contacts/6 hours	0.11	5	0.035	11	0.11	5
Number of porous surface contacts	Number of contacts/6 hours	0.081	6	0.079	5	0.083	6

Table 4
Sensitivity analysis on the effects of assumed baseline hand hygiene compliance rates and surface recontamination time on percent reductions in average estimated cumulative dose

	Percent reduction in average estimated cumulative dose					
	15% Increase in HH with 36% baseline (primary model)	15% Increase in HH from 51% baseline	15% Increase in HH from 75% baseline	**1 cleaning with 2-hour recontamination time (primary model)**	1 cleaning with 4-hour recontamination time	1 cleaning with 6-hour recontamination time
Rotavirus	**20.86**	16.22	15.11	**17.11**	20.97	39.35
Rhinovirus	**17.65**	14.29	9.00	**17.65**	21.43	40.00
Influenza	**20.51**	16.13	15.25	**16.92**	20.99	39.51

HH, hand hygiene.
Bolded values indicate baseline values to which intervention values can be compared.

infection risk than a single cleaning event for this exposure scenario. However, it is acknowledged that the effect of a 15% increase in hand hygiene compliance on infection risks may be dependent on the assumed baseline hand hygiene compliance rate, and that, as demonstrated by the sensitivity analysis, the effect of increased hand hygiene compliance on infection risks may decrease as baseline hand hygiene compliance increases (Tables 3 and 4). The diminishing returns of increased hand hygiene compliance as baseline compliance increases has been noted in other studies.[35]

This model also suggests that 2 cleaning events may yield an even greater risk reduction than a 15% increase in hand hygiene compliance alone, supporting the inclusion of frequent cleaning events in infection control practices, in addition to supporting hand hygiene compliance (Table 2). However, the assumed efficacy of cleaning products may be higher than what is typically achieved in real-world health care environments, especially for porous surfaces, potentially resulting in overestimated risk reductions for cleaning events.

Surface cleaning paired with hand hygiene compliance can provide additional reductions in infection risk, as demonstrated by the 15% hand hygiene compliance paired with a 2 cleaning event scenario in this study that reduced average estimated infection risks by 20.93%-47.55% (Table 2). However, it is also possible that because this model does not account for the effect of hand hygiene compliance on the overall concentrations on health care surfaces, the total effect of hand hygiene compliance on infection risk is not captured. Even when gloves are worn, health care worker hands can still become contaminated. Bingham et al[36] found that 17.4% of gloved health care workers who had participated in wound care had contaminated hands after glove removal. Further studies quantifying the relationship between health care worker hand hygiene compliance and surface concentration could add increased confidence in the comparisons made between these modeled intervention efficacies. Although several models have addressed the exchanges between surface contamination and hand-to-surface contacts, assumptions are sometimes made about the number of surfaces touched by 1 health care worker (in which multiple surfaces may be grouped together represented by a single compartment) and the frequency of contact with these surfaces.[37,38] It has been acknowledged that a common challenge in health care mathematical modeling is that assumptions and results may not be generalizable, owing to a wide variety of health care indoor environments and scenarios.[39]

This model can account for high exposure moments and does not assume a constant concentration on hands over the exposure time. Additionally, this model predicts average baseline risks that are larger than those predicted by models that assume a constant concentration on hands using the same exposure time and distribution of virus concentrations on surfaces. In this study, baseline average infection risks for rotavirus, rhinovirus, and influenza A virus were 0.43, 0.20, and 5.51×10^{-6}, respectively, whereas in a study assuming the same exposure period conducted, reported average infection risks for rotavirus, rhinovirus, and influenza A virus as 7.24×10^{-2}, 2.78×10^{-2}, and 3.43×10^{-7}, respectively, under an assumption that virus concentrations on hands were constant for the exposure period.[17] The discrepancy between these estimated infection risks demonstrates the importance of models that can account for fractions of exposure time in accurately estimating infection risks.

A limitation in this study was the use of microactivity data, or second-by-second behaviors, that were not specific to a health care exposure scenario. The sensitivity analysis revealed that microactivities were included in the top 5 most influential parameters for rotavirus, rhinovirus, and influenza A virus, in which the hand-to-orifice contacts that could result in a dose was influential for each virus (Table 3). Additional knowledge about health care worker behaviors, especially hand-to-orifice contacts that may result in infection, would further inform discrete event models so that more confidence could be placed in the predicted infection risks. The order of events likely influences exposure in addition to the probability of events occurring per second. For example, although moments of high exposure are possible, hand-to-orifice contacts do not always follow hand-to-fomite contacts that result in a higher than average viral load on hands. This means that spikes in viral load on hands may not always result in elevated exposure or infection risk. Over time, if hand-to-orifice contacts rarely follow these high viral loading moments, high viral load on hands does not necessarily have risk implications for an individual. However, high viral load on hands and after hand-to-fomite contacts may affect the exposure and infection risk of others sharing the environment, as this behavior would spread high concentrations of virus around an environment.

Few studies have addressed the timing of recontamination of surfaces postcleaning. In a study conducted by Bogusz et al,[23] surface recontamination was measured in time intervals of 1, 2, 4, 8, 12, 24, and 48 hours postcleaning under normal operating conditions in a 450-bed hospital. It was found that both aerobic colony counts and methicillin-susceptible or methicillin-resistant *Staphylococcus aureus* did not reach precleaning concentration levels after 48 hours.[23] However, this study did not link health care worker behaviors or patient behaviors to the recontamination observed, and the mechanisms of recontamination discussed were focused around microbial growth.[23] The current study assumed a much faster recontamination time because contamination of surfaces was observed as quickly as 2 hours after seeding in a viral tracer study.[12] In another study conducted in an intensive care unit, it was found that surfaces became recontaminated within 4 hours.[40] There are a lack of data characterizing recontamination time, but the results of the previously mentioned studies demonstrate that there could be large differences in recontamination time because of many scenario- and environmental setting-specific parameters. More data are needed to understand how recontamination occurs as a function of health care worker surface contacts, patient surface contacts, patient microbial shedding onto surfaces, and the settling of aerosolized microbes.

This study assumed a linear change in viral concentration over time when surface recontamination was simulated. However, the data

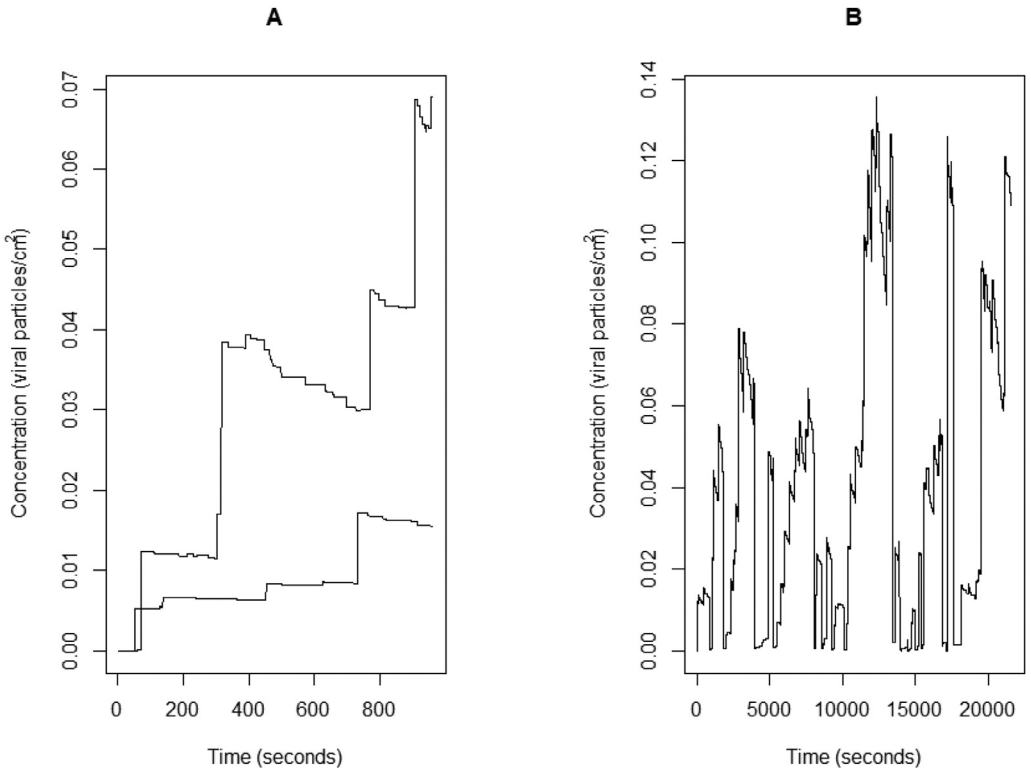

Fig 1. Examples of viral accumulation on hands: (A) virus accumulation on 2 simulated nurses' hands over 16 minutes, (B) virus accumulation on 1 simulated nurse over approximately 6 hours.

reported by Bogusz et al[23] demonstrate that changes in concentration on surfaces over time can take many different polynometric forms, with overall increases. A study conducted by Pittet et al[41] showed linear increases in bacterial colony counts on health care workers hands over 16 minutes of care. However, it is unknown if this accruement continues linearly on the scale of hours, considering that microbial losses from the hand may occur because of contacts with clean surfaces and/or hand hygiene practices. The model used in this study demonstrates that virus can accumulate on the hand stepwise and approximately linearly within the first 16 minutes of exposure (Fig 1A), but accumulation may not continue linearly (Fig 1B). Future environmental microbiology studies are needed to understand the range of variability in recontamination of hands and surfaces. This will continue to inform future efforts to optimize cleaning frequency and duration.

Although simulation models are useful tools in exploring how interventions will affect health outcomes, such as infection risk, the authors acknowledge that changes in behavior, such as a 15% increase in hand hygiene, may not be easily measured or verified in real-world applications for each individual. Therefore, further studies are needed to quantify variability in hand hygiene compliance and from person-to-person and errors in hand hygiene compliance measurement so that this variability can be addressed in future simulation models.

CONCLUSIONS

This study demonstrates the usefulness of mathematical modeling as a tool for predicting infection risk reductions associated with various infection control interventions. We additionally identified areas of future model improvement and gaps in knowledge regarding contamination and exposure mechanisms, and the results reiterate the importance of hand hygiene and surface cleaning interventions in risk mitigation.

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