pathogens. A history of animal or arthropod exposure is a risk factor that can alert laboratory staff to the possibility of tularemia, enabling the application of appropriate precautions (*4*). Pinpoint colonies of gram-negative coccobacilli growing aerobically on chocolate agar 48 hours after plating might indicate the presence of *F. tularensis* and should prompt BSL-3 precautions, as emphasized by the Centers for Disease Control and Prevention's Laboratory Response Network in affiliation with the American Society for Microbiology (*5*,*6*). Of 42 cases of laboratory-acquired tularemia documented by Overholt et al. (*7*), 16 were unsuspected by microbiologists and occurred outside of a known exposure.

These 2 cases caused by *F. tularensis* subspecies *holarctica* support veterinary studies suggesting that this subspecies might be more common in the Canadian prairies than the more virulent *F. tularensis* subspecies *tularensis* identified elsewhere in North America (*8*–*10*). The milder symptoms associated with *F. tularensis* subspecies *holarctica* might require a higher index of clinical suspicion, especially among patients with exposure to arthropods or wild mammals.

About the Author

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Risk for Fomite-Mediated Transmission of SARS-CoV-2 in Child Daycares, Schools, Nursing Homes, and Offices

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Severe acute respiratory syndrome coronavirus 2 can persist on surfaces, suggesting possible surface-mediated transmission of this pathogen. We found that fomites might be a substantial source of transmission risk, particularly in schools and child daycares. Combining surface cleaning and decontamination with mask wearing can help mitigate this risk.

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the causative agent of coronavirus disease, can be transmitted through close contact. However, the virus also persists for up to 28 days on surfaces (*1*–*3*), suggesting that surface-mediated (e.g., fomite) transmission might also occur.

Conventional epidemiologic studies cannot distinguish between competing transmission pathways (e.g., droplet or fomite) when they act simultaneously. Therefore, we used a transmission model to explore the potential for fomite transmission without other pathways. We adapted a published fomite transmission model (*4*) for SARS-CoV-2 (Appendix Figure 1, https://wwwnc.cdc.gov/EID/article/27/4/20-3631-App1.pdf). In our model, persons are classified as susceptible, infectious, or recovered. We explicitly tracked contamination on hands, which is independent of whether or not a person is currently infected. Infectious persons shed pathogens onto fomites or hands, but only a fraction of surfaces (λ) are accessible for contamination. Hands might become contaminated from viral excretion or from touching virus-contaminated fomites. Susceptible persons might become infected through touching their face and mouth with contaminated hands (Appendix).

By using this model, we explore how fomite transmission varies by location (comparing child daycares, schools, offices, and nursing homes), disinfection strategy, and surface type. Although precise values likely vary on a case-by-case basis, child daycares are assumed to have higher frequency of fomite touching (ρ_T) and the fraction of surfaces susceptible to contamination (λ) than offices, whereas schools are likely intermediate for both factors (*4*). Nursing homes are assumed to have similar amounts of surfaces susceptible to contamination to offices, but higher fomite touching rates.

We considered the following surface cleaning and disinfection frequencies: every 8 hours (1×/workday), every 4 hours (2×/workday), and hourly. We also considered handwashing interventions, but they had minimal impact in our model and were not included in our main results (Appendix). Because SARS-CoV-2 persistence varies by surface, we compared transmission for stainless steel, plastic, and cloth. As a sensitivity analysis, we also varied viral shedding rates in our analysis for 2 reasons: initial data are uncertain because of small sample sizes (*5*), and shedding rates are likely to vary on the basis of mask-wearing practices (*6*,*7*; Appendix). In our model, situations in which the basic reproduction number (R_0) for the fomite route exceeds 1 could sustain ongoing transmission in a given setting, whereas transmission could be interrupted when R_0 falls below 1. We explored what interventions could interrupt fomite transmission.

Our estimates suggest that fomite transmission could sustain SARS-CoV-2 transmission in many settings. The fomite R_0 ranged from 10 in low-risk venues (offices) to ≈25 in high-risk settings such as child daycares. SARS-CoV-2 transmission risk is generally higher than influenza and rhinovirus (Appendix Figure 6).

We found that hourly cleaning and disinfection alone could interrupt fomite transmission in some office settings, particularly combined with reduced shedding, but would be inadequate in child daycares and schools (Figure; Appendix Figure 3). If shedding is reduced through mask wearing, transmission from surfaces became unlikely, even with infrequent surface decontamination. Decay rates were similarly low for plastic and stainless steel (Appendix Table 2), leading to substantial transmission potential (Figure). Decay rates on cloth were high and were unlikely to sustain transmission. Therefore, cleaning and disinfection frequencies could vary by surface, with hourly interventions being helpful for frequently touched nonporous surfaces and with porous surfaces (such as plush toys) being cleaned and sanitized less frequently. In child daycares, intervening directly after high-risk shedding

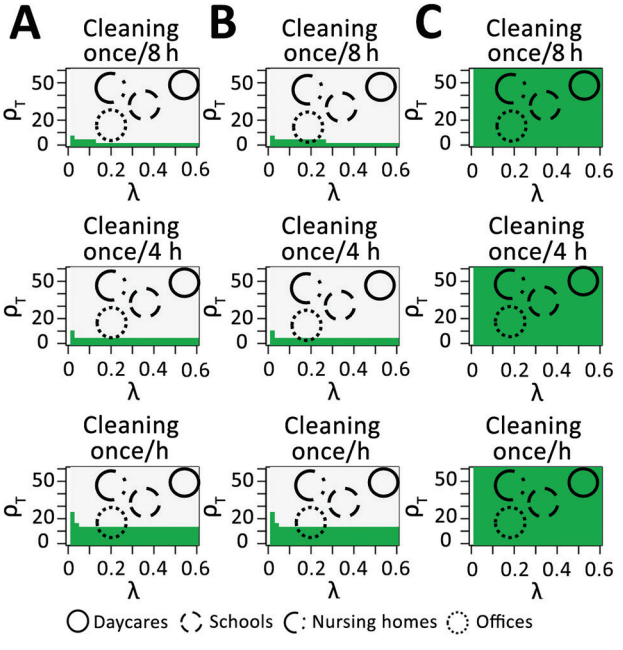

Figure. Reductions in the basic reproduction number for the fomite pathway for severe acute respiratory syndrome coronavirus 2 on stainless steel (A), plastic (B), and cloth surfaces (C), by setting (defined by hourly fomite touching rates [$\rho_T$and proportion of accessible surfaces [λ). For areas in green, the projected reproduction number from fomite transmission is <1. For comparison, cleaning every 2 hours was considered as a sensitivity analysis.

events (e.g., a feverish person coughs directly on a surface) in addition to intervening at standard intervals (such as hourly) would be beneficial.

Because of our emphasis on the basic reproduction number rather than simulating infection dynamics, these results describe transmission potential if outbreaks begin with a single case as opposed to many cases being introduced simultaneously, which could occur when transmission is high. Thus, these results apply when SARS-CoV-2 incidence is low, which might be achievable in individual locations even if community incidence is high. Near the epidemic peak, more detailed simulations are needed because environmental contamination might exceed the linear range of the dose-response curve (*8*), which could lead to an overestimate of the risk for fomite transmission. Because our objective was to assess the potential impact of fomite transmission alone, we did not account for direct transmission through direct droplet spray, aerosols, or hand-to-hand contact, all of which are likely major contributors to transmission in many settings (*9*). Our model suggests fomites can also transmit virus, which is important for indirect exposures. For simplicity, we assume that fomite transmission is similar for symptomatic and asymptomatic infections (Appendix). We also assume that the dose-response curve for fomite transmission is the same as other transmission routes, which might lead to an overestimate of fomite transmission if pathogens from surfaces are less efficiently absorbed into the lungs from hands when they are not aerosolized.

In summary, fomite transmission might be an important source of risk for SARS-CoV-2. However, both mask wearing and frequent cleaning and disinfection can reduce this risk.

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Appendix

Model Details

This model has previously been described in detail, but in brief, the population is classified as susceptible (S), infectious (I), or recovered (R). Infectious individuals can shed pathogens directly onto fomites (F) or onto hands (H compartments, either H_S, H_I, or H_R). Hands have the same infection status as individuals (i.e., all susceptible individuals, S, have susceptible hands H_S). Hands may become contaminated through direct excretion (self-inoculation) by an infectious individual or through touching contaminated fomites. Hand contamination is not dependent on an individual's infection status, thus, a recovered individual (H_R) could have contaminated hands. Susceptible individuals can become infected through touching their face and/or mouth with contaminated hands (self-inoculation) based on the linear dose-response function, P. Pathogens on hands and on fomites are inactivated based on the persistence properties of the pathogen. In addition to natural inactivation, pathogens may be removed from hands or surfaces through cleaning interventions, which is determined by both the frequency and efficacy of the cleaning. The parameter values considered in this model and their definitions are shown in Appendix Table 1. The model equations and the corresponding R_0 equations are shown below. A model diagram is shown in Appendix Figure 1.

In our model, we assume a cleaning and decontamination efficacy of 100%, consistent with initial data on the efficacy of standard cleaning agents (*1*). However, if cleaning without disinfection is used (i.e., removing only organic matter on surfaces without using disinfection agents, such as bleach), higher frequencies of cleaning would be unlikely to reduce risk unless pathogen shedding on surfaces can also be substantially reduced.

$$\frac{dS}{dt} = -\rho P\left(\frac{\chi H_S}{S}\right)S$$

$$\frac{dI}{dt} = \rho P\left(\frac{\chi H_S}{S}\right)S - \gamma I$$

$$\frac{dR}{dt} = \gamma I$$

$$\frac{dF}{dt} = \alpha_F I - (\rho_{FH} N + \mu_F + \theta_F)F + \rho_{HF}(H_S + H_I + H_R)$$

$$\frac{dH_S}{dt} = \rho_{FH} SF - (\mu_H + \rho_{HF} + \chi\rho + \theta_H)H_S - (1-\chi)\rho P\left(\frac{\chi H_S}{S}\right)H_S$$

$$\frac{dH_I}{dt} = \rho_{FH} IF - (\mu_H + \rho_{HF} + \chi\rho + \theta_H)H_I + \alpha_H I + (1-\chi)\rho\, P\left(\frac{\chi H_S}{S}\right)H_S - \gamma H_I$$

$$\frac{dH_R}{dt} = \rho_{FH} RF - (\mu_H + \rho_{HF} + \chi\rho + \theta_H)H_R + \gamma H_I$$

$$R_0 = \frac{1}{\gamma}\left[\frac{\chi\rho\pi}{\mu_H + \rho_{HF} + \chi\rho + \theta_H}\right]\left[\left(\frac{\frac{\rho_{FH}N}{\rho_{FH}N + \mu_F + \theta_F}}{1 - \frac{\rho_{HF}\rho_{FH}N}{(\mu_H + \rho_{HF} + \chi\rho + \theta_H)(\rho_{FH}N + \mu_F + \theta_F)}}\right)\left(\alpha_F + \alpha_H\left[\frac{\rho_{HF}}{\mu_H + \rho_{HF} + \chi\rho + \theta_H}\right]\right) + \alpha_H\right]$$

Deriving model parameters for SARS-CoV-2 and uncertainty

For unknown persistence and transfer efficiency parameters, we used influenza values because SARS-CoV-2, like influenza, is an enveloped virus, which tend to have lower persistence than non-enveloped viruses on surfaces (*34*). For uncertain infectivity parameters, we used values from other coronaviruses (if available) or rhinovirus because of the similarity in symptoms likely to drive transmission.

Infectivity parameters—shedding rate and dose response curves

To provide the most accurate infectivity data, we used shedding rates that were in terms of $TCID_{50}$ units, as this excludes potential bias from measuring genetic material from inactive virus. $TCID_{50}$ units describe the concentration of virus that would be expected to infect 50% of challenged cells, whereas PFU units describe the number (count) of plaques likely to be formed

in a cell culture containing a given concentration of infectious virus. Based on this definition, when $TCID_{50} = 1$, the expected number of observed plaques is 0. Therefore, for parameters in PFU units, we converted between the two scales as follows, applying the Poisson distribution, where the expected number of plaques observed is dependent on μ, the PFU/mL (*35*,*36*):

$$P(Count\ of\ plaques = y) \sim Pois(\mu = PFU) = \frac{e^{-\mu}\mu^{y}}{y!}$$

$$P(Count\ of\ plaques = 0) = \frac{e^{-\mu}\mu^{0}}{0!}$$

$$0.5\ TCID_{50}/mL = e^{-PFU}$$

Thus,

$$TCID_{50} = -\ln(0.5)\ PFU$$

Decay rates on fomites

For SARS-CoV-2, decay rates on different surfaces were calculated based on the slope of the decay curves first 24 hours for cloth (highest decay rates), plastic (lowest decay rates) and stainless steel (medium decay rates). For cloth, the decay rates were calculated based on the first 30 minutes of decay because most samples became undetectable after the first 30 minutes of observation and were then multiplied by 2 to get the corresponding hourly decay rates. Because experimental data was presented on the log10 scale, the decay rates from the different experimental studies were initially calculated on the log10 scale. To convert these numbers to the exponential scale, the decay rate on the log10 scale was multiplied by ln(10).

Decay rates on hands

For SARS-CoV-2, there were no reliable pathogen-specific data for the decay rate of pathogen on hands. One study used skin samples from corpses to estimate this quantity and calculated decay rates for both influenza and SARS-CoV-2. In this study, decay rates on hands were substantially lower for SARS-CoV-2 compared with influenza, with SARS-CoV-2 surviving for about eight times as long as influenza (*22*). However, the decay rates from influenza obtained using this method differed substantially from prior decay rate estimates for influenza (*14*,*21*), casting doubt on this model system for estimating the absolute decay rates. Moreover, this study used a method that pipetted pathogen directly onto hands rather than first

transferring pathogen from an inanimate surface to hands. Given that decay rates on hands vary based on the surface from which they are transferred (even after accounting for transfer efficacy) (*21*), these data were inappropriate for use in our fomite model. For this reason, we relied on decay rates previously estimated for influenza and considered a lower hand decay rate as a sensitivity analysis, where the decay rate on hands for SARS-CoV-2 was 11.02/hour, ≈1/8 the decay rate previously estimated for influenza.

Transfer efficacy

For SARS-CoV-2, there were no data available on transfer efficacy, so we relied on the transfer efficacy parameters previously estimated for influenza.

Handwashing

We initially considered a handwashing intervention that removed all pathogens on hands at regular intervals. However, SARS-CoV-2 is predicted to have very low persistence on hands (Appendix Table 1). For this reason, even hourly handwashing with 100% effectiveness had almost no impact on transmission risk. To illustrate, we reran Figure (https://wwwnc.cdc.gov/EID/article/27/4/20-3631-F1.htm) assuming hourly handwashing and the results were nearly identical to the version in the main text with no handwashing (Appendix Figure 2).

Shedding Sensitivity Analysis

Baseline hand persistence

As a sensitivity analysis, we considered how the basic reproduction number would change if the hourly shedding rate were slightly different, using stainless steel as the surface for comparison. Preliminary data suggest a shedding rate of 1800 pathogens/person/hr (1.8E3) (*6*), which is the estimate used in the main text. We considered 2E3 as a potential upper bound on shedding for SARS-CoV-2 (Appendix Figure 3). Widespread use of masks might also lower the overall shedding rate. To approximate the impact of mask wearing, we reduced both shedding (α) and the rate of self-inoculation (ρ) by 75% ($\alpha = 450, \rho = 3.95$), consistent with estimates of viral reduction in droplets during speech with mask wearing (*13*).

Shedding might be lower or higher than expected for several reasons. First, initial studies were limited in sample size, which may have reduced the precision and accuracy of early

estimates. In addition, overall shedding might vary by level of symptoms. While viral load has been found to be similar in secretions of symptomatic and asymptomatic cases, they may not have similar contributions to overall transmission if shedding is influenced strongly by symptoms. For example, the number of droplets expelled during coughing, a common symptom of SARS-CoV-2, is much higher than during speech (*37*). On the other hand, symptomatic individuals may be more likely to take precautions to limit viral spread, making asymptomatic transmission relatively more important. With mask wearing, transmission from surfaces was unlikely, even when surface decontamination was set to its minimum value (once every 8 hours).

Higher hand persistence

We repeated the same sensitivity analysis varying shedding while also allowing persistence to be higher, based on data from (*22*). When hand persistence was higher, transmission from surfaces could not be controlled unless both mask wearing and frequent surface disinfection were used (Appendix Figure 4). Even with hourly handwashing and mask wearing, transmission from surfaces would only be controlled in office settings.

Alternative cleaning frequencies

For comparison, we reran Figure 2 considering cleaning frequencies every 2 hours (Appendix Figure 5). The impacts were intermediate between cleaning every 4 hours and hourly cleaning.

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Appendix Table 1. List of pathogen-specific parameters values and references*

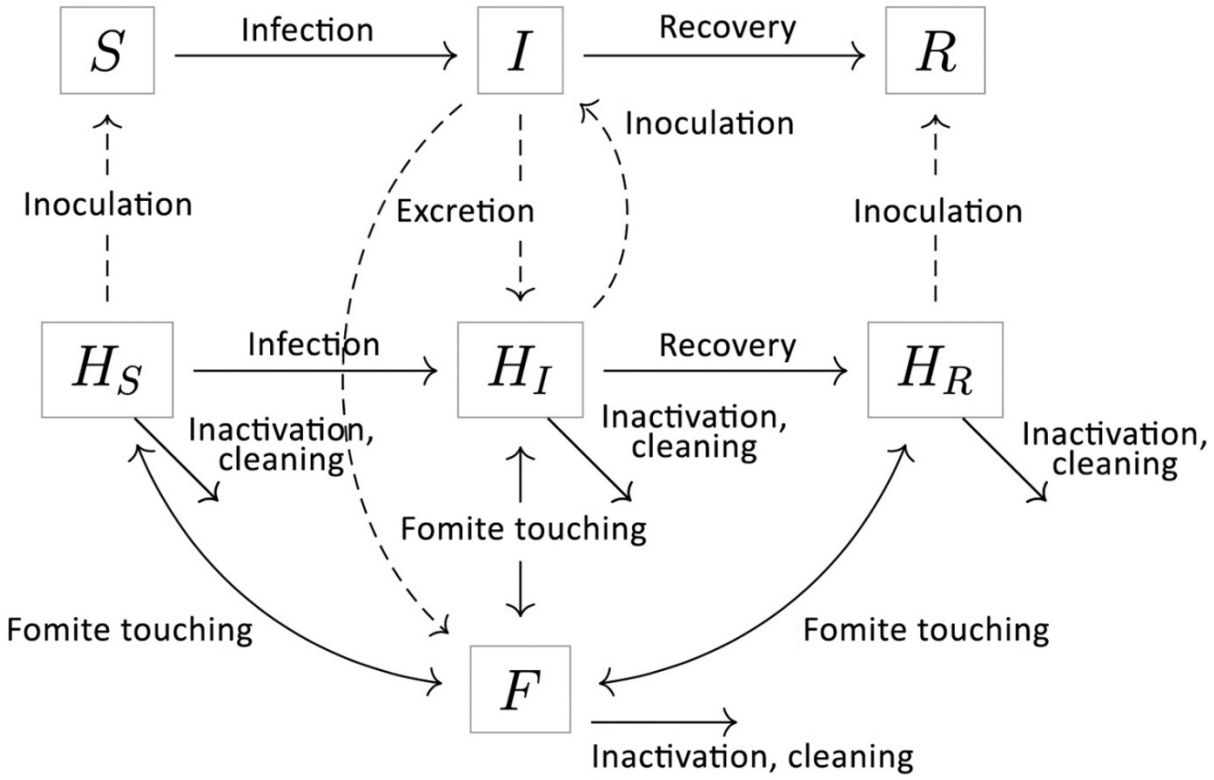
Parameter type	Influenza	Rhinovirus	SARS-CoV-2
Pathogen-specific parameters			
$1/\gamma$: Infectious period (days)	6 (*2–4*)	10.4 (*5*)	8 (*6,7*)
α: Shedding rate (pathogen hours^{-1} people^{-1})	1×10^4 *(8–10)*	1×10^3 *(11,12)*	1.8×10^3 *(6,13)* ($450, 2 \times 10^3$)
μ_F: Inactivation rate in fomites (hours^{-1})	0.121 (*14–16*)	1.44 (*17,18*)	0.148 (*1,19*) (0.131, 9.21)
μ_H: Inactivation rate in hands (hours^{-1})	88.2 (*14,16,20*)	0.767 (*21*)	88.2 (*14,16,20,22*)
τ_{FH}: *Transfer efficacy (F to H) (proportion)*	0.1 (*14,20,23,24*)	0.2 (*21,25–27*)	**0.1** (*14,20,23,24*)
τ_{HF}: *Transfer efficacy (H to F) (proportion)*	0.025 (*14,20,23,24*)	0.2 (*21,25–27*)	**0.025** (*14,20,23,24*)
ϕ_H: *Pathogens excreted to hands (proportion)*	0.15	0.15	0.15
ϕ_F: *Pathogens excreted to fomites (proportion)*	$1 - \phi_H$	$1 - \phi_H$	$1 - \phi_H$
π: Infectivity parameter in contact with x pathogens (unitless, $P'(0)$)†	6.93×10^{-5} *(28)*	2.46×10^{-3} *(28)*	3.55×10^{-3} *(29–31)*
α_H: rate pathogens are added to hands (pathogen hours^{-1} people^{-1})	$\alpha\phi_H$	$\alpha\phi_H$	$\alpha\phi_H$
Venue-specific parameters			
λ: Accessible surfaces (proportion)	(0, 0.6)	(0, 0.6)	(0, 0.6)
κ: fingertip to surface ratio per individual (1/people)‡	$\frac{6 \times 10^{-6}}{\lambda}$	$\frac{6 \times 10^{-6}}{\lambda}$	$\frac{6 \times 10^{-6}}{\lambda}$
ρ_T: rate of fomite touching (hours^{-1})	(0, 60)	(0, 60)	(0, 60)
ρ_{FH}: *rate of fomite pick up from fomites to hand (1/(hours x people))*	$\rho_T\tau_{FH}\kappa$	$\rho_T\tau_{FH}\kappa$	$\rho_T\tau_{FH}\kappa$
ρ_{HF}: *rate of fomite pick up from hands to fomites (1/(hours x people))*	$\rho_T\tau_{HF}$	$\rho_T\tau_{HF}$	$\rho_T\tau_{HF}$
α_F: rate pathogens are added to fomites (pathogen hours^{-1} people)	$\alpha\phi_F\lambda$	$\alpha\phi_F\lambda$	$\alpha\phi_F\lambda$
Cleaning parameters			
Θ_F: Rate of fomite cleaning (hours^{-1})	(1/8, 1)	(1/8, 1)	(1/8, 1)
Θ_H: Rate of hand cleaning (hours^{-1})	(1/8, 1)	(1/8, 1)	(1/8, 1)
q_F: *Fomite cleaning efficacy (proportion)*	1	1	1
q_H: *Hand cleaning efficacy (proportion)*	1	1	1
θ_F: Effective fomite cleaning rate (hours^{-1})	$q_F\Theta_F$	$q_F\Theta_F$	$q_F\Theta_F$
θ_H: Effective hand cleaning rate (hours^{-1})	$q_H\Theta_H$	$q_H\Theta_H$	$q_H\Theta_H$
Fixed parameters (across pathogens and venues)			
ρ: Self-inoculation (hours^{-1})	15.8 (*32,33*)	15.8 (*32,33*)	15.8 (3.95, 15.8) (*13,32,33*)
χ: Proportion of pathogens absorbed when self-inoculation occurs (proportion)	1	1	1

*Parameter values for rhinovirus and influenza are shown for comparison. A range is also included for parameters that were used to perform a sensitivity analysis (frequency of cleaning, infectious period, shedding rate, and persistence on surfaces). Derived parameters are shown as a function of the parameters used to derive them. Decay rates on fomites for influenza and rhinovirus are for stainless steel. For SARS-CoV-2, the decay rates shown in parentheses are the range of observed for plastic (lowest decay rates), stainless steel, and cloth (highest decay rates). For SARS-CoV-2, parameters that were extrapolated based on data from other pathogens are shown in bold.
†Parameter fixed based on linearization of the dose-response curve, P.
‡Parameter fixed based on relative finger to body size

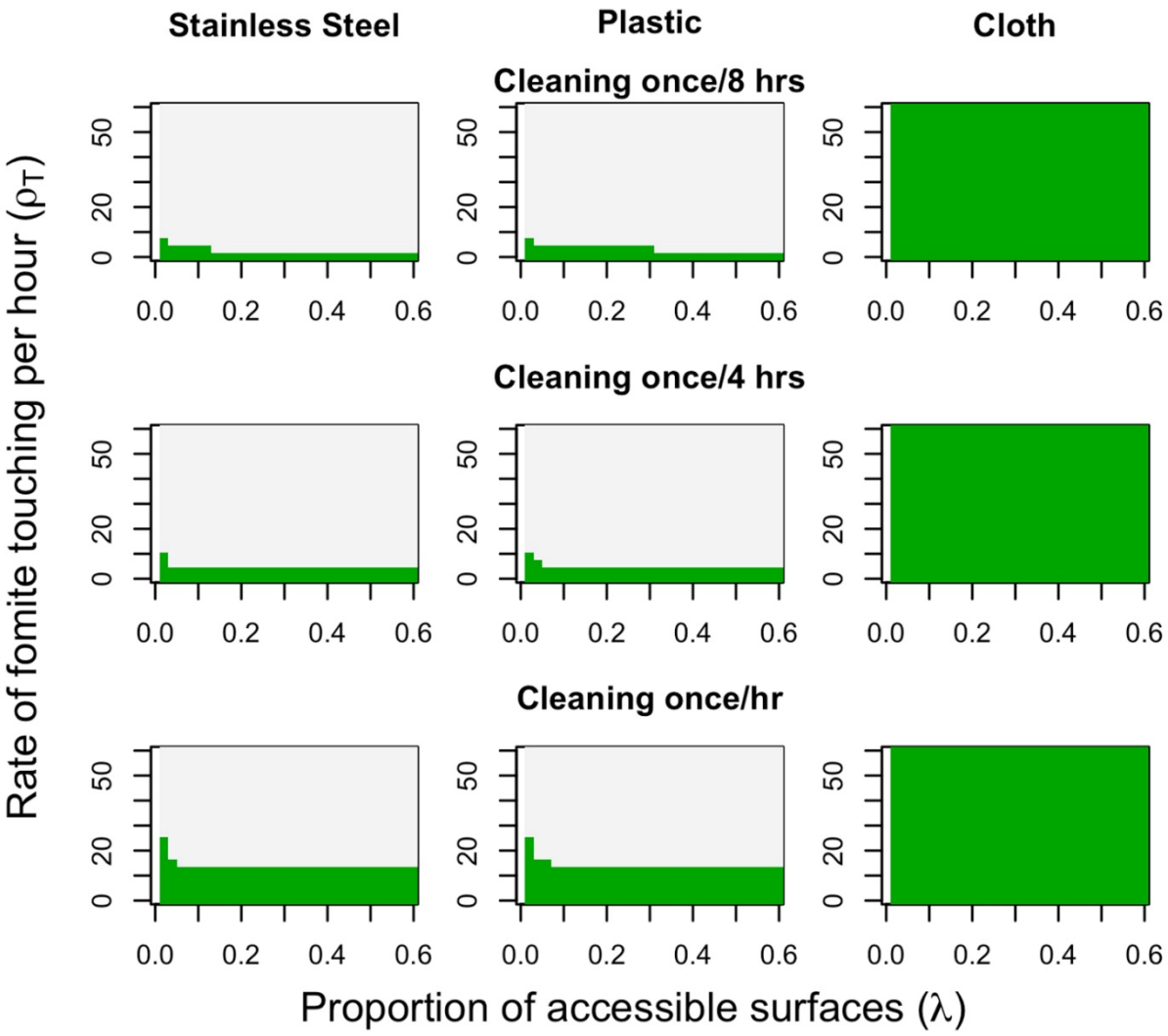
Appendix Table 2. Decay rates for each surface*

Surface	Doremalen et al. (*19*)	Chin et al. (*1*)	Average
Stainless steel (ln/hr)	0.170	0.092	0.131
Plastic (ln/hr)	0.108 (3.64, 2.52)	0.184	0.146
Cloth (ln/hr)	NA	9.21	9.21

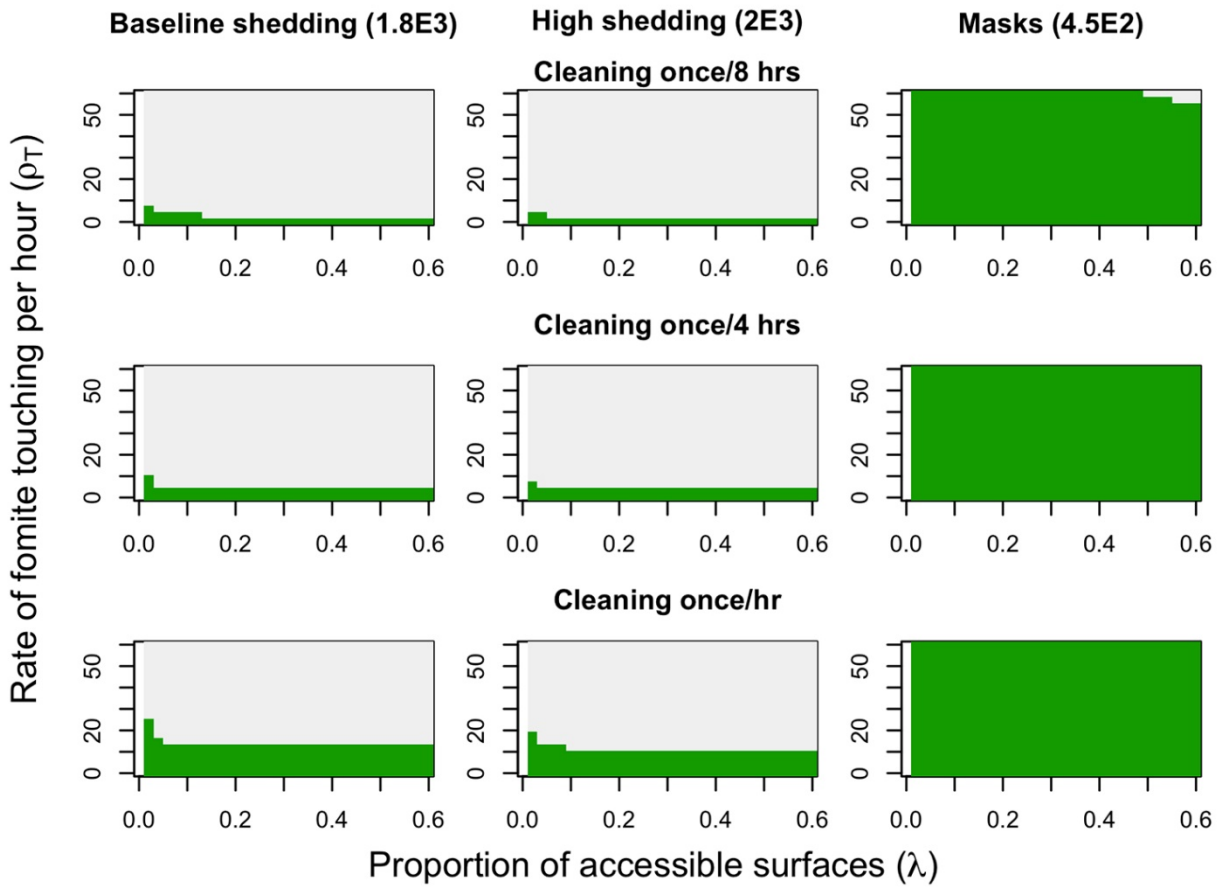
*NA, not available.



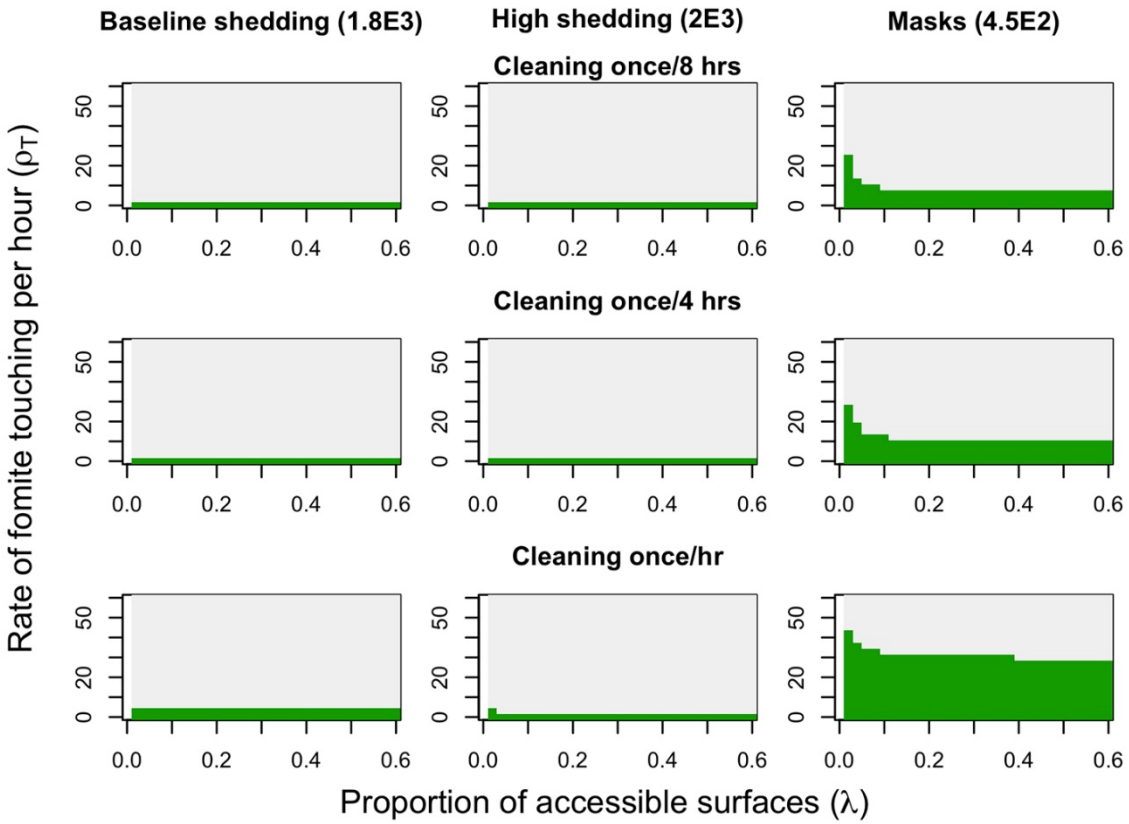
Appendix Figure 1. Model tracks individuals (in compartments *S*, *I* or *R*) and pathogens on fomites (*F*) and hands (hands of susceptible individuals, H_S; hands of infected individuals, H_I; hands of recovered individuals; H_R). The six events (inoculation, fomite touching, excretion, pathogen inactivation, cleaning, and recovery) are represented by arrows in the direction of the corresponding flow.



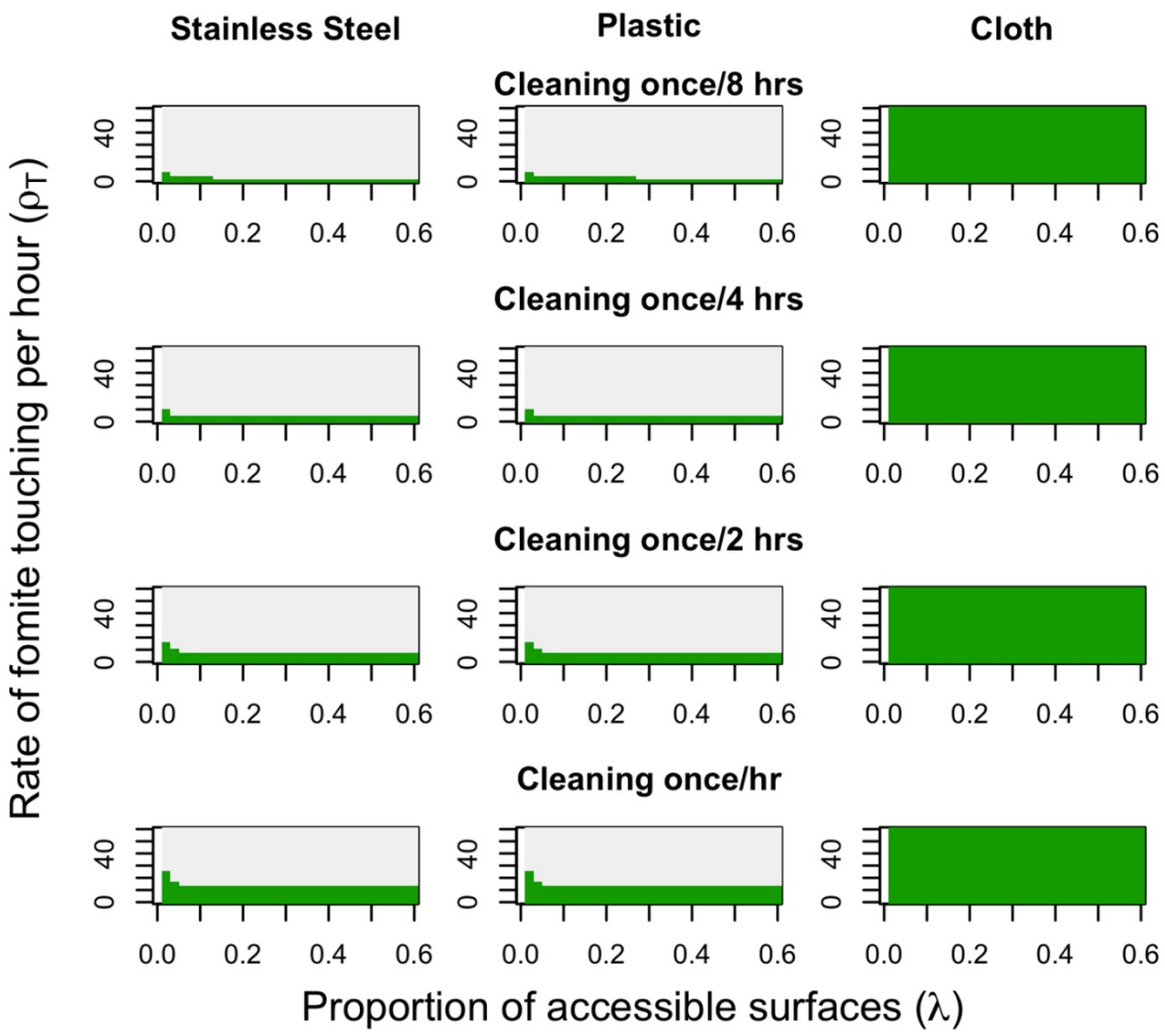
Appendix Figure 2. Reductions in the basic reproduction number by cleaning strategy and surface with hourly handwashing included. For areas in green, the projected reproduction number from fomite transmission is below 1.



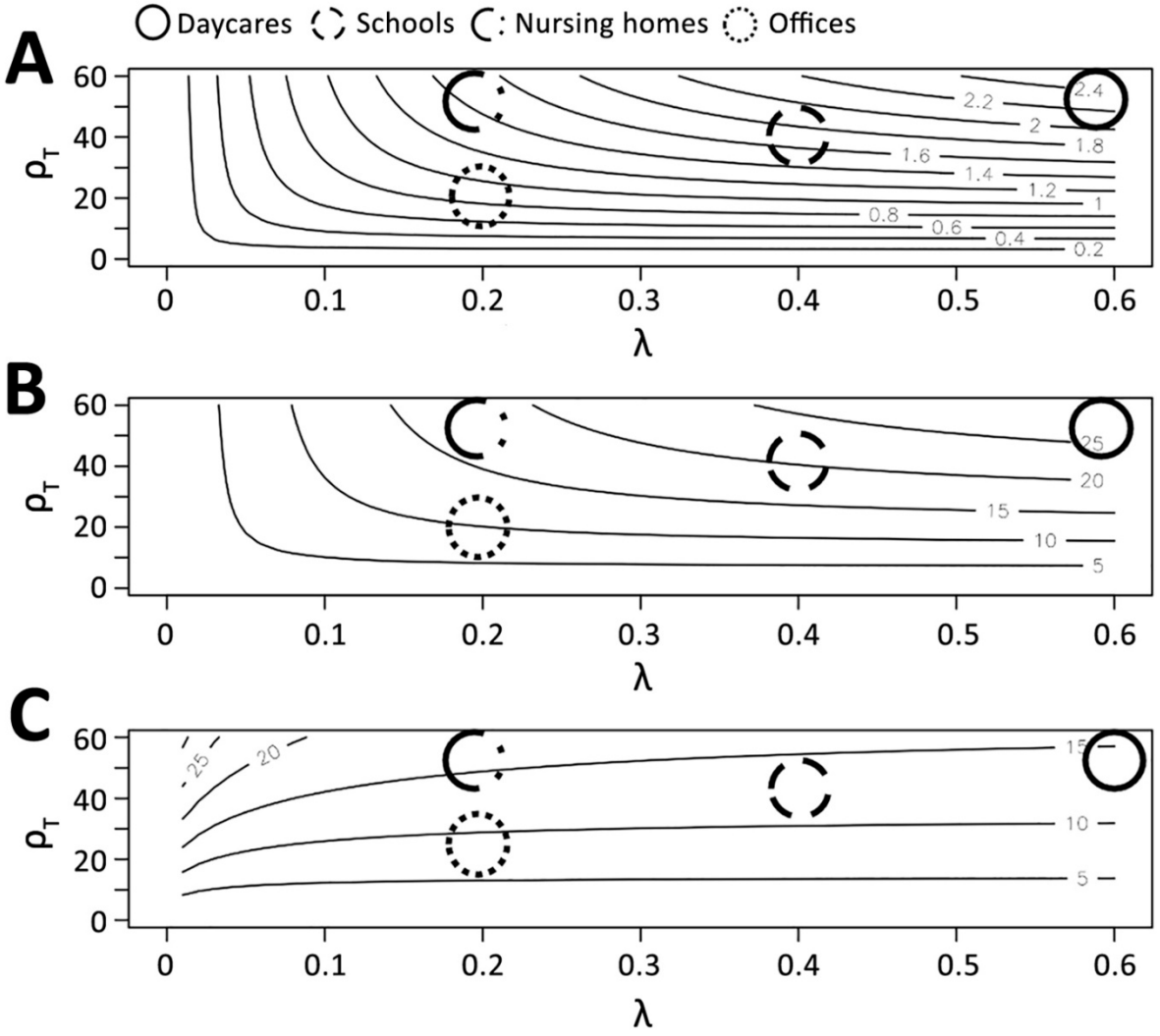
Appendix Figure 3. Reductions in the basic reproduction number by cleaning strategy for different shedding rates (baseline, upper bound of shedding, and mask wearing). For areas in green, the projected reproduction number from fomite transmission is below 1.



Appendix Figure 4. Reductions in the basic reproduction number by cleaning strategy for different shedding rates (baseline, upper bound of shedding, and mask wearing) for stainless steel surfaces assuming higher hand persistence. For areas in green, the projected reproduction number from fomite transmission is below 1.



Appendix Figure 5. Reductions in the basic reproduction number for different surfaces. For areas in green, the projected reproduction number from fomite transmission is below 1.



Appendix Figure 6. Predicted basic reproduction number for the fomite pathway for A) influenza, B) severe acute respiratory syndrome coronavirus 2, and C) rhinovirus without any interventions, by setting. Hourly fomite touching rates (ρ_T) and proportion of accessible surfaces (λ) are not known precisely, so larger circular symbols are used to reflect uncertainty, highlighting the plausible range. All 3 pathogens are shown using decay rates from stainless steel surfaces. Parameters used for each pathogen are shown in Appendix Figure 1.