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Quantitative microbial risk assessment of human norovirus infection in environmental service workers due to healthcare-associated fomites

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SUMMARY

Background: Healthcare-associated norovirus outbreaks place a large burden on healthcare staff. Environmental service workers (ESWs), however, remain understudied despite high contact with potentially contaminated surfaces. Understanding the magnitude of the risk of norovirus infection in healthcare ESWs can protect workers and improve infection control.

Aim: This study simulated the risk of norovirus infection for unprotected ESWs after a single fomite contact, assuming no disinfection or protective equipment, in norovirus-positive patient rooms. In addition, the risk of secondary surface transmission from norovirus-exposed ESWs was simulated.

Methods: A quantitative microbial risk assessment employing two-dimensional Monte Carlo simulation with parameters extracted from the literature was used to estimate norovirus infection from multiple fomite contact scenarios defined by: norovirus source (patient vomit/diarrhoea), location (bathroom/patient room) and target outcome (ESW/secondary illness).

Findings: Unprotected ESWs have a maximum estimated risk of norovirus infection of 33% (1:3) for a single fomite contact in a room where a norovirus-positive patient had a diarrhoeal event. Patient vomit events lead to fomite contact risk estimates that are four orders of magnitude

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Conflict of interest statement

None declared.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jhin.2021.08.006.

lower than those for diarrhoeal events. The estimated risk of secondary illness from touching a common surface is as high as 25% (1:4) after single fomite exposure following a diarrhoeal event.

Conclusions: A single fomite contact may lead to sizable risk of norovirus infection in ESWs if personal protective equipment and disinfection are not used appropriately. ESWs can also transfer virus to secondary surfaces, initiating further infections. Interventions are needed to reduce fomite transfer of norovirus, and protect patients and staff from nosocomial infections.

Keywords

Norovirus; Environmental service workers; Health care; Fomite; Risk assessment

Introduction

Human noroviruses, often generically referred to as ‘norovirus’, cause approximately 685 million illnesses and cost the global economy $64.5 billion each year [1,2]. In many countries, including the USA, the majority of norovirus outbreaks occur in healthcare facilities, including acute care hospitals and long-term care facilities [3,4]. The burden of healthcare-associated norovirus infections is high, and outbreaks may be prolonged and difficult to control in areas caring for immunocompromised or elderly patients, ultimately exposing healthcare providers (HCPs) for months until transmission ceases [5–8]. In many healthcare-associated outbreaks, HCPs represent the majority of cases [5,7]. Norovirus infections in healthcare facilities can lead to significant monetary losses and reduction in hospital capacity due to closures and ill staff [9]. For example, a single norovirus outbreak in a tertiary care hospital in 2004 led to 265 HCP illnesses and an estimated cost of $657,644 [5].

Norovirus possesses multiple characteristics that make it a significant healthcare pathogen. Norovirus is shed in high titres from the vomit and faeces of symptomatic individuals [10]. There is also frequent asymptomatic shedding of infectious virus [11], and the infectious dose of norovirus is very low with the median infectious dose estimated to be between 10 and 100 particles [12]. In a healthcare setting, the presence of immunocompromised patients can increase the likelihood of norovirus transmission as these patients can chronically shed high titres of norovirus [3,13].

While multiple studies have quantified the impact of norovirus on HCPs, the majority have focused on nurses, doctors and other clinical staff [7–9,14]. In many labour sectors, including health care, cleaning and facilities staff are frequently understudied [15]. In the healthcare setting, environmental service workers (ESWs) clean and disinfect facilities and are responsible for executing infection control protocols. ESWs are often in patient areas with a similar frequency as some clinical staff, but very little research exists on their risk of infection [15]. A recent study found that the prevalence of coronavirus disease 2019 was higher among cleaning staff in a clinic than among clinical staff; this points to a potential undetected disease burden in ESWs, although the source of infections was not determined [15]. The US Department of Labor reports that caretakers and cleaning staff have a higher incidence of illness and injury than registered nurses [16].

Significant resources have been dedicated to assessing knowledge, providing education and changing behaviours among ESWs to improve cleaning and disinfection [17–20]. There is evidence that integrating ESWs into healthcare infection control planning results in reduced patient illness [8]. However, current research fails to examine the health burden placed on ESWs who work in close contact with ill individuals and contaminated environments.

The majority of healthcare-associated norovirus outbreaks begin due to environmental contamination, and fomites represent a significant environmental route of exposure [8,21–23]. Despite the concerns with fomite transmission of norovirus, there is evidence that HCPs lack knowledge about fomites as a pathogen vector, and may not follow appropriate hand hygiene practices after fomite contact [24,25]. When focusing on ESWs, the role of fomite transmission of norovirus becomes even more important as cleaning activities result in a high number of fomite contacts [26]. ESWs are also likely to underestimate the risks posed to them from fomites, and may not follow strict hand hygiene practices after contacting fomites [20].

A quantitative microbial risk assessment (QMRA) is a valuable tool to quantify the risk of norovirus infection faced by ESWs. QMRA is a technique to model infection and illness risk in a population exposed to micro-organisms, based on a four-step process: hazard identification, exposure assessment, dose–response and risk characterization [27]. QMRA allows for the quantification of risk of infection under multiple scenarios, which can be useful in estimating health burdens, designing intervention strategies and identifying research needs. Each step requires detailed analysis of a scenario of interest, and requires quantitative information to develop a final model. In QMRA, it is possible to integrate both variability, which describes inherent differences in the population of interest, and uncertainty, which reflects imperfect knowledge [28]. In general, uncertainty can be reduced with additional data and improved models, while variability will not change with more measurements although it may be better characterized [28]. Analysis of both variability and uncertainty in a QMRA can be achieved through the use of two-dimensional Monte-Carlo (2D MC) simulation [29].

The goal of this work was to use a 2D MC method to quantify the risk of norovirus infection resulting from a single fomite contact within patient areas occupied by norovirus-positive patients who are actively shedding norovirus. Illness in unprotected ESWs was considered as an endpoint, in addition to secondary illness as a result of fomite transfer from ESWs. This study focused on unprotected ESWs and did not include the use of personal protective equipment (PPE), disinfection or handwashing, although these measures are well known to control norovirus. This study aimed to provide a worst-case scenario estimate of the risk of norovirus infection, and to use the resulting model to understand fomite transmission and deposition dynamics of norovirus.

Methods

Model scenarios

Eight scenarios were developed to compare the risk of norovirus infection from different norovirus sources, locations and populations experiencing the health outcome. For each

scenario, this study aimed to develop a QMRA model using 2D MC that captured a plausible upper bound of risk by modelling norovirus exposure using the highest values reported in current literature. The risk of norovirus infection from a single fomite contact that occurred after a single norovirus source event – either vomit or diarrhoea from an infected patient – was simulated (Figure 1). Multiple scenarios were modelled to examine risk differences between the type of source event and the location of the source event, either in the patient's bathroom or in their main room. Additionally, this study was interested in two populations for the final health outcome of norovirus infections: (1) ESWs who touch contaminated fomites directly ('primary outcome'); and (2) other HCPs who touch a shared fomite that was first touched by an ESW after their exposure to a contaminated patient's space ('secondary outcome'). In all scenarios, the initial fomite contact was made by an ESW, and PPE, disinfection and handwashing were not considered.

For in-bathroom scenarios, both source events were assumed to occur in the toilet, while for in-room events, the use of a bed pan for diarrhoea and a basin for vomiting was assumed. The touch fomite of interest in bathroom scenarios was the bathroom door handle, as ESWs are assumed to touch the bathroom door handle in order to perform bathroom cleaning. For in-room scenarios, the fomite of interest was the patient bed rail, as the literature has reported this to be the most commonly touched near-patient fomite for HCPs [26]. For secondary fomite infections, the contact surface was modelled as a door handle in common space on the ward. Model assumptions are listed in Table I.

Model parameters

The corresponding variable notation listed in Table II is provided parenthetically in the text below, and corresponds to variable names in the R code included with the online supplementary material; variables are presented in the order they are used in the corresponding R code. In general, uncertainty distributions were truncated at minimum and maximum values of corresponding variability distributions, and variability distributions were truncated at biologically impossible values (excluding stool production <0, transfer proportions >1).

Norovirus shedding

The mean concentration of norovirus in vomit (*m.vv*) was modelled as an uncertainty distribution using data from human challenge studies. This study focused on research by Kirby *et al.* [30] of infected patients with GII.2 snow mountain virus, which measured mean shedding of 1.6×10^5 norovirus genome equivalents (GE)/mL vomitus [standard deviation (SD) 4.5×10^4 GE/mL] [30]. The study by Kirby *et al.* was selected because it used a genotype II norovirus, which is widely circulated in healthcare facilities [4,22]. The mean virus concentration in vomit was modelled using a normal distribution. Based on a previously conducted QMRA, the present authors chose to model variability in norovirus concentration in vomit (*c.vv*) using a BetaPert distribution, with the mode selected from the uncertainty distribution, a minimum of 2.2×10^3 GE/mL and a maximum of 1.2×10^7 GE/mL [31,32].

The next parameter modelled was the volume of vomit produced in a single event. A vomit event was defined as the production of vomitus that occurs within a 15-min period [30]. It was assumed that the vomit event was early in the course of norovirus disease, and not later when dry-heave or retching events may be more likely. Previous literature indicates that vomitus >800 mL is considered abnormally high for patients with norovirus, and vomitus <50 mL is considered a 'dry-heave' event [33]. In norovirus challenge studies, mean production of vomitus during an entire course of infection with norovirus GII.2 snow mountain virus resulted in mean vomitus of 845 mL (SD 227 mL), and patients experienced an average of two vomit events during the course of their infection [30]. To model uncertainty of the mean volume of vomit (*m.vlv*) from a single event, the total volume of vomit was modelled with a normal distribution with a mean of 845 mL (SD 227 mL), and this value was divided by 2 to obtain the estimated mean volume of vomit for one event. The resulting estimated mean was used as the mode for a BetaPert distribution, with a minimum of 50 mL and maximum of 800 mL to model variability (*vl.v*).

The mean mass of faeces in one diarrhoeal event (*m.mf*) was estimated using mean diarrhoea amounts from Read *et al.* [34] to obtain an average daily mass of 437 g (SD 76 g). These values were divided by the estimated average number of daily bowel movements for a norovirus patient (4.45) [35]. This resulted in a normal distribution with mean of 98.2 g (SD 17.08 g). The final mass of faeces (*m.f*) was modelled using a BetaPert distribution with a range of 14.6–449.4 g which represent minimum and maximum daily mass, divided by an estimated 4.45 daily bowel movements [35]. The concentration of norovirus in faeces (*c.vf*) was modelled as a variability parameter with a BetaPert distribution using values from a previous QMRA [32].

Aerosolization and fomite deposition

For the amount of virus released into room air, two parameters were modelled: the proportion of virus aerosolized during vomiting, and the proportion aerosolized during flushing a toilet. These two values were assumed to be additive, and degradation of virus in vomitus was not considered. Additionally, it was assumed that diarrhoeal events only produced aerosol from flushing and not during defaecation, as individuals will block air movement while seated.

Particles released from vomit and flush events were considered to reach their maximum number in the 1-m^3 space around the toilet, as research has indicated initial dispersion of particles post flush approximately 1 m above the ground [36]. Particles were subsequently assumed to disperse evenly through the room within 1 min [37]. The total amount of virus per m^3 was calculated by dividing total particles aerosolized by room volume [36].

Data reported by Tung-Thompson *et al.* [33] were used to calculate the aerosolization of norovirus from a vomit event (*p.av*). Values from a high-pressure simulated vomiting event with low-viscosity vomitus were chosen, as these values reflect a worst-case scenario. Variability in the proportion of particles aerosolized from one vomit event was modelled as a normal distribution based on the concentration of virus in vomit. For low-titre vomitus ($<10^{10}$ GE), a mean of 2.8×10^{-5} (SD 1×10^{-5}) was used, while for high-titre vomitus ($\geq 10^{10}$ GE), a mean of 1.3×10^{-4} [SD 1×10^{-4}] was used. Cut-off values from 10^7 to 10^{10}

for low-vs high-titre vomitus were tested, and no difference was observed in the final risk estimates (data not shown).

For variability in aerosolization of norovirus from toilet flushing (*p.af*), data from Barker and Jones [38] were used, where a toilet bowl was inoculated with a known amount of the norovirus surrogate MS2, flushed, and the air concentration was measured. For one flush, 2.42×10^{-7} of in-toilet virus was aerosolized, and this was modelled as the mean of a normal distribution (SD 6.91×10^{-8}).

Bathroom volume was calculated from a study that measured areas of bathrooms in a modern tertiary care hospital [39]. The height of the room was assumed to be 2.39 m, which meets Facility Guidelines Institute guidelines for hospital construction [40]. Area values were multiplied by 2.39, and values were averaged to obtain an estimated bathroom volume of 12.4 m^3. For patient room volumes, suggested room areas from the literature were multiplied by an assumed room height of 2.39 m and averaged, resulting in an estimated patient room volume of 26.5 m^3 [41]. It was assumed that patient room doors and bathroom doors were closed during source events, and that air mixing between the rooms did not occur. The concentration of norovirus per m^3 in the room air was calculated for each scenario using the amount of virus aerosolized from the vomit event and/or toilet flush.

Equations for the concentration of norovirus in the air in each scenario were:

Vomit in bathroom, immediately post flush

$$(c.vv * vl.v * (p.av + p.af))/vlr \quad \text{[c.va1]}$$

Diarrhoea in bathroom, immediately post flush

$$(c.vf * m.f * p.af)/vlr \quad \text{[c.va2]}$$

Vomit in patient room, immediately after event

$$(c.vv * vl.v * p.av)/vlr \quad \text{[c.va3]}$$

For in-room diarrhoeal scenarios, no aerosolization was assumed due to the absence of both vomiting and toilet flushing.

Air sampling and settle plate data from Barker and Jones [38] were used to model the settling rate of aerosolized virus on to fomites. After flushing a toilet inoculated with MS2, the authors measured a 93% reduction in virus particles in air 30 min post flush. It was estimated that 93% of virus per m^3 area will fall out on 1 m^2 in 30 min, which results in a fomite deposition rate of 0.031 from m^3 to m^2 per min for up to 30 min. After 30 min, it was assumed that 100% of aerosolized particles deposited on to fomites. Time since event (*delt.t*) was modelled as a uniform distribution from 1 to 1440 min, assuming an ESW would enter a room once per day for cleaning [42].

The surface area of hand contact (*ah*) was estimated by painting a standard metal lever-type door handle, author KO holding the handle to simulate opening a door, and measuring the surface area of paint transferred to a sheet of paper. This value was 0.001268 m^2 and was compared with data on average human hand size from the US Environmental Protection Agency's Exposure Factors Handbook to verify that the value was plausible [43]. It was assumed that the hand–fomite contact area was constant for both fomites used in the scenarios: door handles and bed rails.

To obtain estimates of finger pad area (*af*), a study by Dandekar *et al.* was used which found that the average width of an adult index finger ranged from 16 to 20 mm, and a square finger pad area was assumed for a resulting uniform distribution that ranged from 2.56×10^{-4} m^2 to 4×10^{-4} m^2 [44]. To calculate virus particles on finger pads, the concentration of particles on the hand was multiplied by the ratio between hand touch area and finger pad (*r.fh*).

Transfer efficiencies

To calculate the proportion of faeces transferred to hands during wiping (*t.fh*), previous data on mass of faeces transfer to hands and an estimate of 98.2 g mean faeces per event were used to model this value as a BetaPert distribution with a mode of 10^{-6}, minimum of 10^{-10} and maximum of 10^{-3} [32]. Transfer of norovirus particles from hands to fomites and from fomites to hands was estimated using data from Julian *et al.* [45]. Reported transfer values for the surrogate virus MS2 were chosen due to its similar size and surface structure compared with norovirus. In addition, values reported for recently washed hands were used under the assumption that recent handwashing is likely to have occurred in a hospital setting. However, it was assumed that surfaces were not disinfected, and that neither ESWs nor ill patients used any PPE or handwashing between source event and exposure.

Transfer of norovirus particles from hands to fomites (*t.hs*) was modelled as a normal distribution with a mean of 0.15 (SD 0.16). A normal distribution with a mean of 0.26 (SD 0.19) was used for transfer of virus from fomites to hands (*t.sh*). Transfer of virus particles from finger pad to mouth (*t.hm*) was modelled with a normal distribution with a mean of 0.34 (SD 0.25), as reported previously [46–49].

Fomite and hand concentrations

Equations to calculate total amount of norovirus deposited on touch area were:

Vomit in bathroom, immediately post flush

$$s.as * c.va * ah \quad \text{[c.vs1]}$$

Diarrhoea in bathroom, immediately post flush

$$(s.as * c.va * ah) + (m.f * t.fh * c.vf * t.hs) \quad \text{[c.vs2]}$$

Vomit in patient room, immediately after event

$$s.as * c.va * ah \quad \text{[c.vs3]}$$

Diarrhoea in patient room, immediately after event

$$m.f * t.fh * c.vf * t.hs \quad \text{[c.vs4]}$$

For vomit scenarios, it was assumed that no transfer of virus from hands to fomites occurred.

Next, the concentration of virus particles on a fomite was used to calculate the number of virus particles on an ESW's hands after contact with the fomite:

$$c.vs * t.sh \quad \text{[c.vh]}$$

In addition, the number of particles that an ESW would transfer to secondary contact fomites was calculated:

$$c.vh * t.hs \quad \text{[c.v2s]}$$

Dose

Norovirus dose was considered as the amount of virus particles transferred from a finger pad to an ESW's mouth or secondary contact's mouth. The authors chose to model ingestion as a result of finger pad contact because previous work has found that 90% of touches to mucous membranes are by fingers [50]. It was assumed that transfer from finger pad to mouth resulted in ingestion of 100% of transferred particles. This resulted in the following dose equations:

Norovirus dose for ESW

$$c.vh * r.hf * t.hm \quad \text{[D]}$$

Norovirus dose for secondary fomite contact

$$c.vs2 * t.sh * r.hf * t.hm \quad \text{[D2]}$$

Dose response and endpoint

The endpoint for this risk model was norovirus illness in either an ESW or a secondary-fomite-exposed contact. A previously reported dose–response model was chosen to estimate probability of illness, with parameters n and r that take the values 2.55×10^{-3} and 0.086, respectively [12,51]:

$$p(ill/dose) = 1 - (1 + n * dose)^{\wedge} - r \quad \text{[risk.ill]}$$

Modelling approach

A 2D MC approach was used to model variability and uncertainty of inputs. The R package 'mc2d' was used to build and evaluate all models and the final code is provided in the online supplementary material [29]. Distributions for inputs were designated as variable (inherent population differences), uncertain (result of imperfect knowledge) or both. In some instances, fixed values were used for inputs. For inputs that were defined by variability alone, distributions were sampled at random to provide final estimates. For inputs defined by both uncertainty and variability, uncertain parameters were sampled at random and then these uncertain parameters were carried into a variability distribution and sampled at random.

All parameters, distributions and sources used in these models are shown in Table II. The final model resulted in risk estimates that reflect variability across the population, conditional upon uncertainty parameters. Model iterations were set at 1000 for the variability dimension and 100 for uncertainty. The stability of measurements was tested by running 10 different seed values (3, 43, 69, 323, 402, 1111, 58423, 438035, 7915116, 26022021). For each scenario, the different seed values resulted in SD intervals for median risk estimates that overlapped with results from the final seed value (323) used in the model (data not shown).

Sensitivity analysis

A sensitivity analysis was conducted by calculating the Spearman rank correlation coefficient (SRC) for each baseline parameter. SRC values can range from −1 to 1, and an |SRC| value closer to 1 indicates higher correlation and higher importance of the factor on the final risk value. Baseline parameters were defined as values that were supplied to the model as distributions and not calculated within the model. Values that were supplied as constants were excluded from sensitivity analyses. Baseline parameters evaluated in the final sensitivity analysis were concentration of norovirus in vomit (*c.vv*); volume of vomit (*vl.v*); concentration of norovirus in faeces (*c.vf*); mass of faeces (*m.f*); proportion of virus aerosolized from vomit and toilet flush (*p.av*, *p.af*); settling rate of norovirus from air (*s.as*); time between source event and fomite contact (*delt.t*); area of finger pad (*af*); and transfer of norovirus from faeces to hands (*t.fh*), from hands to fomites (*t.hs*), from fomites to hands (*t.sh*), and from hands to mouth (*t.hm*).

Results

Median risk estimates and 95% credible intervals for a single fomite contact are shown in Table III and Figure 2. Risk values for diarrhoeal scenarios were the same when compared across the two locations (bathroom and patient room). Median risk of norovirus infection for diarrhoea source events was calculated to be 1:3 for ESWs whether they were exposed in the bathroom or in the patient room, and 1:4 for secondary contacts. Risk of norovirus infection from a single fomite contact was lower for vomit scenarios, and ranged from a median risk of ESW illness of 1:23,928 following a vomit event in a patient's bathroom to a median risk for a secondary contact of 1:252,185 after a vomit event in a patient room. Median estimated ESW infection risks from a vomit event were higher in the scenarios where the source

event occurred in the bathroom compared with the patient room. Secondary transmission of norovirus by ESWs resulted in an 80% decrease in median infection risk for vomit scenarios and a 32% decrease for diarrhoeal scenarios.

SRC values from the sensitivity analysis for baseline parameters in each scenario are shown in Figure 3. The settling rate of norovirus from air to fomites had the lowest SRC in all scenarios where it was included, indicating low correlation with the final risk estimate. In vomit scenarios, the starting concentration of norovirus in vomit had much less effect on the final risk model, compared with the impact of concentration of norovirus in diarrhoea. In the vomit in bathroom scenario, the value for aerosolization from vomit had equivalent SRC values compared with aerosolization from toilet flush. In all scenarios with an aerosolization component (vomit in bathroom/room, diarrhoea in bathroom), the time between event and fomite contact was strongly correlated with the final risk model (SRC >0.8). All modelled transfer rates had SRC ≥0.8, and in diarrhoeal scenarios, transfer rates were highly correlated with final risk values (SRC = 0.99).

Discussion

This study used QMRA to model the risk of norovirus infection in ESWs from fomite contacts in healthcare settings. For a single fomite contact in a room where a patient experienced a diarrhoeal event, the risk of norovirus infection for an unprotected ESW was as high as 1:3. This risk represents a plausible upper bound from a worst-case scenario where no viral die-off occurs, and where neither the patient nor ESW use PPE, disinfectants or hand hygiene. This risk estimate is in line with previous reports of norovirus attack rates in staff of 30–90% in hospital outbreaks, although reported attack rates from norovirus outbreaks are expected to be higher than those estimated in this work as this study did not include all possible norovirus exposure routes and did not account for multiple exposure events [5,9,13]. For scenarios where an ill patient vomited without a diarrhoeal event, the estimated risk to ESWs from fomite contact dropped four orders of magnitude to 1:23,928 for an in-bathroom event and 1:51,504 for an in-room event. The observed decrease in the risk of fomite transmission of norovirus in vomit scenarios, compared with diarrhoeal scenarios, is mainly due to the reduced initial viral load reported in vomit compared with diarrhoea.

It is important to emphasize that the risk estimates presented in this study represent the risk of infection from a single fomite contact. They do not include aerosol transmission and should not be interpreted as absolute infection risk posed after interacting with a norovirus-positive patient. The authors chose to focus specifically on fomites to understand the role that fomites play in transmission of norovirus to ESWs, as data indicate that, during cleaning, an ESW will touch nine fomites per room visit on average, and up to 34 fomites per room visit [26].

This study found much higher risks of infection from diarrhoeal events compared with vomit events. The impact of norovirus transmission from diarrhoeal events compared with vomit events remains unclear in the literature. One study reported that vomiting patients infect twice the number of people as those who are not vomiting, while patients with diarrhoea

infect 1.4 times the number of people as those without diarrhoea [52]. However, another study indicated that diarrhoea is almost ubiquitous in index cases for norovirus outbreaks, and that diarrhoea had a higher association with outbreak development than vomiting [53]. One reason for the low estimated risks from vomit source events is the focus on the fomite route of transfer and the exclusion of any inhalation of viral particles. Previous literature has indicated that aerosol transmission of norovirus is driven primarily by vomit events [54]. Aerosolization of norovirus is thought to be highest immediately after vomiting, and the presence of norovirus in the air ablates 3–6 h after the vomit event [54]. The present study demonstrates that fomite exposure to norovirus may represent an important exposure pathway, independent of inhalation. Additionally, risk for fomite exposure may increase with time post event as further settling can occur, which can significantly affect long-term exposure to the virus. It is important to note, however, that the exclusion of inhalation transmission in this model precludes direct translation between estimated risks and data from documented norovirus outbreaks.

For secondary norovirus infection from contact with a contaminated fomite, risks were 32% lower than ESW infection risks in diarrhoeal scenarios and 80% lower than ESW infection risks in vomit scenarios. This aligns with evidence that proximity to patients with norovirus increases the likelihood of viral spread in health care [55]. The infection risks faced by secondary fomite contacts are affirmed by previous work that shows spread of viral surrogates between multiple rooms and fomites in a hospital [23].

The sensitivity analyses indicated that the concentration of norovirus in vomit was much less important in relevant scenarios compared with the concentration of norovirus in diarrhoea. This is likely due to less overall transmission of norovirus via fomites in vomit scenarios compared with diarrhoeal scenarios. This study also found that a longer time between the vomit source event and ESW fomite contact led to a greater risk. This occurred because norovirus inactivation was not included, as inactivation is minimal for the 24-h time frame of these models [56–58], and a longer time period results in higher settling of virus on to fomites. However, the finding that a longer time since the source event leads to greater risk of infection is not likely to occur in scenarios that include inhalation transmission of norovirus, which was not considered in this work. This study also found that transfer rates were a significant driver of infection risk in the models, especially in diarrhoeal scenarios. This serves to underscore the need for more robust and specific data on transfer of norovirus across different fomites. Lack of these data contribute to a significant gap in understanding of healthcare-associated outbreaks of norovirus [57].

This work was subject to several limitations. Scenarios only accounted for norovirus transfer from patients with active symptoms. Asymptomatic shedding of norovirus is well documented, but the literature indicates that symptomatic patients remain the main drivers of transmission in healthcare settings [59]. The authors chose not to examine the role of gowns and gloves used routinely for cleaning the rooms of patients with known norovirus infection, and also did not account for hand hygiene or disinfectant usage. This was done to capture a worst-case scenario of transmission and to elucidate the potential transmission dynamics of norovirus. Further, there is evidence of poor hand hygiene compliance among ESWs after fomite contact [26]. Evidence also exists that norovirus persists on fomites

even after cleaning, and transmission of norovirus in healthcare settings is likely to occur even in the presence of interventions [14,22]. The decision to focus on unprotected ESWs yields a risk of norovirus infection that represents an upper bound and serves to illustrate the importance of proper interventions, including disinfection and hand hygiene. Future work should include risk reduction measurements for intervention efforts. Additionally, only direct ingestion from fomite transmission was considered in the models; no other infection routes for norovirus, including inhalation, were examined. The resulting risk estimates should not be interpreted as absolute possible risk of norovirus infection to ESWs, but rather as evidence of the importance that fomites play in norovirus transmission and as a framework to evaluate possible exposure pathways and intervention opportunities in healthcare facilities.

This model did not assume any additive effect from previous norovirus source events, although this is likely to occur in real-world settings. The authors did not differentiate between different norovirus genogroups and genotypes. It was assumed that all norovirus produced in the source event was infectious, when it is likely that a subset of measured noroviruses are non-infectious particles [60]. The recent development of a cell culture model for norovirus represents an interesting future opportunity to further refine these data [3,61].

This study aimed to model only those parameter values that reflected data on norovirus behaviour or that of applicable viral surrogates, as has been done in prior norovirus QMRAs [51]. However, the authors were unable to model all parameters using norovirus-specific values. In particular, transfer rates for faeces to hands during wiping were challenging to find in existing literature. Previous studies have addressed this by assuming a mass transfer of 0.1 g [62], while another study assumed a BetaPert distribution with a mode of 1×10^{-3} g faeces, a minimum of 1×10^{-8} g and a maximum of 1×10^{-1} g, although the rationale for these values was not provided [32]. When reported mass transfer from these studies was combined with the assumed mean stool mass of 98.2 g, the mode percentage transfer of faeces to hands was 0.1% [62] and 0.001% [32]. A third study measured transfer of the surrogate feline calicivirus to hands from artificial faeces, and found that approximately 3% of virus was transferred in a high-contact scenario [63]. These values result in faeces to hand transfer rates that span four orders of magnitude. Similar variability was observed in the literature on aerosolization of particles from a toilet flush. The generation rates calculated in this work were similar to that found by Johnson *et al.* [37] who estimated approximately 0.072 droplets forming for every 100 million particles, resulting in an aerosolization proportion of 1.3×10^{-10}. However, another study estimated that between 33.3% and 60% of the total particles in a toilet rise above the toilet seat during a flush event [36]. This results in possible aerosolization proportions from a toilet flush that differ by >10 orders of magnitude. These data gaps highlight crucial areas for future research.

In conclusion, this study adds to the growing body of literature that points to fomite transmission as a significant pathway in the spread of norovirus [56,58,64,65]. It showed that it is feasible for a single fomite contact to lead to a sizable risk of norovirus infection in ESWs, and that ESWs are able to transmit a significant amount of norovirus to secondary fomites, initiating further infections. This work highlights the importance of studying ESWs as a unique population in healthcare settings, independent of clinical staff. ESWs are

essential to infection control in the healthcare setting, yet understanding of the specific health risks they face remains minimal. This QMRA shows that ESWs likely face important occupational health risks from fomite-mediated norovirus infections. Fomite cleaning and disinfection procedures should be designed in collaboration with ESWs, and future work must focus on quantifying and mitigating any infection-related risks posed to these essential workers.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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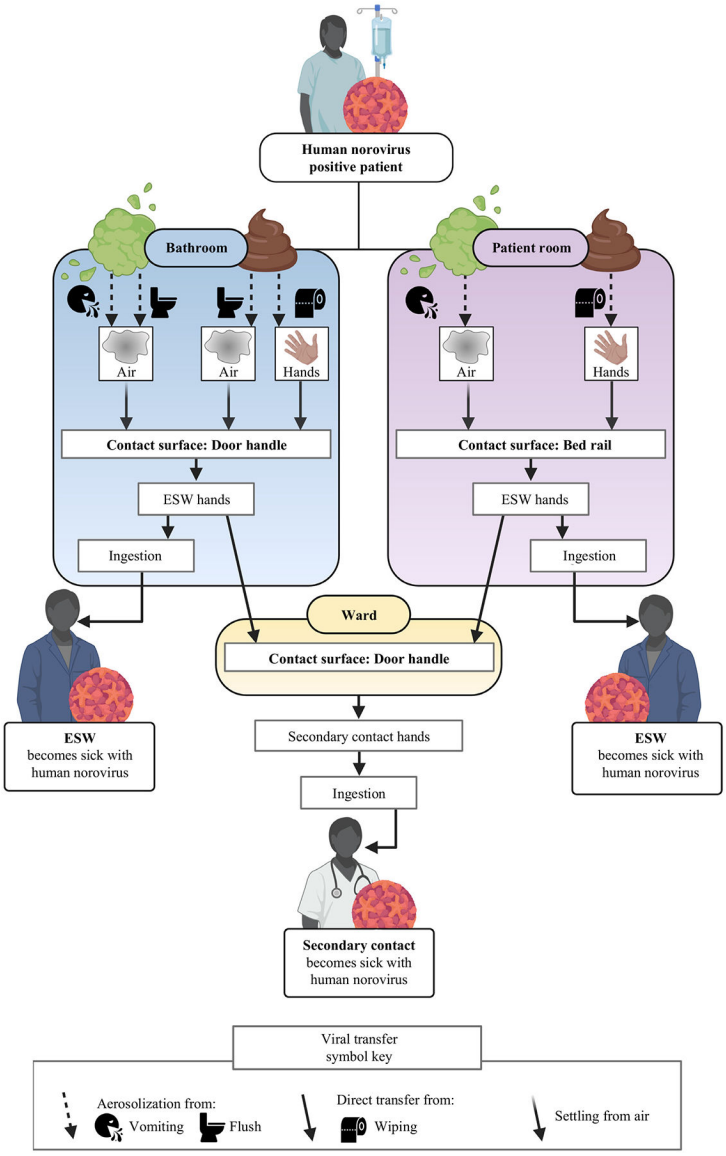

Figure 1.

Conceptual exposure model of norovirus fomite exposure for environmental service workers (ESWs) and secondary contacts in each scenario, created with BioRender.com.

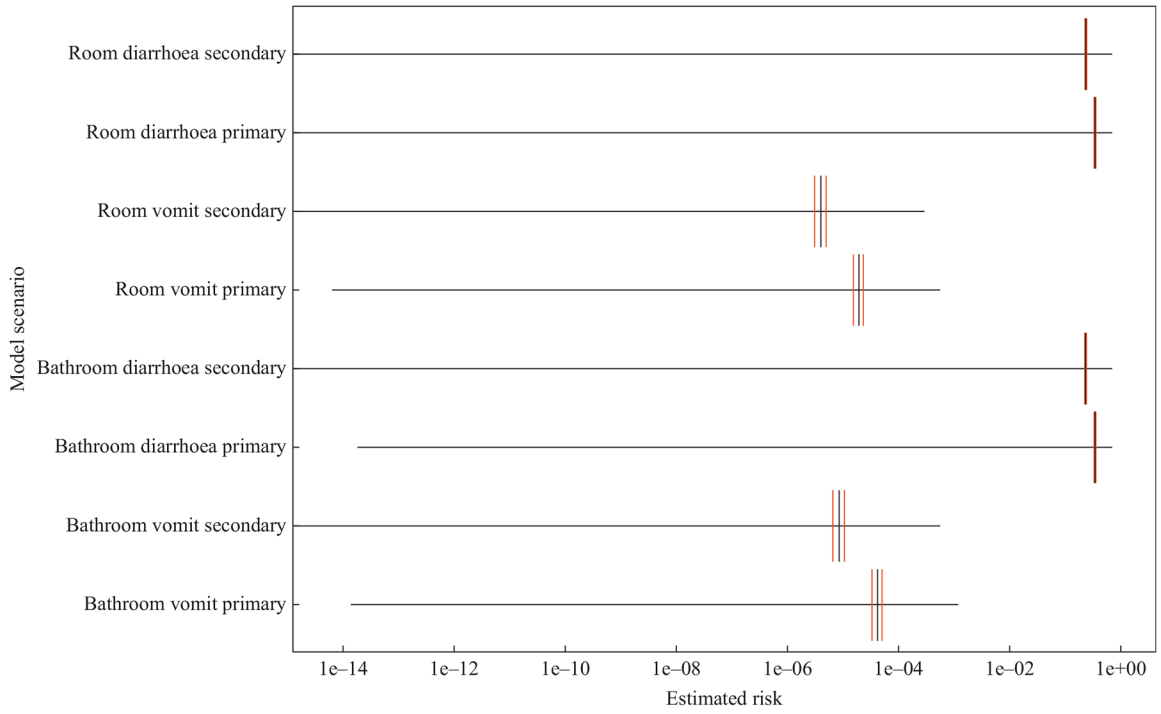

Figure 2.

Estimated median, 95% confidence interval (red lines), minimum and maximum risk of norovirus illness from single source event.

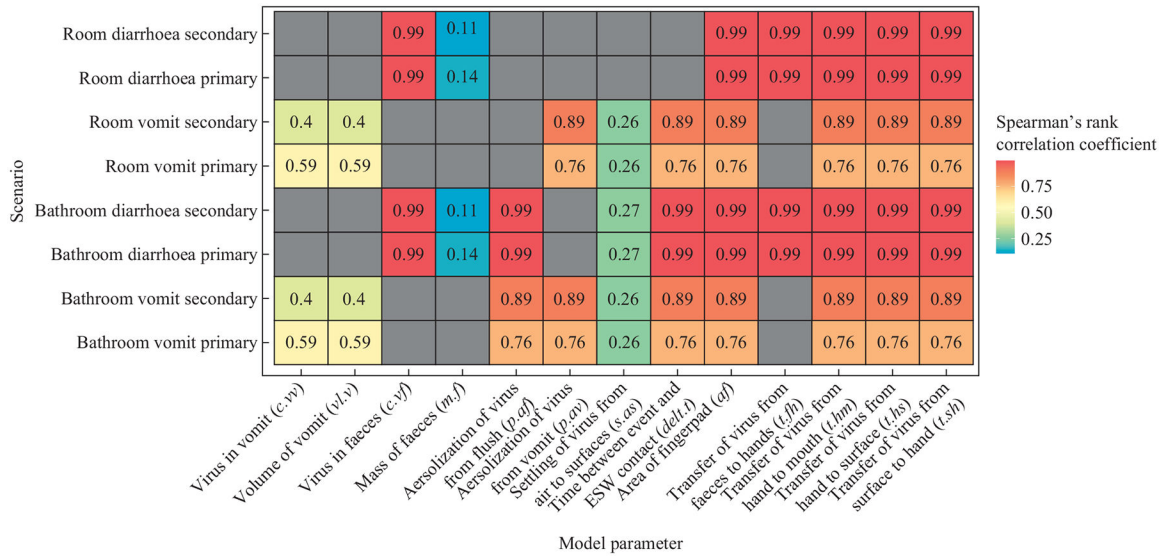

Figure 3.

Spearman's rank correlation coefficient for baseline parameters in each modelled scenario. ESW, environmental service worker.

Table I

Major assumptions of the models

Assumptions related to norovirus symptoms and shedding
All particles released from ill patient are initially infectious
Source of virus is either bowel movements or vomit events – the patient does not shed passively
All persons exposed to norovirus particles are susceptible to infection
Assumptions related to aerosolization of viral particles
Bathroom door is closed during vomit/diarrhoeal event
Air mixing between rooms does not occur
Air distribution of viral particles post flush and/or post vomit event is uniform and occurs within 1 min
No aerosolization occurs directly from a diarrhoeal event
Assumptions related to fomite deposition of viral particles
Virus in air does not settle on hands; it only deposits on fomites
Deposition of virus from air to fomites occurs uniformly across the room
Assumptions related to transfer of viral particles from contact
Environmental service worker cleans room once per day
Virus does not inactivate in air or on fomites
Transfer of virus from the hands of ill patient does not occur during vomit events and only results from wiping after a diarrhoeal event
Transfer of viruses between fomites and hands is one-directional, moving from fomite with high concentration to fomite with low concentration
Distribution of virus is constant across touch area
Viral transfer rates are the same for each fomite
Assumptions related to calculated fomite concentration and dose of norovirus
Contact between hand and mouth is done via a single finger pad
Only one hand-to-face contact event occurs before handwashing
Dose to lip is considered ingestion

Table II

Model parameter distributions and sources

Description	Units	Distribution (values)[a]	Truncation	Type[b]	Source
Virus shedding					
m.vv = mean concentration of norovirus in vomitus	Genome equivalents/mL	Normal (1.6×10^5, 4.5×10^4)	Min = 2200 Max = 1.2×10^7	U	[30]
c.vv = starting concentration of norovirus in vomitus	Genome equivalents/mL	BetaPert (2.2×10^3, *m.vv*, 1.2×10^7)	Min = 0	VU	[31,32]
m.vlv = mean volume of vomit in one event	mL	Normal (845, 226.7)	Min = 500 Max = 1000	U	[33,66]
vl.v = volume of vomit in one event	mL	BetaPert [50 (*m.vlv*/2), 800]	Min = 0	VU	[30,33,66]
m.mf = mean mass of faeces from one event	g	Normal (98.2, 17.08)	Min = 14.6 Max = 449.4	U	[34,35]
m.f = mass of faeces from one event	g	BetaPert (14.6, *m.mf*, 449.4)	Min = 0	VU	[34,35]
c.vf = starting concentration of norovirus in faeces	Genome equivalents/g	BetaPert (10^4, 10^8, 10^{10})	Min = 10^4 Max = 1.6×10^{12}	V	[11,32,67]
Aerosolization					
p.av = proportion of virus particles released into air during vomit event	Proportion	Low titre (*c.vv* <10^{10}): normal (2.8×10^{-5}, 1×10^{-5}) High titre (*c.vv* ≥ 10^{10}): normal (1.3×10^{-4}, 1×10^{-4})	Min = 0 Max = 1	V	[33,68]
p.af = proportion of virus particles released into air during flush	Proportion	Normal (2.42×10^{-7}, 6.91×10^{-8})	Min = 0 Max = 1	V	[38]
vlr = volume of room	m^3	Bathroom: 12.4 Patient room: 26.5	-	V	[39–41]
c.va = concentration of virus in room air	Virus particles/m^3	Vomit in bathroom, immediately post flush [*c.vv* * *vl.v* *(*p.av* + *p.af*)]/*vlr* Diarrhoea in bathroom, immediately post flush (*c.vf* * *m.f* * *p.af*)/*vlr* Vomit in patient room, immediately after event (*c.vv* * *vl.v* * *p.av*)/*vlr* Diarrhoea in patient room, immediately after event 0	-	V	Calculated
Fomite deposition					
delt.t = time between event and HCW fomite contact	min	Uniform (1,1440)	-	V	Assumed
s.as = virus settling rate from air to fomite during total time since event	Proportion/m^2	≤30 min since event: 0.031 * *delt.t* >30 min since event: 1	-	V	[38]
ah = area of hand that contacts fomite	m^2	0.001268	-	0	This study
af = area of finger pad	m^2	Uniform (0.000256, 0.0004)	-	V	[44,48]
r.fh = ratio between area of hand touch area and finger pad	m^2	*af*/*ah*	-	-	Calculated
Transfer efficiencies					

Description	Units	Distribution (values)[a]	Truncation	Type[b]	Source
t.fh = proportion of faeces transferred to hands during wiping	Proportion	BetaPert (10^{-10}, 10^{-6}, 10^{-3})	Min = 0 Max = 1	V	[32,62,63]
t.hs = transfer of virus particles from hand to fomite	Proportion	Normal (0.15, 0.16)	Min = 0 Max = 1	V	[45]
t.sh = transfer of virus particles from fomite to hand	Proportion	Normal (0.26, 0.19)	Min = 0 Max = 1	V	[45]
t.hm = transfer between hand and mouth	Proportion	Normal (0.34, 0.25)	Min = 0 Max = 1	V	[46–49]
Fomite and hand concentrations					
c.vs = concentration of virus particles on contact area at time t	Virus particles	Vomit in bathroom, immediately post flush *s.as * c.va * ah* Diarrhoea in bathroom, immediately post flush (*s.as * c.va* ah*) + (*m.f * t.fh * c.vf * t.hs*) Vomit in patient room, immediately after event *s.as * c.va * ah* Diarrhoea in patient room, immediately after event *m.f* t.fh * c.vf * t.hs*	-	-	Calculated
c.vh = concentration of virus on ESW hands	Virus particles	*c.vs * t.sh*	-	-	Calculated
c.v2s = concentration of norovirus on secondary contact fomite	Virus particles	*c.vh * t.hs*	-	-	Calculated
Dose					
D = dose of virus ingested from primary fomite contact	Particles	*c.vh * r.hf * t.hm*	-	-	Calculated
D2 = dose of virus ingested from secondary fomite contact	Virus particles	*c.vs2 * t.sh * r.hf * t.hm*	-	-	Calculated
Dose response					
n = dose–response constant	-	0.00255	-	-	[12,51]
r = dose–response constant	-	0.086	-	-	[12,51]
risk.ill = risk of illness as a function of dose	Risk	1 − (1 + *n**dose))^-*r*	-	-	[12,51]

[a] Distribution types and values: normal (mean, standard deviation); BetaPert (minimum, mode, maximum); uniform (minimum, maximum); empirical (observed values).

[b] U indicates uncertainty distribution, V indicates variability distribution, U + V is distributions with both an uncertainty and variability component.

Table III

Human norovirus infection risk for each scenario

Scenario	Median risk of illness from single source event	95% confidence interval for risk of illness	Minimum risk estimate	Maximum risk estimate	Median number of touch events required to cause one illness
Bathroom vomit primary	4.18×10^{-5}	(3.32×10^{-5} to 5.02×10^{-5})	1.40×10^{-14}	1.15×10^{-3}	23,928
Bathroom vomit secondary	8.54×10^{-6}	(6.50×10^{-6} to 1.07×10^{-5})	$<1 \times 10^{-16}$	6.02×10^{-4}	117,104
Bathroom diarrhoea primary	3.46×10^{-1}	(3.38×10^{-1} to 3.53×10^{-1})	1.80×10^{-14}	6.84×10^{-1}	3
Bathroom diarrhoea secondary	2.35×10^{-1}	(2.27×10^{-1} to 2.41×10^{-1})	$<1 \times 10^{-16}$	6.69×10^{-1}	4
Room vomit primary	1.94×10^{-5}	(1.54×10^{-5} to 2.33×10^{-5})	6.27×10^{-15}	5.36×10^{-4}	51,504
Room vomit secondary	3.97×10^{-6}	(3.02×10^{-6} to 4.97×10^{-6})	$<1 \times 10^{-16}$	2.80×10^{-4}	252,185
Room diarrhoea primary	3.46×10^{-1}	(3.38×10^{-1} to 3.53×10^{-1})	1.79×10^{-14}	6.84×10^{-1}	3
Room diarrhoea secondary	2.35×10^{-1}	(2.27×10^{-1} to 2.41×10^{-1})	$<1 \times 10^{-16}$	6.69×10^{-1}	4