

REVIEW ARTICLE

## Toilet hygiene—review and research needs

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### Abstract

The goal of good toilet hygiene is minimizing the potential for pathogen transmission. Control of odours is also socially important and believed to be a societal measure of cleanliness. Understanding the need for good cleaning and disinfecting is even more important today considering the potential spread of emerging pathogens such as SARS-CoV-2 virus. While the flush toilet was a major advancement in achieving these objectives, exposure to pathogens can occur from failure to clean and disinfect areas within a restroom, as well as poor hand hygiene. The build-up of biofilm within a toilet bowl/urinal including sink can result in the persistence of pathogens and odours. During flushing, pathogens can be ejected from the toilet bowl/urinal/sink and be transmitted by inhalation and contaminated fomites. Use of automatic toilet bowl cleaners can reduce the number of microorganisms ejected during a flush. *Salmonella* bacteria can colonize the underside of the rim of toilets and persist up to 50 days. Pathogenic enteric bacteria appear in greater numbers in the biofilm found in toilets than in the water. Source tracking of bacteria in homes has demonstrated that during cleaning enteric bacteria are transferred from the toilet to the bathroom sinks and that these same bacteria colonize cleaning tools used in the restroom. Quantitative microbial risk assessment has shown that significant risks exist from both aerosols and fomites in restrooms. Cleaning with soaps and detergents without the use of disinfectants in public restrooms may spread bacteria and viruses throughout the restroom. Odours in restrooms are largely controlled by ventilation and flushing volume in toilet/urinals. However, this results in increased energy and water usage. Contamination of both the air and surfaces in restrooms is well documented. Better quantification of the risks of infection are needed as this will help determine what interventions will minimize these risks.

### Introduction

The invention of the flush toilet over 150 years ago had a major impact of toilet waste disposal within the household. It eliminated the need to transport faecal wastes out of the household by container handling. It also provided plumbed water increasing the ease of hand washing (Aiello *et al.* 2007). While the flush toilet was a major advancement in achieving these objectives, exposure to pathogens can still occur from failure to clean and disinfectant areas within a restroom, as well as poor hand hygiene (Aiello

*et al.* 2007). Outbreaks of infectious agents-associated diseases from toilets have been documented, largely from improper cleaning and disinfection of restroom facilities (Palmer *et al.* 1981; Rajaratnam *et al.* 1992). However, evidence indicates that contamination of areas outside of the toilet bowl/urinal can occur from aerosols generated from flushing resulting in potential transmission by inhalation and indirectly by fomite contamination (Gerba *et al.* 1975). Fomite contamination can also occur directly by hand and body contact with high touch/contact areas within a restroom (Boone and Gerba 2007).

This review summarizes the current state of knowledge on microbial (pathogens) contamination and odours in restrooms and approaches to better define these risks and potential interventions to reduce these risks.

### Occurrence and concentration of pathogens in stools and urine

Many enteric pathogens are found in high titres in stools and therefore in toilets after defecation, particularly during episodes of acute diarrhoea. Some enteric pathogens such as noroviruses are also found in high concentrations in vomitus and can also thus contaminate toilets including other areas indoor via a person vomiting. An infected person can shed up to  $10^{11}$  colony forming units (CFU) of *Salmonella* (Thomson 1954) and *Shigella* per stool (Newsom 1972). Persons infected with enteric viruses may shed  $10^{10}$ – $10^{12}$  virus per gram of faeces (Table 1). During a bout of acute diarrhoea, there is often splashing that may contaminate the bowl sides and the recess under the toilet bowl rim (Barker and Bloomfield 2000). After flushing, the bacteria and viruses may be dispersed onto the external parts of the toilet such as the seat, the handle, and to other bathroom surfaces (Newsom 1972; Gerba *et al.* 1975). Bacteria generally do not survive well under conditions of desiccation; however, Newsom (1972) demonstrated the survival of *Salmonella* on surfaces for up to 9 days, *Escherichia coli* for up to 8 days, and *Shigella* for up to 5 days in faeces dried onto toilet seats.

Bacteria and viruses may also be present in the urine during infection (Table 2). Infectious viruses causing insect-borne encephalitis have been documented, but other viruses such as smallpox and adenoviruses, SARS-CoV-2 virus have also been detected in the urine (Sinclair *et al.*

**Table 1** Concentration for pathogens and faecal bacteria in stools

Microorganism	Concentration g <sup>-1</sup> ml <sup>-1</sup>	Reference
Coliforms	$10^7$ – $10^9$	Haas <i>et al.</i> (2014)
Faecal coliforms	$10^6$ – $10^9$	Haas <i>et al.</i> (2014)
<i>Escherichia coli</i>		
<i>Salmonella</i>	$10^4$ – $10^{10}$	Haas <i>et al.</i> (2014)
<i>Campylobacter jejuni</i>		
<i>E. coli</i> 0157:H7		
<i>Shigella</i>	$10^5$ – $10^9$	Haas <i>et al.</i> (2014)
Enterovirus	$10^3$ – $10^8$	Pepper <i>et al.</i> (2014)
Hepatitis A	$10^8$	Pepper <i>et al.</i> (2014)
Rotavirus	$10^{10}$ – $10^{12}$	Pepper <i>et al.</i> (2014)
Norovirus	$10^{10}$ – $10^{12}$	Pepper <i>et al.</i> (2014)
Adenovirus	$10^{11}$	Haas <i>et al.</i> (2014)
SARS-CoV-2	$10^1$ – $10^3$	Xiao <i>et al.</i> (2020)
<i>Cryptosporidium</i>	$10^6$ – $10^7$	Pepper <i>et al.</i> (2014)
<i>Giardia</i>	$10^1$ – $10^6$	GWPP (2020)
<i>Ascaris</i>	$10^4$ – $10^5$	Haas <i>et al.</i> (2014)

**Table 2** Pathogens (infectious) excreted in the urine

Microorganism	Reference
<i>Coxiella burnetii</i>	Sinclair <i>et al.</i> (2008)
Viral encephalitis viruses	Sinclair <i>et al.</i> (2008)
Nipah virus	Sinclair <i>et al.</i> (2008)
Rabies virus	Sinclair <i>et al.</i> (2008)
Smallpox virus	Sinclair <i>et al.</i> (2008)
Cytomegalovirus	Paduch (2007)
SARS-CoV	Xu <i>et al.</i> (2005)
SARS-CoV-2	Sun <i>et al.</i> (2020)
Adenovirus	Echavarria <i>et al.</i> (1998)
Measles (rubella)	Gresser and Katz (1960); Paduch (2007)
<i>Salmonella typhi</i> ; <i>Salmonella paratyphi</i>	Crowdy 1984
<i>Leptospira interorgans</i>	Crowdy 1984

2008; Sun *et al.* 2020). Variola major, the virus which causes smallpox, is released for up to 19 days after infection at concentrations of  $10^2$ – $10^5$  ml<sup>-1</sup> of urine (Sinclair *et al.* 2008). In many infections, the greatest concentrations are released during the first few days after the initial infection. *Brucella abortus* is excreted in concentrations as high as  $10^6$  ml<sup>-1</sup> of urine for up to 12 weeks (Sinclair *et al.* 2008). SARS-CoV-2 concentrations are low, but infectious virus detected in urine 12 days after onset of disease (Sun *et al.* 2020). Significant amounts of pathogens can be released in the urine considering people excrete from 700 to 2000 ml of urine per day (Crowdy 1984).

During cases of viral gastroenteritis, up to  $10^{12}$  virus particles have been detected per gram of stool. The average human adult stool weighs approximately 100 g (range 100–400 g per day for an adult) and contains about  $10^{12}$  bacteria (Gerba *et al.* 1975), including  $10^{10}$  coliforms (Thomson 1954). Therefore, the toilet bowl could potentially contain up to  $10^{14}$  virus particles (Barker and Jones 2005).

Many pathogens that are transmitted via the faecal-oral route are believed to have low infectivity such as *Shigella*, *Campylobacter*, *E. coli* O157:H7, rotavirus, and norovirus (LeBaron *et al.* 1990). Ingestion of as low as 1–10 noroviruses are needed to cause an infection (Teunis *et al.* 2008). The infectivity of *Salmonella* is generally thought to be higher; however, depending upon the bacterial strain it can be as low as 10–100 CFU (Hockin *et al.* 1989; Barker and Bloomfield 2000).

### Outbreaks associated with flush toilets

There have been reported outbreaks that provide evidence to support the toilet as a source of infection for enteric pathogens (Table 3). In a norovirus outbreak aboard an

**Table 3** Outbreaks associated with toilets

Pathogen	Source location	Reference
Norovirus	Airplane	Hutson <i>et al.</i> (2002); Widdowson <i>et al.</i> (2005)
Hepatitis A virus	Cruise ship	Ho <i>et al.</i> (1989)
	Primary school	Leoni <i>et al.</i> (1998)
SARS-CoV	Apartment building	McKinney <i>et al.</i> (2006)
SARS-CoV-2	Hospital	Ding <i>et al.</i> (2021)
Salmonella	University	Palmer <i>et al.</i> (1981)
Shigella	Hospital	Korpela <i>et al.</i> (1995)
MRSA	Children's Hospital	Giannini <i>et al.</i> (2009)
Legionella pneumophila	Hospital	Couturier <i>et al.</i> (2020)

international flight, the onset of illness was consistent with a point-source outbreak but also suggested secondary transmission among passengers that subsequently embarked on a cruise. The index cases had experienced episodes of vomiting and diarrhoea in the airplane lavatories. Many of the passengers that became ill following this flight were sitting near this block of bathrooms and presumably were somewhat likely to have used these facilities (Holmes and Simmons 2008). In a norovirus outbreak among the cabin staff on an airplane, visibly unsoiled toilets were reported to be the likely source of infection for passengers (Hutson *et al.* 2002; Widdowson *et al.* 2005). In a cruise ship outbreak of norovirus, the use of a specific toilet contaminated by vomit during a highly attended event was associated with increased odds of illness (Chimonas *et al.* 2008). During a similar cruise ship outbreak, the risk of gastroenteritis for those sharing restroom facilities was twice that of those who had a private bathroom. The risk of infection was also related to the number of people sharing the communal restroom; passengers sharing restrooms with more than 60 people had an attack rate twice that of passengers sharing restrooms with 20 or fewer people (Ho *et al.* 1989).

In an outbreak of hepatitis A virus (HAV) within a middle school, the use of a particular toilet for defecation was linked to the source of infection. This toilet had been used by the index case during a bout of diarrhoea (Rajaratnam *et al.* 1992). In a similar outbreak in a primary school in Italy, the critical exposure of the school children was deemed to have taken place in the boys' toilet. The toilet had been contaminated by a child with an HAV infection acquired via consuming infected clams outside of the school. None of the primary school girls or the secondary school children, all who used separate

toilets, were infected (Leoni *et al.* 1998). An outbreak of SARS-CoV in an apartment building with improper restroom ventilation was believed to be involved in its spread via aerosols generated from toilet flushing (McKinney *et al.* 2006).

The spread of enteric bacterial pathogens has also been linked to toilets. In a *Salmonella* Typhimurium outbreak in university students, contamination of toilets was believed to be the cause of secondary cases (Palmer *et al.* 1981). Outbreaks of shigellosis in schools linked to faecal material in and around toilets have also been reported (Hutchison 1956). In a nosocomial (hospital) outbreak caused by *Shigella sonnei*, sharing of a toilet with the index patient was believed to be the most probable source of infection (Korpela *et al.* 1995).

Non-enteric bacteria can also potentially be spread by toilets. Giannini *et al.* (2009) reported that the use of alcohol wipes on toilet seats resulted in a 50-fold reduction in MRSA infections in a children's hospital. Genome sequencing data suggested that two cases of *Legionella pneumophila* in a hospital in France were aerosolization and inhalation of the bacteria from toilets was the cause as this was believed to be the only significant route of exposure (Couturier *et al.* 2020).

### Contamination of flush toilets by pathogens

During an outbreak of shigellosis, contamination of toilet seats was observed following flushing of liquid faeces containing greater than  $10^7$  CFU per g of *S. sonnei*. *Shigella sonnei* was present on 11 of 34 toilet seats tested (Hutchison 1956). In a hospital ward, faecal bacteria grew on 27% of settle plates exposed near ward toilets, suggesting aerosolization of the toilet water. *Escherichia coli* was also isolated from flush handles, toilet seats, and the underside of toilet lids (Newsom 1972). Similar to the results found by other researchers (Barker and Bloomfield 2000; Barker and Jones 2005), the number of bacteria in the toilet was reduced by 2-log<sub>10</sub> by a single flush (Newsom 1972).

Barker and Bloomfield (2000) found *Salmonella enteritidis* in the homes of four recovering Salmonellosis patients in persistent biofilms under the rim of the toilet bowl and in the scaly biofilm layer in the toilet bowl just below the water line. *Salmonella* was not isolated from the outside of the toilet bowl. In all cases, the serotype of the bacteria isolated from the environment was identical to that isolated from the patient. In all four homes, toilet cleaning products were used on a daily to weekly basis. Despite this, *Salmonella* bacteria were isolated from the toilet for weeks, particularly from the biofilms. In one home, *S. enteritidis* was found 4 weeks after the patient's diarrhoea had ceased. Pitts *et al.* (1998) have found

biofilms in the toilet bowl below the water line measuring up to 20  $\mu\text{m}$  thick.

### Contamination of flush toilets by use of bidet

Iyo *et al.* (2016) found *Pseudomonas aeruginosa* on 2% of bidet toilets in a restroom on a university campus. Warm-water tanks used for bidet toilets also showed a decreased concentration of residual chlorine and thereby an increase in heterotrophic and viable bacteria concentrations. Bacterial colonization of bidet toilets in hospitals was found in 87% of bidet nozzle surfaces and 94% of spray waters—where *Pseudomonas* spp. were isolated from 11 nozzle surfaces (5.7%) and 17 spray water (8.8%) (Tsunoda *et al.* 2019). In a university affiliated hospital, 3.4% ( $n = 10$ ) of bidet nozzles contained isolated of *Staphylococcus aureus*. Methicillin resistant *S. aureus* was found on one bidet nozzle and one toilet seat (Katsuse *et al.* 2017). Consistent users of bidet toilets who are female report higher rates of abnormal vaginal *E. coli* colonization and pre-term birth (Kim *et al.* 2019).

### Aerosols produced by flush toilets

The potential for aerosolization of pathogens during toilet/urine flushing has received a lot of attention, especially in concerning emerging pathogens such as SARS, Ebola, and *Clostridium difficile*. All the research indicates that significant aerosolization can occur resulting in potential transmission of pathogens by inhalation and via fomite contamination (Johnson *et al.* 2013a). The degree of aerosolization is dependent upon several factors listed in Table 4. While large droplets settle out within a few minutes, smaller may persist and continued to settle out on surfaces for 90 min (Best *et al.* 2012; Knowlton *et al.* 2018). Residual levels of microorganisms may also remain in the bowl after the initial flush, resulting in aerosolization of bacteria after repeated flushes (Gerba *et al.* 1975; Johnson *et al.* 2013a, 2017).

**Table 4** Factors that influence the aerosolization of microbes from toilet flushing

Design of toilet
Amount of water in bowl
Waste (and type) in the bowl
Water pressure
Biofilm
Automatic toilet bowl cleaner
Chlorine in the tap water
Volume of water used in a flush
Lid down

In a seeded toilet experiment (Barker and Bloomfield 2000), *Salmonella* could be isolated from the air, the toilet seat and lid following flushing of the toilet. In addition, the bacteria were released and could be found in subsequent flushing aerosols, but in incrementally decreasing numbers. After 6 days, the bacteria were no longer found in the water in the toilet bowl. Nevertheless, *Salmonella* was isolated from the biofilm below the water line in the bowl for up to 50 days (Barker and Bloomfield 2000). Barker and Jones (2005) observed similar results for environmental contamination caused by flushing a toilet seeded with *Serratia marcescens* and MS2 bacteriophage. The toilet water after flushing was reduced by approximately 2-log<sub>10</sub> of *Serratia* after 60 min. Similarly, bacterial counts in the air decreased from approximately 1300 to 500 and 128 CFU per  $\text{m}^3$  after subsequent toilet flushing. The bacterial contamination of external surfaces was greatest in areas closest to the seeded toilet bowl (i.e., the toilet seat).

Barker and Jones (2005) concluded that both the bacteria attached to the sidewalls of toilet bowl and those in the water contribute to the formation of aerosols. The bacterial numbers on the sidewalls and under the toilet bowl rim were not significantly decreased by multiple flushing and thus were probably the reservoir for continuing residual contamination of the toilet bowl water. Also, closing the toilet lid did little to prevent the release of bacteria into the air. In a similar study, closing the toilet lid was also found to be ineffective at reducing bacterial air counts (Bound and Atkinson 1966).

Darlow and Bale (1959) demonstrated that toilet water artificially contaminated with *Chromobacterium prodigiosum* produced a widely disseminated and persistent aerosol after flushing. The aerosol was not prevented by weak disinfectants or by closing the toilet lid during flushing. Gerba *et al.* (1975) found that large numbers of seeded bacteria and viruses remained in the toilet bowl following flushing and even continual flushing could not entirely remove them. In addition, both *E. coli* and MS2 bacteriophage were detected in droplets generated by flushing. Aerosols were found to persist for at least 12 min and could disseminate the microorganisms to surfaces throughout the bathroom. The particles in these aerosols were a size capable of being inhaled and of reaching the lower respiratory tract.

Gerba *et al.* (1975) found that bacteria and viruses seeded into the toilet bowl before flushing were ejected from the bowl during flushing and settled on surfaces throughout the restroom for up to 2 h. The number ejected was directly related to the number in the bowl. Aerosolized droplets derived from the toilet bowl are generated even when the toilet bowl is covered and can lead to accumulation of aerosolized particles over time

(Aithinne *et al.* 2019; Schreck *et al.* 2021). Modern flush toilets generate significant amounts of aerosols less than 2  $\mu\text{m}$  diameter (95%) and fomite droplets greater than 5  $\mu\text{m}$  (>99%) (Johnson *et al.* 2013b).

Air quality in hospital wards are largely regulated through mixed ventilation systems, however in the case of an outbreak the use of displaced ventilation systems achieves better microbial air quality when not influenced by the proximity of restroom exhaust vents (Yin *et al.* 2009). Verani *et al.* (2014) detected adenoviruses in 67% of air samples collected from the restrooms in offices and 55% in air from hospitals. Toque teno virus was present in 15–18% of the air samples collected in these environments. Transmission of SARS-CoV-2 and norovirus is influenced by airflow direction produced by air-conditioning units in restaurants and classrooms, respectively (Zhang *et al.* 2017; Lu *et al.* 2020). The finding of infectious SARS-CoV-2 in urine has created speculation that urinal flushing could be involved in the transmission of this virus (Sun *et al.* 2020). In studying droplets generated by urinal flushing Wang *et al.* (2020) found that droplet could reach a height to be inhaled by the average adult, making it useful to wear mask while using public rooms particularly during the ongoing SARS-CoV-2 pandemic.

### Surface fomite contamination

Mendes and Lynch (1976) concluded that faecal bacteria are found on bathroom surfaces in sufficient numbers to allow the transfer of infection via the hands. Scott and Bloomfield (1985) found opportunistic pathogens such as *P. aeruginosa* and *E. coli* as well as other Enterobacteria frequently on sites such as the toilet seat and handle in addition to the toilet bowl, suggesting transfer from the toilet. They deemed that the extent of this transfer was limited under normal conditions and therefore the risk of infection from such transfer was low (Bloomfield and Scott 1997). Nevertheless, under atypical conditions, such as when a person is experiencing bouts of acute diarrhoea with watery stools containing a high titre of the enteric pathogen, this risk may be greatly elevated.

Several studies have reported the contamination of hospital patient toilets shared by patients. Amoah *et al.* (2020) studying community toilets ( $n = 8$ ) in South Africa found that 53–63% of the restroom surfaces were contaminated with SARS-CoV-2 by qPCR and droplet digital PCR. The concentration of virus ranged from 25.9 to 132.69 genome copies per  $\text{cm}^2$ , the highest of that being the toilet seat and the cistern flush handle. SARS-CoV-2 virus has been recovered from toilet seat, bathroom door handle, and sinks in bathrooms housing patients with SARS-CoV-2 infections (Ding *et al.* 2021). In a study of office and hospital restrooms human

adenoviruses were detected on ~70% of the surfaces, torque teno virus on 9% of restrooms surfaces, 44% of hospital restroom surfaces. Norovirus was only detected once on a surface in a hospital restroom. The common occurrence of adenovirus probably results from prolonged shedding from the respiratory and urinary tracts as well as faeces. Torque teno virus infection has not been found to be associated with any specific illness but leads to life-long shedding of the virus without illness but is shed in the faeces and believed to be transmitted by the faecal oral route (Griffin *et al.* 2008). Noroviruses are commonly detected on public restroom surfaces after outbreaks. In a study of outbreaks of norovirus outbreaks in restrooms over 2 years in restrooms norovirus was detected in 8% of 630 samples (8.6%; Kimoto *et al.* 2016). In a study of norovirus outbreaks on cruise ships norovirus was detected on 56.3% of the toilet seats in concentrations range from  $10^{3.1}$  to  $10^{7.4}$  genome copies (an average of  $10^{5.5}$ ). CrAssphage, which is an indicator of human faecal contamination, was found on 68.5% of the toilet seats (Park *et al.* 2020).

### Impact of cleaning on spread of enteric pathogens in restrooms

The spread of enteric pathogens from the toilet via aerosols is not the only route for contamination of bathroom surfaces. Sponges and clothes are routinely used in US households to clean kitchen and toilet surfaces (Enriquez *et al.* 1997) Faecal coliforms were identified from 12 different surfaces in 20 residential bathrooms after the homeowners' regular cleaning regimen (Bright and Gerba, Home Survey of Household Restrooms and Effects of Disinfectants, University of Arizona, Tucson, Arizona, unpublished). The cleaning tool (e.g., sponge, cloth) was also collected and examined for contamination by coliforms and faecal coliforms. Coliforms were found in all 20 homes and faecal coliforms were detected on bathroom surfaces in eight of the 20 homes. Bacterial isolate identification was determined using API 20E strips (BioMerieux, Inc., Hazelwood, MO) as *E. coli*, *E. hermannii*, *Klebsiella pneumoniae*, or *Klebsiella oxytoca*. Further typing of each isolate was accomplished via biochemical fingerprinting (Kühn 1985), ribotyping, and serotyping (*E. coli* only). In seven of the eight homes with identified faecal coliforms, identical strains were isolated from either the toilet itself (toilet bowl, toilet seat bottom, flush handle) or the cleaning tool and at least two other surfaces (up to eight surfaces) in the bathroom (e.g., sink bowl, sink drain, sink countertop, sink faucet handle, shower/bath drain, shower/bath surface, floor 12 inches in front of the toilet). In the eighth home, an identical strain was isolated from the cleaning tool and one other surface. The results of this study are highly

suggestive of the cleaning tool being the instrument of transfer from the toilet to other surfaces in the bathroom.

### Risk assessment of infections from restroom use

Quantitative microbial risk assessment (QMRA) is an approach that can be used to assess the risks of infectious disease transmission by water, food, air, and inanimate objects (Haas *et al.* 2014). It has been used to develop guidelines for setting standards for microbial risks of infection for drinking water by the United States Environmental Protection Agency and regulatory agencies of several countries (Haas *et al.* 2014). Carducci *et al.* (2016) used it to study the risk of infection from aerosols of adenoviruses in different occupational settings including wastewater treatment plants, solid waste landfills and toilets in healthcare and office buildings. Virological monitoring showed the presence of adenoviruses in the air of all these settings. The results of QMRA showed that the risks of infection from airborne transmission was the greatest from the aerosols present in public restrooms. They found the number of genome copies in office buildings averaged  $10^{4.81} \text{ m}^{-3}$  and was greater in four room hospital patient rooms ( $10^{7.9}$  genome copies per  $\text{m}^3$ ). Amoah *et al.* (2020) estimated the risk of infection from SARS-CoV-2 from touching various surfaces in public restrooms. They used qPCR to quantify the number of genome copies of the viruses on surfaces. They calculated that the greatest risk of infection ( $4.3 \times 10^{-2}$  to  $6.0 \times 10^{-4}$ ) is when a person uses the toilet once in a day, increasing to  $1.0 \times 10^{-1}$  to  $1.4 \times 10^{-3}$  if they used the toilet three times in a day. Risks of infection for a one-time exposure are considered significant if less than  $1 \times 10^{-6}$  (Signor and Ashbolt 2009).

### Interventions to reduce risk of transmission

In addition to improper cleaning procedures, many environmental surfaces in the bathroom do not receive adequate cleaning or, in some locations, no cleaning at all, even in hospital environments. This includes 'high risk' objects such as the toilet area handholds, bathroom door knobs, and light switches (Carling *et al.* 2008).

Hypochlorite cleaners have been shown to be effective at reducing the levels of faecal bacteria on bathroom surfaces (Rusin *et al.* 1998). Nevertheless, Barker and Bloomfield (2000) found that *Salmonella* persisted in toilet biofilms for long periods even with disinfection. The area under the toilet rim was particularly difficult to disinfect, even when using cleaners with bottles designed to deliver product to this problematic area. Pitts *et al.* (2001) found that bacteria were able to form biofilms in toilets, even in the presence of continuous  $9 \text{ mg l}^{-1}$  chlorine and up to  $27 \text{ mg l}^{-1}$  free chlorine.

Scott and Bloomfield (1985) determined that continuous release system disinfectants were more efficient at reducing contamination levels of the toilet itself (e.g., water, toilet bowl, toilet bowl rim) than daily disinfection or daily cleaning. In another study, automatic toilet bowl cleaners that did not contain any disinfectant, but rather varying levels of surfactants were found to reduce the number of bacteria ejected from the bowl in droplets or aerosols. The cleaner with the highest surfactant concentration was the most effective at limiting aerosols (Yahya *et al.* 1992).

Although enteric viruses cannot grow in biofilms, they may persist for long periods in the toilet environment and can be difficult to remove via normal decontamination or cleaning procedures. Noroviruses are very stable at room temperature requiring almost 17 h at room temperature for a 99.9% decrease in titre at room temperature (Duizer *et al.* 2004) and chlorine (Barker *et al.* 2004). Multiple outbreaks of norovirus have occurred following environmental contamination with the virus, despite numerous efforts to clean the contaminated surfaces with detergents (Cheesbrough *et al.* 1997; Barker *et al.* 2004; Jones *et al.* 2007).

### Malodours

It is believed that the worst indoor air quality occurs in restrooms (Qiuchen 2018). Malodours in toilets are believed to be associated with butyric acid, p-cresol, and sulphur compounds mainly hydrogen sulfide, methyl sulfide, monosulfide, disulfide and trisulfide (Sharma *et al.* 2020). In urinals, ammonia and amines are important odour producers (Perry and Schroeder 1963; Troccaz *et al.* 2013). The volume of flushing water in urinals is important in the control of odours from urine, and any residual urine leads to its degradation by bacteria and the production of ammonia (Hashemi and Han 2017). Thus, low flush water urinals designed to save water may result in greater generation of odours.

Adequate ventilation of a restroom is believed the most important factor in odour control in restrooms (Qiuchen 2018; Kimura *et al.* 2019). For this reason,  $\text{CO}_2$  concentration in restroom has been shown to be a good measure of malodours in restrooms (Qiuchen 2018). Unfortunately, enhanced ventilation for restrooms for odour control can put a significant demand on energy use (Kimura *et al.* 2019).

Sharma *et al.* (2020) found that daily spray of toilets/urinals with 1.0% sodium hydroxide and hydrogen peroxide (0.05%) could control the breakdown of malodor producing bacteria and oxidizing odorous compounds at household and public restrooms.

## Future research and directions to improve toilet hygiene

Toilet hygiene is important in the control of both enteric and respiratory pathogens-associated illness both in public toilets and the home. Soap and detergents alone if not used properly cause cross contamination throughout a restroom. Use of disinfectants is critical to preventing movement of enteric microorganisms throughout the restroom. Pathogen contamination of both the air and surfaces in restrooms is well documented. Better quantification of the risks of infection using QMRA are needed as this will help determine what interventions will minimize these risks. Colonization of biofilms and hard to clean area (the rim under the toilet) by pathogenic enteric bacteria such as *Salmonella* appear to be a problem, which has not been completely resolved. There is also a need for improved methods for disinfecting problem areas such as the region below the toilet rim and the area just below the water line where the formation of biofilms has been observed. New methods such as the use of a disinfectants that would adhere to the side of the toilet bowl for a longer period or ones with a longer residual effect or new methods for the physical removal of the area by cleaning should be investigated, such as improved scrubbing device or a chemical method (Wang *et al.* 2019; Krishnan 2020). These approaches would aid in the reduction of odours and aerosols. Also, this area as a source of odours appears not to have been studied in detail or at least reported in the scientific literature. These biofilms may also be areas where enteric viruses could persist for long periods of time. Cross contamination in homes during cleaning of the toilet and restroom also appears to be a problem, needs investigational work to provide mitigation guidelines for consumers.

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## Conflict of Interest

Drs Julie McKinney and M. Khalid Ijaz are engaged in R&D at Reckitt Benckiser LLC.

## Author contributions

Sarah E. Abney: Conceptualization (equal); investigation (lead); visualization (lead); writing—original draft (equal); writing—review and editing (lead). Kelly Bright: Writing—review and editing (equal); visualization

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