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Review article

Lifting the lid on toilet plume aerosol: A literature review with suggestions for future research

David L. Johnson PhD [a,*], Kenneth R. Mead PhD [b], Robert A. Lynch PhD [a], Deborah V.L. Hirst PhD [b]

[a] Department of Occupational and Environmental Health, University of Oklahoma College of Public Health, Oklahoma City, OK
[b] Division of Applied Research and Technology, National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, Cincinnati, OH

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Background: The potential risks associated with "toilet plume" aerosols produced by flush toilets is a subject of continuing study. This review examines the evidence regarding toilet plume bioaerosol generation and infectious disease transmission.

Methods: The peer-reviewed scientific literature was searched to identify articles related to aerosol production during toilet flushing, as well as epidemiologic studies examining the potential role of toilets in infectious disease outbreaks.

Results: The studies demonstrate that potentially infectious aerosols may be produced in substantial quantities during flushing. Aerosolization can continue through multiple flushes to expose subsequent toilet users. Some of the aerosols desiccate to become droplet nuclei and remain adrift in the air currents. However, no studies have yet clearly demonstrated or refuted toilet plume-related disease transmission, and the significance of the risk remains largely uncharacterized.

Conclusion: Research suggests that toilet plume could play a contributory role in the transmission of infectious diseases. Additional research in multiple areas is warranted to assess the risks posed by toilet plume, especially within health care facilities.

An association between inhalable bioaerosols produced from disturbed sewage and the transmission of infectious disease has been proposed for over 100 years. However, little study has been devoted to characterizing the potential risks posed by the "toilet plume" aerosols created by toilet flushing. We summarize the related scientific literature and identify gaps in the knowledge base, addressing the following questions: (1) "Do flush toilets produce potentially infectious aerosols?" (2) "Do toilet plume aerosols pose a risk for the spread of infectious disease?" and (3) "What future research is needed to further characterize the risks of exposure to toilet plume aerosols within a health care setting?"

* Address correspondence to David L. Johnson, PhD, Department of Occupational and Environmental Health, University of Oklahoma College of Public Health, P.O. Box 26901, Room 431, Oklahoma City, OK 73126-0901.

E-mail address: David-Johnson@ouhsc.edu (D.L. Johnson).

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DO FLUSH TOILETS PRODUCE POTENTIALLY INFECTIOUS AEROSOLS?

The potential for airborne transmission of sewage-related infectious disease was demonstrated by Horrocks over 100 years ago[1] when he cultured airborne microorganisms from sewage drain systems and also detected airborne transport from one hospital building to another via the sewer drains. Similar results were seen by others including Andrewes.[2]

Bioaerosol production during toilet flushing was first reported in the 1950s by Jessen,[3] who "seeded" several types of toilets with *Serratia marcescens* (then termed *Bacillus prodigiosus*) and measured bioaerosols produced by flushing. Agar-filled "settle plates" caught bioaerosols that fell out of the air because of gravity, and a Bourdillon slit impactor[4] collected air samples. Cistern-fed, gravity-flow toilets and a mains-fed pressure-valve toilet were examined. In addition to colonies found on the floor-based settle plates, microbes were still being captured from the air 8 minutes after the flush, indicating collection of "droplet nuclei" bioaerosols. Droplet nuclei are the tiny particles that remain after the water in a droplet evaporates. They have negligible settling velocity and will

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float with natural air currents.[5] Jessen observed that the amount of bioaerosol increased with increasing flush energy.[3]

Darlow and Bale[6] seeded a "wash-down" type toilet with *S marcescens* and sampled air above the toilet with liquid impingers and a Bourdillon impactor. A wash-down toilet releases the flush water from the toilet rim where it flows down the bowl walls and washes the waste into the S-shaped exit trapway.[7] Bioaerosol was detected in samples collected above the toilet 5 to 7 minutes after the flush, indicating droplet nuclei bioaerosol. Despite over 99% reductions in bowl water microbial concentrations with each flush, air samples indicated only 50% to 60% bioaerosol reductions. They concluded that this was at least partially attributable to a reduction in the number of bacteria per droplet rather than a reduction in the number of droplets containing bacteria because both a multi-organism droplet and a single-organism droplet would appear as 1 colony when deposited on an impactor agar plate.

Siphonic toilets, which feature a submerged jet that propels the waste into the trapway to initiate a siphon action that clears the waste, have generally replaced wash-down models. Bound and Atkinson[8] found that the higher energy siphonic toilet produced approximately 1/14th as much bioaerosol as the wash-down design for the same flush volume. Newsom also demonstrated higher bioaerosol production with higher flush energy when he compared high and low cistern toilets seeded with homogenized feces or suspensions of various bacteria.[9]

Gerba et al[10] seeded a siphonic gravity-flow toilet with *Escherichia coli* and sequentially placed 3 arrays of settle plates on the floor around the toilet, with each set exposed for 2 hours. For the first sample set (0-2 hours), cultured bacteria were predominantly from plates near the toilet, whereas, in later sample sets (2-4 and 4-6 hours), the positive plates were more randomly distributed around the room. This was consistent with an initial deposition of large droplets close to the toilet immediately after the flush, followed by dispersion and mixing of the droplet nuclei into the air with delayed deposition throughout the room. The *E coli* bioaerosol remained airborne and viable for at least 4 to 6 hours postflush.

Barker and Bloomfield[11] seeded a gravity-flow toilet with *Salmonella enteritidis* PT4 and collected surface wipe and air samples after flushing. They observed contamination of the toilet seat and the underside of the lid and also cultured *Salmonella* from the air sample. They detected *Salmonella* in the bowl water after 12 days and in biofilm below the bowl waterline for 50 days after seeding, which suggested a possible role of biofilm as a long-term reservoir and active source of pathogenic organisms in the bowl water.

Barker and Jones seeded a toilet with *S marcescens* or MS2 bacteriophage.[12] Air samples were collected in front of and above the toilet seat with the toilet seat lid open. They also exposed settle plates at 5 locations around the toilet, including 2 above and behind the seat. Bioaerosols were present up to 60 minutes after flushing, and all settle plates were positive for all test conditions and sampling locations, demonstrating droplet nuclei bioaerosol. They also examined toilet bowl clearance and bioaerosol production during sequential flushes without reseeding, with results similar to those of Darlow and Bale,[6] Newsom,[9] and Gerba et al[10] in that bioaerosol concentration did not decrease in proportion to bowl water concentration.

Recently, Best et al[13] flushed a toilet seeded with fecal suspensions of *Clostridium difficile*. Settle plates were placed near the toilet and air was sampled at seat height, flush handle height, and midway in-between, with the toilet lid both up and down. Settle plates showed widespread dissemination of large droplets with the lid up but not with the lid down. *C difficile* was recovered from air sampled at heights up to 25 cm above the toilet seat and up to 90 minutes after flushing, at concentrations 12-fold greater with the lid up than with the lid down. They concluded that lidless conventional toilets increase the risk of *C difficile* environmental contamination and thus discouraged their use. In the United States, however, this would contradict current Uniform Plumbing Code specifications regarding toilet seat design and the installation of toilet seat lids on health care and other public facility "water closets"[14] as well as similar requirements for gap-front seats without cover for water closets in the US Veterans Administration specifications often cited for health care facility design.[15]

It may be concluded from the above that flush toilets produce substantial quantities of toilet plume aerosol capable of entraining microorganisms at least as large as bacteria, that sufficiently small microbe-laden droplets will evaporate to form droplet nuclei bioaerosols small enough to be inhaled deep into the lung, and that these bioaerosols may remain viable in the air for extended periods and travel with air currents. Production of these bioaerosols during multiple flushes after contamination suggests a long-term potential for a contaminated toilet to be an infectious bioaerosol generator.

DOES TOILET PLUME POSE A RISK FOR THE SPREAD OF INFECTIOUS DISEASE?

Contact transmission risk because of surface contamination by flush droplets

A number of studies have demonstrated the contamination of toilet seats and lids, the surrounding floors, and the nearby surfaces by toilet flush aerosols.[3,6,9,10,12,13,16] Because both the vomit and feces of infected persons may contain extremely high pathogen concentrations, eg, 10^5 to 10^9 *Shigella*,[17] 10^4 to 10^8 *Salmonella*,[17] and 10^8 to 10^9 norovirus[18] per gram of stool and at least 10^6 norovirus per milliliter of vomit,[19] some fraction of the aerosol droplets produced during toilet flushing may be expected to contain microbes.[20]

A critical determinant of the infection risk posed by a deposited pathogen will be the organism's ability to survive on a surface.[21] Many pathogens, including *Shigella*, *E coli*, *C difficile*, severe acute respiratory syndrome (SARS) coronavirus, and norovirus can survive on surfaces for weeks or even months.[22] These pathogens can also be present in vomit or stools of infected persons.

In 1956, Hutchinson associated the transmission of Sonne dysentery with *Shigella* contamination on toilet seats,[23] and a number of subsequent field studies have detected contamination on toilet seats and surrounding surfaces with fecal organisms.[9,11,24,25] Thorough cleaning and disinfection of environmental surfaces in health care facilities is a foundational component of infection control programs,[26] and disinfection is particularly important because many studies have shown that microbial surface contamination (including *C difficile*, vancomycin-resistant *Enterococcus*, and methicillin-resistant *Staphylococcus aureus* [MRSA]) may persist even after cleaning.[27-29] The limits of environmental cleaning in preventing spread of viral disease are apparent with acute gastroenteritis (AGE). AGE is frequently caused by norovirus, and the diarrhea and vomiting typically associated with AGE as well as the high viral loads in both stools and vomit suggest a likely toilet role in disease transmission. Environmental contamination has been shown to be a major source of AGE infection on ships,[30-35] including during sequential voyages of a cruise ship in spite of aggressive sanitation efforts and a documented history of good Centers for Disease Control and Prevention (CDC) Vessel Sanitation Program inspection scores.[34] This may be due in part to the ability of toilets to continue generating contaminated toilet plume during multiple flushes after original contamination as well as the apparent resistance of norovirus[36] and perhaps other viruses to cleaning and disinfection. Gerba et al observed that MS2 bacteriophage and poliovirus were not completely cleared from a toilet even after 7 flushes and that scrubbing with or without addition of

a surfactant to the water was only minimally effective in eliminating these residual organisms.[10] The manner in which cleaning and disinfection is performed is also important in ensuring complete disinfection of surfaces, especially when surfaces are heavily contaminated.[37]

Airborne transmission risk because of toilet plume droplet nuclei

Although Chapin dismissed the airborne route as unimportant in his 1912 review of infectious disease transmission,[38] by the 1960s it was accepted that droplet nuclei microbial aerosols were important in the transmission of many infectious diseases in both indoor and outdoor environments.[39,40] We now understand that whether pathogenic droplet nuclei bioaerosols actually cause infection and disease will depend on numerous factors including the organism's viability under existing environmental conditions, the size and chemical composition of the droplet nuclei matrix, the number of organisms inhaled and their virulence, and the exposed person's immune status.[40]

A number of diseases are known or suspected to be transmissible by the airborne route. However, most are either not transmissible from human to human or are not present in feces or vomit and so are not relevant to the present discussion. Toilet-related pathogens that are of interest include those causing gastroenteritis, and a number of gastroenteritis-causing bacteria, protozoa, and especially viruses will be shed in stool and vomit. *Giardia* and *Cryptosporidium* protozoa may be present in feces, have low infective dose, and are stable in the environment, but aerosolization of oocyst-containing droplet nuclei has not been documented.[41] Gram-negative bacteria, with the notable exception of *Legionella*, are susceptible to drying and do not usually spread by the airborne route. The gram-positive MRSA is an airborne nosocomial infection concern,[42] but the potential for toilet plume bioaerosols to cause nosocomial MRSA infection has not yet been assessed.

Mycobacterium tuberculosis (TB) appears to be most efficiently transmitted via droplet nuclei[43] and is an occupational hazard to health care workers as well as a nosocomial infection hazard to patients.[44] TB affects primarily the lungs, but TB bacilli can also be swallowed in sputum to infect the gastrointestinal (GI) tract.[45] At least 21% of 2009 US TB cases involved this "extrapulmonary" infection including infection of the GI tract,[46] although perhaps less than 5% of all TB cases involve lower GI tract infection.[45] The bacilli can survive intestinal transit to be shed in stool,[47,48] and, because one of the symptoms of GI TB is diarrhea,[45] there appears to be a possibility of aerosolizing infectious TB droplet nuclei in toilet flush aerosol. *M tuberculosis* is a lipid-rich, hydrophobic bacterium, and hydrophobic bacteria have been shown to concentrate on the surface of aqueous suspensions[49,50] and to be aerosolized with even slight disturbance of liquid surfaces.[51,52]

The most significant toilet plume airborne infection risks are likely to be due to viruses, and perhaps the most significant of these is norovirus. Norovirus accounts for 73% to 95% of nonbacterial gastroenteritis outbreaks and half of all gastroenteritis outbreaks, worldwide.[53] It may also be transmitted in aerosol and has a low infectious dose.[54] It is shed both before and after—sometimes long after—the symptomatic phase of infection, is resistant to inactivation, and can persist on environmental surfaces for extended periods.[55-57] Diarrhea and vomiting are both common with norovirus AGE, so both the use of toilets by infected persons and the toilet disposal of feces or vomit by other persons could produce norovirus bioaerosols.

Another important viral pathogen is the SARS coronavirus (SARS CoV), which is known to be shed in both feces[58-60] and vomit.[60] A number of studies (discussed below) have suggested that it can be spread by the airborne route,[61-64] and, although not presently a common disease, it has demonstrated its potential for explosive spread and high mortality.

Novel influenza A virus H1N1 has also demonstrated some important epidemiologic features that indicate a potential for airborne transmission via toilet plume. Seasonal influenza does not normally present with diarrhea or vomiting, but each had a prevalence of 25% in the first 642 US cases[65] and 17% and 22%, respectively, among the first 938 US cases[66] diagnosed during the 2009 pandemic H1N1 influenza outbreak. It has been measured in respirable-size aerosol in health care and other facilities,[67,68] has been detected in both stools and urine of H1N1 patients even in the absence of significant GI symptoms,[69] and has shown a potential for extended virus shedding in stool.[70] The presence of H1N1 in vomit seems likely, and, although no report documenting this was found, a suggestive study by Papenburg et al noted that the likelihood of H1N1 transmission in a household was greatest for patients with both diarrhea and vomiting.[71]

Epidemiologic studies of disease outbreaks possibly related to toilet plume

Widdowson et al investigated AGE among passengers on an 8-hour international flight[72] on which 8 of 14 flight crew members experienced vomiting and diarrhea. No episodes of diarrhea or vomiting occurred outside of a restroom, and there were no reported indications of restroom soiling with vomit or feces. Passengers who developed probable norovirus illness 18 to 60 hours after disembarkation were found to have visited a restroom significantly more often than noncases. The authors concluded that "inapparent environmental contamination" may have been an exposure source.

Ho et al studied an outbreak of viral AGE during a transatlantic passenger ship voyage.[73] They compared disease frequency in cabins varying from 1 to 4 occupants either having or not having a private bathroom and showed an increasing AGE risk with increasing number of occupants where a private bathroom was available as compared with cabins where one was not available. AGE incidence among those using communal bathrooms correlated significantly with the bathroom usage density. It was also shown that, in cabins with multiple occupants, the risk of a second person developing disease was higher in cabins where the first person had vomited, even though none of the subsequent cases either assisted the ill person or cleaned up the vomit. Presence in the room when vomiting actually occurred also did not appear to matter. The authors concluded that person-to-person and aerosol routes were the likely modes of transmission, with vomit being implicated as a source, and suggested that contact spread was facilitated by contaminated communal bathrooms.

Marks et al[74] studied restaurant diners who developed AGE following nonprojectile vomiting by the source diner and showed a pattern of decreasing attack rate with increasing distance from the source: 91% at the source's table; 71% and 56% at the 2 adjacent tables, respectively; and lower rates farther away. This study strongly implicated airborne norovirus transmission by vomit aerosol and thus the likelihood of airborne transmissibility by toilet plume aerosol contaminated with vomit.

Epidemiologic, experimental, and modeling studies of SARS are among the most compelling indicators of the potential for toilet plume to cause airborne disease transmission. A report on the 2003 SARS outbreak in Hong Kong's Amoy Gardens apartment complex concluded that exposure and disease propagation was likely due to virus-laden aerosols originating in the sanitary system.[64] The system was contaminated with SARS CoV when the index patient, who was suffering from diarrhea, visited one of the apartments and used the toilet. Sewer drain bioaerosol was believed to be drawn through dry floor drain U-tube traps into the bathrooms of other

apartments by bathroom exhaust fans, and some may have then been exhausted to the outside of the multistory building and carried upward to other apartments. Prevailing winds were thought to be responsible for carrying the infectious aerosol to nearby buildings where cases also occurred.[63,75] These studies suggest that SARS CoV droplet nuclei bioaerosols produced from contaminated sewage may have been highly infectious for significant periods and over long distances. Because the infectious waste, whether feces or vomit, is most concentrated in the toilet bowl and substantial quantities of aerosol are known to be produced during flushing, it might reasonably be expected that infectious SARS CoV droplet nuclei bioaerosol would also be produced during toilet flushing. To date, however, this has not been either experimentally or epidemiologically demonstrated.

No epidemiologic studies of the 2009 H1N1 pandemic have conclusively shown airborne transmission via droplet nuclei, and the primary transmission mode is still considered to be by contact with large particle respiratory droplets or contaminated surfaces.[76] Studies involving confined environment exposures in aircraft and buses concluded that the airborne route did not appear to be an important transmission mode,[77-80] in contrast to the high influenza transmission rate observed on an older aircraft with poor ventilation.[81] Although these epidemiologic studies have not demonstrated airborne infection, 2 recent environmental studies measured influenza A virus in respirable size aerosols collected in health care facilities, day care centers, and aircraft.[67,68] This finding, the shedding of influenza virus in stool and perhaps vomit, and the prevalence of diarrhea and vomiting in Novel H1N1 patients encourage exploration of the potential for toilet plume to contain infectious virus-containing droplet nuclei aerosols.

WHAT FUTURE RESEARCH IS NEEDED TO FURTHER CHARACTERIZE THE RISKS OF EXPOSURE TO TOILET PLUME?

Epidemiologic and laboratory studies provide evidence that potentially infectious aerosols may be produced during flushing of toilets contaminated with vomit or diarrhea from infected persons. Further assessment of the airborne infection risk requires research to address the following questions: (1) What are the physical properties of toilet plume? (2) How much toilet plume is produced, and which toilet design or operating characteristics most influence aerosol production? (3) How persistent are flush-generated droplet nuclei bioaerosols in the air? (4) Can infection be transmitted by toilet flush droplet nuclei bioaerosols, and, if so, what are the airborne concentrations and dispersion patterns of flush-generated pathogenic droplet nuclei bioaerosols in health care environments? (5) What interventions or practices might prove effective in controlling toilet plume bioaerosols?

SUMMARY AND CONCLUSIONS

Contaminated toilets have been clearly shown to produce large droplet and droplet nuclei bioaerosols during flushing, and research suggests that this toilet plume could play an important role in the transmission of infectious diseases for which the pathogen is shed in feces or vomit. The possible role of toilet plume in airborne transmission of norovirus, SARS, and pandemic influenza is of particular interest. Additional research is needed to assess the exposure risk posed by toilet flush bioaerosols in health care facilities.

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