The Microbiology of the Restroom

Why are public toilets, which offer many attractive features to pathogens intent on finding human hosts, so rarely identified as sources of gastrointestinal infections?

Bernard Dixon

Arriving on Earth for the first time, a visitor from Mars would soon recognize the terrestrial ubiquity of gastroenteritis, both trivial and lethal. He, she, or it would find that the human alimentary tract is a permanent battleground in which invading bacteria and viruses seek to establish themselves. Damaging tissues, fabricating and releasing toxins, they proliferate exponentially whenever possible before moving onwards in astronomical numbers to cause trouble in other susceptible hosts.

Equally clear would be the many different types of place where these microorganisms begin their journey. Some are relatively obvious—kitchens, restaurants, cafes, fast food outlets, street stalls, nurseries, old peoples’ care homes, farms and other areas where conditions can favour the dissemination of enteric pathogens. Other locations, to our Martian at least, are less obvious—swimming baths, hospitals and ambulances, for example.

Even assiduous reading of the microbiological literature, however, would reveal few if any food poisoning incidents traced to what an extraterrestrial might imagine to be one of the most plausible of all breeding grounds—the public toilet. Martian reasoning would run as follows. Whether in an airport, railway station, motorway service station or large store, a public convenience is a place where a never-ending succession of people deposit feces that may contain vast quantities of potentially dangerous bacteria or viruses. Although the great majority of these individuals are not clinically ill, some will have gastroenteritis and others will be carrying and shedding pathogens. Vomiting, generating aerosols, is not uncommon, either.

Even the most scrupulous individuals using public conveniences would find it virtually impossible not to pick up a few of their voided organisms and transfer them to sites such as door knobs. Many people are less fastidious about their personal hygiene. Children tend to be particularly careless. Some public toilets are cleaned less frequently and less thoroughly than others. Toilet flushes generate aerosols. Also, the temperature is ideal for gastrointestinal bugs to survive and proliferate. Even hand driers, installed specifically to combat problems of this sort, can disperse microorganisms on dust- and water-droplets. From a microbial perspective, conditions are quite delicious.

Our extraterrestrial visitor would notice something else. Although microbiologists have been investigating the translocation of bacteria, viruses, microfungi, and protozoa for many decades, they have rarely entered the toilet cubicle to do research. Using classical and more recently molecular biological techniques, investigators have built up a huge dossier of data concerning infectious hazards in other parts of the environment. They have assiduously swabbed chairs and tables, walls and floors, plated out the material recovered, and identified their microbial populations. Only last month, I read about two highly sophisticated studies using DNA probes to survey mobile phones and computer keyboards.

Yet the public toilet has been largely ignored. Until very recently. Thanks to Timo Hammer and his colleagues at the Institute for Hygiene and Biotechnology at Schloss Hohenstein, Boeningheim, in Germany, we now have the results of the first-ever rigorous investigation into the possible risks of infection within public lavatories. While their findings do not ring immediate alarm bells, they do confirm and to some degree
quantify the hazards. Based on their experimental model, the authors rightly suggest that further work is required to clarify the extent of the danger.

As the Boenningheim group points out, infectious intestinal disease is widespread (causing 17 million cases in the United Kingdom alone each year), and has considerable economic consequences. Domestic kitchens and bathrooms, serving as reservoirs of pathogens, are assumed to be the origins of most of these infections. Papers published during the last decade have extended our knowledge of places, such as chopping boards, that can harbor potentially harmful microbes. Yet we know very little concerning the identity, proliferation, and dissemination of microorganisms in public lavatories.

How to evaluate the risks? Timo Hammer and his coworkers could not build up a picture based on published conclusions from specific outbreaks centered on public toilets, simply because there are none. They might have generated a computer model, involving data on parameters such as numbers of people, excretion rates, and the survival of organisms outside the body. Instead, they developed an experimental model, using three materials to simulate the relevant inanimate surfaces: a plastic toilet brush, a Falcon tube representing a plastic door handle in size and material, and a piece of acrylic glass representing the grip on a water tap. Before each experiment, the investigators disinfected all of these objects and pairs of disposable latex gloves. The whole process was designed to simulate a possible chain of infection.

Firstly, an experimenter’s hand (wearing a sterilized glove) was contaminated with a test organism and allowed to dry, before the experimenter grasped a labelled area at the top of the brush. The doses were calculated on the basis of realistically reflecting the levels of pathogens found in feces. The glove was then removed and turned inside out before sterile saline solution was added to resuspend any organisms. Next, another experimenter, also wearing a disinfected glove, touched the toilet brush and then the Falcon tube, before the glove was removed as before. Finally, another person touched the Falcon tube followed by the acrylic glass. Culture tests on the saline washings from the gloves revealed the microbial load in each case. The test organisms were *Escherichia coli* K-12 and bacteriophage MS2 (as surrogates for enteropathogenic *E. coli* and norovirus, respectively, both having low infectious doses), plus *Candida albicans* and *Bacillus atrophaeus*. The entire protocol was rigorous, with adequate controls.

The results, reported in the *Journal of Applied Microbiology* (112:614, 2012), are to some degree predictable—but not entirely. They certainly indicate that successive titers of the organisms transferred in each step of the chain usually declined. For pathogens with a low infectious dose, however, the results suggested that a high probability remained of exposure to a potentially infectious dose. This might well occur in the case of noroviruses. And in some instances the touching hand removed even more microorganisms than remained on the initially contaminated surfaces.

Added to these findings, the researchers point to previous studies that have revealed the transfer of bacteria from porous surfaces (fabrics) to hands and thence to other fabrics. “Given the important role that textiles play in chains of infection,” they write, “it is easy to imagine that in a lavatory scenario microorganisms are further transmitted from the last object, the water tap, to a towel, thus turning the fabric into an infectious device.”

To return to our Martian visitor. He, she, or it, being aware of the inherent plausibility of the idea that human gastrointestinal infections can be purveyed through public toilets, would be fascinated by the complementary evidence coming from Boenningheim. Together, the hypothetical notion and the experimental findings reinforce the puzzle as to why virtually no cases or outbreaks have ever been traced to this type of source. The explanation must surely be that, as compared with a restaurant, food supplier, school, nursery or cruise liner, there is never a pattern of pathology that attracts attention and leads to an investigation.

In other words, it is inconceivable that public conveniences do not act as reservoirs of enteric pathogens and as foci of gastrointestinal disease. Over the years, we must all pick up some of the relevant organisms, but then move quickly onwards and never even suspect the cause of any symptomology that emerges a day or two later. What other explanation can there be?