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## Commentary

### Down the drain and back up a drain



In this issue the paper by Aranega-Bou *et al.* provides one more piece to the jigsaw of our understanding on drains and spread of organisms [1].

A survey conducted by the British Medical Journal in 2007 asked a group of experts and doctors what they consider the greatest medical advance since 1840. The answer was sanitation. First developed in ancient civilizations, used by the Romans but neglected by Britain until the industrial revolution, when the influx of population into cities lacking safe water and sanitation fuelled the spread of disease and associated stench. The need to segregate sewage from water intended for human use led to re-invention of sewer (drainage) systems.

Within buildings water supply and drainage systems interface – for example, in hand wash stations (HWSs), toilets and showers. Outbreaks related to hospital drainage systems have been reported over the years but in-depth knowledge of how this occurs has been lacking. By placing fluorescein in a drain, Hota *et al.* showed dispersal of drain contents over at least a metre when the outlet was run [2]. Much greater understanding came from Kotay *et al.* who showed the mere inoculation of siphon with bacteria was not enough to disperse bacteria when water from an outlet was run directly into the drain of an HWS [3]. However, placing a nutrient source down the drain (frequently occurs in clinical practice) led to biofilm growing up the drain at a rate of 2.54 cm/day. After seven days, this reached the drain sieve in the sink which it enveloped. Running the outlet then led to widespread dispersal of organisms. Additionally, using a series of HWSs draining into a common drain they were able to show bacteria introduced into the siphon of one HWS were able to reach the siphons of other HWS through the drain system.

The paper by Aranega-Bour *et al.* confirms the findings of Katay *et al.* and provides new information, primarily that rate of drainage is an important confounding variable. By artificially contaminating a siphon and running water directly into a drain dispersal only occurred when drainage was impaired. Next, using a siphon with biofilm extending up the drain dispersal occurred with fast drainage (as would be expected) but was exacerbated by slow drainage. With rear draining sinks, even with biofilm extending up the drain, dispersal was not an issue until drainage was impaired. In a very simple experiment we placed fluorescein dye down the rear drain of a sink in which drainage was impaired (but not reported by staff) and under

ultraviolet light using a video camera recorded running the outlet. Dye emerged out of the drain for up to nine times of running the outlet. Each time the outlet ran, the initial thrust of water would cause dye to regurgitate into the sink after which the main body of water would remove it. Occasionally, whilst the water was draining a jet of dye would be expelled against the flow from the drain into the sink.

UK guidance is for clinical handwash basins to have rear drains to minimize risk of dispersal of organisms which works until drainage becomes impaired [4]. How often is drainage impaired? Aranega-Bou *et al.* quote Breathnach *et al.* who, in an excellent investigation of an outbreak of multi-drug-resistant bacteria linked to drains, found an average of 391 notifications of blocked sinks, toilets or sluices per year in a hospital [5]. Perversely, rear draining sinks in our experience are more prone to poor drainage. This is because the equivalent to the stainless-steel sieve found in conventionally sited drains is a relatively large piece of plastic inserted in the drain. Viewed as ideal biofilm-forming surfaces, the plastic sieves are frequently not used, as is the case in our hospital. Consequently, pieces of plastic such as small syringes find their way down the drain, becoming trapped in the waste tap leading to poor draining. It is a concern that this happens, but it is equally concerning that staff are unaware of the risk that is caused by poor drainage, and only report problems when sinks eventually block.

Carbapenemase-producing Enterobacteriaceae (CPE) are a major concern, threatening the end of the antibiotic era. Increasingly reports are linking CPE outbreaks to waste water systems. However, it may be that transmission events are being recognized because antibiotic-resistant organisms attract immediate attention and investigation, whereas their antibiotic-sensitive equivalents usually do not. It is also important to note that it is not just drains in clinical areas that present a risk. Kitchen sinks, where outlet water may directly hit the drain sieve, can disperse antibiotic-resistant bacteria that can then reach patients via food or other vehicles have caused CPE outbreaks [6]. A CPE outbreak in a German hospital was attributed to salads contaminated by water splashes from a drain in a nearby sink (Professor Exner, personal communication). Most patient showers require the patient to stand directly on, or close to the drain. As such, it does not require a great leap of faith to envisage how patients become exposed to drain organisms; indeed, the problem may be exacerbated by the fact that most hospital shower rooms have a toilet in close vicinity that is likely to be connected to same drain network. CPE challenge our approach to water management as up till now (excluding legionella) the UK focus has been on augmented care units where patients have a susceptibility due to underlying disease. By contrast, CPEs are a threat anywhere in the

hospital, and they are potentially more pervasive within water and sanitation systems.

Biofilms are bacteria's adaption to an aquatic environment, and which thwart our ability to provide safe water or sanitation to patients. The complexity of biofilm and the systems operating therein can only be marvelled at [7]. Antibiotics enter hospital drainage systems in high concentration while still biologically active. A recent report measured antibiotic levels in siphon water, then flushed the siphon with 15 L of water and measured antibiotic levels preventing use of the outlet. Surprisingly it was found that antibiotic levels increased; the authors suggested that this was due to antibiotics leaching back out of the biofilm [8]. What role, if any, this function of the biofilm has in building antibiotic or maintaining antibiotic resistance has yet to be determined.

In the meantime, the work of Aranega-Bou *et al.* teaches us that poor drainage is a risk we need to address. Water safety groups should monitor blockages and find ways of ensuring staff report poorly draining devices (HWS, toilet, shower) as soon as they are detected, as well as addressing underlying causes of the blockage.

For the future it is not clear whether we should most fear the bacteria or our own intransigence. Microbiologists in 1967 preferred folklore (organisms go from patient to HWS not vice versa) to Joachim Kohn demonstrating transmission from HWS to patient. Furthermore, Kohn thought the drain was often the source and invented a drain-sterilizing device in 1970 [9]. The drainage system is where bacteria have installed their fortress, biofilm. The question remains: are we prepared to do anything about it?

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