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Letters to the Editor

Preventing hospital-onset *Escherichia coli* bacteraemia: is hydration the solution?

Sir,

A recent publication in the *Journal of Hospital Infection* by Omar *et al.* demonstrated the difficulties associated with keeping inpatients hydrated [1]. Hydration in inpatients remains a complex issue, and we commend the study's aim to highlight and tackle this important issue.

Most previous research on urinary tract infections (UTIs) and dehydration has been undertaken in animal models. A 1966 study on the kinetics of UTIs showed that normal physiological flow of urine keeps the bacterial population at a steady level, with lower rates of flow tending to increase the bacterial population [2]. In 1970, Andriole used a rat model to demonstrate that *Escherichia coli* pyelonephritis can be induced by increasing the osmolality of medullary tissue by decreasing water intake [3]. Additionally, granulocyte mobilization and phagocytosis are highly inhibited in concentrated urine [4]. These animal model studies played a role in highlighting the role of hydration in UTIs.

In England, *E. coli* bacteraemia rates continue to increase and are associated with increased mortality. Hospital-onset cases often exhibit increased resistance [5–7]. Abernathy *et al.* used surveillance data to highlight risk factors for *E. coli* bacteraemia [8]. The study did not consider hydration specifically, but highlighted that 51.2% of the cases reviewed were secondary to a urogenital tract focus with urinary tract catheterization and prior healthcare contact as potential risk factors.

At Nottingham University Hospitals, we have achieved a reduction in hospital-onset *E.coli* bacteraemia through a multifaceted approach [9]. Over a period of three years, hospital-onset *E. coli* bacteraemia has reduced by 36%, whilst there has been no appreciable change in community-onset bacteraemia (Table I). This has been achieved through a multidisciplinary approach which has included specialist continence nurses on medical wards, review of patients attending with biliary sepsis for possible earlier intervention, changes in empirical treatments for UTIs, education on urine dipsticks and a focus on antimicrobial stewardship.

The European Food Safety Authority recommends a minimum daily fluid intake of 2 L in women and 2.5 L in men [10]. However, elderly patients who are admitted to hospital usually suffer from multiple co-morbidities, such as heart failure or hyponataraemia that may require restriction of fluids to lower amounts. Excessive hydration for elderly patients, in order to theoretically prevent *E. coli* bacteraemia, can worsen other medical conditions and lead to poorer outcomes. Another factor to consider is the 'tea and toast' phenomenon, where patients with impaired renal function are on a diet poor in salt and protein, and drinking large volumes of liquid (e.g. tea, coffee). This results in increased water re-absorption, leading to hyponatraemia [11]. Even mild hyponatraemia can contribute to gait and attention impairments, which adds to the multifactorial problem of falls [12].

It is also important to highlight that although animal studies have shown some evidence for dehydration leading to *E. coli* pyelonephritis, no human studies have demonstrated a causal link between hydration and *E. coli* bacteraemia. Some association between hydration and *E. coli* bacteraemia in humans comes from epidemiological data that show an increase in rates during summer months within community-onset cases [13]. However, this may only be an association, and further studies are needed to understand the link between hydration and *E. coli* bacteraemia. Furthermore, this seasonal variation is not detected in hospital-onset cases [13]. Hence we would recommend that the message of hydration in hospitals needs to carefully target appropriate groups of patients. Although oral hydration fluids should be accessible to those who need it, in our view, a blanket approach of 'more fluids' should not be utilized. The required hydration needs to be considered carefully for each individual patient in hospital, taking into consideration their co-morbidities and reasons for admission, because the evidence for hydration in preventing *E. coli* bacteraemia is weak.

Table I
Hospital- and community-onset bacteraemia in Nottingham as defined by national surveillance definition [9]

Year	Cases of hospital-onset bacteraemia (*N*)	Cases of community-onset bacteraemia (*N*)
April 2016–March 2017	247	558
April 2017–March 2018	181	504
April 2018–March 2019	159	553

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None declared.

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Contamination of sink drains with carbapenemase-producing Enterobacteriaceae in intensive care units: a concern but don't worry so much!

Sir,

Recent reports have implicated hand-washing sinks as a primary reservoir of resistant pathogens, including carbapenemase-producing *Pseudomonas aeruginosa* [1] and carbapenemase-producing Enterobacteriaceae (CPE) [2], within patient care environments. Although CPE outbreaks are mostly attributed to patient-to-patient transmission via healthcare workers, recent studies identified the sink drains (SDs) as a possible source of transmission in intensive care units (ICUs) [3–5]. Isolation of closely related strains in SDs and in patients, discontinuation of outbreaks, or decrease in acquisition of CPE after the implementation of SD disinfection measures supported this hypothesis. As these studies have been conducted during outbreaks, data concerning the risk of patient contamination within long periods of exposure to contaminated SD outside an epidemic context are lacking.

In 2015, environmental sampling was performed in the 12-bed surgical ICU (SICU) in our 1500-bed French university hospital, as part of the investigation into a carbapenemase-producing *Klebsiella pneumoniae* (KPC) outbreak. Outbreak discontinuation was achieved by implementing a bundle of preventive measures. All SDs were sampled on the same day in 2015; they were re-sampled on a single day in April 2019, outside any epidemic context. Sampling was performed by rotating sterile swabs inserted to a depth of around 5 cm through the sink drain. Specimens were stored in a transport medium (eSwab, Copan, Brescia, Italy) and were then plated on to selective agar plates (chromID CARBA SMART, bioMérieux, Marcy l'Etoile, France). Identification of suspicious colonies was performed by matrix-assisted laser desorption ionization – time of flight mass spectrometry (MALDI-TOF MS) using a Vitek MS mass spectrometer (bioMérieux). For suspicious colonies, carbapenemases were detected by immunochromatography (RESIST-4 O.K.N.V., Coris Bioconcept, Gembloux, Belgium) and confirmed by the method of combined tests (Rosco Diagnostica, Taastrup, Denmark). Isolates were compared using pulsed-field gel electrophoresis (PFGE) as previously described [6] and strains were categorized according to the criteria of Tenover *et al.* [7]. Data concerning CPE isolated in patients hospitalized in the SICU within the 42 months separating the two sets of sink sampling were obtained retrospectively from the laboratory database (GLIMS 8, MIPS Diagnostics Intelligence, Gent, Belgium).