Antimicrobial Resistance Surveillance System in South Korea.[2–4]

Among 1492 patients with BSIs due to *E coli*, 141 (10%) died within 30 days after BSI onset. Of the 1492 *E coli* blood isolates, 359 (24·4%) belonged to sequence type (ST) 131, 140 (9·5%) belonged to ST95, and 126 (8·6%) belonged to ST69. ST131 was a risk factor for 30-day mortality. Carbapenem resistance was observed in three (0·2%) of 1492 *E coli* blood isolates devoid of carbapenemase production. All patients with BSIs caused by carbapenem-resistant *E coli* took inadequate empirical antimicrobial therapy, and all survived until the end of follow-up. The proportion of patients receiving improper empirical antimicrobial therapy was 347 (23·3%) of 1489 patients with BSIs due to carbapenem-susceptible *E coli*. Notably, resistance of *E coli* isolates to third-generation and fourth-generation cephalosporins was high (35·1%, 524 of 1492 isolates), the associated bla_{CTX-M} extended-spectrum beta-lactamase genes were widespread (31·0%, 463 of 1492 isolates), and the high proportion of inadequate empirical antimicrobial therapy (52·7%, 276 of 524 isolates) and 30-day mortality (12·8%, 67 of 524 patients) were affected by the third-generation and fourth-generation cephalosporin resistance.

Of the 579 *K pneumoniae* blood isolates, 81 (14·0%) were ST23, 32 (5·5%) were ST86, and 31 (5·4%) were ST65. The hypervirulent K1 (13·5%, 78 of 579 isolates), which was associated with liver abscess, and K2 (20·9%, 121 of 579 isolates) were the predominant capsular types.[3] The 30-day mortality rate of patients with BSIs because of *K pneumoniae* was 16·9% (98 of 579 patients), and the capsular type wzi50, which is associated with neither K1 nor K2, was a risk factor. The proportion of carbapenem resistance in *K pneumoniae* isolates was 18 (3·1%) of 579 isolates of which 13 (72·2%) of these 18 isolates were associated with improper empirical antimicrobial therapy and eight (44·4%) of 18 patients with associated BSIs died within 30 days. These results were much higher than for those patients with BSIs due to carbapenem-susceptible *K pneumoniae* of which 72 (12·8%) of 561 isolates were associated with improper empirical antimicrobial therapy and 88 (15·7%) of 561 patients with associated BSIs died within 30 days. Eight (44·4%) of 18 carbapenem-resistant *K pneumoniae* isolates possessed the bla_{KPC} gene; six (75·0%) of these eight isolates were associated with inadequate empirical antimicrobial therapy, and five (62·5%) of eight patients with associated BSIs died within 30 days.

The overall incidence of carbapenem-resistant Enterobacteriaceae and carbapenemase-producing Enterobacteriacea in South Korea is still modest among blood isolates. However, their growing dominance necessitates a close look because antimicrobial resistance is a public health concern for all countries. International travel has an important role for the worldwide spread of antimicrobial resistance,[5] and carbapenem-resistant Enterobacteriaceae and carbapenemase-producing Enterobacteriacea spread intercontinentally at an enormous speed.[6] The time to call for collaborative action to prevent the spread through a systematised plan, including antimicrobial stewardship led by specialists worldwide, is now.

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Carbapenem-resistant *Klebsiella pneumoniae* with low chlorhexidine susceptibility

Carbapenem-resistant *Klebsiella pneumoniae* (CRKP) is an emerging pathogen of nosocomial outbreaks. In 2018, in our 1400-bed hospital, we identified eight patients colonised with CRKP by rectal screening, of whom four were epidemiologically related. All isolates were sequenced by Illumina MiSeq (San Diego, CA, USA). Six isolates were closely related (sequence type 258, complex type 123) and harboured the plasmid-encoded β-lactamase bla_{KPC-2} carbapenemase, as well as $bla_{TEM-122}$, bla_{SHV-12}, and bla_{OXA-9}. On the basis of genetic distances and the epidemiological data, we detected a nosocomial cluster in the intensive care unit (ICU) in July, 2018.

Subsequently, we sequenced six stored isolates from a local outbreak in 2017. All isolates were clonally related to the cluster in our hospital (appendix). We investigated the biocide susceptibility of all isolates by analysing their minimum inhibitory concentrations (MICs) and minimum bactericidal concentrations (MBCs).

See **Online** for appendix

For all isolates, we identified reduced susceptibility to chlorhexidine (MIC 32 mg/L, MBC 32–128 mg/L). According to epidemiological cutoff values (ie, 64 mg/L for both MIC and MBC), the MIC was at the upper limit of the normal MIC distribution.[1] Three isolates had an MBC of 128 mg/L. Additionally, two isolates exhibited resistance to colistin (MICs of 16 mg/L and 32 mg/L), which was conferred by efflux, as confirmed by an inhibitor test with cyanide 3-chlorophenylhydrazone. One of these isolates exhibited single-nucleotide polymorphisms in the *phoPQ* two-component regulatory system and genes regulated by PhoPQ (*pmrK*, *smvA*, and *smvR*).[2] For the other isolate, we could not find mutations within these genes, suggesting that other mechanisms of resistance were involved.

Notably, a chart review revealed that four patients colonised with isolates that were non-susceptible to chlorhexidine (one with an MBC of 128 mg/L) were washed regularly with a chlorhexidine washcloth (20 mg/mL chlorhexidine gluconate; SAGE Products, Cary, NC, USA) during their ICU stay. Although some studies have shown that daily chlorhexidine bathing is effective for reducing the risk of Gram-negative infections,[3] others have not confirmed this observation.[4] The fact that the chlorhexidine concentration on the skin fell below effective concentrations after 1–3 days questions the use of chlorhexidine bathing, particularly in the case of a clinical outbreak.[5]

This outbreak is the first report of nosocomial transmission of CRKP non-susceptible to chlorhexidine within an ICU using daily chlorhexidine bathing. In addition, for the first time, we have shown colistin resistance related to efflux in chlorhexidine-adapted clinical isolates. To our knowledge, these data add the first real-world evidence for the hypothesis of Wand and colleagues[2] that wide-ranging chlorhexidine exposures increase the risk of colistin resistance emerging in CRKP clones.

We declare no competing interests.

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Should use of online photographs be exempt from ethical guidelines?

The use of a photo of a clearly identifiable Ugandan adolescent girl accompanying the Comment by Rosanna Wai Wan Peeling and David Mabey[1] is deeply troubling. The inclusion of this photo violates ethical principles paramount to biomedical research: justice and autonomy.[2] The use of this adolescent girl as artwork to illustrate a problem to which this individual has no clear connection is an unjust misrepresentation that undermines her autonomy through absence of consent and reinforces damaging stereotypes.

The Comment specifically discusses the women's improvement of sexual and reproductive health (WISH) study in Rwanda and the implications of the study's findings to all low-resource settings. The photograph, obtained from Flickr, is captioned on Flickr with only the words, "Girls, Uganda." The selection of this photograph suggests that either a Ugandan adolescent girl is being used to illustrate an issue in Rwanda, or a black adolescent girl is being used to illustrate all low-resource settings; both are inappropriate. If this young person is a minor—which cannot be determined from the image or caption—this is an additional ethical issue.

In prior eras, this adolescent girl or anyone from her community would be highly unlikely to ever access the Comment. But in today's digital age, inaccessibility is no longer the case, and she might be stigmatised by her community for the implication that she needs testing for sexually transmitted infections (STIs). Nevertheless, even if no one in her community ever sees her photo in the journal, that does not negate the injustice. If given an opportunity to consent, she, like most adolescent girls, would likely object to being made a poster child for STI testing.

Participants in clinical trials undergo a detailed consent process before any anonymous inclusion in a study. This same code of ethics, which emphasises protecting the rights and privacy of research participants, should be applied to photographs used in biomedical research journals and conference presentations. Within journalism, a code of ethics[3] on visual representation has been developed and upheld, and inappropriate use of photos, leading to misrepresentation, has been written about for many years.[4] In biomedical research, despite strong guiding principles[2], the conversation on visual representation has not yet