

## BACKGROUND

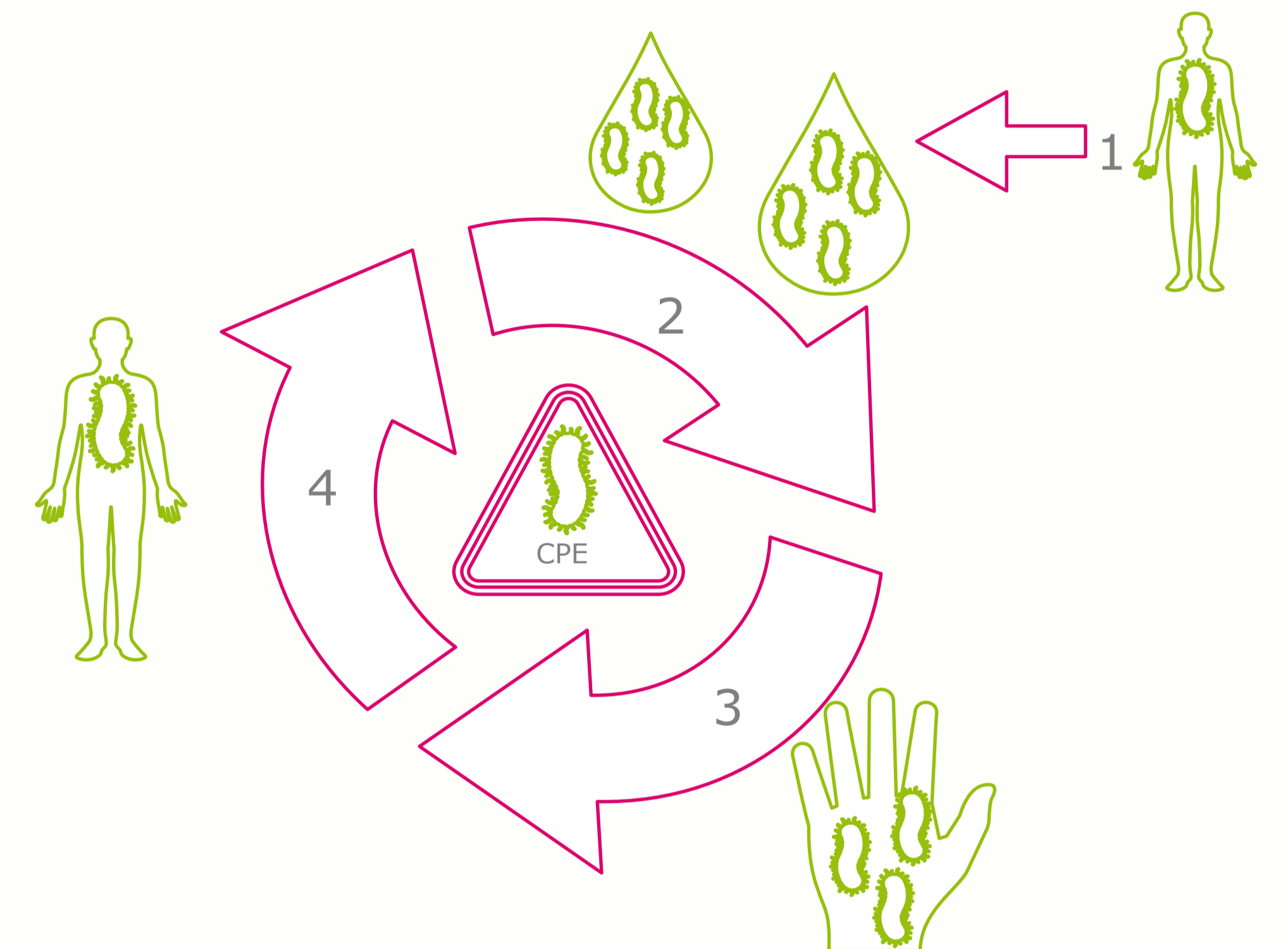
Carbapenemase-producing Enterobacteriaceae (CPE) are emerging pathogens in the last decade. In Belgium, the OXA-48 carbapenemase resistance gene is most frequently identified. Sink drains in ICUs are known to be colonized by Gram negative bacilli. A correlation between environmental contamination and CPE infections at ICUs has been established. A long term CPE epidemic in one of our ICU wards proved difficult to control.

## SETTING

Ziekenhuis Netwerk Antwerpen (ZNA) is a hospital group in Antwerp, Belgium with 2,200 beds over nine campuses. There are 8 ICUs in total. The ICU we study is a general 14-bed adult ICU of a 400-bed hospital site. All rooms have a sink. The distance between the sink and the patients bed is often less than one meter. Patient fluids are occasionally disposed of in the sinks. Tube feeding, aspirated stomach fluids, water for washing the patient, body hair and dialysate are all disposed of in the drain.

## HYPOTHESIS

1. Siphons are the reservoir of CPE. These siphons get colonized through body fluids that are disposed of in the sinks.
2. Colonized siphons are a major culprit of CPE infections at the ICU.
3. Transmission of CPE happens through droplets and aerosols from the siphon, spreading through the environment and hands of hospital personnel.
4. The colonized patient keeps the cycle going.
5. Break the cycle by intervening in step 2.



## METHODS & INTERVENTION

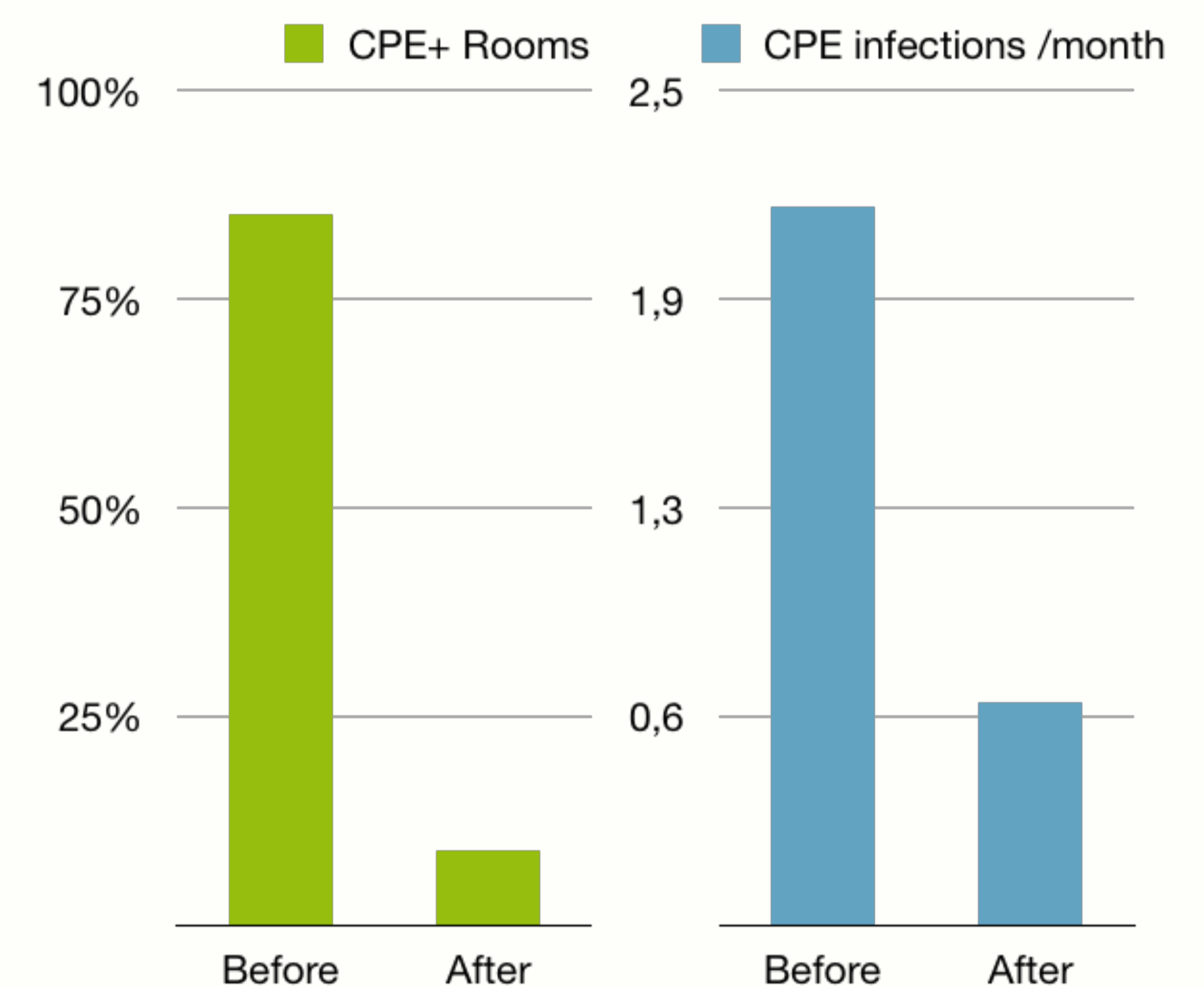
1. Three times weekly we took a swab of the sink drains at the ICU. We cultured them on a selective agar for CPE. When there was growth, the OXA-48 gene, antibiotic and acetic acid resistance were tested.
2. We decontaminated the sinks with 250mL 25% acetic acid three times weekly. We looked at the rate of new CPE infection at the ICU before intervention and after.

## RESULTS

Before decontamination started 4 out of 5 rooms were positive for CPE in the sinks. When decontamination started, 0-2 out of 5 rooms were positive during this period. This means out of 1672 patient admittance days 1426 (85,3%) of these days were in a CPE positive room. After decontamination there were 81 (9,1%) CPE positive admittance days over a total of 888 days. This is a reduction of 89,3% in exposure to CPE colonized sinks.

All of the cultured CPE were OXA-48 positive. The MIC of meropenem varied from 0,5 – 2. The duration of exposure to acetic acid influenced the resistance. 5 Minutes of exposure gave a MBC for acetic acid of 0,86 to 13,8% and after 30 minutes this was reduced to 0,43 - 3,4%.

Initially there were 2,15 new CPE infections per month. After the start of decontamination there were only 0,67 per month.



## DISCUSSION

A study in Sweden<sup>1</sup> also used acetic acid for decontamination. They used 24% once weekly for metallo-beta-lactamase-producing *Pseudomonas aeruginosa* (Pae-MBL). Their intervention resulted in negative sink cultures and termination of transmission at the ICU. They tested the in vitro susceptibility of Pae-MBL biofilms to acetic acid. They found that 0.75% of acetic acid was enough to eradicate the biofilm. Our results of a mean 1.32% is higher. However, we pooled susceptibility of Enterobacteriaceae, *Pseudomonas* and *Stenotrophomonas*. *Pseudomonas* species tested 0.86%, which is in line with what others found.

Cases of CPE infection/colonization still occurring could be either due to missing the CPE with initial screening or due to transmission from other patients in the ICU.

## CONCLUSION

Our method of decontaminating sink drains with acetic acid helps in reducing new CPE infections and colonization at our ICU. It is a valuable alternative to other methods like heated sinks and water-free care. Especially when other options are not feasible in the near term. Acetic acid is cheap, widely available, effective and manageable from a safety and technical point of view.

1. Stjarne Aspelund A, Sjoström K, Olsson Liljequist B, Morgelin M, Melander E, Pahlman LI Acetic acid as a decontamination method for sink drains in a nosocomial outbreak of metallo- beta-lactamase-producing *Pseudomonas aeruginosa*. J Hosp Infect 2016; 94: 13-20.