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Reply to "Colistin Resistance during Selective Digestive Tract Decontamination Is Uncommon"

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We thank Zandstra and colleagues for their comments (1). The outcome of our analysis cannot be ignored, and we disagree with the conclusion of Zandstra et al. that our study is an inappropriate study and provides a low level of evidence. We analyzed a complete set of microbiological data obtained before the start, during the use, and after the stop of selective digestive tract decontamination (SDD). The introduction of SDD, started during an outbreak of extended-spectrum β-lactamase (ESBL)-producing *Klebsiella pneumoniae* (ESBL-Kp), was followed by a clear increase in the detection of pathogens intrinsically resistant to colistin (CIR). Segmented regression analysis revealed that also the proportion of CIR isolates that were tobramycin resistant increased significantly after SDD initiation. When SDD was stopped, these increasing rates of tobramycin resistance reversed toward baseline. In addition, SDD was associated with the emergence of colistin resistance among the ESBL-Kp isolates, while ESBL-Kp isolates were colistin sensitive before the start of SDD. Among ESBL-Kp isolates obtained after the stop of SDD, colistin resistance was no longer observed. These data altogether demonstrate a strong association between SDD and the course of resistance as described.

With respect to the specific issues raised by Zandstra et al., we have the following comments. Our study is a microbiological study, focused on the susceptibility characteristics of all ESBL-Kp strains and all CIR strains isolated in the intensive care unit (ICU) between 2001 and 2008. SDD was used between October 2002 and April 2007, according to classical SDD rules (2). We do not understand how patient data could influence the appearance and disappearance of colistin and tobramycin resistance and why Zandstra et al. ask for these data.

We used the term occurrence in its epidemiological sense, which can be either incidence or prevalence. Our data are incidence data (number of new isolates/month) when detailing the course of the outbreak. Regarding analysis of CIR strains, we used the prevalence and the proportion of tobramycin-resistant isolates.

Indeed, we did not distinguish between imported and ICU-acquired pathogens: we show the global increase in colistin resistance among ESBL-Kp strains and the total increase in CIR strains. Whether the pathogens are imported and then selected by SDD use or whether they arise during SDD would be impossible to distinguish anyway. Certainly, SDD did not eliminate colistin-resistant strains from the ICU, neither imported ones nor ICU-acquired ones.

Sampling for surveillance purposes was started in January 2002, 10 months before the start of SDD, according to the surveillance scheme that is used during SDD.

Finally, as to the authors' comment about published evidence on the association of SDD with a reduction of antibiotic resistance and control of outbreaks, we did not ignore the literature on this subject. We report our findings at face value and agree with the work of Daneman et al. (3), who concluded in their recent meta-analysis on the effect of SDD on antimicrobial resistance in the ICU that "the effect of decontamination on ICU-level antimicrobial resistance rates is understudied."

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