suggests nosocomial transmission. Owing to limited resources, the current routine microbiology workflow at our setting—as is the case in most laboratories in developing countries—does not include bacterial typing to identify nosocomial infections. The hospital infection prevention strategies are generic and focus on hygienic procedures rather than identification of microorganisms.

Availability of robust and rapid WGS allowing simultaneous genotyping of different microorganisms within a relatively short time holds the potential of controlling nosocomial infections and improving care. The decreasing initial and recurrent costs of WGS give optimism that in the near future this technology will be applied more widely in resource-limited settings, which are struggling with a disproportionately high burden of infectious diseases with suboptimal infection control strategies.

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Revised Risk Estimates for MRSA Infection in Patients with Intermittent Versus Persistent MRSA Nares Colonization

We thank Beyersmann and Schrade[1] for their comments, and we agree that the use of an estimator that accounts for death without prior infection is appropriate for our data. As they point out, the raw incidence proportions of methicillin-resistant *Staphylococcus aureus* (MRSA) we reported (16.33% for persistently colonized, 11.18% for intermittently colonized, and 0.51% for non-colonized) reflected observed infections, and did not account for infections that may have occurred after administrative censoring at the study end date. Thus, these data represent an underestimation of risk.

For this reason, we reported proportions based on Kaplan-Meier analysis: 21.26% for persistently colonized, 12.83% for intermittently colonized, and 0.55% for non-colonized. Beyersmann and Schrade aptly pointed out that this analysis does not account for death without prior infection and thus overestimates risk. They propose that the Aalen-Johansen method is a better estimator in this case because it accounts for competing causes. We agree, and we conducted an additional analysis. These new calculations produced results that fall between the other 2 estimates: 20.61% for persistently colonized, 12.16% for intermittently colonized, and 0.54% for non-colonized. The use of the Aalen-Johansen estimator increases the precision of the estimates without changing our overall conclusions.

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Colistin and Tigecycline Resistance in Carbapenem-Resistant Enterobacteriaceae: Checkmate to Our Last Line Of Defense

To the Editor—The emergence and spread of antimicrobial drug resistance in Enterobacteriaceae is posing a serious threat to the treatment of nosocomial infections. Of particular importance are the pathogens of this family that produce metallo-β-lactamases (IMP-type carbapenemases [IMP], New Delhi metallo-β-lactamase, or Verona integron-encoded metallo-β-lactamase [VIM]), non-metallo enzymes (*Klebsiella pneumoniae* carbapenemase and Oxacillinase [OXA]-48), β-lactamases with a broad profile of substrate activity such as extended-spectrum β-lactamases, or AmpC enzyme with porin loss. Until recently, carbapenems have been successfully used for the treatment of infections caused by Enterobacteriaceae, including those producing extended-spectrum β-lactamases.[1] Antibiotic treatment options for these emerging carbapenem-resistant Enterobacteriaceae (CRE) are becoming limited.[2] Colistin and tigecycline have been reported as the remaining armamentarium against the species of Enterobacteriaceae.[3,4] In the present study, a total of 210 clinically significant Enterobacteriaceae isolates were collected from various clinical samples of admitted patients (blood, urine, wound, and burn) over a period of 2 years (2013–2015). Susceptibility testing was performed by Clinical and Laboratory Standards Institute broth microdilution method, and isolates with a meropenem or imipenem minimum inhibitory concentration (MIC) of at least 4 mg/L were categorized as CRE. *Escherichia coli* ATCC 25922 was used as the control strain. Among these 210 isolates, 31 bacteria showed resistance to both imipenem (MIC ≥32 mg/L) and meropenem (MIC 32 mg/L). By means of 16S rRNA sequence of 31 CRE isolates, the following members of the Enterobacteriaceae family were identified: *Enterobacter cloacae* (5), *Enterobacter aerogenes* (4), *Serratia marcescens* (2), *Providencia stuartii* (2), *Klebsiella pneumoniae* (6), *Citrobacter freundii* (2), *Proteus mirabilis* (4), and *E. coli* (6). CRE are increasingly prevalent in many parts of the world.[5] These CRE isolates were further screened for their resistance toward colistin (MIC 32–64 mg/L) and tigecycline (MIC 16-64 mg/L). All the strains of *S. marcescens*, *K. pneumoniae*, and *E. coli* showed resistance to both colistin and tigecycline (Figure 1). Resistance to colistin was 100% for all the strains of *E. cloacae*, *P. stuartii*, *C. freundii*, and *P. mirabilis*, whereas only 75% of strains of *E. aerogenes* were colistin resistant (Figure 1). A total of 80% of strains of *E. cloacae* and 75% of strains of *P. mirabilis* were resistant to tigecycline (Figure 1). Similarly, 50% of strains of *E. aerogenes*, *P. stuartii*, and *C. freundii* were tigecycline resistant (Figure 1). In the past few years, there have been sporadic reports of colistin-resistant CRE cases from various parts of the world, such as Greece, Israel, South Korea, United States, and Singapore,[6] and of tigecycline-resistant CRE.[7] Colistin- and tigecycline-resistant CRE cases have never been reported from India. It should be further noted that co-resistance to both colistin and tigecycline among the CRE strains was not reported before.

Thus, there is clearly a need for the development and screening of new antimicrobial agents to keep pace with the

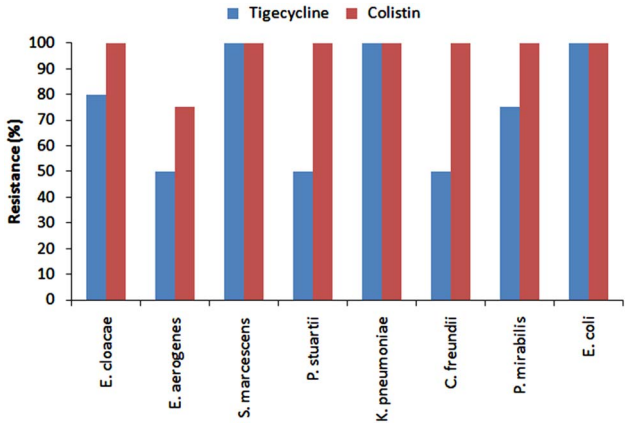

FIGURE 1. Resistance pattern of carbapenem-resistant *Enterobacteriaceae to last resort antibiotics. C. freundii, Citrobacter freundii; E. aerogenes, Enterobacter aerogenes; E. cloacae, Enterobacter cloacae; E. coli, Escherichia coli; K. pneumoniae, Klebsiella pneumoniae; P. mirabilis, Proteus mirabilis; P. stuartii, Providencia stuartii; S. marcescens, Serratia marcescens.*