Letters to the Editor

Enterobacter Plasmids: Molecular Epidemiology

To the Editor:

In the article by Jarvis[1] from the Third International Conference on the Prevention of Infection, there are several excellent molecular epidemiologic presentations. The last one, however, is supposed to represent an *EcoRI* digestion of a set of *Enterobacter cloacae* plasmids. It looks in the figure as though the plasmids in fact either were not cut or had no *EcoRI* sites. The former is more likely, and I suspect that the figure is supposed to represent only the redundant plasmid that was seen in the isolates from patients, technician, and the environment. Most *E cloacae* plasmids of the size portrayed in the figure would be expected to have one or more *EcoRI* sites, thus suggesting that the figure represents only undigested plasma DNA.

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REFERENCE

1. Jarvis WR. Usefulness of molecular epidemiology for outbreak investigations. *Infect Control Hosp Epidemiol 1994:15:500503.*

The author *replies.*

I thank Dr. John for bringing to my attention the error in my manuscript. He is correct that Figure 6 included in my article "Usefulness of Molecular Epidemiology for Outbreak Investigations" is of the plasmid analysis of the *Enterobacter cloacae* isolates obtained from the patient's blood cultures, the laboratory technician handwashings, and the laboratory environment. As mentioned in the article, we performed both plasmid analysis and restriction endonuclease analysis of the plasmids using *EcoRI.* Inadvertently, the figure of the plasmid analysis was included rather than the figure of the restriction endonuclease analysis. Shown here is the figure that should have been included (Figure 1). Note that the lane placement of the isolates in the two gels is identical in the two figures.

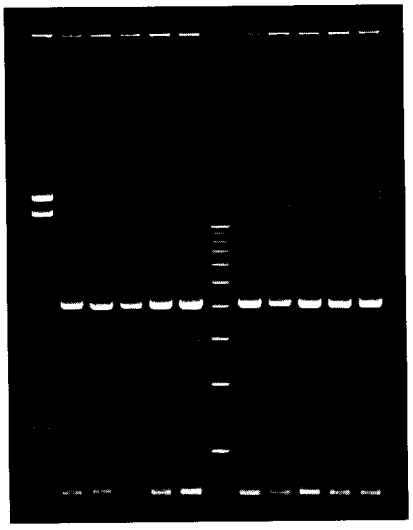

FIGURE 1. Restriction endonuclease analysis of *Enterobacter cloacae* isolates.

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CDAD Rates

To the Editor:

We applaud Olson et al for their recent comprehensive report on the epidemiology of *Clostridium difficile*-associated diarrhea (CDAD) at their medical center over a 10-year period.[1] However, several points of their article deserve further clarification and discussion.

First, although the rate of CDAD per hospital admissions was calculated for the entire 10-year period of surveillance, no such rate was furnished for each year of the study. Therefore, any trends in CDAD during the study period would be difficult to interpret. Did the number of yearly admissions and patient hospital days remain constant during the study period?

Second, it was reported that 93% of CDAD cases were acquired nosocomially. What was the definition of nosocomial CDAD in this study? Because CDAD may not become clinically manifest until after discontinuation of antibiotic therapy,[2] was there a mechanism by which development of CDAD in discharged patients receiving antibiotics was monitored? If so, did this mechanism remain constant during the study period?

Third, the authors report that implementation of body substance isolation was associated with a decrease in CDAD during the first 2 years of its implementation, and the subsequent increase in the rate of new CDAD cases in 1990 and 1991 at their medical center might have been related to the introduction of more virulent strains of *C difficile.* An alternative explanation to this apparent increase in cases of CDAD may be overuse of gloves and delay in their removal following their soilage, resulting in an increase in contamination of patients and their environment. Recent reports of *Acinetobacter,* methicillin-resistant *Staphylococcus aureus,* and CDAD outbreaks in the setting of universal precautions[3,4] seem to support this view.

We also have reviewed the yearly incidence of nosocomial CDAD

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