

Why were molecular studies not considered to classify *Clostridium difficile* isolates implicated in an outbreak?

To the Editor:

We read with interest the article, "*Clostridium difficile* outbreak in Costa Rica: control actions and associated factors," by Roy A. Wong-McClure and colleagues, recently published by this journal (1).

This work is of great value because it broadens the scarce knowledge regarding infections caused by *C. difficile* in hospitals in Latin America (2). It describes the measures taken for a 4-month *C. difficile* hospital diarrhea outbreak, and confirms that age > 59 years, hospitalization for 7 days or more, diabetes or chronic kidney failure, and therapy with ceftazidime and cefotaxime are risk factors associated with *C. difficile* associated disease (CDAD), as previously reported (3). On the other hand, despite the authors' explanation, the lack of association between CDAD and the administration of fluoroquinolones is noteworthy as it contradicts previous studies (3).

Our laboratory experience with *C. difficile* (4–5) has led us to confirm that strict procedures should be followed in order to achieve high cultivation rates from clinical samples. Thus, it would be interesting to know the isolation rate obtained by the authors, and to verify if all "toxin-positive isolates were confirmed by culture," especially since no enrichment procedure was used—contrary to what is recommended (6). In addition, the authors should clarify the number of isolates studied, so that readers might corroborate whether the isolates analyzed were representative of the outbreak.

Since no references are given in the laboratory analysis section, it is not clear whether *tcdC* deletions were detected by molecular methods. This information is important because there is considerable controversy regarding the role of these mutations in the toxin hyperproduction of epidemic strains (7).

The authors state that the isolates were non-BI strains characterized by the presence of the *tcdA* and *tcdB* genes. However, this information is somewhat vague from an epidemiological standpoint. It would be interesting to learn the intraspecific diversity of

C. difficile in the outbreak, so as to compare it with the diversity of pulsotypes that we have previously found in another Costa Rican hospital (4) and to clarify if there were dominant pulsotypes. This information is crucial in light of our most recent studies, which reveal differences in the amounts of toxins produced by autochthonous pulsotypes (unpublished results).

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