- Welford M, Bossak B. Body lice, *Yersinia pestis* Orientalis, and Black Death [letter]. Emerg Infect Dis. 2010;16:1651.
- Welford MR, Bossak BH. Validation of inverse seasonal peak mortality in medieval plagues, including the Black Death, in comparison to modern *Yersinia pestis*variant diseases. PLoS One. 2009;4:e8401. DOI: 10.1371/journal.pone.0008401
- Drancourt M, Roux V, Dang LV, Tran-Hung L, Castex D, Chenal-Francisque V, et al. Genotyping, Orientalis-like *Yersinia pestis*, and plague pandemics. Emerg Infect Dis. 2004;10:1585–92.
- Pusch CM, Rahalison L, Blin N, Nicholson GJ, Czarnetzki A. Yersinial F1 antigen and the cause of Black Death. Lancet Infect Dis. 2004;4:484–5. DOI: 10.1016/ S1473-3099(04)01099-0
- Bianucci R, Rahalison L, Massa ER, Peluso A, Ferroglio E, Signoli M. Technical note: a rapid diagnostic test detects plague in ancient human remains: an example of the interaction between archeological and biological approaches (southeastern France, 16th–18th centuries). Am J Phys Anthropol. 2008;136:361–7. DOI: 10.1002/ajpa.20818
- Wiechmann I, Grupe G. Detection of *Yersinia pestis* DNA in two early medieval skeletal finds from Aschheim (Upper Bavaria, 6th century AD). Am J Phys Anthropol. 2005;126:48–55. DOI: 10.1002/ ajpa.10276
- McLean RG, Fall MW. Body lice, *Yersinia pestis* Orientalis, and Black Death [letter]. Emerg Infect Dis. 2010;16:1651–2.
- Blanc G, Baltazard M. Recherches expérimentales sur la peste. L'infection du pou de l'homme: *Pediculus corporis* de Geer. CR Acad Sci. 1941;213:849–51.
- Raoult D, Dutour O, Houhamdi L, Jankauskas R, Fournier PE, Ardagna Y, et al. Evidence for louse-transmitted diseases in soldiers of Napoleon's Grand Army in Vilnius. J Infect Dis. 2006;193:112–20. DOI: 10.1086/498534
- Raoult D, Roux V. The body louse as a vector of reemerging human diseases. Clin Infect Dis. 1999;29:888–911. DOI: 10.1086/520454

Address for correspondence: Michel Drancourt, Unité des Recherche sur les Maladies Infectieuses et Tropicales Emergent, CNRS UMR 6236, Faculté de Médecine, 27 Blvd Jean Moulin, 13385 Marseille CEDEX 5, France; email: michel.drancourt@univmed.fr

All material published in Emerging Infectious Diseases is in the public domain and may be used and reprinted without special permission; proper citation, however, is required.

Clostridium difficile Infections among Hospitalized Children, United States, 1997–2006

To the Editor: Zilberberg et al. described a notable increase in rates of *Clostridium difficile* infection (CDI)–related hospitalizations of children during 1997–2006 on the basis of analysis of data from 2 national administrative databases (*I*). As the authors acknowledge, they used administratively coded databases, which have inherent misclassification and testing biases.

Detection of *C. difficile* toxin indicates that bowel flora have been perturbed. However, the clinical role of toxin detection or isolation of *C. difficile* organisms in children is controversial. Although primary CDI is a recognized pathologic entity in children, one needs to consider whether another etiology related to a concomitant infection, antimicrobial drug administration, or alteration in enteral nutrition may be the precipitating event resulting in *C. difficile* toxin production.

It is our clinical observation that availability of testing for C. difficile and rapidity of assay results play a role in the submission of stool specimens for analysis. In 2007, we conducted a 5-month retrospective chart review of C. difficile testing practices at 2 local tertiary-care pediatric hospitals. Of 796 stool specimens submitted, 42 (5%) were notable for the detection of toxin A or B; these samples represented 35 patients (2). Medical coders likely face the same challenges as clinicians who must interpret toxin assay results and their clinical role with regard to hospitalized children. Although the \approx 2-fold increase in CDI-associated hospitalization rates reported by Zilberberg et al. in their time series and cross-sectional analyses is notable, these results should be interpreted within the context of clinical and epidemiologic factors contributing to generation of this data.

Stephen M. Vindigni and Andi L. Shane

Author affiliation: Emory University School of Medicine, Atlanta, Georgia, USA

References

- Zilberberg MD, Tillotson GS, McDonald LC. *Clostridium difficile* infections among hospitalized children, United States, 1997– 2006. Emerg Infect Dis. 2010;16:604–9.
- Vindigni SM, Sullivan DH, Shane AL. To treat or not to treat? Optimizing pediatric *Clostridium difficile* management. Poster presented at Fifth Decennial International Conference on Healthcare-Associated Infections. 2010 Mar 18–22; Atlanta, GA, USA.

Address for corrrespondence: Andi L. Shane, Division of Pediatric Infectious Diseases, Emory University School of Medicine, 2015 Uppergate Dr NE, Atlanta, GA 30322, USA: email: ashane@emory.edu

In Response: I appreciate the letter by Vindigni and Shane pointing out the need for a cautious approach to treatment for infection with Clostridium difficile isolated from the stool of children, given their propensity for colonization by this organism (1). I could not agree more. In our report, we noted an increase over time in the rate of hospitalizations for not only C. difficile infections (CDIs) but also for rotavirus infections in children. This finding led us to acknowledge the possibility of a reporting bias for CDI (2). Other studies have detected a similar increase in CDIs among hospitalized children and have reported greater severity of associated disease (3-5). Such epidemiologic data, combined with emergence of the BI/NAP1/027 hypervirulent strain of C. difficile in the United States and abroad, support

LETTERS

a real increase in CDIs in a population for which the clinical definition is likely less specific than for adults. Although a more precise clinical definition for CDI in children (primarily those <2 years of age) is needed, studies like ours, which are necessarily limited methodologically, can serve to alert clinicians to be more vigilant to the possibility of disease caused by this evolving pathogen, even in a population thought to be at low risk.

Marya D. Zilberberg

Author affiliations: University of Massachusetts, Amherst, Massachusetts, USA; and Evi*Med* Research Group, LLC, Goshen, Massachusetts, USA

DOI: 10.3201/eid1610.101080

References

- Vindigni SM, Shane AL. Clostridium difficile infections among hospitalized children, United States, 1997–2006. Emerg Infect Dis. 2010;16:1651.
- Zilberberg MD, Tillotson GS, McDonald LC. *Clostridium difficile* infections among hospitalized children, United States, 1997– 2006. Emerg Infect Dis. 2010;16:604–9.
- Kim J, Smathers SA, Prasad P, Leckerman KH, Coffin S, ZaoutisT. Epidemiological features of *Clostridium difficile*– associated disease among inpatients in the United States, 2001–2006. Pediatrics. 2008;122:1266–70. DOI: 10.1542/ peds.2008-0469
- Toltzis P, Kim J, Dul M, Zoltanski J, Smathers S, Zaoutis T. Presence of the epidemic North American pulsed field type 1 *Clostridium difficile* strain in hospitalized children. J Pediatr. 2009;154:607–8. DOI: 10.1016/j.jpeds.2008.10.016
- Suh KN, Gravel D, Mulvey MR, Moore DL, Miller M, Simor AE, et al. *Clostridium difficile*-associated infections in children admitted to acute care hospitals participating in the Canadian Nosocomial Infections Surveillance Progran (CNISP), 2004–2005 [abstract 306]. In: Program of the 18th Annual Scientific Meeting of the Society of Healthcare Epidemiology of America; 2008 Apr 5–8; Orlando, FL. Arlington (VA): The Society; 2008.

Address for correspondence: Marya D. Zilberberg, Evi*Med* Research Group, LLC, PO Box 303, Goshen, MA 01032, USA; email: marya@evimedgroup.org

