Our findings are unique and relevant to healthcare systems and to decision-makers seeking to reduce infection spread. Despite the intensive infection prevention interventions (enteric isolation, discharge terminal cleaning with sporicidal, and ultraviolet disinfection), our findings show that more patient transfers are associated with increased risk of HO-CDI. This raises the likelihood that with less intensive environmental disinfection, there might be an even larger effect of transfer. This research supports investigating the utility of bringing equipment for testing/care to patients, to reduce unnecessary patient movement within hospitals. This would mandate meticulous attention to disinfection of mobile equipment after each patient use or ideally single-patient use. By providing more insight into the mechanisms that propagate HO-CDI, this research provides a new avenue of study regarding the reduction of HO-CDI outbreaks.

Conflict of interest statement
None declared.

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M. McHaney-Lindstrom[a,*]
C. Hebert[b]
J. Flaherty[c]
J.E. Mangino[c]
S. Moffatt-Bruce[b]
E. Dowling Root[a]
[a]*Department of Geography, Ohio State University, Columbus, OH, USA*
[b]*Department of Biomedical Informatics, Ohio State University, Columbus, OH, USA*
[c]*Department of Clinical Epidemiology, Ohio State University, Columbus, OH, USA*

* Corresponding author. Address: 1070 Derby Hall, 154 N Oval Mall, Columbus, OH 43210, USA. Tel.: +1 614-292-2514.
E-mail address: Lindstrom.42@osu.edu (M. McHaney-Lindstrom)

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Increased hospital-specific nosocomial rates of *Clostridium difficile* infection in Finnish hospitals with high prevalence of imported cases at admission, 2008–2015☆

Sir,

Surveillance of *Clostridium difficile* infections (CDI) has been conducted since 2008 as part of the Finnish Hospital Infection Program. Hospital-specific CDI rates are ranked in feedback to participating hospitals to improve control measures. During 2008–2010 the overall nosocomial rate of CDI decreased by 26%; however, the rates and trends differed between hospitals [1]. Besides gaps in infection control, several other factors may influence the hospital-specific CDI rates, such as awareness of CDI, diagnostic methods and activity, prevalence of colonization and infection at admission, antimicrobial usage, and emergence of certain *C. difficile* ribotypes. We aimed to determine whether hospital-specific nosocomial CDI rates are associated with testing frequency, use of polymerase chain reaction (PCR) in CDI diagnosis and/or rate of imported CDI cases.

Data were reported by 19 hospitals participating in the hospital-based CDI surveillance between 2008 and 2015, representing four out of five tertiary, nine out of 15 secondary and three out of ~20 other acute care facilities in Finland. Case-finding was laboratory-based, and the European Centre for Disease Prevention and Control definition for CDI was used [1]. A hospital-specific nosocomial case was defined as a patient with CDI onset 72 h after admission or in the community within four weeks from the last discharge to the same hospital. CDI cases with disease onset before that time were defined as linked to community, to another healthcare facility, or of unknown origin.

We obtained data on stool specimens tested for CDI per 1000 patient-days between 2014 and 2015 from two hospital surveys. Use of PCR on stool specimens in laboratories serving the participating hospitals was obtained from surveys conducted by the national reference laboratory [2]. We used patient-days as

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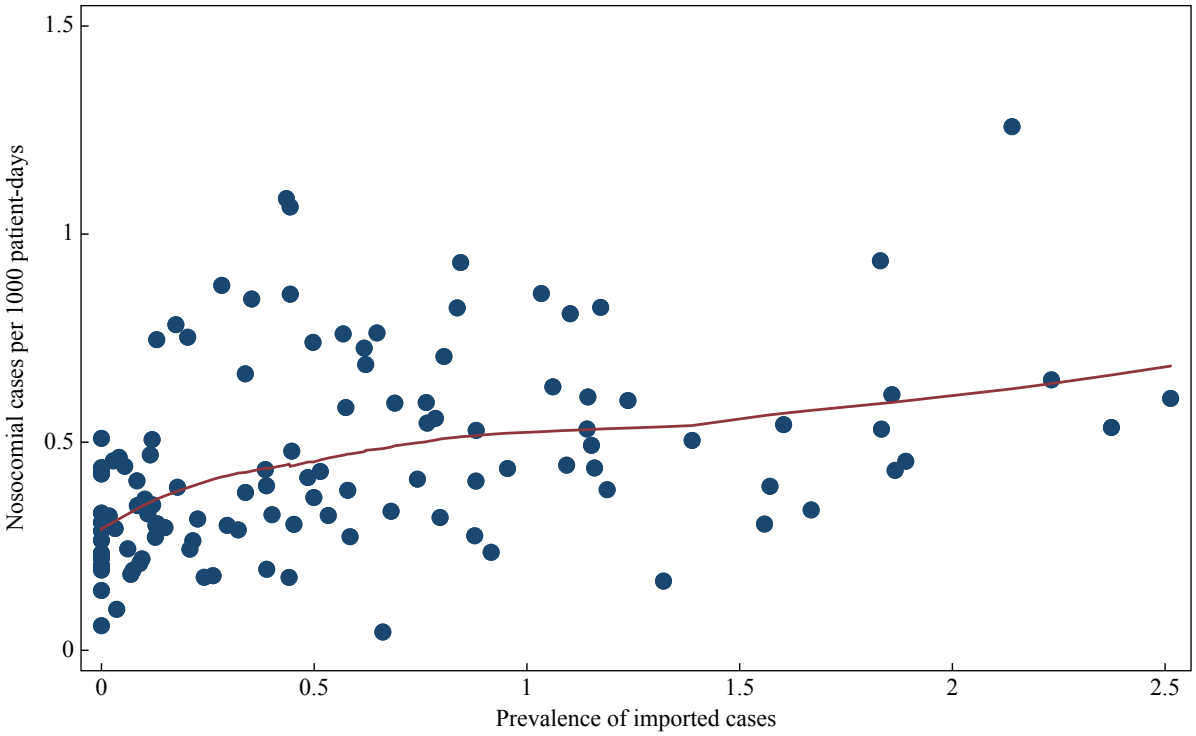

Figure 1. Association between hospital-specific nosocomial rates (cases/1000 patient-days) and prevalence of imported cases at admission (cases/1000 admissions) in 19 Finnish hospitals, 2008–2015 (line indicates the smoothed association between the study variables).

denominators to calculate hospital-specific nosocomial rates, and CDI cases linked to the community or another healthcare facility as numerators and admissions as denominators to calculate prevalence of imported CDI cases at admission. To examine the relationship between annual CDI rates and testing frequency, PCR use and prevalence at admission, we calculated Spearman correlation coefficients (ρ) and incidence rate ratios (IRRs) using mixed-effect negative binominal regression [3].

A total of 6664 CDI cases were identified: 4928 (74%) were hospital-specific nosocomial onset, 950 (14%) community-acquired, 603 (9%) originated from another healthcare facility, and 183 (3%) were of unknown origin.

The overall annual nosocomial rate decreased during 2008–2015 from 0.59 per 1000 patient-days to 0.36 ($P < 0.001$), and it varied between hospitals ten-fold (0.14–1.33 per 1000 patient-days). PCR was introduced in 2008 in six hospitals and was used in all by 2014. The overall annual prevalence of imported CDI cases varied from 0.99 per 1000 admissions in 2008 to 0.33 in 2014, and it ranged between hospitals from 0.00 to 2.51 per 1000 (median: 0.44 per 1000). During 2014–2015, testing frequency differed between hospitals from 0.92 to 41.53 per 1000 patient-days (median: 12.22 per 1000 patient-days). There was a correlation with annual hospital-specific nosocomial CDI rates ($\rho = 0.30$; 95% CI: 0.132–0.463). The hospital-specific nosocomial CDI rates increased with increasing prevalence of imported CDI cases (IRR: 1.21; 95% CI: 1.04–1.42) (Figure 1), and there was a tendency in testing frequency (1.02; 1.00–1.04) but not in PCR use (0.94; 0.77–1.15).

According to our results, one-quarter of CDI cases originated from the community or another healthcare facility, emphasizing that prevention of CDI should go beyond hospital settings. This is in line with our previous study in which one-third of CDIs diagnosed in Finland during 2008–2013 were community-associated (CA) [4]. The previous study also showed an increase in CA-CDI rates which mostly occurred among elderly living in the community or long-term facilities. Inpatient community-onset prevalence is also considered as an adjusting parameter when CDI rates are compared between acute care hospitals in the USA [5].

The German nosocomial infection surveillance system KISS uses a similar surveillance protocol to ours. Our nosocomial rates were in line with German rates but their prevalence at admission was higher than ours [6]. The KISS protocol calculates the prevalence at admission using all patients with CDI, including nosocomial cases related to previous hospitalizations.

Like Goldenberg et al., we found no association between PCR diagnostics and hospital-specific nosocomial CDI rates [7]. Notably, we had no detailed information on methods (toxin detection, toxigenic culture, or cytotoxicity assay) from when a particular laboratory changed to PCR. Knowing that culturing was previously widely used in Finnish laboratories, it is likely that the change happened mostly from toxigenic culture, not from antigen testing [2]. Thus, the change in sensitivity was not large. Nor did we find an association between testing frequency and CDI rates, as previously reported [8]. Our study might have been underpowered, since the data on testing frequency were only available for two years.

Our findings suggest that imported CDI cases should be taken into account when hospital-specific CDI rates are ranked in feedback to participating hospitals to encourage healthcare workers to follow control measures. Controlling CDI in a single hospital may also need collaboration and training in all healthcare facilities in the same region, including long-term care facilities.

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J. Prattingerová[a,b,*,1]
E. Sarvikivi[a,1]
J. Ollgren[a,1]
O. Lyytikäinen[a,1]
[a]*Department of Health Security, National Institute for Health and Welfare, Helsinki, Finland*
[b]*European Programme for Intervention Epidemiology Training, European Centre for Disease Prevention and Control, Stockholm, Sweden*

[1] All authors contributed equally to this article.

* Corresponding author. Address: National Institute for Health and Welfare, Mannerheimintie 166, FI-00271 Helsinki, Finland. Tel.: +420 603 295 280.
E-mail address: jana.prattingerova@gmail.com (J. Prattingerová)

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How do we define recurrence in *Clostridium difficile* infection?

Sir,

Clostridium difficile infection (CDI) causes a range of symptoms from mild diarrhoea to life-threatening pseudomembranous colitis [1—4]. The majority of patients experience a single episode of infection; however, despite treatment, some develop further episodes termed 'recurrent CDI' (rCDI) [1]. Enoch *et al.* stated that rCDI is associated with significant morbidity and mortality, with recurrence rates around 25% [5]. Recurrences are a serious, difficult and still unsolved management problem, increasing the length and overall cost of hospitalization [5]. rCDI is an arbitrary term and it is often difficult to define a true recurrence. Public Health England (PHE) define CDI as diarrhoea [at least three consecutive type 5—7 stools on the Bristol Stool Chart (BSC)] and a positive toxin test with clinical suspicion of CDI [6]. rCDI is defined as recurrence of diarrhoea within approximately 30 days of a previous CDI with a positive *C. difficile* toxin test [6]. Enoch *et al.* similarly defined rCDI as the reappearance of clinical CDI (microbiologically confirmed or clinically suspected) on review at day 30 [5]. At Queen Elizabeth Hospital Birmingham (QEHB), we used the PHE guidance to work out our rCDI rate over one year [6]. Using this definition, the recurrence rate was low; however, we suggest that this is misleading and the definition needs careful interpretation by healthcare workers. In addition, we describe some interventions that QEHB have put in place to reduce the rate of rCDI.

At QEHB (a tertiary referral teaching hospital), *C. difficile* testing is undertaken in line with national guidance using a three-stage algorithmic approach [7,8]. In the current study, we carried out an audit, analysing the BSC and clinical symptoms of all the patients who were positive for CDI by glutamate dehydrogenase (GDH) and nucleic acid amplification test (NAAT) in 2015 [7,9]. We used the PHE criteria to identify recurrence which is reappearance of diarrhoea within 30 days of a previous CDI episode (where diarrhoea resolved following treatment) with a positive toxin test [6].

During 2015, 327 stool samples from 249 inpatients tested positive for CDI via GDH and NAAT. There were 41 patients (16%) with more than one positive CDI test, representing either a recurrence or a relapse in infection. Of these 41 patients,