CSIRO PUBLISHING

Healthcare Infection, 2012, **17**, 127–132
http://dx.doi.org/10.1071/HI12029

An increase in community onset *Clostridium difficile* infection: a population-based study, Tasmania, Australia

Brett G. Mitchell[1,2,3,5] MAdvPract, DTN, CICP, BN

Fiona Wilson[1] RN

Alistair McGregor[1,4] FRACP, FRCPA

[1]Tasmanian Infection Prevention and Control Unit, Department of Health and Human Services, GPO Box 125, Hobart, Tas. 7001, Australia.
[2]Australian Catholic University, School of Nursing, Midwifery and Paramedicine, PO Box 256, Dickson, ACT 2602, Australia.
[3]University of Tasmania, School of Nursing and Midwifery, Private Bag 135, Hobart, Tas. 7000, Australia.
[4]Royal Hobart Hospital, 48 Liverpool Street, Hobart, Tas. 7000, Australia.
[5]Corresponding author. Email: bgmitc001@myacu.edu.au

Abstract. ***Background*:** In early 2012, the Tasmanian Infection Prevention and Control Unit identified a 53% increase in the number of cases of *Clostridium difficile* infection (CDI) identified in Tasmanian public hospitals. To understand this issue further, we undertook a population-based study. The aim of this research was to examine the epidemiology of CDI in Tasmania, with an overarching objective of understanding whether the increase seen in late 2011 was isolated to hospitals or represented a wider phenomenon.

***Methods*:** A population-based study design was used. All cases of laboratory diagnosed CDI that occurred during 2010 and 2011 in Tasmania were identified. Association of the cases with healthcare were determined using national and international CDI surveillance definitions.

***Results*:** A total of 459 cases of CDI from 438 individuals were identified. The incidence of CDI for the study period was 45 per 100 000 persons per year, 95% CI [41–49]. The relative risk (RR) of CDI was significantly higher in females, compared with males, RR 1.27, $P = 0.01$, 95% CI [1.06–1.54]. We estimate that the incidence of community associated CDI increased from 10 per 100 000 population in 2010, 95% CI [7.5–13.2] to 17 per 100 000 population in 2011 95% CI [14–21.5].

***Conclusion*:** Tasmania experienced a sudden and substantial increase in the number of CDI cases in late 2011. This was most likely linked to transmission and infection pathways in the community, not inside hospitals. This hypothesis requires further testing on a larger scale.

Received 19 July 2012, accepted 18 October 2012, published online 21 November 2012

Background

Clostridium difficile is a bacterium that commonly causes diarrhoea in hospitalised patients. The spectrum of disease caused by *C. difficile* ranges from uncomplicated diarrhoea through to pseudo-membranous colitis and toxic megacolon. The condition is frequently described as '*Clostridium difficile*-associated diarrhoea' (CDAD) or, more recently, '*Clostridium difficile* infection' (CDI).[1–4] Surveillance for CDI primarily focuses on the hospital environment, yet not all cases of CDI occur in hospitals or can be attributed to the hospital in which the diagnosis of CDI was made. In Tasmania, the Tasmanian Infection Prevention and Control Unit (TIPCU) has undertaken continuous surveillance of CDI in acute public hospitals since 2009 and these data are published quarterly on its website.

In early 2012, the TIPCU identified a marked increase in the number of CDI cases in Tasmanian Public Hospitals that occurred in the last quarter of 2011.[5] The TIPCU immediately issued an alert to hospitals and reinforced information about infection prevention and control strategies. To further investigate this increase, we designed and conducted a study to examine whether the cause of the increase in CDI could be identified at the population level.

Methods

The aim of this research was to examine the epidemiology of CDI in Tasmania. Its overarching objective was to understand whether the increase seen in late 2011 was isolated to hospitals or represented a wider phenomenon.

Implications

- Recent increases in *Clostridium difficile* infection in Tasmania have been driven by increases in community associated cases.
- Insight into the epidemiology of this infection at a population level.

By comparing data from 2010 and 2011, the following questions were addressed:

1. What was the overall incidence of CDI?
2. What was the incidence of hospital identified CDI?
3. What was the incidence of non-hospital identified CDI?
4. What was the incidence, categorised by case exposure of hospital identified CDI?

The term 'case exposure' describes the physical location and relationship to the healthcare system of the individual with CDI at the time of its onset. The definitions of case exposures and the terminology used are consistent with national and international CDI surveillance definitions.[2,6]

The report also explored issues that could not be addressed by the current CDI surveillance system in Tasmania.

Design, setting and timeframe

A population-based study design was used. All cases of laboratory-diagnosed CDI that occurred in Tasmania in 2010 and 2011 were identified. The use of the Public Health Act (1997) was used to support this investigation.

Selection of cases of CDI

A case of CDI was defined as an infection in a person who had a positive stool sample result for *C. difficile*, using either a laboratory assay (enzyme immunoassay or polymerase chain reaction) detecting toxin A and/or toxin B, or culture, resulting in the isolation of *C. difficile* that was subsequently shown to produce toxin A and/or toxin B. Details of all cases of CDI identified by all microbiology laboratories in Tasmania between 1 January 2010 and 31 December 2011 were provided to the TIPCU. Microbiology laboratories within Tasmania only test diarrhoeal stool samples for *C. difficile.*

In defining cases of CDI, two exclusion criteria were used, consistent with national CDI surveillance definitions:[7] (1) cases occurring within 8 weeks of a previous positive sample; and (2) persons aged less than 2 years old.

Classification of cases

Cases of CDI were first classified as either hospital-identified or non-hospital-identified. Hospital-identified cases were defined as CDI diagnosed in a patient attending an acute care facility. This is consistent with the Australian national CDI surveillance definitions.[6,7] Non-hospital-identified cases were defined as CDI diagnosed in a patient attending or residing in a healthcare facility or service other than an acute care facility. The total number of cases of CDI equals the total number of cases of hospital identified CDI plus the number of non-hospital identified cases of CDI.

Public hospital identified cases were classified into the following four exposure groups, based on national and international CDI surveillance definitions:[6,7]

- Healthcare-associated, healthcare facility onset (HCA HCF): cases of CDI that occurred $\geq$48 h after admission.
- Healthcare-associated, community onset (HCA COM): cases of CDI where symptom onset occurred in the community or $\leq$48 h after admission to a healthcare facility, provided that the onset of symptoms was less than 4 weeks after the last discharge from a healthcare facility where the patient had had a length of stay of $\geq$48 h.
- Community-associated (COM): cases of CDI where symptom onset was in the community or within 48 h of admission to a public hospital provided that symptom onset was more than 12 weeks after the last discharge from a healthcare facility in which skilled nursing care is provided, excluding residential aged care.
- Indeterminate onset (IND): cases that do not fit any of the above criteria for exposure setting (that is, onset in community between 4 and 12 weeks of discharge from a healthcare facility in which skilled nursing care is provided, excluding residential aged care.

Cases from private hospitals were categorised into three exposure groups, namely HCA HCF, COM and UN. The categories HCA COM and IND were not used as we could not obtain consistent data regarding previous contact with healthcare facilities in these cases and, therefore, we could not accurately determine cases of HCA COM or IND identified in private hospitals. Figure 1 summarises the categorisation of CDI cases.

Data collection

For each CDI case, the following data were obtained: case initials, age at time of specimen collection, sex, location where specimen was collected (where known), laboratory, postcode (home), date of specimen collection, date of admission to hospital if patient admitted. Only deidentified patient data was used, meaning that no person with CDI could be identified from this study. The use of initials and date of birth was sufficient to manage duplicate cases of CDI, consistent with the Australian national CDI surveillance definitions.[6,7]

Data analysis

Descriptive analysis on the characteristics of the admissions was performed in SPSS Version 20.0.[8] New variables were computed in SPSS based on the data collected. To test for normal distribution, data were analysed using Q-Q plots and the Kolmogorov–Smirnov test. The calculation of relative risk was performed to compare occurrence of CDI in different groups. Fisher's exact test was used to calculate confidence intervals.

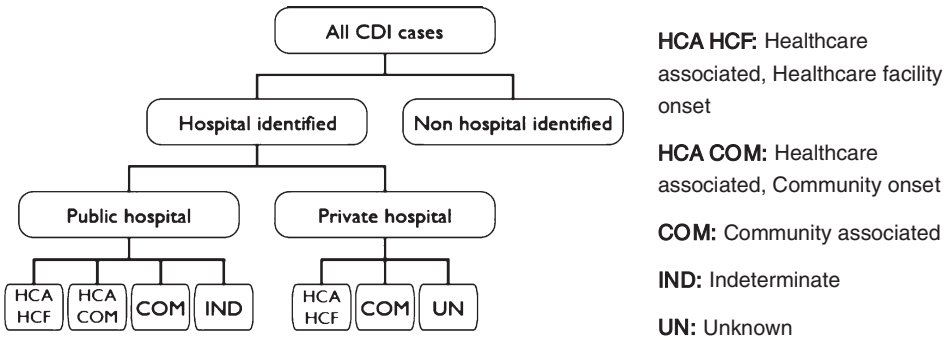

Fig. 1. Categorisation of CDI cases used in this study.

Results

Overview

The incidence of CDI for the study period was 45 per 100 000 persons per year, 95% CI [41–49]. The incidence of CDI by infection type and year is summarised in Table 1. This shows a substantial increase in the total number of CDI cases between 2010 and 2011, paralleled by an increase in hospital identified CDI during the same period. By contrast, the number of non-hospital identified cases remained stable over time.

A total of 459 cases of CDI from 438 individuals were identified. Of these, 198 (43.1%) were male. The age distribution was not normal, with a median age of 71 years (Table 2).

Incidence of CDI by sex and age group

Table 2 provides details on the incidence of CDI in Tasmania by sex and age group. Over the total study period the incidence of CDI was 39.5 per 100 000 in males and 50.5 per 100 000 in females. In 2010 there was no significant difference between males and females in the incidence of CDI, while in 2011, the incidence of CDI was significantly ($P = 0.04$) higher in females. The relative risk (RR) of CDI was significantly higher in females, compared with males, RR 1.27, $P = 0.01$, 95% CI [1.06–1.54]. Using the 70–79 year age group as a reference category, as the median age fell in this age group, the relative risk of CDI was significantly higher in the 80–89 year age group, RR 1.42, 95% CI [1.09–1.87] and in the 90+ age group, RR 2.11, 95% CI [1.37–3.24].

Table 1. Incidence of *Clostridium difficile* infection per 100 000 population in Tasmania during 2010–2011
CDI, *Clostridium difficile* infection

Infection type	2010			2011		
	Cases	Incidence per 100 000 population	95% CI	Cases	Incidence per 100 000 population	95% CI
Hospital-identified CDI	158	31.1	26.5–36.4	232	45.4	39.8–51.6
Non-hospital-identified CDI	34	6.7	4.6–9.4	35	6.9	4.9–9.4
Total	192	37.9	32.7–43.6	267	52.3	46.2–58.9

Table 2. Incidence of CDI per 100 000 population in Tasmania during 2010–2011, categorised by sex and age

Characteristic	2010			2011		
	Cases	Incidence per 100 000 population	95% CI	Cases	Incidence per 100 000 population	95% CI
Sex						
Male	84	33.6	26.8–41.6	114	45.5	37.6–54.5
Female	108	42.0	34.6–50.7	153	58.9	50.1–68.8
Age group						
0–9	0	N/A	N/A	5	7.7	2.5–18.0
10–19	4	5.9	1.6–15.0	6	8.9	3.3–19.4
20–29	9	14.8	6.7–28.0	15	24.3	13.6–40.1
30–39	4	6.5	1.8–16.8	17	28.4	16.6–45.5
40–49	9	12.7	6.2–23.3	14	19.9	10.9–33.3
50–59	25	35.3	23.3–51.3	29	40.3	27.5–57.1
60–69	28	49.4	32.9–71.5	53	90.1	68.2–117
70–79	54	161.1	121–210	69	202.0	157.0–256.0
80–89	49	275.0	203.5–363.6	44	243.7	177.0–327.1
90+	10	318.4	161.7–567.5	15	445.9	259.1–718.9

Incidence of CDI by place of residence

The total number of CDI cases in residents from Southern Tasmania increased from 114 to 162 between 2010 and 2011. A similar increase occurred in Northern Tasmania, where the number of cases rose from 23 to 44. There was a very small decrease in CDI cases in the North West of Tasmania, falling from 50 to 47 cases. The remaining cases were from interstate or overseas or the residential status was unknown.

Types of CDI: hospital identified versus non-hospital identified

A total of 390 cases of hospital-identified CDI occurred during the study period, equating to an incidence of 38.3 cases per 100 000 population, 95% CI [34.6–42.3]. There were 158 and 232 cases of hospital-identified CDI that occurred in 2010 and 2011, respectively. The median time from admission to CDI diagnosis, as defined by the faecal collection date, was 4 days (range: 0–291 days). Forty-five percent of hospital-identified cases of CDI were male. The median age of persons with hospital-identified CDI was 72 years. The case exposure classification of hospital identified cases of CDI, based on national surveillance definitions are provided in Table 3. These show a significant increase in the incidence of community assocaited CDI between 2010 and 2011. Of the 120 HCA HCF onset cases of CDI, the median time from admission to diagnosis was 7 days (range 2–291 days) and the median age of cases was 73 years. The median age of cases of community associated CDI was 70 years.

A total of 69 cases of non-hospital identified CDI occurred. No significant increase in this category of CDI occurred between 2010 and 2011, with 34 and 35 cases reported during 2010 and 2011, respectively. The median age of patients with non-hospital identified CDI was 63 years.

Discussion

This study was able to document the incidence of CDI at a population level by summarising data on the incidence of hospital-identified and non-hospital-identified CDI cases. It also examined the incidence of CDI in various case exposure categories. The data clearly show an increase in the total number of CDI cases between 2010 and 2011, mostly accounted for by community associated infections identified within acute hospitals.

Incidence of CDI

The overall incidence of CDI was 45 cases per 100 000 population. *C. difficile* infection in Tasmania appears more prevalent than in the rest of Australia (incidence rate = 25.6) based on data from 48 laboratories.[9] One possible reason for the higher reported incidence of CDI in Tasmania is that Tasmania has a more robust system to identify and report cases of CDI than other jurisdictions. Other Australian studies on CDI have been based solely on hospital data and, hence, are not necessarily representative of the whole population.[10,11] Our data also suggest that the risk of CDI increases with age, which is consistent with the literature.[12–14]

Using incidence rates from this study and an estimated Australian population of 23 million, we calculate that there would be 10 350 cases of CDI occurring annually in Australia, compared with the estimate of 5900 cases using the incidence rate of Ferguson *et al*.[9]

Hospital-identified CDI

The number of hospital-identified cases of CDI increased between 2010 and 2011. We examined the case exposure of these persons more closely and were able to ascertain that there was a significant increase in the number of community-associated cases of CDI between 2010 and 2011. Therefore, we believe that the main driver of the observed increase were community-associated infections. Our data also indicates an increase in the number of indeterminate and unknown case exposure classifications for hospital-identified CDI between 2010 and 2011. In the case of indeterminate cases of CDI, there is no obvious reason for this increase. The increase in the number of cases being classified as ‘unknown’ was due to a lack of information to classify cases.

Non-hospital-identified CDI

Our study identified 69 cases of CDI that were not identified in an acute care hospital. These cases were identified in settings other than acute hospitals and could include community health centres, general practitioner clinics or residential and aged care facilities. We could not identify whether these 69 cases were attributable to a specific healthcare facility as this

Table 3. Summary of the incidence of hospital identified cases of *Clostridium difficile* infections per 100 000 population in Tasmania during 2010–2011

HCA HFO, healthcare-associated, healthcare facility onset; HCA CO, healthcare-associated, community onset

Case exposure classification[A]	2010			2011		
	Cases	Incidence per 100 000 population	95% CI	Cases	Incidence per 100 000 population	95% CI
HCA HCF	120	23.6	19.7–28.2	132	25.9	21.7–30.1
HCA COM	13	2.5	1.4–4.4	17	3.3	1.9–5.3
Community	17	3.4	2.0–5.4	54	10.6	8.0–13.8
Indeterminate	5	1.0	0.3–2.3	13	2.5	1.4–4.4
Unknown	3	0.6	0.1–1.7	16	3.1	1.8–5.1

[A]Based on national surveillance definitions.[6,7]

information was not available. Due to these limitations, these 69 cases cannot be accurately defined as community-associated CDI, although we believe that this is the most likely scenario.

Most studies examining CDI use data from hospitals and do not explicitly consider the role of infections in the community.[15] If we assume that all cases of non-hospital-identified CDI were community-associated CDI and these data were combined with cases of community-associated CDI from hospital-identified cases, there would be a total of 51 cases of community-associated CDI in 2010 and 89 cases in 2011. This would be a significant increase, with the incidence of community-associated CDI being calculated as 10 per 100 000 population in 2010, 95% CI [7.5–13.2] and 17 per 100 000 population in 2011 95% CI [14–21.5]. Over the 2 years, the incidence of community-associated CDI would be 13.7 per 100 000 population. Based on these data, we estimate that there are 3151 CDI cases that originate in the community in Australia each year.

Potential causes for the observed increase in CDI in Tasmania

There are several potential causes for the observed increase in CDI including: (1) a change of circulating strains of *C. difficile*; (2) changes in laboratory practice; (3) an increase in the use of antimicrobials; (4) changes in infection control practices such as hand hygiene or isolation of patients with CDI; and (5) an increased risk of transmission in the healthcare environment.

There is some data available regarding the types of strains of *C. difficile* currently circulating within Tasmania and while this does not suggest that the strains have changed over the past 3 years, the data is limited.

During the study period, one laboratory changed their testing methodology. This laboratory previously tested all diarrhoeal faecal samples using an enzyme immunoassay (EIA). These faecal samples were also cultured and culture positive isolates were subsequently tested by Techlab (C.DIFF QUICK CHEK COMPLETE), a combination assay that detects glutamate dehydrogenase (GHD) antigen), toxin A and toxin B. In August 2010, the laboratory changed their practice. Samples that gave discordant GDH and toxin results (one positive and the other negative) were subsequently tested using polymerase chain reaction (PCR). The practice of stool culture continued to provide isolates for subsequent typing, if required, and culture positive specimens that were negative using the Techlab also underwent PCR (L. Cooley, pers. comm.). This change did not alter the detection of CDI, as evidenced by similar numbers of cases being identified in the months following this change. Importantly, during this time, there was no change in the criteria used to determine which faeces samples were tested for *C. difficile* and as a result, no significant changes in testing rates.

Although there was no change in the criteria used to determine which faeces samples were tested for *C. difficile* by laboratories during the study period, it is important to note that in the case of private laboratories, a specific request to test for *C. difficile* was required, whereas in public hospital laboratories testing for *C. difficile* could potentially be instituted by the laboratory itself, when deemed appropriate. This has the potential to affect case ascertainment of community cases of CDI, although the size of the effect is unknown. As no change to testing criteria was made during the study period, this would not explain the increase witnessed.

All public hospitals in Tasmania contribute to the National Antibiotic Utilisation Surveillance Program (NAUSP). Data from the NAUSP do not suggest that the observed increase in CDI was caused by an increase in specific classes of antibiotics known to increase the risk of CDI. We did not have data available on antibiotic use in the community so we cannot make comments in relation to this.

Hand hygiene compliance in Tasmanian hospitals has increased from 2010 to 2011,[4] but as this study was not designed to evaluate the effect of hand hygiene on the incidence of CDI, no conclusions from this can be drawn. Hospitals in Tasmania isolate persons with CDI in accordance with the National Health and Medical Research Council's guidelines and protocols published by the Australian Infection Control Association and Australasian Society for Infectious Diseases.[16,17] Compliance with infection control guidelines was not evaluated so we cannot definitively make many any conclusions regarding whether non-compliance with transmission based precautions played any role in the observed increase. That said, we believe this is unlikely, particularly given the increase in CDI was largely caused by community associated cases of CDI.

Work is underway in Tasmania on methods to evaluate environmental cleanliness in hospitals; however, there is no data to suggest there were any changes to environmental cleaning methods during the study period.[18]

In summary, data from our study shows that there was an increase in hospital identified cases of CDI between 2010 and 2011. From the data available, we believe the primary cause of this was an increase in cases of community-associated CDI. Two plausible explanations for this increase are a change in the circulating strains of *C. difficile*, an increase in the use of antimicrobials in the community, or both. The former is being further investigated as part of a nationally coordinated response to the problem of CDI, whereas reliable data on antimicrobial use in the community are not available.

We suggest that more comprehensive population based studies are required in Australia to obtain more robust quantitative data on the incidence of CDI, and that work continues to define the identity and comparative virulence of different strains of *C. difficile* circulating within Australia. It will also be helpful to obtain reliable data on the use of antimicrobial agents in the wider community.

Limitations

Hospital-identified CDI cases were classified by their exposure to a healthcare facility. This could not be done for the non-hospital-identified CDI cases as information about

contact with hospitals was not available for these patients. Therefore, as we combined data from community-associated cases of hospital-identified CDI with non-hospital-identified cases of CDI to calculate the total incidence of community-associated CDI, the incidence may have been overestimated. This may have occurred because an assumption was made that all non-hospital-identified cases of CDI originated in the community. Paradoxically, this limitation does not change our main conclusion that the most likely drivers of the observed increase in CDI cases in Tasmania were probably associated with community associated transmission and infection pathways.

Conclusion

Tasmania experienced a sudden and substantial increase in the number of CDI cases in late 2011. We undertook a population based study to examine the incidence of CDI in Tasmania between 2010 and 2001. This showed that the observed increase in CDI was most likely linked to transmission and infection pathways in the community, not inside hospitals. This hypothesis, based on limited data and analysis, requires further robust testing on a larger scale.

Conflict of interest

One of the authors of the paper is part of the journal Editorial team. This author played no role in the editorial decision making of this paper whatsoever. All other authors have no conflicts to declare.

Funding

No funding was received to support this research or the publication of this manuscript.

Acknowledgements

The authors wish to acknowledge Professor Tom Riley (PathWest) and his team for their work in typing *C. difficile* isolates. We also wish to acknowledge the microbiology laboratories in Tasmania for their assistance in providing us with the necessary data.

References

1. Heinlen L, Ballard JD. *Clostridium difficile* infection. *Am J Med Sci* 2010; 340(3): 247–52. doi:10.1097/MAJ.0b013e3181e939d8
2. McDonald LC, Coignard B, Dubberke E, Song X, Horan T, Kutty PK, *et al*. Recommendations for surveillance of *Clostridium difficile*-associated disease. *Infect Control Hosp Epidemiol* 2007; 28(2): 140–5. doi:10.1086/511798
3. Stuart R, Marshall C. *Clostridium difficile* infection: a new threat on our doorstep. *Med J Aust* 2011; 194(7): 331–2.
4. Van Gessel H, Riley T, McGregor A. *Clostridium difficile* infection: an update for infection control practitioners. *Healthc Infect* 2009; 14: 115–8. doi:10.1071/HI09105
5. Mitchell B, McGregor A, Wells A, Wilson F. Tasmanian Acute Public Hospitals Healthcare Associated Infection Surveillance Report, Report No.11. Hobart: Tasmanian Infection Prevention and Control Unit, Department of Health and Human Services; 2012.
6. Van Gessel H, McCann R, Peterson A, Cope C, Wilkinson I, Mitchell B, *et al*. Implementation Guide for Surveillance for *Clostridium difficile* Infection. Sydney: Australian Commission on Safety and Quality in Health Care; 2011.
7. Australian Commission on Safety and Quality in Health Care. Data Dictionary and Collection Guidelines for the Surveillance of Healthcare Associated Infections: *Staphylococcus aureus* bacteraemia & *Clostridium difficile* Infection. 3rd edn. Sydney: Australian Commission on Safety and Quality in Health Care; 2010.
8. International Business Machines Corporation. SPSS Statistics. Version 20.0. New York: IBM; 2011.
9. Ferguson JK, Cheng AC, Gilbert GL, Gottlieb T, Korman T, McGregor A, *et al*. *Clostridium difficile* laboratory testing in Australia and New Zealand: national survey results and Australasian Society for Infectious Diseases recommendations for best practice. *Pathology* 2011; 43(5): 482–7. doi:10.1097/PAT.0b013e328348c9b4
10. Mitchell B, Ware C, McGregor A, Brown S, Wells A. *Clostridium difficile* infection in Tasmanian Public Hospitals 2006–2010. *Healthc Infect* 2011; 16(3): 101–6. doi:10.1071/HI11009
11. Van Gessel H. Measuring the incidence of *Clostridium difficile*-associated diarrhoea in a group of Western Australian hospitals. *Healthc Infect* 2008; 13(2): 56–62. doi:10.1071/HI08010
12. McFarland LV, Surawicz CM, Stamm WE. Risk factors for *Clostridium difficile* carriage and *C. difficile*-associated diarrhea in a cohort of hospitalized patients. *J Infect Dis* 1990; 162(3): 678–84. doi:10.1093/infdis/162.3.678
13. Starr JM, Martin H, McCoubrey J, Gibson G, Poxton IR. Risk factors for *Clostridium difficile* colonisation and toxin production. *Age Ageing* 2003; 32(6): 657–60. doi:10.1093/ageing/afg112
14. Vaishnavi C. Established and potential risk factors for *Clostridum difficile* infection. *Indian J Med Microbiol* 2009; 27(4): 289–300. doi:10.4103/0255-0857.55436
15. Barbut F, Richard A, Hamadi K, Chomette V, Burghoffer B, Petit JC. Epidemiology of recurrences or reinfections of *Clostridium difficile*-associated diarrhea. *J Clin Microbiol* 2000; 38(6): 2386–8.
16. Stuart R, Marshall C, McLaws M, Boardman C, Russo R, Harrington G, *et al*. ASID/AICA position statement – infection control guidelines for patients with *Clostridium difficile* infection in healthcare settings. *Healthc Infect* 2011; 16: 33–9.
17. National Health and Medical Research Council. Australian Guidelines for the Prevention and Control of Infection in Healthcare. Canberra: National Health and Medical Research Council; 2010.
18. Mitchell B, Wilson F, McGregor A, Dancer S, eds. Evaluating Environmental Cleanliness in Hospitals and other Healthcare Settings. What are the Most Effective and Efficient Methods to Use? Hobart: Department of Health and Human Services; 2012.