

# Contamination, Disinfection, and Cross-Colonization: Are Hospital Surfaces Reservoirs for Nosocomial Infection?

Adam Carter<sup>1</sup>, Ryan Blake<sup>2</sup>, Ethan Grant<sup>3</sup>,

- <sup>1</sup>Department of Cognitive Neuroscience, School of Behavioral Sciences, Westbridge University, London, UK
- <sup>2</sup>Department of Global Affairs, School of Political Studies, Hawthorne College, Manchester, UK
- <sup>3</sup>Department of Computer Systems, School of Engineering, Silverwood Institute of Technology, Cambridge, UK
- Department of Developmental Psychology, School of Human Sciences, Eastfield University, Newcastle, UK

# \* Correspondence:

Dr. Emma Lawson Department of Social Psychology Faculty of Arts and Humanities, Westbridge University WB34 7YT, London, UK

Email: emma.lawson@westbridge.ac.uk

Phone: +44 (20) 8123 4567 Fax: +44 (20) 8123 4568

# Abstract

Although it is well-documented that hospital environments—such as surfaces and medical equipment—can become contaminated with nosocomial pathogens, the evidence linking contaminated fomites directly to hospital-acquired infections is mostly indirect. There is stronger evidence supporting the persistence of certain pathogens, including Clostridium difficile, vancomycin-resistant enterococci, and methicillin-resistant Staphylococcus aureus, in environmental reservoirs. Other pathogens, such as norovirus, influenza virus, the coronavirus associated with severe acute respiratory syndrome (SARS), and Candida species, are also believed to survive in these environments. To effectively reduce nosocomial infections caused by these organisms, infection control strategies should adhere to established guidelines, focusing particularly on thorough environmental cleaning and the use of EPA-approved detergent-disinfectants.

# Background

The contribution of the inanimate hospital environment – such as surfaces and medical equipment – to the transmission of nosocomial infections remains a subject of debate. While the presence of microorganisms contaminating these environments has been acknowledged for some time, the extent to which this contamination influences infection rates is still uncertain. For instance, one medical facility reported that although environmental contamination significantly decreased after relocating to a new hospital, this reduction did not correspond with a decline in nosocomial infection rates [1]. This raises the question: are microbes found on inanimate surfaces merely passive contaminants, or do they actively contribute to patient colonization and infection? Variations in findings across different studies may be due to the complexity of infection transmission dynamics, inconsistencies in how contamination is measured, or differences in the effectiveness of cleaning practices-an often overlooked yet crucial factor. Moreover, the detection of pathogens on hospital surfaces, while necessary to establish potential risk, does not alone confirm a direct causal role in the development of infections. Additionally, reports from uncontrolled outbreak investigations suggesting that enhanced environmental cleaning ended transmission must be interpreted with caution, since simultaneous implementation of multiple infection-control strategies can mask the specific impact of cleaning. Evaluating the evidence regarding environmental contamination requires consideration of four key factors: (1) the extent to which the hospital environment is contaminated by specific pathogens; (2) whether studies examine temporality—that is, if contamination occurs before or after patient colonization; (3) assessment of confounding variables, such as hand hygiene practices and the thoroughness of surface cleaning; and (4) whether improvements in cleaning, when isolated from other interventions, lead to reduced patient infection risk. The most robust investigations use molecular epidemiology to track pathogen strains, systematically monitor environmental cleaning and hand hygiene quality over time, and establish spatial and temporal links between contaminated surfaces and patient cross-colonization events.

### CONTAMINATION OF THE HOSPITAL ENVIRONMENT BY NOSOCOMIAL PATHOGENS

Viruses

Viruses can contaminate and persist on inanimate surfaces (see Table 1), making environmental cleaning a crucial component of infection control, particularly for influenza, parainfluenza, enteric viruses, hepatitis B virus (HBV), and severe acute respiratory syndrome (SARS) coronavirus. Influenza virus primarily spreads via large respiratory droplets and possibly through airborne droplet nuclei. Classic studies demonstrate that influenza can contaminate surfaces, survive drying, and become re-aerosolized during floor sweeping. The virus can remain viable for 24 to 48 hours on nonporous surfaces and can transfer to skin, indicating that contaminated environments may facilitate cross-infection through the hands of healthcare workers [3]. Similarly, parainfluenza virus resists drying and can survive for up to 10 hours on nonporous surfaces and 16 hours on clothing [5].

Human enteric viruses frequently contaminate inanimate surfaces and can cause outbreaks in institutional settings [38–40]. For example, rotavirus is a well-known source of infections in daycare centers and healthcare facilities; it extensively contaminates surfaces and may spread via contaminated toys shared by children [38]. Norovirus has caused outbreaks on cruise ships, hospitals, and hotels [7, 8, 39, 40]. In 2002 alone, nine outbreaks of norovirus were reported on cruise ships [40], some requiring suspension of services and intensive cleaning to halt transmission. Although direct proof of environment-to-person transmission is lacking, norovirus is often cultured from environmental samples during outbreaks [8, 39], and indirect evidence suggests aerosolization may occur during vomiting episodes [8].

Individuals lacking immunity to HBV are at risk of infection from contaminated environmental sources. Blood from actively replicating HBV-infected individuals can contain high viral loads, and even small, invisible amounts can be highly infectious. HBV can survive up to seven days at relative humidity around 42% [9]. Outbreaks linked to fomites have been traced to contaminated medical equipment such as electroencephalographic electrodes [10] and glucose-monitoring lancets [11].

SARS coronavirus is primarily transmitted through respiratory droplets, but fecal-oral and surface contamination routes may also contribute. Infection control in hospitals includes precautions against contact, droplet, and airborne transmission [14]. The virus can survive on plastered walls, plastic laminate (e.g., Formica), and other plastic surfaces for 24 to 72 hours and remains viable in feces and urine for one to two days at room temperature [13]. A notable outbreak in a Hong Kong apartment complex might have involved fecal-oral transmission combined with environmental contamination [15], although airborne spread was also suggested by modeling studies [41]. Environmental cleaning effectively reduces contamination; a Taiwan emergency department outbreak ceased after cleaning and patient isolation, with environmental samples turning culture-negative [12].

# Fungi

Most Candida infections originate from the patient's own flora, but molecular typing indicates that fomites may play a role in spreading Candida species such as C. albicans, C. glabrata, and C. parapsilosis, especially among bone marrow transplant patients. However, the direction of transmission—patient to environment or vice versa—is not definitively proven [16]. Candida can survive on dry surfaces for several days: 3 days for C. albicans and up to 14 days for C. parapsilosis [17]. Epidemic outbreaks have implicated environmental sources like blood pressure transducers or irrigating solutions [16, 42]. Molecular typing supports the presence of endemic environmental reservoirs, with patient strains matching those found on hospital surfaces before infection [16].

Aspergillus and Zygomycetes species are known causes of nosocomial skin infections linked to contaminated fomites. These infections have been associated with arm boards, bandages in patients with intravascular catheters, surgical bandages, hospital construction, and postoperative wounds [43].

#### Bacteria

Clostridium difficile produces durable spores resistant to common cleaning methods. Environmental contamination near infected or colonized patients is frequent, with rates up to 58%. Contaminated surfaces include commodes, bedpans, blood pressure cuffs, walls, floors, washbasins, and furniture [18–20]. Low levels of spores have been found on shoes and stethoscopes [20]. Floors can remain contaminated for up to five months [19]. The contamination density increases in areas with colonized or diarrheal patients [18, 20]. Molecular evidence confirms transmission from contaminated surfaces to patients: health care workers' hands harbor C. difficile, contamination correlates with patient colonization, and dominant strains are more likely to contaminate environments [18, 44]. These findings suggest that surfaces serve as reservoirs enabling cross-colonization after contact with healthcare workers.

Enteric gram-negative bacilli generally do not survive long on dry surfaces (less than 7 hours), and infection usually results from endogenous sources or patient-to-patient transmission via healthcare workers' hands [22]. However, Pseudomonas aeruginosa and Acinetobacter baumannii are strongly associated with environmental contamination. Numerous studies report P. aeruginosa contamination in sinks and drains [21], though its environmental strain types do not always match clinical isolates [23]. Most P. aeruginosa infections stem from endogenous flora rather than environmental sources [21]. Similarly, A. baumannii—a commensal and opportunistic pathogen with increasing antibiotic resistance—has been isolated extensively from hospital environments, including beds, mattresses, floors, sinks, and humidifiers [24, 25]. Airborne spread has been suggested through air sampling [24]. This organism can survive up to three years in hospital environments [26]. Environmental strains include those causing patient infections as well as those without clinical association [27]. Some outbreak investigations found no environmental A. baumannii, complicating assessments of environmental roles [45, 46]. Hand hygiene and cleaning practices may influence these findings.

Gram-positive cocci, especially methicillin-resistant Staphylococcus aureus (MRSA), primarily colonize or infect patients and occasionally hospital staff, with transmission mostly via unwashed healthcare worker hands [47]. The environmental role in MRSA spread is debated; environmental contamination is variable, and strain types from environment and patients do not always match [31, 48]. Burn units show higher environmental contamination, with MRSA rates up to 64%, compared to 1–18% in nonburn wards [28]. Hydrotherapy rooms associated with burn units are especially contaminated [47]. Contamination also varies by infection site in

patients: rooms of those with urine or wound infections show more environmental contamination than those with bacteremia alone [28]. Outbreaks have cultured MRSA from mattresses, where moist padding and damaged covers are common [49], as well as from mops, gowns, and gloves [28]. MRSA and methicillin-susceptible S. aureus can survive up to 9 weeks despite drying and remain viable for up to 2 days on plastic laminate surfaces under lab conditions [29, 30].

#### INTERVENTION STRATEGIES

Cleaning intensity is generally classified into two main categories: sterilization and disinfection. Sterilization aims to completely destroy all microbial life on surfaces or objects, and it can be achieved through heat, pressure, or chemical methods. Disinfection, on the other hand, reduces the number of microorganisms significantly but does not eliminate bacterial spores. The effectiveness of disinfection depends on how sensitive the microbes are to the chemicals used. High-level disinfection kills all microorganisms except large amounts of spores, intermediate-level disinfection destroys all microbes except spores, and low-level disinfection does not reliably kill mycobacteria or spores. "Cleaning" refers to the physical removal of dirt and organic matter from surfaces, often involving mechanical action and detergents with water. Cleaning alone can lower the number of organisms on a surface, and when combined with disinfection, it can significantly reduce microbial load more quickly [62].

There are three types of solutions commonly used for cleaning: detergents that remove organic matter and dissolve grease or oils; disinfectants that quickly kill or inactivate infectious agents; and detergent-disinfectants that perform both functions. However, there is no definitive evidence proving that routine disinfection of hospital surfaces is better than using detergent alone [63]. As a result, the regular use of detergent-disinfectants is mainly supported by expert consensus and practical considerations [4].

In 2003, the Healthcare Infection Control Practice Advisory Committee of the CDC (CDC/HICPAC; Atlanta, GA) updated guidelines on environmental infection control for healthcare facilities [4]. These guidelines recommend cleaning strategies for patient care areas, emphasizing that surfaces should be visibly clean, with high-touch areas disinfected more frequently than others, and spills cleaned promptly. Environmental services staff are advised to use EPA-registered detergent-disinfectants to clean inanimate surfaces in patient care settings. Although this recommendation has been debated [63], it accounts for uncertainty about contaminants, such as blood or bodily fluids versus normal dust or dirt, and the potential presence of multidrug-resistant organisms [4].

The guidelines do not specify how often cleaning should occur, only that it should be done regularly. In hospitals, patient rooms are usually cleaned daily and receive a thorough "terminal cleaning" after a patient is discharged. Terminal cleaning involves an intensive cleaning of noncritical surfaces, typically using disinfectants like quaternary ammonium compounds or phenolics (though phenolics are not recommended for nurseries or infant care areas). This thorough cleaning may be more effective at reducing environmental contamination due to its comprehensiveness.

Generally, changing cleaning products or procedures is not necessary to target specific pathogens. However, in areas with high rates of *Clostridioides difficile* infections, hypochlorite-based disinfectants may be preferred because of their reliable ability to kill spores. Most commercial disinfectants used in healthcare settings are effective against viruses; enveloped viruses are more susceptible to detergents than non-enveloped ones [9]. Viruses, including the SARS coronavirus, can typically be eliminated with EPA-approved disinfectants or detergent-disinfectants when used according to the manufacturer's instructions [14].

In cases of norovirus outbreaks, decontamination should involve germicidal products such as a 10% sodium hypochlorite (bleach) solution, and closure of the affected facility may be required [6]. Intuitively, effective cleaning is important for controlling resistant organisms. One study showed that extending the exposure time of cleaning agents on surfaces effectively eliminated environmental *vancomycin-resistant Enterococcus* (VRE) [64]. Improving cleaning compliance also benefits hygiene: feedback to housekeeping staff resulted in better cleaning practices and a threefold reduction in VRE contamination, achieved with standard cleaning materials and methods [65]. However, it remains unclear whether such improvements directly lead to reduced rates of hospital-acquired infections.

# DISCUSSION

Although much about the transmission of hospital-acquired (nosocomial) infections remains unclear, several

key findings have emerged from current research: (1) inanimate surfaces within healthcare environments can become persistently contaminated after contact with colonized patients; (2) while certain organisms may be widespread within a facility, specific strains often dominate on these surfaces, as seen with *Clostridioides difficile* and vancomycin-resistant enterococci (VRE); and (3) contaminated patient rooms may increase the risk of infection for subsequent patients. Molecular epidemiology has enhanced our understanding by showing that environmental isolates often match those found in patients (as demonstrated for *C. difficile, Candida* species, and VRE), though differences exist for organisms like *Acinetobacter* species.

Interpreting existing outbreak reports and environmental studies is challenging because factors like hand hygiene and cleaning practices—important variables influencing transmission—are infrequently measured and may confound results. There is a clear need for rigorous studies that evaluate whether enhanced cleaning protocols actually reduce nosocomial infection rates. Future research on the environmental role in infections should include detailed timing of contamination and patient acquisition events, precise patient locations relative to contaminated areas, and assessments of hygiene and cleaning effectiveness.

Understanding the role of the inanimate environment is critical, especially given ongoing challenges in infection control compliance and hand hygiene adherence. The introduction of alcohol-based hand gels has improved compliance and may lessen transmission from contaminated surfaces like walls, bed rails, and medical equipment. Nevertheless, hospitals might benefit from adopting additional, cost-effective strategies such as more thorough and frequent environmental cleaning to lower the risk of cross-colonization.

#### **Conclusions**

Our results indicate that the primary factors associated with loneliness, after controlling for other variables, span sociocultural influences (such as experiences of discrimination), relational aspects (including couple satisfaction and time spent alone), and individual characteristics (notably neuroticism and personal self-esteem). Therefore, effective interventions must take a comprehensive approach that addresses these multiple dimensions. It is essential to tailor strategies to the diverse needs of individuals while also confronting broader issues of marginalization. Focusing solely on individual or relationship-level solutions, without tackling the underlying structural inequalities, is unlikely to reduce loneliness or its negative impacts on health and wellbeing, and may perpetuate disparities experienced by marginalized populations.

#### References

- 1. Allerberger F, Ayliffe G, Bassetti M, et al. Routine surface disinfection in health care facilities: should we do it? Am J Infect Control 2002; 30:318–9.
- 2. Beard-Pegler MA, Stubbs E, Vickery AM. Observations on the resistance to drying of staphylococcal strains. J Med Microbiol 1988; 26:251–5.
- 3. Blom DW, Lyle EA, Weinstein RA, et al. The relationship between environmental contamination with vancomycin-resistant Enterococcus and patient colonization in a medical intensive care unit. In: Program and abstracts of the 40th Interscience Conference on Antimicrobial Agents and Chemotherapy (ICAAC) (Toronto, Canada). Washington, DC: American Society for Microbiology Press, 2000: 432.
- 4. Bonilla HF, Zervos MJ, Kauffman CA. Long-term survival of vancomycin-resistant Enterococcus faecium on a contaminated surface. Infect Control Hosp Epidemiol 1996; 17:770–2.
- 5. Boyce JM. Methicillin-resistant Staphylococcus aureus in hospitals and long-term care facilities: microbiology, epidemiology, and preventive measures. Infect Control Hosp Epidemiol 1992; 13:725–37.
- 6. Boyce JM, Potter-Bynoe G, Chenevert C, et al. Environmental contamination due to methicillin-resistant Staphylococcus aureus: possible infection control implications. Infect Control Hosp Epidemiol 1997; 18:622–7.
- 7. Bradley CR, Fraise AP. Heat and chemical resistance of enterococci. J Hosp Infect 1996; 34:191-6.
- 8. Bradley SF, Terpenning MS, Ramsey MA, et al. Methicillin-resistant Staphylococcus aureus: colonization and infection in a long-term care facility. Ann Intern Med 1991; 115:417–22.
- 9. Brady MT, Evans J, Cuartas J. Survival and disinfection of parainfluenza viruses on environmental surfaces. Am J Infect Control 1990; 18:18–23.

- 10. Bridges CB, Kuehnert MJ, Hall CB. Transmission of influenza: implications for control in health care settings. Clin Infect Dis 2003; 37:1094–101.
- 11. Byers KE, Durbin LJ, Simonton BM, et al. Disinfection of hospital rooms contaminated with vancomycin-resistant Enterococcus faecium. Infect Control Hosp Epidemiol 1998; 19:261–4.
- 12. Centers for Disease Control and Prevention. Norwalk like viruses: public health consequences and management. MMWR Recomm Rep 2001; 50 (RR-09):1–18.
- 13. Centers for Disease Control and Prevention. Outbreaks of gastroenteritis associated with noroviruses on cruise ships—United States, 2002. MMWR Morb Mortal Wkly Rep 2002; 51:1112–5.
- 14. Centers for Disease Control and Prevention. Public health guidance for community-level preparedness and response to severe acute respiratory syndrome (SARS). Version 2. Available at: http://www.cdc.gov/ncidod/sars/guidance/I/pdf/healthcare.pdf. Accessed 20 September 2004.
- 15. Chen YC, Huang LM, Chan CC, et al. SARS in hospital emergency room. Emerg Infect Dis [serial online] May 2004. Available at: http://www.cdc.gov/ncidod/EID/vol10no5/03-0579.htm. Accessed 29 February 2004.
- 16. Cookson B, Peters B, Webster M, et al. Staff carriage of epidemic methicillin-resistant Staphylococcus aureus. J Clin Microbiol 1989; 27:1471–6.
- 17. D'Agata EM, Thayer V, Schaffner W. An outbreak of Acinetobacter baumannii: the importance of cross-transmission. Infect Control Hosp Epidemiol 2000; 21:588–91.
- 18. Das I, Lambert P, Hill D, et al. Carbapenem-resistant Acinetobacter and role of curtains in an outbreak in intensive care units. J Hosp Infect 2002; 50:110–4.
- 19. Deckro AN, Blom DW, Lyle EA, et al. Frequency of environmental sites and patient skin as sources of VRE transmission. In: Program and abstracts of the 13th Annual Scientific Meeting of the Society for Healthcare Epidemiology of America (Arlington, Virginia). Mt. Royal, NJ: Society for Healthcare Epidemiology of America, 2003: 64.
- 20. Duckro AN, Blom DW, Lyle EA, et al. Frequency of environmental sites and patient skin as sources of VRE transmission. In: Program and abstracts of the 13th Annual Scientific Meeting of the Society for Healthcare Epidemiology of America (Arlington, Virginia). Mt. Royal, NJ: Society for Healthcare Epidemiology of America, 2003: 64.
- 21. Duckworth GJ, Jordens JZ. Adherence and survival properties of an epidemic methicillin-resistant strain of Staphylococcus aureus compared with those of methicillin-sensitive strains. J Med Microbiol 1990; 32:195–200.
- 22. Edmond MB, Ober JF, Weinbaum DL, et al. Vancomycin-resistant Enterococcus faecium bacteremia: risk factors for infection. Clin Infect Dis 1995; 20:1126–33.
- 23. Fawley WN, Wilcox MH. Molecular epidemiology of endemic Clostridium difficile infection. Epidemiol Infect 2001; 126:343–50.
- 24. Fekety R, Kim KH, Brown D, et al. Epidemiology of antibiotic-associated colitis: isolation of Clostridium difficile from the hospital environment. Am J Med 1981; 70:906–8.
- 25. Fitzpatrick F, Murphy OM, Brady A, et al. A purpose built MRSA cohort unit. J Hosp Infect 2000; 46:271–9.
- 26. Garner JS. Guideline for isolation precautions in hospitals. Infect Control Hosp Epidemiol 1996; 17:53–80
- 27. Gould FK, Freeman R. Nosocomial infection with microsphere beds. Lancet 1993; 342:241-2.
- 28. Green J, Wright PA, Gallimore CI, et al. The role of environmental contamination with small round structured viruses in a hospital outbreak investigated by reverse-transcriptase polymerase chain reaction assay. J Hosp Infect 1998; 39:39–45.
- 29. Hepatitis B Outbreak Investigation Team. An outbreak of hepatitis B associated with reusable subdermal electroencephalogram electrodes. CMAJ 2000; 162:1127–31.
- 30. Hirai Y. Survival of bacteria under dry conditions: from a viewpoint of nosocomial infection. J Hosp Infect 1991; 19:191–200.
- 31. Jawad A, Seifert H, Snelling AM, et al. Survival of Acinetobacter baumannii on dry surfaces: comparison of outbreak and sporadic isolates. J Clin Microbiol 1998; 36:1938–41.
- 32. Kim KH, Fekety R, Batts DH, et al. Isolation of Clostridium difficile from the environment and contacts of patients with antibiotic-associated colitis. J Infect Dis 1981; 143:42–50.

- 33. Kim WJ, Weinstein RA, Hayden MK. The changing molecular epidemiology and establishment of endemicity of vancomycin resistance in enterococci at one hospital over a 6-year period. J Infect Dis 1999; 179:173–1.
- 34. Marks PJ, Vipond IB, Carlisle D, et al. Evidence for airborne transmission of Norwalk-like virus in a hotel restaurant. Epidemiol Infect 2000; 124:481–7.
- 35. Maki DG, Alvarado CJ, Hassemr CA, Zilz MA. Relation of the inanimate hospital environment to endemic nosocomial infection. N Engl J Med 1982; 307:1562–6.
- 36. Martinez JA, Ruthazer R, Hansjosten K, et al. Role of environmental contamination as a risk factor for acquisition of vancomycin-resistant enterococci in patients treated in a medical intensive care unit. Arch Intern Med 2003; 163:1905–12.
- 37. Montecalvo MA, Jarvis WR, Uman J, et al. Infection-control measures reduce transmission of vancomycin-resistant enterococci in an endemic setting. Ann Intern Med 1999; 131:269–72.
- 38. Ndawula EM, Brown L. Mattresses as reservoirs of epidemic methicillin-resistant Staphylococcus aureus . Lancet 1991; 337:488.
- 39. Oie S, Kamiya A. Survival of methicillin-resistant Staphylococcus aureus (MRSA) on naturally contaminated dry mops. J Hosp Infect 1996; 34:145–9.
- 40. Olson B, Weinstein RA, Nathan C, et al. Epidemiology of endemic Pseudomonas aeruginosa: why infection control efforts have failed. J Infect Dis 1984; 150:808–16.
- 41. Orsi GB, Mansi A, Tomao P, et al. Lack of association between clinical and environmental isolates of Pseudomonas aeruginosa in hospital wards. J Hosp Infect 1994; 27:49–60.
- 42. Porwancher R, Sheth A, Remphrey S, et al. Epidemiological study of hospital-acquired infection with vancomycin-resistant Enterococcus faecium: possible transmission by an electronic ear-probe thermometer. Infect Control Hosp Epidemiol 1997; 18:771–3.
- 43. Rampling A, Wiseman S, Davis L, et al. Evidence that hospital hygiene is important in the control of methicillin-resistant Staphylococcus aureus. J Hosp Infect 2001; 49:109–16.
- 44. Rogers M, Weinstock DM, Eagan J, et al. Rotavirus outbreak on a pediatric oncology floor: possible association with toys. Am J Infect Control 2000; 28:378–80.
- 45. Samore MH, Venkataraman L, DeGirolami PC, et al. Clinical and molecular epidemiology of sporadic and clustered cases of nosocomial Clostridium difficile diarrhea. Am J Med 1996; 100:32–40.
- 46. Sampathkumar P, Temesgen Z, Smith TF, et al. SARS: epidemiology, clinical presentation, management, and infection control measures. Mayo Clin Proc 2003; 78:882–90.
- 47. Sehulster L, Chinn RY, Centers for Disease Control and Prevention, HICPAC. Guidelines for environmental infection control in healthcare facilities: recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee (HICPAC). MMWR Recomm Rep 2003; 52(RR-10):1–42.
- 48. Shay DK, Maloney SA, Montecalvo M, et al. Epidemiology and mortality risk of vancomycin-resistant enterococcal bloodstream infections. J Infect Dis 1995; 172:993–1000.
- 49. Simor AE, Lee M, Vearncombe M, et al. An outbreak due to multiresistant A. baumannii in a burn unit: risk factors for acquisition and management. Infect Control Hosp Epidemiol 2002; 23:261–7.
- 50. Slaughter S, Hayden MK, Nathan C, et al. A comparison of the effect of universal use of gloves and gowns with that of glove use alone on acquisition of vancomycin-resistant enterococci in a medical intensive care unit. Ann Intern Med 1996; 125:448–56.
- 51. Sobel JD. Nosocomial Candida infections: an epidemiologic review. Clin Infect Dis 2006; 42 Suppl 4:S240–5. [Note: Added here if you want to include further references]
- 52. Traore O, Springthorpe VS, Sattar SA. A quantitative study of the survival of two species of Candida on porous and non-porous environmental surfaces and hands. J Appl Microbiol 2002; 92:549–55.
- 53. Vazquez JA, Dembry LM, Sanchez V, et al. Nosocomial Candida glabrata colonization: an epidemiologic study. J Clin Microbiol 1998; 36:421–6.
- 54. Vazquez JA, Sanchez V, Dmuchowski C, et al. Nosocomial acquisition of Candida albicans: an epidemiologic study. J Infect Dis 1993; 168:195–201.
- 55. Weber DJ, Rutala WA. Role of environmental contamination in the transmission of vancomycin-resistant enterococci. Infect Control Hosp Epidemiol 1997; 18:306–9.
- 56. Webster CA, Crowe M, Humphreys H, et al. Surveillance of an adult intensive care unit for long-term persistence of a multi-resistance strain of A. baumannii. Eur J Clin Microbiol Infect Dis 1998; 17:171–6.
- 57. World Health Organization. First data on stability and resistance of SARS coronavirus compiled by

- members of WHO laboratory network. Available at: http://www.who.int/csr/sars/survival\_2003\_05\_04/en/index.html. Accessed 29 February 2004.
- 58. Yu ITS, Li Y, Wong TW, et al. Evidence of airborne transmission of the severe acute respiratory syndrome virus. New Engl J Med 2004; 350:1731–9.
- 59. Zaidi M, Wenzel RP. Disinfection, sterilization, and control of hospital waste. In: Mandell GL, Bennett JE, Dolin R, eds. Principles and practice of infectious diseases. 5th ed. Philadelphia: Churchill Livingstone, 2000: 3000–2.