



REVIEW

The role of environmental cleaning in the control of hospital-acquired infection

S.J. Dancer*

Department of Microbiology, Hairmyres Hospital, East Kilbride, UK

Available online 1 September 2009

KEYWORDS

Acinetobacter;
Environmental
cleaning;
C. difficile;
Hospital-associated
infection;
Infection control;
MRSA;
Norovirus;
VRE

Summary Increasing numbers of hospital-acquired infections have generated much attention over the last decade. The public has linked the so-called 'superbugs' with their experience of dirty hospitals but the precise role of environmental cleaning in the control of these organisms remains unknown. Until cleaning becomes an evidence-based science, with established methods for assessment, the importance of a clean environment is likely to remain speculative. This review will examine the links between the hospital environment and various pathogens, including meticillin-resistant *Staphylococcus aureus*, vancomycin-resistant enterococci, norovirus, *Clostridium difficile* and acinetobacter. These organisms may be able to survive in healthcare environments but there is evidence to support their vulnerability to the cleaning process. Removal with, or without, disinfectants, appears to be associated with reduced infection rates for patients. Unfortunately, cleaning is often delivered as part of an overall infection control package in response to an outbreak and the importance of cleaning as a single intervention remains controversial. Recent work has shown that hand-touch sites are habitually contaminated by hospital pathogens, which are then delivered to patients on hands. It is possible that prioritising the cleaning of these sites might offer a useful adjunct to the current preoccupation with hand hygiene, since hand-touch sites comprise the less well-studied side of the hand-touch site equation. In addition, using proposed standards for hospital hygiene could provide further evidence that cleaning is a cost-effective intervention for controlling hospital-acquired infection.

© 2009 The Hospital Infection Society. Published by Elsevier Ltd. All rights reserved.

* Address: Department of Microbiology, Hairmyres Hospital, Eaglesham Road, East Kilbride G75 8RG, UK. Tel.: +44 1355 585000; fax: +44 1355 584350.

E-mail address: stephanie.dancer@lanarkshire.scot.nhs.uk

Introduction

There has been much debate over hospital cleanliness and increasing numbers of hospital-acquired

infections (HAIs). The public have linked their visual experience of dirty hospitals with the risk of HAI but there is little evidence to support this at present.¹ Cleaning has never been regarded as an evidence-based science and consequently receives little attention from the scientific community. Since there are no scientific standards to measure the effect of an individual cleaner, or assess environmental cleanliness, finding the evidence for benefit in the control of infection is further hampered.² There are always basic aesthetic considerations that cannot be disregarded; a perception of cleanliness, however defined, is expected for patients, their relatives and staff from healthcare environments.

Cleaning is routinely monitored by visual audit in the UK. Looking to see if a ward is clean may fulfil aesthetic obligations but it does not provide a reliable assessment of the infection risk for an individual patient on that ward.³ The organisms that cause infection are invisible to the naked eye and their existence is not necessarily associated with the presence of visual dirt. Furthermore, the impression of cleanliness is confounded by clutter, and fabric and maintenance deficits.⁴ Visual assessment will inevitably be subject to bias under these circumstances. It is more difficult to clean a crowded, cluttered environment, perhaps related to a cleaner's incentive, when confronted with peeling plaster, cracked tiles or worn floor coverings.⁴

Sites that are frequently touched by hands are thought to provide the greatest risk for patients, and those situated right beside patients provide the biggest risk of all.^{5–7} The responsibility for cleaning near-patient hand-touch sites does not always rest with the ward cleaners, however, since beds, drip stands, lockers and overbed tables are more usually cleaned by nurses.^{7,8} Nurses are also responsible for the decontamination of more delicate clinical equipment. This overlapping of cleaning responsibilities has created some confusion; it has also meant that cleaning opportunities of some items are missed or abandoned.^{9,10}

The microbial pathogens that cause HAI have two special properties: first, they are recognised as hospital pathogens; second, they have an innate ability to survive on surfaces in the hospital environment for long periods of time.¹ They include organisms such as meticillin-resistant *Staphylococcus aureus* (MRSA), *Clostridium difficile*, vancomycin-resistant enterococci (VRE), *Acinetobacter* spp. and norovirus.^{1,6} This mini-review will summarise the evidence for the presence and survival of these organisms in the clinical environment as well as support for cleaning as a valid

infection control intervention for patients. There will also be some discussion on the measurement of 'cleanliness' of the healthcare environment and why this is important for future work evaluating the role of hospital cleaning and HAI.

Meticillin-resistant *Staphylococcus aureus*

MRSA resists desiccation and can survive in hospital dust for up to a year.¹¹ It is found throughout the hospital environment, particularly around patients known to be colonised or infected with the bacterium. Molecular fingerprinting of these strains shows that MRSA-positive patients tend to shed their own strain of MRSA into the near-patient environment.¹² If staff enter a room containing an MRSA patient, two-thirds of them will acquire the patient's strain on gloved hands or apron.¹² Even if they do not touch the patient directly, four in ten will still exit the room carrying the patient's strain of MRSA on hands or apron.¹²

MRSA can be found on general surfaces such as floors and radiators, furniture such as beds and lockers, and clinical equipment.^{7–10,12–14} Some sites, e.g. linen, curtains, beds, lockers and overbed tables, tend to harbour MRSA more frequently than others.^{7,8,13} It is thought that contamination of near-patient hand-touch sites provides the biggest risk of MRSA acquisition for patients.^{2,6} In addition, there is a small but significant increase in the risk of acquiring MRSA if a patient is admitted into a room previously occupied by carrier patients.¹⁵

There is some evidence that cleaning removes MRSA from the ward environment with benefit for patients.^{6,16} An outbreak of MRSA lingered for several months on a urological ward, resisting all the usual infection control interventions such as promotion of hand hygiene and isolation of patients.¹⁶ The investigating team found the outbreak strain of MRSA scattered throughout the ward environment and doubled the number of domestic cleaning hours from 60 per week to 120. Following this, there was no further isolation of the outbreak strain from the environment and the number of patients affected decreased immediately. The cleaning intervention was thought to have played a significant role in the termination of the outbreak and was estimated to have saved at least £28,000.¹⁶

Another outbreak of glycopeptide-intermediate *S. aureus* (GISA) in an intensive therapy unit proved difficult to control until a wave of further control measures, including enhanced cleaning,

was introduced.¹⁷ The outbreak encompassed two patient clusters, although genotyping showed that all cases were caused by the same strain. The second cluster occurred despite the institution of maximum contact-isolation procedures. This directed attention towards the inanimate environment as the major source of contamination, since it was thought that re-emergence of the strain could be explained by its ability to survive on inert surfaces. The meticulous cleaning procedures finally implemented probably helped to stop the outbreak, although it was not possible to determine the relative roles of barrier precautions and environmental decontamination in eradicating the strain.¹⁷

Outwith the outbreak situation, another study examined the effects of enhanced cleaning on two matched surgical wards in a controlled cross-over trial for two six-month periods.¹⁸ There were nine ward-acquired MRSA infections during routine cleaning periods, but only four when the wards received extra cleaning, notably targeting hand-touch sites and clinical equipment. More MRSA patient-days during the enhanced cleaning periods predicted at least thirteen new cases instead of the four that actually occurred. The study concluded that targeted cleaning using detergent wipes and water could be a cost-effective mechanism of reducing MRSA infection on a surgical ward.¹⁸

Vancomycin-resistant enterococci

VRE are not credited with the same degree of pathogenicity as MRSA, but they may still cause infections in vulnerable patients, including outbreaks that are difficult to control. In addition, the *vanA* gene has been shown to transfer to MRSA, making the latter even more difficult to treat.¹⁹ Part of the problem of controlling VRE in hospitals is due to their extreme longevity in the hospital environment and their resistance to routine cleaning.¹ Even bleach-based cleaning sometimes fails to eradicate these hardy organisms.²⁰ Contact with contaminated surfaces in the rooms of colonised patients results in transfer of VRE to gloved hands, despite cleaning with disinfectants.^{5,21,22} In addition, patients admitted into rooms previously accommodating patients with VRE are themselves more likely to acquire the organism.¹⁵

It has already been suggested that environmental cleaning might be important in the control of VRE.²³ Additional work has highlighted its presence on hand-touch sites near the patient, just as for MRSA.^{10,22} A recent paper describes the impact of

improved environmental cleaning on the spread of VRE in a medical intensive care unit (ICU), with and without promotion of hand hygiene compliance.²⁴ In this study, there were four periods: the first served as a baseline period without any interventions; the second began after a 30 day period during which there was an education and intensified observation programme to improve cleaning; the third served as a 'washout' period without further interventions; and the fourth included an intensive hand hygiene campaign. The study found that enforcing cleaning measures along with encouraging hand hygiene was associated with less surface contamination with VRE, cleaner healthcare worker hands and a significant reduction in VRE cross-transmission among patients. The authors concluded that decreasing environmental contamination might help to control the spread of VRE in hospitals.²⁴

Clostridium difficile

C. difficile is a spore-forming anaerobe that has been recovered in abundance from the environment of symptomatic patients.^{1,25} The more patients there are with *C. difficile* on the ward, the more likely it is that other patients will also acquire the organism.²⁶ Environmental contamination with spores is now well accepted as a risk factor for the acquisition of *C. difficile*.²⁷ Furthermore, as the level of environmental contamination increases, so does the amount of *C. difficile* on the hands of healthcare workers, and once again near-patient hand-touch sites are regarded as a particular risk.^{25,28}

Having established that there is a dynamic transmission cycle for *C. difficile* similar to that for MRSA, there is additional evidence to support the value of cleaning in its control.^{1,29–31} Following a rise in *C. difficile* cases, one hospital introduced enhanced cleaning with hypochlorite into two ICUs.³¹ One of the ICUs applied the extra cleaning to all areas, including rooms used solely by staff, and sensitive clinical equipment was wiped over twice daily using hypochlorite-impregnated cloths. The other unit introduced the intensive hypochlorite clean into isolation rooms housing patients already infected with *C. difficile*. Rates of infection decreased in both units over several months and appeared to be maintained at a lower rate for at least two years after the cleaning intervention, despite some relaxation of the initial regimen.³¹

Infection control teams do not question the importance of thorough environmental cleaning

for controlling *C. difficile*, although whether cleaners should use disinfectants or detergents for routine cleaning continues to be hotly debated.^{25,29,30} Although bleach cleaning certainly appears to have an effect on both environmental contamination and patient acquisition of *C. difficile*, it could be the increased physical effort of enhanced cleaning that contributes towards the overall result rather than a direct effect of the bleach itself.³⁰ It is known that cleaning of toilets and other sites using appropriate disinfectants does not totally remove all traces of *C. difficile*.^{27,30,32,33} It is likely that agreement on the best way of removing *C. difficile* from the environment will elude us for some time to come.

Acinetobacter

Acinetobacter can also be recovered from the hospital environment with ease, including inanimate hand-touch sites near the patient.³⁴ Seeding clinical and environmental strains onto Formica surfaces demonstrated survival of between one and two weeks, although some strains are known to survive for much longer.³⁵ The importance of cleaning in controlling outbreaks of *Acinetobacter* spp. has been emphasised in previous studies.^{36,37} One of these describes an outbreak caused by multiply resistant strains of *A. baumannii* involving more than thirty patients in two ICUs.³⁷ Environmental contamination was recognised as an important reservoir of the epidemic strains and the outbreak ceased only after both ICUs were closed for terminal cleaning and disinfection.³⁷

Another study examined the levels of environmental contamination with acinetobacter in a neurosurgical ICU during a prolonged outbreak.³⁸ As with MRSA and *C. difficile*, there were many near-patient hand-touch sites that yielded the epidemic strain. This study also demonstrated a significant association between the amount of environmental contamination and patient colonisation. The conclusion was that high standards of cleaning play an integral role in controlling outbreaks of acinetobacter in the intensive care setting, although once again, little is known about the best way to clean in non-outbreak settings.³⁸

A further study describes what happened following the introduction of bedside computers in a paediatric burns ward.³⁹ There was a sudden increase in the number of patients acquiring acinetobacter and environmental screening demonstrated the organism on various surfaces in the patients' rooms, especially the plastic covers over the bedside computer keyboards. Targeted

infection control measures that included the use of gloves before using the computer and thorough disinfection of the plastic covers effectively terminated the outbreak. Up until the outbreak occurred, no one had thought to include the computer keyboards in a routine cleaning specification.³⁹ Similarly, during a period of high endemicity of acinetobacter in another burns unit, the endemic strain was identified from surfaces close to the patient, including bed linen, medication table and the display surface of an overbed monitor.⁴⁰

Norovirus

Norovirus can be found on a huge variety of surfaces both in hospitals and in the community.^{41–43} Several studies cite the association of norovirus with hand-touch sites such as toilet taps, door-handles, hospital equipment, elevator and microwave buttons, switches and telephones.^{41,42,44} When fingers come into contact with virus-contaminated material, norovirus is consistently transferred to typical hand-touch sites.⁴⁴

The importance of environmental cleaning in the control of outbreaks of norovirus is widely accepted.^{41,45} This includes clinical equipment, as well as floors, toilets and general surfaces. Curtains should be removed and sent to the laundry, and other soft furnishings either washed down or, preferably, steam-cleaned. All general cleaning, especially the toilets and bathroom areas, should be with a chlorine-containing disinfectant or bleach at a specified concentration. Cleaning policies should always include the use of these disinfectants since detergent-based cleaning often fails to eradicate the virus from the environment.⁴⁴ Without scrupulous attention to the environment, outbreaks not only continue, but will resume within a short space of time. This is because norovirus survives in the warm clinical environment with ease while retaining its infectivity, readily transferring to patients from hands that have just touched a contaminated site.

Outside hospitals, norovirus outbreaks can be devastating in closed or semi-closed communities.⁴⁶ These include sudden and extensive outbreaks in hotels or cruise-liners, but outbreaks can also occur in nursing and residential homes, prisons and schools. An outbreak reported recently in a primary school involved 79 pupils and 24 members of staff.⁴⁷ Subsequent investigation of the outbreak showed that person-to-person contact was a major factor in the transmission of the virus, but there was evidence that poorly cleaned computer

equipment was also implicated. A strain of norovirus, identical by RNA sequencing to two strains retrieved from patients, was isolated from one computer keyboard and mouse in one particular classroom. This occurred even after instituting a bleach clean the previous day. Public health officials recommended good hand-washing practices, exclusion of symptomatic persons and thorough environmental disinfection with a diluted (1:50 concentration) household bleach solution, to include sites that are shared but not commonly cleaned.⁴⁷

Discussion

There is plenty of evidence supporting the role of domestic cleaning in hospitals as an important intervention in the control of HAI. Unfortunately, it often constitutes part of an overall infection control package in response to an outbreak and its importance as a stand-alone activity remains controversial. This does not encourage on-going managerial support for cleaning services in the hospital, particularly if resources are limited. The situation is hampered by the lack of scientific standards for hospital cleaning, rather than the subjective visual assessment practised at present. The following three sections argue for a measured, targeted and methodological approach to domestic cleaning in hospitals.

What is 'clean'?

If we state that a hospital is clean, we assume that it looks clean and that it is safe for patients. Unfortunately, the microbes responsible for HAI are invisible to the naked eye. This means that visual assessment is insufficient for defining cleanliness, nor will it accurately predict the infection risk for patients.² Cleanliness should not actually be defined without indicating how it is assessed. A recent study compared visual assessment against both biochemical (ATP bioluminescence) and microbiological screening of the hospital environment.³ The results showed that whereas most surfaces looked clean, less than a quarter were free from organic soil (ATP) and less than half were microbiologically clean.³ Given the risk of acquiring hospital pathogens from a hospital ward, visual assessment is outdated, inadequate and scientifically obsolete. The only benefit from a visual inspection is to appease aesthetic obligations.

There has been suggestion that hospitals would benefit from cleaning standards emulating those implemented in the food industry.^{2,48} Food preparation surfaces are subjected to routine sampling

using a range of techniques in a coordinated and integrated approach.⁴⁸ Any isolation of pathogens, or pathogen indicators, causes concern and warrants immediate action. By contrast, environmental surface sampling in hospitals usually only takes place in response to an outbreak – and then only if the infection control team responsible has the motive, means and interest to initiate environmental screening.⁴⁸ It is time that high risk surfaces were subjected to routine screening in order to monitor overall levels of microbial dirt and the results used to generate increased or targeted cleaning before a hospital outbreak occurs. It is false economy to wait until an outbreak occurs before the clinical environment receives the attention it deserves.

There is no reason why cleaning should not become an evidence-based science, particularly when we know that lack of it is associated with human infection. Microbiological standards have been proposed, using long-established principles from the food industry as well as from standards governing media such as air and water.² Since the pathogens of interest are widely scattered in time and space, these standards make use of quantitative and qualitative microbial indicators rather than focus on trying to find a discrete pathogen. Practical applications for high risk surfaces in hospitals would not be difficult to institute although the finer details related to risk, and site, require further evaluation.^{2,49} Surfaces in outpatient corridors do not present a similar risk of infection as hand-touch sites might, situated beside a ventilated patient in an ICU. Attempts have already been made to compare benchmark visual, ATP bioluminescence and microbiological values against each other, and one study has attempted to evaluate microbiological standards against infection risk in an ICU.^{8,50}

Where to clean?

There is increasing evidence regarding the importance of hand-touch sites in the transmission of pathogens to healthy persons, as well as to patients.^{51,52} It is also becoming apparent that the sites closer to the patient are more likely to furnish an infection risk than those situated further away.^{7,8} The role of these near-patient hand-touch sites in MRSA transmission and, indeed, other hospital pathogens, has not been given the priority that it deserves. Ward cleaners work to a set specification that encompasses general surfaces and bathrooms, with emphasis on the cleaning of floors and toilets.⁵³ These are not necessarily near-patient hand-touch sites. Examples of the latter include bed rails, bedside lockers, infusion pumps, door handles and various switches,

including the nurse-call button, and they rarely feature in the domestic specification.^{2,7,8}

It is already known that traditional sites such as toilets, general surfaces and sinks tend to attract high rates of cleaning but that hand-touch sites, which are more likely to harbour and transmit microbial pathogens, are only poorly cleaned.^{10,54} The responsibility for cleaning many hand-touch sites usually rests with the ward nurses, who are often very busy and almost permanently understaffed in many hospitals. Two recent studies in ICUs have demonstrated an increased risk of infection following periods of inadequate nurse staffing, or conversely, excess workload.^{55,56} It may be that concentrating available cleaning resources on high-risk hand-touch sites would be the most cost-effective cleaning strategy at the present time.¹⁸ A recent study has shown that it is possible to improve environmental cleaning following an educational campaign and feedback on adequacy of discharge cleaning.¹⁰

How to clean?

Most of the studies describing the benefits from cleaning in this review used disinfectants to clean the hospital environment. Virtually all were reported as part of the response to an outbreak. Only a few UK-based studies used detergent and water, and even fewer reported cleaning benefits in the absence of an outbreak.^{18,30} It appears that when reviewing the evidence for the role of cleaning in the control of HAI, there are several issues which still require clarification. First, is there any difference between the quantity, quality and methods for routine cleaning compared with what is needed in the event of an outbreak; and second, is it sufficient to proclaim the benefits from cleaning with disinfectants without establishing what can be achieved using soap and water alone? These questions require an evidence-based approach before we can set the best specification for cleaning in our hospitals. In addition, no one has yet modelled different cleaning methods against the infection risk for patients, their degree of vulnerability and the clinical area in which they are exposed.

Most hospitals have their own domestic specifications for wards, operating theatres, outpatient and non-clinical areas. More countries are producing national standards for environmental cleaning. These set a necessary and valuable precedent but they are not based on sufficient scientific evidence to justify their contents. At the beginning of the twenty-first century, we simply do not know how to clean our hospitals in order to create the safest environment for patient care – hence

a veritable blunderbuss approach to cleaning in the event of an outbreak and managerial reluctance to protect, let alone prioritise, routine standards of cleaning outwith the outbreak situation. There is a lot of work still to do to establish cleaning as an evidence-based science, and to translate the evidence into cleaning practices in healthcare environments. Given the importance of controlling HAI, it makes sense to support current cleaning practises until such time as sufficient evidence is forthcoming.⁶

There is plenty of evidence to support basic cleaning in hospitals. More and better-directed cleaning reduces the risk of acquisition of a variety of hospital organisms. It might also affect the number of patients with these so-called 'superbugs' in the community, since hospital acquisition invariably leads to patients taking them home.⁵⁷ More interest in basic hygiene is warranted because microbes are becoming increasingly resistant due to the inappropriate, inadequate and uncontrolled use of antibiotics and there are fewer and fewer agents in the developmental pipeline.⁵⁸ A century ago, people died from trivial wounds because there were no antibiotics.

Simple hygiene could be our only defence when Darwinian evolution finally terminates the antibiotic era.⁵⁹ We need to raise the level of awareness regarding hygiene, and its importance, throughout society and it is hospitals that should lead the way. If they at least start by cleaning up the way they could, then this would at least offer some semblance of safety to patients.

Conflict of interest statement

The author has received research funding from UNISON for studies on hospital cleaning.

Funding sources

None.

References

1. Dancer SJ. Mopping up hospital infection. *J Hosp Infect* 1999;43:85–100.
2. Dancer SJ. How do we assess hospital cleaning? A proposal for microbiological standards for surface hygiene in hospitals. *J Hosp Infect* 2004;56:10–15.
3. Griffith CJ, Cooper RA, Gilmore J, Davies C, Lewis M. An evaluation of hospital cleaning regimes and standards. *J Hosp Infect* 2000;45:19–28.
4. Maurer IM. *Hospital hygiene*. 3rd edn. Bristol: Wright PSG; 1985.
5. Bhalla A, Pultz NJ, Gries DM, *et al*. Acquisition of nosocomial pathogens on hands after contact with environmental surfaces near hospitalised patients. *Infect Control Hosp Epidemiol* 2004;25:164–167.

6. Dancer SJ. Importance of the environment in methicillin-resistant *Staphylococcus aureus* acquisition: the case for hospital cleaning. *Lancet Infect Dis* 2008;**8**:101–113.
7. Dancer SJ, White L, Robertson C. Monitoring environmental cleanliness on two surgical wards. *Int J Environ Health Res* 2008;**18**:357–364.
8. White L, Dancer SJ, Robertson C, McDonald J. Are hygiene standards useful in assessing infection risk? *Am J Infect Control* 2008;**36**:381–384.
9. Blythe D, Keenlyside D, Dawson SJ, Galloway A. Environmental contamination due to methicillin-resistant *Staphylococcus aureus* (MRSA). *J Hosp Infect* 1998;**38**:67–70.
10. Goodman ER, Platt R, Bass R, Onderdonk AB, Yokoe DS, Huang SS. Impact of an environmental cleaning intervention on the presence of methicillin-resistant *Staphylococcus aureus* and vancomycin-resistant enterococci on surfaces in intensive care unit rooms. *Infect Control Hosp Epidemiol* 2008;**29**:593–599.
11. Wagenvoort JH, Sluijsmans W, Penders RJ. Better environmental survival of outbreak vs. sporadic MRSA isolates. *J Hosp Infect* 2000;**45**:231–234.
12. Boyce JM, Potter-Bynoe G, Chenevert C, King T. Environmental contamination due to methicillin-resistant *Staphylococcus aureus*: possible infection control implications. *Infect Control Hosp Epidemiol* 1997;**18**:622–627.
13. Lemmen SW, Hafner H, Zollman D, Stanzel S, Lutticken R. Distribution of multi-resistant Gram-negative versus Gram-positive bacteria in the hospital inanimate environment. *J Hosp Infect* 2004;**56**:191–197.
14. Hardy KJ, Oppenheim BA, Gossain S, Gao F, Hawkey PM. A study of the relationship between environmental contamination with methicillin-resistant *Staphylococcus aureus* (MRSA) and patients' acquisition of MRSA. *Infect Control Hosp Epidemiol* 2006;**27**:127–132.
15. Huang SS, Datta R, Platt R. Risk of acquiring antibiotic-resistant bacteria from prior room occupants. *Arch Intern Med* 2006;**166**:1945–1951.
16. 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**:109–116.
17. de Lassence A, Hidri N, Timsit JF, et al. Control and outcome of a large outbreak of colonization and infection with glycopeptide-intermediate *Staphylococcus aureus* in an intensive care unit. *Clin Infect Dis* 2006;**42**:170–178.
18. Dancer SJ, White LF, Lamb J, Girvan EK, Robertson C. Measuring the effect of enhanced cleaning in a UK hospital: a prospective cross-over study. *BMC Infect Med* 2009;**7**:28.
19. Sievert DM, Rudrik JT, Patel JB, McDonald LC, Wilkins MJ, Hageman JC. Vancomycin-resistant *Staphylococcus aureus* in the United States, 2002–2006. *Clin Infect Dis* 2008;**46**:668–674.
20. Noble MA, Isaac-Renton JL, Bryce EA, et al. The toilet as a transmission vector of vancomycin-resistant enterococci. *J Hosp Infect* 1998;**40**:237–241.
21. Ray AJ, Høyen CK, Das SM, Taub TF, Eckstein EC, Donskey CJ. Nosocomial transmission of vancomycin-resistant enterococci from surfaces. *J Am Med Assoc* 2002;**287**:1400–1401.
22. Hayden MK, Blom DW, Lyle EA, Moore CG, Weinstein RA. Risk of hand or glove contamination after contact with patients colonized with vancomycin-resistant enterococcus or the colonized patients' environment. *Infect Control Hosp Epidemiol* 2008;**29**:149–154.
23. Martinez JA, Ruthazer R, Hansjosten K, Barefoot L, Snyderman DR. 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**:1905–1912.
24. Hayden MK, Bonten MJM, Blom DW, Lyle EA, van de Vijver DAMC, Weinstein RA. Reduction in acquisition of vancomycin-resistant enterococcus after enforcement of routine environmental cleaning measures. *Clin Infect Dis* 2006;**42**:1552–1560.
25. Verity P, Wilcox MH, Fawley W, Parnell P. Prospective evaluation of environmental contamination by *Clostridium difficile* in isolation side rooms. *J Hosp Infect* 2001;**49**:204–209.
26. Dubberke ER, Reske KA, Olsen MA, et al. Evaluation of *Clostridium difficile*-associated disease pressure as a risk factor for *C. difficile*-associated disease. *Arch Intern Med* 2007;**167**:1092–1097.
27. Kaatz GW, Gitlin SD, Schaberg DR, et al. Acquisition of *Clostridium difficile* from the hospital environment. *Am J Epidemiol* 1998;**127**:1289–1294.
28. Samore MH, Venkatamaran L, DeGirolami PC, Arbeit RD, Karchmer AW. Clinical and molecular epidemiology of sporadic and clustered cases of nosocomial *Clostridium difficile* diarrhoea. *Am J Med* 1996;**100**:32–40.
29. Mayfield JL, Leet T, Miller J, Mundy LM. Environmental control to reduce transmission of *Clostridium difficile*. *Clin Infect Dis* 2000;**31**:995–1000.
30. Wilcox MH, Fawley WN, Wrighlesworth N, Parnell P, Verity P, Freeman J. Comparison of the effect of detergent versus hypochlorite cleaning on environmental contamination and incidence of *Clostridium difficile* infection. *J Hosp Infect* 2003;**54**:109–114.
31. McMullen KM, Zack J, Coopersmith CM, Kollef M, Dubberke E, Warren DK. Use of hypochlorite solution to decrease rates of *Clostridium difficile*-associated diarrhoea. *Infect Control Hosp Epidemiol* 2007;**28**:205–207.
32. Eckstein BC, Adams DA, Eckstein EC, et al. Reduction of *Clostridium difficile* and vancomycin-resistant Enterococcus contamination of environmental surfaces after an intervention to improve cleaning methods. *BMC Infect Dis* 2007;**7**:61.
33. Alfa MJ, Dueck C, Olson N, et al. UV-visible marker confirms that environmental persistence of *Clostridium difficile* spores in toilets of patients with *C. difficile*-associated diarrhea is associated with lack of compliance with cleaning protocol. *BMC Infect Dis* 2008;**8**:64–70.
34. Getchell-White SI, Donowitz LJ, Groschel DH. The inanimate environment of an intensive care unit as a potential source of nosocomial bacteria: evidence for long survival of *Acinetobacter calcoaceticus*. *Infect Control Hosp Epidemiol* 1989;**10**:402–407.
35. Wendt C, Dietze B, Dietz E, Ruden H. Survival of *Acinetobacter baumannii* on dry surfaces. *J Clin Microbiol* 1997;**35**:1394–1397.
36. Scerpella EG, Wanger AR, Armitige L, Anderlini P, Ericsson CD. Nosocomial outbreak caused by a multiresistant clone of *Acinetobacter baumannii*: results of the case-control and molecular epidemiologic investigations. *Infect Control Hosp Epidemiol* 1995;**16**:92–97.
37. Tankovic J, Legrand P, de Gatines G, Chemineau V, Brun-Buisson C, Duval J. Characterisation of a hospital outbreak of imipenem-resistant *Acinetobacter baumannii* by phenotypic and genotypic typing methods. *J Clin Microbiol* 1994;**32**:2677–2681.
38. Denton M, Wilcox MH, Parnell P, et al. Role of environmental cleaning in controlling an outbreak of *Acinetobacter baumannii* on a neurosurgical intensive care unit. *J Hosp Infect* 2004;**56**:106–110.
39. Neely A, Maley MP, Warden GD. Computer keyboards as reservoirs for *Acinetobacter baumannii* in a burn hospital. *Clin Infect Dis* 1999;**29**:1358–1359.
40. Seifert H, Boullion B, Schulze A, Pulverer G. Plasmid DNA profiles of *Acinetobacter baumannii*: clinical application

- in a complex endemic setting. *Infect Control Hosp Epidemiol* 1994;**15**:520–528.
41. Wu HM, Fornek M, Schwab KJ, *et al.* A norovirus outbreak at a long-term-care facility: the role of environmental surface contamination. *Infect Control Hosp Epidemiol* 2005;**26**: 802–810.
 42. Gallimore CI, Taylor C, Gennery AR, *et al.* Environmental monitoring for gastroenteric viruses in a pediatric primary immunodeficiency unit. *J Clin Microbiol* 2006;**44**:395–399.
 43. Evans MR, Meldrum R, Lane W, *et al.* An outbreak of viral gastroenteritis following environmental contamination at a concert hall. *Epidemiol Infect* 2002;**129**:355–360.
 44. Barker J, Vipond IB, Bloomfield SF. Effects of cleaning and disinfection in reducing the spread of norovirus contamination via environmental surfaces. *J Hosp Infect* 2004;**58**:42–49.
 45. Green J, Wright PA, Gallimore CI, Mitchell O, Morgan-Capner P, Brown DWG. 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.
 46. Love SS, Jiang X, Barrett E, Farkas T, Kelly S. A large hotel outbreak of Norwalk-like virus gastroenteritis among three large groups of guests and hotel employees in Virginia. *Epidemiol Infect* 2002;**129**:127–132.
 47. CDC. Norovirus outbreak in an elementary school – District of Columbia, February 2007. *Morb Mortal Wkly Rep* 2008;**56**:1340–1343.
 48. Griffith CJ. Hospital-acquired infection: are there lessons from the food industry? *Biomed Sci* 2006 August;**697**–699.
 49. Al-Hamad A, Maxwell S. How clean is clean? Proposed methods for hospital cleaning assessment. *J Hosp Infect* 2008;**70**:328–334.
 50. Lewis T, Griffith C, Gallo M, Weinbren M. A modified ATP benchmark for evaluating the cleaning of some hospital environmental surfaces. *J Hosp Infect* 2008;**69**:156–163.
 51. Oelberg DG, Joyner SE, Jiang X, Laborde D, Islam MP, Pickering LK. Detection of pathogen transmission in neonatal nurseries using DNA markers as surrogate indicators. *Pediatrics* 2000;**105**:311–315.
 52. Rheinbaben F, Schunemann S, Gross T, Wolff H. Transmission of viruses via contact in a household setting: experiments using bacteriophage straight phiX174 as a model virus. *J Hosp Infect* 2000;**46**:61–66.
 53. Anonymous. *New model cleaning contract*. London: NHS Estates; December 2004.
 54. Carling PC, Briggs JL, Perkins J, Highlander D. Improved cleaning of patient rooms using a new targeting method. *Clin Infect Dis* 2006;**42**:385–388.
 55. Dancer SJ, Coyne M, Speekenbrink A, Samavedam S, Kennedy J, Wallace PGM. Methicillin-resistant *Staphylococcus aureus* (MRSA) acquisition in an intensive care unit (ICU). *Am J Infect Control* 2006;**34**:10–17.
 56. Hugonnet S, Chevrolet J-C, Pittet D. The effect of workload on infection risk in critically ill patients. *Crit Care Med* 2007;**35**:76–81.
 57. Scanvic A, Denic L, Gaillon S, Giry P, Andremont A, Lucet JC. Duration of colonisation by methicillin-resistant *Staphylococcus aureus* after hospital discharge and risk factors for prolonged carriage. *Clin Infect Dis* 2001;**32**: 1393–1398.
 58. Budd R. *Penicillin: triumph and tragedy*. Oxford: Oxford University Press; 2007.
 59. Dancer SJ. Back to cleanliness. Rapid response. *Br Med J* 2008;**336**.