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## Cleaning and decontamination of the healthcare environment

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**Abstract:** Evidence is accumulating for the role of cleaning in controlling hospital infections. Hospital pathogens such as meticillin-resistant *Staphylococcus aureus* (MRSA), vancomycin-resistant enterococci (VRE), norovirus, multi-resistant Gram-negative bacilli and *Clostridium difficile* persist in the healthcare environment for considerable lengths of time. Cleaning with both detergent and disinfectant-based regimens help control these pathogens in both routine and outbreak situations. The most important transmission risk comes from organisms on frequently handled items because hand contact with a contaminated site could deliver a pathogen to a patient. Cleaning practices should be tailored to clinical risk, near-patient areas and hand-touch-sites and scientifically evaluated for all surfaces and equipment in today's hospitals.

**Key words:** cleaning, detergent, disinfectants, infection control, decontamination.

### 15.1 Introduction

There remains debate over clean hospitals when considering hospital-acquired infections (HAIs).<sup>1,2</sup> A visual experience of dirty hospitals is automatically linked with infection risk but this is difficult to prove for a number of reasons. Firstly, there are already several known risks for patients acquiring infection in hospital – antimicrobial consumption, insufficient isolation rooms and poor hand hygiene, for example. Secondly, since cleaning has never been investigated as a discrete scientific entity, it is impossible to determine just how important it might be towards overall infection control.<sup>1</sup> Finding the evidence to support cleaning as a significant intervention for preventing infection has been seriously disadvantaged because there are no accepted risk-based standards to verify whether a hospital is truly clean and safe.<sup>3</sup> Finally, there is confusion between nursing and domestic personnel over allocation of cleaning responsibilities. Even established cleaning regimens do not necessarily target high-risk reservoirs due to a lack of evidence and education.

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Visual inspection of the hospital environment does not provide a reliable qualitative nor quantitative assessment of the infection risk for patients.<sup>2,3</sup> Microbes are invisible and they are not necessarily associated with visual dirt. The impression of cleanliness is confounded by clutter, excess equipment; cramped wards and fabric deficits. Visual assessment will inevitably be subject to bias under these circumstances, as well as subject to an individual's perception.<sup>1</sup> Despite this, there is general consensus that environmental cleanliness is important for controlling infection. This is largely due to historical influences as well as the large number of outbreak reports, which nearly always mention cleaning as an integral part of the control package.<sup>4</sup>

There has been much recent interest in the role of the environment in promoting transmission of pathogens as well as articles examining basic principles underlying the importance of cleaning.<sup>5,6</sup> Novel biocides, antimicrobial coatings and new types of cleaning equipment are constantly appearing, although few have been modelled against patient outcome. There is an increasing need to prioritise surface level cleaning in the prevention and control of hospital infection, particularly as pathogens are becoming more resistant to antimicrobial agents. Hospital cleaning in the 21st century deserves further investigation for both routine and outbreak practices.

## 15.2 Pathogen survival time in the hospital environment

If a microbial pathogen can retain viability on surfaces outside the human body, there is a risk that it could be picked up by hands or air currents and transmitted to a patient.<sup>4</sup> The longer it survives, the more likely it will ultimately reach a patient at a vulnerable site and cause infection. Robust pathogens will persist in an appropriate environmental niche for days unless removed through some cleaning process.<sup>1,7</sup> Organisms that are particularly good at resisting drying or desiccation are more likely to be associated with epidemic spread.<sup>8-10</sup>

Whilst the ability of bacterial spores to withstand intemperate environments is well known, survival patterns of vegetative bacteria and viruses in healthcare institutions are less predictable. It has been assumed that Gram-negative bacilli are more vulnerable to exposure on surfaces, and therefore pose less of a risk to patients.<sup>1</sup> This assumption has been challenged by studies that detail prolonged survival periods for some Gram-negative species.<sup>7</sup> For example, *Escherichia coli* and *Klebsiella* spp. have been shown to survive more than a year under certain conditions, and *Serratia marcescens* up to 2 months.<sup>7</sup> In contrast, methicillin-resistant *Staphylococcus aureus* (MRSA) has been shown to survive for a year in

hospital dust, the spores of *Clostridium difficile* for five months and epidemic vancomycin-resistant enterococci (VRE) for up to four years.<sup>10-12</sup> *Acinetobacter* can survive in surface dust for at least a month, with some strains reportedly surviving for up to 3 years.<sup>8</sup> *Pseudomonas aeruginosa* usually only survives for a couple of days but will persist for five weeks on a dry floor.<sup>7,13</sup> Along with *Stenotrophomonas maltophilia*, *Pseudomonas* spp. demonstrate long-term persistence within biofilm adherent to hospital plumbing components and other water-exposed sites.<sup>14,15</sup> Most respiratory viruses such as coronavirus, rhinovirus and influenza can survive on dry surfaces for a few days, with gastrointestinal viruses retaining viability for a couple of months. Norovirus is found in the hospital environment for days after an outbreak, demonstrating survivability despite terminal cleaning with bleach-based products.<sup>16,17</sup> Fungi such as *Candida* spp. may persist in hospitals for up to four months, although there are very few reports detailing the risk of cross-infection from an environmental source.<sup>7,18,19</sup>

Given the proven ability of these microorganisms to survive on surfaces for relatively long periods of time, it is obvious that the healthcare environment facilitates cross-transmission and outbreaks of many hospital pathogens. The risks of cross-transmission are exaggerated by heavy workload, understaffing, high bed occupancy rates and rapid bed turnover. Furthermore, in an era of cost cutting, those with cleaning responsibilities cannot hope to decontaminate all high-risk surfaces as often as required when a hospital is full to capacity and patients with attendant microorganisms are transferred between wards day and night.<sup>4</sup>

### 15.3 Identifying the main reservoirs of microorganisms

Pathogens can be recovered from the environment using a variety of microbiological techniques. Most organisms can be found in the air and ultimately on the floors, but almost any surface can host a range of microbes for differing lengths of time. These include general surfaces such as shelves and ledges; curtains, linen and clothes; furniture and computers, telephones, patients' beds and items of clinical equipment.<sup>1,12,20-25</sup>

Gram-negative organisms such as *Pseudomonas* spp. and *Stenotrophomonas* are associated with damp places such as taps, sinks, showers and baths.<sup>1,13,15,26,27</sup> Coliforms such as *Klebsiella* and *Serratia* have been identified from buckets, bowls, mops and liquids.<sup>1,28</sup> Thus, traditional sites for Gram-negative microbes have been sites constantly or intermittently exposed to water, but this is not always the case. Dry sites, e.g. patient charts, can also host a range of Gram-negative organisms.<sup>23</sup>

About 5% of near-patient sites demonstrate presence of Gram-negative bacilli indistinguishable to those from the patient.<sup>22</sup> Organisms identified

included *E.coli*, *Enterobacter*, *Serratia* and *Klebsiella*, and these organisms were recovered from a range of sites including linen and nightwear; bedside table, bed rail and chair; door handle; infusion pump and respirator; and expected bathroom sites. The most prevalent site for the patients' own isolate was the perineal region of the patients themselves, thus demonstrating the major reservoir for Gram-negative bacteria.<sup>22</sup> The perineum has already been highlighted as an important source of environmental contamination for hands of both patients and staff.<sup>29</sup>

Pathogens normally resident in the gastrointestinal system, such as norovirus, *C. difficile* and VRE, are predominantly recovered from bathrooms, toilets or commodes, although the propensity for survival of these particular organisms means that they can be found from many other sites in the healthcare environment.<sup>12,17,30,31</sup> Indeed, spores of *C. difficile* persist on hands and under fingernails, and could be carried between wards on the soles of shoes.<sup>12,32,33</sup> Spores may disseminate through the air, confounding attempts at controlling infection and invalidating terminal cleaning protocols.<sup>34</sup>

Norovirus easily spreads in air and on surfaces throughout an entire ward, although this usually reflects the situation during seasonal outbreaks.<sup>17</sup> Dust-loving MRSA and *Acinetobacter* contaminate rarely cleaned and/or inaccessible surfaces, such as shelves, tops of monitors, patient notes and computer keyboards.<sup>25,35-38</sup> The most frequently contaminated sites for MRSA on an acute ward are top of the bedside locker; overbed table and bed frame.<sup>22,39</sup> Airborne spread of MRSA and *Acinetobacter* has also been documented but remains poorly investigated.<sup>40-43</sup> Even *Pseudomonas* has been recovered from air, and similarly implicated in spread between patients.<sup>13</sup> The more traditional airborne pathogens, ubiquitous *Aspergillus* and *Bacillus* spp., are dispersed through the hospital particularly during hot dry weather, and often associated with construction or renovation.<sup>1,44,45</sup>

Coliforms and *Pseudomonas* may frequent 'wet' sites such as sinks and baths, with differences between the recovery rate from sinks on separate wards within the same hospital.<sup>46</sup> Few coliforms persisted in intensive care unit (ICU) sinks, as opposed to sinks on medical wards, with *Pseudomonas*-type bacteria more frequently isolated from ICU sinks than those on the medical wards. This was attributed to more frequent dispensing of alcohol and chlorhexidine for the purposes of hand disinfection in ICU. All environmental bacteria recovered from ICU were significantly more resistant to antibiotics than those from the medical wards. Antibiotic consumption appears to influence the resistance profiles of organisms on floors and other surfaces within a defined local environment such as a hospital ward.<sup>46</sup>

Prior room occupancy has been shown to be a risk for acquisition of both Gram-negative and Gram-positive organisms.<sup>5,47-51</sup> This suggests that

terminal cleaning and/or disinfection regimens for isolation rooms containing patients colonised and/or infected with MRSA, VRE, *C. difficile*, *Acinetobacter* and *Pseudomonas* fail to remove all microbial contamination, thus exposing a new admission to the remnants of a persistent environmental reservoir.<sup>52</sup> Given that this risk has been verified by many different authors, it strengthens the role of the environment in HAI.<sup>53</sup> A patient admitted into a room previously occupied by an infected patient remains at risk of acquiring the same organism, regardless of hand hygiene compliance rates by attendant clinical staff.<sup>54</sup>

Once a hospital pathogen reaches an appropriate environmental niche, it will persist, unless removed through some cleaning process. It then has the potential to contaminate hands or be uplifted by air currents and deposited upon a patient or onto surfaces beside the patient.<sup>29,34,55,56</sup> The greatest risk for infection for most patients emanates from surfaces beside or on beds, e.g. linen, bed frames, lockers and overbed tables.<sup>3,24,39,57</sup> Contamination of near-patient bedside sites provides an opportunity for everyone's hands, including those of the patient, to acquire pathogens and/or transfer them elsewhere.<sup>29,58,59</sup>

## 15.4 Transmission of contaminants by hands during healthcare

Items or surfaces that are frequently touched provide the largest risk of contamination by pathogens spread on hands.<sup>3,25</sup> These sites then act as reservoirs for subsequent dispersal. Seeding pieces of cauliflower mosaic virus onto a ward telephone allowed researchers to track the movement and spread of the viral marker around the unit, from hand-touch site to hand-touch site over the course of hours and days.<sup>60</sup> A similar community-based study placed viral components onto a door handle in a students' flat and charted the movement of the markers from site to site via hands.<sup>61</sup> These studies confirm the role of hands in mobilising microbial markers between hand-touch reservoirs, and the sites most likely to host pathogens. In addition, the community study showed how direct hand-to-hand contact, as occurs during handshaking, was able to spread marker virus to a succession of people following initial contamination of a door handle.<sup>61</sup>

Many previous studies have demonstrated transient and persistent carriage of hospital organisms on the hands of healthcare workers.<sup>9,27,29,32,55,62–68</sup> About 40% nurses' hands yield coliforms without prior disinfection, although rates depend upon the type of unit in which sampling takes place.<sup>29,63</sup> Another study showed that 17% ICU staff carried *Klebsiella* on their hands, and that these strains were probably related to those colonising or infecting patients resident on the unit.<sup>55</sup> Of clinical staff caring for patients with *C. difficile*, 59% had positive cultures for *C. difficile*

from their hands.<sup>32</sup> About 17% (9–25%) of contacts between a healthcare worker and an MRSA-colonised patient result in transmission of MRSA from a patient to the gloves of a healthcare worker.<sup>66</sup>

Staff also acquire organisms directly from the hospital environment.<sup>29,55,65,69–72</sup> Indeed, they are just as likely to acquire pathogens after touching environmental sites as they are after caring for patients.<sup>65,70–72</sup> Coliforms can be recovered from the hands of nurses after touching patients' washing materials and clothing, as well as after bed making, sluice room activities, handling bedlinen and curtains, and even after a drug round.<sup>29</sup> Once acquired, hands may then be responsible for contaminating additional environmental sites.<sup>7,56,73</sup>

Contamination of hands or gloves with hospital organisms provides a highly plausible route of transmission between patients on a ward.<sup>7,55,63,64</sup> Hand hygiene is an easy practical method of interrupting such transmission but compliance rarely reaches the levels required to remove the risk of HAI for many different reasons.<sup>54,74</sup> Even when staff know that a patient is isolated because they are colonised or infected with a hospital pathogen, hand hygiene compliance is still only about 50%.<sup>74</sup> It is also possible that hand hygiene is insufficient to stop pathogen transmission.<sup>70,75</sup> Neither chlorhexidine, alcohol, nor soap and water necessarily remove contamination from hands, and some hand cleansing products are ineffective against specific pathogens.<sup>63,70,76–78</sup>

## 15.5 The role of cleaning in reducing the infection risk for patients

Whilst there remains little evidence for the benefits of general surface cleaning alone, cleaning is often mentioned as an integral part of a multifaceted response to an outbreak lacking an identified common source.<sup>79</sup> Numerous reports detail cleaning as a major control component for outbreaks of MRSA; VRE; *C. difficile*; norovirus; and drug-resistant *Acinetobacter*. These pathogens thrive in dust and dirt in the temperate hospital environment and contaminate numerous sites on surfaces and equipment, particularly during an outbreak.

### 15.5.1 Cleaning and MRSA

There is some evidence that cleaning removes MRSA from the ward environment with benefit for patients.<sup>35,59</sup> MRSA was isolated from 13 patients on the dermatology ward over a 14-month period.<sup>80</sup> Extensive environmental culturing revealed that a blood pressure cuff and the patients' communal shower were positive for MRSA, with pulsed field gel electrophoresis (PFGE) demonstrating identical DNA typing patterns from

the majority of patient isolates and both environmental sources. Control was achieved after changing of blood pressure cuffs between patients and more stringent cleaning of communal areas.<sup>80</sup> Another MRSA outbreak on a urological ward resisted all the usual infection control interventions such as hand hygiene promotion and isolation of patients.<sup>81</sup> After the outbreak strain was isolated from the ward environment, the number of domestic cleaning hours was doubled from 60 to 120 hours per week and the number of patients affected immediately decreased. The cleaning intervention was thought to have played a significant role in the termination of the outbreak and saved at least £28000.<sup>81</sup>

An outbreak of glycopeptide-intermediate *Staph. aureus* (GISA) in an intensive therapy unit (ITU) proved difficult to control until further control measures, including enhanced cleaning, were introduced.<sup>82</sup> Again, this probably helped to stop the outbreak, although it was not possible to determine the relative roles of barrier precautions and environmental decontamination. Outwith the outbreak situation, the effects of enhanced cleaning were monitored on two matched surgical wards in a prospective controlled cross-over trial for two 6-month periods.<sup>59</sup> 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 13 new cases instead of the 4 that actually occurred. The study concluded that targeted cleaning using detergent wipes and water could be a cost-effective mechanism of reducing MRSA infections.<sup>59</sup>

### 15.5.2 Cleaning and VRE

Environmental cleaning might also be important for controlling VRE.<sup>83,84</sup> One study describes the impact of improved cleaning on the spread of VRE in a medical ICU, with and without promotion of hand hygiene compliance.<sup>85</sup> Enforcing cleaning measures along with improved 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.<sup>85</sup> Introducing an educational programme, contact precautions and reinforcement of environmental cleaning was the response to escalating VRE cases in a Brazilian hospital.<sup>86</sup> Enhanced cleaning emphasised use of bleach for bathroom surfaces and 70% alcohol for furniture and patient equipment. The overall package helped prevent dissemination of VRE throughout the hospital, including intensive care, with a decrease in attack rate from 1.49 to 0.33 ( $p < 0.001$ ).<sup>86</sup> Bleach-based terminal cleaning was used for an earlier



study to control VRE in a haemato-oncology unit, again as part of an intervention package.<sup>84</sup>

Another package of interventions, including extensive cleaning of environmental surfaces and environmental cultures, was implemented in three ICUs by a team in South Korea.<sup>87</sup> During the outbreak, a total of 50 patients with VRE were identified by clinical and surveillance cultures, and 46 had vancomycin-resistant *Enterococcus faecium* (VREF). PFGE analysis of VREF isolates during the initial two months disclosed six types and clusters of two major types. Environmental surfaces were rigorously cleaned three times a day using 5% sodium hypochlorite. The outbreak was terminated 5 months after implementation of the interventions, with the weekly prevalence rate reduced from 9.1/100 to 0.6/100 patient-days, and rectal acquisition rate down from 6.9/100 to 0/100 patient-days.<sup>87</sup>

### 15.5.3 Cleaning and *C. difficile*

The benefits of cleaning for control of *C. difficile* are well established.<sup>88,89</sup> Following a rise in *C. difficile* cases, one hospital introduced enhanced cleaning with hypochlorite into two ICUs.<sup>90</sup> 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 2 years after the cleaning intervention, despite some relaxation of the initial regimen.<sup>90</sup>

Increased rates of *C. difficile* infection (CDI) in three American hospitals prompted terminal room cleaning of those affected with dilute bleach instead of the usual quaternary ammonium compound.<sup>91</sup> All surfaces, floor to ceiling, were wiped with dilute bleach applied with towels to thoroughly wet the surfaces. The prevalence density of *C. difficile* fell by 48%, with a sustained and significant reduction on the rate of nosocomial CDI. Another group implemented daily cleaning with 0.55% bleach wipes on two medical wards with a high incidence of *C. difficile*.<sup>92</sup> Pre-intervention, there was a total of 31 new cases of *C. difficile* on the wards. After the cleaning strategy, there were 4 cases on these wards over the following year, representing a 7-fold decrease in cases of *C. difficile*. There were no other interventions introduced other than targeted cleaning with bleach wipes.<sup>92</sup>

Use of chlorine-releasing disinfectants in rooms contaminated with *C. difficile* spores reduces the number of spores in the environment, with some evidence to suggest that this reduces the risk of recurrence and spread *C. difficile*-associated disease of (CDAD).<sup>93</sup> Evidence is strongest for products with higher concentrations of disinfecting agents (e.g. 5000mg/L free

chlorine). The benefits of chlorine use might be greater in units where rates of CDAD are high (e.g. geriatric rehabilitation or assessment units) or in response to outbreaks. Additionally, effectiveness of cleaning agents used in the hospital environment on levels of spores and, more important, rates of CDAD, might be related to training and time-constraints of cleaning staff.<sup>93,94</sup>

#### 15.5.4 Cleaning and *Acinetobacter*

The importance of cleaning in controlling outbreaks of *Acinetobacter* spp. has been emphasised in previous studies.<sup>95-97</sup> One of these describes an outbreak caused by multiply resistant strains of *A. baumannii* involving more than 30 patients in two ICUs.<sup>96</sup> 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.<sup>96</sup> Another study examined the levels of environmental contamination with *Acinetobacter* in a neurosurgical ICU during a prolonged outbreak.<sup>98</sup> Near-patient hand-touch sites frequently yielded the epidemic strain, and there was 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 little is known about the best way to clean in non-outbreak settings.<sup>98</sup>

A further study describes what happened following the introduction of bedside computers in a paediatric burns ward.<sup>37</sup> 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 on the 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. Before the outbreak occurred, no one had thought to include the computers in a routine cleaning specification.<sup>37</sup>

A 3-year prospective study was conducted in intensive care and coronary care units to evaluate interventions including contact isolation precautions, hand hygiene, active surveillance, cohorting patients colonised or infected with pandrug-resistant *A. baumannii* and environmental cleaning with 1:100 sodium hypochlorite.<sup>99</sup> The rate of *A. baumannii* colonisation and/or infection was 3.6 cases per 1000 patient-days before the intervention. One year after the intervention, the rate of *A. baumannii* colonization and/or infection decreased by 66% to 1.2 cases per 1000 patient-days ( $p < .001$ ) and two years later by 76% to 0.85 cases per 1000 patient-days ( $p < .001$ ).<sup>99</sup>

### 15.5.5 Cleaning and multi-drug-resistant coliforms

The importance of cleaning in controlling outbreaks of Gram-negative microorganisms other than *Acinetobacter* is difficult to ascertain, given that enhanced cleaning usually comes as part of an overall package in response to cross-infection or outbreak. Despite this, there is general consensus that environmental cleanliness is important for controlling infection. Certainly, the literature is littered with reports of outbreaks of coliforms traced to discrete pieces of equipment, specific environmental site or particular product or practice.<sup>1</sup> This is probably because terminating an outbreak caused by single source contamination is a lot easier than implementing a routine cleaning regimen that prevents infection from a multitude of general surfaces. Identification of a single reservoir and eradicating it usually curtails the outbreak.<sup>28,100–102</sup>

Persistent reservoirs of resistant *Klebsiella pneumoniae* were traced to multiple contaminated sink components in an Aberdeen teaching hospital.<sup>100</sup> More recently, four patients in a neurosurgical ITU became infected or colonised with extended-spectrum  $\beta$ -lactamase-(ESBC)-producing *K. pneumoniae* over a period over 7 months.<sup>102</sup> An investigation revealed that the source of this outbreak was also a contaminated sink. By replacing the sink and its plumbing and improving routines regarding sink usage and cleaning, the outbreak was terminated.<sup>102</sup> Biofilm-forming *K. pneumoniae* strains, such as those demonstrating prolonged survival in plumbing components, are more likely to produce ESBLs.<sup>103</sup>

Another outbreak of resistant *K. pneumoniae* among neonates highlights the risks of reusing disposable equipment.<sup>101</sup> The investigation showed that most of the babies were infected a few days after birth or just after hospitalisation. The only common feature was that those presenting with respiratory distress received mucus aspiration a few days before becoming symptomatic. Although a new aspiration tube was used for each case, the tubes had been rinsed in tap water between each aspiration for the same baby. This tap water was not changed between babies and the bowl used was not properly cleaned either. The tap water was found to be contaminated with the same type of *K. pneumoniae*.<sup>101</sup>

The lack of evidence for benefits from general surface cleaning alone is well recognised, even as a response to an outbreak.<sup>104</sup> There is one recent report emphasising additional cleaning following the identification of a carbapenemase-producing *K. pneumoniae* in a ward in a UK hospital.<sup>105</sup> Chlorine-based cleaning was implemented throughout the ward, including patient-related items. Enhanced cleaning was only part of the overall infection control package, however, along with a urinary catheter care bundle; patient note tagging; hand hygiene emphasis; and contact precautions

for patient cases.<sup>105</sup> Another report describes an educational intervention to improve hand hygiene and environmental cleaning in an 11-bedded ICU.<sup>106</sup> The number of patients colonised with ESBL Enterobacteriaceae during a three month pre-intervention period decreased from 70% to 40% during a post-intervention period, although it is possible that the initial high proportion of colonised patients represented an underlying outbreak.<sup>106</sup>

### 15.5.6 Cleaning and *Pseudomonas/Stenotrophomonas*

*Pseudomonas* and *Stenotrophomonas* originating from water outlets have the potential to colonise and infect patients despite a lack of evidence for specific transmission pathways.<sup>107</sup> Only one previous report details transmission of *P. aeruginosa* from sinks to hands during handwashing.<sup>27</sup> Whilst survival on dry surfaces may be only transient, persistent reservoirs of these organisms can be traced to biofilm within sink components, water lines and hospital drains.<sup>13,108</sup> This complex living deposit on internal plumbing surfaces hosts and protects a multitude of water-loving organisms, some of which pose a threat to debilitated patients, particularly those in ITU. Bacteria within biofilm are more likely to be able to withstand chlorine and other types of disinfectants along with greater capacity for antimicrobial resistance.<sup>109</sup>

Various outbreak investigations have shown that recovery of pathogens from water sources, surrounding surfaces, and patient isolates demonstrate indistinguishable strains of *Pseudomonas* and *Stenotrophomonas*.<sup>26,98,102,110,111</sup> An outbreak of *Burkholderia cepacia* on a paediatric unit was attributed to sinks, possibly linked with the presence of aerator filters fitted to the taps.<sup>112</sup> Tap aerators have also been shown to be a source of patient colonisation with *Stenotrophomonas maltophilia*.<sup>26</sup> For this reason, aerators should be replaced with flow straighteners in healthcare premises. Disinfection using chlorinated products, without disruption of biofilm, only offers limited control; a comprehensive cleaning initiative is required to physically remove the biofilm lining the surfaces of affected plumbing components.<sup>111,113</sup> These are often difficult to access and require close collaboration between Estates and domestic staff. Complete eradication is almost impossible but regular cleaning and disinfection with chlorine products will hinder further cases if it is part of a long-term maintenance programme.<sup>4,107</sup>

### 15.5.7 Cleaning and norovirus

The importance of environmental cleaning in the control of outbreaks of norovirus is widely accepted.<sup>114,115</sup> All general cleaning, especially toilet and bathroom areas, should use a chlorine-containing disinfectant or bleach

at a specified concentration. Detergent-based cleaning often fails to eradicate the virus from the environment.<sup>56</sup> One study recently reported indistinguishable genotypes of norovirus from both patient and environmental sources, including detection of viable virus in the environment following terminal cleaning.<sup>17</sup> The authors found expected reservoirs near toilets in bathrooms but also on numerous types of clinical equipment, e.g. pulse and blood pressure machine; alcohol gel containers, and near-patient sites. Persistence of viral reservoirs means that new admissions will be exposed to norovirus, and with current pressures to reduce the length of stay, there is a higher throughput of increasing numbers of patients vulnerable to norovirus.<sup>17</sup> Without scrupulous cleaning attention, outbreaks will quickly resume.

Outside hospitals, norovirus outbreaks can be devastating in closed or semi-closed communities.<sup>116</sup> These include sudden and extensive outbreaks in hotels or prisons, but outbreaks can also occur in nursing and residential homes, cruise-ships and schools.<sup>117,118</sup> An outbreak reported recently in a primary school involved 79 pupils and 24 members of staff.<sup>118</sup> 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 the environment was also implicated. A strain of norovirus, indistinguishable from patient strains, was isolated from a computer keyboard and mouse in one particular classroom despite cleaning with bleach the previous day. Public health officials recommended hand hygiene, exclusion of symptomatic persons and thorough environmental disinfection with a diluted (1:50 concentration) bleach solution, to include sites that were not commonly cleaned.<sup>118</sup>

## 15.6 Contaminated cleaning equipment and fluids

There is not much point in implementing comprehensive cleaning schedules if cleaning agents or equipment themselves are contaminated. Poor choice of cleaning methods or agents, or inadequate maintenance of equipment will result in environmental contamination of the very surfaces that need attention. There are numerous examples of different types of cleaning cloths, including microfibre products, that spread microbes across surfaces rather than removing them.<sup>56,73,119–121</sup> Cleaning equipment may also become contaminated with hospital pathogens and disperse these into the hospital environment.<sup>1,28,122,123</sup> Disinfectants are more effective at killing pathogens than in-use detergents but some hospital pathogens can resist bactericidal effect of a particular agent.<sup>124</sup> Both multi-drug resistant *S. marcescens* and extremely-drug-resistant strains of *K. pneumoniae* have demonstrated increasing tolerance to chlorhexidine.<sup>125,126</sup> Other cleaning fluids can become contaminated with Gram-negative bacilli during use; indeed, some

formulations may encourage acquisition of resistance elements by Gram-negative organisms.<sup>127,128</sup>

Microorganisms use an inadequately cleaned niche to exchange genetic material coding for antimicrobial resistance and other survival mechanisms, including resistance or tolerance to disinfectants.<sup>129,130</sup> Once established, these hardy strains will ultimately infect debilitated patients.<sup>130</sup> Hospital waste water has been shown to harbour KPC-2-producing *K. pneumoniae*, suggesting widespread contamination throughout the healthcare environment.<sup>131</sup>

## 15.7 Assessment of environmental cleanliness

Various scientific methods to measure environmental soil have been devised, since visual inspection cannot ascertain the infection risk for patients.<sup>2</sup> A validated and risk assessed technique is needed to determine cleanliness, rather than the subjective assessment currently provided by visual inspection and clipboards. Chemical (ATP bioluminescence) and microbiological methods have been utilised by the food industry for years, and have been tested in hospitals.<sup>2,3</sup> Measurements from these systems have provided a range of values to model against patient risk; from these, it might be possible to choose an appropriate benchmark for routine monitoring. Hospital staff need to know exactly which levels are acceptable for patient safety purposes.<sup>2,3,4,132</sup>

Currently, aerobic colony counts of <2.5–5 colony forming units (cfu) per cm<sup>2</sup> on hand-touch sites have been tested as microbiological benchmarks.<sup>2,3,39,59,132–134</sup> These levels have not been standardised for use in hospitals, but similar counts have been established in the food industry. Retail and food manufacturers, plus additional agencies, also use microbiological standards incorporating the presence of indicator organisms, identification of which depends upon risk to human health from the medium monitored.<sup>135,136</sup> Since coagulase-positive staphylococci provide a reliable indicator of environmental hygiene, studies examining the utility of microbiological standards in hospitals have chosen both *Staph. aureus* and MRSA to help monitor cleanliness.<sup>3,39,132–134,137</sup>

ATP systems have varying benchmarks depending upon the type of luminometer used. These range from 25 to 500 relative light units (RLUs) for 10–100 cm<sup>2</sup> on hospital surfaces,<sup>133,134,138</sup> but there is concern that some systems are not sufficiently sensitive to detect very low microbial counts (<10 cfu/cm<sup>2</sup>).<sup>139,140</sup> One study found that benchmark categories of 100 RLUs and microbial growth <2.5 cfu/cm<sup>2</sup> were loosely associated, since there was approximately 60% agreement between them on whether a surface should pass or fail.<sup>134</sup> Clearly, more work needs to be done on finding the most appropriate method for detecting microbial soil.<sup>141</sup> ATP

measurements can be confounded by food and drink residues; disinfectants; microfibre products; and manufactured plastics found in the cleaning and laundering industries.<sup>2,141,142</sup> Chosen benchmarks should reflect patient risk; surfaces in outpatient clinics are not necessarily as critical for infection risk as sites beside a ventilated patient receiving intensive care. Once these benchmarks are established, routine monitoring should be able to indicate trends in hospital cleanliness and workload, and, most importantly, when enhanced cleaning activity is required before patients are exposed to serious risk of infection or even an outbreak.<sup>132,133</sup>

There are alternative methods of environmental assessment, notably cleaning inspections; education; monitoring; and feedback; all of which encourage enhanced performance by housekeepers. Placing invisible fluorescent markers at key sites for later inspection and feedback for domestic staff also improves overall cleaning compliance, along with reduction of key hospital pathogens.<sup>143</sup> Use of ATP monitoring demonstrates pronounced effect on cleaners when they received concomitant educational guidance.<sup>138</sup> Domestic staff react quickly to an environmental monitoring programme because they are concerned that their jobs may be at risk.<sup>4,85</sup> Further studies have demonstrated differing effects between direct observation, supervision and education of staff as they clean, again showing reduction of important hospital pathogens.<sup>144–146</sup> There is a concern that these interventions might lose impact over time, since cleaning is physically demanding, poorly paid and subject to inadequate staffing.<sup>147</sup> Hence training and continual evidence-based reassessment are required as part of staff development.

## 15.8 Current and future trends

New methods for environmental decontamination are constantly appearing on the market. Disinfectants tend to be expensive and environmentally unfriendly.<sup>148</sup> Some formulations persist in the water courses underlying towns and cities and exert long-term effects on other biological systems. This has encouraged 'greener' alternatives, particularly those that ultimately degrade into harmless components. Examples include ultra-heated steam; electrolysed water; ozone and hydrogen peroxide, amongst others.<sup>149–152</sup> Electrolysed water products have already shown potential for decontamination of the healthcare environment.<sup>153,154</sup>

In addition to these, are novel cleaning materials and equipment such as microfibre; scrubbing machines; microbicidal gases, vapours and anti-fogging or mist systems; ultraviolet (UV) light-emitting devices; air ionisers; and a range of high-pressure steam cleaners.<sup>152,155–162</sup> A recent paper describes the effect of a newly developed portable pulsed ultraviolet (UV) radiation device on bactericidal activity and its impact on the labour burden when



implemented in a hospital ward.<sup>161</sup> The use of pulsed UV in daily disinfection of housekeeping surfaces reduced the working hours by half in comparison with manual disinfection using ethanol wipes.<sup>161</sup>

There are new types of antimicrobial coatings available for linen, equipment, furniture and general surfaces such as floors, walls and doors.<sup>163,164</sup> Practically anything that can be impregnated with chemicals, or coated with microbicidal paint, could potentially be marketed as 'antibacterial' for healthcare environments. Bioactive surfaces or coatings can contain heavy metals (or their derivatives) such as copper, zinc, silver or titanium, or antiseptics and biocides.<sup>165-170</sup> There is evidence that coating near-patient hand-touch sites with copper reduces organisms such as MRSA and consequently, the risk of HAI.<sup>169,171</sup>

There are also electrostatic and inhibitory surfaces that repel microbial adhesion and even coatings marketed as 'self-cleaning'.<sup>163,172</sup> Different variations on a theme appear at frequent intervals, using ever more innovative technology. One recent example is a coating of nano-silver particles combined with titanium dioxide to form highly reactive TiO<sub>2</sub> Ag particles.<sup>173</sup> This invisible protective nanocoating can be applied onto a range of surfaces under low temperatures, which means that virtually all environmental surfaces in a hospital could theoretically be treated.<sup>173</sup>

Numerous guidelines emphasise the importance of cleaning but offer little practical advice on how to achieve this, or how often sites should receive cleaning attention. Microbial reservoirs are likely to fluctuate at different sites throughout the day. However delivered, viable organisms will persist according to their capacity for survival, unless removed by an effective cleaning regimen. Even if cleaning occurs on a regular basis, pathogens may recontaminate surfaces or equipment immediately after the cleaning process.<sup>174-176</sup> Using agents or surfaces with residual microbicidal activity to repel contamination would mean that hand hygiene might not be quite so critical, although staff should obviously not abandon their hand washing.<sup>54,177,178</sup> Provided such products could be shown to function as postulated, without significant toxicity, surface cleaning might also become less of an issue, although as with clinical staff and hand hygiene, routine cleaning practices should still be retained. Alternatively, a high-frequency cleaning regimen would theoretically discourage the accumulation of potential pathogens continually deposited on surfaces.<sup>179</sup>

Despite future promise from these products, traditional cleaning methods should not be relaxed or abandoned even if the whole hospital is treated to novel cleaning methods or coated with bioactive veneer.<sup>30</sup> No one single process will remove all relevant microbial soil from the hospital. There have already been problems reported with some of the methods mentioned, such as microfibre, steam cleaning, ozone, hydrogen peroxide and high-intensity light irradiation.<sup>121,151,155,156,160, 174,180-184</sup> Furthermore, concern remains over



activity of disinfectants in the field, since laboratory testing does not necessarily predict what happens on hospital surfaces.<sup>185</sup> There are always toxicity and cost issues to consider, and potential cross-resistance between disinfectants, antiseptics and antimicrobial agents.<sup>148,186</sup>

All novel cleaning methods require a comprehensive assessment in association with patient outcome before widespread adoption in healthcare systems. Cash-strapped hospitals should not invest in potentially toxic and/or expensive cleaning methods or agents without good reason. It goes without saying that traditional detergent-based cleaning should also receive a full and thorough appraisal.<sup>79</sup> It is possible that simply increasing the cleaning frequency of established high-risk sites with detergent could be a crucial factor in reducing infection risk, rather than an expensive and potentially toxic biocide.<sup>79,179</sup>

## 15.9 Conclusion

Microbiological screening has demonstrated numerous types of pathogen surviving throughout our hospitals. There is evidence to show that these resilient organisms pose a real and substantive infection risk for patients. The cleaning process could potentially have a huge impact on this risk, if it is aimed at the most frequently contaminated sites, physically removes or kills viable organisms; and takes place a sufficient number of times to inhibit the accumulation of pathogens delivered by air or hands. Whilst there are a flourishing number of novel disinfectants, antimicrobial surfaces and equipment now on the market, few have been properly assessed against patient infection risk and potential toxicity represents a long-term environmental threat. Establishing risk-based standards for all surfaces throughout the hospital would allow monitoring, measurement and scientific evaluation for the benefit of all patients and staff.<sup>187</sup> Frequent application of detergent-based cleaning requires urgent appraisal in order to be able to adequately and comprehensively compare future alternatives for cleaning our hospitals.

## 15.10 References

1. Dancer SJ. Mopping up hospital infection. *J Hosp Infect* 1999; **43**: 85–100.
2. Malik RE, Cooper RA and Griffith CJ. Use of audit tools to evaluate the efficacy of cleaning systems in hospitals. *Am J Infect Control* 2003; **31**: 181–7.
3. 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.
4. Dancer SJ. Hospital cleaning in the 21st century. *Eur J Clin Microbiol Infect Dis* 2011; **30**: 1473–81.

5. Carling PC and Bartley JM. Evaluating hygienic cleaning in healthcare settings: What you do not know can harm your patients. *Am J Infect Control* 2010; **38**: S41–50.
6. Weber DJ, Rutala WA, Miller MB, Huslage K and Sickbert-Bennett E. Role of hospital surfaces in the transmission of emerging health care-associated pathogens: norovirus, *Clostridium difficile*, and *Acinetobacter*. *Am J Infect Control* 2010; **38**: S25–33.
7. Kramer A, Schwebke I and Kampf G. How long do nosocomial pathogens persist on inanimate surfaces? A systematic review. *BMC Infect Dis* 2006; **6**: 130.
8. Jawad A, Seifert H, Snelling AM, Heritage J and Hawkey PM. Survival of *Acinetobacter baumannii* on dry surfaces: comparison of outbreak and sporadic isolates. *J Clin Microbiol* 1998; **36**: 1938–41.
9. Casewell MW and Desai N. Survival of multiply-resistant *Klebsiella aerogenes* and other Gram-negative bacilli on finger-tips. *J Hosp Infect* 1983; **4**: 350–60.
10. Wagenvoort J, MRSA Wagenvoort JH, Sluijsmans W and Penders RJ. Better environmental survival of outbreak vs. sporadic MRSA isolates. *J Hosp Infect* 2000; **45**: 231–4.
11. Wagenvoort JHT, De Brauwier EIGB, Penders RJR, Willems RJ, Top J and Bonten MJ. Environmental survival of *Enterococcus faecium*. *J Hosp Infect* 2011; **77**: 282–3.
12. Fekety R, Kim K, Brown D, Batts DH, Cudmore M and Silva J, Jr. Epidemiology of antibiotic-associated colitis: isolation of *Clostridium difficile* from the hospital environment. *Am J Med* 1981; **70**: 906–8.
13. Panagea S, Winstanley C, Walshaw MJ, Ledson MJ and Hart CA. Environmental contamination with an epidemic strain of *Pseudomonas aeruginosa* in a Liverpool cystic fibrosis centre, and study of its survival on dry surfaces. *J Hosp Infect* 2005; **59**: 102–7.
14. Donlan RM and Costerton JW. Biofilms: survival mechanisms of clinically relevant microorganisms. *Clin Microbiol Rev* 2002; **15**: 167–93.
15. Denton M, Todd NJ, Kerr KG, Hawkey PM and Littlewood JM. Molecular epidemiology of *Stenotrophomonas maltophilia* isolated from clinical specimens from patients with cystic fibrosis and associated environmental samples. *J Clin Microbiol* 1998; **36**: 1953–8.
16. d'Souza DH, Williams K, Jean J, Sair A and Jaykus L. *Persistence of Norwalk virus on environmental surfaces and its transfer to food*: Washington, D.C. American Society for Microbiology; 2003.
17. Morter S, Bennet G, Fish J, *et al.* Norovirus in the hospital setting: virus introduction and spread within the hospital environment. *J Hosp Infect* 2011; **77**: 106–12.
18. Phelps M, Ayliffe GAJ and Babb JR. An outbreak of candidiasis in a special care baby unit: the use of a resistogram typing method. *J Hosp Infect* 1986; **7**: 13–20.
19. Clark TA, Slavinski SA, Morgan J, *et al.* Epidemiologic and molecular characterization of an outbreak of *Candida parapsilosis* bloodstream infections in a community hospital. *J Clin Microbiol* 2004; **42**: 4468–72.
20. Sanderson PJ and Rawal P. Contamination of the environment of spinal cord injured patients by organisms causing urinary tract infection. *J Hosp Infect* 1987; **10**: 173–8.

21. Sanderson PJ and Ashalfi KM. Environmental contamination by organisms causing urinary tract infection. *J Hosp Infect* 1995; **29**: 301–3.
22. Lemmen SW, Hafner H, Zolldan D, Stanzel S and Luttkicken R. Distribution of multi-resistant Gram-negative versus Gram-positive bacteria in the hospital inanimate environment. *J Hosp Infect* 2004; **56**: 191–7.
23. Getchell-White SI, Donowitz LJ and Groeschel 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**: 402–7.
24. Malnick S, Bardenstein R, Huszar M, Gabbay J and Borkow G. Pyjamas and sheets as a potential source of nosocomial pathogens. *J Hosp Infect* 2008; **70**: 89–92.
25. Bures S, Fishbain JT, Uyehara CF, Parker JM and Berg BW. Computer keyboards and faucet handles as reservoirs of nosocomial pathogens in the intensive care unit. *Am J Infect Control* 2000; **28**: 465–71.
26. Weber DJ, Rutala WA, Blanchet CN, Jordan M and Gergen MF. Faucet aerators: a source of patient colonisation with *Stenotrophomonas maltophilia*. *Am J Infect Control* 1999; **27**: 59–63.
27. Döring G, Jansen S, Noll H, *et al.* Distribution and transmission of *Pseudomonas aeruginosa* and *Burkholderia cepacia* in a hospital ward. *Pediatr Pulmonol* 1996; **21**: 90–100.
28. Joynson DHM. Bowls and bacteria. *J Hyg Cambr* 1978; **80**: 423–5.
29. Sanderson PJ and Weissler S. Recovery of coliforms from the hands of nurses and patients: activities leading to contamination. *J Hosp Infect* 1992; **21**: 85–93.
30. 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 diarrhoea is associated with lack of compliance with cleaning protocol. *BMC Infect Dis* 2008; **8**: 64–70.
31. Noble MA, Isaac-Renton JL, *et al.* The toilet as a transmission vector of vancomycin-resistant enterococci. *J Hosp Infect* 1998; **40**: 237–41.
32. McFarland LV, Mulligan ME, Kwok RY and Stamm WE. Nosocomial acquisition of *Clostridium difficile* infection. *N Engl J Med* 1989; **320**: 204–10.
33. Nath SK, Thornley JH, Kelly M, *et al.* A sustained outbreak of *Clostridium difficile* in a general hospital: persistence of a toxigenic clone in four units. *Infect Control Hosp Epidemiol* 1994; **15**: 382–9.
34. Roberts K, Smith CF, Snelling AM, *et al.* Aerial dissemination of *Clostridium difficile* spores. *BMC Infect Dis* 2008; **8**: 7.
35. Dancer SJ. Importance of the environment in meticillin-resistant *Staphylococcus aureus* acquisition: the case for hospital cleaning. *Lancet Infect Dis* 2008; **8**: 101–13.
36. Thom KA, Johnson JK, Lee MS and Harris AD. Environmental contamination because of multidrug-resistant *Acinetobacter baumannii* surrounding infected or colonised patients. *Am J Infect Control* 2011; **39**: 711–15.
37. Neely A, Maley MP and Warden GD. Computer keyboards as reservoirs for *Acinetobacter baumannii* in a burn hospital. *Clin Infect Dis* 1999; **29**: 1358–9.
38. Seifert H, Boullion B, Schulze A and Pulverer G. Plasmid DNA profiles of *Acinetobacter baumannii*: clinical application in a complex endemic setting. *Infect Control Hosp Epidemiol* 1994; **15**: 520–8.

39. Dancer SJ, White L, Robertson C. Monitoring environmental cleanliness on two surgical wards. *Int J Environ Hygiene* 2008; **18**: 357–64.
40. Shiomori T, Miyamoto H, Makishima K, *et al.* Evaluation of bedmaking-related airborne and surface methicillin-resistant *Staphylococcus aureus* contamination. *J Hosp Infect* 2002; **50**: 30–5.
41. Shiomori T, Miyamoto H and Makishima K. Significance of airborne transmission of methicillin-resistant *Staphylococcus aureus* in an otolaryngology-head and neck surgery unit. *Arch Otolaryngol Head Neck Surg* 2001; **127**: 644–8.
42. Bernards AT, Frénay HM, Lim BT, Hendriks WD, Dijkshoorn L and van Boven CP. Methicillin-resistant *Staphylococcus aureus* and *Acinetobacter baumannii*: an unexpected difference in epidemiologic behavior. *Am J Infect Control* 1998; **26**: 544–51.
43. Allen KD and Green HT. Hospital outbreak of multi-resistant *Acinetobacter anitratus*: an airborne mode of spread? *J Hosp Infect* 1987; **9**: 110–19.
44. Balm MN, Jureen R, Teo C, *et al.* Hot and steamy: outbreak of *Bacillus cereus* in Singapore associated with construction work and laundry practices. *J Hosp Infect* 2012; **81**: 224–30.
45. Fournel I, Sautour M, Lafon I, *et al.* Airborne *Aspergillus* contamination during hospital construction works: efficacy of protective measures. *Am J Infect Control* 2010; **38**: 189–94.
46. Dancer SJ, Coyne M, Robertson C, Thomson A, Guleri A and Alcock S. Environmental organisms from hospital wards with differing antibiotic exposure. *J Hosp Infect* 2006; **62**: 200–6.
47. Huang SS, Datta R and Platt R. Risk of acquiring antibiotic-resistant bacteria from prior room occupants. *Arch Intern Med* 2006; **166**: 1945–51.
48. Shaughnessy MK, Micielli RL, DePestel DD, *et al.* Evaluation of hospital room assignment and acquisition of *Clostridium difficile* infection. *Infect Control Hosp Epidemiol* 2011; **32**: 201–6.
49. Wilks M, Wilson A, Warwick S, *et al.* Control of an outbreak of multidrug-resistant *Acinetobacter baumannii calcoaceticus* colonization and infection in an Intensive Care Unit (ICU) without closing the ICU or placing patients in isolation. *Infect Control Hosp Epidemiol* 2006; **27**: 654–8.
50. Drees M, Snyderman DR, Schmid CH, *et al.* Prior environmental contamination increases the risk of acquisition of vancomycin-resistant enterococci. *Clin Infect Dis* 2008; **46**: 678–85.
51. Nseir S, Blazejewski C, Lubret R, Wallet F, Courcol R and Durocher A. Risk of acquiring multi-drug-resistant Gram-negative bacilli from prior room occupants in the intensive care unit. *Clin Microbiol Infect* 2010; **17**: 1201–8.
52. Vickery K, Deva A, Jacombs A, Allan J, Valente P and Gosbell IB. Presence of biofilm containing viable multiresistant organisms despite terminal cleaning on clinical surfaces in an intensive care unit. *J Hosp Infect* 2012; **80**: 52–5.
53. Dancer SJ and Carling PC. All that glistens may be neither gold nor clean. *J Hosp Infect* 2010; **76**: 177–8.
54. Dancer SJ. Control of transmission of infection in hospitals requires more than clean hands. *Infect Control Hosp Epidemiol* 2010; **31**: 958–60.
55. Casewell M and Phillips I. Hands as route of transmission for *Klebsiella* species. *BMJ* 1977; **2**: 1315–17.

56. Barker J, Vipond IB and Bloomfield SF. Effects of cleaning and disinfection in reducing the spread of norovirus contamination via environmental surfaces. *J Hosp Infect* 2004; **58**: 42–9.
57. 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**: 164–7.
58. Smith SJ, Young V, Robertson C and Dancer SJ. Cross-transmission audit of environmental surfaces, clinical equipment and patient: who touches what? *J Hosp Infect* 2012; **80**: 206–11.
59. Dancer SJ, White LF, Lamb J, Girvan EK and Robertson C. Measuring the effect of enhanced cleaning in a UK hospital: a prospective cross-over study. *BMC Infect Dis* 2009; **7**: 28.
60. Oelberg DG, Joyner SE, Jiang X, Laborde D, Islam MP and Pickering LK. Detection of pathogen transmission in neonatal nurseries using DNA markers as surrogate indicators. *Pediatrics* 2000; **105**: 311–15.
61. Rheinbaben F, Schunemann S, Gross T and 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**: 61–6.
62. Salzman TC, Clark JC and Klemm LM. Hand contamination of personnel as a mechanism of cross-infection in nosocomial infections with antibiotic resistant *Escherichia coli* and *Klebsiella-Aerobacter*. *Antimicrob Agents Chemother* 1967; **6**: 97–100.
63. Guenther SH, Owen Hendley J and Wenzel RP. Gram-negative bacilli as non-transient flora on the hands of hospital personnel. *J Clin Microbiol* 1987; **25**: 488–90.
64. Farrington M, Ling J, Ling T and French GL. Outbreaks of infection with methicillin-resistant *Staphylococcus aureus* on neonatal and burns units of a new hospital. *Epidemiol Infect* 1990; **105**: 215–28.
65. Hayden MK, Blom DW, Lyle EA, Moore CG and 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**: 149–54.
66. McBryde ES, Bradley LC, Whitby M and McElwain DL. An investigation of contact transmission of methicillin-resistant *Staphylococcus aureus*. *J Hosp Infect* 2004; **58**: 104–8.
67. Duckro AN, Blom DW, Lyle EA, Weinstein RA and Hayden MK. Transfer of vancomycin-resistant enterococci via healthcare worker hands. *Arch Intern Med* 2005; **165**: 302–7.
68. Morgan DJ, Rogawski E, Thom KA, *et al.* Transfer of multidrug-resistant bacteria to healthcare workers' gloves and gowns after patient contact increases with environmental contamination. *Crit Care Med* 2012; **40**: 1045–51.
69. Boyce JM, Potter-Bynoe G, Chenevert C and King T. Environmental contamination due to methicillin-resistant *Staphylococcus aureus*: possible infection control implications. *Infect Control Hosp Epidemiol* 1997; **18**: 622–7.
70. Creamer E, Dorrian S, Dolan A, *et al.* When are the hands of healthcare workers positive for methicillin-resistant *Staphylococcus aureus*? *J Hosp Infect* 2010; **75**: 107–11.
71. Guerrero DM, Nerandzic MM, Jury LA, Jinno S, Chang S and Donskey CJ. Acquisition of spores on gloved hands after contact with the skin of patients

- with *Clostridium difficile* infection and with environmental surfaces in their rooms. *Am J Infect Control* 2012; **40**: 556–8.
72. Stiefel U, Cadnum JL, Eckstein BC, Guerrero DM, Tima MA and Donskey CJ. Contamination of hands with methicillin-resistant *Staphylococcus aureus* after contact with environmental surfaces and after contact with the skin of colonized patients. *Infect Control Hosp Epidemiol* 2011; **32**: 185–7.
  73. Scott E and Bloomfield SF. The survival and transfer of microbial contamination via cloths, hands and utensils. *J Appl Bacteriol* 1990; **68**: 271–8.
  74. Scheithauer S, Oberröhrmann A, Haefner H, *et al.* Compliance with hand hygiene in patients with methicillin-resistant *Staphylococcus aureus* and extended-spectrum  $\beta$ -lactamase-producing enterobacteria. *J Hosp Infect* 2010; **76**: 320–3.
  75. Eckmanns T, Schwab F, Bessert J, *et al.* Hand rub consumption and hand hygiene compliance are not indicators of pathogen transmission in intensive care units. *J Hosp Infect* 2006; **63**: 406–11.
  76. Ehrenkranz NJ, Alfonso BC. Failure of bland soap handwash to prevent hand transfer of patient bacteria to urethral catheters. *Infect Control Hosp Epidemiol* 1991; **12**: 654–62.
  77. Jabbar U, Leischner J, Kasper D, *et al.* Effectiveness of alcohol-based hand rubs for removal of *Clostridium difficile* spores from hands. *Infect Control Hosp Epidemiol* 2010; **31**: 565–70.
  78. Lages SLS, Ramakrishan MA and Goyal SM. *In-vivo* efficacy of hand sanitisers against feline caliciviruses: a surrogate for norovirus. *J Hosp Infect* 2008; **68**: 159–63.
  79. Dancer SJ. The role of environmental cleaning in the control of hospital-acquired infection. *J Hosp Infect* 2009; **73**: 378–85.
  80. Layton MC, Perez M, Heald P and Patterson JE. An outbreak of mupirocin-resistant *Staphylococcus aureus* on a dermatology ward associated with an environmental reservoir. *Infect Control Hosp Epidemiol* 1993; **14**: 369–75.
  81. 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.
  82. 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**: 170–8.
  83. Martinez JA, Ruthazer R, Hansjosten K, Barefoot L and 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. *Archives Int Med* 2003; **163**: 1905–12.
  84. Sample ML, Gravel D, Oxley C, Toye B, Garber G and Ramotar K. An outbreak of vancomycin-resistant enterococci in a hematology–oncology unit: control by patient cohorting and terminal cleaning of the environment. *Infect Control Hosp Epidemiol* 2002; **23**: 468–70.
  85. Hayden MK, Bonten MJ, Blom DW, Lyle EA, van de Vijver DA and Weinstein RA. Reduction in acquisition of vancomycin-resistant *Enterococcus* after enforcement of routine environmental cleaning measures. *Clin Infect Dis* 2006; **42**: 1552–60.



86. Rossini FA, Fagnani R, Leichsenring ML, *et al.* Successful prevention of the transmission of vancomycin-resistant enterococci in a Brazilian public teaching hospital. *Rev Soc Bras Med Trop* 2012; **45**: 184–8.
87. Yoon YK, Sim HS, Kim JY, *et al.* Epidemiology and control of an outbreak of vancomycin-resistant enterococci in the intensive care units. *Yonsei Med J* 2009; **50**: 637–43.
88. Kaatz GW, Gitlin SD, Schaberg DR, *et al.* Acquisition of *Clostridium difficile* from the hospital environment. *Am J Epidemiol* 1998; **127**: 1289–94.
89. Wilcox MH, Fawley WN, Wrigglesworth 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**: 109–14.
90. McMullen KM, Zack J, Coopersmith CM, Kollef M, Dubberke E and Warren DK. Use of hypochlorite solution to decrease rates of *Clostridium difficile*-associated diarrhoea. *Infect Control Hosp Epidemiol* 2007; **28**: 205–7.
91. Hacek DM, Ogle AM, Fisher A, Robicsek A and Peterson LR. Significant impact of terminal room cleaning with bleach on reducing nosocomial *Clostridium difficile*. *Am J Infect Control* 2010; **38**: 350–3.
92. Orenstein R, Aronhalt KC, McManus JE Jr and Fedraw LA. A targeted strategy to wipe out *Clostridium difficile*. *Infect Control Hosp Epidemiol* 2011; **32**: 1137–9.
93. MacLeod-Glover N and Sadowski C. Efficacy of cleaning products for *Clostridium difficile*. *Can Family Physician* 2010; **56**: 417–23.
94. Doan L, Forrest H, Fakis A, Craig J, Claxton L and Khare M. Clinical and cost-effectiveness of eight disinfection methods for terminal disinfection of hospital isolation rooms contaminated with *Clostridium difficile* 027. *J Hosp Infect* 2012; **82**: 114–21.
95. Scerpella EG, Wanger AR, Armitige L, Anderlini P and 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**: 92–7.
96. Tankovic J, Legrand P, de Gatines G, Chemineau V, Brun-Buisson C and Duval J. Characterisation of a hospital outbreak of imipenem-resistant *Acinetobacter baumannii* by phenotypic and genotypic typing methods. *J Clin Microbiol* 1994; **32**: 2677–81.
97. Chmielarczyk A, Higgins PG, Wojkowska-Mach J, *et al.* Control of an outbreak of *Acinetobacter baumannii* infections using vaporized hydrogen peroxide. *J Hosp Infect* 2012; **81**: 239–45.
98. 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**: 106–10.
99. Apisarnthanarak A, Pinitchai U, Thongphubeth K, *et al.* A multifaceted intervention to reduce pandrug-resistant *Acinetobacter baumannii* colonization and infection in 3 intensive care units in a Thai tertiary care center: a 3-year study. *Clin Infect Dis* 2008; **47**: 760–7.
100. Hobson RP, MacKenzie FM and Gould IM. An outbreak of multiply-resistant *Klebsiella pneumoniae* in the Grampian region of Scotland. *J Hosp Infect* 1996; **33**: 249–62.

101. Randrianirina F, Vedy S, Rakotovao D, *et al.* Role of contaminated aspiration tubes in nosocomial outbreak of *Klebsiella pneumoniae* producing SHV-2 and CTX-M-15 extended-spectrum  $\beta$ -lactamases. *J Hosp Infect* 2009; **72**: 23–9.
102. Starlander G and Melhus A. Minor outbreak of extended-spectrum  $\beta$ -lactamase-producing *Klebsiella pneumoniae* in an intensive care unit due to a contaminated sink. *J Hosp Infect* 2012; **82**: 122–4.
103. Yang D and Zhang Z. Biofilm-forming *Klebsiella pneumoniae* strains have greater likelihood of producing extended-spectrum  $\beta$ -lactamases. *J Hosp Infect* 2008; **68**: 369–71.
104. Goddard S and Muller MP. The efficacy of infection control interventions in reducing the incidence of extended-spectrum  $\beta$ -lactamase-producing Enterobacteriaceae in the non-outbreak setting: a systematic review. *Am J Infect Control* 2011; **39**: 599–601.
105. Virgincar N, Iyer S, Stacey A, *et al.* *Klebsiella pneumoniae* producing KPC carbapenemase in a district general hospital in the UK. *J Hosp Infect* 2011; **78**: 293–6.
106. Soulier A, Barbut F, Ollivier JM, Petit JC and Lienhart A. Decreased transmission of Enterobacteriaceae with extended-spectrum beta-lactamases in an intensive care unit by nursing reorganisation. *J Hosp Infect* 1995; **31**: 89–97.
107. Kerr K and Snelling AM. *Pseudomonas aeruginosa*: a formidable and ever-present adversary. *J Hosp Infect* 2009; **73**: 338–44.
108. Tall BD, Williams HN, George KS, Gray RT and Walch M. Bacterial succession within a biofilm in water supply lines of dental air-water syringes. *Can J Microbiol* 1995; **41**: 647–54.
109. Costerton JW, Cheng K-J, Geesey GG, *et al.* Bacterial biofilms in nature and disease. *Ann Rev Microbiol* 1987; **41**: 435–64.
110. Doring G, Ulrich M, Muller W, *et al.* Generation of *Pseudomonas aeruginosa* aerosols during hand-washing from contaminated sink drains, transmission to hands of hospital personnel, and its prevention by use of a new heating device. *Zentralbl Hyg* 1991; **191**: 494–505.
111. Hota S, Hirji Z, Stockton K, *et al.* Outbreak of multi-drug resistant *Pseudomonas aeruginosa* colonisation and infection secondary to imperfect Intensive Care Unit room design. *Infect Control Hosp Epidemiol* 2009; **30**: 25–33.
112. Lucero CA, Cohen AL, Trevino I, *et al.* Outbreak of *Burkholderia cepacia* complex among ventilated pediatric patients linked to hospital sinks. *Am J Infect Control* 2011; **39**: 775–8.
113. Rutala WA and Weber DJ. Inorganic hypochlorite (bleach) use in healthcare facilities. *Clin Microbiol Review* 1997; **10**: 597–610.
114. Green J, Wright PA, Gallimore CI, Mitchell O, Morgan-Capner P and 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.
115. 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**: 802–10.
116. Love SS, Jiang X, Barrett E, Farkas T and 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**: 127–32.



117. Carling PC, Bruno-Murtha LA and Griffiths JK. Cruise ship environmental hygiene and the risk of norovirus infection outbreaks: an objective assessment of 56 vessels over 3 years. *Clin Infect Dis* 2009; **49**: 1312–17.
118. CDC. Norovirus outbreak in an elementary school – District of Columbia, February 2007. *Morb Mortal Wkly Rep* 2008; **56**: 1340–3.
119. Dharan S, Mourouga P, Copin P, Bessmer, G, Tschanz B and Pittet D. Routine disinfection of patients' environmental surfaces: Myth or reality? *J Hosp Infect* 1999; **42**: 113–17.
120. Moore G and Griffith C. A laboratory evaluation of the decontamination properties of microfibre cloths. *J Hosp Infect* 2006; **64**: 379–85.
121. Bergen LK, Meyer M, Hog M, Rubenhagen B and Andersen LP. Spread of bacteria on surfaces when cleaning with microfibre cloths. *J Hosp Infect* 2009; **71**: 132–7.
122. Engelhart S, Krizek L, Glasmacher A, Fischnaller E, Marklein G and Exner M. *Pseudomonas aeruginosa* outbreak in a haematology-oncology unit associated with contaminated surface cleaning equipment. *J Hosp Infect* 2002; **52**: 93–8.
123. Forder AA. Buckets and mops in operating theatres. *Lancet* 1973; **1**: 1325.
124. Wishart MM and Riley TV. Infection with *Pseudomonas maltophilia*: hospital outbreak due to contaminated disinfectant. *Med J Aust* 1976; **2**: 710–12.
125. McAllister TA, Lucas CE, Mocan H, *et al.* *Serratia marcescens* outbreak in a paediatric oncology unit traced to contaminated chlorhexidine. *Scott Med J* 1989; **34**: 525–8.
126. Naparstek L, Carmeli Y, Chmelnitsky I, Banin E and Navon-Venezia S. Reduced susceptibility to chlorhexidine among extremely-drug-resistant strains of *Klebsiella pneumoniae*. *J Hosp Infect* 2012; **81**: 15–19.
127. Werry C, Lawrence JM and Sanderson PJ. Contamination of detergent cleaning solutions during hospital cleaning. *J Hosp Infect* 1988; **11**: 44–9.
128. Hegstad K, Langsrud S, Lunestad BT, Scheie AA, Sunde M and Yazdankhah SP. Does the wide use of quaternary ammonium compounds enhance the selection and spread of antimicrobial resistance and thus threaten our health? *Microb Drug Resist* 2010; **16**: 91–104.
129. Reed CS, Barrett SP, Threlfall EJ and Cheasty T. Control of infection with multiple antibiotic resistant bacteria in a hospital renal unit: the value of plasmid characterisation. *Epidemiol Infect* 1995; **115**: 61–70.
130. Shlaes CL, Currie-McCumber C, Eanes M, Rotter G and Floyd R. Gentamicin-resistance plasmids in an intensive care unit. *Infect Control* 1986; **7**: 355–61.
131. Chagas TPG, Seki LM, da Silva DM and Asensi MD. Occurrence of KPC-2-producing *Klebsiella pneumoniae* strains in hospital wastewater. *J Hosp Infect* 2011; **77**: 281–2.
132. White L, Dancer SJ, Robertson C and McDonald J. Are hygiene standards useful in assessing infection risk? *Am J Infect Control* 2008; **36**: 381–4.
133. Lewis T, Griffith C, Gallo M and Weinbren M. A modified ATP benchmark for evaluating the cleaning of some hospital environmental surfaces. *J Hosp Infect* 2008; **69**: 156–63.
134. Mulvey D, Redding P, Robertson C, *et al.* Finding a benchmark for monitoring hospital cleanliness. *J Hosp Infect* 2011; **77**: 25–30.
135. Kay D, Bartram J, Pruss A, *et al.* Derivation of numerical values for the World Health Organization guidelines for recreational waters. *Water Res* 2004; **38**: 1296–304.

136. Pasquarella C, Pitzurra O and Savino A. The index of microbial air contamination. *J Hosp Infect* 2000; **46**: 241–56.
137. Sherlock O, O’Connell N, Creamer E and Humphreys H. Is it really clean? An evaluation of the efficacy of four methods for determining hospital cleanliness. *J Hosp Infect* 2009; **72**:140–6.
138. Boyce JM, Havill NL, Dumigan DG, Golebiewski M, Balogun O and Rizvani R. Monitoring the effectiveness of hospital cleaning practices by use of an adenosine triphosphate bioluminescence assay. *Infect Control Hosp Epidemiol* 2009; **30**: 678–84.
139. Aiken ZA, Wilson M and Pratten J. Evaluation of ATP bioluminescence assays for potential use in a hospital setting. *Infect Control Hosp Epidemiol* 2011; **32**: 507–9.
140. Silliker Inc. *Performance evaluation of various ATP detecting units*. Food Science Center, South Holland, Illinois. 2010.
141. Malik DJ and Shama G. Estimating bacterial surface contamination by means of ATP determinations: 20 pence short of a pound. *J Hosp Infect* 2012; **80**: 354–5.
142. Brown E and Eder AR, Thompson KM. Do surface and cleaning chemistries interfere with ATP measurement systems for monitoring patient room hygiene? *J Hosp Infect* 2010; **74**: 193–5.
143. Carling PC, Briggs JL, Perkins J and Highlander D. Improved cleaning of patient rooms using a new targeting method. *Clin Infect Dis* 2006; **42**: 385–8.
144. 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 Infectious Diseases* 2007; **7**: 61.
145. Guerrero D, Carling P, Jury L, *et al.* Beyond the ‘Hawthorne Effect’: Reduction of *Clostridium difficile* environmental contamination through active intervention to improve cleaning practices. Abstract 60; Fifth Decennial: International Conference on Healthcare-Associated Infections, March 2010, Atlanta USA.
146. Goodman ER, Platt R, Bass R, Onderdonk AB, Yokoe DS and 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**: 593–9.
147. Fitzgerald T, Sholtz LA, Marion N, Turner P, Carling PC and Rupp ME. Maintenance of environmental services cleaning and disinfection in the ICU after a performance improvement project. Poster no. 15-216. *Am J Infect Control* 2012; **40**: e159.
148. Kümmerer K. Significance of antibiotics in the environment. *J Antimicrob Chemother* 2003; **52**: 5–7.
149. Rutala WA and Weber DJ. Infection control: the role of disinfection and sterilization. *J Hosp Infect* 1999; **43**: S43–55.
150. Mandal J, Kate A and Parija SC. Microbicidal effect of electrolysed water. *J Hosp Infect* 2010; **76**: 94–5.
151. Berrington AW and Pedler SJ. Investigation of gaseous ozone for MRSA decontamination of hospital side-rooms. *J Hosp Infect* 1998; **40**: 61–5.

152. Department of Health. An integrated approach to hospital cleaning: microfibre cloth and steam cleaning technology. DoH, London (2007).
153. Meakin NS, Bowman C, Lewis M and Dancer SJ. Comparison of cleaning efficacy between in-use disinfectant and electrolysed water in an English residential care home. *J Hosp Infect* 2012; **80**: 122–7.
154. Boyle MA, O'Donnell MJ, Miller A, Russell RJ and Coleman DC. Control of bacterial contamination of washbasin taps and output water using Ecasol: a one-year study. *J Hosp Infect* 2012; **80**: 288–92.
155. Moore G and Griffith C. A laboratory evaluation of the decontamination properties of microfiber cloths. *J Hosp Infect* 2006; **64**: 379–85.
156. Falagas ME, Thomaidis PC, Kotsantis IK, Sgouros K, Samonis G and Karageorgopoulos DE. Airborne hydrogen peroxide for disinfection of the hospital environment and infection control: a systematic review. *J Hosp Infect* 2011; **78**: 171–7.
157. Sharma M and Hudson JB. Ozone gas is an effective and practical antibacterial agent. *Am J Infect Control* 2008; **36**: 559–63.
158. Jury LA, Cadnum JL, Jennings-Sanders A, Eckstein EC, Chang S and Donskey CJ. Evaluation of an alcohol-based power sanitizing system for decontamination of hospital rooms of patients with methicillin-resistant *Staphylococcus aureus* carriage. *Am J Infect Control* 2010; **38**: 234–6.
159. Maclean M, MacGregor SJ, Anderson JG, *et al.* Environmental decontamination of a hospital isolation room using high-intensity narrow-spectrum light. *J Hosp Infect* 2010; **76**: 247–51.
160. Nerandzic MM, Cadnum JL, Pultz MJ and Donskey CJ. Evaluation of an automated ultraviolet radiation device for decontamination of *Clostridium difficile* and other healthcare-associated pathogens in hospital rooms. *BMC Infect Dis* 2010; **10**: 197.
161. Umezawa K, Asai S, Inokuchi S and Miyachi H. A comparative study of the bactericidal activity and daily disinfection housekeeping surfaces by a new portable pulsed UV radiation device. *Curr Microbiol* 2012; **64**: 581–7.
162. Shepherd SJ, Beggs CB, Smith CF, Kerr KG, Noakes CJ and Sleigh PA. Effect of negative air ions on the potential for bacterial contamination of plastic medical equipment. *BMC Infect Dis* 2010; **10**: 92.
163. Page K, Wilson M and Parkin IP. Antimicrobial surfaces and their potential in reducing the role of the inanimate environment in the incidence of hospital-acquired infections. *J Mater Chem* 2009; **19**: 3819–31.
164. De Mynke W, De Belie N and Verstraete W. Antimicrobial mortar surfaces for improvement of hygienic conditions. *J Appl Microbiol* 2009; **108**: 62–72.
165. Nanda A and Saravanan N. Biosynthesis of silver nanoparticles from *Staphylococcus aureus* and its antimicrobial activity against MRSA and MRSE. *Nanomed* 2009; **5**: 452–6.
166. Weaver L, Michels HT and Keevil CW. Survival of *Clostridium difficile* on copper and steel: futuristic options for hospital hygiene. *J Hosp Infect* 2008; **68**: 145–51.
167. Taylor L, Phillips P and Hastings R. Reduction of bacterial contamination in a healthcare environment by silver antimicrobial technology. *J Infect Prevent* 2009; **10**: 6–12.

168. O'Hanlon SJ and Enright MC. A novel bactericidal fabric coating with potent *in vitro* activity against methicillin-resistant *Staphylococcus aureus* (MRSA). *Int J Antimicrob Agents* 2009; **33**: 427–31.
169. Casey AL, Adams D, Karpanen TJ, *et al.* Role of copper in reducing hospital environment contamination. *J Hosp Infect* 2010; **74**: 72–7.
170. D'arcy N. Antimicrobials in plastics: a global review. *Plast Addit Compd* 2001; **3**: 12–15.
171. Schmidt MG, Attaway HH, Sharpe PA, *et al.* Sustained reduction of microbial burden on common hospital surfaces through introduction of copper. *J Clin Microbiol* 2012; **50**: 2217–23.
172. Parkin IP and Palgrave RG. Self-cleaning coatings. *J Mater Chem* 2005; **15**: 1689–95.
173. Su W, Wei SS, Hu SQ and Tang JX. Preparation of TiO<sub>2</sub>/Ag colloids with ultraviolet resistance and antibacterial property using short chain polyethylene glycol. *J Hazard Mater* 2009; **172**: 716–20.
174. Hardy KJ, Gossain S, Henderson N, *et al.* Rapid recontamination with MRSA of the environment of an intensive care unit after decontamination with hydrogen peroxide vapour. *J Hosp Infect* 2007; **66**: 360–8.
175. Aldeyab MA, McElnay JC, Elshibly SM, *et al.* Evaluation of the efficacy of a conventional cleaning regimen in removing methicillin-resistant *Staphylococcus aureus* from contaminated surfaces in an intensive care unit. *Infect Control Hosp Epidemiol* 2009; **30**: 304–6.
176. Brady MJ, Lisay CM, Yurkovetskiy AV and Sawan SP. Persistent silver disinfectant for the environmental control of pathogenic bacteria. *Am J Infect Control* 2003; **31**: 208–14.
177. Attaway HH 3rd, Fahey S, Steed LL, Salgado CD, Michels HT and Schmidt MG. Intrinsic bacterial burden associated with intensive care unit hospital beds: Effects of disinfection on population recovery and mitigation of potential infection risk. *Am J Infect Control* 2012; **40**: 907–12.
178. von Kohn B. Closing the gap of inconsistent hand and surface sanitation. Poster no. 10-140. *Am J Infect Control* 2012; **40**: e114.
179. Bogusz A, Stewart M, Hunter J, Yip B, Reid D, Robertson C and Dancer SJ. How quickly do hospital surfaces become contaminated after detergent cleaning? *Healthcare Infection* 2013; **18**: 3–9.
180. Davies A, Pottage T, Bennett A and Walker J. Gaseous and air decontamination technologies for *Clostridium difficile* in the healthcare environment. *J Hosp Infect* 2011; **77**: 199–203.
181. Griffith CJ and Dancer SJ. Hospital cleaning: problems with steam cleaning and microfibre. *J Hosp Infect* 2009; **72**: 360–1.
182. Diab-Elschahawi M, Assadian O, Blacky A, *et al.* Evaluation of the decontamination efficacy of new and reprocessed microfiber cleaning cloth compared with other commonly used cleaning cloths in the hospital. *Am J Infect Control* 2010; **38**: 289–92.
183. Memarzadeh M, Olmstead RN and Bartley JM. Applications of ultraviolet germicidal irradiation disinfection in health care facilities: effective adjunct, but not stand-alone technology. *Am J Infect Control* 2010; **38**: S13–24.
184. Sweeney CP and Dancer SJ. Can hospital computers be disinfected using a hand-held UV light source? *J Hosp Infect* 2009; **72**: 92–4.

185. Sattar SA. Promises and pitfalls of recent advances in chemical means of preventing the spread of nosocomial infections by environmental surfaces. *Am J Infect Control* 2010; **38**: S34–40.
186. Russell AD. Bacterial adaptation and resistance to antiseptics, disinfectants and preservatives is not a new phenomenon. *J Hosp Infect* 2004; **57**: 97–104.
187. Dancer SJ. Hospital cleanliness: establishing a new science. *J Hosp Infect* 2012; **80**: 355.