The impact of environmental cleanliness on infection rates

variables such as staffing levels, staff compliance with infection-control procedures, the condition of work surfaces in the old and new hospitals, and the transfer of microbial contamination via equipment and furniture from the old to the new hospital. These factors may well have played a role in the incidence of infection.

However, it is significant that, despite the striking contrasts in the physical make-up of the two hospitals and the levels of environmental microbial contamination, the incidence of nosocomial infection remained the same.

**Study two**
In a more recent study, undertaken by Blythe et al. (1998), the rooms of patients who were both colonised by and infected with MRSA were swabbed to establish any evidence of environmental MRSA contamination. The screening was undertaken after terminal cleaning of the rooms was performed using detergent and disinfectant, namely hypochlorite solution, with 1,000ppm of available chlorine.

Areas sampled included those in close contact with the patient and areas likely to be frequently touched by the patient and/or staff. Sampling was undertaken using pre-moistened swabs, by the same two infection control nurses on all occasions. About 1,000 specimens were collected from 41 rooms. Nineteen rooms (46 per cent) showed evidence of environmental MRSA after terminal cleaning. The study concluded that environmental contamination with MRSA can readily occur and could act as a reservoir of infection, although there was no attempt made to quantify this data.

This study appears to have been well conducted, as a large number of swabs were taken, providing a large sample size. Two infection control nurses undertook the sampling, achieving both uniformity and eliminating the potential for bias. However, while the study demonstrated the presence of MRSA in the environment, it did not attempt to quantify any infection risk.

**Study three**
Another outbreak investigation was provided by Teare et al. (1998). They report that 24 patients on an elderly care ward developed diarrhoea over a three-and-half-month period. Twenty-one specimens were sent, in 16 of which *Clostridium difficile* was isolated.

Throughout the outbreak, strict attention was given to environmental cleaning, staff movement, handwashing and antibiotic usage, all of which are known risk factors for *C. difficile*. Despite these control measures and two complete ward closures the outbreak continued.

Eventually, it was observed that each of the radiators was surrounded by a ‘cage’ that was fixed to the wall, making removal difficult. As a result, routine cleaning behind the cage was not undertaken. On investigation, thick dust and dried faecal matter was found behind the cages. Microbiological testing of this material indicated the presence of *C. difficile*. Teare et al. (1998) noted that the outbreak began when the radiators were turned on in November and postulated that thermal convection from the radiators may have played a part in disseminating spores to vulnerable patients.

Clearly, this investigation identified *C. difficile* as being the causative agent. However, the theory that thermal convection disseminated spores to susceptible patients does not appear to have been tested or confirmed.

The investigation raises other questions. Most significantly, it does not indicate whether the isolated *C. difficile* samples were all the same strain, raising some doubt as to whether all the affected patients were indeed outbreak cases, or whether some were sporadic, individual cases.

Furthermore, it may be questioned how well antibiotic prescribing was controlled. This is particularly relevant considering that the outbreak took place during the winter months when antibiotic prescribing tends to be high, owing to clinical need such as treatment for chest infections. It may be that some of the patients developed diarrhoea and had confirmed *C. difficile* as a consequence of antibiotic regimes that they received rather than from exposure to spores via thermal convection from the radiators.

**Study four**
Wagenvoort et al. (2000) looked at whether there is better environmental survival of MRSA outbreak strains compared with sporadic strains. Five MRSA strains were isolated from five adult patients who were unknowingly colonised with MRSA and, therefore, admitted to hospital without specific infection control precautions.

The MRSA strains from patients one and two caused extensive outbreaks in the intensive care unit and surgical wards, with 32 and 14 patients affected respectively. Contact tracing was undertaken after detection of the different MRSA strains in the three other patients, but they did not cause any spread.

Microbiological testing was undertaken for all five strains. Survival was measured at two-weekly intervals, or longer, for about one year. The environmental survival patterns of the five MRSA strains showed qualitative and quantitative differences between the two outbreak and three sporadic strains. All survived longer than six months, but the two outbreak strains survived significantly better, in higher quantities and for one to three months longer.
Much has been said in recent years about the lack of environmental cleanliness in hospitals. The House of Lords Select Committee (1998) talked about falling standards in hospital cleaning and, more recently, the government acknowledged in the NHS Plan that hospitals are not always clean. Such comments may affect people’s perceptions of hospitals, and, rather than regarding them as safe environments, they may be viewed as dangerous places that might cause vulnerable patients greater harm. While it is known that about 10 per cent of hospital patients have a nosocomial infection at any one time (Emmerson et al, 1996), there is uncertainty about whether there is a sound evidence base to support the theory that dirty hospitals cause infection.

In any environment occupied by humans, microbes will be present in varying numbers (Collins, 1988). They enter the environment through a variety of routes, for instance, in body fluids, faeces, the respiratory route, and skin scales that have been shed. Dust is a hazard because it is largely made up of skin scales, and they are constantly being shed into the environment.

Each individual scale may be covered with a number of micro-organisms (Finn, 2000). ‘Super shredders’ may cause even greater environmental contamination. These are patients who, for example, may have a methicillin-resistant Staphylococcus aureus (MRSA) infection in wounds and in urinary catheters, rather than those who simply have MRSA nasal carriage (Boyce et al, 1997).

While it is accepted that microbes are all around us, there remains debate about whether dirty hospitals cause infections.

**Reviewing the literature**

A systematic review commissioned by the Department of Health and undertaken by Pratt et al (2001) for the formulation of national evidence-based guidelines on the prevention of hospital-acquired infection, including environmental hospital hygiene (Box 1), revealed that currently there is only limited research available of an acceptable quality.

However, a great deal of clinical evidence is available in the form of case reports and outbreak investigations (Dancer, 1999).

**Study one**

Maki et al (1982) undertook a prospective study involving a comparison between an old hospital environment and a new, more spacious, facility that provided more floor space, modern ventilation and single rooms for patients with infections. Environmental screening swabs were randomly taken from a number of surfaces, including sinks, taps and ice machines. Randomisation was achieved using a table of random numbers.

In the old hospital 276 swabs were taken, resulting in the isolation of common nosocomial pathogens, such as pseudomonas, acinetobacter species and S. aureus in 47 cases (17 per cent of the swabs taken). In the new hospital, 311 swabs were taken prior to occupancy, with common pathogens isolated in 14 cases (4.5 per cent).

After six to 12 months occupancy, this increased to 11.3 per cent in the new hospital. Except for the floors, all the surfaces sampled showed a lower level of microbial contamination than in the old hospital. Floors had a much higher level of contamination comparable with the old hospital.

A comparison was also made between the incidence of nosocomial infection in the old and new hospitals. In the last two months in the old hospital and in the first two months in the new hospital, the incidence of nosocomial infection was the same, namely, 6.9 infections per 100 discharges. After 10–12 months in the new hospital, the rate had changed, although not significantly (7.5 infections per 100 discharges).

The study found no decline in the rate of infection after moving into the new hospital; the incidence per week in the first two months remained constant.

Although acinetobacter was most frequently found in the environmental screening, it only accounted for two of the 369 nosocomial infections identified during the entire study. Neither of these two infections occurred in the old hospital, where the organism was predominantly isolated (9.1 per cent of all infection in the old hospital compared with 2.3 per cent in the new one). Hence, the study concluded that the environment did not alter nosocomial infection rates.

However, the study had limitations. It did not discuss...
This strain was also widespread in the ward environment. It was confirmed that the isolates from the patients and the environment were of the same strain. About a third of postoperative patients who acquired an infection were found to have this strain, despite implementation of standard infection control precautions, including hand hygiene, isolation of affected patients, and the staggered closure and cleaning of ward bays.

In September 1999 domestic cleaning hours were raised by 57 hours per week. Dust removal by vacuum cleaning and allocation of responsibility for the routine cleaning of shared medical equipment was also emphasised. In the subsequent six months only three patients were found to be colonised with the same strain of MRSA.

Monthly surveys of the environment also failed to find this MRSA strain. The study concluded that the dusty ward environment was an important source of MRSA during the outbreak and that standard infection control precautions alone were insufficient to end the outbreak.

However, as Rampling et al (2001) point out the connecting female surgical ward, which was equally dirty, did not experience a single case of the outbreak MRSA strain. This challenges the theory that a dusty ward environment causes MRSA infection. Other predisposing factors such as age and gender may also have been relevant. Previous studies have linked being male and aged 70–89 years with the acquisition of MRSA (Morgan et al, 2000; Johnson et al, 1997).

The study gave no indication of the staffing levels or the use of bank/agency staff on either the male outbreak ward or the adjoining female ward. Poor staffing levels may have resulted in a lack of compliance with infection control practices. Finally, the study did not discuss whether there was a so-called super-spreader on the male ward, who could have been responsible for the outbreak.

**Conclusion**

The belief that dirty hospitals cause infections is not one that can be readily substantiated on the basis of the available evidence (Box 2). This is because both the prevention and control of infection are determined not only by how clean or dirty a hospital is, but also by a host of other variables that include staffing levels, use of bank and agency staff, staff training on infection control, staff compliance with infection control procedures, and antibiotic prescribing regimes.

It may be concluded from the evidence that the hospital environment plays a notable role in infection control, but whether it causes infection is not known. There is some evidence to suggest that the environment plays a role in outbreaks of infection, rather than sporadic infections.

What is clearly known is that good hand hygiene is crucial to preventing and minimising the spread of infection (Pratt et al, 2001). Therefore, it is important that all health care professionals, as well as cleaning staff, at all times maintain the highest standards of infection control, including hand hygiene and hospital environmental hygiene.

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**Box 2. Summary of literature findings**

- Maki et al (1982) concluded that the environment did not alter nosocomial infection rates, although they did not look at variables such as compliance with infection control and the condition of work surfaces.

- Blythe et al (1998) found that environmental contamination with MRSA can occur even after terminal cleaning of rooms using detergent and disinfectant, although there was no attempt to quantify any infection risk.

- Teare et al (1998) concluded that thermal convection of spores from dirty radiators was the cause of an outbreak of diarrhoea. However, the finding was not tested and there are questions over whether all the affected patients were outbreak cases or whether some were individual sporadic cases.

- Wagenvoort et al (2000) found that outbreak strains of MRSA may have the capacity to spread more readily than sporadic strains as they are present in the environment for longer periods and in greater numbers. However, the study was conducted under experimental conditions, which may not apply to all hospitals.

- Rampling et al (2001) concluded that a dusty ward environment contributed to one particular MRSA outbreak and that standard infection control procedures were insufficient to end it. However, variables such as staffing levels or the presence of super-spreaders were not considered.

This study seems to indicate that outbreak MRSA strains are characteristically different from sporadic strains and may have the potential to spread more readily because they are present in the environment for longer and in greater numbers. This suggests that hospital hygiene may be significant in an outbreak rather than the cause of sporadic infections. However, the outbreak strain may have survived longer because of other factors, such as a higher initial inoculum and variations in temperature and light. It could also be argued that the study results obtained under experimental conditions are not applicable to real hospital conditions, as these vary from one hospital to another.

Wagenvoort et al’s study supports earlier work undertaken by Collins (1988), Maki et al (1982) and Aylliffe et al (1967), among others. They also suggest that the hospital environment plays a minor role in the spread of nosocomial infection.

**Study five**

In a more recent study undertaken by Rampling et al (2001), observational and microbiological data was collected from both patients and the environment on a male general surgical ward, over a 27-month period. From January 1998 to September 1999, 69 patients acquired an epidemic strain of MRSA, namely E-MRSA 16.