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Review

# Antimicrobial strategies for polymeric hygienic surfaces in healthcare

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ABSTRACT

Within healthcare facilitates, environmental contamination is often identified as a major contributor in the transfer of pathogenic hospital acquired infection (HAI). These infections although mainly from a small collection of bacteria, have given rise to one of the most severe and devastating complications associated with hospital stays. Further to this, data appears to indicate that increased length of stay significantly increases the likelihood of acquiring a HAI. For decades manufactures have used biocides to try and reduce the negative impacts of microbial colonisation on specific products. Historically, strategies have focused on incorporating biocidal preservative compounds to impede biofilm formation, with the aim to reduce diminished aesthetic or structural integrity (biodeterioration). However, more recently focus has shifted in trying to provide either a contact-killing surface or a surface that can significantly reduce microbial colonisation to a level below the threshold for which there is significant pathogen survival, with the aim to reduce transfer of infection. In this review, the focus is on the inclusion of antimicrobials into the almost ubiquitous polymer based surfaces, as a means of reducing contamination within the healthcare environment. This includes already established techniques, such as incorporating inorganic and organic biocides into the polymer matrix and surface coatings, as well as more modern techniques such as light activation and nanoparticles. Further to this, the importance of cleaning and personal hygiene is considered, as well as the potential impact of future policy and legislation strategy.


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## 1. Introduction

In contrast with recent times, hospitals in Asia and Southern Europe around 2500 years ago provided hygienic and clean conditions that seem not to have been matched in western Europe until less than a hundred years ago (Selwyn, 1991). Whilst the ancient conditions were mandated by religion, the process that lead to improved modern standards was founded on the scientific investigations which examined the relationship between microorganisms and disease (Forder, 2007). Agostino Bassi (1773–1856), first presented in 1835, the pathogenic potential of a microorganism when he demonstrated that a silkworm disease was due to a fungal infection. Louis Pasteur (1822–1895) later demonstrated a different silkworm disease which was blighting the French silk industry, and was due to a protozoan parasite. Indirectly, Joseph Lister (1827–1912) highlighted the importance of microorganisms in human disease, with his strategy for preventing wound infection by means of a phenol spray which killed bacteria. Florence Nightingale's observations also provided further epidemiological data in support of a substantial link between hygiene and infection (Keith, 1988; Sofiyanti et al., 2015). However, final definitive proof of the involvement of microorganisms in human disease was provided by Robert Koch (1843–1910), when he proved that *Bacillus anthracis* was the aetiological agent of anthrax. His experiments identifying the relationship between bacteria and disease led to development of Koch's postulates, a procedure for classifying an infection caused by a microorganism (Koch, 1890).

The association between environmental contamination within healthcare and potential pathogenic infection is now well established. Even with the prevalence of hospital acquired infections (HAI's) in the UK's National Health Service (NHS) falling from 9.2% of patients in 1980, to 6.4% in 2011, this magnitude of infection can still have significant health and economic impacts when annual patient admittance is considered (Public Health England, 2016). This problem is also echoed across Europe with around 4 million people annually acquiring a HAI (Public Health England, 2016). HAI's as a high as 23.4% have been recorded in intensive care units (ICUs) patients and 8% in surgical ward patients (Hopkins et al., 2012). In the USA the Centers for Disease Control (CDC) reported a 4% likelihood of a patient contracting a HAI throughout the duration of their hospital care (Centers for Disease Control and Prevention, 2016). The length of hospital stay has also been shown to significantly increase a patient's likelihood of contracting an HAI (Carr et al., 2007; Freeman and McGowan, 1984; Nguyen-Van-Tam et al., 1999). Two theories currently attempt to explain this phenomenon. Firstly longer hospital stays equate to a longer exposure time to potential pathogens within the healthcare environment (Weber et al., 2010). Secondly, longer hospital stays are often the result of more serious conditions, or the result of invasive procedures, which carry with them a greater risk (Emori and Gaynes, 1993; Freeman and McGowan, 1984; Higgins et al., 2003; Vilins et al., 2009). In practice, the mechanism most likely involves a combination of environmental, clinical and biological factors. Interestingly, Jeon et al. (2012) evaluated 113,893 inpatient hospital admissions in order to study the effect of hospital stay length and the probability of developing a healthcare-associated bloodstream infection (HABI). The authors used a risk-adjusted time model which accounted for more serious procedures and therefore, increased risk of infection, often resulting in longer stays. Their results indicated that the effect of stay length in hospital on HABI's, was greatly reduced when the implicit infection risk of more complicated procedures was taken into account. However, the authors did highlight a number of potential shortcomings of the study, suggesting that some unmeasured cofactors may, if accounted for, change the findings drastically (Jeon et al., 2012).

Whilst almost all pathogens have the potential to become problematic in hospitalised patients, the aetiology of most HAI's can be traced back to only a few bacteria, with data showing that *Staphylococcus aureus, Pseudomonas aeruginosa, Escherichia coli,* coagulase-negative staphylococci (CoNS) (predominantly *S. epidermidis)*, *Enterococcus* spp. (predominately *E. faecalis* and *E. faecium)* are reportedly the most often species isolated in nosocomial infection (Bereket et al., 2012; Emori and Gaynes, 1993; Martin et al., 1989). Potentially the small number of responsible pathogens could make the task of controlling nosocomial infection sound simple, however, the ubiquitous nature of these species and the difficulties that come with dissemination practices or events mean this isn't the case. Furthermore, the ever increasing prevalence of antimicrobial resistant (AMR) pathogens has led to global concern (Andersson and Hughes, 2010; Jones, 2001; Laxminarayan et al., 2013). Microbial resistance has now been reported to the majority of currently used antibiotics (Jones, 2001), resulting in over 2 million AMR infections in the USA in 2013 alone (CDC, 2013). As a consequence, the CDC and Public Health England (PHE) have expressed concern over the increasing prevalence of antimicrobial resistance (CDC, 2013; Public Health England, 2014), especially that associated with *Clostridium difficile* and carbapenems, and third-generation cephalosporin resistance among enterobacteriaceae (Falagas et al., 2014; Huang et al., 2012; Metri et al., 2013; Poire et al., 2011; Wilcox et al., 2013). For example in 2015, PHE reported an increase in *E. coli* and *Klebsiella pneumoniae* bloodstream infections, between 2010 and 2014, by 15.6% and 20.8% respectively. These problems are further highlighted by continued methicillin-(oxacillin-) resistant *S. aureus*, multidrug-resistant pneumococci, and vancomycin-resistant enterococci (Anstead et al., 2014; Cetinkaya et al., 2000; Cillóniz et al., 2016; Lee, 2003; Van Tonder et al., 2015). Additionally, health organisations have expressed significant alarm at the potential for a pandemic development of extended-spectrum β-lactamases (ESBLs) in *K. pneumoniae*, *E. coli* and *Proteus mirabilis* (Coudron et al., 2000; Pitout and Laupland, 2008; Pitout et al., 1998; Weinstein et al., 2005). The emergence of multidrug- and pandrug-resistance serotypes of *Pseudomonas aeruginosa*, Acinetobacter, and *Stenotrophomonas maltophilia* have also placed increased stress on the already limited potential