Chapter 2
Survival of Microorganisms on Inanimate Surfaces

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Abstract In healthcare settings microbial contaminated surfaces play an important role in indirect transmission of infection. Especially surfaces close to the patients’ environment may be touched at high frequencies, allowing transmission from animated sources to others via contaminated inanimate surfaces.

Therefore, the knowledge on the survival of bacteria, fungi, viruses and protozoa on surfaces, and hence, in a broader sense, in the human environment, is important for implementing tactics for prevention of Healthcare-acquired Infections (HAI).

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This chapter will elaborate the role of surfaces in the transmission of pathogens. Particular emphasis is laid on the current knowledge of the survival time and conditions favouring survival of the pathogens. Finally, mechanisms of transmission from inanimate surfaces to patients are highlighted.

Within the multi-barrier strategy of the prevention of HAI, environmental disinfection policies should be based on risk assessments for surfaces with different risks for cross contamination such as high- and low-touched surfaces with appropriate standards for adequate disinfection measures under consideration of the persistence and infectious dose of the pathogens. As a result, surface disinfection is indicated in the following situations:

- Frequently touched surfaces adjacent to patients
- Surfaces with assumed or visible contamination
- Terminal disinfection in rooms or areas where infected or colonized patients with easily transferable nosocomial pathogens are cared for, and
- in outbreak situations.

Furthermore, the knowledge of the persistence of pathogens will also support ensuring the biosafety in microbiological and biomedical laboratories, food-handling settings, and for hygienic behaviour in the everyday life to prevent transmission of infectious diseases.

**Keywords** Persistence • Bacteria • Fungi • Viruses • Protozoa transmission mechanisms • Surface disinfection

**List of Abbreviations**

| Abbreviation | Description                                      |
|--------------|--------------------------------------------------|
| HAI          | Healthcare-acquired infections                   |
| MRSA         | Methicillin-resistant *Staphylococcus aureus*    |
| MSSA         | Methicillin-sensible *Staphylococcus aureus*     |
| RH           | Relative humidity                                |
| SARS         | Severe acute respiratory syndrome                |
| VRE          | Vancomycin-resistant enterococci                 |

**2.1 Introduction**

Microorganisms may be transmitted from animated sources to inanimate environmental sources, which may become secondary reservoirs if they meet the needs of transmitted pathogens to survive and to multiply. In healthcare settings, however, contaminated surfaces, which may not always be optimal for microbial survival and multiplication, still may play a role in the chain of infection, since surfaces close to the patients’ environment may be touched at high frequencies, allowing transmission from animated sources to others via contaminated inanimate surfaces.
Because of this, the knowledge on the survival of bacteria, fungi, viruses and protozoa on surfaces, and hence, in a broader sense, in the human environment, is important for planning and implementing tactics for prevention of Healthcare-acquired Infections (HAI). Furthermore, such knowledge will also assist ensuring the biosafety in microbiological and biomedical laboratories, food-handling settings, and for hygienic behaviour in the everyday life to prevent transmission of infectious diseases.

One example of microorganisms with relatively short ability of persisting in the environment is the severe acute respiratory syndrome (SARS) coronavirus (CoV), which became pandemic within months in China in 2002. This virus retains infectivity on different substrates up to 9 days, as compared to the influenza virus, which demonstrates a relatively long persistence in the environment up to 4 weeks [112]. Both viruses are airborne transmitted infectious agents, however, they may also be transmitted via hand-surface contacts, supporting the relevance of hand hygiene and personal protection against infection.

Because of a number of microorganisms’ ability to persist and survive for long-term periods on surfaces, particularly in healthcare settings, the usage of antimicrobially impregnated surfaces is increasingly discussed [82]. However, because of the required long contact times of microorganisms on antimicrobial surfaces [64, 65, 25, 45], such technologies may be useful for surfaces with low frequency of hand contacts.

2.2 The Role of Surfaces in the Transmission of Pathogenic Microorganisms Causing Healthcare-Acquired Infections (HAI)

In healthcare settings, bacteria, bacterial spores, viruses and yeasts are mainly transmitted from infected and/or colonized patients, but also from staff, and in some situations from visitors to the inanimate hospital environment, particularly to areas adjacent to patients and frequently touched surfaces by hands (“high-touch surfaces”). Potential pathogenic microbial flora of the respiratory tract and of the vestibulum nasi, such as methicillin-sensible (MSSA) or resistant *Staphylococcus aureus* (MRSA), is correlated with a higher risk of contamination of surrounding surfaces through direct or indirect contact with hands [81]. Intestinal infections caused i.e. by *Clostridium difficile* and Norovirus, or enteral colonization with nosocomial pathogens such as vancomycin-resistant enterococci (VRE) may also be associated with a risk of widespread environmental contamination [30]. Compared with the large number of published literature on environmental contamination with MRSA, VRE, and *C. difficile*, there are relatively few published studies on environmental contamination by Gram-negative bacteria [64, 65]. Aside of a possible publication bias in the past, one reason for this is the different ability of Gram-positive and Gram-negative bacteria to survive in the inanimate environment.

The level of microbial bio-burden on surface in healthcare settings is low compared to the numbers on patients’ skin or in faeces. However, even at low particle numbers
there is a risk of transmission (Table 2.1). In immuno-compromised patients, the required numbers of microorganisms for causing infectious diseases is even lower, increasing the risk of HAI in these populations. Inanimate surfaces have been described as source for HAI-outbreaks. Hayden et al. [49] demonstrated that touching the environment contaminated with relatively low pathogen concentrations in a room occupied by a patient colonized with VRE is associated with approximately the same risk of VRE acquisition on hands as touching an affected patient directly. Evidence of the importance of environmental transmission is further provided by studies showing an increased risk of infection in patients admitted to the same rooms previously occupied by other infected/colonised cases. This has been shown for \textit{C. difficile} [101], VRE and MRSA ([54, 55], and also own observations). Environmental Norovirus contamination has been repeatedly found to be correlated with continuing outbreaks [128], although the significance of this pathway has not been fully elucidated.

The importance of surface contamination is also shown by reduction in the rate of HAI when effective measures of environmental disinfection are implemented [50, 10, 26]. A recent observational study showed a significant reduction in \textit{C. difficile} infection rates following the introduction of sporicidal wipes in an environmental cleaning regimen in an acute London trust [16]. However, not all studies have shown a direct link between surface disinfection and reduction in infection rates, probably because of the complex interactions and transmission routes in the clinical practice.

Yet, in summary it is undisputed that contaminated surfaces may contribute to the transmission of pathogens and may thus pose a critical element in the chain of transmission of microorganisms [41].

### 2.3 Persistence of Microorganisms on Inanimate Surfaces

The risk for transmission of HAI depends of the persistence of nosocomial pathogens on surfaces. The longer a microorganism may persist on a surface, the longer the contaminated surface may be a source of transmission and thus endanger a susceptible patient or healthcare worker of becoming the target of infection. In order to estimate the risk of cross contamination, Kramer et al. [64, 65] have published a systematic review on persistence of pathogens on surfaces.
The following findings are based on this review; however, knowledge on persistence of microorganisms on inanimate surfaces is now expanded by additional findings published after 2005/2006.

### 2.3.1 Persistence of Bacteria

In most reports, persistence was studied on dry surfaces using artificial contamination of a standardized type of surface in a laboratory. Bacteria were prepared in broth, water or saline.

Most Gram-positive bacteria, such as *Enterococcus* spp. including VRE, *S. aureus* including MRSA, or *Streptococcus pyogenes* survive for months on dry surfaces (Table 2.2). In general, there is no observable difference in survival between multi-resistant and susceptible strains of *S. aureus* and *Enterococcus* spp. [78]. Only in one study [118] a difference of survival time between antibiotic resistant and susceptible bacteria was suggested, yet, the susceptible strains demonstrated only a non-significant shorter survival time on surfaces. The factors why the same bacteria may persist more or less on a surface (i.e. from hours to days as detailed in Table 2.2) will be discussed later in Sect. 2.3.5.

Many Gram-negative species, such as *Acinetobacter* spp., *Escherichia coli*, *Klebsiella* spp., *Pseudomonas aeruginosa*, *Serratia marcescens*, or *Shigella* spp. can survive on inanimate surfaces even for months (Table 2.2). These species are found among the most frequent isolates from patients with HAI [64, 65]. However, a few others Gram-negative bacteria, such as *Bordetella pertussis*, *Haemophilus influenzae*, *Proteus vulgaris*, or *Vibrio cholera* persist only for days (Table 2.2).

Mycobacteria, including *Mycobacterium tuberculosis*, and spore-forming bacteria, such as *C. difficile*, can survive for many months on surfaces (Table 2.2).

Because paper still is omnipresent in healthcare settings worldwide today, Hübner et al. [56] have analysed the persistence of various Gram-positive and Gram-negative bacteria including *E. coli*, *S. aureus*, *P. aeruginosa*, and *Enterococcus hirae* on office paper after contamination with standardised inocula of bacterial suspensions in the range of $2.8 \times 10^7$ cfu/mL. Opposite to *E. coli*, all other organisms were more stable at room conditions and were reduced on paper only by $3 \log_{10}$ after 7 days, whereas *E. coli* was reduced by $5 \log_{10}$ within 24 h. Furthermore, the transmissibility of bacteria from hands to paper and back could be demonstrated for all bacteria strains. Similar investigations showed that paper money notes could harbour and transmit pathogens [62, 111, 115].

### 2.3.2 Persistence of Viruses

In order to estimate the persistence of viruses on inanimate surfaces, usually cell culture media are prepared [64, 65]. Most viruses from the respiratory tract such as Corona-, Coxsackie-, or Influenza virus, SARS, or rhinovirus can persist on surfaces
Table 2.2 Published data on survival of nosocomial and community acquired pathogens on various inanimate surfaces

| Organism                        | Range of survival (environment)                                                                 | Reference |
|--------------------------------|-------------------------------------------------------------------------------------------------|-----------|
| Acinetobacter spp.             | 3 days to 1 year (in-vitro) 36 days within biofilm vs. 15 days for non-biofilm-forming strains | Wagenvoort and Joosten [117], Espinal et al. [36]a |
|                                |                                                   | Hunter [57], Walther and Ewald [121]a            |
| Bordetella pertussis           | 3 to >10 days; in pernasal swabs: >4 days                                                      | González and Hänninen [44]a                      |
| Campylobacter jejuni           | >6 days, in water >60 days                                                                      |           |
| Clostridium difficile spores   | 5 months                                                                                        | Weber et al. [123]a                              |
| C. difficile, vegetative form  | 15 min (dry surface) 6 h (moist surface)                                                       |           |
| Chlamydia pneumoniae           | ≤96 h                                                                                            | Fukumoto et al. [40], Haider et al. [51], Matsuo et al. [70]a |
| C. trachomatis                 | <1 week                                                                                         |           |
| Chlamydia psittaci             | 15 days to months (environment)                                                                 | Wendel [125]a                                   |
| Corynebacterium diptheriae     | 7 days to 6 months                                                                              | Walther and Ewald [121]p                         |
| Corynebacterium pseudotuberculosis | 1–8 days, up to several weeks (environment)                                                      | Yeruham et al. [129]a, Dorella et al. [31]       |
| Enterococcus spp. including VRE| 5 days up to 30 months                                                                          | Robine et al. [97], Wagenvoort et al. [116]a     |
| Escherichia coli               | 1.5 h to 16 months                                                                              | Guan and Holley [46], Erickson et al. [35], Chaur et al. [19]a, Duffitt et al. [33] |
| E. coli O157:H7                | 27 days on spinach leaves, 179 days in soil, 98 days in water                                    |           |
| Haemophilus influenzae         | 12 days                                                                                         |           |
| Helicobacter pylori            | ≤90 min; in water: 2–30 days                                                                    | West et al. [124], Percival and Thomas [89]a     |
| Klebsiella spp.                | 2 h to >30 months, ≤144 h in detergent solution                                                 | Beadle and Verran [6]a                          |
| Listeria spp.                  | 1 day–months, 141 days in water                                                                 | Busdzińska et al. [13]a                         |
| Mycobacterium bovis            | >2 months                                                                                       |           |
| Mycobacterium tuberculosis     | 1 day up to 4 months                                                                             | Walther and Ewald [121]p                         |
| Neisseria gonorrhoeae          | 1–3 days                                                                                       |           |
| Neisseria meningitidis         | 72 h                                                                                           | Tzeng et al. [110]a                             |
| Parachlamydia acanthamoebae    | <4 weeks, in presence of blood <7 weeks                                                          | Fukumoto et al. [40]a                           |
| Proteus vulgaris               | 1–2 days                                                                                       |           |
| Pseudomonas aeruginosa         | 6 h up to 16 months; on dry floor: 5 weeks; in aerosol: few hours                               | Clifton et al. [21]a                            |
| Salmonella typhi               | 6 h up to 4 weeks                                                                               |           |
| Salmonella typhimurium         | 10 days up to 4.2 years                                                                        |           |

(continued)
only for a few days [18]. Herpes viruses such as Cytomegalie virus or Herpes simplex virus type 1 and 2 have been shown to persist from only a few hours up to 7 days. Viruses from the gastrointestinal tract, such as Astrovirus, Hepatitis A virus, Polio- and Rotavirus persist significantly longer for approximately 2 months. Blood-borne viruses, such as Hepatitis B virus or Human Immunodeficiency virus can persist for more than 1 week (Table 2.3).

### 2.3.3 Persistence of Fungi

*Candida albicans*, the most important nosocomial yeast, can survive up to 4 months on surfaces. Persistence of other yeasts was described to be similar (*Torulopsis glabrata*: 5 months) or shorter (*Candida parapsilosis*: 14 days) (Table 2.4). The survival of fungi in the environment, however, is strongly influenced by physical factors in nature, such as temperature and relative humidity (see Sect. 2.3.5).
| Organisms                           | Range of survival (environment)                                                                                   | Reference                                      |
|------------------------------------|------------------------------------------------------------------------------------------------------------------|-----------------------------------------------|
| Adenovirus                         | <6 h up to 3 months (type dependent), ≤301 days (in water)                                                       | Hara et al. [48], Rigotto et al. [95]²         |
| Astrovirus                         | 7–90 days                                                                                                        |                                               |
| Avian metapneumonovirus            | ~48 h up to 6 days                                                                                                | Tiwari et al. [107]²                          |
| SARS Coronavirus                   | <5 min up to 24 h (on paper)                                                                                       | Lai et al. [66], Rabenau et al. [93], Guionie et al. [47] |
| Coxsackievirus                     | 7–10 days, up to >2 weeks                                                                                         | Wong et al. [127]²                            |
| Cytomegalovirus                    | 1–8 h                                                                                                             | Faix [37], Stowell et al. [102]²              |
| Echovirus                          | Up to 7 days                                                                                                      |                                               |
| Hepatitis A virus                  | 2 h up to 60 days                                                                                                |                                               |
| Hepatitis B virus                  | ≥1 week                                                                                                           |                                               |
| Human immunodeficiency virus       | Up to 7 days, 7 days (in peritoneal dialysis effluent), 48 h (on peritoneal dialysis exchange and tubing), 4–8 weeks (on glass cover slides) | Van Bueren et al. [113], Farzadegan et al. [38]² |
| Herpes simplex virus, Type 1 & 2   | <2 h up to 8 weeks                                                                                                | Larson and Bryson [67], Bardell [2], Rabenau et al. [93]² |
| Influenza virus                    | 1–28 days (strain dependent)                                                                                       | Edward and Derrick [34], Walther and Ewald [121], Tiwari et al. [107]², Thomas et al. [106] |
| Marburg virus (strain Popp)        | 4–5 days                                                                                                          | Belanov et al. [7]²                           |
| Para-influenza virus               | 10 h                                                                                                               | Brady et al. [11]²                            |
| Norovirus, Feline calici virus (FCV), Murine norovirus (MNV) | 8 h up to 7 days, MNV > 40 days (in diapers and gauze)                                                        | Cannon et al. [14], Lee et al. [69]²          |
| Papillomavirus 16                  | ≤7 days                                                                                                            | Hsueh [53]²                                   |
| Papovavirus                        | 8 days                                                                                                             |                                               |
| Parvovirus                         | >1 year                                                                                                            |                                               |
| Poliovirus type 1                  | 4 h to <8 days                                                                                                     |                                               |
| Poliovirus type 2                  | 1 day up to 8 weeks                                                                                                |                                               |
| Pseudorabies virus                 | ≥7 days, <1 h (in aerosol infectivity decreases by 50 % per hour)                                                  | Schoenbaum et al. [100]                       |
| Respiratory syncytial virus        | up to 6 h                                                                                                          |                                               |
| Rhinovirus                         | 2 h up to 7 days                                                                                                   |                                               |
| Rotavirus                          | 30 min, 6–60 days                                                                                                  | Keswick et al. [61]²                          |
| Vaccinia virus                     | 3 weeks up to >20 weeks                                                                                           |                                               |

²Additional references in Kramer et al. [64, 65]
Moulds are ubiquitous in nature, thermo-tolerant, and can survive in house dust for long time. Indoor airborne mould measurements underline the survival for several months [4, 5].

**2.3.4 Persistence of Other Pathogenic Microorganisms**

*Cryptosporidium* spp. can induce water-born infection. Their oocysts can survive for months in surface water [96, 20, 75, 15], and up to 120 days in soil [60].

Acanthamoeba are one of the most common protozoa in soil, and frequently found in fresh water and other environmental habitats. An important habitat and vector for infection are hydrogel contact lenses, resulting in contact lens associated keratitis caused by acanthamoeba and fusarium [87], particularly since the contact lenses’ moist condition supports survival protozoa.

**2.3.5 Factors Influencing the Survival of Microorganisms in the Environment**

**2.3.5.1 Relative Humidity (RH)**

Generally, viruses with lipid envelops, such as most respiratory viruses including Influenza virus, Para-Influenza virus, Corona virus, Respiratory syncytial virus, Herpes simplex virus, Measles virus, Rubella virus, and Varicella zoster virus will tend to survive longer at lower relative humidity (20–30 % RH) [103]. However,
Cytomegalie virus makes an exception, as it was more likely isolated from moist surfaces [102].

Conversely to enveloped viruses, non-lipid enveloped viruses such as Adenovirus, Enterovirus, and Rhinoviruses tend to survive longer at higher relative humidity (70–90 % RH) [103]. For Rotavirus and Poliovirus conflicting results were reported [64, 65].

*S. aureus* can persist longer at low humidity [74]. However, for *Enterococcus faecalis* the survival kinetic is decreased at 25 % RH compared to 0 % RH [97].

The survival of aerosolized Gram-negative bacteria including *Pseudomonas* spp., *Enterobacter* spp. and *Klebsiella* spp. improved at higher relative humidity and low temperature [103]. Studies on airborne Gram-negative bacteria such as *S. marcescens*, *E. coli*, *Salmonella pullorum*, *Salmonella derby*, and *Proteus vulgaris* showed decreased survival at intermediate (approx. 50–70 % RH) to high (approx. 70–90 % RH) relative humidity. For some airborne Gram-positive bacteria, such as *Staphylococcus epidermidis*, *Streptococcus haemolyticus*, *Bacillus subtilis*, and *Streptococcus pneumoniae*, their survival rate also decreased at intermediate relative humidity ranging at 50–70 % RH [103]. Gram-positive cocci were most prevalent in indoor air, followed by Gram-positive rods (e.g. *Bacillus* spp. and *Actinomycetes* spp.), Gram-negative rods and Gram-negative cocci [103]. The reason for this bacterial behaviour is the design of bacterial cell wall, which allows Gram-positive organisms to tolerate dry conditions better than Gram-negative organisms. Because of a lipid double-layer structure with a thin peptidoglycan (Murein) layer consisting of alternating residues of β-(1,4) N-acetylglucosamine and N-acetylmuramic acid, the later are not so well protected against physical stress and need higher RH in order to survive.

### 2.3.5.2 Temperature

The viral genome (viral DNA or RNA) is sensitive to the surrounding temperature. Indeed, temperature is an important factor influencing the survival of a number of viruses. Higher temperatures impact viral proteins and enzymes, as well as the viral genome. In general, DNA viruses are more stable than RNA viruses; yet, high temperature also will affect DNA integrity.

For most viruses, such as Astrovirus, Adenovirus, Poliovirus, Herpes simplex virus, and Hepatitis A virus, low temperature is associated with a longer persistence [64, 65]. Constant temperatures >24 °C appear universally to decrease airborne bacterial survival [103].

### 2.3.5.3 Biofilm

Biofilm is the predominant form of life for microorganisms in a nutrient-sufficient ecosystem. Adhesion triggers the expression of a sigma factor that depresses a large number of genes so that bacteria within the biofilm are at least 500 times more
tolerable against antimicrobial agents [23] as well as against physical cold plasma [71, 72]. The reason for the unspecific increased tolerance is the production of extracellular substances like polysaccharides, proteins and DNA after attachment to surfaces. A precondition for biofilm formation is the presence of certain amounts of humidity. The biofilm matrix restrains water and nutrients and protects the microorganisms against environmental influences [28, 39]. Because of that, once formed biofilms are an important factor of persistence of microorganisms on surfaces in nature as well as in industrial or medical areas [22, 29, 12]. The persistence on inanimate surfaces is prolonged and depends on the environmental conditions, especially the humidity. Also on hospital surfaces biofilms were demonstrated on a number of objects and surfaces, such as sterile supply buckets, opaque plastic doors, venetian blind cords, and sink rubbers, and it was possible to cultivate viable bacteria. Currently, there is not enough research to elucidate whether presence or absence of biofilm affect the risk of transmission or possibility for cross-transmission. However, multi-drug resistant bacteria may not only be protected within biofilms, which may be the mechanism why they persist within the hospital environment [114], but may also exchange virulence factors among their own species or to other species present in biofilms as well [29, 43, 109].

2.3.5.4 Other Factors

A number of other factors may influence the survival of microorganisms on surfaces. Clearly, the material character of a surface itself may play in important role. However, inconsistent results are reported for the influence of type of materials on microbial survival. Some authors described that the type of material did not affect the persistence of Echovirus, Adenovirus, Para-Influenza virus, Rotavirus, Respiratory syncytial virus, Poliovirus, or Norovirus. Other investigators found that persistence was favoured on non-porous surfaces for Influenza virus on formica and gloves for Respiratory syncytial virus, and on hand pieces of telephones for Feline calicivirus [64, 65]. Other factors for a longer persistence of viruses include the presence of faecal suspension and a higher bio-inoculum [66, 64, 65]. Interestingly and by nature, Urease activity enhances the survival of *Haemophilus influenzae* at a reduced pH [77].

2.3.6 Limitations on the Knowledge of Microbial Survival on Inanimate Surfaces

Laboratory studies to determine the survival and persistence do not reflect the clinical situation, in which surfaces can be simultaneously contaminated with various nosocomial pathogens, different types of bodily and other fluids, secretions,
and antimicrobial residues, i.e. from the last surfaces disinfection. However, little dispute exists that beside the hands of healthcare workers surfaces in the close vicinity of patients may play a key role for the transfer of nosocomial pathogens.

### 2.4 Mechanisms of Transmission from Inanimate Surfaces to Susceptible Patients and Consequences Thereof

The main route of transmission of HAI is via transiently contaminated hands of healthcare workers, but contaminated surfaces may serve as important vectors for cross transmission after hand contact as well (Fig. 2.1).

A single hand contact with a contaminated surface results in a variable degree of pathogen transfer. Transmission from surfaces to hands was most successful with *E. coli*, *Salmonella* spp., *S. aureus* (all 100 %), *C. albicans* (90 %), Rhinovirus (61 %), Hepatitis A virus (22–33 %), and Rotavirus (16 %) [64, 65]. Other transfer rates were calculated for Echovirus, Poliovirus, and Rotavirus with 50 % transmissibility, and for *Salmonella enteritidis*, *Shigella* spp., and *E. coli* O157:H7 with 33 % [104]. Contaminated hands can transfer viruses to 5 more surfaces or 14 other subjects. Contaminated hands can also be the source of re-contamination of the surface, as demonstrated with Hepatitis A virus [64, 65].

Because of this, it is critical to note that healthcare workers’ compliance with hand hygiene varies between 13 % and 94 % with a median of less than 50 % [91]. Moreover hand hygiene is performed less frequently after contact with the environment than with the patient [94]. Both facts underline the necessity to perform additional surface decontamination procedures to interrupt the transmission of nosocomial pathogens. Due to the overwhelming evidence of low compliance of hand disinfection, the risk from contaminated surfaces cannot be overlooked and must not be downplayed by hospital administrations.

![Fig. 2.1 Transmission routes for nosocomial pathogens](image-url)
During outbreaks, the role of the patients’ environment is particularly evident, as suggested by observed evidence for *Acinetobacter baumannii*, *C. difficile*, MRSA, *P. aeruginosa*, VRE, Adenovirus, SARS virus, Rotavirus, and Norovirus [64, 65, 54, 55, 99, 9, 123, 83, 58]. The role of contaminated surfaces is also underlined by the observation that after environmental disinfection, significant decrease of transmissions and HAI have been shown, i.e. for *C. difficile* [73, 126], for VRE [50], for MRSA [32], for multidrug-resistant *A. baumanii* [84], for *S. marcescens* [3], and for other multidrug-resistant Gram-negative rods [86].

If performed correctly, also the burden of microbial airborne transmission can be significantly decreased by surface disinfection. This again may have an impact on healthcare organisations, resulting in i.e. higher clean room class of drug manufacturing areas [8] by elimination of critical bacterial and fungal contamination [63]. As consequence for the successful interruption of cross contamination and infections a multi-barrier approach is required with the key points of hand hygiene and surface disinfection, appropriate used of antisepsis, barrier nursing, and safe reprocessing of contaminated medical devices. Within such multi-barrier strategy, environmental disinfection policies should be based on risk assessments for surfaces with different risks for cross contamination such as high- and low-touched surfaces with appropriate standards for adequate disinfection measures. Generally, surface disinfection is indicated in the following situations:

- Frequently touched surfaces adjacent to patients
- Surfaces with assumed or visible contamination
- Terminal disinfection in rooms or areas where infected or colonized patients with easily transferable nosocomial pathogens are cared for, and
- in outbreak situations.

The purpose of preventive or targeted disinfection on inanimate surfaces is the killing or irreversible inactivation of pathogens to an extent which prevents subsequent infection transmission [41]. In order to ensure the success of environmental disinfection, education, training [52], and targeted microbiological control are important measures and have been shown to improve both, cleaning performance and infection prevention [50]. Increasingly, novel technologies are introduced, which may be used additionally to cleaning. Such technologies may include antimicrobial surfaces on basis of different antimicrobial compounds and are provided for hospital door handles, alarm knobs, curtains, and other objects with high frequencies of hand contact. However, such technologies must be used appropriately and as an adjunct measure to meaningful cleaning and disinfection processes.

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