How long do bacteria, fungi, protozoa, and viruses retain their replication capacity on inanimate surfaces? A systematic review examining microbiological environmental resilience and healthcare-associated infection (HAI) risk by "fomite-borne risk assessment"

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Table of Contents

Summary

In healthcare settings, contaminated surfaces play an important role in transmission of nosocomial pathogens potentially resulting in healthcare-associated infections (HAI). Pathogens can be transmitted directly from frequent hand-touch surfaces close to patients or indirectly by staff and visitors. HAI risk depends on exposition option, extent of contamination, infectious dose (ID), virulence, hygiene practices and patient vulnerability. This review attempts to close a gap in previous reviews on persistence/ tenacity by only including articles (n=179) providing quantitative data on re-cultivable pathogens from fomites for a better translation into clinical settings. We have therefore introduced the new term "replication capacity" (RC). The RC is affected by degree of contamination, surface material, temperature, relative humidity, protein load, organic soil, UV-light exposure (sun) and pH-value. In general, investigations into surface RC are mainly performed *in vitro* using reference strains with high inocula. *In vitro* data from studies on 13 Gram-positive, 25 Gram-negative bacteria, 18 fungi, 4 protozoa and 36 viruses spp. should be regarded as worst case scenario indicating upper bounds of risks when using such data for clinical decision making.

Information on RC after surface contamination could be seen as an opportunity to choose the most appropriate infection prevention and control (IPC) strategies. To help with decision-making, pathogens characterized by an increased nosocomial risk for transmission from inanimate surfaces (fomite-borne) are presented and discussed in this systematic review. Thus, the review offers a theoretical basis to support local risk assessments and IPC recommendations.

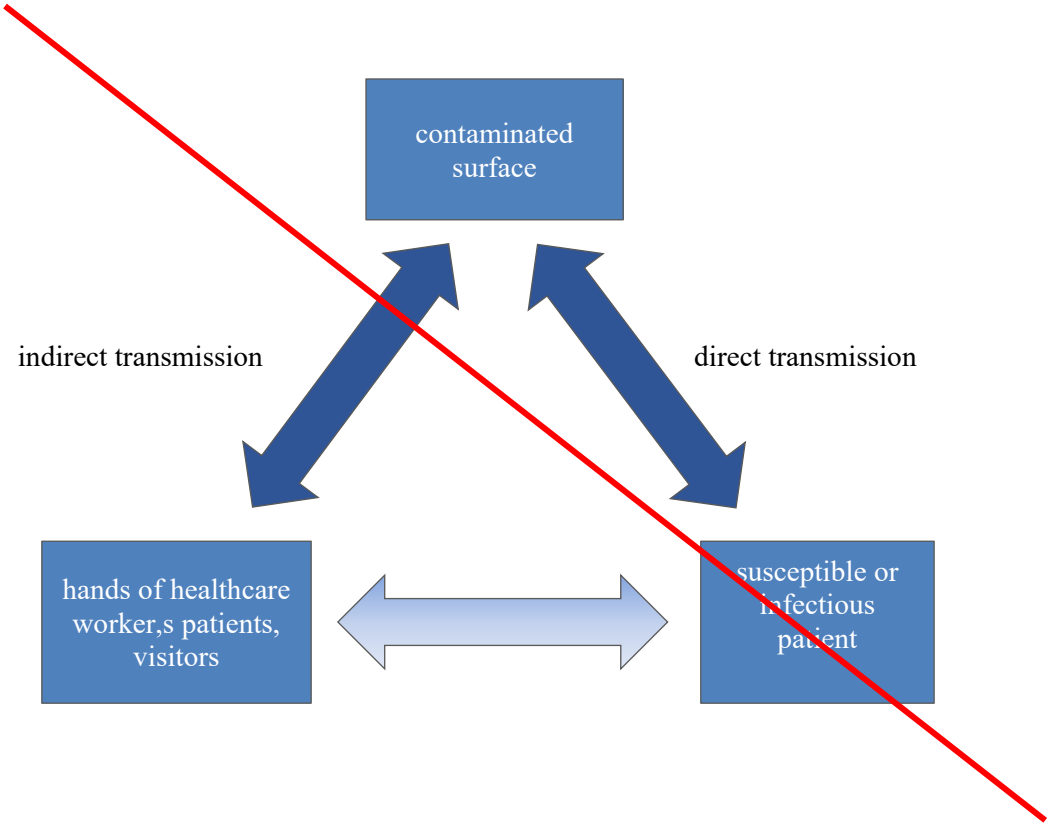
Keywords: replication capacity, persistence, tenacity, viability, resilience, transmission, bacteria, fungi, protozoa, viruses, inanimate surfaces, fomites, fomite-borne risk pathogens, HAI

Introduction

Information about pathogen replication capacity (RC) after surface contamination is an important basis for infection prevention and control (IPC) including the risk assessment of healthcare-associated infections (HAI) and nosocomial outbreaks. In addition, this information is of high importance for outpatient settings and community outbreaks.

Pathogens can be spread from contaminated surfaces by direct patient contact, airborne dispersal (small and large aerosols) or indirectly via hand and medical devices after contamination from hand-touch surfaces (Fig. 1). Exogenous transmission of HAIs in Europe corresponds to only about 5-20 % of the total number of HAI incidents (1), making the hand the main vector for pathogen transmission from contaminated inanimate surfaces (2-31). Consequently, international guidelines assign a key role of cleaning / disinfection of areas beside patients, especially surfaces receiving frequent hand / skin contact (32-35). An additional benefit is the relatively low cost of interventions aiming at controlling this source as opposed to many others, e.g. impregnated catheters (36). However, as recently witnessed during the SARS-CoV-2 pandemic, the role of decontamination of inanimate surfaces can also be overrated (37). Inappropriate use of disinfectants leads to costly interventions alongside risk of disinfectant tolerance and even antibiotic resistance, environmental pollution (38-40) and adverse effects for humans (41-44). Therefore, it would be useful to obtain greater insight on the RC of pathogens on inanimate surfaces in order to implement the most appropriate, risk assessed decontamination procedures.

Since hands are the main vehicle for potential nosocomial pathogens, hand hygiene and surface cleaning should complement each other to prevent HAI (45).


contaminated surface
indirect transmission
direct transmission
hands of healthcare worker,s patients, visitors
susceptible or infectious patient

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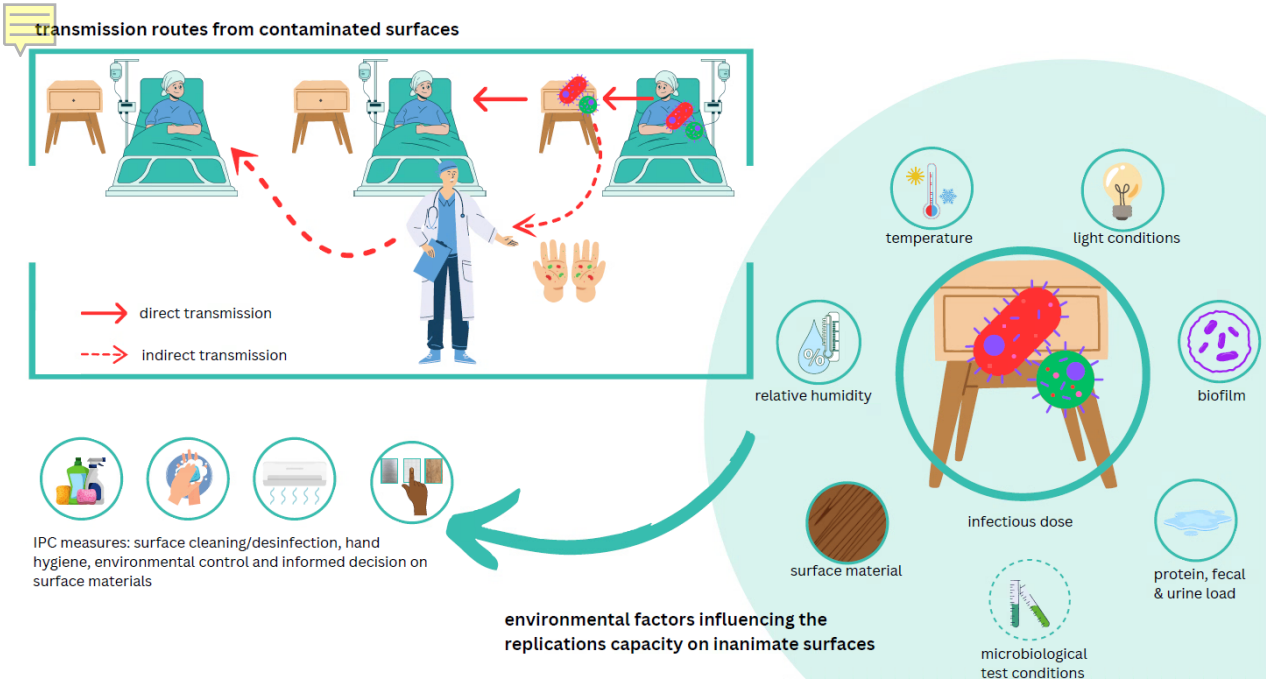
transmission routes from contaminated surfaces
direct transmission
indirect transmission
IPC measures: surface cleaning/desinfection, hand hygiene, environmental control and informed decision on surface materials
temperature
light conditions
relative humidity
biofilm
infectious dose
surface material
protein, fecal & urine load
microbiological test conditions
environmental factors influencing the replications capacity on inanimate surfaces

94

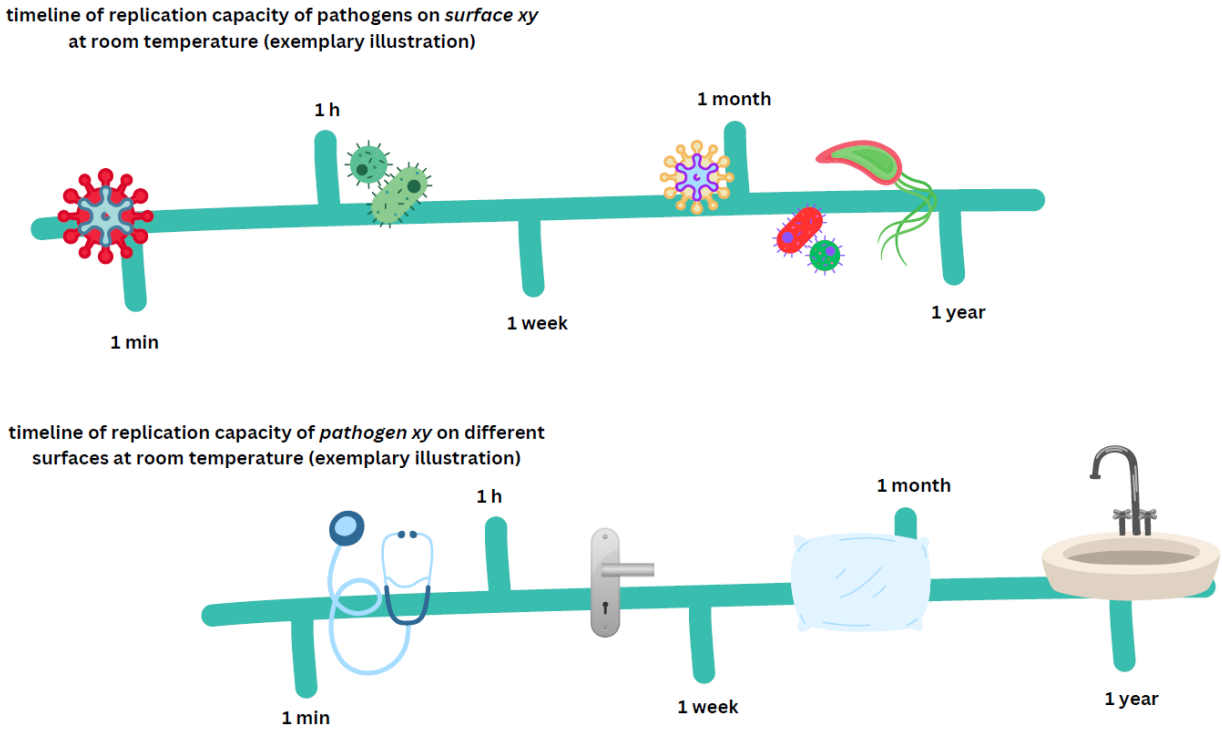

FIGURE 1 Transmission routes from contaminated inanimate surfaces and environmental influences

Defining terms of cultivable pathogens from inanimate surfaces

Resilience is to withstand or to recover quickly from difficulties and therefore being able to keep or come back to the standard or previous condition. Resilience is a positive characteristic from the perspective of the microbes, which in the medical context can have negative implications from the patient's perspective.

To determine the environmental resilience of pathogens, different methods of recovery are available to describe their burden on inanimate surfaces. For viruses only indirect cultivation is possible because cells are needed for replication. Unfortunately, (reverse transcriptase) polymerase chain reaction ((RT-)PCR) does not allow a conclusion to be drawn about remaining infectivity of viruses (e.g. plaque forming units (PFU)). Pathogen dependent, different terms with different meanings are used for the ability of pathogens to be recovered from inanimate surfaces . In order to have the same understanding, some common terms will be preceded by a brief explanation. Von Sprockhoff (46) proposed 'survivability' synonymously to 'tenacity' as the robustness of microorganisms to defined exogenous factors. The term 'tenacity' refers to the resistance of bacteria, fungi, protozoa and viruses to

environmental influences. In the Anglo-American language, the term ‘tenacity’ is uncommon; instead, terms such as ‘resistance’, ‘sensitivity’ or ‘survival’ are used more often (47). The Latin origin ‘tenacitas = to hold on’ is not helpful for understanding what the term means. In the broader sense, tenacity means, ‘the determination to continue what you are doing’ (48). Another comprehensive definition is, ‘the quality or state of being tenacious’ (49). Professionals in clinical disciplines are unaware of the term ‘tenacity’ for microorganisms. Therefore, we need something that linguistically expresses the viability of bacteria, fungi, protozoa and viruses when they contaminate surfaces, in order to be able to assess the risk of onward spread of nosocomial pathogens emanating from that surface.

Since bacteria, fungi and protozoa function autonomously, the terms ‘persistence’, i.e. ‘viability’, or ‘survival’ are used synonymously. Survival can be understood as persisting viability under disadvantageous circumstances (50). Some microorganisms persist through an adaptive reaction to survive in the environment by reducing metabolism and by morphological, biochemical and / or genetic adaptations, especially for bacteria in biofilms and/ or as bacterial spores (51-53). Another mode of adaptation is the transition to viable but non-cultivable (VBNC) cells, which can only be converted back to a replicative, virulent state through certain stimuli (54, 55). Protozoan cysts act as a survival niche and protective shelter (56). The criterion for determining the persistence of microorganisms is whether it can replicate after it has contaminated a surface.

Unlike bacteria, viruses need the synthetic apparatus of intact host cells for their replication. Viruses have neither their own metabolism and energy production nor the possibility of protein synthesis. Therefore, strictly speaking, they are not living beings. The criterion for viral infectivity is the ability to replicate in host cells so that quantification *in vitro* is possible by resuspension from the surface, transfer to the cell culture and counting dead cells, the so-called cytopathic effect. Not every virus is capable of inducing CPE, while demonstrating other significant features. The viral ability to replicate is referred as ‘replication capacity’ (57), which is used in different contexts, e.g. for change under antiviral therapy (58). In parallel, the ability of vectors to transfer antibiotic resistance genes can also be termed ‘replication capacity’ (59). Viral persistence, on the other hand, is understood as the genetic information of viruses presenting in cells of the host organism and the possibility of a virus reactivation under certain circumstances, e.g. in the case of immunosuppression of the host (e.g. herpes viruses).

In summary, only RC reflects the viral load on a surface, because viral RC correlates with the viral infectivity (60). Given that for microorganisms and protozoa, as well as viruses, the criterion of replication determines infectivity and because the term 'replication capacity' does not allow different interpretations, the term 'replication capacity' (instead of tenacity, persistence, survival or viability) is proposed to describe recovery from inanimate surfaces.

Risk assessment from inanimate surfaces as origin of HAI

Information on RC of pathogens on inanimate surfaces could assist with the following aims:

- To determine the most effective decontamination strategy, firstly, for known nosocomial pathogens, and secondly, in the event of the emergence of a new pathogen with initially unknown properties and potential for epi- or pandemic spread;
- Generally, to provide a risk assessment for IPC measures after pathogen release from patients to interrupt further transmission;
- To provide a risk assessment of the need for final disinfection measures required after hospital discharge of pathogen carriers, especially for isolated patients;
- To inform control methods for nosocomial outbreaks;
- To help determine standard operating procedures (SOP) for surface cleaning and / or disinfection, especially hand-touch sites without any knowledge about the presence of potential pathogens;
- To help determine SOP for surface cleaning and / or disinfection, following incidents such as sewage or floodwater spillage, building works, etc.;
- To assess the risk of the possibility of further spread of pathogens after hand contact of contaminated surfaces and medical devices especially for research purposes;
- To assess the risk-benefit between disinfection efficacy, expense and environmental impact and thus finally IPC;
- To analyze the RC under influence of probiotic cleaning as new option for ~~IPC~~.

Walther and Ewald (61) distinguished a highly virulent long-lasting group containing variola (smallpox) virus, *Mycobacterium tuberculosis*, *Corynebacterium diphtheriae, Bordetella (B.) pertussis,*

166 *Streptococcus (Str.) pneumoniae*, and (avian) Influenza A Virus (virulence determined from mortality
167 rate or case mortality). These pathogens have a mean percent mortality ≥ 0.01 % and a mean survival
168 time > 10 days (d). In contrast, a low-virulence and low-persistent group (mean percent mortality <
169 0.01 % and time of survival < 5 d) includes viruses such as Rubeola, Mumps, Parainfluenza, Respiratory
170 syncytial, Varicella-zoster, Rubella and Rhinovirus, alongside the bacteria *Mycoplasma pneumoniae*
171 and *Haemophilus (H.) influenzae*. This is even more interesting, since these bacteria and viruses belong
172 to totally different species, families and genera, respectively. While our review focuses on transmission
173 modes via inanimate surfaces (fomite-borne), another category of pathogens is relevant for risk
174 assessment (see Fig. 1). The longer a nosocomial pathogen persists on a surface, the longer the surface
175 may be a source of transmission and endanger a susceptible patient or healthcare worker. Furthermore,
176 a correlation between virulence and persistence is reported (62), the sit-and-wait hypothesis predicts that
177 virulence should be positively correlated with persistence in the external environment because
178 persistence reduces the dependence on host mobility for transfer to a patient. This has been confirmed
179 for respiratory tract pathogens (62). The pathogenicity including factors as infectious dose (ID), RC and
180 risk of transmission determines the outbreak potential of a pathogen and must be considered as basis for
181 the IPC strategy. For surfaces as (temporary) origin of HAI, the RC of pathogens from fomites is
182 essential. The main focus in this context was the transmission mode from inanimate surfaces. High
183 virulent pathogens with outbreak potential due to low ID, long-lasting RC require additional to the non-
184 targeted near-patient (high-touch) surface disinfection, a targeted cleaning and disinfection as patient-
185 remote (low-touch) surface disinfection and final surface disinfection. Such pathogens with increased
186 "fomite-borne risk", characterized by an increased nosocomial risk for transmission from inanimate
187 surfaces, are marked in blue in the tables 3-7. Of course, disinfection measures are only one part of the
188 IPC strategy combined with the other standard precaution such as hand hygiene and additional pathogen-
189 related measures such as barrier nursing, isolation, antimicrobial chemotherapy and antiseptic
190 decolonization. With growing knowledge, the classification of "pathogens with nosocomial risk for
191 spread from inanimate surfaces" can be further developed.
192 There is a practical way of looking at this. For example, admission to a room previously occupied by a
193 patient infected and/ or colonised with a pathogen is a known risk factor for acquisition of that pathogen

(63). This risk can be quantitated and it appears that the relative differences in acquisition risk between the pathogens mirror environmental longevities. As expected, organisms such as *Acinetobacter baumannii* complex and *C. difficile* present the highest risk for acquisition, and they also happen to be the most resilient in the healthcare environment (64). This begs the question even over the need for cleaning / disinfection priorities for a recently vacated room, depending on which pathogen infected the previous patient. So, in accordance with survival and replicative properties, decontamination strategies could range from a quick wipe over the hand-touch surfaces for MRSA, disinfection of the sink / shower for ESBLs and comprehensive air and surface disinfection for *C. difficile*, etc. If pathogens released from the respiratory tract, knowledge of the RC makes it possible to assess whether surfaces outside the patient's contact area should also be included in the final disinfection, e.g. wall surfaces and slatted curtains. A focus on targeted cleaning and disinfection allows pathogen-related risk to dictate the most appropriate decontamination practice for all patient spaces (45). This risk assessment is the logical consequence of a basic risk without knowledge of existing pathogens and enables a - in theory - most effective strategy.

To assess the timeline of RC for risk of further spread, it is necessary to consider RC in more detail. This includes baseline inoculum, the surface material, temperature, relative humidity (RH), protein load, organic soil, light exposure, and pH-value. Thus, it is not just the type of pathogen or evidence for them (e.g. DNA, RNA), but whether they are capable of being transmitted to, and replicating in, the host (Fig.1). Transmission potential of pathogens on surfaces is not restricted to the direct and indirect contact transmission route, as illustrated in Fig. 1. Some, but not all potential pathogens on inanimate surfaces can be aerosolized and transmitted contact-free. This potential additional risk is not within the scope of this review. But, if the RC is known, the infection risk can be estimated for respiratory released and aerogenic transmissible pathogens.

The aim of this review was to collect and assess published data related to RC of all types of nosocomial pathogens contaminating inanimate healthcare surfaces as basis for evaluating healthcare-associated infection risk by fomite-borne risk assessment. For determination of IPC strategies both RC and ID should be considered. This data might assist by evaluating the transmission and infection risk and therefore guide most appropriate IPC measures.

Method

The basis of this review made use of three reviews (65-67) with at least partly similar aims, from which literature was screened and adopted. In order to update and expand the current reviews, afterwards a systematic literature search was conducted and reported in accordance with the PRISMA guideline and the German Manual for literature research in databases (68).

Based on the modified PICO scheme (table 1) the search strings were compiled. The search was restricted to publications from 2020 onwards to obtain only hits that were not already included in the search of the latest included review (67). The language was limited to German and English. The databases PubMed and Web of Science were searched due their medical focus. The search was conducted on the 26th of January 2023.

TABLE 1 Search strategy; segments and search terms

Segment	Search terms
Pathogens	Bacteria, virus, fungi, protozoa
Conditions	Surface, fomite, inanimate, temperature, humidity, light
Setting	Nosocomial, hospital-acquired
Outcome	Persistence, survival, transmission, tenacity

Duplicates were removed using *Citavi 6* (Swiss Academic Software GmbH). Four reviewers carried out the screenings blinded (two reviewers per article) using an online document to record the decisions. The articles were compared against predetermined inclusion and exclusion criteria (table 2).

In case of different assessments, a third reviewer joined the discussion, and a consensus was reached. Firstly, the titles and abstracts were screened and then the full texts of the included records. Eligible reviews were not included but searched for primary studies, which were then also screened as described above.

TABLE 2 Inclusion and exclusion criteria

Inclusion	Exclusion

Narrative review, rapid review, scoping review, systematic review, randomized controlled trial, quasi randomized controlled intervention study, not randomized controlled studies, pro- and retrospective cohort studies, case control studies, historically controlled studies, cross-sectional studies	Single-arm follow-up studies (case reports, case studies, ...), commentaries, study protocols, conference abstracts, books, editorials, model studies
Human pathogenic species within the following groups: viruses, bacteria, protozoa, fungi that are relevant for hospital acquired infections from surfaces[1]	Other pathogens
Inanimate surfaces – specifically surfaces relevant in hospital settings. Cave: if the only information found was not on hospital relevant surfaces, the information is reported to give insight into possible tenacity of the pathogen.	Animate surfaces
Persistence, tenacity, survival, temerity, recultivable, replicable; a resuspension has to be made from the test surface and then transferred to the cell culture or nutrient medium	Anything concerning the treatment, symptoms, or genetic surveillance; studies on the effect of disinfectants; studies on the effect of antibacterial / antiviral surfaces
Since 2020	Before 2020
English, German	Other languages
	Relevant data / methodology (e. g. inocolum concentration) not given
[1]Although ectoparasites can also be transmitted nosocomial (69), they were excluded because they are multicellular arthropods reproducing outside the human organism.	

The data was extracted into an online table by the reviewers. A cross check was conducted afterwards.

Tables 3-7 were completely modified from the informative appendix (only in German) (70) of the recommendation of the Commission for Hospital Hygiene and Infection Prevention (KRINKO) on Hygiene requirements for cleaning and disinfection of surfaces (71). Table 8 was modified from Jawad et al. (72).

Evaluable publications: Out of the three reviews this review is based on, 145 publications were included. Additionally, through the systematic search 495 records were identified via the databases (Fig. 2). 152 duplicates were removed. The title and abstract of the remaining 343 records were screened leading to the inclusion of 40 reports. 32 of these were excluded during the full text screening. Four

primary studies and four reviews were included. The reference lists of the reviews were screened for other eligible studies which lead to the inclusion of another 22 primary studies. Within the scope of the systematic search, a total of 26 primary studies were included. Adding the studies from the three initial reviews, a total of 171 publications were included.

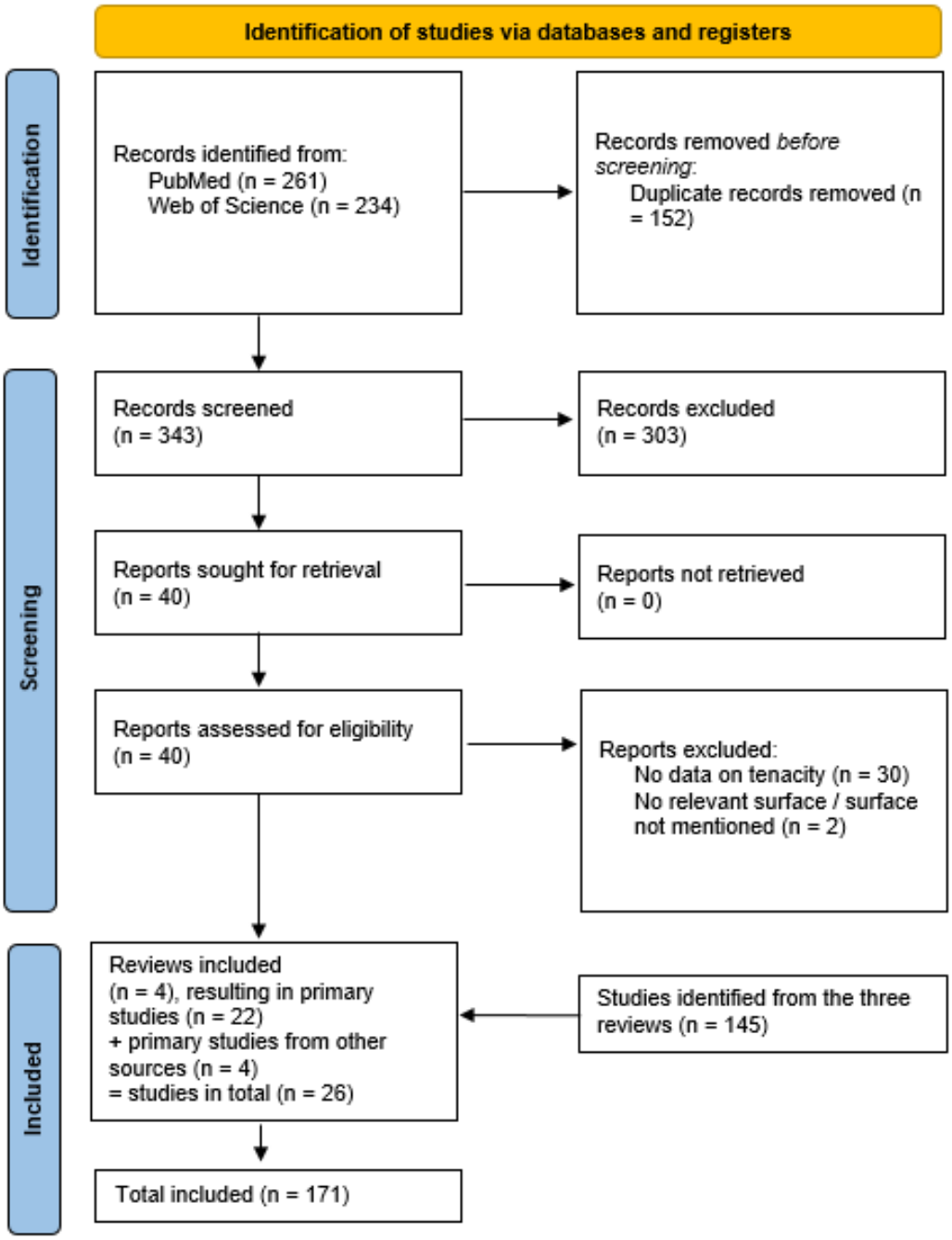

FIGURE 2 Flow chart (modified from (73))

Our review does not claim to completeness all pathogens with ability to induce outbreaks; such as *Mycobacterium chimera*. The priority for us was to observe the transmission possibility from hand-touch inanimate surfaces; this is why we did not consider pathogens dominating in other hospital hygiene relevant settings (e.g. water , air and food).

The tables 3-7 focus on the most important pathogens and relevant environmental (temperature, RH, light, surfaces) data for clinical settings. For better clarity, inocula were reported by waiving application conditions. Due to the inconsistencies in the kind of units used to report results, the initial inoculum (starting point) was converted into decadic logarithm. For additional data and details of recultivation and expanded environmental conditions, see supplementary material. Pathogens with an increased fomite-borne transmission potential were highlighted in blue. For this tentatively introduced classification we used a simple scoring system: Pathogens are characterized with **firstly)** a high virulence and / or **secondly)** a long RC and / or **thirdly)** a high potential for nosocomial spread. A pathogen belongs to the fomite-borne risk group if at least two of the three statements are fulfilled. This is to be understood explicitly as a basis for discussion and is summarized illustratively in the figure.

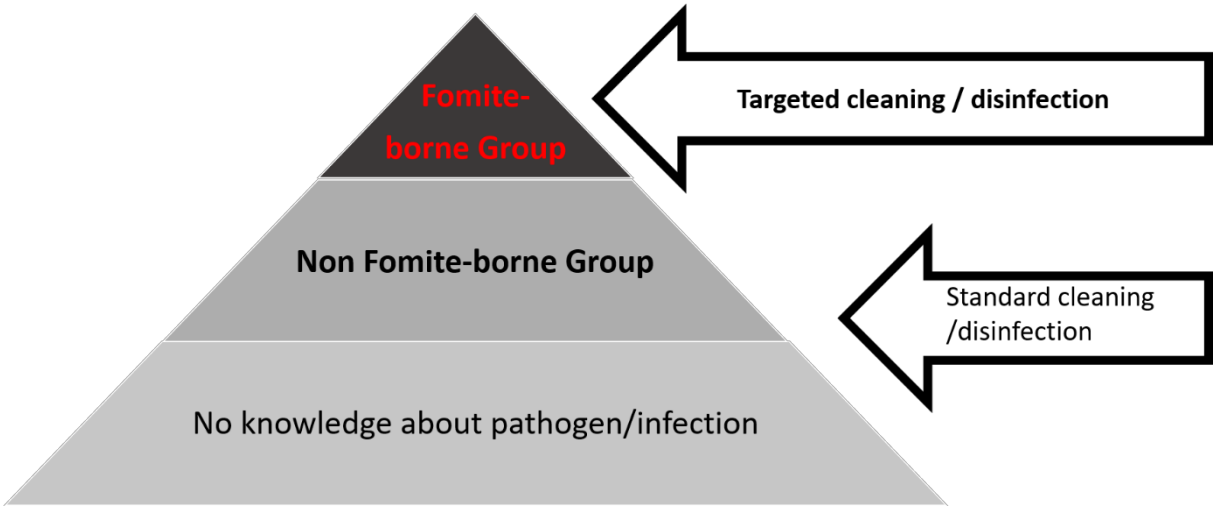

FIGURE 3 Introduced classification of pathogens with fomite-borne transmission potential and derived IPC strategies

Replication capacity of bacteria

Especially in the near-patient environment of microbial colonized or infected patients, the responsible species underlying the colonization or infection can be detected, especially if no surface cleaning or disinfection has been carried out. In order to clarify transmission routes, such detection has been carried out primarily for resistant species such as Methicillin-resistant *Staphylococcus (S.) aureus* (MRSA) (74, 75), vancomycin-resistant enterococci (VRE) (74, 76), carbapenem-resistant enterobacterales (CRE) (77, 78), *Acinetobacter baumannii* complex (79), *Clostridioides (C.) difficile* (79, 80) and recently for the high pathogenic yeast Candida *(C.) auris*. For species regularly detected in nosocomial outbreaks or which frequently colonize or subsequently infect subsequently admitted patients after patient discharge, the knowledge of RC is of special interest, because intensified surface cleaning with disinfection as part of an intervention bundle proved effective in controlling transmissions and even an outbreak. This has been proven for nosocomial outbreaks by VRE (18, 25), *C. difficile* (16), MRSA (81), *Acinetobacter (A.) baumannii* (4, 8, 22, 28), CRE (14, 25) and C. auris (Ahmad et al. 2023). The acquisition of pathogens from the discharged patients caused by deficiencies in final disinfection is repeatedly described (5-7, 9, 15, 23, 82) and evaluated in meta-analyses (21, 31). However, none of these studies have shown by genomic surveillance that the previous and the new patient were colonized with the same clone of the respective species. Recent work suggests, that clonality cannot be assumed, but there is a high likelihood of clonality depending on species (87).

In most reports, RC 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 and removed from the germ carrier by different rinsing solution e.g. dist. water, physiol. NaCl, phosphate buffered salt solution (PBS), or Triton X-100, sometimes in combination with ultrasound (table 3 and 4).

After this preparation, members of the Gram-positive genera enterococcus (e.g. VRE) and staphylococcus (e.g. MRSA) survive for months on dry surfaces. Among streptococci, RC differs depending on the species, i.e. for *Strepotococccus (Str.) pneumoniae* < 24 h, *Str. pyogenes* 1-3 d and *Str. salivarius* > 88 h. In addition, *Corynebacterium pseudotuberculosis* survives 1-4 d on dry plastic surfaces. In contrast, *C. diphtheriae*, isolated from dust in patient rooms, survives 7-90 d, depending on

303 species. By daylight *Mycobacterium tuberculosis* survives for 2-5 d. In darkness the recultivation is
304 possible up to 200 d (table 3).
305 There are only a few studies in which wildtype and antibiotic resistant representatives of the same
306 species were compared with each other. For enterococcus there are hints of higher RC for VRE
307 compared with sensible enterococci present. Moreover, in dust a Methicillin-sensitive *S. aureus* (MSSA)
308 demonstrated a shorter survival time on surfaces than MRSA (table 3).
309 Spores of *Bacillus* und *Clostridioides (C.)* spp. survive depending on the material > 6 mon. In contrast,
310 the vegetative form of *C. difficile* drops to the detectable threshold within 15 minutes (min) (table 3).
311

312 **TABLE 3** Replication capacity of Gram-positive bacteria from inanimate surfaces modified from (70) (pathogens with "fomite-borne risk", characterized by an
313 increased nosocomial risk for transmission from inanimate surfaces, are marked in blue; for additional data and details of recultivation and environmental
314 conditions, see supplementary material)

Pathogen	Initial inoculum	Replication capacity	Surface	Reference
Bacillus subtilis spores	~ 8 lg CFU	After 15 d reduction by ~ 0.3 lg, after 56 d: reduction by ~ 0.7 lg	Glass	(84)
	7.1-9.5 lg CFU	> 200 d: reduction by ~ 2 lg	Polycarbonate	(85)
	6 lg CFU	≥ 1 d: 5 lg	Stainless steel	(86)
Clostridioides (C.) difficile spores	6 lg CFU	After 2 d: reduction by ~ 2 lg, after 4 wk: 8 CFU, after 5 mon: 1 CFU	Floor	(87)
	6-7 lg CFU	After 6 wk: reduction by ~ 0.5 - 0.8 lg; after 12 wk: reduction by < 3 lg	Steel	(88)
C. difficile veg.	~ 6 lg CFU	15 min: reduction by ~ 4 lg	Glass	(89)
Corynebacteria generic	2.7-3.8 lg CFU	≥ 48 h: mean recovery 3.6 %	Cotton	(90)
Corynebacterium diphtheriae	up to 155 CFU	7-90 d (strain-dependend)	Dust	(91)
Corynebacterium pseudo-tuberculosis	~ 6 lg CFU	3 d	Plastic	(92)
Corynebacterium striatum	6 lg CFU	After 48 h: 7.7 lg / 6.8 lg / 2.6 lg	Polyvinyl chloride (PVC)/ silicone / stainless steel	(93)
Enterococcus faecium	6-7 lg CFU	After 12 wk: reduction by < 3 lg	Steel	(88)
	~ 6.5 lg CFU	49 d / 51 d / 49 d	Cotton / wool / silk	(94)
	250 CFU	7 d up to 28 d: 250 - 70 CFU / 250 - ~ 32 CFU / 250 - 160 CFU / 250 - ~ 50 CFU	Glass / PVC / stainless steel / aluminum	(95)
	8 lg CFU	1 to 16 wk	PVC	(96)
	8 lg CFU	< 4 mon: ~ 2 lg recultivable	Ceramic / PVC / rubber / steel	(97)
	~ 5 lg CFU	33 / > 90 / > 90 d	Cotton / polyester / polypropylene	(98)

	5-6 lg CFU	≥ 7 d (3 lg / 3 lg)	Polyester / Terrycloth	(99)
	10 lg CFU	≥ 21 d (4-5 lg)	Cotton	(100)
Enterococcus faecalis	6-7 lg CFU	After 6 wk: reduction by < 1.8 lg	Steel	(88)
	7.5 lg CFU	After 8 wk: 6.5 lg	Ceramic / cotton / synthetic fibers	(101)
	5.2 lg CFU	After 1 d: survival of 3 %	Cotton	(102)
	~ 5 lg CFU	> 90 / > 90 / > 90 d	Cotton / polyester / polypropylene	(98)
	6 lg CFU	≥ 1 d: 5 lg	Stainless steel	(86)
Enterococcus spp.	7.2 lg CFU	Mean survival rate 3 d (dried in water), 43 d (dried in egg white)	Glass	(72)
Vancomycin resistant Enterococcus (VRE)	~ 6 lg CFU	After 6 wk: reduction by ~ 3 lg	Steel	(88)
	5 lg CFU	≥ 7 d	Furnishings	(103)
	E. faecalis 4.5 lg	Dried 60 min: 3 lg CFU; dried 90 min: 3.6lg CFU	Stainless steel	(104)
	8 lg CFU	1 to 16 wk	PVC	(105)
	E. faecalis: ~ 5 lg CFU	22 / > 80 / > 80 d	Cotton / polyester / polypropylene	(98)
	E. faecium: ~ 5 lg CFU	> 90 / > 90 / > 90 d		
Micrococcus luteus	7.1-9.5 lg CFU	After 120 d: reduction by ~ 6 lg	Polycarbonate	(85)
	5.2 lg CFU	After 2 d: survival of 20 %	Cotton	(102)
Mycobacterium tuberculosis	0.1 mg / ml	Recultivable in daylight after 1 d, recultivable in darkness for 9 d, not recultivable after 40 d	Coverslip	(106)
Staphylococcus aureus, methicillin-susceptible (MSSA)	7.3 lg CFU	≥ 11 d	Glass	(72)
	5.2 lg CFU	After 25 d: survival of 0.8 %	Cotton	(102)
	7.5 lg CFU	After 8 wk: ~ 6.5 lg CFU / ml	Ceramic / cotton / synthetic fibers	(101)
	8 lg CFU	2 d / 18 d / > 45 d / 43 d	Latex / cotton / vinyl flooring / granite	(107)
	~ 6.5 lg CFU	37 d / 37 d / 41 d /37 d	Cotton / cotton polyester / wool / silk	(94)
	6 lg CFU	9 d / 10 d / 3 d	Formica / stainless steel / enamel	(108)
	250 CFU	After 21 d: 5 CFU/ after 7 d: ~ 5 CFU / after 21 d: 0 CFU / after 7 d:~ 10 CFU	Glass / PVC / stainless steel / aluminium	(95)

	7.2 lg CFU	Mean survival 26 d (dried in water), 35 d (dried in egg white); after 12 d: ~ 3 lg CFU loss (water); after 18 d: ~ 5.7 lg loss (egg white)	Glass	(72)
	Desiccation: 7.3 lg CFU Wet : 3-4 lg CFU	After 25 d desiccation: 4.4 lg Wet: after 7 d not recultivable	Aluminium	(109)
	6-7 lg CFU	Dry < 7 mon, at 32 % RH > 5 mon	Dust	(110)
	a) dry inoculum: 5-6 lg CFU b) liquid inoculum: ~ 6 lg CFU	a) After 24 h: 6.7 lg CFU, after 7 d: 22 CFU /after 24 h: 6.3 lg CFU, after 7 d: 1 CFU b) After 7 d: 16.2 lg / 6.1 lg	Polymer without silver / with silver	(36)
	8 lg CFU	With dust: < 28 d, without dust: < 35 d	Bottles with and without dust	(111)
	7 lg CFU	≥ 12 d / 12 d / ≥ 14 d	Plastic / laminated plastic / polyester	(112)
	5-6 CFU (matress cover) 14-34 CFU (drapes) 5-6 CFU (bed sheets)	Recovery after 72 h at 22 °C: 98 CFU / / 1 CFU / 17 CFU /3 lg / 1 CFU / 1 CFU	Dry mattress cover / wet mattress cover / dry drapes / wet drapes / dry bed sheets / wet bed sheets	(113)
	8 lg CFU	< 21 d / ≥ 21 d (6 lg)	Polyester / cotton	(114)
	5-6 lg CFU	≥ 206 d / 25 d / 11 d / ≥ 206 d	Mattress inner foam / PVC / cotton / polyester	(115)
	9 lg CFU	≥ 21 d: 4-5 lg CFU	Cotton	(100)
	5.7 lg CFU	≥ 11 d: 4 lg CFU	PVC	(86)
	5.7 lg CFU	≥ 11 d: 3 lg CFU / ≥ 11 d: 3 lg CFU / ≥ 11 d: 3 lg CFU	Aluminum / plastic / stainless steel	
	6 lg CFU	≥ 1 d: 6 lg CFU	Stainless steel	
	0.05 OD_{600}	≥ 7 d: survival rate: 4 %	Polypropylene	(116)
Staphylococcus aureus, methicillin-resistant, Epidemic (EMRSA)	8.7 lg CFU	≤ 60 min / 270 min / ≥ 360 min	Copper / brass (80 % Cu, 20 % Zn) / stainless steel	(117)
Staphylococcus aureus,	6-7 lg CFU	After 6 wk: reduction by 5-6 lg CFU	Steel	(88)
	8 lg CFU	1 d / 18 d / 41 d / 40 d	Latex / cotton / vinyl flooring / tile	(107)

methicillin-resistant *(MRSA)*	3.2-4.9 lg CFU	After 7 d: recovery 59-125 %; after 14 d: 26-42 %; after 28 d: 0.2-16 %; after 56 d: 0-1 %	Dry mop	(118)
	9 lg CFU	< 318 d	Plastic	(119)
	8 lg CFU	With dust: < 126 d; without dust: < 175 d	Bottles with and without dust	(111)
	5.6 lg CFU)	< 21 / 14 / 3 / 40 / > 51 d	Cotton / cotton terry / cotton and polyester / polyester / polypropylene	(98)
	~ 7.3 lg CFU	< 96 d	Glass	(120)
	6 lg CFU	≤ 63 d / ≤ 56 d / ≤ 21 d / ≤ 14 d / ≤ 14 d / ≤ 3 d / ≤ 5 min	Vinyl / plastic / ceramic / bed sheets / towels / wood / razors	(121)
	7 lg CFU	≥ 12 d / 11 d / 9 d	Plastic / laminated plastic / polyester	(112)
	6.3-6.7 lg CFU or 4.3-4.7 lg CFU	≤ 8 d or < 2 d	Polypropylene	(122)
	5-6 lg CFU	≥ 7 d: < 1 lg / 1 lg	Polyester / terrycloth (towel)	(99)
Staphylococcus aureus, Vancomycin intermediate (VISA)	8 lg CFU	1 d/ 3 d / > 45 d / > 45 d	Latex / cotton / vinyl flooring / granite	(107)
Streptococcus faecalis	Desiccation: 6.9 lg CFU Wet: 3-4 lg CFU	After 25 d desiccation: 4.6 lg Wet: after 10 d not recultivable	Aluminium	(109)
Streptococcus pyogenes	~ 7.7 lg CFU	< 2 h	Plastic and ceramic / plastic / stainless steel	(123)
	8 lg CFU	planktonic: 3 d; as biofilm: > 120 d	Plastic / textiles	(124)
	5-6 lg CFU	≥ 206 d / 25 d / 11 d / ≥ 206 d	Mattress inner foam / PVC / cotton / polyester	(115)
Streptococcus pneumoniae	2.8-3.6 lg CFU	≥ 48 h: mean recovery 0.2 %	Cotton	(90)
Streptococci, Staphylococci from saliva; combined analysis	5.3 lg CFU for *Staphylococcus aureus*; 5.9 lg CFU for *Streptococcus pyogenes;* 5.8 lg CFU for *Streptococcus salivarius*	> 88 h	Glass / latex / wood	(125)

Legend: CFU = colony forming units, lg = decadic logarithm, min = minute, h = hour, d = day, wk = week, mon = month, PVC = polyvinyl chloride

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Initial comment is that neither Gram-positive nor Gram-negative organisms represent a uniform group regarding recultivation potential from inanimate surface (Tables 3 and 4). Some species can survive for month, such as *Escherichia (E.) coli, Klebsiella* spp.*, Pseudomonas aeruginosa, Serratia marcescens, Enterococci*, *Acinetobacter* ssp. and *Clostridioides* ssp.. This is also reflected in infection epidemiology since these pathogens can cause ongoing transmission incidents and outbreaks. The Salmonella genus behaves very differently: *Salmonella (S.) typhimurium* is still present in garden soil 280 d after contamination (126), *S. paratyphi B* survives in soil up to 259 d (127) and *S. enteritidis* for more than 11 month, whereas *S. typhi* survives only 4 d.

Conversely, Mitscherlich and Marth (128) demonstrate the persistence of *Proteus* spp. in the environment with 1-2 d. *P. morganii, P. rettgeri, P. vulgaris* and *P. mirabilis* survive in sterile clay loam at 18-20°C species-dependant 35-40 d. The decimal reduction time was about 6 d (129). *Shigella flexneri* persists for 6 d (130). *B. pertussis, H. influenzae,* and *Vibrio cholerae* persist only few days ((131); Table 3). Aerosolized *H. influenzae* is characterized by short survival on glass (0.29 d), wood (0.08 d), and fabric (< 1 d) (128, 132).

331 **TABLE 4** Replication capacity of Gram-negative bacteria from inanimate surfaces modified from (70) (pathogens with "fomite-borne risk", characterized by an
332 increased nosocomial risk for transmission from inanimate surfaces, are marked in blue; for additional data and details of recultivation and environmental conditions,
333 see supplementary material)

Pathogen	Initial inoculum	Replication capacity	Surface	Reference
Acinetobacter baumannii	~ 6.5 lg CFU	19 d / 19 d / 7 d / 19 d	Cotton / cotton polyester / wool / silk	(94)
	6-7 lg CFU	After 6 wk: reduction by 4-5 lg	Steel	(88)
	6 lg CFU	11 d / 12 d / 6 d	Formica / stainless steel / enamel	(108)
	250 CFU	After 28 d: ~ 112 CFU / ~ 112 CFU / ~ 18 CFU / ~ 20 CFU	Glass / PVC / stainless steel / aluminum	(95)
	7.1-9.5 lg CFU	After 20 d reduction by about 5.5 lg	Polycarbonate	(85)
	1200 resp. 1100 CFU	Biofilm-forming < 36 d / non-biofilm-forming <15 d	Glass	(133)
	7.3 lg CFU	3 d	Glass	(134)
	7.3 lg CFU	up to 33 d	Glass	(135)
	7.3 lg CFU	7 - 70 d (strain-dependent)	Glass	(136)
	~ 8 lg CFU)	3 - 90 d (strain-dependent)	Polystyrene	(137)
	~ 7.3 lg CFU	< 96 d	Glass	(120)
	8 lg CFU	50 % of strains mean survival at least 2 wk (< 2 lg recultivable), strain-dependent < 4 mon (7 lg recultivable)	Ceramic / PVC / rubber / steel	(97)
	4.1 lg CFU	Dried 60 min: 4 lg; dried 90 min: 3.9 lg	Stainless steel	(104)
	6 lg CFU	≥ 1 d: 4 lg	Stainless steel	(86)
	7 lg CFU	≥ 60 d: survival rate: 10 %, 40 %, 40 %	Cotton / plastic / glass	(138)
	5-6 lg CFU	≥ 7 d: 2 lg / 3 lg	Polyester / Terrycloth	(99)
	7.2 lg CFU	Mean survival rate strain-dependent 2-29 d (dried in water); < 59 d (dried in egg white); after 18 d ~ 5.5 lg loss	Glass	(72)
Acinetobacter johnsonii		Mean survival rate 3 d (dried in water); 12 d (when dried in egg white)		
Acinetobacter junii		Mean survival rate 2 d (dried in water); 13 d (dried in egg white)		
Acinetobacter lwolffi		Mean survival rate 6 d (dried in water); 8 d (dried in egg white)		
	7.3 lg CFU	3 d	Glass	(134)

Acinobacter calcoaceticus anitratus	4 lg CFU	after 1 h: 3 lg	Hardboard	(139)
	5.2 lg CFU	After 25 d survival of 0.6 % of the CFU / after 7 h survival of 40 % of the CFU	Cotton / glass	(102)
Acinetobacter calcoaceticus lwoffii	4 lg CFU / sample	After 1 h: 3 lg CFU	Hardboard	(139)
	5.2 lg CFU	After 7 d not recultivable	Cotton	(102)
Acinetobacter radioresistens	7.3 lg CFU	157 d	Glass	(134)
Bordetella pertussis	8 lg CFU (0.01 ml)	< 0.04 h - 5 d / 3-5 d / < 0.04 h - 5 d / < 0.04-4 d /0.2-1 d	Glass / plastic / rubber / / fabric / / paper	(140)
Campylobacter jejuni	0.1 ml contaminated water from screw coolers	4 h / 4h / 7 h/ 7 h	Aluminum / stainless steel / formica / ceramic	(141)
	8-9 lg CFU	After 28 d: ~ 5 lg (without wood 0 lg after 2 d) / polyurethane and glass: ~ survival for 2 d (pore-size-dependent)	Wood / polyurethane / glass	(142)
	7 lg CFU	≤ 250 min (4 lg) / ≥ 250 min (3 lg) / < 250 min (1 lg) / < 180 min	Stainless steel / formica / ceramic / cotton	(143)
Enterobacter cloacae	250 lg CFU	After 3 d: ~ 14 CFU / after 2 d: ~ 12 CFU / after 3 d: ~ 13 CFU / after 2 d: ~ 5 CFU	Glass / PVC / stainless steel / aluminum	(95)
Escherichia coli	6 lg CFU	After 48 h: ~ 1.5 lg / after 24 h: ~ 1.5 lg	Plastic / carton	(144)
	9 lg CFU	After 100 d: 1 lg	Plastic	(145)
	7.3 lg CFU	After 7 d (dry): not recultivable; after > 28 d humidity	Wood / steel	(146)
	7-8 lg CFU	< 120 min	Plastic / wood	(147)
	5.2 lg CFU	After 7 h: not recultivable/ after 7 h: survival of 0.8 % of CFU	Cotton / glass	(102)
	7.5 lg CFU	After 8 wk: ~ 6.5 lg CFU / ml	Ceramic / cotton / synthetic fibers	(101)
	7-9 lg CFU	After 2 h decrease by: 1.7 lg / 0.37 lg / 1.09 lg / 0.44 lg / after 24 h: 0.06 lg	New dry Wood / new wet wood / used dry wood / used wet wood / plastic	(148)
	8 lg CFU	< 4 mon (~ 2 lg recultivable)	Ceramic / PVC / rubber / steel	(97)
	~ 6.5 lg CFU	45 d / 37 d / 45 d / 45 d	Cotton / cotton-polyester / wool / silk	(94)
	250 CFU	After 1 d: ~ 5 CFU / after 1 d: 2 CFU / after 2 day: 1 CFU / after 2 d: 1 CFU	Glass / PVC / steel / aluminum	(95)

	7.1-9.5 lg CFU	After 6 h: decrease by about 6.5 lg	Polycarbonate	(85)
	7.2 lg CFU	Mean survival rate 1 d (dried in water), 3 d (dried in egg white)	Glass	(72)
	6-7 lg CFU	At 58 % RH > 8 mon	Dust	(110)
	Desiccation: 6.9 lg CFU Wet: 3-4 lg CFU	After 25 d desiccation: 0.7 lg CFU / cm^2 Wet: > 12 d	Aluminum	(109)
	5-6 lg CFU	After 24 h: 0.2 CFU, after 7 d: not recultivable / after 7 d: 8 CFU	Polymer without silver / with silver	(36)
	1-2 CFU (mattress cover) 2 CFU (drapes) 1-2 CFU (bed sheets)	Recovery after 72 h at 22 °C: 4 lg / 4 lg / 3.7 lg / 5.7 lg / 3.2 lg / 4.2 lg	Dry mattress cover / wet mattress cover / dry drapes / wet drapes / dry bed sheets / wet bed sheets	(113)
	8 lg CFU	< 10 d / ≥ 21 d (6 lg)	Polyester / Cotton	(114)
	5-6 lg CFU	≥ 206 d / 11 d / 7 d / ≥ 206 d	Mattress inner foam / PVC / cotton / polyester	(115)
	2.7 - 3.2 lg CFU	≥ 48 h: mean recovery too numerous to count	Cotton	(90)
	5.7 lg CFU	≥ 1 d: 2 lg	Vinyl chloride	(86)
	5.7 lg CFU	≥ 4 d: 1 lg / ≥ 7 d: 1 lg / ≥ 4 d: 1 lg	Aluminum / plastic / stainless steel	
	6 lg CFU	≥ 1 d: 3 lg	Stainless steel	
	5.7 lg CFU	≥ 7 d: 3 lg	Plastic	
Francisella tularensis	~ 8 lg CFU	After 240 h: 4 lg / after 96 h not recultivable	Glass / paper	(149)
Haemophilus influenzae	6 lg CFU	after 1 h: 99.99 % reduction	Aerosol	(150)
	2.8-3.5 lg CFU	≥ 48 h: mean recovery 1.8 %	Cotton	(90)
Helicobacter (H.) pylori	9 lg CFU	After 30 min: 7.8 lg, after 60 min: ~ 1.1 lg / after 30 min: 8 lg, after 60 min: ~ 1.3 lg	plastic / ceramic	(151)
Klebsiella pneumoniae	5.2 lg CFU	After 1 h not recultivable	Cotton	(102)
	7.5 lg CFU	After 8 wk: ~ 6.5 lg CFU / ml	Ceramic / cotton / synthetic fibers	(101)
	~ 6 lg CFU	After 6 wk: ~1 lg	Steel	(88)
	250 lg CFU	After 3 d: ~ 25 CFU / after 3 d: 17 CFU / after 2 d: 21 CFU / after 2d: 13 CFU	Glass / PVC / stainless steel / aluminium	(95)
	7 lg CFU	After 25 d desiccation: 1.8 lg	Aluminium	(109)
	6-7 lg CFU /	At 58 % RH > 15 mon	Dust	(110)

	3.9 lg CFU	Dried 60 min: 3.4 lg; dried 90 min: 1.8 lg	Stainless steel/plastic	(104)
	5-6 lg CFU	< 3 d / < 7 d	Polyester / terrycloth	(99)
Listeria monocytogenes	6 lg CFU	After 48 h: ~ 3.4 lg / ~ 1.2 lg	Plastic / carton	(144)
	7-8 lg CFU	After 180 min: 4 lg	Wood / plastics	(147)
	6 lg CFU	After 10 d: 5 lg / after 5 d: 1.5 lg	Stainless steel / acrylonitrile butadiene rubber (ABK)	(152)
	9 lg CFU	After 50 d: ~7.5 lg CFU; after 50 d (biofilm): ~7.3 lg CFU	Stainless steel	(153)
	8 lg CFU	After 20 d: 2 lg	Stainless steel	(154)
	7.3 lg CFU (biofilm)	After 21 d: 5.3 lg	Stainless steel	(155)
Neisseria gonorrhoeae	2 x ~ 20 µl Patient exudate (with proven infection)	At least until 24 h recultivable	Plastic / cotton-polyester	(156)
	1 drop of positive urethral secretion	Until 17 h: recultivable, after 24 h: not recultivable / until 24 h: recultivable, after 48 h: not recultivable	Glass / textile	(157)
Pseudomonas aeruginosa	a) dry inoculum: 5-6 lg CFU b) liquid inoculum: ~ 6 lg CFU	a) After 7 d: 6.2 lg / 6.2 lg b) After 7 d: 7.8 lg / 7.8 lg	Polymer without silver / with silver	(36)
	8 lg CFU	After 48 h: average < 2 lg	Door handles / chairs / spirometer tubing	(158)
	7.5 lg CFU	After 8 wk: 6.5 lg	Ceramic / cotton / synthetic fibers	(101)
	5.2 lg CFU	After 2 h: not recultivable	Cotton	(102)
	~ 6.5 lg CFU	13 d / 23 d / 33 d	Cotton / cotton polyester / wool / silk	(94)
	250 CFU	after 2 d on all surfaces < 2 lg	Glass / PVC / stainless steel / aluminium	(95)
	6 lg CFU	4 d / 5 d / 1 d	Formica / stainless steel / enamel	(108)
	Desiccation: 6.4 lg CFU Wet: 3-4 lg CFU	After 2 d desiccation: not recultivable; wet: > 12 d	Aluminum	(109)
	6-7 lg CFU	At 58 % RH > 8 mon	Dust	(110)

	1-4 CFU (mattress cover) 2 CFU (drapes) 1 CFU (bed sheets)	Recovery after 72 h at 22 °C: 3.9 lg / 4 lg / 3.5 lg / 5.5 lg / 4 lg / 4.1 lg	Dry mattress cover / wet mattress cover / dry drapes / wet drapes / dry bed sheets / wet bed sheets	(113)
	8.7 lg CFU	20 d, 5 d, 4 d	Cotton	(100)
	6 lg CFU	≥ 1 d: 4 lg	Stainless steel	(86)
	5 lg CFU	≥ 7 d / 24 h / 24 h / 24 h / 24 h / ≥ 7 d / 24 h / 24 h / 24 h / ≥ 7 d / ≥ 7 d / 5 min / 24 h / ≥ 7 d	Paper-backed wallcovering / vinyl composition tile / micro vented perforated vinyl wallcovering / latex paint / vinyl wallcovering, nonwoven backing / linoleum / vinyl sheet goods flooring / rubber tile flooring / synthetic-backed carpet / vinyl-backed carpet / fabric upholstery / polyester and acrylic blend upholstery / vinyl upholstery / 100 % polyester upholstery	(103)
Salmonella enteritidis / enterica	~ 5 lg CFU	After 8 h: 2 lg / not recultivable	Plastic / carton	(144)
	7 lg CFU	< 1680 min / ≥ 1920 min: 1 lg / < 480 min / < 240 min	Stainless steel / formica / ceramic / cotton	(143)
	9 lg CFU	*Salmonella chester* after 100 d: 3 lg; *Salmonella oranienburg* > 200 d	Plastic	(145)
	~ 9.3 lg CFU	> 48 h	Petri dish	(159)
Salmonella typhimurium	5.2 lg CFU	After 7 h: not recultivable	Cotton cloth / glass	(102)
	3.6 lg CFU	< 6 wk	Stainless steel	(160)
	1 µl of overnight cultures inoculated on agar and incubated at 25 °C	ST19: after 1 mon 59.7 ± 12.3 % recultivable; ST313: after 1 mon 13.1 ± 9.6 % recultivable	Plastic	(161)
	2 drops bacterial suspension	Up to 50 mon	Dust	(162)
	5.2 lg CFU	After 1 d: not recultivable	Cotton	(102)
	6 lg CFU	After 3 d: 2 lg / after 1 d: 1.75 lg	Stainless steel / acrylonitrile butadiene rubber	(152)
	6-7 lg CFU	> 30 d: reduction between 3-6 lg	Stainless steel	(163)

	7-8 lg CFU	≥ 28 d: 2-3 lg / ≥ 24 h: 3 lg / ≥ 24 h: 4.5 lg	Tile / wood / carpet	(164)
Serratia liquefaciens	7.2 lg CFU	Mean survival rate 3 d (dried in water), 43 d (dried in egg white)	Glass	(72)
Serratia marcescens	250 lg CFU	After 3 d: ~ 40 CFU / after 3 d: ~ 15 CFU / after 2 d: ~ 1 CFU / after 3 d: ~ 2 CFU	Glass / PVC / stainless steel / aluminum	(95)
	7.2 lg CFU	Mean survival 12 d (dried in water), 9 d (dried in egg white)	Glass	(72)
	Desiccation: 7.3 lg CFU Wet : 3-4 lg	After 25 d desiccation: 2.6 lg; wet: > 12 d	Aluminum	(109)
	5.2 lg CFU	After 1 h: not recultivable	Cotton cloth / glass	(102)
	6 lg CFU	≥ 1 d: 4 lg	Stainless steel	(86)
Shigella dysenteriae	~ 5 lg CFU	After 4 h: not recultivable	Plastic / glass / aluminum / wood / textile	(165)
Shigella sonnei	9 lg CFU	≤ 10 d / ≤ 27 d / ≤ 23 d / ≤ 9 d / ≤ 28 d	Glass / cotton / wood / metal / paper	(166)
	~ 5.7 lg CFU	Survival after 24 h: 100 % / 100 % / 100 %; after 48 h: 75 % / 63 % / 50 %; after 72 h: 13 % / 0 % / 0 %	PVC / polystyrene / sprelacart	(167)
Shigella flexneri		Survival after 24 h: 100 % / 100 % / 83 %; after 48 h: 67 % / 58 % / 33 %; after 72 h: 0 %		
Stenotrophomonas maltophilia	~ 6.5 lg CFU	7 d / 7 d / 7 d	Cotton / cotton-polyester / wool / silk	(94)
Vibrio cholerae	8.2 lg CFU	Normal cultivable status 1 h / 1 h / 1.5 h / 1.5 h / 3.5 h / 4 h / 4 h; VBNC status < 7 d	Aluminum / glass / plastic / steel / iron / paper / textile / wool	(168)
	8.2 lg CFU	4 h: 2 lg / 4 h: 2 lg / 3.5 h: 3.5 lg / 1 h:3 lg / 1.5 h:2.5 lg / 1.5 h: 0.5 lg / 1.5 h: 3 lg / 1 h: 3 lg	Cotton / wood / paper / glass / plastic / stainless steel / iron / aluminum	(169)
Legend: CFU = colony forming units, lg = decadic logarithm, min = minute, h = hour, d = day, wk = week, mon = month, PVC = polyvinyl chloride VBNC = viable but non-culturable				

Replication capacity of fungi

For RC determination, fungi were removed from the germ carrier mostly by dipping or vortex in bouillon or tryptic-soy-broth (TSB), sometimes in combination with ultrasound, and by contact with agar plate, overlaying with agar or smear (Table 5).

Moulds occur ubiquitously in nature, are thermotolerant and can survive on surfaces for 2 d to > 30 d depending on the material (Table 5). Indoor airborne mould measurements underline the survival for several months (170, 171). Moulds can multiply at a RH ≥ 75 % at room temperature (RT), which can lead to mould infestation (172). The species *Cladosporium*, *Aspergillus* and *Penicillium* are the most frequently detected moulds on hospital surfaces (173-175). *Mucor* and *Aspergillus (A.) spp.* were isolated from room air and dust from an air-conditioning system with a defective filter and were linked with mycotic endocarditis in patients undergoing open heart surgery (176). Moreover, Mucorales (*Rhizopus* spp.), recovered from linen were associated with a Mucormycosis outbreak (177, 178), and even survived a certified health care laundry process (178). Other Mucorales (*Mucor* spp.) persisted on various materials for weeks (179).

The dermatophytes *Epidermophyton (E.) floccosum, Trichophyton* (T.) *mentagrophytes* and *Tricholosporum violaceum* survived in skin scales for 10 years at -20 °C, while *T. rubrum* and *T. verrucosum* could no longer be cultivated under the same conditions (180). *Microsporum canis* has been detected on hospital surfaces (175). In Germany in the 1920s *E. floccosum* and *Microsporum (M.) audouinii* dominated as pathogens of human dermatophytoses and *T. rubrum* was almost insignificant; dermatophyte isolates increased from 41.7 % in 1950 to 82.7 % in 1993, so that *T. mentagrophytes var. interdigitale* was gradually replaced by *T. rubrum* as the main pathogen of tinea pedis and onychomycosis. With the introduction of griseofulvin in 1958, both, *M. audouinii* and *T. schoenleinii* were virtually eradicated (181). In the case of tinea pedis, *T. rubrum* was detectable in 86 % of patients, *T. mentagrophytes* in 81 % of patients in house dust (182). Both dermatophyte species could also be detected and cultivated on the bare soles of the feet after leaving public baths. Washing and drying only did not result in complete elimination (183). Since the beginning of the 20th century, the incidence of *Microsporum canis* infections in Europe, especially in Mediterranean countries and Slovenia, has been

increasing sharply, with dogs and cats being the natural reservoir (184). However, further spread is also possible via combs, brushes, hats, furniture, bedding, etc.

Candida (C.) albicans, the most common nosocomial yeast, can survive up to 4 mon on surfaces. RC for *C. glabrata* (*Nakaseomyces glabratus*) was described to be similar but shorter for *C. parapsilosis* (Table 5). In the patient environment, *C. glabrata* (*Nakaseomyces glabratus*), *C. parapsilosis, C. tropicalis, C. albicans, C. metapsilosis and C. lusitaniae* were detected on dry surfaces in ~ 3 %, on moist surfaces in ~ 14 % (185).

Several recent outbreaks have been caused by the new emerging multidrug-resistant *C. auris* (~~186~~) which differs from other yeasts and dermatophytes in nosocomial spread (187, 188). *C. auris* is capable of colonizing patients and it can persist on a patient for over a year (189, 190). It can be transmitted through direct contact, e.g. hands, but also through indirect contact via fomites, such as medical devices, other devices and surfaces that directly contact the patient (188, 191, 192). From 2015 to 2017 an outbreak with 70 patients occurred in a neuroscience intensive care unit of the Oxford University Hospitals, United Kingdom. The outbreak was linked with the use of reusable skin-surface axillary temperature probes, suggesting that *C. auris* persisted in the environment and initiated a large outbreak (193). By now, several outbreaks have been reported from different countries and hospitals reflecting the high relevant transmission capacity of this new pathogen. This is particularly important since this species is highly virulent, reflected by a substantial high proportion of invasive isolates leading to a high blood culture positivity rate in outbreaks. The risk of nosocomial spread through surfaces is represented by a higher RC in *in vitro* settings. Moreover, *C. auris* is often resistant to many antifungals which complements higher risk of colonization and probable outbreak potential, with special regard to pan-resistant strains of *C. auris* (194). *C. auris* is now established in 43 countries across five continents (195).

389 **TABLE 5** Replication capacity of moulds and yeasts from inanimate surfaces modified from (70) (pathogens with "fomite-borne risk", characterized by an increased
390 nosocomial risk for transmission from inanimate surfaces, are marked in blue; for additional data and details of recultivation and environmental conditions, see
391 supplementary material)

Pathogen	Initial inoculum	Replication capacity	Surface	Reference
A. brasiliensis	4 CFU	Recovery after 72 h at 22 °C: 0 CFU / 0 CFU / 0 CFU / 3 CFU / 0 CFU / 2 CFU	Dry mattress cover / wet mattress cover / dry drapes / wet drapes / dry bed sheets / wet bed sheets	(113)
A. flavus	4-5 lg CFU	2 to > 30 d / 2 - 20 d / > 30 d / 8 to > 30 d	Cotton / polyester / polyethylene / polyurethane	(179)
	~ 5.5 lg CFU	After 24 h: ~ 5.4 lg, after 48 h: ~ 5.2 lg, after 5 d: ~ 5.6 lg / after 24 h: ~ 5.3 lg, after 48: h ~ 3.8 lg, after 5 d: 0 lg	Aluminum / copper	(196)
A. fumigatus	4-5 lg CFU	1 to > 30 d / 5 to > 30 d / > 30 d / 5 to > 30 d	Cotton / polyester / polyethylene / polyurethane	(179)
	~ 6.8 lg CFU	After 24 h: ~ 6.3 lg, after 5 d: ~ 6.4 lg, / after 48 h: ~ 6 lg, after 5 d: ~ 1.7 lg	Aluminum / copper	(196)
	~ 6.5 lg CFU	> 30 d / > 30 d / > 30 d / 27 d	Cotton / polyester / wool / silk	(94)
A. niger	4-5 lg CFU	3 to > 30 d / > 30 d / > 30 d / 2 to > 30 d	Cotton / polyester / polyethylene / polyurethane	(179)
	~ 5.3 lg CFU	After 4 d: ~ 5.2 lg, after 24 d: ~ 5.5 lg / after 4 d: ~ 5 lg; after 5 d: ~ 5.1 lg, after 24 d: ~ 5.4 lg	Aluminum / copper	(196)
A. terreus	4-5 lg CFU	2 to > 30 d / 2 to > 30 d / > 30 d / 12 to > 30 d	Cotton / Polyester / Polyethylene / Polyurethane	(179)
C. albicans	4-5 lg CFU	1-3 d / 1 d / 5-6 d / 4-5 d	Cotton / polyester / polyethylene / polyurethane	(179)
	6 lg CFU	< 7 d	Stainless steel (dry) / moist agar without nutrients	(185)
	6 lg CFU	Survival after 2 d: ~ 1 %, after 3 d: ~ 0.2 % / 0.3 %, after 7 d: 0 %	Stainless steel / glass	(197)
	~ 7.5 lg CFU	After 5 d: ~ 6.5 lg / after 6 h: 5 lg, after 24 h: 0 lg	Aluminium / copper	(196)
	6.5 lg CFU	6 d / 6d / 12 d / 12 d	Cotton / polyester / wool / silk	(94)
	~ 6.1 lg CFU	6 d	Glass	(198)
	~ 4.8 lg CFU	48 d	Textile	
	5-6 lg CFU	after 7 d: 6.3 lg / after 7 d: 5.1 lg	Polymer without silver / with silver	(36)

C. auris	6 lg CFU	Survival after 7 d: ~ 38 % / ~ 93 %	Stainless steel (dry) / moist agar without nutrients	(185)
	~ 4.8 lg CFU	After 4 d: ~ 3.5 lg, after 14 d: ~ 0.4 lg	Plastic	(199)
	8 lg CFU	After 14 d: ~ 4.3 lg (biofilm formation)	Plastic	(200)
C. candidum	~ 6.5 lg CFU	21 d / 6 d / 12 d / 6 d	Cotton / polyester / wool / silk	(94)
C. glabrata (*Nakaseomyces glabratus*)	6 lg CFU	Survival after 7 d: ~ 60 % / ~ 90 %	Stainless steel (dry) / moist agar without nutrients	(185)
	~ 4.8 lg CFU	12 d /97 d	Glass / Textile	(198)
	~ 6.5 lg CFU	> 30 d	Cotton / polyester / wool / silk	(94)
C. krusei (*Pichia kudriavzevii*)	4-5 lg CFU	1 d / 8 d / 3-7 d / 4 d	Cotton / polyester / polyethylene / polyurethane	(179)
	~ 6.5 lg CFU	3 d / 6 d / > 30 d / 21 d	Cotton / polyester / wool / silk	(94)
C. parapsilosis	4-5 lg CFU	9-27 d / 27 to > 30 d / > 30 d / > 30 d	Cotton / polyester / polyethylene / polyurethane	(179)
	6 lg CFU	Survival after 14 d: ~ 1.3 % / ~ 4.1%	Stainless steel / glass	(197)
	6 lg CFU	Survival after 7 d: 60 % / 100 %	Stainless steel (dry) / moist agar without nutrients	(185)
	~ 4.7 lg CFU	After 21 d: ~ 2.5 lg, after 28 d: 0.4 lg	Plastic	(199)
	~ 6.5 lg CFU	> 30 d	Cotton / polyester / wool / silk	(94)
	~ 6.1 lg CFU	55 d	Glass	(198)
C. tropicalis	4-5 lg CFU	1-2 d / 1-8 d / 7-18 d / 6-12 d	Cotton / polyester / polyethylene / polyurethane	(179)
	~ 6.6 lg CFU	3 d / 9 d / > 30 d / 21 d	Cotton / polyester / wool / silk	(94)
	~ 6.1 lg	8 d	Glass	(198)
Cryptococcus neoformans	~ 6.5 lg CFU	> 30 d	Cotton/ polyester / wool / silk	(94)
	~ 6.1 lg CFU	27 d	Glass	(198)
Fusarium solani	~ 5.8 lg CFU	After 5 d: ~ 4.4 lg / after 6 h: ~ 3.6 lg, after 24 h: 0 lg	Aluminium / copper	(196)
Mucor spp.	4-5 lg CFU	20-24 d	Cotton / polyester / polyethylene / poly urethane	(179)
Paecilomyces spp.	4-5 lg CFU	< 1 d / 5 d / 4 d / 11 d	Cotton / polyester / polyethylene / polyurethane	(179)
	~ 6.1 lg CFU	40 d	Glass	(198)

Rhodotorula rubra	~ 4.8 lg CFU	205 d	Textile	
Saccharomyces cerevisiae	6 lg CFU	After 48 h: 3.9 lg / 1.5 lg	Plastic / carton	(144)
	1 CFU	Recovery after 72 h at 22 °C: 5 CFU / 2.1 lg / 3.3 lg / 4 lg / 5 CFU 2.9 lg	Dry mattress cover / wet mattress cover / dry trilaminate drapes / wet trilaminate drapes / dry bed sheets / wet bed sheets	(113)
Legend: CFU = colony forming units, lg = decadic logarithm, min = minute, h = hour, d = day, wk = week, mon = month				

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Replication capacity of protozoa

Protozoa are unicellular heterotrophic eukaryotic organisms. They are considered to be a subkingdom of the kingdom Protista, although in the classical system they were placed in the kingdom Animalia (201). The cultivation techniques for protozoa differ from that used for bacteria and fungi, involve highly complex and require different culture parametersdepending on the life cycle stage (). The RC distinguishes between the vegetative form of protozoa, the trophozoite, and the inactive infectious form, the oocyst or cyst (Table 6).

~~Prevention and~ The interruption of infection chains are the main strategies in the field of combating protozoonoses. Depending on habitat, hygienic measures for water and sewage and personal hygiene are of particular importance. Against this background, understanding the RC of protozoa relevant to human medicine is of particular interest.

Giardia (G.) intestinalis is the commonest cause of parasitic diarrhea in high-income countries, the most common enteric protozoan infection in the US, and is also prevalent in middle and low-income countries. Amoebiasis is the third leading cause of death from parasitic diseases worldwide, with greatest impact in low-income countries. Cryptosporidiosis is becoming more prevalent in both developed and developing countries among patients with AIDS and among children aged less than five years (202, 203). However, there are several other protozoa of relevance for the hospital setting. Several outbreaks of diarrheal diseases caused by *Cyclospora (Cy.) cayetanensis* have been reported recently (204, 205). *Trichomonas vaginalis* is the most common non-viral sexually transmitted disease worldwide (206), but transmission via fomites is ~~rare~~ ().

G. intestinalis and *Cryptosporidium (Cr.)* spp. survive in both aquatic and terrestrial environments. Giardia cysts may remain infectious for months in water or in cool damp areas (207). At temperatures below 15 °C *Cryptosporidium* oocysts can maintain high levels of infectivity in water for at least 24 wk (208-211) and up to 120 d in soil (212). The survival of oocysts of *Cr. parvum* and *G. muris* was inversely correlated with the storage temperature and porosity of the surface (Table 6). Under various

test conditions, the overall trends of the *Cryptosporidium* oocysts die-off were similar to the one of *Giardia* cysts (213). Outbreaks of *Cryptosporidium* spp. and *G. intestinalis* generally occur via drinking water and food which were if inadequately treated to kill or to remove these parasites (214). Other less frequent water-associated outbreaks include *Entamoeba (E.) histolytica / E. dispar, Balantidium (Bal.) coli, Cy. cayetanensis, Microsporidium* spp., *Toxoplasma (T.) gondii* and the free living *Acanthamoeba* species. *Cryptosporidium* spp. can also be transmitted nosocomial via hands and indirect via surfaces (215). In China, an outbreak of cryptosporidiosis was associated with HAI by *G. intestinalis*, *Enterocytozoon bieneusi* and *C. difficile* infection. Poor diaper changing and hand hygiene were probably responsible for this multi-pathogen outbreak (216).

Survival of anaerobic *Entamoeba* spp. in environments is highly dependent on temperature. Survival was determined in faeces and soil at 28-34 °C for 8-10 d, in water and sewage sludge at 0-4 °C for 60-365 d, in surface water resp. wastewater at 20-30 °C for 15 d resp. 10 d (217).

Multiple experiments in soils showed that *T. gondii* oocysts may remain viable for at least 1 year when covered and in cool temperatures (4 °C). Under warm climate conditions in dry soils from Kansas, USA, oocysts remained viable for 18 mon. In fresh or marine waters, oocysts were shown to be viable for at least 4.5 and 2 years, respectively, reviewed by (218). To determine the survival dynamics 2.5 g of soil are inoculated with 1 ml of suspension containing 2×10^5 oocysts. The proportion of oocysts surviving after 100 d was estimated to be 7.4 % under dry conditions and 43.7 % under damp conditions (219).

Babesia (B.) spp. are intraerythrocytic protozoan parasites transmitted primarily by tick vectors, rare also congenital and by blood transfusion (220). Normally, it has its origin in endogenously infected blood donors. A nosocomial transmission in blood products is only indirectly imaginable during the preparation process of blood products in blood bank via hands contaminated from surfaces. Refrigeration decreases the parasite numbers, but parasites survive 31 d at 2-4 °C and yield high end-point parasitemia, proofed by inoculation of hamsters (221). *B. microti* survives in red cells at 4 °C in EDTA-coated blood collection tubes for at least 21 d. Blood held at room temperature did not infect any hamsters (222). Under normal blood bank conditions, a 35-day-old red cell unit was cause of a

transfusion transmitted babesiosis (TTB) (223). Similarly, TTB case reports implicating cryopreserved red cell units indicate that *B. microti* can survive indefinitely in the presence of glycerol cryopreservation (224, 225), but in the absence of cryopreservation, the parasite is rapidly killed by pathogen reduction technology, which uses riboflavin (RB) and ultraviolet (UV) light (226). Theoretically, a single parasite is capable of transmitting infection. Experimental studies, however, have shown that 30 organisms infected about 2 / 5 inoculated hamsters, and 300 organisms infected all animals (227).

Protozoa play a minor role in HAI, but in our increasingly complex healthcare environment with a growing proportion of immunocompromised patients they should be respected, because certain protozoa may cause morbidity and even mortality in both normal and immunocompromised patients (204). Furthermore, climate change with increasing temperatures and heavy rainfall could promote their nosocomial potential in future. There is also the possibility that HAI could be missed because the incubation period may be days to weeks (wk) and the parasite is endemic. It is likely that nosocomial transmission of protozoa may be an even greater problem in tropical hospitals, where comprehensive hygienic measures are costly or otherwise more difficult to maintain and growth conditions more beneficial for the protozoa. Up to 1 % of HAI were caused by parasites depending on geographic region (228), but in this estimation no distinction was made between protozoa and other endo- or ectoparasites. Jarrin et al. (229) assumed that intestinal parasites can cause diarrhoea in 12-17 % of nosocomial epidemics and 1 % of endemic outbreaks, especially on surgical wards. Immunosuppressed patients and those with prolonged antibiotic courses are at higher risk. Enteric protozoa, especially *Cr. parvum*, *G. intestinalis, E. histolytica / E. dispar, Bal. coli, Cy. cayetanensi*s, and *Cystoisospora belli* (syn. *Isospora (I.) belli*) are the most common species involved in nosocomial outbreaks (229).

Spread of enteric protozoa in developing countries usually occurs through fecal contamination due to sewage exposure, poor quality of water and zoonotic exposure, but also via transplantation (230-232). The 50 % infectious dose (ID_{50}) of *C. parvum* has been estimated at 132 oocysts; with some infections followed by ingestion of 30 oocysts (233). Ingestion of at least 10 to 25 *G. intestinalis* oocysts can cause infection in humans (234, 235). Infection after ingestion of a single oocyst has been reported (233). The small ID, the faecal-oral route of transmission, and prolonged environmental survival in water allows

Cryptosporidium to spread in healthcare facilities as well as child-care centers. *Cryptosporidium* can transmit by hands after contact with contaminated environmental surfaces (236). The cysts are highly resistant to environmental conditions and most of the disinfectants commonly used have low or none antiparasitic activity (236). For *Giardia* and *Cryptosporidium* spp. person-to-person transmission is possible (237, 238). For *Cryptosporidium* spp. transmission is primarily found among children and staff members in nurseries, day-care centers, and schools (239). HAI by direct and indirect person-to-person transmission is documented, causing secondary cases among roommates (237). In an outbreak of giardiasis at two day-care nurseries *G. intestinalis* appeared to be transmitted person to person (240). Conversely, ingestion of approximately 200–49,000 oocysts at healthy volunteers did not experience gastroenteritis, and no oocysts were detected in any stool samples over the following 16 wks (241). Therefore, there is minimal risk of nosocomial transmission. Sporulated oocysts of *I. belli* can survive for years in the environment (242). Although the transmission of protozoa via surfaces in hospitals is negligible for most species, awareness of surface persistence is important for assessing the risk of surfaces as a reservoir for food, water, and hands (table 6). *Cr. parvum* oocysts survived in stool on wood up to 72 h, and differed between stool samples (210). Survival was shorter than in water, because other fecal microorganisms such as bacteria may be associated with the shortened survivability (243). Also, the presence of ammonia, which may be present in faeces in high concentrations. This is a significant inactivation agent for oocysts (244, 245). Oocysts have been shown to survive for hours on wet surfaces, including stainless steel, but they resist desiccation and die rapidly on dry surfaces (246).

For virgin girls with high prevalence of trichomoniasis resulting in multivariate analysis, the only statistically significant risk factor for trichomoniasis was inconsistent use of soap. The authors postulate that the high prevalence of trichomoniasis in virgins in Ndola is due to non-sexual transmission of trichomoniasis via shared bathing water and inconsistent use of soap (247).

Acanthamoeba are one of the most common protozoa in soil, and frequently found in fresh water and other environmental habitats such as pools, lakes, brackish water, seawater, heating, ventilating, air-conditioning filters and medical equipment, such as gastric wash tubing and dental irrigation units (248). An important habitat and vector for infection are hydrogel contact lenses, resulting in contact lens

associated keratitis caused by *Acanthamoeba* and *Fusarium* (249), particularly since the contact lenses' moist condition supports survival protozoa. *Acanthamoeba, Vahlkampfia* and *Vermamoeba* spp. have been detected in dust on internal, surgical and open heart surgery intensive care units (ICUs), on equipment, doors and in the air conditioning system (250). *Acanthamoeba* cysts are double-walled, highly resistant dormant stages that remain viable (and infective) for several years (251, 252) and in a state of desiccation up to 21 years (Table 6).

507 **TABLE 6** Replication capacity of protozoa from inanimate surfaces (pathogens with "fomite-borne risk", characterized by an increased nosocomial risk for
508 transmission from inanimate surfaces, are marked in blue; for additional data and details of recultivation and environmental conditions, see supplementary
509 material)

Pathogen	Initial inoculum	Replication capacity	Surface	Reference
Acanthamoeba trophozoites morphological group II	Large numbers of trophozoites	2-21 years	After amoebae differentiated into cysts, agar plates were tightly wrapped with parafilm	(248)
Cryptosporidium parvum oocysts	(Oo)cysts	Survival at 25 °C: > 60 d / > 24 d / > 60 d / > 60 d	Stainless steel / skin / formica / fabric	(213)
	Oocysts	Recovery at 21 °C up to 75 d	Water	(253)
	6 lg / ml oocysts	Recultivation rate after 0 h: 76.3 %; after 2 h: 3 %; after 4 h: 0 %	Glass slide	(210)
	7 lg oocysts	After 30 min: 4.1 lg; after 60 min: 3.2 lg; after 90 min: < 3 lg	Stainless steel	(254)
	≥ 100 oocysts	After 24 h desiccation: no infectivity after 1 – 4 d	Cryptosporidia-laden calf faeces	(255)
Giardia muris cysts	(Oo)cysts	Recovery at 25 °C: 45 d / > 24 d / 21 d / 21 d	Stainless steel / skin / formica / fabric	(213)
Trichomonas vaginalis trophozoites	2-3 lg for human samples; 3-4 lg from culture	Recultivation rates after 120 min: 5.1 % / 30.5 %; survival 24 h	Textil / plastic	(256)
	Trophozoites	Recultivation rates after 15 min at 26 °C: < 10 %	Water	(257)
Legend: lg = decadic logarithm, min = minute, h = hour, d = day				

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Replication capacity of viruses

To determine the RC of viruses, applied material was removed from the germ carrier by scraping or rinsing in cell culture medium; sometimes combined with vortexing and transfer of the sample usually into cell culture. Recultivability is determined, based on the number of infectious virus particles, by growing the remaining virus particles with subsequent determination of the virus titre. In contrast, molecular biological detection alone does not allow any conclusions regarding infectivity. For hepatitis B virus (HBV), infectivity was proven by application of the rehydrated inoculum in chimpanzees due to lack of cultivation in cell culture in the past. Nowadays, it can be analyzed in a HBV susceptible cell culture system using hepatoma cells expressing the Na^+- taurocholate co-transporting polypeptide (NTCP)-HBV cell entry factor (258) (Table 7). However, this method is only available in specialized laboratories and cannot be used routinely.

Gastrointestinal transmissible viruses remain infectious on inanimate surfaces. The longest has an average of 1 - 6 w, followed by blood-borne (average 1 to 6 w), respiratory (average 1 to 3 d) and sexually transmitted viruses (2 h to < 7 d) (table 7).

Non-enveloped viruses are more resistant to extreme pH, heat, dryness, disinfectants in general and some can intrinsically resist certain disinfectants such as the parvovirus or hepatitis A virus (HAV). In contrast, most enveloped viruses such as herpes viruses (cytomegalovirus), human immunodeficiency virus (HIV) and respiratory syncytial virus (RSV) are less environmentally stable since they possess an outer lipid bilayer membrane. Small viruses, e.g. hepatitis B virus (HBV) or the members of the picornavirus or parvovirus family, are much more resistant than larger complex viruses, e.g. members of the herpes or retrovirus families (259). Some non-enveloped viruses, such as enteroviruses belonging to the picorna viridae, are sensitive to drying, e.g. dried inoculum of the Coxsackie B4 (CVB4) virus was easier to recover when CVB4 was spiked in media containing any concentration of NaCl instead of protein load (260).

The relevance of surfaces in healthcare facilities as a contamination source for viruses is even more difficult to prove than for bacteria and fungi, because surface isolation is more complex. Virus infection can so far only be indirectly deduced by tracking the spread of the virus from the patient and its presence in the patient environment, as the ID is not known with a few exceptions. However, in both situations

the risk of infection increases with higher RC. A few examples illustrate the importance of surfaces for the spread of viral infections. After discharge of patients with norovirus infection, the number of new cases has continued to rise, most likely due to the low ID of norovirus (1 to 10 to 100 virus particles) (261). A large outbreak due to noroviruses infections could therefore be controlled by closing the affected departments, implementing extensive disinfection measures, and reducing the exposition risk, i.e. from infected healthcare workers (262). However, if recognized at an early stage, most norovirus outbreaks can be controlled easily without these intensified intervention strategies. A retrospective cohort study showed a very low risk of general infection by only 2 of 1106 exposed patients had acquired the identical norovirus strain from the discharged patient (263). Although the direct hand transmission dominates nosocomial transmission of rotaviruses, surfaces are also relevant for spread (264). A simulation experiment on virus inoculated over surfaces using Cauliflower mosaic virus showed that the virus was detectable on 41 % of the sampled surfaces within 10 h outside of the isolation unit (265). Whether this amount was sufficient to transmit infection was not investigated. After the emergence of MERS-CoV, although the origin is zoonotic, the risk of further spread via surfaces was investigated. The contamination with viral RNA was detected in the environment of hospitalized ventilated patients despite a strict disinfection regimen and negative pressure ventilation. Due to the RC of up to 9 d and the detection in the patient environment, the authors concluded that careful surface disinfection, especially near the patient, can help with prevention (266). Thus, detecting RNA does not necessarily coincide with infectivity.

Other viruses from the gastrointestinal tract such as Astrovirus, HAV, Polio- and Rotavirus can retain their infectivity at RT for quite a long time, with the spectrum varying from several hours to 3 mon. HBV belonging to the group of blood-borne or sexually transmitted viruses play a very high stability with a RC of 50 % of more than 22 d at 37 °C and a persisting infectivity for up to 9 mon at 4 °C (258). In contrast, most respiratory viruses retain their infectivity on inanimate surfaces for a few days only (Table 7).

Herpes viruses such as cytomegalovirus, mainly transmitted through contact with infectious body fluids, e.g. through breastfeeding, kissing, sexual contact, herpes simplex virus (HSV) type 1, mainly transmitted via contact, and HSV 2, mainly transmitted during sex, have been shown to persist from only a few hours up to days (Table 7).

Mpox virus (MPXV)

Since summer 2022, non-travel associated outbreaks of monkeypox have occurred in several non-endemic countries. Person-to-person transmission can occur through exposure to close contact with respiratory secretions, infectious skin lesions (e.g. via ruptured blisters) of an infected person, or recently contaminated objects (sex toys) and surfaces (267); nosocomial infections are described as well (268-271). Recently, the WHO recommended using a new preferred term 'Mpox' as a synonym for monkeypox (272). Investigations with the vaccinia virus – a virus related to the MPXV – showed that this virus can remain 'infectious' on surfaces for up to 56 d (67). Stability on textile fibers was also investigated for the vaccinia virus. Accordingly, this virus could still be recovered from wool fabric after up to 4 wk and from cotton after four to 8 d; textiles contaminated with virus-containing dust even remained infectious for up to 12 wk (273, 274). Adler et al. indicates that in some patients the virus could be detected in the throat swab by PCR test for up to 3 wk (in one case from 2018 even up to 41 d) after diagnosis (275). Whether this was only 'residual nucleic acid' or infectious virus was not investigated. However, viable virus was identified in two (50 %) of four samples selected for viral isolation, including air samples collected during bedding change via air and surface sampling for MPXV in a UK hospital (276). In another study, there was no statistical difference ($p = 0.94$) between MPXV-WA PCR positivity of porous (9 / 10, 90 %) vs. nonporous (19 / 21, 90.5 %) surfaces, but there was a significant difference ($p < 0.01$) between viable virus detected in cultures of porous (6 / 10, 60 %) vs. nonporous (1 / 21, 5 %) surfaces. These findings indicate that porous surfaces (e.g., bedding, clothing) may pose more of a MPXV exposure risk than nonporous surfaces (e.g., metal, plastic). Viable MPXV was detected on household surfaces after at least 15 d (277). Therefore, the CDC recommends minimizing the spread in household by cleaning and disinfection laundry, hard and soft surfaces, carpet and flooring when exposed to an infected person (278).

SARS-CoV-2

Severe acute respiratory syndrome coronavirus type 2 (SARS-CoV-2) demonstrates how infection control of a new infectious disease can be established, and continuously adapted, at breathtaking speed based on hospital hygiene strategies using RC, biocide resistance and transmission. Similar to other coronaviruses, SARS-CoV-2 has been detected on surfaces (279) with a correlation between patient proximity and surface contamination (280), so that the risk of further spread due to RC of up to 7 d on surfaces (Table 7) could be prevented by disinfecting surfaces (281, 282). Even simple wiping with hard water or detergent-based cleaning are effective decontamination strategies against SARS-CoV-2 (283). This applies to all materials (Table 7), even if their influence on RC varies (284). Depending on the exposure time, the recoverable virus quantity decreases almost linearly and is not critical on plastic after 72 h, stainless steel after 48 h, cardboard after 24 h and copper after 4 h (285). Since the ID is unknown, the risk assessment remains open. In a case report, the detection of SARS-CoV-2 on surfaces in the household is interpreted in such a way that transmission from surfaces is possible if they have recently been contaminated by coughing or sneezing, touched and subsequently transferred to mouth, nose or eyes (286). In this investigation, transmission via the respiratory tract cannot be ruled out in the few other cases where transmission via surfaces is suspected (287). Presumably, however, the risk of infection is not very high, because in swab samples from surfaces in an emergency ward and an infectious disease sub-intensive care ward, small amounts of SARS-CoV-2 RNA were detectable in only two of 26 samples and did not cause cytopathic effect in cell culture (288). It is possible that residues of surface disinfectants used were able to reduce RC. In contrast, it is also possible that residues of disinfectants may induce tolerance. Similarly, quantitative microbial risk assessment (QMRA) studies indicate that the risk of SARS-CoV-2 infection via the surface transmission route is low and generally less than 1:10.000, meaning that the probability of infection for each contact with a contaminated surface is less than 1:10.000 (289-291). These results suggest that transmission of SARS-CoV-2 via surfaces in public areas is irrelevant (292). In isolation units / rooms for patients with SARS-CoV-2 infection and in units or rooms for suspected patient cases of SARS-CoV-2 infection, surface disinfection and cleaning is indicated based on the observation that SARS-CoV-2 can be detected in the entire patient environment. Moreover, the RC is up to 7 d, although the infectivity of the surfaces is apparently only

623 low. In a retrospective questionnaire-based study, it was shown that even at home the use of protective
624 masks and daily use of chlorine- and ethanol-based disinfectants for surface decontamination and hand
625 antisepsis significantly reduced the risk of infection (293). Santarpia et al. (294) deduced from the data
626 that in cases of suspected or confirmed SARS-CoV-2 infection within the last 24 h in the household,
627 surfaces should also be decontaminated.

628 **TABLE 7** Replication capacity of viruses after isolation from inanimate surfaces modified from (70) (pathogens with "fomite-borne risk", characterized by an
629 increased nosocomial risk for transmission from inanimate surfaces, are marked in blue; for additional data and details of recultivation and environmental conditions,
630 see supplementary material)

Pathogen	Initial inoculum	Replication capacity/ residual virus titer	Surface	Reference
Predominant transmission through contact				
Adenovirus	~ 7 lg $CCID_{50}$	> 12 wk; after 8 wk: 3.4-5.7 lg	Glass / plastic / porcelain / stainless steel	(295)
	2000 PFU	< 49 d; after 14 d: ~ 8 % / ~ 3 %	Plastic / aluminum foil	(296)
	~ 6 lg PFU	15 d / 15 d / 30 d / > 30 d	Aluminum / porcelain / latex / paper	(297)
Adenovirus type 3	~ 7 lg $TCID_{50}$	> 9 d: 4.2 lg	Polystyrene	(298)
Cytomegalo-virus	4-6.9 lg PFU	1-2 h / 4-8 h	Cotton / plexiglass	(299)
Ebola virus	4-6 lg $TCID_{50}$	At 4 °C > 50 d: 2 lg	Plastic / glass / stainless steel	(300)
	7 lg PFU;	6.2 d	Paper	(301)
	7.3 lg PFU	> 5.9 d: 4 lg	Glass / silicone / aluminum	(302)
	6-7 lg $TCID_{50}$	14 d / 8 d / 11 d	Tyvek / stainless steel / plastic	(303)
	7 lg $TCID_{50}$	> 192 h / > 192 h / < 24 h / > 192 h; 3-4 lg	Stainless steel / surgical mask / cotton / plastic	(304)
Hendra virus (HeV)	~ 6.25 lg $TCID_{50}$	60 min; after 30 min: ~ 2.7 lg	Polystyrene	(305)
Lassa virus	7.1 lg PFU	> 9.7 d: 4 lg	Glass / silicone / aluminum	(302)
Mpox	Household setting after disease	At least 15 days: ≤ 2 lg / 0-≤ 2 lg	Porous surfaces / non-porous	(277)
Marburgvirus	4-7 lg $TCID_{50}$	> 50 d: 2 lg	Plastic / glass	(300)
Nipah virus (NiV)	~ 6.25 lg $TCID_{50}$	After 60 min: ~ 2.7 lg	Polystyrene	(305)
Sindbis virus	7.2 lg PFU	> 14.6 d: 4 lg	Glass / silicone / aluminum	(302)

Vaccinia virus	7 lg $CCID_{50}$	> 4 wk: 2 lg	Glass	(295)
	8 lg $CCID_{50}$	14 wk: 3 lg / up to 10 wk: 3.5. lg	Wool / cotton	(273)
	8 lg $CCID_{50}$ / ml	1 wk: 4 lg	Cotton	(274)
	2.8 lg $TCID_{50}$	14 d: < 1 lg	Gauze bandage	(306)
	8 lg PFU	< 56 d: ~ 4.5 lg	Stainless Steel	(307)
	6-6.5 lg KID_{50}	< 20 wk: 4.3 lg	Glass	(308)
Transmission by contact, starting from the gastrointestinal tract (+ surrogate viruses)				
Adenovirus type 40	5-5.7 lg IU	> 7 d: 3.8 lg	Paper / porcelain	(309)
Astrovirus, serotype 4	5-5.7 lg IU	60 d / after 7 d: 1.7 lg	Paper / porcelain	(309)
Coxsackie virus	6.8 lg $CCID_{50}$	2 wk: 2 lg	Glass	(295)
	6.5 lg $TCID_{50}$	< 6 wk	Petri dish	(310)
Echovirus	max. 300 PFU	42 h	Cellulose	(311)
Feline calicivirus	9 lg PFU	> 7 d: 2 lg	Laminate / ceramic / stainless steel	(312)
	7 lg $TCID_{50}$	90 % reduction in viral titers: up to 24 h	Computer / brass / telephone	(313)
	6 lg PFU	< 15 d / <3 d / < 7 d	Wool / nylon / glass	(314)
Hepatitis A virus (HAV)	6 lg PFU	> 1 mo	Wood / stainless steel	(315)
	3-4 lg PFU	4 h to > 7 d	Stainless steel	(316)
	5-5.7 lg IU	After 7 d: ~ 3.3 lg / ~ 5 lg	Paper / porcelain	(309)
	6.4 lg	After 90 d on PVC: 10 % of initial loading	Stainless steel / PVC	(317)

	~ 6 lg PFU	> 60 d / > 60 d / > 60 d / > 30 d	Aluminum / porcelain / latex / Paper	(297)
Hepatitis E virus (HEV)	~ 4 lg FFU	After 28 d: ~ 1 lg / 1 lg / 0.4 lg / 0 lg	Plastics / ceramics / stainless steel / wood	(318)
	3.9 lg FFU	D value: 5.95 d	Stainless steel	(319)
Escherichia virus (MS2 phage)	6 lg PFU	D value: 19.8 d / 13.2 d	Wood / stainless steel	(315)
Murine hepatitis virus and (MHV) Transmissible gastroenteritis virus (TGEV)	4-5 lg PFU	MHV: after 5 d 3 lg; TGEV: after 3 d 2 lg	Stainless steel	(320)
Murine norovirus	4-4.5 lg PFU	> 120 min except copper; after 120 min: 3.1 lg for stainless steel	Copper 100 % / 95 % / 70 % / stainless steel	(321)
Poliovirus type 1	4.4 lg PFU	> 90 min; after 20 min: 2.6 lg	Worktop	(322)
	~ 6 lg PFU	3 d / 1 d / 30 d / > 30 d	Aluminum / porcelain / latex / Paper	(297)
	max. 300 PFU	42 h	Cellulose	(311)
	~ 12 lg PFU	> 3 wk on all surfaces; 99 % reduction after 5.2 d / 7.4 d / 5.9 d	Steel / cotton / plastic	(323)
	3-4 lg PFU	12 h	Stainless steel	(316)
Poliovirus type 2	8.1 lg PFU	After 14 d: > 3 lg	Glass	(295)
	5-5.7 lg IU	> 7 d	Paper / porcelain	(309)
Rotavirus	~ 6 lg PFU	> 60 d	Aluminum / porcelain / latex / paper	(297)
	3-4 lg PFU	< 90 min	Worktop	(322)
	7 lg PFU	> 10 d	Glass / smooth plastic / rough plastic	(324)
	5-5.7 lg IU	> 7 d	Paper / porcelain	(309)
Tulane virus (Rhesus enteric calicivirus)	4.7 lg PFU	D value: 18.8 d / 13.3 d	Acrylic / stainless steel	(325)
Respiratory and/or aerogenic transmission (+ surrogate viruses)				
Endemic human coronaviruses	5.7 lg $TCID_{50}$	HCoV-229E: > 12 h, >12 h, >6 h; HCoV-OC43: > 3 h, > 1 h, >1 h	Aluminum / cotton / latex	(326)

	3 lg PFU	3 d / 5 d / ≤ 40 min / 120 min / 30 min	Silicone / PVC, ceramic, glass, steel / brass / 70 % copper / 90 % copper	(327)
	~ 7 lg $TCID_{50}$	48 h: 2 lg	Polystyrene	(298)
Influenza A virus	3.1 lg $TCID_{50}$ (A/NC-H1N1); 4.8 lg $TCID_{50}$ (A/Br-H1N1)	7 d	Stainless steel	(328)
	5.5 lg $TCID_{50}$	> 24 h / > 48 h / > 24 h / 8 h	Stainless steel / wood / plastic / cotton	(329)
	5.3 lg $TCID_{50}$	≥ 60 min / 30 min / 15 min / < 15 min / < 15 min	Cotton / formica / vinyl / stainless steel / facial tissue	(330)
	5 lg $TCID_{50}$	< 5 d	Petri dish	(310)
	4-6 lg PFU	After 7.3 d / 17.7 h / 34.3 h 99 % reduction	Stainless steel / cotton / microfiber	(331)
	3-4 lg $TCID_{50}$	48 h / 72 h / 24 h / 24 h / 12 h	Plastic / stainless steel / magazine / cotton / paper	(332)
	6 lg PFU	2-9 h	Telephone receiver / wood / keyboard / stainless steel / dishcloth	(333)
	6 lg $TCID_{50}$	< 4 h	Stainless steel / plastic	(334)
Influenza B virus	4 lg $TCID_{50}$	48 h / 48 h / 8 h / 12 h / 8 h	Plastic / stainless steel / magazine / cotton / paper handkerchief	(332)
Middle East respiratory syndrome coronavirus (MERS-CoV)	6 lg $TCID_{50}$	< 72 h	Stainless steel / plastic	(334)
Parainfluenza virus	3.2 lg $TCID_{50}$	4 h	Stainless steel / laminate	(335)
Respiratory syncytial virus	5 lg $TCID_{50}$	8 h; ~ 2.5 h; ~ 5.3 h; 1 h; 1 h	Laminate / cotton-polyester / rubber / paper / hands	(336)
Rhinovirus type 14	7 lg PFU	< 25 h; $TCID_{50}$: 0.55 h	Stainless steel	(337)
Rhinovirus type 2	2 lg PFU	After 3 d: ~ 0.6 lg	Stainless steel	(338)
SARS-CoV-1	6 lg $TCID_{50}$	4 d / 4 d / 4 d / 5 d / 5 d	Wood / glass / paper / metal / textile /	(339)

	7 lg $TCID_{50}$	28 d: ~ 2 lg	Plastic	(340)
	3.4 lg $TCID_{50}$	72 h / 48 h / 8 h / 8 h	Plastic / stainless steel / paper / copper	(341)
	6 lg $TCID_{50}$ / ml	1 h / 24 h / 2d	Paper / cotton / disposable gown	(342)
	7 lg $TCID_{50}$	After 13 d: 2.3 lg	Plastic	(340)
	~ 7 lg $TCID_{50}$	After 9 d: 2 lg	Polystyrene	(298)
	6 lg $TCID_{50}$	4 d / 4 d / 4 d / ≥ 5 d / ≥ 5 d / 4 d	Plastic / wood / glass / metal / cloth / paper	(339)
SARS-CoV-2	5.5 lg $TCID_{50}$	D values: ~ 6 d / ~ 6.9 d / ~ 9.1 d / ~ 6.3 d / ~ 5.6 d / ~ 6.3 d	Stainless steel / paper / polymer / glass / cotton / vinyl	(343)
	7.9 lg $TCID_{50}$	After 7 d: ~ 2.7 lg / 2 lg / 2.8 lg / not detectable / 2.3 lg / 2.3 lg / 1.1 lg / not detectable	Stainless steel / face shield / nitrile glove / chemical glove / N95 mask / N100 mask / Tyvek suit / cotton	(344)
	3.6 lg $TCID_{50}$	72 h / 48 h / 24 h / < 4 h	Plastic / stainless steel / cardboard / copper	(341)
	7.8 lg $TCID_{50}$	< 3 h / < 3 h / < 2 d / < 2 d / 4 d / 4 d / < 7d / < 7 d / 7 d	Paper / handkerchief / wood / clothes / glass / paper / stainless steel / plastic / surgical mask	(345)
	6.2 ± 5.9 lg $TCID_{50}$	13 min at 0.3 W / cm^2: 90 % reduction	Stainless steel	(346)
	6.5 lg $TCID_{50}$	< 20 min exposed to sunlight	Stainless steel	(347)
	~ 2.8 lg $TCID_{50}$	≤18.6 h	Stainless steel / plastic / nitrile	(348)
	5.23 lg $TCID_{50}$	2 d: ~ 1.2 lg	Glass	(349)
Predominant sexual transmission				
Herpes simplex virus type 1	7.9 $TCID_{50}$	After 2 h: 6.7 lg	Plastic / chrome	(350)
		After 2 h: 5.2 lg		(351)
	5.6 lg PFU	After 1 d: 4 lg	Glass	(295)
	~ 7 lg $TCID_{50}$	After 9 d: 1.9 lg	Polystyrene	(298)
Herpes simplex virus type 2	4.2 lg $TCID_{50}$	4.5 h: 2.9 lg $TCID_{50}$	Polystyrene	(352)
Human immunodeficiency virus (HIV)	Liquid / dry inoculum: 128000 / 25000 cpm / ml reverse transcriptase	> 20 d / ~ 10 d	Petri dish	(353)
Papillomavirus	~ 100-434 FFU	< 7 d	Pipe / cotton / microcentrifuge tube	(354)

Transmission through blood				
Hepatitis B virus (HBV)	0.1 ml HBsAg positive plasma	1 wk	Silanized tube	(355)
	0.1 ml HBV-positive blood	> 2 wk	Stainless steel / cotton swab	(356)
	> 6 lg $TCID_{50}$	After 28 d: ~ 10 % reduction	PCR tubes	(258)
Hepatitis C virus (HCV)	4-6 lg IE	>40 d	24-well plates	(357)
	~ 4.75 lg $TCID_{50}$	After 7 d: ~ 1.5 lg	Stainless steel	(358)

Legend: cmp = counts per minute, D value = time in which the virus titer is reduced by 1 lg, Z value (thermal death time) = number of degrees the temperature has to be increased to achieve a 10-fold decrease in decimal reduction time (D-value), ATCC =American Type Culture Collection, BSA = bovine serum albumin, CCID = cell culture infectious dose, CPE = cytopathic effect, d = day, FFU = focus forming units, h = hours, HBsAg = Hepatitis B surface Antigen, HBVcc = HBV derived from cell cultures, IU = infectious units, lg = decadic logarithm, min = minute, mon = month, N/A = not available, PBS = phosphate-buffered saline, PCR = polymerase chain reaction, PFU = plaque forming unit, PPE = personal protection equipment, PVC = polyvinyl chloride, RH = relative humidity, RIA = Radioimmunoassay, RT = room temperature, $TCID_{50}$ = 50 % tissue culture infectious dose, US = ultrasound, W = watt, wk = week

631

Factors influencing the replication and infection capacity of microorganisms, protozoa and viruses in the environment

Microbiological test conditions: For bacteria, surface desiccation on the surface after contamination (rapid or slow), relative humidity (RH) and temperature during storage, recultivation conditions, and stage of cultivability (VBNC) are of influence on RC (Tables 3 and 4). The origin of the pathogen is also influential. *A. baumannii* strains isolated from clinical settings were more often resistant to desiccation than ATCC strains (Table 3). As expected, the RC is influenced by the initial bio-inoculum of faeces, demonstrated for *E. faecalis*, MRSA, *A. baumannii, C. jejuni* (table 3), *E. coli, P. aeruginosa* of recovery (Table 4), *C. albicans, C. auris, C. krusei (Pichia kudriavzevii), C. parapsilos*is and *C. tropicalis* (Table 5). Similarly for viruses smaller inocula were associated with shorter RC, e.g. for transmissible gastroenteritis virus, mouse hepatitis (320) and SARS-CoV-2. The latter lost infectivity after 2-4 d (341, 345) compared with longer times of 21 d (344) or 7-28 d (343) for larger inocula (Table 7). Finally, the RC depends on recovery method (Tables 3-7).

Surface material: The RC of bacteria, fungi and viruses was significantly shorter on copper surfaces than on textile materials, plastics and steel, due to the oligodynamic effect of copper ((359, 360); Table 7).

On porous surfaces, e.g. coronavirus, influenza virus, avian metapneumovirus, poliovirus type 1 and human enteric adenovirus type 40 (297, 361), survival is longer than on non-porous surfaces (Table 7). One reason may be the lower virus elution during recovery from porous materials (362). A recently published scoping review comes to the same conclusion ().The capillary effect within the cavities and the faster evaporation of the aerosols could also be influential (363).

RH: Gram-positive bacteria tolerate dry conditions better than Gram-negative bacteria due to cell wall properties (364). *S. aureus* persisted longer at low RH (365), while survival kinetics for *E. faecalis* were lower at 25 % RH than at 0 % RH (366). *Acinetobacter* spp. suspended in distilled water survived significantly longer at room temperature (RT) at RH of 28-34 % and 93 %, respectively, compared to 10 % RLF, while survival did not differ between 28-34 % and 93 %, respectively (72). Survival of

Gram-positive bacteria was reduced most at RLF of 50-70 %, while death rates of Gram-negative bacteria were highest at RLF of 50-70 % and 70-90 %, respectively (364).

Enveloped viruses, especially respiratory viruses such as influenza, parainfluenza, corona-, respiratory syncytial, measles and rubella viruses, but also herpes simplex and varicella-zoster viruses, retain their RC longer with a low RH of 20-30 % (364). Only cytomegalovirus is isolated more frequently from moist surfaces (367). Non-enveloped viruses such as adenoviruses, enteroviruses and rhinoviruses are replicable for longer at 70-90 % RH (table 7, (368)).

Temperature: Constant temperatures > 24 °C seem to reduce the replication and infection capacity of airborne bacteria, shown for representatives of Gram-positive, Gram-negative and intracellular bacteria (364).

For 15 yeast species, the survival time increased when the ambient temperature was reduced. Overall, the survivability of the species studied was longest at 4 °C and 1 % RH and shortest at 37 °C and 96 % RH (198). The situation is different for the release of bioaerosols indoors. At 25 °C, more fungi (mainly Fusarium and Penicillium spp.) were released than at 37 and 15 °C, whereby the composition of the mould species differed significantly across these three temperature ranges (369).

The viral genome (viral DNA or RNA) is sensitive to the surrounding temperature. Indeed, temperature is an important factor influencing the RC of several viruses. Higher temperatures affect / impact viral proteins and enzymes, as well as the viral genome. In general, DNA viruses are more stable than RNA viruses; yet high temperature will also affect DNA integrity. For most viruses, such as astro-, adeno-, polioviruses, herpes simplex and HAV, low temperatures (4 °C) are associated with longer duration of replicability (65). For enteric viruses, RC in water increased with increasing temperature > 20 °C (370, 371). For rota-, poliovirus and HAV, RC was higher at > 80 % RH (297). This was confirmed for poliovirus in that stability was significantly greater at 95 % RH than at 25 % RH (316). For coronaviruses, the influence of RH was different with higher RC at 20 % and 80 % and comparatively

688 lower RC at 50 % (320). For SARS-CoV-2, interfering substances, temperature (20 or 35 °C) and RH
689 were only of moderate influence (Table 7). Morris et al. (372) developed an original prediction model
690 of how temperature and humidity alter RC by using a mechanistic quantitative approach that was based
691 on testing the stability of SARS-CoV-2 on an inert surface for a range of temperature and humidity
692 conditions. SARS-CoV-2 remained infectious longest at low temperatures and extreme humidity (up to
693 85 %). The estimated mean half-time of RC was > 24 h at 10 ˚C and 40 % RH, but ~ 1.5 h at 27 ˚C and
694 65 % RH. The model uses basic chemistry to explain why the sensitivity of enveloped viruses increases
695 with higher temperatures and has a U-shaped dependence on humidity. The model accurately predicts
696 existing results on the influence of temperature and RLF for five different human coronaviruses. This
697 suggests that common mechanisms may influence the stability of many viruses.
698
699 **Light conditions:** Light, especially sunlight, or lack of it influences the RC. The survival time of *C.*
700 *albicans* and *Rhodotorula rubra* on smooth glass surfaces doubled when they were kept in darkness
701 compared with daylight and extended from 44 to 98 d for *C. albicans*(198).
702
703 Under the influence of simulated sunlight, 90 % of SARS-CoV-2 applied to the surface in artificial
704 saliva were inactivated every 6.8 min during simulated summer exposure, but every 14.3 min during
705 winter exposure (346). In contrast, no significant decrease was detectable within 1 h in the dark (Table
706 7; (346)). The effect of sunlight was also reproducible in aerosol, while RH alone (20-70 %) had no
707 influence (373). Irradiation (distance 3 cm) with UVC (dose 1.048 mJ / cm^2) completely inactivated
708 SARS-CoV-2 (infectious titre of 5×10^6 $TCID_{50}$ / ml) after 9 min, while UVA (dose 292 mJ / cm^2)
709 reduced the titre by only 1 lg after 9 min (374).
710
711 **Protein, fecal and urine load:** Desiccation in protein-containing media prolongs persistence, e.g. for
712 *A. baumannii* (table 8), *Escherichia (E.) coli* (102), *Neisseria (N.) meningitidis* (375) and yeasts (198).
713 Fecal load had little effect on the RC of HAV and rotaviruses. For adenoviruses, the RC only tended to
714 increase (Table 7).

TABLE 8 Persistence of different *A. baumannii* strains suspended in water or bovine serum albumin (BSA) and dried on glass at different RH (modified from (72))

Average persistence	Strain(s)	Conditions (RH 28-34%, RT)
≤ 5 d	American type culture collection (ATCC) 9955	suspended in water
6-10 d	ATCC 17978, ATCC 19606, R 0211019	
> 10-30 d	ATCC 17904, 18, 49, 16 / 48, 16 / 49, R 447	
<10 d	ATCC 9955	suspended in 7 % BSA
> 10-30 d	ATCC 17978, 18, 16 / 48	
> 29-60 d	ATCC 19606, ATCC 17904, 49, 16 / 49, R 447, R 0211019	

Biofilm: Biofilm is the predominate 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 (376) as well as cold atmospheric plasma (377, 378). For example, *K. pneumoniae* remained viable up to 4 weeks in a dry biofilm, proving the need for robust cleaning regimens (). The reason for the unspecific increased tolerance is the production of extracellular substances such as polysaccharides, proteins and DNA after attachment to surfaces. Besides wet surface biofilm in plumbing systems or other wet surfaces, biofilm on dry inanimate surfaces at room humidity must also be considered (379).The biofilm matrix restrains water and nutrients and protects the microorganisms against environmental influences (380, 381). Once formed, biofilms are important for persistence of microorganisms on surfaces in nature as well as in industrial or medical areas (380-382). The RC on inanimate surfaces is prolonged and depends on environmental conditions, especially humidity. In addition, biofilms have been demonstrated on several objects and surfaces in hospitals, such as sterile supply buckets, opaque plastic doors, venetian blind cords, and sink rubbers, and it is possible to cultivate viable bacteria. Currently, there is insufficient research to elucidate whether presence or absence of biofilm affects the risk of transmission or possibility of cross-transmission. However, multi-drug resistant bacteria may not only be protected within biofilms, but could be the mechanism as to why they persist within the hospital environment (383). They may also exchange virulence factors among their own species or to other species present in biofilms (381, 383-385).

At present there is limited knowledge about the relationship between viruses and biofilms. Since viruses are strict intracellular pathogens, they will be unable to proliferate in biofilms, but they can persist in a reservoir host due to the advantages conferred by the biofilm structure (386). Biofilms may encompass a set of non-enveloped enteric viruses, including caliciviruses, rotavirus spp., astrovirus spp., and hepatitis A virus, alongside other microorganisms such as Gram-negative bacteria and filamentous fungi (387). Biofilms can enhance virion RC in extracellular environments, such as on fomites and in aquatic sediments, allowing viral persistence and dissemination. Importantly, both virions and virus-infected eukaryotic cells embedded in biofilms have been reported to retain infectivity. A study investigated the enveloped virus herpes simplex virus 1 (HSV-1) and the non-enveloped virus coxsackie virus type B5 (CVB5) within fungal *Candida albicans* biofilms (388). Viruses stored in biofilms may be regarded as temporary or long-term reservoirs in the environment (52). The potential of viral spreading via contaminated surfaces depends on the ability of the virus to maintain infectivity while it is in the environment, and biofilms aid protection against desiccation and antimicrobial agents (389).

Discussion

The decisive difference to the first systematic review in 2006 (65) on the resilience of pathogens against environmental influences is that the course of the RC over time was calculated based on the quantity of the inoculum on the surface, expressed as lg reduction. This results in more accurate values and explain different values in some cases of the first review. Additional, the methodological development of laboratory experiments to determine the RC over the last almost two decades also influence the results.

In general, clinical epidemiological evidence for transmission scenarios beyond outbreaks is lacking. However, studies on RC and evidence for persistence on inanimate surfaces in combination with a conspicuous transmission event are available. It is clear that the inanimate environment plays a relevant role in these bacterial transmission pathways in the everyday situation (Fig. 1). As studies using whole genome sequencing indicate, there is a serious underestimation of transmission events when using standard techniques only (390). These analyses tend to focus on resistant, thus easily recognizable

764 pathogens. However, the quantification of transmission events and thus, an appropriate risk assessment
765 is not yet possible.
766 Beyond the epidemiological evidence, the studies were usually generated under laboratory conditions.
767 This means that not all possible environmental influences in hospital settings can be detected, especially
768 any from antimicrobial residues. In addition, the influence of the simultaneous contamination of hospital
769 surfaces with various nosocomial pathogens, with secretions, excretions and dirt will also be
770 disregarded. A growing number of studies report that enveloped and non-enveloped viruses can spread
771 in groups in so-called 'collective infectious units' (391-393). The vehicles mediating collective spread
772 vary widely and include lipid vesicles, protein matrices, diverse forms of aggregation, and binding to
773 the surface of host or non-host cells (391). It seems reasonable, that units like this or interference may
774 also exist for bacteria and / or fungi and / or protozoa. Laboratory studies do not reflect the clinical
775 situation and represent probably a one-sided worst-case scenario assessing the upper bound of infection
776 risk. Furthermore, they cannot represent the complexity of real-life scenarios. When assessing factors
777 that influence the RC, it must be considered that the results only apply to the species investigated and
778 cannot be generalized. Even more so, resistant isolates are often analyzed compared with wild type
779 variants. Sometimes tested microorganisms are poorly characterized so cannot determine the extent of
780 generalizability. Furthermore, it should be noted that data on the RC are often not median values; the
781 maximum was detected and described and these results can, and should, be used as an upper bound
782 approach. Data suggests that no general prediction about RC independent of genus is possible.
783 Additionally, further influences must be considered. Firstly, the dependence of environmental
784 conditions on the RC has not yet been sufficiently studied under real life conditions. Secondly, there is
785 insufficient data on the behaviour of wildtype and/or sensitive strains and variants within a species.
786 Thirdly, no data exist, on whether certain virulence or RC determinants are genetically present in isolates
787 that are particularly well adapted to the hospital setting.
788 In this review, only the risks due to direct or indirect contact transmission from inanimate surfaces were
789 addressed, not the additional risks by potential aerosolization of pathogens from fomites (394-396).
790 Therefore, it should be considered that the RC in aerosols can be significantly lower than on surfaces,
791 as has been proven for different variants of Ebola virus and Marburg virus (397). It is also the case that

high inocula results in longer survival times due to the logarithmic death curve (398), which has been proven for various bacterial species (98, 399) and or fungal spores (198) on surfaces. Considering all background factors, data generated under laboratory conditions can only provide a rough orientation. In case of doubt, the unfavorable situation should be assumed when evaluating the data in Tables 3-7.

Despite knowledge on dependency of replication and infection capacity from factors like pH, temperature, humidity, and others, we cannot easily change these surrounding conditions using their preventive potential. For others, e.g. inocula and biofilms, we can use knowledge covering these aspects from common IPC recommendations.

Another viewpoint for the risk assessment of surface contamination is the minimal infectious dose (MID) to trigger infection. The lower the ID, the greater the risk of acquiring an infection and further transmission as nosocomial outbreaks. It should be noted that the ID can be reduced by a viral infection, which often leads to bacterial co- or superinfection, especially in cases of respiratory viral infections (400-402). In Table 9, examples of different IDs are summarized, mainly taken from reviews. From the clinical perspective it must be considered that this dose depends on the site of infection or at least contamination allowing short-term contamination. For respiratory transmissible viruses with a MID > 10^2 50 % tissue culture infectious dose ($TCID_{50}$), infection by aerosolization from surfaces is unlikely. In contrast, infection is possible via the surface-finger-eye route for keratoconjunctivits epidemica due to the low ID (Table 9) and the surface-finger-nose route, particularly in the case of nasal exposure to respiratory viruses with a MID < 10^1. The same applies to orally transmissible pathogens with a MID <10^1 $TCID_{50}$, CFU resp. oocysts. This is supported by the outbreak potential of pathogens with low MID. For fecal-orally transmissible bacteria and mucorales, transmission from surfaces is unlikely with a MID < 10_2 CFU. However, it should be noted that MID studies do not usually consider the fact that the pathogens multiply from an initially acquired small number and the infection only manifests after the critical quantity has been reached.

The lower the ID and the greater the RC, the greater the risk to acquire an infection by contact with the surface or indirect by aerogenic turbulence from the surface and following inhalative exposition. Likewise the risk of an outbreak emanating from surfaces increases. In both, the ID is likely to have the greater influence. At the same time, the risk of a fomite-borne HAI is influenced by the patients' immune

820 status. The ID, RC and immune status must be considered when deciding upon targeted surface
821 disinfection and additional IPC.

822 **TABLE 9** Minimal infectious dose of selected pathogens

823

Infectious dose	Application	Pathogen	Reference
1-100 virus particles, CFU resp. oocysts	Oral	Noro-, Rotavirus, EHEC, ETEC, *C. difficile*, MRSA, *Cr. parvum, G. intestinalis*	(66, 233, 261, 366, 403-407)
6.6 virus particles	Inhalative	Adenovirus type 4	(408)
10-100 virus particles	Oral	HAV	(409)
30-40 $TCID_{50}$	Intranasal	RS virus	(408)
6 / 71 $TCID_{50}$	Intranasal / oral	Coxsackievirus A21	(408)
0.03 / $>10^1$ - 10^4 $TCID_{50}$	Intranasal / inhalative	Rhinovirus, different serotypes	(408)
< 10^3 CFU	Oral	*Acinetobacter* spp., , *C. jejuni Klebsiella* spp., , VRE,	(66, 410)
≥ 10^3 spores	Chorio-allantois-membran henn egg (equivalent to eye contact)	*Lichtheimia corymbifera*	(411)
≥ 10^3 CFU	Oral	*Salmonella enteritidis*	(412)
≥ 10^3 $TCID_{50}$	Oral	Echovirus	(408)
> 10^3 $TCID_{50}$	Inhalative	Influenzavirus A (H3N2)	(408)
> 10^3 LD_{50}	Intranasal	Congo Basin MPXV	(413)
>10^3 $TCID_{50}$	Inhalative	Influenza A (H3N2)	(408)
≥ 10^4 CFU	Dermal	*P. aeruginosa*	(414)
≥ 10^4 -≥ 10^7	Inhalative	Influenzavirus B, different serotypes	(408)
≥ 10^4 spores		Rhizopus *spp*., *A. fumigatus*	(415, 416)
10^5 $TCID_{50}$	Conjunctival	Respiratory syncytial virus (RSV)	(408)
≥ 10^5 CFU	Intravenious	*C. albicans, C. auris*	(417)
≥ 10^5 spores	Parenteral	*Rhizomucor pusillus*	(415)
> 10^5 CFU	Oral	*E. coli, S. aureus*	(418)

> 10^5 LD_{50}	Intranasal	West African MPXV	(413)
>10^6 $TCID_{50}$	Oral	Adenovirus	(408)
>10^6- >10^7 $TCID_{50}$	Inhalative	Influenza A (H1N1), different serotypes	
>10^8 CFU / ml	Intra-peritoneal	*P. aeruginosa*	(419)
>10^{10} CFU / ml		*S. aureus*	
Legend: CFU = colony forming units, $TCID_{50}$ 50 % tissue culture infective dose, LD_{50} 50% letal dose			

Disinfecting surfaces in hospitals is generally accepted as a key component of infection prevention (32-35, 71, 420-423). But disinfection can also have an influence on the development of tolerance; it is costly and leads to an ecological footprint. Clearly, every disinfection event requires a clear indication. Disinfection must be implemented in a precise and quality-assured manner, since it offers a valuable contribution towards HAI prevention. Regarding environmental protection, probiotic cleaning agents are a promising alternative to chemical disinfection. Surface contamination with pathogens could be reduced by up to 90 % more with probiotic products compared with conventional disinfection wipes (424, 425). SARS-CoV-2 was reduced significantly more by probiotic cleaning than by chemical disinfection (426). In non-intensive care units, routine surface disinfection did not prove superior to soap-based or probiotic cleaning in terms of preventing HAI (427). Of course, no evidence-based practical approach for systematic surface or probiotic cleaning in hospitals can be derived from the RC of nosocomial pathogens.

RC and ID influence the implementation of surface decontamination regarding the extent and the selection of the application concentration and exposure time of the disinfectant. In cases of high RC and low ID, it makes sense to use concentrations that are rapidly effective. For final disinfection after patient discharge, all potential pathogen reservoirs must be eradicated with choice of effective disinfectants. In general, a simple four-step guide for daily decontamination of the occupied bed space can be recommended: Step 1 (LOOK) describes a visual assessment of the area to be cleaned; Step 2 (PLAN) argues why the bed space needs preparation before cleaning; Step 3 (CLEAN) covers surface cleaning /disinfection; and Step 4 (DRY) is the final stage whereby surfaces are allowed to dry. Visible soil should always be removed with detergent and water before using disinfectant (428). Analogous to the 5 moments of hand antisepsis (429), 5 moments of disinfecting surface cleaning can be distinguished: **I.**

Disinfecting surface cleaning as part of standard precautions (non-targeted disinfection) on near-patient (high-touch) sites during patient care, and targeted disinfection as **II.** Disinfecting surface cleaning on the work surface before performing aseptic activities, **III.** Final disinfecting surface cleaning after discharge of patients, **IV.** Two step disinfection surface cleaning after visible surface contamination (first cleaning, thereafter disinfection) and **V:** Disinfection surface cleaning as part of the multi-barrier strategy to control outbreaks (428).

This review can help to reduce the complexity of disinfection choices depending on the range of pathogen properties. At the same time, it proposes the best possible balance between patient and employee safety, i.e. IPC and ecological and economic sustainability. Through a novel classification of pathogens by their fomite-borne potential for transmission - completely independent of the taxonomic approach - a fact-based but also realizable and pragmatic recommendation can be prepared with a view to avoiding transmission. The attempt to classify pathogens by fomite-borne transmission potential should serve only as a first suggestion and should be improved by scientific discussion. In general, further studies should focus beyond the ecological and outbreak assessment –and target real life settings or near real life scenarios in order to emulate endemic settings. There is insufficient evidence regarding the impact of contaminated surfaces for encouraging contact-free transmission risk. Further analysis should cover aspects of ecological sustainability and should weight up the potential benefit for transmission and infection events against the additional ecological footprint from resource consumption, production, and waste management.

Conflicts of interest

None declared

Acknowledgement

The authors thank Isabella Dresselhaus (Department of Infection Control and Infectious Diseases, University Medical Center Göttingen (UMG), Georg-August University Göttingen, Germany) for her important contribution to this review.

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