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

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SUMMARY In healthcare settings, contaminated surfaces play an important role in the 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 exposure, 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 = 171) 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 the degree of contamination, surface material, temperature, relative humidity, protein load, organic soil, UV-light (sunlight) exposure, and pH value. In general, investigations

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into surface RC are mainly performed *in vitro* using reference strains with high inocula. *In vitro* data from studies on 14 Gram-positive, 26 Gram-negative bacteria, 18 fungi, 4 protozoa, and 37 viruses. It should be regarded as a worst-case scenario indicating the 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, viability, inanimate surfaces, fomites, persistence, resilience, tenacity, bacteria, fungi, protozoa, viruses, transmission, HAI, fomite-borne risk pathogens

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. 1a). 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 in 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, for example, impregnated catheters (36). However, as recently witnessed during the severe acute respiratory syndrome coronavirus type 2 (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 into the RC of pathogens on inanimate surfaces 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).

Defining terms of cultivable pathogens from inanimate surfaces

Resilience is the quality to withstand or recover quickly from environmental challenges 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 patients' 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 the 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. 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

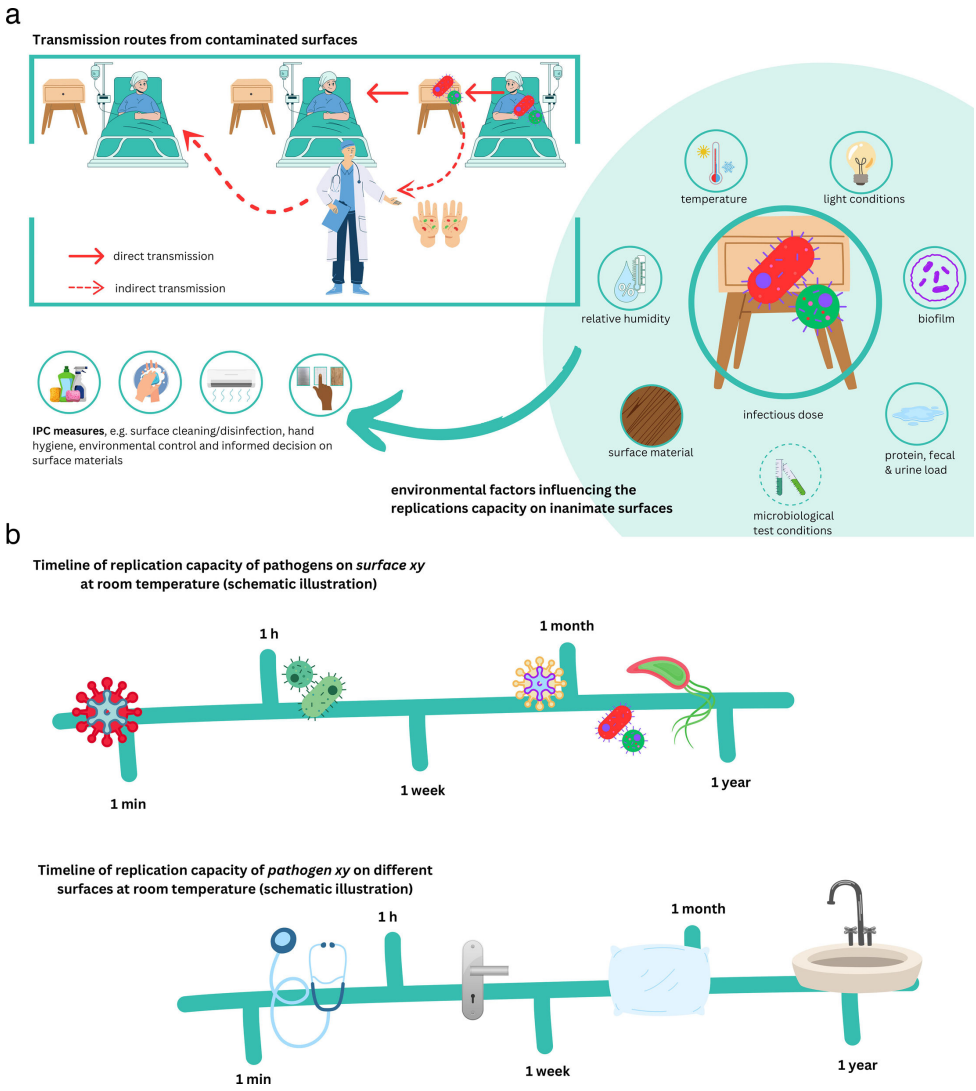

FIG 1 (a) Transmission routes from contaminated inanimate surfaces and environmental influences. (b) Examples of the variety of different replication capacities depending on the pathogen and surface material.

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, 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," that is, "viability," and "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 they 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 (CPE). 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, for example, 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, for example, 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 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 the origin of HAI

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

- To determine the most effective decontamination strategy, first, for known nosocomial pathogens, and second, 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 with 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; and
- To analyze the RC under the influence of probiotic cleaning as a new option for IPC (61).

Walther and Ewald (62) distinguished a highly virulent long-lasting group containing variola (smallpox) virus, *Mycobacterium tuberculosis*, *Corynebacterium diphtheriae, Bordetella (B.) pertussis, Streptococcus (Str.) pneumoniae*, and (avian) Influenza A Virus (virulence determined from mortality rate or case mortality). These pathogens have a mean percent mortality of ≥0.01% and a mean survival time of >10 days (d). By

contrast, a low-virulence and low-persistent group (mean percent mortality <0.01% and time of survival <5 d) includes viruses such as Rubeola, Mumps, Parainfluenza, Respiratory syncytial, Varicella-zoster, Rubella, and Rhinovirus, alongside the bacteria *Mycoplasma pneumoniae* and *Haemophilus (H.) influenzae*. This is even more interesting since these bacteria and viruses belong to totally different species, families, and genera, respectively. While our review focuses on transmission modes *via* inanimate surfaces ["fomite-borne"; e.g., from materials such as glass, stainless steel, textiles (Fig. 1b)], also other transmission modes (e.g., airborne, waterborne/foodborne) of pathogens are relevant for risk assessment. The longer a nosocomial pathogen persists on a surface, the longer the surface may be a source of transmission and endanger a susceptible patient or healthcare worker. Furthermore, a correlation between virulence and persistence is reported (63), the sit-and-wait hypothesis predicts that virulence should be positively correlated with persistence in the external environment because persistence reduces the dependence on host mobility for transfer to a patient. This has been confirmed for respiratory tract pathogens (63). The virulence of pathogens, including factors, such as infectious dose (ID), RC, and risk of transmission, determines the outbreak potential and should be considered as the basis for the IPC strategy. For surfaces as (temporary) origin of HAI, the RC of pathogens from fomites is essential. The main focus in this context was the transmission mode from inanimate surfaces. High virulent pathogens with outbreak potential due to low ID, long-lasting RC require additional to the non-targeted near-patient (high touch) surface disinfection, a targeted cleaning and disinfection as patient-remote (low touch) surface disinfection, and final surface disinfection. Such pathogens with increased "fomite-borne risk," characterized by an increased nosocomial risk for transmission from inanimate surfaces, are marked in gray in Tables 3 to 7. Of course, disinfection measures are only one part of the IPC strategy combined with the other standard precautions such as hand hygiene and additional pathogen-related measures such as barrier nursing, isolation, antimicrobial chemotherapy, and antiseptic decolonization. With growing knowledge, the classification of "pathogens with nosocomial risk for spread from inanimate surfaces" can be further developed.

There is a practical way of looking at this. For example, admission to a room previously occupied by a patient infected and/or colonized with a pathogen is a known risk factor for the acquisition of that pathogen (64). 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 (65). 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 methicillin-resistant *Staphylococcus (S.) aureus* (MRSA), disinfection of the sink/shower for ESBLs and comprehensive air and surface disinfection for *C. difficile*, etc. If pathogens are released from the respiratory tract, knowledge of the RC makes it possible to assess whether patient-remote surfaces should also be included in the final disinfection, for example, 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 the 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. 1a and b). Transmission potential of pathogens on surfaces is not restricted to the direct and indirect contact transmission route. 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 airborne transmissible pathogens.

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

METHOD

Literature from three reviews (66–68), with at least partly similar aims, was screened and examined as a basis for the current review. Then a systematic literature search was conducted in accordance with the PRISMA guideline and the German Manual for Literature Research in Databases (69).

Based on the modified PICO scheme (Table 1), the search terms were compiled. The search was restricted to publications from 2020 onwards to obtain hits that were not already included in the latest review (68). The language was limited to German and English. PubMed and Web of Science were both used for the search, which was conducted on 26th January 2023.

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 the case of different assessments, a third reviewer joined the discussion, and a consensus was reached. First, 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 above.

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

Tables 3 to 7 were modified from the informative appendix (only in German) (71) of the recommendation of the Commission for Hospital Hygiene and Infection Prevention (KRINKO) on Hygiene requirements for cleaning and disinfection of surfaces (72). Table 8 was modified from Jawad et al. (73).

EVALUABLE PUBLICATIONS

There were 145 publications taken from three previous reviews, with an additional 495 records identified *via* the databases (Fig. 2). In all, 152 duplicates were removed. The title and abstract of the remaining 343 records were screened, leading to the inclusion of 40 reports. Of these, 32 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 led to the inclusion of another 22 primary studies. Within the scope of the systematic search, a total of 26 primary studies were included. Together with studies from the three initial reviews, a total of 171 publications were included.

This review does not claim to include all pathogens with the ability to induce outbreaks, for example, *Mycobacterium chimera*. The priority was to gauge transmission potential from near-patient inanimate surfaces. We did not consider pathogens in other hospital hygiene-relevant settings (e.g., water, air, and food).

TABLE 1 Search strategy: segments and search terms

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

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, etc.), commentaries, study protocols, conference abstracts, books, editorials, model studies
Human pathogenic species within the following groups: viruses, bacteria, protozoa, and fungi that are relevant for hospital-acquired infections from surfaces[a]	Other pathogens
Inanimate surfaces—specifically surfaces relevant in hospital settings (e.g., materials such as glass, stainless steel, polymers, textiles). Cave: if the only information found was not on hospital-relevant surfaces, the information is reported to give insight into the possible tenacity of the pathogen.	Animate surfaces (e.g., hands, hair, wounds)
Persistence, tenacity, survival, temerity, recultivable, and 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., inoculum concentration) are given	Relevant data/methodology (e.g., inoculum concentration) not given

[a]Although ectoparasites can also be transmitted nosocomially (70), they were excluded because they are multicellular arthropods reproducing outside humans.

Tables 3 to 7 focus on the most important pathogens in the healthcare setting and the most important parameters for transmission potential (temperature, RH, light, surface material). For better clarity, inocula were reported by waiving application conditions. Due to differences in the choice of units used to report results, the initial inoculum (starting point) was converted into a decadic logarithm. For additional data and details of recultivation and expanded environmental conditions, please see supplementary material (Tables A–E). Pathogens with an increased fomite-borne transmission potential were highlighted in gray. For this tentatively introduced classification, we used a simple scoring system: Pathogens are characterized by (i) a high virulence and/or (ii) a long RC and/or (iii) 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 Fig. 3.

Replication capacity of bacteria

Microorganisms responsible for colonized or infected patients may be isolated from the near-patient environment, especially when surface cleaning or disinfection is inadequate. To clarify transmission routes, screening has been carried out primarily for species such as MRSA (236, 237), vancomycin-resistant enterococci (VRE) (236, 238), carbapenem-resistant enterobacteriaceae (CRE) (239, 240), *Acinetobacter baumannii* complex (241), *Clostridioides (C.) difficile* (241, 242), and recently for the high pathogenic yeast *Candida (C.) auris* (243). For species detected in nosocomial outbreaks, or which frequently colonize or infect newly admitted patients, understanding RC is useful because intensified surface cleaning/disinfection within an intervention bundle has proved effective in controlling cross-infection and even outbreaks. This has been proven for VRE (18, 25), *C. difficile* (16), MRSA (244), *Acinetobacter (A.) baumannii* (4, 8, 22, 28), CRE (14, 25), and *C. auris* (243, 245). The acquisition of pathogens from previous patients caused by deficiencies in final disinfection is well known (5–7, 9, 15, 23, 246) and evaluated in meta-analyses (21, 31). However, none of these studies used genomic surveillance to link isolates from the previous occupant and the new patient admitted into the same room. Recent work suggests that pathogen identity cannot be assumed, but there is a high likelihood of genotypic identity depending on the species (247).

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

TABLE 3 Replication capacity of Gram-positive bacteria from inanimate surfaces[a]

Pathogen	Initial inoculum	Replication capacity	Surface	Ref.
Bacillus subtilis spores	~8 lg CFU	After 15 d: reduction by 0.3 lg, after 56 d: reduction by ~0.7 lg	Glass	(74)
	7.1–9.5 lg CFU	>200 d: reduction by ~2 lg	Polycarbonate	(75)
	6 lg CFU	≥1 d: 5 lg	Stainless steel	(76)
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	(77)
	6–7 lg CFU	After 6 wk: reduction by ~0.5–0.8 lg; after 12 wk: reduction by <3 lg	Steel	(78)
C. difficile veg.	~6 lg CFU	15 min: reduction by ~4 lg	Glass	(79)
Corynebacteria generic	2.7–3.8 lg CFU	≥ 48 h: mean recovery 3.6%	Cotton	(80)
Corynebacterium diphtheriae	Up to 155 CFU	7–90 d (strain-dependent)	Dust	(81)
Corynebacterium pseudotuberculosis	~6 lg CFU	3 d	Plastic	(82)
Corynebacterium striatum	6 lg CFU	After 48 h: 7.7 lg/6.8 lg/2.6 lg	Polyvinyl chloride (PVC)/silicone/stainless steel	(83)
Enterococcus faecium	6–7 lg CFU	After 12 wk: reduction by <3 lg	Steel	(78)
	~6.5 lg CFU	49 d/51 d/49 d	Cotton/wool/silk	(84)
	250 CFU	7 d up to 28 d: 250–70 CFU/250 to ~32 CFU/250–160 CFU/250 to ~50 CFU	Glass/PVC/stainless steel/aluminum	(85)
	8 lg CFU	1 to 16 wk	PVC	(86)
	8 lg CFU	<4 mon: ~2 lg recultivable	Ceramic/PVC/rubber/steel	(87)
	~5 lg CFU	33/>90/>90 d	Cotton/polyester/polypropylene	(88)
	5–6 lg CFU	≥7 d (3 lg/3 lg)	Polyester/Terrycloth	(89)
	10 lg CFU	≥21 d (4–5 lg)	Cotton	(90)
Enterococcus faecalis	6–7 lg CFU	After 6 wk: reduction by <1.8 lg	Steel	(78)
	7.5 lg CFU	After 8 wk: 6.5 lg	Ceramic/cotton/synthetic fibers	(91)
	5.2 lg CFU	After 1 d: survival of 3%	Cotton	(92)
	~5 lg CFU	>90/>90/>90 d	Cotton/polyester/polypropylene	(88)
	6 lg CFU	≥1 d: 5 lg	Stainless steel	(76)
Enterococcus spp.	7.2 lg CFU	Mean survival rate 3 d (dried in water), 43 d (dried in egg white)	Glass	(73)
Vancomycin-resistant *Enterococcus* (VRE)	~6 lg CFU	After 6 wk: reduction by ~3 lg	Steel	(78)
	5 lg CFU	≥7 d	Furnishings	(93)
	E. faecalis 4.5 lg	Dried 60 min: 3 lg CFU; dried 90 min: 3.6 lg CFU	Stainless steel	(94)
	8 lg CFU	1 to 16 wk	PVC	(95)
	E. faecalis: ~5 lg CFU	22/>80/>80 d	Cotton/polyester/polypropylene	(88)
	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	(75)
	5.2 lg CFU	After 2 d: survival of 20%	Cotton	(92)
Mycobacterium tuberculosis	0.1 mg/mL	Recultivable in daylight after 1 d, recultivable in darkness for 9 d, not recultivable after 40 d	Coverslip	(96)
Staphylococcus aureus, methicillin-susceptible (MSSA)	7.3 lg CFU	≥11 d	Glass	(73)
	5.2 lg CFU	After 25 d: survival of 0.8%	Cotton	(92)
	7.5 lg CFU	After 8 wk: ~6.5 lg CFU/mL	Ceramic/cotton/synthetic fibers	(91)
	8 lg CFU	2 d/18 d/>45 d/43 d	Latex/cotton/vinyl flooring/granite	(97)

(*Continued on next page*)

TABLE 3 Replication capacity of Gram-positive bacteria from inanimate surfaces[a] (*Continued*)

Pathogen	Initial inoculum	Replication capacity	Surface	Ref.
	~6.5 lg CFU	37 d/37 d/41 d/37 d	Cotton/cotton polyester/wool/silk	(84)
	6 lg CFU	9 d/10 d/3 d	Formica/stainless steel/enamel	(98)
	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/aluminum	(85)
	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	(73)
	Desiccation: 7.3 lg CFU Wet: 3–4 lg CFU	After 25 d desiccation: 4.4 lg; wet: after 7 d not recultivable	Aluminum	(99)
	6–7 lg CFU	Dry < 7 mon, at 32% RH >5 mon	Dust	(100)
	a. Dry inoculum: 5–6 lg CFU	a. After 24 h: 6.7 lg CFU, after 7 d: 22 CFU /after 24 h: 6.3 lgCFU, after 7 d: 1 CFU		
	b. Liquid inoculum: ~6 lg 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	(101)
	7 lg CFU	≥ 12 d/12 d/≥14 d	Plastic/laminated plastic/polyester	(102)
	5–6 CFU (mattress 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	(103)
	8 lg CFU	<21 d/≥21 d (6 lg)	Polyester/cotton	(104)
	5–6 lg CFU	≥206 d/25 d/11 d/≥206 d	Mattress inner foam/PVC/cotton/polyester	(105)
	9 lg CFU	≥21 d : 4–5 lg CFU	Cotton	(90)
	5.7 lg CFU	≥11 d: 4 lg CFU	PVC	(76)
	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	(106)
Staphylococcus aureus, methicillin-resistant, epidemic (EMRSA)	8.7 lg CFU	≤ 60 min/270 min/≥360 min	Copper/brass (80% Cu, 20% Zn)/stainless steel	(107)
Staphylococcus aureus, methicillin-resistant (MRSA)	6–7 lg CFU	After 6 wk: reduction by 5–6 lg CFU	Steel	(78)
	8 lg CFU	1 d/18 d/41 d/40 d	Latex/cotton/vinyl flooring/tile	(97)
	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	(108)
	9 lg CFU	<318 d	Plastic	(109)
	8 lg CFU	With dust: <126 d; without dust: <175 d	Bottles with and without dust	(101)
	5.6 lg CFU)	<21/14/3/40/>51 d	Cotton/cotton terry/cotton and polyester/polyester/polypropylene	(88)
	~7.3 lg CFU	<96 d	Glass	(110)
	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	(111)
	7 lg CFU	≥12 d/11 d/9 d	Plastic/laminated plastic/polyester	(102)
	6.3–6.7 lg CFU or 4.3–4.7 lg CFU	≤8 d or <2 d	Polypropylene	(112)
	5–6 lg CFU	≥7 d: <1 lg/1 lg	Polyester/terrycloth (towel)	(89)

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TABLE 3 Replication capacity of Gram-positive bacteria from inanimate surfaces[a] (*Continued*)

Pathogen	Initial inoculum	Replication capacity	Surface	Ref.
Staphylococcus aureus, vancomycin intermediate (VISA)	8 lg CFU	1 d/3 d/>45 d/>45 d	Latex/cotton/vinyl flooring/ granite	(97)
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	Aluminum	(99)
Streptococcus pyogenes	~7.7 lg CFU	<2 h	Plastic and ceramic/plastic/ stainless steel	(113)
	8 lg CFU	Planktonic: 3 d; as biofilm: >120 d	Plastic/textiles	(114)
	5–6 lg CFU	≥206 d/25 d/11 d/≥206 d	Mattress inner foam/PVC/cotton/polyester	(105)
Streptococcus pneumoniae	2.8–3.6 lg CFU	≥48 h: mean recovery 0.2%	Cotton	(80)
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	(115)

[a]Table modified from reference 71. Pathogens with fomite-borne transmission potential, characterized by an increased nosocomial risk for transmission from inanimate surfaces, are marked in gray; for additional data and details of recultivation and environmental conditions, see the supplemental material (Table A). Legend: CFU = colony forming units, lg = decadic logarithm, min = minute, h = hour, d = day, wk = week, mon = month, PVC = polyvinyl chloride.

and removed from the germ carrier by different rinsing solutions, for example, dist. water, physiol. NaCl, phosphate-buffered salt solution (PBS), or Triton X-100, sometimes in combination with ultrasound (Tables 3 and 4).

After this preparation, members of the Gram-positive genera enterococci (e.g., VRE) and staphylococci (e.g., MRSA) survive for months on dry surfaces. Among streptococci, RC differs depending on the species, that is, for *Streptococccus (Str.) pneumoniae* <24 h, *Str. pyogenes* 1–3 d, and *Str. salivarius* >88 h. *Corynebacterium pseudotuberculosis* survives 1–4 d on dry plastic surfaces. By contrast, *C. diphtheriae*, isolated from dust in patient rooms, survives 7–90 d, depending on the species. In daylight, *Mycobacterium tuberculosis* survives for 2–5 d, but in darkness, recovery is possible for up to 200 d (Table 3).

There are only a few studies where wild-type and antibiotic-resistant representatives of the same species were compared against each other. For enterococci, VRE has higher RC compared with susceptible enterococci. Similarly, methicillin-sensitive *S. aureus* (MSSA) in dust demonstrated a shorter survival time on surfaces than MRSA (Table 3).

Spores of *Bacillus* and *Clostridioides (C.*) spp. survive for >6 months depending on the material. By contrast, the vegetative form of *C. difficile* drops to the detectable threshold within 15 minutes (min) (Table 3).

An initial comment is that neither Gram-positive nor Gram-negative organisms represent a uniform group regarding recultivation potential from inanimate surfaces (Tables 3 and 4). Some species can survive for a month, such as *Escherichia (E.) coli, Klebsiella* spp.*, Pseudomonas aeruginosa, Serratia marcescens, Enterococcus* spp., *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 (248), *S. paratyphi B* survives in soil up to 259 d (249), and *S. enteritidis* for more than 11 months, whereas *S. typhi* survives only 4 d.

Conversely, Mitscherlich and Marth (250) 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-dependent 35–40 d. The decimal reduction time was about 6 d (251). *Shigella flexneri* persists for 6 d (252). *B. pertussis, H. influenzae*, and *Vibrio cholerae* persist only a few days [(253); Table 3]. Aerosolized *H. influenzae* is characterized by short survival on glass (0.29 d), wood (0.08 d), and fabric (<1 d) (250, 254).

TABLE 4 Replication capacity of Gram-negative bacteria from inanimate surfaces[a]

Pathogen	Initial inoculum	Replication capacity	Surface	Ref.
Acinetobacter baumannii (complex)	~6.5 lg CFU	19 d/19 d/7 d/19 d	Cotton/cotton polyester/wool/silk	(84)
	6–7 lg CFU	After 6 wk: reduction by 4–5 lg	Steel	(78)
	6 lg CFU	11 d/12 d/6 d	Formica/stainless steel/enamel	(98)
	250 CFU	After 28 d: ~112 CFU/~112 CFU/~18 CFU/~20 CFU	Glass/PVC/stainless steel/aluminum	(85)
	7.1–9.5 lg CFU	After 20 d: reduction by about 5.5 lg	Polycarbonate	(75)
	1,200 resp. 1,100 CFU	Biofilm-forming <36 d/non-biofilm-forming <15 d	Glass	(116)
	7.3 lg CFU	3 d	Glass	(117)
	7.3 lg CFU	Up to 33 d	Glass	(118)
	7.3 lg CFU	7–70 d (strain-dependent)	Glass	(119)
	~8 lg CFU)	3–90 d (strain-dependent)	Polystyrene	(120)
	~7.3 lg CFU	<96 d	Glass	(110)
	8 lg CFU	50% of strains mean survival of at least 2 wks (<2 lg recultivable), strain-dependent <4 mon (7 lg recultivable)	Ceramic/PVC/rubber/steel	(87)
	4.1 lg CFU	Dried 60 min: 4 lg; dried 90 min: 3.9 lg	Stainless steel	(94)
	6 lg CFU	≥1 d: 4 lg	Stainless steel	(76)
	7 lg CFU	≥60 d: survival rate: 10%, 40%, 40%	Cotton/plastic/glass	(121)
	5–6 lg CFU	≥7 d: 2 lg/3 lg	Polyester/Terrycloth	(89)
	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	(73)
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	(117)
Acinobacter calcoaceticus anitratus	4 lg CFU	After 1 h: 3 lg	Hardboard	(122)
	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	(92)
Acinetobacter calcoaceticus lwoffii	4 lg CFU/sample	After 1 h: 3 lg CFU	Hardboard	(122)
	5.2 lg CFU	After 7 d not recultivable	Cotton	(92)
Acinetobacter radioresistens	7.3 lg CFU	157 d	Glass	(117)
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	(123)
Campylobacter jejuni	0.1 mL contaminated water from screw coolers	4 h/4 h/7 h/7 h	Aluminum/stainless steel/formica/ceramic	(124)
	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	(125)
	7 lg CFU	≤250 min (4 lg)/≥250 min (3 lg)/<250 min (1 lg)/<180 min	Stainless steel/formica/ceramic/cotton	(126)
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	(85)
Escherichia coli	6 lg CFU	After 48 h: ~1.5 lg/after 24 h: ~1.5 lg	Plastic/carton	(127)
	9 lg CFU	After 100 d: 1 lg	Plastic	(128)

(*Continued on next page*)

TABLE 4 Replication capacity of Gram-negative bacteria from inanimate surfaces[a] (*Continued*)

Pathogen	Initial inoculum	Replication capacity	Surface	Ref.
	7.3 lg CFU	After 7 d (dry): not recultivable; after >28 d humidity	Wood/steel	(129)
	7–8 lg CFU	<120 min	Plastic/wood	(130)
	5.2 lg CFU	After 7 h: not recultivable/after 7 h: survival of 0.8% of CFU	Cotton/glass	(92)
	7.5 lg CFU	After 8 wk: ~6.5 lg CFU/mL	Ceramic/cotton/synthetic fibers	(91)
	7–9 lg CFU	After 2 h: reduction 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	(131)
	8 lg CFU	<4 mon (~ 2 lg recultivable)	Ceramic/PVC/rubber/steel	(87)
	~6.5 lg CFU	45 d/37 d/45 d/45 d	Cotton/cotton-polyester/wool/silk	(84)
	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	(85)
	7.1–9.5 lg CFU	After 6 h: reduction by about 6.5 lg	Polycarbonate	(75)
	7.2 lg CFU	Mean survival rate 1 d (dried in water), 3 d (dried in egg white)	Glass	(73)
	6–7 lg CFU	At 58% RH >8 mon	Dust	(100)
	Desiccation: 6.9 lg CFU Wet: 3–4 lg CFU	After 25 d desiccation: 0.7 lg CFU/cm^2; wet: >12 d	Aluminum	(99)
	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	(103)
	8 lg CFU	<10 d/≥21 d (6 lg)	Polyester/cotton	(104)
	5–6 lg CFU	≥206 d/11 d/7 d/≥206 d	Mattress inner foam/PVC/cotton/polyester	(105)
	2.7–3.2 lg CFU	≥48 h (mean recovery too numerous to count)	Cotton	(80)
	5.7 lg CFU	≥1 d: 2 lg	Vinyl chloride	(76)
	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	
Haemophilus influenzae	6 lg CFU	After 1 h: 99.99% reduction	Aerosol	(132)
	2.8–3.5 lg CFU	≥48 h: mean recovery 1.8%	Cotton	(80)
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	(133)
Klebsiella pneumoniae	5.2 lg CFU	After 1 h not recultivable	Cotton	(92)
	7.5 lg CFU	After 8 wk: ~6.5 lg CFU/mL	Ceramic/cotton/synthetic fibers	(91)
	~6 lg CFU	After 6 wk: ~1 lg	Steel	(78)
	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/aluminum	(85)
	7 lg CFU	After 25 d desiccation: 1.8 lg	Aluminum	(99)
	6–7 lg CFU	At 58% RH >15 mon	Dust	(100)
	3.9 lg CFU	Dried 60 min: 3.4 lg; dried 90 min: 1.8 lg	Stainless steel/plastic	(94)
	5–6 lg CFU	<3 d/<7 d	Polyester/terrycloth	(89)
Listeria monocytogenes	6 lg CFU	After 48 h: ~3.4 lg/~1.2 lg	Plastic/carton	(127)
	7–8 lg CFU	After 180 min: 4 lg	Wood/plastics	(130)

(*Continued on next page*)

TABLE 4 Replication capacity of Gram-negative bacteria from inanimate surfaces[a] (*Continued*)

Pathogen	Initial inoculum	Replication capacity	Surface	Ref.
	6 lg CFU	After 10 d: 5 lg/after 5 d: 1.5 lg	Stainless steel/acrylonitrile butadiene rubber (ABK)	(134)
	9 lg CFU	After 50 d: ~7.5 lg CFU; after 50 d (biofilm): ~7.3 lg CFU	Stainless steel	(135)
	8 lg CFU	After 20 d: 2 lg	Stainless steel	(136)
	7.3 lg CFU (biofilm)	After 21 d: 5.3 lg	Stainless steel	(137)
Neisseria gonorrhoeae	2 x ~ 20 µL Patient exudate (with proven infection)	At least until 24 h recultivable	Plastic/cotton-polyester	(138)
	One 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	(139)
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	(140)
	7.5 lg CFU	After 8 wk: 6.5 lg	Ceramic/cotton/synthetic fibers	(91)
	5.2 lg CFU	After 2 h: not recultivable	Cotton	(92)
	~6.5 lg CFU	13 d/23 d/33 d	Cotton/cotton polyester/wool/silk	(84)
	250 CFU	After 2 d on all surfaces < 2 lg	Glass/PVC/stainless steel/aluminum	(85)
	6 lg CFU	4 d/5 d/1 d	Formica/stainless steel/enamel	(98)
	Desiccation: 6.4 lg CFU Wet: 3–4 lg CFU	After 2 d desiccation: not recultivable; wet: >12 d	Aluminum	(99)
	6–7 lg CFU	At 58% RH >8 mon	Dust	(100)
	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	(103)
	8.7 lg CFU	20 d, 5 d, 4 d	Cotton	(90)
	6 lg CFU	≥ 1 d: 4 lg	Stainless steel	(76)
	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	(93)
Salmonella enteritidis (*S. enterica*)	~5 lg CFU	After 8 h: 2 lg/not recultivable	Plastic/carton	(127)
	7 lg CFU	<1,680 min/≥1,920 min: 1 lg/<480 min/ <240 min	Stainless steel/formica/ceramic/cotton	(126)
	9 lg CFU	*Salmonella chester* after 100 d: 3 lg; *Salmonella oranienburg* >200 d	Plastic	(128)
	~9.3 lg CFU	>48 h	Petri dish	(141)
Salmonella typhimurium	5.2 lg CFU	After 7 h: not recultivable	Cotton cloth/glass	(92)
	3.6 lg CFU	<6 wk	Stainless steel	(142)
	1 µL of overnight cultures inoculated on	ST19: after 1 mon 59.7 ± 12.3 % recultivable; ST313: after 1 mon 13.1 ± 9.6 % recultivable	Plastic	(143)

(*Continued on next page*)

TABLE 4 Replication capacity of Gram-negative bacteria from inanimate surfaces[a] (*Continued*)

Pathogen	Initial inoculum	Replication capacity	Surface	Ref.
	agar and incubated at 25°C			
	Two drops of bacterial suspension	Up to 50 mon	Dust	(144)
	5.2 lg CFU	After 1 d: not recultivable	Cotton	(92)
	6 lg CFU	After 3 d: 2 lg/after 1 d: 1.75 lg	Stainless steel/acrylonitrile butadiene rubber	(134)
	6–7 lg CFU	>30 d: reduction between 3 and 6 lg	Stainless steel	(145)
	7–8 lg CFU	≥28 d: 2–3 lg/≥24 h: 3 lg/≥24 h: 4.5 lg	Tile/wood/carpet	(146)
Serratia liquefaciens	7.2 lg CFU	Mean survival rate 3 d (dried in water), 43 d (dried in egg white)	Glass	(73)
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	(85)
	7.2 lg CFU	Mean survival 12 d (dried in water), 9 d (dried in egg white)	Glass	(73)
	Desiccation: 7.3 lg CFU Wet : 3–4 lg	After 25 d desiccation: 2.6 lg; wet: >12 d	Aluminum	(99)
	5.2 lg CFU	After 1 h: not recultivable	Cotton cloth/glass	(92)
	6 lg CFU	≥1 d: 4 lg	Stainless steel	(76)
Shigella dysenteriae	~5 lg CFU	After 4 h: not recultivable	Plastic/glass/aluminum/wood/textile	(147)
Shigella sonnei	9 lg CFU	≤10 d/≤27 d/≤23 d/≤9 d/≤28 d	Glass/cotton/wood/metal/paper	(148)
	~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 (synthetic resin)	(149)
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	(84)
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	(150)
	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	(151)

[a]Table modified from reference 71. Pathogens with fomite-borne transmission potential, characterized by an increased nosocomial risk for transmission from inanimate surfaces, are marked in gray; for additional data and details of recultivation and environmental conditions, see the supplemental material (Table B). 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 an agar plate, overlaying with agar or smear (Table 5).

Molds 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 mold measurements underline the survival for several months (255, 256). Molds can multiply at an RH of ≥75% at room temperature (RT), which can lead to mold infestation (257). The species *Cladosporium*, *Aspergillus,* and *Penicillium* are the most frequently detected molds on hospital surfaces (258–260). *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 (261). Moreover, Mucorales (*Rhizopus* spp.) recovered from linen were associated with a Mucormycosis outbreak (262, 263) and even survived a certified healthcare laundry process (263). Other Mucorales (*Mucor* spp.) persisted on various materials for weeks (152).

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 (264). *Microsporum canis* has been detected on hospital surfaces (260). 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 (265). In the case of tinea pedis, *T. rubrum* was detectable in 86% of patients and *T. mentagrophytes* in 81% of patients in house dust (266). 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 (267). 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 reservoirs (268). 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 months 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% (154).

Several recent outbreaks have been caused by the new emerging multidrug-resistant *C. auris* (269, 270) which differs from other yeasts and dermatophytes in nosocomial spread (271, 272). *C. auris* is capable of colonizing patients and it can persist in a patient for over a year (245, 273). It can be transmitted through direct contact, for example, hands, but also through indirect contact *via* fomites, such as medical devices, other devices, and surfaces that directly contact the patient (272, 274, 275). 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 to the use of reusable skin-surface axillary temperature probes, suggesting that *C. auris* persisted in the environment and initiated a large outbreak (276). 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 complement a higher risk of colonization and probable outbreak potential, with special regard to pan-resistant strains of *C. auris* (277). *C. auris* is now established in 43 countries across five continents (278).

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 (279). The cultivation techniques for protozoa differ from those for bacteria and fungi, involve highly complex procedures, and depend on the life cycle stage (280, 281). The RC distinguishes between the vegetative stage (trophozoite), and the inactive infectious stage (oocyst or cyst) (Table 6).

The interruption of infection chains is the main strategy 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.

One of the most common causatives for parasitic diarrhea in high-income countries is *Giardia (G.) intestinalis*. It shows also relevant prevalence in middle- and low-income countries and in the United States, it is described as the most common parasitic enteropathy. *Entamoeba histolytica* (Amoebiasis) has the most significant effect in low-income countries and has been globally labeled as the third leading cause of death

TABLE 5 Replication capacity of molds and yeasts from inanimate surfaces[a]

Pathogen	Initial inoculum	Replication capacity	Surface	Ref.
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	(103)
A. flavus	4–5 lg CFU	2 to >30 d/2–20 d/>30 d/8 to >30 d	Cotton/polyester/polyethylene/polyurethane	(152)
	~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	(153)
A. fumigatus	4–5 lg CFU	1 to >30 d/5 to >30 d/>30 d/5 to >30 d	Cotton/polyester/polyethylene/polyurethane	(152)
	~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	(153)
	~6.5 lg CFU	>30 d/>30 d/>30 d/27 d	Cotton/polyester/wool/silk	(84)
A. niger	4–5 lg CFU	3 to >30 d/>30 d/>30 d/2 to >30 d	Cotton/polyester/polyethylene/polyurethane	(152)
	~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	(153)
A. terreus	4–5 lg CFU	2 to >30 d/2 to >30 d/>30 d/12 to >30 d	Cotton/polyester/polyethylene/polyurethane	(152)
C. albicans	4–5 lg CFU	1–3 d/1 d/5–6 d/4–5 d	Cotton/polyester/polyethylene/polyurethane	(152)
	6 lg CFU	<7 d	Stainless steel (dry)/moist agar without nutrients	(154)
	6 lg CFU	Survival after 2 d: ~1%, after 3 d: ~0.2%/0.3%, after 7 d: 0%	Stainless steel/glass	(155)
	~7.5 lg CFU	After 5 d: ~6.5 lg/after 6 h: 5 lg, after 24 h: 0 lg	Aluminum/copper	(153)
	6.5 lg CFU	6 d/6d/12 d/12 d	Cotton/polyester/wool/silk	(84)
	~6.1 lg CFU	6 d	Glass	(156)
	~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	(154)
	~4.8 lg CFU	After 4 d: ~3.5 lg, after 14 d: ~0.4 lg	Plastic	(157)
	8 lg CFU	After 14 d: ~4.3 lg (biofilm formation)	Plastic	(158)
C. candidum	~6.5 lg CFU	21 d/6 d/12 d/6 d	Cotton/polyester/wool/silk	(84)
C. glabrata (*Nakaseomyces glabratus*)	6 lg CFU	Survival after 7 d: ~60%/~90%	Stainless steel (dry)/moist agar without nutrients	(154)
	~4.8 lg CFU	12 d/97 d	Glass/textile	(156)
	~6.5 lg CFU	>30 d	Cotton/polyester/wool/silk	(84)
C. krusei (Pichia kudriavzevii)	4–5 lg CFU	1 d/8 d/3–7 d/4 d	Cotton/polyester/polyethylene/polyurethane	(152)
	~6.5 lg CFU	3 d/6 d/>30 d/21 d	Cotton/polyester/wool/silk	(84)
C. parapsilosis	4–5 lg CFU	9–27 d/27 to >30 d/>30 d/>30 d	Cotton/polyester/polyethylene/polyurethane	(152)
	6 lg CFU	Survival after 14 d: ~1.3%/~4.1%	Stainless steel/glass	(155)
	6 lg CFU	Survival after 7 d: 60%/100%	Stainless steel (dry)/moist agar without nutrients	(154)
	~4.7 lg CFU	After 21 d: ~2.5 lg, after 28 d: 0.4 lg	Plastic	(157)
	~6.5 lg CFU	>30 d	Cotton/polyester/wool/silk	(84)
	~6.1 lg CFU	55 d	Glass	(156)
C. tropicalis	4–5 lg CFU	1–2 d/1–8 d/7–18 d/6–12 d	Cotton/polyester/polyethylene/polyurethane	(152)
	~6.6 lg CFU	3 d/9 d/>30 d/21 d	Cotton/polyester/wool/silk	(84)
	~6.1 lg	8 d	Glass	(156)
Cryptococcus neoformans	~6.5 lg CFU	>30 d	Cotton/polyester/wool/silk	(84)
	~6.1 lg CFU	27 d	Glass	(156)
Fusarium solani	~5.8 lg CFU	After 5 d: ~4.4 lg/after 6 h: ~3.6 lg, after 24 h: 0 lg	Aluminum/copper	(153)
Mucor spp.	4–5 lg CFU	20–24 d	Cotton/polyester/polyethylene/polyurethane	(152)
Paecilomyces spp.	4–5 lg CFU	<1 d/5 d/4 d/11 d	Cotton/polyester/polyethylene/polyurethane	(152)
Rhodotorula rubra	~6.1 lg CFU	40 d	Glass	(156)
	~4.8 lg CFU	205 d	Textile	
Saccharomyces cerevisiae	6 lg CFU	After 48 h: 3.9 lg/1.5 lg	Plastic/carton	(127)
	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	(103)

[a]Table modified from reference 71. Pathogens with fomite-borne transmission potential, characterized by an increased nosocomial risk for transmission from inanimate surfaces, are marked in gray; for additional data and details of recultivation and environmental conditions, see the supplemental material (Table C). Legend: CFU = colony forming units, lg = decadic logarithm, min = minute, h = hour, d = day, wk = week, mon = month.

TABLE 6 Replication capacity of protozoa from inanimate surfaces[a]

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	(159)
Cryptosporidium parvum oocysts	(Oo)cysts	Survival at 25°C: >60 d/>60 d/>60 d	Stainless steel/formica/fabric	(160)
	Oocysts	Recovery at 21°C up to 75 d	Water	(161)
	6 lg/mL oocysts	Recultivation rate after 0 h: 76.3%; after 2 h: 3%; after 4 h: 0%	Glass slide	(162)
	7 lg oocysts	After 30 min: 4.1 lg; after 60 min: 3.2 lg; after 90 min: <3 lg	Stainless steel	(163)
	≥100 oocysts	After 24-h desiccation: no infectivity after 1–4 d	Cryptosporidia-laden calf feces	(164)
Giardia muris cysts	(Oo)cysts	Recovery at 25°C: 45 d/21 d/21 d	Stainless steel/formica/fabric	(160)
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	Textile/plastic	(165)
	Trophozoites	Recultivation rates after 15 min at 26°C: <10%	Water	(166)

[a]Pathogens with fomite-borne transmission potential, characterized by an increased nosocomial risk for transmission from inanimate surfaces, are marked in gray; for additional data and details of recultivation and environmental conditions, see the supplemental material (Table D). Legend: lg = decadic logarithm, min = minute, h = hour, d = day.

from parasitic infections. Another protozoa that shows increasing prevalence all over the world, especially among patients with AIDS and children under 5 years of age, is the *Cryptosporidium* spp. (282, 283). However, there are several other protozoa of relevance for the hospital setting. A number of reports have been published recently describing diarrheal outbreaks caused by *Cyclospora (Cy.) cayetanensis* (284, 285). Another protozoan is *Trichomonas vaginalis* which belongs to one of the most relevant non-viral venereal diseases—although fomite-borne transmission is relatively rare (286).

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 (287). At temperatures below 15°C, *Cryptosporidium* oocysts can maintain high levels of infectivity in water for at least 24 wks (162, 288–290) and up to 120 d in soil (291). 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 (160). Outbreaks of *Cryptosporidium* spp. and *G. intestinalis* generally occur *via* drinking water and food which were inadequately treated to kill or to remove these parasites (292). 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 indirectly *via* surfaces (293). 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 (294).

Survival of anaerobic *Entamoeba* spp. in environments is highly dependent on temperature. Survival was determined in feces and soil at 28°C–34°C for 8–10 d, in water and sewage sludge at 0°C–4°C for 60–365 d, in surface water resp. wastewater at 20°C–30°C for 15 d resp. 10 d (295).

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 months. In fresh or marine waters, oocysts were shown to be viable for at least 4.5 and 2 years, respectively, reviewed by reference (296). To determine the survival dynamics, 2.5 g of soil is 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 (297).

TABLE 7 Replication capacity of viruses after isolation from inanimate surfaces[a,b]

Pathogen	Initial inoculum	Replication capacity/residual virus titer	Surface	Ref.
Predominant contact transmission				
Adenovirus	~7 lg $CCID_{50}$	>12 wk; after 8 wk: 3.4–5.7 lg	Glass/plastic/porcelain/stainless steel	(167)
	2,000 PFU	<49 d; after 14 d: ~8%/~3%	Plastic/aluminum foil	(168)
	~6 lg PFU	15 d/15 d/30 d/>30 d	Aluminum/porcelain/latex/paper	(169)
Adenovirus type 3	~7 lg $TCID_{50}$	>9 d: 4.2 lg	Polystyrene	(170)
Cytomegalovirus	4–6.9 lg PFU	1–2 h/4–8 h	Cotton/plexiglass	(171)
Ebola virus	4–6 lg $TCID_{50}$	At 4°C > 50 d: 2 lg	Plastic/glass/stainless steel	(172)
	7 lg PFU;	6.2 d	Paper	(173)
	7.3 lg PFU	>5.9 d: 4 lg	Glass/silicone/aluminum	(174)
	6–7 lg $TCID_{50}$	14 d/8 d/11 d	Tyvek/stainless steel/plastic	(175)
	7 lg $TCID_{50}$	>192 h/>192 h/<24 h/>192 h; 3–4 lg	Stainless steel/surgical mask/cotton/plastic	(176)
Hendra virus (HeV)	~6.25 lg $TCID_{50}$	60 min, after 30 min: ~2.7 lg	Polystyrene	(177)
Lassa virus	7.1 lg PFU	>9.7 d: 4 lg	Glass/silicone/aluminum	(174)
Mpox	Household setting after disease	At least 15 days: ≤2 lg/0 to ≤ 2 lg	Porous surfaces/non-porous	(178)
Marburgvirus	4–7 lg $TCID_{50}$	>50 d: 2 lg	Plastic/glass	(172)
Nipah virus (NiV)	~6.25 lg $TCID_{50}$	After 60 min: ~2.7 lg	Polystyrene	(177)
Sindbis virus	7.2 lg PFU	>14.6 d: 4 lg	Glass/silicone/aluminum	(174)
Vaccinia virus	7 lg $CCID_{50}$	>4 wk: 2 lg	Glass	(167)
	8 lg $CCID_{50}$	14 wk: 3 lg/up to 10 wk: 3.5. lg	Wool/cotton	(179)
	8 lg $CCID_{50}$/mL	1 wk: 4 lg	Cotton	(180)
	2.8 lg $TCID_{50}$	14 d: <1 lg	Gauze bandage	(181)
	8 lg PFU	<56 d: ~4.5 lg	Stainless steel	(182)
	6–6.5 lg KID_{50}	<20 wk: 4.3 lg	Glass	(183)
Contact transmission, starting from the gastrointestinal tract (+ surrogate viruses)				
Adenovirus type 40	5–5.7 lg IU	>7 d: 3.8 lg	Paper/porcelain	(184)
Astrovirus, serotype 4	5–5.7 lg IU	60 d/after 7 d: 1.7 lg	Paper/porcelain	(184)
Coxsackie virus	6.8 lg $CCID_{50}$	2 wk: 2 lg	Glass	(167)
	6.5 lg $TCID_{50}$	<6 wk	Petri dish	(185)
Echovirus	max. 300 PFU	42 h	Cellulose	(186)
Feline calicivirus	9 lg PFU	>7 d: 2 lg	Laminate/ceramic/stainless steel	(187)
	7 lg $TCID_{50}$	90% reduction in viral titers: up to 24 h	Computer/brass/telephone	(188)
	6 lg PFU	<15 d/<3 d/<7 d	Wool/nylon/glass	(189)
Hepatitis A virus (HAV)	6 lg PFU	>1 mo	Wood/stainless steel	(190)
	3–4 lg PFU	4 h to >7 d	Stainless steel	(191)
	5–5.7 lg IU	After 7 d: ~3.3 lg/~5 lg	Paper/porcelain	(184)
	6.4 lg	After 90 d on PVC: 10% of initial loading	Stainless steel/PVC	(192)
	~6 lg PFU	>60 d/>60 d/>60 d/>30 d	Aluminum/porcelain/latex/paper	(169)
Hepatitis E virus (HEV)	~4 lg FFU	After 28 d: ~1 lg/1 lg/0.4 lg/0 lg	Plastics/ceramics/stainless steel/wood	(193)
	3.9 lg FFU	D value: 5.95 d	Stainless steel	(194)
Escherichia virus (MS2 phage)	6 lg PFU	D value: 19.8 d/13.2 d	Wood/stainless steel	(190)
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	(195)
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	(196)
Poliovirus type 1	4.4 lg PFU	>90 min; after 20 min: 2.6 lg	Worktop	(197)
	~6 lg PFU	3 d/1 d/30 d/>30 d	Aluminum/porcelain/latex/paper	(169)
	max. 300 PFU	42 h	Cellulose	(186)
	~12 lg PFU	>3 wks on all surfaces; 99% reduction after 5.2 d/7.4 d/5.9 d	Steel/cotton/plastic	(198)

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TABLE 7 Replication capacity of viruses after isolation from inanimate surfaces[a,b] (*Continued*)

Pathogen	Initial inoculum	Replication capacity/residual virus titer	Surface	Ref.
	3–4 lg PFU	12 h	Stainless steel	(191)
Poliovirus type 2	8.1 lg PFU	After 14 d: >3 lg	Glass	(167)
	5–5.7 lg IU	>7 d	Paper/porcelain	(184)
Rotavirus	~6 lg PFU	>60 d	Aluminum/porcelain/latex/paper	(169)
	3–4 lg PFU	<90 min	Worktop	(197)
	7 lg PFU	>10 d	Glass/smooth plastic/rough plastic	(199)
	5–5.7 lg IU	>7 d	Paper/porcelain	(184)
Tulane virus (Rhesus enteric calicivirus)	4.7 lg PFU	D value: 18.8 d/13.3 d	Acrylic/stainless steel	(200)
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	(201)
	3 lg PFU	3 d/5 d/≤40 min/120 min/30 min	Silicone/PVC, ceramic, glass, steel/brass/70% copper/90% copper	(202)
	~7 lg $TCID_{50}$	48 h: 2 lg	Polystyrene	(170)
Influenza A virus	3.1 lg $TCID_{50}$ (A/NC-H1N1); 4.8 lg $TCID_{50}$ (A/Br-H1N1)	7 d	Stainless steel	(203)
	5.5 lg $TCID_{50}$	>24 h/>48 h/>24 h/8 h	Stainless steel/wood/plastic/cotton	(204)
	5.3 lg $TCID_{50}$	≥60 min/30 min/15 min/<15 min/<15 min	Cotton/formica/vinyl/stainless steel/facial tissue	(205)
	5 lg $TCID_{50}$	<5 d	Petri dish	(185)
	4–6 lg PFU	After 7.3 d/17.7 h/34.3 h 99% reduction	Stainless steel/cotton/microfiber	(206)
	3–4 lg $TCID_{50}$	48 h/72 h/24 h/24 h/12 h	Plastic/stainless steel/magazine/cotton/paper	(207)
	6 lg PFU	2–9 h	Telephone receiver/wood/keyboard/stainless steel/dishcloth	(208)
	6 lg $TCID_{50}$	<4 h	Stainless steel/plastic	(209)
Influenza B virus	4 lg $TCID_{50}$	48 h/48 h/8 h/12 h/8 h	Plastic/stainless steel/magazine/cotton/paper handkerchief	(207)
Middle East respiratory syndrome coronavirus (MERS-CoV)	6 lg $TCID_{50}$	<72 h	Stainless steel/plastic	(209)
Parainfluenza virus	3.2 lg $TCID_{50}$	4 h	Stainless steel/laminate	(210)
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	(211)
Rhinovirus type 14	7 lg PFU	<25 h; $TCID_{50}$: 0.55 h	Stainless steel	(212)
Rhinovirus type 2	2 lg PFU	After 3 d: ~0.6 lg	Stainless steel	(213)
SARS-CoV-1	6 lg $TCID_{50}$	4 d/4 d/4 d/5 d/5 d	Wood/glass/paper/metal/textile	(214)
	7 lg $TCID_{50}$	28 d: ~2 lg	Plastic	(215)
	3.4 lg $TCID_{50}$	72 h/48 h/8 h/8 h	Plastic/stainless steel/paper/copper	(216)
	6 lg $TCID_{50}$/mL	1 h/24 h/2d	Paper/cotton/disposable gown	(217)
	7 lg $TCID_{50}$	After 13 d: 2.3 lg	Plastic	(215)
	~7 lg $TCID_{50}$	After 9 d: 2 lg	Polystyrene	(170)
	6 lg $TCID_{50}$	4 d/4 d/4 d/≥5 d/≥5 d/4 d	Plastic/wood/glass/metal/cloth/paper	(214)
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	(218)
	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	(219)
	3.6 lg $TCID_{50}$	72 h/48 h/24 h/<4 h	Plastic/stainless steel/cardboard/copper	(216)
	7.8 lg $TCID_{50}$	<3 h/<3 h/<2 d/<2 d/4 d/4 d/<7 d/<7 d/7 d	Paper/handkerchief/wood/clothes/glass/paper/stainless steel/plastic/surgical mask	(220)
	6.2 ± 5.9 lg $TCID_{50}$	13 min at 0.3 W/cm^2: 90% reduction	Stainless steel	(221)
	6.5 lg $TCID_{50}$	<20 min exposed to sunlight	Stainless steel	(222)
	~2.8 lg $TCID_{50}$	≤18.6 h	Stainless steel/plastic/nitrile	(223)

(*Continued on next page*)

TABLE 7 Replication capacity of viruses after isolation from inanimate surfaces[a,b] (*Continued*)

Pathogen	Initial inoculum	Replication capacity/residual virus titer	Surface	Ref.
	5.23 lg $TCID_{50}$	2 d: ~1.2 lg	Glass	(224)
Contact transmission (predominant sexually; also vertically)				
Herpes simplex virus type 1	7.9 $TCID_{50}$	After 2 h: 6.7 lg	Plastic/chrome	(225)
		After 2 h: 5.2 lg		(226)
	5.6 lg PFU	After 1 d: 4 lg	Glass	(167)
	~7 lg $TCID_{50}$	After 9 d: 1.9 lg	Polystyrene	(170)
Herpes simplex virus type 2	4.2 lg $TCID_{50}$	4.5 h: 2.9 lg $TCID_{50}$	Polystyrene	(227)
Human immunodeficiency virus (HIV)	Liquid/dry inoculum: 128,000/25,000 cpm/mL reverse transcriptase	>20 d/~10 d	Petri dish	(228)
Papillomavirus	~100–434 FFU	<7 d	Pipe/cotton/microcentrifuge tube	(229)
Blood-borne transmission				
Hepatitis B virus (HBV)	0.1 mL HBsAg-positive plasma	1 wk	Silanized tube	(230)
	0.1 mL HBV-positive blood	>2 wk	Stainless steel/cotton swab	(231)
	>6 lg $TCID_{50}$	After 28 d: ~10% reduction	PCR tubes	(232)
Hepatitis C virus (HCV)	4–6 lg IE	>40 d	24-well plates	(233)
	~4.75 lg $TCID_{50}$	After 7 d: ~1.5 lg	Stainless steel	(234)

[a]Table modified from reference 71. Pathogens with fomite-borne transmission potential, characterized by an increased nosocomial risk for transmission from inanimate surfaces, are marked in gray; for additional data and details of recultivation and environmental conditions, see the supplemental material (Table E). Legend: cmp = counts per minute, D value = time in which the virus titer is reduced by 1 lg.
[b]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.

Babesia (B.) spp. are intraerythrocytic protozoan parasites transmitted primarily by tick vectors, rare also congenital, and by blood transfusion (298). 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 banks *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 (299). *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 (300). Under normal blood bank conditions, a 35-day-old red cell unit was caused by transfusion-transmitted babesiosis (TTB) (301). Similarly, TTB case reports implicating cryopreserved red cell units indicate that *B. microti* can survive indefinitely in the presence of glycerol cryopreservation (302, 303), but in the absence of cryopreservation, the parasite is rapidly killed by pathogen reduction technology, which uses riboflavin (RB) and ultraviolet (UV) light (304). Theoretically, a single parasite is capable of

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

Average persistence	Strain(s)	Conditions (RH 28%–34%, RT)
≤5 d	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	

[a]Table modified from reference 73.

transmitting infection. Experimental studies, however, have shown that 30 organisms infected about 2/5 inoculated hamsters, and 300 organisms infected all animals (305).

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 (284). Furthermore, climate change with increasing temperatures and heavy rainfall could promote their nosocomial potential in the future. There is also the possibility that HAI could be missed because the incubation period may be days to weeks (wks) 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 (306), but in this estimation, no distinction was made between protozoa and other endo- or ectoparasites. Jarrin et al. (307) assumed that intestinal parasites can cause diarrhea 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 (307).

The 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 (308–310). The 50% infectious dose (ID_{50}) of *C. parvum* has been estimated at 132 oocysts; with some infections followed by ingestion of 30 oocysts (311). Ingestion of at least 10 to 25 *G. intestinalis* oocysts can cause infection in humans (312, 313). Infection after ingestion of a single oocyst has been reported (311). The small ID, the fecal-oral route of transmission, and prolonged environmental survival in water allow *Cryptosporidium* to spread in healthcare facilities as well as child-care centers. *Cryptosporidium* can be transmitted by hand after contact with contaminated environmental surfaces (314). The cysts are highly resistant to environmental conditions and most of the disinfectants commonly used have low or no antiparasitic activity (314). For *Giardia* and *Cryptosporidium* spp., person-to-person transmission is possible (315, 316). For *Cryptosporidium* spp., transmission is primarily found among children and staff members in nurseries, day-care centers, and schools (317). HAI by direct and indirect person-to-person transmission is documented, causing secondary cases among roommates (315). In an outbreak of giardiasis at two day-care nurseries *G. intestinalis* appeared to be transmitted from person to person (318). 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 (319). Therefore, there is minimal risk of nosocomial transmission. Sporulated oocysts of *I. belli* can survive for years in the environment (320). 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 of up to 72 h, and differed between stool samples (162). Survival was shorter than in water because other fecal microorganisms such as bacteria may be associated with the shortened survivability (321), and also with the presence of ammonia, which may occur in feces in high concentrations. Ammonia is a significant inactivation agent for oocysts (322, 323). Oocysts have been shown to survive for hours on wet surfaces, including stainless steel, but they resist desiccation and die rapidly on dry surfaces (324).

One multivariate analysis in a group of virgin females with a high prevalence of trichomoniasis showed that the high prevalence was due to non-sexual acquisition of trichomoniasis, mainly through shared bathing water and inconsistent use of soap (325).

Acanthamoeba is a common protozoa that can be found in diverse environments. Their presence has been documented not only in soil and freshwater but also in

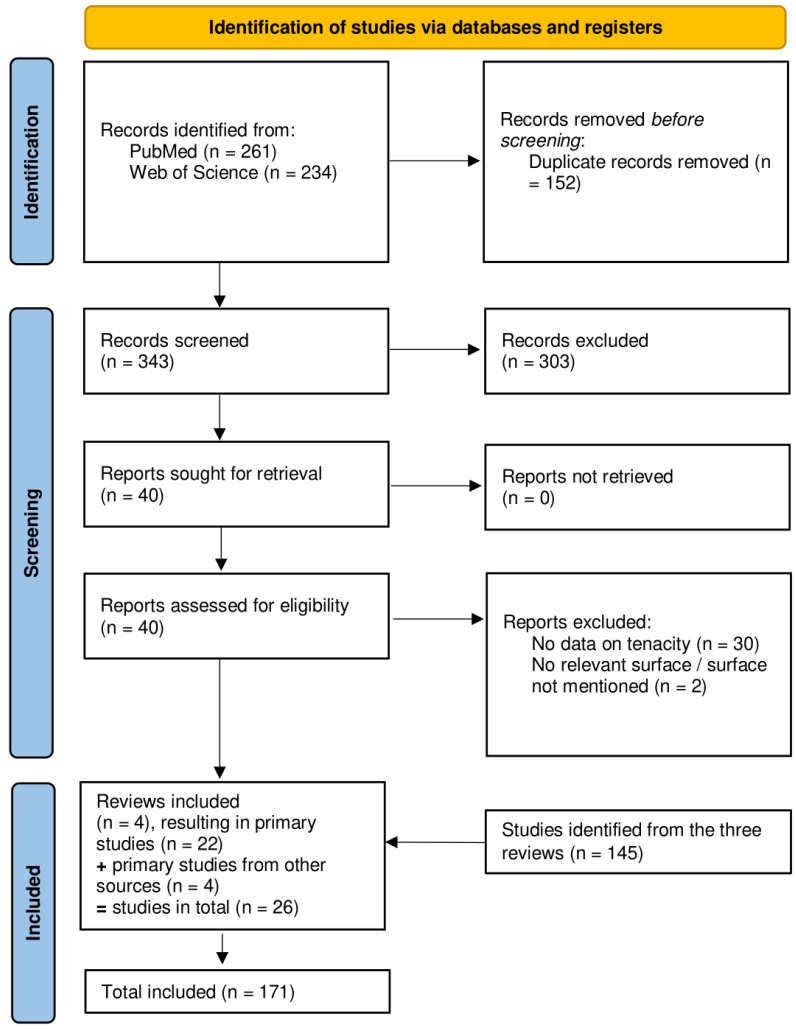

FIG 2 Flow chart (modified from reference 235, published under a Creative Commons license). For more information, visit https://www.prisma-statement.org.

pools, lakes, brackish water, seawater, heating, ventilating, and air-conditioning filters. Moreover, it has been detected on medical devices, such as gastric wash tubing and dental irrigation units (159). Wearing hydrogel contact lenses was associated with keratitis caused by *Acanthamoeba* and *Fusarium* (326), probably due to moist conditions favored by these pathogens. Moreover, the presence of *Acanthamoeba,* together with *Vahlkampfia* and *Vermamoeba* spp., has been verified in the dust of different intensive care wards; on equipment, doors, and in the air-conditioning system (327). With their doubled walls, *Acanthamoeba* cysts are highly resilient, forming dormant stages that remain viable (and infectious) for several years (328, 329) and in a state of desiccation up to 21 years (Table 6).

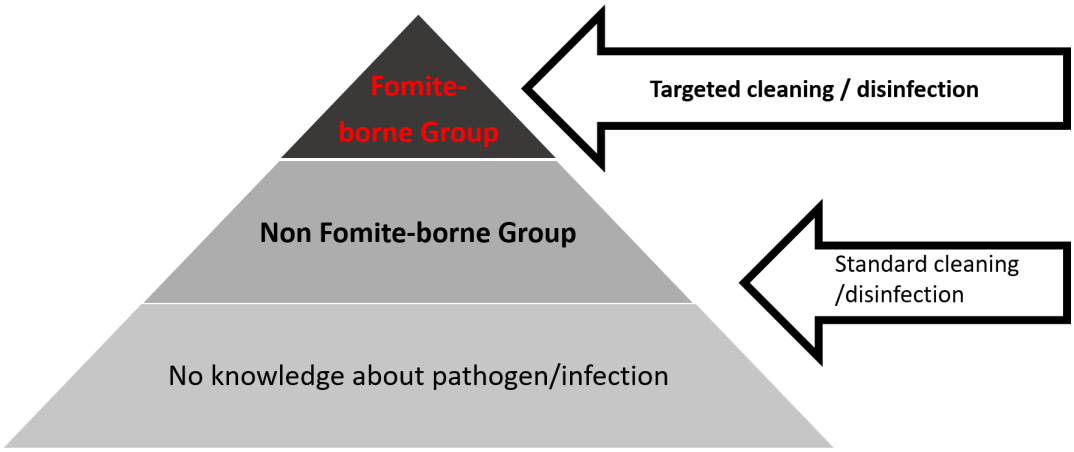

FIG 3 Introduced classification of pathogens with fomite-borne transmission potential and derived IPC strategies.

Replication capacity of viruses

To determine the RC of viruses, applied material was removed from the germ carrier by scraping or rinsing in a 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 titer. By contrast, molecular biological detection alone does not allow any conclusions regarding infectivity. For hepatitis B virus (HBV), infectivity was proven by the application of the rehydrated inoculum in chimpanzees due to lack of cultivation in cell culture in the past. Nowadays, it can be analyzed in an HBV-susceptible cell culture system using hepatoma cells expressing the Na^+- taurocholate co-transporting polypeptide (NTCP)-HBV cell entry factor (232) (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 1–6 w), respiratory (average 1–3 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). By contrast, most enveloped viruses such as herpes viruses (e.g., cytomegalovirus), human immunodeficiency virus (HIV), and respiratory syncytial virus (RSV) are less environmentally stable since they possess an outer lipid bilayer membrane. Small viruses, for example, HBV or the members of the picornavirus or parvovirus family, are much more resistant than larger complex viruses, for example, members of herpes or retrovirus families (330). Some non-enveloped viruses, such as enteroviruses belonging to the picorna viridae, are sensitive to drying, for example, 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 (185).

The relevance of surfaces in healthcare facilities as a contamination source for viruses is even more difficult to prove than for bacteria and fungi, 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's 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 the 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) (331). A large outbreak due to norovirus infections could therefore be controlled by closing the affected departments, implementing extensive disinfection measures, and reducing the exposition risk, that is, from infected healthcare

workers (332). 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 1,106 exposed patients had acquired the identical norovirus strain from the discharged patient (333). Although direct hand transmission dominates nosocomial transmission of rotaviruses, surfaces are also relevant for spread (334). 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 (335). 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 (336). 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 months. HBV belonging to the group of blood-borne or sexually transmitted viruses plays a very high stability with an RC of 50% of more than 22 d at 37°C and a persisting infectivity for up to 9 months at 4°C (232). By contrast, most respiratory viruses retain their infectivity on inanimate surfaces for a few days only (Table 7).

Herpes viruses such as cytomegalovirus are mainly transmitted through contact with infectious body fluids, for example, 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 the summer of 2022, non-travel-associated outbreaks of Mpox have been reported in several non-endemic countries. Human-to-human transmission can occur through close contact with respiratory secretions, infectious skin lesions (such as ruptured blisters) from an infected individual, or recently contaminated objects (e.g., sex toys) and surfaces (337); nosocomial infections have also been documented (338–341). The World Health Organization (WHO) recently recommended adopting the term "Mpox" as a synonym for monkeypox (342). Investigations involving the vaccinia virus, which is related to the MPXV, revealed that it can remain "infectious" on surfaces for up to 56 d (68). Studies on textile fibers showed that the vaccinia virus could be recovered from wool fabric after up to 4 wks and from cotton for up to 8 d; textiles contaminated with virus-laden dust even remained infectious for up to 12 wks (179, 180). Adler et al. found that in some patients the virus could be detected in throat swabs by PCR test for up to 3 wks and in one 2018 case even up to 41 d after diagnosis (343). However, it was not determined whether this represented "residual nucleic acid" or infectious virus. Viable virus was identified in two (50%) of four samples selected for viral isolation, including air and surface MPXV samples collected during bedding change in a hospital in UK (344). 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 suggest that porous surfaces (e.g., bedding, clothing) may pose a higher risk of MPXV exposure than nonporous surfaces (e.g., metal, plastic). Viable MPXV was detected on household surfaces for at least 15 d (178). Therefore, the Centers for Disease Control and Prevention (CDC) recommend minimizing the spread of virus in households by cleaning and disinfecting laundry, hard and soft surfaces, and carpets and flooring when exposed to an infected person (345).

SARS-CoV-2

SARS-CoV-2 illustrates how infection control measures for a new infectious disease can be established, and continuously adapted at breathtaking speed using hospital hygiene strategies including RC, biocide resistance, and transmission considerations. Like other coronaviruses, SARS-CoV-2 has been detected on surfaces (346) showing a correlation between patient proximity and surface contamination (347). Consequently, the risk of further spread due to RC on surfaces of up to 7 d (Table 7) could be prevented by surface decontamination (348, 349). Even simple wiping with hard water or detergent-based cleaning has proven to be an effective decontamination strategy against SARS-CoV-2 (350) applicable to all materials (Table 7), despite variations in their influence on RC (351). Depending on the exposure time, the recoverable virus quantity decreases almost linearly and becomes negligible on plastic after 72 h, stainless steel after 48 h, cardboard after 24 h, and copper after 4 h (352). Since the ID is unknown, the risk assessment remains open. A case report suggests that the detection of SARS-CoV-2 on household surfaces indicates that transmission is possible if surfaces are recently contaminated by coughing or sneezing and then are touched and transferred to the mouth, nose, or eyes (353). However, in other studies where surface transmission was suspected, respiratory transmission could not be entirely ruled out in this study (354). The infection risk is presumed to be low as small amounts of SARS-CoV-2 RNA were detectable in only 2 of 26 samples from an emergency ward and an infectious disease sub-intensive care ward and these did not cause cytopathic effect in cell culture (355). It is possible that residues from surface disinfectants reduced RC. Conversely, there is a possibility that disinfectant residues could induce tolerance. Similarly, quantitative microbial risk assessment (QMRA) studies indicate that the risk of SARS-CoV-2 infection *via* surface transmission is low with a probability of less than 1:10,000 for each contact with a contaminated surface (356–358). These findings suggest that the transmission of SARS-CoV-2 *via* surfaces in public areas is negligible (359). 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 cleaning and disinfection 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 low. In a retrospective questionnaire-based study, it was shown that even at home the use of protective masks and daily use of chlorine- and ethanol-based disinfectants for surface decontamination and hand antisepsis significantly reduced the risk of infection (360). Santarpia et al. (361) deduced from the data that in cases of suspected or confirmed SARS-CoV-2 infection within the last 24 h in the household, surfaces should also be decontaminated.

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

Microbiological test conditions

For bacteria, desiccation on the surface after contamination (rapid or slow), 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 feces, 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, for example, for transmissible gastroenteritis virus, mouse hepatitis (195), and SARS-CoV-2. The latter lost infectivity after 2–4 d (216, 220) compared with longer times of 21 d (219) or 7–28 d (218) for larger inocula (Table 7). Finally, the RC depends on the recovery method (Tables 3 to 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 ((362, 363); Table 7). On porous surfaces, for example, coronavirus, influenza virus, avian metapneumovirus, poliovirus type 1, and human enteric adenovirus type 40 (169, 364), survival is longer than on non-porous surfaces (Table 7). One reason may be the lower virus elution during recovery from porous materials (365). A recently published scoping review draws the same conclusion (366). The capillary effect within the cavities and the faster evaporation of the aerosols could also be influential (367).

RH

Gram-positive bacteria tolerate dry conditions better than Gram-negative bacteria due to cell wall properties (368). *S. aureus* persisted longer at low RH (369), while survival kinetics for *E. faecalis* were lower at 25% RH than at 0% RH (370). *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 (73). 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 (368).

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% (368). Only cytomegalovirus is isolated more frequently from moist surfaces (371). Non-enveloped viruses such as adenoviruses, enteroviruses, and rhinoviruses are replicable for longer at 70%–90% RH [Table 7, (372)].

Temperature

Constant temperatures >24°C seem to reduce the replication and infection capacity of airborne bacteria, as shown for representatives of Gram-positive, Gram-negative, and intracellular bacteria (368). 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 (156). 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 mold species differed significantly across these three temperature ranges (373). The viral genome (viral DNA or RNA) shows especially high sensitivity to the surrounding temperature which influences the RC of some viruses. This is mainly due to their impact and affection not only on the viral genome but also on the viral proteins and the whole enzymatic system. Principally, even though higher temperatures also affect DNA integrity, DNA viruses have more stability than RNA viruses. For certain viruses, including astro-, adeno-, polioviruses, herpes simplex, and HAV, low temperatures (4°C) are associated with longer replicative periods (66). For enteric viruses, RC in water increased with increasing temperature >20°C (374, 375). For rota-, poliovirus, and HAV, RC was higher at >80% RH (169). This was confirmed for poliovirus in that stability was significantly greater at 95% RH than at 25% RH (191). For coronaviruses, the influence of RH was different with higher RC at 20% and 80% and comparatively lower RC at 50% (195). For SARS-CoV-2, interfering substances, temperature (20°C or 35°C), and RH were only of moderate influence (Table 7). Morris et al. (376) developed an original prediction model of how temperature and humidity alter RC using a mechanistic quantitative approach that was based on testing the stability of SARS-CoV-2 on an inert surface for a range of temperature and humidity conditions. SARS-CoV-2 remained infectious longest at low temperatures and extreme humidity (up to 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 65% RH. The model uses basic chemistry to explain why the sensitivity of enveloped viruses increases

with higher temperatures and has a U-shaped dependence on humidity. The model accurately predicts existing results on the influence of temperature and RLF for five different human coronaviruses. This suggests that common mechanisms may influence the stability of many viruses.

Light conditions

Light, especially sunlight, or lack of it influences the RC. The survival time of *C. albicans* and *Rhodotorula rubra* on smooth glass surfaces doubled when they were kept in darkness compared with daylight and extended from 44 to 98 d for *C. albicans* (156). Under the influence of simulated sunlight, 90% of SARS-CoV-2 applied to the surface in artificial saliva were inactivated every 6.8 min during simulated summer exposure, but every 14.3 min during winter exposure (221). By contrast, no significant decrease was detectable within 1 h in the dark [Table 7; (221)]. The effect of sunlight was also reproducible in aerosol, while RH alone (20–70%) had no influence (377). Irradiation (distance 3 cm) with UVC (dose 1.048 mJ/cm^2) completely inactivated SARS-CoV-2 (infectious titer of 5×10^6 $TCID_{50}$/mL) after 9 min, while UVA (dose 292 mJ/cm^2) reduced the titer by only 1 lg after 9 min (378).

Protein, fecal, and urine load

Desiccation in protein-containing media prolongs persistence, for example, for *A. baumannii* (Table 8), *Escherichia (E.) coli* (92), *Neisseria (N.) meningitidis* (379), and yeasts (156). The fecal load had little effect on the RC of HAV and rotaviruses. For adenoviruses, the RC only tended to increase (Table 7).

Biofilm

Several microorganisms form biofilms which is the predominant state of life in nutrient-sufficient habitats. Such life forms lead to more pronounced microorganism adhesion, by which the expression of the so-called sigma factor is triggered. This results in gene activation, making the microorganisms subsequently at least 500 times more tolerable against antimicrobial agents (380) and cold atmospheric plasma (381, 382). Some bacteria such as *K. pneumoniae* can remain viable for up to 4 wks in a dry biofilm, demanding more profound cleaning approaches (383). This may be due to increased tolerance of the production of extracellular substances such as polysaccharides, proteins, and DNA after attaching to surfaces. In addition to biofilm formation under moist conditions, biofilm formation on dry inanimate surfaces at room humidity should also be considered (384). This poses a challenge due to water retention from the biofilm, along with other nutrients, which protects the microorganism itself from various environmental factors (385, 386). This makes biofilms relevant not only for the natural persistence of microorganisms in their native habitats but also for industrial and medical settings (385–387). The RC on inanimate surfaces is prolonged and dependent on environmental aspects, especially humidity. Biofilms have been identified on diverse surfaces in hospitals, that is, on sterile objects, plastic doors, and sanitary areas. Out of these formations, it is possible to cultivate viable bacteria. Available scientific data cannot clarify and elucidate to which extent the risk of transmission and the possibility of cross-transmission is affected by biofilm formation. In the context of multidrug-resistant bacteria, the biofilm could be one additional mechanism for persistence in medical settings (388). Of note, potential intraspecies or interspecies virulence factor exchange may be present in the biofilm (386, 388–390).

The current literature regarding associations between viruses and biofilms is scarce. As viruses are strict intracellular pathogens, they may profit from a prolonged persistence in a reservoir host due to the advantages conferred by the biofilm structure but they will not be able to proliferate (391). Biofilms can contain a range 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 (392). The virion RC in an extracellular context can be promoted by biofilms, both on fomites and aquatic sediments, allowing viral persistence and spread. Therefore, it is necessary to highlight that both virions and virus-infected eukaryotic cells embedded in biofilms can retain their infectivity. The first *in vitro* study provides further information that the enveloped virus herpes simplex virus 1 (HSV-1) and the non-enveloped virus coxsackievirus type B5 (CVB5) can be encompassed within fungal *Candida albicans* biofilms (393). As such viruses stored in biofilms can be depicted as temporary or long-term reservoirs (52). Thus, the viral ability to remain infectious and the potential of fomite-borne transmission can be enhanced by the biofilm, especially due to protection against desiccation and antimicrobial agents (394).

DISCUSSION

The most important difference in this review, compared with the 2006 systematic review (66) on pathogen resilience, is that the course of the RC over time has been calculated based on the quantity of the inoculum on the surface and expressed as log reduction. This has resulted in more accurate values, as well as explaining different values in some cases in the first review. Furthermore, the methodological development of laboratory experiments to determine the RC over the last almost two decades has also influenced overall findings.

In general, good 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 everyday situations (Fig. 1a). Studies using whole-genome sequencing indicate that there is a serious underestimation of transmission events when using standard techniques only (395). These analyses tend to focus on resistant, thus easily recognizable pathogens. However, the quantification of transmission events and thus an appropriate risk assessment are not yet possible.

Beyond the epidemiological evidence, the studies were usually generated under laboratory conditions. This means that not all possible environmental influences in hospital settings can be detected, especially any from antimicrobial residues. In addition, the influence of the simultaneous contamination of hospital surfaces with various nosocomial pathogens, with secretions, excretions, and dirt will also be disregarded. A growing number of studies report that enveloped and non-enveloped viruses can spread in groups in so-called "collective infectious units" (396–398). The vehicles mediating collective spread vary widely and include lipid vesicles, protein matrices, diverse forms of aggregation, and binding to the surface of host or non-host cells (396). It seems reasonable that units like this or interference may also exist for bacteria and/or fungi and/or protozoa. Laboratory studies do not reflect the clinical situation and represent probably an one-sided worst-case scenario assessing the upper bound of infection risk. Furthermore, they cannot represent the complexity of real-life scenarios. When assessing factors that influence the RC, it must be considered that the results only apply to the species investigated and cannot be generalized. Even more so, resistant isolates are often analyzed compared with wild-type variants. Sometimes tested microorganisms are poorly characterized so cannot determine the extent of generalizability. Furthermore, it should be noted that data on the RC are often not median values; the maximum was detected and described and these results can, and should, be used as an upper bound approach. Data suggest that no general prediction about RC independent of the genus is possible.

In addition, further influences must be considered. First, the dependence of environmental conditions on the RC has not yet been sufficiently studied under real-life conditions. Second, there is insufficient data on the behavior of wild-type and/or sensitive strains and variants within a species. Third, no data exist, on whether certain virulence or RC determinants are genetically present in isolates that are particularly well adapted to the hospital setting.

In this review, only the risks due to direct or indirect contact transmission from inanimate surfaces were addressed, not the additional risks by potential aerosolization of pathogens from fomites (399–401). Therefore, it should be considered that the RC in aerosols can be significantly lower than on surfaces, as has been proven for different variants of the Ebola virus and Marburg virus (402). It is also the case that high inocula results in longer survival times due to the logarithmic death curve (403), which has been proven for various bacterial species (88, 404) and or fungal spores (156) on surfaces. Considering all background factors, data generated under laboratory conditions can only provide a rough orientation. In case of doubt, the less favored situation should be assumed when evaluating the data in Tables 3 to 7.

Despite knowledge of the 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, for example, 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 (405–407). 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 of $>10^2$ 50% tissue culture infectious dose ($TCID_{50}$), infection by aerosolization from surfaces is unlikely. By contrast, infection is possible *via* the surface-finger-eye route for *keratoconjunctivitis epidemica* due to 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 of $<10^1$ $TCID_{50}$, 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 of $<10_2$ 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 of acquiring an infection by direct or indirect contact with the surface or by aerosolization from the surface and following respiratory exposure. Likewise, the risk of an outbreak emanating from surfaces increases. In both cases, the ID is likely to have a greater influence. At the same time, the risk of a fomite-borne HAI is influenced by the patient's immune status. The ID, RC, and immune status must be considered when deciding upon targeted surface disinfection and additional IPC.

Disinfecting surfaces in hospitals is generally accepted as a key component of infection prevention (32–35, 72, 424–427). However, 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 toward 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 (428, 429). SARS-CoV-2 was reduced significantly more by probiotic cleaning than by chemical disinfection (430). In non-intensive care units, routine surface disinfection did not prove superior to soap-based or probiotic cleaning in terms of preventing HAI (61). Of course, no evidence-based practical approach for systematic surface or probiotic cleaning in hospitals can be derived from the RC of nosocomial pathogens.

TABLE 9 Minimal infectious dose of selected pathogens

Infectious dose	Application	Pathogen	Reference
1–100 virus particles, CFU resp. oocysts	Oral	Noro-, rotavirus, EHEC, ETEC, *C. difficile*, MRSA, *Cr. parvum, G. intestinalis*	(67, 311, 331, 370, 408–412)
6.6 virus particles	Inhalative	Adenovirus type 4	(331)
10–100 virus particles	Oral	HAV	(413)
30–40 $TCID_{50}$	Intranasal	RSV	(331)
6/71 $TCID_{50}$	Intranasal/oral	Coxsackievirus A21	(331)
0.03/>10^1–10^4 $TCID_{50}$	Intranasal/inhalative	Rhinovirus, different serotypes	(331)
<10^3 CFU	Oral	*Acinetobacter* spp., *C. jejuni*, *Klebsiella* spp., VRE	(67, 414)
≥10^3 spores	Chorio-allantois-membrane hen egg (equivalent to eye contact)	*Lichtheimia corymbifera*	(415)
≥10^3 CFU	Oral	*Salmonella enteritidis*	(416)
≥10^3 $TCID_{50}$	Oral	Echovirus	(331)
>10^3 $TCID_{50}$	Inhalative	Influenza A virus (H3N2)	(331)
>10^3 LD_{50}	Intranasal	Congo Basin MPXV	(417)
≥10^4 CFU	Conjunctival	*P. aeruginosa*	(418)
≥10^4 to ≥10^7 $TCID_{50}$	Inhalative	Influenza B virus	(331)
≥10^4 spores		*Rhizopus* spp., *A. fumigatus*	(419, 420)
10^5 $TCID_{50}$	Conjunctival	RSV	(331)
≥10^5 CFU	Intravenous	*C. albicans, C. auris*	(421)
≥10^5 spores	Parenteral	*Rhizomucor pusillus*	(419)
>10^5 CFU	Oral	*E. coli, S. aureus*	(422)
>10^5 LD_{50}	Intranasal	West African MPXV	(417)
>10^6 $TCID_{50}$	Oral	Adenovirus	(331)
>10^6 to >10^7 $TCID_{50}$	Inhalative	Influenza A virus (H1N1)	
>10^8 CFU/mL	Intraperitoneal	*P. aeruginosa*	(423)
>10^{10} CFU/mL		*S. aureus*	

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 (or terminal) disinfection after patient discharge, all potential pathogen reservoirs must be eradicated with the 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 (431). Analogous to the 5 moments of hand antisepsis (432), 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, (i) disinfecting surface cleaning on the work surface before performing aseptic activities, (iii) final disinfecting of surfaces 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 (431).

This review can 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, that is, 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 to emulate endemic settings. There is insufficient evidence regarding the impact of contaminated surfaces in encouraging contact-free transmission risk. Further analysis should cover aspects of ecological sustainability and should weigh up the potential benefit for transmission and infection events against the additional ecological footprint from resource consumption, production, and waste management.

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ADDITIONAL FILES

The following material is available online.

Supplemental Material

Supplemental Tables A to E (CMR00186-23-S0001.docx). Additional data and details of recultivation and expanded environmental conditions.

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AUTHOR BIOS

Axel Kramer is a medical specialist in hygiene and environmental medicine. From 1990 to 2021, he was the head of the Institute of Hygiene and Environmental Medicine, University Medicine Greifswald and he is still working as a consultant. Since 1990, he has been a founding member of the German Society of Hospital Hygiene (president until 2010); since 1993, he has been a member of the Commission of Hospital Hygiene and Infection Control at the Robert Koch Institute Berlin. From 1994 to 1998, Professor Kramer was vice-dean of the Medical Faculty, at University Greifswald. Since 2006, he has been an Editor-in-Chief of GMS Hygiene and Infection Control. He coordinated several networks, founded by the Federal Ministry of Education and Research: Center of Innovation Competence Plasma—Tissue Interactions (2007–2014); Health Region Baltic Coast—coalition against multi-resistant pathogens (2010–2016); infection prevention of multi-resistant pathogens by UVC irradiation (2019–2021); inactivation of SARS-CoV-2 by UVC light (2021–2024). His work includes 535 research articles, 41 books, 54 patents, and a poetry book "Questioning."

Franziska Lexow completed her studies in veterinary medicine (University of Veterinary Medicine Hannover) in 2011 and wrote her doctoral thesis on biocompatibility studies for implant materials in middle ear surgery. Moreover, she successfully completed Graduate School in "Biomedical Engineering" funded by the German Research Foundation (Collaborative Research Centres SFB599) (2011–2015). Since 2020, Dr. Lexow has been a scientific associate at the Robert Koch Institute (RKI), Department for Infectious Diseases, Unit 14 Hospital Hygiene, Infection Prevention and Control, and working at the scientific office of the Commission for Hospital Hygiene and Infection Prevention (KRINKO). Her focus topic is the development of recommendations on IPC strategies. So far, she has written six research articles and one conference abstract.

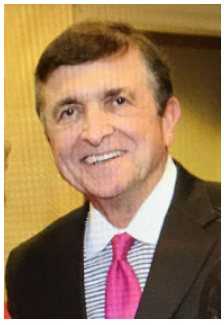
Anna Bludau completed a master's of Public Health and has been a researcher at the Department of Infection Control and Infectious Diseases at the University Medical Center Göttingen, Germany since 2020. Since 2023, she has been a doctoral student (Dr. of Public Health) at the University of Bielefeld, Germany. Since 2023, she has been a member of the German Society for Hygiene and Microbiology and since 2024 a member of the German Public Health Association. Since 2022, she coordinated three projects in the network university medicine, funded by the German Federal Ministry of Education and Research (genomic pathogen surveillance and translational research—GenSurv; molecular surveillance and infection chain tracing for local public health authorities—MolTraX; genomic pathogen surveillance and translational research plus—GenSurv+). So far, her work includes 10 research articles and 8 conference abstracts.

Antonia Milena Köster has a Bachelor of Arts in Social Work (University of Applied Sciences and Arts Hannover) and is a licensed quality manager with a master's degree in organization and leadership (Malmö University). She was in charge of the leadership of an elderly care home from 2016 to 2020. Since 2020, she has been a scientific associate at the Department of Infection Control and Infectious Diseases, University Medical Center Göttingen, and working on small- and large-scale research projects, and for the Commission for Hospital Hygiene and Infection Prevention (KRINKO). Her focus topics are hygiene and sustainability. Her scientific work includes three research articles so far.

Martin Misailovski completed his secondary education as a medical laboratory technician at Skopje's Secondary Medical School and graduated with a medical degree from the University "St. Kiril und Metodij" in 2019. He obtained a master's degree in Cardiovascular Science with a focus on Mass Spectrometry from Georg-August University of Göttingen. Currently, he is pursuing a doctorate (Dr. med.) in Infectious diseases and infection control/prevention and working as a medical doctor at the Department for Infectious Diseases and Infection Control, and the Department of Geriatrics. He is an Associate Fellow at the International Organization for Health Professions Education (AMEE). Moreover, he is a member of the German Society for Hygiene and Microbiology and a member of the European Society of Clinical Microbiology and Infectious Diseases.

Ulrike Seifert is a medical specialist in hygiene and environmental medicine as well as in biochemistry. She has venia legendi in immunology since 2019. Since 2016, she has been a professor and head of virology at the Friedrich Loeffler-Institute of Medical Microbiology, University Medicine Greifswald. Since 2005, she has led a research working group with a focus on the "Ubiquitin-Proteasome-System in infection and disease" and is a principal investigator in several Consortia funded by the German Research Foundation (Collaborative Research Centres SFB421, SFB-TR84, SFB-TR36, SFB854, and Research Training Group 2719-PRO). Since 2022, she has been a member of the section Antiseptic Stewardship of the German Society of Hospital Hygiene. So far, her work includes 47 research articles, 5 review articles, 2 case reports, and 9 book chapters.

Maren Eggers is a specialist in virus hygiene and clinical virology. Since 1997, she has been the head of the virology and disinfectant testing department, Labor Prof. Dr. G. Enders MVZ GbR and Lecturer at the Medical Ruprecht-Karls-University Heidelberg. Professor Eggers is a member of CEN/TC216 WG 1 of the European Committees for Standardization (Chemical disinfectants and antiseptics), and responsible for the virucidal task group since 2018 and chairwoman of the Commission for Virus Disinfection of the German Association for the Control of Virus Diseases (DVV) and the German Society for Virology, on the Board of Association for Applied Hygiene (VAH) and a member of the VAH disinfectant commission since 2011. Moreover, she is a member of the virucidal working group at the Robert Koch Institute (RKI). So far, her scientific work includes 62 research articles, 21 books, 4 European standards, and 2 national guidelines for virucidal testing.



William Rutala is the Director and co-founder of the Statewide Program for Infection Control and Epidemiology and a Professor for the Division of Infectious Diseases at the University of North Carolina's School of Medicine. He was the Director of Hospital Epidemiology, Occupational Health and Safety Program at the University of North Carolina Hospitals for 38 years before retiring in May 2017. He is a retired Colonel with the U.S. Army Reserve and is certified in infection control. He has written more than 700 publications (e.g., peer-reviewed articles, books, book chapters) in the fields of disinfection, sterilization, the healthcare environment, and prevention of healthcare-associated infections. Dr. Rutala has been an invited lecturer at over 400 state, national, and international conferences (>40 states, >40 countries). Dr. Rutala earned his BS from Rutgers University, his MS from UT, and both his MPH and PhD in microbiology from the UNC School of Public Health.

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