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MINIREVIEWS

Stability and infectivity of coronaviruses in inanimate environments

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Abstract

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a highly contagious virus that can transmit through respiratory droplets, aerosols, or contacts. Frequent touching of contaminated surfaces in public areas is therefore a potential route of SARS-CoV-2 transmission. The inanimate surfaces have often been described as a source of nosocomial infections. However, summaries on the transmissibility of coronaviruses from contaminated surfaces to induce the coronavirus disease 2019 are rare at present. This review aims to summarize data on the persistence of different coronaviruses on inanimate surfaces. The literature was systematically searched on Medline without language restrictions. All reports with experimental evidence on the duration persistence of coronaviruses on any type of surface were included. Most viruses from the respiratory tract, such as coronaviruses, influenza, SARS-CoV, or rhinovirus, can persist on surfaces for a few days. Persistence time on inanimate surfaces varied from minutes to up to one month, depending on the environmental conditions. SARS-CoV-2 can be sustained in air in closed unventilated buses for at least 30 min without losing infectivity. The most common coronaviruses may well survive or persist on surfaces for up to one month. Viruses in respiratory or fecal specimens can maintain infectivity for quite a long time at room temperature. Absorbent materials like cotton are safer than unabsorbent materials for protection from virus infection. The risk of transmission *via* touching contaminated paper is low. Preventive strategies such as washing hands and wearing masks are critical to the control of coronavirus disease 2019.

Key words: Severe acute respiratory syndrome coronavirus 2; Coronavirus disease 2019; Inanimate surface; Infectivity; Survival; Transmission

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Core tip: Survival time of severe acute respiratory syndrome coronavirus on inanimate surfaces varied from minutes to up to one month depending on the environmental conditions.

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INTRODUCTION

The coronavirus is a category of enveloped, single-stranded, positive-sense RNA viruses. In the 21st century, the severe acute respiratory syndrome coronavirus (SARS-CoV) and the middle east respiratory syndrome coronavirus (MERS-CoV) both caused worldwide epidemics with high morbidity and mortality[1-3]. Recently, the SARS-CoV-2 that emerged in Wuhan, China in December 2019 has spread globally with alarming infections and deaths[4-10]. The disease caused by SARS-CoV-2 is named as coronavirus disease 2019 (COVID-19)[11], which is also a highly contagious disease[9,12,13]. As of April 11, 2020, there have been 83 400 confirmed infections, 3 349 fatalities in China, and 1 623 873 confirmed cases with 99 617 deaths in 209 other countries[14,15]. The SARS-CoV-2 is presumed to be transmitted by respiratory droplets, viral aerosols, close contacts, and self-inoculation to nose, mouth, or eyes after touching a contaminated surface[16]. However, there are still many controversies about the airborne transmission and infectivity of SARS-CoV on inanimate surfaces. We therefore reviewed the persistence and infectivity of viruses on inanimate surfaces to provide clear information for containing the epidemic of SARS-CoV-2.

SEARCH STRATEGY

The references were systematically searched and reviewed on the homepage of the National Library of Medicine and the web of science without language limitations. The search covered all years available on the internet. The search terms were applied as follows: SARS-CoV-2, SARS-CoV, coronavirus, corona virus, 2019 nCoV, COVID-19, survival, viability, transmission, persistence, surface, and persistence hand. The citations in each article found during the main search were researched for potential relevance. Published articles were included and reviewed, and the related results were extracted given that they provided original data on coronavirus persistence. Data of commercial products based on types of biocidal agents were excluded. Two reviewers screened all identified records against the protocol, undertook the risk of bias assessments, and extracted data using a piloted form.

RESULTS AND DISCUSSIONS

Are there any viruses persisting in the air?

In normal conditions without epidemic, the air should be free of any viruses. Upon SARS-CoV-2 outbreak, when COVID-19 patients cough, sneeze, or even talk, they could shed SARS-CoV-2 into the air, and air transmission may exist within 1 m in close proximity of COVID-19 patients. Thus, the public worry about the possibility of SARS-CoV-2 flowing in the air.

Ong *et al*[17] in Singapore collected air and surface samples from COVID-19 patients bedrooms before and after routine cleaning. Before routine cleaning, reverse transcription-polymerase chain reaction (RT-PCR) results were positive with 13 (87%) of 15 room sites (including air outlet fans) and 3 (60%) of 5 toilet sites (toilet bowl, sink, and door handle), suggesting that the ward environment was extensively contaminated and that viral shedding in stool could be a potential route of transmission. Anteroom and corridor samples were negative. One COVID-19 patient

had upper respiratory tract infection with no pneumonia or diarrhea, and his two stool samples were positive for SARS-CoV-2 on RT-PCR. After routine cleaning of the room, samples collected from another two patients were negative. Only one swab from the surface of a shoe front was positive. All air samples were negative[17] despite the extent of environmental contamination. Swabs taken from the air exhaust outlets tested positive, suggesting that small virus-laden droplets may be displaced by airflows and deposited on equipment such as vents[17]. The risk of transmission from contaminated footwear is likely low, as evidenced by negative results in the anteroom and clean corridor.

Jang *et al*[18] studied 28 air samples and 130 surface samples from isolated wards, outpatient fever clinics, guiding service Intensive Care Units, and nurse stations. RT-PCR results show that only one air sample from Intensive Care Units (1/28, 3.57%) and one surface sample from nurse stations (1/130, 0.77%) were positive for SARS-CoV-2. Tracheal intubation was consistently associated with an increased risk of SARS-CoV transmission among healthcare workers[19]. In 18 hospitalized patients with PCR-confirmed SARS-CoV-2 infection, viral shedding from the nasopharynx was prolonged for 7 d or longer (15/18, 83%). Virus was detectable in the stool (4/8, 50%) and blood (1/12, 8%) by RT-PCR[20].

Significant environmental contamination by COVID-19 patients through respiratory droplets and fecal shedding[17,20] suggests that the environment is a potential medium of transmission and supports the need for strict adherence to environmental and hand hygiene.

To investigate the possibility of SARS-CoV transmission in the air[21], air samples were obtained from seven wards and balconies in hospital at a frequency of three times daily for three continuous days. The N protein gene of SARS-CoV was amplified with RT-PCR, and 29.03% of air samples in the wards and 20.0% of samples in balconies were positive for SARS-CoV; sequential analysis of the positive samples showed that the identity of amplified cDNA fragments to the known SARS-CoV strains was 98%. A strain of live pathogen was isolated and could cause typical cytopathic effects, similar to that of SARS-CoV on Vero-E6 cells, and the effects could be stably passed[21]. This study indicates that SARS-CoV could exist in the air where SARS patients live, but the infectivity of SARS-CoV in air samples is very weak.

Viral nucleic acids, as well as viable seasonal and avian influenza viruses, have been detected in aerosols in the air in healthcare settings or in buses. The viral RNA of respiratory syncytial virus (RSV) was recovered in the air in pediatric or adult ambulatory care clinics, while only a small percentage of them were in particles ≤ 5 μm[22]. A recent report in China shows the clustered aerosol transmission of SARS-CoV-2 in buses[23]. Infected with SARS-CoV-2 but unaware of that, patient zero travelled in a fully enclosed air conditioned bus from place A to B. During his two-hour journey on the bus, he had infected eight passengers, one of which was asymptomatic. At place B, patient zero commuted to another bus right away, and infected two of 12 passengers during this one-hour travel. The bus was not disinfected at the bus station at place B, and 30 min later, shuttled more passengers back to place A. Among them, a passenger sat beside the seat where patient zero was previously seated, and was confirmed to be infected 2 d later, suggesting that the infectivity of SARS-CoV-2 on inanimate surfaces or in aerosol flows in closed buses may last at least 30 min. The distance from patient zero to his infected persons is 0.5-4.5 m. Patient zero and all of the 11 infected passengers in the bus did not wear masks, of which two infected passengers infected two other persons. Thus, patient zero caused a total of 13 human SARS-CoV-2 infections[23]. Another study found infection of RSV virus from the air obtained over 1 m away from RSV-positive hospitalized infants, which were still present 2 h after the infected infants were discharged[24]. Yip *et al*[25] studied the air samples emitted by 16 patients infected with influenza virus. The air samples adjacent to these patients (0.5-1.0 m and 2.1-2.5 m away) were collected using bio-aerosol samplers, respectively. 37.5% of the air samples emitted by these patients were tested positive in viral RNA for all tested particle sizes (< 1, 1-4, and > 5 μm), which indicates that the viruses exist in the air inclose proximity to the patients. There was no correlation between the nasal viral load of the patients and viral RNA recovery from the air, and there was no significant association between RNA detection from the air and demographics or clinical presentation[25].

Air samples collected from patient wards and swab samples from frequently touched surfaces in wards and nurse stations were positive by PCR, indicating that the aerosols bearing SARS-CoV-2 could exist in the air temporarily, especially in poorly ventilated areas, such as buses[23] and wards[18]. The half-life of coronaviruses in aerosols was reported to be 86 h at 80% humidity in the environment[26].

Do respiratory viruses persist in the air in crowded public spaces?

International and national traveling in airports or subways have facilitated the rapid

spread of respiratory infectious diseases such as COVID-19. A relatively prolonged incubation period of SARS-CoV-2 allows travelers with covert coronavirus infection to spread COVID-19 globally. Air transportation appears important in accelerating and amplifying viral transmission aboard airplanes, at the destination, and possibly at airports[27].

In 2018, a pilot study was performed to detect respiratory viruses in Singapore's Mass Rapid Transit network[28]. A total of 89 aerosol samples were collected during peak Mass Rapid Transit ridership hours using noninvasive bioaerosol samplers over 52 wk and tested using real-time RT-PCR: nine (10%) tested positive for adenovirus, four (4.5%) tested positive for RSV type A, and one (1%) tested positive for influenza A virus (IAV). These data show the presence of viruses in crowded public spaces[28].

In Finland from 2015-2016[8], surface and air samples were collected weekly at three different time points during the peak period of seasonal influenza. Swabs from surface samples and air samples were tested by real-time PCR for influenza A and B viruses, RSV, adenovirus, rhinovirus, and coronaviruses (229E, HKU1, NL63, and OC43). The nucleic acid of at least one respiratory virus was detected in 10% (9/90) of surface samples from multiple places, such as children's play grounds, security check areas, buttons of payment terminals, passenger side desks and handrails of stairs. The ten respiratory viruses identified from surfaces and air samples at various sites were rhinovirus (4/10, 40%), coronavirus (3/10, 30%,), adenovirus (2/10, 20%, one air sample, one surface sample), and influenza A (1/10, 10%). The detection of viral nucleic acids indicates surface contamination on multiple sites in airports associated with high touch rates, such as plastic security screening trays, which are almost inevitable for all embarking passengers to touch[8].

Lei *et al*[29] built a model to simulate outbreaks of influenza A H1N1, SARS-CoV, and norovirus in a similar cabin environment. The simulation results suggest that passengers within two rows of the index case had a significantly higher infection risk than others in the outbreak. For SARS-CoV, the airborne, close contact, and fomite routes contributed 21%, 29%, and 50% transmission, respectively. The close contact route was significant in the in-flight transmission of influenza A H1N1. For norovirus, the fomite route played the dominant role in most cases[29].

Viruses exist in a wide range of aerosol particle sizes

Clinically stable asthma patients have similar detection rates of respiratory viruses in samples from the nasopharynx, sputum, and exhaled air[30]. The World Health Organization considers disease transmission with particles ≤ 5 μm as aerosol transmission[31]. When pathogens become airborne, they travel in particles of different sizes and compositions. Particle size determines the distance across which pathogens can be transported, as well as the site of deposition and the survivability of the viruses. The concentration and size distribution of inhalable particles that transport IAV as well as the viral viability for each particle size range were assessed on experimentally infected pigs, including samples from porcine reproductive and respiratory syndrome virus and porcine epidemic diarrhea virus exhaled by acutely infected pigs. An Andersen cascade impactor being able to separate particles by size (0.4-10 μm in diameter) was used to collect aerosols from experimentally infected pigs for 24 d. Infectious status was demonstrated for the three viruses. Quantitative RT-PCR results indicated that airborne porcine epidemic diarrhea virus, IAV, and porcine reproductive and respiratory syndrome virus can be found in a wide range of particle sizes. The virus viability is particle size-dependent[32].

Survival of coronavirus on inanimate surfaces in households or hospitals

A dry or less humid environment is unsuitable for viral survival. During the SARS-CoV-2 outbreak, the contamination of paper documents, paper money, and mail wrapping paper is a concern for people who handle such documents in their daily work. After visiting hospitals and returning home, people worry that their cotton clothes or impervious gowns may carry fomites bearing SARS-CoV-2 into home.

Human coronavirus strain 229E (HuCoV-229E) can remain infectious on inanimate surfaces at RT from 2 h to 9 d on different types of materials[33] (Table 1). At 21 °C and RH of 30-40%, inoculums of 10^3 plaque-forming units (PFU) of HuCoV-229E persisted on polyfluorotetraethylene (Teflon), polyvinyl chloride (PVC), ceramic tiles, glass, and stainless steel for at least 5 d, as well as on silicon rubber for 3 d.

Infectivity of HuCoV- 229E was undetectable after drying on aluminum, sterile latex surgical gloves, and sterile cotton gauze sponges at RT for 3 h; HuCoV OC43 survived 1 h or less[34]. Contaminated droplets will be absorbed faster on cotton materials than on fluid-repellent materials, and cotton gowns offer protection against droplets bearing viruses. Droplets or fomites that persiston a nonabsorbent disposable gown or gloves may be a risk to contaminate the environment.

SARS-CoV strain GVU6109 was isolated from a lung tissue specimen of a SARS

Table 1 Survival time of coronaviruses on different materials of inanimate surfaces

Surface	Virus	Strain	Inoculum	Temperature	Time	Ref.
Paper	SARS-CoV	GVU6109	10^4	RT	< 5 min	[35]
Paper	SARS-CoV	GVU6109	10^5		3 h	[35]
Paper	SARS-CoV	GVU6109	10^6		24 h	[35]
Paper	SARS-CoV	P9	10^5	RT	4-5 d	[26]
Stool normal adult pH7-8	SARS-CoV	GvU6109	10^7	RT 20 °C	1 d	[35]
Baby stool pH6-7					3 h	[35]
Diarrheal stool pH9					4 d	[35]
Baby stool pH6-7	Polivirus				> 4 d	
NPA	SARS-CoV	GvU6109	10^7	RT 20 °C	> 7 d	[35]
				4 °C	21 d	[35]
Cotton gown	SARS-CoV	GvU6109	10^4	RT 20	5 min	[35]
			10^5		1h	[35]
			10^6		24 h	[35]
Disposable gown	SARS-CoV	GUV6109	10^4		1 h	[35]
Disposable gown	SARS-CoV	GUV6109	10^5		24 h	[35]
Disposable gown	SARS-CoV	GUV6109	10^6		2 d	[35]
Cartridge brass	HuCoV	229E	10^3		5 min	
Brasses 70% copper			10^3		< 60 min	
Silicon rubber				RT 21 °C	3 d	
Metal	SARS-CoV	P9	10^5	RT	5 d	[26]
Stainless steel	HCoV	229E	10^3	RT 21 °C	≥ 5 d	[33,34]
Cupper	HCoV	229E	10^3	RT 21 °C	< 5 min	[33]
	MERS-CoV	HCoV-EMC2012	10^5	4 °C	≥ 28 d	[41]
	MERS-CoV	HCoV-EMC2012	10^5	20 °C	48 h	[41]
	MERS-CoV	HCoV-EMC2012	10^5	30 °C	8-24 h	[41]
Aluminum	HCoV	229E OC43	5×10^3	21 °C	2-8 h	[34]
PTFE	HCoV	229E	10^3	21 °C	5 d	[33]
PVC	HCoV	229E	10^3	21 °C	5 d	[33]
Glass	SARS-CoV	P9	10^5	RT	4 d	[26]
Glass	HCoV	229E	10^3	21 °C	5 d	[33]
Ceramic tiles	HCoV	229E	10^3	21 °C	5 d	[33]
Wood	SARS-CoV	P9	10^5	RT	4 d	[26]
Glove latex	HCoV	229E OC43	5×10^3	21 °C	≤ 8 h	
Silicon rubber	HCoV	229E	10^3	21 °C	5 d	[33]
Plastic	HCoV	229E	10^7	RT	2-6 d	[2]
	SARS-CoV	P9	10^5	RT	4 d	[26]
	SARS-CoV	HKU39849	10^5	22-25 °C	5 d-2 wk	[37]
	SARS-CoV	FFM1	10^7	RT	6-9 d	[2]
	MERS-CoV	Isolate		20	48 h	[41]
	HCoV	EMC 2012	10^5	30	8-24 h	[41]

NPA: Nasopharyngeal aspirate; PTFE: Polyfluorotetraethylene; PVC: Polyvinyl chloride; SARS-CoV: Severe acute respiratory syndrome coronavirus; MERS-CoV: Middle east respiratory syndrome coronavirus.

patient during the SARS outbreak in 2003[35]; its infectivity at 10^4 tissue culture infection doses ($TCID_{50}$)/mL vanished within 5 min after drying on paper or a cotton gown at RT[35], showing that the viral infectivity perished faster on the cotton gown than on an impervious surface (*e.g.*, the disposable gown) (5 min *vs* 60 min at 10^4 $TCID_{50}$/mL, 1 h *vs* 24 h at 10^5 $TCID_{50}$/mL)[35].

A piece of sterilized paper was experimentally contaminated with a higher titer virus 10^5 $TCID_{50}$/mL, equivalent to that of fecal excreta. The sample was allowed to be absorbed at RT for 3 h, then placed into a VeroE6 cell culture tube, and no viral infectivity was detected. With 10^6 $TCID_{50}$/mL, no viral infectivity was shown after 24 h. A higher concentration of 10^4 virus that was dropped on paper and allowed to dry

at RT showed no viral infectivity within 5 min. Usually the viral titer in nasopharyngeal aspirate specimens is $10^{2.2}$ $TCID_{50}$/mL[35]. Therefore, the risk of infection by contact with a droplet contaminated paper is small. Hand washing after touching potential materials is effective against SARS-CoV-2 transmission.

Survival of SARS-CoV in liquid: water, urine, and sewage

The survival time of SARS-CoV is impacted by viral stains, the types of solutions it stayed in, temperature, and viral titers. It could survive for 14 d at 4 °C, 2 d at 20 °C in dechlorinated tap water or domestic sewage, and 14 d at 20 °C in BPS. The SARS-CoV strain P9 was isolated from a pharyngeal swab of SARS patients[26]. In testing conditions, it could survive in serum, 1:20 diluted sputum, and feces for at least 4 d (96 h), and in urine for at least 3 d (72 h) with a low level of infectivity[26]. SARS-CoV P9 infectivity at RT persisted for 60 h after exposure, started to drop after 72 h, and was almost detectable after 120 h[26]. It stayed stable at 4 °C, 20 °C and at 37 °C for at least 2 h with infectivity in cells. A short time exposure can inactivate complement at 56 °C, and antibody at 67 °C, which is not enough to eliminate SARS-CoV infectivity; the conditions for SARS-CoV to become non-infectious should be at 56 °C for 90 min, 67 °C for 60 min, and 75 °C for 30 min, respectively[26]. Another study shows that coronaviruses remain infective at 4 °C for several months, and at -60 °C for many years[36], but are inactivated at 56 °C within 10-15 min, and at 37 °C after several days[36].

SARS-CoV GVU6109 (10^6 $TCID_{50}$/mL) can remain infectious in respiratory specimens for >7 d at RT, for > 20 d at 4 °C[35], and for 4 d in diarrheal stool samples at pH 9 at RT[35].

SARS-CoV HKU 39849 is relatively more stable than HuCoV 229E or OC43 and some other viral respiratory pathogens such as RSV. It is stable for 3 wk at RT in a liquid environment and after being dried on plastic, and its viability can be retained from 5 d to 2 wk at 22-25 °C at a relative humidity (RH) of 40-50%[37], which is like air conditioned indoor environments. It is easily killed at 56 °C for 15 min[37]. High RH of > 95% and at 28 °C or 33 °C did not significantly affect the infectivity of SARS-CoV HKU 39849. Increasing the temperature to 38 °C can suppress the virus. The viral viability was rapidly lost (3 log_{10}) at 38 °C and a RH of > 95%[37](Table 1).

At 25 °C, the time required for a 99% reduction in reagent-grade water was 22 d for transmissible gastroenteritis virus (TGEV) and 17 d for mouse hepatitis virus (MHV). In pasteurized settled sewage, a shorter time (9 d for TGEV and 7 d for MHV) was needed for a 99% reduction.These data suggest that contaminated water is a potential vehicle for human exposure[36].

Survival of SARS-CoV in vegetables under household refrigeration conditions

Bovine coronavirus is a pneumo-enteric virus that infects the respiratory tract and intestines of cattle and wild ruminants. Infectivity of bovine coronaviruses strain 88 (BCoV-88) on the surface of romaine lettuce under household refrigeration conditions could sustainfor at least 14 d[38].

The BCoV-88 infectivity in 10% bovine fecal suspension decreased faster than in a minimum essential medium of 2% FBS suspension, yet the reason is unclear. In bovine, there are abundant proteolytic or lipolytic enzymes like proteases or lipases in bovine intestines and bovine fecal suspensions, the virion surface spike glycoproteins are sensitive to protease cleavage, and the envelope is sensitive to lipase. Loss of a functional spike glycoprotein on the virion surface or loss of the virus envelope can confer BCoV-88 infectivity.

HuCoV strains 229E and OC43 cause one-third of common colds and hospital-acquired upper respiratory tract HuCoV infections, and survive in saline solution for 6 d[27]. It is reported that there was an < 1 log_{10} infectivity decrease for both TGEV and MHV at 4 °C after 4 wk[36]. At 4 °C, the time required to achieve a 4 log_{10} reduction in infectivity titer in pasteurized settled sewage was 98 d for TGEV *vs* 139 d for MHV, and the predicted time for an 4 log_{10} infectivity reduction of both TGEV and MHV in reagent-grade water was approximately one year[36].

Temperature and humidity impact the persistence time of viruses

Persistence of most bacteria, fungi, and viruses (*e.g.*, SARS-CoV) on surfaces depends on environmental conditions[37], such as air temperature and RH[39], inoculums, and the materials that they stayed on. Low temperature, high inoculums, and proper RH are associated with longer persistence time for most viruses. The role of the environment on the survival of viruses in the air may be more complex and significant[40]. RH impacts the survival time of viruses, and the relationship between the inactivation of RH and temperature was not monotony. The temperature impacts the survival of the virus more significantly than that of RH. In all water types tested (reagent-grade water, lake water and settled sewage), the titer of infectious virus

declined more rapidly at 25 °C than at 4 °C[36].

A high RH of > 95% and temperature of 28 °C or 33 °C did not significantly affect the infectivity of SARS-CoV HKU 39849. Increasing the temperature to 38 °C disfavors the virus, with the virus rapidly losing viability (3 $\log_{10}$) at 38 °C and RH of > 95% (Table 1)[37]. A high temperature (*e.g.*, 30 °C or 40 °C) reduced the persistence duration of highly pathogenic MERS-CoV, TGEV, and MHV. The persistence of SARS-CoV was longer with higher inocula. HCoV-229E at RT persists longer at RH 50% than at RH 30%.

The survival of airborne HuCoV 229E was investigated under different conditions of temperature and RH. At 20 °C, at 50% RH, aerosolized HuCoV-229E was found to survive best with a half-life of 67.33 ± 8.24 h, with nearly 20% infectious virus detectable at 6 d; while at 30% RH the virus half-life was 26.76 ± 6.21 h. At high 80% RH, the half-life was only about 3 h, and no virus in aerosols could be detected after 24 h. At 6 °C, in either 50% or 30% RH conditions, the survival of HuCoV-229E was significantly enhanced, with the decay pattern essentially similar to that seen at 20 ± 1 °C. At 6 °C in 80% RH, however, the HuCoV-229E half-life increased to 86.01 ± 5.28 h, nearly 30 times that found at 20 °C and 80% RH[40].

Human coronaviruses on metal-containing copper surfaces areeffectively inactivated

TGEV and MHV were used as conservative surrogates for modeling exposure to determine the effects of AT and RH on the survival of coronaviruses on stainless steel[39]. At 4 °C, the infectious virus persisted for 28 d, and the lowest level of inactivation occurred at low RH (*e.g.*, 20%). The results show that high numbers of TGEV and MHV may survive for days on surfaces at ATs and RHs typical of indoor environments. Both viruses were inactivated more rapidly at 40 °C than at 20 °C.

Metal containing copper may demonstrate antiviral activity. Two metal catalysts, Ag/Al_2O_3 and Cu/Al_2O_3, can inhibit the transmission of SARS and other respiratory infectious diseases[3]. Two metal catalysts, Ag/Al_2O_3 and Cu/Al_2O_3, were pressed into wafers. One hundred μL 10^6 $TCID_{50}$/mL SARS-CoV, 100μL 10^6 PFU/mL recombinant baculovirus expressing hamster's prion protein, and roughly 10^6 *E. coli* were slowly dropped onto the surfaces of the catalyst wafers and exposed for 5 min and 20 min, respectively. The infectivity of SARS-CoV in Vero cells and baculovirus in Sf9 cells dropped down to a very low and undetectable level, with no colony detected using a bacterial culture method. The expression of hamster's prion protein reduced to 21.8% in the preparation of Sf9 cells infected with recombinant baculovirus after exposing for 5 min, and was undetectable after exposing for 20 min. Bacterial membranes seemed to be cracked and the cytoplasm seemed to be effluent from cell bodies. Exposures to the surfaces of Ag/Al_2O_3 and Cu/Al_2O_3 for 5-20 min can destroy the replication and propagation abilities of SARS-CoV, baculovirus, and *E. coli*.

HuCoV-229E on copper and copper alloy surfaces was inactivated by released copper ion and reactive oxygen species, resulting in irreversible fragmentation of the viral genome, which was visibly confirmed by morphological changes under transmission electron microscopy. The rate of inactivation was directly proportional to the percentage of copper. Coronavirus was inactivated in 40 min on brass and 120 min on copper nickels containing less than 70% copper. Brass containing at least 70% (*e.g.*,90%, 95%, and 100%) copper effectively inactivated 10^3 PFU HuCoV-229E within 20 min. HuCoV-229E on brass and copper nickel surfaces at RT (21 °C) can be inactivated; 10^3 PFU inoculum was applied as 1μL/cm^2 copper and cartridge brass, respectively, and was dried immediately, and the viruses were inactivated within 5 min, which simulates fingertip touch contamination. Approximately 10^3 PFU in simulated wet-droplet contamination was inactivated in less than 60 min. The SARS-CoV strain P9 in culture medium lost viral infectivity at an undetectable level after UV irradiation for 60 min[26].

CONCLUSION

The most common nosocomial coronaviruses may well survive or persist on inanimate surfaces for up to a month. The risk of transmission *via* touching contaminated paper is low, while respiratory and fecal specimens can maintain infectivity for quite a long time at room temperature. SARS-CoV-2 could exist in the air in poorly ventilated buses for at least 30 min. Absorbent materials like cotton are safer than nonabsorptive materials for protection from viral infection. Proper preventive strategies such as washing hands and wearing masks are critical for containing COVID-19.

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