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Fomites and the COVID-19 pandemic: An evidence review on its role in viral transmission

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Background

Since the beginning of the COVID-19 pandemic, public health messaging continuously evolved as more evidence emerged about the risks associated with SARS-CoV-2 transmission and infection. One of the common control measures suggested from the beginning was to increase cleaning and disinfection in public and private settings based on prior understanding or beliefs about transmission of respiratory viruses. Disinfectants, soaps, and hand sanitizers are known to be effective against coronaviruses and other pathogens at the appropriate concentrations with adequate contact time.[1,2] With heightened concerns about COVID-19, many businesses and public facilities have allocated significant resources to support more stringent and frequent disinfection practices. Some surface disinfection technologies may be very costly especially for small businesses. Some people have also expanded their use of cleaning and disinfection products at home to items beyond frequently touched surfaces (e.g. disinfecting grocery packaging and food take-out containers).[3] Evidence shows that misuse and overuse of cleaning and disinfection products may cause adverse acute and chronic health impacts.[4] In addition, there have been questions about the contribution of fomites to SARS-CoV-2 transmission, and whether excessive surface disinfection is warranted. Some are downplaying the importance of fomites as a transmission pathway as emphasis is shifted to droplet and aerosol transmission.[5]

Thousands of variants of SARS-CoV-2 have been sequenced since the virus began circulating in early 2020, and certain variants have emerged as dominant strains that spread across several continents including Europe and parts of Africa due to quarantine-free travel.[6,7] It is currently unclear whether these mutations in protein spikes on the surfaces of the viruses impart transmission advantages or enhance surface survivability.[6,7] However, some epidemiological evidence and modelling data show that the strain recently identified in the United Kingdom (VOC-202012/01 or lineage B.1.1.7) may be more transmissible.[8]

This review will examine available epidemiological and research evidence on the infection risk of SARS-CoV-2 via fomites. Factors that influence SARS-CoV-2 transfer to and from fomites, as well as how environmental factors may influence the persistence of SARS-CoV-2 on environmental surfaces, will be discussed.

Search methodology

A search for peer-reviewed or grey literature related to this topic was conducted in EBSCOhost databases (includes MEDLINE, CINAHL, Academic Search Complete, ERIC, etc.), Google Scholar, and Google using a variation of the following keywords: (coronavirus OR ncov OR "novel cov" OR COVID-19 OR SARSCOV-2 OR Sars-Cov-19 OR SarsCov-19 OR SARSCOV2019 OR "severe acute respiratory syndrome" OR "2019 ncov" OR "2019ncov" OR nCOV); (bacteria OR contamination OR contaminant) AND (transmit OR transmission OR droplet OR spray or deposition OR deposit OR contact) AND (fomite OR "contact OR "shopping cart" OR elevator OR button OR touchscreen OR "touch screen" OR "grocery bag" OR handrail OR directory OR dispenser OR mailbox OR intercom OR door OR tap OR dryer OR dispenser OR directory OR cart OR buggy OR basket); (supermarket OR "environmental surface" OR "dry surface"); (indoor OR room OR office OR restaurant OR dining OR shop OR business OR premise OR house OR home OR residence OR apartment OR condominium OR condo OR apartment OR high-rise OR mid-rise OR low-rise OR dormitory OR dormitories OR shelter OR flat OR building OR arena OR gym OR classroom OR class OR school OR university OR daycare OR "day care" OR centre OR center OR institution OR hospital OR clinic OR lab OR laboratory OR "confined space"). This was followed by a scan of bibliographies of key papers for additional related literature.

Virology of SARS-CoV-2

SARS-CoV-2 is a single-stranded RNA virus with an outer lipid envelope covered with protein spikes that enable the virus to bind to angiotensin-converting enzyme 2 (ACE2) receptors on host cells. The protein spikes on SARS-CoV-2 are structurally different from those on SARS-CoV, thereby allowing stronger binding to ACE2 receptors.[9] ACE2 receptors are found in several cell types including epithelial, goblet and endothelial cells in respiratory mucosa.[10] ACE2 receptors are expressed in greater abundance in the upper respiratory tract, specifically in the nasopharyngeal region, compared to the lower respiratory tract.[11] Within the lower respiratory tract, ACE2 receptors are more commonly detected in the bronchioles and the alveoli compared to the trachea and bronchi.[11] The distribution and abundance of ACE2 receptors also vary between individuals.[11] ACE2 receptors are also found in vascular epithelia, renal and cardiovascular tissue, as well as the epithelia of the small intestines and testes.[10]

SARS-CoV-2 is primarily transmitted via droplets and aerosols expelled during speaking, coughing, sneezing, and other respiratory actions. It is secondarily transmitted via environmental surfaces (fomites) when infectious viral particles are transferred from an infected person to a surface, and subsequently to the mucous membranes of a susceptible host. Some virus may also be released in other bodily fluids such as feces. To better understand how viruses may settle on or contaminate surfaces and subsequently cause infection, current knowledge on virological concepts including viral load in the nasopharyngeal region and viral infectious dose need to be explored. Viral load refers to the concentrations of viral particles in the bodily fluids of an infected person and can influence how many viruses may end up contaminating surfaces prior to being transferred to the hands and mucous membranes of another person. The infectious dose is the quantity of viral particles required to cause an infection. Given that human studies are very limited, the following sections will also include findings from modelling studies.

Viral load

The number of viruses present in body fluid/excretion samples depends on the number of days since symptom onset, sampling location, type of sample (saliva, throat or nasal swab, urine, or stool) and other host factors. Highest viral loads were observed in the upper respiratory tract during the first week of symptom onset.[12] Peak viral loads in the lower respiratory tract were observed in the second week of illness.[12] Viral loads steadily declined over the duration of illness.[12] Patients with more severe symptoms appeared to have a higher viral load in the nasopharyngeal region compared to patients with mild symptoms.[13,14] Findings from several studies suggest that based on throat and nasal swabs, the viral load in an infected person may range from 641 to 1.34×10^{11} copies per mL depending on the severity of illness and the number of days since symptom onset.[15-18]Median viral loads were found to be 7.99×10^{4} copies/mL in throat samples, 7.52×10^{5} copies/mL in sputum samples, and 1.69×10^{5} copies/mL in one nasal sample.[15] A review of seven studies found that there is generally little or no difference between viral loads in asymptomatic, presymptomatic, and symptomatic patients.[19] In contrast, two studies found lower viral loads in asymptomatic individuals than in symptomatic individuals.[15]

The amount of virus transferred from an infected person to a surface depends not only on the viral load but also on the quantity of bodily fluids in the form of droplets that either settle on surfaces or are transferred from contaminated hands. The size of respiratory droplets expelled during breathing, coughing, sneezing, and other respiratory actions differ. For example, during coughing, the size of expelled droplets typically range between 0.6 and 15 µm.[20] Regular breathing produces microdroplets that are mostly smaller than 1 µm.[20] Larger droplets are more likely to settle on surfaces, whereas small droplets or aerosols may remain suspended. The number of viruses shed in respiratory droplets depends on the viral load in the nasopharyngeal region. Based on previous findings on the viral load found in the nasopharyngeal region, a mathematical model was developed to determine SARS-CoV-2 shedding during breathing and coughing.[20] The model suggests that respiratory actions such as coughing or sneezing expel a greater concentration of viruses compared to breathing or talking. However, the higher frequency of breathing and talking is still an important consideration in viral transmission.[21] Assuming a viral load value of 10^{6} copies/mL to represent a typical emitter, the model determined that a typical emitter may expel 0.277 copies per mL of air in a single cough. Past studies on cough dynamics suggest that the air volume of a single cough may range from 0.8–5.0 L, equating to approximately 221.6–1,385 viral copies per cough for a typical emitter.[22,23] Given this, sequential coughs in a single episode would generate more airborne viruses as small droplets are expelled.[21] Many factors influence the cough volume, including symptom severity, head position, and mouth opening. The number of viral copies per cough would therefore vary widely. Further research is needed to confirm this estimate and determine the number of viral copies via other respiratory actions such as sneezing or shouting.

Infectious dose

Infectious dose is the quantity of infectious viral particles required to cause infection, and differs for different individuals due to biological factors. The infectious dose for different SARS-CoV-2 variants may also differ. Generally, the lower the number of viruses required to initiate infection in a host, the more transmissible the virus is. In laboratory studies, there are a variety of ways to express the infectious dose, including plaque-forming units (PFU), viral particles/copies, human median infectious dose

(human ID_{50}), and median tissue culture infectious dose ($TCID_{50}$). This variation adds to the complexity and difficulty in interpreting and comparing different studies.

The infectious dose also depends on the transmission route and an individual's biological factors such as ACE2 receptor expression and immune response. Some animal studies show that the infectious dose for SARS-CoV-2 is lower through aerosol transmission compared to intranasal inoculation, and may lead to increased mortality and morbidity.[24] Studies on the infectious dose of SARS-CoV-2 are limited. Based on data from human exposure studies, animal studies, and modelling studies, the amount of viral genetic material necessary to initiate infection in humans is around 10-1000 viral copies.[25] This suggests that the minimum infectious dose of SARS-CoV-2 may be slightly higher than that of SARS-CoV-1 (approximately 280 viral particles).[24,26,27] The number of viruses a person is exposed to in a given incident is influenced by duration and intensity of contact with the infected person, either through exposure to a high concentration of viruses in a single incident, or through prolonged exposure to a lower concentration of viruses.

A review of past studies on related respiratory viruses such as influenza, SARS-CoV, and MERS suggests that severity of symptoms exhibits a viral dose-dependent relationship.[28] In three SARS-CoV-2 clusters, people exposed to lower estimated doses of the virus exhibited milder symptoms compared to others who were exposed to a higher viral dose.[29] Further research is required to understand the relationship between initial inoculum, symptom severity, and infection duration.

Fomite contamination and viral transfer dynamics

Fomites may become contaminated in two ways: either from direct deposit of bodily fluids such as respiratory droplets, or via cross-contamination from contaminated hands. It is currently unknown whether airborne particulates such as dust may act as carriers of SARS-CoV-2. Fomite contamination and transfer dynamics are complex due to the numerous variables that influence transfer between hands and environmental surfaces and mucous membranes. The amount of virus that a person may be exposed to via fomites depends on how many viruses were shed by an infected person and what fraction of that initial inoculum is transferred to surfaces, then to hands, and eventually to mucous membranes. Factors that affect viral transfer include the type of surface, skin characteristics, humidity, and temperature. Studies on influenza A inactivation on skin suggest that skin appears to have antiviral properties that cause rapid inactivation of the viruses on human hands.[30] However, no studies have examined whether live human skin is able to inactivate SARS-CoV-2. Additional factors such as personal behaviours, e.g., coughing or sneezing into hands or sleeves, or whether personal protective equipment was worn, also influence how many viruses may be deposited to surfaces.

Generally, viral transfer to hands from porous surfaces is lower than from non-porous surfaces.[31,32] Experimental studies and mathematical modelling can help to provide insight into the likelihood of infection via surfaces. While some studies on microbial transfer from surfaces to/from hands on other respiratory viruses have been conducted, evidence examining SARS-CoV-2 interactions between surfaces and hands is scarce.[33]

Viral transfer between fomites, hands, and mucous membranes

A study demonstrated that SARS-CoV-2 viral RNA levels were found to be lower on environmental surfaces than in the nasopharyngeal samples of the patients in quarantine rooms, indicating that only a

fraction of viruses shed through respiratory droplets end up on surfaces.[34] Evidence from environmental studies confirms that microbial transfer from hands to surfaces is possible.[31,35] A study demonstrated that bacteriophages MS2, fr, and ϕX174 could transfer from finger pads to glass, but transfers were reduced with handwashing.[31] Handwashing removes biological constituents such as sebum, sweat, and microflora from hands while shifting the chemical characteristics of the skin by increasing pH and hydrophobicity. It is unclear from this study how these skin factors influence viral transfer. Further studies are needed to examine the effect of handwashing on SARS-CoV-2 transfer efficiency.

Evidence from past studies shows that the transfer fraction between fomites is dependent not only on the type of surface and relative humidity, but also on the viral species.[32] In a study examining rhinovirus and human parainfluenza virus transfer from finger pads to metal disks, hand-to-disk transfer was greater than disk-to-hand transfer.[36] In another study using hepatitis A virus, the opposite result was found.[37]

A study found that surfaces frequently touched by individuals infected with rhinovirus are easily contaminated with their viruses. Approximately 35% of 150 surfaces sampled tested positive for rhinovirus RNA. Door handles, pens, light switches, TV remote controls, and faucets were the most frequently contaminated surfaces.[35] Similarly, another study demonstrated that 43% of tiles touched by subjects with rhinovirus present on their hands were found positive for rhinovirus.[38]

A study on viral transfer from surfaces inoculated with rhinovirus found that viral transfer to fingertips occurred in 47% of trials.[35] However, infectivity of the transferred rhinoviruses was not assessed. In another study, after handling a coffee cup contaminated with rhinovirus, 50% of test subjects became infected and rhinovirus was recovered in nose/throat samples.[39]

Risk assessment of fomite transmission

The studies in this section are based purely on mathematical models. While these models attempt to use evidence-based values and assumptions, there are many variances in the assumptions in real-world scenarios. Therefore, while informative, results from these studies should not be used to make conclusions.

Several modelling studies were found on SARS-CoV-2 and other related respiratory viruses such as influenza and SARS-CoV. Most studies found that infection risk via fomites in the model scenarios was much lower compared to droplet and aerosol transmission.

Two modelling studies specifically investigating the infection risk of SARS-CoV-2 via fomites were found. The first mathematical model analyzes the contributions of three transmission routes, fomite transmission, droplet transmission, and inhalation of virus-containing droplets, to overall infection risk for healthcare workers providing care to COVID-19 patients with or without personal protective equipment such as surgical masks and eye protection. Results suggest that the contributions of fomite, droplet, and inhalation transmission without any personal protection to overall risk are 6.9%, 32%, and 61% respectively. With personal protective equipment, the mean percent contributions of fomite, droplet, and inhalation transmission are 2.8%, 30%, and 68% respectively.[40] The use of N95 respirators would further lower the infection risks although the specific percent contributions for each transmission route were not quantified. Whether with or without personal protection, the model suggests that the risk of SARS-CoV-2 infection is much greater through droplet and aerosol transmission than through fomite transmission.

The second mathematical model uses data from the outbreak on the *Diamond Princess* cruise ship, and analyzes three transmission routes: contact transmission, short-range (droplet) transmission, and long-range (aerosol) transmission.[41] Results suggest that the mean estimates of the contributions of contact, droplet, and aerosol transmission to the infected cases were 30%, 35%, and 35% respectively, suggesting that the infection risk of droplet and aerosol transmission are greater than fomite transmission.[41]

A modelling study on a large SARS-CoV outbreak in a hospital in Hong Kong was conducted to estimate the contribution of fomites to viral transmission compared to aerosol transmission. The model suggested that fomites play a smaller role in combined SARS-CoV transmission, and airborne transmission is the still predominate pathway for viral spread.[42]

A study by Nicas et al. (2008) developed a quantitative risk assessment model to attempt to assess the infection risk of respiratory viruses based on the viral load from coughs, number of hand-to-facial mucous membrane contacts, number of hand-to-environmental surfaces contacts, viral inactivation rate on hands, and viral transfer efficiency rate from non-porous and porous surfaces to hands.[43] The authors applied this mathematical model to a hypothetical scenario to estimate the infection risk for influenza A transmission from environmental surfaces in a residential bedroom while attending to a sick family member. The estimated infection risk was 0.011%. Due to uncertainties and variations in the factors used in the calculation of the infection risk, this model can only provide a crude first-pass estimate.[43]

Another similar model by Atkinson et al. (2008) was published around the same time as the Nicas et al. study.[44] This study modelled influenza A infection risk for household members living in the same residence as a sick family member, and considers aerosol and fomite transmission. After accounting for viral inactivation on skin and surfaces as well as numerous other parameters, the authors suggested that aerosols play a much greater role compared to fomites in influenza A transmission.[44] This was corroborated by another modelling study for rhinovirus and influenza, in which fomites were also found to have contributed the least to influenza transmission.[45]

Environmental factors influencing persistence of SARS-CoV-2 on surfaces

The infection risk of SARS-CoV-2 via fomites depends greatly on the longevity of SARS-CoV-2 on skin and various types of surfaces. Typically, surface persistence studies use a combination of real-time reverse transcription polymerase chain reaction (rRT-PCR) and cell culture to determine viral infectivity. rRT-PCR detects and quantifies the presence of viral genetic material, but is not able to assess infectivity. Cell culture is needed in order to assess infectivity of the viruses present in the sample. Although environmental surfaces in hospitals, quarantine rooms, and cruise ships that housed COVID-19 patients have been found to be extensively contaminated with SARS-CoV-2 genetic material, many studies do not go a step further to culture the samples to assess infectiousness of the viruses in the samples. The presence of viral RNA does not necessarily indicate infectiousness.

Several studies assessing SARS-CoV-2 contamination on frequently touched surfaces in hospital and healthcare settings were not able to culture viable viruses from the environmental samples.[46-51] One study examining SARS-CoV-2 contamination in two hospital rooms and nine residential isolation rooms housing COVID-19-positive patients was able to detect infectious SARS-CoV-2 via cell culture from one

air sample from a hallway at the residential isolation facility and one surface sample from a windowsill in a residential isolation room out of 163 samples collected in the study.[52]

Surface persistence is influenced by the type of surface as well as environmental factors. Research on the sensitivity of SARS-CoV-2 to various environmental factors is ongoing. Current studies on the effects of temperature and relative humidity on SARS-CoV-2 have been conducted under experimental conditions only. For an overview of current laboratory studies on SARS-CoV-2 persistence on surfaces, see appendix A. Further research is required to determine if new SARS-COV-2 variants of concern behave similarly on surfaces.

Temperature

Temperature may influence the surface persistence of SARS-CoV-2 by affecting the stability of the viral lipid envelope. SARS-CoV-2 and other related coronaviruses have been found to deteriorate faster both in suspension and on surfaces at higher ambient temperatures, but are more resistant to colder temperatures.[53-56] Harbourt et al. (2020) demonstrated that SARS-CoV-2 remains viable for much longer on skin, clothing, and currency at 4°C compared to at 37°C.[54] Chin et al. (2020) found that at 4°C in suspension, there was only a $10^{0.7}$ reduction in infectivity after 14 days.[55] When the temperature was increased to 70°C, inactivation of the suspended viruses occurred in five minutes.[55] Freezing temperatures during storage of foods does not seem to have a noticeable effect on reduction of SARS-CoV-2.[57]

Relative humidity

The ambient humidity of an indoor environment affects the suspension and movement of respiratory droplets and aerosols that may contain SARS-CoV-2. Relative humidity (RH) also affects the rate at which respiratory droplets and aerosols evaporate, which consequently affects the stability of the virus as desiccation inactivates the virus.[58] At room temperature, SARS-CoV-2 appears to be most stable at lower RH (20%) compared to higher RH (80%), with half-lives of ~15.33 hours and ~8.33 hours respectively.[53] The same effect is observed at a higher temperature of 35°C under experimental conditions, with a half-life of ~7.33 hours at 20% RH compared to ~2.26 hours at 80% RH.[53]

Using SARS-CoV-2 surrogate viruses, transmissible gastroenteritis virus (TGEV) and mouse hepatitis virus (MHV), a study found that at 20°C, viruses remained infectious for at least three days at 50% RH compared to up to 28 days at 20% RH.[59] At 40°C, both viruses were able to remain infectious up to five days, but less than six hours at 80% RH.[59] Appendix A outlines the results from laboratory studies on SARS-CoV-2 persistence on various surfaces.

Overall, SARS-CoV-2 appears to be most stable at lower RH and lower temperatures, which may also contain the most comfortable ranges for most people. The persistence of viruses on surfaces is partly due to a complex interaction between humidity and temperature that causes either desiccation or a change in evaporation rate, leading to changes in the viral lipid envelope structure.[59] The interactions between humidity, temperature, and viral inactivation remains to be further verified and explored.

Criticisms on current surface persistence studies

Experiments on SARS-CoV-2 persistence on a variety of surfaces have been conducted in controlled laboratory settings, which are generally more favourable for viral survival. In the real world, indoor environment conditions may vary widely. To the author's knowledge, there have been no studies on SARS-CoV-2 surface persistence in real-world conditions. Additionally, most studies use culture medium, which differs from human respiratory fluids that contain antimicrobial constituents and other properties that may inactivate viruses.[60-62] Bueckert et al. (2020) compared studies using culture medium to others using artificial saliva/mucous mix or human nasal mucus or sputum, and found that culture medium enhanced SARS-CoV-2 persistence on test surfaces.[60] Enriching culture medium with bovine serum albumin (BSA) or fetal calf serum (FCS) also improved SARS-CoV-2 stability on test surfaces.[60]

It has been argued that existing surface persistence studies inoculate experimental surfaces with concentrations and volumes of viral titres that are not reflective of real-world conditions, thereby leading to apparent longer survival times.[63] On the other hand, a study by Biryukov et al. (2020) found that inoculation volume does not have a significant impact on the half-life of SARS-CoV-2 on stainless steel.[53] This may be due to the concentration of the initial viral inoculation in the Biryukov study, which was not clearly mentioned in the study. Further research is needed to determine whether concentration or volume of the initial viral titre affects surface persistence.

More research is also needed to determine how many viruses may realistically contaminate a surface from respiratory actions such as a cough or a sneeze, or from contaminated hands. Only a fraction of virus-containing droplets in a cough or a sneeze may settle on a surface or transfer from surface to hands, and subsequently to mucous membranes.

Apart from determining how long viable viruses remain detectable on surfaces, it is worth noting that data from surface persistence studies summarized in Appendix A demonstrate that the concentration of viable viruses decreases over the duration of the experiments. This suggests that although viable SARS-CoV-2 may be detected on surfaces after a certain period of time, the concentration may be so low that the risk of infection is consequently reduced.

Cases, clusters, and outbreaks potentially linked to fomites

Past cases, clusters, or outbreaks may provide clues as to the likelihood of fomites being the medium for SARS-CoV-2 transmission. The author was only able to find seven published reports. Table 1 outlines outbreaks or clusters in which transmissions have been suspected to be via surfaces; however, the evidence is mostly circumstantial and not definitive. Many of these clusters/outbreaks may have been transmitted through multiple pathways. In most outbreaks, it is difficult to ascertain the implicated transmission pathways. Although outbreak investigators may have determined that only fomites were implicated in viral transmission, respiratory pathways may still have played a role.

Table 1. Outbreaks or clusters in which surfaces potentially contributed to transmission

Authors	Number of cases	Epidemiological findings	Suspected transmission route(s) identified by the authors
Brlek et al.[64]	6	All six cases played squash in court hall number 1, which is a small confined space with poor ventilation. Only one case had direct contact with index case; all other cases denied close contact with other cases. All cases used the dressing room to change, but could not confirm if they used the same stalls. Index case used the shower facilities as did 2 separate cases. No one else in the sports facility developed symptoms. No environmental sampling was conducted.	Respiratory Fomites
New Zealand Ministry of Health[65]	3	Case A completed 14-day quarantine in a managed isolation facility, then flew from Christchurch (location of the facility) to Auckland. A week later case A tested positive. Case B was seated behind case A on the same chartered flight from Christchurch to Auckland. Case B was asymptomatic on the flight but tested positive a few days after case A. Investigators hypothesized that case B may have been infected by another case at the managed isolation facility via the surface of a shared rubbish bin.	Fomites
Xie et al.[66]	5	Individuals from two separate families in a 61-resident apartment dwelling tested positive for COVID-19 (three from family A, two from family B). Both families denied contact with each other. Investigators hypothesized that a case from family B was infected through touching an elevator button contaminated with nasal discharge from a case in family A on the same day.	Fomites
Liu et al.[67]	72	Patient A returned to China from the United States and quarantined alone in her apartment building. A neighbour (patient B) in her building tested positive for the same strain and transmitted it to a friend (patient C). Patient C and close contacts tested positive for the same strain as patient A, which differed from the strain circulating in China at the time. Hospital staff and patients at the hospitals to which patient C was admitted for stroke prior to testing positive also eventually tested positive for the same strain. Investigators hypothesized that patient B was most likely infected by patient A via surfaces in the shared elevator.	Fomites Respiratory

Lessells et al.[68]	135	Investigators hypothesized that the virus was introduced by a patient admitted to the Emergency Department at a hospital, which spread rapidly throughout the hospital due to movement of staff and patients between and within wards. This main outbreak likely spread to a local nursing home and an outpatient dialysis unit on the same campus. Evidence indicated that transmission may have been facilitated through direct droplet as well as indirect fomite transmission throughout the hospital.	Respiratory Fomites

Suggested measures to reduce fomite transmission

With the understanding that viral transmission occurs when viruses in droplets or on contaminated hands are transferred to surfaces and subsequently to hands and mucous membranes, severing one aspect of this chain of transmission would reduce the risk of infection. There are some conventional methods as well as novel technologies that may be applied at different stages of the transmission pathway.

Conventional interventions to reduce fomite transmission include surface cleaning and disinfection, face coverings, and hand hygiene. With adequate disinfectant concentration and contact time, cleaning and disinfection are effective methods to drastically reduce the presence of SARS-CoV-2 on environmental surfaces.[69,70] Proper hand hygiene, including using soap and water or hand sanitizers, is another effective intervention to prevent SARS-CoV-2 transmission. When used appropriately, face coverings may also reduce the likelihood of virus-containing droplets contaminating surfaces, or the touching of mucous membranes with contaminated hands.[71]

Novel technologies have also emerged that either possess antimicrobial properties or reduce viral transfer from surfaces to hands. Past studies have assessed the effectiveness of copper against a variety of respiratory viruses. It is proposed that copper is able to inactivate pathogens by damaging their ability to replicate, thereby reducing infectivity.[72,73] Copper-containing materials and coatings have been found to be effective against a variety of bacterial species and enteric viruses. Copper reduced the infectivity of SARS-CoV and *Escherichia coli* to undetectable levels after a five-minute exposure.[74] Copper alloy surfaces were able to cause complete destruction of SARS-CoV surface proteins and envelope as well as damage to genetic material in less than 60 minutes.[75] A review of several studies using a variety of copper forms (copper alloy dry surface, sodium copper, ionic copper oxide, copper iodide, Cu2+, and lay copper) found that they are effective against several types of influenza viruses.[73] Copper cold spray coating was able to reduce influenza A viruses by 97.7–99.3% with adequate time.[72] A study in a nursing home compared patient areas equipped with copper surfaces versus non-copper surfaces and found that outbreaks of keratoconjunctivitis and gastroenteritis possibly by adenovirus and norovirus were reduced in patient areas using copper surfaces.[76] However, a reduction in influenza A outbreaks was not observed, possibly due to the airborne transmission nature of the virus. Other novel technologies include antiviral polymers surface coatings that either repel viruses or incorporate a virucidal substance that may inactivate viruses upon contact.[77] Nanomaterials may also inactivate enveloped viruses such as SARS-CoV-2. Effectiveness against SARS-CoV-2 still needs to be determined.

Apart from coatings that have antiviral properties, a new technology employing engineered micropattern coating alters the structure of the surface. The coating proposes to increase the hydrophobicity of the surface, thereby reducing the amount of fluids that are transferred upon contact.[78] The micropattern also reduces the surface area for contact with viruses, which interferes with surface attachment and persistence. Lastly, while the droplet dries, capillary action pulls the viruses into the base of the pattern, thereby reducing the transfer of viruses onto hands during a subsequent contact. Experiments using human coronavirus 229E resulted in a 67.3–69.3% reduction in subsequent viral transfer after inoculation.[78]

Key messages

- SARS-CoV-2 viral load in the nasopharyngeal region may range from 641 to 1.34×10^{11} copies per mL depending on the severity of the illness and the number of days since symptom onset.
- Animal, human, and modelling studies suggest that the infectious dose of SARS-CoV-2 ranges from 10–1000 viral copies, which is slightly higher than that of SARS-CoV-1 and lower than that of Middle East Respiratory Syndrome (MERS).
- There is limited epidemiological evidence to support SARS-CoV-2 transmission via fomites, compared to transmission via droplets.
- There is limited evidence that may be inferred from surface persistence studies due to lack of generalizability to real-world situations.
- While surface cleaning and disinfection is the easiest control measure to implement, it should be balanced with other interventions to reduce viral transmission through all pathways.

Knowledge gaps

Further research is needed to verify mathematical models, to confirm available research, and to fill knowledge gaps. Several knowledge gaps were identified through this evidence review:

- What fraction of SARS-CoV-2 in respiratory emissions may be transferred from contaminated hands to surfaces, what fraction of viruses on surfaces may transfer onto hands, and what fraction of viruses on hands may transfer onto mucous membranes?
- Can airborne particulates such as dust act as carriers for SARS-CoV-2?
- How long is SARS-CoV-2 able to persist on live human skin?
- Do new SARS-CoV-2 variants of concern behave differently on surfaces?

Conclusion

Although limited, current evidence indicates that the risk of infection from fomites is low, and fomites are not likely to be the major transmission pathway for SARS-CoV-2 in most situations. However, SARS-CoV-2 RNA has been found on environmental surfaces in hospital rooms, quarantine rooms, and other community settings, implying that the surfaces can become contaminated with SARS-CoV-2 despite few studies being able to culture live viruses.[46,52,79-82] Additionally, new variants with potentially greater transmissibility continue to emerge, and they may behave differently on surfaces than the strains that have been studied to date.

It is important to continue to follow the multilayered control measures approach in order to sever the chain of transmission at all possible links, including proper hand hygiene and appropriate cleaning and

disinfection. Given that available evidence suggests that droplet and aerosol transmission continue to be the primary transmission route for SARS-CoV-2, it would be prudent to balance surface disinfection with other interventions to prevent droplet and aerosol transmission. Public health messaging should also emphasize the safe use of disinfection products to prevent acute and chronic health impacts from overuse and misuse of these products. To learn more about the overuse and misuse of disinfectant products, please see the NCCEH resource: "A rapid review of disinfectant chemical exposures and health effects during COVID-19 pandemic."

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Appendix A: *Summary of current laboratory studies on SARS-CoV-2 persistence on surfaces*

Authors	Temperature	Relative humidity	Inoculation medium	Inoculation titre concentration	Volume of viral titre	Surface types	Results
Hirose et al.[83]	25°C	45-55%	• DMEM† supplemented with 5% FBS‡ • Human mucus/ sputum	10^5 $TCID_{50}$/mL	5 µl	• Skin (forensic sample) • Stainless steel Borosilicate glass • Polystyrene plastic	**Skin (forensic sample):** Culture medium: 9 hours; half-life ($t_{1/2}$) = 3.53 hours Mucus: 11 hours; $t_{1/2}$ = 4.16 hours **Stainless steel:** Culture medium: ~3.5 days; $t_{1/2}$ = 32.62 hours Mucus: ~2.5 days; $t_{1/2}$ = 25.53 hours **Borosilicate glass:** Culture medium: ~3.5 days; $t_{1/2}$ = 33.24 hours Mucus: ~2.5 days; $t_{1/2}$ = 23.63 hours **Polystyrene plastic:** Culture medium: ~2.4 days; $t_{1/2}$ = 22.58 hours Mucus: ~1.5 days; $t_{1/2}$ = 13.17 hours
Chin et al.[55]	22°C	65%	Not mentioned	$10^{7.8}$ TCID50/mL	5 µl	• Paper • Tissue paper • Wood • Cloth • Glass • Banknote • Stainless steel • Plastic • Mask (inner layer) • Mask (outer layer)	**Paper**: viable up to 30 minutes **Tissue paper**: viable up to 30 minutes **Wood**: Viable up to 24 hours **Cloth**: Viable up to 24 hours **Glass**: Viable up to 2 days **Banknote**: Viable up to 2 days **Stainless steel**: Viable up to 4 days **Plastic**: Viable up to 4 days **Mask (inner layer)**: viable up to 4 days **Mask (outer layer)**: Viable up to 7 days
Biryukov et al.[53]	24°C 28°C	20% 40%	Simulated saliva	Not clearly indicated;	1 µl 5 µl	• Stainless steel	Authors combined results from different surfaces and titre volumes as half-lives ($t_{1/2}$) were not

	35°C	60% 80%		diluted 1:10 from viral stock	50 µl	• ABS plastic (similar to office electronics) • Nitrile rubber glove	significantly different. Not all temperatures and RH were tested due to test system limitations. **24°C at 20% RH**: $t_{1/2}$= 15.33±2.75 hours **24°C at 40% RH**: $t_{1/2}$= 11.52±1.72 hours **24°C at 60% RH**: $t_{1/2}$= 9.15±3.39 hours **24°C at 80% RH**: $t_{1/2}$= 8.33±1.80 hours **28°C at 40% RH**: $t_{1/2}$= 6.11±3.02 hours **35°C at 20% RH**: $t_{1/2}$= 7.33±1.33 hours **35°C at 40% RH**: $t_{1/2}$= 7.52±1.22 hours **35°C at 60% RH**: $t_{1/2}$= 2.26±1.42 hours
van Doremalen et al.[84]	21-23°C	40%	DMEM with 10% FBS	$10^{5.25}$ $TCID_{50}$/mL	50 µl	• Plastic • Stainless steel • Copper • Cardboard	**Plastic**: $10^{3.7}$ reduced to $10^{0.6}$ $TCID_{50}$/mL after 72 hours **Stainless steel**: $10^{3.7}$ reduced to $10^{0.6}$ $TCID_{50}$/mL after 48 hours **Copper**: Viable viruses detected up to 4 hours **Cardboard**: Viable viruses detected up to 24 hours
Liu et al.[85]	Room temperature	Not mentioned	Not mentioned	10^{6} $TCID_{50}$/mL	50 µl	• Plastic • Stainless steel • Glass • Ceramics • Wood • Latex gloves • Surgical mask • Cotton cloth • Paper	Remained viable for 7 days on **plastic, stainless steel, glass, ceramics, wood, latex gloves, and surgical mask** but declined slowly over the days. No viable virus on **cotton cloth** after 4 days. No viable virus on **paper** after 5 days
Harbourt et al.[54]	4°C± 2°C 22°C± 2°C 37°C ± 2°C	40-50%	EMEM* with 10% FBS at 5% CO2	4.5 ± 0.5log10 PFU (~$10^{6.4}$ $TCID_{50}$/mL)	50 µl	• Skin • Currency • Clothing	**At 4°C± 2°C:** Skin: remained viable for duration of experiment Clothing: remained viable up to 4 days Currency: remained viable up to 7 days **At 22°C± 2°C:** Skin: viable virus found at 4 days

							Clothing: remained viable up to 4 hours Currency: remained viable up to 24 hours **At 37°C ± 2°C:** Skin: remained viable up to 8 hours Clothing: Viable up to 4 hours Currency: Viable up to 8 hours At all temperatures the virus exhibited log reductions over time to varying degrees
Telang et al.[86]	34°C outdoors	54% outdoors	N/A	COVID-19 patients coughed into their hands	N/A	Fruits and vegetables	Subjects coughed into hands and manipulated fruits and vegetables at least 5 times. No SARS-CoV-2 RNA was found on any of the fruits and vegetables
Pastorino et al.[87]	19-21 °C	45-55 %	Culture medium containing 5% FBS Added BSA to examine effects of higher protein concentration	10^6 $TCID_{50}$/mL	50 µl	• Polystyrene plastic • Aluminum • Glass	**Plastic:** No BSA: Viable for duration of experiment (~$10^{3.3}$ $TCID_{50}$/mL at 4 days) BSA: Viable past 4 days (~$10^{4.1}$ $TCID_{50}$/mL at 4 days) **Aluminum:** No BSA: Viable up to 2 hours (~10^4 $TCID_{50}$/mL at 2 hours) BSA: Viable for duration of experiment (~$10^{3.6}$ $TCID_{50}$/mL at 4 days) **Glass:** No BSA: Viable up to 24 hours (~$10^{2.7}$ $TCID_{50}$/mL remaining at 24 hours) BSA: Viable throughout duration of experiment (~$10^{3.9}$ $TCID_{50}$/mL remaining at 4 days)

† Dulbecco's modified Eagle's medium

‡ Fetal bovine serum

* Eagle's minimum essential medium

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