SARS-CoV-2 Detection from Surface Samples

Subjects: Public, Environmental & Occupational Health

Contributor: Günter Kampf

Contaminated surfaces have been discussed as a possible source of severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2). Under experimental conditions, SARS-CoV-2 can remain infectious on surfaces for several days. However, the frequency of SARS-CoV-2 detection on surfaces in healthcare settings and the public is currently not known. A systematic literature review was performed. On surfaces around COVID-19 cases in healthcare settings (42 studies), the SARS-CoV-2 RNA detection rates mostly were between 0% and 27% (Ct values mostly > 30). Detection of infectious SARS-CoV-2 was only successful in one of seven studies in 9.2% of 76 samples. Most of the positive samples were obtained next to a patient with frequent sputum spitting during sampling. Eight studies were found with data from public surfaces and RNA detection rates between 0% and 22.1% (Ct values mostly >30). Detection of infectious virus was not attempted. Similar results were found in samples from surfaces around confirmed COVID-19 cases in non-healthcare settings (7 studies) and from personal protective equipment (10 studies). Therefore, it seems plausible to assume that inanimate surfaces are not a relevant source for transmission of SARS-CoV-2. In public settings, the associated risks of regular surface disinfection probably outweigh the expectable health benefits.

Keywords: SARS-CoV-2; surface; contamination; RNA; infectious virus

1. Introduction

The global spread of SARS-CoV-2 in 2020 has resulted in a variety of strategies for transmission control. Early laboratory data obtained after an artificial contamination of carrier surfaces with a high viral load suggested that coronaviruses in general may remain infectious on inanimate surfaces at room temperature for up to 9 days [1] and in the dark and in the presence of bovine serum albumin for even up to 28 days [2]. Similar results, though with much shorter stability times, were obtained with SARS-CoV-2 under laboratory settings [3]. The relevance of the rather long persistence on surfaces remains controversial because viruses from respiratory secretions are embedded in mucus and saliva, which probably contain specific antibodies against the virus, high numbers of leukocytes, and intrinsic antiviral activity because of their polyanionic charge which binds to viruses as well as bacteria and fungi, which may influence the environment around the virus [4]. The applicability of the findings to real life has also been questioned because in the studies, a high load of infectious virus was applied to a small surface area, which is much higher than those in droplets in real-life situations. As a result, the amount of virus actually deposited on surfaces could be several orders of magnitude smaller [5]. Nevertheless, these findings obtained under laboratory conditions raised the concern that viral shedders in the public may contaminate frequent touch surfaces, finally resulting in viral transmission via uncontrolled hand-face-contacts. As a result, many public surfaces were subjected to disinfection, e.g., in shops, museums, restaurants, public transportation, or sports facilities.

Recent data suggest that infectious SARS-CoV-2 is rarely found on surfaces around confirmed COVID-19 cases in healthcare settings despite variable detection rates of viral RNA $^{[6][7]}$. Laboratory data with SARS-CoV-2 show that Ct (cycle threshold) values of 29.3 (steel surface) or 29.5 (plastic surface) correlate with detection of culturable virus, whereas Ct values of 32.5 (steel surface) or 32.7 (plastic surface) correlate with the detection of non-culturable virus $^{[6]}$. It was implicated that a Ct value > 30 obtained from a surface sample has probably no epidemiological relevance $^{[6]}$. In contrast, dried inocula with Ct values < 30 (corresponding to an E gene copy number of \geq 10⁵ per mL) yielded SARS-CoV-2 that could be cultured $^{[6]}$. A simple binary approach to the interpretation of PCR results obtained from surface samples and not validated against viral culture will probably result in unnecessary, regular disinfection of surfaces $^{[8]}$. The frequency of SARS-CoV-2 detection by PCR on surfaces in healthcare settings and the public is currently not known. In addition, the corresponding Ct values have not been comprehensively evaluated.

2. SARS-CoV-2 Detection Rates from Surface Samples

SARS-CoV-2 is rarely detected on surfaces in the areas surrounding confirmed COVID-19 patients, mainly when a patient is coughing during sampling. In addition, viral RNA can be detected in variable proportions but mostly with Ct values > 30

suggesting a low viral RNA load. It is therefore assumed that surfaces in hospitals have probably no relevance as a potential source for transmission, especially when regular disinfection and cleaning is done as recommended by the WHO [9]. Similar findings were described for SARS-CoV-2 from public surfaces and PPE surfaces. The results are in line with very low detection rates of infectious influenza virus in 90 households (0%) or on 671 frequently touched surfaces in hospital rooms with confirmed influenza infection (0.3%) [10][11].

The CDC has recently published a science brief on the possible transmission of SARS-CoV-2 from surfaces and concluded that it is possible for people to be infected through contact with contaminated surfaces or objects (fomites), but the risk is generally considered to be low $\frac{[12]}{}$. Based on different quantitative microbial risk assessments, it was considered to be generally less than 1 in 10,000 $\frac{[13][14]}{}$. Under low viral bioburden conditions (<1 genome copy per cm²), it was described to be below 1:1,000,000 $\frac{[15]}{}$.

The major limitation of the currently available studies is the lack of evidence that COVID-19 patients in healthcare settings were still shedding infectious SARS-CoV-2, as only viral RNA was detected for confirmation of the diagnosis. It has been described that infectious SARS-CoV-2 is typically detected for 7 days in respiratory tract samples, whereas viral RNA may be found for up to 28 days after beginning of the symptoms [16][17]. If patients do not shed infectious SARS-CoV-2 anymore but only viral RNA, it would be plausible to detect mainly viral RNA on surfaces and only rarely infectious virus. Future research on surface contamination need to also address the question of whether the patient carries infectious SARS-CoV-2 at the time of surface sampling. Another limitation is that the incidence of COVID-19 in the various public settings described in the studies is variable and often not known.

Whereas regular and targeted disinfection of surfaces in the areas surrounding critically ill patients in healthcare settings remains an important measure to control the spread not only of viruses but also bacteria and fungi [18], there is currently no evidence that suggest an important role of fomite transmission in the public setting. The available data do not support the necessity of regular disinfection procedures of public surfaces as currently observed in many countries. WHO still recommends reducing potential for COVID-19 virus contamination in non-healthcare settings, such as in the home, office, schools, gyms, or restaurants [19]. High-touch surfaces in these non-health care settings should be identified for priority disinfection. These include door and window handles, kitchen and food preparation areas, counter tops, bathroom surfaces, toilets and taps, touchscreen personal devices, personal computer keyboards, and work surfaces [19]. CDC advocates the cleaning and disinfection of surfaces in community facilities only after persons with suspected or confirmed COVID-19 have been in the facility [20]. The Robert Koch Institute in Germany describes cleaning of surfaces as the preferred option because it is still unknown if a surface disinfection outside healthcare facilities is overall necessary. A routine disinfection at home or in public places, including surfaces with frequent hand contacts, is currently not recommended [21]. In public settings, the contamination with high-titre infectious virus is even less likely compared to the immediate surrounding of confirmed COVID-19 cases in healthcare settings or at home. Viral contamination can possibly occur in the unlikely event of a symptomatic or an asymptomatic COVID-19 case near the surface. However, unlike in patient rooms or the domestic setting, it is not expected that there is a permanent presence of a potential virus source next to the surface.

A possible transmission from surfaces could occur via transiently contaminated hands after contact with a viruscontaminated surface followed by a hand-nose or hand-mouth contact. Several studies have analysed the likelihood of fomite transmission for respiratory viruses. One study highlighted the importance of aerosols for rhinovirus transmission in contrast to a neglectable role for surfaces. In this study, two groups of men played poker, one group sick with the common cold and the other group healthy. The healthy group was exposed to infectious virus aerosols simply by being in the same room with the sick group; however, they were restrained so that participants could not touch their faces. Cards and chips used in the poker game were transferred to a group of healthy men to play with, and they were instructed to touch their faces frequently. Interestingly, the aerosol-exposed group got sick, while the surfaces-exposed group did not [22]. Another study could show that, on hands, only a small fraction of infectious virus is usually found after contact with artificially contaminated surfaces, such as 1.5% with parainfluenza virus and 0.7% with rhinovirus [23]. In addition, only a small fraction of the viral load can be transferred from contaminated hands to a surface (0% with parainfluenza virus and 0.9% with rhinovirus) [23]. Importantly, the risk of disease transmission by hand contact with a contaminated surface followed by a single hand-nose-contact is for rhinovirus low (0.0486%) and for influenza virus very low (0.0000000256%) [24]. Of note, seasonality of virus transmission should be considered when interpreting these results as some factors including humidity can directly influence aerosol stability. Under tropic conditions (warm and humid climates), aerosols or droplets evaporate less water and therefore readily settle on surfaces, which could favour fomite transmission as hypothesized for influenza viruses [25]. In addition, it was shown under experimental laboratory conditions at 24 °C that the half-life of SARS-CoV-2 infectivity is 15 h at 20% relative humidity, 12 h at 40% humidity, and 9 h at 60% humidity, suggesting a longer persistence of SARS-CoV-2 in dry air [26]. In addition, viral half-life was shorter at 35 °C compared to 24 °C [27]. Comparative data at 10 °C and 22 °C at different relative humidities show a longer persistence of SARS-CoV-2 at the lower temperature [27]. Nevertheless, hand washing is recommended for the public especially when returning home because the hands may also get contaminated from other people who are coughing or sneezing [28].

Especially in the public setting, as exemplified by a study analysing bus terminals in Brazil, it was interesting to see that all seven positive samples (RNA detection) were found at entrance handrails of the bus terminals. This may be explained by droplets coming from viral carriers close to the handrails. It may also be explained by SARS-CoV-2-positive passengers wearing face masks during coughing, sneezing, or talking because SARS-CoV-2 RNA may be found on the outer surface of a face mask. By touching the face mask, the hands may get contaminated, which may finally result in a handrail contamination. The corresponding Ct values, however, were so high that the RNA-positive handrails are probably not a relevant source of transmission because only a fraction of the virus remains on the hands after a hand-surface contact.

Cleaning of surfaces by a single, two second wipe has been described to reduce infectious coronavirus by 2.4 \log_{10} [29]. Similar results (2.5 \log_{10}) were obtained with a five second single wipe against ebolavirus [30]. These results suggest that in most settings, a simple cleaning procedure with a moist wipe will be sufficient to control the very low risk attributed to public surfaces.

A health benefit of regular disinfection of public surfaces is unlikely, given the currently assumed low transmission risk via this route. Furthermore, it is important to note that regular disinfection of surfaces also carries costs, such as reducing the diversity of the microbiome and increasing the diversity of bacterial resistance genes [31]. Microbiome diversity on surfaces is especially important for babies to ensure a balanced and healthy gut microflora [32]. An increased diversity of resistance genes enhances the occurrence of multi-resistant bacteria, which is a major burden for healthcare in Europe [33] and elsewhere. Permanent exposure of bacteria to subinhibitory concentrations of some biocidal agents used for surface disinfection can cause a strong, adaptive cellular response resulting in a stable tolerance to the biocidal agents and rarely, in a few species, in a new antibiotic resistance [34]. The daily number of calls to U.S. poison centres has substantially increased in 2020, mainly for bleach (+62.1%) and other disinfectants (+36.7%). Inhalation represented the largest percentage increase among all exposure routes (+35.3% for cleaners like bleach; +108.8% for all other disinfectants) [35]. The non-targeted, regular surface disinfection in many public places will probably have no health benefit but may have some negative side effects, similar to the broad non-targeted use of triclosan in the past [36].

3. Conclusions

Currently, available data do not support surfaces as a relevant source of SARS-CoV-2 transmission. In healthcare settings with confirmed COVID-19 cases, regular surface disinfection remains a precautionary element of infection control. In public settings, however, the associated risks and harms of regular surface disinfection probably outweigh the expected health benefits.

References

- 1. Kampf, G.; Todt, D.; Pfaender, S.; Steinmann, E. Persistence of coronaviruses on inanimate surfaces and its inactivation with biocidal agents. J. Hosp. Infect. 2020, 104, 246–251.
- 2. Riddell, S.; Goldie, S.; Hill, A.; Eagles, D.; Drew, T.W. The effect of temperature on persistence of SARS-CoV-2 on common surfaces. Virol. J. 2020, 17, 145.
- 3. Van Doremalen, N.; Bushmaker, T.; Morris, D.H.; Holbrook, M.G.; Gamble, A.; Williamson, B.N.; Tamin, A.; Harcourt, J.L.; Thornburg, N.J.; Gerber, S.I.; et al. Aerosol and surface stability of SARS-CoV-2 as compared with SARS-CoV-1. N. Engl. J. Med. 2020, 382, 1564–1567.
- 4. Eccles, R. Respiratory mucus and persistence of virus on surfaces. J. Hosp. Infect. 2020, 105, 350.
- 5. Goldman, E. Exaggerated risk of transmission of COVID-19 by fomites. Lancet Infect. Dis. 2020, 20, 892-893.
- 6. Zhou, J.; Otter, J.A.; Price, J.R.; Cimpeanu, C.; Garcia, D.M.; Kinross, J.; Boshier, P.R.; Mason, S.; Bolt, F.; Holmes, A.H.; et al. Investigating SARS-CoV-2 surface and air contamination in an acute healthcare setting during the peak of the COVID-19 pandemic in London. Clin. Infect. Dis. 2021, 72.
- 7. Ahn, J.Y.; An, S.; Sohn, Y.; Cho, Y.; Hyun, J.H.; Baek, Y.J.; Kim, M.H.; Jeong, S.J.; Kim, J.H.; Ku, N.S.; et al. Environmental contamination in the isolation rooms of COVID-19 patients with severe pneumonia requiring mechanical ventilation or high-flow oxygen therapy. J. Hosp. Infect. 2020, 106, 570–576.

- 8. Kampf, G.; Lemmen, S.; Suchomel, M. Ct values and infectivity of SARS-CoV-2 on surfaces. Lancet Infect. Dis 2021, 21
- WHO. Infection Prevention and Control during Health Care When Novel Coronavirus (NCOV) Infection Is Suspected. Interim Guidance. 19 March 2020. Available online: https://www.who.int/publications-detail/infection-prevention-and-control-during-health-care-when-novel-coronavirus-(ncov)-infection-is-suspected-20200125 (accessed on 16 April 2020).
- 10. Simmerman, J.M.; Suntarattiwong, P.; Levy, J.; Gibbons, R.V.; Cruz, C.; Shaman, J.; Jarman, R.G.; Chotpitayasunondh, T. Influenza virus contamination of common household surfaces during the 2009 influenza a (H1N1) pandemic in Bangkok, Thailand: Implications for contact transmission. Clin. Infect. Dis. Off. Publ. Infect. Dis. Soc. Am. 2010, 51, 1053–1061.
- 11. Killingley, B.; Greatorex, J.; Digard, P.; Wise, H.; Garcia, F.; Varsani, H.; Cauchemez, S.; Enstone, J.E.; Hayward, A.; Curran, M.D.; et al. The environmental deposition of influenza virus from patients infected with influenza a (H1N1)pdm09: Implications for infection prevention and control. J. Infect. Public Health 2016, 9, 278–288.
- 12. CDC. Science Brief: SARS-CoV-2 and Surface (Fomite) Transmission for Indoor Community Environments. 5 April 2021. Available online: https://www.cdc.gov/coronavirus/2019-ncov/more/science-and-research/surface-transmission.html (accessed on 5 May 2020).
- 13. Harvey, A.P.; Fuhrmeister, E.R.; Cantrell, M.E.; Pitol, A.K.; Swarthout, J.M.; Powers, J.E.; Nadimpalli, M.L.; Julian, T.R.; Pickering, A.J. Longitudinal monitoring of SARS-CoV-2 RNA on high-touch surfaces in a community setting. Environ. Sci. Technol. Lett. 2021, 8, 168–175.
- 14. Pitol, A.K.; Julian, T.R. Community transmission of SARS-CoV-2 by surfaces: Risks and risk reduction strategies. Environ. Sci. Technol. Lett. 2021, 8, 263–269.
- 15. Wilson, A.M.; Weir, M.H.; Bloomfield, S.F.; Scott, E.A.; Reynolds, K.A. Modeling COVID-19 infection risks for a single hand-to-fomite scenario and potential risk reductions offered by surface disinfection. Am. J. Infect. Control 2020.
- 16. Wolfel, R.; Corman, V.M.; Guggemos, W.; Seilmaier, M.; Zange, S.; Muller, M.A.; Niemeyer, D.; Jones, T.C.; Vollmar, P.; Rothe, C.; et al. Virological assessment of hospitalized patients with COVID-2019. Nature 2020, 581, 465–469.
- 17. To, K.K.; Tsang, O.T.; Leung, W.S.; Tam, A.R.; Wu, T.C.; Lung, D.C.; Yip, C.C.; Cai, J.P.; Chan, J.M.; Chik, T.S.; et al. Temporal profiles of viral load in posterior oropharyngeal saliva samples and serum antibody responses during infection by SARS-CoV-2: An observational cohort study. Lancet Infect. Dis. 2020, 20, 565–574.
- 18. Weber, D.J.; Anderson, D.; Rutala, W.A. The role of the surface environment in healthcare-associated infections. Curr. Opin. Infect. Dis. 2013, 26, 338–344.
- 19. WHO. Cleaning and Disinfection of Environmental Surfaces in the Context of COVID-19. Interim Guidance. 19 May 2020. Available online: https://www.who.int/publications/i/item/cleaning-and-disinfection-of-environmental-surfaces-inthe-context-of-COVID-19 (accessed on 18 August 2020).
- 20. CDC. Cleaning and Disinfection for Community Facilities. Interim Recommendations for U.S. Community Facilities with Suspected/Confirmed Coronavirus Disease 2019 (COVID-19). 27 May 2020. Available online: https://www.cdc.gov/coronavirus/2019-ncov/community/organizations/cleaning-disinfection.html (accessed on 18 August 2020).
- 21. Robert Koch-Institut. Hinweise zu Reinigung und Desinfektion von Oberflächen Außerhalb von Gesundheitseinrichtungen im Zusammenhang Mit der COVID-19-Pandemie. Available online: https://www.rki.de/DE/Content/InfAZ/N/Neuartiges_Coronavirus/Reinigung_Desinfektion.html (accessed on 4 April 2020).
- 22. Dick, E.C.; Jennings, L.C.; Mink, K.A.; Wartgow, C.D.; Inhorn, S.L. Aerosol transmission of rhinovirus colds. J. Infect. Dis. 1987, 156, 442–448.
- 23. Ansari, S.A.; Springthorpe, V.S.; Sattar, S.A.; Rivard, S.; Rahman, M. Potential role of hands in the spread of respiratory viral infections: Studies with human parainfluenza virus 3 and rhinovirus 14. J. Clin. Microbiol. 1991, 29, 2115–2119.
- 24. Wilson, A.M.; Reynolds, K.A.; Sexton, J.D.; Canales, R.A. Modeling surface disinfection needs to meet microbial risk reduction targets. Appl. Environ. Microbiol. 2018, 84, e00709–e00718.
- 25. Moriyama, M.; Hugentobler, W.J.; Iwasaki, A. Seasonality of respiratory viral infections. Ann. Rev. Virol. 2020, 7, 83–101.
- 26. Biryukov, J.; Boydston, J.A.; Dunning, R.A.; Yeager, J.J.; Wood, S.; Reese, A.L.; Ferris, A.; Miller, D.; Weaver, W.; Zeitouni, N.E.; et al. Increasing temperature and relative humidity accelerates inactivation of SARS-CoV-2 on surfaces. mSphere 2020, 5.

- 27. Morris, D.H.; Yinda, K.C.; Gamble, A.; Rossine, F.W.; Huang, Q.; Bushmaker, T.; Fischer, R.J.; Matson, M.J.; Van Doremalen, N.; Vikesland, P.J.; et al. Mechanistic theory predicts the effects of temperature and humidity on inactivation of SARS-CoV-2 and other enveloped viruses. eLife 2021, 10, e65902.
- 28. Jefferson, T.; Del Mar, C.; Dooley, L.; Ferroni, E.; Al-Ansary, L.A.; Bawazeer, G.A.; van Driel, M.L.; Foxlee, R.; Rivetti, A. Physical interventions to interrupt or reduce the spread of respiratory viruses: Systematic review. BMJ Clin. Res. 2009, 339, b3675.
- 29. Malenovská, H. Coronavirus persistence on a plastic carrier under refrigeration conditions and its reduction using wet wiping technique, with respect to food safety. Food Environ. Virol. 2020, 12, 1–6.
- 30. Cutts, T.A.; Robertson, C.; Theriault, S.S.; Nims, R.W.; Kasloff, S.B.; Rubino, J.R.; Ijaz, M.K. Assessing the contributions of inactivation, removal, and transfer of ebola virus and vesicular stomatitis virus by disinfectant presoaked wipes. Front. Public Health 2020, 8, 183.
- 31. Mahnert, A.; Moissl-Eichinger, C.; Zojer, M.; Bogumil, D.; Mizrahi, I.; Rattei, T.; Martinez, J.L.; Berg, G. Man-made microbial resistances in built environments. Nat. Commun. 2019, 10, 968.
- 32. Tun, M.H.; Tun, H.M.; Mahoney, J.J.; Konya, T.B.; Guttman, D.S.; Becker, A.B.; Mandhane, P.J.; Turvey, S.E.; Subbarao, P.; Sears, M.R.; et al. Postnatal exposure to household disinfectants, infant gut microbiota and subsequent risk of overweight in children. Can. Med. Assoc. J. 2018, 190, e1097–e1107.
- 33. Cassini, A.; Hogberg, L.D.; Plachouras, D.; Quattrocchi, A.; Hoxha, A.; Simonsen, G.S.; Colomb-Cotinat, M.; Kretzschmar, M.E.; Devleesschauwer, B.; Cecchini, M.; et al. Attributable deaths and disability-adjusted life-years caused by infections with antibiotic-resistant bacteria in the eu and the european economic area in 2015: A population-level modelling analysis. Lancet Infect. Dis. 2018, 19, 56–66.
- 34. Kampf, G. Antiseptic Stewardship: Biocide Resistance and Clinical Implications; Springer International Publishing: Cham, Switzerland, 2018.
- 35. Chang, A.; Schnall, A.H.; Law, R.; Bronstein, A.C.; Marraffa, J.M.; Spiller, H.A.; Hays, H.L.; Funk, A.R.; Mercurio-Zappala, M.; Calello, D.P.; et al. Cleaning and Disinfectant Chemical Exposures and Temporal Associations with COVID-19-National Poison Data System, United States, 1 January 2020–31 March 2020. MMWR Morb. Mortal. Wkly. Rep. 2020, 69, 496–498.
- 36. McNamara, P.J.; Levy, S.B. Triclosan: An instructive tale. Antimicrob. Agents Chemother. 2016, 60, 7015–7016.

Retrieved from https://encyclopedia.pub/entry/history/show/30253