

Aerosol and Surface Stability of SARS-CoV-2 as Compared with SARS-CoV-1

Neeltje van Doremalen, Ph.D., Trenton Bushmaker, B.Sc., [...], and Vincent J. Munster, Ph.D.

To the Editor: A novel human coronavirus that is now named severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) (formerly called HCoV-19) emerged in Wuhan, China, in late 2019 and is now causing a pandemic.¹ We analyzed the aerosol and surface stability of SARS-CoV-2 and compared it with SARS-CoV-1, the most closely related human coronavirus.²

We evaluated the stability of SARS-CoV-2 and SARS-CoV-1 in aerosols and on various surfaces and estimated their decay rates using a Bayesian regression model (see the Methods section in the [Supplementary Appendix](#), available with the full text of this letter at NEJM.org). SARS-CoV-2 nCoV-WA1-2020 (MN985325.1) and SARS-CoV-1 Tor2 (AY274119.3) were the strains used. Aerosols (<5 µm) containing SARS-CoV-2 ($10^{5.25}$ 50% tissue-culture infectious dose [TCID₅₀] per milliliter) or SARS-CoV-1 ($10^{6.75-7.00}$ TCID₅₀ per milliliter) were generated with the use of a three-jet Collison nebulizer and fed into a Goldberg drum to create an aerosolized environment. The inoculum resulted in cycle-threshold values between 20 and 22, similar to those observed in samples obtained from the upper and lower respiratory tract in humans.

Our data consisted of 10 experimental conditions involving two viruses (SARS-CoV-2 and SARS-CoV-1) in five environmental conditions (aerosols, plastic, stainless steel, copper, and cardboard). All experimental measurements are reported as means across three replicates.

SARS-CoV-2 remained viable in aerosols throughout the duration of our experiment (3 hours), with a reduction in infectious titer from $10^{3.5}$ to $10^{2.7}$ TCID₅₀ per liter of air. This reduction was similar to that observed with SARS-CoV-1, from $10^{4.3}$ to $10^{3.5}$ TCID₅₀ per milliliter (Figure 1A).

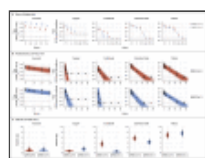


Figure 1
Viability of SARS-CoV-1 and SARS-CoV-2 in Aerosols and on Various Surfaces.

SARS-CoV-2 was more stable on plastic and stainless steel than on copper and cardboard, and viable virus was detected up to 72 hours after application to these surfaces (Figure 1A), although the virus titer was greatly reduced (from $10^{3.7}$ to $10^{0.6}$ TCID₅₀ per milliliter of medium after 72 hours on plastic and from $10^{3.7}$ to $10^{0.6}$ TCID₅₀ per milliliter after 48 hours on stainless steel). The stability kinetics of SARS-CoV-1 were similar (from $10^{3.4}$ to $10^{0.7}$ TCID₅₀ per milliliter after 72 hours on plastic and from $10^{3.6}$ to $10^{0.6}$ TCID₅₀ per milliliter after 48 hours on stainless steel). On copper, no viable SARS-CoV-2 was measured after 4 hours and no viable SARS-CoV-1 was measured after 8 hours. On cardboard, no viable SARS-CoV-2 was measured after 24 hours and no viable SARS-CoV-1 was measured after 8 hours (Figure 1A).

Both viruses had an exponential decay in virus titer across all experimental conditions, as indicated by a linear decrease in the log₁₀TCID₅₀ per liter of air or milliliter of medium over time (Figure 1B). The half-lives of SARS-CoV-2 and SARS-CoV-1 were similar in aerosols, with median estimates of approximately 1.1 to 1.2 hours and 95% credible intervals of 0.64 to 2.64 for SARS-CoV-2 and 0.78 to 2.43 for SARS-CoV-1 (Figure 1C, and Table S1 in the [Supplementary Appendix](#)). The half-lives of the two viruses were also similar on copper. On cardboard, the half-life of SARS-CoV-2 was longer than that of SARS-CoV-1. The longest viability of both viruses was on stainless steel and plastic; the estimated median half-life of SARS-CoV-2 was approximately 5.6 hours on stainless steel and 6.8 hours on plastic (Figure 1C). Estimated differences in the half-lives of the two viruses were small except for those on cardboard (Figure 1C). Individual replicate data were noticeably “noisier” (i.e., there was more variation in the experiment, resulting in a larger standard error) for cardboard than for other surfaces (Fig. S1 through S5), so we advise caution in interpreting this result.

We found that the stability of SARS-CoV-2 was similar to that of SARS-CoV-1 under the experimental circumstances tested. This indicates that differences in the epidemiologic characteristics of these viruses probably arise from other factors, including high viral loads in the upper respiratory tract and the potential for persons infected with SARS-CoV-2 to shed and transmit the virus while asymptomatic.^{3,4} Our results indicate that aerosol and fomite transmission of SARS-CoV-2 is plausible, since the virus can remain viable and infectious in aerosols for hours and on surfaces up to days (depending on the inoculum shed). These findings echo those with SARS-CoV-1, in which these forms of transmission were associated with nosocomial spread and super-spreading events,⁵ and they provide information for pandemic mitigation efforts.

Supplementary Appendix

[Click here for additional data file.](#) (1.4M, pdf)

Disclosure Forms

[Click here for additional data file.](#)^(276K, pdf)

Notes

The findings and conclusions in this letter are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention (CDC). Names of specific vendors, manufacturers, or products are included for public health and informational purposes; inclusion does not imply endorsement of the vendors, manufacturers, or products by the CDC or the Department of Health and Human Services.

This letter was published on March 17, 2020, at NEJM.org.

Footnotes

Supported by the Intramural Research Program of the National Institute of Allergy and Infectious Diseases, National Institutes of Health, and by contracts from the Defense Advanced Research Projects Agency (DARPA PREEMPT No. D18AC00031, to Drs. Lloyd-Smith and Gamble), from the National Science Foundation (DEB-1557022, to Dr. Lloyd-Smith), and from the Strategic Environmental Research and Development Program of the Department of Defense (SERDP, RC-2635, to Dr. Lloyd-Smith).

[Disclosure forms](#) provided by the authors are available with the full text of this letter at NEJM.org.

Article information

N Engl J Med. 2020 Mar 17 : NEJMc2004973.

Published online 2020 Mar 17. doi: [10.1056/NEJMc2004973](https://doi.org/10.1056/NEJMc2004973)

PMCID: PMC7121658

PMID: [32182409](https://pubmed.ncbi.nlm.nih.gov/32182409/)

[Neeltje van Doremalen](#), Ph.D. and [Trenton Bushmaker](#), B.Sc.

National Institute of Allergy and Infectious Diseases, Hamilton, MT

[Dylan H. Morris](#), M.Phil.

Princeton University, Princeton, NJ

[Myndi G. Holbrook](#), B.Sc.

National Institute of Allergy and Infectious Diseases, Hamilton, MT

[Amandine Gamble](#), Ph.D.

University of California, Los Angeles, Los Angeles, CA

[Brandi N. Williamson](#), M.P.H.

National Institute of Allergy and Infectious Diseases, Hamilton, MT

[Azaibi Tamin](#), Ph.D., [Jennifer L. Harcourt](#), Ph.D., [Natalie J. Thornburg](#), Ph.D., and [Susan I. Gerber](#), M.D.

Centers for Disease Control and Prevention, Atlanta, GA

[James O. Lloyd-Smith](#), Ph.D.

University of California, Los Angeles, Los Angeles, CA, Bethesda, MD

[Emmie de Wit](#), Ph.D. and [Vincent J. Munster](#), Ph.D. 

National Institute of Allergy and Infectious Diseases, Hamilton, MT vincent.munster@nih.gov

 Corresponding author.

Dr. van Doremalen, Mr. Bushmaker, and Mr. Morris contributed equally to this letter.

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See commentary "[Stability and Viability of SARS-CoV-2](#)" in *N Engl J Med*, volume 382 on page 1962.

This article is based on a previously available preprint: "[Aerosol and surface stability of HCoV-19 \(SARS-CoV-2\) compared to SARS-CoV-1](#)".

This article has been [cited](#) by other articles in PMC.

References

1. Coronavirus disease (COVID-2019) situation reports. Geneva: World Health Organization, 2020. (<https://www.who.int/emergencies/diseases/novel-coronavirus-2019/situation-reports/>).
2. Wu A, Peng Y, Huang B, et al. Genome composition and divergence of the novel coronavirus (2019-nCoV) originating in China. *Cell Host Microbe* 2020;27:325-328. [[PMC free article](#)] [[PubMed](#)] [[Google Scholar](#)]
3. Bai Y, Yao L, Wei T, et al. Presumed asymptomatic carrier transmission of COVID-19. *JAMA* 2020. February 21 (Epub ahead of print). [[PMC free article](#)] [[PubMed](#)] [[Google Scholar](#)]
4. Zou L, Ruan F, Huang M, et al. SARS-CoV-2 viral load in upper respiratory specimens of infected patients. *N Engl J Med*. DOI: 10.1056/NEJMc2001737. [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
5. Chen YC, Huang LM, Chan CC, et al. SARS in hospital emergency room. *Emerg Infect Dis* 2004;10:782-788. [[PMC free article](#)] [[PubMed](#)] [[Google Scholar](#)]