

Availability of diagnostics and antifungals, and training in their use, will reduce deaths from advanced HIV disease (by up to 30%).² Mistaken diagnoses of pulmonary tuberculosis when actually the problem is a fungal lung infection will be averted. Implementation of these priorities will strengthen public health systems, support antimicrobial stewardship,⁹ develop clinician skills, and appropriately diversify differential diagnosis. New approaches have to be explored, such as the implementation of artificial intelligence, to address the shortage of health-care workers in the Latin American and Caribbean region, Africa, and southeast Asia. We anticipate that the enhancement, innovation, and increased integration of fungal disease diagnosis and management within the health system will benefit not only those with fungal disease, but also improve the effectiveness, efficiency, and quality of the entire health-care system.

We declare no competing interests.

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- 1 Global Fungal Infection Forum 4 in Lima. Geneva, London: Global Action Fund for Fungal Infections, 2019. <https://www.gaffi.org/global-fungal-infection-forum-4-in-lima/> (accessed June 3, 2020).
- 2 Denning DW. Minimizing fungal disease deaths will allow the UNAIDS target of reducing annual AIDS deaths below 500 000 by 2020 to be realized. *Philos Trans R Soc Lond B Biol Sci* 2016; **371**: 20150468.
- 3 Samayoa B, Aguirre L, Bonilla O, et al. The diagnostic laboratory hub: a new health care system reveals the incidence and mortality of tuberculosis, histoplasmosis, and cryptococcosis of PWH in Guatemala. *Open Forum Infect Dis* 2019; **7**: ofz534.
- 4 Bongomin F, Govender NP, Chakrabarti A, et al. Essential in vitro diagnostics for advanced HIV and serious fungal diseases: international experts' consensus recommendations. *Eur J Clin Microbiol Infect Dis* 2019; **38**: 1581–84.
- 5 WHO. Second WHO model list of essential in vitro diagnostics. Geneva: World Health Organization, 2019. https://www.who.int/docs/default-source/nutritionlibrary/complementary-feeding/second-who-model-list-v8-2019.pdf?sfvrsn=6fe86adf_1 (accessed June 3, 2020).
- 6 WHO. Global Antimicrobial Resistance Surveillance System: early implementation protocol for inclusion of *Candida* spp. Geneva: World Health Organization, August, 2019. <https://apps.who.int/iris/bitstream/handle/10665/326926/WHO-WSI-AMR-2019.4-eng.pdf?sequence=1&isAllowed=y> (accessed June 3, 2020).
- 7 Cole DC, Govender NP, Chakrabarti A, Sacarlal J, Denning DW. Improvement of fungal disease identification and management: combined health systems and public health approaches. *Lancet Infect Dis* 2017; **17**: e412–19.
- 8 Pan-American Health Organization. PAHO strategic fund. Washington, DC: Pan-American Health Organization. https://www.paho.org/hq/index.php?option=com_content&view=article&id=12163:paho-strategic-fund&Itemid=1694&lang=en
- 9 Denning DW, Perlin DS, Muldoon EG, et al. Delivering an antimicrobial resistance agenda not possible without improving fungal diagnostic capabilities. *Emerg Infect Dis* 2017; **23**: 177–83.



Exaggerated risk of transmission of COVID-19 by fomites

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A clinically significant risk of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) transmission by fomites (inanimate surfaces or objects) has been assumed on the basis of studies that have little resemblance to real-life scenarios.

The longest survival (6 days) of severe acute respiratory syndrome coronavirus (SARS-CoV) on surfaces was done by placing a very large initial virus titre sample (10^7 infectious virus particles) on the surface being tested.¹ Another study that claimed survival of 4 days used a similarly large sample (10^6 infectious virus particles) on the surface.² A report by van Doremalen and colleagues found survival of both SARS-CoV and SARS-CoV-2 of up to 2 days (on surfaces) and 3 days (in aerosols generated in the laboratory), but again with a large inoculum (10^5 – 10^7 infectious virus particles per mL in aerosols, 10^4 infectious virus particles on surfaces).³ Yet another study found long survival (5 days)

of human coronavirus 229E on surfaces with what I would still consider a substantially large viral load (10^3 plaque-forming units) in a cell lysate.⁴ However, using a cell lysate rather than purified or semipurified virus might enable initial viral proliferation or protection from the effects of the sample drying out.

None of these studies present scenarios akin to real-life situations. Although I did not find measurements of coronavirus quantities in aerosol droplets from patients, the amount of influenza virus RNA in aerosols has been measured, with a concentration equivalent to 10–100 viral particles in a droplet, with even fewer infectious influenza virus particles capable of growth in a plaque assay.⁵ By contrast, one study found human coronavirus 229E to survive for only 3–6 h (depending on the surface tested), and human coronavirus OC43 to survive for 1 h, after drying on various surfaces including aluminum, sterile latex surgical gloves, and

sterile sponges.⁶ In a study in which the authors tried to mimic actual conditions in which a surface might be contaminated by a patient, no viable SARS-CoV was detected on surfaces.⁷

A 2020 literature review⁸ included most of the studies I have cited here (and others), but adds no new research, and in my view, does not critically evaluate previously published studies. I am not disputing the findings of these studies, only the applicability to real life. For example, in the studies that used a sample of 10^7 , 10^6 , and 10^4 particles of infectious virus on a small surface area,¹⁻³ these concentrations are a lot higher than those in droplets in real-life situations, with the amount of virus actually deposited on surfaces likely to be several orders of magnitude smaller.⁵ Hence, a real-life situation is better represented in the work of Dowell and colleagues⁷ in which no viable virus was found on fomites.

In my opinion, the chance of transmission through inanimate surfaces is very small, and only in instances where an infected person coughs or sneezes on the surface, and someone else touches that surface soon after the cough or sneeze (within 1–2 h). I do not disagree with erring on the side of caution, but this can go to extremes not justified by the data. Although periodically disinfecting surfaces and use of gloves

are reasonable precautions especially in hospitals, I believe that fomites that have not been in contact with an infected carrier for many hours do not pose a measurable risk of transmission in non-hospital settings. A more balanced perspective is needed to curb excesses that become counterproductive.

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- 1 Rabenau HF, Cinatl J, Morgenstern B, Bauer G, Preiser W, Doerr HW. Stability and inactivation of SARS coronavirus. *Med Microbiol Immunol* 2005; **194**: 1–6.
- 2 Duan SM, Zhao XS, Wen RF, Huang JJ, Pi GH, Zhang SX. Stability of SARS coronavirus in human specimens and environment and its sensitivity to heating and UV irradiation. *Biomed Environ Sci* 2003; **16**: 246–55.
- 3 van Doremalen N, Bushmaker T, Morris DH, et al. Aerosol and surface stability of SARS-CoV-2 as compared with SARS-CoV-1. *N Engl J Med* 2020; **382**: 1564–67.
- 4 Warnes SL, Little ZR, Keevil CW. Human coronavirus 229E remains infectious on common touch surface materials. *mBio* 2015; **6**: e01697–15.
- 5 Lindsley WG, Blachere FM, Thewlis RE, et al. Measurements of airborne influenza virus in aerosol particles from human coughs. *PLoS One* 2010; **5**: e15100.
- 6 Sizun J, Yu MW, Talbot PJ. Survival of human coronaviruses 229E and OC43 in suspension and after drying on surfaces: a possible source of hospital-acquired infections. *J Hosp Infect* 2000; **46**: 55–60.
- 7 Dowell SF, Simmerman JM, Erdman DD, et al. Severe acute respiratory syndrome coronavirus on hospital surfaces. *Clin Infect Dis* 2004; **39**: 652–57.
- 8 Kampf G, Todt D, Pfaender S, Steinmann E. Persistence of coronaviruses on inanimate surfaces and their inactivation with biocidal agents. *J Hosp Infect* 2020; **104**: 246–51.