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## Monkeypox and the health-care environment



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Historically, fewer than a dozen cases of health care-associated human monkeypox have been reported in endemic African countries, including in the Democratic Republic of Congo (1983),<sup>1</sup> Republic of the Congo (2003),<sup>2</sup> Central African Republic (2015–16),<sup>3</sup> and Nigeria (2017–18).<sup>4</sup> Although all of these exposures occurred in hospital environments, the exact modes of transmission could not be identified. Among healthcare-associated monkeypox cases exported to countries outside of Africa between 2003 and 2021, at least 250 health-care workers have had variable unprotected exposures to the monkeypox virus in the hospital setting;<sup>5</sup> however, only one case of nosocomial transmission has been reported in the literature.<sup>6</sup> This case was a health-care worker diagnosed in the UK in 2018 whose only identifiable exposure risk was the changing of potentially contaminated bedding of a patient with confirmed monkeypox.<sup>6</sup>

Environmental contamination of the household of monkeypox cases<sup>7</sup> and patient care environment<sup>8</sup> with monkeypox virus DNA has been reported, including detection of replication-competent virus from a household 3 days after the patient had been admitted to hospital.<sup>7</sup> Animal models suggest that aerosol transmission of monkeypox virus is possible, but corroborating human studies have been scarce.

Published in *The Lancet Microbe*, Susan Gould and colleagues<sup>9</sup> investigated surface and aerosol-related monkeypox virus DNA contamination of the environment in four respiratory isolation rooms in the Royal Free Hospital (London, UK). The isolation rooms were occupied at various times between May 24 and June 17, 2022, by six patients with laboratory-confirmed symptomatic monkeypox. 73 environmental surfaces were swabbed in patient rooms, including high-touch areas, floors of anterooms, air vents, bathrooms, and personal protective equipment (PPE) worn by health-care workers. Air samples were also collected from patient rooms (including before and during bedding changes), anterooms, and corridors adjacent to isolation rooms. Analysis of the extracted nucleic acid from all samples showed that 56 (93%) of the 60 samples were positive for monkeypox virus DNA, with crossing threshold (Ct) values of 24.7–37.4. Surface contamination included high-touch areas in patient

rooms, bathroom surfaces, anteroom floors after PPE doffing, and health-care worker PPE after use. Five of 15 air samples were positive, including one air sample collected in the anteroom before doffing, and four samples collected before and during bedding changes in two patient rooms. The detection of monkeypox virus on non-touch surfaces—such as air vents and surfaces at distances relatively far (>1.5 m) from patient beds—led the authors to propose that monkeypox virus DNA in aerosols, skin flakes, or dust, could be suspended in the air through respiratory droplets or from activities such as changing bedding. Viral isolation was attempted in four samples, and replication-competent virus was identified in two of the four samples, including from the floor of an anteroom after PPE doffing and from the air samples collected near the patient bed during a bedding change.

Gould and colleagues' findings further substantiate the potential of the monkeypox virus to contaminate diverse environmental surfaces within and outside the immediate vicinity of the patient care environment. The identification of viable monkeypox virus in air samples during bedding changes is novel and noteworthy, especially as the only confirmed case of healthcare-associated monkeypox in non-endemic countries was associated with the changing of bedding in a patient room.<sup>5,6</sup> However, this finding must be interpreted with caution as only one air sample from one patient room yielded replication-competent virus and the identification of viable virus does not necessarily translate to potential aerosol transmission of monkeypox virus in human populations.

Overall, this study highlights some key points relevant to public health. First, the widespread surface contamination of the patient care environment calls for a systematic standardised approach to surface cleaning of hospital rooms and households of patients diagnosed with monkeypox. Second, the detection of monkeypox virus DNA on PPE, in doffing areas, and in air samples at various distances from the patient bed and during bed linen change reiterates the importance of appropriate use and removal of PPE by health-care workers to prevent exposure to monkeypox virus during patient care. Third, since the changing of bedding might be associated with the suspension of monkeypox virus particles, staff should protect their mucous membrane

by wearing surgical masks (with or without face shield) at a minimum. Fourth, in view of some of the study limitations highlighted by Gould and colleagues in their Article, including the small numbers of viral cultures, further studies are needed to understand the infectious potential of fomites and aerosols in the transmission of monkeypox virus in patient-care settings. Until asymptomatic aerosol-related transmission is proven, current epidemiological data does not support airborne transmission of the monkeypox virus.

We declare no competing interests.

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