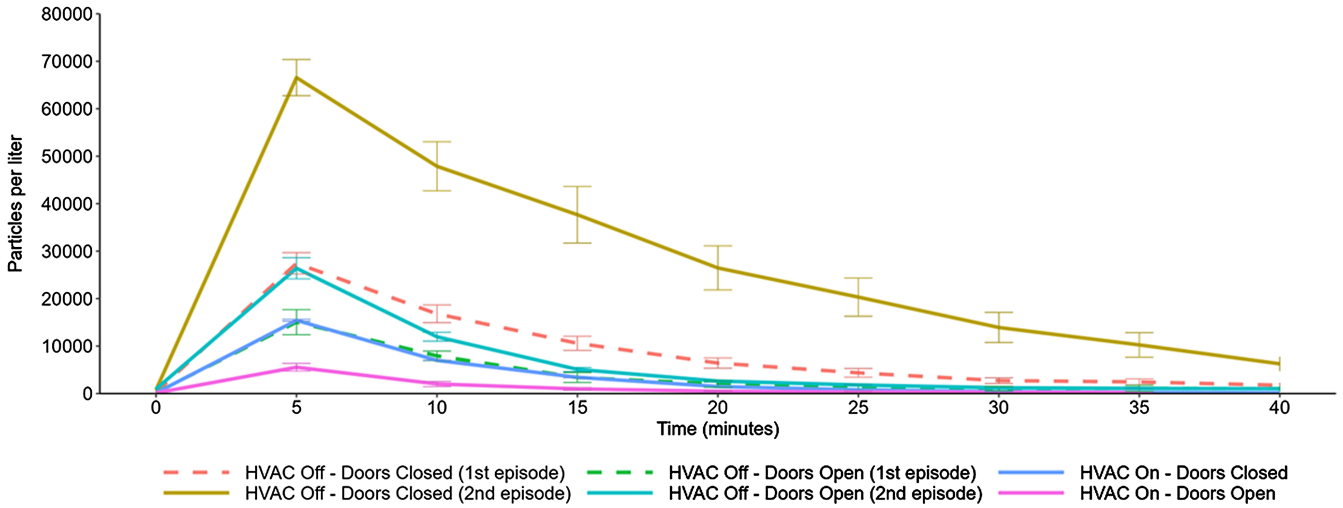


**Figure 1.** Clearance of 5% sodium chloride solution aerosol particles (1–10 μm in diameter) in patient rooms with the door open versus closed during periods when the heating, ventilation, and air conditioning (HVAC) system was off for maintenance versus on. During episode 1, the air handler was able to vent some recirculated air to the outside. Average results for 3 rooms for each test condition are shown. Error bars represent standard error.

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# Discordant antimicrobial susceptibility and polymerase chain reaction (PCR) testing in a *Klebsiella pneumoniae* isolate with a carbapenemase gene

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*To the Editor*—Rapid blood-culture identification techniques are increasingly common in hospitals across the United States. Rapid diagnostics can quickly identify resistance genes in bacteria that may otherwise have taken days to be identified, thus shortening the time until patients are placed on the appropriate

transmission-based precautions and antimicrobial treatment. Because shortened time to appropriate therapy has been linked to improved mortality, this change has critical implications for patient care.[1] Although numerous studies have described early identification of resistance genes that are later confirmed by traditional antimicrobial susceptibility testing (AST), few have reported cases of gram-negative bacteria with resistance genes identified by rapid tests but with AST showing antibiotic susceptibility. We present a case of a patient with a *Klebsiella pneumoniae* carbapenemase (KPC) gene identified by the ePlex Blood Culture Identification (BCID) panel (GenMark, Carlsbad, CA) whose subsequent AST showed susceptibility to carbapenems.

A man aged in his thirties with a medical history of necrotizing pancreatitis and type 2 diabetes mellitus was admitted to the intensive care unit for abdominal pain and diabetic ketoacidosis. Blood cultures collected on admission became positive 12 hours later for gram-negative rods. A known abdominal fluid collection was postulated as the source of his bacteremia. BCID detected *Klebsiella pneumoniae* and a KPC gene, and the patient was placed on contact precautions. Subsequent AST using the Vitek2 GN74 card (bioMérieux, Durham, NC) showed *Klebsiella pneumoniae* with susceptibility to meropenem (minimum inhibitory concentration [MIC] $\leq$ 0.25 μg/mL) and ertapenem (MIC $\leq$ 0.5 μg/mL) (Supplementary Table 1 online). Given this discrepancy, the isolate was sent to our investigational clinical microbiology core, where testing by Carba NP (performed according to guidelines[2]) showed carbapenemase activity. Gradient diffusion testing revealed a main population of bacteria susceptible to meropenem; however, satellite colonies were noted within the zone of inhibition (Fig. 1). Further testing in a research laboratory showed heteroresistance to meropenem. The patient was transitioned to ceftazidime-avibactam, and his blood cultures cleared within 48 hours.

This phenomenon (presence of resistance genes on PCR, and antibiotic susceptibility on AST) has been well documented in *Staphylococcus* and other gram-positive species.[3] Suggested mechanisms for this include empty *mec* cassettes or multiple populations of bacteria yielding conflicting results. Few prior studies have mentioned similar discrepancies in Enterobacterales.[4–7] Bratu et al[5] described a multihospital outbreak of carbapenem-resistant Enterobacterales (CRE) in which many isolates appeared susceptible to imipenem (despite possessing KPC genes) when a lower inoculum was used during standard broth microdilution testing.[5] Other studies suggest that when using more modern AST techniques, an unexpressed carbapenemase gene may lead to apparent susceptibility on standard AST despite presence of the resistance gene.[4,6] Our findings suggest that heteroresistance, whereby an established subpopulation of resistant bacteria proliferates under antibiotic pressure, may also play a role in these discrepant results.

The Centers for Disease Control and Prevention (CDC) recommends that healthcare workers use gowns and gloves when caring for patients with CRE and that these patients be placed in single-bed rooms when available (ie, contact precautions). Prior to instituting BCID, this patient would not have been identified as harboring CRE and would not have been placed under contact precautions. Surveillance for CRE in cultures, along with isolating and placing patients with CRE under contact precautions, have been shown to reduce the transmission of this class of organisms.[8,9] Our findings suggest that relying solely on AST to guide isolation decisions may miss some carbapenemase-producing CRE, potentially increasing the chance of undetected patient-to-patient transmission. As rapid diagnostics become more prevalent, more discrepancies between gene detection and AST will be identified.

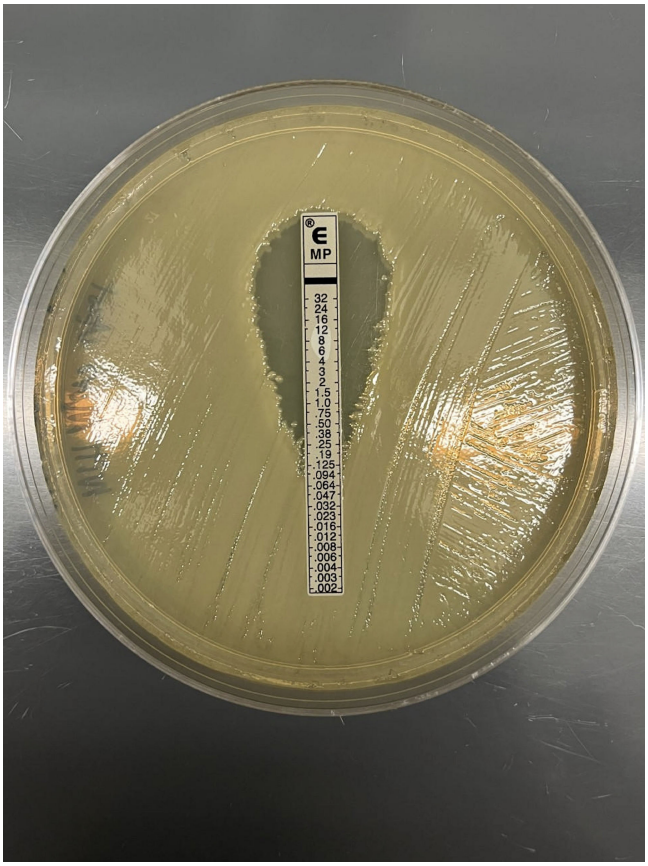


**Figure 1.** Gradient diffusion susceptibility test of meropenem showing colonies within the zone of inhibition.

Further analysis should be undertaken to determine the transmission risk of unexpressed carbapenemase genes and their implications for infection control and prevention.

**Supplementary material.** To view supplementary material for this article, please visit https://doi.org/10.1017/ice.2023.176

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**Conflict of interest.** All authors report no conflicts of interest relevant to this article.

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# Viable mpox in the inanimate environmental and risk of transmission

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As of August 23, 2023, 30,767 mpox cases have been reported in the United States (https://www.cdc.gov/poxvirus/mpox/response/2022/index.html). Although mpox is primarily transmitted through contact with an infected individual, recent investigations have demonstrated potential mpox transmission from patients to healthcare workers after contact with contaminated bedding[1] or other fomites.[2] In support of such findings, viable mpox virus has been detected on various surfaces in home and hospital settings of infected individuals (Table 1).[3–8] One quantitative study of viable mpox virus in a residential setting found the highest level on underwear.[3] Viable mpox has been detected on household surfaces for up to 15 days, but at low titers suggesting a lesser potential for transmission.[3] Mpox survival in the environment is highly dependent on surrounding temperature and humidity,[9] as well as the porosity of a contaminated object.[3] When mpox mixed with blood or albumin was inoculated on stainless steel at 37°C, no viable mpox could be recovered after 6 and 7 days, respectively, 10 and 11 days, respectively at 22°C, but up to 30 days at 4°C for mpox mixed with either blood or albumin.[9]

Based on the data reviewed above, healthcare workers should follow guidance regarding personal protective equipment upon entering the immediate environment of a patient with known or suspected mpox, regardless of whether or not the healthcare worker intends to have direct contact with the patient.[10] In addition, emphasis should also be placed on careful removal of personal protective equipment to prevent self-contamination while doffing and practicing hand hygiene thereafter. Lastly, cleaning environmental surfaces in the rooms of such patients should be done using products with mpox cidal activity.[9,10]

**Table 1.** Detection of Mpox on Surfaces in Home and Hospital Settings

| Authors | Home | Hospital |
|---|---|---|
| | Viable mpox virus detected on household surfaces | Viable mpox virus detected on hospital room surfaces |
| Morgan et al[3] | Paper towels, underwear, blanket, towel, mattress cover, tabletop | . . . |
| Atkinson et al[4] | Mattress and sheet, towel, iPad, door handle, sink tap, duvet, sofa, hall light switch | . . . |
| Pfeiffer et al[5] | . . . | No viable virus detected |
| Nörz et al[6] | . . . | Soap dispenser handle, towel, glove after touching objects |
| Gould et al[7] | . . . | Anteroom floor after PPE doffing |
| Marimuthu et al[8] | . . . | Chair in patient room, toilet seat, linen dust |

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