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Short Communications

Revisiting SARS-CoV-2 environmental contamination by patients with COVID-19: The Omicron variant does not differ from previous strains



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ABSTRACT

SARS-CoV-2 Omicron strain emergence raised concerns that its enhanced infectivity is partly due to altered spread/contamination modalities. We therefore sampled high-contact surfaces and air in close proximity to patients who were verified as infected with the Omicron strain, using identical protocols applied to sample patients positive to the original or Alpha strains. Cumulatively, for all 3 strains, viral RNA was detected in 90 of 168 surfaces and 6 of 49 air samples (mean cycle threshold [Ct]=35.2±2.5). No infective virus was identified. No significant differences in prevalence were found between strains.

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SARS-CoV-2 was shown to be transmitted mainly by respiratory droplets and direct contact with contagious individuals. Previously, we characterized this transmissibility through contaminated inanimate surfaces, which is indirect contact (Ben-Shmuel et al., 2020). Recently, emergence of the Omicron (B.1.1.529) variant caused a global surge in new cases, rapidly spreading while seemingly causing an altered, less severe disease (Araf et al., 2022; Kannan et al., 2021). The infection rate raised concerns regarding the variant's mode of spread in the populace. Thus, debate has arisen whether Omicron's increased infectivity is due to altered contamination/persistence on surfaces and/or the gain of airborne transmissibility (Riediker et al., 2022; Wong et al., 2022). We therefore executed a contamination sampling campaign in proximity to patients positive for the original, Alpha (B.1.1.7), or Omicron SARS-CoV-2 strains.

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Equal contribution

2022 in a hospital isolation ward (n=12, mild-to-severe condition) and in a quarantine hotel (n=4, mild condition). Original strainor Alpha variant–infected patients (n=15 and n=18, respectively) were sampled during February 2021 in a hospital ward (mild-tosevere condition). Inclusion criteria were newly hospitalized patients (within 24-48 hours of RT-PCR) and high upper respiratory viral loads (by reverse transcriptase-PCR [RT-PCR], 12-36 hours before sampling). Patients were not masked and were in bed during the entire sampling process. As shown (Figure 1), all 3 groups were comparable, with statistically similar mean Ct values of 23.1 (13.1-33.3), 23.3 (12.1-32.5), and 21.1 (17.4-26.8) for patients infected with the original, Alpha, or Omicron strains, respectively. Surface samples were taken from high-contact objects in close proximity to each patient (bedrails, bedside tables, and patients' monitors). Each surface was swabbed with 3 sterile 6" applicators, sampling an area of 20 \times 20 cm, pooled into a 15 ml tube containing 2 ml viral transfer medium (Minimal Essential Media [MEM] supplemented with 2% fetal calf serum [FCS] and 200 Units/ml Penicillin, 0.2% streptomycin, and 25 units/ml Nystatin). Air sampling was performed using an MD8 air sampler (Sartorius, Gottingen, Ger-

Omicron-infected patients (n=16) were sampled during January

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Figure 1. Patient viral load. Upper respiratory SARS-CoV-2 viral load in patients as determined by RT-PCR of nasal and mouth swabs (original strain n= 15, Alpha strain n=18, Omicron strain n=16). Data symbols present individual patient Ct values from 12-36 hours before sampling; black bars present patients' group mean \pm SD. Statistical analysis carried by Prism 6 for Windows (GraphPad Software, San Diego, California, USA). Ct, cycle threshold; RT-PCR, reverse transcriptase–PCR.

many) equipped with 3.0 µm gelatin membranes, at 50 liter/min for 20 minutes (1000 liters per sample), positioned facing the patient, 1-1.5 m away. After sampling, each gelatin membrane was immediately dissolved in a 50 ml tube containing 10 ml viral transfer medium. Cold-chain transport of samples was maintained (4-8°C), with sample processing performed within 2-3 hours of sampling. RNA extraction, RT-PCR assays, and virus infectivity cytopathic effect (CPE) assays were performed as previously described (Ben-Shmuel et al., 2020).

Viral RNA was detectable on 60% of high-contact surfaces near Omicron-infected patients (table 1). Contamination, inferred by Ct values, was relatively low (Ct=35.2±2.5). For the original and Alpha strains, each group included ventilated patients (original 6/15, Alpha 6/18); similar levels of contamination were detected, at a slightly lower prevalence (46%), not a statistically significant difference (p=0.29). Patient monitors were sampled to assess medical staff contamination via gloves. Viral RNA was detected on 68%, 52%, and 41% of the patients' monitors for patients infected with the original, Alpha, and Omicron strains, respectively (statistically insignificant, p=0.26). Contamination levels (by Ct) were similar for all strains. None of the surfaces sampled contained viable infectious virus (0/168, table 1). Taken together, high-contact surface and monitor contamination rates were virtually identical for all strains (55.6%, 49.2%, and 56.7% for the original, Alpha, and Omicron strains, respectively). Finally, we detected traces of viral RNA in 3 of 16 air-sample filters next to Omicron-infected patients, 2 of 18 filters next to Alpha-infected patients, and 1 of 15 filters next to original strain-infected patients. These differences were also statistically insignificant (p=0.59). All air samples were found to be free from infective virus (0/49, table 1).

This study aimed to test whether the Omicron variant is better at contaminating surfaces, surviving on real-world surfaces, or being an airborne infective pathogen. Although surface contamination was substantial for all strains (90/168 RT-PCR positive samples, 53%), viral viability in the clinical setting was negligible, with no samples containing cultivable virus. The rates and Ct values observed for contaminated samples were similar between variants. Compared with this relatively high rate, air sample contamination was both significantly less frequent and much lower: 12% contamination, mean Ct value of 35.6 (32.1-37.1). Differences between strains were statistically insignificant. Viable SARS-CoV-2 was not isolated from any air sample. Our results show that in terms of contamination, viability, and stability on surfaces and in

F able 1 SARS-CoV-2 contamina	ation on surfaces and air samples	in proximity to patients with COVID-	19		
SARS-CoV-2 strain	Sample type	description	Positive/total samples(%)	Mean Ct value(range)	Infective virus/total samples(%)
Original strain	Surface samples (400 cm²)	Bed rails, bedside table Patient's monitor	12/26 (46%) 13/19 (68%)	33.3 (31.7-35.5) 33.4 (30.1-36.2)	0/26 (0%) 0/19 (0%)
	Air Sampling (1 m ³)	1-1.5 m in front of the patient	1/15 (6%)	35.1 (35.1)	0/15 (0%)
Alpha	Surface samples	Bed rails, bedside table	20/42 (47%)	31.6(24.4-36.3)	0/42 (0%)
(B.1.1.7)	(400 cm^2)	Patient's monitor	11/21(52%)	33.2 (26.4-37.1)	0/21 (0%)
	Air Sampling	1-1.5 m in front of the patient	2/18 (11%)	34.2 (32.1-36.3)	0/18 (0%)
Omicron	surface samples	Bed rails, bedside table	29/48 (60%)	35.2 (29.5-39.5)	0/48 (0%)
(B.1.1.529)	(400 cm^2)	Patient's monitor	5/12 (41%)	36.7 (34.5-38.6)	0/12 (0%)
	Air Sampling	1-1.5 m in front of the patient	3/16 (19%)	36.7 (36.3-37.1)	0/16 (0%)
	(1 m ³)				
Total	surface samples (400 cm ²)		90/168 (53%)		0/168 (0%)
	Air samples		6/49 (12%)		0/49 (0%)
Abbreviation: Ct, cycle	threshold.				

air samples, the Omicron variant is remarkably similar to previous COVID-19 strains (original and Alpha strains). This correlates with previous studies conducted by us (Ben-Shmuel et al., 2020) and others (Lane et al., 2021; Ong et al., 2021; Thakar et al., 2021; Winslow et al., 2021) in healthcare settings, isolation units, quarantine hotels, and schools. Moreover, it was recently shown that SARS-CoV-2 significantly loses viability, with near complete viability loss occurring within 2-5 minutes, in controlled laboratory aerosolized dispersion at normal relative humidity (Oswin et al., 2022).

This preliminary work suggests that Omicron's increased transmissibility does not result from acquiring airborne infectivity, higher environmental contamination, or better resilience on surfaces. Alternatively, plausible explanations may include the mutated spike protein's higher affinity toward the angiotensinconverting enzyme 2 (ACE2) receptor and lower immune recognition. In addition, enhanced viral loads in clinically milder patients facilitate spread by having more infective, less symptomatic individuals in the community for longer durations before diagnosis and isolation.

Conflict of interest

None declared.

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Ethical approval

The study was approved by the ethics committee of Rambam hospital and and the Institutional Biosafety Committee at Israel Institute for Biological Research.

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