Title: Investigating SARS-CoV-2 surface and air contamination in an acute healthcare setting during the peak of the COVID-19 pandemic in London

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Summary: The role of surface and air contamination in SARS-CoV-2 transmission was evaluated in a London hospital. Whilst SARS-CoV-2-RNA was detected, no viable virus was recovered. This underlines the potential risk of environmental contamination and the need for effective IPC practices.

ABSTRACT

Background: Evaluation of SARS-CoV-2 surface and air contamination during the COVID-19 pandemic in London.

Methods: We performed this prospective cross-sectional observational study in a multi-site London hospital. Air and surface samples were collected from seven clinical areas, occupied by patients with COVID-19, and a public area of the hospital. Three or four 1.0 m^3 air samples were collected in each area using an active air sampler. Surface samples were collected by swabbing items in the immediate vicinity of each air sample. SARS-CoV-2 was detected by RT-qPCR and viral culture; the limit of detection for culturing SARS-CoV-2 from surfaces was determined.

Results: Viral RNA was detected on 114/218 (52.3%) of surfaces and 14/31 (38.7%) air samples but no virus was cultured. The proportion of surface samples contaminated with viral RNA varied by item sampled and by clinical area. Viral RNA was detected on surfaces and in air in public areas of the hospital but was more likely to be found in areas immediately occupied by COVID-19 patients than in other areas (67/105 (63.8%) vs. 29/64 (45.3%) (odds ratio 0.5, 95% confidence interval 0.2-0.9, p=0.025, Chi squared test)). The high PCR Ct value for all samples (>30) indicated that the virus would not be culturable.

Conclusions: Our findings of extensive viral RNA contamination of surfaces and air across a range of acute healthcare settings in the absence of cultured virus underlines the potential risk from environmental contamination in managing COVID-19, and the need for effective use of PPE, physical distancing, and hand/surface hygiene.

Key words: SARS-CoV-2, COVID-19, transmission, air contamination, surface contamination

INTRODUCTION

The severe acute respiratory syndrome coronavirus (SARS-CoV-2) virus has rapidly spread around the world since it emerged in late 2019, resulting in a coronavirus disease 2019 (COVID-19) pandemic.[1] Evidence from SARS, influenza, and SARS-CoV-2 suggests droplet and contact spread as primary transmission routes with evidence of airborne spread during aerosol generating procedures (AGPs).[1, 2]

Hospital-onset COVID-19 infection (HOCI) has been reported, and is probably linked to ineffective implementation of infection prevention and control measures.[1, 3-5] The transmission dynamics in healthcare environments are unclear and likely to be multifactorial. Contaminated surfaces and air were a key part of the transmission dynamics of SARS, MERS, influenza, and other organisms in hospitals.[1, 2, 6] Laboratory evidence suggests that the SARS-CoV-2 virus can survive on dry surfaces and in aerosols for days to weeks, particularly on non-porous surfaces.[7, 8] Furthermore, SARS-CoV-2 RNA has been detected on surfaces and in the air in hospitals that are caring for patients with COVID-19.[9-17]

However, our understanding of the role of surface and air contamination in the transmission of SARS-CoV-2 is limited. Most studies to date have relied on PCR, and have not attempted to culture virus thereby limiting the ability to interpret PCR-based detection; have focussed upon one geographical region (Asia); and have included a limited selection of clinical and non-clinical areas with no evidence from operating theatre environments.[9, 10, 12, 13, 15, 16] In mid-April 2020, the UK experienced the first wave of the COVID-19 pandemic. During this period, there was evidence for HOCI.[5] Therefore, to inform and optimise infection prevention and control interventions, we evaluated SARS-CoV-2 surface and air contamination across a range of clinically-relevant locations (including operating theatres) and public areas during the peak of the COVID-19 pandemic in

London, using both RT-PCR and viral culture. We also performed supporting laboratory experiments to assess SARS-CoV-2 viability on surfaces, with associated limits of detection, to qualify our findings.

METHODS

Setting

Sample collection for this prospective cross-sectional study was performed between April 2nd and 20th 2020 on selected wards at a large North West London teaching hospital group comprising five hospitals across four sites with 1,200 acute beds. Most sampling was conducted on one hospital site during the peak of the COVID-19 pandemic (Supplemental Figure 1) when most patients were managed in cohort wards.

Clinical areas selected for air and surface sampling

Seven clinical areas (emergency department, an admissions ward, two COVID-19 cohort wards, theatres during tracheostomy procedures, an admissions ward, an intensive care unit, and a 6-bedded bay converted into a negative pressure area for management of continuous positive airway pressure (CPAP) on patients with COVID-19) and a public area of the hospital were selected to represent the diversity of clinical environments (Supplemental Table 1).

All inpatient wards were fully occupied by adult patients with COVID-19 at the time of sampling, apart from the Emergency Department. In the part of the Emergency Department dedicated for patients with confirmed or suspected COVID-19, two of the cubicles were occupied and one patient was in the ambulatory wait area at the time of sampling.

All areas were disinfected daily with an additional twice daily disinfection of high touch surfaces using a combined chlorine-based detergent/disinfectant (Actichlor Plus, Ecolab).

In each clinical area, between three and five air samples were collected. High touch surfaces in the immediate vicinity of each air sample were sampled, including bed rails, clinical monitoring devices (blood pressure monitors), ward telephones, computer keyboards, clinical equipment (syringe pumps, urinary catheters), hand-cleaning facilities (hand washing basins, alcohol gel dispensers). In each clinical area, sampling was performed in both patient (i.e. bays and single rooms) and non-patient care areas (i.e. nursing stations and staff rooms). Environmental sampling was conducted during three tracheostomy procedures. During the first procedure, air sampling was performed before and during the procedure; for the other procedures, air sampling was performed during the procedure only.

Sampling methods

1 m^3 air samples were collected into a conical vial containing 5 mL Dulbeccos's minimal essential medium (DMEM) using a Coriolis µ air sampler (Bertin Technologies). Surface samples were collected by swabbing approximately 25 cm^2 areas of each item using flocked swabs (Copan, US) moistened in DMEM and then deposited into 1.0 mL of DMEM. Temperature, humidity and time of day were recorded at the time of sampling.

Detection and quantification of SARS-CoV-2 viral RNA genome and viral culture

Viral RNA detection and absolute quantification was performed using quantitative real-time reverse transcription polymerase chain reaction (RT-qPCR). Samples were extracted from 140 µL of the DMEM medium using the QIAamp viral RNA mini kit (Qiagen, Germany). Negative controls (water) were extracted and included in the PCR assays. SARS-CoV-2 viral RNA was detected using AgPath-ID One-Step RT-PCR Reagents (Life Technologies) with specific primers and probes targeting the envelope (E) gene.[18] A standard curve with six serial dilutions of $1x10^5$ – 1×10^0 copies/µL E gene was included in each RT-qPCR run. E gene copies per m^3 air and copies per swab were calculated.

Duplicate PCRs were run from each sample. Samples were defined as positive if both duplicates had Ct< 40.4, and defined as suspected if one of the two have Ct<40.4, equivalent to one genome copy.

Viral culture: Vero E6 (African Green monkey kidney) and Caco2 (human colon carcinoma) cells were used to culture virus from air and environmental samples using a method adapted from one previously used to culture influenza virus.[19] The cells were maintained in DMEM supplemented with heat inactivated fetal bovine serum (10%) and Penicillin/Streptomycin (10, 000 IU/mL &10, 000 µg/mL). For virus isolation, 200 µL of samples were added to 24 well plates. On day 0 and after 5-7 days, cell supernatants were collected, and RT-qPCR to detect SARS-CoV-2 performed as described above. Samples with at least one log increase in copy numbers for the E gene (reduced Ct values relative to the original samples) after 5-7 days propagation in cells compared with the starting value were considered positive by viral culture.

We performed a laboratory experiment to determine the limit of detection for culturing SARS-CoV-2 dried on surfaces. A log 10 dilution series from solution containing $8.25x10^6$ PFU/mL SARS-CoV-2 (titred by plaque assay in Vero cells) from 10^{-3} to 10^{-6} (covering Ct values from 26 to 36 and E gene copies from 10^6 to 10^3) was produced in DMEM and 50 µL of each dilution was inoculated in triplicate onto the surface of plastic (standard keyboard key) or stainless steel (2 x 1 x 0.2 cm) pieces. The inoculated surfaces were dried in a safety cabinet for 2 hours after which they were visibly dry. They were then sampled using flocked swabs. Swabs were deposited into 1.5 mL of DMEM for 1 hour, and then 100 µL used to inoculate wells of VeroE6 cells culture in 24 well plates. RT-qPCR was used to determine viability following 7 days of culture as follows. 140 µL was used for RNA extraction and qPCR immediately (0 days post inoculation, dpi) and after incubation for 7 days in a 24-well plate with VeroE6 cells (7 dpi). Samples with an increase in copy numbers for the E gene

(reduced Ct values relative to the original samples) after propagation in Vero E6 cells were considered positive by viral culture.

Statistical analysis

A Chi square test was used to compare the proportion of environmental samples (surfaces or air) that were positive or suspected for SARS-CoV-2 RNA in areas immediately occupied by patients with COVID-19 with other areas. The mean concentration of air and surface contamination in each of the areas was log transformed and then compared by one-way ANOVA followed by Tukey's multiple comparisons test.

Ethics approval

The work was registered locally as an NHS service evaluation (#434).

RESULTS

114/218 (52.3%) of surface samples were suspected (91/218 (41.7%)) or positive (23/218 (10.6%)) for SARS-CoV-2 RNA but no virus was cultured (Table 1). The proportion of surface samples with suspected or positive RNA varied by item, including >80% of computer keyboards/mice, alcohol gel dispensers, and chairs, and >50% of toilet seats, sink taps, and patient bedrails (Figure 1).

14/31 (38.7%) of air samples were suspected (12/31 (38.7%)) or positive 92/31 (6.4%)) for SARS-CoV-2 RNA but no virus was cultured (Table 1). 10^1 to 10^3 genome copies / m^3 of SARS-CoV-2 RNA was detected in air from all eight areas tested (Table 1); there was no significant difference in mean viral RNA concentration across the eight areas tested (p=0.826). Similarly, 10^1 to 10^4 genome copies per swab SARS-CoV-2 RNA was detected in surface samples from all eight areas tested (Figure 2). There was a significant difference in the mean SARS-CoV-2 surface viral load across the eight areas tested

(p=0.004), with both Cohort Ward A (mean = 1.76 $\log_{10}$ copies/swab, p=0.015) and the Temporary CPAP ward (mean = 1.69 $\log_{10}$ copies/swab, p=0.016) showing higher levels of viral RNA than the adult ICU (mean = 0.0018 $\log_{10}$ copies/swab).

SARS-CoV-2 RNA was detected in several clinical areas where AGPs are commonly performed, including a resuscitation bay in the emergency department and a bay temporarily converted for CPAP, where SARS-CoV-2 RNA was detected from air both within and outside the bay. No patient was undergoing CPAP at the time of sampling, but one patient was undergoing high-flow nasal cannula (HFNC) oxygen therapy. In operating theatres, 1/3 air samples collected during three tracheostomy procedures were positive.

SARS-CoV-2 RNA was detected in surface and air samples in parts of the hospital hosting staff but not being used for direct patient care, including the ICU staff room, the nursing station outside of the CPAP unit, and the hospital main entrance and public toilets. However, SARS-CoV RNA detection in air and surface samples was significantly more likely in areas immediately occupied by COVID-19 patients than in other areas; (67/105 (63.8%) in areas immediately occupied by COVID-19 patients vs. 29/64 (45.3%) in other areas (odds ratio 0.5, 95% confidence interval 0.2-0.9, p=0.025).

Since viable virus was not cultured from any of the air or surface samples, we performed laboratory experiments to determine the limit of detection of SARS-CoV-2 dried onto surfaces. Four different dilutions of virus deposited onto two non-porous surfaces determined that dried inocula with a Ct value <30 (corresponding to an E gene copy number of $\geq 10^5$ per mL) yielded SARS-CoV-2 that could be cultured (Table 2). In our study, all surface and air samples from the hospital environment had a Ct value >30.

DISCUSSION

SARS-CoV-2 RNA was detected frequently from surface and air samples but was not cultured. SARS-CoV-2 RNA was identified across the eight areas that we tested, and was detected more frequently in areas occupied by COVID-19 patients than in other areas.

A direct comparison between our findings and other studies evaluating SARS-CoV-2 surface and air contamination is not possible due to differences in: environmental sampling strategy; experimental methods (including sampling methods); local SARS-CoV-2 epidemiology; the physical layout of buildings and clinical spaces; patient characteristics and shedding;[4, 20] and patient and staff testing approaches and cleaning/disinfection protocols. Nonetheless, our finding of widespread detection of viral RNA on surfaces (114/218, 52.3%) and to a lesser extent air (14/31, 38.7%) is broadly consistent with the findings of most others although the proportion of surface and air samples positive for viral RNA is higher in our study.[9-14] For example, Ye et al. found that 14% of 626 surface samples were positive for viral RNA, with a higher proportion of surface samples positive in the ICU (32% of 69 samples), when sampling a range of clinical settings in a hospital caring for patients with COVID-19 in Wuhan, China.[10] However, other studies have identified very little or no surface or air contamination.[9, 11] Other studies have observed higher frequencies of contamination in patient-care vs. non-patient-care areas,[9, 10, 12] and variation in the frequency of contamination across different clinical areas, which is in line with our findings.[10, 12] One surprising finding in our study was that the level of contamination on ICU surfaces was lower than in a cohort general ward or in the temporary CPAP ward, in contrast to other findings.[10] This may be because patients sampled in the ICU were on closed circuit ventilation systems through cuffed endotracheal tubes, which may have a lower risk of producing surface and air contamination than other ventilation systems such as CPAP.

We did not identify viable virus on any surface or air sample. Few studies have attempted to culture SAR-CoV-2 from healthcare environments, and no viable virus was detected.[11, 15] Our laboratory study of the viability of virus dried on surfaces helps to qualify our findings and the findings of others, suggesting that Ct values of >30 corresponding to an E gene copy number of $<10^5$ per mL are unlikely to be culturable (Table 2). This finding parallels studies of viral infectivity from clinical specimens.[21, 22] Bearing in mind that the viral RNA detected in the hospital setting might have been deposited more than two hours previously, we cannot differentiate whether our inability to culture virus from the samples is explained by the low RNA levels or the length of time since deposition which may reflect non-viable viral RNA. It is also possible that virus was infectious but not culturable in the laboratory.

Surface contamination was detected on a range of items, especially computer keyboards, chairs, and alcohol dispensers. Other studies have also identified computer keyboards and/or mice as a risk for SARS-CoV-2 RNA contamination.[9, 10, 12] Many of the computers that we sampled were in shared staff clinical areas (such as nursing stations), so this argues for frequent disinfection of these items. The contamination of alcohol gel dispensers is unsurprising since staff hands activate these before hand hygiene is performed. However, alcohol gel dispensers should be included in routine cleaning and disinfection protocols or designed such that they can be activated without touching.

We sampled several areas where AGPs are commonly performed There was no difference in the viral load of the air across the eight areas sampled, suggesting that AGPs do not produce persistently high levels of air contamination. However, we did not sample the air over time, and our air sampling method did not differentiate particle size so we are unable to distinguish droplets from aerosols (< 5 µM). One recent study identified SARS-CoV-2 RNA at low levels (in the 10^1-10^2 range copies per m^3) in patient care areas in a permanent and field hospital in Wuhan, China.[14] Positive samples were identified in a range of particle sizes, including those <5 µM, which would typically be considered as

aerosols.[2] It seems likely, therefore, that the positive and suspected air samples identified in our study included a range of particle sizes spanning 5 µM, particularly in areas where aerosol generating procedures are common.

Whilst we sampled in a temporary CPAP ward, no patient was undergoing CPAP at the time of sampling. However, one patient was undergoing HFNC during sampling, and air contamination was identified <1 m from this patient. A recent summary of evidence concludes that HFNC is a lower risk procedure in terms of aerosol generation than CPAP, which should be a topic for future studies.[23]

We identified surface and air contamination during three tracheostomy procedures. Several studies and commentaries have evaluated the potential for surgical procedures to produce aerosols for patients with COVID-19.[24-26] One study evaluated the spread of droplets during tracheostomies, although did not include sampling for SARS-CoV-2.[24] Our findings highlight a potential theoretical risk of COVID-19 transmission during these procedures. However, a larger sample size is required to understand this risk.

Strengths of our study includes our sampling strategy encompassing contemporaneous surface and air samples from a range of patient and non-patient care areas with diversity in physical layout and ventilation and including operating theatres and areas dedicated to known and potential AGPs; each sample was tested using PCR and viral culture, and we performed laboratory experiments to quality our findings; the sampling was conducted during the peak of the pandemic (and so likely represents a worst-case scenario). Limitations include not collecting patient samples to better understand how our findings links to patient samples, particularly during tracheostomies and AGPs; no asymptomatic patient or staff testing at the time of sampling, which means asymptomatic patients and staff could have shed SARS-CoV-2; challenges in interpreting the significance of samples with low viral loads; a

lack of resolution of particle sizes for contamination of the air; and as no longitudinal sampling was performed these findings represent a “snapshot”.

Our findings may have implications for future policy and guidelines. Most international guidelines recommend enhanced surfaces disinfection during the management of COVID-19. For example, Public Health England recommends enhanced disinfection using a chlorine-based disinfectant (or a disinfectant with effectiveness against coronaviruses).[27] Our finding of widespread RNA contamination of clinical areas used to care for patients with COVID-19 supports this. Physical distancing is recommended by most governments and personal protective equipment (PPE) is recommended during contact with patients with COVID-19 plus higher levels of PPE for performing aerosol generating procedures. Whilst we did not measure particle sizes during our air sampling, our findings highlight a potential role for contaminated air in the spread of COVID-19. Our finding of air contamination outside of clinical areas should be considered when making respiratory PPE recommendations in healthcare settings.[28]

Whilst SAR-CoV-2 RNA was detected within healthcare environments, further research linking patient, staff and environmental samples is required to better understand transmission routes. Longitudinal environmental and clinical sampling across healthcare settings is required to understand risk factors associated with viral shedding and transmission. Our findings can be used to parameterise mathematical models of COVID-19 transmission. Our methods can be used to assess the risks associated with various procedures including surgery, AGPs, and nebulisation of medications. Findings from these studies may influence PPE recommendations for specific procedures.[29-31]

Our findings of extensive viral RNA contamination of surfaces and air across a range of acute healthcare settings in the absence of cultured virus underlines the potential risk from surface and air

contamination in managing COVID-19, and the need for effective use of PPE, physical distancing, and hand/surface hygiene.

Author contributions

All authors met the ICMJE criteria for authorship. JZ and JAO conceived the study, collected and analysed data, and wrote the manuscript; JRP conceived the study, collected data, and contributed to the manuscript; CP, DMG, PRB, SM collected data and contributed to the manuscript; FB, AHH, and WSB conceived the study, analysed data, and contributed to the manuscript. JAO is the study guarantor.

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DISCLAIMER

The views expressed in this publication are those of the author(s) and not necessarily those of the NHS, the National Institute for Health Research, the Department of Health and Social Care or Public Health England. Professor Alison Holmes is a National Institute for Health Research (NIHR) Senior Investigator. International Severe Acute Respiratory and Emerging Infection Consortium (ISARIC) provided funding for JZ and laboratory materials used for this study.

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CONFLICT OF INTEREST STATEMENT

All authors have completed the ICMJE uniform disclosure form at www.icmje.org/coi_disclosure.pdf and declare: JAO reports personal fees from Gama Healthcare Ltd and Pfizer in the past three years

outside the submitted work; JK reports grants from H2020 (ITN grant), NIHR (i4i grant), CRUK fellowship, J+J Educational grant, personal fees from Verb robotics / Ethicon and Medtronic, and other relationships with Cerulean Health, One Welbeck day surgery, and LNC therapeutics; all other authors report no financial relationships that could appear to have influenced the submitted work.

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FIGURE LEGENDS

Figure 1. Proportion of environmental samples suspected or positive by item sampled. The number of the x axis represented the number of each item sampled.

Figure 2. SARS-CoV-19 E gene copy number from surface swabs. The quantity of E gene copy number per swab is shown. Suspect samples = blue dots; positive samples = red dots; negative samples = black dots.

Figure 1

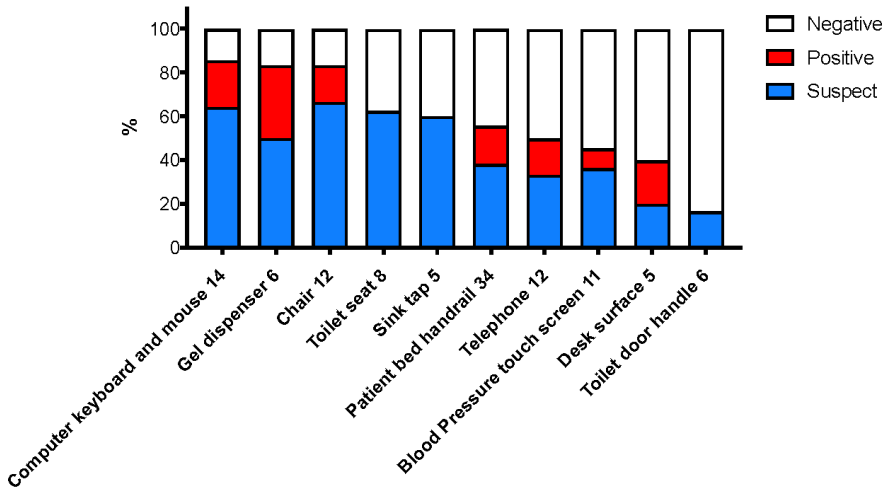

Figure 2

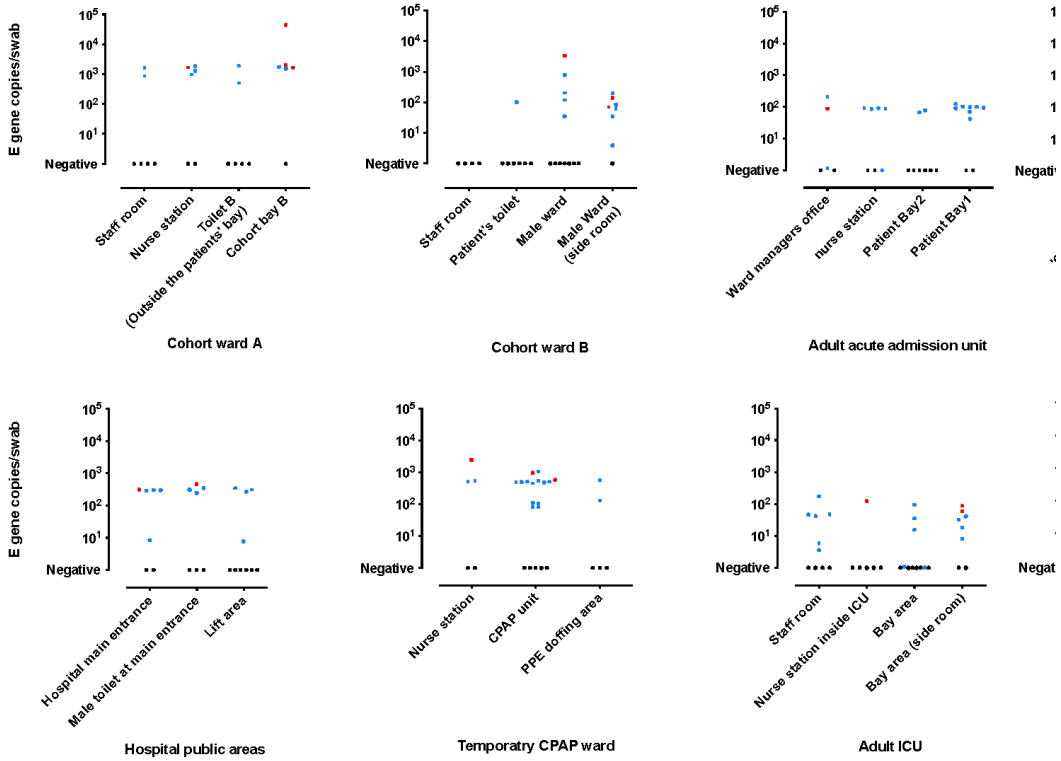

Table 1. PCR results from surface and air samples.

		SURFACE SAMPLES							AIR SAMPLES		
		Total	positive	%positive	suspect	%suspect	positive or suspect	% positive or suspect	Result	Concentration (copies/m^3)	Notes
Cohort ward A	Staff room	6	0	0.0	2	33.3	2	33.3	Negative		
	Nurse station	6	1	16.7	3	50.0	4	66.7	Negative		
	Toilet B (outside the patients' bay)	6	0	0.0	2	33.3	2	33.3	Negative		
	Cohort bay B	6	3	50.0	2	33.3	5	83.3	Positive	7048	
Cohort ward B	Staff room	4	0	0.0	0	0.0	0	0.0	Negative		
	Patients' toilet (in the ward)	7	0	0.0	1	14.3	1	14.3	Suspect	464	
	Male bay	12	1	8.3	4	33.3	5	41.7	Suspect	1335	
	Male bay (side room)	8	2	25.0	5	62.5	7	87.5	Suspect	163	
Adult acute admission unit	Ward managers office	5	1	20.0	2	40.0	3	60.0	Negative		
	Nurse station	7	0	0.0	5	71.4	5	71.4	Positive	404	
	Patient bay 2	8	0	0.0	2	25.0	2	25.0	Negative		
	Patient bay 1	10	0	0.0	8	80.0	8	80.0	Negative		
Adult emergency department	'Green' majors	10	1	10.0	5	50.0	6	60.0	Negative		
	Nurse station	4	2	50.0	0	0.0	2	50.0	Negative		
	Ambulatory waiting	3	2	66.7	1	33.3	3	100.0	Negative		
	Patient assessment cubicles	3	0	0.0	1	33.3	1	33.3			
	Male toilet (next to the nurse station)	2	0	0.0	1	50.0	1	50.0			
	Resus bay (last patient > 2 hours)	10	0	0.0	4	40.0	4	40.0	Suspect	35	
Hospital public areas	QEQM main entrance	7	1	14.3	4	57.1	5	71.4	Suspect	1574	
	Male toilet at QEQM main entrance	7	1	14.3	3	42.9	4	57.1	Suspect	1545	
	Lift area QEQM ground floor	10	0	0.0	4	40.0	4	40.0	Negative		
Temporary CPAP ward	Nurse station	5	1	20.0	2	40.0	3	60.0	Suspect	1922	
	CPAP unit	19	2	10.5	12	63.2	14	73.7	Suspect	31	< 1m from 2 patients
									Negative		> 2 m from patients
	PPE doffing area	5	0	0.0	2	40.0	2	40.0	Negative		
Adult ICU	Staff room	10	0	0.0	6	60.0	6	60.0	Suspect	249	
	Nurse station inside ICU	6	1	16.7	0	0.0	1	16.7	Negative		
	Bay area	11	0	0.0	5	45.5	5	45.5	Suspect	164	
	Side room bay area	8	2	25.0	4	50.0	6	75.0	Suspect	307	
Theatres	Theatres	13	2	15.4	1	7.7	3	23.1	Negative		Before tracheostomy
									Negative		During tracheostomy
									Suspect	1163	During tracheostomy
									Negative		During tracheostomy
	Total	**218**	**23**	**10.6**	**91**	**41.7**	**114**	**52.3**	**2/31 (6.4%) positive; 12/31 (38.7%) suspect**		

Table 2: Viability of SARS-CoV-2 dried onto steel or plastic surfaces from a dilution series; viability determined through RT-PCR from cultures immediately after drying, 0 days post inoculation (dpi) with Vero E6 cells compared with after culture (7 dpi). Means and standard deviations of Ct values are shown.

	Steel surface				Plastic surface			
Inoculum (PFU)	**Swab (Ct)**	**E gene copies/mL**	**After culture (Ct)**	**Interpretation**	**Swab (Ct)**	**E gene copies/mL**	**After culture (Ct)**	**Interpretation**
41.25	26.23 ± 0.30	$1.86x10^6 \pm 3.66x10^5$	12.65 ± 0.51 Pos	Culturable	25.95 ± 0.06	$2.23x10^6 \pm 9.74x10^4$	11.16 ± 0.19 Pos	Culturable
4.125	29.27 ± 0.04	$2.30x10^5 \pm 5.10x10^3$	12.86 ± 0.01 Pos	Culturable	29.51 ± 0.29	$1.97x10^5 \pm 3.71x10^4$	12.58 ± 1.47 Pos	Culturable
0.4125	32.54 ± 0.06	$2.47 \times 10^4 \pm 9.23 \times 10^2$	36.48 ± 1.80 Neg	Non-culturable	32.67 ± 0.07	$2.23 \times 10^4 \pm 1.04 \times 10^3$	37.39 ± 0.21 Neg	Non-culturable
0.04125	39.22 ± 5.13	$1.68 \times 10^3 \pm 1.92 \times 10^3$	41.33 ± 3.45 Neg	Non-culturable	36.55 ± 0.23	$1.63 \times 10^4 \pm 2.85 \times 10^2$	39.76 ± 4.61 Neg	Non-culturable