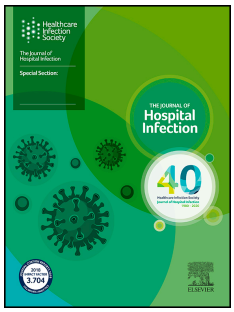

SARS-CoV-2 surface and air contamination in an acute healthcare setting during the first and second pandemic waves

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Running Title: SARS-CoV-2 air and surface contamination

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Summary

Background: Surfaces and air in healthcare facilities can be contaminated with SARS-CoV-2. In a previous study, we identified SARS-CoV-2 RNA on surfaces and air in our hospital during the 'first wave' of the COVID-19 pandemic (April 2020).

Aim: To explore whether the profile of SARS-CoV-2 surface and air contamination had changed between April 2020 and January 2021.

Methods: A prospective, cross-sectional, observational study in a multisite London hospital. In January 2021, surface and air samples were collected from comparable areas to those sampled in April 2020 comprising six clinical areas and a public area. SARS-CoV-2 was detected using RT-PCR and viral culture. Sampling was additionally undertaken in two wards with only natural ventilation. The ability of the prevalent variants at the time of the study to survive on dry surfaces was evaluated.

Findings: No viable virus was recovered from surfaces or air. 5% (14) of 270 surfaces and 4% (1) of 27 air samples were positive for SARS-CoV-2, which was significantly lower than in April 2020 (52% (114) of 218 of surfaces and 48% (13) of 27 air samples ($p<0.001$, Fisher's Exact Test)). There was no clear difference in the proportion of surfaces and air samples positive for SARS-CoV-2 RNA based on the type of ventilation in the ward. All variants tested survived on dry surfaces for at least 72 hours with a $<3\text{-log}_{10}$ reduction in viable count.

Conclusion: Our study suggests that enhanced infection prevention measures have reduced the burden of SARS-CoV-2 RNA on surfaces and air in healthcare.

Key words: SARS-CoV-2, COVID-19, variants of concern, transmission, air contamination, surface contamination, infection prevention and control

Introduction:

The COVID-19 pandemic continues with epidemic waves affecting various parts of the world [1]. Several epidemic waves have occurred in the UK resulting in a peak of hospitalisations in April 2020 and a second, larger peak of hospitalisations in January/February 2021 [2, 3]. The second wave of hospitalisations in early 2021 was associated with increased community prevalence of COVID-19 infection and a second wave of COVID-19 in healthcare workers [4].

Respiratory viruses like influenza, SARS-CoV-1, SARS-CoV-2 and others are able to transmit via the air and via contact under some circumstances [5, 6]. There is considerable controversy around the relative importance of different transmission routes involving air as a vector, with some arguing that transmission over short and long range via small aerosolised particles is the predominant transmission route [7, 8]. The virus has been shown to survive on surfaces and in air for days to weeks [9, 10]. SARS-CoV-2 RNA has been identified in hospital air, and viable SARS-CoV-2 has been cultured from a small number of samples in these studies [11-13]. SARS-CoV-2 RNA has also been identified on surfaces in hospitals, although viable SARS-CoV-2 that can be cultured has not been identified [12, 14]. The role of contaminated surfaces and air in the spread of SARS-CoV-2 within healthcare environments is unclear [14].

An important feature of the epidemiology of SARS-CoV-2 is the emergence and international spread of several different variants, which vary in their transmissibility, virulence, and vaccine response [15]. During the second wave of hospitalisations, the Alpha variant (B.1.1.7) emerged as the predominant cause of COVID-19 in the UK [15]. This variant has been found to be more transmissible than other SARS-CoV-2 variants circulating at the time [15, 16]. The reasons for increased transmissibility of the Alpha and other variants are unclear, but do not appear to be as a result of fundamental differences in transmission routes [15]. The Alpha variant also has a characteristic “S gene” knockout mutation, which has proven to be a useful way to rapidly identify it presumptively from other types of SARS-CoV-2 [15].

During the 'first wave' of COVID-19, environmental sampling of air and surfaces at our London hospital group was undertaken in seven clinical areas and a public entrance [12]. This work identified extensive SARS-CoV-2 RNA contamination of surfaces and air in patient-care and non-patient-care areas, but that viable virus could not be cultured from any samples. In order to re-evaluate surface and air contamination in our hospitals during the second wave, and in the context of the emergence of SARS-CoV-2 variants, we used the same sampling methods to test for SARS-CoV-2 surface and air contamination in comparable areas to those sampled during the 'first wave'. We also aimed to understand patterns of surface and air contamination with SARS-CoV-2 variants so we inferred the genotype of SARS-CoV-2 in patients on the day of sampling, and in SARS-CoV-2 detected from surface and air samples. Renal dialysis represents a particular and complex risk and challenge at the interface of community and healthcare in the context of COVID-19 [17]. Therefore, we performed additional sampling in a renal dialysis setting. Given the role of ventilation in preventing the spread of COVID-19 [18], air and surfaces were sampled for SARS-CoV-2 in wards with a range of ventilation approaches, including some with only natural ventilation. Finally, given limited data on the capacity for SARS-CoV-2 variants to survive on surfaces, we performed a laboratory experiment to evaluate the ability of the Alpha variants to survive on dry surfaces compared with other variants.

Methods:

Selecting clinical areas to sample

To provide a comparison of surface and air contamination in the second wave compared with the first wave, surface and air samples were collected from seven comparable areas to those sampled in the first wave [12], which represent a range of clinical services provided by the hospital group. These comprised:

- Adult emergency department, which included sections dedicated for suspected and confirmed COVID-19 patients and for patients not suspected to have COVID-19.
- A COVID-19 cohorting adult acute admissions unit.
- A COVID-19 cohorting adult intensive care unit.

- Two adult COVID-19 cohort wards: one with physically separated 4-bedded bay areas, and one with large open bay areas.
- An adult ward area used for the management of non-invasive ventilation / continuous positive airway pressure, procedures that (at the time) are thought to be a high risk of generating infectious SARS-CoV-2 aerosol.
- The entrance and public area of the main hospital building.

Each of these clinical areas had either mechanical ventilation, recirculated air, or natural ventilation and mechanical ventilation (Table 1). In addition, two wards cohorting patients with COVID-19 with only natural ventilation were sampled to explore the possible role of different ventilation systems in determining surface and air contamination. Sampling was also undertaken in a renal dialysis unit at one of our hospitals.

Sample collection

Surface samples were taken from high touch areas, including bed rails, ward telephones, computers, clinical equipment (syringe pumps, blood pressure monitors), and hand hygiene facilities (hand washing basins, alcohol gel dispensers); air samples were collected in parallel. Samples were collected from the lowest to highest perceived risk of SARS-CoV-2 contamination. Samples were collected between 6-18th January 2021.

Sampling methods

Air sampling was performed using a 'Coriolis Micro' air sampler (referred to as Coriolis hereafter) (Bertin Technologies), which collects air at 300 litres per minute (LPM). After 10 min sampling at 300 LPM, a total of 3.0 m^3 air was sampled into a conical vial containing 5 mL Dulbeccos's minimal essential medium (DMEM). Surface samples were collected by swabbing approximately 25 cm^2 areas of each item using flocked swabs (Copan, US) moistened in DMEM. Swabs were deposited into 1 mL of DMEM.

Detection and quantification of SARS-CoV-2

Viral RNA detection and absolute quantification was performed using quantitative real-time reverse transcription polymerase chain reaction (RT-qPCR). Samples were extracted from 200 µL of the DMEM medium using the QIAsymphony SP (Qiagen, Germany) instrument according to the manufacturer's instructions. 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) [19] and ORF1a genes [20]. A standard curve with six serial dilutions of $1x10^5$ – 1×10^0 copies/µL E gene was included in each run of the RT-qPCR. The number of SARS-CoV-2 virus E gene copies per m^3 air and copies per swab were calculated. Samples were considered positive for SARS-CoV-2 RNA if E or ORF1a RT-qPCR assays gave Ct value less than 45. Human biological material in air samples was quantified by RT-PCR assays targeting human ribonuclease P (RNaseP) and 18S ribosomal RNA (18s rRNA) [20].

Genotyping SARS-CoV-2 from air and surface samples

The proportion of air and surface samples with mutations consistent with SARS-CoV-2 variants of concern (VOCs) were determined by PCR. The primers (Forward 5'-ACTTTCCTTTACAATCATATGGT-3' and Reverse: 5'- ACTACTCTGTATGGTTGGTAACC-3') and probes (5'-FAM-TTTCCAACCCACTAAT-MGB-3' and 5'-VIC- TTTCCAACCCACTTAT-MGB-3') were used for the assay to differentiate Asparagine or Tyrosine at residue 501 of spike protein. The primers (Forward: 5'- ACCTTTCTTTTCCAATGTTACTT-3' and Reverse 5'-TTAAATGGTAGGACAGGGTTATCAAA-3') and probes (5'-FAM- TTGGTTCCATGCTATCTC–MGB-3' and 5'-VIC- GTTCCATGCTATACATGT–MGB-3') were used to differentiate between the 69/70 deletion and wildtype spike protein.

Virus culture

Only samples with a Ct value of <30 would be cultured, because previous work showed that surface and air samples with a Ct value of >30 will not be culturable [12]. Vero E6 (African Green monkey

kidney) cells were used to culture virus from air and environmental samples. 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 [21].

SARS-CoV-2 laboratory surface stability assay

We performed a laboratory experiment to examine the stability and infectivity of SARS-CoV-2 dried on plastic surfaces. Three SARS-CoV-2 representative variants: Alpha (GISAID: EPI_ISL_693401), Beta (GISAID: EPI_ISL_770441), and Wildtype_D614G (GISAID: EPI_ISL_660788) were diluted to 1 x 10^5 PFU/mL. Five 2 µL droplets of virus culture were pipetted on a plastic surface (cell plates). The inoculated surfaces were dried in a safety cabinet for one hour after which they were visibly dry. The inoculated surfaces were soaked with 1 mL of virus transport medium for 30 minutes to elute the virus at three time points: 1, 24 and 72 hours. The samples were titred by the plaque assay as described previously [22].

Prevalence of variants in patients on the day of sampling

S gene target failure was being used routinely as a proxy to indicate infection caused by the Alpha (B.1.1.7) variant. We used ward admission and discharge dates in electronic patient records to determine which patients were in the clinical area on the day of sampling. A patient considered to have COVID-19 is one who had at least one positive SARS-CoV-2 PCR test within 14 days before the sampling day.

Ethics

In 2020, Imperial NIHR Biomedical Research Centre (BRC) developed the secure Clinical Analytics, Research and Evaluation (iCARE) high-performance analytics environment, which hosts secondary care data from Imperial College Healthcare NHS Trust (ICHT), and COVID-19 test results from North West London Pathology. The iCARE system provides linked health records from ICNT and NWL pathology, which have been de-identified and made available for approved research. This study was approved by the Imperial Academic Health Science Centre (AHSC) COVID Research Committee, the COVID-19 NWL Data Prioritisation Group, and the Discover Research Advisory Group (DRAG), which jointly provide a governance mechanism.

Results

No viable virus was recovered from any of the surface or air samples (Table 2). In the clinical areas that were selected for sampling in January 2021 as being comparable to those sampled in April 2020, the overall percentage of air and surface samples from which SARS-CoV-2 RNA was detected by PCR was significantly lower in January 2021 vs. April 2020 (Figure 1, Table 2). The overall percentage of surfaces contaminated with detectable SARS-CoV-2 RNA in April 2020 was 52% (114) of 218 surfaces compared with 5% (14) of 270 surfaces in January 2021 (p<0.001, Fisher's Exact Test). The overall percentage of air samples contaminated with detectable SARS-CoV-2 RNA in April 2020 was 48% (13) of 27 air samples compared with 4% (1) of 27 air samples in January 2021 (p<0.001, Fisher's Exact Test). SARS-CoV-2 RNA was detected in patient care areas and in nursing stations and staff rooms in April 2020, whereas SARS-CoV-2 RNA was only detected in areas occupied by patients or patient bathrooms in January 2021 (except for the lift buttons in the lift lobby of the main hospital building) (Figure 2, Table 2). At least one positive air sample was identified from every ward / area sampled in April 2020. In January 2021, the one positive air sample was detected in a bay dedicated to patients undergoing aerosol generating procedures.

There was no clear difference in the proportion of surfaces and air samples positive for SARS-CoV-2 RNA based on the type of ventilation in the ward. SARS-CoV-2 RNA was identified by PCR from 6% (5) of 80 surfaces and 12% (1) of 8 air samples from the two wards selected because they were naturally ventilated. The proportion of surface and air samples from naturally ventilated wards was not significantly different when compared to areas with mechanical ventilation (p>0.05 for both). There

was also no clear difference in the proportion of surface and air samples positive for SARS-CoV-2 in the renal dialysis unit: 2% of 40 surfaces samples and none of the four air samples.

51% of 180 of patients in the areas that were sampled had S gene knockouts consistent with the Alpha variant. 13/21 (62%) surface and air samples that detected SARS-CoV-2 RNA could be genotyped by PCR; 8 (38%) were Alpha variants.

In the laboratory stability assay, all three variants tested survived for at least 72 hours with a $<3\text{-log}_{10}$ reduction in viable count (Figure 3).

Discussion:

We undertook this study to compare SARS-CoV-2 surface and air contamination in the second wave of COVID-19 infection in acute care hospitals in London, UK compared with the first wave. Whilst SARS-CoV-2 RNA was detected in clinical and non-patient-care areas, no viable virus was recovered. Despite similar levels of bed occupancy by patients with COVID-19, the levels of air and surface RNA contamination in a range of clinical areas chosen to be comparable to the areas sampled in the first wave was significantly lower in the second compared with the first wave. There was no obvious correlation between the type of ventilation in the area and the level of surface and air contamination with SARS-CoV-2 RNA. SARS-CoV-2 RNA surface and air contamination was not notably different in a renal dialysis unit compared with the general ward setting. Approximately half of SARS-CoV-2 from patients in the clinical areas at the time of sampling and of SARS-CoV-2 RNA identified in surface and air samples was the Alpha variant. A laboratory study showed that the Alpha variant did not have notably different environmental survival properties compared with other variants.

The proportion of surface and air samples from which SARS-CoV-2 was detected was considerably lower in January 2021 during the second wave in the UK compared with April 2021 during the first wave. There may be several factors driving this difference, including enhanced prevention measures (summarised in Table 3) implemented between the COVID-19 waves, the emergence of new variants, changes in patient mix, the introduction of patient and staff vaccination, and changes in the use of clinical areas. It seems most likely that changes in prevention measures implemented between the two waves had the greatest impact on the levels of surface and air contamination that we measured.

Other studies have investigated surface and air contamination with SARS-CoV-2 [11-14, 23, 24]. Consistent with our findings, whilst most studies have identified at least some SARS-CoV-2 RNA on surfaces and air in patient care areas, few have been able to culture viable virus [13, 23, 24]. It is not clear why no viable virus was cultured from surfaces or air in our study. This could have been due to low viral load, methodological issues (such as choice of surface and air sampling technique, viral transport medium, or laboratory culture methods), or a combination of these factors. One study from the US found, as we did, a reduction in the proportion of surfaces from which SARS-CoV-2 RNA was detected from 11% to 2%, which they attributed to improved environmental and patient management practices [25]. Genotyping of the environmental samples found strong evidence that they originated from patients on the ward at the time of sampling.

Our laboratory study suggested that the three variants tested could survive for more than 72 hours when dried onto a plastic surface with a <3-$\log_{10}$ reduction. This rate of decay was not different to the other two variants that we tested, suggesting that differences in environmental persistence are not a factor driving the increased transmissibility of the Alpha variant [15]. Our findings on the environmental stability of these viruses is in line with the findings of others [9, 10, 26]. For example, one study evaluated the capacity of a range of SARS-CoV-2 variants, including the Alpha variant, to survive on stainless steel surfaces [26]. In this study, there was no clear difference in the capacity of the variants tested to survive on the steel surface, and all survived more than 72 hours with an approximate 3-$\log_{10}$ reduction.

SARS-CoV-2 is able to transmit more efficiently in indoor spaces with inadequate ventilation [6-8, 18]. Therefore, we evaluated whether differences in ward ventilation impacted the level of surface and in particular air contamination. We did not identify any differences in contamination level based on ward ventilation system. However, it's important to note that natural ventilation can provide efficient air changes if optimally designed [18], and we did not measure the effectiveness of the ventilation system as part of this study. There has been much debate about the role particle size in transmission of SARS-CoV-2 via the air [8]. Since we did not measure particle size in this study, we cannot add to this debate.

Patients who are dialysis dependent are particularly at risk of COVID-19 [17, 27]. They require regular visits to dialysis units with consequent contact with other dialysis dependent patients, live outside of the hospital, and require regular travel to dialysis units. The first wave of COVID-19 resulted in outbreaks in patients undergoing renal dialysis, with poor clinical outcomes [17, 28]. Our finding suggest that a high burden of surface and air contamination was not a feature of the epidemiology of COVID-19 in renal dialysis units.

Strengths of our study include the selection of a range of clinical and non-clinical areas to represent a breadth of clinical services provided by our hospitals, including a renal dialysis unit. The selection of clinical areas allowed us to compare contamination levels between the first and second waves. We sampled some wards with only natural ventilation to provide information on whether ward-level ventilation system affects contamination levels. The sampling methods used included both PCR and an attempt to culture live virus from environmental specimens. We made use of routinely collected data on the inferred genotype of SARS-CoV-2 from patients and undertook PCR genotyping on SARS-CoV-2 positive air and surface samples, which allowed us to comment on the proportion of the Alpha variant in patient and environmental samples. We undertook a laboratory evaluation of the survival properties of a range of SARS-CoV-2 variants, including the Alpha variant that was of particular interest at the time of the study.

Limitations of the study include that each area was sampled only once; without longitudinal sampling, our findings provide only a snapshot of contamination levels. We sampled exactly the same area where possible, but in some cases changes in the use of clinical areas between April 2020 and January 2021 meant that we had to choose comparable areas to sample. Whilst all clinical areas sampled were fully occupied by patients with COVID-19, we did evaluate the role of viral load and patient vaccination status on the shedding of SARS-CoV-2 into the environment. Samples were not collected from patients, air, and surfaces contemporaneously, meaning that we cannot link contamination levels to individual patients. The methods being used to provide regular reports on the inferred prevalence of the Alpha variant in patients assumed that patients were physically in the ward; this may not have been the case if patients were temporarily in different parts of the hospital, for example, for a procedure. Whilst we sampled two wards with only natural ventilation, several of the

other wards included parts of the ward with only natural ventilation. Also, we did not measure airflows or another measure of air quality (e.g. CO_2 levels of bacterial counts). Our study was undertaken before the emergence of the Omicron variant.

Our findings underline the potential risk of surface and air contamination in managing COVID-19, particularly during direct patient care. The findings suggest that COVID-19 prevention measures that have been introduced have reduced the level of surface and air contamination. The findings also suggest that enhanced ability to shed or survive on surface and / or in air are not the key driver for increased transmissibility of variants that have emerged recently. Based on these results, no changes in current practice are recommended. However, a continued focus on infection prevention and control activities is required to prevent the in-hospital transmission of COVID-19 [29].

Further work that would follow-on from our study includes longitudinal environmental sampling of surfaces and air in clinical and non-clinical areas to understand how patterns of contamination change over time. Further sampling should consider measurement of airflows to correlate airflows and environmental hygiene measures with contamination levels, measurement of particle size, genotyping of isolates, and linking environmental sampling to contemporaneous patient samples would allow to evaluate patient level risk factors for the shedding of SARS-CoV-2 such as viral load, duration of illness, and symptoms. Further work is required to understand the increased transmissibility of SARS-CoV-2 variants, and evaluating the role of patient and staff vaccination in the shedding of SARS-CoV-2 into the environment.

Our study reinforces that SARS-CoV-2 RNA can contaminate surfaces and air in healthcare settings, and suggests that enhanced infection prevention measures have reduced the burden of SARS-CoV-2 RNA on surfaces and air in healthcare. We did not find evidence that enhanced environmental survival properties are linked to the Alpha variant that was prevalent at the time of the study.

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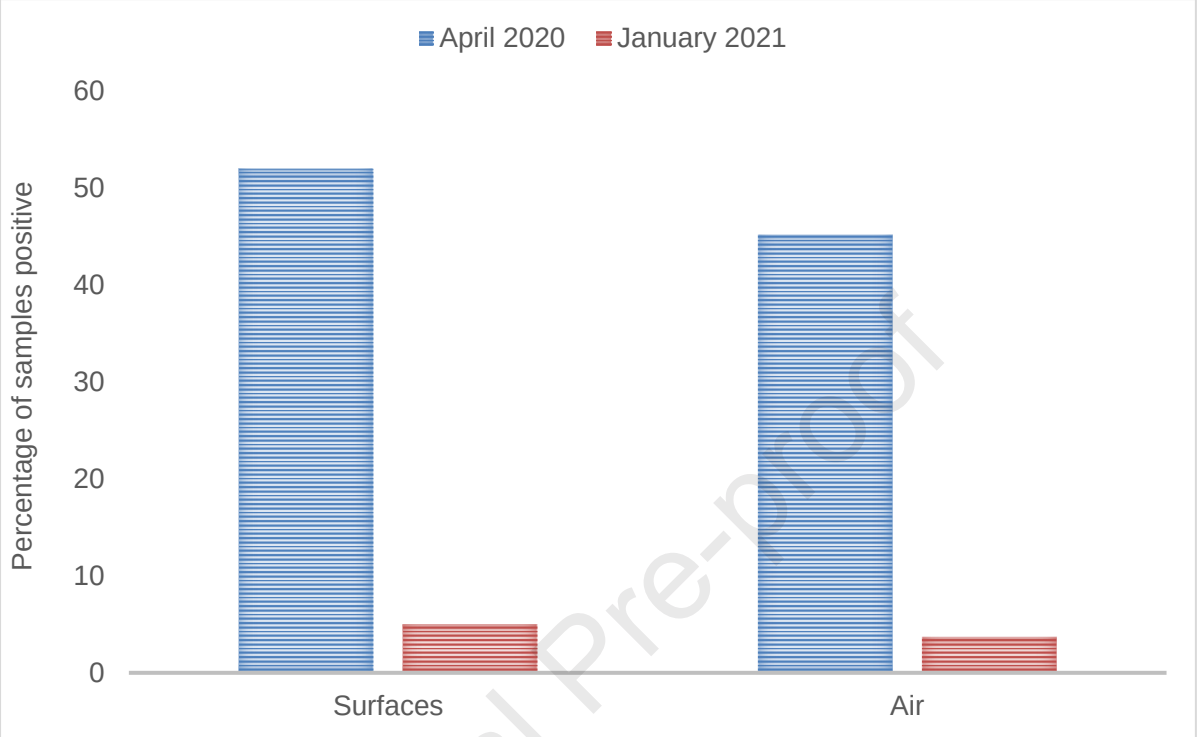
Potential conflicts of interest: JAO is a consultant to Gama Health Ltd and has given paid talks for ASP, Diversey, Ecolab, and Knowlex. All other authors declare no potential conflicts of interest related to this study.

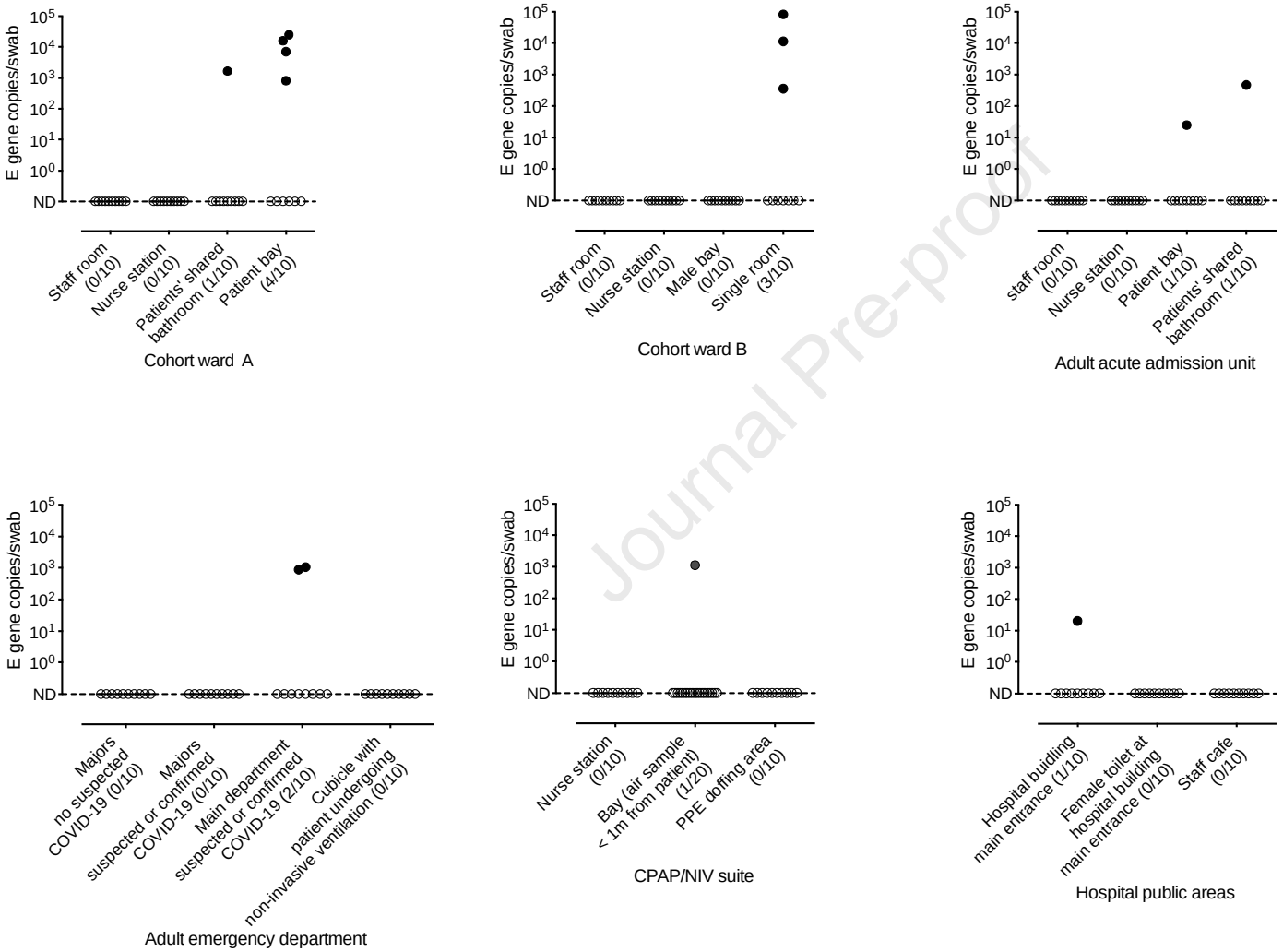
359 **Figure 1**: Overall percentage of surface and air samples positive for SARS-CoV-2 RNA in April 2020
360 vs. January 2021. 218 surfaces samples were collected in April 2020 and 270 in January 2021; 27 air
361 samples were collected in both April 2020 and January 2021.



362

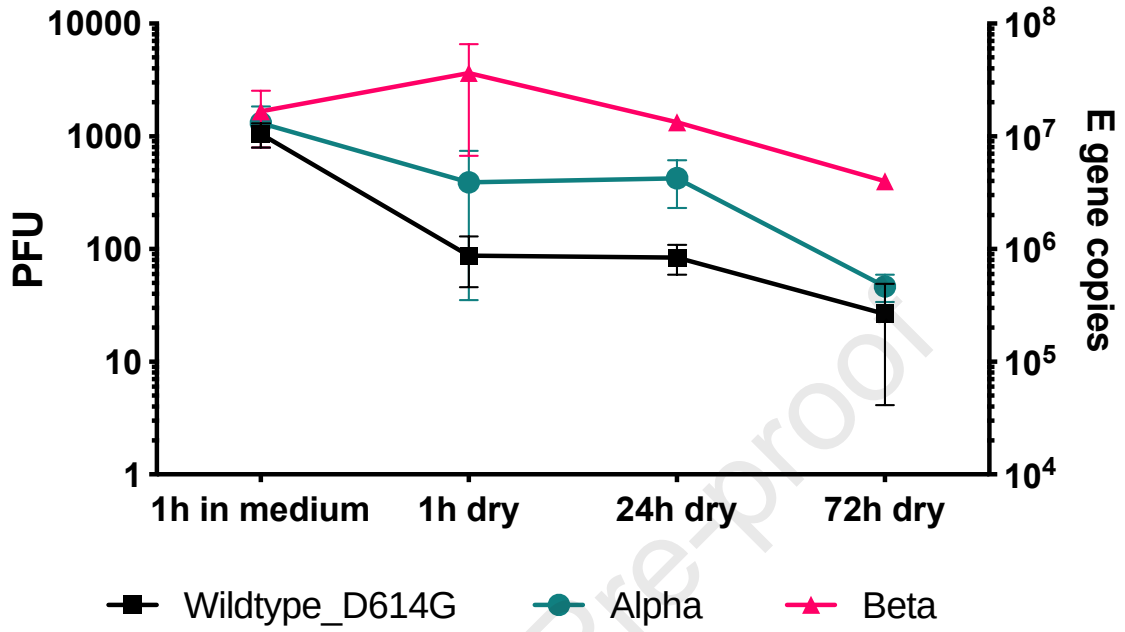
363 **Figure 2**: SARS-CoV-19 E gene copy number from surface swabs. The quantity of E gene copy number per swab is shown. Positive swabs and negative

364 swabs are indicated by solid dots and open dots respectively. All samples from the adult ICU were negative, so are not shown.



365

366 **Figure 3:** Survival of SARS-CoV-2 variants dried onto plastic surfaces. Mean and standard deviation
367 of PFU (plaque forming units) and E gene copies are shown.
368



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370 **Table 1:** Summary of areas sampled

Ward type	Ward details	Patient group	Ventilation type
Cohort ward A (patient bays and single rooms)	April 2020 (ward 1)	COVID-19 cohort ward	Mechanical supply and extract
	January 2021 (ward 2)	COVID-19 cohort ward	Mechanical supply and extract
Cohort ward B ("nightingale" design)	April 2020 (ward 3)	COVID-19 cohort ward	Recirculating
	January 2021 (ward 4)	COVID-19 cohort ward	Recirculating
Adult acute admission unit	-	Mixed cohort of patients with COVID-19 and other patients	Mechanical supply and extract
Adult emergency department	-	Mixed cohort of patients with COVID-19 and other patients	Mechanical supply and extract
Hospital public areas	-	-	Mechanical supply and extract
CPAP/NIV suite	April 2020 (ward 5)	CPAP/NIV for patients with COVID-19	Mechanical supply and extract
	January 2021 (ward 2)	CPAP/NIV for patients with COVID-19	Mechanical supply and extract
Adult ICU	-	Mixed cohort of patients with COVID-19 and other patients	Mechanical supply and extract
Inpatient dialysis unit	-	Mixed cohort of patients with COVID-19 and other patients	Mechanical supply and extract
Cohort wards with natural ventilation	Ward 6	COVID-19 cohort ward	Natural ventilation
	Ward 7	COVID-19 cohort ward	Natural ventilation

371

372 **Table 2**. PCR results from surface and air samples.

	Apr-20	Surfaces sampled	Surfaces positive	% positive	Air positive	Jan-21	Surfaces sampled	Surfaces positive	% positive	Air positive
Cohort ward A (Ward 1 April 2020; Ward 2 January 2021)	Staff room	6	2	33.3	Negative	Doctors' office	10	0	0.0	Negative
	Nurse station	6	4	66.7	Negative	Nurse station	10	0	0.0	Negative
	Patients' shared bathroom	6	2	33.3	Negative	Patients' shared bathroom	10	1	10.0	Negative
	Patient bay	6	5	83.3	**Positive**	Patient bay	10	4	40.0	Negative
Cohort ward B (Ward 3 April 2020; Ward 4 January 2021)	Staff room	4	0	0.0	Negative	Staff room	10	0	0.0	Negative
	Patients' toilet (in the ward)	7	1	14.3	**Positive**	Nursing station	10	0	0.0	Negative
	Male bay	12	5	41.7	**Positive**	Male bay	10	0	0.0	Negative
	Single room	8	7	87.5	**Positive**	Single room	10	3	30.0	Negative
Adult acute admission unit	Ward managers office	5	3	60.0	Negative	Staff room	10	0	0.0	Negative
	Nurse station	7	5	71.4	**Positive**	Nurse station	10	0	0.0	Negative
	Patient bay 2	8	2	25.0	Negative	Patient bay	10	1	10.0	Negative
	Patient bay 1	10	8	80.0	Negative	Patients' shared bathroom	10	1	10.0	Negative
Adult emergency department	'Green' majors (no suspected COVID-19)	10	6	60.0	Negative	Majors - no suspected COVID-19	10	0	0.0	Negative

	Nurse station	4	2	50.0	Negative	Majors - suspected or confirmed COVID-19	10	0	0.0	Negative
	Ambulatory waiting	3	3	100.0	Negative	Main department - suspected or confirmed COVID-19	10	2	20.0	Negative
	Patient assessment cubicles	3	1	33.3						
	Male toilet (next to the nurse station)	2	1	50.0						
	Resus bay (last patient > 2 hours)	10	4	40.0	**Positive**	Cubicle with patient undergoing non-invasive ventilation	10	0	0.0	Negative
Hospital public areas	Hospital building main entrance	7	5	71.4	**Positive**	Hosptial building main entrance	10	1	10.0	Negative
	Male toilet at hospital building main entrance	7	4	57.1	**Positive**	Female toilet at hospital building main entrance	10	0	0.0	Negative
	Lift area hospital building ground floor	10	4	40.0	Negative	Staff café	10	0	0.0	Negative
Continuous positive airway pressure (CPAP) / non-invasive ventilation (NIV) suite (Ward 5 April 2020; Ward 2 January 2021)	Nurse station	5	3	60.0	**Positive**	Nurse station	10	0	0.0	Negative
	Bay (air sample <1m from patient)	19	14	73.7	**Positive**	Bay (air sample <1m from patient)	20	1	5.0	Negative
	Bay (air sample >1m from patient)				Negative	Bay (air sample >1m from patient)				Positive
	PPE doffing area	5	2	40.0	Negative	PPE doffing area	10	0	0.0	Negative
Adult intensive care unit	Staff room	10	6	60.0	**Positive**	Staff room	10	0	0.0	Negative
	Nurse station inside intensive care unit	6	1	16.7	Negative	Nurse station inside intensive care	10	0	0.0	Negative
	Bay area	11	5	45.5	**Positive**	Bay area	10	0	0.0	Negative

	Single room	8	6	75.0	**Positive**	Single room	10	0	0.0	Negative
	Total	**218**	**114**	**55.6**	**13/27 (48.1%)**	**Total**	**270**	**14**	**5.2**	**1/27 (3.7%)**

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375 **Table 3: Key changes in COVID-19 prevention measures implemented between April 2020 and**

376 **January 2021**

	April 2020	**January 2021**
Patients	Symptomatic testing of patients.	Asymptomatic testing of all elective and non-elective admissions, and serial SARS-CoV-2 testing of all inpatients in place so more rapid identification of infected patients.
	No recommendation for surgical masks for patients.	Surgical masks for all patients (where possible).
	Standard bed spacing.	Improved bed spacing.
	No requirement for active identification and management of COVID-19 outbreaks amongst patients.	Active identification and management of COVID-19 outbreaks amongst patients.
Staff	No recommendation for surgical masks outside of direct patient care.	Universal surgical masks for all staff in healthcare buildings including in all clinical areas.
	No specific measures for office spaces.	'COVID-secure' measures in office spaces (including physical distancing).
	No routine staff testing.	Twice weekly lateral flow testing.
	Challenges with PPE use.	Improved compliance with recommended PPE (reductions in both excessive use and under use) and hand hygiene.
	No vaccination.	Initial implementation of a staff vaccination programme.
	Normal footfall on wards.	Reduced footfall on wards.
	No requirement for active identification and management of COVID-19 outbreaks amongst staff.	Active identification and management of COVID-19 outbreaks amongst staff.
Visitor/carer restrictions	Visiting permitted.	No ward visitors (outside of exceptional circumstances).
	No specific provision for enhanced hand hygiene at 2hospital entrances.	Welcome stations introduced to promote hand hygiene and masks at the entrance to our hospitals.
Environmental hygiene	No specific increase in ward cleaning.	Cleaning frequency increased to meet national recommendations. This included one additional clean for each clinical area, plus a further additional touchpoint clean. In addition, a new touchpoint cleaning programme began in public spaces.
Ventilation	No specific improvements in ventilation.	Exterior windows opened where safe and possible.

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379 **References**

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