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Quantifying the effectiveness of ultraviolet-C light at inactivating airborne *Mycobacterium abscessus*

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SUMMARY

Background: *Mycobacterium abscessus* (MABS) group are environmental organisms that can cause infection in people with cystic fibrosis (CF) and other suppurative lung diseases. There is potential for person-to-person airborne transmission of MABS among people with CF attending the same care centre. Ultraviolet light (band C, UV-C) is used for *Mycobacterium tuberculosis* control indoors; however, no studies have assessed UV-C for airborne MABS.

Aim: To determine whether a range of UV-C doses increased the inactivation of airborne MABS, compared with no-UVC conditions.

Methods: MABS was generated by a vibrating mesh nebulizer located within a 400 L rotating drum sampler, and then exposed to an array of 265 nm UV-C light-emitting diodes (LED). A six-stage Andersen Cascade Impactor was used to collect aerosols. Standard microbiological protocols were used for enumerating MABS, and these quantified the effectiveness of UV-C doses (in triplicate). UV-C effectiveness was estimated using the difference between inactivation with and without UV-C.

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Findings: Sixteen tests were performed, with UV-C doses ranging from 276 to 1104 μW s/cm^2. Mean (±SD) UV-C effectiveness ranged from 47.1% (±13.4) to 83.6% (±3.3). UV-C led to significantly greater inactivation of MABS (all *P*-values ≤0.045) than natural decay at all doses assessed. Using an indoor model of the hospital environment, it was estimated that UV-C doses in the range studied here could be safely delivered in clinical settings where patients and staff are present.

Conclusion: This study provides empirical in-vitro evidence that nebulized MABS are susceptible to UV-C inactivation.

Introduction

Mycobacterium abscessus (MABS) group are ubiquitous, rapidly growing non-tuberculous mycobacteria (NTM) comprising three subspecies — *M. abscessus* subsp. *abscessus*, subsp. *massiliense*, and subsp. *bolletii* [1]. MABS causes infection in people with cystic fibrosis (CF), has innate multidrug resistance, and its prevalence has increased in recent decades [2–4]. MABS is a substantial challenge for people with CF and the clinical teams treating them [5–7]. People with CF are more susceptible to MABS infection than the general population, and MABS infection leads to worse clinical outcomes [3,7,8].

Traditionally it was thought that MABS was acquired from environmental niches, as no clear evidence of cross-transmission had been observed in people with CF, but emerging evidence suggests person-to-person transmission of MABS can occur in CF centres [9–13]. All modes of transmission could contribute to MABS transmission indoors, including direct contact, indirect contact (i.e. surface contamination), large droplets (less or approximately equal to 20 μm in aerodynamic diameter), and airborne droplet nuclei (i.e. aerosols, less or approximately equal to 5–10 μm in aerodynamic diameter) [14,15].

The relative importance and plausibility of transmission modes is unknown, creating infection control and logistical challenges in the delivery of care for people attending specialist CF centres. For example, whole genome sequencing of MABS isolates highlighted possible transmission between five individuals with concurrent attendance at the same CF centre; no further cases occurred after both airborne isolation precautions and rigorous surface cleaning were introduced [16]. Airborne droplet nuclei survival was demonstrated by Fletcher *et al.* who showed that artificially generated MABS isolated from people with CF survived in air for up to 80 s and travelled 4 m, under controlled experimental conditions [17]. Similarly, under controlled conditions, airborne MABS was detected in cough aerosols from an individual with CF, chronically infected with MABS, at a distance of up to 4 m from the source and surviving for up to 45 min in air [15].

Ultraviolet light in band C (UV-C, wavelength 200–280 nm) has well-known microbicidal properties that inactivate or kill airborne microbes, and UV-C has been used for airborne *Mycobacterium tuberculosis* (TB) infection control indoors since the 1950s [18–29]. We recently undertook a systematic literature review on indoor air disinfection techniques for airborne respiratory bacteria, identifying 17 studies of UV-C, none of which included MABS [30]. Given the potential for MABS to survive in the airborne phase and the lack of previous studies on UV-C and MABS, we aimed to: (i) determine whether UV-C leads to a greater reduction in airborne MABS than in the absence of UV-C, and (ii) quantify the relationship between UV-C dose and airborne MABS survival under controlled conditions.

Methods

Tandem aged respiratory droplet investigator system (TARDIS)-rotator

The TARDIS-rotator (Supplementary Figure S1) is a sealable, horizontally oriented, mobile cylinder (diameter: 0.65 m; length: 1.2 m; volume: 400 L), which we have described in detail previously [31]. Briefly, the system allows storage and ageing of nebulized bioaerosols. It rotates at ∼1.7 rpm to limit particle deposition due to gravitational settling and inertial impaction. The drum is connected to compressed air, and high-efficiency particulate air (HEPA) filters, and has two outlet tubes connected to a TSI Model 9303 AeroTrak Particle Counter (OPC) (TSI, Shoreview, MN, USA) and an Andersen Cascade Impactor (ACI, Thermo Fisher Scientific, Waltham, MA, USA). In this study, the drum was additionally fitted with a UV-C light-emitting diode (LED) light source (265 nm) and a Mesh nebulizer placed and mounted inside the drum. Details of the TARDIS-rotator, UV-C design, and empirical UV-C validation are in the Supplementary Appendix.

Culture preparation and nebulization

A control strain of *M. abscessus* subsp. *abscessus* (ATCC 19977) *Mycobacterium abscessus* ATCC 19977 was used to prepare MABS suspensions for nebulization. The details are outlined in the Supplementary Appendix. MABS was aerosolized using a vibrating mesh nebulizer (VMN), described and validated previously [32–34]. The VMN generates aerosols without the need for a carrier air flow. Two millilitres of MABS suspension (∼5×10^4 cfu/mL) was injected to the nebulizer using a disposable syringe connected by a tube to the nebulizer. The aerosols were delivered into the drum at a rate of 1.5 mL/min. Each test, a total of 0.25 mL of the suspension was delivered over 10 s, leaving 1.75 mL remaining in the nebulizer.

Bioaerosol sampling

Aerosols were collected using a six-stage Andersen Cascade Impactor (ACI) at a flow rate of 28.3 L/min. The impactors have

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six size ranges (with lower cut-points ranging from 0.65 μm to >7 μm) and have been described previously [35]. Middlebrook 7H11 agar plates were loaded into the impactors. The OPC was used to measure particle number, concentration and size distribution of aerosols inside the drum at a flow rate of 2.83 L/min. There are six size channels with lower diameter cut-points from 0.3 to 10.0 μm. The air extracted by the impactor and the OPC was replaced with HEPA-filtered air [31]. The air exchange rate (AER) was ~0.36 AER of filtered air for each sample extracted (i.e. ~1.1 AER after extracting three samples). After finishing each test, the drum was flushed with HEPA-filtered air to purge residual aerosols through another HEPA filter and into a biosafety cabinet.

Enumeration of airborne MABS by culturing

Microbiological control plates were sealed by parafilm, put in a gas permeable bag, and incubated at 37 °C for at least 14 days for evidence of colony growth. The Andersen impactor plates were sealed by parafilm, covered with aluminium foil to negate potential for photoreactivation, placed in gas-permeable bags and incubated at 37 °C for 14 days. Plates were read at 7 and 14 days of incubation. The total airborne MABS colony counts were determined by summing all colonies (14-day reading) over impactor stages 1–6.

UV-C doses

Several UV-C doses were investigated, with the aim of spanning a range of UV-C effectiveness values. The power of the UV-C LEDs was 20, 40, 60, and 80% each, delivered over a duration of 20 min continuous exposure. The corresponding mean UV-C intensities were 0.23, 0.46, 0.69, and 0.92 $\mu W/cm^2$ respectively. The estimated doses (intensity multiplied by time (seconds)) were therefore 276, 552, 828, and 1104 $\mu W\ s/cm^2$, respectively.

Test and cleaning procedures

Each UV-C test took about 3 h to complete, including extensive post-test cleaning. Each test was repeated three times. Test and cleaning procedures are described in the Supplementary Appendix. Briefly, each test comprised a decay measurement with 5 min of impactor sample extraction, commencing approximately 5, 30, and 40 min after the MABS suspension had been initially nebulized into the drum (termed extractions A, B, and C, respectively). UV-C was delivered between extraction A and B. Each testing day, one control (no UV-C) test was performed to measure the natural decay of MABS in the drum, driven by the inherent biological decay of the organism and dilution with HEPA-filtered replacement air from sample extraction as described above.

Data analysis

To account for the probability that more than one viable MABS organism was deposited through one impactor hole on to agar plates (and therefore miscounted as a single colony-forming unit (cfu)), coincidence correction methods were used as described by Macher [36].

Estimates were based on the decay of airborne MABS over time, with and without UV-C, informed by standard analysis methods for bioaerosols published elsewhere [21,37]. Briefly, without UV-C, the exponential decay in MABS over time was driven by natural decay due to factors other than UV-C (i.e. replacement ventilation with HEPA-filtered air and the inherent biological decay of the micro-organism) [38]. When UV-C was operating, the total reduction in airborne MABS represented the combined effects of natural decay and additional UV-C inactivation. The equivalent air exchange rate (eAER) attributable specifically to UV-C (i.e. the amount of fresh air ventilation that would be required to yield the same removal as UV-C in a well-mixed space [37]) was defined as the difference in the decay slopes (natural logarithm of cfu count versus elapsed time in hours) of UV-C on and UV-C off [37].

The estimates of eAER were expressed as percentages (i.e. UV-C effectiveness, defined as the percentage of total MABS inactivation that is attributable to UV-C), following the methods of Ko *et al.*, who proposed that percentages are potentially more relevant to human infection risk assessment [18]. Descriptive statistics were calculated using both methods for comparison, while the additional analyses described below were based on percentages.

Continuous variables were summarized as mean and standard deviation (SD) if the data set was normally distributed. Otherwise, the median and range or interquartile range (IQR) were presented. Overall means and 95% confidence intervals (CI) were estimated for UV-C effectiveness for replicated tests. Analysis of variance was used to compare the means of UV-C effectiveness across UV-C doses. Tukey's honestly significant difference (HSD) was used as a post-hoc test. A paired *t*-test was used to compare the natural decay and total reduction across UV-C dose groups. $P < 0.05$ was considered significant and data were analysed using SPSS version 27.0 (IBM Corp., Armonk, NY, USA).

A multiple linear regression model was used to assess the effects of relative humidity (RH) inside the drum on UV-C effectiveness (with RH as a continuous variable and UV-C dose as a categorical variable). RH was controlled for, as it has been observed to affect the UV-C susceptibility of droplet nuclei of other *Mycobacterium* species [30]. Estimated marginal means and standard errors were calculated.

Finally we used a model of upper air UV-C, described elsewhere, to estimate whether required upper-air UV-C intensities to deliver the doses fall within occupational exposure limits [39,40]. This was based on actual outpatient clinical rooms in a CF centre we had previously measured (Supplementary Appendix) [41].

Ethics/safety approvals

The study was approved by the Queensland Children's Hospital and Health Service Human Research Ethics Committees (HREC/2007/RQCH/9, HREC/2014/QRCH/88) and QIMR Berghofer Medical Research Institute approval (P2074).

Results

Experiment summary and descriptive statistics

A total of 16 experiments were performed, of which four were control tests and 12 were UV-C tests (four doses, each performed in triplicate) undertaken over four days of testing.

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All swabs collected from the surfaces inside the drum at the beginning of the experimental days were culture negative for MABS.

The mean RH inside the drum and in the room where the experiments were performed was 79.2% (SD: 8.4) and 71.8% (SD: 5.0), respectively. The median temperature in the drum and room was 24.6 °C (IQR: 24.2–24.8) and 23.3 °C (IQR: 23.2–23.4), respectively.

The mean proportion of droplet nuclei <3 μm particle size measured by the OPC was 98.2% (SD: 0.3) at extraction A, 98.9% (SD: 0.2) at extraction B, and 99.2% (SD: 0.2) at extraction C. This remained consistent between tests of different doses (Supplementary Table S1). The mean viable MABS <4.7 μm (stages 3–6 of Andersen impactor plates) by extraction and UV-C dose are shown in Supplementary Table S2. On average, MABS <4.7 μm comprised 72.5% (SD: 19.4) of the total viable MABS in the impactor.

MABS inactivation and UV-C effectiveness

In control tests with no UV-C, the median natural decay of airborne MABS at extraction B (~30 min after nebulization) was 62.2% (IQR: 58.7–63.8) and at extraction C (~40 min after nebulization) was 75.5% (IQR: 72.1–76.9). Supplementary Figure S2 shows the change in airborne MABS over time, compared to extraction A.

The mean total reduction in airborne MABS increased from 83.1% (SD: 5.2) to 99.6% (SD: 0.3) as UV-C dose increased from 276 to 1104 μW s/cm^2. Corresponding total eAER ranged from 4.8 (SD: 0.9) to 15.4 (SD: 2.2) (Supplementary Table S3). The total reduction was significantly higher than natural decay at all doses assessed ($P \leq 0.045$) (Supplementary Table S4).

UV-C effectiveness is presented in Table I and Figure 1. In general, as UV-C doses increased, there was an increase in mean UV-C effectiveness. However, no significant difference in UV-C effectiveness was observed at doses ≥552 μW s/cm^2 (*P*-values ≥0.084, Supplementary Table S5). There was a significant difference in UV-C effectiveness between the lowest (276 μW s/cm^2) and two highest UV-C doses (828 and 1104 μW s/cm^2) assessed (Supplementary Table S5, $P = 0.002$). Corresponding eAER values attributable to UV-C ranged from 2.4 (SD: 1.1) to 12.9 (SD: 2.3) (Table I).

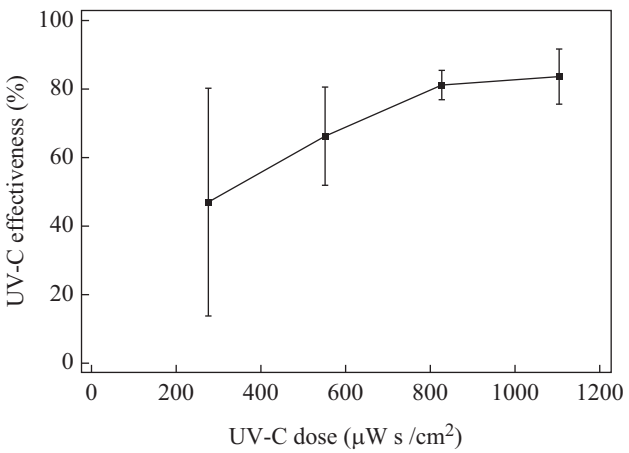

Figure 1. UV-C effectiveness (the proportion of total inactivation of airborne *Mycobacterium abscessus* (MABS) specifically attributable to UV-C after accounting for natural decay (units: %)) for airborne MABS at different UV-C doses. Points are means obtained from triplicates performed at different days of testing and error bars show 95% confidence intervals.

The multiple linear regression model with categorical terms for UV-C effects showed no apparent impact of RH on UV-C effectiveness (Supplementary Table S6). The addition of an interaction term between dose and RH was not significant ($P = 0.808$).

Indoor model

The estimated exposure times of MABS droplet nuclei in the upper 0.675 m of clinical rooms with ceiling height of 2.7 m (i.e. irradiated zone) were 1.2, 1.4, and 1.6 min/h, respectively (Supplementary Table S7). To deliver the maximum dose (at which we observed 84% inactivation of airborne MABS (1104 μW s/cm^2)), average UV-C intensities of 16, 13, and 11 μW/cm^2 in the irradiated zone would be required (Supplementary Table S8). For context, US Centers for Disease Control and Prevention (CDC)/National Institute for Occupational Safety and Health (NIOSH) guidelines for airborne *Mycobacterium tuberculosis* in occupied indoor healthcare settings recommend an average of 30–50 μW/cm^2 (for 254 nm UV-C) in the irradiated zone [42].

Assuming a traditional louvred wall-mounted UV-C fixture installed in the upper-room, CDC/NIOSH guidelines report that measured mean eye-level (1.5 m) intensity is 0.5% of that in upper air [42]. Applying 0.5% to the required intensities above yielded mean eye-level intensities of 0.07, 0.06, and 0.05 μW/cm^2 for 84% inactivation, respectively. Those estimates are 56%, 46%, and 40%, respectively, of the occupational limit for 265 nm UV-C over 8 h in any 24 h period (37 J/m^2) (Supplementary Table S9) [43]. The results for other UV-C doses are shown in Supplementary Table S9.

Discussion

Our study found that nebulized MABS remains viable in airborne droplet nuclei under controlled conditions, demonstrating the ability of MABS to survive for a longer period of time after aerosolization (up to 42.5 min) than 80.6 s as

Table I
Estimated mean UV-C effectiveness (the proportion of total inactivation of airborne *Mycobacterium abscessus* (MABS) specifically attributable to UV-C after accounting for natural decay), also expressed as their equivalent air exchange rates per hour (eAER)

UV-C dose (μW s/cm^2)	Total ($N = 16$)	UV-C effectiveness (%, SD)[a]	Equivalent air exchange rate due to UV-C (eAER/h, SD)
0[b]	4	61.3 (3.4)	2.5 (0.2)[c]
276	3	47.1 (13.4)	2.4 (1.1)
552	3	66.2 (5.7)	5.4 (1.6)
828	3	81.1 (1.7)	11.0 (0.03)
1104	3	83.6 (3.3)	12.9 (2.3)

[a] Estimated from eAER, as described in the main text.
[b] No UV-C test denoted as zero dose.
[c] Non-UVC eAER due to effects of natural decay from dilution ventilation and biological decay based on extractions A, B, and C.

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previously reported [17]. These findings are pertinent since previous observational epidemiological studies suggested that patient-to-patient transmission of MABS may occur via fomite or airborne routes among people with CF [14–16,44]. Collectively, these findings emphasize the importance of policies for control of airborne transmission of MABS between people with CF, particularly for CF inpatients or those attending the same clinic.

The observations described above, in addition to our previous study on the effectiveness of UV-C for airborne *Pseudomonas aeruginosa*, prompted our interest in determining whether UV-C could reduce the levels of viable airborne MABS under conditions broadly representative of indoor healthcare settings, as has been documented for TB and other NTM [21,23,24] (Nguyen *et al.*, unpublished study). UV-C increased the inactivation of MABS droplet nuclei among the doses we assessed, but no statistically significant reductions were observed at doses $\geq$552 μW s/cm^2. However, the change in effectiveness was seventeen percentage points between doses 552 and 1104 μW s/cm^2, which may be clinically important despite non-significant *P*-values. These results need to be corroborated with a larger number of replicates and doses to understand their repeatability, and the shape of the dose–effectiveness relationship.

The models we used, informed by measurements of room layout and ventilation rates, estimated that the highest UV-C dose we assessed can be safely delivered in occupied clinical settings. At the doses in the upper room required to achieve 84% MABS inactivation (i.e. the maximum dose we assessed), the equivalents in the lower room were from 40% to 56% of the 8 h occupational exposure limit for 265 nm UV-C, depending on the room. These estimates assume continuous eye exposure to UV-C for 8 h, a highly unlikely scenario in practice. An empirical study demonstrated that measured doses were between 0.5% and 8.8% of those calculated assuming continuous exposure, even for those who rarely leave their room [41]. In the scenario we modelled above, if 8.8% of the calculated continuous dose is received, that would equate to approximately 3.5–4.9% of the 8 h limit in the occupied part of the room. We caution that the estimates are indicative only and are intended to provide context for the results and inform hypothesis development for future studies.

While *M. tuberculosis* and its surrogates (*M. bovis* Bacillus Calmette–Guérin (BCG)) were the focus of most previous studies of UV-C, including pioneering work of Riley and colleagues, to our knowledge no comparable studies of airborne MABS have been reported [21]. Riley *et al.* also reported that *M. phlei* is one order of magnitude less susceptible than virulent *M. tuberculosis* and its surrogates (*M. bovis* BCG) [15]. Among other NTM species, Xu *et al.* reported average (SD) eAER values from 1.2/h (1.1) to 17/h (2.4) for *M. parafortuitum*, in a full-size test room at 3 AER and UV-C (254 nm) doses of approximately 1500 and 5000 μW s/cm^2 respectively (Z-value of 1.2 $\pm$ 0.15 $\times$ 10^{-3} cm^2/μW s) [45]. The eAER range we observed for MABS spanned the same order of magnitude, although the doses were lower in our study, which may be due to the different species, UV-C wavelength (265 versus 254 nm), or differences in experimental methods. Overall, the findings above highlight the need for further studies on UV-C and airborne NTM, as historical findings for *M. tuberculosis* and proxies may differ markedly from that required for NTM species.

This study had several limitations. (i) The number of experiments was limited (four UV-C doses, each tested in triplicate, plus four control tests), and each test had measurements at three time-points for capturing the decay of MABS. This may affect the representativeness of the results. (ii) We did not attempt to assess the effects of varying environmental conditions that may affect UV-C performance (e.g. room ventilation rates, levels of RH and temperature). Moreover, RH in this study (median: 79.9; range: 66.2–91.6) was somewhat higher than would be expected indoors (50–65%). Higher RH (greater than ~80%) might cause a reduction in UV-C effectiveness compared with low (less than ~30%) and medium (~40–60%) RH, as demonstrated in previous studies of UV-C for airborne bacterial pathogens [19,22,24,25]. Further studies are required to understand the extent to which UV-C effectiveness may vary for MABS over a wider range of RH conditions. (iii) UV-C doses were delivered over 20 min. Whereas previous studies suggest that UV-C effects on airborne bacteria do not substantially depart from the Bunsen–Roscoe law (i.e. that the effects of UV-C dose are not dependent on the time over which it is delivered), we could not confirm this empirically for MABS. (iv) One laboratory strain of MABS (*Mycobacterium abscessus* ATCC 19977) was used for nebulization and UV-C inactivation, which may not represent the clonal strains from CF patients and other strains and subspecies of MABS. (v) The suspension used for in-vitro nebulization in our study was 10% fetal bovine serum in phosphate-buffered saline. This may have different composition and hygroscopicity characteristics and it is unclear if our methods represent the in-vivo generation of respiratory aerosols containing MABS droplet nuclei indoors.

In conclusion, this study demonstrated that the addition of UV-C resulted in significantly greater inactivation of nebulized MABS in air, compared with no UV-C. This provides empirical in-vitro evidence that nebulized MABS are susceptible to UV-C. Modelling estimates suggested that UV-C doses can be delivered to the upper room air of occupied clinical settings, without posing undue safety risks to patients and staff. Future studies are warranted to determine UV-C effectiveness *in vivo*, using human-generated MABS aerosols from people with CF and other suppurative lung diseases.

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Conflict of interest statement

L.D. Knibbs reports financial support was provided by the Thoracic Society of Australia and New Zealand/Vertex and by the Rebecca L. Cooper Medical Research Foundation. S.C. Bell reports financial support was provided by the Cystic Fibrosis Foundation and by the National Health and Medical Research Council. Graham R. Johnson reports financial support was provided by the Australian Research Council. T.T. Nguyen reports financial support was provided by the University of Queensland. S.C. Bell reports administrative support, equipment, drugs, or supplies and statistical support were provided by the QIMR Berghofer Medical Research Institute, and administrative support, equipment, drugs, or supplies were provided by the Children's Health Queensland

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Appendix A. Supplementary data

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