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Plastic pollution as a novel reservoir for the environmental survival of the drug resistant fungal pathogen *Candida auris*

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ABSTRACT

The WHO recently classified *Candida auris* as a fungal pathogen of "critical concern". Evidence suggests that *C. auris* emerged from the natural environment, yet the ability of this pathogenic yeast to survive in the natural environment is still poorly understood. The aim of this study, therefore, was to quantify the persistence of *C. auris* in simulated environmental matrices and explore the role of plastic pollution for facilitating survival and potential transfer of *C. auris*. Multi-drug resistant strains of *C. auris* persisted for over 30 days in river water or seawater, either planktonically, or in biofilms colonising high-density polyethylene (HDPE) or glass. *C. auris* could be transferred from plastic beads onto simulated beach sand, particularly when the sand was wet. Importantly, all *C. auris* cells recovered from plastics retained their pathogenicity; therefore, plastic pollution could play a significant role in the widescale environmental dissemination of this recently emerged pathogen.

1. Introduction

Since the first report of *Candida auris* infection in 2009, cases of this human pathogenic yeast have been confirmed in over 45 countries and it is now recognised as a significant public health challenge (Akinbobola et al., 2023; Du et al., 2020). The magnitude of this is further amplified by the widespread resistance of *C. auris* to antifungal drugs, which limits treatment options and contributes to the high mortality rate associated with invasive *C. auris* infection (Carolus et al., 2021). Consequently, *C. auris* has recently been identified by the WHO as a critical pathogen requiring urgent research (World Health Organization, 2022). While previous studies have provided insight into the biology, genomics, and phylogeny of *C. auris*, the potential for this pathogenic fungus to survive and be disseminated in the natural environment is still poorly understood (Akinbobola et al., 2023), despite evidence suggesting that *C. auris* originally emerged from the natural environment (Arora et al., 2021; Escandón, 2022).

The role the environment plays in the epidemiology of pathogenic opportunist species of *Candida* is becoming increasingly recognised (Morio, 2020; Opulente et al., 2019). This is partly due to the number of at-risk individuals, such as the elderly or those with a compromised immune system, who could become colonised either directly or indirectly by pathogenic *Candida* species (Douglass et al., 2018; Steffen et al., 2023). Pathogenic *Candida* species have previously been isolated from environmental matrices, such as soil and river water, which offers the potential for subsequent human exposure (Opulente et al., 2019; Sautour et al., 2021; Steffen et al., 2023). Human interaction with these opportunistic pathogens can either lead to infection or asymptomatic colonisation, with the potential for subsequent transfer to a susceptible host. The first cases of *C. auris* being isolated from the environment came from coastal and wetland environments, demonstrating that *C. auris* can persist in marine environments (Arora et al., 2021; Escandón, 2022), where it is hypothesised this opportunist pathogen evolved from (Casadevall et al., 2019).

The ability of *C. auris* to form biofilms and persist on abiotic and biotic surfaces is critical for its persistence and nosocomial transmission (Ahmad and Alfouzan, 2021; Horton and Nett, 2020). Such persistence on plastic surfaces may also provide a pathway for survival and dissemination of *C. auris* in both clinical settings and the natural environment. Microbial biofilms can colonise the surfaces of environmental plastic pollution to form unique microbial communities known as the plastisphere (Zettler et al., 2013). Pathogenic bacteria are commonly detected in the plastisphere (Metcalf et al., 2022a) and can survive transitions through varying environmental matrices, with the potential for widescale dissemination within the landscape (Junaid et al., 2022; Meng et al., 2021; Metcalf et al., 2022b). However, despite the growing

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number of studies reporting human pathogenic bacteria and viruses in the plastisphere (Moresco et al., 2022; Silva et al., 2023), there is a significant lack of comparable studies quantifying the association of human pathogenic fungi with the plastisphere (Gkoutselis et al., 2021; Ormsby et al., 2023a).

The ongoing global spread of drug-resistant strains of *C. auris* in clinical and healthcare settings could lead to pathogenic strains of *C. auris* being released into the environment either on fomites such as contaminated clinical materials (Akinbobola et al., 2023; Welsh et al., 2017), or via wastewater effluent (Barber et al., 2023; Welsh et al., 2017). Consequently, there is an urgent need to understand the potential for *C. auris* to persist in the plastisphere in a range of environmental matrices with the potential for human exposure (Akinbobola et al., 2023). Therefore, the aim of this study was to investigate the persistence of *C. auris* on plastic surfaces in river water and seawater and the potential for subsequent transfer to beach sand.

2. Methods

2.1. Survival of planktonic C. auris cells in mesocosms

Two fluconazole resistant *C. auris* isolates (both from Clade 1), *C. auris* NCPF 8973 (a non-aggregating strain) and *C. auris* NCPF 8983 (an aggregating strain) were grown overnight in Yeast Peptone Dextrose (YPD) (Sigma-aldrich, UK) broth at 37 °C for 18 h. *C. auris* cells were recovered by centrifugation at x8,000*g* for 8 min, washed twice in PBS and resuspended in sterile distilled water. Glass wide-mouth conical flasks (four replicate flasks for each type of water) containing 198 mL of either freshly collected river water, seawater, or sterile distilled water were inoculated with cells of *C. auris* by adding 2 mL of inoculum (1×10^8 CFU/mL). All flasks were plugged with foam stoppers and incubated at 15 °C with continuous shaking at 60 RPM. Salinity, electrical conductivity (EC) and turbidity of the water prior to inoculation were measured with, a salinity refractometer (RGBS), a HI2550 EC meter, and HI88703 turbidimeter respectively (Hanna Instruments, UK) (Table S1). The concentration of *C. auris* was measured at days 0, 1, 2, 3, 4, 5, 6, 8, 10,13,16,19, 25 and 31 by removing 1 mL of water from each flask, serially diluting in PBS, and plating out on Sabouraud Dextrose Agar (SDA) (Sigma-aldrich, UK) supplemented with chloramphenicol (50 μg/L), gentamicin (50 μg/L), and fluconazole (45 μg/L). All plates were incubated at 37 °C, and colony forming units (CFU) enumerated after 48 h.

2.2. Persistence of C. auris cells on plastic and glass surfaces

Bespoke mesocosms were constructed to provide replicate plastic surfaces for colonisation by *C. auris* in simulated environmental conditions. A detailed description of the design of these mesocosms is provided in (Ormsby et al., 2023b) but briefly, sheets of blue high-density polythene (HDPE) (20 μm; Thali outlet, Leeds, UK) were sandwiched between two high-grade stainless-steel frames (200 mm × 140 mm × 1.5 mm) with six 40 mm × 40 mm cut outs. In addition, replicate frames were used to hold microscopic glass slides to provide glass surfaces for colonisation. To form a natural biofilm on the surface of both the plastic and glass, all frames were submerged in fresh river water in a 4 L glass tank, kept aerated with an air stone. The frames, now colonised by a natural biofilm, were removed after 72 h and allowed to dry at room temperature.

Inoculum from overnight cultures of the two *C. auris* strains were generated as described above, resuspended in a suspension of sterile human faecal material (10 mg/mL), and thoroughly homogenised by vortexing. Plastic and glass surfaces on each replicate frame were inoculated with a 300 μL *C. auris* suspension and allowed to dry at room temperature for 48 h before the frames were submerged in tanks containing 3 L of freshly collected river water or seawater. A set of replicate frames was also added to a tank with no water to mimic environmental desiccation conditions. The physiochemical conditions of the water were measured as described above, prior to the inoculated frames being added to the tanks (Table S2), and temperature was monitored throughout the duration of the experiment (Table S2). At days 0, 1, 2, 4, 6, 9,12,16, 20, 25 and 30, replicate frames were removed from the tanks, and *C. auris* cells recovered by adding 300 μL of PBS onto the plastic or glass surface and gently scraped with a sterile plastic inoculating loop. In addition, the water in each replicate tank was sampled to quantify those *C. auris* cells that had been transferred into the water from the biofilm. Viable *C. auris* cells were enumerated by culturing on supplemented SDA as described above.

2.2.1. Virulence of C. auris recovered from plastic and glass surfaces

To determine the retention of virulence following their persistence in simulated environmental conditions, cells of *C. auris* recovered on Day 0 from both the plastic and the glass surfaces, and the cells recovered from the longest time points (where there were still at least three replicates of viable cells) were introduced into a *Galleria mellonella* infectivity model according to (Romera et al., 2020). *C. auris* cells were grown overnight and resuspended in PBS prior to this assay, as described above. Healthy *G. mellonella* larvae (Livefood, Axbridge, UK), measuring from 2 to 2.5 cm in length and showing no signs of melanisation were used for all virulence assays. Groups of 10 larvae were each injected with 10 μL of *C. auris* cells (approx. 10^5 cells/larva) into the hemocoel via the last right pro-limb by using a Hamilton syringe. Following pathogen challenge, larvae were placed in an incubator at 37 °C, and survival monitored over a 5-day period, with larvae considered dead when non-responsive to touch. Experiments were conducted in biological triplicate. Virulence was assessed relative to *C. auris* cells recovered at Day 0. For all challenge experiments, an inoculation of 10 μL of PBS was used as a control ($n = 10$ larvae) to account for mortality caused by physical injury or infection by a contaminant.

2.3. Persistence of C. auris cells on microplastic particles and subsequent transfer into beach sand

Environmental biofilms were formed on the surfaces of 2 mm polyethylene plastic microbeads (Goodfellow, Cambridge, UK) and 2 mm glass microbeads (Sigma-Aldrich, Canada) by placing 120 plastic or 120 glass microbeads in replicate spherical stainless-steel metal cages (45 mm high, 38 mm diameter, 1 mm pore size; Golf, China) and submerged in fresh river water in a 4 L glass tank. After 72 h, the metal cages were removed from the tanks, and 120 plastic and 120 glass microbeads transferred into replicate wide neck conical flasks containing 100 mL seawater that had been inoculated with *C. auris* NCPF 8983 to a final concentration of 1×10^6 CFU/mL. All flasks were incubated at 15 °C with continuous shaking at 60 RPM for 48 h to allow *C. auris* to form biofilm on the microbeads.

To simulate contaminated microbeads washing up on the beach, Petri dishes were packed with either dry or wet freshly collected beach sand (the 'wet sand' was kept moistened by the addition of fresh seawater). Selected physicochemical properties (pH and EC) of the dry and wet soil samples were measured at the beginning of the experiment (Table S3), while the temperature and moisture content of the sand samples were measured at intervals throughout the duration of the experiment. Microbeads were placed on the surface of the sand (120 beads of plastic or 120 glass microbeads for each replicate Petri dishe) and all Petri dishes were kept at room temperature throughout the duration of the experiment. Survival of viable *C. auris* cells colonising the microbeads was quantified at 0, 1, 2, 4, 6, 9,12,16, 20, 25 and 30 days by using sterile forceps to transfer 100 beads from each replicate Petri dish into a 15 mL Falcon tube containing 2 mL of PBS. This tube was vortexed vigorously at 1500 rpm for 3 min and the wash solution removed and serially diluted in PBS and plated out onto supplemented SDA as described above for enumeration. The transfer of *C. auris* into the sand from the plastic and glass microbeads was also quantified at each

time point by adding 5 g of sand to 5 mL of PBS, vortexing and plating out as described above. Four replicate Petri dishes were used at each time point for both plastic and glass microbeads.

The virulence of recovered *C. auris* cells from both the plastic and glass microbeads at Day 0 and the longest time point that still had at least three replicates of viable cells was evaluated using the *G. mellonella* model as described above.

2.4. Statistical analysis

Significant differences in persistence over time between aggregating phenotypes of *C. auris*, or between persistence on the surface of plastic and glass was evaluated using a two-way ANOVA in GraphPad prism software (V6.07). *C. auris* virulence in *G. mellonella* was expressed as percentage survival of *G. mellonella* larvae inoculated with *C. auris* over a duration of five days. Kruskal-wallis and a post hoc Dunn's test was used to compare the survival of *G. mellonella*.

3. Results

3.1. Persistence of planktonic C. auris in the aquatic environment

Both *C. auris* strains persisted for over 30 days in all three water types (Fig. 1), with greater survival in sterile distilled water (<1 Log_{10} reduction in the number of viable *C. auris* cells) compared to river water and seawater after 30 days. There was an initial rapid reduction in viable cells during the first 12 days in both river water and seawater mesocosms after which the concentration of viable cells stabilised for the rest of the sampling period. Approximately 2 Log_{10} CFU/mL of viable *C. auris* cells persisted in the river water and seawater mesocosms by the end of the sampling period.

There was no difference in the survival of either the non-aggregating or aggregating strains in the three water types (Fig. 1). Overall, the initial decline in viable cells of both *C. auris* strains was more rapid in river water compared to seawater. A significantly higher number of

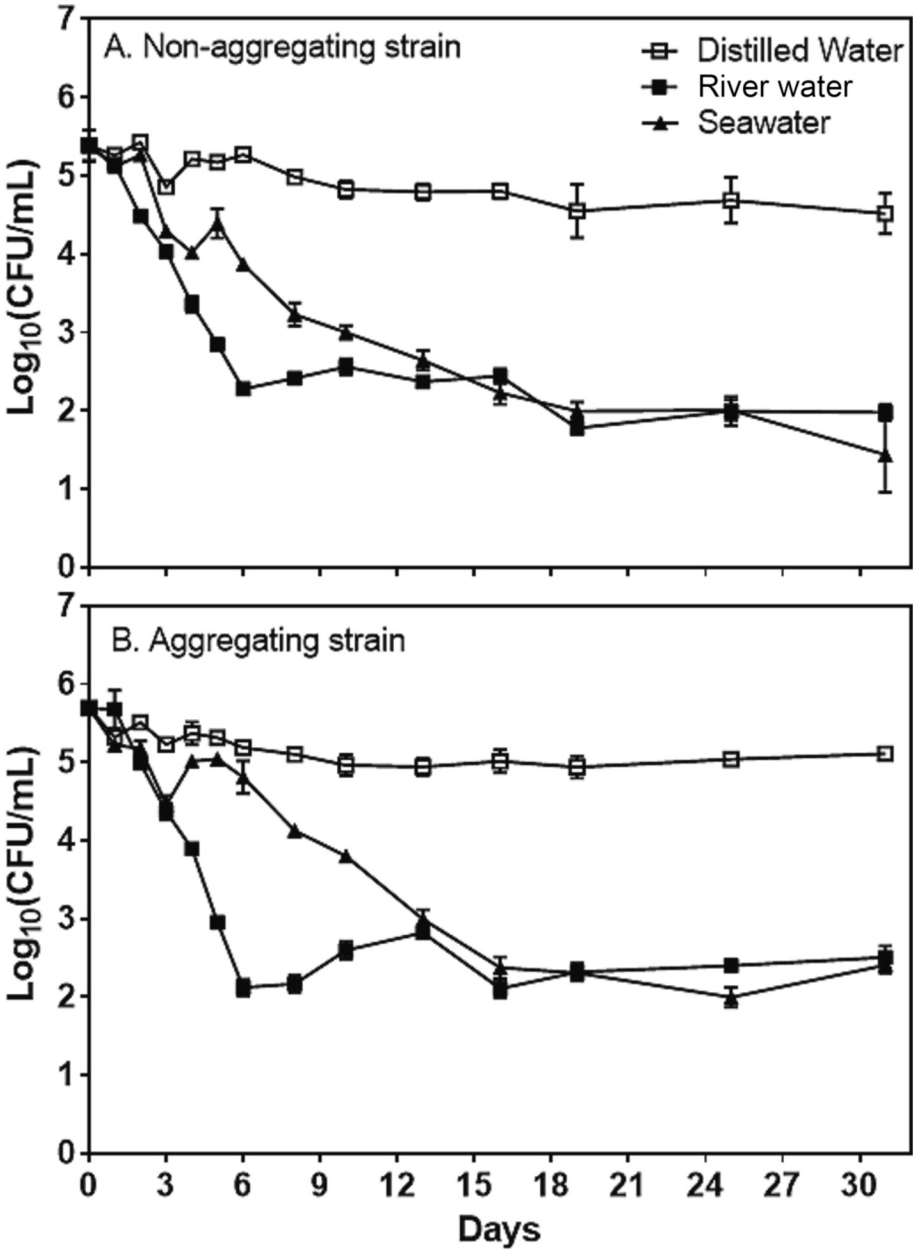

Fig. 1. Persistence of, (A) a non-aggregating strain of *C. auris* and (B) an aggregating strain of *C. auris* in sterile distilled water, river water and seawater mesocosms. Data points are the mean of four replicates ± SE.

viable cells of the non-aggregating strain was recovered from seawater compared to river water from day 4 to 8 (Fig. 1A; $p < 0.01$). Similarly, a significantly higher number of viable cells of the aggregating strain was recovered from the seawater compared to the river water from days 4 to 10 (Fig. 1B; $p < 0.001$).

3.2. Persistence of C. auris on plastic and glass surfaces submerged in river water or seawater

The non-aggregating strain survived better compared to the aggregating strain on dry unsubmerged surfaces, with significantly more viable cells ($p < 0.001$) recovered from both plastic and glass surfaces compared to the aggregating strain after day 25 (Fig. 2A). Both *C. auris* strains persisted on plastic and glass surfaces submerged in river water and seawater for at least 30 days (Fig. 2), with a gradual linear decline. There was approximately a 4 Log_{10} reduction in the viability of both *C. auris* strains on both plastic and glass submerged in river water, although there was no significant difference in the die-off rate between the non-aggregating and aggregating strains (Fig. 2B). In seawater, the concentration of *C. auris* cells on the surfaces of both plastic and glass followed a triphasic die-off (Fig. 2C). The number of viable cells recovered from the surfaces submerged in seawater generally declined until day 9 for the aggregating strain on plastic surfaces and day 15 for the non-aggregating strain, after which there was a temporal increase before a further gradual reduction in the number of viable cells (Fig. 2C). However, there was no significant difference in the die-off rate between the non-aggregating and aggregating *C. auris* strains on either the plastic or glass submerged in the seawater mesocosms.

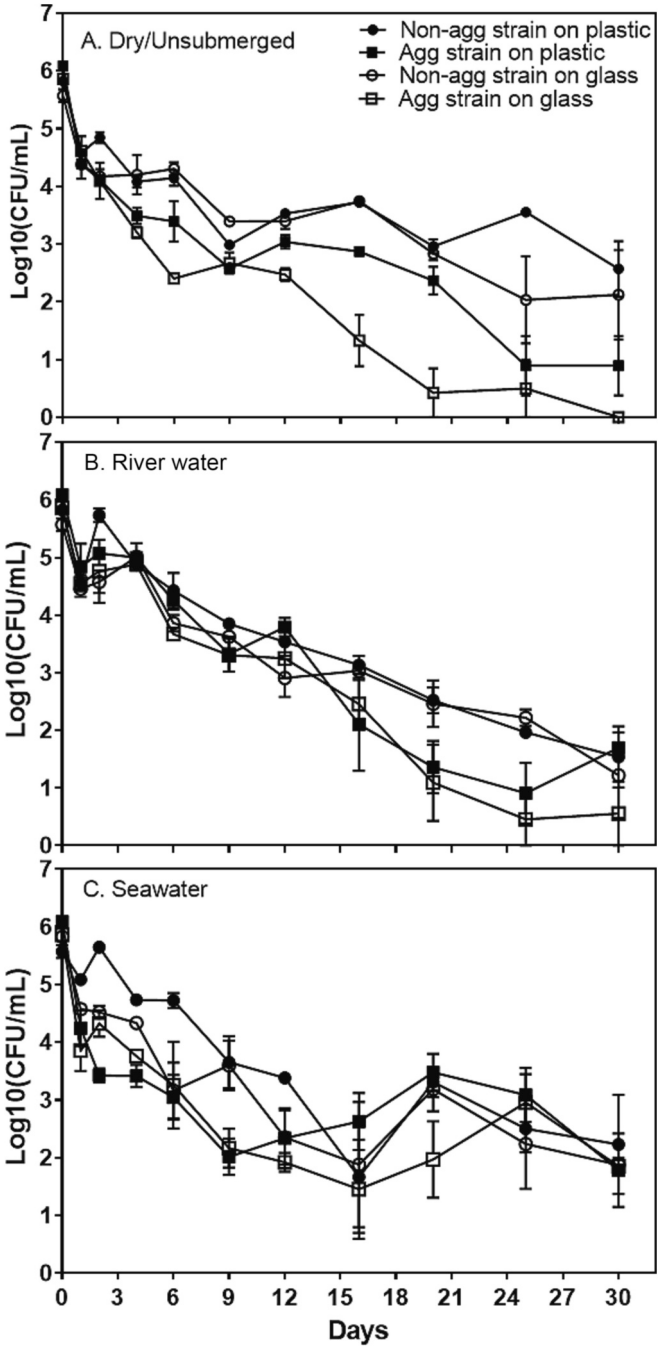

Fig. 2. Persistence of *C. auris* NCPF8973 (non-aggregating strain) and *C. auris* NCPF8983 (aggregating strain) on the surface of either plastic or glass in, (A) dry unsubmerged conditions; (B) submerged in river water; and (C) submerged in seawater. Data points are the mean of four replicates ± SE.

Virulence of *C. auris* cells recovered from plastic and glass surfaces at selected time points was compared to *C. auris* cells recovered at day 0 (Fig. 3). All isolates recovered from the surfaces of plastic and glass, exposed to dry conditions or submerged in either river water or seawater, retained their virulence in the *G. mellonella* model, with no significant difference in pathogenicity compared to *C. auris* cells recovered at day 0 (Fig. 3).

3.3. Persistence of C. auris colonising microbeads on the surface of beach sand

A higher concentration of *C. auris* cells initially colonised plastic microbeads compared to the glass microbeads following the initial 48 h in contaminated seawater. *C. auris* was recovered from the plastic microbeads on the dry sand for the whole 30 d period, whereas on the glass microbeads *C. auris* was only recovered for the first 12 days (Fig. 4A). Significantly more *C. auris* cells were recovered from plastic ($p < 0.05$) and glass ($p < 0.005$) microbeads on wet sand compared to dry sand. On the plastic microbeads, there was an approximately 1 Log_{10} reduction in viable *C. auris* cells on the wet sand, compared to a 2 Log_{10} reduction on dry sand. On the wet sand, *C. auris* survived better on the plastic microbeads compared to the glass microbeads, with significantly more cells ($p < 0.001$) recovered from the plastic microbeads after 30 days. There was a 3 Log_{10} reduction in the concentration of viable cells recovered from glass microbeads after 30 days on wet sand, compared to a 1 Log_{10} reduction of *C. auris* cells on plastic microbeads on wet sand.

There was transfer of *C. auris* from both plastic and glass microbeads into both wet and dry sand for at least the first nine days (Fig. 4B). *C. auris* continued to be transferred from the biofilm on the plastic microbeads and/or persist in both the dry and wet sand (Fig. 4B). However, although *C. auris* continued to persist in the wet sand following transfer from the glass microbeads, after nine days there was no subsequent survival in the dry sand. Although similar concentrations of viable cells were initially transferred to the sand with the plastic microbeads, the concentration of viable cells recovered after 30 days from wet sand was significantly higher ($p < 0.001$) than from the dry sand. The concentration of viable *C. auris* cells in the wet sand transferred from both the plastic and glass microbeads was approximately 3 Log_{10}CFU/g after 30 days compared to approximately 1 Log_{10} CFU/g transferred from the plastic microbeads into the dry sand.

Virulence of cells recovered from the microbeads on day 0, was compared to the virulence of cells recovered from plastic microbeads in both the wet and dry sand mesocosms on day 30, and the cells recovered from the glass microbeads on the wet sand on day 30 and glass microbeads on dry sand on day 9. All isolates recovered from the surfaces of the plastic and glass microbeads, and from both the wet and dry sand, retained their virulence in the *G. mellonella* model, with no significant difference in the pathogenicity compared to *C. auris* cells recovered at day 0 (Fig. 5).

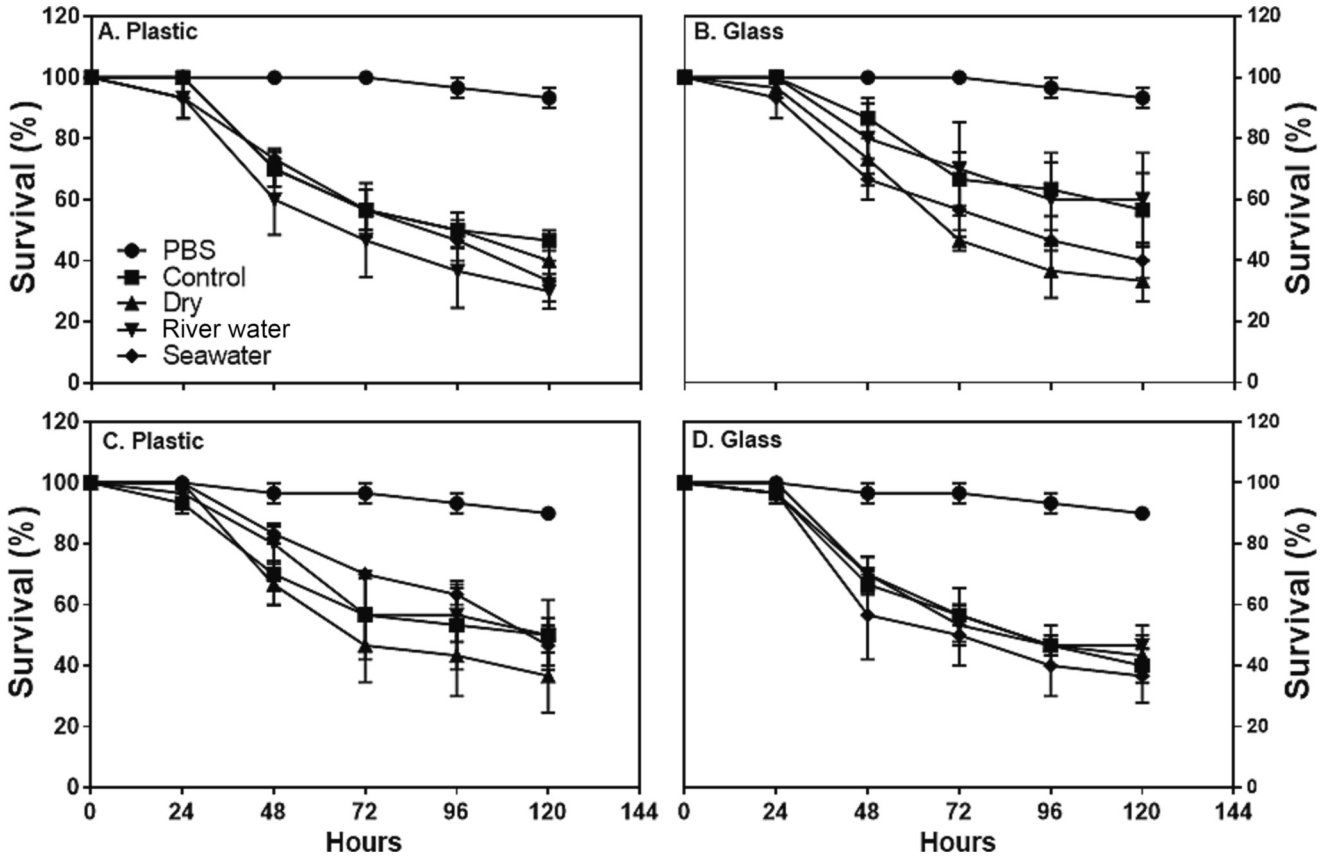

Fig. 3. Survival of *G. mellonella* larvae inoculated with a non-aggregating strain of *C. auris* (A and B) and an aggregating strain of *C. auris* (C and D) recovered from the surfaces of plastic or glass that had been kept in either freshwater, seawater, or unsubmerged mesocosms. An isolate of *C. auris* that had not been through the mesocosms was used as the 'control' and an inoculation of just PBS was used as a non-*Candida* control.

4. Discussion

The persistence of *C. auris* in the clinical environment has been widely studied, yet despite its likely origin in the natural environment the ability of *C. auris* to persist under environmental conditions has never before been reported. Here, we have demonstrated that clinical strains of *C. auris* can persist in both river water and seawater for at least a month, with both non-aggregating and aggregating strains showing similar survival profiles. During the first ten days the rate of die-off was less rapid in seawater, supporting the halotolerant attribute of *C. auris* (Welsh et al., 2017). However, the greater rate of survival in distilled water demonstrates that the combination of abiotic conditions and autochthonous microbial communities can influence the survival of *C. auris* in the environment, with competition for limited resources and predation likely contributing to this initial decline. Similarly, *C. albicans,* the most common human pathogenic candida species, can persist in filtered water for >240 days (Chaieb et al., 2011), but only about four days when suspended in-situ in marine and river water (Valdes-Collazo et al., 1987).

Recently, *C. auris* has been detected in both influent and effluent from wastewater treatment plants (WWTP) serving hospitals, particularly in areas reporting *C. auris* outbreaks (Babler et al., 2023; Barber et al., 2023; Rossi et al., 2023). As microplastics are also major components of wastewater effluents (Okoffo et al., 2019), there is the opportunity for pathogenic *Candida* to colonise and form biofilms on the surfaces of plastic pollutants. Subsequent discharge of wastewater into rivers or coastal environments, could then facilitate the widescale dissemination of *C. auris* on the surfaces of microplastics, particularly as plastics are so buoyant and resistant to degradation. Transfer of microplastics to areas such as sandy beaches and bathing waters is well documented (Bridson et al., 2020; Rapp et al., 2020) and facilitates the persistence of pathogens in areas where human exposure is more likely (Metcalf et al., 2022b).

C. auris colonises human skin where it can persist and subsequently be transferred to other hosts (Schelenz et al., 2016; Tharp et al., 2023), and the survival of *C. auris* in aquatic environments could facilitate increased exposure to human skin during recreation, swimming, or bathing. Contaminated coastal water is a well-known exposure route for pathogenic bacteria such as *Staphylococcus aureus* and *Escherichia coli* (Leonard et al., 2015; Plano et al., 2011); however, it has also been suggested that yeasts are frequently shed from humans into bathing waters (Brandão et al., 2021; Stevens et al., 2012). Thus, the potential shedding of *C. auris* from humans directly into either freshwater or marine bathing waters (together with subsequent persistence), could facilitate the potential transfer between humans over a significant period of time. The likelihood for this is further amplified by the ability of *C. auris* to persist asymptomatically on human skin where it can be subsequently transmitted to susceptible individuals.

In clinical settings, *C. auris* can survive desiccation for >14 days on non-porous surfaces (Horton et al., 2020). However, our study has demonstrated that when introduced into the environment as desiccated biofilm on the surface of either plastic or glass, *C. auris* can persist for at least a month, with increased survival on plastic surfaces compared to glass. Cell phenotype also influences *C. auris* die-off dynamics; for example, on dry unsubmerged surfaces, the decline of the non-aggregating strain was slower than the aggregating strain. This may be due to the different biofilm forming abilities of these isolates, with strains of non-aggregating *C. auris* capable of forming better biofilms compared to the aggregating strains (Sherry et al., 2017; Singh et al., 2019). Colonisation of plastic surfaces will have important implications

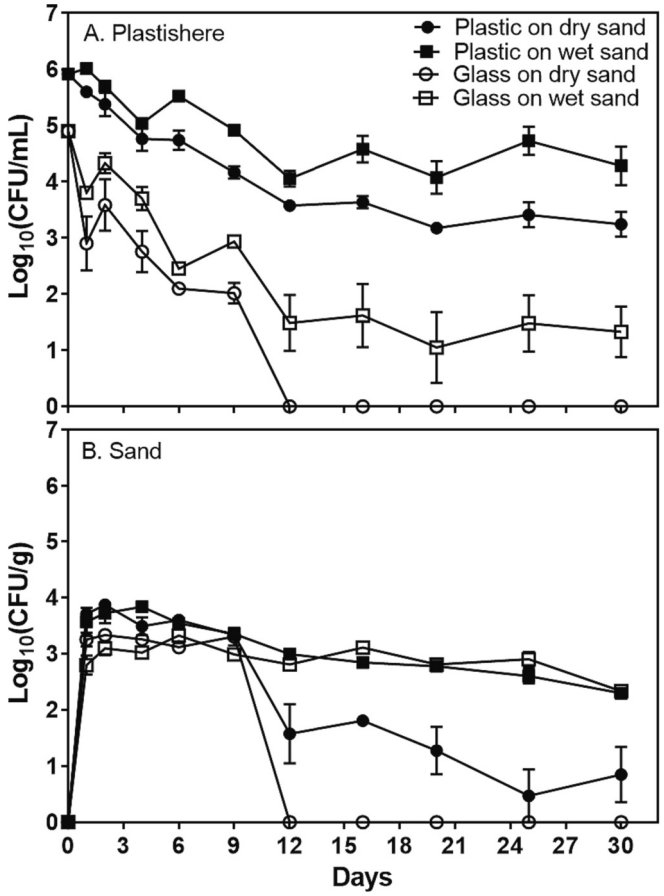

Fig. 4. Persistence of an non-aggregating strain of *C. auris*, (A) colonising plastic and glass microbeads placed on top of either dry or wet sand; and (B) transfer and subsequent persistence of *C. auris* into the dry or wet sand from the plastic or glass microbeads. Data points are the mean of four replicates ± SE.

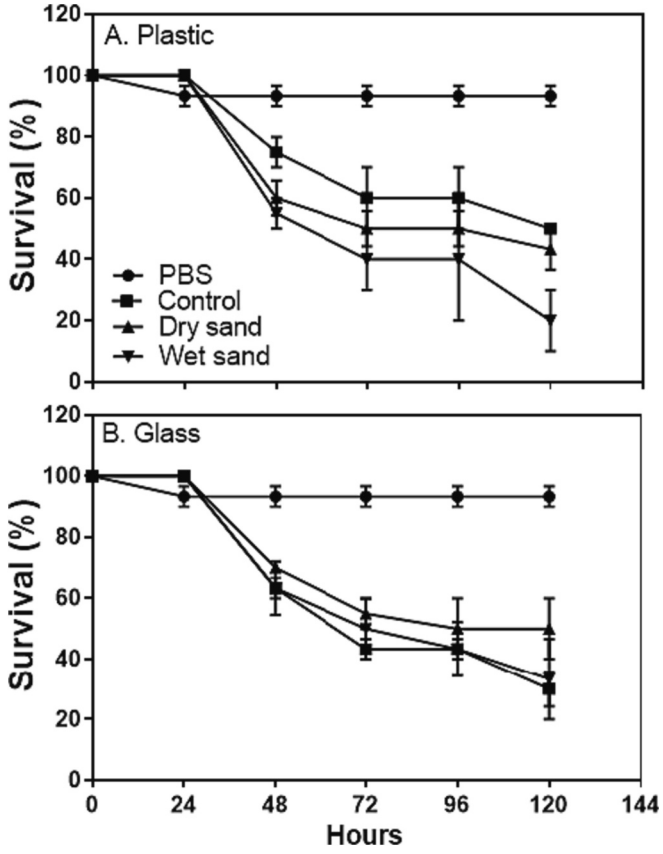

Fig. 5. Survival of *G. mellonella* larvae inoculated with a non-aggregating strain of *C. auris* recovered from (A) plastic and (B) glass microbeads maintained on dry or wet sand. An isolate of *C. auris* that had not been through the mesocosms was used as the 'control' and an inoculation with just PBS was used as a non-*Candida* control.

for human exposure to, and interaction with, plastic pollution (Metcalf et al., 2022a), and for the further dissemination of fungal pathogens if contaminated plastics are introduced into an aquatic environment, e.g., through flooding or transfer into rivers and coastal environments, or if they become incorporated into soil (Gkoutselis et al., 2021).

The difference in die-off dynamics of *C. auris* colonising plastic and glass surfaces in river water and seawater suggests a differential tolerance of these environmental conditions. In contrast to the linear decline of viable cells in surface biofilms in river water, the population of viable cells on both the plastic and glass surfaces submerged in seawater declined for the first two weeks before stabilising, or even increasing, in concentration. This greater halotolerance may reflect the evolution of *C. auris* (Casadevall et al., 2019) or demonstrate the protective role of the plastisphere for tolerating environmental stressors such as salinity (Metcalf et al., 2022a). There was no difference in the survival dynamics of *C. auris* on thin plastic sheets compared to glass; in contrast, *C. auris* generally survived better on plastic microbeads compared to glass microbeads. Increased colonisation of plastic surfaces has previously been attributed to surface properties such as the porosity, texture and surface roughness of plastic, together with physio-chemical properties such as hydrophobicity and charge (Zhao et al., 2021).

Beach sand provides an ideal opportunity for environmental exposure to skin pathogens such as *Candida* (Brandão et al., 2021); and the transfer of *C. auris* from microplastics washed up onto the beach represents an additional pathway for the delivery of this pathogen to areas of high human contact. In clinical settings, *C. auris* and other pathogenic *Candida* species survive better on moist surfaces compared to dry surfaces (Piedrahita et al., 2017), and in a beach mesocosm, once transferred onto beach sand, *C. auris* persisted better on the wet sand compared to sand allowed to dry out. At beach environments, the constant rewetting of sand at the foreshore would enhance the survival of pathogenic yeasts like *C. auris* at the beach, which is a main area for children playing in the sand (Tomenchok et al., 2020).

Importantly, surviving environmental conditions in the plastisphere, e.g., salinity and desiccation stressors, had no significant impact on the virulence of *C. auris* cells, thus indicating that *C. auris* persisting in natural environmental niches retain their pathogenicity. Although the direct or indirect exposure to pathogenic *Candida* species in the environment can lead to infection in immunocompromised individuals (Steffen et al., 2022), the human health hazard posed by opportunistic pathogenic yeasts in natural environments is still poorly understood (Steffen et al., 2023; Weiskerger and Brandão, 2020). This is partly due to the lack of information about median infectious doses for opportunistic fungal pathogens, which is influenced by the host-specific levels of immunity of individuals (Weiskerger and Brandão, 2020). However, exposure to yeasts, including species of *Candida*, during swimming has previously been identified as a risk factor for fungal infections such as otomycosis (Gharaghani et al., 2015; Ozcan et al., 2003) and contact lens-associated fungal keratitis (Zimmerman et al., 2016).

5. Conclusion

The public health significance of multi-drug resistant strains of *C. auris* is so great that in October 2022, the WHO classified *C. auris* as a fungal pathogen of "critical concern" and placed it at the top of its 'fungal pathogen priority list' compiled to guide research and development. Here, we have shown that *C. auris* can persist, and remain

pathogenic, in simulated environmental matrices contaminated with plastic pollution, which demonstrates the potential for the wide scale disseminated of *C. auris* within the natural environment. Therefore, there is an urgent need for a better understanding of *C. auris* survival and transfer between environmental niches and reservoirs, in order to develop more effective environmental surveillance and management responses to this newly emerged fungal pathogen.

CRediT authorship contribution statement

Ayorinde Akinbobola: Conceptualization, Methodology, Writing – original draft, Investigation, Writing – review & editing. **Ryan Kean:** Resources, Methodology, Writing – review & editing. **Richard S. Quilliam:** Conceptualization, Methodology, Writing – review & editing, Funding acquisition.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:

None.

Data availability

Data will be made available on request.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.marpolbul.2023.115841.

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