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MAJOR ARTICLE

Genomic Dynamics of the Emergent *Candida auris*: Exploring Climate-dependent Trends

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Candidozyma auris (formerly known as Candida auris) (*C auris*) is a multidrug-resistant fungal pathogen that has emerged as a significant threat to global health. Shifts in climatic conditions may be driving its adaptation and pathogenicity. Its increased ability to tolerate higher temperatures has been suggested as the first adaptation led by anthropogenic climate change in a pathogenic organism. In this study, we analyzed 801 whole-genome sequences isolated in clinical settings from the New York-New Jersey region from 2016 to 2024. Using Bayesian hierarchical logistic regression models, we identified previously described antifungal resistance genes, their associated point mutations, heat tolerance genes, and their link with key climatic variables using mixed-effects logistic regression models. Our analysis revealed that the heat tolerance genes *HSP90* and *HSP104* were present in >98% of isolates. Among the antifungal resistance-related genes, several showed significant associations with climatic variables, particularly with precipitation and temperature. Elevated precipitation was consistently linked to increased prevalence in antifungal resistance genes and their associated point mutations, suggesting that elevated moisture levels may promote favorable conditions for fungal growth and biofilm formation. Additionally, the interaction between climatic variables showed a stronger association with the presence of resistance genes, evidencing the multifactorial nature of climate change in shaping pathogen adaptations. These findings emphasize the influence of climatic variables on the resistome of *C auris,* which is crucial for predicting the spread and resistance patterns of *C auris* as climate change continues.

Keywords. antifungal resistance; *Candida auris*; climatic drivers of resistance; multivariate climatic analysis.

Candidozyma auris (formerly known as Candida *auris) (C auris)* is an emergent fungus that causes severe healthcare-associated infections and outbreaks [1, 2]. *C auris* was first described in 2009 [3]. Since its emergence, *C auris* has been isolated from all continents except for Antarctica, with more than 4733 reported infection cases from at least 33 countries between 2009 and 2019 [4, 5]. The US Centers for Disease Control and Prevention classified *C auris* as an urgent public health threat, the first fungal pathogen to be classified as such [6]. This classification arises because of several concerning characteristics, including its high levels of antifungal resistance [7], efficient transmission within healthcare settings, ability to survive in the environment, high rates of mortality, and the challenge to detection via routine diagnostic tests [8, 9]. In healthcare settings, patients colonized with *C auris* are an important source of transmission as they can shed large concentrations of the pathogen to surrounding environmental surfaces and equipment [10]. The reasons behind *C auris* emergence and its original ecological niche outside healthcare settings remain unknown.

The low number of human fungal pathogens compared to other taxonomic groups has been proposed to result from the combination of advanced host-defense mechanisms and mammal high basal temperatures, which create a restriction zone [11]. The optimal growth temperature of *Candida* is 37°C, preferentially colonizing the cooler skin. However, *C auris'* tolerance is higher than other phylogenetically related species [12] and can reach up to 42°C, moving it closer to internal mammalian temperatures and, therefore, facilitating infection [13, 14]. The thermal restriction zone that protects mammals from fungal infections is based on the difference between their basal and environmental temperatures, and therefore, climate change-induced global warming will reduce the magnitude of the gradient between them [14], leading to the positive selection of more thermotolerant fungal lineages [12]. It has been previously suggested that every 1°C gained in the global average temperature will reduce this gradient by about 5% [14]. The differential capacity of *C auris* to survive at higher temperatures than its phylogenetic counterparts has

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been proposed as a recent acquisition, suggesting that this species could be the first example of a pathogenic fungus emerging from human-induced global warming [12].

Previous literature has suggested that the increase in temperature and antifungal resistance in *Candida* are interlinked and may be related to the presence and acquisition of genes and point mutations such as *HSP90* [15] or *HSP104* [16]. These genes play a critical role in both heat tolerance and drug resistance in *C auris*, particularly resistance to azoles [17]. The high level of baseline antifungal resistance present in *C auris* has been suggested as 1 of the leading causes of its emergence because of the widespread use of antifungal drugs in agriculture worldwide, which could have modified and expanded the ecological niche of *C auris* [7, 18]. It has been described that the influence of climate change on agricultural fungicide use leads to cross-resistance between environmental and clinical antifungal treatments, exacerbating the problem of *C auris* resistance [19]. Furthermore, there is evidence that increased heat stress accelerates the appearance of genetic mutations in fungi, promoting the appearance of antifungal resistance by enhancing transposable DNA elements, which contribute to the development of thermotolerance and drug resistance [20]. However, these hypotheses have not yet been quantitatively demonstrated for *C auris*, and thus, the genomic landscape of this fungi and its relationship with environmental variables needs further investigation.

In this study, we used 801 whole-genome clinical isolates of *C auris* from the states of New York and New Jersey to explore the potential relationship between the presence of genes and point mutations associated with heat tolerance and antifungal resistance and the changes in variables related to climate over 8 years. This study highlights the critical need for active environmental surveillance of *C auris* in its natural environment and may serve as the reference for future research aiming to link the presence of antifungal and heat tolerance genes and point mutations in fungi with climatic patterns once a more extensive body of genomic data is available.

METHODS

Data Collection and Curation

Our dataset comprises 801 whole-genome *C auris* sequences isolated in the states of New York (87.4%) and New Jersey (12.6%) from April 2016 to February 2024 (Supplementary Table 1). The raw whole-genome sequences were obtained from the National Center for Biotechnology Information (NCBI) Entrez database [21] in Sequence Read Archive format [22]. The quality assessment of the raw sequences was done using FastQC v0.12.1 [23], followed by their trimming using Trimmomatic [24]. The resulting high-quality reads were then assembled into contigs using SPAdes v3.15.5 [25]. We then evaluated the assembly quality using QUAST v5.2.0 [26].

Table 1. List of All Genes and Their Point Mutations Associated With Antifungal Resistance

Class	Gene	Point Mutation(s)
Azoles	*CDR1*	*V704L*
	ERG11	*K143R, Y132F, F126L, V125A, K177R, N335S, E343D, I466 M, F444L, F126T, K143F, Y132H, G458S*
	MRR1	*N647T, P683S*
	TAC1a	*A640V*
	TAC1b	*A640 V, A651T, A657 V, F214L, F214S, K247E, M653 V, R495G, S611P*
Flucytosines	*ADE17*	*G45V*
	FCY2	*M128fs*
	FUR1	*F211I, Q64**
Echinocandins	*FKS1*	*F635C, S638Y, S639Y, S639F, S639P, D642Y*
	FKS2	…

Gene Selection and Detection

We searched the literature to find the most relevant genes and point mutations conferring antifungal resistance and heat tolerance in *C auris*. Gene sequences were downloaded from the Candida Genome Database [27] (accession numbers available in Supplementary Table 2). To scan for the presence of the selected genes and point mutations (Table 1), we used NCBI-BLASTn, a specific version of NCBI-BLAST focused on nucleotide-nucleotide sequence alignments [28]. The search was conducted considering 100% coverage and identity. This process allowed us to prepare a comprehensive presence-absence matrix for the selected antifungal resistance and heat tolerance genes and point mutations.

The complete pipeline used in this study is shown in Figure 1.

Climatic Analysis Pipeline

We performed a literature search to identify the most relevant climatic variables for fungal spread. Our final section included the following 11 variables: average precipitation, average temperature, average wind speed, extreme maximum temperature for the period, minimum temperature, maximum temperature, number of days with maximum temperature > 70°F (21.1°C), number of days with maximum temperature > 90°F (32.2°C), number of days with greater than or equal to 0.1 inches of precipitation, number of days with greater than or equal to 1.0 inch of precipitation, and runoff, which quantifies water discharged in surface streams.

We then gathered these climatic data from the National Oceanic and Atmospheric Administration National Climatic Data Center [29] using the R package "*httr*" [30]. The variable runoff was downloaded using the R package "*dataRetrieval*" [31]. The collected information underwent processing, data manipulation, and quality control measures using the R packages "*dplyr*" [32] and "*lubridate*" [33].

To maintain a higher number of isolates per unit of time and to include the effect of climatic seasonality on the models, we divided the time periods into seasonal quarters, where

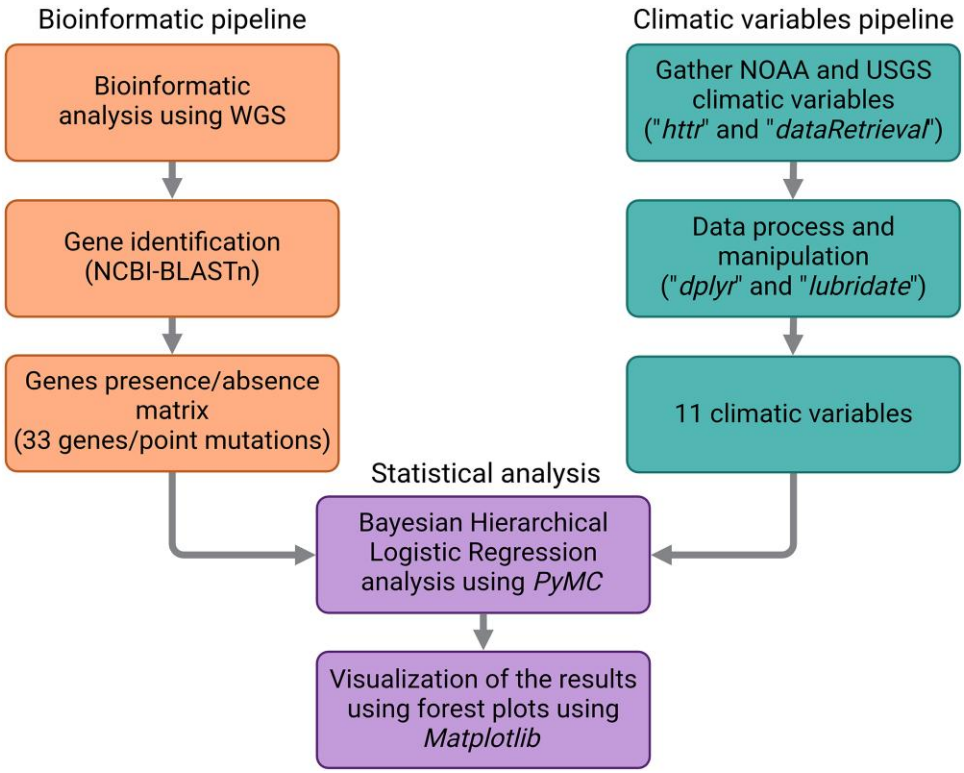


Figure 1. Project workflow illustrating the step-by-step processes involved in both the bioinformatic pipeline for processing genomic data and the climatic variables pipeline for processing environmental data. Abbreviations: NOAA, National Oceanic and Atmospheric Administration; SRA, Sequence Read Archive; USGS, United States Geological Survey; VIF, variance inflation factor; WGS, whole-genome sequencing.

Q1 = December, January, and February, Q2 = March, April, and May, Q3 = June, July, and August, and Q4 = September, October, and November.

Association Between Genes and Climatic Variables

To assess the association between gene presence and climatic variables while accounting for multiple comparisons, we employed a Bayesian hierarchical logistic regression model using PyMC version 5.12 [34]. In this model, each isolate's binary variable (gene presence vs. absence) was treated as a Bernoulli random variable with a logit link. The linear predictor is composed of 3 key components: first, a global intercept representing the baseline log-odds of gene presence; second, fixed effects for the 11 climatic variables, all centered and scaled to unit standard deviation so that reported offs ratios (ORs) correspond to a 1-standard deviation increase, for which we imposed independent Laplace (L1) priors to shrink small coefficients toward zero, thereby effectively performing variable selection and reducing the risk of spurious associations; and third, a random intercept for "quarter year" that captures temporal clustering and unobserved heterogeneity across different quarters, with the random intercept assumed to follow a normal distribution with mean zero and an unknown standard deviation (σ_q). We performed posterior sampling using the No-U-Turn Sampler, running 4 chains with 2000 post-warmup draws per chain (after 1000 tuning iterations) and setting the target acceptance rate to 0.95 to improve convergence. Convergence diagnostics, including Rhat values and effective sample sizes, were evaluated using ArviZ [35], and all key parameters (global intercept, climate coefficients, and σ_q) demonstrated satisfactory convergence (Rhat < 1.01). Posterior summaries comprising means, standard deviations, and 94% highest density intervals (HDI) were computed for each parameter, and forest plots were generated to visually represent the estimated effects of climatic variables using the Matplotlib Python library version 3.5.1 [36]. Prior-versus-posterior overlays for every coefficient whose 94% HDI excluded zero (Supplementary Fig. 2) and trace plots with effective-sample statistics (Supplementary Fig. 3) provide additional convergence and information-content diagnostics.

The code of the pipeline used for these analyses is available at https://github.com/manuelncsu/C_auris-climate_change/tree/main.

RESULTS

Distribution of Antifungal Resistance and Heat Tolerance Genes and Point Mutations

Across all our isolates, we identified varying levels of presence for the 29 antifungal resistance plus relevant point mutations. These results show a widespread prevalence of several key antifungal resistance genes, particularly those associated with azoles and flucytosines (most genes present in >98% of isolates). However, specific resistance mutations, such as *FKS1* (*S639F*) and *ERG11* (*Y132F*) were less frequent (<5%). Regarding the prevalence of heat tolerance genes, notably, *HSP90* and *HSP104*, were found to be highly prevalent, with >98% occurrence in our dataset, and therefore were not suitable to use for further analysis (Supplementary Figure 1).

Interactions Between Climate and Resistance Genes and Point Mutations

Our analysis revealed several significant associations between the presence of specific resistance genes in *Candida auris* and climatic variables (Supplementary Table 3). Our results show that hydrological variables were the dominant drivers. Higher total precipitation (PRCP) raised the odds of detecting *FKS1* (OR = 1.7; 1.4–2.1), its point mutant *D642Y* (1.9; 1.4–2.6), *ADE17* (1.6; 1.3–1.9), and *FUR1* (*F211I*) (1.7; 1.3–2.2). Surface runoff showed parallel positive effects on *FKS1* (1.5; 1.2–1.8), *FKS1* (*S639F*) (2.0; 1.5–2.7), *FLO8* (2.1; 1.4–3.0), and again *FUR1* (*F211I*) (1.6; 1.2–2.1). The frequency of light-rain days (DP01) likewise increased the odds of *FLO8* (1.8; 1.3–2.4). On the other hand, temperature exerted gene-specific effects. Warmer nightly minima (TMIN) reduced *ADE17* prevalence (OR = 0.55; 0.43–0.70), whereas warmer daily maxima (TMAX) elevated the odds of the *CDR1 V704L* variant (1.7; 1.3–2.3); a higher mean temperature (TAVG) increased *FUR1 F211I* (1.5; 1.2–2.0). No other climatic covariate showed a credible association after Laplace shrinkage. These findings are supported by the prior-posterior overlays in Supplementary Figure 2, which show minimal prior influence, and by the trace-mixing diagnostics in Supplementary Figure 3. These findings underscore the multifactorial role of climate in shaping the *C auris* resistome, highlighting the importance of considering climatic contexts in genomic epidemiology studies (Figure 2).

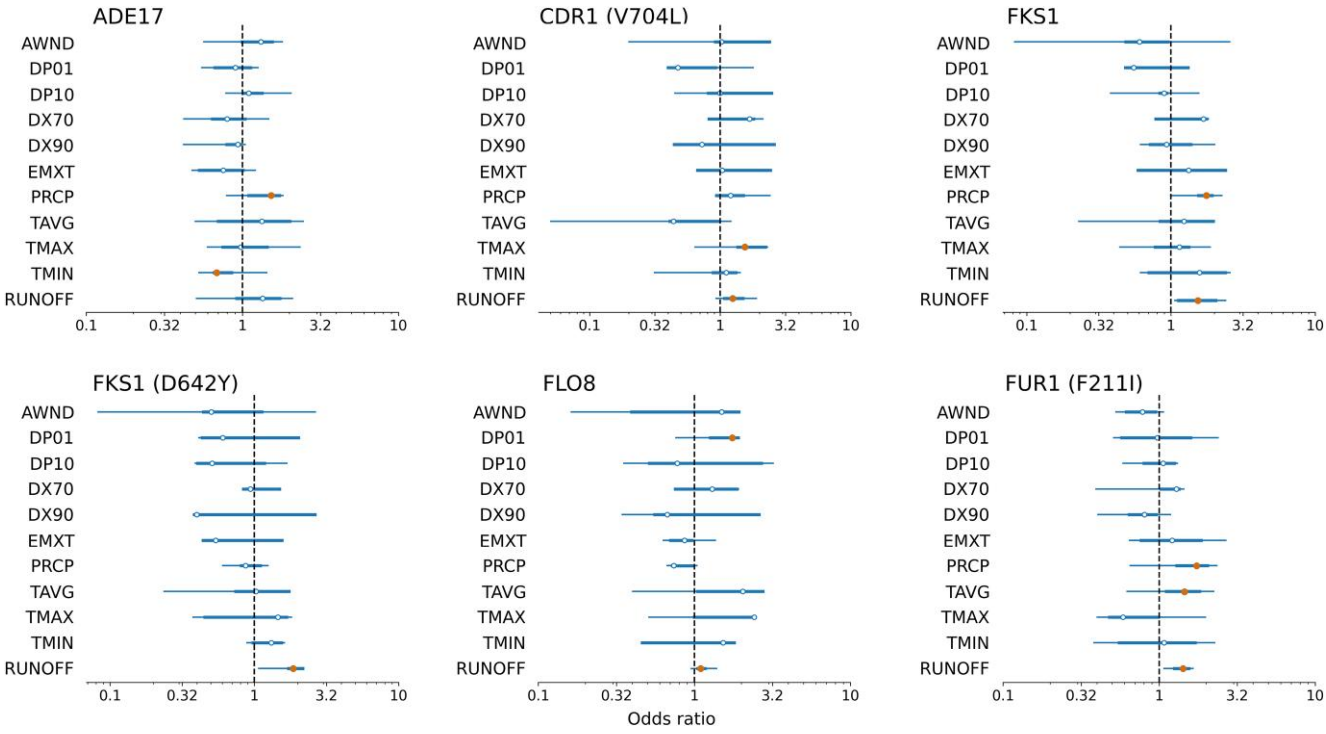


Figure 2. Forest plots of climatic effects on gene presence for genes with the highest associations. These genes were chosen based on their strong evidence of association with climatic factors in our Bayesian hierarchical logistic regression analysis, which incorporated a random intercept for quarter-year and Laplace (L1) priors on the climate coefficients to control for multiple comparisons. Each panel shows posterior odds ratios (OR, blue points ± 94% highest-density interval) for 1 focal gene; the vertical dashed line marks the null value OR = 1. Climate covariates were z-scaled, so ORs correspond to a 1-standard deviation increase. Parameters whose 94% HDI excludes 1 are highlighted in orange. AWND, average wind speed; DP01, number of days with precipitation ≥ 0.1 inch; DP10, number of days with precipitation ≥ 1.0 inch; DX70, number of days with maximum temperature > 70°F (21.1°C); DX90, number of days with maximum temperature > 90°F (32.2°C); Abbreviations: EMXT, extreme maximum temperature for the period; PRCP, average precipitation; TAVG, average temperature; TMAX, maximum temperature; TMIN, minimum temperature; RUNOFF, water discharged in surface streams.

DISCUSSION

Over the past decade, the emergence and spread of *C auris* as an antifungal-resistant pathogen and its elevated thermotolerance have become significant concerns for modern medicine [37]. This study aimed to identify climatic variables associated with the presence of antifungal resistance and heat tolerance genes and point mutations in *C auris* using isolates from the New York and New Jersey areas. By combining genomic data with high-resolution climatic information, this study identified associations between climate and resistance at gene level, supporting previously proposed hypotheses about *C auris* evolutionary adaptation and revealing novel insights into the interconnection between environment and the genetic basis of resistance in this pathogen.

Global warming has previously been proposed as the driver for *C auris* to efficiently overcome the mammalian thermal barrier, leading to its emergence as a human pathogen [37, 38]. Although climate change is a known driver for species adaptations [39], the acquisition of thermotolerance in *C auris* has been suggested to be the first caused by anthropogenic climate change in a pathogenic organism [38, 40, 41], highlighting the importance of further studies on the relationship between climate change and the pathogenicity of emerging *C auris* strains. Previous studies have stated the essential roles in stress responses (i.e., heat tolerance) of the *HSP90* and *HSP104* genes in *C auris* [17, 42]. This was reflected in the high prevalence of these genes among our isolates (>98% of our whole database), which were collected from symptomatically infected patients in clinical settings, supporting their key role in the adaptation of *C auris* to surpass the thermal barrier imposed by homeotherm organisms [12].

Heat tolerance and antifungal resistance in *C auris* have been described to be interlinked due to the function of genes such as *HSP90*, which is associated with *C auris* growth and tolerance to azole antifungals, which block ergosterol biosynthesis [17]. Furthermore, numerous studies have described the association between the emergence of antimicrobial resistance and climate change in fungi [20] and bacteria [43], particularly in relation to global warming, supporting our observed positive associations between the presence of antifungal resistance genes and point mutations in *C auris* with variations in average temperature.

Our findings reinforce that temperature extremes and moisture significantly influence the genetic makeup of *C auris*. Particularly, lower minimum temperatures negatively correlated with genes such as *ADE17*, whereas higher average temperatures increased the odds of detecting genes like *FKS1* and the variant *CDR1_V704L*, indicating selective pressures imposed by temperature fluctuations. We also found a strong positive association between precipitation and the rise of antifungal resistance genes in *C auris,* which can be attributed to moisture fostering fungal proliferation, creating favorable conditions for its growth and survival [44]. Humidity associated variables (i.e., precipitation, runoff) produced the most consistent associations, where in some genes it doubled the odds of harboring resistance determinants, including *ADE17*, *FKS1, FKS1 (D642Y), FKS1 (S639F),* and *FUR1 (F211I*) (Figure 2, Supplementary Table 3). The *CDR1* gene has a critical role in antifungal resistance and is also present in >98% of our isolates. This gene facilitates biofilm formation, which protects the fungus from environmental stressors [45, 46]. Past studies have explored how precipitation and its influence on local humidity contribute to the formation and maintenance of biofilms in natural and clinical settings [44, 47, 48], providing a protective niche where resistant strains of *C auris* can proliferate.

However, climate change is a multifactorial process [49], and therefore, evolutionary adaptations are generally driven by the interconnection of multiple variables. In the case of *C auris* emergence, it has been previously described that the optimal conditions for its survival and propagation also include the combination of humidity with temperature, evidencing an important contribution to its spread inside and outside of healthcare settings [40, 50]. The seasonality found in the presence of antifungal resistance genes and its association with variations in precipitation and temperature suggests that *C auris* may be adapting in response to predictable environmental changes. For that reason, understanding seasonal dynamics is crucial for the prediction of potential outbreaks and the implementation of control and prevention strategies for this fungus based on historical patterns and trends of these variables.

Although our study provides valuable insights, there were still some limitations. Primarily, even though the area combining New York and New Jersey is one of the regions with the highest number of detected cases in the United States [51], this small geographical sector represents a limitation for this type of study. Future studies should include expanding the access to genomic datasets to other regions with contrasting environmental conditions. The second limitation faced by this study is the lack of information from nonclinical environments. The analysis of mostly clinical isolates from symptomatic patients likely poses a bias toward genomes that are suspected to be thermally tolerant based on their isolation source, which would be explained by the generalized presence of genes as *HSP90* and *HSP104*. This potential relationship has been suggested in previous work, where clinical samples present increased *HSP90* expression under heat stress and during infection, supporting survival in febrile and host-imposed stress conditions [52]. Access to isolates from nonclinical sites would be an indicator of the most likely environmental reservoirs of *C auris*, would provide a more comprehensive picture of the genomic diversity of *C auris* strains in a region, and better inform of the differences in gene presence between pathogenic and nonpathogenic strains. In that context, an additional limitation to consider is that without antifungal susceptibility testing, there is no way of knowing whether the

presence of the gene or the necessary mutation will lead to the expression of a resistant phenotype; thus, studies that combine genomic and phenotypic testing are needed. Finally, our analysis time frame (8 years) is constrained by the new emergence of the pathogen. Because of this short period, our results are arguably exploratory, and further analysis over a longer period will be needed to validate our findings and provide more robust estimations of the identified relationships. Additionally, the period studied was influenced by the SARS-CoV-2 pandemic, which particularly affected healthcare systems and increased the number of detected cases of *C auris* worldwide [53].

In conclusion, this study applied an integrative method that allowed us to investigate the potential climate-dependent trends in the temporal and genomic evolution of *C auris*. Our results support previously proposed hypotheses on the relationship between climatic variables and the appearance of antifungal resistance in *C auris*, particularly temperature and precipitation. The associations found between the presence of resistance genes and point mutations and the seasonality of these climatic variables emphasize the adaptation plasticity of this pathogen. This study highlights the need for increased surveillance of *C auris* and provides a starting point for the development of future studies in other geographical areas, including isolates from both hospital and external environments that could better capture the ecological niche and evolutionary trajectory of *C auris* in response to climate change.

Supplementary Data

Supplementary materials are available at *Open Forum Infectious Diseases* online. Consisting of data provided by the authors to benefit the reader, the posted materials are not copyedited and are the sole responsibility of the authors, so questions or comments should be addressed to the corresponding author.

Notes

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Potential Conflicts of Interest. The authors report no conflicts of interest.

Patient Consent Statement. No patient-related data were used in this project. Thus, no ethical committee approval was required.

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