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Candida auris: A Comprehensive Review of an Evolving Pathogen and Strategies for Management

Saud Abdullah Bukhari[1]*, Saleh M. Al-Maaqar [1], Wael A. Alsubhi[3], and Ahmed M. Al-Hejin [1,2,] *

[1]Department of Biological Sciences, Faculty of Science, King Abdulaziz University, 21589, Jeddah, Jeddah, Saudi Arabia.
[2]Microbiology Level 2 Laboratory, King Fahd Medical Research Center, King Abdulaziz University, Jeddah, Saudi Arabia.
[3]Department of Pharmacy Practice, College of Pharmacy, University of Hafr Al-Batin, Saudi Arabia.

***Corresponding authors**: sbukhari0060@stu.kau.edu.sa (S.A.B)|aalhejin@kau.edu.sa (A.M.A.H)

Abstract

This review aims to synthesize the current understanding of the emerging fungal pathogen *Candida auris*, focusing on its epidemiology, mechanisms of multidrug resistance, challenges in diagnosis and infection control, and future directions for management. A comprehensive literature review was conducted, analyzing data from global surveillance reports, clinical studies, and molecular research published up to 2024. The review systematically examines the pathogen's origin, clonal spread, antifungal resistance mechanisms, biofilm formation, and diagnostic limitations. *Candida auris* exhibits high mortality rates (30–60%), rapid nosocomial transmission, and frequent resistance to multiple antifungal classes, including azoles, echinocandins, and amphotericin B. Its persistence on skin and environmental surfaces facilitates outbreaks, while standard phenotypic methods often lead to misidentification. Genomic analysis has revealed four major clades with distinct geographic distributions. *Candida auris* represents a serious and evolving global health threat requiring enhanced surveillance, rapid diagnostics, and strict infection control measures. The development of novel antifungals and decolonization strategies, alongside international collaborative efforts, is urgently needed to curb its spread and improve outcomes in at-risk patient populations.

Keywords: *Candidozyma auris* | *Candida auris* | *Candidemia* | clades | resistance | prevalence.

1. Introduction

Since these eukaryotic pathogens currently infect billions of people worldwide and cause over 1.5 million deaths each year, the prevalence of human fungal infections is increasing rapidly. The high mortality rates from invasive fungal infections are similar to those of common parasitic and bacterial diseases like malaria and TB. Species such as *Aspergillus*, *Cryptococcus*, and *Candida*

are responsible for nearly 90% of all fungal infection-related fatalities [1, 2]. According to the World Health Organization's newly published list of fungal priority infections, these three pathogens are classified as critical priority infections [2, 3]. *Candida* spp. is the primary causative agent of invasive fungal infections. The predominant etiology of invasive fungal infections is *Candida* spp. The associated mortality rate, which has stayed steady for decades, ranges from 46% to 75% [2, 3]. Because drug-resistant infections, especially those caused by non-albicans *Candida* spp., have risen sharply, the US Centers for Disease Control and Prevention has labeled *Candida* spp. as a serious threat to human health [2]. *Candida auris* (*C. auris*) is a new pathogenic yeast causing concern worldwide. An inpatient at a Japanese hospital had a new ascomycetous yeast species, *C. auris*, obtained from the external auditory canal [2, 4]. Since its initial isolation and characterization in 2009, *Candidozyma auris*, commonly known as *C. auris*, has emerged as a novel fungal pathogen responsible for severe infections and fungemia among vulnerable groups, posing a worldwide public health challenge [4-6]. Based on chemotaxonomic research, the 26S rDNA D1/D2 domain and the nuclear ribosomal DNA internal transcribed spacer (ITS) region were analyzed, and the taxonomic description of *C. auris* spp. was proposed in 2009. The research indicated that the species is closely linked to *Candida ruelliae* and *Candida haemulonii* within the *Metschnikowiaceae* group [2,4]. In the same year, *C. auris* was isolated from ear canal samples in South Korea [2,7]. In 2014, five years post-discovery, *C. auris* was identified as the causative agent of bloodstream infection epidemics in South Korea, India, and South Africa [2,8-10]. Within just over a decade of its discovery, *C. auris* has become a primary nosocomial infection worldwide. *C. auris* is not a commensal yeast residing in the gastrointestinal tract and mucosal surfaces, in contrast to other pathogenic *Candida* species. However, it shows distinct skin affinity and a prolonged half-life on human skin [2,11]. Evidently, in healthcare settings, *C. auris* persists on human skin and non-living surfaces, facilitating intra- and inter-hospital clonal transmission that causes significant outbreaks. Its easy transmission through skin-to-skin contact, especially in hospitals, helps spread *C. auris*. Resistance to antifungal drugs is another notable feature of *C. auris*; some isolates are resistant to all three antifungal drug classes [2,11,12]. Recent findings of clinical isolates of *C. auris* that are pan-resistant to the three major antifungal classes Azoles, echinocandins, and amphotericin B (AmB) highlight the urgent and unresolved therapeutic challenges in treating infections linked to *C. auris* [2,13]. There exist distinct clonal strains exhibiting various mechanisms of antifungal resistance. It has been observed that various antifungal susceptibility profiles exist and resistance develops after exposure to antifungals. Control has been impeded by the inability to ascertain, by conventional phenotypic and genetic techniques, the unknown population prevalence, ambiguous environmental niches, and unexplained transmission mechanisms [14]. The increasing use of preventive antifungal medications, such as fluconazole, is believed to have significantly contributed to the rising rates of colonization and infection with non-*albicans Candida* species in recent years. Invasive candidiasis was formerly mostly caused by *Candida albicans (C. albicans)*. Fluconazole can no longer serve as the primary empirical antifungal treatment due to the shift towards non-albicans *Candida* species, which exhibit unique susceptibility profiles, including the rise of multidrug-resistant strains. *C. auris* has the potential to become a dominant opportunistic pathogen in critically ill patients because of its ability to spread rapidly within these groups [14]. The aim of this review is to synthesize the current global knowledge on *C. auris*, an emerging multidrug-

resistant fungal pathogen, by critically examining its global spread, epidemiology, genomics and molecular epidemiology, mechanisms of antifungal resistance, diagnostic challenges, clinical manifestations, and infection control strategies, treatment strategies. This article seeks to provide clinicians, researchers, and public health professionals with an updated, evidence-based resource to enhance recognition, management, and containment of *C. auris*, while also identifying critical gaps in knowledge and proposing prioritized directions for future research and public health action.

1.2. Epidemiology and global spread

Candida species are the main cause of nosocomial fungal infections and the fourth most common cause of diseases acquired in hospitals. Approximately 400,000 bloodstream infections caused by *Candida* species are recorded worldwide each year, with over 40% of those cases ending in fatality [15]. A recent addition to the *Candida Clavispora* group is *C. auris*. Because of its multidrug resistance and rapid global spread, *C. auris* infections have become an international issue over the last 10 years. The Centers for Disease Control and Prevention (CDC) issued a clinical alert to healthcare institutions in 2016 regarding the high mortality associated with emerging international *C. auris* infections. In 2017, the CDC issued an update on the spread of *C. auris* in the United States of America, along with treatment recommendations and disinfection information. Both the clinical and fundamental scientific research domains have given *C. auris* considerable attention since its discovery in 2009 through June 2020. According to the 2020 CDC report (https://www.cdc.gov/) and published research, *C. auris* is found in over 40 countries on six continents. Additionally, it has caused many hospital epidemics [16-19]. Retrospective research conducted in South Korea following the initial reports of *C. auris* infections in clinical settings revealed that the earliest *C. auris* isolates were from 1996. Previously, *C. haemulonii* was wrongly identified as these isolates. Four *C. auris* isolates, obtained in 2009, 2013, 2014, and 2015, were found within a worldwide surveillance culture collection of *Candida* isolates (the SENTRY Antimicrobial Surveillance Program, including 15,271 *Candida* isolates collected from 2004 to 2015). This suggests that *C. auris* is a recently emerged pathogen [15]. Genomic investigations show that *C. auris*' most recent common ancestor appeared about 360 years ago, with some subclades arising as recently as 38 years ago, lending credence to this notion. Numerous novel clonal strains of *C. auris*, together with isolates resistant to amphotericin B and fluconazole, have been identified in two investigations conducted in India [15]. Infections linked to *C. auris* have since been documented in America, Europe, and South Africa. To notify healthcare professionals about *C. auris* as a novel infectious agent, the CDC, the European Centre for Disease Prevention and Control (ECDC), and Public Health England issued several advisories in 2016. A significant study conducted by Lockhart and colleagues in 2017 offered a comprehensive genomic and epidemiological analysis of various genetic populations of *C. auris* strains that appeared almost simultaneously across three continents. Subsequently, *C. auris* isolates have been identified in a minimum of 40 countries worldwide [15]. The South Asia Clade (I), the East Asia Clade (II), the South Africa Clade (III), and the South America Clade (IV) are the four main distinct genetic clades of *C. auris*, which are based on genetic and genomic data as well as the locations of the earliest isolates. Sequencing data typically reveals less than 70 single-nucleotide polymorphisms (SNPs) within each haplogroup. A potentially novel *C.*

auris clade has recently been identified in Iran, characterized by more than 200,000 SNPs that differentiate it from earlier clades [15].

Recent and almost coincidental finding of genetically different clades of *C. auris* on various continents indicates a worldwide selective pressure and not a single point of origin and subsequent spread [15,20]. One of the main hypotheses is related to the role of ecological and anthropogenic elements, especially climate change and the use of antifungal agents on a large scale in the agricultural sector [21]. *C. auris* exhibits a notable thermotolerance, allowing it to grow at temperatures reaching up to 42°C, which is atypical for most *Candida* species [15, 22]. This adaptation can be important evolutionary process, enabling it to cross the mammalian thermal barrier that generally limits majority of environmental fungi. At the same time, the presence of azole fungicides in the environment (e.g., in soil and water) might have selected the azole-resistance mechanisms currently common in clinical isolates, predisposing the pathogen to success in health care environments where antifungals are extensively used [2,21]. Nevertheless, the exact environmental niches (e.g., wetlands, marine, or plant surface) which form the natural reservoir of *C. auris* are not well understood yet, which is a major gap in the ecology [14]. The finding of the phylogenetically divergent Clade V in Iran, that has more than 200,000 SNPs perceivable separation with other clades, is a powerful assail of the model of numerous autonomous emergences out of distinct environmental forfeits and not a recollection increase of a solitary ancestor [15,23]. To conclude, the emergence of *C. auris* is probably a product of a set of global events: a warmer climate permitting thermal selection, agricultural practices selected to favor antifungal resistance, and the modern medical population that has resulted in a vulnerable target population. An intensive, one-health strategy in determining its environmental reservoirs is necessary not only to the understanding of the genesis of this pathogen, but also to the anticipation and prevention of the rise of new fungal hazards.

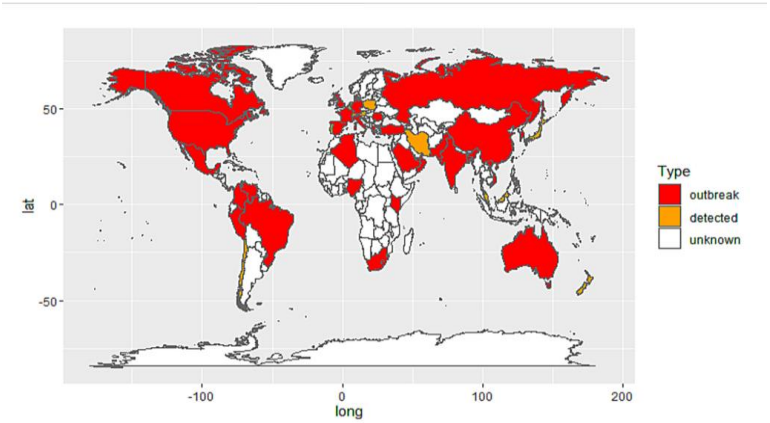

Figure 1. Global distribution of *C. auris* worldwide by Zhang W et al (24).

C. auris has now been isolated from patients in the United States, Canada, Europe (including the United Kingdom, Norway, Germany, and Spain), South Korea, India, Pakistan, Kuwait, Oman, South Africa, Colombia, Venezuela, and the United States [14]. In areas where *Candida* infection has long been recognized, *C. auris* may account for a sizable percentage of cases. *C. auris* was isolated in 19 out of 27 critical care units (ICUs), accounting for 5.2% of cases, according to prospective multi-center research conducted in India that reviewed cases of

candidemia obtained from ICUs. Prevalence at public hospitals was 8.2%, while that in private hospitals was 3.2% [14]. Two peculiar aspects of its epidemiology that distinguish it from other clinical yeasts are its remarkable ability to propagate clones and its quick transformation from *an* obscure species into a significant disease. The transformation is still not fully understood. It was demonstrated to have just recently emerged as a clinical pathogen. Retrospective investigations of yeast stock collections have refuted the idea that *C. auris* was already extensively distributed before its later designation as a separate species [20]. More than 4,000 isolates were found from blood and other materials from many nations across all inhabited continents within ten years of its identification as a new bloodstream pathogen [25]. Nosocomial outbreaks, which are being reported more frequently and appear to involve an increasing number of patients, have replaced the sporadic invasive infections of the early years, causing substantial changes in the epidemiology of invasive *C. auris* infections. According to studies, *C. auris* spreads quickly among vulnerable patients once it is brought into a medical facility [25].

One of *C. auris*'s many distinctive traits is its ability to persist for several months despite the application of conventional disinfectants. This is probably because the bacteria form biofilms on plastic surfaces, in hospitals, and on medical equipment. The therapy of *C. auris* infections is further complicated by varied sensitivity to various azoles, amphotericin B, and echinocandins, as well as very high rates of fluconazole resistance. In several investigations, crude death rates among individuals infected with *C. auris* have ranged from 0% to 72% [25]. In hospitalized patients, the axilla, groin, nares, respiratory tract, and urinary tract are often colonized with *C. auris.* Environmental assessment of the patient's vicinity, encompassing their attire and apparatus, along with the surfaces inside their room, has identified *C. auris* isolates exhibiting comparable fingerprinting patterns, indicating that infected persons may disseminate *C. auris* into the environment. The increased isolation frequency of *C. auris* from the axilla of colonized patients compared to other body locations is consistent with the fact that *C. auris* has also been demonstrated to survive on reusable skin-surface axillary temperature sensors [25]. According to research, skilled nursing facilities that care for patients who are reliant on ventilators had a tenfold greater rate of *C. auris* colonization than nursing facilities that do not provide ventilator support. The same risk factors that apply to other species of *Candida* also apply to invasive *C. auris* infections. Two significant risk factors for the development of invasive infections caused by *C. auris* are the use of mechanical ventilation and the implantation of invasive devices. Prior research has demonstrated that approximately 10% of infected individuals develop invasive infections due to *C. auris* colonization [25]. According to two recent investigations, total parenteral nutrition, sepsis, prolonged arterial or central venous catheterization, severe chronic renal disease, past antibiotic usage, prior surgery, extended intensive care unit stay, and multifocal colonization are additional prevalent risk factors for the development of candidemia in individuals colonized with *C. auris*. Additionally, *C. auris* can produce aggregative phenotypes and "dry" biofilms that are difficult to remove. Hospital illnesses spread more easily via direct or indirect contact due to these features [25]. Fungal infections in humans are becoming increasingly common, affecting billions of people worldwide. These illnesses kill more than 1.5 million individuals each year. On October 25, 2022, the World Health Organization (WHO) published a list identifying 19 priority fungal infections that pose significant threats to public health. The World Health Organization has classified priority fungal

pathogens into three categories: critical, high, and medium. Among fungal diseases, *Candida* species are the main culprits behind invasive fungal infections (IFIs) [26]. The CDC received 127 confirmed and 27 suspected cases from eleven states in September 2017. New York and New Jersey had the most instances, with 86 and 26 confirmed (https://www.cdc.gov/fungal/diseases/candidiasis/tracking-c-auris.html). Increased monitoring efforts, geographic interconnection of health care facilities, and/or population density may all contribute to the rise of isolates from the New Jersey and New York regions [22]. New Jersey isolates were comparable to South Asian isolates but different from those found in New York, while Illinois samples were grouped with South American isolates. After analysis, the first isolates from New York were found to be similar to those from South Asia. Epidemiologic data indicate that a significant proportion of these illnesses was acquired in the US. Furthermore, in the United Kingdom, three of the four established lineages have been identified. The hypothesis of several separate introductions of *C. auris* into the US and UK, followed by local dispersion, is supported by the finding that distinct clonal lineages are now dispersed over great geographic distances [22]. Geographical regions have seen substantial variations in mortality rates. According to reports from the US, Asia, and the Far East, people with invasive diseases have reported fatality rates of more than 50%. In comparison, 72% of people in Venezuela survived for 30 days after contracting candidemia. In Colombia, a delayed diagnosis of *C. auris* was linked to a 30-day death rate of 35.2% [14]. The most notable spike occurred between 2020 and 2021, when the number of cases recorded nearly tripled to 4401. The CDC reports 2377 documented clinical cases and 5754 screens between January 2022 and December 2022. Clinical concerns about the future are growing as research indicates annual multiplicative growth in positive culture [23, 27].

1.3. Clinical manifestations and pathogenesis

C. auris is typically acquired in hospitals and has been isolated from several infection sites across the body. Samples have been obtained from multiple sources, such as urine, bile, blood, wounds, nasal passages, axilla, skin, and rectum of individuals showing signs of illness. Although *C. auris* is thought to colonize the skin mainly, it has occasionally been isolated from the gut, mouth, and esophageal mucosa of infected people. This is not the same as *C.* albicans, which lives in the genitourinary and gastrointestinal systems of healthy people. Clinical symptoms and *in vivo* investigations collectively suggest that *C. auris* is unable to colonize anaerobic habitats, such as the gut, consistent with the rarity of isolating the bacteria from the gut. The salivary antimicrobial peptide histatin 5 was shown to have a strong antifungal impact on *C. auris* in connection to the oral mucosa during a recent study. The fact that *C. auris* is seldom isolated from the oral mucosa may be due to the fact that this peptide may hinder the bacteria from colonizing the oral mucosa. *C. auris* is most frequently linked to bloodstream infections in clinical settings. According to one study, *C. auris* was responsible for about 5% of candidemia cases in Indian intensive care units (ICUs). Critically sick patients in intensive care units are more likely to get invasive infections brought on by *C. auris*. Invasive *C. auris* infections are linked to high worldwide death rates of 30% to 60%, just as other invasive *Candida* infections [15]. Candidemia, including instances linked to CVC usage, pericarditis, and respiratory and urinary tract infections, has all been strongly linked to

invasive *C. auris* infections. Most often, critically sick patients, those in intensive care units and undergoing invasive procedures, are the ones who get an invasive infection with *C. auris*. Hematological malignancies and other illnesses that cause immunosuppression are among the major underlying medical disorders that these individuals typically have—an instance of donor-derived *C. auris* infection after lung transplantation was described in one publication. Initially misdiagnosed by both biochemical and molecular tests, yeast was detected in bronchoalveolar lavage samples before and after implantation [14]. Infections with *C. auris* have the same risk factors as infections with other *Candida* species. Considering that numerous *Candida* species are opportunistic pathogens predominantly linked to severely ailing and immunocompromised individuals, this observation is not surprising. Elderly age, diabetes mellitus, recent surgery, the presence of an indwelling medical device (such as a central venous catheter), immunosuppression, hemodialysis usage, neutropenia, chronic renal illness, and the use of broad-spectrum antibiotics and/or antifungal medications are risk factors for *C. auris* infections [15]. Contact with individuals or their surroundings who are known to have *C. auris* is one risk factor for colonization. Within 48 hours of being admitted to intensive care units, invasive infections have been acquired, and the contact time for acquiring *C. auris* from a colonized patient or environment is as little as 4 hours. In cases where a patient colonized with *C. auris* shows progression, it is advisable to consider empirical antifungal therapy (14). Our understanding of the pathogenic effects of *C. auris* in the invertebrate *Galleria mellonella* was aided by an *in vivo* model that contrasted the pathogenic effects of UK isolates of the fungus with those of other pathogenic species. The findings revealed that isolates of *C. auris* may demonstrate different behaviors, with certain strains forming aggregates while others do not. In comparison to *C. albicans*, isolates that do not form aggregates exhibited greater pathogenicity in larvae than those that do form aggregates. There was no correlation found between this with the creation of hyphae or pseudohyphae, which are formed by *C. auris* only in a very basic way and only very seldom [14]. According to reports, *C. auris* can flourish at temperatures higher than 40 ℃, in contrast to its closely related species global warming, in particular, may have played a role in the emergence of *C. auris* as a human pathogen and in its ability to thrive at high temperatures, according to a recent study that compared the species' temperature tolerance to that of other *Candida* species. Unlike other *Candida* spp, *C. auris* can withstand high salt concentrations (>10% NaCl, wt/vol). According to the findings of two separate experiments, *C. auris* reacts to high salt concentrations by producing morphologies that are similar to pseudohyphae. This morphological alteration is indicative of an adaptive response to stressful situations. Long-term persistence and survival of *C. auris* on biotic and abiotic surfaces may be attributed to traits like thermotolerance and osmotolerance. Like *C. albicans*, *C. auris* produces numerous recognized virulence factors, such as lipases and saps, to degrade and infiltrate host tissues, according to recent findings. Because *C. auris*, *C. glabrata*, and *C. haemulonii* does not produce hyphae or pseudohyphae in the mammalian host—which are essential for tissue penetration during infections—they are less virulent than *C. albicans* [15]. On human skin and environmental surfaces, *C. auris* may, in fact, withstand exposure to several widely used disinfectants for several weeks. One explanation for the often noted intrahospital transmission of *C. auris* in medical settings might be persistence on surfaces. An instance of *C. auris* was connected to the use of reusable axillary temperature sensors at the neurology intensive care

unit of Oxford University Hospitals in the United Kingdom. One of the *C. auris*'s distinguishing characteristics compared with most other human fungal infections is its ability to survive under adverse environmental conditions. The cultivation of *C. auris* on CHROMagar has demonstrated a progression of morphological changes, transitioning from pink to white and ultimately to dark purple. Compared to the white-opaque switch frequencies seen in *C. albicans*, these different *C. auris* phenotypes switch at greater frequency. The inheritance of the phenotypic shift observed in *C. auris*, characterized by variations in colony color from pink to white and dark purple, remains uncertain. There were no cellular photos of the cell morphologies inside the various colored colonies in the first report of this phenotypic transition in *C. auris*. Based on the documented colony morphologies, the phenotypic switch seen in *C. auris* may be similar to the core phenotypic switch system observed in *C. glabrata* when grown on nutritious agar medium containing copper (II) sulfate or phloxine B [15]. Serum, N-acetyglucosamine (GlcNAc), and elevated CO_2 are among the environmental variables that strongly stimulate filamentous development in *C. albicans*. These variables were shown not to affect filamentous development in *C. auris*. Two traits that set *C. auris* apart are thermotolerance and osmotolerance. *C. auris* was cultivated in one recent investigation using a medium supplemented with 10% NaCl and yeast extract, peptone, and dextrose (YPD). Under these conditions, long, pseudohyphal-like cells formed at 37 and 42 degrees Celsius. Heat shock protein 90 (Hsp90) is a crucial molecular chaperone that controls temperature-dependent filamentation in *C.* albicans. *C. auris* cells treated with a Hsp90 inhibitor produced cells that resembled pseudohyphals, according to a recent study. Filamentous growth was produced by similar Hsp90 inhibition in *C. albicans*, indicating that *C. albicans* and *C. auris* at least partially share some filamentation regulatory mechanisms. These investigations also suggest that, under certain environmental conditions, some *C. auris* isolates may undergo filamentation [15]. In natural biological niches, bacteria prefer to form biofilms. A biofilm that develops on human tissue (such as a mucosal layer) or on an implanted medical device (such as a central venous catheter) might act as an infection source in a clinical environment and spread to other body areas. Research indicates that *C. auris* is capable of forming biofilms on surfaces; however, these biofilms are not as resilient as those generated by *C. albicans*. Comparing *C. auris* biofilm cells to their free-floating (planktonic) counterparts, it has been demonstrated that both have high levels of resistance to antifungal drugs. Different *C. auris* isolates and clades have different capacities for biofilm production. Although both aggregated and non-aggregated *C. auris* cells may produce biofilms, it has been shown that the latter generate more robust biofilms [15]. Genes encoding putative adhesins, efflux pumps, and virulence factors were shown to be increased during *C. auris* biofilm formation, according to time-course RNA-sequencing experiments. *C. auris* biofilms contribute to its virulence, antifungal resistance, and survival in the environment and presumably in the host, although their activities are not fully known. Therefore, developing treatment plans to address *C. auris* biofilms in patients and the environment is a major area of interest for future research [15].

1.3.1. Biofilm formation

Its resistance to treatment and capacity to survive in medical environments are largely due to the intricate, multi-layered structure of the biofilm that *C. auris* forms. Gaining insight

into the formation, structure, and makeup of *C. auris* biofilms is vital for devising practical approaches to tackle these infections [28]. The first phase of biofilm development involves *C. auris* cells attaching to surfaces, including both living and non-living materials [28, 29]. Certain adhesins and surface proteins act as mediators of this attachment, and genes linked to cell walls anchored by glycosylphosphatidylinositol (GPI), including IFF4, CSA1, PGA26, and PGA52, exhibit elevated expression at this stage [28, 30]. The discovery that a null mutant of iff4Δ showed decreased virulence and adherence during the early stages of biofilm generation highlights the importance of these genes [28, 31]. Cells start to proliferate and group together after the first adhesion. The clusters of cells function as fundamental components of the biofilm, playing a crucial role in its mass and structural integrity. In the proliferation phase, a range of genes is activated, such as RDC3, SNQ2, CDR1, and YHD3. The efflux pumps that these genes encode safeguard the biofilm throughout its formation. The structural growth of the biofilm is further aided by the upregulation of genes that encode extracellular matrix components, such as KRE6 and EXG, which encodes glucan-1,3-beta-glucosidase [28, 30].

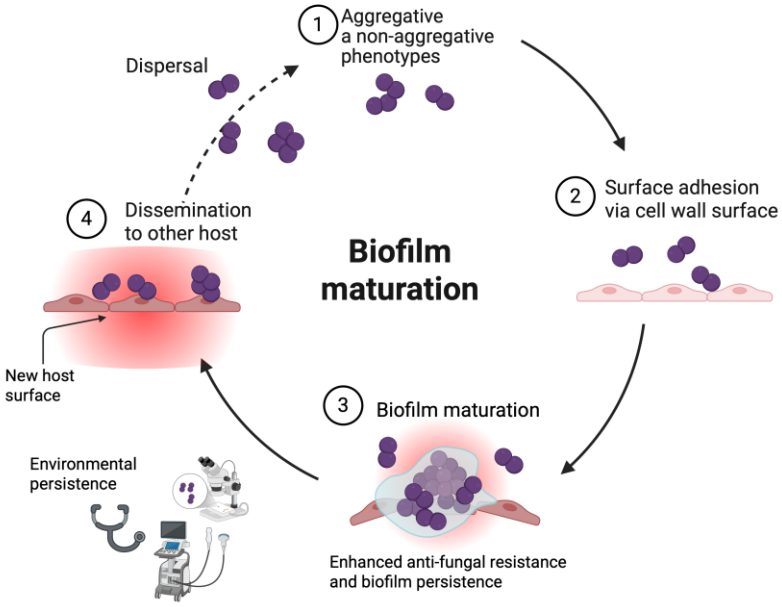

Figure 2. Biofilm formation mechanism in *C. auris.*

These clusters eventually grow into a complete biofilm with a more complex and well-organized structure. When *Candida* biofilms reach maturity, they usually show many fungal cell layers with gaps and channels that allow waste and nutrients to be exchanged [28, 32]. Alpha-glucans and mannans are among the several polysaccharides found in the extracellular matrix of the mature *C. auris* biofilm [28, 33]. A gel-like structure is formed by the polysaccharides, which not only serves as a barrier to antimicrobial agents but also confers structural stability to the biofilm. Cell-cell adhesion and biofilm durability are improved by the presence of many proteins in the *C. auris* biofilm matrix. Additionally, host tissues and immune system components may interact with these proteins. In contrast to planktonic cells, Paudyal and

Vediyappan's research revealed many proteins, such as Spe3p, Tdh3p, Sod2p, Ywp1p, and Mdh1p, that are overexpressed in *C. auris* biofilms. This underscores the biofilm's distinct composition and resistant features [28, 34]. Cell proliferation and matrix production continue to occur as the biofilm grows and spreads. Because of its increased complexity, the biofilm may stay on surfaces for longer. In order to create new biofilms and keep the infection cycle going, cells inside the biofilm may also separate and adhere to other surfaces [28, 35].

1.3.2. Diagnosis:

Conventional testing techniques in the microbiology laboratory often lead to misidentification of *C. auris*. Therefore, a comprehensive diagnostic approach is required [36-38]. This approach should incorporate multiple diagnostic modalities, including complementary tests such as C-reactive protein measurement, which is particularly important for monitoring disease progression and evaluating treatment efficacy [37, 38]. Owing to their accessibility and lower cost compared with blood tests, the neutrophil-to-lymphocyte ratio and platelet-to-lymphocyte ratio have been widely investigated as indicators of systemic inflammation in recent studies. While the neutrophil-to-lymphocyte ratio is recognized as a valid inflammatory marker in solid tumors, bronchiectasis, sleep apnea, and various other diseases, the platelets-to-lymphocyte ratio also demonstrates significant clinical relevance [38]. The Centers for Disease Control and Prevention (CDC) recommends species-level identification of *Candida* isolated from non-sterile sites under specific circumstances. These circumstances include the presence of clinical indications during patient care, a reported case of *C. auris* infection or colonization within an institution or unit that necessitates additional patient screening, or a patient's overnight stay in a healthcare facility outside the United States within the previous year. This guidance is supported by evidence indicating that some patients with *C. auris* may remain colonized for over one year [38, 39]. The prevalence and geographic distribution of *C. auris* infection, which primarily affects low- and middle-income countries, may be underestimated. Two primary factors contribute to this data scarcity: the absence of a global identification strategy and the limited accuracy of current traditional diagnostic tools [38, 40]. Clinical specimen recovery times are shortened and sensitivity and specificity are increased when selective enrichment broth media are optimized for the growth characteristics of *C. auris*. Using a 10% salt Sabouraud Dulcitol broth medium supplemented with gentamicin and chloramphenicol, stirring the inoculum, and incubation at 37 to 40°C may all increase isolation rates. With this technique, the special ability of *C. auris* to grow in salty conditions (10% w/v) and at high temperatures is exploited [38, 41]. While *C. haemulonii* and *C. duobushaemulonii* need glucose to grow in the same circumstances, *C. auris* can grow in similar conditions when dulcitol or mannitol is supplied as a carbon source (Table 1) [38, 42, 43].

On Sabouraud dextrose agar, *C. auris* strains usually produce white to cream-colored colonies; on CHROMagar, they look pink to beige. N-acetylglucosamine, succinate, and gluconate may be assimilated by the bacteria, which develop robustly at 37°C and 42°C. Conversely, *C. duobushaemulonii* and *C. haemulonii* cannot digest these substrates and do not develop at 42°C [38, 46]. For the purpose of detecting *C. auris*, new chromogenic medium compositions have been created [37, 38]. *C. auris* has thermotolerance, which enables it to grow

at temperatures over 37°C and stay viable up to 42°C, in contrast to other fungi, which cannot live at temperatures lower than human body temperature [38, 47]. Significant resistance to a variety of osmotic stressors and high salinity conditions (>10% NaCl, w/v) is shown by this organism [21, 38]. Single cells, pairs, or clusters of the budding yeast *C. auris* are often encountered. These cells have ovoid to elongated, ellipsoidal forms and range in size from 2.5 to 5.0 μm [38, 48]. Filamentation in this organism is a rare event that constitutes a crucial phase in the invasion of host tissues by fungi. Similar to other *Candida* species, it generates both pseudohyphae and true hyphae [38, 40]. Basic pseudohyphae are formed by *C. auris* in high-salinity environments and during biofilm formation [37, 38]. Both hyphal cell wall protein (HWP1) and candidalysin (ECE1), which are necessary for full hyphal growth, are lacking in *C. auris* [38, 49]. At lower temperatures (20°C and 25°C), *C. auris* forms filaments, whereas *C. albicans* forms hyphae at higher temperatures [38, 50].

Table 1. Growth characteristics of *C. auris* and similar *Candida* spp [38, 39, 44, 45].

Characteristic	*C. auris*	*C. haemulonii*	*C. duobushaemulonii*	*C. pseudohaemulonii*
Growth on Sabouraud with dextrose	+	−	−	−
Growth on Sabouraud with dulcitol	+	−	−	−
Growth on Sabouraud with mannitol	+	−	−	−
Growth in 60% glucose	−	−	+	ND
Growth in vitamin-free medium	+	−	−	ND
Growth at 37 ◦C	+	−	+	+
Growth at 40 ◦C	+	−	−	−
Assimilation of L-Sorbose	−	−	+	+
Assimilation of Arbutin	ND	−	+	ND

Table 2. Aspects of different culture media of *C. auris* and other frequently identified *Candida* species [38, 43].

Culture Medium	*C. auris*	*C.albicans*	*C. parapsilosis*	*C. glabrata*
Sabouraud dextrose agar	Medium White to cream			
BrillianceTM Candida Agar	Medium Beige to pink	Green	Beige/yellow/brown	Beige/yellow/brown
CHROMagarTM Candida	Medium Pale pink	Green	White, pale pink, or light lavender	Dark pink to purple

Conventional phenotypic techniques for yeast identification, including the VITEK 2 YST, API 20C, BD Phoenix yeast identification system, and MicroScan, may misidentify *C. auris* as several distinct organisms, such as *C. haemulonii, Rhodotorula glutinis, C. sake, C. intermedia, Saccharomyces kluyveri, C. parapsilosis, C. duobushaemulonii, C. famata, C. catenulate, C. guilliermondii, C. lusitaniae, and C. catenulate* [38, 51]. In comparison to previous technologies, matrix-assisted laser desorption ionization-time-of-flight mass spectrometry (MALDI-TOF MS)

is able to identify *C. auris* with a higher degree of precision, and it also makes it easier to conduct additional epidemiological analysis of strains after identification [37, 38]. Several commercial systems have been developed for the identification of *C. auris* nucleic acids, demonstrating sensitivities of 89% to 100% and specificities of 85% to 100% [38, 52]. Certain molecular assays exhibit diagnostic accuracy comparable to that of the reference method, culture combined with MALDI-TOF identification, supporting their utility for rapid surveillance and diagnosis. For instance, the DiaSorin Molecular Simplexa® Detection Kit provides results within two hours of swab collection, thereby enhancing clinical decision-making [38, 53].

1.4. Antifungal resistance and treatment options

In light of the fact that these organisms have the ability to host or acquire multidrug resistance, the appearance of *C. auris* is certainly cause for concern [22, 38]. Currently authorized antifungal medications fall into four broad classes: azoles, echinocandins, polyenes, and the less common flucytosine. Alterations in cell wall biosynthesis enzymes, mutations in the ergosterol biosynthesis pathway, and alterations in membrane transporters are the leading known causes of antifungal drug resistance. Additionally, reports of pan-resistant and echinocandin-resistant *C. auris* isolates spreading in the US indicate that *C. auris*'s high transmissibility may drive the spread of drug-resistant clones in healthcare environments [2]. Its inherent resistance to one or more kinds of antifungal medications that are already on the market is a significant factor in the fact that *C. auris* is regarded as a "superbug" and is posing a growing threat to human health. The majority of *C. auris* isolates show fluconazole resistance, based on conservative antifungal medication breakpoints for *C. albicans* and other species. Compared to a subset of *C. auris* isolates, amphotericin B and echinocandin drugs have lower minimum inhibitory concentrations (MICs), and certain strains of *C. auris* are resistant to all presently available antifungal medications [15]. The *C. auris* isolates had a wide range of MICs for other antifungal classes, but strikingly comparable fluconazole resistance, as assessed by the European Committee on Antimicrobial Susceptibility Testing (EUCAST) and Clinical and Laboratory Standards Institute (CLSI) methods. Common resistance to one or more antifungal drug classes is a remarkable characteristic of the closely related species of *C. auris*, *C. haemulonii*, and *C. lusitaniae*. Though *C. haemulonii* and *C. lusitaniae* are seldom isolated as infectious organisms, our result suggests that the antifungal resistance traits of *C. auris*, *C. haemulonii*, and *C. lusitaniae* are caused by same genetic mechanisms [15]. Ergosterol, which is the main component of fungal membranes, is the target of polyenes (like amphotericin B) and azoles (like fluconazole). By targeting the enzyme lanosterol demethylase, which is dependent on fungal cytochrome P450 and necessary for ergosterol synthesis, fluconazole, the first-line antifungal medication in clinical use, prevents cellular ergosterol synthesis. ERG11 encodes lanosterol demethylase in the *Candida* species. It is interesting to note that fluconazole-resistant *C. auris* strains belonging to several genetic clades have been identified with three hot-spot mutations in Erg11 (Y132F, K143R, and F126L or VF125AL) [15]. While fluconazole and amphotericin B-resistant *C. auris* isolates are often found, echinocandin-resistant isolates (such as caspofungin) are comparatively uncommon. FKS1 encodes 1,3-beta-D-glucan synthase's catalytic component, which is necessary for the synthesis and maintenance of cell walls in *Candida* species. While isolates of *C. auris* harboring a wild-type Fks1 were responsive to

caspofungin at human therapeutic dosages, isolates with an S639F mutation in Fks1 were resistant to the drug [15]. Currently, susceptibility breakpoints specific to *C. auris* have not been established. Consequently, characterization relies on expert interpretation and on breakpoints established for related *Candida* species (Table 3). Currently, it is uncertain if microbiologic breakpoints and clinical results are related [38, 51].

C. auris presents a formidable antifungal resistance profile, frequently exhibiting resistance to multiple drug classes, with pan-resistant isolates increasingly reported [2,13]. Azole resistance is commonly mediated by mutations in the *ERG11* gene (e.g., Y132F, K143R) and efflux pump overexpression [2,13,44], while echinocandin resistance is strongly linked to hotspot mutations in *FKS1*, such as S639F [2]. Amphotericin B resistance, though less common, can arise from alterations in the ergosterol biosynthesis pathway and membrane composition [2, 54]. Current first-line therapy relies on echinocandins, but this is challenged by rising resistance and the absence of species-specific clinical breakpoints [14, 38]. Emerging therapies such as ibrexafungerp (a triterpenoid) and rezafungin (a long-acting echinocandin) show promising activity against resistant strains *in vitro* and in animal models [23]. Effective management therefore hinges on mandatory antifungal susceptibility testing, robust stewardship programs, and the integration of novel agents into the therapeutic arsenal to address this evolving threat.

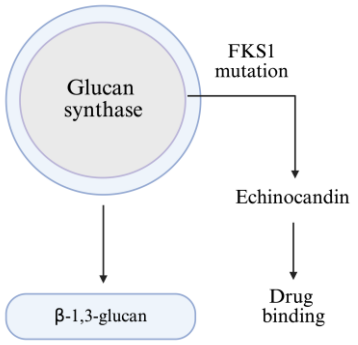

Figure 3. Echinocandin resistance mechanism in *C. auris*

Table 3. Resistance breakpoints as in (38, 51).

Antifungal	Proposed resistance breakpoint (μg/mL)
Fluconazole	≥32
Amphotericin B	≥2
Anidulafungin	≥4
Caspofungin	≥2
Micafungin	≥4

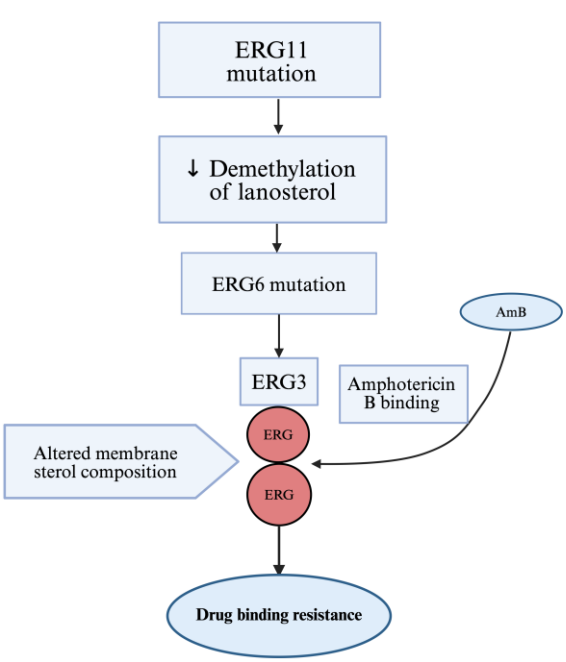

Figure 4. Mechanism of polyene resistance.

The fact that genetically similar isolates harbor distinct resistance gene alleles suggests that *C. auris* resistance is most likely an acquired rather than an innate trait. Clade-specific mutations linked to azole resistance have emerged only recently, within the previous 37 years, according to population-based research [2]. Aneuploidy-induced genetic diversity, chromosomal rearrangements, mutations in the target enzyme, overexpression of the drug target, and reduced drug uptake/efflux are the usual causes of azole resistance in *C. auris*. Point mutations in hot-spot regions of the target gene ERG11, which are known to confer resistance in other *Candida* species, are a primary mechanism of fluconazole resistance. Erg11Y132F and Erg11K143R are the most frequent alterations in Clades I and IV, although Erg11F126L is often present in isolates from Clade III. Interestingly, strains from Clade II frequently lack specific mutations in ERG11. The high azole resistance in *C. auris* is primarily due to overexpression of ERG11, which can occur through aneuploidy or the duplication of tiny chromosomal sections, as in other *Candida* species [2].

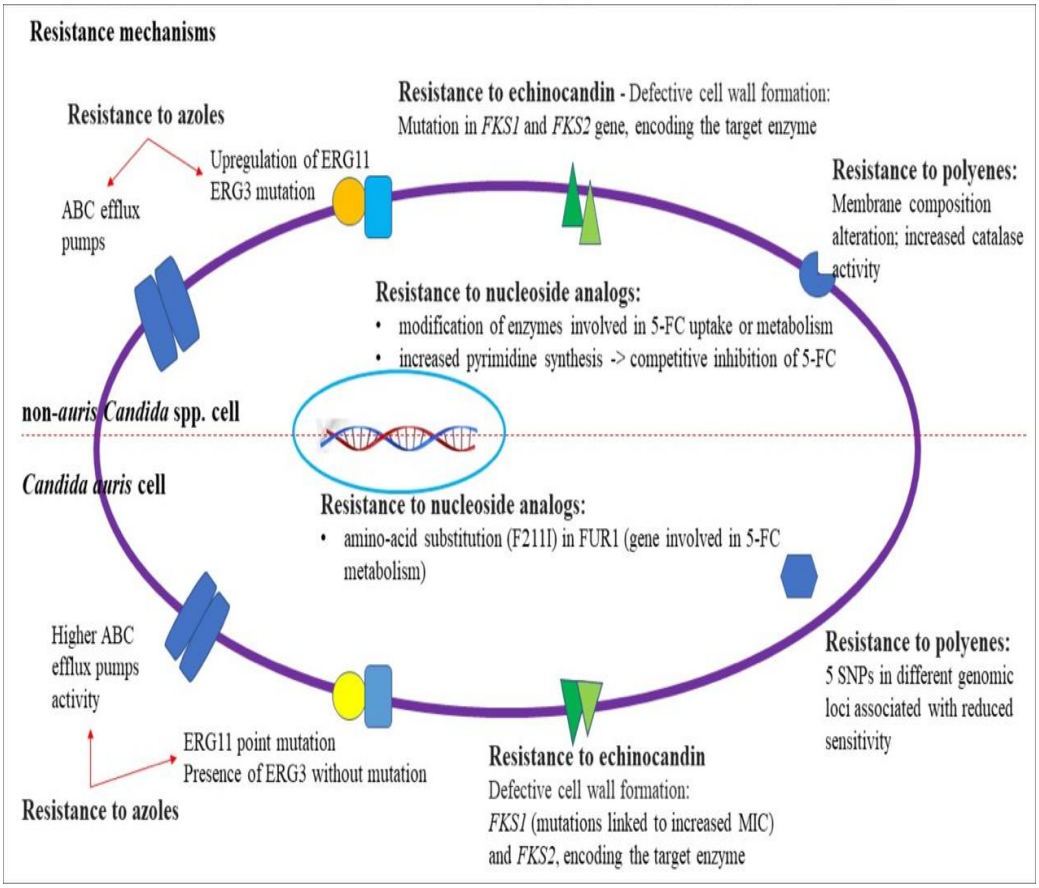

Figure 5. *Candida* spp. and *C. auris* resistance mechanisms [38, 54, 55].

ERG11 and other isolates with segmental duplication of chromosome 1 show minimum inhibitory concentrations of fluconazole that are noticeably greater than those of isolates with only one copy. Chromosome V aneuploidy, which includes the genes TAC1B, NCP1, ERG9, and ERG13, also rapidly confers fluconazole resistance in *C. auris*. High levels of fluconazole resistance are also associated with transcriptional overexpression of efflux pumps due to mutations in the transcriptional regulator TAC1B, and resistance is eliminated when TAC1B is deleted [2]. The expression of many transporter genes (CDR1, FRP1, FTH1, HGT7, HGT13, HSP70, NGT1, OPT1, PTR22, and SEC26) differs between azole-resistant and azole-susceptible strains, according to transcriptomic profiling data. A comparison of the proteome and lipidomic profiles of *C. auris* and *C. albicans* isolates showed that *C. auris* is more abundant in lysophospholipids, suggesting elevated phospholipase activity [2]. Ergosterol appears to be sequestered from the fungal membrane by AmB. Only a few fungal species have incredibly high levels of clinical AmB resistance, including *C. auris*. *C. albicans* and *C. glabrata* have been linked to elevated AmB resistance through mutations in the ergosterol biosynthesis pathway. However, because most cases lack alterations in genes involved in ergosterol biosynthesis, additional mechanisms underlying AmB resistance have yet to be identified. Current research has demonstrated that an AmB-resistant strain of *C. auris* regulates the ergosterol pathway and that resistance to AmB arises during treatment. Furthermore, *C. auris* has been shown to exhibit heightened resistance to AmB due to mutations in ERG6. Reduced membrane lipid permeability

may be a factor in *C. auris*'s resistance to AmB, according to a transcriptional study of isolates of the fungus resistant to the protein [2]. Mutations in the hot-spot regions of the β-1,3-glucan synthase components FKS1 and FKS2 often confer resistance to echinocandins. These mutations are linked to both clinical failures and poor medication response in pharmacodynamic investigations in animal infection models. The enzyme glucan synthase produces β-glucan, one of the primary components of the fungal cell wall. Glucan synthase inhibition by echinocandins is fungicidal because it prevents Fks1-mediated β-glucan deposition in the cell wall. Notably, data indicate that the medication may act on the extracellular face of the membrane, raising questions about whether entry into fungal cells is necessary for effective antifungal activity [2]. Additionally, echinocandin resistance in patient isolates has been linked to membrane transporters, including Cdr2; however, the role of efflux-based resistance remains debated. Echinocandin resistance in *C. auris* is mainly determined by mutations that result in substitutions at amino acid position S639 (S639F, S639P, and S639Y) in Fks1. Nevertheless, little is known about the cellular processes underlying *C. auris*'s echinocandin resistance phenotypes [2]. It is noteworthy that echinocandin resistance in *C. auris* may be promoted in concert by hyperosmotic glycerol 1 (Hog1) MAP kinase signaling and a two-component signaling pathway. Echinocandin-resistant isolates of *C. auris* have been found to have higher mannan and glucan content, exhibit improved adherence to plastic, and show differential susceptibility to the cell wall-perturbing chemical calcofluor white, according to recent transcriptional profiling research. Furthermore, it appears that *C. auris*'s resistance to echinocandins is associated with a fitness cost. Compared to susceptible isolates, macrophages preferentially phagocytose echinocandin-resistant isolates [2]. There are currently no approved human studies or vaccines that generate antibodies against *C. auris*. Results against *C. auris* have been favorable for a number of new antifungal medicines that are now completing Phase II or Phase III clinical studies. Ibrexafungerp demonstrated strong efficacy against echinocandin-resistant *C. auris* isolates. In vitro tests on Rezafungin also revealed possible effects. Tetrazole, another medication, reduces the burden and improves survival in mice infected with *C. auris* [23].

As of right now, anidulafungin, caspofungin, and micafungin, three medications from the echinocandin class, are prescribed to prevent the infection from spreading. An earlier study revealed that *C. auris* mutations in the FKS1 and ERG3 genes confer high echinocandin resistance, a property that is being evaluated in Phase II or Phase III clinical trials and has demonstrated therapeutic efficacy against *C. auris*. To date, the most promising medicine in the triterpenoid family is ibrexafungerp, which has potent activity against isolates of *C. auris*, including those resistant to echinocandins. It inhibits cell division and causes cellular deformation and pore formation [23]. Although rezafungin, a member of the echinocandin class, has demonstrated in vitro activity against clinical *C. auris* isolates and is a potential remedy, there is insufficient data to support its effectiveness against isolates resistant to other echinocandins [23]. Furthermore, in a neutropenic mouse model of *C. auris* isolation, a tetrazole known as VT-1598 was investigated and shown to significantly improve survival and reduce fungal burden in the kidneys and brain. This was particularly important because the majority of individuals who had already had medical problems were the ones who got *C. auris* isolates.

It would minimize the consequences of *C. auris* if relief were provided to areas that are likely already harmed [23].

At this time, there are no antifungal clinical breakpoints that have been specifically identified for *C. auris*. Studies assessing this organism's sensitivity to antifungals have used the E-test, the Vitek 2 yeast susceptibility method, and the Clinical and Laboratory Standards Institute (CLSI) broth microdilution. Breakpoints established for other *Candida* species (CLSI and EUCAST clinical breakpoint tables) have been compared with the MICs obtained for *C. auris* isolates. Pharmacodynamic/pharmacokinetic (PK/PD) findings from a *C. auris* candidemia mouse model support this strategy, albeit there isn't yet proof that it correlates with clinical outcomes. Resistance is not universal, although it has been shown that all geographic clusters had elevated fluconazole MICs in a significant percentage of patients (>64 mg/liter) (14). In the US, reports indicate that fluconazole-sensitive isolates are not responding to treatment. Voriconazole, itraconazole, and isavuconazole are some examples of additional triazole antifungals that have been shown to have reduced susceptibility to the infection. Furthermore, the isolates' susceptibilities to amphotericin B vary widely. As with invasive candidiasis in certain areas, the use of echinocandins as empirical therapy before susceptibility test results are available is due to concern about resistance to amphotericin B and triazole antifungal drugs [14].

1.5 Infection control strategies

Rapid acquisition, a strong correlation with high mortality rates, and high levels of antifungal resistance underscore the importance of promptly implementing Infection Prevention and Control (IPC) measures to stop transmission. The United States, Europe, South Africa, and the United Kingdom have all issued guidelines that include recommendations for patient isolation, contact precautions, and cleaning of surfaces and equipment that come into contact with affected patients [14]. Reducing hospital visits and avoiding contact with an infected individual are generally advised. The CDC does not recommend testing for *C. auris* for healthy people who come into contact with an infected person. People who have *C. auris*, a fungus, may suffer from severe illness. Its symptoms, which range from undetectable to bloodstream, wound, and/or ear infections, are most frequently observed in hospitalized patients [23]. For optimal patient care, *C. auris* must be precisely isolated in culture. Only patients with invasive fungal infections should begin treatment; those with a culture-positive status should not. Identification of patients is necessary for the implementation of suitable infection prevention and control strategies. Patients with a variety of medical conditions, including those who are immunocompromised, diabetic, or have tubes that resemble devices in their bodies, are susceptible to *C. auris* [23].

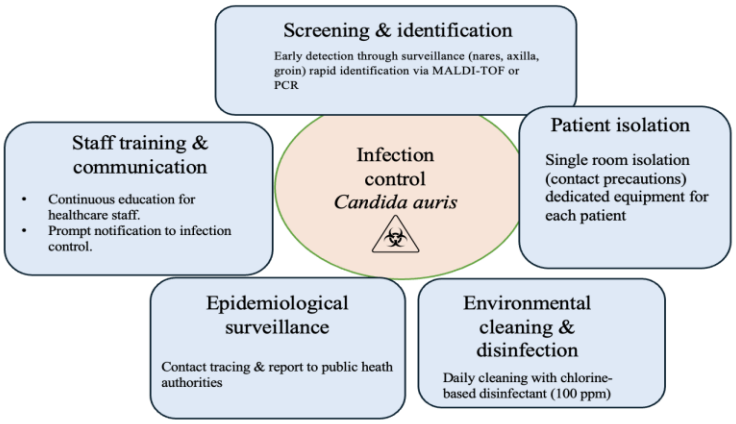

Figure 6. Continuous infection control cycle for *C. auris*.

The CDC states that hospital staff must take extra precautions if a patient is found to have contracted *C. auris*. Among the necessary countermeasures include forcing family members and medical personnel to thoroughly wash their hands after each visit, transferring the infected patient to a separate room, and wearing gowns and gloves while caring for patients. Even after the infection has been treated, it is important to take care since the microorganism may continue to be present on the skin or other regions of the body for lengthy periods of time. *C. auris* is thought to be exceedingly rare to be contracted by healthy members of a family, despite the fact that it is possible. Since the immune system can fight off *C. auris*, the CDC generally advises healthy people not to get screened for it, even if they come into contact with an infected person [23]. Public Health England (PHE) currently advises local units to create screening policies based on risk assessment. As with MRSA and CRE, screening is recommended for patients transferred from affected units, both locally and internationally. Every patient suspected of having a *C. auris* infection or a *C. auris* colony should be segregated, ideally in *en-suite* facilities. All previously positive patients should be screened upon readmission to the hospital to determine longitudinal carriage [14]. Currently, the CDC advises that patients who have at least two negative screens spaced more than a week apart and are not on antifungals be released from isolation. Except for those in units with prior experience managing *C. auris*, PHE has recommended against de-isolating patients with a positive *C. auris* sample [14]. Decolonization of patients using chlorhexidine gluconate body washes, mouthwashes, and pads impregnated with chlorhexidine for CVC exit sites are among the steps taken to stop the spread of *C. auris*. Information on how chlorhexidine body washes inhibit the growth of *C. auris* at contact times and concentrations typical of hand washing has revealed a several-log difference in inhibition when compared to *C. albicans.* On the other hand, povidone-iodine appears to be active at concentrations lower than those found in antiseptic solutions. It is currently unknown how skin disinfection practices affect colonization and shedding [14].

It has been shown that *C. auris* can live on a variety of surfaces, including plastic, both dry and moist. On plastic, the organisms can remain viable for up to 14 days. On both wet and dry surfaces, *C. auris* recovered more quickly than *C. albicans* over 7 days, suggesting the possible importance of environmental contamination [14]. Although it showed promise against several organisms, a synthetic polymer with antimicrobial properties intended for possible use

in medical devices showed no efficacy against *C. auris* [14]. When a variety of disinfectants were compared for their effectiveness against MRSA and *Candida* species, hydrogen peroxide and sodium hypochlorite produced the most considerable decrease in *C. auris* CFU. However, the CFU reduction for acetic acid, ethyl alcohol, and quaternary ammonium compounds was much less than that of MRSA [14]. Using high-concentration chlorine solutions in conjunction with hydrogen peroxide vapor or ultraviolet light to decontaminate patient areas after discharge seems to be an effective way to eradicate the organism. The necessity of thoroughly decontaminating terminally patient-contact items, such as axillary temperature probes and pulse oximeter probes, has also been highlighted by the UK's experience. Patients receiving care in community settings should, whenever feasible, be treated with the same isolation, contact, and cleaning measures as those in hospital settings. In the absence of single rooms with private bathrooms, patients colonized with *C. auris* are advised to avoid sharing facilities with immunocompromised individuals [14].

1.5. Genomics and molecular epidemiology

Techniques such as multi-locus sequence typing (MLST), whole-genome sequencing (WGS), amplified fragment length polymorphism (AFLP), and proteomic analysis using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) are utilized in order to determine the degree of genetic relatedness that exists between different strains of *C. auris*. Use of WGS has increased dramatically for the detection and description of outbreaks as well as the dynamics of *C. auris* transmission. The four major clades into which geographically distinct lineages have been identified worldwide are Clade I in Southern Asia, Clade II in Eastern Asia, Clade III in Africa, and Clade IV in South America. It was discovered that an isolate taken from a patient in Iran belonged to a different clade, which is referred to as Clade V. There were more than 200,000 single-nucleotide polymorphisms (SNPs) that differentiated this cluster from the other clades using WGS analysis. Furthermore, this cluster diverged prior to the other clades splitting apart [23]. Utilizing phenotypic and biochemical methods such as API 20C, Vitek 2 (bioMérieux), Phoenix (BD), and MicroScan (Beckman Coulter, Pasadena, California), some isolates of *C. auris* have been misidentified as belonging to a wide range of other *Candida* species. Most often, these isolates have been confused with the uncommon human infection vector *C. haemulonii*. There have been cases when *Saccharomyces*, *Rhodotorula glutinis*, *Rhodotorula mucilaginosa*, and *C. famata* were mistakenly thought to be distinct species. It has been observed that *C. auris* has been mistakenly identified as *C. catenulata*, *C. lusitaniae*, *C. guilliermondii*, or *C. parapsilosis* on few occasions. This identification has only occurred at the species level [14]. *C. auris* can be identified using many trustworthy molecular methods, including internal transcribed spacer (ITS) region sequencing, real-time polymerase chain reaction (qPCR) DNA amplification, and MALDI-TOF MS. Unfortunately, countries with insufficient healthcare resources will not be able to afford these advanced methods due to their high cost and the specific instruments and training they need. Consequently, developing a straightforward technique for reliably diagnosing *C. auris* infections is of immense importance. *C. auris* may be identified in a short amount of time and with high precision using a number of different PCR techniques [23]. Though it has been useful in separating *C. auris* from other species, sequencing genomic loci is not particularly successful in discriminating various strains

of the fungus. The D1/D2 and ITS alignments of South African isolates revealed 99% and 98% similarity to Kuwaiti and Indian isolates, respectively. One Indian *C. auris* sample showed 100% similarity with an epidemiologically unrelated strain and 98% identity with isolates from South Korea and Japan, according to ITS sequencing. An unrelated strain's large ribosomal subunit sequences were identical to this one [14].

4. Discussion

In recent years, a global increase in *C. auris* cases has drawn the world's attention to overcoming the obstacles caused by the emerging resistance of this organism, with the recent discovery of today's fearful levels of resistance. Unfortunately, most studies show a high level of resistance in *C. auris*. Some recent studies were discussed to highlight the severity of *C. auris*. The findings of the present review point to the fact that *C. auris* is more than a just another form of pathogenic yeast, it is a paradigm shift in fungal healthcare-associated infections. The fact that it simultaneously appeared in several continents in a relatively brief period as shown by genomic research [15, 24, 47] indicates that converging forces must have existed, possibly such as the antifungal use and environmental adaptation [2, 21]. In *C. auris*, the ecological niche is more specific when compared to that of its relative *C. albicans*, considering that the latter is more likely to be gastrointestinally carried with skin colonization and abiotic surface maintenance [2, 11, 15]. This characteristic is the core of its epidemiology and it has enabled the silent, clonal infections which have overwhelmed the infection control measures in health institutions all over the world [14, 25]. The most important issue is the fact that the organism is highly resistant to large antifungal classes, and most of them are simultaneously. Although mutations in ERG11 (azole resistance) and FKS1 (echinocandin resistance) are well-established [2, 15], the pan-resistance of certain strains points to a more complex, multifactorial resistance profile that is not limited by the mutations of target-sites. These involve the possibility of efflux pump overexpression, tolerance by biofilm, and cell wall remodelling [2, 28, 54]. The correlation between certain genetic clades with specific resistance mechanisms (e.g., ERG11 Y132F in Clade I) [2] is an additional epidemiology tool, with additional emphasis on the autonomous development of resistance in populations living in different geographical locations. The bottleneck of containment is a failure of diagnosis. Table 1 illustrates that traditional biochemical practices often cannot tell the difference between *C. auris* and other *Candida* spp. postponing suitable patient isolation and providing an incorrect estimate of its actual prevalence [38, 39]. This survey confirms that the key to correct identification is currently the availability of specialized methods such as MALDI-TOF MS with current databases or molecular assays [38, 52, 53], which are sometimes inaccessible in low-resource locations where the burden may be greatest. Therefore, the clinical treatment of *C. auris* infections is not without difficulties. Echinocandins are now the first-line treatment of choice, but an increasing resistance is posing a threat to this final effective class [23]. There is an encouraging and positive in vitro behaviour of new drugs such as ibrexafungerp and rezafungin [23], but their clinical success in diverse patient groups against resistant strains needs further demonstration. Moreover, the treatment is not enough; the experience of the UK outbreak highlights that the control of infection should be active, including patient seclusion, careful disinfection of the environment using sporicidal chemicals, and screening of risk contacts [14, 25]. Finally, *C. auris* represents a medical

mycological hurricane: easy to spread, endures in the environment, is unnoticed in diagnostics, and highly resistant to drugs. The fight to contain the spread necessitates a two-pronged approach consisting of urgent and stringent measures of public health and long-term funding of both basic and translational research. The nascent strategies should focus on the creation of quick-point of-care diagnostics, new therapeutic and decolonization agents, and a clearer insight into its environmental and host determinants of persistence and pathogenicity.

4. Future perspectives and research directions:

The evolving threat of *Candida aureus* requires an active and multidimensional research plan which goes past characterization to action plan. The future should be pro-actively planned on various key areas to reduce its present effects and avoid future disasters.

4.1 Diagnostic innovation and implementation science:

It should be prioritized to come up with and spread rapid, economical, and point of care tests. Although MALDI-TOF MS and PCR assays can be used, they have gaps in surveillance because they are not available in resource-constrained environments. The low-cost chromogenic media, lateral flow assays, or portable molecular device should be investigated to develop *C. aureus* specific. At the same time, studies on implementation are required to put these tools into real clinical and population health practice, particularly in long-term care homes and areas with new outbreaks.

4.2. Elucidating resistance and persistence mechanisms

A deeper understanding of the **molecular drivers of multidrug and pan-resistance** is essential. This includes investigating non-canonical resistance pathways, the role of the extracellular matrix and biofilm architecture in drug tolerance, and the impact of chromosomal rearrangements and segmental duplications on gene expression. Furthermore, its ecological and genetic attributes associated with its long-term survival on skin and abiotic surfaces, which is one of the major transmission characteristics, needs a special investigation with the help of the highly developed in vitro models coupled with the application of the appropriate models of animals colonization.

4.3. Therapeutic and preventative development:

The antifungal pipeline should be hastened. Emphasis of research should be placed on agents with novel mechanisms of action that would circumvent current resistance like exploiting fungal-specific pathways in the sphingolipid biosynthesis or cell wall integrity system. Also, it is essential to invest in non-traditional strategies; these are:

- Anti-virulence compounds that inhibit biofilm formation or adhesion.
- Immunotherapeutic approaches, such as vaccines or passive antibody therapies, particularly for high-risk, immunocompromised populations.
- Effective decolonization protocols using topical agents or microbiome-based interventions to reduce the reservoir of colonized patients.
-

4.4. Environmental ecology and one-health surveillance

There is a poor definition of the *C. auris* origins and environmental reservoirs. One-Health approach is required to sample systematically wetlands, marine and agricultural places in order to detect potential niches. The questions of the possible use of climate change to select thermotolerant environmental fungi could be informative on its relatively recent evolution into a human pathogen. It will be crucial to establish more integrated genomic surveillance networks that would integrate human clinical isolates with environmental and animal isolates in order to understand its evolution and its future spread.

4.5. Strengthening global health infrastructure and policy

Lastly, the success of translation can be achieved by building global health systems. This involves the creation of international consortia to share real-time data and isolate information, the standardization of the infection prevention and control (IPC) guidelines of local contexts, and the encouragement of antifungal stewardship programs in the world arena. Funding agencies, and the public health organizations should consider invasive fungal diseases a top priority and allocate resources to develop diagnostic and research capabilities especially in the low and middle-income countries to establish a fair and coordinated global response against *C. aureus* and future fungal infections.

It is time to the scientific and medical communities to shift the focus of their research energies towards these interlaced areas, including diagnostics, basic pathogenesis, therapeutic innovation, ecological knowledge, and health system preparedness; in that manner, the world will be able to switch the paradigm of reactively containing the disease but rather proactively manage the disease and eventually mitigate the burden of this powerful pathogen on the world.

5. Conclusions

The emergence of *C. auris* as a formidable global health threat underscores the urgent need for enhanced surveillance, rapid and accurate diagnostics, and effective therapeutic strategies. Its propensity for nosocomial transmission, high mortality rates, and frequent multidrug resistance present escalating challenges to healthcare systems worldwide. Current evidence highlights that containment requires a multifaceted approach integrating robust infection control, antimicrobial stewardship, and ongoing genomic surveillance to track emerging clades and resistance patterns. Looking forward, future efforts must prioritize the development of rapid point-of-care diagnostics, novel antifungal agents, and targeted decolonization protocols. Furthermore, international collaboration and data sharing are essential to understand the pathogen's evolution and spread. By combining vigilant infection prevention with innovative research, the global health community can mitigate the impact of *C. auris* and better protect vulnerable patient populations from this resilient pathogen.

Author Contributions

Author Contributions: Conceptualization, S.A.B. and A.M.A, S.A.B., S.M.A., and W.A.A.; Writing – Original Draft Preparation, S.A.B. and A.M.A, S.A.B., S.M.A., and W.A.A.; Writing – Review & Editing, S.A.B. and A.M.A, S.A.B., S.M.A., and W.A.A. All authors have read and agreed to the published version of the manuscript.

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Conflicts of Interest
The authors declare no conflicts of interest.

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Arabic translation of title page with Arabic abstract

الكانديدا أوريس: مراجعة شاملة لمسبب مرض متطور واستراتيجيات لإدارته

سعود عبد الله بخاري[1]*، صالح م. المعقر[1]، وائل أ. الصبحي[3]، وأحمد م. الهجن [1،2]
[1]**قسم العلوم البيولوجية، كلية العلوم، جامعة الملك عبد العزيز، ٢١٥٨٩، جدة، المملكة العربية السعودية.**
[2]**مختبر الأحياء الدقيقة المستوى ٢، مركز الملك فهد للأبحاث الطبية، جامعة الملك عبد العزيز، جدة، المملكة العربية السعودية.**
[3]**قسم ممارسة الصيدلة، كلية الصيدلة، جامعة حفر الباطن، المملكة العربية السعودية.**

*المؤلف المسؤول عن المراسلات:
sbukhari0060@stu.kau.edu.sa (S.A.B)|aalhejin@kau.edu.sa (A.M.A.H)

الملخص

الهدف: تهدف هذه المراجعة إلى تجميع الفهم الحالي للفطر الممرض الناشئ المبيضات الأذنية(Candida auris) ، مع التركيز على وبائيته، وآليات مقاومته المتعددة للأدوية، والتحديات التي تواجه تشخيصه ومكافحة العدوى، والتوجهات المستقبلية لإدارته. **المنهجية:** أُجريت مراجعة شاملة للدراسات العلمية، شملت تحليل البيانات من تقارير المراقبة العالمية، والدراسات السريرية، والأبحاث الجزيئية المنشورة حتى عام ٢٠٢٤. وتتناول المراجعة بشكل منهجي أصل الفطر الممرض، وانتشاره السلالي، وآليات مقاومته لمضادات الفطريات، وتكوين الأغشية الحيوية، والقيود التشخيصية. **النتائج:** تُظهر المبيضات الأذنية معدلات وفيات عالية (٣٠-٦٠%)، وانتقالًا سريعًا داخل المستشفيات، ومقاومة متكررة لفئات متعددة من مضادات الفطريات، بما في ذلك الأزولات، والإكينوكانديناتات، والأمفوتريسين ب. ويُسهّل وجوده المستمر على الجلد والأسطح البيئية تفشي المرض، بينما غالبًا ما تؤدي الطرق النمطية القياسية إلى تشخيص خاطئ. وقد كشف التحليل الجينومي عن أربع مجموعات رئيسية ذات توزيعات جغرافية متميزة. **الخلاصة:** يُمثل فطر المبيضات الأذنية (C. auris) تهديدًا صحيًا عالميًا خطيرًا ومتطورًا، مما يستدعي تعزيز المراقبة، والتشخيص السريع، وتطبيق إجراءات صارمة لمكافحة العدوى. ويُعدّ تطوير مضادات فطرية جديدة واستراتيجيات فعّالة للقضاء على استعمار الفطر، إلى جانب الجهود التعاونية الدولية، أمرًا بالغ الأهمية للحد من انتشاره وتحسين نتائج العلاج لدى الفئات السكانية المعرضة للخطر.

الكلمات المفتاحية: المبيضات الأذنية (Candidozyma auris)| داء المبيضات في الدم|السلالات، المقاومة| الانتشار.