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Candida tropicalis in animal health: Virulence genomics, clinical impact, and the one health challenge of azole resistance

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Abstract

Candida tropicalis, historically categorized as an innocuous commensal of mammalian and avian mucosal membranes, is rapidly gaining recognition as an aggressive, highly virulent non-albicans *Candida* (NAC) pathogen in veterinary medicine. This review provides a comprehensive, state-of-the-art evaluation of *C. tropicalis* pathobiology across production livestock, companion animals, wildlife, and aquatic ecosystems. We dissect the genomic architecture driving its virulence machinery, detailing the functions of Secreted Aspartyl Proteinases (SAPs), phospholipases, lipases, and the core transcriptional networks (*BCR1*, *EFG1*) that orchestrate robust biofilm matrix deposition and yeast-to-hyphae dimorphism.

Furthermore, we evaluate host-specific clinical manifestations, including bovine mycotic mastitis, porcine gastric ulceration syndrome, avian crop candidiasis, and systemic cetacean mycoses. A primary focus is dedicated to the escalating global crisis of triazole resistance. We trace how the heavy application of sterol 14 α -demethylase inhibitors (DMIs) in agricultural crop protection creates an environmental selective pressure, selecting for mutations (*ERG11*, *ERG3*) and efflux pump hyperactivation (*CDR1*, *MDR1*) that cross-resist veterinary clinical triazoles. Framed within a One Health paradigm, this paper underscores the critical need for advanced molecular diagnostics, routine veterinary mycological surveillance, and strict antifungal stewardship.

Keywords: *Candida tropicalis*; Non-albicans *Candida*; Virulence genomics; Triazole resistance; Bovine mastitis; One Health; Demethylation inhibitors (DMIs); Fungal pathobiology; Biofilm dynamics

Introduction

In contemporary veterinary mycology, the paradigm of opportunistic yeast infections is undergoing a significant epidemiological shift. While *Candida albicans* has historically been diagnosed as the primary etiologic agent of animal candidiasis, non-albicans *Candida* (NAC) species are increasingly isolated from critical clinical cases worldwide (Barchiesi & Arzeni, 2023) [6]. Among these, *Candida tropicalis* represents an escalating threat within both production and companion animal medicine (Andrade & Silva, 2025; Colombo & de Almeida, 2022) [2, 10]. As a diploid, polymorphic yeast belonging to the CTG clade—where the CTG codon translates to serine instead of leucine—*C. tropicalis* exhibits an evolutionary plasticity that facilitates rapid adaptation to distinct anatomical and environmental microenvironments (Quynh & Turner, 2024; Zuza-Alves *et al.*, 2017) [27, 42].

The epidemiology of veterinary candidiasis is undergoing a profound shift. While *Candida albicans* was historically the dominant etiological agent, non-albicans *Candida* (NAC) species are increasingly isolated from severe veterinary cases worldwide (Barchiesi & Arzeni, 2023) [6]. Among these, *Candida tropicalis* has emerged as a particularly virulent pathogen, capable of clonal spread across the human–animal–environment interface (Zhang & Mitchell, 2024) [41]. Its rise is especially notable in Asia, where changing epidemiological patterns highlight NAC species as significant contributors to systemic infections (Tan & Chakrabarti, 2023) [32]. The clinical upsurge of *C. tropicalis* within veterinary facilities is multifactorial, driven by:

- The widespread, often indiscriminate usage of broad-spectrum antibacterial therapies
- that decimate protective mucosal microbiomes (Pappas & Kauffman, 2024) [23].
- Enhanced survival rates of immunosuppressed veterinary patients undergoing prolonged intensive care, oncology protocols, or corticosteroid regimens (Tortorano & Prigitano, 2024) [35].
- The increased deployment of invasive biomedical devices, such as indwelling intravenous catheters, urinary lines, and mechanical milking systems, which serve as perfect substrates for fungal colonization (Silva *et al.*, 2012) [30].

Crucially, *C. tropicalis* is not merely an incidental bystander; epidemiological tracking demonstrates its capacity to rapidly transition from a benign mucosal symbiont into an invasive systemic pathogen capable of inducing lethal candidemia and disseminated granulomatous lesions (Deorukhkar & Saini, 2024; Pfaller & Diekema, 2007) [11, 24]. This review provides a technically rigorous, state-of-the-art analysis of *C. tropicalis* pathobiology at the intersection of animal health, environmental pressures, and the global One Health framework.

The clinical surge of *C. tropicalis* driven by indiscriminate antibiotic use (Pappas & Kauffman, 2024) [23], prolonged immunosuppressive therapies (Tortorano & Prigitano, 2024) [35], and invasive biomedical devices that provide substrates for colonisation (Silva & Negri, 2012) [30]. Surveillance confirms its ability to cause lethal candidemia and disseminated granulomatous lesions (Deorukhkar & Saini, 2024; Pfaller & Diekema, 2007) [11, 24].

2. Physiological and Taxonomic Distinctions

To understand the unique clinical threat of *C. tropicalis*, it must be compared to the historical reference species, *Candida albicans* (Silva *et al.*, 2012) [30]. Both organisms are diploid yeasts belonging to the CTG clade (Zuza-Alves *et al.*, 2017) [42]. However, their metabolic, physiological, and genomic traits differ significantly.

[Candida Genus]

+---> *Candida albicans* (True hyphae dominate; chlamydospores present; green on CHROM agar)

+---> *Candida tropicalis* (Abundant pseudohyphae; sucrose/maltose fermentation; dark blue on CHRO Magar)

2.1 Metabolic Phenotypization

Unlike *C. albicans*, *C. tropicalis* vigorously ferments and assimilates a wide array of carbohydrates, including sucrose, maltose, and galactose, producing a characteristic acid-gas profile during biochemical tube testing (Zuza-Alves *et al.*, 2017) [42]. On standard Sabouraud Dextrose Agar (SDA), *C. tropicalis* develops smooth, creamy, off-white to cream-colored colonies (Al-Obaidi & Mohammad, 2024) [1]. Over time, these colonies develop wrinkled or fringed borders due to extensive pseudohyphal growth into the agar layer. It lacks the ability to form true chlamydospores under nutrient-deprived conditions (e.g., on cornmeal agar), which serves as a key morphological differentiator from *C. albicans* (Silva *et al.*, 2012) [30].

2.2 Chromogenic Divergence

On specialized chromogenic media such as CHROM agar Candida, *C. tropicalis* isolates produce smooth, raised, distinctly dark blue to metallic blue-grey colonies, often surrounded by a diffuse purple-pink halo in the surrounding medium (Deorukhkar & Saini, 2024) [11]. This presentation clearly contrasts with the bright emerald-green colonies produced by *C. albicans* and the fuzzy, pinkish colonies characteristic of *C. krusei* (Tortorano & Prigitano, 2024) [35]. This colour variance is driven by the specific breakdown of chromogenic substrates by species-specific intracellular β -glucosidase enzymes (Zuza-Alves *et al.*, 2017) [42].

3. Virulence Genomics and Pathogenicity Pathways

The capacity of *C. tropicalis* to transition from a harmless commensal to an aggressive tissue invader relies on an intricate arsenal of virulence factors (Morace & Borghi, 2024) [22].

[Adhesion to Mucosa] —> [Bud-to-Hyphae Transition] —> [Extracellular Enzyme Secretion] —> [Biofilm Formation & Tissue Invasion]

3.1 Adherence and Biofilm Regulatory Networks

The initial stage of infection requires the attachment of yeast cells to host endothelial or epithelial surfaces (Silva *et al.*, 2012) [30]. *C. tropicalis* possesses a family of genes homologous to the *C. albicans* Agglutinin-Like Sequence (ALS) proteins, which encode cell-surface glycoproteins that bind to host ligands (Zuza-Alves *et al.*, 2017) [42]. Following initial attachment, the expression of *HWPI* (Hyphal Wall Protein 1) homologs mediates covalent cross-linking with host cells (Quynh & Turner, 2024) [27].

The transition from localized attachment to mature biofilm formation is highly aggressive in *C. tropicalis*, frequently yielding structurally denser and more chemically complex extracellular matrices than those of *C. albicans* (Scorzoni & de Lucas, 2024) [29]. This process is strictly orchestrated by master transcriptional regulators:

- **BCR1 (Biofilm Cascade Regulator 1):** Regulates the downstream expression of cell-surface adhesins, crucial for establishing the foundational framework on both biotic tissue and abiotic medical polymers (Quynh & Turner, 2024) [27].
- **EFG1 (Enhanced Filamentous Growth 1):** Coordinates the structural maturation of the biofilm, embedding budding yeasts within a dense network of pseudohyphae and true hyphae (Quynh & Turner, 2024) [27].

The resulting extracellular matrix consists of a complex network of glucans, mannans, and extracellular DNA (eDNA) (Scorzoni & de Lucas, 2024) [29]. This matrix acts as a physical and chemical shield, neutralizing the oxidative burst of host neutrophils and restricting the physical diffusion of conventional polyene and triazole antifungals by up to 1000-fold (Scorzoni & de Lucas, 2024; Whaley & Rogers, 2017) [29, 40].

3.2 Morphological Plasticity (Dimorphism)

The capacity to switch reversibly between ovoid blastoconidia (yeast form) and elongated filamentous structures (pseudohyphae and true hyphae) is an absolute prerequisite for full virulence (Morace & Borghi, 2024) [22]. This morphogenetic switching is regulated primarily through the cyclic adenosine monophosphate-protein kinase A (cAMP-PKA) signalling pathway, activated by environmental cues

such as elevated host core temperature (37 °C to 39 °C), ambient CO₂ concentration, and neutral-to-alkaline pH (Quynh & Turner, 2024) [27].

- **The Yeast Morphotype:** Mechanically optimized for dissemination via the hematogenous and lymphatic routes, allowing the pathogen to rapidly seed deep visceral organs (Thompson & Patterson, 2024) [34].
- **The Hyphal Morphotype:** Displays mechanical rigidity and directional growth (thigmotropism), which pierces host mucosal barriers and vascular endothelium (Morace & Borghi, 2024) [22]. Hyphae actively secrete tissue-destructive enzymes and physically disrupt macrophage membranes following phagocytosis, causing immune cell lysis.

3.3 The Extracellular Secretome

Once tissue penetration is initiated, *C. tropicalis* deploys an extensive secretome composed of hydrolytic enzymes that catalyze tissue breakdown and facilitate nutrient acquisition (Andrade & Silva, 2025) [2].

Secreted Aspartyl Proteinases (SAPs)

Encoded by a multigene family (*SAPT1* through *SAPT4*), these enzymes cleave vital host structural barriers, including collagen, laminin, fibronectin, and secretory immunoglobulin A (sIgA) (Morace & Borghi, 2024) [22]. *SAPT1* and *SAPT2* are highly up-regulated during mucosal colonization, whereas *SAPT3* and *SAPT4* expression spikes during systemic vascular invasion and parenchymal organ destruction (Zuza-Alves *et al.*, 2017) [42].

Phospholipases and Lipases

Phospholipases (specifically Class B, *PLB*) target the ester linkages of host cell-membrane phospholipids, inducing localized membrane destabilization, cytolysis, and deep tissue necrosis (Deorukhkar & Saini, 2024) [11]. Concurrently, extracellular lipases (*LIP* gene family) hydrolyse host lipid reserves, providing the fungus with essential carbon sources in nutrient-deprived tissue microenvironments (Morace & Borghi, 2024) [22].

Hemolysins

Systemic persistence requires iron acquisition. *C. tropicalis* secretes distinct hemolysins (both alpha and beta-type) that bind to and lyse host erythrocytes (Deorukhkar & Saini, 2024) [11]. This action releases haemoglobin, which is then processed via specialized fungal cell-surface iron-reuptake systems, depleting host iron reserves and contributing to systemic anaemia (Zuza-Alves *et al.*, 2017) [42].

4. Host-Specific Pathobiology and Clinical Manifestations

The susceptibility to *C. tropicalis* spans across multiple animal phyla, manifesting in distinct clinical syndromes based on anatomical vulnerability.

4.1 Bovine Mastitis and Reproductive Tract Pathologies

In production dairy medicine, *C. tropicalis* is an escalating cause of mycotic mastitis (Garcia & Blanco, 2024) [13]. It typically enters the mammary gland via ascending contamination through the teat canal, frequently exacerbated by contaminated teat-dipping solutions, poor sanitization of automated milking clusters, or the abusive intramammary administration of broad-spectrum antibiotics for bacterial mastitis (Garcia & Blanco, 2024) [13].

- **Pathology:** The yeast colonizes the lactiferous ducts and alveolar spaces, establishing dense biofilms that induce chronic granulomatous inflammation (Santos & Ribeiro, 2026) [28]. Affected quarters exhibit marked induration, fibrosis, and a dramatic drop in milk production. The milk secretion shifts to a serous, yellowish fluid containing thick, flaky fungal clumps.
- **Reproductive Failure:** *C. tropicalis* is frequently isolated from cows presenting with chronic endometritis, vulvovaginitis, and repeat-breeding syndrome (Santos & Ribeiro, 2026) [28]. It can cross the placental barrier, leading to mycotic abortion storms characterized by foetal cotyledonary necrosis and cutaneous foetal dermatitis.

4.2 Porcine Gastrointestinal Ulceration Syndrome

Within intensive swine production systems, *C. tropicalis* causes acute, often fatal gastrointestinal mycoses in sows and post-weaning piglets (Sun & Wang, 2021) [31]. The pathogen targets the pars oesophageal—the non-glandular stratified squamous epithelial region of the porcine stomach—which is highly susceptible to mechanical micro-trauma from finely ground feed rations (Sun & Wang, 2021) [31].

Pathology: Following mechanical mucosal abrasion, *C. tropicalis* rapidly transitions to its hyphal form, penetrating deep into the submucosa and muscularis layers (Sun & Wang, 2021) [31]. This rapid invasion induces vast, crateriform ulcers characterized by extensive coagulative necrosis, fibrinopurulent exudates, and direct erosion of major gastric blood vessels, resulting in catastrophic, fatal intra-gastric haemorrhaging.

4.3 Avian Upper Gastrointestinal Candidiasis

Avian candidiasis, colloquially termed "sour crop" or "crop mold," severely impacts both commercial poultry operations and companion psittacine (Tell & Woods, 2016) [33]. *C. tropicalis* proliferates within the crop, oral cavity, and oesophagus, particularly following nutritional deficiencies (e.g., Hypovitaminosis A) or prolonged tetracycline therapy (Tell & Woods, 2016) [33].

Pathology: The macroscopic hallmark is the presence of elevated, confluent, yellowish-white pseudomembranous across the mucosal lining, imparting a corrugated "Turkish Towel" appearance (Tell & Woods, 2016) [33]. Histologically, these lesions consist of hyperkeratotic epithelial debris heavily infiltrated by interlacing networks of pseudohyphae and blastoconidia. Clinical signs include delayed crop emptying, regurgitation of foul-smelling fluids, severe dysphagia, rapid weight loss, and high mortality in juvenile birds.

4.4 Companion Animal Dermatmycoses and Otitis

In canine and feline practices, *C. tropicalis* presents as an opportunistic cutaneous and mucosal pathogen (Silva *et al.*, 2012) [30]. It is a primary or secondary driver of chronic, non-responsive otitis externa, typically complicating underlying primary atopic dermatitis or food allergies (Andrade & Silva, 2025) [2]. Dogs and cats suffer otitis externa and urinary tract infections, particularly in immunocompromised states (Andrade & Silva, 2025; Walsh & Anaissie, 2024) [2, 39]. Ocular mycoses, including fungal keratitis, have also been reported (Vicente & Azevedo, 2024) [38].

Equine cutaneous infections have been successfully managed

with systemic antifungals (van Asbeck & Clemons, 2025) [37]. In dolphins, *C. tropicalis* causes necrotising pneumonia, while in fish it induces visceral granulomas (Reidarson & McBain, 2024; Kano & Hasegawa, 2024) [26, 16].

Clinical Signs: Marked erythema of the vertical and horizontal ear canals, stenotic stenosis, and a thick, sweet-smelling, dark mucoid discharge.

Systemic Urogenital Infections: In animals managed with long-term indwelling urinary catheters or those with metabolic comorbidities like poorly controlled diabetes mellitus or hyperadrenocorticism, *C. tropicalis* establishes ascending infections, leading to necrotizing cystitis and pyelonephritis (Walsh & Anaissie, 2024) [39].

4.5 Marine Mammal and Aquatic Wildlife Mycopathology
The impact of *C. tropicalis* extends into marine and freshwater ecosystems, where it presents unique conservation and management challenges.

Cetacean Respiratory Mycoses: In captive and wild bottlenose dolphins (*Tursiops truncatus*), *C. tropicalis* acts as a highly dangerous respiratory pathogen (Reidarson &

McBain, 2024) [26]. It colonizes the blowhole and larynx, tracking down the trachea into the pulmonary parenchyma. This causes necrotizing granulomatous pneumonia, often culminating in fatal systemic fungemia characterized by multi-organ failure.

Teleost Disseminated Infections: In unique cold-water aquatic species, such as the Okhotsk snailfish (*Liparis ochotensis*), environmental strains of *C. tropicalis* have been isolated from deep visceral granulomas (Kano & Hasegawa, 2024) [16]. The pathogen induces disseminated multi-organ focal necrosis within the gills, renal tissue, and hepatic parenchyma, confirmed via internal transcribed spacer (ITS) rRNA gene sequencing.

5. Diagnostic Frameworks in Veterinary Mycology
Accurate identification is vital to differentiate *C. tropicalis* from other yeast pathogens and guide effective treatment (Barchiesi & Arzeni, 2023) [6]. Phenotypic assays (CHROM agar), histopathology (PAS/GMS), and molecular assays (ITS-rDNA PCR) remain essential (Araujo & Rego, 2024)-[4]. Multicenter surveillance confirms rising resistance patterns, highlighting the need for molecular confirmation (Guinea-*et al.*, 2014) [14].

Diagnostic Modality	Analytical Characteristics & Visual Presentation	Clinical Utility
Phenotypic Medium (CHROM agar)	Produces smooth, distinctly blue to blue-grey colonies. (Deorukhkar & Saini, 2024) [11]	Rapid, cost-effective differentiation from green <i>C. albicans</i> colonies. (Tortorano & Prigitano, 2024) [35]
Microscopic Morphology	Shows oval budding yeast cells (3–6 µm) with abundant, elongated pseudohyphae. (Silva <i>et al.</i> , 2012) [30]	Confirms active tissue invasion and structural fungal morphology. (Morace & Borghi, 2024) [22]
Histopathology (PAS / GMS)	Reveals intra-alveolar exudates, ulceration, and direct vascular infiltration. (Sun & Wang, 2021) [31]	Direct visualization of the pathogen within granulomas or biopsied tissues. (Thompson & Patterson, 2024) [34]
Molecular Assays (ITS-rDNA PCR)	Sequences the internal transcribed spacer region of ribosomal DNA. (Kano & Hasegawa, 2024) [16]	Gold standard for definitive, species-level differentiation. (Araujo & Rego, 2024) [4]

6. The Antifungal Resistance Dilemma: A One Health Crisis

The most alarming aspect of *C. tropicalis* epidemiology is its rapidly expanding resistance to azole therapeutics, particularly fluconazole and itraconazole (Araujo & Rego, 2024; Lackner & Lass-Flörl, 2023) [4, 18]. Surveillance studies confirm that resistance patterns are not isolated to *C. tropicalis* but extend across multiple NAC species, reflecting a broader clinical challenge (Astvad *et al.*, 2018) [5].

[Agricultural Azole Overuse] —> [Environmental Selection in Soil/Water] —> [Animal Colonization & Infection] —> [Therapeutic Failure]

6.1 Molecular Mechanisms of Triazole Resistance
Veterinary isolates of *C. tropicalis* exhibit complex, multi-tiered genetic strategies to counter azole stress (Jiang & Wang, 2025; Whaley & Rogers, 2017) [15, 40].

Target-Site Mutations

Missense mutations within the *ERG11* gene (which encodes lanosterol 14α-demethylase, the target enzyme of triazoles) induce conformational shifts near the catalytic iron centre (Jiang & Wang, 2025) [15]. Common veterinary substitutions, such as Y132F, K143R, and S154F, drastically decrease the binding affinity of triazoles while preserving the enzyme's capacity to process its natural sterol substrate (Lackner & Lass-Flörl, 2023) [18].

Efflux Pump Overexpression
Resistance is frequently mediated by the active exclusion of the drug from the fungal cytoplasm (Berkow & Lockhart, 2017) [7]. This is achieved via two main classes of efflux pumps:

- **ATP-Binding Box (ABC) Transporters:** Encoded by *CDR1* (Candida Drug Resistance 1) and *CDR2*, these pumps rely on ATP hydrolysis to actively transport a wide array of azoles across the plasma membrane (Berkow & Lockhart, 2017) [7].
- **Major Facilitator Superfamily (MFS) Pumps:** Encoded by *MDR1*, this proton-motive force-dependent pump specifically drives the efflux of fluconazole (Berkow & Lockhart, 2017) [7]. The hyperactivation of these pumps is typically driven by gain-of-function mutations in transcription factors like *TAC1* and *MRR1* (Whaley & Rogers, 2017) [40].

Ergosterol Bypass Pathways

Loss-of-function mutations within the *ERG3* gene (encodes the enzyme sterol C-5(6)-desaturase) represents an advanced resistance mechanism (Jiang & Wang, 2025) [15]. When triazoles inhibit Erg11, intact Erg3 normally processes aberrant sterol intermediates into toxic methylated sterols that disrupt membrane integrity (Whaley & Rogers, 2017) [40]. Mutations in *ERG3* halt this process. This allows *C. tropicalis* to incorporate non-toxic alternative sterols into its plasma membrane, preserving viability even in the complete absence of ergosterol (Jiang & Wang, 2025) [15].

Large collections of isolates confirm diverse susceptibility patterns (Castanheira *et al.*, 2016) [8]. Surveillance studies highlight azole resistance in agricultural settings (Chowdhary *et al.*, 2024) [9].

6.2 The Environmental and Agricultural Nexus

Unlike other *Candida* species that rely heavily on host-to-host transmission, *C. tropicalis* is highly ubiquitous in soil, freshwater systems, and agricultural environments (Lima & Colombo, 2023) [19]. The widespread application of triazole-based fungicides (demethylation inhibitors, DMIs) in crop protection exerts continuous selective pressure (Fan & Xiao, 2024) [12]. This creates an environmental reservoir of azole-resistant *C. tropicalis* (ARCT) strains (Fan *et al.*, 2023) [12]. Animals contract these resistant fungi directly from their surroundings, leading to primary therapeutic failures upon clinical presentation (Andrade & Silva, 2025; Colombo & de Almeida, 2022) [2, 10]. Resistant genotypes have been traced to orchards in Taiwan, linking agriculture directly to clinical outbreaks (Tseng *et al.*, 2024) [36].

6.3 Global Perspective

Simultaneous emergence of multidrug-resistant *Candida* species across continents underscores the global nature of the crisis (Lockhart, Etienne, Vallabhaneni, Farooqi, Chowdhary, Govender, Colombo, Concepcion-Acevedo, Alpaesan, Chiller, & Litvinseva, 2017) [20]. Stewardship is urgently needed to mitigate resistance spread (Megri & Arastehfar, 2020) [21].

7. Therapeutic Options and Management Strategies

Treating *C. tropicalis* requires a structured approach tailored to the infection's location and severity, ideally guided by standardized antifungal susceptibility testing (AFST) protocols (Pappas & Kauffman, 2024) [23].

Localized infections respond to topical polyenes and antiseptics (Silva & Negri, 2012) [30]. Systemic infections require echinocandins or amphotericin B, though cost and toxicity limit use (Kuse & Chetchotisakd, 2024; Pappas & Kauffman, 2024) [17, 23]. MIC correlation studies provide interpretive breakpoints for voriconazole therapy (Pfaffer, Diekema, Rex, Alexander, Andes, Brown, Chaturvedi, Ghannoum, Knapp, Lockhart, Walsh, & Werner, 2011) [23].

Localized Interventions

Superficial mucosal infections or otitis externa are managed using topical polyenes (e.g., nystatin) or structural antiseptic flushes (Silva *et al.*, 2012) [30]. If azole susceptibility is confirmed via broth microdilution, oral fluconazole remains a practical choice for canine or feline cystitis (Walsh & Anaissie, 2024) [39].

Systemic Protocols

For deep organ involvement or systemic candidemia, treatment choices are limited (Kuse & Chetchotisakd, 2024) [17].

- **Echinocandins (Caspofungin / Micafungin):** These agents inhibit β -(1,3)-D-glucan synthesis, offering excellent efficacy and minimal resistance profile (Kuse & Chetchotisakd, 2024) [17]. However, high costs often restrict their use in production medicine.
- **Polyenes (Amphotericin B):** Targets cell membrane ergosterol directly (Pappas & Kauffman, 2024) [23]. It retains high stability against azole-resistant strains but requires close monitoring for nephrotoxicity in

companion animals.

Biosecurity and Stewardship

To manage the emergence of *C. tropicalis*, veterinary operations must implement rigorous antifungal stewardship programs (Colombo & de Almeida, 2022) [10]. This includes minimizing the prophylactic use of broad-spectrum antibiotics, maintaining routine disinfection protocols for milking machinery using verified fungicidal agents, and systematically monitoring regional antifungal susceptibility patterns (Garcia & Blanco, 2024; Santos & Ribeiro, 2026) [13, 28].

8. Conclusion and Future Directions

Candida tropicalis can no longer be viewed as an inconsequential commensal organism in veterinary spaces. Driven by robust biofilm synthesis and extensive environmental azole selection, this pathogen presents an escalating threat to domestic animals, wildlife, and veterinary public health.

Future research must focus on defining its precise zoonotic transmission dynamics, expanding molecular sequencing data for veterinary isolates, and identifying novel non-fungicidal therapeutic targets to bypass established efflux mechanisms.

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