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Overcoming Cryptococcus Neoformans Pathogenicity Based on Copper Homeostasis Interference

Jingjie Xu

Department of Biology, College of Science, Shantou University, Shenzhen, China
xu-jingjie0305@outlook.com

Abstract. Cryptococcus neoformans, a neurotropic fungus that opportunistically affects individuals with weakened immune systems, has the ability to transmit to the central nervous system and induce severe meningitis, representing a global challenge. Copper is an integral and essential part in regulating the interplay between fungal pathogen and host. The host mounts immune defenses by accumulating copper at infection sites to exert antifungal effects, while the fungus utilizes an intricate copper homeostasis regulatory network to adapt to varying microenvironments within the host. Copper homeostasis also offers a vast, yet untapped space for potential treatment options and varieties of compounds have shown efficacy. However, there may be major limits to clinical application. Altogether, future efforts should focus on developing targeted delivery platforms, and improved understanding of copper homeostasis may provide promising treatments to address C. neoformans and other invasive fungal pathogens.

Keywords: Cryptococcus neoformans, Copper, Homeostasis.

1. Introduction

As a typical opportunistic pathogenic fungus, Cryptococcus neoformans has a wide range of ecological adaptability. Environmental microbiology studies have shown that it has been isolated in soil, humus of trees, and bird feces [1]. Cryptococcus neoformans exhibit complex mechanisms as an opportunistic pathogen. Specifically, its capsular polysaccharide can inhibit phagocytosis by phagocytes; The virulence factors such as phospholipase and protease secreted from the cytoplasm can degrade the components of the host extracellular matrix and weaken the immune effect. After breaking through the innate immune defense, Cryptococcus neoformans undergoes hematogenous dissemination, preferentially binding to specific cell surface receptors expressed by cerebrovascular endothelial cells. It then breaks through the blood-brain barrier (BBB) through an endocytosis mediated mechanism [2]. In the central nervous system (CNS), pathogens enhance invasion ability through years-mycelia phase transition, and release toxic metabolites such as melanin, which eventually leads to Cryptococcal meningitis (CM) [3]. Patients often have severe headache, fever, vomiting, neck stiffness and other symptoms. Unfortunately, even after receiving standard antifungal treatment, survivors still have permanent neurological sequelae such as cognitive dysfunction, hearing loss and motor dysfunction. In 2022, C. neoformans was listed as a primary control

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pathogen by World Health Organization (WHO). It has the most urgent need for prevention and control.

The application of traditional antifungal drugs faces significant difficulties. In the case of amphotericin B (AmB), intravenous infusion led to acute infusion reactions, and patients often presented with chills, high fever, and headache. Meanwhile, this drug can specifically bind to ergosterol on the cell membrane, disrupt the mitochondrial function of renal tubular epithelial cells, trigger irreversible nephrotoxicity, and lead to serious consequences such as abnormally elevated serum creatinine levels and electrolyte metabolism disorders. Flucytosine (5-FC) exerts antibacterial activity by inhibiting fungal DNA synthesis, but it is easy to induce severe bone marrow suppression and liver function damage during the treatment. The standard treatment of Cryptococcus neoformans recommended by WHO is the combination of AmB and 5-FC, which requires patients to receive continuous treatment. This usually requires an extension of the dosing period by 6 weeks, posing a significant challenge in ensuring patient compliance. Effective therapy for CM remains limited. Thus, the development of new therapeutic strategies for novel Clostridium neoflorum is particularly urgent.

Recent years, nutritional immunity has provided new insights for understanding host-pathogen interactions. Its core is that the host creates a microenvironment that is not conducive to the growth and reproduction of pathogens by regulating the distribution and availability of essential micronutrients [4]. In fact, the importance of metals in pathogenicity has been extensively studied; among them, copper has attracted much attention. Copper exhibits dual function in the host-pathogen interface, serving as an indispensable micronutrient while concurrently exerting effective defense effects. Biochemically, copper ions act as cofactors for numerous enzymatic reactions critical to both host and pathogen metabolism. Conversely, the innate immune response of mammals actively utilizes copper, generating reactive oxygen species (ROS), which directly target and eliminate invading pathogens. Although the molecular mechanisms may differ, the basic problems faced by microorganisms are essentially the same, and both groups of fungal pathogens may have found similar solutions. As a successful pathogen, Cryptococcus neoformans also has a set of complex copper homeostasis regulation mechanisms to survive and establish infection in this challenging host environment. This research focuses on the host's and Cryptococcus neoformans' intense offensive and defensive battles over copper elements and how the host uses copper to implement immune defense and how C. neoformans effectively regulate copper homeostasis to maintain virulence. Ultimately, the study also explores potential therapeutic strategies for developing treatments against invasive CM infections.

2. Copper serves as a nutrient and weapon for the host

Copper, a vital micronutrient for living organisms, functions as a REDOX active core in numerous crucial enzymes. Its distinctive coordination stability and electron transfer properties are fundamental to the efficient operation of enzymatic reactions. Notably, cytochrome c oxidase (COX), a key component of mitochondrial respiratory chain complex IV, relies on copper ions to catalyze the terminal reduction of oxygen to water, thereby regulating cellular energy production. Similarly, copper-zinc superoxide dismutase (Cu/Zn-SOD) dismutase leverages the redox cycling of copper to detoxify intracellular reactive oxygen species. Moreover, copper-dependent enzymes such as tyrosinase and lysyl oxidase are essential for neurotransmitter synthesis and connective tissue integrity, respectively. Normal copper levels are crucial for cellular aerobic respiration, oxidative stress homeostasis, neural signal transduction and connective tissue integrity [5].

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The imbalance of copper metabolic homeostasis (including copper deficiency or excess) will lead to a series of pathophysiological changes in the host: Hematopoietic dysfunction, abnormal neural development and immune dysfunction, while copper excess will cause tissue damage through the accumulation of oxidative stress, especially causing specific toxic effects on the liver, kidney and brain. Typical diseases include Wilson disease, Menkes disease and other hereditary copper metabolism disorders [6].

On the other hand, when pathogens are phagocytosed by macrophages, the host will use copper enrichment to kill pathogens. Macrophages are the key effector cells to execute the copper enrichment strategy, and the copper transport mechanism involves the coordinated action of multiple key proteins. In the resting state, copper homeostasis in macrophages is mainly maintained by the copper transporters Ctr1 and the copper transport atpase ATP7A. In the study of Cryptococcus neoformans infection, host macrophages can up-regulate the expression of Ctr1, on the other hand, promote the localization ATP7A to the phagosome membrane, and the two synergistically act to kill the invading fungi [7,8]. This disruption impairs essential pathogen functions, including DNA repair, metabolic pathways, and respiratory chain electron transfer. Copper-induced multi-target protein damage synergizes with reactive oxygen species-mediated oxidative stress within phagolysosomes, establishing the molecular foundation for copper's broad-spectrum defense efficacy.

3. Regulation of copper homeostasis and pathogenicity in Cryptococcus neoformans

Cryptococcus neoformans also possesses a complex copper homeostasis regulation mechanism to counter the host's complex copper-based immune defense. It not only enables pathogens to thrive in hostile host environments but also enhances pathogenic potential. Compared with the two copper sensing pathways of typical pathogenic fungi, such as Saccharomyces cerevisiae Ace1 and Mac1, C. neoformans have a unique bidirectional regulation [7]. This regulatory framework encompasses a specific copper uptake mechanism and an efficient copper detoxification pathway, which works in synergy to lay a crucial foundation for the survival and virulence of C. neoformans in the complex host microenvironment.

3.1. Copper-dependent virulence expression

Copper serves for the construction of the virulence of Cryptococcus neoformans, which is the core link for it to break through the host immune defense and establish invasive infection. Melanin is the shield of Cryptococcus neoformans against host clearance and drug attack. The biosynthesis and functional activation of C. neoformans depend on the catalysis of laccase mediated by copper ions, which is an irreplaceable cofactor of laccase. In terms of structural insight, laccase (mainly encoded by the LAC1 gene) belongs to the multi-copper oxidase superfamily, and its active center contains three types of highly conserved copper ion binding sites, which is responsible for the capture of electrons from the substrate, transferring electrons to molecular oxygen and catalyzing the oxidation and polymerization of the substrate [9]. During the pathogenic process of C. neoformans, laccase utilizes the abundant endogenous polyphenol substrates in the host microenvironment (such as dopamine, norepinephrine, catecholamines, etc.) to generate quinone intermediates through single-electron oxidation reactions, which then polymerize to form insoluble melanin granules, deposit on the cell wall and construct a defense barrier [10].

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3.2. Copper acquisition and detoxification process

Based on the comprehensive analysis of RNA-Seq and ChIP-Seq, the core of copper acquisition system is a transcription factor Cuf1 and high-affinity copper transporter CTR4. As the central commander of copper homeostasis in Cryptococcus neoformans, Cuf1 localizes to the nucleus and activates the expression of copper absorption-related genes under low copper condition. Under the condition of high copper, Cuf1 transferred to the cytoplasm. CTR4 gene expression is tightly regulated by Cuf1 and is induced under low copper conditions [11]. The copper acquisition strategy of C. neoformans in the brain also involves copper binding protein Bim, a protein associated with lytic polysaccharide monooxygenase (LPMO), which is strongly induced during copper restriction and whose expression drives brain colonization in a mouse model of infection [12,13].

When Cryptococcus neoformans are trapped within the macrophage phagosome, its survival depends on the robust copper detoxification system. In the copper-laden environment of host phagolysosomes, Cryptococcus neoformans deploys a dual-pronged detoxification strategy. The metallothionein CMT1 and CMT2 form an intracellular chelating defense. Their cysteine-rich structure stabilizes copper binding and is synergistic when co-expressed, exhibiting high selectivity for Cu^{+}. Furthermore, their expression is strictly regulated by the copper-reactive transcription factor Cuf1, which is rapidly induced under high copper stress (e.g., in macrophage phagosomes) to isolate excessive Cu^{+} and prevent it from attacking the key metabolic enzyme iron-sulfur (Fe-S) cluster exposed to solvents. Further, this chelation function is complementary to the ATM1-mediated Fe-S cluster repair system [14]. In survival, if missing Atm1, cytoplasmic Fe-S function to the collapse, even if the Cu were CMT1 /CMT2 collectively chelate, still cannot maintain basal metabolism, leading to a 40% drop in overall CFU in macrophages ($P = 0.03$). The demonstration of its importance in host copper stressed microenvironment provides a potential target for the interference of antifungal drugs with copper chelating.

3.3. Copper regulation strategies of Cryptococcus neoformans during infection stages

The infection of Cryptococcus neoformans begins with inhalation of the lungs and can subsequently spread to CNS and other sites. There is a great difference in the availability of copper between the alveolar and cerebrospinal fluid microenvironments. For instance, in the early stage of lung infection, Cuf1 transcription factor of Cryptococcus neoformans upregulates CMT1/CMT2 to resist the copper attack of macrophages [8]. On the other hand, the BBB strictly restricts the entry of copper, resulting in an extremely low copper concentration in cerebrospinal fluid (0.5 μmol/L) [15]. Thus, when infection spreads to the brain, C. neoformans in turn significantly upregulate the expression of CTR4 in order to grab the copper necessary for survival from the low-copper environment. Analysis of cryptococcus clinical isolates from solid organ transplant recipients showed that strains with high CTR4 expression levels were more likely to cause disseminated infection in CNS, suggesting that efficient copper acquisition ability is a key predictor of neuroinvasiveness [16]. Interestingly, Cir1, which is involved in iron regulation, affects the formation of C. neoformans cell wall and has cross-regulation with Cuf1, which synergistically enhances the pathogen's ability to adapt to the host microenvironment.

4. Copper-targeted antifungal drugs and transformation potential

With copper steady state in the core position in the host-pathogen interaction, as well as pathogen relies heavily on to it, targeting the steady-state equilibrium has become an attractive new direction

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of antifungal therapy. The strategies fall into two main categories: copper starvation strategies, which cut off the copper supply to the fungus, and copper poisoning strategies, which use the toxicity of copper to kill the fungus.

4.1. Copper starvation strategy

Copper starvation strategy aims to deplete copper within the host microenvironment or fungal cells by using high-affinity copper chelating agents, thereby inhibiting the activity of copper-dependent virulence factors and the basal metabolism of fungi.

The therapeutic paradigm was initially verified in cancer research. In the clinical transformation of copper starvation strategy, Tetrathiomolybdate (TM) is the most deeply studied representative copper chelator. The active component of TM, MoS_4^{2-}, forms ternary complexes with plasma Cu ions and albumin to reduce bioavailability Cu level [17]. The reutilization of TM that originally treated Wilson's disease may lay the foundation for further design of Cryptococcus neoformans combined with copper starvation strategies.

Another interesting example is antimicrobial peptides (AMPs), which are small molecular peptides with broad spectrum antimicrobial activity and widely exist in organisms. In earlier work, Silva et al. showed that the core of the anti-cryptococcal activity of tick-derived AMPs is the competitive chelation of essential fungal copper- both blocking copper-dependent laccase mediated melanin synthesis and interfering with copper-involved mitochondrial respiratory chain function. This Cu starvation strategy targets the vulnerability of fungi to Cu metabolism [18]. Anyhow, experiments with antimicrobial peptides imply that the potential of some protein compounds of natural origin may be underestimated and more comprehensive investigations are needed.

4.2. Copper poisoning strategy

Unlike copper chelators, copper ion molecules chelate metal ions in the extracellular space, which are then transported through the biofilm and released inside the cell. It exerts an antimicrobial effect with the help of increased intracellular copper concentrations.

In the family of ionophores, Disulfide (DSF) as an alcoholics drug that has been used in clinical practice for over 70 years, forms a complex with copper that can penetrate the cell membrane of pathogens. Peng et al. systematically explored the in vivo activity of DSF against Cryptococcus by means of the Galleria mellonella infection model, opening up a new path for its clinical application [19]. The Galleria mellonella model has become a preferred tool for antifungal drug screening due to its few ethical restrictions, similar immune mechanism to that of mammals, and rapid evaluation of drug efficacy. Cryptococcus neoformans strain H99 was used to infect the larvae of borocella molis, and the 7-day survival rate was observed after intraperitoneal injection. DSF monotherapy significantly improved the survival rate of infected mellonella, with a survival rate of more than 40% in the high concentration group. Notably, as a highly accessible copper ionophore, DSF has the advantage of low-cost translation in the development into a new therapy.

Chloroiodohydroxyquinoline (CLQ), chemically known as 5-chloro-7-iodo8-hydroxyquinoline, was first launched as an anti-amoeba drug and has since been widely used due to its antibacterial and anti-inflammatory effects in dermatology. de Oliveira et al. conducted a screening study among 1,600 clinical compounds in the Pharmakon library [20]. They conducted systematic susceptibility tests against 24 Cryptococcus neoformans (H99, R265 standard strains and clinical isolates). Results demonstrated that CLQ exerted potent antifungal activity against all tested strains, with MIC ranging from 0.625 to 1.25 μM. It is worth noting that as early as 1983, CLQ was forced to

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withdraw from the market due to severe neurotoxicity problems. Hence, its modified derivatives are expected to effectively reduce toxic and side effects while retaining the metal chelating properties.

4.3. Direct inhibition of copper regulatory elements

As discussed earlier, copper homeostasis is principally regulated at the transcriptional level. Cryptococcus neoformans in copper response transcription factor Cuf1 as core components show a key role in regulating the copper steady-state, a direct hit the total commander in theory is attractive. In fact, the targeting potential of TFs has been confirmed.

Bahn's classic review reviewed the pleiotropic characteristics of transcription factors of Cryptococcus neoformans and Candida albicans, respectively [21]. For example, through the full coverage deletion screening of non-essential DNA-binding TF of C. neoformans, it was found that Gat201 deletion reduced the pathogenicity of C. neoformans, demonstrating the targeting value of multi-effects. The narrow-spectrum TF target was also highlighted, a drug designed to be structurally distinct from its human counterpart. This suggests that reference the cancer field use of ubiquitin ligase degradation Cuf1, relying on DNA library high-throughput screening solutions like to intervene directly in the protein interactions may be feasible currently [22]. Likewise, metal transporters and trafficking proteins are promising drug targets. Despite this, to the knowledge of this study, fewer small-molecule inhibitors have been applied to Cryptococcus neoformans experiments in vitro, which may be due to undesired effects on the host.

5. Prospective

Although targeting copper homeostasis brings hope for antifungal therapy, there are still many challenges in the process of moving it from the laboratory to clinical application.

In summary, the greatest challenge lies in how to achieve selective killing of pathogens while avoiding severe interference with the host's own copper homeostasis. Copper is an indispensable trace element for the human body to maintain normal physiological functions and participates in key physiological processes such as the mitochondrial respiratory chain and the antioxidant defense system. Systematic use of copper chelating agents or copper ion carriers may disrupt the intricate copper metabolic network within host cells, triggering severe off-target effects. Balancing antifungal efficacy with host safety remains a key issue that needs to be addressed.

Cryptococcal meningitis (CM) poses unique therapeutic challenges that exacerbate the limitations of targeting copper homeostasis. A paradox is: even if copper homeostasis targeting drugs inhibit fungal growth in peripheral tissues, they fail to clear the fungus resident in the CNS, resulting in recurrent infections.Therefore, the development of delivery systems that can cross the BBB and target CNS fungi is a prerequisite for advancing steady-state targeted therapy for copper. First of all, through the BBB liposomes modified copper ions regulator can effectively through the BBB [23]. Additionally, fungal cell wall targeting liposomes improves the specificity of the copper ion regulator. Ambati et al. coated the β-glucan binding domain of Dectin-1 with AmB liposomes, and the affinity of this liposome for Cryptococcus neoformans was enhanced [24]. By targeted delivery, drugs could bypass the homologous proteins of host cells, reduce the systemic exposure dose and thereby minimize side effects to the greatest extent. Meanwhile, exploring a synergistic treatment strategy that combines copper homeostasis regulators with traditional antifungal drugs may delay the development of drug resistance.

Given safety management of copper ionophore therapy, organs sensitive to copper toxicity should be considered. Among them, the liver is an important organ of copper metabolism in the body.

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Referring to the early warning standard of copper deposition in hepatolenticular degeneration is helpful to avoid liver damage caused by long-term drug use. For instance, the dosing regimen can be adjusted with the help of ATP7B and Ctr1 [25]. In addition, the combination of target organ function assessment results is conducive to further optimize the drug cycle.

6. Conclusion

Mounting evidence suggests copper plays a crucial regulatory role in the pathogenesis of Cryptococcus neoformans, which is mainly reflected in pathogen copper uptake, internal environmental regulation, toxicity tolerance, and the nutritional immune response initiated by the host. Targeting the copper requirements of pathogenic fungi represents a promising avenue for antifungal therapy. Nevertheless, current therapeutic approaches centered on copper homeostasis face multiple technical challenges. As a result, the toxicity index system monitoring, targeted strategy optimization of continuous exploration, and the application of drug combinations, will bring new hope for the treatment of cryptococcal meningitis infection. Copper homeostasis, as the foundation of life activities, also plays key role in the pathogenic processes of other important pathogenic fungi. Validating and extending the targeted copper steady-state strategy for broad-spectrum antifungal therapy holds significant theoretical and translational implications.

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