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Review

Plant Derived Metabolites and Nanoparticles as a Revolutionary Strategy for Managing Human Pathogenic Fungal Biofilms

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ABSTRACT: Fungal biofilms (FBs) are a major threat to the health of immunocompromised individuals. In humans, common FB-associated infections include pulmonary aspergillosis and gingivitis, as well as skin, gut, and vaginal infections. Major human-pathogenic FBs include *Candida* spp. *Aspergillus* spp., *Cryptococcus* spp., etc. Although several medicines control infections caused by these fungi, drug resistance makes infections more fatal and requires more effective treatments. In such cases, plant-based compounds offer potentially safer and effective alternatives, with the main limitation of a short shelf life. Nanotechnology-based interventions, which enable the use of plant extracts to synthesize nanoparticles, address this drawback and support targeted, sustained delivery of plant bioactive compounds in managing FBs. In conclusion, nanotechnology holds significant potential for plant metabolites, provided frameworks and setups monitor the long-term effects of nanomaterials to ensure safe and effective use without harmful impact.

Keywords: Bioactive Compounds, Fungal Biofilms, Human Diseases, Nanomaterials, Nanotechnology.
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1. INTRODUCTION

Fungal biofilms (FBs) are a major threat to human health, and causes high mortality rates across the globe. FB-associated infections are more pronounced in immunocompromised patients suffering from chronic diseases like diabetes mellitus, liver cirrhosis, cancer, HIV/AIDS, etc. (Kuhn et al., 2002; Garvey & Rowan 2023; Janeczko & Skrzypek 2025). Additionally, patients with medical implants such as catheters, pacemakers, and artificial hip, knee, and shoulder joints are also more prone to infections caused by FBs (Kuhn et al., 2002; Garvey & Rowan 2023; Janeczko & Skrzypek 2025). Bloodstream infections inflicted by FBs are known to cause mortality of 30- 60% (Rajendran et al., 2016). During the COVID-19 pandemic, mortality increased due to the exacerbated effects of pulmonary aspergillosis (Arastehfar et al., 2020). FBs in the oral cavity cause severe conditions such as oral candidiasis, denture stomatitis, and periodontal diseases.

FBs formed by *Candida albicans* are associated with gingivitis (Aldosere et al., 2024; Di Spirito et al., 2025), and can grow together with *Streptococcus mutans* to cause severe periodontal conditions (Sangavi et al., 2025). *Aspergillus fumigatus* causes fatal pulmonary aspergillosis and also affects the immune response, worsening disease through certain epigenetic modifications (Chaves et al., 2015; Zanganeh et al., 2018; Rozaliyani et al., 2022; Rai & Rai, 2024; Shishodia et al., 2024; Guo et al., 2025). Other severe infections caused by FBs include gut, skin, and vaginal infections (Bojarska et al., 2024; Cakar et al., 2024; Wang et al., 2024b). In conclusion, the problematic FBs include species belonging to the

genera *Candida*, *Aspergillus* and *Cryptococcus* which are of wide occurrence in hospital atmosphere and can easily lead to formation of biofilms on medical devices, mucosal surfaces, and tissues (Martinez & Casadevall 2015; Rajasingham et al., 2017; Janeczko & Skrzypek 2025).

The plethora of antifungal drugs like fluconazole, echinocandins, amphotericin B, etc. available in the market is not sufficient to tackle these FBs due to the development of resistance within the fungal cells (Kuhn et al., 2002; Jabra-Rizk et al., 2004; Prasad et al., 2014; Mishra et al., 2023; Janeczko & Skrzypek 2025). FBs resist antifungals through several mechanisms involving a combination of genetic, biochemical, and physiological adaptations. FBs have a thick extracellular polymeric substance (EPS) layer that hinders antifungal drug penetration (Langford et al., 2009; Gulati et al., 2016; Dizova & Bujdakova 2017; Massey et al., 2023; Zheng et al., 2024). Moreover, FBs can form persister cells, a type of dormant cell within biofilms that reduces the effect of antifungal drugs (LaFleur et al., 2006). FBs can undergo epigenetic modifications that alter drug targets and reduce drug efficacy (Al-Sheikh et al., 2020; Wassano et al., 2020; Rao et al., 2021; Abdallah et al., 2023). Due to the development of such drug resistance, these FBs pose serious challenges in clinical settings, resulting in enhanced mortality, prolonged infections, limited treatments and higher healthcare costs (Ramage et al., 2025). Because of these mechanisms, there is a need to develop antifungal agents that can target FBs despite these resistance mechanisms.

Plants produce several bioactive compounds, categorised into major classes such as polyphenols, alkaloids, flavonoids, terpenoids, and essential oils, that show antifungal/anti-FB activity through multifaceted mechanisms (Sovljanski et al., 2023). Additionally, plant-based antifungals offer advantages such as lower toxicity to human cells, reduced likelihood of resistance development, and synergistic effects with existing antifungal drugs (Arip et al., 2022; Khwaza & Aderibigbe 2023). Additionally, medicinal plant extracts have been used to synthesize nanoparticles (NPs), which are used to manage human-pathogenic FBs. Synthesizing NPs from medicinally important plant materials is considered economically sustainable and provides formulations with extended shelf-life of bioactive compounds, a notable limitation of using plant extracts directly. These benefits make plant-based compounds and their biogenic NPs promising alternatives for developing next-generation antifungal therapies. In view of these aspects, this article summarizes information on human pathogenic FBs, enumerates plant-derived compounds and their mechanisms of action, and highlights applications of biogenic NPs against clinically relevant FBs. This information is crucial in bridging the gap between traditional medicine and contemporary therapeutic strategies, paving the way for safer, eco-friendly, and more effective antifungal interventions.

2. FUNGAL BIOFILMS: PROCESS OF FORMATION

In general, biofilms refer to structured microbial communities that adhere to surfaces and are embedded within a self-synthesized extracellular polymeric matrix (Flemming et al., 2024). These biofilms can be formed by bacteria, fungi or even mixed microbial populations (Singh et al., 2021; Bohning et al., 2024; Wang et al., 2024a). However, differences in structural constituents make FBs more complex and distinct from bacterial biofilms (Bohning et al., 2024; Wang et al., 2024a). FBs are multilayered, three-dimensional, and filamentous; their EPS matrix is composed of glucans, mannans, chitin, and proteins (Fanning et al., 2012; Guimaraes & Takahashi, 2014; Mitchell et al., 2016; Pham et al., 2020). In addition, they are highly resistant due to cell wall, EPS and persistent cells. Additionally, ergosterol-based membranes confer multidrug resistance (Davies 2003; Ruepp et al., 2004; Buzejic et al., 2016; Grujic et al., 2018). FBs fall into two categories: yeast cell biofilms and hyphal biofilms, but the formation process is largely the same, as shown in Figure 1 and explained below.

Initial attachment: In fungal biofilm formation, the first step is attachment at an interface, which depends on several factors, including nutrient supply, cell-surface proteins, surface properties of the adhering surface and microbes, and signalling cascades (Stoodley et al., 2002; Ding et al., 2025). Fungal spores or hyphae can adhere to various interfaces, including medical devices, epithelial tissues, muscular tissues, and cells. This adhesion is crucial because it allows biofilms to access external nutrients essential for growth and development (Mayer et al., 2013; Wall et al., 2019). During this stage, fungal cells also synthesize various compounds, including sugars, proteins, and lipids. These biomolecules further enhance cell adhesion, promote aggregation, and provide cohesion essential for the pre-biofilm stage (Lohse et al., 2018; de Barros et al., 2020). This synthesis further reduces mobility, a prerequisite for the next stages of biofilm development.

Proliferation and microcolony development: After initial attachment, the cells or hyphae proliferate and aggregate together to form microcolonies (Mehmood et al., 2019; Wall et al., 2019). The duration of this phase usually varies from a few hours to a day depending upon the fungal species (Alim et al., 2018; Uppuluri et al., 2018; Deng et al., 2021).

Maturation: After microcolonies develop, the next stage is encasing these colonies within EPS, which provides structural integrity and protects cells within the biofilm from abiotic stresses and even antifungal agents (Chandra et al., 2001; Siqueira & Lima 2013). Unlike the last stage, the maturation phase can last several days, during which the biofilm architecture becomes more complex and heterogeneous (de Barros et al., 2020; Sasani et al., 2021).

Dispersion: Eventually, mature biofilms undergo a phase of dispersal. In this stage, cells or cell clusters break off from established biofilms to colonize new surfaces, continuing the biofilm development cycle (Stoodley et al., 2002; Deng et al., 2021; Sasani et al., 2021).

3. FACTORS INFLUENCING FUNGAL BIOFILM DEVELOPMENT

FB formation depends on several factors, including nutrient availability, pH, and interface type (Beauvais et al., 2007; Al-Jubouri & Dapagh, 2024). The most critical factor in FB development is nutrient acquisition, which influences overall growth, virulence, and antifungal resistance (Kashyap et al., 2024). Biofilms on host tissues entrap nutrients using adhesins like ALS3 and HWP1, and they also use a holozoic nutritional mode in which they secrete hydrolytic enzymes, including proteases, phospholipases, and lipases (Sahoo & Rao, 2024). FBs are mostly dependent on host-derived iron, carbon sources, glucose, amino acids, and lipids (Alonso et al., 2023). For iron acquisition, heme oxygenases extract iron from haemoglobin, which is then transported by FTR1 and FET3 for assimilation (Al-Jubouri & Dapagh 2024; Kong et al., 2025). Some biofilms at liquid-air interfaces, such as the lungs and sinuses, use extracellular vesicles (EVs) for nutrient exchange and uptake of airborne organic matter (Soni et al., 2024; Wijeesinghe & Nobbs, 2025). Biofilm stability and nutrient retention depend on the composition of the EPS matrix, especially constituents such as β-glucans, mannoproteins, chitin, and others (Soni et al., 2024).

Abiotic factors such as pH, oxygen, and temperature are indispensable for FB development (Dishan et al., 2025; Bayer, 2025). pH affects initial fungal cell adhesion, biofilm thickness, and EPS production (Ramage et al., 2025). Most fungi prefer neutral to slightly acidic pH (6-7), while extreme pH conditions induce stress responses that ultimately hinder biofilm formation (Bage et al., 2025). On the other hand, temperature influences biofilm maturation, metabolism, and antifungal resistance. The optimal temperature for biofilm growth lies between 30-37°C, and any increase in temperature (>42°C) stimulates gene expression of heat shock proteins, which further enhance biofilm stability and antifungal resistance (Silva et al., 2022). FB metabolism has been reported to be influenced by oxygen availability in particular. Most FBs acquire resistance to antifungals under hypoxic conditions (Abdallah et al., 2014), while some may undergo growth arrest under such conditions. In conclusion, nutrient acquisition, pH, temperature, and oxygen play a critical role in regulating fungal biofilm formation, with significant differences from one species to another.

4. PLANT SECONDARY METABOLITES AS FUNGAL ANTIBIOFILM AGENTS

As discussed above, FBs pose significant health challenges in immunocompromised patients because of their high resistance to antifungal treatments (Al-Majedy et al., 2016). Because of the limitations of conventional antifungal drugs and the rise of multidrug-resistant species/strains, interest in plant-derived metabolites has grown as a potential source of FB inhibitors (Table 1). The major categories of these compounds are alkaloids, flavonoids, terpenes, and phenolics, which mostly exert their antifungal effects through anti-adhesion, membrane disruption, and hindrance of biofilm initiation (Zhan et al., 2022; Azizi & Soheilivand, 2024; Li et al., 2025). Aristolochic acid, a bioactive secondary metabolite derived from *A. indica*, has been reported to exhibit potent antifungal activity against *A. flavus,* which causes pulmonary aspergillosis. In a study, Al-Zubairi et al. (2017) demonstrated that treatment with aristolochic acid at concentrations ranging from 0.1% to 1% can lead to up to a 79% reduction in fungal hyphal growth, indicating its fungistatic and fungicidal properties. This compound is mainly reported to disrupt fungal cell wall synthesis and interfere with ergosterol synthesis, thereby compromising membrane integrity and communication. In addition, aristolochic acid may exert its antifungal effects by inducing oxidative stress-mediated apoptosis, thereby inhibiting fungal proliferation and biofilm establishment. Another equally important metabolite, catechin, extracted from plants like Camellia sinensis, Canarium patinervium, etc., has been reported to have broad-spectrum antimicrobial effects against *C. albicans* and *E. coli*. Various studies have evaluated these compounds at 25-50 mg/mL concentrations and observed significant reduction in biofilm stability, impairing microbial adhesion and structural integrity (Hirasawa & Takada 2004; Haghighi et al., 2011; Jubair et al., 2022).

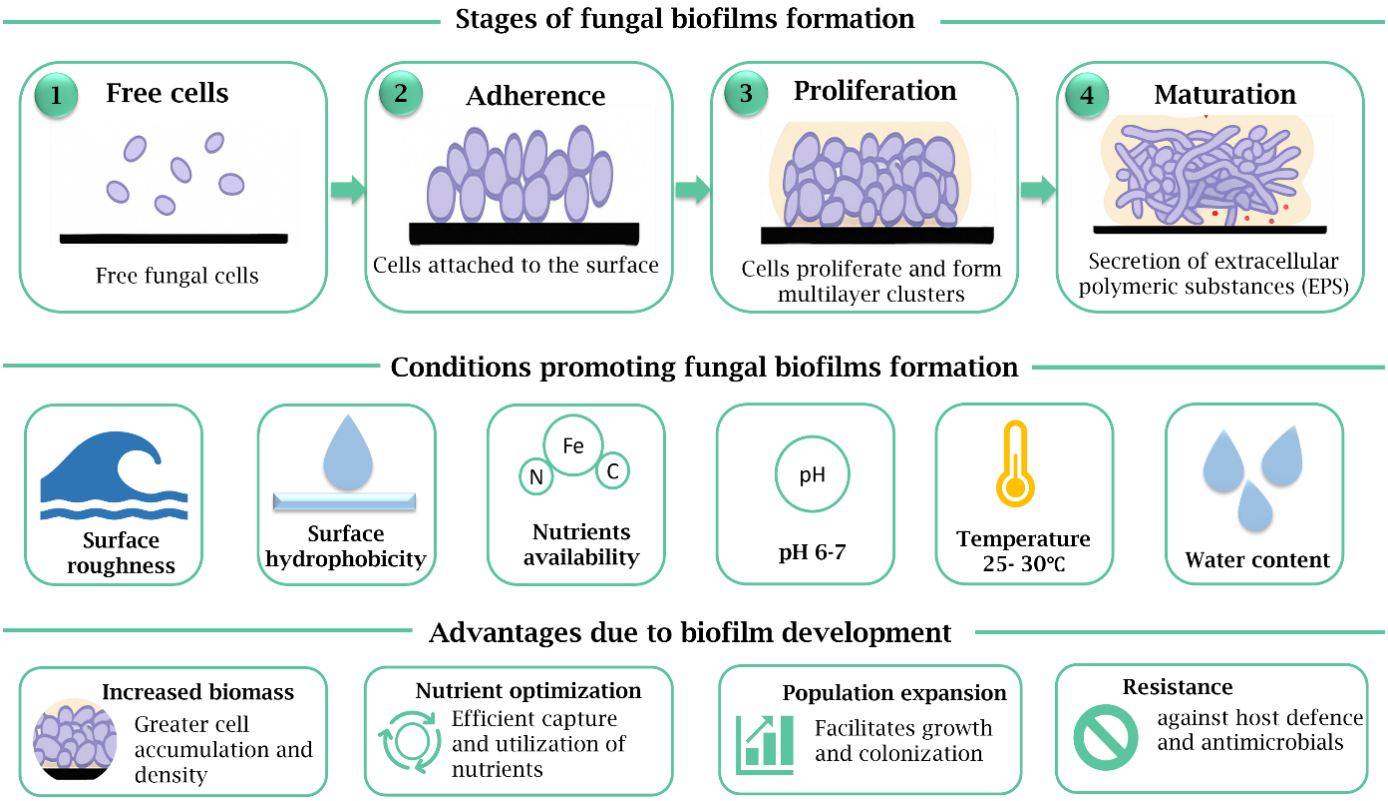

Figure 1. A schematic overview of the life cycle of human pathogenic FBs, showing the sequential stages: initial adhesion, proliferation of cells, and biofilm maturation. The middle panel summarizes key environmental and surface-related factors that facilitate biofilm progression across these developmental stages. The lower panel highlights major advantages conferred by biofilm formation.

Similar antifungal bioactive compounds, artemisinin and scopoletin, obtained from Artemisia annua, have been shown to exhibit potent antifungal activity against *Candida* spp. According to Das et al. (2020), treatment with these metabolites at concentrations ranging from 21.83 to 119.4 μg/mL significantly inhibited biofilm formation by promoting ROS accumulation, disrupting EPS integrity, weakening biofilm structure, and making it susceptible to antifungal agents. Similarly, allicin, a bioactive compound extracted from *A. sativum*, has been evaluated to exhibit significant antifungal properties against *C. albicans* biofilms. For instance, studies by Zhao et al. (2021) and Shariati et al. (2022) reported a 50% reduction in biofilm formation at concentrations ranging from 100 to 500 μg/mL. This inhibitory effect is mainly exercised by allicin's potential to inhibit the synthesis of extracellular vesicles, which play a major role in EPS formation, biofilm stability and fungal communication. Hence, by disrupting vesicle-mediated cell signalling, allicin weakens biofilm structure and increases the susceptibility of *C. albicans* to antifungal agents.

Similar to the aforementioned studies, Tubeimoside extracted from *Bolbostemma paniculatum* has been reported to have strong anti-biofilm formation activity against *S. pombe*. Liu et al. (2024) reported that at 4 μg/mL, it significantly disrupts cell wall synthesis and interferes with Prz1 signalling pathway, which regulates fungal stress adaptation. Therefore, by destabilizing biofilm architecture and increasing susceptibility to antifungal agents, these bioactive compounds offer valuable natural alternatives for controlling fungal biofilm-based infections. However, plant metabolites have limited shelf life and require optimal conditions to act (Arip et al., 2022; Khwaza & Aderibigbe, 2023; Sovljanski et al., 2023). Additionally, as shown in Table 1, we can conclude that doses of these bioactive metabolites vary depending on the source plant and the target pathogenic FB species/strain. Because of this need for more specific trials, more extensive experimentation is required, demanding more sophisticated extraction techniques for higher compound yields, which makes the use of plant-derived metabolites less economical.

Table 1: Comprehensive list of various plant-based compounds in management of common human pathogenic FBs

Plant source	Name of compound	Target pathogens	Dose	Mediated effects	References
Allium sativum	Allicin	*C. albicans*	100- 500 µg/mL	Inhibition of production of extracellular vesicles	Zhao et al. 2021; Shariati et al. 2022
Aristolochia indica	Aristolochic acid	*A. flavus*	0.1-1% (v/v)	ROS generation	Al-Zubairi et al. 2017
Artemisia annua	Artemisinin, scopoletin	*C. albicans* 1372, *C. dubliniensis* 1470, *C. tropicalis* 1368	21.83-119.4 µg/mL	Promotion of ROS, disruption in formation of EPS	Das et al. 2020
Azadirachta indica	Azadirachtin, Nimbin, Nimbdin	*C. albicans, Streptococcus mutans*	50-100 µg/mL	Disruption of quorum sensing	Priya et al. 2021
Bolbostemma paniculatum	Tubeimoside I	*Schizosaccharomyces pombe*	4 µg/mL	Disruption in cell wall formation and Prz1 signalling pathway	Liu et al. 2024
Berberis spp.	Berberine	*C. albicans* SC5314, *C. Tropicalis* ATCC 750, *C. tropicalis* strains 2203, 2317, 2006	64- 256 µg/mL	Complete inhibition of FBs	Shi et al. 2017
Camellia sinensis	Epigallocatechin Gallate, Catechin, Terpinene-4-ol	*C. albicans, E. coli*	25- 50 mg/mL	Reduced biofilm formation	Hirasawa & Takada 2004
Cannabis sativa	Cannabidiol	*C. albicans* SC5314	25- 100 µg/mL	Reduction in ATP levels	Feldman et al. 2021
Cinnamomum zeylanicum	Cinnamaldehyde	*C. albicans, A. flavus, A. fumigatus*	0.5-1% (v/v)	Disruption of biofilm initiation	Al-Zubairi et al. 2017; Rangel et al. 2018
Citrus paradisi	Citric acid	*C. albicans*	2-5 % (v/v)	Blocks initial adhesion	Ma et al. 2019
Clitoria ternatea	Ternatins, Delphinidin	*Fusarium* sp.	40- 90%	Blocks initial adhesion and cause degradation	Hujjatusnaini et al. 2025
Curcuma longa	Curcumin	*C. albicans* SC5314	200 µg/mL	Prevent the initial attachment to denture base material	Alalwan et al. 2017; Dong et al. 2021; Mustafa et al. 2024
Eucalyptus spp.	Eucarobustol E	*C. albicans* SC5314, Fluconazole-resistant *C. albicans*	>32 µg/mL	100% inhibition and eradication of FBs	Liu et al. 2017
Eucalyptus globulus	Eucalyptol	*C. albicans* 475/15, *C. albicans* ATCC 1023, *C. parapsilosis* ATCC 22019	23 mg/mL	Reduced cell viability	Ivanov et al. 2021
Gynostemma pentaphyllum	Dammarane	Flu-resistant *C. albicans* CA10 and CA16	128 µg/mL	Inhibition and reduction in biofilm formation	Liu et al. 2020

Heteroscyphus coalitus	Heteroscyphin D	*C. albicans* DSY654, *C. albicans* S5314	≥8 µg/mL	Complete prevention of biofilm formation	Wang et al. 2020
Lithospermum erythrorhizon	Shikonin	*C. albicans* SC5314	0.5 -32 µg/mL	65.4- 92.8% reduction in biofilm formation	Yan et al. 2019
Magnolia officinalis	Magnolol	*C. dubliniensis* CDC 27897, *C. albicans* CDC 27907, *C. glabrata* CDC 28621	32 µg/mL	35.6-69.5% inhibition of biofilms	Behbehani et al. 2017
Marchantia polymorpha	Plagiochin E	*C. albicans* CA2	16 µg/mL	Disrupt membrane integrity	Wu et al. 2008
Origanum vulgare	Carvacrol, Cinnamaldehyde, Citral, Menthol Thymol	*Cryptococcus neoformans* NCIM 3541, *C. laurentii* NCIM 3373	256 µg/mL	Reduced EPS formation	Kumari et al. 2017; Butassi et al. 2021
Podocarpus nagi	Nagilactone E	*Saccharomyces cerevisiae*	25- 50 µg/mL	Disrupt nutrient acquisition	Hayashi et al. 2018
Phytolacca tetramera	Phytolaccoside B	*C. albicans* ATCC 10231, *S. cerevisiae* ATCC 9763	74- 188 µg/mL	Enzyme inhibition	Escalante et al. 2008
Psidium spp.	Eucarobustol E	*C. albicans* SC5314, Fluconazole-resistant *C. albicans*	>32 µg/mL	100% inhibition and eradication of FBs	Liu et al. 2017
Rubia tinctorum	Purpurin	*Candida* spp.	1.28- 5.12 µg/mL	Alter membrane potential and efflux pumps	Kang et al. 2010
Radix glycyrrhizae	Licochalcone A	*C. albicans* SC5314, *C. albicans* ATCC 24433	4-128 µg/mL	Prevent aggregation	Messier & Grenier 2011
Scutellaria baicalensis	Baicalein	*C. albicans* ATCC 10231, *C. albicans* 5314	1-8 µg/mL	Cause apoptosis of fungal cells	Cao et al. 2008; Huang et al. 2008; Fu et al. 2011; Douka et al. 2024
Syzygium aromaticum	Eugenol	*C. albicans*	0.1-1% (v/v)	Disrupts maturation of biofilms	Butassi et al. 2021
Tritomaria quinquedentata	Ent-Isoalantolactone	*C. albicans* DSY654	8 µg/mL	Reduction in ergosterol content	Li et al. 2017
Trogonella foenum-graceum	Saponins, Steroidal Saponins	*A. niger*	10% (v/v)	Disruption in biofilm formation	Kulkarni et al. 2020
Vanilla planifolia	Vanillin	*C. albicans* SC5314	125 µg/mL	Renders ergosterol depletion	Saibabu et al. 2020
Warburgia ugandensis	Warburganal, Polygodial	*C. albicans*, *C. glabrata* ATCC 2001	10-50 µg/mL	Reduction in formation of biofilms	Kipanga et al. 2020
Zingiber officinale	6-gingerol, 8-gingerol, 6-shogaol	*C. albicans* resistant for Fluconazole	10- 100 µg/mL	85-94% reduction in biofilm formation	Lee et al. 2018

5. MECHANISMS BEHIND MANAGEMENT OF FBS BY PLANT METABOLITES

The unique properties and biological nature of plant metabolites make them efficient biofilm control agents. Plant metabolites employ several mechanisms to combat FBs, including inhibition of initial adhesion, interference with quorum sensing, disruption of EPS and cell wall, etc. As discussed in previous sections, the first phase of biofilm formation is the initial adhesion phase, a decisive step that allows pathogens to attach to available interfaces (Chauhan et al., 2024). Several plant metabolites, such as baicalein from S. baicalensis, curcumin from C. longa, and catechins from C. sinensis, interfere with adhesion proteins and cell surfaces, preventing fungal attachment (Douka et al., 2024; Mustafa et al., 2024). Next most important mechanism to inhibit fungal biofilm formation is interference with cell signalling pathways such as quorum sensing (Maggio et al., 2024). For example, eugenol from *Ocimum sanctum*, berberine from *Berberis* spp., and thymol from *T. vulgaris* disrupt quorum-sensing messengers such as farnesol, disrupting cellular communication (Ćirić et al., 2019; Iungin et al., 2024; Juszczuk-Kubiak, 2024).

Another common target of natural compounds is EPS degradation, which has been attributed to polyphenols from *C. sinensis* and terpenes from *Citrus* spp. (Wang et al., 2024a, b), ellagic acid from *P. granatum*. Another mechanism is disrupting fungal membrane integrity through allicin and artemisinin (Mahomoodally et al., 2022). In cellular fungi, yeast-to-hyphal transition is essential for biofilm development and structural stability. Alkaloids like berberine and resveratrol can inhibit morphogenetic signalling pathways such as mitogen-activated protein kinase (MAPK) signalling, impairing fungal hyphal growth (Faria et al., 2017; Bu et al., 2022; Bejenaru et al., 2024). Thus, plant-based antifungal biomolecules offer a multifaceted antifungal strategy that targets biofilm formation at various stages (Figure 2). By preventing initial adhesion, disrupting cell signalling, degrading EPS, destabilizing cell walls and inhibiting fungal growth, these compounds provide a promising natural alternative to conventional antifungal drugs.

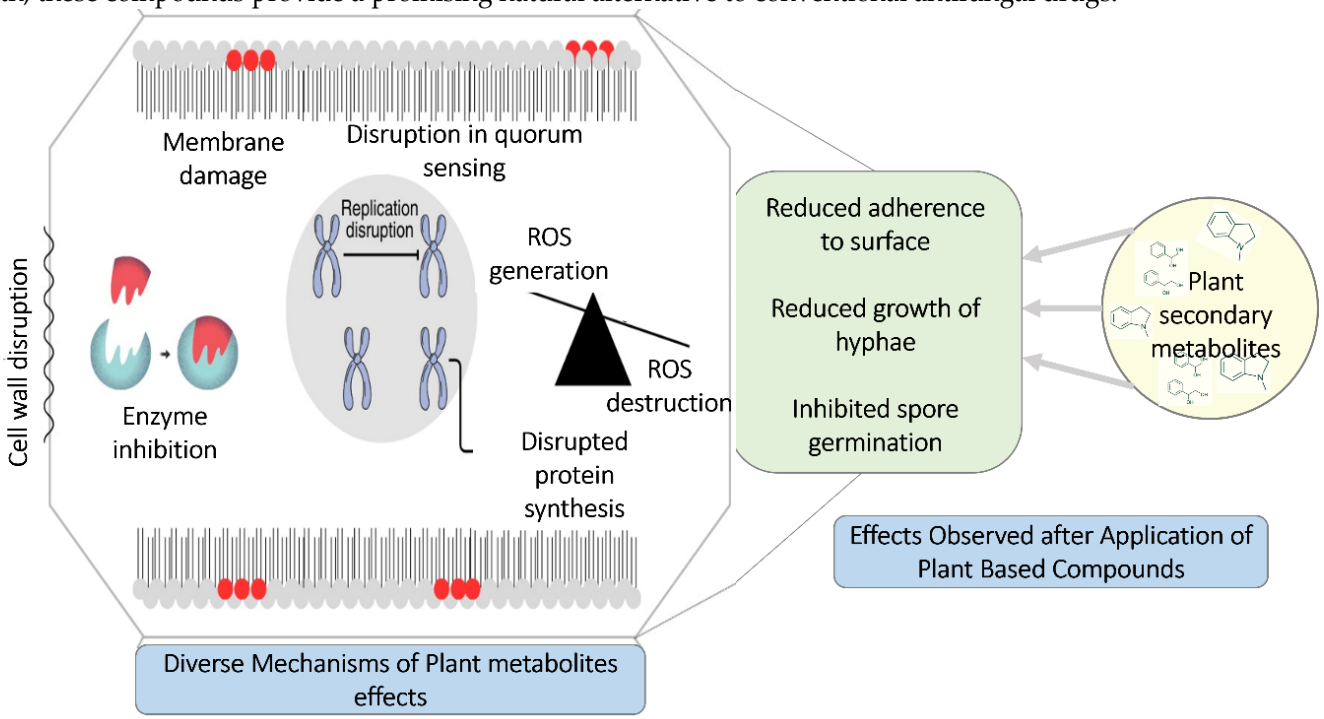

Figure 2. A schematic summary of the multiple mechanisms through which plant-derived metabolites control human pathogenic fungal biofilms. These metabolites act by generating ROS, damaging cell membranes and interfering with quorum sensing, thereby impairing biofilm maturation, reducing

6. EFFICACY OF PLANT-BASED NPS AGAINST DIFFERENT FBS

Several NPs, such as Ag NPs, CuO NPs, ZnO NPs, and Au NPs, are synthesized using a diverse range of medicinally important plants. Several researchers have described their role in managing human-pathogenic FBs (Table 2). Ag NPs synthesized using extracts from *A. indica* have shown potent antifungal activity against *A. flavus* and *F. oxysporum* at a concentration of 200 µg/mL. The proposed mechanisms include disruption of the fungal cell wall and inhibition of key enzymatic pathways, leading to growth suppression and structural damage (Latif et al., 2025). Similarly, Ag NPs synthesized using *C. sinensis* exhibit strong antifungal efficacy against multiple *Candida* species at concentrations

ranging from 125 to 250 μg/mL. Their antifungal action is primarily attributed to reactive oxygen species (ROS) generation and subsequent membrane damage in target pathogens.

Other than Ag NPs, CuO NPs synthesized using extracts from *Grewia asiatica* were reported to have significant antifungal activity against *A. niger* (Tahir et al., 2022). In another study, CuO NPs synthesized using *E. royleana* extracts showed antifungal activity against F. oxysporum at 125 μg/mL. The proposed mechanism involves electrostatic binding of metal ions to fungal membranes, disrupting membrane integrity and causing cell damage (Kumari et al., 2023). Similarly, Au NPs synthesized from O. vulgare were effective against C. albicans, with an average particle size of 28 nm, acting by disrupting spore germination and inhibiting hyphal growth (Benedec et al., 2018). Similarly, cobalt oxide NPs synthesized from *Withania coagulans* showed antifungal effects against A. niger and C. albicans at 10,000 μg/mL, indicating their potential for controlling pathogenic FBs (Hasan et al., 2020). Additionally, ZnO NPs prepared using *S. officinalis* extracts showed potent antifungal activity against *C. albicans* at 250 μg/mL. These ZnO NPs killed the fungi and markedly suppressed their growth, further supporting their role as effective antifungal agents (Abomuti et al., 2021).

Table 2: Plant mediated NPs exhibiting antibiofilm activities against human pathogenic FBs

Plants	NPs	Size (nm)	Target pathogen species/strain	Dose (μg/mL)	Mediated effects	References
Azadirachta indica	Ag	22	*A. flavus*, *F. oxysporum*	200	Cell wall disruption and enzyme inhibition	Latif et al. 2025
Camellia sinensis	Ag	52	*C. albicans*, *C. tropicalis*, *C. dubliniensis*, *C. parapsilosis*	125-250	ROS generation and membrane damage	Ali et al. 2022
Citrus limon	Ag	7-28	*C. albicans*	100	-	Khane et al. 2022
Cleome rutidosperma	Ag	15	*C. albicans*	30-50	-	Ganesh Kumar et al., 2024
Dodonaea viscosa	Ag	40-55	*C. albicans* ATCC 90028 *C. glabrata* MTCC3019 *C. tropicalis* MTCC 184	10	79–88% inhibition of biofilm growth	Muthamil et al. 2018
Eucalyptus camaldulensis	Ag	9	*C. albicans*	1	Disruption of fungal morphology and reduced cell viability	Wunnoo et al. 2022
Euphorbia royleana	Ag	20	*F. oxysporum*	400	Disruption of membrane integrity	Kumari et al. 2023
Ferulago macrocarpa	Ag	17	*C. albicans*	50-250	Reduced cell viability	Azarbani et al. 2020
Hyptis suaveolens	Ag	40-55	*C. albicans* ATCC 90028 *C. glabrata* MTCC3019 *C. tropicalis* MTCC 184	10	79–87% inhibition of biofilm formation	Muthamil et al. 2018
Jatropha curcas	Ag	47	*C. albicans* 3153A	12.14	Disrupted morphology and reduced aggregation	Kumar et al. 2017
Lannea grandis	Ag	8	*C. albicans* 3153A	10.78	Disrupted cell morphology and aggregation	Kumar et al. 2017
Lotus lalambensis	Ag	6-26	*C. albicans*	50-100	Reduced adhesion among individual cells	Abdallah & Ali 2021
Momordica charantia	Ag	5-29	*A. flavus*, *A. fumigatus*, *F. oxysporum*	-	Cell wall disruption, and enzyme inhibition	Nguyen et al. 2020

Moringa oleifera	Ag	3-5	*A. flavus, A niger*	1000	Disrupts spore germination and hyphal growth	Prajapati et al. 2023
Ocimum sanctum	Ag	50	*C. albicans, C. kefyr, A. niger, C. tropicalis, C. krusei, A. flavus, A. fumigatus*	50	Inhibits mycelial growth	Rout et al. 2012
Psidium guajava	Ag	5-53	*A. flavus, A. fumigatus, F. oxysporum*	-	Cell wall disruption and enzyme inhibition	Nguyen et al. 2020
Rosa canina	Ag	13-21	*C. albicans*	128	Inhibited hyphal growth	Gulbagca et al. 2019
Ruta graveolens	Ag	30-50	*C. albicans*	25-50	Disruption in membrane integrity	Sivakamavalli et al. 2014
Seripheidium quettense	Ag	50-96	*A. niger, A. fumigatus, A. flavus, Mucor* sp.	-	-	Nasar et al. 2019
Synedrella nodiflora	Ag	19	*Aspergillus* sp., *Penicillium* sp.	50	Reduced cell viability	Vijayan et al. 2017
Terminalia catappa	Ag	4-10	*Candida albicans*	10	Cell wall disruption and enzyme inhibition	Ansari et al. 2021
Blumea sinuata	Ag	8	*A. flavus, A. niger, F. oxysporum*	500-1500	Disruption in membrane integrity	Pradhan et al. 2025
Alhagi graecorum	Ag	22-36	*C. albicans., C. glabrata, C. krusei C. parapsilosis, C. tropicales*	50-150	Disruption in membrane integrity	Hawar et al. 2022
Teucrium polium	Ag	10-100	*F. oxysporum*	50-1500	Reduced adhesion among individual cells	Ghojavand et al. 2020
Jasminum nudiflorum	Ag	25	*A. longipes*	32	Disrupted membrane integrity	Yang et al. 2023
Synedrella nodiflora	Au	22	*Aspergillus* sp., *Penicillium* sp.	50	Reduced cell viability	Vijayan et al. 2017
Origanum vulgare	Au	52	*C. albicans* ATCC 10231	28	Disrupts spore germination and hyphae growth	Benedec et al. 2018
Celastrus paniculatus	CuO	2-10	*F. oxysporum*	-	Disruption of DNA replication and transcription	Mali et al. 2020
Withania coagulans	CoO	-	*A. niger, C. albicans*	10	-	Hasan et al. 2020
Euphorbia royleana	CuO	3	*F. oxysporum*	125	Disrupted membrane integrity	Kumari et al. 2023
Grewia asiatica	CuO	2	*A. niger*	-	-	Tahir et al. 2022
Salvia officinalis	ZnO	26	*C. albicans* SC5314	250	Reduced cell viability	Abomuti et al. 2021

Although Table 2 collates numerous reports of plant-mediated NPs with apparent antibiofilm activity against *Candida*, *Aspergillus* and *Fusarium* species, the evidence base is uneven and methodologically heterogeneous. Most

studies focus on Ag NPs and in vitro models of *C. albicans*, which very few investigations of other nanomaterials or clinically relevant non-*Candida* pathogens. The cumulative evidence shows that concentrations vary by several orders of magnitude and are often reported without standardized MIC/MFC or biofilm-specific endpoints, hampering quantitative comparison. Mechanistic descriptions are largely generic, and studies rarely integrate detailed toxicity assessment or resistance profiling. Different types of NPs, such as Ag NPs, CuO NPs, and ZnO NPs, synthesized using a variety of medicinal plant extracts, have shown broad-spectrum and potent antifungal effects against FBs. Further, diverse mechanisms of action, including cell membrane disruption, inhibition of spore germination, induction of oxidative stress, and enzyme inhibition, make them more target-specific. Although these biogenic NPs offer lower toxicity and reduced chances of resistance, a wide gap remains in understanding their detailed mechanisms of action and long-term effects on human health.

7. CONCLUSION

Diseases caused by FBs are a significant health threat, due to the increased number of resistant strains against available traditional antifungal drugs. To control such resistant strains, there is an immediate need to develop novel antifungal compounds. Plant-based secondary metabolites have the potential to cure several bacterial and fungal infections. As highlighted throughout the article, plant-based biomolecules exhibit promising antifungal activities against *C. albicans*, *A. niger*, *A. flavus*, *A. fumigatus*, etc. Despite these promising results, they face hurdles, such as expensive and time-consuming extraction processes. Moreover, some compounds show effects at low concentrations, while others require higher doses to reduce biofilm growth significantly. These factors pose challenges in practical applications, as higher concentrations may increase exploitation of plants and put certain medicinal plant species at risk of overexploitation. Building on the strong foundation of plant secondary metabolites, plant-based NPs are an emerging strategy in antifungal therapy. These biogenic NPs have been reported to show enhanced activity at lower doses. Despite these encouraging initial findings, research on plant-based NPs is still in its infancy, and much remains to be discovered. As traditional plant extracts continue to provide valuable alternatives, the field of plant-based NPs as antifungal agents will continue to evolve. Comprehensive studies focusing on the optimization, safety, and regulatory oversight of plant metabolites and plant-based NPs should be conducted. By addressing these knowledge gaps, we can fully harness the synergistic potential of both plant extracts and their nanoparticle formulations, ultimately advancing the fight against resistant FBs and improving therapeutic outcomes.

Ethical Statement

Not Applicable.

Conflicts of Interest

The authors declare no competing interests.

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