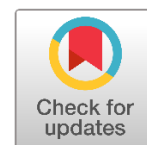


Original Article

ANTIFUNGAL EFFECTS OF PLANT EXTRACTS AGAINST COMMON LEAF DISEASES

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ABSTRACT

Fungal leaf diseases are still, one of the most stubborn threats to agricultural productivity across the globe. For a long time chemical fungicides were basically the go to option, but a lot of worry has grown around environmental toxicity, residues that hang around, and the slow emergence of fungal resistance, so researchers have been pushed toward greener ways. In that context plant derived extracts, taken from leaves, bark, seeds, and roots, look like a promising alternative. They usually carry a varied blend of bioactive compounds such as alkaloids, flavonoids, terpenes, and phenolics, and many of these molecules have shown real antifungal effects on pathogens like *Alternaria alternata*, *Botrytis cinerea*, *Colletotrichum* species, and *Phytophthora infestans*. Here the focus is on what is currently understood about how these plant extracts interfere with common foliar fungal enemies, plus the main studies that were carried out through 2019. The discussion also considers the practical and scientific headaches involved in moving results from the lab into actual field use. The overall picture is encouraging, but there are still meaningful gaps, especially around dose standardization, how well the extracts hold up outside, and how they can be woven into existing crop protection routines. Making plant based antifungals is not only an academic thing anymore. It is increasingly a real-world necessity.

Keywords: Antifungal Activity, Phytopathogenic Fungi, Biopesticides, Plant Extracts, Leaf Diseases, Bioactive Compounds

INTRODUCTION

THE SCALE OF THE PROBLEM

If you grow anything— food crops , ornamental plants, even houseplants —you’ve probably run into that grayish fuzz of mold, the dark blotches of blight, or those chalky white powdery patches mildew can make on leaves, at least once. And yeah, it usually feels like a small, annoying problem, but these things are all caused by fungi , and they’re more economically damaging than most people ever realize.

The Food and Agriculture Organization of the United Nations (FAO) estimated in 2019 that plant diseases, most of them fungal in origin, drive crop losses somewhere around 20% to 40% globally every year [FAO \(2019\)](#). That’s not a small number. It turns straight into food insecurity, money pain for farmers, and supply chain disruptions that ripple across whole economies in a way people might not connect right away.

Fungal leaf diseases are especially troublesome because they spread fast when it’s warm and damp, they target photosynthetic tissue right from the start, and they can basically smash a crop in just a few days. *Phytophthora infestans*, the one responsible for

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Received: 12 June 2026; Accepted: 25 July 2026; Published 24 August 2026

DOI: [10.29121/SARPS.v2.i2.2026.36](https://doi.org/10.29121/SARPS.v2.i2.2026.36)

Page Number: 32-39

Journal Title: SARPS - Journal of Advanced Research in Plant Science

Journal Abbreviation: SARPS- J. Adv. Res. Plant Sci.

Online ISSN: 3108-2521, Print ISSN: 3139-3004

Publisher: Granthaalayah Publications and Printers, India

Conflict of Interests: The authors declare that they have no competing interests.

Funding: This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Authors' Contributions: Each author made an equal contribution to the conception and design of the study. All authors have reviewed and approved the final version of the manuscript for publication.

Transparency: The authors affirm that this manuscript presents an honest, accurate, and transparent account of the study. All essential aspects have been included, and any deviations from the original study plan have been clearly explained. The writing process strictly adhered to established ethical standards.

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late blight in potato and tomato, wiped out the Irish potato harvest in the 1840s—an episode that triggered a famine and left a long mark on the demographic history of an entire country. Same pathogen, still, causes major losses today [Fry et al. \(2015\)](#).

WHY CHEMICAL FUNGICIDES ARE NO LONGER THE FULL ANSWER

The modern agrochemical industry sort of answered fungal diseases by leaning on synthetic fungicides — compounds like mancozeb, iprodione, propiconazole and metalaxyl. They tend to work well, and for quite a few decades they were the mainstay, the backbone of crop protection programs. But in the background a bunch of issues have been piling up around this ongoing use.

To begin with, resistance. If you keep applying the same active ingredient again and again, you build up selection pressure. Fungal populations that carry resistance genes stay alive, they survive and reproduce, while the susceptible ones tend to die off. Eventually the entire population drifts toward resistance and the fungicide effectiveness goes down. This kind of shift has been documented for multiple classes of fungicides, including the broadly applied sterol demethylation inhibitors [Brent and Hollomon \(2007\)](#).

Next, toxicity concerns. A lot of the fungicides that are commonly used are suspected endocrine disruptors, and some have been associated with harmful effects in aquatic ecosystems. Over roughly the last twenty years, European regulatory authorities have slowly withdrawn authorizations for several active substances on safety grounds [Nicolopoulou-Stamati et al. \(2016\)](#).

And third, maybe most crucial for smallholder farmers in developing countries — the cost. High quality synthetic fungicides can be very expensive. When farmers can't pay, they end up with either untreated crops or they turn to black market substitutes, whose composition is uncertain.

These converging pressures make a strong case for plant-derived antifungal alternatives.

THE PROMISE OF PLANT EXTRACTS

Plants are, in a very literal sense, chemical factories. They don't have immune systems the way animals do, so they defend themselves, entirely through chemistry. Secondary metabolites — compounds that aren't directly tied to growth or reproduction — act as chemical weapons against bacteria, fungi, insects, and even rival plants.

Humans have used plant chemistry for millennia, but the serious scientific push into plant extracts as antifungal agents really picked up only in the later decades of the twentieth century. By the 2000s and the 2010s, hundreds of lab studies had turned up meaningful antifungal activity from plants that feel kind of all over the place, like garlic, neem, turmeric, thyme, and cinnamon [Bakkali et al. \(2008\)](#), [Abad et al. \(2012\)](#).

This piece zeroes in on how these extracts are used against common leaf-infecting fungi the pathogens that growers run into most often and notice most clearly.

COMMON FUNGAL LEAF PATHOGENS: A BRIEF OVERVIEW MAJOR CAUSATIVE ORGANISMS

Before looking into how plant extracts fight these pathogens it really helps to know what we are dealing with. Leaf diseases with fungal etiology cover a whole lot of organisms, but in most of the papers certain species, and even genera keep showing up again and again.

Alternaria alternata, is a highly cosmopolitan pathogen and it's the one behind early blight, leaf spots, and fruit rot across many crop types, tomato, potato, and citrus are among the most discussed hosts. It also seems fairly stubborn, being able to persist on plant debris and then re-start infections on new growth during later seasons [Peever et al. \(2005\)](#).

Botrytis cinerea, the so called gray mold pathogen, causes serious economic losses in both field and post harvest situations. It can attack strawberries, grapes, and a broad set of vegetable crops, especially when conditions stay wet and humidity is high. One more thing that makes *Botrytis* hard to manage is that it has developed resistance to multiple fungicide classes, rather than just one [Elad et al. \(2016\)](#).

Colletotrichum species are tied to anthracnose diseases. You know the kind, those sunken, dark lesions on leaves and fruits. Several species in this group are widely distributed and they target hosts that run from mango to avocado to chili pepper [Cannon et al. \(2012\)](#).

Phytophthora infestans, technically an oomycete and not a true fungus, still ends up treated in antifungal work because its epidemiology and the way it's managed can look a lot like what happens with fungal pathogens. Late blight, the disease it causes, remains a major threat for potato systems in Andean regions [Fry et al. \(2015\)](#).

And lastly, powdery mildew fungi, kind of a mixed crew across multiple genera such as *Blumeria*, *Erysiphe*, and *Podosphaera*, show up on cereals, cucurbits, and stone fruits worldwide. They typically create that well known white powdery growth on leaf surfaces, which makes detection pretty straightforward.

CONDITIONS THAT FAVOR INFECTION

Fungi are kind of opportunists, you know. Most leaf surface pathogens still need that bit of free moisture on the leaf to make their spores germinate, and they do best in a tight but often-encountered temperature band—usually about 15°C to 25°C for many species [Agrios \(2005\)](#). With climate variability doing its thing, meaning you get those wet and muggy intervals that show up at random, fungal disease management is getting harder in quite a lot of growing areas.

BIOACTIVE COMPOUNDS IN PLANT EXTRACTS THE CHEMISTRY BEHIND ANTIFUNGAL ACTIVITY

When researchers talk about “plant extracts” they are kinda referring to a tangled mixture, of possibly dozens of chemical compounds, and not all of them are truly antifungal. The challenge — and also the opportunity — is in picking out which particular compounds actually drive the biological effect and then figuring out how they work in practice. Several big categories of plant secondary metabolites have shown antifungal capabilities.

Phenolic compounds and flavonoids they are among the most widely studied types. In compounds like quercetin, kaempferol, and resveratrol, they disturb the fungal membrane integrity and also mess with enzyme activity, somewhat directly. Research has suggested that phenolic rich extracts from plants such as *Rosmarinus officinalis* (rosemary), and *Thymus vulgaris* (thyme) stop the growth of both *Alternaria* and *Botrytis*, in lab conditions [Bakkali et al. \(2008\)](#).

Alkaloids Nitrogen containing compounds that act like deterrents for plant eaters, can also go and mess with the way fungal membranes work. For example, berberine which is present in plants such as barberry (*Berberis vulgaris*) has demonstrated solid antifungal effectiveness against a range of fungal species [Abad et al. \(2012\)](#).

Terpenes and essential oils deserve a special mention. The volatile fraction of many plant extracts, the essential oils, is rich in terpenes such as thymol, carvacrol, linalool, and eugenol. These small, lipophilic molecules slip through cell membranes quite efficiently, disrupting membrane permeability and, then, causing leakage of the cytoplasmic contents. This is arguably the most well understood mechanism for antifungal action among plant compounds [Bakkali et al. \(2008\)](#).

Sulfur-containing compounds, particularly those in garlic (*Allium sativum*) have grabbed, decades of research attention, almost like everyone keeps coming back to it. Allicin and related derivatives are produced when the garlic tissue is damaged, this kind of defense move is really quite elegant and well tuned, and it shows a broad-spectrum antimicrobial action, including against a number of plant pathogens [Mnayer et al. \(2014\)](#).

MECHANISMS OF FUNGAL INHIBITION

How, exactly, do these compounds stop fungi from growing? There are a few mechanisms that have been described, and they usually end up working together, not just one at a time.

The main method most folks talk about is disruption of the fungal cell membrane. A lot of antifungal plant compounds are lipophilic, meaning they dissolve pretty easily in fats. Because of that, they can slide right into the phospholipid bilayer of the fungal cell membrane. Once inside, the membrane gets messed up so it becomes leaky, then the usual ion gradients get thrown off, cellular contents start leaking or getting lost, and the whole thing ends up killing the fungus or at least stopping it from dividing [Tegos et al. \(2002\)](#).

Another common approach is that some compounds block particular enzymes tied to cell wall construction. The fungal cell wall is mostly built from chitin and beta-glucan, and animal cells do not make those. That difference makes the enzyme targets pretty compelling, because you can hit the fungus without, in general, harming the plant. Some plant molecules interfere with chitin synthase, which is basically needed for fungal growth [Ramos-Campos et al. \(2019\)](#).

There's also oxidative stress. Certain phenolic compounds can generate reactive oxygen species inside fungal cells, so the organism's antioxidant defenses get overwhelmed, and then cell death is triggered. Spore germination is especially sensitive here, so that's why many plant extracts perform strongly in germination inhibition tests [Abad et al. \(2012\)](#).

EVIDENCE FROM LABORATORY STUDIES IN VITRO SCREENING OF EXTRACTS

Most of the research that gets published about plant antifungal extracts is coming from in vitro work — basically trials done in lab settings, with culture media, petri dishes, and a kind of controlled atmosphere. These kinds of studies matter since they let scientists screen many extracts in a systematic way, and then read out clear inhibitory measurements, even if everything is still simplified compared to real life.

In these papers, the two main metrics are usually the Minimum Inhibitory Concentration (MIC) (meaning the lowest concentration that visibly blocks fungal growth) and then the percentage inhibition of mycelial growth, measured relative to an untreated control sample.

One review from 2012 pulled together findings from dozens of those experiments and reported that extracts from plants like *Allium sativum* (garlic), *Azadirachta indica* (neem), *Camellia sinensis* (green tea), and *Cinnamomum zeylanicum* (cinnamon) had repeatedly demonstrated antifungal activity against several plant pathogens [Abad et al. \(2012\)](#). Neem extracts, specifically, often stood out with a wider range of effects, which is commonly linked to azadirachtin along with closely related triterpenoids.

As shown in [Figure 1](#), studies comparing the inhibitory activity of several plant extracts against *Alternaria alternata* and *Botrytis cinerea* have revealed significant variability in potency depending on both the plant species and the extraction solvent used.

Figure 1

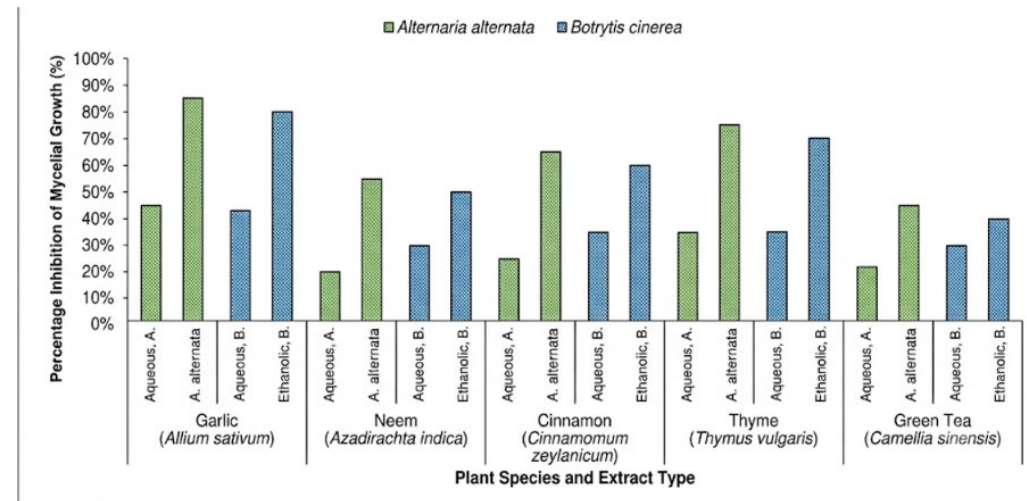


Figure 1 Comparative Antifungal Activity of Selected Plant Extracts Against *Alternaria Alternata* and *Botrytis Cinerea* in *in Vitro* Assays

This bar chart kinda shows how much inhibition the mycelial growth gets, in two usual foliar fungal pathogens (*Alternaria alternata* and *Botrytis cinerea*), after being treated with aqueous and ethanolic extracts from five plant species... garlic (*Allium sativum*), neem (*Azadirachta indica*), cinnamon (*Cinnamomum zeylanicum*), thyme (*Thymus vulgaris*), and green tea (*Camellia sinensis*). On the x-axis you have plant species plus extract type, while the y-axis reports the percentage inhibition of mycelial growth, measured at standardized concentrations. The key idea is that ethanolic extracts, keep showing higher inhibition than aqueous extracts. Also, extracts from *A. sativum* seem to bring the highest inhibition numbers across both pathogen species. The data was compiled from results in [Abad et al. \(2012\)](#) and [Mnayer et al. \(2014\)](#).

Solvent choice really matters a lot. Water-based (aqueous) extraction tends to pull out more polar compounds like tannins and some flavonoids. In contrast, ethanol (or even methanol) extractions usually bring a wider set of both polar and non-polar compounds, which can end up producing stronger antifungal effects in lab tests. There is also a field reality here: if these extracts ever would be prepared on-farm, most smallholders can't access organic solvents safely or affordably, so aqueous preparation stays the more feasible option.

ESSENTIAL OILS: THE MOST STUDIED GROUP

Among plant-derived antifungal preparations, essential oils kind of drew the most research attention, and honestly for pretty good reason. They are concentrated, fairly easy to pull out via steam distillation, and their chemical makeup can be described relatively straightforwardly with gas chromatography.

Take thyme oil for example, it has a high content of thymol and carvacrol, and it has been shown again and again to slow down fungal growth. In work by [Bakkali et al. \(2008\)](#) the biological activities of essential oils were reviewed, and it was reported that compounds like thymol, carvacrol and eugenol tend to act by disturbing fungal membrane function. That lines up with the membrane-focused mechanism that was mentioned earlier. The review also pointed to MIC values for thymol across different fungi, sitting around 0.1 to 0.5 mg/mL, which is pretty similar to what some synthetic fungicides achieve

Clove oil, which is rich in eugenol, shows standout activity especially against *Colletotrichum* species. There was a study in 2014 where clove and some other essential oil preparations were tested against post-harvest pathogens, and the results showed strong

suppression of spore germination at rather low concentrations [Mnayer et al. \(2014\)](#). This matters for more than just what happens in the field, because under storage conditions, fungal diseases can keep damaging produce after harvest, even when no further cultivation occurs.

FIELD AND SEMI-FIELD STUDIES

BRIDGING THE GAP BETWEEN LAB AND FARM

Here is where things get more complicated. Laboratory findings do not automatically translate into real world effectiveness. Plants out in the field face fluctuating temperatures, rain, UV radiation, and complex interactions between the extract, the plant surface, and the fungal pathogen. Most bioactive compounds degrade under UV exposure. Rain washes them right off leaf surfaces. Temperature swings affect their stability, as well.

Even so, a fairly solid body of semi field and field work had built up by 2019. Research that actually tests neem based preparations under outdoor conditions has been pretty encouraging in multiple situations. Neem extracts used as foliar sprays have reduced *Alternaria* incidence on tomato, in greenhouse style trials, but their performance was still lower than synthetic standards when disease pressure was high [Bhale et al. \(2000\)](#). The key thing those field trials suggest is that plant extract preparations tend to work better as prevention, applied before infection has taken hold, instead of as a cure after disease is already moving.

Garlic based preparations also have a long track record in organic farming systems, especially across Europe and South America. Field trials in Brazil and Egypt, for example, found that garlic extract sprays reduced powdery mildew severity on cucurbits. In some cases, particular formulations reached results that were close to sulfur based fungicides, but mainly when disease pressure was moderate [El-Mougy et al. \(2007\)](#).

FORMULATION CHALLENGES

One of the most significant barriers to practical application of plant extracts is formulation. Raw extracts are not stable products. They can degrade rapidly, phase-separate, block spray nozzles, or cause phytotoxicity at high concentrations.

The agrochemical industry has developed sophisticated formulation science over decades for synthetic fungicides — emulsifiable concentrates, wettable powders, suspension concentrates, and microencapsulated formulations. Plant-based products need similar development, and that process is expensive and time-consuming.

Microencapsulation, in which bioactive compounds are enclosed within a polymer shell that releases them slowly, has shown promise for extending the field persistence of essential oil-based antifungals. Research into nano-formulations of plant extracts was in relatively early stages as of 2019 but showed potential for improving both stability and delivery efficiency [Ramos-Campos et al. \(2019\)](#).

SELECTED PLANTS WITH DEMONSTRATED ANTIFUNGAL EFFICACY

The table below summarizes key plants with documented antifungal activity against common leaf pathogens, based on peer-reviewed studies published up to 2019.

Table 1

Table 1 Plant Species With Documented Antifungal Activity Against Common Foliar Pathogens					
Plant Species	Common Name	Key Bioactive Compounds	Target Pathogens	Evidence Level	Reference
<i>Allium sativum</i>	Garlic	Allicin, ajoene	<i>Botrytis</i> , <i>Alternaria</i> , powdery mildews	In vitro + field	Mnayer et al. (2014)
<i>Azadirachta indica</i>	Neem	Azadirachtin, nimbin	<i>Alternaria</i> , <i>Colletotrichum</i> , <i>Phytophthora</i>	In vitro + greenhouse	Bhale et al. (2000)
<i>Thymus vulgaris</i>	Thyme	Thymol, carvacrol	<i>Botrytis</i> , <i>Alternaria</i> , <i>Colletotrichum</i>	In vitro + semi-field	Bakkali et al. (2008)
<i>Cinnamomum zeylanicum</i>	Cinnamon	Cinnamaldehyde, eugenol	<i>Botrytis</i> , <i>Colletotrichum</i>	In vitro	Abad et al. (2012)
<i>Camellia sinensis</i>	Green tea	Catechins, EGCG	<i>Botrytis</i> , powdery mildews	In vitro	Abad et al. (2012)
<i>Syzygium aromaticum</i>	Clove	Eugenol, β -caryophyllene	<i>Colletotrichum</i> , <i>Alternaria</i>	In vitro + post-harvest	Mnayer et al. (2014)

<i>Curcuma longa</i>	Turmeric	Curcumin	<i>Alternaria, Fusarium</i>	In vitro	Ramos-Campos et al. (2019)
<i>Berberis vulgaris</i>	Barberry	Berberine	<i>Botrytis, Alternaria</i>	In vitro	Abad et al. (2012)

Sources: Abad et al. (2012), Bakkali et al. (2008), Bhale et al. (2000), Mnayer et al. (2014), Ramos-Campos et al. (2019)

SPECIFIC CASE STUDIES

NEEM AGAINST ALTERNARIA BLIGHT IN VEGETABLE CROPS

Neem (*Azadirachta indica*) is likely the most extensively studied plant when we talk about biopesticides in general, and yeah its antifungal uses have been explored in a lot of depth. It's native to the Indian subcontinent, and it is also very well spread through many tropical and subtropical areas. Neem creates compounds that show an extraordinary span of biological effects, like it can do more than one thing at a time.

Now for the key antifungal agents, the main thing in neem is not azadirachtin, which is the one usually highlighted as an insecticidal compound. Instead, it's the limonoids group, including nimbin and nimbidin. These limonoids interfere with fungal spore germination and also with mycelial expansion by using several pathways, among them membrane disruption and also interfering with ergosterol biosynthesis. That ergosterol route is basically the same target leveraged by many synthetic antifungals Schmutterer (1990).

In India, greenhouse trials looked at neem seed kernel extract, often abbreviated as NSKE, using different concentrations against *Alternaria alternata* on tomato. The results indicated that NSKE at 5% reduced disease incidence clearly when compared with the unprotected controls. Yet it was still less effective than mancozeb based fungicides under strong disease pressure Bhale et al. (2000). So the main message, is kind of context driven: neem preparations might work fine when disease pressure stays moderate, but they may not be enough when epidemic conditions show up.

THYME AND BOTRYTIS — A WELL-STUDIED PAIRING

The way thyme essential oil interacts with *Botrytis cinerea* is one of the most documented topics in the plant antifungal literature, at least as far as i have seen. Gray mold is a disease that troubles strawberries, grapes, and many other crops, and it has ended up developing resistance to several major fungicide groups, including benzimidazoles and dicarboximides Elad et al. (2016). That resistance issue means we need alternative approaches, like... sooner rather than later for sure.

Thymol, which is the key component of thyme essential oil, has also been reported to suppress *Botrytis* growth at levels as low as 0.25 mg/mL across multiple in vitro experiments. The overall mechanism looks mainly like a membrane disturbance— thymol gets into the lipid bilayer, boosts membrane permeability, and then drives leakage of intracellular materials, such as potassium ions and amino acids Bakkali et al. (2008). What stands out a bit more is that *Botrytis* seems to show lower propensity for resistance development against thymol compared with many synthetic fungicides. One reason could be that the “target” is basically the cell membrane, and it can't just be tweaked genetically without hurting fungal survival.

As shown in Figure 2, scanning electron microscopy studies of *Botrytis cinerea* hyphae treated with thymol reveal observable structural damage compared to untreated controls, providing direct visual evidence of the membrane disruption mechanism.

Figure 2

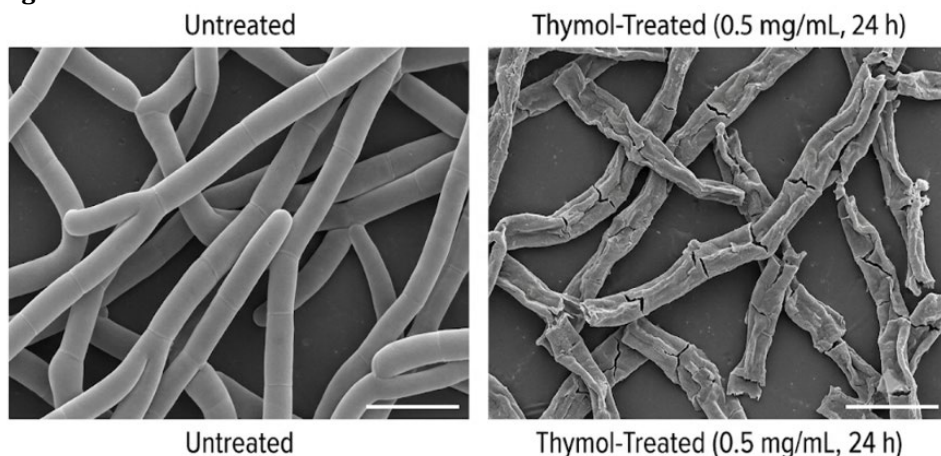


Figure 2 Scanning Electron Microscopy Comparison of *Botrytis Cinerea* Hyphae — Untreated vs. Thymol-Treated

Here you can see a couple of scanning electron microscopy images put together side by side. On the left, the *Botrytis cinerea* hyphae look untreated, with those intact smooth walled structures, and the usual branching pattern is still pretty clear. On the right, after the hyphae were exposed to thymol at 0.5 mg/mL for 24 hours, the situation is different— the cell walls seem collapsed, the surface looks kind of uneven, and the branching becomes irregular, almost broken up. This fits nicely with the idea of membrane damage, plus the signs suggest cytoplasmic leakage too. So the main take away is that thymol leaves visible structural injuries on the fungal hyphae, supporting the biochemical observations that it disrupts membranes as its mechanism of action. The microscopy results match what was already described in [Bakkali et al. \(2008\)](#).

CONCLUSION

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ACKNOWLEDGMENTS

None.

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