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Comparative Genomics of Plant and Mushroom Pathogenic Fungi for Identification of Conserved Virulence Factors

Dr. Nadia Jabeen
Department of Agriculture, Hazara University Mansehra, kpk
nadia_khan909090@yahoo.com
nadia.agri@hu.edu.pk
ORCID ID: 0000-0001-6617-8301

Dr Saba Tabasum
Assistant Professor
Department of Plant Breeding and Genetics College of Agriculture University of Sargodha
saba.tabasum@uos.edu.pk

Talha
Department of Botany, University of Makran, Panjgur
talha@uomp.edu.pk

ABSTRACT

Fungal pathogens pose severe threats to agricultural production and mushroom cultivation, yet comparative genomic analyses across these divergent pathosystems remain limited. This review synthesizes current knowledge on conserved virulence mechanisms shared between phytopathogenic and fungicolous fungi, with emphasis on genomic architectures, carbohydrate-active enzyme repertoires, effector biology, signal transduction, secondary metabolism, and emerging RNAi-based control strategies. Comparative genomics reveals that while plant pathogens possess expanded pectinolytic and cellulolytic enzyme families targeting cellulosic host walls, mushroom pathogens exhibit major expansions in chitinases (GH18), β-glucanases, and secreted peptidases tailored to dismantle fungal cell walls composed of chitin-glucan matrices. Both pathogen groups deploy LysM effector proteins that sequester chitin oligomers to suppress host immunity and protect hyphae from enzymatic hydrolysis, representing a conserved virulence strategy across kingdoms. Functional analyses demonstrate divergence in MAPK signaling requirements, with Pmk1 orthologs essential for plant infection but dispensable for mushroom pathogenicity, reflecting differences in penetration mechanisms. Secondary metabolite arsenals, particularly peptaibols and biosynthetic gene clusters, alongside expanded drug efflux transporters, facilitate niche colonization and chemical warfare. The identification of conserved, essential virulence factors enables targeted disease management through spray-induced gene silencing (SIGS), with validated targets including dicer-like genes, chitin synthases, and cell wall integrity kinases. Advanced nanocarrier delivery systems address environmental stability challenges,

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promising broad-spectrum control strategies applicable to both crop protection and mushroom cultivation. This comparative framework advances understanding of fungal virulence evolution and facilitates development of sustainable disease management approaches.

Keywords: Comparative genomics, fungal pathogens, virulence factors, mycoparasites, effector proteins, RNA interference, carbohydrate-active enzymes, signal transduction

1. Introduction

Fungal pathogens represent one of the most critical threats to agricultural production, ecosystem stability, and global food security. Historically, molecular plant pathology has focused predominantly on phytopathogenic species such as *Magnaporthe oryzae*, *Botrytis cinerea*, *Fusarium oxysporum*, and *Zymoseptoria tritici* which infect autotrophic host tissues by breaching cellulosic host cell walls, suppressing plant immune cascades, and assimilating host nutrients (Lo Presti et al., 2015). Concurrently, an equally destructive group of fungal pathogens operates within commercial mushroom production and natural ecosystems: fungicolous fungi and mycoparasites that parasitize other fungi. Major mushroom pathogens, including *Mycogone perniciosa* (the causal agent of wet bubble disease), *Lecanicillium fungicola* (dry bubble disease), *Trichoderma aggressivum* (green mold disease), and species within the genus *Cladobotryum* (cobweb disease), inflict severe yield losses on edible basidiomycete crops such as *Agaricus bisporus*, *Pleurotus ostreatus*, and *Morchella sextelata* (Gea et al., 2021).

Despite fundamental differences in host tissue composition plant cell walls being composed primarily of cellulose, hemicellulose, and pectin, whereas fungal cell walls consist of chitin, β-glucans, and mannosylated proteins phytopathogenic and mushroom-pathogenic fungi share broad infection paradigms. Both pathogen groups rely on extracellular secretomes comprising carbohydrate-active enzymes (CAZymes), surface-sensing signal transduction networks, small secreted effector proteins, toxic secondary metabolites, and drug efflux transporters to establish infection, overcome host defenses, and extract nutrients (Rovenich et al., 2014).

Recent advances in high-throughput whole-genome sequencing, transcriptomics, and phylogenomics have enabled rigorous comparative analyses across plant and mushroom pathogens (Mondo et al., 2017). Comparative genomics facilitates the identification of conserved "core" virulence factors required for fungal parasitism across divergent kingdoms, while highlighting lifestyle-specific genomic adaptations. Unraveling these shared versus specialized pathogenic mechanisms provides critical insights into the evolution of fungal virulence and informs the development of broad-spectrum biocontrol strategies, including trans-kingdom RNA interference (RNAi) applications (Wang et al., 2016).

2. Genomic Architectures and Evolutionary Dynamics Across Trophic Lifestyles

Comparative genome profiling across filamentous fungi reveals distinct evolutionary trajectories shaped by nutritional lifestyle, host range, and ecological niche adaptation. The genomic assemblies of major hypocrealean mushroom pathogens display genome sizes predominantly ranging from 34.5 Mbp to 44.0 Mbp, encoding between 8,400 and 12,300 predicted proteins (Fisher et al., 2012). For instance, *Mycogone perniciosa* possesses a genome spanning ~39.0 to

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44.0 Mbp encoding 9,276 to 10,077 predicted genes, characterized by a transposable element (TE) content reaching up to 25.3%. Similarly, *Cladobotryum protrusum* and *Cladobotryum mycophilum* exhibit high-quality genome assemblies of 38.56 to 40.7 Mbp encoding 8,428 to 12,282 protein-coding genes (Dean et al., 2012).

In contrast, phytopathogenic fungi demonstrate widely variable genomic architectures dependent on their infection strategy. Specialized biotrophic phytopathogens often maintain streamlined genomes, whereas broad-host-range necrotrophs, such as *Macrophomina phaseolina*, feature expanded assemblies (50.2 Mbp, 14,633 genes) enriched with repeat elements and duplicate gene clusters (Fletcher & Gaze, 2008). High repeat content and active transposable elements frequently generate flexible genomic regions commonly termed "two-speed genomes" where virulence factors, including effector suites and biosynthetic gene clusters (BGCs), undergo accelerated gene duplication, positive selection, and horizontal gene transfer (HGT) under host-driven selective pressures (Savary et al., 2019).

Figure 1. Evolutionary timeline, morphological diversity, and sexual life cycle of Homobasidiomycetes.

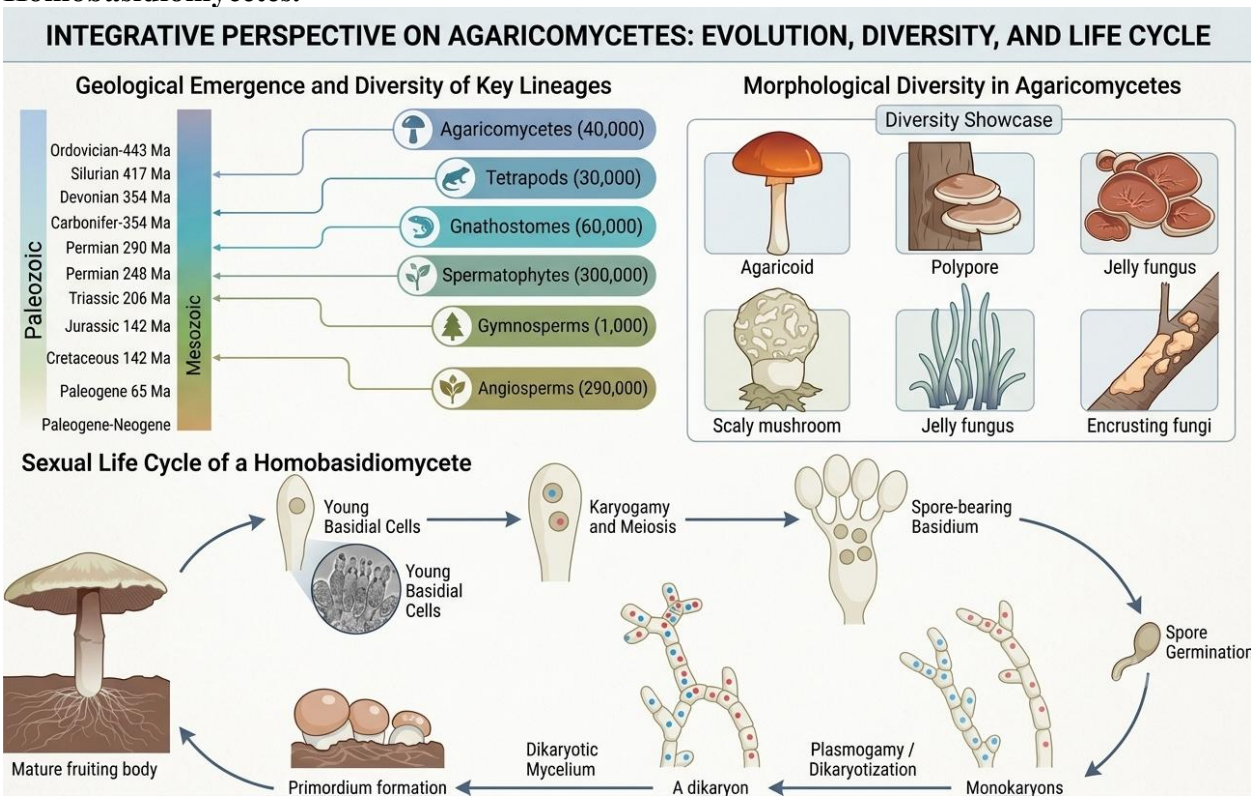

Table 1. Comparative Genomic Statistics of Representative Plant and Mushroom Pathogenic Fungi.

Pathogen Species	Target Host System	Trophic Lifestyle	Genome Size (Mb)	Predicted Genes	Key Genomic and Virulence Features

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Mycogone perniciosa	*Agaricus bisporus*	Necrotrophic Mycoparasite	39.0–44.0	9,276–10,077	Expanded GH18 chitinases (41 genes), peptidases, 25.3% TEs, ABC transporters
Trichoderma aggressivum	*Agaricus bisporus*	Competitive Mycoparasite	38.95	11,015	430 CAZymes, 970 secretome proteins, 58 BGCs, laccase *lcc2*, peptaibols
Cladobotryum protrusum	*Agaricus bisporus*, *Pleurotus* spp.	Necrotrophic Mycoparasite	39.09	11,003	Expanded secreted peptidases, 412 CAZymes, P450s, mycotoxin BGCs
Cladobotryum mycophilum	*Agaricus bisporus*, *Morchella* spp.	Necrotrophic Mycoparasite	38.56–40.7	8,428–12,282	499 CAZymes (30 GH18s), 106 BGCs, evidence of horizontal gene transfer
Clonostachys rosea	Fungal pathogens, Plant roots	Necrotrophic Mycoparasite	~38.0–40.0	~11,000–14,000	Expansions in ABC/MFS transporters, GH18 chitinases, LysM effectors
Calcarisporium cordycipiticola	*Cordyceps militaris*	Biotrophic-to-Necrotrophic	34.51	10,443	High host specificity, compact genome, reduced CAZyme count
Macrophomina phaseolina	Broad plant host range	Polyphagous Necrotroph	50.2	14,633	639 CAZymes (301 GHs, enriched

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					PL1/PL41), 73 BGCs, 1,070 secretome proteins
Metarhizium anisopliae	Insects, Rhizosphere	Entomopathogen / Endophyte	38.5	10,817	Core secreted hydrolytic secretome, cuticle-degrading proteases

Gene expansion analyses across hypocrealean mushroom pathogens indicate that strong positive selection acts on genes involved in cellular degradation, membrane transport, and secondary metabolism. In *M. perniciosa*, duplication events under positive selection are specifically concentrated within family GH18 chitinases, directly driving its specialized mycoparasitism on basidiomycete host tissue. Conversely, comparative phylogenomics demonstrates gene cluster contraction and lineage-specific losses in biotrophic mushroom pathogens such as *Calcarisporium cordycipiticola*, who's compact 34.51 Mbp genome reflects an obligate biotrophic-to-necrotrophic lifestyle tailored exclusively to its host *Cordyceps militaris* (Lo Presti et al., 2015).

3. Comparative Analysis of Carbohydrate-Active Enzyme (CAZyme) Repertoires

The carbohydrate-active enzyme (CAZyme) profile of a fungal pathogen is closely linked to the structural polysaccharides present in its host cell wall. Phytopathogenic fungi possess extensive suites of plant cell wall-degrading enzymes (CWDEs) designed to depolymerize cellulose, hemicellulose, and pectin (Kubicek et al., 2014). Pectinolytic enzymes belonging to Polysaccharide Lyase families PL1, PL3, and PL41, alongside Glycoside Hydrolase family GH28 (polygalacturonases), are markedly expanded in hemibiotrophic and necrotrophic plant pathogens such as *Macrophomina phaseolina*, *Fusarium oxysporum*, and *Phytophthora* species. In addition, cellulolytic degradation machinery including GH6 and GH7 cellobiohydrolases, GH61/AA9 lytic polysaccharide monooxygenases (LPMOs), and Carbohydrate-Binding Module family CBM1 modules ar ubiquitous among phytopathogens to facilitate outer wall penetration (Grigoriev et al., 2014).

Table 2 delineates the primary structural distinctions in CAZyme and hydrolytic enzyme repertoires between plant-infecting and fungicolous fungal pathogens (Torres et al., 2020).

Table 2. Divergent CAZyme Profiles Across Plant-Infecting and Fungicolous Pathogenic Fungi.

CAZyme / Protein Category	Dominant Subclasses / Domains	Phytopathogen Target	Mushroom Pathogen / Mycoparasite Target	Primary Functional Role in Pathogenesis
Pectinases & Polysaccharide Lyases	PL1, PL3, PL41, GH28	Pectin matrix in plant middle lamella	Absent or minimal	Breaches plant primary cell wall;

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				absent in fungal wall degradation
Cellulases & Hemicellulases	GH6, GH7, GH61/AA9, CBM1	Cellulose microfibrils and xylan	Low abundance / opportunistic	Hydrolyzes plant structural polysaccharides
Chitinases & Chitin-Binding	GH18, CBM18, CBM50 (LysM)	Fungal wall auto-remodeling / immunity	Fungal host cell wall chitin degradation & protection	Hydrolyzes fungal chitin; LysM shields pathogen hyphae
β-Glucanases	GH16, GH55, GH81	Plant glucans (minor)	β-1,3- and β-1,6-glucan matrix of host hyphae	Dissolves fungal cell wall structural matrix; drives cell lysis
Secreted Peptidases	Serine proteases, Metalloproteases	Plant structural proteins (extensins)	Outer glycoprotein layer of host fungal wall	Cleaves protective protein-mannan outer matrix of host hyphae

Mushroom pathogens and mycoparasites display a clear evolutionary shift away from pectinolytic and cellulolytic enzyme families, diverting genomic capacity toward fungal cell wall-degrading enzymes (FCWDEs). The fungal cell wall is a complex structural matrix consisting of an inner shell of chitin (β-1,4-linked N-acetylglucosamine) covalently cross-linked to β-1,3- and β-1,6-glucans, enclosed by an outer layer of heavily mannosylated proteins (Abbas et al., 2025). As a result, mushroom pathogens such as *M. perniciosa*, *C. protrusum*, *C. mycophilum*, and *Trichoderma aggressivum* feature major expansions in GH18 chitinases, GH16 and GH55 β-glucanases, and secreted peptidases (Stajich, 2017).

In *M. perniciosa*, genome-wide identification revealed 41 GH18 chitinase genes categorized into distinct phylogenetic groups based on domain architecture. Transcriptomic analysis during *Agaricus bisporus* infection demonstrated that 23 of these GH18 chitinase genes are significantly upregulated in a stage-specific manner, initiating as early as 3 days post-inoculation. Similarly, *C. mycophilum* encodes 499 total CAZymes, including 30 GH18 chitinases, exceeding the chitinolytic capacity of related non-pathogenic hypocrealean fungi (Cai et al., 2018). The concerted action of expanded GH18 chitinases, β-1,3-glucanases, and subtilisin-like serine proteases enables mushroom pathogens to systematically dismantle the structural glucan-chitin scaffold of host basidiomes, giving rise to wet bubble deformations, stipe collapse, and tissue necrosis (Zhao et al., 2013).

4. Effector Biology and Immunity Evasion Mechanisms

Beyond secretome-driven wall degradation, fungal pathogens deploy small secreted effector proteins (SSEPs) into host extracellular spaces (apoplast) or intracellular compartments to manipulate host physiology, suppress defense responses, and prevent recognition. Computational secretome analysis combining SignalP, DeepTMHMM, EffectorP 3.0, and PHI-base functional

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annotations reveals that both plant and mushroom pathogens allocate significant genetic resources to host-modifying effectors (Sahu et al., 2024).

A widespread and highly conserved effector family across fungal pathogens is the Lysin Motif (LysM) effector class. LysM domains are approximately 50-amino-acid carbohydrate-binding modules that bind chitin oligomers and peptidoglycan. In phytopathogenic fungi such as *Cladosporium fulvum* (Ecp6) and *Zymoseptoria tritici* (Mg1LysM, Mg3LysM), LysM effectors function through two main mechanisms during host colonization (Vincent et al., 2024):

First, LysM effectors suppress microbe-associated molecular pattern (MAMP)-triggered immunity through chitin oligosaccharide sequestration. During infection, host-derived chitinases hydrolyze the fungal cell wall, releasing free chitin fragments into the apoplast. LysM effectors bind these soluble chitin oligomers with ultra-high picomolar to nanomolar affinity outcompeting host immune receptors such as AtCERK1 or AtLYK5 thereby preventing chitin-induced immune activation (Presti & Kahmann, 2017). Second, LysM effectors protect fungal hyphae from enzymatic hydrolysis. Effector molecules like Mg1LysM form chitin-independent homodimers that polymerize upon binding cell wall chitin, constructing a physical protective coat over pathogen hyphae that blocks host chitinases from accessing their substrate (Rynkiewicz, 2021).

Comparative genomics confirms that LysM effectors are equally vital in fungal-fungal pathogenic interactions. The necrotrophic mycoparasite *Clonostachys rosea* encodes three functional LysM proteins. Deletion of *lysm1* and *lysm2* in *C. rosea* leads to a significant reduction in biocontrol efficacy against *Fusarium graminearum* and impairs root colonization capabilities, demonstrating that chitin-binding effectors are conserved virulence factors operating across plant, fungal, and rhizosphere interfaces (Meyer et al., 2009). Similarly, whole-genome annotation of the wet bubble pathogen *M. perniciosa* identifies conserved LysM domain proteins that are active during host parasitism on *A. bisporus*, indicating that fungicolous pathogens deploy LysM coats to resist host-derived chitinolytic defenses during basidiome invasion (Kumar et al., 2021).

In addition to LysM effectors, comparative analysis using EffectorP 3.0 differentiates apoplastic effectors (characterized by small size and enriched cysteine residues forming stabilization disulfide bonds) from cytoplasmic effectors (enriched in positively charged amino acids). While phytopathogens express diverse cytoplasmic effectors designed to target host organellar pathways, necrotrophic mushroom pathogens secrete diverse apoplastic effectors, hydrophobins, and killer toxins (e.g., KP3-like killer proteins in *Cladobotryum* species) to compromise cell membrane integrity and evade host-derived lectins (Li et al., 2019).

Figure 2. Multi-component interaction network of entomopathogenic/endophytic fungi (Metarhizium spp.) across Host, Microbiome, and Substrate interfaces.

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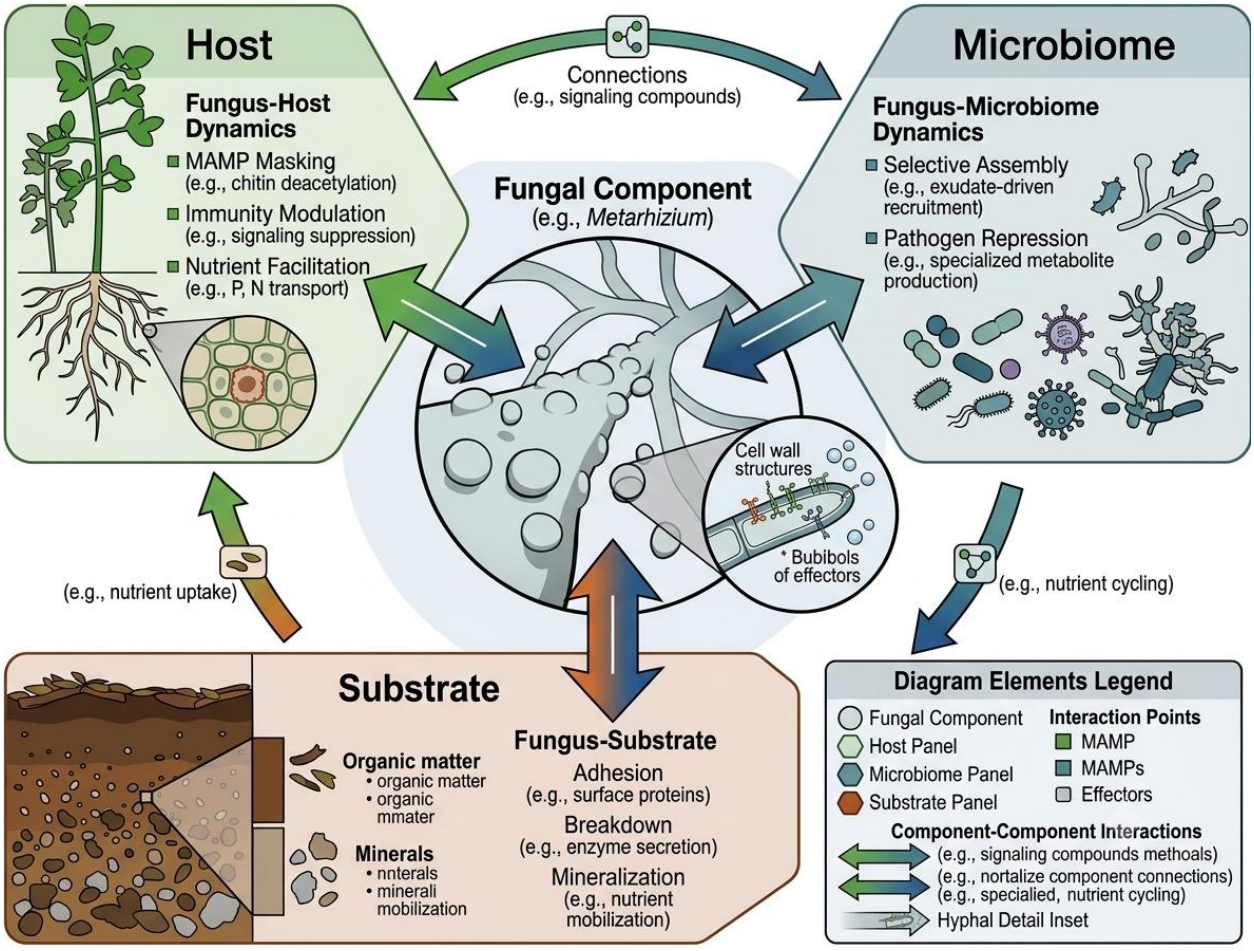

5. Signal Transduction and Regulatory Cascades: Convergence and Divergence

5.1 G-Protein Coupled Signaling and Host Recognition

Fungal sensing of host surfaces and subsequent developmental transitions require conserved intracellular signal transduction pathways. Heterotrimeric G-protein signaling complexes, cAMP-dependent Protein Kinase A (PKA) cascades, Mitogen-Activated Protein Kinase (MAPK) networks, and Cell Wall Integrity (CWI) pathways govern key stages of infection in both plant and mushroom pathogens (Zhao et al., 2007).

G-protein coupled receptors (GPCRs) located within the fungal plasma membrane perceive external stimuli, such as host surface topography, physical contact, cell wall fragments, and host secondary metabolites. In *Trichoderma* species, heterotrimeric G-protein α subunits belonging to Subgroups I and III regulate the downstream transcription of mycoparasitism-associated genes, controlling the secretion of cell wall-degrading CAZymes, the production of secondary metabolites, and the formation of infectious coiling structures or appressoria-like structures around host hyphae. In *A. bisporus–L. fungicola* interactions, physical recognition is mediated by specific

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host mushroom cell wall lectins binding to cell wall glucogalactomannans on *L. fungicola*, triggering downstream signaling cascades that initiate dry bubble disease (Zheng et al., 2000).

5.2 The MAPK Signaling Paradox: Pmk1 Divergence

A notable functional divergence revealed by comparative fungal genomics concerns the Mitogen-Activated Protein Kinase (MAPK) cascade orthologous to the *Saccharomyces cerevisiae* Fus3/Kss1 pathway. In phytopathogenic fungi, including *Magnaporthe oryzae* (Pmk1), *Botrytis cinerea* (Bmp1), and *Fusarium oxysporum* (FoPmk1), this MAPK cascade is essential for virulence. Targeted disruption of *pmk1* in plant pathogens consistently abolishes appressorium differentiation, host cuticle penetration, and invasive hyphal growth, resulting in total loss of pathogenicity (Di Pietro et al., 2001).

Functional gene disruption experiments in mushroom pathogens demonstrate that this signaling requirement is not universally conserved. Targeted deletion of the Pmk1 ortholog in *Lecanicillium fungicola* (*Lfpmk1*) revealed that *Lfpmk1* is not required for virulence on *Agaricus bisporus*. Although *Lfpmk1* transcript levels increase five-fold during cap lesion development compared to axenic culture, *Lfpmk1* knockout mutants produce typical dry bubble malformations and cap lesions, maintain wild-type growth rates, and exhibit normal sporulation on *A. bisporus* (Amey et al., 2003).

This divergence highlights fundamental differences in host tissue penetration mechanisms. Penetrating rigid, wax-coated plant cuticles require appressorium-mediated mechanical force and turgor pressure strictly orchestrated by Pmk1 signaling. In contrast, parasitizing mushroom fruiting bodies relies primarily on enzymatic wall dissolution, surface lectin-glycan interactions, and secreted toxin action, bypassing the requirement for Pmk1-dependent penetration structures (Eastwood et al., 2011).

5.3 Cell Wall Integrity Pathways

Conversely, the Slt2/Mpk1-mediated Cell Wall Integrity (CWI) MAPK pathway remains highly conserved across both plant and mushroom pathogens. In *M. oryzae*, regulators such as MoHG1 coordinate with hexokinases (MGG_00623) to modulate chitin biosynthesis, cell wall repair, and stress responses. Mushroom pathogens similarly rely on CWI pathways to maintain cell wall structure under environmental osmotic stress and to withstand host-derived defense enzymes, such as host laccases secreted by *A. bisporus* during mycoparasitic attack (Xu & Hamer, 1996).

6. Secondary Metabolite Arsenals, Toxic Peptides, and Efflux Transporters

6.1 Biosynthetic Gene Clusters and Toxic Peptides

Secondary metabolites play central roles in fungal chemical interactions. Genome mining using antiSMASH shows that both plant and mushroom pathogens assign significant genomic capacity to Non-Ribosomal Peptide Synthetases (NRPS), Polyketide Synthases (PKS), terpene synthases, and hybrid clusters. For example, the morel pathogen *Pseudodiploöspora longispora* encodes 58 secondary metabolite biosynthetic gene clusters (BGCs), including 30 NRPS-related clusters, while *Cladobotryum mycophilum* harbors up to 106 BGCs representing one of the highest cluster densities among fungicolous species (Amey et al., 2003).

A major class of non-ribosomal secondary metabolites deployed by mycoparasites and mushroom pathogens is peptaibols. Peptaibols are linear, non-proteinogenic amino acid-rich peptides

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containing α-aminoisobutyric acid (Aib) that insert into lipid bilayers to form voltage-dependent ion channels, disrupting membrane potential and inducing cellular leakage. In *P. longispora* infection of *Morchella sextelata*, peptaibols including alamethicin F-50, polysporin B, septocylindrin B, and longisporin A act as primary virulence factors (Ramachander Turaga, 2020). Direct application of purified peptaibols reproduces tissue softening and white mold symptoms on *M. sextelata* basidiomes, confirming that peptaibol-mediated membrane disruption drives tissue collapse during infection. Similarly, peptaibol production in *Trichoderma* species (e.g., trichokonins) facilitates rapid hyphal lysis of competing fungal species during mycoparasitic contact (Zeilinger, 2015).

6.2 Xenobiotic Resistance and Detoxification Machinery

Because fungal-fungal interactions involve reciprocal chemical defense, mushroom pathogens require efficient self-defense and detoxification mechanisms (Lo Presti et al., 2015). Comparative genomics reveals two main mechanisms:

First, ATP-Binding Cassette (ABC) and Major Facilitator Superfamily (MFS) drug efflux transporters are significantly expanded in the genomes of *Clonostachys rosea*, *M. perniciosa*, and *T. aggressivum*. In *C. rosea*, drug efflux pumps provide protection against host-derived mycotoxins, such as zearalenone secreted by *Fusarium* species, allowing the pathogen to survive in competitive microenvironments (Savary et al., 2019). Second, mushroom pathogens deploy enzymatic detoxification systems, including cytochrome P450 monooxygenases and laccases. In *Trichoderma aggressivum*, laccase activity encoded by the *lcc2* gene metabolizes host-derived toxins and reactive oxygen species (ROS), enhancing pathogen resistance during green mold invasion of *A. bisporus* compost (Fletcher & Gaze, 2008).

7. Emerging RNAi-Based Cross-Kingdom Control Strategies

The identification of conserved, essential virulence factors across plant and mushroom pathogens creates opportunities for targeted disease management using RNA interference (RNAi). RNAi is a conserved eukaryotic gene-silencing mechanism wherein double-stranded RNA (dsRNA) is processed by Dicer-like (DCL) enzymes into small interfering RNAs (siRNAs). These siRNAs assemble into the RNA-Induced Silencing Complex (RISC) to direct sequence-specific messenger RNA degradation or translational inhibition (Kubicek et al., 2014).

Two main RNAi application strategies have been developed: Host-Induced Gene Silencing (HIGS) involves transgenically expressing dsRNA in host plants to silence essential pathogen genes during infection. Spray-Induced Gene Silencing (SIGS) utilizes the exogenous application of synthetic dsRNA directly onto crop surfaces or mushroom casing layers. Fungal hyphae take up the environmental dsRNA, triggering target gene knockdown without requiring transgenic host modifications (Wang et al., 2016).

Comparative genomic data facilitate the selection of conserved, high-efficacy RNAi targets present across phytopathogenic and fungicolous species. Key target gene classes validated in model phytopathogens (*Botrytis cinerea*, *Fusarium graminearum*, *Verticillium dahliae*) that can be applied to mushroom pathogens include:

- Dicer-like genes (*DCL1*, *DCL2*): Silencing core RNAi machinery disrupts the pathogen's ability to generate sRNAs that suppress host defenses (Sahu et al., 2024).

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- Chitin synthases (*CHS*) and glucan synthases: Downregulating cell wall biogenesis enzymes compromises structural integrity and increases osmotic sensitivity.
- Mitogen-activated protein kinases (*MAPKs*): Silencing conserved signaling components, such as Cell Wall Integrity kinases, impairs growth and stress adaptation (Stajich, 2017).

To address challenges related to environmental dsRNA stability, nuclease degradation, and cellular uptake across thick fungal cell walls, advanced nanocarrier delivery systems are being established. Nanocarriers such as synthetic layered double hydroxide (sLDH) clay nanosheets, lipid nanoparticles, and polymer complexes protect dsRNA from degradation and provide sustained release, improving the feasibility of SIGS applications in commercial crop production and mushroom cultivation (Rovenich et al., 2014).

Conclusion

Comparative genomic analyses of plant-pathogenic and mushroom-pathogenic fungi reveal both conserved virulence strategies and lifestyle-specific adaptations shaped by fundamentally different host tissue compositions. The identification of shared virulence factors, particularly LysM effectors, expanded CAZyme repertoires targeting host cell walls, and secondary metabolite arsenals, underscores the evolutionary convergence in pathogenic strategies across phylogenetically diverse fungal lineages. Notably, while plant pathogens depend heavily on Pmk1-mediated appressorium formation for host penetration, mushroom pathogens bypass this requirement through enzymatic wall dissolution and toxin-mediated membrane disruption, highlighting critical functional divergence in infection mechanisms. The conservation of essential pathogenicity determinants across both pathosystems provides a robust foundation for developing broad-spectrum RNAi-based control strategies targeting conserved genes such as chitin synthases, MAPK components, and dicer-like enzymes. However, successful implementation of SIGS approaches requires overcoming challenges related to dsRNA stability, cellular uptake, and environmental persistence, which are being addressed through nanocarrier delivery systems. Future research should prioritize functional validation of candidate virulence genes across multiple fungal species, elucidation of effector translocation mechanisms in fungal-fungal interactions, and optimization of RNAi delivery protocols for commercial mushroom production systems. This integrated comparative framework not only advances fundamental understanding of fungal virulence evolution but also accelerates translation toward sustainable disease management strategies applicable across agricultural and horticultural systems.

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