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Review

Fungal Functional Differentiation in Grapevine Esca: From Pioneer Vascular Pathogens to White-Rot Basidiomycetes

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Abstract

Esca remains one of the most conceptually challenging disease syndromes of grapevine because it cannot be reduced to a single pathogen, lesion type or external phenotype. Foliar symptoms are erratic, internal wood damage is heterogeneous, and fungi associated with the syndrome may persist in asymptomatic vines. Much of this difficulty stems from treating esca-associated fungi as functionally equivalent, even though they occupy different niches and contribute differently to disease development. Here, we revisit grapevine esca through a fungal-biology-centred framework focused on *Phaeomoniella chlamydospora*, *Phaeoacremonium minimum* and white-rot basidiomycetes, especially *Fomitiporia mediterranea*. We argue that the esca pathosystem is best understood as the interaction between two biologically connected but non-equivalent fungal layers: a pioneer vascular phase and a later white-rot phase. Within this perspective, Petri disease and esca proper are interpreted as stages along a continuous host-fungus trajectory shaped by colonization strategy, tissue occupation, decay biology and pathobiome context. This distinction clarifies why host phenotypes are so difficult to interpret. Current evidence supports partially independent layers of host response, including resistance to pioneer colonization, tolerance to hydraulic dysfunction, resistance to wood decay and resilience under chronic infection. We therefore propose that "esca resistance" should be interpreted not as a unitary host trait, but as a multidimensional consequence of fungal functional differentiation, host physiology and environmental modulation.

Keywords: esca; grapevine trunk diseases; fungal lifestyles; pioneer vascular pathogens; white rot; *Phaeomoniella chlamydospora*; *Phaeoacremonium minimum*; *Fomitiporia mediterranea*; xylem; pathobiome

1. Introduction

Esca remains one of the most conceptually difficult trunk disease syndromes of grapevine. More than two decades after its modern redefinition, it still cannot be reduced to a single pathogen, lesion type or visible phenotype (Mugnai et al., 1999; Surico, 2009; Lecomte et al., 2012). Intermittent foliar symptoms, frequent detection of associated fungi in asymptomatic vines, and weak proportionality between internal damage and external expression argue against linear causal models. Recent syntheses instead favour pathobiome-aware frameworks in which disease expression emerges from interactions among wood-colonizing fungi, host vascular and metabolic responses, wood decay and environmental modulation (Gramaje and Eichmeier, 2026).

This distinction is important for the present review. Within the scope of esca, the fungi most consistently implicated do not represent a single functional guild. *Phaeomoniella chlamydospora* and *Phaeoacremonium minimum* are pioneer vascular pathogens shared with Petri disease of young vines and are primarily associated with xylem occupation, chronic vascular dysfunction, wound-healing

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disruption and long-term persistence within woody tissues (Gramaje and Armengol, 2011). By contrast, white-rot basidiomycetes, especially *Fomitiporia mediterranea* in Mediterranean viticulture, represent a biologically distinct layer centred on lignocellulose degradation, structural decay of the woody cylinder and advanced internal deterioration (Moretti et al., 2021). Understanding esca therefore requires more than listing associated fungi: it requires distinguishing their functional roles within the pathosystem.

This fungal differentiation also helps explain why host phenotypes are difficult to interpret. In esca, "resistance", "susceptibility", "tolerance", and sometimes "resilience" are often used loosely, although they refer to different biological properties. A vine may restrict pioneer colonization yet remain vulnerable to later white rot, or sustain chronic infection while better preserving hydraulic function or conductive renewal. Conversely, apparent "resistance" may simply reflect environmentally suppressed symptom expression. Host response is therefore informative only when interpreted against the correct fungal phase and biological process. For clarity, operational definitions of key host-response, lesion and disease-stage terms used throughout this review are provided in Table S1.

A second source of confusion has been the tendency to treat all esca-associated fungi as if they contributed in equivalent ways to disease expression. They do not. The present review is therefore deliberately restricted to *P. chlamydospora*, *P. minimum*, and white-rot basidiomycetes, especially *F. mediterranea*. This restriction is heuristic rather than reductive: it does not dismiss the broader pathobiome context of esca (Gramaje and Eichmeier, 2026), but focuses on the fungal components whose biological roles are most consistently supported across histopathological, epidemiological, and experimental studies. Within this restricted framework, we argue that the esca pathosystem is best understood as the interaction between two connected but biologically distinct fungal layers: a pioneer vascular phase and a later white-rot phase. This two-layer model should therefore be read as a fungal-biology framework embedded within, rather than opposed to, wider host-microbiome-environment interactions. This framework allows Petri disease and esca proper to be interpreted not as unrelated disorders, but as stages along a continuous host-fungus trajectory shaped by colonization strategy, tissue occupation, wood decay, and environmental modulation.

This perspective also clarifies why field susceptibility often appears unstable. The same cultivar may rank differently depending on whether the phenotype measured is pruning-wound susceptibility, lesion length after artificial inoculation, annual foliar incidence, cumulative multi-year prevalence, white-rot severity, or vine loss. Such discrepancies are not noise. They reflect the fact that different studies capture different outputs of a multilayered fungal pathosystem. A more informative question is therefore not whether a cultivar is "resistant to esca" in the abstract, but rather: resistant to which fungal phase, at which tissue level, and under which environmental filter? Reframed in this way, host variation becomes a means to interpret the distinct biological roles of pioneer vascular fungi and white-rot basidiomycetes, rather than a reason to collapse them into a single disease category.

2. Defining the Pathosystem

The esca pathosystem can be usefully defined by the coexistence of two fungal layers with distinct biological roles (Fig. 1). The first is a pioneer vascular layer, dominated by *P. chlamydospora* and *P. minimum*. The second is a white-rot layer, dominated in Mediterranean viticulture by *F. mediterranea* and, more broadly, by basidiomycetes associated with white rot (Mugnai et al., 1999; Surico, 2009; Fischer, 2006; Lecomte et al., 2012; Cloete et al., 2015). This distinction is better understood as biological rather than merely semantic. Pioneer fungi occupy xylem-associated niches, persist for long periods in vascular tissues, interact strongly with wound healing and compartmentalization, and are central to Petri disease in nursery material and young vineyards (Mugnai et al., 1999; Gramaje and Armengol, 2011). White-rot basidiomycetes, by contrast, degrade the structural matrix of the woody cylinder, alter the physicochemical environment of the trunk, and

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are more tightly associated with advanced internal decline (Mugnai et al., 1999; Cloete et al., 2015; Moretti et al., 2021; Fig. 1).

Taxonomic clarity is essential in this context. *Phaeoacremonium aleophilum* and *Togninia minima* should be treated as synonyms of *P. minimum* (Gramaje et al., 2015), otherwise older and newer evidence becomes artificially fragmented. The same applies conceptually to studies in rooted cuttings, nursery stock and rootstocks: these correspond clinically to Petri disease, but remain highly relevant because they address the same pioneer pathogens that later participate in esca.

At the same time, esca should not be expanded so broadly that it becomes indistinguishable from all Grapevine Trunk Diseases (GTDs). *Botryosphaeriaceae*, *Diaporthe* spp. and *Eutypa lata* frequently coexist in symptomatic vines and complicate field diagnosis (Gramaje et al., 2018), but their main importance for the present review is contextual. They explain why natural-infection studies often measure susceptibility to a mixed field syndrome rather than to a "pure" esca process. The purpose of this restriction is not to reduce esca to a narrow etiological model, but to distinguish the fungal functional layers most relevant for interpreting host phenotypes within a broader, context-dependent syndrome. The mechanistic core considered here remains the pioneer vascular duo plus the white-rot basidiomycete layer.

Microbiome studies have further refined this view. *P. chlamydospora* is frequently abundant even in non-necrotic tissues of asymptomatic vines (Hofstetter et al., 2012; Elena et al., 2018), which argues against using its mere presence as a disease-defining signal. By contrast, *F. mediterranea* and white-rotted tissues produce a much clearer disease-associated signature (Del Frari et al., 2019, 2021; Bruez et al., 2020). A small but informative genomic clue supports the same interpretation. The first draft genome of *P. chlamydospora* suggested a relatively modest repertoire of canonical wall-degrading enzymes and secondary-metabolite genes compared with strongly wood-degrading fungi, consistent with a biology of chronic vascular colonization rather than large-scale structural decomposition (Antonielli et al., 2014). This does not make the species biologically minor; rather, it reinforces its interpretation as a persistent pioneer vascular pathogen.

3. From Petri Disease to Esca Proper: A Continuum

Petri disease and esca proper can be usefully understood as a continuum, even though they are not clinically identical. Petri disease describes early infection of young plants by pioneer vascular fungi, typically associated with poor establishment, vascular discoloration and reduced vigour (Gramaje and Armengol, 2011). Esca proper describes the chronic mature-vine condition in which vascular lesions, internal necroses and often white rot accumulate in an ageing woody axis and may become associated with foliar symptoms and long-term decline (Lecomte et al., 2012).

The continuity is biological, not necessarily symptomatic. Young vines infected by *P. chlamydospora* or *P. minimum* may remain externally silent for long periods (Hrycan et al., 2020). Mature vines may carry these fungi for years while expressing foliar symptoms only intermittently (Dewasme et al., 2022). Some symptomatic vines mainly show vascular discoloration, while others accumulate extensive white rot. Nevertheless, the same pioneer fungi recur in both Petri disease and esca-associated wood lesions, and infection introduced early through propagation material or wounds can persist through the life of the plant.

Field epidemiology supports this long view. Romanazzi et al. (2009) showed that annual symptom incidence in young vineyards can be low while cumulative multi-year incidence becomes much higher, indicating that symptom absence in a given year does not equal absence of the disease process. In mature vineyards, the same principle applies: annual expression is erratic, whereas cumulative prevalence reveals that many vines eventually enter the syndrome.

Anatomically, the continuum is visible in the progressive transformation of the woody cylinder. Kassemeyer et al. (2022) distinguished two lesion types that are complementary rather than mutually exclusive: brown wood streaking linked to *P. chlamydospora*, involving localized groups of vessels, fibres and parenchyma with tyloses and dark deposits, and white rot linked to *F. mediterranea*,

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involving demarcation zones and progressive destruction of fibre and vessel walls. These lesion types represent different histopathological layers that can coexist in the same trunk.

Experimental studies reinforce the same logic. Artificial inoculation with *P. chlamydospora* and *P. minimum* usually reproduces wood symptoms, vascular streaking and reduced vigour, but the grapevine only rarely expresses the full mature esca phenotype over short time scales (Eskalen et al., 2001; Feliciano et al., 2004; Gramaje et al., 2010; Travadon et al., 2013). This does not weaken their role; it clarifies it. They are strong models of the early vascular phase, but incomplete models of the mature syndrome. Basidiomycete inoculations, conversely, can generate wood lesions and even esca-like foliar features under some conditions, but still without reproducing the full chronic field phenotype unless broader co-infection or tissue context is present (Brown et al., 2019). A coherent interpretation is therefore that Petri disease and esca proper lie along the same chronic infection trajectory, but that mature esca includes an added structural layer associated with wood decay.

4. Host Variation and Phenotyping of Resistance

Host variation in esca is real, but its meaning depends on what is measured. Multi-year field studies established cultivar differences in apparent susceptibility (Marchi, 2001; Murolo and Romanazzi, 2014), and recent work has made this evidence more robust. In a common garden vineyard experiment including 46 cultivars, Gastou et al. (2024) demonstrated stable cultivar-dependent differences in esca expression. Etienne et al. (2026), using two decades of surveys, similarly showed strong cultivar effects on prevalence, despite major contributions of vineyard age and year. Lecomte et al. (2024) further showed that susceptibility has a temporal component, with cultivars differing not only in total symptom incidence but also in the kinetics and seasonal probability of symptom emergence.

Yet susceptibility observed in the field is not a unitary phenotype. It integrates chronic infection history, white rot, hydraulic condition, climate and management, not simply direct incompatibility with a pathogen. This is why natural field rankings often diverge from inoculation rankings. Chacón-Vozmediano et al. (2021), under natural infection in a Spanish germplasm collection, identified substantial cultivar differences, but these rankings reflected the full GTD field reality. By contrast, inoculation studies with *P. chlamydospora* measure resistance to pioneer vascular infection rather than to full esca expression.

This distinction is important. Martínez-Diz et al. (2019) showed that all cultivars inoculated with *P. chlamydospora* developed vascular lesions, indicating absence of qualitative immunity. What varied was lesion extent, that is, quantitative restriction of damage. Sofia et al. (2018) and Markakis et al. (2017) reached related conclusions in Portuguese and Greek germplasm, respectively. These are genuine resistance traits, but they concern pioneer vascular susceptibility, not the whole syndrome.

A further refinement is the distinction between resistance and tolerance. García-García et al. (2024) showed that cultivar Pardina appeared more tolerant than cultivar Tempranillo to esca-related challenge. Crucially, Pardina did not dramatically suppress *P. chlamydospora* multiplication; instead, it suffered less structural and physiological damage and activated an earlier host response. This is one of the clearest demonstrations that tolerance and resistance must be distinguished in esca. Genetics strengthens this section further. Arnold et al. (2025) identified Esca Necrosis Susceptibility 1 (ENS1), a major locus associated with necrosis-related susceptibility in mature field-grown vines. ENS1 does not resolve the genetics of esca resistance, but it shows that internal wood susceptibility can be mapped genetically under realistic conditions. This represents an important conceptual step.

Host variation also exists below the cultivar level. Clone-dependent esca expression and metabolomic responses have been reported in both Chardonnay and Trousseau cultivars, showing that symptom-associated signatures are strongly shaped by genotype and vintage rather than reflecting a single stable disease fingerprint (Moret et al., 2019, 2021). An additional unresolved dimension is the contribution of rootstock-scion interaction to these response layers. Existing evidence indicates that rootstock xylem anatomy can influence susceptibility to *P. minimum* and *P. chlamydospora* (Ramsing et al., 2021), but we still lack robust experimental data on whether particular

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rootstock-scion combinations show non-additive effects on pioneer colonization, hydraulic tolerance, white-rot susceptibility or long-term esca expression. This remains a priority both for mechanistic work and for applied breeding and rootstock selection.

For this reason, resistance phenotyping in esca should be stratified into at least six levels: susceptibility to pioneer vascular colonization, severity of internal necrosis, white-rot susceptibility, seasonal timing of foliar expression, tolerance to physiological dysfunction, and long-term decline or vine loss (Fig. 2).

5. Structural Resistance to Pioneer Vascular Pathogens

The most consistently proposed structural candidate for resistance to pioneer fungi is xylem architecture. A recurrent hypothesis in the literature is that wider vessels facilitate pathogen spread and increase the hydraulic cost of defence, whereas narrower and more numerous vessels favour compartmentalization. Pouzoulet et al. (2017, 2020) gave this idea its clearest formulation, linking vessel diameter and vessel density to susceptibility to esca-associated fungi. Ramsing et al. (2021) extended the same logic to rootstocks, showing that susceptibility to *P. minimum* and *P. chlamydospora* correlated with xylem anatomy, with wider vessels associated with greater colonization. Gerin et al. (2024) reinforced the anatomical layer across a broader set of genotypes.

The attraction of this model lies in its ability to connect fungal spread and host function. Xylem design is always a trade-off between transport efficiency and safety. Wide vessels maximize conductance under normal conditions, but they are also potentially more costly to isolate during infection. In esca-associated pioneer infections, this trade-off is central because tyloses, gums and dark deposits may both restrict fungal advance and compromise hydraulic continuity (Troccoli et al., 2001).

However, anatomy cannot be treated as a universal explanation of esca. Foglia et al. (2022), examining 51 cultivars under the same field conditions, found strong differences in xylem traits but no simple linear relationship between vessel size and field incidence of esca. This provides an important corrective. It suggests that anatomy predicts pioneer spread and internal damage more directly than erratic annual foliar symptom incidence.

A recent common garden physiopathological analysis also tempers overly deterministic interpretations of xylem anatomy (Gastou et al., 2025b). Across cultivars spanning a broad susceptibility gradient, several constitutive stem traits were not significantly correlated with cultivar mean esca susceptibility despite substantial genotypic variation. Together with the lack of a simple relationship between vessel size and field incidence reported elsewhere, this suggests that constitutive stem anatomy may shape local pioneer colonization and vascular damage, but does not by itself explain whole-plant susceptibility or the transition from latent infection to visible esca expression.

Histological work clarifies what structural resistance actually looks like. Troccoli et al. (2001) showed that *P. chlamydospora* progresses slowly through xylem and that its advance is accompanied by tyloses, phenolic accumulation and deposition of defensive materials. Pierron et al. (2016) demonstrated that wound response itself is strong, but that *P. chlamydospora* specifically disrupts wound healing and reduces new xylem formation more than *P. minimum*. Structural resistance therefore includes both pre-existing xylem architecture and the capacity of wood to rebuild conductive continuity after wounding and infection.

Kassemeyer et al. (2022) further bridge resistance and lesion anatomy by showing that brown wood streaking linked to *P. chlamydospora* involves localized vessel groups, fibres and parenchyma with tyloses and dark inclusions, whereas white rot reflects a much more destructive lesion pattern. The early structural struggle in esca is thus a struggle over vascular integrity and compartmentalization. In the pioneer phase, structural resistance may be usefully defined as the capacity to contain fungi spatially while maintaining enough xylem continuity to avoid progressive functional decline. Yet the later transition from chronic vascular occupation to whole-plant

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dysfunction appears to depend less on anatomy alone than on how host physiology, lesion development and pathobiome state become configured over time.

6. Chemical Resistance of the Wood: Constitutive and Induced Components

Wood susceptibility in esca cannot be understood through anatomy alone. The chemical environment of the trunk is equally important, and here a distinction must be made between constitutive chemistry and induced defence chemistry.

Constitutive chemistry appears particularly relevant to white rot. Gastou et al. (2025a) showed that cultivar susceptibility aligned more strongly with wood composition than with the mycobiome of healthy wood, implicating extractives and structural fractions such as hemicellulose in the predisposition of wood to decay. Schilling et al. (2022) complemented this from the fungal side by showing that *F. mediterranea* degrades grapevine wood more efficiently than non-host hardwood, while mobilizing oxidoreductases and removing grapevine extractives, including stilbenes and flavonoids. Puca et al. (2025) further broadened this view by showing that several esca-associated white-rot basidiomycetes share the capacity to activate chelator-mediated Fenton-like pathways, suggesting that non-enzymatic radical chemistry is likely widespread in this system.

Induced chemistry appears more closely linked to the pioneer vascular layer. Amalfitano et al. (2000) showed that brown-red wood accumulates trans-resveratrol and ε-viniferin, demonstrating that this lesion type is not merely passive discoloration but also a site of phytoalexin accumulation. Del Río et al. (2004) added a mechanistic dimension by showing that grapevine phenolics inhibit fungal ligninolytic enzymes, suppress fungal growth and sporulation, and are associated with improved vascular recovery when phenolic synthesis is stimulated. These data support the view that phenolics in esca act as defence components rather than merely as markers of stress. Proteomic and transcript-metabolite studies point in the same direction. In grapevine trunks affected by esca proper or apoplexy, both asymptomatic and black-streaked wood showed reprogramming of stress-, antioxidant-, pathogenesis-related and phenylpropanoid-related functions, together with shifts in stilbene and phenolic accumulation, indicating that induced defence chemistry extends beyond visibly altered tissues (Magnin-Robert et al., 2014, 2016).

Additional studies broadened the chemical repertoire. Rusjan et al. (2017) showed spatial heterogeneity of responses. Pierron et al. (2016) linked induced chemistry to early activation of phenylalanine ammonia-lyase (PAL) and stilbene synthase (STS), while Romeo-Oliván et al. (2024) showed that induced signatures differ between *P. minimum* and *P. chlamydospora*. Vásquez-Ocmín et al. (2026) expanded the detectable response beyond classic phenolics by identifying lipid-related signatures and candidate oxylipin signals.

This broader chemistry is especially important from a fungal biology perspective. Basidiomycete white-rot fungi are not only confronted with host extractives; they actively detoxify them. Pioneer fungi, in turn, encounter a chemically fortified wound environment that may slow or redirect colonization. Thus, constitutive and induced chemistry interact dynamically with fungal strategy. Resistance at this level is not simply "high stilbenes" or "more phenolics", but the combination of a wood chemistry difficult to degrade and an inducible local chemistry able to constrain infection.

7. Early Host Responses and Pathogen-Specific Signalling

One of the strongest corrections to older views of esca is the realization that woody tissues actively perceive fungal challenge. The trunk is not a chemically inert cylinder that merely scars after invasion. Pierron et al. (2016) provided one of the clearest demonstrations of this principle: wounding alone induces a strong response, but the response changes depending on whether the wound contains *P. minimum*, *P. chlamydospora*, or both. The host is therefore not responding only to injury. It is responding to fungal identity.

The timing of that response is particularly informative. In Pierron et al. (2016), *P. minimum* elicited a clearer early response, whereas *P. chlamydospora* later exerted a stronger effect on wound

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closure and new xylem formation. Romeo-Oliván et al. (2024) reinforce this interpretation with local transcriptomic and metabolomic data showing that the host response differs in both timing and quality depending on the pioneer pathogen. This is important because it suggests that early resistance in wood is at least partly a matter of pathogen-specific signalling.

This is where Ferrandino et al. (2023) are conceptually useful: they place these early responses within a broader grapevine framework of transcriptional, metabolic and hormonal crosstalk. In this view, pioneer infection is not just a localized insult but an entry point into wider regulatory networks involving phenylpropanoid metabolism, stress hormones and defence signalling. Earlier work also supports the idea that lower susceptibility may depend on the speed and amplitude of defence activation. Using a leaf elicitation system based on *P. chlamydospora* culture filtrate, Lambert et al. (2013) showed that less susceptible grapevine cultivars displayed earlier and stronger induction of phenylpropanoid- and pathogenesis-related (PR) genes, together with greater accumulation of stilbenoid-associated defences, than a more susceptible cultivar. Although this system does not reproduce the full wood pathosystem, it supports the broader view that timing and intensity of host response are likely to be important components of apparent resistance. Rakotoniaina et al. (2025) provide a complementary proof-of-concept from a different tissue system. Their stomatal model showed that *P. chlamydospora* mycelium and cell-wall fractions induce stomatal closure, at least partly through ethylene signalling, whereas fungal extracellular compounds can instead promote opening. Although this is not a trunk model, it is conceptually relevant because it supports the idea that pioneer fungi both trigger and modulate host defence, potentially through distinct molecular fractions.

A main insight of this section is that early host response in esca can be viewed as a decision layer. It is where wound response, fungal identity and fungal interaction first begin to shape the later course of infection. If that response is efficient, colonization may remain restricted. If it is delayed, misdirected or excessively costly, chronic occupation and later dysfunction become more likely.

8. *Phaeomoniella chlamydospora* Versus *Phaeoacremonium minimum*: Same Syndrome, Different Biology

Although *P. chlamydospora* and *P. minimum* frequently co-occur in esca-associated wood, current evidence indicates that they are not biologically equivalent (Table 1). Valtaud et al. (2009) provided one of the clearest mechanistic distinctions. Under their conditions, *P. minimum* penetrated lignified xylem walls, formed cavity-like alterations in the secondary wall, and caused substantially greater wood mass loss than *P. chlamydospora*. Del Río et al. (2004) similarly detected ligninolytic-related enzymatic activities in *P. minimum* but not in *P. chlamydospora* in their assays. This does not make *P. minimum* a white-rot fungus, but it does support a more active role in wall alteration.

By contrast, *P. chlamydospora* is repeatedly associated with slow vascular colonization, strong local host response, and interference with wound closure (Troccoli et al., 2001; Pierron et al., 2016). The first draft genome of *P. chlamydospora* fits this interpretation well, suggesting a relatively modest canonical degradative arsenal compared with more aggressive wood degraders (Antonielli et al., 2014). Its biology is therefore more consistent with that of a chronic pioneer vascular colonizer than with that of a major decomposer.

The host also perceives the two fungi differently. Pierron et al. (2016) and Romeo-Oliván et al. (2024) both show that wood responses differ depending on whether the inoculum is *P. minimum* or *P. chlamydospora*. Even susceptibility patterns in rootstock and cutting assays are not perfectly overlapping, suggesting that the same host architecture does not constrain both fungi in exactly the same way (Markakis et al., 2017; Martínez-Diz et al., 2019; Ramsing et al., 2021).

The fungal side is also asymmetric in genomic plasticity. Massonnet et al. (2018) showed that *P. minimum* exhibits substantial structural variation among isolates, especially in biosynthetic gene clusters associated with secondary metabolism. Comparative pangenomics placed metabolite-related clusters and transporters in the dynamic fraction of the *P. minimum* pangenome (Garcia et al., 2024). This suggests that chemical plasticity may represent an important axis of adaptation in *P. minimum*.

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Taken together, the evidence points to a functional contrast within the pioneer vascular phase. *P. chlamydospora* is more strongly associated with chronic vascular occupation, altered wound healing and localized dysfunction, whereas *P. minimum* appears more chemically and structurally versatile, with clearer wall-altering potential and greater genomic variability in metabolite-related functions. Both are pioneer fungi, but they are not functionally redundant. The main contrasting biological features of these two pioneer vascular pathogens are summarized in Table 1.

9. White-Rot Basidiomycetes as a Distinct Second Layer of Susceptibility

White-rot basidiomycetes introduce a biologically distinct dimension to esca. They are not merely signs of advanced deterioration, nor passive successors of pioneer infection. Their significance lies in their ability to attack the structural matrix of the woody cylinder and thereby redefine host susceptibility.

Del Frari et al. (2021) argued persuasively that foliar esca is better explained when white rot and *F. mediterranea* are treated as central components rather than as secondary background factors. This view is supported by both microbial and anatomical evidence. Bruez et al. (2020) found that the clearest microbial signal of diseased wood emerged in white-rotted tissues, where *F. mediterranea* dominated a simplified fungal consortium. Elena et al. (2018) similarly linked *F. mediterranea* mainly to white-rot tissues rather than to healthy wood. Kassemeyer et al. (2022) provided the anatomical counterpart by showing that white rot follows a distinct pattern of wall degradation and tissue collapse.

The biology of *F. mediterranea* further supports its importance in this context. Schilling et al. (2022) demonstrated that the fungus is particularly efficient on grapevine wood, combining simultaneous degradation of structural polymers with detoxification of host extractives. Puca et al. (2025) further showed that several esca-associated basidiomycetes share the capacity to activate chelator-mediated Fenton-like pathways, implying that non-enzymatic radical chemistry is probably widespread among white-rot agents in this system. Brown et al. (2019) then challenged the older notion that basidiomycetes require prior pioneer infection before becoming true pathogens. In their system, several basidiomycetes caused wood lesions on their own, and some combinations with *P. chlamydospora* increased the frequency of esca-like foliar symptoms. Although these experiments do not reproduce the full mature field syndrome, they show that esca-associated basidiomycetes can act as autonomous wood pathogens.

From the host perspective, susceptibility to white rot is not the same as susceptibility to pioneer fungi. Gastou et al. (2025a) suggest that cultivar differences in esca susceptibility are linked more strongly to wood chemistry and degradability than to the microbiome of healthy wood. This interpretation fits well with Schilling et al. (2022), in which host extractives are direct fungal targets.

White rot is therefore best treated as a distinct second layer of susceptibility: the vulnerability of the woody substrate to basidiomycete decay, and the capacity of fungi such as *F. mediterranea* to neutralize host defences and degrade lignocellulose. A key unresolved issue is causality. Current evidence supports a closer relationship between white rot, *F. mediterranea* activity and foliar symptom expression than between pioneer vascular colonization alone and visible disease, but the mechanistic chain remains incompletely demonstrated. A plausible working model is that decay-driven loss of wood integrity and xylem continuity amplifies hydraulic dysfunction, favours vessel occlusion and increases the probability of foliar symptom expression. Testing this model will require controlled longitudinal systems combining basidiomycete inoculation, lesion tracking, hydraulic monitoring and temporal symptom recording, ideally in designs that also distinguish the contribution of pioneer fungi from that of decay fungi.

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10. Co-Infection, Succession, Pathobiome and Environmental Modulation of Apparent Resistance

A characteristic feature of esca is the weak and inconsistent correspondence between internal infection and external symptom expression (Fig. 3). This discrepancy cannot be understood without considering co-infection, tissue succession, pathobiome structure, hydraulics and environmental modulation together. In non-necrotic or apparently healthy wood, fungal communities often differ more according to microhabitat, tissue age or terroir than according to symptom status, as shown by Del Frari et al. (2019), Elena et al. (2018) and Geiger et al. (2022). By contrast, the clearest microbial signal emerges in necrotic tissues, especially white rot, where Bruez et al. (2020) identified a highly specific consortium dominated by *F. mediterranea* and *P. chlamydospora*. This may help explain why bulk surveys of wood communities often fail to clearly distinguish symptomatic from asymptomatic vines.

Pathobiome therefore matters, but not as a synonym for richness. What matters is which organisms coexist within a given niche and how that coexistence influences fungal activity, metabolite production and host response. Travadon et al. (2016) showed that community composition explained necrosis better than richness alone, and Karácsony et al. (2023) further suggested that even taxa usually regarded as neutral or beneficial, such as *Aureobasidium pullulans*, may alter symptom expression through interaction with *P. chlamydospora*. Apparent resistance is thus shaped not only by which fungi are present, but also by how host and fungal processes are configured within particular tissues.

Environmental forcing adds a further layer of complexity. Calvo-Garrido et al. (2021) linked lower water availability and reduced transpiration to greater symptom expression, while Calzarano et al. (2018) and Monod et al. (2025) showed that climate and soil context also influence symptom emergence. Gastou et al. (2026) sharpened this point by showing that drought and esca expression do not affect the fungal component of the vine in the same way: drought primarily altered Ascomycota communities in apparently healthy trunk wood, whereas esca expression more clearly affected young organs, and the strongest community shifts remained associated with necrotic tissues. Crucially, in that system drought suppressed foliar symptoms while still enriching putative pathogens and depleting putative antagonists in trunk wood, making clear that absence of symptoms does not equate to absence of disease. These drought-driven shifts should also not be interpreted as pre-symptomatic proxies of later foliar expression, because current evidence indicates that enrichment of *P. chlamydospora* and related drought-associated changes does not reliably predict subsequent foliar symptom incidence (Leal et al., 2024; Hrycan et al., 2025).

A critical distinction emerging from recent multi-omics and longitudinal work is that the processes driving black necrosis and those driving foliar symptom expression are not necessarily the same (Fig. 3). Current evidence suggests that *P. chlamydospora*-associated black necroses can accumulate under chronic infection or drought-linked states without tightly tracking foliar symptom expression, whereas white rot and *F. mediterranea* activity show a closer relationship with visible symptom development. Across cultivars, black necrosis extent and foliar symptom expression do not align as consistently as white rot extent and symptom expression do, supporting the view that *P. chlamydospora*-driven vascular dysfunction and *F. mediterranea*-driven decay represent partially distinct pathobiome states rather than successive readouts of a single process (Del Frari et al., 2021; Gastou et al., 2025a, 2026; Chambard et al., 2025).

Nutritional status appears to act as a similar environmental filter. Dell'Acqua et al. (2025) showed that low nitrogen nutrition reduced esca leaf symptom incidence while modifying vigour, gas exchange, senescence, and both leaf and trunk metabolomic profiles, whereas trunk fungal community diversity changed little and no robust fungal biomarkers consistently explained symptom expression. A field study on tiger-stripe expression points in the same direction. In diseased but still asymptomatic vines, higher calcium contents were repeatedly associated with the asymptomatic state, and vines treated before symptom onset with a calcium-magnesium-based fertilizer mixture showed higher early-season water and vegetation indices together with reduced

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symptom incidence and severity (Calzarano et al., 2021). Taken together, these studies suggest that visible esca expression can shift through host physiological state and mineral status without requiring major reorganization of the overall fungal community in apparently healthy trunk wood.

This interpretation is reinforced by hydraulic and multi-omic evidence. Bortolami et al. (2019) linked esca with leaf vessel occlusions and hydraulic dysfunction; Bortolami et al. (2021a) showed that annual shoots and permanent woody structures cannot be treated as a single hydraulic unit; Bortolami et al. (2021b) demonstrated that drought suppresses esca leaf symptoms; and Bortolami et al. (2023) framed symptom expression as a process involving hydraulic failure and premature senescence. Ouadi et al. (2021) similarly identified sap flow disruption as an early integrative signal of esca expression and a markedly higher proportion of white rot in symptomatic vines. In parallel, Gastou et al. (2025b) and Chambard et al. (2025) showed that symptom-associated hydraulic impairment, metabolomic reprogramming, and pathogen activity were more informative than stem microbiome structure alone, with *F. mediterranea* more transcriptionally active in esca-expressing plants and *P. chlamydospora* more strongly associated with drought.

Management fits within the same framework. Severe pruning was associated with higher GTD symptom burden, greater pathogen load and altered wood mycobiome structure, whereas minimal pruning was associated with lower necrosis (Meza et al., 2024; Travadon et al., 2016). In parallel, an integrated management protocol based on the foliar formulation AF5 reduced esca incidence and severity and, in symptomatic plants, was associated with improved stomatal function and induction of defence-related transcripts, suggesting that visible disease expression can be partly mitigated through physiological maintenance and defence priming of the host (Chitarra and Nerva, 2026).

Taken together, these studies indicate that apparent resistance may reflect very different underlying processes: reduced pathogen establishment, stronger compartmentalization, lower sensitivity of hydraulic function to chronic lesions, delayed white rot, drought- or nutrition-driven suppression of foliar symptoms, or a pathobiome configuration less conducive to visible expression in a given year. Apparent resistance is therefore best understood as the visible outcome of a compartment-dependent and environmentally filtered system.

11. Integrated Framework, Research Gaps and Conclusions

11.1. An Integrated Framework

A coherent framework for esca emerges more clearly when the syndrome is decomposed into interacting fungal and host layers rather than treated as a single phenotype.

The first layer is the pioneer vascular phase, dominated mainly by *P. chlamydospora* and *P. minimum*, and the corresponding host resistance to vascular colonization. This layer depends on xylem architecture, wound-healing capacity, compartmentalization and local induced chemistry in wood. However, the evidence reviewed here also indicates that traits relevant to local pioneer spread do not necessarily predict whole-plant susceptibility or the later transition to visible esca expression. In this sense, structural predisposition and disease expression should not be collapsed into the same phenotype.

The second layer is tolerance or resistance to hydraulic dysfunction and remote symptom expression. Here, the key issue is not only whether pioneer fungi are present, but how strongly their chronic occupation compromises sap flow, new xylem formation and leaf hydraulics. The work of Bortolami et al. (2019, 2021a, 2021b, 2023) is central because it places hydraulic failure, seasonal organ behaviour and premature senescence at the core of symptom development, while Gastou et al. (2026) further show that drought-driven symptom suppression is not accompanied by uniform microbial suppression across the vine. This reinforces the view that symptom expression is a filtered physiological outcome rather than a direct measure of fungal presence.

The third layer is the white-rot phase and the corresponding resistance to wood decay. This concerns the recalcitrance of the woody substrate, its extractives and structural fractions, and the ability of basidiomycetes such as *F. mediterranea* to detoxify defences and degrade lignocellulose.

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Importantly, recent evidence suggests that this layer is not simply a late extension of the pioneer vascular phase. Black necrosis associated mainly with *P. chlamydospora* and white rot associated with *F. mediterranea* appear to represent partially distinct pathobiome states, with different relationships to foliar symptom expression. Across cultivars, black necrosis extent does not align with foliar symptom expression as consistently as white rot extent does, which supports the view that vascular occupation and decay-driven symptom expression should be analytically separated even when they coexist in the same trunk. At the same time, recent physiopathological work indicates that cultivar susceptibility may emerge more from the interaction between constitutive physiological strategy and symptom-associated functional collapse than from any single constitutive anatomical or microbial determinant. In the VitAdapt common garden vineyard experiment, weakly susceptible cultivars tended to display lower stomatal conductance, whereas constitutive stem anatomy and stem microbial community structure did not clearly predict susceptibility; by contrast, esca expression was associated with hydraulic impairment, starch depletion and strong metabolomic reprogramming, particularly in more susceptible cultivars (Gastou et al., 2025b).

The fourth layer is resilience, understood as the capacity to maintain cambial renewal, generate new conductive tissues and remain functional under chronic infection. Dell'Acqua et al. (2024) are especially valuable here because they frame resilience in terms of stem radial growth, xylem development and physiological acclimation rather than as a vague synonym of survival. Read together with the hydraulic studies and the new compartment-based microbiome evidence, this suggests that resilience in esca should be understood as the capacity to sustain or rebuild functional wood despite chronic infection, white-rot progression and shifting environmental constraints.

A fifth, cross-cutting layer is environmental and pathobiome modulation, which determines how much of the underlying biological vulnerability becomes visible in a given season. In this respect, Gastou et al. (2026) make a particularly important point: trunk condition, young-organ communities and foliar symptoms are connected, but only partially. Esca must therefore be analysed as a compartment-dependent system, not as a single whole-plant state.

11.2. Main Research Gaps

Several gaps follow directly from this framework, but they are not all equally urgent.

Immediate priorities concern causal structure and temporal resolution. First, the pioneer and white-rot phases are still too often studied separately. Longitudinal studies following the same vines across years, lesion types and fungal phases are particularly needed. Second, microbiome studies must move beyond description toward functional pathobiome analysis. Taxonomic composition in bulk, apparently healthy wood is often insufficient to explain symptom expression. The next step is to connect compartment-specific communities with activity, interaction, metabolite exchange and contribution to tissue function or dysfunction. The contrasts highlighted by Bruez et al. (2020), Gastou et al. (2026) and Chambard et al. (2025) all point in the same direction: pathogen activity may diverge more clearly than taxonomic composition, so metabarcoding increasingly needs to be complemented by metatranscriptomic and metabolomic approaches. Future studies should also explicitly separate lesion type, fungal activity and organ compartment when inferring disease mechanisms.

Second-order priorities concern integration across biological scales. Host genetics and host physiology remain insufficiently connected. ENS1 is an important start, but we still lack a mechanistic bridge between susceptibility loci, vessel anatomy, wound healing, wood chemistry and hydraulic resilience. Biomarkers also remain overinterpreted. Foliar metabolomic and transcriptomic signatures are informative, but highly dependent on genotype, year and symptom stage (Moret et al., 2021; Weiller et al., 2024). Lecomte et al. (2024) further reminds us that timing is part of susceptibility, while Gastou et al. (2026) shows that the microbial signal of disease depends strongly on organ and tissue type. Epigenetic regulation may also deserve more attention, as Berger et al. (2025) suggests that recurrent symptom expression may involve systemic DNA methylation states not captured by visible phenotype alone. Likewise, Magnin-Robert et al. (2017) indicates that preapoplectic vines show foliar alterations before visible collapse, but these are better interpreted as trajectory-specific

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signals than universal biomarkers. Nutritional status should also be integrated more explicitly, since nitrogen availability can modify esca incidence, physiology and metabolomic responses without equally strong shifts in trunk fungal community structure (Dell'Acqua et al., 2025). Future work therefore needs to integrate time, compartment and physiological state rather than relying on one-off sampling.

Longer-term priorities concern system durability and reconstruction of function. Resilience remains underdeveloped as a biological framework. The integration of Dell'Acqua et al. (2024) suggests that future esca research should pay much more attention to cambial continuity, xylem renewal and physiological acclimation in chronically infected vines, especially under environmental modulation.

11.3. Broader Implications and Future Perspectives

Beyond grapevine, the framework proposed here may also be relevant to other woody plant disease complexes in which chronic vascular occupation, tissue decay and symptom expression are only partially coupled. One general implication is that disease expression in perennial woody hosts may often emerge from interacting but non-equivalent microbial and host-functional layers rather than from a single linear pathogenic process. A second implication concerns climate change: shifts in drought frequency, heat load and seasonal water balance are unlikely to affect pioneer vascular phases and white-rot phases in the same way, potentially increasing the decoupling between internal infection and visible symptom expression. Finally, the framework has practical value because it redirects breeding and management from the abstract search for "disease resistance" toward the more operational question of which biological layer is being targeted: pioneer restriction, hydraulic tolerance, substrate recalcitrance to decay, or long-term resilience under chronic infection.

11.4. Conclusions

A central conclusion of this review is that grapevine resistance to esca-associated fungi is not a single trait, because esca itself is not a single biological process. What is often called susceptibility may reflect vulnerability to pioneer vascular colonization, vulnerability of hydraulic function, vulnerability of the woody substrate to white rot, or vulnerability of symptom expression to environmental triggering. What is often called tolerance may reflect the ability to maintain function at similar infection levels. What is often called resilience may reflect the capacity to regenerate conductive tissues and remain productive despite chronic infection.

This layered view helps reconcile long-standing contradictions and disparate insights in the literature. It explains why cultivars may differ in common garden vineyard experiments and long-term field surveys yet appear similar in short inoculation assays; why *P. chlamydospora* can be abundant in asymptomatic wood; why drought-driven enrichment of pioneer fungi or black necrosis does not necessarily predict subsequent foliar symptom expression; why white rot is highly informative without perfectly predicting symptom severity; and why annual foliar incidence is such a poor standalone proxy for disease burden. The addition of Gastou et al. (2026) strengthens this framework by showing that drought and esca expression do not imprint the same fungal signal across the grapevine: symptom suppression in drought can coexist with pathogen-enriched trunk communities and substantial internal damage, whereas symptom expression in well-watered plants is associated with stronger changes in young organs and lower proportions of healthy wood. The visible disease phenotype is therefore best understood as a filtered output of a compartment-dependent system in which black necrosis, white rot and foliar expression may be connected, but not linearly or equivalently.

A more informative future question is therefore not "which cultivar is resistant to esca?" in the abstract. It is: resistant to which layer, in which compartment, under which environmental filter, and against which fungal strategy? A fungal-biology framework does not make esca simpler. But it makes host resistance more interpretable, and probably more actionable.

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