FEMS Microbiology Ecology, 2026, **102**(4), fiag028
https://doi.org/10.1093/femsec/fiag028
Published: 13 March 2026
Minireview

Beyond Koch's postulates: the pathobiome paradigm in grapevine esca disease

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Editor: Marcus Horn

Abstract

Esca is one of the most damaging fungal diseases of grapevine and continues to defy Koch's postulates. Although *Phaeomoniella chlamydospora*, *Phaeoacremonium minimum*, and *Fomitiporia mediterranea* are consistently associated with wood necrosis in esca-symptomatic vines, they also occur in asymptomatic vines and even in apparently healthy wood tissues without visible necrosis, and single-species but also mixed-species inoculations rarely reproduce the characteristic foliar symptoms. We hypothesize that esca is best understood as a stress-mediated pathobiome disorder of the grapevine holobiont rather than a predictable outcome of specific fungal combinations, shifting focus from pathogen identity to holobiont functional state and environmental context. In this Review, we integrate evidence from community ecology, vascular biology, and multi-omics studies to link microbial community structure and activity with host hydraulics, defence, and environmental drivers. Metabarcoding and metatranscriptomics indicate that symptom expression correlates with functional reprogramming of trunk-inhabiting fungi more than their mere presence, while metabolomics and epigenomics reveal localized physiological disruption combined with systemic regulatory responses. Climatic and edaphic stresses, particularly drought, are strongly associated with holobiont destabilization and dysbiosis, altering symptom expression without necessarily modifying pathogen occurrence. We propose a temporal, multi-phase model integrating colonization history, microbiome restructuring, and host stress physiology through long-term feedbacks. This framework emerges through convergent multi-omics evidence and generates testable predictions for early detection, microbiome-informed biocontrol, and resilience-oriented vineyard management strategies.

Keywords abiotic stress, dysbiosis, esca, holobiont, multi-omics, pathobiome

Introduction: esca as a complex pathosystem

Esca is one of the most enigmatic diseases affecting grapevine (*Vitis vinifera* L.). Historically described as a single entity, it is now recognized as a complex of wood decay syndromes encompassing young esca (Petri disease) in nursery material and esca proper in mature vines (Mugnai et al. 1999). This Review focuses specifically on esca proper in mature vineyards. Internally, affected vines display brown wood streaking, sectorial necrosis, and white spongy rot (Mugnai et al. 1999, Fischer 2006), while externally they may show chronic "tiger stripe" foliar patterns or sudden apoplexy (Surico et al. 2006, Lecomte et al. 2012). At the vineyard scale, esca incidence and symptom expression are notoriously erratic across years, vines and plots, complicating diagnosis, etiology, and management (Lecomte et al. 2012, Dewasme et al. 2022).

For many years, research on esca was guided by a traditional etiological framework grounded in Koch's postulates. *Phaeomoniella chlamydospora* (W. Gams, Crous, M.J. Wingf. and Mugnai) Crous and W. Gams and *Phaeoacremonium* spp. were initially identified as early colonizers of the xylem in both young and mature vines, while basidiomycetes such as *Fomitiporia mediterranea* M. Fisch. were implicated as the main agents responsible for white rot in older vines (Mugnai et al. 1999, Fischer 2006, Mostert et al. 2006, Cloete et al. 2015). Field surveys have reported *P. chlamydospora* in up to 94.9% of vines showing internal wood symptoms, along with high frequencies of *Phaeoacremonium minimum* (Tul. & C. Tul.) Gramaje, L. Mostert & Crous and basidiomycetes in vineyards affected by esca (Mostert et al. 2006, Fischer 2006, Díaz and Latorre 2014, Cloete et al. 2015). These fungi also occur widely in symptomless vines and in apparently healthy wood tissues without visible necrosis across diverse viticultural regions (Hofstetter et al. 2012, Choueiri et al. 2014, Bruez et al. 2014, Elena et al. 2018, 2020, Geiger et al. 2022), indicating that mixed

Received: 10 December 2025. **Revised:** 2 March 2026. **Accepted:** 12 March 2026

infections, most of them remaining latent, are extremely common. This widespread asymptomatic colonization highlights the limits of applying Koch's postulates to perennial woody hosts, where disease seems to arise not from a single causal agent but from shifting interactions among the plant, its microbiome and the environment.

A growing body of evidence has increasingly challenged this pathogen-centred perspective. Single- or dual-isolate inoculations consistently reproduce internal wood necroses but rarely give rise to the characteristic foliar symptoms, even after several seasons (Larignon and Dubos 1997, Mugnai et al. 1999, Sparapano et al. 2000, 2001, Gramaje et al. 2010, Mohammadi et al. 2013, Díaz and Latorre 2014). High-throughput surveys further reveal polymicrobial communities where esca-associated fungi coexist with saprotrophs, endophytes and potential antagonists (Monod et al. 2025a, Pinto et al. 2014, Bruez et al. 2020, Bekris et al. 2021, Geiger et al. 2022, Azevedo-Nogueira et al. 2025). Severe foliar damage has even been linked to unexpected fungal combinations, such as *Aureobasidium pullulans* (de Bary) G. Arnaud with *P. chlamydospora* (Karácsony et al. 2023).

Environmental conditions add another layer of complexity. Variability in climate and soil properties strongly influences disease expression: drought, heat waves, and heterogeneity in soil water-holding capacity have all been repeatedly associated with fluctuations in symptom incidence and vine mortality (Bortolami et al. 2021a, Monod et al. 2025b, Fischer and Peighami-Ashnaei 2019). Together, these observations indicate that esca cannot be explained by individual pathogenic species alone, but rather appears to emerge from context-dependent interactions among the host, its microbiome and the environment.

Esca sits within a larger group of decline syndromes that affect woody plants in forests and perennial cropping systems worldwide (Ciesla and Donaubauer 1994, Desprez-Loustau et al. 2006, Martelli et al. 2016). These disorders share key features with esca: they rarely follow simple causal models, typically involve multiple microbial taxa acting simultaneously, exhibit extended periods of latent infection, and display symptom expression that fluctuates with environmental conditions. Notably, abiotic stresses, particularly drought, appear to shift latent or benign endophytes into more damaging roles across diverse systems (Desprez-Loustau et al. 2006, Martelli et al. 2016, Songy et al. 2019). Oak decline under drought, for example, has been linked to the activation of endophytic fungi that remain inconspicuous under milder conditions (Sallé et al. 2014), while similar dynamics have been described in trunk and crown rots affecting fruit trees and kiwifruit (Spigaglia et al. 2020).

Despite the biological and ecological diversity of these systems, recurring patterns emerge that parallel esca's behaviour. Decline syndromes are often associated with reductions in microbial diversity, shifts toward opportunistic or stress-tolerant taxa, and progressive impairment of vascular integrity (Desprez-Loustau et al. 2006, Bettenfeld et al. 2020, 2022). Such trends reflect the nature of long-lived woody hosts, which assemble intricate microbial communities over decades. The composition and function of these microbiomes are shaped not only by plant age but also by the cumulative influence of local environmental conditions and past stress exposure. Grapevines similarly harbour diverse fungal and bacterial endophytes whose contributions to growth promotion, defence modulation and metabolic adjustments vary with pedoclimatic context, cultivar, vine age, and management practices (Pacifico et al. 2019).

Viewed through this comparative lens, disease emergence in woody plants, including esca, often coincides with a breakdown in holobiont stability rather than the invasion of novel pathogens. Across decline syndromes, stress-induced disruption of host-microbiome homeostasis appears to play a more central role in symptom development than the activity of any single pathogenic agent (Vandenkoornhuyse et al. 2015, Bettenfeld et al. 2020, Li et al. 2023). This convergence suggests that esca is not an isolated phenomenon but part of a broader pattern requiring conceptual frameworks capable of integrating microbial complexity, environmental context, and long-term host-microbiome feedbacks.

The purpose of this review is to present esca as a multifaceted pathosystem that is more accurately interpreted through the lenses of the pathobiome and the holobiont. By integrating these conceptual frameworks with recent multi-omics insights, we show how they help resolve long-standing inconsistencies in esca research and offer a more robust foundation for disease prediction and management. We first revisit esca in light of the pathobiome paradigm, then examine the grapevine holobiont and vascular pathobiology, integrate multi-omics insights, and finally propose a temporal, multi-phase model with implications for management under climate change.

The pathobiome paradigm and the disease triangle in grapevine esca

From single pathogens to microbial communities

The "pathobiome" concept refers to the entire community of microorganisms (including recognized pathogens, beneficial or neutral endophytes, wood-decomposing saprotrophs, epiphytic colonizers, and mycorrhizal symbionts) whose collective activities contribute to disease (Vayssier-Taussat et al. 2014, Bass et al. 2019). This systems-oriented view contrasts with the single-pathogen model derived from Koch's postulates and reframes pathogenicity as an emergent property of community structure, microbial interactions, host physiology, and environmental context (Lamichhane and Venturi 2015).

Esca aligns well with this paradigm. Studies conducted in nurseries and vineyards consistently show that vines harbour diverse communities of trunk-associated fungi, including *P. chlamydospora*, *Pm. minimum*, *Fomitiporia* spp., and numerous endophytes (Hofstetter et al. 2012, Bruez et al. 2020, Gramaje et al. 2022, Hrycan et al. 2023, Garcia et al. 2025). Many of these organisms display considerable lifestyle plasticity, shifting between latent and pathogenic modes depending on the physiological state of the host and environmental stressors (Hrycan et al. 2020, Bettenfeld et al. 2020, 2022). From a network-oriented viewpoint, disease does not arise simply from the presence of pathogenic taxa but from the way these fungi interact within a dynamic, multispecies community under specific environmental conditions and host stress states.

This interpretation expands the disease triangle (Fischer and Peighami-Ashnaei 2019)—host, pathogen, environment—into a tetrahedral or multidimensional framework that explicitly incorporates the microbiome (Leveau 2024) (Fig. 1). In this expanded model, esca reflects interacting processes involving (i) host hydraulic and defence status, (ii) specific pathogenic fungi,

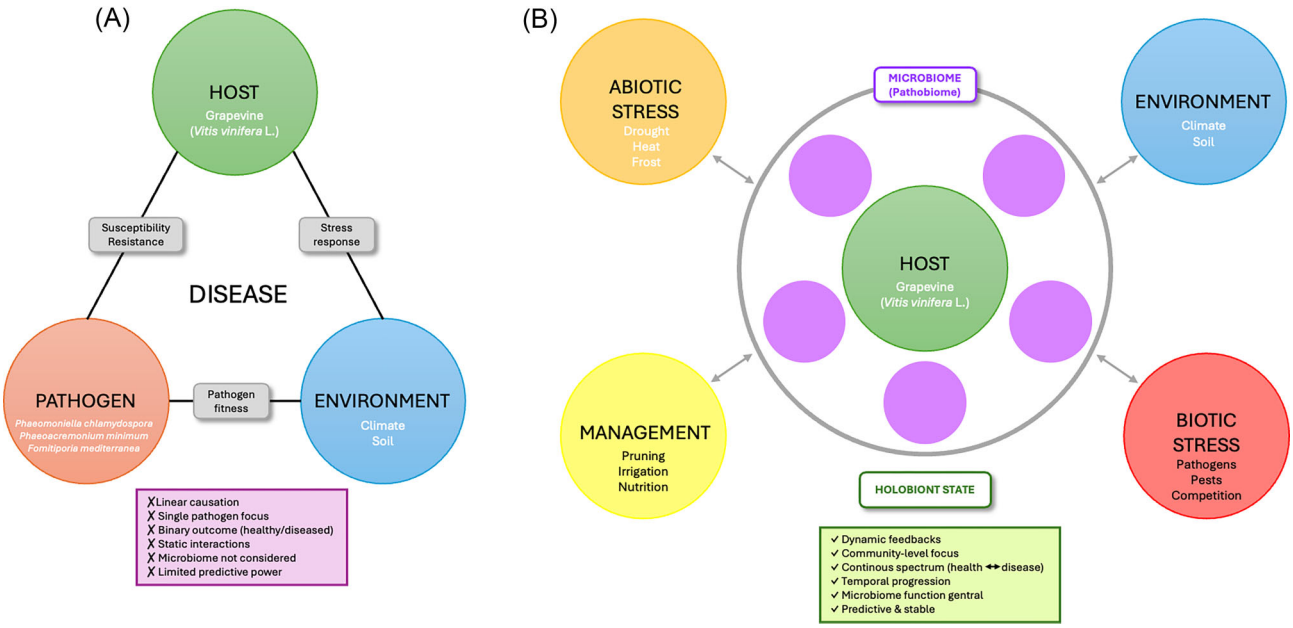

Figure 1 Paradigm shift from the disease triangle to a holobiont framework in grapevine esca. (A) Disease triangle: disease risk is conceptualized as emerging from the intersection of host susceptibility, pathogen pressure, and environment, typically emphasizing single causal agents and largely one-way effects. (B) Holobiont framework: esca is interpreted as an emergent outcome of a coupled host–microbiome system in which disease develops under stress-driven dysbiosis, shaped by bidirectional feedback among the host, its microbiota, abiotic conditions, management, and biotic stress. This framework helps explain why pathogen presence alone poorly predicts symptom expression.

(iii) resident microbial communities with antagonistic or facilitative roles, and (iv) environmental drivers. The association of *P. chlamydospora* or *F. mediterranea* with diseased vines therefore reflects only part of a larger ecological system: symptom development ultimately depends on how these fungi behave within a community embedded in a stressed holobiont (Leal et al. 2024a, Monod et al. 2025a, Bruez et al. 2020, Hrycan et al. 2025). In the following section, we examine how this community-based perspective is reflected in the etiological complexity of esca, where co-infection, latency, and context-dependent symptom expression blur the boundaries of classical disease definitions.

Etiological complexity of esca: co-infection, latency, and limits of Koch's postulates

The classification of esca has long reflected its etiological complexity. It has been described as a single but multifaceted syndrome (Lecomte et al. 2012) or as a group of overlapping diseases (Mugnai et al. 1999, Andolfi et al. 2011). Surico (2009) further distinguished white rot, chronic foliar symptoms, apoplexy, and brown wood streaking. Although white rot, almost always linked to *F. mediterranea*, was traditionally viewed as independent from tiger-stripe symptoms, recent studies challenge this separation: the development of white rot (Maher et al. 2012, Ouadi et al. 2019) or high *F. mediterranea* abundance (Del Frari et al. 2021) is strongly associated with leaf-stripe expression and apoplexy. Curettage trials also show that removing white rot reduces symptom incidence (Cholet et al. 2021, Pacetti et al. 2021, Lecomte et al. 2022), echoing early interpretations by Ravaz (1909), Viala (1926), and Arnaud and Arnaud (1931) that these manifestations reflect one disease process.

Mixed infections are the rule rather than the exception. Surveys often detect several trunk disease complexes within a single vine, and individual lesions typically contain multiple fungal taxa (Luque et al. 2009, Choueiri et al. 2014, Elena et al. 2018, Bruez et al. 2020). Metabarcoding studies show that esca-associated fungi occur within diverse communities that vary across tissues, vineyards, and cultivars (Monod et al. 2025a, Bruez et al. 2020, Azevedo-Nogueira et al. 2025). Although *P. chlamydospora*, *Pm. minimum*, and *F. mediterranea* are among the most consistently reported taxa, numerous other fungi are frequently detected, including additional *Phaeoacremonium* spp., and various basidiomycetes beyond *Fomitiporia* (e.g. *Fomitiporella*, *Inonotus*, *Stereum*) (Cloete et al. 2015, Brown et al. 2020). Importantly, these fungi, including the most frequently cited esca-associated taxa, are consistently found at similar abundances in both symptomatic and asymptomatic vines (Elena et al. 2018, Del Frari et al. 2019, Bruez et al. 2020, Geiger et al. 2022, Gramaje et al. 2022, Hrycan et al. 2023, Garcia et al. 2025), underscoring that pathogen presence alone is a poor predictor of symptom expression (Hofstetter et al. 2012).

Geographic variation in esca-associated mycobiomes further underscores the pathobiome nature of the disease. In some viticultural regions (e.g. parts of Australia, South America, and North America), *F. mediterranea* is absent or occurs at low frequency, yet esca-like foliar symptoms are still observed (Fischer et al. 2005, Lupo et al. 2006, Úrbez-Torres et al. 2014, Brown et al. 2020). In these areas, other basidiomycetes such as *Fomitiporella* spp., *Inonotus* spp., or *Stereum hirsutum* may fulfill similar ecological roles in wood decay (Cloete et al. 2015, Brown et al. 2020), reinforcing the view that symptom expression depends on community-level functional traits rather than the presence of specific taxonomic markers.

Despite this polymicrobial context, pathogenicity tests have historically relied on single-isolate inoculations. While these experiments can reproduce internal wood necroses, they rarely induce tiger-stripe symptoms or apoplexy (Larignon and Dubos 1997, Mugnai et al. 1999, Sparapano et al. 2000, 2001, Gramaje et al. 2010, Mohammadi et al. 2013, Díaz and Latorre 2014). Notable exceptions include Úrbez-Torres et al. (2014) and Brown et al. (2020), who obtained tiger-stripe-like foliar symptoms in young potted vines single or co-inoculated with *P. chlamydospora* and *Phaeoacremonium* spp. and basidiomycetes (*Fomitiporia* spp.), though these symptoms have not been consistently reproduced in mature field vines, highlighting the limitations of short-term, single-environment assays.

Overall, foliar symptoms appear to arise from interactions among toxin production, xylem dysfunction, hydraulic bottlenecks, and environmental stress (Surico et al. 2006, Bortolami et al. 2023). Similar dynamics occur in Petri disease: *P. chlamydospora* behaves as a latent pathogen whose virulence is strongly modulated by water deficit, which increases its abundance and associated necrosis without major community shifts (Hrycan et al. 2025). Disease expression therefore depends less on the pathogens themselves than on how stress reshapes host physiology and activates otherwise quiescent endophytes (Hofstetter et al. 2012).

Taken together, these findings illustrate the limitations of Koch's postulates in long-lived woody hosts with chronic mixed infections. They support the need for experimental designs incorporating multispecies inoculations, stress manipulations, and longitudinal profiling of host physiology and microbial activity (Morales-Cruz et al. 2018a,b, Chambard et al. 2025). Recognizing esca as an emergent property of host-microbiome-environment interactions naturally leads toward broader ecological frameworks, where holobiont dynamics and community assembly offer more coherent explanations than classical pathogen-centric models.

The grapevine holobiont and vascular pathobiology

Grapevine as a holobiont: microbial community structure in wood

The holobiont concept, viewing the plant and its associated microbiota as an integrated unit, provides a powerful framework for interpreting esca's complexity (Zilber-Rosenberg and Rosenberg 2008, Vandenkoornhuyse et al. 2015). Grapevines do not host a single microbiome but a set of them, spread across soil, roots, the woody cylinder, xylem sap, and even the leaf surface or interior (Berlanas et al. 2019, Niem et al. 2020, Darriaut et al. 2022, Bettenfeld et al. 2022). Moreover, fungal communities differ substantially between trunk wood (older, perennial tissue) and stem wood (younger, annual growth), reflecting age-dependent colonization dynamics and environmental exposure history (Gastou et al. 2026). Each of these compartments contributes in different ways to nutrient uptake, stress tolerance, and suppression of some pathogens, yet they also harbour fungi that may stay latent for years or behave opportunistically when conditions change (Pacifico et al. 2019). In many respects, these microbial communities seem to reflect long-term co-associations shaped by the vine's perennial nature, local terroir, and the filtering imposed by particular genotypes (Gramaje et al. 2022).

Wood and xylem tissues, long assumed to be almost sterile, actually host a surprisingly diverse set of bacterial and fungal groups (Haidar et al. 2021, Bettenfeld et al. 2022). Both culture-based methods and metabarcoding studies routinely recover common bacterial genera such as *Pseudomonas*, *Bacillus*, and *Methylobacterium*, along with numerous fungi, including well-known trunk pathogens and potential antagonists with biocontrol activity against esca-associated fungi (Pinto et al. 2014, Bruez et al. 2020, Bekris et al. 2021). Still, the overlap between these approaches is far from complete. Molecular surveys usually reveal higher richness, particularly in older vines, and they also show strong microhabitat differentiation between bark, sapwood, and heartwood (Dissanayake et al. 2018, Deyett and Rolshausen 2020). This suggests that the internal structure of the trunk acts as a fairly tight filter on microbial distributions.

Regional metabarcoding studies reinforce this idea: endophytic communities in trunks vary with terroir, cultivar, vine age, and rootstock, and Grapevine Trunk Disease (GTD)-associated families like *Phaeomoniellaceae* and *Togniniaceae* frequently dominate wood tissues across sites (Azevedo-Nogueira et al. 2025). Interestingly, alpha diversity often remains similar between healthy and symptomatic vines, while beta diversity is mostly driven by vineyard identity and tissue type (Del Frari et al. 2019, Bekris et al. 2021). Altogether, this supports the view that many esca-associated fungi are persistent components of the grapevine mycobiome, and that their behaviour, rather than their simple presence, is what shifts under stress.

The host also shapes the holobiont through its own structural traits. Wood anatomy, vessel diameters, and the vine's ability to compartmentalize damaged tissues all influence how microbes establish and spread. Cultivars with wider vessels or less effective compartmentalization tend to show more extensive necrosis and stronger hydraulic impairment in experimental inoculation studies (Bortolami et al. 2021b, Pouzoulet et al. 2014, 2017, 2020, Ramsing et al. 2021). However, recent evidence across a large cultivar panel ($n = 23$) reveals no consistent relationship between constitutive vessel anatomy (vessel diameter, ray parenchyma fraction, pit structure) and esca foliar symptom expression in the field (Gastou et al. 2025b), suggesting that anatomical traits do not determine disease susceptibility at the whole-plant level. In practice, the trunk behaves as a long-term reservoir for a diverse suite of microbes whose ecological dynamics, especially how they interact with the vascular system, appear to determine whether endophytism remains benign or moves toward disease. Understanding these transitions sets the stage for the mechanistic aspects discussed in the following section.

Vascular colonization, xylem dysfunction, and symptom expression

Esca-associated fungal pathogens can colonize grapevines through multiple routes. Pruning wounds, graft interfaces, and other entry points provide opportunities for infection by airborne or tool-borne inoculum (Mugnai et al. 1999, Fischer 2006, Gramaje and Armengol 2011). However, *P. chlamydospora* and, to a lesser extent, *Phaeoacremonium* spp. are frequently detected in nursery

propagation material (scion and rootstocks) prior to field planting (Gramaje and Armengol 2011), and their widespread occurrence in asymptomatic young and mature vines suggests that these fungi may be vertically transmitted and constitute latent components of the grapevine holobiont from the outset (Hofstetter et al. 2012, Bruez et al. 2020, Hrycan et al. 2020). Once established, ascomycetes such as *P. chlamydospora* and *Phaeoacremonium* spp. typically colonize younger woody tissues and xylem vessels, whereas basidiomycetes such as *F. mediterranea* and related taxa establish in older wood and drive white-rot formation (Mugnai et al. 1999, Fischer 2006, Maher et al. 2012, Cloete et al. 2015), a feature strongly associated with chronic esca symptoms (Maher et al. 2012). Metabarcoding studies confirm this age-dependent pattern: decay-inducing fungi are abundant in permanent structures but largely absent from current-season canes, which instead host non-pathogenic endophytes and saprotrophs (Del Frari et al. 2019, Martínez-Diz et al. 2020). The dual origin of esca-associated fungi, both as latent endophytes inherited from propagation and as wound-invading opportunists, underscores that colonization alone does not explain the timing or intermittence of foliar symptoms.

At the organ scale, white rot represents a simplified and distinct pathobiome: *F. mediterranea* may account for 60–90% of fungal sequences in necrotic cordons at symptom onset, co-occurring with *P. chlamydospora*, while adjacent sound wood retains diverse communities (Bruez et al. 2014, 2020). Bacterial assemblages are similarly simplified, enriched in *Sphingomonas* and *Mycobacterium* (Bruez et al. 2020). White rot therefore reflects a functional microbial state rather than just advanced decay. Notably, wood beneath tiger-striped leaves can resemble that beneath asymptomatic leaves (Del Frari et al. 2019), suggesting that symptoms depend more on functional states than on taxonomic shifts. Building on this, Del Frari et al. (2022) proposed the "edge hypothesis," in which symptoms arise when leaf-stripe-inducing molecules (LSSIM) surpass a plant- and environment-dependent threshold; management that reduces LSSIM or raises the threshold may suppress symptoms without removing esca-associated fungi.

The mechanisms underlying foliar symptom development remain one of the major unanswered questions in esca research. Claverie et al. (2020) reformulated two major, non-exclusive mechanistic hypotheses: (i) toxin hypothesis-systemic transport of phytotoxic compounds produced by trunk pathogens or associated microbes (Andolfi et al. 2011); and (ii) hydraulic-failure hypothesis-disruption of sap flow due to vessel blockage and loss of hydraulic conductivity (Lecomte et al. 2012, Bortolami et al. 2019, 2021a, 2023). These hypotheses imply that foliar symptoms may arise from toxin-mediated processes, vascular dysfunction or, most plausibly, their interaction under environmental stress. Recent work shows symptom onset is associated with tyloses and gels in symptomatic leaves (Bortolami et al. 2023) and young shoots (Bortolami et al. 2021a). These occlusions, consistent with non-gaseous embolisms, may generate internal streaking. Although occlusions can form under aging or environmental stress (De Micco et al. 2016), their concentration in leaves appears specific to esca. Air embolism has not been detected (Bortolami et al. 2021a), and reduced xylem transport does not necessarily alter whole-plant water status (Bortolami et al. 2021b), leaving the initial trigger uncertain, especially under combined heat, drought, and fungal activity. Xylem-mobile signals (toxins, elicitors, or both) may contribute to tylose formation and hydraulic collapse (Bortolami et al. 2021a, Ouadi et al. 2019), alongside hormonal pathways involving ethylene and auxin (De Micco et al. 2016), which may explain partial leaf abscission and, in severe cases, apoplexy.

Host responses compound vascular dysfunction. Tyloses and gels compartmentalize decay but progressively restrict water flow (Pouzoulet et al. 2014, 2017, Ouadi et al. 2019, 2020), and symptomatic stems often show 50%–70% loss of conductivity (Bortolami et al. 2021b, Ouadi et al. 2019). Chronic symptoms likely reflect cumulative hydraulic decline, whereas apoplexy results from acute failure during high evaporative demand (Surico et al. 2006, Bortolami et al. 2019, 2023). Yet vascular dysfunction alone cannot explain strong variability in symptom expression among similarly colonized vines. This has shifted attention toward the functional state of the pathobiome rather than its composition. Multi-omics approaches linking community structure, gene expression, and metabolite profiles have therefore become essential for understanding the mechanistic basis of esca.

Multi-omics insights into the esca pathobiome

Metabarcoding, metatranscriptomics, metabolomics, and epigenomics in esca-affected vines

High-throughput omics technologies have transformed our understanding of esca by capturing microbial composition, activity, functional traits, and host regulatory states at different levels. Together, these complementary approaches show that esca is associated with shifts in community function rather than the simple presence of trunk pathogens, host physiology, and stress integration within the grapevine holobiont.

Metabarcoding: who is there

Extensive metabarcoding studies have shown that grapevine wood hosts complex and taxonomically diverse microbial communities. These assemblages include not only fungi typically linked to esca, but also saprotrophs, endophytes, pathogens, and many poorly characterized taxa (Pinto et al. 2014, Bruez et al. 2020, Bekris et al. 2021, Azevedo-Nogueira et al. 2025). Still, taxonomic resolution remains limited when multi-locus data from cultured isolates are lacking (Dissanayake et al. 2018), and metabarcoding cannot distinguish active microbes from dormant or dead cells (Eichmeier et al. 2018). The recent detection of abundant mycoviruses in esca-associated fungi (Debat et al. 2025) adds yet another layer of complexity to trunk microbiome interactions.

Metatranscriptomics: who is active and what they are doing

Metatranscriptomics overcomes the inability to infer activity from presence alone. Using a closed-reference, multi-species pipeline, Morales-Cruz et al. (2018a) demonstrated that co-infecting fungi express distinct virulence suites *in planta* and that esca has a characteristic community-level expression signature. Subsequent studies confirmed strong species- and tissue-specific expression of virulence, wood-decay, and stress-response genes (Morales-Cruz et al. 2018b, Massonnet et al. 2018, Nerva et al. 2022). Stress

conditions modulate these activities: for example, water deficit increases *P. chlamydospora* abundance and expression of transport and antioxidant genes, whereas esca symptom expression is associated with strong *F. mediterranea* activity (elevated relative abundance and upregulation of virulence-related genes, including CAZymes, peroxidases and detoxification pathways) (Chambard et al. 2025). Importantly, these microbiome shifts primarily involve changes in fungal relative abundance rather than complete community turnover, with *F. mediterranea* transcriptional activity playing a central role in symptom development.

Metabolomics: what biochemical states emerge in the holobiont

Metabolomic profiling serves as a critical tool for linking microbial activity to biochemical transformations within plant tissues. Esca-affected wood displays accumulations of stilbenes, flavonoids, and phenylpropanoids, as well as shifts in central carbon and lipid metabolism (Morales-Cruz et al. 2018b, Dell'Acqua et al. 2025a, Lima et al. 2017). These analyses reveal that stress-induced defense reprogramming is associated with changes in the wood niche that favour *F. mediterranea* under esca physiology and *P. chlamydospora* under drought, even when overall diversity remains stable.

Epigenomics: how the host stores and transmits stress information

Recent work has added an epigenomic layer to our understanding of esca. Berger et al. (2025) found that transcriptional and metabolic shifts are confined to symptomatic leaves, while DNA methylation changes occur throughout the vine, even in tissues that look healthy. Symptomatic leaves tend to be broadly hypomethylated, consistent with defence activation, whereas asymptomatic leaves show their own distinct methylation signatures. Notably, pre-symptomatic vines display stable differentially methylated regions across years, hinting at epigenetic marks that persist regardless of environment or symptom expression. Altogether, these results suggest that esca involves localized physiological disruption coupled with systemic regulatory reprogramming, a form of holobiont "memory" that may shape how vines respond to future stress and infection.

Figure 2 integrates evidence from metagenomics, metatranscriptomics, metabolomics, and epigenomics, demonstrating convergence on the pathobiome model. Across all molecular levels, symptomatic vines are characterized by dysbiosis-compositional shifts toward pathogen dominance, functional reprogramming of virulence and decay pathways, metabolic changes that restructure the wood niche, and epigenetic alterations that persist systemically. Together, these multi-omics layers show that esca is associated with functional state of the holobiont rather than simple pathogen presence.

Microbial networks and dysbiosis signatures under stress

Network analysis has emerged as a valuable tool to infer potential ecological interactions from co-occurrence patterns in microbiome data (Bass et al. 2019, Lv et al. 2023). In grapevine wood, co-occurrence networks built from metabarcoding datasets generally show that healthy or asymptomatic tissues harbour complex, modular networks with numerous positive and negative associations among taxa (Bekris et al. 2021, Azevedo-Nogueira et al. 2025).

From healthy networks to dysbiosis: simplification under disease and stress

By contrast, diseased or stressed tissues typically exhibit reduced network complexity, marked by fewer connections, loss of modular structure and the dominance of only a few pathogenic taxa (Leal et al. 2024a, Monod et al. 2025a). These features match the concept of dysbiosis, where a once diverse and resilient community transitions into a simplified, unstable assemblage prone to opportunism (Bettenfeld et al. 2020, 2022).

White rot as terminal dysbiosis

In esca-affected trunks, white-rot areas represent the most advanced stage of this shift, hosting a much simpler fungal assemblage, largely composed of *F. mediterranea* and *P. chlamydospora*, and a limited group of bacteria including *Sphingomonas* and *Mycobacterium* (Bruez et al. 2020), compared with the surrounding sound wood. These communities, dominated by pathogenic taxa, mark a terminal dysbiosis state in the trunk-associated pathobiome.

Stress and genotype modulate microbial interactions

Recent experimental evidence refines this picture. Gastou et al. (2026) showed that intense summer drought was associated with a pronounced collapse in Ascomycota diversity in healthy trunk wood and a concomitant increase in wood pathogens, even in the absence of foliar symptoms. Beyond global network properties, correlation analyses indicate that pathogen-pathogen and pathogen-endophyte interactions are modulated by genotype and region. SparCC analysis by Azevedo-Nogueira et al. (2025) revealed that esca-related genera display both cooperative and antagonistic interactions, which may switch between competition and facilitation depending on cultivar, vine age or rootstock. A number of endophytic fungi repeatedly displayed negative correlations with trunk pathogens, pointing to their potential value for biocontrol.

Dysbiosis as network reconfiguration rather than diversity loss

Overall, dysbiosis in esca is not merely a reduction in diversity but a profound reconfiguration of interaction networks, whose topology is shaped by host genotype and environmental context. Within this dysbiotic microbiome, the functional activation of *F. mediterranea*, characterized by virulence gene upregulation, represents the critical transition that triggers symptom expression, suggesting that dysbiosis provides the ecological context enabling keystone pathogen activity rather than directly causing symptoms. This view highlights that assessments of disease progression and microbiome-guided management must integrate information on community architecture and microbial interactions, moving beyond taxonomic inventories alone.

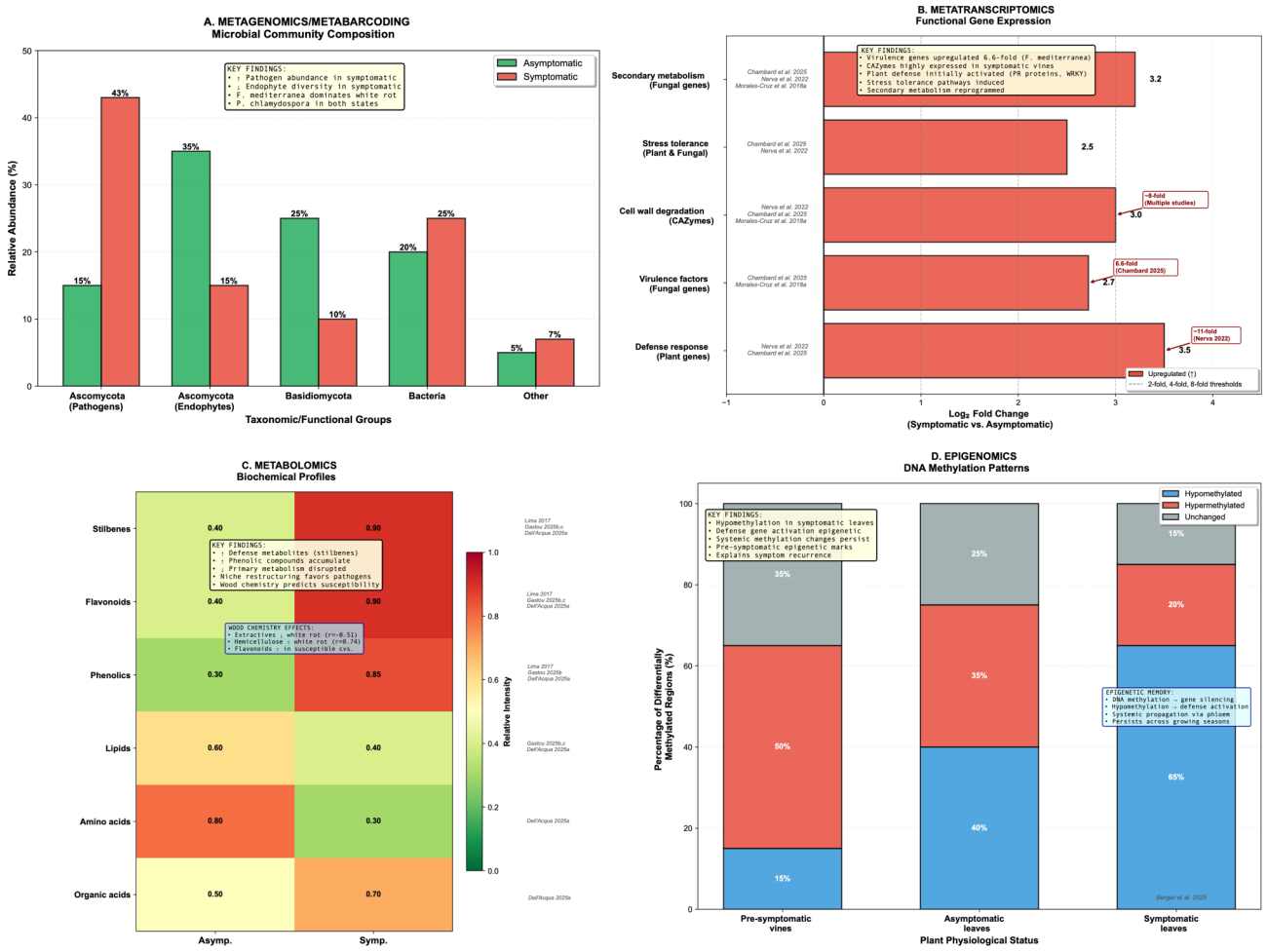

Figure 2 Multi-omics evidence supporting the pathobiome paradigm in grapevine esca. (A) Community structure (metabarcoding/metagenomics). Symptomatic vines typically show pathogen-biased, low-diversity microbiomes compared with more diverse communities in asymptomatic vines. Bars are schematic and illustrate the qualitative pattern of dysbiosis using approximate groupings from Bruez et al. (2020) (pathogenic Ascomycota, endophytic Ascomycota, Basidiomycota, Bacteria). Reported Shannon indices: 0.97 (white-rot necrotic tissue) vs 2.77 (non-necrotic tissue). (B) Community activity (metatranscriptomics). Symptom expression is associated with strong activation of wood-decay/virulence functions, including high expression of carbohydrate-active enzymes (CAZymes genes) and oxidative enzymes in wood-decay basidiomycetes (e.g. *F. mediterranea*), alongside activation of host defence pathways (e.g. PR proteins, WRKY-related responses) (Morales-Cruz et al. 2018a, Nerva et al. 2022, Chambard et al. 2025). Bars summarize published $\log_2$ fold-change trends across major functional categories (schematic synthesis; not a *de novo* re-analysis). (C) Biochemical niche restructuring (metabolomics/wood chemistry). Symptomatic vines show accumulation of defence-related metabolites (stilbenes, flavonoids, phenolics) and shifts in primary metabolism (Lima et al. 2017, Dell'Acqua et al. 2025a, Gastou et al. 2025a, b). Wood chemical traits correlate with susceptibility and wood decay (e.g. extractives vs susceptibility; hemicellulose vs white-rot proportion), supporting wood biochemistry as a key determinant of outcome (Gastou et al. 2025a). (D) Systemic regulation and "memory" (epigenomics). Esca expression is associated with differential DNA methylation patterns in leaves, including predominantly hypomethylated regions in symptomatic tissue and systemic methylation changes detectable beyond symptomatic leaves, yielding candidate epigenetic signatures associated with symptom recurrence or predisposition (Berger et al. 2025). Stacked bars depict proportions of differentially methylated regions across plant statuses (schematic). Abbreviations: CAZymes, carbohydrate-active enzymes; DEG, differentially expressed gene; DMR, differentially methylated region; PR, pathogenesis-related.

Abiotic stress, holobiont imbalance, and woody decline syndromes

The drought-esca paradox and latent infections

Among the environmental drivers implicated in esca, drought has received the most research attention due to its paradoxical effects and its central role in the esca disease triangle (Fischer and Peighami-Ashnaei 2019). However, other factors, including temperature extremes, soil properties, nutrient availability, and their interactions, remain understudied and may be equally or more important in shaping disease outcomes (Dell'Acqua et al. 2025a, Monod et al. 2025b, Etienne et al. 2026). Moderate to severe water deficit can markedly suppress foliar symptoms in previously symptomatic vines (Bortolami et al. 2021a, Calvo-Garrido et al. 2021). Lower transpiration restricts sap movement and may limit the systemic transport of metabolites or signalling compounds required for symptom development (Bortolami et al. 2021a, 2023), which is broadly consistent with models proposing the

involvement of long-distance chemical signalling (Andolfi et al. 2011, Claverie et al. 2020).

At the same time, drought imposes strong ecological stress on the wood microbiome. Water deficit often reduces fungal diversity and shifts communities toward simpler, pathotroph-enriched assemblages dominated by opportunistic species (Leal et al. 2024a, Monod et al. 2025a). Despite the variety of approaches used, the results converge on the same pattern: multi-omics analyses showed that drought favoured *P. chlamydospora* activity, whereas *F. mediterranea* activity remained largely unchanged (Chambard et al. 2025); greenhouse and field experiments on young vines revealed that intense water stress increased *P. chlamydospora* colonization and necrosis, accelerating vine decline in some cases (Hrycan et al. 2025); and long-term greenhouse trials found reduced microbial diversity and enrichment of *P. chlamydospora* in the xylem of drought-stressed plants (Leal et al. 2024a). This selective response, *P. chlamydospora* upregulation without *F. mediterranea* activation, helps explain why drought-induced microbiome shifts do not predict subsequent foliar symptom expression (Gastou et al. 2026, Dell'Acqua et al. 2025b).

Drought also affects wood structure, moisture content and resource availability in ways that can favour the expansion of latent fungi (Pouzoulet et al. 2014, 2017, Ouadi et al. 2019, 2020). While anecdotal observations have suggested that symptoms may reappear after drought stress is relieved (Lecomte et al. 2012), evidence for a consistent post–drought increase in incidence has not been reported consistently (Gastou et al. 2025a, Dell'Acqua et al. 2025b, Bortolami et al. 2019, 2021a, 2023). Instead, vineyard-scale climatic patterns reveal a different relationship. Using two decades of data from 493 commercial vineyards, Etienne et al. (2026) showed that esca incidence increases under wet conditions, particularly when soil wetness and evapotranspiration are high. By contrast, warm and dry springs and summers are associated with reduced incidence. The climatic conditions of the current year had the strongest influence, and consecutive wet years amplified symptom expression. These findings capture the drought-esca paradox: drought suppresses visible symptoms even though latent infections remain widespread, yet drought-stressed vines do not exhibit increased symptom expression upon rehydration.

Nutrient availability introduces an additional layer of complexity. In naturally infected Sauvignon Blanc vines, nitrogen deficiency was associated with lower esca incidence compared with moderate fertilization (Dell'Acqua et al. 2025a). While nitrogen strongly influenced growth, photosynthesis and plant metabolomes, fungal community composition remained largely unchanged, suggesting that host-driven factors such as canopy structure, transpiration and defence pathways, can shape symptom expression independently of pathogen composition. Reduced transpiration and elevated phenylpropanoid synthesis under low nitrogen align with this pattern.

In summary, drought and nutrient status can temporarily suppress symptom expression through reductions in transpiration or enhanced defence responses. However, the relationship between stress relief and symptom resurgence remains poorly understood. While moderate water stress has been associated with increased symptom expression in some field observations (Calvo-Garrido et al. 2021), severe experimental drought (ΨPD ~ -1.0 MPa) completely inhibits symptom development (Bortolami et al. 2021a). Moreover, long-term monitoring has not confirmed a consistent pattern of increased symptom incidence in years following drought stress (Gastou et al. 2026, Dell'Acqua et al. 2025b). Acute events, including apoplexy, have been hypothesized to reflect the combined pressures of high evaporative load, altered microbial activity and cumulative vascular dysfunction (Surico et al. 2006, Bortolami et al. 2019, Claverie et al. 2020, 2023), though the precise triggers and mechanisms remain unclear.

Infection-stress causality: competing and integrative models

The causal interplay between infection, stress and symptom expression in esca can be framed through three non-exclusive models that differ in how they assign primacy to colonization versus environmental perturbation.

1. *Stress-driven dysbiosis.* Abiotic stress, including drought, heat, and fluctuating moisture, acts as the main destabilizing force. Stress weakens host defences and alters wood chemistry, which is associated with reduced microbial diversity, breakdown of antagonistic interactions and enrichment of opportunistic fungi already present in the holobiont (Leal et al. 2024a, Desprez-Loustau et al. 2006, Lv et al. 2023). This perspective echoes the broader GTD framework of Hrycan et al. (2020), who argued that many trunk pathogens behave as latent endophytes until environmental stress disrupts host-microbiome equilibrium. Recent experimental work under drought confirms that trunk endophytic communities can be strongly restructured, with loss of Ascomycota diversity and increased abundance of wood pathogens even in the absence of foliar symptoms (Gastou et al. 2026). Under this model, colonization is widespread but remains asymptomatic until stress pushes the system beyond a resilience threshold (Bettenfeld et al. 2020, 2022). However, current evidence is heavily biased toward water-related stressors; other abiotic factors (e.g. nutrient imbalances, soil pH, heavy metals, salinity) and biotic stresses (e.g. co-infection with viruses, nematodes, or other pathogens; herbivory; root competition) remain largely unexplored and may prove equally or more important in triggering dysbiosis and symptom expression.
2. *Latent infection with stress activation.* Esca-associated fungi may establish in vines early, either through nursery transmission or field infection, and persist for years without producing visible symptoms (Monod et al. 2025a, Hofstetter et al. 2012, Bruez et al. 2020). This raises a fundamental question: should these fungi be considered "latent pathogens" that infected the plant and await activation, or "resident endophytes" that are natural components of the grapevine holobiont from the outset? The evidence supports elements of both perspectives. *Phaeomoniella chlamydospora* and *Phaeoacremonium* spp. clearly can infect through pruning wounds in the field (Mugnai et al. 1999, Fischer 2006, Gramaje et al. 2018), yet they are also ubiquitous in nursery material and asymptomatic vines across regions, cultivars, and age classes (Hofstetter et al. 2012, Gramaje et al. 2022, Hrycan et al. 2023), suggesting that many vines harbor these fungi as inherited or early-acquired endophytes rather than as discrete infection events. From a holobiont perspective, the critical question shifts from "when did infection occur?" to "what triggers the transition from benign to pathogenic behavior?" (Hrycan et al. 2020,

Bettenfeld et al. 2020). Whether acquired vertically or horizontally, the functional outcome is similar: stress is associated with increased fungal metabolism, microbial activity and modifies host responses, consistent with transcriptomic and metabolomic shifts observed in natural infections (Morales-Cruz et al. 2018b, Pouzoulet et al. 2014, 2017, Ouadi et al. 2019, 2020, Chambard et al. 2025). Work on young vine decline (Petri disease), while distinct from esca proper in mature vines, provides relevant insights into *P. chlamydospora* behaviour under stress: water stress increased *P. chlamydospora* abundance and necrosis and, in some cases, led to collapse, but without guaranteeing symptom expression (Hrycan et al. 2025). This pattern, stress-driven fungal proliferation without deterministic symptom outcomes, appears conserved across both young vine decline and esca proper, reinforcing that fungal presence, whether from external infection or inherited endophytism, and stress do not translate linearly into visible symptoms. Importantly, this conceptual ambiguity has practical implications: if esca-associated fungi are integral microbiome components, eradication-focused strategies are unlikely to succeed, and management must instead prioritize holobiont stability through stress mitigation and microbiome support.

3. *Integrated feedback model.* Increasing evidence supports a dynamic feedback model in which infection and stress reinforce each other. Episodic stresses (e.g. drought, pruning wounds, frost) are associated with progressive shifts in microbiome composition, host physiology and wood structure toward states that correlate with enhanced fungal activity and reduced resilience (Desprez-Loustau et al. 2006, Bettenfeld et al. 2020, 2022, Del Frari et al. 2023). Accumulated vascular and metabolic disturbances then amplify vulnerability to subsequent stress, producing non-linear trajectories of symptom onset, remission or collapse (Monod et al. 2025b, Lecomte et al. 2012, Calvo-Garrido et al. 2021, 2024). Within this feedback framework, *P. chlamydospora* and *F. mediterranea* play distinct but complementary roles: stress-driven proliferation of *P. chlamydospora* progressively impairs vascular function and destabilizes the microbiome (Leal et al. 2024a, Hrycan et al. 2025), creating conditions that may facilitate *F. mediterranea* activity, whose virulence gene upregulation is strongly associated with foliar symptom expression (Chambard et al. 2025). Results from Hrycan et al. (2025), where water stress increased mortality in vines infected with *P. chlamydospora*, fit this framework by illustrating how infection and stress co-escalate rather than acting independently. However, the precise mechanisms linking *P. chlamydospora*-driven wood dysfunction to *F. mediterranea* activation remain unclear, representing a critical knowledge gap in understanding symptom expression.

Overall, the convergent evidence throughout this Review strongly supports the integrated feedback model as the most coherent framework for understanding esca dynamics. Multi-omics data strongly support this integrative model, showing that microbial gene expression, host transcriptomic profiles, and metabolite signatures differ markedly between drought stress and esca symptom expression (Morales-Cruz et al. 2018b, Leal et al. 2024a, Chambard et al. 2025). Ecological models of pathogen emergence also highlight the roles of microbial cooperation, interkingdom signalling and context-dependent shifts in microbial behaviour (Lv et al. 2023). Appreciating these feedbacks is crucial for building predictive disease models and devising effective management strategies.

Towards a holistic, multi-stage model of esca disease

Dissociating wood necrosis from foliar symptom expression

A critical distinction emerging from recent multi-omics and longitudinal studies is that processes driving wood necrosis, particularly black necroses associated with *P. chlamydospora*, may be largely decoupled from those driving foliar symptom expression (Gastou et al. 2026, Chambard et al. 2025). Across a large panel of cultivars, black necrosis extent does not correlate with foliar symptom expression, whereas white rot extent does (Gastou et al. 2025a), reinforcing that *P. chlamydospora*-driven black necrosis and *F. mediterranea*-driven white rot represent functionally distinct pathobiome states with different relationships to symptom development.

By contrast, foliar symptom expression is strongly associated with *F. mediterranea* activity and white rot development (Gastou et al. 2025a, Maher et al. 2012, Del Frari et al. 2021, Chambard et al. 2025). Metatranscriptomic data show that *F. mediterranea* virulence genes, including CAZymes, peroxidases, and detoxification pathways, are specifically upregulated in symptomatic vines, whereas *P. chlamydospora* virulence is elevated under drought but not necessarily during symptom expression (Chambard et al. 2025). Paradoxically, esca-symptomatic vines may even display enhanced drought tolerance due to reduced canopy area and lower whole-plant transpiration (Dell'Acqua et al. 2025b), further supporting an antagonistic rather than synergistic relationship between drought and esca foliar symptoms.

These findings indicate that drought-driven dysbiosis (enrichment of *P. chlamydospora*, black necrosis) and esca foliar symptom expression (driven by *F. mediterranea*, white rot) represent partially independent pathobiome states. The temporal model below integrates this distinction.

A temporal, multi-phase framework

Synthesizing current evidence, esca can be conceptualized as a multi-stage, context-dependent process rather than a linear infection-symptom sequence (Desprez-Loustau et al. 2006, Lecomte et al. 2012, 2024). A heuristic framework includes the following overlapping phases:

Phase I: Latent colonization and microbial establishment (Fig. 3, green zone).

Trunk pathogens and other endophytes colonize wood via nursery infections, pruning wounds or natural openings (Gramaje et al. 2018). During this stage, host defences and anatomical compartmentalization limit pathogen spread (Pouzoulet et al. 2017), and the microbiome remains relatively stable. This phase can persist for many years without visible symptoms. As summarized in Table 1, strong observational support for this latent stage comes from metabarcoding surveys (Bruez et al. 2020, Gramaje et al.

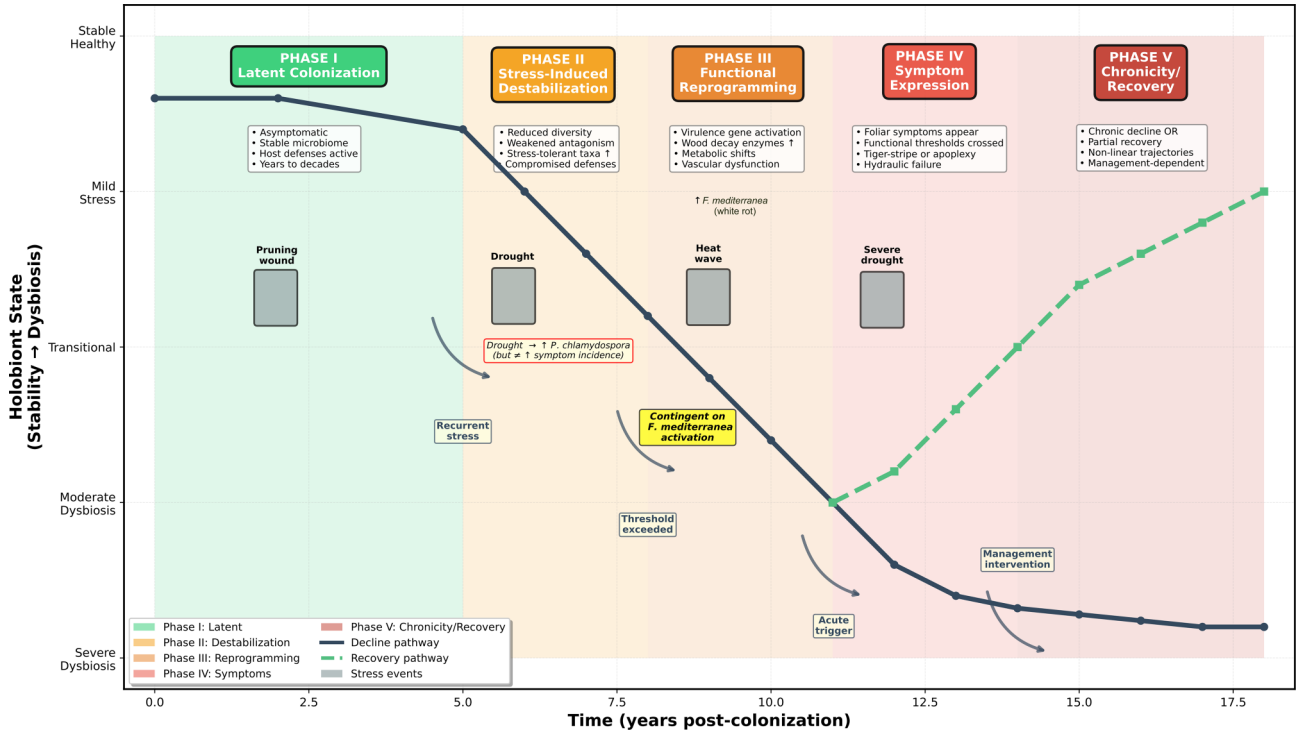

Figure 3 A temporal, multi-phase framework for grapevine esca pathobiome dynamics. Esca is represented as a non–linear, multi–phase trajectory of the trunk pathobiome across time since colonization (*x*–axis), with the *y*–axis indicating a shift from holobiont stability to dysbiosis. Colored backgrounds indicate phase–specific states; gray boxes denote stress events that may trigger transitions. Solid line indicates a decline trajectory; dashed line indicates partial recovery. Timelines vary by cultivar, environment, and management. Phase I (Latent colonization / stable holobiont): high microbial diversity and balanced interactions with limited functional disruption. Phase II (stress–induced destabilization): abiotic stress (e.g. drought, heat) can reduce diversity and weaken host defences, favouring stress–responsive taxa, including increased abundance/activity of *P. chlamydospora* and associated black necrosis processes. Importantly, longitudinal evidence indicates that drought–driven enrichment of *P. chlamydospora* and related shifts do not reliably predict subsequent foliar symptom incidence (Bortolami et al. 2021a; Gastou et al. 2026; Dell'Acqua et al. 2025b). Phase III (Functional reprogramming / decay activation): progression toward symptom–linked states is contingent on activation of wood–decay basidiomycetes (e.g. *F. mediterranea* or functionally similar taxa), with increased expression of decay/virulence pathways (e.g. CAZymes, oxidative enzymes) and advancing white rot (Chambard et al. 2025). Phase IV (Symptom expression): tiger–stripe foliar symptoms and/or apoplexy emerge when physiological thresholds are crossed under conducive environmental conditions, reflecting combined effects of microbial activity, vascular dysfunction, and host responses (conceptual; mechanisms remain debated). Phase V (Divergent outcomes): vines may partially recover (e.g. via restoration of hydraulic function and management–mediated stabilization) or enter chronic decline.

2022), although the processes maintaining long-term latency remain unclear.

Phase II: Stress-induced holobiont destabilization (Fig. 3, orange zone).

Recurrent or intense abiotic stress (drought, heat or restrictive soils) disrupts host homeostasis and reshapes the internal microbiome. Diversity declines, antagonistic interactions weaken and opportunistic or stress-tolerant fungi increase in relative abundance (Gastou et al. 2026). These shifts occur alongside compromised host defences (Bortolami et al. 2021a, Chambard et al. 2025) and stress-mediated changes in wood chemistry (Pouzoulet et al. 2014, 2017, Ouadi et al. 2019, 2020) that favour pathogen activity. Table 1 indicates that this phase has the strongest experimental basis, with drought manipulation studies providing partial causal evidence (Gastou et al. 2026). In Fig. 3, this transition is shown as being triggered by episodic stress events such as heat waves or pruning injuries. Importantly, recent longitudinal studies reveal that stress-driven microbiome shifts, particularly increased *P. chlamydospora* abundance under drought, do not necessarily predict subsequent foliar symptom expression (Bortolami et al. 2021a, Gastou et al. 2026, Dell'Acqua et al. 2025b). This suggests that Phase II dysbiosis may represent a predisposing context, but it is not sufficient and is not consistently predictive of foliar symptom expression; progression depends on additional triggers, including the functional activation of wood–decay basidiomycetes such as *F. mediterranea* (Chambard et al. 2025).

Phase III: Functional reprogramming and dysbiosis (Fig. 3, dark orange zone).

As dysbiosis sets in, fungal communities shift from coexistence to active wood decay. Virulence factors, CAZymes and secondary metabolism pathways become upregulated (Morales-Cruz et al. 2018a,b, Chambard et al. 2025), as illustrated in Fig. 2B. Correspondingly, host metabolism changes: stilbenes, phenolics and other defence compounds accumulate and reshape the wood environment (Fig. 2C). Early vascular dysfunction also appears during this phase, including reduced hydraulic conductivity and initial vessel occlusion (Bortolami et al. 2019, 2021a). Table 1 notes that transcriptomic

Table 1 Evidence summary for temporal multi-phase model components.

Model Component	Study Type	Key Studies	Evidence Level	Causality	Validation Status
Phase I: Latent colonization	Observational, Metabarcoding	Bruez et al. 2014, 2020	Strong	Correlative	Established
Phase II: Stress-induced destabilization	Experimental, Observational, Physiological	Gastou et al. 2026; Dell'Acqua et al. 2024	Strong	Partial	Replicated
Phase III: Functional reprogramming	Metatranscriptomics, Physiological, Anatomical	Morales-Cruz et al. 2018a,b; Dell'Acqua et al. 2024	Strong	Partial	Replicated
Phase IV: Symptom expression	Field surveys, Physiological, Anatomical	Lecomte et al. 2012, 2024; Dell'Acqua et al. 2024	Strong	Correlative	Established
Phase V: Chronicity/Recovery	Longitudinal observations, Physiological	Dewasme et al. 2022; Dell'Acqua et al. 2024	Moderate-Strong	Correlative	Ongoing
Pathogen presence in both states	Metabarcoding, Culture	Hofstetter et al. 2012	Very Strong	Correlative	Established
Reduced microbial diversity → symptoms	Metabarcoding, Statistical	Bruez et al. 2020	Strong	Correlative	Replicated
Virulence gene upregulation	Metatranscriptomics	Chambard et al. 2025	Strong	Correlative	Replicated
Wood decay enzyme activation	Metatranscriptomics, Enzymatic	Morales-Cruz et al. 2018b	Strong	Correlative	Replicated
Metabolic niche restructuring	Metabolomics	Lima et al. 2017	Moderate-Strong	Correlative	Replicated
Epigenetic marks (systemic)	Epigenomics (WGBS)	Berger et al. 2025	Moderate	Correlative	Single study
Drought → microbiome collapse	Experimental manipulation	Gastou et al. 2026	Strong	Partial	Experimental
Hydraulic dysfunction	Physiological measurements, Anatomical analysis	Bortolami et al. 2019, 2021a; Dell'Acqua et al. 2024	Very Strong	Partial	Replicated
Toxin production hypothesis	Biochemical, *In vitro*	Andolfi et al. 2011	Moderate	Not demonstrated	Inferred
Functional thresholds (LSSIM)	Conceptual model	Del Frari et al. 2022	Weak (hypothesis)	Not demonstrated	Proposed
Compensatory xylem develpment	Physiological, Anatomical, Experimental	Dell'Acqua et al. 2024	Strong	Partial	Experimental

NOTES:
- Evidence Level: Strength of empirical support based on number of studies, sample sizes, and consistency of findings.
- Causality: Whether relationship is correlative, partially causal (experimental evidence), or causal (demonstrated)
- Validation Status: Degree of independent replication and experimental validation.
- Correlative evidence: Strong associations observed but causality not experimentally demonstrated.
- Partial causality: Some experimental evidence (e.g., drought manipulation) but full causal chain not established.
- Preprint data (Berger et al. 2025): Included for novelty but marked as single study requiring validation.

INTERPRETATION:
- Most model components have STRONG correlative evidence from multiple independent studies.
- Few components have DEMONSTRATED CAUSALITY (requires targeted manipulation experiments).
- This is acknowledged in main text with explicit statement on causality vs. correlation (Section Conclusions and Outlook).
- The temporal model should be viewed as a mechanistically plausible, evidence-supported conceptual framework that generates testable prediction, rather than a definitively proven causal mechanism.

Evidence Level
- Very strong
- Strong
- Moderate-Strong
- Moderate
- Weak

Causality
- Correlative
- Partial
- Not demonstated

Validation Status
- Established
- Replicated
- Experimental
- Single study
- Ongoing
- Inferred
- Proposed

evidence for this functional reprogramming is strong and reproducible, though direct causal tests (e.g. gene disruption) are still lacking.

Phase IV: Symptom expression and functional thresholds (Fig. 3, red zone).
Foliar symptoms arise once key physiological thresholds are crossed. Their appearance reflects the combined impact of fungal activity (toxins, enzymes), vascular impairment (hydraulic decline, vessel blockage), and environmental triggers such as heat or high evaporative demand (Lecomte et al. 2012, Bortolami et al. 2019, Claverie et al. 2020, 2021a, Del Frari et al. 2022). Acute tiger-stripe symptoms often coincide with periods of high transpiration, whereas chronic symptoms signal long-term hydraulic decline. According to Table 1, symptom patterns are well documented, but the underlying mechanisms remain debated: both toxin-based and hydraulic hypotheses have moderate support, yet neither is conclusively demonstrated.

Phase V: Chronicity, recovery or collapse (Fig. 3, divergent pathways).
After symptom onset, vines may follow different trajectories: progressive decline (solid line in Fig. 3), partial recovery (dashed line) or sudden collapse (apoplexy). Recovery typically depends on stress relief, re-establishment of holobiont stability and cultivar-specific resilience. Recent evidence indicates that vines retain a capacity to regenerate functional xylem vessels even after hydraulic disruption: symptomatic stems show increased density of non-occluded vessels in the outermost (most recent) xylem tissue, often with smaller, hydraulically safer vessel diameters, enabling partial compensation for vascular dysfunction (Dell'Acqua et al. 2024). In contrast, recurrent symptoms and vascular dysfunction may lock vines into chronic decline. Table 1 shows that this phase is supported mainly by longitudinal field observations; however, the mechanisms governing recovery or long-term collapse remain poorly resolved.

The temporal model depicted in Fig. 3 emphasizes that esca is not a linear progression from infection to disease, but rather emerges through long-term feedbacks between colonization history, microbiome restructuring and host stress physiology. The model generates testable predictions for each phase transition, as detailed in Table 1, including specific experiments needed to establish causality for currently correlative relationships.

Testable predictions for microbiome-informed disease ecology

The holistic model proposed here generates several testable predictions that can guide future work on esca. One expectation is that vines which later develop symptoms should already show early signs of dysbiosis—lower microbial diversity, simplified co-occurrence networks and a shift toward pathotroph dominance—well before any visual expression (Leal et al. 2024a, Monod et al. 2025a, Bruez et al. 2020). It also follows that practices which mitigate environmental stress, such as targeted irrigation to avoid severe drought stress, canopy shading to reduce evaporative demand, or soil management to improve nutrient availability and root health, should reduce the likelihood of transitions from latency to destabilization and symptom onset, even in vines already colonized by esca-associated fungi (Bortolami et al. 2021a, Calvo-Garrido et al. 2021, Lecomte et al. 2024). A third prediction is that effective biocontrol should not only suppress pathogen abundance but also favour the re-establishment of more complex and modular microbial networks enriched in antagonistic taxa (Leal et al. 2024a, Niem et al. 2020, Bettenfeld et al. 2022).

Host genotype plays a key role, though recent multi-omics analyses across diverse cultivars reveal that varietal differences in esca susceptibility are driven primarily by wood biochemical

composition, particularly extractive and hemicellulose content, rather than by constitutive anatomical traits or microbiome composition (Gastou et al. 2026 b, c). Cultivars with higher extractive content and lower hemicellulose proportions display reduced esca incidence and white-rot necrosis, suggesting that wood chemistry directly influences pathogen establishment and activity (Gastou et al. 2025a). Moreover, susceptible cultivars exhibit stronger metabolic responses to esca, including elevated accumulation of phenylpropanoids, terpenes, and lipids (Gastou et al. 2025b). While earlier studies suggested that cultivars capable of maintaining stable microbiomes and vascular function under stress might display lower esca incidence (Pouzoulet et al. 2017, Bekris et al. 2021, Azevedo-Nogueira et al. 2025), the recent evidence indicates that wood chemistry and vascular regeneration capacity (Dell'Acqua et al. 2024) are stronger determinants of disease outcomes than microbiome stability per se. Genetic mapping may help identify loci involved in extractive biosynthesis, hemicellulose regulation, and traits supporting holobiont stability (Vandenkoornhuyse et al. 2015). Finally, integrated omics approaches, combining metagenomics, transcriptomics and metabolomics, should allow discrimination among disease phases, enabling the development of diagnostic markers for early detection and risk forecasting (Morales-Cruz et al. 2018b, Monod et al. 2025b, Massonnet et al. 2018, Azevedo-Nogueira et al. 2022, Chambard et al. 2025). Confirming these predictions would help move esca research beyond descriptive pathogen surveys toward a more predictive, mechanistic disease ecology (Desprez-Loustau et al. 2006, Lecomte et al. 2024).

Implications for management and future research

From pathogen eradication to holobiont management

The pathobiome-holobiont perspective calls for reorienting esca management beyond attempts to eliminate individual pathogens. Classical practices such as sanitation and pruning-wound protection (Gramaje et al. 2018) help limit new infections but do not address the ecological imbalance that underlies disease development (Desprez-Loustau et al. 2006, Bettenfeld et al. 2020, 2022). In this context, Table 2 outlines an integrated management framework that links preventive measures, monitoring tools and intervention strategies to different vineyard stages.

Stress mitigation

Abiotic stress plays a central role in holobiont destabilization. Although water deficit may temporarily suppress foliar symptoms, it also promotes trunk-wood dysbiosis by reducing fungal diversity and favouring trunk pathogens (Leal et al. 2024a, Gastou et al. 2026). Practices that moderate hydraulic stress (e.g. targeted irrigation during periods of high evaporative demand, soil management that improves root function and buffering capacity against extreme water deficits, while avoiding prolonged waterlogging, or canopy cooling) help stabilize both host hydraulics and the endophytic microbiome (Bortolami et al. 2021a, Calvo-Garrido et al. 2021). Dell'Acqua et al. (2025a) further showed that nitrogen deficiency reduced esca incidence without altering fungal community composition, suggesting that host-driven shifts in transpiration, morphology and phenylpropanoid accumulation can modulate symptom expression independently of pathogen abundance.

Microbiome support and manipulation

Strengthening beneficial microbial assemblages can enhance holobiont resilience. Endophytic communities provide growth-promoting and defence-related functions, including hormone modulation, antioxidant activity, ACC deaminase production, VOC signalling and immune priming, that help buffer vines against stress (Pacifico et al. 2019). Biocontrol agents such as *Trichoderma* spp. or beneficial bacteria show variable performance depending on environment and microbial context (Pertot et al. 2016, Berbegal et al. 2020, Bigot et al. 2020, Niem et al. 2020, Visagie et al. 2023, Leal and Gramaje 2024, Leal et al. 2024b, Abarquero et al. 2025). Network analyses reveal consistent negative associations between certain endophytes and trunk pathogens (Azevedo-Nogueira et al. 2025), reinforcing the value of management strategies that maintain or restore diverse, modular and functionally redundant communities.

Host-oriented strategies

Recent multi-omics evidence indicates that cultivar selection should prioritize wood biochemical composition, particularly high extractive content and low hemicellulose proportions, as these traits appear to be stronger determinants of esca susceptibility than vessel anatomy or microbiome composition alone (Gastou et al. 2026 b, c). Similar patterns emerge in Eutypa dieback, where cultivar resistance is associated with specific phenolic profiles (stilbenoids vs. proanthocyanidins) rather than total phenolic content (Sinclair et al. 2025), suggesting that wood chemical composition may be a common determinant of susceptibility across grapevine trunk diseases. Cultivar-rootstock combinations that support vascular regeneration capacity following hydraulic disruption may also enhance recovery potential (Dell'Acqua et al. 2024). While stable microbiomes and resilient hydraulic traits remain relevant under climate stress (Pouzoulet et al. 2017, Azevedo-Nogueira et al. 2025), breeding and selection programs should prioritize wood chemical traits that directly limit pathogen activity. Nursery practices also remain critical for minimizing early colonization while supporting beneficial endophytes (Gramaje and Armengol 2011).

Altogether, these insights shift the emphasis from eliminating pathogens to maintaining holobiont stability and resilience (Vandenkoornhuyse et al. 2015, Bettenfeld et al. 2020). The repeated observation that symptom expression can be decoupled from pathogen abundance, whether under drought or nitrogen deficiency, highlights that durable management must target holobiont function rather than pathogen presence alone.

Research priorities in the era of multi-omics and climate change

Several priorities emerge from this ecological reconceptualization of esca:

(i) *Mechanistic integration of omics and physiology.* Future studies should integrate metagenomics, metatranscriptomics, metabolomics, and detailed plant hydraulic/physiological measurements on the same vines (Morales-Cruz et al. 2018b, Leal et al. 2024a, Massonnet et al. 2018, Chambard et al. 2025,

Table 2 Integrated management framework for esca disease.

Phase	Vineyard age	Primary strategy	Specific actions	Evidence level
PREVENTIVE	NurseryYoung (1–5 years)	Minimize colonizationEarly detection	• Cleaning of nursery compartments • Dips for cuttings in chemicals/BCA • Certified low-pthogen nursery stock • Molecular diagnostics (qPCR, ddPCR, NGS) • Wound protectants • Avoid overcropping	Moderate
MONITORING	Established (5+ years)	Early detection	• Wound protectants • Annual symptom surveys • Microbiome profiling for dysbiosis • Remote sensing (multispectral, thermal) • Precision viticulture platforms	Moderate/emerging
INTERVENTION	Symptomatic or high-risk	Holobiont stabilization	• Supplement irrigation during stress • Canopy management • Trunk surgery (valuable vines only) • Curettage • Removal/replanting (severe cases)	Variable
ADAPTIVE	All ages	Continous improvement	• Evaluate intervention effectivenness • Adjust based on climate/disease pressure • Regional monitoring networks • Knowledge sharing	Ongoing

Hrycan et al. 2025). Multi-omic integration is crucial to disentangle abundance, activity, and functional impact (Gastou et al. 2026).

(ii) *Longitudinal and experimental designs.* Disease expression depends on temporal dynamics, including stress exposure, microbial activity, and host responses. Long-term monitoring of vineyards, combined with experimental manipulations of drought, heat, and co-infections, will be essential to capture transitions between latent, chronic, and apoplectic phases (Bortolami et al. 2021a, Monod et al. 2025b, Lecomte et al. 2012, 2024).

(iii) *Microbiome-based diagnostics and forecasting.* Diagnostic markers based on community structure, dysbiosis indicators or network architectures could enable early detection of holobiont destabilization (Monod et al. 2025a, Bruez et al. 2020, Bekris et al. 2021, Azevedo-Nogueira et al. 2022). Integrating these markers with climate and soil data may improve risk forecasting under climate change.

(iv) *Ecological foundations of biocontrol.* To improve biocontrol efficacy, research must clarify how introduced agents integrate into existing microbial networks, how competitive or mutualistic interactions shift across regions and cultivars, and how environmental stress modulates these interactions (Niem et al. 2020, Bettenfeld et al. 2022, Lv et al. 2023). Findings from Azevedo-Nogueira et al. (2025) showing region- and genotype-dependent pathogen-endophyte correlations provide a foundation for context-specific biocontrol design.

(v) *Holobiont-informed breeding.* Breeding programs should incorporate traits related to microbiome assembly, network stability, endophytic colonization patterns, and drought resilience, alongside classical resistance traits (Vandenkoornhuyse et al. 2015, Pouzoulet et al. 2017). Genotypes showing stable trunk microbiomes across contrasting environments may be better adapted to future climatic variability.

(vi) *Broadening the stress landscape.* Most experimental work has focused on drought and temperature stress. Future research should systematically evaluate other abiotic (nutrient imbalances, soil chemistry, salinity) and biotic (viral co-infections, nematodes, root pathogens) stressors, as well as their synergistic interactions, to determine which factors are most critical in triggering holobiont destabilization and symptom expression across diverse viticultural contexts.

Climate change will intensify environmental pressures and likely exacerbate trunk disease dynamics, though effects will differ among fungal species with contrasting biology (Desprez-Loustau et al. 2006). Drought intensification may favour stress-responsive ascomycetes such as *P. chlamydospora*, driving wood necrosis and vascular dysfunction (Gastou et al. 2026, Chambard et al. 2025), whereas increased frequency of wet, warm periods may promote basidiomycete activity, particularly *F. mediterranea*, accelerating white-rot progression and foliar symptom expression (Etienne et al. 2026). The net effect on esca incidence will depend on the balance between these opposing climatic drivers and their interaction with host genotype and microbiome composition. Adopting a holobiont-centred approach, supported by multi-omics, ecological theory and genotype-environment interactions, is therefore both conceptually necessary and practically urgent.

Conclusions and outlook

Esca continues to challenge a pathogen-centred disease framework. Although certain fungi are consistently associated with wood necroses (Luque et al. 2009, Choueiri et al. 2014, Díaz and Latorre 2014, Elena et al. 2018), classical inoculations seldom reproduce the full foliar syndrome (Larignon and Dubos 1997, Mugnai et al. 1999, Sparapano et al. 2000, 2001, Gramaje et al. 2010). Multi-omics studies increasingly show that esca arises from shifts

in microbial activity, host physiology, and stress responses rather than pathogen presence alone (Morales-Cruz et al. 2018b, Leal et al. 2024a, Massonnet et al. 2018, Bruez et al. 2020, Chambard et al. 2025).

The pathobiome and holobiont concepts reconcile these patterns by framing esca as a stability disorder of the grapevine holobiont (Zilber-Rosenberg and Rosenberg 2008, Vayssier-Taussat et al. 2014, Vandenkoornhuyse et al. 2015, Bass et al. 2019). Stress-driven dysbiosis, conditional pathogenicity, and cumulative vascular dysfunction interact over time to generate the intermittent and unpredictable symptoms typical of the disease (Bortolami et al. 2021a, Monod et al. 2025b, Desprez-Loustau et al. 2006, Lecomte et al. 2012, 2023, 2024). This view aligns esca with other woody decline syndromes and emphasises shared ecological principles such as network stability and host-microbiota-environment feedbacks (Spigaglia et al. 2020, Carluccio et al. 2025).

Management approaches should therefore prioritize holobiont resilience (stress mitigation, microbiome-supportive strategies and host traits linked to hydraulic and microbial stability) over attempts to eliminate individual pathogens (Bettenfeld et al. 2022). Within this microbiome-oriented perspective, there is particular value in supporting the natural, locally adapted microbiota already present in vineyards: preserving endemic endophyte pools, minimizing disruptive practices, and favouring nursery and field management that transmit and maintain beneficial local communities (Giffard et al. 2022). Research, in parallel, must integrate longitudinal designs with multi-omics to disentangle microbial abundance, activity, and functional impact (Morales-Cruz et al. 2018b, Massonnet et al. 2018).

A key limitation, however, is that most current evidence remains correlational. The associations highlighted in this review, connecting microbial assemblages, host physiology, environmental constraints, and symptom expression, are compelling but rarely causal. Addressing this gap will require targeted experimentation: (i) microbiome transplantation or synthetic communities to test whether pathogen-biased consortia can induce symptoms; (ii) functional disruption of virulence genes in key taxa; (iii) controlled environmental manipulations paired with time-series multi-omics to resolve event sequences; and (iv) epigenetic engineering or chemical modulation of DNA methylation to test its role in symptom development (Morales-Cruz et al. 2018b, Lv et al. 2023, Berger et al. 2025). Until such studies become routine, the pathobiome framework should be regarded as a coherent and testable model rather than a complete mechanistic explanation.

Grapevine, with its genomic resources and tractable experimental systems, is well suited to advance our understanding of decline syndromes in woody perennials (Deyett and Rolshausen 2020, Bettenfeld et al. 2022). Viewing esca as a pathobiome-driven holobiont disorder, and treating this framework as a set of hypotheses to be tested under climate change, offers a more predictive foundation for future vineyard resilience.

Acknowledgements

We gratefully acknowledge the researchers from diverse disciplines whose work on grapevine esca has substantially enriched our understanding of this complex disease. We also gratefully acknowledge all active members of the International Council on Grapevine Trunk Diseases (ICGTD).

Author contributions

David Gramaje (Conceptualization, Validation, Visualization, Writing – original draft, Writing – review & editing), Ales Eichmeier (Conceptualization, Visualization, Writing – original draft, Writing – review & editing).

Conflicts of interest

None declared.

Funding

This work was supported by the following grants:

VITicultura RESiliente en el territorio POCTEFA (VITRES), EFA033/01, European Regional Development Fund.

Rhizobiome improvement to mitigate grapevine root diseases under drought stress (RHIZOIMPROVE), Ministry of Science, Innovation and Universities, Spain (PID2023-147360OR-C32).

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Received: 10 December 2025. **Revised:** 2 March 2026. **Accepted:** 12 March 2026