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Enhancing Plantation Forest Sustainability: A Review of *Eucalyptus* Defence Mechanisms to Foliar Fungal Pathogens

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Abstract

Purpose of the Review In this review, we synthesize knowledge generated over many decades on the main defence responses of *Eucalyptus* to fungal leaf pathogens with the aim of identifying targets for breeding disease tolerant trees. We highlight physiological and molecular traits associated with host defence in relation to pathogen life-style. Overall, the purpose of this review is to identify resistance mechanisms that offer improved resilience of *Eucalyptus* plantations in the face of increasing threats by foliar fungal pathogens. The broad aim is to promote sustainable forestry through appropriate selection of resistance traits in trees that are widely planted for commercial timber production.

Recent findings *Eucalyptus* is among the most important tree genera planted for commercial timber production worldwide. Numerous foliar pathogens have been reported on these trees in the last 30 years with numbers of recent reports increasing exponentially. The majority of these diseases affect the leaves and shoots of the trees. Knowledge on resistance traits in *Eucalyptus* to fungal foliar pathogens is limited. This is in part due to the high intra- and inter-species variation in molecular and physiological responses of the host and variation in responses to different pathogens, especially those with different trophic modes. A well-founded understanding of such host responses will provide valuable knowledge required to maintain healthy, sustainable *Eucalyptus* plantations, especially in the face of changing environmental conditions, where new diseases are caused by fungi previously not considered relevant.

Summary Foliar diseases are among the most important challenges for *Eucalyptus* plantations globally. The effects of climate change and new or more serious outbreaks present an important threat to the sustainability of *Eucalyptus* plantations worldwide. Due to restrictions on the use of chemicals, more feasible solutions for disease management lie in selecting planting material with resistance traits. To achieve that goal, it is essential to understand the most important physiological and molecular responses of *Eucalyptus* to infection by pathogens that infect their foliar tissues. In this review we summarise the available knowledge of the main physiological defence barriers and genetic traits that play key roles in the broad defence against foliar fungal pathogens. Furthermore, we consider defence pathways that are specifically related to the lifestyle and trophic mode of the pathogens. In order to ensure the future sustainability of *Eucalyptus* plantations, it will be necessary to understand how disease resistance is affected by climate change, as well as the adaptability of the hosts and pathogens to newly emerging environmental conditions.

Keywords *Eucalyptus* · Foliar fungal pathogens · Disease resistance · Management · Forest diseases · *Austropuccinia psidii* · *Calonectria pseudoreteaudii*

Introduction

The global demand for wood and wood products has increased significantly in recent decades. The only sustainable timber sources to meet this demand are high-yielding forest plantations [1, 2]. Consequently, global plantations have expanded by an average of 4.4 million ha annually from 168 million ha in 1990 to nearly 278 million ha in 2015 [3, 4] and are predicted to expand further by 25–67 million by 2030 [5].

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Eucalyptus species are native to Australia and nearby islands [6]. They are dominant, fast-growing hardwood trees, planted commercially in the tropics, temperate regions of the southern hemisphere and the Iberian peninsula due to their high productivity and adaptability to a large range of soil and environmental conditions [7–9]. Their ability to yield substantial economic returns, typically over short rotations, has led many countries with mild to tropical climatic conditions to select these trees for their plantation forestry programmes [9]. In these introduced ranges, *Eucalyptus* plantations currently cover over 22 million hectares worldwide [10].

Amongst the most important threats to forest plantations are pests and pathogens. *Eucalyptus* species are not highly susceptible to any particular pathogen in their natural range [11]. In these situations, coevolution between pathogens and their hosts, together with pressures from competition, predation, and parasitism, contribute to the stability of a natural ecosystem [12]. Plantations of non-native *Eucalyptus*, at the early establishment stages, are typically free of diseases. This period can, however, be short-lived, as pathogens and pests usually follow their hosts into their new environments or, in some cases, the transient disease free stages in non-native *Eucalyptus* plantations is interrupted due to host shifts by native pathogens, becoming an important constraint to the sustainability of *Eucalyptus* plantations [11, 13, 14].

Several recent studies have shown that the global movement of germplasm, particularly of living plants, seeds as well as plant products such as untreated timber, are amongst the most important sources for the spread of plant pathogens and pests globally [15–21]. Leaf pathogens are among the more common pathogens appearing in plantations of non-native *Eucalyptus* species, and are in fact considered the principal invaders of exotic *Eucalyptus* plantations [11, 22].

Established technologies such as breeding and selection of resistant species and hybrids, and vegetative propagation of high-quality clones has already substantially reduced the impact of foliar diseases [14]. The first studies considering resistance of *Eucalyptus* to foliar pathogens in the 20th century utilised microscopy and biochemical assays [23]. Later, the availability of the *Eucalyptus grandis* genome [24] provided new opportunities to study responses to these diseases at the molecular level. This has been further supported by knowledge of the signalling pathways that lead to active defence against different groups of pathogens in model plants such as *Arabidopsis thaliana* [25, 26]. Currently, one of the most informative approaches to elucidate resistance mechanisms in non-model plants, such as *Eucalyptus*, is using RNA-sequencing, which involves studying the genetic response networks of plants during infection [27–29].

Resistance mechanisms of *Eucalyptus* to fungal pathogens that affect the shoots and leaves (foliar pathogens) have not been extensively studied. While there are numerous reviews on plant fungal interactions [30–32], this review specifically considers *Eucalyptus* pathosystems and leaf and shoot pathogens of relevance to southern hemisphere plantations. *R*ecent findings on *Eucalyptus* defence responses and resistance mechanisms against pathogens with different life styles that represent important threats to *Eucalyptus* plantations, such as for example the biotrophic pathogen *Austropuccinia psidii* and the necrotrophic pathogen *Calonectria pseudoreteaudii* will be highlighted.

Fungal Foliar Diseases Threaten *Eucalyptus* Plantation Sustainability

The most economically important foliar diseases of *Eucalyptus* plantations include myrtle rust [33, 34], Calonectria leaf blight [35–37], Teratosphaeria leaf blight (TLB) and Mycosphaerella leaf disease (MLD).

[38–42].

Myrtle rust caused by *Austropuccinia psidii* is a native pathogen of Myrtaceae in South and Central America that underwent a host-shift [43] to infect *Eucalyptus* that have been introduced into that region to establish timber plantation trees [34]. Myrtle rust was one of the first serious diseases to affect *Eucalyptus* in Brazilian plantations with the first report of the disease by Joffily [44]. The average impact of the disease on stem growth is 20% in *Eucalyptus*, but the pathogen has been reported to cause losses of more than 60% stem volume on nutrient-stressed trees generating important economic losses [45–47].

Infection by *A. psidii* becomes visible when yellow pustules appear on the leaves and shoots, and symptoms include necrosis and leaf deformation, affecting mainly young trees less than 3–4 m tall [48]. Susceptibility of *Eucalyptus* to this rust is dependent on leaf development, where younger leaves and shoots are more susceptible. When leaves achieve 50–60% of their final length, they are more resistant to infection by *A. psidii*, with few pustules visible on the leaf surface [49].

Recently, a resistant *E. grandis* × *E. urophylla* clone widely planted in Brazil in 2013 was affected by a highly aggressive race of *A. psidii* [50], illustrating the continual evolution of the pathogen. It is of concern that the pathogen has spread worldwide and has been reported in Japan [51], Australia [33], South Africa [52], Uruguay [53], Colombia [54], Indonesia [55], Singapore and New Zealand [56].

Calonectria leaf blight (CLB) is also considered an aggressive disease on *Eucalyptus* plantations particularly in tropical and subtropical areas of the world [36, 37, 57]. The symptoms of CLB include damping-off and collar-rot of nursery plants, leaf spot, root rot, shoot blight, and stem canker [57]. Different species of *Calonectria* cause CLB and this differs in different parts of the world. Some that represent a threat to *Eucalyptus* plantations include *Calonectria pseudoreteaudii* [58], *C. pteridis* [59] and *C. spathulate* [37].

C. pseudoreteaudii in one of the most widely-distributed and aggressive species [60]. Leaf symptoms begin with water-soaked lesions, which rapidly lead to necrosis under high humidity, resulting in severe defoliation and eventually tree death [61]. It is estimated that annual economic losses due to this disease are over $7.8 million in Fujian alone [62].

Leaf blotch and defoliation caused by various species in the Teratosphaeriaceae and Mycosphaerellaceae [63] reduce the photosynthetic area, which negatively affects growth and can lead to malformation of the tree and productivity losses in affected plantations [41, 64–67]. Most of these species are apparently facultative pathogens and do not cause significant disease [68]. However, three species of *Teratosphaeria* including, *Teratosphaeria nubilosa*, *T. cryptica* and *T. destructans* are highly aggressive pathogens, mainly affecting juvenile and intermediate foliage, causing premature defoliation and, during severe infections, shoot blight and branch death [38, 40–42]. For example *T. nubilosa* can cause leaf area losses of up to 80% in *Eucalyptus globulus* plantations [69, 70]. *Teratosphaeria destructans*, first recorded in Indonesia [71] has caused serious losses in *Eucalyptus* plantations in various south east Asian countries [72] and in South Africa has reached incidence levels above 90% in subtropical areas affecting both nurseries and young plantations.

The impact of foliar diseases on *Eucalyptus*, the rapid spread of their causal agents between continents and increasing reports of new disease outbreaks, for example *Elinoe necatrix* in Indonesia [73] and *Elsinoe masingae* in South Africa [74], requires the urgent development of suitable management strategies. However, due to costs and environmental impacts, management options at plantation scale are limited to sustainable cultural practices and planting resistant varieties. The availability of genetic resources, including chip-based genotyping tools [75] allow targeted breeding of trees with higher resistance to fungal foliar pathogens. However, targets for breeding resistant trees are currently still limited. Exploring the knowledge gained from previous research on the morphological adaptations and molecular responses of *Eucalyptus* to infection by fungal foliar pathogens, should provide clues to identifying genetic targets for breeding trees with high resilience to leaf and shoot diseases.

A First Line of Defence: Preformed and Induced Anatomical Barriers

Leaf Cuticles and Epicuticular Waxes

The first physical barrier to foliar pathogens is the wax layer on the leaf surface (Fig. 1), which has been shown to provide strong protection to biotrophic and hemibiotrophic pathogens. The wax layer is hydrophobic and reduces the leaf wetness which is necessary for pathogen germination [76]. Studies on the structure of waxes in different species of *Eucalyptus* have shown that the length, diameter of wax tubes, and distribution varies between species and the age of the leaf [77, 78]. Ontogenic variations in the thickness of the wax layers could explain why many *Eucalyptus* foliar pathogens only infect younger leaves. Pathogens that penetrate *via* stomata are more likely to overgrow stomata on leaves with thicker wax layers rather than penetrating and infecting them [79]. For example, in *E. globulus*, a wax layer covering the stomata restricted infection by *T. nubilosa*, a pathogen that enters via the stomata [80].

In the interaction between *Eucalyptus grandis* and *A. psidii*, where the pathogen infects via direct penetration [81], the thickness of the waxy layer and the structure of the wax on the surface of old leaves resulted in low levels of adhesion of urediniospores and restriction of the development of appressoria [82]. The resistance of *Eucalyptus* to *A. psidii* was also shown to be influenced by wax abundance, its composition and morphology, where parallel and uniform leaf wax platelets were associated with host resistance [83]. Waxes can also increase resistance to foliar pathogens by inhibiting spore germination as has been reported in *Eucalyptus bicostata* against *T. epicoccoides* [23] and recently reported in *Eucalyptus grandis* x *urophylla* against *T. destructans* [84]. Leaf waxes are, therefore, important traits that influence *Eucalyptus* resistance against foliar pathogens and should be considered as targets for resistance breeding.

Cuticle thickness also plays a role in fungal penetration of leaves. For example, in the interation between *Eucalyptus* and the necrotrophic *C. pseudoreteaudii*, the thickness of the cuticle was an important factor determining resistance to this pathogen [85]. The pathogen also showed high levels of cutinase gene expression at an early infection stage [85], suggesting that necrotrophic pathogens that penetrate leaves directly must invest significant energy in breaking down this defence.

Leaf Cellular Structure and Organization

Variation in specialized leaf cells such as palisade and spongy parenchyma ratios, airspace, and sclerified tissues, also contribute to foliar pathogen resistance (Fig. 1) [86, 87]. Park and Keane [88] showed that the hyphae of some species of *Teratosphaeria* (previously *Mycosphaerella*) may not be able to penetrate the intercellular spaces of tightly packed cells such as those in palisade mesophyll layers. Isobilateral palisade mesophyll layers with lignin deposition in *E. nitens* also provided a barrier for hyphal proliferation of *T. nubilosa*. In contrast, juvenile *E. globulus* leaves are thought to be more susceptible to *T. nubilosa*, because they have only one layer of palisade mesophyll [89]. Mesophyll cells of *E. nitens*, in contrast, can undergo cell division including anaplasia, where cells

that are already differentiated can revert to embryonic, meristematic cells [89]. This de-differentiation, was not observed in the susceptible *E. globulus* [89]. Recently, a study using different *E. globulus* genotypes reported that resistance to *T. nubilosa* was also related to a greater percentage leaf mass per area (LMA) and fewer airspaces in the palisade parenchyma [90]. Thicker leaves with multiple layers of tightly packed palisade parenchyma cells with additional sclerification, seem to be highly effective barriers against infection by Teratoshaeriaceae pathogens. However, as cellular organization is controlled by many different loci [91] it could be difficult to select for these traits in resistance breeding programs.

Stomata

Stomatal position and their abundance on the leaf surface have been reported as a preformed barrier against several pathogens that penetrate directly through these structures *(*Fig. 1*)*. For example, *Eucalyptus* resistance to *Mycosphaerella fijiensis* [92], frog-eye leaf spot (*Cercospora sojina*) [93] and *Mycosphaerella berkeleyi* [94] was associated with lower stomatal densities. Resistance facilitated by lower stomatal numbers can be enhanced by a thick wax coverage of the leaf cuticle [95]. This is also considered an important factor in the resistance of *E. globulus* against *T. nubilosa* [80]. Migacz et al. [77] suggested that stomatal distribution on the leaf surface together with other anatomical features plays a significant role in the resistance of different *Eucalyptus* species to foliar diseases. However, similar to the cellular organization discussed above, stomatal density is mostly a multigenic trait [96] and thus selection of breeding material with low stomatal density could be challenging.

Constitutive Phytochemicals

Chemical inhibitors of fungal growth on the leaf surface such as phenolic compounds, including flavonoids and catechins, have been reported as barriers for pathogens on *Eucalyptus* leaves [89]. Essential oils have also been widely reported to function as preformed chemical defences [97]. They consist of a complex mixture of terpenes, predominantly monoterpenes and sesquiterpenes, which are produced and stored in subdermal secretory cavities or "sebaceous glands" [98]. The defensive action of essential oils can be via direct toxicity to microbes or indirect priming of systemic defences in both the host and neighbouring plants [99, 100]. The foliar secretory cavities of various *Eucalyptus* species have been shown to contain not only volatile essential oils but also non-volatile resinous compounds with unknown function, which could play an important role in *Eucalyptus* defence against pathogens [101]. Furthermore, *Eucalyptus* essential oils have been reported to reduce mycelial growth [102] and inhibit spore production of *Pythium aphanidermatum* [102, 103] and *Colletotrichum* sp. [104].

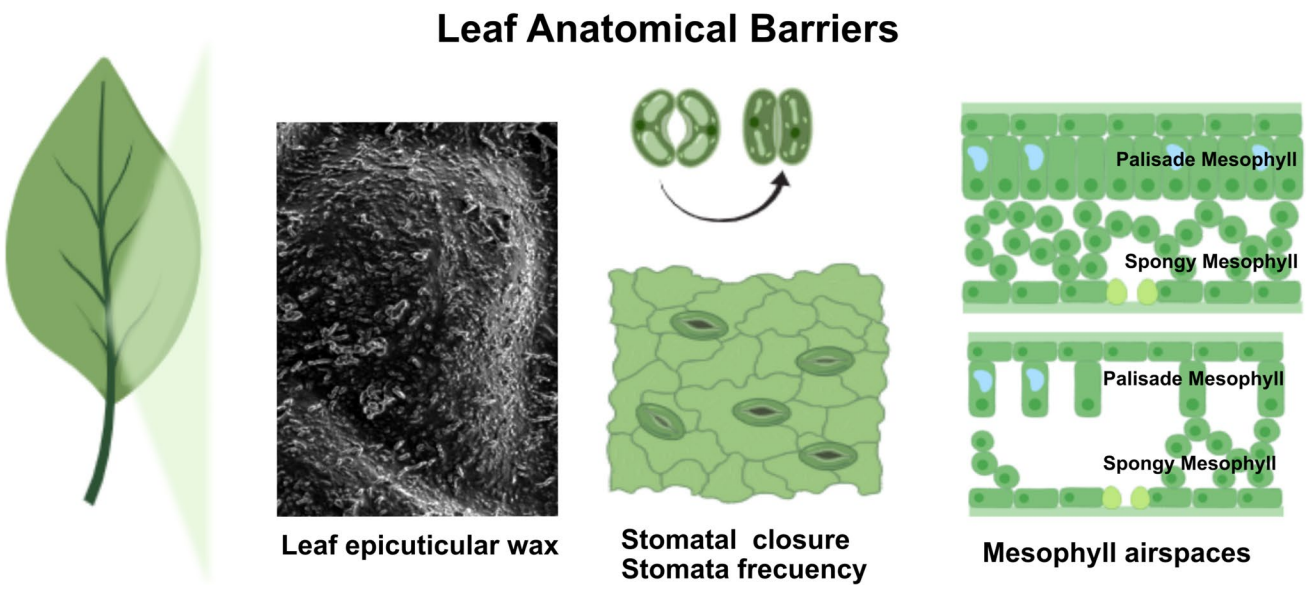


Fig. 1 Main preformed barriers of *Eucalyptus* associated against fungal foliar pathogens. Constitutive defence responses mainly include physical barriers such as leaf wax morphology (density and composition), stomata and their closure to inhibit pathogen penetration. Mesophyll density and lower number of airspaces can prevent pathogen spread, thereby restricting the colonization

Innate Immunity of *Eucalyptus*

Three Main Components of Plant Immunity

After pathogens overcome the leaf preformed barriers, inter and/or intracellular host colonization takes place. Plants have an innate immune system to perceive and respond to different pathogen infection strategies [30, 105–107]. Overall the prevalent defence mechanisms have three main biomolecular components; pathogen-associated molecular pattern (PAMP)-triggered immunity (PTI) [108], secondary defences known as effector-triggered immunity (ETI) [109], and additional defences known as exosome-mediated cross-kingdom RNA interference (CKRI) [110].

Pattern Triggered Immunity (PTI)

Pattern Triggered Immunity (PTI), is based on the "zig-zag" model by Jones and Dangl [30], where plants recognize pathogens via pattern recognition receptors (PRRs). These receptors are located on the cell surface and detect PAMPs, which are conserved pathogen or microbial components, such as chitin or exopolysaccharides [111]. Some PRRs can also recognize damage-associated molecular patterns (DAMPs), which are molecules secreted by the plants as a result of exposure to biotic and abiotic stresses [112]. PAMPs and DAMPs trigger the first line of immunity, known as PTI. Structurally the main receptors located on the plant cell surface are receptor-like kinases (RLK) and receptor-like proteins (RLP) [31]. There are a number of RLKs/RLPs involved in plant immunity, which have been well summarized by Tang et al. [113].

In *Eucalyptus*, the main receptors known to be involved in pathogen recognition are leucine-rich repeat transmembrane protein kinases (LRR-RLK). During the interaction between *Eucalyptus* and the necrotrophic pathogen *C. pseudoreteaudii* [102], a *LRR-RLK* receptor gene (*Eucgr. F01306.1*) was up-regulated in a resistant *E. urophylla* × *E. tereticornis host* (Fig. 2B). The expression of this receptor was also validated by qRT-PCR, where it was not detected in the susceptible *E. grandis* host [114]. In the same study, higher expression of a *calmodulin-domain protein kinase 9 gene* (*CDPK*, *Eucgr.F00761.1*) was reported in the resistant host [114]. CDPK is an important factor in activating the immune reactions of plants, such as the synthesis of reactive oxygen species (ROS), changes in phytohormone synthesis, signalling (Ca^{2+}) and cell death [115]. PTI has also been studied in the interaction of *Eucalyptus* with the biotrophic pathogen *A. psidii*. The main difference between the constitutively overexpressed proteins in a susceptible and a resistant *E. grandis* host were cysteine-rich receptor genes (CRR) expressed in the susceptible host, while the resistant host expressed the *LRR-RLK* receptor genes [116]. The differences in efficiency of pathogen recognition by LRR and CRR are not understood, but could be notable targets in breeding Eucalyptus trees with resistance to foliar pathogens.

Effector Triggered Immunity/Defence

Effector triggered immunity (ETI), targets pathogens with intracellular growth, where successful pathogens release effectors into the cytoplasm [30]. Effectors are small secreted proteins (SSPs) that interfere with PTI to reprogram host cell metabolism and physiology to aid in host colonization [26, 117]. Effectors can be perceived by specific co-evolved nucleotide-binding leucine-rich receptors (NB-LRRs) in the cytoplasm. Plants possess highly variable NB-LRRs known as disease resistance proteins (R-proteins). Many R genes have been identified in *Arabidopsis*, and their role in suppressing pathogens and activating defence mechanisms have been characterized [117, 118]. The R proteins can recognize specific effectors or their products, resulting in the activation of ETI [117].

The NB-LRR proteins in plants form two classes: with terminal Toll/interleukin-1 receptors (TIR-NB-LRR) or CC coiled coil receptors (CC-NB-LRR) [105]. The diversity of *NBS-LRR* genes in the *E. grandis* genome has been investigated by Christie et al. [119] showing a higher ratio of *TIR-NB-LRR* to *CC-NB-LRR* compared to other woody plants. However, the differences in the function of TNL and CNL proteins in *Eucalyptus* remain unknown.

There is only one example where a NB-LRR protein has been clearly linked to *Eucalyptus* resistance to a foliar pathogen. A locus associated with *A. psidii* resistance in *E. grandis* was identified more than 30 years ago (*Puccinia psidii* Resistance 1). This locus encodes numerous NB-LRRs [120], which suggests that resistance against *A. psidii* might be controlled by a strong ETI response. Unfortunately, a highly virulent strain of the pathogen has recently overcome the mechanism of this defence and resistance in plantation trees has failed [50]. Thus, to breed trees with durable resistance, it will be important not only to rely on a single dominant molecular trait, such as ETI, but to diversify and include multiple mechanisms.

In the response to necrotrophic pathogens, ETI is associated with transcription of pathogenesis-related (PR) proteins such as PR3, PR4 and PR12. These lead to an accumulation of their products locally, and hence provide only local acquired resistance (LAR) [121]. In the interaction between *Eucalyptus* and *C. pseudoreteaudii*, a PR10 protein (Eucgr. A00133.1) was transcribed in a resistant host [114]. PR

proteins are commonly regulated by WRKY transcription factors through a W-box motif, which bind to WRKY factors, thus activating plant defence responses [122] (Fig. 2B). Interestingly, *WRKY33* was significantly up-regulated in the resistant host, but suppressed in the susceptible host during *C. pseudoreteaudii* infection [114]. This discovery is relevant due to the importance of *WRKY33* as a transcription factor expressed in response to necrotrophic pathogens such as *B. cinerea* and *Alternaria brassicicola* in Arabidopsis [123] and could also be an appropriate target for resistance breeding in *Eucalyptus.*

Effectors of biotrophic and hemibiotrophic pathogens may trigger a systemic signal from the point of infection to distal tissues that induces a secondary resistance response, known as SAR [124]. This activation leads to an accumulation of phytohormones, and alters the cellular redox balance causing an activation and translocation of NONEXPRESSOR OF PR1 (NPR1) from the cytoplasm to the nucleus to interact with TGA-type transcription factors (TF's) that promote transcriptional activation of genes related to pathogen defence, such as PR and WRKY encoding genes [117, 123, 125]. In the *Eucalyptus* - *A. psidii* interaction, genes involved in glutathione (GSH) metabolism and *WRKY* transcription factors were highly expressed in resistant hosts [126]. *In* particular a putative PR gene (Eucgr.C01921), showed significantly higher levels of expression in a resistant host than in a susceptible host [126] (Fig. 2A).

Pathogens with intercellular growth display effectors that are detected by receptor-like proteins (RLP) located on the cell surface [127]. This constitutes an exception to the zig-zag model [30]. Stotz et al. [127] provided the term effector-triggered defence (ETD) for this effect, and the new invasion model was refined by Kanyuka and Rudd [128]. This new model is based on the fact that not all pathogen effectors, including those of biotrophic pathogens, are delivered or translocated into the host cell cytoplasm in contrast to some effectors that can be recognized in the apoplast by extracellular receptors structurally similar to PRRs in PTI [128]. This is relevant for pathogens that colonise the extracellular spaces in plants. For example, the *Eucalyptus* foliar pathogens, *Quambalaria eucalypti* [129] and *T. destructans* [130] do not form specialized feeding structures or penetrate host cells during their life cycle. Therefore, these pathogens most likely produce apoplastic effectors to invade their hosts. Although no RLP proteins have been linked to resistance to specific leaf diseases in *Eucalyptus*, they are highly expressed in buds and shoots of *Eucalyptus* [131], which are the tissues most severely affected by *Q. eucalypti* and *T. destructans*. This is an important gap in our current knowledge and deserves furhter study.

Phytohormones Amplify the Defence Signal

The main plant mechanisms triggered by PTI and ETI to sense the presence of fungal pathogens are changes in the cellular redox status and cytoplasmic Ca^{2+} levels, modification of specific proteins (e.g., by phosphorylation) and generation of signalling molecules e.g., salicylic acid (SA), jasmonic acid (JA) and ethylene (ET) as reviewed by Tsuda and Katagiri [132]. In general, there are two mains defensive signalling routes. The SA-dependent pathway is associated with activation of SA-dependent defence mechanisms in both local and distal parts of the plants, leading to systemic acquired resistance (SAR) or rapid activation of the hypersensitive response [132, 133]. The SA-dependent signalling pathway is best described for plant interactions with biotrophic pathogens [134]. SA and JA-ET pathways appear to act antagonistically in herbaceous plants such as *A. thaliana* [135], but have been reported to act synergistically in woody plants such as poplar [136]. In herbaceous plants, SA induces redox changes, mediated by thioredoxins and glutaredoxins, which modify transcription factors (TFs) that are involved in the suppression of JA-dependent genes [137].

In Eucalyptus hosts that are resistant to A. psidii, Swanepoel et al. [126] showed that LRR proteins activated genes involved in SA signaling with the highest level of expression of these genes at two days post inoculation (Fig. 2A). Similar to the synergy observed in poplar in response to a biotrophic pathogen [136], the authors also showed that genes related to ABA, JA and ET signaling were also upregulated in resistant *E. grandis* hosts at the same time point [126]. Additionally, genes involved in BR signaling were expressed at higher levels in resistant *E. grandis*, compared to the susceptible host [126]; Fig. 2A). *The* BRI1-associated receptor kinase (BAK1) is a co-receptor of BR-insensitive 1 (BRI1) which plays a role in BR signal transduction [138]. In *E. grandis*, nine *BAK1* genes were differentially expressed in resistant hosts in response to infection by *A. psidii*, indicating a possible direct role of this receptor in resistance, as has been reported previously in tomato challenged by *Verticillium dahliae* [139] and tobacco challenged by *Phytophthora infestans* [140].

In contrast to biotrophic pathogens, necrotophic pathogens, induce the JA signaling pathway that concomitantly activates the ET signaling pathway [141]. Early JA activation inhibits SA accumulation through modulation of multiple TFs such as the basic helix-loop-helix (bHLH) transcription factor MYC2 that binds to the secondary wall thickening-promoting factor NAC, which triggers the expression of benzoic acid/SA carboxyl methyltranferase1 (BSMT1), a gene involved in SA methylation [142]. JA and ET also play an important role in the regulation of *Eucalyptus* defence (Fig. 2B) to necrotrophic pathogens [143–145].

For example, the JA signaling pathway was strongly activated in a genotype with resistance to the necrotrophic pathogen, *C. pseudoreteaudii*. This was shown by high levels of expression of a lipoxygenase gene (LOX) and an allene oxide synthase gene *(AOS)*, *(Eucgr.J00821.1)* in response to infection. ET biosynthesis was also up-regulated with a high expression of a 1-aminocyclopropane-1-carboxylic acid gene *(ACS1)* [114]. This shows that *Eucalyptus* responds to necrotrophic pathogens using similar signalling cascades as described in model species and thus resistance breeding should clearly focus on enhancing these signal transduction pathways.

Cross-Kingdom RNA Interference (CKRI)

Small RNAs (sRNAS) also play a role in plant response to fungal infection. The microRNAs are 20–24 nucleotide long and act as shields against infection. At the same time pathogens use sRNAs to suppress plant responses [105]. This phenomenon, where RNAs are used to silence the expression of target genes in a different organism, is known as RNA cross-kingdom/organism interference (CKRI) [105, 146, 147]. These RNAs have been considered as targets to develop new control strategies, referred to as spray-induced gene silencing (SIGS) where dsRNAs and sRNAs are sprayed onto plant surfaces to target pathogen effectors [105]. Recently, this strategy was successfully shown to silence different fungal genes in *Eucalyptus*, such as *β-tubulin* and *translation elongation factor 2*, reducing the growth and production of *A. psidii* infection structures [148], presenting a possible new avenue to control this important leaf and shoot pathogen.

Other Important Receptors in Plant-Pathogen Interactions with Unknown Function in *Eucalyptus*

Another important family of receptors involved in foliar pathogen recognition is CERK1 (Chitin Elicitor Receptor Kinase 1), a RLK receptor for a fungal cell wall polysaccharide known as chitin [149–153]. CERK1 receptors have been reported in *A. thaliana* and are involved in recognition of powdery mildew caused by *Blumeria graminis* [151]. In rice, CERK1 required a receptor-like protein CEBiP (Chitin Elicitor Binding Protein*)*, to recognize the leaf pathogen *Magnaporte oryzae* [154]. The homologues of these receptors in wheat are necessary for *Zymoseptoria tritici* recognition [155, 156]. Although this receptor has not been reported in the interaction of *Eucalyptus* with shoot-leaf pathogens, five *CERK* genes are currently annotated in the *E. grandis* genome (https://phytozome-next.jgi.doe.gov/) with an unknown, but putative role in the recognition of fungal pathogens.

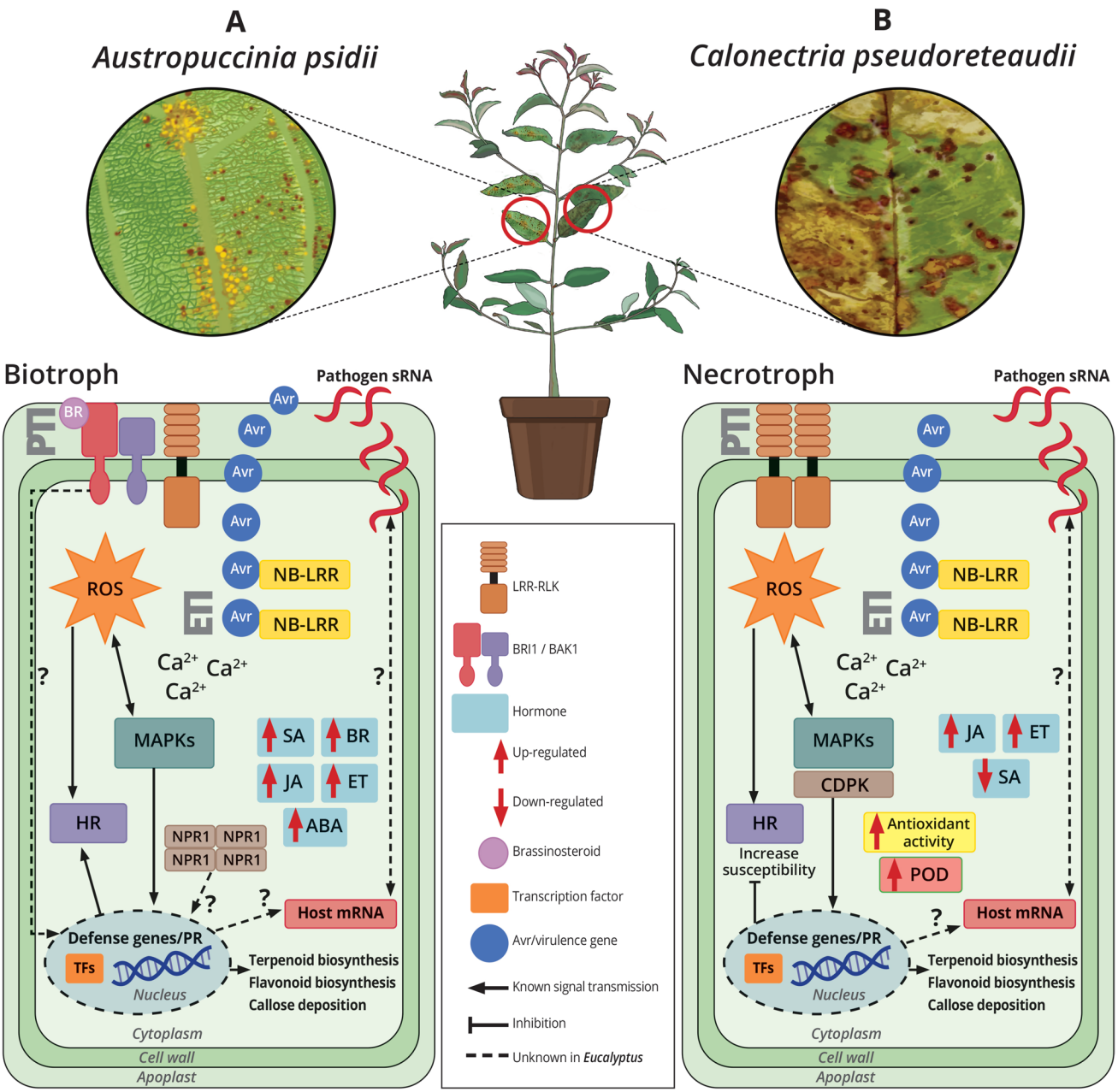
Plants can also perceive endogenous molecules that are produced during the infection. One example is the receptor wall-associated kinase 1 (WAK1) that binds cell wall-derived oligogalacturonides released during pathogen infection and serve as DAMPs [157]. In the *E. grandis* genome, 98 genes have been annotated as WAK's (https://phytozome-next.jgi.doe.gov/). Cell wall degrading enzymes (CWDEs) produced by pathogens during infection can facilitate pathogen invasion, as well as providing pathogens with nutrition [158]. In the interaction between *E. urophylla* and the powdery mildew pathogen *Podosphaera pannosa*, glycoside hydrolase (GHs) family protein genes are expressed. These proteins are known to be involved in host cell-wall degradation [159], the products of which may be detected by *Eucalyptus WAK* genes.

The Importance of Quantitative Disease Resistance (QDR)

Knowledge of the major genes involved in plant defence and the development of DNA-based molecular markers has made it possible to investigate the relationship between *Eucalyptus* genotypes and phenotypes to identify quantitative trait loci (QTL) [160, 161]. The development of genetic markers predictive of phenotypes, known as marker-assisted selection (MAS), has facilitated the improvement of different traits in plants [162–165], especially those with complex inheritance that are difficult or expensive to phenotype, such as quantitative disease resistance (QDR) [166–168]. In *Eucalyptus*, quantitative disease resistance (QDR) represents the predominant form of resistance in the interaction with *A. psidii* [169, 170]. The identified loci encoding different resistance genes against *A. psidii* have been reported as *Ppr1*, *Ppr2*, *Ppr3*, *Ppr4*, *and Ppr5* [169, 170]. As mentioned above, the resistance trait *Ppr1* was lost in a homozygous genotype in 2013 due to the outbreak of a more virulent race of the pathogen [50]. Thus, the stability of these QTL markers should be monitored, as new pathogen strains evolve continuously and could overcome the resistance trait of the host.

Recent studies of *Eucalyptus* host responses to *A. psidii* have shown that two BR-mediated signaling genes were identified within the *Ppr3* locus and four in the *Ppr5* locus [126]. Interestingly, the genes present at these loci that were highly expressed 2 dpi were associated with genes involved in both PTI and ETI [126]. PTI and ETI employ distinct immune receptors, but they appear to use a similar signaling network [171], and have recently been shown to influence each other by requiring PRR/co-receptor signaling for ETI [32] or through ETI enhancement by activation of surface receptors [172].

Delplace et al. [173] considered the main limitations of the zig-zag model, where not all pathogenic determinants fit the classical definition of PTI and ETI. This became more evident in quantitative forms of immunity such as QDR, where plant immunity should rather be seen as a distributed



and highly connected molecular network including diverse perception and signaling functions, which combines PTI and ETI genes. Recently this observation has been reported in rice infected by the foliar pathogen *M. oryzae* [174]. This could also explain the complexity of foliar resistance mechanisms in *Eucalyptus*.

Phytochemicals Synthetized by *Eucalyptus*

Phytochemicals, such as secondary metabolites, are involved in plant defence responses to pathogens [175, 176]. In *Eucalyptus*, genes involved in flavonoid and terpenoid biosynthesis play a role in the resistance to biotrophic [126] and necrotrophic pathogens [114]. In the interaction of *Eucalyptus* with *C. pseudoreteaudii*, high expression of a gene encoding phenylalanine ammonia lyase (PAL), PAL2 (*Eucgr.J01079.1*), a key enzyme in the regulation of carbon flow into the phenylpropanoid pathway, together with significantly higher expression of genes involved in lignin biosynthesis, was reported in a clone resistant to this pathogen [114] (Fig. 2B).

In the interaction with biotrophic pathogens, the expression of genes involved in flavonoid and terpenoid biosynthesis, as well as genes involved in callose deposition were

◂ **Fig. 2** A model of the main *Eucalyptus* molecular responses against biotrophic and necrotrophic foliar pathogens based on studies on the interaction with *Austropuccinia psidii* and *Calonectria pseudoreteaudii*. **(A) Main molecular responses against biotrophic pathogens (example*A. psidii*).** Pathogen recognition includes the pattern recognition receptors (PPR) LRR-RLK and BRI1-associated receptor kinase (BAK1) a co-receptor of BRI1 (BR-insensitive 1) which induce Brassinosteroid (BR) signal transduction with direct activation of pathogenesis-related (PR) genes. These pattern recognition receptors instantly trigger a number of downstream responses, such as; activation of mitogen-activated protein kinases (MAPKs) and Ca^{2+} accumulation, involved in multiple signalling defence responses, including the biosynthesis/signalling of plant stress/defence hormones, such as BR, salicylic acid (SA), abscisic acid (ABA) and jasmonic acid and ethylene pathway (JA/ET) which can directly activate transcription factors (TFs) to induce the expression of PR genes, terpenoid and flavonoid biosynthesis, callose deposition, and activates the hypersensitive response (HR) causing cell death to restrict the pathogen colonization. The possible role of mRNA cross-kingdom interference is still unknown. **(B) Main molecular responses against necrotrophic pathogens (Example*C. pseudoreteaudii*).** The sensing of necrotrophic pathogens by PRR genes is triggered by LRR-RLK receptor which may activate PR genes. Downstream calmodulin-domain protein kinase 9 (CDPK) activates ROS accumulation, as well as the elevation of antioxidant activity to induce resistance to cell death which increases susceptibility to necrotrophic pathogens. Up-regulated peroxidase (POD) play a role in lignin, suberin biosynthesis and in the regulation of ROS. This activates downstream changes in phytohormone biosynthesis, such as the activation of JA/ET to activate PR/defence genes in the nucleus. The SA pathway has been reported to be down-regulated, preventing the induction of HR which can increase the host susceptibility to necrotrophic pathogens and their colonization. The upstream signalling might also activate terpenoid, flavonoid and lignin biosynthesis to restrict the colonization by necrotrophic pathogens as reported for *(C) pseudoreteaudii.* The possible role of mRNA cross kingdom RNA interference has not been yet been demonstrated for necrotrophic pathogens in *Eucalyptus*

reported in the *Eucalyptus* - *A. psidii* interaction [126]. Interestingly, a recent study on clones with different levels of susceptibility to *A. psidii* showed that the resistant clone had higher essential oil yield compared with those that were susceptible. Limonene was the main compound induced in the resistant host, while this compound was synthesized in low concentrations in the leaves of the susceptible clones [177] *(*Fig. 2A*)*. Monoterpenes and sesquiterpenes have also been implicated as important compounds involved in resistance of *Eucalyptus* to *T. nubilosa*, and have been recently reported as disease biomarkers [178]. These could potentially be used as biomarkers for resistance to this and related foliar pathogens of *Eucalyptus.*

Conclusions and Future Perspectives

We are still in our infancy in our attempt to understand the specific defense responses of *Eucalyptus* to foliar pathogens compared with well-studied model plant systems, as well as the significance of expansion/contraction of genes families. This review highlights the fact that there are different mechanisms that play a role in the defence of *Eucalyptus* against fungal pathogens. It is also apparent that the responses involve similar pathways that can be activated differently depending on pathogen lifestyles. For example, *Eucalyptus* transcriptome analyses have revealed a strong expression of LRR-RLK, BR signaling and BR receptors, and genes encoding PR proteins appear to be activated early during the biotrophic rust pathogen *A. psidii* in a resistant genotype (Fig. 2A). On the other hand, the necrotrophic pathogen *C. pseudoreteaudii* elicits defences by activating specific LRR-RLK receptors (*Eucgr.F01306.1*), triggering the up-regulation of JA and ET signaling pathways (Fig. 2B). Thus, studies considering the basic biology of *Eucalyptus* pathogens should include a detailed analysis of the mode and temporal dynamics of the infection process and subsequent colonization events to better reveal the expected physiological and molecular defence mechanisms.

The knowledge of specific defence mechanisms against pathogens may be deployed in different ways, including breeding approaches and new breeding technologies (NBT). In traditional breeding, the focus is on the selection of healthy trees based on observations. A QTL approach in structured populations allows the discovery of the genetic architecture governing resistance to a pathogen. Once QTLs are validated in subsequent populations, further characterization of the underlying candidate genes in these regions can be performed. The Eucalyptus 72 K SNP chip offers the tools for genotyping. However, more recent work involves whole genome sequencing for genotyping. The latter approach allows the discovery of genes that could be causal. Additionally, genome wide association studies (GWAS), can be used, to identify the associations between markers and disease resistance traits and their stability across large unrelated populations [179]. This information would then provide a foundation for studies on this and others important *Eucalyptus* leaf pathogens with long biotrophic phases, such as the aggressive leaf pathogen *T. destructans* [130].

Breeding and genomic selection for disease resistance is a promising approach to ensure future plantation productivity. There are, however, surprisingly few programs that have successfully applied genetic resistance in forestry [180], mainly due to the long breeding cycles of forest trees with long periods to reach sexual maturity affecting the crosses between selected parents, together with large experimental size related to the extended geographic territories that the trees covered with different levels of adaptation, thus the testing must be extensive and be conducted over extended geographic areas known as target planting zones [180, 181]. Despite these limitations, there are successful programs, including breeding of pines with resistance to fusiform rust and white pine blister rust in North America [182]. In these cases, both quantitative and qualitative resistance have been included the breeding

populations to accommodate the effects of pathogen evolution and a rapidly changing environment [167, 183, 184]. It will therefore remain important to maintain high levels of genetic diversity in the breeding population to allow sufficient phenotypic plasticity to accommodate changing environmental conditions and disease outbreaks.

New breeding technologies like TALEN (transcription activator-like effector nucleases) and (clustered regularly interspaced palindromic repeats) (CRISPR)-Cas9 types of editing are anticipated to assist in the development of *Eucalyptus* populations with genetic resistance to foliar pathogens. Such approaches could be considered for the future but the technology must be refined to ensure that adaptive traits needed for the species to thrive across vastly different ranges and environments are conserved [185]. A challenge that is currently experienced in terms of the production of edited trees is that there are still negative societal perceptions about biotechnology and the Forestry Stewardship Council (FSC) will currently not tolerate genetically modified trees on FSC certified forests. Edited plants are more frequently being considered non-GM and this could translate to trees too, making editing a viable option to generate disease resistant trees.

The field is changing and the technologies are maturing. This will undoubtedly contribute to uncovering how *Eucalyptus* trees defend themselves against foliar pathogens and will, in consequence, strongly underpin the future sustainability of plantation forestry. Our priority is to study the effect of these host defence genes on other metabolic processes, the heritability of resistance traits within populations, their interaction with other adaptative traits and the impact of climate change on their activity. There are still several questions that remain unanswered regarding *Eucalyptus* defence mechanisms. It is intriguing that there is an expansion of several defence gene families, including the terpene synthase genes in *Eucalyptus grandis*. The relevance of this genetic diversity in the host remains to be explored. The sequencing of 10,000 *Eucalyptus* genomes, will provide a resource to mine to identify associations with genetic variation and host resistance. This, coupled with the knowledge of adaptation to specific environments, will provide clues to sustain *Eucalyptus* plantations well into the future.

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Data Availability No datasets were generated or analysed during the current study.

Declarations

Competing Interests The authors declare no competing interests.

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