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Antioxidant Systems of Plant Pathogenic Fungi: Functions in Oxidative Stress Response and Their Regulatory Mechanisms

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During the infection process, plant pathogenic fungi encounter plant-derived oxidative stress, and an appropriate response to this stress is crucial to their survival and establishment of the disease. Plant pathogenic fungi have evolved several mechanisms to eliminate oxidants from the external environment and maintain cellular redox homeostasis. When oxidative stress is perceived, various signaling transduction pathways are triggered and activate the downstream genes responsible for the oxidative stress response. Despite extensive research on antioxidant systems and their regulatory mechanisms in plant pathogenic fungi, the specific functions of individual antioxidants and their impacts on pathogenicity have not recently been systematically summarized. Therefore, our objective is to consolidate previous research on the antioxidant systems of plant pathogenic fungi. In this review, we explore the plant immune responses during fungal infection, with a focus on the generation and function of reactive oxygen species. Furthermore, we delve into the three antioxidant systems, summarizing their functions and regulatory mechanisms involved in oxidative stress response. This comprehensive review provides an integrated overview of the antioxidant mechanisms within plant pathogenic fungi, revealing how the oxidative stress response contributes to their pathogenicity.

Keywords **:** antioxidant system, oxidative stress response, pathogenicity, plant pathogenic fungi

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Reactive oxygen species (ROS) such as superoxide anion ($O_2^{\bullet-}$), hydrogen peroxide (H_2O_2), and hydroxyl radical ($^{\bullet}OH$), are highly reactive oxygen molecules that are generated as byproducts during various cellular metabolisms (Reczek and Chandel, 2015; Yang et al., 2013). Most intracellular ROS is produced during the electron transport chain of mitochondrial respiration (Angelova and Abramov, 2018). Additionally, ROS are generated in peroxisome and cytosol through several metabolic reactions, including the β-oxidation of fatty acids and metal ion catalysis (Avery, 2001; del Río and López-Huertas, 2016).

At low concentrations, ROS serve primarily as signaling molecules and play essential roles in the regulation of cellular pathways and physiological functions (D'Autréaux and Toledano, 2007; Mittler et al., 2011; Reczek and Chandel, 2015). Modified intracellular redox states facilitate cell signaling by oxidizing specific proteins, such as kinases, phosphatases, and transcription factors (Clempus and Griendling, 2006; McCubrey et al., 2006; Schieber and Chandel, 2014; Son et al., 2011). Oxidative modification can modulate protein activity and function, thereby regulating cellular responses and gene expression.

However, highly elevated ROS levels result in oxidative stress, causing impairment and dysfunction of the cellular components. Oxidative stress manifests as damage to DNA, proteins, and lipids, ultimately affecting cell viability. ROS accumulation induces DNA damage, contributing to double-strand break formation and the oxidation of

nucleotide bases (Srinivas et al., 2019). Furthermore, inappropriate protein oxidation due to ROS can lead to protein dysfunction, resulting in compromised structural integrity and function (Stadtman and Levine, 2000). ROS-induced lipid peroxidation affects membrane fluidity and integrity, potentially disrupting cellular compartments and organelles (Avery, 2011). Therefore, preserving the balance of ROS is essential for organisms, and the physiological and biochemical responses that work to counteract ROS and manage oxidative stress are collectively known as the oxidative stress response.

During plant infection, plant pathogens are exposed to oxidative stress conditions induced by the plant's immune response. To adapt to and counteract oxidative stress, pathogens have developed efficient oxidative stress responses, which have been the subject of investigation in studies focused on plant pathogenic fungi. This review specifically examines the oxidative stress environments encountered by plant pathogenic fungi during fungal infections. Our discussion centers on examining the importance of the oxidative stress response in influencing pathogenicity through an extensive exploration of diverse fungal antioxidant mechanisms. The objective of this review is to provide a comprehensive perspective on the role of oxidative stress responses in shaping plant-pathogen interactions.

Oxidative Stress Conditions in the Host Environment

Plants deploy a broad spectrum of defense mechanisms to counteract pathogen attacks, employing two immune systems: pattern-triggered immunity (PTI) and effector-triggered immunity (ETI). PTI acts as the first line of defense, activated upon pathogen-associated molecular patterns (PAMPs) recognition (Schwessinger and Zipfel, 2008; Zipfel and Felix, 2005). PAMPs encompass various non-self microbial molecules widely conserved across pathogen genera, such as bacterial flagellin, peptidoglycan, chitin, and fungal cell wall polysaccharides (Kaku et al., 2006; Lee et al., 2008; Sanabria et al., 2010). PAMPs are recognized by pattern recognition receptors in the plant cell membrane, which triggers a series of downstream responses, including ion fluxes, ROS production, and activation of mitogen-activated protein kinase (MAPK) cascades, and leads to defense-related gene expression (Peng et al., 2018; Tena et al., 2011; Yuan et al., 2021a; Zipfel, 2009). Pathogens have developed several strategies to counteract PTI through the release of effector proteins. These effectors manipulate various aspects of plant immunity, targeting pathways involving protein degradation, hormone signaling, transcription, and the host plant's microbiome, resulting in effector-triggered susceptibility (Irieda et al., 2019; Schreiber et al., 2021; Snelders et al., 2020; Wang and Jiao, 2019). Conversely, plants have evolved to detect these effectors using nucleotide-binding and leucine-rich repeat receptors (NLRs). Recognition of effectors by NLRs triggers a robust immune response, known as ETI, leading to enhanced resistance and hypersensitive response (HR) (Cui et al., 2015; Ngou et al., 2022). However, pathogen effectors have also evolved to suppress ETI, and this ongoing arms race between pathogens and plants is depicted by the zig-zag model (Boller and He, 2009; Jones and Dangl, 2006).

Several key immune mechanisms are commonly shared between PTI and ETI, and ROS production, one of the overlapped mechanisms, is an important defense component in both immune systems (Kadota et al., 2019). In PTI, a rapid and strong production of ROS is induced by the perception of PAMP. The NADPH oxidases belonging to the respiratory burst oxidase homolog (RBOH) family are responsible for ROS burst with a low-amplitude transient phase (Kadota et al., 2014; Sagi and Fluhr, 2006; Torres et al., 2006). ETI-mediated ROS production is also predominantly dependent on RBOH and exhibits a stronger and more persistent ROS burst than that of PTI (Kadota et al., 2015; Tsuda and Katagiri, 2010; Yuan et al., 2021a). ROS in these immune responses function as signaling molecules, controlling the redox state and modifying target sensor proteins. ROS-mediated signaling plays an important role in the regulation of hormone response, transcription factor expression, and activation of protein kinase required for immune response (Marcec et al., 2019; Suzuki et al., 2011; Torres, 2010). Furthermore, H_2O_2 production is required for activating HR that causes cell death, restricting invasive growth of pathogens, and the oxidative burst displays antimicrobial properties capable of directly killing pathogens (Delledonne et al., 2001; Li et al., 2005). Collectively, during the two-layered innate immune response within plant cells, plant pathogenic fungi face formidable challenges to their survival.

Antioxidant Systems and Their Role in Pathogenicity

In order to withstand and adapt to oxidative stress conditions, pathogens possess various antioxidant systems for detoxifying or scavenging ROS. These antioxidant systems can generally be categorized into enzymatic and non-enzymatic groups. Non-enzymatic antioxidants include glutathione, ascorbic acid, and carotenoids, and enzymatic

antioxidants include superoxide dismutase (SOD), catalase, and glutathione peroxidase (GPx) (Santos-Sánchez et al., 2019). The components of these two groups function together to eliminate ROS and maintain intracellular ROS levels (Garcia-Caparros et al., 2021). In this section, we focus on the mechanisms that directly scavenge ROS: SOD, catalase, and glutathione, and discuss the contribution of each mechanism to the oxidative stress response and pathogenicity reported in plant pathogenic fungi.

Superoxide dismutase. SOD is an enzyme that plays a role in catalyzing the conversion of superoxide radical (O_2^-) into dioxygen (O_2) and hydrogen peroxide (H_2O_2). SODs, as metalloenzymes, use metal ions for their dismutation of superoxide (McCord and Fridovich, 1969). Depending on the metal cofactor, they are usually classified into three groups: copper/zinc-containing SOD (Cu/Zn-SOD), manganese-containing SOD (Mn-SOD), and iron-containing SOD (Fe-SOD) (Fridovich, 1986). As Fe-SOD has been reported to be rarely found in the Fungi kingdom, Cu/Zn-SOD and Mn-SOD have been extensively investigated in plant pathogenic fungi (Table 1) (Miller, 2012).

Cu/Zn-SODs are divided into intracellular Cu/Zn-SOD and extracellular Cu/Zn-SOD (EC-SOD) based on the presence of an N-terminal signal peptide for secretion (Zelko et al., 2002). Intracellular Cu/Zn-SOD, found in the cytoplasm, plays a pivotal role in scavenging endogenous O_2^- and is crucial for the virulence of plant pathogenic fungi. The depletion of *SOD1* resulted in increased sensitivity to menadione, an intracellular superoxide-generating agent, observed in *Fusarium graminearum* and *Sclerotinia sclerotiorum* (Veluchamy et al., 2012; Yao et al., 2016). In *F. graminearum*, the expression of *SOD1* was strongly induced during plant infection, and the *SOD1* deletion caused reduced virulence (Yao et al., 2016). Consistent with the results of *F. graminearum*, the *SOD1* deletion mutants of *Botrytis cinerea* and *S. sclerotiorum* had defects in virulence and led to altered ROS accumulation in plant cells (López-Cruz et al., 2017; Veluchamy et al., 2012). Some cytoplasmic Sod1 proteins that lack the signal peptide have been observed to be secreted through unconventional secretion pathways (Kinseth et al., 2007; Nickel, 2003; Tian et al., 2021a). Sod1 from *Verticillium dahliae* lacking the signal peptide was found to be secreted through the Golgi reassembly stacking protein and contributed to fungal virulence by scavenging endogenous and exogenous O_2^- (Tian et al., 2021a).

EC-SOD has a canonical signal peptide or the glycophosphatidylinositol-anchor, enabling their release outside the cells or anchored to the cell wall. In *Puccinia striiformis*, PsSOD2 was observed to localize on the plasma membrane, suppressing ROS accumulation in plant cells (Zheng et al., 2020). Another EC-SOD of *P. striiformis*, PsSOD1 transiently expressed in *Nicotiana benthamiana*, inhibited programmed cell death, highlighting its role as a virulence factor (Liu et al., 2016). The *FoSOD5* and *VdSOD5*, the EC-SOD of *Fusarium oxysporum* and *V. dahliae*, respectively, were upregulated in early infection stages, work-

Table 1. Superoxide peroxidases characterized in plant pathogenic fungi

	Species	Localization/ Subclassification	Gene name	References
Cu/Zn-SOD	*Botrytis cinerea*	IC	*Bcsod1*	López-Cruz et al. (2017)
	Claviceps purpurea	IC	*Cpsod1*	Moore et al. (2002)
	Fusarium graminearum	IC	*SOD1*	Yao et al. (2016)
	Fusarium oxysporum	EC (Cu-only SOD)	*FoSOD5*	Wang et al. (2021)
	Puccinia striiformis	EC (Zn-only SOD)	*PsSOD1*	Liu et al. (2016)
	Puccinia striiformis	EC (Cu-only SOD)	*PsSOD2*	Zheng et al. (2020)
	Sclerotinia sclerotiorum	IC	*Sssod1*	Veluchamy et al. (2012)
	Verticillium dahliae	IC/US	*VdSOD1*	Tian et al. (2021)
	Verticillium dahliae	EC (Cu-only SOD)	*VdSOD5*	Li et al. (2021)
Mn-SOD	*Colletotrichum graminicola*	IC	*SOD2*	Fang et al. (2002)
	Verticillium dahliae	IC/US	*VdSOD3*	Tian et al. (2021)

IC, intracellular region; EC, extracellular region/secreted; US, unconventionally secreted; SOD, superoxide dismutase.

ing to detoxify ROS generated by the host plant (Li et al., 2021; Wang et al., 2021).

Mn-SODs are usually localized within the mitochondria or the cytosol. However, similarly to observations in Cu/Zn-SODs, some cytosolic Mn-SOD can be released into the extracellular space without the signal peptide. The study of *V. dahliae* found that VdSOD3, identified as Mn-SOD, could be translocated extracellularly during infection and required for full virulence, but the specific function of VdSOD3 needs to be investigated (Tian et al., 2021b).

Most identified fungal SODs play a critical role in fungal virulence by effectively detoxifying host-generated O_2^-. However, not all SODs seem to contribute to fungal virulence during infection. For example, the deletion of *SOD2* in *Colletotrichum graminicola*, which encodes Mn-SOD, did not result in any significant changes in sensitivity to menadione and virulence compared to the wild type (Fang et al., 2002). Furthermore, the study on *Claviceps purpurea* revealed that *cpsod1* does not play a crucial role in pathogenicity (Moore et al., 2002). As these two SODs were not observed to be induced or expressed under oxidative stress conditions or during infection, it is suggested that they might be involved in other cellular functions.

Catalase. Catalase (EC 1.11.1.6) catalyzes the dismutation of H_2O_2 into a water and oxygen molecule. There are three types of catalases: typical monofunctional heme-containing catalases, bifunctional catalase-peroxidases, and Mn-containing catalases (Nicholls, 2012). Since Mn-containing catalase has been reported to exist only in prokaryotes and archaea (Whittaker, 2012), the other two classes will be discussed in this review.

Monofunctional heme-containing catalases are most widespread in all aerobic organisms, and are divided into three clades based on molecular phylogeny: clade 2 groups the large-subunit catalases, and clades 1 and 3 include small-subunit catalases (Zamocky et al., 2008). Clade 1 comprises catalases from bacteria, green algae, and plants, and typical fungal catalases belong to clades 2 and 3 (Chelikani et al., 2004; Hansberg et al., 2012; Zámocký et al., 2012b). As shown in Fig. 1, the monofunctional catalases identified in plant pathogenic fungi are largely divided into two groups, corresponding to clades 2 and 3. Two groups diverge in clade 2 based on the presence of signal peptides.

Studies of monofunctional catalases in plant pathogenic fungi have reported that several catalases are highly associated with oxidative stress or *in planta* responses. In *Cochliobolus heterostrophus*, the *CAT3* deletion mutants showed increased sensitivity to oxidative stress (Robbertse et al., 2003). Similarly, the treatment of H_2O_2 resulted in increased transcript levels of *Bccat2* and the disruption of *Bccat2* caused the increased sensitivity to oxidative stress in *B. cinerea* (Schouten et al., 2002). In *Cladosporium fulvum*, *Cat2* expression was induced upon exogenous oxidative stress treatment, but the deletion of *Cat2* did not cause phenotypic changes under oxidative stress conditions (Bussink and Oliver, 2001). Deletion of each monofunctional catalase-encoding gene did not result in a phenotypic difference compared to the wild-type strain in *F. graminearum* (Lee et al., 2018), while the transcript levels of all five catalases significantly increased upon H_2O_2 treatment (Lee et al., 2014), suggesting their redundant functions in the oxidative stress response. Among the identified typical catalases, PsCAT1 of *P. striiformi*, SsCAT1 and SsCAT2 of *S. sclerotiorum*, and CATB of *Magnaporthe oryzae* are required for fungal virulence (Huang et al., 2021; Skamnioti et al., 2007; Yarden et al., 2014; Yuan et al., 2021b). PsCAT1 and SsCAT2 are responsible for resistance to oxidative stress and it was confirmed that the deletion of both genes caused H_2O_2 accumulation in host plant cells, indicating that two catalases are important for scavenging host-derived ROS. Notably, the deletion mutants *Sscat1* of *S. sclerotiorum* and *CATB* of *M. oryzae* exhibited increased tolerance to H_2O_2, indicating that their role in the virulence is via other mechanisms such as regulation of cell wall integrity rather than detoxification of ROS. Rather, the *CATB* deletion mutant exhibited an increased accumulation of intracellular H_2O_2 in appressoria, which affects the viability and penetration ability in *M. oryzae* (Skamnioti et al., 2007).

The bifunctional catalase-peroxidases, called KatGs, are distributed in prokaryotes and lower eukaryotes, with the exception of mammalian, and possess both peroxidase and catalase activity within a single active site (Baker et al., 2006; Fraaije et al., 1996). There are two distinct groups of fungal KatG: intracellular catalase-peroxidases (KatG1) and extracellular catalase-peroxidases (KatG2) (Zámocký et al., 2009b), and these KatGs serve as primary catalysts and play a role in the oxidative stress response. In *F. graminearum*, Fca6 and Fca7 were identified as KatG1 and KatG2, respectively, and Δ*fca6* and Δ*fca7* deletion mutants showed hypersensitivity to oxidative stress, exhibiting a drastic reduction in total peroxidase enzyme activity (Lee et al., 2018). Studies on *M. oryzae* also reported that *CPXA*, corresponding to KatG1, is continuously expressed in normal and oxidative stress conditions and *CPXB* encoding KatG2 is upregulated under oxidative stress conditions (Zamocky et al., 2009a; Zámocký et al., 2012a). Additionally, *CPXB* deletion significantly reduced the level of catalase activity in *M. oryzae* (Tanabe et al., 2011).

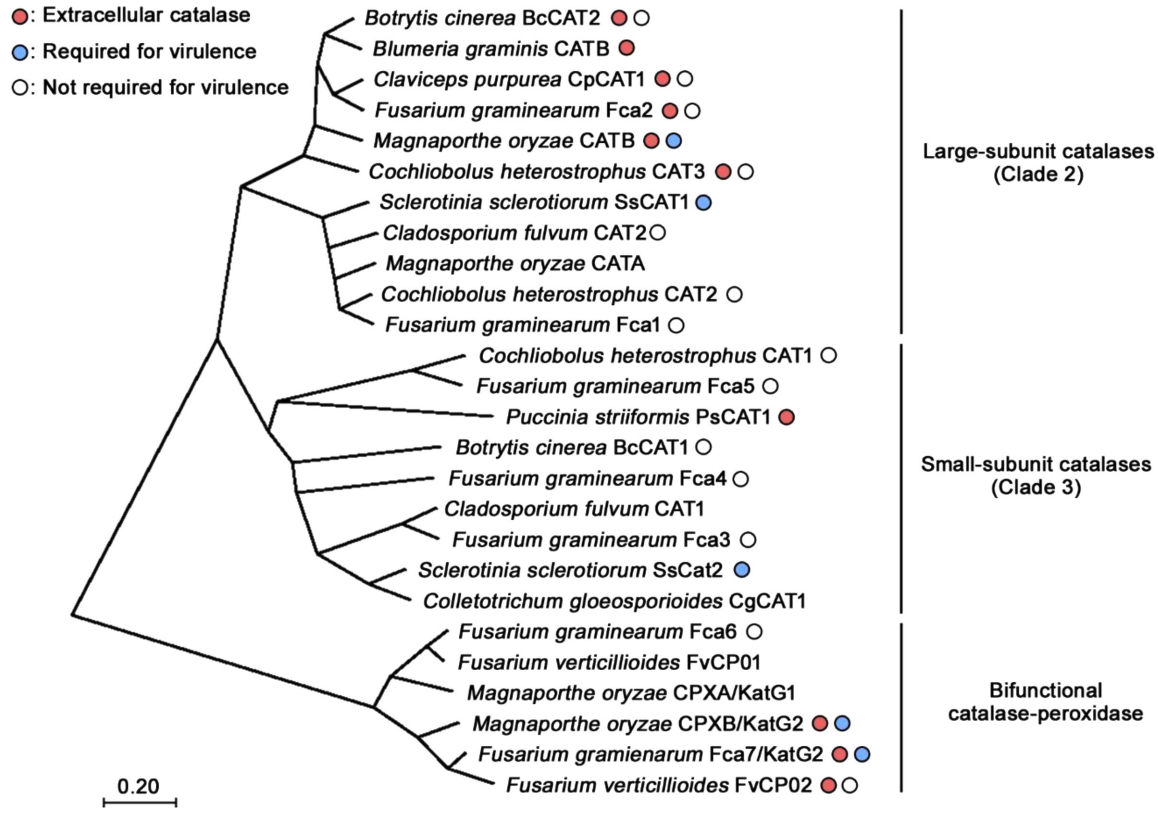

Fig. 1. A phylogenetic tree of monofunctional and bifunctional catalases characterized in plant pathogenic fungi. Sequences were aligned with the MUSCLE algorithm, and the phylogenetic tree was constructed by the neighbor-joining method with 1,000 bootstrap replicates, using the MEGA11 program.

Especially, KatG2, as an extracellular protein, plays a vital role in scavenging host-derived ROS at the pathogen-host interface. In *F. graminearum*, KatG2 is localized in the cell wall of invading hyphae, aiding in ROS scavenging generated by the host cells, ultimately contributing to virulence (Guo et al., 2019). In *M. oryzae*, the expression of *CPXB* was transiently upregulated at the early stage of infection in the leaf sheath (Tanabe et al., 2011), and the catalase activity of CPXB is responsible for the removal of H_2O_2 in rice leaf sheath epidermal cells (Tanabe et al., 2009). Furthermore, deletion of *CPXB* showed a defect in infection during the early stage, indicating that CPXB contributes to the virulence of *M. oryzae* (Tanabe et al., 2011). Although the gene encoding KatG2, *FvCP02*, showed increased expression under *in planta* oxidative stress conditions, the deletion of *FvCP02* causes only a minor effect on virulence in *Fusarium verticillioides* (Gao et al., 2018). These findings suggest that while KatG2 is responsible for scavenging external ROS, its impact on virulence varies among different fungal pathosystems (Fig. 1).

Glutathione mechanisms. Glutathione, a ubiquitous tripeptide composed of glutamic acid, cysteine, and glycine, is an important antioxidant. Glutathione plays a crucial role in neutralizing and detoxifying ROS, and several glutathione metabolism-related enzymes are known to be involved in the synthesis, utilization, and recycling of glutathione (Gullner and Kömives, 2001; Meister and Anderson, 1983). GPx is involved in the removal of H_2O_2 and hydroperoxides by converting reduced glutathione (GSH) to its oxidized form (GSSG) (Wendel, 1980). Glutathione reductase (GR) is responsible for recycling GSSG into GSH (Couto et al., 2016).

Studies on plant pathogenic fungi have demonstrated that both GPx and GR are required for resistance to oxidative stress. The deletion of GPx-encoding genes resulted in increased sensitivity to H_2O_2 and menadione in *Alternaria alternata*, *Valsa mali*, and *M. oryzae* (Feng et al., 2021; Huang et al., 2011; Yang et al., 2016). In. *M. oryzae*, *GTR1*, which encodes GR, is responsible for resistance to H_2O_2 and menadione (Fernandez and Wilson, 2014). Similarly, in *A. alternata*, the *Glr1* deletion mutant had increased sensitivity to oxidants (Ma et al., 2018).

The enzyme activities of GPx and GR are required for

fungal virulence. The expression of genes encoding GPx and GR was upregulated during plant infection in *M. oryzae* and *Blumeria graminis* (Mir et al., 2015; Zhang et al., 2004), and the deletion of genes encoding GPx and GR resulted in attenuated virulence in *A. alternata*, *V. mali*, and *M. oryzae*. However, the *FPX21* gene that encodes GPx is not required for oxidative stress resistance and virulence in *F. graminearum* (Lee et al., 2018), which suggests that the contribution of GRs to oxidative stress resistance and virulence may vary among different fungal species.

The role of glutathione mechanisms in the oxidative stress response has also been confirmed through functional analyses of enzymes within the glutathione biosynthesis pathway. Glutathione is synthesized by the consecutive actions of two enzymes, γ-glutatmylcysteine ligase (Gsh1) and glutathione synthetase (Gsh2). A recent study in *F. graminearum* reported that glutathione deficiency caused by the deletion of *GSH1* led to increased sensitivity to oxidative stress (Park et al., 2024). Furthermore, the accumulation of the metabolic intermediate γ-glutamylcysteine was able to restore the hypersensitivity of glutathione-deficient mutant to oxidative stress, indicating the role of γ-glutatmylcysteine as an intracellular antioxidant (Park et al., 2024). The deletion of *GSH1* and *GSH2* from *F. graminearum* led to a decrease in virulence, and, notably, the contribution of glutathione to virulence is considered to be separate from its antioxidant activity in *F. graminearum* (Park et al., 2024). Despite the restoration of tolerance to oxidative stress through overexpression of the key catalase Fca7, virulence was not restored, indicating that the role of glutathione in fungal virulence is independent of its antioxidant activity (Park et al., 2024). These findings are supported by a previous study which reported that GPx is not involved in virulence in *F. graminearum* (Lee et al., 2018). However, further research is still required to investigate the role of glutathione in virulence and its underlying mechanism.

The Regulatory Mechanisms of Antioxidant Systems

Signal transduction mechanisms play a crucial role in detecting external stress stimuli and orchestrating downstream responses. In eukaryotes, these mechanisms such as a MAPK pathway, the cyclic AMP (cAMP) signaling pathway, and the nutrient-sensing target of the rapamycin (Tor) signaling pathway, are well conserved, and their roles for responding to environmental stimuli have been extensively investigated (Seger and Krebs, 1995; Wullschleger et al., 2006). Here, we present the main signaling pathways or regulatory mechanisms responsible for orchestrating oxidative stress response; the high-osmolarity glycerol (HOG) MAPK pathway and the cooperation of two transcription factors activator protein-1 (AP-1) and Skn7 (Fig. 2).

HOG pathway and Atf1. The MAPK pathway is a representative signaling cascade that coordinates the cellular response for adaptation in their extracellular environment. The MAPK cascade consists of a MAPK, a MAPK kinase (MAPKK), and a MAPKK kinase (MAPKKK), and activated MAPKKK in response to an extracellular signal regulates the downstream pathway by sequential phosphorylation (Qi and Elion, 2005). In model fungi such as *Saccharomyces cerevisiae* and *Schizosaccharomyces pombe*, MAPK cascades are well investigated (Chen and Thorner, 2007), and several studies on fungi revealed that MAPK cascades are classified according to the external stress type or biological functions: mating pheromone responses, filamentous growth, cell wall integrity, and HOG. Among them, the HOG MAPK cascade has been identified as playing a role in the oxidative stress response (Bahn, 2008; Ikner and Shiozaki, 2005).

In *S. cerevisiae*, the HOG pathway is proceeded by consecutive phosphorylation with two upstream branches, Sln1 and Sho1 (O'Rourke and Herskowitz, 2004). The Sln1 branch is controlled by a two-component signaling pathway composed of a dimeric sensor kinase (Sln1), the histidine-containing phosphotransfer protein (Ypd1), and a response regulator (Ssk1) (Macia et al., 2009). Sln1 is a membrane-localized histidine kinase and acts as a negative regulator of the HOG pathway (Hohmann, 2002). Under non-osmotic stress conditions, the active form of Sln1 is maintained, and the phosphoryl group is continuously transferred through Ypd1 to Ssk1, which inhibits the function of Ssk1 and inactivates the downstream pathway (Kaserer et al., 2009). Upon hyperosmolarity, Sln1 kinase activity is reduced causing a reduction in phosphotransfer activity. Unphosphorylated Ssk1 activates MAPKKK Ssk2/Ssk22, and through sequential phosphorylation of MAPKK Pbs2 and MAPK Hog1, regulates osmolarity-related response genes (Hohmann, 2002; Posas et al., 1996). Sho1 is also a sensor of another branch for controlling HOG pathway. Upon osmotic stress, Sho1 activates MAPKKK Ste11, which is directed to phosphorylate downstream kinases Pbs2 and Hog1 (Zarrinpar et al., 2004). In fission yeast *S. pombe*, the stress-activated protein kinase (SAPK) pathway serves an equivalent function to the HOG pathway in yeast, which is responsive to global stress, including hyperosmotic stress and heat and cold stress. The SAPK pathway is composed of MAPK Sty1 (also known as Spc1 and Phh1), MAPKK

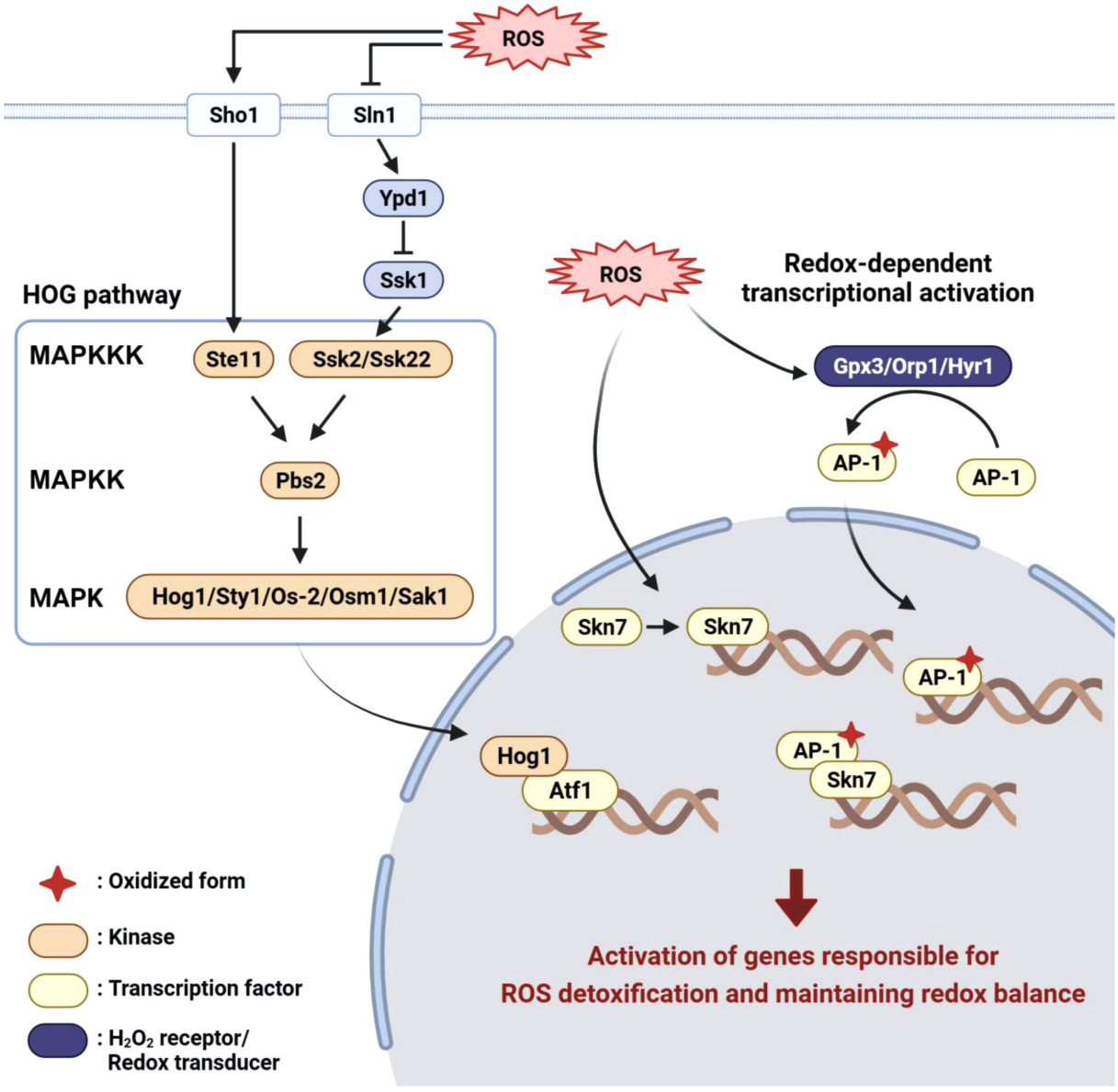

Fig. 2. Scheme of oxidative stress response pathways in plant pathogenic fungi. There are two conserved pathways that activate the genes responsible for reactive oxygen species (ROS) detoxification and maintaining redox balance: the high-osmolarity glycerol (HOG) pathway and redox-dependent activation via two transcription factors, activator protein-1 (AP-1) and Skn7. In the HOG pathway, oxidative stress-induced signals are transferred via phosphorylation of the mitogen-activated protein kinase (MAPK) cascade, and phosphorylated Hog1 (also known as Sty1, Os-2, Osm1, and Sak1 in *Schizosaccharomyces pombe*, *Fusarium graminearum*, *Magnaporthe oryzae*, and *Botrytis cinerea*, respectively) localizes to the nucleus and activates the Atf1 transcription factor. The activation of Skn7 and AP-1 transcription factors is mediated by ROS. Under oxidative stress conditions, AP-1 is oxidized by redox transducer such as Gpx3 of *Saccharomyces cerevisiae*, and is accumulated in nucleus regions. The transcription activity of Skn7 was induced by oxidative stress, and collaborating with AP-1, these transcription factors regulate the genes responsible for the oxidative stress response. This figure was created using BioRender.

Wis1, and MAPKKK Wak1 (also known as Wis4) and Win1 (Gacto et al., 2003; Smith et al., 2010). Similar to the Sln1 branch of *S. cerevisiae*, the response regulator Mcs4, a functional homolog of Ssk1, is activated upon the extracellular stress signals and activates multistep phosphorelay of Wak1-Wis1-Sty1 (Buck et al., 2001; Smith et al., 2010; Vivancos et al., 2006).

These MAPK/SAPK pathways have been reported to be

responsible for the oxidative stress response (Quinn et al., 2002; Vivancos et al., 2006). Several studies have reported that the phosphorylated level of Hog1 is increased in response to oxidative stress in *A. alternata*, *Heterobasidion annosum*, and *B. cinerea* (Lin and Chung, 2010; Raffaello et al., 2012; Segmüller et al., 2007). The absence of the HOG pathway component genes results in fungal strains that are sensitive to oxidative stress. In *F. graminearum*, the deletion of components of the HOG pathway such as *HOG1*, *PBS2*, and *SSK2*, resulted in heightened sensitivity to oxidative stress and a defect in virulence (Zheng et al., 2012). Similarly, the *HOG1* homolog deletion in *F. proliferatum*, *B. cinerea*, and *Bipolaris oryzae* increased sensitivity under oxidative stress conditions (Ádám et al., 2008; Moriwaki et al., 2006; Segmüller et al., 2007). Furthermore, the absence of the *HOG1* homolog gene led to reduced expression of antioxidant system components such as catalases and GPx in *B. oryzae* and *A. alternata* (Moriwaki et al., 2006; Yang et al., 2016).

Studies on *S. pombe* revealed that the SAPK pathway, homolog of the HOG pathway, is involved in the oxidative stress response by interacting with the ATF/CREB-type transcription factor Atf1 (Lawrence et al., 2007; Rep et al., 2001; Wilkinson et al., 1996). Stress-triggered activation of Sty1 prompts its migration to the nucleus, where it engages with and phosphorylates Atf1, thus stimulating the initiation of transcription for stress-related downstream genes (Hohmann, 2002; Lawrence et al., 2007; Salat-Canela et al., 2017). Consistent with reports in model organisms, a study in *M. oryzae* showed that MoOsm1, the ortholog of Hog1, is phosphorylated and localized to nuclei under oxidative stress conditions and *in planta* (Liu et al., 2020). Additionally, MoOsm1 regulates MoAtf1 through phosphorylation, which regulates genes involved in the oxidation-reduction process (Liu et al., 2020). The functions of Atf1 in the oxidative stress response have also been reported in several plant pathogenic fungi including *V. dahliae*, *F. oxysporum*, and *C. purpurea* (Fang et al., 2017; Nathues et al., 2004; Qi et al., 2013; Szabó et al., 2020). In detail, the study of *C. purpurea* revealed that CpTF1, a homolog of Atf1, is required for the expression of catalase genes (Nathues et al., 2004). Similarly, the deletion of *Foatf1* caused an increased sensitivity to oxidative stress and resulted in a decrease in catalase activity in *F. oxysporum* (Qi et al., 2013). In *V. dahliae*, the deletion mutant of the *Atf1* homolog caused a decrease in extracellular peroxidase activity and produced more intracellular ROS compared to the wild type (Fang et al., 2017). These findings suggest that Atf1 is clearly responsible for the expression of oxidative stress response-related genes, and its function is well conserved in phytopathogenic fungi.

However, a study on *F. graminearum* showed that the regulation of antioxidant mechanisms by Atf1 operates *in planta*, not under *in vitro* oxidative stress conditions. Furthermore, the Atf1 functions as a repressor of catalase expression under oxidative stress response, and the deletion of *ATF1* showed increased resistance to oxidative stress (Nguyen et al., 2012; Van Nguyen et al., 2013), which is consistent with the report in *S. cerevisiae* showing tolerance of the *sko1* (ortholog of Atf1) mutant to oxidative stress (Rep et al., 2001). These findings suggest that although the HOG pathway is responsible for the oxidative stress response, the regulation of Atf1 on downstream genes has evolved to function differently depending on fungi species.

Cooperation of two transcription factors AP-1 like protein and Skn7. In *S. cerevisiae*, AP-1 like proteins were first identified on the basis of the similarity of their bZIP DNA binding domains with that of the AP-1 of mammalian cells (Angel et al., 1988; Toone et al., 2001). Yap1, a yeast AP-like protein has been reported to play an essential role for oxidative stress, and the machinery of Yap1 in the oxidative stress response has been well characterized. The Yap1 regulation is mediated in a redox-dependent manner. Under normal conditions, Yap1, which possesses a nuclear export sequence (NES), shuttles freely between the nucleus and the cytoplasm (Kuge et al., 1997; Yan et al., 1998). When exposed to oxidants, the GPx-like enzyme Gpx3 (also known as Orp1) serves as a sensor of H_2O_2 and catalyzes the formation of disulfide bonds between the N-terminal and C-terminal cysteine-rich domains of Yap1 (Delaunay et al., 2002). During these processes, NES is masked in the oxidized sulfide-bridge structure of Yap1, which leads to nuclear localization of Yap1 and triggers the expression of oxidative stress-related genes such as *GSH1*, *GPX*, and thioredoxin (Delaunay et al., 2000; Fassler and West, 2011; Toone and Jones, 1999; Wood et al., 2004).

The function of Yap1 orthologs on oxidative stress response have been extensively investigated in plant pathogenic fungi. In *M. oryzae*, *B. cinerea*, *U. maydis*, *A. alternata*, and *F. graminearum*, deletion of the Yap1 ortholog genes led to hypersensitivity to oxidative stress and decreased the expression level of genes related to ROS detoxification, including extracellular peroxidase (Guo et al., 2011; Lee et al., 2018; Molina and Kahmann, 2007; Montibus et al., 2013; Park et al., 2023; Temme and Tudzynski, 2009). Additionally, the study of *A. alternata* demonstrated the involvement of AaAp1 in oxidative stress resistance by affecting the activity of catalase, peroxidase, and SOD (Lin et al., 2009). Furthermore, inhibition of NADPH oxidase

in plants restored the pathogenicity of the *AaAP1* deletion mutant, indicating that the function of AaAp1 in the oxidative stress response is essential for fungal virulence in *A. alternata* (Lin et al., 2009). But still, the machinery required for AP-1 activation was only explored in the study of *U. maydis*, uncovering the importance of two disulfide bridges for AP-1 functionality (Molina and Kahmann, 2007). Research in *M. oryzae* revealed the involvement of Hyr1, the ortholog of H_2O_2 sensor Gpx3 of *S. cerevisiae*, in detoxifying ROS (Huang et al., 2011), but in *B. cinerea*, the ortholog of Gpx3 is not required for nuclear translocation of AP-1 (Viefhues et al., 2015).

The function of AP-1, which regulates the expression of genes related to oxidative stress response, is highly conserved in plant pathogenic fungi. However, its role in fungal virulence varies depending on the pathogen's lifestyle. Yap1 orthologs have been known to be crucial for virulence in several biotrophic fungi. In *U. maydis*, Yap1 is activated during the early stage of biotrophic growth and regulates the downstream genes required to detoxify ROS and maintain redox homeostasis, and deletion of *YAP1* caused the reduced virulence on the host plant (Molina and Kahmann, 2007). An ortholog of Yap1 in *Monilinia fructicola*, MfAP1, is activated to respond to oxidative burst of host plants, and the appropriate expression of *MfAP1* during infection is essential for fungal virulence (Yu et al., 2017). However, in necrotrophic fungi, including *B. cinerea* and *C. heterostrophus*, AP-1 is not required for its virulence (Lev et al., 2005; Temme and Tudzynski, 2009). In *C. heterostrophus*, it was observed that *ChAP1* is expressed upon oxidative stress or plant signals at the early stage of plant infection, activating the antioxidant genes, but the deletion of *ChAP1* did not affect virulence (Lev et al., 2005). Interestingly, the host-derived oxidative stress condition did not induce the expression of the AP-1-regulated oxidative stress response in *B. cinerea* (Temme and Tudzynski, 2009). However, some of the necrotrophic fungi, such as *A. alternata* described above, showed the requirement of AP-1 for their virulence (Lin et al., 2009, 2011). In hemibiotrophic fungi, the contribution of AP-1 to virulence differs between species. For example, Yap1 orthologs are important for virulence in *M. oryzae*, *C. gloeosporioides*, and *Lasiodiplodia theobromae* (Guo et al., 2011; Li et al., 2017; Sun et al., 2016; Zhang et al., 2021). In contrast, Fgap1, the ortholog of Yap1 in *F. graminearum*, is dispensable for virulence (Montibus et al., 2013). Together, AP-1 extensively regulates genes associated with the oxidative stress response under oxidative stress conditions, and this AP-1-mediated oxidative stress response significantly contributes to the virulence of most biotrophic fungi and some necrotrophic/hemibiotrophic fungi, highlighting the importance of the host-derived ROS-eliminating process during plant infection.

Skn7, along with Ssk1, functions as a response regulator affected by Sln1-Ypd1 signal transduction. Unlike Ssk1 which is activated upon hyperosmotic stress, Skn7 is activated under hypo-osmotic conditions, which compensates for the shutdown of the HOG pathway (Levin, 2011). Additionally, Skn7 activated by the Sln1-Ypd1 pathway is responsible for the response to cell wall stress, participating in the regulation of downstream genes related to the cell wall integrity (Fassler and West, 2011; Levin, 2005; Li et al., 2002). However, in response to oxidative stress, Skn7 is activated independently of the Sln1-Ypd1-mediated activation and governs the expression of genes involved in the oxidative stress response (Charizanis et al., 1999; Morgan et al., 1997). In plant pathogenic fungi, the role of Skn7 in the oxidative stress response is well conserved. In *C. heterostrophus*, *B. cinerea*, *F. graminearum*, and *V. dahliae*, the *SKN7* deletion mutant showed increased sensitivity to oxidative stress (Lee et al., 2018; Shalaby et al., 2014; Tang et al., 2017; Yang et al., 2015), and deletion of *SKN7* led to a reduction of catalase and peroxidase activities in *A. alternata* (Chen et al., 2012). The functions of Skn7 orthologs in virulence have also been investigated in several plant pathogenic fungi. In *A. alternata*, the Δ*skn7* mutant showed attenuated virulence (Chen et al., 2012), while *SKN7* orthologs of *B. cinerea*, *F. graminearum*, and *C. heterostrophus* were not required for virulence (Jiang et al., 2015; Shalaby et al., 2014; Yang et al., 2015), aligned with the observed function of *AP-1* in each fungal species.

Ap-1 and Skn7, both activated in a redox-dependent manner, cooperate in regulating oxidative stress response-related genes. A significant portion of downstream genes regulated by each transcription factor overlaps, and studies in *S. cerevisiae* showed that the receiver domain of Skn7 is required for activating oxidative stress response genes by interacting with Ap-1 (He et al., 2009; Mulford and Fassler, 2011). And also, in plant pathogenic fungi, their collaboration in the transcriptional regulation of oxidative stress response genes has been observed. In *B. cinerea*, the genes encoding thioredoxin reductase (*trr1*), glutaredoxin (*grx1*), glutathione reductase (*glr1*), and glutathione peroxidase (*gpx3*) were co-regulated by both Skn7 and Ap-1 (Viefhues et al., 2015). Notably, Skn7 binds directly to the promoter regions of *glr1*, and not to *grx3* in *B. cinerea* (Viefhues et al., 2015), which suggests that Ap-1 and Skn7 function as a complex in activating specific target genes or that they share other downstream regulators. In *C. heterostrophus*, the expression of GR, thioredoxin reductase, and

glutathione biosynthetic genes were regulated by ChAP1, but not affected by the deletion of *SKN7*. In contrast, the catalase 2-encoding gene is under the regulation of Skn7 alone (Shalaby et al., 2014), indicating that they also have unique roles in the management of oxidative stress. But, the Δ*chap1* and Δ*chskn7* deletion mutants did not show a defect in virulence, but the double deletion of orthologs of *AP-1* and *SKN7* led to the decreased virulence in *C. heterostrophus*, showing the synergism of Ap-1 and Skn7 on oxidative stress resistance and virulence, and they operate as pivotal regulators of the oxidative stress response.

Conclusions and Future Perspectives

In this review, we examine the oxidative stress environments encountered by plant pathogenic fungi during plant infection and summarize the function of antioxidant mechanisms in ROS scavenging and virulence. The coordinated signaling pathways and regulatory mechanisms are involved in adequate activation of antioxidant systems, including SOD, catalase, and glutathione mechanisms. Antioxidant systems are involved in the maintenance of intracellular ROS levels and function in redox signaling, which contributes to virulence by affecting growth, penetration ability, and the formation of invasive structures. Additionally, some antioxidants are located in the cell wall or secreted into the plant cell and are essential for virulence by directly scavenging host-derived ROS.

This review has two implications for research on oxidative stress responses in plant pathogenic fungi. First, the contribution of the oxidative stress response to virulence depends on the lifestyle of the fungus. The discussion based on the characterization of the ortholog of Yap1 in various fungi showed that resistance to oxidative stress contributes differently to virulence depending on fungal lifestyles. For biotrophic fungi, the ability to cope with oxidative stress is critical for their virulence. With some exceptions, this ability does not appear to contribute significantly to virulence in necrotrophic fungi, suggesting that the impact of host-derived oxidative stress on pathogens with different lifestyles is clear.

Second is the presence of antioxidant mechanisms that specifically function during the process of plant infection. As described above, in *F. graminearum*, the regulation of Atf1 on the oxidative stress response genes differs between *in planta* and *in vitro* oxidative stress conditions. Additionally, in *F. verticillioides*, KatG2 exhibits a significant increase in expression *in planta*, remaining uninduced under *in vitro* exogenous oxidative stress conditions. These findings suggest the existence of *in planta*-specific antioxidant mechanisms and underscore the limitations of *in vitro* oxidative conditions in mimicking host-derived ROS stress conditions. Therefore, it is necessary to investigate beyond *in vitro* investigations and delve into actual host-pathogen interaction scenarios to elucidate the role of the oxidative stress response in fungal virulence.

Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

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