Opposing effects of MYZUS PERSICAE-INDUCED LIPASE 1 and jasmonic acid influence the outcome of Arabidopsis thaliana–Fusarium graminearum interaction

Syeda T. Alam1,2 | Sujon Sarowar1 | Hossain A. Mondal1,3 | Ragiba Makandar1,4 | Zulkarnain Chowdhury1,2 | Joe Louis1,5 | Jyoti Shah1,2

1Department of Biological Sciences, University of North Texas, Denton, Texas, USA
2BioDiscovery Institute, University of North Texas, Denton, Texas, USA
3College of Postgraduate Studies in Agricultural Sciences (CPGS-AS), under Central Agricultural University, Imphal, India
4Department of Plant Sciences, University of Hyderabad, Gachibowli, India
5Department of Entomology and Department of Biochemistry, University of Nebraska, Lincoln, Nebraska, USA

Correspondence
Jyoti Shah, Department of Biological Sciences, University of North Texas, Denton, TX 76203, USA. Email: Jyoti.Shah@unt.edu

Present address
Sujon Sarowar, Genetic Improvement of Fruits and Vegetables Laboratory, United States Department of Agriculture-Agricultural Research Service, Chatsworth, New Jersey, USA

Funding information
U.S. National Science Foundation, Grant/Award Number: MCB# 1412942; U.S. Department of Agriculture’s National Institute of Food and Agriculture, Grant/Award Number: 2021-67013-33573; U.S. Department of Agriculture, Grant/Award Number: #59-0790-8-060

Abstract
Fusarium graminearum (Fg) is an important fungal pathogen of small grain cereals that can also infect Arabidopsis thaliana. In Arabidopsis, jasmonic acid (JA) signalling involving JASMONATE RESISTANT 1 (JAR1), which synthesizes JA-isoleucine, a signalling form of JA, promotes susceptibility to Fg. Here we show that Arabidopsis MYZUS PERSICAE-INDUCED LIPASE 1 (MPL1), via its influence on limiting JA accumulation, restricts Fg infection. MPL1 expression was up-regulated in response to Fg infection, and MPL1-OE plants, which overexpress MPL1, exhibited enhanced resistance against Fg. In comparison, disease severity was higher on the mpl1 mutant than the wild type. JA content was lower in MPL1-OE and higher in mpl1 than in the wild type, indicating that MPL1 limits JA accumulation. Pharmacological experiments confirmed the importance of MPL1-determined restriction of JA accumulation on curtailment of Fg infection. Methyl-JA application attenuated the MPL1-OE-conferring resistance, while the JA biosynthesis inhibitor ibuprofen enhanced resistance in mpl1. Also, the JA biosynthesis-defective opr3 mutant was epistatic to mpl1, resulting in enhanced resistance in mpl1 opr3 plants. In comparison, JAR1 was not essential for the mpl1-conferring susceptibility to Fg. Considering that methyl-JA promotes Fg growth in culture, we suggest that in part MPL1 curtails disease by limiting the availability of a plant-derived Fg growth-promoting factor.

KEYWORDS
callose, disease resistance, fungal disease, lipase, oxylipin, salicylic acid
Salicylic acid (SA) and jasmonic acid (JA) are important signalling metabolites in plant growth, development, and stress responses (An & Mou, 2014; Chaturvedi & Shah, 2007; Dempsey & Klessig, 2017; Huang et al., 2017; Rivas-San Vicente & Plasencia, 2011; Yan & Xie, 2015; Zhang et al., 2017). Barring some exceptions, the involvement of SA and JA in plant responses to biotic stress is largely viewed as SA contributing to plant defence against pathogens with a biotrophic lifestyle and JA contributing to defence against necrotrophic pathogens and herbivores (Glazebrook, 2005; Yan & Xie, 2015). Although SA and JA signalling have been reported to work synergistically in controlling some diseases (Bostock, 2005), in general, mutually antagonistic interactions between SA and JA influence the outcome of plant–pathogen interaction (Gimenez-Ibanez & Solano, 2013; Kunkel & Brooks, 2002; Thaler et al., 2012). Many pathogens have evolved strategies to exploit this antagonistic interaction between SA and JA signalling to their benefit. For example, the bacterial pathogen *Pseudomonas syringae* pv. *tomato* DC3000 uses coronatine, a toxin that mimics JA-isoleucine, a signalling form of JA, to bind the JA-isoleucine receptor CORONATINE-INSENSITIVE 1 (COI1) to activate JA signalling and thus attenuate SA signalling to the pathogen’s benefit (Bender et al., 1999; Brooks et al., 2004, 2005; Katsir et al., 2008; Kloek et al., 2001). Coronatine biosynthesis genes are present in other pathogens, suggesting that using coronatine-like metabolites to hijack host defences might be a strategy used by many phytopathogens (Zhang et al., 2017). JA signalling is also targeted for activation by effector proteins like HopZ1a and HopX1 produced by other *P. syringae* strains (Gimenez-Ibanez et al., 2014; Jiang et al., 2013). These effectors act on JAZ proteins, which are inhibitors of JA signalling, to promote their degradation by the proteasome and thus stimulate the JA signalling machinery. Other pathogens, especially some necrotrophs and hemibiotrophs, have evolved strategies to metabolize JA and/or suppress JA signalling to facilitate infection (Zhang et al., 2017). In addition, some necrotrophic pathogens produce factors that activate SA signalling to suppress JA signalling in the plant and thus facilitate infection. For example, j1-(1,3) (1,6)-β-glucan, an exopolysaccharide produced by *Botrytis cinerea*, promotes SA accumulation, thereby dampening JA signalling (El Oirdi et al., 2011).

JA-mediated attenuation of SA signalling facilitates plant infection by *Fusarium graminearum* (Fg) (Makandar et al., 2010, 2012). Fg, a hemibiotrophic fungus, causes Fusarium head blight, a devastating disease of floral tissues in wheat (*Triticum aestivum*) and barley (*Hordeum vulgare*) that in the past has resulted in annual losses ranging from $0.3 billion to $3 billion (Bai & Shaner, 2004; Johnson et al., 2003; Wilson et al., 2017; Xu & Nicholson, 2009). Gene-for-gene resistance, which provides strong race-specific immunity against pathogens, is not available for Fusarium head blight in wheat and barley, highlighting the need for identifying plant genes and mechanisms that can be targeted to control this pathogen. In the laboratory, Fg can also infect inflorescence and leaf tissues of *Arabidopsis thaliana*. Brewer and Kosack (2015) have reviewed several studies with the *Arabidopsis–Fg* pathosystem that have uncovered host genes/mechanisms that contribute to defence and susceptibility to this fungus. SA signalling was identified as one component of *Arabidopsis* defence against Fg (Makandar et al., 2006, 2012, 2015). SA application enhanced resistance against Fg in *Arabidopsis* (Makandar et al., 2010). In addition, while resistance against Fg was enhanced in plants overexpressing the SA receptor NPR1 (NONEXPRESSER OF PR GENES 1), it was attenuated in the npr1 mutant, in mutants deficient in SA biosynthesis, and in plants that express an SA-metabolizing salicylate hydroxylase encoded by a bacterial NahG gene (Makandar et al., 2006, 2010; Nalam et al., 2015). The importance of SA signalling in defence against Fg has been extended to wheat and barley (Diethelm et al., 2014; Hao et al., 2018; Makandar et al., 2006, 2012).

It has been suggested that early during infection, which coincides with the biotrophic phase of the infection, SA has an important function in defence against Fg (Ameye et al., 2015; Makandar et al., 2010, 2012). During this stage, SA accumulation and the activation of SA signalling are attenuated by JA signalling. Exposure of *Arabidopsis* to methyl-JA (MeJA) either prior to inoculation or within 6 h of inoculation with Fg resulted in increased disease severity (Makandar et al., 2010). Furthermore, MeJA pretreatment attenuated the NPR1 overexpression-conferred resistance to Fg. Disease severity was reduced in the 12-oxophytodienoate reductase 3-lacking opr3 mutant, which is deficient in JA biosynthesis, the jasmonate resistant 1 (*jar1*) mutant, which is deficient in the synthesis of JA-isoleucine, and the JA-isoleucine coreceptor mutant coi1 (Makandar et al., 2010). MeJA application also attenuated the NPR1 overexpression-conferred resistance in wheat (Makandar et al., 2012). Similarly, Ma et al. (2021) showed that in maize, JA signalling contributes to susceptibility to Gibberella stalk rot (GSR) caused by Fg (teleomorph *Gibberella zeae*). They showed that the JA-deficient maize opr7-5 opr8-2 double mutant and the JA signalling-deficient coi1a mutant exhibited enhanced resistance to GSR. In contrast, MeJA application enhanced GSR severity and the jaz15 mutant, which is deficient in JAZ15, a COI1a-interacting protein that presumably suppresses JA signalling, resulted in increased susceptibility to GSR (Ma et al., 2021).

In *Arabidopsis* and wheat, however, when plants were exposed to MeJA 24 h after inoculation with Fg, disease severity was lower than in plants that received a control treatment. These results led to the suggestion that JA signalling has a dichotomous role in the *Arabidopsis* and wheat interaction with Fg, contributing to susceptibility during the early phase of infection via its effect on curtailing SA signalling. However, after this period of SA signalling activation, JA signalling is proposed to contribute to resistance (Makandar et al., 2010, 2012). The temporal fashion of SA and JA accumulation and expression of SA- and JA-responsive genes supports this model of a sequential involvement of SA and JA in defence against Fg (Ameye et al., 2015; Ding et al., 2011; Steiner et al., 2009). Thus, mechanisms that impact JA accumulation could serve as potential targets for enhancing host plant resistance to Fg infection.

JA belongs to a class of oxidized lipids (oxylipins) that are derived from polyunsaturated fatty acids. Fungi and other eukaryotes also produce oxylipins (Brodhun & Feussner, 2011; Nover et al., 2003).
In fungi, oxylipins are involved in controlling density-dependent processes, facilitating colonization of the host plant, and regulating mycotoxin production (Brown et al., 2009; Tsitsigiannis & Keller, 2007). In some cases, fungal oxylipins have been shown to mimic plant signals and plant oxylipins shown to substitute for fungal oxylipins (Brodhagen et al., 2008; Burow et al., 1997; Christensen & Kolomiets, 2011). The concept of cross-kingdom communication has been proposed to describe the contribution of these oxylipins in symbiotic interactions (Tsitsigiannis & Keller, 2007), wherein oxylipins produced by one symbiont are perceived by the other symbiont to modulate the interaction. JA and derivatives are produced by some plant-pathogenic fungi, including Fusarium spp. (Cole et al., 2014; Miersch et al., 1999; Oliw & Hamberg, 2017; Tsukada et al., 2010). A functional JA signalling mechanism in Arabidopsis is critical for root colonization by Fusarium oxysporum varieties that synthesize JA-isoleucine, thus confirming the responsiveness of Arabidopsis to JA-isoleucine synthesized by the fungal pathogen (Cole et al., 2014). However, whether Fusarium spp. perceive and respond to jasmonates produced by plants is not known.

Here we show that MYZUS PERSICAE-INDUCED LIPASE 1 (MPL1), which encodes an α/β-fold lipase that was previously shown to be involved in controlling green peach aphid (Myzus persicae) infestation on Arabidopsis (Louise et al., 2010), promotes defences against pathogens and limits JA accumulation and Fg infection. Pharmacological and genetic experiments suggest a relationship between the ability of MPL1 to control JA accumulation and to limit Fg disease severity. However, the impact of MPL1 on limiting disease caused by Fg is largely independent of JAR1 and is not mediated by the attenuation of the repressive effect of JA on SA accumulation and signalling. Because JA directly promotes Fg growth in culture, we suggest that in addition to promoting defences, MPL1 restricts Fg infection by limiting availability of a potential JA-dependent Fg growth-promoting factor.

2 | RESULTS

2.1 | MPL1 is expressed at high levels in Arabidopsis inflorescence tissue

A meta-analysis of gene expression data available in the public domain (https://www.genevestigator.com/) indicated that compared to the developed rosette, MPL1 expression is higher in young and developed flowers and highest in mature siliques and germinating seeds of Arabidopsis (Louise et al., 2010). To confirm that MPL1 expression is higher in flowers, reverse transcription-quantitative PCR (RT-qPCR) analysis was conducted on RNA extracted from flowers, the inflorescence stem, cauline leaves, and control rosette leaves of the Arabidopsis accession Columbia-0 (Col-0). As shown in Figure S1a, the MPL1 transcript level was higher in flowers compared to the rosette and cauline leaves. Expression in the inflorescence stem was intermediate between that in leaves and flowers. We further tested the expression pattern of MPL1 in Arabidopsis containing a MPL1::uidA chimera, which expresses the Escherichia coli uidA-encoded β-glucuronidase (GUS) reporter under the control of the MPL1 promoter. In congruence with the RT-qPCR data (Figure S1a), histochemical analysis showed stronger MPL1 promoter-driven GUS activity in the inflorescence stems and flowers, and relatively poor activity in leaves (Figure S1b). In the flowers, GUS activity was observed in sepal (Figure S2a–d), in petals (Figure S2e), in the pistil in an area immediately below the stigma (Figures S1b and S2f), at the base of the style (Figure S1b), and in the stamen filament (Figure S2g). GUS activity was also observed at the pedicel and the apex of developing siliques (Figure S2h), at the seed base (Figure S2i), and at the radicle of germinating seeds (Figure S1b). In rosette leaves, GUS activity was largely restricted to the hydathodes (Figure S1b).

2.2 | MPL1 expression is up-regulated in response to Fg infection

While studying the association of genes that were discovered in our laboratory with biotic stress, we observed that MPL1 expression is up-regulated in Fg-inoculated inflorescence tissue (Figure 1a). This increase in MPL1 expression was validated in experiments with MPL1::uidA plants. MPL1 promoter-driven GUS activity was stronger in the flowers of Fg-inoculated plants compared to mock-inoculated plants (Figure 1a). Likewise, stronger GUS activity was observed in the siliques of fungus-inoculated plants compared to mock-inoculated plants (Figure 1a). Fg infection of leaves also resulted in an increase in MPL1 transcript accumulation and activity of the MPL1::uidA reporter (Figure 1b). GUS activity was strongest at the site of Fg inoculation.

2.3 | MPL1 contributes to Arabidopsis defence against Fg

Encouraged by the increase in MPL1 expression in response to Fg infection of Arabidopsis inflorescence tissues and leaves, we hypothesized that MPL1 has a role in the Arabidopsis–Fg interaction. To test this hypothesis, we compared Fg disease severity on the wild-type (WT) accession Col-0, two previously described T-DNA-containing mpl1-1 and mpl1-2 knockout mutants (Louise et al., 2010), and two independent MPL1-overexpressing (MPL1-OE) lines (Louise et al., 2010) in a Col-0 background. As shown in Figures 2a and S3a, compared to the WT, disease severity on inflorescence tissues, represented as the Fusarium–Arabidopsis Disease (FAD) score (Nalam et al., 2016; Urban et al., 2002), was higher on the mpl1-1 and mpl1-2 mutants. In contrast, the FAD score was lower on both MPL1-OE lines than on the WT. Similarly, disease severity was also lower on MPL1-OE leaves compared to WT (Figures 2b and S3b). In contrast, disease progression was faster on leaves of the mpl1-1 and mpl1-2 mutants. At 4 days postinoculation (dpi) but not at 5 dpi, disease severity was higher on mpl1-1 and mpl1-2 leaves than on WT leaves (Figures 2b and S3b). This increase in disease severity correlated with increased...
Fg DNA accumulation observed in mpl1-1 and mpl1-2 mutants than in the WT (Figure 2c). In contrast, Fg growth was slower on the MPL1-OE lines than on the WT (Figure 2c). These results suggest that MPL1 contributes to Arabidopsis defence against Fg. In support of this proposition, callose deposition and reactive oxygen species (ROS) accumulation, two processes associated with defence against pathogens, were found to be higher in the fungus-inoculated leaves of MPL1-OE than in those of WT plants (Figure 3a,b).

### 2.4 MPL1 overexpression overcomes the requirement of SA for conferring resistance to Fg

SA has a critical function as a signalling metabolite in plant defence against Fg (Diethelm et al., 2014; Hao et al., 2018; Makandar et al., 2006, 2010, 2012). In addition, under some growth conditions SA inhibits Fg mycelial growth (Figure S4) and conidial germination (Qi et al., 2012). SA content was higher in uninfected and Fg-inoculated MPL1-OE plants compared to WT plants (Figure 4a,b). To determine if this high SA content was responsible for the higher level of resistance to Fg observed in MPL1-OE plants, we tested Fg disease severity in MPL1-OE nahG plants, which express the Pseudomonas putida nahG-encoded salicylate hydroxylase, which removes SA by metabolizing it to catechol. As anticipated, the presence of nahG resulted in a lower SA content in MPL1-OE nahG compared to MPL1-OE plants (Figure 4b). Furthermore, unlike the WT and MPL1-OE plants, the presence of nahG prevented SA increases in the Fg-inoculated MPL1-OE nahG plants. However, despite the nahG-conferred reduction in SA content, the MPL1-OE-conferred resistance against Fg was retained in the leaves and inflorescences of MPL1-OE nahG plants (Figure 4c). Previously it was reported that resistance to Fg was not affected by catechol (Makandar et al., 2012); thus, it is unlikely that the resistance preserved in the MPL1-OE nahG plants is due to catechol.

As a control to monitor the effectiveness of nahG in attenuating SA-mediated defence in MPL1-OE nahG plants, we also compared the resistance level of MPL1-OE nahG and MPL1-OE plants to the...
bacterial pathogen *P. syringae* pv. *maculicola*. SA is essential for defence against *P. syringae* (Chaturvedi & Shah, 2007; Shah, 2003) and MPL1 expression is induced in response to *P. syringae* pv. *maculicola* infection (Figure S5a). Furthermore, compared to the WT, mpl1-1 plants are more susceptible and MPL1-OE plants are more resistant to *P. syringae* pv. *maculicola* (Figure S5b). However, unlike resistance against Fg, the MPL1-OE-conferring resistance against *P. syringae* pv. *maculicola* was attenuated in MPL1-OE nahG plants (Figure S5c), confirming the effectiveness of nahG in abolishing SA-dependent resistance in MPL1-OE nahG plants. These results indicate that the resistance-promoting effect of MPL1-OE towards Fg results from factors that are independent of MPL1-OE’s effect on promoting SA accumulation.

We also did not see any correlation between resistance to Fg and expression of the SA-responsive PR1 gene in mpl1-1 and MPL1-OE plants. PR1 expression was up-regulated to comparably high levels in response to Fg inoculation in the Fg-resistant MPL1-OE and the more susceptible mpl1-1 plants (Figure S6). Furthermore, SA application restored resistance in the mpl1-1 mutant to levels comparable to that in WT plants (Figure S7), confirming that the mpl1-1 mutant is responsive to SA. Taken together, these results indicate that SA does not make a major contribution to MPL1’s involvement in defence against Fg.

### 2.5 | MPL1 limits JA accumulation to promote resistance against Fg

To determine if there is a relationship between JA and MPL1 that influences the *Arabidopsis* interaction with Fg, we determined the JA contents in mpl1 and MPL1-OE plants. As shown in Figure 5a,b, the JA content was higher in the mpl1 mutants and lower in the MPL1-OE lines compared to the WT. Fg infection did not result in a significant change in JA content in the WT (Figure 5b). Similarly, no changes in JA content were observed between the mock- and Fg-inoculated MPL1-OE plants (Figure 5b). While expression of the JA-responsive gene LOX2 was up-regulated in response to Fg infection, another JA-responsive gene (MYC2) was down-regulated in response to Fg infection in the WT. In contrast, both JA-responsive genes were down-regulated in the fungus-inoculated MPL1-OE plants. In contrast, Fg infection resulted in the up-regulation of both JA-responsive genes in the mpl1-1 mutant (Figure 5c), suggesting hyperstimulation of JA signalling in the Fg-challenged mpl1-1 and repression of JA signalling in MPL1-OE plants.

**FIGURE 2  *Fusarium graminearum* disease severity in *Arabidopsis* mpl1 mutants and MPL1-overexpressing plants.** (a) Disease severity expressed as the Fusarium–*Arabidopsis* Disease (FAD) score in inflorescences of *Arabidopsis* wild-type (WT) accession Columbia-0, the mpl1-1 and mpl1-2 mutants, and two independently derived MPL1-overexpressing (MPL1-OE) lines. Disease was scored at 7 days postinoculation (dpi). Error bars represent SE (n = 10). (b) *F. graminearum* disease severity in leaves of *Arabidopsis*. Upper panel: Disease severity in WT and MPL1-OE plants at 5 dpi. Lower panel: Disease severity in WT and mpl1-1 and mpl1-2 mutants at 4 and 5 dpi. Error bars represent SE (n = 30). (c) Fungal DNA accumulation in fungus-inoculated leaves of *Arabidopsis* genotypes. Quantitative PCR evaluation of the *F. graminearum* nahG gene relative to *Arabidopsis* ACT8 in WT, mpl1-1 and mpl1-2 mutants, and the MPL1-OE lines at 5 dpi. Error bars represent SE (n = 3). Different letters above bars indicate values that are significantly different from each other (p < 0.05). The above experiments were conducted three times with similar pattern of results.
were exposed to MeJA vapour for a 24 h-period prior to inoculation with Fg. As shown in Figure 5d, unlike the low disease severity observed in the control (0.1% ethanol-treated) MPL1-OE plants, disease severity in inflorescence and leaf tissues was comparably high in the MeJA-treated MPL1-OE and WT plants. The contribution of JA in conferring susceptibility to Fg in the mpl1-1 mutant was further tested by pretreating plants with ibuprofen, an inhibitor of JA biosynthesis (Bohlmann et al., 1998; Louis et al., 2015). As shown in Figure 5e, ibuprofen treatment reduced Fg disease severity to comparable levels in the WT and the mpl1-1 mutant. In comparison, ibuprofen did not enhance resistance in MPL1-OE plants. Taken together, these results suggest that MPL1’s negative effect on JA accumulation is relevant to MPL1’s involvement in promoting resistance to Fg.

The above hypothesis was further tested by evaluating disease severity in mpl1-1 opr3-2 double mutant plants. The opr3-2 mutant lacks OPDA reductase activity, which is involved in JA biosynthesis (Bi et al., 2010; Shiva et al., 2020). Previously it was shown that plants lacking OPR3 exhibit enhanced resistance to Fg (Makandar et al., 2010). Furthermore, similar to MPL1-OE, the reduced Fg disease severity in opr3-2 compared to the WT is accompanied by increased deposition of callose (Figure S8a). As shown in Figure 5f, opr3-2 was epistatic to mpl1-1. Similar to opr3-2, Fg disease severity was lower in mpl1-1 opr3-2 compared to mpl1-1, supporting the suggestion that MPL1 contributes to Arabidopsis defence against Fg by controlling JA accumulation. The fact that no higher ROS levels were observed in the opr3-2 mutant compared to the WT (Figure S8b) suggests that MPL1’s impact on ROS production is probably independent of its impact on limiting JA accumulation.

Previously, it was shown that JAR1 is required for restricting the activation of SA signalling in response to Fg infection (Makandar et al., 2010). Compared to the WT, the jar1-1 mutant exhibits higher resistance to Fg (Figure 5f) (Makandar et al., 2010). To determine if JAR1 is required for the mpl1-1-conferring enhanced susceptibility to Fg, we compared disease severity between the mpl1-1 jar1-1 and the mpl1-1 and jar1-1 mutants. We hypothesized that if the higher disease severity caused by Fg in the mpl1-1 mutant is mediated by JAR1, then Fg disease severity in the mpl1-1 jar1-1 double mutant should be low and resemble the disease severity in jar1-1. However, counter to our hypothesis, as shown in Figure 5f, disease severity on mpl1-1 jar1-1 was higher than on jar1-1 and not significantly different from that on mpl1-1 and WT, suggesting that JAR1 and hence JA-isojleucine are not major contributors to the higher Fg disease severity in mpl1-1.

To determine if the low level of JA is associated with the high level of resistance to Fg in MPL1-OE plants, we tested disease severity in inflorescence and leaf tissues of WT and MPL1-OE plants that

2.6 | MeJA promotes Fg growth in culture

In the case of Arabidopsis and wheat interaction with Fg, an oxylipin-synthesizing lipoxygenase serves as a susceptibility factor (Nalam et al., 2015). Oxylipins influence growth, development, and mycotoxin production by some fungal pathogens, and in some cases plant oxylipins can substitute for fungal oxylipins (Brodhagen et al.,
oxylipins like JA could directly affect Fg growth. We tested this possibility by studying the impact of MeJA on Fg macroconidia germination and fungal growth in culture. As shown in Figure 6a,b, compared to the control treatment, Fg macroconidia germination and growth were better in the presence of MeJA, thus raising the possibility that MPL1 curtails the availability of a JA-dependent Fg growth-promoting factor via its impact on limiting JA accumulation, which along with MPL1's ability to promote defences contributes to limiting Fg disease severity on Arabidopsis.

### DISCUSSION

Studying the function of MPL1 in Arabidopsis, we found that MPL1 is involved in limiting JA accumulation. JA levels were lower in MPL1-OE and higher in mpl1 mutant plants. How MPL1, which encodes a lipase (Louis et al., 2010), limits JA accumulation is unclear. MPL1 has homology to eukaryotic proteins involved in formation of lipid droplets and thus lipid compartmentation (Chapman et al., 2012), which could affect the availability of lipids for JA biosynthesis. Alternatively, MPL1 could be involved in promoting the turnover of JA or its precursor.

JA has an important role in Arabidopsis and wheat susceptibility to Fg. The following observations suggest that the diminution of JA accumulation by MPL1 contributes to limiting Fg infection on Arabidopsis: (a) MeJA application suppressed the MPL1-OE-conferred enhanced resistance against Fg; (b) in contrast, the JA biosynthesis inhibitor ibuprofen restored resistance of the mpl1-1 mutant to WT levels; and (c) opr3-2, which is deficient in JA biosynthesis, was epistatic to mpl1-1, resulting in higher than WT levels of resistance against Fg in mpl1-1 opr3-2.

JA’s involvement in Arabidopsis and wheat susceptibility to Fg has been attributed to the antagonistic effect of JA on SA accumulation and signalling (Makandar et al., 2010, 2012). In Arabidopsis, this antagonism is mediated by JAR1 (Makandar et al., 2010). SA accumulation and signalling in response to Fg infection were not adversely impacted in the mpl1-1 mutant compared to the WT. The comparably low Fg disease severity in MPL1-OE and the SA-deficient MPL1-OE nahG plants further confirms that MPL1's impact on limiting Fg infection is independent of SA. Taken together, the results presented here indicate that the impact of MPL1 on the Arabidopsis–Fg interaction is not associated with JA's antagonistic effect on SA accumulation and signalling. Experiments with mpl1-1 jar1-1 plants further support our suggestion that the antagonism between JA signalling and SA is not critical to the involvement of MPL1 in the interaction of Arabidopsis with Fg. While Fg disease severity is higher in mpl1-1 and lower in jar1-1 than in the WT, Fg disease severity in mpl1-1 jar1-1 was higher than in jar1-1 and not significantly different from that in mpl1-1. Hence, we conclude that JAR1 function is not important to
MPL1-1 conferred susceptibility to Fg. However, considering that disease severity in the mpl1-1 jar1-1 was also not significantly different from WT, we cannot rule out a small contribution of JAR1 to the higher susceptibility to Fg in plants lacking MPL1 function.

We noted that Fg growth in culture is promoted by MeJA. This raises the possibility that elevated JA content in mpl1-1 directly stimulates Fg growth. In contrast, the lower levels of JA in MPL1-OE plants may limit availability of an Fg growth-promoting factor, which when combined with the higher level of defence activities like callose deposition and ROS production may contribute to the higher level of resistance against Fg in MPL1-OE plants. As noted above, the cross-kingdom communication during the symbiosis between plants and fungi invokes oxylipins. Oxylipins control density-dependent processes, plant colonization, and...
regulation of mycotoxin production in some fungi (Brown et al., 2009; Tsitsigiannis & Keller, 2007). Similarly, plant-derived JA, or a dependent product, might control Fg growth/development. A lipoxygenase serves as a susceptibility factor in the Arabidopsis and wheat interaction with Fg (Nalam et al., 2010). Asp and His, which correspond to Asp360 and His393 in MPL1, are present in the wheat MPL1-like proteins. The sequences around these Ser, Asp, and His residues are also conserved between MPL1 and the wheat MPL1-like proteins. Whether these MPL1-like wheat proteins are the wheat orthologs of MPL1 remains to be determined.

In conclusion, we provide evidence that MPL1 via its effect on limiting JA accumulation promotes resistance against Fg, an important phytopathogen. Past studies have shown that SA signalling is an important target for controlling Fg infection in Arabidopsis, wheat, and barley (Diethelm et al., 2014; Hao et al., 2018; Makandar et al., 2006, 2012). This work shows that JA accumulation and the MPL1-dependent mechanism provide additional targets for controlling Fg infection and limiting agricultural losses due to infections by Fg. However, considering that JA has a dichotomous involvement in wheat interaction with Fg (Makandar et al., 2010), it is important to understand the mechanism underlying MPL1’s ability to limit JA accumulation and to identify the JA-dependent factor that promotes Fg spor germination and growth.

FIGURE 6  Methyl-jasmonic acid (MeJA) promotes Fusarium graminearum (Fg) growth. (a) Fg spor germination in potato dextrose broth in the presence of different concentrations of MeJA. Spor germination was monitored under the microscope at 6 h after seeding of culture media with Fg macroconidia. Error bars represent SE (n = 5). Asterisks indicate values that are significantly different (p < 0.05) from the control without MeJA. (b) Fg growth on potato dextrose agar plates incubated in the presence of MeJA vapour and as control in plates exposed to ethanol, which was used as a solvent for MeJA. The four sectors on each plate were spotted with 0, 50, 200, and 2000 macroconidia and the plates were imaged 3 days postinoculation. The experiments in (a) and (b) were conducted twice with similar results.

4 | EXPERIMENTAL PROCEDURES

4.1 | Plant genotypes and cultivation

Arabidopsis was cultivated in growth chambers at 22°C under a 14-h light (80–100 μmol-m⁻²-s⁻¹) and 10-h dark regime as previously described (Nalam et al., 2016). The Salk_101919 (mpl1-1) and Salk_082589 (mpl1-2) lines and the MPL1-OE lines, which are in a Col-0 background, were previously described (Louis et al., 2010). The opr3-2 allele, which contains a G-to-A base substitution that results in the replacement of Trp138 by a stop codon (Bi et al., 2010; Shiva et al., 2020), was a gift of Dr Jian-Min Zhou. A cross between opr3-2 and mpl1-1 was used to generate the mpl1-1 opr3-2 double mutant. Presence of the mpl1-1 allele was followed by PCR with the primers mpl1-1-F and LhB1.3, which yield a product only with the mpl1-1 template. A dCAPS was used to differentiate between the OPR3 and opr3-2 alleles. PCR was performed with the primers OPR3-dCAPS-F and opr3-2-dCAPS-BsuRI-R to yield a 192-bp ampliion. The ampliion derived from the OPR3 template contains a BsuRI site that upon digestion with BsuRI yields two products of 31 and 161 bp. In comparison, the ampliion derived from opr3-2 lacks the BsuRI site and hence is recovered as the undigested 192-bp DNA fragment. A cross between mpl1-1 and jar1-1 was used to generate the mpl1-1 jar1-1 double mutant. CAPS was used to differentiate between the JAR1 and jar1-1 alleles. PCR was performed with the primers jar1-1-CAPS-HindIII-F and jar1-1-CAPS-R to yield a 143-bp ampliion, which was treated with the restriction enzyme HindIII. The ampliion derived from the jar1-1 template contains a HindIII site and upon digestion with HindIII yields two products of 27 and 116 bp. In comparison,
the amplicon derived from JAR1 lacks the HindIII site and hence is recovered as the undigested 143-bp DNA fragment.

### 4.2 Pathogen strains, culture conditions, and plant infection

Cultivation of *P. syringae* pv. *maculicola* ES4266, inoculation of *Arabidopsis* leaves, and the monitoring of bacterial numbers were conducted as previously described (Nandi et al., 2003). Cultivation of Fg isolate Z-3639, sporulation, fungal inoculation of leaf and inflorescence tissues, and disease evaluation and scoring were conducted as previously described (Nalam et al., 2016). Leaf infection assays were performed on 4-week-old plants by infiltrating leaves with a suspension containing fungal mycelial fragments. Unless stated otherwise, the extent of chlorosis was scored 5 days later. A minimum of 30 leaves from 8-12 plants of each genotype were used for each experiment. Based on the area of the leaves exhibiting chlorosis, the inoculated leaves were grouped into four categories: category 1 (chlorosis covering <25% of the leaf area), category II (chlorosis covering 25%–50% of the leaf area), category III (chlorosis covering 51%–75% of the leaf area), and category IV (chlorosis covering >75% of the leaf area). The numbers of leaves in each category were used to calculate the disease severity index as previously described (Nalam et al., 2016).

Inflorescence infections were carried out by spraying Fg macroconidia on the inflorescences of 6–7-week-old plants. Disease severity on inflorescence tissue was expressed as the FAD score, which was calculated as previously described (Nalam et al., 2016). A minimum of 10 plants per genotype were used for each experiment. Fungal DNA accumulation was quantified by qPCR performed on DNA isolated from Fg-infected tissues using primers FgNahG-F and FgNahG-R specific to the fungal gene FGSG_08116 as previously described (Nandi et al., 2003). Two technical replicates were included for each biological sample. The expression levels of each biological sample for gene expression studies. Two technical replicates were included for each biological sample. The expression levels of MPL1, PR1, LOX2, and MYC2 were normalized to that of ACT8 and/or the

### 4.3 RNA isolation and RT-qPCR

An acid guanidinium thiocyanate/phenol/chloroform mix (Chomczynski & Sacchi, 1987) was used to extract total RNA from *Arabidopsis* leaf and inflorescence tissues. Contaminating DNA was removed by RQ1 DNase (Promega) followed by cDNA synthesis with GoScript reverse transcriptase (Promega) and an oligo(dt) 18-mer primer (New England Biolabs). qPCR was carried out with iTaq UniversalSYBR Green mix (Bio-Rad) on an Eco qPCR system (https://illumina.com). Each biological sample for gene expression studies consisted of two or three leaves or inflorescences, each harvested from a separate plant. A minimum of three biological samples were included for gene expression analysis. Two technical repeats were included for each biological sample. The expression levels of MPL1, PR1, LOX2, and MYC2 were normalized to that of ACT8 and/or the

### 4.4 GUS reporter construct and histochemical GUS staining

A 1785-bp PCR product containing the MPL1 promoter was amplified with the primers MPL1-Pro-F and MPL1-Pro-R (Table S1) and cloned into the pCR8/GW/TOPO Entry vector (Life Technologies), from which the insert was mobilized with the Gateway LR cloning system (Life Technologies) into the Destination binary vector pMDC162 (Curtis & Grossniklaus, 2003). The resultant plasmid pMDC162-MPL1:uidA, which contains the MPL1 promoter placed upstream of the E. coli uidA coding sequence, was transformed into *Agrobacterium tumefaciens* GV3101, from where it was delivered into *Arabidopsis* Col-0 by the floral dip method (Clough & Bent, 1998). Transgenic plants containing the MPL1:uidA chimera were selected on Murashige and Skoog agar plates based on their resistance to hygromycin (20 mg/L) as previously described (Oono et al., 1998). The presence of the insert was confirmed by PCR on DNA extracted from the transgenic plants. Histochemical analysis of GUS activity was performed using 5-bromo-4-chloro-3-indolyl-β-D-glucopyranosiduronic acid (Gold Biotechnology) as the substrate.

### 4.5 Hormone and chemical treatment of plants

SA treatment was performed by irrigating and simultaneously spraying the plants with a 200 µM solution of sodium salicylate in water. Plants were inoculated with Fg 24 h later, and disease severity was evaluated at 7 dpi. Plants irrigated and sprayed with water served as the controls.

MeJA treatment was performed according to Makandar et al. (2010). A cotton plug containing 1 ml of a MeJA solution (200 µM) prepared in 0.1% ethanol was placed in a Petri plate at the centre of an airtight plexiglass chamber (volume, 48 L) that contained *Arabidopsis* plants. The plants were left in the chamber for 24 h. Plants similarly placed in a chamber containing 1 ml 0.1% ethanol served as the controls for these experiments. At the end of the treatment, plants were inoculated with Fg as described above.

Ibuprofen treatment was performed by irrigating *Arabidopsis* plants with a solution (100 ppm) of ibuprofen in water (controls were irrigated with water). Twenty-four hours later, the inflorescence was sprayed with Fg spores and disease severity was evaluated 7 days later (Nalam et al., 2016).

### 4.6 Effect of SA and JA on fungal growth

The spore germination assay was conducted by diluting freshly harvested macroconidia to a concentration of 10,000 macroconidia/ml in potato dextrose broth containing different concentrations of
MeJA or 0.01% ethanol (control), which was used to dissolve MeJA. All macroconidia contained multiple cells. The emergence of hyphae from these cells was monitored under a compound microscope at various time points after addition of MeJA or ethanol. Each emerged hypha was considered as one germinated spore cell.

To study the effect of MeJA on fungal growth, different amounts of fungal macroconidia were spotted on potato dextrose agar. One set of potato dextrose agar plates spotted with Fg macroconidia were placed in a plexiglass chamber (volume, 48 L) that contained a cotton plug soaked in 1 ml MeJA solution (200 µM) prepared in 0.1% ethanol. As a control, the second set of potato dextrose agar plates spotted with Fg macroconidia were placed in a plexiglass chamber along with a cotton plug soaked in 1 ml ethanol (0.1%). Fg was allowed to grow on these plants at 28°C. To monitor the effect of SA on fungal growth, different amounts of fungal macroconidia were spotted on potato dextrose agar supplemented with 200 µM sodium salicylate. Potato dextrose agar lacking sodium salicylate served as the control.

### 4.7 Hormone quantification

Fully expanded leaves of approximately 4-week-old plants were used for JA and SA estimations. To monitor the effect of fungal infection on JA and SA levels, Fg-inoculated and water-treated (control) leaves were harvested 24 h postinoculation. A minimum of three biological samples were included for hormone quantification. Each sample consisted of two leaves, each harvested from a separate plant. JA extracted from leaves was purified, derivatized, and quantified against a D5-JA standard by gas chromatography–mass spectrometry (Kilaru et al., 2007) or by high-performance liquid chromatography–mass spectrometry (Pan et al., 2010). SA content in *Arabidopsis* leaves was measured using a modified biosensor strain of *Acinetobacter* ADP1 (Huang et al., 2005, 2006).

### 4.8 Quantification of callose deposition

Leaf tissues were used for callose staining and quantification, which was conducted by a previously described method (Ton & Mauch-Mani, 2004) as modified by Mondal et al. (2018). Callose deposits were observed with an epifluorescence microscope and a UV filter (bandpass filter, 340–380 nm; longpass filter, 425 nm). Digital images were used to quantify callose spots in the leaf tissues.

### 4.9 ROS determination

ROS production was examined using a previously described method of staining with 3,3′-diaminobenzidine tetrahydrochloride (DAB) (Gao et al., 2013). Briefly, leaves from 4-week-old plants were inoculated with Fg. The leaves were harvested 18–24 h later and immediately immersed in a 1 mg/ml DAB solution (pH 3.8; Sigma-Aldrich). Vacuum was applied followed by slow release to allow the solution to infiltrate the leaf. Following an 8 h incubation in the dark at room temperature, the stained leaves were fixed and cleared in alcoholic lactophenol (2:1:1 mix of 95% ethanol, lactic acid, and phenol) at 65°C, and then rinsed once with 50% ethanol and twice with water. Cleared leaves were stored in 50% glycerol prior to observation under a light microscope.

### 4.10 Statistical analysis

When comparing two genotypes or treatments, the two-tailed Student’s t test was used to assess the significance of variance (p < 0.05). When comparing multiple genotypes or treatments to each other, analysis of variance (ANOVA) was used following the general linear model. Tukey’s test was used to assess the significance of variance (p < 0.05).

### ACKNOWLEDGEMENTS

The authors would like to thank Dr Jian-Min Zhou for the opr3-2 allele, Dr Monika Patel and Elena Shulaev for assistance with *Arabidopsis* crosses and nucleic acid extraction from plant tissues, and Dr Jantana Keereetaweep and the Kansas Lipidomics Research Center for help with the JA measurements. This work was partially supported at different times by grants from the U.S. National Science Foundation (MCB award # 1412942), the U.S. Department of Agriculture (agreement #59-0790-8-060), and the U.S. Department of Agriculture’s National Institute of Food and Agriculture (award # 2021-67013-33573) to J.S. S.T.A. and Z.C. were supported by a graduate assistantship from the University of North Texas.

### CONFLICT OF INTEREST

The authors declare no conflict of interest.

### DATA AVAILABILITY STATEMENT

All data supporting the findings of this study are available within the paper and its supplementary data published online.

### ORCID

Jyoti Shah 🌼 https://orcid.org/0000-0002-5604-1771

### REFERENCES

Ameye, M., Audenaert, K., De Zutter, N., Steppe, K., Van Meulebroek, L., Vanhaecke, L. et al. (2015) Priming of wheat with the green leaf volatile Z-3-hexenyl acetate enhances defense against *Fusarium graminearum* but boosts deoxynivalenol production. *Plant Physiology*, 167, 1671–1684.

An, C. & Mou, Z. (2014) Salicylic acid and defense responses in plants. In: Tran, L.S. & Pal, S. (Eds.) *Phytohormones: a window to metabolism, signaling and biotechnological applications*. New York, NY: Springer.

Bai, G.H. & Shaner, G. (2004) Management and resistance in wheat and barley to Fusarium head blight. *Annual Review of Phytopathology*, 42, 135–161.

Bender, C.L., Alarcón-Chávez, F. & Gross, D.C. (1999) *Pseudomonas syringae* phytopathogens: mode of action, regulation, and biosynthesis
by peptide and polyketide synthetases. Microbiology and Molecular Biology Reviews, 63, 266–292.

Bi, D., Cheng, Y.T., Li, X. & Zhang, Y. (2010) Activation of plant immune responses by a gain-of-function mutation in an atypical receptor-like kinase. Plant Physiology, 153, 1771–1779.

Blow, D. (1990) More of the catalytic triad. Nature, 343, 694–695.

Bohlmann, H., Vignutelli, A., Hilpert, B., Miersch, O., Westernack, C. & Apel, K. (1998) Wounding and chemicals induce expression of the Arabidopsis thaliana gene Thi2.1, encoding a fungal defense thionin, via the octadecanoic pathway. FEBS Letters, 437, 281–286.

Bostock, R.M. (2005) Signal crosstalk and induced resistance: straddling the line between cost and benefit. Annual Review of Phytopathology, 43, 545–580.

Brady, L., Brzozowski, A.M., Derewenda, Z.S., Dodson, E., Dodson, G.G., Tolley, S. et al. (1990) A serine protease triad forms the catalytic centre of a triacylglycerol lipase. Nature, 343, 677–679.

Brewer, H.C. & Hammond-Kosack, K.E. (2015) Host to a stranger: Arabidopsis and Fusarium ear blight. Trends in Plant Science, 20, 651–663.

Brodhagen, M., Tsitsigiannis, D.I., Hornung, E., Goebel, C., Feussner, I., Brooks, D.M., Hernández-Guzmán, G., Kloek, A.P., Alarcón-Chaidez, F., Bostock, R.M. (2005) Signal crosstalk and induced resistance: straddling the line between cost and benefit. Annual Review of Phytopathology, 43, 545–580.

Christensen, S.A. & Kolomiets, M.V. (2011) The lipid language of plant–microbe interactions. Phytochemistry, 72, 1339–1348.

Cole, S.J., Yoon, A.J., Faull, K.F. & Diener, A.C. (2014) Host perception of jasmonates promotes infection by Fusarium oxysporum formae specialis that produce isoleucine- and leucine-conjugated jasmonates. Molecular Plant-Microbe Interactions, 27, 1047–1063.

Gimenez-Ibanez, S. & Solano, R. (2013) Nuclear jasmonate and salicylate signaling and crosstalk in defense against pathogens. Frontiers in Plant Science, 4, 72.

Gimenez-Ibanez, S., Boter, M., Fernandez-Barbero, G., Chini, A., Rathjen, J.P. & Solano, R. (2014) The bacterial effector HopX1 targets JAZ transcriptional repressors to activate jasmonate signaling and promote infection in Arabidopsis. PLoS Biology, 12, e1001792.

Glazebrook, J. (2005) Contrasting mechanisms of defense against biotrophic and necrotrophic pathogens. Annual Review of Phytopathology, 43, 205–227.

Hao, Q., Wang, W., Han, X., Wu, J., Lyu, B., Chen, F. et al. (2018) Isochorismate-based salicylic acid biosynthesis confers basal resistance to Fusarium graminearum in barley. Molecular Plant Pathology, 19, 1995–2010.

Huang, H., Liu, B., Liu, L. & Song, S. (2017) Jasmonate action in plant growth and development. Journal of Experimental Botany, 68, 1349–1359.

Huang, W.E., Huang, L., Preston, G.M., Naylor, M., Carr, J.P., Li, Y. et al. (2006) Quantitative in situ assay of salicylic acid in tobacco leaves using a genetically modified biosensor strain of Acinetobacter sp. ADP1. The Plant Journal, 46, 1073–1083.

Huang, W.E., Wang, H., Zheng, H., Huang, L., Singer, A.C., Thompson, I. et al. (2005) Chromosomally located gene fusions constructed in Acinetobacter sp. ADP1 for the detection of salicylate. Environmental Microbiology, 7, 1339–1348.

Jiang, S., Yao, J., Ma, K.W., Zhou, H., Song, J., He, S.Y. et al. (2013) Bacterial effector activates jasmonate signaling by directly targeting JAZ transcriptional regulators. PLoS Pathogens, 9, e1003715.

Johnson, D.D., Flaskerud, G.K., Taylor, R.D. & Satyanarayana, V. (2003) Quantifying economic impacts of Fusarium head blight in wheat. In: Leonard, K.J. & Bushnell, W.R. (Eds.) Fusarium head blight of wheat and barley. St Paul, MN: American Phytopathological Society Press, pp. 461–483.

Katsir, L., Schlimmer, A.L., Stawiski, P.E., He, S.Y. & Howe, G.A. (2008) COII is a critical component of a receptor for jasmonate and the bacterial virulence factor coronatine. Proceedings of the National Academy of Sciences of the United States of America, 105, 7100–7106.

Kilaru, A., Bailey, B.A. & Hasenstein, K.H. (2007) Moniliformis permiscia produces hormones and alters endogenous auxin and salicylic acid in infected cocoa leaves. FEMS Microbiology Letters, 274, 238–244.

Kloek, A.P., Verbsky, M.L., Sharma, S.B., Schoelz, J.E., Vogel, J., Klessig, D.F. et al. (2001) Resistance to Pseudomonas syringae conferred by an Arabidopsis thaliana coronatine-insensitive (coi1) mutation occurs through two distinct mechanisms. The Plant Journal, 26, 509–522.

Kunkel, B.N. & Brooks, D.M. (2002) Cross talk between signaling pathways in pathogen defense. Current Opinion in Plant Biology, 4, 325–331.
Louis, J., Basu, S., Varsani, S., Castano-Duque, L., Jiang, V., Williams, W.P. et al. (2015) Ethylene contributes to maize insect resistance-mediated maize defense against the phloem sap-sucking corn leaf aphid. *Plant Physiology*, 169, 313–324.

Louis, J., Lorenc-Kukula, K., Singh, V., Reese, J., Jander, G. & Shah, J. (2010) Antibiosis against the green peach aphid requires the *Arabidopsis thaliana* MYTZUS PERSICAE-INDUCED LIPASE1 gene. *The Plant Journal*, 64, 800–811.

Ma, L., Sun, Y., Ruan, X., Huang, P.-C., Wang, S., Li, S. et al. (2021) Genome-wide characterization of jasmonates signaling components reveals the essential role of ZmCOI1a-ZmA215 action module in regulating maize immunity to Gibberella stalk rot. *International Journal of Molecular Sciences*, 22, 870.

Makandar, R., Essig, J.S., Schapauge, M.A., Trick, H.N. & Shah, J. (2006) Genetically engineered resistance to Fusarium head blight in wheat by expression of Arabidopsis NPR1. *Molecular Plant-Microbe Interactions*, 19, 123–129.

Makandar, R., Nalam, V., Chaturvedi, R., Jeannotte, R., Sparks, A.A. & Shah, J. (2010) Involvement of salicylate and jasmonate signaling pathways in *Arabidopsis* interaction with *Fusarium graminearum*. *Molecular Plant-Microbe Interactions*, 23, 861–870.

Makandar, R., Nalam, V.J., Chowdhury, Z., Sarowar, S., Klossner, G., Lee, H. et al. (2015) The combined action of ENHANCED DISEASE SUSCEPTIBILITY1, PHYTOALEXIN DEFICIENT4 and SENESCENCE-ASSOCIATED101 promotes salicylic acid-mediated defenses to limit *Fusarium graminearum* infection in *Arabidopsis thaliana*. *Molecular Plant-Microbe Interactions*, 28, 943–953.

Makandar, R., Nalam, V.J., Lee, H., Trick, H.N. & Shah, J. (2012) Salicylic acid regulates basal resistance to Fusarium head blight in wheat. *Molecular Plant-Microbe Interactions*, 25, 431–439.

Miersch, O., Bohlmann, H. & Wasternack, C. (1999) Jasmonates and related compounds from *Fusarium oxysporum*. *Phytochemistry*, 50, 517–523.

Mondal, H.A., Louis, J., Archer, L., Patel, M., Nalam, V.J., Sarowar, S. et al. (2018) *Arabidopsis ACTIN-DEPOLYMERIZING FACTOR3* is required for controlling aphid feeding from the phloem. *Plant Physiology*, 176, 879–890.

Nalam, V.J., Alam, S., Keeretaweep, J., Venables, B., Burdan, D., Lee, H. et al. (2015) Facilitation of *Fusarium graminearum* infection by 9-lipoxygenases in Arabidopsis and wheat. *Molecular Plant-Microbe Interactions*, 28, 1142–1152.

Nalam V.J., Sarowar S. & Shah J. (2016) Establishment of a *Fusarium graminearum* infection model in *Arabidopsis thaliana* leaves and floral tissues. *BioProtocol*, 6. https://doi.org/10.21769/bioprotoc.1877

Nandi, A., Kachrro, P., Fukushige, H., Hildebrand, D.F., Klessig, D.F. & Shah, J. (2003) Ethylene and jasmonic acid signaling pathways affect NPR1-independent expression of defense genes without impacting resistance to *Pseudomonas syringae* and *Peronospora parasitica* in the *Arabidopsis* ssi1 mutant. *Molecular Plant-Microbe Interactions*, 16, 588–599.

Nover, M.C., Erb-Downward, J. & Huffnagle, G.B. (2003) Production of eicosanoids and other oxylipins by pathogenic eukaryotic microbes. *Clinical Microbiology Reviews*, 16, 517–533.

Oliw, E.H. & Hamberg, M. (2017) An allene oxide and 12-oxophytodienoic acid are key intermediates in jasmonic acid biosynthesis by *Fusarium oxysporum*. *Journal of Lipid Research*, 58, 1670–1680.

Oono, Y., Chen, Q.G., Overvoorde, P.J., Köhler, C. & Theologis, A. (1998) Age mutants of Arabidopsis exhibit altered auxin-regulated gene expression. *The Plant Cell*, 10, 1649–1662.

Pan, X., Welti, R. & Wang, X. (2010) Quantitative analysis of major plant hormones in crude plant extracts by high-performance liquid chromatography-mass spectrometry. *Nature Protocols*, 5, 986–992.

Qi, P.F., Johnston, A., Balcerzak, M., Rocheleau, H., Harris, L.J., Wei, Y.M. et al. (2012) Effect of salicylic acid on *Fusarium graminearum*, the major causal agent of fusarium head blight in wheat. *Fungal Biology*, 115, 413–426.

Rivas-San, V.M. & Plascencia, J. (2011) Salicylic acid beyond defence: its role in plant growth and development. *Journal of Experimental Botany*, 62, 3321–3338.

Shah, J. (2003) The salicylic acid loop in plant defense. *Current Opinion in Plant Biology*, 6, 365–371.

Shiva, S., Samarakoon, T., Lowe, K., Roach, C., Vu, H.S., Colter, M. et al. (2020) Leaf lipid alterations in response to heat stress of *Arabidopsis thaliana*. *Plants*, 9, 845.

Steiner, B., Kurz, H., Lemmens, M. & Buerstmayr, H. (2009) Differential gene expression of related wheat lines with contrasting levels of head blight resistance after *Fusarium graminearum* inoculation. *Theoretical and Applied Genetics*, 118, 753–764.

Thaler, J.S., Humphrey, P.T. & Whitman, N.K. (2012) Evolution of monate and salicylate signal crosstalk. *Trends in Plant Science*, 17, 260–270.

Ton, J. & Mauch-Mani, B. (2004) Beta-amino-butyric acid-induced resistance against necrotrophic pathogens is based on ABA-dependent priming for calllose. *The Plant Journal*, 38, 119–130.

Tsitsigiannis, D.I. & Keller, N.P. (2007) Oxylipins as developmental and host-fungal communication signals. *Trends in Microbiology*, 15, 109–118.

Tskadu, K., Takahashi, K. & Nabetta, K. (2010) Biosynthesis of jasmonic acid in a plant pathogenic fungus, *Lasiodiplodia theobromae*. *Phytochemistry*, 71, 2019–2023.

Urban, M., Daniels, S., Mott, E. & Hammond-Kosack, K. (2002) *Arabidopsis* is susceptible to the cereal ear blight fungal pathogens *Fusarium graminearum* and *Fusarium culmorum*. *The Plant Journal*, 32, 961–973.

Wilson, W.W., McKee, G., Nganje, W., Dahl, B. & Bangsund, D. (2017) Economic impact of USWB5’s Scab Initiative to reduce FHB. *Agribusiness and Applied Economics Report*, 774. https://doi.org/10.22004/ag.econ.264672

Winkler, F.K., D’Arcy, A. & Hunziker, W. (1990) Structure of human pancreatic lipase. *Nature*, 343, 771–774.

Xu, X. & Nicholson, P. (2009) Community ecology of fungal pathogens causing wheat head blight. *Annual Review of Phytopathology*, 47, 83–103.

Yan, C. & Xie, D. (2015) Jasmonate in plant defence: sentinel or double agent? *Plant Biotechnology Journal*, 13, 1233–1240.

Zhang, L., Zhang, F., Melotto, M., Yao, J. & He, S.Y. (2017) Jasmonate related compounds from *Fusarium graminearum* and *Fusarium culmorum* infection in *Arabidopsis thaliana*. *Theoretical and Applied Genetics*, 109–118.

**SUPPORTING INFORMATION**

Additional supporting information may be found in the online version of the article at the publisher’s website.

How to cite this article: Alam, S.T., Sarowar, S., Mondal, H.A., Makandar, R., Chowdhury, Z., Louis, J. et al (2022) Opposing effects of *MYTZUS PERSICAE*-INDUCED LIPASE 1 and jasmonic acid influence the outcome of *Arabidopsis thaliana–Fusarium graminearum* interaction. *Molecular Plant Pathology*, 23, 1141–1153. https://doi.org/10.1111/mpp.13216