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SHORT COMMUNICATION

Trichoderma virens colonization of maize roots triggers rapid accumulation of 12-oxophytodienoate and two γ-ketols in leaves as priming agents of induced systemic resistance

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ABSTRACT

Two oxylipins 12-OPDA (12-Oxo-10(*Z*),15(*Z*)-phytodienoic acid) and an γ-ketol, 9,10-KODA (10-oxo-9-hydroxy-12(*Z*), 15(*Z*)-octadecadienoic acid) were recently identified as important long-distance-induced systemic resistance (ISR) signals in *Trichoderma virens*-treated maize. On the other hand, jasmonic acid (JA), long believed to be a major signal of ISR, was not involved, as the JA-deficient mutant, *opr7 opr8*, retained the capacity for *T. virens*-triggered ISR. In order to further understand the biochemical basis for ISR priming in maize leaves, diverse oxylipins and phytohormones in the leaves of wild-type maize or ISR-deficient *lox10-3* mutants treated with *T. virens* were quantified. This analysis revealed that 12-OPDA and two novel γ-ketols, 9,12-KOMA (12-Oxo -9-hydroxy-10(*E*)-octadecenoic acid) and 9,12-KODA (12-Oxo-9-hydroxy-10(*E*),15(*Z*)-octadecadienoic acid), accumulated at high levels in ISR-positive plants. In support of the notion that 12-OPDA serves as a priming agent for ISR in addition to being a xylem-mobile signal, leaf pretreatment with this JA precursor resulted in increased resistance to *Colletotrichum graminicola*. Furthermore, the injection of 9,12-KODA or 9,12-KOMA in wild-type plants enhanced resistance against *C. graminicola* infection, suggesting that they play roles in ISR priming.

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Aside from promoting plant growth and development, the colonization of plant roots by beneficial microorganisms triggers induced systemic resistance (ISR) to pathogen infection in aboveground tissues.[1–3] ISR involves the movement of long-distance, root-derived signals that travel systemically through the vasculature to prime defenses against pathogen attack.[1,4,5] In general, ISR signaling has been postulated to involve jasmonic acid (JA) and ethylene (ET) in a salicylic acid (SA)-independent manner.[6] The beneficial fungus, *T. virens*, induces systemic resistance in several host plants.[7,8] In *T. virens* interactions with maize (*Zea mays*), the small-secreted proteins Sm1 (protein ID 110852) and Sir1 (protein ID 77560) have been identified as positive and negative regulators of ISR, respectively.[9–11] Specifically, the knockout mutant *Δsm1* is unable to trigger ISR in cotton or maize,[9,10] while *Δsir1* enhanced resistance in maize against the pathogens, *Cochliobolus heterostrophus* and *Colletotrichum graminicola*.[11,12] Previously, we showed that Sm1 suppressed expression of a 9-lipoxygenase (LOX) gene, *LOX3*, in maize roots that acts as a negative regulator of ISR.[13] Maize *lox3-4* mutants over-accumulated the defense hormones SA, JA, and ET in the roots and displayed strong constitutive resistance against several pathogens.[14] The subsequent search for a positive ISR regulator in *lox3-4* mutant resulted in the identification of a 13-LOX gene, *LOX10*,[12] that is responsible for the biosynthesis of green leaf volatiles and an array of wound-induced oxylipins, including jasmonates and 12-OPDA (12-Oxo-10(*Z*),15(*Z*)-phytodienoic acid), a precursor of JA biosynthesis.[15,16] *LOX10* is overexpressed in *lox3-4* roots[14] and acts as a positive regulator of ISR, as *lox10-3* mutants displayed induced systemic susceptibility (ISS) when colonized by *T. virens*.[12] By metabolite screening of xylem sap from *T. virens*-treated wild-type, B73 inbred line, and near-isogenic *lox3-4* and *lox10-3* mutants, we identified 12-OPDA and 9-hydroxy-10-oxo-12(*Z*),15(*Z*)-octadecadienoic (9,10-KODA, formerly named as KODA), an α-ketol of linoleic acid, as major novel ISR long-distance signals.[12] Surprisingly, JA-deficient *opr7 opr8* mutants of maize displayed normal ISR, suggesting that JA is not a major long-distance ISR signal induced by *T. virens*. This current study extends the previously published metabolite profiling of xylem sap and roots with metabolite profiling of early leaf responses of maize to *T. virens*. These analyses uncover new potential priming compounds responsible for ISR.

To better understand the biochemical basis for ISR priming in maize leaves, we quantified diverse metabolites in the leaves of B73 or *lox10-3* mutants treated with wild-type *T. virens* (TvWT). The plants were treated with ISR-positive or ISR-negative strains of *T. virens*, and shoot tissue was collected at 6 (fungal recognition) and 54 hours (advanced colonization) post-treatment for metabolite quantification.[12,17] The results are presented in Figure 1, where the samples and individual metabolites (full names listed in Table 1) are grouped based on genotype/treatment and similarity in accumulation patterns (dendrogram not shown), respectively. The metabolites were separated into three groups. The most striking observation was that *lox10-3* plants had a lower concentration of almost all the measured metabolites compared to B73, regardless of *T. virens* treatments, with few exceptions. Two of these exceptions were

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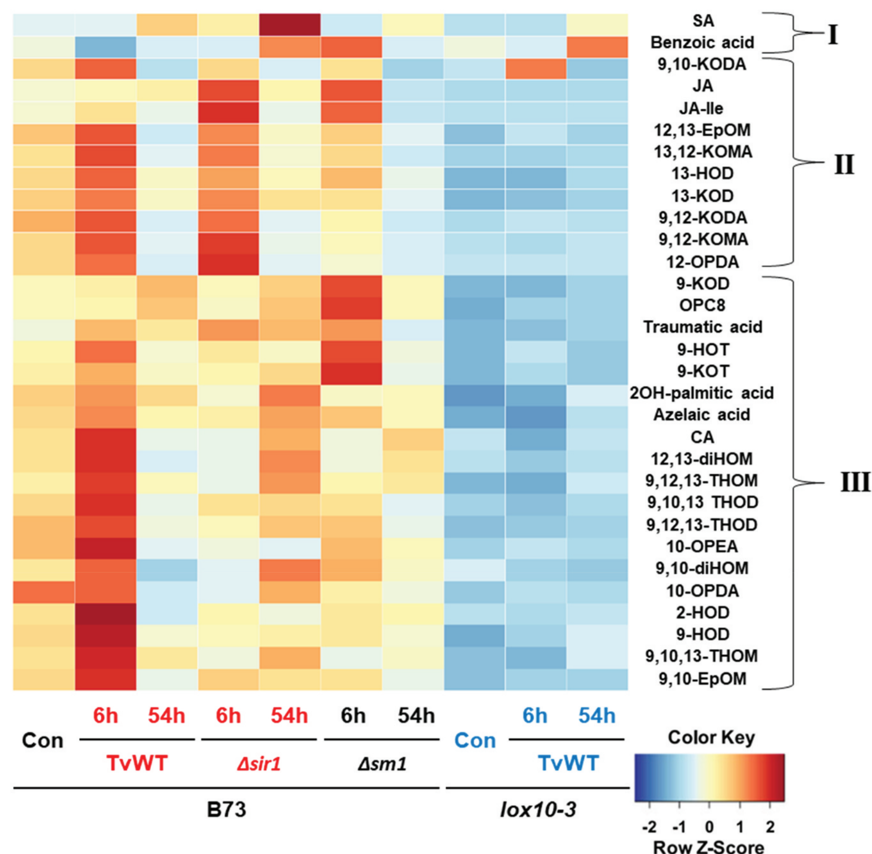


Figure 1. Metabolite and phytohormone levels in the shoot tissue of B73 (control or treated with TvWT, *Δsm1*, or *Δsir1*) and *lox10-3* (control or treated with TvWT) are displayed as a heatmap with Z-score transformed concentrations. Controls (Con) are plants that were not treated by *T. virens*. The treatments were separated by maize genotype, *T. virens* strain, and time, while the metabolites were separated into Groups I, II, and III based on similarity in the accumulation patterns (dendrogram not shown). Treatment designations in black font indicate lack of ISR, while red font indicates ISR-positive genotype combinations and blue font indicates ISS-positive genotype combinations.

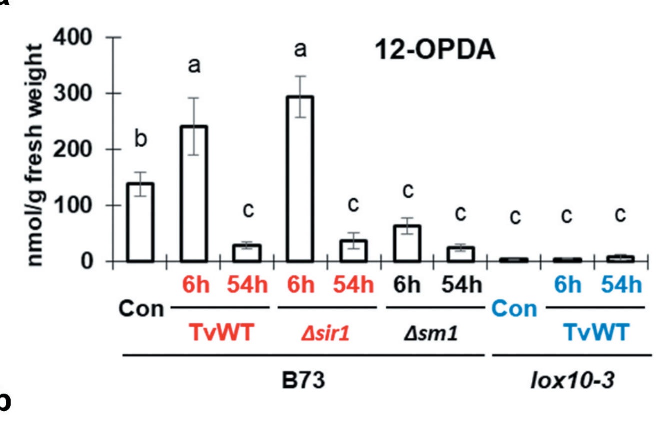
Table 1. Common and formal names of oxylipins and phytohormones measured in this study.

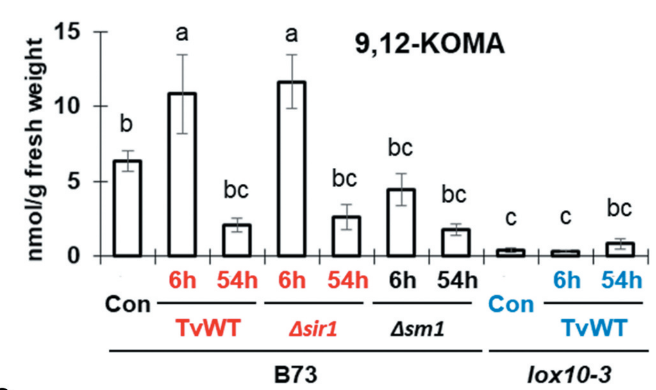
Class	Pathway	Lipid	Compound	Full Name
9-LOX	REDUCTASE/PEROXYGENASE	18:2	9-HOD	9(*S*)-Hydroxy-10(*E*),12(*Z*)-octadecadienoic acid
9-LOX	REDUCTASE/PEROXYGENASE	18:3	9-HOT	9(*S*)-Hydroxy-10(*E*),12(*Z*),15(*Z*)-octadecatrienoic acid
9-LOX	PEROXYGENASE	18:2	9,10-EpOM	9(*R*),10(*S*)-Epoxy-12(*Z*)-octadecenoic acid
9-LOX	PEROXYGENASE	18:2	9,10-diHOM	9(*R*),10(*S*)-Dihydroxy-9(*Z*)-octadecenoic acid
9-LOX	LIPOXYGENASE	18:2	9-KOD	9-Oxo-10(*E*),12(*Z*)-octadecadienoic acid
9-LOX	LIPOXYGENASE	18:3	9-KOT	9-Oxo-10(*E*),12(*Z*),15(*Z*)-octadecatrienoic acid
9-LOX	ALLENE OXIDE SYNTHASE	18:3	9,10-KODA	10-Oxo-9-hydroxy-12(*Z*), 15(*Z*)-octadecadienoic acid
9-LOX	ALLENE OXIDE SYNTHASE	18:3	10-OPDA	10-Oxo-11(*Z*),15(*Z*)-phytodienoic acid
9-LOX	ALLENE OXIDE SYNTHASE	18:2	10-OPEA	10-Oxo-11(*Z*)-phytoenoic acid
9-LOX	EPOXY ALCOHOL SYNTHASE	18:2	9,12,13-THOM	9(*S*),12(*S*),13(*S*)-Trihydroxy-10(*E*)-octadecenoic acid
9-LOX	EPOXY ALCOHOL SYNTHASE	18:3	9,12,13-THOD	9(*S*),12(*S*),13(*S*)-Trihydroxy-10(*E*),15(*Z*)-octadecadienoic acid
9-LOX	EPOXY ALCOHOL SYNTHASE	18:2	9,10,13-THOM	9(*S*),10(*S*),13(*S*)-Trihydroxy-11(*E*)-octadecenoic acid
9-LOX	EPOXY ALCOHOL SYNTHASE	18:3	9,10,13-THOD	9(*S*),10(*S*),13(*S*)-Trihydroxy-11(*E*),15(*Z*)-octadecadienoic acid
9-LOX	HYDROPEROXIDE LYASE	18:2	Azelaic acid	1,9-Nonanedioic acid
13-LOX	REDUCTASE/PEROXYGENASE	18:2	13-HOD	13(*S*)-Hydroxy-9(*Z*),11(*E*)-octadecadienoic acid
13-LOX	PEROXYGENASE	18:2	12,13-EpOM	12(*R*),13(*S*)-Epoxy-9(*Z*)-octadecenoic acid
13-LOX	PEROXYGENASE	18:2	12,13-diHOM	12,13-Dihydroxy-9(*Z*)-octadecenoic acid
13-LOX	LIPOXYGENASE	18:2	13-KOD	13-Oxo-9(*Z*),11(*E*)-octadecadienoic acid
13-LOX	ALLENE OXIDE SYNTHASE	18:2	9,12-KOMA	12-Oxo-9-hydroxy-10(*E*)-octadecenoic acid
13-LOX	ALLENE OXIDE SYNTHASE	18:3	9,12-KODA	12-Oxo-9-hydroxy-10(*E*),15(*Z*)-octadecadienoic acid
13-LOX	ALLENE OXIDE SYNTHASE	18:2	12,13-KOMA	12-Oxo-13-hydroxy-9(*Z*)-octadecenoic acid
13-LOX	ALLENE OXIDE SYNTHASE	18:3	12-OPDA	12-Oxo-10(*Z*),15(*Z*)-phytodienoic acid
13-LOX	ALLENE OXIDE SYNTHASE	18:3	OPC-8	3-Oxo-2-(2-Pentenyl)-Cyclopentane-1-Octanoic Acid
13-LOX	ALLENE OXIDE SYNTHASE	18:3	JA	7-Iso-Jasmonic acid
13-LOX	ALLENE OXIDE SYNTHASE	18:3	JA-Ile	7-Iso-Jasmonoyl-L-isoleucine
13/ 9-LOX	HYDROPEROXIDE LYASE	18:2/ 3	Traumatic acid	2(*E*)-Dodecenedioic acid
Other	α-DIOXYGENASE	16:3	2OH-palmitic acid	2-Hydroxy-hexadecanoic acid
Other	α-DIOXYGENASE	18:2/ 3	2-HOD	2(*R*)-Hydroxy-9(*Z*),12(*Z*)-octadecadienoic acid
Other	PHENYLALANINE AMMONIA LYASE	NA	CA	Cinnamic acid
Other	PHENYLALANINE AMMONIA LYASE	NA	BA	Benzoic acid
Other	PHENYLALANINE AMMONIA LYASE/ISOCHORISMATE SYNTHASE	NA	SA	Salicylic acid

SA and benzoic acid, a precursor of SA,[18,19] which was moderately induced in TvWT-treated *lox10-3* at 54 h. This is not surprising, as *lox10-3* has been demonstrated to accumulate elevated levels of SA when infected by *C. graminicola*[20] and is deficient in many 13-oxylipins compared to B73.[16] While SA is the major hormone for defense against this hemibiotrophic pathogen,[20] the induction of SA in *lox10-3* mutants was not sufficient to induce ISR, suggesting a combination with other defense metabolites, including those in Group III, may be necessary. Interestingly, *Δsir1*-treated B73 at 54 h accumulated the highest levels of SA, providing one mechanistic explanation for the superior ISR triggered by this strain. Surprisingly, 9,10-KODA (formerly designated as KODA)[12] also showed increased accumulation in TvWT-treated *lox10-3* at 6 h. Overall, these results did not provide any additional clues that explain ISS observed in TvWT-treated *lox10-3*. Interestingly, TvWT-treated B73 at 6 h accumulated higher amounts of metabolites in Groups II and III compared to untreated B73, though those subside to near control levels at 54 h. Most of the compounds in Group III that were upregulated in TvWT-treated B73 were not induced in *Δsm1*- or *Δsir1*-treated B73, suggesting that these compounds, while dependent on both functional Sm1 and Sir1 peptides, may not be necessary for the establishment of ISR. Elevated levels of several 9-LOX and α-dioxygenase (*a*-DOX) products in TvWT-treated B73 at 6 h suggest that these oxylipins may play defensive roles against *C. graminicola*, as was previously demonstrated with other pathogens.[21,22]

Of special relevance are several compounds in Group II that accumulated to the highest levels only in TvWT- or *Δsir1*-treated B73, suggesting that these compounds may be involved in ISR priming. This group of metabolites included 12-OPDA (Figure 2a), which was high only at 6 h, consistent with our previous findings.[12] Two other interesting compounds identified in Group II are the two poorly characterized $_{\gamma}$-ketols, 9,12-KODA and 9,12-KOMA (Figure 2b and c). These oxylipins are formed by hydrolysis of epoxides produced by reactions of 13-LOX and allene oxide synthase (AOS) on linoleic and linolenic acids, respectively (Figure 3). This is similar to the synthesis of the xylem-mobile ISR signal 9,10-KODA,[12] except that this α-ketol is synthesized by 9-LOX and 9-AOS branch of the LOX pathway.

The early and strong induction of 12-OPDA, 9,12-KODA, and 9,12-KOMA in leaves prompted a hypothesis that these (and other metabolites in group II) are ISR priming agents. We tested this hypothesis by foliar application of 20 µM 12-OPDA, which significantly reduced lesion development on the leaves of W438 maize inbred line, while lesions of near-isogenic *lox10-3* in W438 background remained unchanged (Figure 4a and b). Interestingly, this increased resistance is opposite to that of exogenous application of JA, which increased susceptibility to *C. graminicola*.[20] These findings further support the role of 12-OPDA as a major long-distance signal and potent priming agent that is integral to *T. virens*-triggered ISR. The early induction of 9,12-KODA and 9,12-KOMA suggested roles in ISR priming of shoots. To test the relevance of these two α-ketols, we injected B73 plants with 10 µL of 10 nM or 100 nM concentrations of 9,12-KODA or 9,12-KOMA before infecting with *C. graminicola*. The plants pretreated with 10 nM of either compound exhibited no change in resistance against infection (Figure 4c). On the other hand,





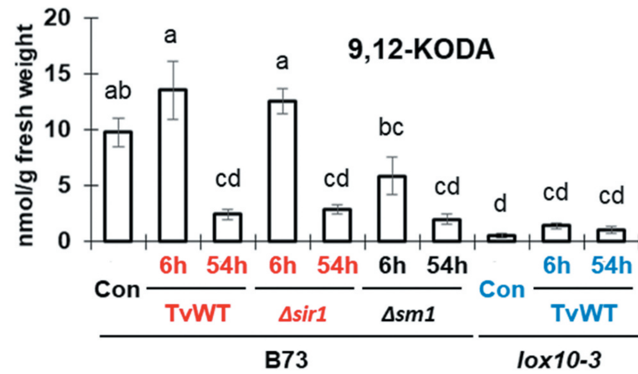


Figure 2. Accumulation of 12-OPDA, 9,12-KOMA, and 9,12-KODA in shoot tissues of B73 and *lox10-3* treated with *T. virens*. Bar graphs for (a) 12-OPDA, (b) 9,12-KOMA, and (c) 9,12-KODA, represent means, with error bars representing standard error. Letters indicate significant differences among all treatments (Tukey's HSD test, $p < .05$). ISR-negative genotype combinations of *T. virens*-treated maize are represented in black font, while ISR-positive genotype combinations are in red font and ISS-positive combinations are in blue font.

treatment with 100 nM 9,12-KODA or 9,12-KOMA significantly increased resistance, suggesting that they act as important signals in *T. virens*-triggered ISR in maize and shed new light on the functional relevance of these oxylipins toward defense against pathogen infection. As other compounds in Group II, such as 12,13-EpOM, 12,13-KOMA, 13-HOD, and 13-KOD, also accumulated highly in ISR-positive plants, we plan to expand our testing of these molecules to determine if they may play any roles in regulating ISR. Two other compounds in Group II, JA and JA-Ile, were not induced as strongly by Tv-WT as other metabolites. Importantly, our previous studies provided strong genetic and pharmacological evidence that these compounds

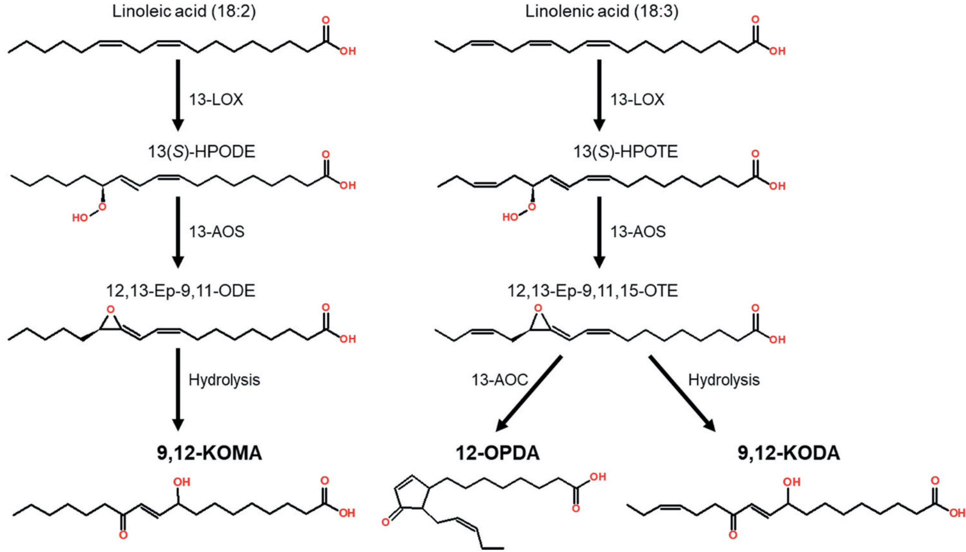


Figure 3. The biosynthesis pathways and chemical structures of 9,12-KOMA, 12-OPDA, and 9,12-KODA.

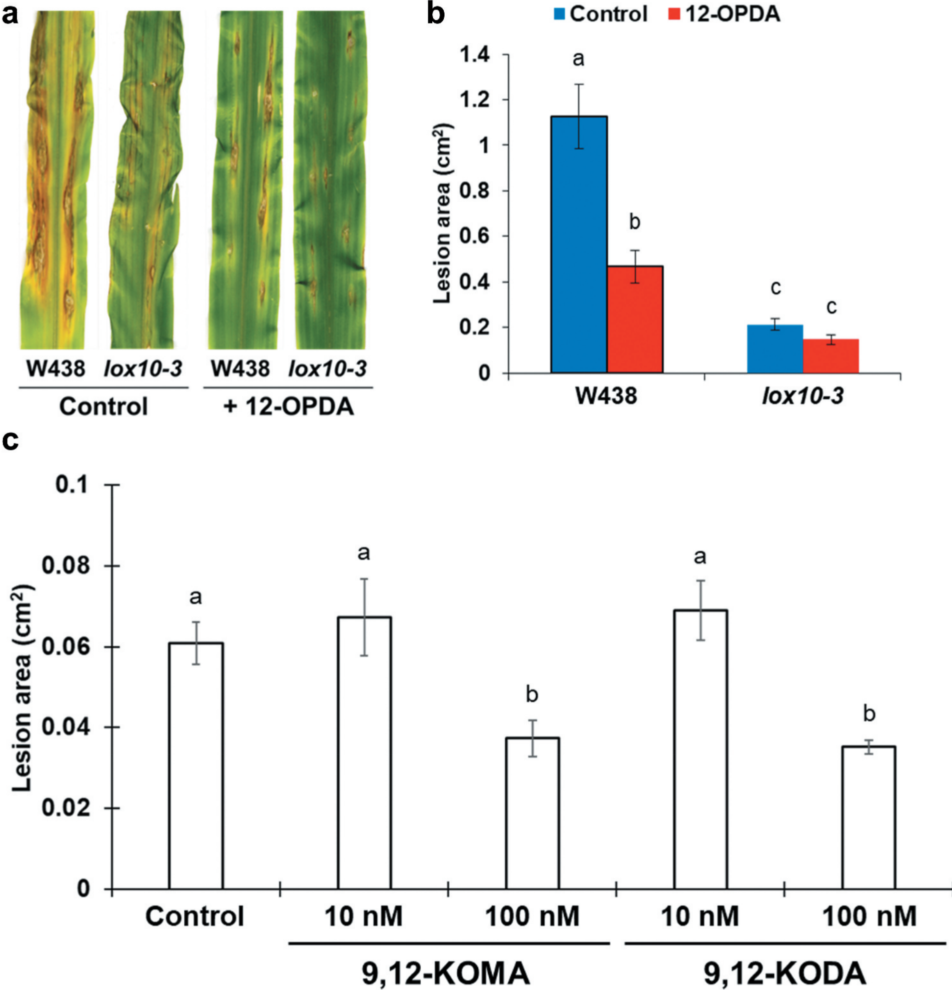


Figure 4. Exogenous application of 12-OPDA, 9,12-KOMA, and 9,12-KODA increases maize resistance to *C. graminicola*. (a) Lesions caused by *C. graminicola* infection of leaves of untreated by *T. virens* W438 inbred line and *lox10-3* mutants in W438 background sprayed with 1 mL of 20 µM 12-OPDA (dissolved in ethanol) or ethanol (control). Spores of *C. graminicola* (approximately 1×10^6 spores/mL) were drop inoculated on leaves 6 h after spray treatment, and leaves were scanned 5 d after infection. (b) Bar graph represents average lesion area of W438 inbred maize and *lox10-3* mutants in the W438 background sprayed with 20 µM 12-OPDA (dissolved in ethanol) or ethanol (control) and infected with *C. graminicola*. Error bars represent standard error. Letters indicate significant differences among all treatments (Tukey's HSD test, $p < .05$). (c) Bar graph represents average lesion area of B73 plants transfused with sap supplemented with 100 nM or 10 nM 9,12-KODA or 9,12-KOMA and infected by *C. graminicola*, with error bars representing standard error. Letters indicate significant differences among all treatments (Tukey's HSD test, $p < .05$).

promote virulence of *C. graminicola*,[12,20] and therefore, cannot be considered as priming agents of ISR.

In summary, this study was carried out to better understand the phytohormones and metabolites associated with regulating *T. virens*-triggered ISR in maize. Through metabolite screening, we determined that *lox10-3* responded to *T. virens* very differently than B73, with a notable lack of accumulation of most detectable oxylipins. We showed the relevance of 12-OPDA as an inducer of resistance in maize leaves against *C. graminicola* infection. Additionally, we further demonstrated that treatment with 9,12-KODA and 9,12-KOMA enhanced maize resistance against pathogen infection, suggesting previously unknown roles for both oxylipins as potential ISR priming agents.

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Disclosure of potential conflicts of interest

No potential conflicts of interest were disclosed.

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