Potential roles for pattern molecule of PAMP-triggered immunity in improving crop cold tolerance

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Received: 4 September 2021 / Accepted: 4 November 2021 / Published online: 24 November 2021
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Abstract
Key message The application of flagellin 22 (flg22), the most widely studied PAMP, enhance crop cold tolerance.
ICE1-CBF pathway and SA signaling is involved in the alleviation of cold injury by flg22 treatment.

Abstract Pathogen infection cross-activates cold response and increase cold tolerance of host plants. However, it is not possible to use the infection to increase cold tolerance of field plants. Here flagellin 22 (flg22), the most widely studied PAMP (pathogen-associated molecular patterns), was used to mimic the pathogen infection to cross-activate cold response. Flg22 treatment alleviated the injury caused by freezing in Arabidopsis, oilseed and tobacco. In Arabidopsis, flg22 activated the expression of immunity and cold-related genes. Moreover, the flg22 induced alleviation of cold injury was lost in NahG transgenic line (SA-deficient), sid2-2 and npr1-1 mutant plants, and flg22-induced expression of cold tolerance-related genes, which indicating that salicylic acid signaling pathway is required for the alleviation of cold injury by flg22 treatment. In short flg22 application can be used to enhance cold tolerance in field via a salicylic acid-depended pathway.

Keywords Freezing tolerance · Flagellin 22 · Cross-talk · Salicylic acid

Introduction

Plants have worldly monitoring systems to adapt a variety of environmental disturbances including biotic and abiotic stress, such as drought, heat, cold, salinity, pathogen infection, insect feeding (Suzuki et al. 2014). Upon perceiving stress, plants trigger a series of signal transduction leading to reprogramming of gene expression to establish a new balance between growth and defense response against the adverse environment to finish lifecycle and produce seeds (Zhu 2016).

Cold stress, including chilling stress (0–15 °C) and freezing stress (<0 °C), restricts plant growth and development, limits plant geographical location and reduces crop production (Shi et al. 2018). C-repeat binding factors (CBFs), conserved transcription factors in many plant species, are critical for cold acclimation (Jia et al. 2016). CBF genes are rapidly induced by cold stress, and then activate the expression of downstream cold-responsive gene (COR) to increase plant cold tolerance (Chinnusamy et al. 2007). CBFs are regulated by various transcription factors, which recognize the cis-elements in CBFs’ simultaneously promoters under cold stress, including ICE1 (CBF expression1), CAMTA (calmodulin-binding transcription activator) proteins, MYB15, EIN3 (Ethylene insensitive 3) (Shi et al. 2018).

Under field conditions, cold stress affects the growth and development of plants simultaneously with other stress, such as pathogen infection. Cold stress cross activates disease resistance by activating the expression of PR1, PR2, and PR5 (Pathogenesis-Related 1, 2, and 5) through activating the function of transcription factor NAC062 (NAC for NAM, ATAF1,2, CUC2) (Seo et al. 2010), activating the pattern-triggered immunity in Arabidopsis plants in a HAT1 (HISTONE ACETYL TRANSFERASE 1)-dependent manner (Singh et al. 2014) and releasing CAMTA (calmodulin-binding transcription activator) transcription factors mediated repression of salicylic acid biosynthesis (Kim et al. 2017). Our previous research showed that cold stress cross-activates basal immunity against the bacterial...
pathogen \textit{Pseudomonas syringae pv tomato} DC3000 (\textit{Pst DC3000}) though activating SA pathway and inhibiting JA pathway (Wu et al. 2019). Interestingly this pathogen infection also cross-activates cold response and thus increase cold tolerance of host plants (Tuang et al. 2020). These investigations provide an idea to exploit immunity to increase plants abiotic stress tolerance.

Plants have a multilayered system to defense against pathogens, plant cells are able to recognize conserved epitopes of pathogen, which are called pathogen-associated molecular patterns (PAMPs), such as bacterial flagellin (Wyrsch et al. 2015). Plasma membrane-localized pattern recognition receptors (PRRs) activates PAMP-triggered immunity (PTI), a mild and wide-spectrum disease resistance against various pathogens (Bigeard et al. 2015). Flagellin-22 (Flg22), the most widely-studied PAMP, has a conserved 22-amino acid domain shared by the bacterial flagellum. Flg22 is recognized by FLAGELLIN-SENSITIVE 2 (FLS2) and activates a mitogen-activated protein kinase (MAPK) cascade that regulates the activation of defense-related genes (Zipfel et al. 2004), including salicylic acid (SA) pathway maker genes such as \textit{PR1} (\textit{Pathogenesis-Related 1}), \textit{PAD4} (\textit{PHYTOALEXIN-DEFICIENT4}), \textit{EDS1} (\textit{ENHANCED DISEASE SUSCEPTIBILITY1}), \textit{NPR1} (\textit{non-expresser of pathogenesis-related [PR] 1}) (Denoux et al. 2009), callose deposition (Muthamilarasan and Prasad 2013), and production of reactive oxygen species (ROS) (Bigeard et al. 2015).

In this work, it is investigated whether flg22, a well-studied PAMP is able to cross-activate cold response and thus increase the cold tolerance of plants and our data indicated that the PAMP is a potential activator useful for the improvement of the cold tolerance of crops.

\section*{Results}

\textbf{Flg22 treatment alleviated the injury caused by freezing in Arabidopsis}

In previous work, it was shown that bacterial pathogen \textit{Pst DC3000} infection cross-activates the cold response and thus increased cellular viability under freezing (Tuang et al. 2020). As the pathogen infection leads to serious disease to plants, it is not possible to use the infection to increase freezing tolerance of field plants. Thus we investigated whether flg22, (a pathogen-associated molecular patterns PAMPs), a maker peptide for PTI in freezing stress response, derived from bacterial flagellin proteins, could mimic the pathogen infection to cross-activate cold response to some extent.

Five days old Arabidopsis seedlings were treated with flg22, and 2 days later the infiltrated (2 dpi) seedlings were stressed under $-4 \, ^\circ \text{C}$ for 1 h, 2 h, and 2.5 h, respectively. Then extracted the chlorophyll and determine the contents using UV-Spectrometer. As shown in Fig. 1, both chlorophyll ‘a’ and ‘b’ of flg22 treated seedlings showed lower chlorophyll contents compared to control seedlings under control condition ($22 \, ^\circ \text{C}$). However, at 1 h freezing stress, chlorophyll ‘a’ and ‘b’ in control and flg22 treated seedlings showed no difference. At 2 h freezing stressed flg22 treated seedlings showed higher contents compared to those of control seedlings (Fig. 1). The results indicated that freezing stress does not much effect chlorophyll contents in a test temperature and time points, but prior flg22 treatment-induced chlorophyll contents upon freezing stress in Arabidopsis seedlings.

To further examine whether Flg22 treatment functions as pathogen infection under freezing stress. Arabidopsis plants were infiltrated by flg22, and 2 days later the infiltrated plants were stressed under freezing temperature ($-4 \, ^\circ \text{C}$) for 6 h or 8 h, and the cellular viability was evaluated by trypan blue staining and ion leakage measurement. As shown in Fig. 2, upon freezing, the CK (non-infiltrated), Mock (infiltrated by water), and flg22-infiltrated plants were significantly decreased in cellular viability and increased ion leakage level compared with their counterpart controls (non-freezing) (Fig. 2). However, after 8 h of freezing stress, flg22-infiltrated plants showed a significant higher cellular viability and lower ion leakage level than CK and mock plants (Supplement Figures 1, 2a, c). These results indicate that prior flg22 treatment could alleviate cellular injury caused by subsequent freezing stress.
Flg22 infiltration induced chlorophyll contents of Oilseed and Tobacco seedlings upon freezing

To know whether flg22 treatment affects freezing tolerance in crops as found in Arabidopsis, we also treated five days old Brassica napus (winter Oilseed Rape) seedlings by flg22, and 2 dpi seedlings were stressed under −4 °C. As shown in Fig. 3, chlorophyll ‘a’ of flg22 treated seedlings showed higher contents than those of control seedlings in 1 h, 2 h, and 2.5 h of freezing stressed (Fig. 3); and chlorophyll ‘b’ of flg22 treated seedlings showed higher contents than those of control seedlings in 2 h, and 2.5 h of freezing stressed (Fig. 3). This result suggests that prior flg22 treatment-induced chlorophyll contents upon freezing stress.

Then we checked the chlorophyll contents of Nicotiana tabacum (Tobacco) seedlings upon freezing stress. Similar to Oilseed, Tobacco seedlings also showed higher chlorophyll contents in flg22 treated seedlings (Fig. 4) compared...
to control seedlings upon freezing stress. Under normal conditions (22 °C), control and flg22 treated seedlings showed no significant contents to each other. However, chlorophyll contents of flg22 prior treated seedlings showed higher contents compared to control seedlings after freezing stress (Fig. 4). All chlorophyll contents were down to basal level at 2.5 h of freezing stress (Fig. 3, 2.5 h). The results suggest freezing stress adversely affects the chlorophyll contents of Tobacco, and flg22 treatment indeed induced chlorophyll contents upon freezing stress in tested conditions. All these above indicates that flg22 treatment enhanced freezing tolerance in oilseed and tobacco.

Flg22 treatment activated the expression of cold-related genes

As higher cellular viability was detected in flg22-treated plants stressed by freezing (Fig. 4a, b) and our previous work showed the pathogen infection activated the expression of cold-responsive genes in host plants (Tuang et al. 2020), it was asked whether flg22 treatment also cross-induces these genes expression. We checked the expressions of some cold stress-related genes. As expected, some key genes involved in cold tolerance were activated by flg22 at early (6 hpi) or/and late (48 hpi) stages. Interestingly, some of the tested genes were only upregulated at an early stage, including ICE1, CBF3, COR15A (Fig. 5a, d, e). CBF1, COR15B, COR27 and COR413M2 were upregulated at both early and late stages (Fig. 5b, f–h).

To find out the genetic mechanism on flg22 treatment increasing cold tolerance, we analyzed the expression of more genes which plays a positive function in cold tolerance. The expression of CHS1, ATERDJ3A, DREB2A, MBF1C (Fig. 6a, c, g, i) were induced at both early and late stages. The expression of AZII, MKK2, DREB2C, EALII, NAC019, NAC042 (Fig. 6b, f, g–k) were only upregulated at early stage. The expression of flg22-activated immunity genes such as EDS1, EDS5, EDS16 and PRI which were upregulated at both early and late stages (Supplement Figure 2). All these results indicate flg22 treatment, similar to pathogen injection, activates the expression of genes involved cold response pathway in host plants.

Flg22-activated cold tolerance is dependent on SA

Salicylic acid (SA) is required for the alleviation of cold injury by pst Dc3000, therefore we wondered whether the alleviation of cold injury by Flg22 was associated with SA accumulation. NahG transgenic plant, containing a salicylate hydroxylase gene NahG, was an SA deficient line (Tuang et al. 2020). As shown in Fig. 7a, c under freezing for 6 h flg22 treated NahG plants showed no difference in cell viability and ion leakage with control and mock-treated plants. After − 4 °C treatment for 8 h flg22 infected NahG plants were more severely damaged by freezing than CK and mock ones (Fig. 7a, c Supplement Figure 3a), which was in contrast with the results detected in wildtype. NahG transgenic plant, SA deficient line, lost the capacity of

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**Fig. 5** RT-qPCR to check the expression of several marker genes in cold tolerance upon 2 mg/L flg22 treatment at 6 hpi, and 48 hpi. TUB2 (AT5G62690) was used as an internal reference. The data were shown as the average ± SD of three biological repeats. The different lower letters, a–c, etc. in graphs indicate the significant difference between different samples ($p < 0.05, n = 3$).
alleviating freezing injury by flg22 treatment, indicating SA was required in the alleviating.

To confirm the function of SA in the alleviation of cold injury by flg22 treatment, the cold tolerance of flg22 treated sid2-2 and npr1-1 plants was checked. SID2 plays an important role in SA accumulation, pathogen-induced SA biosynthesis was significantly decreased in sid2-2 mutant plants (Dempsey et al. 2011). The SA induced by pathogen induce the expression of downstream genes by activate NPR1 (Pajerowska-Mukhtar et al. 2013). NPR1 is the regulator of SA signaling pathway. As shown in Fig. 7e–l and Supplement Figure 3b, c, flg22 treated sid2-2 and npr1-1 plants showed no difference in cell viability and ion leakage with control and mock-treated plants under freezing for 6 h. Flg22 treated sid2-2 plants were more severely damaged by freezing for 8 h than CK and mock ones. These above indicated the requirement of SID and NPR for the alleviation of freezing injury by flg22 treatment.

The effect of flg22 treatment on the expression of cold tolerance genes was lost or compromised in NahG, sid2-2, and npr1-1 plants

To check whether SA was required for the activation of genes which were related to the cold tolerance and induced by flg22 treatment, the expression of these genes were detected in flg22 treated NahG transgenic, sid2-2, npr1-1 plants. As shown in Fig. 8 the induction by flg22 infection of genes, including CBF1, CBF3, ATERDJ3A, NAC042, was lost in NahG transgenic plants. Only the expression of DREB2C and MBF1C was partially induced. In sid2-2 mutant plants the induction of CBF1, CBF3, DREB2C, MBF1C was completely lost (Fig. 8). In npr1-1 mutant plants the induction of CBF1, CBF3, DREB2C, NAC042 and MBF1C was completely lost (Fig. 8). All these above indicated that NPR1-mediated SA signaling pathway is required for the activation of these cold tolerance-related genes.

Discussion

Flagellin-22 treatment alleviates the injury caused by subsequent freezing stress

A great deal of evidences showed the crosstalk between abiotic stress and immune response, environmental factors are proved to module plant immunity (Saijo and Loo 2020). For example, UV–B or excessive light stress induced the synthetize of flavonoids (Schenke et al. 2019), which weaken SA-based immunity (Serrano et al. 2012); heat stress induced the expression of HSP, which enhanced plant immunity by the accumulation and stability of pathogen-related (PR) proteins (Ul Haq et al. 2019); cold stress-activated disease resistance through SA dependent pathway (Wu et al. 2019). Our earlier experiment showed that pst DC3000 infection enhanced the tolerance of freezing and heat injury in Arabidopsis (Tuang et al. 2020). However,
Fig. 7  cell viability analysis on 2-dpi 2 mg/L flg22 treated NahG transgenic (a–d), sid2-2 (e–h), and npr1-1 mutant (i–l) plants under freezing (−4 °C). 2-dpi mutant plants were treated by freezing (−4 °C) for the indicated time. a, e, i Cell viability assay by trypan blue staining. b, f, j Ion leakage assay. c, d, g, h, k, l Ratio of freezing treatment value to control in b, c, d, g, h, l, m, respectively. The quantified values in a, b, e, f, i, j are the average ± SD from three biological repeats, and different lowercase letters, a–c, showed the significant difference (p < 0.05).
these findings are not possible for use in the field because of lack of practicability or the damage to plants.

Here, flagellin-22 treatment not only alleviated freezing injury in Arabidopsis (Figs. 1, 2) but also in oilseed (Fig. 3) and tobacco (Fig. 4). Flg22, a conserved peptide with 22 amino acid, has been widely used to induce PTI responses and does not lead to serious disease to plant (Wyrsch et al. 2015). All these above suggested that flg22 application may be used to enhance cold tolerance in field. Phytohormones and their functional analog were also used to prime plants to mitigate the effects of abiotic and biotic stresses (Conrath et al. 2015). However, these molecules were often used in seed priming to increase the percentage and rate of germination and improve seedling growth (Rhaman et al. 2020). Flagellin-22 application can be used at any stage of plant life.

Flagellin-22 treatment activates signaling pathway of cold stress

When exposed to cold stress, plant rapidly induces the expression of many transcription factors, including the APETALLA2 (AP2)-domain proteins CBFs (CBF1, CBF2 and CBF3). The expression of CBF genes was regulated by ICE1, a bHLH transcription factor (Chinnusamy et al. 2007). In Arabidopsis, CBF2 is suggested as a negative regulator of CBF1 and CBF3 expression during cold response (Novillo et al. 2004). The cold-induced CBFs bind C-repeat dehydration-responsive elements in the promoters of downstream genes and activate the expression of downstream genes, such as numerous COR genes (Zhu 2016). Interestingly, in our work, the expression of ICE1 was induced by flg22 treatment at early stage (6 h after injection), the expression of CBF1 and CBF3 was up-regulated by flg22 and the expression of CBF2 was depressed by flg22 treatment at early and late stages (Fig. 5). Furthermore, a sum of COR genes were up-regulated by flg22 treatment, including COR15A, COR15B, COR27, COR143M2 (Fig. 5). These results suggested that the ICE1-CBF pathway is activated by flg22, thus increasing cold tolerance of host plants.

SA is required for alleviating the injury caused by cold stress

Salicylic acid (SA) has emerged as a key plant defense hormone for both pathogen-associated molecular patterns (PAMPs)-triggered immunity (PTI) and effector-triggered immunity (ETI) (Zhang and Li 2019). SA also plays an important role in the plant responses to different abiotic stresses (Rivas-San and Plasencia 2011). Cold was shown to induce SA biosynthesis (Kim et al. 2013, 2017), meanwhile exogenous SA treatment was proved to provide protection against cold stress (Miura and Furumoto 2013). Flg22 treatments caused SA accumulation through...
SID2-mediated SA biosynthesis (Tsuda et al. 2008). SID2, which encodes isochorismate synthase, is a SA biosynthetic enzyme (Wildermuth et al. 2001).

Our current work showed Flg22 treatment-induced SA was required for the alleviation of subsequent cold injury, since both NahG line and sid2-2 mutant plants lost the ability to alleviating the injury (Fig. 7). Consistently, in these plants, Flg22 treatment-activated expression of tested cold stress genes was lost or compromised (Fig. 8). Moreover, the alleviation of cold injury by Flg22 was lost in npr1-1 mutant (Figs. 7 and 8), which suggest SA signaling pathway also play a vital role in flg22 enhanced cold tolerance. NPR1 protein not only plays an essential role in SA-mediated defense resistance, but also is involved in crosstalk inhibition of jasmonic acid (JA)-mediated defense responses (Dong 2004). Flg22-triggered oxidative burst was suppressed by the activation of JA signaling (Yi et al. 2014). The above indicated that JA signaling pathway may also involve in flg22 enhanced cold tolerance.

In summary, our work suggested that flg22 application may be used to enhance cold tolerance in field, indicated that flg22 treatment enhance of host plants to cold stress by activating cold signaling pathway ICE1-CBF. NPR1-mediated SA signaling is involved in the alleviation of cold injury by prior flg22 treatment.

Materials and methods

Plant materials and flg22 treatment

The Arabidopsis materials including ecotype Col-0, npr1-1 and sid2-1, NahG transgenic plants and growth conditions were described in Tuang et al. (2020). The 4-week-old plants with the 8th true leaf emerging were vacuum-infiltrated by 2 mg/L of Flg22 (Peptide Sequence: QRLSTGSRINSKDDAAGLQIA, Beijing XMJ Scientific Co., Ltd, China) at − 0.1 MPa for 1 min, followed by culturing under the normal condition for 2 days. Then, the plants were moved to − 4 °C for a different time. The cellular viability, Chlorophyll contents and ion leakage were measured upon each time-point. Freezing treatment was performed under a dark condition.

For oilseed and tobacco materials, seeds were sown on half MS basal agar plates, and cold acclimated at 4 °C for 48 h, then transferred to 22–23 °C for germination. 5-day-old seedlings on growing plates were sprayed by 2 mg/L flg22 solution, and sequentially cultured for 2 days. Then plates were moved to − 4 °C incubator for 1 h, 2 h, 2.5 h, and the chlorophyll contents were measured upon each time-point for evaluating the freezing damage.

Cellular viability assay, ion leakage measurement and Chlorophyll contents measurement

Electrical conductance and trypan blue staining were used to evaluate the cellular viability as described in Tuang et al. (2020). Ion leakage measurement was performed by following the method from Tuang et al. (2020). Chlorophyll extraction was performed by following the method from Karan and Subudhi (2012).

RNA isolation and real-time PCR

Total RNAs were extracted from Arabidopsis leaves with NucleoZol solution (Macherey–Nagel GmbH & Co. KG, Germany) following the manufacturer’s instructions. Prime Script™ RT Reagent Kit with gDNA Eraser (Takara Bio Inc. China) was used to remove DNA contamination and synthesize cDNA. All qPCR reactions were performed using the UNICON™SYBR® Green master mix (Yeasen Co. Ltd, China). TUB2 (At5g62690) was used as the internal control. The data were shown as the average ± SD from three biological replicates. Primers were described in Supplement Table 1.

Statistics

The statistics analysis was performed using the software GraphPad Prism (Version 7.00). Student’s t was used to test the significance of a single pair of sample differences. One-way ANOVA analysis of variance was used to test the significance of the differences between multiple pairs of samples, with Tukey’s multiple comparisons test for a post-hoc test for pairwise significant comparison. A difference at $p < 0.05$ was considered significant.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1007/s00299-021-02811-4.

Author contribution statement

WY conceived and designed research. ZT, YJ, YW and ZW conducted experiments. YJ wrote the manuscript. All authors read and approved the manuscript.

Funding

This work was financially supported by the National Natural Science Foundation of China (Grant numbers 31470365 and 31700216).

Data availability

All data generated or analysed during this study are included in this published article and its supplementary information files.
Declarations

Conflict of interest The authors declare that they have no conflict of interest.

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