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REVIEW
Salicylic Acid Signaling Pathway in Rice and the Potential Applications of Its Regulators

Hiroshi TAKATSUJI*, Chang-Jie JIANG and Shoji SUGANO
Plant Disease Resistance Research Unit, Division of Plant Sciences, National Institute of Agrobiological Sciences (Tsukuba, Ibaraki 305−8602, Japan)

Abstract
The salicylic acid (SA) signaling pathway plays a crucial role in systemic acquired resistance in dicots. Several chemical inducers, which are also called "plant activators," such as benzothiadiazole (BTH), protect plants from diseases by acting on the SA signaling pathway. Several studies by us and other groups have revealed that rice also has the SA signaling pathway that shares signaling components with the SA signaling pathway in dicots. OsNPR1 is a rice counterpart of NPR1, a transcriptional coactivator that plays a central role in the SA pathway in *Arabidopsis*. OsWRKY45 is a BTH-inducible rice-specific transcription factor. Knockdown experiments have demonstrated that both these transcriptional regulators are essential for BTH-induced blast resistance in rice. Unlike the SA pathway in *Arabidopsis*, wherein most of the SA/BTH-regulated genes are controlled by NPR1, the pathway in rice branches into OsNPR1- and OsWRKY45-dependent pathways. OsWRKY45 overexpression confers very high resistance to blast and leaf blight diseases in rice, and it causes only minor growth retardation through the "priming effect"-an action mechanism characteristic of plant activators. OsWRKY45 is a potential target for genetic manipulation of rice in order to confer broad-spectrum disease resistance; however, it is necessary to improve the construct to drive *OsWRKY45* expression in rice in order to optimize the growth and disease resistance of the plants. Here, we have reviewed recent advances in the studies on the SA pathway in rice, with particular focus on the potential practical applications of the signaling components.

Discipline: Plant disease
Additional key words: blast, leaf blight, OsNPR1, OsSSI2, OsWRKY45

Introduction

Rice is not only the most important cereal crop for human consumption worldwide, but has also recently emerged as a forage crop and a resource for biofuel. Rice diseases such as fungal blast and bacterial leaf blight occasionally cause devastating damage to rice crops. In Japan, blast disease causes the most severe damage annually. For decades, various approaches have been sought to prevent and treat rice diseases. Agricultural chemicals have greatly contributed to modern practices for rice cultivation; however, in addition to the economic costs, there has been a growing concern about the health and environmental risks associated with the use of chemicals. In recent decades, new type of agrochemicals, called "plant activators," which protect plants by activating their defense system, have been widely used because of their low health and environmental risks. Another approach for disease prevention is through the use of host defense genes. Disease resistance (R) genes that mediate 'gene-for-gene' resistance are effective and economical. However, incorporation of R genes into rice varieties has not achieved durable resistance to the rice blast fungus, *Magnaporthe oryzae*; this is because of the outbreak of fungal races that have evaded surveillance systems by R genes, which results in the breakdown of resistance after a few years of use of the resistant rice variety in fields. The possibility of using genes encoding pathogenesis-related (PR) proteins, such as chitinase and ß-1,3 glucanase, for disease prevention has also been explored[1]; however, in general, disease resistances conferred by these genes were not so

* Corresponding author: e-mail takatsuh@affrc.go.jp
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strong. In an attempt to develop a new approach for the molecular breeding of disease-resistant rice, we functionally characterized signaling components involved in the salicylic acid (SA)-mediated defense-signaling pathway; these components are also involved in the action mechanism of plant activators. OsWRKY45, a central transcription factor regulating the SA signaling pathway in rice, currently seems to be the most promising target for this approach.

SA signaling pathway in rice

In dicots, a defense-signaling pathway mediated by SA has been proposed to play a crucial role in systemic acquired resistance (SAR)-a natural defense system against attacks by microbial pathogens[20]. Endogenous SA levels rapidly elevate both locally and systemically upon pathogen attacks[16], leading to the activation of defense reactions. In contrast to dicots wherein the basal levels of SA are low, rice plants accumulate high levels of SA in the absence of pathogen infection, and these levels do not seem to be sensitive to infection[24]. This implies that the defense mechanism in rice plants is different from that in dicots. However, several studies have also demonstrated the presence of an SA-mediated defense-signaling pathway in rice. SA-deficient transgenic plants that overexpress the *nahG* gene, encoding a salicylate hydroxylase, exhibited reduced resistance to blast disease, accompanied by increased susceptibility to oxidative damage due to biotic and abiotic stresses[29]. Moreover, a correlation has been recognized between the SA content and blast resistance of various rice varieties[24].

Recent studies have revealed that rice shares several components of the SA signaling pathway with *Arabidopsis*. NPR1 is a central positive regulator of SAR in *Arabidopsis* that transmits the SA signal to downstream *PR* gene activation[2]. SA accumulation triggered by pathogen infection alters the cellular redox potential, resulting in the nuclear translocation of NPR1[17]. NPR1 then interacts with transcription factors (TFs) of the TGA family[33] and activates target defense genes. Overexpression of the *Arabidopsis NPR1* gene in rice enhanced the resistance of the latter to the bacterial pathogen, *Xanthomonas oryzae* pv. *oryzae*[4, 6] and the fungal pathogens, *M. oryzae* and *Fusarium verticillioides*[19]. Moreover, overexpression of OsNPR1/NH1, an NPR1 counterpart in rice[3, 32], enhances the resistance of rice to bacterial blight disease, while knockdown of this gene increases the susceptibility of rice plants to this disease[3]. These reports suggest that rice has a defense-signaling pathway similar to the SA/NPR1-mediated pathway in *Arabidopsis*.

Plant activators act on the SA signaling pathway

Plant activators, such as benzothiadiazole (BTH)[8, 15], probenazole[11, 31] and tiadinil[30] effectively protect many plant species from diseases. These chemicals do not have a direct effect on pathogens but enhance the defense responses of the plants by acting on the SA signaling pathway. While probenazole acts upstream of SA[10], BTH and tiadinil, which are functional analogues of SA, act downstream of SA (Fig. 1). Plant activators are recognized for their broad spectrum of resistance; in general, they are effective against a variety of biotrophic pathogens. The “priming” effect is a characteristic feature in the action mechanism of plant activators. In most cases, defense responses negatively affect plant growth and development due to allocation of limited resources to defensive compounds or toxicity of defensive products[9, 27]. When plant activators are administered to plants at high doses, they constitutively activate the defense responses, including upregulation of defense genes, a process designated “direct defense”[14, 27]. In contrast, when plant activators are administered at appropriately low doses, plants become “primed” for defense responses, i. e. , their defense response are effectively activated after pathogen infection[5].

The transcription factor OsWRKY45 plays an essential role in plant activator-induced disease resistance

In general, pathogen infection alters the transcription levels of numerous defense genes, as a consequence of the activation of the plant defense response. TFs play a crucial role in this process by controlling multiple genes that cumulatively drive defense reactions. The modulation of key TFs in the defense mechanism should lead to a wide range of expressional changes and thus considerably alter the defense-related phenotypes. Therefore, TFs are suitable targets for genetic manipulation of plants in order to improve disease resistance. To identify the key TFs involved in BTH-induced disease resistance, we characterized the expression of BTH-responsive genes in wild-type rice (*Oryza sativa* cv. Nipponbare) by using an Agilent Rice Oligo Microarray. We identified 326 BTH-responsive genes expressed at statistically significant levels ($q < 0.05$); these include the genes encoding 4 WRKY-type TFs. Because WRKY-type TFs have been implicated in the defense mechanism of various plant species[18], we generated rice transformants that overexpressed their cDNAs under the

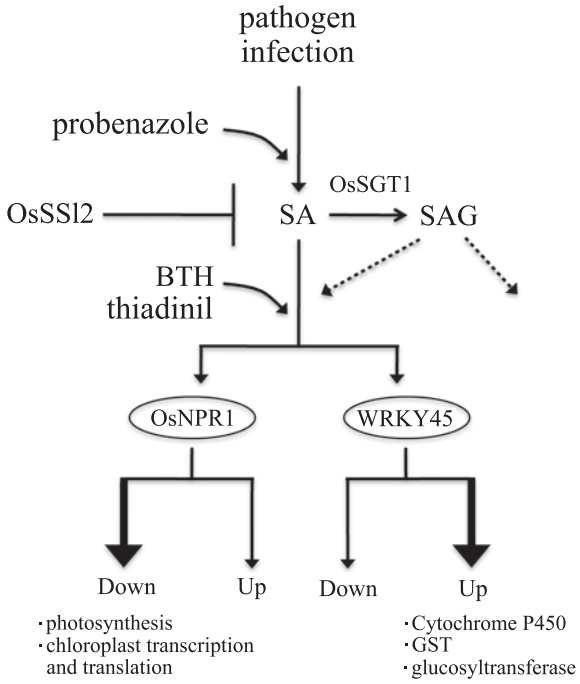

Fig. 1. Current model of the SA signaling pathway in rice

The SA pathway in rice branches into OsWRKY45- and OsNPR1-dependent pathways. OsWRKY45 and OsNPR1 are mainly involved in the upregulation and downregulation of genes, respectively. Probenazole acts upstream of SA, whereas BTH and tiadinil act downstream of SA. OsSSI2 negatively regulates the SA pathway, whereas OsSGT1 plays a positive role in plant defense.

control of a maize *ubiquitin* promoter. Blast resistance tests of these transformants showed that only those transformants that expressed the *OsWRKY45* cDNA exhibited greatly enhanced resistance to a compatible race of blast fungus (race 007, Fig. 2A). We also generated *OsWRKY45*-knockdown (*OsWRKY45*-kd) rice plants (by RNAi technology) and tested them for BTH-inducible blast resistance; we found that this resistance was largely compromised in the *OsWRKY45*-kd plants[23]. *OsWRKY45* knockdown also reduced the blast resistance induced by probenazole and tiadinil (Shimono et al., submitted elsewhere); this indicated that OsWRKY45 plays a crucial role in blast resistance induced by plant activators in general. It is noteworthy that an OsWRKY 45 counterpart has not been identified in *Arabidopsis*.

OsWRKY45 overexpression induces strong resistance to blast and leaf blight diseases

To evaluate the degree of blast resistance of *OsWRKY45*-overexpressing (*OsWRKY45*-ox) rice plants, we compared them with several blast-resistant rice varieties. A blast-resistance test conducted with a compatible race indicated that *OsWRKY45*-ox rice plants are even more blast resistant than Sensho plants (Shimono et al., submitted elsewhere), which are known to be a highly blast resistant rice line due to a single recessive mutation[7]. Taking into account the high blast resistance of Sensho plants that is comparable with those of gene-for-gene-based true blast resistant lines, the blast resistance of *OsWRKY45*-ox rice plants is outstanding. Moreover, *OsWRKY45*-ox rice plants were also resistant to panicle blast caused by fungus infection from the panicle necks (Shimono et al., submitted elsewhere). This is noteworthy because panicle blast directly affects the rice crop yield and grain quality thereby affecting rice production more severely than leaf blast does.

BTH also induces resistance to bacterial leaf blight disease in rice plants[25]. We tested the leaf blight resistance of *OsWRKY45*-ox rice plants and found that these plants were highly resistant to the disease (Fig. 2A, Shimono et al., submitted elsewhere). These results suggest that WRKY45 confers broad-spectrum resistance to rice, and they are consistent with the notion that OsWRKY45 is a key regulator of BTH-induced defense responses.

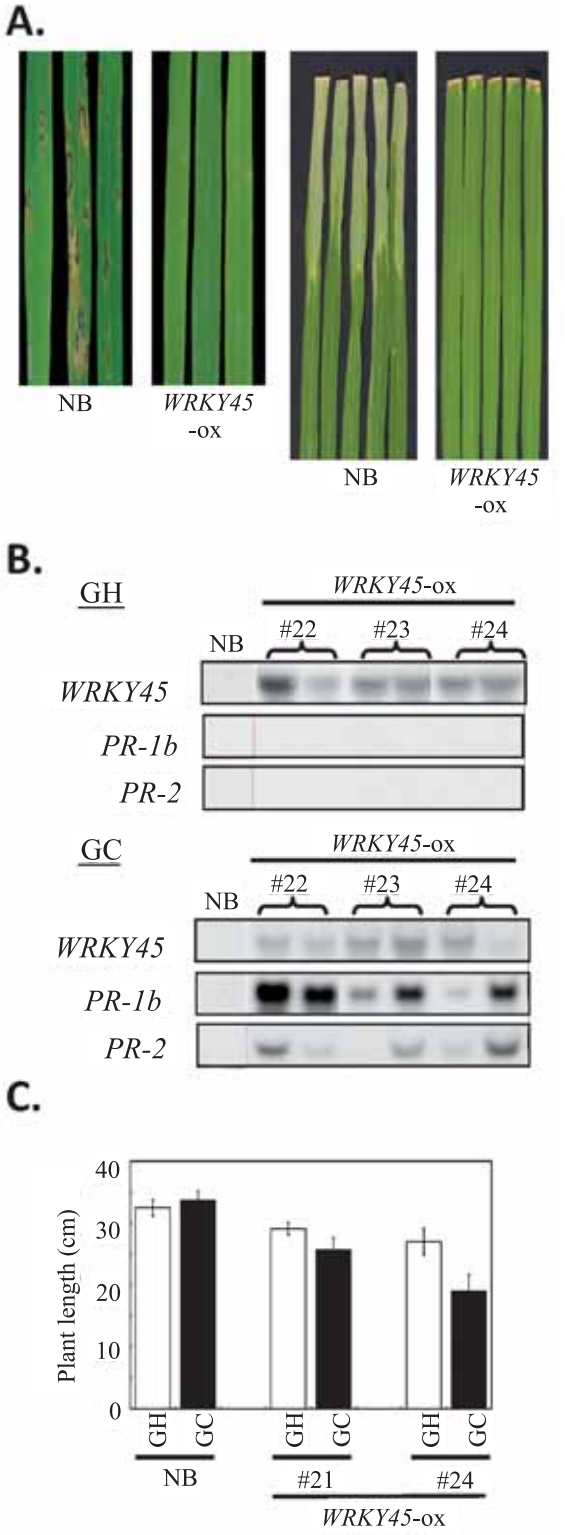

Fig. 2. Phenotypes of OsWRKY45-ox rice plants

A: *OsWRKY45*-ox rice plants are blast (left) and leaf blight (right) resistant.

B: *PR* genes were not expressed in *OsWRKY45*-ox rice plants when they were grown in a growth chamber (GC, upper); however, these genes were highly expressed when the plants were grown in a greenhouse (GH, lower). Results of two plants for each line are shown. NB: control Nipponbare.

C: *OsWRKY45*-ox rice plants grow to shorter heights in a growth chamber (GC) than in a greenhouse (GH).

Environmental factors affect plant growth of OsWRKY45-ox rice

OsWRKY45-ox rice plants grown in our greenhouse did not express *PR1b* and *PR2*, which are marker genes of the SA signaling pathway (Fig. 2B)[23]. Nevertheless, these *OsWRKY45*-ox rice plants were highly blast resistant. This implies that *OsWRKY45*-ox rice plants were primed for defense responses as in the case of plants treated with plant activators. In contrast, *PR1b* and *PR2* were induced at high levels in the *OsWRKY45*-ox rice plants that were grown in our growth chamber. Wild-type rice plants grew to similar heights when they were grown under either of the above-mentioned conditions (Fig. 2C). In contrast, the growth of the *OsWRKY45*-ox rice plants was influenced by the growth conditions; those grown in the growth chamber were notably shorter than those grown in the greenhouse (Fig. 2C). The plant heights were apparently correlated with the *PR* gene expression under different growth conditions (Fig. 2B); this finding suggested that defense responses that were activated in the absence of pathogen infection entailed fitness costs on the *OsWRKY45*-ox rice plants grown in the growth chamber. Under both growth conditions, the growth of the OsWRKY45-ox rice plants was largely restored after prolonged cultivation under long-day conditions, which suggests that the growth phenotypes were attributable to growth retardation rather than irreversible changes. Although the environmental factor(s) responsible for the growth phenotypes have not yet been identified, the candidate influencing factors include the intensity and/or quality of light, temperature and wind.

OsNPR1 also plays a role in the SA signaling pathway in rice

OsNPR1 is another important component of the signaling pathway for BTH-induced disease resistance in rice. *OsNPR1* overexpression driven by the maize *ubiquitin* promoter enhanced the resistance of rice plants to bacterial blight disease[3, 32], while knockdown of this gene increased the susceptibility of the plants to the disease[32]. It has been reported that *OsNPR1* overexpression has no effects on blast resistance[32]; however, the results from our recent study demonstrated that this overexpression in rice conferred enhanced resistance to blast disease (Sugano et al., submitted elsewhere). It is possible that experimental conditions such as the transgene expression levels, growth conditions or plant age, influence the blast-resistance phenotype. The growth of *OsNPR1*-ox rice plants (expression driven by the *ubiquitin* promoter) was comparable with that of the wild-type rice[3]. However, the *OsNPR1*-ox rice plants exhibited severe growth defects when they were grown in a growth chamber. These growth defects were attributed to the low light intensity in the growth chamber[2]. Thus, the growth conditions influenced the growth of both *OsNPR1*-ox and *OsWRKY45*-ox rice plants. However, it remains unclear whether the same environmental factor was responsible for the growth phenotypes of both the transformants.

The SA signaling pathway in rice branches into OsNPR1- and OsWRKY45-dependent pathways

In *Arabidopsis*, NPR1 plays a major role in the SA signaling pathway, more than 99% of BTH-responsive genes are regulated by NPR1[28]. Several WRKY TFs are regulated downstream of NPR1 in *Arabidopsis*. We have provided evidence to prove that the SA signaling pathway in rice branches into OsNPR1- and OsWRKY45-dependent pathways (Fig. 1)[23]. Genome-wide transcript profiling by using *OsNPR1*-kd rice has revealed that the repertoire of OsNPR1-dependent genes is rather limited as compared to that of NPR1-dependent genes in *Arabidopsis*. It is noteworthy that most genes that are downregulated by BTH are OsNPR1 dependent, whereas only a few of those that are upregulated by BTH are OsNPR 1 dependent (Fig. 1) (Sugano et al., submitted elsewhere). Similar experiments using *OsWRKY45*-kd plants demonstrated that among the BTH-responsive genes, OsWRKY45-dependent genes were mostly upregulated (Fig. 1) (Nakayama et al., in preparation). Thus, the OsNPR1- and OsWRKY45-dependent pathways appear to play complementary roles in the defense mechanism mediated by the SA signaling pathway, which is consistent with the branched SA signaling pathway in rice. It is easy to imagine that the branched SA pathway is more adaptable to different situations such as attacks by different types of pathogens under different environmental conditions; however, the precise biological significance of the branched pathway remains unclear.

The genes downregulated by BTH in an OsNPR1-dependent manner include many genes involved in photosynthesis and in transcription and translation in the chloroplasts. These results strongly suggest that OsNPR 1 suppresses chloroplast activity and photosynthesis in response to the SA/BTH signal. Since photosynthesis is a major source of reactive oxygen species (ROSs), the suppression of photosynthesis could reduce the content of ROSs, thereby protecting plants from ROS-induced cellular damage during defense responses. OsWRKY45-dependent genes include the genes encoding several cytochrome P450s, glutathione-S-transferases (GSTs) and

UDP-glucosyl transferases. These genes may be involved in the biosynthesis of defense metabolites that are yet to be identified. GST is also implicated in ROS scavenging; therefore, the OsWRKY45-regulated defense responses may also include ROS reduction.

Other factors that influence the SA signaling pathway in rice

Fatty acids and their derivatives are emerging as important signaling molecules in plant defense pathways. The *Arabidopsis ssi2* mutant, which was identified as a suppressor of the *npr1* mutant[22], is defective in a gene encoding a stearoyl-acyl carrier protein (ACP) desaturase[13]. The *ssi2* plants accumulate endogenous SA at high levels; they constitutively express *PR1* and *PR2* and exhibit enhanced resistance to multiple pathogens[13, 21]. However, the dependence of the *ssi2* defense phenotypes on the SA pathway remains unclear, because the defense phenotypes appear even in SA-deficient *nahG*-overexpressing plants, albeit not prominently[22]. We recently reported that knockdown of *OsSSI2* (*OsSSI 2*-kd), the rice homolog of *SSI2*, resulted in the constitutive expression of *OsWRKY45* and noticeably enhanced the plant resistance to fungal blast and bacterial leaf blight diseases[12]. Microarray transcript profiling of *OsSSI2*-kd rice has revealed an extensive overlap between *OsSSI2*-regulated genes and BTH-responsive genes. Therefore, taken together with the upregulation of *OsWRKY45*, our results suggest that the activation of the SA pathway is responsible, at least in part, for enhanced disease resistance in *OsSSI2*-kd rice plants[12]. Thus, OsSSI2 negatively regulates the SA signaling pathway (Fig. 1).

SA O-ß-glucoside (SAG) is an SA derivative with unknown function. The probenazole-inducible gene *OsSGT1* encodes UDP-glucose:SA glucosyltransferase (OsSGT1), which catalyzes the conversion of free SA into SAG[26]. RNAi-mediated silencing of the *OsSGT1* gene significantly reduces the probenazole-dependent development of blast resistance. This observation suggests that OsSGT1 and its product SAG play positive roles in the defense mechanism in rice, and that SAG is not merely a reservoir for free SA (Fig. 1). These facts should be considered when attempting genetic modification of the SA signaling pathway to improve disease resistance in rice.

Possible applications of OsWRKY45 for developing rice varieties that are resistant to multiple diseases

OsWRKY45 overexpression seems to have effects similar to those of plant activators in conferring broad-spectrum disease resistance. Its effects mimic the priming effect of plant activators, thereby alleviating the cost of defense reactions on plant growth and development. These favorable traits render OsWRKY45 a promising target for the genetic manipulation of rice in order to develop multi-disease-resistant varieties. However, the growth retardation due to OsWRKY45 overexpression driven by the maize *ubiquitin* promoter, which can be exacerbated by an environmental factor(s), is not negligible. Therefore, optimization of the transgene expression is essential for circumventing the growth retardation. A realistic approach for this optimization would be to lower the transgene expression levels by using appropriate constitutive promoters to establish a more stable primed state. The use of pathogen-inducible promoters would be an alternative approach. However, in this case, a system wherein OsWRKY45 expression responds to infection with multiple pathogens should be established in order to achieve resistance of multiple diseases.

Central TFs are often regulated at multiple levels. In fact, accumulating evidence suggests that OsWRKY 45 activity is regulated by complicated mechanisms at both the transcriptional and post-transcriptional levels. These regulatory mechanisms appear to be relevant to the priming effect, the infection strategy of rice pathogens, and environment-triggered activation of OsWRKY 45. It is important to comprehend these regulations in order to optimize the functioning of OsWRKY45 in transformants. OsWRKY45 is a potentially powerful tool, and although the well-controlled use of this TF is a challenge, it could lead to a technology breakthrough that is applicable not only to rice but also to other grasses.

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