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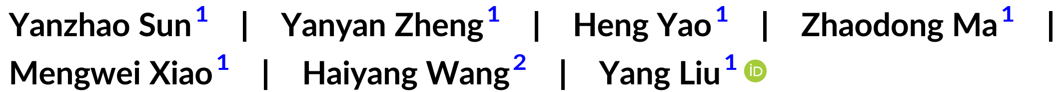
RESEARCH ARTICLE

Light and jasmonic acid coordinately regulate the phosphate responses under shade and phosphate starvation conditions in Arabidopsis



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Abstract

In the natural ecosystem, plants usually grow at high vegetation density for yield maximization. The high-density planting triggers a variety of strategies to avoid canopy shade and competes with their neighbors for light and nutrition, which are collected termed shade avoidance responses. The molecular mechanism underlying shade avoidance and nutrition has expanded largely in the past decade; however, how these two responses intersect remains poorly understood. Here, we show that simulated shade undermined Pi starvation response and the phytohormone JA is involved in this process. We found that the JA signaling repressor JAZ proteins directly interact with PHR1 to repress its transcriptional activity on downstream targets, including phosphate starvation induced genes. Furthermore, FHY3 and FAR1, the negative regulators of shade avoidance, directly bind to promoters of *NIGT1.1* and *NIGT1.2* to activate their expression, and this process is also antagonized by JAZ proteins. All these results finally result in attenuation of Pi starvation response under shade and Pi-depleted conditions. Our findings unveil a previously unrecognized molecular framework whereby plants integrate light and hormone signaling to modulate phosphate responses under plant competition.

KEYWORDS
Arabidopsis, phosphate, regulatory mechanism, shade avoidance

1 | INTRODUCTION

Phosphorus (P) is an essential element of many vital compounds to maintain a wide array of physiological and biochemical processes in plant cells. Plants acquire P through the uptake of soluble inorganic phosphate (Pi) in soils. As a nutrient with low solubility, low mobility, and high fixation by the soil matrix, Pi is inefficiently utilized, with approximately 10–15% recovery rate from fertilizers (Kirkby & Johnston, 2008). To cope with such limited Pi availability, plants have developed a series of adaptive changes to enhance Pi acquisition. These include root architecture remodeling with enhanced growth of lateral roots and root hairs, accumulation of anthocyanins, and upregulated expression of the high-affinity Pi transporter (Lopez-Arredondo et al., 2014).

During the adaption to Pi deficiency, thousands of Pi starvation-responsive (PSR) gene changes were identified in transcriptomic

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analyses in multiple plant species (Barragán-Rosillo et al., 2021; Bustos et al., 2010). Early PSR genes whose expression responded rapidly to Pi deprivation are general stress-responsive and signal transduction-related genes, whereas late PSR genes that responded over 1 day are mainly involved in Pi uptake and redistribution, facilitating Pi acquisition and utilization (Zhang et al., 2014). It has been proposed that MYB-type transcription factor PHR1 and its close homolog PHL1 (PHR1-Like1) are master regulators of transcriptional responses to Pi starvation. They orchestrate the vast majority of this Pi depletion-mediated transcriptional reprogramming, as 80% of the strongly induced genes by Pi-starvation displayed lower expression levels in *phr1phl1* mutant (Bustos et al., 2010; Rubio et al., 2001). Through directly binding to the cis-element P1BS, PHR1 and PHL1 activate the Pi starvation-induced (PSI) genes to coordinate Pi deficiency responses. Earlier studies revealed that multiple regulators modulate PHR1 activity at the transcriptional and posttranscriptional levels, the latter of which is Pi status dependent. For instance, proteins with SYG1/PHO81/XPR1 (SPX) domains, SPX1, can interact with PHR1 protein, thereby sequestering PHR1 and thus inhibiting its binding to downstream target genes in Arabidopsis and rice (Puga et al., 2014; Wang et al., 2014). Studies of PHR1 gene expression have begun to be revealed recently. Light, ethylene, and auxin induce PHR1 and PSI gene expression to enhance Pi uptake under Pi deprivation. Specifically, FHY3, EIN3, and ARF7/19 directly occupy the *PHR1* promoter and activate its expression (Huang et al., 2018; Liu et al., 2017). Recently, NITRATE-INDUCIBLE, GARP-TYPE TRANSCRIPTIONAL REPRESSOR1 (NIGT1) is simultaneously activated by Pi deprivation via PHR1-mediated transcriptional activation (Maeda et al., 2018). During Pi starvation, high activity of NIGT1s activates the expression of the Pi transporter genes (PHT1;1 and PHT1;4), thereby leading to enhancement of Pi uptake (Maeda et al., 2018; Medici et al., 2015; Ueda et al., 2020; Wang et al., 2020).

Nutrition signaling is modulated by various internal and external factors. In agricultural and natural systems, plants typically grow at high densities, which urge plants to perceive the proximity of competitors, thus triggering a series of growth adjustments termed shade avoidance syndrome (SAS), to compete for limited light and nutrition (Casal, 2012; Craine & Dybzinski, 2013; Smith & Whitelam, 1997). Despite the comprehensive understanding of light quality-mediated SAS mechanism, the nutritional signal controlled by light quality is much less discussed. Previous studies have documented that FR light application also changes root system architecture with shorter main root and fewer lateral roots (Salisbury et al., 2007; van Gelderen et al., 2018). These observations imply that the nutrition uptake may be altered under simulated shade conditions. Several SAS factors are found to regulate the expression of genes involved in P metabolism. For example, FHY3 and FAR1, which negatively regulate shade avoidance-mediated growth, are responsible for light-induced *PHR1* expression (Liu et al., 2017, 2019). However, the sophisticated mechanisms to sense and integrate the light quality signal and correspondingly adjust the nutritional system to forage for more favorable conditions are less known.

Jasmonic acid (JA) is an important hormone for plants to respond to defense against pests and diseases, and it is also involved in plant growth and development (Browse, 2009). When plants are stimulated by a series of biotic or abiotic stimuli, the generated JA-Ile directly binds to the COI1-JAZ complex and promotes the degradation of JAZ protein, thereby releasing the inhibition of downstream transcription factors such as MYC2 (Sheard et al., 2010). Earlier studies have demonstrated that the ambient light environment with decreased R/FR ratio stabilizes the JAZ protein and promotes the degradation rate of transcription factors MYC2, MYC3, and MYC4, thus inhibiting JA action (Chico et al., 2014; Leone et al., 2014; Robson et al., 2010). The role of JA in modulating Pi nutrients has just begun to be revealed. For example, Pi deficiency induces JA biosynthesis and the expression of responsive genes that enhance plant resistance to pathogens and herbivorous insects, whereas such induction is attenuated in *phr1*mutant, suggesting that PHR1 plays a pivotal role in this process (Castrillo et al., 2017; Khan et al., 2016). However, the precise mechanism by which JA integrate ambient light to modulate the acquisition and utilization of Pi is not well understood.

In this study, we show that Pi starvation responses are compromised under simulated shade conditions. Low R/FR light reduces Pi content and the inducement of PSI, whereas the application of JA alleviates such responses. We found that JAZ proteins interact with PHR1/PHL1 and repress their activity, thus affecting PSI gene expression. The shade negative regulators FHY3 and FAR1 are shown to bind to *NIGT1.1* and *NIGT1.2* promoters and activate their expression, thereby leading to upregulation of NIGT1s level. In contrast, the increased JAZ1 by extra shade repress PHR1 activity during Pi starvation, thus downregulating the NIGT1s expression compared with white light.

2 | RESULTS

2.1 | Simulated shade conditions attenuate phosphate starvation responses

To examine whether simulated shade conditions would affect the efficiency of Pi utilization, we measured the total Pi content of seedlings in different light conditions. Wild-type seedlings were grown in a high Pi or low Pi medium for 3d under white light (WL) and then were either maintained under WL or transferred to light supplemented with FR (WL + FR) for another 3 days. The results showed that Pi accumulation in WL + FR conditions is significantly reduced compared with WL in either high or low Pi medium, indicating that simulated shade impaired Pi acquisition (Figure 1a). Moreover, Pi starvation-inhibited primary root elongation was prominent in simulated shade (Figure 1b,c). As transcriptional factors PHR1 and its homologs control a majority of Pi uptake and utilization under Pi limited environment, we monitored its protein accumulation in response to simulated shade exposure. Strikingly, PHR1 level was markedly reduced by FR supplementation (Figure 1c). The reduction of PHR1 protein might change the level of PSI gene expression. Consistent with this notion, Pi deficient-induced expression of PSI genes, which function downstream of PHR1 (*PHT1.1*, *RNS1*, *IPS1*, *SPX1*, *PRE8*, and *ACP5*)

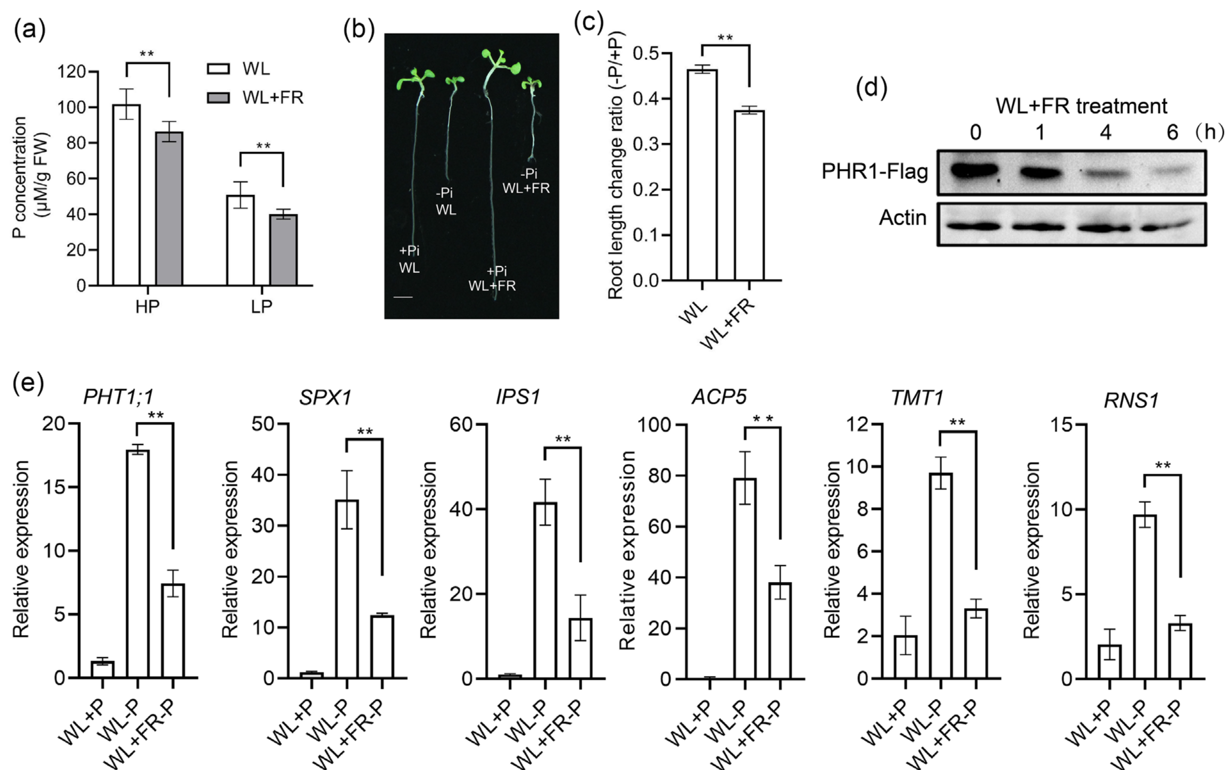

FIGURE 1 FR-enrichment light conditions attenuate Pi starvation responses. (a) Histogram showing Pi content of wild-type (Col-0) seedling grown under white light and simulated shade conditions. Three-day-old seedlings grown on high P medium (HP, 1 mM KH_2PO_4) and low P medium (LP, 10 μM KH_2PO_4) were either retained in white light (WL, high R/FR) or moved to simulated shade (WL + FR, low R/FR) for 7 days. Error bars indicate SD ($n = 10$ plants). Asterisks indicate significant differences between WL and WL + FR grown conditions ($P < .01$, Student's t test). FW, fresh weight. (b and c) Comparison of primary root lengths of wild-type (Col-0) grown in +P and −P medium under white light and simulated shade conditions. Bar = 50 mm. Quantification of root length change is shown using the value of −P/+P. $^{**}P < .01$, Student's t test. Data are means ± SD, $n = 10$ plants. (d) Immunoblot assay shows that accumulation of PHR1 protein decreased in the *35S:PHR1-Flag* seedlings treated with WL + FR light irradiation for the indicated times. Actin was used as a loading control. (e) Relative expression of PSI genes in wild-type plants grown under WL + P, WL−P, and WL + FR-P conditions. For −P conditions, wild-type seedlings were grown on −P medium in white light for 3 days; then, the plants are retained in white light or transferred to simulated shade conditions for 3 days. Asterisks indicate significant differences between WL−P and WL + FR-P ($P < .01$, Student's t test). Values are means ± SD; $n = 3$ biological replicates

(Cardona-Lopez et al., 2015; Rubio et al., 2001), was significantly attenuated in WL + FR light compared with WL-grown seedlings (Figure 1e). Moreover, the inactivation of phyB, which is responsible for the perception of decreased R:FR, resulted in severely reduced PSI induction by Pi starvation (Figure S1). These results indicate that shade represses PHR1 accumulation and Pi starvation response.

2.2 | JA is involved in shade-repressed phosphate starvation responses

Previous studies have shown that low ratios of R/FR light repress, while Pi deficiency induces JA pathway (Castrillo et al., 2017; Khan et al., 2016). Therefore, we hypothesized that JA pathway might participate in the shade-repressed Pi starvation responses. To test this idea, we first examined the PHR1 level changes in response to JA application. White light-grown seedlings are transferred to WL + FR, with or without JA supplementation in the medium. Interestingly, both PHR1 mRNA transcript and protein accumulation are reduced upon FR supplementation, while additional JA treatment partially rescued such reduction (Figures 2a and S2). In addition, the decreased Pi content under low R/FR is also recovered when JA is present (Figure 2b). These results implied that JA might alleviate the Pi starvation response. To this end, we investigated the role of JA on PSI gene expression by RT-qPCR. Seedlings transferred from WL to WL + FR were either grown in a Pi-depleted medium with or without JA supplementation. As expected, JA application significantly enhanced four PSI induction (*RNS1*, *PHT1;1*, *SPX1*, and *IPS1*) under simulated shade, suggesting that JA positively regulates Pi starvation responses (Figure 2c). Furthermore, *coi1* mutant with interrupted JA reception (Xie et al., 1998) and *JAZ1OE* plants with repressed JA downstream signaling (Thines et al., 2007) were used to detect PSI's expression profile when Pi is depleted. Consistent with the above observation, induction of PSI expression by Pi deprivation is significantly reduced in *coi1* mutant and *JAZ1OE* plants compared with wild type (Figure 2d). These results suggest that suppression of JA action under shade conditions is partially causal for the impairment of Pi starvation response.

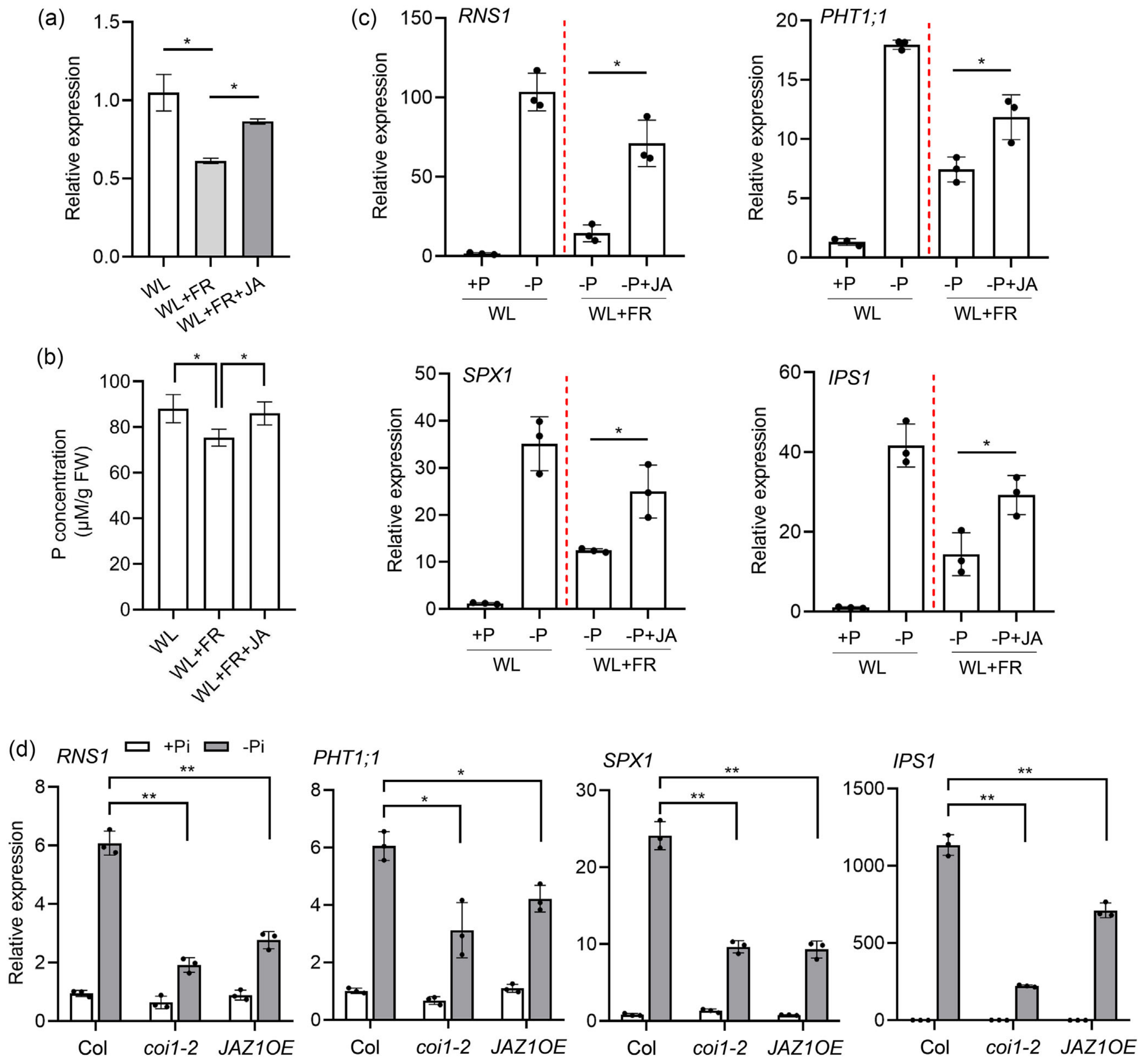

FIGURE 2 Jasmonic acid is involved in shade-mediated inhibition of phosphate starvation responses. (a) Relative expression of *PHR1* in wild type seedlings grown 3 days in WL, transferred to ±1 μM MeJA medium and then shifted to WL ± FR at day 3. Tissues were harvested at day 6. *$P < .05$, Student's t test. Values are means ± SD; $n = 3$. (b) Histogram showing Pi content of wild-type seedlings grown 3 days in WL, transferred to ±1 μM MeJA medium and then shifted to WL ± FR at day 3. Tissues were harvested at day 6. *$P < .05$, Student's t test. Values are means ± SD; $n = 10$ plants. (c) Relative expression of *PSI* genes in wild type plants grown under WL ± P and WL−P ± FR conditions. For −P conditions, wild type seedlings were grown on −P medium in WL for 3 days, transferred to ±1 μM MeJA medium and then shifted to WL + FR conditions for 3 days. Tissues were harvested at day 6. *$P < .05$, Student's t test. Values are means ± SD; $n = 3$. (d) Relative expression of PSI genes in wild type, *coi1-2* and *JAZ1OE* seedlings grown +P and −P medium. *$P < .05$, Student's t test. Values are means ± SD; $n = 3$

2.3 | PHR1 and PHL1 interact with JAZs

As general repressors of the JA pathway, JAZ proteins are stabilized by an FR-enriched light environment, which is causal for the undermined defensive capability in shade conditions (Leone et al., 2014). To investigate how JAZs are involved in the interaction between JA and shade-mediated PSR disruption, we tested if JAZs interact with PHR1 to modulate Pi starvation-related gene expression. Because there are 11 JAZ family members in Arabidopsis, we first adopted yeast two-hybrid assays to detect their pair-wise protein–protein interactions with PHR1 and PHL1. Notably, multiple JAZs (JAZ1, JAZ3, JAZ8, JAZ10, and JAZ11) directly interacted with PHR1 and PHL1 (Figure 3a). Additionally, the interaction between JAZ1 and PHR1 was confirmed by glutathione S-transferase (GST) pull-down using His-tagged JAZ1 protein and GST-tagged PHR1 (Figure 3b). We next tested the interaction of JAZ1 with PHR1 and PHL1 in vivo by a

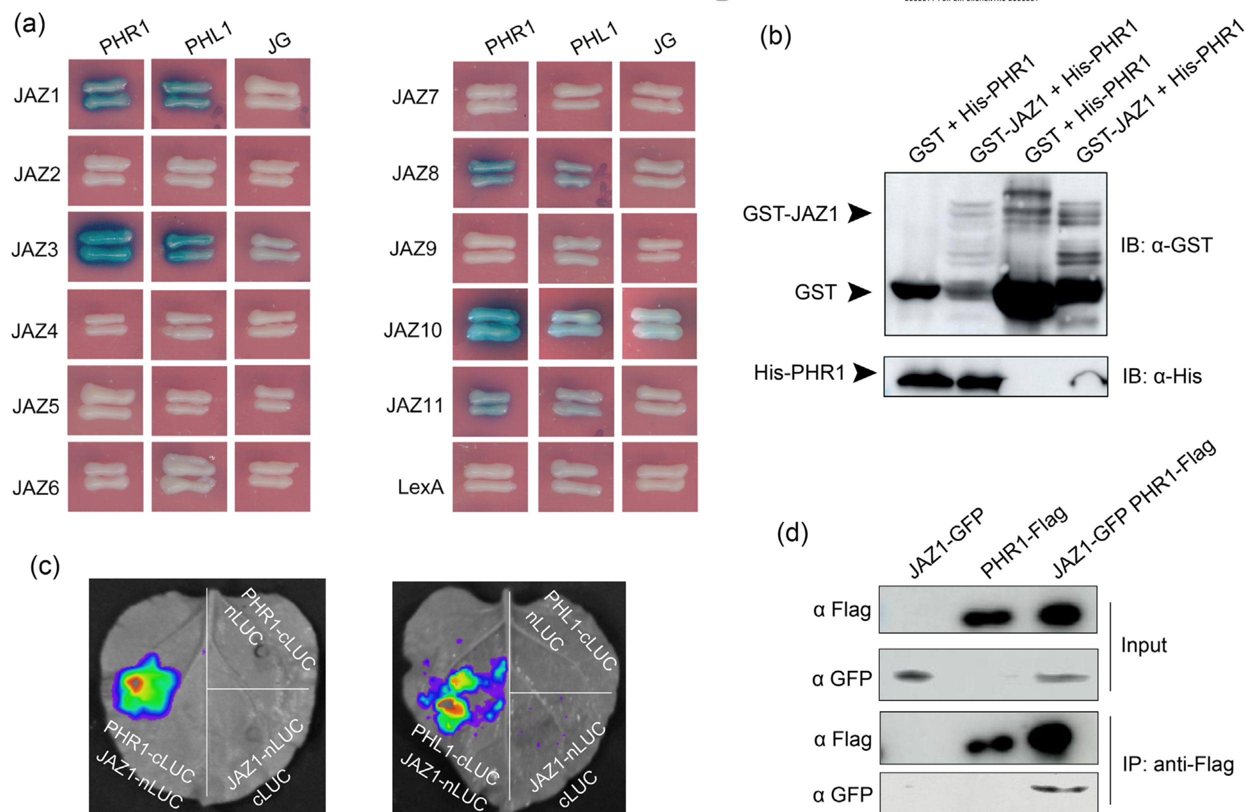

FIGURE 3 PHR1 and PHL1 interact with JAZs. (a) Yeast two-hybrid assay showing interactions of JAZs with PHR1 and PHL1. The JAZ proteins were fused with the LexA DNA binding domain in pEG202. PHR1 and PHL1 were fused with the activation domain (AD) in pB42AD. (b) In vitro pull-down assay verifying the interaction of PHR1 with JAZ1. GST and GST-JAZ1 expressed in *Escherichia coli* were incubated with His-PHR1. Proteins were pulled down with glutathione sepharose resin and detected in immunoblotting with anti-His antibody. (c) LCI assay showing that PHR1 and PHL1 interact with JAZ1 in planta. The C-terminal half of firefly LUC (cLUC) was fused to PHR1 or PHL1, and the N-terminal half of firefly LUC (nLUC) was fused to JAZ1. (d) Co-IP assay showing that PHR1 associates with JAZ1 in *N. benthamiana* leaves in vivo. Protein extracts expressing *35S:PHR1-Flag* and *35S:JAZ1-GFP* were immunoprecipitated using anti-Flag antibody and detected using anti-Flag (1:4,000) or anti-GFP (1:1,000) antibody, respectively.

luciferase complementation imaging (LCI) assay with PHR1 and PHL1 fused to the C terminus of LUC (cLUC) and JAZ1 fused to the N terminus of LUC (nLUC). When transiently co-expressed in *N. benthamiana* leaves, JAZ1-nLUC with PHR1-cLUC or PHL1-cLUC resulted in a reconstituted LUC signal, while no signal was observed in negative controls (Figure 3c). Moreover, the in vivo interaction of JAZ1 with PHR1 was further confirmed by coimmunoprecipitation (Co-IP) assay, with PHR1-Flag precipitating JAZ1-GFP in *N. benthamiana* leaves (Figure 3d). These data collectively suggested the interaction of JAZ1 with PHR1.

2.4 | JAZ1 represses PHR1 activity and phosphate starvation response

Given the reduced PSI expression in *JAZ1OE* plants, we hypothesized that JAZ1 might function to repress PHR1 and PHL1 to regulate the PSI expression. To test this, we chose IPS1 as the representative target of PHR1 and PHL1 to investigate the effect of JAZ1 on its transcription in a transient expression assay using *N. benthamiana* leaves. As shown in Figure 4a,b, PHR1 or PHL1 effectively activated *IPS1pro::LUC* reporter gene expression, whereas co-expression of JAZ1 with PHR1 or PHL1 significantly repressed the expression of the reporter gene.

A previous study has documented that FHY3 plays an important role in the light-induced activation of PHR1 (Liu et al., 2017). Since JAZ1 also interacts with FHY3 and represses its transcriptional activity, it prompted us to determine whether JAZ1 restrain PHR1 transcript via interference of FHY3 activity. Transient expression assay verified this presumption with FHY3-activated PHR1 reporter gene severely repressed when JAZ1 co-expressed (Figure 4c). In support of this notion, qRT-PCR experiments showed that light-induced *PHR1* expression is significantly reduced in *JAZ1OE* plants compared with wild type (Figure 4d).

Next, we characterized the Pi concentration in wild type and *JAZ1OE* plants grown in high Pi and low Pi conditions, respectively. As expected, Pi accumulation is significantly reduced when JAZ1 is overexpressed (Figure 4e). Furthermore, phenotypic analysis revealed that the main root of *JAZ1OE* plants grown in Pi deprivation medium was severely inhibited, showing a onefold reduction of the -P/+P ratio

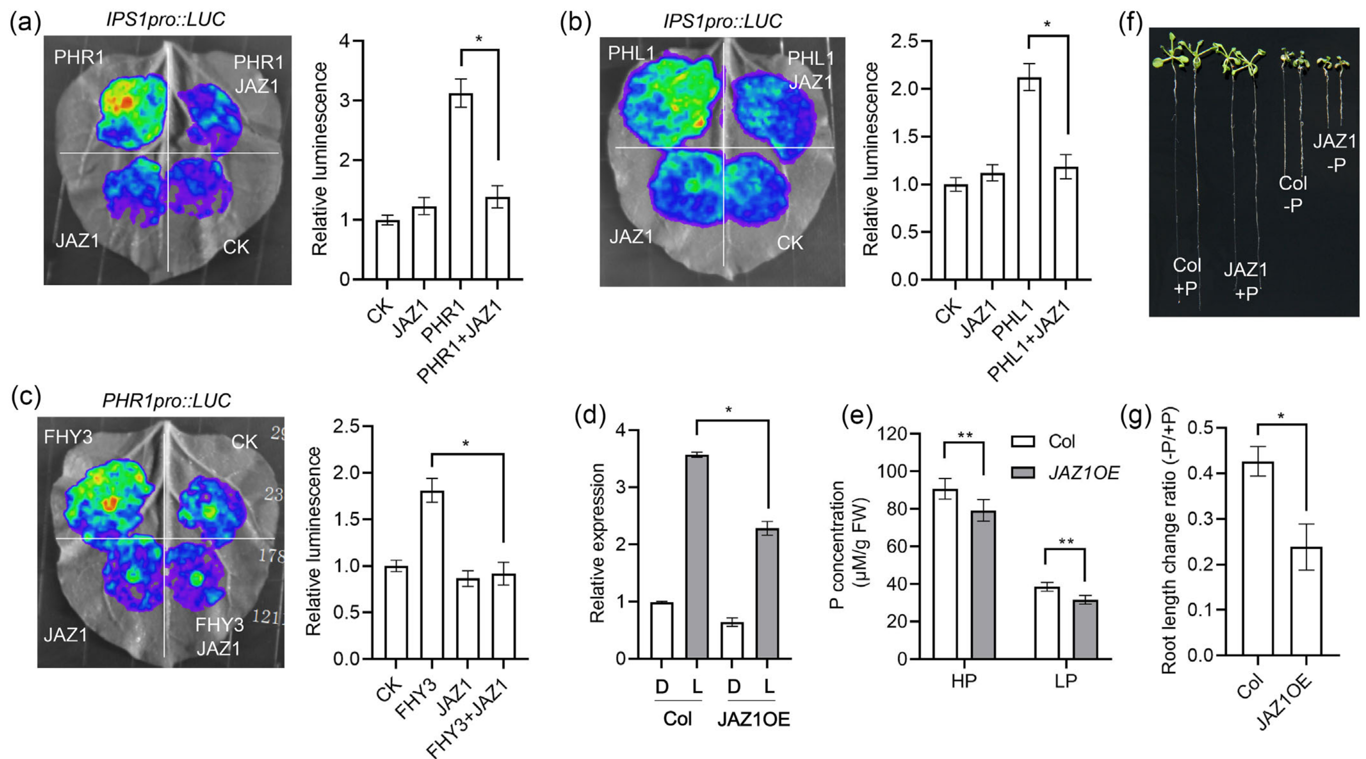

FIGURE 4 JAZ1 inhibit Pi starvation responses by inhibiting PHR1 activity. (a and b) Transient expression assay shows that JAZ1 represses the transcriptional activity of PHR1 and PHL1 on *IPS1pro::LUC* in *N. benthamiana* leaf. The right panels of (a) and (b) show quantification of the transient expression assay. *$P < .05$, Student's *t* test. Values are means ± SD; $n = 3$. (c) Transient expression assay shows that JAZ1 represses the transcriptional activity of FHY3 on *PHR1pro::LUC* in *N. benthamiana* leaf. The right panel shows quantification of the transient expression assay. *$P < .05$, Student's *t* test. Values are means ± SD; $n = 3$. (d) Relative expression of *PHR1* in light- and dark-grown wild type and *JAZ1OE* seedlings. Six-day-old seedlings grown on +P and −P medium were collected for RNA extraction. *$P < .05$, Student's *t* test. Values are means ± SD; $n = 3$. (e) Histogram showing Pi content of wild-type and JAZ1OE seedlings grown under HP and LP conditions. Ten-day-old seedlings grown on high P medium (HP, 1 mM KH_2PO_4) and low P medium (LP, 10 μM KH_2PO_4) were collected. Error bars indicate SD ($n = 10$). *$P < .05$, Student's *t* test. (f and g) Comparison of primary root lengths of wild-type and JAZ1OE seedlings grown in +P and −P medium for 10 days. Bar = 50 mm. Quantification of root length change is showed using the value of −P/+P in (G). *$P < .05$, Student's *t* test. Data are means ± SD, $n = 10$ plants.

compared with wild type (Figure 4f,g). Also, the shorter root of the *JAZ1OE* transgenic plants under −Pi conditions was fully rescued by overexpression of PHR1 (Figure S3). Taken together, our results suggested that JAZ1 repress PHR1 activity and thus negatively regulates Pi starvation response.

2.5 | FHY3 and FAR1 directly activate the expression of NIGT1.1 and NIGT1.2

Besides PHR1, other Pi starvation-induced transcription factors have been uncovered. For example, the MYB-related transcription factors NIGT1s play important roles in Pi Pi uptake capacity under low Pi stress (Maeda et al., 2018; Medici et al., 2015; Ueda et al., 2020; Wang et al., 2020). Thus, we attempt to identify whether shade conditions could modulate NIGT1s expression. RT-qPCR assay indicated that three out of four NIGT1s (*NIGT1.1*, *NIGT1.2*, and *NIGT1.3*) displayed compromised upregulation by Pi starvation when growing under WL + FR conditions (Figure S4). The change of *NIGT1s* expression in response to WL + FR conditions indicates that they might be modulated by the shade action. To identify the potential regulators that conferred to shade altered *NIGT1s* expression, we conducted a cis-regulatory element analysis of NIGT1 promoters. Notably, canonical FHY3/FAR1 binding sites (FBS; CACGCGC) were identified in *NIGT1.1* and *NIGT1.2* promoters (Figure 5a). A yeast one-hybrid assay showed that FHY3 and FAR1 were able to bind to the promoters of *NIGT1.1* and *NIGT1.2*. No binding was observed in promoters of *NIGT1.3* and *NIGT1.4* (Figure 5b). The binding of FHY3 to the *NIGT1.1* and *NIGT1.2* promoters was further verified by an electrophoretic mobility shift assay (EMSA) and chromatin immunoprecipitation (ChIP) assay (Figure 5c–f). Next, we performed a transient expression assay to examine the regulatory effect of FHY3 on *NIGT1.1* and *NIGT1.2* expression in *N. benthamiana* leaf cells. FHY3 activated the expression of the *NIGT1.1pro:LUC* and *NIGT1.2pro:LUC* reporters (Figure 5g,h). Moreover, RT-qPCR showed that expression of *NIGT1.1* and *NIGT1.2* are significantly declined in *fhy3 far1* mutant compared with wild type in either WL or WL + FR conditions, with a very significant difference occurring in WL + FR (Figure 5i,j). All these results suggest that FHY3 and FAR1, the negative regulators of shade avoidance, directly activate *NIGT1.1* and *NIGT1.2* expression.

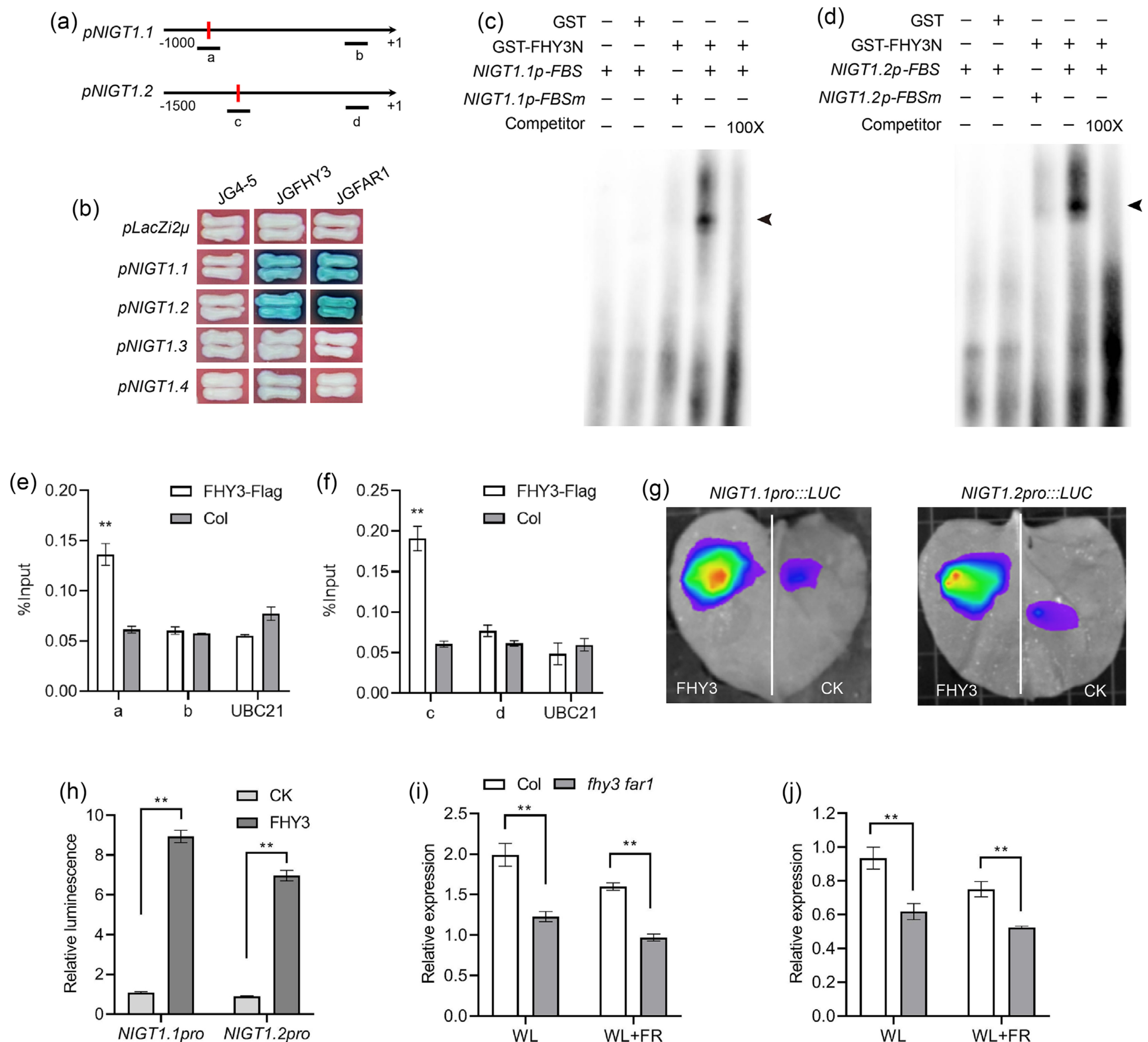

FIGURE 5 FHY3 and FAR1 directly upregulate the expression of *NIGT1.1* and *NIGT1.2*. (a) Schematic diagram of the promoter regions for *NIGT1.1* and *NIGT1.2*. Red lines indicate FBS cis-elements. Letters a, b, c, and d represent the fragments used for amplification in the ChIP-qPCR assay. (b) Yeast one-hybrid assay showing that FHY3 and FAR1 bind to the *NIGT1.1* and *NIGT1.2* promoters. Empty vector expressing the AD alone was used as the negative control. (c and d) Competitive EMSA showing that GST-FHY3N specifically bind to the *NIGT1.1p-FBS* and *NIGT1.2p-FBS* probes, respectively. The arrowheads indicate N-terminal GST-FHY3 protein. One-hundred-fold molar excesses of unlabeled probes were used in the competition assay. (e and f) ChIP-qPCR analysis of FHY3 binding to the *NIGT1.1* and *NIGT1.2* promoter regions. Ten-day-old seedlings of *35S:FHY3-Flag* and the wild type were harvested and immunoprecipitated using anti-Flag antibody. Values are means ± SD; $n = 3$. (g and h) Transient expression assay showing that FHY3 activates expression of *NIGT1.1* and *NIGT1.2* in *N. benthamiana* leaf cells ($^{**}P < .01$, Student's *t* test). Values are means ± SD; $n = 3$. (i and j) Relative expression of *NIGT1.1* and *NIGT1.2* in wild type and *fhy3 far1* seedlings grown 3 days in WL and WL + FR conditions. $^{**}P < .01$, Student's *t* test. Values are means ± SD; $n = 3$

2.6 | JAZ1 represses expression of *NIGT1.1/1.2* in Pi-starvation conditions

Since *NIGT1.1* and *NIGT1.2* are both directly targeted by PHR1 and FHY3 in response to light and Pi availability, respectively, it prompted us to test the synergistic effect of these two transcription factors on expression of *NIGT1.1* and *NIGT1.2*. Transient expression assay in *N. benthamiana* leaves showed that FHY3 and PHR1 cooperatively promoted *NIGT1.1* and *NIGT1.2* expression (Figure S5). Consistent with this, RT-qPCR analysis showed that Pi-deficient activated expression of *NIGT1.1* and *NIGT1.2* was significantly reduced in the *fhy3 far1 phr1 phl1* quadruple mutant compared with the *fhy3 far1* and *phr1 phl1* mutants (Figure 6a,b). Based on this finding, the factors that influence PHR1 and FHY3 activity would also have an impact on

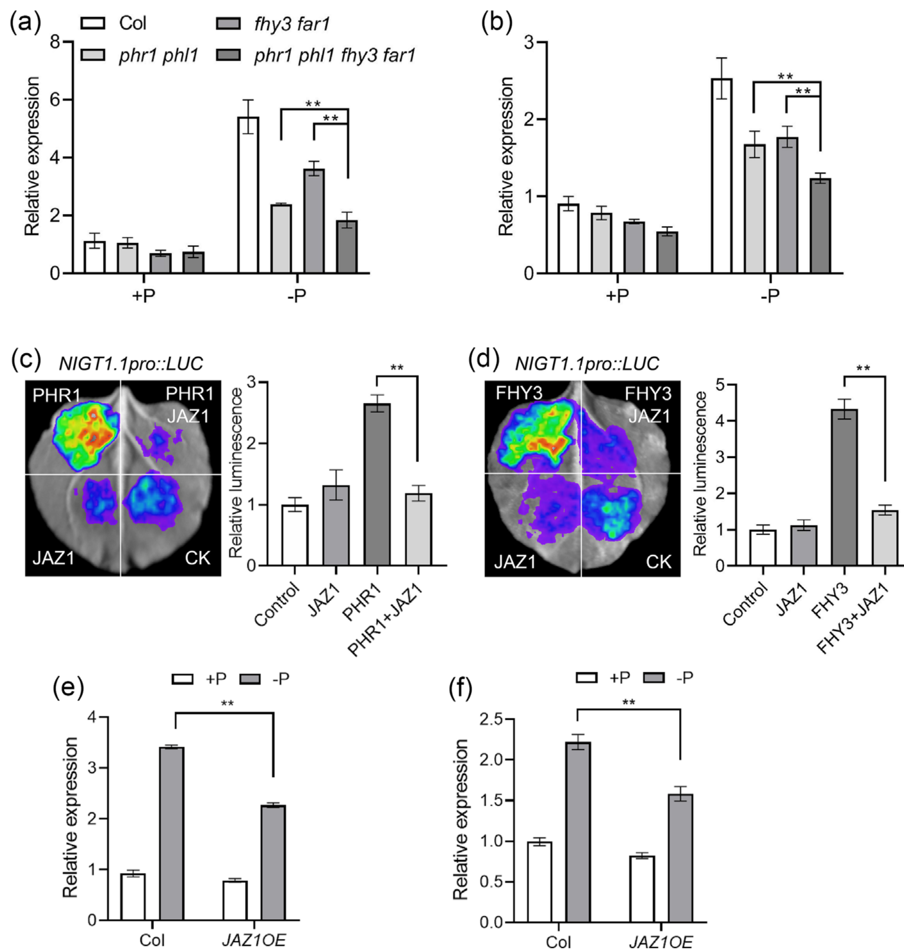

FIGURE 6 JAZ1 inhibits expression of *NIGT1.1* and *NIGT1.2* through repression of PHR1 and FHY3 activities. (a and b) Relative expression of *NIGT1.1* and *NIGT1.2* in wild type (Col), *phr1 phl1*, *fhy3 far1* and *phr1 phl1 fhy3 far1* seedlings grown under +P and −P conditions. $^{**}P < .01$, Student's *t* test. Values are means ± SD; $n = 3$. (c and d) Transient expression assay shows that JAZ1 represses the transcriptional activity of PHR1 and FHY3 on *NIGT1.1pro::LUC* and *NIGT1.2pro::LUC* in *N. benthamiana* leaf. The right panels of (c) and (d) show quantification of the transient expression assay. $^{**}P < .01$, Student's *t* test. Values are means ± SD; $n = 3$. (e and f) Relative expression of *NIGT1.1* and *NIGT1.2* in wild type (Col) and *JAZ1OE* seedlings grown under +P and −P conditions. $^{**}P < .01$, Student's *t* test. Values are means ± SD; $n = 3$

NIGT1s expression. Because JAZ1 represses the transcriptional activity of PHR1 and FHY3, it is very likely that expression of *NIGT1s* is also modulated by JAZ1 protein. Transient expression assay showed that co-expression of JAZ1 with PHR1 or FHY3 significantly repressed the expression of *NIGT1.1pro:LUC* reporters (Figure 6c,d). Also, RT-qPCR showed that Pi limitation-induced expression of *NIGT1.1* and *NIGT1.2* is substantially declined when JAZ1 is overexpressed (Figure 6e,f). These results support the notion that JAZ1 represses the expression of *NIGT1.1* and *NIGT1.2*.

3 | DISCUSSION

Plants are sessile organisms and do not grow in isolation, especially those grown in densely planted environments need to compete with neighboring vegetation for limited light and nutrients. Thus, it is necessary to pay attention to the impact of neighbor proximity in the natural ecosystem regardless of whether studying light signal transduction or nutrient absorption and utilization. In this study, we unveil a novel molecular framework in which the phytohormone JA integrates shade with Pi starvation. We show that under simulated shade, Pi starvation response is attenuated partially through downregulation of PHR1 activity. JAZ proteins, the repressors of JA pathway, accumulate under shade and inhibit the transcriptional activity of PHR1 downstream PSI expression. We further show that FHY3, the negative regulator of shade avoidance, directly binds to the promoters of *NIGT1.1/1.2* and activates their expression and that this process is also repressed by JAZ proteins. Our results provide insight into the complex interplay between nutrition, light and hormone that help plants to change the mineral homeostasis in an unfavorable environment.

Pi acquisition and other PSRs in Pi-limited conditions are determined by various factors. Light, the essential external factor that provides energy and serves as an information signal to promote growth, has been shown to affect Pi starvation responses (Chen et al., 2004). For instance, light supply and active photosynthesis are required for PSR and expression of PSI under Pi-limited conditions (Lei et al., 2011; Liu et al., 2017). A recent study has documented that expression of *PHR1* is induced during light, while repressed by dark, via phytochrome-FHY3 pathway (Liu et al., 2017). As FHY3 has been confirmed to play a negative role in shade avoidance, and its activity is reduced on more prolonged exposure to FR irradiance (Liu et al., 2017; Xie et al., 2017), thus, it is inferred that simulated shade ought to downregulate *PHR1* transcript and the relevant PSRs, results of which are proved in this study. Mechanistically, we provide another two regulatory modes of PHR1 at a posttranscriptional level under shade conditions. First, we demonstrate that PHR1 protein accumulation is reduced on low R/FR irradiance (Figure 1d). Second, the

transcriptional activity of PHR1 is strongly repressed by JAZ proteins (Figure 4). All these overlying results ultimately reinforce the reduction of PHR1 activity and thus PSRs under shade conditions. Consistent with this notion, expression of Pi deprivation-induced PSI, as well as total Pi concentration, is compromised under simulated shade conditions (Figure 1e).

Under the adverse environment where Pi is limited, plants have evolved a long-distance signaling pathway to adjust Pi utilization, including enhancement of Pi uptake, reallocation, and recycling to ensure the metabolic balance of P at the whole-plant level (Thibaud et al., 2010). Several molecules, such as Pi, sugars, hormones, and RNAs, have been recognized as systemic signals (Chien et al., 2018). Recently, the action of light has been indicated to implicate in this process. A recent work identified phyB-PIF4/PIF5/HY5 pathway as a regulatory module in controlling red-light-mediated Pi uptake. They found that PIF4 and PIF5 directly repress *PHT1;1*, the Pi uptake-related gene, and *PHL1*, while HY5 activates *PHT1;1* when plants grown under low Pi stress (Sakuraba et al., 2018). Due to the capability of HY5 as a shoot-to-root mobile signal, it might be involved in long-distance signaling in regulating Pi acquisition under red light or white light. As for plants grown under shade conditions, it is supposed that FR-enriched irradiation impedes the systemic or long-distance responses through modulation of the PHR1 level at the whole plant level. Two lines of scenarios are proposed to support this notion. First, it has been shown that FR-supplementation promotes HY5 stabilization and facilitates its transport to the root (van Gelderen et al., 2018). Meantime, HY5 can directly bind to *PHR1* promoter and repress its expression (Liu et al., 2017). Second, HY5 has been shown to repress the activity of FHY3 by inhibiting its DNA binding (Liu et al., 2017). Hence, it is estimated that the shoot-to-root transportation of HY5 restricts the activity of FHY3 and their downstream target PHR1 in the root, which allows the root to perceive the change of ambient light in the aboveground and correspondingly adjust the Pi metabolism underground. Accordingly, based on the finding that (1) shade inhibits lateral root emergence (van Gelderen et al., 2018); (2) shade downregulates PHR1 level (this study); and (3) low Pi induced lateral root formation is dependent on PHR1 (Huang et al., 2018), the Pi starvation promoted lateral root growth is very likely decreased as a result of ambient FR enrichment perceived by shoot under shade conditions.

Based on these findings in conjunction with earlier reports, we unveil a previously unrecognized molecular framework whereby plants integrate light and hormone signaling to modulate Pi responses under plant competition. Previous studies have demonstrated that shade (low R/FR ratios) stabilize JAZ proteins (Leone et al., 2014; Robson et al., 2010). Under simulated shade conditions, the activity of FHY3 and PHR1 is repressed by JAZ proteins. On the one hand, PHR1 activity and expression level is reduced, leading to downregulation of PSI and Pi uptake. On the other hand, expression of *NIGT1.1* and *NIGT1.2*, which are the targets of FHY3 and PHR1, declined, further declining the expression of Pi transporter genes. Under white light conditions, JAZ proteins are destabilized, while FHY3 and PHR1 accumulate. These changes increased the expression levels of FHY3 and PHR1 downstream targets: *PHR1*, various PSI, *NIGT1.1*, and *NIGT1.2*, which enhance Pi uptake in low Pi stress.

4 | EXPERIMENTAL PROCEDURES

4.1 | Plant materials and growth conditions

The wild-type and mutant Arabidopsis (*Arabidopsis thaliana*) plants used in this study are of the Col-0 genetic background. The *coi1-2* (Xu et al., 2002), *fhy3-11 far1-4* (Liu et al., 2019), *phr1* (salk_067629, Liu et al., 2017), and *phl1* (SAIL_731_B09; Bustos et al., 2010) mutants and the transgenic lines *35S:JAZ1-GUS* (JAZ1OE; Thines et al., 2007); *35S:PHR1-Flag* (Liu et al., 2017); and *35S:FHY3-Flag* (Liu et al., 2020) have been reported. The *phr1 phl1* and *fhy3-11 far1-4 phr1 phl1* mutants were generated by genetic crossing. Genotyping of the T-DNA insertion mutants was performed using the T-DNA left border primer and gene-specific primers designed in a web tool (at http://signal.salk.edu/tdnaprimers.html).

After vernalization for 2 days at 4°C, plates were incubated in a growth chamber (Percival Scientific, cool white fluorescent bulb at 22°C) under continuous white light (16/8 light/dark photoperiod, LED light, PAR = 40 μmol m^{-2} s^{-1}; red: 640–670 nm, 15 μmol m^{-2} s^{-1}; FR: 720–750 nm, 1.2 μmol m^{-2} s^{-1}) for 3 days. The plates were then either kept in white light or transferred to simulated shade (LED continuous white light plus FR, PAR = 36 μmol m^{-2} s^{-1}; red: 640–670 nm, 20 μmol m^{-2} s^{-1}; FR: 720–750 nm, 90 μmol m^{-2} s^{-1}) for 3 days. For Pi+ medium, plants were grown in Murashige and Skoog (MS) medium supplemented with 1 mM KH_2PO_4. For the Pi-deficient medium, KH_2PO_4 was replaced with equimolar amounts of KCl.

4.2 | Plasmid construction

All plasmids were constructed using an In-Fusion HD cloning kit (Clontech). To generate the *pNIGT1.1:LacZ* and *pNIGT1.2:LacZ* reporter constructs, oligonucleotides were synthesized as two complementary oligo primers with an EcoRI site overhang at the 5′ end and an XhoI site overhang at the 3′ end. The oligo primers were annealed, and the double-stranded oligonucleotides were ligated into the EcoRI-XhoI sites of the pLacZi2μ vector. AD-PHR1 and AD-PHL1 were generated by subcloning the full-length coding sequence (CDS) of PHR1 and PHL1 into the pB42AD vector through EcoRI-XhoI sites. AD-FHY3, AD-FAR1, AD-PHR1, AD-PHL1, and various LexA-JAZs were previously described by Liu et al. (2017) and Liu et al. (2019). To generate *NIGT1.1pro::LUC* and *NIGT1.2pro::LUC*, promoters of NIGT1.1 and NIGT1.2 were amplified and cloned into pGreenII 0800-LUC through PstI and BamHI sites. *IPS1pro::LUC* and *PHR1pro::LUC* were previously described (Liu et al., 2017). For the 35S promoter-driven effector constructs, the cDNAs of PHR1 and PHL1 were amplified and cloned into the SPYNE vector at the BamHI and SalI sites. For the vectors in firefly LCI assays, the CDSs of PHR1 and PHL1 were ligated into the KpnI/SalI sites of the p1300- 35S-nLUC

vector, and the JAZ1 CDS was ligated into KpnI/SalI sites of the p1300-35S-cLUC vector (Chen et al., 2008).

4.3 | Yeast assay

Yeast one-hybrid and yeast two-hybrid assays were performed as described previously (Liu et al., 2017).

4.4 | RNA extraction and RT-qPCR

The seedling samples were harvested, frozen immediately in liquid nitrogen, and stored at −80°C until use. Total RNA was extracted from the seedlings using Trizol (Invitrogen) following the manufacturer's protocols. The first-strand cDNA was synthesized from 1 μg of RNA using reverse transcriptase (Tiangen, FastQuantRT Kit) following digestion with gDNase from the kit to remove genomic DNA contamination. The cDNA was diluted 1:10 and subjected to qPCR using SuperReal PreMix Plus (Tiangen) and a Q3 Real Time PCR System (Applied Bio-systems) cycler. Gene expression levels were normalized to *UBQ5* using the comparative C_T method. All experiments were replicated two or three times with similar results. Primers are listed in Dataset S6.

4.5 | ChIP-qPCR

ChIP experiments were performed as described with modifications (Liu et al., 2020). Briefly, ∼2 g of wild-type and *35S:FHY3-Flag* transgenic seedlings were cross-linked for 10 min in 1% (v/v) formaldehyde solution under a vacuum. Then, it was isolated using nuclear lysis buffer (50 mM Tris–HCl at pH 8.0, 10 mM EDTA, 1% SDS, 1 mM PMSF, and 1 × protease inhibitor cocktail), diluted fivefold in ChIP dilution buffer (16.7 mM Tris–HCl at pH 8.0, 167 mM NaCl, 1% Triton X-100, 1.2 mM EDTA, 1 mM PMSF, and 1 × protease inhibitor cocktail), and sheared by sonication. The sonicated chromatin complex was immunoprecipitated using anti-Flag antibodies (2 μl; Cali-Bio). The beads were washed with low-salt buffer (150 mM NaCl), high-salt buffer (500 mM NaCl), LiCl buffer (10 mM Tris–HCl at pH 8.0, 1 mM EDTA, .25 M LiCl, .5% NP-40, and .5% deoxycholate), and TE buffer (10 mM Tris–HCl at pH 8.0 and 1 mM EDTA) and eluted with elution buffer (1% SDS and .1 M $NaHCO_3$). After reverse cross-linking, the DNA was precipitated with phenol/chloroform/isoamyl alcohol and analyzed by qPCR. Primers used for ChIP-qPCR are listed in Dataset S1.

4.6 | EMSA

EMSA was performed using a LightShift Chemiluminescent EMSA kit (Pierce) according to the manufacturer's instructions. GST-FHY3N fusion proteins were described previously (Liu et al., 2017). For probe preparation, two complementary oligonucleotides (60 bp long) of the *NIGT1.1* and *NIGT1.2* promoters containing the FBS sites were synthesized, annealed, and labeled with biotin. The oligonucleotide sequences are listed in Dataset S1. Briefly, biotin-labeled probes were incubated for 20 min with the expressed proteins in binding buffer at room temperature. The DNA-protein complexes were separated on 6% (w/v) native polyacrylamide gels, and the signal was detected using the Amersham ImageQuant 800 system.

4.7 | Immunoprecipitation assay

35S:PHR1-Flag and *35S:JAZ1-GFP* constructs were introduced into *A. tumefaciens* strain EHA105 and cotransformed into the *N. benthamiana* leaves. After 3 days of incubation, total protein was extracted using a homogenization buffer (50 mM Tris–HCl, pH 7.5, 150 mM NaCl, 10 mM $MgCl_2$, .1% Tween 20, 1 mM PMSF, and 1 × protease inhibitor cocktail), and the extract was mixed with anti-Flag magnetic agarose beads (MBL, M185–10). After incubation overnight at 4°C, the beads were centrifuged and washed. The protein was eluted with 40 ml of loading buffer and analyzed by immunoblotting using anti-GFP antibody (MBL,598).

4.8 | Pull-down assay

In vitro expressed and purified GST fusion proteins (GST-JAZ1 and GST) and His fusion protein (His-PHR1) were incubated in binding buffer (50 mM Tris–HCl, pH 7.5, 100 mM NaCl, and .6% Triton X-100) for 4 h at 4°C. Glutathione Sepharose 4B beads were added and incubated for 1 h. After washing five times with binding buffer, precipitated sepharose beads were collected by brief centrifugation (500 g, 15 min) and then resuspended in protein extraction buffer. The proteins were then size fractioned on 10% SDS-PAGE and immunoblotted by anti-GST (1:5000) and anti-His (1:5000) antibodies (MBL; PM013-7, D291-7), respectively.

4.9 | LCI assays

Both the nLUC- and cLUC-fused proteins were coinfiltrated into *N. benthamiana leaves* via Agrobacterium-mediated coinfiltration. The infiltrated plants were incubated for 3 days and examined using the NightSHADE LB985 Plant Imaging System (Berthold).

4.10 | Transient expression assay

Transient expression assays were performed as described previously (Liu et al., 2020). The reporter and effector constructs were transformed into Agrobacterium strain EHA105. The Agrobacterium solutions containing the reporter or effector constructs were coincubated for 2 h and infiltrated into 3- to 4-week-old *N. benthamiana* leaves. Plants were incubated under continuous white light for 3 days after

infiltration. The firefly LUC activity was photographed after spraying with 1 mM luciferin (Goldbio).

4.11 | Physiological measurements

The Pi concentration measurement was performed as described previously (Liu et al., 2017).

4.12 | Statistical analysis

The significance of differences between the mean values was assessed with Student's *t* test using the SPSS software (n.s., not significant; $^{*}P < .05$; $^{**}P < .01$). Standard deviation (±SD) was provided as an error bar to indicate the variations associated with the particular mean values.

AUTHOR CONTRIBUTIONS

Yang Liu and Haiyang Wang designed the experiment. Yang Liu, Yanzhao Sun, Yanyan Zheng, Heng Yao, Zhaodong Ma and Mengwei Xiao performed the experiment. Yanzhao Sun and Yanyan Zheng analyzed data. Yang Liu wrote the article.

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CONFLICT OF INTEREST STATEMENT

The authors have no conflict of interest to declare.

PEER REVIEW

The peer review history for this article is available in the supporting information for this article.

DATA AVAILABILITY STATEMENT

All relevant data are within the paper and its Supporting Information files.

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SUPPORTING INFORMATION

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