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Research Article

Gibberellin signaling modulates flowering via the DELLA–BRAHMA–NF-YC module in Arabidopsis

Chunyu Zhang,[1,†] Mingyang Jian,[1,2,†] Weijun Li,[1,2] Xiani Yao,[1,2] Cuirong Tan,[1,2] Qian Qian,[1] Yilong Hu,[1,2] Xu Liu[1,2] and Xingliang Hou[1,2,*]

1 Guangdong Provincial Key Laboratory of Applied Botany and State Key Laboratory of Plant Diversity and Prominent Crops, South China Botanical Garden, Chinese Academy of Sciences, Guangzhou 510650, China
2 College of Life Sciences, University of the Chinese Academy of Sciences, Beijing 100049, China

*Author for correspondence: houxl@scib.ac.cn
†These authors contributed equally to this work.
The author responsible for distribution of materials integral to the findings presented in this article in accordance with the policy described in the Instructions for Authors (https://academic.oup.com/plcell/) is: Xingliang Hou (houxl@scib.ac.cn).

Abstract

Gibberellin (GA) plays a key role in floral induction by activating the expression of floral integrator genes in plants, but the epigenetic regulatory mechanisms underlying this process remain unclear. Here, we show that BRAHMA (BRM), a core subunit of the chromatin-remodeling SWItch/sucrose nonfermentable (SWI/SNF) complex that functions in various biological processes by regulating gene expression, is involved in GA-signaling-mediated flowering via the formation of the DELLA–BRM–NF-YC module in Arabidopsis (*Arabidopsis thaliana*). DELLA, BRM, and NF-YC transcription factors interact with one another, and DELLA proteins promote the physical interaction between BRM and NF-YC proteins. This impairs the binding of NF-YCs to *SOC1*, a major floral integrator gene, to inhibit flowering. On the other hand, DELLA proteins also facilitate the binding of BRM to *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS1* (*SOC1*). The GA-induced degradation of DELLA proteins disturbs the DELLA–BRM–NF-YC module, prevents BRM from inhibiting NF-YCs, and decreases the DNA-binding ability of BRM, which promote the deposition of H3K4me3 on *SOC1* chromatin, leading to early flowering. Collectively, our findings show that BRM is a key epigenetic partner of DELLA proteins during the floral transition. Moreover, they provide molecular insights into how GA signaling coordinates an epigenetic factor with a transcription factor to regulate the expression of a flowering gene and flowering in plants.

Introduction

In flowering plants, the precise transition from vegetative growth to reproductive development is crucial for successful propagation. The timing of this transition is tightly controlled by environmental cues and intrinsic signals. Six flowering pathways have been identified in Arabidopsis (*Arabidopsis thaliana*) via various genetic and molecular biological studies: the photoperiod, vernalization, thermosensory, autonomous, gibberellin (GA), and age pathways (Michaels 2009; Amasino 2010; Li et al. 2016a; Bao et al. 2020). These pathways converge to regulate the expression of various floral integrator genes, including *FLOWERING LOCUS T* (*FT*), *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS1* (*SOC1*), and *LEAFY* (*LFY*), which subsequently activate several downstream floral meristem identity genes, such as *LFY*, *APETALA1* (*AP1*), and *FRUITFULL* (*FUL*), to initiate the formation of floral meristems (Kardailsky et al. 1999; Blazquez and Weigel 2000; Abe et al. 2005; Wigge et al. 2005; Lee and Lee 2010).

The crucial role of GA in floral induction in Arabidopsis has been extensively studied in the past 2 decades (Wilson et al. 1992; Griffiths et al. 2006; Porri et al. 2012; Wang et al. 2016). Prior to floral initiation, the levels of bioactive GAs increase, which promotes flowering by activating the expression of

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Background: The timing of floral induction is tightly controlled by environmental cues and intrinsic signals. The critical role of gibberellin (GA) in this process has been extensively studied in past decades. DELLA proteins serve as the central regulatory hubs of GA signaling. The mechanism of GA-dependent transcription involves the recruitment of DELLA proteins to transcription factors. In general, epigenetic modifiers are believed to cooperate with transcription factors to regulate gene expression, but how epigenetic regulation participates in GA-dependent transcription of the floral integrator genes in plants remains unclear.

Question: Which epigenetic modifiers participate in GA-dependent transcription of the floral integrator genes? What is the detailed molecular mechanism involved in this process?

Findings: BRAHMA (BRM), a core catalytic subunit of the SWI/SNF-type chromatin-remodeling complex, is involved in GA-signaling-mediated flowering via the formation of the DELLA–BRM–NF-YC module in Arabidopsis. DELLA proteins promote the interaction of BRM with the transcription factor NUCLEAR FACTOR Y-C (NF-YC), impairing the binding of NF-YC to the floral integrator gene *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS1* (*SOC1*), resulting in late flowering. Meanwhile, DELLA proteins accelerate the binding of BRM to *SOC1*. In the presence of GA, GA-triggered DELLA degradation disturbs the DELLA–BRM–NF-YC module and the H3K4me3 level at *SOC1* chromatin increases, resulting in higher gene expression and early flowering.

Next steps: We will investigate whether the BRM–NF-Y module is also responsive to other phytohormone signals and how BRM integrates different phytohormone signals during plant development.

SOC1 and *LFY* in the shoot apex (Eriksson et al. 2006). Bioactive GAs can bind to their receptor GA-INSENSITIVE DWARF 1 (GID1) and, in turn, recruit DELLA proteins (DELLAs) for ubiquitination and degradation. This process, which is mediated by the F-box type E3 ubiquitin ligase SLEEPY 1 (SLY1), modulates plant responses to GAs (Dill et al. 2004; Ueguchi-Tanaka et al. 2005; Willige et al. 2007; Murase et al. 2008). DELLA proteins play pivotal negative roles in the GA signal transduction pathway. *Arabidopsis* contains 5 DELLAs: REPRESSOR OF ga1-3 (RGA), GA-INSENSITIVE (GAI), RGA-LIKE1 (RGL1), RGL2, and RGL3 (Sun and Gubler 2004). DELLAs can interact with epigenetic factors, such as SWI3C and PICKLE (Sarnowska et al. 2013; Zhang et al. 2014; Park et al. 2017), suggesting a connection between GA signaling and the epigenetic regulatory mechanisms. In addition, DELLAs can mediate transcriptional control to repress flowering by interacting with many transcription factors, such as CONSTANS (CO), MYC3, FLOWERING LOCUS C (FLC), SQUAMOSA PROMOTER BINDING-LIKEs (SPLs), WRKY75, bHLH48, bHLH60, and nuclear factor Y (NF-Y; Yu et al. 2012; Hou et al. 2014; Wang et al. 2016; Li et al. 2016b, 2017; Bao et al. 2019).

In eukaryotes, NF-Y complexes, which comprise 3 distinct subunits including NF-YA, NF-YB, and NF-YC, regulate the expression of their target genes by binding to DNA with the central pentamer CCAAT box (Nardini et al. 2013). To date, several NF-YA, NF-YB, and NF-YC transcription factors have been shown to modulate flowering in Arabidopsis (Ben-Naim et al. 2006; Wenkel et al. 2006; Cai et al. 2007; Kumimoto et al. 2008, 2010; Siriwardana et al. 2016). The best understood role of the NF-Y subunits (NF-Ys) during this process is their transcriptional activation of *FT*. Briefly, NF-Y subunits form the canonical NF-YB/NF-YC/NF-YA and non-canonical NF-YB/NF-YC/CO complexes, which bind to the distal enhancer bearing a CCAAT box and the proximal CO-responsive elements (COREs), respectively, in the *FT* promoter. The interaction of the 2 distally separated DNA-bound complexes is stabilized by the formation of a chromatin loop (Cao et al. 2014; Gnesutta et al. 2017a, b; Myers and Holt 2018). Additionally, NF-Ys can recruit different transcription factors and histone modifiers to regulate flowering (Liu et al. 2018; Luo et al. 2018; Myers and Holt 2018; Hwang et al. 2019; Li et al. 2021). We previously demonstrated that NF-Ys, acting as flowering activators, are sequestered from binding to *SOC1* by interacting with DELLAs, which results in the downregulation of *SOC1* expression and the repression of flowering (Hou et al. 2014). Although the molecular and genetic relationships between NF-Ys and DELLAs have been characterized in recent years (Hou et al. 2014; Liu et al. 2016; Hu et al. 2018), the detailed molecular mechanism underlying how DELLAs inhibit NF-Y activity during flowering remains unclear.

Chromatin-remodeling complexes (CRCs) play key roles in transcriptional regulation in eukaryotes. SWItch/sucrose non-fermentable (SWI/SNF) complexes are the best studied ATP-dependent CRCs, which utilize the energy derived from ATP hydrolysis to regulate the interaction between histones and DNA (Clapier and Cairns 2009; Clapier et al. 2017). BRAHMA (BRM) is a key catalytic subunit in the SWI/SNF complex that serves as an important regulator of the growth and development of Arabidopsis by modifying the expression of its target genes (Farrona et al. 2004, 2011; Hurtado et al. 2006; Kwon et al. 2006; Tang et al. 2008; Han et al. 2012; Wu et al. 2012; Vercruyssen et al. 2014; Li et al. 2015, 2022;

Yang et al. 2015; Zhang et al. 2017). Several studies over the past 2 decades have revealed the role of BRM in regulating flowering. BRM negatively regulates flowering by repressing the expression of *CO*, *FT*, and *SOC1* (Farrona et al. 2004, 2011; Hurtado et al. 2006). BRM also directly activates the expression of *SHORT VEGETATIVE PHASE* (*SVP*) and *TARGET OF FLC AND SVP1* (*TFS1*) by altering chromatin modifications at these loci (Li et al. 2015; Richter et al. 2019). Although recent studies have shown that BRM associates with transcription factors to regulate flowering (Richter et al. 2019; Yang et al. 2022), the relationship between BRM and NF-Ys in this process remains unclear.

In this study, we show that BRM is involved in GA-signaling-mediated flowering by forming a GA-sensitive module with DELLAs and NF-YCs. Specifically, the interaction of BRM with NF-YCs inhibits its binding to *SOC1*, a major floral integrator gene, and the interaction between BRM with NF-YCs can be significantly promoted by DELLAs. Meanwhile, DELLAs also promote the binding of BRM to *SOC1*. Both mechanisms decrease the deposition of H3K4me3 at *SOC1* chromatin, resulting in reduced *SOC1* gene expression and late flowering. GA triggers the degradation of DELLAs to release the repression of BRM on NF-YC activity and *SOC1* transcription, thus promoting early flowering. These findings provide important insights into the epigenetic regulatory mechanism by which GA signaling accelerates flowering via BRM in plants.

Results

DELLAs physically interact with BRM

DELLAs are important repressors of the GA response that physically interact with other proteins to control flowering (Bao et al. 2020). However, few epigenetic factors associated with DELLAs that function in this process have been identified. To investigate potential epigenetic partners of DELLAs, we performed a series of yeast 2-hybrid assays. BRM, a core subunit of the SWI/SNF complex, strongly interacted with all 5 Arabidopsis DELLAs in yeast (Fig. 1, A and B; Supplemental Fig. S1). Region mapping assays showed that RGA^N (amino acids 1 to 199) and BRM^N (amino acids 1 to 952) were necessary and sufficient for the interaction of RGA with BRM (Fig. 1, A, C, and D). The N-terminal region of RGA (RGA^N) contains a conserved DELLA domain (Zentella et al. 2016), and the N-terminal region of BRM (BRM^N) normally serves as a docking site for the recruitment of transcription factors (Peirats-Llobet et al. 2016). We thus used BRM^N instead of the full-length BRM protein in subsequent experiments. In vitro pull-down assays revealed that His-DELLAs were successfully pulled down by GST-BRM^N but not by the GST control, suggesting that BRM^N physically interacts with DELLA proteins in vitro (Fig. 1E; Supplemental Fig. S2).

To further test the interaction between DELLAs and BRM in vivo, we used RGA as the representative DELLA homolog in subsequent analyses. We first performed split luciferase (split-LUC) complementation assays in *Nicotiana benthamiana* leaves. A strong interaction signal was observed when BRM^N-nLUC and cLUC-RGA were co-expressed, whereas there was no signal in the negative controls (Fig. 1F). Next, we generated double transgenic Arabidopsis plants harboring *35S:RGA-FLAG pBRM:BRM-GFP* (*RGA-FLAG BRM-GFP*) to perform a co-immunoprecipitation (co-IP) assay. RGA-FLAG co-immunoprecipitated with BRM-GFP (Fig. 1G), which confirms the interaction between RGA and BRM in Arabidopsis. These findings indicate that BRM interacts with DELLA proteins both in vitro and in vivo.

DELLAs regulate GA-mediated flowering via BRM

The role of BRM in flowering control was previously reported (Farrona et al. 2004; Li et al. 2015; Yang et al. 2022). In light of the DELLA–BRM interaction identified in this study, we speculated that these 2 proteins might function together to control flowering. Consistent with previous studies (Yan et al. 2020; Yang et al. 2022), we determined that both DELLAs and BRM negatively regulate flowering. Remarkably, the *brm-3* mutant completely or partially restored the late-flowering phenotype of *35S:RGA-FLAG* or *pRGA:RGA⊿17* (GA-insensitive form of *RGA*; Yu et al. 2012) plants, respectively (Fig. 2, A and B; Supplemental Fig. S3, A and B). The quadruple mutant (*dellaq*) of *RGA*, *GAI*, *RGL1*, and *RGL2* exhibited an early flowering phenotype, as expected, and the *brm-3 dellaq* mutant displayed a similar flowering phenotype to either the *brm-3* or *dellaq* mutant (Fig. 2, C and D). Furthermore, the *brm-3* mutant significantly rescued the late-flowering phenotype of *ga1* (Fig. 2, E and F), a GA-deficient mutant in which DELLA proteins highly accumulate. These observations suggest that *BRM* may be epistatic to *DELLA* genes during flowering.

The flowering genes *SOC1* and *FT* contribute to the regulation of GA-mediated flowering (Bao et al. 2020). We thereby investigated whether DELLAs and BRM co-regulate the expression of these 2 flowering genes by reverse transcription quantitative PCR (RT-qPCR). Consistent with the flowering phenotypes, the transcript levels of *SOC1* and *FT* were markedly altered in *brm-3*, *35S:RGA-FLAG*, *pRGA:RGA⊿17*, and *ga1* plants compared with those in Col-0, and the loss of *BRM* function resulted in the substantial de-repression of *SOC1* and *FT* expression in *35S:RGA-FLAG*, *pRGA:RGA⊿17*, and *ga1* plants (Fig. 2G; Supplemental Figs. S3C and S4). The expression of *SOC1* in the *brm-3 dellaq* mutant was not examined because no seeds from the homozygous *brm-3 dellaq* mutant plants could be obtained due to its abnormal fertility. We thus treated the *brm-3* mutant with GA instead of the *brm-3 dellaq* mutant prior to RT-qPCR. GA treatment did not further promote the expression of *SOC1* and *FT* in the *brm-3* background, even though GA treatment significantly increased their expression in Col-0 plants (Fig. 2G; Supplemental Fig. S4), which is also consistent with the flowering phenotypes of the *brm-3* and *brm-3 dellaq* mutants (Fig. 2, C and D).

Chromatin immunoprecipitation (ChIP) assays showed that BRM was enriched at the chromatin of *SOC1* but not

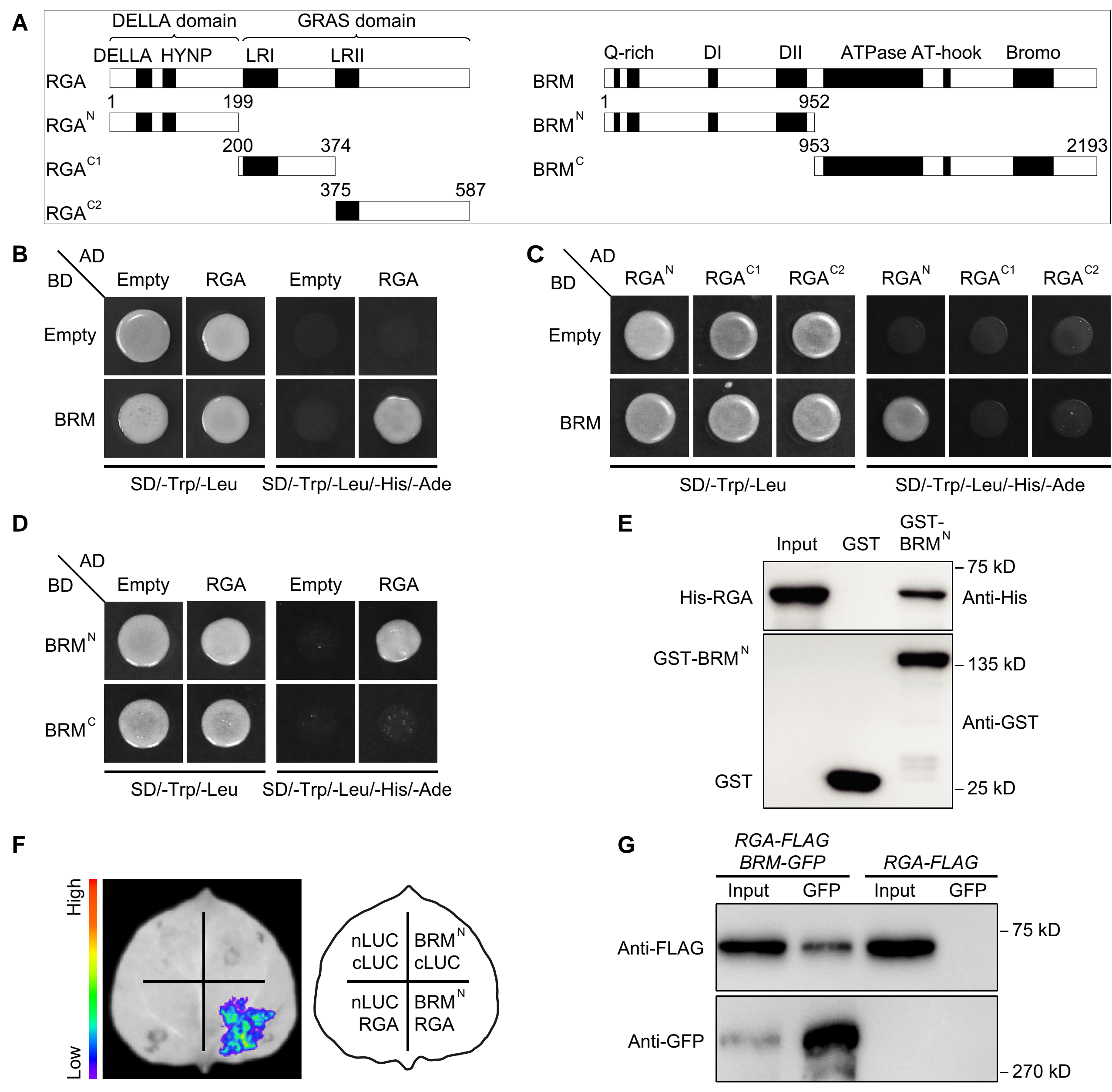

Figure 1. RGA physically interacts with BRM in vitro and in vivo. **A)** Schematic diagram of full-length RGA, BRM and their truncated derivatives used in the yeast 2-hybrid assay. The conserved domains are marked and the amino acid positions of these derivatives are numbered. **B–D)** Yeast 2-hybrid assays showing the interactions between RGA and BRM. Transformed yeast cells were grown on SD/-Trp/-Leu medium or SD/-Trp/-Leu/-His/-Ade medium. BD, GAL4 DNA-binding domain; AD, GAL4 DNA-activation domain. **E)** Pull-down assay showing direct interactions between His-RGA and GST-BRM^{N} fusion proteins in vitro. His-RGA protein was incubated with immobilized GST-BRM^{N} or GST protein. The pulled down proteins were detected by anti-His or anti-GST antibody. **F)** Split-LUC complementation imaging assay showing the interaction of RGA and BRM^{N} in *N. benthamiana* cells. **G)** Co-IP assay showing the interaction of RGA and BRM in *Arabidopsis*. Plant extracts from 9-d-old *35S:RGA-FLAG pBRM:BRM-GFP* (*RGA-FLAG BRM-GFP*) or *RGA-FLAG* seedlings under long-day conditions (LDs) were immunoprecipitated by using a GFP trap. The precipitated proteins were detected by anti-FLAG or anti-GFP antibody.

FT (Supplemental Fig. S5). We thus asked whether the DELLA–BRM module co-regulates flowering by altering the expression of *SOC1*. Transient expression assays in *N. benthamiana* leaves revealed that BRM or RGA decreased the activity of LUC driven by the *SOC1* promoter, and the co-expression of BRM and RGA substantially increased the inhibition of LUC driven by the *SOC1* promoter (Fig. 2H), suggesting that the DELLA–BRM module negatively regulates the expression of *SOC1*.

DELLAs can affect the binding of transcription factors to their target genes (Hou et al. 2014; Liu et al. 2016; Hu et al. 2018). To explore how the DELLA–BRM module regulates *SOC1* expression, we performed ChIP assays to determine whether DELLAs affect the association of BRM to the

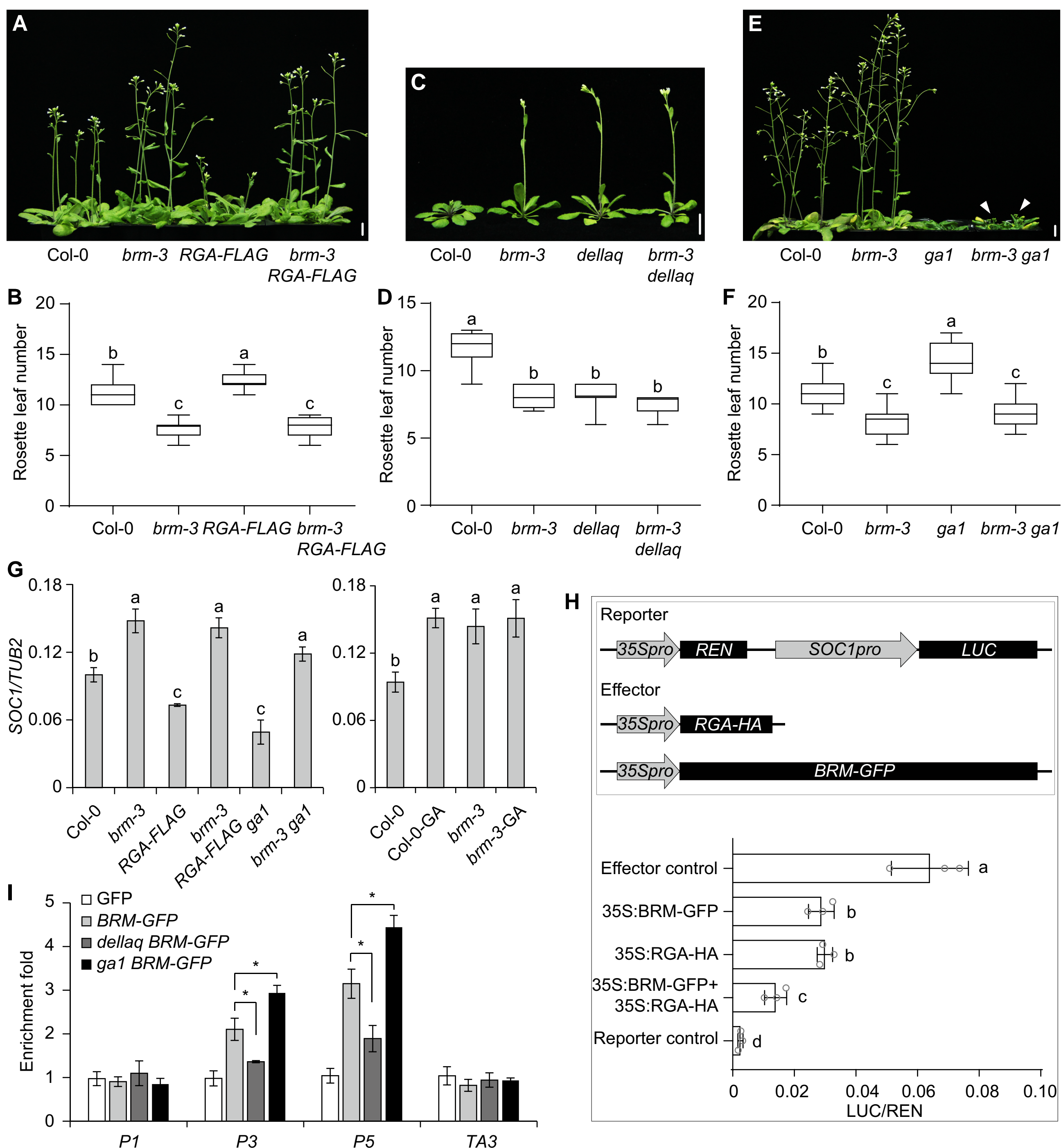

Figure 2. BRM is epistatic to DELLAs in regulating flowering time. **A and B)** Flowering phenotypes of Col-0, *brm-3*, *35S:RGA-FLAG* (*RGA-FLAG*), and *brm-3 RGA-FLAG*. **C and D)** Flowering phenotypes of Col-0, *brm-3*, *dellaq*, and *brm-3 dellaq*. **E and F)** Flowering phenotypes of Col-0, *brm-3*, *ga1*, and *brm-3 ga1*. White arrowheads show flowering. Scale bars in **A**), **C**), and **E**), 1 cm. Values in **B**), **D**), and **F**) are shown as boxplots, with the box representing the interquartile range, the central line indicating the median, and the whiskers showing the minimum or maximum value ($n \geq 20$). Different lowercase letters indicate statistically significant differences (1-way ANOVA, $P < 0.01$). **G)** RT-qPCR analysis of *SOC1* expression in 9-d-old Col-0, *brm-3*, *RGA-FLAG*, *brm-3 RGA-FLAG*, *ga1*, *brm-3 ga1*, GA-treated Col-0 (Col-0-GA), and GA-treated *brm-3* (*brm-3*-GA) seedlings. For the Col-0-GA and *brm-3*-GA samples, 8-d-old Col-0 and *brm-3* seedlings treated with 100 μM GA_3 for 24 h were collected. *TUB2* was used as an internal control. **H)** Transient expression assay indicating that the expression of *SOC1* is co-regulated by BRM and RGA. **I)** ChIP analysis of BRM-GFP binding to the *SOC1* locus in Col-0, *dellaq*, and *ga1* mutant. Nine-day-old Col-0, *p35S:GFP* (*GFP*), *BRM-GFP*, *dellaq BRM-GFP*, and *ga1 BRM-GFP* seedlings were collected for ChIP assay. All plants used for analyses were grown at 22 °C under LDs. The enrichment of a TA3 genomic fragment was used as the negative control. Values in **G**), **H**), and **I**) represent means ± SD of 3 independent experiments. Different lowercase letters indicate statistically significant differences (1-way ANOVA, $P < 0.01$). Asterisks indicate significant differences between the selected samples (*$P < 0.01$, Student's *t*-test).

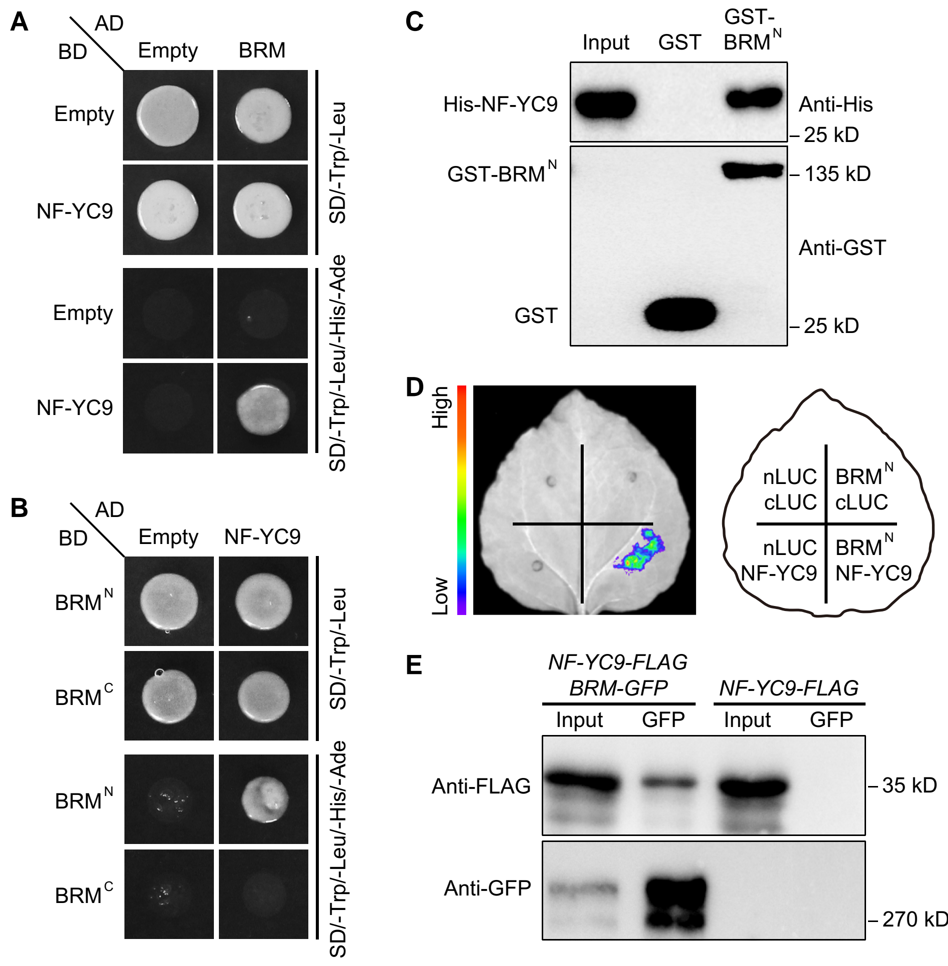

Figure 3. BRM physically interacts with NF-YC9 in vitro and in vivo. **A)** Yeast 2-hybrid assay showing the interactions between BRM and NF-YC9. **B)** Yeast 2-hybrid assay showing that BRM^N directly interacts with NF-YC9. Transformed yeast cells were grown on SD/-Trp/-Leu medium or SD/-Trp/-Leu/-His/-Ade medium. **C)** Pull-down assay showing direct interactions between GST- BRM^N and His-NF-YC9 fusion proteins in vitro. His-NF-YC9 protein was incubated with immobilized GST- BRM^N or GST protein. The pulled down proteins were detected by anti-His or anti-GST antibody. **D)** Split-LUC complementation imaging assay showing the interaction of BRM^N with NF-YC9 in *N. benthamiana* cells. **E)** Co-IP assay showing the interaction of NF-YC9 and BRM in *Arabidopsis*. Plant extracts from 9-d-old *nf-yc9-1 pNF-YC9:NF-YC9-FLAG pBRM:BRM-GFP* (*NF-YC9-FLAG BRM-GFP*) or *NF-YC9-FLAG* seedlings under LDs were immunoprecipitated by GFP trap. The precipitated proteins were detected by anti-FLAG or anti-GFP antibody.

SOC1 promoter. The enrichment of BRM at *SOC1* chromatin was drastically reduced in the *dellaq* mutant but increased in the *ga1* mutant background (Fig. 2I). However, the level of BRM or RGA protein was not altered in the *dellaq/ga1* or *brm-3* mutant compared with Col-0 plants, respectively (Supplemental Figs. S6 and S7), indicating that DELLAs enhance the binding of BRM to *SOC1* rather than altering the protein level of BRM during flowering. Consistent with this observation, GA significantly reduced the binding of BRM-GFP to *SOC1*, whereas paclobutrazol (PAC), an inhibitor of GA biosynthesis, increased the binding of BRM-GFP to *SOC1*, even when the protein levels of BRM-GFP were not altered by these treatments (Supplemental Fig. S8). Overall, these findings suggest that DELLAs regulate the expression of *SOC1* via BRM to control flowering.

BRM physically interacts with NF-YCs

We previously showed that DELLAs function together with NF-Y subunits to control flowering in the GA pathway (Hou et al. 2014). Considering that DELLAs also interact with BRM, we investigated the potential biological relationships among these proteins. NF-YC3, NF-YC4, and NF-YC9 have redundant functions in regulating flowering (Kumimoto et al. 2010; Liu et al. 2018). All 3 of these proteins interacted with BRM in yeast (Fig. 3A; Supplemental Fig. S9A). Region mapping assays revealed that BRM^N, but not BRM^C, mediates interactions with NF-YCs (Fig. 3B; Supplemental Fig. S9B). In in vitro pull-down assays, His-NF-YC3, His-NF-YC4, and His-NF-YC9 could be pulled down by GST-BRM^N but not by the GST control, suggesting that BRM^N physically interacts with NF-YCs in vitro (Fig. 3C; Supplemental Fig. S10).

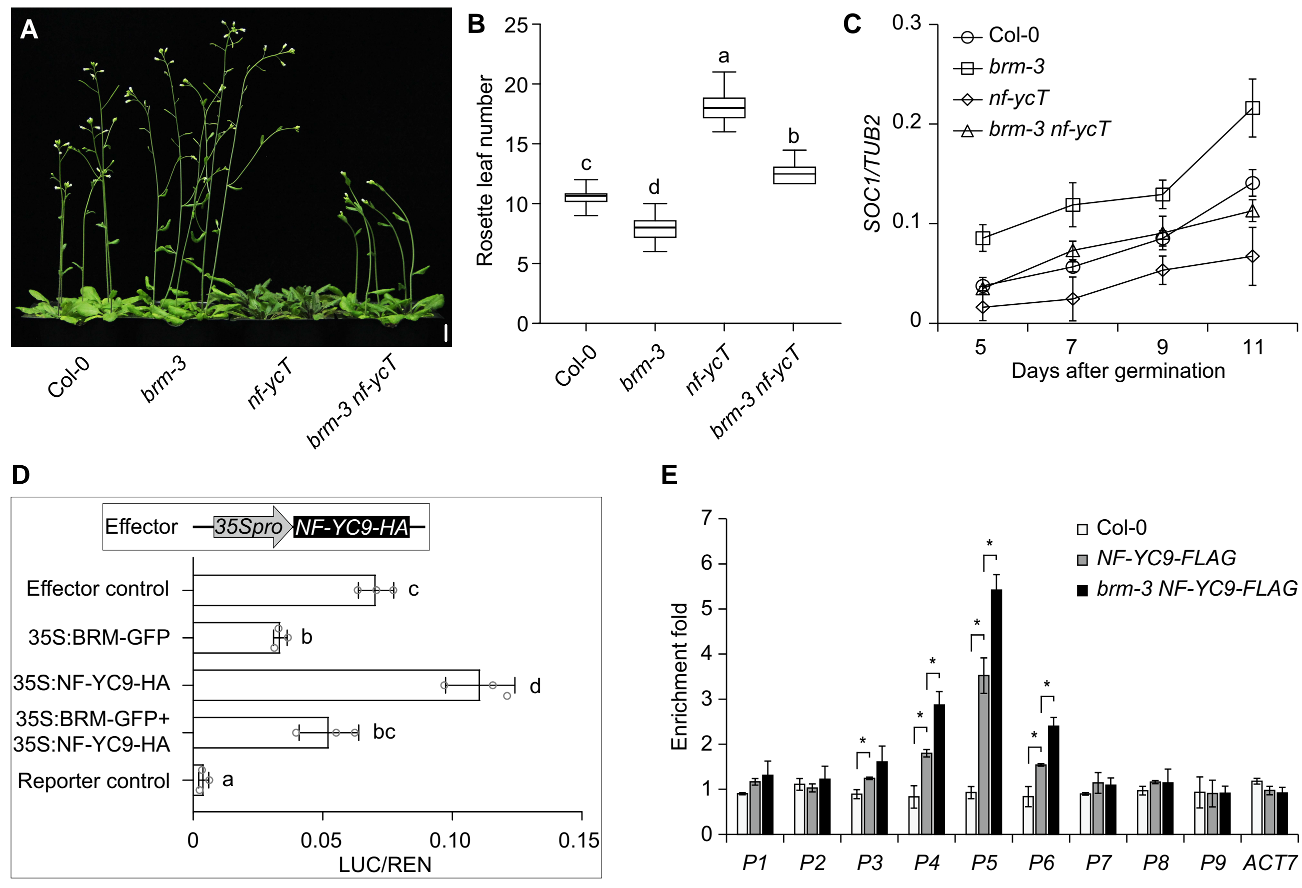

Figure 4. NF-YCs and BRM antagonistically regulate flowering by directly modulating *SOC1* expression. **A and B)** Flowering phenotypes of Col-0, *brm-3*, *nf-ycT*, and *brm-3 nf-ycT*. Scale bar, 1 cm. Values in **B)** are shown as boxplots, with the box representing the interquartile range, the central line indicating the median, and the whiskers showing the minimum or maximum value ($n \geq 20$). **C)** RT-qPCR analysis of *SOC1* expression in developing Col-0, *brm-3*, *nf-ycT*, and *brm-3 nf-ycT* seedlings. *TUB2* was used as an internal control. **D)** Transient expression assay indicating that the expression of *SOC1* is regulated by NF-YC9 and BRM. Either the reporter or the relevant empty vector (Reporter control) was co-transformed with the effector or the relevant empty vector (Effector control) into *N. benthamiana* leaves. *SOC1* promoter activity was calculated as the ratio of LUC to REN. **E)** ChIP analysis of NF-YC9-FLAG binding to the *SOC1* locus. Nine-day-old Col-0, *NF-YC9-FLAG*, and *brm-3 NF-YC9-FLAG* seedlings were collected for ChIP assay. The enrichment of a *ACT7* genomic fragment was used as the negative control. All plants used for analysis were grown at 22 °C under LDs. Values in **C)**, **D)**, and **E)** represent means ± SD of 3 independent experiments. Different lowercase letters indicate statistically significant differences (1-way ANOVA, $P < 0.01$). Asterisks indicate significant differences between the selected samples (*$P < 0.01$, Student's *t*-test).

We next chose NF-YC9 as the representative of NF-YCs to investigate the interaction between BRM and NF-YCs in planta. Split-LUC complementation assays revealed a strong signal in *N. benthamiana* leaves when BRM^{N}-nLUC and cLUC-NF-YC9 were co-expressed; however, no such signal was observed in the negative controls (Fig. 3D). Furthermore, a co-IP assay using *nf-yc9-1 pNF-YC9:NF-YC9-FLAG pBRM:BRM-GFP* (*NF-YC9-FLAG BRM-GFP*) Arabidopsis plants also showed that NF-YC9-FLAG protein could be co-immunoprecipitated with BRM-GFP (Fig. 3E). Taken together, these findings suggest that BRM interacts with NF-YCs both in vitro and in vivo.

BRM genetically interacts with NF-YCs during *SOC1*-mediated flowering

To clarify the biological function of the BRM–NF-YC interaction, we evaluated the genetic relationship between BRM and NF-YCs during flowering. Consistent with previous findings, *brm-3* and *nf-ycT* (*nf-yc3/4/9* triple mutant) plants showed early and late flowering, respectively (Liu et al. 2018; Yang et al. 2022). Notably, the quadruple *brm-3 nf-ycT* mutant flowered significantly earlier than the *nf-ycT* mutant and later than the *brm-3* mutant (Fig. 4, A and B), suggesting that BRM and NF-YCs might antagonistically regulate flowering time. Consistently, the expression of *SOC1* and *FT* was significantly higher in *brm-3 nf-ycT* than in *nf-ycT* but lower than in *brm-3* (Fig. 4C; Supplemental Fig. S11). We also examined *SOC1* promoter activity regulated by BRM and NF-YC9 using a dual-luciferase reporter assay in *N. benthamiana* leaves (Fig. 4D). The transient assay revealed that LUC activity was enhanced by NF-YC9; in contrast, LUC activity was inhibited by BRM (Fig. 4D). These findings suggest that BRM and NF-YCs antagonistically regulate the expression of *SOC1*.

To characterize the opposite functions of BRM and NF-YCs in regulating the expression of *SOC1*, we performed ChIP assays using *brm-3 nf-yc9-1 pNF-YC9:NF-YC9-FLAG* (*brm-3 NF-YC9-FLAG*) plants. The enrichment of NF-YC9 on *SOC1* rather than *FT* chromatin was higher in the *brm-3* vs. the Col-0 background (Fig. 4E; Supplemental Fig. S12A). Given that the level of NF-YC9 protein was not affected by the loss of function of *BRM* (Supplemental Fig. S12B), the greater enrichment of NF-YC9 on *SOC1* chromatin stems from the lack of BRM inhibition rather than alterations in the level of NF-YC9 protein.

Since both BRM and NF-YCs were shown to regulate the expression of genes via histone modifications (Hou et al. 2014; Yang et al. 2022), we examined the deposition of H3K4me3 and H3K27me3, key marks involved in transcriptional modulation at *SOC1* chromatin. BRM and NF-YCs had opposite effects on the levels of H3K4me3 at the *SOC1* locus, and these effects were abolished in the *brm-3 nf-ycT* mutant. In contrast, H3K27me3 deposition was only mediated by NF-YCs but not by BRM (Supplemental Fig. S13). Taken together, these results suggest that BRM regulates flowering time by antagonizing NF-YC activity via the deposition of H3K4me3 on *SOC1* chromatin.

DELLA proteins enhance the interaction of BRM with NF-YCs

In light of the finding that DELLAs interact with both BRM and NF-YCs, we determined whether these proteins function together to regulate GA-mediated flowering. Among the 5 DELLAs in Arabidopsis, RGA and GAI play major roles in this process (Tyler et al. 2004); we thus selected these 2 DELLAs for further analyses. Yeast 3-hybrid assays revealed that the interaction between BRM and NF-YC9 was strengthened in the presence of RGA or GAI (Fig. 5, A and B; Supplemental Fig. S14, A and B). Split-LUC complementation assays also showed that the co-expression of RGA or GAI with BRM and NF-YC9 enhanced LUC activity compared with co-expression exclusively with BFP (Fig. 5, C and D; Supplemental Fig. S14, C and D), even when the protein level of BFP was markedly higher than that of RGA or GAI (Fig. 5E; Supplemental Fig. S14E). These results suggest that DELLAs increase the strength of the interaction between BRM and NF-YC9 in vivo. To confirm these findings, we verified the effect of RGA on the physical interaction between BRM and NF-YC9 via co-IP. We generated transgenic plants co-expressing *NF-YC9-FLAG* and *BRM-GFP* in the Col-0 and *35S:RGA-HA* (*RGA-HA*) backgrounds and found that more NF-YC9-FLAG coimmunoprecipitated with BRM-GFP in the *RGA-HA* background than in the Col-0 background (Fig. 5, F and G; Supplemental Fig. S15).

DELLA proteins, the key repressors in GA signaling, regulate the expression of *SOC1* by inhibiting the binding of NF-Y to *SOC1* (Hou et al. 2014). This observation, coupled with the results of the current study, suggests that DELLAs might recruit BRM to inhibit the binding of NF-Y to *SOC1* by enhancing the physical interaction of BRM with NF-YCs. To test this hypothesis, we treated *NF-YC9-FLAG* and *brm-3 NF-YC9-FLAG* seedlings with GA or PAC and performed ChIP assays to evaluate the binding ability of NF-YC9 to *SOC1* chromatin. As expected, GA or PAC treatment had a significant effect on the binding of NF-YC9 to the *SOC1* promoter in the Col-0 background. However, the loss-of-function mutation of *BRM* abolished this effect (Fig. 5H), indicating that BRM is essential for the effect of GA signaling on the binding of NF-YC9 to *SOC1*. Consistent with this finding, the flowering of *brm-3* plants was not responsive to GA treatment, even though GA treatment significantly accelerated the flowering of Col-0 plants (Fig. 5, I and J). Overall, our findings demonstrate that DELLA proteins facilitate the formation of the DELLA–BRM–NF-YC module, which is essential for GA-mediated flowering.

Discussion

GA signaling regulates flowering by inducing the degradation of DELLA proteins, which are the key repressors of the GA-signaling pathway (Sun 2008). DELLA proteins physically interact with a series of transcription factors and modulate their transcriptional activities on their target genes to regulate flowering (Bao et al. 2020). For instance, DELLAs prevent NF-Y from binding to the *SOC1* locus via protein–protein interactions, which inhibits flowering (Hou et al. 2014). However, the molecular mechanisms underlying the ability of DELLAs to mediate the epigenetic regulatory effects of NF-Y on gene expression remain unclear.

Here, we show that DELLA proteins can recruit a catalytic subunit of the SWI/SNF complex, BRM, to inhibit the binding of NF-YCs to *SOC1*. In addition, DELLAs promote the binding of BRM to the *SOC1* locus during GA-signaling-mediated flowering. In the presence of GA, DELLA proteins are degraded, which reduces the binding of BRM to chromatin and the strength of the interaction between BRM and NF-YCs, thus activating the expression of *SOC1* and promoting flowering (Fig. 6). Notably, the expression of *FT* is also regulated by BRM, but it does not affect the binding strength of NF-YC9 to *FT* chromatin, suggesting that the DELLA–BRM–NF-YC module may not occur on the *FT* locus. BRM represses *FT* expression by directly altering the expression of its upstream repressor *SVP* (Lee et al. 2007), which is a direct target of BRM (Li et al. 2015). The differential regulation of *SOC1* and *FT* suggests that the formation of the DELLA–BRM–NF-YC module may have spatial and temporal characteristics. Further studies on where and when the heterotrimer module functions in plants should help address this interesting issue.

Several recent reports provide structural insights into how SWI/SNF complexes recognize and remodel nucleosomes in humans and yeast (Han et al. 2020; He et al. 2020; Mashtalir et al. 2020), illustrating the detailed role of SWI/SNF complexes in gene transcription. However, no evidence supports the function of the ATPases in these complexes (such as BRM in plants) in DNA recognition. Three

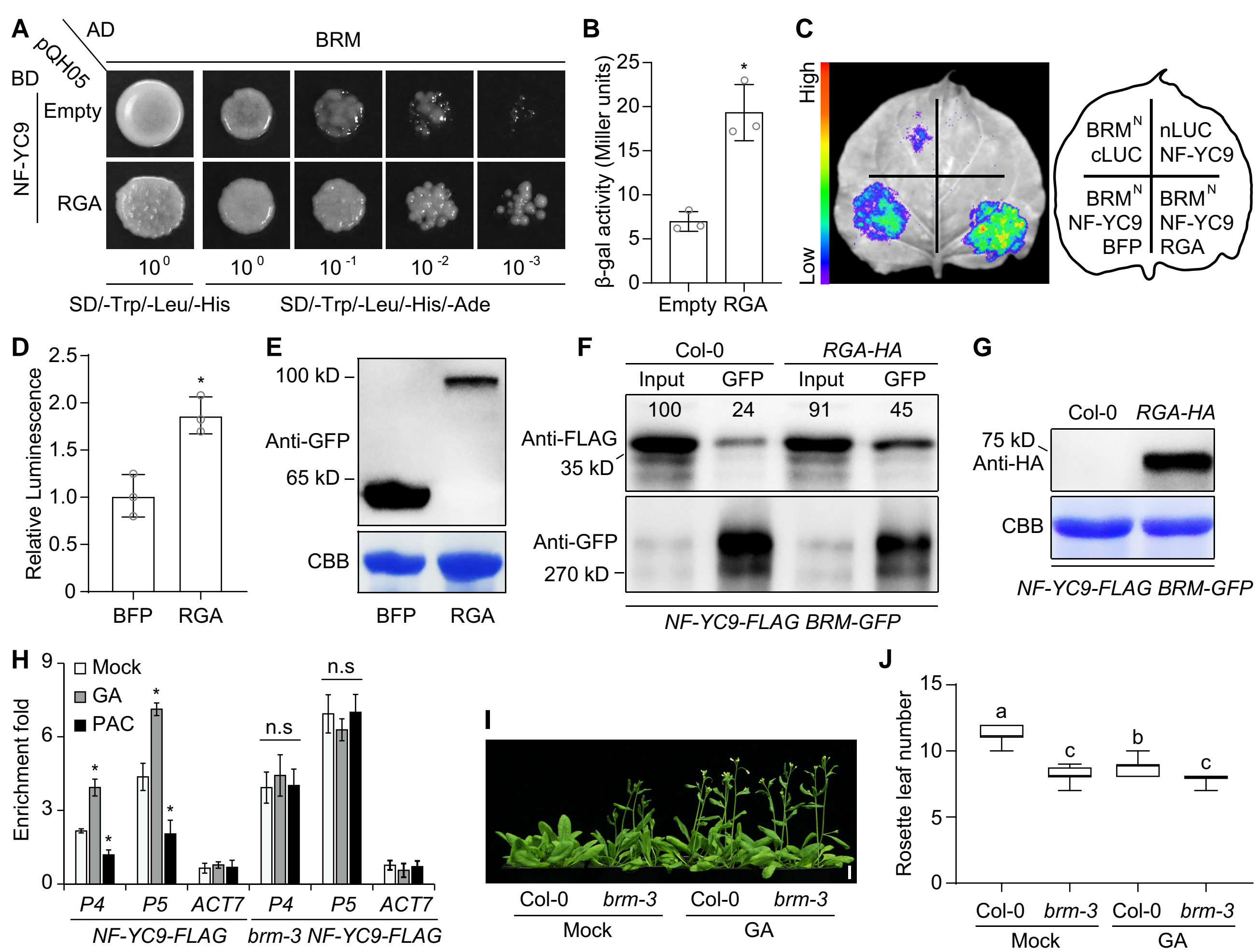

Figure 5. RGA enhances the interaction of NF-YC9 with BRM. **A)** Yeast 3-hybrid assay showing the enhanced interaction between NF-YC9 and BRM in the presence of RGA. Transformed yeast cells were grown on SD/-Trp/-Leu/-His medium or SD/-Trp/-Leu/-His/-Ade medium. **B)** Quantitative yeast 3-hybrid assay defining the strength of the interaction in **A)**. **C and D)** Split-LUC complementation imaging assay showing the enhanced interaction of NF-YC9 and BRM^N by RGA in *N. benthamiana* cells **C)**, and the quantification of luciferase activity **D)**. **E)** The protein levels of BFP-GFP and RGA-GFP in *N. benthamiana* cells determined by probing with anti-GFP antibody. **F)** Co-IP assay showing that RGA promote the interaction of NF-YC9 and BRM in *Arabidopsis*. Plant extracts from 9-d-old *NF-YC9-FLAG BRM-GFP 35S: RGA-HA* (*RGA-HA*) or *NF-YC9-FLAG BRM-GFP* (Col-0) seedlings under LDs were immunoprecipitated by GFP trap. The precipitated proteins were detected by either anti-FLAG or anti-GFP antibody. The protein levels of NF-YC9-FLAG were analyzed using Image J and presented as numbers at the top. **G)** Protein expression analysis of RGA-HA in *NF-YC9-FLAG BRM-GFP* and *NF-YC9-FLAG BRM-GFP RGA-HA* lines. The immunoblot was probed with anti-HA antibody (Sigma, H3663). The bottom gels in **E)** and **G)** were stained with Coomassie Brilliant Blue as loading controls. **H)** ChIP analysis of NF-YC9-FLAG binding to the *SOC1* locus in Col-0 and *brm-3* backgrounds mock treated or treated with 100 μM GA_3 or 10 μM PAC for 24 h. Nine-day-old *NF-YC9-FLAG* and *brm-3 NF-YC9-FLAG* seedlings under LDs were collected for ChIP assay. The enrichment of a *TA3* genomic fragment was used as the negative control. Values in **B)**, **D)**, and **H)** represent means ± SD of 3 independent experiments. n.s., no significance (Student's *t*-test). Asterisks in **B)**, **D)**, and **H)** indicate significant differences (*$P < 0.01$, Student's *t*-test). **I and J)** Flowering phenotypes of Col-0 and *brm-3* grown at 22 °C under LDs treated with mock or 100 μM GA_3 once every 2 d until bolting. Scale bar, 1 cm. Values in **J)** are shown as boxplots, with the box representing the interquartile range, the central line indicating the median, and the whiskers showing the minimum or maximum value ($n \geq 20$). Different lowercase letters indicate statistically significant differences (1-way ANOVA, $P < 0.01$).

DNA-binding factors are required for the binding of BRM to its targets (Li et al. 2016; Zhang et al. 2017; Yang et al. 2022). Among these, GNC is a GATA transcription factor that mediates the recruitment of BRM to *SOC1* to regulate flowering. It is thus possible that GNC or other transcription factors recruit the DELLA–BRM module to *SOC1* (Fig. 6). However, we cannot exclude the possibility that other NF-YCs besides those examined in our study mediate the recruitment of BRM to *SOC1*, since additional NF-YC family members in plants evolved to allow subtle adjustments to many different environmental conditions (Petroni et al. 2012). The crystal structure revealed that the NF-YC/NF-YB dimer coordinates with NF-YA or the CCT domain to specifically target DNA sequences (Gnesutta et al. 2017a, b; Shen et al. 2020).

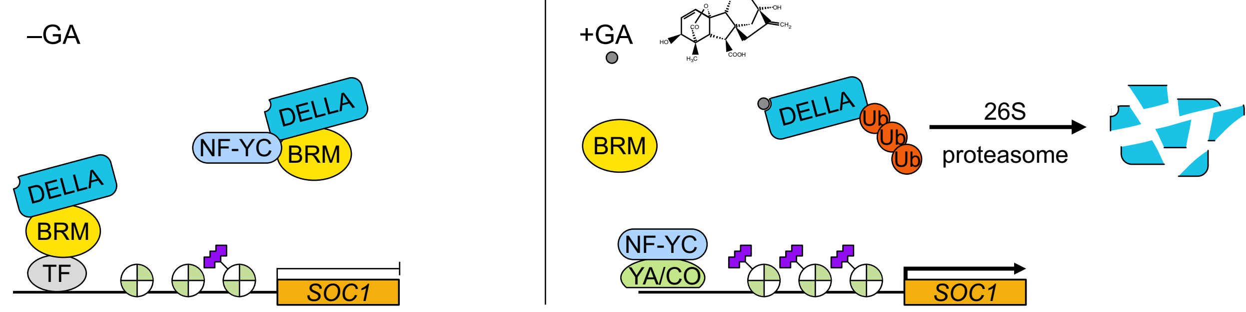

Figure 6. A proposed model illustrating how the DELLA–BRM–NF-YC module regulates GA-signaling-mediated flowering. In the absence of GA, DELLA proteins accumulate and promote the formation of the DELLA–BRM–NF-YC module, thereby inhibiting the binding of NF-YCs to the *SOC1* locus by BRM. At the same time, DELLA proteins promote BRM binding to *SOC1*, thereby inhibiting the expression of *SOC1* by decreasing the deposition of H3K4me3 at *SOC1* chromatin, resulting in late flowering. In the presence of GA, DELLA proteins are ubiquitinated and degraded by the 26S proteasome. This releases the inhibition of NF-YCs by disturbing the DELLA–BRM–NF-YC module and reduces the binding of BRM to *SOC1*, which enhances the expression of *SOC1* by increasing the deposition of H3K4me3 at *SOC1* chromatin, resulting in early flowering. TF, transcription factor; YA, NF-YA; CO, CONSTANS. One (No GA) and 3 (GA) purple squares represent higher and lower H3K4me3 levels, respectively.

Therefore, NF-YC/NF-YB/NF-YA or a CCT domain-containing protein that represses flowering might also be involved in this process, but not CO, which functions as a positive regulator of flowering (Putterill et al. 1995).

BRM is a core SWI/SNF chromatin-remodeling ATPase that uses the energy from ATP hydrolysis to alter the position and occupancy of nucleosomes, leading to either the activation or repression of gene transcription (Clapier et al. 2017). BRM is involved in regulating several plant hormone pathways, such as the abscisic acid, auxin, cytokinin, and GA pathways (Han et al. 2012; Archacki et al. 2013; Efroni et al. 2013; Wu et al. 2015; Peirats-Llobet et al. 2016; Vain et al. 2019). In the GA pathway, BRM affects GA biosynthesis and signaling by regulating the expression of a large of GA-responsive genes (Archacki et al. 2013); however, the precise mechanism underlying how BRM regulates in GA signaling remains unclear. In the current study, we determined that BRM interacts with the GA-signaling hubs DELLA proteins and NF-YC transcription factors to form the DELLA–BRM–NF-YC module to inhibit the effects of NF-YCs. On the other hand, the binding of BRM to the chromatin of flowering-related genes can be accelerated by DELLA proteins. These mechanisms underlie the ability of BRM to regulate the expression of flowering genes and flowering via the GA-signaling pathway. These findings also demonstrate that BRM fine-tunes GA responses at multiple levels.

Many proteins mediate the GA-signaling pathway by physically interacting with DELLA proteins. In general, interactions between DELLAs and transcription factors regulate the transcriptional activities or binding of transcription factors to their target genes (Yu et al. 2012; Fukazawa et al. 2014; Hou et al. 2014; Liu et al. 2016; Wang et al. 2016; Xu et al. 2016; Li et al. 2016b, 2017; Zhang et al. 2018; Bao et al. 2020). DELLAs can also disrupt the interaction of CO with NF-YB2 by the interactions among DELLAs, CO, and NF-YB2 (Xu et al. 2016) or compete with MYC2 to interact with JASMONATE-ZIM1 (JAZ1; Hou et al. 2010). These findings indicate that DELLA proteins modulate the functions of different protein partners. Indeed, our findings suggest that DELLAs enhance the association of BRM with NF-YCs to prevent NF-YCs from binding to the promoter of *SOC1*. Furthermore, DELLAs can promote BRM binding to the *SOC1* promoter during flowering. These results also support the notion that the DELLA-mediated effects on GA signaling might partially depend on the interactions of DELLA proteins with CRCs (Sarnowska et al. 2013). Additional studies of the interactions between DELLAs and other proteins are needed to increase our understanding of the functions of DELLA proteins.

The epigenetic regulatory effects of the subunits of the NF-Y complex on plant growth and development have been a major focus of our research over the past decade (Hou et al. 2014; Tang et al. 2017; Liu et al. 2018; Zhang et al. 2021). Temporal changes in the interaction of NF-YCs with CURLY LEAF (CLF) occur and counteract the deposition of H3K27me3 at the *FT* locus to regulate flowering time (Liu et al. 2018). NF-Y can also modulate the level of H3K27me3 at the *SOC1* locus and promote its expression by recruiting the H3K27 demethylase REF6 (Hou et al. 2014). BRM is a core catalytic subunit of the SWI/SNF-type CRC (Clapier et al. 2017). Recent studies have shown that BRM affects the level of H3K4me3 but not H3K27me3 at the *SOC1* locus, suggesting that the effects of BRM on *SOC1* expression are independent of the effects of the BRM-REF6 module (Li et al. 2015; Yang et al. 2022). In this study, we revealed that BRM and NF-YCs antagonistically regulate H3K4me3 occupancy at the *SOC1* locus through physical interactions, thus altering flowering time. BRM can repress the binding of NF-YCs to *SOC1* chromatin, which may be due to the competitive binding of BRM with NF-YCs to DNA or changes in the chromatin state induced by BRM. Given that NF-Ys are associated with multiple epigenetic factors, it will be interesting to investigate how NF-Ys orchestrate these factors and to uncover the fundamental roles of NF-Ys in epigenetic regulation during plant growth and development.

Materials and methods

Plant materials and growth conditions

All Arabidopsis (*A. thaliana*) plants used in this study are in the Col-0 background and were grown at 22 °C under long-day conditions (LDs, full-spectrum white fluorescent light intensity of 100 μmol m^{-2} s^{-1} with 16 h light/8 h dark photoperiod). All plant materials used in this study are listed in Supplemental Table S1. NF-YC-related plants, *brm-3*, *p35S:GFP*, *pBRM:BRM-GFP*, *35S:RGA-FLAG*, *pRGA:RGAΔ17*, *ga1*, and *dellaq* were described previously (Yu et al. 2012; Yang et al. 2015; Hu et al. 2018; Yan et al. 2020; Zhang et al. 2021). To generate *35S:RGA-6HA*, the coding region of *RGA* was inserted into the pGreen-35S:6HA vector (Hou et al. 2014). Primers used are listed in Supplemental Table S2. The floral dip method was used to generate the transgenic plants, and positive lines were selected by Basta treatment in soil. Seeds with a *ga1* background were imbibed in 100 μM GA_3 at 4 °C for 7 d and rinsed thoroughly with water before sowing.

Yeast 2-hybrid and 3-hybrid assays

The coding regions of genes for NF-YCs, BRM, and truncated versions of BRM were amplified and cloned into the *Eco*RI/*Pst*I, *Nde*I/*Eco*RI, or *Nde*I/*Xma*I restriction site of pGBKT7 (Clontech). The coding regions of genes for NF-YCs, BRM, DELLAs, and truncated versions of RGA were amplified and cloned into the *Xma*I/*Bam*HI, *Nde*I/*Xma*I, or *Nde*I/*Eco*RI restriction site of pGADT7 (Clontech). To analyze the effects of DELLAs on the interaction of BRM with NF-YC9, the *RGA* and *GAI* coding regions were amplified and cloned into the *Xho*I/*Xma*I or *Xma*I restriction site of pQH05 (Hou et al. 2014). Primers used are listed in Supplemental Table S2. Yeast 2-hybrid assays were performed using the Yeastmaker Yeast Transformation System 2 (Clontech). Yeast AH109 cells were co-transformed with specific bait and prey constructs. All yeast transformants were grown on SD/-Trp/-Leu medium for selection. To assess protein interactions, the transformed yeast cells were resuspended in liquid SD/-Leu/-Trp. Three microliters of suspended yeast cells were spotted onto SD/-Trp-Leu or SD/-Trp-Leu-His-Ade dropout plates to detect direct interactions between 2 proteins following incubation at 30 °C. Yeast 3-hybrid assays were performed as described previously (Hou et al. 2014). Measurement of β-galactosidase activity was performed according to the Yeast Protocols Handbook (Clontech) using chlorophenol red-β-D-galactopyranoside (Roche) as the substrate. Yeast 2-hybrid and 3-hybrid assays were repeated at least 3 times with similar results.

In vitro pull-down assay

The coding regions of genes for NF-YCs and DELLAs were cloned into the *Bam*HI/*Hind*III, *Bam*HI/*Sal*I, *Sac*I/*Xma*I, or *Kpn*I/*Sal*I restriction site of pQE30 (Qiagen) to produce *His-NF-YCs* and *His-DELLAs* constructs. The coding region of gene for *BRM^N* was cloned into the *Bam*HI/*Sal*I restriction site of pGEX-4T-1 (Pharmacia) to produce *GST-BRM^N* construct. Primers used are listed in Supplemental Table S2. These GST and His fusion recombinant proteins were induced and expressed in *Escherichia coli* Rosetta cells. The soluble His-NF-YC and His-DELLA proteins were extracted and immobilized on nickel-nitrilotriacetic acid agarose beads (Qiagen, 30210), while the soluble GST-BRM^N and GST proteins were extracted and immobilized on Glutathione Sepharose Beads (GE Healthcare, 17-0756-01). For pull-down assays, the purified His-NF-YC or His-DELLA proteins were incubated with immobilized GST or GST-BRM^N in binding buffer (50 mM Tris-HCl [pH 8.0], 100 mM NaCl, and 1 mM EDTA) at 4 °C overnight. After washing with binding buffer, proteins retained on the beads were subsequently resolved by SDS–PAGE and detected by immunoblotting with anti-HIS antibody (BGI, AbM59012-18-PU, 1: 10,000 dilution) or anti-GST antibody (TianGen, AB101-02, 1:10,000 dilution). Pull-down experiments were repeated 3 times with similar results.

Split-luciferase assay

The coding region of gene for BRM^N was cloned into the *Kpn*I/*Sal*I restriction site of pCAMBIA1300-nLUC to produce *BRM^N-nLUC* construct. The coding regions of genes for NF-YC9 and RGA were cloned into the *Kpn*I/*Sal*I restriction site of pCAMBIA1300-cLUC to produce *cLUC-NF-YC9* and *cLUC-RGA* constructs (Chen et al. 2008). Primers used are listed in Supplemental Table S2. These constructs were transformed individually into *Agrobacterium tumefaciens* strain GV3101. GV3101 cells harboring the indicated constructs expressing nLUC or cLUC fused proteins were mixed at a 1:1 ratio and introduced into *N. benthamiana* leaves. To determine the effects of RGA and GAI on the interaction of NF-YC9 with BRM^N, the coding regions of genes for RGA, GAI, and BFP (negative control) were cloned into the *Pst*I restriction site of pGreen-35S:GFP vector (Qian et al. 2021) to produce *35S: RGA-GFP* (*RGA-GFP*), *35S: GAI-GFP* (*GAI-GFP*), and *35S: BFP-GFP* (*BFP-GFP*) constructs, respectively. Primers used are listed in Supplemental Table S2. GV3101 cells harboring the constructs expressing *BRM^N-nLUC*, *cLUC-NF-YC9*, and *RGA-GFP*, *GAI-GFP*, or *BFP-GFP* were mixed at a 1:1 ratio and infiltrated into *N. benthamiana* leaves. At 2 to 3 d after infiltration, the leaves were incubated with 1 mM D-luciferin sodium salt substrate (Abcam, ab145164) and kept in the dark for 10 min. The luminescence imaging workstation (Tanon 5200) was used to capture luciferase images. Split-luciferase experiments were repeated 3 times with similar results. LUC activity was measured using the Dual-Luciferase Reporter Assay System (Promega, E1910) according to the manufacturer's instructions. The relative luminescence was presented with 3 independent experiments.

Co-IP assay

Seedlings with various genetic backgrounds were grown at 22 °C under LDs for 9 d and harvested for total protein extraction in co-IP buffer (50 mM HEPES [pH 7.5], 150 mM KCl, 10 mM $ZnSO_4$, 5 mM $MgCl_2$, 1% Triton X-100, and

0.05% SDS). The total proteins were incubated with GFP trap beads (Chromotek, gtak-20) at 4 °C overnight. The beads were washed 3 times with co-IP buffer, and the precipitated proteins were eluted in 1×SDS loading buffer by boiling for 10 min. The immunoprecipitated proteins were separated on a 6% or 10% SDS–PAGE gel and detected by immunoblotting with anti-GFP (TransGen, HT801-01, 1:5,000 dilution) and anti-FLAG (Sigma, F3165, 1:10,000 dilution) antibodies. Co-IP experiments were repeated 3 times with similar results.

Quantitative RT-PCR

Growth conditions and treatment of seedlings were described in the text. Total RNA was extracted from the samples using a Plant RNA Kit (Promega, LS1040) and reverse transcribed to cDNA using MMLV-RTase (Promega, M1701) according to the manufacturer's protocols. Gene expression levels were determined by RT-qPCR on a Light Cycler 480 thermal cycler system (Roche) with KAPA SYBR Fast qPCR Kit Master Mix (Kapa Bio, KK4680). The relative expression level of each gene was quantified in triplicate and normalized to that of *TUB2* (as an internal control). Primers used are listed in Supplemental Table S2.

Transient expression assay

To generate the *pSOC1:LUC* reporter construct, ~2 kb *SOC1* promoter was cloned into the *Hind*III/*Bam*HI restriction site of the pGreenII 0800-LUC vector. The *Renilla Luciferase* (*REN*) gene under the control of the 35S promoter in the pGreenII 0800-LUC vector was used as the internal control. The coding region of *BRM* was cloned into the *Pst*I restriction site of pGreen-35S:GFP to produce the *35S:BRM-GFP* (*BRM-GFP*) construct and used as an effector. The coding region of *NF-YC9* or *RGA* was cloned into the *Eco*RI/*Spe*I or *Hind*III/*Eco*RI restriction site of pGreen-35S:6HA to produce the *35:NF-YC9-HA* or *35S:RGA-HA* construct and used as another effector. Primers used are listed in Supplemental Table S2. These effector and reporter or control constructs were transformed individually into *A. tumefaciens* strain GV3101. GV3101 cells harboring the indicated constructs were mixed at a ratio of 1:1 and introduced into *N. benthamiana* leaves. The LUC and REN activities were measured using the Dual-Luciferase Reporter Assay System according to the manufacturer's instructions. The LUC/REN ratio was presented with 3 independent experiments.

ChIP qPCR assay

Seedlings grown under LDs were used for the ChIP assays, which were performed as described previously (Hou et al. 2014). Briefly, seedlings at 9 d after germination were vacuum-infiltrated with 1% formaldehyde for cross-linking, which was stopped by adding 150 mM glycine. Chromatin was isolated from the samples and sonicated to generate DNA fragments with an average size of ~500 bp. Subsequently, the chromatin complexes were immunoprecipitated by GFP trap beads or Protein G PLUS/Protein A agarose (Millipore, 16-201) plus anti-FLAG antibody at 4 °C overnight. The precipitated DNA fragments were recovered and quantified by qPCR with SYBR Premix ExTaq Mix using the primers shown in Supplemental Table S2. Relative enrichment fold was calculated by normalizing the amount of a target DNA fragment against that of a *PP2A* genomic fragment and then against the respective input DNA samples.

Observation of GFP fluorescence

GFP fluorescence in primary roots was observed under a confocal laser scanning microscope (Leica TCS SP5). A 488 nm laser was used to detect GFP excitation. All images were obtained with the same modifications and intensity parameters.

Statistical analysis

GraphPad Prism 8.0 and Microsoft Office Excel were used for statistical analysis of the numerical data. The statistically significant differences between 2 groups or multiple samples were determined by using a 2-tailed Student's *t*-test or 1-way ANOVA, respectively. Statistical data are provided in Supplemental Data Set 1.

Accession numbers

Sequence data from this article can be found in the TAIR website under the following accession numbers: *NF-YC3* (*AT1G54830*), *NF-YC4* (*AT5G63470*), *NF-YC9* (*AT1G08970*), *BRAHMA* (*AT2G46020*), *RGA* (*AT2G01570*), *GAI* (*AT1G14920*), *RGL1* (*AT1G66350*), *RGL2* (*AT3G03450*), *RGL3* (*AT5G17490*), *SOC1* (*AT2G45660*), *FT* (*AT1G65480*), *TUB2* (*AT5G62690*), *ACT7* (*AT5G09810*), *PP2A* (*AT1G69960*), *TA3* (*AT1G37110*), *Cinful-like* (*AT4G03770*).

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Author contributions

C.Z. and X.H. designed the research. C.Z., M.J., W.L., X.Y., and C.T. performed the research. C.Z., M.J., Q.Q., Y.H., X.L., and X.H. analyzed data; C.Z. and X.H. wrote the article.

Supplemental data

The following materials are available in the online version of this article.

Supplemental Figure S1. BRM interacts with DELLA proteins in yeast.

Supplemental Figure S2. BRM^N interacts with DELLA proteins in vitro.

Supplemental Figure S3. The *brm-3* mutant can rescue the late-flowering phenotype of *pRGA:RGAΔ17* (*RGAΔ17*) plants.

Supplemental Figure S4. Expression analysis of *FT*.

Supplemental Figure S5. The binding of BRM on *SOC1* and *FT*.

Supplemental Figure S6. The protein levels of BRM-GFP in *BRM-GFP*, *dellaq BRM-GFP*, and *ga1 BRM-GFP*.

Supplemental Figure S7. The protein levels of RGA in the *brm-3* mutant.

Supplemental Figure S8. GA reduces BRM binding to *SOC1*.

Supplemental Figure S9. BRM interacts with NF-YC proteins in yeast.

Supplemental Figure S10. BRM^N interacts with NF-YC proteins in vitro.

Supplemental Figure S11. Analysis of the regulation of *FT* by BRM and NF-YCs.

Supplemental Figure S12. BRM does not affect the binding of NF-YC9 to the *FT* locus.

Supplemental Figure S13. BRM and NF-YCs antagonistically regulate H3K4me3 level at *SOC1*.

Supplemental Figure S14. GAI enhances the interaction of NF-YC9 with BRM.

Supplemental Figure S15. Biological replicates for the Co-IP assays in Fig. 5F.

Supplemental Table S1. Sources of the plant materials.

Supplemental Table S2. List of primers used in this study.

Supplemental Data Set S1. Statistical analyses.

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Conflict of interest statement. The authors declare that they have no competing interests.

Data availability

All data to support the conclusions of this manuscript are provided in the main figures and supplementary information.

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