Functional Analysis of RF2a, a Rice Transcription Factor*

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RF2a is a bZIP transcription factor that regulates expression of the promoter of rice tungro bacilliform badnavirus. RF2a is predicted to include three domains that contribute to its function. The results of transient assays with mutants of RF2a from which one or more domains were removed demonstrated that the acidic domain was essential for the activation of gene expression, although the proline-rich and glutamine-rich domains each played a role in this function. Studies using fusion proteins of different functional domains of RF2a with the 2C7 synthetic zinc finger DNA-binding domain showed that the acidic region is a relatively strong activation domain, the function of which is dependent on the context in which the domain is placed. Data from transgenic plants further supported the conclusion that the acidic domain was important for maintaining the biological function of RF2a. RF2a and TBP (TATA-binding protein) synergistically activate transcription in vitro (Zhu, Q., Ordiz, M. I., Dabi, T., Beachy, R. N., and Lamb, C. (2002) Plant Cell 14, 795–803). In vitro and in vivo assays showed that RF2a interacts with TBP through the glutamine-rich domain but not the acidic domain. Functional analysis of such interactions indicates that the acidic region activates transcription through mechanisms other than via the direct recruitment of TBP.

The severe stunting symptoms of rice tungro disease are caused by infection of rice tungro bacilliform virus (RTBV), a double-stranded DNA badnavirus. Understanding the transcriptional regulation of RTBV is an important factor to elucidate the basis of the disease. RTBV carries a single, vascular tissue-specific promoter with several defined DNA cis-elements (1–4). Box II, one of the DNA cis-elements in the promoter, is essential for phloem-specific expression of the promoter (3, 4).

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EXPERIMENTAL PROCEDURES

Plasmid Construction

Plasmid Constructions for Protein Purification—Mutants of RF2a were created through PCR amplification. A NdeI restriction site was added at the 5′ end of all primers, and the ATG in the restriction site was in frame with the Hix4 tag in vector pET28a (Invitrogen) and served as the transcription start codon for plasmids described under “Plant Expression Constructs with RF2a Deletion Mutants.” A BamHI site was added to all 3′ primers with a stop codon in front of the restriction site. The primers used for amplification of different fragments of RF2a are listed below. The α fragment was amplified using primers RF2aA and RF2aC; ΔQ was amplified using primers RF2aA′ and RF2aQ5′; ΔPA was amplified using primers RF2aA′ΔPA5′ and RF2aC′; and ΔPAQ was amplified using primers RF2aΔP′A′ and RF2aQ5′. All of the fragments were restricted with Ndel and BamHI and were cloned into pET28a through the same set of restriction sites. All of the mutations were verified by DNA sequence analysis. The derived plasmids were designated pET-RF2a-ΔP, pET-RF2a-ΔQ, pET-RF2a-ΔPA, and pET-RF2a-ΔPAQ. TBP coding sequence was released from pOrTBP2 (25) through NcoI digestion (made blunt with Klenow DNA polymerase) followed by XhoI digestion. The released fragment was cloned into pGEX-4T-1 (Amersham Biosciences) through BamHI and XhoI sites (pGST-TBP) to produce a GST-TBP fusion protein with a thrombin recognition site in frame with the His6 tag in vector pET28a (Invitrogen) and served as the protein to the 3-ml reaction buffer and incubated at 4°C overnight. After intensive washing with TBST, the membrane was blocked with 5% nonfat milk in TBST for 1 h and electrophoresed in a 5% acrylamide gel (4). For supershift assays, various amounts of TBP were added to the reactions as indicated.

Transfection of Tobacco BY-2 protoplasts

The protoplasts were isolated from tobacco cell line BY-2 as described by Watanabe et al. (43). Approximately one million protoplasts were transfected by electroporation with 20 μg of effector DNA, 15 μg of herring sperm DNA, 2.5 μg of reporter gene DNA, and 15 μg of pCI-GFP DNA (constructed by C. Reichel). In samples with reporter gene alone, the total amount of DNA was adjusted by adding 20 μg of herring sperm carrier DNA. The electroporation parameters used were 300 V and 250 microfarads with the Bio-Rad electroporation system. Protoplast samples were cultured in Murashige and Skoog medium with 0.4 M mannitol, pH 5.8, at 28°C. The protoplasts were collected 24 h after electroporation.

Quantitative Analysis of GUS Activity and GFP

Protein samples from protoplasts were prepared using protein extraction buffer (27) and quantified using DC protein assay kit (Bio-Rad). GUS activity was measured using 4-methylumbelliferone as substrate as described by Jefferson et al. (27). GFP was quantified by spectrometry with excitation at 480 nm and emission at 510 nm.

Transformation

Tobacco Transformation

pGA482-derived plasmids were introduced into Agrobacterium tumefaciens strain LBA4404 and used for tobacco transformation. Leaf discs from Nicotiana tabacum cv. Xanthi NN were used following the protocol of Horsch et al. (28). At least 15 independent transgenic lines were produced with each gene construct. Transgenic plants were self-fertilized, and T1 seeds were collected. The T1 seeds were germinated on Murashige and Skoog medium (29) with kanamycin (100 mg/liter) selection, and KanR seedlings were grown in a greenhouse for observation of phenotypic changes.

Protein Overlay

2 μg of each protein sample was subjected to SDS-PAGE, and the proteins were transferred to nitrocellulose membranes (Schleicher & Schuell). The transferred proteins were denaturred in 20 mM Hepes-KOH, pH 7.9, 10% glycerol, 60 mM KCl, 6 mM MgCl2, 0.6 mM EDTA, 1 mM dithiothreitol, and 6 M guanidine hydrochloride for 45 min. The proteins were then renatured by incubation in the same buffer containing decreasing amounts of guanidine hydrochloride (3 μM, 1.5 μM, 0.75 μM, 0.3 μM, and 0.1 μM) for 20 min each followed by two washes without guanidine hydrochloride. The membrane was blocked in TBST buffer (50 mM Tris-HCl, pH 8.0, 150 mM NaCl, and 0.2% Tween 20) containing 5% nonfat milk powder for 1 h before adding 20 μg of GUS-TBP fusion protein to the 3-ml reaction buffer and incubated at 4°C overnight. After intensive washing with TBST, the membrane was blocked with TBST containing 5% milk for 30 min prior to addition of goat anti-GST antibody (Amersham Biosciences). After 2 h of incubation at room temperature, the membrane was intensively washed with TBST and

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blocked again with TBST containing 5% milk for another 30 min before adding the horseradish peroxidase-conjugated anti-goat secondary antibody. The membrane was washed with TBST after 2 h of incubation. The GST was detected by applying Super Signal Substrates for horse-

Radish Peroxidase-conjugated to Goat Anti-rabbit Antibody (Southern Protection. The primary antibody used in the immunodetection was raised with Ponceau S (Sigma) to monitor protein loading prior to immunode-

bition mixture 10 min prior to the addition of the whole cell extract. Protein RF2a, mutants of RF2a, and rice TBP2 were added to the 

Promoter Ligated to the 

GST was detected by applying Super Signal Substrates for horse-

rsldish Peroxidase (Pierce) and exposure to x-ray film. The membrane 

Western Immunoblot Reactions 

in plants (5). To analyze the function of each region, mutants of 

et plasmids were named pET-RF2a- 

Western Immunoblot Reactions 

Whole cell extracts of rice cv. IR72 suspension cultures were used to transcribe a template comprising nucleotides 164 to +45 of the RTBV promoter ligated to the uidA gene as described (5, 25). The recombiant 

proteins of mutants of RF2a were detected through the same Western 

Functional Dissection of bZIP Protein RF2a 

RESULTS 

RF2a Mutants with Deletions of Functional Domains Bind to the DNA Target Sequence—In previous studies, we showed that the bZIP protein RF2a enhances transcription in vivo and in vitro (5, 6). Based on the enrichment of amino acids in specific regions of RF2a, we proposed that proline-rich, acidic, and glutamine-rich regions might play specific roles in the function of RF2a (5). To analyze the function of each region, mutants of 

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In Vitro Transcription Assay 

Transcription products were analyzed by primer extension with a primer located in the GUS coding region (2).

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It has been reported that in some cases, protein domains other than the bZIP domain are required for establishing stable protein-DNA complexes (30, 31). Box IIm1 is a mutant of Box II fused to a minimal CaMV 35S promoter comprising nucleotides 8 to +8. The chimera was ligated to the uidA coding sequence to create the reporter plBI-48Ca:GUS. The activity of this chimeric pro-

FIG. 1. Electrophoretic mobility shift assay of protein-DNA complexes formed between mutants of RF2a and Box II cis-

clement of the RTBV promoter. A, schematic diagram of mutants of RF2a. A, acidic domain; P, proline-rich domain; Q, glutamine-rich do-

moter is about 16% of the activity of RTBV promoter activity and 6.5% of the activity of the enhanced CaMV 35S promoter in BY-2 protoplasts (not shown).

To analyze the function of domains of RF2a, effectors were created by inserting coding sequences of mutants of RF2a downstream of the enhanced CaMV 35S promoter in pMON999 vector (a gift from Monsanto Co., St. Louis, MO). The resultant constructs, p35S:RF2a, p35S:RF2a-ΔP, p35S:RF2a-ΔQ, p35S:RF2a-ΔPA, p35S:RF2a-ΔPAQ, and p35S:RF2a-3A, were co-transfected into BY-2 protoplasts with plBI-48Ca:GUS (Fig. 2A). Plasmid pCat-GFP, in which the GFP gene was driven by CaMV 35S promoter, was co-introduced to serve as an internal control.

As shown in Fig. 2B, the transactivation function of RF2a was not decreased by removing either the proline-rich (RF2a-ΔP) or glutamine-rich (RF2a-ΔQ) domains or both of the domains (RF2a-ΔPAQ). In fact, the activation function of each of these mutants was greater than that of full-length RF2a; RF2a-ΔP was significantly different from RF2a at the P 0.05 level, whereas RF2a-ΔQ and RF2a-ΔPAQ were significantly different from RF2a at the P 0.01 level (Student’s t test). Also, the difference between the activation function of RF2a-ΔP and RF2a-ΔQ was significant at the P 0.01 level, and there was no difference between RF2a-ΔQ and RF2a-ΔPAQ. The data suggest that the proline-rich and glutamine-rich domains do not contribute in a positive way to the activation function of RF2a; on the contrary,
these domains may reduce the activity of RF2a. In contrast, the activity dropped to near basal level when the acidic domain was removed (RF2a-ΔPA and RF2a-3A) (Fig. 2B). These results suggest that the acidic domain is responsible for the activation of gene expression by RF2a.

Functions of RF2a Domains in Fusion Proteins with 2C7 DBD—To determine whether domains of RF2a can serve as independent modules to regulate transcription, putative functional domains were fused with the synthetic 2C7 protein, a synthetic zinc finger DBD that specifically binds to the 2C7 DNA-binding site (26, 32) (Fig. 3A); domains were placed either at the N terminus or the C terminus of the DBD. The reporter construct pC7er2:GUS carried the uidA coding sequence located downstream of a chimeric promoter comprising 6x2C7-binding sites ligated with the minimal promoter of erbB-2 (26). p35S:2C7 encodes the 2C7 protein without an activation domain and served as a control (Fig. 3A) (26).

As shown in Fig. 3B, when domains of RF2a were placed C-terminal of the 2C7 protein, 2C7-A and 2C7-PA showed significant activation function. When the domains were fused individually at the N terminus of 2C7, the acidic domain (A-2C7) gave stronger activation than the P (P-2C7) or Q (Q-2C7) domains did. The function of the acidic domain in the fusion proteins is consistent with its function in RF2a, although the position of this domain in the fusion domain affects its activity. The proline- and glutamine-rich domains had no effect on gene expression when they were placed at the C terminus of the 2C7 DBD; however, these two domains showed mild activation function when they were fused at the N terminus of the 2C7 DBD. Neither of these two domains showed repression function as they did in the context of RF2a.

Impact of Mutants of RF2a on Plant Development—We previously demonstrated that transgenic rice (5) and tobacco (6) plants that overexpressed RF2a were normal in appearance and reproduction. To determine whether mutants of RF2a from which one or more domains were removed had a positive or negative effect on plant development, we produced transgenic plants that overexpress mutants of RF2a. Fifteen or more independent transgenic tobacco lines were developed with each construct through Agrobacterium-mediated transformation. After PCR analysis, transgenic lines with each mutant were observed for phenotypic changes. T1 generation plants with 35S:RF2a, 35S:RF2a-ΔPA, 35S:RF2a-ΔQ, and 35S:RF2a-ΔPAQ did not exhibit abnormal phenotypes (Fig. 4A). However, 11 of 15 independent transgenic lines with 35S:RF2a-ΔPA exhibited mild to severe stunting with curved leaves and substantial delay in flowering times (Fig. 4, A and B). The internodal elongation of transgenic plants was strongly repressed by RF2a-ΔPA (Fig. 4C, panel 1). The phenotype caused by 35S:RF2a-ΔPA was similar to but less severe than the phenotype caused by 35S:RF2a-3A (6). Cross-sections of the stem of transgenic plants with either RF2a-ΔPA or RF2a-3A showed that the xylem of stunted plants was not uniformly lignified and...
that phloem development was altered (Fig. 4C).

To confirm that the phenotype was related to transgene expression, leaf protein samples of transgenic plants with RF2a-ΔPAΔA were analyzed via a Western blot assay using an antibody against RF2a. Fig. 5 shows that there is a direct correlation between the abnormal phenotype and the accumulation of RF2a-ΔPAΔA.

**RF2a Interacts with TBP via the Glutamine-rich Domain—Zhu et al. (25)** reported that RF2a physically interacts with rice TBP, and the results of current studies raised the question of whether this interaction affects RF2a function. To address this issue, we first conducted experiments to identify the regions of RF2a that interact with TBP. Gel mobility shift assays of mutants of RF2a were carried out with and without the addition of purified TBP (Fig. 6A). 32P-labeled Box II1m1 DNA element was used as probe in the gel shift assays. As shown in Fig. 6B, RF2a-ΔQ and RF2a-ΔPAΔA form DNA-protein complexes with Box II1m1. When TBP was added to the reactions, only the RF2a-ΔPAΔA-DNA complex exhibited a band with slower mobility (supershift) compared with the sample without TBP. When a higher amount of TBP was added to the reaction, both the RF2a-ΔPAΔA-DNA complex and the supershift band was enhanced. In samples with RF2a-ΔQ, no supershift of DNA-protein complexes was observed. This experiment shows that the proline-rich and acidic domains did not directly interact with TBP, whereas the glutamine-rich domain did.

To confirm the data from gel supershift assays, protein overlay assays were carried out. Mutants of RF2a were separated by SDS-PAGE and blotted to nitrocellulose membrane, and the
RF2a binds to Box II, a cis-element adjacent to the TATA box, to regulate expression of the RTBV promoter (5, 6). Results of in vitro transcription studies indicated that interactions of RF2a with TBP contribute to the function of RF2a (25). In the present study, we defined the functional domains of RF2a using in vivo assays and examined the mechanisms by which these domains contribute to its activity.

The proline-rich and glutamine-rich domains of RF2a do not directly contribute to the activation function of RF2a, although the acidic (A) domain is essential for its activity. The A domain is a strong activation domain when fused with a heterologous DNA-binding domain. The P and Q domains showed weak activation function only when they were placed at the N terminus of the 2C7 DBD; this activity is different from functions in the context of RF2a. Nevertheless, RF2a is a strong activator of the expression of the RTBV promoter (6).

The bZIP domain of RF2a shares high similarity with a small family of bZIP proteins that include members from Arabidopsis, tobacco, tomato, and other plants (34–36). This group of proteins has a lysine residue at the −10 position relative to the first leucine residue of the leucine zipper domain (5). The amino acid sequence signature of the DNA-binding regions of the proteins of this class is NXXXSAXXXSK (37). The identification of this subgroup of bZIP proteins may imply that members are essential for plant growth and development. Based upon the finding that the RTBV promoter is expressed in vascular tissues in rice (2, 4), tobacco (6), and Arabidopsis, we suggest that this group of bZIP proteins is involved in regulating gene expression in these tissues. Yin et al. (5) used an antisense approach to demonstrate that RF2a is important for development of the vascular system in rice seedlings. Ectopic expression of RF2a-ΔPΔA and RF2a-3A in transgenic tobacco (Fig. 4) and rice caused severe stunting and abnormal development. These phenotypes apparently result from dominant negative effects of RF2a-ΔPΔA and RF2a-3A on the expression of genes that require function of RF2a-like homologs in tobacco plants, such as RSG (34, 38). Fukazawa et al. (34) determined that RSG regulates GA biosynthesis through the GA3 gene.

Interestingly, the proline-rich and glutamine-rich domains of RF2a did not directly contribute to the activation function of RF2a in BY-2 protoplasts even though these two domains showed limited activation functions in fusions with 2C7 DBD.

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Like the proline-rich domain in p45 NF-E (39), the proline-rich domain of RF2a may be dispensable for the activity of RF2a. In contrast, the C-terminal region of RF2a, including the glutamine-rich domain, may repress the function of the acidic domain (Fig. 3). Previous studies demonstrated that glutamine-rich domains can act as repression domains (23, 24). It will be important to further map and characterize the potential repression domain at the C terminus of RF2a.

The binding of TBP is important for formation of the transcription preinitiation complex (7, 19). Therefore, interactions between gene-specific transcription factors and TBP or TAFs and recruitment of TFIIID are important mechanisms of gene regulation. Because the acidic domain is a primary contributor to the activation of RF2a in vivo, we expected that this domain would interact with TBP. Surprisingly, as in the case of AtHSF1, which interacts with AtTBP1 and AtTBP2 through its N-terminal sequence rather than the acidic C terminus (40), the glutamine-rich domain was responsible for interaction of RF2a with TBP (Figs. 6 and 7). Our studies indicate that the acidic domain activates transcription through mechanisms other than direct binding to TBP. For example, the acidic domain may interact with other components of the transcriptional machinery upon binding of RF2a to the cis-element. Alternatively, the acidic domain may recruit TFIIID through interaction with certain TAFs (18).

The roles of the glutamine-rich domain and TBP interaction were somewhat different in vitro and in vivo (Fig. 7). In the cell-free in vitro assay, RF2a and TBP enhanced transcription in a synergistic manner when the glutamine-rich domain was obviated the repression effect of the glutamine-rich domain but will be important to further map and characterize the potential repression domain at the C terminus of RF2a.

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