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Xanthomonas TAL effectors hijack host basal transcription factor IIA α and γ subunits for invasion

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ABSTRACT

The *Xanthomonas* genus includes Gram-negative plant-pathogenic bacteria, which infect a broad range of crops and wild plant species, cause symptoms with leaf blights, streaks, spots, stripes, necrosis, wilt, cankers and gummosis on leaves, stems and fruits in a wide variety of plants via injecting their effector proteins into the host cell during infection. Among these virulent effectors, transcription activator-like effectors (TALEs) interact with the γ subunit of host transcription factor IIA (TFIIAγ) to activate the transcription of host disease susceptibility genes. Functional TFIIA is a ternary complex comprising α, β and γ subunits. However, whether TALEs recruit TFIIAα, TFIIAβ, or both remains unknown. The underlying molecular mechanisms by which TALEs mediate host susceptibility gene activation require full elucidation. Here, we show that TALEs interact with the α+γ binary subcomplex but not the α+β+γ ternary complex of rice TFIIA (holo-OsTFIIA). The transcription factor binding (TFB) regions of TALEs, which are highly conserved in *Xanthomonas* species, have a dominant role in these interactions. Furthermore, the interaction between TALEs and the α+γ complex exhibits robust DNA binding activity *in vitro*. These results collectively demonstrate that TALE-carrying pathogens hijack the host basal transcription factors TFIIAα and TFIIAγ, but not TFIIAβ, to enhance host susceptibility during pathogen infection. The uncovered mechanism widens new insights on host-microbe interaction and provide an applicable strategy to breed high-resistance crop varieties.

1. Introduction

Xanthomonas, a genus of phytopathogenic bacteria, cause devastating diseases and severely impact the yield quantity and quality of important crops including at least 124 monocots and 268 dicots, such as rice, soybean, cassava, brassica, cotton, pepper, and citrus [1,2]. During *Xanthomonas* infection, a plethora of diverse virulent factors are injected into host cells through the type III secretion system and are thus called type III effectors [3]. These virulence factors typically mimic the structure and function of host proteins to adapt to the host cell, and they suppress host innate immunity to benefit pathogen colonization [2,4].

Transcription activator-like effectors (TALEs) are members of the type III effectors family that are secreted into the host cytosol and subsequently translocated into the nucleus to reprogram the host transcriptome [3]. Similar to transcription factors, these TALE proteins specifically bind to TAL effector-binding elements (EBEs) on either strand of host target genes with a unique protein-DNA modular and to activate downstream host gene transcription [5,6]. Evolution-driven mutations in the EBEs that alleviated or disrupted TALE binding can improve plant resistance to diseases, and these mutations have been harnessed to breed resistant plant varieties to control TALE-mediated diseases [2,7–10].

Typically, the TALE protein contains five domains: an N-terminal type III secretion signal sequence; a central DNA binding domain (DBD); a transcription factor binding region (TFB); three nuclear localization signals; and a C-terminal acidic transcription activation domain [1,11]. The DBD of TALEs contains a set of tandem repeats, each of which precisely targets one DNA base with two residues (called repeat variable di-residues, RVDs). The RVD combinations generate a DNA recognition alphabet and have been well studied [3]. The C-terminal acidic transcription activation domain, which has acidic residues interspersed with bulky and hydrophobic residues, is highly conserved and mediates host gene transcription [12].

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Although the mechanisms of the DBD and the acidic transcription activation domain are clear, the role of the TFB region in TALEs remains elucidation.

In eukaryotes, transcription is regulated by numerous factors. A key intermediate in transcription initiation is the multicomponent preinitiation complex (PIC), which assembles over the core promoter of a gene [13]. PIC assembly is initiated by the recruitment of transcription factor IID (TFIID) and transcription factor IIA (TFIIA) to the core promoter, followed by the recruitment of other transcription factors [14]. TFIID recognizes the core promoter through one of the components, the TATA box-binding protein (TBP), and consequently triggers PIC assembly. The specific DNA binding activity of TBP is a key event initiating PIC assembly [15]. As a basal transcription factor, TFIIA contains α, β, and γ subunits that form a ternary complex; two of these subunits (β and γ) mediate the primary interaction with TBP. This interaction stabilizes TBP binding to the TATA box [16–18].

The TFB region of TALEs harbors an imperfect leucine zipper motif that typically mediates protein-protein interactions [19]. Previous studies have demonstrated that the TFB region of TALEs of *Xanthomonas* directly interact with the TFIIAγ subunit of host plant, which plays a pivotal role during pathogen invasion by regulating host susceptibility gene transcription to promote disease [11,20]. Considering that TFIIAγ recruits the other two subunits, TFIIAα and TFIIAβ, it remains unknown whether TALEs can associate with TFIIAα or TFIIAβ or both.

In this study, we carried out biochemical assays to examine the interaction between TALEs and rice holo-TFIIA (OsTFIIA) or subcomplexes. The results demonstrated that TALE dHax3, a designed hybrid TALE of Hax3 produced by the *Brassicaceae* pathogen *X. campestris* pv. *armoraciae* strain [21], was unable to form a complex with either the un-cleaved (αβ+γ) or cleaved (α+β+γ) form of holo-OsTFIIA. In contrast, dHax3 robustly interacts with the α+γ subcomplex of OsTFIIA through the TFB region. Consistently, the TFB region of TALE PthXo1 also assembles a stable complex with the α+γ subunits of OsTFIIA. Furthermore, an electrophoretic mobility shift assay (EMSA) showed that the reconstituted ternary complex of dHax3 with OsTFIIA (α+γ) retained clear DNA binding activity. Totally, we propose that TALE-carrying pathogens can hijack host transcription initiation factors TFIIAα and TFIIAγ, but not TFIIAβ, to enhance host susceptibility for infection. This finding provides new insights into the functions of TALEs and suggests a potential strategy for breeding high-resistance crop varieties.

2. Materials and methods

2.1. Vectors and constructs

The cDNAs encoding dHax3 and DBD were subcloned into pET-15b (Novagen, Darmstadt, Germany). The TALE TFB DNA were amplified and subcloned into pGEX6p-1 (Novagen). To generate well-expressed TFB proteins, a series of boundaries were screened and constructed in the same vector. The cDNAs encoding the cleaved form of holo-OsTFIIA (α+β+γ) were subcloned into pRSFDuet-1. The OsTFIIAα (M1 to S59) and OsTFIIAβ (A305 to F347) subunits were fused together with a linker (GGGGSDEVDA) and cloned into MCS1 of pRSFDuet-1 with 6 His at the N-terminus, whereas the OsTFIIAγ5 (M1-Q106) subunit was cloned into MCS2 with no tag. DEVD↓A is the cutting site for the drICE protease. The cDNAs encoding the un-cleavable form of holo-OsTFIIA (αβ+γ) were subcloned into pQLinkH (GenBank: EF025688) [22]. To generate the α+γ complex, OsTFIIAα and OsTFIIAγ5 were also fused together with the linker (GGGGSDEVDA) and subcloned into pQLinkH. The β+γ complex was constructed in the same manner as the α+γ complex.

2.2. Protein expression and purification

All the plasmids were transformed into *Escherichia coli* BL21 (DE3). A procedure for the overexpression and purification of dHax3, DBD, TFBs, holo-OsTFIIA (α+β+γ), holo-OsTFIIA (αβ+γ), α+γ, and β+γ was performed as previously [23]. The TFB supernatant was applied to Glutathione Sepharose 4B Affinity Resin (Qiagen, Hilden, Germany), while the others to Ni^{2+} affinity resin (Ni-NTA, Qiagen). The dHax3 was further purified using heparin affinity column (Heparin, GE Healthcare, Chicago, USA), while the others using ion exchange column (Source 15Q, GE Healthcare). After that each protein was subjected to gel filtration chromatography (Superdex-200 Increase 10/300, GE Healthcare). The buffer for gel filtration chromatography contained 25 mM Tris-HCl (pH 8.0), 250 mM NaCl, and 5 mM DTT. The peak fractions from size exclusion were collected. Holo-OsTFIIA (α+β+γ), α+γ, and β+γ were cleaved by drICE overnight at 4 °C prior to performing gel filtration chromatography.

2.3. Size exclusion chromatography (SEC)

All the proteins used in the SEC assays were purified as described above. SEC assays were performed as described previously [24]. However, the assays that referred to proteins dHax3 and DBD used a 250 mM NaCl concentration. Samples from relevant fractions were examined by SDS-PAGE and visualized with Coomassie blue staining. Tricine-SDS-PAGE was used when the proteins were smaller than 14 kDa.

2.4. Electrophoretic mobility shift assay (EMSA)

A single-stranded DNA oligonucleotide containing a dHax3 binding site (5′-CCACATATGTCATACGTGTCCCTTTATCTCTCTCCAGCTCGAGGAATTC-3′) was labeled with 5-carboxyfluorescein (FAM) at the 5′ end. The dsDNA was generated by mixing equal molar quantities of forward and reverse strands. The mixture was subsequently boiled and cooled to room temperature. For the EMSA, increasing amounts of the dHax3+α+γ complex and α+γ complex (0.25, 0.5, 1, and 1.5 μM) were incubated with approximately 100 nM FAM-labeled dsDNA on ice in binding buffer containing 25 mM Tris-HCl (pH 8.0), 150 mM NaCl, 5 mM MgCl2, 10% glycerol (w/v), 50 ng/ml heparin, 5 mM DTT, and 0.1 mg/ml bovine serum albumin (BSA) for 15 min. The reactions were then resolved on 6% native acrylamide gels (37.5:1 for acrylamide: bisacrylamide) in 0.25 × Tris borate buffer under an electric field of 15 V/cm for 2 h. Gels were visualized using a Typhoon Trio Imager (Amersham Biosciences).

3. Results and discussion

3.1. dHax3 cannot interact with holo-OsTFIIA

We previously demonstrated that the TFB motif of TALEs of *Xanthomonas* directly interacts with the γ subunit of host plant TFIIA to facilitate pathogen invasion [11,20]. In higher eukaryotes, purified TFIIA contains α, β, and γ subunits. TFIIAαβ is encoded by a single gene that is posttranslationally cleaved into α and β subunits by threonine aspartase1 (taspase1) to form a TFIIA ternary complex with TFIIAγ [25,26]. Both un-cleaved (αβ+γ) and cleaved (α+β+γ) forms of holo-TFIIA mediate transcription initiation [25,27]. To exam whether holo-TFIIA (αβ+γ/α+β+γ) is involved in TALE-mediated gene activation, we performed SEC to assess the interactions between PthXo1, a TALE of *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) strain $PXO99^{A}$ and holo-OsTFIIA (αβ+γ/α+β+γ) *in vitro*.

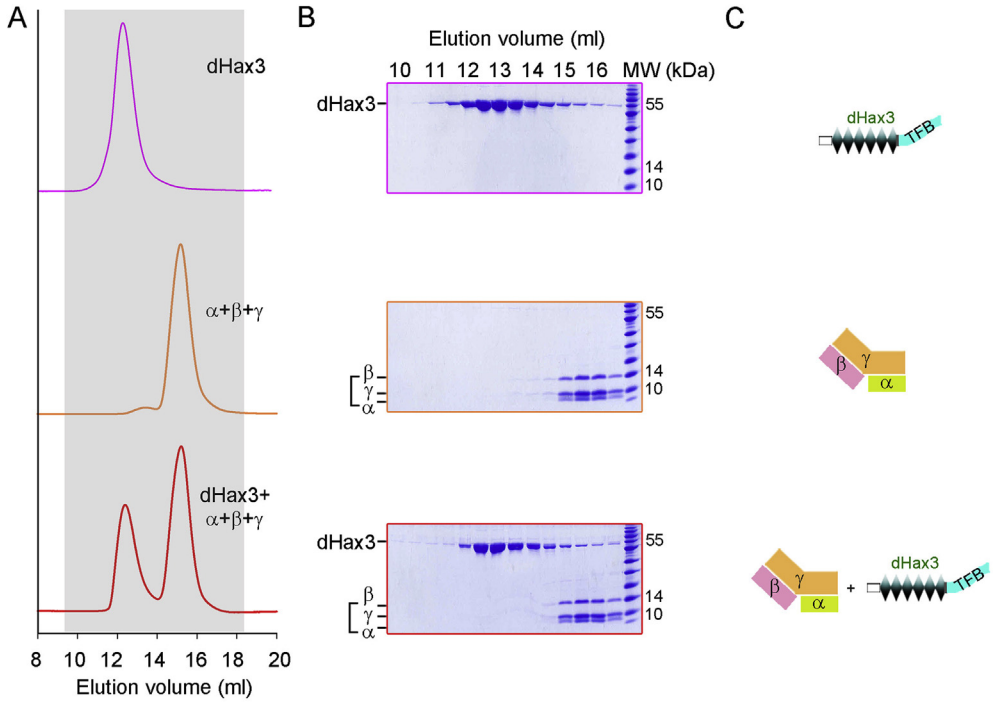

Fig. 1. The dHax3 cannot interact with the cleaved form of holo-OsTFIIA (α+β+γ). (A) Peaks fractions of proteins dHax3, α+β+γ, and dHax3 and α+β+γ in SEC. (B) SDS–PAGE of peak fractions in (A) at the same elution volume from individual injections stained by Coomassie blue. (C) Model of proteins in SEC.

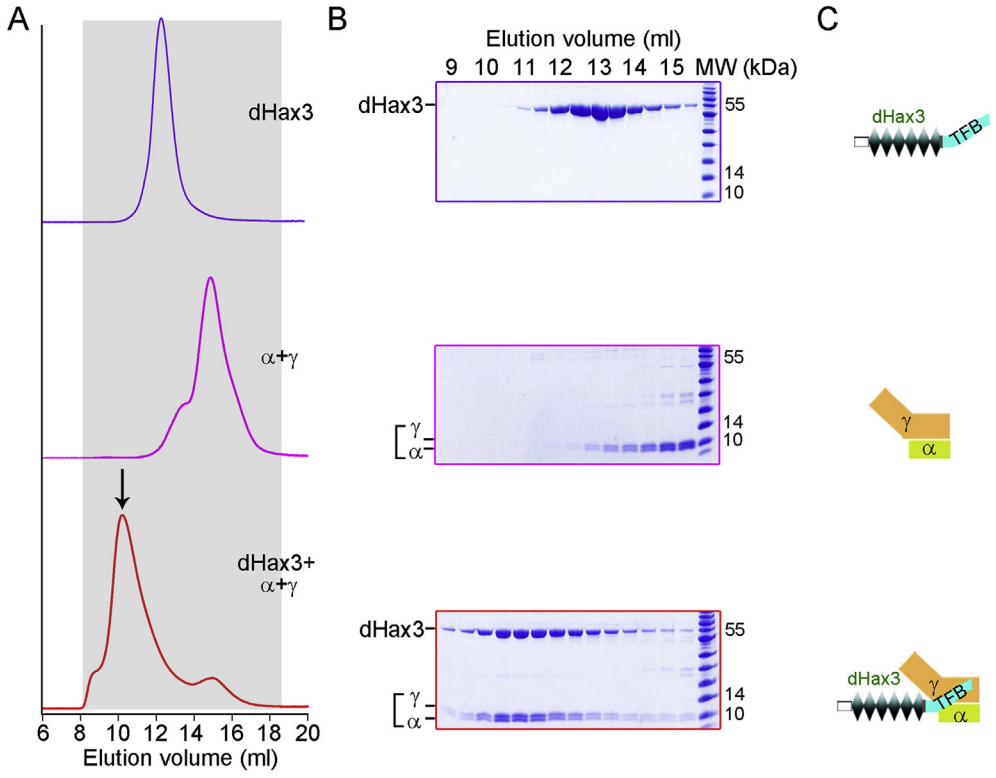

Fig. 2. The dHax3 interacts with the α+γ subcomplex of OsTFIIA. (A) Peaks fractions of protein dHax3, α+γ, and dHax3 in complex with α+γ in SEC. The arrow denotes the peak fractions of the dHax3+α+γ complex that were co-eluted. (B) SDS–PAGE of peak fractions in (A) at the same elution volume from individual injections stained by Coomassie blue. The apparent co-migration of dHax3 with α+γ subunits indicates their interaction (the lower panel). The controls are shown in the upper and middle panels. (C) Models of proteins in SEC.

Full-length recombinant PthXo1 protein is difficult to express (data not shown). Thus, we used a previously designed functional TALE dHax3, which contains a central DNA binding region and a TFB region (residues Q231 to M816) [21]. First, we assessed whether dHax3 interacts with the un-cleaved form of holo-OsTFIIA (αβ+γ). The elution volumes of dHax3 and holo-OsTFIIA (αβ+γ) were approximately 12.5 and 13 ml, respectively (Supplemental Figs. 1A and 1B). After co-incubation, no obvious shifted peak was observed (Supplemental Fig. 1A), suggesting that dHax3 is unable to interact with the un-cleaved form of holo-OsTFIIA (αβ+γ) (Supplemental Fig. 1C).

A long flexible middle region (245 residues) existed between the α and β subunits that might block the interaction between dHax3 and holo-OsTFIIA. Thus, we expressed and purified the cleaved form of holo-OsTFIIA (α+β+γ) by replacing the flexible region between α and β subunits with a linker (GGGGSDEVDA) and co-expressed OsTFIIAα-GGGGSDEVDA-OsTFIIAβ and OsTFIIAγ5 in the same vector. After anion-exchange chromatography, the recombinant protein was cleaved by drICE protease which could cut at the DEVD↓A site [28], to generate the α+β+γ ternary complex. Subsequently, we examined the interaction of dHax3 with the cleaved form of holo-OsTFIIA (α+β+γ). The elution volumes of dHax3 and the cleaved form of holo-OsTFIIA (α+β+γ) were approximately 12.5 and 15.5 ml, respectively (Fig. 1A and B, upper and middle panels). After co-incubation, dHax3 and holo-OsTFIIA (α+β+γ) proteins were eluted separately (Fig. 1A and B, lower panel), indicating that no direct interaction occurred between dHax3 and the cleaved form of holo-OsTFIIA (α+β+γ) (Fig. 1C). Taken together, the results indicated that dHax3 was unable to interact with either the cleaved or un-cleaved form of the ternary holo-OsTFIIA complex.

3.2. dHax3 directly interacts with OsTFIIA (α+γ) subcomplex

Although dHax3 interacted with OsTFIIAγ5 (Supplemental Fig. 2), this protein failed to form a quaternary complex with holo-OsTFIIA (Fig. 1 and Supplemental 1). We speculated that OsTFIIAα or OsTFIIAβ might block the interaction between dHax3 and OsTFIIAγ5. Thus, we examined the interaction of dHax3 with the OsTFIIA (α+γ) subcomplex (without β) or the OsTFIIA (β+γ) subcomplex (without α) of holo-OsTFIIA. The OsTFIIA (α+γ) subcomplex was expressed and purified to homogeneity (Fig. 2, middle panels), while the OsTFIIA (β+γ) subcomplex is prone to aggregation (data not shown). Therefore, we tested the interaction of dHax3 with the OsTFIIA (α+γ) subcomplex. The elution volumes of dHax3 and the OsTFIIA (α+γ) subcomplex were approximately 12.5 and 15.5 ml, respectively (Fig. 2A and B, upper and middle panels). After co-incubation, in sharp contrast to the results obtained with holo-OsTFIIA (α+β+γ/αβ+γ), dHax3 and the OsTFIIA (α+γ)

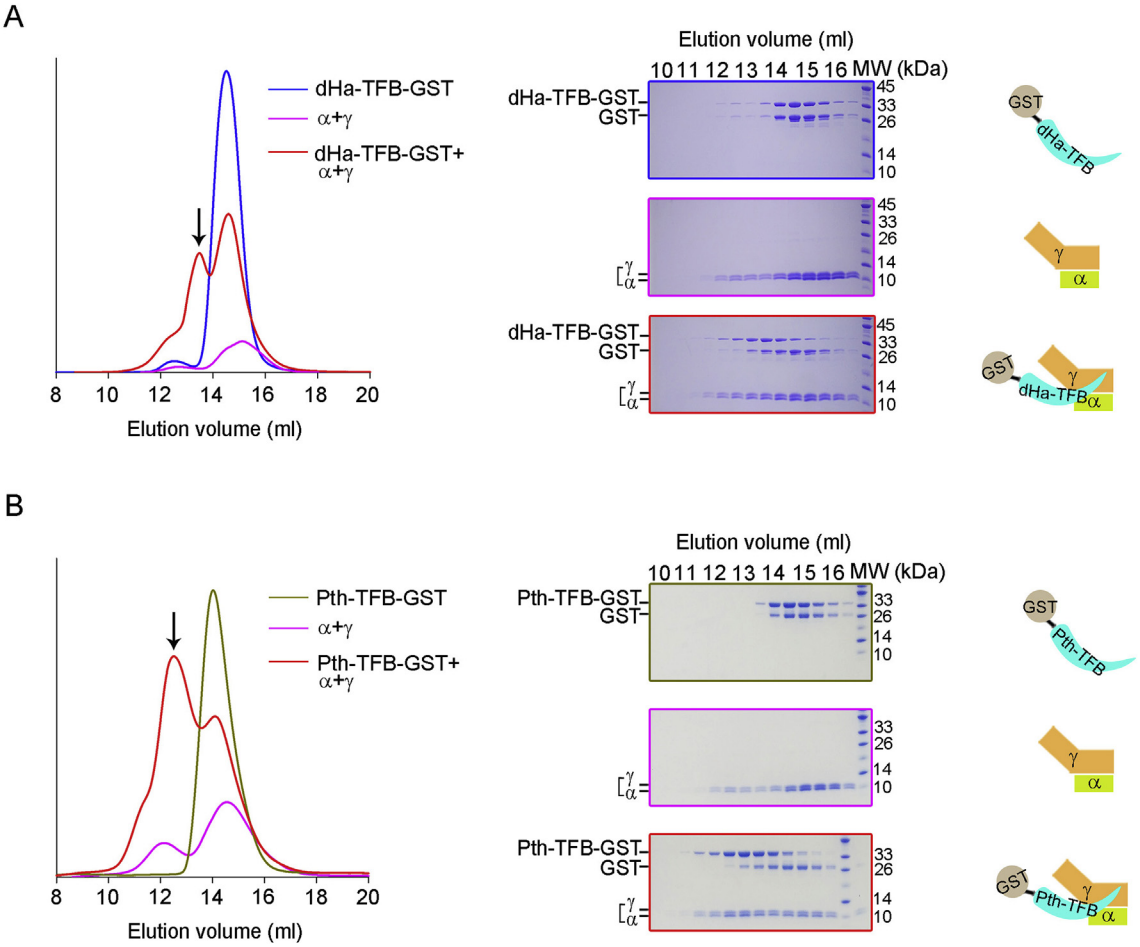

Fig. 3. The TFB region of TALEs is sufficient for a stable association with the α+γ subcomplex of OsTFIIA. Analysis of the interactions between dHa-TFB-GST (A) and Pth-TFB-GST (B) with the α+γ subcomplex of OsTFIIA by SEC was examined. DHa, dHax3; PthXo1, Pth. Left panels are the peak fractions of proteins in SEC. Middle panels are SDS-PAGEs of relevant peak fractions at the same elution volume from left panel stained by Coomassie blue. Right panels are models of proteins in SEC. The arrows denote the co-eluted peak fractions.

subcomplex were co-eluted at 11 ml (Fig. 2A and B, lower panels). These results clearly demonstrated that dHax3 interacts with the OsTFIIA (α+γ) subcomplex (Fig. 2C).

3.3. TFB regions of TALEs predominantly contribute to the OsTFIIA (α+γ) subcomplex interaction

Our previous studies showed that the TFB motif plays a crucial role in TALE interaction with OsTFIIAγ5 [11,20]. To examine whether the TFB region of dHax3 recruits the OsTFIIA (α+γ) subcomplex, we expressed and purified the GST-tagged TFB region of dHax3 (dHa-TFB, residues A745-M816) and confirmed its interaction with the OsTFIIA (α+γ) subcomplex by SEC. The dHa-TFB and OsTFIIA (α+γ) subcomplex were eluted at approximately 14.5 and 15.5 ml, respectively (Fig. 3A). After co-incubation, dHa-TFB co-migrates with the OsTFIIA (α+γ) subcomplex. In contrast, the central DBD of dHax3 was unable to form a complex with the OsTFIIA (α+γ) subcomplex (Supplemental Fig. 3A). These results suggest that dHax3 recruits the OsTFIIA (α+γ) subcomplex through the TFB motif.

Moreover, the TFB motif is highly conserved in diverse TALEs from *Xanthomonas* species (Supplemental Fig. 4). Thus, we assessed whether the TFB motif of PthXo1 (Pth-TFB, residues A1044-M1115) from *Xoo* strain PXO99^{A} interacted with the OsTFIIA (α+γ) subcomplex. The SEC assay clearly showed that Pth-TFB co-eluted with the OsTFIIA (α+γ) subcomplex (Fig. 3B), indicating that Pth-TFB also recruited the OsTFIIA (α+γ) subcomplex similar to dHax3 recruitment. In contrast, Pth-TFB failed to form a complex with the un-cleaved (αβ+γ) and cleaved (α+β+γ) forms of holo-OsTFIIA (Supplemental Fig. 5). These results are consistent with those for dHax3, suggesting conserved roles of the TFB motif of TALEs in hijacking the OsTFIIA (α+γ) subcomplex.

3.4. TALEs+α+γ complex exhibits DNA binding activity

TALEs typically bind to target DNA to modulate gene transcription [29,30]. Our results showed that TALEs recruit the OsTFIIA (α+γ) subcomplex. However, whether this recruitment influences TALEs DNA binding activity remains unclear. To answer this question, we preformed EMSA analyses. Increasing band retardation was detected with increasing amounts of the dHax3+α+γ ternary complex (Fig. 4A). In contrast, the α+γ binary complex did not exhibit DNA binding activity, even at high protein concentrations. These results indicate that the dHax3+α+γ complex retains DNA binding activity.

The molecular mechanism by which TALEs mediate the activation of susceptibility genes during *Xanthomonas* infection is poorly understood. Recent progress, mostly via genetic studies, has

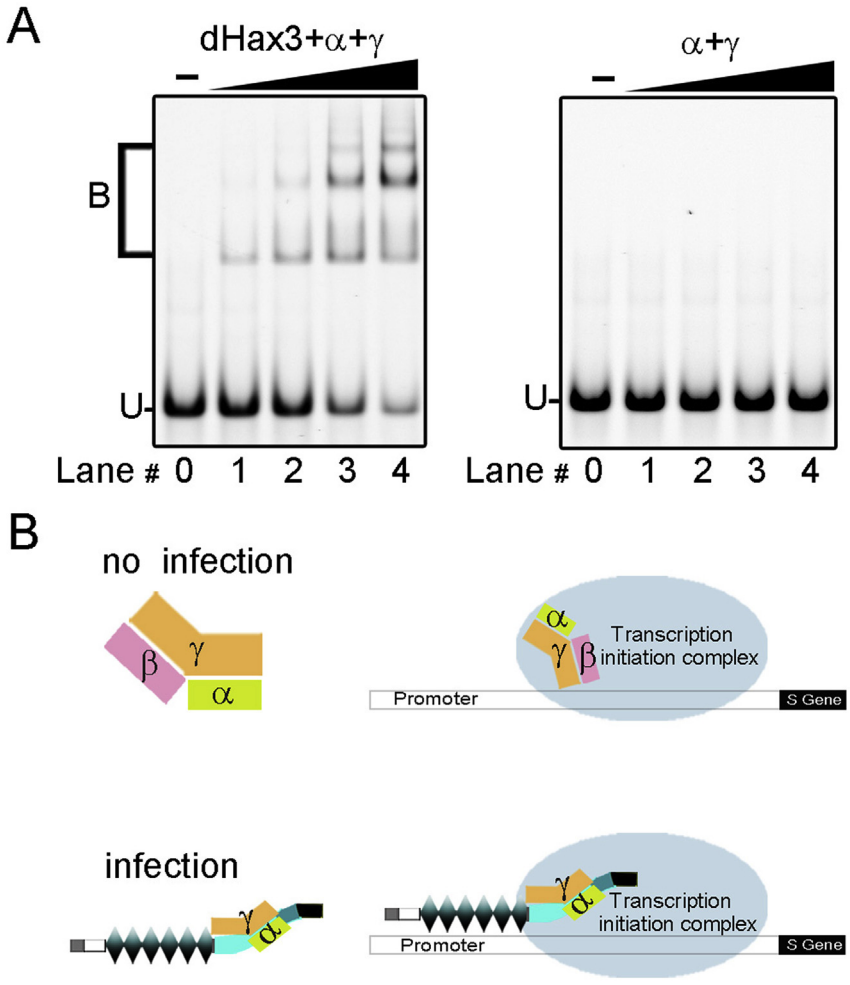

Fig. 4. Proposed model of infection with TALE-carrying bacteria. (A) The DNA binding activity of dHax3+α+γ and α+γ complexes revealed by EMSA. The final protein concentrations of lanes 0–4 are 0, 0.25, 0.5, 1, and 1.5 mM. B, bound; U, unbound. (B) Proposed model of infection with TALE-carrying bacteria.

revealed that TALE-carrying bacteria recruit host TFIIAγs, thereby inducing diseases in many plant [11,20]. In eukaryotic transcription initiation, TFIIA exists as a ternary complex comprising α, β and γ subunits to stabilize the binding of TFIID (TBP component) to the TATA box, triggering the assembly of the preinitiation complex. Whether TALEs recruit TFIIAα or TFIIAβ, or both, during *Xanthomonas* infection remains unknown. In this study, we biochemically demonstrate that TALEs directly interact with the OsTFIIA (α+γ) subcomplex but not the holo-OsTFIIA (αβ+γ/α+β+γ) ternary complex. The TFB motifs of TALEs, which are highly conserved among *Xanthomonas* species (Supplemental Fig. 4), predominantly contribute to OsTFIIA (α+γ) subcomplex interactions (Fig. 3). Moreover, the reconstituted TALEs+α+γ complex retained DNA binding activity. Based on these results, we propose a model for TALE-mediated transcriptional activation of downstream susceptibility genes. That is, without bacterial infection, TFIIA normally exists as a ternary complex to initiate transcription, whereas upon bacterial infection, the injected TALEs may hijack only the α+γ subunits but not the β subunit of TFIIA to reconstitute the TALEs+α+γ hetero-ternary transcription complex, which binds target gene promoters. By mimicking the function of the basal TFIIAβ subunit, TALEs successfully recruit other transcription factors to assemble the preinitiation complex to activate the transcription of host disease susceptibility genes (Fig. 4B). Future studies determining the structure of TALEs+α+γ or TFB+α+γ will reveal the underlying molecular mechanism of infection with TALE-carrying bacteria. Together, these findings suggest a new mechanism for infection with TALE-carrying bacteria, which can provide new insights into host-microbe interactions and applicable strategies for breeding high-resistance crop varieties.

Conflicts of interest

None.

Acknowledgments

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Appendix A. Supplementary data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.bbrc.2018.01.059.

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