Alternative plant host defense against transposon activities occurs at the post-translational stage

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Abstract

The *Antirrhinum* DNA transposon Tam3 uniquely demonstrates low temperature-dependent transposition (LTDT), so transposition does not occur at high temperatures. We previously showed that the detainment of Tam3 transposase (TPase) at the plasma membrane occurs when transposition is inactive, and that TPase is released at the permissive state of Tam3 transposition. LTDT of Tam3 is attributed to interactions between Tam3 and its host. In this addendum, we propose a model to explain the LTDT of Tam3, which is regarded as an equilibrium state reached between the host and parasite to maximize the fitness of both.

Key words: Transposon Tam3; low temperature-dependent transposition (LTDT); post-translational control; BED-zinc finger (Znf-BED) domain; co-evolution; plasma membrane

Plant genomes are occupied by massive transposable elements (TEs), which can lead to conflicts following the generation of an active TE. TEs must reproduce to ensure their propagation and survival, but this is detrimental for the host genome so they are rigorously controlled by powerful host mutagens. Plant hosts have developed various mechanisms to prevent the transposition of TEs and maintain the stability and integrity of their own genomes, including DNA methylation[1, 2], histone modification[3], mRNA degradation[4], and translation inhibition[5, 6]. These known mechanisms are

epigenetic-dependent, but we recently discovered a novel mechanism for controlling TE activity that is independent of epigenetic regulation[7].

Unlike the majority of transposons, Tam3 in *Antirrhinum* exhibits the unusual and remarkable feature of low temperature-dependent transposition (LTDT) that is activated at low growth temperatures (around 15°C) and inhibited at high ones (above 25°C)[8, 9]. Of particular note, we found that LTDT of Tam3 is epigenetic-independent and is associated with the temperature-dependent nuclear transfer of Tam3 transposase (TPase)[7, 10]. A low temperature allows some amount of TPase to enter the nuclei, resulting in the transposition of Tam3, but high temperatures inhibit this nuclear import, causing the silencing of Tam3[7, 10]. Our data revealed that host factor(s) take part in this process and that at high temperatures they firmly detain Tam3 TPase at the plasma membrane (PM) through protein–protein interactions to inhibit transposition[7]. However, some amount of TPase escapes this low-temperature regulation, most likely through the down-regulation of host factor(s). Because the expression of Tam3 *TPase* was previously shown to be similar under low and high temperatures[11], we deduced that the host factor(s) were temperature-responsive.

Based on these findings, we propose a model to explain LTDT (Fig. 1). Several pieces of evidence show that TE activity can be rapidly silenced because epigenetic defenses respond quickly to TE activation[12]. Indeed, reactivated elements from the retrotransposon Evadé were shown to be completely silenced after ~15 host generations[13]. Because *Antirrhinum* Tam3 has maintained its host–parasite relationship over a long evolutionary time course[14, 15], this suggests that an evolutionary equilibrium has been reached between Tam3 and *Antirrhinum*, which may enable the fitness of both host and parasite to be maximized.

We found that the host factor(s) detain Tam3 TPase at the PM through the BED-zinc finger (Znf-BED) domain located in the N-terminal region of the TPase (Fig. 1)[7]. The Znf-BED domain is common to many TPases of hAT superfamily DNA transposons, including Tam3[7], and is capable of binding to its own element DNAs to initiate transposition[16-18]. Interestingly, our data also suggested that Tam3 TPase has the ability to bind proteins. In *Antirrhinum*, the Znf-BED domain of Tam3 TPase has

bidirectional functions in regulating the activity of Tam3, which depends on the subcellular localization of the TPase. Besides its DNA binding ability in targeting transposon sequences to the nucleus, the Tam3 Znf-BED domain also facilitates the interaction with certain host factor(s) to inhibit TPase nuclear transport, resulting in the inactivation of Tam3 (Fig. 1)[7]. This epigenetic-independent post-translational control is a novel means of transposon silencing.

We speculate that the bidirectional functions of the Tam3 TPase Znf-BED domain have been acquired during the co-evolution between Tam3 and its host[7], and confirm the hypothesis that TE cis-elements are foci for an evolutionary arms race between TEs and host defenses[12]. Moreover, the post-translational suppression of TPase is not the only example of this between Tam3 and *Antirrhinum*. Previous analyses of Ac and Mutator transposons in maize revealed that excess TPase production caused aggregation and transpositionally inactive forms of the proteins[19-21]. This autonomous regulation might be concerned with the potential protein–protein binding ability of TPase. Accordingly, the interaction between TPase and host factor(s) at the post-translational level might occur in a wide range of organisms with longstanding evolutionary relationships with TEs.

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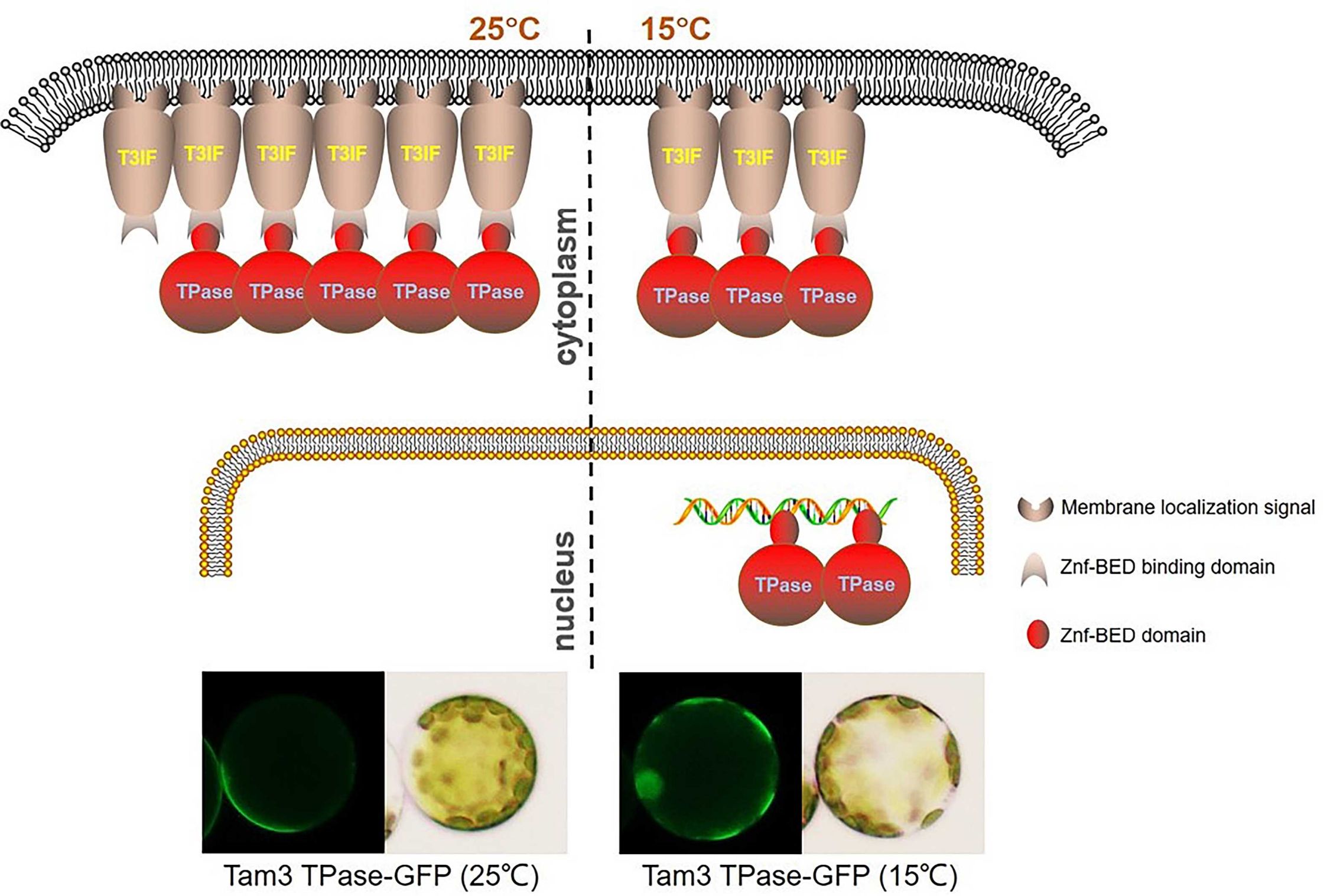
Figure Legend

A model for the low-temperature dependent transposition of Tam3.

At high temperatures, the transposition activity of Tam3 is completely silenced by the host factor(s) (Tam3 TPase interacting factor(s), T3IF) detaining Tam3 TPase at the plasma membrane. At low temperatures, the host factor(s) is not fully functional so some amount of Tam3 TPase can enter the nucleus to initiate the transposition of Tam3.


25°C
15°C
T3IF
TPase
cytoplasm
nucleus
Membrane localization signal
Znf-BED binding domain
Znf-BED domain
Tam3 TPase-GFP (25°C)
Tam3 TPase-GFP (15°C)