



Title	Alternative plant host defense against transposon activities occurs at the post-translational stage
Author(s)	Zhou, Hua; Kishima, Yuji
Citation	Plant signaling & behavior, 12(5), e1318238 https://doi.org/10.1080/15592324.2017.1318238
Issue Date	2017-06-05
Doc URL	http://hdl.handle.net/2115/70683
Rights	This is an Accepted Manuscript of an article published by Taylor & Francis in Plant Signaling & Behavior on May 2017, available online: https://www.tandfonline.com/doi/full/10.1080/15592324.2017.1318238
Type	article (author version)
File Information	Zhou&Kishima addendum for PSB 4.6.2017.pdf



[Instructions for use](#)

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

Hua Zhou* and Yuji Kishima

Laboratory of Plant Breeding, Research Faculty of Agriculture, Hokkaido University, Sapporo 060-8589, Japan

***Corresponding author: zhouhua@abs.agr.hokudai.ac.jp**

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 modification³, mRNA degradation⁴, 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 regulation⁷.

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 transposition⁷. 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 temperatures¹¹, 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 activation¹². Indeed, reactivated elements from the retrotransposon *Evadé* were shown to be completely silenced after ~15 host generations¹³. 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 Tam3⁷, and is capable of binding to its own element DNAs to initiate transposition¹⁶⁻¹⁸. 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 host⁷, and confirm the hypothesis that TE cis-elements are foci for an evolutionary arms race between TEs and host defenses¹². 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 proteins¹⁹⁻²¹. 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.

References

1. Pikaard CS, Haag JR, Ream T, Wierzbicki AT. Roles of RNA polymerase IV in gene silencing. *Trends in Plant Science* 2008; 13:390-7.
2. Matzke MA, Mosher RA. RNA-directed DNA methylation: an epigenetic pathway of increasing complexity. *Nat Rev Genet* 2014; 15:394-408.
3. Saze H, Tsugane K, Kanno T, Nishimura T. DNA Methylation in Plants: Relationship to Small RNAs and Histone Modifications, and Functions in Transposon Inactivation. *Plant and Cell Physiology* 2012; 53:766-84.
4. Zhang B, Wang Q, Pan X. MicroRNAs and their regulatory roles in animals and plants. *Journal of Cellular Physiology* 2007; 210:279-89.
5. Li S, Liu L, Zhuang X, Yu Y, Liu X, Cui X, et al. MicroRNAs Inhibit the Translation of Target mRNAs on the Endoplasmic Reticulum in Arabidopsis. *Cell* 2013; 153:562-74.
6. Iwakawa H-o, Tomari Y. Molecular Insights into microRNA-Mediated Translational Repression in Plants. *Molecular Cell* 2013; 52:591-601.
7. Zhou H, Hirata M, Osawa R, Fujino K, Kishima Y. Detainment of Tam3 Transposase at Plasma Membrane by Its BED-Zinc Finger Domain. *Plant Physiology* 2017; 173:1492-501.
8. Harrison BJ, Fincham JRS. Instability at the Pal locus in *Antirrhinum majus*. *Heredity* 1964; 19:237-58.
9. Carpenter R, Martin C, Coen ES. Comparison of genetic behaviour of the transposable element Tam3 at two unlinked pigment loci in *Antirrhinum majus*. *Molecular and General Genetics MGG* 1987; 207:82-9.

10. Fujino K, Hashida SN, Ogawa T, Natsume T, Uchiyama T, Mikami T, et al. Temperature controls nuclear import of Tam3 transposase in *Antirrhinum*. *Plant J* 2011; 65:146-55.
11. Uchiyama T, Saito Y, Kuwabara H, Fujino K, Kishima Y, Martin C, et al. Multiple regulatory mechanisms influence the activity of the transposon, Tam3, of *Antirrhinum*. *New Phytologist* 2008; 179:343-55.
12. Bousios A, Gaut BS. Mechanistic and evolutionary questions about epigenetic conflicts between transposable elements and their plant hosts. *Current Opinion in Plant Biology* 2016; 30:123-33.
13. Mari-Ordonez A, Marchais A, Etcheverry M, Martin A, Colot V, Voinnet O. Reconstructing de novo silencing of an active plant retrotransposon. *Nat Genet* 2013; 45:1029-39.
14. Yamashita S, Takano-Shimizu T, Kitamura K, Mikami T, Kishima Y. Resistance to gap repair of the transposon Tam3 in *Antirrhinum majus*: a role of the end regions. *Genetics* 1999; 153:1899-908.
15. Kishima Y, Yamashita S, Martin C, Mikami T. Structural conservation of the transposon Tam3 family in *Antirrhinum majus* and estimation of the number of copies able to transpose. *Plant Molecular Biology* 1999; 39:299-308.
16. Hashida SN, Uchiyama T, Martin C, Kishima Y, Sano Y, Mikami T. The temperature-dependent change in methylation of the *Antirrhinum* transposon Tam3 is controlled by the activity of its transposase. *Plant Cell* 2006; 18:104-18.
17. Mack AM, Crawford NM. The *Arabidopsis* TAG1 Transposase Has an N-Terminal

Zinc Finger DNA Binding Domain That Recognizes Distinct Subterminal Motifs. *The Plant Cell* 2001; 13:2319-32.

18. Hickman AB, Ewis HE, Li X, Knapp JA, Laver T, Doss AL, et al. Structural basis of hAT transposon end recognition by Hermes, an octameric DNA transposase from *Musca domestica*. *Cell* 2014; 158:353-67.

19. Heinlem M, Brattig T, Kunze R. In vivo aggregation of maize Activator (Ac) transposase in nuclei of maize endosperm and *Petunia* protoplasts. *The Plant Journal* 1994; 5:705-14.

20. Boehm U, Heinlein M, Behrens U, Kunze R. One of three nuclear localization signals of maize Activator (Ac) transposase overlaps the DNA-binding domain. *The Plant Journal* 1995; 7:441-51.

21. Ono A, Kim S, Walbot V. Subcellular localization of MURA and MURB proteins encoded by the maize MuDR transposon. *Plant Mol Biol* 2002; 50:599-611.

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.

