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Pathogenesis of begomoviruses in Tomato

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Introduction

Tomato (*Solanum lycopersicum*, Solanaceae) is host of several viral species, according to recent counts in the encyclopedia of plant viruses.[1] More than 50% of these are DNA viruses in the genus Begomovirus of the family Geminiviridae,[1] transmitted through the phloem by feeding of whiteflies *Bemisia tabaci*. In the begomovirus genera, Tomato yellow leaf curl virus (TYLCV), possess the most cause of concern in tomato production due to documented losses reaching 40-100%,[2-4] plus the continuous spread of the vector. Begomovirus infections are a problem common to China, Mexico, the Netherlands, the United States, and Spain, the main producers of tomato in the world.[5] Also severe cases are appearing in Asia, Israel, the Mediterranean and the Middle East where TYLCV, Tomato leaf curl China virus and Tomato leaf curl New Delhi virus can occur alone mixed or in complexes with beta-satellites.[6-8]

Begomovirus infection in tomato

Three categories of symptoms, namely yellow leaf curl disease, mosaic/mottle, and leaf deformation can be established; these come with an intensity, distribution and time of appearance depending on the cultivar and the virus. In typical non-mixed infections by monopartite viruses, like TYLCV, the virulence factor is protein C4;[9] most of the C4 interactors are ribosomal or participate in ubiquitination, but others are key regulators of the plant defenses pathways; the infection induces down-regulation of genes related to basic metabolism and biosynthesis of secondary metabolites, with significative loss of transcripts for photosystem II and up-regulation of anthocyanin biosynthesis.[10-12] Production of cellulose is seriously injured in cultivars susceptible to TYLCV,[10] however rather than deploying a complete antiviral response, infected plants follow a photosynthetically poor life cycle with curled new leaves and reduced growth that reaches senescence 50-60 days post infection.[13] This is critic if the plants become infected soon after transplanting, hence among recommended management strategies is to avoid planting in seasons with high whitefly populations.[2] Noteworthy, some evidence suggests that seed transmission of TYLCV might account to a high percentage of early infections.[14]

In other pathogenic systems, for exampleTomato leaf curl China virus (TYLCCNV) and its satellites, symptoms are modulated by βC1. TYLCCNV itself or with an alpha satellite induces mild symptoms, which turn into severe apical leaf curling if a beta-satellite is added to the complex;[15,16] overexpression βC1 protein in healthy plants produces leaf curling, vein swelling, and blistering of leaves, indicating that, after all, severity in leaf curl diseases is a result of impaired cell wall biosynthesis. The interactions of βC1 proteins, in general, involve polymerases and RNA methylases that participate in RNA silencing plus proteins in the ubiquitination pathway;[15,17] their pathogenicity is negatively regulated by the sucrose-nonfermenting kinase 1 (SnRK1),[15,18] which also phosphorylates proteins Rep and C2, as well, and was linked with resistance to TGMV[19] independent of ty- genes. During interactions of the TYLCCNV with its satellites, expression of genes involved in starch metabolism and glycosaminoglycans synthesis is more often disturbed in presence of the beta-satellite if the alpha satellite is absent,[16] all together suggesting metabolic changes that might lead to impairments in cell-wall biosynthesis.

Bipartite begomoviruses, particularly those in the clade of Abutilon mosaic virus, trend to produce mild yellowing in which the nuclear shuttle protein (NSP) acts as suppressor of antiviral response. NSP main target is a transmembrane leucine rich protein involved in viral PAMPs identification, named NIK1 for NSP-interacting kinase I;[20,21] NIK1 has direct interaction with ribosomal protein L10A (rpL10A), in such a way that its over expression results in severe shut down of the translational machinery;[22] ectopic expression of NIK1 in tomato delays the infection and reduces manifestations of Tomato yellow spot virus, other bipartite begomovirus in the same clade, whereas in tobacco leaves infected with Tomato golden mosaic virus (TGMV) rpL10A distribution is distorted; additionally, Arabidopsis *nik1* null mutants are more susceptible to Cabbage leaf curl virus (CaLCuV),[23] which belongs to in a different clade of American begomoviruses, all together indicating that NIKI plays a central role in response to these pathogens.

The transcriptional activity induced by bipartite begomoviruses in tomato is likely to resemble that of Pepper golden mosaic virus in *Capsicum annum* and CaLCuV in *A. thaliana,* characterized by an increased expression of plant defense genes in the ethylene, jasmonic and oxidative stress pathways, but with down-regulation of particular steps in the ethylene pathway.[24] Conversely, Navqi and co-workers found that polyubiquitinators, 40S ribosomal proteins, ethylene response proteins, and a cell wall protein precursor' are among the genes differentially expressed in infections by the Old Word bipartite virus Tomato leaf curl New Delhi virus.[25]

Conclusion

Begomovirus pathogenicity is determined by a complex interaction between viral and host proteins' that together cause general and specific symptomatology. In emergent of plant viral diseases, genetic changes in viral proteins or interaction between proteins of two or more co-infecting viral species, allow new or stronger reactions that favor viral phatogenesis. Under these circumstances, the symptomatology

is no longer modulated by the typical pathogenicity determinants and becomes dependent on genetic changes such as point mutations,[26,27] recombination events,[28,29] rearrangements and coinfections.[30-37]

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Conflicts of interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

1. Sastry KS, Mandal B, Hammond J, et al. Encyclopedia of plant viruses and viroids. *Springer, New Delhi.* 2019.
2. Fonsah EG, Yu C, Diffie S, Srinivansan RB, Riley D. Economic productivity and profitability analysis for whiteflies and Tomato Yellow Leaf Curl Virus (TYLCV) management options. *Journal of agriculture and environmental sciences*. 2018;7(1):1–9.
3. Hanssen IM, Lapidot M, Thomma BP. Emerging viral diseases of tomato crops. *Mol Plant Microbe Interact*. 2010;23(5):539–548.
4. Beam K and Ascencio–Ibañez JT. Geminivirus Resistance: A Minireview. *Front Plant Sci*. 2020;11:1131.
5. Guan Z, Biswas T, Wu F. *The US tomato industry: An overview of production and trade*. FE1027. Gainesville, FL: University of Florida, Institute of Food and Agricultural Sciences. 2017.
6. Anfoka G, Ahmad FH, Altaleb M, Al Shhab M. Detection of Satellite DNA Beta in Tomato Plants with Tomato Yellow Leaf Curl Disease in Jordan. *Plant Dis*. 2014;98(7):1017.
7. Shahid, Muhammad Shafiq. Tomato yellow leaf curl virus interaction with betasatellites – a global threat to tomato production. *Plant Pathology*. 2020;69.
8. Gelbart D, Chen L, Alon T, Dobrinin S, et al. The recent association of a DNA betasatellite with Tomato yellow leaf curl virus in Israel – A new threat to tomato production. *Crop Protection*. 2020;128:104995.
9. Kim N, Kim J, Bang B, et al. Comparative Analyses of Tomato yellow leaf curl virus C4 Protein–Interacting Host Proteins in Healthy and Infected Tomato Tissues. *Plant Pathol J*. 2016;32(5):377–387.
10. Seo JK, Kim MK, Kwak HR, et al. Molecular dissection of distinct symptoms induced by tomato chlorosis virus and tomato yellow leaf curl virus based on comparative transcriptome analysis. *Virology*. 2018;516:1–20.
11. Huang Y, Ma HY, Huang W, et al. Comparative proteomic analysis provides novel insight into the interaction between resistant vs susceptible tomato cultivars and TYLCV infection. *BMC Plant Biol*. 2016;16(1):162.
12. Miozzi L, Napoli C, Sardo L, Accotto GP. Transcriptomics of the interaction between the monopartite phloem–limited geminivirus tomato yellow leaf curl Sardinia virus and Solanum lycopersicum highlights a role for plant hormones, autophagy and plant immune system fine tuning during infection. *PLoS One*. 2014;9(2):e89951.
13. Gorovits R, Moshe A, Amrani L, Kleinberger R, Anfoka G, Czosnek H. The six Tomato yellow leaf curl virus genes expressed individually in tomato induce different levels of plant stress response attenuation. *Cell Stress Chaperones*. 2017;22(3):345–355.
14. Kil EJ, Kim S, Lee YJ, et al. Tomato yellow leaf curl virus (TYLCV–IL): a seed–transmissible geminivirus in tomatoes. *Sci Rep*. 2016;6:19013.
15. Zhong X, Wang ZQ, Xiao R, et al. Mimic Phosphorylation of a βC1 Protein Encoded by TYLCCNB Impairs Its Functions as a Viral Suppressor of RNA Silencing and a Symptom Determinant. *J Virol*. 2017;91(16):e00300–17.
16. Luo C, Wang ZQ, Liu X, et al. Identification and Analysis of Potential Genes Regulated by an Alphasatellite (TYLCCNA) that Contribute to Host Resistance against Tomato Yellow Leaf Curl China Virus and Its Betasatellite (TYLCCNV/TYLCCNB) Infection in Nicotiana benthamiana. *Viruses*. 2019;11(5):442.
17. Shen Q, Liu Z, Song F, et al. Tomato SlSnRK1 protein interacts with and phosphorylates βC1, a pathogenesis protein encoded by a geminivirus β–satellite. *Plant Physiol*. 2011;157(3):1394–406.
18. Shen W, Hanley–Bowdoin L. SnRK1: a versatile plant protein kinase that limits geminivirus infection. *Curr Opin Virol*. 2021;47:18–24.
19. Shen W, Bobay BG, Greeley LA, et al. Sucrose Nonfermenting 1–Related Protein Kinase 1 Phosphorylates a Geminivirus Rep Protein to Impair Viral Replication and Infection. *Plant Physiol*. 2018;178(1):372–389.
20. Martins LGC, Raimundo GAS, Ribeiro NGA, et al. A Begomovirus Nuclear Shuttle Protein–Interacting Immune Hub: Hijacking Host Transport Activities and Suppressing Incompatible Functions. *Front Plant Sci*. 2020;11:398.
21. Ferreira MA, Teixeira RM, Fontes EPB. 2021. Geminivirus–Host interactions: Action and reaction in receptor–mediated antiviral immunity. *Viruses*. 2021;13:840.
22. Zorzatto C, Machado JP, Lopes KV, et al. NIK1–mediated translation suppression functions as a plant antiviral immunity mechanism. *Nature*. 2015;520(7549):679–682.
23. Carvalho CM, Santos AA, Pires SR, et al. Regulated Nuclear Trafficking of rpL10A Mediated by NIK1 Represents a Defense Strategy of Plant Cells against Virus. *PLoS Pathog*. 2008;4(12):e1000247.
24. Góngora–Castillo E, Ibarra–Laclette E, Trejo–Saavedra DL, Rivera–Bustamante RF. Transcriptome analysis of symptomatic and recovered leaves of geminivirus–infected pepper (Capsicum annuum). *Virol J*. 2012;27(9):295.
25. Naqvi AR, Sarwat M, Pradhan B, et al. Differential expression analyses of host genes involved in systemic infection of Tomato leaf curl New Delhi virus (ToLCNDV). *Virus Res*. 2011;160(1–2):395–9.
26. Zhao W, Ji Y, Wu S. et al. Single amino acid in V2 encoded by TYLCV is responsible for its self–interaction, aggregates and pathogenicity. *Sci Rep*. 2018;8:3561.
27. Wang D, Zhang X, Yao X, et al. A 7–Amino–Acid Motif of Rep Protein Essential for Virulence Is Critical for Triggering Host Defense Against Sri Lankan Cassava Mosaic Virus. *Mol Plant Microbe Interact*. 2020;33(1):78–86.
28. Xie Y, Zhao L, Jiao X, et al. A recombinant begomovirus resulting from exchange of the C4 gene. *J Gen Virol*. 2013;94(Pt 8):1896–1907.
29. Shilpi S, Kumar A, Biswas S, et al. A recombinant Tobacco curly shoot virus causes leaf curl disease in tomato in a north–eastern state of India and has potentiality to trans–replicate a non–cognate betasatellite. *Virus Genes*. 2015;50(1):87–96.
30. Chakraborty S, Vanitharani R, Chattopadhyay B, Fauquet CM. Supervirulent pseudorecombination and asymmetric synergism between genomic components of two distinct species of begomovirus associated with severe tomato leaf curl disease in India. *J Gen Virol*. 2008;89(Pt 3):818–828.
31. Rentería–Canett I, Xoconostle–Cázares B, Ruiz–Medrano R, Rivera–Bustamante RF. Geminivirus mixed infection on pepper plants: synergistic interaction between PHYVV and Pep GMV. *Virol J*. 2011;8:104.
32. Sufrin–Ringwald T, Lapidot M. Characterization of a synergistic interaction between two cucurbit–infecting begomoviruses: Squash leaf curl virus and Watermelon chlorotic stunt virus. *Phytopathology*. 2011;101(2):281–9.

33. Basu S, Singh AK, Singh D, Sahu SK, Chakraborty S. Role of viral suppressors governing asymmetric synergism between tomato–infecting begomoviruses. *Appl Microbiol Biotechnol*. 2021;105(3):1107–1121.

34. Li T, Huang Y, Xu ZS, et al. Salicylic acid–induced differential resistance to the Tomato yellow leaf curl virus among resistant and susceptible tomato cultivars. *BMC Plant Biol*. 2019;19(1):173.

35. Melgarejo TA, Kon T, Rojas MR, et al. Characterization of a new world monopartite begomovirus causing leaf curl disease of tomato in Ecuador and Peru reveals a new direction in geminivirus evolution. *J Virol*. 2013;87(10):5397–413.

36. Pérez–Padilla V, Fortes IM, Romero–Rodríguez B, et al. Revisiting Seed Transmission of the Type Strain of Tomato yellow leaf curl virus in Tomato Plants. *Phytopathology*. 2020;110(1):121–129.

37. Romay G, Geraud–Pouey F, Chirinos DT. et al. Molecular variability of Merremia mosaic virus infecting tomatoes in Venezuela. *Australasian Plant Dis Notes*. 2016;11:11.

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