VirusDis. (April–June 2019) 30(2):245–251
https://doi.org/10.1007/s13337-019-00521-w

ORIGINAL ARTICLE

In silico analyses of molecular interactions between groundnut bud necrosis virus and its vector, *Thrips palmi*

Shounak S. Jagdale[1] · **Amalendu Ghosh**[2]

Received: 27 December 2018 / Accepted: 13 March 2019 / Published online: 26 March 2019

Abstract Groundnut bud necrosis virus (GBNV) is an economically important tospovirus transmitted by *Thrips palmi* (Thysanoptera: Thripidae). The current understanding of thrips-tospovirus interactions is largely based on the tomato spotted wilt virus-*Frankliniella occidentalis* relationship. Only limited information is available for the GBNV-*T. palmi* system. In the present study, available genome data of *T. palmi* and GBNV were used to predict the protein partners that may play a crucial role in the internalization of GBNV virions into thrips cells. Computational analyses showed that the GBNV precursor glycoprotein bears a signal peptide of 24 amino acids and a secondary cleavage site at position 434–435 separates the amino-terminal mature glycoprotein (G_N) from the carboxyl-terminal glycoprotein (G_C). Potential interactions of GBNV glycoproteins were predicted with *T. palmi* enolase, cathepsin, C-type lectin, clathrin and vacuolar ATP synthase subunit E. The in silico analyses suggested that C-type lectin is the primary cellular receptor to interact with GBNV-G_N. After receptor binding, virus particles probably enter vector cells by clathrin-mediated endocytosis. This is the first in silico evidence of GBNV-*T. palmi* protein interaction.

Keywords Melon thrips · GBNV · Modeling · Docking · Virus-vector relationship · Protein–protein interaction

Introduction

Groundnut bud necrosis virus (GBNV; genus *Tospovirus*, family *Peribunyaviridae*, order *Bunyavirales*) is one of the predominant plant viruses causing substantial losses of agricultural produce worldwide. GBNV accounts for losses of US$ 89 million annually with 70–90% yield loss in groundnut and 30% in potato [29, 22]. Infection of GBNV causes mild chlorotic spots on young quadrifoliate leaves, and subsequently necrosis and chlorotic rings develop. Secondary symptoms include stunting, axillary shoot proliferation, and malformation of leaflets. *Thrips palmi* (Thysanoptera: Thripidae) is known to vector GBNV [40, 9]. Not much is known about the relationships of thrips and tospoviruses. Based on the well-studied tomato spotted wilt virus (TSWV)-*Frankliniella occidentalis* relationship, it is understood that virus propagates in the thrips body and is transported through the midgut to the salivary gland [38, 39, 43, 44]. Virus transmission occurs exclusively by adult thrips while virus acquisition takes place during the early larval stages [30]. Differential transcriptomics and proteomics of *F. occidentalis* in response to TSWV have predicted few probable receptors for TSWV [3, 33]. The glycoprotein G_N of TSWV is involved in the primary binding to the thrips midgut epithelial cells, whereas G_C is necessary for fusion [42–44, 7]. But no such study has been undertaken to unveil the interaction between GBNV and *T. palmi*. The primary reason for this was a lack of structural data of GBNV proteins and receptors of *T. palmi*. In the present study, we have utilized the available data resources to understand the protein–protein interactions between

Electronic supplementary material The online version of this article (https://doi.org/10.1007/s13337-019-00521-w) contains supplementary material, which is available to authorized users.

✉ Amalendu Ghosh
amal4ento@gmail.com

[1] Institute of Bioinformatics and Biotechnology, Savitribai Phule Pune University, Pune 411007, India

[2] Insect Vector Laboratory, Advanced Centre for Plant Virology, ICAR-Indian Agricultural Research Institute, New Delhi 110012, India