

# Neofusicoccum Parvum Causes a Pear Stem Canker and Twig Dieback Disease in China's Mainland, Which has been Identified and Characterised

Tong Zhang\*

Guangdong Province Key Laboratory of Microbial Signals and Disease Control, College of Plant Protection, South China Agricultural University, Guangzhou, 510642, China

## Letter to Editor

The pears (*Pyrus spp.*) are one of China's most popular fruits, but their supply is under threat from deadly illnesses. In this paper, we describe two cases of stem canker and twig dieback disease on pear plants in Guangxi and Jiangsu provinces, which resulted in the death of pear seedlings (about 10% of total plants). The fungus *Neofusicoccum parvum* was identified as the disease's causal agent in these two locations using a combination of morphological and molecular diagnostics, as well as a pathogenicity test. The isolates were classified into two clades: the CY-2 isolate and the other four isolates, ZL-4, BM-9, BM-10, and BM-12, may have split into two *N. parvum* groupings [1]. For further examination, two representative isolates (CY-2 and ZL-4) were chosen. The best temperature for in vitro infection on pear trees of these two isolates was around 25°C, according to our findings. In vitro, both CY-2 and ZL-4 could infect many sand pear cultivars as well as other horticultural plants, however CY-2 was more virulent on certain pear varieties, including Nanyue, Lvyun, Qiushui, and Ningmenghuang. Additionally, the efficacy of fungicides against these two isolates was assessed, with carbendazim and flusilazole proving to be the most efficient fungicides in preventing the growth of these fungal infections. These findings, taken together, characterise the *N. parvum* species and offer viable solutions for the disease's future management [2].

Virus-derived small interfering RNAs (vsiRNAs) can trigger symptom development in plants by targeting host genes. The most devastating rice-infecting virus, Southern rice black-streaked dwarf virus (SRBSDV), produces severe stunting and poorly developed roots in rice plants, posing a substantial danger to rice output. We show that a vsiRNA (vsiR-S9-18) derived from SRBSDV genome segment 9 binds the transcription factor ROC1 in rice in this work. In rice plants, SRBSDV infection resulted in the synthesis of vsiR-S9-18 and the down regulation of ROC1. The particular connection of vsiR-S9-18 with ROC1 was further shown by transient expression of vsiR-S9-18 in rice protoplasts and tobacco leaves. Furthermore, the ROC1-knockout rice plants had shorter roots, which was similar to the phenotype of SRBSDV infection-induced root growth suppression. We believe that vsiR-S9-18 decreases root elongation by inhibiting ROC1, a known regulator of root growth. This discovery adds to our knowledge of the role of vsiRNA in viral illness progression and aids in the development of new antiviral strategies [3].

Plant pathogenic oomycetes are a major hazard to crop production and ecosystems all over the world. Oomycete pathogens produce a number of effectors to modulate plant immunity during infection. Many of these effectors, which have been identified as avirulence gene candidates, will use components of the immune system to cause plant cell death. Plants' response is referred to as effector-triggered immunity (ETI). The discovery of virulence genes in pathogenic oomycete species pave the door for further research into their virulence function and the discovery of comparable R gene repertoires in resistant plants [4]. We investigated the requirements of ETI signalling components to elicit cell death in *N. benthamiana* by screening eight cell death-inducing effectors from oomycete species. PcRXLR25, PsAvh163,

PsAvh241, and PsCRN63-triggered cell death were eliminated when NbHSP90 was silenced, whereas PcRXLR25, PsAvh163, PsAvh241, and PsCRN63-triggered cell death were all abolished when NbHSP90 was silenced. EDS1, NDR1, NRG1, and ADR1 have no influence on cell death triggered by the examined effectors. NRC2/3/4 is required for cell death triggered by PcRXLR25 and PsAvh163, indicating that these two effectors are avirulence protein candidates. Finally, we discovered that auto-activated NRC2/3/4 induced hypersensitivity by requiring SGT1 and HSP90 [5].

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\*Corresponding author: Tong Zhang, Guangdong Province Key Laboratory of Microbial Signals and Disease Control, College of Plant Protection, South China Agricultural University, Guangzhou, 510642, China, E-mail: zhangtong@scau.edu.cn

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