DOI 10.18699/SBB-2022-365

Transcriptional profiling of fusarium wilt in flax

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Key words: Fusarium wilt, flax, transcriptional profiling, cytochrome, host-parasite interaction

Motivation and Aim: Fusarium wilt of flax is an aggressive disease caused by soil-borne fungal pathogen *Fusarium oxysporum* f. sp. *lini* posing a major threat to flax production worldwide [1–3]. Recently we reported chromosome-level assemblies of 5 highly pathogenic *F. oxysporum* f. sp. *lini* strains, including a highly virulent MI39 isolate, thus providing a background for detailed genomic studies of host-pathogen interaction mechanisms on multiple levels [4, 5]. Here, we present a first insight into molecular mechanisms involved in response to infection. Such data may be of use when integrated with the infection resistance markers emerging from GWAS studies, thus opening up new possibilities for disease control and prevention.

Methods and Algorithms: In this study we examined susceptible and resistant flax varieties, namely, LM98 and Atalante, infected by MI39 *Fusarium* isolate. Uninfected plants and pure culture of the fungus served as controls. Flax samples were harvested on the third and the fifth day post inoculation (dpi). A total RNA mixture (i.e., flax and fungus) was extracted from infected plant roots and sequenced. The RNASeq data was processed in parallel with kallisto/sleuth suite [6, 7] using flax and *Fusarium* gene models. The functional annotation and GO enrichment analyses of resulting sets of differentially expressed genes was done with XGR package [8].

Results: *Fusarium* infection produces a diverse spectrum of transcriptional states both of the host and the pathogen. The resistant plants exhibited a sharp, prolonged response to the infection as early as the third dpi. In line with previous work [9–11], we observed changes in expression of genes controlling membrane remodelling, secondary metabolite synthesis, synthesis of lipids and cytochrome-related proteins, transcription factors in response to infection. Furthermore, an activation of Germin-like genes provides a clear indication of flax response to the biotic stress associated with disease progression. In contrast, the susceptible plants provided no sign of significant transcriptional response on the third dpi. However, the delayed changes in transcriptional profiles were visible on the fifth dpi, manifesting in expression changes of genes controlling protein kinases, transmembrane transport, cytochrome-related genes and glycoproteins associated with plant resistance to diseases.

Upon invasion the fungus exhibits upregulation of genes associated with pathogen penetration through the host's cell walls and further progression (e.g., proteases and cellular energetics) irrespective of the host's resistance status.

Conclusion: Previous research [12] has highlighted the significance of cytochrome-related genes in plant defense. Also, earlier we reported QTN in a gene encoding flax' cytochtrome P450 protein associated with resistance to *Fusarium* infection [13]. Our findings further emphasize the indispensable role the cytochrome superfamily in plant pathogen response. Furthermore, our results also provide first insights on the whole genome level into transcriptional activity of the plant pathogen upon penetration into host. The data generated in the framework of this project lay a solid foundation for in-depth delineation of the regulatory mechanisms of plant-pathogen interaction.

Acknowledgements: This work is supported by Peter the Great St. Petersburg Polytechnic University in the framework of Russian Federation's Priority Strategic Academic Leadership Programme (Agreement 75-15-1038-2021-1333).

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