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Fusarium wilt of flax: multi-omics elucidation of host-specific virulence and epigenetically regulated resistance

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Motivation and aim: Fusarium wilt, a devastating disease affecting flax (*Linum usitatissimum*), is attributed to the host-specific pathogen *Fusarium oxysporum* f. sp. *lini* (Fol). Although selective breeding has successfully developed resistant flax varieties, the molecular mechanisms governing flax immunity remain poorly understood. This study employs a multi-omics approach to (1) pinpoint genomic loci and virulence factors specific to *F. oxysporum* f. sp. *lini,* (2) analyze transcriptional and epigenetic responses to infection in both resistant and susceptible flax cultivars, and (3) elucidate the spatial-temporal dynamics of fungal colonization throughout the disease progression.
Methods and algorithms: Fungal virulence was assessed in controlled greenhouse conditions using reference flax cultivars that show varying levels of susceptibility. To identify host-specific genomic regions in *F. oxysporum* f. sp. *lini*, orthogroup identification was conducted with mmseqs2 using a dataset comprising 556 *F. oxysporum* genomes from strains infecting different hosts. Infection progression was monitored in the plant using GFP-tagged *F. oxysporum* (MI39 strain). Transcriptome and methylome data were analyzed using a bioinformatics pipeline that included Kallisto, DESeq2, and other tools for differential expression and methylation profiling.
Results: We identified several specific orthogroups of *F. oxysporum* f. sp. *lini* that are functionally annotated as potential virulence factors. In resistant cultivars, the challenge posed by the pathogen resulted in significant demethylation events in genes associated with immune signaling pathways. Expression profiling of SIX effector genes, which are critical for fungal virulence, demonstrated that SIX13 was uniquely upregulated during the course of plant infection. Additionally, resistant cultivars were able to effectively limit fungal colonization to the root tissue, thereby preventing systemic movement to the stems.
Conclusions: This study enhances our comprehension of flax-Fusarium interactions by identifying genomic signatures associated with host specificity, the dynamic epigenetic regulation of defense mechanisms, and the activity of effector genes crucial for pathogenicity. The discovery of fungal-specific genes and immune-related methylation markers lays the groundwork for the development of molecular diagnostics aimed at detecting *F. oxysporum* f. sp. *lini*. Additionally, these insights could inform strategies to bolster flax resistance through either conventional breeding approaches or advanced genetic engineering techniques.
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