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Fusarium head blight of wheat: Involvement of salicylic acid and jasmonic acid in disease resistance response

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Abstract

Fusarium head blight (FHB) is a devastating disease affecting maize and small grain cereals mainly caused by the hemi-biotrophic fungus *Fusarium graminearum*. Infection of wheat results in severe grain yield losses and trichothecene contamination (e.g. deoxynivalenol). This is not only toxic for the plant itself, but more importantly also for humans and livestock. Controlling FHB outbreaks and the disease is difficult and achieved by cultural techniques and the use of chemicals. Therefore, breeding of highly resistant cultivars is the most effective way of coping with the disease. FHB resistance in hexaploid wheat is quantitative. More than 200 QTL have been detected in various studies. Only a few are stable in different environments and diverse genetic backgrounds and therefore useful for breeding. An accepted resistance mechanism is the prevention of fungal spread within the spike (type II resistance). However, the underlying molecular mechanisms are still poorly understood. Further research at the molecular level of the *Triticum aestivum* - *F. graminearum* interaction will deepen our understanding and forward FHB resistance breeding.

In our study, four bread wheat genotypes differing in FHB resistance level were evaluated: highly-resistant 'Sumai 3', moderately-resistant 'Dream', moderately-susceptible 'Lynx' and highly-susceptible 'Florence Aurore'. Single-floret inoculation was used to infect wheat heads at early anthesis (1000 *F. graminearum* macroconidia per floret, or distilled water). Spike samples were collected at 12 time points after inoculation, i.e. 0, 0.33, 1, 1.33, 2, 3, 4, 5, 7, 14, 21 days after inoculation and at maturity. Furthermore, spike samples were dissected in four subsamples, which were analyzed independently: inoculated spikelets, rachis, uninoculated spikelets and upper stem. Fresh weights were recorded. Quantification of salicylic acid, jasmonic acid, fungal biomass and DON are in progress. Also, expression analysis of marker genes associated with salicylic- and jasmonic acid-dependent signaling pathways will be performed.

Comparison of *F. graminearum* and mock-inoculated subsample weights showed the importance of the rachis tissue in highly-resistant 'Sumai 3'. Here, the weight was not affected by *F. graminearum* attack, whereas in highly-susceptible 'Florence Aurore' the rachis was completely blighted 14 days after inoculation. Gene expression analysis confirmed importance of the rachis tissue. The gene expression of *Non-Expressor Of Pathogenesis-Related Gene 1* (*NPR1*) was examined. *NPR1* is a master regulator controlling immune responses in the systemic acquired resistance (SAR) pathway. Three days after inoculation the *NPR1* gene was 5-fold up-regulated compared to the water control in 'Sumai 3', and seven days after inoculation still 3.5-fold. However, this gene was not up-regulated in 'Florence Aurore'. Indicating an absence of, or late response to *F. graminearum* attack. Fungal biomass quantification using qPCR resulted in a clear differentiation between FHB resistance levels of the two cultivars. In the rachis of 'Sumai 3', 2.5-fold less *F. graminearum* biomass was detected than in 'Florence Aurore' after 21 days. Furthermore, in the 'Sumai 3' subsample from uninoculated spikelets 0.21 ng fungus per mg plant fresh weight were found at 21 days after inoculation, i.e. 4-fold less than in 'Florence Aurore'. Our results show that analyzing the dissected spike provides a more detailed insight into systemic signaling and defense response of wheat attacked by *F. graminearum*.

Keywords

Disease resistance, *Fusarium graminearum*, gene expression, NPR1, *Triticum aestivum*

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