

**PI:** Gary Muehlbauer

**PI's E-mail:** [muehl003@umn.edu](mailto:muehl003@umn.edu)

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**Project Title:** Molecular Genetics Approaches to Developing Scab Resistant Barley

### PROJECT 1 ABSTRACT

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Fusarium head blight (FHB), a fungal disease of small grain crops caused by *Fusarium graminearum*, threatens to reduce wheat and barley to economically unviable crops. The overall project goals are to develop genetic tools for increasing FHB resistance in barley. Three objectives will be addressed including: (1) characterize the impact of trichothecenes on infection and host responses; (2) identify resistant mutants; and (3) fine map and characterize the chromosome 2H bin8 and chromosome 6H bin7 FHB resistant QTL.

We developed transgenic barley overexpressing *HvUGT13248* and identified barley lines carrying mutations in *HvUGT13248*. Our results showed that the transgenic plants exhibit high levels of DON resistance in a root assay and rapid conjugation of DON to DON-3-glucoside (D3G); whereas the *HvUGT13248* mutants exhibit susceptibility to DON in a root assay and reduced conjugation of DON to D3G. We will use a strain of *F. graminearum* that provides the opportunity to track trichothecene production and infection in the transgenics and mutants. Host responses will also be examined in both the transgenics and mutants. These efforts will potentially provide novel strategies and genetic tools for DON and FHB resistance in barley.

Transgenic plants overexpressing *HvUGT13248* were developed in cv. Golden Promise, which precludes FHB screening in the field due to the inability of the spike to emerge from the boot in Upper Midwestern climates. Therefore, we introgressed the *HvUGT13248* transgene into the cv. Rasmusson and derived lines that are available for field screening. These lines will be tested in 2020 and 2021 in collaboration with Ruth Dill-Macky.

We plan to screen mutagenized populations for mutants that confer resistance to DON and FHB. Mutants that exhibit resistance will be further characterized for FHB resistance, the causative gene(s) mapped, and the lines and markers provided to breeding programs.

My laboratory, in collaboration with Kevin Smith, mapped QTL on chromosomes 2H bin8 and 6H bin 7 associated with FHB resistance. The size of both QTL intervals have been significantly reduced. We plan to continue to fine map both QTL through identifying recombinants in each region and testing the recombinants in the field for FHB resistance. These efforts will enable identifying lines and markers for breeding programs, and candidate genes for each QTL.

Stakeholders (breeders and geneticists) will benefit from our work through new genetic tools (markers, mutants, lines and transgenics) that can be used to increase FHB resistance in barley.