Project Abstract

Project Title:	**Molecular genetics approaches to developing scab resistant barley**	
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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 goal is to develop genetic tools for increasing FHB resistance in barley. Three objectives will be addressed including: (1) characterize the role of trichothecenes on infection and host responses; (2) fine map and characterize the chromosome 2H bin8 FHB resistant QTL; and (3) identify DON and FHB resistant mutants.

We have identified plants carrying mutations in *HvUGT13248* and showed that *HvUGT13248* mutants exhibit reduced conjugation of DON to D3G and increased FHB severity. Preliminary analysis suggests that *HvUGT13248* is required for type II resistance in barley. To further characterize the role of *HvUGT13248* in type II resistance and understand infection pathways in more detail, we will inoculate the *HvUGT13248* mutant and wildtype plants with a strain of *F. graminearum* that provides the opportunity to track trichothecene production and infection. To identify novel genes and mechanisms that function to detoxify or eliminate DON in plants that are unable to conjugate DON to D3G, we will examine the host response in the *HvUGT13248* mutant and wildtype. These efforts will potentially provide novel strategies and genetic tools for developing DON and FHB resistance in barley.

My laboratory, in collaboration with Kevin Smith, has mapped QTL on chromosomes 2H bin8 associated with FHB resistance. We plan to continue to fine map this 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 the QTL.

Examples of susceptibility genes that when mutated exhibit FHB resistance have been identified in wheat. To identify susceptibility genes in barley, we will screen mutagenized populations for mutants that confer resistance to DON and FHB. Our hypothesis is that susceptibility genes that are mutated will confer resistance. 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.