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Besides genes involved in defense, host genes associated with susceptibility, for example those required for pathogen growth, development and pathogenicity, also provide targets for controlling disease. With previous support from the USWBSI we identified 9-lipoxygenases (9-LOXs) as susceptibility factors in plant interaction with *Fusarium graminearum*. In *Arabidopsis thaliana* and in wheat, knockdown of 9-LOX function resulted in enhanced resistance to *Fusarium* head blight (FHB) caused by *F. graminearum*. RNA-interference (RNAi)-mediated silencing of *Lpx3* in the hexaploid wheat cv Bobwhite resulted in resistance that was characterized by lack of spread of the infection from the inoculated spikelet. However, whether the effect of *Lpx3* knockdown on promoting FHB resistance is also effective in other wheat backgrounds needs to be assessed. Furthermore, if a specific *Lpx3* homeolog is the susceptibility factor, or whether more than one *Lpx3* homeologs contribute to susceptibility remains to be determined.

The goals of this project are: (i) to confirm if knock-down of *Lpx3* in other wheat varieties can similarly enhance FHB resistance, and (ii) simultaneously to identify *Lpx3* variants that confer resistance to FHB in wheat varieties that can be utilized by breeding programs. As a first step in this direction, several TILLING mutants have been identified in the hexaploid and tetraploid wheat varieties Cadenza and Kronos, respectively that are predicted to yield prematurely truncated *Lpx3* protein. These mutants cover all three *Lpx3* homeologs on chromosomes 4A, 4B and 4D. Additional strong missense alleles are also available.

1. Isolate homozygous *Lpx3* mutant lines from the three sub-genomes, and backcross them to clear background mutations.
2. Characterize the response of *Lpx3* mutants to *F. graminearum*.
3. Develop wheat lines containing combinations of *Lpx3* mutant alleles at the homeologous chromosomes.

Homozygous lines are available for some of the *Lpx3* mutants, but not for all. Under objective 1, PCR and/or DNA sequencing will be used to follow the genotypes at the *Lpx3* locus to isolate homozygous mutants at the *Lpx3* locus on chromosome 4A, 4B and 4D. Objective 2 will assess FHB resistance and mycotoxin accumulation in individual *Lpx3* mutant lines. Under objective 3, crosses will be made between the homeologous *Lpx3* mutants on chromosome 4A, 4B and 4D with the goal of developing plants in which multiple *Lpx3* homeologs have been knocked out such that the effect of combined knockout of these *Lpx3* homeologs on FHB severity can be studied.

The proposed project is relevant to the GDER initiative of USWBSI to *identify candidate genes for resistance against FHB and/or reduced DON accumulation*. Our approach and the genes/mechanisms being targeted complement the activity of other USWBSI sponsored projects.