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Ultimately the food industry needs wheat with low deoxynivalenol (DON), the toxin produced by the fungus that causes Fusarium Head Blight (FHB). Breeders are making progress in developing cultivars with low visual symptoms of FHB. Selection is often based on FHB index (IND), with the assumption that selecting lines with low index will reduce DON concentration. In general, this happens. However, this association becomes highly variable and weak at low IND levels indicating independent genetic control once moderate resistance has been achieved. Indeed many cultivars with low visual symptoms still have unacceptable levels of DON. We need to understand variation for DON concentration among moderately resistant lines to efficiently obtain cultivars with low IND and low DON required by the wheat industry.

It has been suggested that there is resistance to DON accumulation per se (type III resistance) and variation for type III resistance could explain variation for DON among moderately resistant genotype. Type III resistance may be needed to obtain cultivars that repeatedly have with low IND and low DON. Unfortunately, there is no conclusive data characterizing this resistance. We need to determine to what extent environment, genetic (for type III), and genotype x environment interaction effects contribute to variation for DON among moderate resistance. This information is crucial to determine if it is possible to select for low DON among moderately resistant lines, or if other measures are required to reliably produce grain with low DON. Our objectives are:

2. Determine underlying mechanism for resistance to DON accumulation per se (type III resistance).

We will use a moderate sized population of moderately resistant soft wheat lines in this study to minimize the confounding affects of segregation for type I and II resistance. We will assess index of each genotype as well as the DON and fungal biomass of grain from each. We will then determine of some genotypes repeatedly have lower DON than expected based on their index and fungal biomass. In addition we will sample grain from each genotype from spikes with equal levels of infection (severity = 0, 7, 14, and 21%). This will allow us to determine how DON concentration in each genotype responds to increased disease severity and fungal biomass and how fungal biomass of each genotype responds to increase severity. Genotypes with low response have type III resistance. The lines in the study have already been genotyped with markers from key chromosome region affecting type I and II resistance. We will use association mapping to determine if these regions affect type III resistance. The study will determine 1) the heritability of type III resistance, 2) the mechanism of type III resistance (resistance to fungal infection of seed or favorable DON metabolism), and 3) if type III resistance is independent from type I and II resistance.