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PROJECT 1 ABSTRACT

(1 Page Limit)

Scab or Fusarium head blight (FHB), caused by *Fusarium graminearum* Schwabe, and sometimes by other *Fusarium* species, is a destructive disease of wheat and barley that causes significant reductions in yield and quality in many wheat growing regions of the world. Scab resistance in wheat is complex and different “types” of resistance have been described. As is well known, Type II (resistance to spread within the head) has been studied the most, and the Type II resistance derived from Sumai 3 and its derivatives has been widely used in breeding for scab resistance. A number of genes on various chromosomes have been shown to be involved in controlling scab resistance in wheat. In particular, a region of the 3BS chromosome has been shown to have a significant effect on scab resistance. Information about quantitative trait loci (QTL) for types of resistance other than Type II would be useful in developing genotypes with a higher level of resistance than those currently available. Our objective is to begin to identify QTL for ability to produce unshriveled kernels when infected with scab.

Some breeding lines produce sound, unshriveled kernels when infected with scab. In these lines the percentage of shriveled kernels produced is lower than would be expected on the basis of the level of symptoms observed in the heads. This type of resistance has been described previously. We will refer to this as “kernel retention” throughout the proposal. We have observed that Coker 9474 and IL94-1653 exhibit this type of resistance. Using RIL (recombinant inbred line) populations involving Coker 9474 and IL94-1653, we plan to work on identification of SSRs associated with QTL for this type of scab resistance. We plan to select microsatellite markers (SSRs) dispersed across the genome, but markers reported to be associated with scab resistance QTL will be high priority. Markers will first be screened on the parents to ascertain which ones are polymorphic in these RIL populations.

Research on “kernel retention” scab resistance will provide information on the inheritance of “kernel retention” resistance, may lead to identification of SSRs associated with QTL for the kernel retention type of scab resistance, and may eventually lead to an efficient marker assisted selection system for this type of scab resistance. New scab resistance QTL from a source other than Sumai 3 might also be identified. This research may also lead to identification of lines with high levels of “kernel retention” resistance that could be useful as parents in breeding for scab resistance.