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Fusarium head blight (FHB) has recently become a major disease in hard winter wheat (HWW) in the northern plain area, a major wheat production region in the USA. Currently, HWW lacks of FHB resistance resources and needs to introgress effective resistance genes from other sources like spring wheat and wild relatives. Several major FHB-resistant QTLs have been discovered in spring wheat and they are great resources for improving FHB resistance in HWW. Of them, *Fhb1* has been introgressed into several HWW lines by marker-aided backcrossing. How effectively *Fhb1* works under the new HWW genetic backgrounds has been questioned since a case has emerged showing the introgressed *Fhb1* does not work in the HWW background. Our previous research in spring wheat has identified an *Fhb1* functional gene *WFhb1_c1*, and found that it exists in all wheat lines, and that it is not the coding sequence of *WFhb1_c1* (and thus *Fhb1*) but the regulatory component of the gene that may play the key role in FHB resistance. This proposed research aims at improving FHB resistance in HWW by molecular breeding/manipulation. It will do research to develop new breeding technology and germplasm to further enhance short-term and long-term improvement of FHB resistance and to efficiently introgress effective FHB resistance genes into HWW breeding germplasm. It will also develop a full understanding of specific biological factors influencing FHB infection/toxin accumulation. Therefore, it fits both the 1st and 2nd objectives of the HWW-CP for FY14-15. Our working hypothesis for this proposed research is that not every *Fhb1*-introgressing HWW line we have created gets the functional genic component of this QTL. We plan to test our hypothesis by achieving the following objectives: 1) understanding how *WFhb1_c1* is expressed in *Fhb1*-introgressing HWW lines; and 2) elucidating the regulatory component of the native *WFhb1_c1* in HWW that makes it FHB-susceptible. First, we will screen the current *Fhb1* introgressing HWW lines with gene-specific marker *Fhb1_c1* to see if an FHB-resistant copy of *WFhb1_c1* does exist in them. Expression pattern of *WFhb1_c1* during the early stage of FHB pathogenesis will then be investigated with qRT-PCR. *WFhb1_c1* regulatory sequences of interest will be amplified from HWW genomic DNA by PCR with *WFhb1_c1*-specific PCR primers, and be cloned and sequenced. Fulfilling the first objective will directly answer the question how *Fhb1* works in the *Fhb1*-introgressing HWW lines. This study may find that the introgressed *Fhb1* works well or not as well in HWW as it does in Sumai 3. In the later case, we need to understand why by elucidating the regulatory mechanism of *WFhb1_c1* in HWW. Even if *Fhb1* is found to work well in HWW, knowledge about how *WFhb1_c1* is regulated natively in HWW is important to enhancing FHB resistance in HWW by manipulating the regulatory mechanism for the benefits of HWW. This project should generate information about how *Fhb1_c1* functions in HWW and how we could manipulate it to improve FHB resistance in HWW in particular and in all small grains in general. This information is very useful to our understanding of FHB susceptibility, and should help us coming up a new strategy to control FHB epidemics in HWW. This project will develop allelic-specific markers for *WFhb1_c1* for marker-aid selection of HWW.