

**PI: Lewis, Janet****PI's E-mail: lewisja6@msu.edu****Project ID: FY08-SN-129****FY07 ARS Agreement #: 59-0790-6-061****Research Category: VDHR-NWW****Duration of Award: 1 Year****Project Title: Mapping QTL for Type I and II FHB Resistance from CIMMYT Germplasm derived from a Synthetic Hexaploid.****PROJECT 2 ABSTRACT**

(1 Page Limit)

Our objective is to identify unique genes for Type I and II FHB resistance from CIMMYT germplasm derived from a synthetic hexaploid. Multiple sources of resistance are needed to accumulate the complimentary genes needed for FHB resistance. The diploid wheat (D genome) *Triticum tauschii* (= *Aegilops squarrosa*) is potential source of resistance, as are Brazilian wheat. CIMMYT has created synthetic hexaploids derived from *Triticum tauschii* and several have been crossed to bread wheat. One such line, referred to here as "CASS94-A" is derived from the cross MAYOOR//TK SN1081/AE.SQUARROSA (222) (CASS94Y00009S-10PR-2M-0M). CASS94-A was crossed to OH685, an adapted but susceptible soft red winter wheat. A mapping population of winter wheat has been developed from this cross. Preliminary field data of this population shows a large amount of variation in FHB resistance between the different families within the population, and the data suggests that 3-4 genes are involved in resistance, or that large effect genes are present. In addition, some of the families showed better levels of resistance than the well-known resistant genotype 'Truman'. Previous greenhouse evaluation at Michigan State University of Type II resistance of a full-sib line of CASS94-A clearly showed a very prominent QTL for resistance on chromosome arm 2DL. However, field studies and Type I resistance levels were not evaluated on the sib population. In 2008 we will conduct field phenotyping of 176 F4:5 families of this population in two field locations, Ohio and Michigan. Phenotyping will focus on gathering data including both Type I and II levels of resistance. In the first year of the study, we will screen the parents from polymorphisms at 11 regions of the genome that have been associated with FHB resistance in past research (eg 1B, 2B, 2D, 3B, 4A, 4B, 5A, 5D, 6B, 7A, 7B). We will then genotype the F4 derived families with polymorphic markers from select regions. In year 2, if funded, we will also screen the parents with available SSR markers from the D genome. The F4 families will be genotyped with polymorphic DNA markers. In addition to SSR markers on the D genome, DArT analysis will be conducted to further enrich the D genome marker analysis.