COURNEYA., ISAAC*¹, NATHAN WYATT^{1,2}, GARY A. SECOR², VIVIANA V. RIVERA-VARAS², and MELVIN D. BOLTON^{1,2}, ¹USDA-ARS ETSARC, Fargo ND 1307 18th St N, ²North Dakota State University, Dept. of Plant Pathology, 355 Walster Hall, Fargo ND.

Effects of synonymous and nonsynonymous Cyp51 mutations on DMI resistance in Cercospora beticola.

Cercospora leaf spot (CLS), caused by the fungal pathogen *Cercospora beticola*, is the most economically important disease of sugarbeet worldwide. One of the primary means of combatting this disease is the timely application of fungicides. Demethylation inhibitors (DMIs) are a class of fungicide that target Cyp51, a key enzyme in the synthesis of an essential fungal cell membrane component called ergosterol. While DMIs are important for managing CLS, resistance to these fungicides has been observed. Previous work in our lab has shown that resistance to DMIs is highly correlated with synonymous and non-synonymous mutations in the Cyp51 gene. One such mutation, L144F, is found in two codon variants TTC and TTT, where TTC is associated with resistance and TTT is associated with sensitivity even though both codons encode phenylalanine. Notably, resistance is also strongly associated with the synonymous mutation E170. We have identified five Cyp51 haplotypes exhibiting different combinations of these mutations. To improve our understanding of the effects of silent mutations on DMI resistance, we have undertaken a variety of studies to characterize the role of this gene in mediating DMI resistance. The results and potential implications of these studies will be presented.