BOLTON, MELVIN D.[1]*, LUIGI FAINO[2], BART P. H. J. THOMMA[2] and GARY A. SECOR[3], [1]USDA-ARS, Northern Crop Science Laboratory, Fargo, North Dakota, USA, [2]Laboratory of Phytopathology, Wageningen University, The Netherlands and [3]North Dakota State University, Department of Plant Pathology, Fargo, ND, USA. **Insight into sterol demethylation inhibitor (DMI) resistance in *Cercospora beticola* using RNA-Seq.**

ABSTRACT

Cercospora leaf spot (CLS) is a devastating disease of sugarbeet caused by the fungus *Cercospora beticola*. Management measures include the application of sterol demethylation inhibitor (DMI) and quinone outside inhibitor fungicides. Understanding the molecular mechanism of fungicide resistance is critical for fungicide resistance management. We have shown previously that the gene encoding the DMI target enzyme, *CbCyp51*, is over-expressed in DMI-resistant isolates upon exposure to the DMI tetraconazole. However, no mutations in the *CbCyp51* gene or promoter were associated with resistance. Following the same experimental approach, we sequenced the entire transcriptome of a DMI-resistant and -sensitive isolate using next generation RNA-Seq technology to identify genes involved with DMI-resistance.

We identified 104 genes commonly differentially expressed between the two isolates in response to tetraconazole, suggesting a core set of genes are triggered in response to the fungicide and/or associated cellular stress. Interestingly, most of the genes in the ergosterol biosynthetic pathway were induced to similar levels in both isolates. One exception was *CbCyp51*, which was induced in both isolates but was expressed much higher in the DMI-resistant strain.

A set of 110 genes were uniquely induced in the DMI-resistant isolate after exposure to tetraconazole. Several genes encoding transmembrane molecular pumps, such as ATP-binding cassette transporters and major facilitator superfamily transporters, were identified. These genes will be ablated from the *C. beticola* genome to confirm their role in DMI-resistance. Future work directed towards pathway and sequence analysis of these 110 differentially expressed genes will be carried out to identify regions or mutations associated with DMI-resistance, which will be exploited for PCR-based detection analyses.