SAUNDERS, JOSEPH W.[1*], AND SRNIC, GORAN, [1]USDA, Agricultural Research Service, Dept. of Crop and Soil Sciences, Michigan State University, East Lansing MI 48824, and [2]Dept. of Crop and Soil Sciences, Michigan State University, East Lansing MI 48824. **Current understanding of the genetics and physiology of foliar disease lesion mimicry in sugarbeet.**

A foliar disease lesion mimic (DLM) trait visually characterized by brightly pigmented, or watersoaked, or necrotic, spots, lesions, or leaves, or all of the above, has been noticed in various smoothroot breeding lines at East Lansing. Because of its prospective value in elucidating sugarbeet foliar disease resistance mechanisms and genes, research on this trait was initiated nearly ten years ago. Early progress was slow, in part due to non-monogenic segregation patterns. The best inheritance model is that DLM is conditioned by a recessive allele at one locus in combination with a dominant allele at a second locus, to give more or less the classical 13:3 digenic F_2 segregation ratio, although modifier genes are probably involved. To reach this conclusion has required growing more than 5000 progeny plants in the greenhouse for durations of 4-8 months, due to a desire to give the trait time to be expressed in all possible DLM genotypes. This is largely due to higher temperatures favoring the expression of the trait, indicating a degree of inducibility which still needs to be quantified. AFLP research to map the DLM loci has been started. DLM plants have displayed an approximately two-fold higher level of pathogenesis-related (PR) leaf defense protein such as chitinases than related wild type segregates, suggesting that one or more of the DLM loci are acting relatively upstream in the defense reaction sequence. Tying DLM research into Cercospora genetic research is challenging, due to the non-monogenic nature of inheritance of each, and the minimal understanding of the inducibility of the DLM symptoms.