Determination of *Puccinia helianthi* races in the United States

Andrew Friskop\(^1\), Thomas Gulya\(^2\), James Jordahl\(^1\), Scott Meyer\(^1\), Ryan Humann\(^1\), Maricelis Acevedo\(^1\), Robert Harveson\(^3\), and Samuel Markell\(^1\)

\(^1\)North Dakota State University – Department of Plant Pathology, Walster Hall, Fargo, ND
\(^2\)USDA-ARS Sunflower Research Unit, 1307 18th Street N, Fargo, ND
\(^3\)University of Nebraska – Panhandle Research and Extension Center, 4502 Avenue I, Scottsbluff, NE

**INTRODUCTION**

Sunflower rust, caused by *Puccinia helianthi* (Schwein.), is an economically important disease in the major sunflower producing areas of the United States. Management of the disease is accomplished through the use of fungicides and resistant hybrids (if available). Pathogen race changes can occur over time leading to virulence on commercially available resistance genes. In 2008, the first documented sexual recombination event in the Northern Great Plains occurred. This event could have led to the development of new races and may impose a change on race prevalence across the region. Evaluation of pathogen diversity is very important for understanding the risk of sunflower rust epidemics in the future. Previous work on pathogen diversity of bulk field collections was conducted in 2007 and 2008 (Gulya and Markell, 2009). Although this information can be very useful (particularly for breeders), it is insufficient for accurate assessment of true pathogen diversity. Single-pustule (clone) creation is necessary for assessing pathogen diversity using any molecular tools and virulence phenotyping. The objectives of this study are to compare the virulence phenotypes and genotypes of single pustule isolates collected in 2011 and 2012.

**MATERIALS AND METHODS**

Samples were collected randomly by NSA survey teams or from sunflower consultants who submitted samples. Fields sampled during the survey were randomly selected in each county, but total number of fields was based on previous sunflower acreage. Field-derived or greenhouse-derived single pustule isolates were collected from multiple locations in 2011 and 2012. In 2011, 129 single pustule isolates were obtained from ND, NE, and SD. In 2012, 109 single pustule isolates were collected from CA, Canada, IA, MN, NE, ND, SD, and TX. Urediniospores were increased by inoculating a universal susceptible sunflower hybrid. Fresh urediniospores were inoculated on a set of nine differentials (Gulya and Masirevic, 1996) and evaluated 14 days later for reaction types and virulence phenotype. 

**RESULTS AND DISCUSSION**

In 2011, 129 isolates were processed and 17 races were detected. Two races (races 300 and 304) comprised ~70% of tested isolates (Figure 1). The most virulent race detected was 776 (virulent on 8 of the 9 differentials). Also, HAR3 conferred resistance to ~98% of the tested isolates. In 2012, 109 isolates were processed and 27 races were detected (Figure 2). Races 304 and 324 were the most predominant encompassing ~70% of the isolates. The most virulent race detected was race 777 appearing in multiple states. The differential line HAR3 conferred resistance to ~95% of the 2012 isolates.

Collectively, both sampling years yielded 29 sunflower rust races (Figure 3). Only two races were detected in 2011 that were not detected in 2012. The most frequently detected race was race 304. Although the differential lines CM29 and HAR2 conferred resistance to ~90% of the isolates in 2011, the same trend wasn’t noticed in 2012. This could be attributed to sampling bias, sampling area expansion, or race changes. Additionally, sunflower severity was much lower throughout the sampling region in 2012 and rust was very difficult to find in most fields. It is possible that races less likely to result in epidemics were detected in 2012 as a result of very intense searching for rust in fields. Additionally, multiple races were detected within location sites indicating multiple races can co-exist in a given field. Results indicate that a wide variety of virulence exists in the *P. helianthi* population, which suggests that breeding for resistance to all races is challenging. However, it is unclear if all races are capable of causing an epidemic.
Figure 1. Number of sunflower rust races detected and prevalence from 2011 single pustule isolates

2011 Sunflower Rust Race Survey Results

Figure 2. Number of sunflower rust races detected and prevalence from 2012 single pustule isolates

2012 Sunflower Rust Race Survey Results
Figure 3. Number of sunflower rust races detected and prevalence from 2011 and 2012 single pustule isolates

2011 and 2012 Sunflower Rust Race Survey Results

REFERENCES


