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IMPROVING KEY ROOT TRAITS IN SUGAR BEET: FUSARIUM TOLERANCE

Amélioration de caractéristiques centrales de la racine de la betterave sucrière: la résistance au fusarium / Verbesserung zentraler Merkmale der Zuckerrübenwurzel: Fusarien-Toleranz

ABSTRACT

Fusarium oxysporum is an important and widespread soil-borne pathogen of sugar beet causing both root rots and wilting symptoms, alone or together. In this study, we evaluated the resistance to *Fusarium oxysporum* of a collection of sugar beet lines from CRA-CIN Rovigo's genetic pool. Artificial inoculation was done with two fungal isolates from the US (Fob220a and F19). After six weeks, plants were scored for disease symptoms, including leaf stunting, chlorosis and necrosis, on the basis of a phenotypic resistance scale from 1 to 5. We identified lines, showing low and high levels of disease symptoms. These results were in agreement with field observations at two different study sites in the Ukraine where the same lines were classified as resistant and susceptible to root-rot under heavy infection pressure. Further genetic improvement studies at the molecular level based on the identification of SNP molecular markers associated with *Fusarium* resistance are underway.

INTRODUCTION

Fusarium oxysporum, is a ubiquitous soil-borne fungus (HARVESON & RUSH, 1997) that causes two devastating diseases in sugar beet, Fusarium yellows and Fusarium root rot. For both the typical symptoms of the disease are foliar wilting and inter-veinal yellowing, in Fusarium yellows there are no external symptoms on the root, but vascular discoloration is observed (STEWART 1931). A beet with Fusarium root rot shows a black or dark brown rot of the taproot that usually starts at the tip of the root (MARTYN *et al*., 1989). Significant reduction in root yield is shown in sugar beet affected by Fusarium yellows, with a significant loss of sucrose percentage and increased juice purity. Recent work suggests that the same *F. oxysporum* isolate can cause different disease symptoms on different sugar beet lines (HANSON *et al*., 2009). Since there are no efficient fungicides, the best means to control the disease is genetic resistance. For this reason it is critical to understand the genetic basis of the *F. oxysporum* – sugar beet host-pathogen interaction, and develop germplasm capable of control infection with each the three described sub-classes of *F. oxysporum* that cause disease on sugar beet (HILL *et al*. 2010).

MATERIALS AND METHODS

In this work, 19 experimental sugar beet hybrids from CRA-CIN Rovigo's genetic pool and a commercial hybrid control were tested with two *Fusarium oxysporum* isolates from two different genetic sub-groups, F19 (sub-group A) and Fob220a (Sub-group B) (HILL *et al*. 2010). The evaluations were carried out under greenhouse conditions in a peat-based potting mix. Twelve 5-week-old sugar beet plants were soaked in a 4×10^4 spores/ ml suspension for 8 min and then planted in the potting medium. Sterile water was used as negative control. Plants were watered daily. Weekly for 6 weeks, plants were rated using a 0 to 5 scale in which 0 was healthy and 5 was completely dead (tab. 1). AUDPC (Area Under the Disease Progress Curve) was calculated to represent the response of 20 germplasms to two Fusarium isolates. At the final rating, the taproots were removed from the soil and root symptoms were examined. Throughout the rating period roots of any plants that died were plated on potato dextrose agar to confirm the presence of *Fusarium*. Samples also were plated at the end of the experiment to confirm Fusarium in symptomatic but living plants.

RESULTS AND DISCUSSION

In this experiment, two *F. oxysporum* isolates showed different disease levels, with isolate F19 more aggressive than Fob220a, consistent with previous reports (HANSON *et al*., 2009). Disease symptoms increased linearly after the second week post-inoculation.

Among the sugar beet lines there was a significant difference in the tolerance response (fig. 2). Some experimental hybrids were tolerant for both tested sub-groups.

Even though the Fob220a was less aggressive than F19, both F19 and Fob220a caused rot root in this germplasm (fig. 3) unlike previously reports (HANSON *et al*. 2009). Thus, for the first time it showed an unusual root rot. The same strains used for identification were isolated from the rotted tissue.

The next step will be to better understand the variability among the lines and the interaction between pathogen and germoplasm and to make further studies of genetic improvement to develop SNP molecular markers associated with the *Fusarium* tolerance.

Fusarium disease rating system

0 = No visible disease, plants healthy with all green leaves.
1 = May show stunting. Leaves may wilt during the day, but recover turgor overnight or with watering, small chlorotic areas on lower leaves between veins, but most of leaf green.
2 = Chlorosis - leaves either become mottled with interveinal yellow spots or the outer edges of the leaf becomes yellow; up to extreme yellowing of leaves, but no necrosis.
3 = Leaves with necrotic spots or becoming necrotic and dying, but less than half of the leaves showing necrosis. Stunting may be severe.
4 = Half or more of the leaves dead, plant severely stunted, most living leaves showing some chlorosis or necrosis.
5 = Plant dead.

Fig. 1: Fusarium disease rating system used.

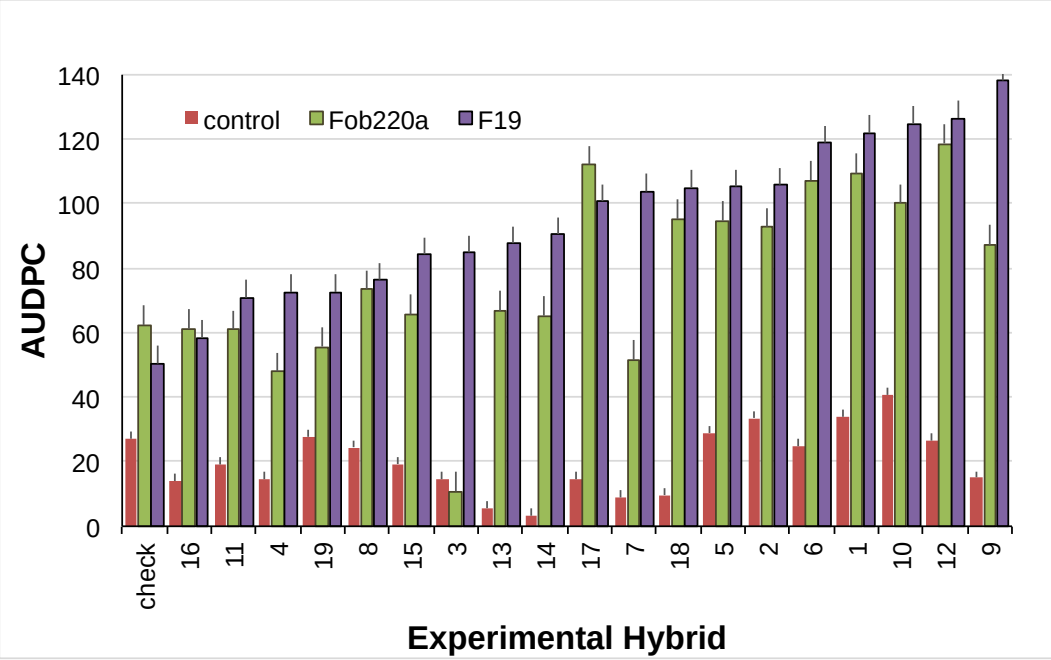

Fig. 2: Response of 20 germplasms to two Fusarium isolates (AUDPC = Area Under the Disease Progress Curve).

Fig. 3: Examples of complete root rot caused by *Fusarium oxysporum*, an unusual result.

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