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RESISTANCE TO LEAF RUST (*PUCCINIA RECONDITA* F.SP.*TRITICI* AT THE SEEDLING STAGE AMONG SINGLE D-GENOME SUBSTITUTION LINES OF TRITICALE PRESTO

(Short communication)

ABSTRACT

There were performed tests on a set of twenty single substitutions of the D-genome chromosomes from various bread wheats into winter triticale Presto, a set of seven disomic addition lines of chromosomes from winter rye Dankowskie Zlote to winter wheat Grana, and all parental forms to study on the chromosome location of major genes that are resposible for seedling stage resistance to *Puccinia recondita* f.sp.*tritici* in triticale, . For each line, seedlings developed to two-leaf stage were exposured to four pathotypes of the pathogen predominant in Poland to test them in the greenhouse conditions. There were high resistance among the substitution lines of Presto, 1D(1A), 4D(4R), and 5D(5R) suggesting that chromosomes introduced from the D-genome carried resistance genes. Among the addition lines, GH2R was highly resistant. Resistance to rust at seedlings stage of Presto lines of D- genome and addition lines Grana (wheat)/Dankowskie Zlote (rye) shows that chromosome 2R is responsible for the resistance to the disease.

Key words: leaf rust, resistance, substitution, triticale, wheat

INTRODUCTION

The most important of the three rusts occurring on wheat in Poland, is the leaf rust, caused by *Puccinia recondita* Rob. ex Desm. f. sp. *tritici* appearing every year that often demonstrates in severe epidemics (Dwuraźna, Gajda 1980, Strzembicka 1997). Recently this pathogen has also become a serious menace for triticale (Zamorski *et al.*1994, Woś *et al.* 1994, 1995, Arseniuk 1996, Strzembicka *et al.* 1998). Severe infections caused by *P. recondita f.sp.tritici* resulted substantial yield losses of winter triticale (Woś *et al.* 1994).

Leaf rust resistance of triticale, similary to that one found in wheat, may be conditioned by numerous genes scattered through the genome what make breeding efforts focused on concentration of profitable loci necessary. Some cultivars, particularly the newer ones, show various degree of susceptibility (Kociuba 1994,

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Woś *et al.*1995, Węgrzyn *et al.* 1996, Strzembicka *et al.*1998). It has been established that responsible for the spread of *Puccinia recondita* f.sp.*tritici* are: the physiological specialization of the pathogen, its high virulence and insufficiency of sources of resistance (Roelfs *et al.*1992, Strzembicka 1997). Taking under consideration that the identification of new sources of resistance would be important in developing of breeding programs, it seems to be reasonable to search such sources. This studies were taken up to identify the chromosomes that possibly carry leaf rust resistance among the substitution and addition lines of winter triticale and winter wheat. The results of the resistance evaluation at the seedling stage were presented in this paper.

MATERIALS AND METHODS

Twenty single D-genome substitution lines of winter triticale Presto (substitution 7D(7R) missing), seven disomic addition lines of chromosomes of winter rye Dankowskie Zlote to winter wheat Grana (GHR1-GHR7) and the parental forms were tested with respect to resistance (Table 1).The winter triticale substitution lines and wheat-rye addition lines were obtained from A.J. Lukaszewski, University of California, Riverside, USA.

Experiments were carried out under greenhouse conditions within the years 2000 - 2002. Seedlings developed to two-leaf stage were inoculated with the same four pathotypes of *Puccinia recondita* f.sp.*tritici*in every year, i.e. 4c, 40b, 83c, and 95b. These four pathotypes were found as predominant ones in the Polish population of the wheat leaf rust (Strzembicka 1997) and were highly virulent to the different set of fifteen isogenic lines carrying the following resistance genes *Lr1,Lr2a, Lr2b, Lr2c, Lr3a, Lr9, Lr11, Lr15, Lr17, Lr19, Lr21, Lr23, Lr24, Lr26* and *Lrr28*, respectively. This set of differentials is used in all of Europe (Mesterhazy *et al.*2000).

The virulence characteristics of the pathotypes of *Puccinia recondita* f.sp.*tritici*used in this study:

PathotypeVirulent on the *Lr* genes

4c2c, 3, 11, 15, 17, 21, 26, 28
40b 3, 11, 15, 17, 21, 26
83c 3, 11, 15, 17, 21, 26, 28
95b 2b, 2c, 3, 11, 15, 17, 21, 26, 28

The inoculation of seedlings was performed by spraying the leaves with a water suspension of spores supplemented Tween 20 used as a wetting agent. Following inoculation, the seedling were placed in a moisture chamber for 24 h and then transferred to the room with 18-20°C and 10-12 h day/night period. The virulence of pathogen against leaves was evaluated according to the scale suggested by Roelfs *et al.*(1992), where: 0-immune (no visible uredia), 0; - very resistant (hypersensitive flecks), 1- resistant (small uredia with necrosis), 2 – resistant to moderately resistant (small to medium uredia often surrounded by chlorosis or necrosis), 3 – moderately susceptible (medium sized uredia with or without chlorosis), 4 –susceptible (large uredia without chlorosis), x – resistant, heterogeneous.

Table 1

Resistance at the seedling stage to *Puccinia recondita* f.sp.*tritici* of some substitution lines of triticale Presto, euploids and addition lines of Grana (wheat)/Dankowskie Zlote (rye)

Substitution,	Pathotypes of *Puccinia recondita* f.sp.*tritici*							
Addition, translocation	4c	40b	83c	95b	4c	40b	83c	95b
Presto 1D(1A)	0;	0;	0;	0;	0;	0;	0;	0;1
Presto 1D(1B)	2	2	2	1,2	0;	0;	1,2	1
Presto 1D(1R)	1,2	x	x	0;	0;	0;	0;	0;
Presto 2D(2A)	0;2	2,x	2	2	0;	1	0;	0;1
Presto 2D(2B)	1,2	2	2	0;2	0;	1	1	0;
Presto 2D(2R)	2	4	4	2	2	3	3	2
Presto 3D(3A)	2	x	0;	2	0;1	0;	0;	1
Presto 3D(3B)	0;	0;	2,x	0;	1	0;	0;	0;
Presto 3D(3R)	1,2	2,x	1,2	x	0;1	2,x	2	2;x
Presto 4D(4A)	2	2	x	0;	0;1	0;1	0;	0;
Presto 4D(4B)	0;	2,x	2	0;	0;	0;	0;1	0;1
Presto 4D(4R)	**0;**	**0;**	**0;1**	**0;1**	**0;**	**0;**	**1**	**1**
Presto 5D(5A)	0;1	1,2	0;1	1	0;	0;	0;	0;
Presto 5D(5B)	1	2	2	1	0;	0;2	0;1	0;1
Presto 5D(5R)	**0;**	**0;1**	**0;**	**1**	**0;**	**0;**	**0;**	**0;1**
Presto 6D(6A)	1	2	x	0;	0;	0;	0;1	0;
Presto 6D(6B)	2	1,2	1,2	0;	0;	1	0;	0;1
Presto 6D(6R)	1,2	1,2	1,2	2	0;	0;	0;	1,2
Presto 7D(7A)	0;1	x	2	1,2	0;	0;	0;	0;1
Presto 7D'N(7B)	1,2	0;2	2	1	0;	0;	1	0;
GH 1R	4	4	4	4	4	4	4	4
GH 2R	**0;**	**0;1**	**0;**	**1**	**0;**	**1**	**0;**	**1**
GH 3R	4	4	4	4	4	4	4	4
GH 4R	4	4	4	4	4	4	4	4
GH 5R	4	4	4	4	4	4	4	4
GH 6R	4	4	4	4	4	4	4	4
GH 7R	4	4	4	4	4	4	4	4
Grana (wheat)	4	4	4	4	4		4	4
Dańk.Złote (rye)	0	0	0	0	0	0	0	0
Presto (triticale)	**0;2**	**0;1**	**1**	**2**	**0;1**	**0;**	**0;**	**0;**
(Valdy- 1) Presto 1R.1D$_{5+10}$ -2/ WR4	-	-	-	-	0;	2	0;x	0;
Valdy - 2 Presto 1R.1D$_{5+10}$ -2/ WR4	-	-	-	-	0;	0;	0;	0;
Check- Kolibri	4	4	4	4	4	4	4	4

Leaf rust reaction scale (Roelfs *et al.* 1992): 0 -immune, 0;-very resistant, 4 - susceptible, x - heterogeneous reaction

RESULTS AND DISCUSSION

In all experiments, a resistant or moderately resistant reactions were typical for all substitution lines of tested triticale; Presto itself had a good level of resistance. Depending on the pathotypes used, the substitution lines showed various host responses, with infections of types of 0, 1, 2, X. Among the D-genome substitution lines of Presto, lines 1D(1A), 4D(4R), 5D(5R) showed higher resistance to *Puccinia recondita* f.sp. *tritici* than the euploid Presto and it was found for both years of testing. It indicates that chromosomes 1D, 4D and 5D present in the set can be identified as ones carrying the resistance genes. Less likely, chromosomes 1A, 4R and 5R can be thought as carrying genes of suppressors of resistance. It may be pointed out that in a different study (Woś *et al.*, this volume) substitution 1D(1A) had the most positive effect on breadmaking quality of all substitutions tested. This study shows that additionally, it improves disease resistance.

Among the disomic wheat-rye addition lines, only GH2R showed high seedling resistance to all four used pathotypes (Table 1). Clearly, this chromosome carries a major resistance locus although its presence was not apparent in the set of Presto substitutions. This locus could be exploited in wheat breeding as there is available a set of 120 2B-2R recombinants and it could be easily used to engineer the proper transfer (A.J. Lukaszewski, pers. comm). While this study gave no indication with regard to the identity of the resistance gene (it may be also *Lr25*), known to be on the segment of 2RL in the Transec translocation (Driscoll and Anderson 1967).

The pathogen isolated from rust-infected triticale fields is usually identified as *Puccinia recondita* f.sp.*tritici* (McIntosh *et al.* 1995, Arseniuk 1996).This implies that like wheat, triticale is generally resistant to f.sp *secalis.* Because f.sp.*secalis* is avirulent on wheat*,* Quinones *et al.*(1972) surmised that resistance in triticale was controlled only by genes deriving from the wheat parent component. If it appeared true in at least some instances, rye resistance genes clearly would operate in other cases.

Singh and McIntosh (1990) identified gene *LrSatu,* located in a rye chromosome, that occurred at high frequency in CIMMYT triticale population. The rye-derived genes *Lr 25* and *Lr26* that showed to be effective in hexaploid wheat backgrounds, in some countries they ought to be effective in triticale (McIntosh *et al.*1995).

Resistance to diseases in triticale has been considered as one of its most important and durable advantages (Arseniuk *et al.*2000).

Resistance to rust at seedlings stage of Presto lines of D- genome and addition lines Grana (wheat)/Dankowskie Zlote (rye) shows that chromosome 2R is responsible for the resistance to the disease.

The results of seedling tests on leaf rust resistance suggest that some of analyzed substitution lines of triticale and addition line GH2R may be the valuable source of resistance. They will be evaluated in the field conditions, in adult plant stage and used for further investigations.

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