Virulence of winter wheat leaf rust isolates

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The pathogenicity of *Puccinia recondita* f. sp. *tritici* was evaluated in 2000 and 2001. The pathogenicity of isolates was determined in tests of detached primary leaf segments kept on water agar suplemented with benzimidazole (30 p.p.m.). The differential genotypes used were 'Thatcher', 31 near-isogenic 'Thatcher' lines each with a single leaf rust resistance gene, and six cultivars/lines with additional resistance genes. All isolates were avirulent for the genes *Lr9, Lr19, Lr24*. All three completely resistant genes are derived from wild relatives. Both virulence and avirulence were detected for the rest 34 genes. The most severely affected genes were *Lr3ka, 17, 18* (90–100%). The effectiveness of *Lr* genes strongly depended on their frequency in cultivars: the more frequent they were in cultivars, the less effective they were. That is due to pathogen adaptation. Most frequent in cultivars are *Lr* genes *Lr3, 10, 13, 26, 34*. Only gene *Lr26* showed a satisfactory resistance. Isolates containing 17–22 virulent genes predominated. The frequency of the other isolates was low, as these isolates had a lower vitality or fewer varieties suitable for them.

Key words: winter wheat, leaf rust, resistance genes

INTRODUCTION

Wheat leaf rust (caused by *Puccinia recondita* f. sp. *tritici*) is one of the most important diseases of wheat in Lithuania and worldwide. It occurs annually throughout the growing areas and in some epidemic years could cause serious yield losses. Susceptible wheat varieties suffer regularly a yield reduction of 5–15% or more, depending on the stage of crop development when the initial rust infection occurs. Although it does not cause severe epidemics in Europe every year, the disease reaches epidemic levels in several areas annualy and causes severe losses [1].

Except for the traditional use of fungicides, the protection against it is mainly based on the application of resistance. This approach is highly preferable because of its economic importance and special biological safety. Up to now more than 45 leaf rust resistance genes have been characterised [2]. Most of these genes are expressed throughout the whole life of the plant (seedling resistance), however, some are expressed at adult plant growth stages only (adult plant resistance, APR). APR to leaf rust in wheat can be under oligogenic or polygenic control. Examples are known in which APR is governed by single genes which confer hypersensitivity (*e.g.*, *Lr12, Lr13, Lr22b*), and many of these have been overcome by the development of corresponding virulence in the pathogen [3].

Partial resistance is a form of moderate resistance in which a hypersensitivity reaction does not occur, and this resistance does not allow for the development of virulent genotypes in pathogen population. Promising ways to achieve durable resistance include pyramiding race-specific resistance genes, combining seedling resistance and APR genes, and combining effective race-specific genes with high levels of partial resistance [2, 3].

Unfortunately, the resistance genes used in breeding lose their effectiveness after commercial growing for some years, due to pathogen adaptation and change in virulence. This is the main reason why regular virulence surveys are needed to develop successful resistance breeding strategies [4].

Besides, isolates with known virulence are suitable for evaluation of newly developed breeding lines and varieties for resistance to leaf rust. If a cultivar has not yet been grown in commercial plots, but is susceptible to all or a major part of isolates, it is possible to reject such variety in early breeding stages or there is a need to create a special cultivation technology, if the variety has other special valuable traits [1, 4].

MATERIALS AND METHODS

Samples of leaf rust uredinospores were collected in 2000 and 2001 from different cultivars and breeding

lines in breeding nurseries. The spores were vacuum-dried and stored until analysis in glass ampoules at 4 ± 2 °C. Suspension of water and spores was sprayed onto seedling leaves of susceptible the wheat variety 'Carsten V'. After a 24-h dark and moist period, inoculated seedlings were placed in a temperature-controlled chamber (20 ± 2 °C). Single pustules were isolated with a leaf segment after appearance of virulence traces in Petri dishes on water agar suplemented with benzimidazole (30 p.p.m.) at a temperature of 20 ± 2 °C. Isolates from single pustules were multiplied on the same cultivar under the same conditions.

The pathogenicity of the isolates was determined in tests of detached primary leaf segments maintained on the same water agar under the same conditions. Seeds of differential set were obtained from Dr. Szunics Laszlo, Agricultural Research Institute of the Hungarian Academy of Sciences, Martonvasar. The differential genotypes used were 'Thatcher', 31 near-isogenic 'Thatcher' lines each with a single leaf rust resistance gene, and six cultivars/lines with additional resistance genes.

The scoring was carried out eight to ten days after inoculation. Infection types of 0 to 2 were classified as resistant and those of 3 to 4 as susceptible.

RESULTS

A total of 100 single uredinospores isolates were analysed (50 in 2000 and 50 in 2001).

The frequency of virulence of leaf rust isolates from seedlings of the 'Thatcher' isolines and additional cultivars/lines is presented in Table. *Lr* genes *9*, *19* and *24* were resistant to all isolates, whereas *Lr* genes *3ka, 17, 18* were susceptible to almost all isolates (90–100%). The effectiveness of the rest resistance genes was different.

The low virulence frequency in the leaf rust population was found for genes *Lr 15, 21, 23, 25, 26, 32, 35, 38, 44* (10–40%). Only genes *Lr15* and *Lr23* are from *T. aestivum*. The diploid *Aegilops* species are excellent sources of resistance to leaf rust. Although suppression of alien resistance gene(s) by gene(s) present on the genomes of cultivated species is a barrier in alien gene transfer, this obstacle can be avoided by selecting wheat stocks or cultivars lacking the suppressing genes [2].

From 41 to 70% of isolates exhibited virulence for genes *Lr 2a, 10, 11, 12, 14a, 20, 22a, 27, 29*. Of them, *Lr 14a, 22a, 29* were from wild relatives. The development of new virulent biotypes as a response to the introduction of resistant cultivars is especially asso-

Table. **Percentage of *Puccinia triticina* isolates virulent at the seedling stage on single-gene differential lines in 2000 and 2001**

No.	Line/Cultivar	Resistance genes	Source of resistance genes	Approximate frequency in cultivars and lines, %	Frequency of virulence, %	
					2000	2001
1	*2*	*3*	*4*	*5*	*6*	*7*
1	Tc* 6 / Centenario	*Lr 1*	*T. aestivum*	5	74	66
2	Tc* 6 / Webster	*Lr 2a*	" –"	3	58	60
3	Tc* 6 / Carina	*Lr 2b*	" –"	0.5	74	88
4	Tc* 6 / Loros	*Lr 2c*	" –"	1	60	80
5	Tc* 6 / Demokrat	*Lr 3*	" –"	10	72	82
6	Tc +Lr 3bg	*Lr 3bg*	" –"	1	76	70
7	Tc + Lr 3ka	*Lr 3ka*	" –"	0.5	90	94
8	**Tc + Lr9**	***Lr 9***	***Ae. umbellulata***	**2**	**0**	**0**
9	Tc * 6 / Exchange	*Lr10*	*T. aestivum*	10	48	68
10	Tc * 6 / Hussar	*Lr11*	" –"	3	64	60
11	Exchange/Tc*6	*Lr 12*	" –"	1	48	40
12	Lr 13	*Lr 13*	" –"	10	46	72
13	Spica	*Lr 14a*	*T. timopheevii*	5	44	42
14	Tc + Lr 14b	*Lr 14b*	"–"	0.5	72	64
15	Tc+Lr14ab	*Lr 14ab*	"–"	0.5	76	52
16	Tc* 6/Kenya.W1483	*Lr 15*	*T. aestivum*	0.5	40	36
17	Tc(6)Exchange	*Lr 16*	" –"	3	68	78
18	Tc + Lr17	*Lr 17*	" –"	2	92	94
19	Tc* 7/Africa 43	*Lr 18*	*T. timopheevii*	1	100	100
20	**Tc* 7/Agropyron**	***Lr 19***	***Ag. elongatum***	**0.5**	**0**	**0**
21	Tc* 6/Jimmer	*Lr 20*	*T. aestivum*	2	52	70
22	Tc* 6/RL 5406	*Lr 21*	*Ae. squarrosa*	0.1	26	30
23	Tc* 6/RL 5404	*Lr 22a*	*Ae. squarrosa*	0.1	52	68

Table continued.

1	*2*	*3*	*4*	*5*	*6*	*7*
24	Tc* 6/Lee.FL310	*Lr 23*	*T. aestivum*	2	22	32
25	**3AG II.1**	***Lr 24***	***Ag. elongatum***	**5**	**0**	**0**
26	Transec	*Lr 25*	*S. cereale*	0.4	22	14
27	Tc* 6/ST-1-25	*Lr 26*	"–"	15	40	36
28	Gatcher	*Lr 27*	*T. aestivum*	2	70	60
29	Tc* 6/CS7D-AG//11	*Lr 29*	*Ae. elongatum*	0.1	68	38
30	Tc +Lr30	*Lr 30*	*T. aestivum*	0.1	64	82
31	Tc * 6/3Ae. sp	*Lr 32*	*T. tauschii*	0.1	36	28
32	Tc*6/PI58548(1+ Gene)	*Lr 33*	*T. aestivum*	1	74	72
33	Tc*6/PI58548(2+ Gene)	*Lr 34*	"–"	10	66	80
34	Tc*6/Rl5711	*Lr 35*	*Ae. speltoides*	0.1	40	32
35	FE51 - 5/86	*Lr 37*	*T. ventricosa*	1	80	64
36	Tc*6/TMR-514-12-24	*Lr 38*	*Ae. intermedium*	0.1	36	24
37	Tc*6/8404	*Lr 44*	*T. spelta*	1	28	10

T. – Triticum, Ae. – Aegilops, Ag. – agropyron, S. – Secale.

ciated with breeding for major gene resistance, and these major genes are selected for wheat breeding for high levels of resistance [5].

The other group of genes with very low effectiveness (infected with isolates 71–90%) was *Lr 1, 2b, 2c, 3, 3bg, 13, 14, 14ab, 16, 30, 33, 34, 37*. Resistance genes *Lr 14, 14ab*, *37* are from wild relatives. The most common resistance genes in cultivars are *Lr3, 10, 13, 26, 34*. These five genes are frequent in more than 50% of the widely grown cultivars. All of them showed low resistance to leaf rust isolates, except *Lr 26* which is from *S. cereale*. All more or less common *Lr* genes are from *T. aestivum*. That is the reason why the effectiveness of all these resistance genes is low. On the other hand, genes that spread poorly in cultivars are from wild relatives or they are overcome completely by leaf rust due to a long time of its adaptation.

Besides, the effectiveness of APR genes *Lr 12, 13, 22a, 34, 35, 37* is not very high at seedling stage due to expression of resistance primarily or only in adult plant stage.

E.g., gene *Lr13* is none the less important since it has, in combination with other genes, continued to provide protection against most pathotypes.

Species related to wheat, including both distantly related and progenitor species, represent a large reservoir of useful variability including rust resistance that can be exploited in wheat improvement. A large number of transfers carrying useful alien genes have been produced, but very few have been exploited commercially. Most of the introduced alien segment from wild relatives into wheat either do not compensate well for the loss of wheat chromatin or contain undesirable genes, causing depression in yield and performance of the plant. Undesirable effects can be avoided by transferring the desired gene(s) with least amount of unwanted alien chromatin [1, 6].

Extensive hybridisation has contributed significantly to germplasm enhancement of bread wheat. Many agronimically important traits, including resistance to diseases and pests and abiotic stresses, have been transferred from related species and genera into wheat. Alien resistance genes are useful only when they are expressed in the cultivated background [7].

The isolates were distributed to 12 groups according to the number of virulent genes – from 15 to 26 genes per group (Fig.). The average of virulent genes per isolate was 19.98 in 2000 and 19.96 in 2001. Groups of isolates containing 19–24 virulent genes predominated. The frequency of the other groups was lower. The fewest genotypes were with very low or very high virulence, as these genotypes had a lower vitality or fewer varieties suitable for them. Changes that occurred in the pathogen virulence over two years were not essential. The pathogen population has un-

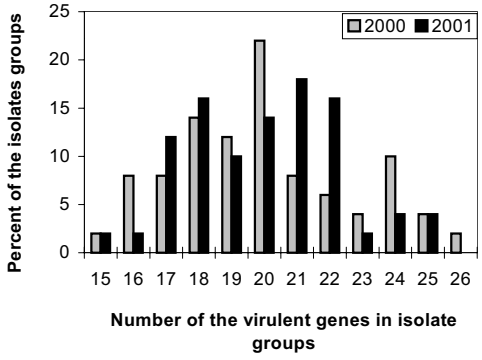

Figure. Distribution of the virulent genes in groups of isolates

derhone important microevolutional changes over a longer period (5–10 years) [2, 7, 8].

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ŽIEMINIŲ KVIEČIŲ RUDŲJŲ RŪDŽIŲ IZOLIATŲ VIRULENTIŠKUMAS

S a n t r a u k a

Žieminių kviečių rudųjų rudžių patogeniškumas buvo tirtas 2000 ir 2001 metais. Izoliatų patogeniškumui nustatyti naudotas 37 diferenciatorių rinkinys su žinomais rudosioms rudims atspariais genais. Veislių diferenciatorių daigų pirmų lapų segmentai laikyti vandens agare su 0,003% benzimidazolo. Visi tirti izoliatai buvo avirulentiški diferenciatoriams – linijoms su genais *Lr9, 19, 24.* Šie trys visišką atsparumą suteikiantys genai yra perkelti iš laukinių giminaičių. Kiti 34 genai daugiau ar mažiau pažeisti. Labiausiai buvo pažeisti *Lr3, 10, 13, 26, 34* atsparumo genai. *Lr* genų efektyvumas labai priklausė nuo jų dažnumo kviečių veislėse: kuo genai buvo dažnesni, tuo jų efektyvumas buvo mažesnis. Šio reiškinio priežastis yra patogeno adaptacija. Dažniausi veislėse yra *Lr3, 10, 13, 26, 34* genai. Tiktai *Lr26* genas užtikrina patenkinamą atsparumą. Dažniausi izoliatai su 17–22 virulentiškumo genais. Kitų izoliatų dažnis nedidelis, kadangi jie buvo mažiau gyvybingi ar turėjo mažiau jiems tinkamų veislių.