www.nsseme.com/journal.html

original scientific paper / originalni naučni članak

Ratar. Povrt. 50:3 (2013) 6-12

Functions of Lr Genes beside Reduction of *Puccinia triticina*

Zoran Jerković • Željana Prijić • Veselinka Đurić • Mirjana Lalošević

received: 16 September 2013, accepted: 29 October 2013
published online: 10 February 2014

doi:10.5937/ratpov50-4482

Summary: Wheat leaf rust race specific resistance controlling genes (Lr) caused 0.01-0.03 decrease of SAGR = divided first two internodes with stem length. In addition, nitrogen transport in upper parts by those genes was 3-19% higher in comparison to near isogenic lines with nonspecific Lr 22a and Lr 22b genes. RAR (divided root nitrogen content with sum of up ground and root ones) values of Lr16, Lr29 and Lr37 between Lr1, Lr15 or Lr19 and nonspecific genes were explained by hydrolytic stability of endo-proteases responsible for cleaving of glutens disulphide bridges. According to RAR, Lr 34 seemed to be specific Lr gene but increased SAGR as was by nonspecific Lr genes focused SUTs as adequate for its conformity. Two different drought and heat stress respond systems were linked to Lr genes; one based on gluten degradation consequential through photosynthesis decrease and another on accelerated transfer of starch products and water in stem.
Key words: gluten, LR genes, *Puccinia triticina*, SAGR, wheat enzymes, wheat leaf rust

Introduction

Leaf rust causer *Puccinia triticina* is not the most destructive of wheat parasites. Stable appearance and large wheat growing areas have explained the responsibility for the major accumulated yield losses from single parasite in history (Chester 1946, Rijsdijk & Zadoks 1976). Considered by Khan et al. (1997), total yield could be reduced for about 1% for each 1% of infection, when higher temperatures begin earlier in the spring. In semiarid regions, grain yield was decreased by approximately 3.5% for each 10% of two last leaves average infection (Jerkovic 2008). Until nowadays, 63 genes were proposed as *Puccinia triticina* reducing ones (Kolmer et al. 2010) and segregated as horizontal or vertical influential on parasites population reduction (Van der Plank 1963), to parasite races specific or nonspecific (Nelson 1978) and adult or seedling stage effective (Dyck 1991, Martinez et al. 2001, Saini et al. 2002). Highest reaction type (RT) was basis for partial resistance definition (Parlevliet & Van Ommeren 1975). Suspicion about huge number of different specific parasite host interactions predicted by gene-to-gene theory (Flour 1955, 1971) was articulated by Parlevliet (1988). Parlevliet (1986) recognized pleiotropic association between latency period (LP) and infection efficiency as well as LP was linked to specific resistance as its last character before overcome by changes in parasitic population (Jerkovic et al. 1993, Jerkovic & Putnik-Delic 2004). Background influence on Lr genes expression was reduced by backcrossing with variety Thatcher (Lr NILs). Negative effects of genes for resistance on wheat genotype fitness (Van der Plank 1976) were described as lower yield of eight lines with genes introduced from relatives (The et al. 1988). Their activity was related to fungal elicitor (De Witt et al. 1993) while previously single glycoprotein in cytoplasm of susceptible variety, same as in the parasite cell wall as cause of lignifications was discovered (Koegel et al. 1988). The nature of nonspecific resistance characterized by decrease of infection efficiency was attempted to be explained across stomata number and function, leaf wax differences (Anker

Z. Jerković* • V. Đurić • M. Lalošević
Institute of Field and Vegetable Crops, 30 Maksima Gorkog, 21000 Novi Sad, Serbia
e-mail: zoran.jerkovic@nsseme.com

Ž. Prijić
Megatrend University, Faculty of Biofarming, 39 Maršala Tita, 24300 Bačka Topola, Serbia

& Nis 2000) or by analyses of early abortion (Niks 1981, Jacobs 1990, Niks & Dekens 1991, Jacobs & Kiriswa 1993). The inheritance of the trait was simple to polygenic (Parlevliet 1978, Lee & Shaner 1985, Dyck 1991, Das et al. 1992, Kolmer & Liu 2001). Across enhancements of the resistance, complementary Lr genes were recognized (Samborski & Dyck 1982, Jerkovic 1992, German & Kolmer 1992). The environmental influence on resistance genes expressions was involved in concept of interorganismal genetics (Loegering 1978) as well as of Lr ones summarised by Browder (1985).

The differences in growth were found to be related with leaf rust severity and regional grain yield potential and linked with nonspecific or adult stage resistance (Jerkovic & Prijic 2009). From another side, it was discovered that Lr 16 and Lr 29, which were confirmed as race specific genes in interactions with parasite, had influence on carbohydrate fungicides accelerated degradation (Jerkovic et al. 2012). It was known that the fungi cell wall was structured mostly from glycoproteins (Harder & Chong 1984) laminated by bridges same in one of basic elements (sulfur) within seed stored proteins (Shewry & Halford, 2002) or mentioned fungicides. Protein transport from seed began in units of molecular weight under 64 Kd continued in 24 Kd and finished below 10 Kd (Masson et al. 1986), so there was a room for their accelerated degradation. The presence of gluten in leaves was also noticed (Cumming et al. 2004), but their enzymatic degradation was not described.

According to previous states, the hypothesis was that specific Lr genes should cleave the disulphide bridges of seed stored proteins during germination visible through their distribution in organs of wheat seedlings, as well as that it will be of influence on growth.

Materials and Methods

Eighteen Lr near isogenic lines were sown in field nursery (susceptible border Novosadska rana 2) in semiarid region (around 600 mm^2 of the annual rainfall). Presented parasite severity on last two leaves was estimated in percents from the second ten-day period of May. Results from 2004 and 2011 were presented because of no or low antagonistic facultative parasites appearance. Two last internode and stem lengths estimated at the third last ten-day period of June 2010 and 2011 were divided and represented as stem growth ratio (SAGR). The second leaf length of seedlings was compared with the first one (LGR) ten days after emerging of 60 seeds sown in 2 dl pots with compost and grown in greenhouse on constant air temperature of 20°C and 11 hours of day light regime, daily watered. Genotypes with near equal lengths of leaves were pronounced medium (M), those with higher second one for more than 1 cm were H_2 as opposite were L. *Puccinia triticina* severity on the last two leaves was presented in percents of the affected area. Lines for the tests divergent by nonspecific and specific resistance control were chosen across the specific resistance character, prolonged latency period in field and low reaction type (RT) according to scale by Stakman et al. (1962) to the races 2 and 77 (Johnston & Browder, 1966) at seedling stage. Seed of Lr lines from same locality in 2010 were sown in 2 dl pots or 3 l cylinder filled by compost or pure sand and grown at the same regime as for LGR analysis. Two or three week old seedlings after emerging were cleared in water, completely dried and fine milled. Nitrogen content in root and up ground part was estimated in two replications by method previous used for seed protein evaluation (ICC method 105-2, 1994). Root (RC) or app ground (AC) values multiplied by 5.7 were adequate to protein based structures amount presented in percent (RPC or APC). RC divided by AC and RC sum was RAR.

Results and Discussion

Differences according to SAGR were in the interval from 0.53 of Lr 1 in 2010 to 0.71 of Lr 12 in 2011. Values of tested genotypes were increased for 0.022 in average when specific genes were present in comparison to adequate nonspecific Lr gene containing NIL. The SAGR of Lr 12 line was higher for 0.01 than of Lr 22b, which could be explained by complementary effect with genes as were Lr 2c, Lr34, Lr 13 etc. SAGR of basic variety Thatcher was closest to Lr 2c line indicating different Lr genes accumulations in NILs. The average severity of *Puccinia triticina* was higher for 16% unexpected in more continuously dry year. Decreased severity of obligate parasite was related more to antagonism with *Pyrenophora tritici repentis* than to *Septoria tritici* (Jerkovic 2008). The 5 Lr lines were of the same maximal rust severities 50 or 70 (SAGR 0.53-0.60) as another 5 were such but with values 40 or 50 (SAGR 0.67-0.71), also indicating the different nonspecific resistance genes effects in near isogenic lines beside race specific ones. Less maximally rusted than expected by SAGR where those with prolonged LP, visible through lower parasite development on last two leafs in period from the end of May (Table 1) and pronounced as race specific resistance genes

carried like Lr 1, Lr 2a, Lr 16, Lr 19,Lr 21, Lr 29 and Lr 37. Decreasing of maximal severity of the parasitic attack was strong negative correlated to SAGR (r= -0.75 to -0.80). Without specific resistant genotypes recognized by lower initial infection severities, r-values were -0.81 to -0.86. Maximal severities of parasite attacks in two years was absolute correlated (r=0.98). Lower maximal intensity than on Thatcher was achieved by genes different positioned on chromosomes (McIntosh et al. 1995; Purnhauser et al. 2000). For confirmation of the above, recent dividing of Lr genes in two groups, the Lr 1 (5D) and Lr 2a (2D) genes were regional express able through lower RT at seedling stage to the particular races of pathogen (Boskovic & Browder 1976, Prijic & Jerkovic 2010), while generally most effective by RT decrease were Lr 19 (7D) and Lr 24 (3D) (Jerkovic 1992); even chromosomes from D genome were described as suppressor genes containing (Dyck 1987, Nelson et al. 1997). Lr 1 and Lr 19 were recently proved as such according to lower RT (Tables 2 and 3). The nature of Lr 15 gene was not possible to discover according to analyses of regional host parasite interactions. Adequate race 162(A) proving its specific character was isolated in India (Gupta et al. 2008).

Genes like Lr 2, Lr 2b, Lr 3 or Lr 3b were SAGR increase able in comparison to basic Thatcher and much larger distanced from Lr 22b by the character than was achieved by specific resistance genes simultaneous presence with at last mentioned gene. Such, they were defined as adult plant or nonspecific resistance controlled genes with lower effect as were Lr 13 or Lr 48 and Lr 49 (Saini et al. 2002). Presence of such genes was followed by leaf tip necrosis (Schnurbrush et al. 2004, Mishra et al. 2005). However, near isogenic lines were multiple based when 2D chromosome was focused, where discovered genes were responsible for day light insensitivity (Worland & Law 1986) likely to be responsible for SAGR increase. For the difference Lr 34 was defined as non-hypersensitive (Rubiales & Niks 1995) but it was LP prolongable as well

Table 1. Leaf rust severities and SAGR of Lr NILs and background variety

Genotype	Leaf rust severities						
	2004			2011		SAGR	
	21.05	3. 06	9.06	16. 06		2010	2011
Lr 1	T	10	40	70	50	0.53	0.56
Lr 2a	T	10	30	60	50	0.54	0.56
Lr 2c	5	25	70	80	60	0.57	0.60
Thatcher	5	20	60	80	60	0.55	0.58
Lr 2b	5	20	60	70	50	0.59	0.63
Lr 3	5	15	60	70	50	0.60	0.63
Lr 3b	5	20	60	70	50	0.60	0.63
Lr 34	5	20	60	70	50	0.59	0.62
Lr 13	10	30	50	60	50	0.62	0.64
Lr 12	5	10	30	40	30	0.71	0.72
Lr 15	5	15	40	60	50	0.66	0.67
Lr 16	0	T	25	30	40	0.67	0.69
Lr 21	5	10	40	50	40	0.66	0.67
Lr 22a	5	30	40	50	40	0.68	0.70
Lr 22b	5	40	50	50	40	0.70	0.71
Lr 29	T	T	10	50	60	0.55	0.57
Lr 19	0	15	40	50	40	0.66	0.68
Lr 37	T	15	40	50	40	0.68	0.68
Lr 38	5	15	40	40	40	0.69	0.69

as adequate NIL was with higher SAGR, than was of Thatcher. Carbon flux discovered by Bolton et al. (2008) and lower RAR value in comparison to nonspecific resistance controlling genes were supplement to define unusual gene type. According to recent results Lr 34 was responsible for the realest of the seed content described by Aoki et al. (2006) and most likely to be sucrose transporter (SUT). Sugar, arabinose, xylose and mannose were adequate for haustorium mother cell formation in vitro, whereas only one hour of water lack during the initial infection was enough for its rejecting (Heat 1990). Enzymes for starch or soluble carbohydrates cleaving were weighted 45-59 Kd (Carneiro et al. 2004) and had to be transported last during heat and drought stress explaining different colours of leaf tips (K and Ca). Their optimum at higher temperatures in acid environment (Halverson & Barry 2003) was adequate to the state.

RAR values of Lr 1, Lr 15, Lr 19 were equal and distanced from Lr 22b line more than Lr 16, Lr 37 and Lr 29. The active sites of adequate enzymes could not be the same also confirmed by different RTs when mono pustule isolate of parasite was applied (Boskovic & Browder 1976, Jerkovic 1992, Mesterhazy et al. 2000, Bulos et al. 2006, Winzeler et al. 2000, Prijic & Jerkovic 2010). Simulated results when RAR values sum was equalized with seven defined those genes as responsible for 3 to 19% of root proteins lack. Results confirmed enough energetic level of all tested enzymes to cleave disulphide linkage of gluten (Tables 2 and 3). Prolamins were of remarkable diversity in size, gliadin, glutenin ratio, frame shift mutations and sulfur content (Nagy et al. 2005). Total of 35-40% of wheat gluten was gliadin characterized by intra chains cross links so called bounds, branches or bridges, as well as such amounted were intra and extra bounded glutenin (Wall 1971). Gliadin first reached their proportion in seed (Wrigley et al. 1980) and indicated the external bounds as the primary target of hydrolysis or enzymes. The protein disulfide isomerases (PDI) were responsible for cysteine join in covalently different polypeptides or stabilization of protein via bounds (Ciaffi et al. 1999). Primarily the gliadin monomers were transported to vesicles via Golgi apparatus (GA) according to Rubin et al. (1992). Bound producing effect of PDI enzymes was linked to maturating conditions characterized by higher temperatures and drought. Fructose-1, 6-diphosphatase from spinach leaf chloroplasts weighted 92-115 Kd, while

Table 2. Reaction to *Puccinia triticina* races, LGR and nitrogen distribution of three-week-old seedlings

Genotype	RT	LGR	RC	AC	RAR	RC+AC	RC/AC	RPC	APC
Lr 1	0;-4	L	1.8	5.4	0.25	7.2	0.33	10.3	30.8
Lr 15	4	H2	1.7	5.2	0.25	6.9	0.33	8.0	29.7
LR 34	4	M	1.9	4.7	0.29	6.6	0.40	10.8	26.8
THATCHER	4	M	2.0	5.0	0.29	7.0	0.40	11.4	28.5
LR 22B	4	H2	2.0	4.5	0.31	6.5	0.44	11.4	25.7
LR 38	4	H2	2.1	4.8	0.31	6.9	0.44	13.1	27.4

Table 3. Reaction to *Puccinia triticina* races, LGR and nitrogen distribution of two-week-old seedlings

Genotype	RT race 2 77	LGR	RC	AC	RAR	RC+AC	RC/AC	RPC	APC
Lr 1	0;-4	M	2.2	5.2	0.30	7.4	0.42	12.5	29.6
Lr 15	4	H2	1.7	3.9	0.30	5.6	0.44	8.0	22.2
Lr 19	0;-4	H2	1.8	4.2	0.30	6.0	0.43	10.3	23.9
LR 16	4	H2	2.0	4.2	0.32	6.2	0.42	11.4	23.9
LR 37	4	H2	2.2	4.4	0.33	6.6	0.51	12.5	25.1
Lr 29	4	H2	2.1	4.0	0.34	6.1	0.52	12.0	22.8
LR 38	4	H2	2.2	4.2	0.35	6.4	0.54	12.5	24.0
Lr 22A	4	H2	2.2	4.1	0.35	6.3	0.54	12.5	23.4
Lr 22B	4	H2	2.8	5.2	0.35	8.0	0.56	16.0	29.6

on pH 8.8 were splitting in native monomers of 54 and 60 Kd. According to Lr 21 cloning by Huang et al. (2003) calculated gene product weight was over 100 Kd. Such, enzymes of Lr genes could not pass through wheat cell wall (Lazaro et al. 1975) and were defined as endo-proteases. Logically, production of lighter units of proteins happened only in wheat germ. Different RAR values were related with hydrolytic stability of enzymes.

Nitrogen and other nutritives from compost were not absorbed in above described growth phases according to Vuurde & Tonneyck (1978). Identified genes in hard red spring wheat (Oelke & Kolmer 2004, 2005, Martinez et al. 2007) as in soft spring ones (Kolmer 2003, Wanishe & Millus 2004) were in agreement with achieved protein contents in seed, as well as state that genes for gluten synthesis were not linked with Lr 19 (Slikova et al. 2003). In addition, there was no relation between protein content and RAR values according to its same result as was of Lr 1 line.

For the digression, hypersensitive reaction was related to same mechanism with different source of sulphur from fungi cell wall or membrane instead of gluten. Sulphur dioxide, which was expected to be consequence of enzymes produced by specific Lr genes, was transferred in acid forms when reached chloroplast while fall of PH below 6.5 caused chlorophyll degradation (Jolivet et al. 1992).

Conclusions

According to recently presented results, genes controlling race specific resistance to *Puccinia triticina* were particular endoprotease also responsible for gluten disulphide bridges degradation consequential through increased transfer of proteins in plant up ground parts from seed, decreased SAGR and increased LGR in comparison to Lr 22b line. Enzymes were with different hydrolytic stability according to different effects to the gluten. Other type of Lr genes cause of increased SAGR were most likely related to starch and its products degrading and had no influence on RAR. For the difference of both above-mentioned gene groups, according to slightly decreased RAR and SAGR, increase of Lr 34 was adequate to transporters enzymatic family. However, two different drought and heat stress respond mechanisms were discovered. When race nonspecific Lr genes were present, plant growth was because of below parts followed by their rejection. Vice versed, specific Lr genes reduced photosynthesis and across locally more viable water prevented starch and gluten formation allowing further growth of recently present organs consequential through prolonged total leaf area duration and equilibrated growth.

Recently proposed method could be used for discovering of Lr genes without interactions with parasite, but did not signal the opportunity for their precise identification.

References

Aoki, N., Scofield, G.N., Wang, X., Offler, C.E., Patrick, J.W., & Furbank, R.T. (2006). Pathway of sugar transport in germinating wheat seeds. *Plant physiology, 141*(4), 1255-63. pmid:16766668. doi:10.1104/pp.106.082719

Anker, C.C., & Niks, R.E. (2000). Genetics of prehaustorial resistance to wheat leaf rust in diploid wheat. *Acta Phiytopathologica and Entomologica Hungarica, 35*(1-4), 23-30.

Bolton, M.D., Kolmer, J.A., Xu, W.W., & Garvin, D.F. (2008). Lr34-mediated leaf rust resistance in wheat: transcript profiling reveals a high energetic demand supported by transient recruitment of multiple metabolic pathways. *Molecular plant-microbe interactions , 21*(12), 1515-27. pmid:18986248

Bošković, M.M., & Browder, L.E. (1976). A comparison of phatogenicity of Puccinia recondita tritici in Europe, The United States and Canada. *Plan Dis. Reptr, 60*, 278-280.

Browder, L.E. (1985). Parasite - host: Environment specificity in the cereal rusts. *Annual Review of Phytopathology, 23*(1), 201-222. doi:10.1146/annurev.py.23.090185.001221

Bulos, M., Echarte, M., & Sal, C. (2006). Occurence of rust resistance gene Lr 37 from aegilops ventricosa in Argentina cultivars of wheat. *Electron. J. Biotechnol, 9*, 5.

Carneiro, C.N., Isejima, E.M., Samuels, R.I., & Silva, C.P. (2004). Sucrose hydrolases from the 9, 5 midgut of the sugarcane stalk borer Diatraea sacchaaralis. *J. Insect Physiol., 50*(11), 1093-1101.

Chester, K.S. (1946). *The cereal rusts*. (p. 269). Waltham, Mass.

Ciaffi, M., Dominici, L., Tanzarella, O.A., & Porceddu, E. (1999). Chromosomal assignment of gene sequences coding for protein disulphide isomerase (PDI) in wheat. *Theoretical and Applied Genetics, 98*(3-4), 405-410. doi:10.1007/s001220051086

Cumming, R.C., Andon, N.I., Haynes, P.A., Park, M., Fisher, W., & Shubert, D. (2004). Protein disulfide bond formation in the cytoplasm during the oxidative stress. *Journal of biological chemistry, 279*, 21749-21758.

Das, M.K., Rajaram, S., Mundt, C.C., & Kronstad, W.E. (1992). Inheritance of slow-rusting resistance to leaf rust in wheat. *Crop Science, 32*, 1452-1458.

de Wit, P.J.G.M., & van Kan, J.A.L. (1993). Is durable resistance against fungi attainable through biotechnological procedures. In T. Jacobs & J.E. Parlevliet (Eds.), *Durability of disease resistance*. (pp. 57-70). Kluwer Academic Publishers.

Dyck, P.L. (1987). The association of a gene for leaf rust resistance with the chromosome 7D suppressor of stem rust resistance in common wheat. *Genome, 29*, 467-469.

Dyck, P.L. (1991). Genetics of adult-plant leaf rust resistance in 'Chinese Spring' and 'Sturdy' wheats. *Crop Science, 31*, 309-311.

Flor, H.H. (1955). Host parasite interaction in flax rust - its genetics and other implications. *Phytopathology, 45*, 680-685.

Flor, H.H. (1971). Current status of the gene-for-gene concept. *Annual Review of Phytopathology, 9*(1), 275-296. doi:10.1146/annurev.py.09.090171.001423

German, S.E., & Kolmer, J.A. (1992). Effect of gene Lr34 in the enhancement of resistance to leaf rust of wheat.*Theoretical and Applied Genetics, 84*, 97-105.

Gupta, V.S., Khan, R.R., Rajwade, A.V., Reddy, D.M.R., Hosmani, P., Chavan, S., . . . Sain, R.G. (2008). Molecular mapping of

leaf rust resistance gene Lr 15 in bread wheat. In: *11th Wheat genetics symposium*. 724-726.
Heath, M.C. (1990). Influence of carbohidrates on the induction of haustoria of the cowpea rust fungus in vitro. *Experimental Mycology, 14*(1), 84-88.
Halverson, K.M., & Barry, B.A. (2003). Sucrose and glycerol effects on photosystem 2. *Biophysical Journal, 85*, 1317.
Harder, D.E., & Chong, P. (1984). Structure and physiology of haustoria. In A.P. Roelfs & W.R. Bushnell (Eds.), *Cereal rust*. (pp. 431-473). Academic press.
Huang, L., Brooks, S.A., Li, W., Fellers, J.P., Trick, H.N., & Gill, B.S. (2005). Map-Based cloning of Leaf Rust Resistance Gene Lr21 From the Large and Polyploid Genome of Bread Wheat. *Genetics, 164*, 656-664.
Jacobs, T.H. (1990). Abortion of infection structures of wheat leaf rust in susceptible and partially resistance wheat genotypes. *Euphytica, 45*, 81-86.
Jacobs, T., & Kiriswa, F.K.L. (1993). The relation between the developmental stage of wheat and barley and the latency period and infection frequency of leaf rust. *Cereal Research Communication, 21*(2-3), 195-200.
Jerković, Z. (1992). Identifying genes for Puccinia recondite f. sp. tritici resistance in wheat lines. *J. Genetics and Breeding, 46*, 163-166.
Jerković, Z., Zadoks, J.C., & Momčilović, V. (1993). Inheritance of two characters of incomplete resistance to Puccinia recondite tritici. *8th International Wheat Genetics Symposium*, China, Abstracts. 110-111.
Jerković, Z., & Putnik-Delić, M. (2004). The forecast of maximal leaf rust intensity of wheat varieties in field according to seed ling resistance in green house. *Acta fytotechnica et zootechnica, 2*, 107-109.
Jerkovic, Z. (2008). Wheat variety specific model of parasite development and effects in semiarid regions. *International Conference "Conventional and Molecular Breeding of Field and Vegetable Crops"*, November 24-27, Institute of Field and Vegetable Crops, Novi Sad, Serbia, Conference Proc. 277-282. Retrieved from
http://www.nsseme.com/about/inc/knjige/Breeding08-ConferenceProceedings.pdf.
Jerković, Z., & Prijić, Ž. (2009). Phytopathology consequence of growing of different wheat variety in semiarid region. *IV Symposium with international participation Innovation in crop and vegetable production.* 23-24. Octobar, Belgrade. Book of abstracts. 34-35.
Jerkovic, Z., & Prijic, Z. (2012). Leaf rust specific resistance genes effect on degradation of fungicides with sulfur.*Plant protection, 63* (1), 29-35.
Johanson, C.O., & Browder, L.E. (1966). Seventh revision of physiologic races of Puccinia recondita f. sp. tritici. *Plant Disease Reporter, 50*, 756.
Jolivet, P., Bergeron, E., & Kein, P. (1992). Oxidation of Elemental Sulfur and Sulfite by Chloroplasts. *Phyton, Annales Rei Botanicae, Horn, 32*(3), 59-62.
Khan, M.A., Trevathan, L.E., & Robin, J.T. (1997). Quantitative relation between leaf rust and wheat yield in Mississippi. *Plant Disease, 81*, 769-772.
Koegel, G., Beissman, H.J., Reisener, H.M., & Kogel, K.H. (1988). A single glycoprotein from Puccinia graminis f. sp. tritici cell walls elicits the hypersensitive lignification response in wheat. *Physiological and Molecular Plant pathology, 33*(2), 173-185.
Kolmer, J.A., & Liu, J.Q. (2001). Simple inheritance of the partial resistance to leaf rust in two wheat cultivars. *Plant Pathology, 50*(5), 546-551.
Kolmer, J.A. (2003). Postulation of leaf rust resistance genes in selected soft red winter wheats. *Crop Science, 43*, 1266-1274. doi:10.2135/cropsci2003.1266
Kolmer, J.A., Anderson, J.A., & Flor, M. (2010). Chromosome location, linkage with simple sequence repeated markers, and leaf rust resistance conditioned by gene Lr 63 in wheat. *Crop Science, 50*, 2303-2395.
Lazaro, J.J., Cueca, A., Gorge, J.L., & Mayor, F. (1975). Fructose-1, 6-diphosphatase from spinach leaf chloroplasts. Molecular weight transitions of the purified enzymes. *Plant Science Letters, 5*(1), 49-56.
Lee, T.S., & Shaner, G. (1985). Oligogenic inheritance of length of latent period in six slow leaf-rusting wheat cultivars. *Phytopathology, 75*(6), 636-646. doi:10.1094/Phyto-75-636
Loegering, W.Q. (1978). Current concepts in interorganismal genetics. *Annual Review of Phytopathology, 16*(1), 309-320. doi:10.1146/annurev.py.16.090178.001521
Martínez, F., Niks, R.E., Singh, R.P., & Rubiales, D. (2001). Characterization of Lr46, a gene conferring partial resistance to wheat leaf rust. *Hereditas, 135*(2-3), 111-4. pmid:12152322. doi:10.1111/j.1601-5223.2001.00111.x
Martínez, F., Sillero, J.C., & Rubiales, D. (2007). Resistance to leaf rust in cultivars of bread wheat and durum wheat grown in Spain. *Plant Breeding, 126*(1), 13-18. doi:10.1111/j.1439-0523.2007.01287.x
Masson, P., Tomè, D., & Popineau, Y. (1986). Peptic hydrolysis of gluten, glutenin and gliadin from wheat grain: Kinetics and characterisation of peptides. *Journal of the Science of Food and Agriculture, 37*(12), 1223-1235. doi:10.1002/jsfa.2740371212
McIntosh, R.A., Wellings, C.R., & Park, R.F. (1995). *Wheat rust. An atlas of resistance genes*. Melbourne, Dordecht, Boston, London: CSIRO and Kluwer Academic Publishers.
Mesterhazy, A., Bartos, P., Goyeau, H., Niks, R.E., & Chosz, M. (2000). European virulence survey for leaf rust in wheat. *Agronomie, 20*(7), 793-804. doi:10.1051/agro:2000104
Mishra, A.N., Kaushal, K., Yadav, S.R., Shirsekar, G.S., & Pandey, H.N. (2005). A leaf rust resistance gene, different from Lr34, associated with leaf tip necrosis in wheat. *Plant Breeding, 124*(5), 517-519. doi:10.1111/j.1439-0523.2005.01140.x
Nagy, I.J., Takács, I., Juhász, A., Tamás, L., & Bedo, Z. (2005). Identification of a new class of recombinant prolamin genes in wheat. *Genome,* 48(5), 840-7. pmid:16391690
Nelson, R.R. (1978). Genetics of horizontal resistance to plant diseases. *Annual review of phytopathology, 16*, 359-378.
Nelson, J.C., Singh, R.P., Autrique, J.E., & Sorrelli, M.E. (1997). Mapping genes conferring and suppressing leaf rust resistance in wheat. *Crop Science, 37*, 1928-1935.
Niks, R.E. (1981). Early abortion of colonies of leaf rust Puccinia hordei in partially resistant barley seedlings. *Canadian Journal of Botany, 60*, 714-723.
Niks, R.E., & Dekens, R.G. (1991). Prehaustorial and posthaustorial resistance to wheat leaf rust in diploid wheat seedlings. *Phytopathology, 81*(8), 847-851. doi:10.1094/Phyto-81-847
Oelke, L.M., & Kolmer, J.A. (2004). Characterisation of leaf rust resistance in hard red spring wheat cultivars. *Plant Disease, 88*(10), 1127-1133. doi:10.1094/PDIS.2004.88.10.1127
Oelke, L.M., & Kolmer, J.A. (2005). Genetics of leaf rust resistance in spring wheat cultivars Alsen and Norm. *Phytopathology, 95*(7), 773-778.
Parlevliet, J.E., & van Ommeren, A. (1975). Partial resistence if barley to leaf rust Puccinia hordei. II: Relationship between field trials, microplot tests and latent period. *Euphytica, 24*(2), 293-303. doi:10.1007/BF00028194
Parlevliet, J.E. (1978). Further evidence of polygenic inheritance of the partial resistance in barley to leaf rust, *Puccina hordei*. *Euphytica, 27*(2), 369-379. doi:10.1007/BF00043161
Parlevliet, J.E. (1986). Pleiotropic association of infection frequency and latent period of two barley cultivars partially resistant to barley leaf rust. *Euphytica, 35*(1), 267-272. doi:10.1007/BF00028565

Parlevliet, J.E., & Kievit, C. (1986). *Development of barley leaf rust, Puccinia hordei, infections in barley. I. Effect of partial resistance and plant stage*. (pp. 953-959). doi:10.1007/BF00028605

Prijic, Z., & Jerkovic, Z. (2010). Genetic base of durable resistance to *Puccinia triticina* of two serbian varieties. *Genetika, 42*(2), 307-312. doi:10.2298/GENSR1002307P

Prunhauser, L., Gyulai, G., Csosz, M., Heszky, L., & Mesterhazy, A. (2000). Identification of leaf rust resistance genes in common wheat bz molecular markers. *Acta Phytopathologia et Entomologica Hungarica, 35*(1-4), 31-36.

Rijsdijk, F.H., & Zadoks, J.C. (1976). Assessment of risks and losses due to cereal rusts in Europe. In: *Proc. 4th Conf. Eur. Mediterr. Cereal Rusts*. 60-62.

Rubin, R., Levanony, H., & Galili, G. (1992). Evidence for the presence of two different types of protein bodies in wheat endosperm. *Plant physiology, 99*(2), 718-24. pmid:16668945

Rubiales, D., & Niks, R.E. (1995). Characterization of Lr34, a major gene conferring nonhypersensitive resistance to wheat leaf rust. *Plant Disease, 79*(12), 1208-1212. doi:10.1094/PD-79-1208

Saini, R.G., Kaur, M., Singh, B., Sharma, S., Nanda, G.S., Nayar, S.K., . . . Nagarajan, S. (2002). Genes Lr 48 and 49 for hypersensitive adult plant leaf rust resistance in wheat (Triticum aestivum L.). *Euphytica, 124*(3), 365-370. doi:10.1023/A:1015762812907

Samborski, D.J., & Dyck, P.L. (1982). Enhancement of resistance to Puccinia recondite by interactions of resistance genes in wheat. *Can. J. of Plant Pathology, 4*, 152-156.

Schnurbusch, T., Paillard, S., Schori, A., Messmer, M., Schachermayr, G., Winzeler, M., & Keller, B. (2004). Dissection of quantitative and durable leaf rust resistance in Swiss winter wheat reveals a major resistance QTL in the Lr34 chromosomal region. *TAG, 108*(3), 477-84. pmid:14523520. doi:10.1007/s00122-003-1444-4

Shewry, R.P., & Halford, G.N. (2002). Cereal seed storage proteins: Structures, properties and role in grain utilization. *Journal of Experimental Botany, 53*(370), 947-958.

Slikova, A., Gregova, E., Bartoš, P., & Kraic, J. (2003). Selection of wheat genotypes with combination of resistant gene Lr 19 and glutenin alleles with positive effect on bread making quality by means of protein markers. In: XVI Slovak and Czech plant protection conference, Nitra, Abstracts. 318-319.

Stakman, E.C., Stewart, D.M., & Loegering, W.Q. (1962). *Identification of physiological races of Puccinia graminis var. tritici*. (pp. 1-53). Washington, DC, USA: United States Department of Agriculture.

The, T.T., Latter, B.H.D., Intosh, M.R.A., Elison, F.W., Brennan, P.S., Fishe, J., . . . Wilson, R.E. (1988). Grain yields of near isogenic lines with added genes for stem rust resistance. In: *Proc. Int. Wheat. Genetic Symp*. 7. 901-906.

Wanishe, Y.A., & Milus, E.A. (2004). Seedling resistance genes to leaf rust in soft red winter wheat. *Plant Disease, 88*(2), 136-141. doi:10.1094/PDIS.2004.88.2.136

Wall, J.S. (1971). Disulfide bonds: determination, location and influence on molecular properties of protein.*Agricultural and food chemistry,* 19(4), 6-19.

Winzeler, M., Mesterhazy, A., & Park, R.F. (2000). Resistance of European wheat germplasm to leaf rust. *Agronomie, 20*(7), 783-792. doi:10.1051/agro:2000175

Worland, A.J., & Low, C.N. (1986). Genetic analysis of chromosome 2D of wheat. I. The location of genes affecting height, day-lenght insensitivity, hybrid dwarfism and yellow rust resistance. *Z. Pflanzenzuchtg, 96*, 331-345.

Wrigley, C.W., Cros, D.L.D., Archer, M.J., Downie, P.G., & Roxburgh, C.M. (1980). The Sulfur Content of Wheat Endosperm Proteins and Its Relevance to Grain Quality. *Australian Journal of Plant Physiology, 7*(6), 755-766. doi:10.1071/PP9800755

van der Plank, J.E. (1963). *Plant diseases: Epidemic and control*. (p. 349). New York-London: Academic Press.

van der Plank, J.E. (1976). Four essays. *Annual Review of Phytopathology, 14*, 1-10.

Vuurde, J.W., & Tonneyck, A.E. (1978). Effect of foliar application of urea on wheat growth in relation to plant age, soil fertility and light intensity. *Plant and Soil, 50*(1-3), 473-477.

Funkcije Lr gena osim redukcije *Puccinia triticina*

Zoran Jerković • Željana Prijić • Veselinka Đurić • Mirjana Lalošević

Sažetak: Geni za specifičnu otpornost prema prouzrokovaču lisne rđe pšenice (Lr) smanjivali su rast sledećih delova stabla (SAGR) za 0,01-0,03 u poređenju sa osnovnom sortom Tačer ili Lr 22b linijom. RAR, odnosno podeljen sadržaj azota u korenu sa zbirom navedene vrednosti i one u nadzemnom delu u stadijumu sejanaca, bio je umanjen u odnosu na poslednje pomenute genotipove. Ubrzanje transporta proteina u nadzemne delove preko gena za specifičnu otpornost je bilo 3-19%. Enzimi za specifičnu otpornost su svrstani u glutenaze, odnosno endoproteaze, odgovorne za oslobađanje sumpora iz disulfidnih mostova pri prolasku proteina kroz klicu. Razlike po RAR-u između Lr 1, Lr 15, Lr 19 sa istim vrednostima i nešto različitih Lr 16, Lr 29 i Lr 37 od Lr 22b i Lr 38 linija nosilaca nespecifičnih Lr gena bilo je objašnjeno različitom hidrolitičkom stabilnošću. Lr 34 je po RAR-u bio sličan grupi specifičnih gena, ali povišen SAGR u odnosu na Tačer i produžen LP ukazivali su na enzimatsku familiju transportera šećera pre nego ABC transportere. Dva različita sistema za tolerantnost prema suši odnosno stresu usled visokih temperatura vazduha povezana su sa genima za otpornost prema *Puccinia triticina*. Rasno specifični geni su umanjivali fotosintezu preko produkata degradacije glutena u listu, ujedno povišujući LGR i snižavajući SAGR, dok su nespecifični delovali obrnuto i stoga povezani sa degradacijom skroba. Kod linija sa poslednje pomenutim genima primećeno je ranije gubljenje fotosintetičke aktivnosti postojećih organa i brži rast sledećih na uštrb prethodno formiranih.
Ključne reči: enzimi pšenice, gluten, lisna rđa pšenice, LR geni, *Puccinia triticina*, SAGR