https://doi.org/10.17816/ecogen14530

GENETIC DIVERSITY OF CEREAL CROPS FOR POWDERY MILDEW RESISTANCE

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Cite this article as: Radchenko EE, Abdullaev RA, Anisimova IN. Genetic diversity of cereal crops for powdery mildew resistance. *Ecological genetics*. 2020;18(1):59-78. https://doi.org/10.17816/ecogen14530.

Received: *28.06.2019* Revised: *12.11.2019* Accepted: *19.03.2020*

❀ Powdery mildew (causal agent *Blumeria graminis*) is a widespread and harmful fungi disease of cereal crops especially in the regions with humid climate. The pathogen is differentially interacting with plant host genotypes. Growing cereal crop varieties protected with different resistance genes is the most rational, costly and ecologically safe way of combating powdery mildew. The supply of effective genes can be increased due to studies of crop genetic resources collection, introgression of resistance from wild relatives, and also at the expense of mutant forms created with the use of traditional (induced mutagenesis) and biotechnological methods including genome editing. This causes the increasing interest to searching and identifying resistance genes, elucidation of their structural and functional organization, and analysis of molecular mechanisms of the character development. The review summarizes modern information on the identified genes of powdery mildew resistance of the main cereal crops – wheat, barley and oat. The list of wheat and barley genes identified at the molecular level is presented. It includes genes encoding NLR and CNL proteins (*Pm2*, *Pm3*, *TaMla2*, *TaMla3* genes of wheat, rye *Pm8* gene, barley *Mla* gene), receptor-like proteins (barley *Mlo* gene), transport proteins and receptor-like kinases (*Lr34*, *Lr67*, *Pm21* of wheat).

❀ **Keywords:** cereals; *Blumeria graminis*; parasite–plant host interaction; resistance; R-genes; proteins; structural and functional organization.

ГЕНЕТИЧЕСКОЕ РАЗНООБРАЗИЕ ЗЕРНОВЫХ КУЛЬТУР ПО УСТОЙЧИВОСТИ К МУЧНИСТОЙ РОСЕ

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Для цитирования: Радченко Е.Е., Абдуллаев Р.А., Анисимова И.Н. Генетическое разнообразие зерновых культур по устойчивости к мучнистой росе // Экологическая генетика. – 2020. – Т. 18. – № 1. – С. 59–78. https://doi.org/10.17816/ecogen14530.

Поступила: *28.06.2019* Одобрена: *12.11.2020* Принята: *19.03.2020*

❀ Мучнистая роса (возбудитель *Blumeria graminis*) — одно из наиболее распространенных и вредоносных грибных заболеваний зерновых культур, особенно в регионах с влажным климатом. Для патогена характерно дифференциальное взаимодействие с генотипами растений-хозяев. Наиболее рациональный, дешевый и экологически безопасный способ борьбы с мучнистой росой — возделывание сортов злаковых культур, защищенных разными генами устойчивости. Запас эффективных генов может пополняться за счет изучения коллекции генетических ресурсов культурных растений, интрогрессии устойчивости от диких родичей, а также за счет мутантных форм, созданных с помощью традиционных (искусственный мутагенез) и биотехнологических методов, включая редактирование генома. В этой связи в последние десятилетия возрос интерес к поиску и идентификации генов устойчивости, выяснению их структурно-функциональной организации, а также анализу молекулярных механизмов формирования признака. В обзоре обобщена современная информация об идентифицированных генах устойчивости к мучнистой росе основных зерновых культур — пшеницы, ячменя и овса. Приведен список идентифицированных на молекулярном уровне генов пшеницы и ячменя. Среди них: гены, кодирующие белки NLR и CLR (*Pm2*, *Pm3*, *TaMla2*, *TaMla3* мягкой пшеницы, *Pm8* ржи, *Mla* ячменя), рецептор-подобные белки (*Mlo* ячменя), транспортные белки и рецептор-подобные киназы (*Lr34*, *Lr67*, *Pm21* пшеницы).

❀ **Ключевые слова:** злаки; *Blumeria graminis*; взаимодействие паразит–хозяин; устойчивость; R-гены; белки; структурно-функциональная организация.

INTRODUCTION

Powdery mildew is a widespread and harmful fungal disease of cereal crops, especially in regions with humid climates. This disease affects all aboveground parts of the plant – leaves, leaf sheath, stem, glumes, and awns – in years of strong development.

In the affected plants, photosynthesis in leaves is reduced, and physiological processes are significantly changed (increased water loss and breathing intensity). Plants exhibit slow growth and reduced tillering ability, decreased grain number per spike and seed weight.

The causal agent of powdery mildew disease of cereal crops is the obligate parasite, *Blumeria graminis* DC. The species exhibits several morphologically similar forms specialized for different host plants [1]. Several specialized forms are found on cereal crops: f. sp. *tritici* Marchal (on species of genera *Triticum* L. and *Aegilops* L. and a number of other wild cereal crops), f. sp. *hordei* Marchal (on species of the genus *Hordeum* L.), f. sp. *secalis* Marchal (on species of the genus *Secale* L.), and f. sp. *avenae* Marchal (on species of the genus *Avena* L.). Until 2001, powdery mildew was not detected on triticale. The spread of this disease to triticale in Europe was a result from the emergence of new forma specialis *triticosecale* [2, 3].

Plant resistance is a primary factor limiting the harm caused by powdery mildew. The selection of resistant genotypes is an effective, inexpensive, and ecologically sound method to combat the disease. Unfortunately, differential interaction with host plant genotypes is typical for the pathogen [4]. This property allows adaptive microevolution of the fungus to resistance genes in uniformly cultivated varieties.

The interaction of *B. graminis* with plants reflects the "gene-for-gene" relations [5]: each gene of resistance in the host plant corresponds to specific virulence gene in the parasite. The mutation of a parasite virulence gene is associated with the loss of effectiveness of a resistance gene in the host. Resistance genes are usually dominant as they are evolutionarily older; parasite (driven partner) virulence is controlled by recessive genes. Resistance and avirulence have "plus"-functions (interacting gene products), whereas susceptibility and virulence have "minus"-functions [6].

Different genes of resistance to different pathogen populations can be expressed in the same variety. Genes of resistance can differ in the stability of manifestation, which depends on environmental conditions and genetic background. Resistance genes manifested in seedlings ("juvenile genes") act, as a rule, during the entire plant life cycle. At the same time, the expression of resistance can change during the plant ontogenesis.

Resistance of the host plant is associated with a hypersensitivity reaction – a plant's defensive reaction manifested in rapid local cell death in response to the penetration of a harmful organism, accompanied by accumulation of toxic products in the dead cells. Interaction with phytopathogens involves several stages: extraction of inducers (elicitors), recognition of elicitors by the plant cell employing receptors, transduction of signal to the genome, activation of transcription of genes of the immune response, and synthesis of defense compounds [7].

To prevent epiphytotic impacts of powdery mildew, cultivating varieties with different resistance genes are required. The stock of effective genes can be replenished by studying the cultivated plant collection, introgression of resistance genes from wild relatives, and production of mutant forms using conventional genetical and biotechnological methods. Introgression greatly impacts the diversity of cereal crop resistance genes. Inheritance of resistance to powdery mildew is well studied for wheat and barley. However, information on the genetic structure of loci and encoded products is known only for a small number of genes.

This review aimed to generalize literature data on the polymorphism of cereal crops and resistance genes for powdery mildew.

GENES CONTROLLING CEREAL CROP RESISTANCE TO POWDERY MILDEW

Currently, 92 alleles in 62 loci (*Pm1–Pm65*) that control wheat resistance to powdery mildew (Table 1) were identified. Most genes were dominant and expressed throughout ontogenesis. About 44 alleles were of *Triticum aestivum* L., 26 alleles were transferred from different species of the genus *Triticum*, 11 were from *Aegilops* spp., 5 were from *Secale cereale* L., and 6 were introgressed from *Dasypyrum villosum* (L.) Borbás (synonym of *Haynaldia villosa* (L.) Schur), *Thinopyrum ponticum* (Podp.) Z.-W. Lin & R.-C. Wang, *Thinopyrum intermedium* (Host) Barkworth & D.R. Dewey, and *Agropyron cristatum* (L.) Gaertn. There were more than 20 resistance genes that were given temporary symbols.

Besides genes with clear phenotypic manifestation, high levels of resistance to fungus (mainly age-specific, manifested in the phase of flag leaves)

can be controlled with small genes (quantitative trait loci – QTL). A minimum of 119 age-specific resistance QTL were mapped on 21 wheat chromosomes. Durable resistance of adult plants to leaf rust, yellow rust, and stem rust, as well as to powdery mildew, was provided by gene clusters *Lr34/Yr18/Pm38/Sr57* (chromosome 7DS), *Lr46/Yr29/Pm39/Sr58* (1BL), and *Lr67/Yr46/Pm46/Sr55* (4DL) [84].

Introgressed genes ensure a wider range of durable resistance in comparison with recipient species genes due to differences in the structure of coding sequences. In China, varieties protected by gene *Pm21* from *D. villosum* (translocation of T6AL.6VS), despite extensive cultivation, were resistant to the pathogen for more than 40 years. Currently, the new breeding material with resistance gene *Pm40* introduced from intermediate wheatgrass considers as very promising [85]. However, both own and introgressed genes in soft wheat have different effectiveness and duration of useful life. The fungus can overcome resistance of varieties with alien genes as quickly as resistance from closely related

Table 1

Wheat powdery mildew resistance genes

Chromosome	Resistance genes of *T. aestivum*	Resistance genes of related species
1A	*Pm3a*, *Pm3b*, *Pm3c* [8, 9], *Pm3d*, *Pm3e*, *Pm3f* [10], *Pm3g* [11], *Pm3i*, *Pm3j* [12], *Pm3l* [13], *Pm3m*, *Pm3n*, *Pm3o*, *Pm3p*, *Pm3q*, *Pm3r* [14]	*Pm3h* (*T. durum*) [12], *Pm3k* (*T. dicoccum*) [13], *Pm25* (*T. boeoticum*) [15], *Pm17* (*S. cereale*) [16, 17]
2A	*Pm4c* (*Pm23*) [18], *Pm65* [19]	*Pm50* (*T. dicoccum*) [20], *Pm4a* (*T. dicoccum*), *Pm4b* (*T. persicum*) [21], *Pm4d* (*T. monococcum*) [22]
3A	*Pm44* [23]	–
4A	*Pm61* [24]	*Pm16* (*T. dicoccoides*) [25]
5A	–	*Pm55* (*D. villosum*) [26]
6A	–	*Pm56* (*S. cereale*) [27], *Pm21* (*Pm31*) (*D. villosum*) [28]
7A	*Pm1a* [29], *Pm1e* (*Pm22*) [30], *Pm9* [31], *Pm59* [32]	*Pm1b*, *Pm1c* (*Pm18*) (*T. monococcum*), *Pm1d* (*T. spelta*) [29], *Pm37* (*T. timopheevii*) [33], *Pm60* (*T. urartu*) [34]
1B	*Pm28* [35], *Pm39* [36]	*Pm32* (*Ae. speltoides*) [37], *Pm8* (*S. cereale*) [38]
2B	*Pm52* [39], *Pm63* [40]	*Pm6* (*T. timopheevii*) [41], *Pm26* (*T. dicoccoides*) [42], *Pm33* (*T. persicum*) [43], *Pm42* (*T. dicoccoides*) [44], *Pm49* (*T. dicoccum*) [45], *Pm64* (*T. dicoccoides*) [46], *Pm57* (*Ae. searsii*) [47], *Pm51* (*Th. ponticum*) [48], *Pm62* (*D. villosum*) [49]
3B	–	*Pm41* (*T. dicoccoides*) [50], *Pm13* (*Ae. longissima*) [51]
4B	–	*Pm7* (*S. cereale*) [52]
5B	–	*Pm30* (*T. dicoccoides*) [53], *Pm36* (*T. dicoccoides*) [54], *Pm53* (*Ae. speltoides*) [55]
6B	*Pm11* [56], *Pm14* [57], *Pm54* [58]	*Pm27* (*T. timopheevii*) [59], *Pm12* (*Ae. speltoides*) [60], *Pm20* (*S. cereale*) [61]
7B	*Pm5b*, *Pm5d* [62], *Pm5e* [63], *Pm47* [64]	*Pm5a* (*T. dicoccum*), *Pm5c* (*T. sphaerococcum*) [62], *Pm40* (*Th. intermedium*) [65]
1D	*Pm10* [66], *Pm24a* [67, 68], *Pm24b* [69]	–
2D	–	*Pm58* (*Ae. tauschii*) [70], *Pm43* (*Th. intermedium*) [71]
4D	*Pm46* [72]	–
5D	*Pm2c* [73], *Pm48* [74, 75]	*Pm2a* (*Ae. tauschii*) [76], *Pm34* (*Ae. tauschii*) [77], *Pm35* (*Ae. tauschii*) [78], *Pm2b* (*A. cristatum*) [79]
6D	*Pm45* [80]	–
7D	*Pm15* [57], *Pm38* [81]	*Pm19* (*Ae. tauschii*) [82], *Pm29* (*Ae. ovata*) [83]

species. For example, the wide use in breeding since the 1970s of gene *Pm8* (T1BL.1RS translocation) and further growing of genetically uniform varieties on vast areas led, at the beginning of the 1990s, to all European fungus populations contained 100% clones virulent to *Pm8* [86]. A rapid loss of resistance can be explained by *Pm8* gene suppression. Experiments with transient expression demonstrated the suppression of resistance in the presence of functional and non-functional alleles of wheat gene, *Pm3*. This process occurred in epidermal cells of wheat lines carrying *Pm8* resistance gene from rye variety Pectus [87]. Unfortunately, the only reliable criterion for the identification of durable resistance is the cultivation of resistant varieties.

There are more than 100 genes known to affect barley resistance to powdery mildew, most of which are allelic variants of *Mla* and *Mlo*. These genes are found in accessions of different origin, mostly from Israel. About 30 alleles of *Mla* (chromosome 1H) [88–90] and 40 alleles of *Mlo* (chromosome 4H) are described [91]. Unfortunately, most alleles are not effective against the disease agent. The durable resistance of barley varieties is caused by *mlo11* and partially by *mlo9*. Currently, 75% of modern varieties of spring barley in Europe are protected with these genes [92].

Eleven genes that control resistance of oat varieties to *B. graminis* (DC.) E.O. Speer f. sp. *avenae* Em. Marchal during the ontogenesis period were identified [93]. Variety Jumbo is protected with a dominant gene, *Pm1*, localized on chromosome 1C [94]. *Pm3* was transferred to cultivated oat (variety Mostyn) from *Avena sterilis* L. var. *ludoviciana* and is localized on chromosome 17A [94–96]. Variety Rollo, besides *Pm3*, also possesses a second dominant resistance gene, *Pm8,* on chromosome 4C [94]. Resistance to the pathogen by introgression line Cc 6490, which is obtained with the involvement of *A. barbata,* is controlled by the *Pm4* gene localized on chromosome 18D. Resistance gene, *Pm5* (chromosome 19A), was introgressed from *A. macrostachya* [94, 97, 98]. The recessive resistance gene, *Pm6*, which was localized on chromosome 10D, was identified in variety Bruno. Breeding line APR122, with *A. eriantha* in its pedigree, is protected with dominant gene, *Pm7,* on chromosome 13A. The localization of *Pm2*, transferred into cultivated oat from *Avena hirula*, is still unknown [94]. Accessions of *A. byzantina* AVE2406 and AVE2925 carry one effective dominant resistance gene: *Pm9 (chromosome 16A)* and *Pm10* (10D), respectively [99]. Effective resistance gene, *Pm11*, was identified in accession CN113536 (*A. sterilis*) [93].

Donors of resistance genes *Pm1*, *Pm3,* and *Pm6*, which are widely used in historical breeding programs in European countries [100–102], are strongly infected by the pathogen. The highest level of resistance is provided by gene *Pm4*. Gene *Pm7* is less effective in Europe [103]. Markers of gene *Pm4* are available for marker-assisted selection [104].

Examination of extensive material of *A. sativa* indicates low-diversity of powdery mildew resistance genes [95, 100–102]. A variety Canyon from Poland was revealed probably protected by new resistance gene (genes) to the pathogen [102, 103]. The sources of resistance are seldom found among hexaploid oat species. Among 350 accessions of *A. sterilis,* only 10 appeared to be resistant [105]. Accessions CN67383 and CN113536 are the most interesting and likely possess new resistance genes [106]. Accessions of tetraploid species *A. magna* and *A. murphyi* can be effective donors of resistance to disease. All forms originate from the Mediterranean region (Morocco and Spain) [107, 108].

Oat also possesses age-specific resistance to powdery mildew. Moreover, adult plants of nine landraces and two commercial varieties were highly resistant [109].

STRUCTURAL AND FUNCTIONAL DIVERSITY OF GENES CONTROLLING CEREAL CROP RESISTANCE TO POWDERY MILDEW

Two types of protection against the pathogen are available at the cell level – external and internal. External protection is based on transmembrane pattern recognition receptors on cell surfaces that can recognize conserved pathogen-associated molecular patterns, such as lipopolysaccharides, peptidoglycans, and bacteria proteins. Primary transmembrane receptors include receptor-like kinases (RLK) and receptor-like proteins (RLP). Internal protection involves cytoplasmic receptors, most of which are coded by resistance genes, or *R*-genes, and assigned to the conserved family of proteins, NLR, characterized by the availability of nucleotide-binding sites (NBS)

and leucine-rich repeat (LRR) domains. Effector proteins are directly recognized by NLR receptors or indirectly via modification of host plant proteins associated with NLR [110–113].

In the molecular identification of *R*-genes, positional cloning and comparative and mutation genomics are used [114]. However, the number of resistance genes of cereal crops cloned and sequenced to date is still small and limited to wheat and barley.

R-genes coding NLR-type receptors are typically members of multigene families. Cluster structure in genomes and a high level of variability are typical, due to segmental duplications, recombinations, unequal crossover, point mutations, and divergent selection [115]. A series of alleles are described for *R*-genes *Pm3* [12] and *Mla* of wheat [116, 117] and barley [90], respectively.

Genes of effectors are probably characterized by higher variability in comparison with resistance genes. Such variability is demonstrated for causal agents of powdery mildew in wheat and barley. Their effectors of virulence evolve more rapidly than many other genes, allowing pathogens to overcome effects of related NLR genes [118]. Protective responses are a result of interactions among various genes, proteins, and regulatory molecules. A formal picture of these interactions in wheat was developed as a reconstructed gene network describing groups of functionally related genes involved in the development of immune responses to pathogenic fungi [119].

Currently, nine *R* genes are identified in wheat at a molecular level. These genes control wheat resistance to powdery mildew. One gene is cloned and sequenced from barley and rye. Genes *Pm2, Pm3,* and *Pm60* of wheat, *Mla* of barley, and *Pm8* of rye, respectively, code proteins assigned to the family of NLR receptors. Durable resistance is determined by genes coding for proteins with kinase activity or transport functions, introgressed from *Haynaldia villosa Pm21*, as well as loci with pleiotropic effects, including *Lr34/Yr18/Sr57/Pm38* and *Lr67/Yr46/Sr55/Pm46*. The biochemical function of MLO, a product of locus *Mlo* (a negative regulator of immune response), is still not studied [120–132] (Table 2).

Genes of loci *Lr34/Yr18/Pm38/Sr57* and *Lr67/Yr46/Pm46/Sr55* were sequenced to ensure simultaneous adult resistance to several pathogens, including leaf, yellow, and stem rust and powdery mildew. Multiple resistance to fungi in carriers of genes *Lr34/Yr18/Pm38/Sr57* and necrosis of leaf tips (marker *Ltn1*) are specified by the effects of gene *Lr34* localized in the short arm of chromosome 7D near locus *Xgwm295*. This gene is identical to *Yr18*, *Pm38,* and *Sr57* [124]. The product of gene *Lr34* belongs to class ABCG of the ATP-binding cassette (ABC) transporters. It includes 1401 amino acid residues. The protein possesses two cytosolic nucleotide-binding domains and two hydrophobic transmembrane domains. *Lr34* alleles from sensitive and resistant genotypes are characterized by two polymorphic sites changing the structure of one transmembrane domain [124]. Gene *Lr34* is involved in the remodeling of the cell plasmalemma, accompanied by inner-cell accumulation of phosphatidic acid and an increase in the removal of phosphatidylserine. Redistribution of phospholipids under the control of gene *Lr34* affects the composition of membrane proteins and responses to stress factors, resulting in the accumulation of neutral lipids in *Lr34*-transgenic barley plants [133].

The product of gene *Lr67* also exhibits a pleiotropic effect. This protein is a supposed hexose transporter STP13 of the H^+-monosaccharide symporter class. The product contains 514 amino acid residues, with 12 transmembrane coils, and transports glucose through the cell membrane. Proteins from resistant (*Lr67res*) and sensitive (*Lr67sus*) genotypes differ only in two amino acid residues, which are conserved in STP-like hexose transporters. Protein, Lr67sus, and related proteins coded by homoalleles function as high-affinity glucose transporters. Allele *Lr67res* exerts dominant-negative effect. Protein Lr67res interacts with the products of homoalleles producing heterodimers, resulting in a reduction of glucose digestion and slow growth of pathogenic fungi [125]. Experimental confirmation of the conserved status of resistance mechanisms determined by gene *Lr67* was shown. Resistance allele, *Lr67res*, from wheat determines the resistance of transgenic barley plants to barley leaf rust and powdery mildew. It also induces upregulation of genes connected with pathogenesis, *PR1*, *PR2*, and *PR3*. However, contrary to wheat, resistance is evident in seedlings, likely as a result of the differences in the level of expression of this gene in different genetic backgrounds [134].

Pm3 and *Pm8* genes, localized in syntenic regions of chromosomes 1AS of wheat and 1RS of rye, respectively, are orthologs. The identified products of gene candidates *Pm3b* (1415 amino acid residues) and *Pm8* (1375 amino acid residues) are characterized by significant similarity. Their protein sequences share 81% of identical amino acid residues. The most polymorphic sites are in the same leucine-rich repeats contacting with cytosol. Sequences homologs of two genes in the different genera of the tribe Triticeae are complexes of the same haplotypes. This finding indicates that the genes evolved independently following the divergence of wheat species from a common ancestor about 7.5 million years ago, yet retained a common function [122].

Locus *Mla* (*mildew resistance locus A*) that defines race-specific resistance of barley to powdery mildew is in the short arm of chromosome 1H. The gene have a series of more than 30 alleles [89]. *Mla*-coded NLR proteins are characterized by an exclusively high level of functional diversity (race specificity). They contain the coiled coil (CC), NBS, and LRR domains (CNL receptors). These genes were introgressed into the genome of cultivated barley from different sources, including wild species *H. spontaneum*. The *Mla*-specified response is characterized

Table 2

List of sequenced genes for powdery mildew resistance of cereal crops

Gene	Protein	Species, genotype	References
Wheat			
Pm2	NLR	*T. aestivum*, line CI12632/8 (from variety Chancellor)	[120]
Pm3b	CNL	*T. aestivum*, landrace Chul, line Chul/8*Chancellor	[121, 122]
Pm21a (*Stpk-V*)	Serine/threonine kinase	*H. villosa*, amphidiploid *T. durum* – *H. villosa*, *T. aestivum-H. villosa* T6VS.6AL translocation line, *T. aestivum* lines with additional *H. villosa* chromosomes	[123]
Lr34/Yr18/Sr57/Pm38	ABC-transporter	*T. aestivum*, lines Thatcher *Lr34*, Avocet *Lr34*, Forno, Chinese Spring	[124]
Lr67/Yr46/Sr55/Pm46	Hexose transporter	*T. aestivum*, line Thatcher *Lr67*	[125]
Pm60	NLR	*T. urartu*, accessions from Lebanon and Turkey	[34]
TmMla1	NLR	*T. monococcum*, line DV92	[126]
TaMla2	CNL	*T. aestivum*, line TAM104R with translocation 6BS.6RL	[127]
TaMla3	CNL	*T. aestivum*, line TAM104R with translocation BS.6RL	[127]
Rye			
Pm8	CNL	*S. cereale*, line from variety Petkus	[122]
Barley			
Mla	CNL	*H. vulgare*, variety Morex	[128]
Mlo (wild type)	Calmodulin-binding protein	*H. vulgare*, variety Ingrid	[129]
mlo1, *mlo3*, *mlo4*, *mlo5*, *mlo7*, *mlo8*, *mlo9*, *mlo10*, *mlo13*, *mlo17*, *mlo26*	Calmodulin-binding protein	*H. vulgare*, induced mutants of varieties Haisa, Maltera Heida, Foma, Carlsberg II, Diamant, Plena	[129]
mlo12, *mlo16*, *mlo27*, *mlo28*, *mlo29*, *mlo30*	Calmodulin-binding protein	*H. vulgare*, induced mutants of wild-type allele carriers and variety Sultan 5 *Mlo*	[130]
mlo11	Calmodulin-binding protein	*H. vulgare*, spontaneous mutation, wild barley accessions from Ethiopia. Lines *H. vulgare* var. *spontaneum* from Israel, Turkey and Iran	[131]
mlo11 (*cnv2*)	Calmodulin-binding protein	*H. vulgare*, spontaneous mutation, Ethiopean barley accession Eth295	[132]

by the rapid development of the hypersensitivity response [135]. *Mla* alleles are highly polymorphic. Each allele likely recognizes gene AVR_a, coding for an effector for avirulence in *B. graminis*. Transcriptome analysis of 17 isolates of *B. graminis* containing different AVR_a genes, identified variants AVR_{a1} and AVR_{a13} encoding presumptive effectors, which are recognized by immune receptors of barley coded by alleles *Mla1* and *Mla13* [115]. Several investigators reported on the structural arrangement of barley *Mla* locus. In particular, locus *Mla* from variety Morex contains a cluster of CNL-coding genes belonging to three divergent subfamilies of homologs of *R* genes (resistance gene homologs, *Rgh*). Resistance is controlled by allelic variants of subfamily *Rgh1* [89, 128, 136]. These conclusions were confirmed by examining the transcriptome of 50 accessions of *H. spontaneum* representing nine populations cultivated in the Fertile Crescent. The diversity of *Mla* transcripts was not associated with the accession origin. However, depending on the structure of two N-terminal coiled-coil signal domains able to mediate the cell death, all identified transcripts were grouped into two subfamilies, one of which included all known variants of receptor MLA that determine resistance to *B. graminis* [137].

Bioinformatic analysis resulted in the detection of 175 genes, CNL, in the barley genome attributed to three phylogenetic groups. The majority of identified clusters were localized in extra-pericentromeric areas associated with a high degree of recombination required for rapid divergence [138].

The family miR9863 of microRNA in genomes of barley and wheat are involved in the initiation of immune response by barley *Mla* gene. Four members of this family performed differential cleavage of *Mla* transcripts and suppressed synthesis of MLA1 protein in the heterologous *Nicotiana benthamiana* Domin expression system. The specificity of interactions was determined by single-nucleotide polymorphism of mature miR9863, as well as by two SNPs in miR9863-binding site sequences of *Mla*, depending on which alleles were attributed to the three groups [139].

Genes of *Mla* locus are linked with *Hor1* and *Hor2* genes controlling the synthesis of hordeins C and B, the main storage proteins of barley seeds [140]. The *Pm3* locus in wheat is bound with complex locus *Glu3/Gli1* coding for seed storage proteins, low-molecular-weight glutenin subunits, and reserve gliadins [122].

Orthologs of *Mla* gene are detected in genomes of different genera of cereal grasses that diverged millions of years ago. The genome of diploid wheat *T. monococcum* contains functional homologs of barley *Mla* gene (*TmMla1*). The amino acid sequences of proteins TmMLA1 and HvMLA1 of barley have 78% identical amino acid residues. The hybrid protein TmMLA1, in which the LRR domain is replaced with that of HvMLA1 appeared to be functional and determined resistance to a previously unknown race of *B. graminis* [126]. Hexaploid wheat contains orthologs of *Mla* genes, *Sr33* and *Sr50*, introgressed from genomes of rye and *A. tauschii*, respectively. These genes provide resistance to stem rust (*Puccinia graminis* f. sp. *tritici*) [141, 142]. Two *Triticum aestivum Mla* orthologs, *TaMla2*, and *TaMla3* were cloned and sequenced. These genes encode CNL proteins and are presented with numerous copies in the genome [127].

The nonspecific durable resistance of barley to *B. graminis* is related to the mutations of *Mlo* locus (*Mildew locus O*), which is located on the long arm of chromosome 4 [143]. The *Mlo* gene contains 12 exons. The encoded RLP protein has a molecular weight of 60 kDa and contains seven transmembrane domains and a calmodulin-binding site located in the intracellular C-terminus [129, 143]. Wild-type *Mlo* genes, are expressed in different organs, tissues, and cell types and play significant roles in the protection against premature cell death. They are also involved in the reactions to biotic and abiotic stressors. However, with infection, *Mlo*-coded proteins suppress defensive reactions to pathogen penetration through Ca^{2+}-dependent interaction with calmodulin. This action prevents damage to the epidermis and mesophyll with hydrogen peroxide. Thus, MLO prevents oxidative burst and thus suppresses cell death reaction [144, 145]. In plants homozygous for recessive alleles, MLO is absent (loss-of-function mutations), and unspecified resistance to *B. graminis* is observed. Complete resistance is observed in the presence of two other genes, *Ror1* and *Ror2* (required for *mlo* resistance) [146]. In resistant *mlo* mutants, the sites of fungus penetration show remodeling and strengthening of cell walls through

rapid oxidative crosslinking of hydroxyproline-rich glycoproteins [135]. Leaf damage is typical for *mlo* mutants. The manifestation of symptoms of premature cell death following apposition of epidermis cell walls (callose sediments on adult leaves) is observed even without the pathogen [147]. A number of restrictions from negative pleiotropic effects of the gene lead to a yield reduction (e.g., premature leaf wilting) and to *mlo* mutant sensitivity to the fungus *Ramularia collo-cygni* Sutton & Waller. The use of *mlo* alleles in the breeding ensured stable durable protection of barley against *B. graminis* in areas with a moderately humid climate [148]. Mutations in the *Mlo* gene result in the inactivation of functional domains and the appearance of terminating codons. The high frequency of intragenic recombinations is typical for *mlo* alleles. Recombinations lead to reversions and recovery of wild-type sequences [129].

To date, more than 40 recessive loss-of-function alleles have been identified at the Mlo locus. These alleles are characterized by different resistance levels, from partial (e. g., alleles *mlo12* and *mlo28* obtained with chemical mutagenesis) to complete (allele *mlo11*). Most mutations are caused by the substitution of a single amino acid residue, seldom by deletions. The phenotypic effects indicated that 12 out of 14 mutants exhibited durable resistance to powdery mildew, and two mutants exhibited reduced sensitivity by reduced binding with calmodulin [144]. Comparative sequence analysis of individual alleles revealed clustered mutations, i. e., their occurrence in the certain exons [130, 131]. Spontaneous *mlo11* mutation, detected initially in barley landrace collected in Ethiopia in 1930, confers durable resistance to all races of *B. graminis*. The mutation is widely distributed among European spring barley varieties. Haplotype *mlo11* is characterized by the availability of a complex tandem repeat of 11–12 repeated units located upstream the wild-type gene [131]. The repeated unit includes 3.5 kb of the 5'-regulatory sequence, as well as 1.1 kb fragment of coding area containing the sequences of the first five exons. Aberrant transcripts of this sequence disturb the accumulation of *Mlo* transcripts and wild-type MLO protein that probably determines resistance. Mutation that led to the appearance of allele *mlo11* likely occurred after the domestication of barley [131]. Accession Eth295 of Ethiopian barley landrace (*H. vulgare* convar. *deficiens* var. *nudideficiens*) from the collection of the Leibniz Institute of Plant Genetics and Crop Plant Research (Gatersleben, Germany) recently displayed another variant of allele *mlo11* characterized by a change in the number of repeats – *mlo11*(*cnv2*) [132]. Mutation *mlo11*(*cnv2*) confers partial resistance of seedlings and complete resistance of adult plants. The mutation exhibits no negative pleiotropic effects associated with cell wall apposition or necrosis. Also, no loss of photosynthetic activity is observed. Resistance associated with the mutation, assessed by the number of colonies and their growth rate, was quantitative. The manifestation of resistance to fungal penetration in the carriers of standard and variant *mlo11* alleles differs on a histological level. The *mlo11*(*cnv2*) genotype in epidermal cells in contact with areas of successful fungal penetration forms appositions of cell walls and show the absence of necrosis and collapse of mesophyll cells. Differences in the repeat methylation levels between standard and variant *mlo11* alleles are correlated with the manifestation of resistance. The allelic variant of *mlo11*(*cnv2*) probably originated through natural selection from an ancestral variant of *mlo11* as a result of recombination between repeated elements and the 3'-terminal of an adjacent area containing a Stowaway-like transposon [132].

Sequences of polymorphic *mlo* alleles were the basis for the development of molecular markers [129, 131] which are successfully used for the screening of breeding material [147] and for searches for the carriers of mutant alleles among collection accessions [149, 150].

MLO genes were detected in plants and green algae. They are represented in small multigene families in higher plants, including cereal grasses and dicotyledons [148]. *HvMlo* homologs in barley were detected in syntenic regions of soft wheat and rice genomes. In soft wheat, homologs *TaMlo-A1*, *TaMlo-B1,* and *TaMlo-D1* are localized on chromosomes 4BL, 4DL, and 5AL, respectively. They encode three related proteins that are 88% identical to MLO from barley. These proteins likely originated from three initial ancestral wheat genomes. The *Mlo* ortholog in the rice genome, *OsMlo2* (linkage group 3), induced sensitivity of barley *mlo* mutants to *B. graminis* in transient expression experiments [151]. Twelve possible members of the *MLO*

family were detected in the rice genome [152]. The authors combined the metadata for expression, transcriptome, and phylogenetic analyses to determine their functions. The members of the family differ in tissue specificity and participate in various physiological functions, including the reactions of stressors. The expression of *OsMLO3* by infection with rice blast causal agent (*Magnaporthe oryzae* (T.T. Hebert) M.E. Barr) indicated the participation of the gene in protective reactions [152].

Genome of the model species *Brachypodium distachyon* (L.) P. Beauv. (purple false broom) contains 11 conserved *BdMLO* genes distributed on five chromosomes. *BdMLO* genes code for seven conserved transmembrane domains and calmodulin-binding sites. One gene is a probable candidate for resistance to powdery mildew [153]. The number of *MLO* homologs in the genomes of other plant species varies from 12 to 19 [154]. *MLO* genes of both monocotyledonous and dicotyledonous plants are characterized by several specific features apparently resulting from negative selection. At the same time, the results of the heterologous complementation experiments (expression of sensitivity alleles of one species in the resistant genotype of another) indicate the availability of conserved functional features that interact with powdery mildew [154].

Various approaches to obtaining new variants of *mlo* were discussed, including suppression of the wild-type *Mlo* allele by RNA interference, using methods without transgenesis (TILLING), and using genome editing systems, TALEN, and CRISP/CAS9) [148]. Practical implementation of the TILLING technology for the modification of homologs *TaMlo-A1*, *TaMlo-B1,* and *TaMlo-D1* of soft wheat variety Cadenza is reported by (Acevedo-Garcia et al. [155]). The authors obtained 16 missense mutations, each caused by the single amino acid substitutions. Lines developed based on triple and, in some cases, double mutants were characterized by resistance to *B. graminis* and did not exhibit negative pleiotropic effects of recessive *mlo* alleles.

The effectiveness of induced mutations was dependent on their position in *Mlo* genes; mutations in the second and third cytoplasmic loops of the membrane protein exhibited the highest effect [156]. Ingvardsen et al. [157] reported differences in the effectiveness of induced mutations in homologous genes. Using the TILLING technology, the authors obtained a series of mutations in homeologs *Mlo-A1* and *Mlo-B1* of the durum wheat variety Kronos. The effects of mutations in *Mlo-B1,* in general, were more dramatic compared with those of the mutations in *Mlo-A1;* however, the best result was observed in genotypes carrying mutations in both *Mlo-A1* and *Mlo-B1*.

Resistance to powdery mildew can be increased by the mutations in other genes involved in protective functions. Using the CRISP/CAS9 technology, Zhang et al. [158] obtained mutations of homeologs of the conserved *Taedr1* gene (enhanced disease resistance) localized on chromosomes 1AS, 1BL, and 1BL of soft wheat. *TaEDR1* is a negative regulator of resistance, and triple *Taedr1* mutants were resistant to powdery mildew.

Only two of more than 40 known mutant *mlo* alleles, spontaneous *mlo11* and induced *mlo9,* were used in barley breeding in the 1970s and the beginning of the 1980s. Currently, immunity of more than half of the varieties of spring barley cultivated in the Central Europe is conditioned by *mlo* alleles [91].

The information accumulated to date for the genetic diversity of cereal crop resistance to *B. graminis* confirms the "laws of natural immunity of plants to infectious diseases" formulated by Vavilov [159]. The number of identified primary resistance genes to powdery mildew is large, and the list is constantly expanding. Genes determining race-specific resistance of cereal crops exhibit a common type of structural organization characteristic of NLR receptors for immune response. This structure helps ensure co-evolution with parasite genes. This finding is consistent with the first law, which assumes that higher parasite specialization is correlated with a higher probability of finding resistant forms.

"The second law for finding immune varieties and species among the cultivated plant is the availability or absence of strong genetic divergence. The greatest contrasts in terms of resistance are cytogenetically strongly differentiated in different species" [159]. The data discussed in the article support this statement. Cultivated species of the genus *Triticum* display complex genomic composition and are characterized by a high level of polymorphism. To the contrary, cultivated barley is characterized by a rather low level of

genetic diversity. Large numbers of powdery mildew resistance genes on different chromosomes (mainly genomes A and B) are identified in wheat, but only two loci (*Mla* and *Mlo*) with large numbers of alleles are found in barley.

Immunity responses reflect plant ecological type, and significant differences in resistance are detected in contrasting environmental conditions. N.I. Vavilov considered that immunity is developed only under conditions that promote the development of infection (the third law) [159]. According to Wolfe and McDermott [160], the probable origin of *B. graminis* f. sp. *hordei* is centered in the Mediterranean region and Near East. All allelic variants of *Mla* and *Mlo* genes that determine race-specific and durable resistance to *B. graminis* are detected only in accessions from East Africa and Near East.

Group or complex resistance is widespread in nature (the fourth law) [159]. Data on structure and function of genes associated with resistance enable the understanding of resistance mechanisms. Age resistance to several pathogens – powdery mildew and leaf, yellow, and stem rust in wheat genotypes carrying clusters of genes *Lr34/Yr18/Pm38/Sr57* and *Lr67/Yr46/Pm46/Sr55* – is specified by the pleiotropic effects of one gene coding for transport protein, ABC-transporter (*Lr34*) and hexose transporter (*Lr67*).

These regularities prompted Vavilov to formulate the fifth and sixth laws. "Knowing the evolutionary history of the cultivated plant, <...> one can foresee, to a significant extent, the location of immune forms interesting for a breeder, and ecological and geographical associations for the detection of immunity are common and inherent for plants assigned to different genera and even families" [159]. These regularities are consistent with the information in this review. For example, the most resistant forms of oat (genus *Avena*) and barley (genus *Hordeum*) came from the Mediterranean region and North Africa [107, 108].

CONCLUSION

Cereal crops are characterized by wide genetic diversity for resistance to powdery mildew. The specificity of the parasite-host relationship causes rapid loss of the effectiveness of many genes, leading to a constant search for new resistance genes. The genetic pool of cultivated species is rather poor in resistant forms. Recent introgression of resistance from wild relatives has become important for replenishing the stock of effective genes. Thus, among 92 identified alleles of resistance to *B. graminis* in soft wheat, 48 were transferred from the genomes of wild relatives, including *Aegilops* sp., *Secale* sp., *Dasypyrum* (*Haynaldia* sp.), *Thinopyrum* sp., and *Agropyron*. New sources of resistance can be obtained through traditional methods of mutagenesis (e.g., numerous *mlo* alleles of barley), and by targeted changes of gene sequences using the TILLING and CRISP/CAS9 technologies. Information on the structural and functional organization of resistance genes and molecular resistance mechanisms is still limited and available only for wheat and barley. Soft wheat genes, *Pm2*, *Pm3*, and *TmMla1*; wild einkorn *T. urartu* gene, *Pm60;* rye gene, *Pm8*; *and* barley gene, *Mla,* are identified at the molecular level to code for proteins NLR and CLR. Barley *Mlo* encodes receptor-like proteins, and wheat genes, *Lr34*, *Lr67*, and *Pm21,* code for transport proteins and receptor-like kinases.

This research was performed under financial support of the Russian Foundation for Basic Research (Project No. 18-016-00075) and within the framework of state task (Budgetary Project No. 0662-2019-0006).

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