

# Genetic Protection of Soft Wheat

Subjects: **Pathology**

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The southern Ural is consistently among the 10 best regions in Russia for agricultural production, including wheat. Breeding in the Chelyabinsk Research Institute of Agriculture aims to develop wheat cultivars genetically protected from the main diseases (leaf and stem rust, septoria leaf blotch and tan spot). The genes for resistance to leaf rust, Lr1, Lr9, Lr10 and Lr26/Sr31, alone or in combination, are widespread in cultivars grown in the southern Ural.

f. sp.

resistance

## 1. Introduction

Despite the contrasting climatic conditions, in the 2010s, the Chelyabinsk was consistently among the 10 best regions in Russia for agricultural production. The total area sown to various agricultural crops represents 2–3% of the total cropping area of Russia. In the region, soft and durum wheat, barley, oats, rye, triticale and millet are grown <sup>[1]</sup>. The largest proportion of cropping in the region is soft wheat (mostly spring wheat), comprising 40–50% of the area sown. In terms of area sown to soft wheat, the Chelyabinsk region is in the top 10 regions of Russia <sup>[2]</sup>.

Until recently, leaf rust was the main contributor to a damaging pathogenic complex in the region. In some conducive years, the yield losses in wheat were more than 35% <sup>[3]</sup>. From mid-2010, the severity of stem rust and leaf blotches (septoria leaf blotch and tan spot) outbreaks began to increase.

Breeding of soft spring wheat at ChRIA commenced in 1937 and has two methodological periods. The first period (1937–1960) was characterized by the use of mainly local wheat cultivars and their hybridization. During this period, the cv. Iskra, Vesna and others were developed. These local cultivars of soft spring wheat had drought tolerance, but lacked the economically important traits of resistance to lodging and diseases due to their long stems.

Breeding for resistance to leaf rust has a long history in the southern Ural, and it has been based on the strategy of producing cultivars with vertical resistance. The use of nonspecific disease resistance has been limited as it has a negative association with drought tolerance. In the mid-2010s, ChRIA, in conjunction with standard breeding for resistance to leaf rust, began using molecular markers and marker assistance selection (MAS).

## 2. Cultivars and Promising Lines of Soft Spring Wheat Produced in ChRIA

Since 1937, ChRIA breeders have produced and introduced to the region 40 cultivars, of which 17 are included in the State Register of Breeding Achievements (SRBA) as approved for use ( **Table 1** ). The widely sown cultivars in southern Ural and Western Siberia at different times were cv. Iskra, Vesna, Uralskaya 52, Rossiyanika, Erythrospermum 59, Niva 2, Duet and Chelyaba 75. The ecological optimums for Chelyabinsk cultivars have now shifted towards more efficient use of the moisture resource due to increased resistance to lodging, leaf rust, EMSD and other stresses.

**Table 1.** Cultivars of the Chelyabinsk Research Institute of Agriculture included in the State Register of Breeding Achievements (SRBA) and promising cultivars progressed to State Cultivar Testing (SCT).

Cultivar	Pedigree	Year of Involvement in the SRBA/ Transfer to SCT	Ripening Time	Grain Quality	Resistance Genes
Iskra	Milturum 321 × Kitchener	1949	late-ripe	weak	
Vesna	-	1961	mid-ripe	weak	
Ural'skaya 52	Cesium 111 × Lutescens 324	1974	mid-ripe	strong	
Rossiyanika	Saunders × Svenno	1981	mid-ripe	strong	
Uralochka	Svenno × (Lee × Kenya Farmer)	1987	mid-ripe	strong	
Eritrospermum 59	Chayka × Irtyskanka 10	1994	mid-ripe	strong	<i>Lr10</i>
Izumrudnaya	Waldron × Ural'skaya 52	1996	mid-ripe	filler	<i>Lr26/Sr31/Pm8/Yr9</i>
Niva 2	Ps 360/76 × Irtyskanka 10	1997	mid-ripe	strong	
Duet	Eritrospermum 59 × (Tselinnaya 20 × ANK-02)	2003	mid-ripe	valuable	<i>Lr9 Lr10</i>
Chelyaba 2	{(Tezpishar × Irtyskanka 10) × Irtyskanka 10} × Tselinnaya 20 × ANK-102)	2005	mid-early	valuable	<i>Lr9 Lr10</i>

Cultivar	Pedigree	Year of Involvement in the SRBA/ Transfer to SCT	Ripening Time	Grain Quality	Resistance Genes
Pamyati Ryuba	Tertsiya × Eritrosperrum 19542	2006	mid-ripe	valuable	<i>Lr9 Lr10</i>
Chelyaba yubileynaya	Eritrosperrum 59 × Tertsiya	2010	mid-late	filler	<i>Lr9 Lr10</i>
Chelyaba stepnaya	Eritrosperrum 59 × Tertsiya	2011	mid-early	valuable	<i>Lr9 Lr10</i>
Chelyaba 75	Chernyava 13 × Eritrosperrum 21338	2012	mid-ripe	valuable	<i>Lr1 Lr10 LrSp</i>
Ural'skaya kukushka	Lutescens 4 × Tulunskaya10 × Lutescens 22178	2016	mid-early	filler	
Chelyaba rannyaya	Chelyaba 2 × ANK-104	2016	early-ripe	filler	<i>Lr9 Lr10</i>
Silach	Lutescens 210/99-10 × Eritrosperrum 23090	2020	mid-late	filler	<i>Lr10 Lr9 Lr26/Sr31/Pm8/Yr9</i>
Chelyaba 80	Cuckoo line 210 × Rossiyanika × Novosibirskaya 15	SCT 2017	mid-late	valuable	<i>LrSp</i>
Il'menskaya 2	Chelyaba 75 × (Chelyaba 2 × Fori 7)	SCT 2018	mid-early	valuable	<i>LrSp</i>
Odintsovskaya	Chelyaba 75 × ANK-17B	SCT 2018	early-ripe	valuable	<i>LrSp</i>
Chelyabinka	Vatan × Duet	SCT 2021	mid-ripe	valuable	<i>Lr9 Lr26</i>

Most of the cultivars grown in the southern Ural before the 2000s were characterized by a high level of susceptibility to leaf rust, and many of them are no longer grown commercially, with the exception of cv. Erythrosperrum 59, given that it is equivalent to many modern cultivars in grain quality and resistance to hydrological and hydrothermal stresses. Cultivar Erythrosperrum 59 is simultaneously resistant to drought and excessive moisture, has high resistant to lodging and is not subject to EMSD. It is also tolerant to leaf rust, but susceptible to stem rust, septoria leaf blotch and other leaf spots.

In the 2000s, to expand genetic diversity of resistance to leaf rust, alien donors began to be widely used by ChRIA. One of them was a cuckoo-type line with some *Aegilops speltoides* genetic background. These lines were developed at the N.I. Vavilov All-Russian Institute of Plant Genetic Resources from crosses and backcrosses with soft wheat of the complex-resistant amphidiploid *Triticum dicoccum* × *Ae. speltoides*, and they carried a block of effective Lr and Sr genes linked to the gametocidal gene ( Gc ). The Gc -gene expression leads to the elimination of gametes carrying the recessive gc allele in the heterozygous sporophyte tissues. Recessive alleles linked to it ( lr and sr ) are also eliminated, and as a result, F1 plants are semi-sterile, and in F2 they are homozygous for resistance [4]. The Gc -gene was named cuckoo. Interest in Lr and Sr gene identification in cuckoo-type lines considered for selection is associated with their linked inheritance of resistance to two damaging pathogens and with a genetic mechanism of selection for resistance ( Gc -gene). The presence of the Gc -gene in the block excludes the use of established hybridological analysis; therefore, molecular screening is an alternative method for identifying these genes. Ibragimova et al. [5] identified the gametocidal gene of the cuckoo-type lines as Gc1 and determined its localization on chromosome 2B.

In the 2000s, ChRIA began to use new donors and lines in breeding with alien genes Lr22a Lr24 , Lr21 , Lr25 , Lr38 , Lr47 (Pavon), Lr48 (CSP44), Lr49 (VL404) and others. A new breeding material of soft spring wheat has been created, combining disease resistance with adaptability to adverse environmental factors, high yield and grain quality [6][7]. Molecular analysis showed a high genetic diversity of all lines in terms of resistance to leaf and stem rusts. Many of the breeding lines have a complexed resistance to the foliar diseases (leaf and stem rust, powdery mildew and blotches) and carry resistance genes previously unused in Russian wheat cultivars (e.g., Lr21 and Lr24 ). More important examples of them are presented in **Table 2** .

**Table 2.** Reaction of breeding lines and cultivars produced at the Chelyabinsk Research Institute of Agriculture (ChRIA) to foliar diseases in seedling and adult plant stages (2019–2020).

Wheat Line/ Cultivar	Identified Resistance Genes	Reaction Type to Foliar Wheat Pathogens at the Seedling Stage									Disease Severity in the Field (%)			
		<i>Puccinia triticina</i>		<i>Puccinia graminis</i>	<i>Parastagonospora nodorum</i>	<i>Parastagonospora avenae</i> f. sp. <i>tritici</i>	<i>Pyrenophora tritici-repentis</i>			Stem Rust	Leaf Rust	Septoria Leaf Blotch	Tan Spot	
		PtK1	PtK2	PtK3	Pg1	Pn	Pa	Ptr ToxA+	Ptr ToxA-					Ptr ToxB+
Lut. 26534		3	0–1	3	3–4	5	3–4	2–3/2–3	2–3/2–3	1–2/1–2	1–5 MS	0	5	1
Er. 26596	<i>Lr10 LrSp/SrSp</i> <i>Lr34/Yr18/Sr57/Pm38</i>	0	0	0	1–2	2–3	2–3	2–3/2–3	2–3/2–3	2–3/2–3	01 MR	0	5	1
Lut. 26708	<i>Lr3 Lr10 Lr6Agi2</i>	0–1	0	0–1	0–1	3–4	3	3/3–4	2–3/2–3	2–3/2–3	0	0	0	0

Wheat Line/ Cultivar	Identified Resistance Genes	Reaction Type to Foliar Wheat Pathogens at the Seedling Stage										Disease Severity in the Field (%)			
		Puccinia triticina			Puccinia graminis	Parastagonospora nodorum	Parastagonospora avenae f. sp. tritici	Pyrenophora tritici-repentis			Stem Rust	Leaf Rust	Septoria Leaf Blotch	Tan Spot	
		PtK1	PtK2	PtK3	Pg1	Pn	Pa	Ptr ToxA+	Ptr ToxA-	Ptr ToxB+					
Lut. 26720	Lr1 Lr3 Lr10 Tsn1	3	3	3	0–1	5		2– 3/2– 3	2/2	1/1	0	0	5	0	
Er. 26725	Lr24/Sr24	0	0	0	0	5	4	1– 2/1– 2	3/3	1– 2/1	0	0	5	10	
Ferr. 26727	Lr10 Lr24/Sr24	0	0	0	0–1	5	4	1– 2/1– 2	1– 2/1– 2	1– 2/1– 2	0	0	15	1	
Er. 26762	Lr26/Sr31/Pm8/Yr9 Sr35	0– 1	0	0	0–1	5	3	1– 2/1– 2	1– 2/1– 2	1– 2/1– 2	0	0	1	1	
Er. 26775	Lr10	1– 2	3	3	0	3–4	3	2– 3/2– 3	2– 3/2– 3	2– 3/2– 3	0	0	5	0	
M. 26690	Lr1 Lr3 Lr10	0	0– 1	0– 1	0	3–4	1–2	1/1	1/1	1/1	0	0	20	1	
Ferr. 26757	Lr1 Lr3 Lr10 LrSp/SrSp	0	0	0	1–2	5	3	2– 3/2– 3	2– 3/2– 3	2– 3/2– 3	0	0	10	10	
Ferr. 26774	Lr10 Lr21 Lr34/Yr18/Sr57/Pm38	0– 1	0	0	0	2–3	1–2	2– 3/2– 3	2– 3/2– 3	1– 2/2	0	0	5	0	
Chelyaba 75	Lr1 Lr10 LrSp/SrSp	0	0	0	1–2	3–4	2–3	2– 3/2– 3	2– 3/2– 3	2– 3/2– 3	1 MR	0	10	1	
Chelyaba yubileynaya	Lr9 Lr10	3	0	0	3	4	3	3/3	3/3	2– 3/2– 3	20 S	10– 20 S	5	5	
Er. 59		3– 4	3– 4	3– 4	3–4	5	3	3/3	3/3	3/3	30 S	70 S	20	10	

Ferr., Ferrugineum; Lut., Lutescens; Er., EritrospERMum; M, Miltrum. MS, moderately susceptible; MR, moderately resistant; S, susceptible. Reaction types for rust were 0–2 for resistance and 3–4 for susceptibility, with MR type 2,

MS types 2–3 and S type 4. Avirulence of test-isolates: *P. tritici* PtK1 for Lr 19, 23, 24, 26, 28, 29 and 44, and virulence for Lr 1, 2a, 2b, 2c, 3a, 3bg, 3ka, 9, 10, 11, 14a, 14b, 15, 16, 17, 18, 20 and 30. PtK2 for Lr 9, 11, 16, 23, 24, 26, 28 and 29, and Lr 1, 2a, 2b, 2c, 3a, 3bg, 3ka, 10, 14a, 14b, 15, 17, 18, 19, 20, 30 and 44. PtK3 or Lr 9, 2a, 15, 16, 19, 20, 23, 24, 26, 28 and 29, and Lr 1, 2b, 2c, 3a, 3bg, 3ka, 10, 11, 14a, 14b, 17, 18, 30 and 44. *P. graminis* Pg for Sr 24, 24 + 31, 24 + 36, 31, 36, and Sr: 5, 6, 7b, 8a, 9a, 9b, 9g, 9e, 10, 11, 17, 21, 30, 9d and Tmp. *P. tritici-repentis*: PtrToxA+/-, producing/not producing toxin ToxA; PtrToxB+, producing toxin ToxB. *P. nodorum*: Pn, isolates from northwestern Russia; *P. avenae* f. sp. *tritici*: Pa2 isolate from Chelyabinsk. -/-, reaction necrosis/chlorosis. *P. tritici-repentis* and *Para-stagonospora* sp. genotypes with reaction types 0–2 are resistant, with 3 are moderately susceptible and with 4–5 are susceptible. The GS11 stages of screening for seeding based on Zadoks growth scale and GS 61–85 for adult stages.

### 3. Variability of Pathogens' Population Structure in Response to Genetically Protected Cultivar Production

Widespread adoption of genetically protected wheat cultivars can increase variability in pathogen populations. Resistance of cultivars to one pathogen produces a favorable niche for the development of another pathogen (in the absence of complex resistance). This can lead to a significant shift in the composition of pathogen populations. For successful disease genetic management, it is necessary to control the variability in pathogen populations and the effectiveness of resistance genes.

**Tan spot:** The recent increase in the importance of tan spot is due partly to reduced or zero-tillage practices that retain stubble on the soil surface, shorter crop rotations or continuous wheat cultivation. The tan spot causal agent, *Pyrenophora tritici-repentis*, is a necrotrophic fungus that produces host-specific toxins. The pathogen has the ToxA gene, which determines the production of necrosis inducing toxin PtrToxA in wheat cultivars having the avirulence gene Tsn1. In addition to necrosis on wheat leaves, chlorosis caused by toxins PtrToxB (race 5) and PtrToxC (race 3) develops. However, the main critical virulence factor is PtrToxA [8]. It has been shown that races producing exotoxin ToxA are potentially damaging (race 1, ToxA + ToxC; race 2, ToxA; race 7, ToxA + ToxB; race 8, ToxA + ToxB + ToxC) [9]. However, in our studies, wheat genotypes at the seedling stage mostly had similar reactions to Ptr isolates producing different toxins (Table 2).

differential wheat lines/cultivars (lines 6B365 and line 6B662, and cv. Glenlea): 6B365 has Tsc1, the gene controlling sensitivity to PtrToxC (races 1, 3, 6 and 8), 6B662 has Tsc2, the gene controlling sensitivity to Ptr ToxB (races 5, 6, 7 and 8), and cv. Glenlea has Tsn1, the gene controlling sensitivity to PtrToxA (races 1, 2, 7 and 8) [10].

In 2017, wheat leaves with spots were collected from samples studied in Chelyabinsk under the Kazakhstan-Siberia Network for the Spring Wheat Improvement program [11]. Nineteen monoconidial isolates of *P. tritici-repentis* were assessed and five races were determined (Table 3). The highest proportion was in races producing Ptr ToxA toxin (86%).

**Table 3.** Frequency of *Pyrenophora tritici-repentis* races in Chelyabinsk region on spring soft wheat (%).

Race	Ptr Toxins	2017	2019	2020
1	<i>PtrToxA, ToxC</i>	26	2	32
2	<i>ToxA</i>	53	8	11
3	<i>ToxC</i>	0	3	8
4	<i>No toxins</i>	5	29	27
5	<i>PtrToxB</i>	0	14	0
6	<i>ToxB + Tox</i>	0	12	8
7	<i>ToxA + ToxB</i>	5	16	0
8	<i>ToxA + ToxB + ToxC</i>	7	16	11
Number of isolates		19	86	37

## 4. Conclusions

This article presented an analysis of wheat genetic variability in the southern Ural, Russia, in terms of population compositions of their obligate and hemibiotrophic pathogen complexes. This helps in understanding plant reactions when they are exposed to multiple pathogens with differing mechanisms of infection and pathogenicity under conducive conditions.

The analysis of the genetic diversity of wheat cultivars grown in the southern Ural, and the pathogenic complex present, indicate that pathogens continuously evolve into novel virulent races. Since leaf rust is the most damaging pathogen in the southern Ural, breeders need to develop cultivars resistant to this disease. The importance of stem rust and leaf blotches increased in the 2010s, when cultivars with the Lr9 gene began to be widely grown in the region. These cultivars were resistant to leaf rust but susceptible to stem rust and leaf blotches. Thus, cultivar resistance to leaf rust provides an opportunity for colonization of leaves by other pathogens. After 10 years of commercial wheat farming, the Lr9 gene has lost effectiveness. However, in the 2010s, the ranged of commercial wheat cultivars grown in the region was expanded to include cultivars having other race-specific resistance genes. The common production of moderately susceptible or only moderately resistant wheat cultivars in farming in the southern Ural, along with changes in agricultural practices, has led to pathogen build-up and providing abundant inoculum to initiate disease cycles, and provided favorable conditions for the leaf blotches to increase in their importance. In addition to these factors, the influence of global climate change favoring disease development in the region cannot be excluded. Knowledge of genetic variation in a pathogen population underpins breeding for disease resistance. Long-term monitoring of the leaf rust pathogen populations in the southern Ural has not revealed significant changes in the pathogen virulence since the 2010s. In 2019–2020, there was a moderate decrease in the frequency of pathogen virulence to a number of Lr genes. This indicates some stability in the genetic management of wheat in the region for this pathogen. However, significant variation in susceptibility to P.

tritici-repentis and *Parastagonospora* races was found; in particular, potentially damaging races of *P. tritici-repentis* producing the exotoxin ToxA were found.

Microevolutionary changes in populations of the tan spot pathogen increase genetic diversity, allowing expansion into new areas and increasing virulence in comparison to previous populations [12]. The southern Ural is an area where *P. tritici-repentis* has appeared recently. Pathogen populations adjust to their environment over time, but the dynamics of emerging pathogens can be difficult to predict. According to Mikhailova et al. [9], the virulence structure is an indicator of population age. Older populations have lower virulence, as natural selection operates against excess virulence. The *Tsn1* gene is not present in most wheat cultivars grown in the southern Ural, but some ChRIA wheat genotypes were susceptible to race PtrToxA.

Under co-infection conditions, pathogens are thought to exploit host-limited resources more efficiently, with natural selection favoring the coexistence of pathogens that are less damaging to their hosts [13][14]. Early during co-infection, the more virulent pathogen may quickly dominate. However, similarly virulent pathogens can coexist and share their hosts, mainly due to conditions favoring the occurrence of multiple pathogens [15]. An unresolved question is how changes in natural (e.g., climate) and man-made (e.g., new cultivars with polygenic or major gene resistances) conditions alter coexistence in the long term. Changes to conditions may favor one pathogen over another, potentially leading to epidemics of more aggressive pathogens [16]. Therefore, to make significant progress in wheat disease management, research efforts need to include field evaluations encompassing the range of environmental conditions and multiple-pathogen infections.

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