How to cite: Genievskaya, Y., Turuspekov, Y. (2026). Genetic resistance to major fungal diseases of barley in Kazakhstan: advances, challenges, and breeding perspectives. *Eurasian Agrotechnical Journal*, 2(130), 28-45. DOI: https://doi.org/10.51452/eaj.2026.2(130).2208.

UDC 633.256 **Review article**

Genetic resistance to major fungal diseases of barley in Kazakhstan: advances, challenges, and breeding perspectives

Yuliya A. Genievskaya, Yerlan K. Turuspekov

Institute of Plant Biology and Biotechnology, Almaty, Kazakhstan

Corresponding author: Yerlan K. Turuspekov: yerlant@yahoo.com
Co-author: (1: YG) julia.genievskaya@gmail.com
Received: 20 April 2026 **Accepted:** 03 June 2026 **Published:** 30 June 2026

Abstract

Background and Aim. Barley (*Hordeum vulgare L.*) is a key cereal crop in Kazakhstan, ranking second after wheat and making a substantial contribution to livestock production and food security. However, barley productivity is strongly constrained by major fungal diseases, including net blotch, barley scald, powdery mildew, and stem rust, causing yield losses of 10–40% and up to 100% under severe epidemics. This review summarizes current knowledge on the genetic basis of resistance to these diseases, with particular emphasis on resistance genes, quantitative trait loci (QTLs), and their application in barley breeding in Kazakhstan.

Materials and Methods. The review is based on the analysis of published scientific literature and recent molecular studies, including genome-wide association studies (GWAS) and SSR-marker analyses conducted on globally diverse and locally adapted barley germplasm. Particular emphasis was placed on studies performed under the agroecological conditions of Kazakhstan.

Results. Numerous resistance loci associated with major barley diseases have been identified and characterized. In Kazakhstan, GWAS identified 59 QTLs for net blotch (25 novel), 7 QTLs for powdery mildew (5 novel), and 19 QTLs for stem rust (13 novel), along with validation of known resistance genes. These studies also confirmed the presence of both globally relevant and region-specific resistance sources in local germplasm. The findings highlight the importance of combining major resistance genes with quantitative resistance loci and adult plant resistance (APR) for durable resistance.

Conclusion. Molecular tools such as GWAS, marker-assisted selection, and genomic selection offer strong potential to accelerate barley breeding for disease resistance. Future research should focus on gene pyramiding, use of diverse germplasm, and advanced approaches such as pangenomics and genome editing. These strategies will develop multi-disease resistant barley cultivars adapted to Kazakhstan conditions.

Keywords: net blotch; barley scald; powdery mildew; stem rust; QTL.

Introduction

Barley (*Hordeum vulgare L.*) is one of the most widely cultivated cereal crops worldwide, ranking fourth after wheat, maize, and rice. Global cultivation exceeds 50 mln hectares, with annual production ranging between 150 and 155 mln metric tons in recent years [1, 2]. Its broad adaptability to diverse agroecological conditions, including drought-prone, saline, and low-fertility environments, makes barley a key crop in marginal regions where other cereals often fail. Beyond its agronomic resilience, barley has substantial economic importance: approximately 70% of global production is used for animal feed, 20–25% for malting and brewing, and a smaller proportion for human consumption [3, 4]. The global barley market was valued at more than USD 20 billion in 2024, underscoring its continued relevance [5].

In Central Asia, Kazakhstan is among the major barley-producing countries, with annual production ranging from approximately 2.6 to 3.8 mln metric tons, depending on climatic conditions [1, 6]. Barley is the second most important cereal crop in the country after wheat and plays a vital role in livestock production, food security, and export markets. The primary production zones are located in northern Kazakhstan, characterized by continental climatic conditions with cold winters, hot summers, and variable precipitation. Despite its adaptability, average yields remain relatively low (1.5–1.7 tons per ha), largely due to combined abiotic stresses and biotic constraints, particularly fungal diseases.

Fungal diseases represent one of the most significant constraints to barley productivity worldwide. They reduce both yield and grain quality, affecting key parameters such as kernel size, thousand kernel weight, and malting quality. Under moderate infection, yield losses typically range from 10% to 40%, but may reach up to 100% in severe epidemics, especially when susceptible cultivars are grown under favorable environmental conditions [7, 8]. In addition, certain pathogens can adversely affect grain quality, leading to economic losses in both feed and malting industries.

Among the most economically important fungal diseases of barley are net blotch (NB), barley scald (BS), powdery mildew (PM), and stem rust (SR). These diseases are widely distributed across barley-growing regions and their epidemiology and severity are increasingly influenced by climatic variability. In Kazakhstan, NB and BS are particularly prevalent, often occurring simultaneously and contributing to cumulative yield losses [9, 10, 11]. PM and SR, although historically less dominant [12, 13, 14], remain important due to their widespread occurrence and the emergence of highly virulent pathogen populations [15].

Disease development is strongly influenced by environmental factors, including temperature, humidity, and leaf wetness duration, resulting in pronounced genotype × environment interactions [16]. This variability complicates disease management and the deployment of resistant cultivars, particularly under the heterogeneous climatic conditions of Kazakhstan.

While fungicide application and agronomic practices can provide partial disease control, these approaches are associated with increased production costs, environmental concerns, and the risk of fungicide resistance [17]. Therefore, the development of resistant cultivars remains the most sustainable and economically viable strategy. However, breeding for resistance is challenged by the quantitative nature of many resistance traits and the rapid evolution of pathogen populations [18].

Recent advances in molecular genetics, including quantitative trait locus (QTL) mapping, genome-wide association studies (GWAS), and marker-assisted selection (MAS), have significantly improved the understanding of the genetic architecture of disease resistance in barley [19]. These approaches enable the identification of resistance loci and molecular markers that can accelerate breeding programs. In Kazakhstan, the application of these tools to local germplasm has begun to reveal resistance mechanisms to major barley diseases [10, 13, 14, 20, 21], although comprehensive integration into breeding pipelines remains limited.

This review summarizes current knowledge on the genetic basis of resistance to four major fungal diseases of barley, with particular emphasis on resistance genes and QTLs identified in globally diverse germplasm. Special attention is given to insights derived from recent molecular studies, including GWAS and SSR-genotyping, with phenotypic evaluation conducted under the agroecological conditions of Kazakhstan, and their implications for the development of resistant cultivars adapted to the region.

Materials and Methods

The review was prepared as a structured analytical synthesis of current knowledge on the genetic basis of resistance in barley to major fungal diseases in Kazakhstan. A comprehensive literature search was conducted using scientific databases (Scopus, Web of Science, Google Scholar, and PubMed), covering peer-reviewed articles, reviews, and relevant studies published up to 2026. Particular emphasis was placed on recent molecular studies, including GWAS, SSR-marker analyses, and phenotypic evaluations performed on globally diverse and locally adapted barley germplasm under the agroecological conditions of Kazakhstan.

Data on resistance genes, QTLs, pathogen populations, and breeding strategies were extracted and synthesized narratively, with special attention given to studies addressing NB, BS, PM, and SR. Tables were used to summarize major resistance loci and GWAS results.

Results

Net blotch (Pyrenophora teres)

NB is one of the most economically important foliar diseases of barley worldwide. The pathogen occurs in two forms: *P. teres* f. *teres* (Ptt), causing the net form (NFNB), and *P. teres* f. *maculata (Ptm)*, responsible for the spot form (SFNB). These forms differ in symptom expression and host-pathogen interactions [22]. NFNB produces elongated necrotic lesions with characteristic net-like patterns (Figure 1), whereas SFNB results in discrete, circular lesions surrounded by chlorotic halos.

Figure 1. Photo of *P. teres* f. *teres* on barley plant in the field of Almaty region [10]

The pathogen survives between growing seasons on infected crop residues and seed, serving as the primary inoculum source. Both sexual (ascospores) and asexual (conidia) spores contribute to disease spread, with secondary infection driven mainly by conidia under favorable environmental conditions [7, 22]. Disease development is promoted by moderate temperatures and moisture, particularly periods of leaf wetness.

NB is widely distributed across major barley-growing regions, including North America, Europe, Australia, and Asia. In Kazakhstan, NB is one of the most prevalent foliar diseases, with infection severity exceeding 40% in northern regions during favorable years. Yield losses typically range from 10% to 40%, although higher losses may occur under severe epidemics [10, 11, 22]. Field evaluations have further demonstrated strong stage-dependent variation in resistance: although up to 37 genotypes exhibited resistance at the stem elongation stage; only 12 genotypes-maintained resistance at heading, indicating substantial changes in host response during plant development [11]. Moreover, the high virulence diversity observed in local Ptt populations, including isolates capable of overcoming resistance in commercial cultivars such as “Ubagan” [11], highlights the dynamic nature of host-pathogen interactions and the need for durable resistance strategies.

In addition to reducing yield, NB negatively affects grain quality by decreasing kernel size and thousand kernel weight [23].

Resistance to NB is commonly evaluated using visual infection-type scales developed for both seedling and adult plant stages. At the seedling stage, the Tekauz scale [24] is widely used to classify reactions from resistant to susceptible based on lesion size, chlorosis, and necrosis patterns. In field

evaluations, disease severity is often estimated as the percentage of infected leaf area using modified Cobb-type scales [25] or a 1–9 rating system, where lower scores indicate greater resistance.

Barley scald (*Rhynchosporium commune*)

BS, caused by R. commune (Rc), is a major foliar disease in temperate regions characterized by cool and humid conditions [26]. Symptoms include irregular lesions that develop into characteristic grayish-white centers with dark margins, often coalescing under severe infection [27] (Figure 2).

Figure 2. Photo of *R. commune* on barley plant in the field of Almaty region [10]

The pathogen survives on crop residues and volunteer plants, with disease spread primarily mediated by rain-splashed conidia. Moisture is a critical factor for infection, with optimal conditions occurring at temperatures of 10-20 °C and prolonged leaf wetness [27]. Consequently, BS is particularly prevalent in regions with frequent precipitation [26].

Globally, BS is widely distributed across Europe, North Africa, South America, and parts of Asia. Yield losses typically range from 10% to 40%, but may approach 100% under continuous barley cultivation without crop rotation [28]. In Kazakhstan, the disease is widespread across major barley-growing regions and shows considerable variation in severity depending on environmental conditions and host genotype, with field studies reporting disease development levels commonly ranging from 5% to 50% in spring barley under natural infection [29]. Also, BS negatively affects grain quality by decreasing kernel weight and uniformity [27].

BS resistance is traditionally assessed using qualitative infection-type scales that consider lesion size, sporulation intensity, and the extent of chlorosis or necrosis. Seedling evaluations commonly employ a 0–4 or 0–9 scale, while adult plant resistance is frequently quantified as the percentage of diseased leaf area under field conditions. These assessments allow differentiation between highly resistant, moderately resistant, and susceptible genotypes.

Powdery mildew (Blumeria graminis f. sp*. hordei)*

PM, caused by the obligate biotrophic fungus *B. graminis* f. sp. *hordei (Bgh)*, is a widespread disease affecting barley across diverse environments. The pathogen forms white, powdery colonies on leaf surfaces and extracts nutrients via haustoria formed within epidermal cells [30] (Figure 3).

Figure 3. Photo of *B. graminis* f. sp. *hordei* on barley plant in the field of Almaty region

Unlike many foliar pathogens, *Bgh* does not require free water for infection. It spreads through airborne conidia and is favored by moderate temperatures (15–22 °C) and high humidity. Its short life cycle and high reproductive capacity enable rapid adaptation, often resulting in the breakdown of resistance genes [31].

Yield losses associated with PM are typically moderate (5–20%), but may be higher under severe infection [32]. The disease reduces photosynthetic efficiency and biomass accumulation, ultimately affecting grain filling [33]. In Kazakhstan, PM occurs in both northern and southeastern regions, with variable severity depending on environmental conditions. Studies have revealed high genetic and phenotypic diversity of *Bgh* in southern regions, with 23 pathotypes identified among 107 isolates and considerable variation in virulence frequencies across locations, indicating strong adaptive potential of the pathogen [12]. Notably, while virulence to several *Mla* resistance genes remains low (0.9-1.8%), all tested isolates were virulent to *Mla8*, highlighting the limited effectiveness of certain resistance sources [12].

PM resistance is typically scored using infection-type scales based on the amount of fungal mycelium and sporulation on leaf surfaces. The most widely used method is a 0–4 scale, where 0 indicates the absence of visible symptoms and 4 represents abundant sporulation and high susceptibility. In field experiments, disease severity may also be recorded as the percentage of leaf area covered by mildew colonies.

Stem rust (Puccinia graminis f. sp. tritici)

SR, caused by *P. graminis* f. sp. *tritici (Pgt)*, is one of the most destructive diseases of cereals. The pathogen has a complex life cycle involving both cereal hosts and *Berberis* spp., producing multiple spore types that facilitate both sexual and asexual reproduction [34].

Infection results in characteristic reddish-brown pustules on stems, leaves, and spikes, which can disrupt vascular tissues and severely impair plant growth (Figure 4).

Figure 4 – Photo of *P. graminis* f. sp. *tritici* on barley plant in the field of Almaty region [14]

The disease is favored by warm temperatures (20–30°C) and sufficient moisture, and airborne spores enable long-distance dispersal [35]. Yield losses due to SR often exceed 50% and may reach up to 100% under severe epidemics [36]. The emergence of highly virulent races, particularly the Ug99 lineage, has increased global concern due to their ability to overcome widely deployed resistance genes. Although historically less prevalent in Kazakhstan compared to foliar diseases, SR poses an increasing risk due to climate change and pathogen migration.

Recent studies in Central Asia, including Kazakhstan, have identified variation in resistance among local cultivars and detected loci associated with adult plant resistance (APR) using molecular approaches such as GWAS [14, 20]. These findings highlight the importance of integrating genetic resistance into breeding programs to mitigate future risks.

SR resistance is traditionally evaluated using the Stakman infection-type scale, which classifies reactions from immune (0) to highly susceptible (4) based on uredinium size and the presence of chlorosis or necrosis [37]. For adult plants, disease severity is commonly estimated using the modified Cobb scale [25], which records the percentage of plant tissue covered by rust pustules and combines severity estimates with host response classes such as resistant (R), moderately resistant (MR), moderately susceptible (MS), and susceptible (S).

Genetic basis of resistance to four fungal diseases of barley in Kazakhstan

The genetic control of resistance to major barley diseases in Kazakhstan reflects a combination of well-characterized major genes and numerous QTLs identified through recent genome-wide studies. As summarized in table 1, resistance to NB, BS, PM, and SR is governed by both seedling/all-stage resistance (ASR) and APR, with clear implications for breeding strategies.

Table 1. The list of major resistance genes to net blotch, barley scald, powdery mildew, and stem rust in barley

Pathogen/disease	Locus	Chr.	Pos. (Mb)	Type of resistance	References
Pyrenophora teres / net blotch	*Rpt1*	3H	541.7 – 574.5	ASR, APR	[38, 39, 40]
	Rpt2	1H	25.7 – 78.9	ASR	
	Rpt3	2H	601.8 – 663.6	ASR, APR	
	Rpt4 / Rpt3	7H	415.1 – 596.7	ASR	
	Rpt5	6H	362.3 – 376.9	ASR	
	Rpt6	5H	2.2 – 16.6	ASR	
	Rpt7	4H	81.9 – 447.3	ASR, APR	
	Rpt8	4H	572.5 – 592.2	ASR, APR	
	Rpt9 / Spt3	7H	587.1 – 632.0	ASR, APR	
	Rpt10 / Spt2	5H	437.1 – 455.5	ASR	
	Rpt11 / Spt4	1H	2.6 – 14.7	ASR	
	SPN1	6H	30.1 – 94.9	ASR	
Rhynchosporium commune / barley scald	*Rrs1 (Rh4)*	3H	446.9	ASR	[26, 41]
	Rrs14	1H	2.8	ASR	
	Rrs17 (Rrs15 (CI8288))	2H	10.4	ASR	
	Rhy	3H	447.3	ASR	
	Rrs4	3H	523.0	ASR	
	Rrs16	4H	1.7	APR	
	Rrs18 (qC174_6)	6H	11.8	APR	
	Rrs13	6H	25.6	ASR	
	Rrs2	7H	5.2	ASR, APR	
	Rrs12	7H	16.0	ASR	
	Rrs15	7H	626.3	ASR	
Blumeria graminis f. sp. *hordei* / powdery mildew	*Mla*	1H	0.24 – 0.36	ASR	[42, 43]
	MILa	2H	NA	APR	
	Mlg, MlBo	4H	550	ASR	
	mlo	4H	589.3	ASR	
	Mlh	6H	NA	ASR	
	Mlf	7H	NA	ASR	
	Mlj	5H	NA	ASR	
	mlmr	6H	NA	APR	
	Mlt	7H	NA	ASR	
	MlSb	7H	NA	ASR	
Puccinia graminis f. sp. *tritici* / stem rust	*Rpg1*	7H	3.73– 3.74	ASR	[38, 44]
	Rpg2	2H	533.3– 551.2	APR	
	Rpg3	5H	448.6 – 519.6	APR	
	rpg4 / Rpg5	5H	562.8 – 562.9	ASR	
	rpg6	6H	0.07 – 25.5	ASR	
	Rpg7	3H	606.1 – 615.9	ASR	
	Rrr1	5H	560.9 – 561.5	ASR	
	Rrr2	7H	6.2 – 6.9	ASR	
	Rpr1	4H	145.5 – 411.4	ASR	
	Rpr2	6H	68.3 – 111.5	ASR	
	Rpr9	3H	502.4 – 503.2	ASR	

Chr. – chromosome, Pos. – position, ASR – seedling/all-stage resistance, APR – adult plant resistance, NA – information is unknown.

For NB, at least 11 major resistance loci (Rpt) and additional loci, such as SPN1, have been identified across all barley chromosomes, as reported in multiple linkage and association mapping studies [38]. Resistance to BS is controlled by a series of Rrs genes, with more than 15 loci described to date, although many exhibit race-specific effects and limited durability [26]. PM resistance represents one of the most extensively characterized systems in barley, with over 30 alleles identified at the Mla locus alone, in addition to other loci such as mlo, which confers durable, broad-spectrum resistance [42]. For SR, at least 10 major genes (Rpg) have been mapped, with Rpg1 being the most widely deployed gene in cultivated barley [38].

Recent studies based on GWAS and SSR genotyping, using globally diverse barley germplasm (accessions from the USA, Middle East, Europe, Africa, and Kazakhstan) assessed under conditions of southern and south-eastern Kazakhstan, have provided valuable insights into the genetic architecture of resistance to four major fungal diseases (Table 2).

Table 2. Number of SNP, SSRs and list of genes identified for net blotch, powdery mildew, and stem rust of barley in Kazakhstan

Pathogen/disease	Number of QTLs from GWAS	Number of presumably novel QTLs from GWAS	Number of effective SSRs	Genes overlapped with QTLs from GWAS and SSR markers	Reference
Pyrenophora teres / net blotch	59	25	3	*Rpt1, Rpt2, Rpt3, Rpt4, Rpt6, Rpt8, Rpt9, SPN1*	[10, 21]
Rhynchosporium commune / barley scald	-	-	3	*Rrs4*	[10]
Blumeria graminis f. sp. *hordei* / powdery mildew	7	5	-	*Mla*	[13]
Puccinia graminis f. sp. *tritici* / stem rust	19	13	-	*Rpg1, Rpg6*	[14]

GWAS conducted in Kazakhstan identified 59 QTLs for NB, of which 25 were considered potentially novel, highlighting the unique genetic diversity of local germplasm. Importantly, several of these QTLs co-localized with known *Rpt* genes (e.g., *Rpt1, Rpt3, Rpt6, Rpt8*), confirming their relevance under regional agro-climatic conditions [21]. Additionally, seven SSRs were used for the screening of local germplasm, revealing that markers *Bmac209, Bmag206,* and *Bmag613* confirmed their association with NB resistance in Kazakhstan [10].

Resistance to BS is primarily associated with major genes such as *Rrs1* and *Rrs2*, alongside additional loci distributed on chromosomes 1H-7H [26]. Although fewer large-scale studies have been conducted in Kazakhstan compared to NB, the presence of *Rrs4* locus suggests a complex genetic architecture requiring further validation under local environments.

In contrast, resistance to PM in Kazakhstan appears to be less genetically diverse, with only seven QTLs identified, five of which are novel [13]. These loci are primarily associated with the well-known *Mla* resistance cluster, indicating a narrower genetic base that may limit long-term durability.

For SR, 19 QTLs have been identified in Kazakhstan, including 13 novel loci, with key resistance genes such as *Rpg1* and *Rpg6* contributing to ASR [14]. The presence of APR loci further supports the importance of combining resistance types to achieve durable protection.

Overall, these findings demonstrate that the diverse barley germplasm tested in Kazakhstan harbors both globally recognized and region-specific resistance loci, emphasizing its value for breeding programs targeting multi-disease resilience under continental climatic conditions.

Discussion

Breeding strategies for disease resistance

The development of disease-resistant barley cultivars relies on the strategic combination of genetic resistance sources, informed by an understanding of pathogen variability, host–pathogen interactions, and the genetic architecture of resistance. Modern breeding approaches increasingly integrate classical selection methods with molecular tools to achieve durable, broad-spectrum resistance against multiple pathogens.

One of the fundamental strategies is the deployment of major resistance (R) genes, which typically confer high levels of race-specific resistance at the seedling stage [45]. Genes such as *Rpg1* for SR [46] and multiple *Mla* alleles for PM [47] have been widely utilized due to their strong phenotypic effects. However, the durability of such resistance is often limited, as pathogen populations can rapidly evolve virulence. For example, the emergence of virulent races of *Bgh* and *Pgt* has repeatedly led to the breakdown of single-gene resistance in barley [48, 49]. Consequently, reliance on individual R genes is considered insufficient for long-term disease management.

To overcome this limitation, breeding programs increasingly focus on quantitative resistance, often expressed as APR [45]. This form of resistance is typically controlled by multiple loci with smaller effects and provides partial but more durable protection. Studies have demonstrated that combining several minor-effect QTLs can significantly reduce disease severity and slow pathogen adaptation [31, 50]. In barley, APR has been reported for all four major diseases (Table 1), highlighting its importance for sustainable resistance breeding.

Another widely adopted approach is gene pyramiding, which involves the combination of multiple resistance genes within a single genotype. This strategy enhances both the breadth and durability of resistance by reducing the likelihood that a pathogen can simultaneously overcome all resistance factors. MAS has greatly facilitated gene pyramiding by enabling the precise tracking of resistance loci during breeding cycles. For instance, pyramiding of *Rpg1* with APR loci has been shown to improve resistance stability against diverse SR races [51].

The integration of genomic tools, particularly GWAS and genomic selection (GS), represents a major advancement in resistance breeding. GWAS enables high-resolution identification of resistance loci across diverse germplasm, while GS uses genome-wide marker data to predict breeding values and accelerate selection cycles. In barley, GS has been successfully applied to improve complex traits, including disease resistance, with prediction accuracies often exceeding 0.5 depending on trait heritability and population structure [52, 53].

Figure 5 summarizes a modern breeding framework for developing disease-resistant barley cultivars using GWAS-derived molecular markers. In this approach, diverse germplasm is first phenotyped for disease response and genotyped with genome-wide markers to identify marker–trait associations linked to resistance. Significant loci are subsequently incorporated into breeding programs through marker-assisted selection, enabling the efficient identification of individuals carrying favorable alleles during population advancement. The integration of GWAS, marker-assisted selection, and conventional breeding accelerates the pyramiding of resistance loci and facilitates the development of cultivars with durable resistance to multiple fungal pathogens.

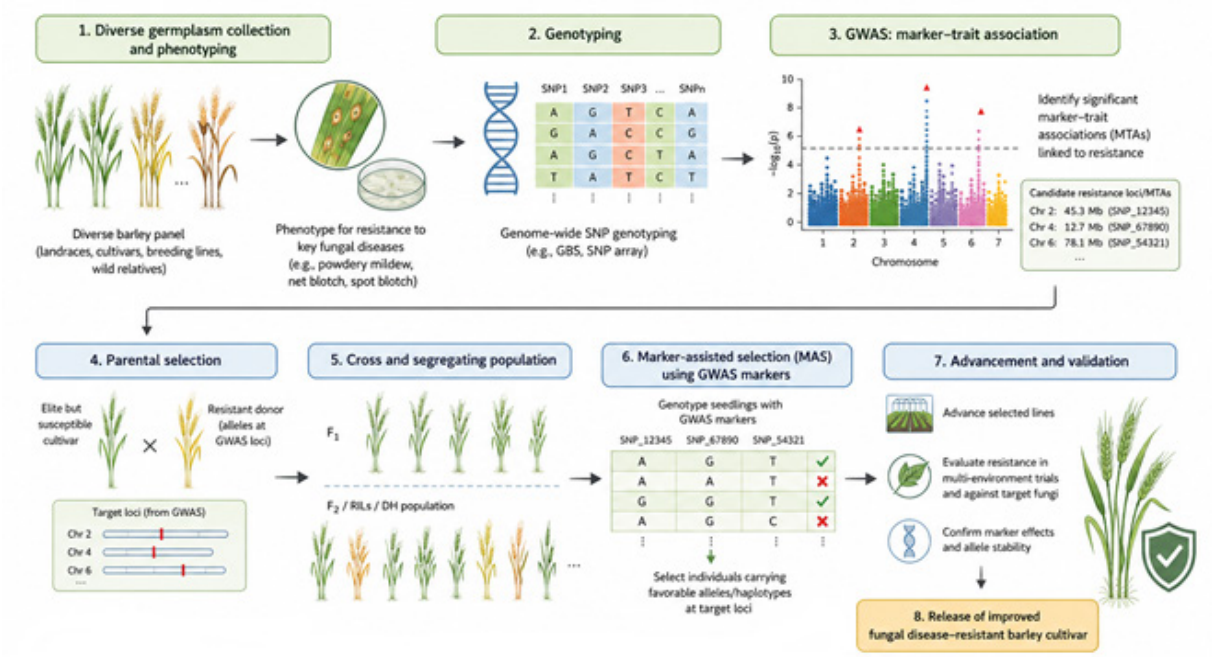

Figure 5. Schematic representation of a barley breeding pipeline for fungal disease resistance using molecular markers identified through genome-wide association studies (GWAS)

Recent examples from barley breeding programs further illustrate the effectiveness of these integrated approaches. In BS resistance, MAGIC populations combined with GWAS and high-density SNP genotyping have enabled pyramiding of loci such as Rrs1 and Rrs18 with additional QTLs, resulting in stable multi-environment resistance [54]. For stripe rust, doubled haploid lines combined with MAS have demonstrated that stacking multiple QTLs across different chromosomes provides substantially higher resistance than single loci [55]. In PM, GWAS-based identification of APR loci, together with selection for *mlo*-based slow-mildewing phenotypes, has contributed to long-term durable field resistance, illustrating the synergy between classical breeding and genomics-assisted selection [56].

In addition, host diversity and cultivar mixtures have been proposed as complementary strategies to reduce disease pressure at the field level. The use of genetically diverse cultivars can limit pathogen spread and delay the emergence of virulent races, contributing to more stable production systems. Although less commonly applied in large-scale agriculture, this approach remains relevant under low-input or environmentally sensitive conditions.

In Kazakhstan, breeding strategies for barley disease resistance are increasingly incorporating molecular approaches, although conventional phenotypic selection under field conditions remains the primary method. Recent GWAS studies on globally diverse germplasm phenotyped under Kazakhstan's agroecological conditions have identified QTLs for NB, PM, and SR [13, 14, 21], including a substantial proportion of novel loci, providing a valuable resource for marker-assisted breeding. However, the integration of these markers into routine breeding programs is still at an early stage [57]. Current efforts focus on combining locally adapted germplasm with identified resistance sources to develop cultivars with improved multi-disease resistance under continental climatic conditions.

Challenges in breeding for resistance

Despite substantial progress in identifying resistance genes and QTLs, breeding for durable disease resistance in barley remains a complex and multifaceted challenge. One of the most critical constraints is the high evolutionary potential of fungal pathogens, which enables rapid adaptation to host resistance. Pathogen populations of *Ptt, Rc, Bgh*, and *Pgt* exhibit high levels of genetic diversity driven by mutation, recombination, gene flow, and large population sizes [58]. These processes facilitate the emergence of new virulent pathotypes capable of overcoming resistance genes deployed in commercial cultivars [59].

A well-documented consequence of this evolutionary capacity is the so-called "boom-and-bust" cycle associated with major resistance genes [60]. Race-specific resistance, particularly in pathosystems governed by gene-for-gene interactions such as PM, is often rapidly rendered ineffective after widespread

deployment. For example, more than 85 race-specific resistance genes have been identified for PM in barley, yet many have lost effectiveness due to pathogen adaptation [61]. Similarly, studies emphasize that hypersensitive resistance conferred by major genes is frequently short-lived, necessitating continuous replacement of cultivars.

Another major challenge is the quantitative nature of durable resistance, which is typically controlled by multiple loci with small individual effects. While such resistance is more stable, it is also more difficult to select and validate, particularly under variable environmental conditions. Genotype × environment interactions significantly influence the expression of quantitative resistance, reducing selection accuracy and slowing breeding progress [62]. Moreover, the accumulation of favorable alleles through conventional breeding requires multiple generations and extensive phenotyping, increasing both time and cost.

The genetic uniformity of modern cultivars further exacerbates vulnerability to disease outbreaks [63]. As barley is a predominantly self-pollinated species, commercial varieties tend to be highly homogeneous, providing ideal conditions for rapid pathogen spread and adaptation. This lack of genetic diversity at the field level reduces the buffering capacity of cropping systems and increases the risk of large-scale epidemics.

In addition, fungicide resistance in pathogen populations represents an emerging constraint that indirectly affects resistance breeding. Intensive and repeated use of fungicides imposes strong selection pressure, leading to the development of resistant pathogen strains. Reduced sensitivity to major fungicide classes, including azoles, strobilurins, and SDHIs, has already been reported in several barley pathogens, complicating integrated disease management strategies [64]. This limits the effectiveness of chemical control and increases reliance on genetic resistance, thereby intensifying selection pressure on host resistance genes.

Another important limitation is the complexity of pyramiding resistance loci. Although combining multiple genes is essential for durable resistance, linkage drag, epistatic interactions, and limited availability of tightly linked markers can hinder efficient gene stacking. Furthermore, some resistance genes may be associated with undesirable agronomic traits, such as yield penalties or reduced adaptability, complicating their deployment in elite cultivars [65].

Finally, gaps in phenotyping and pathogen monitoring remain a bottleneck. Accurate assessment of resistance requires multi-environment trials and detailed characterization of pathogen populations [66]. However, limited availability of standardized phenotyping platforms and insufficient surveillance of pathogen virulence restrict the ability to match resistance genes with current pathogen races. Continuous monitoring is essential to anticipate resistance breakdown and guide breeding strategies.

In Kazakhstan, these challenges are particularly pronounced due to the combination of high environmental variability and dynamic pathogen populations. Continental climatic conditions, characterized by fluctuating temperature and moisture regimes, lead to strong year-to-year variation in disease pressure, complicating the evaluation of resistance. In addition, studies have shown that local populations of *Bgh* exhibit high intrapopulation variability and a strong capacity to overcome host resistance, while many commercial cultivars remain insufficiently resistant. Although recent GWAS studies have identified numerous resistance loci in local germplasm, their practical implementation in breeding programs is still limited. Strengthening the integration of molecular tools with long-term field validation and pathogen monitoring will be essential to overcome these constraints and develop resilient barley cultivars adapted to Kazakhstan's agroecological conditions.

Future perspectives

Future progress in breeding barley for disease resistance will largely depend on the integration of advanced genomic technologies, expanded genetic diversity, and predictive breeding frameworks. One of the most promising directions is the transition from MAS to gene-level and genome-wide approaches, enabling more precise identification and deployment of functional resistance alleles. Recent advances in barley pangenomics, supported by the sequencing of over 1,300 accessions, provide unprecedented opportunities to capture structural variation and rare alleles associated with disease resistance [67]. These resources facilitate the discovery of novel resistance mechanisms beyond classical *R genes.*

The application of genome editing technologies, particularly CRISPR/Cas systems, is expected to accelerate the development of resistant cultivars by directly modifying susceptibility genes or

enhancing quantitative resistance pathways. This approach has already demonstrated potential in barley improvement, allowing targeted manipulation of genes involved in plant–pathogen interactions [68]. In parallel, GS combined with artificial intelligence-based models is anticipated to significantly increase selection accuracy and reduce breeding cycles, especially for complex traits controlled by multiple loci.

Another key perspective is the utilization of wild relatives and landraces as sources of novel resistance alleles. Studies have shown that a substantial proportion of heritage barley varieties exhibit enhanced resistance to *Fusarium*-associated traits compared with modern cultivars, reflecting the value of genetic diversity preserved in landraces [69]. However, this resistance is often associated with undesirable agronomic traits, including increased plant height and delayed maturity, indicating linkage between resistance loci and adaptive growth characteristics. Overcoming such trade-offs through pre-breeding and genomic introgression will be essential for broadening the genetic base of resistance.

Future strategies will also increasingly emphasize multi-trait and climate-resilient breeding, integrating resistance to both biotic and abiotic stresses. Climate change is expected to alter pathogen distribution and disease dynamics, requiring the development of cultivars with combined resistance and environmental adaptability. The integration of transcriptomics, phenomics, and high-throughput phenotyping platforms will further enhance the understanding of resistance mechanisms and their stability across environments.

In Kazakhstan, future perspectives are closely linked to the effective utilization of locally adapted germplasm and recently identified resistance loci. The discovery of numerous novel QTLs for NB, PM, and SR highlights the unique genetic resources available in the region. However, their practical application requires the establishment of integrated breeding pipelines combining GWAS, GS, and multi-environment validation. Strengthening collaboration between research institutes and breeding programs, along with improved pathogen monitoring systems, will be critical for translating genetic discoveries into resistant cultivars adapted to the highly variable continental climate.

Overall, the convergence of genomics, biotechnology, and data-driven breeding approaches provides a strong foundation for developing next-generation barley cultivars with durable, broad-spectrum disease resistance.

Conclusion

Breeding for genetic resistance remains the most sustainable and cost-effective strategy to mitigate the impact of major fungal diseases on barley production in Kazakhstan. Significant progress has been achieved through GWAS and molecular marker analyses, which have successfully identified both known and novel QTLs for resistance to net blotch, barley scald, powdery mildew, and stem rust under conditions of Kazakhstan. These findings confirm that global barley germplasm harbors valuable genetic diversity, including novel resistance loci that can enhance breeding programs. Nevertheless, several challenges remain, including the high evolutionary potential of pathogens, strong genotype × environment interactions, and the slow integration of modern genomic tools into practical breeding pipelines. The frequent “boom-and-bust” cycles associated with race-specific resistance genes further underscore the need for durable resistance strategies based on gene pyramiding and the accumulation of quantitative resistance loci. Looking forward, the effective deployment of advanced technologies such as genomic selection, pangenome resources, and CRISPR-based genome editing, combined with systematic pathogen monitoring and multi-environment phenotyping, offers promising avenues for accelerated breeding progress. Strengthening collaboration between research institutions and breeding centers will be essential to translate these genetic discoveries into high-yielding, multi-disease resistant barley cultivars adapted to the variable continental climate of Kazakhstan. Ultimately, the development of such resilient varieties will contribute substantially to the stability of barley production, livestock feed security, and agricultural sustainability in the region.

Author contribution

YG: conceptualization, literature search and analysis, data curation, writing – original draft, writing – review and editing, visualization, and preparation of tables and figures; YK: conceptualization, supervision, project administration, resources, validation, writing – review and editing, and funding acquisition.

Funding information

This research was supported by the Science Committee of the Ministry of Science and Higher Education of the Republic of Kazakhstan, Project No. AP23485118: Association mapping of resistance loci to net blotch and leaf scald of barley at seedling and adult plant growth stages (2024-2026).

References

1 Food and Agriculture Organization of the United Nations. (2024). A*gricultural production statistics 2010–2023 (FAOSTAT Analytical Brief No. 96).* Rome, Italy.

2 Lammas, M.E., Shitikova, A.V. (2024). Spring barley is a promising crop in agricultural production. *In BIO Web of Conferences*, 139, 01012. DOI: 10.1051/bioconf/202413901012.

3 Tricase, C., Amicarelli, V., Lamonaca, E., Rana, R.L. (2018). Economic analysis of the barley market and related uses. *Grasses as Food and Feed,* 10, 25–46. DOI: 10.5772/intechopen.78967.

4 Perera, W.N.U., Abdollahi, M.R., Zaefarian, F., Wester, T.J., Ravindran, V. (2022). Barley, an undervalued cereal for poultry diets: Limitations and opportunities. *Animals,* 12(19), 2525. DOI: 10.3390/ani12192525.

5 Global Market Insights. (2024). *Barley market size & share, global statistics report 2025–2034* https://www.gminsights.com/industry-analysis/barley-market

6 Bureau of National Statistics, Agency for Strategic Planning and Reforms of the Republic of Kazakhstan. (2025). https://stat.gov.kz/en/

7 Tomić, A., Trkulja, V., Matić, S., Trkulja, N., Iličić, R., Scortichini, M., Popović Milovanović, T. (2024). Net blotch (*Pyrenophora teres* Drechsler): An increasingly significant threat to barley production. *Plant Protection Science*, 60(1), 1–30. DOI: 10.17221/122/2023-PPS.

8 Walters, D.R., Avrova, A., Bingham, I.J., Burnett, F.J., Fountaine, J., Havis, N.D., Newton, A. C. (2012). Control of foliar diseases in barley: Towards an integrated approach. *European Journal of Plant Pathology*, 133(1), 33–73. DOI: 10.1007/s10658-012-9948-x.

9 Sultanova, N.Z., Uspanov, M., Bekezhanova, M., Sarsenbayeva, G.B., Rysbekova, A.M. (2021). Morphological signs of barley spot pathogens in the conditions of the Almaty region. *Online Journal of Biological Sciences*, 21(2), 223–231. DOI: 10.3844/ojbsci.2021.223.231.

10 Genievskaya, Y., Maulenbay, A., Zatybekov, A., Abugalieva, S., Turuspekov, Y. (2026). SSR-based analysis of genetic diversity and resistance to barley scald and net blotch in a collection of barley from Kazakhstan. *Genes*, 17(3), 261. DOI: 10.3390/genes17030261.

11 Rsaliev, A.S., Chudinov, V.A., Amirkhanova, N.T. (2016). Ustoichivost' selekcionnyh materialov yachmenya Karabalykskoi sel"skohozyaistvennoi opytnoi stancii k setchatoi pyatnistosti i muchnistoi rose. *Reports of the national academy of sciences*, 2224. [*in Russ*].

12 Rsaliyev, A., Pahratdinova, Z., Rsaliyev, S. (2017). Characterizing the pathotype structure of barley powdery mildew and effectiveness of resistance genes to this pathogen in Kazakhstan. *BMC Plant Biology,* 17(Suppl 1), 178. DOI: /10.1186/s12870-017-1098-4.

13 Genievskaya, Y., Zatybekov, A., Abugalieva, S., Turuspekov, Y. (2023). Identification of quantitative trait loci associated with powdery mildew resistance in spring barley under conditions of southeastern Kazakhstan. *Plants,* 12(12), 2375. DOI: 10.3390/plants12122375.

14 Genievskaya, Y., Maulenbay, A., Rsaliyev, S., Abugalieva, S., Rsaliyev, A., Zatybekov, A., Turuspekov, Y. (2025). Dissecting adult plant resistance to stem rust through multi-model GWAS in a diverse barley germplasm panel. *Frontiers in Plant Science,* 16, 1681398. DOI: 10.3389/fpls.2025.1681398.

15 Jørgensen, J.H., Wolfe, M. (1994). Genetics of powdery mildew resistance in barley. *Critical Reviews in Plant Sciences,* 13(1), 97–119. DOI: 10.1080/07352689409701910.

16 Hoheneder, F., Biehl, E.M., Hofer, K., Petermeier, J., Groth, J., Herz, M., Hückelhoven, R. (2022). Host genotype and weather effects on Fusarium head blight severity and mycotoxin load in spring barley. *Toxins,* 14(2), 125. DOI: 10.3390/toxins14020125.

17 Corkley, I., Fraaije, B., Hawkins, N. (2022). Fungicide resistance management: Maximizing the effective life of plant protection products. *Plant Pathology,* 71(1), 150–169. DOI:10.1111/ppa.13467.

18 Sakr, N. (2022). Erosion of Quantitative Resistance in Wheat and Barley to Fusarium Head Blight: Gene Pyramiding Achieves and Durability Study. *The Open Agriculture Journal,* 17(1). DOI:10.2174/18743315-v16-e221117-2022-HT14-3623-3.

19 Kunze, K.H., Meints, B., Massman, C., Gutiérrez, L., Hayes, P.M., Smith, K.P., Sorrells, M.E. (2024). Genome-wide association of an organic naked barley diversity panel identified quantitative trait loci for disease resistance. *The Plant Genome,* 17(4), e20530. DOI:10.1002/tpg2.20530.

20 Turuspekov, Y., Ormanbekova, D., Rsaliev, A., Abugalieva, S. (2016). Genome-wide association study on stem rust resistance in Kazakh spring barley lines. *BMC Plant Biology,* 16(Suppl 1), 6. DOI:10.1186/s12870-015-0686-z.

21 Genievskaya, Y., Maulenbay, A., Zatybekov, A., Turuspekov, Y. (2026). Genome-wide association mapping and functional annotation of loci conferring resistance to *Pyrenophora teres* f. *teres* in barley. *Frontiers in Agronomy.*

22 Backes, A., Guerriero, G., Ait Barka, E., Jacquard, C. (2021). Pyrenophora teres: Taxonomy, morphology, interaction with barley, and mode of control. *Frontiers in Plant Science,* 12, 614951. DOI:10.3389/fpls.2021.614951.

23 Liu, Z., Ellwood, S.R., Oliver, R.P., Friesen, T.L. (2011). Pyrenophora teres: Profile of an increasingly damaging barley pathogen. *Molecular Plant Pathology,* 12(1), 1–19. DOI:10.1111/j.1364-3703.2010.00649.x.

24 Tekauz, A. (1985). A numerical scale to classify reactions of barley to Pyrenophora teres. *Canadian Journal of Plant Pathology*, 7(2), 181-183. DOI: 10.1080/07060668509501499.

25 Peterson, R.F., Campbell, A.B., Hannah, A.E. (1948). A diagrammatic scale for estimating rust intensity on leaves and stems of cereals. *Canadian Journal of Research, Section C,* 26(5), 496–500. DOI: 10.1139/cjr48c-033.

26 Zhang, X., Ovenden, B., Milgate, A. (2020). Recent insights into barley and Rhynchosporium commune interactions. *Molecular Plant Pathology*, 21(8), 1111–1128. DOI:10.1111/mpp.12945.

27 Avrova, A., Knogge, W. (2012). Rhynchosporium commune: A persistent threat to barley cultivation. Molecular Plant Pathology, 13(9), 986–997. DOI:10.1111/j.1364-3703.2012.00811.x.

28 Ababa, G., Kesho, A., Tadesse, Y., Amare, D. (2023). Reviews of taxonomy, epidemiology, and management practices of the barley scald (*Rhynchosporium graminicola*) disease. *Heliyon*, 9(3). DOI: 10.1016/j.heliyon.2023.e14315.

29 Zhunusova, A., Sarbaev, A. (2022). Arpa sort-ülgileriniñ negizgi sañyrauqūlaq aurularyna tözimdiligi. *Biosafety and biotechnology*, 10, 31–43. DOI:10.58318/2957-5702-2022-9-31-43. [*in Kaz*].

30 Lambertucci, S., Orman, K.M., Das Gupta, S., Fisher, J.P., Gazal, S., Williamson, R.J., Bindschedler, L.V. (2019). Analysis of barley leaf epidermis and extrahaustorial proteomes during powdery mildew infection reveals that the PR5 thaumatin-like protein TLP5 is required for susceptibility towards *Blumeria graminis* f. sp. *hordei. Frontiers in Plant Science,* 10, 1138. DOI:10.3389/fpls.2019.01138.

31 Dreiseitl, A. (2020). Specific resistance of barley to powdery mildew, its use and beyond: A concise critical review. *Genes*, 11(9), 971. DOI:10.3390/genes11090971.

32 Chaure, P., Gurr, S.J., Spanu, P. (2000). Stable transformation of Erysiphe graminis, an obligate biotrophic pathogen of barley. *Nature Biotechnology,* 18(2), 205–207. DOI:10.1038/72666.

33 Balkema-Boomstra, A.G., Mastebroek, H.D. (1995). Effect of powdery mildew (*Erysiphe graminis* f. sp. *hordei*) on photosynthesis and grain yield of partially resistant genotypes of spring barley (*Hordeum vulgare L.*). *Plant Breeding*, 114(2), 126–130. DOI:10.1111/j.1439-0523.1995.tb00776.x.

34 Leonard, K.J., Szabo, L.J. (2005). Stem rust of small grains and grasses caused by Puccinia graminis. *Molecular Plant Pathology*, 6(2), 99–111.

35 McIntosh, R.A., Wellings, C.R., Park, R.F. (1995). *Wheat rusts: An atlas of resistance genes.* CSIRO Publishing.

36 Van Niekerk, B.D., Pretorius, Z.A., Boshoff, W.H.P. (2001). Potential yield losses caused by barley leaf rust and oat leaf and stem rust to South African barley and oat cultivars. *South African Journal of Plant and Soil*, 18(3), 108–113.

37 Stakman, E.C., Stewart, D.M., Loegering, W.Q. (1962). Identification of physiologic races of *Puccinia graminis* var. *tritici.* U.S. Department of Agriculture, ARS-E-617. 53 p.

38 Clare, S.J., Novakazi, F., Hayes, P.M., Moscou, M.J., Brueggeman, R.S. (2024). Colocalization of genetic regions that confer resistance/susceptibility against Puccinia species and association with Pyrenophora teres loci within the barley genome. *Frontiers in Agronomy,* 6, 1451281. DOI:10.3389/fagro.2024.1451281.

39 Adhikari, A., Steffenson, B.J., Smith, M.J., Dill-Macky, R. (2019). Genome-wide association mapping of seedling net form net blotch resistance in an Ethiopian and Eritrean barley collection. *Crop Science,* 59, 1625–1638. DOI:10.2135/cropsci2019.01.0003.

40 Muria-Gonzalez, M.J., Lawrence, J.A., Palmiero, E., D'Souza, N.K., Gupta, S., Ellwood, S.R. (2023). Major susceptibility gene epistasis over minor gene resistance to spot form net blotch in a commercial barley cultivar. *Phytopathology*, 113, 1058–1065. DOI:10.1094/PHYTO-10-22-0376-R.

41 Looseley, M.E., Griffe, L.L., Büttner, B., Wright, K.M., Middlefell-Williams, J., Bull, H., Avrova, A. (2018). Resistance to Rhynchosporium commune in a collection of European spring barley germplasm. *Theoretical and Applied Genetics,* 131(12), 2513–2528. DOI:10.1007/s00122-018-3168-5.

42 Backes, G., Madsen, L., Jaiser, H., Stougaard, J., Herz, M., Mohler, V., Jahoor, A. (2003). Localisation of genes for resistance against Blumeria graminis f. sp. hordei and Puccinia graminis in a cross between a barley cultivar and a wild barley *(Hordeum vulgare* ssp. *spontaneum*) line. T*heoretical and Applied Genetics,* 106(2), 353–362. DOI:10.1007/s00122-002-1148-1.

43 Pogoda, M., Liu, F., Douchkov, D., Djamei, A., Reif, J.C., Schweizer, P., Schulthess, A.W. (2020). Identification of novel genetic factors underlying the host–pathogen interaction between barley (*Hordeum vulgare L.*) and powdery mildew (*Blumeria graminis* f. sp. *hordei*). *PLoS ONE,* 15(7), e0235565. DOI: 10.1371/journal.pone.0235565.

44 Mirlohi, A., Brueggeman, R., Drader, T., Nirmala, J., Steffenson, B.J., Kleinhofs, A. (2008). Allele sequencing of the barley stem rust resistance gene Rpg1 identifies regions relevant to disease resistance. *Phytopathology*, 98, 910–918. DOI:10.1094/phyto-98-8-0910.

45 Chełkowski, J., Tyrka, M., Sobkiewicz, A. (2003). Resistance genes in barley (Hordeum vulgare L.) and their identification with molecular markers. *Journal of Applied Genetics*, 44(3), 291–310.

46 Brueggeman, R., Rostoks, N., Kudrna, D., Kilian, A., Han, F., Chen, J., Kleinhofs, A. (2002). The barley stem rust-resistance gene Rpg1 is a novel disease-resistance gene with homology to receptor kinases. *Proceedings of the National Academy of Sciences,* 99(14), 9328–9333. DOI:10.1073/pnas.142284999.

47 Caldo, R.A., Nettleton, D., Wise, R.P. (2004). Interaction-dependent gene expression in Mla-specified response to barley powdery mildew. *The Plant Cell*, 16(9), 2514–2528. DOI:10.1105/tpc.104.023382.

48 Cieplak, M., Nucia, A., Ociepa, T., Okoń, S. (2022). Virulence structure and genetic diversity of *Blumeria graminis* f. sp. *avenae* from different regions of Europe. *Plants,* 11(10), 1358. DOI:10.3390/plants11101358.

49 Cao, Y., Si, B., Zhu, G., Xu, X., Li, W., Chen, S., Li, T. (2019). Race and virulence of asexual and sexual populations of Puccinia graminis f. sp. tritici in China from 2009 to 2015. *European Journal of Plant Pathology*, 153(2), 545–555. DOI:10.1007/s10658-018-1581-x.

50 Kolkman, J.M., Sepp, S.S., Kunze, K.H., Bergstrom, G.C., Sorrells, M.E. (2026). Genome-wide association analysis of resistance to scald in an adapted multiparent winter malting barley population. *bioRxiv.* DOI:10.1101/2026.03.

51 Sharma Poudel, R., Al-Hashel, A. F., Gross, T., Gross, P., Brueggeman, R. (2018). Pyramiding rpg4- and Rpg1-mediated stem rust resistance in barley requires the Rrr1 gene for both to function. *Frontiers in Plant Science,* 9, 1789. DOI:10.3389/fpls.2018.01789.

52 Lorenz, A.J., Smith, K.P., Jannink, J.L. (2012). Potential and optimization of genomic selection for Fusarium head blight resistance in six-row barley. *Crop Science*, 52(4), 1609–1621. DOI:10.2135/cropsci2011.09.0503.

53 Tiede, T., Smith, K.P. (2018). Evaluation and retrospective optimization of genomic selection for yield and disease resistance in spring barley. *Molecular Breeding,* 38(5), 55. DOI:10.1007/s11032-018-0820-3.

54 Hautsalo, J., Novakazi, F., Jalli, M., Göransson, M., Manninen, O., Isolahti, M., Bengtsson, T. (2021). Pyramiding of scald resistance genes in four spring barley MAGIC populations. *Theoretical and Applied Genetics*, 134(12), 3829–3843. DOI:10.1007/s00122-021-03930-y.

55 Singh, D., Dracatos, P.M., Loughman, R., Park, R.F. (2017). Genetic mapping of resistance to Puccinia hordei in three barley doubled-haploid populations. *Euphytica,* 213(1), 16. DOI:10.1007/s10681-016-1799-7.

56 Czembor, J.H., Czembor, E., Suchecki, R., Watson-Haigh, N.S. (2021). Genome-wide association study for powdery mildew and rusts adult plant resistance in European spring barley from Polish gene bank. *Agronomy*, 12(1), 7. DOI:10.3390/agronomy12010007

57 Shumenova, N. Z., Nauanova, A. P., Tyfekciuglu, A. (2023). Biotyñaitqyştardyñ soltüstik qazaqstan jağdaiynda jazdyq arpanyñ sañyrauqūlaq aurularynyñ taraluyna äseri. Herald of science of S. Seifullin Kazakh agrotechnical university: *Multidisciplinary,* 3(118), 28-36. DOI: 10.51452/kazatu.2023.3(118).1476. [in Kaz].

58 Kusch, S., Qian, J., Loos, A., Kümmel, F., Spanu, P.D., Panstruga, R. (2024). Long-term and rapid evolution in powdery mildew fungi. *Molecular Ecology,* 33(10), e16909. DOI:10.1111/mec.16909.

59 McDonald, B.A., Linde, C. (2002). Pathogen population genetics, evolutionary potential, and durable resistance. *Annual Review of Phytopathology,* 40(1), 349–379. DOI:10.1146/annurev.phyto.40.120501.101443.

60 Dreiseitl, A. (2025). Major genes for powdery mildew resistance in research and breeding of barley: A few brief narratives and recommendations. Plants, 14(14), 2091. DOI:10.3390/plants14142091.

61 Jørgensen, J.H., Wolfe, M. (1994). Genetics of powdery mildew resistance in barley. *Critical Reviews in Plant Sciences*, 13(1), 97–119. DOI:10.1080/07352689409701910.

62 Ankamah-Yeboah, T., Janss, L.L., Jensen, J.D., Hjortshøj, R.L., Rasmussen, S.K. (2020). Genomic selection using pedigree and marker-by-environment interaction for barley seed quality traits from two commercial breeding programs. *Frontiers in Plant Science,* 11, 539. DOI:10.3389/fpls.2020.00539.

63 Muñoz-Amatriaín, M., Cuesta-Marcos, A., Hayes, P.M., Muehlbauer, G.J. (2014). Barley genetic variation: Implications for crop improvement. *Briefings in Functional Genomics,* 13(4), 341–350. DOI:10.1093/bfgp/elu006.

64 Hawkins, N.J., Fraaije, B.A. (2018). Fitness penalties in the evolution of fungicide resistance. *Annual Review of Phytopathology, 5*6(1), 339–360. DOI:10.1146/annurev-phyto-080417-050012.

65 Xu, Y., Crouch, J.H. (2008). Marker-assisted selection in plant breeding: From publications to practice. *Crop Science*, 48(2), 391–407. DOI:10.2135/cropsci2007.04.0191.

66 Xu, Y. (2016). Envirotyping for deciphering environmental impacts on crop plants. *Theoretical and Applied Genetics*, 129(4), 653–673. DOI:10.1007/s00122-016-2691-5.

67 Jayakodi, M., Lu, Q., Pidon, H., Rabanus-Wallace, M.T., Bayer, M., Lux, T., et al. (2024). Structural variation in the pangenome of wild and domesticated barley. *Nature,* 636(8043), 654–662. DOI:10.1038/s41586-024-08187-1.

68 Jash, S., Chetri, A., Syed, R., Upadhyay, D.K., Mandal, S. (2026). CRISPR-Cas-based gene editing in barley for trait enhancement. In Gene editing in cereals: Approaches and applications. *Springer Nature Singapore*. 259–291. DOI:10.1007/978-981-95-6722-5_10.

69 Massman, J., Cooper, B., Horsley, R., Neate, S., Dill-Macky, R., Chao, S., Smith, K.P. (2011). Genome-wide association mapping of Fusarium head blight resistance in contemporary barley breeding germplasm. *Molecular Breeding*, 27(4), 439–454. DOI:10.1007/s11032-010-9442-0.

Генетическая устойчивость ячменя к основным грибным болезням в Казахстане: достижения, проблемы и перспективы селекции

Гениевская Ю.А., Туруспеков Е.К.

Аннотация

Предпосылки и цель. Ячмень (*Hordeum vulgare L.*) является одной из ключевых зерновых культур в Казахстане, занимая второе место после пшеницы и играя важную роль в животноводстве и продовольственной безопасности. Однако его продуктивность существенно ограничивается

грибными заболеваниями, включая сетчатую пятнистость, ринхоспориоз, мучнистую росу и стеблевую ржавчину, вызывающими потери урожая от 10 до 40%, а при эпифитотиях – до 100%. Цель обзора – обобщение современных данных о генетической основе устойчивости к этим болезням с акцентом на гены и локусы количественных признаков (ЛКП) и их использование в селекции Казахстана.

Материалы и методы. Обзор основан на анализе опубликованных данных и молекулярно-генетических исследований, включая полногеномный анализ ассоциаций (GWAS) и SSR-маркерный анализ коллекций генетически разнообразного ячменя. Особое внимание уделено исследованиям, проведённым в агроэкологических условиях Казахстана.

Результаты. Выявлено значительное число локусов устойчивости к основным болезням ячменя. В Казахстане с помощью GWAS идентифицировано 59 ЛКП для сетчатой пятнистости (25 новых), 7 ЛКП для мучнистой росы (5 новых) и 19 ЛКП для стеблевой ржавчины (13 новых), а также подтверждена роль известных генов устойчивости. Показано наличие как широко распространённых, так и регионально специфичных источников устойчивости. Подчёркнута важность сочетания major genes и количественной устойчивости, включая устойчивость взрослого растения (APR), для обеспечения её долговечности.

Заключение. Молекулярные подходы, такие как GWAS, маркер-ассоциированная и геномная селекция, значительно ускоряют селекцию ячменя на устойчивость к грибным болезням. Перспективными направлениями остаются пирамидирование генов, расширение генетического разнообразия исходного материала, а также внедрение пангеномики и геномного редактирования для создания устойчивых сортов, адаптированных к климату Казахстана.

Ключевые слова: сетчатая пятнистость; ринхоспориоз; мучнистая роса; стеблевая ржавчина; ЛКП.

Қазақстандағы арпаның негізгі саңырауқұлақ ауруларына генетикалық төзімділігі: жетістіктер, мәселелер және селекциялық перспективалар

Гениевская Ю.А., Туруспеков Е.К.

Түйін

Алғышарттар мен мақсат. Арпа (*Hordeum vulgare L.*) Қазақстандағы негізгі дәнді дақылдардың бірі болып табылады, бидайдан кейін екінші орын алады және мал шаруашылығы мен азық-түлік қауіпсіздігін қамтамасыз етуде маңызды рөл атқарады. Алайда оның өнімділігі негізгі саңырауқұлақ ауруларымен, атап айтқанда торлы дақ, ринхоспориоз, ақ ұнтақ ауруы және сабақ таты ауруларымен айтарлықтай шектеледі. Бұл аурулар өнімділіктің 10–40% төмендеуіне, ал эпифитотия жағдайында 100%-ға дейін жоғалуына әкелуі мүмкін. Осы шолудың мақсаты – аталған ауруларға төзімділіктің генетикалық негіздері туралы заманауи мәліметтерді жинақтау, оның ішінде төзімділік гендері, сандық белгілер локустары (СБЛ) және оларды Қазақстан жағдайында селекцияда қолдану.

Материалдар мен әдістер. Шолу жарияланған ғылыми еңбектерді және заманауи молекулалық-генетикалық зерттеулерді талдауға негізделген, соның ішінде толықгеномдық ассоциациялық зерттеулер (GWAS) және SSR-маркерлерді талдау, әлемдік және жергілікті бейімделген арпа генқорында жүргізілген. Ерекше назар Қазақстанның агроэкологиялық жағдайларында орындалған зерттеулерге аударылды, олар негізгі фитопатогендерге төзімділікті бағалауға және сәйкес генетикалық локустарды анықтауға бағытталған.

Нәтижелер. Соңғы зерттеулер арпаның негізгі ауруларына төзімділікпен байланысты көптеген генетикалық локустарды анықтады. Қазақстанда жүргізілген GWAS нәтижесінде торлы даққа қатысты 59 СБЛ (оның ішінде 25 жаңа), ақ ұнтақ ауруы 7 СБЛ (5 жаңа) және сабақ татына 19 СБЛ (13 жаңа) анықталды, сондай-ақ белгілі төзімділік гендерінің бірқатары расталды. Бұл нәтижелер жергілікті генқорда жаһандық деңгейде белгілі және аймаққа тән төзімділік көздерінің бар екенін көрсетеді. Сонымен қатар, төзімділіктің тұрақтылығын арттыру үшін негізгі гендерді

сандық локустармен, оның ішінде ересек өсімдік кезеңіндегі төзімділікпен (APR), біріктірудің маңыздылығы дәлелденді.

Қорытынды. GWAS, маркерге негізделген селекция (MAS) және геномдық селекция (GS) сияқты молекулалық әдістерді қолдану арпаның ауруларға төзімділігін арттыру селекциясын жеделдетуге мүмкіндік береді. Дегенмен, патогендердің жоғары өзгергіштігі, генотип × орта өзара әрекеттесуі және геномдық технологиялардың селекцияға жеткіліксіз енгізілуі сияқты мәселелер сақталуда. Болашақта гендерді пирамидалау, генетикалық әртүрлі генқорды пайдалану, сондай-ақ пангеномика және геномды редакциялау сияқты озық технологияларды енгізу арқылы Қазақстанның континенттік климатына бейімделген, бірнеше ауруға төзімді арпа сорттарын шығару маңызды болып табылады.

Кілт сөздер: торлы дақ; ринхоспориоз; ақ ұнтақ ауруы; сабақ таты; СБЛ.