ANNUAL REVIEWS

Annual Review of Phytopathology

Challenges to Wheat Disease Resistance and Current Global Strategies

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https://doi.org/10.1146/annurev-phyto-121923-082727

Keywords

Triticum, pathogen, epidemiology, resistance, breeding, biotechnology

Abstract

Wheat yields have continued to increase globally at a steady pace over the past decade despite challenges faced by breeding programs from evolving and migrating races of rust and other wheat disease–inducing fungi. Additionally, pathogens are becoming tolerant to fungicides because of their injudicious use. We highlight the challenges in breeding and deploying resistant varieties and discuss global strategies to protect wheat from diseases. The continuous identification, utilization, and deployment of diverse resistance genes and quantitative trait loci for durable adult plant resistance, supported by precision phenotyping, marker-assisted and genomic selection, real-time pathogen diagnostics, and the rapid diffusion of resistant varieties, are helping to minimize crop losses while enhancing productivity. The potential for genetic engineering, including the introduction of resistance gene cassettes

and precise genome editing of susceptibility or resistance genes, has also increased because of the recent acceptance of genetically modified wheat carrying the HB4® drought tolerance gene in some countries.

INTRODUCTION

More than 220 million hectares of wheat are sown annually across all continents, encompassing a diverse range of latitudes, altitudes, crop rotations, and production practices. This vast cultivation area creates favorable environmental conditions for more than 50 important diseases and pests (119). The diseases of global and regional significance are predominantly fungal and caused by either biotrophic or necrotrophic fungi. Recent estimates suggest that potential yield losses in wheat due to diseases range from 10 to 28% globally (97). As a result, controlling these diseases has become a high priority, leading to reductions in incidence, severity, and the magnitude of epidemics (106).

King et al. (62) estimated that the cultivation of resistant wheat varieties has allowed for the avoidance of more than a billion liters of fungicide applications since 2000. Furthermore, the compound average annual growth in wheat production from 2014 to 2024 was 0.77%, with nearly 790 million tons harvested from 220 million hectares in marketing year 2023/2024 (111). Improved disease resistance, along with effective chemical control strategies and agronomic practices, has ensured that higher yields of newer varieties remain protected, even in disease-favorable conditions created by intensive farming, high nitrogen use, zero or reduced tillage, stubble retention, and crop rotations such as maize–wheat.

Approximately a dozen diseases, caused by both biotrophic and necrotrophic fungi, continue to pose considerable threats in specific areas or regions or worldwide. These diseases continue to receive substantial research and breeding investments, despite the implementation of successful management strategies—be they genetic, chemical, or a combination of both. In this review, we have focused on three rust diseases (stripe rust, stem rust, and leaf rust), Fusarium head blight (FHB), and wheat blast (WB) to illustrate challenges and global strategies employed for their management. The review highlights key challenges in resistance breeding for wheat diseases, including the continuous evolution and migration of pathogens, fungicide resistance, the replacement of susceptible varieties, and breeding complexity. It outlines global strategies to address these issues, such as enhancing knowledge and diversity of resistance, precision phenotyping, leveraging resistance (*R*) genes and quantitative adult plant resistance (APR), breeding for specific diseases like FHB and WB, genomic selection, genetic engineering, rapid molecular diagnostics for pathogen monitoring, and accelerating the adoption of resistant varieties.

Rust Diseases

Stem (or black) rust (SR), stripe (or yellow) rust (YR), and leaf (or brown) rust (LR) are caused by the biotrophic fungi *Puccinia graminis tritici* (*Pgt*), *Puccinia striiformis tritici* (*Pst*), and *Puccinia triticina*, respectively. These diseases are present in nearly all wheat-growing regions and have consistently hindered global wheat production since domestication, continuing to threaten global wheat supplies (100, 106). The rust fungi survive during the offseason on voluntary wheat plants and secondary host grasses in areas where alternate hosts are absent. Cooler highland areas with wet conditions due to frequent rains and dew provide the most suitable environment for pathogen survival. Continuous wheat cropping in the eastern African highlands further supports high inoculum loads throughout the year, maintaining a continuous supply of

inoculum in the region and beyond (102, 103). Similarly, summer wheat crops and the presence of secondary and alternate hosts in other mountainous regions worldwide also facilitate rust survival.

The airborne urediniospores of rust fungi can travel short to long distances, even intercontinentally, spreading diseases and new virulent races (101, 106). Highly diverse populations of rust fungi, with both asexual and sexual reproduction mechanisms, continuously evolve, leading to localized epidemics in various wheat-growing regions. The deployment of single race-specific *R* genes or their combinations has resulted in boom-and-bust cycles due to the rapid selection of virulent races targeting these *R* genes (102). Furthermore, several *R* genes are effective only in specific geographies, limiting their global deployment.

Fusarium Head Blight

Also known as head scab, FHB is one of the most significant wheat spike diseases worldwide, caused by several species in the *Fusarium* genus, predominantly the *Fusarium graminearum* clade (73). Most FHB pathogens thrive in warmer, humid environments, making wheat cultivation areas in East Asia, Europe, North America, the Southern Cone of South America, and Eastern and Southern Africa the most severely affected. Yield losses attributed to this disease are substantial in epidemic regions; for instance, FHB caused approximately $7.67 billion in yield losses for wheat and barley in the United States between 1993 and 2001 (76). In recent years, there has been increasing concern over mycotoxins produced by these pathogens, particularly deoxynivalenol (DON), which is toxic to humans and animals (11). The incidence, severity, and spread of FHB are linked to climate change, maize–wheat rotations, monoculture of wheat and other small grains, and conservation agriculture practices, including reduced tillage and stubble retention (129). Although various management methods have been developed to combat FHB, none provide complete control, leading to the widespread adoption of integrated FHB management strategies that prioritize varietal resistance and chemical fungicides.

Wheat Blast

It is a relatively new disease, initially confined to South America since its first report in 1985, until the 2016 outbreak in Bangladesh. The pathogen, *Magnaporthe oryzae* pathotype *Triticum* (*MoT*), can infect all aerial parts of the wheat plant, but head blast is the most prominent symptom in the field, causing yield reductions that range from negligible to 100%, depending on weather conditions, varietal resistance, and management approaches (23). The disease is favored by hot and humid conditions around the heading stage and develops rapidly, allowing little time for remedial measures; therefore, prevention is key. Disease forecasting is essential for implementing preventive measures, and several WB prediction models have been developed (24). Cultural management strategies are also effective; it is widely accepted that early sowing in South America and late sowing in South Asia should be avoided to prevent severe WB infection (99). Some chemicals, primarily strobilurins and triazoles, have been identified for field application against WB, but they often yield unsatisfactory results and may lead to fungal resistance when used at high doses or increased frequencies (14).

CHALLENGES TO RESISTANCE BREEDING

Continuous Evolution and Migration of Pathogens

The evolution and spread of new virulent pathotypes present a continual challenge for effective wheat disease control strategies. Climate change and global trade further accelerate their

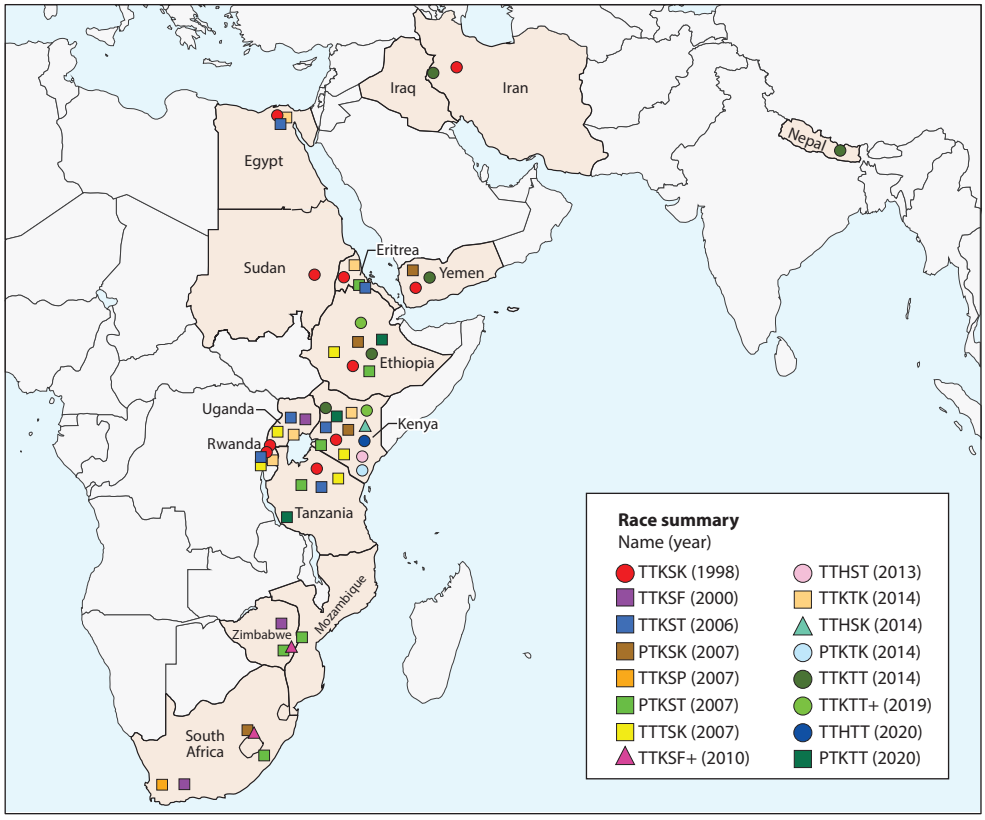

Figure 1

Summary and spread of detected races belonging to the Ug99 (TTKSK; belonging to Clade I) lineage of stem rust fungus between 1998 and 2024 (52, 83, 84, 86, 89, 92, 106). Figure adapted from Reference 101; 2011 *Annu. Rev. Phytopathol.*

migration, leading to emerging disease outbreaks. Understanding pathogen dynamics is crucial for developing durable disease-resistant wheat cultivars and effective management strategies.

Rust diseases. In recent decades, the emergence and migration of significant race lineages of both *Pgt* and *Pst* have posed a substantial threat to wheat production across multiple continents (43, 101). The Ug99 lineage of *Pgt* has been a focal point for monitoring and exemplifies the rapid changes occurring. First detected in Uganda in 1998 (92), the Ug99 (race TTKSK) lineage now comprises 16 races that have spread across 15 countries and two continents (**Figure 1**).

More than two decades after its initial detection, Ug99 races still dominate in most East African countries. Over time, additional virulence has developed, notably affecting resistance genes *Sr24*, *Sr36*, *SrTmp*, and *Sr8155B1*, rendering many commercial cultivars susceptible (52, 84, 86). Migration out of Africa has also been documented, with the highly virulent Ug99 variant TTKTT—exhibiting combined virulence to *Sr31*, *Sr24*, and *SrTmp*—detected in Iraq (83), Yemen (K. Nazari, personal communication), and, most recently, Nepal (89). The recent spread of the Ug99 lineage into other significant wheat-growing regions, such as South Asia, raises concerns and necessitates close monitoring.

In addition to Ug99, other lineages of *Pgt* also threaten wheat production. In 2013–2014, *Pgt* race TKTTF caused a major epidemic in Ethiopia, affecting the widely grown cultivar

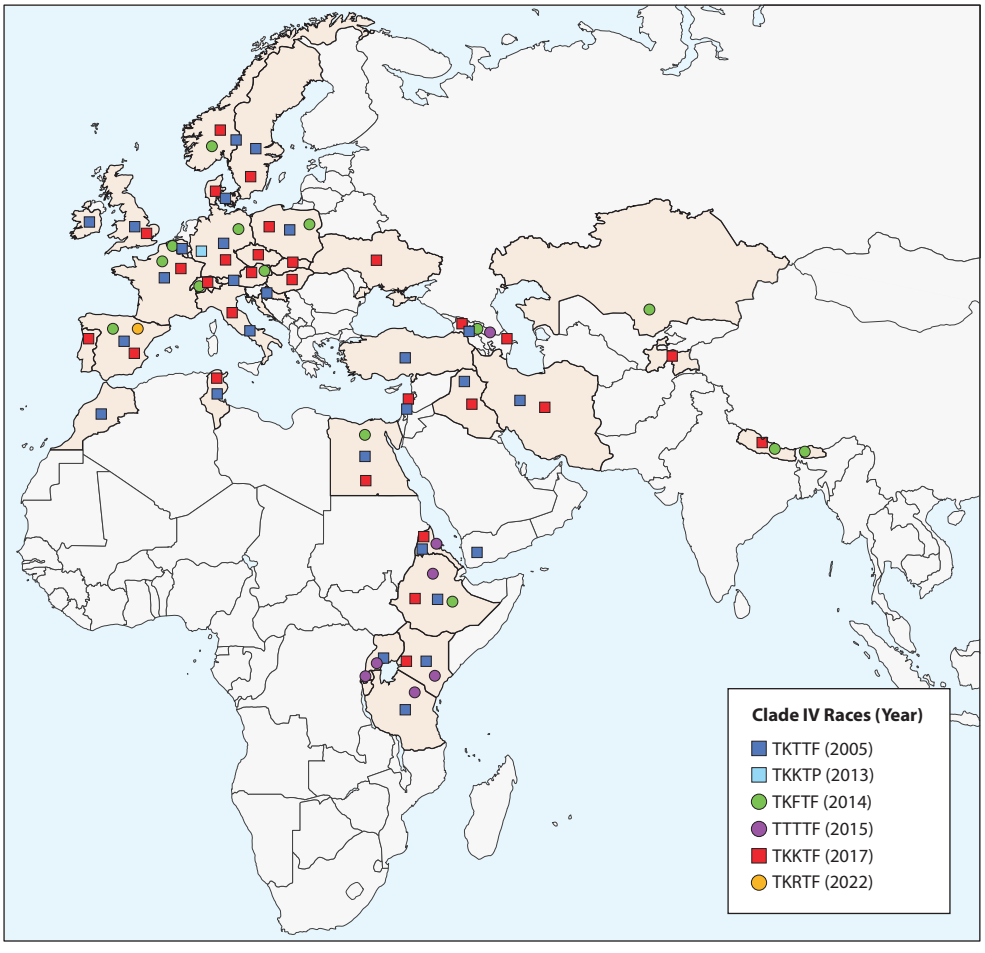

Figure 2

Summary and spread of detected races belonging to the Clade IV lineage of stem rust fungus between 1998 and 2024 (85, 90, 103).

'Digalu', which carries the *SrTmp* resistance gene (85). This race, believed to have originated in the Turkey/Caucasus region, was genetically distinct from Ug99 and classified as Clade IV (subclade B) (103). Since the 2013–2014 epidemic, Clade IV races have dominated in Ethiopia, even with the ongoing presence of Ug99 races. This clade, which comprises eight identified subclades and six races, has spread rapidly in recent years (**Figure 2**). In fewer than two decades, the Clade IV lineage has expanded across the Middle East, Europe, East Africa, and North Africa (90), and in 2024 it was detected for the first time in South Asia, with races TKKTF and TKFTF confirmed in Nepal and race TKFTF confirmed in Bhutan (Cereal Disease Laboratory, United States; Global Rust Reference Center, Denmark; National Agricultural Research Center, Nepal, unpublished results). The wide geographical spread and ongoing evolution of lineages like Ug99 and Clade IV underscore the re-emerging threat that SR poses to wheat production.

YR has emerged as an increasingly important disease since the 1960s (6) and is now considered one of the most significant wheat diseases globally (17). *Pst* has also exhibited significant changes over the past two decades, with the detection and rapid spread of important new genetic groups. The spread of *Pst* genetic groups has occurred on a global scale, with movements

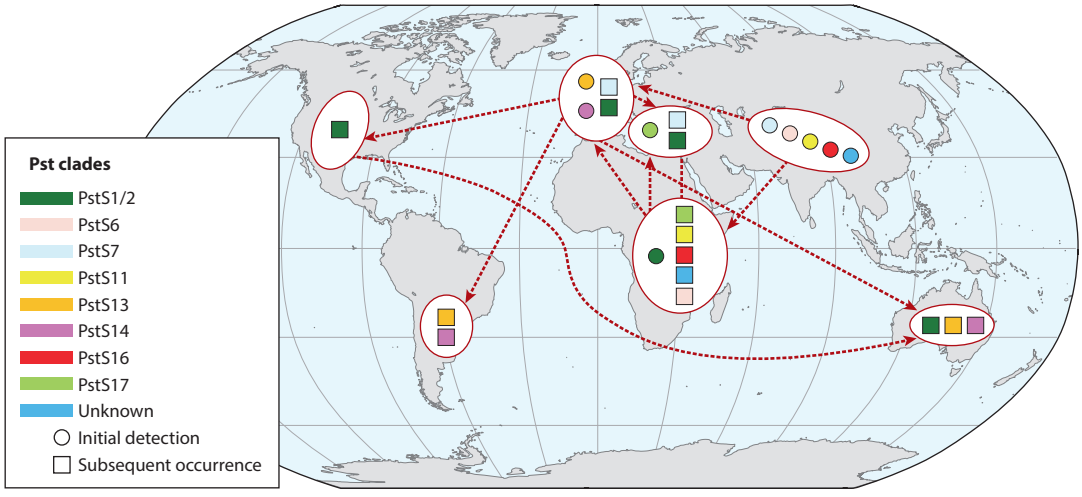

Figure 3

Summary and spread of recent races belonging to different lineages of stripe rust fungus (4, 44–47, 106, 115, 120). Circles represent initial detection regions and squares represent subsequent occurrence regions. Figure adapted from Reference 106; 2016 *Annu. Rev. Phytopathol.*

detected between all continents except Antarctica (**Figure 3**). The genetic group *PstS1/S2* is believed to have originated in East Africa (115) and has rapidly spread to Europe, North America, and, subsequently, Australia (4, 45, 120). This genetic group has demonstrated increased aggressiveness, characterized by shorter latent periods and adaptation to higher temperatures (78). These traits likely contributed to its successful and rapid colonization across several continents, including areas where YR was previously not a concern, exemplifying the increased adaptability of YR.

The *PstS1/S2* race group likely migrated from California, USA, to Mexico, and was first detected in Baja California in 2002 and then spread to the central Mexican highlands, where wheat is grown in summer, within a year. New variants were identified in subsequent years carrying virulence to resistance genes *Yr1*, *Yr8*, *Yr10*, *Yr17*, *Yr24* (=*Yr26*), *Yr27*, *Yr31*, and *YrPoll* (an unidentified gene in triticale) (47).

The Himalayan region is recognized as a center of pathogen diversity for *Pst* (3), with barberry reported to play a significant role in the emergence of new races in China (127) and strong indications of a similar role in Pakistan (2, 3, 77). Monitoring has shown a strong connection between the Himalayan region and East Africa, with at least four genetic groups believed to have originated in the Himalayas migrating into East Africa within a short time frame (**Figure 3**). The postulated migration of genetic groups *PstS7* (Warrior) and *PstS8* (Kranich) from the Himalayas into Western Europe has also had significant implications, resulting in a notable shift in the *Pst* population in Western Europe (44, 46). For YR, both wind and human-related movements contribute to its rapid global spread across continents.

Wheat blast. WB is both windborne and seedborne; the former facilitates local dispersion, whereas the latter accounts for both local and long-distance spread. It has been confirmed that the *MoT* isolates from Bangladesh were introduced from South America via the international wheat trade (16). The recent incursion in Zambia further underscores the risk of intercontinental disease spread, emphasizing the importance of quarantine measures to prevent the introduction of *MoT* (109) (**Figure 4**). Mottaleb et al. (82) predicted that under favorable climatic conditions, 17% of

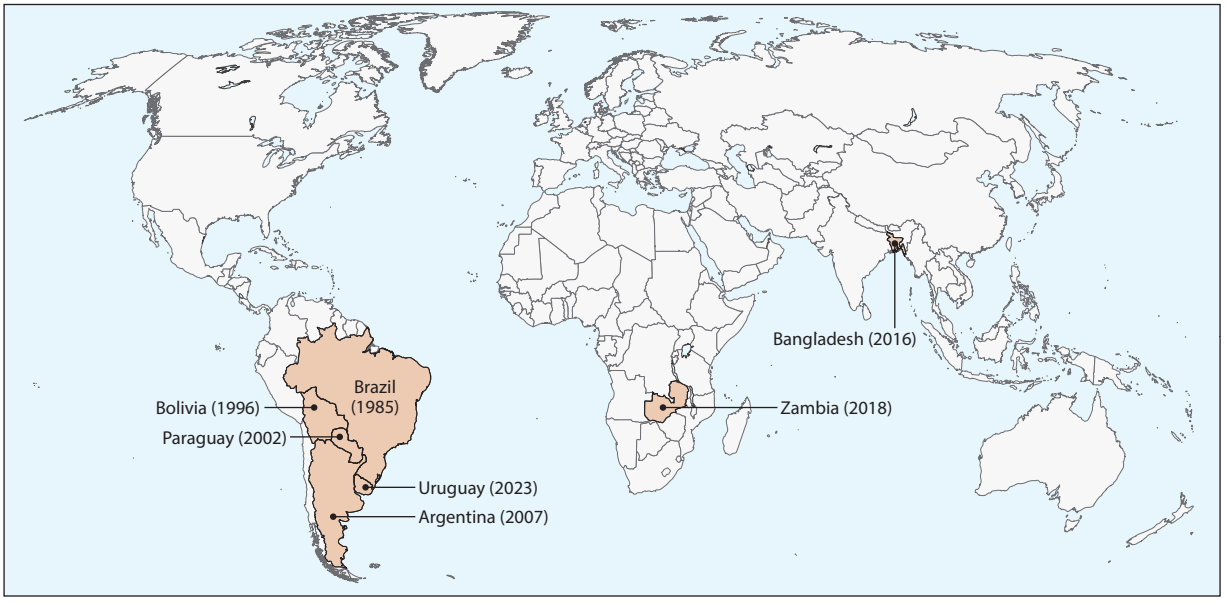

Figure 4

Intercontinental and intracontinental spread of wheat blast through seedborne and airborne mechanisms (99).

the wheat-growing area in South Asia could be at risk, potentially resulting in an annual loss of 0.88 million tons of production. Therefore, it is crucial for countries vulnerable to this disease to carefully design and enforce seed entry and quarantine regulations to restrict the import of seeds and grains from endemic regions.

Fungicide Resistance in Pathogen Populations

Unlike other pathogens, rusts have historically been considered to pose the lowest risk for developing fungicide resistance or insensitivity (9); however, a high frequency of fungicide resistance-associated mutations in *Pst* has been documented (19). In China, resistance to the fungicide Triadimefon in *Pst* isolates has been observed (128), and, more recently, fungicide insensitivity has been reported in barley LR, wheat LR, and barley grass YR fungi in Australia (88). These findings serve as a reminder of the remarkable ability of rust pathogens to adapt and circumvent both genetic resistance and chemical control strategies.

Fungicide resistance has also been a significant issue for other wheat pathogens, such as Septoria tritici blotch (STB) fungus. This pathogen rapidly adapted to several fungicides, including quinone outside inhibitors (QoIs) like strobilurins, demethylation inhibitors (DMIs) such as azole fungicides, and succinate dehydrogenase inhibitors (112). For FHB, reduced fungicide efficacy has been widely reported, particularly with benzimidazole fungicides in China and azole fungicides in Europe and the United States. *Fusarium* species can develop resistance through mutations and copy number variation in target genes like *CYP51*, which encodes sterol 14α-demethylase, the enzyme targeted by triazoles. Additionally, *Fusarium* spp. can deploy efflux pumps to expel fungicide molecules, further diminishing their effectiveness (79).

Similarly, the WB pathogen *MoT* has developed resistance to key fungicides, particularly in Brazil, where blast control heavily relies on fungicide applications due to high disease pressure. Resistance, especially to strobilurins (QoI fungicides), has emerged through mutations in the

cytochrome b gene, rendering these fungicides ineffective. Triazole fungicides have also shown declining effectiveness (113, 114).

To combat fungicide resistance, it is essential to reduce reliance on chemical control by increasing varietal resistance and rotating fungicides, including using mixtures that contain different modes of action. For example, QoI fungicides are often applied in combination with DMI fungicides to minimize the selection pressure for QoI resistance (91).

Replacement of Susceptible Varieties

Susceptible varieties are a key driver of disease epidemics, and replacing older, vulnerable varieties with newer ones, ideally those with durable resistance, is crucial for effective disease control. However, in many parts of the world, varietal turnover rates are low, and outdated varieties persist in farmers' fields. These old varieties often serve as reservoirs for the buildup of inoculum for wheat rusts and other diseases. Tracking varietal use and turnover reliably is challenging, although the increasing use of DNA fingerprinting is now yielding more accurate results (65).

Recent studies on wheat varietal adoption using DNA fingerprinting in Ethiopia (41) and Nepal (28) offer insights into the challenges and progress of varietal replacement. In Nepal, during the 2018–2019 wheat season, it was found that 38% of the wheat area was planted with varieties released 20 years ago, and old, decommissioned varieties (often phased out due to high susceptibility to wheat rusts) were still being grown by 8% of farmers. Conversely, a relatively recent and rust-resistant variety, 'Vijay', covered approximately 20% of the wheat area. Older varieties were more prevalent in the less accessible hill regions with no formal seed system, whereas newer varieties were more common in the plains, where access to seed companies was better. The continued presence of old, susceptible varieties in hill areas remains a major driver of recurrent YR outbreaks in Nepal.

In Ethiopia, disease pressure from both SR and YR has driven varietal turnover, with recurrent epidemics prompting farmers to replace popular but susceptible varieties (51, 85). The DNA fingerprinting study provided strong evidence that newer, rust-resistant varieties like 'Kakaba' and 'Danda'a' were being grown on a large scale and spread across wheat-growing regions (41); however, older YR susceptible varieties like 'Kubsa' persisted in Ethiopian fields as farmers still valued its other characteristics.

Breeding Complexity and Capacity

Breeding programs must continuously achieve genetic gains across multiple traits to develop future varieties that can meet the demands of a growing population, evolving markets, and consumer needs while mitigating the effects of climate change. The success of a breeding program, whether public or private, is measured by its ability to consistently produce competitive varieties and capture a significant share of the market. This typically requires a collaborative effort involving scientists from various disciplines, which often is lacking.

Many wheat breeding programs in the Global South are small and resource constrained. As a result, the International Wheat Improvement Network, managed by CIMMYT, continues to serve as a crucial source of improved parental lines and new varieties (68, 105). Although most resistance genes in wheat and their associated molecular markers are publicly available, accessing germplasm that carries some of the more recently identified resistance genes has become increasingly difficult because of intellectual property rights or restrictions imposed by institutions or funding agencies. The exchange and utilization of diverse resistance sources are essential for managing diseases, particularly those caused by fast-evolving pathogens (62).

Wheat genetic resources have proven to be a valuable source of new resistance genes. However, only a limited number of research programs—primarily in the Global North—have maintained

the capacity to identify, harness, and transfer these genes into wheat. Strengthening the capacity of such programs in the Global North is vital to ensuring a continuous supply of new and diverse resistance genes as global public goods.

GLOBAL STRATEGIES TO SAFEGUARD WHEAT FROM DISEASES

Enhancing the Knowledge, Diversity, and Durability of Disease Resistance

Significant progress has been made over the years in advancing knowledge of resistance genes, their diversity, and the durability of resistance to wheat diseases. These advancements have been crucial for breeding resistant varieties and safeguarding yields. Below, we summarize key developments for rusts, FHB, and WB.

Rust diseases. Rust resistance phenotyping at the seedling stage in the greenhouse and in the adult plant stage in field environments has greatly facilitated the precise characterization of numerous resistance genes. The hexaploid nature of wheat has allowed for the transfer of chromosome segments carrying additional resistance genes from related species and genera. These efforts have resulted in the formal designation of 67, 84, and 86 resistance genes conferring resistance to SR, LR, and YR fungi, respectively (30). Over a third of these genes, including 38 for SR, 45 for LR, and 23 for YR, have been sourced from 21 wheat-related species and genera (62).

Most cataloged genes are race-specific (*R* genes), often providing moderate to high levels of resistance at all stages of development to fungal races that possess corresponding avirulence alleles. Of the 33 cloned wheat rust *R* genes, most belong to the nucleotide-binding site–leucine-rich repeat family. Other genes such as *Yr15* and *Yr36*, transferred to wheat from *Triticum turgidum* ssp. *dicoccoides* (emmer wheat), belong to the tandem kinase-pseudokinase family or possess a kinase and a putative START lipid-binding domain (25, 63, 110). Although deploying *R* genes individually often leads to the rapid emergence of virulent races and resistance breakdown, certain *R* genes have remained effective for longer periods and their overutilization increases genetic vulnerability, as observed with the SR resistance gene *Sr31* (101). Deploying diverse combinations of effective *R* genes is a better strategy, and both gene-specific and linked molecular markers for several genes are now available to aid their selection.

Race-nonspecific resistance, typically effective during the postseedling growth stages, is often referred to as APR. This form of resistance is prevalent in wheat and is governed by quantitative trait loci (QTL), some of which have been characterized by their encoding genes (100). In a recent study, Tong et al. (110) compiled a genomic atlas of 920 rust resistance QTL and genes based on data from 170 publications over the past two decades. They mapped these across the 21 wheat chromosomes using the latest wheat reference genome (IWGSC RefSeq v2.1), identifying high-confidence regions harboring 83 YR, 43 LR, and 28 SR QTL, including 26 regions with pleiotropic effects on multiple rust diseases.

Although individual APR genes and QTL provide only modest to intermediate levels of partial or slow rusting resistance, combining 4 to 5 of these genes can achieve near-immune levels of durable resistance (102, 104). Two cloned pleiotropic APR genes, *Lr34/Yr18/Sr57/Pm38* and *Lr67/Yr46/Sr55/Pm46*, confer partial resistance to all three rusts and powdery mildew, mediated by adenosine triphosphate–binding cassette and altered hexose transporters, respectively (66, 80). Another widely deployed pleiotropic APR gene is *Lr46/Yr29/Sr58/Pm39*, which is used in both hexaploid bread wheat and tetraploid durum wheat (67, 100). The SR APR gene *Sr2*, transferred to bread wheat from emmer wheat, also provides partial resistance to YR (*Yr30*), LR, and PM (*Pm48*) (100, 122). The use of these pleiotropic APR genes, in combination with *Lr68* and other QTL, has contributed to stabilizing LR resistance in CIMMYT-derived bread wheat varieties across Asia, Africa, and Latin America, with good progress toward achieving

durable resistance to SR and YR (7, 100, 101). Gene-based markers for the two cloned genes and tightly linked markers for others are facilitating their verification and marker-assisted selection (MAS).

Fusarium head blight. Resistance to FHB is typically inherited quantitatively, with no immunity present, and limited resistant sources. Resistance genes and QTL have been identified across all wheat chromosomes, although the majority have minor effects. Nine QTL with larger effects have been designated as *Fhb* resistance genes (*Fhb1* through *Fhb9*) (123). To date, only two genes, *Fhb1* and *Fhb7*, have been cloned, and functional markers for these are available. However, the lack of diagnostic markers for the remaining genes/QTL significantly limits the effectiveness of MAS in breeding for FHB resistance (12, 125). Resistance to DON is quantitative, too, and the genes/QTL for DON are largely overlapping with those for FHB (12). Nevertheless, a few QTL that were exclusively associated with DON have been reported (35), warranting additional research on DON resistance.

Wheat blast. Both *R* genes and QTL have been identified for WB, with *R* genes emerging from seedling studies (e.g., *Rmg1* through *Rmg9*), and QTL from field experiments at adult plant stages (36). Among these, the $2N^{v}S$ translocation (also referred to as 2NS/2AS or 2NS) is the only one with stable, major phenotypic effects and has been widely used in all WB-affected regions. However, its resistance is becoming less effective against new *MoT* isolates. Regarding *Rmg* genes, most do not confer head blast resistance at higher temperatures. Some, like *Rmg8*, have shown promising effects at the heading stage but need validation in large-scale field trials (36).

Precision Phenotyping Platforms

The expression of resistance to diseases and pests is strongly influenced by environmental conditions, disease intensity, and the diversity, virulence, and aggressiveness of pathogen populations that vary across geographical regions. Breeding programs must also anticipate changes in pathogen populations or incursions of new diseases and take preemptive measures. Well-structured and well-resourced breeding programs perform resistance selection and phenotyping using desired pathogen races under artificially created epidemics and uniform disease pressures. Phenotyping in controlled field environments is especially important when selecting for quantitative APR, which is effective across wide geographical regions, diverse environments, and varied pathogen populations.

To build better, more diverse, and more stable resistance to significant diseases and pests, CIMMYT and international partners have established a global network of precision phenotyping platforms (PPPs) representing key environments, such as disease hotspots and future-climate analog sites (**Supplemental Figure 1**). These platforms serve as vital hubs for germplasm exchange, breeding, capacity building, and international collaboration. The PPP concept was initiated to facilitate the precise characterization and selection of resistance to pathogens or their critical biotypes, such as the Ug99 races of SR and WB fungi, which are absent in Mexico, where CIMMYT's international wheat breeding program is based.

Supplemental Material >

The first formal PPP became fully operational in 2008 at Njoro, Kenya, to improve resistance to *Pgt* races belonging to the Ug99 lineage and diverse *Pst* races prevalent in eastern Africa. CIMMYT's breeding program utilized this platform to shuttle segregating populations between Mexico and Kenya, which helped build complex APR to both rusts and enrich breeding germplasm with diverse *R* genes (101). Another PPP for characterizing and selecting resistance to durum wheat–specific *Pgt* races was established at Debre Zeit, Ethiopia. Phenotyping data from Kenya and Ethiopia have been instrumental in the release and promotion of resistant varieties in various

countries across Africa and Asia, as well as in the identification of numerous *R* genes and QTL (8).

For WB, three platforms have been established—two in Bolivia (Quirusillas and Okinawa) and one in Bangladesh (Jashore)—representing different geographical regions with distinct cropping cycles. The screening capacity at each location is approximately 8,000 plots, generating high-quality phenotypic data. This has greatly facilitated WB resistance breeding, germplasm screening, and genetic studies (36).

Utilizing *R* Genes Using Conventional and Molecular Approaches

The selection of combinations of major *R* genes for rust fungi remains challenging despite the availability of functional or closely linked molecular markers. Breeding programs often struggle to develop successful cultivars containing multiple *R* genes that are effective against the predominant pathogen populations. Typically, molecular markers are used to genotype parents for targeted crossing, and the best selections of progeny are genotyped after yield phenotyping. Segregating populations in different generations undergo selection and culling in fields under natural or artificial epidemics, with MAS limited to only a few targeted populations. In breeding programs that have adopted speed breeding, disease resistance screening is often deferred until advanced generations, further reducing the likelihood of identifying progeny with the desired combination of multiple *R* genes alongside other necessary traits.

With advances in sequencing technology, a series of high-density single nucleotide polymorphism (SNP) arrays have been developed for gene cloning and genomic selection in wheat. Examples include the Illumina Wheat 9K and 90K iSelect SNP genotyping arrays (15, 117), the Axiom® Wheat 660K SNP array and the Axiom® HD Wheat genotyping (820K) array (121), the Wheat 50K *Triticum* TraitBreed array (94), and the *Triticum aestivum* Next Generation array (13). Although these SNP arrays have been widely used for mapping resistance genes (50), they are not cost-effective for routine use in wheat breeding because of their high cost and limited diagnostic value for *R* genes.

To support MAS in wheat and other food crops, CGIAR's shared services platform offers a low-density, cost-effective genotyping service with rapid turnaround through Intertek, Sweden. This service is based on Kompetitive Allele Specific PCR (KASP) markers. Markers for various genes and QTL associated with resistance to wheat diseases, pests, and other traits are available for MAS. Additionally, CIMMYT's wheat breeding program has made significant progress in developing a low-cost genotyping service with DArTAG-2 (29), which integrates 156 gene-based SNPs/indels and 312 QTL-associated SNPs, including markers for resistance genes and QTL. This platform is being used for genomic selection, incorporating information on the likely presence of resistance genes and QTL. However, further optimization of about half of the markers is needed for diagnostic accuracy. Despite these advancements, the logistics and costs of genotyping large numbers of plants or fixed lines remain impractical and beyond the capacity of most wheat breeding programs.

An approach being employed by CIMMYT's wheat breeding program is the rapid trait integration strategy. This strategy involves incorporating different resistance genes into high-value parents, new varietal candidates, and recently released varieties that are already resistant to the targeted disease. The use of speed breeding facilities and MAS allows for the completion of trait integration through 4–5 backcrosses with the recurrent parent, achieving homozygous progenies with resistance genes within 2 to 3 years. In the absence of linkage drag associated with the targeted resistance gene, this approach has the potential to ensure that the derived variety retains a similar grain yield to the recurrent parent while providing resistance through multiple *R* genes.

Utilizing Quantitative Adult Plant Resistance to Achieve Resistance Durability

The utilization of genetically complex APR is undoubtedly the key to breeding wheat varieties with durable rust resistance, essential for protecting the livelihoods of millions of farmers, especially smallholders, across Asia, Africa, and Latin America. This approach helps mitigate the evolution and spread of new, virulent races of rust pathogens. Because the resistance provided by individual APR genes, such as the pleiotropic genes *Lr34/Yr18/Sr57/Pm38*, *Lr46/Yr29/Sr58/Pm39*, and *Lr67/Yr46/Sr55/Pm46*, is only partial and exhibits small to intermediate effects, breeding programs must focus on selecting diverse combinations of four or five APR genes. This strategy aims to achieve higher resistance levels and significantly reduce crop losses (102, 104).

Employing multipathogen APR genes and QTL simplifies the selection process for resistance to multiple pathogens, as fewer genes are required for pyramiding. The distinct resistance mechanisms and interactions among APR genes further reduce opportunities for mutation and selection within pathogen populations, enhancing resistance durability. Maintaining QTL diversity in breeding populations is essential in case certain QTL prove to be race-specific and lose effectiveness upon deployment. The ongoing nature of breeding allows for the replacement of these QTL with other effective APR options present in the breeding population.

For more than four decades, rust research at CIMMYT has focused on characterizing, elucidating, and selecting diverse APR genes within breeding populations. This work has revitalized research on APR both within and beyond CIMMYT while also supplying elite lines with APR to national partners, leading to the release and widespread cultivation of numerous varieties (8). Although breeding for quantitative APR can be cumbersome initially, the additive and often pleiotropic effects of multiple APR genes facilitate the simultaneous selection of plants with high resistance levels and desirable agronomic and grain traits (100, 104). Once progress has been made, most parental lines used to initiate new breeding cycles possess at least moderate resistance and often share some common APR genes. This enables the selection of progeny with similar, and often superior, resistance levels because of transgressive segregation. Phenotypic selection and the deployment of quantitative APR against the powdery mildew fungus in breeding programs across the United Kingdom and other northern European countries have resulted in durable resistance, despite limited knowledge of the specific genes contributing to this resistance (10).

The release and widespread cultivation of CIMMYT-derived varieties with moderate to high levels of APR have stabilized the LR situation in Mexico and many other countries prone to LR for more than two decades. In 2008, a Mexico–Kenya shuttle breeding scheme was initiated to identify and enhance the frequency of resistance to Ug99 races within breeding materials. This approach emphasized building APR based on *Sr2* and other genes/QTL to achieve durable resistance. As a result, APR gene frequencies were significantly increased alongside high yield potential through field-based selection (7). The presence of various aggressive *Pst* races in Kenya further supported the selection of effective APR gene combinations for YR in the East African environment. Wheat varieties with APR to both SR and YR that have been released and cultivated in Ethiopia include ‘Kakaba’, ‘Danda’a’, ‘Kingbird’, and ‘Deka’. The first two, released in 2010, now account for about 40% of Ethiopia’s wheat-growing areas and continue to perform well against SR and YR, unlike other *R* gene–protected varieties such as ‘Digalu’, which have succumbed to new *Pgt* and *Pst* races (41).

Progress and Prospects of Breeding Resistance to Fusarium Head Blight and Wheat Blast

Breeding for FHB resistance in high-yielding, semidwarf wheat has a shorter history compared to rust resistance, primarily because of its significance being limited to specific regions in a few

countries, such as China and South America. At CIMMYT, breeding efforts began in 1985 to transfer FHB resistance from Chinese sources (37), which is why Chinese parents are often included in the pedigrees of CIMMYT's FHB-resistant lines. Increased FHB severity and associated losses in the United States during the 1990s heightened the urgency for FHB resistance breeding and research. Continuous selection using phenotyping, MAS, and a combination of both over two decades has led to improved resistance conferred by genes and QTL from both native and exotic Chinese sources; however, there has been no clear trend toward the preferential utilization of a gene or QTL (27). Similarly, breeding programs in FHB-affected regions of China have produced competitive varieties possessing *Fhb1* and other genes/QTL, although these varieties generally exhibit only moderate resistance levels (129).

Developing varieties with high yield, strong resistance, and low grain toxins remains challenging worldwide. This difficulty is largely due to the need to pyramid multiple genes and QTL, all of which typically have minor to moderate effects when present individually under high disease pressure. Furthermore, phenotyping for FHB resistance and toxin levels is cumbersome and costly and requires multiple seasons of screening.

The *Fhb1* gene was utilized by CIMMYT initially but was gradually phased out because of its repulsion linkage with *Sr2*, which was heavily employed to restore durable SR resistance to mitigate threats from Ug99 and other *Pgt* races. Recognizing the importance of *Fhb1*, researchers developed recombination lines with *Fhb1* and *Sr2* in coupling linkage (34). A recent study identified two inhibitors of *Fhb1* that act additively; the presence of either inhibitor significantly diminishes the effect of *Fhb1*, whereas both substantially mask the gene's effectiveness and complicate resistance selection (70). Using flanking markers, CIMMYT lines carrying *Fhb1* and lacking the two inhibitors have been identified and are being used as resistance donors in crosses aimed at improving FHB resistance. Genetic studies and haplotyping of FHB-resistant lines revealed that a QTL on chromosome 2DL is the most common native QTL with a significant effect on FHB resistance within CIMMYT germplasm (87, 130). This QTL is present in nearly 20% of CIMMYT's advanced breeding lines and is being stacked with *Fhb1*/*Sr2*. Pyramiding additional resistance genes such as *Fhb5* and *Fhb7* through marker-assisted backcrossing is also in progress, which is expected to enhance FHB resistance given the quantitative nature of these resistance QTL and genes.

Annually, a panel of 40–50 breeding lines is selected based on multiyear FHB phenotyping, yield, and agronomic traits, designated as the FHB Screening Nursery. This panel is distributed to various countries where FHB is a significant issue, allowing for further field experiments and local utilization when promising resistance is identified (38).

The utilization of the $2N^VS$ translocation has significantly advanced breeding efforts against WB. This translocation has been frequently observed in recent CIMMYT germplasm, as it provides broad resistance against rusts, nematodes, spot blotch, and lodging, and enhances yield potential (22, 54). 'Milan' is a notable CIMMYT variety featuring the $2N^VS$ translocation, extensively used across South America. Derivatives of Milan, such as 'Sausal CIAT', 'CD 116', and 'Canindé 1', have been released in regions of Bolivia, Brazil, and Paraguay affected by WB (64). 'BARI Gom 33', a zinc-biofortified, blast-resistant variety released in Bangladesh in 2017, exemplifies another successful application of the $2N^VS$ translocation in breeding (42). All recent wheat varieties released in South Asia and Zambia that exhibit high WB resistance carry this translocation (99). However, excessive reliance on the $2N^VS$ translocation is increasing its vulnerability to new *MoT* isolates because of strong directional selection, with virulent strains against the $2N^VS$ translocation already reported in South America (21). This situation underscores the urgent need to identify new resistance sources.

Non-2N^{V}S sources of WB resistance are scarce in both wheat and its relative species, particularly those exhibiting high levels of WB resistance (99). CIMMYT has identified some non-2N^{V}S materials that show moderate resistance in both field and controlled conditions; however, their progenies exhibit typical quantitative segregation for WB resistance in mapping studies, indicating a lack of major QTL. *Rmg8* and *RmgGR119* are being transferred to elite lines with promising results for *Rmg8*; however, the MAS for *RmgGR119* is hindered by the absence of markers and the lack of disease presence in Mexico. Additionally, efforts are underway to eliminate nonhost susceptibility genes, *rwt3* and *rwt4*, to mitigate the risk of wheat varieties being infected by non-*MoT* pathotypes of *M. oryzae* and ensure optimal expression of *Rmg8*, as its resistance can be masked by *rwt4* (49).

Genomic Selection for Disease Resistance Breeding

Quantitative disease resistance is key to developing durable APR in wheat, but traditional MAS has serious limitations due to the lack of molecular markers for most genes/QTL associated with it. Genomic selection offers a solution by using genome-wide marker data to predict breeding values based on the genetic profiles of plants, known as genomic estimated breeding values (39). Unlike MAS, genomic selection integrates genotypic and phenotypic data into a predictive model, improving selection accuracy and efficiency. Genomic selection can support resistance selection, shorten the breeding cycle length, and reduce costs associated with extensive phenotyping particularly when applied for multiple traits simultaneously or when the cost of phenotyping exceeds genotyping. In genomic selection, a training population of genotyped and phenotyped individuals is used to develop predictive models, which are then applied to a selection population to select the best candidates.

A key aspect of implementing genomic selection in breeding programs is understanding trait prediction accuracies in different populations and environments, and numerous studies have reported cross-validation prediction accuracies for disease resistance in wheat (**Figure 5**, **Supplemental Table 1**). Under the within-population scenario for different cohorts of CIMMYT advanced breeding lines, the prediction accuracies ranged from 0.41 to 0.52 for LR, 0.41 to 0.71 for YR, 0.43 to 0.64 for SR, 0.38 to 0.53 for STB, 0.42 to 0.56 for tan spot, 0.34 to 0.61 for spot blotch, and 0.66 to 0.8 for WB (55–59, 95). These prediction accuracies are similar to those reported in other studies for these and other diseases (**Supplemental Table 1**). The promising prediction accuracies for key wheat diseases across various breeding populations suggest that disease responses in untested wheat genotypes can be effectively predicted within different populations and environments, allowing breeders to save resources by sparsely testing some lines and using data from related lines to predict others within that environment.

Supplemental Material >

Although cross-validation prediction accuracies are only informative for integrating genomic selection in selections within populations and environments, forward prediction accuracies are critical from a more practical genomic selection deployment context in breeding programs, and few studies have looked at them. The forward prediction accuracies for resistance to SR, YR, STB, and spot blotch in CIMMYT's breeding lines were 0.6, 0.37, 0.29, and 0.37, respectively (57). Forward prediction accuracies for FHB severity varied across studies. Larkin et al. (69) reported accuracies ranging from 0.08 to 0.16, whereas Moreno-Amores et al. (81) and Zhang et al. (124) observed higher accuracies of 0.36 and 0.59, respectively. These studies demonstrate that although forward predictions show promise for some diseases in wheat, they are challenging for others because of the complex genetic architecture and genotype × environment effects. Other factors influencing prediction accuracies include trait heritabilities, the size and composition of the training population, marker densities, marker platforms, and choice of prediction models.

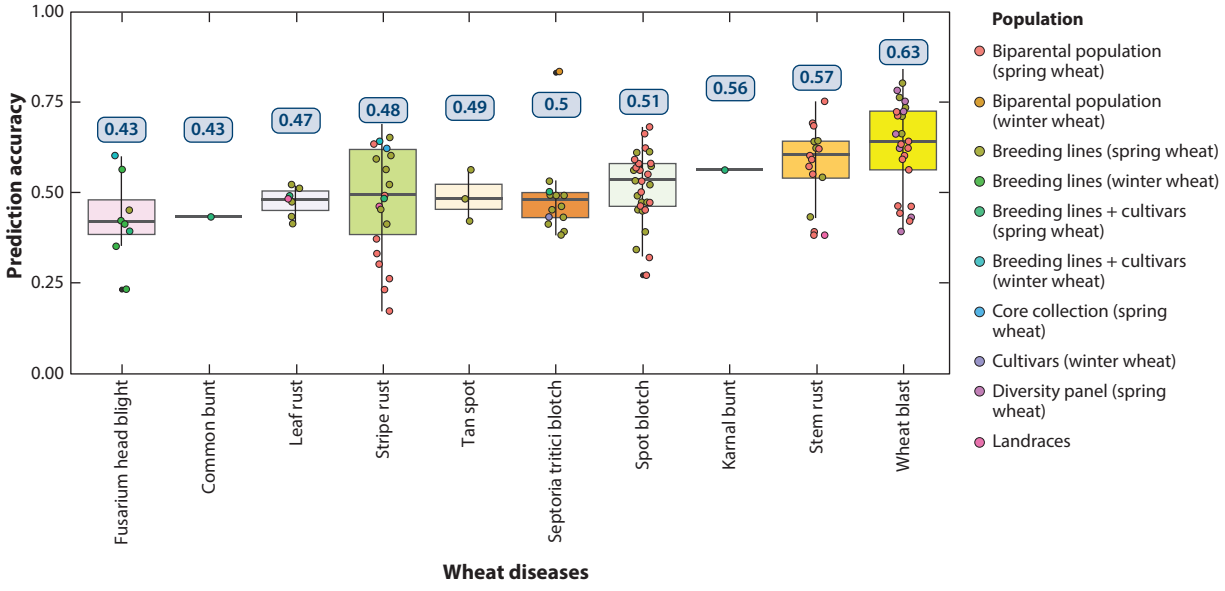

Figure 5

Prediction accuracies for wheat diseases. The highest prediction accuracies from a genomic prediction model for resistance to diseases based on various research publications listed in **Supplemental Table 1**.

Integrating genomic selection into disease resistance breeding also presents several challenges. First, although theoretical models indicate the potential benefits of genomic selection, empirical studies remain limited on its advantage and relative gain over phenotypic selection and its long-term effect on variance and inbreeding (40, 96). Second, genomic selection cannot help in selecting for resistance to novel pathogen races, as genomic prediction models rely on training datasets that may not encompass emerging threats. Additionally, it is a resource-intensive process, as it necessitates creating and updating multiple training populations for each pathogen race phenotyped in multiple environments. Another challenge lies in predicting resistance in novel genetic sources, which requires these alleles to be at sufficient frequencies in the training population for effective utilization. Furthermore, the stage of the breeding cycle at which genomic selection can be implemented needs more investigation. Although early stages involve segregating generations and a significant cost associated with genotyping a large number of individuals, advanced stages often lack sufficient variation because of high selection pressure applied in earlier stages by the breeding programs. These complexities underscore the importance of empirical research and innovation to successfully integrate genomic selection into wheat breeding programs.

Supplemental Material >

Genetic Engineering and Genome Editing

Recent advances in genetic engineering and genome editing have opened new avenues for accelerating the development of disease-resistant wheat varieties. To address the challenges associated with the simultaneous selection of multiple *R* genes in wheat, effective against *Pgt* races from the Ug99 lineage and others, Luo et al. (74) introduced a multigenic resistance strategy. They developed a transgene cassette containing five resistance genes (*Sr22/Sr35/Sr45/Sr50/Sr55*) inserted at a single locus and demonstrated that at least four of the five genes were functional. These wheat lines exhibited strong resistance in field trials, with effectiveness against various *Pgt*

races, including Ug99, in greenhouse studies. The monogenic inheritance of this multigene locus simplifies its selection in breeding programs, whether through field-based phenotyping or MAS. This approach can be extended to develop multigenic cassettes for cloned resistance genes against other diseases, facilitating their use in breeding efforts. Additionally, genes encoding antifungal proteins, such as chitinases, have been successfully introduced into wheat, resulting in reduced FHB severity and lower mycotoxin accumulation (98).

Gene editing platforms like the CRISPR-Cas9 and TALEN systems have emerged as revolutionary tools for precise genome editing, allowing for targeted disruptions or modifications of genes involved in disease susceptibility or resistance. Several susceptibility genes have been edited in wheat with the first being the *TaMlo* gene, which encodes a protein that facilitates powdery mildew infection. Knockouts of *TaMlo* across all three wheat genomes using CRISPR-Cas9 conferred broad-spectrum resistance to powdery mildew in wheat, eventually without detrimental agronomic effects (71, 118). Other gene editing (33, 60, 61, 72, 116, 126) or TILLING (20, 48) knockouts collectively have given resistance to YR, LR, wheat yellow mosaic virus, and further powdery mildew resistance, highlighting the diversity of wheat pathogens that this approach can be applied to.

Despite the potential of genetic engineering and genome editing in enhancing wheat disease resistance, several challenges remain. Regulatory barriers, public acceptance of genetically modified organisms, and concerns about the off-target effects of genome editing must be addressed. Additionally, the durability of resistance conferred by these technologies needs to be evaluated under field conditions, where pathogen populations can evolve rapidly. Positive developments in favor of GM wheat have been the acceptance of simple SDN1 gene editing events not requiring regulation in some countries (1) and approval being granted in Argentina and Brazil for the cultivation of HB4® wheat varieties, which carry a drought-responsive *Habb4* transgene from sunflower. These HB4® varieties have also been approved for consumption in approximately 10 countries (31). This recent progress, combined with the value of targeted traits and their natural availability, should further support the adoption of these technologies in the development of disease-resistant wheat varieties.

Rapid Molecular Diagnostics for Pathogen Surveillance and Monitoring

Although conventional pathotyping of wheat rusts through seedling testing of differential sets under controlled conditions remains an invaluable diagnostic technique, molecular diagnostics are increasingly important in surveillance programs for wheat pathogens. Recent advances in genomics technologies are providing new opportunities for the rapid development of molecular diagnostics for pathogen surveillance and monitoring, particularly for *Pgt* and *Pst*. A core set of 17 SNP markers has been established that accurately diagnoses a diverse range of *Pgt* isolates at the genetic group level (107). This rapid SNP assay is now routinely applied to ethanol-killed *Pgt* samples collected during surveys in Africa and Asia.

The development of field pathogenomics, based on RNA-seq data obtained directly from field-infected plants, has marked a significant advancement in diagnostics relevant to surveillance programs for *Pst* lineages in UK wheat (46). The advent of low-cost, portable nanopore sequencing using MinION technology developed by Oxford Nanopore has further facilitated the implementation of point-of-care diagnostics for wheat rusts in resource-constrained settings.

The mobile and real-time plant disease (MARPLE) diagnostic system (93) is the first operational system to utilize nanopore sequencing to determine clade-level identities for both *Pst* and *Pgt* from infected field samples within 2–3 days. Fully mobile and deployable in field settings, MARPLE is tailored for use in low-resource environments. It employs an innovative

methodology to rapidly diagnose complex fungal pathogens with large genomes. Originally developed for *Pst*, this methodology has now been successfully adapted for *Pgt* as well.

A set of highly variable genes that differentiate the main genetic groups of *Pst* has been identified from a comprehensive collection of genomic data from global yellow rust isolates. Targeted amplicon resequencing of this differential gene panel (approximately 200 genes) using MinION nanopore sequencing and a custom-built open-source bioinformatics pipeline provides an accurate diagnostic tool. This technique can easily accommodate additional key genes and be applied to other pathogens. Notably, one gene already included in the panel, *cyp51*, is critical for fungicide resistance, potentially allowing the MARPLE system to monitor emerging fungicide resistance in *Pst* isolates. MARPLE has been successfully deployed in Ethiopia, Kenya, Nepal, and South Africa.

Accelerating Varietal Diffusion and Replacement

Wheat is a self-pollinated crop that requires large seed rates for successful crop establishment. This limits the capacity of organized seed sectors and often favors the use of farm-saved seeds or the acquisition of low-cost seeds from informal seed producers. Reducing the lifespan of a variety is crucial to continuously delivering genetic gains in yield, disease resistance, and other traits while also promoting genetic diversity (5, 53).

To accelerate varietal turnover, several measures and policies have been implemented, including continuous releases of new varieties, fast-tracking variety approvals when necessary, halting seed production of susceptible varieties, pre-release seed multiplication of candidate varieties, engaging many small- and mid-sized seed producers—including progressive farmers—and conducting awareness and promotional campaigns. These efforts have contributed to improving varietal turnover in Ethiopia (108) and other countries where wheat breeding is primarily conducted by public sector institutions.

After WB was first detected in Bangladesh in 2016 (75), the popular variety 'Bari Gom 26' was found to be highly susceptible. In response, the Bangladesh Wheat and Maize Research Institute discontinued breeder seed production of 'Bari Gom 26' and promoted the resistant variety 'Bari Gom 33'. Although 'Bari Gom 33' covered less than 4% of the wheat area in the 2018–2019 season (26), by the 2023–2024 season, it had expanded to more than 30% of the wheat area. Aggressive seed-scaling strategies have also accelerated the adoption of new varieties in India (18) and Pakistan (32), and these strategies can be applied by other countries to speed up varietal replacement.

CONCLUSION

Over the past decade, wheat yields have increased at a healthy pace, and major disease epidemics have been avoided. However, localized crop losses continue to occur as new, more virulent, aggressive, and fungicide-tolerant fungal races evolve and spread to new areas. Wheat breeding programs have made significant strides, delivering multiple disease-resistant varieties with superior grain yields that are continuously replacing susceptible varieties and boosting productivity. Nevertheless, susceptible varieties continue to persist, undermining disease management strategies. The increased availability and use of multiple race-specific resistance genes, through both field-based selection and molecular marker-assisted integration, have contributed to improved resistance. Increased focus on breeding for moderate to high levels of APR, using combinations of additive pleiotropic multipathogen resistance genes and quantitative trait loci, ensures the durability of resistance is enhanced. Cultivation of resistant varieties has reduced the need for harmful chemicals on millions of hectares of wheat crops. Looking forward, genomic selection, genome editing, and

the use of cloned wheat resistance genes in multigene resistance cassettes are expected to further accelerate resistance breeding and provide durable control of major diseases.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

AUTHOR CONTRIBUTIONS

Ravi P. Singh structured the review, contributed to writing, and oversaw overall editing. David P. Hodson contributed to sections on rust epidemiology, fungicide resistance, varietal replacement, and pathogen diagnostics, and developed **Figures 1–3**. Pawan K. Singh and Xinyao He prepared sections related to Fusarium head blight and wheat blast, and developed **Figure 4** and **Supplemental Figure 1**. Caixia Lan, Evans S. Lagudah, Sridhar Bhavani, and Julio Huerta-Espino contributed to sections on rust resistance. Philomin Juliana wrote the section on genomic selection and developed **Figure 5** and **Supplemental Table 1**. Michael Ayliffe contributed to the section on genetic engineering and genome editing. Diane G.O. Saunders contributed to the pathogen diagnostics section. All authors participated in editing and revising the final version of the review.

Supplemental Material >

ACKNOWLEDGMENTS

Some of the research results and information used in this review were generated by the Accelerating Genetic Gain (AGG) in Maize and Wheat Project Grant INV-003439 to CIMMYT; Wheat Disease Early Warning Advisory System (Wheat DEWAS) project grant INV-048345 from the Bill and Melinda Gates Foundation (BMGF) and the Foreign and Commonwealth Development Office (FCDO) to CIMMYT and John Innes Centre; Feed the Future project #AID-OAA-A-13-00051 funded by the United States Agency for International Development (USAID) CIMMYT; and fundings to CIMMYT from the Australian Council of International Agricultural Research (ACIAR), Indian Council of Agricultural Research (ICAR), CGIAR's Applied Breeding Initiative (ABI) and Plant Health Initiative (PHI).

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