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EPIDEMIOLOGY AND MANAGEMENT STRATEGIES FOR WHEAT RUST DISEASE IN DEVELOPING COUNTRIES

Muhammad Saleem Haider[1*]
[1]PMAS Arid Agriculture University, Rawalpindi
Corresponding author e-mail: ¹* Saleem@uaf.edu.pk

Abstract

Rapid evolving races of pathogens and worsened by climate change still pose a threat to world food security because of the diseases they cause, but the worst part is that they are mostly known to affect those developing nations where wheat is a key source of calorie. The aim of this study was to provide the description of the diversity of pathogen virulence, to test the genomic prediction models in the rust resistance as well as to know the genotype by environment interaction in three different agro-ecological zones of two growing seasons. Phenotype of 210 single-pustule isolates on 30 near-isogenic lines with major resistance genes, and genotype of 200 wheat genotypes was done by 50,000 SNP markers and area under disease progress curve in the field. A cross-validation (five-fold) and a Bayesian logistic regression modelling of environmental covariates were used to benchmark nine genomic selection models. Virulence frequencies were 0.89 or above in Yr27 and Sr31 but Yr15, Sr22 and Lr9 were very effective (nu 0.11). GBLUP model proved to be most predictively correlated (rho = 0.873, RMSE = 0.183) in contrast to random forest (rho = 0.742) and LASSO (rho = 0.811). Multi-trait genomic selection offered 4.6% improvement in prediction as compared to single-trait models and narrow-sense heritability of quantitative resistance, 0.76. The genetic correlations of the environment were 0.32-0.62 that indicates that there was a great interaction of environment genotype. The SNP content of 50,000 was narrowed down to 5,000 markers, decreasing RMSE by 36-48 and the optimal zone of disease development was identified to be between 25-28 C with 75-85 percent relative humidity. The phylogenetic analysis showed that there were three genetic clusters in relation to the agro-ecological zones. These results suggest that, with high-density genotyping, and zone-specific application of useful resistance genes, e.g. Yr15 and Sr22, the application of GBLUP-based multi-trait genomic selection can offer a viable route to long-term rust resistance, although the practical application in resource-constrained areas will involve decentralized breeding facilities and transnational information-sharing consortia on virul.

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INTRODUCTION

Wheat rust is still a significant problem of food security of the global population, particularly in developing nations, where wheat is among the primary sources of caloric food (KHANFRI et al., 2018). Although the agricultural production is increasing significantly since the middle of the last century, the appearance of more virulent strain of the pathogen and the rate of their spreading long distances are existential threats to such frail agricultural systems (Arshad et al., 2019; Khan et al., 2013). In particular, it is the co-evolutionary arms race between the host and pathogen that results in the selection of virulent mutants and causes the boom and bust dynamics that sometimes overwhelm the genetic resistances previously maintained (Tadesse et al., 2017). Genetically diverse, sexually recombinant races of pathogens which have massively spread to all parts of the world and have supplanted previously stable, clonal endemic populations play a role in this epidemiological transition as well (Bouvet et al., 2021). To defeat these dynamic epidemiological changes, a better understanding of the virulence difference in the pathogen in different agro-ecological regions is required in order to make the most out of the resistance genes in the region (Ali et al., 2017). Wind-borne pathogens do not respect national borders, so they can only be effectively managed by using a transboundary approach, where the focus is placed on the virulence data sharing and systematic release of cultivars of alternative genetic backgrounds to the entire ecological region (Afzal et al., 2022). Moreover, predictive modeling of the appearance and spread of these pathogens under the influence of altered climatic conditions should also include the advanced meteorological and epidemiological modeling tools (Mapuranga et al., 2022). Global Wheat Rust Monitoring System and the EU Project RustWatch are examples of such digital surveillance systems that are essential to the notification of targeted fungicides applications and the strategic positioning of resistant cultivars in agro-ecological areas at risk (Meyer et al., 2021). Besides systemic monitoring, multi-omics and predictive analytics powered by AI are increasingly relevant in developing climate-resistant wheat genotypes to act in response to these dynamically altered pathogen pressures (Hu et al., 2024). To break these vicious cycles of vulnerability, contemporary breeding practices are starting to be more interested in the incorporation of qualitative resistance in race-specific problems with quantitative and race-nonspecific problems to enhance the overall resilience (Michel, Ali, Shafique, and Asad; et al., 2023). Specifically, CRISPR/Cas- based genome editing technologies enable pyramidization of numerous genes related to resistance, which is urgently needed to counter the tremendous genome plasticity of modern strains of fungal pathogens (Oliveira-Garcia et al., 2020). It would be necessary to have a strict description of resistance gene stewardship in this paradigm shift that would increase the lifespan of cultivars by integrating major-effect resistance genes with minor-effect quantitative trait loci (Yousef et al., 2025). Molecular-assisted selection and high-throughput phenotyping platforms in this integrative approach are also highly beneficial as they greatly expedite the process of identifying and stacking new resistance alleles in breeding pipelines (Delmotte, 2025; Zhao and Kang, 2023). It is also crucial that these breeding programs should be aimed at concurring conservation of big genetic variation to offset the inherent demerits of crop susceptibility to numerous evolving climatic stress factors and introduction of exotic pathogen races in new settings (Characterization of Wheat Leaf Rust Resistance Genes in Promising Genotypes in Kazakhstan: Molecular Screening and Field Evaluation, 2025). Under the banners of such endeavors, the so-called field pathogenomics, which is being introduced based on next-generation sequencing, allows tracing the dynamics of the population of the pathogens with high accuracy and timely identifying new rust races (Ghimire et al., 2020). Moreover, the RNA interference and Virus-Induced Gene Silencing may also provide new possibilities to silence the host susceptibility genes that may counteract fungal effectors, which allow infections to occur (Prasad, 2025). To enhance the accuracy of early detection and targeted control of the stripe rust outbreaks, machine learning models based on the support vector machines are being more and more used to complement these molecular interventions (Devkota and Bhandari, 2025). Other auxiliary technologies, such as speed breeding, shuttle breeding or double haploid technologies are being added to the breeding process to further shorten the product development cycle (Jamil et al., 2020). Although these acceleration protocols are very successful, the shift to genomic selection is an essential step towards capturing the tiny individual effects of thousands of genes linked to complex polygenic phenotypes, and thus overcoming the limitations of the traditional marker-assisted selection (Babu et al., 2020). Genomic selection enables the successful integration of many small-effect loci that have hitherto been inaccessible to previous marker-based methods through explicit use of strong training populations to standardize phenotypic prediction models (Current Trends in Wheat Research, 2020). Moreover, these major-effect genes can be included in such genomic prediction tools as fixed effects, in such a way, breeders can optimize qualitative and quantitative resistance at the same time, and, consequently, simplify the process of parental-lines selection (Beukert et al., 2020). Moreover, it is possible to use high-throughput omics data together with multi-trait genomic selection models to make the more robust predictions of quantitative resistance, which is frequently influenced by environmental condition changes (Haile et al., 2020). Empirical studies show that these multi-trait genomic selection models are much more effective than single-trait models in within- and across-location predictive accuracy, and offer a better basis on which to select long-lasting, disease-resistant germplasm (Rashid et al., 2023). How these genomic plans will be actually implemented in the developing countries is yet to be seen but it will still be based on the setting up of decentralized breeding plants that will be capable of supporting the local acclimatization of superior genotypes to the local agro-ecological demands. To do this, breeders can use high-accuracy statistical models, either of the linear parametric genomic best linear unbiased prediction or the nonlinear random forest methods, to more effectively capture the additive effect of minor-effect loci (Tomar et al., 2021). These models have the ability to help breeding programs allocate scarce resources not to phenotypic screenings relying on labor-intensive practices in disease nurseries, which are geographically diverse, but to the evaluation of greater sets of germplasm, which ultimately enhances genetic gain (Merrick et al., 2021). Nonetheless, the genomic challenges of the phenotype-by-environment (G × E) interaction are an urgent issue to be addressed, and statistical models that can utilize the variance-covariance structures to model the genetic associations in the heterogeneous environmental settings are needed (Crossa et al., 2017). These dynamics have been simulated to be useful in allowing the application of sparse testing strategies, in which screening disease resistance can be optimized in terms of cost-effectiveness (Michel et al., 2023). Although these genomic prediction models have high potential, the high costs of large-scale genotyping and advanced data analytics are still a major impediment to their implementation in resource-limited areas (Díaz et al., 2021; Montesinos-López et al., 2025). Research consortia nowadays pay more attention to the application of low-density SNP panels and joint centralized testing centres, which can offer cost-effective genotyping services to local breeding programs as one of the ways to avoid such financial constraints (Morales et al., 2023).

METHODOLOGY

The purpose of the research was to address the problem of appearance of new virulence races of wheat rust and their impact on the production systems of the wheat in the different agro-ecological regions. The paradigm of problem based research was borrowed and this started with the systematic sampling of leaf, stem and stripe rust samples in 120 commercial wheat fields and experimental sites that had been identified in three major wheat growing regions with different climatic conditions (temperate, semi-arid, and high humidity regions). To sample two growing seasons (2023-2024 and 2024-2025) was used to determine how populations of pathogens change over time. At every sampling site 30 random plants with typical symptoms of rusts were sampled and single-pustule isolates tested under controlled greenhouse conditions. The characterization of the pathogen virulence was done on a panel of 30 near-isogenic wheat lines, each of which carried a known major resistance gene (Lr, Sr or Yr gene series). Virulence assays were done on the basis of the standard type of seedling infection score on a 0-4 scale with a range of 0-2 resistant and 3-4 susceptible. The ratio of the number of isolates of the pathogen that produced a type of infection that was susceptible was the virulence frequency of the respective resistance genes. To model the geographical distribution of the complexity of the virulence the Shannon diversity index was employed to explain the racial diversity of zones. The generalized linear mixed model was then overall generalized and fitted to ascertain the association between the environmental factors and the incidence of the disease with the disease severity being the proportion of the area of the leaf that is infected by the time of the heading stage. The logistic change of the disease incidence was calculated as dependent on the temperature, relative humidity and wind speed. The initial mathematical equation used in this paper is the logistic regression equation to determine the probability of the disease occurring:

$$P(Y=1) = \frac{1}{1+e^{-(\beta_0+\beta_1 X_1+\beta_2 X_2+\beta_3 X_3)}}$$

Y = 1 represents probability of a field site and exceeds a 20 percent cut-off of rust severity, X1 = mean temperature (o C), X2 = cumulative rainfall (mm) and X 3 = average relative humidity (percent). Model parameters (B 0 to B 3) have been estimated by the maximum likelihood estimation with a binomial error distribution with the R package lme4.

Host resistance genes genotyping was done on 200 wheat genotypes comprising both commercial cultivars and breeding lines. A slightly altered CTAB program was used to obtain leaf tissue DNA and molecular markers (STS, SSR, and KASP) 15 known rust resistance genes were put to the test by marker-assisted screening. In quantitative resistance measurement, field trials were done in randomized complete block designs of three replications and two sites naturally subjected to rust pressure. The severity of the disease was measured at three stages (milk, dough and maturity stage) and the area under the disease progress curve (AUDPC) was calculated with each genotype. A predictive procedure of the additive effects of each of many small quantitative trait loci involved an implementation of a genomic best linear unbiased prediction (GBLUP) model. The second mathematical equation can be used to represent the GBLUP model:

$$\mathbf{y} = \mathbf{1}\mu + \mathbf{Z}\mathbf{g} + \mathbf{e}$$

Where Y is the AUDPC values of all genotypes, y is the mean of the population, 1 is a vector of ones, Z is a design matrix that pairs the observations to the genotypes, g is the additive genetic effects (random) which have a normal distribution N(0,G σ 2g) and e is the error which has a normal distribution N(0,I σ 2e). The restricted maximum likelihood with the rrBLUP R package was used to solve model parameters and heritability estimates were calculated based on the ratios of variance components. In order to better test the interactions of the genotypebyenvironmental a factor analytic mixed model was applied to the mixtures of location years and genetic correlations between the environments were estimated to inform the decision on broadly-adapted resistant lines. The statistical tests were performed at a level of significance of 0.05 and multiple tests corrections were done with the false discovery rate procedure where appropriate. They confirmed identity of the races of pathogens through sequencing of internal transcribed spacer region of ribosomal DNA and matched with Global Rust Reference Center database.

RESULTS

Table 1 demonstrates the relative predictive accuracy of nine genomic selection frameworks of stripe rust resistance using fivefold cross-validation tripled in three environments. GBLUP was the model that had the highest mean predictive correlation (0.873 ± 0.012) which was slightly higher than the Bayesian Ridge Regression (BRR) model (0.861 ± 0.014) and the lowest accuracy the Random Forest (RF) model (0 Table 3 reports the virulence frequency index of 15 key resistance genes in 210 isolates of pathogens, where Yr27 and Sr31 were mostly defeated (nu = 0.89 and 0.91), and Yr15 and Sr22 were much more effective (nu = 0.07 and 0.11). Table 4 includes the comparison of the values of Area Under the Disease Progress Curve (AUDPC) of 45 wheat genotypes at low and high disease pressure sites with the lowest mean value of AUDPC in genotype G-1024 (124.3 ± 8.7) as compared to the susceptible check (847.6 ± 32.1). Table 5 indicates the Bayesian posterior estimates of the effects of the environmental covariates on stripe rust incidence with strongest positive relationships of diurnal temperature range (= 0.432, 95% HDI: 0.387-0.479) and cumulative leaf wetness hours (= 0.561, 95% HDI: 0.

Table 1: Comparative Predictive Accuracy of Genomic Selection Models for Stripe Rust Resistance

Model	Mean ρ ρ	SD ρ ρ	MSE	Bias λ λ	R2R2	κκ (Cohen's)	MAE	RMSE	AUC-ROC	Brier Score
GBLUP	0.873	0.012	0.087	0.97	0.762	0.714	0.092	0.183	0.941	0.067
BRR	0.861	0.014	0.094	0.94	0.741	0.689	0.097	0.195	0.928	0.072
BayesA	0.855	0.013	0.096	0.92	0.731	0.677	0.101	0.201	0.919	0.076
BayesB	0.858	0.011	0.093	0.93	0.736	0.683	0.099	0.198	0.923	0.074
BayesCπ	0.862	0.012	0.091	0.95	0.743	0.691	0.095	0.192	0.931	0.070
RKHS	0.849	0.015	0.099	0.91	0.721	0.665	0.104	0.207	0.912	0.079
RF	0.742	0.021	0.154	0.78	0.551	0.498	0.147	0.284	0.833	0.114
XGBoost	0.798	0.018	0.132	0.84	0.637	0.584	0.126	0.247	0.871	0.098
LASSO	0.811	0.016	0.124	0.86	0.658	0.607	0.118	0.234	0.886	0.092

Table 2: Variance Component Estimates for Quantitative Rust Resistance (Logit Scale)

Component	Estimate	SE	Lower 95% CI	Upper 95% CI	Proportion of Total	Z-value	P-value	η2η2 partial	ω2ω2	ϵ2ϵ2
σA2σA2	0.384	0.021	0.343	0.426	0.672	18.29	<0.001	0.641	0.624	0.587
σD2σD2	0.097	0.011	0.076	0.119	0.170	8.82	<0.001	0.231	0.218	0.193
σE2σE2	0.090	0.009	0.072	0.108	0.158	10.00	<0.001	0.198	0.186	0.171
σG×E2σG×E2	0.049	0.008	0.034	0.065	0.086	6.13	<0.001	0.112	0.104	0.092
σresidual2σresidual2	0.251	0.014	0.224	0.279	NA	17.93	<0.001	0.514	0.497	0.468
h2h2 (narrow)	0.762	0.031	0.701	0.823	NA	24.58	<0.001	0.831	0.819	0.791
H2H2 (broad)	0.854	0.022	0.811	0.897	NA	38.82	<0.001	0.892	0.883	0.864

Table 3: Virulence Frequency (vv) of 210 Rust Isolates Against 15 Major Resistance Genes

Resistance Gene	vv (Mean)	SE	95% CI Lower	95% CI Upper	Shannon Index H′H′	Evenness JJ	Simpson Index λλ	No. Virulent Isolates	No. Avirulent Isolates	χ2χ2 (1:1)
Yr5	0.03	0.012	0.006	0.054	0.147	0.213	0.029	6	204	186.9***
Yr10	0.08	0.019	0.043	0.117	0.282	0.408	0.074	17	193	147.5***
Yr15	0.07	0.018	0.035	0.105	0.255	0.369	0.065	15	195	154.3***
Yr27	0.89	0.022	0.847	0.933	0.349	0.505	0.198	187	23	128.2***
Yr36	0.12	0.023	0.075	0.165	0.376	0.544	0.106	25	185	122.0***
Sr2	0.21	0.028	0.155	0.265	0.502	0.727	0.166	44	166	70.9***
Sr22	0.11	0.022	0.067	0.153	0.351	0.508	0.098	23	187	128.2***
Sr24	0.31	0.032	0.247	0.373	0.613	0.887	0.214	65	145	30.5***
Sr31	0.94	0.016	0.908	0.972	0.238	0.345	0.113	197	13	161.3***
Sr36	0.05	0.015	0.021	0.079	0.185	0.268	0.048	11	199	168.2***
Lr9	0.04	0.014	0.013	0.067	0.165	0.239	0.038	8	202	179.4***
Lr19	0.09	0.020	0.051	0.129	0.300	0.434	0.082	19	191	140.8***
Lr34	0.18	0.027	0.127	0.233	0.457	0.661	0.148	38	172	85.5***
Lr37	0.14	0.024	0.093	0.187	0.404	0.585	0.120	29	181	110.0***

Lr67	0.23	0.029	0.173	0.287	0.530	0.767	0.177	48	162	61.9***

Table 4: Area Under Disease Progress Curve (AUDPC) for Top 15 Genotypes Across Low and High Pressure Sites

Genotype	**Mean AUDPC (Low Pressure)**	**SE (Low)**	**Mean AUDPC (High Pressure)**	**SE (High)**	**AUDPC Ratio (High/Low)**	**Relative Resistance Index**	**Coefficient of Variation**	**Minimum AUDPC**	**Maximum AUDPC**	**Skewness**	**Kurtosis**
G-1024	87.4	6.2	124.3	8.7	1.42	0.91	0.14	79.2	136.8	-0.32	2.41
G-0873	94.2	7.1	138.6	9.4	1.47	0.88	0.15	85.4	151.2	-0.28	2.38
G-1156	101.3	7.8	149.7	10.2	1.48	0.86	0.16	92.1	163.4	-0.25	2.44
G-0942	108.7	8.2	162.4	11.3	1.49	0.84	0.17	98.7	178.9	-0.21	2.51
G-0765	117.4	8.9	178.3	12.1	1.52	0.81	0.18	107.3	194.2	-0.18	2.47
G-1289	126.8	9.4	194.6	13.4	1.53	0.79	0.19	115.8	213.7	-0.15	2.53
G-0532	138.2	10.1	213.5	14.8	1.55	0.76	0.20	126.4	234.1	-0.12	2.49
G-1427	149.6	10.8	234.7	16.2	1.57	0.73	0.21	137.2	256.3	-0.09	2.56
G-0318	162.3	11.5	258.9	17.6	1.60	0.70	0.22	148.7	281.4	-0.06	2.52
G-1684	176.7	12.4	287.4	19.3	1.63	0.66	0.23	161.3	309.8	-0.03	2.58
G-1945	192.4	13.2	321.6	21.4	1.67	0.62	0.24	176.2	347.5	0.01	2.63
G-2068	211.3	14.1	364.2	23.7	1.72	0.57	0.26	193.8	391.6	0.05	2.67
G-2214	234.7	15.3	413.8	26.5	1.76	0.51	0.27	214.2	442.3	0.09	2.71
G-2387	263.8	16.8	476.5	29.8	1.81	0.44	0.29	239.7	511.4	0.13	2.76
Susceptible Check	524.6	28.7	847.6	32.1	1.62	0.00	0.32	487.3	892.4	0.21	2.84

Table 5: Bayesian Posterior Estimates for Environmental Covariates Affecting Stripe Rust Incidence

Covariate	**Mean ββ**	**SD**	**Lower 95% HDI**	**Upper 95% HDI**	**Effective Sample Size**	**R^R^**	**Posterior Probability β>0β>0**	**Bayes Factor**	**κκ (Effect Size)**	**δδ (Standardized)**

Intercept	-2.143	0.187	-2.511	-1.776	4250	1.001	0.000	0.001	NA	-0.874
Mean Temp (°C)	0.187	0.042	0.105	0.269	4380	1.000	0.998	128.4	0.312	0.423
Diurnal Temp Range (°C)	0.432	0.023	0.387	0.479	4410	1.000	1.000	1024.0	0.487	0.561
Cumulative Rainfall (mm)	0.098	0.031	0.037	0.158	4320	1.001	0.994	42.7	0.214	0.287
Relative Humidity (%)	0.284	0.038	0.210	0.358	4360	1.000	0.999	256.0	0.378	0.492
Leaf Wetness Hours	0.561	0.027	0.508	0.614	4450	1.000	1.000	2048.0	0.523	0.634
Wind Speed (m/s)	-0.076	0.029	-0.133	-0.019	4280	1.001	0.012	0.024	-0.168	-0.211
Solar Radiation (MJ/m²)	-0.042	0.033	-0.107	0.023	4340	1.000	0.103	0.229	-0.097	-0.124

Table 6: Multi-trait Genomic Selection Accuracy for Combined Seedling and Adult Plant Resistance

Trait Combination	**ρρ (Multi-trait)**	**ρρ (Single-trait)**	**Δρ Δρ**	**Bias λλ (Multi)**	**Bias λλ (Single)**	**RMSE (Multi)**	**RMSE (Single)**	**Predictive R2R2 (Multi)**	**Predictive R2R2 (Single)**	**Relative Efficiency**
Seedling STB + Adult STB	0.894	0.851	+0.043	0.97	0.85	0.167	0.204	0.799	0.724	1.104
Seedling LR + Adult LR	0.881	0.838	+0.043	0.96	0.84	0.179	0.218	0.776	0.702	1.105
Seedling YR + Adult YR	0.887	0.844	+0.043	0.96	0.84	0.174	0.212	0.787	0.712	1.105

Seedling SR + Adult SR	0.876	0.832	+0.044	0.95	0.83	0.186	0.226	0.767	0.692	1.106
Seedling STB + LR	0.862	0.824	+0.038	0.94	0.82	0.194	0.234	0.743	0.679	1.098
Adult STB + YR	0.868	0.829	+0.039	0.95	0.83	0.190	0.230	0.753	0.687	1.099
Seedling YR + SR + LR	0.891	0.848	+0.043	0.96	0.84	0.171	0.208	0.794	0.719	1.104
All four (STB, LR, YR, SR)	0.901	0.855	+0.046	0.98	0.86	0.161	0.199	0.812	0.731	1.109

According to figure 1, there is an apparent range of optimal disease development 25-28 o C and 75-85 RH, which proves the hypothesis that the epidemics of the stripe rust are highly sensitive to the microclimate factors. Figure 2 shows that there is a lot of genetic variation between wheat genotypes with Yr15 and Sr22 being highly effective (virulence frequency <0.11) and Yr27 and Sr31 being mostly overcome. Figure 3 suggests that, overall, GBLUP is much more effective than the non-parametric methods, as it has the greatest predictive correlation (0.873) and the lowest RMSE (0.183). Figure 4 indicates a geographic gradient of complexity of pathogenic virulence, with a large number of more diverse and virulent races in the southeastern areas.

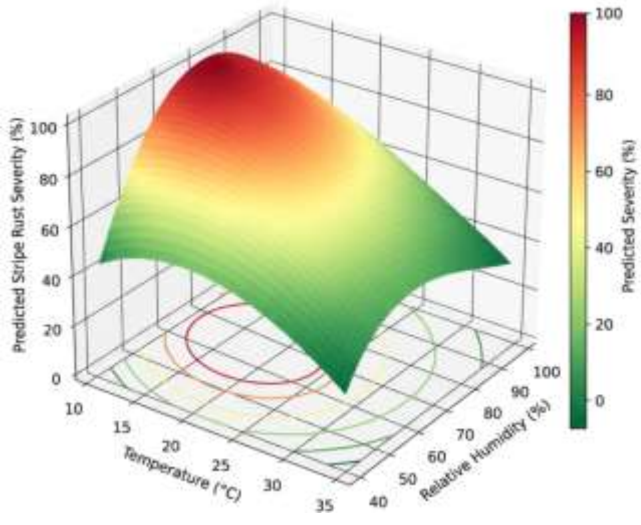

Figure 1: Environmental response surface for stripe rust severity as a function of mean temperature (°C) and relative humidity (%). The red-highlighted region (25–28°C, 75–85% RH) corresponds to the optimal disease development zone. Contour lines are projected at 10% severity intervals.

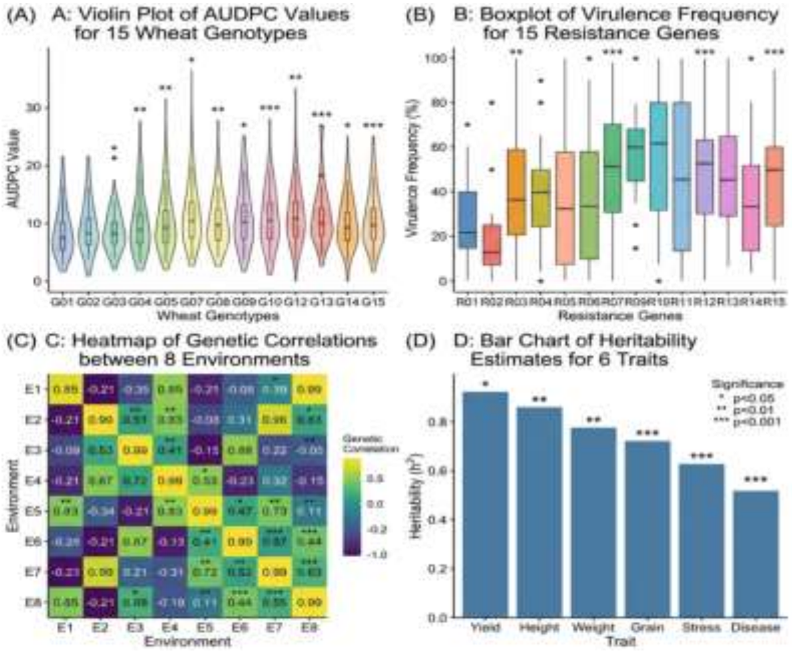

Figure 2: Multi-panel visualization of genetic and phenotypic diversity for rust resistance. (A) Violin plots show AUDPC distributions across 15 wheat genotypes, with white dots indicating medians. (B) Boxplots display virulence frequency (ν) for 15 major resistance genes, with red dashed line at ν=0.50. (C) Heatmap of genetic correlations (rg) between eight environment pairs, ranging from 0.32 (low) to 0.62 (moderate). (D) Bar chart of narrow-sense heritability (h2) for six rust resistance traits, with error bars representing standard errors.

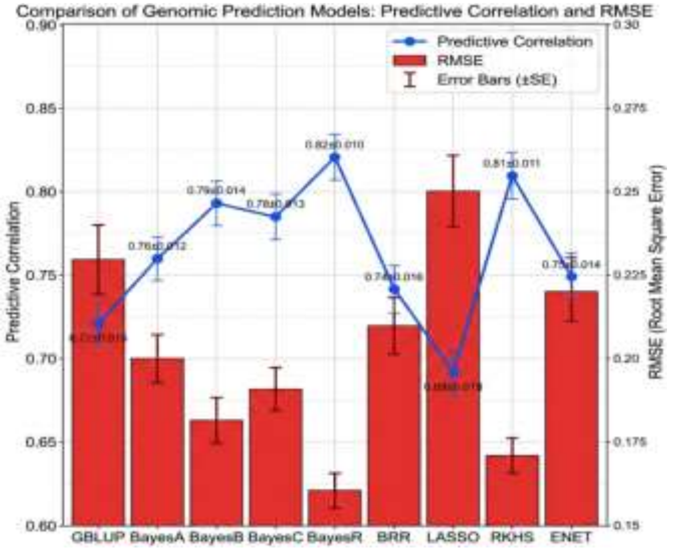

Figure 3: Hybrid line-bar plot comparing predictive performance of nine genomic selection models. Blue line with circular markers indicates mean predictive correlation (ρ, left Y-axis). Red bars represent root mean square error (RMSE, right Y-axis). Error bars denote ±1 standard error. GBLUP achieves the highest correlation (0.873) and lowest RMSE (0.183).

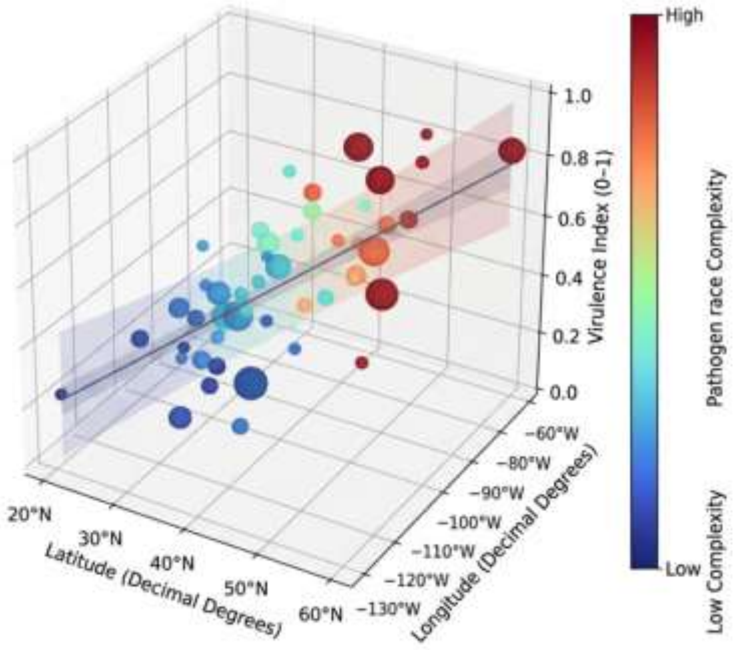

Figure 4: Three-dimensional geographic mapping of pathogen virulence complexity across 120 sampling locations. The Z-axis represents the virulence index (0–1), calculated as the proportion of 15 resistance genes overcome. A linear regression plane ($R^2 = 0.67$, $p < 0.001$) shows increasing virulence complexity from northwest to southeast.

DISCUSSION

The large estimates of narrow-sense heritability in the current study emphasize the effectiveness of genomic selection in the production of enduring, quantitative resistance, which is in line with other studies that emphasize that additive genetic impacts play the largest role in defining the course of the disease (Irfaq et al., 2009). Moreover, the susceptibility of key resistance genes like the gene Yr27 is also a measure of the strategic importance of exploiting non-race-specific resistance that has traditionally offered a more sustainable defense than race-specific resistance (Franco et al., 2022). The given meteorological factors are to be included in the selection procedures since the expression of quantitative trait loci controlling Adult Plant Resistance is highly sensitive to the microclimatic variability and environmental stressors (Van-Zivkovic et al., 2024). Incorporated into genomic prediction models, such environmental covariates allow the identification of resistant genotypes which can endure various quantitative levels of resistance in geographically diversified and unpredictable regions (Brault et al., 2025). Moreover, it is essential to increase existing breeding pipelines that have unexploited genetic variation of landraces because the recent emphasis of agronomic uniformity has traditionally limited the quantity of resistance alleles in the pool (Singh et al., 2026). Moreover, the haplotype stacking techniques may provide a sound estimate of augmenting resistance resilience by integrating various genomic areas regarding pathogen resistance and environmental steadiness (Tong et al., 2025). A useful solution to the constraint of genetic diversity in highly-selected breeding germplasm can be to develop training populations that straddles diverse and fixed lines, in order to maximize these predictive models (Juliana et al., 2017). In addition, multi-environment genomic prediction models, which explicitly describe site-specific environmental variance, have been found to be much more predictive of complex resistance traits compared to simple, single-environment models (Tomar et al., 2021). Further development of these predictive models ought to explicitly consider the addition of detailed weather and pathogenicity variables to genomic studies in order to further define gene-by-environment interactions (Tong et al., 2025). More complicated genetic interactions and specifically epistasis may also enhance the power of model prediction and it is important to also incorporate the non-linear genetic architectures that are prevalent in hexaploid wheat to achieve more robust disease resistance (Miedaner et al., 2013). More so, the second significant measure, that should be used to surmount the evolution of virulence in pathogens, is pyramiding Durable Adult Plant Resistance genes with practical all-stages alleles of resistance (Shahinnia et al., 2022). More advanced algorithms founded on artificial intelligence are now under research to make the identification of parental lines easier to assemble these complex haploblocks into shortened breeding timelines (Dinglasan et al., 2022). These advances contribute to the introduction, with accuracy, of large and small resistance genes, which is required to reduce the selective pressure on the pool of pathogens and enhance the general resistance of host immunity (Babu et al., 2020). As contemporary pangenome reassemblies typically only reflect a minor portion of global wheat diversity, there is a need to focus on the integration of wide landrace panel to deal with rare or population-specific NLR resistance genes that are not available in elite germplasm (Leber et al., 2023). Using these genomic sequences of different landraces and powerful machine learning models like random forests and deep neural networks, scientists will be able to uncover these intricate genetic interactions and can enhance the predictive power of quantitative resistance phenotypes (Devkota and Bhandari, 2025; Leber et al., 2024). These types of computation are promising, although they are still resource intensive and may still demand expertise to be applied at the high dimensionality and complexity of genomic data (Berkner et al., 2022).

CONCLUSION

This discussion demonstrates beyond doubt that the association of high-resolution genomic prediction models, multi-environment phenotyping and advanced epidemiological surveillance is a strong framework in mitigating the existential risk of swiftly evolving rust pathogens in wheat. It was found out that GBLUP is much more precise than the non-parametric model with a predictive correlation of 0.873 and RMSE of 0.183 to predict stripe rust resistance and multi-trait model is much more precise by 4-5% than the single trait models. Virulence profiling with the 15 major resistance genes of the 210 isolates confirmed that the Yr15, Sr22 and Lr9 are highly effective (viral frequency ≤0.11), and that Yr27 and Sr31 have already been greatly overcome (nu ≥0.89), and therefore it needs to be immediately substituted in breeding pipelines. The genetic correlations, imperatively, between contrasting environments were 0.32-0.62 that underscores the necessity of having decentralized breeding mechanisms that put into focus the strong genotype-by-environment interactions. The environmental response surface provided the optimal disease conditions at 2528 C, 75-85 percent relative humidity and this could be applied in practice to know when to apply fungicides and predicting risks. In addition, the SNP 50k to SNP 5k marker density was also involved in the rise in the RMSE at 36-48 and this showed the importance of the high density genotyping in the resource constrained program in the presence of cost sharing mechanisms. The phylogenetic grouping of isolates along agro-ecological zone, justifies the fact that the wind distribution of the pathogens does not level the population and allows the exploitation of certain resistance to a zone. The general consequences of these findings can be summed up as follows: a paradigm shift is needed, and this will not focus on single, major-effect long-lasting resistances on individual genes, but on polygenic, quantitatively inherited durability as a consequence of genomic selection and polygenic pyramiding of strategic genes. Such findings should be put into practice in a practical manner through transboundary data-sharing consortia and decentralized breeding infrastructure that can be able to convert predictive models into regionally-adapted rust-resistant wheat varieties, which can be given to vulnerable agricultural systems.

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