



RESEARCH ARTICLE

Identification of novel sources of yellow rust resistance in wheat-*Aegilops* derivatives through multi-location trials in Northern India

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Abstract

Yellow rust, also referred to as stripe rust is caused by the fungus *Puccinia striiformis* Westend f.sp. *tritici*. This pathogen leads to considerable reductions in both grain quality and crop yield worldwide, making it one of the most damaging fungal disease affecting wheat cultivation. The present study evaluated a set of 103 wheat-*Aegilops* derivatives along with their parental lines for resistance against yellow rust across three different locations viz. Baru Sahib (Himachal Pradesh), Palampur (Himachal Pradesh) and Ludhiana (Punjab) using a 0–9 disease rating scale. Significant differences in disease responses were observed among different genotype and environmental conditions. The analysis of variance indicated significant effects due to both genotype and location, with genotype accounting for the majority of variation at 58.63 %, while location contributed 17.39 %. Six parent genotypes viz., *Aegilops kotschy* (Ae. kot. 396, Ae. kot. 3573, Ae. kot. 3790) and *Aegilops peregrina* (Ae. per. 3519, Ae. per. 3774 and Ae. per. 3772) demonstrated immunity across all locations. The derivative 75-1-4-23-3-2-4 showed a highly resistant response at 2 locations, whereas, the derivatives, 75-1-4-16-3-5 and MB-11-2-17 showed consistent resistance over 3 locations. The derivatives 75-1-4-16-3-5, 77-33-2-5-9-(x)-2, MB-11-2-17, MB-4-2-2 and PBW-343LrYr exhibited significant resistance, as evidenced by their low disease scores at 2 locations. The analysis of stability revealed a specific group of genotypes that demonstrated reliable performance across various environments. The results highlight the potential of wild *Aegilops* species as novel genetic reservoirs for the introgression of yellow rust resistance. The identified resistant and stable genotypes present a valuable opportunity for pre-breeding efforts aimed at developing robust, rust-resistant wheat cultivars.

Keywords: *Aegilops* species; disease resistance; pre-breeding; *Puccinia striiformis*; rust resistance breeding; yellow rust

Introduction

Wheat is one of the leading cereals in the world, belonging to the family Poaceae and is the most widely cultivated cereal crop globally in various agro-climatic regions, with a production of 778.6 million tons (t) during 2021–22 and a productivity of 3.2 t/ha¹ of land (1). It is cultivated on over 221 million hectares of land. Bread wheat (*Triticum aestivum* L.) is primarily consumed and used as an ingredient in a variety of baked goods, accounting for more than 90 % of global wheat output. To address the global issue of food security, wheat production and productivity must be enhanced through improvements in yield and efficiency. At the same time, wheat production is facing challenges from both biotic and abiotic constraints, especially an increased pressure from pathogens such as wheat rusts, among which the yellow rust pathogen is one of the most problematic (2). Globally, approximately 91 % of the wheat production area is affected by various stresses (3), yet annual losses

to wheat rust pathogens range between 4.3 and 5.0 billion US dollars (4). Wheat rusts, including leaf rust, stem rust and yellow rust are common, invasive and extremely destructive diseases that significantly reduce wheat yields (5). Yellow rust is considered a globally destroy fungal disease of wheat caused by the fungal pathogen *Puccinia striiformis* Westend f. sp. *tritici*, an obligate biotrophic pathogen (6).

Wheat yields may decrease by 20–30 % during epidemic years and in extreme situations, by up to 60 %, or possibly be destroyed entirely. Wheat is grown in five crucial zones across India, namely: Himachal Pradesh, Uttarakhand, the Union territories of Jammu and Kashmir and Ladakh are all part of the Northern hill zone. Wheat is grown on approximately 0.8 million ha in this location, where the temperature is quite cold and humid therefore, leaf rust occurs late in the season (7). The maximum yellow rust outbreaks were detected in 2010 and 2011 and again in 2016–2018

(8). Yellow rust infection is often visible on the adaxial leaf surface as parallel rows of yellow to orange pustules that release uredinio spores, causing cycles of re-infection and cross-infection in wheat (9). In recent years, the susceptibility of wheat to yellow rust has increased among dwarf and semi-dwarf cultivars, as well as with the adoption of improved irrigation and fertilisation practices (10). Wheat rust disease spreads rapidly in temperatures ranging from 10–30 °C (11). The rust pathogen can spread within 3 hr during humid periods with an air condition of 20 °C. Also, sufficient precipitation is required for the spread of infection in low-temperature conditions. Certain diseases can develop when the temperature ranges between 2 °C and 32 °C and there is sufficient humidity (12). Yellow rust significantly reduced germination and thousand kernel weights by 72 % and 56 %, respectively. It minimises the area covered by the green leaves, which impairs the supply of sugar to growing seeds. The two most significant leaves for sugar production in developing grains are the flag leaf and the second leaf. Significant yield losses are expected as a result of flag leaf infection, which is responsible for more than 70 % of grain filling, preventing the plant from completing its life cycle and thus affecting yield by impacting grain filling, grain quality, flour yield and durability (13).

Yellow rust is asymptomatic in the early stages of infection and can usually be identified by manual inspection of visual symptoms that appear later in the infection. It typically appears in late December in Peninsular India, whereas in wheat-growing regions of Northern and Central India, early infections develop by the end of January or sometimes later (7). The management of yellow rust severity on plants is addressed through various strategies, including physical, biological, chemical and cultural methods (12). Also, planting date is a beneficial agronomic method for reducing yellow rust and increasing crop output. Farmers can reduce the severity of yellow rust infections by deliberately adjusting the planting date. Fungicides have been widely utilised in the treatment of plant diseases affecting crops. Fungicides are the most effective way to manage yellow rust, including chemicals that prevent or inhibit the spread of fungal infections. Fungicides protect plant tissues by forming a barrier between the host plant and the fungus (14). The severity of the disease depends on the plant developmental stage as well as its susceptibility (15). Present investigation deals with the novel and stable sources of resistance from wheat-*Aegilops* derivative to support future breeding programs for durable yellow rust resistance in wheat.

Materials and Methods

The experimental material includes 103 wheat-*Aegilops* derivatives that were derived from the crossing between commercial wheat cultivars (Chinese Spring, HD2687, WL711, UP2338, PBW343) with related non-progenitors *Aegilops* species (*Aegilops kotschy* 396, *Aegilops kotschy* 3573, *Aegilops kotschy* 3790, *Aegilops peregrine* 3519, *Aegilops peregrine* 3774, *Aegilops peregrina* 3772) at the Indian Institute of Technology, Roorkee (IITR) (Uttarakhand) and Eternal University, Baru Sahib, Sirmaur (Himachal Pradesh) using pollen radiation hybridisation and induced homeologous pairing approaches (16–18). The experiment material; wheat-*Aegilops* derivatives along with parents and checks were sown during the rabi season of 2024, in the last week of October at three different environments, viz Eternal University, Baru Sahib, Himachal Pradesh; Rice and Wheat Research Station, Palampur, Himachal Pradesh and Punjab Agricultural University, Ludhiana, Punjab, India. Detailed pedigree information of the wheat-*Aegilops* derivatives is provided in Supplementary Table S1. Each genotype was represented by a plot of two rows of 1m and row to row spacing was maintained at 0.20 cm for introgression line. The experiment was conducted using an augmented block design (ABD) comprising 5 blocks, with 109 genotypes and 3 check varieties. Four blocks accommodated 22 test entries each, while the 5th contained 21 entries; 'Chinese Spring' was included as a filler to maintain uniformity. Check varieties were replicated across all blocks, whereas test genotypes remained unreplicated. The layout of the experiment ensured random distribution of entries within each block to minimise environmental variation. The disease score data for yellow rust has been collected using the rating scale (0–9) (19) (Fig. 1, Table 1). The severity of diseases was scored as an average score across the entire plot, on a scale of 0–9, with 0 and 9 corresponding to 0–10 % and 90–100 % disease coverage, respectively (Fig. 2). The infection with the yellow rust pathogen can also cause host cell death, appearing as chlorotic or necrotic flecks to blotches on seedling 14 leaves or chlorotic or necrotic flecks to stripes on adult-plant leaves (2).

Statistical analysis

The recorded disease reaction data over the three locations were subjected to statistical analysis following standard procedures for the estimation of genetic variance components, each location and pooled over the environments. All analyses were performed using Python, employing relevant libraries such as Pandas, SciPy and stats models, along with Minitab for validation and supplementary statistical computations.

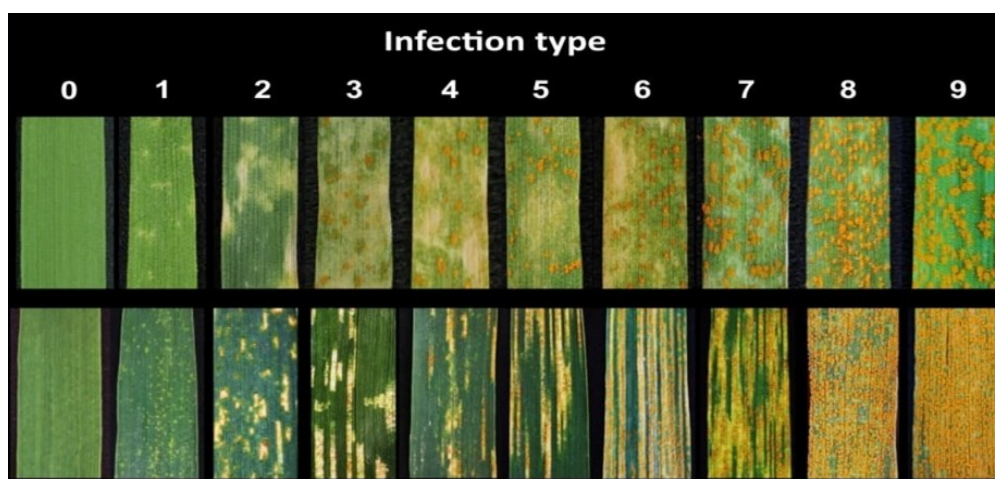


Fig. 1. Infection type scale of yellow rust on seedlings (the top row) and adult-plant leaves (the bottom row) showing various proportions of necrosis and sporulation.

Table 1. Yellow rust disease scored scales (0–9) based on infection types at seedling stage

Scale	Type's infection	Symptom
0	Low	No visible symptoms
1	Low	Necrotic/chlorotic flecks-no sporulation
2	Low	Necrotic/chlorotic stripes-no sporulation
3	Low	Necrotic/chlorotic stripes-trace sporulation
4	Moderate	Necrotic/chlorotic stripes-light sporulation
5	Moderate	Necrotic/chlorotic stripes-intermediate sporulation
6	Moderate	Necrotic/chlorotic stripes-moderate sporulation
7	High	Necrotic/chlorotic stripes-abundant sporulation
8	High	Chlorosis behind sporulating area-abundant sporulation
9	High	No necrosis/chlorosis-abundant sporulation

**Fig. 2.** Yellow rust destroyed the crop of a susceptible variety in an experimental plot.

Results

Analysis of variance (ANOVA)

The evaluation of the 103 wheat-*Aegilops* derivatives, along with parental lines and checks, across three distinct locations viz.; Baru Sahib (BS), Palampur (PL) and Ludhiana (LDH) revealed significant variation in yellow rust severity. Two-way ANOVA confirmed that both genotype and location effects were highly significant ($p < 0.001$), indicating substantial variation in yellow rust response among genotypes and testing environments (Table 2). Furthermore, the significant non-additivity component ($F = 14.28$, $p < 0.001$) indicated the presence of significant genotype $\times$ environment ($G \times E$) interaction, suggesting the differential response of the genotypes across locations.

Disease severity across location

Descriptive statistics across the three locations revealed that LDH experienced the highest mean disease severity (5.28) followed by PL (3.60) and BS (3.56). The higher standard deviation and wider range of disease scores observed at LDH suggest both greater genetic diversity in response and more intense disease pressure at this location. Palampur and BS locations exhibited comparatively moderate disease severity (Table 3).

Mean performance and frequency distribution across locations

The distribution of yellow rust disease scores across the three testing locations clearly demonstrated substantial variation in host response (Fig. 3, Table 4). At Eternal University, Baru Sahib, Himachal Pradesh a total of 7 genotypes including 6 wild parents and 1 derivate exhibited highly resistant reactions with the disease rating 0 whereas, 13 genotypes were classified as resistant (rating 1–2). The majority of derivatives [66] fell under the moderately resistant category with the disease rating 3–4, followed by 24 moderately susceptible (5–6) and only two genotypes obsessed as susceptible reactions with the disease rating 7–8. At PL, 7 genotypes remaining highly resistant while, 21 genotypes observed as resistant response. A total of 49 genotypes were moderately resistant, whereas 31 and 4 genotypes were categorised as moderately susceptible and susceptible, respectively. In contrast, LDH emerged as the most conducive environment for disease development, where only 6 genotypes maintained highly resistant reactions for four remained resistant. Twenty-four genotypes maintained moderately resistant reactions. A clear shift towards susceptibility was observed, with 39 genotypes each categorised as moderately susceptible and susceptible. Across the three testing locations, 6 parental genotypes viz. *Ae. kot.* 396, *Ae. kot.* 3573, *Ae. kot.* 3790, *Ae. per.* 3519, *Ae. per.* 3774

Table 2. Analysis of variance for yellow rust disease scores across genotypes and locations

Sources	Degree of freedom	Mean square	F-value	p-value
Genotypes	111	6.53***	4.89	< 0.01
Locations	2	107.48***	80.50	< 0.01
Residual ($G \times L$)	222	1.34	-	-
Non-additivity ($G \times L$)	17.98	17.98***	14.28	0.00
Balanced (Remainder)	278.38	1.26	-	-

*** significant at $p < 0.001$. Residual represents genotype $\times$ location ($G \times E$) interaction.

Table 3. Descriptive statistics of yellow rust disease across location

Locations	Total observations	Mean $\pm$ SE	Standard	Minimu	Maximu	Median	95 % Confidence
Baru Sahib	112	3.56 $\pm$ 0.138	1.46	0	7	4.0	3.292–3.833
Palampur	112	3.60 $\pm$ 0.166	1.76	0	7	4.0	3.273–3.924
Ludhiana	112	5.28 $\pm$ 0.188	1.99	0	8	6.0	4.908–5.646

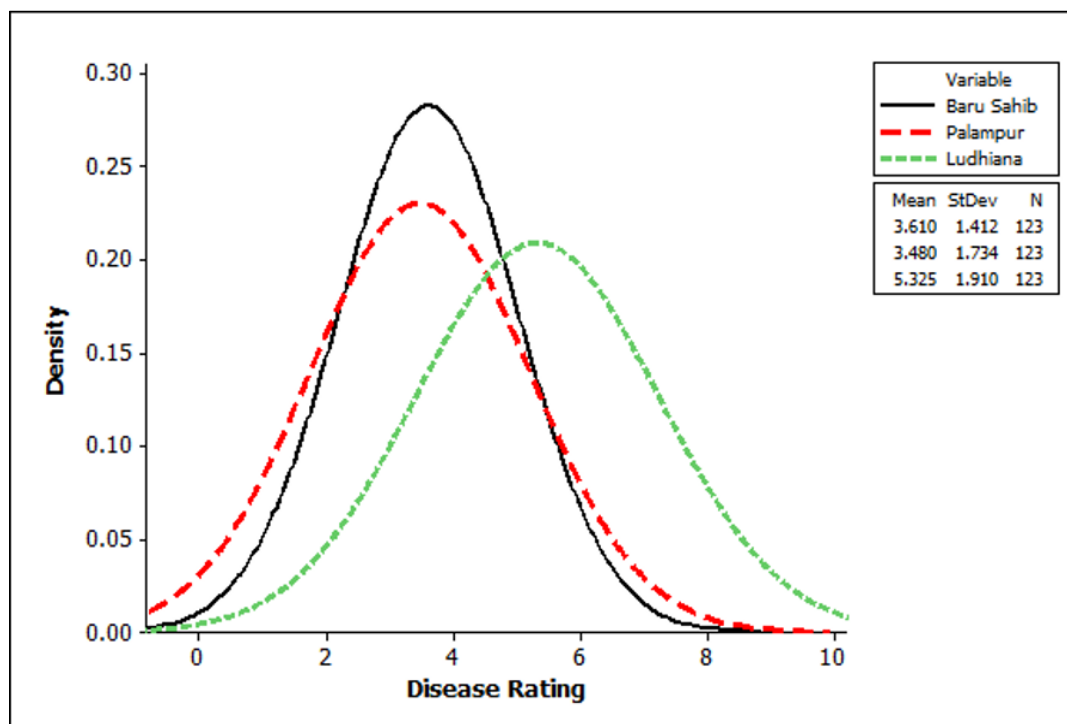


Fig. 3. Histogram showing the distribution of rating of wheat-*Aegilops* derivatives for resistance to yellow rust in Baru Sahib, Palampur and Ludhiana.

and *Ae. per.* 3772 consistently displayed an immune reaction with the disease rating 0, reflecting stable and broad-spectrum resistance. The derivative 75-1-4-23-3-2-4 demonstrated a highly resistant (immune reaction) at BS and PL, while exhibiting a resistant response at LDH (disease rating 1). Among resistant genotypes, 75-1-4-16-3-5 and MB-11-2-17 maintained consistent resistance across all three locations, emphasising their stability. Under the moderately resistant category genotypes such as PRH2-312-18-1, 16-1-8-56-4-4-2, PRH2-312-22-1, 48-41-6-6-5-4-1 and PRH2-331-4-1 showed consistent performance across all locations, suggesting partial but stable resistance. Other genotypes including MB-4-2-2 and PBW-343Lyr exhibited stable resistance at BS and PL, whereas 77-33-2-5-9-(x)-2 showed resistance at BS and LDH.

Comparative analysis among locations

The analysis demonstrated a significant overall location effect as indicated by the Kruskal-Wallis test ($H = 65.99, p < 0.001$), reflecting substantial variation among locations. Pairwise comparisons using the Mann-Whitney U test showed no significant difference between BS and PL, whereas LDH differed significantly from both locations. These findings were further supported by Tukey's HSD test, which also revealed significant differences involving LDH, while no significant difference was observed between BS and PL (Table 5).

Stability analysis

The stability analysis conducted with the Eberhart and Russell model showed significant differences in the adaptability of genotypes. Among the 112 genotypes, 17 were recognised as stable ($bi \approx 1$ with minimal deviation), while 43 genotypes showed adaptation to favourable environments (high disease conditions) and 36 genotypes were suited for unfavourable environments (low disease conditions). In addition, 6 genotypes were identified as immune. The environmental index analysis indicated that LDH ($EI = +1.131$) offered a favourable environment for disease development, whereas BS ($EI = -0.583$) and PL ($EI = -0.548$) showed negative indices, suggesting less conducive environments.

Correlation and consistency across locations

Spearman rank correlation analysis revealed a strong positive association between BS and LDH ($r_s = 0.66$), whereas moderate correlations were observed between PL and LDH and weaker correlation between BS and PL. These results indicate partial stability in genotype performance across environments, with some variation in ranking (Table 6). The Friedman test further confirmed significant differences among locations ($\chi^2 = 96.66, p < 0.001$), confirming the influence of environmental factors on disease expression.

Pooled classification of genotypes based on mean disease score across locations

Based on the average disease score across locations genotypes classification indicated that 5.4 % were immune, 3.6 % as resistant, 30.4 % as moderately resistant, 57.1 % as moderately susceptible and 3.6 % as susceptible. The large proportion of moderately susceptible genotypes indicates a requirement for further improvement in resistance. Among the resistance genotypes such as 75-1-4-23-3-2-4, 75-1-4-16-3-5, MB-11-2-17 and MB-4-2-2 consistently exhibited low disease scores highlighting their potential as valuable genetic donors for breeding initiatives.

Discussion

The present study evaluated a set of wheat-*Aegilops* derivatives for yellow rust resistance across three locations, viz. Eternal University, Baru sahib; Rice and Wheat Research Station, Palampur; Punjab Agricultural University, Ludhiana. Yellow Rust is a critical constraint in wheat production with the potential to reduce yields by up to 30 % and cause severe crop losses over a short period, thereby posing a major threat to food security. The evaluation indicated significant variation in disease response among genotypes. Six genotypes (*Ae.kot.* 396, *Ae.kot.* 3573, *Ae.kot.* 3790, *Ae.per.* 3519, *Ae.per.* 3774 and *Ae.per.* 3772) exhibited complete immunity (score 0) across all environments, indicating the presence of strong and stable resistance, consistent with earlier findings (20). In addition, genotype

Table 4. Rust reactions in wheat-*Aegilops* derivatives at various locations

Eternal University, Baru Sahib			Rice and Wheat Research Station, Palampur		Punjab Agricultural University,	Common genotype across the locations
Disease	Disease	Reaction	Wheat- <i>Aegilops</i> derivatives			
0	0	Highly resistant (HR)	<i>Ae. Kot.</i> 396, <i>Ae. Kot.</i> 3573, <i>Ae. Kot.</i> 3790, <i>Ae. Per.</i> 3519, <i>Ae. Per.</i> 3774, <i>Ae. Per.</i> 3772, 75-1-4-23-3-2-4	<i>Ae. Kot.</i> 396, <i>Ae. Kot.</i> 3573, <i>Ae. Kot.</i> 3790, <i>Ae. Per.</i> 3519, <i>Ae. Per.</i> 3774, <i>Ae. Per.</i> 3772, 75-1-4-23-3-2-4	<i>Ae. Kot.</i> 396, <i>Ae. Kot.</i> 3573, <i>Ae. Kot.</i> 3790, <i>Ae. Per.</i> 3519, <i>Ae. Per.</i> 3774, <i>Ae. Per.</i>	<i>Ae. Kot.</i> 396, <i>Ae. Kot.</i> 3573, <i>Ae. Kot.</i> 3790, <i>Ae. Per.</i> 3519, <i>Ae. Per.</i> 3774, <i>Ae. Per.</i> 3772
1–10 %	1–2	Resistant (R)	75-1-4-16-3-5, MB-4-2-2, MB-11-2-17, PBW-343Lrry, MB-33-2-3, 77HD2967, MB-2-4, MB-16-1-33-2-5-9-(x)-2, 77-46-6-17-9-2, 49-1-11-28-2-5, 77-36-6-15-1-8-5, PRH3-64-1-1, 49-1-11-9-7-1-2, NAPHAL, PRH3-62-1	75-1-4-16-3-5, MB-4-2-2, MB-11-2-17, PBW-343Lrry, MB-33-2-3, 77HD2967, MB-2-4, MB-16-1-33-2-5-9-(x)-2, 4, PBW343+Lr28+GPCB1, HPW349, 49-1-11-9-7-1, HPW89, MB-13-2-2, PBW343Unnat, PBWZn1, HS240, PRH3-23-6, PRH3-	75-1-4-23-3-2-4, 75-1-4-16-3-5, MB-11-2-17, 77-33-2-5-9-(x)-2	75-1-4-16-3-5, MB-11-2-17
11–30 %	3–4	Moderately resistant (MR)	HD2967, MB-2-4, MB-16-1-4, PBW343+Lr28+GPCB1, HPW349, PRH2-312-18-1, 49-1-11-9-7-1, HPW89, 49-1-73-8-5, P-174-2-2, 16-1-8-56-4-4-2, PRH2-312-22-1, 48-41-6-6-5-4-1, PRH2-331-4-1, MB-13-2-2, PBW343Unnat, PBWZn1, HS240, MB-64-1-1, 46-1-15-15-3-1, MB-27-4-12, MB-27-1-1, PRH3-23-6, B-56-2-5-1, PRH2-487-6-2, PRH3-15-12, 17-1-2-5-4-6-5, B-56-2-5-4, PRH3-77-5, 79-1-4-8-1, 1-1-7-18-5, B-52-1-8, PRH3-1-3, PRH3-30-3, PRH3-71-3-2, PRH3-71-3-2, PRH3-83-1-4, 77-50-33-2-5-2, B-14-1-2-4, MB-39-2-6, MB-67-1-2, 17-1-2-5-4-9-3, 48-44-23-3-6-4-4, MB-31-1-5, 14-1-1-18-6-4-6-2, 79-2-1-14-1-6-1, PRH3-73-2, PRH3-67-1, 77-50-8-4-2, PRH3-13-5, PRH3-45-3, PRH3-81-6-2, PAVON76, 14-1-8-18-6-8-5, P-163-2-4, 63-2-12-14-10-2-4, 77	MB-33-2-3, PRH2-312-18-1, 49-1-73-8-5, P-174-2-2, 16-1-8-56-4-4-2, PRH2-312-22-1, 48-41-6-6-5-4-1, PRH2-331-4-1, 77-46-6-17-9-2, MB-64-1-1, PRH3-64-1-1, 46-1-15-15-3-1, MB-27-4-12, MB-27-1-1, 17-1-2-5-4-6-5, B-56-2-5-4, PRH3-77-5, 79-1-4-8-1, PRH3-1-3, PRH3-30-3, PRH3-71-3-2, PRH3-83-1-4, PRH3-46-4-2, PRH3-13-5, PRH3-45-3, PRH3-81-6-2, 63-2-12-14-10-2-4, 77-46-6-2-4-6-6-4, 79-1-4-2-3, PRH2-87-4-5, PRH3-51-5, 17-3-37-16-2-7-1, PRH2-124-5-1, PRH3-74-3-1, PRH3-79-2, B-16-1-5, B-52-2-6, MB-27-2-10, B-	MB-4-2-2, PBW-343Lrry, HD2967, MB-2-4, MB-33-2-3, MB-16-1-4, PBW343+Lr28+GPCB1, HPW349, PRH2-312-18-1, 16-1-8-56-4-4-2, PRH2-312-22-1, 48-41-6-6-5-4-1, PRH2-331-4-1, 49-1-11-28-2-5, 77-36-6-15-1-8-5, B-56-2-5-1, PRH2-487-6-2, 1-1-7-18-5, B-52-1-8, 17-1-2-5-4-9-3, 48-44-23-3-6-4-4, MB-31-1-5, 14-1-8-18-6-8-5, P-163-2-4	PRH2-312-18-1, 16-1-8-56-4-4-2, PRH2-312-22-1, 48-41-6-6-5-4-1, PRH2-331-4-1
31–50 %	5–6	Moderately susceptible (MS)	CS, PRH3-46-4, PRH3-3-1, PRH3-80-4, PRH3-78-1-3, 17-3-37-16-2-7-1, PRH2-124-5-1, PRH3-74-3-1, PRH3-79-2, B-56-2-5-5, B-16-1-5, B-52-2-6, MB-27-2-10, 1RS40:9, B-52-1-4, PRH3-61-1, PRH3-68-1-5, 16-1-8-4-1-1, 17-3-8-4-6-3, PRH3-33-2, 77-3-37-16-2-7-1, PRH3-43-1, MA1PAVON, PRH3-74-3-2	1-7-18-5, B-52-1-8, 77-33-2-5-2, B-14-1-2-4, MB-39-2-6, 49-1-11-9-7-1-2, 17-1-2-5-4-9-3, 48-44-23-3-6-4-4, MB-31-1-5, NAPHAL, PAVON76, 14-1-8-18-6-8-5, P-163-2-4, PRH3-62-1, 1RS44:38, B-56-2-5-5, B-58-3-3, PRH3-70-6, 1B+38, 1RS40:9, 46-1-27-10-6-8-2-5, 49-1-11-6-10-2, 77-46-6-8-2-1-1, 16-1-8-4-1-1, 17-3-8-4-6-3, 17-1-2-2-3	49-1-11-9-7-1, HPW89, 49-1-73-8-5, P-174-2-2, MB-13-2-2, 77-46-6-17-9-2, PBW343Unnat, PBWZn1, HS240, MB-64-1-1, 46-1-15-15-3-1, MB-27-4-12, MB-27-1-1, PRH3-15-12, CS, 17-1-2-5-4-6-5, B-56-2-5-4, PRH3-77-5, PRH3-1-3, PRH3-30-3, PRH3-71-3-2, PRH3-83-1-4, 77-33-2-5-2, B-14-1-2-4, MB-39-2-6, 49-1-11-9-7-1-2, 17-1-2-5-4-9-3, 48-44-23-3-6-4-4, 79-1-4-2-3, PRH2-87-4-5, PRH3-51-5, 17-3-37-16-2-7-1, PRH2-124-5-1, PRH3-74-3-1, PRH3-79-2, B-16-1-5, B-52-2-6, MB-27-2-10, 1RS40:9, 46-1-27-10-6-8-2-5, 49-1-11-6-10-2, 77-46-6-8-2-1-1, 16-1-8-4-1-1, 17-3-8-4-6-3, 17-1-2-2-3	B-56-2-5-5
51–75 %	7–8	Susceptible	1-1-7-18-5-5-18, 17-1-2-2-3	PRH3-33-2, PRH3-43-1, MA1PAVON, PRH3-74-3-2	PRH3-33-2, PRH3-43-1, MA1PAVON, PRH3-74-3-2	---
76–100 %	9	Highly Susceptible	---	---	---	---

Table 5. Effect of locations based on non-parametric and parametric tests

Comparison/Parameter	Test used	Statistics value	p-Value	Significance	Interpretation
Overall location effect	Kruskal-Wallis Test	H = 65.99	4.67×10^{-15}	***	Significant differences among locations
Baru Sahib vs Palampur	Mann-Whitney U	U = 6161.50	1.00	Ns	No significant differences
Baru Sahib vs Ludhiana	Mann-Whitney U	U = 2746.50	4.74×10^{-13}	***	Significant difference
Palampur vs Ludhiana	Mann-Whitney U	U = 3084.50	8.87×10^{-11}	***	Significant difference
Baru Sahib vs Ludhiana	Tukey HSD	Mean diff = 1.71	0.00	***	Significant
Baru Sahib vs Palampur	Tukey HSD	Mean diff = 0.04	0.987	Ns	Not significant difference
Ludhiana vs Palampur	Tukey HSD	Mean diff = -1.68	0.00	***	Significant

Table 6. Spearman rank correlation between locations

Locations pair	Spearman's r_s	p-value	Significance	Interpretation
Baru Sahib vs Palampur	0.26	5.37×10^{-3}	**	Weak correlation
Baru Sahib vs Ludhiana	0.66	3.49×10^{-15}	***	Moderate-strong correlation
Palampur vs Ludhiana	0.33	4.35×10^{-4}	***	Low-moderate correlation

75-1-4-23-3-2-4 exhibited a highly resistant response at two locations (BS and PL) whereas, resistance at LDH, suggesting location-specific expression of resistance. Furthermore, genotypes such as 75-1-4-16-3-5, 77-33-2-5-9-(x)-2, MB-11-2-17, MB-4-2-2 and PBW-343LrYr demonstrated resistance reaction, suggesting their potential utility in resistance breeding programme. The average disease severity was highest at LDH, followed by PL and BS, indicating that LDH provided a high disease pressure environment. The greater variability and wider range of disease scores at this location reflect enhanced pathogen activity and differential genotypic responses under conditions favourable for disease development. Comparable findings have been documented in studies across various environments, indicating that climatic factors specific to each location had a substantial impact on the severity of yellow rust and the performance of different genotypes (21, 22). The observed distribution pattern showed that the only a small proportion of genotypes exhibited highly resistant or resistant reactions, whereas the majority were confined to the moderate resistance classes (scale 3-4). The predominance of moderate resistance responses indicates the quantitative nature of resistance, consistent with recent findings (23).

The stability analysis indicated that a limited number of genotypes maintained high stability across multiple environments, whereas the majority were adapted to specific favourable or unfavourable conditions, indicating significant genotype $\times$ environment interaction. Genotypes such as MB-33-2-3, MB-16-1-4 and PBW343+Lr28+GPCB1 exhibited consistent performance ($b_i \approx 1$ with minimal deviation), suggesting their wide adaptability. Similar observations of limited stability coupled with specific adaptation have been reported in earlier studies (24, 25) emphasising the role of stable genotypes in breeding programs aimed at achieving durable resistance.

The non-parametric analysis provided additional confirmation of these results, with the Kruskal-Wallis test indicating a highly significant effect of location. Pairwise comparisons revealed significant differences among LDH, BS and PL suggesting that LDH represents a distinct environment influencing disease expression, as reported in earlier study (26, 27). The variation observed in rust response may be attributed to the complex interaction between host resistance and pathogen variability. This highlights the strong evolutionary potential of the yellow rust pathogen, enabling rapid adaptation to diverse environmental conditions and potentially leading to resistance breakdown in wheat cultivars (2, 28). The identification of immune and stable genotypes emphasises the importance of *Aegilops*-derived material as key genetic resources for

resistance breeding. These genotypes hold significant promise as pre-breeding resources for the incorporation of durable resistance genes into elite wheat cultivars. The presence of the moderately resistant genotypes further emphasises the importance of quantitative resistance strategies for sustainable disease management.

Conclusion

The multi-location evaluation revealed significant variation in yellow rust resistance, with strong effects of genotype, environment and their interaction. Ludhiana (LDH) was the most disease-prone location, while Baru Sahib (BS) and Palampur (PL) showed moderate disease pressure. Six parental lines (*Ae. Kot.* 396, *Ae. Kot.* 3573, *Ae. Kot.* 3790, *Ae. Per.* 3519, *Ae. Per.* 3774 and *Ae. Per.* 3772) exhibited stable immunity across locations. Among derivatives, 75-1-4-16-3-5 and MB-11-2-17 showed consistent resistance across locations, while 75-1-4-23-3-2-4 showed an immune reaction at two locations and resistance at one, indicating location-specific response. The derivatives 75-1-4-16-3-5, 77-33-2-5-9-(x)-2, MB-11-2-17, MB-4-2-2 and PBW-343LrYr exhibited significant resistance, as evidenced by their low disease scores two locations. The identified stable highly resistant derivatives can be utilised as valuable donors in breeding programmes for the development of yellow rust-resistant wheat varieties.

Authors' contributions

ND initially collected data and identification of novel sources of yellow rust resistance in wheat-*Aegilops* derivatives through multi-location trials in Northern India. PC provided valuable guidance in interpreting the disease data and offered continuous support throughout the research work, including during the preparation of the final manuscript draft. HSD, VT and IS were involved in the development of the material, evaluation of the lines, manuscript writing and finalising the manuscript. SK assisted and facilitated the field research trial at PAU, Ludhiana and also reviewed the manuscript. VR supported and facilitated the necessary activities to conduct the field research trial at CSKHPKV, Palampur.

Compliance with ethical standards

Conflict of interest: Authors do not have any conflict of interests to declare.

Ethical issues: None

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