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Pathotype Analysis of *Blumeria graminis* f. sp. *tritici* and Characterization of Some Powdery Mildew Resistance Genes in Egyptian Wheat Genotypes

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Abstract

Ensuring sustainable wheat production is crucial for food security, particularly in regions facing plant diseases and environmental challenges. In the Middle East, wheat powdery mildew, caused by *Blumeria graminis* f. sp. *tritici*, significantly threatens yields and quality, making it essential to comprehend local pathotype virulence and resistance genes for effective disease management. Therefore, 252 infected leaves were sampled from cultivated bread wheat genotypes in Egypt Delta region during 2024–2025 growing seasons. Forty-one powdery mildew monogenic lines (*Pm*) and twelve new bread wheat genotypes were evaluated at seedling and adult stages. Pathotype analysis identified six pathotypes in 2024 (IOEE most common, 22.91%) and four in 2025 (POKE dominant, 34.25%). Virulence diversity declined significantly: Shannon diversity decreased from 5.90 to 3.73, and Simpson diversity decreased from 5.81 to 3.58, indicating reduced pathotype complexity. No virulence was detected against *Pm*11, *Pm*13, *Pm*24, *Pm*25, *Pm*29, *Pm*32, *Pm*34, *Pm*35, *Pm*36, *Pm*37, *Pm*43, NCA9, and MIAB10 genes. PERMANOVA analysis showed no significant compositional difference between seasons ($R^2 = 0.112$, $p = 0.481$), with low genetic differentiation (mean character difference = 0.1894; Roger's distance = 0.1914). Sakha96 wheat genotype displayed complete resistance at both growth stages, while Misr4, Misr5, Sakha95, Sids15, and Gemmeiza13 showed adult-stage resistance only in both seasons. Molecular analysis revealed that *Pm*24, *Pm*35 and *Pm*37 were absent in all genotypes. Pedigree analysis demonstrated *Pm*35 and *Pm*37 share common ancestral origins across Egyptian wheat germplasm. So, these resistant genotypes and broadly effective resistance genes provide valuable resources for breeding durable powdery mildew resistance in Egyptian wheat.

Keywords: wheat; *Pm* genes; virulence analysis; pathotype diversity; powdery mildew; molecular analysis; pedigree

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1. Introduction

Powdery mildew, *Blumeria graminis* f. sp. *tritici* (*Bgt*), is one of the devastating diseases affecting wheat (*Triticum aestivum* L.), which results in a significant reduction in wheat quality and grain yield in case of severe infection [1–4]. *Blumeria graminis* ranks sixth of the

ten most urgent fungal pathogens affecting wheat worldwide due to the emergence of new virulent isolates, environmental changes, and the susceptibility of wheat cultivars [5,6]. Wheat yield losses ranged from 10 to 62% [7–9], with severe infection resulting in yield losses up to 100% [10]. In the absence of resistant varieties, different chemical fungicides were applied for powdery mildew management [11]. Application of fungicides is often unsustainable because of the emergence of isolates with acquired resistance and environmental and economic concerns [12]. Additionally, *Bgt* has a diverse population composition, fast mutation rates, and virulent pathotypes [13,14]. Therefore, investigating population dynamics and genetic evolution, tracking the pathogen's spread across different regions, and assessing the pathogenic isolates' diversity are crucial for enabling scientists to monitor the efficiency of resistance genes [15,16]. These efforts provide early warnings of resistance loss in wheat varieties [13,17]. Therefore, host plant resistance remains a key component of integrated powdery mildew management [18,19], which includes both race-specific and non-race-specific resistance [20,21]. Race-specific resistance has been widely used in breeding programs; however, the narrow genetic base of wheat cultivars often limits its durability. Moreover, race-specific resistance is ephemeral and easily defeated by emerging virulent races [22,23]. Conversely, adult resistance is long-lasting, robust, more durable, and highly efficient for multiple pathogens [24,25]. To date, about 110 to 140 *Pm* genes/alleles have been characterized in common wheat and its relatives, including 69 formally designated genes [19,26,27], many of which confer race-specific resistance. Consequently, it is crucial to assess disease resistance across diverse wheat germplasm and understand its genetic basis to support the effective deployment of resistance genes in wheat production [28]. Historically, the identification of *Pm* genes relied on the phenotypic screening of monogenic lines against specific *Bgt* isolates. This approach often struggles to distinguish between individual resistance sources in complex backgrounds. To overcome these limitations, the integration of molecular markers (e.g., SSRs, STSs, and SNPs) has become indispensable in modern breeding programs. Molecular markers allow for the precise characterization of a cultivar's genetic profile, enabling researchers to identify specific *Pm* genes and confirm gene pyramiding. The main wheat pathogens in Egypt are stripe rust and powdery mildew, which significantly reduce yield [29]. Powdery mildew is now considered a major threat, as the majority of widely cultivated Egyptian wheat varieties are susceptible [11,30]. Furthermore, limited investigations have addressed the genetic basis of powdery mildew resistance, particularly for the new wheat varieties as well as the current population structure of *Bgt* across major wheat- growing regions [31]. Therefore, this study aims to identify the pathotypes of *Bgt* isolates, test the virulence and diversity of the Egyptian powdery mildew populations, and study the effectiveness of *Pm* genes and the new released breeding genotypes in both stages and molecular and pedigree analysis of some *Pm* genes in the new released bread wheat genotypes.

2. Materials and Methods

2.1. Collecting Samples of Infected Leaves

The naturally infected leaves with conidia of *Bgt* were collected from the susceptible wheat genotypes (Misr1, Misr2, Gemmeiza 12, Giza 171, Sakha95, Misr3), grown in fields of the middle governorates of Delta Egypt in the 2024–2025 seasons (Table 1). These governorates include Gharbia, Kafr El Sheikh, Dakahlia, and Menoufia. Two hundred and fifty-two isolates, 144 in 2024 and 108 in 2025, were collected.

Table 1. Distribution and number of infected wheat leaf samples collected across different governorates and locations in the 2024 and 2025 growing seasons.

Governorate	Location	No. of Samples	
		2024	2025
Gharbia	Gemmeiza Res. Station	26	20
	Tanta	10	10
	El-Santa	20	15
	Kafer ElZayat	20	10
	Kotor	15	10
Menoufia	Sers Elayan	10	10
	Shebein Elkom	10	10
Kafr El Sheikh	Sakha Research Station	15	7
Dakahlia	Shirbin	8	6
	Mansoura	10	10
Total		144	108

2.2. Plant Material and Inoculation

A total of 53 genotypes, including 41 *Pm* differential lines provided from North Carolina State University (Table 2) and 12 new released spring wheat genotypes, sourced from the International Wheat Program, Field Crops Research Institute, were selected for virulence analysis (Table 3). The pedigree of the powdery mildew cultivars/lines, sources and chromosomal location is listed according to [32,33]. All the genotypes were cultivated in plastic pots (10 × 10 cm). A fresh colony of each fungal isolate was inoculated into the susceptible wheat variety Chancellor seedlings for multiplication using the pot-brushing technique. Then, single pustules were separated by transferring conidia to a non-infected seedling using a sterile dissecting needle. All the isolates were kept in a separate transparent unit at 20–22 °C. All *Pm* gene lines and local wheat genotypes were inoculated separately with each pure isolate using the pot-brushing technique [34].

Table 2. List of the 41 *Pm* monogenic lines evaluated for powdery mildew resistance, including the cultivar/line, gene and gene location.

No	Cultivar/Line	Gene	Gene Location	References
1	CI14114	*Pm1a*	7*AL*	[35]
2	MocZlatka	*Pm1b*	7*AL*	[36]
3	*Pm*1c (*Pm*18)	*Pm1c*	7*AL*	[36]
4	W.W.	*Pm1d*	7*AL*	[36]
5	*Pm*1e (*Pm*22)	*Pm1e*	7*AL*	[37]
6	CI14118	*Pm2*	5*DS*	[38,39]
7	CI14120	*Pm3a*	1*AS*	[40]
8	C14121	*Pm3b*	1*AS*	[40]
9	CI14122	*Pm3c*	1*AS*	[40]
10	Ralle	*Pm3d*	1*AS*	[41]
11	W150	*Pm3e*	1*AS*	[41]
12	CI15888	*Pm3f*	1*AS*	[41]
13	Aristide	*Pm3g*	1*AS*	[42]
14	CI14124	*Pm4a*	2*AL*	[43]
15	Ronos	*Pm4b*	2*AL*	[43]
16	CI14125	*Pm5a*	7*AL*	[44]
17	Kormoran	*Pm5b*	7*AL*	[45]
18	15	*Pm5d*	7*AL*	[45]
19	Coker 747	*Pm6*	2*DL*	[46]

Table 2. *Cont.*

No	Cultivar/Line	Gene	Gene Location	References
20	Transec	*Pm7*	4*BS*.4*BL*2*Rl*	[47]
21	Kavakza	*Pm8*	1*RL*.1*BL*	[48]
22	N 14	*Pm9*	7*A*	[36]
23	Chinese Spring	*Pm11*	6*BS*	[49]
24	Wembley	*Pm12*	6*BS*	[49]
25	*Pm13*	*Pm13*	*T3BL*.3*S*	[50]
26	*Pm16*	*Pm16*	5*BS*	[51]
27	Amigo	*Pm17*	*T1AL*.1*RS*	[52]
28	*Pm19*	*Pm19*	7*D*	[37]
29	TamW-104	*Pm20*	6*BS*.6*RL*	[47]
30	DH2	*Pm21*	*T6AL*.6*VSS*	[53]
31	*Pm24*	*Pm24*	*1DS*	[54]
32	NC96BGTA5-Ab	*Pm25*	1*AS*	[55]
33	*Pm29*	*Pm29*	*7DL*	[56]
34	*Pm32*	*Pm32*	*1BL*.*1SS*	[57]
35	NC96BGTD7-Ab	*Pm34*	5*DL*	[58]
36	NC96BGTD3-Ab	*Pm35*	5*DL*	[59]
37	5-BIL29 (durum)	*Pm36*	5*BL*	[60]
38	NC96BGTAG11-Ab	*Pm37*	7*AL*	[61]
39	*Pm43*	*Pm43*	2*DL*	[62]
40	NC97BGTAB9-Ab	*NCA9*	3*BL*	[63]
41	NC97BGTAB10-Ab	*MIAB10*	2*BL*	[64]
42	Chancellor	-	-	
43	Coker 68-15	-	-	

-: absence of resistance genes.

Table 3. List of twelve new bread wheat genotypes and their pedigrees evaluated in the present study, including their specific parental crosses, pedigree information and year of release.

No	Wheat Genotype	Pedigree	Year of Release	Reference
1	Misr1	Oasis/skauz//4*BCN/3/2*PastorCMss00Y01881T-050M-030Y-030M-030WGY-33M-0Y-0S.	2011	[65]
2	Misr2	SKAUZ/BAV92.CMss96M03611S-1M-0105Y-010M-010SY-8M-0Y-0S.	2011	[65]
3	Misr3	ATTILA*2/PBW65*2//KACHU/	2019	[66]
4	Misr4	Ns-732//HER/3/PRL/SARA//TS1/Vee 5/4/FR6T2/5/WHEAR/SOKOLL	2022	[67]
5	Misr5	KACHU//WBLLi*2/BRAMBLING *2/3/KACHU/KiRiTATi		Still under registration
6	Misr6	SUP152/AkuRi//SUP152/3/MUCUY		Still under registration
7	Misr7	WBLLI//PUB94.15.1.12/WBLLI/3/Mucy		Still under registration
8	Sakha95	PASTOR//SITE/MO/3/CHEN/AEGILOPSSQUARROSA(TAUS)//BCN/4/WBLL1(CMSA01Y00158S-040P0Y-040M-030ZTM-040SY-26M-0Y-0SY-0S)WBLL1(CMSA01Y00158S-040P0Y-040M-030ZTM-040SY-26M-0Y-0SY-0S).	2018	[68]
9	Sakha96	MINO/6/Sakha12/5/Kvz//Cno67/Pj62/2/Yds”/Blo”s”/4/K 134 (60)/Vee.	2024	[69]
10	Sakha97	HUBARA-21//WAXWING2/HEILO(S.2013-095-010S-011S-55-0S)	2025	[70]
11	Sids15	PARUS/PASTOR//FIDIY-20/3/PASTOR//MILAN/KAUZ		Still under registration
12	Gemmeiza13	Gemmeiza3/HUBRAS/8/BUC//7C/ALD/5/MAYA74/ON//1160.147/3/BB/GLL/4/CHATS”/6/MAYA/VUL//CMH74A.6340/4SX/7/SIDS7.(CGM12-103979-2GM-1GM-1GM-0GM)		Still under registration

2.3. Disease Assessment and Virulence %

Disease severity of powdery mildew was assessed 10 days post-inoculation using the 0–9 scale [71], when control plants exhibited maximum severity. The virulence of each isolate was evaluated according to [72].

2.4. Pathotype Identification

Pathotypes were identified from 144 and 108 *Bgt* isolates, conducted in a controlled glass house environment (12 h light at 20 °C), with units covered in cheesecloth to prevent contamination at the Gemmeiza Agricultural Station. Sixteen *Pm* differential lines were used (Table 4), and pathotypes were designated according to the system described by [73]. Heatmap and principal component analysis (PCA) were performed to show the relationship between *Pm* pathotypes and/or their virulence formula on 41 *Pm* genes across the two seasons. The virulence/avirulence formula was converted to a binary data matrix (0 = resistant and 1 = susceptible). All statistical analysis and visualizations were performed using R software (version 4.5.1), using the R packages FactoMineR, factoextra and ggplot2. For assessing the powdery mildew pathotypes diversity, the following indices were calculated: Shannon index (q = 1), Simpson (q = 2), Richness (q = 0), and divergence was measured by mean character difference (MCD) and Roger's distance [74] based on virulence reactions of the differential lines. Differences in virulence composition between seasons were evaluated using permutational multivariate analysis of variance (PERMANOVA), while homogeneity of multivariate dispersion was tested using beta-dispersion analysis. Statistical analyses were conducted by the iNEXT package in R Studio (v4.5.1).

Table 4. Designation codes and characteristics of the 16 wheat differentials used for the identification and pathotype characterization of *Blumeria graminis* f. sp. *tritici* pathotypes.

Host set 1	***Pm1a***	***Pm2***	***Pm3a***	***Pm4a***
Host set 2	***Pm3c***	***Pm8***	***Pm17***	***Pm34***
Host set 3	***Pm4b***	***Pm5a***	***Pm20***	***Pm35***
Host set 4	***Pm7***	***Pm11***	***Pm25+3a***	***Pm37***
A	*R*	*R*	*R*	*R*
B	*R*	*R*	*R*	*S*
C	*R*	*R*	*S*	*R*
D	*R*	*S*	*R*	*R*
E	*S*	*R*	*R*	*R*
F	*R*	*R*	*S*	*S*
G	*R*	*S*	*R*	*S*
H	*S*	*R*	*R*	*S*
I	*R*	*S*	*S*	*R*
J	*S*	*R*	*S*	*R*
K	*S*	*S*	*R*	*R*
L	*R*	*S*	*S*	*S*
M	*S*	*R*	*S*	*S*
N	*S*	*S*	*R*	*S*
O	*S*	*S*	*S*	*R*
P	*S*	*S*	*S*	*S*

R—Resistant, *S*—Susceptible.

2.5. Field Experiment

Seeds of twelve new bread wheat genotypes and forty-one *Pm* monogenic lines were sown in rows 1.5 m long and spaced 50 cm apart at the Gemmeiza Agricultural Research Station during the 2024–2025 seasons using a randomized complete block design with three replicates. The experiment was bordered by two highly susceptible cultivars, Coker 68-15 and Chancellor. The final disease severity was assessed under natural infection conditions as the susceptible check varieties exhibited maximum powdery mildew severity using the 0–9 scale [71]. Then, data for both seasons were statistically analyzed using IBM SPSS Statistics (version 20), and mean comparisons were performed using the least significant

difference (LSD) test and Duncan's multiple range test at $p \leq 0.05$; means followed by the same letter are not significantly different.

2.6. Molecular Analysis

DNA Extraction and PCR Amplification Conditions

This work was conducted in the Plant Pathology and Biotechnology Lab, Faculty of Agriculture, Kafr Elsheikh University in 2025. Total DNA of 3 highly resistant monogenic lines (*Pm* genes) and the 12 new bread wheat genotypes was extracted from 250 mg of 15-day seedlings. The fresh leaf tissues were ground in liquid nitrogen with a mortar and pestle, and subsequently, DNA extraction was accomplished using the Invisorb® Spin Plant Mini Kit (STRATEC Molecular, Berlin, Germany) to purify the DNA, according to the manufacturer's instructions. The extraction method and amplification conditions were carried out as reported by [75]. The SSR markers Xgwm337, Xcfd7, and Xgwm332 linked to *Pm*24, *Pm*35, and *Pm*37 were used [59,61,76]. Each PCR mixture (25 µL) includes 1 µL of 25 ng nucleic acid, 1 µL of each primer (10 pmol), 12.5 µL of GoTag® Colorless Master Mix (Promega Corporation, Madison, WI, USA), and 9.5 µL of nuclease-free water (Promega). A total of 15 µL of all PCR products was analyzed by electrophoresis through a 2% agarose gel, stained with ethidium bromide, and DNA bands were visualized using a UV transilluminator. SSR profiles were scored as binary data, based on the presence (1) or absence (0). Genetic similarity among wheat genotypes was estimated using the Jaccard similarity coefficient. Pairwise similarity coefficients were calculated based on shared and non-shared alleles. All analysis was performed using the R environment. The Jaccard distance matrix was computed using the vegan and ape packages, and hierarchical clustering was conducted using the base hclust function.

2.7. Pedigree Analysis

To know the possible pedigree relationship between *Pm*24, *Pm*35 and *Pm*37 and 12 Egyptian wheat genotypes, the detailed pedigree was downloaded from https://www.cimmyt.org/about/data/?utm_source=chatgpt.com (accessed on 11 May 2025) and https://npgsweb.ars-grin.gov/gringlobal/accessiondetail/ (accessed on 11 May 2025) (Supplementary Table S1). Data of the pedigree and ancestry networks were constructed and visualized using the R statistical environment (version 4.3.0). Data manipulation and relationship mapping were conducted using the packages tidyverse, igraph, ggraph, and ggrepel.

3. Results

3.1. Disease Assessment

A total of 252 *Bgt* isolates were investigated for their virulence frequencies % on 41 known *Pm* genes during the 2024–2025 seasons (Table 5). In the 2024 season, virulence frequencies % ranged from 0 to 95.13%. Most isolates were virulent to the tested *Pm* genes, whereas resistance was observed to *Pm*11, *Pm* 13, *Pm*24, *Pm*25, *Pm*29, *Pm*32, *Pm*34, *Pm*35, *Pm*36, *Pm*37, *Pm*43, NCA9, and MIAB10. Five *Pm* genes, *Pm*16, *Pm*12, *Pm*20, *Pm*2, and *Pm*3d, respectively, showed virulence frequencies from 7.63 to 47.91% (less than 50%). However, 24 *Pm* genes (58.53%) showed virulence frequencies % of more than 50%. In the 2025 season, the tested *Pm* genes exhibited the same trend in their virulence frequencies % against 108 isolates of *Bgt*, with different values. *Pm*17 exhibited the highest virulence frequency in both seasons, with frequencies of 95.13% and 95.37%, respectively.

Table 5. Virulence frequency % of 144 and 108 isolates of *Bgt* evaluated under conditioned glass house in the 2024–2025 growing seasons.

No	Gene	2024 Season 2024		Virulence Frequency %	2025		Virulence Frequency %
		S	R		S	R	
1	*Pm1a*	89	55	61.80	76	32	70.37
2	*Pm1b*	103	41	71.52	96	12	88.88
3	*Pm1c*	108	36	75.00	92	16	85.18
4	*Pm1d*	102	42	70.83	87	21	80.55
5	*Pm1e*	90	54	62.50	92	16	85.18
6	*Pm2*	44	100	30.55	12	96	11.11
7	*Pm3a*	110	34	76.38	98	10	90.74
8	*Pm3b*	105	39	72.91	100	8	92.59
9	*Pm3c*	104	40	72.22	88	20	81.48
10	*Pm3d*	69	75	47.91	40	68	37.03
11	*Pm3e*	90	54	62.50	76	32	70.37
12	*Pm3f*	100	44	69.44	78	30	72.22
13	*Pm3g*	112	32	77.77	85	23	78.70
14	*Pm4a*	120	24	83.33	78	30	72.22
15	*Pm4b*	123	21	85.41	90	18	83.33
16	*Pm5a*	98	46	68.05	76	32	70.37
17	*Pm5b*	120	24	83.33	81	27	75.00
18	*Pm5d*	105	39	72.91	88	20	81.48
19	*Pm6*	102	42	70.83	75	33	69.44
20	*Pm7*	123	21	85.41	90	18	83.33
21	*Pm8*	110	34	76.38	78	30	72.22
22	*Pm9*	130	14	90.27	84	24	77.78
23	*Pm11*	0	144	0.00	0	108	0.00
24	*Pm12*	14	130	9.72	9	99	8.33
25	*Pm13*	0	144	0.00	0	108	0.00
26	*Pm16*	11	133	7.63	8	100	7.41
27	*Pm17*	137	7	95.13	103	5	95.37
28	*Pm19*	100	44	69.44	100	8	92.59
29	*Pm20*	24	120	16.66	15	93	13.88
30	*Pm21*	99	45	68.75	75	33	69.44
31	*Pm24*	0	144	0.00	0	108	0.00
32	*Pm25*	0	144	0.00	0	108	0.00
33	*Pm29*	0	144	0.00	0	108	0.00
34	*Pm32*	0	144	0.00	0	108	0.00
35	*Pm34*	0	144	0.00	0	108	0.00
36	*Pm35*	0	144	0.00	0	108	0.00
37	*Pm36*	0	144	0.00	0	108	0.00
38	*Pm37*	0	144	0.00	0	108	0.00
39	*Pm43*	0	144	0.00	0	108	0.00
40	NCA9	0	144	0.00	0	108	0.00
41	MIAB10	0	144	0.00	0	108	0.00
	Chancellor	144	0	100.00	108	0	100.00
	Coker 68-15	144	0	100.00	108	0	100.00

3.2. Pathotype Identification of Bgt

Six pathotypes were identified among 144 isolates of *Bgt* collected from different wheat genotypes in the 2024 season. These pathotypes were POKE, PJOE, MKOE, PIKE, MOKE, and IOEE (Table 6). Among the identified pathotypes, IOEE showed the highest occurrence (22.91%), while PJOE exhibited the lowest (10.41%). All of them ended with KE, OE, and EE letters and

were characterized by virulence to the *Pm* genes 1a, 2, 3a, 4a, 3c, 8, 17, 5a, and *Pm*7. Heatmap and PCA were performed based on the response of *Pm* genes to six identified pathotypes (Figure 1A). The PCA scatterplot explained a total variance of 40.5%, with PC1 (Dim1) accounting for 32.7% and PC2 (Dim2) explaining 27.8% of the total (Figure 1B).

Table 6. Occurrence and virulence formulae of the *Bgt* phenotypes identified among 144 isolates collected in 2024 season.

Pathotype	No. of Isolates	Occurrence %	Virulence/a Virulence Formulae	No. of Genes	Virulence %
POKE	26	18.05	1a,2,3a.4a,3c,8,17,4b,5a,7/	10	62.50
PJOE	15	10.41	1a,2,3a.4a,3c,17,4b,5a,20,7/	10	62.50
MKOE	28	19.44	1a,3a,4a,3c,8,4b,5a,20,7/	9	56.25
PIKE	17	11.80	1a,2,3a.4a,8,17,4b,5a,7/	9	56.25
MOKE	25	17.36	1a,3a.4a, 3c,8,17, 4b,5a,7/	9	56.25
IOEE	33	22.91	2,3a,3c,8,17,4b,7/	7	43.75

Occurrence % = number of isolates that have the same pathotype/total no. of isolates. Virulence % = number of virulent genes/no. of genes used for identification.

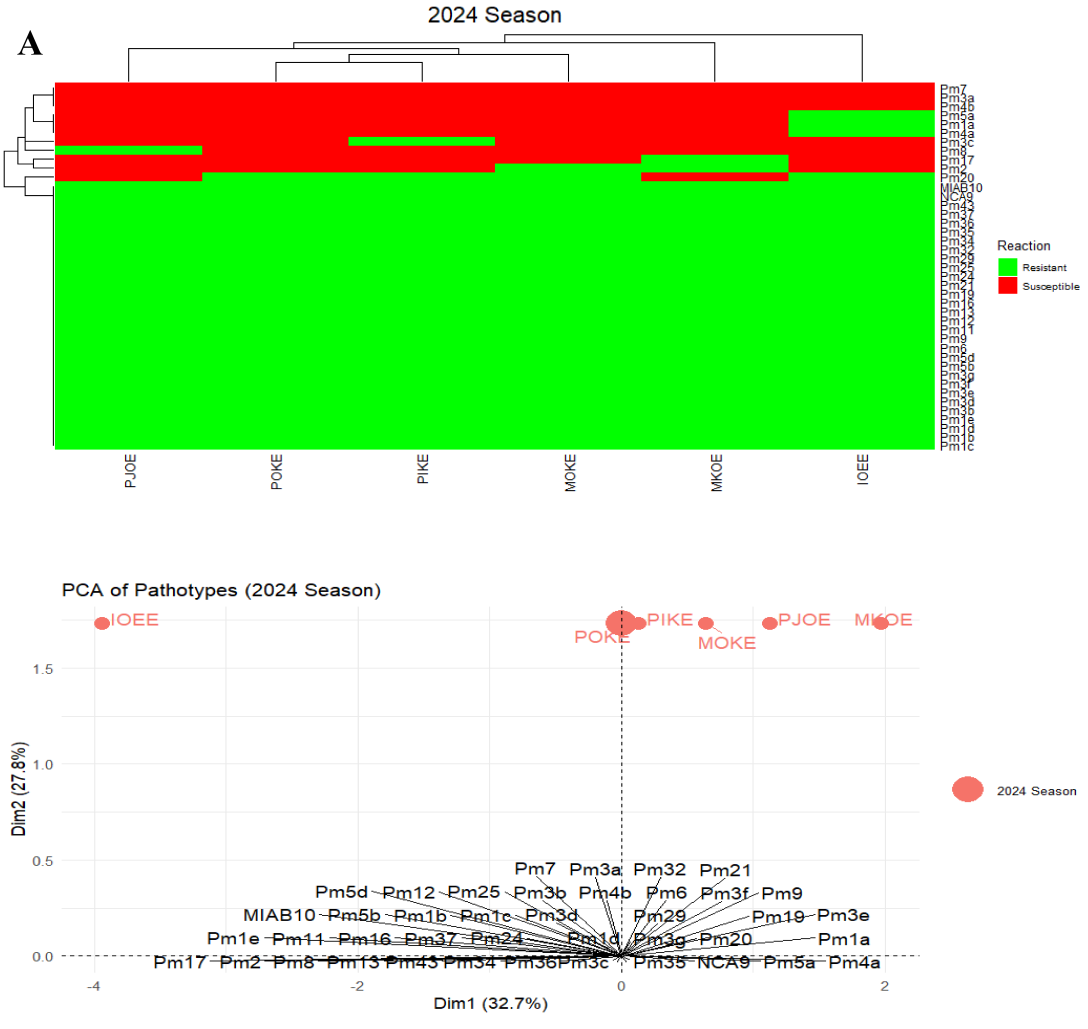


Figure 1. Multivariate analysis of six *Bgt* pathotypes evaluated across 41 *Pm* monogenic lines during the 2024 growing season. (**A**) Heatmap illustrating the infection response patterns and interaction profiles, where color intensity reflects disease reaction levels. (**B**) Principal component analysis (PCA) scatter plot showing the grouping and variance distribution among the tested pathotypes.

Four *Bgt* pathotypes, POOE, MOOE, PJOE, and POKE, were identified from 108 isolates collected in the 2025 season. (Table 7). The POKE pathotype was the most prevalent, accounting for 34.25%, whereas POOE was the least frequent (9.25%). The pathotypes POKE and MOOE showed the highest occurrence (%), 34.25% and 28.70%, respectively. Virulence % of the pathotypes MOOE, PJOE, and POKE were 62.50% for each, while the fourth pathotype (POOE) showed 68.75% (Figure 2A). Also, all of them ended by OE or KE letters, with virulence against the *Pm* genes 1a, 2, 3a, 4a, 3c, 8, 17, 5a, 20 and *Pm*7. Heatmap and PCA were performed based on the response of *Pm* genes to four identified pathotypes. The PCA scatterplot explained 48.8% of the variance, with PC1 (Dim1) accounting for 44.4% and PC2 (Dim2) explaining 44.4% (Figure 2B).

Table 7. Occurrence and virulence formulae of the *Bgt* phenotypes identified among 108 isolates collected in the 2025 season.

Pathotype	No. of Isolates	Occurrence%	Virulence/a Virulence Formulae	No. of Virulence Genes	Virulence %
POOE	10	9.25	1a,2,3a.4a,3c,8,17,4b,5a,20,7/	11	68.75
MOOE	31	28.70	1a,3a,4a,3c,8,17,4b,5a,20,7/	10	62.50
PJOE	30	27.77	1a,2,3a.4a,3c,17,4b,5a,20,7/	10	62.50
POKE	37	34.25	1a,2,3a.4a,3c,8,17,4b,5a,7/	10	62.50

Occurrence % = number of isolates that have the same pathotype/total no. of isolates. Virulence % = number of virulent genes/no. of genes used for identification.

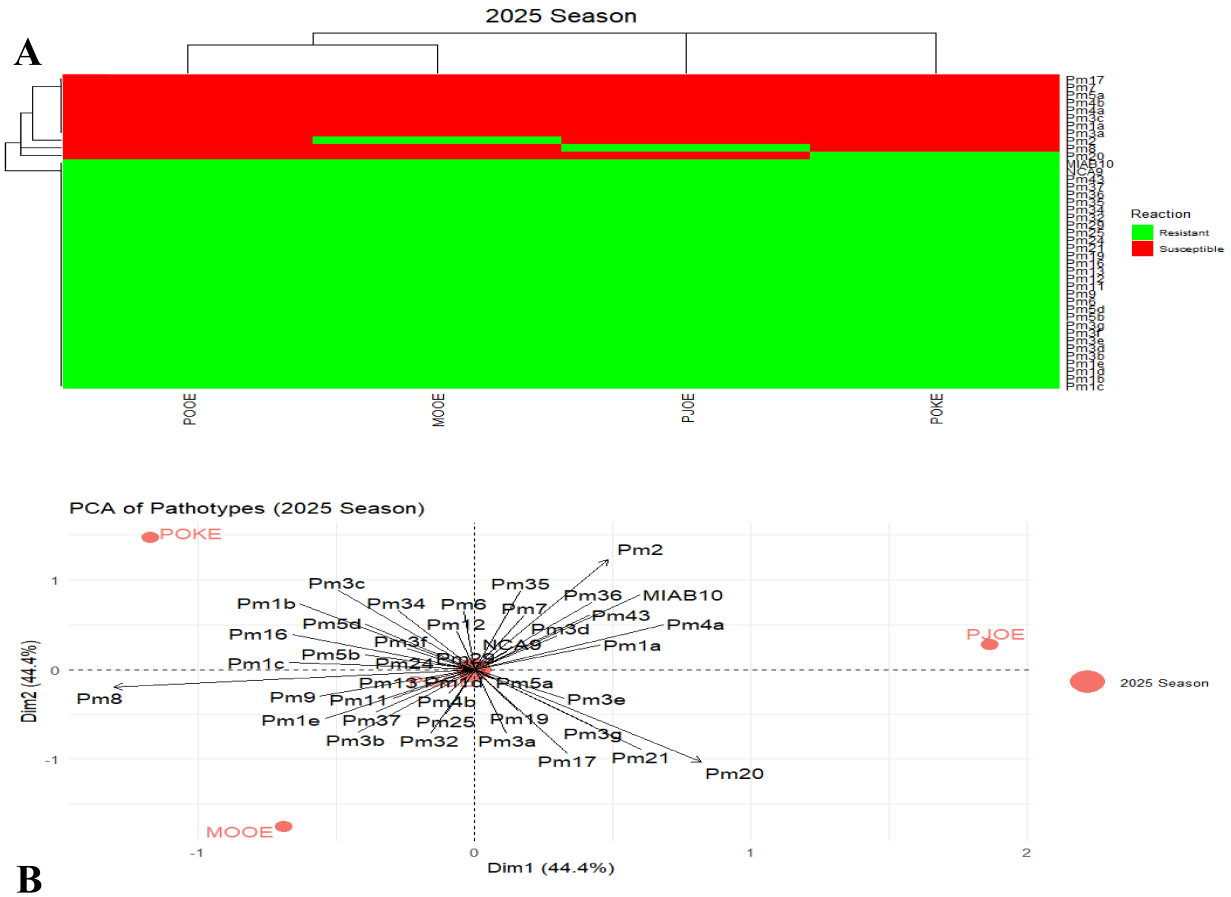


Figure 2. Multivariate analysis of four *Bgt* pathotypes evaluated across 41 *Pm* monogenic lines during the 2025 growing season. (**A**) Heatmap illustrating the infection response patterns and interaction profiles, where color intensity reflects disease reaction levels. (**B**) Principal component analysis (PCA) scatter plot showing the grouping and variance distribution among the tested pathotypes.

3.3. Virulence Diversity Shifts in Both Seasons

To investigate the diversity in wheat powdery mildew pathotypes in both seasons, Hill-number analysis was conducted. Hill-number analysis revealed a reduction in pathotype richness and diversity from 2024 to 2025, with richness decreasing from six to four pathotypes and Shannon and Simpson diversities declining from 5.90 to 3.73 and from 5.81 to 3.58, respectively (Table 8 and Figure 3). The test for homogeneity of multivariate dispersions was not significant (F = 0.836, p = 0.387), confirming comparable within-season variability (Table 9). PERMANOVA showed that season accounted for only 11.18% of the total variation in virulence profiles and did not significantly affect population structure (F = 1.007, p = 0.481). Likewise, the low mean character difference (MCD = 0.1894) and Roger's distance (R = 0.1914) indicated a high degree of similarity between the 2024 and 2025 pathogen populations (Table 10).

Table 8. Genetic diversity indices of *Blumeria graminis* f. sp. *tritici* pathotypes populations sampled during the 2024 and 2025 growing seasons. Metrics include the number of distinct phenotypes, Shannon–Wiener diversity index, Shannon, and Richness measures.

Season	Diversity	Observed	Estimator	S.E.	95% LCL	95% UCL
2024	Richness (q = 0)	6	6	0	6	6
2024	Shannon (q = 1)	5.79	5.90	0.146	5.61	6.18
2024	Simpson (q = 2)	5.622	5.81	0.244	5.30	6.31
2025	Richness (q = 0)	4	4	0	4	4
2025	Shannon (q = 1)	3.67	3.73	0.138	3.47	3.98
2025	Simpson (q = 2)	3.50	3.58	0.178	3.26	3.91

Stander error = S.E., lower confidence level (LCL), upper confidence level (UCL).

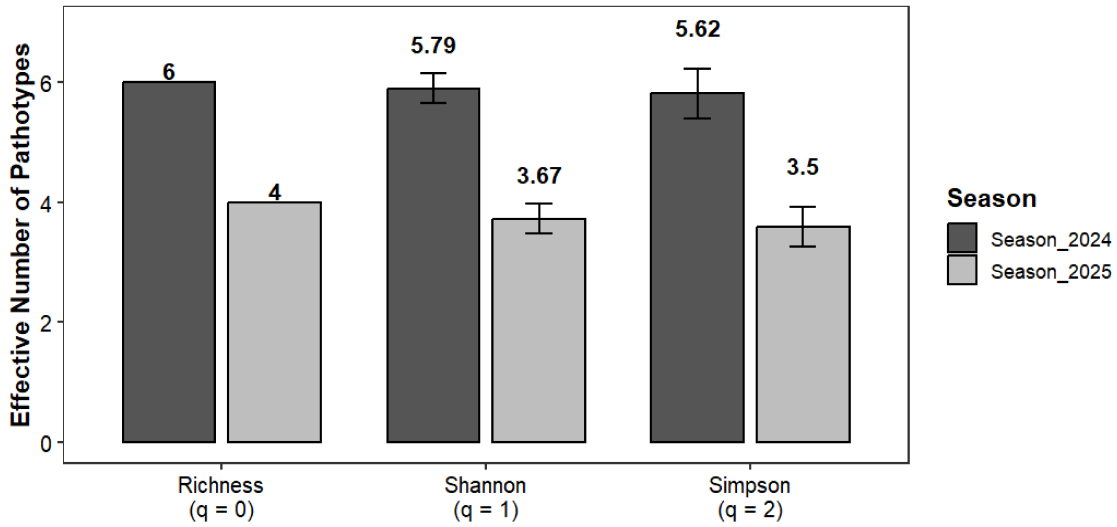


Figure 3. Diversity of wheat powdery mildew pathotypes during the 2024 and 2025 growing seasons based on Hill numbers.

Table 9. Beta dispersion and PERMANOVA comparisons of two *Bgt* pathotypes populations across the 2024 and 2025 growing seasons.

Beta Dispersion					
Source	**df**	**Sum. Sq**	**Mean. Sq**	**F. Value**	***p* Value**
Groups	1	0.0098	0.0098	0.836	0.387
Residuals	8	0.0938	0.0117		

Table 9. *Cont.*

Beta Dispersion					
PERMANOVA					
	df	**Sum. Sq**	**R^2**	**F**	***p* (>F)**
Model	1	0.0281	0.11	1.007	0.481
Residual	8	0.2229	0.888	-	-
Total	9	0.2510	1	-	-

Table 10. Genetic distance metrics for mean character difference (MCD) and Rogers distance (R) between the *Bgt* pathotypes in 2024 and 2025 seasons on differential lines (n = 41).

Pathotypes	**Mean Character Difference (MCD)**		**Rogers Distance (R)**	
	2024	**2025**	**2024**	**2025**
2024	0	0.1894	0	0.1914
2025	0.1894	0	0.1914	0

3.4. Response of the Pm to Bgt at Adult Stage

A significant difference was observed between resistant and susceptible monogenic lines in both seasons. Among the 41 evaluated *Pm* monogenic lines, 32 showed complete resistance at adult growth stage across both seasons, and nine were susceptible (Table 11). The resistant *Pm* genes were *Pm* 1d, 2, 3a, 3b, 3d, 3c, 3g, 4a, 4b, 5a, 5b, 6, 8, 12, 16, 17, 19, 20, 21 11, 13, 24, 25, 29, 32, 34, 35, 36, 37, 43, NCA9 and MIAB10 throughout both seasons. Meanwhile, the susceptible *Pm*s were *Pm* 1a, 1b, 1c, 1e, 3e, 3f, 5d, 7 and 9 in both seasons.

Table 11. Mean powdery mildew disease severity of 41 *Pm* monogenic lines at the adult stage across the 2024 and 2025 growing seasons.

No	***Pm* Gene**	**Mean Disease Severity Season 2024**	**Mean Disease Severity Season 2025**	**No**	***Pm* Gene**	**Mean Disease Severity Season 2024**	**Mean Disease Severity Season 2025**
1	*Pm*1a	9 ^{a*}	8 ^{b}	23	*Pm*11	0.00 ^{e}	0.00 ^{g}
2	*Pm*1b	7 ^{c}	8 ^{b}	24	*Pm*12	0.00 ^{e}	1 ^{f}
3	*Pm*1c	9 ^{a}	8 ^{b}	25	*Pm*13	0.00 ^{e}	0.00 ^{g}
4	*Pm*1d	0.00 ^{e}	2 ^{f}	26	*Pm*16	0.00 ^{e}	0.00 ^{g}
5	*Pm*1e	7 ^{c}	9 ^{a}	27	*Pm*17	0.00 ^{e}	3 ^{d}
6	*Pm*2	0.00 ^{e}	0.00 ^{g}	28	*Pm*19	0.00 ^{e}	0.00 ^{g}
7	*Pm*3a	3 ^{d}	0.00 ^{g}	29	*Pm*20	0.00 ^{e}	0.00 ^{g}
8	*Pm*3b	0.00 ^{e}	0.00 ^{g}	30	*Pm*21	0.00 ^{e}	0.00 ^{g}
9	*Pm*3c	0.00 ^{e}	0.00 ^{g}	31	*Pm*24	0.00 ^{e}	0.00 ^{g}
10	*Pm*3d	0.00 ^{e}	0.00 ^{g}	32	*Pm*25	0.00 ^{e}	0.00 ^{g}
11	*Pm*3e	8 ^{b}	9 ^{a}	33	*Pm*29	0.00 ^{e}	0.00 ^{g}
12	*Pm*3f	9 ^{a}	7 ^{c}	34	*Pm*32	0.00 ^{e}	0.00 ^{g}
13	*Pm*3g	0.00 ^{e}	0.00 ^{g}	35	*Pm*34	0.00 ^{e}	0.00 ^{g}
14	*Pm*4a	0.00 ^{e}	0.00 ^{g}	36	*Pm*35	0.00 ^{e}	0.00 ^{g}
15	*Pm*4b	0.00 ^{e}	0.00 ^{g}	37	*Pm*36	0.00 ^{e}	0.00 ^{g}
16	*Pm*5a	0.00 ^{e}	0.00 ^{g}	38	*Pm*37	0.00 ^{e}	0.00 ^{g}
17	*Pm*5b	0.00 ^{e}	2 ^{e}	39	*Pm*43	0.00 ^{e}	0.00 ^{g}
18	*Pm*5d	7 ^{c}	7 ^{c}	40	NCA9	0.00 ^{e}	0.00 ^{g}
19	*Pm*6	0.00 ^{e}	0.00 ^{g}	41	MIAB10	0.00 ^{e}	0.00 ^{g}
20	*Pm*7	8 ^{b}	9 ^{a}		Chancellor	9	9
21	*Pm*8	0.00 ^{e}	0.00 ^{g}		Coker 68-15	9	9
22	*Pm*9	9 ^{a}	8 ^{b}				
LSD at 5%		0.01	0.14	LSD at 5%		0.01	0.14

* The numbers with different letters mean there are significant differences between the disease severities of the wheat genotypes.

3.5. Response of the New Wheat Genotypes to Bgt Pathogen

Twelve new wheat genotypes were evaluated against powdery mildew disease at both stages in both seasons. Significant differences were observed among the evaluated wheat genotypes at the seedling stage, with Sakha96 being the only genotype resistant to all tested *Bgt* isolates (Table 12). In contrast, the other eleven genotypes showed high susceptibility, with scores ranging from 6 to 9 on the scale (Figure 4). However, at the adult stage, Misr4, Misr5, Sakha95, Sakha96, Sids15, and Gemmeiza13 genotypes showed an immune reaction. While Misr3, Misr6, Misr7, and Sakha97 genotypes showed susceptible reaction (7 scale), Misr1 and Misr2 were highly susceptible (9 scale).

Table 12. Assessment of mean powdery mildew disease severity percentages evaluated on 12 new bread wheat genotypes at both the seedling and adult plant stages across the 2024 and 2025 growing seasons.

Wheat Genotype	Reaction to *Bgt* Pathogen	
	At Seedling Stage	At Adult Stage
Misr1	9 ^{a*}	9 ^{a}
Misr2	7 ^{c}	9 ^{a}
Misr3	7 ^{c}	7 ^{b}
Misr4	7 ^{c}	0 ^{c}
Misr5	9 ^{a}	0 ^{c}
Misr6	8 ^{b}	7 ^{b}
Misr7	6 ^{d}	7 ^{b}
Sakha95	7 ^{c}	0 ^{c}
Sakha96	0 ^{e}	0 ^{c}
Sakha97	8 ^{b}	7 ^{b}
Sids15	8 ^{b}	0 ^{c}
Gemmeiza13	6 ^{d}	0 ^{c}
LSD at 0.05%	0.2	0.3

* Numbers followed by different letters indicate significant differences among the mean disease severities of the wheat genotypes.

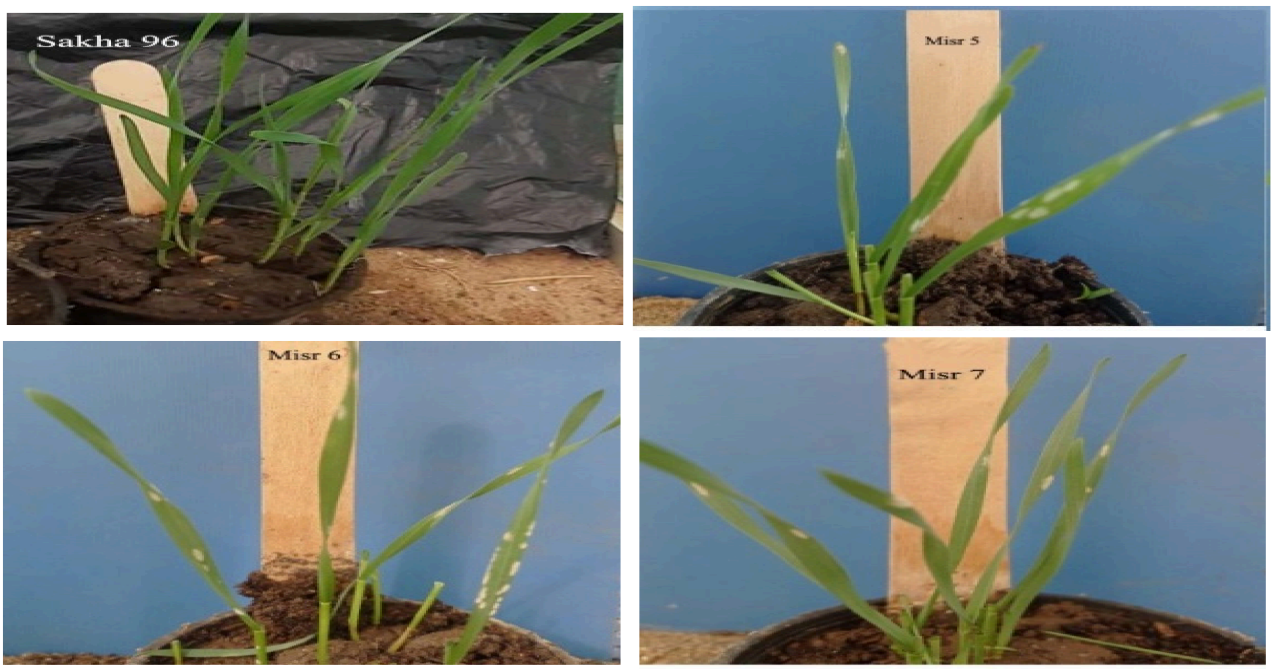


Figure 4. Response of Sakha96 and Misr5, Misr6, and Misr7 wheat genotypes to powdery mildew pathogen under conditioned glass house conditions at seedling stage.

3.6. Molecular Detection of Resistance Pm Genes

This study used three closely linked primers, Xgwm337, Xcfd7, and Xgwm332, to detect *Pm*24, *Pm*35, and *Pm*37 in twelve new wheat genotypes. Specific bands of 200, 251, and 193 bp correspond in size to the diagnostic controls for *Pm*24, *Pm*35, and *Pm*37, respectively. However, the analysis of the PCR profiles showed that the diagnostic marker bands associated with *Pm*24, *Pm*35, and *Pm*37 were not detected in any of the tested wheat genotypes (Figures 5–7). Additional polymorphic bands were amplified by Xgwm337 (350 and 150 bp), xcfd7 (200 bp) and xgwm332 (230 bp). The additional bands were used to construct the relationships among the wheat genotypes (Table 13 and Figure 8). These results indicate the presence of marker alleles corresponding in size to those associated with the respective resistance loci. Based on UPGMA cluster analysis, the genotypes were grouped into two main clusters. The first cluster comprised the highly resistant Sakha96 and the adult-stage-resistant Sids15, together with several highly susceptible cultivars (Sakha97, Misr1, Misr3, and Misr7). The second cluster also comprised adult-stage-resistant varieties together with susceptible cultivars (Misr6 and Misr2).

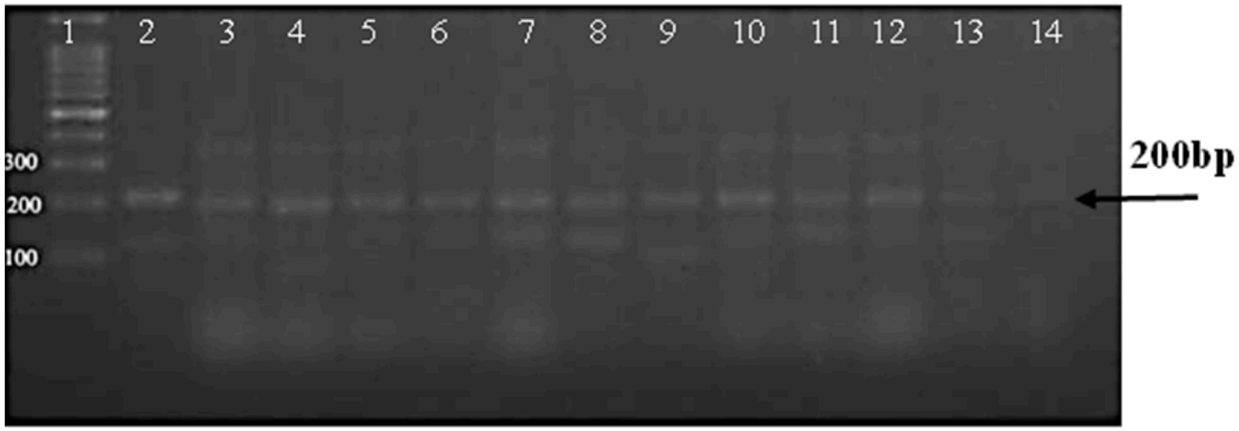


Figure 5. *Pm*24 linked primer (200 bp) and 12 wheat genotypes. 1: 100 bp DNA ladder RTU (Gene Direx), 2: *Pm*24, 3: Misr1, 4: Misr2, 5: Misr3, 6: Misr4, 7: Misr5, 8: Misr6, 9: Misr7, 10: Sakha95, 11: Sakha 96, 12: Sakha97, 13: Sids15, 14: Gemmeiza13.

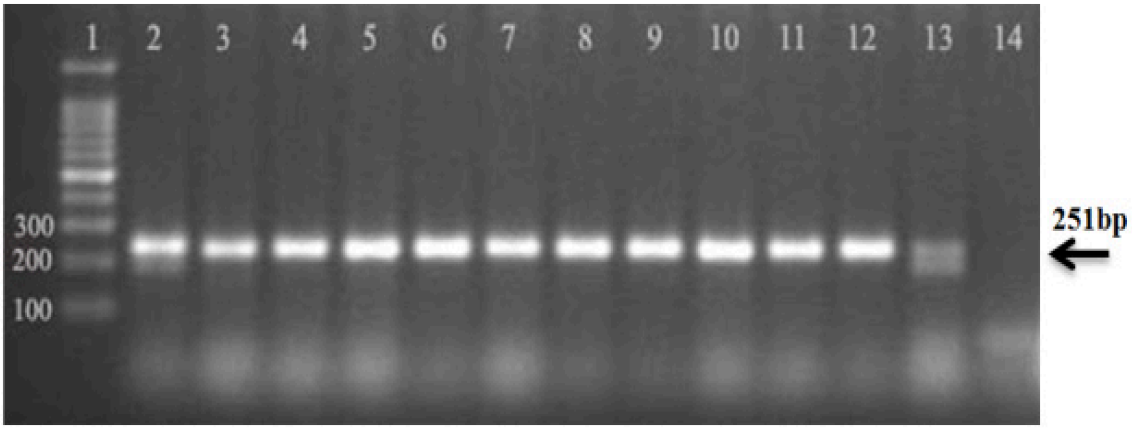


Figure 6. *Pm*35 linked primer (251 bp) and 12 wheat genotypes. 1: 100 bp DNA ladder RTU (Gene Direx), 2: *Pm*35, 3: Misr1, 4: Misr2, 5: Misr3, 6: Misr4, 7: Misr5, 8: Misr6, 9: Misr7, 10: Sakha95, 11: Sakha96, 12: Sakha97, 13: Sids15, 14: Gemmeiza13.

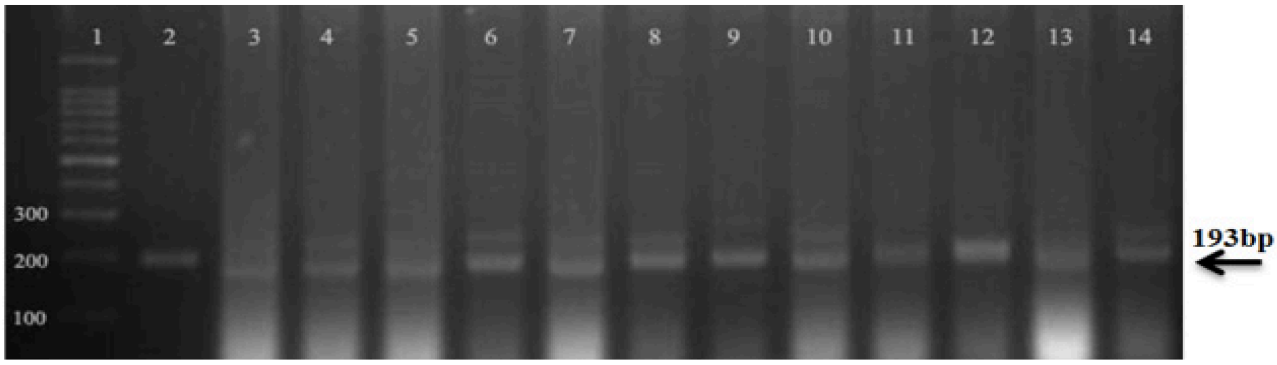


Figure 7. *Pm37* linked primer (193 bp) and 12 wheat genotypes. 1: 100 bp DNA ladder RTU (Gene Direx), 2: *Pm37*, 3: Misr1, 4: Misr2, 5: Misr3, 6: Misr4, 7: Misr5, 8: Misr6, 9: Misr7, 10: Sakha95, 11: Sakha96, 12: Sakha97, 13: Sids15, 14: Gemmeiza13.

Table 13. Pairwise genetic similarity matrix among the twelve bread wheat genotypes based on molecular marker data. Values represent the calculated similarity coefficients between pairs of cultivars, with diagonal values indicating self-comparison (1.000).

	Misr1	**Misr2**	**Misr3**	**Misr4**	**Misr5**	**Misr6**	**Misr7**	**Sakha95**	**Sakha96**	**Sakha97**	**Sids15**	**Gemmeiza13**
Misr1	1.000											
Misr2	0.800	1.000										
Misr3	1.000	0.800	1.000									
Misr4	0.600	0.800	0.600	1.000								
Misr5	0.667	0.833	0.667	0.667	1.000							
Misr6	0.500	0.667	0.500	0.800	0.833	1.000						
Misr7	0.750	0.600	0.750	0.750	0.500	0.600	1.000					
Sakha95	0.667	0.833	0.667	0.667	1.000	0.833	0.50	1.000				
Sakha96	0.800	0.667	0.800	0.500	0.833	0.667	0.60	0.833	1.000			
Sakha97	1.000	0.800	1.000	0.600	0.667	0.500	0.75	0.667	0.800	1.000		
Sids15	0.600	0.500	0.600	0.600	0.667	0.800	0.75	0.667	0.800	0.600	1.000	
Gemmeiza13	0.500	0.667	0.500	0.800	0.571	0.667	0.60	0.571	0.429	0.500	0.500	1.000

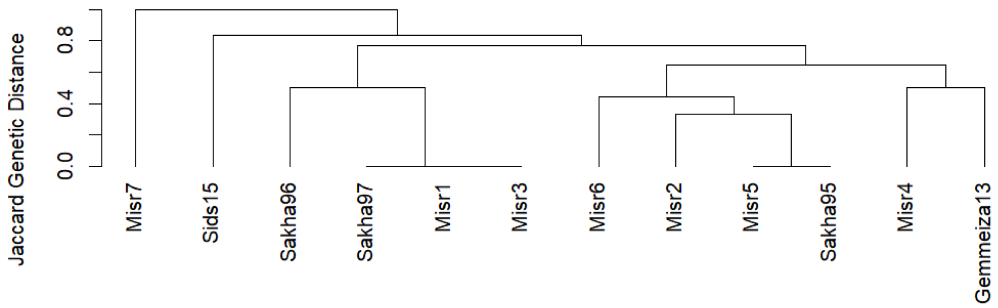


Figure 8. UPGMA cluster analysis, showing the genetic relationships and hierarchical clustering among the twelve bread wheat genotypes based on simple sequence repeat (SSR) marker data.

3.7. Pedigree Analysis of Pm Genes

To assess the ancestral relation between *Pm24*, *Pm35* and *Pm37* and the 12 Egyptian wheat genotypes, R packages were used. Data showed common ancestors between *Pm35* and *Pm37* and the wheat genotypes (Figures 9 and 10), while *Pm24* does not have a cleared pedigree.

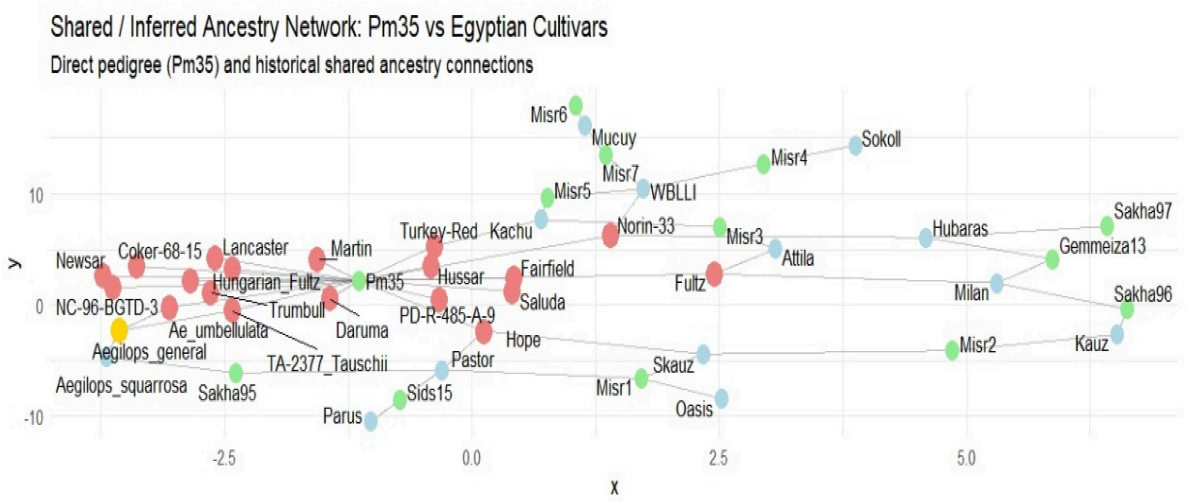


Figure 9. Shared/inferred ancestry network between *Pm*35 and Egyptian wheat cultivars. Green, blue, red, and yellow nodes represent Egyptian cultivars, Egyptian ancestors, *Pm*35 ancestors, and shared backbone ancestors, respectively. Lines indicate direct pedigree relationships or historically inferred shared ancestry connections.

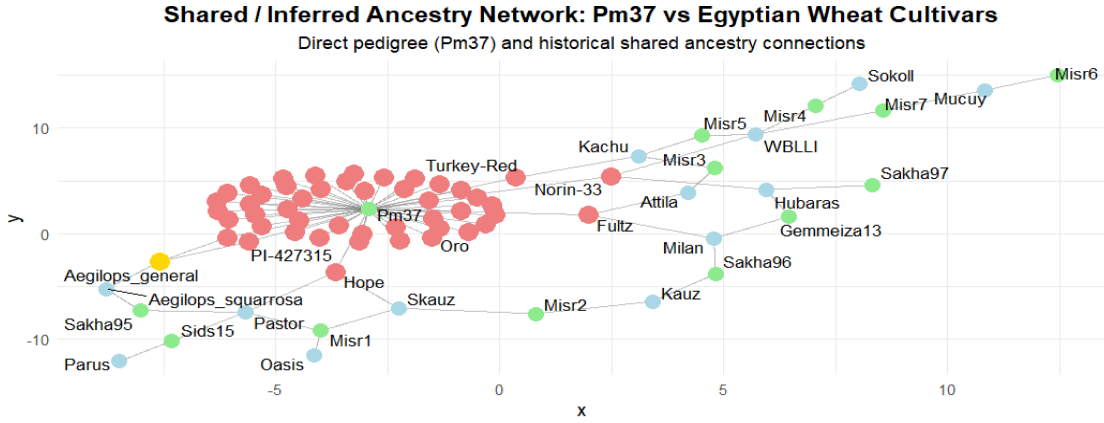


Figure 10. Shared/inferred ancestry network between *Pm*37 and Egyptian wheat cultivars. Green, blue, red, and yellow nodes represent Egyptian cultivars, Egyptian ancestors, *Pm*37 ancestors, and shared backbone ancestors, respectively. Lines indicate direct pedigree relationships or historically inferred shared ancestry connections.

4. Discussion

Among the 41 evaluated *Pm* monogenic lines, *Pm*11, 13, 24, 25, 29, 32, 34, 35, 36, 37, *Pm*43, NCA9, and MIAB10 (31.70%) demonstrated complete resistance at both seedling and adult plant stages across both seasons. Of these, *Pm*13, 24, 35, 36, and 37 were previously reported as completely resistant under Egyptian conditions [31,76], while the complete resistance of *Pm*11, 25, 29, 32, 34, *Pm*43, NCA9, and MIAB10 against Egyptian *Bgt* populations is documented here. Their consistent performance across developmental stages and seasons implies stable, potentially durable resistance, a prerequisite for breeding cultivars with long-term disease management efficacy. In contrast, 21 lines, *Pm*1d, 2, 3a, 3b, 3c, 3d, 3g, 4a, 4b, 5a, 5b, 5d, 6, 8, 12, 16, 17, 19, 20, and 21, were susceptible at the seedling stage yet resistant at the adult plant stage, indicative of adult plant resistance (APR). Previous Egyptian studies covered only a subset of these lines, with an APR, while *Pm*8 and *Pm*9 were fully susceptible at both stages. Notably, the APR responses now observed for several lines previously classified as completely resistant [31,76] provide direct evidence of ongoing pathogen evolution under Egyptian conditions. Some *Pm*s, such

as *Pm*21, *Pm*37, and *Pm*4b, which have consistently conferred complete or near-complete resistance across *Bgt* populations in China and the United States [77,78], exhibited variable responses in Egypt. Collectively, these findings highlight that resistance gene effectiveness cannot be predicted across regions without local validation, reinforcing the necessity of continuous, region-specific virulence monitoring to anticipate pathotype shifts and guide evidence-based resistance gene deployment strategies.

The nomenclature system was adapted from cereal rust classification and provided a standardized framework for identifying and comparing pathogen races over time and across regions [72,79]. Such a standardized system is crucial because the number of pathotypes increases rapidly with more resistance genes, complicating communication and data interpretation. Moreover, it provided an effective, logical, and concise method capable of illustrating relationships among pathotypes [80]. The obtained data revealed that six (POKE, PJOE, MKOE, PIKE, MOKE, and IOEE) and four (POOE, MOOE, PJOE, and POKE) pathotypes were identified in the 2024 and 2025 seasons. Results also showed a significant increase in virulence of pathotypes to *Pm* genes 1a, 2, 3a, 4a, 3c, 8, 17, 5a, and *Pm*7. Moreover, all pathotypes shared the KE, OE, and EE suffixes, suggesting shared characteristics with previous pathotypes documented in Egypt [11]. The observed decline in pathotype richness from six to four between seasons was accompanied by a significant reduction in diversity indices: Shannon diversity decreased from 5.90 to 3.73, and Simpson diversity decreased from 5.81 to 3.59. This reduction in pathotype complexity, coupled with the emergence of a single dominant pathotype (POKE) in 2025, may reflect competitive exclusion under directional selection, wherein highly virulent genotypes conferring fitness advantages on prevalent host cultivars displace less competitive pathotypes within the population. Similar results were found by [81], who monitored *Bgt* populations in spring wheat-growing regions of Northeast China from 2004 to 2010 and found that the dominant race varied across three time periods and virulence to *Pm*2, *Pm*4b, *Pm*2+6, *Pm*4+8, *Pm*12, *Pm*16, *Pm*18, *Pm*21, *Pm*22, and *Pm*23 remained relatively low, ranging from 0 to 26.5%. Regional variation in pathotype diversity was further evidenced by [82], who identified 32 and 30 pathotypes in Lithuanian and Ukrainian *Bgt* populations, respectively, with NGDE and NGKE as their corresponding dominant types, highlighting the extent of geographic structuring within European *Bgt* metapopulations. Despite the inter-seasonal decline in pathotype number observed in the present study, PERMANOVA analysis revealed no statistically significant difference in overall virulence composition between the 2024 and 2025 populations ($R^2 = 0.112$, $p = 0.481$), with low genetic differentiation values (mean character difference = 0.1894; Roger's distance = 0.1914), indicating limited seasonal differentiation in virulence composition. Collectively, these findings suggest that, although pathotype diversity declined in 2025, the overall virulence composition remained largely stable between seasons.

Heatmap and PCA were used to analyze the virulence patterns of powdery mildew pathotypes and their interactions with 41 *Pm* genes during the 2024 and 2025 seasons. PCA analysis revealed differences in the structure of powdery mildew pathotypes between 2024 and 2025, with higher variance in 2025 (88.8%) compared to 2024 (70.5%). In 2024, most pathotypes were closely clustered, suggesting similar virulence patterns, with only a few distinct types. In contrast, 2025 showed clear separation of specific pathotypes (e.g., PJOE, POKE, MOOE), indicating increased diversity and possible pathogen adaptation. The tighter clustering of *Pm* genes in 2024 suggests limited discrimination among pathotypes, whereas their wider distribution in 2025 indicates greater effectiveness in differentiating virulence profiles. Overall, these results suggest a dynamic pathogen population, highlighting the importance of continuous monitoring and the use of diverse resistance genes to ensure durable wheat resistance. Annual surveys for prevalence and virulence for the *Bgt*

population must be the target of wheat pathologists to draw a strategy for maintaining durable resistance to the disease in wheat cultivars.

In the current study, the Sakha96 wheat variety exhibited high resistance (reaction 0) during both the seedling and adult stages across both seasons. This suggests the presence of race-nonspecific or broad-spectrum resistance in Sakha96 makes it an excellent choice for further wheat breeding programs. However, the wheat genotypes Misr4, Misr5, Sakha95, Sids15 and Gemmeiza13 showed susceptibility at the seedling stage and complete resistance at the adult stage, meaning they have adult plant resistance (quantitative resistance). Such APR resistance is crucial in mature plants but may not be expressed effectively during the seedling stage [83,84], so it is more durable than seedling resistance for durable disease control under field conditions [85]. The remaining six genotypes showed susceptibility at both developmental stages, indicating a lack of currently effective resistance genes or the presence of resistance alleles whose expression is suppressed under the virulence spectrum of prevailing *Bgt* pathotypes. These results are consistent with those of [86], who evaluated 370 Indian varieties under polyhouse conditions using a mixture of natural *Bgt* pathotypes and found that only 23 varieties were fully immune, while the majority exhibited partial or no resistance, highlighting the challenge of identifying varieties with broad and stable resistance across diverse pathotype backgrounds.

To fight powdery mildew disease, it is essential to utilize and determine resistance genes and unravel the genetic basis for this resistance [87]. Molecular markers serve in breeding programs for marker-assisted selection and gene pyramiding, as they are not influenced by environmental variability [16]. SSR (simple sequence repeat) markers, with their high polymorphism and gene specificity, offer a practical and reliable approach for assisting in the identification and selection of resistant genotypes [88]. SSR markers are used to confirm the presence of target genes and provide insight into the genetic background in selected varieties [89]. Three SSR markers linked to different *Pm* genes were deployed to identify the selected resistant genes in the new release cultivars. The markers were Xgwm337, Xcfd7, and Xgwm332, linked to *Pm*24, *Pm*35, and *Pm*37, respectively. *Pm*24, *Pm*35, and *Pm*37 were selected because they are conferring stable resistance under Egyptian field conditions [74,76] and derived from cultivars rather than breeding lines, thereby reducing the agronomic penalties associated with linkage drag. Actually, in breeding programs, it is generally preferred to introgress *Pm* genes from cultivars or advanced breeding lines because they possess superior agronomic traits with linkage drag reduction [90,91]. SSR analysis detected marker alleles associated with *Pm*24, *Pm*35 and *Pm*37 in all examined genotypes. Emara [74] reported that, although Mis1 and Misr2 showed susceptibility at both stages to powdery mildew, they contain the *Pm*24, *Pm*35, and *Pm*37 resistance genes. These findings could be explained by the negative expression of resistance in cultivars, which may be affected by their genetic backgrounds and/or environmental conditions. Consequently, the reliability and effectiveness of these markers across diverse genetic backgrounds require validation before their broad application [92]. Moreover, the SSR markers used in this study were developed and validated in specific genetic backgrounds distinct from those of modern Egyptian cultivars, raising the possibility of imperfect co-segregation between the marker allele and the functional resistance allele due to historical recombination. Therefore, pedigree analysis of *Pm*24, *Pm*35, and *Pm*37 across the 12 Egyptian wheat genotypes was performed to investigate their genetic backgrounds. The widespread detection of *Pm*35 and *Pm*37 among the evaluated Egyptian wheat genotypes suggests a possible shared genetic background; however, the available pedigree information and marker data are insufficient to establish a common ancestral origin for these two resistance genes. Although *Pm*24 originated from the Chinese landrace bread wheat cultivar Chiyacao, whose pedigree remains unclear [93,94], it was detected in all 12 Egyptian bread wheat

genotypes. This widespread occurrence may reflect a shared genetic background resulting from the extensive use of related germplasm in international wheat-breeding programs, including CIMMYT-derived breeding materials that have contributed to the development of several Egyptian wheat genotypes. In contrast, *Pm*35 and *Pm*37 showed more distant relationships, with *Pm*35 tracing to CIMMYT lineages involving Hope and Fultz and *Pm*37 associated with the broader Hope–Norin–Fultz–Turkey Red lineage to modern bread wheat. SSR marker data were used to assess the genetic relationships among the twelve Egyptian wheat genotypes. UPGMA cluster analysis grouped the genotypes into two main clusters that did not correspond to their phenotypic resistance profiles. The first cluster comprised the highly resistant Sakha96 and the adult-stage-resistant Sids15, together with several highly susceptible cultivars (Sakha97, Misr1, Misr3, and Misr7). The second cluster likewise comprised adult-stage-resistant varieties together with susceptible cultivars (Misr6 and Misr2). This clustering pattern indicates substantial genetic similarity among some of the tested genotypes, irrespective of their phenotypic resistance profiles. This situation underscores the urgent need to broaden the genetic diversity of resistance sources deployed in Egyptian wheat breeding programs. This can be achieved by prospecting for novel *Pm* genes from synthetic wheats, wild relatives, and geographically distant germplasm collections that remain unexploited in current local programs, as well as by integrating pathological and molecular data. Several limitations should be considered when interpreting the present findings. First, the molecular analysis included a relatively limited number of Egyptian wheat cultivars, which may not fully represent the genetic diversity of the broader Egyptian wheat germplasm. Second, pathogen sampling was concentrated within the surveyed regions, and therefore, the identified *B. graminis* f. sp. *tritici* pathotypes may not represent the complete virulence spectrum occurring throughout Egypt. Third, the pathotype analysis covered only two growing seasons, and temporal fluctuations in pathogen populations may occur across years in response to environmental conditions, host deployment, and selection pressure. Consequently, longer-term and geographically broader surveillance involving larger numbers of cultivars and isolates will be required to determine the stability and wider distribution of the observed resistance and virulence patterns. Finally, although the molecular markers provided evidence for the presence of *Pm35* and *Pm37*-associated loci, marker detection alone does not definitively establish the functional identity or effective pyramiding of these resistance genes.

5. Conclusions

The current study revealed that 13 *Pm* genes have all-stages resistance and 20 have adult resistance to *Bgt*. Out of 12 new bread wheat genotypes, only Sakha96 showed a complete resistance at both stages, while Misr4, Misr5, Sakha95, Sakha96, Sids15, and Gemmeiza13 genotypes showed adult plant resistance. Molecular detection of the three *Pm* genes revealed that *Pm*24, *Pm*35 and *Pm*37 are absent in all tested wheat genotypes. Also, pedigree analysis showed an ancestral relationship between the Egyptian wheat genotypes and *Pm*35 and 37. The widespread susceptibility among local genotypes highlights an urgent need to incorporate durable resistance sources into Egyptian wheat improvement programs. Consequently, gene pyramiding of *Pm*24, *Pm*35, and *Pm*37 via marker-assisted backcrossing (MABC) is strongly recommended to develop broad-spectrum, durable resistance against evolving *Bgt* populations.

Supplementary Materials: The following supporting information can be downloaded at https://www.mdpi.com/article/10.3390/ijpb17100098/s1, Table S1: Detailed pedigree for *Pm*24, *Pm*35 and *Pm*37; Table S2: Pedigree for some Egyptian wheat genotypes Misr1, Misr 2 and Sakha and Gemmeiza13. Detailed pedigree for some Egyptian wheat genotypes Misr1, Misr 2 and Sakha and

Gemmeiza13 downloaded from CIMMYT database (https://www.cimmyt.org/about/data/?utm_source=chatgpt.com, accessed on 11 May 2025).

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