Evidence of a distinct *Blumeria graminis* f. sp. *tritici* pathotype structure in Australian wheat powdery mildew: implications for resistance breeding

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Abstract

The obligate biotrophic fungus *Blumeria graminis* f. sp. *tritici* (*Bgt*) is the causal agent of wheat powdery mildew. To investigate virulence diversity, 30 Australian *Bgt* isolates collected between 2020 and 2024 were screened against a panel of 24 wheat lines carrying defined resistance (*R*) genes. Pathotyping analysis indicated that the Australian *Bgt* population is diverse in its virulence profile. Non-metric multidimensional scaling analysis of disease phenotyping revealed evidence of six distinct *Bgt* pathotype groups. In addition, we identified wheat varieties carrying defined *R* genes and their combinations that remain effective against the contemporary Australian *Bgt*. Notably, wheat cvs Khapli and Tabasco, possessing

Pm4a and *Pm2,* respectively, in addition to unknown *R* gene(s), were highly resistant to all the isolates tested. Furthermore, elite European varieties that possess durable resistance, such as Alchemy and Cordiale, display complete to moderate resistance against all selected isolates representing the Australian pathotype groups.

Key words: *Blumeria graminis* f. sp. *tritici,* powdery mildew resistance, pathotype structure, virulence diversity, resistance breeding.

The obligate fungal pathogen *Blumeria graminis* f. sp. *tritici* (*Bgt*) causes wheat powdery mildew (WPM) that results in typical yield losses of 10–15% and up to 50% under favourable conditions (Rana et al. 2022). In Australia, around 12.6 million hectares are sown with wheat annually, providing a large breeding ground for *Bgt* (Department of Agriculture 2025). The fungus develops haustoria in epidermal cells to extract nutrients and reproduces asexually through airborne conidia, while chasmothecia are formed sexually under stress or at the end of the season (Spanu 2014; Wicker et al. 2013). The deployment of genetic resistance in wheat is an environmentally sustainable strategy to minimize fungicide dependence and mitigate associated ecological impacts. Resistance to WPM comprises race-specific resistance, mediated by major WPM resistance (*R/Pm*) genes that induce hypersensitive responses, and adult-plant or partial resistance, mostly a slow-mildewing form associated with broad-spectrum and durable protection (Alam et al. 2011; Ge et al. 2021). To date, over 100 *Pm* genes and allelic variants (*Pm1-Pm71*) conferring major and minor resistance to *Bgt* have been characterized in wheat, but only a small subset of these have been cloned (Kunz et al. 2025; Li et al. 2025). To achieve durable resistance, resistance-associated quantitative trait loci must

47 exhibit consistent effects across multiple years and diverse environmental conditions,
48 while maintaining stable expression throughout plant development (Kang et al. 2020).

49 Most wheat cultivars currently grown in Australia exhibit partial to complete
50 susceptibility to *Bgt* (Shackley et al. 2025). The limited presence of widespread
51 resistance to WPM in current germplasm highlights the urgent need for improvement,
52 especially in light of proliferating fungicide resistance (Ireland et al. 2022).
53 Demethylation inhibitor (DMI) and quinone-outside inhibitor resistance have recently
54 become widespread in Eastern Australia, and DMI-resistance mutations have also
55 been detected in Western Australia, as reported by the Australian Fungicide
56 Resistance Extension Network (2025). This underscores declining fungicide efficacy
57 against WPM (GRDC 2025; Lopez-Ruiz et al. 2023).

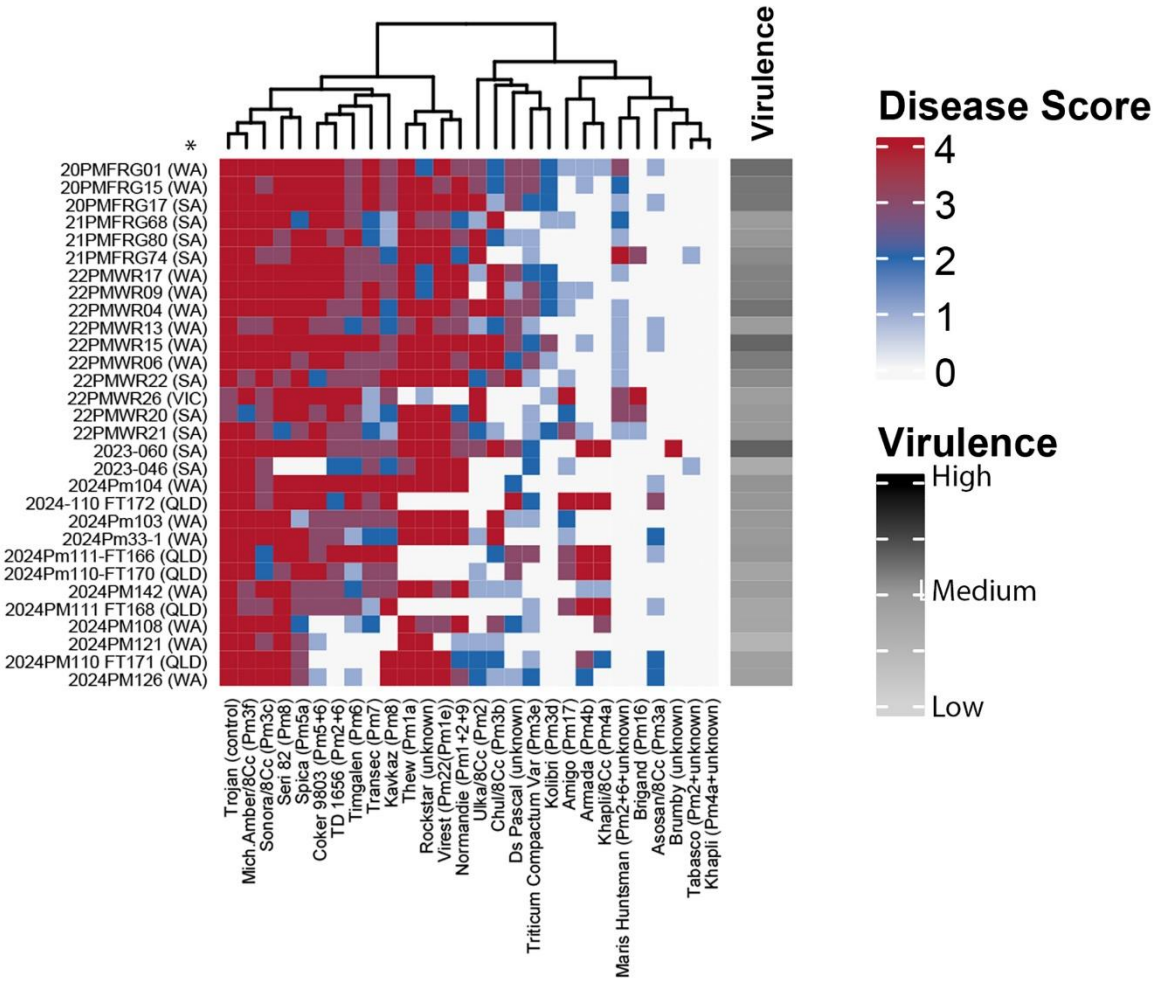
58 Race-specific resistance underlies the existence of pathotypes, or variants with a
59 distinct virulence pattern on host genotypes (Thrall et al. 2001). Monitoring WPM
60 pathotype structure across Australian agro-ecological zones is crucial for tracking
61 virulence variation and guiding effective, region-specific resistance deployment in
62 geographically isolated growing regions, as different commercial wheat varieties
63 adopted by Australian regions affect *Bgt* adaption to varietal differences (Brown and
64 Harris 2020; Shackley et al. 2023). The aims of this study were to determine the
65 pathotypes present in Australia, analyse virulence differences among geographic
66 locations, and determine the resistance status of selected sources of *Pm* genes
67 against the current Australian *Bgt* population.

68 Thirty *Bgt* isolates were collected from major wheat-growing regions across Australia
69 between 2020 and 2024 (Supplementary Table 1 and Supplementary Figure 1).
70 Monoconidial isolates were established and maintained on the susceptible wheat cv.

71 Trojan using a high-throughput detached leaf assay (DLA) method for infection studies
72 as previously described (Lopez-Ruiz et al. 2023; Tucker et al. 2013). To assess
73 pathogenicity, a panel of 24 wheat varieties carrying different combinations of defined
74 *Pm* genes sourced from the Australian Grains Genebank (AGG) was evaluated to
75 identify effective *Pm* genes and determine any evidence of local *Bgt* pathotypes
76 (Supplementary Table 2). Selected Australian commercial wheat lines were also
77 evaluated against WPM. These varieties are Brumby (rated as resistant except to a
78 pathotype), DS Pascal (resistant to moderately resistant), Rockstar (Moderately
79 susceptible to susceptible) and Trojan (susceptible) (Shackley 2022). All wheat
80 varieties were sown in individual pots with three replicates and grown in an
81 environment-controlled growth chamber. Seven-day-old seedling leaf sections were
82 then excised and placed onto benzimidazole agar plates. Isolate inoculation was
83 performed using an inoculation tower and plates were incubated at 20°C under a
84 12:12 h light:dark photoperiod as previously described (Tucker et al. 2013). Disease
85 severity was scored at seven days past infection based on a five-point (0-4) infection
86 type (IT) scale (Supplementary Figure 2). 0 and 1 were considered highly resistant
87 (R), 2 moderately resistant (MR), 3 moderately susceptible (MS), and 4 highly
88 susceptible(S) (Li et al. 2019).

89 We used a heatmap approach to visualise the virulence profile of all 30 *Bgt* isolates
90 and observed significant variations in the virulence profiles (Figure 1). Isolates 2023-
91 060 (SA), 22PMWR15 (WA) and 20PMFRG01 (WA) were highly virulent on the wheat
92 panel, whereas 2024PM121 (WA), 2023-046 (SA), 2024PM111 FT168 (QLD) and
93 2024PM108 (WA) were the least. Wheat cvs Khapli and Tabasco, both carrying an
94 unknown *Pm* gene as well as *Pm4a* and *Pm2,* respectively, exhibited complete
95 resistance to all the isolates tested. The Australian wheat cv Brumby possesses the

highest disease resistance rating against WPM. However, resistance was overcome by the highly virulent isolate 2023-060 from a field in Malinong (SA), as was previously observed in another field location elsewhere in SA (Shackley 2022). Despite this, our study indicates that wheat cvs Khapli and Tabasco possess highly effective resistance genes against isolate 2023-060 (Supplementary Table 3). Wheat cvs Mich.Amber/8Cc, Seri 82, and Sonora/8Cc possess *Pm3f*, *Pm8* and *Pm3c,* respectively, and displayed the lowest levels of resistance. Thus, these *Pm* genes are ineffective against the current Australian *Bgt* population. It was observed that cv Ulka/8Cc, possessing *Pm2* (Briggle 1969), shows a mixed resistance response to the isolate panel, whereas Tabasco, also possessing *Pm2* (Wu et al. 2023), is resistant to all isolates. This strongly suggests that cv Tabasco carries an unknown *Pm* gene in addition to *Pm2*. Likewise, although cvs Kavkaz and Seri 82 both carry *Pm8* (Hurni et al. 2013; Villareal et al. 1998), their contrasting responses suggest the presence of additional resistance in Kavkaz.



* WA = Western Australia, SA = South Australia, VIC = Victoria, QLD = Queensland

Figure 1: Virulence profiling of Australian *Bgt* isolates on differential and commercial wheat lines. The heatmap represents the virulence spectrum of *Bgt* isolates (*x*-axis) tested on a set of differential wheat lines (*y*-axis). The heatmap was generated using *ComplexHeatmap* package (ver.2.22.0) in R (ver.4.4.1) (Gu et al. 2016). Compiled using Adobe Photoshop (ver. 25.12.3). Detailed disease scores are provided in Supplementary Table 3.

119 A two-dimensional non-metric multidimensional scaling (2D nMDS) analysis was
120 performed to assess whether virulence variability across the panel reflected any
121 underlying population structure. The disease severity score data were exported and
122 analysed in PRIMER 7 (ver. 7.0.24), and a 2D nMDS ordination plot was constructed
123 based on the Euclidean distance. A similarity profile then used to visualise the *Bgt*
124 clustering pattern that is indicative of pathotypes (M. J. Anderson 2008) (PRIMER 7
125 PERMANOVA+ add-on, PRIMER-E Ltd). 2D nMDS ordination identified six distinct
126 pathotype groups (Figure 2). Correlation vectors demonstrate the contribution of
127 specific host *R* genes to the observed variation (Figure 2A; Supplementary Figure 3).
128 Thus, the longer the vector, the greater its influence in differentiating isolates and their
129 orientation reflects the direction of increasing susceptibility to those isolates. cv Khapli,
130 which exhibited an IT of 0 against all isolates, is positioned near the centre of the
131 correlation vectors due to the lack of variation in its effectiveness. Wheat cvs Virest,
132 Thew, Normandie and Rockstar exhibited similar susceptibility patterns and were the
133 major contributors to pathotype IV formation. Brumby was susceptible only to isolate
134 2023-060, and it aligns with pathotype V, which carries the corresponding virulence.
135 Wheat cvs Khapli/8Cc and Armada displayed related *Bgt* susceptibility profiles, with
136 both showing pronounced susceptibility to QLD-derived isolates that predominantly
137 define pathotype I. Wheat cvs TD 1656, Timgalen and Coker 9803 exhibited similar
138 *Bgt* susceptibility profiles and contributed to the clustering of isolate 22PMWR26 (VIC),
139 together with the variety Brigand, which showed complete susceptibility exclusively to
140 this isolate. Out of these pathotypes, III and V, each carrying a single isolate, were
141 collected in 2023 from South Australia. However, III contains the second least virulent
142 isolate (2023-046), and the most virulent isolate (2023-060) is assigned to pathotype
143 V. To assess the geographical variation of these pathotypes, a 2D nMDS plot was

144 generated to determine if there is any correlation between state collection location and
145 pathotype. Clustering of isolates by location is difficult to conclude due to insufficient
146 isolates collected from VIC and among pathotype groups (Figure 2B). We then
147 examined for temporal variation (Figure 2C). This indicated pathotypes I and II were
148 comprised solely of isolates from 2024. Some isolates collected from 2024 are also
149 found in pathotype IV, which consists of isolates collected in 2020, 2021, 2022 and
150 2024 (Figure 2C). However, more isolates are needed to conclusively determine
151 whether there is a spatial-temporal effect on virulence.

152

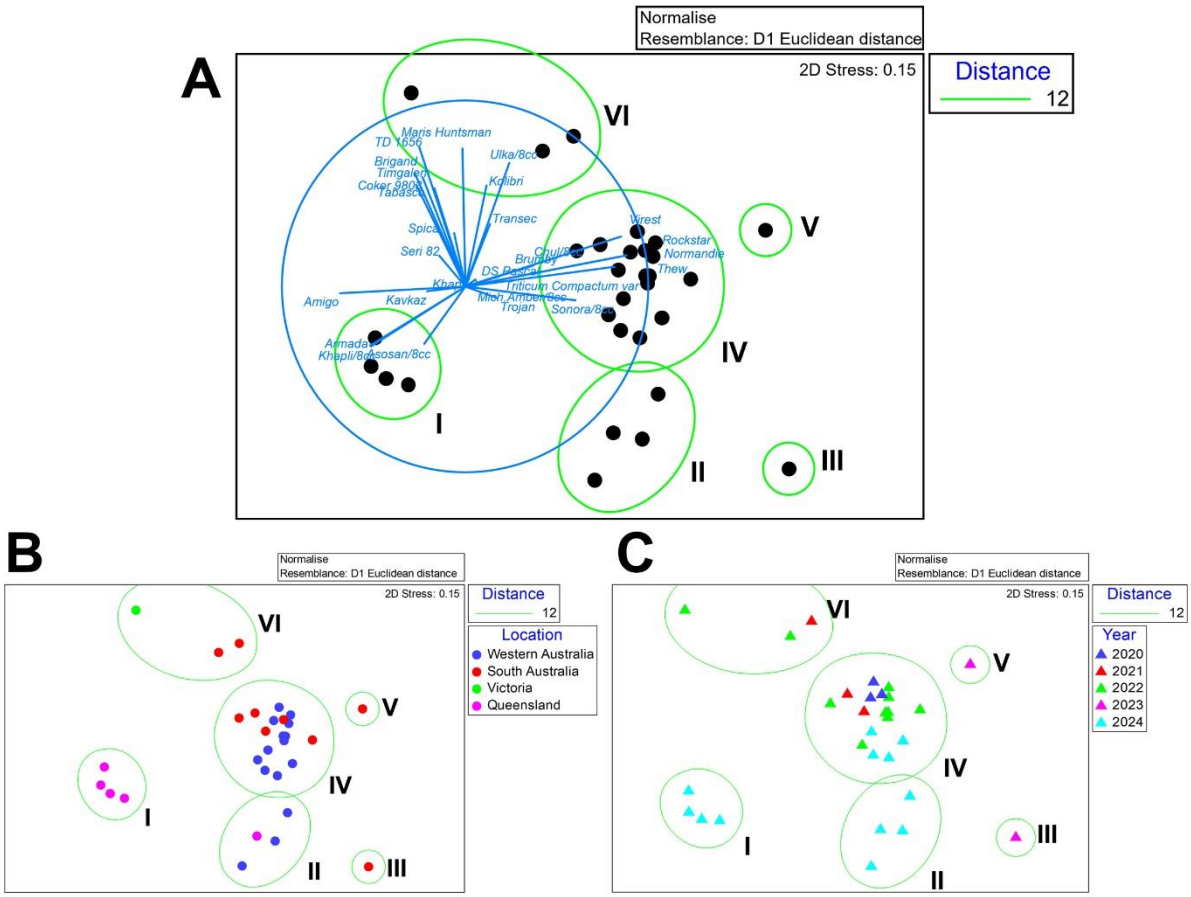


Figure 2: 2D nMDS scaling ordination plot reveals distinct Australian *Bgt* pathotypes. A Euclidean distance similarity matrix was calculated based on the pairwise virulence profile of 30 *Bgt* isolates and used to generate nMDS coordinates of each sample and was overlaid with the hierarchical clustering analysis of these isolates, denoted as green ellipses or as groups in roman numerals. **A**, Each isolate, denoted by black dots and is overlaid with the correlation vectors calculated based on the Pearson correlation, denoted as blue lines with wheat varieties. **B**, Isolate plotted based on collection location. **C**, Isolate plotted based on the year of collection.

164 Single major *R* genes are often rapidly overcome by new virulent pathogen races due
165 to strong selection pressure, whereas durable resistance remains effective over time
166 and across environments. Durable polygenic resistance provides complete to partial
167 protection against a wider range of pathotypes by lowering the selection pressure
168 (Burdon et al. 2014). In the United Kingdom, Alchemy, Cordiale, and Einstein
169 demonstrated long-lasting resistance against the local *Bgt* population over a 10-year
170 disease trial survey (Brown 2015). These varieties were screened to assess their
171 effectiveness against the Australian *Bgt* population by selecting representative isolates
172 from each pathotyping group and screened using seedling DLAs. Pathotype VI was
173 excluded due to loss of isolates. Wheat cvs Alchemy and Cordiale were resistant to all
174 pathotypes, whereas Einstein displayed moderate resistance across all pathotypes
175 (Table 1). Even though pathotype V contains the most virulent isolate, all three elite
176 European cultivars showed moderate to strong resistance. This suggests the potential
177 effectiveness of these varieties in providing long-term broad-spectrum WPM
178 resistance in Australia. However, further testing over several years across diverse
179 geographical and environmental conditions is required.

180 **Table 1 here**

181 Knowledge on Australian *Bgt* aggressiveness is limited, with only two existing
182 published data focused exclusively on WA isolates. For instance, Golzar et al.(2016)
183 pathotyped cumulative pools of WA *Bgt* isolates collected between 2011 and 2014,
184 whereas Kloppe et al. (2022) characterised 15 WA isolates collected in 2016. We then
185 compared these earlier findings with those from the contemporary Australian isolates
186 analysed in this study (Table 2). For statistical analysis, the disease scores from both
187 studies were converted to a 0–4 numerical scale and assigned infection types
188 following the method described by Li et al. (2019). Comparisons of WA isolates

189 revealed that *Pm3b*, *Pm3e*, *Pm1+2+9*, and *Pm2+6* exhibited a loss of effective
190 resistance over time. In contrast, *Pm4b* and *Pm17* demonstrated renewed
191 effectiveness against the WA *Bgt* isolates examined in this study. *Pm3a, Pm4a* and
192 the *Pm4a+unknown R* gene (from cvs. Asosan/8Cc, Khapli/8Cc and Khapli,
193 respectively) maintained their effectiveness over time in WA, with *Pm3a* and
194 *Pm4a+unknown* also remaining effective against *Bgt* isolates from other Australian
195 states. The effectiveness of these identified *Pm* genes require further experimental
196 validation to ensure that WPM resistance in varieties that carry them is not due to
197 unknown background resistance. However, Golzar et al.(2016) observed that Khapli
198 demonstrated adult plant WPM resistance.

199 **Table 2 here**

200 Since 2020, severe WPM outbreaks have occurred across the eastern states of
201 Australia, causing yield losses of up to 50% (Ellwood et al. 2024). Strategic use of
202 resistance genes is only effective when supported by detailed characterisation of *Bgt*
203 virulence and pathotype diversity (Czembor et al. 2014; El-Shamy and Mohamed
204 2022; Lalošević et al. 2021; Parks et al. 2008). Since the deployment of a major *R*
205 gene often leads to boom-and-bust disease dynamics driven by the emergence of new
206 pathogen races (Pink and Puddephat 1999). This underscores the need for continued
207 monitoring of pathogen diversity and resistance gene efficacy to guide region-specific
208 disease management (Zou et al. 2023).

209 This study represents the first reported simultaneous pathotyping of *Bgt* across a
210 broad coverage of major wheat-growing regions in Australia, resulting in the
211 identification of multiple virulences and six distinct pathotype groups. As many of the
212 most popular wheat varieties in Australia are susceptible to WPM, this suggests a

213 history of exposing *R*-genes in the field, many of which have lost their effectiveness.
214 Despite this, wheat cvs Tabasco and Khapli exhibit complete resistance to the tested
215 Australian *Bgt* population and represent valuable sources of resistance that can be
216 deployed by the cereal industry into existing parental varieties. In addition to major *R*
217 genes, the use of durable broad-spectrum resistance from Alchemy and Cordiale to
218 minimise the impact of WPM in Australia highlights the potential of tapping into
219 international germplasm as a resource for WPM resistance breeding in Australia.
220 Findings from this study support targeted deployment and stacking of effective *R*
221 genes and, ideally, implementing broad-spectrum WPM resistance. Materials
222 collected for the current research can be used to study WPM evolution, reveal their
223 origins, and identify their virulence factors. Furthermore, we hypothesise novel *Pm*
224 genes effective against Australian *Bgt* are present and remain undiscovered in the
225 differential wheat panel. Efforts are underway to locate these unknown genes and to
226 assess their efficacy in disease control.

227

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335 **Table 1:** Disease phenotypes of European durable WPM resistant wheat lines to
336 Australian *Bgt.* Each column contains representative isolates of different pathotypes,
337 and each row represents wheat lines. Wheat cv. Khapli (resistant) and Trojan
338 (susceptible) were used as controls and scored/rated based on 0-4 numerical scale
339 as described above (Li et al. 2019).

	Pathotype I	**Pathotype II**	**Pathotype III**	**Pathotype IV**	**Pathotype V**
	2024PM110172	**2024PM126**	**2023-046**	**20PMFRG01**	**2023-060**
Alchemy	R	R	R	R	R
Cordiale	R	R	R	R	R
Einstein	MR	MR	R	MR	MR
Khapli	R	R	R	R	R
Trojan	S	S	S	S	S

340

Table 2: Comparative analysis of previous pathotyping studies on Australian *Bgt* with this study. 'NA' indicates *Pm* genes not examined. Seedling infection scores from these studies were converted to a 0 to 4 numeric scale for statistical comparisons (Li et al. 2019). * Indicates that the wheat varieties used for the particular *Pm* genes differ among studies.

	(Golzar et al. 2016)	**(Kloppe et al. 2022)**	**This study**			
Region	**WA**	**WA**	**WA**	**SA**	**QLD**	**VIC**
R gene/sampling year	**2011-2014**	**2016**	**2020-2024**			
*Pm1a**	S	S	S	S	R	R
Pm2	NA	R	MR	MR	R	S
Pm3a	R	R	R	R	R	R
Pm3b	NA	MR	MS	MR	R	R
Pm3c	S	NA	S	MS	MS	MS
*Pm3e**	R	NA	MR	R	R	R
Pm4a	R	R	R	R	S	R
Pm4a+unknown	R	NA	R	R	R	R
*Pm4b**	S	R	R	R	S	R
Pm5a	S	NA	MS	MS	S	S
*Pm6**	S	S	MR	MS	MR	S
Pm7	MS	NA	MS	MR	MR	R
*Pm8**	S	MR	MR	MR	S	MS

Pm17	S	R	R	R	MS	S
Pm1+2+9	R	NA	S	MS	R	R
Pm2+6	R	NA	MS	MS	MR	S

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347