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

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

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

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

Thirty *Bgt* isolates were collected from major wheat-growing regions across Australia between 2020 and 2024 (Supplementary Table 1 and Supplementary Figure 1). 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 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 Tabasco carries an unknown *Pm* gene in addition to *Pm2*. Likewise, although 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.

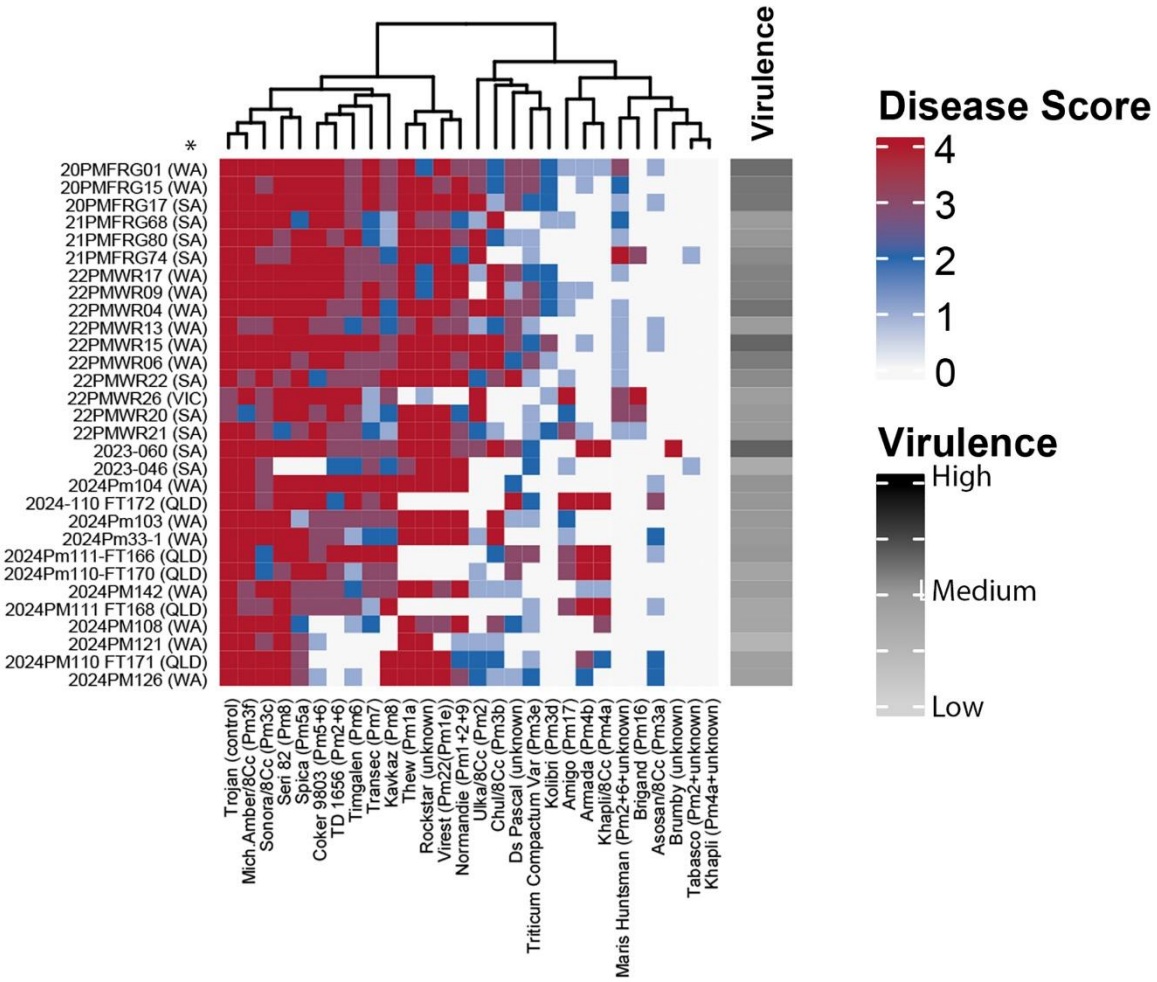

Figure 1: Virulence profiling of Australian *Bgt* isolates on differential and commercial wheat lines. The heatmap represents the virulence spectrum of *Bgt* isolates (*x*-axis) tested on a set of differential wheat lines (*y*-axis). The heatmap was generated using *ComplexHeatmap* package (ver.2.22.0) in R (ver.4.4.1) (Gu et al. 2016) and 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. 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 group IV formation. Brumby was susceptible only to
134 isolate 2023-060, and it aligns with pathotype V, which carries the corresponding
135 virulence. Wheat cvs Khapli/8Cc and Armada displayed related *Bgt* susceptibility
136 profiles, with both showing pronounced susceptibility to QLD-derived isolates that
137 predominantly define pathotype I. Wheat cvs TD 1656, Timgalen and Coker 9803
138 exhibited similar *Bgt* susceptibility profiles and contributed to the clustering of isolate
139 22PMWR26 (VIC), together with the variety Brigand, which showed complete
140 susceptibility exclusively to this isolate. Out of these pathotypes, III and V, each
141 carrying a single isolate, were collected in 2023 from South Australia. However, III
142 contains the second least virulent isolate (2023-046), and the most virulent isolate
143 (2023-060) is assigned to pathotype V. To assess the geographical variation of these

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

153

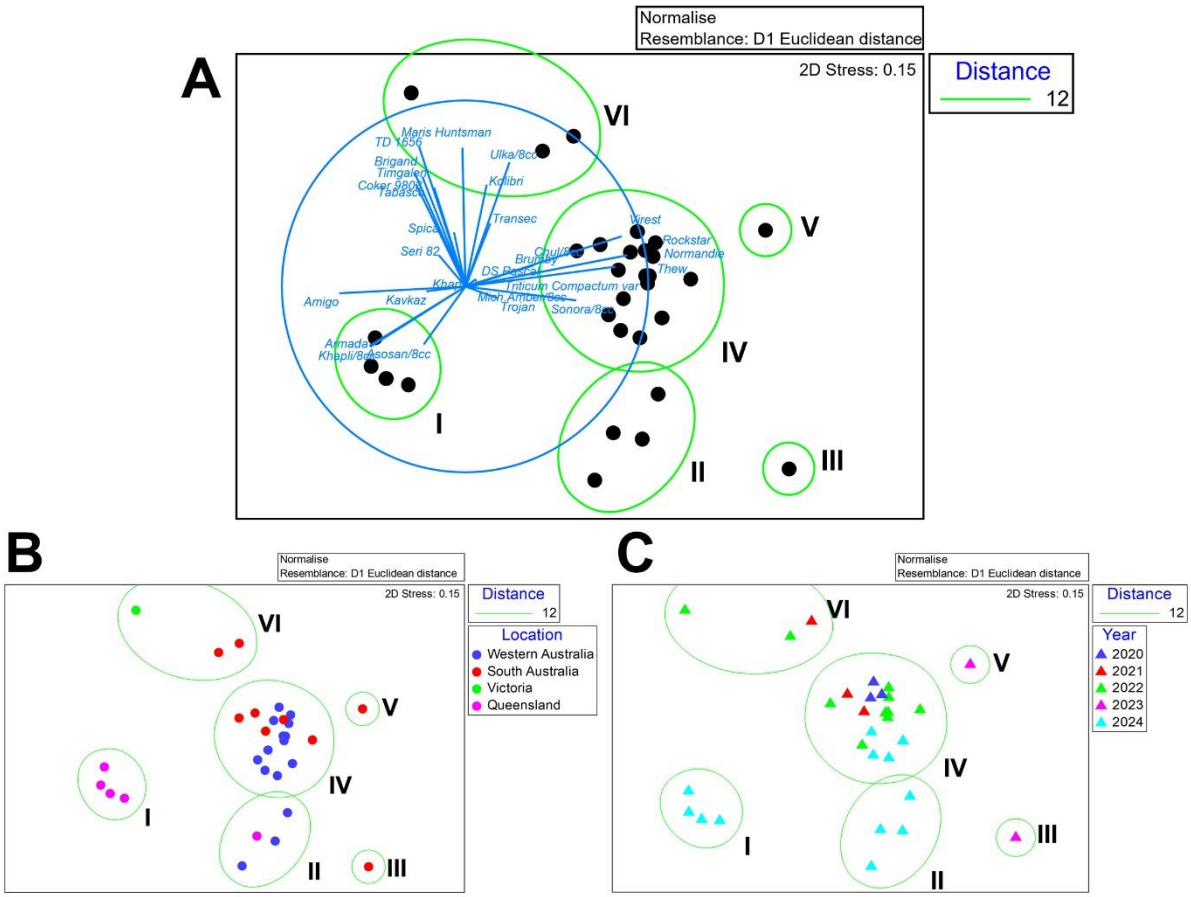

Figure 2: 2D nMDS scaling ordination plot reveals distinct Australian *Bgt* pathotypes. A Euclidean distance similarity matrix was calculated based on the pairwise virulence profile of 30 *Bgt* isolates and used to generate nMDS coordinates of each sample and was overlaid with the hierarchical clustering 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.

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

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

208 This study represents the first reported simultaneous pathotyping of *Bgt* across a
209 broad coverage of major wheat-growing regions in Australia, resulting in the
210 identification of multiple virulences and six distinct pathotype groups. As many of the
211 most popular wheat varieties in Australia are susceptible to WPM, this suggests a
212 history of exposing *R*-genes in the field, many of which have lost their effectiveness.
213 Despite this, wheat cvs Tabasco and Khapli exhibit complete resistance to the tested
214 Australian *Bgt* population and represent valuable sources of resistance that can be

deployed by the cereal industry into existing parental varieties. In addition to major *R* genes, the use of durable broad-spectrum resistance from Alchemy and Cordiale to minimise the impact of WPM in Australia highlights the potential of tapping into international germplasm as a resource for WPM resistance breeding in Australia. Findings from this study support targeted deployment and stacking of effective *R* genes and, ideally, implementing broad-spectrum WPM resistance. Materials collected for the current research can be used to study WPM evolution, reveal their origins, and identify their virulence factors. Furthermore, we hypothesise novel *Pm* genes effective against Australian *Bgt* are present and remain undiscovered in the differential wheat panel. Efforts are underway to locate these unknown genes and to assess their efficacy in disease control.

Acknowledgements

We thank the AGG (Horsham, Australia) for the provision of wheat germplasm used in this study. Sam Trengove (Trengove Consulting), Hossein Golzar (DPIRD), Andrea Hills (DPIRD) and Ciara Beard (DPIRD) for the provision of *Bgt* samples. Julie Lawrence (Curtin University) for valuable technical assistance. Kul Adhikari (Curtin University), Steven Chang (Curtin University), Marta Lopez-Ruiz (Curtin University), and Nirmala Sharma (Curtin University) for isolate maintenance.

Funding: Support was provided by Intergrain Pty Ltd (Grant CTR18532) and the Centre for Crop and Disease Management, a co-investment between the Grains Research and Development Corporation and Curtin University (Grant CUR1403-002BLX).

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

339

340 **Table 2:** Comparative analysis of previous pathotyping studies on Australian *Bgt* with
341 this study. 'NA' indicates *Pm* genes not examined. Seedling infection scores from these
342 studies were converted to a 0 to 4 numeric scale for statistical comparisons (Li et al.
343 2019). * Indicates that the wheat varieties used for the particular *Pm* genes differ
344 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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346