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REVIEW ARTICLE

Barley powdery mildew control in Western Australia and beyond

Simon R. Ellwood | **Francisco J. Lopez-Ruiz** | **Kar-Chun Tan**

Centre for Crop and Disease Management, School of Molecular and Life Sciences, Curtin University, Bentley, Western Australia, Australia

Correspondence
Simon R. Ellwood, Centre for Crop and Disease Management, School of Molecular and Life Sciences, Curtin University, Bentley, WA 6102, Australia.
Email: srellwood@gmail.com

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Abstract

Australia is one of the largest barley exporters in the world, with Western Australia accounting for some 40% of national production. The crop is predominantly grown in the south and south-west of the state in winter and spring, where temperate conditions and higher rainfall levels are more suited to barley than northern and eastern regions. Between 2007 and 2013, prolonged outbreaks of barley powdery mildew (BPM) occurred. This was brought about by a combination of the extensive use of susceptible cultivars and an over-reliance on a small number of single mode-of-action demethylation inhibitor fungicides, which select for mutations in the C14α-demethylase (*Cyp51A*) gene. This review highlights the steps taken to reduce losses to BPM, breeding efforts to introduce resistance into cultivars and the success of pre-breeding research to find new and durable resistance genes. We also draw comparisons with powdery mildew in Australian wheat, where similar factors are leading to substantial outbreaks.

KEYWORDS
Blumeria graminis s. s., *Blumeria hordei*, broad-spectrum resistance, fungicide resistance

1 | POWDERY MILDEWS, MOBILE AND ADAPTABLE ADVERSARIES

Barley powdery mildew (BPM) is caused by the ascomycete fungus *Blumeria hordei* (Bh), formerly known as *Blumeria graminis* f. sp. *hordei* (Liu et al., 2021). The pathogen, in common with all mildews, is an exclusively obligate biotroph, causing stunting and reduced numbers of tillers, particularly following early infection in susceptible cultivars in the absence of chemical control. Growers are impacted by both yield reductions and downgrades in grain quality from malting to animal feed. In south-western Western Australia (WA) the disease is endemic, with regional variations in infection severity that correlate with higher rainfall. Disease severity is generally low, although grain yield losses can range from 12% to 60%, with the potential for substantial losses in some areas and seasons (Chan et al., 1990). As an obligate biotroph, Bh has dispensed of many genes essential to primary and secondary metabolism (Spanu et al., 2010). Instead, like all powdery mildews sequenced to date, Bh has an elaborate host relationship, secreting numerous effector proteins with roles in establishing infection and manipulating host immunity at several levels by interfering in reactive oxygen species homeostasis, key defence responses and cell death (Bourras et al., 2018; Li et al., 2021; Pennington et al., 2016; Yuan et al., 2021). The pathogen also has a large genome expanded by repetitive DNA caused by retrotransposon activity, a feature that facilitates adaption to different host genetic backgrounds by rapid transposon-mediated expansion and turnover of effectors (Menardo et al., 2017; Pedersen et al., 2012).

The pathogen may survive hot arid Australian summers in fruiting structures known as chasmothecia containing ascospores (Figure 1), as found in Israel which shares similar conditions (Koltin

FIGURE 1 Field symptoms of barley powdery mildew on a highly susceptible line with withering and collapse of lower leaves in the background. Inset: Older colonies may develop groups of small black fruiting bodies called chasmothecia, indicated with an arrow.

& Kenneth, 1970) and as opposed to over-wintering in the vegetative stage in the northern hemisphere (Cherewick, 1944). However, Bh may also persist in mixed pastures that receive summer rainfall such as in coastal zones, or on volunteer plants near water sources. Infection is promoted by cool humid conditions (Ward & Manners, 1974), which stimulates the germination of asexual reproductive spores known as conidia. A mixed reproductive system, very large effective population size and vast numbers of vegetative polycyclic conidia with associated mutation risks mark powdery mildew out as having a high adaptive potential (McDonald & Linde, 2002). This is evidenced by almost all European virulence genes having recombined into just two isolates (Spies et al., 2012). Bh is highly mobile as the airborne conidia are able to travel hundreds of kilometres under favourable conditions and, with repeated dispersals, cover ever larger areas allowing rapid establishment of virulent new pathotypes (Wolfe et al., 1992). Evidence for migration of Bh across large bodies of water between continents is lacking as the conidia are fragile and short-lived compared to most fungi. However, Bh probably has a similar epidemiology to the closely

related wheat powdery mildew (WPM, caused by *B. graminis* f. sp. *tritici*), where genomic analyses have established historical dispersals associated with human trade and migration (Sotiropoulos et al., 2022), together with evidence for hybridization between colonizing and distantly related local lineages. In a study based on genome-wide synonymous single-nucleotide polymorphisms (SNPs) (Lu et al., 2016), WA Bh isolates were enigmatically distinct from European, Japanese and American isolates. This may be due to hybridization with a resident Bh relative, adaption of such a mildew to domestic barley or migration from an unsampled population.

2 | LOSS OF BARLEY POWDERY MILDEW CONTROL IN WESTERN AUSTRALIA

Reports of powdery mildew outbreaks in south-western WA started to become common in 2007, with epidemic levels experienced in 2010 and 2011 (Oliver & Jayasena, 2012). At the time, predominantly susceptible cultivars were grown (Figure 2) and the role played by a loss of control by fungicides was initially unclear as no systematic mutation studies had been carried out, although reports of declining fungicide efficiency appear to go back to 2005. This changed in 2009, when Tucker et al. (2020) initiated the first study that revealed a sudden increase in the *CYP51* non-synonymous amino acid mutation S524T from 0% in 2009 to 90% in 2011. *CYP51* encodes C14α-demethylase (Aoyama et al., 1996; Yoshida, 1993), which is the target site enzyme for demethylation inhibitor (DMI) fungicides and in powdery mildews is essential for biosynthesis of the plasma membrane component ergosta-5,24(24^1)-dien-3β-ol (Debieu et al., 1995; Loeffler et al., 1992). DMI fungicides are also known as FRAC mode of action Group 3 (https://www.frac.info/knowledge-database/knowledge-database), which is mainly composed of the chemical family triazoles. Two new *CYP51* haplotypes identified in WA were associated with strong resistance to the DMI fungicide tebuconazole. One contained mutation S524T, in combination with a pre-existing Y137F mutation found in all Australian isolates, while

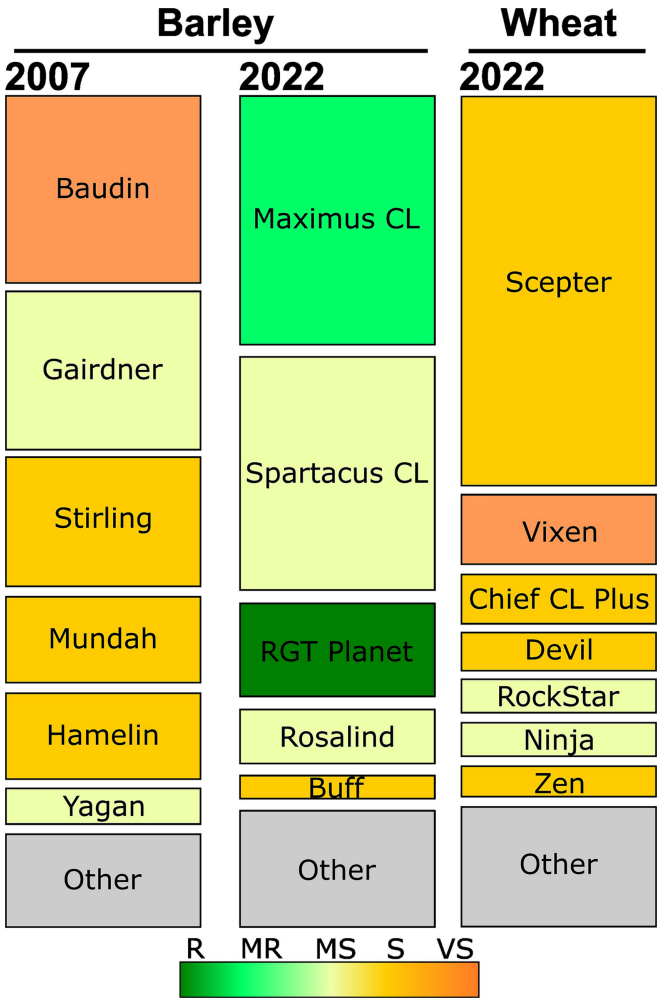

FIGURE 2 Disease phenotypes of major barley and wheat cultivars grown in Western Australia. Over 80% of the barley area was sown to susceptible cultivars in 2007 and a similar proportion to susceptible wheat cultivars in 2022. Boxes are proportional to the area grown, with 'other' composed of new or less popular cultivars. Colour scale: R, resistant; MR, resistant to moderately resistant; MS, moderately susceptible; S, susceptible; VS, very susceptible. Data obtained from Tucker et al. (2015), Paynter and Khan (2023) and Shackley and Nicol (2023).

the second carried additional mutations M304I and R330G. These T524-containing haplotypes had a tebuconazole resistance factor (RF) of 17.6 compared to the S524 wild-type allele. Individual expression of K172E, Y137F and M304I in *Saccharomyces cerevisiae* indicated RFs of approximately 1, while S524T had a RF of 3.7. However, heterologous expression confirmed a synergistic effect of the combined Y137F and S524T mutations with a RF of 33, while the solo Y137F construct had a RF of 9.7 against fluquinconazole (Tucker et al., 2020). The spread of the T524-containing haplotypes across the barley-growing regions accounted for the loss of fungicide field efficacy, as fungal disease control at the time was overwhelmingly achieved by older and cheaper DMI formulations containing primarily straight tebuconazole (Tucker et al., 2015).

The rapid loss of control by single DMI actives is characteristic of single gene mutations that confer resistance and, similar to other pathogens, in WA Bh isolates this appears to have followed on from the Y137F mutation. Fungal pathogens that produce large numbers of spores such as Bh have a greater probability of mutations leading to fungicide resistance and, inevitably, pathogens reproduce at higher rates on susceptible cultivars compared to those with some level of resistance. Susceptible cultivars also equate to higher selection pressure as more fungicide applications at higher rates are needed for effective disease control. This causes a rapid swing towards resistance against single actives, leading to a population that is predominantly resistant in a relatively short time frame.

Various estimates of the costs of powdery mildew damage and the value of control have been proposed. However, these are inevitably generalized and exclude costs to individual growers, especially those in areas prone to more severe outbreaks and where additional fungicide treatments may be needed or where decisions are taken to reduce the area sown. Although the cost of older DMI products such as tebuconazole and propiconazole was relatively cheap at the time of the epidemic, the use of alternative chemistries due to resistance was comparatively expensive, with more recent prices of $17–27 per hectare (Bullen, 2020) combined with variable application costs dependent on the mode of delivery. Nonetheless, there is no doubt the WA BPM epidemic was costly, with annual losses estimated by Tucker et al. (2015) at AU$100 m.

3 | CHANGES IN BREEDER AND GROWER PRACTICES

In response to the compromised DMI products, 2013 saw the first registration in Australia of a non-DMI fungicide product, spiroxamine from the FRAC Group 5 carboxamide chemical family, followed by a Group 7 succinate dehydrogenase inhibitor (SDHI) containing fluxapyroxad in 2015. The registration of a SDHI may have been influenced by a need to control other diseases, particularly barley net blotches, as they are not particularly effective against powdery mildew. Other fungicides available to Australian growers include Group 11 quinone-outside inhibitors (QoIs) azoxystrobin and pyraclostrobin and the powdery mildew-specific Group 13 quinoline quinoxyfen. QoIs have remained viable against BPM in Australia, although these are known to have lost effectiveness overseas and against WPM in the eastern states of Australia (Dodhia et al., 2021), while Groups 5 and 13 are regarded as low to medium risk of breakdown by FRAC, offering longer-term control prospects.

To lower selection pressure and the risk of resistance emergence, specific fungicide management guidelines have been developed as part of the Australian Fungicide Resistance Extension Network (AFREN) initiative (https://afren.com.au/resources/#management-guide). Growers are recommended to mix and rotate fungicides and mode of action groups using established resistance management principles to reduce pressure on any one individual fungicide or mode of action. Even though strobilurin fungicides are present in most barley chemical programmes and BPM is often exposed to off-target applications of these chemicals, resistance to this high-risk mode of action group has not been reported yet in Australia, with the delay attributed to adoption of AFREN guidelines by the industry.

Arguably, growing less susceptible barley varieties is the most effective management approach to reduce the reliance on fungicides to control BPM, but growers are advised of significant non-chemical integrated disease management options through industry newsletters and AFREN (https://afren.com.au/). These include inoculum control through the removal of volunteer barley plants during fallow periods and the reduction of infected stubble, which limits the volume of spores spreading into adjacent or subsequent barley crops; rotation of both different crops and more tolerant barley varieties carrying different resistance genes, which reduces selection for virulence and inoculum carryover from year to year; avoiding warm and damp conditions that favour BPM disease during early winter by practising delayed sowing, thus limiting infection of young plants, although this strategy can result in reduced yield potential in some environments; and finally, high nitrogen levels and limited air circulation are associated with increased BPM incidence and as such, adjusting nitrogen levels and lowering relative humidity by adopting wider row spacing, lowering seeding rates and grazing all contribute to reducing disease levels.

The mildew outbreaks also led to greater interest in powdery mildew resistance in new cultivar releases, which have the added benefit of reducing selection pressure for fungicide resistance because fewer applications are needed for effective disease control. As many barley *R*-genes have yet to be isolated, their presence in Australian cultivars was postulated by Dreiseitl and Platz (2012) on the basis of disease phenotypes using pathogen isolates with known virulences. These mostly contained *MlLa*, a major dominant resistance (*R*) gene that, often in combination with a second *R* gene, had proved robust in Australia up to that point. The most popular of these were Buloke, reaching over 25% of the area sown by 2012, followed by Hindmarsh at over 35% by 2014, then Scope and La Trobe later in the decade. As typically follows when strong selection pressure is exerted by major *R*-genes grown over sufficiently large areas, *MlLa* has since been defeated by the pathogen, first in the eastern states by 2015 and later confirmed in WA by 2021 (Paynter, 2021). *MlLa* is still used in the current cultivars, notably Spartacus CL and Rosalind (Figure 2), but their field ratings

have reduced from moderately resistant to moderately susceptible. Other examples of major *R*-genes failing during this period and confirmed by independent testing in our laboratory included *MlSt* in Oxford and *MlCh* and *Mlra* in Yagan (authors' unpublished data).

The most significant change of attitude in achieving stable mildew resistance in Australia was the gradual acceptance of the *mlo* gene. *mlo* provides broad-spectrum resistance to all Bh pathotypes and has been used in European agriculture for some 40 years (Büschges et al., 1997; Jørgensen, 1992; Kusch & Panstruga, 2017). The phenotype results from inactivation, or suppression of expression, of the wild-type *Mlo* gene that negatively regulates plant defence responses (Acevedo-Garcia et al., 2014; Büschges et al., 1997). The prevailing view up to this point had been that the gene was unsuited to the Australian environment, causing excessive yield losses. Indeed, *mlo* is known to have several pleiotropic effects including spontaneous necrosis leading to a reduction in photosynthetic capacity and reduced fertility (Behn et al., 2005; Kjær et al., 1990; Thomas et al., 1998). However, breeders in Europe had largely compensated for this by a process known as top-crossing to incorporate 'helper' genes (Kjær et al., 1990). The identity of these is unknown. However, Li et al. (2022) recently discovered activation of a conserved tonoplast monosaccharide transporter (*TMT3*) in wheat alleviates *mlo* growth and yield penalties. The gene is yet to be extensively tested in the field and may still enable undesirable *mlo* pleiotropic effects but represents a promising alternative to traditional breeding strategies in diverse crops through gene editing.

Ian Edwards of Edstar Genetics pioneered the first Australian introductions of *mlo* in the early 2010s with the European varieties Granger, Henley and Westminster. Field trials showed no yield penalties across different environments and, although these cultivars were superseded, cultivars such as RGT Planet, which was grown in over 20% of the WA barley area in 2020 (Paynter & Khan, 2023), have shown how successfully the gene can be deployed. RGT Planet has since declined in popularity, down to 12% in 2022 and with reports suggesting very little will be grown in 2024. This is mainly due to severe outbreaks of net form net blotch (NFNB), caused by *Pyrenophora teres* f. *teres*, a pathogen which, like Bh, has a high adaptive potential (Ellwood et al., 2019; Mair et al., 2016; Syme et al., 2018). RGT Planet NFNB resistance reduced from an intermediate moderately resistant–moderately susceptible rating (Paynter et al., 2017) to very susceptible (Paynter & Khan, 2023), while at same time the pathogen has gained resistance to both DMIs and SDHIs.

4 | PRE-BREEDING RESEARCH FOR DURABLE RESISTANCE

In response to the severe mildew outbreaks in WA, Australia's national agricultural research, development and extension platform, the Grains Research and Development Corporation, funded a project at Curtin University to find new resistance genes. From the outset, the project sought to avoid major *R*-genes by excluding lines where strong levels of resistance were active in seedling leaves, a characteristic feature of the class. This was based on the hypothesis that partial, slow mildewing or adult plant resistance (APR) may be considered non-race-specific and potentially durable (Hwang & Heitefuss, 1982). APR is generally most effective from the fifth-leaf stage (Heitefuss et al., 1997) but its underlying genetic basis was poorly understood. To discover new genes, hundreds of landraces and wild barley relatives were screened from across original centres of barley diversity: Ethiopia, countries bordering the Fertile Crescent, the Caucasus and eastwards to the Himalayas.

We discovered several lines with phenotypes matching those described by Heitefuss et al. (1997). Detailed cytological experiments established that the genes triggered unique host responses and, with one exception, were found in new chromosome locations. Despite exhibiting partial resistance phenotypes, particularly after the fifth-leaf stage, all were single major effect genes, rather than genes within minor quantitative trait loci (QTLs). Furthermore, they provided APR resistance to all Bh pathotypes tested both at Curtin University and overseas. All the genes allow small sporadic mildew pustules to form after the fifth-leaf stage, which is characteristic of APR, although there were minor differences in their onset and appearance. However, the plants were effectively immune. The developmental aspect of the genes was most obvious in plants grown over summer in a glasshouse, where 16-h days and warm overnight temperatures promotes heading at or before the fifth leaf and where pustules are apparent over the whole plant (authors' unpublished data). Normally, in winter/spring field conditions, fifth-leaf plants would be small with initial tillers emerging (Zadoks stage 15). Conceivably, by allowing some mildew development at earlier growth stages, such genes may place less selection pressure on the pathogen.

Conspicuous among the APR genes discovered was *rbgh2* on chromosome 7H from an Azerbaijani landrace (Ge et al., 2021; Moolhuijzen et al., 2023). *rbgh2* is one of the few recessive resistance genes against BPM to be found that notably includes *mlo*, a potentially durable gene *mlmr* on 6H from Morocco (Piechota et al., 2020) and the race-specific *mlt* (Schönfeld et al., 1996). *rbgh2* has features consistent with penetration resistance, with cell wall appositions (CWAs) localized to attempted entry sites, which represents a form of basal penetration resistance (Aghnoum et al., 2010). Only infrequent epidermal cell death (the hypersensitive response), which is normally associated with major *R*-genes, was observed. In addition, and unlike *mlo* alleles, there is a lack of spontaneous necrosis and mesophyll cell death (Behn et al., 2005; Ge et al., 2016). However, in common with *mlo*, *rbgh2* resistance is associated with distinctive large halos after staining with 3,3′-diaminobenzidine (indicative of defence-related hydrogen peroxide generation) around appressorial penetration sites and defence-related vesicle-like bodies diffused throughout but aggregated at the penetration sites (Collins et al., 2003; Ge et al., 2021).

Rather intriguingly, *rbgh2* may act through suppression of genes involved in cAMP/G-protein-coupled signalling and induction of calmodulin-binding genes (Moolhuijzen et al., 2023). *rbgh2* influences gene expression both genome-wide by inducing resistance gene homologues and through genes associated with innate immunity together with co-expression of genes at the *rbgh2* locus. Conspicuous among the genes associated with innate immunity was suppression of a WD40 repeat-containing protein. A wheat orthologue, *TaHOS15*, increased susceptibility to *B. graminis* sensu stricto (Bg) via a transcriptional repressor complex acting on the chromatin of defence-related genes (Liu et al., 2019). Also suppressed was a ZIM-domain (JAZ) jasmonate repressor, thought to be associated with host and non-host production of reactive oxygen species in the chloroplast and cell death (Ishiga et al., 2013), while activated genes included members of the tryptophan biosynthetic pathway, cytochrome P450 monooxygenases and the pathogenesis-related genes *PR5* and *PR7*. Two other genes with superficially similar characteristics were reported by Ge et al. (2021): *RBgh1*, from a Turkish landrace and *RBgh3*, from the same line as *rbgh2*. Both have similar cytological features to *rbgh2*, with minor differences in vesicle sizes and distribution observed.

The research also uncovered the novel *mlo* variant, *mlo-11* (*cnv2*) (Ge et al., 2016). As the name suggests, *mlo-11* (*cnv2*) is a *mlo-11* repeat subunit copy number variant with only two copies rather than some 11–12 originally used in breeding programmes (Ge et al., 2016). The repeats consist of a section of the *Mlo* promoter region and the first five exons of the gene that appear to be the result of rolling-circle DNA replication used by some plant viruses and transposons. Rather than suppression acting via transcriptional read-through interference as indicated by Piffanelli et al. (2004), we found *Mlo* expression was conditioned by copy number-dependent DNA methylation. Further experiments confirmed gene silencing by quantifiable differences in small interfering RNA counts and histone modification levels between copy number variants (Ge et al., 2020). Thus, the expression of *mlo-11* (*cnv2*) is a stably-inherited phenomenon that may provide advantages over the original *mlo-11* and other alleles, as these show increased susceptibility to physiological spotting (Behn et al., 2005; Makepeace et al., 2007), facultative diseases such as spot blotch (Kumar et al., 2001) and reduced mycorrhization (Jacott et al., 2020). These traits were not assessed by the authors for *mlo-11* (*cnv2*), either in a standard or *mlo*-adapted background, as they require field trials in different genetic backgrounds across a range of field sites.

mlo-11 (*cnv2*) resistance was developmentally controlled and quantitative as with the APR genes described above, consistent with the observations of Wolter et al. (1993), who found the formation of CWAs of different *mlo* alleles was under developmental control up to 21 days. More obvious mildew colonies in *mlo-11* (*cnv2*) seedling leaves than other alleles studied by Ge et al. (2016) indicated attenuation by the milder variant and, in addition, there was a lack of spontaneous CWAs or the same degree of necrosis of mesophyll cells and loss of photosynthetic tissue found in conventional *mlo* alleles.

5 | RECENT OUTBREAKS OF WHEAT POWDERY MILDEW IN AUSTRALIA, A CASE OF HISTORY REPEATING ITSELF?

Reports of WPM outbreaks have been circulating since 2020 in New South Wales and northern Victoria (Simpfendorfer et al., 2023) and in 2023, particularly in eastern South Australia, there have been anecdotal descriptions of substantial losses of around 50%, with some growers considering reducing next year's plantings. These coincide with the detection of reduced sensitivity and resistance to DMIs and QoIs (Dodhia et al., 2021; Lopez-Ruiz et al., 2023). In WA, although there are no reports of fungicide resistance to date, sporadic outbreaks going back several years have occurred both in high rainfall zones (Lopez-Ruiz et al., 2022) and also in drier wheatbelt regions. A grower in a low rainfall area in WA described that even in hot conditions with low humidity and low plant biomass (dense crop canopies encourage mildew) WPM was becoming more prevalent (https://www.youtube.com/watch?v=pECd0YgT5hg). This may be the pathogen adapting to become hypervirulent on an already susceptible cultivar such that the disease is evident even in less conducive conditions. As with BPM at the time of the WA epidemic, the majority of the area sown to wheat consists of susceptible cultivars (Figure 2). With fungicide resistance already present in the eastern states, the outlook for wheat is clear and a need for improved powdery mildew resistance levels is perhaps even greater than in barley, as the vast areas under cultivation means there are difficulties of getting a spray across a susceptible crop in a timely manner where there is a high disease loading in the environment, even where fungicide resistance is not an issue (Daniel Mullan, InterGrain, Bibra Lake, WA, Australia, personal communication).

Preliminary pathotyping studies using contemporary Bg isolates collected from across Australian wheat-growing regions have revealed modest pathotype diversity and evidence that many *R*-genes currently offer highly effective resistance (authors' unpublished data). Moreover, a large repertoire of genes is available to breeders to protect the local industry from the impending threat of fungicide-resistant Bg populations, with over 100 known race-specific *R*-genes (Wang et al., 2023). These may be stacked for improved longevity but there are good existing sources of resistance in high-yielding cultivars, particularly in Europe, for breeders to incorporate (AHDB, 2024). Potentially long-lasting genes also exist such as the interspecific *Pm40*, an apparent successor to *Pm21*, which has been successfully used in China for over 40 years (Tang et al., 2018). Furthermore, although no naturally occurring *mlo* mutations have been found in wheat, the inactivation of all three *Mlo* homoeoalleles using mutational and gene-editing approaches to produce broad-spectrum resistance has been carried out (Acevedo-Garcia et al., 2017; Wang et al., 2014). Acevedo-Garcia et al. (2017) found no evidence of pleiotropic effects observed in their allelic variants, which may now also be ameliorated by *TMT3* (Li et al., 2022), the conserved wheat *TMT3* described above. Perhaps the main drawback to wheat *mlo* resistance is enhanced allele-dependent susceptibility to wheat blast (Gruner et al., 2020), which has yet to reach Australia.

6 | PROSPECTS FOR BARLEY POWDERY MILDEW RESISTANCE AND NEW BREEDING APPROACHES

The examples above illustrate the strong need for minimum powdery mildew resistance levels in Australian wheat and barley. Even modest levels of resistance are worth incorporating as this slows disease progression and reduces overall conidia numbers where conditions are not particularly conducive to disease, as well as decreasing the pressure on currently effective fungicides. Both at the Curtin University and overseas, diverse new sources of BPM resistance have been discovered (e.g., Ames et al., 2015; Czembor & Czembor, 2021; Dreiseitl, 2017a, 2017b; Romero et al., 2018). To achieve durable resistance, a range of breeding strategies are available dependent upon introgression timeframes, technology platforms and cost. The genes discovered by Ge et al. (2016, 2021) may in themselves provide longer-term resistance but combinations of such genes with different underlying mechanisms is perhaps the optimal approach that the pathogen is unlikely to overcome, especially where the genes have not been individually exposed in cultivars. Gene editing or mutagenesis of defence response repressors and targets of secreted Bh effectors offer the prospect of expanding the diversity of available mechanisms, although there is the possibility of unforeseen pleiotropic effects. A more sophisticated approach is to combine nonspecific minor partial resistance genes with additive effects, which has been highly successful in providing long-term resistance in UK winter wheat (Brown, 2015). Indeed, such genes may now be readily identified through large-scale germplasm sequencing and genome-wide association mapping to identify co-inherited haplotype blocks based on linkage disequilibrium (Bhat et al., 2021). From the breeding perspective, backcrossing to remove undesirable genetic backgrounds and precision introgression is now possible by haplotype-assisted genomic selection (Brinton et al., 2020), complementing selection for polygenic traits such as yield and malting quality.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no datasets were generated or analysed.

ORCID

Simon R. Ellwood https://orcid.org/0000-0002-2673-0816

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