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Allocation of the oat powdery mildew resistance gene *Pm3* to oat chromosome 1A

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Abstract
Besides the mode of inheritance, the knowledge of the chromosome location and allelic relationships are the essentials towards a successful deployment and stacking of divergent disease resistance genes for a given pathogen in breeding programs. Powdery mildew of oats, to which 11 major resistance genes in the host *Avena sativa* L. have been characterized so far, is a prevalent fungal disease of the crop in Northwestern Europe. In the present study, the resistance gene *Pm3* was mapped by linkage analysis relative to molecular markers from oat consensus linkage group Mrg18 which was recently determined to represent oat chromosome 1A. *Pm3* was located at 67.7–72.6 cM on Mrg18 of the oat consensus map, a position at which also stem and crown rust resistance genes *Pg13* and *Pc91* and a large cluster of resistance gene analogs have been previously mapped. The closely linked marker GMI_ES03_c2277_336 was found to be useful for the prediction of *Pm3* in gene postulation studies. Although the major effect of the widespread gene got lost over time, the known genome location with associated markers will assist revealing in future genetic studies whether there is a possible residual effect of the gene contributing to adult plant resistance.

Keywords *Avena sativa* · *Blumeria graminis* · Disease resistance · Oat consensus map · 7C-17A translocation

Introduction

Cultivated oat (*Avena sativa* L.) to which approved health claims have been granted (Storsley et al. 2014) is, though of its high value for human nutrition, low in acreage compared to other cereals like rice, wheat, and maize. The crop is an allohexaploid species ($2n = 6x = 42$, AACCDD subgenomes) with a large genome (12.5 Gbp; Yan et al. 2016) and made up of genotypes that contain variable chromosome rearrangements (Singh and Kolb 1991; Chaffin et al. 2016).

Breeding for disease resistance needs to be addressed in every crop breeding program as pathogen attacks can significantly reduce crop yields and grain quality in susceptible cultivars. Powdery mildew of oats, caused by the biotrophic fungus *Blumeria graminis*, is a major disease in the humid temperate climates of Northwestern Europe (Schwarzbach and Smith 1988; Roderick et al. 2000). Previous studies have shown that losses in grain yield caused by the disease could be attributed to reductions in numbers of fertile panicles and thousand grain weights, while quality parameters such as percentage protein contents and specific weights were negatively correlated with levels of powdery mildew (Roderick and Jones 1988).

Eleven major genes for resistance to powdery mildew in cultivated oats have been catalogued so far (*Pm1-Pm11*; Hsam et al. 2014; Herrmann and Mohler 2018; Ociepa et al. 2020), but many more sources, as yet uncharacterized, for resistance to powdery mildew do exist in oat landraces and wild relatives of different ploidy levels (Herrmann and Roderick 1996; Okoń et al. 2014, 2016, 2018; Okoń and Kowalczyk 2020). Although the access to genes from lower ploidy levels for enhancing cultivated oat germplasm is challenging (e.g., Aung et al. 1977, 2010; Thomas et al. 1980), agriculture will finally benefit from this work.

The advent of a robust genotyping-by-sequencing approach (Elshire et al. 2011; Huang et al. 2014), i.e., an all-in-one approach combining single nucleotide polymorphism (SNP) discovery and SNP scoring and analyzing

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pooled samples in a highly multiplexed fashion, allowed establishing the first true road map of the complex oat genome, with 9 of 21 consensus linkage groups unequivocally assigned to physical chromosomes (Chaffin et al. 2016). This map was then substantially extended in the work of Bekele et al. (2018). Recently, a publicly available hexaploid oat genome sequence of oat variety OT3098 was released (https://oatnews.org/).

Of the 11 documented powdery mildew resistance genes, *Pm9*, *Pm10*, and *Pm11* were allocated to the oat consensus map by GBS marker sequence information (Herrmann and Mohler 2018; Ociepa et al. 2020). It is expected that further consensus map-based genetic mapping studies, e.g., crown rust resistance gene *Pc39* was allocated to Mrg11 (Sowa and Paczos-Gręda 2020; Zhao et al. 2020a), *Pc53* to Mrg08 (Admassu-Yimer et al. 2018), and *Pc98* to Mrg20 (Zhao et al. 2020b), will facilitate determining how resistance genes are distributed across the oat genome. Furthermore, the integrative analysis of historical data from both cytogenetic and genetic mapping studies can further increase this knowledge.

The widespread, dominant resistance gene *Pm3*, derived from the wild oat *A. sterilis* L. var. *ludoviciana*, was assigned to chromosome 17A by monosomic analysis (Hsam and Zeller 1998; Hsam et al. 2014). The gene was mapped relative to restriction fragment length polymorphism (RFLP) markers cMWG706 and cMWG733 from homoeologous group-1 chromosomes of the *Triticeae* (Mohler et al. 2012). In the present study, we used these historical data for the targeted re-mapping of *Pm3*. Furthermore, we explored the prediction potential of SNP markers with linkage to *Pm3*.

Materials and methods

Plant materials and DNA isolation

The *Pm3* mapping population Kanota × Rollo comprised 79 $F_{2:3}$ lines and was reported in Mohler et al. (2012). A set of 104 oat cultivars/lines (Table S1) was used to assess the genotype frequency and the predictive ability of SNP markers linked to *Pm3*. For the diversity panel, genomic DNA was extracted from lyophilized primary wheat leaves as described by Plaschke et al. (1995). For 53 oats of the collection, the *Pm3* and other *Pm* phenotypes were known from previous studies (Hsam et al. 1997, 1998, 2014; Yu and Herrmann 2006; Herrmann and Mohler 2018). The pedigrees of the oat lines carrying *Pm3* (Table S2) were accessed from the POOL database (Tinker and Deyl 2005; https://triticeaetoolbox.org/POOL/).

Phenotypic data

The phenotypic data for Kanota × Rollo originated from Mohler et al. (2012) and are based on seedling inoculation tests that used 12 to 16 plants for each $F_{2:3}$ line. The isolate HGB2/1, known to carry avirulence for *Pm3* from monosomic analysis of powdery mildew resistance in Rollo (Hsam et al. 2014), was spread in a settling tower on leaf segments at densities of 400–500 spores/cm^2. The leaf segments were cultured in plastic dishes on 6 g/l agar and 35 mg/l benzimidazole. The conditions of incubation were under continuous lighting at 10 μE/m^{2}s in a growth chamber at 17 °C and at 70% relative humidity. Ten days after inoculation, two classes of host reactions relative to the susceptible control cultivar Fuchs were distinguished: resistant (0–20%) and susceptible (> 50% infection); no intermediate (30–50%) infections were observed.

BLASTn analysis

Similarity to oat DNA sequences was searched for barley cDNA RFLP markers cMWG704 and cMWG733 by using their DNA sequences as queries against genetically mapped sequences and the hexaploid oat genome assembly lodged in the T3/oat database using default settings (https://triticeaetoolbox.org/oat/). The RFLP marker sequences were obtained from the GrainGenes database (https://wheat.pw.usda.gov). Matched sequences as well as all other oat marker sequences from the target linkage group Mrg18 used for genetic mapping were subsequently blasted against the wheat reference genome sequence (RefSeq_v1.0; International Wheat Genome Sequencing Consortium, 2018) in the Ensembl Plants database (http://plants.ensembl.org/index.html). The function of the detected high confidence protein-coding genes was retrieved from the T3/wheat database (https://triticeaetoolbox.org/wheat/) (Tables S3 and S4).

Genetic mapping

A total of 32 framework markers consisting of 6 K array SNP markers (GMI) and GBS markers (avgbs) and distributed along the linkage group Mrg18 of the oat consensus map (Chaffin et al. 2016) were used for SNP development and data collection (Table S3). The SNP assays were designed by Fluidigm Corporation (South San Francisco, USA). SNP marker genotypes were recorded on an EP1 genotyping platform using 192.24 Dynamic Array integrated fluidic circuits. All SNP genotyping analysis protocols can be found in the user guide published by the manufacturer (https://www.fluidigm.com). Genotyping with polymorphic SNP markers was done in double. The SNP data of

the mapping population Kanota×Rollo were merged with previously established genotypic data, i.e., RFLP and amplified fragment length polymorphism (AFLP) markers, and the binary *Pm3* phenotype (Mohler et al. 2012). To avoid complications in positioning tightly linked dominant markers from opposite linkage phases as accurately as possible (Mester et al. 2003), two separate but related linkage maps, both share the co-dominant markers, were computed with JoinMap® software version 5.0 (Kyazma BV, Wageningen, The Netherlands). The "maternal" map included dominant markers that were scored as heterozygous in the female parent (Kanota) and homozygous in the male parent (Rollo), while "paternal" markers were heterozygous in Rollo and homozygous in Kanota. Linkage of loci was claimed at a logarithm of the odds ratio (LOD) score ≥ 3.0, with a maximum recombination fraction of 0.4. Regression mapping was performed using the Haldane mapping function. Genetic linkage maps were drawn with Mapchart 2.1 software (Voorrips 2002). Chi-squared tests for goodness of fit were used to test for deviation of observed data from theoretically expected segregation ratios. Chi-squared values were corrected for continuity (http://vassarstats.net/csfit.html).

Results

Assignment of *Pm3* to linkage group Mrg18 of the oat consensus map

BLASTn search for the cDNA sequence of barley RFLP marker cMWG706, which is closely linked to oat powdery mildew resistance gene *Pm3*, in the T3/oat database produced significant alignments to GBS markers avgbs_249172 (bit score 80.6), avgbs2_5635 (bit score 78.8), avgbs2_165855 (bit score 73.4), and avgbs_670066 (bit score 66.2) of which avgbs_249172 and avgbs2_5635 represent the same sequence stretch (Fig. S1). The markers avgbs2_5635 and avgbs2_165855 were known to map at 69.9 cM and 20.2 cM, respectively, of linkage group Mrg18 of the oat consensus map (2016ExpandedConsensus_Mrg18). All five sequences identified the same high confidence protein-coding gene, a phosphoethanolamine methyltransferase triplicated on the long arm of homoeologous group-1 chromosomes, in the wheat reference genome sequence of Chinese Spring (Table S4). No significant hit to genetically mapped oat markers was obtained for cMWG733; however it produced, as cMWG706, significant alignments with oat chromosomes 1A and 1D and targeted a high confidence protein-coding gene of unknown function located on each of the three homoeologous group-1 chromosomes in the wheat reference genome sequence (Table S4).

Genetic mapping of *Pm3* relative to SNP markers from Mrg18

Of the 32 framework markers mainly chosen from the 56.0–76.7 cM region of linkage group Mrg18 of the oat consensus map, 13 showed polymorphism between the parental lines of the mapping population, 14 were monomorphic and 5 were failures (Table S3). Four SNP markers showed co-dominant inheritance, while all nine dominant markers were heterozygous for the paternal parent Rollo. The maternal genetic map had a map length of 13.1 cM and comprised seven markers including co-dominant RFLP marker cMWG706 and two dominant markers, one RFLP and AFLP each, from Mohler et al. (2012) (Fig. 1a). The resistance gene was bracketed by the RFLP marker loci cmwg706_*Dra*I and cmwg733_*Hind*III at map distances of 1.1 cM and 4.0 cM, respectively. The closest SNP marker locus was GMI_ES03_c2277_336 and mapped 3.8 cM proximal to *Pm3*. The paternal genetic map contained 17 markers and spanned a map distance of 31.7 cM (Fig. 1b). Two AFLP markers, E37M53-398 and E42M56-149, completed the data set. Eight markers were located within a 3.2 cM-interval around *Pm3*: 4 SNPs, 3 AFLPs, and RFLP cMWG706. No linkage in both maps was found for GMI_ES02_lrc12474_560 and GMI_ES03_c609_767 located at 36.2 cM and 56.0 cM, respectively, on consensus linkage group Mrg18. Six and 13 molecular markers had distorted segregation ratios in the maternal and the paternal map, respectively (Table S5). The marker with the greatest degree of segregation distortion in the Kanota×Rollo linkage group was GMI_ES03_c2277_336. The *Pm3* resistance locus and most of the SNP markers showed a deficiency of Kanota—the female parent—homozygotes.

Evaluation of the predictive ability of SNP markers for *Pm3*

Table S1 shows the genotypes and the genotype frequencies of the diversity panel for the 11 mapped SNP markers where the markers were ordered according to their position on the consensus linkage group Mrg18. Altogether, seven markers showed genotype frequencies ≤ 5% in the set of the 104 oat samples. For the remaining four markers, the minor genotype frequency ranged between 0.12 and 0.39.

For 53 oats of the genotype collection, the status of major powdery mildew resistance genes was known: 15 oats carried *Pm3* singly or in combination, 19 possessed other *Pm* genes, one was postulated to have an unknown *Pm* gene, and 18 reacted susceptible. Looking at these 53 oats, GMI_ES03_c2277_336 showed a distribution of genotype A:A similar to *Pm3*. Except for the oat cultivars Hinoat and Pendrwm, which were postulated to carry *Pm* gene combinations, the prediction of the presence or absence of *Pm3* in the
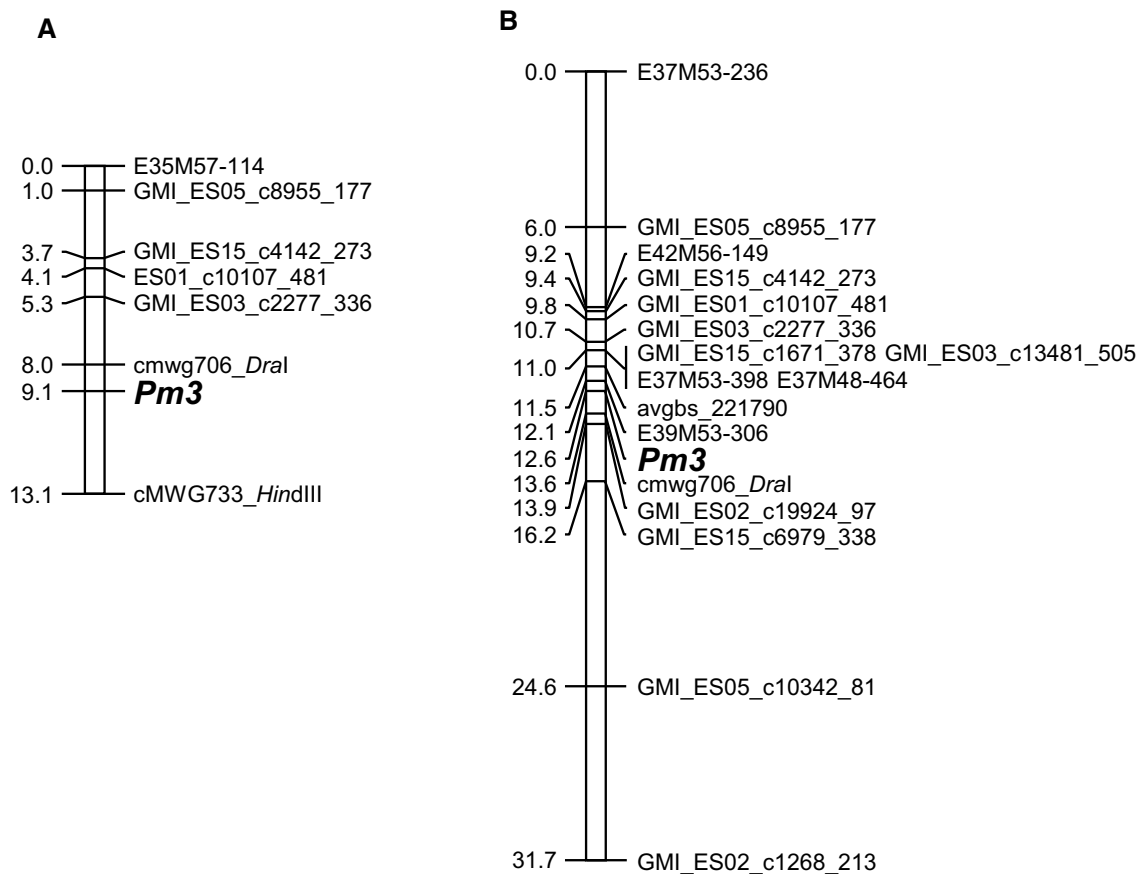
A

B

Fig. 1 Maternal **a** and **b** paternal genetic maps for the linkage group representing Mrg18 of the oat consensus map in mapping population Kanota×Rollo. Co-dominant markers are common to both maps. Absolute map positions in cM and marker names are shown on the left and right sides, respectively, of each genetic map

validation set was accurate (Table 1). The Swedish oat culti-var Galopp carrying *Pm3* was heterozygous for GMI_ES03_c2277_336. When extending the prediction to the full panel, oat cultivars AC Marie, Curley and Keely were suggested to carry *Pm3* (Table S1). Two other markers, GMI_ES15_c1671_378 and GMI_ES15_c4142_273, were also evaluated for their potential to predict *Pm3* in the sub-panel. However, prediction was limited for both markers. There were seven (1 false-positive and 6 false-negatives) and eight misclassifications (2 false-positives and 6 false-negatives) for GMI_ES15_c1671_378 and GMI_ES15_c4142_273, respectively (Table 1). For both markers, false-negative classifications were common to Mostyn and its derivatives Avalanche, Johanna and Manoire. The remaining 2 false-negative predictions were for Hinoat and Pendrwm which already were wrongly determined by GMI_ES03_c2277_336. Cultivar Galopp and *Pm10*-carrier AVE2925 showed a heterozygous allele configuration for both markers. The prediction for Cc 4146 possessing *Pm1* by GMI_ES15_c4142_273 was rated as false-positive.

Discussion

The present study reported the genetic mapping of the oat powdery mildew resistance gene *Pm3* relative to SNP markers derived from linkage group Mrg18 (recently determined to represent oat chromosome 1A) of the oat consensus map, and thus helped to gather information about the distribution of disease resistance genes across the oat genome. The initial identification of the target consensus linkage group was achieved by BLASTn similarity search for barley cDNA RFLP maker cMWG706, which was previously mapped in the vicinity of *Pm3*, in oat DNA sequences for which genetic map locations were also lodged in a database. The precise location of the matched oat marker allowed targeted marker enrichment for the genome region containing *Pm3*. The RFLP makers cMWG706 and cMWG733 (Table S4) and other 13 SNP markers chosen for the genetic mapping study (Table S3) identified high confidence protein-coding genes in the wheat reference genome sequence which all were located on each of the three homoeologous group-1 chromosomes

Table 1 Prediction of *Pm3* allelic status using linked SNP markers in a validation set of 53 oat genotypes

Genotype	Gene status	GMI_ES03_c2277_336	GMI_ES15_c1671_378	GMI_ES15_c4142_273
Cc4146	*Pm1*	G:G	C:C	T:T
Jumbo	*Pm1*	G:G	C:C	C:C
Lutz	*Pm1*	G:G	C:C	C:C
Maris Tabard	*Pm1*	G:G	C:C	C:C
Nelson	*Pm1*	G:G	C:C	C:C
Solva	*Pm1*	G:G	C:C	C:C
9065 Cn 18/53	*Pm3*	A:A	A:C	T:T
Avalanche	*Pm3*	A:A	C:C	C:C
Fuwi	*Pm3*	A:A	A:C	T:T
Galop	*Pm3*	A:G	A:C	C:T
Johanna	*Pm3*	A:A	C:C	C:C
Lowi	*Pm3*	A:A	A:C	T:T
Manoire	*Pm3*	A:A	C:C	C:C
Mostyn	*Pm3*	A:A	C:C	C:C
Nordstern	*Pm3*	A:A	A:C	T:T
Pewi	*Pm3*	A:A	A:C	T:T
Barra	*Pm3+Pm1*	A:A	A:C	T:T
Pendrwm	*Pm3+Pm6*	G:G	C:C	C:C
Rollo	*Pm3+Pm8*	A:A	A:C	T:T
NIC-91–7026	*Pm3+U*	A:A	A:C	T:T
Hinoat	*Pm3+U*	G:G	C:C	C:C
Av 1860	*Pm4*	G:G	C:C	C:C
Cc 6490	*Pm4*	G:G	C:C	C:C
Adamo	*Pm6*	G:G	C:C	C:C
Bruno	*Pm6*	G:G	C:C	C:C
Manod	*Pm6*	G:G	C:C	C:C
Maris Osprey	*Pm6*	G:G	C:C	C:C
APR122	*Pm7*	G:G	C:C	C:C
Bison	*Pm7*	G:G	C:C	C:C
Delfin	*Pm7*	G:G	C:C	C:C
Harmony	*Pm7*	G:G	C:C	C:C
Yukon	*Pm7*	G:G	C:C	C:C
AVE2406	*Pm9*	G:G	C:C	C:C
AVE2925	*Pm10*	G:G	A:C	C:T
Extraklock	U	G:G	C:C	C:C
Alf	no	G:G	C:C	C:C
AVA 572	no	G:G	C:C	C:C
Avesta	no	G:G	C:C	C:C
Condor	no	G:G	C:C	C:C
Ebene	no	G:G	C:C	C:C
Flämingsnova	no	G:G	C:C	C:C
Fuchs	no	G:G	C:C	C:C
Kanota	no	G:G	C:C	C:C
Klaus	no	G:G	C:C	C:C
Leo	no	G:G	C:C	C:C
Lorenz	no	G:G	C:C	C:C
Maris Quest	no	G:G	C:C	C:C

Table 1 (continued)

Genotype	Gene status	GMI_ES03_c2277_336	GMI_ES15_c1671_378	GMI_ES15_c4142_273
Milford	no	G:G	C:C	C:C
Pennel	no	G:G	C:C	C:C
Petra	no	G:G	C:C	C:C
Silwi	no	G:G	C:C	C:C
Tomba	no	G:G	C:C	C:C
Victory	no	G:G	C:C	C:C

further confirming the relationship of these chromosomes from oats and wheat (Jellen et al. 1995).

The powdery mildew resistance gene was located at 67.7–72.6 cM on Mrg18 of the oat consensus map, a position similar to the widely deployed oat stem rust resistance gene *Pg13* and crown rust resistance gene *Pc91* recently described in Kebede et al. (2020) indicating a possible clustering of disease resistance genes in this region of the oat genome. The likely occurrence of disease resistance gene clusters in the oat genome was recently predicted by annotating resistance gene analogs in the genomes of the diploid *A. atlantica* (A_sA_s) and *A. eriantha* (C_pC_p) species (Maughan et al. 2019). A cluster of resistance gene analogs on Mrg18 coincided with SNP marker GMI_ES03_c2277_336 which was found to be closely linked to *Pm3* in this study. In the study of Oliver et al. (2013), Mrg18 was associated with physical chromosomes assumed to carry the reciprocal translocation 7C-17A which is common to cultivated oats (Jellen and Beard 2000). In addition, the results presented by Kebede et al. (2020) indicated that *Pg13* and *Pc91* are near the 7C-17A translocation breakpoint. It is not clear whether this is also true for *Pm3*. However, a significant portion of the markers located on Mrg18 in the Kanota × Rollo population showed distorted segregation similar to the markers that were mapped to Mrg18 in the AC Morgan × CDC Morrison *Pg13* mapping population for which variation for the 7C-17A translocation was assumed (Kebede et al. 2020). Distorted segregation is often observed in crosses involving parental lines with and without the 7C-17A translocation (Wight et al. 2003). As our maternal line *A. byzantina* cv. Kanota does not carry the translocation (Jellen and Beard 2000), the skewed segregation of markers in the population could be due to the presence of this widespread chromosome mutation in the paternal line Rollo. In previous monosomic analyses involving the *Pm3*-carriers Mostyn (Hsam and Zeller 1998) and Rollo (Hsam et al. 2014), *Pm3* was located on chromosome 17A corresponding to the missing chromosome in the Kanota monosomic line K11 which was involved in the critical crosses. Thus, it can be concluded that the linkage group in Kanota × Rollo

corresponding to consensus linkage group Mrg18 represents chromatin from chromosome 17A. This conclusion is further supported by the observation that the part of Mrg18 targeted by GMI_ES03_c2277_336 corresponds to the distal part of chromosome AA2 of the diploid A-genome accession *A. atlantica* (Maughan et al. 2019).

Except for the cultivars Pendrwm and Hinoat, which were postulated to carry gene combinations, SNP marker GMI_ES03_c2277_336 showed a high prediction in the validation panel of 53 oats. It appears that there must be no doubt about previous gene postulations as the reaction patterns of Pendrwm and Hinoat fully corresponded to a combination of the reaction pattern of single-gene lines for *Pm1* and *Pm3* and *Pm3* and U from cultivar Extraklock, respectively (Hsam et al. 1997, 1998). However, the haplotypes of Pendrwm and Hinoat across the eleven mapped markers were similar to many other oats from the diversity panel (Table S1). In contrast to line 9065 Cn 18/53, which was the primary resistance source for the distribution of *Pm3* in oat cultivars and for which the marker prediction was true, line CD 3820, a diploid oat from the species *A. strigosa* recognized to carry *Pm3* (Hsam et al. 1997) and involved in the development of cultivar Hinoat, was not available for genotyping. The cultivar Mostyn, for which 9065 Cn 18/53 via line 05,443 was used as *Pm3* source, and its derivatives Avalanche, Johanna and Manoire were correctly determined by SNP marker GMI_ES03_c2277_336. This was also observed for *Pm3* resistance derived from sources of unknown origin in cultivars Pewi, Fuwi, Lowi, Rollo, Nordstern and breeding line NIC-91–7026 (Hsam et al. 1997). Barra was derived from a cross Selma//Palu/Saxo. The cultivar Palu in turn was developed from the cross Seger/von Lochow's Gelbhafer//Minor. As cultivars Minor, Selma and Saxo were susceptible (Hsam et al. 1998), Seger, which is a selection from the Swedish cultivar Milton, or von Lochow's Gelbhafer could have contributed the *Pm3* resistance in cultivar Barra. For cultivar Galopp, which was heterozygous for GMI_ES03_c2277_336, the origin of *Pm3* is even less clear (Hsam et al. 1998).

Although *Pm3* became ineffective over time (Okoń 2015; Okoń and Tociepa 2017) possibly due to its frequent use as sole resistance component in cultivars, the usefulness of *Pm3* in gene combinations is unknown, especially when there is high virulence for the gene in oat powdery mildew field populations. The knowledge of the genome location of *Pm3* and the availability of a highly informative linked marker will help in future QTL mapping studies to recognize whether this defeated gene contributes to adult plant resistance to powdery mildew. The availability of a consensus map along with its reference genome sequence has been found to be highly useful for the purpose of expanding the knowledge of the genetics of powdery mildew resistance in cultivated oat.

Declarations

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