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Mapping of Crown Rust (*Puccinia coronata* f. sp. *avenae*) Resistance Gene *Pc54* and a Novel Quantitative Trait Locus Effective Against Powdery Mildew (*Blumeria graminis* f. sp. *avenae*) in the Oat (*Avena sativa*) Line Pc54

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ABSTRACT

The Pc54 oat line carries the crown rust resistance gene *Pc54* and an unknown gene effective against powdery mildew. In this study, two recombinant inbred line (RIL) populations were developed to identify the genomic locations of the two genes and produce lists of molecular markers with a potential for marker-assisted selection. The RILs and parents were phenotyped for crown rust and powdery mildew in a controlled environment. They were also genotyped using the 6K Illumina Infinium iSelect oat single nucleotide polymorphism (SNP) chip. Multiple interval mapping placed *Pc54* on the linkage group Mrg02 (chromosome 7D) and the novel powdery mildew quantitative trait locus (QTL) *QPm.18* on Mrg18 (chromosome 1A) both in mapping and in the validating populations. A total of 9 and 31 significant molecular markers were identified linked with the *Pc54* gene and *QPm.18*, respectively. Reactions to crown rust inoculations have justified separate identities of *Pc54* from other genes and QTLs that have previously been reported on Mrg02 except for *qPCRFd*. *Pm3* is the only powdery mildew resistance gene previously mapped on Mrg18. However, the *pm3* differential line, Mostyn, was susceptible to the powdery mildew race used in this study, suggesting that *Pm3* and *QPm.18* are different genes. Determining the chromosomal locations of *Pc54* and *QPm.18* is helpful for better understanding of the molecular mechanism of resistance to crown rust and powdery mildew in oats. Furthermore, SNPs and single sequence repeats that are closely linked with the genes could be valuable for developing PCR-based molecular markers and facilitating the utilization of these genes in oat breeding programs.

Keywords: disease resistance, genetics, host parasite interactions

Crown rust caused by the fungus *Puccinia coronata* f. sp. *avenae* and powdery mildew caused by *Blumeria graminis* f. sp. *avenae* are two of the most important foliar diseases of cultivated oats, *Avena sativa* L. Crown rust is the main production constraint of oats in North America (Carson 2011) and is also present in most oat-producing countries of the world (Cabral and Park 2014; Sebesta et al. 2003; Simons 1985). Powdery mildew, on the other hand, is mainly common in cooler humid regions of Europe and South America (Roderick et al. 2000). The annual grain yield losses from crown rust and powdery mildew infections are estimated to range from 1.7 to 20% and 5 to 40%, respectively (Carson 2011; Okoń 2015). The use of fungicides to manage both diseases in oats is not recommended, as it is not economically justifiable or environmentally desirable, and excessive use may lead to the development of fungicide resistance in the pathogen populations (Martinelli 2004; May et al. 2014; Stevens et al. 2004). The use of resistant cultivars carrying effective genes against the pathogens is generally accepted as the best alternative to control crown rust and powdery mildew diseases in oats (May et al. 2014; McCartney et al. 2011; Stevens et al. 2004). Development and use of resistant cultivars against crown rust and powdery mildew should be a continuous process, as the virulence evolution of the pathogens is continuous and resistance breakdown is common (Carson 2011; Okoń and Ociepa 2018).

There are >100 crown rust race-specific resistance genes that have been described in oats. Some of these genes, such as *Pc38*, *Pc39*, *Pc48*, *Pc68*, and *Pc94*, have been deployed in cultivars that were commercialized in North America (McCallum et al. 2007). Of the >100 *Pc* genes, the map location of only a handful of them (*Pc38*, *Pc45*, *Pc53*, *Pc58 Pc68*, *Pc71*, *Pc91*, and *Pc94*) have been identified (Admassu-Yimer et al. 2018a; Bush and Wise 1998; Chen et al. 2006; Chong et al. 2004; Gnanesh et al. 2014; Hoffman et al. 2006; Wight et al. 2004) and were subsequently positioned on the oat consensus map (Chaffin et al. 2016). The lack of information about the chromosomal location of *Pc* genes and molecular markers linked to *Pc* genes have limited the utilization of genomic tools in oat breeding and caused difficulty when determining the novelty of newly identified quantitative trait loci (QTLs) that are effective against crown rust (Admassu-Yimer et al. 2018b).

There are 11 major powdery mildew resistance genes that have been described in oat (Herrmann and Mohler 2018; Hsam et al.

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2014; Ociepa et al. 2020; Okoń and Ociepa 2018). Six of these genes (*pm1*, *pm3*, *pm6*, *pm8*, *pm9*, and *pm10*) originated from cultivated oat (*A. sativa*), while the remaining genes were introgressed from wild oat species such as *A. hirtula* (*pm2*), *A. barbata* (*pm4*), *A. macrostachya* (*pm5*), *A. eriantha* (*pm7*), and *A. sterilis* (*pm1*, *pm3*, and *pm11*; Aung et al. 1977; Hsam et al. 2014; Ociepa et al. 2020). Some of these powdery mildew resistance genes have been utilized in oat breeding programs (Hsam et al. 2014). For example, *pm7* was successfully deployed in the German oat cultivars Canyon, Harmony, Delfin, Bison, and Yukon (Herrmann and Mohler 2018). Unfortunately, virulence in the *B. graminis* f. sp. *avenae* populations exist against all known resistance genes (Okoń and Ociepa 2018). Identification and characterization of additional powdery mildew resistance genes is imperative in the fight against powdery mildew in oats. Some types of oat germplasm exist that are resistant to powdery mildew, but they are not well characterized in terms of genetic control (Hsam et al. 1997; Okoń and Ociepa 2018). One such source of resistance to powdery mildew is the Pc54 oat line.

The *Pc54* gene, conferring seedling resistance to crown rust, was originally identified in an accession of the wild oat species, *A. sterilis* (CAV 1832), collected in the Mediterranean region of Turkey (Plant Gene Resources of Canada; http://pgrc3.agr.gc.ca/cgi-bin/npgs/html/acc_search.pl?accid=CN+21030). The accession was crossed and backcrossed with the *A. sativa* cultivar Pendek, to develop the single gene crown rust differential line, Pc54 (https://triticeaetoolbox.org/POOL; Martens et al. 1980). In addition to the crown rust resistance gene (*Pc54*) this line also carries an unknown gene effective against powdery mildew and the stem rust resistance gene *Pg15* (Martens et al. 1980; Roderick et al. 2000; Sebesta et al. 1993). The crown rust resistance gene *Pc54* has been effective against large proportion of the *P. coronata* f. sp. *avenae* isolates prevalent in North America compared with most known *Pc* genes (Carson 2008, 2011; Leonard 2007), suggesting that it might be a useful gene to utilize in oat breeding for crown rust resistance. The powdery mildew resistance in the Pc54 line is a widely deployed source in resistance breeding programs in Europe along with *Pm7* (Hsam et al. 2014). This study complements other studies that have mapped other crown rust and powdery mildew resistance genes and would improve the availability of information on chromosomal location of *Pc* and *pm* genes. Moreover, molecular markers linked to the genes may facilitate oat breeding for crown rust and powdery mildew resistance. Therefore, the objectives of this study were to map *Pc54* and the powdery mildew resistance on the oat genome using the 6K Infinium SNP chip (Illumina, San Diego, CA), and identify single nucleotide polymorphisms (SNPs) closely linked with *Pc54* and the powdery mildew resistance gene.

MATERIALS AND METHODS

Plant material. A population of F_5-derived recombinant inbred lines (RILs; $F_{5:6}$) was developed from a cross between the Pc54 differential line, Pendek*2/CAV 1832 (Pc54) and the susceptible cultivar Otana, developed by the U.S. Department of Agriculture's Agricultural Research Service (USDA-ARS), Aberdeen, ID (CI 5345/Zanster [CI 5345/2*Overland]). Quantities of 204 and 178 RILs were used to map the powdery mildew QTL and Pc54, respectively. The map locations of the powdery mildew resistance gene and *Pc54* were validated using 137 and 115 F_5-derived RILs from a cross between the Pc54 line and the Pc96 single gene line, RL1730 (Pc96), respectively. All mapping populations were developed by single seed descent at the Small Grains and Potato Germplasm Research Facility of the USDA-ARS in Aberdeen, ID. One hundred and sixty-five oat cultivars and breeding lines, predominantly originating from Aberystwyth University with additional material from the Germplasm Resource Unit at the John Innes Centre, UK, and varieties obtained commercially and from the Oat Core Collection (Esvelt Klos et al. 2017), were also used to validate the powdery mildew marker association.

Evaluation for crown rust reaction. Seedling tests were carried out on the two RIL populations (Pc54 × Otana and Pc54 × Pc96) at the Small Grains and Potato Germplasm Research Facility of the USDA-ARS in Aberdeen, Idaho in 2017 to 2018. The *P. coronata* f. sp. *avenae* race LGCG was used to evaluate the mapping population, Pc54 × Otana. The race was virulent against Otana, but avirulent against Pc54. Race LLMG was used to evaluate the validating population, Pc54 × Pc96. It was virulent against Pc96 but avirulent against Pc54. Each RIL was planted in three containers (five seeds per container), for a total of 15 seedlings evaluated per line. Seedlings were inoculated with spore suspension of the respective race at a concentration of 1×10^5 spores/ml at 2 weeks after planting, and later transferred into a dew chamber in the dark. After 18 h, the seedlings were placed in a growth chamber adjusted to 20 to 22°C. Disease reactions were recorded 14 days after inoculation as infection type (IT) on a 0 to 4 scale, where ITs 0 to 2 were considered as resistant (incompatible) and 3 to 4 were considered as susceptible (compatible) reactions, respectively (Chong et al. 2000). The detailed seedling inoculation of RILs and disease rating procedures were described in Admassu-Yimer et al. (2018a).

Evaluation for powdery mildew reaction. Plants of the two RIL populations (Pc54 × Otana and Pc54 × Pc96) and a range of control, modern, and historic varieties (Supplementary Table S1) were grown in 0.5-liter pots filled with peat/sand (3:1) in a glasshouse in Aberystwyth, UK under natural lighting and temperatures of 22°C during the day and 18°C at night. Powdery mildew inoculation and resistance assessment were conducted as described in Montilla-Bascón et al. (2015) using three independent plants per RIL or variety in a randomized block design. Inoculation was done by applying conidia onto leaves using a settling tower. After inoculation, plants were maintained in the glasshouse for a further 10 days before assessment of powdery mildew infection. For assessment of seedling resistance, plants were inoculated when the second leaf was fully expanded, and for adult plant resistance, plants were inoculated when the fifth leaf was fully expanded and macroscopically assessed relative to the susceptible control cultivar Selma without excising the leaves from the plant.

Genotyping. Isolation of high-quality DNA from F_5 plants of the two populations (Pc54 × Otana and Pc54 × Pc96) and the parents was performed as described in Admassu-Yimer et al. (2018a). Genotyping was performed with an Illumina Infinium iSelect oat SNP chip containing 4,975 SNPs at the Cereal Crops Research Unit of USDA-ARS in Fargo, ND. Genotype calling for each RIL and the parental lines was performed automatically using the DBSCAN procedure in GenomeStudio v.2.0 (Illumina), and was manually inspected for call accuracy. The marker data set was extended with 50 microsatellite markers using fluorescent primers and fragment analysis using an ABI 3730 sequencer (Applied Biosystems) and the software GeneMapper v.3.7 (Thermo Fisher Scientific, Waltham, MA).

Statistical analysis. Individual RILs in each population were classified as susceptible or resistant to crown rust and powdery mildew based on the reaction of $F_{5:6}$ families. The goodness-of-fit of the observed disease reaction to the expected segregation ratio of 1:1 for a single gene was tested using Pearson's χ^2 (X^2) distribution analyses.

Genetic mapping. Polymorphic SNP markers with <20% missing data were selected, and only SNPs that were previously assigned to the oat consensus map of Chaffin et al. (2016) were used for analyses. A total of 773 and 555 mapped polymorphic SNP markers were used in the Pc54 × Otana and Pc54 × Pc96 populations, respectively (Table 1). In addition, 42 and 25 polymorphic microsatellite loci were included in the development of the genetic linkage maps of the Pc54 × Otana and Pc54 × Pc96 populations, respectively. Population-specific linkage maps were developed from the genotyping data of both mapping populations using the software from JMP Genomics (v.9.0; SAS Institute, Cary, NC). The presence of main QTL effects was tested with a multiple interval

mapping (MIM) approach with forward selection in the software JMP Genomics. After running a permutation test, a minimum logarithm of odds (LOD) score of 3.0 was set to determine statistical significance. Genetic distances between markers and resistance loci were calculated in centiMorgans (cM) using the Kosambi map function (Kosambi 1943). Markers with significant association with either crown rust or mildew resistance were blasted against the *A. sativa* – OT3098 v2 sequence (https://wheat.pw.usda.gov/jb?data=/ggds/oat-ot3098v2-pepsico) in the database GrainGenes (https://wheat.pw.usda.gov/GG3/).

RESULTS

Crown rust reaction. The *P. coronata* f. sp. *avenae* isolate LGCG produced compatible ITs of 3 to 4 on the susceptible parent (Otana) while an incompatible IT value of 1 was observed on the resistant parent Pc54. Isolate LLMG produced ITs of 1 and 43+ on Pc54 and Pc96, respectively. Phenotyping of $F_{5:6}$ families indicated that the F_5 individuals in the Pc54 × Otana and Pc54 × Pc96 populations segregated at a ratio of 84R:94S and 56R:59S, respectively, indicating the presence of a single gene. Based on χ^2 tests, both populations segregated to fit to the Mendelian 1R:1S ratio (Table 2) indicating that the single-seed descent procedure worked as expected without introducing selection bias.

Powdery mildew reaction. The oat line Pc54 had a resistant and susceptible reaction to powdery mildew at the adult and seedling stages, respectively. The other parental lines, Otana and Pc96, were susceptible to powdery mildew at both seedling and adult plant stages. The segregation of powdery mildew resistance did not fit the expected 1:1 ratio with 61 and 64% of progeny displaying susceptibility in the mapping and validating populations, respectively (Table 2).

Genetic mapping of *Pc54* and powdery mildew resistance genes. After running the MIM procedure, evidence of linkage between the crown rust resistance gene *Pc54* and mapped markers was detected on Mrg02 of the oat consensus map (Chaffin et al. 2016). Four markers, GMI_ES03_c13331_202, GMI_ES03_c95_413, GMI_ES22_c2813_554, and GMI_ES15_c15279_258 had the highest LOD value at 62.1, followed by GMI_ES02_c16953_600 (LOD = 62.0), making them closely linked to *Pc54* (Table 3). The single sequence repeat (SSR) markers AME097_148 and AM07_152 were the others that were linked to *Pc54* with LOD = 25 and 7.4, respectively (Table 3). This region displayed collinearity to the 60 to 88 cM on Mrg02 of the oat consensus map (Chaffin et al. 2016), and has been identified as chromosome 7D (*A. sativa* – OT3098 v2, PepsiCo, https://wheat.pw.usda.gov/jb?data=/ggds/oat-ot3098v2-pepsico). The additive effect values of markers linked with the gene were aligned with the crown rust score of Pc54 line, indicating that the effective allele is from the resistant parental line.

MIM placed the powdery mildew resistance gene, *QPm.18*, on linkage group Mrg18 of the oat consensus map (Chaffin et al. 2016) at 67.7 to 72 cM. There were seven SNP and three SSR markers at the same de novo map position with the highest LOD value at 59.4 (Table 4). In total, there were 17 SNPs and six SSR markers with LOD = 20.2 to 59.4. BLAST analysis (https://blast.ncbi.nlm.nih.gov/Blast.cgi) of these markers identified them as all except for two being found on chromosome 1A of the OT3098 v2 reference genome (Table 4). Markers linked to *QPm.18* had additive effect values linking the source of resistance to the resistant parental line. Considerable segregation distortion was observed in the QTL region with an average 60% of the alleles present coming from the susceptible parent. This distortion was similar to that observed for the disease resistance phenotype.

Validation. One-hundred-and-fifteen RILs from the Pc54 × Pc96 population were used to validate the chromosomal locations of *Pc54* and the powdery mildew resistance gene. MIM placed *Pc54* on linkage group Mrg02 at 60 to 84.6 cM (Table 3) There were four SNPs and one SSR that were closely linked with *Pc54* in the validating population (Table 3). GMI_ES22_c2813_554 had the highest LOD value at 36.5. GMI_ES03_c95_413, GMI_ES15_c15279_258, and GMI_ES22_c2813_554 were common markers between the mapping and validating populations that were closely linked with *Pc54*.

The validating population placed the powdery mildew resistance gene on linkage group Mrg18 of the consensus map. Four SSR markers, MAMA13_238, OL0435_178, AB_AM907_249, and OL0221_144, with LOD = 44.7, 44.7, 48.7, and 42.5, respectively, were closely linked markers to the QTLs in the validating population, but their exact position on Mrg18 is not determined. Nine out of twelve of the SNP markers had LOD = 36.3 (Table 4). In total, there were 12 SNPs and four microsatellite loci that were significantly linked with the gene with LOD values ranging from 4.4 to 48.7 and spanning from 24.7 to 67.7 cM of the consensus map (Table 4). Again, segregation distortion was apparent in this region of the genome with 61% of alleles originating from the susceptible parent matching the distortion found in the powdery mildew resistance phenotype. The SNP loci GMI_GBS_4425, GMI_DS_CC7281_186, GMI_ES17_c806_849, and GMI_DS_LB_4840 along with the microsatellites AB_AM907_249, OL0435_178, MAMA13_238, and OL0221_144 were common to both maps and were linked with the powdery mildew resistance QTLs in both populations.

A survey of 168 oat breeding lines and varieties including the *Pm3* differential line Mostyn indicated that most lines displayed adult plant susceptibility to powdery mildew. All accessions that possessed the 180, 240, and 144 alleles from OL0435, MAMA13, and OL0221, respectively, displayed resistance (Table 5; Supplementary Table S1), suggesting that they possess the Pc54-derived mildew resistance. This was confirmed by examination of their pedigrees. Five other accessions (Blythe, Olympic, Anchor, Dyfed, and Orlando) also displayed resistance to powdery mildew but did not possess these alleles.

DISCUSSION

Crown rust resistance. *Pc54* was originally identified from *A. sterilis* accession CAV 1832, obtained from Turkey. The Pc54 differential line was developed using F_2 families in the Pendek × (CAV1832 × Pendek) backcrosses (Martens et al. 1980). This line was used as one of a set of single gene *Pc* differential lines for designating virulence phenotypes of *P. coronata* f. sp. *avenae* (Chong

TABLE 1. Number of single nucleotide polymorphisms (SNPs) between parental lines and mapped SNPs in the two mapping populations

Population	Number of SNPs (polymorphic)	Number of SNPs (mapped)	Coverage (cM)
Pc54 × Otana	859	773	2,285
Pc54 × Pc96	670	555	2,122

TABLE 2. Segregation ratio and χ^2 test analyses of $F_{5:6}$ progenies

Disease	Population	Generation	Resistant	Susceptible	Total	Expected ratio	χ^2	*P* value
Crown rust	Pc54 × Otana	$F_{5:6}$	84	94	178	1:1	0.46	0.497
	Pc54 × Pc96	$F_{5:6}$	56	59	115	1:1	0.04	0.842
Powdery mildew	Pc54 × Otana	$F_{5:6}$	79	125	204	1:1	10.4	0.001
	Pc54 × Pc96	$F_{5:6}$	39	98	137	1:1	26.3	0.0007

et al. 2000). Virulence to *Pc54* is present in the *P. coronata* f. sp. *avenae* populations of North America (Carson 2008, 2011; Leonard 2007). However, the proportion of isolates collected in North America with virulence to *Pc54* has never been >25%. In contrast, all isolates collected in 2009 were virulent against the widely deployed *Pc38*, *Pc39*, *Pc63*, *Pc67*, and *Pc96* genes (Carson 2011). This suggests that *Pc54* may have sufficient effectiveness to be useful in variety development when deployed in combination with other *Pc* genes.

QTLs conditioning crown rust resistance in oats were previously mapped on the same linkage group as *Pc54* (Mrg02). There are seedling resistance genes (*Pc58* complexes, *Pc38*, *Pc62*, and *Pc63*)

TABLE 3. Single nucleotide polymorphisms and simple sequence repeat markers closely linked with *Pc54* in two mapping populations indicating de novo map position, linkage group (Mrg), and position on the consensus map of Chaffin et al. (2016), and chromosome and position on the OT3098 Reference Sequence v.2[a]

		Consensus linkage map			*Pc54*		Location OT3098RefSeq v2	
Population	Marker	Mrg	Position (cM)	De novo map position (cM)	LOD	Additive effect	Chromosome	Position
Pc54 × Otana	AM07_152	2	nd	62.0	7.4	0.62	7D	460099831
	GMI_ES03_c13331_202	2	61.0	66.3	62.1	0.99	7D	448990058
	GMI_ES03_c95_413	2	84.6	67.3	62.1	0.98	7D	462940037
	GMI_ES02_c16953_600	2	85.2	67.8	62.0	0.98	7D	477997993
	GMI_ES22_c2813_554	2	87.3	68.3	62.1	0.98	7D	465109615
	GMI_ES15_c15279_258	2	87.3	68.3	62.1	0.98	7D	461451176
	AME097_148	2	nd	73.3	25.0	0.86	2A	431836859
Pc54 × Pc96	AM07_154	2	nd	38.8	10.9	0.73	7D	460099831
	GMI_ES03_c7453_413	2	60.0	63.2	8.1	0.53	2A	441406181
	GMI_ES03_c95_413	2	60.4	75.3	35.5	0.88	7D	462940037
	GMI_ES22_c2813_554	2	75.0	75.3	36.5	0.91	7D	461451176
	GMI_ES15_c15279_258	2	84.6	78.1	22.6	0.78	7D	465109615

[a] cM, centiMorgans; LOD, logarithm of odds; nd, not determined.

TABLE 4. Single nucleotide polymorphisms and simple sequence repeat markers closely linked with *QPm.18* in two mapping populations indicating de novo map position, linkage group (Mrg), and position on the consensus map of Chaffin et al. (2016), and chromosome and position on the OT3098 Reference Sequence v.2[a]

		Consensus linkage map			*QPm.18*		Location OT3098RefSeq v2	
Population	Marker	Mrg	Position (cM)	De novo map position (cM)	LOD	Additive effect	Chromosome	Position (bp)
Pc54 × Otana	GMI_GBS_4425	18	38.7	27.2	20.2	0.75	1A	461367099
	GMI_ES03_c11836_276	18	41.5	27.2	20.2	0.75	1A	459096341
	GMI_ES15_c8164_563	18	42.8	38.3	51.1	0.93	1A	353568418
	GMI_DS_CC7281_186	18	43.2	38.3	51.1	0.93	2D	47044888
	GMI_ES17_c806_849	18	46.5	38.3	51.1	0.93	1A	420951569
	GMI_ES03_c609_767	18	56.0	38.3	51.1	0.93	2D	62742151
	GMI_DS_LB_4840	18	56.0	38.3	51.1	0.93	1A	406190646
	GMI_ES13_c2873_647	18	67.7	38.3	51.1	0.93	1A	381674095
	GMI_GBS_9360	18	67.7	38.3	51.1	0.93	1A	393087077
	GMI_ES15_c8008_218	18	72.0	38.3	51.1	0.93	1A	371747613
	AB_AM907_249	nd	nd	38.3	51.1	0.93	nd	
	CDC1	nd	nd	38.3	51.1	0.93	1A	403213006
	OL0221_144	nd	nd	38.3	57.7	1.00	1A	442258110
	GMI_ES02_c17762_447	18	67.2	38.3	59.4	0.97	1A	401976834
	GMI_ES14_c8352_658	18	67.7	38.3	59.4	0.97	1A	390020240
	GMI_ES17_c19933_225	18	72.0	38.3	59.4	0.97	1A	371490307
	OL0435_178	nd	nd	38.3	59.4	0.97	1A	410258704
	MAMA03_333	nd	nd	38.3	59.4	0.97	1C	92964268
	MAMA13_238	nd	nd	38.3	59.4	0.97	1A	387351490
	GMI_ES15_c5371_294	18	72.6	38.3	59.4	0.97	1A	377728675
	GMI_ES17_c4128_744	18	72.6	38.3	59.4	0.97	1A	377584318
	GMI_ES15_c1671_378	18	72.6	38.3	59.4	0.97	1A	373686312
	GMI_ES15_c4142_273	18	72.6	38.3	59.4	0.97	1A	373529439
Pc54 × Pc96	GMI_ES02_lrc12474_560	18	36.2	24.7	4.4	0.30	1A	471882595
	GMI_GBS_4425	18	38.7	25.4	27.6	0.63	1A	461367099
	GMI_DS_CC7846_153	18	43.2	27.0	36.3	0.93	1A	426916285
	GMI_DS_CC7281_186	18	43.2	27.0	36.3	0.93	2D	47044888
	GMI_GBS_103591	18	43.2	27.0	36.3	0.93	1A	439637724
	GMI_ES17_c806_849	18	46.5	27.0	36.3	0.93	1A	420951569
	GMI_ES17_c5090_114	18	47.9	27.0	36.3	0.93	1A	411470502
	GMI_ES03_c13481_505	18	47.1	27.0	36.3	0.93	1A	409620742
	GMI_DS_LB_4840	18	56.0	27.0	36.3	0.93	1A	406190646
	GMI_ES02_c3392_447	18	67.7	27.0	36.3	0.93	1A	409039365
	GMI_DS_LB_10616	18	67.7	27.0	36.3	0.93	1A	380425584
	MAMA13_238	nd	nd	29.9	44.7	0.90	1A	387351490
	OL0435_178	nd	nd	29.9	44.7	0.90	1A	410258704
	AB_AM907_249	nd	nd	29.3	48.7	0.90	1A	442258110
	OL0221_144	nd	nd	33.4	42.5	0.80	1A	442258110
	GMI_GBS_90976	18	67.7	48.5	22.0	0.45	1A	434587553

[a] cM, centiMorgans; LOD, logarithm of odds; nd, not determined.

and QTLs (*QPc.Core.02*, *qPCRFd*, *Prq1a*, *Prq1b*, and *QCR.TxH-Mrg02*) previously reported on Mrg02 (Acevedo et al. 2010; Esvelt Klos et al. 2017; Hoffman et al. 2006; Jackson et al. 2007; Loarce et al. 2009; Portyanko et al. 2005; Sunstrum et al. 2019; Wight et al. 2004). Of the *Pc58* complex genes, *Pc58a* and *Pc58c* are located at 10.8 cM on the consensus map while *Pc58b* is at 110.4 cM (Hoffman et al. 2006; Jackson et al. 2007; Oliver et al. 2013). *Pc54*, at 84.6 cM, is unlikely to be one of the *Pc58* cluster genes. Similarly, the map location of SNPs associated with *QPc.Core.2* is 28 to 34 cM (Esvelt Klos et al. 2017), also far from *Pc54*. Crown rust resistance *Pc38* clusters with *Pc62* and *Pc63* (Harder et al. 1980) appears to overlap with the location of *Pc54*. The location of *qPCRFd* (~85 cM) also maps in this position (Wight et al. 2004). However, there is sufficient evidence documenting the difference between *Pc38* and *Pc54* in their reaction to *P. coronata* f. sp. *avenae* inoculation at seedling and adult plant stages to justify a separate identity (Martens et al. 1980). *qPCRFd* was detected in the oat line TAM-O-301 (the Pc58 differential line) in greenhouse and field studies using a single race isolate (Jackson et al. 2007). Although TAM-O-301 was initially described as carrying a single gene (*Pc58*), it was later confirmed to carry more than one (Hoffman et al. 2006). This suggests that additional studies may be needed to audit all the QTLs present in the Pc58 differential line, which may shed light on the relationship between *qPCRFd* and *Pc54*.

TABLE 5. Allele sizes found for simple sequence repeat markers in OL0435, MAMA13, and OL0221 for a subset of oat varieties and breeding lines[a]

Accession	Allele size			Adult plant mildew response
	OL0435	MAMA13	OL0221	
14063 Cn	180	240	144	R
14662Cn3/1	180	240	144	R
14675Cn1/1	180	240	144	R
14803Cn20/1	180	240	144	R
2008-32Cn1/1	180	240	144	R
2008-98Cn2/2	180	240	144	R
2009-18Cn17	180	240	144	R
Beacon	180	240	144	R
Elgar	180	240	144	R
Fergus	180	240	144	R
Galloway	180	240	144	R
Peloton	180	240	144	R
Rhapsody	180	240	144	R
Selwyn	180	240	144	R
Tardis	180	240	144	R
Olympic	178	230	134	R
Blythe	178	243	134	R
Dyfed	178	243	134	R
Orlando	178	243	134	R
Anchor	178	243	134	R
Barra	178	230	134	S
Caron	178	230	134	S
Chapline	178	230	134	S
Gandalf	178	230	134	S
Melys	178	230	134	S
Milo	178	230	134	S
Mostyn	178	230	134	S
SW Argyle	178	230	134	S
Buffalo	178	243	134	S
Buggy	178	243	134	S
Condor	178	243	134	S
Delfin	178	243	134	S
Dominik	178	243	134	S
Eagle	178	243	134	S
Flamingsgold	178	243	134	S
Lutz	178	243	134	S
Maldwyn	178	243	134	S
Mascani	178	243	134	S
Yukon	178	243	134	S
Red Rustproof	176	237	138	S
Akiyutaka	176	237	138	S
Black Mesdag	173	237	136	S
Stormugul II	173	237	136	S
ND9508252-9	173	241	136	S
Stainless	173	241	136	S

[a] R, resistant; S, susceptible.

Powdery mildew resistance. Resistance of the oat Pc54 line to powdery mildew was described in the literature (Jones and Hayes 1971; Roderick et al. 2000; Sebesta et al. 1993). They showed that the resistance in line Pc54 was conditioned by a single incompletely dominant gene. Since then, the Pc54 line has served as a source of resistance to powdery mildew in several oat breeding programs in Europe, and has been transferred to several oat varieties (Hsam et al. 2014; Sebesta et al. 1993).

In this study, line Pc54 was susceptible to powdery mildew at the seedling (two- to three-leaf) stage. However, as the plants grow (four- to five-leaf stage), the spread of powdery mildew on line Pc54 and resistant $F_{2:3}$ lines was restricted, expressed as necrotic flecks. On the other hand, the susceptible parents (Otana and line Pc96) and some of the $F_{5:6}$ lines were susceptible both at the seedling and adult plant stages. This concurs with previous literature for Pc54 (Jones and Hayes 1971; Sebesta et al. 1993), where the first leaf was infected by mildew, but the resistance improved from the third leaf upward, and with complete resistance at the inflorescence stage. This study also showed similarities in the reaction of the oat cultivar Maldwyn (Jones 1983) and Pc54 to powdery mildew infection, where disease resistance in both germplasms was expressed at adult plant stage.

All known powdery mildew resistance genes except *pm2* have been located on the oat genome. According to Hsam et al. (2014), updated by Herrmann and Mohler (2018), *pm1*, *pm3*, *pm4*, *pm5*, *pm6*, *pm7*, and *pm8* have been, respectively, mapped on Mrg11, Mrg18, Mrg04, Mrg20, Mrg05 or Mrg15, Mrg12, and Mrg03 of the oat consensus map of Chaffin et al. (2016). Similarly, Herrmann and Mohler (2018) located *pm9* and *pm10* on Mrg21 and Mrg03, respectively, and Ociepa et al. (2020) mapped *pm11* on Mrg12. According to Mohler et al. (2012), the powdery mildew resistance gene *pm3* is positioned between the barley cDNA RFLP marker loci cmwg706 and cmwg733, which have also been mapped to chromosome 1H of barley. Chromosome 1H of barley is homologous to the rice chromosome Os05 (Mayer et al. 2011). In turn, chromosome Os05 is homologous to the oat linkage group 7C-17A (= Mrg18 and Mrg28; Chaffin et al. 2016; Oliver et al. 2013). Hsam and Zeller (1998) have also confirmed the location of *pm3* on oat chromosome 17A using the *pm3* carrier Mostyn and monosomic F_1 plants, indicating that *pm3* is located on the same linkage group as the powdery mildew resistance in Pc54. Mohler (2021) assigned *pm3* to the region 67.7 to 72.6 on the oat Mrg18 of the oat consensus map in a similar position to the mildew resistance QTL found in this study, with the SNPs GMI_ES15_c1671_378 and GMI_ES15_c4142_273 found in common between the two studies. However, the *pm3* differential line, Mostyn (Jones 1983), was susceptible to the oat powdery mildew race used (Table 5; Supplementary Table S1), suggesting that *pm3* and the QTL in Pc54 may be different genes or alleles of the same gene. Several genes that are effective against other oat diseases were also identified in the same genomic region as the powdery mildew resistance QTL (*QPm.18*) identified in this study (Esvelt Klos et al. 2017; Gnanesh et al. 2014; Kebede et al. 2020; McCartney et al. 2011; McNish et al. 2020). Maughan et al. (2019) found that the *Pc91* gene, along with the QTLs *QPc.CORE.18.2* and *QPc.CORE.18.3*, map to the *A. atlantica* chromosome AA2 colocating with a predicted disease gene cluster. This region of the genome is associated with the 17A-7C translocation breakpoint found in many oat cultivars (Jellen and Beard 2000) and clustering of the disease resistance genes listed above may be associated with this region. This also is likely the reason for the distorted segregation of both the resistance phenotype and associated markers in this region found in this study, as is common in crosses that involve parental lines that contrast for the 17A-7C translocation (Wight et al. 2004).

In summary, we find that the *Pc54* and *QPm.18* genes conferring crown rust and powdery mildew resistance in the oat line Pc54 are linked with SNP markers placed on the oat consensus linkage groups Mrg02 (chromosome 7D) and Mrg18 (chromosome 1A), respectively. The identification of validated PCR-based markers (SSRs) enables the rapid selection of genotypes with *Pc54* and *QPm.18* as well as in the identification of homozygous and heterozygous genes in breeding programs. Determining the chromosomal location of *Pc54* and the powdery mildew resistance gene will help in understanding the molecular mechanism of resistance to crown rust and powdery mildew in oats. SNPs identified in this study that are closely linked with the genes can be used to develop further PCR-based molecular markers and facilitate the utilization of these genes in oat breeding programs. The results of this mapping data complement other similar literature, and contribute toward a more complete understanding of oat genomics.

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