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Preliminary study of QTLs for susceptibility to blue mold in apple (*Malus* x *domestica*) identified with genome-wide association mapping

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ABSTRACT

Purpose: Apple is one of the commercially most important fruits hosting a wide range of postharvest diseases that can threaten grower economy resulting in a yearly yield loss. Fungal diseases are the major problem in many countries, especially in organic culture as well as in countries with humid condition. Among the postharvest diseases, blue mold (caused by *Penicillium expansum*) is one of the most important. This disease is very destructive on apple, not only due to the economic damage but also because of mycotoxin patulin production. **Research method:** In the present study, we used wound inoculation and fruit trait data along with a large set of SNP data from the Axiom®Apple 480 K array to explore possible QTLs associated with rate of lesion decay, ripening period, fruit firmness and softening. **Findings:** Due to the very large number of SNPs in the present study, the significance threshold (5.63) was higher than in most other Genome-Wide Association Studies. However, some close to significant associations involving lesion decay were found on linkage group 3 (LG3), spanning a distance of 537 Kb (from 30,527,077 to 31,064,205). Interestingly, two SNPs associated with ripening period were also found on LG3 at the same position (30,494,523–30,885,771). **Research limitations:** This study could be extended with inclusion of more cultivars from different locations in order to minimize the impact of environment and to enhance the study power. **Originality/Value:** Although the genetic mechanism of blue mold resistance seems to be controlled by several genes in apple, our results indicate a possible association on LG3 which needs to be further investigated for candidate gene targeting. This finding may help to understand the genetic mechanism of blue mold disease in apple.

INTRODUCTION

Domesticated apple (*Malus* × *domestica*) is one of the most important fruits in the world but grower economy is severely threatened by different factors such as the occurrence of postharvest diseases. Storage rots caused by different fungi are especially problematic in commercially low-input orchards such as organic production. Frequently, the harvest has to be marketed after only a short storage period in order to avoid serious loss due to extensively damaged fruit, thereby lowering grower revenues. One of the most destructive postharvest diseases is blue mold, caused by *Penicillium expansum*. This pathogen is not only able to infect fruit through wounds or scars, both in the orchard and during storage, but is also capable of producing the mycotoxin patulin.

Previous studies have demonstrated considerable inter-cultivar genetic variation in the amount of blue mold damage shown after inoculation with spores of the fungus (Ahmadi-Afzadi et al., 2013; Naeem-Abadi et al., 2014). In most of these studies, a number of quantitatively inherited traits were associated with the level of resistance to blue mold, initial fruit firmness and softening rate i.e. loss of firmness during storage (Ahmadi-Afzadi et al., 2013; Naeem-Abadi et al., 2014), fruit ripening period (Ahmadi-Afzadi et al., 2013; Tahir et al., 2015, Nybom et al., 2020), fruit maturity stage (Torres et al., 2003; Janisiewicz et al., 2008; Chávez et al., 2014), fruit acidity (Prusky et al., 2004; Janisiewicz et al., 2008; Naeem-Abadi et al., 2014; Vilanova et al., 2014a) and content of phenolic compounds (Ahmadi-Afzadi et al., 2015; Naeem-Abadi & Keshavarzi, 2016; Sun et al., 2017; Ahmadi-Afzadi et al., 2018).

Quantitative trait loci (QTL) for some of these traits have already been mapped to the apple genome; QTL related to softening and fruit texture are co-located e.g. with *Md-PG1* on linkage group (LG) 10 (Costa *et al.* 2010; Longhi *et al.* 2012), fruit maturity is co-located with *MdMYB10* on LG9 (Morimoto et al., 2013), and a QTL associated to phenolic compounds is co-located with *LAR1* on LG16 (Chagné et al., 2012). For fruit ripening period, 6 SNPs were recently identified on LG3 (four SNPs), LG10 and LG16 in a GWAS approach (Urrestarazu et al., 2017). Recently, one study reported that the transcription factor NAC18.1 (MD03G1222600) on LG3 is the strongest predictor of fruit ripening (Migicovsky et al., 2021).

There have been no reports of major gene(s) for genetically determined resistance to blue mold in domesticated apple (*M.* x *domestica*) but two QTLs were reported on LG3 and LG10 in a mapping population of 'Royal Gala' × *Malus sieversii* PI613981 (Norelli et al., 2017). The stronger of these, on LG3, apparently derived from the *M. sieversii* parent. Analyses of the response to wound-inoculating wild genotypes of *M. sieversii* suggest that several mechanisms are involved in the high levels of resistance encountered in some of these genotypes (Janisiewicz et al., 2016). The extent to which the resistance mechanisms are present also in domesticated apple germplasm is not yet known, or whether some of them may be governed by the QTL on LG3.

Association mapping uses the linkage disequilibrium (LD) present among individuals from germplasm collections or natural populations to analyze the genetic basis of complex traits (Myles et al., 2009). Generally, germplasm collections contain more genetic variation than segregating populations. Since association mapping covers all the recombination events that have occurred in the evolutionary history of the studied populations, a more accurate mapping resolution is expected (Zhu et al., 2008; Myles et al., 2009; Ingvarsson & Street, 2011). Moreover, number of QTLs that can be mapped for a phenotype is not restricted to the segregating individuals in a specific cross, but rather by the number of QTLs associated to the trait and the degree to which the studied population reflects the genetic diversity (Zhu et al.,

2008; Yano et al., 2016). Association mapping has been applied for different traits in apple, especially after the release of high-density SNP arrays with uniform coverage of the whole apple genome (Chagné et al., 2012; Bianco et al., 2014; Urrestarazu et al., 2017).

Aim of the present study was to identify QTLs for susceptibility to blue mold by application of the high density Axiom®Apple 480 K SNP array (Bianco et al., 2016) developed within the EU-FruitBreedomics project (http://www.fruitbreedomics.com).

MATERIALS AND METHODS

Plant material

The association panel consisted of 182 unique diploid apple genotypes, all of which had been sampled in the genetic resource collections at SLU, Sweden (Supplementary Table 1). Depending on the harvest date of fruit, cultivars were ranged from early to late maturing cultivars. The majority of these is well adapted to cultivation in northern regions, and represents old local or national cultivars from Europe and N. America. For fruit phenotyping, fruits were harvested at a pre-climacteric stage, i.e. a harvest date suitable for long-term storage, according to the iodine starch test (starch conversion value 4–5 on a 9-point scale) (Smith et al., 1979).

Phenotypic data analysis

Phenotypic measurements for ripening period (i.e. harvest date), fruit firmness, fruit softening rate during storage were performed in several years during 2010-2015. Fruit firmness was measured on opposite, peeled sides of each fruit with a penetrometer (model FT-327, Effigy, Alfonsine, Italy, plunger diameter 11.1 mm, depth 7.9 mm). Fruit softening rate during storage was calculated as below (1):

$$Softening\ rate = \frac{(\text{fruit firmness at harvest} - \text{fruit firmness after 6–12 weeks storage})}{\text{number of weeks in storage}} \quad (1)$$

The rate of lesion decay after challenge by spores of *Penicillium expansum* inoculation was measured on fruits. Lesion decay was measured as the lesion diameter divided by weeks in storage before evaluation. The annual variation in estimation of lesion diameter was corrected based on averaged values for a set of control cultivars analyzed each year. Above-mentioned data collected in 2010 (Ahmadi-Afzadi et al., 2013), 2012 and 2013 (Tahir, 2015) and in 2015.

SNP genotyping

One hundred and eighty-two cultivars were genotyped as part of a larger set (1,168 unique genotypes; Urrestarazu et al., 2017) with the Axiom®Apple 480 K array containing 487,249 SNPs evenly distributed over the 17 apple chromosomes (Bianco et al., 2016). Application of stringent filters produced a set of 275,223 robust SNPs for GWAS (Bianco et al., 2016). SNP positions were determined according to the most recent version of the apple genome based on the doubled haploid GDDH13 (Daccord et al., 2017).

Kinship and population structure

The standardized relatedness matrix (**K**) was estimated using GEMMA software (Zhou & Stephens 2012). PLINK (Purcell et al., 2007) was used to perform a principal component analysis (PCA) of the SNP data, and the ten largest Eigenvalues were used to correct for population structure (**Q**). Matrix **Q** was constructed for all genotypes (Urrestarazu et al., 2017).

Table 1. List of the most significant SNPs associated to lesion decay in 182 apple cultivars resulted from GWAS study.

SNP No.	Linkage Group	position	-Log 10 (pvalue)
SNP1	3	30915232	5.29
SNP2	3	31027483	4.87
SNP3	3	31027563	4.87
SNP4	3	31027811	4.87
SNP5	3	30982672	4.80
SNP6	2	35320971	4.09
SNP7	7	21676244	4.08
SNP8	7	21691013	4.07
SNP9	12	27921051	3.97
SNP10	17	12535393	3.97

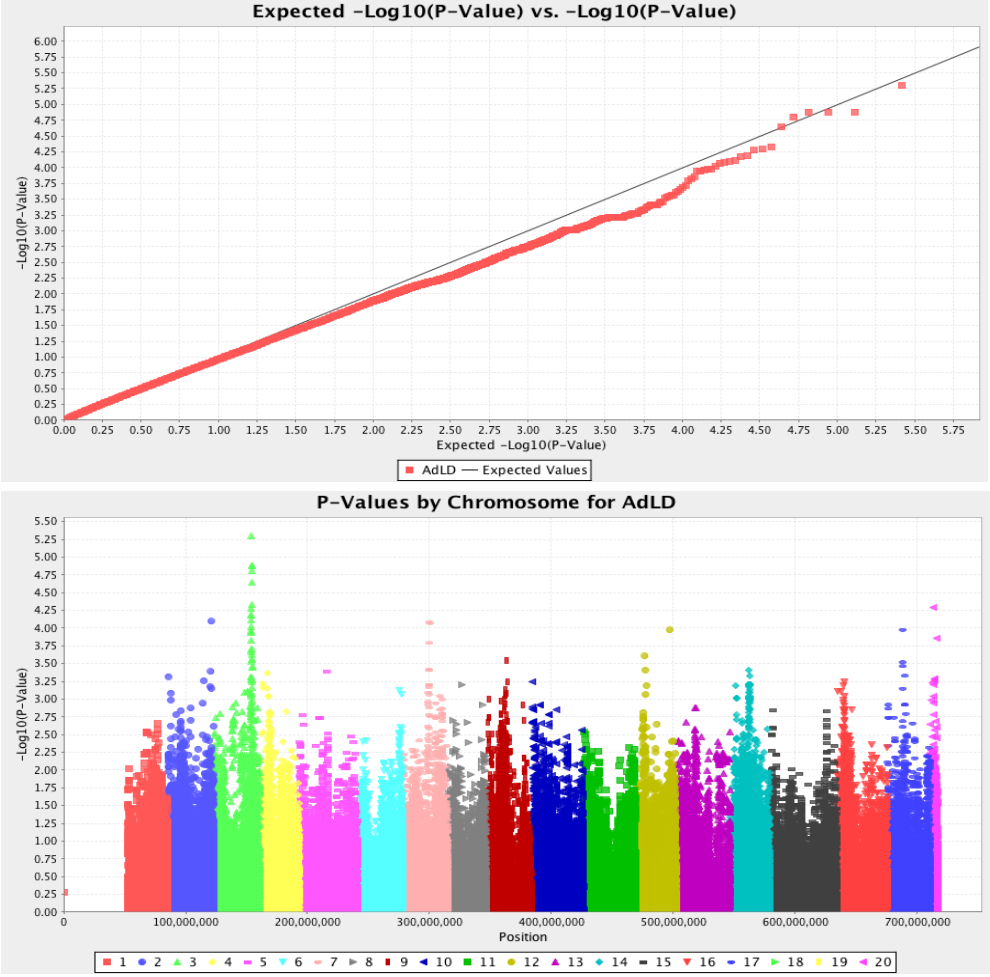

Fig. 1. Q-Q plot and Manhattan plots for lesion decay at a Bonferroni-corrected threshold of 0.05. Genome-wide significance threshold of $-\log_{10}(Pval) = 5.63$.

Genome-wide association study (GWAS)

GWAS was performed using TASSEL with correction for population structure (Q) and modeling phenotypic covariance with the kinship matrix (K) applied in a modified version of the multi-locus mixed model (MLMM) published by Segura et al. (2012). The Extended Bayesian Information Criterion (EBIC) proposed by Chen & Chen (2008) was conducted to select the best fitted model to our data. Coupel-Ledru et al. (2022) estimated the number of nearly independent SNPs among the 275,223 robust SNPs using a LD pruning method and obtained 23,200 independent SNPs, with a genome-wide significance threshold of $-\log_{10}(P\text{value}) = 5.63$.

RESULTS AND DISCUSSION

Due to the very large number of SNPs in the present study, the significance threshold (5.63) became higher than in most other GWAS. Although using a model with control for population structure and relatedness, none of the values obtained for analyses of the different traits reached above the significance threshold, and all $-\log_{10}$ (*P* value) were under the threshold of 5.63. Nevertheless, some of these traits produced data that indicate presence of a QTL, since it was possible to compare with previously reported QTL in the same positions for the same or similar traits.

Lesion decay

In our study, the strongest association involving lesion decay was found on LG3, involving four QTLs with $-\log_{10}$ (*P*value) ranged from 4.6 to 5.3 (Table1). This linkage group explained 69.5% of the genetic variation in resistance to blue mold. Interestingly, most of these SNPs spanned a distance of 537 Kb (from 30,527,077 to 31,064,205) on LG3 (Fig. 1). These SNPs are located at almost the same position (bottom) of LG3 as the 70 SNPs for ripening period reported by Urrestarazu et al. (2017). Most of these 70 SNPs on LG3 discovered at the bottom of this chromosome, spanned a distance of 2.05 Mb (29,196,200 to 31,243,065 bp). When a series of GWAS was performed in their materials, 31 of those SNPs showed a significant association not only in the six individual genotype collections from different countries but also in the whole population taken together. When analyses were carried out for individual collections, all the above-mentioned SNPs were located on LG3, except for three that were unmapped (Urrestarazu et al., 2017).

Interestingly, Norelli et al. (2017) found one QTL on LG3 (qM-*Pe*3.1) which explained 27.5% of the variation in terms of resistance to *P. expansum* in their material representing offspring from a cross between 'Royal Gala' (susceptible) and the *M. sieversii* genotype PI613981 originating in Central Asia (resistant). The qM-*Pe*3.1 QTL mapped from 67.3 to 74 cM on LG3 of the GMAL4593 genetic linkage map.

The location of our identified SNPs on LG3 overlaps with the determined new position of qM-*Pe*3.1 in GDDH13 v1.1 (Daccord et al., 2017) based on the previously reported physical location of the left border of qM-*Pe*3.1 (i.e. 26,848.396 in M × *d* v.1; Velasco et al., 2010). Interestingly, our data suggest that our set of samples, containing exclusively cultivars belonging to *M* × *domestica*, also harbors considerable variation for the same QTL on LG3. Selection of the best *domestica* genotypes for future plant breeding should make it possible to significantly improve the status of blue mold resistance within a single generation without losing too much in yield and fruit quality.

In the study by Norelli et al. (2017), a second QTL on LG10 (qM-*Pe*10.1) explained 14% of the variation in resistance to *P. expansum*. Second best associations (but much weaker) in

our study instead occurred on LG2 (position: 35,320,971), LG7 (position: 21,676,244–21,691,013), LG12 (position: 27,921,051) and LG17 (position: 12,535,393) (Table 1).

Table 2. List of the most significant SNPs associated to ripening period (harvest date) in 182 apple cultivars resulted from GWAS study.

SNP No.	Linkage Group	position	-Log 10 (p value)
SNP1	12	7257366	4.58
SNP2	12	7257850	4.58
SNP3	12	7264752	4.58
SNP4	3	30494523	4.57
SNP5	3	30885771	4.47
SNP6	16	8706306	4.26
SNP7	16	8707165	4.26
SNP8	16	8725879	4.26

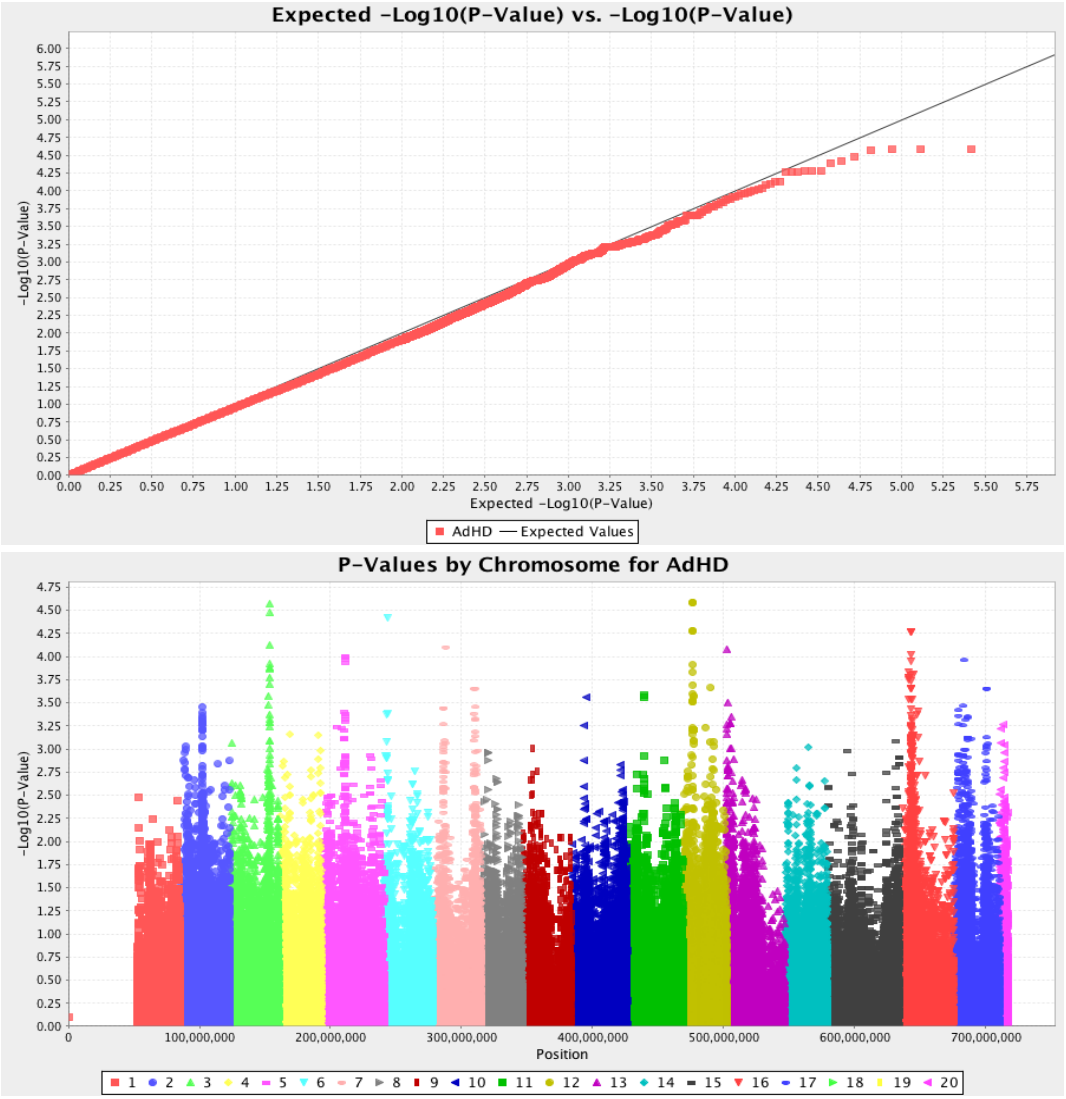

Fig. 2. Q-Q plot and Manhattan plots for ripening period at a Bonferroni-corrected threshold of 0.05. Genome-wide significance threshold of $-\log_{10}(Pval) = 5.63$.

Ripening period

The strongest associations involving ripening period (Table 2) in the present study were found on LG3 with –log10 = 4.6 at position 30,494,523–30,885,771 (spanning a distance of 391 kbp), and on LG12 with –log10 = 4.6 at position 7,257,366–7,264,752 (spanning a distance of 7386 bp) (Fig. 2). Interestingly, this is close to the position on LG3 (at the bottom) of the 4 SNPs (after application of EBIC criterion) previously found to be associated with ripening period (Urrestarazu et al., 2017). Migicovsky et al. (2021) has reported that the transcription factor *NAC18.1* is a strong predictor and may have a functional role in the fruit ripening process. They reported that the D5Y marker in *NAC18.1* had a 3 to 14 times greater effect on firmness at harvest and harvest date compared to the well-known genes *MdACO1*, *MdACS1* and *MdPG1* that are often screened in apple breeding programs.

Our third strongest association was located on LG16 (highest –log10 = 4.26) (Table 2). This may correspond to a QTL on the same LG found in Fuji (Kunihisa et al., 2016), and also reported previously in other studies (Kenis et al., 2008, Kunihisa et al., 2014). In Urrestarazu et al. (2017), one SNP was detected on LG16 (after EBIC criterion). In other studies (e.g. Kunihisa et al., 2014 and Urrestarazu et al., 2017), LG10 (bottom) has also been shown to harbor some QTLs but we did not find any indications in this region.

Fruit maturity stage but not ripening time was associated with blue mold symptoms in a set of apple genotypes investigated in Mexico (Chávez et al., 2014). However, fruit ripening time has been shown to be associated with blue mold resistance in previous studies when great care was taken to harvest all cultivars at the same stage of maturation (commercial harvest time) (Ahmadi-Afzadi et al., 2013; Tahir et al., 2015).

Firmness at harvest and softening

Results of GWAS did not show any strong signal for fruit firmness, instead several SNPs on different linkage groups showed weak associations (Fig. 3). The best (still weak) association with fruit firmness was found on LG16, LG15 and LG11 (the highest –log10 value close to 4) (Table 3). Previous studies have reported QTL for firmness on LG11 (Vilanova et al., 2014b; Sun et al., 2017) and we found weak associations in two regions of this linkage group (position: 11,196,126–11,230,129), but no genes have as yet been determined on this linkage group.

A QTL for the fruit firmness gene *MdPG1* has instead been reported on LG10 (Costa et al., 2010) but only a weak signal (max 3.75) was found in our study, possibly because there is little variation for *MdPG1* in the Balsgård gene bank (as determined by DNA marker analysis, Nybom et al., 2013). Also, *MdACO1* resides on this LG (Costa et al., 2005) but generally has smaller effects on firmness.

Table 3. List of the most significant SNPs associated to fruit firmness in 182 apple cultivars resulted from GWAS study.

SNP No.	Linkage Group	position	-Log 10 (pvalue)
SNP1	16	38364682	3.96
SNP2	15	50159612	3.90
SNP3	11	11230129	3.87
SNP4	17	9904088	3.87
SNP5	8	28215693	3.86
SNP6	11	11196126	3.84
SNP8	11	11196576	3.64
SNP9	10	39970155	3.63

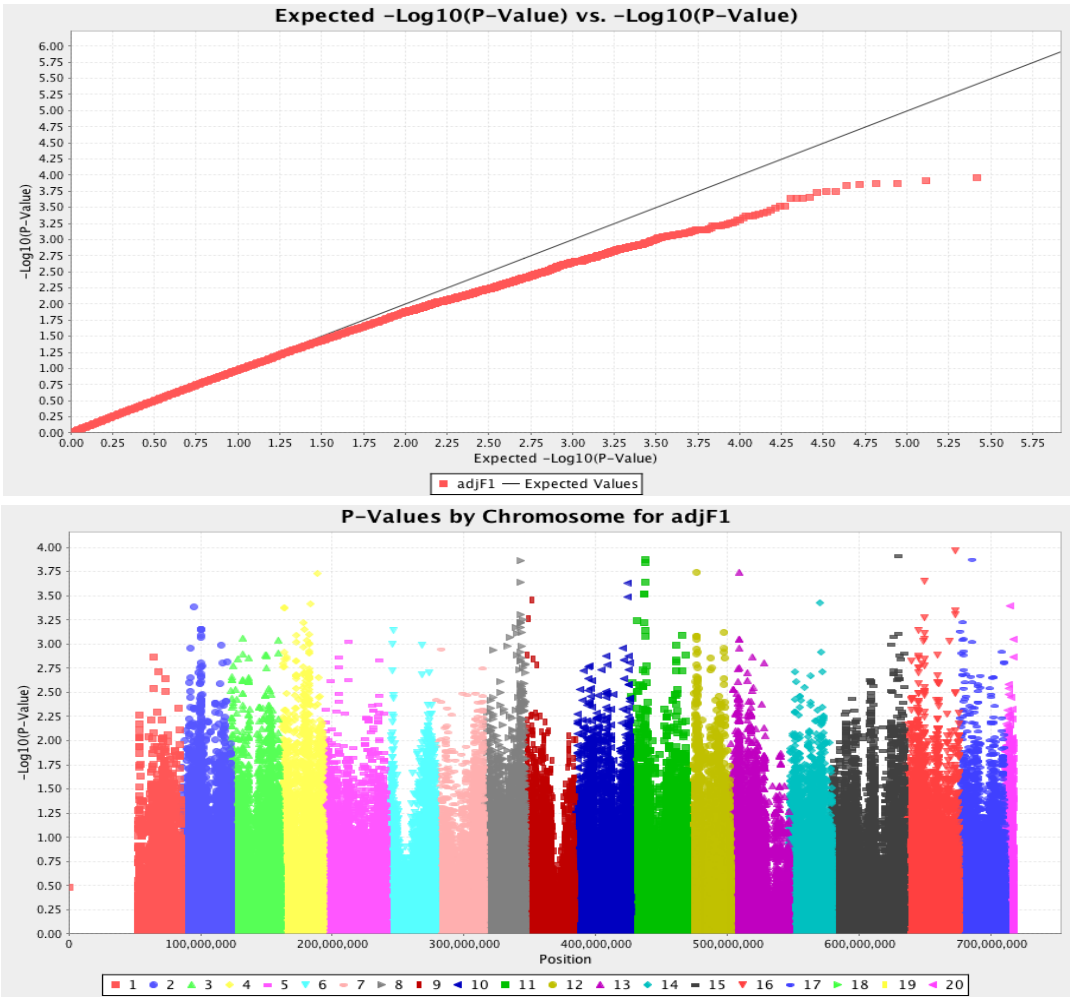

Fig. 3. Q-Q plot and Manhattan plots for firmness at harvest at a Bonferroni-corrected threshold of 0.05. Genome-wide significance threshold of $-\log_{10}(P\text{val}) = 5.63$.

CONCLUSION

The genetic mechanism of blue mold resistance seems to be controlled by several genes in apple. In addition, different pomological traits as well as the chemical contents of the fruit can indirectly affect lesion development when challenged by *P. expansum*. However, we found four closes-to- significant SNPs in the bottom part of LG3 at the same position as QTLs previously described by Norelli et al. (2017). This finding is highly interesting, since these SNPs would indicate that there is considerable variation for *P. expansum* resistance not just in the wild species of *M. sieversii*, but also in *M.* × *domestica*. Nevertheless, due to very large number of SNPs in the present study, the significance threshold (5.63) was higher than in most other GWAS, thus the significance level of our QTLs was below the threshold.

This finding may indicate that a larger number of genotypes from several locations/countries are needed to achieve more significant results. We thus recommend increasing sample size in order to minimize the impact of environmental factors, and to obtain a higher level of association between SNPs and traits of interest. The likelihood of finding SNPs with small impact sizes can be increased by reducing random variation using a larger sample size. However, this preliminary finding has highlighted the bottom section on LG3 as the most important chromosomal location involved in lesion development, thereby suggesting that it may contain several important candidate genes.

Conflict of interest

The authors have no conflict of interest to report.

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