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Genome-wide association studies of resistance to dieback diseases in a pseudo-F2 mango population

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ABSTRACT. *Lasiodiplodia theobromae* and *Neofusicoccum parvum* are important fungi affecting mango trees in Northeast Brazil, a prominent region of mango export. Genome-wide association studies in a 'Haden' × 'Tommy Atkins' mango pseudo-F_2 population were performed for symptoms of both fungal diseases to support the development of new cultivars by applying marker-assisted selection. 'Haden' is resistant while 'Tommy Atkins' is susceptible to both fungal diseases. Single nucleotide polymorphism (SNP) and microsatellite data of 95 progenies were analyzed by allelic and genotypic association and by general (GLM) and mixed linear models (MLM). Artificial pathogen inoculation was performed on 15-year-old progenies by manually spraying a 10^3 conidia mL^{-1} suspension on young branches and leaves. The plants were considered resistant when the absence of symptoms was ≥ 90% over three different evaluations. Consensus genomic associations were identified on chromosome 12; position 10.60 Mb (Mi_0096) and 1 (Mango_rep_c1316) position 14.67 Mb, with a significant association with *L. theobromae* symptoms, accounting for 20% of the total variation. Additional regions identified exclusively by GLM and MLM analysis, in chromosomes 11 and 8 (positions 24.57 Mb and 9.34 MB, respectively), explain 36% of the symptoms variation of this disease. Consensus genomic associations were identified on

chromosomes 2, position 20.49 Mb (Mango_rep_c9407) and 9, position 15.01 Mb (Mango_rep_c8984), with a significant association with *N. parvum* symptoms, accounting for 21% of total resistance to this fungus. An additional region identified exclusively by GLM and MLM analysis, in chromosome 12 (Mango_rep_c7620, position 14.29 MB), explains 29% of the total variation of this disease. Qualitative and quantitative genome association methods run together enabled the identification of consensus chromosomal regions controlling resistance to these diseases. These chromosome regions are candidates for saturation with SNPs or further genome data mining to apply marker-assisted selection in mangoes.

Key words: *Mangifera indica*; *Lasiodiplodia theobromae*; *Neofusicoccum parvum*; GWAS; Plink; Tassel

INTRODUCTION

The world production of mangoes (*Mangifera indica* – Anacardiaceae) reached, approximately, 57 million metric tons in 2021, increasing 8.3% since 2017, with India, China, and Thailand accounting for 55% of global production (FAO, 2021). Mexico, Thailand, the Netherlands, Peru, Brazil, and India accounted for 70% of mango exports in 2021, while the United States of America, the Netherlands, Vietnam, Germany, the United Kingdom of Great Britain and Northern Ireland, and the United Arab Emirates accounted for 60% of mango imports in 2021 (FAO, 2021). Mango fruits have an attractive visual appearance (Wang et al., 2020) and mango popularity is on the rise due to its high nutraceutical and pharmaceutical values, including the polyphenol mangiferin, the most abundant and bioactive compound in this fruit (Masibo and He, 2009). According to Galán Saúco (2017), European and North American consumers prefer brightly colored mangoes with a well-balanced sugar/acidity ratio, and Florida cultivars dominate the global fresh-fruit export market.

Mango ($2n$ = 40 chromosomes) is a fruit tree that originated in Eastern India, Assam to Myanmar, and the species was spread to Southeast Asia and later to Africa and America (Duval et al., 2005). In Brazil, Portuguese navigators introduced the first mango varieties in the 17th century (Mukherjee and Litz, 2009). The juvenile mango period ranges from three to seven years, and trees can remain in orchards for hundreds of years (Mukherjee and Litz, 2009). The mango's long juvenile period highlights the importance to develop new methods, such as marker-assisted selection, to speed up the development of new cultivars.

Mango crops are affected by several diseases; those caused by *Lasiodiplodia theobromae* and *Neofusicoccum parvum*, Botryosphaeriaceae complex, are the most common in Northeast Brazil. Dieback is one of the main symptoms observed in plants infected by these two fungi (Coelho et al., 2018). Without any control measures, plants affected by these fungi begin to decline and die, and pruning to restore plant health makes the process more expensive because it is not possible to cure a plant through fungicide applications alone (Coelho et al., 2020).

Mango disease resistance inheritance studies are restricted to genetic parameter estimations, and genome or linkage mapping of diseases is not available. The sequencing and assembling of the mango genome at a chromosome level for cultivar Alphonso (Wang et al., 2020), makes it possible to perform trait association and fine mapping of this species. Dautt-Castro et al. (2019) report genome-wide identification of mango polygalacturonases based on an unpublished Tommy Atkins mango genome, making additional analysis impossible. Harper et al. (2016) report associative transcriptomics of dieback disease in the European ash tree (*Fraxinus excelsior*), identifying markers strongly associated with canopy damage in infected trees, enabling the identification of individuals with a low level of susceptibility to the disease in unrelated trees.

Here we report the application of genome-wide association studies (GWAS) to identify single-nucleotide polymorphisms (SNPs) and microsatellite loci associated with *L. theobromae* and *N. parvum* dieback diseases to support the development of marker-assisted selection for the improvement of new mango cultivars.

MATERIAL AND METHODS

Plant Material and DNA extraction

Ninety-five plants of a 'Haden' × 'Tommy Atkins' pseudo-F_2 population were obtained by identifying two isolated 'Haden' plants surrounded by hundreds of 'Tommy Atkins' plants in a commercial orchard, located in Petrolina, PE, Brazil. Progenies were developed in a nursery and, after reaching 50 cm height, were established in the field in 2002 at Mandacaru Experimental Station at the Brazilian feceral agricultural research institution - Embrapa, in Juazeiro, BA. Crop management, widely adopted in this region for mango, was applied, including early floral induction with the plant growth treatment paclobutrazol (PBZ) and potassium nitrate applications associated with pruning and irrigation measures. Selfed and off-typed plants were identified beforehand by SNPs and excluded from the genetic analyses. According to Coelho et al. (2020), 'Haden' is resistant while 'Tommy Atkins' is susceptible to both fungi, based on evaluation in three different inoculation periods. This population is part of an ongoing Embrapa mango breeding program in the Brazilian semi-arid region, with Petrolina, PE as a reference city, being one of the most prominent regions of mango export.

DNA was extracted from healthy and young leaves sampled at the Mandacaru Experimental Station for each pseudo-F_2 plant, according to the CTAB 2x protocol, with minor modifications (Ribeiro et al., 2012). After extraction, DNA integrity was checked on 0.8% agarose gel, followed by fluorescence quantification and dilution to 10 ng/µL.

Phenotyping for *L. theobromae* and *N. parvum*

Isolates of *L. theobromae* and *N. parvum* were provided by the mycology laboratory of the Universidade Federal Rural de Pernambuco (UFRPE), Recife, PE. The fungi were grown for 15 days in a 2% water-agar culture medium in Petri dishes and the pycnidia were released by flooding the fungal colony with sterile distilled water and the surface scraped with glass slide. The solution containing reproductive structures was poured off and filtered through sterile gauze and harvested into a beaker. Pycnidia were softly macerated with a

sterilized pestle in a sterilized mortar containing distilled-sterilized water. The content was filtered and the concentration of the suspension determined with a Neubauer chamber and then adjusted to a concentration of 10^3 conidia mL^{-1}.

An inoculum suspension of 10^3 conidia mL^{-1} was softly sprayed, without provoking injuries to the plant, with the aid of a hand sprayer, on healthy young single branches with young leaves, for each fungal inoculum, until runoff occurred (Coelho et al., 2018). After spraying, the branches were protected in a wet chamber made of plastic bags moistened with distilled-sterilized water, for 48 hours. To prevent burning of leaves and branches by the incidence of solar radiation, the plastic bags were covered with paper bags. The dieback symptoms were evaluated five days after opening the wet chamber. This procedure was repeated three times for both pathogens, at different periods, in each 'Haden' × 'Tommy Atkins' progeny, separately for each fungus.

The method for disease symptom evaluation was an adaptation of the percentage infection method described by Coelho et al. (2018): plants without symptoms = (Number of branches without symptoms/total number of inoculated branches) x 100. Plants without symptoms with a mean greater than 90% were considered as resistant to *L. theobromae* or *N. parvum*. Plants with a percentage smaller than 80% in a unique evaluation were considered as susceptible to the pathogens.

Genotyping, physical mapping and statistical analysis

Pseudo-F_2 plants were genotyped with 705 SNPs, as described by Kuhn et al. (2017). Fifty-four microsatellite loci were genotyped, as described by Ribeiro et al. (2012), after initial screening for a large number of such markers published by Duval et al. (2005), Viruel et al. (2005), Honsho et al. (2005), Schnell et al. (2005), Ravishanka et al. (2011), Alves et al. (2016) and Dillon et al. (2014). Markers and plants with missing data greater than 10% were deleted from the dataset.

For qualitative genomic associations, based on the presence or absence of disease symptoms, allelic and Fisher's exact tests were performed, as well as genotypic association by the Cochran-Armitage trend test and these analyses were implemented with the DOS Plink program (Purcell et al., 2007). Plants were coded 1 for susceptible (presence of symptoms <90%) and 2 for resistant (absence of symptoms ≥ 90%) for both *L. theobromae* and *N. parvum* fungi.

For quantitative genomic associations and the percentage evaluations for the absence of symptoms, analyses were performed with the general (GLM) and the mixed linear model (MLM) available in the Tassel 5.2.65 software (Bradbury et al., 2007). In the GLM analysis for *L. theobromae*, genotyping and phenotyping data plus principal components (PC) were considered, and for the MLM analysis, an identical by state (IBS) matrix, estimated using Plink 1.9 (Purcell et al., 2007), was included. In the PC estimation, the accumulation of 50% of the total variation in the first eigenvectors was considered (Bradbury et al., 2007). In the GLM analysis for *N. parvum*, only data from genotyping and phenotyping were considered, while in the MLM analysis, the IBS matrix estimated using the Plink 1.9 program was also considered (Purcell et al., 2007). Percentage data were transformed to arcsine + 0.01 and arcsine for *L. theobromae* and *N. parvum*, respectively.

SNP sequences, published by Kuhn et al. (2017), and microsatellite sequences, obtained from the GenBank accession number, were blasted into the genome of *M. indica*

(Wang et al., 2020) available at NCBI. Loci were assigned to mango chromosomes based on high e-value and bit score. SNPs and microsatellites were ordered for each chromosome in an Excel spreadsheet.

RESULTS

Only four and 14 pseudo-F_2 plants were resistant (>93%) to *L. theobromae* and *N. parvum*, respectively, in the three field inoculation tests. The symptoms of the evaluated inoculations were characteristic of dieback disease, noted by a progressive drying out of the leaves and branches. Pruning was done to stop advances towards the trunk that can result in plant death and loss of the segregating population.

Qualitative genomic association - allelic and genotypic tests

For *L. theobromae*, four SNPs were identified on chromosome 12 by the allelic association, one SNP by Fisher's exact test, on chromosome 12, and four SNPs by the Cochran-Armitage trend genotypic test: one on chromosome 12, two on chromosome 1 and one on chromosome 16 (Table 1). The common SNP in these analyses was Mi_0096, with *P-value*<0.008397 (Table 1), on chromosome 12, thus making it a strong candidate for association with *L. theobromae* resistant loci.

Table 1. SNPs with allelic and genotypic statistical association (*P-value*<0.02) to *Lasiodiplodia theobromae* resistance loci in a 'Haden' × 'Tommy Atkins' mango pseudo-F_2 population.

SNP	Chromosome	Position (bp)	*P-value*
	Fisher's exact test		
Mi_0096	12	10604128	0.008184
	Allelic test		
Mi_0096	12	10604128	0.007949
Mango_rep_c59614	12	14675191	0.0142
Mi_0287	12	14543232	0.0154
Mi_0070	12	15285720	0.01555
	Cochran-Armitage trend genotypic test		
Mi_0096	12	10604128	0.008397
Mango_rep_c1316	1	24576970	0.008895
Mango_rep_c5591	16	8818242	0.01818
Mi_0110	1	2534890	0.01978

For *N. parvum*, seven SNPs were identified on chromosomes 4 and 11 by the allelic test, five SNP by Fisher's exact test, on chromosomes 4 and 11, and eight SNPs by the Cochran-Armitage trend genotypic test, on chromosomes 11, 4, 1 and 9 (Table 2). SNPs Mi_0140, Mi_0244, Mi_0433 and Mi_0294 were common in these analyses, with *P-value*<0.006940 (Table 2), on chromosomes 4 and 11, being strong candidates for association with *N. parvum* resistance loci.

Table 2. SNPs with an allelic and genotypic statistical association (*P-value*<0.009) with *Neofusicocum parvum* resistance loci in a 'Haden' × 'Tommy Atkins' mango pseudo-F_2 population.

SNP	Chromosome	Position (bp)	*P-value*
	Fisher's exact test		
Mi_0140	4	17199651	0.002642
Mi_0244	11	869763	0.008104
Mi_0433	11	2102539	0.008104
Mi_0294	11	896127	0.008657
Mi_0405	4	17245187	0.009457
	Allelic test		
Mi_0140	4	17199651	0.00228
Mi_0244	11	869763	0.004439
Mi_0433	11	2102539	0.004439
Mi_0294	11	896127	0.006527
Mi_0405	4	17245187	0.008002
Contig2867	4	15515955	0.00903
Mango_rep_c9407	2	20496307	0.00974
	Cochran-Armitage trend genotypic test		
Contig2867	4	15515955	0.002721
Mi_0030	1	22890486	0.003674
Mi_0140	4	17199651	0.004609
Mi_0244	11	869763	0.004927
Mango_rep_c8984	9	15016915	0.005247
Mi_0294	11	896127	0.006439
Mi_0433	11	2102539	0.006940
Mango_c33092	4	18064217	0.007244

Quantitative genomic association - general and mixed linear models

Four SNPs were associated with loci-controlling *L. theobromae* resistance (*P-value* <0.0049) in the GLM analysis and these same SNPs presented a significant association, but with a different *P-value*, in the MLM analysis (Table 3, Figure 1). SNPs were identified on chromosomes 12, 1, 11 and 8 of the mango genome, with R^2 ranging from 7.27% to 11.37% (Table 3).

Table 3. SNPs with significant association* (*P-value*<0.007) and coefficient of determination (R^2) by the General (GLM) and Mixed Linear Model (MLM) to *Lasiodiplodia theobromae* resistance loci in a 'Haden' × 'Tommy Atkins' mango pseudo-F_2 population.

SNP	Chromosome	Position (bp)	*P-value*	R^2
GLM				
Mi_0096	12	10604128	0.00162	0.11371
Mango_rep_c1316	1	24576970	0.00195	0.08715
SSKP031C1_A370G	11	10604128	0.00223	0.08963
Mi_0278	8	9349041	0.0049	0.07272
MLM				
Mango_rep_c1316	1	24576970	0.00339	0.08715
SSKP031C1_A370G	11	10604128	0.00435	0.08963
Mi_0096	12	10604128	0.00446	0.11371
Mi_0278	8	9349041	0.00717	0.07272

* Percentage data for symptoms of *L. theobromae* transformed to arcsene + 0.01

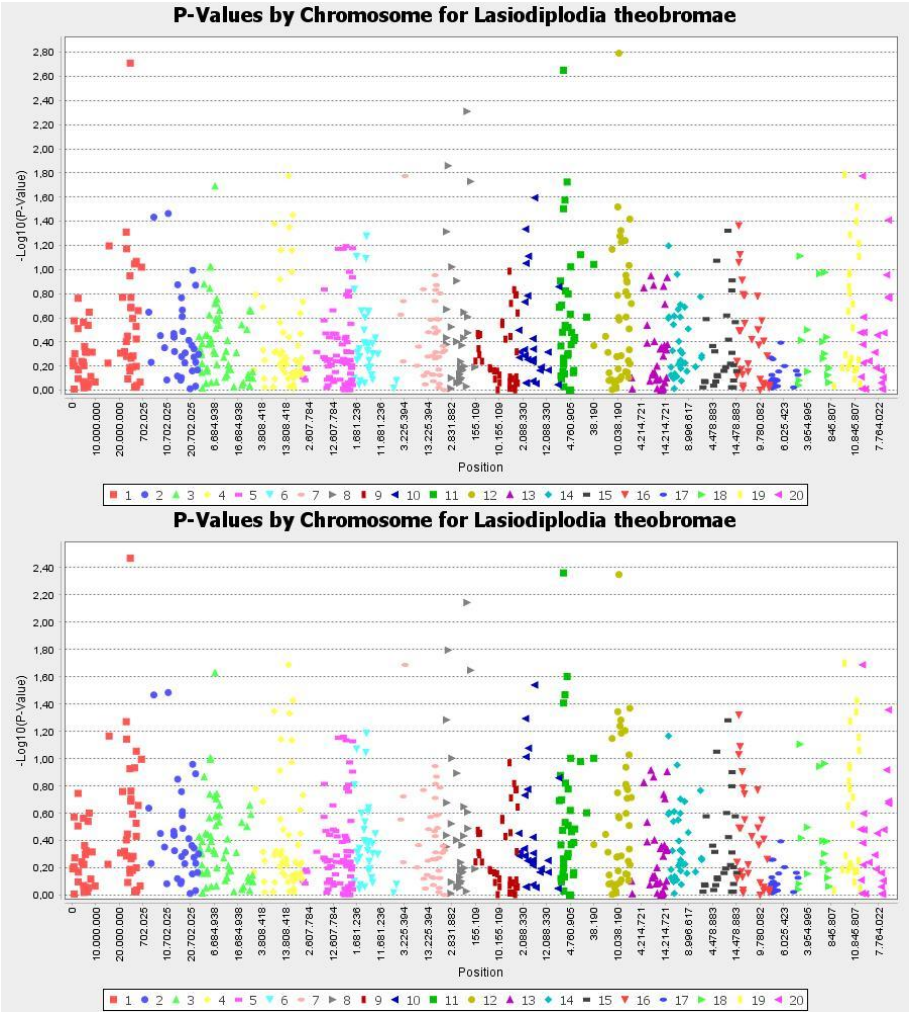

Figure 1. Manhattan plot for SNPs with association for resistance to *Lasiodiplodia theobromae* in a 'Haden' × 'Tommy Atkins' mango pseudo-F_2 population: (A) General (GLM), (B) Mixed Linear Model (MLM).

Three SNPs presented a significant association with loci-controlling *N. parvum* resistance (*P-value* <0.006) in the GLM and MLM analysis (Table 4, Figure 2). SNPs were identified on chromosomes 2, 9 and 12 of the mango genome, with R^2 ranging from 8.0% to 11.2% (Table 4).

The SNPs Mi_0096 and Mango_rep_c1316 presented an association with resistance loci to *L. theobromae*, both in the 'Cochran-Armitage trend' genotypic test and in the GLM and MLM analyses (Tables 1 and 3), thus making them strong candidates for further analysis. The SNPs Mango_rep_c9407 and Mango_rep_c8984 presented an association with resistance loci to *N. parvum* both in the allelic and genotypic analyses, as well as in the GLM and MLM analyses (Table 2 and 4), thus making them strong candidates for additional analysis.

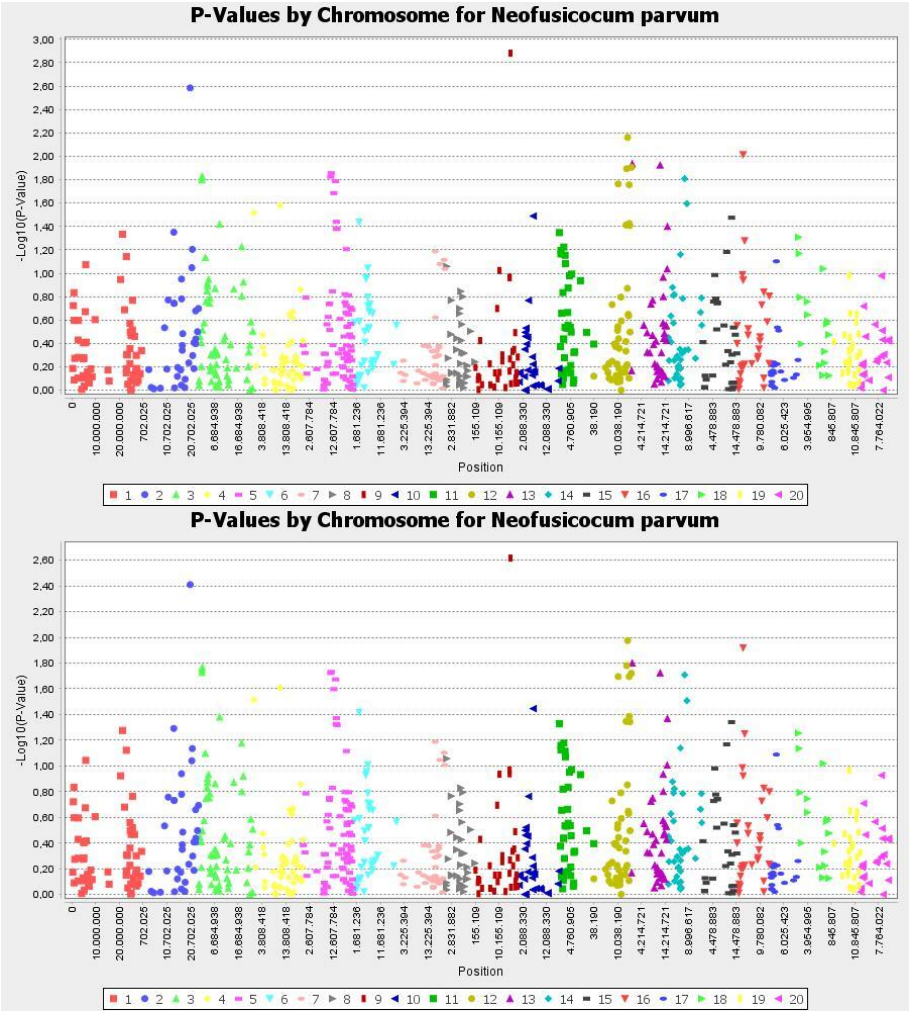

Figure 2. Manhattan plot for SNPs with associated resistance to *Neofusicocum parvum* in a 'Haden' × 'Tommy Atkins' mango pseudo-F_2 population: (A) General (GLM), (B) Mixed Linear Model (MLM).

Table 4. SNPs with a significant association* (*P-value*<0.02) and coefficient of determination (R^2) by the General (GLM) and Mixed Linear Model (MLM) to *Neofusicocum parvum* resistance loci in a 'Haden' × 'Tommy Atkins' mango pseudo-F_2 population.

SNP	Chromosome	Position (bp)	*P-value*	R^2
GLM				
Mango_rep_c8984	9	15016915	0.001318	0.112434
Mango_rep_c9407	2	20496307	0.002605	0.094316
Mango_rep_c7620	12	14293252	0.006896	0.080037
MLM				
Mango_rep_c8984	9	15016915	0.002425	0.112434
Mango_rep_c9407	2	20496307	0.003893	0.094316
Mango_rep_c7620	12	14293252	0.010592	0.080037

*Percentage data for symptoms of *N. parvum* transformed to arcsine.

DISCUSSION

Mango genetic studies and the development of new cultivars are hampered by the long juvenile period, by polyembryony in Southeast Asia accessions, cultivar incompatibility, plant size, and the use of small populations (Bally et al., 2009). These disadvantages are partially compensated by the vegetative multiplication of plants with commercial characteristics; if a superior plant is identified, its clonal maintenance is possible forever.

With the release and public availability of mango genomic sequencing (Wang et al., 2020) and the development of SNPs, still in small numbers, it became possible to carry out studies to identify alleles and/or chromosomal regions associated with many traits of this tree. The final goal is to apply marker-assisted selection to release new mango cultivars with desirable and attractive traits and with resistance to many mango diseases. Here we report a pioneering GWAS applied to key fungal diseases affecting commercial orchards in Northeast Brazil, the most important growing area in the country.

Allelic and genotypic analyses for *L. theobromae* indicate two regions on chromosome 12, one at position 10604128 bp (Mi_0096) and another at position 14675191 bp (Mango_rep_c59614), two other regions on chromosome 1 at positions 24576970 bp (Mango_rep_c1390) and 2534890 (Mi_0110), and a region at position 8818242 bp (Mango_rep_c5591) on chromosome 16. In the GLM and MLM analyses for this same fungus, two additional regions were identified: one on chromosome 11, position 10604128 bp (SSKP031C1_A370G) and another on chromosome 8, position 9349041 bp (Mi_0278). Taken together, genotypic, allelic, GLM and MLM analyses indicate two regions, one in chromosome 12 (Mi_0096) and another in chromosome 1 (Mango_rep_c1316) with a strong association to *L. theobromae* resistance loci. These two regions account for 20% of the total variation of resistance to this disease. If the other regions of chromosomes 11 and 8 are added, the regions identified in the present study explain 36% of the total variation of this disease.

For *N. parvum*, the allelic and genotypic analyses indicate two regions on chromosome 4 at positions 117199651/17245187 bp, two on chromosome 11, at positions 869763/896127 bp and three at chromosomes 2, 1 and 9, at positions 20496307, 22890486 and 15016915 bp, respectively. The GLM and MLM analyses indicate three regions on chromosomes 9, 2 and 12 at positions 15016915, 20496307 and 14293252 bp, respectively. Taken together, genotypic, allelic, GLM and MLM analyses indicate two regions on chromosomes 2 (Mango_rep_c9407) and 9 (Mango_rep_c8984) respectively, with a strong association to *N. parvum* resistance loci. These two regions explain 20.6% of total resistance to this fungus, which could be 28.7% of the total variation of this disease if one region of chromosome 12 (Mango_rep_c7620) is added.

A considerable proportion of the total variation of resistance to both fungi is still missing, 64% and 71% for *L. theobromae* and *N. parvum*, respectively, probably due to low genome coverage. Many of the SNPs in the present study were selected from thousands of RNA transcripts (Kuhn et al., 2016), some of which may be coding regions involved in the resistance of these two fungi. Saturation of the regions identified in the present study with additional SNPs will make it possible to develop markers with high efficiency for marker-assisted selection in mango and provide insight into other systems in which such pathogens are important. Resende et al. (2018) report missing heritability ($h^2=R^2$) up to ~64% for

three quantitative traits of *Phaseolus vulgaris*, probably due to LD between markers and genes and/or rare allele variants not sampled. Nonetheless, the results of the present study are in accordance with simple inheritance reported by Coelho et al. (2018) applying classical parameter estimation, when studying both fungi in three mango crosses, including the same 'Haden' × 'Tommy Atkins' pseudo-F_2 population.

For qualitative analysis, presence × absence of symptoms and allelic and genotypic methods have different assumptions, with Fisher's test analysing allele as the sampling unit, in Hardy-Weinberg equilibrium (HWE), while the Cochran-Armitage trend has the individual as a sampling unit, without the need for HWE, but in general, the qualitative analyses were consistent, presenting the same SNPs. The GLM and MLM analyses presented the same results, without impact of the principal components or IBD matrices, since the pseudo-F_2 populations are structured as one, without the formation of clusters, as occurs in accessions or populations from different origins.

Linkage map development in the Haden × Tommy Atkins mango pseudo-F_2 population resulted in up to 23 groups (data not shown), (*M. indica* n = 20), hindering traditional quantitative trait loci (QTL) analyses and comparisons with GWAS estimates in the present study. Zuiderveen et al. (2016) report independent analyses of GWAS and QTL to confirm regions controlling resistance to *Colletotrichum lindemuthianum* in *Phaseolus vulgaris* L.

According to Nagel et al. (2019), *N. parvum* and *L. theobromae*, family Botryosphaeriaceae, are well-known pathogens of economically important, commercially propagated plant crops, and the present study could support chromosomal mapping of a broad range of trees, including grapevine, pome and stone fruits, *Eucalyptus* spp., *Pinus* spp., cocoa and coconut. Shen et al. (2019) provide an example of rapid candidate gene mapping for resistance/susceptibility to four isolates of *Botryosphaeria dothidea* (Botryosphaeriaceae complex) in apple trees.

Analyses of resistant and susceptible plants, coded for 2 and 1, respectively, are a simplification of a complex plant-host system, but they enable the identification of chromosomal regions for both diseases, as proven when regions were also significant in GLM and MLM models, applied to the quantitative trait. Thus, the SNP regions Mi_0096 (chromosome 12) and Mango_rep_c1316 (chromosomes 1) for *L. theobromae* and the regions of the SNPs Mango_rep_c9407 (chromosomes 2) and (Mango_rep_c8984 (chromosomes 9) for *N. parvum* are strong candidates for saturation with a greater number of SNPs or further data mining in the mango genome.

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CONFLICTS OF INTEREST

The authors declare no conflict of interest.

REFERENCES

Alves EOS, Lima Neto FP, Santos CAF, Ribeiro ICNS, et al. (2016). Genetic diversity of mango accessions (*Mangifera indica*) using new microsatellite markers and morphological descriptors. *Aust. J. Crop Sci.* 10: 1281-1287.

Bally IS, Lu P and Johnson PR (2009). Mango Breeding. In: Breeding Plantation Tree Crops: Tropical Species (Jain, M and Priyadarshan, PM, eds). Springer New York, New York. Pp. 51-82.

Bradbury PJ, Zhang Z, Kroon DE, Casstevens TM, Ramdoss Y, et al. (2007). TASSEL: software for association mapping of complex traits in diverse samples. *Bioinformatics*. 23: 2633-2635.

Coelho WCP, Santos CAF and Batista DDC (2018). Inheritance of resistance to dieback disease in mango (*Mangifera indica*). *Aust. J. Crop Sci.* 12: 467-471.

Coelho WCP, Santos CAF and Batista DDC (2020). Variability of mango accessions resistance to dieback disease caused by *Lasiodiplodia theobromae* and *Neofusicoccum parvum*. *Amaz. J. Plant Res*. 4: 462-468.

Dautt-Castro M, López-Virgen AG, Ochoa-Leyva A, Contreras-Vergara CA, et al. (2019). Genome-Wide Identification of Mango (*Mangifera indica* L.) Polygalacturonases: Expression Analysis of Family Members and Total Enzyme Activity During Fruit Ripening. *Front. Plant Sci*. 10: 969.

Dillon N, Innes D, Bally I, Wright C, et al. (2014). Expressed Sequence Tag-Simple Sequence Repeat (EST-SSR) Marker Resources for Diversity Analysis of Mango (*Mangifera indica* L.). *Diversity*. 6: 72-87.

Duval MF, Bunel J, Sitbon C and Risterucci AM (2005). Development of microsatellite markers for mango (*Mangifera indica* L.). *Mol. Ecol. Notes*. 5: 824-826.

FAO - Food and Agriculture Organization of the United Nations (2020). FAOSTAT Food and Agriculture data. FAO, Rome. Accessed at [https://www.fao.org/faostat/en/#data/QCL]. Accessed 15 February 2023.

Galán Saúco V (2017). Trends in world mango production and marketing. *Acta Hortic*. 1183: 351-364.

Harper AL, Mckinney LV, Nielsen LR, Havlickova L, et al. (2016). Molecular markers for tolerance of European ash (*Fraxinus excelsior*) to dieback disease identified using Associative Transcriptomics. *Sci. Rep*. 6: 19335.

Honsho C, Nishiyama K, Eiadthong W and Yonemori K (2005). Isolation and characterization of new microsatellite markers in mango (*Mangifera indica*). *Mol. Ecol. Notes*. 5: 152-154.

Kuhn DN, Bally ISE, Dillon NL, Innes D et al. (2017). Genetic Map of Mango: A Tool for Mango Breeding. *Front. Plant Sci*. 8: 577.

Kuhn DN, Dillon N, Innes D, Wu L-S et al. (2016). Development of single nucleotide polymorphism (SNP) markers from the mango (*Mangifera indica*) transcriptome for mapping and estimation of genetic diversity. *Acta Hortic*. 1111: 315–322.

Masibo M and He Q (2009). Mango Bioactive Compounds and Related Nutraceutical Properties - A Review. *Food Rev. Int*. 25: 346-370.

Mukherjee SK and Litz RE (2009). Introduction: botany and Importance. In: The mango: botany, production and uses: (Litz RE, ed.). CAB International, Wallingford, UK. Pp. 1-18.

Nagel JH, Cruywagen EM, Machua J, Wingfield MJ, et al. (2020). Highly transferable microsatellite markers for the genera *Lasiodiplodia* and *Neofusicoccum*. *Fungal Ecol*. 44: 100903.

Purcell S, Neale B, Todd-Brown K, Thomas L, et al. (2007). PLINK: A Tool Set for Whole-Genome Association and Population-Based Linkage Analyses. *Am. J. Hum. Genet*. 81: 559-575.

Ravishankar KV, Mani BH-R, Anand L and Dinesh MR (2011). Development of new microsatellite markers from Mango (*Mangifera indica*) and cross-species amplification. *Am. J. Bot*. 98: e96-99.

Resende RT, Resende MDVD, Azevedo CF, Silva FFE, et al. (2018). Genome-Wide Association and Regional Heritability Mapping of Plant Architecture, Lodging and Productivity in *Phaseolus vulgaris*. *G3 (Bethesda)* 8: 2841-2854.

Shen F, Huang Z, Zhang B, Wang Y, et al. (2019). Mapping gene markers for apple fruit ring rot disease resistance using a multi-omics approach. *Genes Genomes Genet*. 9: 1663-1678.

Ribeiro IDS, Lima Neto FP and Santos CAF (2012). Allelic database and accession divergence of a Brazilian mango collection based on microsatellite markers. *Genet. Mol. Res*. 11: 4564-4574.

Schnell RJ, Olano CT, Quintanilla WE and Meerow AW (2005). Isolation and characterization of 15 microsatellite loci from mango (*Mangifera indica* L.) and cross-species amplification in closely related taxa. *Mol. Ecol. Notes* 5: 625-627.

Viruel MA, Escribano P, Barbieri M, Ferri M, et al. (2005). Fingerprinting, embryo type and geographic differentiation in mango (*Mangifera indica* L., Anacardiaceae) with microsatellites. *Mol. Breeding* 15: 383-393.

Wang P, Luo Y, Huang J, Gao S, et al. (2020). The genome evolution and domestication of tropical fruit mango. *Genome Biol*. 21: 60.

Zuiderveen GH, Padder BA, Kamfwa K, Song Q, et al. (2016). Genome-Wide Association Study of Anthracnose Resistance in Andean Beans (*Phaseolus vulgaris*). *PLoS One* 11: e0156391.