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Genome-wide association study for adult plant resistance to yellow rust in spring bread wheat (*Triticum aestivum* L.)

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Abstract Yellow rust, caused by *Puccinia striiformis* f. sp. *tritici*, is a common and serious fungal disease of wheat (*Triticum aestivum* L.) all over the world and particularly in the Central and West Asia and North Africa region. To identify effective yellow rust resistance loci, genome-wide association study (GWAS) was performed using 196 bread wheat genotypes based on 10,477 single nucleotide polymorphisms markers. Adult plants were evaluated under field conditions for resistance to yellow rust for 2 years (2014–2015) at ICARDA station, Marchouch, Morocco. Out of the 196 genotypes, 85 genotypes (43.37%) were resistant, 22 genotypes (11.22%) were moderately resistant, 13 genotypes (6.63%) were moderately susceptible, 48 genotypes (24.49%) were moderately susceptible to susceptible and 28 genotypes (14.29%) were susceptible to *Puccinia striiformis* f. sp. *tritici*. GWAS using mixed linear model identified 23 markers on chromosomes 2A, 2B, 2D and 7B significantly associated with adult plant resistance at false discovery rate (FDR-adjusted $P \leq 0.05$). Of which, three markers were within 10 wheat functional genes involved in several plant disease resistance and defense mechanism. Five of the reported functional genes are annotated as disease resistance proteins with nucleotide-binding site leucine repeat domains. BLAST analysis confirmed that *YrR61* and *Yr17* were mostly the candidate genes linked to the marker *Tdurum_contig29983_490* on chromosome 2A. Moreover, markers identified on chromosome 7B and *Kukri_c12648_434* on 2D were not mapped within any of previously reported gene/QTL region hence, representing novel resistance loci for *Pst* and needs to be confirmed using an allelism test.

Keywords Adult plant resistance · GWAS · *Puccinia striiformis* f. sp. *Tritici* · SNP · Stripe rust · Wheat

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s10681-021-02803-1.

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Introduction

Wheat is one of the most important food crops cultivated globally with annual production of more than 750 million tons in around 220 million hectares (FAO 2019). Numerous biotic and abiotic stresses, which are affecting individual regions in different/unique combinations, cause dramatic/significant yield losses each year. However, yellow (stripe) rust (YR), caused by *Puccinia striiformis* f. sp. *tritici* (*Pst*), is the most severe biotic stress globally, causing yield losses of about 5.5 million tones, or 979 million dollars, per year (Beddow et al. 2015).

Even though many new resistant varieties are released carry major stripe rust resistance genes, a cultivars' resistance can break down quickly when new virulent pathotypes evolve in the ongoing arms race between host plant and pathogen. In 2016, 88% of the global wheat production is assumed to be susceptible (Beddow et al. 2015). Consequentially, future yellow rust epidemic outbreaks remain a constant threat to yield stability. A prominent example is the breakdown in resistance conferred by *Yr27* that forced farmers in Ethiopia to spend over 3.2 million dollars in fungicides in 2010 to save their crop (Ali et al. 2017). Later on, in 2017, severe epidemics in major wheat growing area in the CWANA region like Algeria, Morocco, Syria and Turkey were reported (Morgounov et al. 2012).

In the course of 40 years, ICARDA has worked alongside communities and national research centers in the CWANA region to develop genotypes with new sources of resistance to YR to keep ahead of evolutionary potential outbreak of the pathogens. Pyramiding of diverse resistance genes in elite wheat cultivars is a successful strategy to achieve durability (Chen and Line 1995). Through the combination of multiple minor and major resistance genes, the likelihood of new pathotypes overcoming resistance is minimized (Pedersen and Leath 1988).

Over the last decade, new resistance genes and QTLs that could be employed in pyramiding have been identified through classical genetics and breeding efforts. To date, 80 yellow rust resistance genes (Yr) have been permanently named. Of which, 67 have been temporary designated and hundreds of quantitative trait loci (QTL) have been reported and mapped (Wang and Chen 2017; Feng et al. 2018; Nsabiyera et al. 2018). Six QTL newly reported in Yao et al (2019) were mapped far from resistance regions previously identified. Among the formally designated stripe rust resistance genes, 55 confer seedling resistance and 24 genes are recounted as adult plant resistance (APR) genes. Combining of two or more of such resistant genes would provide durable resistance. However, gene pyramiding through conventional methods is often challenging and time-consuming.

Therefore, the identification of molecular markers closely associated with yellow rust resistance is a promising approach to accelerate the breeding process by discovering novel resistance genes and pyramiding multiple genes in a robust and simplified manner. With the advances in sequencing technologies, new genotyping methods allowed the development of high-throughput and low cost-effective marker systems. For instance, single nucleotide polymorphism (SNP) markers have been routinely used in plant breeding research for their availability and their abundance across the genome. Development of SNP marker panels enabled genome wide association studies (GWAS) to increase the chances of identifying genomic regions that underlay genes of interest. Association mapping has been successfully used in mapping agronomically important genes/QTL in bread wheat, such as grain quality traits (Tadesse et al. 2015), kernel size and milling quality (Breseghello and Sorrells 2006), disease resistance (Mourad et al. 2018; Riaz et al. 2018) major insect pest resistances (Ando et al. 2018) and many more.

The objectives of the study were (1) to access the extend of field resistance to stripe rust among the 196 bread wheat genotypes, (2) to determine the genetic structure within these wheat genotypes and (3) to identify closely associated markers with YR resistance using 10,477 polymorphic SNP markers over the 2 years.

Materials and methods

Germplasm development and phenotyping

In this study, a population of 196 spring bread wheat genotypes, from the International Center for Agricultural Research in Dry Areas (ICARDA) advanced yield trials, was used (Supplemental Table S1). This population was previously used to identify QTL linked to yield potential and heat stress tolerance in Sudan

and Egypt (Tadesse et al. 2019). The 196 genotypes were planted using a lattice design in two replications in 3 m^2 plot basis at Marchouch station, Morocco (33° 36′ N, 6° 43′ W, 394 masl) for two cropping seasons (2013/2014 and 2014/2015) under rainfed conditions to identify genotypes resistant to yellow rust. The trials management followed common local practices.

Yellow rust assessment

The 196 genotypes were evaluated for yellow rust under natural field infection at adult-plant stage. ZEMAMRA-8 and QADANFER-11 cultivars were used as susceptible checks. The responses for the major infection types were recorded according to Roelfs (1992). Disease severity as a percentage of covered areas was assessed following modified Cobb's scale (Peterson et al. 1948). Field responses were recorded 2–3 times and the final scoring at soft-dough stage was considered for the association mapping analysis. The data on disease severity and host reaction were combined to calculate the coefficient of infection (CI) following Pathan and Park (2006), by multiplying the severity value by a value of 0.2, 0.4, 0.6, 0.8, or 1.0 for host response ratings of resistant (R), moderately resistant (MR), intermediate (M), moderately susceptible (MS), or susceptible (S), respectively.

Statistical analysis

Analysis of variance (ANOVA) were performed across years using a linear mixed model to determine the effects of genotype, year and genotype × year interaction. Genotype and genotype × year were considered as (1) fixed to get the means and their standard errors and (2) as random to compute the genetic variance and hence the heritability. Heritability for coefficient of infection (CI) was calculated using the formula $H = \sigma^2 g/(\sigma^2 g + \sigma^2 e/ne + \sigma^2 e/ne\ nr)$, where $\sigma^2 g$ represented the genetic variance, $\sigma^2 e$ the error variance, ne was the number of environments (years) and nr the number of replicates.

Genotyping by SNP markers

Genomic DNA extraction of the 196 genotypes, genotyping, SNP calling are described in earlier paper (Tadesse et al. 2019). Monomorphic and SNP loci with > 10% missing values, vague SNP calling, and minor allele frequencies < 5% were excluded.

Population structure

The genetic structure of the 196 genotypes was assessed with 102 unlinked SNP markers distributed across the wheat genome with at least two loci on each wheat chromosome using the STRUCTURE software (Pritchard et al. 2000). This population was previously used by Tadesse et al. (2019) as mentioned above. Analysis of population structure, discriminant analysis of principal components (DAPC) performed using the 'adegenet' package 1.4-1 in Rstudio (Jombart et al. 2010) and Bayesian Information Criterion (BIC) using find.clusters function in R have shown all three subpopulations.

Linkage disequilibrium

TASSEL 5.0 (Bradbury et al. 2007) was used to estimate LD as squared allele frequency correlation estimates (R^2) and to measure the significance of R^2 at *P* values for each pair of loci on different chromosomes (interchromosomal LD) and within the same chromosome (intrachromosomal LD).

Association mapping

In the present study, stripe rust resistance data were used to identify SNPs associated with the resistance. The 2 years average phenotypic stripe rust coefficient of infection (YR CI value) were used separately for the association mapping analysis. The association of the SNP markers retained after quality filtering and stripe rust measurements was carried out using TASSEL 5.0 software (Bradbury et al. 2007). A mixed linear model (MLM) was performed taking into consideration both kinship (*K*) and population structure (*Q*) matrices as covariates. To eliminate linear dependence between columns of the *Q* matrix, the last column was removed prior to GWAS. Individuals with > 10% missing SNP calls and markers with > 10% missing and < 5% minor allele frequency (MAF) were eliminated. The Manhattan plots of GWAS were re-drawn using R library "CMplot" package in R 3.3.1.

Putative candidate gene identification

Putative candidate genes associated with the significant markers were identified on the basis of LD using the International Wheat Genome Sequencing Consortium (IWGSC) RefSeq v1.0 annotations (IWGSC 2018). Based on basic local alignment search tool (BLAST) in the server, nearby genes in the linkage regions of significant SNP-trait associations with putative functions were selected as candidates. The list of candidate genes was further narrowed down by extracting the nucleotide-binding and leucine-rich repeat (NLR) resistance genes annotated by NLR Annotators under loci/QTL intervals (Steuernagel et al. 2018).

Results

Response of genotypes to yellow rust

Phenotypic variation was observed for the infection types and level of YR severity for the 196 genotypes during the two cropping seasons. The final score ranged from 5R (resistant) to 100S (highly susceptible). Both the susceptible controls, ZEMAMRA-8 and QADANFER-11 were scored 100S in the first and second year. The reaction type for some of the genotypes changed between cropping seasons. The genotypes showed variable reaction responses from moderately resistant to susceptible and the severity level ranged from 10 to 100%.

Based on the coefficient of infection, we have classified different severity classes. Out of 196 genotypes tested for 2 years, 85 genotypes (43.37%) were resistant (exhibited resistance reaction response CI 0–20); 22 genotypes (11.22%) were moderately resistant (CI 20–30); 13 genotypes (6.63%) were moderately susceptible (CI 30–40); 48 genotypes (24.49%) were moderately susceptible to susceptible (CI 40–60) and 28 genotypes (14.29%) were susceptible to *Puccinia striiformis* f. sp. *tritici* (CI 60–100) (Fig. 1). The resistant lines based on CI scores for yellow rust dropped down over the 2 years (Fig. 2). And the CI recorded in the two cropping seasons were significantly correlated with a coefficient of 0.39 ($P < 0.001$) (Fig. 3). Field response scores across years were normally distributed with 13.85% CV and heritability of 0.65% (Table 1). The analysis of

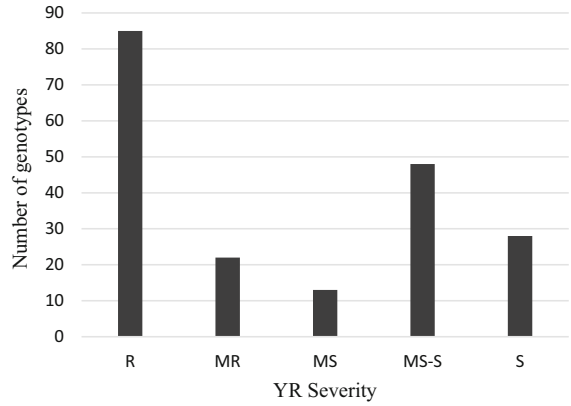

Fig. 1 Average response of 196 spring bread wheat genotypes to yellow rust at Marchouch station (Morocco) during 2013–2014 and 2014–2015 cropping seasons

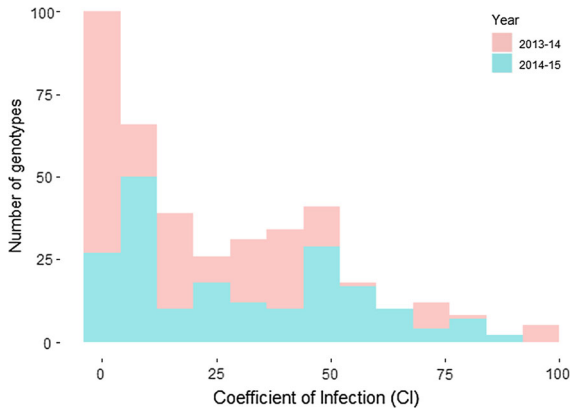

Fig. 2 Coefficient of infection of the 196 spring wheat genotypes to yellow rust at Marchouch station (Morocco) during 2013–2014 and 2014–2015 cropping seasons. (Color figure online)

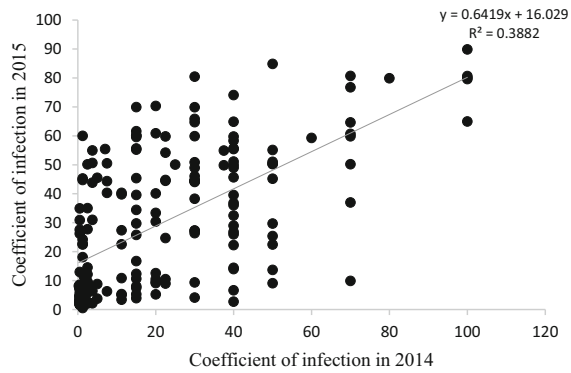

Fig. 3 Scatter plot of the coefficient of infection over the two cropping seasons

Table 1 Analysis of variance of stripe rust resistance in the studied 196 wheat genotypes

	df	Mean square	F value	Pr
Year	2	1647.2	768.5	2.31E−136**
Genotype	195	987.6	2.89	1.71E−19**
Year × rep	2	200.8	0.57	0.563
Year × genotype	194	452.4	1.31	0.014*
Error	356	363		
C.V	13.85			
R	0.83			
Heritability	0.65			

C.V coefficient of variation, *R* variance

**,*Significant at 0.001 and 0.05 probability level, respectively

variance for CI scores revealed significant effect of the 2 years and genotypes as well as significant genotypes × years interaction in response to stripe rust infection.

There was significant difference in grain yield performance among the genotypes and the average yield ranged from 1.801 to 7.275 t/ha. Those with maximum relative disease severity (QADANFER-11) produced minimum grain yield (2.34 t/ha). Whereas, the genotypes with resistant or moderate resistant (CI 0–30) showed higher yield productivity (Fig. 4). Among the genotypes in the resistant group, 20 elite lines were identified combining YR resistance and high yield potential (Table 2). These elite genotypes have been part of the international nurseries distributed to the national partners for release or further use as parents in the wheat breeding programs.

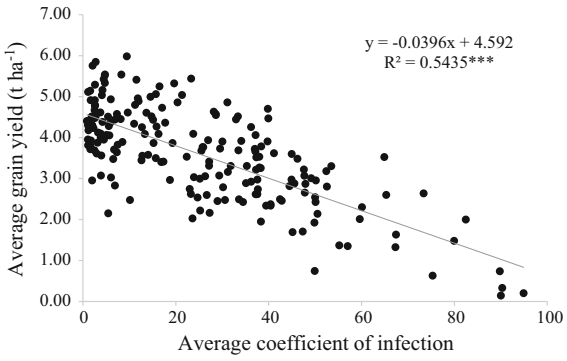

Fig. 4 Scatter plot of average yield performance of the 196 genotypes of spring bread wheat and average coefficient of infection during the 2013–2014 and 2014–2015 cropping seasons

Population structure

The population structure of the 196 genotypes showed 3 sub-populations. A detailed analysis was described in earlier publication (Tadesse et al. 2019). The top 23 high yielding genotypes were distributed across the three subpopulations.

Linkage disequilibrium

A total of 10,477 out of 12,725 SNPs, retained after removal of those with MAF $< 5\%$ and missing values over 10%, were used to identify markers associated to stripe rust response using Q + K MLM method. A total of 3937 SNP markers were located on the A genome, whereas 5317 and 1223 polymorphic markers were distributed on the B and D genomes, respectively. The poor-quality markers with minimum allele frequency less than 5% were excluded from further analysis. Chromosomes with the largest number of markers were 2B with 1015 markers followed by 5B with 894 markers. Chromosomes 3D and 4D showed the least number of markers, 92 and 49, respectively.

The LDs for locus pairs within the same chromosomes and between chromosomes were calculated separately. For the whole wheat genome, 58.8% of the pair-wise LD comparisons were significant at $P < 0.01$, with the mean R^2 of 0.36. The A genome had 35.78% significant marker pairs with an average R^2 of 0.3, the B genome had 54% significant marker pairs and an average of R^2 0.35 while the D genome had 9.22% significant marker pairs and an average R^2 of 0.23. Chromosomes 2B (10.8%) has the highest percentage of significant marker pairs of LD R^2, whereas chromosomes 4D (0.25%) showed the lowest percentage.

The scatter plots of LD (R^2) as a function of the intermarker distance (Mbp) within the same chromosome for all genotypes indicated a clear LD decay with genetic distance (Fig. 5). LDs with $R^2 > 0.23$ extended to distances up to 6Mbp suggesting that the mapping resolution using these genotypes would generally be well below the 6Mbp.

Table 2 Mean yellow rust response and grain yield performance of elite wheat genotypes at Marchouch, during 2014 and 2015 seasons

No	Name/pedigree	Selection history	YR response	Mean yield (t/ha)
1	SERI.1B//KAUZ/HEVO/3/AMAD/4/ATTILA//PSN/BOW/3/ ATTILA/5/KAUZ'S'/SHUHA-15	ICW06-50236-13AP-0AP-0AP-02 SD	10MR	7.275
2	SERI.1B//KAUZ/HEVO/3/AMAD/4/ATTILA//PSN/BOW/3/ ATTILA/5/KAUZ'S'/SHUHA-15	ICW06-50236-11AP-0AP-0AP-1 SD	10MR	6.657
3	SERI.1B//KAUZ/HEVO/3/AMAD/4/ATTILA//PSN/BOW/3/ ATTILA/5/KAUZ'S'/SHUHA-15	ICW06-50236-12AP-0AP-0AP-03 SD	15 M	6.524
4	CHILERO-1/4/VEE'S'/3/HORK/4MH//KAL-BB/5/PFAU/MILAN	ICW05-0634-11AP-0AP-0AP-2AP-0AP	10MR	6.189
5	KAUZ//ALTAR 84/AOS/3/MILAN/DUCULA	AISBW05-0103-7AP-0AP-0AP-8AP-0AP	10MR	6.049
6	VEE/PJN//2*KAUZ/3/SHUHA-4/FOW-2	ICW06-00166-10AP-0AP-1 SD	10MR	5.961
7	OPATA/RAYON//KAUZ/3/2*MILAN/DUCULA	ICW06-50333-4AP-0AP-0AP-1 SD	10MR	5.853
8	SERI.1B//KAUZ/HEVO/3/AMAD/4/PFAU/MILAN	ICW06-00151-8AP-0AP-03 SD	10MR	5.709
9	OPATA/RAYON//KAUZ/3/2*MILAN/DUCULA	ICW06-50333-4AP-0AP-0AP-03 SD	10MR	5.653
10	P1.861/RDWG//DAJAJ-10/3/MILAN/PASTOR	ICW06-50250-11AP-0AP-0AP-05 SD	10MR	5.651
11	ATTILA 50Y//ATTILA/BCN/3/KAUZ//MON/CROW'S'/4/ MILAN/PASTOR	ICW06-50018-12AP-0AP-0AP-02 SD	10MR	5.644
12	ATTILA*2/PBW65//PFAU/MILAN	ICW05-0450-8AP-0AP-0AP-3AP-0SD	10MR	5.596
13	KAUZ//ALTAR 84/AOS/3/MILAN/DUCULA	AISBW05-0103-7AP-0AP-0AP-5AP-0AP	10MR	5.565
14	OPATA/RAYON//KAUZ/3/2*MILAN/DUCULA	ICW06-50333-2AP-0AP-0AP-04 SD	10MR	5.548
15	KAUZ//ALTAR 84/AOS/3/TNMU/MILAN/4/MILAN//PSN/BOW	ICW06-50377-5AP-0AP-0AP-04 SD	10MR	5.542
16	CROC1/AE.SQUARROSSA(205)//KAUZ/3/ATTILA/4/ LAG-1	ICW06-00907-4AP/0KUL-0DZ/0AP-0DZ/ 0AP-1AP-0AP	10MR	5.501
17	KAUZ//ALTAR 84/AOS 3/KAUZ/3/SHUHA-4//NS732/HER/4/ QAFZAH-33	ICW06-50344-3AP-0AP-0AP-1 SD	10MR	5.397
18	ATTILA*2/PBW65//PFAU/MILAN	ICW05-0450-8AP-0AP-0AP-2AP-0SD	10MR	5.365
19	KAUZ//ALTAR 84/AOS 3/KAUZ/3/ATTILA 50Y//ATTILA/ BCN/4/PASTOR-6	ICW06-50246-7AP-0AP-0AP-1 SD	10MR	5.344
20	KAUZ//ALTAR 84/AOS/3/TNMU/MILAN/4/MILAN//PSN/BOW	ICW06-50377-5AP-0AP-0AP-02 SD	10MR	5.312
	ARREHANE		70S	3.905
P value				< 0.001
CV[a] (%)				9.6
SE[b] (kg)				0.576
LSD[c] at 5% (kg)				0.782

[a]Coefficient of variation

[b]Standard error of the mean

[c]Least significant difference

Association analysis of resistance to yellow rust

A total of 76 significant marker trait associations (MTAs) representing 9 chromosomes were identified to be significantly associated ($P < 0.001$) with stripe rust resistance at adult plant stage. These markers are located on 1A, 2A, 2B, 2D, 3B, 3D, 5A, 7B and 7D of which 25 markers were identified in 2014 and 51 markers in 2015 (Supplementary Table S2, Fig. 6). The big portion of the markers found associated to YR resistance was located on chromosomes 2A, 2B and 7B. The percentage of phenotypic variation explained by marker for the resistance to YR ranged from 22.27 to 42.35%. *Tdurum_contig29983_490* in 2A exhibited

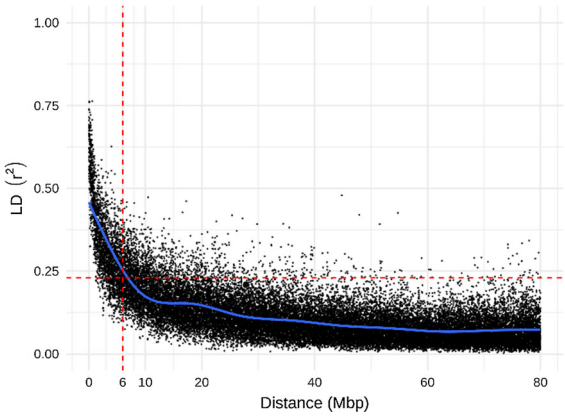

Fig. 5 Plot of pair-wise single-nucleotide polymorphism LD R^2 values as a function of intermarker map distance (Mbp). The blue curve represents the model fit to LD decay. The red line represents the 6 (Mbp) confidence interval for the quantitative trait loci regions in which LD $R^2 = 0.23$, beyond which LD is likely due to linkage. (Color figure online)

the highest level of contribution to YR resistance. Six markers, *Ku_c7740_879* (2B), *Excalibur_c45094_602* (2B), *wsnp_BE399688B_Ta_2_1* (2B), *Excalibur_c73922_55* (2B), *Excalibur_c73922_109* (2B) and *RAC875_c34939_86* (7B), were found common in both years. The marker *Ku_c7740_879* on chromosome 2B was present in 177 genotypes (91.7%), of which 137 genotypes (70.9%) showed moderately resistant to resistant responses to YR in the first year. However, only 64 (36.15%) genotypes were scored resistant to moderately resistant for the same marker. Out of the 76 significantly associated markers, 23 passed a threshold of FDR ≤ 0.05. *Ku_c7740_879* and *RAC875_c34939_86* were common in both years but significant at FDR ≤ 0.05 in 2014 and 2015, respectively. Thirteen of these markers on chromosomes 2A, 2B and 2D were mapped close to previously identified rust resistance genes and QTL and the rest are most likely novel markers for stripe rust resistance (Table 3).

Out of the 6 common significant MTAs found across 2 years, *Ku_c7740_879* and *RAC875_c34939_86* located on chromosomes 2B and 7B, respectively, expressed the highest frequency of favorable alleles for yellow rust resistance in the population. The results showed that wheat genotypes carrying the cytosine and adenine bases at the *Ku_c7740_879* and *RAC875_c34939_86* markers combination, respectively, significantly out-yielded the other bases combinations. The combination of the thymine and adenine bases shown to have low yield performance and that's linked to the low cumulative number of favorable alleles of the two significant markers (Fig. 7). We have investigated the relationship between the cumulative favorable alleles and response to stripe rust in every single genotype. Pearson correlation coefficient showed significant negative correlation between the number of favorable alleles ($r = -0.614$, P value < 0.01) with respect to stripe rust intensity score (Fig S1).

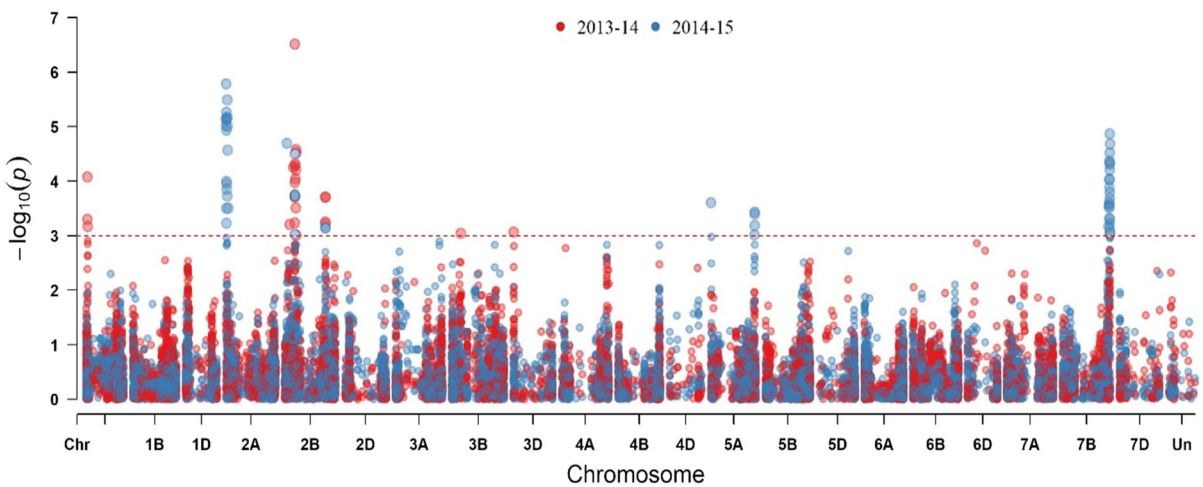

Fig. 6 Manhattan plot for stripe rust results obtained from genome-wide association mapping for 196 genotypes in Marchouch during 2014 and 2015 field experiments

Table 3 Chromosome, position, LOD, R^2, MAF and effect of the 23 significantly associated SNP markers with yellow rust (YR) resistance at FDR ≤ 0.05 in Marchouch in the 196 bread wheat genotypes

Marker	Chr	Pos (cM)	Physical position (bp)[a]	LOD[b]	R^2	MAF	Effect	Year[c]	Previously mapped Yr gene/QTL[d]
Tdurum_contig29983_490	2A	0	259186	5.76*	0.42	0.16	− 2.03	2015	*YrR61*, *Yr61*, *Yr56*, *Yr17*, *Yr2A.1PBL*, *QYr.tam-2AS_TAM 111*, *QYr.uga-2AS_26R61*
Kukri_c22599_114	2A	0	397564	5.12*	0.41	0.17	− 2.23	2015	*YrR61*, *Yr61*, *Yr56*, *Yr17*, *Yr2A.1PBL*, *QYr.tam-2AS_TAM 111*, *QYr.uga-2AS_26R61*
tplb0041b11_529	2A	0	532014	5.00*	0.41	0.16	− 0.90	2015	*YrR61*, *Yr61*, *Yr56*, *Yr17*, *Yr2A.1PBL*, *QYr.tam-2AS_TAM 111*, *QYr.uga-2AS_26R61*
Excalibur_c62893_629	2A	0	4213194	5.26*	0.41	0.17	0.68	2015	*YrR61*, *Yr61*, *Yr56*, *Yr17*, *Yr2A.1PBL*, *QYr.tam-2AS_TAM 111*, *QYr.uga-2AS_26R61*
RAC875_c829_1215	2A	8.86	17000797	5.14*	0.41	0.15	2.38	2015	*QYr.inra-2AL_CampRemy*
Ra_c26532_894	2A	8.86	21532691	4.68*	0.41	0.15	0.35	2015	*QYr.inra-2AL_CampRemy*
wsnp_Ku_c33374_42877546	2A	11.14	4789997	4.93*	0.41	0.15	− 2.77	2015	*Yr2A.2PBL*, *QYrst.orr-2AS_Stephens*, *QYr.ucw-2A_PI610750*
BS00093990_51	2A	12.31	24278839	4.56*	0.40	0.15	− 12.70	2015	*Yr2A.2PBL*, *QYrst.orr-2AS_Stephens*, *QYr.ucw-2A_PI610750*
TA003766-0683	2A	–	24410117	4.99*	0.41	0.16	− 1.99	2015	*Yr2A.2PBL*, QYrst.orr-2*AS_Stephens*, *QYr.ucw-2A_PI610750*
Ku_c7740_879	2B	78.5	160217138	6.49**	0.30	0.09	0.59	2014[c]	*QYr.cim-2BS_Francolin*
Tdurum_contig11802_864	2D	0	2478926	5.10*	0.41	0.18	0.11	2015	*QYr.caas-2DS_Libellula*
BS00067382_51	2D	0	4329746	5.16*	0.41	0.17	2.02	2015	*QYr.caas-2DS_Libellula*
Excalibur_c25599_358	2D	0	8294994	3.99*	0.39	0.20	− 0.26	2015	*QYr.caas-2DS_Libellula*
Kukri_c12648_434	2D	8.86	17955919	5.48*	0.42	0.16	− 5.98	2015	–
RAC875_c34939_467	7B	159	741576947	4.31*	0.40	0.33	0.11	2015	–
RAC875_c5744_115	7B	171.25	740055207	4.02*	0.39	0.46	0.88	2015	–
Excalibur_c81824_411	7B	172.24	739931797	4.03*	0.39	0.30	1.13	2015	–
BobWhite_c43557_103	7B	172.24	741577054	4.33*	0.40	0.32	1.25	2015	–
RAC875_c34939_86	7B	172.24	741577327	4.87*	0.41	0.39	− 0.96	2015[c]	–
tplb0060b03_432	7B	181.24	742465197	4.51*	0.40	0.46	4.80	2015	–
RAC875_c38693_319	7B	191.24	740056879	4.36*	0.40	0.46	− 1.51	2015	–
RAC875_c8752_705	7B	191.24	741575116	4.20*	0.40	0.32	− 4.39	2015	–
RAC875_rep_c108382_824	7B	191.24	750083809	4.68*	0.41	0.33	− 3.39	2015	–

LOD − Log10(P), *MAF* major allele frequency

[a]Physical position based on the IWGSC RefSeq. 1.0

[b]Significance. Experiment-wise (Benjamini–Hochberg FDR adjusted): *FDR (q) ≤ 0.05, **FDR (q) ≤ 0.01

[c]Marker appeared in both years but passed FDR ≤ 0.05 in 1 year only

[d]References are given in the text

Candidate genes and function annotations

The genetic frame of the polymorphic SNP markers was constructed using BLAST alignment of each allele sequence to the reference Chinese Spring genome RefSeq v1.0. Based on NLR-Annotators, we aligned sequences of flanking markers to Chinese spring, defined their physical interval and determined the NLR-associated loci (Table 4). Ten candidate genes were annotated for functional proteins involved in plant disease resistance and defense mechanism. Five NLR loci (*NLR43*, *NLR143*, *NLR147*, *NLR146*

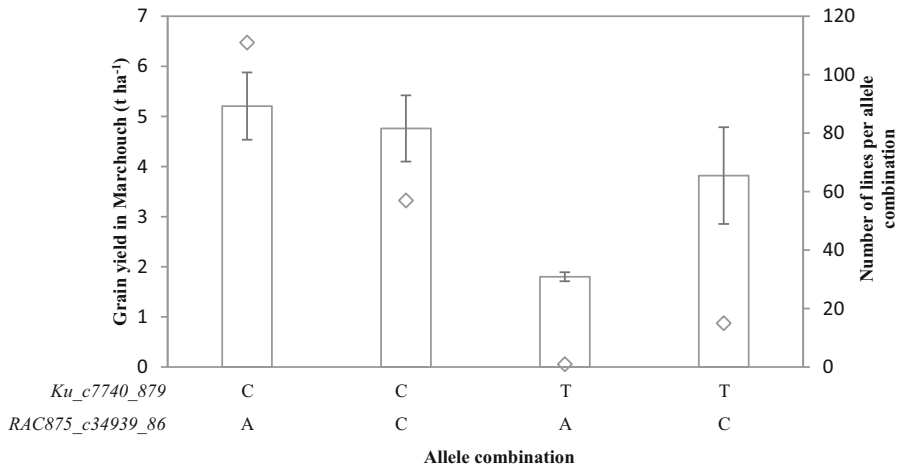

Fig. 7 Average grain yield (bars) from the two field experiments conducted in 2014 and 2015 at Marchouch, Morocco, with 196 bread wheat genotypes having four base–pair combinations of two independent single nucleotide polymorphism markers associated with yellow rust resistance (logarithm of odds > 3.0) common in both years: *Ku_c7740_879* and *RAC875_c34939_86*. The number of lines per base–pair combination is indicated in open quadrangle (open diamond). Confidence level at $\alpha = 0.05$ is also shown for each combination

Table 4 Identification of genes and NLR of yellow rust resistance underlying QTLs ± LD

Chr	Marker	LOD	Left flanking position	Right flanking position	Number of genes	Published YR in region	NLR annotator
2A	Tdurum_contig29983_490	5.77	0	6259186	153	*YrR61/BV211615, YR17*	
2B	Ku_c7740_879	6.14	154217138	166217138	112	*QYr.cim-2BS_Francolin*	*NLR43*; *NLR143*
7B	RAC875_c34939_86	3.78	735577327	747577327	155	*?*	*NLR80*; *NLR146*; *NLR147*

and *NLR80*), corresponding to one or more genes in the RefSeq v1.0, were detected. The putative proteins/enzymes related to these genes include serine/arginine (SR) domain, StAR-related lipid transfer domain, Leucine-rich repeat (LRR) domains, TNF receptor-associated factor family protein, glycosyltransferase 1 family and Chromodomain-helicase-DNA-binding family protein. Gene function, genome and protein sequence of the putative NLRs identified are provided in Supplementary Table S3 and File S1.

Discussion

As yellow rust becomes the most important disease across the CWANA region, ICARDA's germplasm development approach aims to combine resistance to yellow rust with high yield and wide adaptation. Therefore, understanding of the genetic basis of yellow rust resistance could facilitate the transfer of existing or novel resistance genes into high yielding and widely adapted bread wheat lines. In Morocco, where yellow rust is the major disease for bread wheat production, yield losses are estimated at 20% in 2009 with the variety Achtar severely affected (Ezzahiri et al. 2009). However, under conducive environment, the losses can be even worse up to 100% in susceptible varieties if the infection occurs in early growth stages (Yuan et al. 2018). Aggressive races with virulence to the most common genes (*Yr9*, *Yr27* and others) are widely spread in many countries where susceptible varieties are still grown at large scale as it is the case of variety Achtar, one of the most widely grown in Morocco. Race dynamics and virulence evolution of yellow rust are responsible for the resistance outbreaks. Consequently, continuous screening in

hotspots areas is crucial to identify and integrate new sources of resistance genes that could be valuable for yellow rust resistance breeding progress. Hence, we carried out this study to determine the resistance level of diverse ICARDA materials as compared with susceptible checks against Moroccan pathotypes of yellow rust and identify the associated SNP markers with disease resistance.

Considerable variation was observed for reaction response to stripe rust across the years. Among the 196 genotypes, 147 and 96 genotypes showed strong to moderate resistance based on the disease intensity in the first and second year, respectively. More disease severity was observed in the second year; The number of the resistant genotypes in the second year was less than what was scored in the first year and this might be attributed to the conducive conditions for infection that causes greater disease pressure, variation of environmental conditions that favor the level of disease expressions and/or a new race emergence.

The Warrior (*PstS7*) race was confirmed in 2013 in Morocco and continue to be spread widely in the Northern African countries, Europe and East Asia (Tadesse et al. 2014; Hovmøller et al. 2016). *PstS10* known as Warrior(–) (virulence pattern: [1, 2, 3, 4, –, 6, 7, –, 9, –, –, 17, –, 25, –, 32, Sp, AvS, –]) was also reported in Algeria and Spain in 2014 and perhaps in Morocco as well but no data is available to confirm (Hovmøller et al. 2018). In 2015 season, the virulence in Morocco again was unknown. However, the next season, in 2016, a sampling was done systematically and a new distinct race (*PstS14*, virulence pattern: [–, 2, 3, –, –, 6, 7, 8, 9, –, –, 17, –, 25, –, 32, Sp, AvS, –]) was detected in Morocco (Hovmøller et al. 2018). So, it is possible that the race change occurred in 2015. In fact, giving that the resistance rating dropped from 147 to 96 resistant genotypes in 2015 might indicate that *PstS10* and *PstS14* (or may be other unknown races) possibly contributed to variation in the reaction type and the resistance.

Appearance of new virulent races is leading to the breakdown of the resistance to stripe rust. Almost all the commercial bread wheat cultivars grown in Morocco became highly susceptible to stripe rust (Ramdani et al. 2018). The cultivar Arrihane, which used to have moderate resistance to stripe rust in the past few years, is becoming highly susceptible and scored 70-90S (personnel score). Similarly, in the current study, we observed changes in the intensity and virulence of the yellow rust and some of the resistance genes, perhaps, no longer provide effective resistance due to emergence of the *PstS14*. The genotype SERI.1B*2/3/KAUZ*2/BOW//KAUZ/4/KAUZ/GYS//KAUZ/5/MUNIA/ALTAR84//MILAN showed a 5R response to yellow rust in the first year but changed drastically to 60S in the second year. Moreover, yellow rust infections increased for all the checks in the second years. The check PASTOR-2, which was moderately susceptible (20MS) in the first year, scored 70S in the next year. Therefore, the surveillance through race phenotyping and genotyping in hotspot regions is crucial for an early warning and anticipatory crop management and breeding strategies.

The host plant resistance is generally the most economically effective strategy to manage stripe rust in the developing countries. Thus, the deployment of resistance genes in the development of effectively resistant cultivars is important for the ongoing control against stripe rust in wheat (Todorovska et al. 2009). Up to date, ICARDA continues to deliver germplasm that possesses adult plant resistance to stripe rust combining several genes with minor effects, mostly *Yr18*, *Yr29* and *Yr30* (Tadesse et al. 2014). SNINA cultivar, of ICARDA origin, has been recently released in Morocco and demonstrated its broad resistance to YR. In the current study, we applied genome wide association mapping approach using 10,477 polymorphic SNP markers. We run the GWAS using the mean values of every year separately because of the significant genotypes × years interaction. This may indicate that the pathogenicity during the 2 years experiment was different. A total of 76 markers at different genomic regions were detected significatively associated with yellow rust resistance of which 23 were at experiment-wise FDR ≤ 0.05 on chromosomes 2A, 2B, 2D and 7B. We have observed that the markers on chromosome 1A that confer yellow rust resistance in the first year are no longer present in the second year. The LOD score in *Ku_c7740_879* reduced from 6.49 to 4.49 in the first and the second year, respectively indicating that possible new source of virulence has contributed to the variation in the resistance level.

In order to determine the genes/QTL associated with the YR resistance previously reported, we compared their physical location by BLAST analysis of the International Wheat Genome Sequencing

Consortium (IWGSC) RefSeq v1.0 genome. Identified QTL were further investigated by extracting all genes annotated in the region of the highest associated marker ± LD of 6 Mbp. The list of candidate genes was further narrowed down by extracting the NLR resistance genes annotated by NLR Annotators within the loci/QTL. Results of the GWAS are discussed in detail focusing only on markers at experiment-wise FDR ≤ 0.05.

Based on the information of genes and QTL previously reported, we compared the locations of the significant markers found in the present study. Seven QTL were identified on the short arm of the chromosome 2A. Of these, *QYr.tam-2AS_TAM 111* and *QYr.uga-2AS_26R61* have been previously mapped within the flanking positions of the markers *Tdurum_contig29983_490*, *Kukri_c22599_114*, *tplb0041b11_529* and *Excalibur_c62893_629* markers (Basnet et al. 2014; Hao et al. 2011). Ledesma-Ramírez et al. (2019) has reported a new *QTL Yr2A.1PBL* that overlapped with the same markers where *YrR61*, *Yr69*, *Yr56* and *Yr17* are located. Markers *RAC875_c829_1215* and *Ra_c26532_894* were found in the region of previously identified *QTL QYr.inra-2AL_CampRemy* (Mallard et al. 2005). Three other QTL *QYrst.orr-2AS_Stephens*, *QYr.ucw-2A_PI610750* and *Yr2A.2PBL* were identified by the SNPs *wsnp_Ku_c33374_42877546*, *BS00093990_51* and *TA003766-0683* on chromosome 2A (Lowe et al. 2011; Vazquez et al. 2012; Ledesma-Ramírez et al. 2019) showed effectiveness at adult-plant stage to *Pst*. BLAST analysis and anchoring the flanking markers of stripe rust resistance locus on the reference genomes allowed the identification of two candidate genes *TraesCS2A01G009400* and *TraesCS2A01G010200* with arginine/serine-rich splicing factor and StAR-related lipid transfer proteins, respectively (IWGSC 2018). *YrR61* and *Yr17*, which overlapped with *TraesCS2A01G009400* and *TraesCS2A01G010200*, respectively, were mostly the candidate genes tagged by *Tdurum_contig29983_490*.

On chromosome 2B, 112 genes were mapped so far. Among previously published Yr genes/QTL, *QYr.cim-2BS_Francolin* was located within the flanking region of the marker *Ku_c7740_879* on the short arm of chromosome 2B (Lan et al. 2014). *QYr.cim-2BS* was designed as *YrF* gene that conferred intermediate seedling reactions and moderate resistance at the adult plant stage in both Mexican and Chinese environments (Yuan et al. 2018). BLAST analysis of *SNP Ku_c7740_879* identified one gene (*TraesCS2B01G190200*) that serves as TNF receptor-associated factor (TRAF) (IWGSC 2018). TRAF proteins are adaptor proteins for a wide variety of receptors that are involved in regulating cell death and survival and cellular responses to stress (Bradley and Pober 2001). Moreover, Steuernagel et al. (2018) annotated two NLR putative resistance genes (*NLR 43* and *NLR 143*) in *QYr.cim-2BS* region, which are overlapping with *TraesCS2B01G180700* and *TraesCS2B01G182800* genes, respectively. The sequence analysis revealed that *TraesCS2B01G180700* belongs to resistance gene cluster on chromosome 2B encoding leucine-rich repeat (LLR) proteins (IWGSC 2018). While *TraesCS2B01G182800* has different functions based on the annotation of Chinese spring reference genome (nucleotide-binding site and leucine-rich repeats (NBS-LRR) protein, winged helix-turn-helix DNA-binding domain, and P-loop containing nucleoside triphosphate hydrolase) (IWGSC 2018). The function of NBS-LRR genes is to participate in plant resistance to pathogens by directly or indirectly interacting with the pathogen effectors. Signal transduction in plants towards activating defense genes is mainly accomplished by leucine-rich repeat regions in resistance genes (Gong et al. 2013).

In chromosome 7B, several Yr genes (*Yr2*, *Yr6*, *Yr39*, *Yr52*, *Yr59*, *Yr63*, and *Yr67*) and quantitative-trait loci (QTL) have been reported (El-Bedewy and Robbelen 1982; McIntosh et al.1998; Lin and Chen 2007; Ren et al. 2012; Zhou et al. 2014; McIntosh et al. 2014; Maccaferri et al. 2015). The significant markers identified in chromosome 7B, except *AC875_rep_c108382_824*, appear to be in the same region based on LD (6 Mbp). These markers are mapped close to *YR52* and *QTL-7BL.3* as previously reported by Yung et al. 2019. AC875_rep_c108382_824 was far away from *Yr52* by about 16 Mbp and hence, it is tagging novel genomic regions. According to the annotation of Chinese Spring reference sequence, SNP *RAC875_c34939_86* reside within coding regions of three NLR putative genes (*NLR80*, *NLR146* and *NLR147*). *NLR1147* overlaps with three functional genes *TraesCS7B01G482200*, *TraesCS7B01G482300* and *TraesCS7B01G48240* while NLP146 and NLR80 overlap with *TraesCS7B01G482800* and *TraesCS7B01G483800*, respectively. *TraesCS7B01G482200* is a gene coding for sucrose synthase (SuSy) that belongs to the glycosyl

transferase family 1 (IWGSC 2018). SuSy is a key enzyme of sucrose metabolism in plant cell that catalyzes the reversible cleavage of sucrose into fructose and either uridine diphosphate glucose (UDP-G) or adenosine diphosphate glucose (ADP-G) (Stein and Granot 2019). The putative genes *TraesCS7B01G48240*, *TraesCS7B01G482800* and *TraesCS7B01G483800* encode NBS-LRR disease resistance proteins which are induced during disease response in plants (IWGSC 2018). The gene *TraesCS7B01G482300* encodes for chromodomain-helicase-DNA-binding (CHD) domain having P-loop motif containing nucleoside triphosphate hydrolase (IWGSC 2018).

Of the markers found on chromosome 2D, *Tdurum_contig11802_864*, *BS00067382_51* and *Excalibur_c25599_358* located on the short arm of the chromosome were associated with YR resistance. In line with their distant map positions, these markers are in LD with each other (R^2 = 0.76–0.92) and thus tag same genomic region. Based on the information from adjacent markers, these markers are classified into the QTL *QYr.caas-2DS_Libellula* (Lu et al. 2009). However, we are uncertain whether these markers are potentially novel resistance loci because we are unable to compare the relative distances between this marker and the flanking markers (*Xcfd51* and *Xgwm261*) of *Qyr.caas-2DS*. The QTL was only detected in the 2015 trial with high LOD score and PVE values which indicates either race specificity or environmental factors impacting on effectiveness of resistance. The confidence interval of *Kukri_c12648_434* do not overlap with positions of any of the previously reported Yr genes or QTL. Thus, it likely represents novel resistance loci for *Pst* and needs to be confirmed using an allelism test.

The identification of favorable alleles or stable QTL for stripe rust resistance is a prerequisite to enhancing the resistance in area where the breakdown can occur and new races are present or likely to emerge. The introgression of these favorable alleles into adapted wheat cultivars is important for the improvement of stripe rust resistance. In this study, we have observed that the level of resistance increases by increasing the number of alleles conferring resistance that both contribute for better yield performance. We have identified proteins, involved in plant disease resistance and defense mechanism, linked to previously reported and novel genes/QTL. The novel markers found might be useful in marker assisted selection, but it is essentially important to validate them by using different genetic background and test them across multiple disease hotspots.

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