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ORIGINAL PAPER

Identification of adult plant resistance to stripe rust in the wheat cultivar Cappelle-Desprez

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Abstract Following the appearance of stripe rust in South Africa in 1996, efforts have been made to identify new sources of durable resistance. The French cultivar Cappelle-Desprez has long been considered a source of durable, adult plant resistance (APR) to stripe rust. As Cappelle-Desprez contains the seedling resistance genes *Yr3a* and *Yr4a,* wheat lines were developed from which *Yr3a* and *Yr4a* had been removed, while selecting for Cappelle-Desprez derived APR effective against South African pathotypes of the stripe rust fungus, *Puccinia striiformis* f. sp. *tritici.* Line Yr16DH70, adapted to South African wheat growing conditions, was selected and crossed to the stripe rust susceptible cultivar Palmiet to develop a segregating recombinant inbred line mapping population. A major effect QTL, *QYr.ufs-2A* was identified on the short arm of chromosome 2A derived from Cappelle-Desprez, along with three QTL of smaller effect, *QYr.ufs-2D, QYr.ufs-5B* and *QYr.ufs-6D. QYr.ufs-2D* was located within a region on the short arm of chromosome 2D believed to be the location of the stripe rust resistance gene *Yr16*. An additional minor effect QTL, *QYr.ufs-4B,* was identified in the cv. Palmiet. An examination of individual RILs carrying single or combinations of each QTL indicated significant resistance effects when *QYr.ufs-2A* was combined with the three minor QTL from Cappelle-Desprez, and between *QYr.ufs-2D* and *QYr.ufs-5B.*

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Introduction

Stripe rust, caused by the fungus *Puccinia striiformis* f. sp. *tritici,* was first observed in South Africa in 1996 in the winter rainfall region of the Western Cape Province (Pretorius et al. 1997). Subsequent surveys at the time of detection showed that the disease was well established in the Western, Northern and Eastern Cape Provinces, with trace amounts present on irrigated wheat further north in the summer rainfall region. The above-average rainfall, exacerbated by lower than average temperatures recorded during August and September 1996 contributed significantly to the establishment, spread and subsequent epidemic outbreak of stripe rust (Pretorius et al. 1997). The spread of stripe rust in 1997 led to the disease becoming endemic in all major wheat producing areas of South Africa within 2 years (Boshoff et al. 2002a). Only one pathotype, 6E16A- (virulent to *Yr2*, *Yr6*, *Yr7*, *Yr8* and *Yr17*) was detected in 1996 (Pretorius et al. 1997; Boshoff et al. 2002b). This pathotype was previously detected in East and North Africa, the Middle East, and Western Asia (Badebo et al. 1990; Louwers et al. 1992). Using DNA marker data, Hovmøller et al. (2008) confirmed similarities between South African stripe rust pathotypes and some Central and Western Asian and European isolates. During 1998, the presence of a second pathotype, 6E22A- with

added virulence to *Yr25,* was recorded. In 2001 a new variant, 7E22A-, virulent to *Yr1*, was detected in a trap nursery in the highlands of Lesotho (Pretorius et al. 2007). Pathotype 6E22A+, defeating *YrA,* was identified in 2005 (Pretorius ZA, unpublished data), resulting in four stripe rust pathotypes in total to date.

Internationally, the emergence of new, more aggressive strains of the stripe rust fungus is of concern to the wheat industry (Chen 2005; Hovmøller et al. 2008). In addition to increased aggressiveness of the pathogen, several previously effective resistance genes are now failing to provide adequate disease protection (Chen 2007; Germán et al. 2007; Hovmøller and Justesen 2007; Wellings 2007, 2011). It is therefore crucial to identify additional sources of durable resistance, and improve the characterisation of known sources, to facilitate gene pyramiding in breeding programs. In contrast to seedling resistance genes, which are normally expressed throughout the lifespan of the host plant, i.e. all-stage resistance, adult plant resistance (APR) genes are predominantly expressed later in plant development. APR that falls in the race-nonspecific class (Lagudah 2011) usually results in partial resistance or slow-rusting phenotypes, and is considered to be durable. The incorporation into new cultivars of a combination of APR genes, often resulting in a high level of resistance, is deemed an effective management strategy (Singh et al. 2011).

In view of the importance of stripe rust, South African wheat breeders are making a conscious effort to introduce durable stripe rust resistance genes into new breeding material. A targeted research effort has identified rust resistance QTL in the South African cv. Kariega (Ramburan et al. 2004; Prins et al. 2011). The major QTL, *QYr.sgi-2B* and *QYr.sgi-4A,* and the *Lr34/Yr18* gene present in Kariega are currently being utilised in breeding programs. However, to extend the diversity of stripe rust resistance QTL available to South African wheat breeders, other sources of resistance are continuously being investigated. One of these sources, with a history of durable resistance, is the European winter wheat cv. Cappelle-Desprez.

After its release in France in 1946, Cappelle-Desprez was widely cultivated in Western Europe through to the 1970s (Lupton and Macer 1962; Worland and Law 1986; Bonjean et al. 2001). When released, the cultivar exhibited complete resistance to stripe rust. Cappelle-Desprez is known to possess the seedling resistance genes *Yr3a* and *Yr4a* (De Vallavieille-Pope et al. 1990) on chromosome 1B and 6B, respectively (Chen et al. 1996), but reports on the APR in Cappelle-Desprez are less definitive. Early genetic and cytogenetic studies implicated a locus on chromosome 2D, designated *Yr16* (Worland and Law 1986), and the 5BS-7BS translocation (Law and Worland 1997) as conferring stripe rust APR. *Yr16* and the 5BS-7BS translocation are, however, unlikely to be the only contributors to stripe rust APR in Cappelle-Desprez. While the identity of the full complement of stripe rust resistance in Cappelle-Desprez remains unclear, its resistance is believed to have been incorporated into many European varieties. Under controlled environmental conditions, adult plants of Cappelle-Desprez also showed resistance to leaf rust (Poyntz and Hyde 1987), which raised the question as to whether Cappelle-Desprez possessed *Lr34/Yr18* (McIntosh 1992; Bossolini et al. 2006). However, Lagudah et al. (2009), using gene specific markers, showed that *Lr34/Yr18* is not present in Cappelle-Desprez.

Capelle-Desprez has shown excellent stripe rust resistance since it was first evaluated in South Africa in 1998 (Boshoff et al. 2002b; Pretorius ZA, unpublished). To diversify the South African stripe rust resistance gene pool, Cappelle-Desprez was introduced through a cross to the susceptible South African cv. Palmiet. By means of a selection process an adapted breeding line, Yr16DH70 was developed which retained Cappelle-Desprez APR, but lacked the seedling stripe rust resistance genes *Yr3a* and *Yr4a*. The objective of this study was to confirm the presence and location of *Yr16* on chromosome 2D and the role of the 5BS-7BS translocation in stripe rust resistance, while investigating whether additional stripe rust resistance QTL, derived from Cappelle-Desprez, are effective under field conditions in South Africa.

Materials and methods

Plant material

An initial cross was made between the French winter wheat cv. Cappelle-Desprez (pedigree: Vilmorin-27/Hybride-du-Joncquois) and the South African cv. Palmiet (pedigree: SST3*//Scout*5/Agent). Palmiet is a stripe rust susceptible spring wheat carrying the stem rust resistance genes *Sr2* and *Sr24,* as well as the leaf rust resistance gene *Lr24* (Smit et al. 2010). Cappelle-Desprez × Palmiet F_2 seedlings were infected with spores of *P. striiformis* f. sp. *tritici* pathotype 6E16A- (avirulent/virulent for *Yr1*,*3a*,*4a*,*4-b*,*5*,*9*,*10*,*15*,*25*,*27*,*A*,*Sp*/*Yr2*,*6*,*7*,*8*,*17*) in greenhouse tests. Susceptible seedlings were kept, and those plants expressing APR in the field after infection with the stripe rust pathotype 6E22A- (avirulent/virulent for *Yr1*,*3a*,*4a*,*4-b*,*5*,*9*,*10*,*15*,*27*,*A*,*Sp*/*Yr2*,*6*,*7*,*8*,*17*,*25*) were advanced to the F_4 generation through selfing. One F_4 plant was selected for backcrossing to Palmiet based on its high level of stripe rust resistance and other favourable agronomic characters. Doubled haploid (DH) lines were produced from the resulting F_1 plants. One DH line, Yr16DH70, was subsequently selected and crossed to Palmiet to generate a recombinant inbred line (RIL) mapping population,

consisting of 201 individuals, by advancing random individual F_2 plants to the F_7 generation by single seed descent.

Disease evaluation

Field trials were conducted during 2009 and 2010 at the PANNAR Research Station, Greytown, KwaZulu-Natal, South Africa. The 201 RILs were planted in 2 randomised replicates. Entries were planted in 1-m row plots spaced 90 cm apart. Each replicate contained several Palmiet and Yr16DH70 parental controls. The highly susceptible cv. Morocco was planted in spreader rows bordering the trial area, in all pathways perpendicular to plots, and as every tenth entry within the trial. Control plots of Cappelle-Desprez were also included. Field trials were infected with a spore suspension of pathotype 6E22A+ of *P. striiformis* f. sp. *tritici* (avirulent/virulent for *Yr1*,*3a*,*4a*,*4b*,*5*,*9*,*10*,*15*,*27*,*Sp/Yr2*,*6*,*7*,*8*,*17*,*25*,*A*). Stripe rust severity was scored using the modified Cobb scale (0–100% leaf area infected, LAI) as a quantitative measure of disease infection (Peterson et al.1948; McIntosh et al. 1995). Host reaction type (RT) was scored on an ordinal scale as resistant (R); moderately resistant (MR); moderately susceptible (MS) and susceptible (S) (McIntosh et al. 1995), augmented with resistant–moderately resistant (RMR); moderately resistant–moderately susceptible (MRMS) and moderately susceptible–susceptible (MSS). These seven classes of reaction types were assigned a numerical value of 1 (resistant)–7 (susceptible). Phenotyping was performed once the first signs of stripe rust infection were visible on Palmiet, and until the reaction type classes could no longer be accurately distinguished. For both years, this was between plant growth stages 41 (flag leaf sheath extending) and 69 (anthesis completed) (Zadoks et al. 1974). LAI was scored at the Greytown site on 14 September 2009 (LAI14Sept09), 23 September 2009 (LAI23Sept09), 5 October 2009 (LAI5Oct09), 27 September 2010 (LAI27-Sept10) and 11 October 2010 (LAI11Oct10). RT was scored on 14 September 2009 (RT14Sept09), 23 September 2009 (RT23Sept09) and 27 September 2010 (RT27Sept10).

Statistical analyses

All statistical analyses were performed using the statistical package Genstat for Windows, release 12 (Genstat 5 Committee 2005; Payne et al. 2009). The LAI and RT scores were transformed to achieve near normality and independence of the means and variances. LAI data were converted to proportions and transformed to arcsin (Sokal and Rohlf 1995), while the RT data were transformed with the log function (ln(score + 1)) (Ramburan et al. 2004). For the seedling stripe rust resistance tests, segregation ratios were used to test gene postulations by Chi-square goodness-of-fit. Comparisons between field phenotypic data sets were carried out for both LAI and RT scores using a 2-sided test of correlation. Analysis of variance (ANOVA) was carried out using the General Linear Regression (GLR) model in Genstat v12, which supports unbalanced designs and missing data. The effects of field replicates and genotypes were accounted for in the model. Predicted means for each RIL, for all LAI and RT data sets, were extracted and used in further analyses to minimise error variation in mean values. To identify significant effects between different QTL combinations, the GLR model compared RILs within QTL groups and between groups using *t* test comparisons, with all QTL groups being compared to all other groups (Jagger et al. 2011).

Marker linkage analysis and QTL identification

Genomic DNA was isolated from the parents and a representative individual from the Palmiet × Yr16DH70 F_6 and F_7 RIL generations using the cetyltrimethyl ammonium bromide (CTAB) DNA extraction method (Doyle and Doyle 1990). This DNA was used for simple sequence repeat (SSR) marker analysis. For diversity arrays technology (DArT) analysis, DNA was extracted using the Zymo Research Plant/Seed DNA Kit™. DNA concentrations were determined with the NanoDrop Spectrophotometer ND-1000 and diluted to 50 ng/μL. The SSR marker technique described by Röder et al. (1998) was followed.

The SSR primers used included markers from the WMC (Wheat Microsatellite Consortium, Gupta et al. 2002); CFA (Clermont-Ferrand A genome, Sourdille et al. 2003); CFD (Clermont-Ferrand D genome, Guyomarc'h et al. 2002); GWM (Gatersleben Wheat Microsatellite, Röder et al. 1998); GDM (Gatersleben D genome Microsatellite, Pestsova et al. 2000); PSP (Bryan et al. 1997) and BARC (Beltsville Agriculture Research Center, Song et al. 2005) series. Fluorescently labelled primers were synthesised by Applied Biosystems. Markers were amplified and subsequently analysed on the 3730*xl* Genetic Analyzer (Applied Biosystems) using GeneScan™ 500 LIZ® as the internal size standard. Data were analysed using GeneMapper v4.0 (Applied Biosystems). The parental lines, Palmiet and Yr16DH70, were screened with 465 SSR markers. These markers were uniformly distributed across the three wheat genomes, with each of the 21 chromosomes being well represented. Polymorphic SSR markers and DArT markers (using the *PstI*/*TaqI* v3.0 array enriched for the D-genome, Triticarte P/L and Diversity Arrays Technology P/L, Canberra, Australia) were typed in the RIL population. One sequence tagged site (STS) marker for the gene *Pina-D1* (US public MAS wheat breeding programs, http://maswheat.ucdavis.edu) and one converted DArT marker,

wPt-0600 (Yu et al. 2009) were also screened in the population.

Marker data were used to construct a linkage map. Markers were ordered according to the RECORD output (Van Os et al. 2005). Map distances were calculated using the Kosambi map function from MapManager QTXb20 (Manly et al. 2001) and linkage groups were declared based on map distances and linkages between markers at a significance level of $P = 0.001$. Chromosomes were assigned to linkage groups by referring to a consensus map (Appels 2003). Heterozygote scores were replaced with a missing data score. Redundant loci were hidden and unlinked markers were excluded for the purpose of QTL analysis.

For QTL analyses, composite interval mapping (CIM) was performed with Windows QTL Cartographer v2.51 (Wang et al. 2011), using a forward regression model with a window size of 10 cM and a walk speed of 1 cM. Both transformed and untransformed LAI and RT phenotypic data were analysed to increase confidence in the QTL identified. Predicted means obtained for each RIL from the GLR analyses in Genstat v12 were also used in the QTL analysis. To determine the LOD threshold value above which a QTL is considered significant, 1,000 permutations were performed ($P = 0.05$) (Doerge and Churchill 1996) for the LAI and RT traits at each score date. Maps were prepared using MapChart v2.1 (Voorrips 2002). RILs were placed in QTL groups depending on the combination of QTL they carried. The presence of a QTL was based on the genotype of the QTL-associated markers with the highest LOD values in the QTL interval. The predicted means for the LAI and RT phenotypes were calculated for each QTL group.

Results

Development of the mapping population

In order to eliminate seedling stripe rust resistance, F_2 seedlings from the cross Cappelle-Desprez × Palmiet were screened in greenhouse tests with the pathotype 6E16A-. These tests yielded 98 resistant and 6 susceptible plants, giving a good fit to the expected 15:1 ratio for the segregation of the two dominant resistance genes, *Yr3a* and *Yr4a* from Cappelle-Desprez ($X^2 = 0.041$; $P = 0.84$). Adult plant ratings of the susceptible seedlings were 0R (five plants) and 70MS (one plant). The resistant plants were advanced to the F_4 generation through selfing, with F_3 and F_4 progeny being tested for APR in the field. One F_4 plant, displaying complete stripe rust field resistance and other favourable agronomic characters was crossed to Palmiet, a series of DH lines being produced from the resulting F_1 seed to obtain a pure breeding line. The DH line Yr16DH70 was subsequently selected for backcrossing to Palmiet. Yr16DH70 did not display seedling resistance against the South African pathotypes tested (infection type 2+ to 3), but retained APR which was effective under field conditions in South Africa (30R at the end of the growing season). A RIL mapping population (201 lines) was derived from the cross Palmiet × Yr16DH70.

Field assessment of stripe rust resistance

Good stripe rust establishment was seen in the field in both years, the highly susceptible cv. Morocco displaying 100S midway through the scoring seasons. Although Palmiet is fully susceptible to stripe rust in the winter rainfall regions of South Africa (Boshoff et al. 2002a), severe infection is associated with chlorosis and necrosis in the warmer Greytown environment. In 2009 Palmiet scored 70–80MS, while the resistant parent, Yr16DH70 scored 20R at the last assessment (Online Resource 1). Less infection was seen in the dryer and warmer 2010 season, with Palmiet scoring 40–50MR and Yr16DH70 10–15R (Fig. 1). In general, Cappelle-Desprez expressed marginally higher levels of resistance, having scores from 0R through to 20R in field tests from 1999 to 2011.

Although Palmiet was not fully susceptible in these field trials, ANOVA showed clear segregation of resistance in the RIL population for all LAI and RT data sets (variation between RIL, F value prob. < 0.001; Fig. 1). Some variation between replicates was seen, although this was only significant for the data sets LAI23Sept09 and RT27Sept10 (F prob. < 0.001). Transgressive segregation was seen in both years, with some lines being more resistant than Yr16DH70 and others more susceptible than Palmiet (Fig. 1). Comparing phenotypic data sets showed high, positive correlations for LAI scores, both within and across years ($r = 0.61$–0.73). For RT, the correlation between the two score dates in 2009 ($r = 0.74$) was greater than between years ($r = 0.53$ and 0.58), although all correlations were significant ($P < 0.001$). Significant correlations were also observed when comparing LAI and RT phenotypes between the RIL, with correlations being greater within years ($r = 0.59$–0.76) than between years ($r = 0.52$–0.64).

Genetic linkage map construction

Seventy-three of the 465 (15.7%) SSR markers screened were polymorphic between Palmiet and Yr16DH70. These were mapped in the RIL population along with 336 DArT markers and 2 STS markers (one converted DArT and the *Pina-D1* quality marker). At the F_6 generation, an average of four heterozygotes (2%) per SSR marker could still be detected in the RIL population. Fifteen linkage groups, containing 3 or more loci were identified, representing 12 chromosomes (Online Resource 2). All the DArT markers

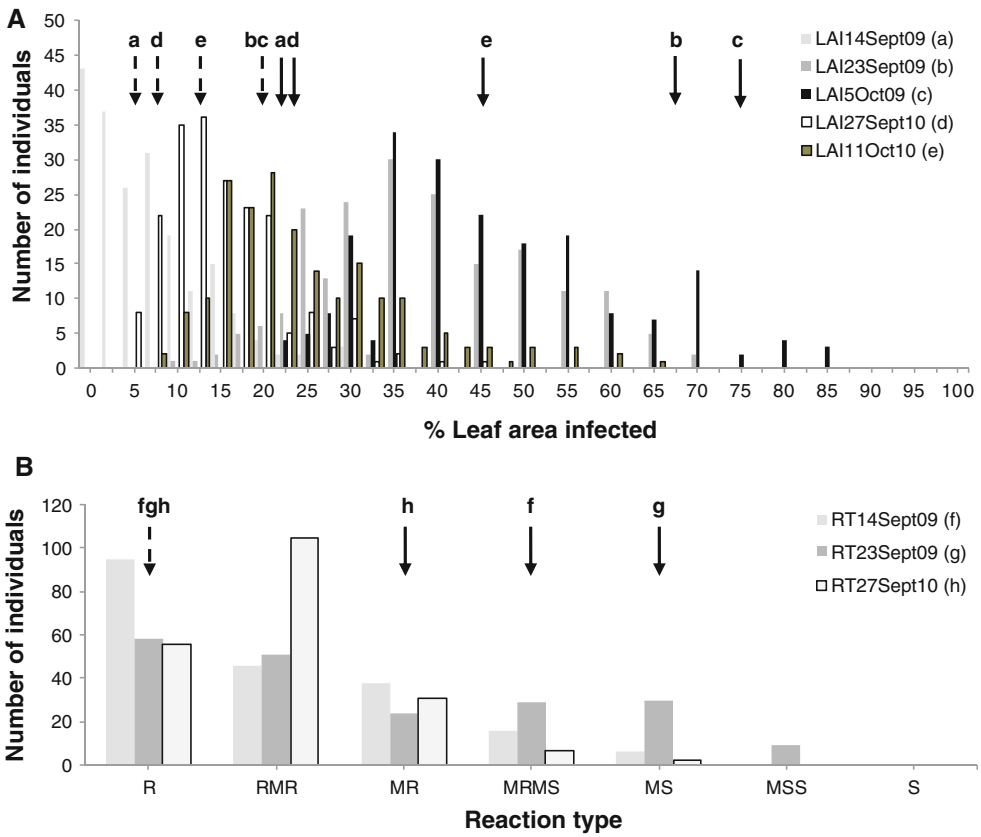

Fig. 1 Segregation of leaf area infected (LAI) (**a**) and reaction type (RT) (**b**) phenotypes in the Palmiet × Yr16DH70 RIL mapping population for the 2009 and 2010 seasons. The *broken arrow* represents the disease scores for Yr16DH70 and the *solid arrow* the scores for Palmiet for the eight phenotypic data sets as indicated by *lowercase letters*

could be incorporated into linkage groups, but four SSR markers (WMC173, GDM111, WMC285 and BARC110) remained unlinked at a significance level of $P = 0.001$.

In particular, good marker coverage was obtained for chromosomes 2A and 2D (Online Resource 2). The linkage group representing chromosome 2A contained 32 markers with a total map distance of 138.7 cM, while the 2D linkage group contained 96 markers and covered a distance of 158.2 cM. Chromosomes 5B and 7B were both represented by two, unlinked linkage groups, representing the short and long arms of each chromosome (Online Resource 2). This would be expected if Yr16DH70 had inherited the 5BS-7BS translocation from Cappelle-Desprez. Chromosome 5D was also represented by two linkage groups (Online Resource 2). Chromosomes 1A, 1D, 2B, 3A, 4A, 4D, 6A, 6B and 7D were not represented by linkage groups, indicating chromosomes represented primarily by Palmiet DNA. The high number of chromosomes fixed for Palmiet DNA was supported by the lower level of polymorphism found for SSR markers between Palmiet and Yr16DH70 (15.7%), as compared to Palmiet and Cappelle-Desprez (68.0%).

QTL analysis of stripe rust resistance

CIM analysis detected QTL on chromosomes 2A (*QYr.ufs-2A*), 2D (*QYr.ufs-2D*), 5B (*QYr.ufs-5B*) and 6D (*QYr.ufs-6D*) from Yr16DH70 and one QTL on chromosome 4B (*QYr.ufs-4B*) from Palmiet. Between 33.6 and 75.2% of the total phenotypic variation could be explained by the identified QTL, depending on the phenotypic data set analysed (Table 1). QTL analysis was carried out using both transformed and untransformed phenotypic scores, identifying the same QTL, with very similar LOD scores. Only the analyses using the untransformed data are reported (Table 1).

A major effect QTL, *QYr.ufs-2A* was detected on the short arm of chromosome 2A with all the phenotypic data sets, explaining up to 53.2% of the phenotypic variation for RT and 47.8% of the variation for LAI (Fig. 2). The QTL mapped between the marker loci *wPt-733314* and *wPt-0003* with the peak located between *Xgwm636* and *wPt-0003*. With the LAI phenotype data sets, a potential QTL was also detected at the opposite end of the 2A linkage group, having a maximum LOD value of 3.2 (Fig. 2a). However, the presence of this second QTL on 2AL requires further investigation to provide confirmation. Three further, minor QTL were derived from Yr16DH70. *QYr.ufs-2D* was detected on the short arm of chromosome 2D flanked by the marker loci *Xgwm102* and *wPt-664520* and explained up to 10.3% of the phenotypic variation, being detected by both the LAI and RT phenotypic data sets (Fig. 3; Table 1). *QYr.ufs-5B* explained up to 5.7% of the

Table 1 Summary of stripe rust APR QTL detected with CIM using LAI and RT phenotypic data sets

QTL interval[a]	Chr[b]		Percentage leaf are infected (LAI)					Host reaction type (RT)			Origin
			14 Sept 09	23 Sept 09	5 Oct 09	27 Sept 10	11 Oct 10	14 Sept 09	23 Sept 09	27 Sept 10	
QYr.ufs-2A	2A	LOD	10.7	18.8	13.7	11.6	13.8	10.6	16.2	8.6	Yr16DH70 (CD[d])
wPt-733314–wPt-0003		%VAR[c]	28.8	47.8	39.7	32.6	36.9	30.5	53.2	23.4	
QYr.ufs-2D	2D	LOD	–	–	–	2.9	5.4	–	6.6	6.1	Yr16DH70 (CD)
Xgwm102–wPt-664520		%VAR				4.7	8.4		10.3	9.9	
QYr.ufs-4B	4B	LOD	–	–	–	–	–	4.7	7.6	–	Palmiet
Xgwm165–Xgwm495		%VAR						7.0	11.7		
QYr.ufs-5B	5B	LOD	3.2	3.4	2.8	–	–	3.8	–	3.5	Yr16DH70 (CD)
wPt-7114–Xbarc74		%VAR	4.8	4.9	4.3			5.7		5.3	
QYr.ufs-6D	6D	LOD	–	4.2	2.8	4.2	–	–	–	–	Yr16DH70 (CD)
Xgwm325–Xbarc175		%VAR		7.6	4.5	6.2					
Total variance explained (%)			33.6	60.4	48.4	43.5	45.3	43.2	75.2	38.6	

[a] Only QTL with LOD values above the significance levels that were determined after 1,000 permutations ($P = 0.05$) are shown

[b] Chromosome

[c] Percentage phenotypic variance explained (R^2) with '–' meaning not significant

[d] Derived from Cappelle-Desprez

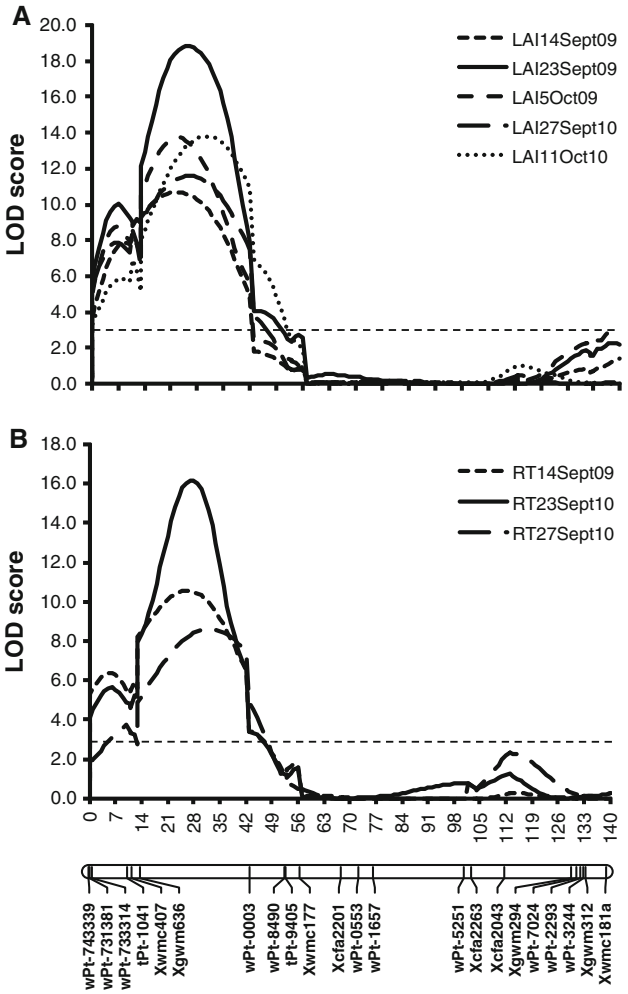

Fig. 2 Stripe rust resistance QTL, *QYr.ufs-2A* on chromosome arm 2AS identified with the leaf area infected (**a**) and reaction type (**b**) data sets. The distances between markers are shown in centiMorgans. The LOD threshold, as determined from 1,000 permutations using Cartographer v2.51 (Wang et al. 2011) is shown

phenotypic variation, and was detected by both the LAI and RT phenotypes (Table 1; Online Resource 3). *QYr.ufs-5B* spanned an interval of 11.1 cM and was flanked by marker loci *wPt-7114* and *Xbarc74*, placing the QTL near to the centromere. *QYr.ufs-6D* was detected only with the LAI phenotype (Table 1; Online Resource 3). It explained up to 7.6% of the phenotypic variance and was located between the SSR marker loci *Xgwm325* and *Xbarc175*, placing the QTL on the long arm of chromosome 6D. A minor QTL was also detected in Palmiet on chromosome 4B flanked by marker loci *Xgwm165* and *Xgwm495* (Table 1; Online Resource 3). *QYr.ufs-4B* explained up to 11.7% of the phenotypic variance, and was only detected using the RT phenotypic data (Table 1).

Each RIL was placed in one of 32 groups depending on the five QTL they carried (Online Resource 4). The predicted means of each QTL group were obtained from an

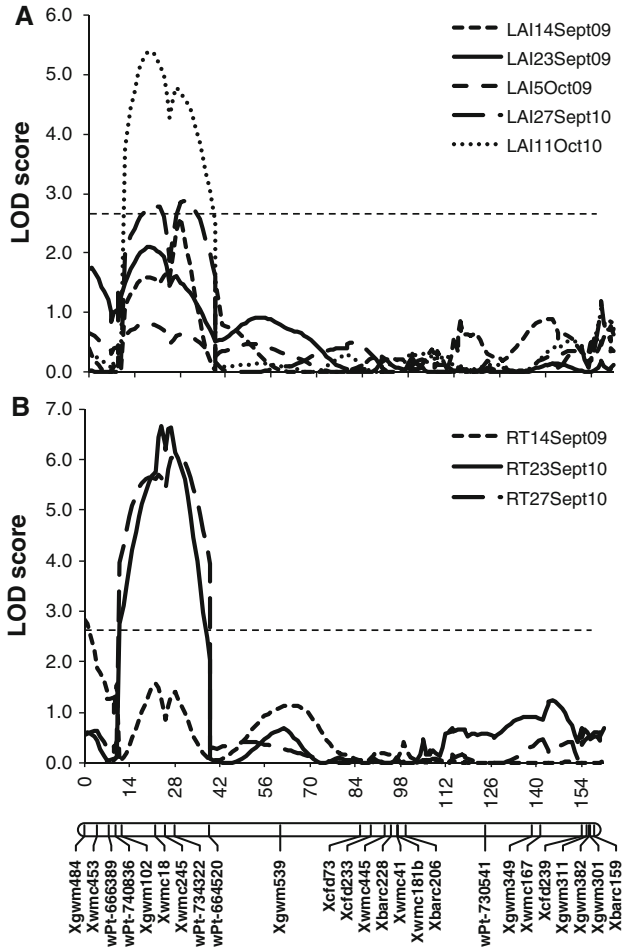

Fig. 3 Stripe rust resistance QTL, *QYr.ufs-2D* on chromosome arm 2DS identified with the leaf area infected (**a**) and reaction type (**b**) data sets. The distances between markers are shown in centiMorgans. The LOD threshold, as determined from 1,000 permutations using Cartographer v2.51 (Wang et al. 2011) is shown

ANOVA, using the GLR model in Genstat v12, for all eight LAI and RT datasets (Fig. 4; only QTL groups carrying Yr16DH70-derived QTL are shown). With both LAI and RT phenotypes, significant differences were found between QTL groups (F prob. < 0.001), while no differences were found between RILs within QTL groups. Following the ANOVA, t test comparisons of the RILs within each QTL group were undertaken, comparing all 32 QTL groups to each other. A significant effect on stripe rust phenotypes was seen when *QYr.ufs-2A* was combined with the small QTL derived from Yr16DH70, removal of one or more small effect QTL altering the level of resistance (t test prob. < 0.01–0.05; Fig. 4). Lower disease scores were also seen for both LAI (t test prob. < 0.05) and RT (t test prob. < 0.001 to 0.05) when the minor QTL *QYr.ufs-2D* and *QYr.ufs-5B* were combined. A significant reduction in disease was not consistently seen with any other pairwise combinations of the Yr16DH70-derived QTL.

Discussion

Cappelle-Desprez was first released in France in 1946 and occupied a considerable acreage across Western Europe until the late 1970s (Lupton and Macer 1962; Worland and Law 1986; Bonjean et al. 2001). The longevity of this cultivar was exceptional, which in part was due to its high levels of APR to a number of major diseases, including stripe rust. Consequently, Cappelle-Desprez was used extensively in breeding programs within the UK and Western Europe (Angus 2001; Bonjean et al. 2001; Porche 2001). Cappelle-Desprez is still recognised as a good source of stripe rust APR and in South Africa has maintained high levels of resistance since being tested for the first time in 1998 (Boshoff et al. 2002b).

Previous studies of stripe rust resistance in Cappelle-Desprez have suggested several APR loci (Worland and Law 1986; Law and Worland 1997) in addition to the seedling resistance genes *Yr3a* and *Yr4a* (Lupton and Macer 1962; De Vallavieille-Pope et al. 1990). To increase the sensitivity of detection of minor APR loci derived from Cappelle-Desprez, the line Yr16DH70 was developed, whereby the race-specific, seedling resistance genes *Yr3a* and *Yr4a* had been removed. The development of Yr16DH70 resulted in much of the genome of this line consisting of Palmiet DNA. Twenty-three percent of all polymorphic SSR markers between Cappelle-Desprez and Palmiet (316 SSR markers) were also polymorphic between Yr16DH70 and Palmiet (73 SSR markers). This would fit with the expected genetic structure of Yr16DH70, where 25% of the genome should be derived from Cappelle-Desprez and 75% from Palmiet. Genetic mapping of the Palmiet × Yr16DH70 RIL population therefore resulted in the identification of 12 of the potential 21 chromosomes of hexaploid wheat.

Subsequent QTL analyses identified a major effect stripe rust resistance QTL on the short arm of chromosome 2A (*QYr.ufs-2A*) and three minor QTL on chromosomes 2D (*QYr.ufs-2D*), 5B (*QYr.ufs-5B*) and 6D (*QYr.ufs-6D*). In addition, a minor QTL was detected in the South African cv. Palmiet on chromosome 4B (*QYr.ufs-4B*). While up to 75.2% of the variation seen for RT was explained by the QTL identified, only 60.4% of the variation seen for LAI was explained. Although heavy selection pressure for adult plant stripe rust resistance was imposed during the development and selection of Yr16DH70, minor QTL and QTL where expression is heavily influenced by environmental factors (Boukhatem et al. 2002), may not have been retained in Yr16DH70. This may account for the consistently, but marginally higher disease rating obtained for Yr16DH70 compared to Cappelle-Desprez in each year of testing.

QYr.ufs-2A was responsible for a significant proportion of the stripe rust resistance displayed by Yr16DH70 (up to

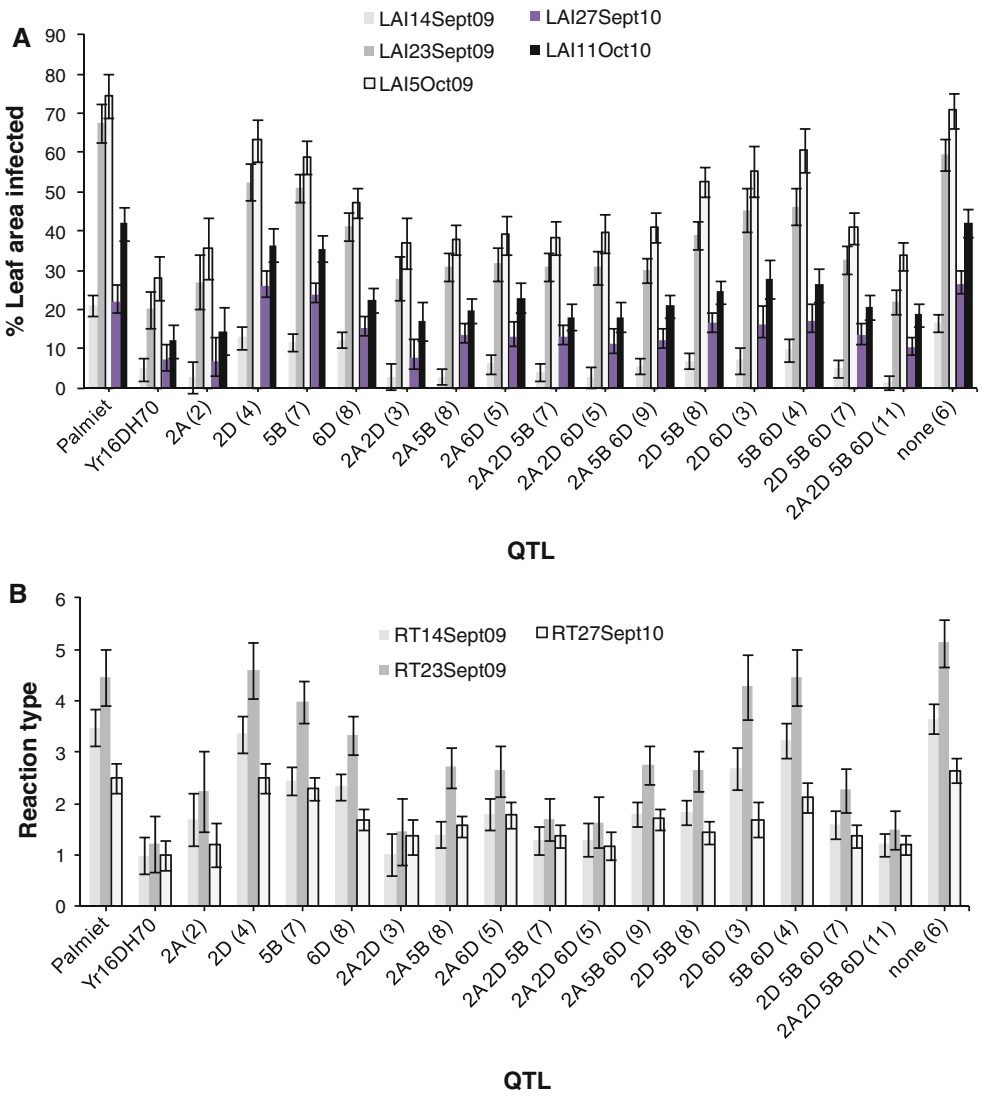

Fig. 4 **a** Mean percentage leaf area infected (LAI) and **b** reaction type (RT) of the RIL defined by the four stripe rust APR QTL derived from Yr16DH70. Groups containing the Palmiet QTL (*QYr.ufs-4B*) are not shown in the figure to simplify the display. The phenotypes of the individual QTL *QYr.ufs-2A, QYr.ufs-2D, QYr.ufs-5B* and *QYr.ufs*-6D are shown, as well as the phenotypes of selected QTL combinations. The number of genotypes in each group is indicated in *brackets*. The reaction type scores are represented on an ordinal scale with *1* resistant, *2* resistant–moderately resistant, *3* moderately resistant, *4* moderately resistant–moderately susceptible, *5* moderately susceptible and *6* moderately susceptible–susceptible. *Error bars* show standard errors of the means

53.2%) and was consistently detected by both LAI and RT scores. The association between low LAI and low RT scores would indicate a QTL for stripe rust resistance exhibiting a necrotic phenotype typical of race-specific resistance. Statistical analyses of the LAI and RT scores of the RIL within each QTL group indicated significant interactions between *QYr.ufs-2A* and the three smaller QTL derived from Yr16DH70, indicating that although *QYr.ufs-2A* conferred a significant level of stripe rust resistance this could still be enhanced by small effect QTL. SSR markers defined the location of *QYr.ufs-2A* to the short arm of chromosome 2A. The stripe rust seedling resistance gene *Yr17* was transferred to chromosome 2AS from *Triticum ventricosum* (Helguera et al. 2003) and maps to the same region as *QYr.ufs-2A* (Christiansen et al. 2006). However, the detection of seedling resistance genes is improbable as the population was developed from the seedling susceptible, Cappelle-Desprez derived line Yr16DH70. Furthermore, the release of Cappelle-Desprez predates the development of the *Yr17* translocation. In addition, the 2NS-specific marker VENTRIUP/LN2 (Helguera et al. 2003) was used to show that the *Lr37/Yr17/Sr38* segment is not present in Cappelle-Desprez or Yr16DH70 (Online Resource 5). This does not exclude *QYr.ufs-2A* from representing an adult plant race-specific resistance locus which has not as yet been overcome by South African pathotypes. Boukhatem et al. (2002) detected an APR QTL (*QYR2*) in the cv. Camp Rémy, a descendant of Cappelle-Desprez which is also described as having durable stripe rust resistance. *QYR2* was located to a region on the long arm of chromosome 2A which is defined by the SSR marker loci *Xgwm356* and *Xgwm382*. Mallard et al. (2005) also detected an APR locus in Camp Rémy on chromosome 2A and designated the QTL, *QYr.inra-2AL*. However, the designation of this QTL to the long arm of 2A may be in question, as all the SSR markers in this QTL

interval, except one, GWM382, have been assigned to the short arm of chromosome 2A (Appels 2003). As defined by the 2A chromosome short arm SSR marker loci, *QYr.inra-2AL* and *QYr.ufs-2A* would lie within a common interval. Mallard et al. (2005) attributed between 20 and 40% of the phenotypic variance to *QYr.inra-2AL,* which is comparable to the phenotypic variance explained by *QYr.ufs-2A*. In the study of Boukhatem et al. (2002), *QYR2* accounted for only 15.4% of the phenotypic variation. Bariana et al. (2010) identified an APR QTL proximal to the region defining *QYr.ufs-2A* in the cv. Kukri, linked to the DArT marker *wPt-0003*, a locus flanking the *QYr.ufs-2A* interval. In contrast to *QYr.ufs-2A* this QTL explained only 12–15% of the phenotypic variance and was not detected across years. Recently Lowe et al. (2011) identified a small effect QTL, *QYr.ucw-2AS*, contributing 2.3% to the stripe rust resistance expressed by a synthetic derivative [Croc/*Aegilops tauschii* (Synthetic 205)//Kauz]. *QYr.ucw-2AS* was located between the markers *wPt-3896* and *Xwmc177*, placing it in the same region as *QYr.ufs-2A*. Yet another QTL, *QYr.uga-2AS,* was detected in the *QYr.ufs-2A* interval in the cv. Pioneer 26R61, flanked by the markers *Xbarc124* and *Xgwm359* (Hao et al. 2011). This QTL explained up to 56.0% of the phenotypic variation and was consistently expressed across different environments. The short arm of chromosome 2A is therefore a region of interest in terms of stripe rust resistance.

A gene for stripe rust resistance, *Yr16* was located to the centromeric region of chromosome 2D in Cappelle-Desprez through cytogenetic analyses (Worland and Law 1986; Worland et al. 1988). The gene was placed 9.3 cM from the centromere between RFLP marker loci *Xpsr641-2D* and *Xpsr681-2D* on a consensus map, Ta-Gale-2D (http://wheat.pw.usda.gov/GG2/index.shtml; Devos et al. 1993). Mallard et al. (2005) identified a QTL in the cv. Camp Rémy near the centromere on 2DS, *QYr.inra-2DS*. *QYr.inra-2DS* was located between the loci *Xgwm102* and *Xgwm539* and was responsible for 24–69% of the observed phenotypic variance. In this study, *QYr.ufs-2D* mapped to the same region, although defined by a smaller interval (*Xgwm102-wPt-664520*), but contributed significantly less to the phenotypic variance (maximum of 10.3%). *QYr.inra-2DS* and *QYr.ufs-2D* probably represent the same QTL, the differences in phenotypic variance indicating the influence of genetic background and/or environmental conditions. *QYr.ufs-2D* appears to perform less well under heavy disease pressure, not being detected late in 2009, but still being effective late in the drier, low stripe rust year of 2010. The sensitivity of expression of *QYr.ufs-2D/QYr.inra-2DS* would be supported by the fact that Boukhatem et al. (2002) did not identify a QTL on 2D in their Camp Rémy × Michigan Amber study. Various other stripe rust resistance QTL have been mapped to the short arm of chromosome 2D within the same region as *QYr.ufs-2D*. However, the ancestral relationship between these resistance sources and Cappelle-Desprez is not as clear as for the Cappelle-Desprez Camp Rémy relationship. Temperature-sensitive seedling resistance, *YrCK* was reported on chromosome 2D in the Australian cv. Cook and a derivative, cv. Sunco (Park et al. 1992; Bariana et al. 2001), contributing 13–19% of the phenotypic variance for stripe rust and 9–13% of the phenotypic variance for leaf rust resistance (Navabi et al. 2005). A stripe rust resistance QTL was identified in the Italian cv. Libellula, *QYr.caas-2DS* explaining 8.4–12.1% of the phenotypic variance (Lu et al. 2009), while Suenaga et al. (2003) detected a stripe rust resistance QTL in this region of 2DS in the cv. Oligoculm which explained less than 10% of the phenotypic variance. In the UK cvs Guardian (Melichar et al. 2008) and Claire (Powell 2010), both of which have Cappelle-Desprez in their pedigrees, stripe rust resistance QTL were found that mapped close to the *QYr.ufs-2D* region on the short arm of chromosome 2D.

Cappelle-Desprez carries the reciprocal, centromeric translocations 5BL-7BL and 5BS-7BS (Riley et al. 1967; Badaeva et al. 2007) which were common in Western European wheat cultivars in the 1960s and 1970s (Riley et al. 1967). The 5BS-7BS chromosome was previously shown to contribute substantially to stripe rust resistance in Cappelle-Desprez. The parents of Cappelle-Desprez, Vilmorin-27 and Hybride du Joncquois, both possess this translocation (Law et al. 1978; Law and Worland 1997). However, while Vilmorin-27 had a high level of stripe rust resistance, Hybride du Joncquois was susceptible. Ditelosomic 5BS and 7BS Cappelle-Desprez lines subsequently showed the resistance in Cappelle-Desprez to be located on the 5BS arm, close to the chromosomal breakpoint, while the genetic background in which the 5BS chromosome arm resides also appeared to influence the resistance (Law et al. 1978; Law and Worland 1997). The presence of the 5BS-7BS translocation in Yr16DH70 was confirmed by C-banding data (Pretorius ZA, unpublished data). The marker data placed *QYr.ufs-5B* near to the centromeric region on the long arm of chromosome 5B. It has not been defined exactly where the physical breakpoint of the 5BS-7BS and 5BL-7BL translocations are positioned with respect to the centromeres of each chromosome, therefore *QYr.ufs-5B* may represent the stripe rust resistance located on the short arm of chromosome 5B in Cappelle-Desprez. *QYr.ufs-5B* did not make a large contribution to the stripe rust resistance observed in Yr16DH70, however, lower disease scores were seen for both LAI and RT when *QYr.ufs-5B* was combined with *QYr.ufs-2D*.

Mallard et al. (2005) detected two QTL (*QYr.inra-5B.1* and *QYr.inra-5B.2)* in Camp Rémy which mapped to the telomeric region on the long arm of chromosome 5B.

Based on the known map location of the markers defining these QTL (Appels 2003) the authors suggested that the telomeric end of 5BL contained a translocation of a fragment of 5BS. However, this proposal was not confirmed by cytological data. Boukhatem et al. (2002), in their study of the cv. Camp Rémy, did not identify a QTL on chromosome 5B. Stripe rust resistance QTL on chromosome 5B have been detected in the Italian cvs Libellula and Strampelli (Lu et al. 2009), the Israeli cv. Oligoculm (Suenaga et al. 2003), the Australian cv. Janz (Bariana et al. 2010) and the French cv. Flinor (Feng et al. 2011). In Flinor, two QTL were detected on chromosome 5B which are expressed at the seedling stage and at higher temperatures (*QYr-tem-5B.1* and *QYr-tem-5B.2*; Feng et al. 2011). Flinor shares ancestry with Cappelle-Desprez and one of these QTL, *QYr-tem-5B.1,* overlaps with the region defining *QYr.ufs-5B*. *QYr-tem-5B.1* explained up to 33% of the phenotypic variation in Flinor which is substantially greater than the resistance phenotype explained by *QYr.ufs-5B*. It has yet to be determined if there is a relationship between *QYr-tem-5B.1* and *YrDru* from the cv. Druchamp (Chen et al. 1996).

No stripe rust APR QTL have been mapped to chromosome 6D, although the seedling resistance genes *Yr20* and *Yr23* were located on this chromosome (Chen et al. 1995). *QYr.ufs-6D* represents a small effect QTL, only detected with the LAI scores and then not consistently across years. A small effect QTL was also found on chromosome 4B, *QYr.ufs-4B*, derived from Palmiet. Depending on environment and disease pressure, a low level of stripe rust resistance was observed for Palmiet, supporting the detection of this QTL. QTL for stripe rust APR have been reported on chromosome 4B in the cvs Alcedo (Jagger et al. 2011), Oligoculm (Suenaga et al. 2003), Avocet S (William et al. 2006), Guardian (Melichar et al. 2008) and cvs Libellula and Strampelli (Lu et al. 2009). The stripe rust QTL from Avocet S was also reported to have an effect on leaf rust (William et al. 2006). Except for the QTL from Alcedo, which explained up to 29% of the phenotypic variance, none of the above-mentioned QTL accounted for more than 15% of the phenotypic variance. This is in line with the relatively small effect seen for *QYr.ufs-4B*. However, the large interval defining *QYr.ufs-4B* does not allow direct comparison to these other published 4B QTL.

The RILs were assigned to 32 genotype groups depending on which of the 5 QTL they contained. In general, QTL intervals were fairly large, therefore QTL assignment to RILs was based on the marker closest to the QTL peak (highest LOD) (see Online Resource 4). Selection based on markers flanking the QTL interval would have eliminated several lines from each QTL group, this being most evident for the major QTL *QYr.ufs-2A*. *Xgwm636* was used to select for this QTL, having the highest LOD value with all eight traits, the LOD value associated with the flanking marker *wPt-0003* sharply decreases to below 3.0 with most trait data sets. More variation was generally seen in the QTL interval between traits for the minor QTL. Where the marker locus with the highest LOD differed between trait data sets, markers that flanked this locus were also considered in the assignment of RILs to groups. In the case of the problematic region on chromosome 6D, the *Xgwm325–Xbarc202* interval was considered when assigning RILs to QTL groups. The assignment of each QTL was therefore determined by the resolution of the QTL interval, but for each QTL the same selection criteria were consistently applied. The assignment of RILs into QTL groups using this method was supported by the similar stripe rust resistance phenotypes seen for the RILs within each QTL group. Clearly, the QTL intervals need to be delimited to smaller regions in order to facilitate the use of an interval for QTL identification. This would improve the accuracy of QTL assignment and avoid the incorrect assignment of QTL which have been removed via recombination.

In South Africa, Cappelle-Desprez and the Cappelle-Desprez derived breeding line, Yr16DH70, selected to retain the stripe rust APR found in Cappelle-Desprez, exhibits complete resistance to stripe rust under field conditions. In the UK, stripe rust resistance in Cappelle-Desprez is now only partial (Boyd LA, unpublished data). While the *Yr16* (*QYr.ufs-2D*) resistance is still believed to be effective in the UK, as seen in the UK cvs Guardian (Melichar et al. 2008) and Claire (Powell 2010), it may well be that the major effect QTL, *QYr.ufs-2A* detected in Yr16DH70 is no longer effective against UK pathotypes of *P. striiformis* f. sp. *tritici*. *QYr.ufs-2D*, which is in all likelihood the *Yr16* locus previously reported in Cappelle-Desprez is therefore more likely to be of long term value. The value of *QYr.ufs-5B* and *QYr.ufs-6D* is less clear and an improved linkage map of these QTL intervals would help define their value as stripe rust APR genes. The stripe rust APR QTL from Cappelle-Desprez have already been transferred into a desirable South African spring wheat background through the development of the breeding line, Yr16DH70. This study has now defined these QTL and provides markers by which each QTL can be identified as this breeding line is taken forward in the development of new wheat cultivars.

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