Aus der Landessaatzuchtanstalt
der Universität Hohenheim
apl. Prof. Dr. T. Miedaner

Mapping of quantitative-trait loci (QTL) for adult-plant resistance to *Septoria tritici* in five wheat populations (*Triticum aestivum* L.)

Dissertation
zur Erlangung des Grades eines Doktors
der Agrarwissenschaften
vorgelegt
der Fakultät Agrarwissenschaften

von
Master of Science
Peter Risser
aus
Kirchheimbolanden

Stuttgart-Hohenheim
2010

Die vorliegende Arbeit wurde am 24. August 2010 von der Fakultät Agrarwissenschaften der Universität Hohenheim als „Dissertation zur Erlangung des Grades eines Doktors der Agrarwissenschaften“ angenommen.

Tag der mündlichen Prüfung:	13. September 2010
1. Prodekan:	Prof. Dr. A. Fangmeier
Berichterstatter, 1. Prüfer:	apl. Prof. Dr. T. Miedaner
Mitberichterstatter, 2. Prüfer:	Prof. Dr. R. Vögele
3. Prüfer:	Prof. Dr. W. Claupein

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Abbreviations

AFLP	amplified fragment length polymorphism
ANOVA	analysis of variance
AUDPC	area under the disease progress curve
BBCH	decimal code of phenological growth stages, which is divided into principal and secondary growth stages based on the cereal code developed by Zadoks et al. (1974); the abbreviation BBCH derives from **B**iologische **B**undesanstalt, Bundessortenamt and **CH**emical industry (Meier 2001)
CIM	composite interval mapping
cM	centiMorgan
DArT	diversity array technology
df	degrees of freedom
dt	1 dt = 100 kg
e.g.	for example
FHB	fusarium head blight
GS	genetic similarity
HED	heading date
MAS	marker-assisted selection
No.	number
nR^2_{adj}	normalized adjusted R^2
pers. comm.	personal communication
PLH	plant height
QTL	quantitative-trait locus/loci
R^2	phenotypic variance explained by detected QTL
RFLP	restriction fragment length polymorphism
SGB	stagnospora glume blotch
SNP	single nucleotide polymorphism
SSR	simple sequence repeat
STB	septoria tritici blotch

1 INTRODUCTION

1.1 Importance of wheat (*Triticum aestivum* L.) and *Septoria tritici*

Wheat is one of the most important food crops worldwide covering more land area than any other crop (FAOSTAT 2007). In Germany, arable land comprises about 12 million hectare including 3.2 million hectare of wheat. The acreage increased from 2.3 million hectare in 1990 to 3.2 million hectare in 2008, thus more than every fourth hectare in Germany is grown with wheat. In parallel, yield increased from 63 dt/hectare to 81 dt/hectare (Bundessortenamt 2009). In comparison to other wheat growing regions worldwide with an average yield of 10 to 30 dt/hectare (FAOSTAT 2007), Germany is one of the highest yielding areas. Here, winter wheat is grown predominantly, whereas spring wheat is of less importance. Much of the yield increase is due to efforts in plant breeding. In combination with crop management including crop rotation, soil tillage, and plant protection, resistant varieties are of growing importance to protect the high yield against upcoming disease.

Grown wheat varieties belong to three main groups: (i) diploid wheat (einkorn, *T. monococcum*, AA, *2n* = 14), (ii) tetraploid wheat (emmer, durum wheat, *T. turgidum*, AABB, *2n* = 28), and (iii) hexaploid wheat (spelt, bread wheat, *T. aestivum*, AABBDD, *2n* = 42). Einkorn with the A genome is evolutionary the earliest group. Out of it, emmer was formed by hybridization with another wheat carrying the B genome. Recently, our most grown bread wheat evolved from cultivated tetraploid wheat and wild diploid species carrying the D genome (Bonjean et al. 2001, p. 7; Miedaner 2009, p. 11).

During the long wheat growing season in middle Europe, starting with sowing in September or October and ending with harvesting in July or August, a variety of diseases can reduce yield. Septoria tritici blotch (STB), caused by the fungus *Septoria tritici* (teleomorph *Mycosphaerella graminicola*), is one of the most important foliar diseases of wheat worldwide causing yield losses between 30 and 40 % (Eyal et al. 1987, p. 1). *S. tritici* infects both bread wheat and durum wheat. Infections on flag leaves cause most severe yield losses by limiting production of assimilates during the grain filling phase. Sexual as-

cospores and asexual pycnidiospores both germinate on the leaf (Palmer and Skinner 2002). The disease cycle of *S. tritici* (**Figure 1**) starts with windborne ascospores produced on stubble remaining in the fields (Shaw and Royle 1989). Throughout the winter and early spring, the spores are blown to wheat seedlings and establish primary infections. In spring, under conducive weather conditions with rainfall and high humidity, rain splashed pycnidiospores infect upper leaf layers. The fungus penetrates the leaf surface through stomata (Palmer and Skinner 2002). The latency period under our central European conditions ranges from 22 to 28 days depending on variety, temperature, and humidity. After successful infection, symptoms of STB occur as characteristic necroses bearing visible black pycnidia arranged parallel to leaf veins. Out of these pycnidia, new cycles of disease can develop several times during the growing season of wheat.

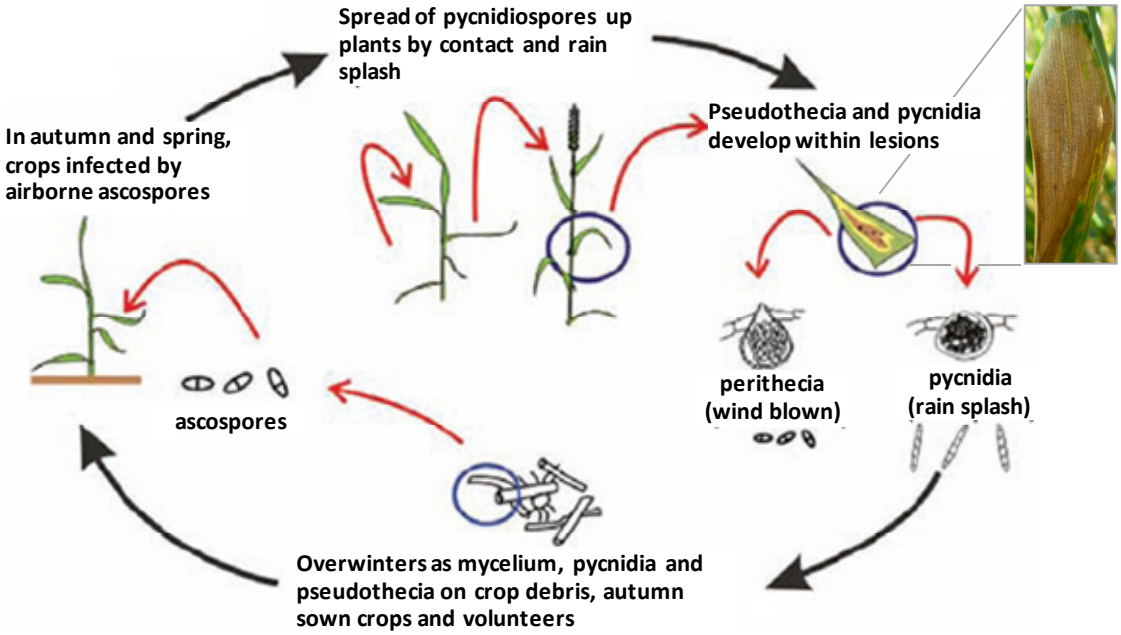


Figure 1: Life cycle of *Septoria tritici* (Source: http://www.hgca.com/cde, 7 Jan 2010)

Means of control of STB infections include crop rotation, soil tillage, fungicide application, and cultivation of resistant varieties. Profit-making wheat growers are forced to apply narrow crop rotations planting wheat crop after wheat crop under reduced tillage, where stubbles remain on the field. Thus, fungicide applications are routine to protect high yielding varieties against diseases. The most effective fungicides belong to the groups of strobilurins and azoles. However, strobilurins are no longer effective, due to recurring mutations in the pathogen population of *S. tritici* (Torriani et al. 2009). Most azole fungi-

cides continue to be effective against *S. tritici*, although decline in the efficiency of some azoles has been reported (Cools and Fraaije 2008). Consequently, cultivation of STB resistant varieties is a cost effective strategy of controlling the disease.

1.2 Resistance breeding and mapping

European wheat breeders consider *S. tritici* to be one of the major targets for resistance breeding (Arraiano and Brown 2006). There are two different genetic mechanisms for disease resistance: (i) qualitative, isolate-specific, vertical resistance based on major genes and (ii) quantitative, isolate non-specific, horizontal resistance, which is polygenic (Keller et al. 2000). In order to distinguish between these two types of resistance, a number of host genotypes (e.g. wheat varieties) are tested against a number of pathogen genotypes (e.g. fungal isolates) (Parlevliet 1977). Qualitative resistance is characterized by the interaction between varieties and isolates (Parlevliet and Zadoks 1977). This resistance is based on the successful interaction of the resistance gene of the plant and the avirulence gene of the host, according to Flor (1971) who formulated the gene-for-gene relationship. In contrast, there is also horizontal or quantitative resistance since the total non-environmental variance in levels of disease resistance is explained by the main effects (varieties and isolates) showing no genotype-by-isolate interactions. This type of resistance slows down the disease development by increasing latency period. In practical resistance breeding a combination of both types of resistance is often used. Major genes are easy to handle, whereas quantitative resistance is more complex and harder to select, but also thought to be more durable and, therefore, desirable in resistance breeding (Chartrain et al. 2004b).

In the pathosystem wheat/*S. tritici* we can find both qualitative, isolate-specific, vertical resistance depending on one major gene and quantitative, isolate non-specific, horizontal resistance with polygenic inheritance. Until now, thirteen major genes for resistance to STB, *Stb1* to *Stb12* and *Stb15*, have been mapped (Arraiano et al. 2001b; Brading et al. 2002; Adhikari et al. 2003; McCartney et al. 2003; Adhikari et al. 2004a; Adhikari et al. 2004b; Adhikari et al. 2004c; Chartrain et al. 2005a; Chartrain et al. 2005c; Arraiano et al. 2007) and only two studies detected quantitative-trait loci (QTL, Eriksen et al. 2003; Char-

train et al. 2004b). *Stb6* and *Stb15* are the most common resistance genes in European germplasm (Arraiano and Brown 2006). Isolate-specific resistance genes could be effective at seedling and adult-plant stage (Arraiano et al. 2001a) or just at seedling and not at adult-plant stage (Kema and van Silfhout 1997). Since quantitative resistance is sometimes only expressed in adult-plant stage, it is also described as adult-plant resistance (Keller et al. 2000).

Plant breeders have the choice whether they use isolate-specific resistance genes, quantitative resistance, or a combination of both. Thus, pyramiding effective major genes with closely linked diagnostic markers is one option, whereas selecting lines with good quantitative resistance in the absence of isolate-specific resistance is another strategy (Brown et al. 2001). The pathogen population of *S. tritici* shows high levels of genetic diversity on three different scales within and among (i) continents, (ii) wheat fields, and even (iii) one lesion on a wheat leaf (McDonald et al. 1999; Linde et al. 2002). Factors that contribute to the high genetic diversity are regional gene flow and frequent sexual recombination (Zhan et al. 2003). Thus, considering the high evolutionary potential of *S. tritici* (McDonald and Linde 2002) quantitative resistance seems more durable and, therefore, a more sustainable strategy in breeding wheat which is resistant to STB.

In order to search for loci controlling quantitative resistance (quantitative-trait loci, QTL), populations segregating for the resistance trait are used. QTL analysis includes construction of genetic maps and searching for association between resistance trait and polymorphic markers (Liu 1998, p. 375). Therefore, all individuals of the population are evaluated for resistance trait (phenotyping) and, in parallel, polymorphic genetic markers are generated (genotyping). Genetic maps are obtained by assignment and ordering of these markers to linkage groups, which correspond to one of the 21 wheat chromosomes (Keller et al. 2000, p. 132). Based on such genetic maps, the number, positions, and genetic effects of QTL can be determined by computer programs, e.g. PLABMQTL, a program for composite interval mapping of QTL (Utz and Melchinger 1996). QTL are defined as significant statistical association between genotypic values and phenotypic variability among the segregating population (Beavis 1998, p. 146). Until now, little has been known about inheritance of adult-plant resistance to STB. Only two studies reported about QTL

mapping in two populations limited in size and number of evaluated environments (Eriksen et al. 2003; Chartrain et al. 2004b), which is critical in QTL analysis (Melchinger et al. 2004; Schön et al. 2004). In this study, we used five biparental populations with higher numbers of progenies segregating for STB resistance to search for loci controlling the disease. Marker-assisted selection (MAS) of major QTL, explaining most of phenotypic variance, is the obvious application in resistance breeding (Asíns 2002).

QTL meta-analysis is state-of-the-art, after QTL mapping established a routine in genetic analysis of complex traits like quantitative disease resistance. Meta-analysis is defined as the integration of individual studies with a comparative map-based approach on three different levels: (i) different populations within the same crop inoculated with one pathogen (e.g. wheat/*Septoria*); (ii) one population of the same crop inoculated with different pathogens (e.g. wheat/*Fusarium*/*Septoria*/*Stagnospora*); (iii) populations of different crops inoculated with the same pathogen (e.g. wheat/maize/*Fusarium*). Recently published QTL meta-analyses (Liu et al. 2009; Löffler et al. 2009) looked for common QTL regions across wheat populations for one resistance trait using data given in the literature. Up to now, however, no studies exist using raw data of independent field experiments evaluated for resistance to several severe pathogens of wheat to reveal multiple-disease resistance QTL within mapping populations.

Wheat breeders consider resistance to STB to be an important trait. However, the challenge in breeding is to achieve most of the breeding goals in a balanced proportion. Successful varieties combine high yield and baking quality with several disease resistances. Thus, resistance to Fusarium head blight (FHB, caused by *Fusarium graminearum*) and Stagnospora glume blotch (SGB, caused by *Stagnospora nodorum*), causing yield losses and poor grain quality, is of relevance in wheat breeding. Resistance to FHB and SGB is quantitatively inherited and controlled by multiple genes (Schnurbusch et al. 2003; Paillard et al. 2004; Semagn et al. 2007; Uphaus et al. 2007; Holzapfel et al. 2008; Shankar et al. 2008; Bonin and Kolb 2009). QTL were carried out for resistance to FHB and SGB in mapping population Arina/Forno (Schnurbusch et al. 2003; Paillard et al. 2004) and for resistance to FHB in History/Rubens (Holzapfel et al. 2008). Evaluation of resistance to STB is missing in these segregating populations.

1.3 Aims of this study

In this study, we analyzed the genetic diversity of European wheat varieties after inoculation with different isolates of *S. tritici*. Segregating populations were used to detect chromosomal regions for quantitative adult-plant resistance of winter wheat to Septoria tritici blotch (STB). Furthermore, QTL meta-analysis was applied to reveal multiple-disease resistance QTL.

Firstly, we evaluated 24 winter wheat varieties after inoculation with four preselected isolates of *S. tritici* in multienvironmental field trials (test of isolates and varieties).
The objectives were to

(1) compare natural infection and inoculation,
(2) evaluate genotypic variation of adult-plant resistance to STB, and
(3) analyze genotype x environment (G x E) interaction.

Secondly, we mapped quantitative-trait loci (QTL) for STB resistance, heading date (HED), and plant height (PLH) in five wheat populations inoculated with *S. tritici* in field trials across four to six environments (mapping populations).
The objectives were to

(1) evaluate and analyze phenotypic data including STB severity, HED, and PLH,
(2) construct genetic linkage maps using AFLP, DArT, and SSR markers,
(3) determine number, positions, and genetic effects of QTL for evaluated traits.

Thirdly, raw data of four different field experiments including phenotypic evaluations for resistance to STB, Fusarium head blight (FHB), and Stagnospora glume blotch (SGB) in mapping population Arina/Forno, as well as for resistance to STB and FHB in History/Rubens were used to detect multiple-disease resistance QTL (QTL meta-analysis).
The objectives were to

(1) identify position, support interval, and genetic effects of meta QTL and
(2) investigate impact of QTL meta-analysis for applications in practical plant breeding programs.

2 MATERIAL AND METHODS

This study describes the genetic diversity between wheat varieties and the genetic variation within several populations in field trials after inoculation with *S. tritici*. Plant material and fungal isolates were obtained from cooperating partners. Field trials were conducted at four locations in North and South Germany. A combination of several marker technologies was used to identify genome regions for resistance. The closing biometrical analyses yield an estimation of variance components and genomic regions of interest for resistance breeding. Two experiments were conducted to test isolates and varieties and to analyze mapping populations.

2.1 Plant material

Test of varieties and isolates

Twenty-four European wheat varieties differing in resistance to Septoria tritici blotch (STB) were tested in field trials (**Table 1**). All are winter types and have been released as commercial varieties including parents of mapping populations.

Mapping populations

In total, we used five populations consisting of crosses of a resistant and a susceptible winter wheat variety. Four populations are recombinant inbred lines (RILs) and one is a doubled haploid (DH) population provided by cooperating partners in the CEREHEALTH project (**Table 2**). Two populations, History/Rubens and Arina/Forno, have already been published and were phenotyped for Fusarium head blight (FHB) (Paillard et al. 2004; Holzapfel et al. 2008) and Stagnospora glume blotch (SGB) (Schnurbusch et al. 2003), respectively. QTL meta-analysis makes it possible to detect common QTL regions across pathosystems.

Table 1: Wheat varieties in multi-environmental field trials 2007 to 2009

No.	Variety [1]	Origin	Breeder / Originator	Pedigree [3]
1	Ambition	Denmark	Abed Fonden	Ritmo / A0119.7
2	Apache	France	Nickerson (Limagrain)	Axial / NRPB-84-4233
3	**Arina**	Switzerland	Federal Research Station for Agronomy	Moisson // Can3842 / Heines VII
4	Atlantis	Germany	Saatzucht Schweiger	Disponent / Kronjuwel // Kanzler
5	**Biscay**	Germany	KWS LOCHOW	CPB 79 / Hussar
6	**Bussard**	Germany	KWS LOCHOW	Kranich / Maris-Huntsman // Monopol
7	Cliff	n.l. [2]	RAGT	Rialto / Torfrida // Brutus
8	Contra	Germany	Saatzucht Breun	Kronjuwel / Maris-Marksman
9	Dream	Germany	Saatzucht Schweiger	Disponent / Kronjuwel // Monopol / Orestis
10	Drifter	Germany	Nickerson (Limagrain)	Ronos / Estica
11	Flair	Germany	Saatzucht Schweiger	Ares / Marabu
12	**Florett**	Germany	RAGT	PBIS 95-82 / Cortez
13	**History**	Germany	Bayerische Pflanzenzuchtgesellschaft	Isidor / Kronjuwel // Huntsman / Götz / 3 / Granada / Huntsman // Diplomat / Kronjuwel
14	Lindos	Germany	Saatzucht Strube	W549-70 / Benno / Maris-Huntsmann // Kormoran / Kronjuwel
15	Lynx	UK	Cambridge Plant Breeders	Rendevous / Heaven
16	Meteor	Germany	SW Seed	Tarso / Contra // Hadm 91952-88
17	Piko	Germany	Nordsaat	CWW-3319.5 / 3 / Kraka // Maris-Huntsman / Frühgold
18	Robigus	UK	KWS UK	Z836 / Putch
19	**Rubens**	France	Verneuil-Recherche (Limagrain)	MD-286 / Pernel // Genial
20	Senat	Denmark	Sejet	Ritmo / Sj7830
21	Skalmeje	Germany	KWS LOCHOW	Greif / Pastiche // SB8681
22	Sobi	Germany	Saatzucht Breun	1553fl32 / 1730d53 // Transit
23	**Solitär**	Germany	Saatzucht Schweiger	Flair / Piko
24	**Tuareg**	Germany	Nordsaat	Kris / Dekan

[1] Parents of mapping populations in green (resistant) and red (susceptible)

[2] Not licensed

[3] Information of breeder or online: http://genbank.vurv.cz/wheat/pedigree/pedigree.asp http://www.sortinfo.dk/Sorter.asp (14. Dec. 2009)

Table 2: Details of the five wheat mapping populations

Population	Source [1]	Type [2]	Generation	No. of lines	No. of environments
1 **Florett**/Biscay	KWL	RIL	$F_{7:8}$	316	6
2 **Tuareg**/Biscay	KWL	RIL	$F_{7:8}$	269	5
3 **History**/Rubens	LfL	RIL	$F_{6:9}$	103	6
4 **Arina**/Forno	UZH	RIL	$F_{5:8}$	200	4
5 **Solitär**/Bussard	KWL	DH	-	81	4

[1] Mapping populations were provided by KWS LOCHOW GMBH (KWL), Bayerische Landesanstalt für Landwirtschaft (LfL), and Universität Zürich (UZH)

[2] RIL, recombinant inbred lines; DH, doubled haploid lines

2.2 Fungal isolates, inoculum production, and inoculation

Four *S. tritici* isolates comprised of BAZ 6/1/04, BAZ 8/8/04, and D 12/5 from Germany (provided by Julius-Kühn-Institut) as well as IPO 94269 from the Netherlands (provided by G. J. Kema) were chosen for the variety test both because they were virulent at seedling stage (**Table 3**, results with additional isolates are presented in supplement table **S 1**) and also because of differentiate parents of mapping populations at adult-plant stage (Risser 2007; Schilly 2009). This is important, because we want to focus on quantitative resistance in the field. All mapping populations except History/Rubens were inoculated with a mixture of two isolates (BAZ 6/1/04, BAZ 8/8/04) virulent to *Stb6* and *Stb15* (**Table 3**). History/Rubens was inoculated with the single isolate BAZ 6/1/04.

Table 3: Test of parents of mapping populations in seedling test with four international isolates for resistance to Septoria tritici blotch (% leaf necrosis)

Parents	*Septoria tritici* isolates [1]				Postulated
	IPO 323 [2]	IPO 88004	BAZ 6/1/04	BAZ 8/8/04	*Stb* genes
Arina	8	10	90	100	*Stb6, Stb15*
Biscay	80	100	100	90	-
Bussard	100	85	80	100	-
Florett	3	3	70	85	*Stb6, Stb15*
Forno	33	100	100	100	*Stb6*
History	80	88	100	95	-
Rubens	0	100	100	100	*Stb6*
Solitär	3	100	95	90	*Stb6*
Tuareg	0	3	58	85	*Stb6*
Mean	34	65	88	94	

[1] Seedling test by G. J. Kema, Wageningen, the Netherlands; % leaf necrosis on the primary seedling leaves (21 dpi)

[2] IPO 323 (avir. *Stb6*) and IPO 88004 (avir. *Stb15*) were provided by G. J. Kema, the Netherlands, BAZ 6/1/04 and BAZ 8/8/04 were provided by Julius-Kühn-Institut, Germany; the latter two isolates were also used in field trials

Inoculum was produced from sporulating cultures of *S. tritici*, grown on yeast malt agar (YMA) for 3 to 5 days under ultraviolet (UV) light for 16 h per day at 18°C and 8 h per night at 12°C. This starter culture was used to produce huge amounts of inoculum for field trials. Inoculum was prepared by inoculating 150 ml of liquid yeast-glucose medium (4 g yeast, 4 g malt and 4 g glucose in 1 l distilled water) in 300 ml Erlenmeyer flasks with fresh *S. tritici* spores from agar plates. Several flasks per isolate were incubated for 3 to 5 days in reciprocal shakers (175 rpm) under UV light like the agar plates. The resultant

spore suspensions were concentrated into 200 ml tubes using separating funnels. The concentrate was stored in freezers at - 20°C until 24 h before inoculation. At each location the inoculum was adjusted to a density of 5 x 10^6 spores/ml as determined by hemacytometer counts (Neubauer improved, depth 0.1 mm, 0.0025 mm², Laboroptik GmbH, Germany).

Inoculation was done after rainfall and during cloudy weather conditions so that moisture was retained on the leaf surface for several hours. All trials were inoculated once at growth stage BBCH 39 to 55 (Meier 2001), after late genotypes' flag leaves had been fully unrolled. The variety test and the populations were inoculated using a pneumatically driven hand sprayer and a tractor mounted sprayer, respectively (**Figure 2**).

Figure 2: Inoculation and field design of mapping populations

2.3 Field trials

Trials were conducted at four locations over three years in Germany (**Figure 3**). Freising (FRE, latitude 48.45°, longitude 11.72°, 448 m a.s.l., 7.5°C mean annual temperature, 775 mm mean annual precipitation), Stuttgart-Hohenheim (HOH, latitude 48.80°, longitude 9.20°; 400 m a.s.l., 8.5°C mean annual temperature, 685 mm mean annual precipitation) and Oberer Lindenhof (OLI, latitude 48.52°, longitude 9.05°, 700 m a.s.l., 6.6°C mean annual temperature, 960 mm mean annual precipitation) are located in South Germany, Wohlde (WHO, latitude 52.80°, longitude 9.98°, 80 m a.s.l., 8.8°C mean annual temperature, 753 mm mean annual precipitation) in North Germany.

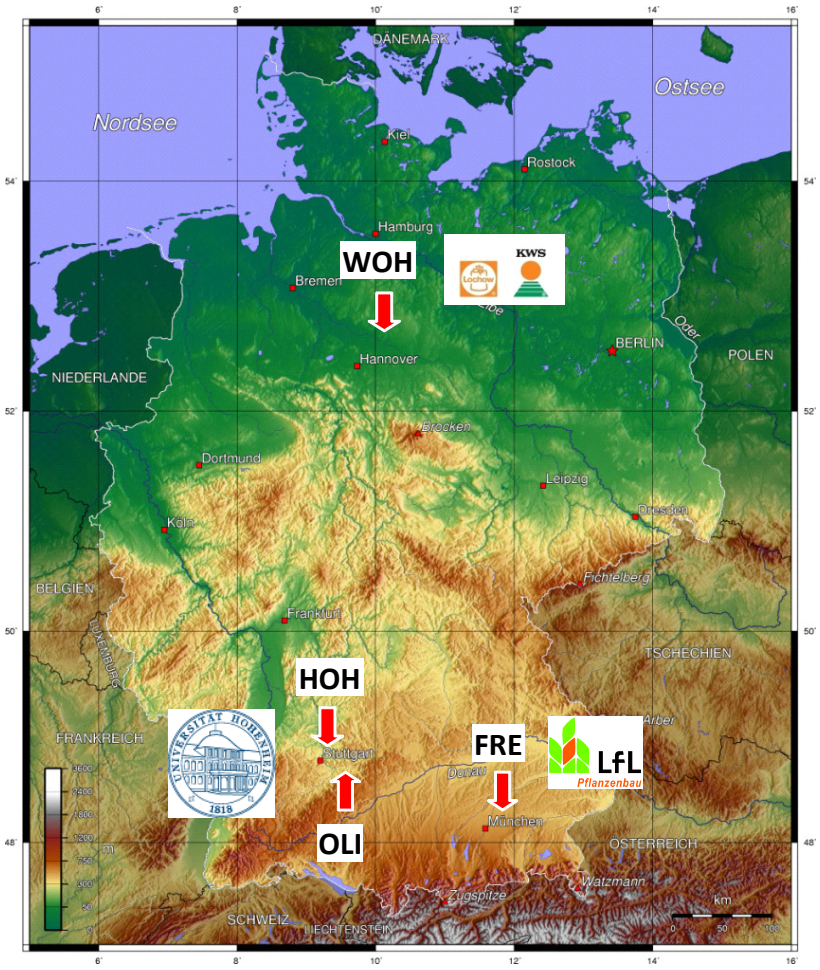


Figure 3: Location of field trials at Freising (FRE), Hohenheim (HOH), Oberer Lindenhof (OLI) and Wohlde (WHO) 2007 to 2009 (Source: http://www.mygeo.info/, 10 Oct 2008)

The variety test was sown in a split-plot design with four isolates and one untreated control as main-plot factor and the 24 varieties as sub-plot factor. Main plots, inoculated with different isolates, were separated from each other by double rows of triticale. The populations were grown together with five replicated entries of the parental lines as α-design with two replications at each location as well as the variety test (**Figure 2**). Each entry of both trials was sown in sets of two 1 m rows with approximately 40 to 60 kernels per row. Solitär/Bussard was tested in only one replication and one row, because of problems in seed production of DH lines.

In all field trials Septoria tritici blotch (STB), heading date (HED), and plant height (PLH) were evaluated plotwise. STB severity was visually scored plotwise as percent coverage with lesions bearing pycnidia. Flag leaves of double rows were assessed three times at an interval of four to seven days, starting from about 30 days after inoculation. The arithmetic mean of two scorings, representing middle and end of disease, was used in the follow-

ing analyses. HED was scored as days after 1st January when 50 % of spikes had emerged. PLH was measured from the soil surface to the middle of the spike on the main tillers.

Field trials were allocated across four locations within two years for mapping populations and test of isolates and varieties (**Table 4**). The test of isolates and varieties was phenotyped additionally at Freising and Hohenheim 2007 after inoculation with nine different *S. tritici* isolates (Risser 2007). In this study, the data of four selected isolates and the not inoculated variant evaluated across ten environments is presented. At every location the test of isolates and varieties was organized next to the mapping populations. Due to adverse weather conditions during and after inoculation in Northern Germany 2009 disease symptoms were inadequate to differentiate genotypes for STB. Therefore, STB was missing at Wohlde 2009. HED was missing at Freising 2007 and 2009 and at Hohenheim 2007.

Table 4: Allocation of field trials in 2008 and 2009 with the number of plots phenotyped at each environment

Field trial	Environment [1]								Total number of environments
	2008				2009				
	FRE	HOH	OLI	WOH	FRE	HOH	OLI	WOH	
Populations (α-lattice)									
Florett/Biscay	684	684	-	684	684	-	684	684	6
Tuareg/Biscay	576	-	576	576	576	-	-	576	5
History/Rubens	252	252	252	-	252	252	252	-	6
Arina/Forno	-	-	-	-	432	432	432	432	4
Solitär/Bussard [2]	-	-	-	-	108	108	108	108	4
Test of isolates and varieties (split-plot)	240	240	240	240	240	240	240	240	10 [3]
Total number of plots	1,752	1,176	1,068	1,500	2,292	1,032	1,716	2,040	

[1] Environment = year x location combination; locations were Freising (FRE), Hohenheim (HOH), Oberer Lindenhof (OLI), and Wohlde (WOH)

[2] Solitär/Bussard was phenotyped only one replication per location

[3] Test of varieties and isolates was phenotyped additionally at Fresing and Hohenheim 2007

In meta-analysis, raw data of initial studies (Schnurbusch et al. 2003; Paillard et al. 2004; Holzapfel et al. 2008) was reanalyzed for resistance traits of Arina/Forno (FHB and SGB) and History/Rubens (FHB), whereas STB, PLH, and HED were used from this study for both mapping populations. In Arina/Forno, Schnurbusch et al. (2003) scored twice the percentage of the infected glume area per spike after natural SGB infestation across five environments in Switzerland. The area under the disease progress curve (AUDPC) was calculated based on two SGB scorings per environment. In this study, we used the arithmetic mean of two SGB scorings according to our STB rating. Paillard et al. (2004) calculated

percentage of diseased spikelets after inoculation with *F. graminearum* across six environments in Switzerland. Two FHB scorings were chosen to calculate the AUDPC. As for SGB, we used the arithmetic mean of the two FHB scorings in our meta-analysis. Thus, all resistance traits are given in percentage of disease severity, comparable between experiments. In History/Rubens Holzapfel et al. (2008) the scored FHB severity averaged over five visual ratings as a percentage of infected spikelets per plot after spray inoculation with *F. culmorum* across five environments in Germany, which was used in meta-analysis.

2.4 Marker analysis and genetic mapping

Marker analysis

In order to construct genetic maps with broad coverage enabling QTL mapping, we used a combination of AFLP, DArT, and SSR markers. Because all markers were provided by cooperating partners or by companies, the reader is referred to further literature and information.

AFLP markers were provided by LfL Freising. The AFLP (amplified fragment length polymorphism) technique is a DNA fingerprinting technology applicable to linkage mapping without the need for prior sequence information. Vuylsteke et al. (2007) and Vos et al. (1995) described the technique in detail. The name of the AFLP markers consisted of the applied primer combination followed by the estimated fragment size in base pairs (see Holzapfel et al. 2008).

DArT markers are a product of Triticarte (http://www.triticarte.com.au). DArT is an abbreviation for **D**iversity **Ar**ray **T**echnology. In contrast to SNP and SSR markers, DArT detects single base changes and INDELs without relying on DNA sequence information. This array based technology enables high throughput entailing low costs. We used Triticarte service Wheat PstI(TaqI) v2.3 (2,500 markers) in Tuareg/Biscay and History/Rubens as well as v2.5 (5,000 markers) in Florett/Biscay and Bussard/Solitär. Akbari et al. (2006) described an integrated map for a cross between the wheat varieties Cranbrook and Halbred using RFLP, SSR, AFLP, STM, and DArT markers that was used as a reference map.

KWS LOCHOW GMBH (Dr. Viktor Korzun) provided three to five SSR (simple sequence repeats) anchor markers per chromosome in Florett/Biscay, Tuareg/Biscay, and Bussard/Solitär to facilitate assignment of linkage groups to chromosomes. SSR marker tech-

nique is frequently used in plant breeding, especially in genetic mapping and marker-assisted selection (Landjeva et al. 2007).

Genetic linkage mapping

Genetic linkage maps with AFLP, DArT, and SSR markers of Florett/Biscay, Tuareg/Biscay, and Bussard/Solitär were generated using JoinMap 3.0 (Van Ooijen and Voorrips 2001) assuming Haldane's mapping function (Haldane 1919). Markers were assigned to linkage groups at logarithm of odds (LOD) ≥ 3.0 with a maximum recombination fraction of 0.4. Because of clustering of markers, we decided to delete markers without additional information within one cM. Clustered DArT markers were deleted first, followed by AFLP. SSR markers remained in the map. In summary, we started with a larger set of polymorphic markers and ended with a subset of these as mapped markers used for QTL analysis. The maps of History/Rubens and Arina/Forno have been published (Paillard et al. 2003; Holzapfel et al. 2008) and were provided by the authors. Additionally, DArT markers were mapped in History/Rubens with an increase of genome coverage. The number of mapped markers ranged from 221 to 491 covering 1,314 to 3,305 cM of polymorphic regions (**Table 5**). The average interval distance was small (2.3 to 8.0) indicating high density maps, although differing in genome coverage.

Table 5: Summary of five wheat mapping populations used to construct genetic maps

Population	No. of lines for QTL mapping	Marker types	No. of marker		Polymorphic regions (cM)	Avg interval distance (cM) [1)]
			polymorphic	mapped		
Florett/Biscay	301	SSR, DArT, AFLP	609	221	1,341	2.3
Tuareg/Biscay	263	SSR, DArT, AFLP	384	262	1,326	3.6
History/Rubens [2)]	94	SSR, DArT, AFLP	939	491	2,361	2.6
Arina/Forno [3)]	200	SSR, RFLP	458	440	3,305	8.0
Bussard/Solitär	80	SSR, DArT	844	239	1,314	6.1

[1)] Average interval distance in cM

[2)] Holzapfel et al. 2008, additional DArT marker were integrated in the genetic linkage map

[3)] Paillard et al. 2003, modified

2.5 Data analysis

Analysis of variance (ANOVA)

Plot means were calculated and used for the statistical analysis of the field trials. Estimation of variance components for evaluated traits (STB, HED, PLH) in variety test and mapping populations was done using PLABSTAT (Utz 2001). STB disease severity was tested for assumptions. Residuals of multienvironmental variety test were normally distributed. Residuals of mapping populations did not follow normal distribution. Appropriate transformations were evaluated, but did not improve the normality of the data. Therefore, untransformed data were used for analysis of variance (ANOVA) and QTL analysis.

The following statistical model was used for split-plot design of test of varieties and isolates across environments considering genotype as random effect:

$$y_{ijkl} = \mu + E_i + I_j + G_k + (RE)_{li} + (IE)_{ij} + (GI)_{jk} + (GE)_{ik} + (GIE)_{ijk} + b_{ijl} + e_{ijkl}$$

μ = general mean

E_i = main effect of *i*th environment

I_j = main effect of *j*th isolate

G_k = main effect of *k*th genotype

R_l = main effect of *l*th replication

$(RE)_{li}$ = replication-by-environment interaction

$(IE)_{ij}$ = isolate-by-environment interaction

$(GI)_{jk}$ = genotype-by-isolate interaction

$(GE)_{ik}$ = genotype-by-environment interaction

$(GIE)_{ijk}$ = genotype-by-isolate-by-environment interaction

b_{ijl} = main-plot error

e_{ijkl} = sub-plot error

Post hoc comparisons between means of inoculated and not inoculated plots were performed using a Dunnett test. Multiple comparisons of genotypes were done with the Tukey-Kramer test using statistical software SAS 9.1.

In the α-lattice of mapping populations prior to ANOVA, adjusted means were calculated for genotypes in each environment. ANOVA across environments was performed using a general linear model with genotype and environment effects, considering genotype as

random. Entry-mean heritability (h^2) was calculated by PLABSTAT as the ratio of genotypic to phenotypic variance (Knapp et al. 1985).

Stability analysis across environments

A regression approach was used with the coefficient of regression (b_i) and the deviation mean square (MS_{dev}) as important parameters to describe environmental stability (Eberhart and Russell 1966; Becker and Léon 1988). The b_i value characterizes the specific response of genotypes to increasing epidemic pressure and the MS_{dev} value describes the contribution of a variety to G x E interaction (Miedaner and Flath 2007).

QTL analysis

QTL analysis was performed using composite interval mapping (CIM) with PLABMQTL (Utz and Melchinger 1996; Utz 2009 pers. comm.). For detection of QTL LOD threshold was set to 3.0. After QTL detection, critical LOD scores were determined for all traits in all populations based on 1,000 permutations (α = 10 %) as recommended by Churchill and Doerge (1994). Additionally, five-fold cross validation was applied to determine the magnitude of bias of phenotypic variance (R^2) explained by detected QTL. The entire data set (DS) is split into five genotypic subsamples. Means from four out of five subsamples serve as the estimation set (ES) for QTL detection, localization, and estimation of genetic effects. The remaining subset forms the test set (TS) in which predictions derived from ES are tested for their validity by correlating predicted and observed data. By permutating the respective subsets used for ES and TS, five different cross validation runs are possible (Utz et al. 2000). In this study, five-fold cross validation with 200 replicated runs was used. Detected QTL are presented with genome position, flanking markers, distance to next marker, confidence interval (CI), and normalized adjusted R^2 (nR^2_{adj}). The 95 % CI is calculated after Darvasi and Soller (1997). R^2 was adjusted (R^2_{adj}) to get more adequate estimation of explained phenotypic variance (Hospital et al. 1997), and was normalized (nR^2_{adj}) so that the sum across detected QTL is equal to model R^2_{adj} (see Zhu et al. 2004). In this study QTL was declared major if it explained more than 10 % of nR^2_{adj} (Draeger et al. 2007; Semagn et al. 2007). In the final simultaneous fit, the detected QTL and their positions were used to obtain estimates of additive effects. These effects were calculated for each envi-

ronment illustrating QTL-by-environment (QTL x E) interaction. QTL x E interactions were tested for significance by sequentially rejective Bonferroni F-test according to Bohn et al. (1996). Genetic maps and QTL positions were drawn using MapChart 2.1 (Voorrips 2002).

Meta QTL-analysis

Meta QTL-analysis was applied within History/Rubens and Arina/Forno across three pathogens: Fusarium head blight (FHB, caused by *Fusarium graminearum*), Stagnospora glume blotch (SGB, caused by *Stagnospora nodorum*), and STB. Schnurbusch et al. (2003), Paillard et al. (2004), and Holzapfel et al. (2008) provided phenotypic raw data of SGB (percentage of the infected glume area per spike) and FHB (percentage of diseased spikelets). In order to compare QTL effects between different experiments, AUDPC was not used for SGB and FHB in Arina/Forno, but rather the arithmetic mean of two scorings, as described previously. QTL mapping was conducted with PLABMQTL using equal QTL-mapping procedures for all three diseases to get comparable LOD curves and estimations of QTL effects. Meta-analysis was done by adding LOD scores of disease traits within each population. History/Rubens was analyzed for resistance to FHB and STB, Arina/Forno for resistance to FHB, SGB, and STB. In contrast to other meta QTL studies (Hanocq et al. 2007; Griffiths et al. 2009; Liu et al. 2009; Löffler et al. 2009; Mao et al. 2010) searching for common QTL regions of one or two trait(s) across populations using data from the literature, we looked for meta QTL within one population across different resistance traits using raw data of field evaluations. With this approach, overlapping QTL regions for multiple-disease resistance were detected. Because the detection of meta QTL depends on addition of LOD scores, all scores larger than six were declared as meta QTL. Also major QTL for one resistant trait with high LOD scores appeared as meta QTL. With focus on multiple-disease resistance, meta QTL were selected showing significant ($P < 0.01$) QTL effects across at least two resistance traits and with a LOD score ≥ 6.

3 RESULTS

3.1 Test of varieties and isolates

Comparison of inoculation and natural infection

We inoculated field trials with four isolates of *S. tritici* to ensure the development of disease symptoms in a total of nine environments. Additionally, one main plot was not inoculated to get information about natural infection each year (**Table 6**). The four isolates used in field trials showed the same level of aggressiveness with mean STB ratings > 30 % across 24 varieties and nine environments. The inoculated plots showed significantly higher mean STB ratings (Dunnets's test, P < 0.01) compared to the not inoculated plots.

Table 6: Comparison of mean STB rating of plots inoculated with four *S. tritici* isolates and not inoculated plots across 24 wheat varieties evaluated in nine environments

Variant	Mean STB rating (% flag leaf area infected)			Mean (N = 9)
	2007 (N = 2) [1)]	2008 (N = 4)	2009 (N = 3)	
***Septoria tritici* inoculated**				
BAZ 6/1/04	14	37	41	**33.6** **
BAZ 8/8/04	15	34	47	**34.2** **
D 12/5	25	28	45	**32.6** **
IPO 94269	16	34	42	**32.9** **
Not inoculated	4	25	31	**22.0**

[1)] N = number of environments

** Significance of comparison with not inoculated plots (Dunnett's test, P < 0.01)

Disease symptoms caused by natural infection were comparatively low in 2007 and were exceeded each time by inoculated plots (**Table 7**). Levels of disease severity increased the following years favoured by rainfall events after flag leaf emergence (BBCH 39) with leaf wetness and high humidity. The parallel rise of symptoms of both, the not inoculated and inoculated plots, indicates that natural infection contributed to the symptoms of inoculated plots. Correlation with the mean STB rating of inoculated plots over all environments with means of not inoculated *versus* inoculated plots in each year resulted in positive correlation coefficients (**Table 7**). While natural infection 2007 showed a moderate correlation of r = 0.46, correlation of inoculated plots with overall mean (r = 0.84) was considerably high. Inoculation is therefore a successful method to obtain infections and to

detect the full range of genotypic variance, even under conditions with low rates of natural infection.

Table 7: Mean percentage of not inoculated *versus* inoculated flag leaf area with Septoria tritici blotch of 24 wheat varieties across three experimental years including nine environments

Variety [1)]	2007 (N = 2) [2)]		2008 (N = 4)		2009 (N = 3)	
	Not inoculated	Inoculated	Not inoculated	Inoculated	Not inoculated	Inoculated
Ambition	3	6	18	21	9	14
Apache	4	42	31	47	53	80
Arina	3	13	12	18	19	31
Atlantis	3	13	18	27	20	33
Biscay	3	19	56	58	58	72
Bussard	7	40	40	54	55	71
Cliff	6	11	44	55	52	64
Contra	12	38	41	61	57	77
Dream	3	22	17	26	20	36
Drifter	5	20	48	59	48	67
Flair	2	14	11	15	26	32
Florett	2	8	13	15	12	16
History	3	14	7	14	11	16
Lindos	3	34	27	45	53	75
Lynx	3	12	38	44	41	50
Meteor	2	25	15	35	31	51
Piko	1	13	14	24	26	42
Robigus	2	8	8	19	24	34
Rubens	3	34	51	62	62	85
Senat	8	8	18	20	11	19
Skalmeje	5	15	18	25	14	33
Sobi	1	6	19	21	11	21
Solitär	1	5	5	9	7	9
Tuareg	2	5	29	26	14	20
Mean	**4**	**18**	**25**	**33**	**31**	**44**
Correl. [3)]	**0.46**	**0.84**	**0.85**	**0.97**	**0.98**	**0.99**

[1)] Parents of mapping populations in green (resistant) and red (susceptible)

[2)] Means across locations per year (N = number of locations)

[3)] Correlation with the overall mean STB rating (% flag leaf area infected) of inoculated plots across nine environments

Analysis of variance and correlations

In analysis of variance for HED, PLH, and STB, there were highly significant ($P < 0.01$) differences between genotypes for all traits evaluated (**Table 8**, **Table 9**). For STB, genotypes

had the highest variance component followed by the environment (**Table 8**). A significant G x E interaction was detected, but it was considerably smaller than the effect of genotypes. The effects of isolates and genotype-by-isolate interaction were negative and with low impact. Thus, the following values of STB rating are shown as means across isolates.

Table 8: Estimates of variance components for heading date, plant height, and STB rating across 24 European wheat varieties

Source	Heading date (days) [1)]	Plant height (cm) [2)]	STB rating (%) [3)]
Environment (E)	79.7 **	28.7 **	119.2 **
Isolate (I)	na [4)]	na	- [5)]
I x E	na	na	39.2 **
Genotype (G)	6.0 **	91.4 **	314.8 **
G x I	na	na	4.1 **
G x E	1.4 **	9.6 **	168.6 **
G x I x E	na	na	18.8 **
Error	0.7	6.9	76.4

[1)] Days from 1st January, means across seven environments and two isolates

[2)] Means across ten environments and two isolates

[3)] Septoria-tritici blotch (% flag leaf area infected), means across nine environments and four isolates

[4)] na = not applicable

[5)] Negative variance component

** F-test significant at $P < 0.01$

Heading date ranged from 148.6 (Apache) to 158.5 (Solitär) days from first January with mean of 155.3 days evaluated across seven environments (**Table 9**). The tallest variety was Arina (104.2 cm) and the shortest Lynx (70.1 cm) measured in ten environments. There was no correlation between STB disease severity and PLH ($r = -0.04$). The correlation with HED was moderate ($r = -0.53$), just depending on the two very early and susceptible varieties Apache and Rubens and the very late resistant variety Solitär (**Table 9**).

Table 9: Means of 24 wheat varieties for heading date, plant height, and Septoria tritici blotch (STB) over three years (2007 to 2009) sorted by STB

Variety [1]	Heading date (days) [2]	Plant height (cm) [3]	STB rating (%) [4]	Multiple comparisons [5]
Solitaer	**158.5**	99.3	8.1	a
Florett	154.0	81.2	13.6	b
History	157.3	94.3	14.6	bc
Ambition	157.0	82.9	15.6	bcd
Senat	157.9	74.1	17.1	cde
Sobi	154.0	90.6	17.8	def
Tuareg	157.1	83.2	19.1	efg
Flair	154.7	93.7	20.7	fg
Arina	153.4	**104.2**	21.3	g
Robigus	156.0	75.6	21.5	g
Skalmeje	155.4	86.5	25.5	h
Atlantis	155.1	95.4	26.0	h
Piko	157.9	92.1	27.6	h
Dream	157.5	100.3	28.5	h
Meteor	155.0	85.1	37.8	i
Lynx	157.4	**70.1**	38.8	i
Cliff	156.1	77.7	48.3	j
Lindos	153.6	96.4	52.4	k
Drifter	155.4	95.7	53.2	k
Biscay	156.2	78.7	54.1	kl
Apache	**148.6**	79.9	56.6	l
Bussard	154.5	103.6	56.7	l
Contra	154.3	90.2	61.5	m
Rubens	149.3	85.4	63.4	m
Mean	155.3	88.2	33.3	-
$LSD_{5\%}$ [6]	0.44	1.16	2.86	-
Heritability (%)	99.58	99.81	99.66	-

[1] Parents of mapping populations in green (resistant) and red (susceptible)

[2] Days from 1st January, means across seven environments, min. and max. in bold

[3] Means across ten environments, min. and max. in bold

[4] Septoria tritici blotch (% flag leaf area infected), means across nine environments

[5] Varieties with the same letter are not significantly different in STB (Tukey-Kramer test, $\alpha = 5\%$)

[6] Least significant difference for pairwise comparisons ($\alpha = 5\%$)

Entry-mean heritabilities for HED, PLH, and STB were high (> 0.99) indicating a high level of accuracy of field trials. Multiple testing identified genotypes with different letters as significantly different from each other in STB (Tukey-Kramer test, α = 5 %). Solitär is the most resistant variety followed by Florett and History with mean STB ratings < 15 %. In contrast, the most susceptible wheat varieties were Rubens and Contra with mean STB ratings > 60 %. The chosen parents of mapping populations are a good representation of the genetic variance for STB including the most resistant and susceptible varieties (**Table 9**).

G x E interaction and environmental stability

G x E interaction on resistance traits is well known. It is also true for STB in wheat varieties as shown for the parents of mapping populations in detail (**Table 10**). For instance, Tuareg showed the same level of resistance as Solitär in 2007, but was highly susceptible at Freising 2008. Another example is Arina, which was resistant at Freising and Hohenheim 2008, but moderately susceptible at Oberer Lindenhof and Wohlde in 2008 as well as at Freising, Hohenheim, and Oberer Lindenhof in 2009.

Table 10: Means of Septoria tritici blotch (% flag leaf area infected) of parents of mapping populations and overall means across 24 varieties and across four isolates in nine environments (= location x year combination): Freising (FRE), Hohenheim (HOH), Oberer Lindenhof (OLI), and Wohlde (WOH) 2007, 2008, and 2009

Parents	Environment									Mean
	2007		2008				2009			
	FRE	HOH	FRE	HOH	OLI	WOH	FRE	HOH	OLI	(N = 9)
Solitär	7	2	3	4	8	21	12	9	7	**8**
Florett	13	3	26	7	6	20	18	14	16	**14**
History	22	5	6	10	24	15	20	16	13	**15**
Tuareg	8	2	60	13	10	20	18	26	14	**19**
Arina	14	12	9	6	25	31	48	23	23	**21**
Biscay	18	20	76	73	58	26	51	77	88	**54**
Bussard	32	49	48	57	79	33	58	80	75	**57**
Rubens	46	22	68	76	79	24	73	88	94	**63**
Mean (N = 24)	**19**	**17**	**41**	**28**	**40**	**24**	**40**	**46**	**44**	

Scale Septoria tritici blotch (% flag leaf area infected)	
0 - 10	light green
11 - 20	light yellow
21 - 50	gold
> 50%	brown

Environmental stability of varieties is a major breeding goal to reduce G x E interaction. A regression approach was used with the coefficient of regression (b_i) and the deviation mean square (MS_{dev}) to describe environmental stability. The smaller both values are, the higher is the stability and the level of resistance to STB (**Figure 4**). With focus on the parents of mapping populations, indicated in the figure, Solitär, History, and Florett were the best genotypes. Even under very high epidemic pressure they remained resistant in most environments, with mean disease severities of 8 to 15 % (**Table 10**). Tuareg and Biscay showed the highest MS_{dev} indicating low stability. While Tuareg is rather resistant, Biscay is one of the most susceptible varieties in the trials. Arina, Rubens, and Bussard showed a moderate to high MS_{dev} with increasing susceptibility from 2007 to 2009 starting from different levels of resistance (**Table 10**). Hence, Solitär, History, and Florett are examples for a stable resistant variety, whereas Tuareg is rather resistant but not stable and Biscay is susceptible and not stable.

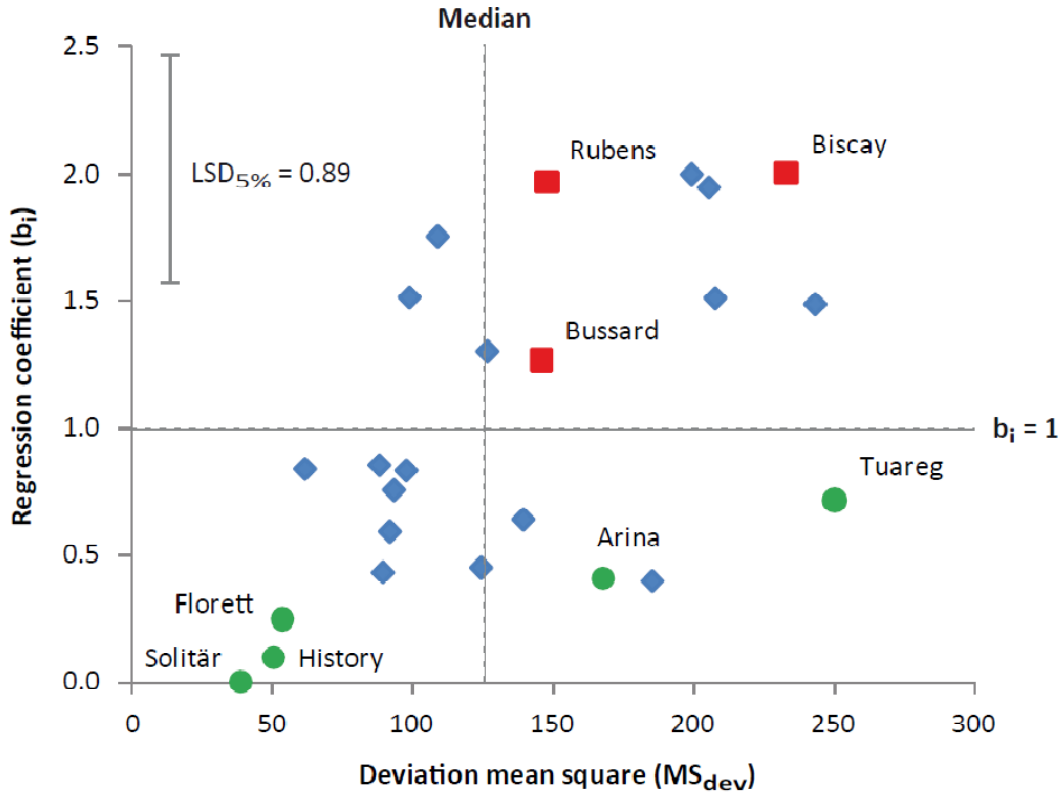


Figure 4: The relation of deviation mean square (MS_{dev}) and regression coefficient (b_i) of 24 wheat varieties in stability analysis of Septoria tritici blotch severity across nine environments; resistant and susceptible parents of mapping populations are named and indicated by green circles and red squares, respectively; $LSD_{5\%}$ = least significant difference for pairwise comparisons of b_i ($P < 0.05$)

3.2 Mapping populations

Phenotypic data

Field trials revealed differentiation for STB, HED, and PLH between the parents and within all five mapping populations (**Table 11**). The populations differed on average by 12 % for STB, 5 days for HED, and 21 cm for PLH. Differentiation was significant for all traits. Heritabilities (h^2) ranged from 0.69 to 0.87 for STB, the only exception was Tuareg/Biscay (h^2 = 0.38). For HED (h^2 = 078 to 0.93) and PLH (h^2 = 0.92 to 0.98), heritabilities were high throughout mapping populations.

Table 11: Means of parents (P 1 and P 2) and populations for Septoria tritici blotch, heading date, and plant height evaluated in three to six environments 2008 and 2009, $LSD_{5\%}$ = Least significant difference ($P < 0.05$), h^2 = heritability ± standard error

Traits	# E [1)]	Parents			Population				
Populations		P 1	P 2	Mean	Mean	Min	Max	$LSD_{5\%}$	h^2
Septoria tritici blotch (%)									
Florett/Biscay	5	25.1	59.4	42.3	36.1	7.9	73.1	17.27	0.73 ± 0.02
Tuareg/Biscay	4	24.9	48.7	36.8	36.6	14.1	65.2	19.15	0.38 ± 0.06
History/Rubens	6	13.2	87.1	50.2	47.7	13.2	88.2	16.90	0.87 ± 0.02
Arina/Forno	3	23.7	55.0	39.4	42.9	18.4	87.4	20.85	0.73 ± 0.03
Solitär/Bussard	3	13.1	81.9	47.5	35.5	8.7	73.8	19.73	0.69 ± 0.06
Heading date (days)									
Florett/Biscay	5	152.7	154.6	153.6	154.8	149.8	160.1	1.09	0.93 ± 0.01
Tuareg/Biscay	4	157.9	156.7	157.3	156.7	153.6	160.4	1.55	0.88 ± 0.01
History/Rubens	5	159.3	151.0	155.1	155.4	151.5	160.4	1.31	0.93 ± 0.01
Arina/Forno	3	152.0	150.6	151.3	151.3	145.9	155.7	1.45	0.88 ± 0.02
Solitär/Bussard	3	157.2	153.9	155.5	156.8	153.0	160.7	2.42	0.78 ± 0.04
Plant height (cm)									
Florett/Biscay	6	79.1	76.8	77.9	77.3	62.0	97.8	2.81	0.95 ± 0.00
Tuareg/Biscay	5	81.2	77.9	79.6	79.1	64.6	95.9	2.94	0.95 ± 0.01
History/Rubens	6	93.0	81.8	87.4	95.0	64.4	116.4	4.94	0.98 ± 0.00
Arina/Forno	4	99.7	88.5	94.1	92.6	72.1	113.7	4.18	0.96 ± 0.00
Solitär/Bussard	4	94.9	100.9	97.9	97.3	76.3	117.0	5.68	0.92 ± 0.01

[1)] Number of environments; each population together with parents was evaluated in different sets of environments

All correlations between STB and HED as well as between STB and PLH are negative and moderate in most cases (**Table 12**), although significant ($P < 0.01$, $P < 0.05$). Two populations, Florett/Biscay and Tuareg/Biscay, were fixed for the reduced height (rht) allele at *Rht-D1* locus, and two populations, Arina/Forno and Bussard/Solitär, for the tall allele.

History/Rubens population is segregating at *Rht-D1* locus (**Figure 5**) and shows a considerably higher correlation between STB and PLH (r = -0.55).

Table 12: Correlation of Septoria tritici blotch with heading date and plant height in five wheat populations with different alleles at *Rht-D1* locus assessed in three to six environments 2008 and 2009

Population	*Rht-D1* locus [1)]	Coefficients of correlation	
		Heading date (days)	Plant height (cm)
Florett/Biscay	fixed for *Rht-D1b*	-0.19 **	-0.13 *
Tuareg/Biscay	fixed for *Rht-D1b*	-0.18 **	-0.20 **
History/Rubens	*segregating*	-0.30 **	-0.55 **
Arina/Forno	fixed for *Rht-D1a*	-0.23 **	-0.45 **
Bussard/Solitär	fixed for *Rht-D1a*	-0.33 **	-0.26 *

[1)] *Rht-D1b* = reduced height allele, *Rht-D1a* = tall allele

* Significant (P < 0.05)

** Significant (P < 0.01)

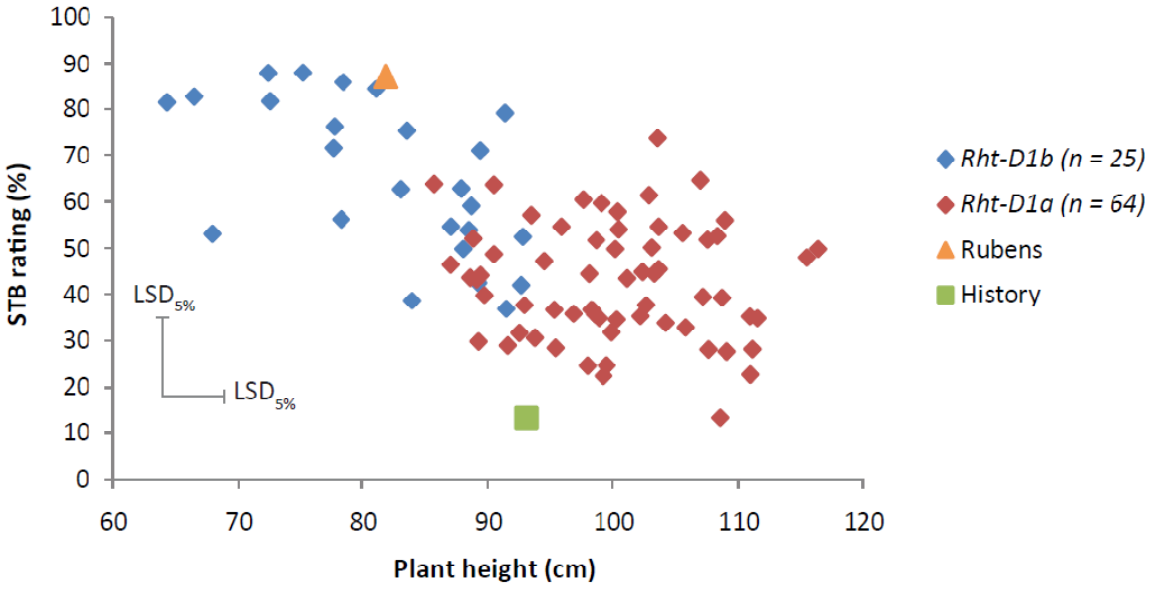


Figure 5: Relationship between mean STB rating and plant height for progeny of the population History/Rubens, separated into subpopulations homozygous for either the *Rht-D1b* semi-dwarf allele or *Rht-D1a* wild-type allele across six environments, n = number of progeny in the respective class

ANOVA for STB revealed highly significant (P < 0.01) variance components of genotype, environment, and G x E interaction (**Table 13**). Also, for PLH and HED these variance components were significant (P < 0.01). Tuareg/Biscay population had the smallest genotypic

variance of all populations with an extremely high G x E interaction. For the other populations G x E interaction was similar or smaller than genotypic variance.

Table 13: Estimates of variance components for Septoria tritici blotch, heading date, and plant height of five wheat populations evaluated in three to six environments

Trait/Source	Environment (E)		Genotype (G)		G x E		Error	
Population	DF	Var.cp. [1)]	DF	Var.cp.	DF	Var.cp.	DF	Var.cp.
Septoria tritici blotch (%)								
Florett/Biscay	4	172.6 **	315	104.3 **	1,260	143.8 **	1,525	49.8
Tuareg/Biscay	3	151.7 **	268	28.7 **	804	140.9 **	1,012	49.5
History/Rubens	5	118.0 **	102	255.9 **	508	158.5 **	544	63.4
Arina/Forno	2	33.4 **	197	149.6 **	394	110.1 **	543	58.5
Bussard/Solitär	2	1.3	80	111.1 **	160	149.8 [2)]	-	-
Heading date (days)								
Florett/Biscay	4	70.4 **	315	2.0 **	1,260	0.3 **	1,525	0.4
Tuareg/Biscay	3	84.7 **	268	2.3 **	804	0.5 **	1,012	0.7
History/Rubens	4	90.7 **	102	3.1 **	406	0.7 **	453	0.4
Arina/Forno	2	163.5 **	197	1.9 **	394	0.4 **	543	0.4
Bussard/Solitär	2	161.4 **	80	2.7 **	160	2.3 [2)]	-	-
Plant height (cm)								
Florett/Biscay	5	19.8 **	315	20.1 **	1,575	2.6 **	1,830	3.5
Tuareg/Biscay	4	36.6 **	268	20.1 **	1,072	2.0 **	1,012	3.6
History/Rubens	5	14.7 **	102	128.7 **	508	13.0 **	544	6.0
Arina/Forno	3	34.0 **	197	54.7 **	591	4.6 **	724	4.5
Bussard/Solitär	3	91.3 **	80	46.9 **	240	16.6 [2)]	-	-

[1)] DF = degress of freedom; Var.cp. = variance component

[2)] Error included in G x E because only one replication

** F-test significant at P < 0.01

The five mapping populations showed a wide and continuous distribution of mean STB severity averaged across three to six environments in field trials (**Figure 6**). Parental lines were located on either end of the distribution. History/Rubens population showed the widest range whereas Tuareg/Biscay had a smaller variation.

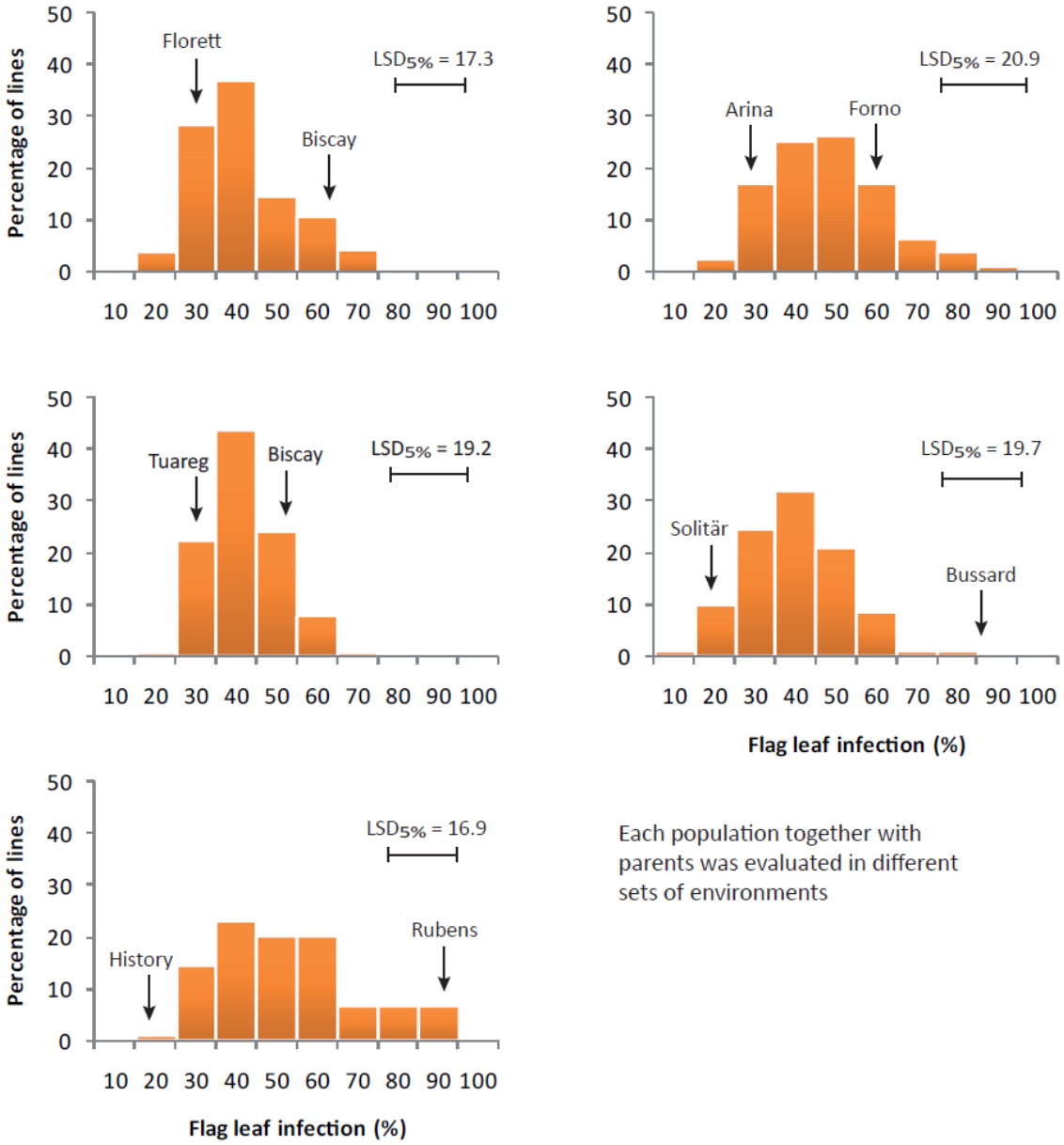


Figure 6: Histograms showing the results for Septoria tritici blotch (% flag leaf area infected) in five wheat populations from field trials (means across three to six environments)

Genetic linkage mapping and genetic similarity

A combination of SSR, DArT, AFLP, and RFLP markers were used to construct genetic maps in five wheat populations (**Table 5**). Total number of linkage groups varied among mapping populations with 22 in Florett/Biscay, 20 in Tuareg/Biscay, 35 in History/Rubens, 26 in Arina/Forno, and 22 in Bussard/Solitär. Each of the linkage groups could be assigned to one of the chromosomes. Except Tuareg/Biscay, all 21 wheat chromosomes were represented by linkage groups (**Table 14**). In Tuareg/Biscay chromosomes 3D and 6D were

missing. The number of markers per chromosome ranged from 3 to 42 (A genome), 6 to 41 (B genome), and 2 to 32 (D genome). Generally, the D genome was less represented by markers than A and B genome.

Table 14: Number of mapped SSR, AFLP, RFLP, and DArT markers on chromosomes and genomes in five wheat populations

Population		Chromosome									Genome coverage	
	Genome	1	2	3	4	5	6	7	Total	%		cM
Florett/Biscay	A	16	23	13	7	6	9	23	97	43.9		583
	B	17	11	10	6	11	10	7	72	32.6		427
	D	10	18	2	5	10	3	4	52	23.5		331
											Sum	1,341
Tuareg/Biscay	A	11	3	26	7	3	16	9	75	28.6		402
	B	24	19	26	7	12	19	21	128	48.9		598
	D	12	19	-	5	13	-	10	59	22.5		327
											Sum	1,327
History/Rubens	A	22	25	19	31	17	25	38	177	36.0		755
	B	40	39	36	18	26	22	41	222	45.2		1,010
	D	12	14	9	11	18	10	18	92	18.7		597
											Sum	2,361
Arina/Forno	A	15	27	21	18	25	15	42	163	37.5		1,221
	B	23	27	25	10	23	20	28	156	35.9		973
	D	14	14	22	6	16	12	32	116	26.7		1,112
											Sum	3,305
Bussard/Solitär	A	17	11	15	13	18	6	11	91	38.6		411
	B	17	17	17	6	14	8	19	98	41.5		576
	D	5	8	4	2	16	6	6	47	19.9		328
											Sum	1,314

In order to assess the level of genetic diversity among parents of mapping populations, the genetic similarity (*GS*) was estimated using 221 preselected SSR markers (Korzun 2009, pers. comm.). The dendrogram obtained from cluster analysis using the software NTSYS PC2.0 resulted in separation among varieties according to their *GS* (**Figure 7**). Estimates of *GS* ranged from 0.58 for History and Rubens to 0.73 for Tuareg and Biscay.

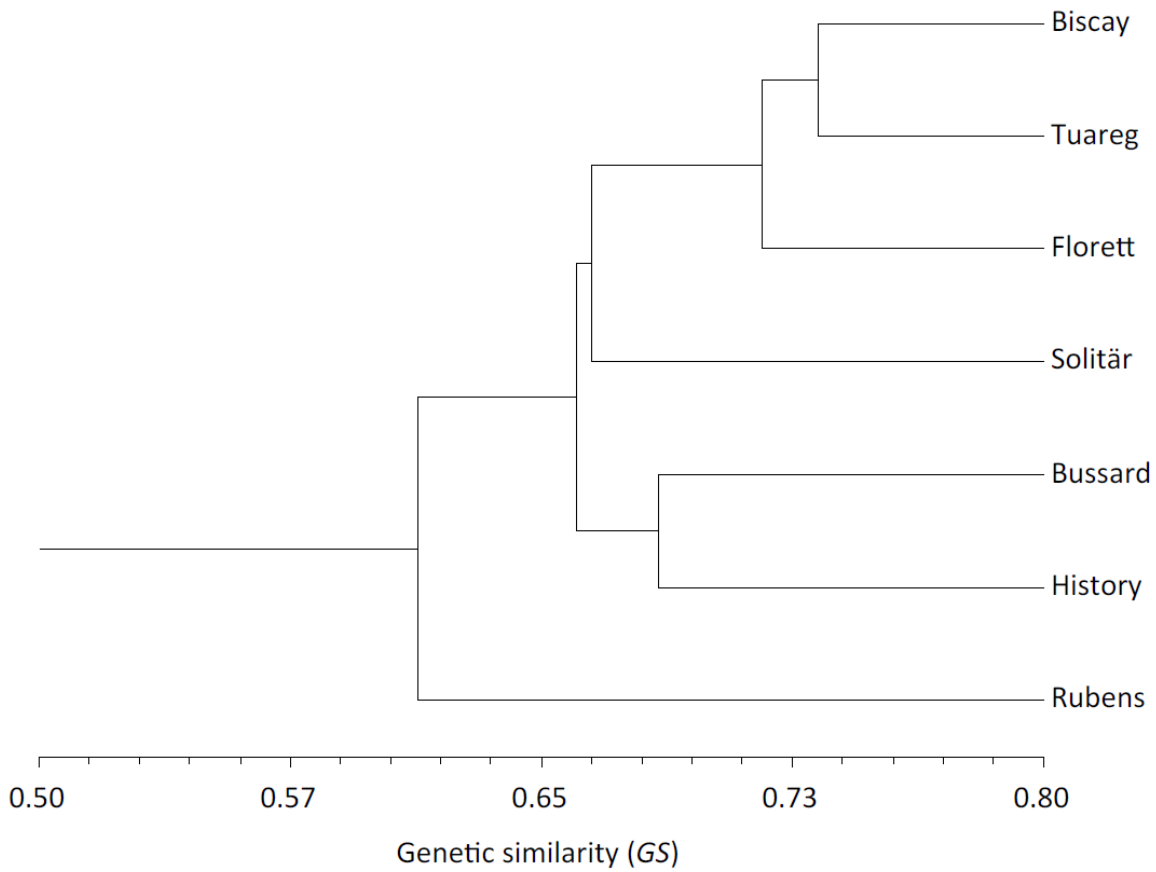


Figure 7: Dendrogram of the parents of mapping populations revealed by cluster analysis based on genetic similarity (*GS*) estimates calculated from a set of 221 SSR markers (Korzun 2009 pers. comm.)

QTL analysis

In total, one to nine, zero to nine, and four to eleven QTL were detected for STB, HED, and PLH, respectively, across five wheat populations using composite interval mapping (CIM) with a LOD threshold of 3.0 (**Table 15**). Critical LOD scores based on 1,000 permutations ranged from 3.0 to 7.7 among traits and mapping populations, with the exception of History/Rubens which had critical LOD scores ranging from 15.5 to 15.8 among traits. One to two major QTL (nR^2_{adj} ≥ 10 %) for resistance to STB were detected per population, all derived from the resistant parent. Altogether, resistance QTL explained 14 to 55 % of the total phenotypic variance (total R^2_{adj}). In most populations the susceptible parent also contributed resistance QTL. Astonishingly, in the Bussard/Solitär population only one QTL for STB was detected.

Table 15: Summary of QTL detected by composite interval mapping for all traits evaluated in field trials across three to six environments in five mapping populations

Traits Populations	Critical LOD α = 10 % [1]	No. of QTL detected LOD > 3.0	No. of major QTL nR^2_{adj} ≥ 10 % [2]	Total R^2_{adj} all QTL (%) [3]
Septoria tritici blotch (%)				
Florett/Biscay	3.2	9 (**5**) [4]	2 (**2**)	54.9
Tuareg/Biscay	3.1	6 (**5**)	2 (**2**)	51.3
History/Rubens	15.8	5 (**5**)	2 (**2**)	54.8
Arina/Forno	4.5	5 (**3**)	1 (**1**)	33.2
Bussard/**Solitär**	7.5	1 (**1**)	1 (**1**)	14.1
Heading date (days)				
Florett/Biscay	3.2	9	1	38.8
Tuareg/Biscay	3.0	6	1	43.8
History/Rubens	15.8	5	3	50.0
Arina/Forno	4.4	2	1	16.5
Bussard/Solitär	7.7	0	-	-
Plant height (cm)				
Florett/Biscay	3.2	4	0	25.3
Tuareg/Biscay	3.1	8	2	69.5
History/Rubens	15.5	5	2	68.4
Arina/Forno	4.4	11	1	60.0
Bussard/Solitär	7.5	1	0	8.4

[1] Critical LOD scores (α = 10 %) based on 1,000 permutations

[2] Number of detected QTL, each explaining ≥ 10 % of normalized adjusted phenotypic variance (nR^2_{adj})

[3] Total adjusted phenotypic variance explained by all detected QTL

[4] Number of QTL derived from resistant parent in brackets (**bold**)

QTL for STB resistance are all located on different chromosomes, except of History/Rubens chromosome 5B where two QTL were found (**Table 16**). The confidence intervals (CI) of these two QTL on chromosome 5B do not overlap, indicating independency. The distance of QTL to next flanking marker ranged from 0 to 4 cM across all populations. Small distances between marker and QTL resulted from high-density genetic linkage maps. Comparisons of QTL positions between populations were not possible because of missing common markers across genetic maps (**S 5**).

The majority of resistance QTL showed QTL x E interactions. The frequency of significant ($P < 0.01$) QTL x E interaction for STB resistance (69 %) is much higher than for PLH (24 %) or HED (36 %) (supplement tables **S 2** and **S 3**). Major QTL on chromosomes 5B (History/Rubens) and 7A (Bussard/Solitär) showed no QTL x E interactions.

Table 16: Localisation of QTL with LOD > 3.0 for resistance to Septoria tritici blotch (% flag leaf area infected) in five wheat populations (means across environments); major QTL explaining ≥ 10 % of phenotypic variance are highlighted

No.	Chrom.	Pos.	Flanking marker [1]		d [2]	CI [3]	nR^2_{adj} [4]	QTL x E [5]
		(cM)	left	right	(cM)	(cM)	(%)	
Florett/Biscay (N = 301)								
1	1A	44	**Xwmc0312**	XwPt-7030	0	27- 56	3.0	**
2	1B	78	XwPt-0260	**Xwmc0419**	0	68- 88	2.9	**
3	2B	4	**XP2553-222**	Xwmc0344	4	0- 10	4.8	**
4	3B	60	XP2553-237	**Xstb10**	1	57- 61	12.6	**
5	4B	6	XP1754-147	**XwPt-8092**	1	2- 10	9.3	**
6	5B	2	Xgwm0408	**XwPt-4577**	0	0- 19	2.3	**
7	6D	16	Xgwm0469	**Xcfd0013**	0	13- 16	11.9	**
8	7A	36	**Xbarc0108**	Xwmc0009	0	30- 42	5.1	**
9	7D	2	**XwPt-7842**	XwPt-7368	2	0- 13	3.0	
						Total R^2_{adj} (%)	**54.9** [6]	
Tuareg/Biscay (N = 263)								
1	1A [ns 7)]	44	Xwmc0024	**XwPt-3904**	0	29- 59	3.1	**
2	4A	6	**XwPt-5434**	Xwmc0219	0	0- 13	5.8	
3	4B	22	Xwmc0471	**Xwmc0238**	0	7- 29	17.3	**
4	4D	2	Xcfd0071	**Xgwm0129**	0	0- 12	5.2	
5	6B	24	**XwPt-6286**	XP1459-119	0	18- 30	11.8	**
6	7B	4	**Xwmc0517**	XP2255-118	0	0- 14	8.0	**
						Total R^2_{adj} (%)	**51.3**	
History/Rubens (N = 94)								
1	4D	4	***Rht-D1***	Xbarc0105	0	0- 9	21.2	**
2	5B [ns]	34	XwPt-4996	**Xgwm0274**	0	29- 39	10.4	
3	5B [ns]	68	Xbarc0142	**XP7152-196**	0	57- 79	9.8	**
4	6B [ns]	16	**XP7162-180**	XP7256-485	0	11- 21	7.1	**
5	7B [ns]	24	Xgwm0263	**XP6653-115**	0	19- 29	6.2	
						Total R^2_{adj} (%)	**54.8**	

Table 16: Continued

No.	Chrom.	Pos.	Flanking marker [1]		d [2]	CI [3]	nR^2_{adj} [4]	QTL x E [5]
		(cM)	left	right	(cM)	(cM)	(%)	
Arina/Forno (N = 200)								
1	2B	140	Xpsr0540	**Xcfd0276**	0	133- 147	7.0	**
2	3B	112	Xcfab2134	**Xgwm0131**	2	96- 128	10.4	**
3	5B	44	Xwmc0473	**Xpsr0574**	0	37- 51	4.9	
4	6D	106	Xcfd0019	**Xgdm0014**	2	99- 113	6.9	
5	7B [ns]	148	XksuD2	**Xgwm0146**	0	130- 157	4.1	**
						Total R^2_{adj} (%)	**33.2**	
Bussard/Solitär (N = 81)								
1	7A [ns]	38	**Xwmc0790**	XwPt-8067	1	23- 53	14.1	
						Total R^2_{adj} (%)	**14.1**	

[1] Closest marker in bold

[2] Distance in cM to the next flanking marker

[3] 95 % confidence interval after Darvasi and Soller (1997)

[4] Normalized partial phenotypic variance explained by detected QTL

[5] QTL-by-environment interaction tested for significance (sequentially rejective Bonferroni F-test)

[6] Adjusted phenotypic variance explained by detected QTL (final simultanous fit) across environments

[7] LOD > 3.0 but not significant (ns) according to critical LOD score after 1,000 permutations (α = 10 %)

** F-Test significant at P < 0.01

The most effective QTL was detected in History/Rubens explaining 21 % nR^2_{adj}. This QTL resides at the *Rht-D1* locus conferring reduced plant height. The tall allele has a significant higher resistance, than the short allele (**Figure 5**).

QTL x E interaction was frequently detected for STB resistance. This is illustrated in detail with the additive effects of resistance QTL at each location for Florett/Biscay, to give just one example (**Table 17**, for the other populations, see supplement table **S 4**). With focus on the two major QTL, they had a quite small additive effect at Freising and Wohlde 2008, but a very high impact at Hohenheim 2008 and Oberer Lindenhof 2009. In the series analysis, the resistant alleles of the two major QTL reduced STB rating by 5.2 and 5.8 % flag leaf infection. Both parents, susceptible and resistant, were donors of resistance alleles, although the susceptible Biscay contributed only QTL with small effects. This is also true for Tuareg/Biscay and Arina/Forno, whereas in History/Rubens and Bussard/Solitär only the resistant parent is donor of resistance alleles (**S 4**).

Table 17: Additive effects of resistance alleles illustrating QTL-by-environment interaction for Septoria tritici blotch (% flag leaf area infected) in the population Florett/Biscay at five environments; major QTL explaining ≥ 10 % of phenotypic variance are highlighted

QTL designation	Donor of resistance	Additive effect of resistance allele (%) [1]					
		2008			2009		Series
		FRE	HOH	WOH	FRE	OLI	
Florett/Biscay (N = 301)							
QStb.lsa_fb-1A	Florett	-4.1	-3.3	-0.1	-1.0	-3.2	**-2.3**
QStb.lsa_fb-1B	Biscay	-3.2	-3.9	-0.5	-0.9	-4.6	**-2.4**
QStb.lsa_fb-2B	Biscay	-1.1	-4.4	-1.0	-3.6	-4.4	**-2.9**
QStb.lsa_fb-3B	Florett	-2.0	-12.9	-0.8	-6.2	-7.4	**-5.8**
QStb.lsa_fb-4B	Florett	-1.1	-7.6	-0.7	-3.5	-9.2	**-4.5**
QStb.lsa_fb-5B	Biscay	-2.5	-4.9	-0.6	-0.6	-3.5	**-2.1**
QStb.lsa_fb-6D	Florett	-3.4	-10.2	-0.7	-2.5	-9.6	**-5.2**
QStb.lsa_fb-7A	Florett	-6.2	-3.0	-1.1	-2.2	-2.5	**-3.1**
QStb.lsa_fb-7D	Biscay	-2.7	-3.8	-0.7	-1.6	-2.8	**-2.2**
	Total R^2_{adj} (%) [2]	**15.7**	**49.8**	**16.4**	**40.0**	**42.7**	**54.9**

[1] Estimated additive effects (less % flag leaf infection) in final simultaneous fit of the resistance allele at the locations Freising (FRE), Hohenheim (HOH), Oberer Lindenhof (OLI), Wohlde (WOH), across two years (2008, 2009) and in the series

[2] Adjusted phenotypic variance explained by detected QTL in final simultanous fit in each environment and in the series

Five-fold cross validation revealed lower number of QTL detected in estimation set (ES) compared to data set (DS) in three cases (**Table 18**). In addition, the R^2_{adj} decreased from DS to ES and even more in the test set (TS). Factors influencing power, precision, and accuracy (Beavis 1998, p. 150) of QTL mapping are population size, heritability, genome coverage, and method of QTL analysis. There is a tendency that all parameters are higher in larger populations like Florett/Biscay and Tuareg/Biscay, than in smaller populations like History/Rubens and Bussard/Solitär. It is not the case for entry-mean heritabilities in these five wheat populations.

Table 18: Results of five-fold cross validation with 200 replicated runs in five wheat populations for resistance QTL against Septoria tritici blotch

Population	No. of lines	Heritability	No. of QTL		R^2_{adj} (%) [3]		
			DS [1]	ES [2]	DS	ES	TS
Florett/Biscay	301	0.73	9	6.3	54.9	48.5	38.3
Tuareg/Biscay	263	0.38	6	6.4	51.3	52.2	40.2
History/Rubens	94	0.87	5	3.1	54.3	47.4	23.6
Arina/Forno	200	0.73	5	3.7	33.2	30.2	14.2
Bussard/Solitär	81	0.69	1	1.1	14.1	14.0	4.3

[1] DS, data set; ES, estimation set; TS, test set

[2] Number of QTL in ES calculated as the mean

[3] Adjusted phenotypic variance explained by all detected QTL in each set

In plant breeding, the size of QTL effects is crucial to apply marker-assisted selection. Florett/Biscay was divided in four subpopulations, each carrying different alleles of the two major QTL (*QStb.lsa_fb-3B* and *-6D*), to demonstrate the size of QTL effects (**Figure 8**). The first subpopulation carried the susceptible allele (S) at both resistance loci with a mean STB rating of 46 %. The second subpopulation carried the resistant allele (R) at 3B locus and the S allele at 6D locus. In the third subpopulation, the allele situation was vice versa. Mean STB rating was reduced by 10 % in both subpopulations. The fourth subpopulation carried the resistant allele at both loci and reduced STB rating by 20 %, compared to the susceptible allele situation. The most resistant progeny were even better than the resistant parent Florett as indicated by green dashed line (**Figure 8**). In conclusion, combining major QTL in wheat breeding seems promising.

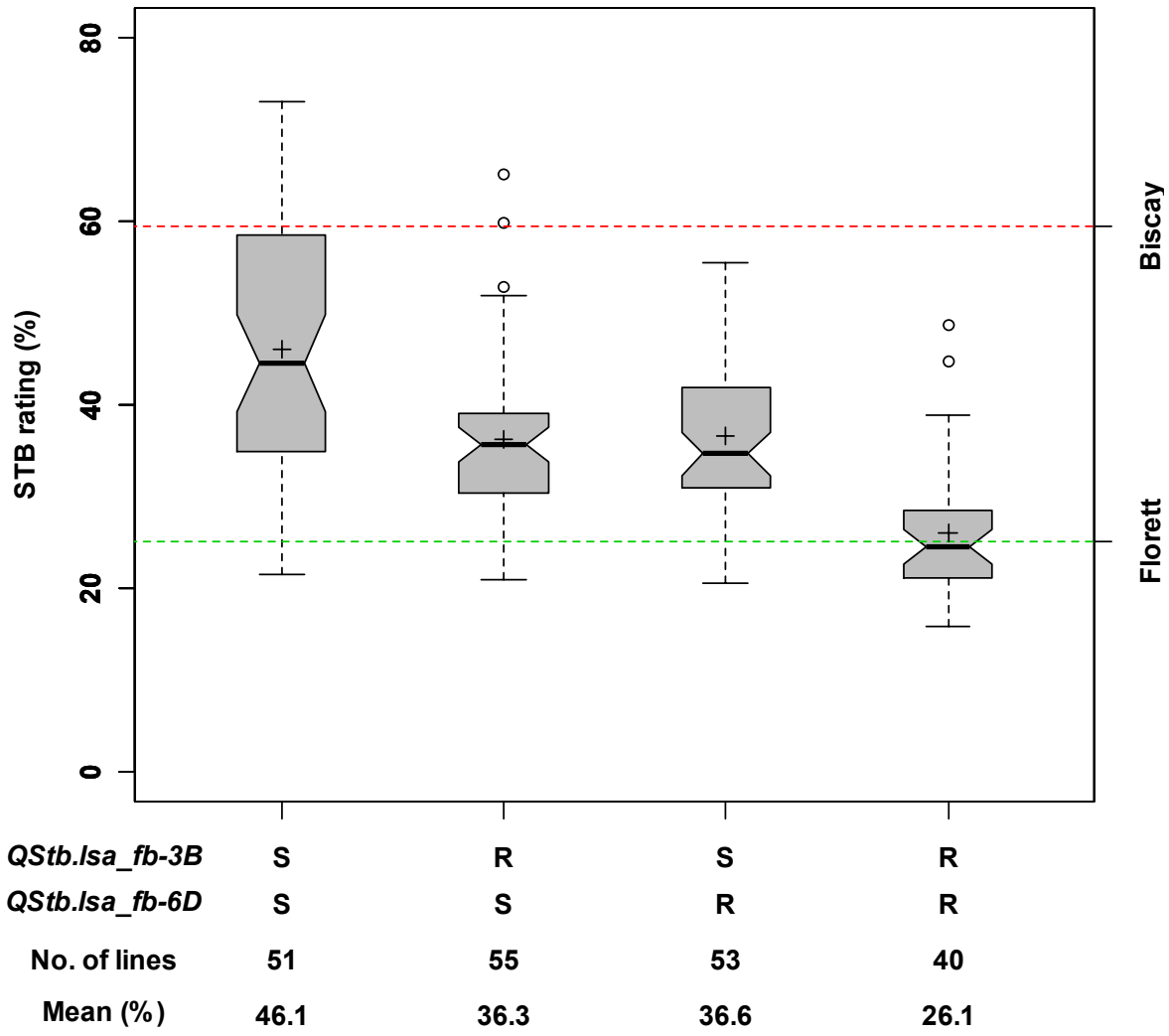


Figure 8: Notched boxplots for subpopulations of Florett/Biscay illustrating four different allele situations at two major QTL loci (*QStb.lsa_fb-3B, -6D*) for resistance to Septoria tritici blotch (S = susceptible allele present, R = resistant allele present); if notches do not overlap, medians are different according to McGill et al. (1978); horizontal line within boxes = median, + = mean, o = outliers, red and green dashed line indicate mean STB rating of susceptible and resistant parent, respectively, across environments

Altogether, 26 QTL for STB resistance were mapped across 13 chromosomes, each explaining 2.3 to 21.2 % of normalized adjusted phenotypic variance (**Table 19**). Additive effects of detected resistance QTL ranged from 1.5 to 10.2 % reduced flag leaf infection among the five mapping populations. Most QTL were mapped on the B genome with four QTL located on chromosome 5B.

Table 19: Distribution of QTL for resistance to Septoria tritici blotch in five wheat populations across chromosomes and genomes; major QTL explaining ≥ 10 % of normalized partial phenotypic variance are highlighted

Genome	Chromosome													
Population	1		2		3		4		5		6		7	
	nR^2_{adj} [1)]	Effect [2)]	nR^2_{adj}	Effect	nR^2_{adj}	Effect	nR^2_{adj}	Effect	nR^2_{adj}	Effect	nR^2_{adj}	Effect	nR^2_{adj}	Effect
A genome														
Florett/Biscay	3.0	-2.3	-	-	-	-	-	-	-	-	-	-	5.1	-3.1
Tuareg/Biscay	3.1	-1.5	-	-	-	-	5.8	-2.2	-	-	-	-	-	-
History/Rubens	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Arina/Forno	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Bussard/Solitär	-	-	-	-	-	-	-	-	-	-	-	-	14.1	-5.5
B genome														
Florett/Biscay	2.9	-2.4	4.8	-2.9	12.6	-5.8	9.3	-4.5	2.3	-2.1	-	-	-	-
Tuareg/Biscay	-	-	-	-	-	-	17.3	-4.0	-	-	11.8	-3.3	8.0	-2.6
History/Rubens	-	-	-	-	-	-	-	-	10.4	-5.6	7.1	-4.7	6.2	-4.2
									9.8	-5.5				
Arina/Forno	-	-	7.0	-4.3	10.4	-5.6	-	-	4.9	-3.5	-	-	4.1	-3.1
Bussard/Solitär	-	-	-	-	-	-	-	-	-	-	-	-	-	-
D genome														
Florett/Biscay	-	-	-	-	-	-	-	-	-	-	11.9	-5.2	3.0	-2.2
Tuareg/Biscay	-	-	-	-	-	-	5.2	-1.9	-	-	-	-	-	-
History/Rubens	-	-	-	-	-	-	21.2	-10.2	-	-	-	-	-	-
Arina/Forno	-	-	-	-	-	-	-	-	-	-	6.9	-4.5	-	-
Bussard/Solitär	-	-	-	-	-	-	-	-	-	-	-	-	-	-

[1)] Normalized partial phenotypic variance (%) explained by resistance QTL

[2)] Additive effect of resistance allele (less % flag leaf infection)

3.3 QTL meta-analysis

QTL meta-analysis was carried out in two wheat populations: Arina/Forno and History/Rubens. Raw data of different experiments was used for initial QTL analysis. In Arina/Forno, seven and three QTL explaining 48.0 and 34.3 % of phenotypic variance (R^2_{adj}) were detected for FHB and SGB resistance, respectively (**Table 20**). In History/Rubens, six QTL for FHB resistance were mapped explaining 57.8 % of R^2_{adj}. Additionally, STB, HED, and PLH of this study was used in meta-analysis.

Table 20: Summary of initial QTL analysis of Arina/Forno and History/Rubens with number of environments (No. env.), years, and heritabilities (h^2) for evaluated traits in field trials and total adjusted phenotypic variance (Total R^2_{adj}) explained by detected QTL with LOD > 3.0 used in meta-anylsis across pathosystems within mapping populations

Population (size) Trait (Abbreviation) (data source)	No. env. (years)	h^2	No. of QTL (LOD > 3.0)	Total R^2_{adj} (%)
Arina/Forno (N = 200)				
Septoria tritici blotch (STB) (1)	3 (1)	0.73	5	33.2
Fusarium head blight (FHB) (2)	6 (3)	0.92	7	48.0
Stagnospora glume blotch (SGB) (3)	5 (2)	0.81	3	34.3
Heading date (HED) (1)	3 (1)	0.88	2	16.5
Plant height (PLH) (1)	4 (1)	0.96	11	60.0
History/Rubens (N = 94)				
Septoria tritici blotch (STB) (1)	6 (2)	0.87	5	54.8
Fusarium head blight (FHB) (4)	5 (2)	0.93	6	57.8
Heading date (HED) (1)	5 (2)	0.93	5	50.0
Plant height (PLH) (1)	6 (2)	0.98	5	68.4

1) Data of this study

2) Paillard et al. (2004)

3) Schnurbusch et al. (2003)

4) Holzapfel et al. (2008)

LOD scores of initial QTL analysis for disease resistance were added to look for common QTL regions across pathosystems within mapping populations. In detail, LOD scores for STB, FHB, and SGB resistance in Arina/Forno as well as for STB and FHB resistance in History/Rubens were added to determine localization, position, flanking markers, support interval (SI), and LOD scores of meta QTL. In total, 12 and 19 genome positions were detected with LOD ≥ 6.0 across three and two pathosystems in Arina/Forno and History/Rubens, respectively. Out of these, meta QTL for multiple-disease resistance were selected showing significant ($P < 0.01$) QTL effects across at least two resistance traits. With PLABMQTL, eight meta QTL for multiple-disease resistance were detected conforming the selection criteria. Meta QTL were located on chromosomes 3B, 4B, 5B, and 6D in Arina/Forno, and on chromosomes 2B, 4D, 5B, and 7B in History/Rubens (**Table 21**). LOD scores ranged from 6.1 to 22.7 and from 7.0 to 48.7.

Table 21: Localization of meta QTL with LOD ≥ 6 for multiple-disease resistance in wheat populations Arina/Forno and History/Rubens across Septoria tritici blotch (STB), Fusarium head blight (FHB), and Stagnospora glume blotch (SGB) and across STB and FHB, respectively

Chrom.	Pos.	Flanking marker [1]		d [2]	SI [3]	LOD [4]
	(cM)	left	right	(cM)	(cM)	
Arina/Forno (N = 200)						
3B	120	**Xgwm0131**	Xgwm0383	5	106- 130	6.1
4B	16	**Xpsr0914**	Xglk0335	1	10- 22	12.0
5B	70	Xgwm0639	**Xpsr0120**	1	68- 72	22.7
6D	106	Xcfd0019.b	**Xgdm0014**	2	100- 110	10.9
History/Rubens (N = 94)						
2B	140	**XwPt-0694**	XP7056-648	2	138- 144	18.1
4D	4	***Rht-D1***	Xbarc0105	0	2- 6	48.7
5B	68	Xbarc0142	**XP7152-196**	0	64- 72	7.0
7B	24	Xgwm0263	**XP6653-115**	1	22- 28	30.6

[1] Closest marker in bold

[2] Distance in cM to the next flanking marker

[3] Support interval of meta QTL with a LOD fall off of 1.0 expressed as position on the chromosome

[4] Added LOD score across analyzed resistance traits: in Arina/Forno LOD scores of resistance to Septoria tritici blotch (STB), Fusarium head blight (FHB), and Stagnospora glume blotch (SGB) were added; in History/Rubens LOD scores of resistance to STB and FHB were added; LOD scores were calculated by composite interval mapping using PlabMQTL

The most effective meta QTL was on chromosome 4D in History/Rubens closely linked to *Rht-D1*. The resistance allele, coming from History, reduces disease severity by 9.8 % for STB and 6.3 % for FHB explaining 47 and 60 % of partial R^2 in QTL meta-analysis (**Table 22**). All resistance alleles at one meta QTL come from the same parent indicated by the same donor of resistance allele across pathogens.

Table 22: Partial R^2 (pR^2) and effects of meta QTL for multiple-disease resistance in wheat populations Arina/Forno and History/Rubens across Septoria tritici blotch (STB), Fusarium head blight (FHB), and Stagnospora glume blotch (SGB) and across STB and FHB, respectively

Chrom.	Pos.	STB			FHB			SGB		
	(cM)	pR^2	Effect [1)]	Donor [2)]	pR^2	Effect	Donor	pR^2	Effect	Donor
Arina/Forno (N = 200)										
3B	120	15.9	-5.9 **	A	8.2	-3.1 **	A	1.8	-0.8	A
4B	16	4.1	-2.5 **	F	0.0	-0.2	F	13.0	-2.1 **	F
5B	70	0.8	-1.2	F	12.9	-3.8 **	F	7.8	-1.7 **	F
6D	106	3.9	-3.3 **	A	5.5	-3.0 **	A	1.1	-0.8	A
History/Rubens (N = 94)										
2B	140	11.6	-3.7 **	R	9.9	-1.7 **	R	-	-	-
4D	4	47.2	-9.8 **	H	60.3	-6.3 **	H	-	-	-
5B	68	27.2	-6.1 **	H	12.8	-1.9 **	H	-	-	-
7B	24	23.9	-5.4 **	H	30.0	-3.1 **	H	-	-	-

[1)] Additive effects (less % infection) of meta QTL for STB, FHB, and SGB; all numbers reflect better resistance

[2)] Donor of resistance allele coming from Arina (A), Forno (F), History (H), Rubens (R)

** F-test significant at $P < 0.01$

The LOD curves are plotted to illustrate meta QTL for multiple-disease resistance in Arina/Forno (**Figure 9**) and History/Rubens (**Figure 10**). LOD curves for PLH and HED are given in comparison to the resistance traits. Influence of PLH and HED to meta QTL is crucial on chromosome 5B in Arina/Forno and chromosomes 2B, 4D, and 7B in History/Rubens indicated by high LOD scores of these traits within support interval of meta QTL.

In addition, significant QTL from initial QTL mapping experiments are labeled at the LOD peak. Initial QTL of Arina/Forno for STB, SGB, and FHB resistance on chromosomes 3B, 4B, 5B, and 6D and of History/Rubens for STB and FHB resistance on chromosomes 4D, 5B, and 7B were confirmed.

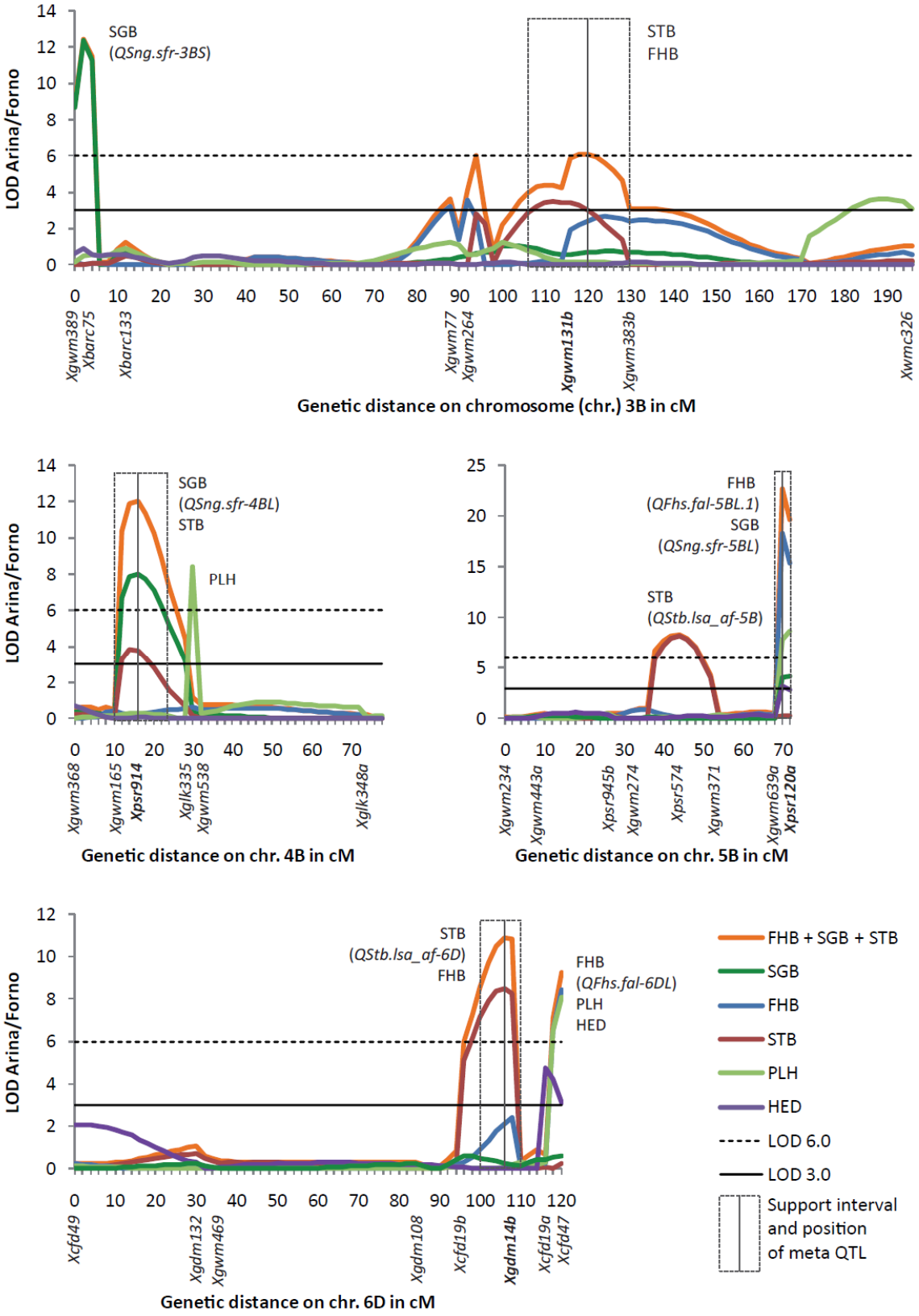


Figure 9: Illustration of meta QTL on chromosomes 3B, 4B, 5B, and 6D in wheat population Arina/Forno for multiple-disease resistance to Fusarium head blight (FHB), Stagnospora glume blotch (SGB), and Septoria tritici blotch (STB); LOD curves for plant height (PLH) and heading date (HED) are given in comparison to resistance traits; rectangles with line insight show support intervals and positions of meta QTL; initial QTL in brackets

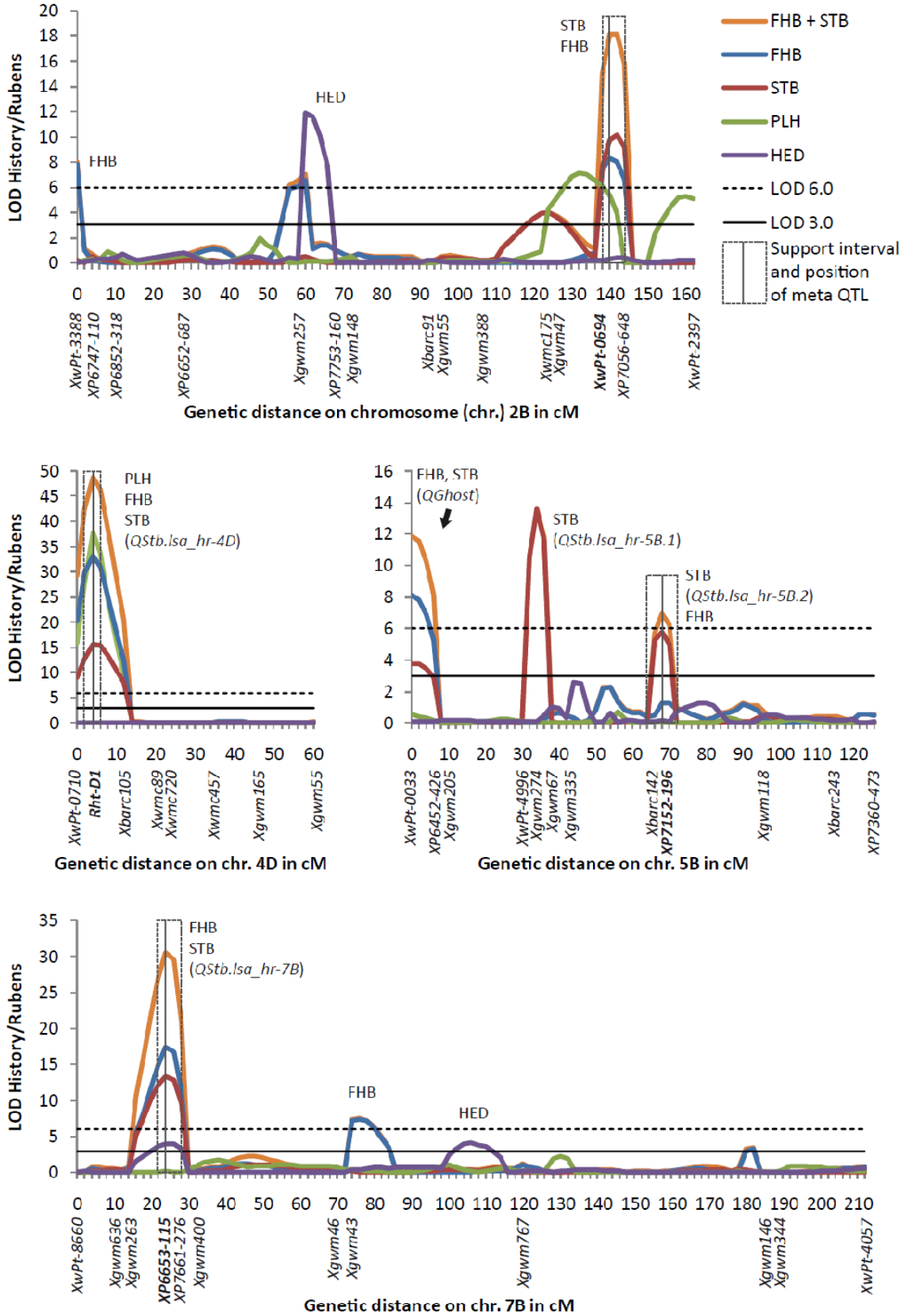


Figure 10: Illustration of meta QTL on chromosomes 2B, 4D, 5B, and 7B in wheat population History/Rubens for multiple-disease resistance to Fusarium head blight (FHB) and Septoria tritici blotch (STB); LOD curves for plant height (PLH) and heading date (HED) are given in comparison to resistance traits; rectangles with line insight show support intervals and positions of meta QTL; initial QTL in brackets

4 DISCUSSION

4.1 Phenotypic evaluations

Field trials

The basis for all studies were multienvironmental field trials organised as split-plot design for varieties and isolates, and as α-lattice design for phenotyping mapping populations. All trials were inoculated with *S. tritici* isolates. Weather conditions during and after inoculation are critical. A long period of leaf moisture is required to get successful infections (Shaw 1991; Shaw and Royle 1993), which are essential to differentiate varieties and populations for resistance to STB. In 2009, rainfall was missing during and after inoculation at Wohlde in Northern Germany and consequently no infections with *S. tritici* occurred despite inoculation. Although, natural infection at Freising and Hohenheim 2007 was low, inoculation was successful due to high relative humidity (> 80 %) and cloudy weather conditions during and after inoculation. For all trials, the inoculation date was adapted to local weather forecast with rainfall events most likely, after flag leaves had been fully unrolled (BBCH 39 to 55). In other field trials (Kema and van Silfhout 1997; Arama et al. 1999; Brown et al. 2001; Eriksen et al. 2003), irrigation was applied to provide favourable relative humidity in evaluated plots, which is recommendable for all field trials with regard to resistance breeding against *S. tritici*.

Heritabilities for STB were high for both trials, the test of isolates and varieties ($h^2 > 0.99$) as well as the mapping populations ($h^2 = 0.69$ to 0.87) indicating high accuracy of field trials. The only exception was population Tuareg/Biscay, where genotypic variation was much smaller than the effect of environment and G x E interaction. In comparison, Rosielle and Brown (1979) reported heritabilities ranging from 0.57 to 0.68 for STB, 0.56 to 0.88 for HED, and 0.41 to 0.49 for PLH in F_2 plants of three spring wheat populations. Field trials were conducted at only one location across two years. Other studies, concerning fungal disease resistance, reported heritabilities ranging from 0.35 to 0.93 for Fusarium head blight (Buerstmayr et al. 2000; Paillard et al. 2004; Semagn et al. 2007; Voss et al. 2008; Bonin and Kolb 2009), from 0.34 to 0.85 for Stagnospora glume blotch (Schnurbusch et al. 2003; Uphaus et al. 2007; Shankar et al. 2008), and from 0 to 0.98 for STB (Van Ginkel and Scharen 1987; Eriksen et al. 2003).

We can conclude that resistance breeding needs highly accurate inoculation methods applicable for large-scale field trials in multiple environments. Our method presented here is sound under conducive weather conditions during and after inoculation. An irrigation system is useful to ensure leaf wetness during the critical period of inoculation and infection of *S. tritici*. Because of high G x E interaction, field trials should be conducted at several locations across a minimum of two years to get accurate genotypic differentiation of wheat varieties and breeding lines for resistance to STB.

Inoculation *versus* natural infection

One rationale for the current field trials was to study whether ratings of inoculated plots coincide with natural infection. Coefficients of correlation between non-inoculated plots and mean STB rating of inoculated plots across nine environments were high, except in 2007, where natural infection was almost missing. Inoculation supports infection with STB under field conditions, thus genotypic differentiation is improved. In field trials natural infection cannot be excluded, therefore, level of natural infection contributes to the symptoms depending on environmental influence. Not inoculated plots 2008 and 2009 were diseased as high as inoculated plots each year. Thus, natural infection played an important role. With selected isolates, similar infection rates and genotypic differentiation were obtained by comparing inoculated plots with not inoculated plots.

Seedling test

Parents of mapping populations were also evaluated at seedling stage to detect genotype-by-isolate (G x I) interactions. The avirulent isolates IPO 323 (avir. *Stb6*) and IPO 88004 (avir. *Stb15*) showed highly significant G x I interactions, thus *Stb6* was postulated in Arina, Florett, Forno, Rubens, Solitär, and Tuareg as well as *Stb15* in Arina and Florett (Kema 2009, pers. comm.). Gene postulation based on these seedling test has to be confirmed by mapping of segregating populations with the respective avirulent isolates. Other studies showed presence of *Stb6* and *Stb15* in Arina, confirming these results (Chartrain et al. 2004a; Arraiano et al. 2007). The isolates (BAZ 6/1/04 and BAZ 8/8/04) chosen to inoculate field trials were virulent to all parents in seedling stage indicating no isolate-specific resistance. Results of QTL analysis revealed no QTL for STB resistance on

chromosomes 3A and 6A in this study, where the most common isolate-specific resistance genes *Stb6* and *Stb15* are located (Arraiano and Brown 2006).

There are many reports about resistance screening to STB in wheat cultivars at the seedling stage in which *Stb* genes were sought (Kema et al. 1996a; Kema et al. 1996b; Arraiano et al. 2001a; Arraiano and Brown 2006). At seedling stage, two different methods were applied: (i) whole seedling test (Kema et al. 1996a) and (ii) detached leaf test (Arraiano et al. 2001a). Both methods were basically used to detect interactions between wheat genotypes and *S. tritici* isolates. A gene-for-gene relationship was described for *Stb6* (Brading et al. 2002), which is widespread in wheat varieties (Chartrain et al. 2005b). Coefficients of correlation across whole seedling test, detached leaf test, and field test were high (Arraiano et al. 2001a), depending, however, on isolates used for inoculation (Kema and van Silfhout 1997). Hence, screening methods at seedling stage are useful to detect isolate-specific resistance. The inoculation at seedling stage cannot predict quantitative resistance in adult plants. Therefore, field trials at several environments inoculated with isolates virulent to common *Stb* genes are the method of choice to analyze quantitative adult-plant resistance. The advantage of field trials compared to tests at seedling stage is the ability to evaluate varieties under natural conditions, which is closest to agricultural practice. Disadvantages are labour, space, and time required to set up and evaluate field trials. However, field trials are of high risk to detect isolate-specific resistance genes due to the influence of natural infections as shown in our trials.

Genotypic differentiation of adult-plant resistance under inoculation

Isolates used to inoculate field trials in this study were selected in order to detect quantitative adult-plant resistance rather than isolate-specific resistance genes. Thus, most of the non-environmental variation in the resulting disease severity was explained by differences between wheat genotypes. In contrast to other studies (Kema and van Silfhout 1997; Arraiano and Brown 2006; Arraiano et al. 2007), no significant G x I interaction was detected indicating quantitative, horizontal resistance (Parlevliet 1977) in our material triggered by the choice of isolates.

The analysis of variance revealed a significant genotypic differentiation for resistance to STB in the test of varieties and in five segregating populations. In the variety test, genotypic variation of STB severity ranged from 8 to 63 % flag leaf area infected, evaluated

across three years including nine environments. Until now, no comparable study has been published giving such evaluation of quantitative adult-plant resistance to STB in European wheat germplasm.

Comparing results for STB in this study with data from official variety testing in Germany (Bundessortenamt 2009, scale from 1 to 9, 1 = no susceptibility, 9 = very high susceptibility) the varieties Biscay (7), Bussard (6), and Drifter (7) are susceptible, whereas Sobi (3) and Solitär (2) are resistant, confirming results presented here. Solitär, the most resistant variety for STB, is also known as a resistant cultivar for Fusarium head blight, powdery mildew (caused by *Blumeria graminis*), and Stagnospora glume blotch (Bundessortenamt 2009). Therefore, this variety seems to be a useful parent for resistance breeding. Its use, however, is limited by tallness and lower yield performance.

Genotypic variation for STB was significant in all five segregating populations. The distribution of wheat lines for flag leaf infections with STB revealed a wide phenotypic variation indicating a quantitative inheritance in all five mapping populations.

We can conclude that varieties as well as segregating populations showed a wide range of genotypic variation for STB resistance which is useful for breeding.

Environmental influence on Septoria tritici blotch resistance

Environment and G x E interaction affected STB ratings in the test of varieties as well as in all five mapping populations. The level of resistance of varieties is one factor that contributes to G x E interaction. Resistant varieties are more stable across environments than susceptible varieties (Miedaner and Flath 2007). Successful new varieties combine high yield and stable resistance over a wide range of different environments. Thus, plant breeders should be interested in stable varieties. We used a regression approach (Eberhart and Russell 1966) to identify varieties stable for STB resistance across nine environments. Concepts of stability and methods to analyse G x E interactions were summarized by several authors (Freeman 1973; Westcott 1986; Becker and Léon 1988). In contrast to multivariate methods, linear regression provides a simple measure of stability, which allows ranking of varieties. The limitation of this approach is the large number of environments needed for an accurate estimate of stability. The importance of assessing stability parameters for resistance traits was shown in this study. Tuareg for instance, was resistant in the first year and highly susceptible in the second year of evaluation, thus

mean rating across all environments would, therefore, indicate a moderate resistant variety. Including MS_{dev} of regression, Tuareg was revealed as not being stable. In conclusion, multienvironmental data of STB severity are necessary. The information on stability is an additional tool to select breeding lines with a high level of resistance which are stable across environments.

Escape mechanisms

Escape mechanisms, like plant height and heading date, reduce the chance of contact between pathogen and host. Influence of PLH and HED to STB resistance must be taken into account (Rosielle 1972; Parlevliet 1977; Jlibene et al. 1992; Simón et al. 2004). In test of isolates and varieties, correlation between STB and PLH was missing, whereas correlation between STB and HED was moderate, just depending on two very early and susceptible varieties (Apache, Rubens) and one late very resistant variety (Solitär). For the remaining 21 varieties correlation was not significant. A significant negative moderate correlation between STB severity and HED as well as PLH was detected in all five mapping populations. The only exception was History/Rubens with a considerably higher correlation between STB and PLH caused by the segregating *Rht-D1* locus. In four out of five mapping populations resistance loci coincided with positions for plant height and/or heading date indicated by overlapping confidence intervals of detected QTL on chromosomes 1B (History/Rubens), 2B (Florett/Biscay), 4B (Arina/Forno), 4D (Tuareg/Biscay, History/Rubens), 5B (Arina/Forno), and 6D (Arina/Forno) (**S 5**). However, there was no severe influence on PLH and HED to disease severity in our study. One explanation could be that inoculation was done once after latest genotypes' flag leaves had been fully unrolled, thus, differences in plant height or heading date had no severe effect on disease development, although, these differences were significant. In natural infected field trials taller plants tend to have less disease because the vertical spread of spores up the plant is reduced (Baltazar et al. 1990; Jlibene et al. 1992). The influence of plant height and heading date to STB was analyzed recently in a set of 226 wheat lines after natural infection (Arraiano et al. 2009). Reduced STB severity was mainly associated with greater plant height ($r = -0.7$, $P < 0.001$), whereas heading date had no severe effect ($r = -0.1$, $P > 0.05$). Other studies showed significant negative coefficients of correlation between plant height and leaf infections after field inoculation with *S. tritici* (Rosielle 1972; Jlibene et al. 1992; Arraiano

et al. 2009), whereas Chartrain et al. (2004b) found a positive correlation that was not significant. There was a significant negative correlation between flowering as well as HED and STB infection reported by several authors (Arama et al. 1999; Chartrain et al. 2004b; Arraiano et al. 2006). Earlier genotypes became more diseased than later genotypes. This was true for Apache and Rubens in this study, but not for other varieties.

In conclusion, assessment of morphological traits is of importance to provide unbiased results and to avoid escape mechanisms as being selected as resistance. Taking into account the influence of PLH and HED to STB severity, data should be corrected according to a covariance analysis (Simón et al. 2004).

Effect of *Rht-D1* on Septoria tritici blotch

Generally, correlations between STB and PLH as well as between STB and HED in this study were of minor importance with one exception regarding correlation between STB and PLH in History/Rubens population, which is segregating at the *Rht-D1* locus. Earlier, the effect of the *Rht-D1* dwarfing locus on FHB rating was shown in History/Rubens and two other populations (Voss et al. 2008). Plant height and FHB rating were significantly negatively correlated in all populations. This correlation decreased within subpopulations homozygous for one or the other height allele (Voss et al. 2008). In our study, the correlation remained significant in the shorter subpopulation (carrying *Rht-D1b*, $r = -0.59$, $P < 0.01$) and vanished only in the taller subpopulation (carrying *Rht-D1a*, $r = -0.10$, $P > 0.05$). Either linkage or pleiotropic effects might be possible explanations for the correlation between plant height and disease rating (Holzapfel et al. 2008; Voss et al. 2008). In another study, Simón et al. (2004) evaluated the influence of *Rht* genes on STB severity. They concluded that shorter distances between leaf layers favoured inoculum transfer and therefore reduced plant height was correlated with higher disease severity. Here, in the mapping population History/Rubens, we found QTL for PLH and STB severity at the same genome position, indicating linkage or pleiotropic effects responsible for negative correlation between PLH and STB severity.

The most effective QTL for resistance to STB is located on chromosome 4D caused by the lower susceptibility of the taller wild-type allele (*Rht-D1a*) at *Rht-D1* locus. However, another major QTL was detected on chromosome 5B. Plant breeders have to select short, high yielding varieties in combination with at least moderate resistance. Thus, it might be

possible to select short varieties in combination with improved resistance, which seems promising taking into account the variance for STB within the shorter subpopulation.

4.2 Genetic mapping and QTL detection

Influence of genetic similarity of parents on rate of polymorphism

In total, five mapping populations were used in our QTL analysis. Two of them, Arina/Forno and History/Rubens, have been used in previous studies to map FHB and SGB resistance. These two genetic maps showed high density and good genome coverage. In History/Rubens additional DArT markers even improved genome coverage and facilitated combining linkage groups within one chromosome. A plant breeding company provided three additional populations using modern varieties for parents. The level of polymorphism between parents of mapping populations Florett/Biscay and Tuareg/Biscay was relatively low. The AFLP polymorphism was lower in Florett/Biscay and Tuareg/Biscay with 3 to 4 AFLPs per primer combination compared to 7 to 8 in History/Rubens (Holzapfel 2009 pers. comm.) and 6.4 in Arina/NK93604 (Semagn et al. 2006). A similar low rate of polymorphism was detected for DArT markers. In Florett/Biscay and Tuareg/Biscay 7 to 8 % of analyzed DArT markers were polymorphic, in History/Rubens 17.6 %, and in Bussard/Solitär 13 %. One explanation for these differences in the level of polymorphism could be genetic similarity (*GS*) between parents. Florett, Tuareg, and Biscay are quite similar to each other (*GS* = 0.70 to 0.73), compared to History/Rubens (*GS* = 0.58) and Bussard/Solitär (*GS* = 0.66) as revealed by cluster analysis (see **Figure 7**).

The genetic maps of Florett/Biscay, Tuareg/Biscay, and Bussard/Solitär span more than 1,300 cM with small average interval distances (2.3 to 6.1 cM) indicating high density maps, although genome coverage is limited. In comparison, Somers' high-density consensus map covered 2,569 cM with an average interval distance of 2.2 cM (Somers et al. 2004). Although in the five mapping populations presented in this study a large number of polymorphic markers were generated (384 to 939), only a smaller proportion remained in the final map (28 to 68 %) due to clustering and missing linkage. This indicates that especially the genetic similar parents Florett/Biscay and Tuareg/Biscay possess highly conserved genomic regions. Due to the lack of coverage of genetic maps compared to History/Rubens (2,361 cM) and Arina/Forno (3,305 cM), QTL could possibly remain unde-

tected. With regard to the results of QTL analysis, the explained phenotypic variance (nR^2_{adj}) by detected QTL was independent from genome coverage, thus most important genomic regions with respect to STB resistance, PLH, and HED should be detected. The exception was Bussard/Solitär, with less than 15 % nR^2_{adj} explained by only one QTL for STB. Therefore, application of additional polymorphic markers could improve genome coverage that reveals additional QTL loci. In parallel, the Bussard/Solitär population needs more precise phenotypic evaluation, such as sowing double rows with two replications across several environments with increased population size.

In all five mapping populations, higher proportions of markers were mapped on the A and B genome compared to the D genome. The low level of polymorphism in this genome is consistent to several other studies (Röder et al. 1998; Eriksen et al. 2003; Akbari et al. 2006) and can be explained by the rather recent introgression of the D genome into bread wheat probably occurring only a few times or even once in history (Salamini et al. 2002). The development of D genome specific markers from the diploid *Aegilops tauschii* (Pestsova et al. 2000) facilitated mapping and improved marker density on the D genome of hexaploid wheat.

We can conclude that the level of marker polymorphism decreases using modern high-yielding varieties as parents in mapping populations, due to highly conserved genomic regions. However, most of phenotypic variance for STB resistance was represented by detected QTL with exception of Bussard/Solitär. These QTL already reside in an adapted genetic background with high agronomic performance useful in resistance breeding.

QTL for STB resistance

In adult plants, resistance to STB can be isolate-specific or quantitative. The phenotypic data of mapping populations suggested a typical quantitative inheritance of STB resistance evaluated in the field at adult-plant stage. The results of QTL analysis confirmed that resistance was inherited quantitatively depending on several QTL, each explaining part of phenotypic variance. In total, 26 QTL for STB resistance were mapped across five wheat populations on all chromosomes excluding 1D, 2A, 2D, 3A, 3D, 5A, 5D, and 6A. QTL for resistance to STB in all five wheat populations were not equally distributed across chromosomes and genomes. Most of STB resistance QTL were mapped on the B genome (60 %).

Altogether, detected QTL in each population explained 14 to 55 % of adjusted phenotypic variance (R^2_{adj}). R^2 was adjusted (R^2_{adj}) to get more adequately estimation of explained phenotypic variance (Hospital et al. 1997) and normalized (nR^2_{adj}) that the sum across detected QTL is equal to model R^2_{adj} (see Zhu et al. 2004). Both parents contributed resistant alleles. Major QTL, however, were all from the respective resistant parent. Eight QTL were declared major explaining more than 10 % of nR^2_{adj}. This is quite strict in comparison to other QTL studies (Draeger et al. 2007; Semagn et al. 2007), which used just R^2 to differentiate between minor and major QTL. Therefore, major QTL in this study were found consistently across environments: *QStb.lsa_fb-3B, QStb.lsa_fb-6D, QStb.lsa_tb-4B, QStb.lsa_tb-6B, QStb.lsa_hr-4D, QStb.lsa_hr-5B.1, QStb.lsa_af-3B, QStb.lsa_bs-7A*. Major QTL from different populations (Florett/Biscay and Arina/Forno) were detected at the same chromosome 3B (**S 5**). Because of missing common markers it was not possible to locate whether these QTL were at the same marker interval. Fine mapping of these QTL with markers common across populations will map QTL positions more precisely, facilitate meta-analysis, and thus promote marker-assisted selection in wheat breeding. This is suggested for all major QTL presented here, because the main limitations to do meta-analysis in wheat are missing common markers in QTL regions.

In two other studies detection of QTL for STB resistance were reported. Eriksen et al. (2003) mapped QTL for STB resistance, some of the alleles providing resistance at seedling stage, whereas others at adult-plant stage and some were effective at both the seedling and the adult-plant stage. With focus on adult-plant stage, four resistance QTL were mapped at chromosomes 2B, 3A, 6B, and 7B (*QStb.risø-2B, QStb.risø-3A.2, QStb.risø-6B.2, QStb.risø-7B*), altogether explaining 62 to 77 % of phenotypic variance. The resistant parent Senat provided all resistant alleles. There was an overlap of resistance QTL and QTL for plant height at chromosome 3A. The authors concluded that linkage is the most likely reason for this event. In this study, we mapped QTL on the same chromosomes (**S 5**), two on chromosome 2B in Florett/Biscay and Arina/Forno (*QStb.lsa_fb-2B*, *QStb.lsa_af-2B*), two on chromosome 6B in Tuareg/Biscay and History/Rubens (*QStb.lsa_tb-6B*, *QStb.lsa_hr-6B*), and even three on chromosome 7B in Tuareg/Biscay, History/Rubens, and Arina/Forno (*QStb.lsa_tb-7B*, *QStb.lsa_hr-7B*, *QStb.lsa_af-7B*). SSR markers *Xwmc0344* on chromosome 2B close to *QStb.lsa_fb-2B* and *Xwmc517* on chromosome 7B

close to *QStb.lsa_tb-7B* were the only markers in accordance to the map of Savannah/Senat. Support intervals of resistance QTL in Savannah/Senat on chromosomes 2B and 7B did not include the common markers indicating independency of QTL detected here.

Chartrain et al. (2004b) detected one QTL (*QStb.psr-6B-1*) explaining 24 % of phenotypic variance (R^2). The resistance allele came from the susceptible parent Riband. The position of the QTL was close to SSR marker *Xgwm0219*, which is also present in Tuareg/Biscay and History/Rubens (**S 5**). Other common markers are missing, thus comparing the position of STB QTL mapped on this chromosome in Tuareg/Biscay and History/Rubens (*QStb.lsa_tb-6B*, *QStb.lsa_hr-6B*) was not possible.

These two studies show that, until now, little has been known about quantitative resistance to STB at adult-plant stage. Population size and number of evaluated environments were very limited in these studies, which has a significant effect on power of QTL detection as well as on accuracy and precision of QTL estimates (Melchinger et al. 2004; Schön et al. 2004). Our results can provide a better understanding of inheritance of STB resistance at adult-plant stage. QTL mapping in five populations, where three were much larger in population size and the multienvironmental evaluations allow accurate estimation of QTL effects confirmed by cross validation, which was not applied by Chartrain et al. (2004b).

The challenges of QTL mapping in wheat populations compared to other crops are (i) the large genome size, (ii) low polymorphism rate between modern high-yielding varieties, and (iii) that the genome has not yet been sequenced. Therefore, a multi-stage-QTL-mapping approach is proposed. Firstly, genetic linkage maps are generated in each population using high throughput low cost marker techniques (DArT, AFLP) in combination with some anchor markers (SSR). Good genome coverage of sufficient density is needed to detect most of responsible resistance-QTL alleles. In parallel, mapping populations inoculated at several environments conducive to disease development are phenotyped. Secondly, QTL mapping is applied in each population to reveal common QTL regions across populations. Thirdly, fine mapping of common QTL regions with a larger set of common markers is conducted across populations. After this step, it should be possible to generate a consensus map of common QTL region via meta analysis.

4.3 QTL meta-analysis

One objective of this study was to reveal common QTL regions for multiple-disease resistance within two mapping populations using meta-analysis. Meta-analysis is defined as the integration of individual experiments with a comparative map-based approach on three different levels: (i) different populations within the same crop inoculated with one pathogen (e.g. wheat/*Septoria*); (ii) one population of the same crop inoculated with different pathogens (e.g. wheat/*Fusarium*/*Septoria*/*Stagnospora*); (iii) populations of different crops inoculated with the same pathogen (e.g. wheat and maize/*Fusarium*). In this study, we present data concerning the first two levels, the third level will be discussed only theoretically.

On chromosome 5B, QTL for STB resistance were located close to *Xgwm0274* in Arina/Forno (*QStb.lsa_af-5B*) and History/Rubens (*QStb.lsa_hr-5B.1*), indicating a meta QTL effective across both populations, according to the first level of definition of meta-analysis. Because of missing common markers between mapping populations it was not possible to create a consensus map and to estimate position, support interval, and effect of meta QTL across segregating populations within different wheat populations inoculated with *S. tritici*. Further marker analyses are needed to fine map detected initial QTL and to reveal meta QTL across mapping populations inoculated with one or several important pathogens.

Meta-analysis revealed QTL for multiple-disease resistance with significant effects across a minimum of two disease traits within Arina/Forno and History/Rubens, according to the second level of definition of meta-analysis. Four meta QTL for different disease combinations were detected in each population, three on the B genome and one on the D genome. Initial QTL in Arina/Forno of Schnurbusch et al. (2003) for SGB and of Paillard et al. (2004) for FHB as well as in History/Rubens of Holzapfel et al. (2008) for FHB were confirmed, revealed by reanalyzing raw data with PLABMQTL using equal settings than the previous authors. Three meta QTL, on chromosome 5B in Arina/Forno and on chromosomes 4D and 7B in History/Rubens with small support intervals (SI ≤ 6), are based on two initial QTL already detected in the single-trait-analysis. All other meta QTL had significant effects because of either one detected initial QTL or even without any detected initial QTL, which is the case for meta QTL on chromosomes 3B and 2B in Arina/Forno and History/Rubens, respectively. The latter two meta QTL as well as the LOD peak detected at

the beginning of chromosome 5B in History/Rubens (**Figure 10**, *QGhost*) could be a non-existing "ghost" meta QTL. By adding two smaller LOD scores, each not detected in the initial QTL mapping experiment, meta QTL with significant effects could be arise. The problem of mistakenly identified QTL in initial QTL analysis was discussed in detail previously (Martinez and Curnow 1992; Jansen 1993) and the same applies for meta QTL. Therefore, threshold for detection of meta QTL as well as validation of detected meta QTL has to be commented upon.

For detection of meta QTL, threshold was set in this study to LOD 6.0, i.e. double the LOD score according to initial QTL analysis using 3.0. No adjustment of critical LOD scores due to permutations are at present possible in the used program PLABMQTL. In total, 12 and 19 positions were detected with LOD ≥ 6.0 in Arina/Forno and History/Rubens, respectively. Out of these, meta QTL for multiple-disease resistance were selected by the following criterion: Significant individual QTL effects for a minimum of two disease traits. According to the selection criterion, eight multiple-disease resistance QTL were found in the two mapping populations. Two meta QTL on chromosome 3B in Arina/Forno and on chromosomes 2B in History/Rubens were detected, although these QTL alone had no significant effects in initial QTL analysis. A LOD threshold in meta-analysis larger than 6 in Arina/Forno and even larger in History/Rubens, according to small population size of mapping populations, is recommended. In combination with the selection criterion of significant QTL effects at a minimum of two disease traits, nonexisting "ghost" meta QTL would not have been selected. Further research is needed to define critical LOD scores for meta QTL. Until now, permutation test and cross validation for meta QTL were not possible in PLABMQTL. In future analyses, such tools are highly recommended to determine the magnitude of bias of number, position, and genetic effects of meta QTL.

In Arina/Forno, multiple-disease resistance QTL were only detected across two pathogens. There was no meta QTL with significant effects across all three pathogens. However, there is a positive side effect, as shown for chromosome 5B, where STB infection is reduced by selecting the resistant allele effective for resistance to FHB and SGB. In comparing meta QTL positions between mapping populations, there is one meta QTL on chromosome 5B effective against FHB and SGB in Arina/Forno and similarly effective against FHB and STB in History/Rubens. Because of missing common markers at this position it is not

possible to specify whether this meta QTL is located at the same position in both populations (**S 5**).

However, it was possible to reveal each of four multiple-disease resistance QTL across each of two pathogens in two mapping populations, which has not been shown before in wheat studies. Closely linked markers are available, and small support intervals indicate high accuracy of meta QTL positions.

In future analysis, synteny between major crops such as rice, maize, and wheat (Bennetzen and Ma 2003), will promote detection of meta QTL in segregating populations across different crops, according to the third level of definition of meta-analysis. *Fusarium* head blight and ear rot are major diseases in wheat and maize both caused by *F. graminearum* provoking losses in yield and quality and accumulation of mycotoxins. Therefore, resistance breeding is one major component to reduce *Fusarium* severity in both crops. Meta-analysis is a useful tool to detect common regions for *Fusarium* resistance across wheat and maize genome or to use synteny to check if QTL appearing in one crop are also present in the other at similar loci. Until now, no study has been published concerning meta-analysis across these two crops. The increasing number of publications concerning *Fusarium* resistance in wheat (e.g. Waldron et al. 1999; Buerstmayr et al. 2002; Paillard et al. 2004; Schmolke et al. 2005; Draeger et al. 2007; Liu et al. 2007; Semagn et al. 2007; Abate et al. 2008; Holzapfel et al. 2008; Bonin and Kolb 2009) and maize (Pérez-Brito et al. 2001; Ali et al. 2005; Robertson-Hoyt et al. 2006; Ding et al. 2008) seems promising with regard to future research using synteny to reveal meta QTL across wheat and maize with the goal of reducing *Fusarium* severity.

4.4 Genetic architecture of STB resistance and significance for resistance breeding

QTL analysis was performed in five segregating populations revealing a total of 26 QTL for resistance to STB allowing some conclusions on genetic architecture of this trait: (i) a large diversity of QTL have been mapped on most wheat chromosomes accounting for quantitative STB resistance at adult-plant stage, (ii) most resistance QTL are located on the B genome in comparison to the A and D genome, and (iii) in every population at least one major QTL was detected explaining more than 10 % of nR^2_{adj}. In summary, adult-plant resistance to *S. tritici* in the analyzed populations was inherited quantitatively depending on several loci each explaining only a smaller part of the phenotypic variance.

Besides quantitative resistance, thirteen isolate-specific resistance genes have been mapped in previous studies. No *Stb* gene was mapped on chromosomes 1A, 2B, 4B, 4D, and 6D whereas in this study 12 QTL for STB resistance have been found. Thus, additional sources for STB resistance have been detected. At the same chromosomal region as QTL in this study some *Stb* genes were mapped on chromosomes 1B, 3B, 4A, 5B, 7A, 7B, and 7D confering to *Stb11*, *Stb2*, *Stb7*, *Stb1*, *Stb3*, *Stb8*, and *Stb4*. Five QTL (*QStb.lsa_fb-1B*, *QStb.lsa_tb-4A*, *QStb.lsa_fb-7A*, *QStb.lsa_bs-7A*, *QStb.lsa_af-7B*) were mapped on chromosomes with common markers next to *Stb* genes (**Table 23**), all the other QTL were mapped on similar chromosomes, but with no common markers. *Xwmc0219* next to *Stb12* on chromosome 4A and *Xgwm0146* next to *Stb8* on chromosome 7B were mapped within the confidence interval (CI) of *QStb.lsa_tb-4A* and *QStb.lsa_af-7B*, respectively, whereas *Xbarc0008* next to *Stb11* and *Xwmc0083* next to *Stb3* were close to CI of QTL, but not within. Therefore, *Stb12* and *Stb8* could have an effect at adult-plant stage detected as QTL in this study. Arraiano et al. (2009) analyzed the contribution of isolate-specific disease resistance to the control of STB in wheat. Most variation in level of STB resistance caused by natural infection was explained by the presence or absence of *Stb6*. It is discussed that defeated *Stb* genes may confer quantitative or partial resistance to STB at adult-plant stage, which could be a residual effect. Another hypothesis was linkage between *Stb* genes and QTL (Arraiano et al. 2009). Further research is required to validate these hypotheses.

Table 23: Comparison of position of QTL and *Stb* genes located on the same chromosome using common markers present in genetic maps of segregating populations and 95 % confidence interval (CI) of QTL position; marker position within CI of QTL are highlighted

Chrom.	QTL designation	*Stb* gene	Common marker	Marker position (cM)	CI of QTL (cM)
1B	*QStb.lsa_fb-1B*	*Stb11*	*Xbarc0008* [1)]	65	68-88
4A	*QStb.lsa_tb-4A*	*Stb12*	*Xwmc0219* [2)]	7	0-13
7A	*QStb.lsa_fb-7A*	*Stb3*	*Xwmc0083* [3)]	0	30-42
7A	*QStb.lsa_bs-7A*	*Stb3*	*Xwmc0083* [3)]	7	23-53
7B	*QStb.lsa_af-7B*	*Stb8*	*Xgwm0146* [4)]	148	130-157

[1)] Chartrain et al. 2005c
[2)] Chartrain et al. 2005a
[3)] Goodwin et al. 2007
[4)] Adhikari et al. 2003

STB resistance is mainly affected by additive effects and, therefore, the combination of major QTL in breeding material seems promising to reduce STB severity. Another strategy is to use markers next to *Stb* genes within CI of QTL to combine isolate-specific resistance and quantitative resistance. Meta-analysis in two populations revealed eight loci responsible for multiple-disease resistance. Closely linked markers allow the implementation in marker-assisted breeding programs. Further research is required to validate major QTL for STB as well as meta QTL for multiple-disease resistance in different genetic backgrounds.

An outlook to future research could be the combination of FHB resistance QTL reported in the literature with detected major QTL for STB resistance. Both diseases are of major interest in a practical breeder's point of view. Thus, using donors for FHB and STB resistance as parents to validate QTL effects in the segregating progenies could be the next step. Another strategy is the validation of meta QTL. Lines carrying resistance alleles of meta QTL can be used in backcrosses with the resistance donor. Progenies selected by flanking markers representing different allele situations at meta QTL locus could be phenotyped in parallel field trials inoculated with *Fusarium*, *Stagnospora*, and *Septoria*.

5 SUMMARY

Septoria tritici blotch (STB), caused by *Septoria tritici* (teleomorph *Mycosphaerella graminicola*), is one of the most important diseases in wheat varieties worldwide, responsible for severe damage of the leaves causing yield losses between 30 and 40 %. Control of STB includes crop rotation, soil tillage, fungicide application, and cultivation of resistant varieties. Profit-making wheat growers are forced to apply narrow crop rotations under reduced tillage. Some fungicides including widely-used strobilurins are no longer effective due to mutations in the highly variable pathogen population of *S. tritici*. Therefore, resistance breeding using genetic mapping to identify quantitative-trait loci (QTL) associated with STB resistance provides a promising strategy for controlling the disease.

The main goal of this study was to detect chromosomal regions for quantitative adult-plant resistance of winter wheat to STB. Besides this, we analyzed the genetic diversity of 24 European varieties after inoculation with four different isolates of *S. tritici*. Multienvironmental field trials inoculated with *S. tritici* were applied to test isolates and varieties and to phenotype mapping populations. In detail, the objectives were to (1) compare natural infection and inoculation, (2) evaluate genotypic variation of adult-plant resistance to STB in European varieties, (3) analyze genotype x environment (G x E) interaction, (4) evaluate and analyze phenotypic data including STB severity, heading date (HED), and plant height (PLH) of five mapping populations, (5) construct genetic linkage maps of these populations using AFLP, DArT, and SSR markers, (6) determine number, positions, and genetic effects of QTL for evaluated traits, and (7) reveal QTL regions for multiple-disease resistance within mapping populations using QTL meta-analysis.

In all trials, inoculation with one to four preselected isolates was performed and STB severity was visually scored plotwise as percentage coverage of flag leaves with lesions bearing pycnidia. 24 winter wheat varieties were chosen with maximal differentiation in resistance to STB and evaluated across three years including nine environments. Five mapping populations, Florett/Biscay, Tuareg/Biscay, History/Rubens, Arina/Forno, and Solitär/Bussard, each comprising a cross of a resistant and a susceptible variety, with population sizes ranging from 81 to 316, were phenotyped across four to six environ-

ments. In parallel, 221 to 491 polymorphic genetic markers were assigned to linkage groups covering 1,314 to 3,305 cM of the genome. Based on these linkage maps, the number, positions, and genetic effects of QTL could be determined by composite interval mapping. Furthermore, raw data of different experiments evaluated for resistance to two other pathogens, Fusarium head blight and Stagnospora glume blotch, were used to reveal multiple-disease resistance QTL within Arina/Forno and History/Rubens populations by the software package PLABMQTL.

Results of inoculated field trials coincided with not inoculated trials showing natural infection ($r = 0.84$ to 0.99, $P < 0.01$), thus inoculation method was accurate to evaluate STB severity in the field. Genotypic variation between 24 varieties ranged from 8 % (Solitär) to 63 % (Rubens) flag leaf area infected. In the analysis of variance, genotypic variance had highest impact followed by G x E interaction ($P < 0.01$). Therefore, environmental stability of varieties should be a major breeding goal. The varieties Solitär, History, and Florett were most stable, as revealed by a regression approach. In contrast, disease symptoms of Biscay ranged from 19 to 72% within the three experimental years.

Phenotypic data revealed significant ($P < 0.01$) genotypic differentiation for STB, HED, and PLH within all five mapping populations and between the parents. Entry-mean heritabilities (h^2) ranged from 0.69 to 0.87 for STB, the only exception was Tuareg/Biscay ($h^2 = 0.38$). For HED ($h^2 = 0.78$ to 0.93) and PLH ($h^2 = 0.92$ to 0.98) heritabilities were high. All correlations between STB and HED ($r = -0.18$ to -0.33) as well as between STB and PLH ($r = -0.13$ to -0.45) were negative and moderate. The exception was History/Rubens which is segregating at the *Rht-D1* locus showing considerably higher correlation between STB and PLH ($r = -0.55$, $P < 0.01$). The five mapping populations showed a wide and continuous distribution of mean STB severity averaged across three to six environments in field trials at adult-plant stage.

In QTL analysis, one to nine, zero to nine, and four to eleven QTL were detected for STB, HED, and PLH, respectively, across five wheat populations using composite interval mapping. One to two major QTL for resistance to STB were detected consistently across environments in each population (*QStb.lsa_fb-3B, QStb.lsa_fb-6D, QStb.lsa_tb-4B,*

QStb.lsa_tb-6B, QStb.lsa_hr-4D, QStb.lsa_hr-5B.1, QStb.lsa_af-3B, QStb.lsa_bs-7A) explaining more than 10 % of normalized adjusted phenotypic variance. Altogether, resistance QTL explained 14 to 55 % of adjusted phenotypic variance. Both parents contributed resistant alleles. Major QTL, however, were all from the resistant parent.

QTL meta-analysis revealed each of four loci for multiple-disease resistance located on chromosomes 3B, 4B, 5B, and 6D in Arina/Forno, and on chromosomes 2B, 4D, 5B, and 7B in History/Rubens. The most effective meta QTL was on chromosome 4D in History/Rubens closely linked to *Rht-D1*. The resistance allele from History reduced disease severity by 9.8 % for STB and 6.3 % for FHB, thus explaining 47 % and 60 % of partial phenotypic variance.

In general, European wheat varieties showed a wide range of genotypic variation for STB resistance useful for breeding. Although the influence of environment and G x E interaction was high, some resistant varieties which were stable across multiple environments were found (Solitär, History, Florett). Genomic regions associated with STB resistance were mapped across 13 out of 21 wheat chromosomes. Together with the continuous distribution of five segregating populations for flag leaf infection, it can be concluded that the adult-plant resistance to *S. tritici* was inherited quantitatively depending on several loci explaining part of phenotypic variance. QTL meta-analysis across three severe pathogens, including Fusarium head blight, Stagnospora glume blotch, and STB, within two populations revealed eight loci for multiple-disease resistance with closely linked markers applicable in resistance breeding. Combining detected major QTL as well as meta QTL in present breeding material by applying marker-assisted selection seems a promising approach to the breeding of varieties with improved resistance to Septoria tritici blotch, Fusarium head blight, and Stagnospora glume blotch.

6 ZUSAMMENFASSUNG

Die Septoria-Blattdürre (Septoria tritici blotch, STB) des Weizens wird durch den Erreger *Septoria tritici* (teleomorph *Mycosphaerella graminicola*) verursacht. Die gefährliche Blattkrankheit hat in den Weizenanbaugebieten der gemäßigten Breiten weltweit große Bedeutung und führt zu Ertragsverlusten zwischen 30 und 40 %. Das Befallsrisiko kann durch Fruchtfolgegestaltung, Bodenbearbeitung, Fungizidapplikation und den Anbau resistenter Sorten verringert werden. Um Kosten zu reduzieren und das Betriebseinkommen zu steigern, sind Landwirte gezwungen, Weizen in engen Fruchtfolgen mit reduzierter Bodenbearbeitung anzubauen. Die als Fungizid weit verbreiteten Strobilurine haben aufgrund von Mutationen in der sehr anpassungsfähigen Pathogenpopulation von *S. tritici* ihre Wirkung verloren. Die Resistenzzüchtung liefert durch die Anwendung der genetischen Kartierung zur Lokalisierung quantitativ vererbter Resistenzloci (sog. QTL) einen vielversprechenden Ansatz zur Kontrolle der Septoria-Blattdürre bei Weizen.

Im Vordergrund dieser Studie stand die Lokalisation chromosomaler Regionen für die quantitativ vererbte Adultpflanzenresistenz von Weizen gegen STB. Außerdem wurde die genetische Diversität von 24 europäischen Sorten nach Inokulation mit vier verschiedenen *S. tritici*-Isolaten untersucht. Es wurden Feldversuche in mehreren Umwelten mit ein bis vier ausgewählten *S. tritici*-Isolaten inokuliert, um Isolate und Sorten zu testen und um Kartierungspopulationen zu phänotypisieren. Diese Isolate wurden so ausgewählt, dass sie nur Adultpflanzenresistenz entdecken. Die Ziele waren im Einzelnen (1) den Krankheitsbefall bei natürlicher Infektion und nach Inokulation zu vergleichen, (2) die genetische Variation für Adultpflanzenresistenz gegen STB in europäischen Sorten zu untersuchen, (3) die Genotyp x Umwelt (G x U)-Interaktion zu analysieren, (4) die phänotypischen Merkmale STB-Befall, Ährenschieben (AES) und Wuchshöhe (WUH) von fünf Kartierungspopulationen zu erheben und varianzanalytisch auszuwerten, (5) genetische Kopplungskarten dieser Populationen mit AFLP, DArT und SSR Markern zu erstellen, (6) Anzahl, Position und die genetischen Effekte der QTL für die erhobenen Merkmale zu bestimmen und (7) Genomregionen für multiple Krankheitsresistenz innerhalb der Kartierungspopulationen mit Hilfe einer QTL Meta-Analyse zu entdecken.

In allen Feldversuchen wurde nach der Inokulation der prozentuale Krankheitsbefall mit STB auf dem Fahnenblatt im Mittel über die Parzelle visuell erfasst. 24 Weizensorten, die sich in der Resistenz gegen STB unterscheiden, wurden über drei Jahre in neun Umwelten angebaut. Fünf Kartierungspopulationen, Florett/Biscay, Tuareg/Biscay, History/Rubens, Arina/Forno und Solitär/Bussard, jeweils Kreuzungen aus einer resistenten und einer anfälligen Sorte mit Populationsgrößen zwischen 81 und 316 Genotypen wurden in vier bis sechs Umwelten phänotypisiert. Parallel wurden je Population 221 bis 491 polymorphe Marker den einzelnen Kopplungsgruppen zugeordnet. Die entstandenen genetischen Karten hatten eine Genomabdeckung von 1.314 bis 3.305 cM. Auf der Grundlage dieser genetischen Karten konnten die Anzahl, die Positionen und die genetischen Effekte der QTL für die erhobenen Merkmale bestimmt werden. Hierzu wurde das Verfahren der Intervallkartierung unter Einbeziehung von Kofaktoren verwendet. Darüber hinaus wurden die ursprünglichen Daten aus anderen Feldexperimenten zur Untersuchung der Resistenz gegen zwei weitere bedeutende Krankheitserreger, Ährenfusarium und Spelzenbräune, verwendet, um QTL für multiple Krankheitsresistenz innerhalb der Populationen Arina/Forno und History/Rubens zu entdecken.

Die Ergebnisse der inokulierten Parzellen stimmten gut mit den Boniturwerten in natürlich infizierten Parzellen überein ($r = 0,84$ bis $0,99$, $P < 0,01$). Daraus lässt sich schließen, dass die eingesetzte Inokulationsmethodik geeignet war, um den Befall mit STB im Feld auch unter ungünstigen Bedingungen zu gewährleisten und eine ähnliche Differenzierung der Weizensorten wie in der Praxis zu ermöglichen. Zwischen den 24 Weizensorten variierte die infizierte Fahnenblattfläche von 8 % (Solitär) bis 63 % (Rubens) im Mittel über neun Umwelten. Die Varianzanalyse ergab einen hoch signifikanten ($P < 0,01$) Effekt des Genotyps und der G x U-Interaktion. Aus diesem Grund ist die Umweltstabilität der Sorten ein wichtiges Zuchtziel. In einem Regressionsansatz zeigten die Sorten Solitär, History und Florett die höchste Umweltstabilität. Im Gegensatz dazu schwankte der Befall der Sorte Biscay zwischen 19 und 72 % innerhalb der Umwelten über drei Versuchsjahre.

Die varianzanalytische Auswertung der phänotypischen Daten ergab sowohl innerhalb der fünf Kartierungspopulationen als auch zwischen deren Eltern eine hoch signifikante ($P < 0,01$) genotypische Differenzierung für alle drei Merkmale STB, AES und WUH. Die

Heritabiltät (h^2) für STB lag zwischen 0,69 und 0,87, außer bei Tuareg/Biscay (h^2 = 0,38). Die Heritabilitäten für AES (h^2 = 0,78 bis 0,93) und WUH (h^2 = 0,92 bis 0,98) waren in allen Populationen sehr hoch. Die Korrelationen zwischen STB und AES (r = -0,18 bis -0,33) als auch zwischen STB und WUH (r = -0,13 bis -0,45) waren negativ und signifikant (P < 0,01), aber von geringer Bedeutung. Die einzige Ausnahme war die Population History/Rubens, die am für die Wuchshöhe relevanten *Rht-D1*-Locus aufspaltet und deshalb eine deutlich höhere signifikant negative Korrelation zwischen STB und WUH aufwies (r = -0,55, P < 0,01). Alle fünf Kartierungspopulationen zeigten im Adultpflanzenstadium eine breite und kontinuierliche Häufigkeitsverteilung der Befallsmittelwerte aus Feldversuchen in drei bis sechs Umwelten.

In der QTL-Analyse wurden in den fünf Weizenpopulationen für die drei Merkmale STB, AES und WUH ein bis neun, null bis neun und vier bis elf QTL detektiert. Für die Resistenz gegen STB wurden in jeder Population ein bis zwei Major-QTL kartiert (*QStb.lsa_fb-3B, QStb.lsa_fb-6D, QStb.lsa_tb-4B, QStb.lsa_tb-6B, QStb.lsa_hr-4D, QStb.lsa_hr-5B.1, QStb.lsa_af-3B, QStb.lsa_bs-7A*), die jeweils mehr als 10 % der normalisierten adjustierten phänotypischen Varianz erklärten. Insgesamt erklärten alle 26 gefundenen Resistenz QTL zusammen 14 bis 55 % der adjustierten phänotypischen Varianz in den fünf Populationen. Resistenzallele stammten vom resistenten aber auch vom anfälligen Elter, während sie bei den gefundenen Major-QTL alle vom resistenten Elter kamen.

Die abschließende Meta-Analyse entdeckte acht Loci für multiple Krankheitsresistenz, vier in Arina/Forno auf den Chromosomen 3B, 4B, 5B und 6D, sowie vier in History/Rubens auf den Chromosomen 2B, 4D, 5B und 7B. Den mit Abstand größten Effekt zeigte der Meta-QTL auf Chromosom 4D in History/Rubens, der eng gekoppelt ist mit *Rht-D1*. Die Resistenz von History reduziert sowohl den Fahnenblattbefall mit STB um 9,8 % als auch den Ährenbefall mit Fusarium um 6,3 % und erklärt damit jeweils 47 % bzw. 60 % der partiellen phänotypischen Varianz.

Die Studie hat gezeigt, dass in europäischen Weizensorten eine für die Züchtung nutzbare, breite genetische Variation für die Resistenz gegen STB vorhanden ist. Obwohl der Einfluss der Umwelt als auch die G x U-Interaktion bedeutend waren, wurden einige resis-

tente Sorten mit hoher Umweltstabilität gefunden (Solitär, History, Florett). Auf 13 von 21 Chromosomen des Weizens wurden QTL für die Resistenz gegen STB kartiert. Die kontinuierliche Häufigkeitsverteilung des Fahnenblattbefalls in allen fünf segregierenden Populationen zusammen mit der großen Anzahl von 26 gefundenen QTL lässt darauf schließen, dass die Adultpflanzenresistenz gegen *S. tritici* quantitativ vererbt wird. Die hier vorgestellte QTL-Meta-Analyse innerhalb zweier Kartierungspopulationen über die drei bedeutenden Pathogene Ährenfusarium, Spelzenbräune und STB konnte acht Loci mit eng gekoppelten Markern für multiple Krankheitsresistenz detektieren. Ein vielversprechender Ansatz in der Resistenzzüchtung ist die Anwendung der markergestützen Selektion. Dadurch ist es möglich sowohl die gefundenen Major-QTL als auch die Meta-QTL im Zuchtmaterial zu kombinieren und somit neue Sorten mit verbesserter Resistenz gegenüber Septoria-Blattdürre, Ährenfusarium und Spelzenbräune zu züchten.

7 REFERENCES

Abate ZA, Liu S, McKendry AL (2008) Quantitative trait loci associated with deoxynivalenol content and kernel quality in the soft red winter wheat 'Ernie'. Crop Sci 48:1408-1418

Adhikari TB, Anderson JM, Goodwin SB (2003) Identification and molecular mapping of a gene in wheat conferring resistance to *Mycosphaerella graminicola*. Phytopathology 93:1158-1164

Adhikari TB, Cavaletto JR, Dubcovsky J, Gieco JO, Schlatter AR, Goodwin SB (2004a) Molecular mapping of the *Stb4* gene for resistance to Septoria tritici blotch in wheat. Phytopathology 94:1198-1206

Adhikari TB, Wallwork H, Goodwin SB (2004b) Microsatellite markers linked to the *Stb2* and *Stb3* genes for resistance to septoria tritici blotch in wheat. Crop Sci 44:1403-1411

Adhikari TB, Yang X, Cavaletto JR, Hu X, Buechley G, Ohm HW, Shaner G, Goodwin SB (2004c) Molecular mapping of *Stb1*, a potentially durable gene for resistance to septoria tritici blotch in wheat. Theor Appl Genet 109:944-953

Akbari M, Wenzl P, Caig V, Carling J, Xia L, Yang S, Uszynski G, Mohler V, Lehmensiek A, Kuchel H, Hayden MJ, Howes N, Sharp P, Vaughan P, Rathmell B, Huttner E, Kilian A (2006) Diversity Arrays Technology (DArT) for high-throughput profiling of the hexaploid wheat genome. Theor Appl Genet 113:1409-1420

Ali ML, Taylor JH, Jie L, Sun G, William M, Kasha KJ, Reid LM, Pauls KP (2005) Molecular mapping of QTLs for resistance to *Gibberella* ear rot, in corn, caused by *Fusarium graminearum*. Genome 48:521-533

Arama PF, Parlevliet JE, van Silfhout CH (1999) Heading date and resistance to septoria tritici blotch in wheat not genetically associated. Euphytica 106:63-68

Arraiano LS, Brading PA, Brown JKM (2001a) A detached seedling leaf technique to study resistance to *Mycosphaerella graminicola* (anamorph *Septoria tritici*) in wheat. Plant Pathol 50:339-346

Arraiano LS, Worland AJ, Ellerbrook C, Brown JKM (2001b) Chromosomal location of a gene for resistance to septoria tritici blotch (*Mycosphaerella graminicola*) in the hexaploid wheat 'Synthetic 6x'. Theor Appl Genet 103:758-764

Arraiano LS, Brading PA, Dedryver F, Brown JKM (2006) Resistance of wheat to septoria tritici blotch (*Mycosphaerella graminicola*) and associations with plant ideotype and the 1BL-1RS translocation. Plant Pathol 55:54-61

Arraiano LS, Brown JKM (2006) Identification of isolate-specific and partial resistance to Septoria tritici blotch in 238 European wheat cultivars and breeding lines. Plant Pathol 55:726-738

Arraiano LS, Chartrain L, Bossolini E, Slatter HN, Keller B, Brown JKM (2007) A gene in European wheat cultivars for resistance to an African isolate of *Mycosphaerella graminicola*. Plant Pathol 56:73-78

Arraiano LS, Balaam N, Fenwick PM, Chapman C, Feuerhelm D, Howell P, Smith SJ, Widdowson JP, Brown JKM (2009) Contributions of disease resistance and escape to the control of Septoria tritici blotch of wheat. Plant Pathol 58:910-922

Asíns MJ (2002) Present and future of quantitative trait locus analysis in plant breeding. Plant Breeding 121:281-291

Baltazar BM, Scharen AL, Kronstad WE (1990) Association between dwarfing genes '*Rht1*' and '*Rht2*' and resistance to Septoria tritici blotch in winter wheat (*Triticum aestivum* L. em Thell). Theor Appl Genet 79:422-426

Beavis WD (1998) QTL analysis: Power, precision, and accuracy. In: Paterson AH (eds) Molecular dissection of complex traits. Boca Raton, USA, CRC Press, pp 145-162

Becker HC, Léon J (1988) Stability analysis in plant breeding. Plant Breeding 101:1-23

Bennetzen JL, Ma J (2003) The genetic colinearity of rice and other cereals on the basis of genomic sequence analysis. Curr Opin Plant Biol 6:128-133

Bohn M, Khairallah MM, González-de-León D, Hoisington DA, Utz HF, Deutsch JA, Jewell DC, Mihm JA, Melchinger AE (1996) QTL mapping in tropical maize: I. Genomic regions affecting leaf feeding resistance to sugarcane borer and other traits. Crop Sci 36:1352-1361

Bonin CM, Kolb FL (2009) Resistance to Fusarium head blight and kernel damage in a winter wheat recombinant inbred line population. Crop Sci 49:1304-1312

Bonjean AP, Angus WJ, editors (2001) The world wheat book : A history of wheat breeding. Paris, France, Intercept, pp 1131

Brading PA, Verstappen ECP, Kema GHJ, Brown JKM (2002) A gene-for-gene relationship between wheat and *Mycosphaerella graminicola*, the Septoria tritici blotch pathogen. Phytopathology 92:439-445

Brown JKM, Kema GHJ, Forrer HR, Verstappen ECP, Arraiano LS, Brading PA, Foster EM, Fried PM, Jenny E (2001) Resistance of wheat cultivars and breeding lines to Septoria tritici blotch caused by isolates of *Mycosphaerella graminicola* in field trials. Plant Pathol 50:325-338

Buerstmayr H, Steiner B, Lemmens M, Ruckenbauer P (2000) Resistance to fusarium head blight in winter wheat: heritability and trait associations. Crop Sci 40:1012-1018

Buerstmayr H, Lemmens M, Hartl L, Doldi L, Steiner B, Stierschneider M, Ruckenbauer P (2002) Molecular mapping of QTLs for Fusarium head blight resistance in spring wheat. I. Resistance to fungal spread (Type II resistance). Theor Appl Genet 104:84-91

Bundessortenamt (2009) Beschreibende Sortenliste Getreide, Mais, Ölfrüchte, Leguminosen (großkörnig), Hackfrüchte (außer Kartoffeln). Hannover, Germany, Bundessortenamt, pp 273

Chartrain L, Brading PA, Makepeace JC, Brown JKM (2004a) Sources of resistance to Septoria tritici blotch and implications for wheat breeding. Plant Pathol 53:454-460

Chartrain L, Brading PA, Widdowson JP, Brown JKM (2004b) Partial resistance to Septoria tritici blotch (*Mycosphaerella graminicola*) in wheat cultivars Arina and Riband. Phytopathology 94:497-504

Chartrain L, Berry ST, Brown JKM (2005a) Resistance of wheat line Kavkaz-K4500 L.6.A.4 to Septoria tritici blotch controlled by isolate-specific resistance genes. Phytopathology 95:664-671

Chartrain L, Brading PA, Brown JKM (2005b) Presence of the *Stb6* gene for resistance to Septoria tritici blotch (*Mycosphaerella graminicola*) in cultivars used in wheat-breeding programmes worldwide. Plant Pathol 54:134-143

Chartrain L, Joaquim P, Berry ST, Arraiano LS, Azanza F, Brown JKM (2005c) Genetics of resistance to Septoria tritici blotch in the Portuguese wheat breeding line TE 9111. Theor Appl Genet 110:1138-1144

Churchill GA, Doerge RW (1994) Empirical threshold values for quantitative trait mapping. Genetics 138:963-971

Cools H, J, Fraaije B, A (2008) Are azole fungicides losing ground against Septoria wheat disease? Resistance mechanisms in *Mycosphaerella graminicola*. Pest Manag Sci 64:681-684

Darvasi A, Soller M (1997) A simple method to calculate resolving power and confidence interval of QTL map location. Behav Genet 27:125-132

Ding JQ, Wang X, Chander S, Yan J, Li J (2008) QTL mapping of resistance to Fusarium ear rot using a RIL population in maize. Mol Breed 22:395-403

Draeger R, Gosman N, Steed A, Chandler E, Thomsett M, Srinivasachary, Schondelmaier J, Buerstmayr H, Lemmens M, Schmolke M, Mesterhazy A, Nicholson P (2007) Identification of QTLs for resistance to Fusarium head blight, DON accumulation and associated traits in the winter wheat variety Arina. Theor Appl Genet 115:617-625

Eberhart SA, Russell WA (1966) Stability parameters for comparing varieties. Crop Sci 6:36-40

Eriksen L, Borum F, Jahoor A (2003) Inheritance and localisation of resistance to *Mycosphaerella graminicola* causing septoria tritici blotch and plant height in the wheat (*Triticum aestivum* L.) genome with DNA markers. Theor Appl Genet 107:515-527

Eyal Z, Scharen AL, Prescott JM, van Ginkel M (1987) The Septoria diseases of wheat: Concepts and methods of disease management. Mexiko, CIMMYT, pp 46

FAOSTAT (2007) Food and agriculture organization of the United Nations. Online, 5 Mar 2010, http://faostat.fao.org/

Flor HH (1971) Current status of the gene-for-gene concept. Annu Rev Phytopathol 9:275-296

Freeman GH (1973) Statistical methods for the analysis of genotype-environment interactions. Heredity 31:339-354

Goodwin SB, Cavaletto JR, Lowe I, Thompson I, Xu SX, Adhikari TB, Dubcovsky J (2007) Validation of a new map location for the *Stb3* gene for resistance to Septoria tritici blotch in wheat. 7th International Mycosphaerella and Stagonospora Symposium, Ascona, Switzerland, poster presentation, Abstract, p 1

Griffiths S, Simmonds J, Leverington M, Wang Y, Fish L, Sayers L, Alibert L, Orford S, Wingen L, Herry L, Faure S, Laurie D, Bilham L, Snape J (2009) Meta-QTL analysis of the genetic control of ear emergence in elite European winter wheat germplasm. Theor Appl Genet 119:383-395

Haldane JBS (1919) The combination of linkage values and the calculation of distances between the loci of linked factors. J Genet 8:299-309

Hanocq E, Laperche A, Jaminon O, Lainé A, Le Gouis J (2007) Most significant genome regions involved in the control of earliness traits in bread wheat, as revealed by QTL meta-analysis. Theor Appl Genet 114:569-584

Holzapfel J, Voss HH, Miedaner T, Korzun V, Häberle J, Schweizer G, Mohler V, Zimmermann G, Hartl L (2008) Inheritance of resistance to Fusarium head blight in three European winter wheat populations. Theor Appl Genet 117:1119-1128

Hospital F, Moreau L, Lacoudre F, Charcosset A, Gallais A (1997) More on the efficiency of marker-assisted selection. Theor Appl Genet 95:1181-1189

Jansen RC (1993) Interval mapping of multiple quantitative trait loci. Genetics 135:205-211

Jlibene M, Gustafson JP, Rajaram S (1992) A field disease evaluation method for selecting wheats resistant to *Mycosphaerella graminicola*. Plant Breeding 108:26-32

Keller B, Feuillet C, Messmer M (2000) Genetics of disease resistance. In: Slusarenko AJ, Fraser RSS, van Loon LC (eds) Mechanisms of resistance to plant diseases. Kluwer Academic Publishers, pp 101-160

Kema GHJ, Annone JG, Sayoud R, Van Silfhout CH, Van Ginkel M, de Bree J (1996a) Genetic variation for virulence and resistance in the wheat-*Mycosphaerella graminicola* pathosystem I. Interactions between pathogen isolates and host cultivar. Phytopathology 86:200-212

Kema GHJ, Sayoud R, Annone JG, Van Silfhout CH (1996b) Genetic variation for virulence and resistance in the wheat-*Mycosphaerella graminicola* pathosystem II. Analysis of interactions between pathogen isolates and host cultivar. Phytopathology 86:213-220

Kema GHJ, van Silfhout CH (1997) Genetic variation for virulence and resistance in the wheat-*Mycosphaerella graminicola* pathosystem III. Comparative seedling and adult plant experiment. Phytopathology 87:266-272

Knapp SJ, Stroup WW, Ross WM (1985) Exact confidence intervals for heritability on a progeny mean basis. Crop Sci 25:192-194

Landjeva S, Korzun V, Börner A (2007) Molecular markers: actual and potential contributions to wheat genome characterization and breeding. Euphytica 156:271-296

Linde CC, Zhan J, McDonald BA (2002) Population structure of *Mycosphaerella graminicola*: From lesions to continents. Phytopathology 92:946-955

Liu BH (1998) Statistical genomics: Linkage, mapping, and QTL analysis. Boca Raton, USA, CRC Press, pp 611

Liu S, Abate Z, Lu H, Musket T, Davis G, McKendry A (2007) QTL associated with Fusarium head blight resistance in the soft red winter wheat Ernie. Theor Appl Genet 115:417-427

Liu S, Hall MD, Griffey CA, McKendry AL (2009) Meta-analysis of QTL associated with Fusarium head blight resistance in wheat. Crop Sci 49:1955-1968

Löffler M, Schön C, Miedaner T (2009) Revealing the genetic architecture of FHB resistance in hexaploid wheat (*Triticum aestivum* L.) by QTL meta-analysis. Mol Breed 23:473-488

Mao S, Wei Y, Cao W, Lan X, Yu M, Chen Z, Chen G, Zheng Y (2010) Confirmation of the relationship between plant height and Fusarium head blight resistance in wheat (*Triticum aestivum* L.) by QTL meta-analysis. Euphytica online, 8 Feb 2010, DOI 10.1007/s10681-010-0128-9, pp. 14

Martinez O, Curnow RN (1992) Estimating the locations and the sizes of the effects of quantitative trait loci using flanking markers. Theor Appl Genet 85:480-488

McCartney CA, Brule-Babel AL, Lamari L, Somers DJ (2003) Chromosomal location of a race-specific resistance gene to Mycosphaerella graminicola in the spring wheat ST6. Theor Appl Genet 107:1181-1186

McDonald BA, Zhan J, Yarden O, Hogan K, Garton J, Pettway RE (1999) The population genetics of *Mycosphaerella graminicola* and *Stagnospora nodorum* . In: Lucas JA, Bowyer P, Anderson HM (eds) Septoria on cereals: a study of pathosystems. Bristol, UK, CABI, pp 44-69

McDonald BA, Linde C (2002) Pathogen population genetics, evolutionary potential, and durable resistance. Annu Rev Phytopathol 40:349-379

McGill R, Tukey JW, Larsen WA (1978) Variations of box plots. The American Statistician 32:12-16

Meier U (2001) Growth stages of mono- and dicotyledonous plants. 2nd edn. Braunschweig, Germany, Federal Biological Research Centre for Agriculture and Forestry, online, 16 Feb 2010, http://www.jki.bund.de/fileadmin/dam_uploads/_veroeff/bbch/BBCH-Skala_englisch.pdf

Melchinger AE, Utz HF, Schön CC (2004) QTL analyses of complex traits with cross validation, bootstrapping and other biometric methods. Euphytica 137:1-11

Miedaner T, Flath K (2007) Effectiveness and environmental stability of quantitative powdery mildew (*Blumeria graminis*) resistance among winter wheat cultivars. Plant Breeding 126:553-558

Miedaner T (2009) Züchtung und Biotechnologie. In: Christen O (eds) Winterweizen: Das Handbuch für Profis. Frankfurt, DLG-Verlags-GmbH, pp 11-37

Paillard S, Schnurbusch T, Winzeler M, Messmer M, Sourdille P, Abderhalden O, Keller B, Schachermayr G (2003) An integrative genetic linkage map of winter wheat (*Triticum aestivum* L.). Theor Appl Genet 107:1235-1242

Paillard S, Schnurbusch T, Tiwari R, Messmer M, Winzeler M, Keller B, Schachermayr G (2004) QTL analysis of resistance to Fusarium head blight in Swiss winter wheat (*Triticum aestivum* L.). Theor Appl Genet 109:323-332

Palmer CL, Skinner W (2002) *Mycosphaerella graminicola*: latent infection, crop devastation and genomics. Molecular Plant Pathology 3:63-70

Parlevliet JE (1977) Plant pathosystems: An attempt to elucidate horizontal resistance. Euphytica 26:553-556

Parlevliet JE, Zadoks JC (1977) The integrated concept of disease resistance: A new view including horizontal and vertical resistance in plants. Euphytica 26:5-21

Pérez-Brito D, Jeffers D, González-de-León D, Khairallah M, Cortés-Cruz M, Velázquez-Cardelas G, Azpıroz-Rivero S, Srinivasan G (2001) QTL mapping of Fusarium moniliforme ear rot resistance in highland maize, Mexico. Agrociencia 35:181-196

Pestsova E, Ganal MW, Röder MS (2000) Isolation and mapping of microsatellite markers specific for the D genome of bread wheat. Genome 43:689-697

Risser P (2007) Untersuchung der Isolatspezifität im Pathosystem Weizen/*Septoria tritici* bei Feldinokulation. Master thesis 1-44

Robertson-Hoyt LA, Jines MP, Balint-Kurti PJ, Kleinschmidt CE, White DG, Payne GA, Maragos CM, Molnar TL, Holland JB (2006) QTL mapping for Fusarium ear rot and fumonisin contamination resistance in two maize populations. Crop Sci 46:1734-1743

Röder MS, Korzun V, Wendehake K, Plaschke J, Tixier M, Leroy P, Ganal MW (1998) A microsatellite map of wheat. Genetics 149:2007-2023

Rosielle AA (1972) Sources of resistance in wheat to speckled leaf blotch caused by *Septoria tritici* . Euphytica 21:152-161

Rosielle AA, Brown AGP (1979) Inheritance, heritability and breeding behaviour of three sources of resistance to *Septoria tritici* in wheat. Euphytica 28:385-392

Salamini F, Ozkan H, Brandolini A, Schafer-Pregl R, Martin W (2002) Genetics and geography of wild cereal domestication in the Near East. Nature Reviews Genetics 3:429-441

Schilly A (2009) Genotyp x Isolat-Interaktion im Pathosystem Weizen / in neun Feldumwelten. Bachelor thesis 1-40

Schmolke M, Zimmermann G, Buerstmayr H, Schweizer G, Miedaner T, Korzun V, Ebmeyer E, Hartl L (2005) Molecular mapping of Fusarium head blight resistance in the winter wheat population Dream/Lynx. Theor Appl Genet 111:747-756

Schnurbusch T, Paillard S, Fossati D, Messmer M, Schachermayr G, Winzeler M, Keller B (2003) Detection of QTLs for Stagonospora glume blotch resistance in Swiss winter wheat. Theor Appl Genet 107:1226-1234

Schön CC, Utz HF, Groh S, Truberg B, Openshaw S, Melchinger AE (2004) Quantitative trait locus mapping based on resampling in a vast maize testcross experiment and its relevance to quantitative genetics for complex traits. Genetics 167:485-498

Semagn K, Bjørnstad Å, Skinnes H, Marøy AG, Tarkegne Y, William M (2006) Distribution of DArT, AFLP, and SSR markers in a genetic linkage map of a doubled-haploid hexaploid wheat population. Genome 49:545-555

Semagn K, Skinnes H, Bjrnstad A, Maroy AG, Tarkegne Y (2007) Quantitative trait loci controlling Fusarium head blight resistance and low deoxynivalenol content in hexaploid wheat population from 'Arina' and NK93604. Crop Sci 47:294-303

Shankar M, Walker E, Golzar H, Loughman R, Wilson RE, Francki MG (2008) Quantitative trait loci for seedling and adult plant resistance to *Stagonospora nodorum* in wheat. Phytopathology 98:886-893

Shaw MW, Royle DJ (1989) Airborne inoculum as a major source of *Septoria tritici* (*Mycosphaerella graminicola*) infections in winter wheat crops in the UK. Plant Pathol 38:35-43

Shaw MW (1991) Interacting effects of interrupted humid periods and light on infection of wheat leaves by *Mycosphaerella graminicola* (*Septoria tritici*). Plant Pathol 40:595-607

Shaw MW, Royle DJ (1993) Factors determining the severity of epidemics of *Mycosphaerella graminicola (Septoria tritici)* on winter wheat in the UK. Plant Pathol 42:882-899

Simón MR, Worland AJ, Struik PC (2004) Influence of plant height and heading date on the expression of the resistance to Septoria tritici blotch in near isogenic lines of wheat. Crop Sci 44:2078-2085

Somers D, Isaac P, Edwards K (2004) A high-density microsatellite consensus map for bread wheat (*Triticum aestivum* L.). Theor Appl Genet 109:1105-1114

Torriani S, FF, Brunner P, C, McDonald B, A, Sierotzki H (2009) QoI resistance emerged independently at least 4 times in European populations of *Mycosphaerella graminicola*. Pest Manag Sci 65:155-162

Uphaus J, Walker E, Shankar M, Golzar H, Loughman R, Francki M, Ohm H (2007) Quantitative trait loci identified for resistance to Stagonospora glume blotch in wheat in the USA and Australia. Crop Sci 47:1813-1822

Utz HF, Melchinger AE (1996) PLABQTL: A program for composite interval mapping of QTL. Journal of Agricultural Genomics 2:1-5

Utz HF, Melchinger AE, Schön CC (2000) Bias and sampling error of the estimated proportion of genotypic variance explained by quantitative trait loci determined from experimental data in maize using cross validation and validation with independent samples. Genetics 154:1839-1849

Utz HF (2001) Plabstat, ein Computerprogramm zur statistischen Analyse pflanzenzüchterischer Experimente.

Van Ginkel M, Scharen AL (1987) Generation mean analysis and heritabilities of resistance to *Septoria tritici* in durum wheat. Phytopathology 77:1629-1633

Van Ooijen JW, Voorrips RE (2001) JoinMap® 3.0, Software for the calculation of genetic linkage maps. Plant Research International, Wageningen, the Netherlands.

Voorrips RE (2002) MapChart: Software for the graphical presentation of linkage maps and QTLs. J Hered 93:77-78

Vos P, Hogers R, Bleeker M, Reijans M, Van de Lee T, Hornes M, Frijters A, Pot J, Peleman J, Kuiper M, Zabeau M (1995) AFLP: A new technique for DNA fingerprinting. Nucleic Acids Res 23:4407-4414

Voss HH, Holzapfel J, Hartl L, Korzun V, Rabenstein F, Ebmeyer E, Coester H, Kempf H, Miedaner T (2008) Effect of the *Rht-D1* dwarfing locus on Fusarium head blight rating in three segregating populations of winter wheat. Plant Breeding 127:333-339

Vuylsteke M, Peleman JD, van Eijk MJT (2007) AFLP technology for DNA fingerprinting. Nat Protoc 2:1387-1398

Waldron BL, Moreno-Sevilla B, Anderson JA, Stack RW, Frohberg RC (1999) RFLP mapping of QTL for Fusarium head blight resistance in wheat. Crop Sci 39:805-811

Westcott B (1986) Some methods of analysing genotype-environment interaction. Heredity 56:243-253

Zadoks JC, Chang TT, Konzak CF (1974) A decimal code for the growth stages of cereals. Weed Res 14:415-421

Zhan J, Pettway RE, McDonald BA (2003) The global genetic structure of the wheat pathogen *Mycosphaerella graminicola* is characterized by high nuclear diversity, low mitochondrial diversity, regular recombination, and gene flow. Fungal Genetics and Biology 38:286-297

Zhu S, Rossnagel BG, Kaeppler HF (2004) Genetic analysis of quantitative trait loci for groat protein and oil content in oat. Crop Sci 44:254-260

8 SUPPLEMENT

S 1: Test of wheat varieties in seedling test with set of international isolates for resistance to Septoria tritici blotch (% leaf necrosis); Isolates used in field trials are highlighted

Septoria tritici isolates [1]		Varieties in seedling test [2]										
Designation	**Origin**	**Arina**	**Biscay**	**Bussard**	**Florett**	**Forno**	**History**	**Julius**	**Rubens**	**Solitär**	**Tuareg**	**Mean**
BAZ 6/1/04	Germany	90	100	80	70	100	100	100	100	95	58	89
BAZ 8/8/04	Germany	100	90	100	85	100	95	55	100	90	85	90
IPO 323	Netherlands	8	80	100	3	33	80	0	0	3	0	31
IPO 2166	Iran	35	100	100	3	100	75	50	100	75	50	69
IPO 86013	Turkey	50	83	90	70	70	68	60	100	95	63	75
IPO 88004	Ethopia	10	100	85	3	100	88	85	100	100	3	67
IPO 90006	Mexiko	15	55	85	70	100	65	53	80	50	45	62
IPO 90015	Peru	80	48	100	75	100	100	90	100	60	95	85
IPO 92034	Algeria	100	100	100	95	100	100	90	100	85	88	96
IPO 94218	Canada	8	100	100	95	100	90	100	100	85	40	82
IPO 95054	Algeria	58	80	100	23	100	75	75	100	100	100	81
IPO 98022	France	85	100	100	85	50	100	93	100	93	80	89
IPO 98033	France	75	100	100	75	100	100	85	100	100	75	91
IPO 98042	France	75	80	95	63	95	85	95	95	90	73	85
IPO 98050	France	45	100	100	85	95	100	30	100	60	100	82
IPO 98051	France	70	90	100	30	100	100	100	80	100	70	84
IPO 99018	France	78	100	100	90	85	95	95	100	80	85	91
IPO 99031	France	88	100	100	90	100	100	100	100	100	75	95
Mean		59	89	96	62	90	90	75	92	81	66	80

[1] BAZ 6/1/04 and BAZ 8/8/04 were also used in field trials and were provided by Julius-Kühn-Institut, Germany; IPO isolates were provided by G. J. Kema, the Netherlands

[2] Seedling test by G. J. Kema, Wageningen, the Netherlands; % leaf necrosis on the primary seedling leaves (21 dpi)

S 2: Localisation and effect of QTL with LOD > 3.0 for plant height (cm) in five wheat populations (means across environments); major QTL explaining ≥ 10 % of phenotypic variance are highlighted

No.	Chrom.	Donor of shortening	Pos. (cM)	Flanking marker [1] left	Flanking marker [1] right	d [2] (cM)	CI [3] (cM)	Effect [4] (cm)	nR^2_{adj} [5] (%)	QTL x E [6]
Florett/Biscay (N = 301)										
1	2B	Florett	4	XP2553-222	**Xwmc0344**	4	0- 15	**-1.0**	3.9	*
2	3B	Biscay	44	**XP2553-237**	Xstb10	6	33- 55	**-1.4**	5.3	**
3	5A	Florett	40	XwPt-3924	**XP1949-263**	1	28- 41	**-1.6**	9.1	
4	5B	Florett	36	XP1856-231	**Xbarc0059**	3	28- 44	**-1.5**	6.9	
								Total R^2_{adj} (%)	**25.3**	
Tuareg/Biscay (N = 263)										
1	1D	Tuareg	8	**XwPt-7946**	XwPt-9664	0	0- 20	**-0.6**	2.8	*
2	2B	Tuareg	104	XP1952-142	**XwPt-4917**	2	97- 110	**-1.0**	6.3	
3	2D	Biscay	4	**Xwmc0112**	XP1761-389	0	1- 7	**-1.6**	13.2	
4	3A	Biscay	6	XP2553-147	**XwPt-7890**	0	0- 25	**-0.8**	3.8	
5	3A	Biscay	74	**Xbarc0045**	Xwmc0264	1	73- 75	**-2.8**	25.2	
6	3B	Tuareg	76	**XP1952-152**	XwPt-6187	5	64- 88	**-1.3**	8.0	
7	5A	Tuareg	0	**Xwmc0096**	XwPt-3620	0	0- 1	**-1.0**	6.5	
8	6A	Biscay	86	Xgwm0570	**Xwmc0201**	1	77- 87	**-0.7**	3.6	
								Total R^2_{adj} (%)	**69.5**	
History/Rubens (N = 94)										
1	2B [ns 7]	History	160	XwPt-7859	**XwPt-2397**	2	149- 162	**-3.3**	9.7	
2	4D	Rubens	4	***Rht-D1***	Xbarc0105	0	1- 7	**-8.6**	29.9	**
3	5A [ns]	History	10	**Xgwm0617**	XwPt-4419	0	6- 14	**-3.6**	12.0	**
4	6A [ns]	Rubens	66	Xgwm0082a	**Xgwm0082b**	0	61- 71	**-2.6**	7.1	
5	7A [ns]	Rubens	60	**Xgwm0276a**	Xgwm0276b	0	55- 65	**-3.0**	9.6	
								Total R^2_{adj} (%)	**68.4**	
Arina/Forno (N = 200)										
1	1A	Forno	62	Xcfd0058	**Xgwm0357**	0	52- 72	**-1.5**	3.9	
2	1B	Arina	48	Xpsr0642	**XOA093**	0	43- 53	**-2.1**	6.6	
3	1D	Arina	64	**Xgdm0019**	Xcfd0019	0	56- 72	**-1.6**	4.7	**
4	2A	Forno	192	**Xgwm0526**	Xcfa2086	1	189- 195	**-2.5**	8.5	**
5	2B	Arina	172	Xgwm0526	**Xpsr0644**	3	169- 175	**-1.9**	3.9	*
6	4A	Arina	102	Xpsr0618	**Xpsr0160**	0	89- 105	**-1.2**	2.6	**
7	4B	Arina	30	**Xgwm0006**	Xgwm0538	0	23- 37	**-1.5**	4.1	
8	5A	Forno	72	Xglk0317	**Xpsr0386**	0	69- 75	**-3.5**	14.4	**
9	5B	Arina	72	Xpsr0120	**Xcfd0007**	0	65- 72	**-1.5**	3.8	
10	6A [ns]	Arina	134	XOA097	**Xnils05**	0	118- 150	**-1.5**	3.6	
11	6D	Forno	120	Xcfd0019	**Xbarc0273**	0	113- 120	**-1.5**	3.8	
								Total R^2_{adj} (%)	**60.0**	

S 2: Continued

No.	Chrom.	Donor of shortening	Pos. (cM)	Flanking marker [1] left	Flanking marker [1] right	d [2] (cM)	CI [3] (cM)	Effect [4] (cm)	nR^2_{adj} [5] (%)	QTL x E [6]
Bussard/Solitär (N = 81)										
1	3A [ns]	Bussard	24	XwPt-1688.1	**Xwmc0532**	3	17- 31	**-2.5**	8.4	
								Total R^2_{adj} (%)	**8.4**	

[1] Closest marker in bold

[2] Distance in cM to the next flanking marker

[3] 95 % confidence interval after Davarsi and Soller (1997)

[4] Estimated additive effects in final simultaneous fit of the QTL allele in the series of field trials; numbers reflect reduced height

[5] Normalized partial phenotypic variance explained by detected QTL

[6] QTL-by-environment interaction tested for significance (sequentially rejective Bonferroni F-test)

[7] LOD > 3.0 but not significant (ns) according to critical LOD score after 1,000 permutations (α = 10 %)

** F-Test significant at P < 0.01

* F-Test significant at P < 0.05

S 3: Localisation and effect of QTL with LOD > 3.0 for heading date (days in year) in five wheat populations (means across environments); major QTL explaining ≥ 10 % of phenotypic variance are highlighted

No.	Chrom.	Donor of prematurity	Pos. (cM)	Flanking marker [1] left	Flanking marker [1] right	d [2] (cM)	CI [3] (cM)	Effect [4] (days)	nR^2_{adj} [5] (%)	QTL x E [6]
Florett/Biscay (N = 301)										
1	1D	Florett	12	**XP1761-205**	XP1952-198	12	7- 17	**-0.5**	5.2	
2	1D [ns 7]	Florett	110	Xcfd0072	**Xgwm0458**	1	93- 111	**-0.3**	2.3	
3	2A	Biscay	20	Xcfd0036	**Xwmc0177**	2	6- 34	**-0.3**	3.4	
4	2B	Florett	18	**XwPt-1140**	XwPt-3132	1	10- 26	**-0.4**	4.6	**
5	2D	Florett	42	XP1761-393	**Xwmc0503**	1	38- 46	**-0.6**	9.9	
6	3B	Florett	40	**XP2553-237**	Xstb10	2	26- 54	**-0.3**	3.8	
7	4A	Biscay	36	XP1354-235	**XP1954-118**	2	18- 38	**-0.3**	2.9	
8	5A [ns]	Biscay	34	**Xwmc0096**	XwPt-3924	0	21- 41	**-0.2**	2.3	
9	5D	Florett	50	**Xcfd0008**	XwPt-2256	0	39- 60	**-0.3**	4.5	
								Total R^2_{adj} (%)	**38.8**	
Tuareg/Biscay (N = 263)										
1	1B	Tuareg	4	XwPt-7160	**XP1753-152**	3	0- 15	**-0.4**	4.9	*
2	2D	Tuareg	30	**Xcfd0043**	XwPt-0330	0	23- 37	**-0.5**	6.4	**
3	3A	Tuareg	86	Xbarc0045	**Xwmc0264**	1	83- 89	**-0.7**	14.0	**
4	4A	Biscay	50	XwPt-5172	**Xwmc0262**	1	44- 51	**-0.5**	8.3	**
5	4D [ns]	Biscay	4	**Xgwm0129**	XP2255-265	1	0- 14	**-0.3**	4.0	
6	7D	Tuareg	50	Xbarc0184	**XwPt-1269**	0	45- 52	**-0.4**	6.3	**
								Total R^2_{adj} (%)	**43.8**	
History/Rubens (N = 94)										
1	1B [ns]	Rubens	190	XP6451-190	**Xgwm0140**	1	173- 192	**-0.6**	7.5	
2	2B [ns]	Rubens	60	Xgwm0257	**XP7753-186**	0	54- 66	**-0.8**	14.1	**
3	5A [ns]	Rubens	6	XP7661-461	**Xgwm0617**	3	0- 13	**-0.9**	13.3	**
4	7B [ns]	History	106	P7056-167	**P7455-119**	2	100- 112	**-0.5**	5.0	
5	7D [ns]	Rubens	10	**XwPt-1859**	Xbarc0111	0	2- 18	**-0.7**	10.1	
								Total R^2_{adj} (%)	**50.0**	

S 3: Continued

No.	Chrom.	Donor of prematurity	Pos. (cM)	Flanking marker [1] left	Flanking marker [1] right	d [2] (cM)	CI [3] (cM)	Effect [4] (days)	nR^2_{adj} [5] (%)	QTL x E [6]
Arina/Forno (N = 200)										
1	6D [ns]	Forno	116	Xpsr0915	**Xcfd0019**	0	104- 120	**-0.4**	6.5	
2	7B	Forno	58	**Xgwm0133**	Xpsr0955	0	54- 62	**-0.6**	10.0	**
								Total R^2_{adj} (%)	**16.5**	
Bussard/Solitär (N = 81)										
No QTL and no QTL effects found										

[1] Closest marker in bold

[2] Distance in cM to the next flanking marker

[3] 95 % confidence interval after Davarsi and Soller (1997)

[4] Estimated additive effects in final simultaneous fit of the QTL allele in the series of field trials; numbers reflect earlier heading

[5] Normalized partial phenotypic variance explained by detected QTL

[6] QTL-by-environment interaction tested for significance (sequentially rejective Bonferroni F-test)

[7] LOD > 3.0 but not significant (ns) according to critical LOD score after 1,000 permutations (α = 10 %)

** F-Test significant at P < 0.01

* F-Test significant at P < 0.05

S 4: Additive effects of resistance alleles illustrating QTL-by-environment interaction for Septoria tritici blotch (% flag leaf area infected) in the populations Tuareg/Biscay, History/Rubens, Arina/Forno, and Bussard/Solitär; major QTL explaining ≥ 10 % of phenotypic variance are highlighted

QTL designation	Donor of resistance	Additive effect of resistance allele (%) [1]							Series
		2008				2009			
		FRE	HOH	OLI	WOH	FRE	HOH	OLI	
Tuareg/Biscay (N = 263)									
QStb.lsa_tb-1A	Biscay	-0.6	-	-3.6	-0.3	-1.4	-	-	**-1.5**
QStb.lsa_tb-4A	Tuareg	-1.6	-	-4.5	-1.3	-1.4	-	-	**-2.2**
QStb.lsa_tb-4B	Tuareg	-3.5	-	-11.5	-1.1	-2.1	-	-	**-4.0**
QStb.lsa_tb-4D	Tuareg	-2.1	-	-2.5	-1.4	-1.7	-	-	**-1.9**
QStb.lsa_tb-6B	Tuareg	-1.1	-	-6.4	-1.1	-4.5	-	-	**-3.3**
QStb.lsa_tb-7B	Tuareg	-0.7	-	-7.7	-0.9	-1.1	-	-	**-2.6**
	Total R^2_{adj} (%) [2]	**12.5**		**42.1**	**3.4**	**22.4**			**51.3**
History/Rubens (N = 94)									
QStb.lsa_hr-4D	History	-3.6	-16.8	-11.4	-	-5.5	-8.8	-14.7	**-10.2**
QStb.lsa_hr-5B.1	History	-5.8	-4.6	-7.7	-	-2.7	-6.5	-6.5	**-5.6**
QStb.lsa_hr-5B.2	History	-2.6	-6.1	-9.0	-	-0.5	-7.9	-6.9	**-5.5**
QStb.lsa_hr-6B	History	-2.1	-3.5	-6.2	-	-2.1	-6.3	-7.8	**-4.7**
QStb.lsa_hr-7B	History	-1.9	-5.4	-6.7	-	-1.5	-4.1	-5.6	**-4.2**
	Total R^2_{adj} (%)	**7.2**	**47.9**	**36.5**		**8.7**	**44.6**	**49.3**	**54.8**

S 4: Continued

QTL designation	Donor of resistance	Additive effect of resistance allele (%) [1]							
		2008				2009			Series
		FRE	HOH	OLI	WOH	FRE	HOH	OLI	
Arina/Forno (N = 200)									
QStb.lsa_af-2B	Forno	-	-	-	-	-1.4	-4.9	-6.5	**-4.3**
QStb.lsa_af-3B	Arina	-	-	-	-	-1.9	-6.2	-8.8	**-5.6**
QStb.lsa_af-5B	Arina	-	-	-	-	-3.9	-1.9	-4.7	**-3.5**
QStb.lsa_af-6D	Arina	-	-	-	-	-2.3	-5.9	-5.3	**-4.5**
QStb.lsa_af-7B	Forno	-	-	-	-	-0.1	-5.3	-4.3	**-3.1**
Total R^2_{adj} (%)						**6.7**	**32.1**	**30.2**	**33.2**
Bussard/Solitär (N = 81)									
QStb.lsa_bs-7A	Solitär	-	-	-	-	-5.1	-5.4	-6.0	**-5.5**
Total R^2_{adj} (%)						**6.8**	**8.9**	**7.8**	**14.1**

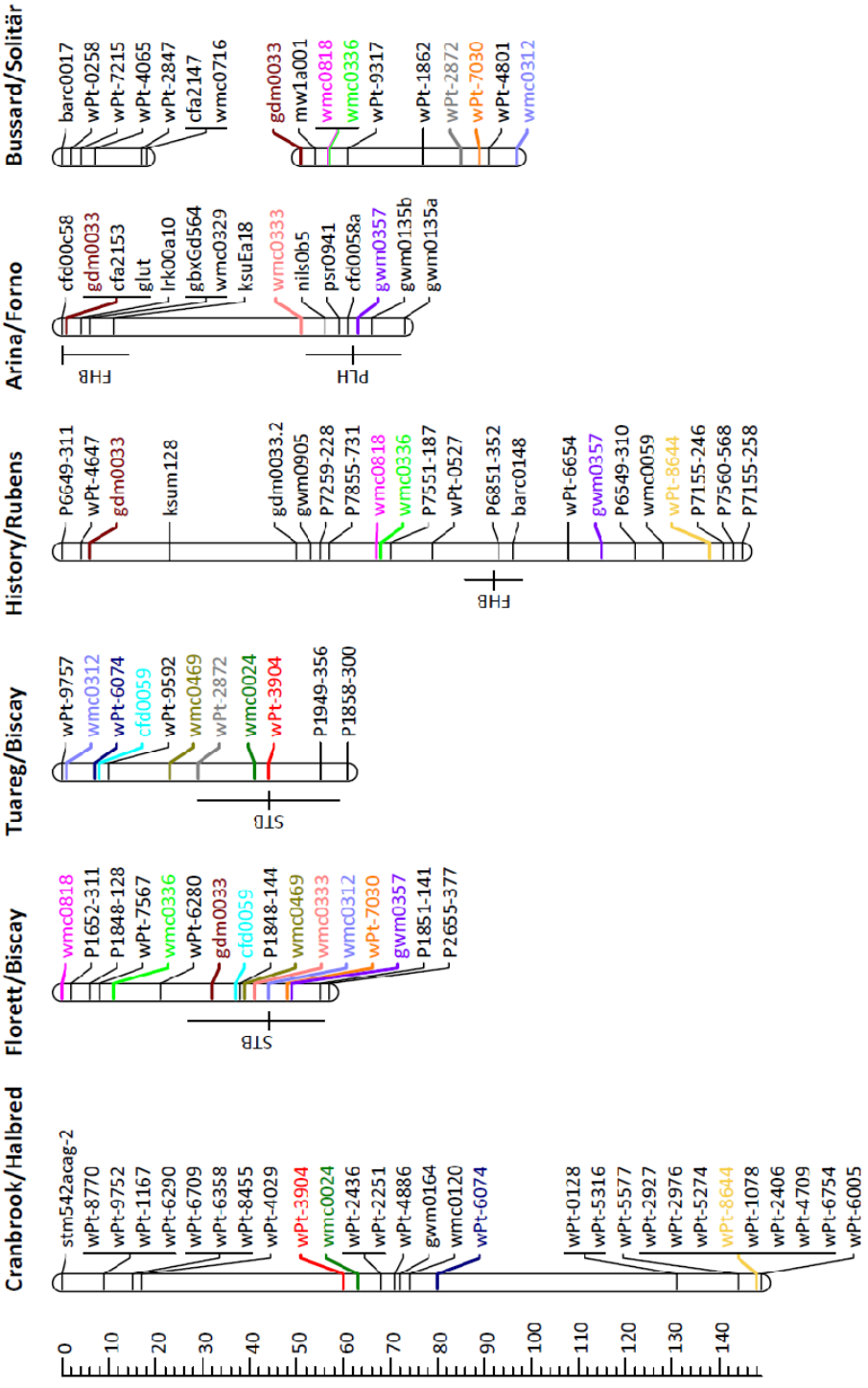
[1] Estimated additive effects (less % flag leaf infection) in final simultaneous fit of the resistance allele at the locations Freising (FRE), Hohenheim (HOH), Oberer Lindenhof (OLI), Wohlde (WOH), across two years (2008, 2009) and in the series

[2] Adjusted phenotypic variance explained by detected QTL in final simultanous fit in each environment and in the series

S 5: Genetic maps with chromosomal locations of QTL for resistance to Septoria tritici blotch (STB), Stagnospora glume blotch (SGB), and Fusarium head blight (FHB) as well as QTL for plant height (PLH), heading date (HED), and meta QTL (MQTL) for multiple-disease resistance in winter wheat populations Florett/Biscay, Tuareg/Biscay, History/Rubens, Arina/Forno, and Bussard/Solitär. Additionally, genetic map of Cranbrook/Halbred is presented as reference map (Akbari et al. 2006). Mapped markers are indicated on the right of each chromosome and their corresponding cumulative genetic distances (cM) are indicated as ruler at the left page margin. Common marker names across mapping populations within chromosomes are highlighted with the same color. Detected QTL in our study are designated with corresponding trait abbreviation left of chromosomes. Line lengths for QTL and length of hatched bars for MQTL represent 95 % confidence intervals and support intervals, respectively. Shorter lines and bars indicate more precise QTL and MQTL locations, respectively

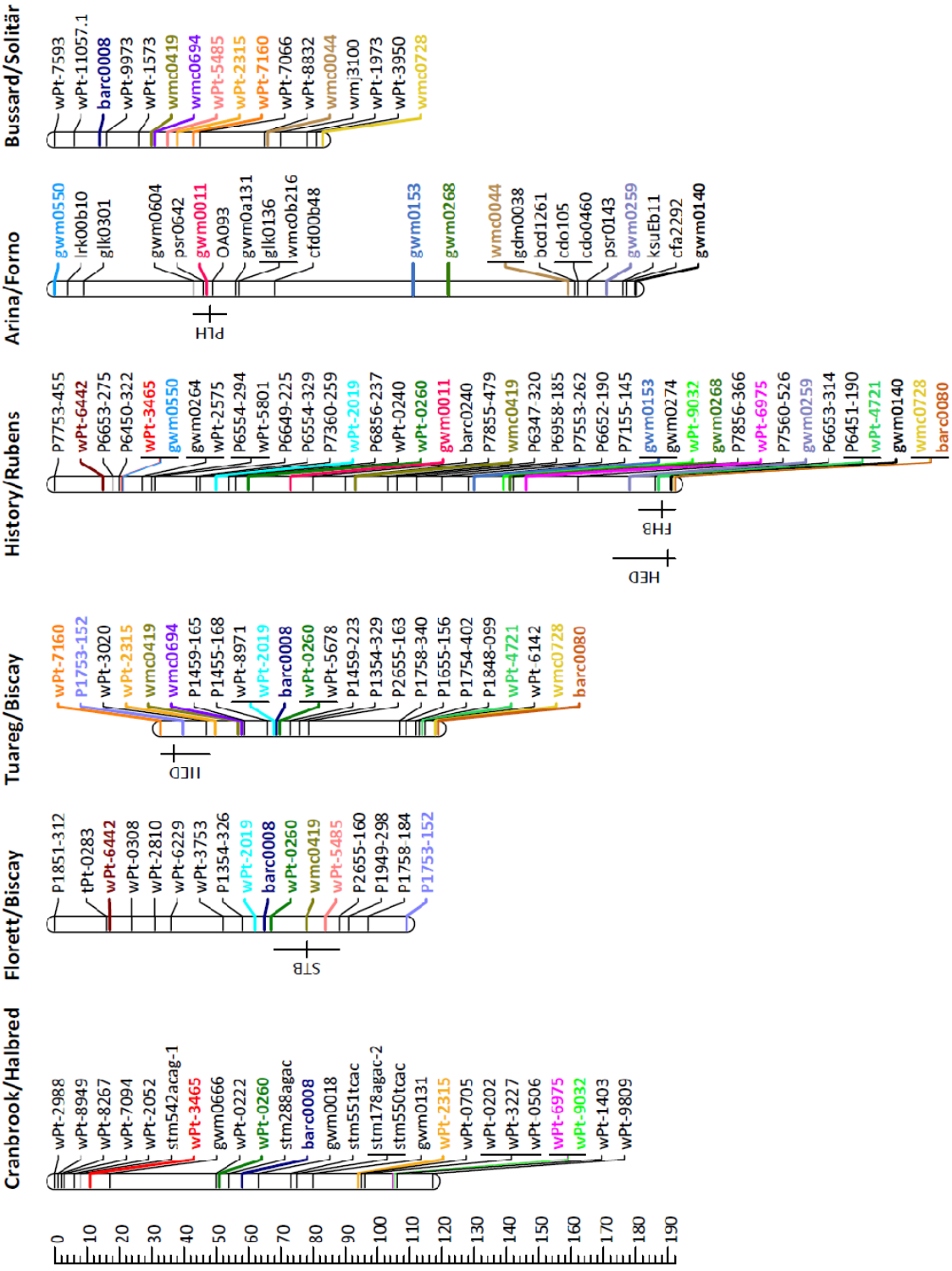
S 5: Continued

Chromosome 1A



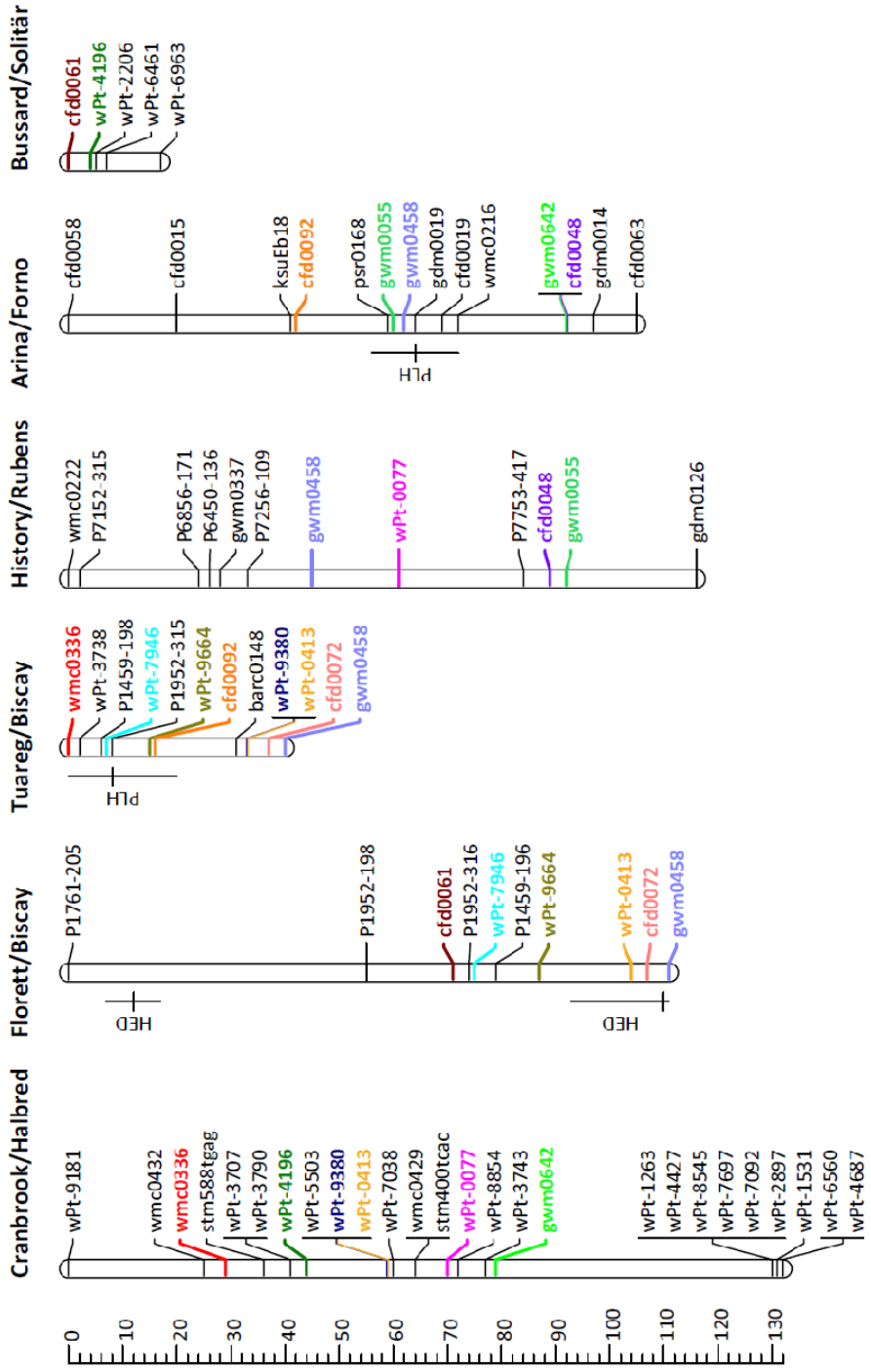
S 5: Continued

Chromosome 1B



S 5: Continued

Chromosome 1D



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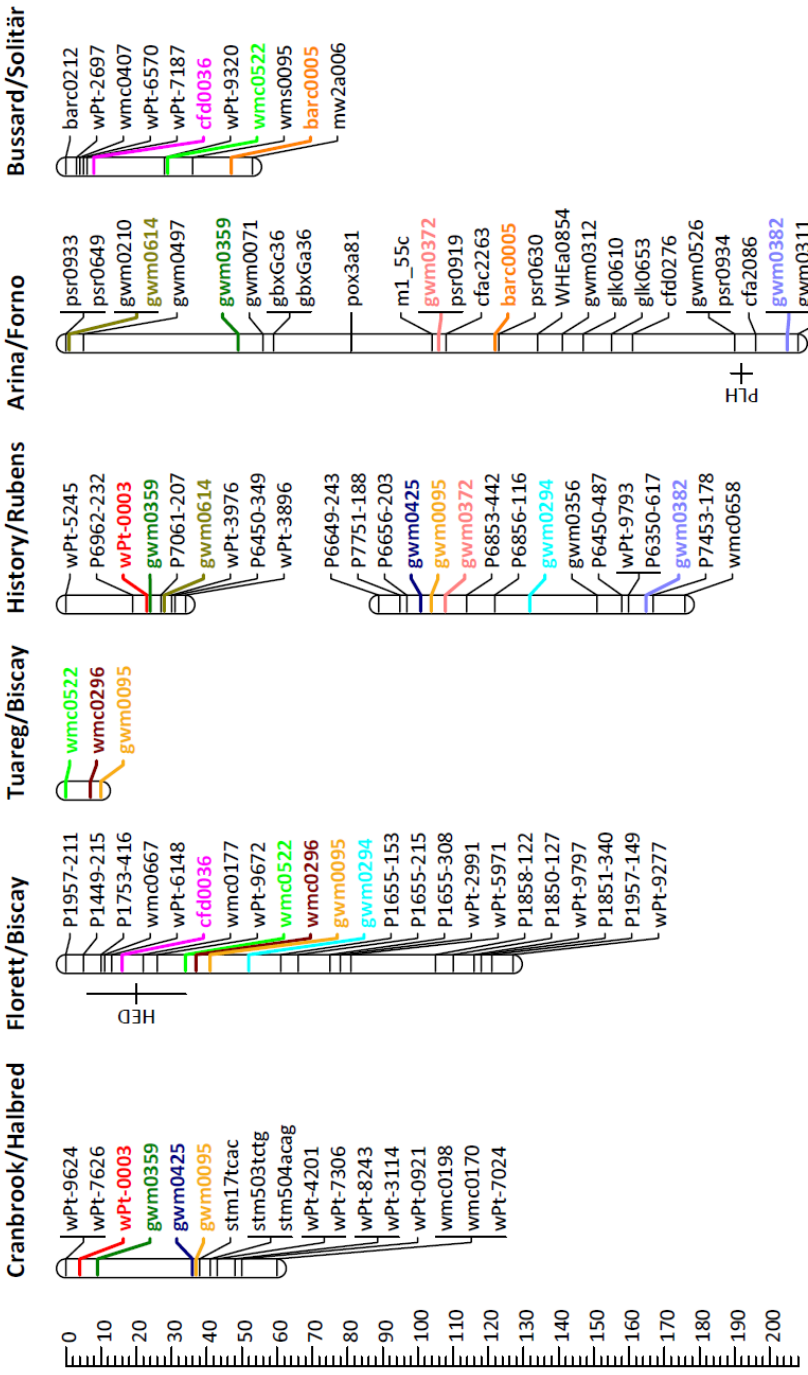

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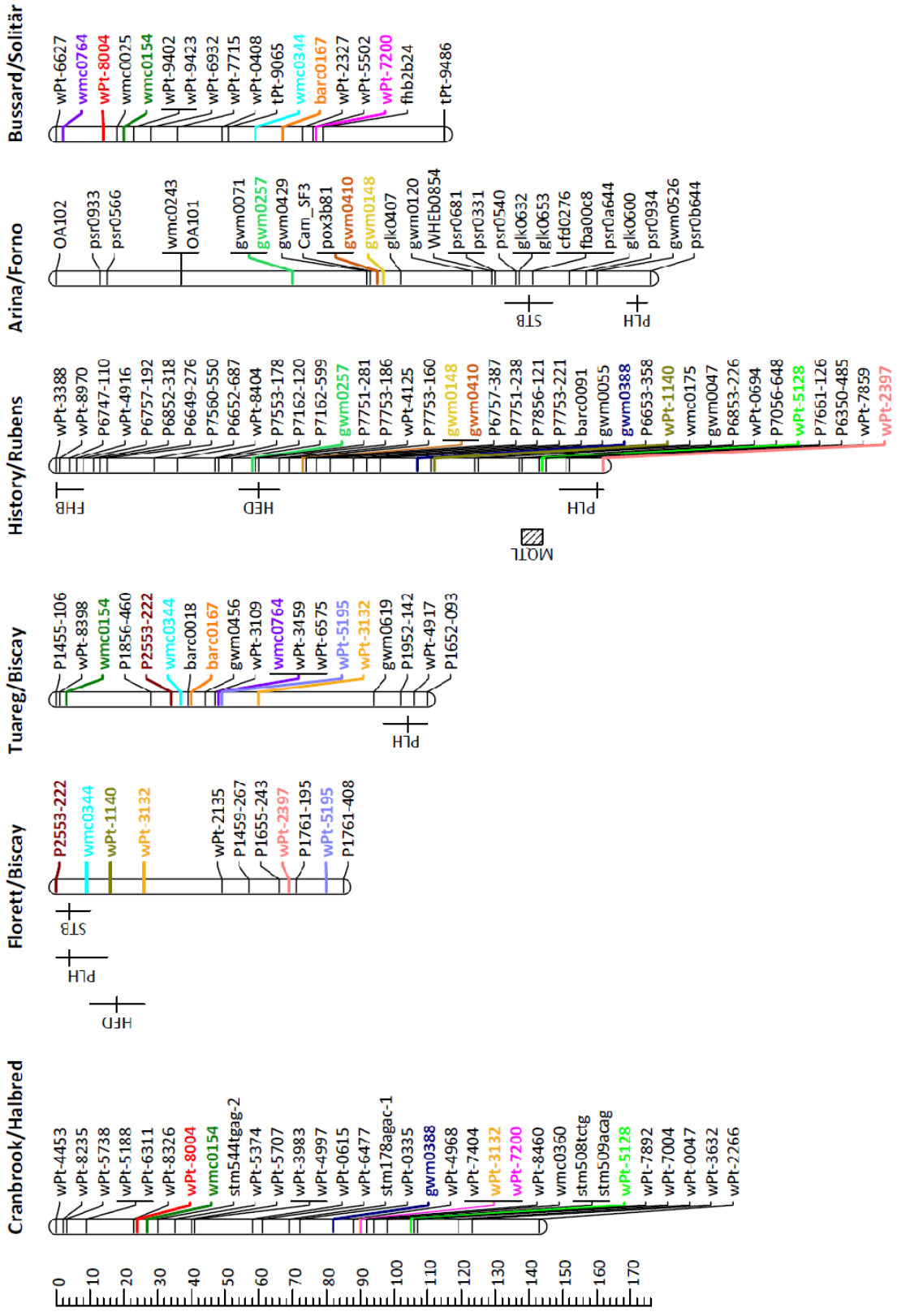

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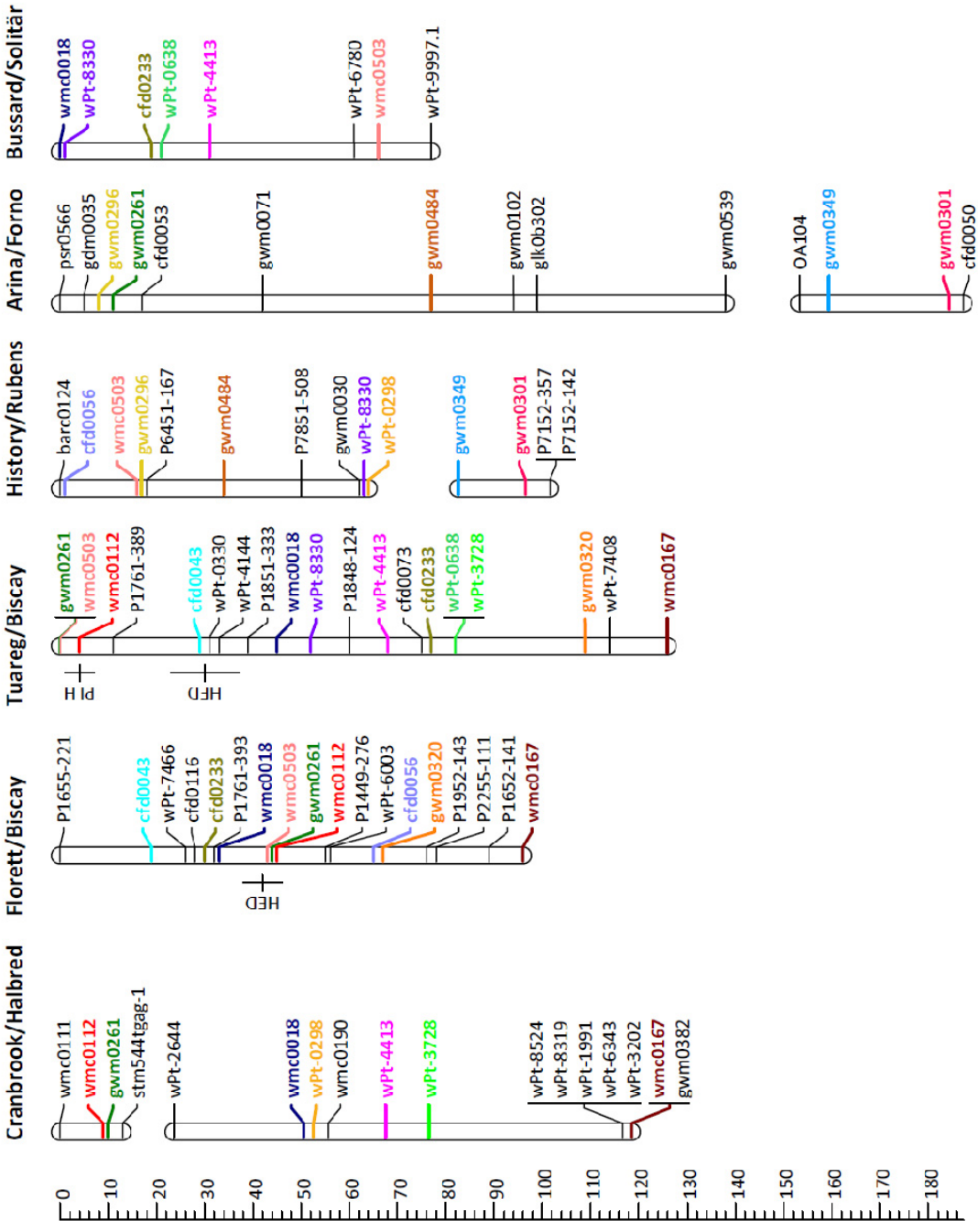
Chromosome 2D



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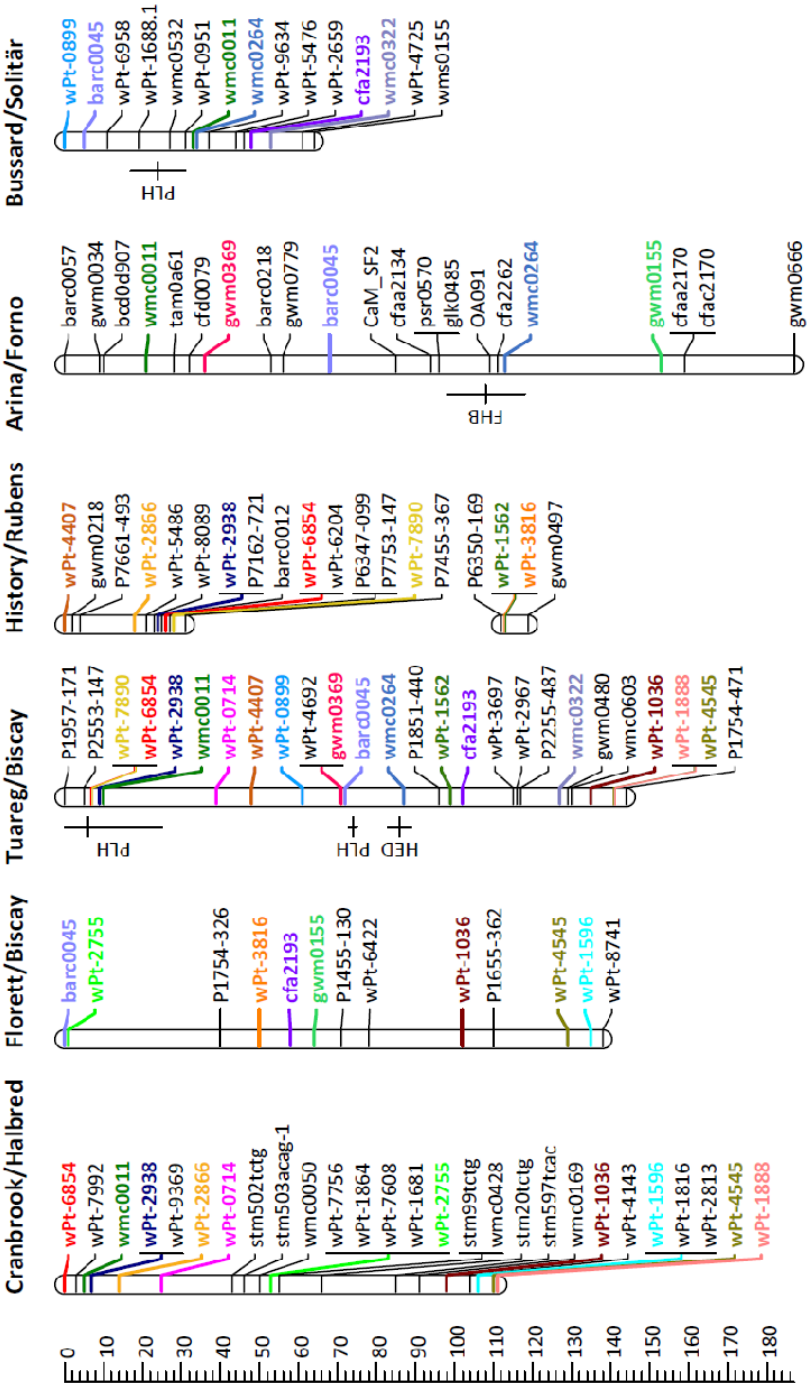
Chromosome 3A



S 5: Continued

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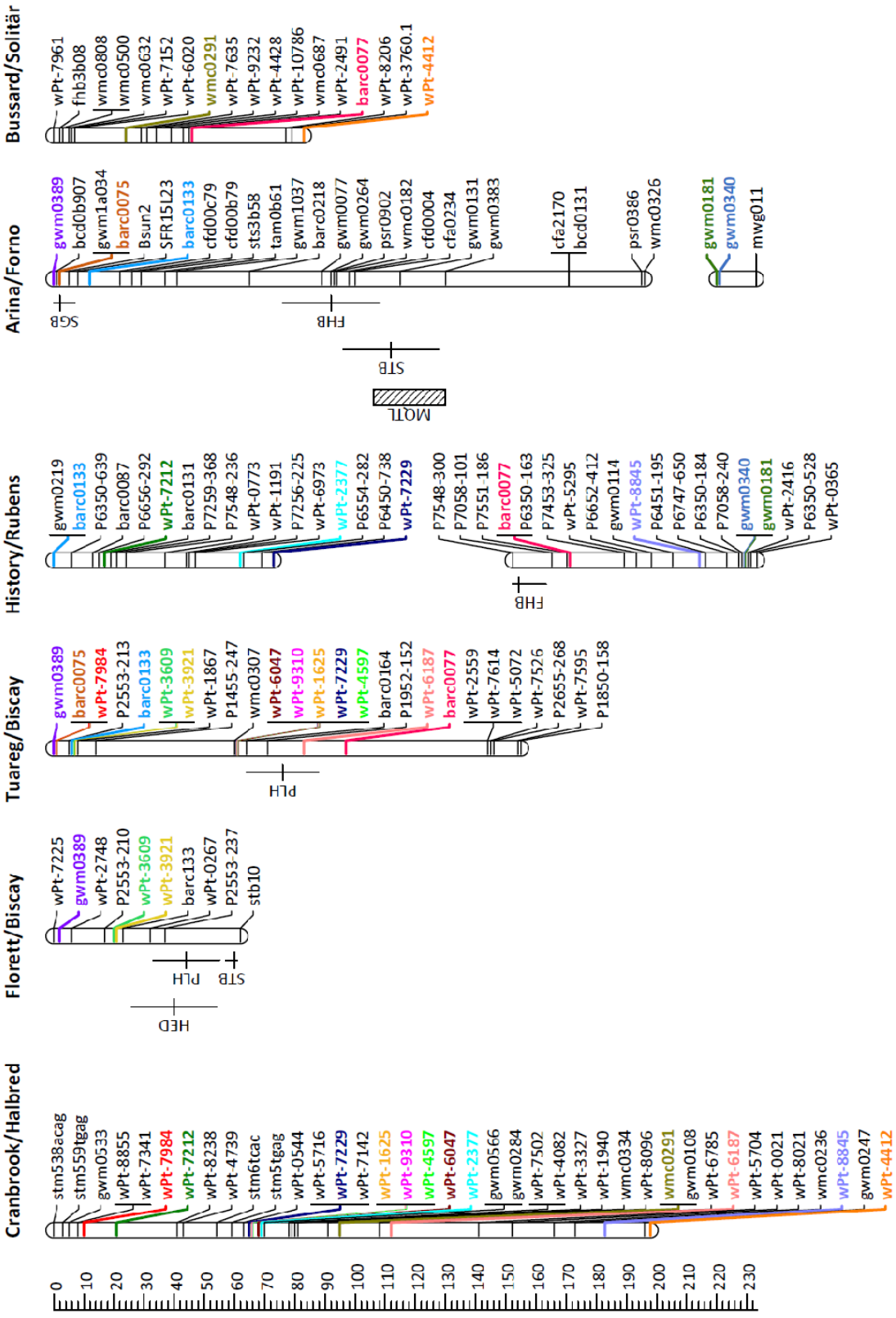

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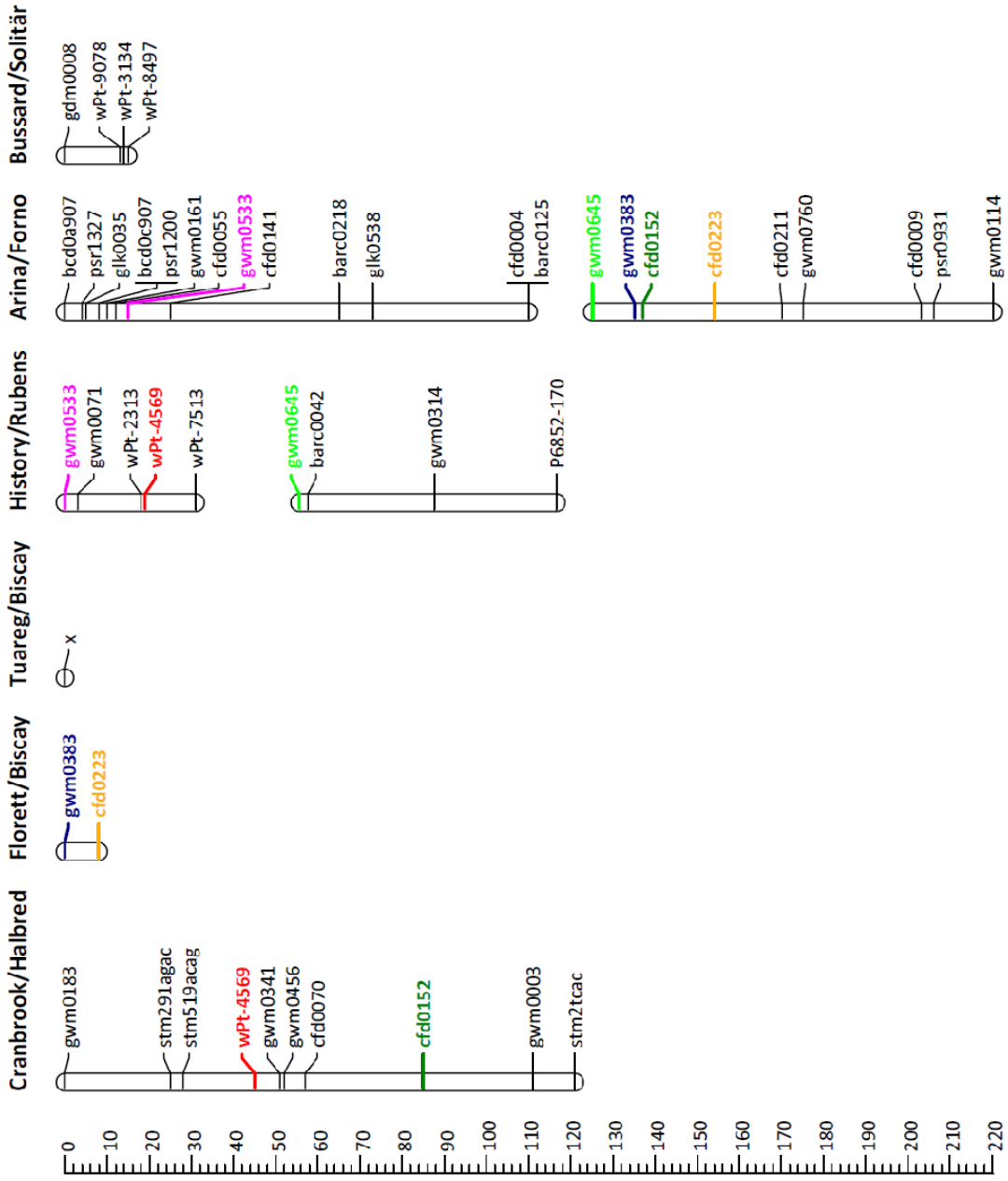
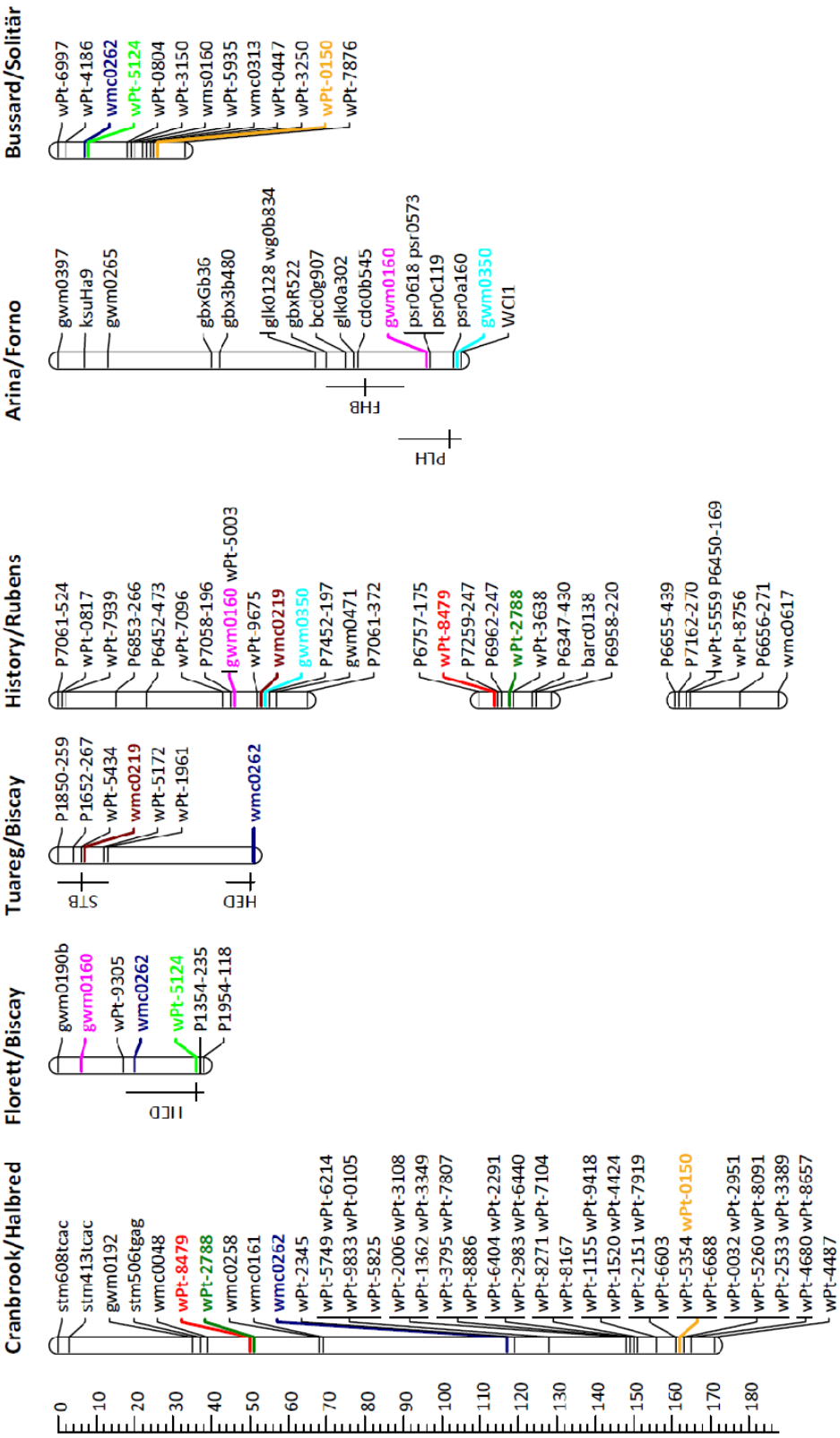


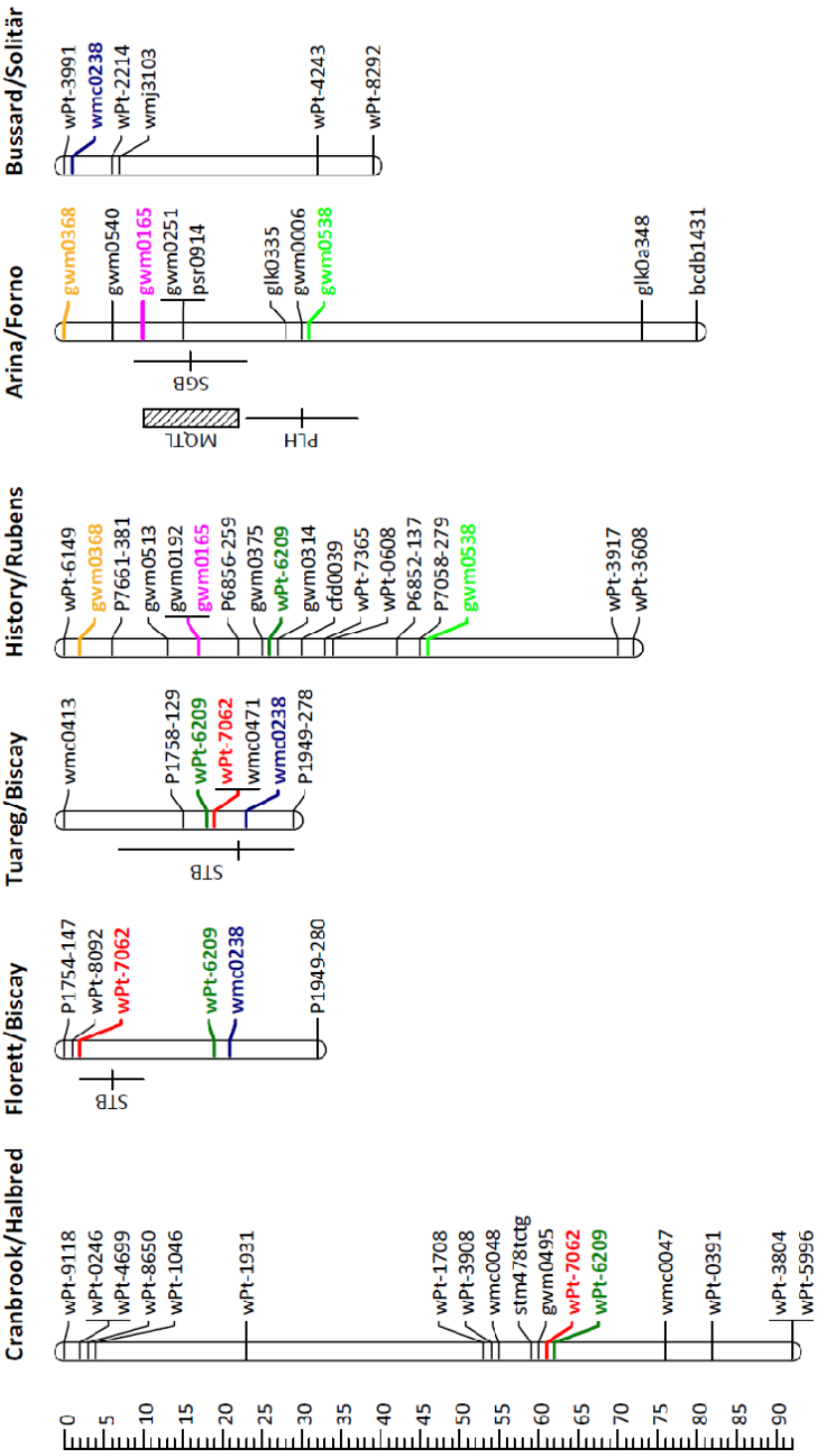
S 5: Continued

Chromosome 4A



S 5: Continued

Chromosome 4B



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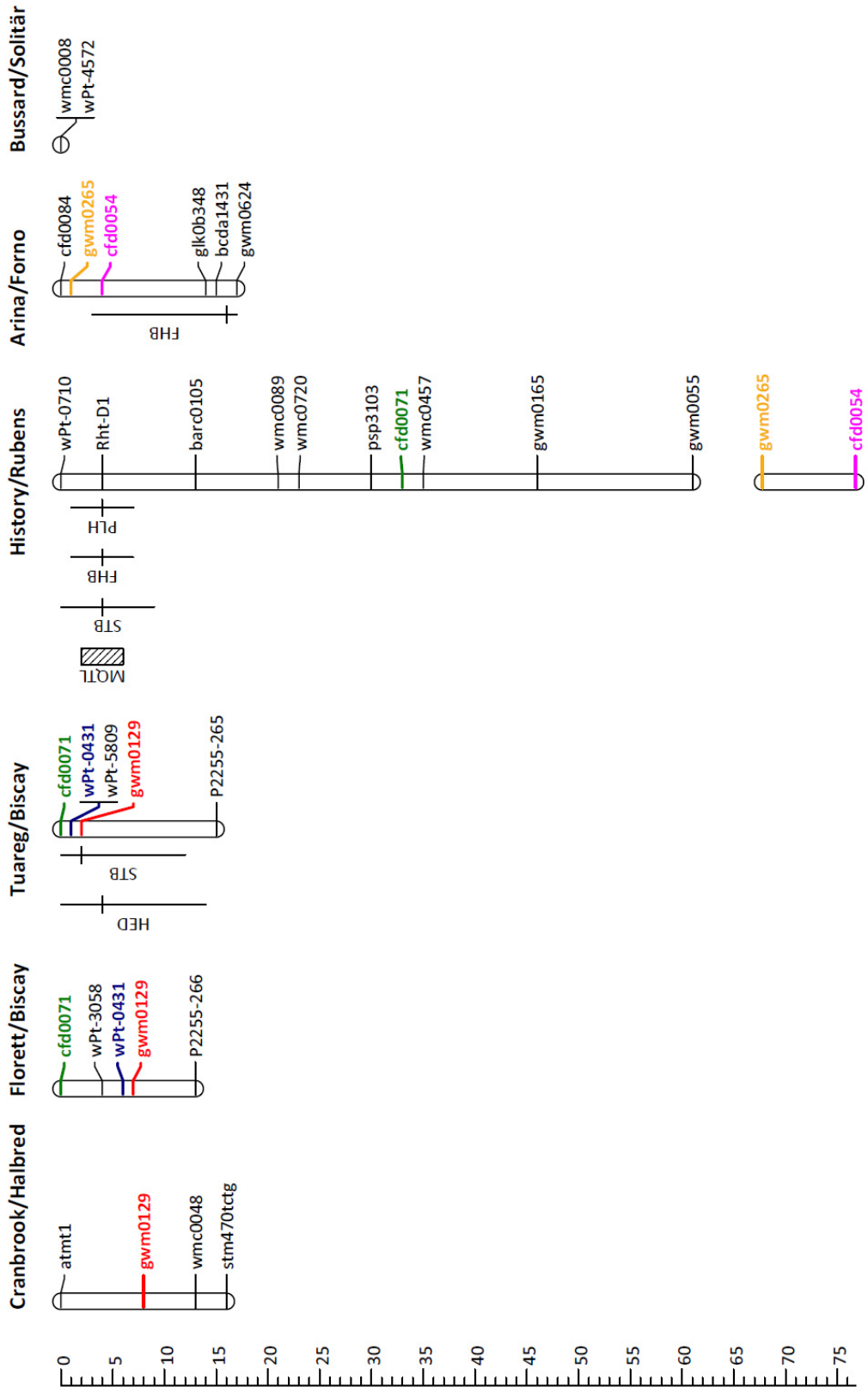
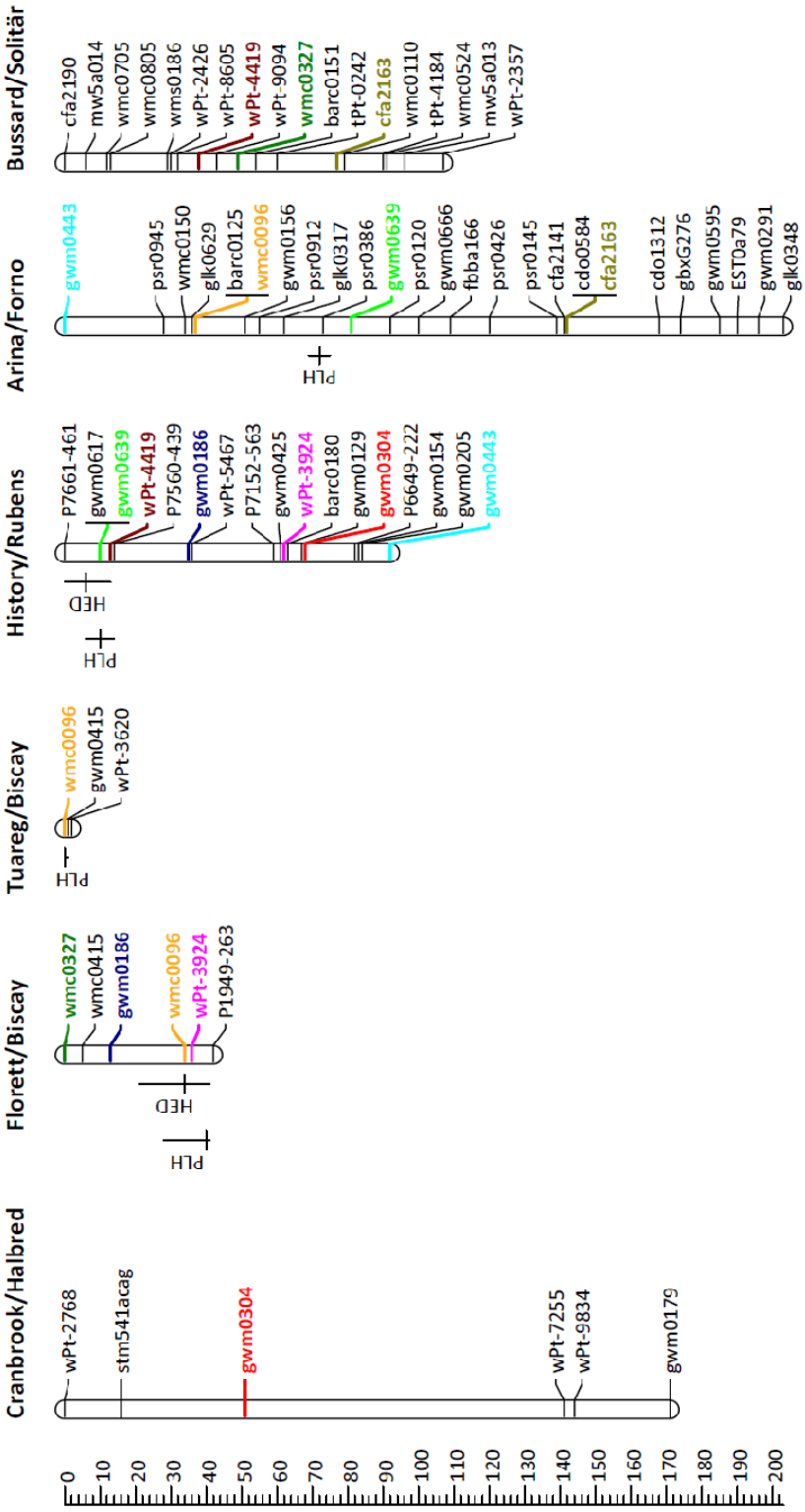


S 5: Continued

Chromosome 5A



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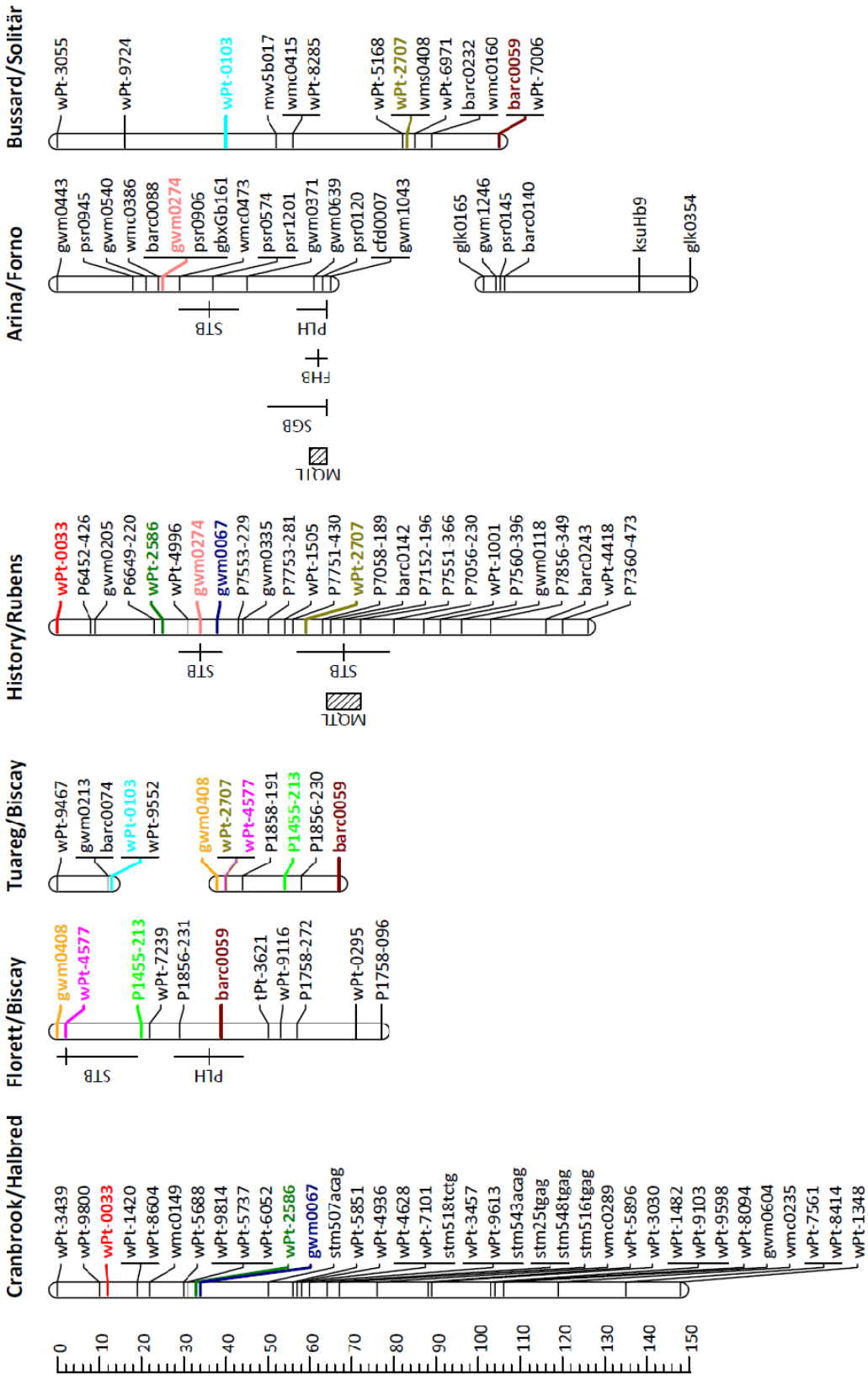

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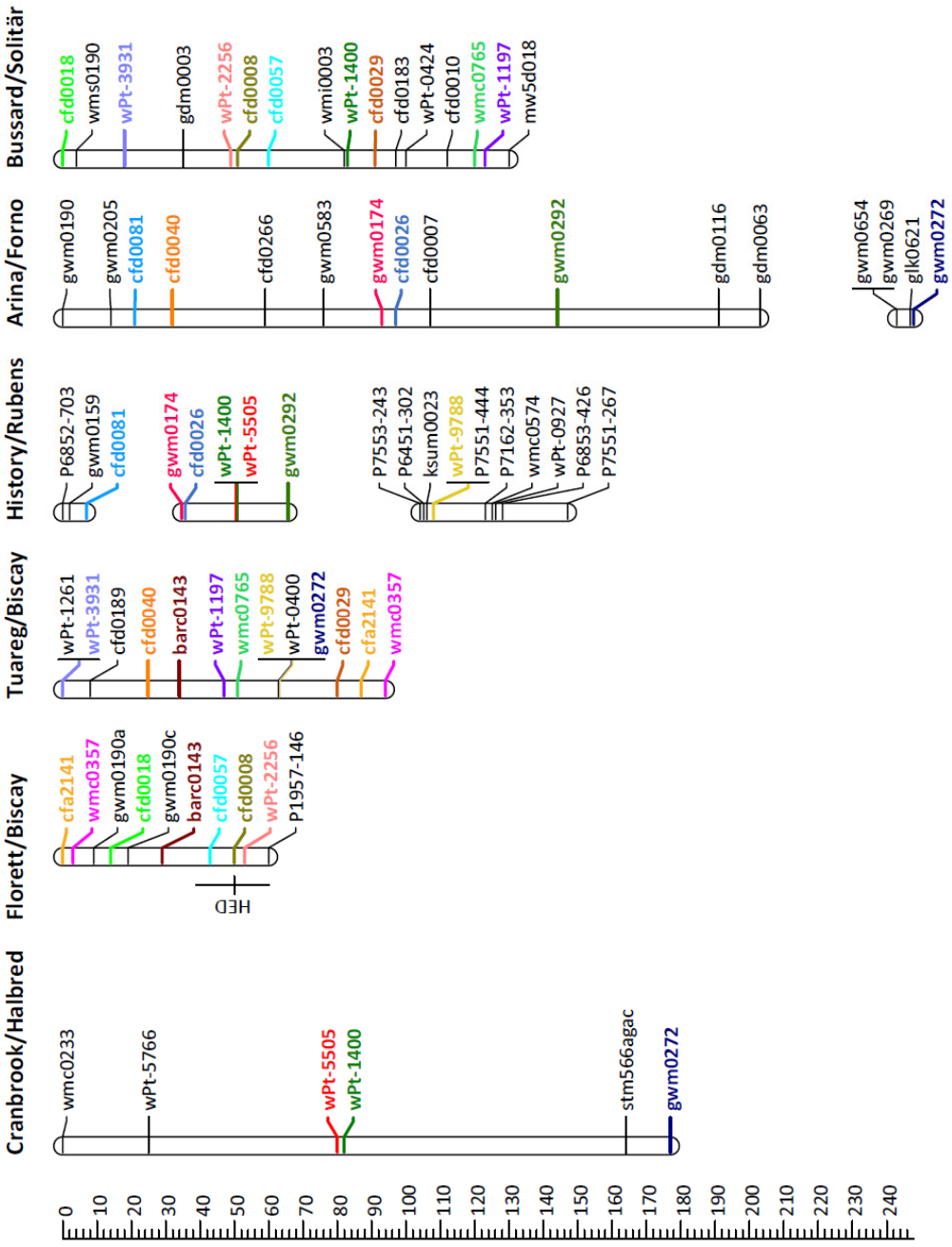
Chromosome 5D



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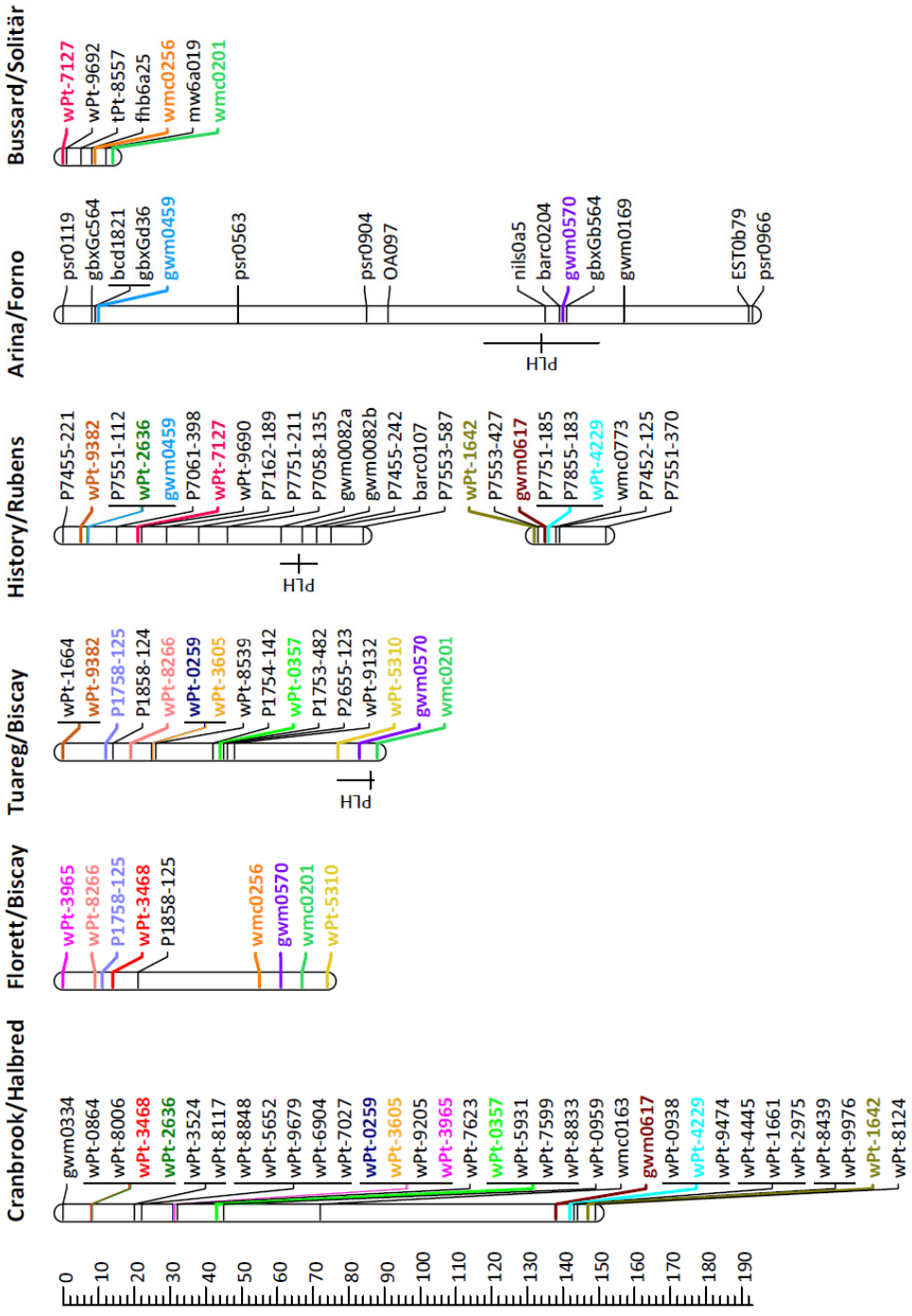
Chromosome 6A



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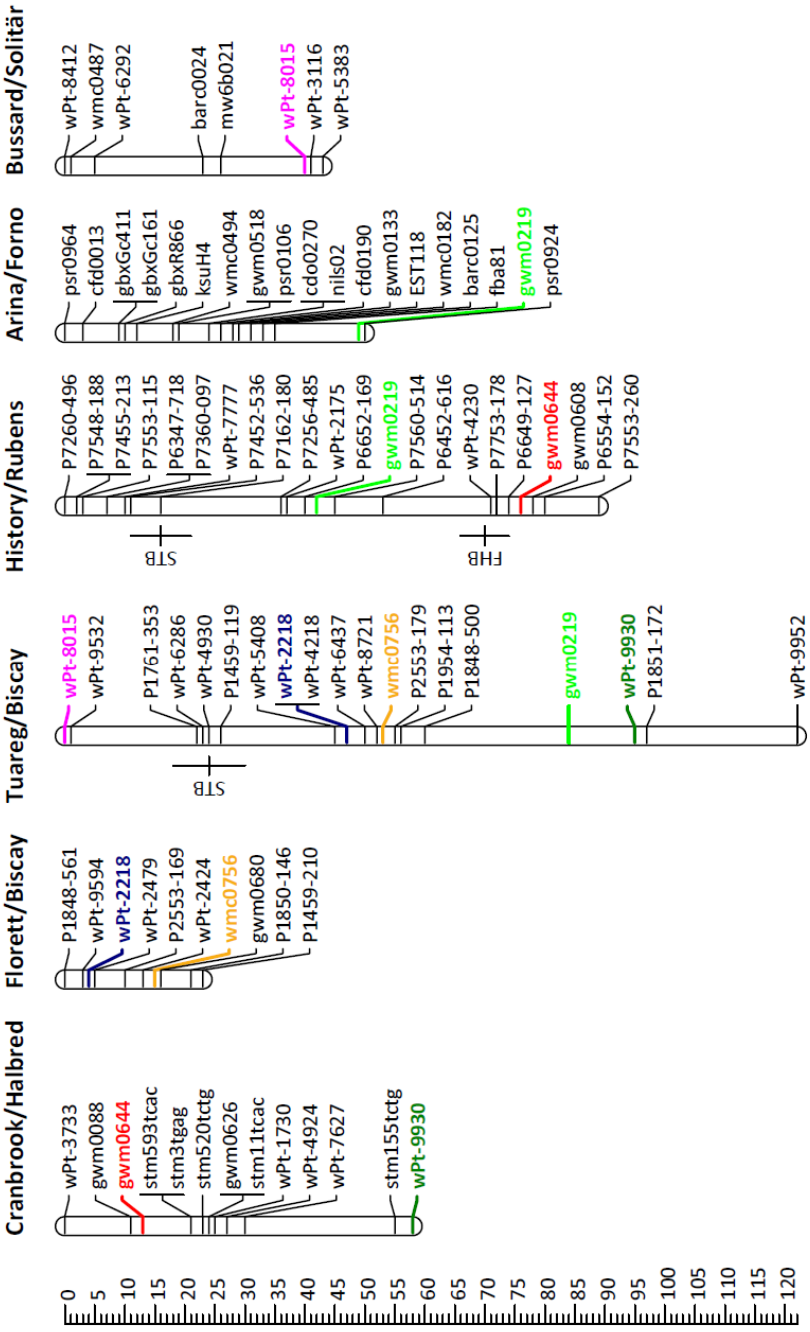
Chromosome 6B



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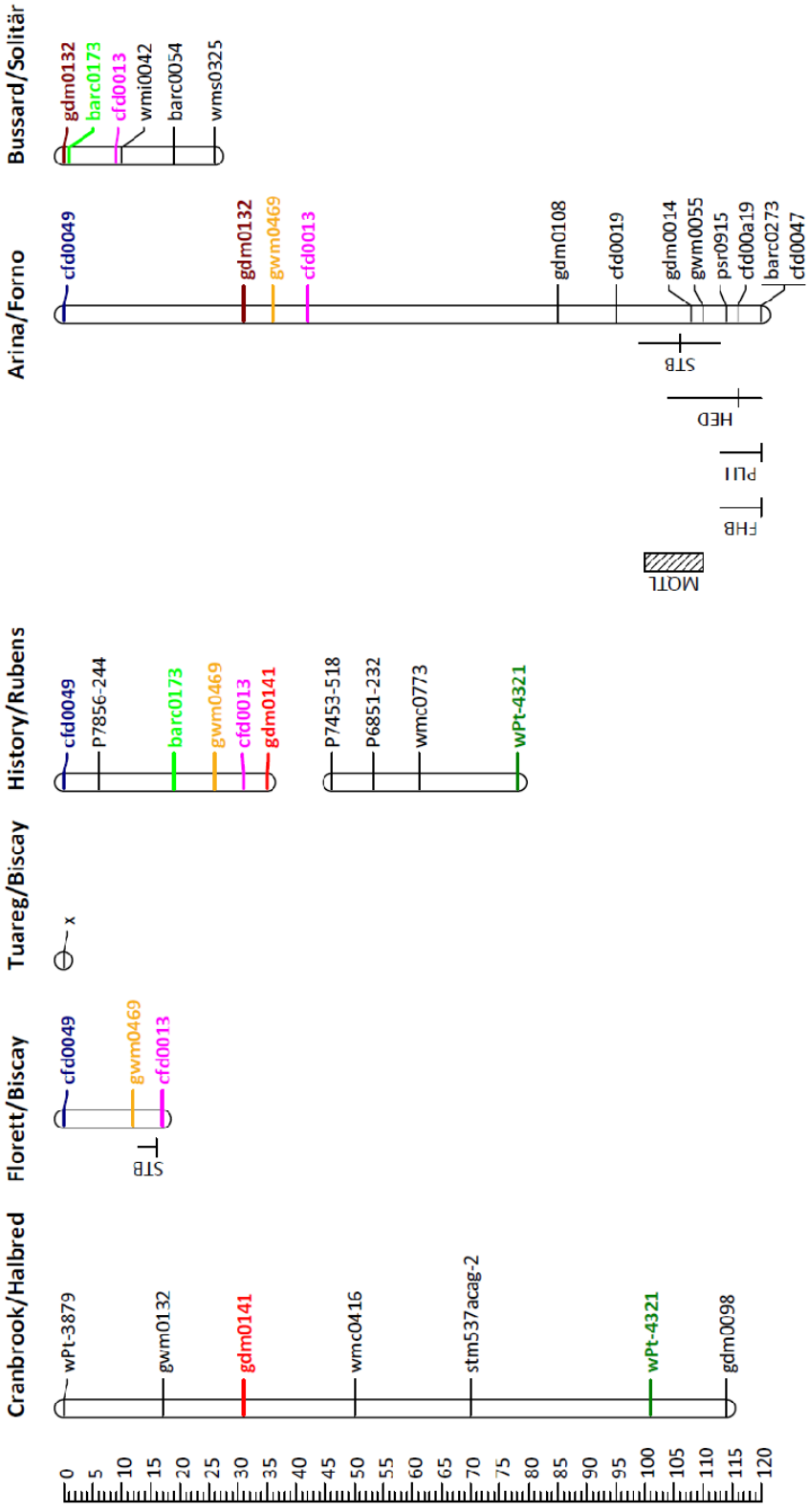
Chromosome 6D



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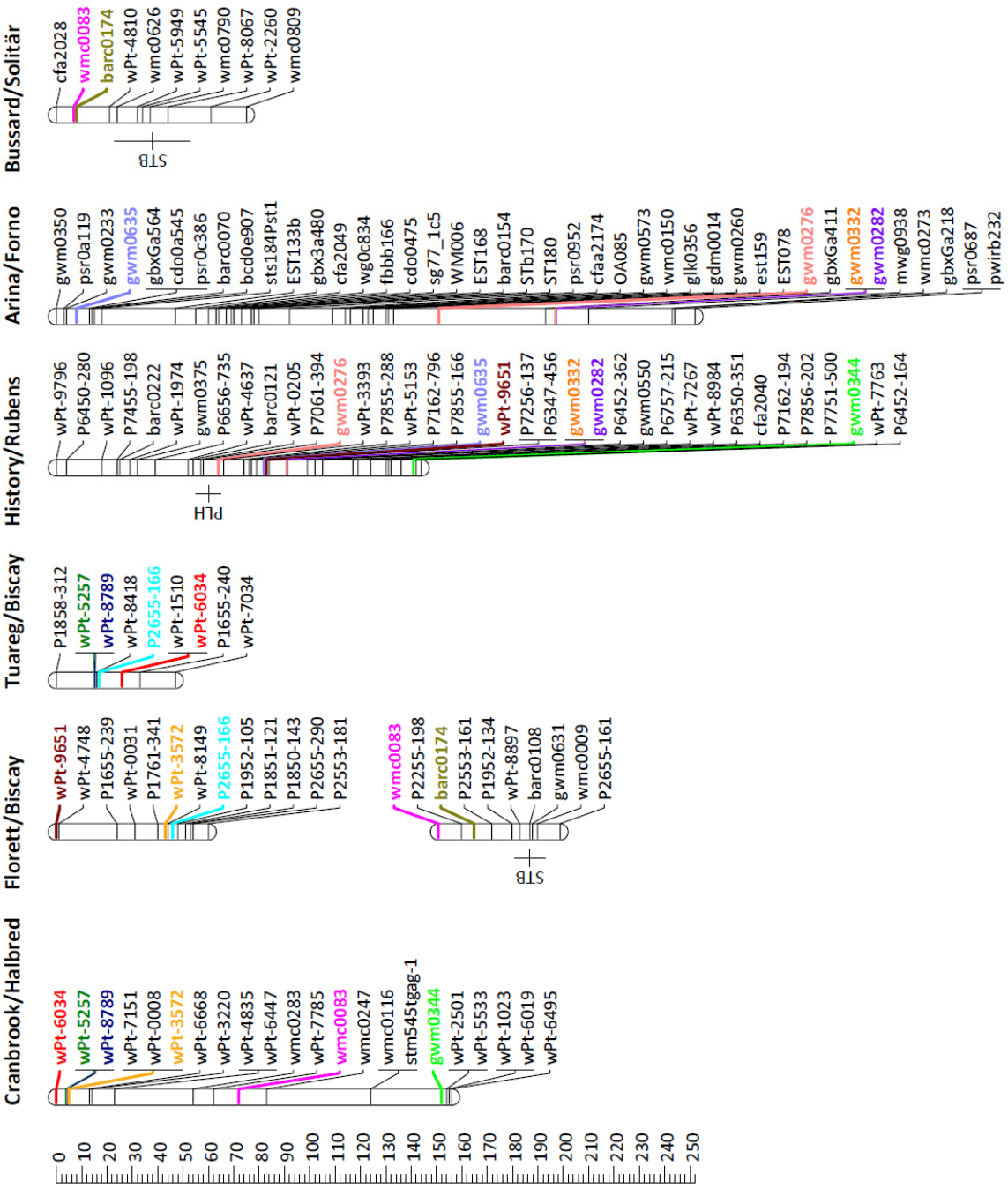
Chromosome 7A



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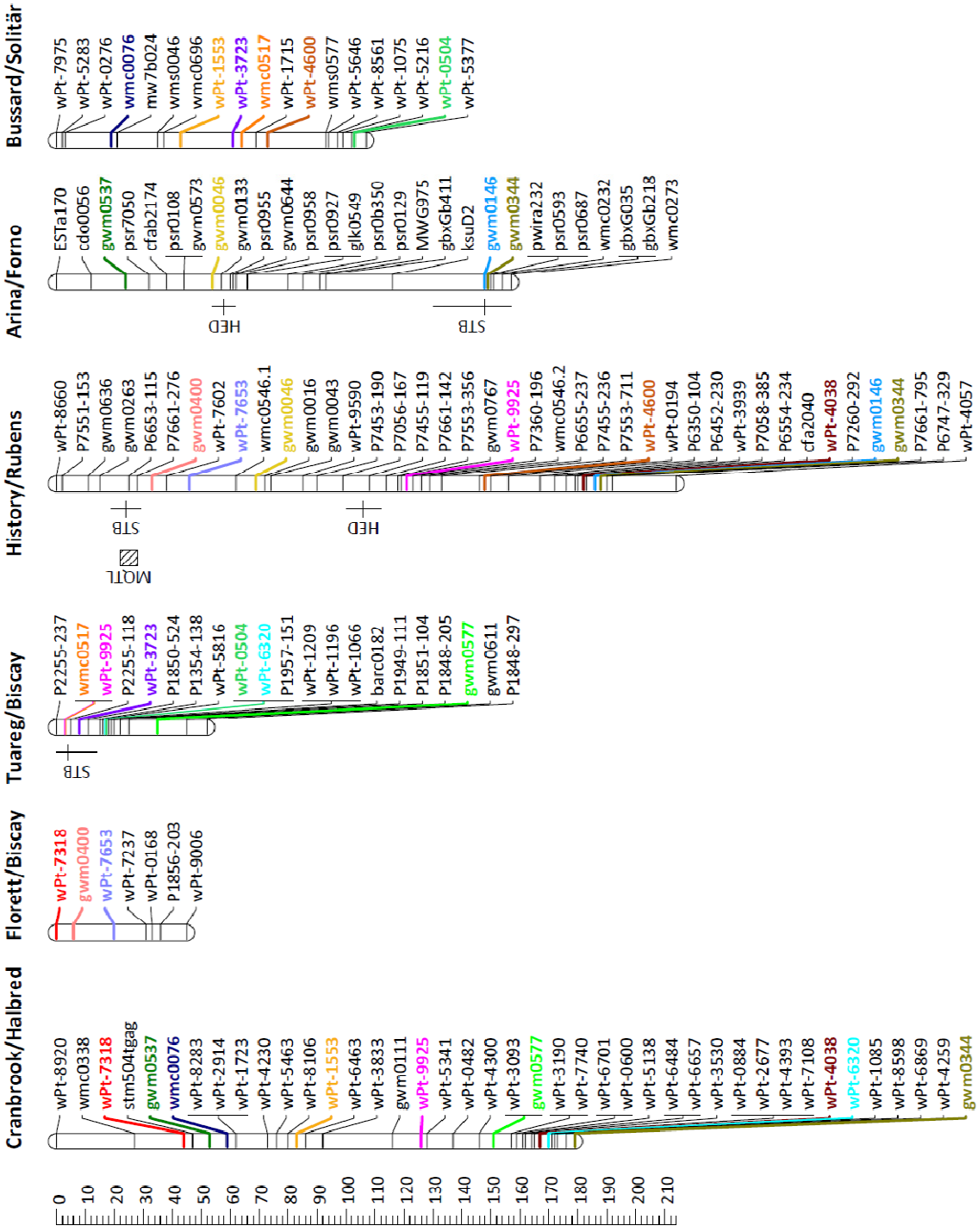
Chromosome 7B



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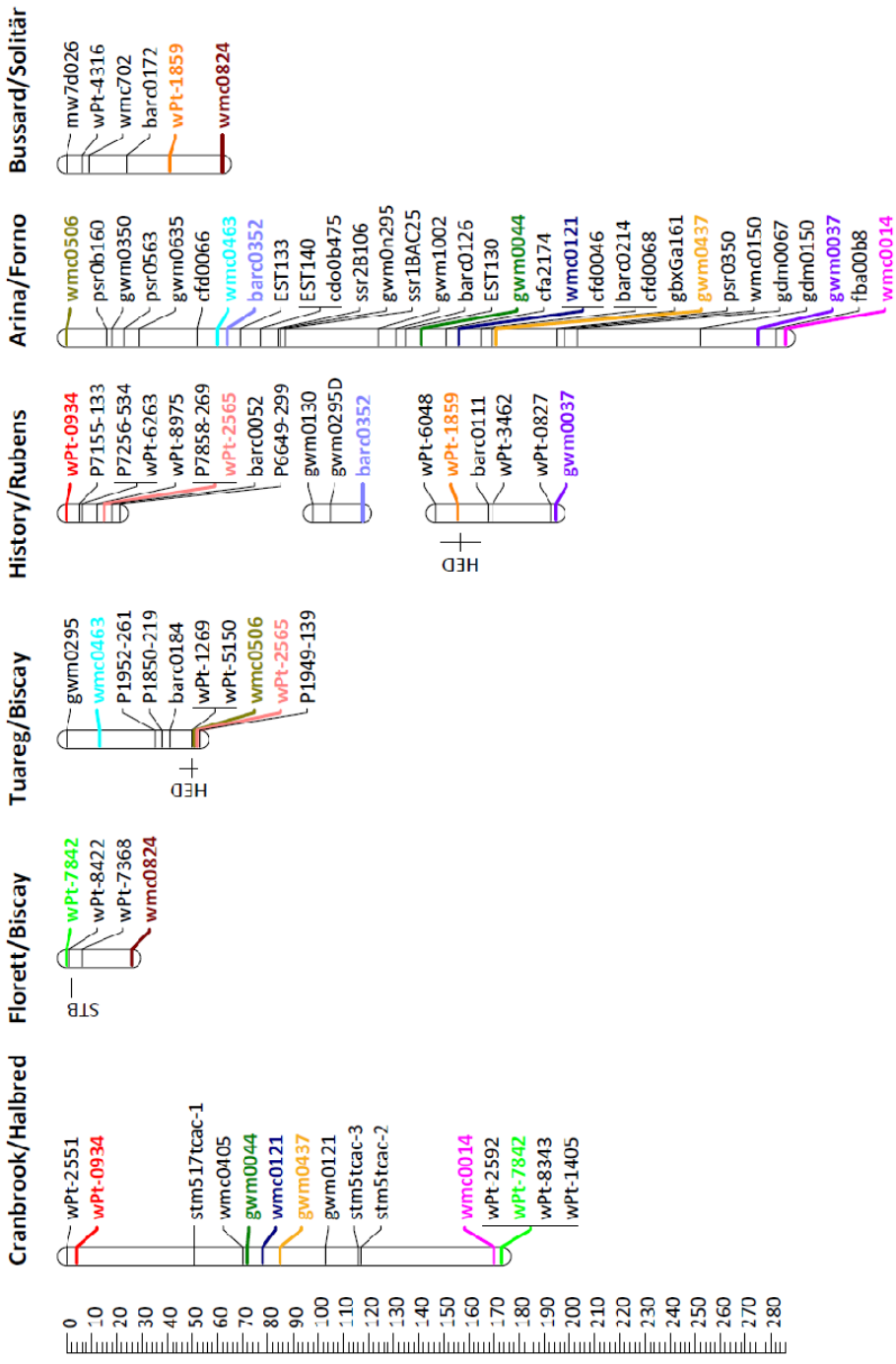
Chromosome 7D



Acknowledgements

I am very grateful to apl. Prof. Dr. T. Miedaner for his advice, suggestions, and continuous support during this thesis work. Thanks to Prof. Dr. R. T. Vögele for serving on my graduate committee.

Special thanks to Bärbel Lieberherr for her excellent assistance during field trials, her helping hand wherever needed, and her open and friendly mood. The help of Sigrid Erath, Marlene Warsow, Mark Raith, Manfred Buck-Finkele, Sabine Pöschel, the whole group AG Roggen, the field stations Oberer Lindenhof, especially Helmut Bimek, and Heidfeldhof, especially Herbert Stelz, is acknowledged. The atmosphere was always friendly and helpful during the field season including inoculation, field evaluations, and even installing an irrigation system.

Many thanks to Prof. Dr. H. F. Utz for fruitful discussions on statistics and QTL mapping and for discussing “other things”.

I would like to thank all present and former members of the State Plant Breeding Institute, Prof. Dr. C. C. Schön, PD Dr. J. C. Reif, Dr. G. Oettler, Dr. C. I. Kling, Dr. E. Bauer, Dr. V. Hahn, Dr. H. P. Maurer, Dr. T. Würschum, G. Hartmann, D. Wahnelt, U. Schrader, L. Handt, and B. Kurka for their help in organizational matters and for the discussions during coffee break.

I really enjoyed the work with Ayla Schilly and Georg Forster during their time preparing Bachelor and Master thesis in this project. Especially their help in field trials beyond their thesis is greatly appreciated.

I thank all my Ph.D. student colleagues, Silke, Firas, Martin, Maren, Christiane, Delphine, Thilo, Vanessa, Christin, Benjamin, Friedrich, Hans-Henning, Matthias, Sebastian, Sandra, Sankalp, Bettina, Greta, Jana, Marlen, Katharina, Christoph, Alexander, and all unmentioned members of the Hohenheimer plant-breeding group for creating a pleasant work environment.

I want to thank all the partners collaborated in the ERA-NET Plant Genomics project called “CEREHEALTH” working with wheat and *Septoria tritici*, namely the KWS LOCHOW GMBH and in particular Dr. Erhard Ebmeyer, Dr. Viktor Korzun, Meike Scholz, Annina Schulz, and the whole team at Wohlde, as well as the Bavarian State Research Center for Agriculture (LfL), Institute for crop science and plant breeding especially Dr. Lorenz Hartl, Dr. Jennifer Häberle, Dr. Josef Holzapfel, Lydia Giehl, Thomas Wirth, and the whole team at Freising.

I dedicate special thanks to INA and my FAMILY for their great support during my thesis.

Curriculum vitae

Personal Details

Name:	Peter Risser
Birth:	2 April 1982 in Kirchheimbolanden
Marital Status:	Married

School Education

1988 to 1992	Grundschule, Stetten
1992 to 2001	Nordpfalzgymnasium, Kirchheimbolanden, Abitur, June 2001

Civilian Service

08/01 to 05/02	Farmhand at 16 different farms

University Education

10/02 to 11/05	Bachelor of Science in Agriculture (Major in Crop Sciences), Universität Hohenheim, Stuttgart
10/05 to 10/07	Master of Science in Plant Breeding, Universität Hohenheim, Stuttgart
06/07 to 04/10	Ph.D. in Agricultural Sciences, Universität Hohenheim, Stuttgart

Practical Experience

07/02 to 08/02	Ervema agrar GmbH, Wiebelsdorf, Germany
08/04 to 10/04	Estancia La Josefina S.A., Province Buenos Aires, Argentina
08/05 to 09/05	KWS LOCHOW GMBH, Bergen-Wohlde, Germany
08/06 to 08/06	KWS Saat AG, Einbeck, Germany

Professional Employment

06/07 to 04/10	Research assistant at the State Plant Breeding Institute, Universität Hohenheim, Stuttgart
Since 05/10	Südzucker AG, Mannheim/Ochsenfurt

Erklärung

Hiermit erkläre ich an Eides statt, dass die vorliegende Arbeit von mir selbst verfasst wurde und lediglich unter Zuhilfenahme der angegebenen Quellen und Hilfsmittel angefertigt wurde. Wörtlich oder inhaltlich übernommene Stellen wurden als solche gekennzeichnet.

Die vorliegende Arbeit wurde in gleicher oder ähnlicher Form noch keiner anderen Institution oder Prüfungsbehörde vorgelegt.

Insbesondere erkläre ich, dass ich nicht früher oder gleichzeitig einen Antrag auf Eröffnung eines Promotionsverfahrens unter Vorlage der hier eingereichten Dissertation gestellt habe.

Stuttgart, im April 2010

_____

Peter Risser