A genome-wide association study of resistance to the yellow rust pathogen (*Puccinia striiformis* f. sp. *tritici*) in elite UK wheat germplasm

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ABSTRACT

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Identification of marker-trait associations (MTA) in germplasm relevant to breeding program via association mapping (AM) can be an effective way to identify loci useful for selection. This approach does not require the generation of specific mapping populations and takes advantage of historical phenotypic data. In the present study, an association panel of 327 bread wheat varieties have been assembled and genotyped with 1806 DArT markers. Genetic structure analysis revealed a low stratification of the panel based on geographical origin (UK versus mixed European varieties) and a close relatedness between lines, which is confirmed by pedigree information. Historical evaluations against the yellow rust pathogen (*Puccinia striiformis* f.sp. *tritici* (Pst)) carried in the United Kingdom between 1990 and 2009, as well as *de novo* evaluations against recent Pst races have been collected and analysed for MTAs.

Association scans considering historical data focused on specific Pst pathotypes and *de novo* seedling tests identified markers linked to known race-specific *Yr* genes *Yr6, Yr7, Yr9, Yr17* and *Yr32.*

When evaluated against current Pst races in the field, 35% of the lines from the panel presented repeatedly a high level of resistance (Area Under the Disease Progress Curve relative<0.2) which is due to the presence of seedling resistances as well as adult plant resistances within the lines. AM with *de novo* phenotypes revealed 23 MTA groups pointing to potential resistance loci, 14 of them were also identified with historical data and six seemed to point to adult plant resistance loci on chromosomes 2A, 2B, 3A, 6A, 6B and 7A.

These results confirm the value of AM using historical data for QTL discovery and suggest the availability of diverse sources of yellow rust resistances within wheat elite UK germplasm.

STATEMENT OF ORIGINALITY

Unless otherwise mentioned in the text, the work presented in this thesis is that of the author.

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LIST OF ACRONYMS

AFLP: amplified fragment length polymorphism
AM: association mapping
APR: adult plant resistance
APT: adult plant test
AUDPC: area under the disease progress curve
AUDPCr: relative AUDPC
Avr: avirulence
BAC: bacterial artificial chromosome
BBSRC: Biotechnology and Biological Sciences Research Council
BSPB: British Society of Plant Breeders
CAPs: Cleaved amplified polymorphic sequence
CIMMYT: International Maize and Wheat Improvement Centre
DArT: diversity array technology
DEFRA: Department for Environment, Food and Rural Affairs
DEL: deletion
DPI: days post inoculation
DT: ditelosomic
ECPGR: European Cooperative Programme for Plant Genetic Resources
EDTA: ethylenediaminetetraacetic acid
EST: expressed sequence tags
FERA: Food and Environment Research Agency
FHR: field host response
GC: growth chamber
GLM: general linear model
GS: Growth stage
GWA: genome wide association
HGCA: Home Grown Cereals Authority
HR: hypersensitive response
HTAP: High-temperature adult plant resistance
IGS: intergenic spacer
INDEL: insertion and deletion
IWGSC: international wheat genome sequencing consortium
IT: infection type
JIC: John Innes Centre
LD: linkage desequilibrium
MAS: marker assisted selection
MLM: mixed linear model
MTA: marker-trait association
NGS: next generation sequencing
NIAB: National Institute of Agricultural Botany
NL: National list trial
NRS: non-race specific
NT: nullisomic-tetrasomic
PBI: Plant Breeding Institute
PBIC: Plant Breeding International Cambridge
PCR: polymerase chain reaction
PCA: principal component analysis
PR : pathogenesis related
Pst: *Puccinia striiformis* Westend f.sp. *tritici*
QTL: quantitative trait loci

RAPD: random amplification of polymorphic DNA
RFLP: restriction fragment length polymorphism
RGA: resistance gene analog
RGAP: resistance gene analog polymorphism
R gene: resistance gene
R protein: resistance protein
RL: recommended list trial
RS: race specific
SASA: Science and Advice for Scottish Agriculture
SCAR: sequence characterized amplified region
SDR: seedling plant resistance
SDT: Seedling test
SFP: single features polymorphism
SRAP: sequence-related amplified polymorphism
SNP: single nucleotide polymorphism
SSR: single sequence repeat
STS: sequence-tagged site
TE: transposable element
UK: United Kingdom
UKCPVS: United Kingdom cereal pathogen virulence survey
WYR: wheat yellow rust

CHAPTER I. GENERAL INTRODUCTION

1 HEXAPLOID WHEAT

1.1 Production and use

Wheat (*Triticum* spp.) is a major cereal crop cultivated worldwide and contributes substantially to human diet and food security. Wheat was ranked third in 2010 for grain production with 651 million tons produced after maize (844 million tons) and rice (672 million tons) (http://faostat.fao.org). Since the end of the Second World War, wheat supply and demand have increased steadily to reach record levels in recent years (Figure I-1). For 2012, the International Grains Council forecasted production of 695 million tons and consumption of 684 million tonnes, the highest levels ever registered (http://www.igc.org.uk).

A tremendous improvement in productivity has been achieved over the past decades, mainly through agricultural development and adoption of green-revolution technologies worldwide (Evenson and Gollin, 2003). At the same time, greater demand is a direct result of the growing global population and changing dietary habits. With a projected world population of 8.27 billon for 2030, the demand for wheat is expected to average 851 million tons (Bruisman, 2003), representing an increase of 24% compared to the current level.

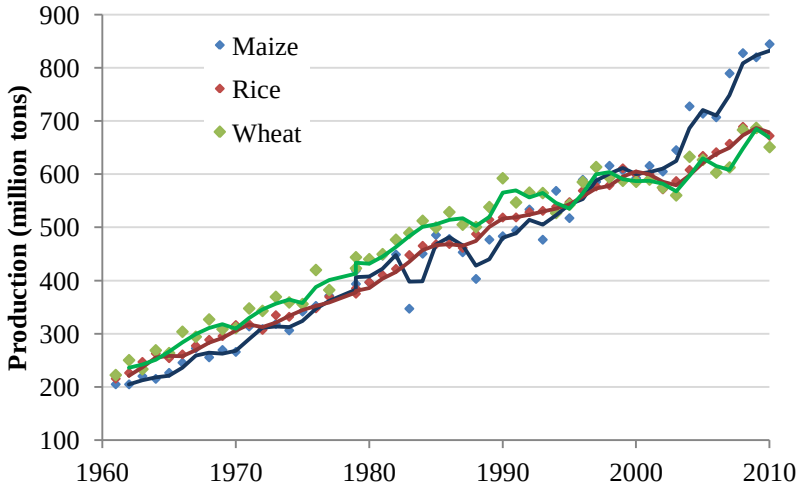

Figure I-1: Trends in Maize, Rice and Wheat global production over the past 50 years.

(Data from FAOSTAT http://faostat.fao.org)

In the UK, common wheat (*Triticum aestivum* L.) is the most important arable crop, with a production of 15.3 million tons and acreage of 1.97 million hectares in 2011, covering 32.2% of all arable land (http://www.defra.gov.uk/statistics/foodfarm). The average yield in 2011 was 7.7 tonnes per hectare. Wheat is predominantly grown as a winter crop, with the spring wheat area representing less than 5 % of the total UK wheat area.

Based on UK National List (NL) and Recommended List (RL) trials over the period 1948–2007, Mackay et al. (2011) showed a yield increase of 4.2 tonnes per hectare over 60 years, with yield gains from the last 20 years being attributed primarily to genetic improvement of UK wheat varieties, whereas both breeding and agronomy contributed to the preceding 40 years of yield gains.

Wheat varieties in the UK are categorized in four so-called 'nabim' groups (http://www.nabim.org.uk), giving an indication of the likely end use of the grain. Groups 1 and 2 varieties have quality characteristics suitable for bread making and have a higher level of protein. Millers generally offer a premium price for Group 1 varieties as they give a more consistent milling performance compared to Group 2. Group 3 is used for cake, biscuit and batter flour, while Group 4 varieties, whilst tending to have higher yields, have low protein levels and consequently are mainly used for animal feed. On average, 39% of the UK production is used for human consumption (mainly flour production) and industry usage (starch or biofuel production), while 42% is directed to animal feed (Table I-1).

Table I-1: UK wheat supply and demand, adapted from HGCA, UK Cereal Supply and demand, update report March 2012 (http://www.hgca.com/).

The data presented are annual average between July 2006 and June 2011, the number indicated in brackets correspond to home grown part.

	Average (millions tons)	% total availability
Opening stocks	2 034	11.3
Production	14 831	82.1
Imports	1 191	6.6
Total availability	**18 056**	**100**
Human and industrial consumption	6 817 (5 792)	37.8
Usage as animal feed	6 420 (6 256)	35.6
Seed	294	1.6
Others	74	0.4
Total domestic consumption	**13 605**	**75.3**
Balance	**4 452**	**24.6**
Exports	**2 470**	**13.7**

1.2 Origin and taxonomy

Wheat was one of the first cereals to be domesticated in the Fertile Crescent in the Neolithic period. Common wheat, *Triticum aestivum* L. (2n=6x=42), cultivated today, is allohexaploid and evolved through two genome hybridisations of *Triticum* species with

closely related *Aegilops* species. Figure I-2 gives a possible model for the phylogeny of *Triticum aestivum*, however the exact speciation events and the species involved are still debated (Salamini et al., 2002).

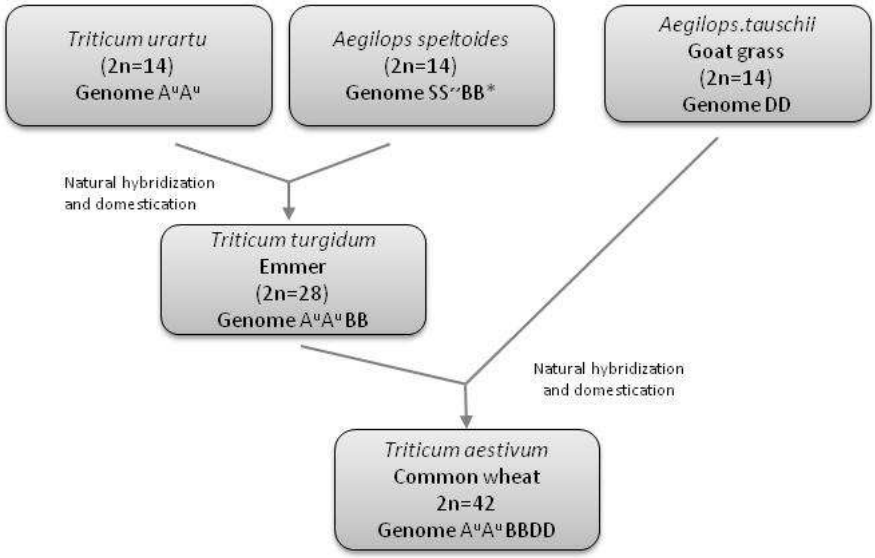

Figure I-2: Schematic description of the origin of hexaploid wheat, *Triticum aestivum*.

(*) a specie related to *Aegilops speltoides* is likely to be the ancestor of the wheat B not directly *A. speltoides*. Adapted from Gill et al. (2004)

Triticum aestivum is classified in the division *Magnoliophyta*, class *Liliopsida*, order *Cyperales*, family *Poaceae* (Gramineae).

1.3 Genetic structure

Among agricultural crops, bread wheat has the largest genome size, estimated to ~17 000 Mb (Bennett and Leitch, 1995). The hexaploid wheat genome consists of seven groups of six chromosomes; each group contains a set of three pairs of homeologous chromosomes from the three ancestral genomes: A, B, and D. Related chromosomes
have similar gene content and order but are divergent in their repetitive DNA content. Based on the analysis of large tracts of genomic sequence from wheat, Sabot et al. (2005) showed that 7.8 % of the wheat genome consist of gene derived sequences, while transposable element (TE) sequences make up 54.7% of the total genome, with the A genome presenting a higher TE content than those of the B and D genomes.

Despite their close homology, homeologues are normally prevented from pairing at meiosis by the *Ph1* (Pairing homeologous 1) locus on 5B (Moore et al., 2009), so hexaploid wheat behaves much like a diploid organism at meiosis.

In wheat, the gene distribution is variable along chromosomes, as genes are clustered in gene-rich regions, separated by long stretch of TEs as demonstrated by physical mapping (Faris et al., 2000; Gill et al., 1996).

Additionally, chromosomal rearrangements are relatively common within hexaploid wheat (Badaeva et al., 2007) and have even been encouraged by breeding programs. The wheat-rye chromosomal translocation 1BL.1RS carrying resistance genes *Lr26*, *Sr31*, *Yr9* and *Pm8* (McIntosh et al., 1998b) has been integrated into lines developed by the International Maize and Wheat Improvement Centre (CIMMYT) and largely distributed worldwide (Rabinovich, 1998). The 5B:7B and 3B:6B translocations are also found frequently in European wheat (Badaeva et al., 2007).

1.4 Genomic resources

A number of *Triticum* genomic resources have been developed or are being developed. These include a collection of over 1 858 000 Triticea expressed sequence tags (EST) available to the public from the National Centre for Biotechnology Information (NCBI) GenBank dbEST database. It also includes bacterial artificial chromosome (BAC) libraries representing several millions clones, and over 16 000 bin-mapped EST markers (Qi et al., 2004).

Hexaploid wheat can tolerate the loss of whole chromosomes, arms or segments as it can compensate by the presence of homologous chromosomes. This tolerance was exploited to create extensive sets of aneuploids from the variety Chinese Spring for each of the 21 chromosome of wheat (Sears, 1954, 1966). In addition, Endo and Gill (1996) developed 436 deletion (del) lines with chromosome-segment deletions. All these genetic stocks has been and are still widely use for cytogenetic studies, particularly to map genes or markers to specific chromosomes, chromosome arms or chromosomal bins (region delineated by neighbouring deletion breakpoint).

For the past 20 years, comparative genomics has contributed greatly to the analysis of complex genome such as the wheat genome. Studies within the grass family *Poaceae*, shown high levels of conservation of gene content and gene orders over millions of years of evolution (Gale and Devos, 1998; Moore et al., 1995). The concept of colinearity (gene order) and synteny (gene content) between grass species provided a useful framework for analysis of the wheat genome: deductions about gene order content and location of putative intron-exon boundaries based on alignments of crop sequences to fully sequenced model genomes, were helpful in the development of generalized monocot genomic tools (Paterson et al., 2009). In the past, rice served as a good model for studies involving comparative

genomics of grass species, because of its small size genome and the availability the whole genome sequence. However, in recent studies, the wild grass *Brachypodium distachyon* has emerged as a better model for temperate cereals including wheat (Kumar et al., 2009). Comparing to rice, *Brachypodium distachyon* is more closely related to wheat at biological and phylogenetic level (Garvin et al., 2008).

In 2005, the international wheat genome sequencing consortium (IWGSC) was created with the aim of accelerating the wheat improvement by developing DNA based tools and products though the creation of a physical map anchored on the genetic map and ultimately obtaining the complete genome sequence of hexaploid bread wheat (http://www.wheatgenome.org). Key to the "chromosome-by-chromosome" strategy adopted by the IWGSC was the ability to flow sort nearly all the chromosomes of hexaploid wheat variety 'Chinese Spring' for which comprehensive nullitetratsomic and deletion line resources were available. The feasibility of this strategy was demonstrated by the creation of an ordered physical BAC contig map of chromosome 3B (Paux et al., 2008) and currently, physical mapping projects have been initiated for each wheat chromosomes. More recently, direct sequencing of a flow-sorted chromosome arm has been carried out, although the assembly is fragmented and relies greatly on conservation of synteny for ordering of contigs (Berkman et al., 2011) and a number of projects are underway toward the sequencing of the reference wheat genotype Chinese Spring (http://www.wheatgenome.org). Next generation sequencing (NGS) technologies have also accelerated SNP discovery. For example, Allen et al. (2011) developed a panel of 1114 single-nucleotide polymorphisms (SNP) markers based on the analysis of expressed sequence tags derived from public sequencing programmes and next-generation sequencing of normalized wheat complementary DNA libraries and more than 100,000 putative varietal SNPs have been databased (Wilkinson et al., 2012).

2 WHEAT YELLOW RUST

2.1 Taxonomy and distribution

Puccinia striiformis Westend f.sp. *tritici* Eriksson (henceforth abbreviated to Pst), the causal agent of yellow (or stripe) rust on wheat (WYR), is currently the most globally damaging cereal rust (Wellings, 2011). Together with the causal agents of brown rust *(Puccinia triticina)*, black rust (*Puccinia graminis* f. sp. *tritici)*, *P. striiformis* f.sp. *tritici* belongs to the family *Pucciniaceae*, order *Uredinales* and class *Basidiomycetes*.

Pst is well distributed worldwide (Wellings, 2011). WYR was previously considered to be a low temperature disease, more adapted to cool and wet climes, but in recent years, isolates adapted to higher temperature emerged. WYR occurs particularly in northern Europe, the Middle East, the east African highlands, China, the Indian sub-continent, the

west coast of the USA, South America, Australia and New Zealand (Saari and Prescott, 1985). Severe infection from the pathogen can reduce grain quality, grain weight, number of grains per spike and plant height (Ma and Singh, 1996). Although in most areas, the range of yield losses observed is between 10% to 70%, the incidence and impact of the disease depends on the cultivar, the date of the initial infection, the disease development and duration (Chen, 2005).

Pst isolates can be categorised into physiological races. A race is defined by Roelfs et al. (1992) as "a non-random assemblage of virulences and avirulences as determined on a series of differential hosts". Races are differentiated by infection types produced on a set of selected plant genotypes, differentials, with one or a limited number of race-specific YR genes. New races are constantly emerging as new cultivars are released (Chen et al., 2009; Chen, 2005; De Vallavieille-Pope et al., 2012). In recent years, Pst strains with wider virulence and increased aggressiveness and adaptability have been described in Europe, Australia and United States (Hovmoller et al., 2008; Milus et al., 2009).

2.2 Life cycle

Pst is a biotrophic obligate parasite dependent on the host plant to complete its life cycle. Pst has a heteroecious life cycle (Figure I-3) involving two unrelated hosts; a microcyclic asexual stage on a graminaceous host and a sexual stage which occurs on an alternative host.

The primary host of Pst is bread wheat (*Triticum aestivum*). Until recently, Pst had no known alternative host. However, in 2010, Jin et al. (2010) demonstrated that *Berberis* spp. may serve as alternate hosts.

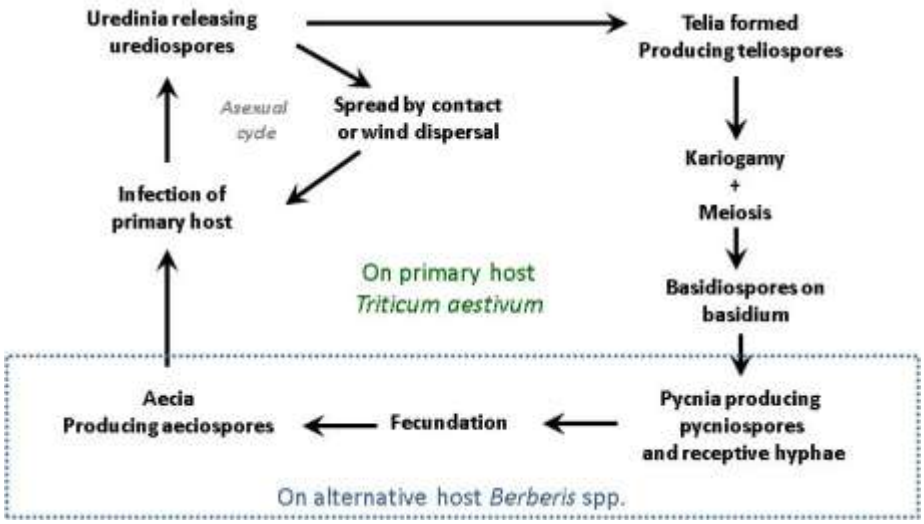

Figure I-3: Schematic diagram of the heteroecial cycle of *Puccinia striiformis* f.sp. *tritici*

2.3 Symptoms

Pst can infect the green tissues of wheat but also some other cereal crops (e.g. Barley, Triticale) at any stage of their development. The fungus forms round, yellow-orange pustules (uredias) which contain thousands of urediospores (Figure I-4). Depending on the susceptibility of the variety, chlorosis and necrotic lesions appear on leaves with or without sporulation. In severe attacks, the fungus can also affect the ears, resulting in the formation of masses of spores between the grain and the glumes.

Figure I-4: Symptoms of WYR

(a) WYR uredias on wheat seedlings (photo UKCPVS). (b) Characteristic symptoms in stripes on adult wheat. (c) Intense sporulation on highly susceptible adult wheat. (d) Necrotic lesions on adult wheat.

2.4 Mode of infection (asexual cycle)

Infection starts by the adhesion of urediniospores on the surface of the leaves with an adhesion pad. Next, the urediospores produce a germ tube which grows toward the stomata guided by the topography of the leaf epidermal pavement (Mendgen and Hahn, 2002). Once the germ tube has reached the stomatal opening, the fungus can form an appressorium which makes an infection peg. However appressoria are rarely observed during Pst infection and are often under-developed (De Vallavieille-Pope et al., 1995; Moldenhauer et al., 2006; Wang et al., 2007). Alternatively, and more commonly, the germ tube directly penetrates the stomatal cavity and successively differentiates into infection structures within the cavity e.g. substomatal vesicles, infection hyphae, haustorial mother cell, penetration peg and finally haustorium. The haustorium is a sophisticated transfer apparatus localized between the mesophyll cell wall and the cell membrane, allowing the fungus to both deliver effectors, which suppress the hosts defence response as well as to withdraw nutrients from

the living host cells, thus redirecting the metabolic flow of the host (Voegele and Mendgen, 2003).

Following the establishment of haustoria in the vicinity of the stomatal cavity, further colonisation occurs through the development of secondary hyphae within the intercellular space between the mesophyll cells and establishment of additional haustoria. Eventually, the elongating hyphae lead to the emergence of urediniospore initials and pedicels in clusters under the epidermis, from which urediniospores differentiate. Under mechanical pressure the epidermis is ruptured releasing spores into the environment.

2.5 Disease development and epidemiology

The epidemiology of yellow rust is highly dependent on three main environmental factors : temperature, moisture and wind (Chen, 2005). Temperature affects spore germination and infection, latent period, sporulation, spore survival, and host resistance. Moisture affects spore germination, infection and survival. Wind plays a major role in the spread of the disease.

The fungus overwinters on autumn sown wheat or volunteers as dormant uredinomycelia or actively sporulating lesions. The latent period for stripe rust during the winter can be up to 118 days (Zadoks and Bouwman, 1985). Rapilly (1979) considered that temperatures under -10˚C stop pathogen development.

In the spring, as soon as daytime temperatures reach 5˚C, pathogen growth can start again to produce sporulating lesions. Yellow rust is spread by the urediospores, by contact for adjacent plants and by wind over longer distance. Viable spore transmission over distances of more than 800km have been reported by Zadoks (1961), despite their susceptibility to ultraviolet light.

The complete cycle from infection to the production of new urediospores takes about two weeks under optimum conditions. Several disease cycles may therefore be completed in one season.

2.6 *Puccinia striiformis* population dynamics and genetics

Pst is thought to have originated from Transcaucasia, where its primary host ancestor also originated. From there, the pathogen would have evolved and dispersed in to Europe, East Africa and along the mountain ranges to China and eastern Asia. It is believed it reached America via land bridge or prehistoric human migration (Stubbs, 1985).

More recently, besides effective step by step wind dispersal, human agency has played an important role in the global spread of Pst. The first Pst detected in Australia in

1979, is believed to have been introduced from Europe via airplane transportation (Wellings et al., 1988). Soon after, wind carried the disease to New Zealand (Wellings and McIntosh, 1990). The last century saw also Pst progression through the African continent towards South Africa where the pathogen was first detected in 1996 (Pretorius et al., 1997).

The increasing global range of Pst and regular emergence of new races with wider virulence and increased aggressiveness demonstrates its great potential for adaptation and for causing renewed epidemics all over the globe.

Until very recently, Pst was considered to reproduce exclusively asexually and the variation of the pathogen was thought to come exclusively from mutations. This view is supported by the progressive virulence accumulation observed following the introduction of new race-specific host resistances (i.e. successive fixation of new factors under positive selection) at the regional level (Bayles et al., 2000; Chen et al., 2009; De Vallavieille-Pope et al., 2012; Wellings, 2007). Additionally, many genetic studies of Pst population based on various molecular markers systems (RAPD, IGS, AFLP, SSR, SRAP), support the hypothesis, as they showed low genetic variation and typical clonal population structures in western Europe (Enjalbert et al., 2005; Hovmøller et al., 2002; Komjáti et al., 2004) , USA (Chen and Line, 1993b) and Australia (Steele et al., 2001). Using high density AFLP, Hovmøller and Justesen (2007b) resolved the single step changes in three European pathogen lineages confirming the stepwise mutation process leading to the acquisition or loss of virulence factors and ultimately to new races.

However, other studies suggested that genetic recombination might exist in some Pst populations. Relatively higher genetic diversity has been reported in China (Duan et al., 2010; Shan et al., 1998), Middle East (Bahri et al., 2009) and Pakistan (Bahri et al., 2011). Mboup et al. (2009) revealed the presence of genetic recombination between molecular markers suggesting the existence of a sexual or parasexual cycle in Pst populations from Gansu province, China. The postulated ability of Pst to complete a sexual cycle seemed to be supported by the description of *Berberis spp.* as alternative hosts (Jin et al., 2010) and the higher rate of sexual spore production in Asiatic genotypes (Ali et al., 2010).

Beside population structure studies of the WYR pathogen, efforts have been made in genomic and functional genomics studies in order to identify molecular mechanisms controlling the host–pathogen interaction and particularly effector genes which subvert the plant innate immune response and enable the infection to occur. Several genetic libraries for Pst have become available, including a BAC library (Chen and Ling, 2004), a cDNA library from urediniospores (Chen and Ling, 2004), a germinated urediniospores EST library (Zhang et al., 2008), and a haustorial EST library (Yin et al., 2009). Based on those resources and the genome sequence of *P. graminis* f. sp. *tritici*, the wheat stem rust pathogen, Ma et al. (2009) produced a preliminary physical map locating 1,432 Pst genes. Using next generation

sequencing (NGS), Cantu et al. (2011) provide a draft of the genome of Pst race PST-130 consisting of an assembly of 29,178 contigs available through GenBank covering at least 88% of the Pst genes; they also predicted 20,423 proteins and listed 1,088 candidate effectors protein based on comparative genetic. Another sequencing project is in progress as part of the Fungal Genome Initiative at the Broad Institute (http://www.broadinstitute.org/) with the objective to release a high quality reference sequence of race Pst-78 from the USA. Once the reference sequence is available, NGS of multiple Pst races and comparative sequence analysis will greatly help to identify candidate genes for effectors recognized by specific resistance genes.

2.7 Control

Wheat yellow rust epidemics can have a devastating impact on yield (Wellings, 2011), so integrated disease control in high risk conditions is essential. There are two main rust control strategies: use of varietal resistance and chemical control. Additional crop management measures can decrease the risk of early infection and disease spread, such as controlling volunteers between harvest and emergence of new crop, delaying sowing date and diversifying varieties grown on a local area.

Chemical control is based on azole, strobilurin, morpholine and succinate dehydrogenase inhibitor fungicides (HGCA, 2012), all known to be effective to control rust diseases (Chen, 2005) and up to now, fungicide resistance in wheat rusts is limited (Bounds et al., 2012). Control is achieved by timely foliar applications. Seed treatment with broad spectrum systemic fungicide can also provide early protection. Despite the availability of efficient fungicides, chemical control does not provide an economically viable solution for the majority of wheat growing regions, where the return on investment is low and access is difficult (Chen, 2007; McIntosh et al., 1995). Moreover, the use of fungicides is not without environmental impact and an unnecessarily high usage of fungicide may favour the emergence of pathogen resistance.

The use of resistant wheat cultivars represents the most efficient alternative to control yellow rust, with the advantages of being both economical and environmentally friendly. Thanks to the breeding effort worldwide, numerous sources of resistance have been deployed in the development of resistant cultivars and commercialised. However, there are many examples of rapid breakdown of resistance genes as illustrated by data gathered over 60 years in the UK (Table I-2). Even the presence of multiple resistance genes within a cultivar does not necessarily provide long lasting resistance.

The control of cereal rusts is particularly marked by a “boom and bust” cycle. The “boom” refers to the sudden increase in the area of a resistant cultivar with a single major

resistance gene effective against the majority of the contemporaneous Pst population. The "bust" refers to the process whereby a new pathotype virulent on the resistance gene deployed in the 'booming' cultivar appears and creates a dramatic epidemic, leading to a rapid adjustment of breeding strategies to select on some other resistance gene which is still effective to the new pathotype.

On the other hand, there are a number of examples where varietal control was a success as wheat cultivars remained resistant for their commercial life, among them: Deben (Christiansen et al., 2006), Alcedo (Jagger et al., 2011), Guardian (Melichar et al., 2008), Cappelle Desprez (Agenbag et al., 2012), Cadenza (personal communication Rosemary Bayles), Luke (Guo et al., 2008), among others

Table I-2: Some of UK wheat cultivars that have seen their resistances broken down in within years of release.

(1)Years up to 1990 refer to Johnson (1992a), after 1990 the year refers to the first UKCPVS description of Pst isolates virulent at adult plant stage on the specified cultivar. (2) It is not clear if virulence for genes in parentheses was necessary. * Adult plant resistance gene

Cultivar name	Year first commercialized	Year resistance breakdown(1)	Gene combination overcome
Nord Desprez	unknown	1952	*Yr3a,Yr4a*
Heines VII	unknown	1955	*Yr2*
Rothwell Perdrix	1964	1966	*Yr1, (Yr2,Yr6)(2)*
Maris Templar	1968	1968	*Yr1, Yr3a, Yr4a*
Maris Beacon	1968	1969	*Yr3b,Yr4b*
Maris Ranger	1968	1969	*Yr3a, Yr4a, Yr6*
Joss Cambier	1968	1971	*Yr2, Yr11**
Talent	1971	1972	*Yr7*
Maris Bilbo	1970	1972	*Yr3a, Yr4a, Yr14**
Maris Huntsman	1972	1974	*Yr2, Yr3a, Yr4a, Yr13**
Maris Nimrod	1971	1974	*Yr2, Yr3a, Yr4a, Yr13**
Kinsman	1975	1975	*Yr3a, Yr4a, Yr6, Yr13**
Clement	1975	1975	*Yr2, Yr9*
Brigand	1979	1981	*Yr2, Yr3a, Yr4a, Yr13*, Yr14**
Stetson	1983	1983	*Yr1, Yr9*
Slejpner	1985	1985	*Yr9, + Yr* unknown*
Hornet	1987	1988	*Yr2, Yr6, Yr9*
Brock	1985	1988	*Yr7, Yr14**
Hereward	1991	1991	*Yr2,Yr3a,Yr4a, Yr32, + Yr* unknown*
Brigadier	1993	1995	*Yr9, Yr17, + Yr* unknown*
Madrigal	1995	1996	*Yr6, Yr9, Yr17*
Oxbow	2000	2000	*Yr9,(Yr17)(2),Yr32*
Robigus	2003	2004	*Unknown (infected by WYR isolates with virulence factor 9,17,32)*
Warrior	2009	2011	*Unknown (infected by WYR isolates with virulence factor 6,7,9,17,32)*

3 PLANT DEFENCE MECHANISM AND DISEASE RESISTANCE

Plants are constantly facing biotic stresses however this constant exposure leads rarely to the development of the disease as plants have developed a wide range of defence

mechanisms, passive as well as active. Interactions between plants and pathogens are generally classified as compatible or incompatible. In compatible interactions, the pathogen is able to develop and spread within the plant host. In incompatible interactions, the growth of a pathogen is limited or stopped, resulting in reduced symptoms or the absence of the disease. To allow the establishment of the disease, the pathogen must overcome constitutive and inducible defence systems which require prior recognition between the plant and the pathogen. Understanding the defence mechanisms involved in resistant cultivars particularly to WYR would have great implications in breeding for durable resistance.

3.1 Constitutive defence mechanisms

The passive or constitutive defence system is composed of physical barriers such as waxy cuticles and reinforced cell walls, and chemical barriers, which protect against the initial pathogen invasion. The constitutive chemical barrier include the presence of secondary metabolites with anti-microbial properties named photoanticipins (Osbourn, 1999), and anti-microbial proteins which inhibit the growth and development of the pathogen including inhibitors of essential pathogen enzymatic activities, hydrolytic enzymes, lectins and defensins (Boman, 1995; Broekaert et al., 1997; García-Olmedo et al., 1998; Heath, 2000; Ryan, 1990; Shewry and Lucas, 1997).

Constitutive defences occur in a non specific manner and are believed to participate to horizontal or non-race specific resistance.

3.2 Inducible defence mechanisms

Alongside preformed defences, plants possess inducible defence mechanisms activated following the recognition of a pathogen. These comprise both rapid structural and biochemical changes.

The first visible line of defence is a cell wall apposition, or papilla (Aist, 1976), a deposit of callose, lignin and proteins between the cell wall and the cell membrane at the site of attempted penetration. Additionally, deposition of lignin (Vance et al., 1980), callose, hydroxyl rich glycoprotein (Mazau and Esquerré-Tugayé, 1986) and cross-linking (Brisson et al., 1994) reinforce the cell walls. Those structural modifications are believed to slow down the pathogen by making the cell wall less digestible and the cell less accessible.

Following pathogen recognition, a modification of membrane permeability generating ions fluxes such as export of K^+ and Cl^- and import of H^+ and Ca^{2+} is seen. In

addition, changes in protein phosphorylation and production of reactive oxygen species (ROS) are observed (Ebel and Mithöfer, 1998). The changes in ion fluxes and particularly the increased concentration of Ca^{2+} in the cytoplasm are involved in the activation of defence related genes. The accumulation of ROS (hydrogen peroxide, superoxide anion and hydroxyl radical), also named oxidative burst, is a rapid reaction (occurring within minutes of perception) of plants to infection (Torres et al., 2006). In addition to being toxic to the pathogen (Mehdy, 1994; Peng and Kuc, 1992), ROS promotes the cross-linking of cell-wall in proteins (Bradley et al., 1992) and activates a cascade of defence-related gene expression. Moreover, the generation of ROS is a characteristic feature of the hypersensitive response (HR), a programmed cell death response which prevents the spread of the pathogen (Heath, 1998; Lamb and Dixon, 1997); however it is still unclear if ROS are directly causing cell death.

Concomitant with the oxidative burst, phytoalexins and pathogenesis related (PR) proteins are synthesised, both present anti-microbial properties. Phytoalexins (reviewed by Ahuja et al., 2012; Sels et al., 2008) are secondary metabolites which accumulate on the site of infection following plant-pathogen interactions, in contrast with phytoanticipants which are constitutive. They are broad spectrum inhibitors and are chemically divers, among them figure some terpenoids and flavonoids. PR proteins (reviewed in Sels et al., 2008; van Loon et al., 2006) are produced during a plant pathogen interaction, they have been classified in 17 PR families according to their biochemical properties and biological activity. Many PR proteins have roles in degrading pathogen cell wall: endo-β-1,3-glucanase (PR-2), endochitinases (PR-3 ,4 , 8, and 11) and endoprotease (PR-7). Others target the plasma membrane of fungal pathogens like defensin (PR-12), thionin (PR-13) and lipid transfer protein (PR-14). Reinforcement of plant cell wall (PR-9 peroxidase), production of hydrogen peroxide (PR 15, 16 and 17 oxidases proteins) are additional activities related to PR proteins.

Besides those localized defence responses following the early stage of the pathogen attack, plants present a more generalized response to face further colonisation, referred as systemic acquired resistance (SAR)(Ryals et al., 1996) . Signal molecules such as jasmonic acid and salicylic acid are synthesised and act as long distance signals to regulate a broad-spectrum of local and systemic defence mechanisms (Heil and Ton, 2008).

The active defence response can occur both in a specific and a non specific manner. The same set of genes seems to be expressed in both susceptible and resistant plants as illustrated by microarray studies (Caldo et al., 2004; Schneider et al., 2011; Tao et al., 2003) with differences in timing and magnitude of response likely to explain the difference between compatible and incompatible plant-pathogen interaction.

3.3 Plant-pathogen interaction and co-evolution

Active plant defence is highly dependent upon plant-pathogen recognition, which involves general and specific pathogen elicitors and plant molecules.

Active plant responses to aggression fall into two categories depending on the specificity of the plant-pathogen recognition. The first category referred to non-specific response as the plant recognizes and responds to molecules common to many classes of pathogens, referred as pathogen-associated molecular patterns (PAMPs, also called MAMPs) (Heath, 2000; Mackey and McFall, 2006), such as chitin polymer derived from cell wall of fungus (Wan et al., 2008). The recognition of those general effectors passes by transmembrane pattern recognition receptor (PRR) and triggers a non-specific defence response commonly referred to as PAMP-triggered immunity (PTI). PTI is generally sufficient to stop the majority of pathogens. Successful pathogens, however, are capable of suppressing PTI through the action of pathogen- specific effector proteins, reducing the host response to its basal level (Bent and Mackey, 2007). For instance, plant pathogen *Phytophthora infestans* secretes extracellular protease inhibitors EPI1 and EPI10 that were shown to inhibit and interact with the PR protein P69B subtilase of tomato (Tian et al., 2005).

In return, plants have evolved R proteins which directly or indirectly detect effectors produced by specific races of pathogens, triggering a race-specific response of large amplitude, referred to as effector-triggered immunity (ETI). The activation of R proteins is often accompanied by a hypersensitive response (HR) that can block the progress of the virulent pathogens (Dangl and Jones, 2001).

Because of its specificity, ETI is often challenged by natural selection. Under selective pressure, pathogens can overcome ETI by evolving alternative effectors that suppress the ETI or are no longer recognized by R proteins (Houterman et al., 2008), reverting resistance response to its basal level. To counteract the pathogen evolution, selective pressure on the host plant could then lead to new R proteins capable to recognize new effectors. The evolutionary battle between plant and pathogens whereby efficiency of defence response of the host population are reduced when individuals within the pathogen population acquire a new suppressive or evasive mechanism and is regained when individuals from the host population evolve new recognition specificities is referred to as the zigzag model (Jones and Dangl, 2006)(Figure I-5).

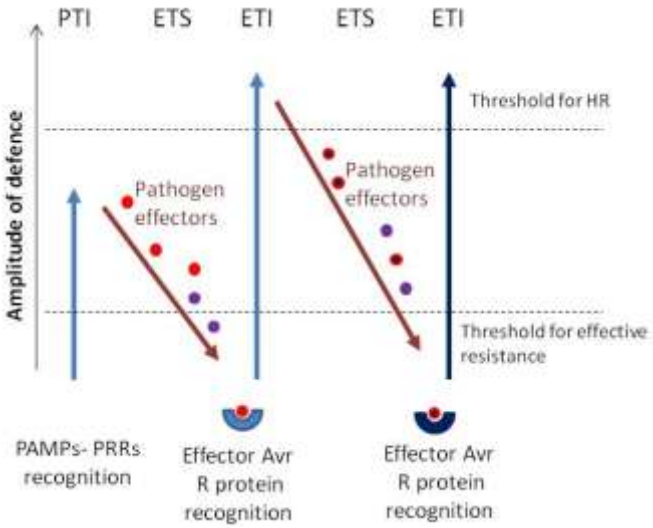

Figure I-5; Evolutionary view of multi-layered molecular plant-pathogen interactions illustrated by the 'zigzag' model.

PAMPs: pathogen-associated molecular patterns; PRRs: pattern recognition receptors; PTI: PAMPs-triggered immunity; ETS: effector-triggered susceptibility; Avr: pathogen-derived avirulence factor; R-protein: resistance protein ETI: effector-triggered immunity; HR: hypersensitive response. Adapted from Jones and Dangl (2006).

3.4 Plant resistance: the molecular approach

One approach to understand the molecular mechanism underlying resistance in plant is the identification and functional analyses of genes that are involved in the resistance.

Many *R*-genes conferring race-specific resistance have been isolated from several plants species (Dangl and Jones, 2001). *R*-genes seem to encode receptors that detect the presence of the pathogen directly or indirectly and activate signalling cascade, which gives rise to resistance reaction (Hammond-Kosack and Jones, 1997). Despite the diversity of host-pathogen systems, resistance genes share common motifs, and five major classes of *R*-genes are now recognised (Figure I-6). The largest group of *R*-genes code for proteins characterized by the presence of a nucleotide binding site NBS and a leucine rich repeat (LRR domain). LRR domains are involved in protein-protein interaction and are the major determinant of recognition specificity (Fluhr, 2001). The NBS region has been proposed to control plant cell death (Van der Biezen and Jones, 1998).

Plant *R*-genes are commonly clustered in the genome and well conserved between plant species (Hammond-Kosack and Jones, 1997; Hulbert et al., 2001a; Michelmore and Meyers, 1998). Resistance gene clusters are thought to be the result of gene duplication and wild range of recombination events; they also appear to evolve more rapidly than other regions of the genome (Richter and Ronald, 2000).

The highly conserved motifs in *R*-gene allows the identification of putative *R*-gene sequence homology as illustrated by the works of Meyers et al. (1999) and Bai et al. (2002). A method referred as NBS profiling takes advantage of well conserved motif identified within the NBS domain to develop marker systems targeting *R*-genes (van der Linden et al., 2004).

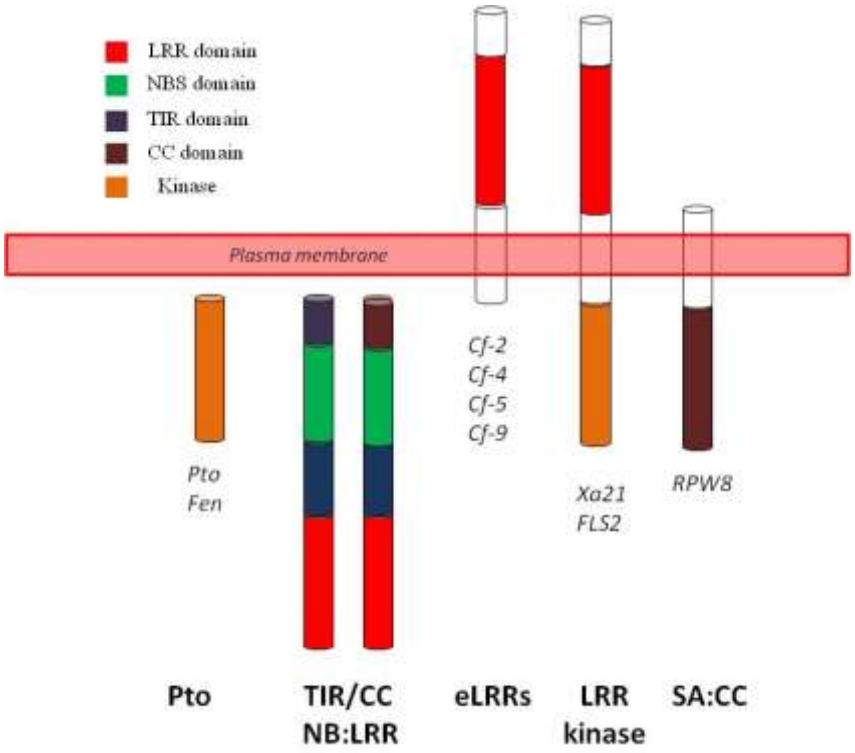

Figure I-6: Schematic representation of main classes of *R* proteins.

Some exemplar *R*-proteins are indicated in italics for each group.
The *Pto* class is characterized by a Serine/threonine protein kinase with a N-terminal myristoylation site. The TIR/CC NB:LRR group includes a nucleotide binding site (NBS), a leucine rich repeat motif (LRR) and either a coiled coil (CC) sequence or a Toll and Interleukin-1 receptor type region (TIR). These two classes are thought to be localized intracellularly. The eLRRs and LRR kinase classes are transmembrane protein with extracellular LRRs, in addition LRR kinases possess a cytoplasmic protein kinase domain. Finally, the SA:CC class carries a putative signal anchor (SA) for membrane insertion, and a putative CC domain.
Adapted from Dangl and Jones (2001) and Jones (2001).

4 HOST RESISTANCE TO WYR

Based on the plant stage they are expressed, their genetic basis and race specificity, different types of WYR resistance have been characterized. Classically, a distinction is made between seedling disease resistance (SDR) and the adult plant resistance (APR).

4.1 Seedling resistance

SDR is better described as 'all stage' resistance; although it is the only type of resistance that can be detected at the seedling stage (hence the name), full resistance continues to be expressed through all subsequent stages of plant growth. SDR is race specific (Chen and Line, 1995). A race-specific resistance is characterized by being expressed by only certain genotypes of the host species and being effective against only certain genotypes or races of the specific pathogen (Heath, 1995), it involves parasite-specific effector-triggered recognition processes that induce the defence mechanisms of the plant. Genetically, this type of recognition is said to involve a gene-for-gene relationship between an avirulence (Avr) gene in the parasite and resistance gene (*R*-gene) in the plant (Flor, 1956). SDR is also characterized by a qualitative (all-or-nothing) resistance response. Although only one WYR NBS-LRR gene (*Yr10*) has been cloned and no WYR avirulence genes or effectors formally identified, it is generally accepted that most avirulence genes are likely to be what are now called 'effectors' and that most SDRs will involve specific recognition of effector proteins by *R*-genes of one of the known classes. This type of monogenic resistance is easily evaded as a single mutation in the pathogen effector/avirulence gene can render an R-gene ineffective; therefore it does not provide long term protection. To infect a host individual with several R-genes, a fully avirulent pathogen would need to evade recognition by each gene in one generation, which is considered unlikely. However if R-genes used in combination are also present singly in some cultivars, the pathogen could become virulent to each one in a stepwise manner rendering the pyramid ineffective. There are many examples of wheat varieties containing combinations of race specific resistances that broke down within a few year of their release (see Table I-2, section 2.7); for example the resistance of variety 'Brigadier' released in 1993 (*Yr9* and *Yr17)* broke down in 1995 (Bayles et al., 2000).

4.2 Adult plant resistance

APR is only expressed as the plant matures into its reproductive phase. The genotypes carrying this type of resistance will appear susceptible at the seedling stage but resistant at adult plant stage. APR can be race-specific, as exemplified by *Yr11, Yr12, Yr13* and *Yr14* or non race specific, for instance *Yr18*. A non-race specific resistance confers a broad spectrum resistance against a range of virulent races belonging to a particular pathogen. APR can be monogenic, controlled by one gene, or polygenic, where the expression of two or more genes with partial effect is required to confer the full level of resistance observed. APR confer a quantitative disease resistance, associated with non-HR

response. Considering the rust infection, APR are usually associated with slow rusting phenotype, which is defined as a reduce rate of development of the pathogen with a longer latent period and a reduce rate of spore production. Quantitative resistances have often shown a greater field resistance and appear to be a more durable form of resistance (Mallard et al., 2005; Parlevliet, 2002).

The term durable resistance was introduced and described by Roy Johnson as a "resistance that remains effective during its prolonged and widespread use in an environment favourable to the disease" (Johnson, 1984). However this definition does not make any statement or inference about the genetic control of the resistance or its race specificity (Johnson, 1981). Nevertheless, rust experts worldwide generally agree that non-specific APR, quantitatively inherited are more likely to provide long term resistance comparing to race specific and/or monogenic resistance (Johnson, 1988; Parlevliet, 2002; Singh, 2012).

Some APR are additionally labelled high-temperature adult plant resistance (HTAP) for instance *Yr18* and *Yr36*, based on the observation that resistance is only expressed above a certain threshold of temperature. HTAP resistance is durable and non-race specific (Chen, 2005), the effectiveness of HTAP increasing as the temperature rises over the course of the growing season and plants mature. HTAP resistance reduces both the rate and severity of infections, as well as lowering the amount of secondary inoculum. HTAP resistance is generally expressed as a partial resistance and its level in fields is highly influenced by inoculum pressure and fluctuations in atmospheric temperature. Thus, the HTAP resistances may not provide adequate resistance level in cooler climates as demonstrated by Johnson (1992a), who evaluated American cultivars Wanser, Gaines, Nugaines and Luke with HTAP resistance in the UK.

4.3 WYR resistances genes

At the time of writing, 52 genes for resistance to yellow rust have been formally named (*Yr* followed by a number) (Table I-3) and assigned to a wheat chromosome apart from four APR genes (*Yr11,12,13,14*). An additional 37 reported genes have been provisionally named (*Yr* followed by letters) (Table I-4), pending a more definitive characterization. Multiple alleles have been reported for the *Yr3* and the *Yr4* loci. Most of the 89 genes are unique as indicated by their different chromosomal locations, their race-specificity and germplasm source. Many genes were assigned to a specific chromosome using cytogenetic methods such as evaluation of monosomic or substitution lines. More recently, the time consuming process of chromosome location using aneuploidy (McIntosh et al., 1995) has been superseded by genetic mapping using molecular markers, which not only

resolves the chromosome on which the R-gene resides, but with today's dense marker coverage can give a relatively accurate intra-chromosomal location for the gene.

Table I-3: List of designated genes for resistance to Pst, chromosomal locations, examples of genotype containing the genes and origins, types of resistance and references.

[a] RS race-specific resistance; NRS Non-race-specific resistance; TSSDR Temperature sensitive seedling disease resistance; APR Adult plant resistance. All APR genes are highlighted with blue background.
[b] *Yr3b* was originally reported in Hybrid 46 by Lupton and Macer (1962), but Chen and Line (1993) found that a second gene in Hybrid 46, presumably this gene, was not located at the *Yr3* locus, and provisionally named it *YrH46*.
[c] *Yr4* first described as undesignated allele for *Yr4a* and *Yr4b* by Lupton and Macer (1962). In 2010, Bansal et al. (2010) concluded was *YrRub* is *Yr4* based on specific similarities and the presence similar molecular markers in cvs. Avalon, Bolac, Emu and Rubric. However, the 3BS location is not consistent with that listed below for *Yr4a and Yr4b*.
[d] *Yr34* Bariana et al. (2006) shows also a weak seedling resistance.

***Yr* gene**	**Alternative name**	**Chromosomal location**	**Germplasm Source**	**Resistance type[a]**	**References**
Yr1	L	2AL	Chinese 166	**RS, SDR**	Lupton and Macer (1962); McIntosh and Arts (1996)
Yr2	U	7B	Kalyansona, Heines VII	**RS, SDR**	Lupton and Macer (1962); Chen et al. (1996)
Yr3	allele *Yr3a,Yr3b*, *Yr3c*	undesignated allele	Vilmorin 23, Minister	**RS, SDR**	Lupton and Macer (1962)
Yr3a	presumably *YrV23*	1B, 2BL	Nord Desprez, Vilmorin 23, Cappelle-Desprez	**RS, SDR**	Lupton and Macer (1962); Chen et al. (1996), Wang et al. (2006)
Yr3b[b]		unknown	Hybrid 46	**RS, SDR**	Lupton and Macer (1962); Chen and Line (1993b)
Yr3c		1B	Minister, Maris Beacon	**RS, SDR**	Lupton and Macer (1962); Chen et al. (1996)
Yr4[c]	presumably *YrRub*	3BS	Avalon, Rubric, Bolac, Emu S	**RS, SDR**	Lupton and Macer (1962) Bansal et al. (2010)
Yr4a		6B	Cappelle-Desprez	**RS, SDR**	Lupton and Macer (1962); Chen et al. (1996)
Yr4b		6B	Hybrid 46	**RS, SDR**	Lupton and Macer (1962); Chen et al. (1996)
Yr5	presumably allelic to *Yr7*and *YrSp*	2BL	Originated from *Triticum aestivum* subsp. *Spelta* cvs. Album	**RS, SDR**	Macer and MacKey (1966); Law (1976);Zhang et al. (2009)
Yr6	B	7B	Heines Kolben	**RS, SDR**	Macer and MacKey (1966);Elbedewy and Robbelen (1982)
Yr7	Presumably allelic to *Yr5* and *YrSp*	2BL	Originated from *Triticum durum* cvs. Lumillo, Lee Thatcher	**RS, SDR**	Macer and MacKey (1966); McIntosh et al. (1981); Zhang et al. (2009)
Yr8		2A or 2D (2AS.2ML.2MS or 2DS.2ML.2MS)	Originated from *Aegilops Comosa*, Compair	**RS, SDR**	Riley et al. (1968); McIntosh et al. (1982);McIntosh (1988);Friebe et al. (1996)
Yr9		1BS (1BL.1RS)	Originated from *Secale cereale* cvs. Petkus, Clement, Kavkaz, Riebesel 47/51	**RS, SDR**	Macer (1975);Zeller (1973)
Yr10 Cloned	*YrVav*, presumably *YrMor*	1BS	Spelt wheat 415, Turkish line pl178383, Moro	**RS, SDR**	Macer (1975);Metzger and Silbaugh (1970); Laroche et al. (2000)
Yr11	R11	unknown	Joss Cambier, Heine VII	**RS, APR**	McIntosh (1988)
Yr12	R12	unknown	Frontier, Mega	**RS, APR**	McIntosh (1988)
Yr13	R13	unknown	Maris Huntsman, Hustler	**RS, APR**	McIntosh (1988)
Yr14	R14	unknown	Hobbit, Kador, Maris Bilbo	**RS, APR**	McIntosh (1988)
Yr15		1BS	Originated from *Triticum dicoccoides* accession G25, Boston,Cortez	**RS, SDR**	Gerechter-Amitai et al. (1989); McIntosh et al. (1996)

Yr gene	Alternative name	Chromosomal location	Germplasm Source	Resistance type[a]	References
Yr16		2D	Bersee, Cappelle-Desprez	**NRS, APR**	Worland and Law (1986)
Yr17		2AS (2NS.2AS)	originated from *Aegilops ventricosa*, VPM1, Rendezvous, Brigadier	**RS, SDR**	Bariana and McIntosh (1993); Jahier et al. (1996)
Yr18 Cloned		7DS	Frontana, Jupateco R	**NRS, HTAP**	Singh (1992); Dyck et al. (1994); Krattinger et al. (2009)
Yr19	*YrCom*	5B	Compair	**RS, SDR**	Chen et al. (1995a)
Yr20	*YrFie*	6D	Fielder	**RS, SDR**	Chen et al. (1995a)
Yr21	*YrLem*	1B	Lemhi	**RS, SDR**	Chen et al. (1995a)
Yr22	*YrLe1*	4D	Lee	**RS, SDR**	Chen et al. (1995a)
Yr23	*YrLe2*	6D	Lee	**RS, SDR**	Chen et al. (1995a)
Yr24	*YrCH42*, identical to *Yr26*	1BS	Originated from *Triticum turgidum* K733, Chuanmai	**RS, SDR**	McIntosh et al. (1998a); McIntosh and Lagudah (2000)
Yr25		1D	TP129, Strubes Dickkopf, Heines Peko, Heine VII	**RS, SDR**	McIntosh et al. (1998a); Calonnec and Johnson (1998)
Yr26	Identical to *Yr24*	1B	Originated from *Triticum turgidum Haynaldia villosa*, R55	**RS, SDR**	Ma et al. (2001), Wang et al. (2008)
Yr27	*YrSk*	2BS	Selkirk, Ciano 79	**RS, SDR**	McDonald et al. (2004)
Yr28		4DS	Originated from *Aegilops tauschii* W-219, Synthetic	**RS, SDR**	Singh et al. (2000b)
Yr29		1BL	Pavon 76	**NRS, APR**	McIntosh et al. (2001); William et al. (2003)
Yr30		3BS	Opata 85, Parula, Pavon 76	**NRS, APR**	McIntosh et al. (2001)
Yr31		2BS	Pastor	**RS, SDR**	McIntosh et al. (2003); Singh et al. (2003)
Yr32	*YrCv*	2AL	Carstens V, Tres	**RS, SDR**	Eriksen et al. (2004)
Yr33		7DL	Batavia	**RS, TSSDR**	McIntosh et al. (2004); Zahravi et al. (2003)
Yr34[d]	*YrWA*	5AL	WAWHT2046=AUS91389	**APR**	McIntosh et al. (2004); Bariana et al. (2006)
Yr35	*Yrs8*	6BS	originated from *Triticum dicoccoides*, 98M71	**RS, SDR**	Marais et al. (2003)
Yr36 Cloned		6BS	Origined from *Triticum dicoccoides*, RSL 65, UC1041	**NRS, HTAP**	(Uauy et al., 2005); Fu et al. (2009)
Yr37		2DL	Originated from *Aegilops kotschyi*, Line S14	**RS, SDR**	Marais et al. (2005a)
Yr38	*YrS12*	6A (6AL.6L^{sh}.6S^{sh})	Originated from *Aegilops sharonensis*, Line 0352-4	**SDR**	Marais et al. (2003); Marais et al. (2006);
Yr39		7BL	Alpowa	**NRS, HTAP**	Chen and Zhao (2007)
Yr40		5DS (5DL.5DS-T5MS^{G}	Originated from *Aegilops geniculata*, TA5602, Chinese common wheats	**RS, SDR**	Kuraparthy et al. (2007);Kuraparthy et al. (2009)
Yr41	*YrCN19*	2BS	Chuan-nong 19, Line AIM6	**NRS, SDR**	Luo et al. (2005)
Yr42		6A (6AL.6^{aen}L.6^{aen} S)	Originated from *Aegilops neglecta*, Line 03M119-71A	**SDR**	Marais et al. (2009)
Yr43		2BL	IDO377s=PI 591045, Lolo	**RS, SDR**	Cheng and Chen (2010a)
Yr44	*YrZak*	2BL	Zak=PI607839	**RS, SDR**	Sui et al. (2009)
Yr45		3DL	PI181434	**RS, SDR**	Li et al. (2010)
Yr46		4D	PI250413, RL6077 =Thatcher*6/PI250413	**APR**	Herrera-Foessel et al. (2011) Hiebert et al. (2010)
Yr47		5BS	AUS28183=V336	**SDR**	Bansal et al. (2011)
Yr48		5AL	UC1110(S)/PI610750 RIL 167(R)	**APR**	Lowe et al. (2011b)
Yr49		3DS	Chuanmai 18	**APR**	McIntosh et al. (2011) Spielmeyer *et al*.,unpublished
Yr50		4BL	Potentially originated from *Thinopyrum intermedium* , line CH2233	**SDR**	Liu et al. (2012)
Yr51		4AL	AUS27854	**SDR**	(Randhawa et al., 2012)
Yr52		7BL	PI 183527	**HTAP**	Ren et al. (2012a)

Table I-4: List of temporarily designated genes for resistance to stripe rust (*Puccinia striiformis* Westend), chromosomal locations, examples of genotype containing the genes, types of resistance and references.

[a] RS race-specific resistance; NRS Non-race-specific resistance; TSS Temperature sensitive seedling resistance.
[b] *YrA* refers to a phenotype controlled by several gene (KOMUGI, 2008).

***Yr* gene**	**Alternative name**	**Chromosomal location**	**Germplasm example and origin**	**Resistance type[a]**	**References**
YrA[b]		3D and unknown	Avocet R	**RS, SDR**	Wellings et al. (1988)
YrAlp		1BS	Alpowa	**RS, SDR**	Lin and Chen (2007b)
YrC142	Presumably *Yr24/Yr26*	1BS	Originated from synthetic wheat CI142	**RS, SDR**	Wang et al. (2009)
YrC591		7BL	C591	**SDR**	Li et al. (2009)
YrCK		2DS	Cook, Sunco	**TSS**	Park et al. (1992); Bariana et al. (2001); Navabi et al. (2005)
YrCle		4B	Clement	**RS, SDR**	Chen et al. (1995b)
YrCN17		1BS (1BL.1RS)	Originated from *Secale cereale* R14,in line CN12, CN17, CN18	**SDR**	Luo et al. (2008); Ren et al. (2009)
YrD		6A	Druchamp	**RS, SDR**	Chen et al. (1994)
YrDa1		1A	Daws	**RS, SDR**	Chen et al. (1995b)
YrDa2		5D	Daws	**RS, SDR**	Chen et al. (1995b)
YrDru		5B,6B	Druchamp	**RS, SDR**	Chen et al. (1994); Chen et al. (1996)
YrDru2		6A	Druchamp	**RS, SDR**	Chen et al. (1996)
YrExp1		1BL	Express	**RS, SDR**	Lin and Chen (2008)
YrExp2		5BL	Express	**RS, SDR**	Lin and Chen (2008)
YrH46		6A	Hybrid 46	**RS, SDR**	Chen et al. (1996)
YrH52		1BS	origined from *Turgidum dicoccoides* H52	**RS, SDR**	Peng et al. (1999)
YrHVII		4A	Heines VII	**RS, SDR**	Chen et al. (1995b)
YrMin		4A	Minister	**RS, SDR**	Chen et al. (1996)
YrMor		4B	Moro	**RS, SDR**	Chen et al. (1995b)
YrND	presumably *YrMin*	4A	Nord Desprez	**RS, SDR**	McIntosh et al. (1995); Chen et al. (1996)
Yrns-B1		3BS	Lgst.79-74	**NRS, APR**	Borner et al. (2000)
YrP81		2BS	P81, Xu29	**SDR**	Pu et al. (2010)
YrR212		1BS (1BL.1RS)	Originated from *Secale cereale*, R185, R205,R212	**SDR**	Luo et al. (2008)
YrR61		2AS	Pioneer 26R61	**APR**	Hao et al. (2011)
YrS		3B	Stephens	**RS, SDR**	Chen et al. (1994)
YrS2199	presumably *Yr5*	2BL	S2199	**SDR**	Fang et al. (2008)
YrSN104		1BS	Line Shaannong 104	**SDR**	Asad et al. (2012)
YrSP		2B	Spadlings Prolific	**RS, SDR**	McIntosh et al. (1995); Gosal (2000)
YrSte		2B	Stephens	**RS, SDR**	Chen et al. (1996)
YrSte2		3B	Stephens	**RS, SDR**	Chen et al. (1996)
YrTr1		6D	Tres	**RS, SDR**	Chen and Line (1995)
YrTr2		3A	Tres	**RS, SDR**	Chen et al. (1995b)
YrTye		6D	Tyee	**RS, SDR**	Chen et al. (1995b)
YrV23	presumably *Yr3a*	2B	Vilmorin23	**RS, SDR**	Chen et al. (1996); Wang et al. (2006)
YrYam		4B	Yamhill	**RS, SDR**	Chen et al. (1994)
YrZH84		7BL	Annong 7959, Zhoumai 11	**SDR**	Li et al. (2006b)

5 WYR RESISTANCE: THE GENETIC APPROACH

5.1 Conventional breeding and marker assisted selection

As the most economic and environmentally friendly strategy for disease control is genetic resistance, breeding programs work to create cultivars with resistance to Pst. The classical approach includes crosses between a resistant and a susceptible cultivar, with backcrosses and phenotypic selection at several steps in order to successfully fix Pst resistance. However, the process is long and the phenotyping is time and resource-intensive particularly when selecting for partial APR, as the plants have to be evaluated at the adult stage and quantitative resistance is more difficult to evaluate (low heritability). With this approach, it is also challenging to select for recessive resistance. For these reasons, breeding programs have concentrated their effort on major resistance genes during the past century. Furthermore, in order to obtain durable resistance, pyramiding genes is recommended, but in absence of suitable races of the pathogens to test for the presence of each individual gene or the resources to adopt this type of approach, conventional breeding does not actively implement pyramiding strategies.

In the past two decades, progress in cereal genomics has resulted in the rapid development of molecular markers, a better understanding of the genome and the function of genes (Gupta and Varshney, 2004). Those progresses allow novel approaches in breeding such as marker assisted selection and genetic engineering.

Marker assisted selection (MAS) uses molecular markers linked with genes of interest to select for specific traits, eliminating the need for direct screening (Koebner and Summers, 2003). The main prerequisite to MAS is a marker-trait association (MTA) study that identifies markers of interest. For MAS purpose, the ideal marker system should present the following qualities: 1) highly polymorphic, 2) high throughput to obtain good genome coverage, 3) reproducible, 4) cost-efficient for screening of large populations (Falconer and Mackay, 1996; Mohan et al., 1997). Besides, the marker loci must be closely linked to the gene of interest and be diagnostic for a wide range of cultivars to be of use in plant breeding programs (Gupta et al., 1999).

MAS presents several advantages compared to conventional breeding for disease resistance. First, it eliminates the need for disease screening tests. Secondly, selection is sped up as plant can be evaluated at seedling stage, even for APR. Finally, subject only to the limits of population size, it allows selection for multiple resistance genes, a promising approach to produce durably resistant cultivars.

MAS is now commonplace in wheat breeding programs all over the world, however, until recently, efforts have been concentrate on monogenic traits.

5.2 Marker–trait association

WYR resistances fall into two categories: qualitative resistances which are controlled by major R-genes; and quantitative resistances. Quantitative resistances, like many agronomically important traits such as yield, yield components and flowering time, are controlled by multiple genetic loci referred to as quantitative trait loci (QTLs).

The association of a trait with specific molecular markers (gene tagging) allows the description and localisation of gene or QTLs of interest within the genome. Besides, it constitutes the starting point toward map-based cloning and the understanding of gene function. In addition, MTA studies provide an insight on the inheritance of traits: polygenic versus monogenic, recessive versus dominance, additive effect and epistasis.

Before the development of molecular markers, the genetic basis of qualitative and quantitative traits was studied through the statistical analysis of phenotypic variation within segregating populations. Such studies were useful to give an indication of the number of genes underlying traits, though they did not reveal their location. In the late 1980s, the development of molecular markers improved the situation. DNA markers facilitated the construction of linkage maps and the identification of chromosomal regions containing genes and QTL of interest.

QTL mapping reveals significant marker loci associated with the trait of interest by looking at the correlation between marker allelic variation and the phenotype in a segregating bi-parental population. The method is based on the presence of linkage between the marker locus and the causative locus due to limited recombination between them. Many statistical methods have been developed for QTL mapping (reviewed by Hackett, 2002) among them figure three widely use methods: single marker analysis, interval mapping and multiple QTL mapping. The most commonly used populations in QTL mapping for cereal are doubled haploid, F2 and backcross populations (Paterson, 1996). The success of QTL mapping greatly depends of the population used (type and size), the marker system (polymorphism and density of markers) and the trait (heritability and complexity). In the past two decades, QTL mapping using bi-parental mapping populations has been widely employed to indentify QTLs. Salvi and Tuberosa (2005) reported that approximately 150 research papers reporting original QTL data were published yearly between 2000 and 2004.

In recent years, an alternative approach known as association mapping gained in importance (Gupta et al., 2010). Association mapping (AM) is a mapping approach based on linkage disequilibrium (LD) within a set of genotypes such as germplasm accessions and cultivated varieties. LD is defined as a non-random association of alleles at different loci that may or may not be on the same chromosome (Abdurakhmonov and Abdukarimov, 2008). In other words, LD describes the presence of combinations of alleles/markers in a population at

a higher or lower frequency than would be expected in a randomly mating population. In AM, QTLs are identified by statistical association between allelic variants of a marker and the mean of the analysed trait. The main advantage of this method lies in the possibility of identifying markers tightly linked to a gene of interest without having to produce a costly mapping population. Positive and negative aspects of the method are discussed further in section 6.2.

Finally, bulk segregant analysis (BSA) is a shortcut to QTL mapping, which can be used to identify markers associated with trait of interest located in specific chromosomal regions (Michelmore et al., 1991). As markers are screened on bulks of DNA from contrasting individuals, it is not necessary to build a complete linkage map or to genotyping an entire segregating population. Markers polymorphic between the bulks directly tag the genomic regions of interest.

5.3 Molecular markers

Accurate tagging of quantitative trait loci (QTLs) and/or genes of interest relies on DNA based molecular markers. In contrast to traditional markers (e.g. morphological and biochemical), molecular markers are not influenced by the environment and are independent of the harvested tissue/organ or the stage of development of the plant. Also, the number of potential DNA based markers is almost unlimited, as recently illustrated by the development of Diversity Array Technology (DArT) markers for wheat (Akbari et al., 2006) and single nucleotide polymorphism (SNP) discovery based on Next-Generation Sequencing technologies (Allen et al., 2011; Poland et al., 2012; Trick et al., 2012).

A molecular marker aims to reveal a variable feature of a DNA fragment that has two or more conformations (alleles), at least one of which can be reliably identified. Where only one variant is unambiguously identified, the assay is a dominant assay, where all variants present are unambiguously scored, the assay is co-dominant.

There are many types of molecular markers, of which restriction fragment length polymorphisms (RFLPs) were the first to be developed (Botstein et al., 1980). The advent of the Polymerase Chain Reaction (PCR) (Mullis et al., 1986) gave rise to new marker systems including random amplification of polymorphic DNAs (RAPDs) (Williams et al., 1990), microsatellites or Single Sequence Repeats (SSRs) (Litt and Luty, 1989), Amplified Fragment Length Polymorphisms (AFLPs) (Vos et al., 1995) and most recently Single Nucleotide Polymorphisms (SNPs). Additionally, PCR-based markers have been derived from RAPD, RFLP and AFLP marker system; they are referred as Sequence Characterized Amplified Regions (SCARS) (Paran and Michelmore, 1993), Sequenced Tagged Sites (STS)

(Olson et al., 1989) and Cleaved Amplified Polymorphic Sequences (CAPS) (Konieczny and Ausubel, 1993).

The different marker systems used in plants have been intensively reviewed (Gupta et al., 1999; Koebner et al., 2001; Semagn et al., 2006a), Table I-5 summarises the properties of the major marker systems.

The recent molecular technique refers to Diversity array technology (DArT) (Jaccoud et al., 2001), was chosen for the present study. DArT genotyping is provided as a commercial service by Diversity Arrays Technology Pty Ltd, Canberra, Australia (http://www.diversityarrays.com/index.html) and has been applied to a wide range of plant species and a few animal species. For wheat, a high density array containing more than 5000 markers is available; the latest array version v.3 is enriched with D genome markers. The genotyping service for wheat is provided by a subsidiary company named Triticarte Pty Ltd (http://www.triticarte.com.au/). DArT is essentially a microarray hybridization-based technique for AFLP. DArT markers are dominant markers, where polymorphisms are based on SNPs, insertions and deletions (INDELs) at restriction sites or large INDELs between restriction sites. The methodology consists of a genome complexity reduction, including fluorescent dye labelling, followed by hybridization on a microarray chip. The chip contains a large number of immobilised cloned AFLP fragments from distinct wheat genotypes and closely related *Triticea* species. Hybridisation of a test DNA sample to an individual feature (clone), measured by above background fluorescence, is scored as '1' and absence of significant fluorescence as '0'.

To specifically target R-genes, an additional molecular technique known as resistance gene analog polymorphism (RGAP) is available. Specific genomic DNA sequences are amplified using degenerate primers based on conserved sequence discovered in cloned R-genes from diverse species, such as leucine-rich repeats (LRR) and nucleotide-binding sites (NBS). Resistance gene analogs (RGAs) are highly non-randomly distributed in the genome and often map to clusters which harbour major resistance genes or QTLs (Hulbert et al., 2001b). Thus, they may provide candidate genes or useful markers for MAS. They also provide information about the organization and evolution of resistance genes and RGAs. Using the RGAP technique, Feuillet et al. (1997) isolated a candidate gene *Lr10* for leaf rust resistance. More recently the RGAP technique has been used successfully to developed molecular markers for WYR resistance genes *Yr5* (Chen et al., 2003a; Yan et al., 2003), *Yr9* (Shi et al., 2001), *Yr21* (Pahalawatta and Chen, 2005), *Yr26* (Wen et al., 2008),*Yr39* and *YrAlp* (Lin and Chen, 2007a). On the same principle, Van der Linden et al. (2004) developed a new technique named NBS profiling to generate polymorphic markers based on conserved NBS domain of R-genes using an adapter primer matching a restriction site and a degenerate primer targeting a NBS domain in PCR reaction.

Table I-5 : Major DNA based marker systems and their properties.

Adapted from Gupta et al. (1999); Rafalski et al. (1996); Semagn et al. (2006a); Jaccoud et al. (2001)

Marker system	Principle	Polymorphism Type	PCR based	Inheritance	Sequence information required?	Quantity of DNA required	Polymorphism level	Development cost	Cost	Throughput	Reliability
RFLP	Digestion of genomic DNA by endonuclease followed by electrophoresis and hybridization of a DNA probe (southern blotting)	SNPs, I NDELs	No	Co-dominant	No	High 5-10µg	Medium	Medium	Medium	Low	High
RAPD	PCR amplification of random fragments of genomic DNA with a single primer of arbitrary nucleotide sequence	SNPs, INDELs	Yes	Dominant	No	Low 10-25ng	High	Low	Low	Medium	Low
AFLP	Selective PCR amplification of restriction fragments generated by specific restriction enzymes and ligation of complementary adaptors	SNPs, INDELs	Yes	Dominant	No	Medium 1-2µg	Very high	Low	Medium	Medium	Medium
SSR	PCR amplification of simple sequence repeats of 1 to 6 base pairs in length (named microsatellites), using primers flanking the microsatellite region	Change in Number of repeats	Yes	Co-dominant	Yes	Low 10-25ng	Very high	High	Low	High	High
SNP	Identification of a single base change within a DNA sequence. SNPs can be detected via different methods, including : competitive allele specific hybridization or amplification, specific primer extension, oligonucleotide ligation, invasive cleavage	SNP	Yes	Co-dominant	Yes	Low 5-25ng	High	High	Low	High	High
SCAR	PCR amplification using a pair of specific primers derived from sequencing of RAPD markers	SNPs, INDELs	Yes	(Co-) Dominant	Yes	Low 10-50ng	-	-	-	-	-
STS	PCR amplification of a short unique sequence using a pair of specific primers derived from sequencing either an RFLP probe or an AFLP fragments	SNPs, INDELs	Yes	(Co-) Dominant	Yes	Low 10-50ng	-	-	-	-	-
CAPS	PCR amplification of a target DNA, followed by digestion with restriction enzymes which cleaves the fragment at a polymorphic site	SNPs at restriction sites	Yes	Co-dominant	Yes	Low 10-50ng	-	-	-	-	-
DArT	Microarray hybridation based technique of cloned AFLP fragments	SNPs, INDELs	Yes	Dominant	No	Low 1-5ng	High	-	Low	High	High

5.4 Mapping of WYR resistance genes and QTLs

Mapping resistance genes constitutes one of the most active areas of research on WYR. A variety of marker systems including RADP, SSR, AFLP, RAGP have been used to map *Yr* genes and QTLs for WYR resistance. An extensive list of mapping information available for *Yr* genes is presented in Appendix 1. Some of the markers closely linked to the resistance genes have been shown to be useful for marker-assisted selection. For instance, *Yr15, Yr17, Yr36* were introgressed using MAS into adapted lines and popular cultivars in the USA (Gupta et al., 2010; Hospital, 2009; Sorrells, 2007). Following physical mapping, three WYR resistance genes have been cloned: *Yr10*, a race specific *R*-gene coding for a NBS-LRR protein (Laroche et al., 2005),*Yr18/Lr34* and *Yr36* involved in non race specific resistances coding respectively for a ABC transporter (Krattinger et al., 2009) and a protein kinase with a START domain (Fu et al., 2009). Based on the gene sequence and functional polymorphism, allele-specific diagnostic markers were developed for *Yr18* and *Yr36* (Fu et al., 2009; Lagudah et al., 2009).

In addition to markers linked to *Yr* genes, an increasing number of QTLs have been described, especially for partial and durable resistance to yellow rust (Wellings et al., 2012). An extensive list of WYR resistance QTLs is available in Appendix 2.

6 ASSOCIATION MAPPING

6.1 Introduction to association mapping

Association mapping (AM), also known as linkage disequilibrium (LD) mapping or association analysis is a population based method used to detect and map QTLs based on the strength of the correlation between a trait and a marker. The method takes advantage of historical LD to identify trait-marker relationships within a panel of individuals. When performed by scanning markers across the entire genome for statistical significant associations between a set of molecular markers and a specific phenotype, this approach is referred to as a genome-wide association (GWA) study. AM studies can also be carried out on restricted genomic regions using a candidate gene approach, an approach which is particularly relevant when candidate genes can be identified and genome wide LD is limited (Hall et al., 2010). Following a GWA study, which identifies regions of interest within the genome, fine mapping studies can be undertaken by increasing the density of markers in the identified candidate genomic regions. This approach potentially allows the identification of the causative polymorphism and candidate gene.

AM was pioneered in human disease genetics and the results of thousands of GWA studies performed on the human genome are collated in a National Institute of Health

database (Hindorff et al., 2009). The statistical approaches to population association studies developed for human genetics have been reviewed in detail by Balding (2006). More recently, methods related to association mapping have been adapted and applied to higher plants (Gupta et al., 2005; Hall et al., 2010; Mackay and Powell, 2007; Sorrells and Yu, 2009; Soto-Cerda and Cloutier, 2012). Supported by the rapid development of molecular markers, the availability of high density maps and in some case sequenced genomes, numerous studies related to LD evaluation, genome wide diversity and AM have been published in recent years for major crops e.g. rice, maize and wheat (Soto-Cerda and Cloutier, 2012; Zhu et al., 2008).

In wheat, association analysis has been used to identify markers linked with numbers of traits including milling/flour quality (Bordes et al., 2011; Breseghello and Sorrells, 2006b), high molecular weight glutenin (Ravel et al., 2006), alpha amylase activity (Emebiri et al., 2010), grain yield, grain weight (Mir et al., 2012; Reif et al., 2011), flowering time (Rousset et al., 2011) ,earliness (Le Gouis et al., 2012), sprouting resistance (Kulwal et al., 2012), seed longevity (Rehman et al., 2012), agronomic traits (Dodig et al., 2012; Neumann et al., 2011; Yao et al., 2009), many disease resistances (e.g. *Stagonospora nodorum* blotch, Fusarium head blight , stem rust, leaf rust, yellow rust, powdery mildew) (Crossa et al., 2007; Gurung et al., 2011; Miedaner et al., 2010; Tommasini et al., 2007; Yu et al., 2012a). So far, AM studies in wheat have provided a long list of MTAs, some of them were validated by comparing to already known QTLs. However, no validation for the new putative QTLs and no causative polymorphism have been reported yet. The earliest wheat AM studies were based on SSR and AFLP. In recent years, high throughput genotyping techniques (e.g. DArT and array based SNPs) appear to be the marker systems of choice for AM, as it provides a cost efficient way to obtain genome wide coverage.

6.2 Association mapping versus QTL mapping in bi-parental population

AM and QTL mapping are both methods currently and widely applied to identify MTA in plants. In this chapter the advantages and limitations of AM compared to QTL mapping are presented, starting with its benefits.

First, with AM, the identification of QTLs does not require the costly and time consuming production of large experimental populations, as association mapping can be apply directly to a panel of individuals such as breeding populations, subsets of germplasm collections, cultivated varieties, landraces or samples from natural populations.

Secondly, the higher level of recombination within AM populations has the potential to give higher genetic resolution comparing to classical bi-parental population. The large

number of recombination is due to many meiotic events that occurred throughout the germplasm development history. Furthermore, AM populations present a wider genetic background and give access to broader genetic variation, meaning that many alleles can be evaluated simultaneously for one locus. The high resolution and the broad genetic diversity investigated via AM are most likely to provide effective gene tagging markers for MAS (Yu and Buckler, 2006).

Thirdly, as AM is based on existing collections of individuals, it is possible to take advantage of historical phenotype data previously collected from the different genotypes, as illustrated by the mining of historical multi-environment trials from CIMMYT to identify markers linked to disease resistance in wheat germplasm (Crossa et al., 2007). Finally, as many valuable traits are likely to segregate within the same AM panel, it is possible to investigate many traits through AM using the same panel of accession and the same genotype data, making the approach particularly cost effective.

On the other hand, AM presents some disadvantages comparing to QTL mapping. Stratification or structuration of the genetic diversity within an AM population leads to spurious associations (Flint-Garcia et al., 2005; Pritchard et al., 2000a). To avoid false discovery, it is necessary to take into account the sub-population structure and individual relatedness (kinship) when testing for MTA. Many AM methods have been developed to correct for population structure, some of which are presented in the section 6.3.5 of this chapter. Nevertheless, corrective models have to be carefully applied as inappropriate use can lead to overcorrection and false negative results. In addition, the necessity to correct for population structure also limits the possibility to detect traits strongly associated with population structure, as differences between sub-populations are disregarded. The difficulties arise particularly for traits under local adaptation like flowering time. In order to study such traits, it is advisable to limit AM to sub-populations.

The detection of MTA via AM is influenced by allele frequency distribution within a population thus causative rare allele cannot be identified (Myles et al., 2009). In a biparental population, the allele frequency is normally distributed around a mean of 0.5, whereas in an AM population, allele frequencies can vary from 0 to 1, and power to detect association drops away when the Minimum Allele Frequency (MAF) at a locus drops below a certain level, making it necessary to exclude low MAF markers from the analysis. Also, AM does not perform as well as QTL mapping to identify QTLs explaining small phenotypic variation (low heritability) as the signal is scattered across numerous QTLs and alleles. Moreover, the study of complex traits such as yield or drought stress via AM is made difficult as they are greatly influenced by phenological characters segregating as well in the population (i.e.

maturity, plant height). As a consequence, true effects tend to be confounded with phenological effects.

Finally, due to the generally higher rate of LD decay, the AM approach requires a higher marker density than QTL mapping but yet does not allow the construction of genetic map which complicates the situation in crop species like wheat where high density consensus maps are not available.

To overcome the limitations of AM, methodologies using multiparent intercross population have been developed recently. Nested association mapping (NAM) populations have been established in maize (Yu et al., 2008) and *Arabidopsis* (Brachi et al., 2010). Multiparent advanced generation intercross (MAGIC) population were proposed by Cavanagh et al. (2008) and several were developed in wheat (Huang et al., 2012) and *Arabidopsis* (Kover et al., 2009). Those approaches combined classical QTL mapping and AM, and are likely to reduce spurious association caused by population structure and allow the detection of rare alleles while maintaining a high mapping resolution and broad genetic diversity.

6.3 Methods in association mapping in plants

The major steps in an AM study: choice of germplasm, trait measurement, estimation of LD, evaluation of population structure and statistical analysis are illustrated in Figure I-7.

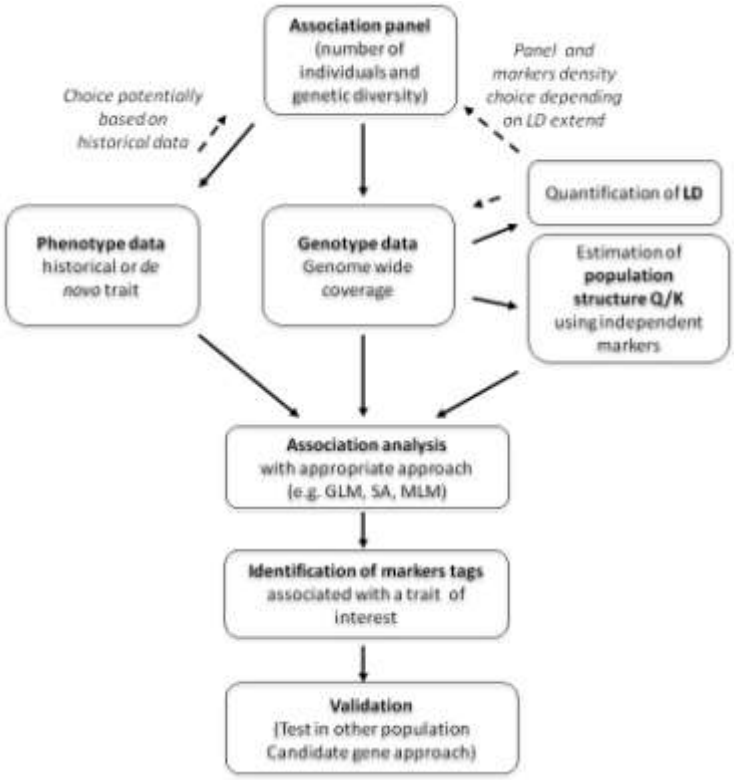

Figure I-7: Scheme of steps involved in association mapping studies.

Adapted from Abdurakhmonov and Abdukarimov (2008); Flint-Garcia et al. (2005); Salvi and Tuberosa (2005).

6.3.1 Choice of germplasm

The choice of germplasm for association analysis is critical for the success of AM and highly dependent on the purpose of the study; which trait is going to be studied and what is the objective, for instance the investigator might want to identify novel alleles from exotic germplasm or identify markers for a useful trait within elite lines. However there is little guidance available regarding criteria that could be used to assemble germplasm panels for plants. Generally, association populations should present phenotypic variation for the trait studied as well as overall genetic diversity. Towards this end, preliminary germplasm evaluation including the use of historical phenotype data is often conducted prior to establishing a new association panel. Breseghello and Sorrells (2006a) distinguished three main categories of populations suitable for AM (germplasm bank collections, elite breeding materials and synthetic populations) and discuss the suitability of each for AM considering their level of LD, structuration, genetic diversity and their suitability for studying various traits.

Population size is another critical point for AM success in detecting and estimating gene effects. Long and Langley (1999) demonstrate through simulations than increasing

population has a more positive effect on power compared with increasing the number of markers.

6.3.2 Genotype data

Any molecular markers can be used in AM studies. The markers systems available in crops are described in Chapter I-5.3. Nevertheless, high-throughput marker systems such as DArT and SNPs providing a high density genome wide coverage are favoured for AM. Lower density marker types like SSRs, which are highly polymorphic, are also of great use to estimate population structure and pairwise relatedness among individuals.

The genotyping approach depends mainly on the availability of molecular markers for the species concerned. The number of markers required in AM is highly dependent on the population studied and the extent of LD decay, as well as the resolution sought.

Another important point to take into account while looking at the genotyping approach is the genotype error rate, as even low error rates (around 3% or less) can have dramatic consequences for the accuracy of estimates of LD and hence on AM (Akey et al., 2001).

In wheat AM, until recently, marker density was the limiting factor, wheat had the fewest molecular markers available among major crops. The first LD studies were based on AFLP and SSR markers (Chao et al., 2007; Maccaferri et al., 2005; Somers et al., 2007). Following the development of DArT markers, several GWA studies were reported. At the same time, some wheat AM studies avoided the issue of lack of genome wide markers by focusing on small genomic regions (Breseghello and Sorrells, 2006b; Ravel et al., 2006; Tommasini et al., 2007). Recently, several thousand SNPs markers have been identified by the wheat research community and during 2012, a 90K SNP Illumina Infinium assay was developed for wheat. The first 90K Infinium SNP genotype data are expected to be made public at the end of 2012 and part of this SNP cohort has been mapped, the resource will undoubtedly boost future AM studies.

6.3.3 Estimation of LD

Once an association panel has been assembled, AM studies always start with the determination of LD decay within an AM population. LD can be viewed as the correlation between polymorphisms in a population. The degree of LD present in the population determines the resolution of the analysis and influences the possibility to identify markers closely linked to causal locus. Where LD is strong in the region of the causal locus, the chance to reveal a marker-trait association (MTA) with a manageable number of markers is high, but the resolution may be low. Where LD is weak, the potential to detect MTA with the

equivalent number of markers will be lower, but the potential for high resolution is greater. Depending of the LD observed within the population, the investigator will be able to adapt the density of markers needed and the accession selected.

The structure of LD is highly dependent on the species, the population and even the locus considered (Caldwell et al., 2006; Chao et al., 2010; Flint-Garcia et al., 2003; Rafalski, 2002). The difference in LD decay rates between species is often related to the breeding system; in cross pollinating species, LD tends to decay faster than in self pollinating species (Myles et al., 2009).

LD is commonly calculated between each pair of polymorphic sites, using D' (standardized disequilibrium coefficient) or r^2 (correlation coefficient between the alleles of two loci). Both parameters vary from 0 (absence of LD) to 1 (complete LD). To visualise the extent of LD, LD measurements can be plotted as a function of genetic (in centiMorgans, cM) or physical (in base pairs, bp) distance and a curve fitted to estimate LD decay. Alternatively, some software programs like TASSEL (Bradbury et al., 2007) display an LD heatmap of all pairwise LD measurements which allows examination of local variation in LD patterns.

6.3.4 Trait evaluation

Once the association panel has been defined, a potentially infinite number of traits can be investigated. However, the better the heritability of the trait, the easier it will be to detect MTAs. The complexity of the trait, the accuracy of measurement and the importance of genotype by environment interaction will influence heritability and hence the success of MTA detection.

AM of complex traits such as yield and insect resistance can result in numerous associations with low statistical significance. To improve the resolution in genotype-phenotype association, one approach suggested is to dissect the complex phenotype into several component traits (Hammer et al., 2006; Kloth et al., 2012). Mapping these components traits will result in fewer genotype-phenotype associations with larger statistical significance. This approach is illustrated by studies on yield component in rice (Li et al., 2011). Taking the example of WYR, instead of scoring the overall disease severity, we can imagine separately targeting different mechanisms of disease resistance by scoring different components of the infection for instance the haustoria formation, the presence of HR and the density of pustules.

Accuracy of phenotype scoring is another important issue. Replication of individual accession measurements within a site is usually needed to increase precision in phenotypic measurement and allow accounting for environmentally induced noise and measurement

error. Additionally, replication across multiple environments can provide important insight into the stability of the phenotype itself, the robustness of the positive association across environments and the importance of genotype-environment interactions. Replicated data can then be combined to produce an estimate of the phenotype for each accession that can be used in the AM analysis. Estimates such as the best linear unbiased predictor (BLUP) or best linear unbiased estimator (BLUE) are less influenced by environment and measurement errors.

6.3.5 Population structure and statistical models

Population structure within an association panel is another serious issue as it often lead to false positive MTA (Myles et al., 2009; Pritchard et al., 2000a; Zhao et al., 2007). The problem arises because any phenotypic trait that is also correlated with the underlying population structure at neutral loci will show an inflated number of positive associations (Hall et al., 2010). The term structure within an association panel refers to the possible presence of hidden sub-groups (stratification at a macro-level) and family relatedness within sub-group also named kinship (micro-level). Complex patterns of population structure are engendered by complex breeding history in crops, gene flow from wild species, and artificial and natural selection.

A common practice to correct for spurious effects of population structure is to use unlinked markers throughout the genome to estimate the genetic variation among accessions and account for population structure within the association tests. Many methods have been developed to correct for genetic relatedness, three of which are commonly applied to crop plant AM studies: general linear model (GLM) method integrating a structure matrix Q based either on co-ancestry coefficients (method also referred as structured association) or on principal components (also referred as PCA model) (Price et al., 2006), and a mixed linear model (MLM) method integrating a kinship matrix K.

The first model, structured association, involves using a program STRUCTURE (Falush et al., 2003; Pritchard et al., 2000a) to identify ancestral sub-populations from which the individuals in association panel originated. This approach is based on an island model where each sub-population is assumed to be in Hardy Weinberg equilibrium. The output from STRUCTURE is a matrix Q of sub-population assignment for each individual (co-ancestry coefficient). The matrix Q can then be used as a covariate in a general linear regression model (GLM) in order to indentify significant MTAs. One of the drawbacks of this method is the intensive computation required to obtain the Q matrix using STRUCTURE.

Alternatively, the Q matrix can be obtained from a principal component analysis (PCA) across markers with a genome wide distribution. PCA summarizes the variation observed across all markers into a small number of variables, the eigenvectors. A limited number of eigenvectors are included in a matrix Q and used to represent the underlying population structure in the association tests. PCA is less computationally demanding and the models obtained have been shown to perform similarly or better than STRUCTURE model (Price et al., 2006; Zhao et al., 2007). However neither PCA nor STRUCTURE models account for close kinship potentially present between accessions, which is critical when accessions are being sampled within a highly intercrossed breeding genepool.

The MLM, outlined by Yu et al. (2006), is the most recent approach and the most promising as it allows the integration of both types of population structure: the population stratification revealed by a Q matrix and the relative kinship of individual captured by a kinship matrix (K). Both matrixes are then fitted into a mixed linear regression model, Q is fitted as fixed effect and K as random effect. The kinship matrix can be estimated from pedigree when available or random genetic markers; however marker-based kinship coefficients are proven to be more accurate than pedigree based estimated (Myles et al., 2009). The marker-based kinship matrix captures the relatedness between each possible pair of individuals. The strength of the MLM lies in its flexibility, whereby it performs well under many types of population structure. Many studies in plants show a significant decrease in false positives and false negatives when correcting for pairwise relatedness over a Q matrix alone (Kang et al., 2008; Yu et al., 2006; Zhao et al., 2007). Additionally, MLM can be adapted to the population studied by removing either the Q or the K matrix from model.

6.3.6 Replication and validation

Looking at the increasing number of significant MTAs published, it is becoming crucial to validate those findings to separate the true from the false positives and provide reliable estimate of allelic effects. The most straight forward way to verify a putative association is to evaluate the candidate polymorphism and replicate the result in a different population either bi-parental or independent AM population. Other validation methods are available when a candidate gene has been identified, including the identification of the causative polymorphism within the genomic sequence and the confirmation of biological function through transgenic misexpression studies, Targeting Induced Local Lesions in Genomes (TILLING) or other molecular techniques.

7 WHEAT YELLOW RUST IN UNITED KINGDOM AND UKCPVS

In the UK, WYR occurs more often in the east of the country and in coastal areas; Lincolnshire, Norfolk, East Midlands and parts of Yorkshire are acknowledged hotspots. Severe epidemics are usually associated with very susceptible varieties, mild winters and cool moist summers. Yield losses of 40-50% have often been recorded in susceptible varieties (Priestley and Bayles, 1988).The continuous threat posed by the disease is regularly brought into focus when new isolates are found to overcome the major resistance genes present in highly cultivated wheat varieties, for instance Brigadier in 1994 (Bayles et al., 2000), Robigus in 2006, Solstice/Oakley in 2009, Warrior in 2011.

The Figure I-8 illustrates the cyclical nature of YR epidemics in the UK and trend towards increasing reliance on fungicide use.

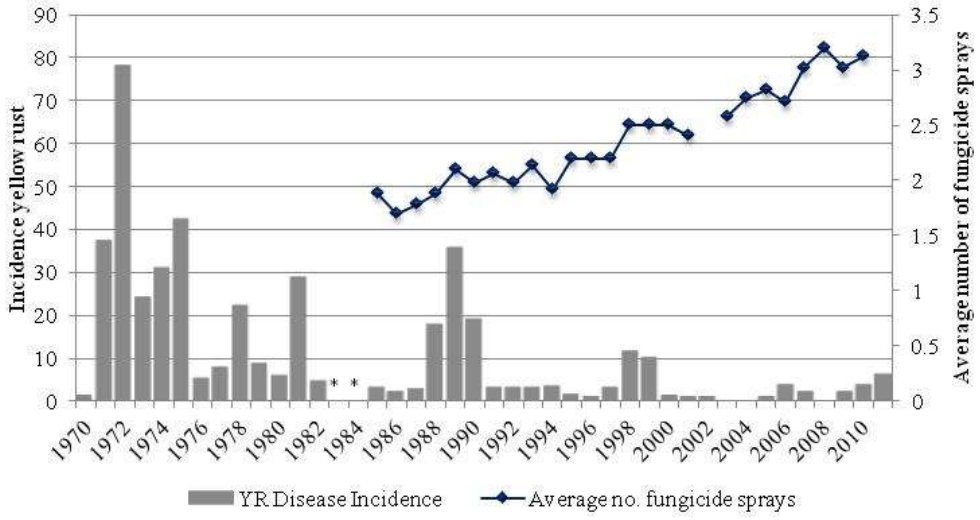

Figure I-8 : Incidence of yellow rust of wheat in the UK and fungicide sprays associated

*no data were available for 1983 and 1984 for yellow rust severity
(Source data: Defra winter wheat disease surveys on www.cropmonitor.co.uk)

Pst virulence monitoring in the UK has been carried out continuously since 1963 by the UK Cereal Pathogen Virulence Survey (UKCPVS) by collecting and pathotyping field isolates from disease observation plots, untreated field trials and disease samples sent to NIAB from growers. Representative isolates are used in inoculated field trials to measure the Adult Plant Resistance (APR) of candidate Recommended List (RL) cultivars.

The UKCPVS published the result of the survey on an annual report describing the virulence of Pst isolates collected within the country; it also offers a guideline for varieties diversification in order to minimize the impact of epidemic within a farm.

In its 40 years of existence, the survey has accumulated detailed resistance data on every major variety of wheat grown in the UK at both seedling and adult plant stages, recorded the emergence of previously unseen virulence on newly introduced specific resistances. UKCPVS data provide also many examples of varieties which display contrasting levels of partial resistance once the specific element of their resistance have been matched by a virulent pathogen isolate.

8 AIMS, OBJECTIVES AND POTENTIAL APPLICATIONS

Despite the importance of wheat yellow rust in the UK, many of the race-specific resistances used by breeders in UK varieties remain unmapped and the relative contribution of race-specific and partial genetic resistance to the overall performance of a variety in the field is still poorly understood.

The first objective was to utilise the unique historic data resource represented by historic UKCPVS APR and seedling test data to analyse the genetic architecture of resistance to yellow rust using an association genetics approach. In pursuit of this goal, a comprehensive panel of contemporary and historic UK wheat varieties which have been tested for phenotypic reaction to yellow rust isolates by the UKCPVS and during the National List and Recommended List testing will be assembled and genotyped using DArT markers.

Based on historical phenotype, genome wide association analysis will be carried out with the aim of rapidly revealing the diversity of resistance present within the UK elite germplasm. This meta-analysis of UKCPVS and related data has never been carried out before, and represents an exciting opportunity to add value to existing resources.

Beside the investigation of historical data, in the context of the development of a new WYR races within the UK with a wide range of virulence, the second major objective of this study was to evaluate the association panel using the more recent isolates. This unusually wide screening of UK wheat varieties will show: (1) remaining sources of resistance to new widely virulent isolates within the UK elite genepool; (2) allow deductions to be made about the extended virulence profile of these isolates as well as improved/updated gene postulations regarding older varieties; (3) provide the means to conduct a genome scan for resistance loci effective against the new isolates.

A second series of association analyses will be conducted using the *de novo* phenotype to identify resistances QTL effective against current WYR races.

To validate some of the association hits, specific WYR races used to screen the association panel will be used to screen a doubled haploid populations (Avalon x Cadenza) which segregate for key resistances deployed in recent years in the UK and the results of mapping resistance in biparental population and association panel compared.

CHAPTER II. YR ASSOCIATION PANEL

1 INTRODUCTION

The first step of the project was to assemble a panel of winter wheat (*Triticum aestivum L.*) varieties; mainly UK elite germplasm, that will be use in the association mapping studies. A variety panel, henceforth referred to as the wheat "YR panel", was assembled to enable mining of historic adult plant resistance data for rapid association mapping of multiple WYR resistance factors. Additionally, with the aim of investigating the source of genetic structure (e.g. historical stratification and kinship) within the YR panel, information such as breeder, country of origin, pedigree were gathered for each variety.

The aims of this chapter are to present the wheat YR panel and describe the information collected on the different wheat varieties in regard to their origin and pedigree. Finally the characteristics of the YR panel will be discussed with respect to association mapping studies.

2 YR ASSOCIATION PANEL

2.1 Choice of varieties studied

Since a central aim of the project was to utilise historic data on adult plant resistance to specific isolates, the main criterion for selection of a variety to join the YR panel was the existence of at least one year of adult plant resistance data as part of evaluations carried out under the auspices of the National and Recommended List trials and UKCPVS. Effectively, this means varieties entered as candidates which reached at least the second full year of NL testing, when specific isolate inoculations are first carried out. Based on historic data available at NIAB, 310 varieties for which well-provenanced seeds could be sourced were selected to comprise the core wheat YR panel. The permission of the breeder of each variety was obtained to access, where appropriate, authenticated seed stocks held by the DUS section at NIAB, as well as historic YR data. A full description of the historic data collected is given as part of Chapter III.

The 310 wheat varieties selected have been tested in the field against at least one single UK Yellow Rust isolate between 1990 and 2009.

An additional 17 winter varieties were included in the YR panel based on their importance in the pedigrees of elite wheat germplasm and the availability of seeds and genotyping information from other projects.

The full wheat YR panel is therefore composed of 327 wheat varieties, mainly elite UK winter wheat varieties, but other European countries are also represented (see Table II-3 at the end of the chapter).

2.2 Seed collection and multiplication

From the 327 cultivars selected, we obtained the seeds from five main collections (see Table II-3 at the end of the chapter) cited in order of preferential sources:

- **DUS wheat reference collection** at NIAB, Cambridge, which contains currently registered or protected cultivars, candidates submitted for Distinctness, Uniformity and Stability test.
- **BSPB collection** held at the John Innes Centre (JIC), Norwich
- **GEDIFLUX winter wheat germplasm collection** conserved by Simon Orford, JIC, Norwich (GEDIFLUX was a project supported by the European Commission under the 5th Framework Programme for Research and Technological Development European, the summary of the results can be found on http://ec.europa.eu/research/)
- **BBSRC Small Grain Cereals Collection**, JIC, Norwich
- **Triticeae Genome (TG) winter wheat collection** conserved by Nick Gosman at NIAB, Cambridge, (TG project is supported by the European Commission under the 7th Framework Programme for Research and Technological Development http://www.triticeaegenome.eu).

Authorization to obtain seeds for lines not registered in the UK national list or any European national lists was obtained from the breeders prior requesting the seeds from the different collections.

To maximise traceability and authenticity of our project materials, we preferred seeds from DUS reference and BSPB collection or from controlled multiplication. By carefully choosing the source of our seeds, we hoped to minimise the risk of drift from type over several years of multiplication between the date when plants were phenotyped by the UKCPVS or during the NL RL trials and the stocks from which plants that will be genotyped and de novo phenotypes are taken.

Once all the seeds have been collected, 4 seeds per cultivars were sown in control environment in April 2009, vernalized for 8 weeks and grown for multiplication. Each plant was bagged individually to minimise the risk of cross pollinisation. Homogeneity amongst

the four plants was assessed and any offtype removed. One of the four plants was identified as reference and sampled for genotyping work. The seeds from the reference plant established a single-plant fully selfed lineage and were used for future multiplication. The seeds issued from the three other plants were bulked and used for phenotype experiments over the course of the project.

An additional multiplication was carried out in the glasshouse in 2010 to further increase seed stocks and obtain a first SSD lineage for the varieties for which the first multiplication failed.

2.3 YR panel description

For each variety included in the wheat YR panel, when available, the Application For Protection[1] (AFP) number corresponding to the UK application code for Plant Breeder Right, the breeder name, the alternative names and the NL and RL status were collected in order to identify with certitude each variety and gather the data describing the cultivar known under different names. Particular attention was paid to variety names known to have been used to refer to different varieties at different time – e.g. Choice, Solstice, Warrior, Marksman and Lynx.

For this step, the 'Interra' database developed by Simon Oldfield at NIAB was used. It summarizes information from the UK Plant Varieties and Seeds Gazette published by DEFRA. The EU Common Catalogues of plant varieties (http://europa.eu/) and national authorities for plants breeder right were another source of information, e.g. GEVES in France (http://www.geves.fr) and Bunderssortenant in Germany (*www.bundessortenamt.de).*

A complete list of the varieties with their origin, year of release or first application for plant breeder rights is available in Table II-3 at the end of the chapter

The great majority of cultivars in the YR panel are wheat varieties originating from UK-based breeding programmes (254 lines), but it also includes a minority of European varieties (67 lines) and a few American varieties (4 lines). Apart from five spring wheats, the panel is composed entirely of winter varieties. It is worth noting that some of the winter wheat lines like Cadenza are known to be alternative or facultative wheat which mean they required limited vernalization to initiate flowering (Stelmakh, 1998), facultative wheats often derived from spring wheat and winter wheat crosses (Braun, 1997). Taken together, this set of varieties represents a broad spectrum of breeders, from large seed companies such as

[1] A unique AFP code is issued to each new candidate variety upon submission of an application to obtain plant breeders rights. Although not widely used outside the regulatory framework, the AFP code is very useful as it establishes not only the date of introduction of a variety but avoids the problem of homonymy.

Syngenta seeds to small independent breeders e.g. Mike Pickford. Nevertheless, three main companies/institutes represent 67% of the panel (Table II-1): the Plant Breeding International Cambridge/RAGT, Nickersons/Limagrain and CPB-Twyfords/KWS. The variety set includes cultivars from 1921, e.g. Carstens V, up to 2006 e.g. Warrior (2) (Figure II-1), however the core of the panel covers primarily the past three decades: 1980-1989 (37 lines), 1990-1999 (181 lines) and 2000-2005 (83 lines). Finally, 85% of the varieties in the YR panel were previously or are currently on the UK wheat national list (

Table II-2). Amongst the varieties that have never been on the UK lists, seven were commercialized prior to the coming into existence of the National Listing system in 1973, four were refused UK National Listing and 18 were withdrawn during the testing process. No application for the UK national list was recorded for the remaining varieties; although they may be registered on National Lists in other European countries.

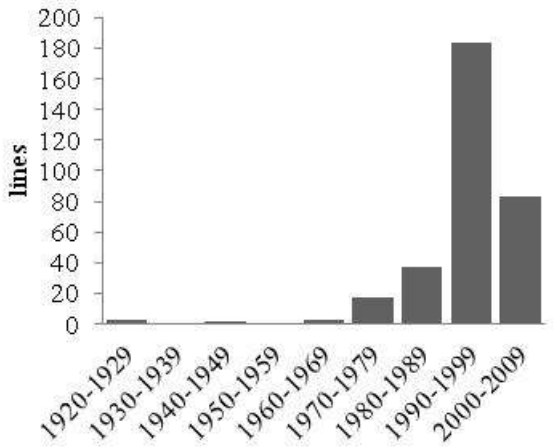

Figure II-1 : Decadal distribution of YR panel varieties

Table II-1 : Main breeders of YR panel varieties

Breeder name	Line
Plant Breeding International Cambridge and associated companies	**Total 99**
Plant Breeding International Cambridge	68
Plant Breeding Institute/NSDO	4
Institute of Plant Science	23
Monsanto	3
Monsanto Technology LCC/Ragt seeds Ltd	1
KWS and associated companies	**Total 50**
KWS UK Ltd /CPB-Twyfords	48
KWS Lochow GmbH	2
Limagrain and associated companies	**Total 73**
Limagrain UK Ltd /Advanta Seeds /Nickerson UK	58
Nickerson International Research	14
Limagrain Netherland BV	1
Syngenta Seeds UK	21
Elsoms Ltd UK	9
SAS Florimond Desprez	5
Other breeders representing less than 5 lines each	74

Table II-2 : Status of YR panel varieties in relation to UK national list

Status	Lines
In 2012 UK national list	57
In previous UK national lists	232
Application for UK national list withdrawn	16
Application for UK national list refused	4
No application for UK national list	4
Unknown	14

2.4 YR panel pedigrees

Pedigrees are a valuable source of information to determine if the YR panel varieties may carry known *Yr* resistance genes, as well as explaining the origin of potential novel resistance genes. For that purpose, pedigree descriptions of the varieties in the YR panel and affiliated lines were collected from the following sources (cited in order of preference):

- **Scottish wheat variety database** (http://wheat.agricrops.org./menu.php) from Science and Advice for Scottish Agriculture (SASA) in Edinburgh.

- **Botanical descriptions of wheat varieties** (http://www.niab.com/pages/id/147/Botanical_Descriptions_of_Varieties) from National Institute of Agricultural Botany in Cambridge.

- **BBSRC wheat collection pedigree report** (http://www.jic.ac.uk/GERMPLAS/bbsrc_ce/Pedw.txt) from the BBSRC Small Grain Cereals Collection in JIC, Norwich.

- **European Wheat Database** (http://genbank.vurv.cz/wheat/pedigree/) from the European Cooperative Programme for Plant Genetic Resources (ECPGR)

- **Germplasm Resources Information Network Database (GRIN),** from the United States Department of Agriculture - Agricultural Research Service. The passports of wheat lines are accessible via the National Small Grains Collection on the following link: http://www.ars.usda.gov/main/docs.htm?docid=2884.

- **Journal articles related to the wheat varieties present in the YR panel**.

Although, all traceable parental lines used and a representation of the complexity of the crossing scheme are given, pedigree information cannot be use to describe accurately the degree of kinship among the varieties within the YR panel as the influence of selection as well as the means of purification i.e., by SSD or doubled haploidy, which will strongly bias the degree of kinship between the parents and the final variety, are unknown or unstated.

General pedigree information for 303 of the 327 varieties composing the YR panel was collected. Some discrepancies in pedigree of a single variety recorded by different sources were noted. In those instances, we recorded the consensus pedigree. The list of pedigrees is available in Table II-3. The pedigree information gathered has to be used with caution as the ad hoc pedigree recording system by which varietal pedigrees come into the public domain (including arbitrary use of codes to protect identity of proprietary material) does not apply any recognised standards that reduces the risk of error or incompleteness and the existence of numerous homonyms (different varieties which carry the same name e.g. Lynx, Warrior) within even recent elite UK wheat germplasm means that there is considerable scope for error.

The pedigree data were formatted for Pedigree Viewer Version: 6.5.2.0. Pedigree viewer is a program developed by University of New England in Australia, allowing drawing and manipulating pedigree diagrams. The latest version can be downloaded at the following link: http://www-personal.une.edu.au/~bkinghor/pedigree.htm.

The diagrams underline the relatedness of varieties in the panel. Key varieties include in the pedigree of modern UK can be identified by the nodes in the diagrams such as Moulin, Haven, Consort, Charger, Lynx and Rialto. A viewing option that shows all the relatives of a single line emphasises the large influence of the older cultivars such as Cappelle Desprez, Rendezvous, Moulin, Maris Huntsman and Thatcher. Unfortunately without the interactive functions given within the program, the Pedigree Viewer diagrams are difficult to read. Therefore, some simplified diagrams of descendants for major varieties in the YR panel were redrawn in Microsoft PowerPoint and made available in Appendix 8.

3 DISCUSSION AND CONCLUSION

Many association panels have been reported in wheat and they are generally highly diverse as they cover a wide range of geographic area and/or include several germplasm groups (landrace, breeding lines, winter and spring wheats). Among the most reported panel in AM studies, we can cite the core collection from Balfourier et al. (2007) of 372 bread wheat including landraces and varieties covering 40 geographical areas (used by Bordes et al. (2008); Le Gouis et al. (2012); Rousset et al. (2011)), the 96 winter wheat collection from 21 countries exploited by Neumann et al. (2011); Rehman et al. (2012) and the 455 European advanced breeding line collection investigated by Miedaner et al. (2010); Reif et al. (2011). Rare are the association panels focusing on limited geographical area and limited breeding period, which makes the YR panel unique. The YR panel including 327 lines represents a large and coherent set of elite wheat focused on UK germplasm from the past three decades.

Additionally, the YR panel is better characterized than most of the panels proposed for association mapping as we have assembled an extensive set of information for each variety including country of origin, breeder, pedigree, alternatives name which are gathered in a database (see database description in the Chapter III).

The panel was assembled with the requirements of association mapping in mind. The association panel size was maximized including not only commercial varieties but also breeding lines that were never commercialized, knowing the population size has a great impact on the statistical power of association (detection of MTA)(Long and Langley, 1999). In addition, a large sample size allows capturing more alleles. Our limiting factors were the availability of reliable seed sources and the availability of reliable phenotypes for yellow rust.

One of the limiting factors in association mapping is the presence of population structure and close relatedness within individuals which lead to false positive MTA. By focussing on UK elite winter wheat germplasm from the last three decades, we wished to eliminate the source of major stratification (structure at macro-level). Breeding pools are known to be a major source of stratification (Breseghello and Sorrells, 2006a). In a collection of US wheat germplasm, two sub-populations corresponding to winter wheat and spring wheat have been identified (Chao et al., 2010). The geographic origin was shown to be a source of stratification in European wheat (Le Couviour et al., 2011; Reif et al., 2011; Tommasini et al., 2007).

Although sampling within a specific breeding pool (UK elite winter wheat) may have limited population structure at the macro-level, the YR panel is still likely to present an important population structure at the micro-level. As stated by Breseghello and Sorrells (2006a), a typical elite breeding pool is derived from a few founders in the recent past and

commonly included closely related lines. The YR panel is no exception as the pedigree information revealed many varieties used multiple times as parental lines (see pedigree diagrams in Appendix 8) as well as many sister lines, for instance Warlock 24 and Scorpion 25 (cross Cadenza x W929029), Equator and Heritage (cross Charger x Equinox). Therefore, a high degree of kinship is expected between lines from the YR panel and will need to be taken into account when running association mapping. In absence of complete pedigree information, the degree of kinship between lines will have to be determined based on independent markers.

While the close relatedness of lines in elites population such as the YR panel is detrimental to association mapping power, elite populations present significant advantages, they are adapted to local growing conditions and therefore allows the detection of low heritability traits. Furthermore, the favourable alleles are directly detected in target populations, elite lines with favourable allele could be directly used in breeding program and significant markers revealed by AM can be used for marker assisted selection in progeny.

Table II-3: Wheat varieties included in the YR panel

AFP is the UK application code for plant breeder right given by DEFRA. ww and sw are seasonal types respectively winter wheat and spring wheat. The year indicated corresponds to the plant breeder right first application, prior 1989 this date was not available, instead the year of released is indicated in reference of the following sources (a) http://www.geves.fr, (b) http://genbank.vurv.cz, (c) *www.bundessortenamt.de*. The country of origin is primarily assigned based on the country of the breeder. The seed source is the collection from where the seeds use to start the present study originated.

Variety name	AFP	ww/sw	Year	Breeder	Country	Seed source	Pedigree	Pedigree source
Aarden	1573	ww	2002	Saaten Union Recherche	France	TG	Aardvark x Denver	NIAB
Aardvark	1111	ww	1995	KWS UK Ltd	UK	TG	(Cadenza x Lynx 'sib') x Lynx	SACA
Abbot	967	ww	1993	Plant Breeding International Cambridge	UK	DUS	(Avalon x Brimstone) x Torfrida 'sib'	SACA
Abele	311	ww	1980(a)	Limagrain UK Ltd	UK	TG	R 68-114 x Maris Beacon	BBSRC
Ac Barrie	1591	sw	2003	Agricuture and Agrifood	Canada	BBSRC	(Neepawa x Columbus) x BW-90	SACA
Access	1266	ww	1998	KWS UK Ltd	UK	TG	90-15 x 91-6	SACA
Acclaim	972	ww	1993	Plant Breeding International Cambridge	UK	DUS	Hussar (gbr) x Beaver	ECPGR
Admiral	759	ww	1992	Limagrain UK Ltd	UK	TG	Mithras x(Hobbit x Hedgehog)	SACA
Adroit	825	ww	1990	Plant Breeding International Cambridge	UK	GEDIFLUX	(Norman x Mercia) x Moulin	BBSRC
Agami	1133	ww	1996	Limagrain UK Ltd	UK	TG	4215-5-1 x Brigadier	NIAB
Alchemist	1276	ww	1998	Plant Breeding International Cambridge	UK	DUS	(Brutus x Lynx) x Rialto	NIAB
Alchemy	1564	ww	2002	Nickerson International Research	UK	TG	Claire x (Consort x Woodstock)	SACA
Alsace	1429	ww	2000	Nickerson International Research	UK	DUS	-	-
Ambrosia	1462	ww	2001	Plant Breeding International Cambridge	UK	TG	(Cantata'sib' x Genesis) x Pinder	SACA
Andante	859	ww	1990	Plant Breeding International Cambridge	UK	GEDIFLUX	Moulin x D172.6.4	SACA
Anglo	1267	ww	1998	KWS UK Ltd	UK	DUS	94-12 x 93-27	SACA
Anvil	260	ww	1982(a)	Limagrain UK Ltd	UK	TG	(((TR.CA x Holdfast) x (Hybrid 46 x Viking)) x ((Chnese 166 x Cappelle) x CI-12633)) x ((RM-6 x Champlein) x (Joss x V-6603) x (RM-97 x RM-49))	ECPGR

Table II-3: Continued

Variety name	AFP	ww/sw	Year	Breeder	Country	Seed source	Pedigree	Pedigree source
Apollo	607	ww	1988	Saatzucht Josef Breun GdbR	Germany	TG	Maris Beacon x Kronjuwel	SACA
Apostle	670	ww	1980(a)	Plant Breeding Institute/NSDO	UK	TG	(Alcedo x Avalon) x Moulin	SACA
Aristocrat	785	ww	1992(a)	Plant Breeding International Cambridge	UK	DUS	(Rendezvous x Moulin) x Mercia	SACA
Ark	1233	ww	1997	KWS Lochow GmbH	Germany	DUS	Greif x Ibis	NIAB
Arlington	1184	ww	1997	Pioneer Hi-Bred	USA	DUS	HGC146 x SVC1141	NIAB
Armada	201	ww	1978	Limagrain UK Ltd	UK	TG	(TP 118 x (Perdix x Hybrid 46)) x (Cappelle x Champlein) x ((Viking x Tetrix) x (Tetrix x Jubilegem))	ECPGR
Arminda	612	ww	1976(a)	DJ Van Der Have BV	Netherland	TG	Carstens 854 x Ibis	BBSRC
Arran	1380	ww	1999	Nickerson International Research	UK	TG	Consort x Asset	SACA
Arriva	1222	ww	1997	KWS UK Ltd	UK	DUS	(Cadenza x Lynx) x Lynx	NIAB
Asagai	1543	ww	2002	Limagrain UK Ltd	UK	TG	(Charger x Maverick) x Savannah	SACA
Ashanti	1333	ww	1999	KWS UK Ltd	UK	DUS	(Cadenza x Lynx) x Lynx	NIAB x
Astron	801	ww	1990	Saatzucht F. Strube	Germany	TG	Blaukorn Abkommling x Monopol	ECPGR
Atla	862	ww	1990	Plant Breeding International Cambridge	UK	DUS	-	-
Atlanta	1477	ww	2001	KWS UK Ltd	UK	TG	(94-32 x Consort) x Krakatoa	SACA
Atoll	1122	ww	1996	Hybritech Europe SNC	France	TG	Moulin x (Drakkar x Marathon)	NIAB
Atou	111	ww	1973	Paul Guillemain	France	GEDIFLUX	Cappelle Desprez x Garnet	SACA
Avalon	287	ww	1980	Institute of Plant Science	UK	TG	Maris Plougham x Bilbo	SACA
Award	1407	ww	2000	Deutsche Saatveredelung AG	Germany	BSPB	Tambor x Charger	NIAB
Axial	707	ww	1989(a)	Etablissements Claude-Camille Benoist	France	DUS	((Talent x Maris Beacon) x Arminda) x Festival	SACA
Axona	544	sw	1983(a)	DJ Van Der Have BV	Netherland	GEDIFLUX	HPG 552-66 x Maris Dove	SACA
Baron	310	ww	1949(a)	Limagrain UK Ltd	UK	TG	R 68-114 x Maris Beacon	SACA
Battalion	1599	ww	2003	Monsanto	UK	TG	98ST08 x Aardvark	SACA
Beaufort	878	ww	1991	Plant Breeding International Cambridge	UK	TG	(Rendezvous x Haven) x Fresco	SACA

Table II-3: Continued

Variety name	AFP	ww/sw	Year	Breeder	Country	Seed source	Pedigree	Pedigree source
Beaver	692	ww	1990	Institute of Plant Science	UK	TG	(Hedgehog x Norman) x Moulin	SACA
Belter	1445	ww	2000	Plant Breeding International Cambridge	UK	DUS	Blaze 'sib' x Consort	NIAB
Benedict	1625	ww	2003	Limagrain UK Ltd	UK	BBSRC	CPBTW48(=Challenge) x Rialto	SACA
Bentley	1447	ww	2000	SAS Florimond Desprez	France	TG	Shango x FD-89034-23	SACA
Biscay	1227	ww	2000(b)	KWS UK Ltd	UK	TG	CPBT79 x Hussar	NIAB
Blaze	1029	ww	1994	Plant Breeding International Cambridge	UK	TG	Hussar x Beaver	SACA
Bogart	1105	ww	1995	KWS UK Ltd	UK	BSPB	-	-
Boston	-	-	2000	Landbouwbureau Wiersum BV	Netherland	TG	-	-
Bounty	274	ww	1979(a)	Institute of Plant Science	UK	TG	Maris ploughman x Durin	SACA
Bouquet	4	ww	1972	SAS Florimond Desprez	France	TG	(2/7 x Cappelle Desprez) x Cappelle Desprez	SACA
Boxer	509	ww	1987(a)	Limagrain UK Ltd	UK	GEDIFLUX	Griffin x RPB 181-70D	SACA
Brando	1156	ww	1996	KWS UK Ltd	UK	TG	(Cadenza x Lynx) x Lynx	ECPGR
Brigadier	818	ww	1992	Limagrain UK Ltd	UK	TG	Squadron x Rendezvous	SACA
Brigand	231	ww	1979	Institute of Plant Science	UK	TG	Maris Huntsman x Bilbo	SACA
Brock	489	ww	1985(a)	Institute of Plant Science	UK	TG	Hobbit '30/2' x Talent	SACA
Broiler	1137	ww	1996	Limagrain UK Ltd	UK	TG	composite cross	SACA
Brompton	1502	ww	2001	Elsoms Ltd UK	UK	DUS	CWW92.1 x Caxton	SACA
Brunel	1327	ww	1999	KWS UK Ltd	UK	TG	Krakatoa x Beaufort	SACA
Bryden	824	ww	1990	Plant Breeding International Cambridge	UK	GEDIFLUX	-	BBSRC
Buccaneer	1044	ww	1994	KWS UK Ltd	UK	DUS	(Gawain x Riband) x CWW 4442/ 64	SACA
Buchan	1100	ww	1995	Limagrain UK Ltd	UK	TG	Beaver x Hussar	SACA
Buster	845	ww	1990	Limagrain UK Ltd	UK	TG	Brimstone x Parade	SACA
Cadenza	833	ww	1990	KWS UK Ltd	UK	TG	Axona x Tonic	SACA
Camp Remy	798	ww	1980(b)	Unisigma GIE/Société Européenne de	France	TG	(362 x Atou) x Hardi	SACA

Table II-3: Continued

Variety name	AFP	ww/sw	Year	Breeder	Country	Seed source	Pedigree	Pedigree source
				Semences Europe NV SA				
Cantata	1030	ww	1994	Plant Breeding International Cambridge	UK	TG	Adroit x Torfrida	SACA
Canterbury	1223	ww	1997	KWS UK Ltd	UK	TG	Riband x Lynx	SACA
Cappelle desprez	-	ww	1946	SAS Florimond Desprez	France	TG	Hybride du Jonquois x Vilmorin 27	SACA
Caphorn	1438	ww	2000	Plant Breeding International Cambridge	UK	BSPB	(S-14579-454 x Rialto) x Beaufort	ECPGR
Capnor	1310	ww	1999	G.A.E. Recherche	France	TG	Fertil x ((Pluton x Armada) x Pernel)	NIAB
Caprimus	783	ww	1994(a)	Plant Breeding International Cambridge	UK	DUS	Gawain x Aquila	SACA
Carlton	1343	ww	1999	Elsoms Ltd UK	UK	TG	CWW-92-1 x FD-92054	SACA
Carstens V	-	ww	1921	Rudolph Carsten	Germany	GEDIFLUX	(Carstens 3 x Dickkopf) x (Dickkopf x Criewener)	ECPGR
Caxton	931	ww	1992	Elsoms Ltd UK	UK	DUS	Moulin x Riband	SACA
Chardonnay	1383	ww	1999	Nickerson International Research	UK	DUS	Spry x Rialto	SACA
Charger	939	ww	1992	Plant Breeding International Cambridge	UK	TG	Fresco 'sib' x Mandate	SACA
Chatsworth	1258	ww	1998	Limagrain UK Ltd	UK	TG	Estica x Genesis	SACA
Chaucer	1039	ww	1994	Elsoms Ltd UK	UK	TG	Apollo(deu)*Rendezvous	SACA
Chequer	1249	ww	1998	Syngenta Seeds UK	UK	DUS	Lynx x NFC-969-27	NIAB
Chester	1511	ww	2001	Nickerson International Research	UK	BBSRC	(Haven x Hussar) x Claire 'sib'	NIAB
Chianti	946	ww	1992	KWS UK Ltd	UK	TG	CWW-4442-64 x Rendezvous	SACA
Chicago	1151	ww	1996	KWS UK Ltd	UK	DUS	Haven x Obelisk	NIAB
Choice	1470	ww	2001	Plant Breeding International Cambridge	UK	DUS	Aardvark x Abbot	NIAB
Claire	1070	ww	1995	Limagrain UK Ltd	UK	TG	Wasp x Flame	ECPGR
Clement	134	ww	1974(a)	Innoseed BV	Netherland	GEDIFLUX	(((Hope x Timstein) x (Heine 7)3) x ((Riebesel 57-41 x (Heine 7)2)) x Cleo	BBSRC
Clove	940	ww	1992	Plant Breeding International Cambridge	UK	DUS	-	-
Comet	1270	ww	1998	Plant Breeding International Cambridge	UK	DUS	((Norman x D84-4-12)) x Haven) x Consort	NIAB x

Table II-3: Continued

Variety name	AFP	ww/sw	Year	Breeder	Country	Seed source	Pedigree	Pedigree source
Commodore	1081	ww	1995	Plant Breeding International Cambridge	UK	BSPB	Hussar x (Haven x Complex cross)	NIAB
Conqueror	1813	ww	2005	KWS UK Ltd	UK	DUS	Robigus x Equinox	SACA
Consort	882	ww	1991	Plant Breeding International Cambridge	UK	TG	(Riband 'sib' x Fresco)xRiband	SACA
Contender	1611	ww	2003	Limagrain UK Ltd	UK	BBSRC	Nelson x Wasmo	NIAB
Context	1331	ww	1999	KWS UK Ltd	UK	DUS	Consort x Lynx	NIAB
Convoy	1332	ww	1999	KWS UK Ltd	UK	DUS	(91-35 x Cadenza) x 91-35	NIAB
Copain	265	ww	1977(a)	Etablissements Claude-Camille Benoist	France	TG	(((Thatcher x Vilmorin 27) x (Petit Quinquin x Hybrid 40)) x ((Thatcher x Vilmorin 27) x Fortunato))x Mexique 50	ECPGR
Cordiale	1388	ww	2000	KWS UK Ltd	UK	TG	(Reaper x Cadenza) x Malacca	SACA
Cranley	1203	ww	1997	Plant Breeding International Cambridge	UK	BSPB	((Norman x D84-4-12) x Haven) x Hussar	NIAB
Crofter	954	ww	1992	Limagrain UK Ltd	UK	TG	Riband x Moulin	SACA
Cyber	1224	ww	1997	KWS UK Ltd	UK	TG	(Talon x Beaver) x Lynx	NIAB
Dart	1439	ww	2000	Monsanto	UK	DUS	(Consort x Madrigal) x Consort	SACA
Datum	1108	ww	1995	KWS UK Ltd	UK	BBSRC	(Talon x Beaver) x Lynx 'sib'	NIAB
Dean	732	ww	1989(a)	Institute of Plant Science	UK	GEDIFLUX	Disponent x Norman	SACA
Deben	1220	ww	1997	Limagrain UK Ltd	UK	TG	(Hunter x Buster) x Wasp	NIAB
Defender	1468	ww	2001	Plant Breeding International Cambridge	UK	TG	Classic x Charger	SACA
Denver	1155	ww	1996	KWS UK Ltd	UK	DUS	(Orestis x Lynx) x Bandit	NIAB
Derwent	1161	ww	1996	Pioneer Hi-Bred	USA	BBSRC	HGC146 x FVL158	NIAB
Diablo	806	ww	1990	Limagrain UK Ltd	UK	GEDIFLUX	BS 934 x BS 948	SACA
Dickins	1115	ww	1995	Syngenta Seeds UK	UK	BSPB	-	-
Dickson	1411	ww	2000	Limagrain UK Ltd	UK	TG	Abbot x Consort	SACA
Director	1542	ww	2002	Limagrain UK Ltd	UK	TG	(Charger x Maverick) x Savannah	SACA
Dorial	1197	ww	1997	Secobra Recherches	France	DUS	(Ares x Severin) x Orestis	NIAB

Table II-3: Continued

Variety name	AFP	ww/sw	Year	Breeder	Country	Seed source	Pedigree	Pedigree source
Dover	1550	ww	2002	KWS UK Ltd	UK	TG	(Biscay x Aardvark)x F86Z46-6-2	SACA
Drake	934	ww	1992	Syngenta Seeds UK	UK	GEDIFLUX	Motto x(Bounty x Galahad)	SACA
Duxford	1725	ww	2004	Syngenta Seeds UK	UK	DUS	Solstice x Scorpion25	SACA
Dynamo	896	ww	1991	Limagrain UK Ltd	UK	GEDIFLUX	Hammer x Parade	SACA
Eclipse	1126	ww	1996	Syngenta Seeds UK	UK	TG	NFC94-334 x Drake 'sib'	SACA
Einstein	1376	ww	1999	Limagrain UK Ltd	UK	TG	NSL 91/1670 x NPL 90-1282	SACA
Ekla	724	ww	1988(a)	Unisigma GIE/Société Européenne de Semences Europe NV SA	France	TG	(Wizard x 1425) x (1144 x Talent)	BBSRC
Electron	1196	ww	1997	Secobra Recherches	France	DUS	Pastiche x Genial	NIAB
Encore	881	ww	1991	Plant Breeding International Cambridge	UK	GEDIFLUX	Apostle x Haven	SACA
Equator	1463	ww	2001	Plant Breeding International Cambridge	UK	DUS	Equinox x Charger	NIAB
Equinox	983	ww	1993	KWS UK Ltd	UK	TG	CWW 4442-64 x (Rendezvous x Obelisk)	SACA
Estica	775	ww	1991(b)	Cebeco Zaden BV/Innoseeds BV	Netherland	TG	Arminda x Virtue	SACA
Exeter	1512	ww	2001	Nickerson International Research	UK	TG	(Flame x Brigadier) x Rialto	SACA
Explosiv	1152	ww	1996	KWS UK Ltd	UK	DUS	Talon x Hussar	NIAB
Exsept	1213	ww	1997	Limagrain UK Ltd	UK	TG	Hereward x (Moulin x Boxer)	ECPGR
Extend	1464	ww	2001	Plant Breeding International Cambridge	UK	DUS	P-1527 x Consort	NIAB
Falstaff	1031	ww	1994	Plant Breeding International Cambridge	UK	TG	(Hussar x Beaver) x Hunter	SACA
Fastnet	1549	ww	2002	KWS UK Ltd	UK	TG	Buster x Equinox	SACA
Feast	1271	ww	1998	Plant Breeding International Cambridge	UK	DUS	((Slejpner x Torfrida 'sib') x Beaufort) x Rialto	NIAB
Fenda	811	ww	1990	A.W. PAGE Plant Breeders Ltd	UK	GEDIFLUX	TJB 989-4 x MMG 4170-7	SACA
Fender	1263	ww	1998	KWS UK Ltd	UK	DUS	93-13 x Beaver	SACA
Fielder	1367	ww	1999	Pioneer Hi-Bred	-	DUS	(Brutus x Lynx) x Rialto	NIAB
Flair	1120	ww	1996	Hans Schweiger & Co. OHG	Germany	TG	Ares x Marabu (Deu)	ECPGR

Table II-3: Continued

Variety name	AFP	ww/sw	Year	Breeder	Country	Seed source	Pedigree	Pedigree source
Flame	847	ww	1990	Limagrain UK Ltd	UK	TG	Taurus x Moulin	SACA
Flaxen	1400	ww	2000	Syngenta Seeds UK	UK	DUS	NFC-6929 x NFC-3436-13-C-A	SACA
Fletum	803	ww	1990	Semundo BV	Netherland	GEDIFLUX	-	-
Frelon	1277	ww	1998	Plant Breeding International Cambridge	UK	DUS	(Genial x Torfrida) x Soissons	SACA
Fresco	672	ww	1988(a)	Plant Breeding Institute/NSDO	UK	DUS	Moulin x Monopol	SACA
Galahad	440	ww	1983(a)	Plant Breeding Institute/NSDO	UK	GEDIFLUX	(Joss Cambier x Durin) x Hobbit 'Sib'	SACA
Galatea	943	ww	1992	Plant Breeding International Cambridge	UK	GEDIFLUX	Sicco 'sib' x Galahad	ECPGR
Gallant	1766	ww	2005	Syngenta Seeds UK	UK	DUS	(Malacca x Charger) x Xi19	SACA
Gatsby	1546	ww	2002	Limagrain UK Ltd	UK	TG	Nelson x Wasmo	SACA
Genesis	800	ww	1990	Serasem Recherches	France	GEDIFLUX	Arminda x TJB 363	SACA
Genghis	1153	ww	1996	KWS UK Ltd	UK	TG	Rialto x 24-1-420	SACA
Gladiator	1442	ww	2000	Plant Breeding International Cambridge	UK	DUS	Falstaff x Shannon	SACA
Glasgow	1482	ww	2001	Saaten Union Recherche	France	TG	(Ritmo x ZE.90 - 2666) x ZE.91.11658	SACA
Goldlace	1206	ww	1997	Plant Breeding International Cambridge	UK	BSPB	Consort x (Rendezvous x Haven)	NIAB
Goodwill	1353	ww	1999	Limagrain UK Ltd	UK	TG	Flame x Hunter	SACA
Granta	296	ww	1980(a)	Limagrain UK Ltd	UK	TG	Cariba x Kranich	BBSRC
Gulliver	1621	ww	2003	Nickerson International Research	UK	DUS	Shamrock x Aardvark	SACA
Harbour	1370	ww	1999	Syngenta Seeds UK	UK	DUS	Consort x NFC-5204	SACA
Harrier	978	ww	1993	Limagrain UK Ltd	UK	DUS	Soldier x Beaver	SACA
Harrow	1251	ww	1998	Syngenta Seeds UK	UK	DUS	NFC-967-18 x Drake	NIAB
Haven	694	ww	1990	Institute of Plant Science	UK	TG	(Hedgehog x Norman) x Moulin	SACA
Hereford	1731	ww	2004	Sejet Plant Breeding	Denmark	DUS	Solist x Deben	SACA
Hereward	736	ww	1991	Institute of Plant Science	UK	TG	Norman 'sib' x Disponent	SACA
Heritage	1443	ww	2000	Plant Breeding International Cambridge	UK	TG	Charger x Equinox	SACA

Table II-3: Continued

Variety name	AFP	ww/sw	Year	Breeder	Country	Seed source	Pedigree	Pedigree source
Hobbit	179	ww	1977(b)	Institute of Plant Science	UK	TG	((CI 12633 x Cappelle desprez) x (Cappelle desprez x Heines 110) x Professeur Marchal) x ((Marne Desprez x VG 9144) x Nord Desprez)	ECPGR
Holster	992	ww	1993	Limagrain UK Ltd	UK	GEDIFLUX	Fresco x Rendezvous	NIAB
Hornet	591	ww	1986(a)	Institute of Plant Science	UK	TG	Norman x Hedgehog	SACA
Hourra	1556	ww	2002	Adrien Momont et Fils	France	TG	M H 91-16 x M H 48-64	NIAB
Hudson	982	ww	1993	KWS UK Ltd	UK	DUS	(Riband x Rendezvous) x Riband	NIAB
Humber	1652	ww	2003	KWS UK Ltd	UK	TG	Anglo x Krakatao	SACA
Hunter	828	ww	1990	Plant Breeding International Cambridge	UK	GEDIFLUX	Apostle x Haven	SACA
Hurley	1577	ww	2002	Elsoms Ltd UK	UK	TG	V1-DH-4 / CE 422-4	SACA
Hussar	817	ww	1989	Limagrain UK Ltd	UK	DUS	Squadron x Rendezvous	SACA
Hustler	230	ww	1978	Institute of Plant Science	UK	TG	Maris Huntsman xTL 365a/25(=Durin)	SACA
Hyperion	1561	ww	2002	Nickerson International Research	UK	TG	Aardvark x (Consort x Woodstock)	SACA
Impala	970	ww	1993	Plant Breeding International Cambridge	UK	TG	Andante x Dean	SACA
Insight	1359	ww	1999	Plant Breeding International Cambridge	UK	DUS	Flame x Hussar	NIAB
Isengrain	-	ww	1997	SAS Florimond Desprez	France	GEDIFLUX	Apollo (Deu) x Soissons	ECPGR
Isidor	1489	ww	2002(b)	Unisigma GIE/Société Européenne de Semences Europe NV SA	France	TG	Victo x UN-47	NIAB
Istabraq	1426	ww	2000	Nickerson International Research	UK	TG	Claire x Consort	SACA
Jacadi	1008	ww	1994	Lemaire Deffontaines	France	BBSRC	FLorin x Brimstone	SACA
JB Diego	1737	ww	2004	Saatzucht Josef Breun GdbR	Germany	DUS	3351B2 x STRU2374	SACA
Joss Cambier	88	ww	1968	Cambier Freres	France	TG	(Heine 7 x Tadepi) x Cappelle Desprez	SACA
Kador	205	ww	1977	Serasem Recherches	France	TG	(Champlein x Cappelle Desprez) x B 21 (=Versailles)	SACA
Kempt	1275	ww	1998	Plant Breeding International Cambridge	UK	DUS	(Madrigal 'sib' x Beaufort) x Consort	NIAB
Ketchum	1765	ww	2005	Syngenta Seeds UK	UK	DUS	Solstice x Xi 19	SACA

Table II-3: Continued

Variety name	AFP	ww/sw	Year	Breeder	Country	Seed source	Pedigree	Pedigree source
Kinsman	178	ww	1970s	Institute of Plant Science	UK	TG	((CI 12633 x Cappelle Desprez) x (Hybrid 46 x Cappelle Desprez) x Professeur Marchal) x Maris Ranger	BBSRC
Kipling	1578	ww	2002	SAS Florimond Desprez	France	BBSRC	Hunter x 92054	SACA
Krakatoa	1047	ww	1994	KWS UK Ltd	UK	TG	Apollo(deu) x CWW-4442-64	SACA
KWS horizon	1882	ww	2006	KWS UK Ltd	UK	DUS	Cordialex Robigus	SACA
Lancelot	770	ww	2002(b)	Verneuil Recherche SA/Limagrain	France	TG	-	-
Leo	826	ww	1990	Nickerson International Research	UK	GEDIFLUX	Kristall x Marksman	BBSRC
Limerick	1726	ww	2004	Syngenta Seeds UK	UK	DUS	Solstice x Scorpion 25	SACA
Longbow	364	ww	1983	Institute of Plant Science	UK	GEDIFLUX	TJB 268-175 x Hobbit	SACA
Lorraine	-	ww	1998	Etablissements Claude-Camille Benoist	France	TG	-	-
Lynx (1)	704	ww	1980s	MMG Agriseed	UK	TG	Arminda x Norman	BBSRC
Lynx (2)	856	ww	1990	KWS UK Ltd	UK	DUS	Rendezvous x CWW-4442-64	SACA
Macro	1262	ww	1998	KWS UK Ltd	UK	TG	(Orestis x 5006)x 91-11	SACA
Madrigal	973	ww	1993	Plant Breeding International Cambridge	UK	DUS	Hussar x Beaver	SACA
Magellan	945	ww	1992	KWS UK Ltd	UK	GEDIFLUX	CWW-4462-64 x Rendezvous	SACA
magnitude	1467	ww	2001	Plant Breeding International Cambridge	UK	DUS	Classic x Charger	NIAB
Malacca	980	ww	1993	KWS UK Ltd	UK	TG	Riband x(Rendezvous x Apostle)	SACA
Mallet	1361	ww	1999	Plant Breeding International Cambridge	UK	DUS	((Obelisk x Haven)x Haven) x Rialto	SACA
Maris Beacon	41	ww	1968(a)	Institute of Plant Science	UK	BBSRC	((CI 12633 x (Cappelle Desprez) 5) x Hybrid 46) x Professeur Marchal	SACA
Maris Freeman	103	ww	1974(a)	Institute of Plant Science	UK	TG	Maris Ranger x Maris Widgeon	SACA
Maris Huntsman	66	ww	1972	Institute of Plant Science	UK	GEDIFLUX	((CI 12633 x (Cappelle Desprez) 5) x Hybrid 46) x Professeur Marchal	SACA
Maris Templar	67	ww	1968(a)	Institute of Plant Science	UK	BBSRC	((((CI-12633 x Cappelle Desprez) x (Heines 110 x Cappelle Desprez)) x Nord desprez) x Viking	ECPGR

Table II-3: Continued

Variety name	AFP	ww/sw	Year	Breeder	Country	Seed source	Pedigree	Pedigree source
Marksman	1687	ww	2004	R2N/RAGT Seeds Ldt	UK	DUS	98ST08 x Aardvark	SACA
Marshal	1078	ww	1995	Limagrain UK Ltd	UK	BBSRC	Kontiki x Brigadier	SACA
Mascot	1532	ww	2002	Monsanto	UK	DUS	Reaper x Rialto	SACA
Maverick	1035	ww	1994	Limagrain UK Ltd	UK	TG	Talon x Torfrida	SACA
Mayfair	1409	ww	2000	Limagrain UK Ltd	UK	DUS	Lynx x (Genesis x Rialto)	NIAB
Mayfield	1461	ww	2001	Plant Breeding International Cambridge	UK	DUS	Falstaff x Ritmo	NIAB
Mega	109	ww	1974	Nickerson UK	UK	GEDIFLUX	(Cappelle Desprez x H 2596) x 6003	SACA
Mercia	533	ww	1986	Institute of Plant Science	UK	TG	(Talent x Virtue) x Flanders	SACA
Milestone	1199	ww	1997	Limagrain UK Ltd	UK	DUS	Vivant x Estica	SACA
Monty	1727	ww	2004	Syngenta Seeds UK	UK	DUS	Robigus x NFC-10035	SACA
Monument	1441	ww	2000	Plant Breeding International Cambridge	UK	DUS	Falstaff x Ritmo	NIAB
Moulin	486	ww	1985(a)	Institute of Plant Science	UK	TG	(CIMMYT CB 306Y70 x Maris Widgeon) x Hobbit 'sib'	SACA
Napier	1147	ww	1996	Plant Breeding International Cambridge	UK	GEDIFLUX	Hussar (Gbr) x Lynx	SACA
Newhaven	942	ww	1992	Plant Breeding International Cambridge	UK	DUS	-	-
Nexus	1240	ww	1997	Svalof Weibull (Plant Breeders Ltd)	Germany	DUS	Hussar x Adroit	NIAB
Nijinsky	1427	ww	2000	Nickerson International Research	UK	TG	Claire x Consort	SACA
Norman	321	ww	1981	Institute of Plant Science	UK	TG	TJB-268-175 x Hobbit 'sib'	SACA
Oakley	1658	ww	2003	KWS UK Ltd	UK	TG	(Aardvark 'sib' x Robigus) x Access	SACA
Ochre	1606	ww	2003	Monsanto Technology LCC/Ragt seeds Ltd	UK	DUS	98ST31 x Cortez	SACA
Odyssey	1211	ww	1997	Syngenta Seeds UK	UK	TG	Rialto x Drake	SACA
Option	1200	ww	1997	Plant Breeding International Cambridge	UK	GEDIFLUX	Vivant x Rialto 'sib'	SACA
Orestis	755	ww	1988(a)	Saatzucht H. Strube	Germany	TG	Obelisk	ECPGR
Orton	1210	ww	1997	Syngenta Seeds UK	UK	DUS	NFC403-1 x Rialto	NIAB
Ostara	863	ww	1990	Plant Breeding International Cambridge	UK	GEDIFLUX	-	-

Table II-3: Continued

Variety name	AFP	ww/sw	Year	Breeder	Country	Seed source	Pedigree	Pedigree source
Oxbow	1202	ww	1997	Plant Breeding International Cambridge	UK	TG	Brigadier x Consort	NIAB
Pagan	1478	sw	2001	KWS UK Ltd	UK	DUS	Savannah x 95-93	NIAB
Parade	565	ww	1987(a)	Limagrain UK Ltd	UK	GEDIFLUX	Granta x Marksman	SACA
Pastiche	671	ww	1988(a)	Plant Breeding Institute/NSDO	UK	TG	Jena x Norman	SACA
Pennant	1450	ww	2000	Elsoms Ltd UK	UK	DUS	Malacca x FD-92054	SACA
Phlebas	1278	ww	1998	KWS UK Ltd	UK	TG	(89-13 x 5006) x 5006A	SACA
Piranha	1541	ww	2002	Limagrain UK Ltd	UK	TG	Claire x Krakatoa	SACA
Posit	1269	ww	1998	Plant Breeding International Cambridge	UK	DUS	Consort x Madrigal	NIAB
Potent	1205	ww	1997	Plant Breeding International Cambridge	UK	TG	Spry x Rialto	NIAB
PR21R60	1321	ww	1999	Pioneer Hi-Bred	USA	DUS	FBP0091 x FVP0040	NIAB
Predator	1499	ww	2001	Syngenta Seeds UK	UK	BBSRC	Stainton x Eclipse 'sib'	SACA
Prophet	867	ww	1990	Syngenta Seeds UK	UK	GEDIFLUX	Moulin x (Braco x VPM)	SACA
QPlus	1789	ww	2005	Nickerson International Research	UK	DUS	Drifter x Solstice	SACA
Quest	1392	ww	2000	KWS UK Ltd	UK	TG	94-35 x Hudson	SACA
Raglan	1456	ww	2001	Mike Pickford	-	TG	Charger x Abbot	NIAB
Raleigh	933	ww	1992	Syngenta Seeds UK	UK	TG	Motto x (Bounty x Galahad)	SACA
Rampart	1272	ww	1998	Plant Breeding International Cambridge	UK	DUS	((Rialto 'sib' x Torfrida) x Rialto) x Brutus	NIAB
Ranger	1157	ww	1996	KWS UK Ltd	UK	DUS	Blitz x CPB188	NIAB
Reaper	932	ww	1992	Syngenta Seeds UK	UK	TG	Haven x NFC-251	SACA
Rendezvous	585	ww	1985(a)	Institute of Plant Science	UK	DUS	(VPM1 x Hobbit 'sib') x Virtue	SACA
Renown	827	ww	1990	Plant Breeding International Cambridge	UK	GEDIFLUX	Squareheads Master*Swedish Squarehead	BBSRC
Reydon	1201	ww	1997	Plant Breeding International Cambridge	UK	BSPB	Beaufort x Haven	NIAB
Rialto	858	ww	1990	Plant Breeding International Cambridge	UK	TG	Haven'sib' x Fresco'sib'	SACA
Riband	628	ww	1989	Plant Breeding International Cambridge	UK	TG	Norman x (Maris Huntsman x TW 161)	SACA

Table II-3: Continued

Variety name	AFP	ww/sw	Year	Breeder	Country	Seed source	Pedigree	Pedigree source
Richmond	1257	ww	1998	Limagrain UK Ltd	UK	TG	Flame x Brigadier	SACA
Ritmo	955	ww	1992	Cebeco Zaden BV/Innoseeds BV	Netherland	TG	((Hobbit x (Line 1320 x Wizard)) x Marksman)	SACA
Rivet	1364	ww	1999	Plant Breeding International Cambridge	UK	DUS	(Hussar x Buster) x Beaufort	NIAB
Robigus	1330	ww	1999	KWS UK Ltd	UK	TG	composite cross	SACA
Rosario	1516	ww	2001	Secobra Recherches	France	DUS	Magellan x Charger	NIAB
Rosette	1088	ww	1995	Plant Breeding International Cambridge	UK	DUS	-	-
Rubens	966	ww	1993	Verneuil Recherche SA/Limagrain	France	TG	(MD-286 x Pernel) x Genial	ECPGR
Russet	948	ww	1992	KWS UK Ltd	UK	DUS	(Fresco x Rendezvous) x 4442/64	NIAB
Sabre	1295	ww	1998	Nickerson International Research	UK	BBSRC	-	-
Sahara	1649	ww	2003	Limagrain UK Ltd	UK	TG	Savannah x Claire	SACA
Samurai	1497	ww	2001	Deutsche Saatveredelung AG	Germany	BBSRC	(Texel x Transit) x Lynx	NIAB
Sancerre	1428	ww	2000	Nickerson International Research	UK	DUS	-	-
Sarek	765	ww	1992(a)	W WEIBULL/Svalof Weibull AB	Sweden	GEDIFLUX	Holger x (Maris Hunstman x Bilbo)	SACA
Savannah	1033	ww	1994	Limagrain UK Ltd	UK	TG	Riband x Brigadier	SACA
Scandia	1469	ww	2001	Plant Breeding International Cambridge	UK	DUS	Aardvark x Charger	NIAB
Scorpion 25	1335	ww	1999	Advanta Seeds UK Ltd	UK	TG	W929029 x Cadenza	SACA
Senator	1395	ww	2000	KWS UK Ltd	UK	TG	Krakatoa x Beaufort	SACA
Shamrock	1092	ww	1995	Limagrain UK Ltd	UK	TG	Fresco x complex TIG323-1-3M	SACA
Shango	-	ww	1994	Plant Breeding International Cambridge	UK	TG	Fresco x Tiresius	ECPGR
Shannon	938	ww	1992	Plant Breeding International Cambridge	UK	DUS	-	-
Slade	1204	ww	1997	Plant Breeding International Cambridge	UK	DUS	((Norman x D84-4-12) x Haven) x Consort	NIAB
Slejpner	537	ww	1986	WEIBULLSHOLM Plant breeding institute/Svalof Weibull	Sweden	GEDIFLUX	(W 20102 x CB 149 x Maris Huntsman) x Maris Bilbo	SACA
Smuggler	1415	ww	2000	Limagrain UK Ltd	UK	BBSRC	W930752 x 6438-283 B	SACA

Table II-3: Continued

Variety name	AFP	ww/sw	Year	Breeder	Country	Seed source	Pedigree	Pedigree source
Soissons	834	ww	1987(a)	SAS Florimond Desprez	France	DUS	Jena x Hybride Naturel 35	SACA
Soldier	760	ww	1980s	Limagrain UK Ltd	UK	BBSRC	Sqaudron x Rendezvous	ECPGR
Soleil	610	ww	1985(a)	Lemaire Deffontaines	France	TG	LD339 x (Joss x Top)	SACA
Solstice	1282	ww	1998	Limagrain UK Ltd	UK	TG	Rialto x Vivant	SACA
Spaldings prolific	-	ww	1923	-	UK	BBSRC	-	-
Spark	808	ww	1990	Limagrain UK Ltd	UK	TG	Moulin x Tonic	SACA
Spry	937	ww	1992	Plant Breeding International Cambridge	UK	DUS	-	-
Squadron	626	ww	1980s	MMG Agriseed	UK	GEDIFLUX	MMG 0370-18 x F1(MMG 442-47-1 x MMG 4668-8)	ECPGR
Steadfast	1440	ww	2000	Plant Breeding International Cambridge	UK	TG	Drake x Madrigal	SACA
Stetson	389	ww	1983	Limagrain UK Ltd	UK	GEDIFLUX	(TP 226 x Lincoln) xBenno	SACA
Storm	1286	ww	1998	Limagrain Nederland BV	Netherland	TG	Apollo x Trawler	SACA
SW Maxi	-	ww	2002	Svalof Weibull AB/Lantmännen SW Seed Hadmersleben GmbH	Germany	TG	-	-
SW Tataros	1435	ww	2000	Lantmännen SW Seed Hadmersleben GmbH	Germany	TG	Rendezvous x tambor	ECPGR
Talon	728	ww	1992(a)	Nordsaat Saatzuchtgesellschaft	Germany	TG	(Maris Huntsman x Sava) x (NS 372 x Maris Huntsman)	SACA
Tambor	-	ww	1995	Lantmännen SW Seed Hadmersleben GmbH	Germany	TG	(Hadmerslebener-26384-78 x Taras) x Taras	ECPGR
Tanker	1234	ww	1997	Elsoms Ltd UK	UK	TG	Beaver x Zodiac	SACA
Tara	735	ww	1989(a)	Institute of Plant Science	UK	GEDIFLUX	(Clement x Marksman) x Brock	SACA
Tellus	1326	ww	1999	KWS UK Ltd	UK	TG	composite cross	SACA
Tempest	1028	ww	1994	Plant Breeding International Cambridge	UK	BSPB	-	-
Temple	1019	ww	1994	Innoseed BV	Netherland	BSPB	-	-
Thatcher	-	sw	1934(a)	Minnesota Agricultural Experiment Station	USA	BSPB	(Marquis x Lumillo) x (Marquis x Kanred)	BBSRC
Tilburi	1020	ww	1994	Hybritech Europe SNC	France	TG	Pernel x Kurt	ECPGR

Table II-3: Continued

Variety name	AFP	ww/sw	Year	Breeder	Country	Seed source	Pedigree	Pedigree source
Tiller	1050	ww	1994	Syngenta Seeds UK	UK	BSPB	Riband x Hussar	SACA
Timber	1644	ww	2003	Saaten Union Recherche	France	TG	Terrier x Hamac	SACA
Tommy	23	ww	1971(a)	Etablissements Claude-Camille Benoist	France	TG	Champlein x (Petit Quinquin x Hybrid 40) x (Thatcher x Vilmorin 27)	BBSRC
Tonic	496	sw	1985(a)	Limagrain UK Ltd	UK	DUS	RPB 87-73 x RPB 94-73	SACA
Torfrida	787	ww	1992	Plant Breeding International Cambridge	UK	TG	Rendezvous x (Moulin x Mercia)	SACA
Toronto	773	ww	1990(c)	Saatzucht Streng GmbH & co./Saatzuchtgesellschaft	Germany	GEDIFLUX	(Disponent x WEIHENSTEPHANER-616-67) x Kronjuwel	ECPGR
Travix	1226	ww	1997	KWS UK Ltd	UK	DUS	Rialto x Lynx	NIAB
Trend	844	ww	1990	KWS Lochow GmbH	Germany	GEDIFLUX	-	-
Turpin	886	ww	1991	Syngenta Seeds UK	UK	GEDIFLUX	-	-
Urban	682	ww	1981(a)	Saarzuchtwirtschafte	Germany	GEDIFLUX	Kranich x Diplomat	SACA
Vault	1273	ww	1998	Plant Breeding International Cambridge	UK	DUS	(Tara x Haven) x (Andante x Haven)	NIAB
Vector	1414	ww	2000	Limagrain UK Ltd	UK	TG	Ekla x Nova x Haven	SACA
Verdon	1195	ww	1997	Secobra Recherches	France	BSPB	Hereward x Genial	NIAB
Veritas	829	ww	1990	Plant Breeding International Cambridge	UK	GEDIFLUX	-	-
Vilmorin 27	-	ww	1928	Vilmorin	France	TG	(Dattel x (Japhet x Parsel)) x (Hatif Inversable x Bon Fermier)	ECPGR
Virtue	276	ww	1979	Institute of Plant Science	UK	TG	Maris Huntsman x Maris Durin	SACA
Virtuose	1292	ww	1998	Institut National de la Recherche Agronomique	France	TG	(VM-713 x CF-1851) x (CF-1616 x C-103)	ECPGR
Vivant	1003	ww	1990	Plant Breeding International Cambridge	UK	DUS	Boxer x Gawain	ECPGR
Vuka	-	ww	1975	Hohenheim University	Germany	GEDIFLUX	Fiorello x U 1(=Osjecka Sisulja)	BBSRC
Warlock 24	1336	ww	1999	Limagrain UK Ltd	UK	TG	W929029 x Cadenza	SACA
Warrior (1)	994	ww	1993	Limagrain UK Ltd	UK	BSPB	Sniper x Rendezvous	SACA

Table II-3: Continued

Variety name	AFP	ww/sw	Year	Breeder	Country	Seed source	Pedigree	Pedigree source
Warrior (2)	1865	ww	2006	Societe R2N/RAGT	France	DUS	CM8228 x Robigus	NIAB
Wasp	776	ww	1990(a)	Limagrain UK Ltd	UK	TG	Galahad x Boxer	SACA
Welford	1449	ww	2000	Elsoms Ltd UK	UK	TG	CWW-9-21 x FD-92054	SACA
Wellington	1048	ww	1994	Syngenta Seeds UK	UK	TG	Haven x NFC257	SACA
Weston	1040	ww	1994	Elsoms Ltd UK	UK	TG	Apollo (Deu) x Rendezvous	SACA
Wickham	1149	ww	1996	Plant Breeding International Cambridge	UK	DUS	Rialto x Morell	NIAB
Windsor	1300	ww	1998	Saatzucht Josef Breun GdbR	Germany	DUS	Apollo (Deu) x Gawain	ECPGR
Wizard	1328	ww	1999	KWS UK Ltd	UK	DUS	Consort x Chianti	SACA
Woburn	1259	ww	1998	Limagrain UK Ltd	UK	DUS	Flame x Hunter	SACA
Woodstock	848	ww	1990	Limagrain UK Ltd	UK	GEDIFLUX	Longbow x Septoria resistant selection	SACA
XI19	1281	ww	1998	Limagrain UK Ltd	UK	TG	(Cadenza x Rialto) x Cadenza	SACA
Zaka	1329	ww	1999	KWS UK Ltd	UK	DUS	Krakatoa x 94-35	NIAB
Zebedee	1545	ww	2002	Limagrain UK Ltd	UK	TG	Claire x Nelson	SACA
Zodiac	810	ww	1990	Limagrain UK Ltd	UK	GEDIFLUX	Hammer x Parade	SACA

CHAPTER III. YELLOW RUST HISTORICAL RESISTANCE PHENOTYPES

1 INTRODUCTION

To study resistance to wheat yellow rust in elite UK wheat germplasm, the first step was to collect historical resistance data. These historical data consisted of evaluations of varietal resistance to contemporaneous wheat yellow rust isolates conducted between 1990 and 2010. They came primarily from the UK Cereal Pathogen Virulence Survey (UKCPVS), laboratory long-term project which describes and monitors virulences in the yellow rust population and evaluates the resistance of wheat varieties on the UK Recommended List against emerging yellow rust isolates. UKCPVS evaluations were complemented by official variety testing conducted in the context of national and recommended list trials. These trials are carried out each year to determine which new candidate varieties can be registered in the Food and Environment Research Agency (FERA) National list and the Home Grown Cereals Authority (HGCA) Recommended List in the UK. The historical data allow us to look at resistance to WYR virulence spectra which are no longer represented in current populations.

Chapter III provides an extensive description of the historical resistance data collected and underlines the unique approach taken to analyse the data prior association mapping.

2 NIAB WYR HISTORICAL RESISTANCE DATA

WYR adult plant field resistance evaluations from 1990 to 2010 have been collected. The resistance evaluations originated from the UKCPVS, the National List (NL) and Recommended List (RL) testing. The tests were carried out in the field or in polythene tunnels on adult plants. Each trial was inoculated with a single Pst isolate, the Figure III-1 providing an overview of the organisation of the WYR testing in the UK from which the historical data collected originate.

As the virulence spectrum of the specific isolates used was critical to the interpretation of the adult plant phenotypes, we also collected seedling test results from the UKCPVS defining the virulence spectrum of Pst isolates used in the adult plants tests. Typically, these seedling tests comprised reaction of a range of differential hosts and known varieties to a given isolate.

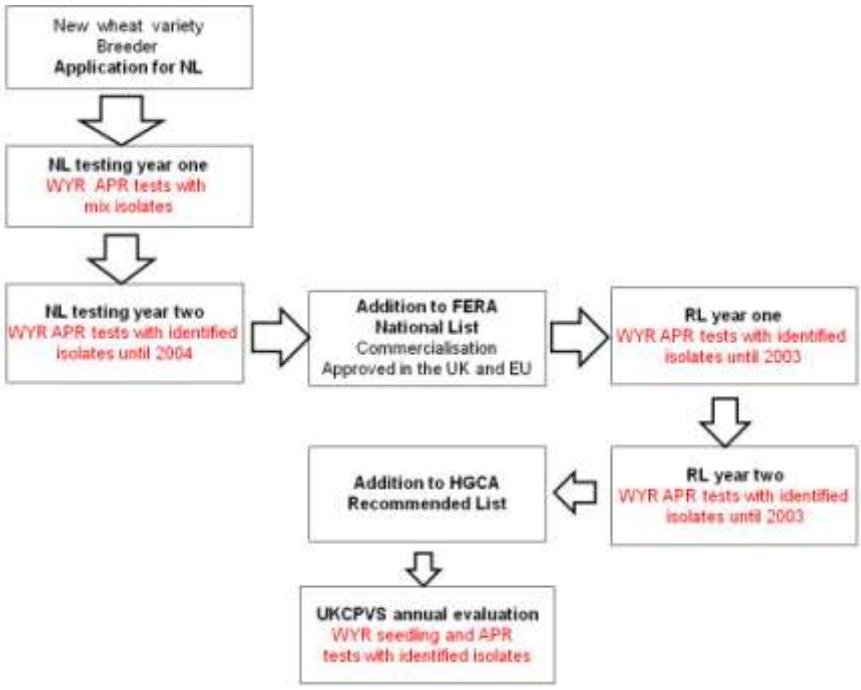

Figure III-1: Organization of the WYR resistance evaluation of wheat varieties in the UK

2.1 UKCPVS Adult plant resistance data

The UKCPVS adult plant resistance (APR) data consists of an annually updated survey of resistance of wheat and barley varieties from the HGCA recommended list. Wheat APR data have been collated from 1990 to 2010. From 1990 to 1993, only the trial means for percentage infection have been retrieved. From 1994 to 2010, plot-level data for the APR test were obtained.

The APR tests from the UKCPVS are inoculated tests with Pst isolates selected from isolates received by the survey over the previous season. The tests including two replicates are sown in tussock plots and inoculated in early spring with a single Pst isolate. The disease severity is recorded as percent leaf area infected using the International Scale (Table III-1) described by Zadoks (1961). The trial is assessed several times during May and June, the timing of the epidemic peak and therefore of scoring depending on environmental influences. All useable scores are selected and averaged to represent the resistance score of each plot. Up to 2001, the UKCPVS trials were conducted under polythene tunnels. From 2002, the trials are conducted in field isolation nurseries each trial with a single Pst isolate being surrounded by one meter high wind breaks as well as being isolated from each other by a

large area of rye. A summary of the UKCPVS trials is published each year in the UKCPVS annual reports.

2.2 National List and Recommended List data

The APR test data from the UKCPVS were complemented with previously unpublished single isolate APR data from National List (NL) trials from 1990 to 2003 and Recommended List trials from 1990 to 2004. Plot data were recovered for trials from 1994 onward. Prior to 1994, only trial means (expressed as percent leaf area infected) were available.

WYR evaluation for NL and RL testing consists of inoculated trials with two replicates. The trials were conducted either in tussock plots for oldest trials or in drilled plots of two one meter row for the most recent trials. The severity of the disease was recorded several time during the epidemic using the international cereal yellow rust key (Table III-1) published by Zadoks (1961). Typically, two to three scores were selected and averaged to represent the resistance score of each plot.

Table III-1: International scale used to score the infection severity in WYR UKCPVS and NL/RL trials

(adapted from Zadoks,1961)

Percent of infection	Visual observation
0	No infection observed
0.1	One stripe per tiller
1	Two stripes per leaf
5	Most tillers infected but some top leaves uninfected
10	All leaves infected but leaves appear green overall
25	Leaves appear half infected and half green
50	Leaves appear more infected than green
75	Very little green tissue left
100	Leaves dead, no green tissue left

2.3 UKCPVS seedling tests

Most Pst isolates used in APR tests have been characterised for the presence or absence of virulence to a series of known (though not necessarily named) resistance genes by means of inoculated tests of a differential set of varieties at seedling stage. Seedling tests from 1987 to 2010, covering the testing period of these isolates, have been obtained from the UKCPVS and NIAB archives. They provide information defining the virulence profile or pathotype for each isolate.

The seedling tests collected are primarily virulence tests based on WYR differential hosts; they are carried out on isolates received by the UKCPVS. Additional seedling tests on recommended varieties were also collected when available. All the seedling tests have been done by the UKCPVS in spore-proof facilities. 10 seedlings of each variety were inoculated with specific Pst isolates and approximately 14 days later, the disease infection type was scored on individual first leaves using a 6 points scale based on Gassner and Straib (1932), published by Zadoks (1961) . The final score recorded is an average infection type calculated from 10 individual leaves score using numerical equivalence (Table III-2).

Table III-2 : WYR seedling infection key used by UKCPVS

Infection type	Description symptoms	Average weighting
i	No observable reaction	-
0	Area of chlorosis /necrosis, No pustulation	0
I	Very few pustules of low spore production with chlorosis /necrosis	1
II	Pustules of low spore production with or without chlorosis /necrosis	2
III	Pustules of high spore production with chlorosis	3
IV	Pustules of high spore production without chlorosis	4

The first system of pathotype description for WYR based on host differential series was proposed by Gassner and Straib (1932). A similar system based on the proposal of Johnson et al. (1972) continues to be use nowadays by the different surveys worldwide (McIntosh et al., 1995). However the composition of differential hosts series in use varies greatly over time and from place to place. The pathotype description is based on the response of a set of differential hosts inoculated with a single Pst isolate. The differentials are selected to carry single or limited resistance genes.

To test the virulence of UK Pst isolates, the UKCPVS uses a set of differential host varieties, including a common core of differentials as described by Johnson et al. (1972), with a number of additions and subtractions made over the past three decades to reflect to local virulence changes and the introduction of novel *Yr* genes.

2.4 Historical data database and organisation

To search the historical data collected depending on the isolate pathotype, a relational database between the resistance phenotype and the pathogen isolate virulence profile has been created. The relational database improves the mining of the historic data and helps to select subsets of phenotypes for subsequent association analyses. Furthermore the database includes information collected on wheat varieties.

The data collected have been organised in five tables, as describes below:

- The “VARIETY” table describes the wheat varieties and gathers information collected during the project (pedigree, genotype, breeder, Yr gene…).
- The “APT mean scores” table contains the average Yellow rust scores for varieties within an adult plant trial.
- The “SDT Scores” table contains the yellow rust score from seedling tests.
- The “APT Description and Link SDT” table describes each adult plant test (year, layout, WYR isolate, source data) and gives a reference seedling test for the pathotype definition.
- The “SDT Description and Pathotype” table describes each seedling test collected (year, WYR isolate, source data) as well as the pathotype description of the isolate for the virulence factor selected.

The five tables have been uploaded to a Microsoft Office *Access*dB and logical relationships implemented as illustrated in Figure III-2. Table I-1 summarizes the different fields used in the database. This organization permits the design of logical queries, for instance to select APT data inoculated with isolates containing specific virulence factors.

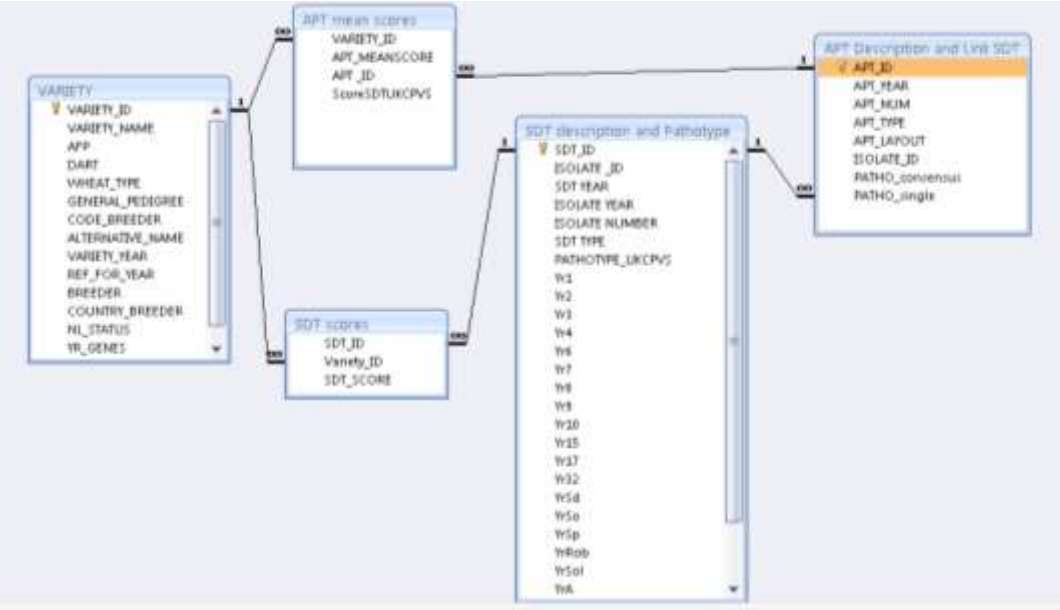

Figure III-2 : Schematic of WYR SDT-APT NIAB Database from Microsoft Office *Access* including relationships between tables.

Table III-3 : Description of WYR SDT-APT NIAB Database fields

Fields	Description
AFP	*AFP number, a unique numerical code given to a variety upon submission of an application for plant breeders rights*
ALTERNATIVE_NAME	*Alternative names known for a variety*
APT_ID	*Unique identifier for each APT*
APT_LAYOUT	*Trial layout (field isolation, polythene tunnel or spreader bed)*
APT_MEANSCORE	*Average percent yellow rust severity for each variety within an APT*
APT_NUM	*Number given to APT the year it was conducted*
APT_TYPE	*Origin of the APT (UKCVPS, National List trial or Recommended List trial)*
APT_YEAR	*Year of APT*
BREEDER	*Name of the breeder*
CODE_BREEDER	*Original code for the variety given by the breeder*
COUNTRY_BRREDER	*Country of the breeder*
DART	*Availability of DArT genotype data at NIAB (Yes or No)*
GENERAL_PEDIGREE	*Pedigree collected*
ISOLATE_ID	*WYR isolate identifier*
ISOLATE_NUMBER	*Number given to WYR isolate in order of reception each year*
ISOLATE_YEAR	*Year of collection of the WYR isolate*
NL_STATUS	*Status of the variety with respect to the 2010 NL*
PATHO_consensus	*SDT identifier for a consensus pathotype*
PATHO_single	*SDT identifier for a single virulence test*
PATHOTYPE_UKCPVS	*Pathotype defined by the UKCPVS and published in UKCPVS reports*
REF_FOR_YEAR	*Reference used for the VARIETY_YEAR (SACA, NIAB, USDA, genbank.vurv.cz)*
SDT_ID	*SDT identifier, unique.*
SDT_SCORE	*Score SDT using 0 to 4 scale*
STD_SCORE_UKCPVS	*Score SDT done in parallel to the UKCPVS APT if available*
SDT_TYPE	*Type of SDT (virulence test or resistance evaluation)*
SDT_YEAR	*Year of SDT*
VARIETY_NAME	*Variety common name*
VARIETY_ID	*Variety identifier, unique.*
VARIETY_YEAR	*Year plant breeder right application or first year of commercialisation of a variety*
WHEAT_TYPE	*Seasonal type of a variety (winter wheat, spring wheat or unknown)*
Yr1 to *YrA*	*Presence of a specific virulence factor, for instance for Yr1 "1" means presence of virulence factor vir1," no1" means absence of vir1,"?1"means presence of Vir1 indeterminate.*
YR_GENES	*Yr genes known and suspected to be present in a variety based on literature*

3 DATA ANALYSIS

3.1 APR data analysis

Prior to analysis, APR data were validated to remove duplicate records. The historical APR data collected came from artificially inoculated trials with selected Pst isolates. For UKCPVS tests, Pst isolates are selected every year to represent the most recent emerging pathotypes, but they do not fully represent the natural virulence frequencies present within the UK Pst population. Some pathotypes will be overrepresented while others will be underestimated. While Pst isolates used in NL/RL trials can be considered representatives of the most predominant pathotypes at the time of testing, only a limited number of varieties have been repeatedly evaluated over the years. NL/RL trials therefore provide a largely incomplete variety-year matrix which does not allow investigation of the evolution of variety performance over time. The limited number of varieties tested in any

given year also precluded conducting meaningful association scans using datasets from an individual isolate-year APT and drove us instead to aggregating data over tests and years.

To derive means from a gappy matrix with common controls, a variety of REML corrective models as implemented in GenStat 13th (Payne et al., 2009), were tested in order to obtain a Best Linear Unbiased Predictor (BLUP) for each variety and dataset considered. The adjusted means from those models (grand mean +BLUP) will be used directly in the association mapping analysis. The adjusted mean is an estimation of the intrinsic resistance value of each line.

Because of computational constraints (power and memory), the analysis have been done in two steps.

To start, a simplified model including fixed effects was used in order to evaluate the effect of replicate within trial. Secondly, a more complex model was fitted to obtain an adjusted mean for each variety representative of the genetic variation while controlling other sources of variation e.g. year, layout, isolates. The objective of the model here is not to investigate the biological background of the variations observed but only to obtain an adjusted mean for each variety suitable for association mapping.

To study the replication effect within trial, plot data from 1994 to 2010 were analysed using REML. The following model was tested:

(III.1)

$$y_{ijk} = \mu + V_i + T_j + B_{jk} + e_{ijk}$$

y_{ijk} is the historical WYR severity of the variety i, in the trial j, in block k;μ is the overall mean; V_i is the effect of the ith variety; T_j is the effect of the trial j (isolate effect is confounded with trial effect here); B_{jk} is the effect of the kth bloc within the trial j ; e_{ijk} is the residual. Varieties, trials and block were treated as fixed factors. For the analyses, severity data are transformed using log(x+1) to obtain a near normal distribution.

To obtain an overall adjusted mean for each variety, a model including all two-way interactions was fitted; three-way interactions were not included because of computational limits. As we were constrained on power and memory, all components of the model were fitted as Random. Thus, in presence of lot of missing data, the Bayesian approach seems to be most appropriate as means are weighed and adjusted depending of the data available. In other words, if only one or two scores are available for a variety, the BLUP will tend to be closer to zero in order to reflect the uncertainty of the evaluation. On the other hand, if many

data are available, the uncertainty decreases and the estimations will be kept further from zero as the confidence on the estimation is higher. Model (III.2) is used as a starting point, it is then adapted to each subset of data studied by dropping terms that do not improve significantly the model. Terms are dropped individually based on a $\chi 2$ test on the deviances between two models.

(III.2)

$$y_{ilmn} = \mu + V_i + R_l + L_m + A_n + VR_{il} + VL_{im} + VA_{in} + RL_{lm} + RA_{ln} + LA_{mn} + e_{ilmn}$$

y_{ilmn} is the historical WYR severity of the Variety i, tested against isolate l, in a trial with a layout m; in year n; μ is the overall mean; V_i is the effect of the ith variety; R_l is the effect of the rust isolate l; L_m is the effect of the layout m; A_n is the effect of the year n; VR_{il}, VL_{im}, VA_{in}, RL_{lm}, RA_{ln}, LA_{mn} are all two way interaction between main effects; e_{ilmn} is the residual. All components were treated as random factors. For the analyses, severity data are transformed using log(x+1) to obtain a near normal distribution.

3.2 Seedling test analysis: determination of virulence factors

Based on the seedling data collected, the virulence of the isolates used in APR tests was investigated. Ongoing evolution of the differential series used by the UKCPVS (Table III-4) means that the full description of virulence spectrum against all differential hosts ever used contains some missing data. Therefore the pathotype of Pst isolates for the purposes of this study was described in terms of virulence of the common core of differential varieties for which a complete dataset was available (Table III-4). For example, to determine if an isolate has the virulence factor *vir3* against *Yr3a* and *Yr4a*, the responses to Vilmorin 23, Nord Desprez and Cappelle Desprez were examined in this order of preference.

Isolate virulence spectra are given based on 12 core *Yr* genes (*Yr1, Yr2, Yr3a+Yr4a, Yr3b+Yr4b, Yr6, Y7, Yr8, Yr9, Yr10, Yr15, Yr17, Yr32*). Virulence for Avocet R, Strubes Dickkopf, Suwon 92/Omar and Spalding prolific were also included to define pathotypes despite a limited number of testing years as they are used in international surveys (Chen, 2005; De Vallavieille-Pope and Line, 1990). Finally, the virulence response at seedling stage against four UK wheat varieties, Solstice, Robigus, Claire and Cadenza, were added to the virulence definition.

The presence of specific virulence factors was determined based on the scores in seedling tests as follows: a. Present if SDT score ≥ 3, b. Absent if score SDT ≤ 2, c. Indeterminate if 2 < SDT score > 3. When two or more seedling tests for a single isolate were available a consensus virulence profile was decided, and inconsistencies between two tests for a specific virulence were shown as undetermined.

Table III-4 : Virulence factors describing the Pst isolates and differentials used

(a) *Yr* genes referring to (Chen and Line, 1992), (Chen and Line, 1993b), (Bayles, 2001),(Boshoff et al., 2002), (Eriksen et al., 2004), (Chen, 2007), (Hovmøller, 2007) and (Lin and Chen, 2009).

			Years differential used in virulence test between 1990 and 2010																					
Virulence factor	**Yr gene associated**	**Differentials associated with known Yr genes in parentheses (a)**	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010
Vir1	*Yr1*	Chinese 166 (*Yr1*)	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x
Vir2	*Yr2*	Kalyasona (*Yr2*)									x	x	x	x	x	x	x	x	x	x	x	x	x
		Heines VII (*Yr2,Yr25,YrHVII*)	x	x	x	x	x	x	x	x													
Vir3	*Yr3a +Yr4a*	Vilmorin (*Yr3a, Yr4a*)									x	x	x	x	x	x	x	x	x	x	x	x	x
		Nord Desprez (*Yr3a, Yr4a+*)									x	x	x	x	x	x	x	x	x	x	x	x	x
		Cappelle Desprez (*Yr3a, Yr4a, APR Yr16*)	x	x	x	x	x	x	x	x													
Vir4	*Yr3b+Yr4b*	Hybrid 46 (*Yr3b, Yr4b*)	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x
Vir6	*Yr6*	Heines Kolben (*Yr2, Yr6*)	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x
		Heines Peko (*Yr2, Yr6, Yr25*)											x	x	x	x	x	x	x	x	x	x	x
Vir7	*Yr7*	Lee (*Yr7,Yr22,Yr23*)	x	x	x	x	x				x	x	x	x	x	x	x	x	x	x	x	x	x
		Brock (*Yr7, APR Yr14*)	x	x						x	x	x	x	x			x	x	x	x		x	x
		Reischerberg 42 (*Yr7, Yr25*)									x				x	x	x	x	x	x			
		Tommy (*Yr7*)	x					x	x	x													
Vir8	*Yr8*	Compair (*Yr8, Yr19*)							x	x	x	x	x	x	x	x	x	x	x	x	x		x
Vir9	*Yr9*	Kavkaz x 4 Federartion (*Yr9*)		x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x
		Clement (*Yr2, Yr9, Yr25, YrCle*)	x	x						x					x	x	x	x	x	x	x	x	x
Vir10	*Yr10*	Moro (*Yr10, YrMor*)											x	x	x	x	x	x					
Vir15	*Yr15*	Yr15 Avocet NIL (*Yr15*)											x	x	x	x	x	x	x	x	x	x	x
Vir17	*Yr17*	VPM1 (*Yr17*)									x	x	x	x	x	x	x	x	x	x	x		x
		Rendezvous (*Yr17*)	x	x	x	x		x	x	x					x	x	x	x	x	x		x	
		Reaper (*Yr17*)							x	x	x	x	x					x	x	x	x	x	
		Brigadier (*Yr9, Yr17*)			x	x	x	x	x	x	x	x			x	x	x	x	x	x	x	x	x
Vir32	*Yr32*	Carstens V (*Yr32, Yr25*)			x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x
VirA	*YrA*	Avocet R (*YrA*)									x		x	x	x	x							
VirSd	*YrSd*	Strubes Dickkopf (*YrSd,Yr25*)									x		x	x	x	x							
VirSo	*YrSo*	Suwon92/Omar (Y*rSo*)									x		x	x	x	x							
VirSP	*YrSP*	Spalding Prolific (*YrSP*)									x		x	x	x	x	x	x	x				
VirRob	-	Robigus													x	x	x	x	x	x	x	x	x
VirSol	-	Solstice												x	x	x	x	x	x	x	x	x	x
VirClaire	-	Claire														x	x	x	x	x	x	x	x
VirCad	-	Cadenza				x	x	x	x	x	x		x	x	x	x	x	x	x	x	x	x	x

The seedling tests collected represent only the highly selected isolates used in APR tests. Therefore, although they are all derived from the natural UK Pst population, virulence frequencies in this set of isolates do not reflect natural virulence frequencies. They are simply used to identify the virulence profile of isolates used in APR tests.

Based on the pathotype as defined above, isolates whose pathotypes all match certain criteria will be grouped and APR tests including those grouped isolates jointly analysed. This grouping of APRs is key to the task of association mapping using historical APR phenotype data as a typical APR test involves inoculation of an average of 52 varieties - a number too low to give any statistical power - whereas the sum of varieties inoculated with isolates matching a given criterion – for instance "virulent on *Yr1, Yr2, Yr3a+Yr4a, Yr3b+Yr4b, Yr6*" (to take one such example) – may return results for a majority of varieties in the panel. This presumed signal increase due to increase in panel size may be eroded by the introduction of greater error in adjusted APR means calculated from grouped tests. This

will be discussed in relation to actual results obtained in Chapter VI. For the purposes of this chapter, the analysis of seedling tests is mainly descriptive and aims to identify subsets of data representing a suitable number of varieties for association mapping.

4 RESULTS

4.1 Adult plant test data: descriptive analyse

Data from a total of 313 WYR adult plant tests were collected, covering 245 UKCPVS and 73 NL/RL trials from 1990 to 2010 (Table III-5), which together represent 20,932 data points. On average, 64 lines were tested each year in NL/RL trials and 40 each year in UKCPVS trials. The distribution of the raw severity scores ranges from 0% to 72.5% and is skewed toward low severity scores (Figure III-5). Two thirds of the scores were less than 5% severity. In 4162 of the 12,906 variety-trial combinations, no symptom of rust (Severity= 0%) was observed. This is not surprising for two reasons. Firstly, the tested wheat lines are potential commercial varieties (proposed for National listing or recently commercialized), so it is to be expected that their level of resistance to be fairly high. Secondly, in keeping with its mission as a forward-looking virulence monitoring study, UKCPVSs pathotype selections often include isolates with an unusual virulence spectrum which (at the time of isolation and testing) may only be virulent of a small number of cultivars.

The complete dataset represents 574 lines evaluated in at least one year against Pst. A total of 310 of those lines were included in the YR Panel.

Table III-5 : Summary APR data collected between 1990 and 2010.

Observations related to lines in the YR panel are indicated in parentheses. (a) Infection levels from 2001 UKCPVS trials were not included as the WYR epidemics within those trials were too low; (b) Starting in 2004 the RL evaluations were carried out using isolate mixtures instead of single isolates, so RL trials were only collected until 2003; (c) in a similar manner, NL trials were only collected until 2004 due to the introduction of isolate mixtures into NL evaluations starting in 2005.

	Lines tested	Total observations	Trials	Pst isolates		
				UKCPVS	NLRL	Total
1990	125 (93)	1212 (1053)	29	22	5	24
1991	75 (69)	669 (639)	20	11	5	11
1992	71 (66)	803 (778)	25	16	5	17
1993	86 (63)	816 (678)	22	16	6	19
1994	85 (78)	870 (828)	24	18	6	23
1995	83 (69)	714 (630)	20	14	6	20
1996	98 (81)	947 (846)	24	18	6	23
1997	117 (95)	585 (475)	10	4	4	8
1998	96 (80)	711 (631)	17	11	5	16
1999	102 (84)	516 (455)	13	10	3	12
2000	95 (83)	620 (564)	16	12	4	16
2001(a)	65 (52)	260 (208)	4	0	4	4
2002	118 (101)	805 (720)	20	10	5	14
2003	143 (125)	992 (920)	18	10	4	13
2004(b)	94 (61)	610 (478)	14	10	4	14
2005(c)	52 (52)	520 (520)	10	10	0	10
2006	49 (49)	343 (343)	7	7	0	7
2007	41 (41)	205 (205)	5	5	0	5
2008	39 (39)	117 (117)	3	3	0	3
2009	44 (38)	220 (190)	5	5	0	5
2010	53 (30)	371 (210)	7	7	0	7

Each trial has been inoculated with a single Pst isolate. Of the 313 trials, only a limited number have been carried using the same isolate as a total of 202 Pst isolates have been used over the years (Figure III-3). The 202 isolates used in APR tests have been isolated between 1987 and 2009; however each year is not represented equally (Table III-6). 71% of the isolates originated from samples received by the UKCPVS between 1989 and 1999, while 27% represent samples from 2000 to 2009.

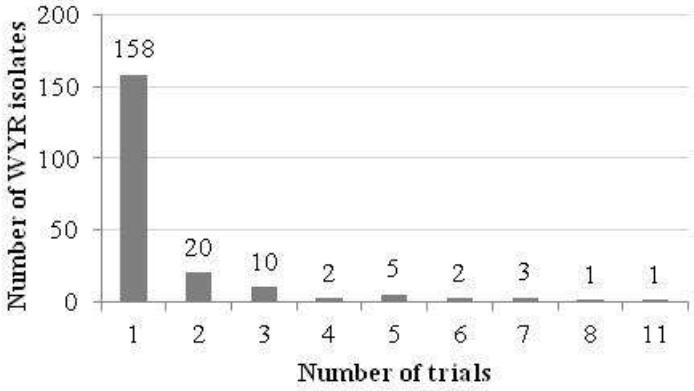

Figure III-3: Frequency of Pst isolates uses

Table III-6: Year of isolations of the 202 Pst isolates used in APR tests

	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
Number of isolates	1	4	17	12	14	14	16	15	18	4	11	10	12	2	7	9	5	6	5	5	3	5	7

The evaluation of lines against different virulence factors does not allow a simple compilation of data for analysis. Depending of the isolate, contradictory resistance levels are observed. The phenomenon is illustrated in Figure III-4 by cultivar Brock, which appears susceptible and highly resistant within the same year of testing particularly in 1998 and 2003 in NL/RL trials. Those observations emphasise the importance to gather the data based on the virulence profile.

Additionally, Figure III-4 illustrated a strong trial effect within year due to the inclusion in the same year of isolates virulent on wider and narrower spectra of lines.

Despite the confounding isolate effect, Figure III-4 suggest also the presence of year effect, for instance 1993 and 1999 trial means show a low level of WYR infection, while 1994 and 2002 show higher rust severity scores. Large annual fluctuations can be observed in WYR severity score, as rust infection is highly influenced by climatic conditions (temperature, hygrometry). For instance Bayles et al. (2002) explained the low level of WYR infection in 2000/2001 season by the combination of a wet autumn, severe winter and wet spring. In our dataset, the yearly environmental effect on WYR severity is confounded with isolate and variety effects, as each year is represented by a different set of varieties and Pst isolates.

Moreover, the date of scoring selected each year will affect the percent of infection recorded; later assessments are most likely to show higher levels of infection as the WYR infection is not static but progresses throughout the growing season until the total senescence of the plants.

Finally, the layout of the trials (in spreader beds or tussock plot, in the field or under polythene tunnels) may influence the severity scores. The different experiment layouts affect the spread of the disease, first because of spatial organization plots and spreaders, secondly because of the micro-climatic conditions created.

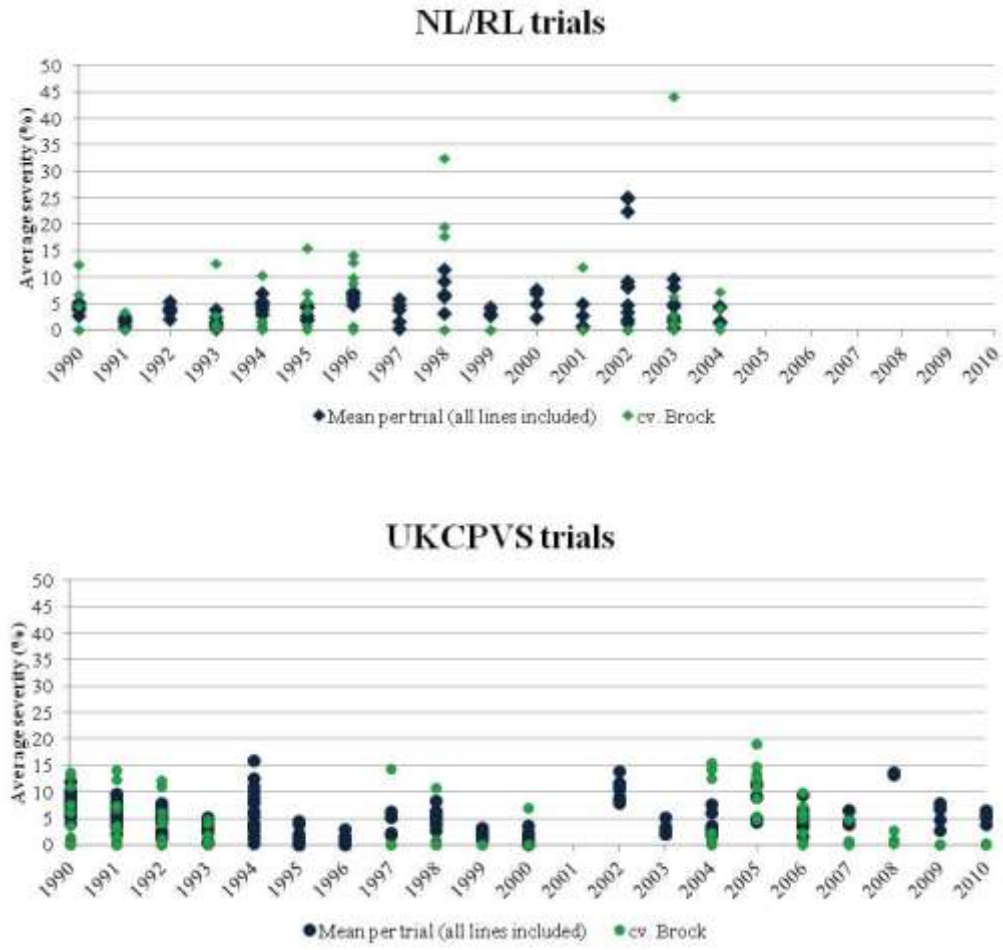

Figure III-4: Average WYR severity in trials collected between 1990 and 2010 and severity observed on cv. Brock

4.2 Replicate effect within APR trial

The analysis using the model (III.1) described previously showed no significant effect of the replications within trial for data collected from 1994 to 2010 (P-value from Wald statistic =1), the relevant output from Genstat is available in Appendix 3.

In the absence of a replicate effect, henceforth, all analyses will be done on means of varieties from each trial. Analysing the means instead of the plot data has the advantage of bringing the dataset from 20,932 to more manageable 12,906 data points, in regards to computational power and memory required.

4.3 Estimation of line resistance value from APR data

The descriptive analysis of APR data demonstrated the major environmental effects that can affect the evaluation of the resistance of varieties and that the mean infection level obtained for each variety depended on the isolates, the year (climatic conditions, date of scoring) and the layout of the test. Therefore, to obtain a single adjusted mean value per variety, model (III.2) was fitted to the complete dataset.

The term Isolate (R_l) and Layout (L_m) did not significantly improve model (III.2) based on $\chi 2$ test on deviance difference, and so the model was simplified as follows:

$$y_{ilmn} = \mu + V_i + A_n + VR_{il} + VL_{im} + VA_{in} + RL_{lm} + RA_{ln} + LA_{mn} + e_{ilmn}$$

(Genstat output is available in Appendix 3)

Adjusted means obtained presented a smoother distribution comparing to the raw data (Figure III-5), they varied between 0 and 17.6%. 44% of the YR panel lines are represented by adjusted severity less than 1% which corresponds to a high level of resistance, most probably explained by the presence of major *R*-genes.

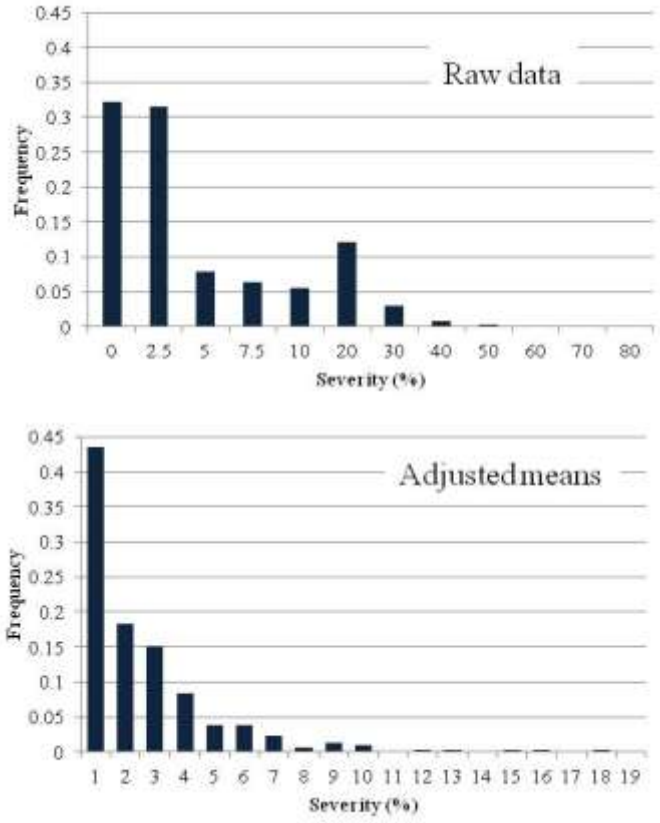

Figure III-5: Distributions of all historical rust severity scores prior correction including 12,906 data points (raw data) and distribution of rust severity score for each cv tested after statistical adjustment integrating the year, the isolate and the layout effects (adjusted means)

By comparing the adjusted resistance value obtained from the fitted model with the HGCA WYR resistance scores published for varieties in the RL (Figure III-6), a moderate correlation was observed (r^2=0.57). Thus, it demonstrates the usefulness of our model to provide a meaningful resistance value for each variety. A reasonably good correlation was expected as the HGCA ratings are derived partly from NIAB inoculated trials results.

Among the varieties highly resistant based on our adjusted score figure many varieties rated resistant (score 7 to 9) by HGCA when last registered on the recommended list: Xi19, Malacca, Deben, Option, Gatsby, Zebedee, Alchemy, Claire and others, a complete list is available in Appendix 4. However a few lines were scored 5 and 4 by HGCA like Solstice, Duxford, Tonic, Mega and Armada. The discrepancies between our resistance estimation and HGCA can be explained in the case of Solstice and Duxford by the very recent downgrading of these varieties by 4-5 points due to the emergence of the 'Solstice' race, which was only represented in 2009 and 2010 historical phenotype data. The varieties Armada and Mega are known to carry the APR gene *Yr12*, the virulence for *Yr12* was frequent when those two varieties were scored by HGCA in the 1970s and 1980s, since the

virulence for *Yr12* seems to have disappeared (Rosemary Bayles personal communication) therefore the recent evaluations showed a higher level of resistance in those varieties.

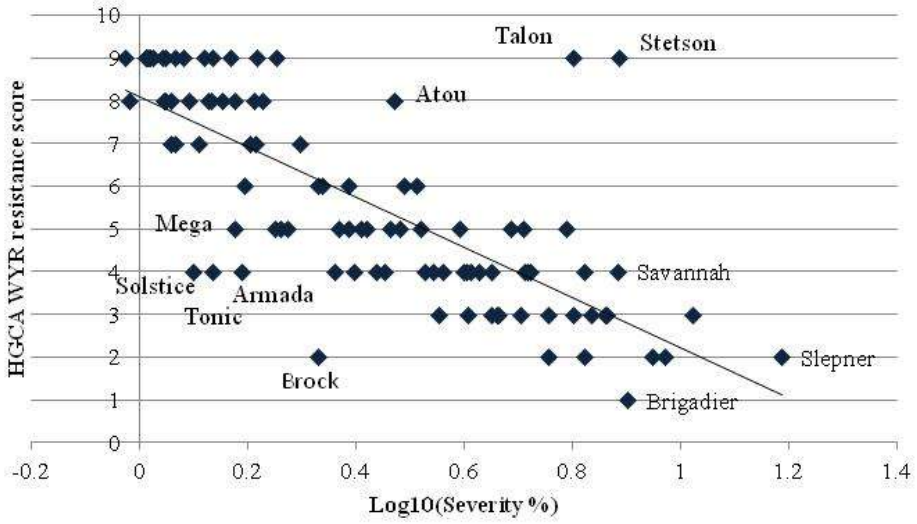

Figure III-6: Correlation between adjusted rust severity and HGCA score for varieties from the recommended list.

The trend line in black has for equation y= 5.8653 x + 8.0757

A corresponding discrepancy is seen at the other end of the scale, whereby varieties such as Stetson and Talon which show highly susceptible adjusted mean severities had a last score of 9 on the HGCA scale. However, those scores were given prior 1992 and do not take into account data generated using isolates which overcame resistance in these varieties during 1992-2010.

Among the most susceptible lines are known susceptible lines without *Yr* genes such as Vuka but also varieties with specific resistance genes that have been overcome by specific UK isolates such Clement (*Yr2*,*Yr9*), Slejpner (*Yr9*), Brigadier (*Yr9, Yr17*), Ritmo (*Yr1*), Savannah (*Yr1,2,3,4,9,17*), Thatcher (*Yr7*).

4.4 Seedling tests: descriptive data analysis

302 seedling tests have been gathered for the 202 Pst isolates used in adult plant tests. For most of the isolates (141), only one seedling test was available. For remaining 61 isolates, two or more tests were available which led to the determination of a consensus pathotype, however many inconsistencies were observed between repeated tests. Where contradictory infection types were observed, the specific virulence factor was noted as "Undetermined".

Table III-7: Virulence factor observed from seedling tests within the 202 Pst

isolates used in historical trials, based on consensus profile.

(a) The presence of virulence factor is stated as undetermined in absence of virulence testing, infection type between 2 and 3 or inconsistency between two seedling tests.

	Isolates with virulence factors		
	Present	Absent	Undetermined(a)
Vir1	179	17	6
Vir2	187	7	8
Vir3	195	1	6
Vir4	154	33	15
Vir6	104	83	15
Vir7	24	158	20
Vir8	0	112	90
Vir9	162	27	13
Vir10	0	39	163
Vir15	0	61	141
Vir17	125	53	24
Vir32	77	78	47
VirA	31	6	165
VirSd	41	2	159
VirSo	31	13	158
VirSP	0	54	148
VirRob	15	37	150
VirSol	11	43	148
VirClaire	41	41	120
VirCad	6	130	66

A large portion of the isolates were described as undetermined for several virulence factors particularly for *vir8* (differential host cv. Compair *Yr8+Yr19*), *vir10* (differential host cv. Moro *Yr10+YrMor*), *vir15* (differential host *Yr15* Avocet NIL *Yr15*), *virA* (differential host Avocet R *YrA*), *virSd* (differential host Strubes Dickkopf *YrSd+Yr25*), *virSo* (differential host Suwon92/Omar *YrSo*), and *virSp* (differential host Spalding Prolific *YrSP*), as the corresponding differential hosts have not been consistently tested over the past 20 years (Table III-7). The virulence factors *vir1, vir2, vir3, vir4, vir6, vir7* and *vir9* present a more consistent data coverage over the past decades. Generally, the isolates present a high frequency of *vir1* (88.6%), *vir2* (92.6%), *vir3* (96.5%), *vir4* (76.2%) and *vir9* (80.2%). Only 51.1% of the isolates were shown to be virulent for *Yr6* (presence of *vir6*), and 11.9% were virulent on *Yr7* (presence of *vir7*).

61.9% of the isolates were determined to be virulent on *Yr17* (presence of *vir17*), and 26.2% were not virulent on *Yr17* (absence *vir17*). Despite the absence of systematic testing for *Yr17* before 1994, only 11.9% of the isolates are undetermined for *vir17* as we were able to determine the virulence based on the infection response obtained on varieties Rendezvous, Reaper and Brigadier. 38.1% of the isolates were virulent on *Yr32* (presence of *vir32*), 38.6% avirulent (absence of *vir32*) while 23.3% remained undetermined.

Virulence was not detected for the following genes or gene combinations *Yr8+Yr19*, *Yr10+YrMor*, *Yr15* and *YrSP*.

The remaining virulence factors *virA*, *virSd*, *virSo* have been tested systematically only between 1998 and 2003. Thus those virulence factors have been elucidated for only 44 isolates within our dataset. Despite a limited number of tests, the results showed that a large proportion of the UK isolates from late 1990s present virulence for the *YrA*, *YrSd* and *YrSo*.

The virulence of a limited number of isolates has been evaluated against major UK wheat varieties Cadenza, Solstice, Robigus and Claire. From the 202 isolates used in APR tests, 52 have been tested against Robigus, 54 on Solstice, 82 on Claire and 136 on Cadenza. Virulence has been identified at seedling stage in six isolates for Cadenza and 41 isolates for Claire, 15 isolates for Robigus and 11 isolates for Solstice. Although virulence has been identified at the seedling stage, Claire and Cadenza remained resistant in the field against all isolates tested during this study.

Based on the presence/absence of virulence factors *vir1, vir2, vir3, vir4, vir6, vir7, vir9, vir17* and *vir32*, the isolates used in APR tests have been grouped in 37 pathotypes (Table III-8), excluding the 86 isolates where at least one virulence factor was stated as undetermined.

Table III-8: Pathotype of the isolates from historic APR tests

Virulence profile	Number of isolates
1,2,3,4,6,7,9,17,32	1
1,2,3,4,6,7,9,17, --	1
1,2,3,4,6,7,9, --,32	2
1,2,3,4,6,7,-, --,32	1
1,2,3,4,6,-,9,17,32	14
1,2,3,4,6,-,9,17, --	13
1,2,3,4,6,-,9, --,32	5
1,2,3,4,6,-,9, --, --	7
1,2,3,4,6,-,-,17,32	2
1,2,3,4,6,-,-, --,32	1
1,2,3,4,-,7,9,17,32	1
1,2,3,4,-,7,9,17, --	2
1,2,3,4,-,7,9, --, --	1
1,2,3,4,-,-,9,17,32	10
1,2,3,4,-,-,9,17, --	13
1,2,3,4,-,-,9, --,32	2
1,2,3,4,-,-,9, --, --	3
1,2,3,4,-,-,-, --,32	1
1,2,3,-,6,7,9, --, --	1
1,2,3,-,6,-,9,17,32	1
1,2,3,-,6,-,9,17, --	2
1,2,3,-,6,-,9, --,32	1
1,2,3,-,-,-,9,17,32	1
1,2,3,-,-,-,9,17, --	13
1,2,3,-,-,-,9, --,32	1
1,2,3,-,-,-,9, --, --	2
1,2,3,-,-,-,-,17,32	2
1,2,3,-,-,-,-, --,32	2
1,-,3,4,6,-,9, --,32	1
1,-,3,4,6,-,-, --,32	1
-,2,3,4,6,-,9, --,32	1
-,2,3,4,6,-,-,17,32	2
-,2,3,4,-,7,9, --,32	1
-,2,3,4,-,7,9, --, --	1
-,2,3,4,-,-,9, --, --	1
-,-,3,4,6,-,-,17,32	1
-,-,3,4,6,-,-, --,32	1
At least one virulence factor undetermined	86

4.5 Derivation of means based on datasets satisfying different virulence criteria

Using the information collected on Pst isolates, the adult plant dataset was divided into subsets based on different virulence criteria and adjusted means were derived for association mapping. Carrying out association analysis on virulence-based derived means instead of the complete set has several advantages. Firstly some environmental variations can be eliminated from the model such as the isolates effects or pathotype effects to obtain a more reliable estimation of the variety resistance.

Secondly, the QTL detection via association analysis can target specific *Yr* genes by analysing well defined pathotypes. For instance in the case of the pathotype "1,2,3,4,6,no7,9,17,32", association mapping can potentially identify QTL associated with *Yr7* but none should be associated with *Yr1,Yr2, Yr3,Yr4, Yr6, Yr9* and *Yr32*. The signal linked to the presence of *Yr7* should appear more detectable as it is not diluted with the action of the *Yr* genes cited in the pathotype. However, it should be noted that any *Yr* genes or QTL not cited in the pathotypes are also potentially present and detectable.

A number of 80 lines have been chosen as arbitrary minimum to define a subset of data that will be analysed via association mapping. Statistical power to detect genetic associations generally decreases with the number of lines analysed (Long and Langley, 1999).

4.5.1 Derived mean based on isolates

From the complete dataset, 12 isolates which have been tested on more than 80 lines included in the YR panel have been identified (Table III-9). The corresponding trials were used to derive adjusted mean which will be used for association mapping analysis.

Table III-9: Pst isolates tested on more than 80 lines from the YR panel

[(a)] Virulence profile defined by UKCPVS following the reception of the isolate.

Isolate	Virulence profile [(a)]	YR panel lines tested	Number of trials	Year tested
1990-505	1,2,3,4,7	190	11	1991, 1993 to 1998, 2003, 2004
1993-24	1,2,3,4,6	95	5	1994 to 1998
1993-54	1,2,3,4,6,9	95	5	1994 to 1998
1994-519	1,2,3,9,17	137	6	1995 to 2000 (except 1998)
1996-31	3,4,6,CV	122	5	1997, 2003 to 2005
1996-502	1,2,3,6,9,17	174	8	1997 to 2002
1998-28	1,2,3,4,6,9,17	94	4	1999, 2003, 2004
1998-96	1,2,3,4,6,9,17	127	6	1999 to 2002
1998-108	3,4,6,17,CV	101	5	1999 to 2002
2000-41	1,2,3,4,9,17,CV	124	7	2001 to 2004
2002-70	1,2,3,9,CV	93	3	2003 to 2005
2002-84	3,4,CV	93	3	2003 to 2005

Despite having been artificially inoculated with a single isolate, it is important to remember when interpreting data from APR tests and postulating resistance genes in lines that contamination of trials by natural Pst populations cannot be completely excluded. Such contamination by natural Pst populations with virulence for *Yr9* and *Yr17* were obvious based on cv. Brigadier in year 1996, 1997 and 1998, for the isolates 1990-505, 1993-24, 1993-54 and 1994-519. For example, cv. Brigadier inoculated with isolate 1990-505 presents

a severity score of 0% from 1991 to 1995, but in 1996 and 1997, the severity observed were respectively 14.5% and 13.5%. Knowing Brigadier has *Yr9* and *Yr17*, severity over 10% in tests with isolate 1990-505 which does not has virulence for *Yr9* and *Yr17*, implied that some contamination occurred from natural population carrying the virulence combination *Yr9+Yr17*. Some level of natural contamination is likely to occur each year, although this will not always be detectable.

4.5.2 Derived means based on virulence on UK wheat varieties

Based on virulence at seedling stage on three UK wheat varieties, two data subsets have been identified (Table III-10) from which adjusted means have been derived.

Table III-10: subset of data based on virulence to four UK wheat varieties

Virulence profile at seedling stage	Number trials	Isolate number	YR panel lines	Year tested
Vir Robigus/ avir Solstice	15	9	155	2001 to 2005, 2007 to 2010
Vir Claire	76	41	290	1991 to 2006, 2009, 2010

The first subset groups trials based on virulence to Robigus and avirulence on Solstice at seedling stage. Robigus was on the RL between 2003 and 2011 and was first rated 3 then 2 on the HGCA scale (susceptible). Despite its poor resistance rating, Robigus had been shown to be resistant to some Pst isolates from the 2000s at seedling and adult plant stage, from which it can be deduced that Robigus carries an unknown source of seedling resistance and potentially an APR resistance. Solstice was on the RL from 2002 until 2012, its HGCA rating started at 9 and dropped to 4 in 2010 when a new highly virulent Pst race overcame Solstice resistance at adult stage. Little is known about Solstice resistance; however it must include a seedling stage resistance component in addition to an APR component. By investigating trials inoculated with isolates virulent on Robigus and avirulent on Solstice at seedling stage, we aim to reveal QTLs conferring resistance to the "Robigus race", among which may figure QTL overcome by the "Solstice race".

The second subset groups trials based on the virulence on the cultivar Claire. Claire is a resistant variety released in 1999 which maintained an HGCA score of 9 (maximum resistance) until now. It is believed to carry seedling resistances *Yr2*, *Yr3*, *Yr4*, *Yr25* and another unknown seedling resistance gene (Lewis, 2006). In addition Claire was found to exhibit quantitative APR, controlled by four QTLs, one of which corresponds to *Yr16* (Powell, 2010).

4.5.3 Derived means based on pathotype description

Five pathotypes represented by more than 10 isolates have been used to interrogate between 96 and 198 varieties (Table III-11). Adjusted means have been derived for each pathotype to carry association mapping analysis. With those subsets, we will investigate specifically QTL linked to the known resistance gene *Yr3b + Yr4b*, *Yr6*, *Yr7*, *Yr17* and *Yr32*, as well as unknown resistance genes.

Table III-11: Subset of data identified based on main pathotypes

Virulence profile	Number trials	Isolate number	YR panel lines	Year tested
1,2,3,4,6,no7,9,17,32	20	14	189	1992 to 1994,1999 to2002, 2009, 2010
1,2,3,4,6,no7,9,17,no32	15	13	141	1992 to 1994, 2000, 2002, 2004 to 2007, 2009,2010
1,2,3,4,no6,no7,9,17,32	19	10	187	1992 to 1994, 2001 to2005, 2007, 2009, 2010
1,2,3,4,no6,no7,9,17,no32	15	13	92	1996 to 2000
1,2,3,no4,no6,no7,9,17,no32	19	13	166	1995 to 2000, 2006

5 DISCUSSION

5.1 APR dataset and estimation of resistance value

The dataset collected from historical WYR evaluations is extensive. It represents 21 years of adult stage testing so a total of 313 APR trials, including 202 WYR isolates and 574 wheat elite lines. However, the dataset includes many missing data which will influence the estimation of resistance value.

(1) Not all lines have been tested against all isolates. On average a variety has been tested against 17 isolates, because each year new isolates representing the latest virulence are substituted for older isolates in keeping with the main purpose of the UKCPVS and NL testing regime to continuously update evaluation of the WYR epidemic risk in the UK.

(2) All lines have not been tested each year. On average, lines have been in test less than 3 years, the number of year tested depends of course of the date of release of the variety and its relative commercial success and longevity. For instance, Hereward was first released in 1991 and remained on the RL until 2010 therefore 20 years of testing are available. On the other end, Acclaim was tested to enter the NL in 1995 but was not retained, thus only one year of testing is available for this line. Similarly to the highly incomplete Line x Isolate matrix, the incomplete Line x Year matrix limits the accuracy with which the resistance value can be estimated for each variety.

To complement and extend the historical data therefore, all the lines included in the YR Panel have been evaluated at seedling stage and adult stage against current Pst isolates, the result of those evaluations are presented in the next Chapter V.

With these caveats in mind, a resistance estimate of each variety was obtained based on a restricted maximum likelihood (REML) model including year, isolate and layout factors.

5.2 Isolate virulence at seedling stage

The presence of many environmental variations in the historic dataset, highlight the importance of analysing data in subgroups to obtain more reliable estimates of resistance value by removing some variation from the equation.

With this aim, isolates used in each APR trial were identified and the corresponding seedling virulence test results were collected. The data from a total of 302 seedling tests were collected; they cover the 202 Pst isolates used in APR tests.

5.2.1 Limitation to the definition of virulence profile

The virulence evaluations did not include the identical set of differential hosts each year, making difficult to obtain an extended virulence profile common to each isolate. The UKCPVS differential set is adapted each year (1) to represent recently discover resistance, (2) to use a more reliable or commonly used differential. For instance, Cadenza and Solstice were added respectively in 1993 and 2002 to the UKCPVS differential set as they were highly resistant to contemporary Pst races, although their source of resistance are still unknown. Some virulence factors were tested for only a short period of time such as *vir8, vir10, vir15, virA, virSd and virSu*. Other virulence factors were evaluated based on several differential hosts; it is the case for *Yr2, Yr3, Yr6, Yr7, Yr9* and *Yr17*. For instance, virulence for *Yr2* was tested based on Heines VII until 1997, it was then replaced by Kalyasona as another resistance specificity was identified in Heines VII using an isolate from Ecuador (Calonnec et al., 1997); however Heines VII did not differentiate European isolates. In the case of *Yr17*, Rendezvous, Reaper and Brigadier were added to VPM1 as alternative differential hosts, in an attempt to elucidate the virulence profile of more isolates.

This approach is not perfect; there is a great uncertainty about resistance genes in differential set as illustrated by Heines VII. The reliability of definition of pathotypes depends on the extent of knowledge about the genetic resistance in the differential set for each virulence factor. One can never be certain that two hosts used as alternative differentials known to share one or more genes, are not differing in some other unknown genes. Therefore, there is a risk for some isolates to be declared wrongly avirulent for *Yr2,*

Yr3, Yr6, Yr7, Yr9 and *Yr17*. This risk has to be bear in mind when examining the virulence profile defined.

In our attempt to define the virulence profile of isolates used in APR tests, we observed many contradictory infection types between seedling tests, meaning an isolate was found virulent for a specific virulence factor in one test and avirulent in another test. The inconsistencies observed can have different origin: experimental error (isolates and /or differential host misidentified, error in data recording), use of alternative differentials, cross contamination during testing and drift from the original isolate following several multiplication cycles. The UKCPVS isolates are not specifically isolated from a single pustule but directly from leaf samples presenting several pustules. Therefore, multiple Pst clones with distinct virulence can occasionally form a single isolate. Several cycles of multiplication of mixed isolates can lead to the selection of a specific clone with a virulence profile slightly different from the original isolate.

In case of contradictory observations, the isolates was classified as undetermined for the virulence factor considered, thus limiting uncertainty of the virulence definition.

5.2.2 Virulence factors in isolates used in APR tests

The isolates used in APR tests present a high frequency (more than 60%) of virulence factors *vir1, vir2, vir3, vir4, vir9* and *vir17*. A little more than half of the isolates were shown to be virulent for *Yr6* (presence vir6), 38.1% of the isolates were virulent on *Yr32* (presence *vir32*), and 12% were virulent on *Yr7* (presence *vir7*).

Based on UKCPVS reports from 1990 to 2010, the frequency of specific *Yr* virulence within the UK can be followed since 1969 (see Appendix 5). The surveys confirmed the presence of virulence for *Yr1, 2, 3a, 3b, 4a, 4b, 6, 7, 9, 17* and *32* in natural Pst populations over the last two decades.

Pathotypes with virulence for the rust genes *Yr1, Yr2, Yr3, Yr4* and *Yr6* were fairly frequent in the UK during the past three decades and were also well represented in the UKCPVS and NL/RL inoculated tests. Based on UKCPVS reports, virulence for *Yr2* and *Yr3* were present in the quasi totality of isolates since 1977. While the virulence frequency for *Yr1* progressively increased to reach 90% in early 1990s. The frequency of *Yr4* varied between 16% and 100%. Similarly, the virulence for *Yr6* varied greatly, oscillating between 1% and 98% during the last three decades.

Despite its first identification in the mid-1970s, *Yr7* frequency remained low in natural populations in the UK. However isolates carrying *Yr7* virulence were often used in inoculated trials.

Virulence for *Yr9* was first detected in 1981 in the UK, since isolates virulent on *Yr9* has become predominant and has been well represented in inoculated trials. Isolates presenting the virulence for *Yr17* were not detected before 1994 in the UK (Bayles et al., 2000), in conjunction to the cultivation of varieties with *Yr17*, the frequency of *Yr17* virulence rose quickly to reach 100% in 1999.

Virulence for *Yr32* (cv Carstens V) was detected prior to 1946 in the Netherlands (Roelfs et al., 1992) and was likely to be present within the UK around the same period of time as Pst isolates have been shown to migrate between France, Netherland and the UK (Hovmøller et al., 2002). Carstens V was first tested by the UKCPVS in 1994 and virulence for this cultivar was rather high (75%), it then decreased to an almost undetectable level in 1998 before reoccurring at a very high level in 2001. Since then, virulence for *Yr32* has been maintained at a high frequency.

Consistently with UKCPVS, no isolates used in APR tests carry the virulence factor *vir8*, *vir10* or *vir15* but some uncertainty remains as they were not tested systematically over the past 20 years. Additionally pathotypes virulent on *Yr8* and *Yr10* have been found in other west European countries in recent years (eurowheat.org).

No isolates used in APR tests were found to be virulent on Spalding Prolific (*virSp*), which is not surprising since only one UK isolate with this resistance has been detected in 2003. Furthermore the virulence was absent in West European countries until 2011.

Despite a limited number of tests for virulence factors *virA*, *virSd* and *virSo*, the results demonstrated a large part of UK isolates from late 1990s present virulence for the *YrA*, *YrSd* and *YrSo*. Those virulence factors are likely to be present in most UK isolates sampled from the last two decades. Based on EuroWheat database (eurowheat.org), the three virulence factors remained at high frequency in Denmark and France between 1993 and 2011.

The virulence for *Yr5* has not been tested on UK isolates by the UKCPVS since 1984. However it is known that virulence for *Yr5* is rare (Stubbs, 1985), thus it is unlikely to find a UK Pst isolates virulent on *Yr5*. Moreover, no isolate virulent on *Yr5* was detected in other west European countries between 1993 and 2011(eurowheat.org).

Isolates virulent on Cadenza at seedling stage have been detected in the early 1990s but none of them were able to infect significantly Cadenza at adult stage. Cadenza has been evaluated each year since 1990 and has always shown a high level of resistance in the field. Its long lasting resistance includes seedling stage components as well as an adult stage component.

Similarly many isolates virulent on Claire at seedling stage have been identified but none of them affected Claire at adult stage. Since its release in 1999, Claire has been widely

grown nonetheless yearly resistance evaluation show a maximum of 2% of infection. Thus the resistance present in Claire was thought to be durable according to the definition of Roy Johnson (Johnson, 1979). Unfortunately, Claire adult plant resistance was finally overcome in 2011/2012 following the emergence of a new Pst race named the "Warrior race". Prior to this Claire resistance had also broken down in New- Zealand.

15 of 55 isolates tested on Robigus were virulent at seedling stage as well as adult stage. VirRob first evaluated in natural population in 2004 and was present in 31% of the isolates received by the UKCPVS, VirRob increased progressively to reach 100% in 2010. Therefore VirRob is most likely to be present at a high frequency in isolates used in APR tests in the 2000s.

11 isolates tested on Solstice were virulent at seedling stage, however only the isolates post 2007 were also virulent at adult stage confirming the presence of a seedling resistance and an adult plant resistance in Solstice. VirSol was found at a low level in natural population in 2002 and 2003 by the UKCPVS, it then decreased to an undetectable level before quickly rising again and reaching a frequency of 98% in 2010.

5.2.3 Pathotype of isolates used in APR tests

Based on virulence factor *1, 2, 3, 4, 6, 7, 9, 17* and *32*, the isolates used in APR tests were grouped in 37 pathotypes. 86 of the isolates were not classified as the complete virulence profile was not available. The five most frequent pathotypes used in APR tests and chosen for further analysis correspond also to the most frequent pathotypes sampled between 2000 and 2010 in the UK based on EuroWheat database (the figure summarizing the pathotypes frequency from eurowheat.org is available in Appendix 6). Thus the subset of data focusing on those pathotypes will provide a good representation of the WYR virulence present in the UK during the first decade of the XXI century.

CHAPTER IV. YELLOW RUST DE NOVO RESISTANCE PHENOTYPES

1 INTRODUCTION

To obtain an updated view of the WYR resistances present in the YR panel and to highlight resistance genes effective against current Pst isolates in the UK, the YR panel was assessed for seedling resistance in controlled environment tests and adult plant resistance in field tests with selected Pst UK isolates of recent origin. In parallel to this *de novo* resistance survey of the complete panel, the virulence of isolates 03/7, 08/501 and 08/21 was evaluated on an extended set of host differential varieties. Additionally, using so-called 'diagnostic' markers, the presence of genes *Yr5, Yr9* and *Yr17* was investigated within the YR panel.

Through *de novo* resistance tests, molecular analysis and extended virulence tests, we wish to discover if the YR panel includes (1) major well characterized seedling resistances e.g. *Yr6, Yr7, Yr9* and *Yr17* (2) uncharacterized seedling resistance effective against the current UK Pst isolates, (3) quantitative adult plant resistance effective against the current UK Pst isolates.

2 MATERIALS AND METHODS

2.1 Plant material: YR panel, differential hosts, reference varieties for WYR resistance

The complete YR panel including 327 varieties was previously described in Chapter II.

An extended set of WYR differential hosts was assembled and used to refine the virulence spectrum description of Pst isolates used in de novo phenotyping.

This extended differential host series comprised thirty commonly used European and American differential hosts complemented with thirty control and reference varieties with unknown *Yr* genes and recently described resistances and thus can be used to evaluate virulence on most of the race-specific *Yr* genes already described in the literature apart from 12 for which we could not obtain seeds from reference cultivars (namely *Yr28, Yr31, Yr35, Yr37, Yr38, Yr40, Yr41, Yr42, Yr45, Yr47, Yr50, Yr51* and *Yr52*). The seeds for the differentials and reference cultivars were provided by the UKCPVS, the USDA small grain collection, Dr Xiaming Chen in University of Washington State (Zak, Express, IDO377s),

Dr Lesley Boyd in John Innes Centre UK(Alcedo, Guardian). The complete list is available in Appendix 7.

In addition, the Pst isolates from the project were evaluated against the WYR near isogenic lines developed by the Plant Breeding Institute (University of Sydney, Australia) in the Avocet susceptible background (Wellings et al., 2004), a list is available in Appendix 7.

2.2 Pathogen, spores production and conservation

2.2.1 Pathogen

Three main Pst isolates, known as the "Solstice" 08/21, "Brock" 03/07 and "Timber" 08/501 WYR isolates were used in seedling and adult plant resistance tests to evaluate the YR panel. Those isolates originated in the UK, having been received by the UKCPVS in 2003 and 2008 and selected for use in National List and Recommended List trials because of their specific combination of virulences. A mixture of those three isolates has been used in 2009, 2010 and 2011 in NL and RL trials. The UKCPVS defined the virulence spectrum of the isolates as described in the Table IV-1.

Table IV-1: Pathotypes of Pst isolates used to evaluate resistance in YR Panel cultivars.

(Adapted from UKCPVS reports 2004 and 2009)

Isolate Code	Origin			*Yr* genes and reference cultivars	
	Year	County	Cultivar	Virulence	Avirulence
08/21	2008	Cambridgeshire	Solstice	1,2,3,4,6,9,17,32,Solstice	7,8,15,Brock,Timber
08/501	2008	Cambridgeshire	Timber	1,2,3,4,6,9,17,Timber	7,8,15,32,Solstice
03/7	2003	Lincolnshire	Brock	1,2,3,4,7, (17), Brock	6,8,9,15,32 ,Solstice, Timber

Freeze-dried, vacuum sealed ampoules of spores were obtained from the UKCPVS. In addition to the re-evaluation of the three main isolates used for de novo phenotyping, to verify the actual virulences present in the inoculated field trials at the time of scoring in case of significant natural infection, a small number of tissue samples were taken each season. From those samples, Pst isolates were retrieved and pathotyped.

2.2.2 Increase of Urediniospores

2.2.2.1 Large scale production

To produce the adequate inocula for the different tests, the spores were multiplied in isolation in a controlled environment on the WYR susceptible cultivar: Victo or the respective susceptible cultivars Solstice, Timber and Brock.

The susceptible cultivar was sown in plastic multi-cells trays (7 x 5 cells of 5 x 5cm), the seeds were arranged in 7 narrow rows. The trays were then placed in a disease free growth chamber (GC) or a glasshouse prior to inoculation. The growth conditions are described in Chapter IV.2.3.

When coleoptiles emerge (GS07 following Zadoks et al. (1974) decimal code for growth stage), a maleic hydrazide solution at 0.1 g/l (Sigma-Aldrich) was applied to the soil surface, at the level of one litre per tray. Maleic hydrazide permits the first foliar leaf to develop and remain erect whilst suppressing growth of secondary leaves thus prolonging sporulation (Rowell, 1984).

When the seedling reached GS11 (first leaf fully expanded), each seedling tray was placed in a polythene bag, inoculated with a spore/talc mixture (1 part spores, 19 part talc) and incubated following the method described in Chapter IV.2.3. Each Pst isolate was multiplied in a separate isolation growth chamber to prevent cross-contamination.

To collect the spores, channels, consisting of light metal strips (4 x 50 cm), were laid between rows of seedlings (Figure IV-1). Once sporulation started, 10 to 14 days post inoculation (DPI), the spores were knocked off the leaves by gently tapping the plants with a plastic stick and collected from the metal strips. The spores could be collected every 2 to 3 days over a two week period until the leaves were dry.

2.2.2.2 Small scale multiplication

Small scale multiplications of the Pst isolates collected in the field were carried out to pathotype them. The susceptible cultivars were sown in individual 12 cm pots (Stewart Plastics Ltd.) fitted with a central tube allowing air flow from to bottom of the pot up to the soil surface. Maleic hydrazyde solution (0.1g/l) was applied at GS07 and plant grown in a free disease growth room until inoculation. The seedlings were inoculated at GS11 manually by brushing a spore/talc mixture or simply rubbing infected leaves on the disease-free plant. Next, the pots were covered with individual perspex domes (Stewart Plastics Ltd.) and incubated at 8°C for 48 h, before being moved to an isolation plant propagator (Burkard Manufacturing Co. Ltd) for individual small pots (Figure IV-1). The propagator consists of a bench with air flow system and artificial lighting. Filtered and humidified air continuously circulates from beneath the pots through the fitted tube to the top of the dome where 2 small apertures allow the air to escape. The air flow creates a constant positive air pressure in each dome preventing spore transfer and cross-contamination between pots.

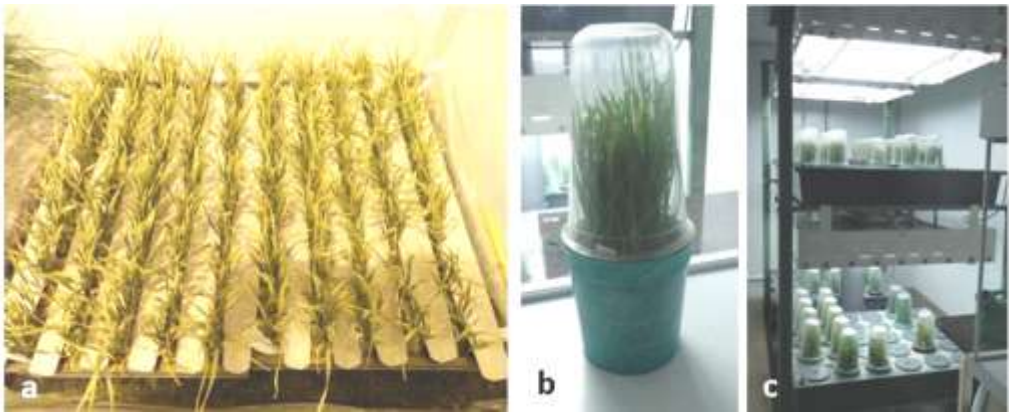

Figure IV-1: WYR spores production

(a) Large scale multiplication in multi-cell trays with metal stripes, (b) 12 cm pots and dome used for small scale spore multiplication, (c) Burkard system.

When sporulation occurred, urediniospores were collected by tapping the rusted plants over a piece of smooth dry paper and transferred into test tubes for storage, before being used in seedling test.

2.2.3 Creation of short-term working stocks and reference stocks for long-term storage

For short term conservation, up to 3 months, and immediate use for seedlings tests, the spores were conserved in half filled test tubes closed with cotton wool, at 4°C in a vacuum desiccator containing silica gel crystals. This was the preferred method of conservation for spores used in seedling tests.

If the spores were not to be used within 2 months, to keep them for future multiplication or for reference, approximately 5 mg of spores were placed in a glass ampoule, freeze-dried for 24h and vacuum sealed. The ampoules were stored at 6 °C. Sealed in this manner, the spores could be conserved for several years and yet maintain their germinebility.

Using the long term conservation method, we created reference stocks of 20 to 30 ampoules for each of the original isolates, each reference stock being the result of one round of multiplication from the UKCPVS ampoules on a selective susceptible cultivar. The ampoule stocks were used throughout the project to multiply spores needed for the different tests, limiting the risk of genetic drift due to successive multiplications.

Spores from ampoules were not used directly in seedling tests as we preferred to use spores not older than 2 months to inoculate seedling tests or transplants.

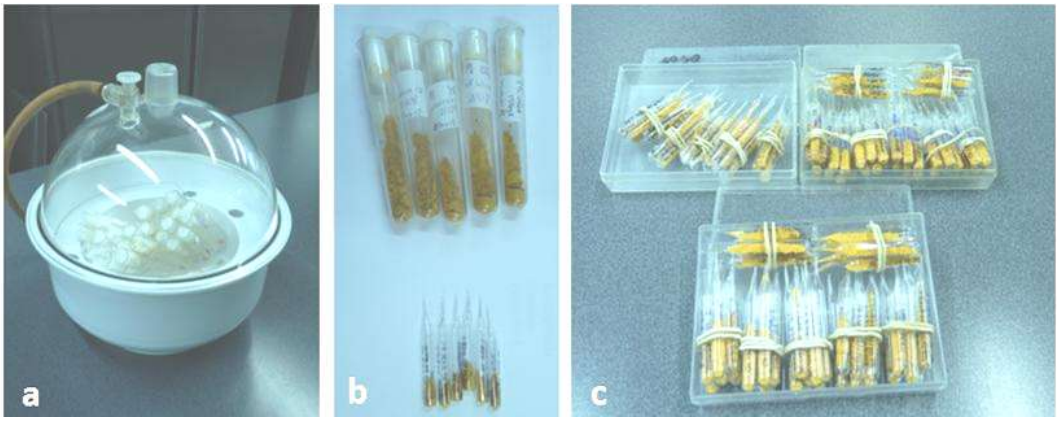

Figure IV-2 : Spore conservation.

(a) Vacuum desiccator, (b) test tube and vacuum sealed glass ampoules containing WYR spores, (c) Reference collection of WYR conserved in sealed glass ampoules

2.3 Seedling tests: evaluation of yellow rust resistance at seedling stage

2.3.1 Seedling test management

To evaluate the resistance of the varieties from the YR panel at seedling stage against some UK Pst isolates, three seedling tests were conducted on the YR panel, two using "Solstice" 08/21 Pst isolate and a third using the "Brock" 07/03 Pst isolate.

The seedling tests were sown in 96 cell trays (34 x 52 cm) or 77 cells trays (29.5 x 60 cm), each cell constituting an experimental unit. Seven to ten seeds were sown in each cell in order to obtain a minimum of three plants for each cultivar at GS11 to inoculate and evaluate the average host response. Each seedling test was composed of 2 replicates organized in a complete randomized block. Following the observation of an edge effect during the first seedling test, the outer cells of each tray were then filled with discard susceptible varieties.

Once sown, the trays were place in a disease free glasshouse or a growth room prior inoculation. Indirect contact with maleic hydrazyde was avoided by rinsing benches prior placing the trays to germinate. Alternatively the trays were placed on support to avoid direct contact with the bench so the chemical will not influence the reaction to the pathogen. When the seedlings were between GS11 and GS12, the average height of the seedlings was recorded. At this point, plots with average seedling size less than 5 cm, limited number of seedlings (less than 3) or discoloration were discarded.

Following the measurements, the trays well watered and placed in individual polythene bags. 20 cm plastic sticks were placed at each corner of the trays to avoid contact between the bag and the plants during the incubation. A 1:19 spore: talc mixture was

prepared and then 3g of the mixture per tray, equivalent to 0.15g spores and 2.85g of talc, was distributed in an individual glass jar. Using an air-blown spore inoculators, each tray was inoculated individually with the contents of a jar (Figure IV-3). The bags were then sealed and placed in an incubator at 8°C, during 24 to 48 hours, in the dark. The bag operates as a dew chamber to keep a high humidity level and provides favourable conditions for spore germination.

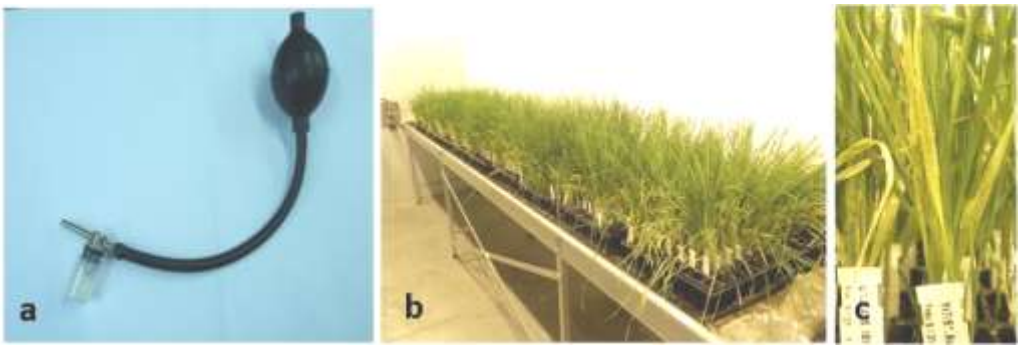

Figure IV-3 : Seedling test.

(a) Glass jar containing spore/talc inoculum mixture and air blow inoculators consisting of a puffer bulb and a nozzle. (b) Seedling test in growth chamber at 21 DPI. (c) Seedling plot sporulating ready to be assessed.

After the incubation, the seedling trays were removed from their bag and placed in a growth room under the following conditions: 11°C at night and 18°C during the day. Depending on the growth chamber, the lighting was composed of high pressure sodium lamps (400 W), metal halide lamps (400 W) or a mix of both. The lamps were set to a 16h day / 8 h night cycle.

Table IV-2: YR panel seedling tests and experimental conditions.

Pst Isolate	Date	Varieties tested	Edge of discards	Pre-inoculation	Post-inoculation	Assessment
08/21"Solstice"	November 2009	299 (77 per trays)	No	Glasshouse Sodium lamps	Growth chamber Sodium lamps	17DPI
08/21"Solstice"	March 2010	317 (60 per trays)	Yes	Glasshouse Natural light	Growth chamber Sodium +metal halide lamps	17DPI
03/07 "Brock"	June 2011	308 (60 per trays)	Yes	Growth chamber Sodium lamps	Growth chamber Sodium + metal halide lamps	19DPI

2.3.2 Seedling test assessment

Depending on the isolate and the environmental conditions of the test (Table IV-2), the tests were scored between 14 to 19 days post inoculation (DPI). The variety Victo was placed in each tray as a susceptible control and to help choosing the most suitable scoring

date. The infection type was assessed on the first leaf of the seedling two times for consistency. Disease assessments followed the 0 to 9 infection type (IF) scoring system (Table IV-3 and Figure IV-4) described by McNeal et al.(1971).

The consistency of the scores between replicates was checked at the end of the second scoring. In case of large variation between replicates, the score for selected varieties was checked a third time.

Lines with an average IF score < were classified resistant and based on a gene for gene relationship were expected to present at least one seedling resistance. Lines with IF>6 were considered susceptible and value 4-6 were interpreted as intermediate response.

Table IV-3: Description of IF scale and class of host response used to evaluate yellow rust seedling resistance

(Adapted from Roelfs et al., 1992).

Mc Neal IF	Disease symptoms
0	No visible infection
1	Necrotic flecks without sporulation
2	Necrotic area without sporulation
3	Trace sporulation with necrotic and chlorotic area
4	Light sporulation with necrotic and chlorotic area
5	Intermediate sporulation with necrotic and chlorotic area
6	Moderate sporulation with necrotic and chlorotic area
7	Moderate sporulation with chlorosis
8	Abundant sporulation with chlorosis
9	Abundant sporulation without chlorosis

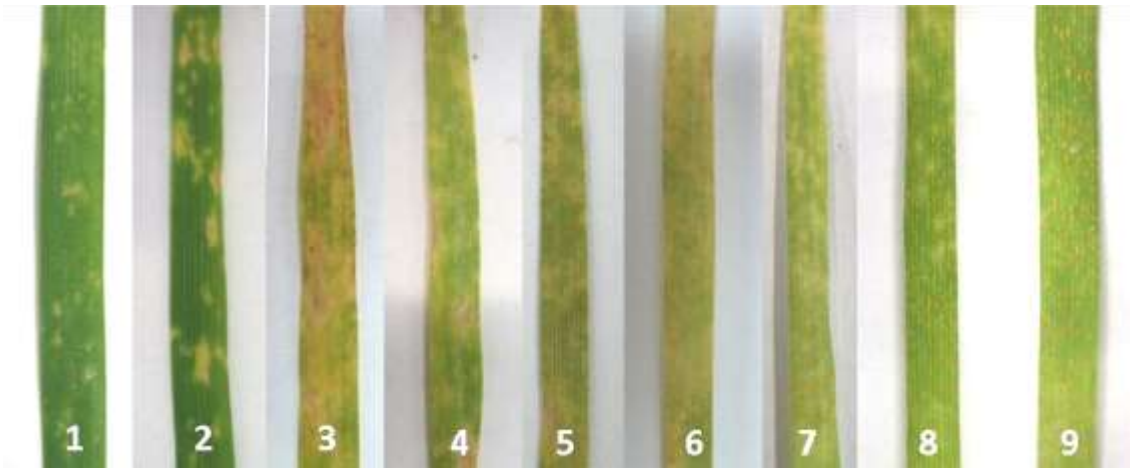

Figure IV-4: Illustration of seedling test infection types 1 to 9 using the McNeal scale (0 IF is not represented)

2.4 Evaluation of Pst isolates virulence

The extended virulence profiling of the Pst isolates followed the same protocol described in preceding section. The objective in this case being to define the pathotype or virulence spectrum of a Pst isolates using differential hosts. We used such tests to define in detail the pathotype of the isolates used in seedling tests and adult plant tests, but also to define the pathotype of isolates recovered from the field. In some cases, the number of replicates was limited to one because of low spore availability.

2.5 Adult plant tests: evaluation of yellow rust resistance at adult plant stage

2.5.1 Field trial management

The YR panel was sown in 3 replicated blocks during the 2009-2010 and 2010-2011 growing season on NIAB trial grounds in Cambridge, UK. Within each block approximately 3g of seeds of each cultivar were sown by hand in tussock plots. The tussock plots were 50 cm apart and organised in 16 rows. Spreader plots with a mixture of susceptible varieties Victo and Vuka were evenly sown throughout the trial at a density of one spreader plot for 4.3 test plots.

Each randomised block was surrounded by 1m high plastic netting acting as windbreak to limit the spread of spores to and from nearby trials.

A minimum of 10 meters of rye was sown around the trial to isolate it from other trials. Following the observation of an edge effect due to the wind breaks, during the first

season of trials (2009-2010), a discard edge of barley was sown around each block for the 2010-2011 season.

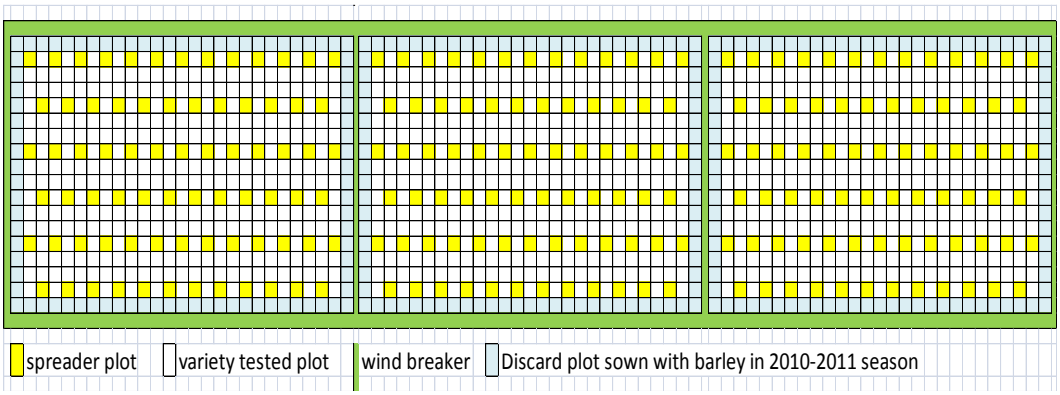

Figure IV-5: Schematic layout of adult plant yellow rust resistance trials

Trials were inoculated at around GS30 by transplanting seedlings infected with the selected Pst isolate in the field. Sporulating seedlings were produced following a similar method to the large scale spore production. Susceptible variety Victo was sown in clumps (10 to 15 seeds per cell) in 77 (29.5 x 60 cm) or 96 cell trays (34 x 52 cm). Maleic hydrazide was applied to encourage sporulation. Once the seedlings started to sporulate, they were transferred from the growth room to an outside covered area (e.g. barn or cold glasshouse) to harden for a few days prior to field transplantation. The seedling plugs were finally planted by hand with a trowel on opposite edges of each spreader plot, with a minimum of 2 transplants per spreader plots.

Figure IV-6 : Adult plant test

(a) Spreader plot with two PST inoculated transplants in March 2010. (b) Overview of adult plant test with windbreaker in March 2010. (c) View of one replicate of an adult plant test in May 2010.

Table IV-4: Description of the adult plant test setup

Season	Varieties tested	Sowing date	Inoculated transplant transfer	Pst isolates	Edge of discard
2009-2010	295	28 October 2009	25 March 2010	08/21 "Solstice" 2 transplants per spreader plot	No
2010-2011	308	28 October 2010	23 March 2011	08/31 "Solstice" 08/501 "Timber" 03/07 "Brock" 1 transplant of each per spreader plot	Yes

2.5.2 Field trial assessment

Yellow rust disease was recorded quantitatively and qualitatively, using the respective measures of severity and field host response (HostR).

Severity corresponds to the percentage of leaf area infected by the disease and was assessed on the entire plot using the modified Cobb scale (Peterson et al., 1948). Figure IV-7 illustrates severity scoring. Lines were classified as followed based on their severity value: 0-5% was interpreted as highly resistant, 5.1- 20% resistant, 21-40% intermediate 41-60% susceptible, 61-100% highly susceptible.

The field host response describes the type of disease reaction (0, R, MR, MRMS, MS, S) on a six-point qualitative scale from immune (0) to susceptible (S) (Table IV-5). Each host response was associated to a numerical constant allowing the estimation of an average host response within trial and the analysis of the data via analyse of variance. An average host response inferior to 0.5 was interpreted as resistant, while an average host superior to 0.7 was interpreted as susceptible

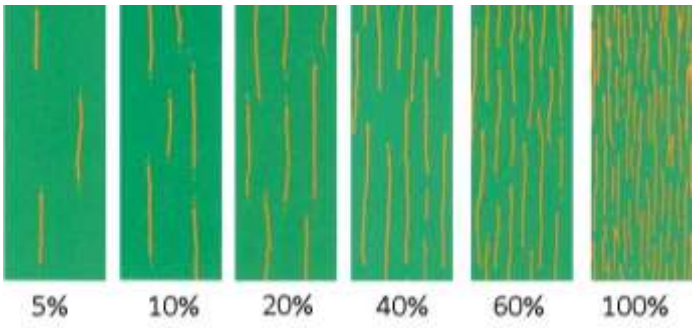

Figure IV-7: Illustration of severity assessments using Modified Cobb scale

(Source : http://wheatdoctor.cimmyt.org)

Table IV-5 : Description of field host response score

Code	Host response	Description	Constant associated
0	**Immune**	No visible infection on plant	0
R	**Resistant**	Visible chlorosis or necrosis, no uredia are present.	0.2
MR	**Moderately Resistant**	Small uredia are present and surrounded by either chlorotic or necrotic areas	0.4
MRMS	**Intermediate**	Variable sized uredia are present; some with chlorosis, necrosis, or both.	0.6
MS	**Moderately Susceptible**	Medium sized uredia are present and possible surrounded by chlorotic areas.	0.8
S	**Susceptible**	Large uredia are present, generally with little or no chlorosis and no necrosis.	1

The level of yellow rust infection was recorded repeatedly during the spring to record both early and late infection and to allow the calculation of Area under Disease Progress Curve (AUDPC). The AUDPC was calculated using the formula proposed by Campbell and Madden (1990) :

(IV-1)

$$\mathrm{AUDPC} = \sum_{i} \left[\frac{x_i + x_{i+1}}{2}\right](t_{i+1} - t_i)$$

Where x_i is the rust severity of the ith note; $x_{i+1}$is the rust severity of the i+1th note, $t_{i+1} - t_i$ is the number of days between the ith note and the i+1th note

Relative AUDPC (AUDPCr) values were calculated for each variety as a percentage of the mean AUDPC value of spreader plots. Lines with AUDPCr value of 0- 0.20, 0.21-0.40 and 0.41-0.60 were regarded as possessing high, moderate and low levels of field resistance, respectively. Lines with AUDPCr above 0.60 were regarded as susceptible, although value between 0.61 and 0.80 could indicate the presence of low level APR.

The date of growth stage GS45 (flag leave sheath swollen) and GS50 (ear emergence) were also recorded respectively in 2010 and 2011, to take into account the development stage effect on rust severity. Additionally, straw length was recorded after flowering for each variety.

Table IV-6: Description of the adult plant test assessments

Season	Period of scoring	Frequency severity / host response scoring	Additional phenotypes
2009-2010	17 May to 18 June	7 days / 15 days	Date GS45 Straw length primary tiller
2010-2011	22 May to 23 June	10 days / 15 days	Date GS50 Straw length primary tiller

2.5.3 Retrieval of Pst isolates from leaf samples and small scale spore production

A small number of leaves presenting active sporulating lesions were collected from the field trials in 2010 at the end of the experiment, to control the virulence in the field. Following the methods given by the UKCPVS, Pst isolates were retrieved from the samples. Spores were produced at a small scale and a seedling tests with a limited set of WYR differentials was realized.

2.6 Detection of *Yr* genes using molecular markers

DNA was extracted from each variety of the YR panel as well as control following the protocol described in Chapter V. To identify some known *Yr* genes present in the YR panel, published linked markers to *Yr5*, *Yr9* and *Yr17* were tested and applied to the YR panel. All these *Yr* genes are carried by alien introgression (*Yr5* from *Triticum spelta*, *Yr9* from *Secale cereale* and *Yr17* from *Aegilops ventricosa*) and the markers selected tag the respective introgressed segments.

2.6.1 Assays for *Yr5*

Two assays were tested to identify the presence of *Yr5*. The first assay uses a CAPS marker described by Chen et al. (2003b), a detailed protocol is available in the website http://maswheat.ucdavis.edu. The second assay uses STS marker described by Smith et al. (2007).

Two primers pairs were tested from Chen et al. (2003b) assay (Table IV-7), to enable the use of a ABI DNA analyser to resolve the amplification products, the reverse primer STS-10 was labelled with a fluorescent dye 6-carboxyfluorescein (6-*FAM*). PCR were carried out in 20µl reaction volume, containing 0.5 µM of each selected primer, 200 µM of each deoxynucleoside triphosphates (dNTP), 1U of *Faststart Taq* (Roche), 2µl *Faststart* 10x buffer with $MgCl_2$ and approximately 20ng of DNA. The reaction conditions were 95°C for 6 min; followed by 45 cycles of 95°C for 30 s, 45°C for 30s and 72°C for 1min. A final step of extension at 72°C for 10 min was added at the end. After amplification,

5µl was used to check the success of amplification on agarose gel. 0.25µl of restriction enzyme *Dpn*II (NEB) equivalent to 2.5U and 1 µl of 10X NEB buffer were added to 9 µl of the remaining PCR product, samples were incubated at 37°C for 2 h. Finally, digestion products were separated either in 2.5% agarose gel containing ethidium bromide or using an ABI 3730xl DNA analyser. Restriction profile was resolved under UV light on the agarose gel. Alternatively, major fragments were resolved using the software GeneMapper version 4 when the ABI analyser was used.

Table IV-7: Primers and product expected for *Yr5* CAPS marker described by Chen et al.(2003b)

Primer pair	Sequences 5'-3'	PCR products expected	Major restriction fragments after restriction by *Dpn*II
STS-7 (forward) STS-10 (reverse)	GTA CAA TTC ACC TAG AGT CAA ACT TAT CAG GAT TAC	472 bp in absence of *Yr*5 478 bp in presence of *Yr*5	182 and 102 bp in absence of *Yr*5 289 bp in presence of *Yr*5
STS-9 (forward) STS-10 (reverse)	AAA GAA TAC TTT AAT GAA CAA ACT TAT CAG GAT TAC	433 bp in absence of *Yr*5 439 bp in presence of *Yr*5	182 and 102 bp in absence of *Yr*5 289 bp in presence of *Yr*5

The primer pair S19M93-100 F/R from Smith et al. (2007) was tested (Table IV-8). PCR were carried out in 10µl reaction volume, containing 5 µM of forward and reverse primers, 200 µM of each dNTP, 1U of *Faststart Taq* (Roche), 1µl *Faststart* 10x buffer with $MgCl_2$ and approximately 10ng of DNA. The reaction conditions were 95°C for 6 min; followed by 40 cycles of 95°C for 30 s, 63°C for 30s and 72°C for 20s; with a final extension at 72°C for 10 min. After the PCR, 5µl of the amplification was used to reveal the presence /absence of 100bp amplification on agarose gel.

Table IV-8: primers and product expected for *Yr5* STS marker described by Smith et al. (2007)

Primer Pair	Sequences 5'-3'	PCR product
S19M93- 100 F / R	TAATTGGGACCGAGAGACG TTCTTGCAGCTCCAAAACCT	100 bp in presence of *Yr*5 2 poorly amplified fragments in absence of *Yr*5

2.6.2 Assay for *Yr9*

The presence of the *Yr9* gene was evaluated indirectly by identifying the presence of the 1BL.1RS translocation, *Yr9* being introduced with the translocation. The 1BL.1RS translocation was assayed using the co-dominant marker developed for rapid detection of the translocation in winter wheat, as described by de Froidmont (1998)(Table IV-9). The assay is a multiplex PCR were carried out in 10µl reaction volume. The reaction mix contained 1µM of each primer (O11B3, O11B5, SECA2 and SECA 3), 200 µM of each dNTP, 0.4U of *Faststart Taq* (Roche), 1µl *Faststart* 10x buffer with $MgCl_2$ and approximately 10ng of

DNA template. The reaction conditions were 95°C for 6 min; followed by 35 cycles of 95°C for 30 s, 60°C for 30s and 72°C for 1 min; with a final extension at 72°C for 10 min. The amplification products were detected after migration on a 2.5%agarose gel.

Table IV-9: Primers and expected product sizes for 1BL.1RS translocation assay designed by de Froidmont (1998)

Locus	Target	Primer pair	Sequence 5'-3'	PCR product
Glu-B3 (wheat 1BS)	Low molecular weight glutenin gene	O11B3 O11B5	GTTGCTGCTGAGGTTGGTTC GGTACCAACAACAACAACCC	636bp
SEC-1b (rye 1RS)	ω-secalin gene	SECA2 SECA3	GTTTGCTGGGGAATTATTTG TCCTCATCTTTGTCCTCGCC	412bp

2.6.3 Assay for *Yr17*

Yr17 was identified based on the SCAR marker SC-Y15 developed by Robert et al. (1999). The primer sequences are available for research purposes upon request to Francoise Dedryver (INRA, Le Rheu, France). The PCR reaction was carried out in 10 µl solution. The reaction mix contained 1µM of the forward and reverse primers, 100 µM of each dNTP, 0.5U of *Faststart Taq* (Roche), 1µl *Faststart* 10x buffer without $MgCl_2$, 2µl *Faststart* 5x GC mix, 25 µM of $MgCl_2$ and approximately 10ng of DNA template. The amplification program was 95°C for 6 min; followed by 35 cycles of 95°C for 1 min, 64°C for 2 min and 72°C for 1 min; with a final extension at 72°C for 10 min. The amplification products were detected after migration on 2% agarose gel. In presence of *Yr17*, a band around 580bp should be observed.

2.7 Data analysis of YR panel seedling and adult plant test

GenStat 13th Edition (Payne et al., 2009) was used to perform the statistical analysis on phenotypic data sets. Analysis of variance using REML were performed on infection type, host response, rust severity and AUDPCr. From the fitted model, the adjusted mean for each varieties based on BLUP were obtained. When necessary, the data were transformed using log (x+1) transformation to achieve near normality (x being the score recorded).

The model fitted for seedling test is as followed:

(IV-2)

$$y_{ikp} = \mu + V_i + B_k + S_{kp} + e_{ikp}$$

y_{ijk} is the infection type of the variety i, in bloc k, in the sub-bloc (tray) p; μ is the overall mean; V_i is the effect of the ith variety; B_k is the effect of the kth bloc; S_p is the effect of the pth sub-bloc within the bloc k ; e_{ikp} is the residual. Variety, block and sub-block were treated as random factors to obtained variance components to calculate adjusted means based on BLUP. A fixed effect model was used to look at significance of effects based on Wald statistic.

The model fitted for APR test is as followed:

(IV-3)

$$y_{ik} = \mu + V_i + B_k + G + e_{ik}$$

y_{ik} is the severity, the host response or the AUDPC of the variety i, in bloc k; μ is the overall mean; V_i is the effect of the ith variety; B_k is the effect of the kth bloc; G correspond to the measure of the plot growth stage (GH50 or GH43) and is used as covariate in the model; e_{ik} is the residual. Variety and block were treated as random factors to obtained variance components and calculate adjusted means based on BLUP. A fixed effect model was used to look at significant effect based on Wald statistic

To compare the genetic (i.e. heritable) and environmental (i.e. non heritable) source of variation within a trait, heritability was calculated. Replicated data on WYR resistance phenotype was used to estimate genetic variance (σ^2_G) and error variance (σ^2_E) within a trial. Heritability (h^2) was calculated as follow:

(IV-4)

$$h^2 = \sigma^2_G/\sigma^2_P = \sigma^2_G/(\sigma^2_G + \frac{\sigma^2_e}{r})$$

Whereσ^2_G, σ^2_P and σ^2_e represent the variances genetic (variety), phenotypic and residual respectively. r is the number of replication per line. The estimates of variance for σ^2_G and σ^2_e were obtained from GenStat output after fitting a simplified random model including only variety and bloc effects.

To obtain an adjusted resistance score for 1) the two seedling tests inoculated with Pst 08/21 and 2) the two APR tests, data were combined. The following model was fitted:

(IV-5)

$$y_{ikj} = \mu + V_i + T_j + TV_{ji} + e_{ikj}$$

y_{ikj} is the yellow rust score (infection type at seedling test, AUDPCr or average severity) for the variety i, in bloc k, in trial j; μ is the overall mean; V_i is the effect of the ith variety; ; T_j is the effect of the jth trial;; TV_{ji} is the interaction between the test and the variety; e_{ikj} is the residual. A fixed effect model was used to look at significant effect based on Wald statistic. Varieties, block, trial and their interactions were treated as random factors to calculate adjusted means based on BLUP.

To compare the genetic and environmental source of variation between trials, heritability was calculated as followed:

(IV-6)

$$h^2 = \frac{\sigma_G^2}{\sigma_P^2} = \sigma_G^2/(\sigma_G^2 + \sigma_E^2)$$

$$\text{where } \sigma_E^2 = \left(\frac{\sigma_e^2}{r_s}\right) + \left(\frac{\sigma_{tv}^2}{s}\right)$$

σ_G^2,σ_P^2, σ_E^2, σ_e^2 represent respectively the variances genetic (variety), phenotypic, environmental and residual. σ_{tv}^2 is the variance of block.variety component. r_s The estimates of variance forσ_G^2, σ_{tv}^2 and σ_e^2 were obtained from GenStat output after fitting random model (IV-5).

The correlation co-efficient (r^2) between infection type at seedling stage, host response at adult stage, severity in the field and AUDPCr were calculated in R (cran.r-project.org/).

3 RESULTS

3.1 Extended virulence profile of Pst isolates

The three Pst isolates used to evaluate the current resistance of the YR panel have been tested at seedling stage against a large set of differentials hosts and reference varieties containing diverse sources of resistance against WYR (Table IV-10, Table IV-11, Table IV-12).

The virulence profiles published by the UKCPVS were confirmed. The profiles were completed for most of the numbered *Yr* genes effective at seedling stage, only a few could not be tested as seed from the reference varieties were not obtained.

The "Solstice race" 08/21 is virulent on *Yr* ***1, 2, 3a, 3b, 4a, 4b, 6, 9, 17, 20, 21, 25, 26, 27, 32, A, Sd, HVII*** and avirulent on Yr ***5, 7, 8, 10, 15, 24, SP***. Virulence of this isolate on *Yr19, Yr22, Yr23* and *YrMor* remains unknown as there is no differential host containing those genes in isolation. In addition 08/21 was shown to be virulent on varieties Paha (*YrPa1, YrPa2, YrPa3),* Produra (*YrPr1, YrPr2),* Druchamps (*Yr3a, Yr4a, YrD, YrDru, YrDru2*), Daws (*YrDa1, YrDa2),* C591 *(YrC591)* and Alpowa (*YrAlp, APR Yr39),*

The "Brock race" 07/3 is virulent on ***Yr 1, 2, 3a, 3b, 4a, 4b, 7, 21, 22, 23, 25, 26, 27, 32, A, Sd, HVII*** and avirulent on ***Yr 5, 6, 8, 9, 10, 15, 24, SP***. As in the previous case,

virulence on *Yr19, Yr20, YrMor* could not be determined. The virulence for *Yr17* remains uncertain as the isolate was avirulent (average IF 3.5) on differential VPM1 for *Yr17*, while it was virulent (average IF 6.5) on Rendezvous - a variety known to carry *Yr17*- and the 'Avocet' *Yr17* isogenic line *(Yr17*/6* AvS.) Contamination by other isolates is unlikely as the seedling test was carried in a growth room entirely dedicated to isolate 03/07. However the UKCPVS tests on 03/07 collected for historical analysis showed contrasting response for the virulence to *Yr17*, the test from 2003 showed avirulence for *Yr17* based on VPM1 and Rendezvous , while the tests from 2004 and 2005 showed virulence on *Yr17* based on VPM1 and Rendezvous. It is possible the susceptible reaction on the isogenic line but the resistance seen on VPM1 (*Yr17*) is due to additional virulence genes within the Pst isolate as the European differentials VPM1 (*Yr17*). However, it is more likely that there is a problem with the seed stock of VPM1. Alternatively, the difference of IF response observed on differential hosts Hyak, VPM1, *Yr17*/6*Avocet and Rendezvous in seedling may be due to unfavourable experimental condition for the expression *Yr17* and a genetic background effect. Moderate variability of the expression of *Yr17* was observed by Bariana et al. (2001) and Bariana and McIntosh (1994) based on the environmental condition and genetic background, cultivars with *Yr17* under low light intensity and low temperature tend to appear more susceptible. Therefore the isolate Brock can be virulent on *Yr17* but the expression of *Yr17* was not expressed fully in Hyak, Rendezvous and *Yr17*/*6Avs. Similar contradiction between *Yr17*/6*Avs and VPM1 IF were observed by Lewis (2006).

In addition, the test revealed the virulence of 03/7 on cvs Paha (*YrPa1, YrPa2, YrPa3)*, Druchamps (*Yr3a, Yr4a, YrD, YrDru, YrDru2*), Alpowa (*YrAlp,* APR *Yr39),* IDO377S (*Yr43*), Zak (*Yr44*) and Daws (*YrDa1, YrDa2).*

The "Timber race" 08/501 is virulent on ***Yr 1, 2, 3a, 3b, 4a, 4b, 6, 9, 17, 20, 21, 25, 26, 27, A, Sd***, ***HVII*** and avirulent on ***Yr 5, 7, 8, 10, 15, 24, 32, SP***. Again, virulence is unknown for *Yr19, Yr22, Yr23* and *YrMor*. Moreover 08/501 present a high virulence on Yamhill (*Yr2, Yr4a, YrYam),* Paha (*YrPa1, YrPa2, YrPa3),* Produra (*YrPr1, YrPr2),* Druchamps (*Yr3a, Yr4a, YrD, YrDru, YrDru2*), Daws (*YrDa1, YrDa2),* Alpowa (*YrAlp,* APR *Yr39)* and C591 *(YrC591).*

The three isolates showed contrasted response against the *Yr* genes *6, 7, 9, 32, 43, 44* and cvs Produra, Express, Brock, Robigus, Timber and Solstice. All three isolates were virulent on *Yr 1, 2, 3, 4, 25, 26, 27* and cv Paha, Druchamps, Daws and Alpowa and all three were avirulent on *Yr 5, 8, 10, 15, 4, SP* and cvs Tres, Tyes, Batavia, PI181434 (=205) and Cadenza. The observation of susceptible response at seedling stage in cultivar carrying APR e.g. Guardian, Alcedo, Cappelle Desprez, Opata and isogenic lines with *Yr18* confirmed those cvs will be useful to identify the effectiveness of their APR in the field.

We observed some conflicting results between differentials for *Yr17*, *Yr27* and *Yr32*.

Differential host VPM1 and Hyak for *Yr17* provided contradictory infection type. For instance with isolate 08/21, VPM 1 was highly susceptible (score IF 9) but Hyak was resistant (IF 3.5). Hyak is likely to carry an additional source of resistance partially effective against UK isolates.

Based on Selkich and the isogenic line *Yr27*/6* AvS, the tested isolates are virulent on *Yr27*, however cv CianoT79 carrying *Yr27* appeared resistance against 08/21 and presented an intermediate response against 03/7 and 08/501. Ciano T79 is likely to carry an additional resistance gene providing some protection against the UK isolates. Similarly, Mac Donal (McDonald and Linde, 2002) noticed a difference of *Yr27* expression depending of the Pst isolates as well as the cv.

Finally, although 08/501 would be classified as avirulent on *Yr32* based on the host response on Carstens V, the isogenic line *Yr32*/6* AvS gave a less clear-cut response with an intermediate IF.

These observations underline the fact that differential varieties which are known to possess the same resistance gene still differ throughout the rest of the genome and these differences may include additional resistance genes that respond to additional unknown avirulence factors present in isolates and can never therefore be considered fully equivalent. This problem is the main driver behind the development of isogenic series containing small introgressions carrying singular *Yr* genes.

Table IV-10: Host response observed on official differential hosts at seedling stage against Pst isolates used on YR panel

R: resistant IF<4; S: susceptible IF>6, I: intermediate IF between 4 and 6, for intermediate response the average IF score is indicated in parentheses

	***Yr* gene**	**08/21 Solstice race**	**03/7 Brock race**	**08/501 Timber race**
Chinese 166	*Yr1*	S	S	S
Kalyansona	*Yr 2*	S	S	S
Heines Kolben	*Yr2, Yr6*	S	R	S
Yamhill	*Yr2,Yr4a, YrYam*	I(4.5)	I(5)	S
Heines VII	*Yr2, Yr25,YrHVII*	S	S	S
Heines Peko	*Yr2, Yr6, Yr25*	S	I(4)	S
Vilmorin 23	*Yr3a,Yr4a*	S	S	S
Nord Desprez	*Yr3a, Yr4a +*	S	S	S
Hybrid 46	*Yr3b, Yr4b*	S	S	S
T. spelta Album	*Yr5*	R	R	R
Fielder	*Yr6, Yr20*	S	R	S
Lee	*Yr7, Yr22, Yr23*	R	S	R
Compair	*Yr8, Yr19*	R	R	R
Federation x4/Kavkaz	*Yr9*	S	R	S
Riebesel 47-51	*Yr9*	-	R	S
Clement	*Yr2,Yr9,Yr25, YrCle*	S	R	S
Moro	*Yr10,YrMor*	R	R	R
Boston	*Yr15*	R	R	R
VPM 1	*Yr17*	S	R	S
Hyak	*Yr17*	R	I(4)	I(6)
Lemhi	*Yr21*	S	S	S
Carstens V	*Yr32, Yr25*	S	S	R
Paha	*YrPa1, YrPa2, YrPa3*	S	S	S
Produra	*YrPr1,YrPr2*	S	R	S
Tres	*YrTr1, YrTr2, Yr32*	R	R	R
Tyee	*YrTye*	R	R	R
Strubes Dickkopf	*Yr25, YSd*	S	S	S
Spalding Prolific	*YrSp*	R	R	R
Express	*YrExp1, YrExp2 +APR*	R	S	R

Table IV-11: Host response observed on isogenic lines at seedling stage against Pst isolates used on YR panel

R: resistant IF<4; S: susceptible IF>6, I: intermediate IF between 4 and 6, for intermediate response the average IF score is indicated in parentheses. – cvs not tested.

WYR near-isogenic line	*Yr* gene	08/21 Solstice race	03/7 Brock race	08/501 Timber race
Avocet S	*none*	S	S	S
Avocet R	*YrA*	S	S	S
Yr1/6* AvS	*Yr1*	S	S	S
Yr5/6* AvS	*Yr5*	R	R	R
Yr6/6* AvS	*Yr6*	S	I(6)	S
Yr7/6* AvS	*Yr7*	R	S	I(4)
Yr8/6* AvS	*Yr8*	R	R	R
Yr9/6* AvS	*Yr9*	S	R	S
Yr10/6* AvS	*Yr10*	R	R	R
Yr15/6* AvS	*Yr15*	R	R	R
Yr17/6* AvS	*Yr17*	S	S	S
Yr18/6* AvS	*Yr18**	S	S	S
Yr24/6* AvS	*Yr24*	R	R	R
Yr26/6* AvS	*Yr26*	S	S	S
Yr27/6* AvS	*Yr27*	S	S	S
Yr32/6* AvS	*Yr32*	S	I(4)	I(6)
YrSP/6* AvS	*YrSP*	R	R	R
Jupateco S	*none*	-	-	S
Jupateco R	*Yr18**	S	-	S

Table IV-12: Host response observed on complementary varieties at seedling stage against Pst isolates used on YR panel

R: resistant IF<4; S: susceptible IF>6, I: intermediate IF between 4 and 6, for intermediate response the average IF score is indicated in parentheses.* indicated adult plant resistance, for which the virulence has not been tested as the test were carried out on seedlings; (a) *YrCk* is a temperature sensitive resistance thus the virulence on this specific gene cannot be evaluated in our testing conditions. * adult plant resistance

	***Yr* genes/QTL**	**8/21 Solstice race**	**03/7 Brock race**	**8/501 Timber race**
Cappelle Desprez	*Yr3a, Yr4a,Yr16* +*	S	S	S
Druchamp	*Yr3a,Yr4a,YrD, YrDru, YrDru2*	S	S	S
Minister	*Yr3c, YrMin*	I(6)	S	S
Madrigal	*Yr6,Yr9,Yr17*	S	R	S
Hornet	*Yr6, Yr9*	S	R	S
Tommy	*Yr7*	R	S	R
Brock	*Yr7, Yr14**	R	S	R
Guardian	*Yr13*,Yr29**	S	S	S
Rendezvous	*Yr17*	S	I(6)	S
Opata 85	*Yr18*,Yr27,Yr30**	S	I(6)	S
Cook	Yr18*, *YrCK*(a)	S	S	S
Talon	*Yr32*	S	R	R
Oxbow	*Yr32 +*	S	R	R
Ciano T79	*Yr27*	R	I(6)	I(5)
Selkirk	*Yr27*	S	S	S
Batavia	*Yr33,YrA,YrBat1,YrBat2*	R	R	I(4)
Alpowa (=WA 7677)	*Yr39*, YrAlp,*	S	S	S
IDO377S	*Yr43*	R	S	R
ZAK	*Yr44 (=YrZac)*	R	S	R
PI181434 (=205)	*Yr45*	R	R	R
C 591	*YrC591*	S	I(6)	S
Daws	*YrDa1, YrDa2*	S	S	S
Cadenza	*unknown*	R	R	R
Claire	*unknown*	I(5.5)	I(6)	S
Robigus	*unknown*	S	R	R
Solstice	*unknown*	S	R	R
Timber	*unknown*	R	R	S
Warrior (RAGT)	*unknown*	I(4)	R	I(4)
Alcedo	*QTL 2DL+ QTL 4BL**	S	S	S
LalBahadur	*none*	S	S	S

3.2 Seedling tests

3.2.1 Seedling test with Brock isolate 03/7

308 varieties from the YR panel have been tested against Pst 03/7 at seedling stage. Four varieties have to be removed from the test because of poor germination. The isolate was highly virulent on Brock (IF=8.5). The IF from the seedling tests with Pst 03/7 displays a positively skewed distribution (Figure IV-8). 66.1% of the varieties were highly resistant with an IF< or =3 and only 18.1% of the varieties were susceptible (IF>6).

Amongst the highly resistant varieties, we observed varieties with *Yr* genes not overcome by the isolates 03/07 such as Boston (*Yr15*), Rialto (*Yr9*), Brigadier (*Yr9, Yr17*), Charger (*Yr6*) and Haven (*Yr6, Yr9*). The high frequency of resistance in the YR Panel is likely to be due to the presence of *Yr9* in many lines. In fact, the 97 lines diagnosed to have *Yr9* based on the 1BL.1RS molecular marker (see section 3.7.2) had an average IF inferior to

2. Only Derwent was diagnosed with *Yr9* but gave a susceptible reaction (average IF =8) which might be explained by an error in sowing. Besides *Yr9*, many lines in the YR panel are likely to carry *Yr6* for which the isolate 03/7 is avirulent explaining the low susceptibility observed such as Shango (*Yr4,Yr6*), Norman (*Yr6*).

Among the highly susceptible varieties we found lines carrying *Yr7* i.e. Camp Remy, Thatcher, varieties with ineffective resistance i.e. Husler (*Yr1*), Joss Cambier *(Yr2+ Yr3*), Avalon *(Yr4*), Prophet *(Yr17*), Armada *(Yr27*), Carstens V *(Yr32*), and varieties with no seedling resistance i.e. Vuka, Soissons.

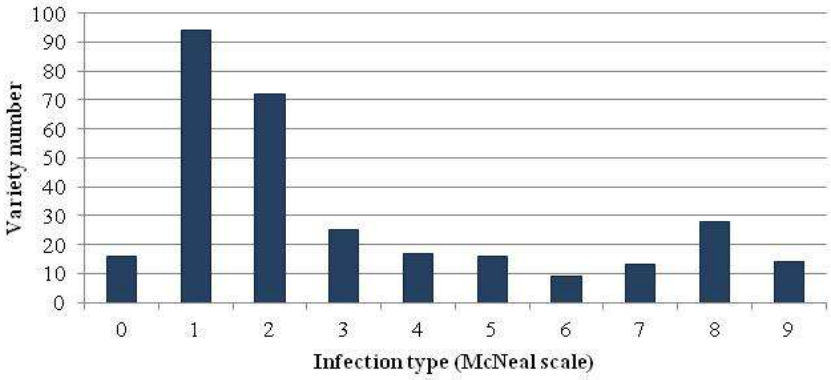

Figure IV-8: Distribution of infection type in seedling test 19 dpi with Pst 03/7

The analysis of variance based on REML showed a negative component of variance for sub-block (tray), therefore the sub-block effect was eliminated from the final model. The analysis showed significant difference in IF ($p<0.001$) between varieties, a significant replicate effect was observed (Table IV-13) but the heritability in the trial was very high (0.97).

Table IV-13: Analysis of variance of infection type at seedling stage against WYR 03/07

(1) numerator degree of freedom; (2) significance probability from Wald tests based on all fixed effect model; (3) estimate of variance component from random model; (4) standard error based on random model; (5) heritability based on simplified random model.

Score	Source	*n.d.f*(1)	*F.pr*(2)	Est. (3)	s.e(4)	H2(5)
IF 19dpi	Variety	*303*	*<0.001*	6.7217	0.5609	0.973
	Block	*1*	*<0.001*	0.0272	0.0401	
	Residual			0.362	0.0289	

3.2.2 Seedling tests with Solstice isolate 08/21

299 and 317 varieties from the YR panel have been tested against Pst 08/21 at seedling stage in November 2009 and March 2010 respectively. Three varieties were removed in each test because of poor germination. The IF from the seedling tests with Pst 08/21 displays a bimodal distribution (Figure IV-9), characteristic of the presence of qualitative resistance. The test run in 2010 presented a higher average IF score, compared to the test run in 2009. The changes in experimental condition e.g. temperature, lighting, presence of discard edges may explain the change of symptom intensity, particularly the lighting during incubation. In addition, powdery mildew contamination was observed at the end of the second test and may have influenced the development of WYR symptoms.

Despite the wide virulence profile of 08/21, many UK elite lines presented highly effective seedling resistance, between 18.7 and 32.4% and of the varieties presented an IF< or =3 depending of the test considered. Among the resistant varieties we found several varieties with *Yr7* for which the isolate is avirulent i.e. Tommy, Thatcher, Brock and varieties with unknown resistance i.e. Ochre, Pennant, Hereford, Timber. The virulence on Solstice was intermediate with IF =6 in 2009 test and IF=5.5 in 2010 test.

41.2% to 42.3 % of the varieties were susceptible (IF>6) to Pst 08/21, among them are varieties with no seedling resistance (Vuka, Soissons), varieties with *Yr* genes overcome by the "Solstice" isolate: Flair *(Yr1)*, Galahad *(Yr1*), Joss Cambier *(Yr2+Yr3)*, Biscay (*Yr2+Yr3+Yr17*), Shango (*Yr4+ Yr6*), Slejpner *(Yr9)*, Apollo (*Yr9*), Prophet *(Yr17)*, Chianti *(Yr17*) and Windsor (*Yr32 + Yr2*).

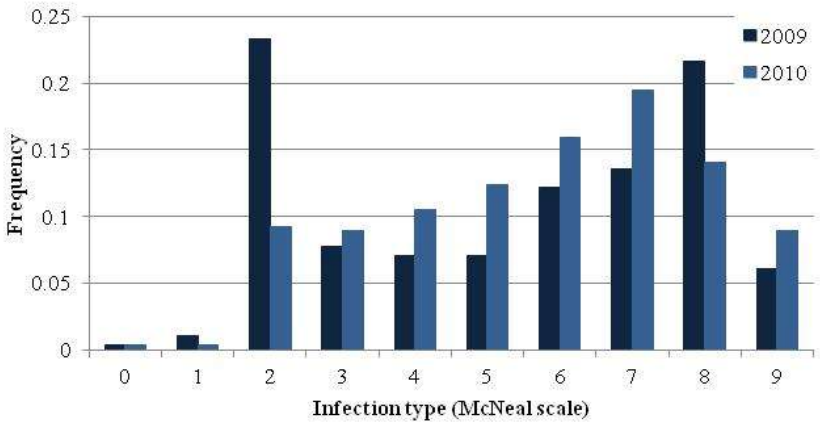

Figure IV-9: Distribution of infection type at 17 dpi in seedling tests from November 2009 and March 2010 with Pst 08/21

The analysis of variance using REML showed a significant difference in IF ($p<0.001$) between varieties, as well as a block effect (trait) in both tests (Table IV-14). The

trait effect is linked with the position of the tray within the growth chamber, with trays positioned in the back of the growth chamber generally showing less sporulation.
Despite the block and sub-block effects, the trials presented of high heritability of 0.92 for the first test and 0.93 for the second test.

Table IV-14: Analyse of variance of infection type from seedling tests against Pst 08/21

(1) numerator degree of freedom; (2) significance probability from Wald tests based on fixed effect model; (3) estimate of variance component from random model; (4) standard error based on random model; (5) heritability based on random model.

Score	Source	*n.d.f*(1)	*F.pr.*(2)	Est.(3)	s.e(4)	$H^{2(5)}$
Test 2009	Variety	*296*	*<0.001*	5.473	0.4927	0.915
IF 17 dpi	Block	*1*	*<0.001*	0.0526	0.0955	
	Tray	*8*	*0.012*	0.0528	0.0434	
	Residual			0.973	0.0817	
Test 2010	Variety	*313*	*<0.001*	4.1968	0.3621	0.929
IF 17 dpi	Block	*1*	*0.041*	0.0019	0.0116	
	Tray	*12*	*0.028*	0.0268	0.0218	
	Residual			0.0622	0.0508	
Tests	Variety	*316*	*<0.001*	3.4954	0.3553	0.807
combined	Test	*1*	*<0.001*	0.0783	0.1189	
IF 17 dpi	Test.Variety	*292*	*<0.001*	1.2742	0.1433	
	Residual			0.859	0.0494	

A relatively low correlation was observed between the two tests (R^2=0.46) (Figure IV-10) which emphasizes the influence of climatic conditions prior and post inoculation on the development of the disease. Temperature and light (intensity and length) pre-inoculation also varied between tests. The first one was sown in late autumn, when light and heat were supplemented. The second one was sown beginning of the spring, no supplemental light was available and the temperature control was limited. After inoculation, both tests were transferred into a growth chamber, with similar temperature set up, however, to help the development of symptoms the lighting was modified by adding metal halide lighting.

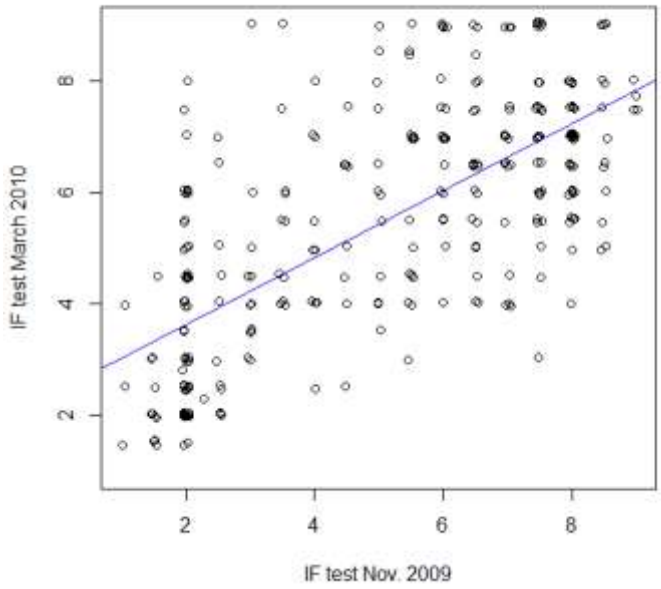

Figure IV-10: Correlation IF score between seedling tests inoculated with Pst 08/21

Plot of the adjusted means from seedling tests inoculated with Pst 08/21 using the jitter function from R (www.r.project.org/). Linear regression line in blue.

To obtain a single rust evaluation for isolate 08/21 at seedling stage, the IF scores from both tests were combined and an adjusted mean based on BLUP was obtained after fitting a model including test effect as described in the section 2.7. The results of the analysis of variance showed as expected a significant tests effect, suggesting the symptoms intensity was generally superior in the second test. This tends to confirm the usefulness of improvement made to the seedling test management between the two tests (higher light intensity post inoculation and presence of discard edges). The analysis of variance showed also some interaction between variety and test (Table IV-14). As illustrated in Figure IV-10, some varieties showing a resistant IF (IF≤4) in the first test such as Hudson, Encore and Context, presented a susceptible response in the second test with IF ≥ 7. The inverse situation was also observed for instance for Norman (IF test 2009=7.5, and IF test 2010=3). The presence of significant interaction suggests that the difference of experimental conditions affected the development of the disease unequally in all varieties. The heritability over the two tests remained high despite the obvious presence of interactions.

3.2.3 Comparison of seedling scores against isolates 08/21 and 03/7

Using adjusted means obtained from GenStat, the IF observed for each variety against isolate 08/21 and isolate 03/7 were compared (Figure IV-11).

A large part of the varieties presented a higher susceptibility against 08/21 which can be explained partly by the additional virulence factors identified in 08/21, particularly *vir6* and *vir9*. 94 lines identified by the red square in the figure were susceptible against 08/21 and resistant against 03/7. Among them, 49 have the 1BL.1RS translocation with *Yr9* based on the genetic marker from de Froidmont (1998)(see section 3.7.2). The 50 remaining includes seven lines known to have *Yr6* (Charger, Comet, Kinsman, Longbow, Maris Freeman, Shango (see Appendix 3 for postulated genes and references) and 27 lines diagnosed to have *Yr17* based on the genetic marker from Robert et al. (2000). No diagnostics markers is available for *Yr6*, however we expected a greater number of lines to carry *Yr6*.

A small part of the varieties had a higher resistance with 08/21 which can be explained by the presence of virulence factor *vir7* in 03/7. Eight lines identified by the green square on the figure (Brock, Camp Remy, Thatcher, Tommy, Cordiale, Ekla, Vilmorin 27, Spark) were highly susceptible against 03/7 and resistant against 08/21. Four of them (Brock, Camp Remy, Thatcher, Tommy) are known to carry *Yr7*, the remaining lines are likely to carry *Yr7* also considering the wild virulence profile of 08/21.

The difference of IF observed between the two seedling tests are supported by the differences in virulence profile of the Pst isolates, particularly for *Yr6, Yr7, Yr9*, we were not able to conclude concerning the virulence of 07/3 on *Yr17* . Additionally, the two isolates presented contrasting virulence for *Yr43* and *Yr44*, however those genes are not likely to be present in the UK winter wheat as *Yr43* and *Yr44* have been identified in American spring wheat, IDO377s and ZAK respectively. Nothing indicated in the pedigrees a common ancestor between those American lines and the YR panel lines.

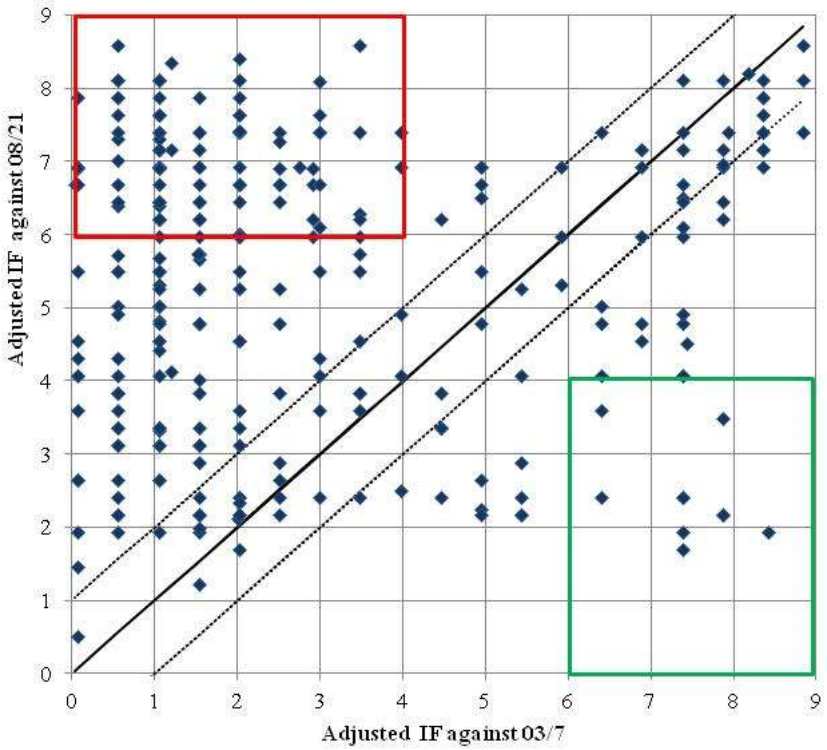

Figure IV-11: Comparison of IF scores with isolates 08/21 and 03/7

The plain line corresponds to IF 03/7 =IF 08/21, the dashed lines delimited the area where IF08/21 = IF 03/7 ± 2 standard error. The upper left corner (red) of the chart includes varieties highly resistant against 03/7 and susceptible against 08/21. The lower right corner (green) of the chart includes varieties susceptible against 03/7 and highly resistant against 08/21.

3.3 Adult tests

3.3.1 Disease pressure and progression in field trials

Two adult plants tests have been carried out on the YR panel in 2010 and 2011. The first one was inoculated with Pst isolates 08/21 characteristic of the Solstice race. The second was inoculated with three Pst isolates (08/21, 03/7 and 08/501) representing current races from the UK, Solstice, Brock and Timber races respectively. The progression of the disease was followed from late May to end of June. The spreader plots composed of the mix of two susceptible varieties Victo and Vuka were scored in conjunction to varieties in test. The average severity in spreader plots (208 plots in 2010 and 225 plots in 2011) provides an indication of disease pressure in the field as well as availability of inoculum. In both trials, the disease severity on spreader plots was particularly high over the period of scoring (Figure IV-12), demonstrating the success of the inoculations and the availability of inoculums in quantity within the field. In 2010, the rust severity observed in spreader plots was 39% on the 17 May and progressed up to 83 % the 18 June. In 2011, despite an exceptional dry

spring, the disease progressed quickly within the spreader plots to reach 67 % on the 22 May, then the disease evolved slowly up to 76% the 23 June.

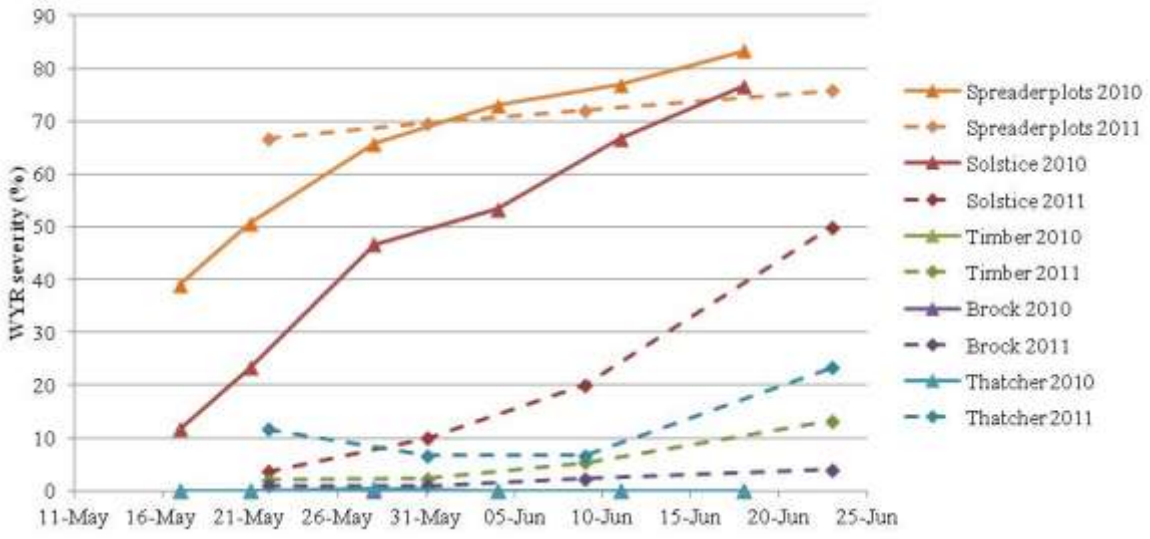

Figure IV-12: Evolution of disease severity (% Cobb scale) in adult plant tests for selected varieties and spreaders plots.

The spreader plots correspond to a mix of cvs Victo and Vuka. The plain lines represent the 2010 trial inoculated with 08/21. The dashed lines represent the 2011 trial inoculated with three Pst isolates 08/21, 03/07, 08/501.

3.3.2 APR with Solstice isolate 08/21

3.3.2.1 Virulence present within the field trial

The YR panel was evaluated against Solstice race 08/21 in an inoculated field trial in 2010. A high infection of Solstice was observed (Figure IV-12). No symptoms were observed on Brock, Timber and lines with *Yr7* such as Thatcher and Lee. Therefore the isolate present within the trial is likely to be 08/21 inoculated, additionally no obvious contamination from natural Pst races was revealed based on differential hosts sown within the trial. Virulence test of Pst isolated from spreader leaves showed a similar virulence profile to 08/21, the test showed virulence for *Yr* genes *1,2,3,4,6,9,17,20,25,32,Sd* and avirulence for *Yr* genes *5,7,8,10,15,SP*, intermediate response were observed for *Yr27* and *YrA*.

The trials included also differential lines for the adult plant resistance. Isogenic lines *Yr18*/6* AvS and Jupateco R (*Yr18*) presented an infection severity between 70 to 90% (Cobb scale) on the 4 June 2010 (middle scoring date) and then dried quickly as they are spring varieties and flowered earlier than the YR panel lines. Those observations highlighted the low efficiency of *Yr18* against UK isolates when included singularly in cv.

The adult plant resistance genes *Yr11*, *Yr12*, *Yr13* and *Yr14* described on European wheat in the UK in the 60 and 70s are most likely overcome by 08/21 as high level of infection were observed at the end of June on variety supposed to carry those genes: Joss Cambier and Heine VII for *Yr11*, Mega and Nord Desprez for *Yr12*, Maris Huntsman and Kinsman for *Yr13*, Avalon and Hobbit for *Yr14*.

3.3.2.2 Severity score and host response

The progression of the disease was followed between 17 May and 18 June, six severity scores using the Cobb scale and three host response scores have been collected over this period of time. Although we disposed of six scores for severity, the analysis focused on three scores: the first from the 17 May, the intermediate from the 4 June and the last from the 18 June.

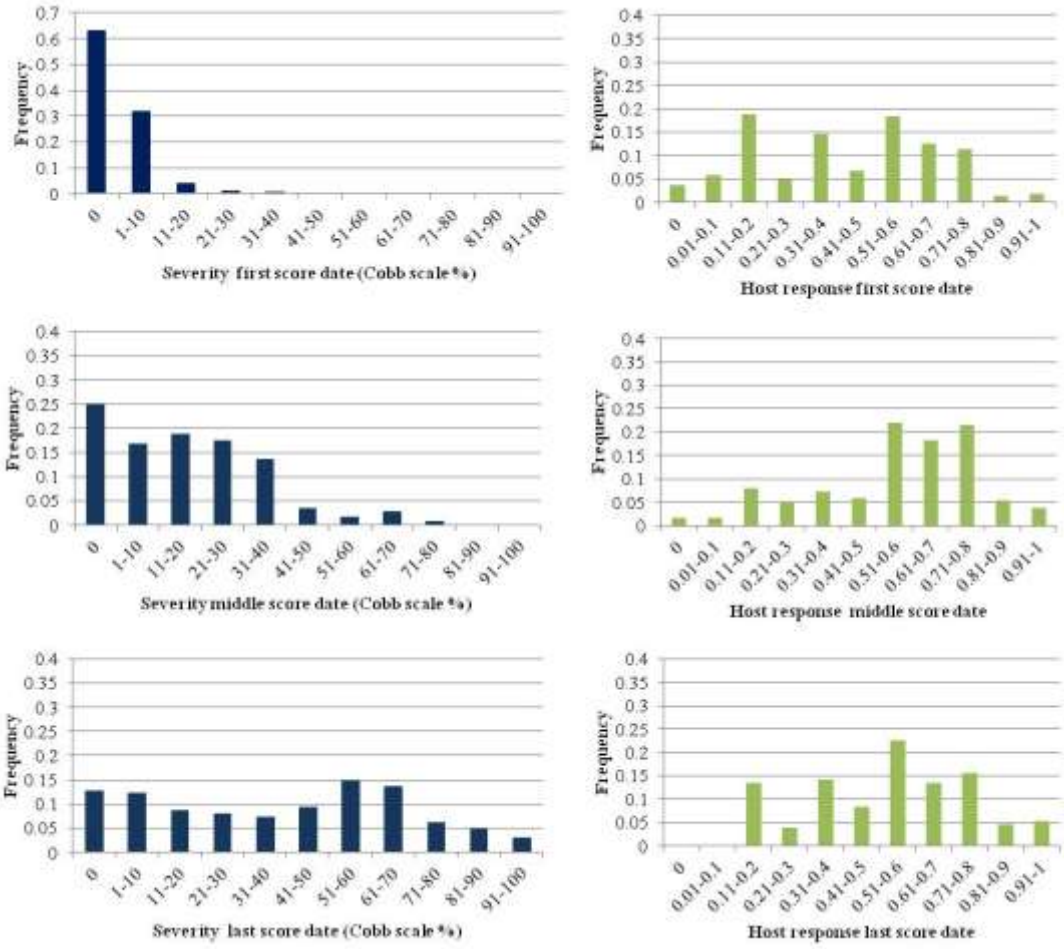

Figure IV-13: Distribution of average severity scores and host response for three dates of scoring in inoculated trial with 08/21.

The severity scores evolved greatly between the 30 days of scoring (Figure IV-13). The first scoring date showed a limited rust severity within the trial with an average of 2.2%, the scores spread from 0 to 40%. The middle date showed intermediate rust severity with an average of 17.9% and the scores went from 0 to 90%. The final scoring presented a high infection of 39.3%; the scores were between 0 and 100%. Based on the final score, the YR panel presented a homogenous distribution of WYR severity, 94 varieties are highly resistant to moderately resistant (severity≤20%), 137 varieties are moderately susceptible to highly susceptible (severity ≥50%).

The analysis of variance based on REML as implemented in GenStat 13th showed significant differences in severity ($p<0.001$) between the YR panel lines across the three scoring dates (Table IV-15). A replicate effect was exhibited for the intermediate score dates ($p<0.001$) and the last score date ($p=0.007$), probably reflecting variation in inoculums density. The heritability value increased from 0.83 to 0.98 and 0.96 between the first and middle/end score dates reflecting a better establishment of the disease at the later dates. No evidence of the influence of the plant stage GS45 on the disease score was revealed by the analysis, thus the covariate GS45 was removed from the model to obtain an adjusted mean.

Table IV-15: Analyse of variance of rust severity (% in Cobb scale) from adult plant test inoculated with Pst 08/21

(1) numerator degree of freedom; **(2)** significance probability from Wald tests based on fixed effect model; **(3)** estimate of variance component from random model; **(4)** standard error based on random model; **(5)** heritability based on simplified random model. – indicated the factor presented a negative variance component estimate, thus was remove from the random model.

Score	**Source**	***n.d.f*(1)**	***F.pr.*(2)**	**Est.** (3)	**s.e**(4)	**H**$^{2(5)}$
Severity	Variety	*292*	*<0.001*	0.09706	0.01012	0.825
First	Block	*2*	*0.071*	0.00037	0.00061	
score	GS45	*1*	*0.265*	-	-	
Log(x+1)	Residual			0.0619	0.0039	
Severity	Variety	*292*	*<0.001*	0.3787	0.03187	0.971
Middle	Block	*2*	*<0.001*	0.00103	0.00117	
score	GS45	*1*	*0.142*	0.00002	0.00005	
Log(x+1)	Residual			0.0338	0.00213	
Severity	Variety	*292*	*<0.001*	0.3953	0.03441	0.957
Last	Block	*2*	*0.007*	0.00083	0.00104	
score	GS45	*1*	*0.639*	-	-	
Log(x+1)	Residual			0.0533	0.00335	

The field host response evolved as the disease progressed during the season (Figure IV-13). Many varieties were classified of resistant (0.2) to moderately R (0.4) in the first scoring date. As more sporulating lesions appeared in June, many lines passed in the classes of intermediate (0.6) to moderate susceptible (0.8). The last scoring date is marked by a regression of lines in classes intermediate to moderately susceptible as the scores focused on the top leaves generally healthier. Only rare lines in the YR panel displayed a totally immune HostR (0), for the last scoring date, only the cvs. Ochre, Hurley, Benedict, Trend and Vector were showing an immune response in all blocks. On the opposite side of the scale, rare were the cvs showing a susceptible HostR(S), only cvs. Jacadi, Tilburi, Lynx (afp 704), Agami, Slejpner and Axial exhibit a constant susceptible response within the trial. It indicates that the YR panel lines, outside the five cited, present some source of resistance against Pst 08/21.

Comparing lines over three score dates, significant differences in HostR (p = <0.001) were observed amongst the YR panel (Table IV-16). No significant replicate effect was shown. The plant stage GS45 did not appeared to influence significantly the HostR. A similar pattern was observed for the heritability of HostR scores as for severity scores, the heritability increased between the first and the second /third scores.

Table IV-16: Analyse of variance of host response from adult plant test inoculated with Pst 08/21

(1) numerator degree of freedom; **(2)** significance probability from Wald tests based on fixed effect model; **(3)** estimate of variance component from random model; **(4)** standard error based on random model; **(5)** heritability based on a simplified random model. – indicated the factor presented a negative variance component estimate, thus was remove from the random model.

Score	**Source**	***n.d.f*(1)**	***F.pr.*(2)**	**Est.** (3)	**s.e**(4)	**H**2(5)
Host R.	Variety	*292*	*<0.001*	0.04823	0.00516	0.804
First	Block	*2*	*0.063*	0.00028	0.00042	
Score	GS45	*1*	*0.179*	0.00003	0.00005	
	Residual			0.0353	0.00222	
Host R.	Variety	*292*	*<0.001*	0.04768	0.00437	0.915
Middle	Block	*2*	*0.06*	0.00011	0.00016	
Score	GS45	*1*	*0.101*	0.00001	0.00002	
	Residual			0.0133	0.00083	
Host R.	Variety	*292*	*<0.001*	0.04382	0.00402	0.915
Last	Block	*2*	*0.691*	-	-	
Score	GS45	*1*	*0.182*	0	0.00001	
	Residual			0.0121	0.00076	

3.3.2.3 AUDPCr

The AUDPC was calculated based on six scoring dates between 17 May and 18 June 2010 and divided by the average AUDPC of the spreader plots (AUDPC=2250) to obtained the relative AUDPC (AUDPCr). Only cv. AC Barrie and Oakley presented a higher AUDPC respectively 2307 and 2256. AC Barrie is a spring varieties from Canada with known high susceptibility, while Oakley is a UK variety also recognized for its susceptibility to UK rust isolates. The distribution of AUDPCr is bimodal, characteristic of the presence of qualitative resistance for the lower scores and more quantitative resistances for the higher scores (Figure IV-14). 67 varieties presented an AUDPCr inferior to 0.1. Among them we found varieties with seedling resistant i.e. Timber, Cyber, Ekla, Buster, Cadenza, Ochre, Malacca. We also recognized long lasting resistant lines with APR such as Deben, Camp Remy and Claire.

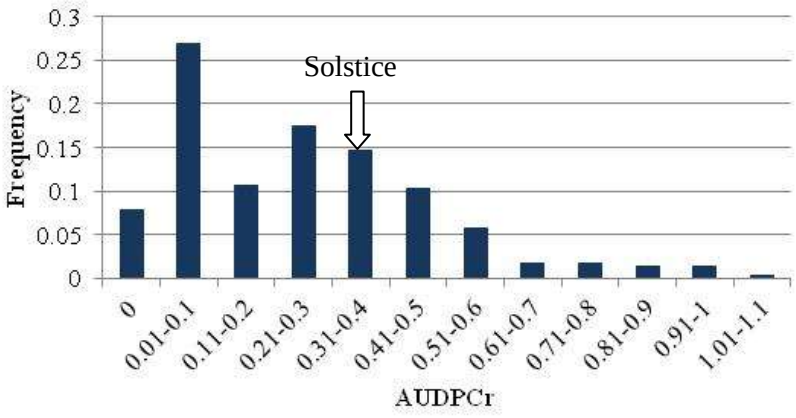

Figure IV-14: Distribution of Area under the disease progress curve relative calculated between 17 May and 18 June in inoculated trial with Pst 08/21.

The analysis of variance on the AUDPCr score showed significant differences between lines of the YR panel ($p<0.001$). A significant replicate effect was also highlighted. No evidence of GS45 influence on the AUDPCr score was demonstrated. The heritability of AUDPCr was particularly high (0.97).

Table IV-17: Analyse of variance of AUDPCr from adult plant test inoculated with Pst 08/21

(1) numerator degree of freedom; (2) significance probability from Wald tests based on fixed effect model; (3) estimate of variance component from random model; (4) standard error based on random model; (5) heritability based on simplified random model.

Score	Source	*n.d.f*(1)	*F.pr.*(2)	Est. (3)	s.e(4)	H2(5)
AUDPCr	Variety	*292*	*<0.001*	0.049957	0.004596	0.968
	Block	*2*	*<0.001*	0.000239	0.000259	
	GS45	*1*	*0.175*	0.000001	0.000005	
	Residual			0.00491	0.00031	

3.3.2.4 Correlation between rust assessments

The correlation coefficient (r^2) was calculated between all the WYR scores collected in the field trial 2010 (Table IV-18). The correlation within severity scores decrease over time. Similar pattern is observed within HostR scores. Generally we found a best correlation between middle and end date than between start and middle date which reflect the low yellow rust infection at the first date. The correlation between the HostR score and the severity scores progressed from 0.14 to 0.70 over the three dates scored in parallel of the rust epidemic development.

We remarked a particularly high correlation between the AUDPCr and the intermediate severity score of 0.95. The middle severity score could therefore be used instead of AUDPCr to look at slow rusting.

Table IV-18: Correlation coefficient R^2 between yellow rusts assessments in inoculated trial with 08/21.

Sev: severity, HostR: host response, start: score on 17 May 2010, middle: score on 4 June 2010, end: score 18 June.

R^2	Sev. start	Sev. middle	Sev. end	HostR. start	Host R. middle	HostR end	AUDPCr
Sev. start	1						
Sev. middle	0.432	1					
Sev. end	0.228	0.697	1				
Host R. start	0.141	0.399	0.374	1			
Host R. middle	0.147	0.493	0.600	0.344	1		
Host R. end	0.193	0.568	0.704	0.358	0.728	1	
AUDPCr	0.472	0.952	0.813	0.421	0.540	0.630	1

3.3.3 APR with a mix of 3 isolates 08/21, 03/07 and 08/501

3.3.3.1 Virulence within the field trial

The YR panel was evaluated against a mix of three Pst isolates 08/21, 03/7, 08/501 in an inoculated field trial in 2011. Those three isolates were chosen to represent a wide range of virulence. The Brock race 03/7 has the virulence for *Yr7* and APR *Yr14* and the Timber race 08/501 is the virulent on Timber, which complemented the Solstice race 08/21 already extended virulence profile: *Yr 1,2,3,4,6,9,17,32,Sd,A*, Solstice.

The WYR infection was evaluated between 22 May and 23 June 2011 (Figure IV-12). Symptoms were observed on Brock, Thatcher (differential host for *Yr7*) and Timber, but the severity score were fairly low even for the last scoring date, respectively 4%, 13% and 23%. Virulence on Solstice was observed, the average severity score progressed from 3.6 to 50% at the end score date. Those observations support the presence of an extended set on virulence within the 2011 trial comparing to 2010 as no symptoms were observed on Brock and Timber in 2010. The average level of infection in 2011 was lower than in the field trial 2010, average severity in 2011 was 22% against 39% in 2010 at the end date.

The difference in severity between susceptible varieties Solstice, Timber and Brock may have several origins :1) a difference of fitness between the three isolates, Solstice isolate 08/21 is believed to be more aggressive than other UK isolates, for instance the latent period in our seedling tests appeared generally shorter with 08/21 comparing to the 03/7; 2) an earlier contamination of the field trial by a race similar to Solstice as sporulation pustules were observed on spreader plots prior the field inoculation and the Solstice race was predominant in 2011 within the UK. 3) Timber and Brock may possess additional minor yellow rust resistances not overcome by their respective isolates.

The trials included also differential lines. The presence of virulence against seedling resistance *Yr2, Yr6, Yr7, Yr22, Yr23, Yr25* was confirmed based on the high level of severity observed on Lee and Heines Peko. No virulence was observed on *Yr5* (*T. spelta album*), *Yr8* (*Yr8*/6* AvS and Compair), *Yr10* (Moro) and *Yr15* (Boston).

Additionally Alcedo (APR QTL on 4BL) and Opata (APR *Yr18, Yr27,* APR *Yr30*), two cvs with adult plant resistance were totally resistant, demonstrating the high efficiency of their respective APR combinations against UK isolates. Guardian (APR *Yr13*, APR *Yr29*) was also evaluated and showed resistance (Severity =20%).

We also remarked a low infection on Vilmorin 23 and Hybrid 46 suggesting the presence of efficient APR within those differential hosts.

3.3.3.2 Severity score and host response

The progression of the disease was followed between 22 May and 23 June 2011, four severity scores using the Cobb scale and two HostR scores have been collected over this

period of time. Although we disposed of four scores for severity, the analysis focused on three scores: the first from the 22 May, the intermediate from the 9 June and the last from the 23June. The HostR was scored at the intermediate and last scoring date.

Severity scores in the 2011 field trial progressed between the 30 days of scoring (Figure IV-15). The first scoring date showed a limited rust severity within the trial with an average of 4.5 %, the scores ranged from 0 to 50%. The middle date showed intermediate rust severity with an average of 12.0% and the scores went from 0 to 90%. The final scoring presented an intermediate high infection of 22.0 %, the scores were between 0 and 100%. Based on the final score, the YR panel presented a skewed distribution toward low severity, 181 varieties showed a severity ≤20%, corresponding to a resistant to moderately resistant response. 55 varieties appeared moderately susceptible to highly susceptible (severity ≥50%).

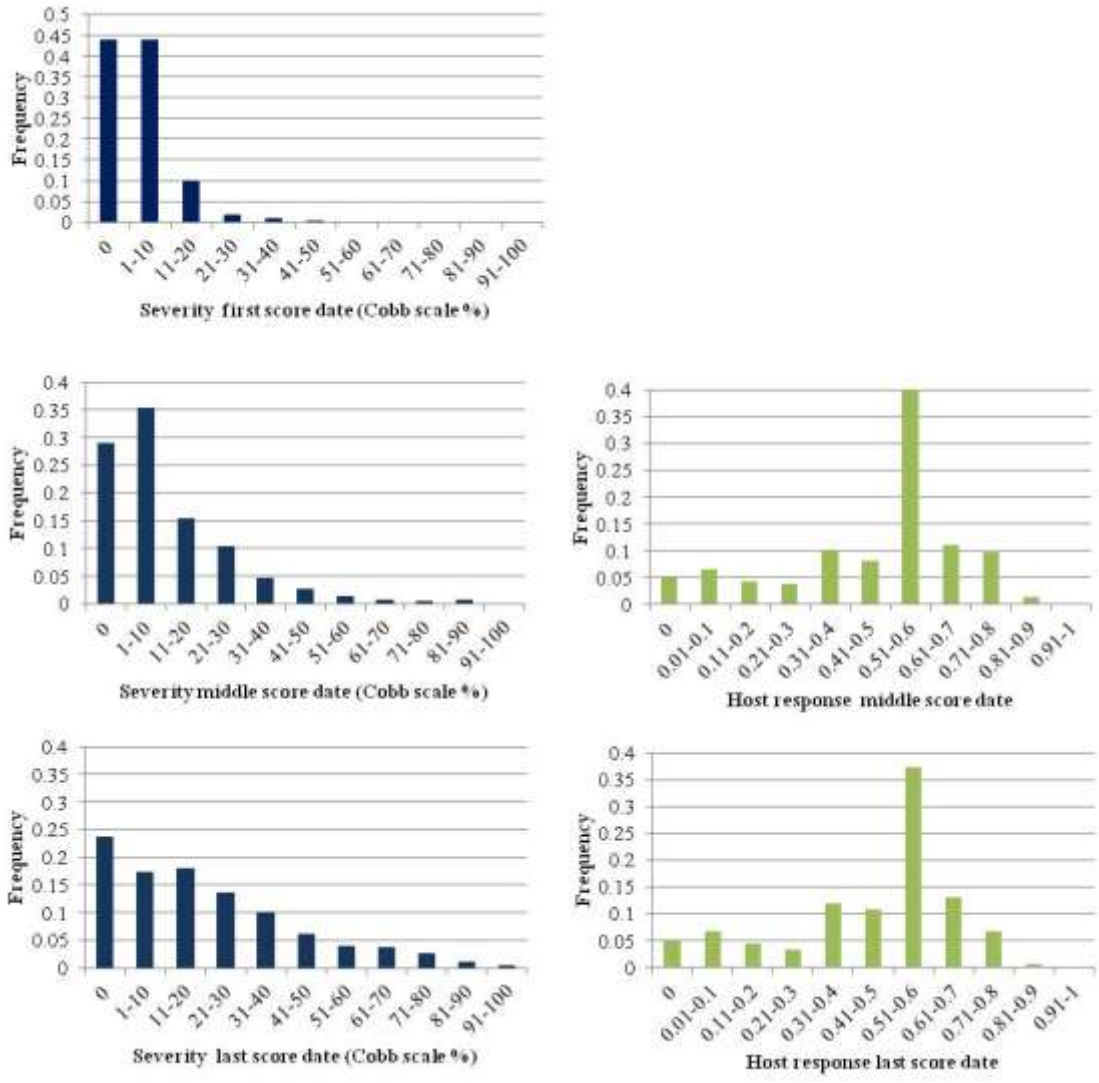

Figure IV-15: Distribution of average severity scores and host response for

three dates of scoring in inoculated trial with three isolates (08/21, 03/7, 08/501).

As the virulence profile of the mix of isolates used for inoculation in 2011 was more extensive than Pst isolate 08/21 alone, a higher number of intermediate to resistant response (equivalent to severity >30) was expected comparing to the 2010 trial. However the intensity of symptoms observed at the end of the epidemic in 2011 was lower than in 2010. The low symptom intensity observed in 2010 was most likely due to the exceptional weather conditions observed in spring 2011, with extensive drought in April and May affecting the plant growth in great extend. The average date for GS50 in 2011 was the 21 May, while in 2010 the average date for an earlier stage GS45 was on the 24 May.

Analysis of variance based on REML showed significant differences in severity ($p<0.001$) between the YR panel line for the three scoring date (Table IV-19). A replicate effect was exhibited for all score date. The heritability value increased between the first and the last score, reflecting a better establishment of the disease at the later dates. Evidence of the effect of the plant stage GS50 were showed at the intermediate scoring date ($p=0.03$).

Table IV-19: Analyse of variance of rust severity from adult plant test inoculated with mix of isolates (08/21, 03/7, 08/501)

(1) numerator degree of freedom; **(2)** significance probability from Wald tests based on fixed effect model; **(3)** estimate of variance component from random model; **(4)** standard error based on random model; **(5)** heritability based on random model.

Score	Source	*n.d.f*[(1)]	*F.pr.*[(2)]	Est.[(3)]	s.e[(4)]	H²[(5)]
Severity	Variety	*307*	*<0.001*	0.16396	0.01458	0.910
First	Block	*2*	*<0.001*	0.00114	0.0013	
score	GS45	*1*	*0.079*	0.00002	0.00007	
Log(x+1)	Residual			0.0483	0.00277	
Severity	Variety	*307*	*<0.001*	0.25525	0.02343	0.950
Middle	Block	*2*	*<0.001*	0.00113	0.00127	
score	GS45	*1*	*0.034*	0.00011	0.0002	
Log(x+1)	Residual			0.0439	0.00252	
Severity	Variety	*307*	*<0.001*	0.37531	0.03129	0.969
Last	Block	*2*	*0.018*	0.00036	0.00048	
score	GS45	*1*	*0.925*	-	-	
Log(x+1)	Residual			0.0365	0.00209	

The field HostR was evaluate at the two latest scoring date. The HostR distribution was similar for both scores. For both dates 9 June and 23 June, the average nominal HostR ranged from 0.1 to 0.9

None of the YR panel lines demonstrated a totally susceptible HostR (1) reflecting the presence of minor resistance within almost all YR panel lines. 11 lines showed a constant immune response among them Contender, Malacca, Ochre, Vector and Gatsby. 50 to 51% of the cvs have an average HostR between 0.51 to 0.7 corresponding to an intermediate response (MSMR).

Based on the analysis of variance, significant differences in HostR (p = <0.001) were observed amongst the YR panel (Table IV-20) for both date. No significant replicate effect was shown. The plant stage GS50 did not appeared to influence significantly the HostR. The heritability of HostR was high for both dates (0.89 and 0.91).

Table IV-20: Analyse of variance of host response from adult plant test inoculated with mix of isolates (08/21, 03/7, 08/501)

(1) numerator degree of freedom; **(2)** significance probability from Wald tests based on fixed effect model; **(3)** estimate of variance component from random model; **(4)** standard error based on random model; **(5)** heritability based on random model.

Score	**Source**	***n.d.f*(1)**	***F.pr.*(2)**	**Est.** (3)	**s.e**(4)	**H**2(5)
Host R.	Variety	*307*	*<0.001*	0.04232	0.00385	0.891
Middle	Block	*2*	*0.428*	0	0.00004	
Score	GS50	*1*	*0.351*	-	-	
	Residual			0.0155	0.00088	
Host R.	Variety	*307*	*<0.001*	0.03971	0.00356	0.904
Last	Block	*2*	*0.388*	0	0.00004	
Score	GS50	*1*	*0.898*	-	-	
	Residual			0.0126	0.00072	

3.3.3.3 AUDPCr

The AUDPC was calculated based on four scoring dates between 22 May and 23 June 2011 and divided by the average AUDPC of the spreader plots (AUDPC=2286) to obtained the relative AUDPC (AUDPCr). Only cv. AC Barrie and Slejpner (*Yr9*) presented a higher AUDPC respectively 2650 and 2317. The distribution of AUDPCr is skewed toward low AUDPCr scores (Figure IV-16). 149 varieties presented an AUDPCr inferior to 0.1; among them we found Timber and Brock as well as varieties with seedling resistance and adult resistance similar to 2010 trial.

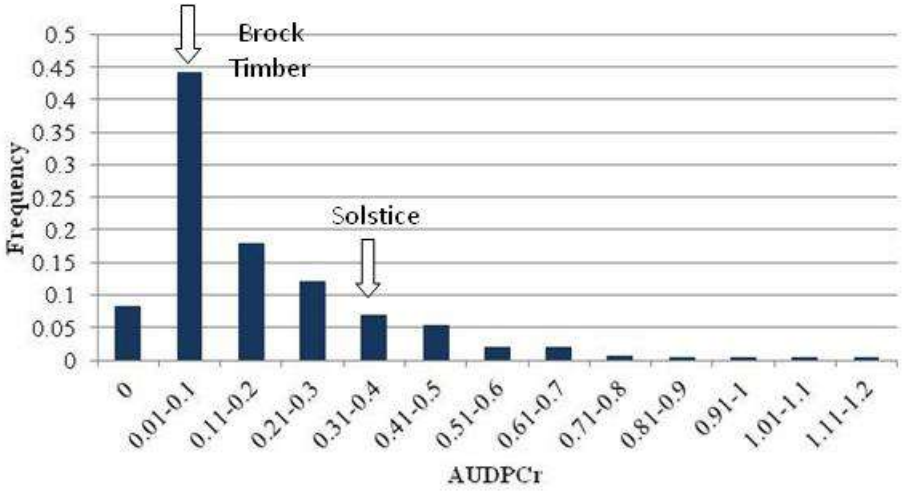

Figure IV-16: Distribution of Area under the disease progress curve relative calculated between 22 May and 23 June in inoculated trial with three isolates (08/21, 03/07, 08/501).

The analysis of variance on the AUDPCr score showed significant differences between lines of the YR panel ($p<0.001$) (Table IV-21). A significant replicate effect was also highlighted. There was no evidence of the effect of the GS50 on the AUDPCr score. The heritability of AUDPCr was particularly high (0.96).

Table IV-21: Analyse of variance of AUDPCr from adult plant test inoculated with mix isolates (08/21, 03/7, 08/501)

(1) numerator degree of freedom; (2) significance probability from Wald tests based on fixed effect model; (3) estimate of variance component from random model; (4) standard error based on random model; (5) heritability based on random model.

Score	Source	*n.d.f*(1)	*F.pr.*(2)	Est.(3)	s.e(4)	H2(5)
AUDPCr	Variety	*307*	*<0.001*	0.034644	0.002926	0.959
	Block	*2*	*<0.001*	0.000096	0.000111	
	GS50	*1*	*0.254*	-	-	
	Residual			0.00467	0.000268	

3.3.3.4 Correlation between rust assessment

The correlation coefficient (r^2) was calculated between all the WYR scores collected in the field trial 2011 (Table IV-22). The correlation within severity scores decrease over time. The correlation value between middle and end date was higher (0.80) than between start and middle date (0.61), reflecting the low yellow rust infection at the first date. The two HostR scores presented an intermediated correlation of 0.62. The correlation between the HostR score and the severity scores progressed from 0.27 to 0.37 from the intermediate to

the last scoring date. The correlation coefficient was particularly high between the AUDPCr and the intermediate severity score of 0.95. The middle severity score could therefore be used instead of AUDPCr to look at slow rusting.

Table IV-22: Correlation coefficient R^2 between yellow rusts assessments in inoculated trial with mix isolates (08/21, 03/7, 08/501).

Sev: severity, Host R: host response, start: score on 22 May 2011, middle: score on 9 June 2011, end: score 23 June 2011.

R^2	Sev start	Sev middle	Sev end	Host R middle	Host R end	AUDPCr
Sev start	1					
Sev middle	0.611	1				
Sev end	0.473	0.796	1			
Host R middle	0.157	0.265	0.364	1		
Host R end	0.132	0.222	0.373	0.616	1	
AUDPCr	0.674	0.965	0.894	0.308	0.279	1

3.4 Comparison of *de novo* adult plant tests

An analysis of variance using REML as implemented in Genstat 13th was carried on the average severity score between three scoring dates from both field trials. The results of the analysis of variance showed as expected a significant tests effect, as the symptoms intensity was generally inferior in the second test. The analysis of variance showed also some interaction between variety and test (Table IV-23) which reflect the presence of additional virulence in 2011 field trial, the presence of potential interaction between rust isolates and generally genotype by environment interactions.

Table IV-23: Analyse of variance of for combined APR field trials

(1) numerator degree of freedom; (2) significance probability from Wald tests based on fixed effect model; (3) estimate of variance component from random model; (4) standard error based on random model; (5) heritability based on random model.

Score	Source	*n.d.f*(1)	*F.pr.*(2)	Est.(3)	s.e(4)	H2(5)
Average	Variety	*323*	*<0.001*	0.23394	0.02163	0.875
severity	Test	*1*	*<0.001*	0.0253	0.03612	
log(x+1)	Test.Var	*276*	*<0.001*	0.05752	0.00577	
	Residual			0.0281	0.00119	

The visual comparison between the severity score at the last scoring date in field 2010 and field 2011 highlighted a few varieties with contradictory responses (Figure IV-17). For instance lines surrounded by a green rectangle in the chart were highly resistant in 2010 but developed symptom in the 2011 field trial as a result of the additional virulence factors.

On the other hand, varieties with intermediate to high level of susceptibility in 2010 trial presented a limited infection in 2011 trial (in the red rectangle in the Figure IV-17), suggesting the presence of negative interactions between Pst isolates and the varieties.

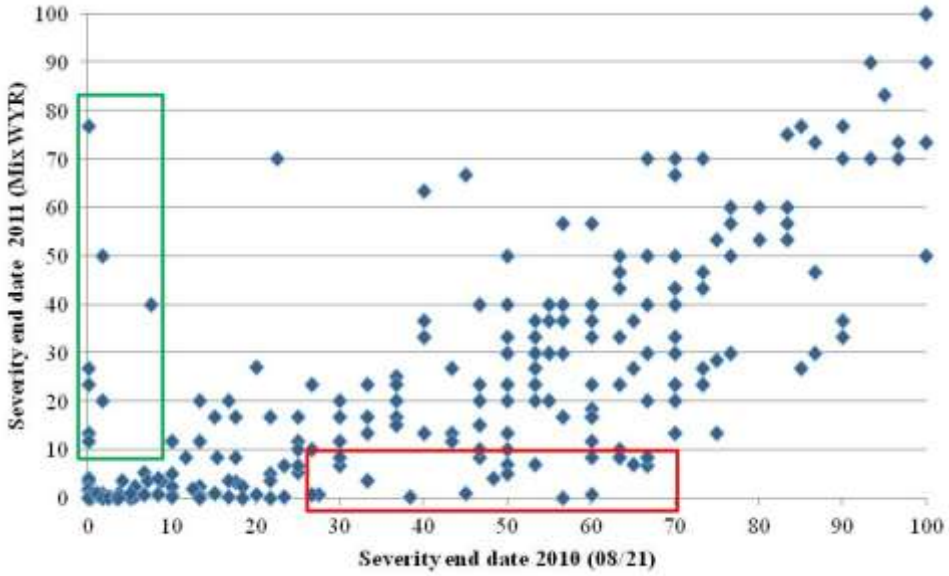

Figure IV-17: Comparison severity last scoring date between field trial 2010 and 2011

The upper left side of the chart (green) includes varieties highly resistant against 08/21 in 2010 and which developed symptoms when inoculated with the mixture of Pst isolates in 2011. The lower right side of the chart (red) includes varieties susceptible against 08/21 in 2010 trial which showed limited symptoms in the 2011 trial inoculated with a mix of isolates

3.5 Comparison of seedling tests and de novo adult plant tests

The Figure IV-18 showed the adjusted IF scores obtained from the two seedling tests inoculated with Pst 08/21 compared to the average adjusted severity scores in field trial 2010 inoculated with Pst 08/21. From the chart we can identify varieties with potential source of adult resistance, the green square contained 34 lines with an IF>6 at seedling stage and an average severity <20% (Table IV-24), some are fairly old lines such as Copain and Maris Huntsman; others are fairly recent such as Brompton, Humber and Award.

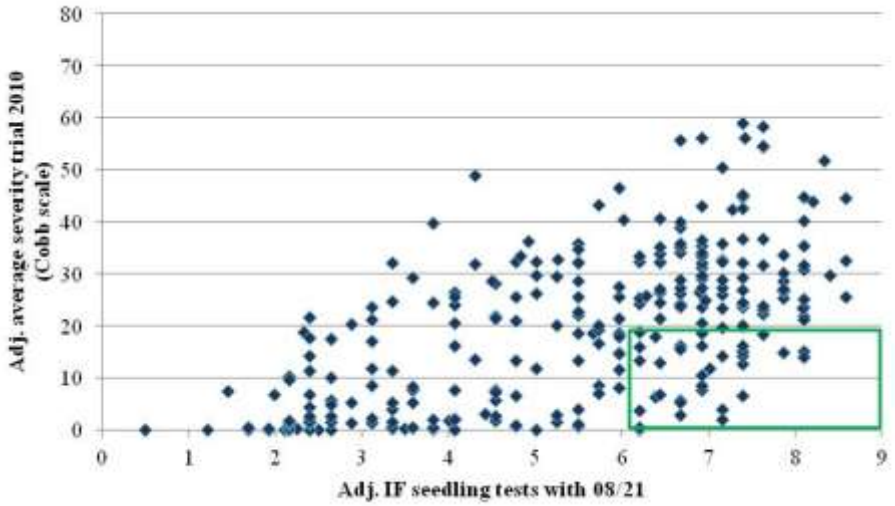

Figure IV-18: Pst 08/21 seedling IF scores versus severity adult plant scores

The plotted scores are adjusted means obtained after fitting a random model as described in section 2.7. The green rectangle identified lines with potential source of adult plant resistance against Pst 08/21.

To obtain a single rust resistance evaluation for both APR tests, the average severity score from both tests were combined and an adjusted mean based on BLUP was obtained after fitting a model including test effect as described in the section 2.7. Similarly, an adjusted IF was obtained from the three seedling tests inoculated with Pst 08/21 and Pst 03/7. The adjusted mean from seedling tests and APR tests are plotted against each others in Figure IV-19, highlighting lines with potential source of adult resistance (orange rectangle) against the mixture of current Pst isolates tested. 30 lines felt in this category, of those 10 were similar to the one identified with only Pst isolate 08/21. The introduction of the IF score against 03/7 is the estimation of the overall seedling resistance score lowered the adjusted IF of many lines with *Yr6* and *Yr9*. Therefore the number of lines with source of adult plant resistance is most likely underestimated.

Note the adjusted scores for each line at seedling and adult plant stage are available in Appendix 4.

Table IV-24: List of varieties with a potential source of adulte resistance effective againt 08/21 (Solstice race):

	Adj. IF seedling tests with Pst 08/21	Adj. average severity trial 2010
Anglo	6.44	6.78
Armada	6.21	0.41
Atla	6.39	6.47
Award	6.92	7.70
Brompton	7.16	4.04
Capnor	6.21	18.85
Carstens V	7.16	19.73
Caxton	6.92	18.59
Charger	6.68	15.53
Chicago	6.68	2.91
Comet	7.02	11.89
Convoy	6.68	5.73
Copain	8.11	14.08
Dart x	6.44	12.98
Drake	6.39	17.99
Electron	6.21	13.45
Explosiv	7.39	16.17
Exsept	6.68	16.12
Falstaff	6.21	16.09
Galahad	7.39	15.04
Galatea	8.11	15.10
Genesis	6.92	10.58
Harrow	7.39	14.25
Humber	6.68	5.24
Longbow	7.39	12.62
Maris Huntsman	6.92	16.17
Matfield	7.63	18.39
Predator	7.16	14.17
Shannon	6.92	8.57
Soissons	7.39	6.63
Spry	7.87	15.01
SW Maxi	6.21	0.30
Tambor	7.16	2.00
Toronto	6.21	3.79
Trend	6.21	0.11

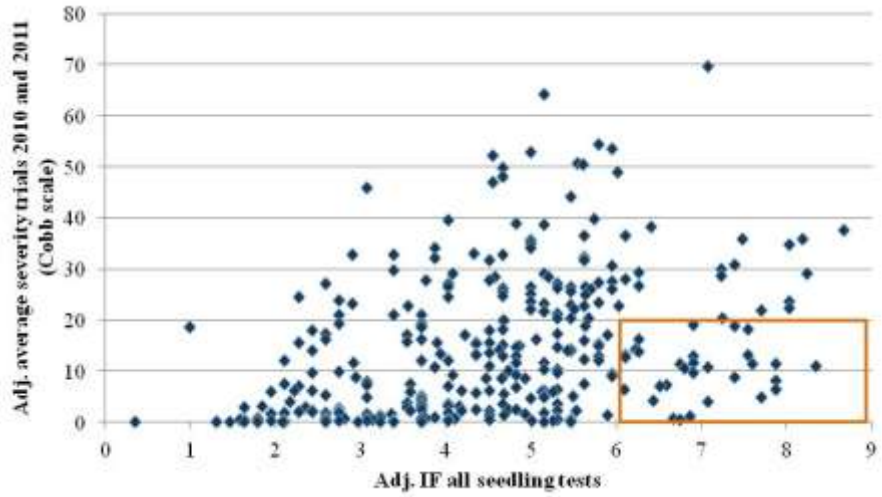

Figure IV-19: Adjusted IF scores from three seedling tests combined versus adjusted severity scores from 2010 and 2011 field trials.

The plotted scores are adjusted means obtained after fitting a random model as described in section 2.7. The orange rectangle identified lines with potential source of adult plant resistance against all isolates tested.

3.6 Comparison of de novo APR test and historical APR

The rust resistance against current Pst isolates evaluated in field trial in 2010 and 2011 were compared visually to historical data resistance scores (see Chapter III for historical data description and adjusted mean calculation) (Figure IV-20). Some similarity were found, many historically susceptible lines appeared also susceptible in the de novo evaluations such as Ac Barrie, Clement, Slejpner, Vuka, Hornet, Blaze, Travix. In addition, many historically resistant lines appeared to have maintained a high level of resistance against current yellow rust isolates, for instance 140 lines have an average severity score <5% in both dataset. The comparison highlighted also some obvious contradictions. Varieties carrying *Yr7* such as Thatcher, Tommy, Camp Remy would be classified as highly resistant based on de novo field evaluation however the historical data showed a lower level of resistance reflecting the lack of strong virulence for *Yr7* in the de novo evaluation. Furthermore, higher susceptibility was observed in many lines when evaluated against current Pst isolates comparing to the historic evaluation, as demonstrated by Vivant, Warrior (afp994), Weston, Dart, Chaucer and Diablo. The increase in susceptibility is supported by a wider virulence profile of current Pst isolates comparing to older isolates evaluated.

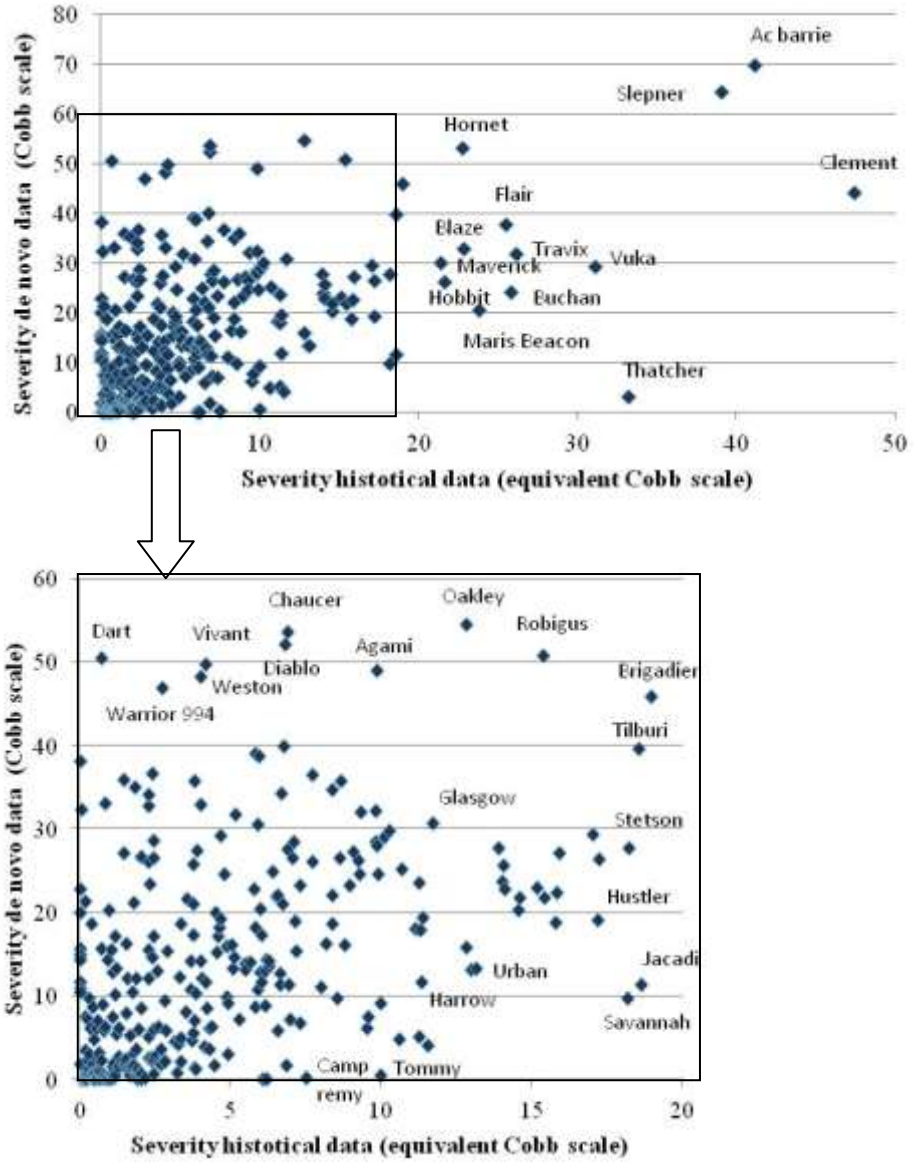

Figure IV-20: Adult plant resistance scores from historical data versus severity in de novo adult plant tests.

The data plotted are adjusted means, de novo data correspond to the average severity between first, intermediate and final score dates for trial 2010 and trial 2011. To ease the comparison, historic data were transformed to match the Cobb scale (equivalent Cobb scale =2.7 x score with International scale).

3.7 Molecular markers

3.7.1 Assays for *Yr5*

Preliminary tests with primer pair STS-9 / STS-10 provided weak amplifications. Therefore, this primer pair was discarded and replaced by STS-7 / STS-10. Similar problem

with the primer pair STS-9 / STS-10 have also been observed in other laboratory as stated in the web site http://maswheat.ucdavis.edu.

Using primer pair STS-7/ STS-10, several differential hosts were evaluated. A great diversity of restriction profiles were observed on agarose gel as illustrated in Figure IV-21. Unfortunately, none of the band observed were specific of lines with *Yr5* e.g. isogenic line *Yr5*/6* AvS and *T. spelta* Album. For instance, Heines Peko, Nord Desprez, Cappelle Desprez and Heines Kolben presented similar restriction profile on agarose gel to lines with *Yr5*. As Cappelle Desprez is known not to carry *Yr5* and entered in the pedigree of many European wheat, the diagnostics of *Yr5* CAPS marker from Chen et al. (2003b) is most likely to be limited in European wheat.

Figure IV-21: *Yr5* assay with primer pair STS7 / STS 10 after digestion by DpnII on European WYR differential hosts.

Agarose gel 2.5%, 1 :*Yr15*/6* AvS; 2: *Yr18*/6* AvS; 3: Chinese 166 ; 4: Compair; 5: Heines Peko, 6: Heines VII; 7: Kavkaz; 8: Kalyansona; 9: Nord Desprez; 10: Reichersberg; 11: Riebesel 47-51; 12: VPM1; 13: Strubes Dickkopf; 14: Suwon/Omar ; 15: Minister; 16: Jupateco R; 17: *Yr5*/6* AvS; 18: *T. Spelta* Album; 19: Heines Kolben; 20: Cappelle Desprez; 21:Hybrid 46; 22: Moro; 23: Lee; 24: Vilmorin 23; L: HyperLadder IV (Bioline).

Using an ABI 3730xl analyser, the YR panel cultivars and WYR differential hosts were classified based on the size of the restriction fragment including the reverse primer Sequence STS-10. Only two fragment sizes were identified using GeneMapper V.4. One fragment was evaluated to be 285bp and was found in *T. spelta* Album and the differential hosts presenting a similar profile to *T. spelta* Album on the agarose gel. The alternative fragment size was 98bp and was found among others in Heines VII, Compair and VPM 1. The varieties of the YR panel are distributed almost equally in the two classes; the 185bp band was present for 149 lines while the 98bp band was present in 147 lines (Table IV-25). The 98bp and 285bp fragments detected by ABI analysis are most likely equivalent to the 102 and 289 bp fragments described by Chen et al. (2003b) minus the 4bp included in the restriction site of DpnII (GATC).

Table IV-25: Distribution of major restriction fragments obtained after digestion by DpnII of amplification using STS-7 /STS-10 primers in YR panel

	Band 285pb	Band 98bp	None detected
Varieties in YR panel	149	147	31

The primer pair S19M93-100 F / R from Smith et al. (2007) was also tested on differential hosts. As expected a 100bp fragment was amplified in lines with *Yr5* e.g. isogenic line *Yr5*/6* AvS and *T. spelta* Album. Similar bands were also amplified in many other differential hosts that do not carry *Yr5 (*Figure IV-22*).* The amplification of 100bp fragments in lines fairly common in pedigree of European wheat Such as Cappelle Desprez and Hybrid 46 render this marker useless to identify *Yr5* within the YR panel. Therefore the STS marker S19M93-100 has not been tested on the complete panel.

Figure IV-22: *Yr5* assay with primer pair S19M93-100 F/R on WYR European differential hosts

Agarose gel 2%, 1 :*Yr15*/6* AvS; 2: Chinese 166 ; 3: Heines Peko; 4: Kavkaz; 5: Nord Desprez; 6: Riebesel 47-51; 7: Strubes Dickkopf; 8: Minister; 9: *Yr18*/6* AvS; 10: Compair; 11: Heines VII; 12: Kalyansona; 13: Reichersberg; 14: VPM1; 15: Suwon/Omar; 16: Jupateco R; 17: *Yr5*/6* AvS; 18: T. Spelta Album; 19: Heines Kolben; 20: Cappelle Desprez; 21: Hybrid 46; 22: Moro; 23: Lee; 24: Vilmorin 23; L: HyperLadder IV (Bioline).

3.7.2 Assay for *Yr9*

The presence of 1BL.1RS rye translocation including *Yr9* have been evaluated via a multiplex PCR developed by de Froidmont (1998) on European differential hosts. The differential hosts for *Yr9*, Kavkaz and Riebesel 47-51, amplified a 412bp fragment characteristic of the rye ω-Secalin gene located on 1RS. The European WYR differentials which do not have *Yr9* amplified a fragment of 636bp characteristic of the wheat low molecular weight glutenin gene located on 1BS. Only differentials Kalyansona and Jupateco R provided a mixed profile with both bands, most likely due to DNA contamination as all wheat lines studied are inbreed lines and should be homozygous at all locus.

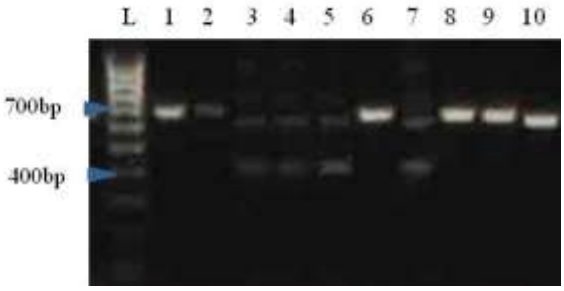

Figure IV-23: Amplification products from 1BL.1RS assay from De Froidmont (1998)

Agarose gel 2.5%. 1: Prophet; 2: Quest; 3: Steadfast; 4: Storm; 5: Weston; 6: Xi19; 7: Kavkaz; 8: Ketchum; 9: Vilmorin 23; 10: VPM 1; L: HyperLadder IV (Bioline).

When applied to the YR panel lines, the multiplex PCR distinguished 104 lines with the 1RS translocation from rye (band 412bp) (Table IV-26), thus carrying *Yr9*. Those varieties represent 32 % of the YR panel. Among the lines amplifying the 412bp product, we recognize varieties such Haven, Hussar, Brigadier and Slejpner known to have *Yr9*. Four lines (Fender, Conqueror, Maverick and Glasgow) appears heterozygote as they amplified a 412bp product and a 636bp. The presence of heterozygote suggests some contaminations of the DNA may have occurred during the extraction and PCR process or it originated from an impure seed source as the YR panel is exclusively composed of inbred lines.

Table IV-26: 1BL.1RS markers distribution for YR panel lines

	Presence of translocation 412bp band	Absence of translocation 636bp band	Heterozygous 412bp and 636bp present	No amplification
Varieties in YR panel	104	213	4	6

3.7.3 Assay for *Yr17*

We used the primers SC-Y15 for *Yr17* developed by Robert et al. (2000) on European WYR differentials hosts and the YR panel lines. The PCR generated a DNA fragment of ~580bp in the VPM 1 line (differential for *Yr17*) and line with Y*r17* such as Hussar, Brigadier, Rendezvous and Madrigal. No fragment was amplified in European WYR differentials with others *Yr* genes. A total of 132 lines from the YR panel amplified the 580 fragments linked to *Yr17* (Table IV-27).

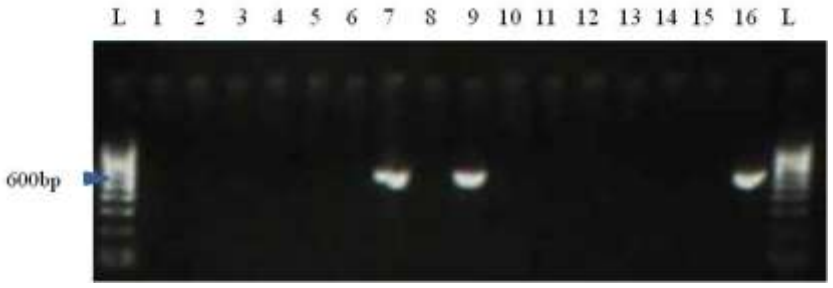

Figure IV-24: Amplification products from SC-Y15

Agarose gel 2%. 1: Windsor; 2: Scorpion 25; 3: Woburn; 4: Hunter; 5: Woodstock; 6: Hunter; 7: Zaka; 8: Cockpit; 9: Wizard afp1828; 10: Riband; 11: Senator; 12: Warlock 24; 13: Armada; 14: negative control (water); 15: Warlock 24; 16: VPM 1; L: HyperLadder IV (Bioline).

Table IV-27: Marker SC-Y15 applied to the YR panel lines

	Presence of 580bp product	Absence of amplification	Not tested
Varieties in YR panel	132	175	20

4 DISCUSSION

4.1 Seedling resistance in YR panel

The YR panel was evaluated as seedling stage against two Pst isolates representing the current virulence in the UK, Brock isolate 03/7 virulent on *Yr7* and Solstice isolate 08/21 virulent on a wide range of *Yr* genes with the exception of Yr7. The test allows us to highlight the presence of seedling resistance within the YR panel as 224 lines tested against 03/7 test and 89 lines in tested against 08/21 had an infection type inferior to 4 (immune to moderately resistant). 63 varieties presented a consistent resistance response (IF <4) against both isolates.

The virulence profile of the isolates differed for *Yr7, Yr6* and *Yr9*, furthermore a difference of response was observed on American line ZACH and IDO3778 carrying *Yr43* and *Yr44* respectively. Extended seedling test confirmed the avirulence of Brock isolate 03/7 for *Yr* genes *5, 6, 8, 9, 10, 15, 24* and *SP* and the avirulence of Solstice isolate 08/21 for *Yr* genes *5, 7, 8, 10, 15, 24 and SP*. The IF type obtained against both Pst isolates complemented by pedigree information and the markers data for *Yr9* and *Yr17* provide an insight concerning the presence of race specific *Yr* genes.

4.1.1 Source of seedling resistance against Brock isolate

4.1.1.1 Yr9

Many qualitative resistances against Brock isolates 03/7 can be explained by the presence of *Yr9* in YR panel lines as confirmed by the molecular assay for the translocation 1BL.1RS. Based on the assay, 98 lines tested against 03/7 have the translocation which contains the resistance gene *Yr9*. 97 lines diagnosed to have *Yr9* based on the 1BL.1RS molecular marker had an average IF inferior to 2. Only Derwent was diagnosed with *Yr9* but present a susceptible reaction (average IF =8). An error in sowing might have occurred.

The presence of the 1BL.1RS was confirmed by the DArT genotype data in 104 YR panel lines (see Chapter 5, 5% of the DArT markers obtained from Triticarte are linked to the presence/absence of the translocation). Cv Alchemy was diagnosed with the 1BL.1RS translocation based on De Froidmont markers but the DArT genotype indicated the contrary, Alchemy presented an intermediate IF of 5 when tested against 03/7 isolate suggesting the absence of *Yr9*. The marker will need to be retested on newly extracted DNA from Alchemy as some contaminations of the DNA may have occurred during the extraction and PCR process explaining the contradiction. The four varieties presenting an heterozygote profile for the marker (Conqueror, Glasgow, Maverick, Fender) do not appear to carry the 1BL.1RS translocation based on the DArT genotype, additionally they presented an intermediate to susceptible IF (4 to 8.5) when tested against 03/7. The presence of a band characteristic of the translocation in the assay is most likely due to DNA contamination.

Most of bread wheat cvs with *Yr9* are believed to derived from *Triticum aestivum x Secale cereale* cv. Petkus lines produced in Germany (Zeller, 1973) such as Aurora, Kavkaz, Benno, Riebesel 47-51. Aurora, Benno and Kavkaz had the translocation 1BL.1RS while Riebesel 47-51 is a substitution line where the chromosome 1R from rye is substitute entirely to the chromosome 1B from wheat. Based on the pedigree information collected for the YR panel, the origin of *Yr9* in the panel can be retraced to three main varieties Haven (11 derivative with *Yr9*), Squadron (25 derivatives with *Yr9*) and Clement (10 derivatives with *Yr9*) (see Appendix 8 for pedigree diagrams). The wheat cv. Clement was assessed in NIAB trials between 1972 and 1975 but never recommended. *Yr9* in Clement most likely originated from Riebesel 47-51 present in its pedigree. The wheat Squadron was developed by Miln Master (now part of Limagrain) and registered in the NL in the 1980 but never recommended little is known about its pedigree. The wheat Haven was developed by the Institute of Plant Science in Cambridge and recommended in the UK between 1990 and 1996; however its HGCA yellow rust resistance score was only 3 (susceptible) due to the high virulence frequencies against *Yr9*. Haven is most likely to have inherited *Yr9* from Riebesel 47-51 present in the pedigree of Hedgehog, one of its parental lines. Hedgehog

appeared also in the pedigree of Admiral (*Yr9*), Hornet (*Yr9*) and Beaver (*Yr9*). Benno was an additional source of *Yr9* in the YR panel for Stetson (*Yr9*), Dean (*Yr9*) and Toronto (*Yr9*).

Yr9 is believed to have been first introduced in the UK in the cultivar Clement in the 1970s, *Yr9* virulent pathotypes were detected in 1975. The deployment of Cvs with *Yr9* in the UK such as Stetson (RL 1983-1984), Slejpner (RL 1986-1991), Hornet (RL 1987-1991), Apollo (1998-1994), Beaver (1990-1995) has been followed by an increase of pathotypes with virulence for this gene, for instance severe epidemics were observed in 1988 and 1989 (Bayles et al., 1990). Therefore *Yr9* is ineffective in the UK; however several current varieties are still carrying the 1BL.1RS translocation. Besides multiple disease resistance (*Yr9, Sr31, Lr26*) that are not always durable, the alien chromatin from rye provide abiotic resistance and some yield advantages (Villareal et al., 1998; Villareal et al., 1994) which could explain its continued use.

4.1.1.2 Yr6

Besides *Yr9*, many YR panel lines are likely to carry *Yr6* and display a resistant infection type against Brock isolate. The race-specific resistance gene *Yr6* was released in the UK cultivars early 1960. *Yr6* is known to be present in two well represented cvs in the pedigree of YR panel lines: Moulin which appeared in the pedigree of 138 YR lines and Norman (*Yr6*) which appeared in 135 YR lines (see Pedigree diagrams in Appendix 8).

Yr6 was also postulated in Hornet (*Yr2, Yr6, Yr9*), Longbow (*Yr1, Yr2, Yr6*), Haven (*Yr6, Yr9*), Lynx afp 856 (*Yr6, Yr9, Yr17*), Madrigal (*Yr6, Yr9, Yr17*), Charger (*Yr3, Yr6, Yr32+*), Encore (*Yr3, Yr6, Yr9, Yr32*), Equinox (*Yr6, Yr9, Yr17*), Hunter (*Yr3, Yr6,Yr9, Yr32*), Rialto (*Yr6, Yr9*), Comet (*Yr3, Yr6, Yr9, Yr32+*), Shango (*Yr4, Yr6*), Cadenza (*Yr6, Yr7*), Riband (*Yr6*) and Spark (*Yr6*) based on multiples seedling tests against Pst isolates with contrasting virulence (Bayles, 2001; Hovmøller, 2001a, 2007; Johnson, 2001; Pathan et al., 2008; Singh et al., 2008). All those lines except Spark and Riband presented a resistant response against Brock isolate 03/7. The difference in isolates and environmental conditions between the present study and previous tests reported by Singh et al. (2008) and Hovmøller (2007) could explain the difference observed. The expression of *Yr6* is known to be influenced by environmental condition (Wellings, 1986), (Dubin et al., 1989) as well as isolates (Elbedewy and Robbelen, 1982). Alternatively an error in seed source could not be excluded.

Virulence against *Yr6* is common on the UK, the pathotypes frequencies varied greatly year to year (see Appendix 5) most likely in association with surface cultivated of wheat carrying *Yr6,* Norman (RL 1981-1994), Longbow (RL 1983-1989), Hornet (RL 1987-1991) (Bayles) and in recent year Solstice (RL 2002-present) (the presence of *Yr6* in Solstice

was postulated from evaluation of double haploid population between Solstice and Robigus). In 2010, pathotypes with virulence for *Yr6* represented 98% of the isolates tested by the UKCPVS. *Yr6* is therefore inefficient against current YR isolates in the UK.

4.1.1.3 Yr17

As the virulence tests of Brock isolate for *Yr17* on several differential hosts did not provide a consistent response and *Yr17* is often present in combination with *Yr6* and/or *Yr9* within cvs, we were not able to conclude whether the presence of *Yr17* within the lines explains part the resistance response observed in many lines tested against Brock isolate.

Yr17 originated from *Aegilops ventricosa*, was transferred to the hexaploid wheat line VPM1 on chromosome 2A in conjunction to resistance genes to leaf rust *Lr37* and stem rust *Sr38* in a cluster (Doussinault et al., 2001). The variety Roazon registered in 1976 in France was the first to carry this cluster. In the UK, *Yr17* was first introduced in cv Rendezvous (RL 1987-1990) then largely deployed in cv Hussar (RL 1992-1999) and Brigadier (RL 1993-1999). Virulent pathotypes were first detected in 1994 in Denmark and UK (Bayles et al., 2000), since pathotypes virulent on *Yr17* have been predominant in the UK.

A SCAR marker developed by Robert et al. (1999) to identify the cluster of gene *Yr17/Lr37/Sr38* was tested on the YR panel. The presence of *Yr17* was postulated in 132 lines (representing 43 % of the 308 lines tested from the YR panel) based on the molecular marker. The diagnostic of *Yr17* by marker was in agreement with previous postulations from Singh et al. (2008), Hovmøller (2007) and Pathan et al. (2008) for 14 cultivars (Hussar, Brigadier, Torfrida, Beaufort , Reaper, Abbot, Equinox, Madrigal, Savannah, Biscay, Smuggler, Andante, Chianti, Prophet), however Caxton and Armada were postulated to carry *Yr17* but did not amplified a specific fragment. As the marker used is dominant, a problem with PCR amplification cannot be excluded. To confirm the negative results, the PCR amplifications will have to be repeated in a second set of DNA extraction. The difference in diagnostic can also be explained by a recombination between the marker and the *Yr17* locus. The marker SCY15 is estimated to be 0.8cm away from the resistance gene *Yr17* by Robert et al. (2000) as one recombinant was found in a F2 population between VPM1 and Thesee.

The result shows that a large part of the YR panel lines have *Yr17* and by extension VMP1 in their pedigree. In spite of the fact that the resistance was overcame mid 1990s, many varieties registered in national list in 2000s for instance Battalion, Hyperion still carry *Yr17*. The continued presence of *Yr17* within current commercialized suggests there is a neutral or positive effect of the translocated fragment from *A, ventricosa* on agronomic value. Dyck and Lukow (1988) showed a higher kernel protein level but lower dough mixing properties. The presence of *Lr37* in association to *Yr17* is another explanation for the

continued presence of *Yr17* in Western Europe as *Lr37* still provide resistance to some leaf rust races and can be used in combination with other rust resistance gene.

The repartition of IF observed in seedling test against Brock isolate depending of genotype score for *Yr9* (based on DArT genotype) and *Yr17* (based on SCAR marker SCY15) is presented in Figure IV-25 and suggest that *Yr17* is generally associated with a higher level of resistance, as *Yr17* in often present in combination with *Yr6* and/or *Yr9*. However 11 lines with *Yr17* showed susceptibility which reflects most probably the ineffectiveness of *Yr17* against Brock isolate instead of unfavourable experimental conditions for the expression of *Yr17* in specific genetic backgrounds as mentioned in the virulence test results on Brock isolates (see section 3.1).

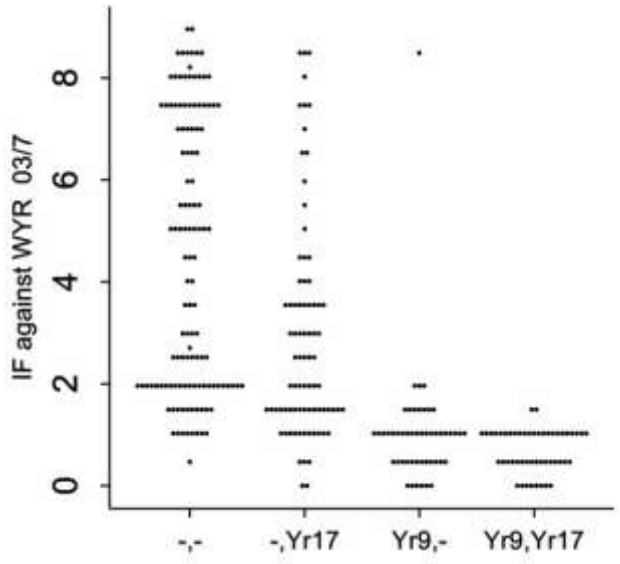

Figure IV-25: Dot plot of infection type observed against Brock isolates 03/7 depending of genotype for *Yr9* and *Yr17*

4.1.1.4 Other source of resistance against Brock isolate

19 YR panel lines were resistant against the Brock isolate (IF 03/7<4) and susceptible against the Solstice isolates (IF 08/21>6) but did not carry *Yr9* and/or *Yr17* (determined with molecular markers). Those lines (Axial, Caxton, Charger, Chicago, Consort, Dickson, Electron, Except, Harbour, Kinsman, Leo x, Longbow, Oakley, Option, QPlus, Robigus, Rosette, Shango and Talon) potentially carry *Yr6* or an uncharacterised resistance gene effective against 03/7 but 08/21. Among them we found lines known to carry *Yr6* such as Kinsman, Charger, Longbow but also lines unlikely to carry Yr6 such Robigus and Consort as they were susceptible in seedling tests carried by the UKCPVS with isolates without *Yr6* virulence.

4.1.2 Source of seedling resistance against Solstice isolate

4.1.3 Yr7

Some qualitative resistances against Solstice isolates 08/21 can be explained by the presence of *Yr7* in YR panel lines alone or in combination with other genes. *Yr7* originated from durum cv Lumillo and was transferred to Thatcher and derivatives cvs Talent, Lee and Hardi. By comparing seedling tests with 03/7 and 08/21, ten lines can be postulated to have *Yr7*: Brock, Camp Remy, Thatcher, Tommy, Cordiale, Ekla, Vilmorin 27, Spark, Vault, Dynamo (IF with 03/7 <6 and IF with 08/21<4) . Brock, Camp Remy, Thatcher and Tommy are known to carry *Yr7*, the remaining lines are likely to carry *Yr7* considering the wild virulence profile of 08/21. Ekla and Vault are likely to have inherited *Yr7* from Talent (*Yr7*) (Figure IV-26) .Cordiale and Spark may have inherited *Yr7* from Cadenza (*Yr6,Yr7*) (Pathan et al., 2008) and Tonic (*Yr7*) (Singh et al., 2008) (Figure IV-27). No link can be made between *Yr7* and Vilmorin 27 and Dynamo. Vilmorin 27 is a fairly old cv. developed prior to Thatcher, so it is unlikely it carry *Yr7*. Vilmorin 27 is also a parental line of Cappelle Desprez. Resistance in those cultivars may be due to uncharacterized resistance gene(s) for which virulence differ between 03/7 and 08/21.

Yr7 is expected to be present in additional YR panel lines in combination with *Yr6* and/or *Yr9*. Cvs carrying those *Yr* genes combinations present a resistant response against both WYR isolates tested. For instance, cv. Cadenza was postulated to carry *Yr6* and *Yr7* by Pathan et al. (2008). Based on pedigree and the seedling tests, Tara is likely to carry *Yr7* in addition to *Yr9*.

The virulence frequency against *Yr7* remained low in the UK, over four decades of virulence survey, the frequency of pathotypes with *Yr7* varied from 0 and 36% between 1967 and 2010 (see Appendix 5). In 2011 and 2012, a spike in *Yr7* virulence was observed by the UKCPVS following the emergence of a new WYR race named Warrior. The Warrior race combined virulence for *Yr6, Yr7, Yr9* and *Yr17*. *Yr7* used singularly proved to be inefficient for many years; however in combination with *Yr6*, it provided a high level of resistance against almost all UK isolates. Only a few Pst isolates from the 1990s presented the virulence combination *Yr6+Yr7*. As today, the Warrior race is predominant in the UK rendering the combination of resistance *Yr6+Yr7* totally inefficient.

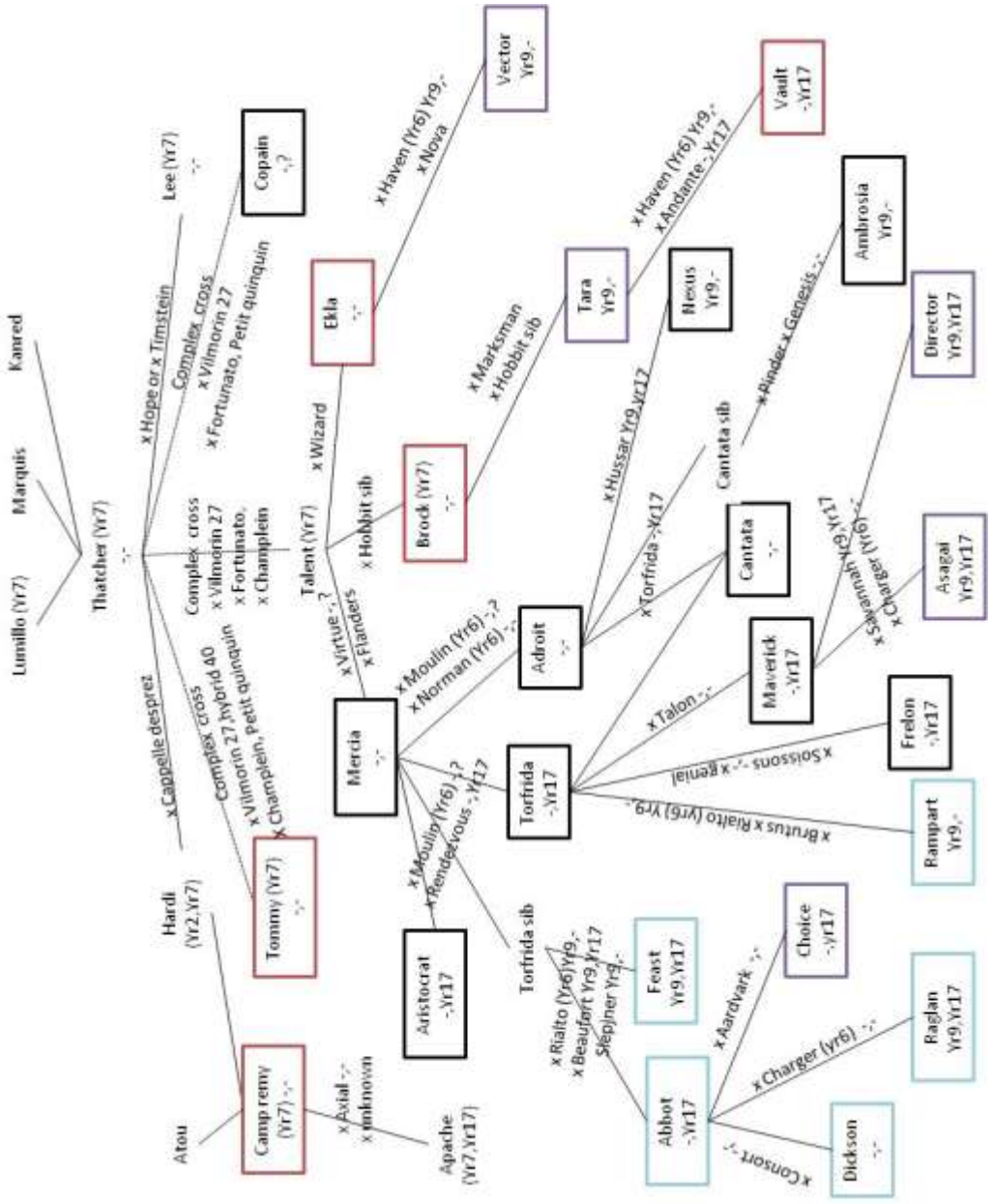

Figure IV-26: Pedigree of cv. Thatcher and descendants

Presence and absence of *Yr9* and *Yr17* based on DArT genotype and *Yr17* assay from Robert et al. (1999) are indicated as followed : "-,-" absence of *Yr9* and *Yr17* , "-,Yr17" absence of *Yr9* and presence of *Yr17*, "Yr9,-" presence of *Yr9* and absence of *Yr17*, "Yr9,Yr17" presence of *Yr9* and *Yr17*, ? for indeterminate; *Yr* genes in parentheses are from Pathan et al. (2008), Hovmøller (2007), Singh et al. (2008), Hovmøller (2001b) and Johnson (2001); cvs are colored based on the IF observed against 03/7 and 08/21 at seedling stage : blue for IF (03/7)<4 (resistant) and IF 08/21>6 (susceptible), violet for IF (03/7)<4 (resistant) and IF 08/21<4 (resistant), red for IF (03/7)>6 (susceptible) and IF 08/21<4 (resistant), green for IF (03/7)>6 (Susceptible) and IF 08/21>6 (susceptible), black for IF between 4 and 6 in one of the test.

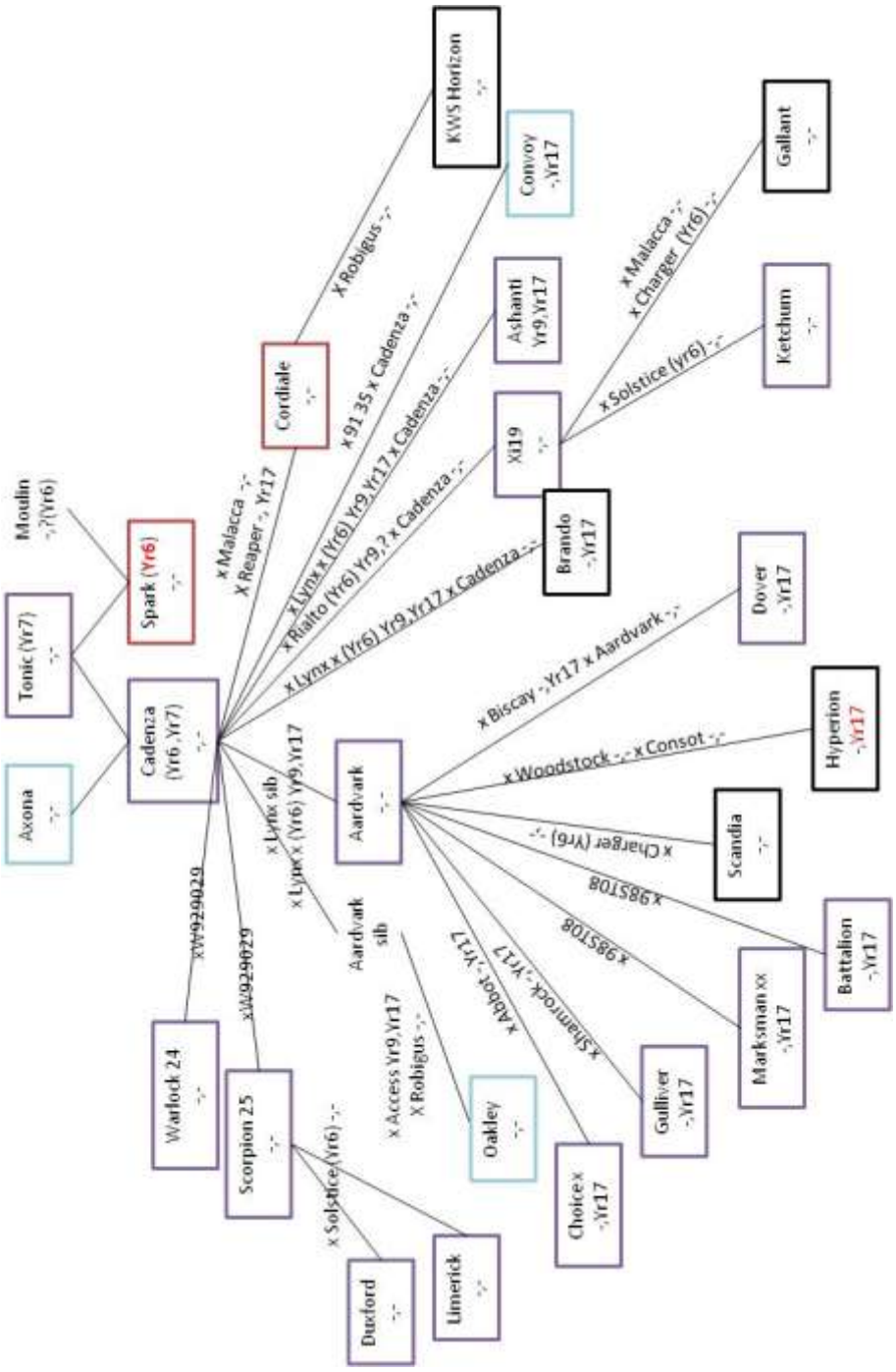

Figure IV-27: Pedigree of cv. Cadenza and descendants

Presence and absence of *Yr9* and *Yr17* based on DArT genotype and *Yr17* assay from Robert et al. (2000) are indicated as followed : "-,-" absence of *Yr9* and *Yr17* , "-,Yr17" absence of *Yr9* and presence of *Yr17*, "Yr9,-" presence of *Yr9* and absence of *Yr17*, "Yr9,Yr17" presence of *Yr9* and *Yr17*, ? for indeterminate; *Yr* genes in parentheses are from Pathan et al. (2008), Hovmøller (2007), Singh et al. (2008), Hovmøller (2001b), and Johnson (2001); cvs are colored based on the IF observed against 03/7 and 08/21 at seedling stage : blue for IF (03/7)<4 (resistant) and IF 08/21>6 (susceptible), violet for IF (03/7)<4 (resistant) and IF 08/21<4 (resistant), red for IF (03/7)>6 (susceptible) and IF 08/21<4 (resistant), green for IF (03/7)>6 (Susceptible) and IF 08/21>6 (susceptible), black for IF between 4 and 6 in one of the test.

4.1.4 Source of seedling resistance against both Pst isolates tested

Thirty five varieties have been found resistant (IF ≤4) against both isolates Brock 03/7 and Solstice 08/21, apart rare case, their resistance genes are unknown.

One of the possibilities is that they carry a combination of resistance genes *Yr7 +Yr6* and *Yr7+Yr9*. This is probably true for a few varieties like Cadenza and Tara (see previous paragraph), however based on pedigree information, *Yr7* is not likely to have been deployed in a wide range of varieties. An evaluation of those varieties with the newly emerged Warrior race from the UK (virulent on *Yr 1, 2, 3, 6, 7, 9, 17, 32, SP*) would help to validate this hypothesis.

The second possibility to explain the constant resistant infection type is the presence of seedling gene *Yr 5, 8, 10, 15, 24, SP* or resistance from cvs Batavia (*Yr33, YrA, YrBat1, YrBat2*), Tres (*YrTr1, YrTr2, Yr32*), Tyee (*YrTye*) or 205 (*Yr45*) as both WYR isolates tested were avirulent on those cvs. However, outside rare occurrences, none of the *Yr* genes cited are known to have been used in UK breeding program. Hovmøller (2007) evaluated 141 European wheats at seedling stage using 16 Pst isolates but found no indication of the presence of *Yr5, Yr7, Yr8, Yr10 and Yr24*.

Yr5 is originated from *Triticum spelta* cv Album and provides a high level of resistance against most WYR races. Yr5 was also reported to be present in several accessions of spelt wheat (Kema, 1992). Virulence to *Yr5* rarely occurs in most wheat producing area. Kema (1992) reported that *Yr5* has been use sporadically in Dutch breeding programs. However, Smith et al. (2007) indicated that *Yr5* was not present in European cultivars. To find if *Yr5* was present in the YR panel, two molecular markers for *Yr5* identified respectively by Smith et al. (2007) and Chen et al. (2003b) in crosses between *T. spelta* cv Album and American cv Lemhi were tested in a subset of the YR panel. As European wheat varieties without *Yr5* e.g. Cappelle Desprez, Nord Desprez, Heines Kolben, Vilmorin 23 amplified similar bands to *T. spelta* Album, we concluded that the two markers tested were not useful to diagnose the presence of *Yr5* in the YR panel. Although the markers developed by Chen et al. (2003b) was not perfect, Zhang et al. (2009) believed the marker would be useful for gene postulation in association studies because of the limited number of original source. An additional marker has been reported for *Yr5* by Sun et al. (2002) and could be tested on the YR panel. Nevertheless the SSR marker *Xgwm501* reported was estimated to be 10.5 to 13.3cM away from the resistance gene loci, therefore many recombinations could occur during the breeding process reducing the diagnostic potential.

Yr8 originally from *Aegilops comosa* was translocated on chromosome 2D in wheat Compair (2D/2M). Additional translocation lines 2A/2M, 2D/2M and 3D/2M were obtained from Chinese spring, Maris Widgeon and Hobbit-sib (Miller et al., 1988),(McIntosh et al., 1982). Compair was deployed in the UK in 1960s, 10 years later the efficiency of the resistance from *Yr8* was re-assessed as a few virulent pathogens have been identified in the UK. Since, *Yr8* was not reported in further cvs from the UK. Virulence for *Yr8* was also detected in the past decade in the south of France (De Vallavieille-Pope et al., 2012) and more recently in Sweden, Denmark and Germany (eurowheat.org).

Yr10 originated from *T. spelta* cv 415 and was deployed in the American cv Moro in the 1960s, soon after virulent pathotypes were reported in the USA. Pathotypes virulent on *Yr10* were also detected in recent years in Denmark, Germany and Sweden (eurowheat.org). There is no report of use of *Yr10* in European varieties (Smith et al., 2002).

Yr15 originated from *Triticum diccocoides* accession G25 and was identified in 1970s (Gerechter-Amitai et al., 1989). *Yr15* is known to be present in a few Danish lines and French lines Boston, Cortez, Agrestis and Legron (Hovmøller, 2007), apart from Ochre (pedigree Cortez x 98ST31) none of the YR lines seems to be related to those varieties. *Yr15* is effective over a wide range of yellow rust race worldwide (Chen, 2005), however isolates virulent on *Yr15* were detected in Denmark in early 2000 (Hovmøller and Justesen, 2007a).

YrSP was identified in English cultivar Spadlings prolific from the 19th century. Pathotype virulent on Spalding prolific have been reported in most geographic area. In the UK, the virulence for *YrSP* was not tested systematically by the UKCPVS apart between 1998 and 2005. Only one isolate virulent on Spalding prolific has been identified in the 2003 survey. It is not know if Spalding has been use as source of resistance in European breeding programs. Allelic tests showed that *YrSP* located on chromosome 2B is likely to be allelic to *Yr5* and *Yr7* (Zhang et al., 2009).

Yr24 is derived from a durum wheat K733 and was identified in a synthetic wheat from Australia (McIntosh and Lagudah, 2000). No link can be made between the synthetic wheat from Australia and the YR panel lines therefore, *Yr24* is not likely to directly explain the resistance observed. However, *Yr24* was described recently, many virulent pathotypes for this gene are already reported, for instance in China (Liu et al., 2010), East Africa (Hovmoller, 2012), and USA (Wan and Chen, 2012). Prior the present study, no UK Pst isolate has been tested for their virulence on *Yr24*, thus it is not known *Yr24 r*emained efficient in the UK against a wide range of race. Li et al. (2006a) reported that *Yr24* is likely allelic to *Yr26* and *YrCH42*, based on allelic tests.

Both Pst isolates 03/7 and 08/21 were also avirulent on Australian cv. Batavia (*Yr33, YrA, YrBat1, YrBat2*) and American cvs Tres (*YrTr1, YrTr2, Yr32*), Tyee (*YrTye*) and 205 (*Yr45*). None of those varieties are known to have been use in European breeding programs.

Thus resistance genes *Yr33, YrBat1, YrBat2, YrTr1, YrTr2, YrTye, Yr45* are not likely to be present in the YR panel.

Molecular markers have been reported for many WYR resistance genes including *Yr5, Yr8, Yr10, Yr15* and *Yr24* (Sun et al., 2002), (Chen and Zhao, 2007), (Wang et al., 2002),(Peng et al., 2000),(Peng et al., 1999), and could be tested to confirm the presence of specific resistance gene within the YR panel and their frequencies.

The third possibility to explain the consistent resistance against Brock and Solstice isolates is the presence of uncharacterised resistance gene(s) or gene combination(s) outside of the *Yr* genes known to be avirulent on both isolates. Therefore some of those resistant cultivars represent potentially new sources of resistance.

4.2 Adult plant resistance in YR panel

Through two year of inoculated field trials, we collected two separate phenotypes related to resistance, first the descriptive host response score which relates to the defence response induced in the plant. Secondly, the quantitative severity (or percent of infection) was scored several times during the season. The severity score can be related to the fitness of the pathogen and the intensity /timing of the plant response which restrain the development of the pathogen. Based on the severity scores, the AUDPCr was calculated for each cv. in order to identify slow rusting resistance, those classes of resistance retard the pathogen development and delay spore production. In both years, in our experiment the AUDPCr was highly correlate to the intermediate severity score, therefore the intermediate severity score can be examine to identify slow rusting resistance.

Looking at the host response of the last scoring date, 84 cvs presented constantly an immune to moderately resistant response (average HostR < 0.5) in 2010 and 2011 with reduce uredias or no uredias. Of the 84, 56 carried a seedling resistance against Solstice isolate explaining the resistant host response observed. 10 were totally susceptible at seedling stage (e.g. Toronto, Armada, Electron, Chicago, Convoy, Award, Tambor, Brompton, Explosiv and Maris Freeman) and 18 presented an intermediate response at seedling stage, therefore suggesting the presence of highly efficient APR in those 28 cultivars. For the cultivars with intermediate response at seedling stage, the resistance observed in the field could have resulted from either APR or an unidentified seedling resistance (not fully expressed in our experimental condition) or both.

Looking at severity score at the end of the season and AUDPCr, some level of resistance was observed at adult stage in most of cvs. Only AC Barrie presented an AUDPCr

superior to 1 (meaning an infection superior to the spreader plots) and can be qualified of highly susceptible. A limited number of cvs were susceptible based on their AUDPCr, as only 20 cvs in 2010 and 12 cvs in 2011 had an AUDPCr superior to 0.6. On the other hand, many cvs were resistant to highly resistant, 119 cv presented an AUDPCr ≤ 0.2 both years and 87 cvs presented a percentage of final infection ≤20% in both field trials. Besides lines carrying seedling resistance identified in seedling tests, 38 to 43 % of the resistant lines observed in the field showed on intermediate to susceptible IF at seedling stage (IF<6). Respectively 12 and 23 cvs susceptible at seedling stage were resistant in the field based on severity and AUDPCr. Therefore we highlighted the presence of slow rusting resistance within the YR panel.

Pathan et al. (2008) evaluated European cultivars at seedling stage and adult plant stage against an Australian isolate of *Puccinia striiformis* f. sp. *tritici*, they observed also a high level of adult plant resistance in cvs with intermediate response (i.e. Monopol, Alcedo, Transit) to susceptible response (i.e. Astron, Charger, Spark, Moulin, Cadenza) at seedling stage. Similarly, Hovmøller (2007) evaluated 141 European wheats against several Pst isolates at seedling and adult plant stage and identified medium level of APR in several cvs among them were lines in the YR panel Hereward, Windsor, Shango, Reaper, Hussar.

Johnson (2000) highlighted the presence of durable resistance in several European cultivars in 1980 (Hybride de Bersee, Bouquet, Cappelle Desprez, Caribo, Holdfast, Hybrid 46, Little Joss, Maris Widgeon), many of which entered in the pedigree of YR panel lines.

In recent years, several genetic studied focused on durable resistance in European cultivars and highlighted many QTL for APR in European lines related to the YR panel.

Using derived lines from Cappelle Desprez excluding the seedling resistance *Yr3* (*Yr3a+Yr4a*), Agenbag et al. (2012) investigated the genetic basis of APR present in Cappelle Desprez. They identified four APR QTLs (2AS, 2DS, 5B, 6DL) effective against South African Pst isolates. The QTL on 2DS is believed to be the APR gene *Yr16*. Cappelle Desprez is present in pedigree of at least 232 YR panel lines (estimated from pedigree viewer), it is a parental line of Maris Marksman, Maris Beacon, Mega, Bouquet, Kador, Joss Cambier, Atou, Arminda and Hobbit and therefore appears in all their descendants (see pedigree diagrams in Appendix 8). Cappelle Desprez showed a low level of resistance in the field at the end of 2010 but a high level in 2011. Therefore it is likely some of those QTLs are present in the YR panel and participate to resistance in the field against current UK isolates.

Mallard et al. (2005) identified five QTL for APR in Camp Remy (1BS, 2AL, 2DS, 5BL, 5BL). In our evaluation Camp Remy was resistant over the two years of experimentation. Camp Remy derived from a cross including two well represented cvs in the pedigree of YR panel e.g. Cappelle Desprez and Thatcher (see Figure IV-26). Therefore

APR present in Camp Remy may also be found in many YR panel lines. The QTL on 2DS is thought to be APR gene *Yr16* inherited from Cappelle Desprez.

Paillard et al. (2012) identified one APR QTL in Apache on chromosome 4B Apache derived from Camp Remy, Axial and an additional unknown component. Axial was highly susceptible in field trial 2010 while it showed a low level of resistance in 2011

Dedryver et al. (2009) identified three stable QTLs for APR in cv Renan (2BS, 3BS, 6B) and one in Recital (3DS). Some parental lines of Renan and Recital are included in the pedigree of the YR panel e.g. Maris Huntsman and VPM1 for Renan, Heines VII and Tadorna for Recital; thus some of those APR QTL could be present in the YR panel.

Jagger et al. (2011) identified quantitative APR in cvs Alcedo (2DL, 4BL) and Brigadier (3BL, 5A), however the QTLs detected in Brigadier provided only low resistance level. Brigadier is included in the YR panel but appeared to be susceptible in both field trials in 2010 and 2011. Alcedo (pedigree (Record x Poros) x Carstens VIII) entered in the pedigree of Apostle (see pedigree diagrams in Appendix 8) which was highly resistant both at seedling and adult stage against current UK isolates. Consequently, QTL from Alcedo may participate to the quantitative field resistance found in the YR panel.

The UK cultivar Guardian also in the YR panel was found to carry three QTL for APR (1B, 2D, 4B), the QTL on 1B is believed to be durable resistance gene *Yr29*. *Yr29* confers a partial resistance, showing a slow rusting phenotype that is not associated with a strong necrotic response (Rosewarne et al., 2006; Singh et al., 2001). Despite Guardian is not well represented in the YR panel, the presence of *Yr29* and APR QTLs in Guardian, a UK cultivar from 1982 developed by Advanta UK (now part of Limagrain), suggests those sources of APR resistance have been introduced in UK breeding programs. Guardian pedigree (Israel-M-46/Maris-Ranger//Siete-Cerros-66/Maris-Ranger) includes also Heines Peko and Cappelle Desprez via Maris Ranger.

Carstens V is another cv thought to have APR resistance (Lewis, 2006) and which have been used extensively in European breeding program (Angus, 2001; Eriksen et al., 2004). Carstens V is known to have at least three races specific resistance efficient at seedling stage (Calonnec et al., 2002; Chen and Line, 1993a). *Yr32* was identified on 2AL (Eriksen et al., 2004), *Yr4 or Yr3*, *Yr25* and *YrSd* were also postulated based on multiple seedling tests (Lewis, 2006). Using bulk segregant analysis, three locus influential on Carstens V adult resistance were identified on 1BL, 2AL and 4DS. Carstens V presented a moderate to low level of resistance in the field trial in 2010 and 2011. Carstens V and its direct descendant Carstens VIII are present in the pedigree of many YR panel lines in particular in Apostle (via Alcedo), Fresco (via Monopol), Parade (via Granta), Boxer and Wasp (via Griffin). Consequently some of the quantitative resistance observed in the field could originate from Carstens V.

Claire is also known for its durable resistance (Powell et al., 2009), and have four QTLs for APR (2 in 2DL, 2BL, 7BL), one QTL on 2DL is likely to be *Yr16* and the other QTL on 2DL is likely to be similar to the 2DL QTL from Alcedo (Powell, 2010). Claire presented a high level of resistance in the field trial in 2010 and 2011 and has for parental line Flame and Wasp. Those two lines carrying also high to moderate APR resistance and both include Carstens V and Cappelle Desprez in their pedigrees.

4.3 Conclusion

Based on seedling tests with two Pst isolates, molecular markers and pedigree analysis, resistance genes *Yr7, Yr6, Yr9* and *Yr17* have been identified in the YR panel, however they have limited breeding value because virulence for all occurs at relatively high frequency in many European countries. Nevertheless, the information presented is useful for plant breeders in rationalizing germplasm enhancement programs. Additionally, resistant cultivars against Brock isolate 03/7 and Solstice isolate 08/21 have been identified and are potential source of uncharacterized resistance.

The extended virulence testing of the three Pst isolates used in National testing between 2009 and 2011 showed us that *Yr* genes *1, 2, 3a, 3b, 4a, 4b, 6 , 7, 9, 17, 20, 21, 22, 23, 25, 26, 27, 32, A, Sd, HVII* were not efficient against at least one of the current UK isolates tested. Additionally, the test confirmed that *Yr* genes *5, 8, 10, 15, 24, SP* are still providing a high level of resistance against the UK isolates 03/7, 08/501 and 08/21. Furthermore, seedling resistances present in cvs, Paha (*YrPa1, YrPa2, YrPa3)*, Druchamps (*Yr3a, Yr4a, YrD, YrDru, YrDru2*), IDO377S (*Yr43*), Zak (*Yr44*), Daws (*YrDa1, YrDa2),* Yamhill (*Yr2, Yr4a, YrYam),* Produra (*YrPr1, YrPr2),* Express *(YrExp1, YrExp2, APR)* and C591 *(YrC591),* revealed to be inefficient against at least one of the UK isolates tested. Only cvs Tres (*YrTr1, YrTr2, Yr32),* Tyes (*YrTye),* Batavia (*Yr33, YrA, YrBat1, YrBat2),* PI181434 (=205) and Cadenza are carrying *Yr* gene or a combination of *Yr* genes efficient against all isolates at seedling stage.

The evaluation of disease resistance of the YR panel at adult stage against Solstice isolate in 2010 and a mix of current UK isolate in 2011 demonstrate the presence quantitative field resistance in most of the lines. Additionally we identified cultivars possessing specific APR which are potentially important sources of yellow rust resistance for use in wheat breeding programs. Based on pedigree information, the YR panel may include APR originated from Carstens V and Cappelle Desprez, but not exclusively. However additional genetic studies will be necessary to characterize the APRs present in the YR panel and evaluate their diversity.

Nevertheless, in the light of the emergence in 2011 of a new Pst race namely the "Warrior" race virulent on cv. Warrior, with additional virulence on *YrSP* and the combination of virulence *Yr 6, 7, 9, 17* a new evaluation of the YR panel would be needed to confirm that the resistant cultivars identified at seedling stage and adult stage will maintain their resistance.

CHAPTER V. YR PANEL GENOTYPING, POPULATION STRUCTURE AND LINKAGE DISEQUILIBRIUM ANALYSIS

1 INTRODUCTION

With historic APR data and *de novo* evaluations of the full wheat YR panel against modern Pst isolates described in the preceding chapters, we dispose of extensive WYR resistance data. In order to conduct GWA scans, genotype data covering the entire wheat genome at an appropriate density is needed. Although a SNP array with several hundred informative wheat SNPs has recently been reported (Chao et al., 2010), the availability of DArT arrays for some years has meant that this platform was the most cost-effective way of sampling genome-wide across the bread wheat genome during the execution of the work described here (Akbari et al., 2006; White et al., 2008). For this reason, DArT was chosen for genotyping the wheat YR panel. Success in GWA mapping depends critically on matching the LD decay rates observed with an appropriate marker density, and in controlling adequately for any population structure evident in the selected panel.

Therefore, objective of this chapter is to explore the genetic diversity, the population structure and LD characteristics of the YR wheat panel. In addition, we present an exemplar GWA scan for straw length to choose the appropriate association analysis model for our wheat panel and genotype dataset.

2 MATERIAL AND METHOD

2.1 Plant material

The 327 varieties from the wheat YR panel were selected to be genotyped. The composition of the YR panel is as described previously in Chapter 2. In addition, 18 European WYR differentials obtained from the UKCPVS were genotyped to provide a control for major race specific *Yr* genes.

2.2 DNA extraction and quality check

DNA was extracted in 96-well microtube collection racks from Qiagen or 2ml eppendorf tubes using a modified microprep protocol from Fulton et al. (1995). 50-100mg ($1cm^2$) of leaf tissue has been collected from 2 week old wheat seedlings and placed in a -80C freezer prior to extraction. A 2mm stainless steel bead was added to each tube and the samples were disrupted mechanically for 30 s at 30 hertz using a Spex Certiprep GenoGrinder 2000. The tubes were briefly spun down at 1500g in a Sigma Qiagen 4-15C centrifuge to prevent contamination prior adding 500ml of fresh extraction buffer to lyse the cells.

The extraction buffer was prepared on the day of the extraction by adding 50 ml of lysis buffer stock solution (0.2 M Tris HCl pH 8.0, 0.05 M EDTA pH 8.0, 2M NaCl, 2% CTAB), 50ml of extraction buffer stock solution (0.35 M Sorbitol, 0.005M EDTA pH 8.0, 0.1M TrisHCl pH 8.0), 20ml Sarkosyl stock solution (5% Sarkosyl w/v) and 0.4g sodiumdisulfite. To reduce the viscosity, the buffer was placed in the oven 20min at 65°C and 240µl of ribonuclease A from Qiagen (100mg/ml) was added to the mix. The tubes were sealed properly and shake an additional 30 s in the GenoGrinder at 30 hertz, spinned again and incubated at 65°C for 60 min. The tubes were inverted half way through.

Once the incubation time was off, the tube was placed in the fridge at 4°C, 10 min to cool down. 300 µl of chloroform: isoamyl alcohol (24: 1) mixture was added to each tube and mixed by shaking the tubes for 5 min. The samples were then centrifuged at 1500g for 5min and 400µl of each supernatant transferred in new tubes. 400µl isopropanol at -20°C was immediately added to precipitate the DNA. The tubes were inverted 10 times; at this stage the nucleic acids became visible. The DNAs were pelleted by centrifugation at 6000g. The supernatant was discarded and the pellets washed twice with 1ml of ethanol at 70% and spun down at 6000g for 7 min. After discarding the ethanol supernatant, the pellet were dried for 1h under the fume hood and finally eluted in 50 µl of TE buffer (0.01M TrisHCl pH 8.0, 0.001M EDTA pH 8.0),

The quality of the DNA samples was controlled on a gel after extraction following Diversity Arrays Technology (DArT) quality check guidance. DArT requires 20 µl of DNA solution at 50-100ul/ng with minimum shearing and high digestibility by the restriction enzyme *Mse*I. The quality control consisted of an electrophoresis on a 0.8% agarose gel of the genomic DNA extracted to verify the integrity of the DNA and the absence of shearing. The quantity of DNA was evaluated by comparing the intensity of the bands on the gel with NEB lambda DNA solutions at 20, 50 and 100 ng/µl. The extracted DNA was also digested with the restriction endonuclease *Mse*1 from NEB during 2h at 37˚C and the completeness of

digestion was checked on 0.8% agarose gel. These steps ensure the absence of contamination which could impact the complexity reduction step from the DArT procedure. Figure V-1 illustrates the DNA quality and quantity check.

The DNA concentration of a tenth of the samples was also quantified on a NanoPhotometer (Implen) to ensure an accurate quantification from the gels. The typical yield ranged from 50 to 100 ng/µl.

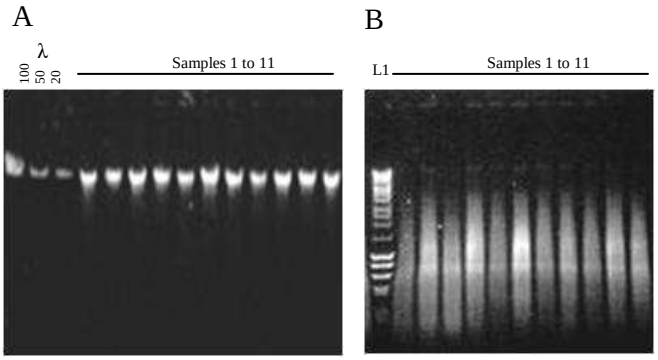

Figure V-1: Example of high quality DNAs for DART genotyping

DNA stained with ethidium bromide on 0.8% agarose gel visualised under UV light A. DNA genomic from 11 varieties, λ lambda DNA at 100, 50 and 20 ng/ul, B. DNA after digestion by *Mse*I, LI: HyperLadder™ from Bioline.

The extraction was repeated until the required quality for DArT for each sample was obtained.

2.3 Genotyping

2.3.1 Genome-wide coverage with DArT markers

The set of 327 varieties included in the YR panel, together with 18 yellow rust differential varieties were genotyped using the Wheat *Pst*I (*Taq*I) high density DArT array v.3 from Diversity Arrays Technology Pty Ltd in separate services. Wheat DArT genotyping is provided by Triticarte Pty Ltd. in Australia (http://www.triticarte.com.au). DArT was chosen for the present work as the most efficient and mature (at the time this study was initiated) technology that could offer unbiased genome-wide coverage of the hexaploid wheat genome (Akbari et al., 2006). In addition the latest array v.3 has been enriched to increase coverage of the D genome of bread wheat. The DArT markers are binary and dominant markers. Polymorphisms, based on SNPs and INDELs at restriction enzyme cutting sites and large INDELs within restriction fragments, are revealed through detection of binding (or not) of the target to a solid-state array of immobilised polymorphic fragments (Jaccoud et al., 2001).

2.3.2 Genotyping for *Rht1*, *Rht2*, *Ppd-1* loci, 1BL.1RS and 2NS.2AS translocations

The panel was genotyped using four primer pairs specific for causative polymorphisms at the *Rht1* (*Rht-B1a* and *Rht-B1b*) and *Rht2* (*Rht-D1a* and *Rht-D1b*) loci, so that allelic status at the two most important semi-dwarfing loci used in European wheat could be ascertained. The assay used was developed by Wilhelm (2011). Primer sequences and PCR conditions are shown in Appendix 9.

The potent photoperiod insensitivity allele Ppd-D1a is caused by a 2kb deletion in the PRR gene homoeologue at the *Ppd-D1* locus and primers specific for this causative polymorphism were used to detect the presence/absence of the deletion in the panel as described by Beales et al. (2007). Primer sequences and PCR conditions are shown in Appendix 10.

The presence of the 1BL.1RS translocation was assayed using the co-dominant marker developed for rapid detection of the translocation in winter wheat, as described by de Froidmont (1998). The same assay was used to detect the *Yr9* gene carried on the translocation (see Chapter IV for details).

The presence of introgressed segment including resistance genes *Yr17, Lr37* and *Sr38* from *Aegilops ventricosa* 2N chromosome in the hexaploid wheat 2A chromosome (2NS.2AS translocation) was assayed using the dominant marker SC-Y15 developed by Robert et al. (1999). The same assay was used to detect *Yr17* gene carried on the translocation (see Chapter IV for details).

2.4 DArT Consensus map

To assess the extent of linkage disequilibrium (LD) and to facilitate the analysis of association mapping results, the construction of a consensus map including DArT markers was necessary.

A consensus map was build from four genetic maps including DArT markers as well as SSR, SNP and STS markers:

- Genetic maps from crosses containing at least one UK elite parent line:
    - Avalon x Cadenza (markers data available from WGIN website http://www.wgin.org.uk and Allen et al. (2011), map created in MapDisto (Lorieux, 2012),

  - Solstice x Robigus (markers data from Wheat Triticarte DArT array v3 provided by Dr. Anna Gordon NIAB, map created in Mapdisto),
  - Claire x Lemhi (map provided by Dr. Lesley Boyd NIAB, map created using MapMaker/Exp (Lander et al., 1987).
- Genetic map from the RIL population UC1110 x PI610750 (Lowe et al., 2011b); this map was selected as it includes an extensive number of DArT markers from Wheat Triticarte array version 3.

Cosegregating DArT markers from each map were included to maximise the number of DArT markers in the consensus map.

The information from the multiple linkage maps was integrated in a consensus map using MergeMap Online (Wu et al., 2008), using the following order of confidence: (1) Avalon x Cadenza, (2) Robigus x Solstice, (3) Claire x Lemhi and (4) UC1110 x PI610750. Maps (1), (2), (3) were weighted 1 each while map (4) was weighed 0.5. The DAGGER algorithms (http://cran.r-project.org/web/packages/DAGGER/) implemented as a package for the R environment (http://www.Rproject.org/) was also tested as Endelman (2011) highlighted its efficiency comparing to MergeMap. However the presence of many cosegregating markers created ordering conflicts that could not be resolved using DAGGER, therefore MergeMap was preferred over DAGGER for the present study.

The new consensus map includes DArT, SSR, SNP and STS markers. The map published by Somers et al. (2004) was used as the reference map for the chromosomal orientation. Furthermore, the new consensus map was compared to the DArT consensus map developed by Crossa et al. (2007) to highlight possible discrepancies.

To complement the consensus map created in MergeMap, DArT markers without map position were placed in the consensus map when they were closely correlated to mapped markers based on estimated pairwise correlation coefficient r^2.

2.5 Linkage disequilibrium analysis

As a measure of linkage disequilibrium (LD), we calculated the correlation coefficient (r^2) between each pair of loci using the software program TASSEL 3.0 (Bradbury et al.). Only markers assigned to a single chromosomal location in the consensus map and with a minimum allele frequency >0.05 were included in the analysis. The significance of the LD pairwise LD (P-value) was computed using the default setting in TASSEL. To evaluate the extent of LD decay, r^2 was plotted against genetic distances for intra-

chromosomal pairs corresponding to each genome. A second degree Loess (locally weighted scatter plot smoothing) curve was then fitted in R and the intercept between the critical value of r^2 and the fitted curve was used as measure of LD decay. The critical value of r^2 was estimated following the procedure suggested by Breseghello and Sorrells (2006b) as the 95% percentile of the square rooted r^2 distribution of unlinked marker pairs e.g. inter-chromosomal pairs. Beyond the critical value, LD is likely caused by real physical linkage. Additionally, the extend of LD decay was evaluated based on the moderate level of LD r^2=0.2 to ease the comparison with published studies.

2.6 Population structure

The program STRUCTURE, which implements a Bayesian algorithm to estimate the number of historical subpopulations present within a variety panel, was used. The basic algorithm (Island-based model) was described by Pritchard et al. (2000a), and an extension to the model incorporating admixture within subpopulation was later introduced (Falush et al., 2003). The principal output is the Q matrix assigning fractional membership of each of K historical subpopulations to each member of the panel. The Q matrix provides a representation of substructure within a variety panel which can then be used as covariate in association mapping.

The software STRUCTURE version 2.3 was run varying K (the postulated number of sub-populations) from 1 to 15, with a burn in of 500,000 cycles and a Markov Chain Monte Carlo (MCMC) phase of 2,000,000 cycles. Two runs for each K value were performed. To prevent bias in the estimation of population structure, we used a subset of DArT markers that have a pairwise r^2 <0.6 and a minimum allele frequency (MAF) >0.1.

The optimum number of sub-populations K was determined using both the ad hoc procedure introduced by Pritchard et al. (2000a) and the rate of change of likelihood distribution method developed by Evanno et al. (2005)

In addition, the examination of population structure was undertaken using Principle Component Analysis (PCA) in R (http://cran.r-project.org/). The main principal components were plotted in R to assess and visualise the association of distinct germplasm pools with specific traits of interest. The analysis was done on the same subset of DArT markers used with STRUCTURE (r2<0.6 and MAF>1) i.e. 442 non-redundant markers.

2.7 Population genetic diversity

The software PowerMarker version 3.25 (Liu and Muse, 2005) was used to determine allele frequencies, polymorphism information content (PIC) and gene diversity of each marker. The calculation of PIC and gene diversity for each marker was according to the formulae:

(V-1)

$$PIC = 1 - \sum_{i=1}^{m} P_{i=1}^{2}$$

(V-2)

$$gene\ diversity = 1 - \sum_{i=1}^{m} P_i^2 - \sum_{i<j} 2P_i^2 P_j^2$$

P_i and P_j are the proportions of the population carrying the ith and jth alleles.

2.8 Preliminary association analysis with height

To test the usefulness of the YR panel for association mapping and select the most appropriate model to apply in order to limit spurious associations due to population structure and kinship, we used plant height data from the 2010 and 2011 YR panel field trial.

2.8.1 Phenotype data: height

The average straw length of lines in the YR panel was scored from each plot in adult plant resistance tests carried in 2010 and 2011 (see Chapter IV section 2.5). GenStat 13th Edition (Payne et al., 2009) was used to perform the statistical analysis on the phenotypic data. Analysis of variance using REML was performed and an adjusted mean based on BLUP was obtained for each line after fitting the following model:

(V-3)

$$y_{ikj} = \mu + V_i + T_j + TV_{ji} + e_{ikj}$$

y_{ikj} is the straw length for the variety i, in bloc k, in trial j; μ is the overall mean; V_i is the effect of the ith variety; T_j is the effect of the jth trial; TV_{ji} is the interaction between the test and the variety; e_{ikj} is the residual. A fixed effect model was used to look at significant effect based on Wald statistic. Varieties, block, trial and their interactions were treated as random factors to calculate adjusted means based on BLUP.

To compare the genetic and environmental source of variation between trials, heritability was calculated as followed:

(V-4)

$$h^2 = \frac{\sigma_G^2}{\sigma_P^2} = \sigma_G^2/(\sigma_G^2 + \sigma_E^2)$$

$$\text{where } \sigma_E^2 = \left(\frac{\sigma_e^2}{r_s}\right) + \left(\frac{\sigma_{tv}^2}{s}\right)$$

$\sigma_G^2, \sigma_P^2, \sigma_E^2, \sigma_e^2$, represent respectively the genetic (variety), phenotypic, environmental and residual variances. σ_{tv}^2 is the variance of block.variety component. r_s is the number of plots per variety, s the number of trials? The estimates of variance for σ_G^2, σ_{tv}^2 and σ_e^2 were obtained from GenStat after fitting a random model
(IV-5).

2.8.2 Association analysis

Associations with height were tested using TASSEL Standalone version 3.0 (Bradbury et al., 2007). A naive model as well as models including correction for population structure were tested (Table V-1). The Q matrix for K=2 (two subpopulations) from STRUCTURE was included in a general linear model (GLM). Alternatively, a matrix including the seven first principal components which explain approximately 25% of the variation was used to control population structure. Furthermore, a mixed linear model (MLM) was applied including a kinship matrix (K matrix) supplemented or not with a structure matrix (Q matrix) in the model. The K matrix representing the proportion of shared alleles between individuals was obtained in TASSEL from spaced markers (MAF >0.1 and r^2<0.6). The underlying equation for the five models tested is:

(V-5)

$$Y = \mu + Xa + Qb + Kv + \varepsilon$$

Y is the phenotypic observation term, μ is the intercept, a is the fixed effect related to the marker, b is the vector of the fixed effect related to population structure, v is the vector of random effects due to relatedness among the lines and ε is the residual effect. X is the marker allele matrix, Q is the population structure matrix either from STRUCTURE software or PCA. K is the kinship coefficient matrix.

Table V-1: Summary of statistical models used to test the data for marker-

trait association

Qs refers to the structure matrix from STRUCTURE and Qpca refers to the structure matrix based on PCA.

Model	Statistical model	Type
Model 0 "Naive"	$Y=\mu+Xa+\varepsilon$	GLM
Model 1	$Y=\mu+Xa+Qs\ b+\varepsilon$	GLM
Model 2	$Y=\mu+Xa+Qpca\ b+\varepsilon$	GLM
Model 3	$Y=\mu+Xa+Kv+\varepsilon$	MLM
Model 4	$Y=\mu+Xa+Qs\ b+Kv+\varepsilon$	MLM
Model 5	$Y=\mu+Xa+Qpca\ b+Kv+\varepsilon$	MLM

Only markers with MAF of 0.05 were investigated to limit spurious association of phenotypes with minor alleles. The efficiency of the different methods employed to correct for spurious association was compared visually using quantile-quantile (Q-Q) plots. The Q-Q plot shows the observed $-\log_{10}$ (P-values) of a genome wide association scan plotted against the expected $-\log_{10}$ (P-values) under the null hypothesis of no association.

Significant associations were detected based on a P-value threshold of $\alpha=0.01$. The use of an adjusted corrective threshold for multiple testing was also investigated. The Bonferroni threshold for multiple testing is defined as α – the probability level - divided by the number of tests, for instance in our study a 5% Bonferroni threshold is calculated as 0.05/number of markers tested. However, since DArT markers are often correlated with each other this is likely to be excessively conservative. Therefore, an alternative threshold for multiple tests based on false discovery rate (FDR) or Q-value was tested using Qvalue software (Storey, 2002). FDR is known to exert a less stringent control on false discovery compared with the Bonferroni correction (Benjamini and Yekutieli, 2001).

3 RESULTS

3.1 Genotyping

3.1.1 DArT markers

A total of 1806 polymorphic, genome wide DArT markers were scored on the panel. Marker prefixes "rPt-" (15), tPt (91) and wPt-" (1700) designated DArT features developed respectively from rye, triticale and wheat. Based on Triticarte assignment, 249 markers were located on genome A, 501 on genome B and 88 on genome D, 78 markers were assigned to multiple locations, 890 markers were not assigned to a chromosome as they were newly developed markers (coded wPt- followed by six digits) present on the Triticarte array version 3. Crossa et al. (2007) already developed a consensus map based on eight genetic maps

available from the Triticarte web site (http://www.triticarte.com.au/) however, the component maps were generated using previous arrays and the consensus map only includes 273 DArT markers scored on the YR panel using the Version 3array. The limited availability of map locations for DArT markers and in particular the absence of any public mapping data for a majority of markers from the Version 3 array at the outset of this project, underlined the necessity to develop an improved consensus maps including markers from the array v.3.

3.1.2 *Ppd-D1* 2kb deletion

We ascertained the allelic status of each entry in our panel for the most common photoperiod insensitivity allele *Ppd-D1a* manipulated by breeders to verify if its distribution reflected a possible geographic division between the UK lines (254 lines) and the European lines (67 lines) composing the YR panel.

Each of the lines tested amplified a single product demonstrating the quality of DNA. The deletion was present in only nine varieties of the YR panel: Axial, Isengrain, Isidor, Lorraine, Soissons, Caphorn, Frelon, Hurley, Ritmo. All these lines except Ritmo are French or include French lines as direct parent. The 318 remaining YR panel lines amplified the fragment characteristic of the absence of the deletion.

3.1.3 *Rht1* and *Rht2*

We genotyped the variants at two major *Rht* genes: *Rht1* on 4B (alleles *Rht-B1a* and *Rht-B1b*) and its homologue on 4D *Rht2* (alleles *Rht-D1a* and *Rht-D1b*)(Table V-2) using assays developed by Wilhelm (2011). *Rht-B1a* (wild allele e.g. tall) appeared to be predominant in the YR panel, 291 lines amplified the fragment characteristic for this allele. While *Rht-D1b* (semi-dwarf allele) is the most common variant for *Rht2* was found in 251 of 327 varieties.

We also detected 9 lines heterozygous for *Rht1* and 2 for *Rht2*, which was unexpected as all lines included in the YR panel are known to be inbred lines thus in principle, homozygous at all loci. A problem with DNA purity or a cross contamination during the multiplication of the lines rather than heterozygosis ‘per se’ of the varieties may explain this result. For this reason, the heterozygote genotypes are indicated as missing for the purpose of association analysis.

Table V-2: Presence of products from *Rht* assays on the YR panel lines

		Rht2 assay			
		Rht-D1a **(tall allele)**	***Rht-D1b*** **(semi-dwarf allele)**	***Rht-D1a* +*Rht-D1b*** **(heterozygote)**	**No amplification**
Rht1 assay	***Rht-B1a*** **(tall allele)**	56	**240**	0	5
	Rht-B1b **(semi-dwarf allele)**	**11**	0	0	0
	Rht-B1a*+*Rht-B1b **(heterozygote)**	0	7	2	0
	No amplification	2	4	0	0

3.1.4 1BL.1RS translocation

The presence of the 1BL.1RS rye translocation (including *Yr9*) in the panel was evaluated via a multiplex PCR developed by de Froidmont (1998). 108 lines showed the band characteristic of the 1RS translocation from rye (band 412bp) (see Chapter IV section 3.7.2), however four of them appeared heterozygous as they also amplified the fragment specific to 1BS.

Comparing the PCR assay to DArT markers, we observed 97 DArT markers with a correlation coefficient r^2 greater than 0.7 (11 rPt-, 20 tPt- and 66 wPt-) with the presence of the 412bp fragment. Therefore 5% the entire set of DArT markers are tightly linked to the presence/absence of the 1BL.1RS translocation. The maximum correlation value of 0.99 was observed for 51 markers. The correlation did not reached 1 as Alchemy was diagnosed with the translocation based on the PCR assay, but the DArT data showed clearly the absence of the translocation in Alchemy. Furthermore, the lines thought to be heterozygous did show DArT markers characteristic of the presence of the 1RS chromosome. Based on DArT markers, a total of 104 YR panel lines include the rye translocation 1BL.1RS.

3.1.5 Alien introgression 2NS-2AS from *Aegilops ventricosa*

The presence of the alien segment 2NS-2AS from *A. ventricosa* carrying resistance genes *Yr17*, *Lr37* and *Sr38* in the YR panel lines was evaluated using the dominant SCAR marker developed by Robert et al. (1999), 132 lines showed a band characteristic (580 bp) of the alien introgression (see Chapter IV section 3.7.3).

3.2 Consensus map

A consensus map was built from four genetic maps using the MergeMap program. The map obtained includes 2449 markers (DArT, SSR, SNP, STS and gene based markers) covering 4576 cM spread over 24 linkage groups (Table V-3, the complete map is presented in Appendix 11). DArT markers represent 72% of the mapped loci. There were several gaps

in the map coverage notably on chromosome 1D, 2A, 2D, 3D, 4A, 4D, 6D and 7D, which were also observed in at least one of the individual genetic maps.

Table V-3: Consensus map, markers and linkage group length in cM

Chromosome and linkage group (LG)	Genome A		Genome B		Genome D	
	Markers	cM	Markers	cM	Markers	cM
1	148	174.0	115	246.1	100	155.5
2	127	281.6	186	218.9	69	162.7
3	96	198.5	327	313.0	106	157.1
4 LG1	128	184.9	50	114.7	24	126.0
4 LG2	-	-	6	32.9	-	-
5	86	248.7	162	225.03	37	258.2
6	127	205.4	159	161.8	22	146.8
7 LG1	164	249.3	142	216.0	49	280.5
7 LG2	-	-	7	7.8	12	11.1
Total	**876**	**1542.4**	**1154**	**1536.2**	**419**	**1297.9**

The spread of the new consensus map (4576 cM) is relatively high comparing to widely used consensus map from Somers et al. (2004) (2569 cM) and Crossa et al. (2007) (2149 cM). This is most likely an artefact of the MergeMap algorithm used, Endelman (2011) using simulation noticed a 46% inflation of linkage group length generated by MergeMap. Therefore, the genetic distance from the new consensus map must be interpreted with caution. One solution to overcome this issue could be to rescale the consensus map to the average length i.e. 2468 cM of the four maps used to build it.

A moderate to high correspondence between the present consensus map and the map from Crossa et al. (2007) was observed for chromosomes 1A, 1D, 2A, 2B, 3A, 3B, 5A, 5D, 6A, 6D, 7A and 7D. The consensus maps for chromosomes 1B, 2D, 4A, 5B and 7B presented limited correlation. The presence of translocations in PI610750 (1BL.1RS) (Lowe et al., 2011a) and chromosomal rearrangements in Claire (5BL.7BL) (Lesley Boyd personal communication) and Avalon (5BL.7BL) (Simon Griffith personal communication) created unusual recombination patterns that could explain the differences observed for 1B, 5B, 7B. Additional, unidentified translocations may be at the origin of other inconsistencies between consensus maps. Furthermore, low frequencies of recombination were observed in chromosomes 4A and 7A in Robigus x Solstice map most likely due to the presence of *T. diccocoides* segments in the Robigus genome, and therefore the genetic distance between markers was under-estimated in the chromosomes specified. Consequently the consensus map will have to be used with caution.

Because of the low number of common markers between the two consensus maps, we were not able to compare the order of DArT markers on chromosomes 3D, 4B, 4D, 6B and 6D.

The consensus map included 722 DArT markers scored on the YR panel (685 were assigned to a unique location and 37 to several locations within the genome).

3.3 Population structure

3.3.1 Subpopulation based on STRUCTURE

The number of historical subpopulations composing the YR panel was investigated using the Bayesian based clustering approach implemented in STRUCTURE (Figure V-2). The LnP(D) value increased continuously without significant change in slope as K (the postulated number of underlying subgroups) was varied from 1 to 15. Therefore the ad hoc statistic ΔK (rate of change of likelihood) was used as suggested by Evanno et al. (2005). According to ΔK approach, the optimal value of K is 2 when ΔK is maximal.

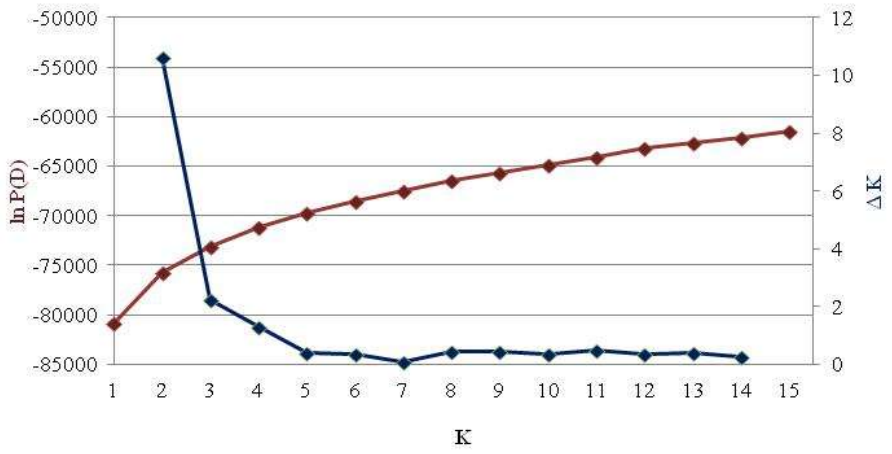

Figure V-2: Estimating the most probable number of sub-populations (*K*) based on Bayesian clustering for *K* = 1 to 15

In red, left-hand axis: mean log-likelihood of the data (Ln *P*(*D*)) per *K* generated from STRUCTURE program (Pritchard et al., 2000a). In blue, right-hand axis: mean absolute difference of the second order rate of change with respect to *K*, i.e. ΔK of LnP(D) following Evanno et al. (2005). The *ad hoc* statistic Δ*K* suggested *K* = 2 as the most likely number of clusters.

3.3.2 Principle Component Analysis

A PCA was run using 442 spaced DArT markers (same subset used with STRUCTURE), to further investigate the population structure. The first 10 eigenvectors from PCA explained 30.4% of the genetic variation; the first three components accounted for 7.3%, 4.5% and 3.4% of the variation respectively. The plots PC1 versus PC2, PC1 versus

PC2 and PC2 versus PC3 do not differentiate clear subgroups within the YR panel lines (Figure V-3). Nevertheless, a few varieties detached from the main cloud in PC1 versus PC2 (bottom left on the graph) and they include spring wheats AC Barrie, Axona, Tonic and their descendants (Cadenza, Spark, Scorpion 25, Warlock 24, Xi19), some of which are known to be alternative wheats.

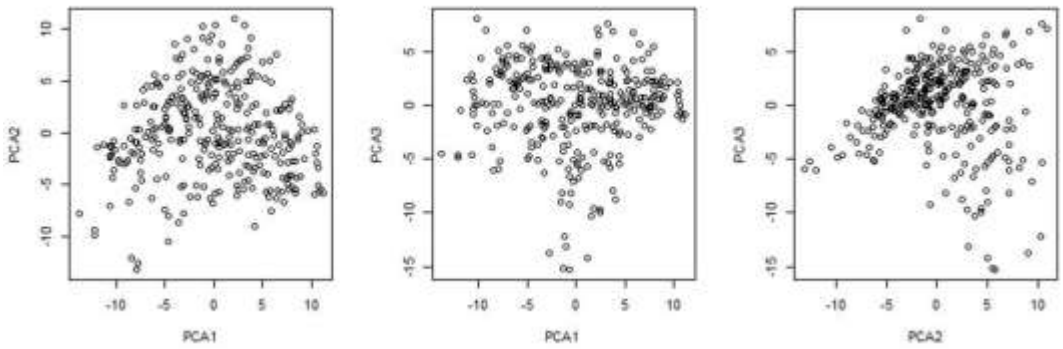

Figure V-3: Plots of the first three principal components with spaced markers, PC1 versus PC2, PC1 versus PC2 and PC2 versus PC3.

To illustrate why 442 spaced markers were used to investigate population structure, PCA done on the full dataset including all 1806 DArT markers is shown in Figure V-4. In this figure, we observe an artificial structuration into two groups according to the presence or absence of the 1BL.1RS translocation. However, this entire translocation behaves as a single (though physically large) recombinational unit, so the use of 97 of the 1806 (~5%) of all DArT markers noted in Section 3.1.4 as tagging the translocation gives a 97-fold redundant weight to this single genetic locus.

This is an extreme case, but DArT markers are documented as being more generally prone to clustering (Akbari et al., 2006; Marone et al., 2012b), therefore when analysing the population structure based on any, but especially DArT, genotypic data, it is important to select spaced markers to avoid spurious structuration.

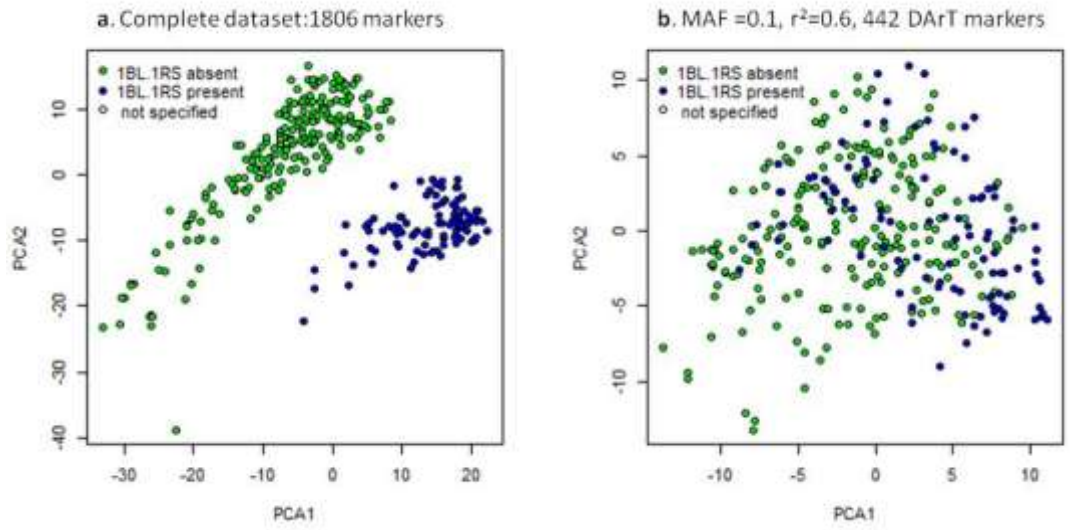

Figure V-4: Differentiation of genotypes based on the first two principal components when PCA applied to the full genotype versus skimmed genotype dataset.

(a) PCA derived from 1806 DArT markers, including redundant markers (b) PCA derived from 442 spaced DArT markers based on r^2=0.6 and MAF=0.1

3.3.3 Structure factors

The Bayesian algorithm for detecting population stratification implemented in the software STRUCTURE showed two clear subpopulations in the panel. The first two principle components from the PCA were used to visualise these two sub-populations in 2D-diversity space. The first principal component (PC1) captures the separation between subpopulations 1 and 2 very clearly (Figure V-5 a). The factors country, breeder and decade were assessed as putative sources of population structure. The country in which the variety was bred was the most powerful explanatory factor (Figure V-5 b). Lines bred in continental Europe formed a small group largely separate from the UK lines. The other factors we could have hypothesized to play a role in creating or reinforcing population stratification i.e. decade of release or breeding programme did not appear to cluster in PCA space.

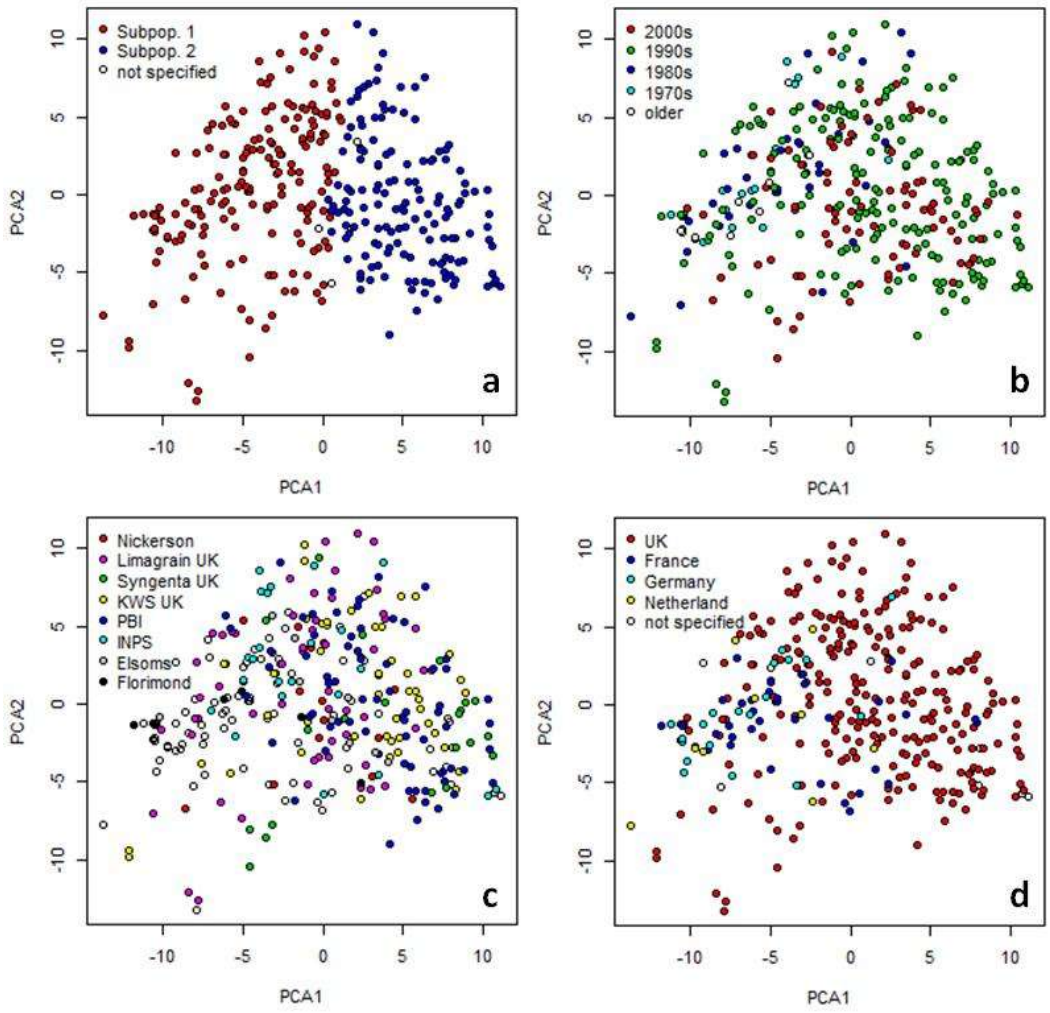

Figure V-5: Distribution of the YR panel lines according to the first two principal components, classified by subpopulations, decades, breeders and countries.

(a) Classification by 2 subpopulations defined in STRUCTURE program (K=2); (b) Classification by decade; (c) classification by breeders (breeders represented by less than 5 lines are not coloured; INPS: Institute of Plant Science; PBI: Plant Breeding Institute, previously INPS); (d) classification by country of breeding.

Looking at the molecular assays carried out on YR panel lines, we investigated the distribution of adaptative genes *Rht1, Rht2, Ppd-D1* and alien introgressions carrying yellow rust resistance genes *Yr9* (1BL.1RS translocation) and *Yr17 (*2A fragment from *A.ventricosa)* in subpopulations (Figure V-4 and Figure V-6). Using spaced markers, the translocation 1BL.1RS does not appear preferentially associated with one or other of the two historical populations revealed by Bayesian analysis or the structure revealed by the first principal components (Figure V-4). The dwarfing allele *RhtB1b* and the photoperiod insensitivity allele *Ppd-D1a* were both rare in the YR panel lines therefore they could not be

associated with a specific sub-population. While the dwarfing allele *RhtD1b* was highly frequent in the YR panel lines, the wild type allele *RhtD1a* seems to be most associated with subpopulation 1 and continental European varieties. The alien fragment carrying *Yr17* is present in greater frequency in subpopulation 2.

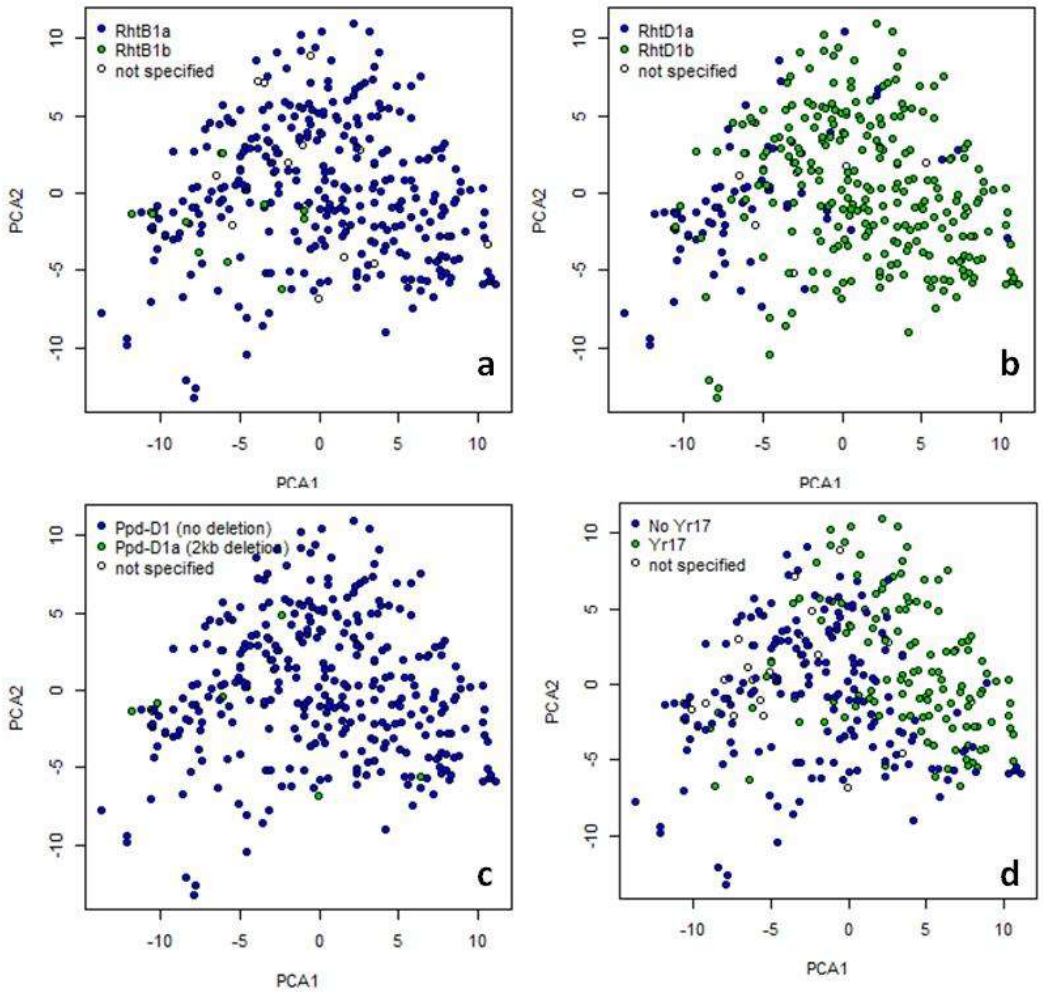

Figure V-6: Distribution of the YR panel lines according to the first two principal components, classified by allelic variation for *Rht1*, *Rht2*, *Ppd-D1* and *Yr17* assays

(a) Classification based on *Rht1* diagnostic assay; (b) Classification based on *Rht2* diagnostic assay; (c) classification based on *Ppd-D1* diagnostic assay; (d) classification based on *Yr17* diagnostic assay.

3.3.4 Linkage disequilibrium analysis

Pairwise LD between all combinations of markers from the consensus DArT map was calculated, 11,699 from intra-chromosomal pairs (referred as linked markers), 162,056 from inter-chromosomal pairs (referred as unlinked markers). 734 marker pairs (corresponding to 6.3% of the linked marker pairs) were in complete LD (r^2=1). 47.2%

(5,525) of the intra-chromosomal marker pairs showed significant LD ($p<0.01$) and the average of r^2 for all intra-chromosomal pairs was 0.159.

Significant inter-chromosomal LD was also detected in 19.9% of the marker pairs, highlighting the presence of population structure and the potential risk of spurious marker-trait association.

The plot of LD over genetic distance showed that LD extended to 16.7 cM based on the critical value of r^2 (Table V-4). LD decayed more rapidly in the D-genome as judged by the genetic distance to critical r^2 value of 9.4 cM compared to the A (15.2 cM) or B (21.0 cM) genomes, although it should be noted that the D-genome LD decay rate was estimated with lower accuracy since less markers were available and the D genome map contained many cosegregating markers.

Table V-4: Overview of LD in the wheat genomes A, B and D based on DArT markers in the YR panel

(a) Average pairwise r^2 for intra-chromosomal marker pairs, (b) average pairwise r^2 for inter-chromosomal marker pairs, (c) critical r^2 as described by Breseghello and Sorrells (2006b)

	DArT markers	Mean r^2 linked (a)	Mean r^2 unlinked (b)	Critical r^2 (c)	Extend of LD for critical r^2 in cM	Extend of LD for r^2 =0.2 in cM
A Genome	219	0.0923	0.0152	0.059	15.2	8.2
B Genome	240	0.0763	0.0125	0.046	21.0	9.4
D Genome	131	0.4047	0.0151	0.063	9.4	5.3
All genome	590	0.1587	0.0146	0.057	16.7	7.3

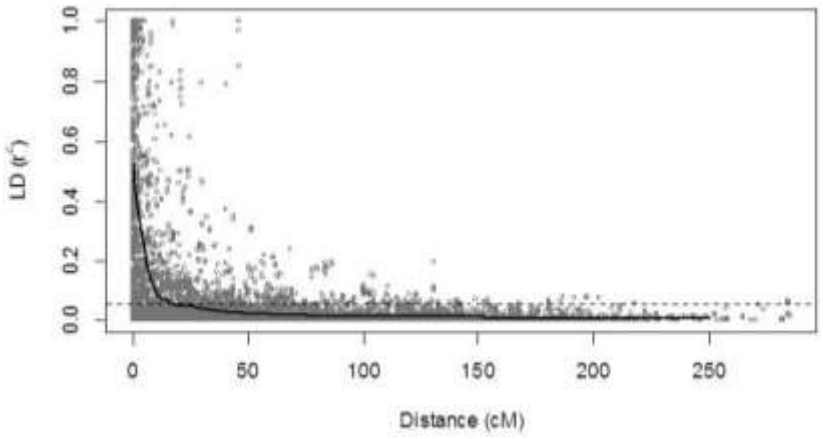

Figure V-7: LD parameter r^2 plotted against the genetic distance in cM for the whole genome

The horizontal dashed line indicated the 95% percentile of the distribution of unlinked r^2 referred as critical value of r^2. The black curve is the second degree LOESS curve fitted to the plot.

3.3.5 Genetic diversity and allele frequencies

For the 1806 DArT markers scored across the 327 lines from the YR panel, the frequency for allele "1" (positive detection of hybridisation to the DArT feature in question) ranged from 0.006 to 0.988. 257 DArT markers had a minimum allele frequency inferior to 0.05 and will not be used for further analysis.

For the 1549 DArT markers remaining, a fairly low mean frequency of missing data was observed (1.99%). The PIC values computed on the complete population ranged from 0.03 to 0.38 with a mean of 0.27. The gene diversity ranged from 0.03 to 0.5 with a mean of 0.33. The observed values of PIC and gene diversity were slightly lower in subpopulation 2 defined by STRUCTURE (for K1: PIC=0.27, gene diversity=0.34; for K2: PIC=0.21, gene diversity=0.26), reflecting a lower genetic diversity in population focused on UK lines (K2) compared to population with lines of mixed origin (K1).

3.4 Preliminary association

3.4.1 Phenotype

The plant height of 324 YR panel lines was evaluated in yellow rust field trials in 2010 and 2011 by measuring the average straw length of each plot which represents three replicates per line. The frequency distribution of straw length scores shows a near normal distribution both years (Figure V-8) reflecting the presence of one dwarfing gene in the majority of the lines; 255 YR lines have been diagnosed with one of the *Rht1* and *Rht2* dwarfing alleles (see Figure V-9). Toward the higher values a secondary normal distribution could be visualized, representing tall lines with wild type alleles for *Rht1* and *Rht2*; 56 YR lines have been diagnosed with both wild type alleles.

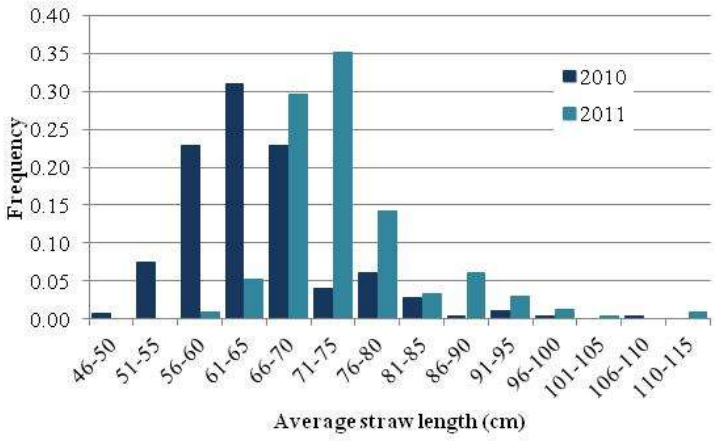

Figure V-8: Distribution of average straw length in field trial 2010 and 2011.

The average height was 64.2 cm in 2010 and 73.8 cm in 2011, which represents a significant difference of 9.6 cm ($p<0.001$ from t-test). As the primary purpose of the trial was to evaluate yellow rust resistance, the plants were highly stressed and the presence of the disease was likely to stunt their growth. As the disease intensity was greater in 2010, the plant growth might have been affected to a greater extent in 2010. The difference in plant height can also be partly explained by drought stress; both spring 2010 and 2011 were notably dry, however the 2011 trial received irrigation in May which may have prevented severe drought symptoms.

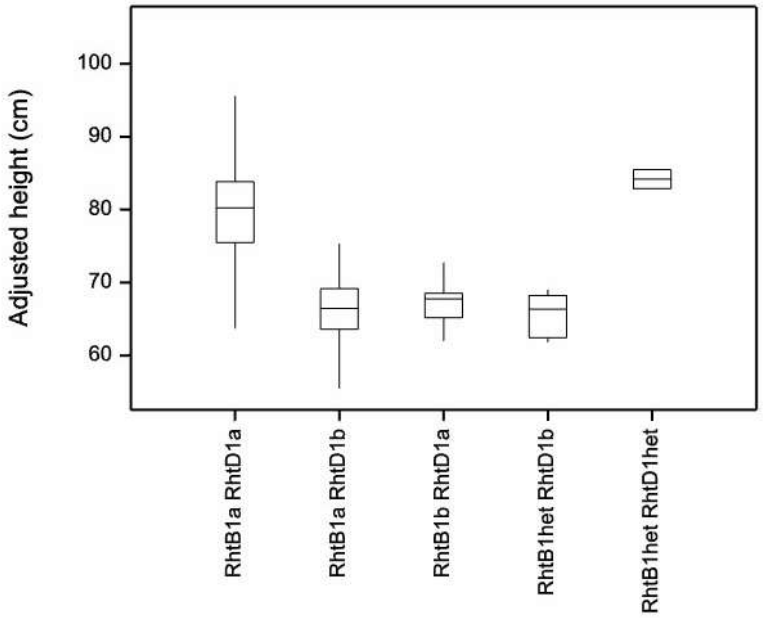

Figure V-9: Box plot of adjusted height in relation to *Rht1* and *Rht2* alleles

The figure illustrates the difference of height observed in the YR panel lines in relation to dwarfing alleles from *Rht1* and *Rht2* gene respectively *RhtB1b* and *RhtD1b*. *RhtB1a* and *RhtD1a* are the wild type alleles associated with a tall phenotype, *RhtB1*het and *RhtD1*het indicated the amplification of fragments characteristic of both alleles.

The analysis of variance using REML as implemented in Genstat confirms the presence of a significant trial effect (Table V-5). There were also significant interactions between the trials and the varieties which probably illustrates differences in varietal resistance to drought and yellow rust. Despite the variable environmental conditions between trials, the overall heritability (H^2) remains moderately high.

Table V-5: Analysis of variance of height data from field trials 2010 and 2011 using the REML adjustment procedure

(1) nominal degree of freedom; (2) significance probability from Wald tests based on fixed effect model; (3) estimate of variance component from random model; (4) standard error based on random model; (5) heritability based on random model.

Score	Source	*n.d.f*(1)	*F.pr.*(2)	Est. (3)	s.e(4)	H2(5)
Straw	Variety	*323*	*<0.001*	56.48	4.98	0.672
Length	Trial	*1*	*<0.001*	48.76	69.01	
Both	Trial.Var	*276*	*<0.001*	5.35	1.04	
Year	Residual			18.21	0.77	

Following the analysis of variance, an adjusted mean based on BLUP (Best Linear Unbiased Predictor) was obtained from Genstat and used for association mapping analysis.

3.4.2 Association analysis

Associations between 1549 DArT markers with MAF>0.05 and the height adjusted mean were tested using six statistical models. The association between the *Rht2* marker and height was also tested. *Rht1* and *Ppd-D1* markers were removed from the analysis as they were present in low allele frequency. Rare alleles are known to induce bias in the calculation of MTA P-value and are likely to cause false positive.

Depending on the corrective model, the significant marker-trait associations detected varied from 17 to 120 at a P-value threshold of α= 0.01 (Table V-6). Using the highly conservative 5% Bonferroni correction threshold (0.05/1549) for multiple comparisons 1 to 17 markers were significantly associated with height. Based on 10% FDR, 1 to 165 markers were associated with height. However the FDR threshold has to be interpreted with caution as the distribution of p-values obtained from AMs were highly distorted due to the presence of many redundant markers in the dataset, it was difficult to obtain a meaningful estimate of the null distribution parameter (Π_0) which is essential in the calculation of FDR.

The highest P-value in all models related to *Rht2* markers for alleles *RhtD1a* and *RhtD1b* (Table V-6).

P-values (transformed to $-\log_{10}(P)$) observed in the different models have been plotted against expected P value from a normal distribution to create a Q-Q plot (Figure V-10).The Q-Q plots allow visual comparison of the effectiveness of the models in correcting for population structure. Without population structure, we should see the P-value uniformly distributed between 0 and 1 and only true associations should deviate from the diagonal. The first Q-Q plot presenting the P-values from the naive model shows actual P values systematically above the diagonal, illustrating a significant overestimation of P-values (based on P-value threshold of α= 0.01, 769 of 1549 markers were associated with height). The second Q-Q plot includes the P-values from each of the five correction models tested.

All correction models gave an improvement compared to the naive model. The GLM model with the structure matrix Qpca corrected more effectively than the GLM model with Qs. The Q matrix for the two subpopulations defined in STRUCTURE software captured only a small part of the actual population structure, while the seven Principal Components modelled population structure far more effectively. MLM models 3 to 5 including Kinship present the best distribution of P-values as they include a kinship matrix representing the relatedness of the individuals within the population. Based on the pedigree collected from the YR panel lines, we already highlighted key varieties such as Moulin, Cappelle Desprez, Squadron, Norman, and Hobbit which appeared in the pedigree of many lines. Thus the major structuration of the YR panel is likely to result from variety relatedness and less from historical sub-populations. Comparing the three MLMs, model 5 including the kinship matrix (K) and the structure matrix Qpca seems to perform the best (P-value from the model followed closely the expected normal distribution of P-value), it decreases the number of probable false discoveries over model 4 and 3.

Table V-6: Number of markers with significant marker traits associations and P value associated with *Rht2* genes

Model	*Rht2* P-value	No of significant markers for different threshold			
		α= 0.05	**α= 0.01**	**Bonferroni 5%**	**FDR 10%**
Model 0: Naive	$4.43\ 10^{-40}$	945	769	300	1262
Model 1 GLM with Qs	$3.23\ 10^{-24}$	260	120	17	165
Model 2 GLM with Qpca	$2.05\ 10^{-20}$	186	82	7	74
Model 3 MLM with K	$1.41\ 10^{-16}$	131	34	1	4
Model 4 MLM with Qs and K	$3.57\ 10^{-19}$	156	56	2	35
Model 5 MLM with Qpca and K	$6.54\ 10^{-13}$	140	17	1	1

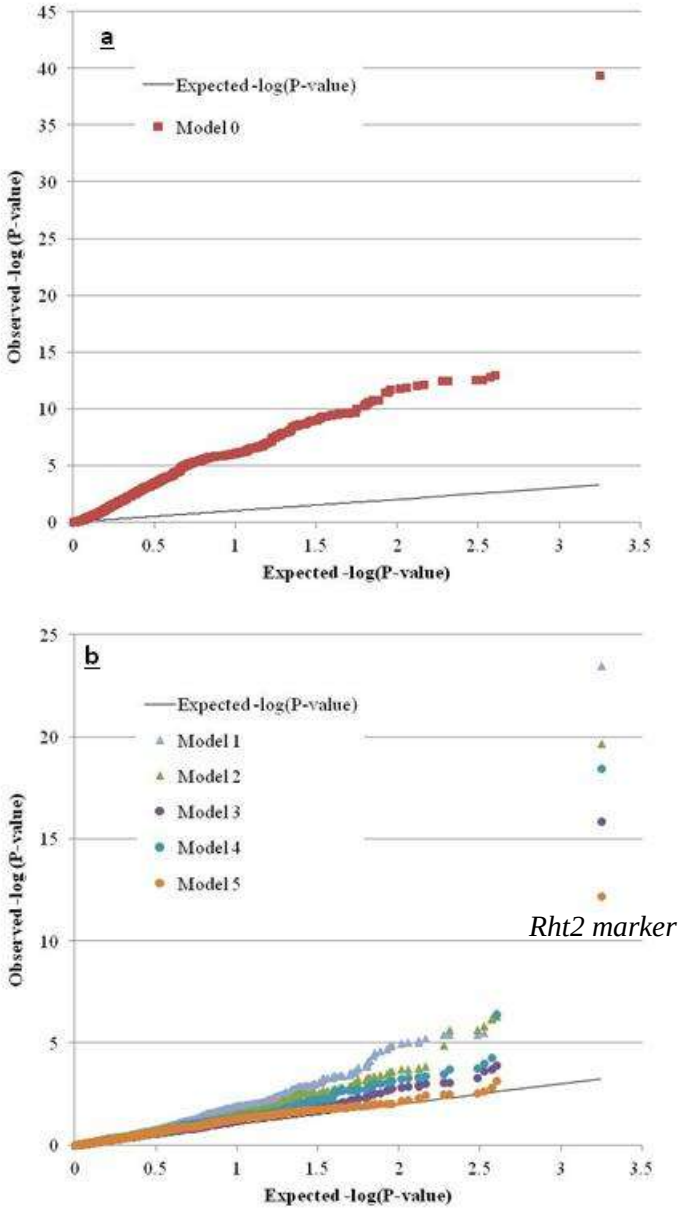

Figure V-10: Q-Qplot of –log (P-value) obtained from the association mapping models applied to heights.

a. Q-Q plot for naive model; b. Q-Q plot for corrective models

Based on MLM correction models, the *Rht2* marker was consistently significant marker using a 5% Bonferroni threshold, none of the other six DArT markers on chromosome 4D (i.e. linked to the *Rht2* gene) appeared above the threshold of significance. DArT marker wPt-9067 on 4B which mapped 15.3 cM from *Rht1* on our consensus map, was the only other markers passing the Bonferroni threshold. Below the Bonferroni threshold, but still with $\alpha = 0.001$ markers on chromosomes 1B, 2A, 3B, 5B, 5D, 7B presented significant association (see list of association hits in Appendix 12) and explained between 2.1 and 4.6% of the phenotypic variation, while the *Rht2* marker explained between

14.5 to 20.3 % of the phenotypic variation depending of the corrective model. The *Rht2* marker used is based on a causal SNP for a notable dwarfing gene and therefore it is expected to present a high level of significance.

4 DISCUSSION

4.1 Construction of a 2449-locus consensus map of hexaploid wheat integrating v3 array DArT markers

The consensus map developed in the present study has a high average density of 1.9 markers per cM and includes an extensive number of DArT markers (1744), 226 of which are novel DArT markers from the wheat DArT array v3. The different genomes A, B and D are represented respectively by 37%, 47% and 16% of the mapped DArT markers. Limited consensus maps have been published for hexaploid wheat since the construction of a reliable consensus map is made difficult by the size and the hexaploidy of the wheat genome as well as the presence of translocations and chromosomes rearrangements, many of which are still unrecognized. Two consensus maps are used as reference in many association mapping publications, (1) the map developed by Somers et al. (2004) which includes 1235 microsatellite loci distributed over 2269 cM (one marker per 2.2 cM); (2) the map developed by Crossa et al. (2007) which includes 1644 markers (813 DArT and 831 SSR, AFLP or RFLP) distributed on 2149 cM (one marker per 1.3 cM)**.** With respect to those two maps, the present consensus map represents a large improvement as it has a greater number of markers (2449 versus 1235 and 1644 markers) and includes novel SNPs developed by Allen et al. (2011). Compared to Crossa et al. (2007), the present map contains novel DArT markers from the latest DArT array v3 (used to genotype two of the four populations underpinning the present consensus map). Additionally, the D genome coverage with DArT markers is improved; 284 markers in the present consensus versus 66 markers in Crossa et al. (2007).

The new consensus map allows assigning 722 DArT markers scored on the YR panel, a great improvement comparing to the 273 markers mapped in the map from Crossa et al. (2007), providing the means to interpret more results from the association analysis. Furthermore, the opportunity is afforded to select additional markers to enrich regions of interest where associated DArT markers lie in close proximity to SSR and SNP markers.

Recently, Marone et al. (2012a) developed a high density consensus map for durum wheat (A and B genomes) including 1898 loci (1185 DArT, 388 SRR, 166 EST derived loci and 159 other markers) distributed on 3059 cM. Despite the absence of information on the D genome, this new consensus map is a valuable resource for hexaploid wheat as it appears to be fairly reliable given the high correspondence between the map position of genomic markers and physical positions given by Francki et al. (2009) and Gadaleta et al. (2009). The

assignment of 712 DArT to specific deletion bins by Francki et al. (2009) also provides valuable information allowing to ascertain the position of DArT markers from the previous DArT array v2, aligning genetic map and estimating the coverage of DNA markers across the wheat genome.

4.2 Genetic diversity of the YR panel

A set of 1549 DArT markers were used to characterize the genetic diversity of 327 elite varieties of hexaploid wheat. The average PIC and gene diversity values in the YR panel were 0.27 and 0.33 respectively, which was consistently lower than other wheat association populations genotyped with DArT markers. For example, Stodart et al. (2007) calculated the average PIC value of 355 DArT markers in 705 worldwide landraces to be 0.43; Le Couviour et al. (2011) estimated the genetic diversity (average PIC) in a set of 252 DArT markers in 195 Western European elite wheat to be 0.38; Benson et al. (2012) found an average PIC value of 0.31 and gene diversity of 0.39 from 900 DArT markers in a panel of 251 US winter wheat enriched for Fusarium head blight resistance; Zhang et al. (2011) calculated a mean PIC value of 1637 DArT markers to be 0.40 in 111 elite Chinese lines. As the YR panel is composed of elite germplasm it is expected its genetic diversity to be lower than landrace populations as in Stodart et al. (2007), however genetic diversity in the YR panel appears lower than many elite populations from different countries. A reduced genetic diversity of elite UK germplasm was also highlighted by White et al. (2008) compared to elite Australian and US lines. White et al. (2008) explained the less diverse gene pool in the UK by the long adaptation of wheat landraces to the UK environment followed by the selection in the late 19th century of well adapted lines creating a narrow genetic base, with the subsequent introgressions of traits from European, US and Japanese material involving backcrossing to maintain a UK tolerant ideotype which would have limited the gene pool expansion from those introductions.

4.3 Population structure of the YR panel

The population structure of the panel was examined through STRUCTURE and PCA using a set of 442 non-redundant DArT. The results of the two approaches were consistent as the two ancestral populations identified via STRUCTURE were also separated on the first PC vector. However the identification of K=2 is likely to be an underestimation of the real population structure, ΔK method of Evanno et al. (2005) is known to underestimated K where the genetic differentiation between subpopulations is weak (Waples and Gaggiotti, 2006). It is therefore appropriate to think of K=2 as having identified the uppermost level of structure in a hierarchically structured population.

Of three potential factors investigated leading to population structure (decade, country of selection and breeding company), the country in which the variety was originally selected had the greatest influence. We saw a division of wheat germplasm into groups from the UK and from continental countries (France, Germany and Netherland). This division is not surprising as climatic, agronomic and market differences between the UK and continental Europe dictate the varietal selection. Our findings are consistent with studies on European wheat collections. Roussel et al. (2005) reported geographical variation as a main factor of genetic structure in a historical European panel including 480 varieties released between 1840 and 2000. Recently, subdivisions between UK and western European countries were reported in elite germplasm collection by Le Couviour et al. (2011) and Reif et al. (2011).Neither the decade of breeding nor the breeding company appeared to explain the population structure of the YR panel. Roussel et al. (2005) and White et al. (2008) reported temporal changes in wheat diversity respectively in European and UK germplasm over the last century. However, these superficially divergent findings are not necessarily mutually exclusive. The Roussel et al. (2005) and White et al. (2008) studies covered a longer period of time (1840-2000 and 1920-2004 respectively) compared to the present study which focused mainly on the last three decades and Roussel et al. (2005) highlighted a lower diversification in recent times.

We can speculate that the consolidation of the breeding industry in the last decades into just a handful of European seed houses (KWS, Syngenta, Limagrain and RAGT being the predominant contributors to the YR panel) coupled with the crossing agreement which allow breeders to use any varieties on National Lists to develop new lines means any putative genetic footprint of an individual breeding programme is now too weak to observe.

4.4 Major gene differences across the YR panel

The genetic composition of the panel was additionally examined with a small set of markers diagnostic for major pleiotropic alleles controlling flowering time (*Ppd-D1*a) and height (*Rht-B1a/b* and *Rht-D1a/b*) and for two widely used translocations 1BL.1RS and 2NS.2AS.

Flowering time in winter wheat is dependent on three key groups of genes controlling the vernalization requirement, photoperiod sensitivity and "Earliness per se" (Worland, 1996). In our study, we focused on *Ppd-D1a*, a major gene-allele controlling photoperiod insensitivity (Beales et al., 2007). Photoperiod insensitive genes are thought to give an adaptative advantage in south and central Europe as photoperiod insensitive winter wheat varieties benefit from early flowering and their consequent earlier maturation helps them to avoid the effect of hot dry summers (Worland et al., 1998). Our allelic analysis

reveal that the majority of the lines in the YR panel except a few lines of French origin carry the photoperiod sensitive allele *Ppd-D1*, which is consistent with previous results. Le Couviour et al. (2011), studying a panel of 195 western European elite wheat varieties highlighted the high frequency of photoperiod sensitive allele *Ppd-D1* in German and UK germplasm contrasting with French varieties. Earlier Worland et al. (1998) looking at the phenotypes of 120 European wheat varieties for photoperiod sensitivity revealed that French varieties mostly photoperiod insensitive, while the majority of UK were photoperiod sensitive.

According to the catalogue of wheat gene symbols available online (http://www.shigen.nig.ac.jp/wheat/komugi/), 21 *Rht* genes involve in reducing plant height have been identified (*Rht1* to *Rht 21*), however *Rht1* (also named *Rht-B1*) and *Rht2* (also named *Rht-D1*) have been the most widely used in breeding program over the past 50 years. The dwarfing alleles *Rht-B1b* and *Rht-D1b* were both derived from the Japanese variety Norin 10 which was used in US breeding programs in the 1950s in order to improve lodging resistance in winter wheat. Soon after thanks to Norman Borlaug and the CIMMYT, both alleles were deployed worldwide and it is estimated that over 70% of commercial wheat varieties contain at least one of these two alleles (Evans, 1998). Both alleles *Rht-B1b* and *Rht-D1b* are semi-dominant and have similar effect on the plant height (reduction of 20 cm (Flintham et al., 1997)). They are generally introduced singularly in commercial wheat as the combination of *Rht-B1b* and *Rht-D1b* produced a strong effect on plant height and is generally associated with a decreased of the agronomical value of the variety (Allan, 1989). In our panel, only 17% of the lines carried the wild type for both genes (*Rht-B1a_Rht-D1a*) and no double dwarf (*Rht-B1b_Rht-D1b*) was detected. Additionally the dwarfing allele *Rht-D1b* appeared predominant over *Rht-B1b*, with the combination *Rht-B1a_Rht-D1b* representing 73% of the varieties. We also noticed that *Rht-D1a* allele was mainly associated with non-UK varieties. This result is consistent with the result presented by Le Couviour et al. (2011) as they found the *Rht-B1a_Rht-D1b* combination mainly in UK varieties and the *Rht-B1b_Rht-D1a* combination in French varieties. The specific use of *Rht-D1b* in the UK is thought to be the result of historical breeding practice with the introduction of *Rht-D1b* in the UK via PBI in early 1970, which predominated wheat breeding in the UK.

The short arm of chromosome 1R from rye (*Secale cereale*) is one of the most widely utilized sources of alien chromatin in wheat breeding (Baum and Appels, 1991), the wheat-rye translocation having been considered favourable for agronomic performance of wheat because it carries genes for disease resistance (Friebe et al., 1996), insect resistance (Marais et al., 1994) and high yield potential (Kim et al., 2004; Villareal et al., 1998). However its agronomic benefits have been inconsistent across wheat class and genetic background (reviewed by Lelley et al. (2004) for review) and 1RS is also known to reduce

bread-quality making. In a review, Rabinovich (1998) highlighted the presence of 1RS translocation in more than 300 genotypes worldwide, the major source namely 1BL.1RS originating from a cross with rye cv Pektus and German wheat in the 1940s. Roder et al. (2002) looked at 502 recent European varieties and identified the presence of 1BL.1RS translocation in 49% of northern European lines (mainly UK lines), the translocation was also present in western and central Europe but at a lower frequency. In the present study, 32% of the YR panel lines presented the 1BL.1RS translocation but they were not associated with a specific geographic origin. The 1BL.1RS derived from Pektus is known to carry the resistance genes *Pm8/Sr31/Lr26/Yr9*, but the resistances to leaf rust *Lr26*, powdery mildew *Pm8* and yellow rust *Yr9* were already overcome in Europe in early 1980s, thus other selective advantages must explain the continued use of the 1BL.1RS translocation within Europe.

The 2NS.2AS translocation, a fragment of 25-38 cM from the short arm of *Aegilops ventricosa* chromosome 2N translocated in bread wheat chromosome 2AS (Helguera et al., 2003), is another important alien translocation that was widely exploited in wheat breeding as it possessed a complex of resistance to rusts *Lr37/Sr38/Yr17* (McIntosh et al., 1995) and a cyst nematode resistance *Cre5* (Jahier et al., 1996). The 2NS fragment was first introgressed into wheat cultivar VPM1 and was later transferred in parallel to resistance to eye spot controlled by *Pch1* on chromosome 7DS to commercial cultivars in France, UK and USA. In Australia, the translocation was initially used to provide rust resistance. Using rust resistance postulation tests, the translocation including the resistance cluster *Lr37/Sr38/Yr17* was shown to be fairly common in European wheat and UK wheat (between 10% to 23% in studies from Pathan et al. (2008), Hovmøller (2007) and Singh et al. (2008)). Similarly a high frequency (40%) of the translocation was revealed in the YR panel and particularly in the UK group using molecular markers. The predominance of the introgression 2NS.2AS from *Aegilops ventricosa* (carrying resistance gene for the three wheat rusts) in UK wheat group compared to the continental group in addition to the quasi absence of *Ppd-D1a* (major photoperiod insensitive allele) suggests that breeders responded to local climatic and pathogen pressures independently of their affiliation. The result of this local adaptation is a reasonably well differentiated (yet homogeneous) UK genepool versus continental genepool.

4.5 Extent of LD

The level of LD is a determinant factor for the success of an association mapping approach, the presence of LD between unlinked markers can lead to false discoveries (as unlinked markers can be found correlated to causal loci) and the level of LD decay intra-chromosomal due to linkage determined the mapping resolution. In presence of a fast LD decay < 1cM over chromosomal distances, the resolution of association mapping would be

rather high at the condition we disposed of high density marker coverage. On the other hand, with a slow LD decay ~10 cM, the chance to identify a marker linked to the causal loci are greater, assuming the same marker coverage, the drawback being a lower resolution.

In the present panel, using genome wide DArT markers, we demonstrated an extensive amount of LD between unlinked markers, 19.9% of inter-chromosomal pair of markers were in significant LD according to TASSEL. The level seems particularly high compared to others studies. Only 5% of unlinked markers were in significant LD in a set of 170 elite spring wheat from CIMMYT (Crossa et al., 2007), 8.1% in a set of 478 elite germplasm from the USA and Mexico (Chao et al., 2010), 3.2% in a set of 90 winter wheat from 21 countries (Neumann et al., 2011). The high level of LD for inter-chromosomal in the YR panel can be explained by the population structure, the high level of relatedness illustrated by pedigree information and the relatively low diversity of the panel. However it is difficult to compare the amplitude of LD level obtained in our study with results obtained in others studies as the size of samples, the marker system as well as the methods used to estimate significant LD vary greatly.

In the YR panel, the LD extend is fairly high 7.3 cM or 16.7 cM depending on the estimation method; the first one is evaluated for r^2=0.2, the second is based on Breseghello and Sorrells (2006b) r^2 critical value, in the present study critical r^2=0.06. Therefore we have a reasonable chance to detect significant loci using our DArT genome wide coverage. Considering only DArT markers with a MAF of 0.05 (1549), our average marker coverage is one DArT marker every 3 cM. In reality as DArT markers have a tendency to cluster (Akbari et al., 2006; Semagn et al., 2006b), our coverage is slightly lower. Recently Marone et al. (2012b) analysed the sequence of 2000 wheat DArT markers and showed that approximately 12% of the sequences were truly redundant (corresponding to nearly identical sequences), which explain the clustering. Despite the apparent adequacy between marker coverage and LD decay, some region of the genome remains uncovered as many gaps have been observed in the genetic map; this will need to be kept in mind when interpreting the results from AM.

The LD decay observed in this study is in the range of reported estimates for genome-wide studies focussed on wheat elite germplasm. Benson et al. (2012) showed an average LD decay of 9.9 cM (for r^2=0.2, evaluated with DArT) considering 251 US elite winter wheat. Reif et al. (2011) saw an extend of LD up to 20 cM in a population of 455 European soft wheat, using SSR markers. Chao et al. (2010) based on SNP data, demonstrated LD decaying from half of its initial value within 6 to 10 cM range (equivalent r^2 ~0.15 to 0.2) in a panel of 478 elite wheat from USA and Mexico. However, other populations showed a faster LD decay, Neumann et al. (2011) found that mean LD decayed permanently below r^2=0.15 within 4 cM in a set of 96 winter wheat originated from 21

different countries. The difference observed in LD decay can be explained by a difference in diversity sampled within each panel; several studies highlighted that LD extended for longer distances in subpopulation (Chao et al., 2010; Somers et al., 2007).

4.6 Structure information and association mapping

It is well known that population structure lead to spurious associations in association studies as it creates correlation between causal factors and (unlinked) non causal markers (Lander and Schork, 1994; Rosenberg and Nordborg, 2006). Having acknowledged the presence of at least two subpopulations/gene pools in the YR panel based on geographical origin, several strategies can be considered for future association studies. (1) The association analysis can be conducted within subpopulations to minimize false positives due to structure as seen in barley (Comadran et al. (2012) for instance focused the analysis on two-rows barley) but this approach will reduce the sample size and most likely the genetic diversity, decreasing therefore the power of the association. This last issue can be resolved by increasing the sampling from respective pools. (2) Alternatively, we can rely on corrective models as many statistical solutions have been proposed to control population structure (Devlin and Roeder, 1999; Price et al.; Pritchard et al., 2000b; Yu et al., 2006). We followed the second option in the present study; the challenge was then to select the optimal association model for our population.

4.7 Association mapping and statistical correction

In addition to historical population structure, the complex evolutionary and breeding history in wheat give rise to complex familial relationships which can also lead to false positive results in AM studies. Therefore to reduce the risk of false positive, an estimate of both population structure and familial relatedness may have to be included in association analysis.

In the present study, in addition to a naive model without correction presenting an extensive number of false positive, five corrective models controlling either population structure, family relatedness or both, were tested using plant height, a trait easy to follow and well characterized in European germplasm. The most appropriated models for the YR panel appeared to be the mixed linear model (MLM) (Yu et al., 2006) integrating a kinship matrix (K) either alone (Model 3) or with Qpca structure matrix based on PCA (Model 5). The kinship matrix defines the degree of genetic covariance among individual and corrects for fine relatedness among individual. Models 3 and 5 performed the best in reducing cofounding population structure and relatedness bias, with P-value distribution most closely resembling a uniform distribution. The large influence of the kinship matrix in the P-value

distribution emphasise the importance of family relatedness within lines in the YR panel, this observation is not surprising as the pedigree information revealed that many varieties share the same parental lines, for instance Rialto and Hussar are the parent of 18 and 17 varieties in the YR panel respectively (see pedigree diagrams in Appendix 8). Limited AM studies on wheat have examined several corrective models; however the use of GLM with Q matrix and/or MLM with Q and K matrix seems to be the most common approaches (Bordes et al., 2008; Breseghello and Sorrells, 2006b; Gurung et al., 2011; Kulwal et al., 2012; Le Gouis et al., 2012; Miedaner et al., 2010; Neumann et al., 2011; Ogbonnaya et al., 2008; Rehman et al., 2012; Yu et al., 2012b). Several authors reported a greater numbers of associated markers with GLM using a Q matrix than for MLM with a Q matrix and a K variance-covariance matrix (Malosetti et al., 2007; Neumann et al., 2011; Pozniak et al., 2012). Recently Le Couviour et al. (2011) discussed the use of different Q matrix (with variable level of co-ancestry (K) to optimize the association for specific traits.

4.8 Significance level and multiple testing

Another problem we face in association mapping is multiple testing, as we are testing numerous hypotheses (one for each marker) in the same dataset. For instance with 100,000 markers, the expectation predicted that approximately 5000 markers will demonstrate a nominal P-value≤0.05 from chance alone, making it difficult to distinguish the true associations from the false. According to DeWan et al. (2007), how to properly adjust for multiple testing is one of the major contentious issues in statistical genetics. We approach this issue in two ways. One method of adjusting for multiple tests is the Bonferroni correction which adjust the cut-off value to declare significance at the α level by the number of test performed (critical P-value = α/ number of markers tested). But this method have been criticised because it assumes independence of all markers which is not true for closely linked markers, therefore the method appears overly conservative for association mapping studies (Zöllner and Pritchard, 2005). An alternative approach is to control the false discovery rate (FDR), the method allows estimating the proportion of significant tests that will be false positive based on the distribution of P-value. It has been shown that this method in more powerful than the Bonferroni correction at the condition there is enough independent markers (Benjamini and Hochberg, 1995; Sabatti et al., 2003; Storey, 2002).

Using this Bonferroni correction we find that only *Rht2* presented a significant MTA using the best performing MLM (namely model 3 and 5), showing that hits above the corrected threshold are very believable. At FDR values of 0.1, we estimated that between 1 to 4 MTA were significant, *Rht2* figured on the top of the list in addition to wPt-4402 on 5BL, SC-Y15 on 2AS and marker wPt-4220 which is assigned to multiple locations (3D and

7A). When it is difficult to conclude about wPt-4220 as the MTA may come from mixed signals from different genomic regions, the MTA associated with wPt-4402 and SC-Y15 are likely to be real. Griffiths et al. (2012) detected height QTLs on 2AS and 5BL in bi-parental populations, these populations include influential lines from the YR panel (Avalon x Cadenza for 2AS; Avalon x Cadenza and Charger x Badger for 5BL). In addition, SC-Y15 was shown to be tightly linked and in strong LD in experimental wheat populations to gwm359 (Rhoné et al., 2007), a marker lying just below the QTL detected in Avalon x Cadenza population, which provides an additive effect between 2.2 to 3.3 cM. In our dataset, FDR of 0.1 provide a highly stringent threshold but does not bring a real improvement comparing to the Bonferroni threshold, as the high number of redundant markers skewed the estimation of FDR. For instance, by removing the P-values linked to the 1BL.1RS translocation, additional markers are found significant based on FDR.

5 CONCLUSION

During the last decade, the complex organization (and re–organization) of wheat breeding program shaped a complex genetic structure in UK winter wheat elite germplasm. Investigating the 327 wheat varieties composing the YR panel, with a large set of DArT markers (1806 in total counting redundant and low frequency marker), we showed a relatively narrow genetic diversity, a high level of LD and a small population stratification based on geographical origin (UK and continental Europe). The rate of LD decay was in line with the markers density making likely the detection of significant association within the panel. The association mapping tests with plant height highlighted large confounding effect due close relatedness within lines and showed that the MLM correction performed well in reducing those confounding effects. The large number of varieties and markers available in the YR panel make it possible for an investigator to initiate a genome-wide association scan with nothing more than a phenotypic screen in place. In the following chapter, we will focussed on resistance to yellow rust but we can imagine to exploit the YR panel via association mapping for a vast range of segregating traits (multiple disease resistance, yield, flowing time, grain quality) as illustrating in the present chapter for plant height.

CHAPTER VI. GENOME WIDE ASSOCIATION SCANS FOR RESISTANCE TO WHEAT YELLOW RUST

1 INTRODUCTION

Association Mapping (AM) arose in medical research and is increasingly applied in crop species. The method allows identifying QTLs within a panel of individuals based on historical linkage desequilibrium. There are several benefits of association mapping over mapping in bi-parental crosses. AM allows to investigate simultaneously multiple quantitative traits (any that vary in the panel under study) and examine more than two alleles per locus (all that occur at reasonable frequency in said panel). In addition, AM is time and cost saving over the development of bi-parental population for mapping studies. Where the population consists mainly of elite varieties in which considerable resource has been invested in the accurate measurement of yield, agronomic and quality traits, further saving can be achieved by using historical phenotypic datasets for detecting marker-trait associations. The UKCPVS and NL/RL trials in the UK constitute a valuable resource of historical yellow rust resistance data for wheat lines registered in the UK and/or evaluated in official pre-registration tests (see Chapter III). These data were derived from adult plant trials inoculated with selected Pst isolates, which represent the most up to date Pst virulence profile in the UK at the time of testing and have been pathotyped at seedling stage against control varieties and *Yr* differential hosts by the UKCPVS. Partitioning this vast amount of historical data and derived means based on different virulence criteria allowed us to target distinct resistance components via association analysis. To do this, we assembled an association panel of 327 elite wheat varieties (for which historical YR data was available) named the YR panel (see Chapter II). The evaluated materials represent the most advanced products of UK wheat breeding programs in the last three decades and are expected to carry a high proportion of relevant, desirable alleles. Therefore, identification of marker-trait associations should be of practical benefit to breeders.

The first objective of the study was to investigate the feasibility of using partitioned historical data to identify QTLs linked to yellow rust resistance at adult stage. Using this approach, we intended to reveal the diversity of yellow rust resistance present in the UK elite germplasm.

Yellow rust is one of the most damaging diseases of wheat worldwide and is particularly renowned for its great propensity to evolve novel virulences and virulence

combinations. The UKCPVS has illustrated the continuous evolution of Pst populations within the UK since late 1960s and has shown in recent years the emergence of new races with ever wider virulence spectra. In this context, the YR panel was evaluated against more recent isolates for which little historic data had yet accumulated (see Chapter IV).

The second objective of the study related in this Chapter was therefore to conduct association analyses using *de novo* phenotypes thus identifying resistance QTL effective against current WYR isolates.

Finally, to validate some of the association hits, the Avalon x Cadenza doubled haploid population which segregates for resistances deployed in recent years in the UK was screened at seedling stage against selected Pst isolates and QTL for resistance were detected using interval QTL mapping.

2 MATERIALS AND METHODS

2.1 Plant material

2.1.1 YR panel

As described in Chapter II, 327 elite wheat lines were selected for the present study. The lines were chosen based on the historical WYR resistance evaluation carried out at NIAB over the last 20 years or for their relevance in the pedigree of modern UK winter wheat varieties. The panel comprised essentially winter wheat varieties bred over the past three decades and adapted to UK growing conditions.

2.1.2 Mapping population

The Avalon x Cadenza (AxC) mapping population was used to identify QTL related to WYR resistance. The population is made of 201 doubled haploid (DH) lines, derived from the F_1 progeny of a cross between cvs Avalon and Cadenza, was developed by Clare Ellerbrook, Liz Sayers and the late Tony Worland (John Innes Centre), as part of a Defra funded project led by ADAS. The parents were originally chosen (to contrast for canopy architecture traits) by Steve Parker (CSL), Tony Worland and Darren Lovell (Rothamsted Research). The population has been widely used by the UK research community to study the genetic basis for several traits including heading date, height and soil-borne cereal mosaic virus resistance (Griffiths et al., 2009; Griffiths et al., 2012) and the most recently published AxC genetic linkage map contains 490 SNP markers as well as 212 SSR and 227 DArT loci (Allen et al., 2011). Seeds of AxC lines were provided by Lesley Fish at the John Innes Centre.

Cadenza is a resistant variety registered on the RL between 1994 and 1998 and rates a 9 and 8 for WYR resistance on the HGCA scale (resistant). Since it was withdrawn from the RL, the UKCPVS has continue to evaluate Cadenza against new WYR races, although no isolate has been found to be virulent on Cadenza at adult stage yet. A few isolates from early 1990s were found to be virulent on Cadenza at seedling stage. Thus Cadenza resistance most likely includes seedling as well as adult stage components. Pathan et al. (2008) postulated the presence of the resistance genes *Yr6* and *Yr7* in Cadenza.

Avalon is a UK variety registered on the RL between 1980 and 1992, believed to carry seedling resistance *Yr3b+Yr4b* (formerly *Yr4*) and the adult plant resistance *Yr14*. However both of these resistances are overcome by modern UK Pst isolates.

2.2 Phenotypic data

2.2.1 Historical data on YR panel

The collection of historical yellow rust adult plant resistance data from the UKCPVS and NL/RL trials has been extensively described in Chapter III. From this extensive dataset, twenty subsets suitable for AM were identified based on virulence criteria (Table VI-1) and adjusted means using BLUP were derived in Genstat 13th as described in Chapter III section 3.1.

Table VI-1: Description of historical adult plant datasets from which adjusted means have been derived for AM studies.

[a] the virulence profile provided for each Pst isolate and pathotype is based on historical seedling tests collected (see Chapter III for historical data description and pathotype definition), vir: presence of virulence factor, avir: absence of virulence factor, ?: indeterminate virulence (either the virulence for the *Yr* gene specified has not been evaluated or the evaluations were contradictory or inconclusive), [b] heritability calculated from a random model fitted to obtain derived means (h^2= genetic variance / sum of variance components).

Virulence criterion	Description: name of Pst isolate and virulence profile criterion	No. derived means	Heritability [b]
None (all historical data)	-	310	0.42
Pst isolate [a]	**1990-505** (vir *1,2,3,4,7* / *avir 6/ ?9,17,32)*	190	0.55
	1993-24 (vir *1,2, 3,4,6,cv* / *avir 7,9 / ?17)*	95	0.59
	1993-54 (vir *1,2, 3,4,6,9* / *avir 32 / ?7,17)*	95	0.58
	1994-519 (vir *1,2, 3,9,17 / avir Yr 4,6,7,32)*	137	0.75
	1996-31 (vir *3,4,6,32 / avir 1,2,7,9,17)*	122	0.56
	1996-502 (vir *1,2, 3,6,9,17 / avir 4,7 / ?32)*	174	0.80
	1998-28 (vir *1,2, 3,4,6,9,17 / avir 32/ ?7)*	94	0.89
	1998-96 (vir *1,2, 3,4,6,9,17,32 / avir 7)*	127	0.86
	1998-108 (vir *3,4,6,17,32 / avir 1,2,7,9)*	101	0.58
	2000-41 (vir *1,2, 3,4,9,17,32 / avir 6,7)*	124	0.67
	2002-70 (vir *1,2,9 / avir 6 / ?3,4,7,17,32)*	93	0.84
	2002-84 (vir *3,32 / avir 9 / ?1,2,4,6,7,17)*	93	0.73
Pathotype regarding nine *Yr* genes [a]	**no7** (vir *1,2, 3,4,6,9,17,32* / avir *7)*	189	0.64
	no7no32 (vir *1,2, 3,4,6,9,17* / avir *7,32)*	141	0.67
	no6no7 (vir *1,2, 3,4,9,17,32* / avir *6,7)*	187	0.66
	no6no7no32 (vir *1,2, 3,4,9,17* / avir *6,7,32)*	92	0.83
	no4no6no7no32 (vir *1,2, 3,9,17* / avir *4,6,7,32)*	166	0.71
Pathotype regarding three UK lines	vir Robigus / avir Solstice	155	0.60
	vir Claire	290	0.47

2.2.2 *De novo* data on YR panel

The YR panel has been evaluated against recent Pst isolates at seedling stage and at adult plant stage in two consecutive years. Chapter IV provides an extensive description of these evaluations. Adjusted means based on BLUP were derived from each assessment (Table VI-2) following the analysis in Genstat 13th as described in Chapter VI section 2.7.

Table VI-2: Description of *de novo* resistance evaluation from which adjusted means have been derived for AM studies.

Phenotype	Assessment description
IF08/21	Infection type scored at 17 dpi from two seedling tests inoculates with Pst isolates 08/21
IF03/07	Infection type scored at 19 dpi from one seedling test inoculated with Pst isolates 03/07
AUDPCr2010	Area under the disease progress curve relative to spreader bed percent infection, calculated between 17 May and 18 June 2010, in field trial 2009/2010 inoculated with 08/21
AUDPCr2011	Area under the disease progress curve relative to spreader bed percentage of infection, calculated between 22 May and 23 June 2011, in field trial 2010/2011 inoculated with a mix of three Pst isolates (08/21, 03/07, 08/501)
Sev2010start, Sev2010mid, Sev2010end	Disease severity (in % leave surface infected) scored respectively 17 May, 4 June and 18 June 2010, in field trial 2009/2010 inoculated with 08/21
HostR2010start, HostR2010mid, HostR2010end	Host response scored respectively 17 May, 4 June and 18 June 2010, in field trial 2009/2010 inoculated with 08/21
Sev2011start, Sev2011mid, Sev2011end	Disease severity (in % leave surface infected) scored respectively 22 May, 9 June and 23 June 2011, in field trial 2010/2011 inoculated with a mixture of three Pst isolates (08/21, 03/07, 08/501)
HostR2011mid, HostR2011end	Host response scored respectively 9 June and 23 June 2011, in field trial 2010/2011 inoculated with a mixture of three Pst isolates (08/21, 03/07, 08/501)

2.2.3 Mapping yellow rust resistance in a biparental mapping population

AxC was evaluated at seedling stage against two UK Pst isolates used to evaluate the YR panel:

(1) 08/21 was found virulent on Avalon and avirulent on Cadenza. 08/21 represents the recent "Solstice race" from the UK which has a wide range of virulence (Table VI-3), it was first isolated in 2008 then spread rapidly throughout the UK to be the predominant race in 2009, 2010 and 2011 (Hubbard and Bayles, 2009, 2010, 2011).

(2) 03/07 was found virulent on Avalon and avirulent on Cadenza. 03/07, which represents the "Brock race", was isolated in 2003 and carries virulence for *Yr7* and Brock.

Both isolates have been evaluated against an extended set of differential host (Chapter IV sections 2.3 and 3.1). Table VI-3 summarizes the virulence profile observed.

Table VI-3: Virulence profile of isolates used in evaluation of AxC population

? Indeterminate virulence for the *Yr* gene specified.

Pst isolate	Pathotype from extended test
08/21	Virulence for *Yr 1,2,3a+4a,3b+4b,6,9,17,20,21,25,26,27,32,A,Sd, Robigus, Solstice* Avirulence for *Yr 5,7,8,10,15,24,SP, Brock* ? *Yr 19,22,23,Mor*
03/07	Virulence for *Yr 1,2, 3a+4a,3b+4b,7,21,22,23,25,26,27,32,A,Sd, Brock* Avirulence for *Yr 5,6,8,9,10,15,24,SP, Solstice,* ? *Yr 19,20,Mor*

The seedlings tests on the AxC population were carried out following the same protocol described for the YR panel in Chapter IV section 2.3. The tests were sown using 96 cells trays, including discard rows around the edge of each tray. Each test was composed of three replicates organized in a complete randomized block. The experimental conditions for each test are provided in Table VI-4.

Table VI-4: Description of seedling tests carried out on AxC population

Pst isolate	Lines tested	Score	Date	Experimental conditions
08/21	201 AxC lines	17DPI	May 2011	Pre and post inoculation in growth chambers, 16 hours light (mixed metal halide and sodium lamps), 18°C day/11°C night.
03/07	201 AxC lines	20DPI	July 2011	

2.3 Genotype data

2.3.1 YR panel

The YR panel was genotyped with 1806 DArT markers from DArT array v3, five genes based markers (*Rht-B1a*, *Rht-B1b*, *Rht-D1a*, *Rht-D1b*, *Ppd-D1a)* and two alien introgression based marker (1BL.1RS, 2AS.2NS) as previously described in Chapter V.

To facilitate the interpretation of genome wide association scans carried on the YR panel, a consensus map including DArT, SSR and SNP markers was developed as described in Chapter V section 2.4. Our consensus map included 722 DArT markers scored on the YR panel (685 assigned to unique location and 37 to several locations within the genome). Additionally, based on pairwise correlation within the YR panel, 356 additional DArT markers were assigned a chromosomal position. The genome coverage of DArTs with known chromosomal location was variable, with sparse coverage on 2A, 4B, 4D, 6D and 5D. Marker density was greater on chromosomes 2B and 3B. The number of mapped markers was greatest on the B genome, followed by the A genome and the D genome.

A DArT consensus map communicated by Andrzej Kilian (Triticarte) in January 2012 was also consulted to locate DArT markers of interest.

2.3.2 AxC DH population

The AxC population has been genotyped with microsatellites, DArT markers from array v2 and SNPs. The mapping data are freely available from the Wheat Genetic Improvement network (WGIN) website (http://www.wgin.org.uk/) and cerealdb website (http://www.cerealsdb.uk.net) (Wilkinson et al., 2012).

2.3.3 Conversion of the wPt-3695 DArT marker into a SCAR marker

Based on the wPt-3695 marker sequence available from Triticarte (http://www.triticarte.com.au/), a new PCR based marker (STSwPt-3695) was developed (Table VI-5) and genotyped on the YR panel as well as AxC population in order to define its map position and test it effects in the bi-parental population.

The adapted marker is a dominant Sequence-Characterised Amplified Region (SCAR) marker amplifying a 457bp fragment. PCR reactions were carried out in 10µl reaction volume, containing 1µM of each primers (AdartwPt-3695-F/R), 200µM of each dNTP, 0.4U of *Faststart* Taq (Roche), 1µl *Faststart* 10x buffer with $MgCl_2$ and approximately 10ng of DNA template. The reaction conditions were 95°C for 6 min; followed by four touchdown cycles where the annealing temperature was decreased by 0.5°C at each cycle starting at 68°C (95°C for 30s, 68°C-66°C for 30s, 72°C 1 min); 30 final cycles with a constant annealing temperature of 66°C (95°C for 30s, 66°C for 30s, 72°C 1 min) and a final extension at 72°C for 10min. The amplification products were detected after migration on a 2% agarose gel.

The genotype (presence/absence) obtained with the adapted marker was identical to the wPt-3695 DArT genotype for 319 lines out of 323 YR panel lines tested. Only one variety (Extend) amplified a fragment when the associated clone was not detected by Triticarte. Three lines (Chatsworth, Dynamo and Arran) did not amplify the fragment while the wPt-3695 clone was detected by Triticarte.

Table VI-5: Primers and product expected for *SCAR* marker developed from wPt-3695

Primer Pair	Sequences 5'-3'	PCR product
AdartwPt-3695-F AdartwPt-3695-R	TGCAGCATCCACATTCTCAT TGCAGGGTGGAGTGCACA	A 457 bp fragment or a poorly amplified smear correspond to the presence or absence of wPt-3695 respectively

2.4 Genetic map for AxC DH population

A linkage map including DArT markers was developed for the AxC population using the program MapDisto 1.7.5 beta (Lorieux, 2012). For the map construction, individuals with high frequency of missing data (<0.2), markers with high frequency of missing data (<0.4) and/or high segregation distortion (p=0.001) were removed. Loci were assembled into linkage groups using likelihood odds (LOD) of 6.0 and a maximum recombination frequency (RF) threshold of 0.3. The linkage groups were ordered using the Seration algorithm and the Sum of Adjacent Recombination Frequencies (SARF) criteria available in MapDisto. The Kosambi mapping function was used to calculate map distances (cM) from recombination frequency. Co-segregating markers have been progressively removed from the map, keeping the markers with lower missing data. However a record of the location of all markers was kept to allow a comparison with the GWA scans. Finally, potential genotype errors detected by MapDisto at a threshold of 0.01 have been replaced by missing data.

2.5 Statistical analysis

The historical and de novo data were analysed respectively in Chapters III and IV, and adjusted means derived.

ANOVA of AxC population seedling tests were carried out with GenStat 13th Edition (Payne et al., 2009). The model (VI-1) was fitted and the adjusted mean for each variety was obtained to carry QTL analysis.

(VI-1)

$$y_{ik} = \mu + V_i + B_k + e_{ik}$$

y_{ik} is the infection type of the variety i, in bloc k; μ is the overall mean; V_i is the effect of the ith variety; B_k is the effect of the kth bloc; e_{ik} is the residual.

To compare the genetic (i.e. heritable) and environmental (i.e. non heritable) sources of variation within a test, heritability was calculated. Replicated data on WYR resistance phenotype was used to estimate genetic variance (σ^2_G) and error variance (σ^2_E) within a trial. Heritability (h^2) was calculated as follow:

(VI-2)

$$h^2 = \sigma_G^2/\sigma_P^2 = \sigma_G^2/(\sigma_G^2 + \frac{\sigma_e^2}{r})$$

Where σ_G^2, σ_P^2 and σ_e^2 represent the variances genetic (variety), phenotypic and residual respectively. r is the number of replication per line. The estimates of variance for σ_G^2 and σ_e^2 were obtained from GenStat output.

The segregation ratio between the resistant and susceptible response groups was tested for its compatibility with monogenic expectations using χ^2 statistic.

2.6 Association mapping

Marker trait association (MTA) tests with yellow rust resistance data were performed within TASSEL v.3.0 software (Bradbury et al., 2007). Markers with MAF<0.05 were removed from the association scans. Adjusted means for the adult plant scores, percentage of infection, severity and AUDPCr, were transformed using log(x+1) prior to analysis to follow a near normal distribution.

The mixed linear model (MLM) was applied, integrating a kinship matrix (K) and a structure matrix (Qpca) derived from seven principal components as this model appeared to be the most efficient for removing the confounding effect due to the YR panel population structure (see Chapter V section 3.4 for more details on population structure of the YR panel and evaluation of alternative AM models). A set of 442 non-redundant DArT markers (r^2>0.6, MAF=0.1) were used to compute the K and Qpca matrix in TASSEL.

Based on (1) map location (P. Bansept consensus map and Andrzej Killian consensus map) and (2) pairwise LD, the markers with an association P-value less than 0.01 were grouped into QTLs. Only groups with at least one MTA with P-value<0.001 for any score considered are reported.

We acknowledge a P-value threshold of 0.01 is relatively low and may include false positives due to multiple testing. However as discussed in the previous chapter, the alternative thresholds provided by the Bonferroni correction and the FDR were both excessively conservative due to the high redundancy and non independence of the markers in our dataset. In addition, the MLM has a known tendency to reduce greatly the MTA significance level even for real association.

The MTAs were additionally scored based on the corrective Bonferroni threshold of 5% to identify the highly significant markers.

2.7 QTL detection in bi-parental populations

QTL analysis of seedling tests were performed using the R/qtl package (http://cran.r-project.org/web/packages/qtl/index.html), a QTL mapping software implemented in R environment. The QTL were detected based on interval mapping function using a Haley-Knott regression. The LOD threshold of detection was derived from one thousand permutations. Percentages of variation explained (r^2) were estimated from single marker analysis with the formula $1\text{-}10^{-2LOD/n}$, where n is the number of lines tested and LOD is the logarithm of the odds ratio.

3 RESULTS AND DISCUSSION

3.1 GWA scans on historical data

Considering all scores analysed for genetic association in the present study (historical and *de novo*), 464 markers with at least one MTA at P-value<0.01 were found. Based on map location (markers within a 10cM interval) and pairwise LD (r^2<0.5), these markers have been grouped in 108 potential QTLs. Of these, 38 included a MTA significant at 0.001 P-value thresholds and were assigned a chromosomal location (Table VI-19). Three other groups presented significant MTA at P-value<0.001 but were unmapped. Only MTAs belonging to the 36 groups are presented further. A complete list of markers with significant MTA within these groups is available in Appendices 13 and 14.

3.1.1 Analysis by isolate

The set of 1806 DArT markers was reduced to conserve a MAF of 0.05 prior to the analysis of each isolate-specific phenotype. Depending on the isolate considered (thus the number of varieties evaluated), between 1384 and 1505 DArT markers were analysed. A total of 111 markers were shown to be associated with resistance to one or more Pst isolate(s), but many of these were redundant. Seventeen markers were found to be highly correlated (r^2>0.8) to marker SC-Y15 linked to *Yr17* (list in Appendix 15). Similarly 51 DArT markers on 3DS mapping within 2.8 cM interval, were found associated with resistance to five Pst isolates (1998-28, 1998-108, 2000-41 2002-70 and 2002-84) (see list in Appendix 15). The 119 MTAs were distributed in 20 groups (Table VI-6), of which four included markers highly significant using the Bonferroni correction threshold (P-value<0.00003). Some MTA groups were specific to one Pst isolate, for instance groups 2A1, 2A2, 2D1, 6A1 and 7B3. Others were detected with four or more isolates like 2B1, 2B4, 3B, 4A and 4D1.

Table VI-6: Groups of MTAs associated with twelve Pst isolates and statistics

from the MLM

[a] chromosomal location from P. Bansept consensus map, [LD] chromosomal location based on LD within YR panel, [b] chromosomal location from A. Killian consensus map, [c] effect based on allele 1 (presence of the DArT clone) and transformed scores e.g. log (percent of infection+1), [d] marker with a low frequency not tested with all isolates, * P-value<0.01, ** P-value<0.001, *** P-value<0.0001, B 5% Bonferroni correction

Group	Major MTA	cM	Significant isolates	r^2	Effect [c]	Allele distribution 1/0
1B1	wPt-2694	36.6 [b]	2000-41*	5.4	+0.196	30/90
			2002-70***B	17.9	+0.450	17/73
2AS.2NS	wPt-744900	12.0 [LD]	1993-24**	11.5	+0.338	44/50
			1993-54*	8.3	+0.297	44/50
2AS.2NS	SC-Y15(*Yr17*)	-	1993-54*	7.9	-0.282	49/45
2A1	wPt-1657	71.2 a	1998-108*	8.8	-.343	88/12
2A2	wPt-4021	unknown	1994-519**	9.9	-0.347	73/55
2B1	wPt-2293	84.2 [b]	1993-54**	11.9	-0.280	33/61
			1996-502***B	12.0	-0.323	70/103
			1998-28**	13.4	-0.341	41/53
			1998-96***B	13.3	-0.367	50/77
2B2	wPt-1489	53.6 [a]	1998-108**	10.6	-0.402	10/91
			2000-41*	5.1	-0.240	15/108
2B4	wPt-3695	180.6 [a]	1990-505***B	8.7	-0.200	45/145
			1993-24**	14.8	-0.400	15/80
			1993-54**	13.3	-0.400	15/80
			1996-31**	10.8	-0.249	41/81
			1998-108*	7.9	-0.286	28/73
			2000-41*	6.8	-0.212	42/82
			2002-70***B	19.7	-0.423	40/53
			2002-84**	12.8	-0.286	40/53
2D1	wPt-6419	49.7 [b]	1996-502**	6.7	+0.378	150/19
2D2	wPt-667054	101.2 [b]	1990-505*	5.4	+0.153	135/54
			1998-108**	11.4	+0.317	75/25
3B1	tPt-7594	132.5 [b]	1993-24**	12.0	-0.330	57/37
			1993-54**	7.0	-0.260	57/37
3D1	wPt-741820 [d]	8.5 [a]	1998-108**	10.8	+0.503	14/87
			1998-28*	8.1	+0.650	8/86
			2002-70*	10.1	+0.533	12/80
			2002-84**	12.4	+0.489	12/80
4A	wPt-8657	20.7 [a]	1990-505*	4.1	-0.257	178/12
			1996-31*	6.0	-0.344	109/13
			2002-70**	12.3	-0.580	81/12
			2002-84*	11.0	-0.454	81/12
4D1	Rht-D1a/b	20.5 [a]	1998-108*	6.3	-0.331	89/11
			1998-96*	5.9	-0.445	116/10
5B2	wPt-8604	57.2 [a]	1990-505*	3.9	+0.180	174/15
			1993-24*	7.9	+0.400	88/6
5B3	wPt-3763 [d]	94.7 [b]	2002-70*	8.3	-0.785	86/7
			2002-84**	12.6	-0.794	86/7
6B1	wPt-2424	83.2 [a]	1996-31*	8.1	-0.332	111/8
			1998-108*	6.4	-0.380	95/5
			2002-70*	7.6	-0.4528	85/6
			2002-84**	14.7	-0.5179	85/6
7A1	wPt-740561	40.6 [a]	1996-502*	4.9	+0.294	17/157
			1998-108**	10.1	+0.428	7/94
7B2	wPt-4814	113.1 [a]	1990-505*	4.0	+0.121	78/101
			2000-41**	7.9	+0.198	51/67
			2002-70*	7.8	+0.255	37/53
7B3	wPt-0752	141.6 [a]	1994-519***B	22	+0.544	83/48

3.1.1.1 Factors influencing MTA detection in historical data

The specificity of association with one or a limited number of pathotype(s) may suggest race specificity of the putative underlying resistance factor. However it is important to remember that many factors could influence the significance of a marker in association studies, including the heritability of the trait in question, the number of genes controlling the trait of interest, the effect of the marker itself (major qualitative effect or smaller quantitative effect) as well as the allele frequency of the marker. Therefore if a resistance gene is not detected, it does not necessarily mean the gene was not present and/or not efficient; it is equally possible that confounding factors may have limited its detection.

With many genes involved, the signal for one specific gene can be diluted to an undetectable level. In the case of yellow rust, it is known that many genes influence the resistance response at the adult stage. At the time of writing 52 *Yr* genes for resistance to yellow rust has been formally named, 37 of which are seedling resistances with major effect, 15 are adult plant resistances likely to present a smaller quantitative effect (see Chapter I section 4.3 for complete list). Additionally a large number of QTLs have been described as contributing to yellow rust resistance (see Appendix 2 for list of QTLs). Taking only the YR panel into account, we highlighted in Chapter IV the presence of race specific resistance genes *Yr6, Yr7, Yr9* and *Yr17*. Additionally, we noticed the rare occurrence of *Yr15* (Boston, Ochre) and *YrSP* (Spalding Prolific) as well as the presence of uncharacterized sources of adult plant resistance. Furthermore based on the literature review, seedling resistance genes *Yr1, Yr2, Yr3a, Yr3b Yr4a, Yr4b, Yr25, Yr32* and adult plant resistance genes *Yr11, Yr12, Yr13, Yr14, Yr16* are also known to be present in some YR panel lines (see Appendix 3 for *Yr* genes postulated in YR panel lines). Only with this brief description, we identified 17 genes likely to control the resistance response in historical data. Considering that in the present analysis we are looking in turn at different subsets of the YR panel and different isolates, the set of discoverable YR resistance genes in each analysis is likely to vary as a function of the different Pst isolate specificities and the subsets of varieties tested.

For a similar marker effect, alleles at a low frequency are less likely to be detected compared to alleles at balanced frequency as they would provide a stronger overall signal. As we are investigating different subsets of the YR panel, allele frequencies are different for each isolate/marker combination, which limit the chance to observe a constant significant MTA.

Finally, our analyses are based on adjusted means derived from unbalanced historical data with low to intermediate levels of heritability (Table VI-1). The great

influence of climatic conditions on yellow rust epidemic (environmental factor), the presence of natural contamination of inoculated trials as well as the specificity of varietal response to environmental condition (genetic x environmental factor) explained the reduced level of heritability observed. It is known the power of detection of a MTA increases with trait heritability thus the chance of detecting a specific effective gene will not be equal for each data subset.

3.1.1.2 Detection of *Yr17*

Taking the example of the major race specific gene *Yr17* identified by markers in the group 2AS.2NS, significant MTAs were detected with two isolates 1993-24 and 1993-54 (Figure VI-1). Despite a well balanced distribution, MTAs for *Yr17* were not detected with isolate 1996-31 which is known to be avirulent on *Yr17*. The yellow rust scores for varieties carrying the allele specific to the translocated segment 2AS.2NS from *Aegilops ventricosa* (SC-Y15) ranged from 0 to 0.78, 0.89 and 1.21 respectively for the isolates 1993-24, 1993-54 and 1996-31 (Figure VI-1). As *Yr17* is a seedling resistance gene with major effect, percentages of infection above 5% (equivalent to a 0.78 YR score after transformation) are not likely when tested against isolates avirulent on *Yr17*. This observation suggests either the presence of cross-contamination of historical trials with isolates virulent against *Yr17* or the presence of varieties without *Yr17* within the lines presenting the "2NS" allele (presence of segment 2NS from *Aegilops ventricosa*). The reality is likely to be a combination of both these factors.

In Chapter III section 4.51, we highlighted the presence of contamination by natural WYR population virulent on *Yr6* and *Yr17* in year 1996, 1997 and 1998 for the isolates1993-24 and 1993-54, this is also true for trials inoculated with 1996-31.

When carrying out the GWA scans on isolate 1993-24 and 1996-54, the STS marker SC-Y15 from Robert et al. (1999) was not the most significant marker (Table VI-7), as we misdiagnosed five varieties using the assay and the genotype was missing for 20 lines (6% of the YR panel). Looking at DArT markers in the 2AS.2NS group, it was obvious Glasgow, Hyperion, Shamrock were wrongly diagnosed to carry the 2AS.2NS segment. Conversely, Rampart and Rosette were likely to carry the 2AS.2NS segment (as judged by DArT marker profile but did not amplify the specific SC-Y15 fragment characteristic of its presence. Markers wPt-8242, wPt-669721, wPt-8464 and wPt-3676 appeared to be perfect markers for the 2AS.2NS introgression and by extension for *Yr17* in the YR panel. It should therefore be possible to use a one or more of this group of DArT markers (wPt-8242-wPt3676, wPt-669721-wPt-3676 or wPt-8464-wPt-3676) as co-dominant markers to diagnose the presence of 2AS.2NS introgression. Finally it is interesting to notice that despite presenting the strongest MTA, marker wPt-744900 was not a perfect marker for *Yr17*

against isolate 1993-24 (P-value=0.0009). Looking at the complete panel, wPt-744900 misdiagnosed four varieties (Astron, Option, Ranger and Veritas) and had 11 missing data points.

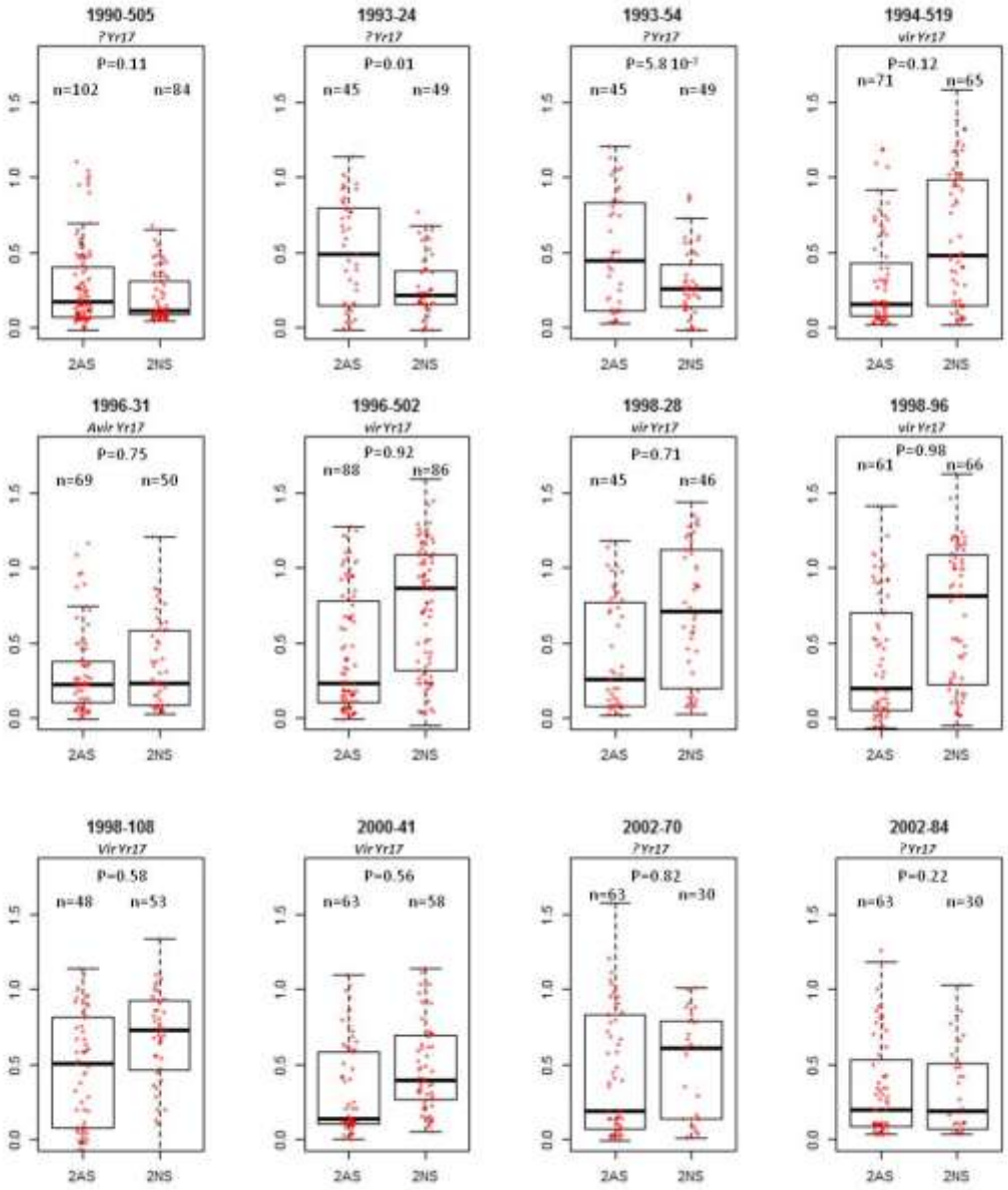

Figure VI-1: Combined box plots and dot plots of YR rust scores for each isolate depending of the genotype for SC-Y15 marker specific of 2AS.2NS translocation carrying *Yr17*

Allele 2AS: absence of segment 2NS including *Yr17;* allele 2NS : presence of segment 2NS including *Yr17;* p: P-value from MLM association test; n: number of allele in each subset. The red dots are adjusted means for each line included in the test. The scores presented are transformed scores e.g. log (percent of infection+1). The isolate used to inoculate the trials from which adjusted means were derived and its respective virulence for *Yr17* is indicated on the top of each boxplot.

Table VI-7: Significance of MTA for selected markers associated with *Yr17*

	SC-Y15	wPt-8242	wPt-669721	wPt-8464	wPt-3676	wPt-744900
Allele associated with resistance	1(580bp)	1	1	1	0	0
P-value for 1993-24	0.010	0.005	0.004	0.004	0.004	0.0009
P-value for 1993-54	0.006	0.006	0.005	0.005	0.005	0.005

3.1.1.3 Absence of detection of *Yr9*

Despite the tests being done with Pst isolates avirulent for *Yr9* namely 1993-24, 1996-31, 1998-108 and 2002-84, no markers linked to the translocation 1BL.1RS carrying *Yr9* was detected. The dot plots of adjusted means for the four isolates avirulent on *Yr9* showed high YR scores (susceptibility) in some lines carrying *Yr9* (Figure VI-2), which is not expected as *Yr9*, a major seedling resistance gene, should provide a complete protection against the four isolates. Those observations could have two origins: (1) biased estimates of the YR scores for some of the lines due to the highly unbalanced historical dataset, (2) the presence of contamination of some field trials with Pst isolates virulent on *Yr9*. Looking back at the raw data, many trials inoculated with *Yr9* avirulent isolates presented natural Pst infection with virulence for *Yr9*, based on the high percentage of infection observed on lines carrying *Yr9* e.g. Clement, Haven, Hornet.

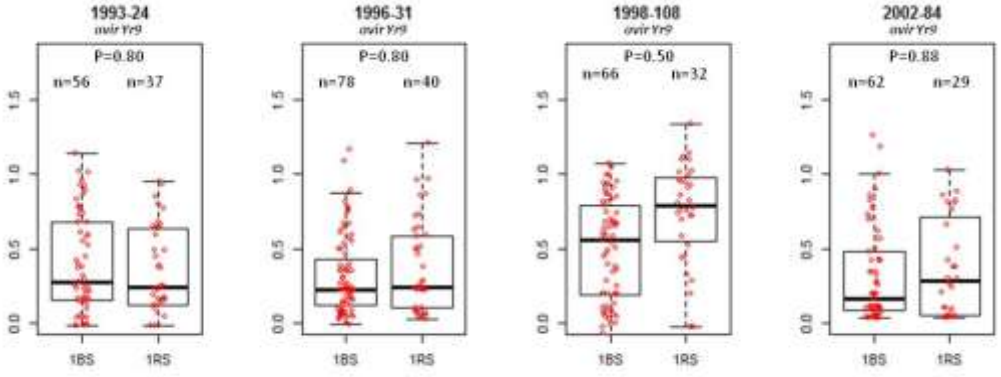

Figure VI-2: Combined box plots and dot plots of transformed YR scores for isolates avirulent on *Yr9* in lines with and without the 1BL.1RS translocation

Allele 1BS: absence of segment 1RS including *Yr9;* allele 1RS : presence of segment 1RS including *Yr9;* p: P-value from MLM association test; n: number of allele in each subset. The red dots are adjusted means for each line included in the test. The scores presented are transformed scores e.g. log (percent of infection+1). The isolate used to inoculate the trials from which adjusted mean were derived and its respective virulence for *Yr9* is indicated on the top of each boxplot.

3.1.2 Analysis by pathotype and identification of resistance matching virulence factors 4, 6, 7 and 32

Depending on the pathotype considered (and thus the number of varieties evaluated), between 1475 and 1561 DArT markers were analysed. A total of 51 markers were identified to be linked with resistance to one or more pathotype(s) and were distributed in 14 MTA groups (Table VI-8). Two groups included markers significant using the Bonferroni correction threshold (P-value<0.00003), these groups pointed to QTLs on 2B (group 2B4) and 7B (group 7B3).

Many MTA groups were specific to one pathotype, for instance groups 2B1, 2B2, and 2D3, which may again seem to suggest "pathotype" specificity of the underlying resistance locus. However, the same remarks made for the isolate based analysis section 3.1.1.1 are applicable to pathotype based analysis. The detection of an MTA in historical datasets is influenced by many factors (heritability, number of gene involved, number of varieties tested, marker frequency ect), thus the absence of detection of an MTA is not synonymous of the absence of a QTL. Moreover, the use of pathotype instead of isolate description to sort the historical data, introduced an additional source of variation as virulence factors not identified by virulence testing can segregate amongst the Pst isolates included in each pathotype.

Table VI-8: Groups of MTAs associated with specific pathotype definition

[a] chromosomal location from P. Bansept consensus map, [LD] chromosomal location based on LD within YR panel, [b] chromosomal location from A. Killian consensus map, [c] effect based on log(YR score+1), * P-value<0.01, ** P-value<0.001, *** P-value<0.0001, B 5% Bonferroni correction.

Group	Major MTA	cM	Score and significance	r^2	Effect [c]	Allele distribution 1/0
2A2	wPt-4021	unknown	no4no6no7no32*	8.38	-0.317	94/60
2B1	wPt-2293	84.2 [b]	no7*	4.65	-0.182	71/115
2B2	wPt-1489	53.6 [a]	no7*	3.68	-0.242	22/166
2B4	wPt-3695	180.6 [a]	no7** no7no32** no6no7***B no4no6no7no32**	6.72 9.08 10.73 6.85	-0.259 -0.313 -0.289 -0.290	53/136 49/92 57/130 46/120
2D2	wPt-667054	101.2 [b]	no7***	8.37	+0.288	135/51
3B2	wPt-6785	210.0 [a]	no7no32*	6.99	-0.235	57/77
3B3	wPt-10537	272.2 [a]	no6no7no32**	16.38	-0.462	30/58
3D1	wPt-741820	8.5 [a]	no6no7*	3.83	+0.265	21/165
4D2	wPt-8657	20.5 [a]	no7no32***	11.47	+0.205	124/15
5B1	wPt-0708	15.2 [b]	no6no7no32**	14.21	-0.452	62/29
5B2	wPt-8604	57.2 [a]	no6no7*	4.67	+0.275	169/16
6A2	wPt-9584	62.1 [a]	no4no6no7no32**	6.99	-0.382	147/17
6B1	wPt-2424	83.2 [a]	no7* no6no7*	4.66 4.99	-0.331 -0.336	174/11 170/11
7B3	wPt-0752	141.6 [a]	no6no7no32*** no4no6no7no32***B	17.21 15.69	+0.578 +0.458	59/30 103/55

The association scans with our pathotypes target four virulence factors corresponding to a few race specific genes known to be present in the YR panel line (*Yr4, Yr6, Yr7 and Yr32*). Thus though our pathotype based analysis, we aimed to identify these specific genes.

3.1.2.1 Virulence factor 4

Virulence factor 4 is based on response observed on the differential host Hybrid 46 and was formerly thought to be specific to *Yr4*. According to Lupton and Macer (1962), Hybrid 46 carries two resistance genes *Yr3* allele b and *Yr4* allele b based on their allelism to *Yr3* allele a and *Yr4* allele a in Cappelle Desprez. We used the nomenclature *Yr3b+Yr4b* throughout the thesis based on those observations; however the location and number of genes mediating susceptibility to the virulence factor 4 remain uncertain. Worland (1988) located *Yr4* on chromosome 3B. Later Chen and Line (1993b) suggested Hybrid 46 does not carry *Yr3b* and reported that Hybrid 46 has two resistance genes namely *Yr4b* and *YrH46*. Subsequently, Chen et al. (1996) showed using crosses between Hybrid 46 and a set of aneuploids that *Yr4b* and *YrH46* were on chromosome 6B and 6A respectively. They also

showed that *Yr3a* and *Yr3c* present in Nord Desprez and Minister respectively mapped to 1B. Recently, Bansal et al. (2010) identified a seedling resistance gene *YrRub* in cv Rubric located to 3BS. Based on markers polymorphism and seedling tests with an Australian isolate, they suggested *YrRub* was present in Hybrid 46 and Avalon, and was likely to be *Yr4*.

In our dataset, only pathotype "no4no6no7no32" should allow the identification genes controlling resistance against virulence factor 4 present in Hybrid 46 (*Yr3b, YrH46, Yr4b or YrRub).* The pathotype "no4no6no7no32" specifically identified markers on 6A, potentially consistent with previous reports of the location of *YrH46*, but no markers were identified on 3B (Chromosome assigned to *YrRub*) or 6B (chromosome assigned to *Yr4*b).

Marker wPt-9584 presented the higher MTA in group 6A2 and the allele providing resistance "1" was frequent in the YR panel (147 lines of 164 tested)(Figure VI-3). Furthermore, allele1 was present in cvs postulated to carry "*Yr4"* (Hybrid 46, Avalon, Claire, Dynamo, Flame, Hereward, Moulin, Savannah and Shango, see Appendix 3). As Moulin and Hybrid 46 appear in the pedigree of many YR panel lines, it would not be surprising to find loci associated with "*Yr4*"at high frequency. Thus, markers in cluster 6A2 may point to *YrH46* on 6A. However the skewed distribution of YR scores toward low values and the highly unbalanced allele leave question marks over the reliability of this MTA (Figure VI-3).

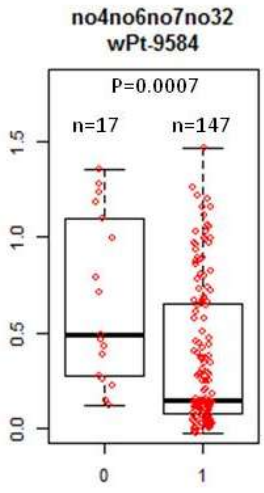

Figure VI-3: Combined box plots and dot plots of YR rust scores for wPt-9584 specific of the pathotype "no4no6no7no32"

Allele 0: absence of hybridisation to DArT clone*;* Allele 1 : positive detection of hybridisation to DArT clone*;* p: P-value from MLM association test; n: number of genotypes in each allelic class. The red dots are adjusted means for each line included in the test. The scores presented are transformed scores e.g. log(percent of infection+1).

3.1.2.2 Virulence factor 6

Virulence factor 6 is considered equivalent to virulence for *Yr6* in the present study. The presence/absence of virulence factor 6 is determined based on infection type observed on differential hosts Heines Kolben (*Yr2, Yr6*) or Heines Peko (*Yr2,Yr6,Yr25*) in conjunction to avirulence for *Yr2*. Thus the absence of *vir6* in a particular isolate may be caused by *Yr25*, however *Yr25* is believed to be widely spread in European isolates (see Chapter III section 4.4) and is not likely to participate to the resistance of Heines Peko against UK isolates. Hovmøller (2007) indicated that *Yr25* does not confer yellow rust control in North West Europe as the matching virulence is close to fixation in present Pst population. Additionally *Yr25* was assigned to chromosome 1D (Calonnec et al., 1997) and no MTAs were detected on chromosome 1D (all analyses considered).

Yr6 is believed to be well represented in the YR panel as previously discussed in Chapter IV section 4.1.1.2, and was mapped to chromosome 7BS by Elbedewy and Robbelen (1982) based on crosses with susceptible monosomic lines. Apart from its assignment to chromosome arm 7BS, little is known about the chromosomal location of *Yr6*, the mapping of *Yr6* in Avalon x Cadenza population will therefore shed some light on this matter (see section 3.2.1.3).

MTA group 7B3 was identified using two pathotypes avirulent on *Yr6* and included a highly significance marker (wPt-0752) likely to be linked *Yr6*. Looking at distribution of YR scores based on wPt-0752 alleles (Figure VI-4), allele 0 is associated with lower YR score on average for the three pathotypes avirulent on *Yr6* ("no6no7", "no6no7no32" and "no4no6no7no32"), while the inverse is observed for pathotype virulent on *Yr6* (no7). Among the lines carrying allele 0, figured many varieties known to carry *Yr6* e.g. Haven, Norman, Hornet, Comet, Cadenza, Madrigal, Lynx afp 856, Hunter, Equinox, Shango, Rialto and differential host Heines Kolben and Heines Peko. However allele 0 is also found in varieties without *Yr6* such as Brock, Thatcher; and some lines postulated to carry *Yr6* (Moulin, Charger, Riband and Spark). Thus wPt-7052 does not constitute a diagnostic marker but is likely to be closely linked to *Yr6*. Increasing the marker density within the vicinity of wPt-7052 may identify a diagnostic marker.

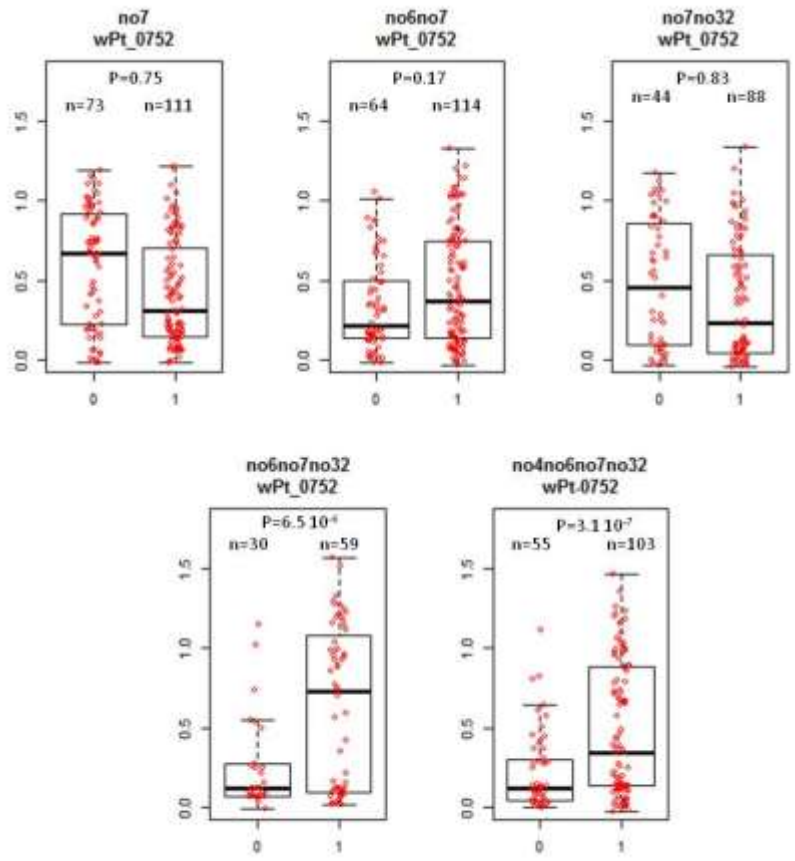

Figure VI-4: Combined box plots and dot plots of YR rust scores for wPt-0752 with five pathotypes

Allele 0: absence of hybridisation to DArT clone*;* Allele 1 : positive detection of hybridisation to DArT clone*;* p: P-value from MLM association test; n: number of genotypes in each allelic class. The red dots are adjusted means for each line included in the test. The scores presented are transformed scores e.g. log (percent of infection+1).

3.1.2.3 Virulence factor 7

Virulence factor 7 is assumed to equate to virulence for *Yr7* in the present study. The presence/absence of vir 7 is determined based on infection type observed on four differential hosts Lee (*Yr7, Yr22*, *Yr23*), Brock (*Yr7*), Reischerberg 42 (*Yr7*, *Yr25*) or Tommy (*Yr7*). Thus the absence of vir 7 may be associated with *Yr22, Yr23* or *Yr25*. For the same reasons mentioned in previous paragraph, *Yr25* is not likely to participate to the resistance of Reischerberg 42 against UK isolates. *Yr22* and *Yr23* have been identified in Lee against North American Pst isolates by Chen et al. (1995a) and were assigned respectively to chromosomes 4D and 6D. The presence of *Yr7* in Lee has been reported by Macer (1975) and is based on evaluation against UK isolates, but he pointed out that the designation was not proven by a complete set of diallel crosses. Therefore the effectiveness of *Yr22* and *Yr23* against UK isolates cannot be ruled out. Nevertheless no MTAs were detected on 6D (where

Yr23 was assigned) in the present study. On the other hand, some MTAs were detected on 4D with pathotype "no7no32" (group 4D1) as well as isolates 1998-108 and 1998-96 which do not carry the virulence factor 7 (group 4D2). These MTA may well be consistent with the reported location of *Yr22* but in absence of precise map location and knowledge of lines in the Yr panel carrying *Yr22*, we are not able to confirm it.

Yr7 is believed to be present in the YR panel but at a relatively low frequency based on postulations made in Chapter IV section 4.1.3. According to infection type observed at seedling stage against isolate 08/21 and 03/07, ten lines are thought to carry *Yr7* alone (Brock, Camp Remy, Cordiale, Ekla, Spark, Tara, Thatcher, Tommy, Tonic, Vault) and up to 63 lines could carry *Yr7* in combination with another effective *Yr* gene as Cadenza (*Yr6,Yr7*).

Yr7 mapped to chromosome 2BL, 21cM from the centromere according to Law (1976) and is believed to be allelic to yellow rust resistance genes *Yr5* and *YrSP* (Zhang et al., 2009). Three MTAs groups were detected on 2B e.g. 2B1, 2B2 and 2B4. Considering group 2B4 was found associated with isolate 1990-505 avirulent on *Yr7* in the previous analysis, it is not likely to be linked to *Yr7*. Markers from group 2B2 mapped to 2BS (position 53.6 cM in our consensus map, see Appendix 11). Marker wPt-2293 (highest significant marker on group 2B1) was not mapped on our consensus map but was located on 2BL on consensus map from A. Killian (position 84.1cM), wPt-2293 was also located on 2BL in the consensus map developed by Marone et al. (2012a). Additionally, in Marone et al. (2012a) map, wPt-2293 was 6.1cM away from SSR marker wmc175 which is associated with *Yr7* in cvs Camp Remy (Mallard et al., 2008). Looking at the allelic distribution of wPt-2293, allele 1 which is associated with resistance, was present in 136 lines within the YR panel. Among these, only Ekla was postulated to carry *Yr7*, while allele 0 was present in eight lines postulated to have *Yr7* (Cadenza, Tonic, Cordiale, Brock, Camp Remy, Spark, Tommy, Tara). Thus, we conclude that wPt-2293 is not linked to *Yr7*. WPt-732882 was the second marker in group 2B1 and is highly correlated with wPt-2293 (r^2 =0.81), allele 1 was associated with resistance and was present in 129 lines, seven of them were postulated to carry *Yr7* (Cadenza, Tonic, Cordiale, Brock, Camp Remy, Thatcher, Ekla). WPt-732882 allele 1 was also present in *Yr7* differential host Lee, but no genotype was available for Spark, Tommy and Tara. Thus, we conclude that wPt-732882 is probably loosely linked to *Yr7*. Similarly to *Yr6*, to identify a marker able to distinguish clearly varieties with and without *Yr7*, a higher marker density close to wPt-732882 would be needed.

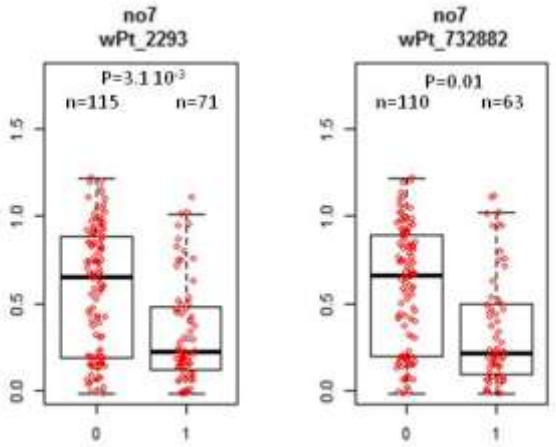

Figure VI-5: Combined box plots and dot plots of YR rust scores for wPt-2293 and wPt-732882 (MTA group 2B1) with pathotype "no7"

Allele 0: absence of hybridisation to DArT clone*;* Allele 1 : positive detection of hybridisation to DArT clone*;* p: P-value from MLM association test; n: number of genotypes in each allelic class. The red dots are adjusted means for each line included in the test. The scores presented are transformed scores e.g. log (percent of infection+1).

3.1.2.4 Virulence factor 32

Virulence factor 32 is based on response observed on the European differential host Carstens V, however it may cover several resistance genes considering studies from Chen and Line (1993a) and Lewis (2006). Stubbs (1985) referred to one presumed resistance gene in Carstens V as *YrCV*. Based on segregation tests in crosses between Carstens V and susceptible lines, Chen and Line (1993a) concluded three resistance genes from Carstens V (*YrCV1, YrCV2, YrCV3*) were effective against selected North American Pst isolates. While in Australia, according to McIntosh et al. (1995), only one gene was detected against Australian Pst isolates. In 2003, using an Australian isolate, Eriksen et al. (2004) identified a major race specific gene in Carstens V namely *Yr32*, which was presumed to be equivalent to *YrCV*. *Yr32* was then mapped in cv. Senat on chromosome arm 2AL within a 5cM interval between SSR marker wmc198 and wmc181 (Eriksen et al., 2004). Later, Lewis (2006) postulated the presence of seedling resistance genes *Yr25, Yr32, YrCV, YrSD* and maybe *Yr3* or *Yr4* in Carstens V using an array of 26 European Pst isolates. *Yr32* and *YrCV* were considered to be distinct in this latter study contrasting with the Eriksen et al. (2004) postulation. Throughout this thesis, we used the nomenclature *Yr32* throughout the thesis as equivalent to *YrCV*.

Yr32 is believed to be present in at least 11 lines from the YR panel based on literature review (Charger, Comet, Consort, Encore, Hereward, Hunter, Oxbow, Robigus, Vivant, Windsor and Carstens V, see Appendix 3 for gene postulation). As Carstens V,

Consort and Charger are present in the pedigree of many lines, we expect *Yr32* to be present at a high frequency in the YR panel. Only one marker on 2A (wPt-4021) presented a significant MTA (P-value =0.0003). WPt-4021 allele 1 was associated with lower YR scores (resistance) with the pathotype "no4no6no7no32" (Figure VI-6) but no map position was available. Allele 1 was present in 180 YR panel lines, among them were found ten lines postulated to have *Yr32*. However, Carstens V, which is known to carry *Yr32*, was genotyped with allele 0 for genotype. It is noteworthy that in the Quality Control data supplied, Triticarte assigned an average P score of 0.75 for wPt-4021 genotype, meaning it may contain a higher rate of scoring error. Normally, Triticarte recommend a P score of 0.8 for high reliability. Apart from Robigus (YR score 0.423), lines postulated with *Yr32* had a YR score less than 0.15 with pathotype "no4no6no7no32", corresponding to a highly resistant response (1.4% infection). Among the lines with allele 1 were also many lines without *Yr32* such as Vuka, AC Barrie, Slejpner, Brock and Tonic.

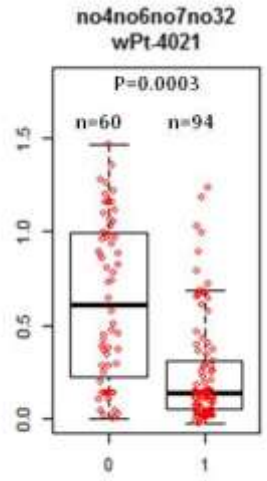

Figure VI-6: Combined box plots and dot plots of YR rust scores for wPt-4121 against pathotype "no4no6no7no32"

Allele 0: absence of hybridisation to DArT clone*;* Allele 1 : positive detection of hybridisation to DArT clone*;* p: P-value from MLM association test; n: number of genotypes in each allelic class. The red dots are adjusted means for each line included in the test. The scores presented are transformed scores e.g. log (percent of infection+1).

WPt-4021 may point to a resistance gene specific to *vir 32*; however considering the information we dispose of, this is difficult to conclude with certainty. WPt-4021 will need to be located on a map integrating SSR markers to compare its location with *Yr32*. Furthermore a seedling test with Pst isolates differing for vir 32 would help with the postulation of *Yr32* in YR panel lines.

3.1.3 Analysis based on virulence response against selected varieties

3.1.3.1 Pathotype virulent on Claire

Claire is a variety released in 1999, susceptible at seedling stage against common UK isolates but resistant at adult stage. Lewis (2006) postulated Claire has five seedling resistance genes, *Yr2, Yr3, Yr4, Yr25* as well as another unknown seedling resistance gene. In addition, Claire was found to exhibit quantitative APR, controlled by four QTLs, one on 2BL, two on 2DL and one on 7B (Powell, 2010). Despite being grown widely in the UK, Claire maintained its adult plant resistance until 2012, when the new emerging "Warrior" race reduced its level of resistance in the field.

By sorting the historical data based on the virulence for Claire at seedling stage, we aimed to focus the association scan toward Claire APR. Nevertheless, it should be remembered that the trials used to derived means for pathotype "Virulent on Claire" have been inoculated with isolates with mixed virulence for major genes known to be present in the YR panel e.g. *Yr6, Yr7, Yr9, Yr17, Yr32* (see Chapter IV section 4 for more details).

After application of a MAF threshold of 0.05, 1540 of 1806 markers were included in the association analysis with pathotype "Virulent on Claire". 38 markers were identified to be linked with resistance to pathotype "vir Claire" and were distributed over six MTA groups (Table VI-9) assigned to chromosomes 2B, 3D, 4D, 6A and 7A. None of the MTAS seemed related to Claire adult plant resistance identified by Powell (2010). Marker wPt-2293 (MTA group 2B1) and wPt-3695 (MTA group 2B4) mapped respectively 17.5 cM and 45.8 cM away from marker wPt-9190 associated with the 2B QTL from Claire (Powell, 2010) on the consensus map from A. Killian (Triticarte). Instead of identifying adult plant resistance, the most significant markers pointed to seedling resistance as all MTA groups except 3D and 4D were also detected in *de novo* seedling tests (see sections 3.2.1 and 3.2.2). The presence of major YR genes within the YR panel, effective against some Pst included in the dataset, has probably masked the action adult plant QTLs with more moderate effect. Additionally, the adult plant QTLs found in Claire may not be deployed at a large frequency in the YR panel, which could limit even more their detection.

Table VI-9: Groups of MTAs associated with pathotype virulent on Claire at seedling stage

[a] chromosomal location from P. Bansept consensus map, [LD] chromosomal location based on LD within YR panel, [b] chromosomal location from A. Killian consensus map, [c] effect for allele 1 given in log (percent of infection+1), * P-value<0.01, ** P-value<0.001, *** P-value<0.0001, B 5% Bonferroni correction

Group	Major MTA	cM	P-value	r^2	Effect [c]	Allele distribution 1/0	Markers significant in group
2B1	wPt-2293*	84.1 [b]	2.45E-3	3.20	-0.139	116/170	1 of 2
2B4	wPt-3695***B	180.6 [a]	1.55E-6	7.77	-0.249	74/216	5 of 9
3D1	wPt-740662*	8.5 [a]	3.58E-3	2.99	+0.196	35/248	27 of 51
4D1	Rht-D1a/b*	43.9 [a]	4.91E-3	2.78	-0.166	237(Rht-D1*b*)/48	1 of 1
6A2	wPt-3965*	55.4 [a]	6.29E-3	2.58	+0.190	24/265	3 of 14
7A1	wPt-740561*	40.6 [a]	6.92E-3	2.50	+0.167	34/256	1 of 2

3.1.3.2 Pathotype virulent on Robigus and avirulent on Solstice

Robigus is a variety commercialized in the UK between 2003 and 2011, it is susceptible at seedling stage and adult stage against most UK Pst isolate but it is believed to carry some seedling resistance and potentially an adult stage resistance based on the infection observed against limited UK isolates in historical data (see description of historical data collected in Chapter III). Hovmøller (2007) postulated the presence of resistance gene *Yr2* and *Yr32* in Robigus using seedling test against 16 Pst isolates of various origin. Solstice was commercialized in the UK between 2002 and 2012 and presented a high field resistance until 2008 when a new race named "Solstice race" overcame its resistance. The source of Solstice resistance is unknown, however all isolates virulent on Solstice were also virulent on Robigus at seedling stage (Rosemary Bayles, NIAB. Personal communication), suggesting Robigus and Solstice may share a common seedling resistance gene with Solstice carrying an additional seedling resistance which was overcome by the "Solstice" race. The Solstice pedigree (Vivant x Rialto) suggests it may have inherited *Yr32* from Vivant and *Yr6* from Rialto.

By sorting the historical data based on the virulence for Robigus and avirulence for Solstice at seedling stage, we aimed to focus on QTLs effective against the "Robigus race" and reveal the seedling resistance differentiating Robigus from Solstice.

After application of the usual MAF threshold 0.05, 1590 DArT markers were included in the association analysis of pathotype "virRob/avirSol". 14 markers were identified to be linked with the pathotype and were distributed over five MTA groups (Table VI-10) assigned to chromosomes 1B, 2B, 5B and 6A. None of those MTAs point to seedling resistance we suspected (based on pedigree) to be present in Solstice; *Yr6* is located on 7B, *Yr32* is likely to be on 2A. Additionally except for group 5B2, all MTA groups were also

detected in *de novo* adult plant test against isolate 08/21 representing the Solstice race which excludes them as potential candidates for Solstice seedling resistance. The MTA group 5B2 does not refer to a resistance in Solstice neither as Solstice allele for wPt-8604 (1) is associated with a +0.289 effect (increased susceptibility). Therefore the dataset based on virulence response against Robigus and Solstice failed to identify the seedling resistance overcome by the new "Solstice" race, for which several reasons can be put forward:

- A low frequency of the Solstice resistance gene in the subset of varieties tested,
- A low heritability of the phenotype (h^2=0.60)
- Insufficient marker coverage in the region of the pertinent resistance loci
- A high number of loci contributing to the phenotype.

Table VI-10: Groups of MTAs associated with the pathotype virulent on Robigus and virulent on Solstice at seedling stage

[a] chromosomal location from P. Bansept consensus map, [LD] chromosomal location based on LD within YR panel, [b] chromosomal location from A. Killian consensus map, [c] effect linked to allele 1 based on log (YR score+1), * P-value<0.01, ** P-value<0.001, *** P-value<0.0001, B P-value fitting the 5% Bonferroni correction threshold (0.05/1590=3.2 E-5)

Group	Major MTA	cM	p-value	r^2	Effect [c]	Alleles 1/0	No. Markers in group
1B1	wPt-2694	37.6 [b]	1.74E-3	6.36	+0.234	41/110	1 of 1
2B2	wPt-1489	53.6 [a]	6.44E-3	4.66	-0.256	18/135	2 of 4
2B4	wPt-3695	180.6 [a]	3.90E-6	12.80	-0.326	53/102	5 of 9
5B2	wPt-8604	57.2[a]	1.99E-3	6.09	+0.289	138/15	3 of 6
6A2	wPt-3965	55.4 [a]	1.21E-3	6.45	+0.329	13/141	3 of 14

3.1.4 Analysis including all historical data and comparison with analysis based on virulence criterion

The derived mean from all merged historical data covered 310 varieties in the YR panel. Within that subset 270 markers had a molecular variant at a frequency of 5% or less, leaving a total of 1541 usable markers.

Seven markers from three MTAs groups were identified; they were located on 1A, 2B and 4D (Table VI-11). Five markers from 2B were highly significant (Bonferroni threshold 5%). Only wPt-3695 was mapped in our consensus map, on 2B at 180.6 cM. The four others markers were highly correlated (r^2>0.9) to wPt-3695 suggesting they mapped to nearby loci.

Table VI-11: Significant MTAs detected with merged historical data for 310 varieties

[a] chromosomal location from P. Bansept consensus map, [LD] chromosomal location based on LD within YR panel, [b] chromosomal location from A. Killian consensus map, [c] effect linked to allele 1 based on log (YR score+1), * P-value<0.01, ** P-value<0.001, *** P-value<0.0001, B P-value fitting the 5% Bonferroni correction threshold (0.05/1541=3.2 E-5)

Group	Chr.	Highest MTA	cM	r^2	Effect [c]	Alleles 1/0
1A2	1A	wPt-665174*	11.4cM [b]	2.2	-0.14	124/184
2B4	2B	wPt-3695***B	180.6 [a]	9.3	-0.25	81/229
2B4	2B	wPt-669273***B	180.6 [a,LD]	10.0	-0.25	80/225
2B4	2B	wPt-732666***B	180.6 [a,LD]	9.3	-0.25	81/229
2B4	2B	wPt-733641***B	180.7 [a,LD]	9.1	-0.24	83/224
2B4	2B	wPt-743307***B	180.6 [a,LD]	9.3	-0.25	81/228
4D1	4D	*Rht-D1a/b***	43.9 [a]	2.3	-0.18	246(Rht-D1*b*)/59

Despite covering a larger number of varieties, the genome wide scan using the entire historical dataset, identified fewer resistance QTLs comparing to scans focused on a specific isolate or pathotype (Table VI-12), which emphasises the advantage of the virulence profile approach. By focussing on a specific virulence profile, the heritability of the trait (YR score) was improved as the number of genes determining yellow rust resistance within the YR panel line was artificially limited, therefore increasing the chance to identify specific resistance QTLs. The heritability of YR scores from the complete dataset was estimated to be 0.42, while heritability within the data subsets corresponding to one isolate or a strict pathotype varied from 0.55 to 0.89. Crossa et al. (2007) using international multi-environment trials for rust association studies acknowledged that the variation of pathogen races in different trials was likely to reduce the identification of race-specific resistances. Therefore it may suggest the three MTA groups detected on 1A, 2B and 4D using all historical data identified resistance effective against a wide range of isolates and are likely to be more durable.

Table VI-12: Summary markers traits association detected with historical data

P-value <0.00005 in dark blue, P-value between 0.0001 and 0.00005 in purple, P-value between 0.001 and 0.0001 in red, P-value between 0.01 and 0.001 in orange, na: not tested because MAF was inferior to 0.05, [a] chromosomal location from P. Bansept consensus map, [b] chromosomal location from A. Killian consensus map, [c] some markers from the group had a low allele frequency (MAF<0.05)

MTA groups and location on chromosome in cM	No. markers	Dataset analysed and avirulence associated																	YR race specific identified
		all historical data	**1990-505** avir 6 /? 9,17,32	**1993-24** avir 7, 9 /?17, 32	**1993-54** avir 32 /?7, 17	**1994-519** avir 4, 6, 7, 32	**1996-31** avir 1, 2, 7, 9, 17	**1996-502** avir 4, 7 /? 32	**1998-28** avir 32 /?7	**1998-96** avir 7	**1998-108** avir 1, 2, 7, 9	**2000-41** avir 6, 7	**2002-70** avir 6 /? 3, 4, 7, 17, 32	**2002-84** avir 9 /? 1, 2, 4, 6, 7, 17	**no7** avir 7	**no6no7** avir 6, 7	**no7no32** avir 7, 32	**no6no7no32** avir 6, 7, 32	**no4no6no7no32** avir 4, 6, 7, 32	
1A2 (11.4-14.8[b])	4	x																	
1B1(37.6[b])	1											x	x						
2AS-2NS(11.9-40[a])	17			x	x														*Yr17*
2A1(61.6-71.2[a])	5[c]		na	na	na	na	na	na	na	na	x	x						na	
2A2(-)	1					x												x	*Yr32*
2B1(84.1[b])	2				x			x	x	x					x				*Yr7*
2B2(53.5-53.6[a])	4										x	x			x				
2B4(179.5-180.6[a])	9	x	x	x	x		x		x		x	x	x	x	x	x	x	x	x	
2D1(48.7[b])	1							x											
2D2(101.2[b])	1		x								x				x				
3B1(125.2-132.5[b])	7			x	x														
3B2(210.0-210.3[a])	3																x		
3B3(272.2-281.5[a])	2														x				
3D1(5.8-8.6[a])	52								x		x		x	x	x	x			
4A1(18.4-23.4[a])	3		x				x						x	x					
4D1(43.9[a])	1	x																	
4D2(-)	1																x		*(Yr22)*
5B1(15.2[b])	2																	x	
5B2(57.2-65.6[a])	6		x	x												x		x	
5B3(94.7[b])	3[c]		na	na	na	na			na				x	x	na	na			
6A2(55.0-62.7[a])	14[c]									na		na				x			x	*(YrH46)*
6B1(72.9-83.2[a])	7[c]						x		x	na	x	na	x	x	x	x			
7A1(40.6[b])	2		x					x			x								
7B2(109-119.5[a])	4		x									x	x	x			x		x	
7B3(141.6-143.0[a])	1					x						x				x		x	x	*Yr6*

Using the isolate and pathotype approaches, we were able to identify markers associated with major resistance gene *Yr6, Yr7, Yr17* and *Yr32*, while the analysis on the complete dataset did not show significant association with these genes. Only MTA group

2B4 including five highly significant markers (wPt-669273, wPt-732666, wPt-733641, wPt-743307 and wPt-3695) was detected with the complete dataset and the isolate/pathotype approach. This QTL is likely to point to a resistance with major effect effective against a wide range of UK isolates. WPt-3695 was mapped on 2BL (180.6cM) on our consensus map. As 2BL is rich in yellow rust resistance genes (*Yr5, YrSP, Yr7, YrV23, Yr43, Yr44, Yr S2199*) and QTLs, it is difficult to narrow MTA group 2B4 to any specific known resistance gene or QTL; however 2B4 is not likely to be *Yr7* as MTA was detected with 1990-505, an isolate virulent on *Yr7*. To investigate further 2B4 QTL, we developed a STS marker for wPt-3695, allowing us to locate and test the effect of the marker in AxC mapping population (see section 3.3 for details).

Besides the major resistance genes *Yr6, Yr7, Yr17, Yr32,* and QTL 2B4 with large effect, the GWA scans on selective subset of historical data, identified 18 additional potential resistance QTLs, spread across the genome which suggests a large diversity of resistance loci within the YR panel. Chromosome groups 2 and 3 had the highest number of MTA groups (6 and 4) while group 1 and 6 only presented two MTA group each. Less MTA groups were detected in Genome D and A comparing to genome B which is partly due to the difference of marker coverage between genome (Genome D presenting less independent polymorphic DArT markers comparing to Genomes A and B).

Many MTA groups point to chromosome arms where *Yr* genes have been previously reported 1BS (*Yr 9,10,15,24,26,Alp,H52,Cl142*), 2AS (*Yr 8, 17*), 2BS (Yr 2*7, 31, 41/CN19, P81*), 2BL (*Yr 5, 7, SP, V23, 43, 44, S199*), 2DS (*Yr 8, CK*), 2DL (*Yr 16, 37*), 4DS (*Yr 28*), 5BL (*Yr Exp2*), 6BS (*Yr 35,36*) and 7BL (*Yr 6, C591*) (see *Yr* genes Tables I.4 and I.5 in Chapter I for references). Other MTAs point to chromosome arms without mapped Yr genes 1AS, 3BL, 3DL, 4AS, 5BS, 6AS, 7AS and could represent novel resistance loci. However they could also represent *Yr* genes not assigned to a specific arm (YrDa1 on 1A, YrS and YrSte2 on 3B, Yr HVII, Min, ND on 4A, Yr19 and YrDru on 5B, YrDru2, YrD, YrH46 on 6A) or one of many WYR resistance QTLs identified (see list in Appendix 2).

As limited *Yr* genes and resistance QTLs have been mapped using DArT markers, we showed with the examples of *Yr7* and *Yr32,* it was difficult to compare the MTAs detected from this study with other resistance studies. Although our consensus has proven to be useful to compare the location of DArT markers with others markers (e.g. SSRs) previously reported. Only resistances known to be present within the YR panel can be identified positively. To understand the role of the region identified in the genome and reveal their true linked with known QTLs and *Yr* genes, each MTA will need to be investigated further.

3.2 GWA scans on *de novo* data and QTL analysis

Responding to the deployment of new resistance genes in wheat varieties, Pst has shown its great potential for evolution by successively acquiring new virulence factors over the past decades in the UK (see Appendix 5 for evolution of virulence factors in the UK). Recently, a new race named the "Solstice" race emerged and overcame many previously resistant UK wheat varieties. Therefore a new question arose: are the resistances identified with historical data efficient against current Pst isolates? Or do they represent race-specific resistance overcome by new Pst isolates. To answer those questions, the YR panel was evaluated against recent Pst isolates covering a wide range of virulence at seedling stage and at adult plant stage. In parallel of the seedling tests on the YR panel, the doubled haploid population Avalon x Cadenza was evaluated at seedling stage against the same Pst isolates to help the validation of some MTAs.

3.2.1 "Brock isolate" seedling tests

3.2.1.1 GWA scan on the complete panel

308 YR panel lines were evaluated against Pst isolate 03/07, the genome wide association scan with infection type observed on revealed 126 markers associated with seedling resistance. The markers were distributed over 9 MTA groups located on chromosomes 1A, 1B, 2B, 3B, 6B and 7B (Table VI-13).

Table VI-13: MTAs detected in the YR panel at seedling stage against Pst isolate 03/7

[a] chromosomal location from P. Bansept consensus map, [b] chromosomal location from A. Killian consensus map, *P-value<0.01, **P-value<0.001, ***P-value<0.0001

MTA group	Highest MTA	cM	r^2	Effect	Alleles 1/0	No. Markers in group
1A1	wPt-4676**	16.6 [a]	3.9	-1.78	22/286	5 of 6
1BS-1RS	wPt-8930***B	9.2 [b]	11.4	+2.86	203/99	102 of 102
1B2	wPt-3465**	135.6 [a]	3.7	+1.30	108/191	1 of 1
2B1	wPt-2293*	84 [b]	2.6	-0.97	129/175	1 of 2
3B1	wPt-5072*	131.5 [b]	2.3	+1.09	112/194	2 of 7
6B2	wPt-3207**	109 [b]	3.7	-1.70	283/23	4 of 4
7B1	wPt-743645**	3.0 [a]	4.2	-1.80	275/22	2 of 2
7B3	wPt-0752**	141.6 [a]	3.7	+1.47	192/105	1 of 2

102 of the markers identified on 1B were correlated (r^2>0.5) to the 1BL.1RS marker from de Froidmont (1998) (see Appendix 15 for complete list), therefore identifying the resistance gene *Yr9*. The markers linked with 1BL.1RS translocation (MTA group 1BS-1RS)

explained between 4.3 to 11.4 % of the phenotypic variation. wPt-8930 presented the highest significance level (P-value =8.3 10^{-10}), but was not a perfect diagnostic marker for *Yr9* as allele 0, associated with lower infection type, was also present in Ochre and Boston which carry resistance gene *Yr15* on 1BS, a major resistance gene effective against Pst 03/7. 27 markers were perfect diagnostic markers for the presence of the rye chromosome arm (1RS) within the YR panel but presented different levels of missing genotypes which influenced their level of significance, among them are rPt-7959 (P-value=4.4 10^{-8}, 2 genotypes missing), tPt-8754 (P-value=4.3 10^{-8}, 4 genotypes missing), tPt-2550 (P-value=6.4 10^{-8}, 6 genotypes missing) and wPt-9883 (P-value=4.4 10^{-8}, 1 genotype missing). Allele 1 from rPt-7959, tPt-8754, and tPt-2550 indicates the presence of the rye chromosome arm, while allele 1 for wPt-9883 indicates the absence of the rye translocation. By combining the genotype for DArT markers rPt-7959 and wPt-9883, we are able to create a virtual co-dominant marker (Figure VI-7), which identified either the presence of the rye chromosome arm 1RS or the presence of the wheat chromosome arm 1BS.

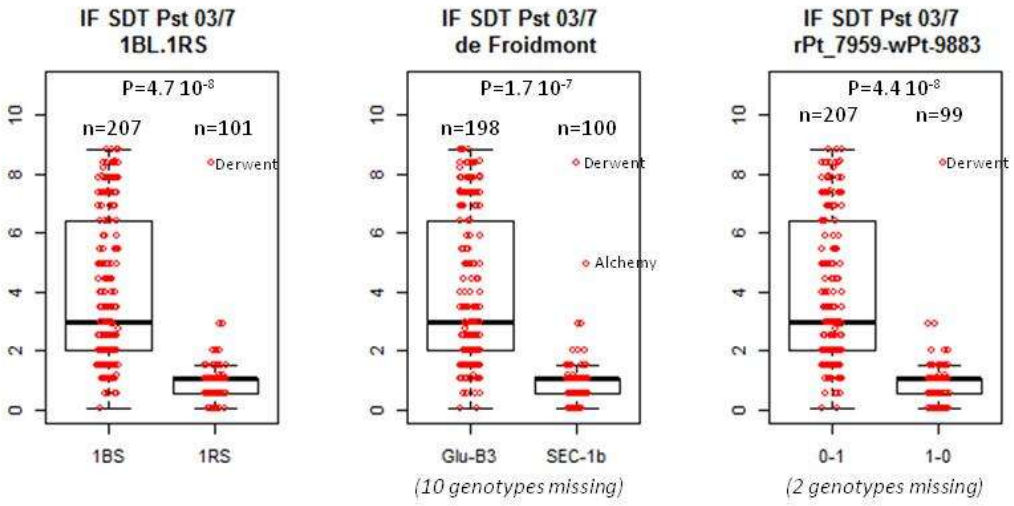

Figure VI-7: Combined box plots and dot plots of infection type observed on the YR panel lines with Pst isolate 03/7 for MTA group 1BS-1RS

The first box plot "1BL.1RS" sorts the lines with the wheat chromosome arm 1BS from the line with the rye chromosome arm 1RS based on the genotype of four DArT markers from MTA group 1BS-1RS: rPt-7959, tPt-8754, tPt-2550 and wPt-9883. "de Froidmont" corresponds to the genotype obtained from the PCR assay developed by de Froidmont (1998). rPt-7959_wPt-9883 combined the genotype for DArT markers rPt-7959 and wPt-9883 to create a co dominant marker. 0: hybridisation to DArT clone not detected; 1: positive detection of hybridisation to DArT clone; p: P-value from MLM association test; n: number of genotypes in each allelic class. The red dots are adjusted infection type for each line. The high infection type in Derwent is likely due to an error in the seedling test, while Alchemy has been genotyped erroneously with the translocation 1BL.1RS using " de Froidmont" assay.

3.2.1.2 GWA scan on lines without 1BL.1RS translocation

As the presence of the rye chromosome arm 1RS and the strong effect of *Yr9* on infection type may prevent the detection of other resistance QTLs on 1BS effective against Pst 03/7 such as *Yr15*, *Yr10*, *Yr24*, an additional scan was done with the 207 lines without the rye translocation. 1518 DArT markers with a MAF 0.05 were included in this association analysis. 12 markers were found significant and assigned to chromosomes 2B, 3B and 7B (Table VI-14). No MTA was found on 1B which suggests the resistance genes *Yr10*, *Yr15* and *Yr24* located on 1BS are either not present in the YR panel lines or are too rare to be detected. It is the case of *Yr15* which is present in Boston and Ochre but no other lines are known to have *Yr15*.

Table VI-14: Comparison of MTAs detected in the complete YR panel and in the subset of lines without the 1BL.1RS translocation against Pst isolate 03/7 at seedling stage

*P-value<0.01, **P-value<0.001, ***P-value<0.0001

		P- value for GWA scan	
Highest MTA	Group	309 lines	207 lines without 1BL.1RS
wPt-4676	1A1	$4.0\ 10^{-4}$**	*0.014*
wPt-8930	1BS-1RS	$8.3\ 10^{-10}$ ***	nc
wPt-3465	1B2	$6.2\ 10^{-4}$ **	*0.12*
wPt-2293	2B1	$4.1\ 10^{-3}$ *	$1.9\ 10^{-4}$ **
wPt-0950	2B3	*0.014*	$4.3\ 10^{-3}$ *
wPt-5072	3B1	$6.2\ 10^{-3}$ *	$1.0\ 10^{-3}$ *
wPt-3207	6B2	$5.7\ 10^{-4}$ **	$3.0\ 10^{-3}$ *
wPt-743645	7B1	$3.0\ 10^{-4}$ **	$1.3\ 10^{-3}$ *
wPt-4814	7B2	*0.024*	$1.6\ 10^{-3}$ *
wPt-0752	7B3	$6.9\ 10^{-4}$ **	$7.2\ 10^{-6}$ ***

Besides markers linked to *Yr9*, the GWA scan with seedling test with 03/7 detected a highly significant marker on 7BL (wPt-0752) which is likely to be *Yr6* as 03/7 is avirulent on *Yr6*. The same marker has been identified using historical data with pathotypes and isolates avirulent on *Yr6*. However wPt-0752 does not distinguish all the cultivars with *Yr6* (see section 3.1.2.2). Despite the high heritability of the infection type (h^2=0.97) and the large number of varieties tested (309 and 207), we did not succeed to identify a usable diagnostic marker, most likely because of the relatively low DArT marker density on 7BL.

3.2.1.3 Validation of MTA group 7B3 in mapping population Avalon x Cadenza

As the precise map location of *Yr6* is not known, we evaluated the doubled haploid population Avalon x Cadenza against Pst isolate 03/7 to map *Yr6*. Cadenza is believed to

carry seedling resistance *Yr6* and *Yr7* (Pathan et al., 2008). The assignment of *Yr6* to a genetic map including DArT markers will allow us to validate the association found on 7BL as being *Yr6.*

The distribution for infection type scored on the 201 AxC doubled haploid lines was bimodal (Figure VI-8). All Cadenza seedlings were resistant (IF= 1 or 2). Avalon seedlings were susceptible (IF between 6 and 8). ANOVA showed a significant genotype effect and no significant block effect (ANOVA outputs are available in Appendix16). Consequently adjusted means were obtained and used for QTL detection. A high heritability within the test was observed (h^2=0.98). Chi-squared analysis showed that resistant and susceptible lines segregated in a 1:1 ratio supporting the hypothesis of a single gene (p=0.40) (see Chi-squared goodness-of-fit results in Appendix 16).

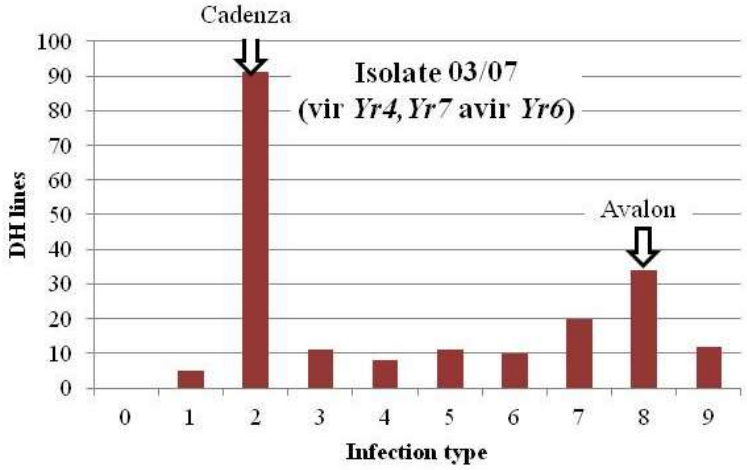

Figure VI-8: Distribution of infection type at seedling stage in Avalon x Cadenza DH lines inoculated with Pst isolate 03/7

A total of 611 markers were included in the AxC genetic map used to detect yellow rust resistance QTLs. The map included 26 linkage groups representing the 21 wheat chromosomes. Using an interval mapping approach, a major QTL from Cadenza was detected on 7BL and a minor QTL from Avalon was detected on 2B (Table VI-15). No interaction were observed between the two QTLs. QTL on 7BL explained 60.6 % of the phenotypic variation and is presumed to be *Yr6,* a major seedling resistance gene effective against Pst isolate 03/7. This QTL designates a similar chromosomal location to MTA groups 7B2 and 7B3 (wPt-0752, wPt-4814) identified by association mapping (Figure VI-9). Thus we confirmed MTA wPt-0752 and wPt-4814 identified *Yr6.*

On the other hand, the minor QTL on 2BL from Avalon was not detected by association mapping, probably because of its small effect.

Table VI-15: Summary of resistance QTL in AxC against yellow rust Pst 03/7

QTL were identified by interval mapping, the threshold of detection was determined using a 1000 permutation test (LOD threshold 5% =3.3).[a] interval of confidence for QTL position using Bayesian estimate as implemented in R/qtl

Chr.	Origin of resistance allele	95% confidence interval [a]	Peak position (cM)	Nearest marker	LOD	R^2	Gene associated
2BL	Avalon	XBS0003585-wPt-2397 (134.8-158.2 cM)	148.0	XBS00010361	4.2	8.7	unknown
7BL	Cadenza	XBS00010660-gwm577 (39.1-49.2 cM)	45.0	gwm577	54.3	60.6	*Yr6*

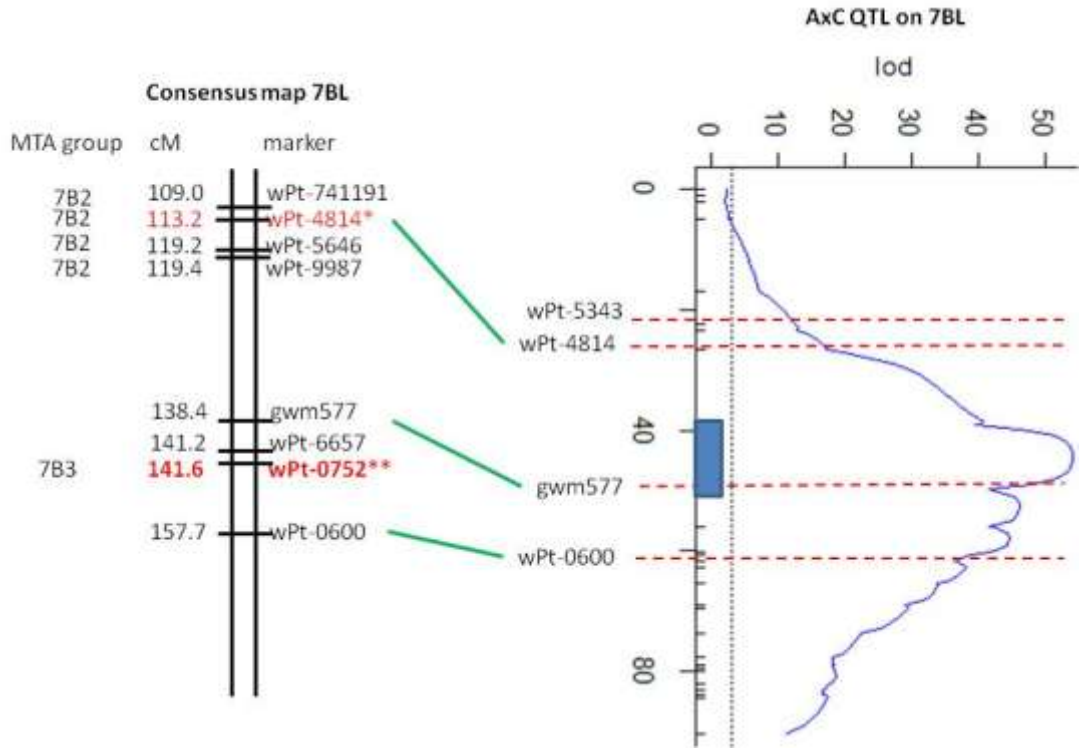

Figure VI-9: Comparison of 7BL QTL in AxC population and MTAs in YR panel identified at seedling stage against Pst isolate 03/7

The dashed line in AxC QTL indicated threshold of detection at 5% error, the blue rectangle shows the 90% interval of confidence for the QTL. The marker highlighted in red were detected by GWA mapping.

3.2.2 "Solstice isolate" seedling tests

3.2.2.1 GWA scans on YR panel

317 YR panel lines were evaluated at seedling stage against Pst isolate 08/21. The genome wide association scan with infection type revealed 17 significant MTAs. The MTA were located on chromosomes 2A (group 2A1), 2B (groups 2B3 and 2B4), 3B (group 3B3) and 6A (groups 6A1 and 6A2) (Table VI-16). MTA group 2B4 presented the highest level of

significance and could explained up to 10% of the phenotypic variation, it includes five markers mapped on 2B, the same already highlighted with the analysis of historical data.

Table VI-16: Groups of MTAs detected at seedling stage against Pst isolate 08/21

[a] chromosomal location from P. Bansept consensus map, [b] chromosomal location from A. Killian consensus map. * P-value<0.01, ** P-value<0.001, *** P-value<0.0001, BP-value fitting the 5% Bonferroni threshold

MTA group	Major MTA	cM	r^2	Effect	Alleles 1/0	No. Markers in group
2A1	wPt-1657*	71.6 [a]	2.9	-1.03	263/53	2 of 5
2B3	wPt-9350*	123.7 [a]	3.2	+0.98	177/138	2 of 3
2B4	wPt-3695***B	180.6 [a]	8.7	-1.59	86/231	5 of 9
3B3	wPt-10537*	272.2 [a]	2.9	+0.83	116/193	2 of 2
6A1	wPt-0228*	17.3 [a]	2.9	+0.99	184/129	3 of 3
6A2	wPt-664552*	62.7 [a]	2.2	-0.90	259/51	3 of 14

Considering the wide virulence profile of the "Solstice" isolate 08/21 (see extended virulence profile on Chapter IV section 3.1) and its avirulence for *Yr 5,7,8,10,15,24,SP*, the GWA scans on infection type should allow us to detect seedling resistance *Yr7* on 2BL known to be present in 11 lines of the YR panel or more. Two MTA groups were found on 2BL (group 2B3 and 2B4). On our consensus map, wPt-9350 (group 2B3) mapped to 123.7 cM and is between 11.1 and 14.2 cM away from SSR markers wmc175, gwm501, cfd73 and gwm120 linked to resistance gene *Yr7* in cv Camp-Remy (Mallard et al., 2005) and cv Apache (Paillard et al., 2012)(Figure VI-12). Group 2B4 mapped 68.2 cM away from the SSR markers wmc175 and is likely to represent a different seedling resistance gene.

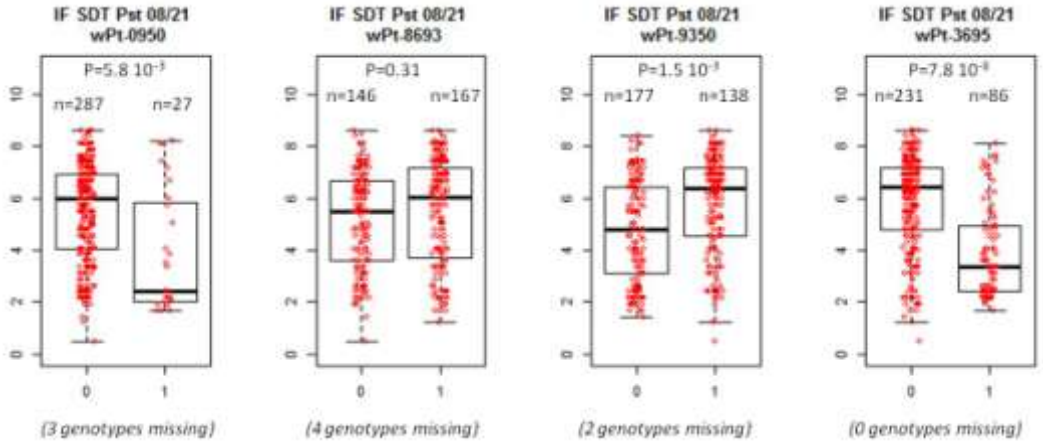

Figure VI-10: Combined box plots and dot plots of infection types observed at seedling stage in YR panel against 08/21 depending of genotype for the MTA groups 2B3 (wPt-0950, wPt-8693, wPt-9350) and 2B4 (wPt-3695).

3.2.2.2 Validation of MTA group 2B3 in mapping population Avalon X Cadenza

A direct validation of the MTAs on 2B was difficult based on published studies due to the lack of DArT markers in published genetic maps. To resolve the problem, we intended to map *Yr7* in Avalon x Cadenza population as its genetic linkage map incorporating DArT, SSR and SNP markers. WPt-9350 and wPt-0950, associated with MTA group 2B3, were mapped on the AxC genetic map.

The AxC doubled haploid population was evaluated at seedling stage against Pst isolate 08/21. The distribution for infection type was bimodal (Figure VI-11). All Cadenza seedlings were resistant (IF=2 or 3). Avalon seedlings were susceptible (IF between 7 and 9). ANOVA showed a significant genotype effect and a significant block effect (ANOVA outputs are available in Appendix 16). Consequently adjusted means were obtained and used for QTL detection. Chi-squared analysis showed that resistant and susceptible lines segregated at a 1:1 a ratio supporting the hypothesis of a single gene (p=0.83) (see Chi-squared goodness-of-fit results in Appendix 16).

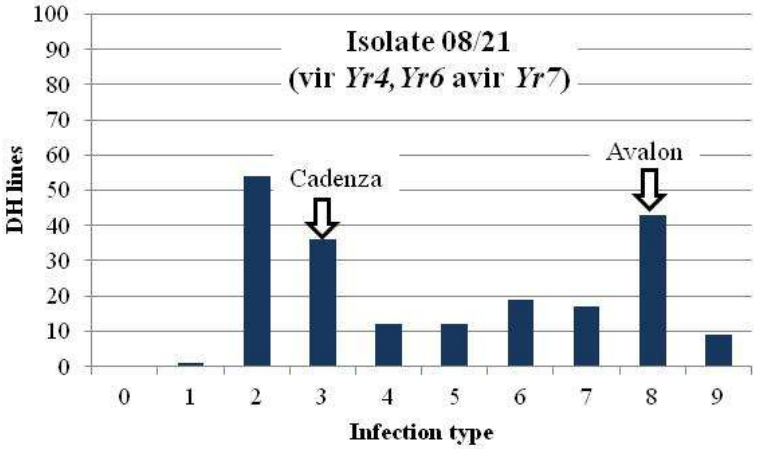

Figure VI-11: Distribution of infection type at seedling stage in Avalon x Cadenza DH lines inoculated with Pst isolate 08/21

Using an interval mapping approach, a major QTL was detected on 2BL and a minor QTL was also detected on 4B (Table VI-15), both resistance QTL were inherited from Cadenza. However, we suspect the (minor) QTL on 4B is an artefact caused by the presence of a high level of linkage disequilibrium between markers from QTL 2BL and 4B.

Table VI-17: Summary of resistance QTL in AxC against yellow rust Pst 08/21

QTL were identified by interval mapping, the threshold of detection was determined using a 1000 permutations test (LOD threshold 5% =3.1).[a] interval of confidence for QTL position using Bayesian estimate as implemented in R/qtl. [b] QTL 4B is likely to be an artefact from QTL 2BL due to high linkage disequilibrium between markers from 2BL and markers from 4B

Chr.	Origin of resistance allele	95% confidence interval [a]	Peak position (cM)	Nearest marker	LOD	R^2	Gene associated
2BL	Cadenza	Xwmc175-XBS00010012 (118.2-119.8cM)	118.7	XBS00009989	66.0	75.7	*Yr7*
4B [b]	Cadenza	XBS0009480-XBS00010409 (0-28.6 cM)	20.6	XBS00009915	7.32	16.2	None

The QTL on 2BL explained 75.7 % of the phenotypic variation and is presumed to be *Yr7*. This QTL designates an identical chromosomal location to MTA group 2B3 (wPt-0950, wPt-9350) identified by association mapping with the *de novo* seedling test with 08/21 (Figure VI-12). In addition, based on A. Killian consensus map, MTA group 2B1 (wpt-2293, wPt-732882) identified by association mapping with historical data is likely to located between marker wPt-0950, wPt-9350. Thus, as suspected MTA groups 2B1 and 2B3 corresponds to the *Yr7* locus.

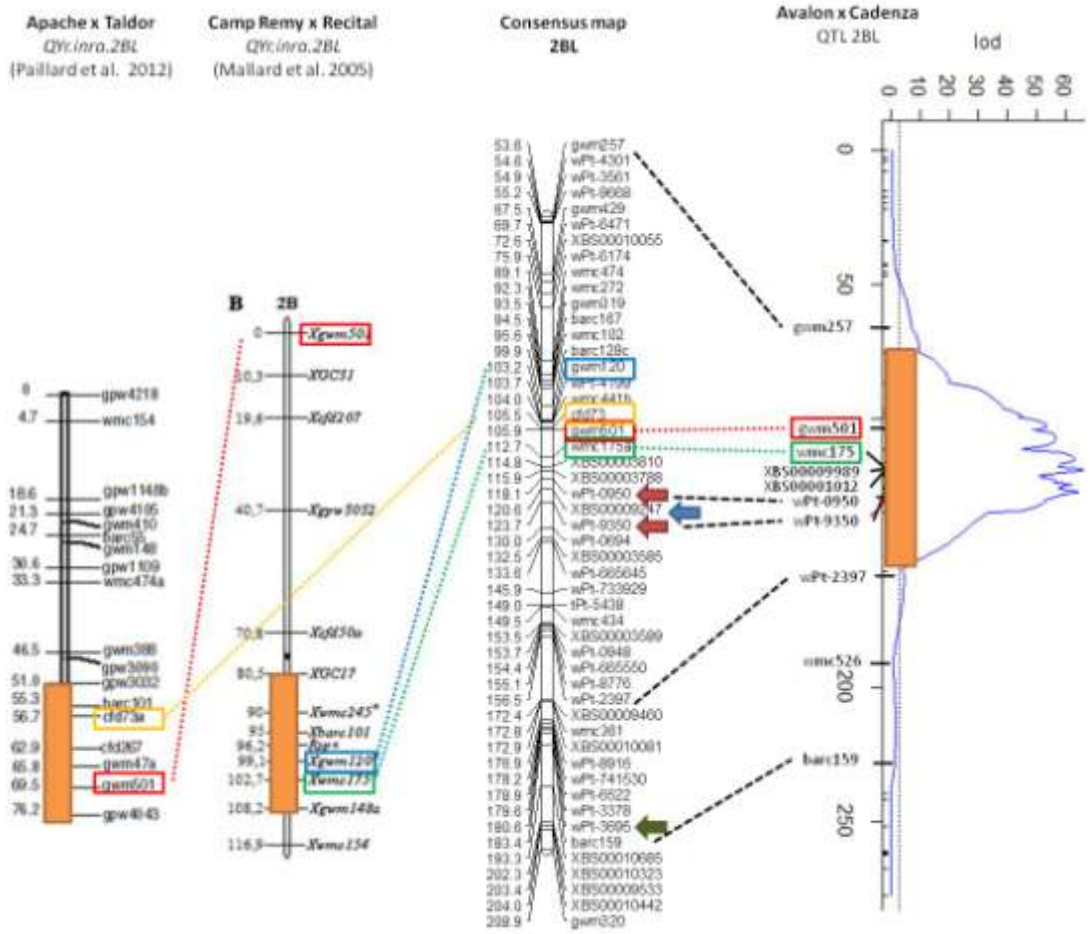

Figure VI-12: Comparative location of QTL*s* identified in bi-parental populations Apache x Taldor, Camp Remy x Recital and Avalon x Cadenza with GWA mapping hits on 2BL

The QTL positions are indicated by orange rectangles and significant MTAs are indicated by coloured arrows on the consensus map, blue for 2B1, red for 2B3 and dark green for 2B4. No marker from MTA group 2B1 were included in our consensus map however based on A. Killian map (Triticarte), 2B1 should be located between wPt-0950 and wPt-9350 from group 2B3. MTA group 2B2 is not represented as it points to 2BS.
Avalon x Cadenza QTL was observed with infection type against Pst 08/21. The dashed line in AXC QTL indicated threshold of detection at 5% error.

Looking at the allele distribution of the markers in group 2B3 (Figure VI-10), despite presenting a lower P-value, wPt-0950 seems to be the closest to a diagnostic marker for *Yr7*. The resistance allele "1" was found on 27 YR panel lines, among which 11 are known to carry *Yr7* (Brock, Cadenza, Camp Remy, Cordiale, Ekla, Thatcher, Spark, Tonic, Tommy and Vault) and nine lines are not likely to have *Yr7* given their intermediate to susceptible response against 08/21 isolate (Vuka, AC Barrie, Prophet, Chicago, Hurley, Copain , Apollo, Bouquet, Hyperion). The remaining varieties Cyber, Fastnet, Orestis, Pagan, Soleil, Vector and Virtuose were resistant against 08/21 isolate and may carry *Yr7*. Except for Copain, the lines in the pedigree of Thatcher and Cadenza carrying the positive allele were resistant against 08/21 and 03/7 (lines outlined in violet in Figure VI-13) or

resistant against 08/21 and susceptible against 03/7 (lines outlined in red in Figure VI-13). WPt-0950 allele 1 was transmitted from Thatcher (*Yr7*) to its descendants carrying *Yr7*, while Cadenza inherited the allele from Tonic (*Yr7*) and transfered it to Cordiale.

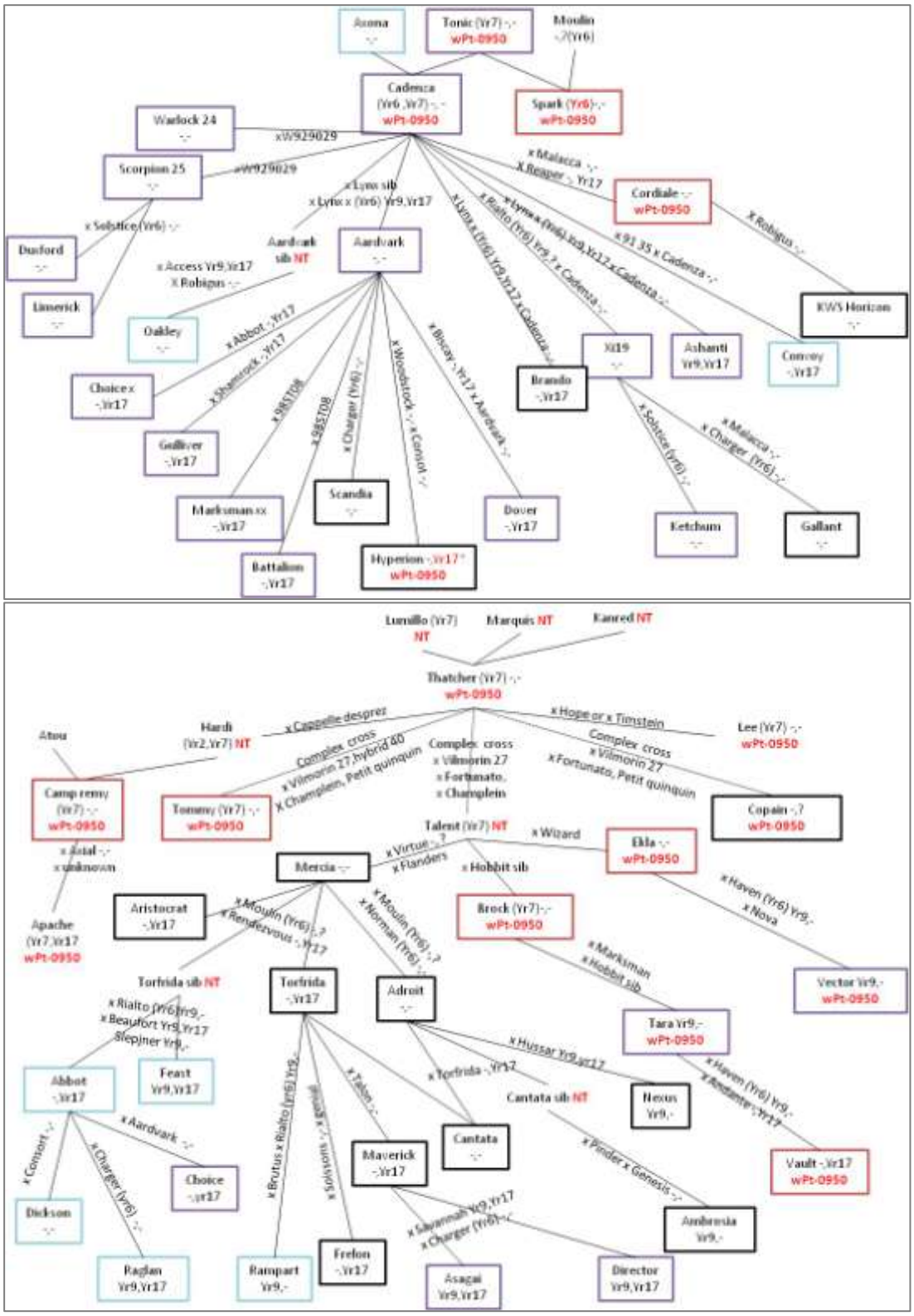

Figure VI-13: Pedigree diagrams of Thatcher and Cadenza descendants including their genotype for wPt-0950

wPt-0950 indicates the presence of allele 1 in the line; NT indicates the genotype information for wPt-0950 was not available. The colour code related to infection type response against 08/21 and 03/21 isolates as already described in Chapter IV Figure IV-27. * Hyperion genotype does not fit the pedigree information for *Yr17* and wPt-0950, which suggests a pedigree error or a genotype error.

3.2.3 Adult plant test

The YR panel was evaluated against selected Pst isolates in the field in 2010 and 2011. The 2010 trial was inoculated with 08/21 isolate representing the "Solstice" race, while the 2011 trial was inoculated with a mix of isolates representing Solstice (08/21), Brock (03/7) and Timber (08/501) races.

When analysing all the AP scores (AUDPCr, Severity and Host response), 86 markers corresponding to 23 MTA groups were associated with at least one yellow rust scores in the field(Table VI-18). Greater associations (P-value<0.001) were observed on chromosomes 1A, 2A, 2B, 3B, 3D, 5D, 6A and 6B. The wide spread of the MTAs over the genome highlighted a great diversity of genes contributing to the resistance response. Considering the gaps in genome coverage, we would expect even a greater number of loci to participate to the yellow rust response.

Several MTA groups were already detected at seedling stage against 08/21 and/or 03/7, they represent race specific, seedling resistances: 1A1, 1BS-1RS, 2A1, 2A3, 2B3 (*Yr7*), 2B4, 6A1, 6A2 and 6B2 (see Table VI-19 for visual help).

Five highly correlated markers assigned to 2B (group 2B4) were consistently associated to rust resistance with all scores, they are identical to the markers point out in seedling test with 08/21, and in historical data. Therefore they may represent a QTL effective at all stage and efficient against a wide range of isolates.

When comparing the 2010 and 2011 field trials, common MTAs were found in chromosomes 1A, 2B, 3A, 4A, 5A, 5B and 6A highlighting potential QTLs efficient against the three Pst isolates tested. Some MTAS in chromosomes 1A, 2A, 3D, 4D, 5D, 6B and 7A were specific of field trial 2010 inoculated with Solstice isolate 08/21 and may represent resistances overcame by isolates 08/501 and 03/07, alternatively those resistance may still be effective in 2011 but not detected. Other MTAs in chromosomes 1B, 3B, 4A, 4D, 5B, 6B were specific of field trial 2011 inoculated with a combination of Pst isolates (08/21, 03/07, 08/501) and represent QTLs effective against the three Pst isolates.

Table VI-18: Groups of MTAs detected for yellow rust resistance scores in the field against current Pst isolates

[a] chromosomal location from P. Bansept consensus map, [b] chromosomal location from A. Killian consensus map, [c] number of scores for which the MTA was detected in 2010 and 2011 ,* P-value<0.01, ** P-value<0.001, *** P-value<0.0001.

		Major MTA				
MTA group	Detection 2010/2011[c]	Marker	Score	cM	r^2	Allele distribution 1/0
1A1	6/0	wPt-4676***B	Sevmid2010	16.5[a]	5.8	20/273
1A2	4/0	wPt-665174**	Sevend2010	11.4[b]	4.5	124/168
1A3	3/2	wPt-666607***	AUDPCr2010	67.5[a]	5.4	234/52
1BSor1BS	0/2	wPt-5740*	HostRend2011	93.3[a]	2.7	196/96
1B1	1/0	wPt-2694*	HostRmid2010	37.6[b]	2.5	66/221
2A1	2/0	wPt-1657**	Sevstart2010	71.2[a]	3.8	243/48
2B2	3/3	wPt-1489*	HostRmid2011	53.6 a	2.8	35/269
2B3	2/3	wPt-0950***B	Sevend2010	118.1[a]	6.2	27/263
2B4	7/6	wPt-3695***B	HostRmid2011	180.6[a]	12.7	82/226
3A1	*2/1*	*wPt-5476**	*HostRend2011*	*196.8*[a]	*2.6*	*289/1*
3B2	0/4	wPt-6785**	Sevmid2011	210.3[a]	2.6	117/178
3D1	4/0	wPt-1336**	Sevmid2010	8.5[a]	3.4	250/43
4A2	*1/2*	*tPt-9400**	*Sevmid2011*	*85.2*[a]	*2.7*	*282/24*
4D1	1/0	Rht-D1a/b*	HostRstart2010	43.9[a]	2.6	225b/64a
5A4	*2/3*	*wPt-1165**	*Sevend2011*	*25.5*[a]	*3.1*	*265/37*
5B1	0/1	wPt-0708*	AUDPCr2011	15.2[b]	2.9	221/81
5B4	1/3	wPt-1548*	*Sevend2010*	125.3[a]	2.7	20/272
5D	2/0	wPt-732418**	HostMid2010	13.2[a]	3.9	226/67
6A1	3/2	wPt-0228**	AUDPCr2010	17.3[a]	4.5	169/120
6A2	3/2	wPt-664552*	Sevstart2010	62.7[a]	2.9	241/44
6B1	7/0	wPt-4893*	HostRend2010	72.8[a]	3.3	236/48
6B2	0/1	wPt-3207**	HostRmid2011	108.7[b]	4.1	283/23
7A1	1/0	wPt-740561*	Sevstart2010	40.6[a]	2.9	34/259

Figure VI-14 illustrated graphically the results from the GWA scans with AUDPCr, severity scores and HostR scores. Many MTAs for HostR and severity were similar within the same trial. Likewise, MTAs for AUDPCr were matching MTAS for severity scores or HostR within the same trials, which was expected given the correlation observed between scores (see Chapter IV, sections 3.3.2.4 and 3.3.3.4). Despite targeting restricted components of rust resistance, HostR rarely provided higher level of significance compared to severity scores and AUDPCr, only associations from group 2B4, 5D and 6B1 were more significant.

The Figure VI-14 also illustrates the lack of marker coverage in several genomic regions (2A, 5D, 6D).

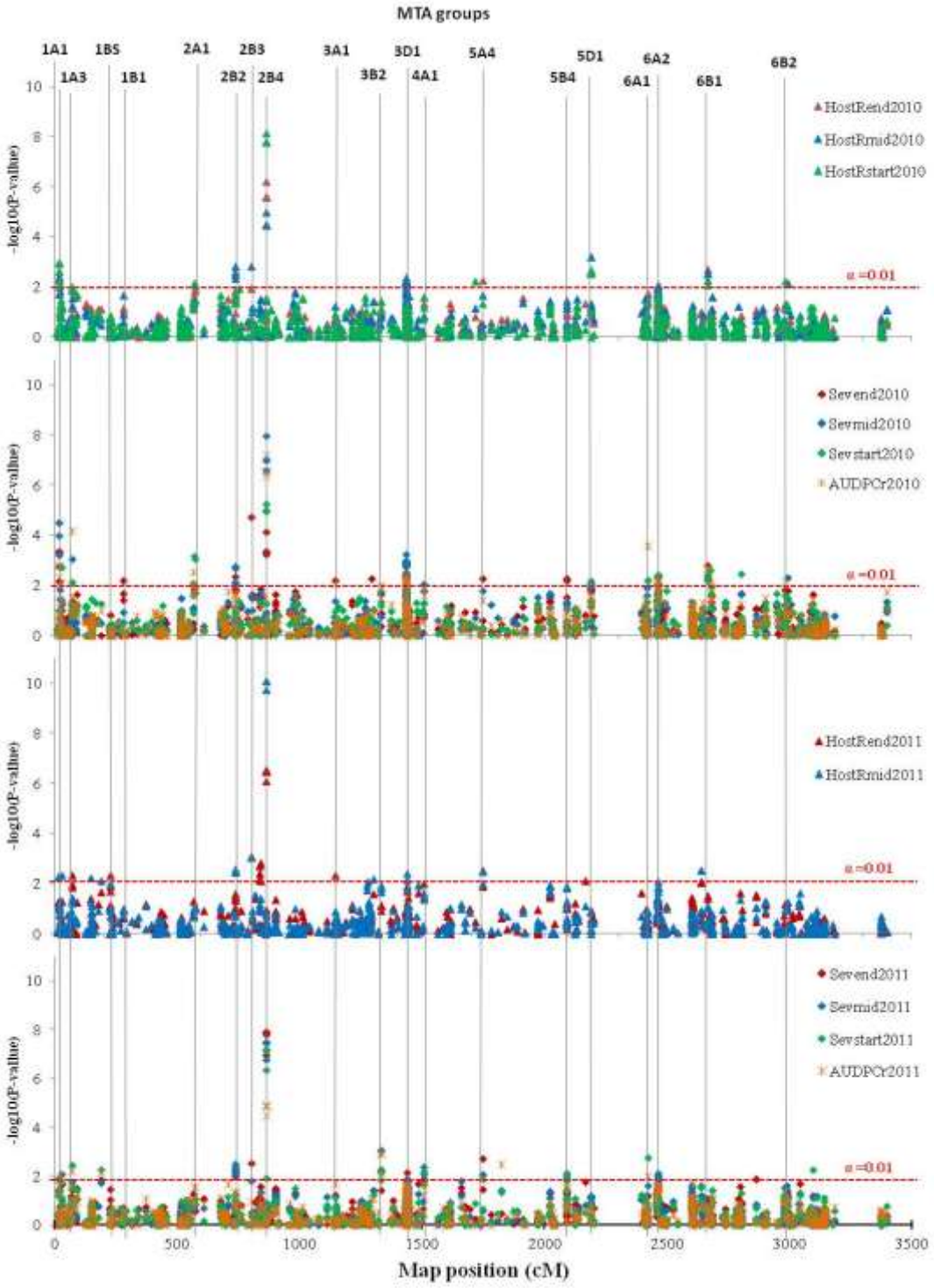

Figure VI-14: Manhattan plots for genome association mapping scans on AUDPCr, severity and host response scores from *de novo* field evaluations

The map position is based on our consensus map following the chromosomes order : 1A (position 9 to 145), 1B (150 to 369), 1D (370 to 513), 2A(514 to 690), 2B (691 to 862), 2D(863 to 987), 3A(988 to 1144), 3B(1145 to 1430), 3D(1431 to 1434), 4A (1435 to 1595), 4B (1596 to 1673), 4D (1674 to 1745), 5A(1747 to1970), 5B (1971 to 2186), 5D (2178 to 2408), 6A (2401 to 2601), 6B (2602 to 2734), 6D(2735 to 2771), 7A (2772 to 2991), 7B (2992 to 3130), 7D (3131 to 3420).

To identify MTAs associated specifically with APR against Solstice isolate 08/21, a second GWA scan was run with 2010 field trial scores including only 174 lines which showed an intermediate to susceptible response at seedling stage against 08/21 (infection type > 5). This additional scan identified markers in MTA groups 2A1, 2B4, 3A1, 6A1, 6B1 and 7A1 and suggests those groups are linked to resistance genes expressed specifically at adult plant stage.

3.3 Toward the identification of MTA group 2B4

Group 2B4 includes the five most significant markers (wPt-3695, wPt-733641, wPt-7326666, wPt-6692273, and wPt-743307) in GWA scans with historical data, *de novo* seedling test against 08/21 as well as *de novo* APT. Only wPt-3695 was included in our consensus map (position 180.6 on 2BL) based on UC1110 x PI610750 genetic map from Lowe et al. (2011b), the other markers were in high LD with wPt-3695.

To confirm the map location and identified the resistance underlying MTA group 2B4, we designed a PCR marker based on the clone sequence of DArT marker wPt-3695 available from Triticarte. The clone sequences for the other markers of the group were not available. Using the sequence, we design specific primers and obtained a dominant marker matching the DArT genotype for 99% of the lines tested.

As Avalon and Cadenza carried opposite alleles for wPt-3695, and Cadenza is known for its durable yellow rust resistance, we used the AxC population to map the STSwPt-3695 marker and tested if it was linked to a seedling resistance in Cadenza using AxC seedling test against 08/21.

195 AxC lines and the parental lines were genotyped with the adapted PCR marker STSwPt-3695. Cadenza amplified a fragment of 457bp (allele 1) while Avalon did not amplify the fragment (allele 0). 115 AxC lines amplified the 457bp fragment. The marker segregation was slightly distorted within the population (p=0.012) and mapped to a linkage group loosely linked to the long arm of 2A. The linkage group includes markers with significant segregation distortion and marker known to map to the long arm of several homeologues from the group 2 e.g. gwm382, gwm526, wmc181 (see linkage map in Appendix 17). Therefore, we suspected markers from the group 2B4 to map to multiple loci of the chromosomal group 2. Looking at A. Killian consensus map, two markers of the group, wPt-743307 and wPt-732666, also mapped to multiple locations 123.3cM on 2B and 101.2cM on 2D. The three other markers, wPt-3695, wPt-733641, and wPt-6692273, mapped on 2B position 129.9cM.

No significant QTL was detected in AxC with marker STSwPt-3695 (LOD=0.12) at seedling stage against Pst 08/21. Thus wPt-3695 was not linked to a resistance in Cadenza. Several reasons can explain this result:

- A recombination between the resistance gene identified by association mapping and marker wPt-3695 may have occur in Cadenza ancestors, so the link between the resistance gene and the marker was lost in Cadenza. Both parental lines Axona and Tonic carry also the wPt-3695 allele 1 associated to resistance.
- As wPt-3695 may be present in several homeologues of the group 2, the positive effect associated with wPt-3695 (group 2B4) in association genetic study may not be linked with 2A locus but may result from loci located in chromosome 2B and/or 2D.
- Alternatively, MTA with wPt-3695 may be a false discovery. Platt et al. (2010) emphasise through simple simulations, the possibility to observe positively misleading associations when studying a trait due to multiple factors using a single locus model that assume unlinked non causal markers are not correlated with the causal factor. In the present study, we applied a corrective model which should reduce the false discovery due to correlation between causal factor and unlinked causal factor. However, our model looks at each marker individually without taking into account the presence of multiple factors (potentially correlated) and their interactions.

Nevertheless, the investigation of pedigree gives some arguments to support a real association between wPt-3695 with a yellow rust resistance gene. Except for lines in Cadenza pedigree and other rare lines, the origin of wPt-3695 can be traced to Carstens V and most of the lines carrying allele 1 present an intermediate to resistance response at seedling stage against 08/21 and a high resistance at adult stage. The pedigree diagrams (Figure VI-15) showed wPt-3695 positive allele was transmitted:

- to Apostle and its descendant via Alcedo,
- to Flame and Claire and their descendants via Caribo and Griffin,
- to Parade and its descendant via Caribo and Granta
- to Arminda and its descendants via Carstens 864.

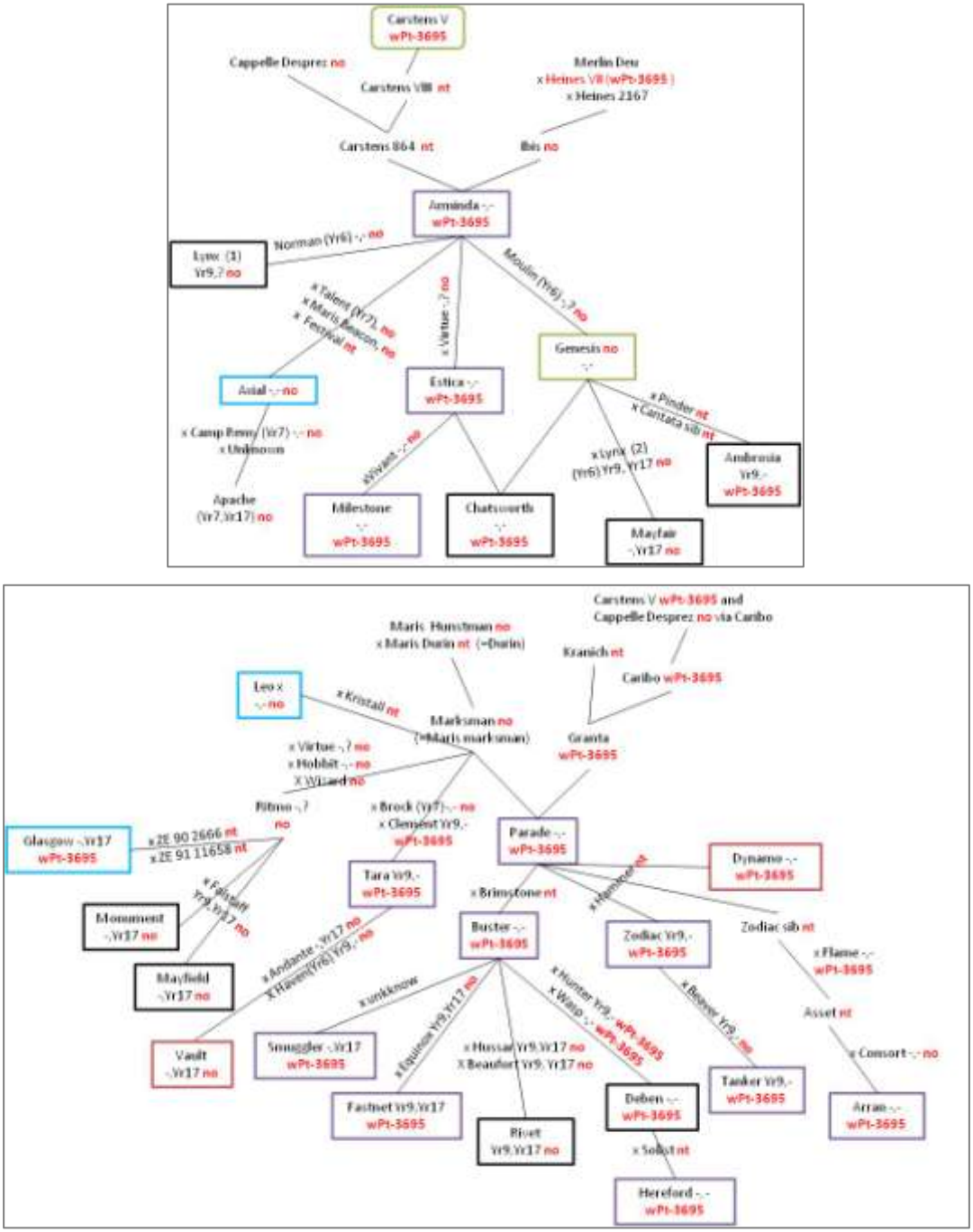

Figure VI-15: Pedigree diagrams of Arminda, Apostle, Armada and Marksman descendants including their genotype for wPt-3695

The genotype for wPt-3695 is indicated in red, wPt-3695: presence of allele 1, no: presence of allele 0, nt: genotype not available. The colour code relates to infection type response against 08/21 and 03/21 isolates: blue for IF (03/7)<4 (resistant) and IF 08/21>6 (Susceptible), violet for IF (03/7)<4 (resistant) and IF 08/21<4 (resistant), red for IF (03/7)>6 (susceptible) and IF 08/21<4 (resistant), green for IF (03/7)>6 (susceptible) and IF 08/21>6 (susceptible), black for IF between 4 and 6 (intermediate response) in one of the test.

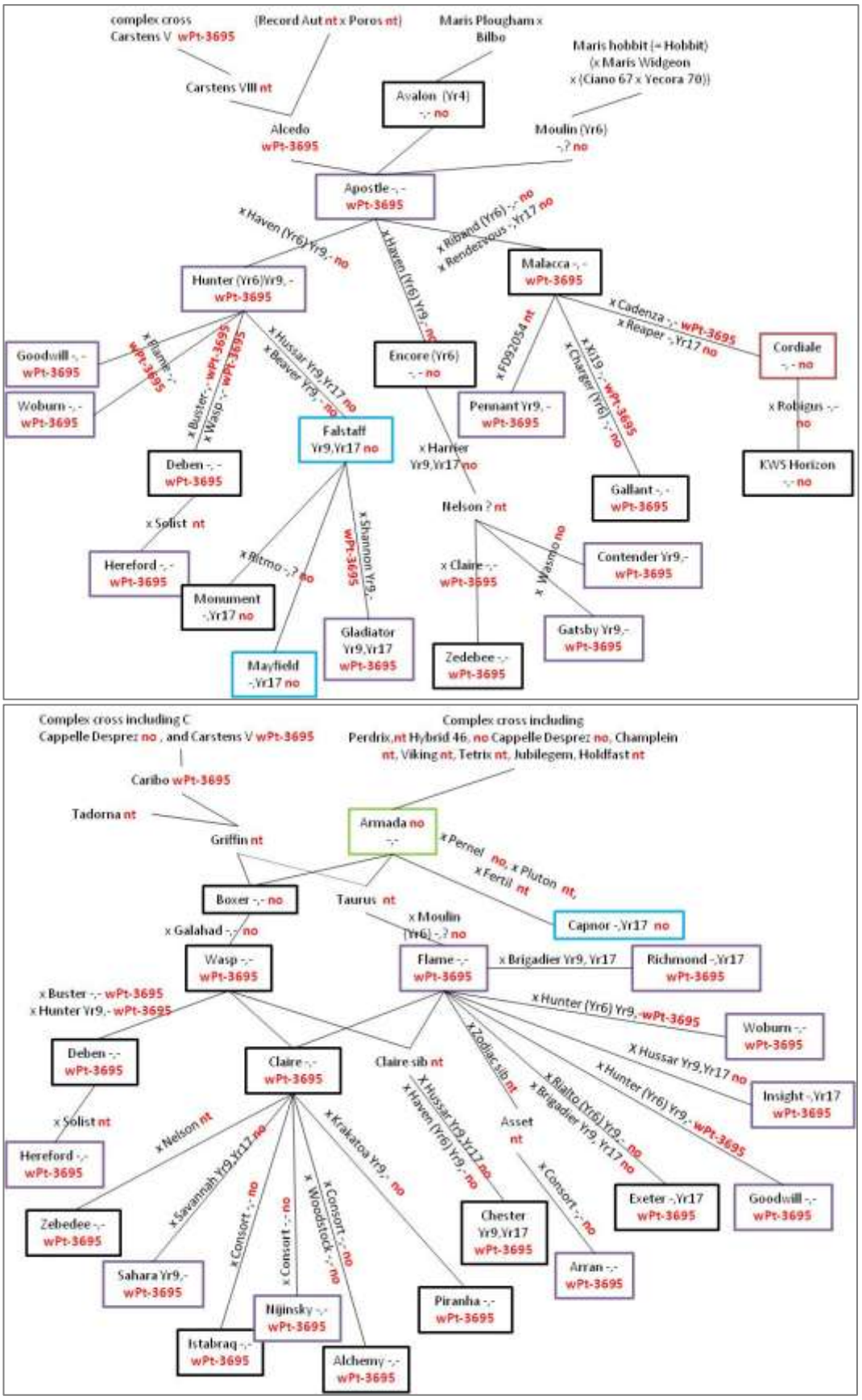

Figure VI-15 continued

Claire and Alcedo are both known for their durable YR field resistance in Europe and the QTLs underlying their resistance have been investigated respectively by Jagger et al. (2011) and Powell (2010). Both varieties presented a QTL on 2DL with major effect directly related to SSR marker gwm301 which suggest their common origin. The comparison between A. Killian consensus map and the linkage maps Claire x Lemhi and Alcedo x Brigadier (Figure VI-16) suggests markers from MTA group 2B4, wPt-743307 and wPt-732666, point to the same QTL on 2D. Additionally, Jagger et al. (2011) reported the resistance from 2D QTL was detected at adult stage but was also likely to be expressed at earlier stage. The intermediate response at seedling stage observed in many lines with wPt-3695 can be the result of a similar partial expression of the same adult plant resistance.

Based on these observations, we suggest allele 1 from markers of group 2B4 point to a 2A locus in Cadenza background which is not linked to a resistance gene, while allele 1 in Carstens V descendants point to a 2D locus linked to an adult plant resistance with main effect, expressed partially at seedling stage. Mapping STS-wPt-3695 in Alcedo x Brigadier and Claire x Lemhi would help to confirm this hypothesis. We suspect the group 2B4 represented also a locus on 2B, but further investigations will be needed to know if this locus is linked to an additional resistance.

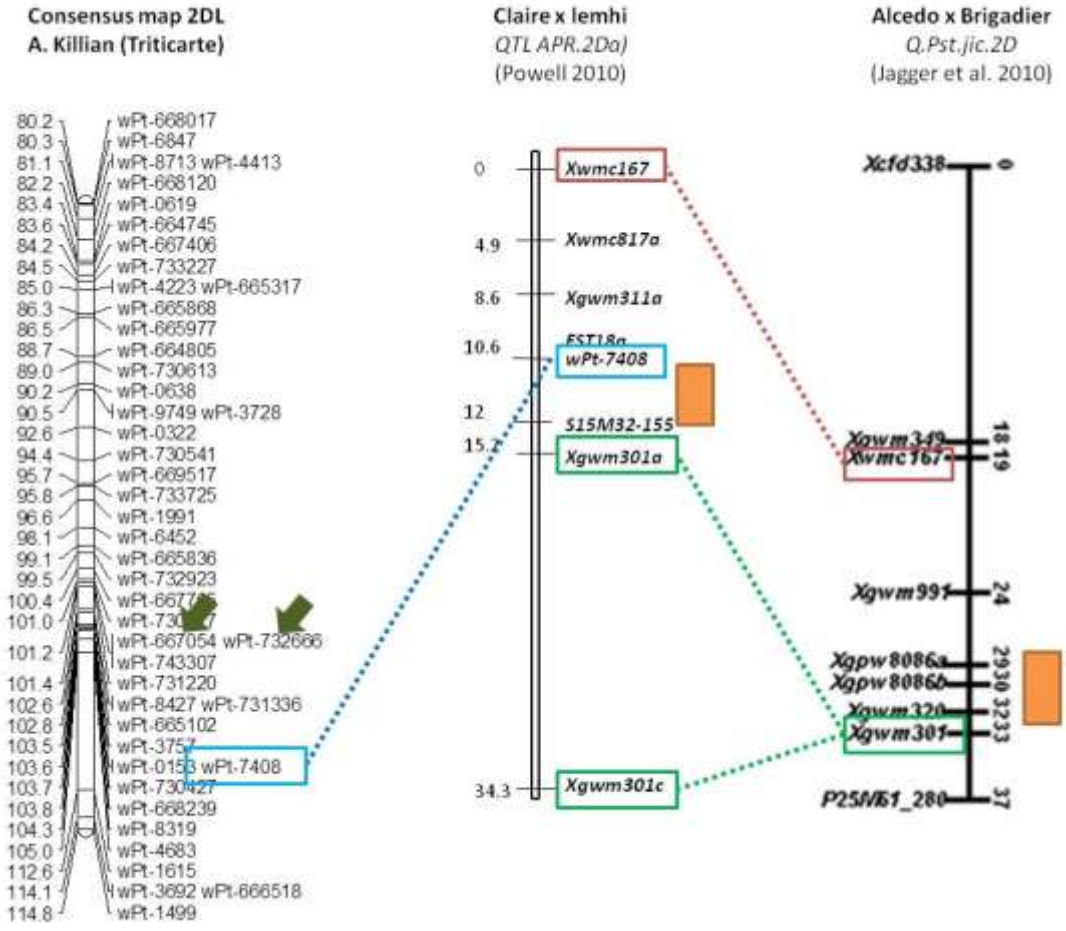

Figure VI-16: Comparison 2DL QTLs identified in bi-parental populations

Alcedo x Brigadier, Claire x Lemhi with major MTA group 2B4 in YR panel

The QTL positions are indicated by orange rectangles and significant MTAs are indicated by green arrows on the consensus map. For illustration purpose only a subset of DArT marker in the consensus map are represented here.

4 COMPARISON GWA SCANS AND CONCLUSION

All the MTA groups identified with historical data and *de novo* phenotypes are summarized in Table VI-19. Considering the *Yr* genes and QTLs already published (see literature review for *Yr* genes and Appendix 2 for QTLs), we mapped theses using the locations given by the authors and use our consensus map to overlay the positions of the markers showing significant association. When a published locus and a MTA mapped to a similar location, we indicated them in the column "*Yr* genes and QTLs". *Yr* genes recently transferred from alien species such as *Yr38, Yr32* were excluded of the postulations since there are not likely to be present in the YR panel. Furthermore, we considered the extended virulence profile of Pst isolates 08/21, 03/7 and 08/501 and excluded seedling resistances not effective from MTA groups identified in *de novo* phenotypes. For instance, race specific *Yr* gene *YrDa1* issue from American cultivar Daws is allocated to chromosome 1A. However, Daws resistance was shown to be overcome by the recent UK Pst isolates 08/21, 08/501 and 03/07 in extended virulence tests, therefore the QTLs identified on 1A are likely to be a new resistance gene.

When the MTAs were mapped on A. Killian consensus map which contains exclusively DArT markers, it was not possible to compare the MTA position to published locus with other marker systems.

Although, our consensus map contains a large number of markers (DArT, SSR, SNP), it was not always possible to relate published QTLs/genes to DArT markers, especially when the QTLs/genes were mapped using AFLP and RAGP markers. Thus the postulated column remains empty for many MTA groups despite QTLs and *Yr* genes have been identified for almost all chromosome arms.

Additionally, all significant DArT markers were compared to DArT markers found associate with yellow rust resistance in CIMMYT elite spring wheat via association mapping (Crossa et al., 2007). Where a common marker was found MTA ESW is indicated in the column "Yr genes and QTL".

Table VI-19: Summary of MTAs detected with historical data and *de novo* phenotypes

The highest P-value for each group is presented: P-value <0.00005 in dark blue, P-value between 0.0001 and 0.00005 in purple, P-value between 0.001 and 0.0001 in red, P-value between 0.01 and 0.001 in orange, [a] chromosomal location from

P. Bansept consensus map, [b] chromosomal location from A. Killian consensus map, [c] markers with low allele frequency (MAF<0.05) not tested with all phenotypes. [d] markers from 2B4 were first assigned to chromosome 2B based on our consensus map but it appears they mapped to multiple homeologues from group 2. HISTO includes all the GWA scans done with historical data. APT includes all the GWA scans done with different scores (Sev, HostR, AUDPCr), SDT includes a single scan for each seedling test. "APT 2010 S lines" is the result of the scan done with lines susceptible at seedling stage against 08/21.
[e] QTLs mentioned mapped within 10 cM of the MTAs identified on our consensus map, the QTLs refer to Bariana et al. (2010) for Janz, Powell (2010) for Claire, Jagger et al. (2011) for Alcedo, Agenbag et al. (2012) for Cappelle Desprez, Christiansen et al. (2006) for Deben, Wasmo and Kris, Mallard et al. (2005) for Camp-Remy, Rosewarne et al. (2012) for Avocet and Pastor, Dolores Vazquez et al. (2012) for Stephens, Hao et al. (2011) for Pioneer 62R61. MTA ESW identified MTAs detected in CIMMYT elite spring wheat by Crossa et al. (2007). No map location was available for *Yr* genes underlined. Postulations in bold correspond to examples described in details elsewhere in this chapter.

Group	Chr.	Interval (cM)	No. markers	Major MTAs	HISTO	SDT 03/7	SDT 08/21	APT 2010 All lines	APT 2010 S lines	APT 2011	*Yr genes and QTLs* [e]
1A1	1AS	10.4-21.1 [a]	6	wPt-4676		xx		xxxB			-
1A2	1AS	11.4-14.8 [b]	4	wPt-665174	*x*			xxxB		x	-
1A3	1AS	67.5-71.0 [a]	3	wPt-666607				xxxB		x	*Qyr.sun-1A Janz*
1BS-1RS	1BS	0-93.4 [a]	102	wPt-8930, 1BL.1RS		xxxB				x	***Yr9*** *,Yr10,Yr15,Yr24*
1B1	1BS	37.6 [b]	1	wPt-2694	*xxx*			x			-
1B2	*1BL*	*136.0* [a]	*1*	*wPt-3465*		xx					*Yr24, YrH52, MTA ESW*
2AS-2NS	2AS	11.9-40 [a]	17	wPt-744900, SC-Y15	*xx*						***Yr17****, MTA ESW*
2A1	2AS	61.6-71.2 [a]	5c	wPt-1657	*xx*		x	xx	xx		*QYr.ufs-2A Cappelle Desprez, QYrst.orr-2AS Stephens*
2A2	2A	unknown	1	wPt-4021	*xx*						***Yr32***
2B1	2BL	84.1 [b]	2	wPt-2293	*xxxB*	x					***Yr7****,Yr5,YrSP*
2B2	2BS	53.5-53.6 [a]	4	wPt-1489	*xx*			x		x	*Yr27, Yr31,MTA ESW*
2B3	2BL	118.1-123.7 [a]	3	wPt-9350, wPt-0950			x	xxxB		xx	***Yr7****, Yr5,YrSP, MTA ESW*
2B4 [d]	2AL 2BL 2DL	179.5-180.6 [a]	9	wPt-3695, wPt-669273	*xxxB*		xxxB	xxxB	x	xxxB	***QTLAPR.2Da Claire, QPst.jic-2DL Alcedo***
2D1	2DS	48.7 [b]	1	wPt-6419	*xx*						-
2D2	2D	101.2 [b]	1	wPt-667054	*xxxB*						-
3A1	*3AL*	*196.8* [a]	*1c*	*wPt-5476*				*x*	*xx*	*x*	*YrTr2*
3B1	3BL	125.2-132.5 [b]	7	tPt-7594	*xx*	x					*MTA ESW*
3B2	3BL	210.0-210.3 [a]	3	wPt-6785	*x*					xx	*YrS, YrSte2*
3B3	3BL	272.2-281.5 [a]	2	wPt-10537	*xx*		x				*YrS, YrSte2*
3D1	3DS	5.8-8.6 [a]	52	wPt-741820, wPt-1336	*xx*			xx			*MTA ESW*
4A1	4AS	18.4-23.4 [a]	3	wPt-8657	*xx*						*YrHVII, YrMin, YrND*
4A2	*4A*	*85.0-85.2* [a]	*3c*	*tPt-9400*				*x*		*x*	-
4D1	4DS	43.9 [a]	1	Rht-D1a/b	*xx*			x			*Yr22,Yr28*
4D2	4D	unknown	1	wPt-731627	*xxxB*						-
5A1	*5AS*	*25.5* [a]	*1*	*wPt-1165*				*x*		*x*	*MTA ESW*
5B1	5BS	15.2 [b]	2	wPt-0708	*xx*					x	-
5B2	5BS	57.2-65.6 [a]	6	wPt-8604	*xx*						-

Group	Chr.	Interval (cM)	No. markers	Major MTAs	HISTO	SDT 03/7	SDT 08/21	APT 2010 All lines	APT 2010 S lines	APT 2011	*Yr genes and QTLs* [e]
5B3	5BL	94.7 [b]	3c	wPt-3763	*xx*						-
5B4	*5B*	*125.3-128.0* [a]	*9*	*wPt-1548*	*x*			*x*		*x*	*QYr.inra-5BL.1 Camp Remy*
5D1	5DS	13.2 [a]	2	wPt732418				xx			-
6A1	6AS	9.3-17.3 [a]	3	wPt-2228			x	xx	x	x	*QYr.uga-6AS Pioneer 62R61*
6A2	6AS	55.0-62.7 [a]	14	wPt-3665,wPt-9584, wPt-664552	*xx*		x	x		x	*QTL 6AS Avocet*
6B1	6BS	72.9-83.2 [a]	7	wPt-2424, wPt-4893	*xx*			x	x		*Qyr.sun-6B Janz, QTL Deben Wasmo, Kris*
6B2	6BL	108.7-113.9[b]	4c	wPt-3207		xx				xx	-
7A1	7AS	40.6b	2	wPt-740561	*xx*			x	xx		-
7B1	7BS	3.0-3.3 [a]	2	wPt-743645		xx					-
7B2	7BL	109.0-119.5 [a]	4	wPt-4814	*xx*						*Yr39,QTL 7BL Pastor, QTLAPR.7B Claire*
7B3	7BL	141.6-143.0 [a]	1	wPt-0752	*xxxB*	xx					***Yr6,** QTL 7BL Pastor, QTLAPR.7B Claire*

14 MTA groups detected using historical data were also detected with de novo phenotypes, confirming the value of GWA scans on historical data and our statistical approach. Seven of the common MTA groups (2A1, 2B1, 2B4, 3B1, 3B3, 6A2, 7B3) were identified in seedling tests and are likely to represent race specific, "all stages" resistance with major effect like *Yr6* (7B3) and *Yr7* (2B1). Nine MTA groups identified were only seen with historical data and may represent resistance genes overcome by the recent Pst isolates like *Yr17, Yr32.* Alternatively the locus identified may still be efficient but not detectable in *de novo* data as their effect may be masked by other genes or their allele frequencies in the complete YR panel may be limited. It is also worth noticing, historical scans identified three MTA groups specific to adult plant resistance identified with seedling susceptible lines with Pst 08/21 (APT 2010 S lines). On the other end, 11 MTA groups were detected only with *de novo* data, the high heritability of *de novo* phenotypes, in conjunction to a higher number of varieties tested are likely to have improved the power of detection by association mapping.

In addition to *Yr6, Yr7, Yr9, Yr17* and *Yr32*, some significant DArT markers proved to be associated with known *Yr* genes locus such as *Yr24* on 1B (1BS-1RS and 1B2) which is closely linked to gwm11 (Zakari et al., 2003) and gwm498 (Li et al., 2006a), *YrH52* on 1B (1B2) which is closely linked to gwm11 and gwm18 (Lin and Chen, 2007a), *Yr15* on 1B (1BS-1RS) closely linked to gwm33 (Chague et al., 1999), *Yr31* on 2BS closely linked to wPt-6268 (Rosewarne et al., 2012) near wPt-1489 (group 2B2) and *Yr39* on 7BL linked to gwm131(Lin and Chen, 2007b) (see consensus map in Appendix 11 for SSR locations). Furthermore, the GWA scan proved to detect QTLs originated from lines linked with the YR panel (Camp-Remy, Claire, Cappelle Desprez, Deben, Wasmo, Alcedo) (Table VI-19)

providing an independent validation. Finally, when compared to the association analysis of historical bread wheat germplasm carried by Crossa et al. (2007), ten significant DArT markers (wPt-3465 1B2, wPt-6207 2AS-2NS, wPt-1489 2B2, wPt-9350 2B3, wPt-3378 2B3, wPt-0950 2B3, wPt-8845 3B1, wPt-1336 3D1, wPt-9401 3D1, wPt-1165 5A1) were common with the present study, suggesting that similar resistance genes have been deployed in CIMMYT elite germplasm and UK breeding germplasm.

Many MTAs could not be linked to published QTLs and *Yr* genes, mainly because a difference in marker system used and limited mapping information for DArT markers. To help with the validation of MTA, we could consider genotyping the YR panel with markers linked to QTLs from mapping populations and study their polymorphism within the YR panel as well as their LD with markers detected by association genetic. Alternatively, as illustrated with STSwPt-3695 (group 2B4), significant DArT markers can be adapted to PCR markers and map in bi-parental populations presenting resistance to yellow rust. However, to design specific PCR primers, DArT clone sequences are required. During our research project, only a limited number of sequences were available.

Finally, MTAs detected with *de novo* adult plant trials 2010 and 2011 are effective sources of field resistance against widely virulent Pst isolates 08/21 (Solstice race). Since the end of our project, a new race named "Warrior" race has emerged in the UK and presented an even wider virulence profile. In addition to the virulence factor of the Solstice race, the Warrior race, which was predominant in the UK in 2012 is virulent on *Yr7*, *YrSP* and reduces the adult plant resistance of Claire and Warrior (personal communication Rosemary Bayles). Therefore some of the MTAs detected in 2010 and 2011 may have lost their effectiveness in the field, in particular MTA group 2B3 which related to the *Yr7/Yr5/YrSP* locus and MTA group 2B4 which related to an APR QTL locus in Claire. This drastic change in Pst populations within the UK is a reminder that when studying disease resistance, we are working in a constantly evolving context. Therefore, it would be advisable to update regularly the YR panel evaluations with new Pst isolates to follow the durability of the loci identified by association mapping.

CHAPTER VII. CONCLUSIONS AND PERSPECTIVES

1 CONTEXT

To feed the world population expected to reach 9. 3 billion in 2050, it is estimated that agricultural output will have to increase by 70 percent between 2005 and 2050 (FAO, 2006). This implies annual cereal production alone would have to grow by almost one billion tonnes. These challenges will have to be met with limited arable land increase, potentially lower inputs and in a changing environment likely to bring new biotic and abiotic threat. Therefore, the increase of production will have to rely heavily on crop improvement to increase yield potential. A strategic target of breeding for food security is to enhance genetic resistance in cultivated crops (CIMMYT, 2011). Yellow rust is a major wheat pathogen and regular rust epidemics worldwide proved to be a major threat for yield stability (Wellings, 2010). The development of resistant varieties appears to be the most environmentally friendly and efficient way to control yellow rust. However, the great adaptability of Pst populations in conjunction to the wide range cultivation of genetically similar varieties has limit the efficiency of conventional breeding approach, which relied heavily on selecting for one major resistance gene based on phenotype. Wellings (2010) reports little progress have been made in containing the worst effect of yellow rust epidemics over the past 50 years. It is becoming increasingly apparent that specific selection strategies are needed to enhance durability of rust resistance. Gene pyramiding constitutes one of the most promising prospects to create durable disease resistances by selecting for two or more resistance genes against a pathogens using markers assisted selection.

To effectively develop and deploy resistance based on diverse yellow rust resistance genes, it is important to determine their chromosomal locations and develop diagnostic markers for marker-assisted selection. It is where association mapping comes handy. Comparing to QTL mapping in bi-parental population, association mapping allows to investigate simultaneous multiple sources of resistance and is likely to identify markers related to locus of interest with an improved resolution. Additionally it is a cost effective method since there is no need to develop expensive bi-parental mapping population. Instead varieties from different germplasm collections can be assembled directly to create an association panel. The cost efficiency can be even increased when historical phenotype data are already available.

Within the UK, the UKCPVS constitute a valuable resource of historical evaluation of elite germplasm against a wide range of Pst isolates. Taking advantage of those data we intended to identify markers linked to specific resistance genes using an exclusive approach based on Pst seedling stage virulence profiles and access the diversity underlying yellow rust resistance in UK elite breeding lines. Additionally, in the light of a new emerging Pst race in 2008, namely the "Solstice" race, with a wide virulence profile, the project was extended to include *de novo* evaluations and identify resistance loci efficient against the current Pst populations.

2 A NEW ASSOCIATION PANEL FOCUS ON UK GERMPLASM: YR PANEL

One of the main achievements of this project is the creation of the YR panel, a new association panel focus on UK elite wheat from the past three decades including 327 lines. The YR panel in conjunction of its genotype with 1806 DArT markers constitute a unique resource to forward UK wheat breeding. One can do an association study and identify MTAs for many traits segregating within the YR panel with nothing more than a new set of phenotypes. Many traits of agronomical interest could be looked at with minor investments. Following this principle, Neumann et al. (2011) studied twenty agronomic traits in the field on a core collection of 96 accessions, the same collection was used later to study seed dormancy, seed longevity and preharvest sprouting (Rehman-Arif et al., 2012; Rehman et al., 2012). Crossa et al. (2007) looked at multiple disease resistances and agronomic traits in two sets of CIMMYT elite spring wheat.

What makes the YR panel so unique is that it represents fairly recent breeding lines adapted to the UK growing environment. Other association panels have been studied but they sampled either a wider growing area (455 soft wheat from continental Europe for (Miedaner et al., 2010), 195 elite wheat from western Europe for (Le Couviour et al., 2011)), a longer breeding history (94 UK wheat varieties from 1845 up to 2002 (White, 2011)) or simply focussed on diversity (96 winter wheat from 21 countries(Neumann et al., 2011), INRA bread wheat core collection of 372 accessions (Balfourier et al., 2007)). Allele of interest identified within the YR panel could be integrated rapidly and easily into UK commercial variety with limited breeding effort as varieties carrying the alleles are themselves adapted to the market and the local growing conditions.

In addition to the seeds and the genotypes, a significant amount of information has been gathered concerning the YR panel lines. Each variety came with its passport data

including its origin (country and breeder), its year of release, its alternative names (code breeder and synonymous) and its pedigree information. They all have been stored in a searchable database including historical yellow rust resistance data from the UKCPVS. Pedigrees have been formatted for pedigree viewer (Kinghorn, 1994), allowing the visualisation of extended links between varieties included in the YR panel but also with a wider set of historical varieties. This information has shown to be valuable to retrace the origin of specific alleles and estimate their spread within the UK germplasm.

3 ASSOCIATION MAPPING FOR DISEASE RESISTANCE AND HISTORICAL DATA: A NEW APPROACH

Although the use of historical data for association studies is often cited of great value, in practice limited studies using historical data have been reported. To my view, three majors factor will influence the success of association mapping with historical data: the completeness of the dataset (matrix year-variety or trial-variety), the heritability of the trait studied (qualitative versus quantitative inheritance and degree of genetic by environment interaction) and finally the number of varieties included in the panel (the number of lines is fixed by the availability of historical data and may be a limiting factor).

One of the major references of association mapping in wheat is the study done by Crossa et al. (2007) using international multi-environment trials to identify MTAs with resistance to stem rust, leaf rust, yellow rust, powdery mildew and grain yield. Using this fairly balanced and extensive dataset on 170 CIMMYT elite spring wheat, they identified multiple LD clusters bearing multiple host resistance genes; most of which co-located with genomic region previously reported with resistance genes or QTLs. Cockram et al. (2010) used replicated registration variety data for 32 qualitative morphological traits available for 200 to 500 elite barley cultivars and reported the GWA mapping for 15 of them. White (2011) explored the feasibility of association mapping using historical data and concluded it was possible to conduct successful association studies in wheat and barley using historical phenotypes, however he observed a lack of power in his wheat population (204 UK winter wheat varieties) to discover QTL for quantitative traits (yield, protein content, specific weight, Hagberg falling number) using a highly incomplete year-phenotype matrix. He was more successful with his barley population as he studies essentially botanical descriptors independent of the environment and his marker density was greater. Recently Pozniak et al. (2012) demonstrated that unbalanced historical data were a useful resource for discovery of MTAs in durum wheat using GWA mapping as they were able to identify genomic region previously identified for cadmium and yellow pigments coloration, but they also highlighted

the fact that unbalanced data and variation affecting measurement of the phenotype would make it difficult to detect QTL with small effect on highly quantitative trait.

In the present study, we explore a highly unbalanced dataset of yellow rust resistance evaluations recorded by the UKCPVS. As our focus was on maximising the number of varieties included in the panel, sometimes only one year of testing was available for a selected line; which will have limited the estimation of variance and covariance between years and therefore limited the accuracy of the estimated phenotypic value via linear modelling. Taken these considerations into account, we chose to calculate and use a phenotypic value based on BLUP for AM, the BLUP had the advantage to reduce the uncertainty brought by a lower number of replication, by shrinking the estimated value in function of the number of data point available for each variety. Additionally, to overcome the limits introduced by the polygenic nature of yellow rust resistance and the high environment x genotype interaction due to the diversity of Pst isolates tested, we considered a new approach and partitioned the historical dataset based on Pst isolate virulence criterion, knowing which isolates were used in trials and their virulence profile. By using this approach, we increased artificially the heritability of the trait and limited the number of gene participating to the phenotype compared to the analysis including the entire dataset. Furthermore, we were able to target the discovery to locus specific of certain virulence factors and/or isolates. Although the number of lines tested was reduced in partitioned data, we indentified a higher number of significant MTAs compare to the un-partitioned dataset. These observations resonate with Neumann et al. (2011) findings as they noted that fewer loci were detected for complexly-inheritated or low–heritability traits.

Using targeted analysis, a total of 23 MTA clusters were identified, 21 of which would have failed to be detected using a classical approach. We also successfully attributed five MTA groups to known race-specific Yr genes (*Yr6, Yr7, Yr17* and *Yr32)* and QTL (2D Claire and Alcedo).

The comparison of scans with unbalanced historical data and scans with balanced de novo data provides a further validation of our methodology (data partitioning and phenotypic value estimation) as we identified 14 common MTA clusters.

We demonstrated we were able to identify genes/QTLs with major effect, but a doubt remained as if we were able to identify loci providing partial resistance, many of the MTA clusters identified by AM remained unaffiliated. Further investigation will be needed to determine if any of the MTAs detected are linked to known quantitative QTL or genes.

GWAS using historical disease resistance evaluations has proven to be a cost effective approach to identify QTL linked to yellow rust race-specific resistance and a

similar method could be applied to brown rust and mildew data from UKCPVS. No further investment will be needed as the same panel and genotyping data can be exploited, only time will be required to collect and organized neatly the historical data from paper archives.

4 SOURCE OF YELLOW RUST RESISTANCE AGAINST RECENT PST ISOLATES

The emergence of the "Solstice" race in the UK in 2008, combining virulence for *Yr6, Yr9, Yr17* and *Yr32* accentuated the need to obtain an updated view of the YR panel resistance level. Therefore the YR panel was evaluated a seedling stage and adult stage against recent UK isolates.

The comparison of seedling tests with isolates 03/07 (Brock race) and 08/21 (Solstice race) in combination to genotype for alien introgression including race specific resistances (*Yr9* from 1BL.1RS and *Yr17* for 2AS.2AN) and thorough review of pedigree information, demonstrated many UK lines presented race specific resistance gene *Yr6 (*minimum of 5%), *Yr9* (32 %) and *Yr17* (40%), additionally *Yr7* was postulated to be present in at least 10 varieties. Unfortunately, all these resistance genes broken down in the UK over the past 50 years (UKCPVS reports frequency virulence summarized in Annexe 5). Only the combination *Yr6-Yr7* was still providing an efficient protection until 2011. In 2011, the "warrior" race carrying virulence for *Yr6, Yr7, Yr9, Yr17* and *Yr32* was identified in the UK and rendered ineffective the gene combination *Yr6-Yr7*.

To my knowledge, we reported the first GWA scans on yellow rust seedling tests. These scans proved to be particularly powerful at identifying race-specific genes *Yr6, Yr7* and *Yr9*. All three MTAs were independently validated, markers linked to *Yr9* were found in high LD with diagnostic marker developed by de Froidmont (1998) and markers for *Yr6* and *Yr7* were found within the *Yr6* and *Yr7* QTL intervals detected in mapping population Avalon x Cadenza.

Prior to this project, *Yr6* was only assigned to chromosome 7B but no map position was available, therefore we showed that AM and seedling tests with Pst isolates with targeted virulence profile could be of great use to map many race-specific *Yr* genes for which mapping information is limited. In principle, using the same approach and the Pst isolate collection from the UKCPVS, we could map *Yr* genes *Yr1, Yr2, Yr3a+b, Yr4a+b* and *Yr32* as those genes are known to be present in relatively high frequencies in the YR panel.

The evaluation of the YR panel in the field against isolate 08/21 in 2010 and the mixture of isolates 08/21, 03/7, 08/501 in 2011, proved the current UK breeding germplasm included a high level of field resistance against recent Pst isolates. By comparing with infection type at seedling stage we deduced 10% of the lines minimum presented a source of APR.

The GWA scans with field scores identified a total of 23 loci participating to field resistance, suggesting the sources of resistances within the YR panel were generally diverse. Some MTAs were identical to those detected with seedling tests, thus pointed to seedling resistance (MTAs on 1AS, 1BS, 2AS, 2BL and 6AS). Other MTAs were likely to be specific of APR as they were not identified at seedling stage or they were detected within varieties lacking major seedling resistance (MTAs on 1BS, 2AS, 2BL, 3AL, 3BL, 3DS, 4DS, 5AS, 5B, 5DS, 6AS, 6BS, and 7A1).

Those results suggest there is a diversity of resistance sources within the UK elite germplasm effective against recent UK isolates that could be exploited to create lines with durable resistance using gene pyramiding.

5 INCREASING THE EFFICIENCY AND THE RESOLUTION OF ASSOCIATION MAPPING

While perfect markers were detected for *Yr9* and *Yr17* due to the high level of LD create by alien introgression, we only identified markers loosely correlated with *Yr6*, *Yr7*, and *Yr32*. Furthermore, many of the MTAs detected presented a limited level of significance.

In addition to the locus frequency within the population, the inherent effect of the locus (large or small effect) and the heritability of the phenotype, two other factors influence greatly the detection and the significance level of MTAs: the marker system (density and homogeneity of marker coverage) as well as the statistical model used.

DArT was chosen for the present work as the most efficient and mature (at the time this study was initiated) technology that could offer unbiased genome-wide coverage of the hexaploid wheat genome). However, DArT showed some limitations. Mapped DArT markers were unevenly distributed in the genome, (1) the marker density was greater on genome B, (2) the marker coverage of several chromosomes was sparse, (3) DArT markers presented a high degree of clustering. Similar observations have been made by Semagn et al. (2006b), Peleg et al. (2008), Akbari et al. (2006), Francki et al. (2009), Marone et al. (2012b) and Marone et al. (2012a). The uneven coverage biased the MTA discoveries toward genomic region with higher marker density. In addition, the marker number was limited,

with 1806 markers, we could achieve at best a density of one marker every 3cM. While this marker density was considered sufficient to identify MTAs taking into account the slow LD decay observed within the YR panel (Chapter V), it is not adequate to detect causative mutations or tightly linked markers usable as diagnostic marker. Chao et al. (2010) estimated that at least 17,500 markers evenly distributed in the wheat genome are needed to detect MTAs within an interval of 0.2 cM of a causative mutation. This amount of markers were not available at the start of our project, but since the situation has greatly improved. We saw in 2012 the development of a wheat iSelect SNP chip including 90,000 SNP markers. Although, the mapping information, the uniformity of distribution and the redundancy of these SNPs are yet to be disclosed, this new state-of-the-art resource will undisputedly improve the marker coverage, increase the discoveries of MTAs and improve the resolution of AM studies. I am happy to say that the YR panel have been integrated in a new project and will be genotyped shortly with the new SNP chip.

Genotyping by sequencing is another strategy that could be explored to increase the marker coverage for association mapping, Elshire et al. (2011) and Poland et al. (2012) demonstrated that genotyping by sequencing in species with large genome including wheat was now feasible.

The second important factor to consider for improving MTA detection is the statistical model applied. In the present study, we used a two steps modelling approach. The first step was to obtain a BLUP for each variety by integrating environmental effects (year, trial, and block). The second step test for association between a single marker and the phenotypic value estimate (BLUP) and include a correction for population structure to limit the risk of false discoveries.

While this approach is widely accepted in plant genetics, it is in some ways restrictive. First, we are studying a polygenic trait using a single locus model which could be a source of spurious association (Platt et al., 2010), but also limit the detection of multiple loci. For instance, adding a marker with main effect as covariate may allow the detection of more MTAs. Additionally our approach does not allow the detection of potentially important interactions between QTLs (QTL X QTL) or between a specific QTL and the genetic background (QTL x genetic background). Including an interaction component in the model could for instance identify valuable haplotypes for use in MAS; it may also allow the identification of inhibitor of resistance. Secondly the two steps approach limits the estimation of QTL x environment interaction (GEI). Wei et al. (2010) in sugarcane compared simple association model with model accounting simultaneous for population structure, GEI and spatial variation and concluded that association mapping failing to account for GEI

would have low value in breeding programs as they found many. A nice example of improved model for multi-locus trait in wheat is provided by the study of Miedaner et al. (2010), they applied a one step approach including a two-dimensional scan for pairwise interaction effect among major loci and showed an improvement in the proportion of genotypic variance explained and identified useful epistatic interaction between reduce height gene *Rht-D1* and Fusarium head blight resistance locus *Fhb1*.

6 VALIDATION OF MTAS

A total of 38 MTAs clusters were identified in our studies. Although we included population structure correction within our model and considered a higher significance threshold (only markers with at least one P-value<0.001 were reported) to limit the detection of spurious associations, some false discoveries may remain. Therefore, an independent validation of MTAs is useful; we showed three ways to validate some of the associations:

- Testing the effect of the marker identified by AM in a bi-parental population (example of *Yr6*, *Yr7* and wPt-3695), which is the only option available for *de novo* QTLs.
- Demonstrating a high correlation between a marker detected by AM and a diagnostic marker (example of *Yr9* and *Yr17*)
- Co-localizing MTAs with published QTLs/genes based on common markers (example of *Yr7* in Camp Remy and Apache, and QTLs from Claire, Alcedo)

Only a few MTAs groups from our study were validated as we faced some difficulties due to the marker system. First, DArT markers are relatively recent and only limited mapping studies used DArT. Secondly, many DArT markers were not mapped which made the comparison of MTAs with published QTL/genes impossible. Thirdly, DArT genotyping is only available as a multi-assay from Triticarte and does not allow single marker genotyping in the lab. Therefore to be used locally either for mapping purpose or application of MAS, DArT markers need to be adapted in PCR- based assay. The development of monopole assay using the sequence of DArT marker was used for wPt-3695, our most significant MTAs, but could not be extended to other markers as limited DArT sequences were available. Over the past three years, things have improved greatly; more mapping studies are published with DArT markers, more DArT markers are mapped and more DArT sequences have been released. In addition the availability of Chinese spring genomic draft genome assembly may give access to the genomic sequence surrounding DArT marker clone and can allow the design of co-dominant SNP assay.

A valuable addition to the present work would be to design mono-plex assays for all significant MTA groups based on the newly available sequences and map them in bi-parental

populations segregating for rust resistance response. The genotyping of the YR panel with SSR linked to yellow rust QTLs and their comparison with DArT genotypes will also help with the validation of MTAs.

7 BEYOND ASSOCIATION MAPPING

The ultimate goal of MTA discoveries is to identify markers useful to develop wheat lines with durable yellow rust resistance via MAS. The consensus is that to achieve durability, several resistant loci must be considered, however the usefulness of major R-genes is sometimes questioned. The utilization of two effective R-genes in combination can enhance resistance durability, but according to Singh (2012), such a strategy much be strictly followed by all breeding programs for a long lasting success, as the release of varieties which carry the same R-genes singly will undermine this approach. Alternatively, durable resistance can also be achieved through pyramiding several minor or partial resistance genes. Implementing gene pyramiding, CIMMYT has developed wheat lines with near immune levels of adult-plant resistance based on 4-5 partial rust resistance genes that have small to intermediate, but cumulative effect (Singh et al., 2000a). Other examples in European wheat showed that long lasting resistance can result of the presence of several major race-specific resistance genes (R-genes) in conjunction with adult plant partial resistance loci(PR-gene), for instance in cultivar Cappelle Desprez (Johnson, 1984), Camp Remy (Mallard et al., 2005), Renan (Dedryver et al., 2009) and Apache (Paillard et al., 2012).

Using the results from our study, we could use the makers linked to major resistance genes *Yr6, Yr7, Yr9, and Yr17* to avoid the introduction of these major R-genes and concentrate the breeding effort on resistance loci identified at adult stage. Alternatively, we could intentionally select some seedling resistance loci to be introduced in complement of adult plant resistance loci.

It is also worth noting that the availability of markers for major R-genes may allow studying potential yield drag and yield depression associated with the ineffective resistance genes present in the YR panel. If the ineffective R-genes prove to be associated with a yield depression like it has been seen for leaf rust resistance *Lr9* (Ortelli et al., 1996), it will provide an additional argument to avoid them in future breeding programs

Finally having identified as many as 38 loci involved in yellow rust resistance give the means to breeder to diversify the source of resistance present within their range of varieties, this will improve the resistance durability at the national level.

Beyond MAS, which rely on almost perfect markers, we could also implement a genomic selection approach directly with the results from our GWA scans against recent isolates. Significant markers and *de novo* phenotypes can be used to build a model predicting the breeding value of the lines (in our case the potential resistance level of a line). Half of the YR panel could be used as a training population and half can be used to validate the model. After validation, the model can be used in subsequent breeding populations developed from YR panel line or related lines to select individuals with the best genotypic breeding value (i.e. the best combination of molecular alleles providing resistance) without having to phenotype them.

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APPENDICES

Appendix 1: Mapping information available for yellow rust resistance genes and references

Appendix 2: QTLs for yellow rust resistance

Appendix 3: Statistical analysis of historical data (Genstat output)

Appendix 4: Summary yellow rust evaluations on the YR panel lines and genotype for *Rht1, Rht2, Ppd-D1,* 1BL.1RS translocation and 2NS.2AS introgressed fragment with *Yr17*

Appendix 5: Virulence frequency observed (%) from Pst isolates tested by the UKCPVS from 1969 to 2010.

Appendix 6: Frequency of pathotype sampled in the United Kingdom from eurowheat.com

Appendix 7: List of differential hosts and varieties control tested to determine the extended virulence profile of Pst isolates used in *de novo* evaluations

Appendix 8: Pedigree diagrams

Appendix 9: Primers sequences and PCR conditions for *Rht1* and *Rht2*

Appendix 10: Primers sequences and PCR conditions for *PpD-D1*

Appendix 11: Consensus map

Appendix 12: Major MTAs for height in YR panel

Appendix 13: MTAs for yellow rust resistance and P-Values for historical data GWA scans

Appendix 14: MTAs for yellow rust resistance and P-values for *de novo* phenotypes GWA scans

Appendix 15: List of markers included in MTA groups 1BS-1RS, 2AS-2AN, 3D1 and 6A2

Appendix 16: Analysis of variance and Chi squared test for goodness of fit for infection type observed in Avalon x cadenza population

Appendix 17: Linkage group 2AL including marker STSwPt-3695 from Avalon x Cadenza mapping population

APPENDIX 1: Mapping information available for Yellow rust resistance genes

[a] Symbol for traits and genes associated to *Yr* genes
Yr followed by a number or letter: reaction to Yellow rust
Pm followed by number or letter: reaction to Powdery mildew,
Sr followed by number or letter: reaction to Stem rust,
Lr followed by number or letter: reaction to Leaf rust,
Bdv1: reaction to barley yellow dwarf virus,
Gli1b: gliadin 1b,
Gpc-B1: Grain protein content,
Ltn and *Ltn2*: Leaf tip necrosis,
Ppd1: photoperiod response,
Rht8: Reduce height,
RspLem: resistance to barley yellow rust (*Puccinia striiformis* f.sp *hordei*)
Rg1: Red glume colour,
Vga1: virginiamycin-like antibiotics gene
[b] Molecular markers acronyms:
AFLP: amplified fragment length polymorphism
CAPS: cleaved amplified polymorphic sequence
RAPG: resistance gen analog polymorphism
RAPD: random amplification of polymorphic DNAs
RFLP: restriction fragment length polymorphisms
SCAR: sequence characterized amplified region
SSR: single sequence repeat
SNP: single nucleotide polymorphism
STS: sequence tagged site

ed

***Yr* gene**	**Mapping information[a]: genes and markers linked to *Yr* genes, flanking markers and genetics distance**	**Marker system[b]**	**References**
Yr1	***Yr1*** - 2 cM - *Pm4a*, ***Yr1*** - 16.5 cM - *Sr48*, Xfba-2A - 1.3 cM - Xstm673acag - 1.1 cM - ***Yr1***	RFLP	McIntosh and Arts (1996)
Yr3a (YrV23)	***YrV23*** - 9.4 cM - Xwmc356-2B	SSR	Wang et al. (2006)
Yr4 (YrRub)	***Yr4***-2.9cM-cfb3530-2.4cM-barc 75	SSR	Bansal et al. (2010)
Yr5	***Yr 5*** completely linked with RGAP Xwgp 17, 19 and 26-2B, Xwgp17-2B was converted into a CAPS PCR marker for ***Yr5,*** ***Yr5***/S19N93-140 - 0.7 cM - S23M41-310, 2 STS markers were derived from those 2 AFLP markers	RGAP, AFLP	Yan et al. (2003), Chen et al. (2003b), Smith et al. (2007)
Yr7	***Yr7*** closely linked with Sr9g, ***Yr7*** - 5.3 cM - Xgwm526-2B	SSR	McIntosh et al. (1981), Yao et al. (2006)
Yr8	***Yr8*** and *Sr34* located on the translocation 2M from *Ae.Comosa*	-	Friebe et al. (1996)
Yr9	***Yr9*** and *Pm8*, *Sr31*, *Lr26* located on the translocation 1RS from Rye Petkus, ***Yr9*** - 3.7 cM - Xgwm582-1BL, 4 RAPG markers Xwgp 4, 7, 8 and 9 coincident with ***Yr9***	SSR, RGAP	Mago et al. (2002), Weng et al. (2005), Shi et al. (2001)
Yr10 (YrVav)	***Yr10*** - 2 cM - Rg1, ***Yr10*** - 1.2cM - Xpsp3000-1BS - 4cM - *Gli1b*, STS marker S26-M42 co segregate with *YrMoro*	SSR, STS	Metzger and Silbaugh (1970),Wang et al. (2002) Smith et al. (2002)
Yr15	Xgwm33-1B - 4.5 cM - ***Yr15*** - 4.7cM - $UBC199_{700}$ - 5.6 cM Nor - B1	SSR, RAPD	Chague et al. (1999)
Yr16	probable gene order *Rht8* - *Ppd1* - 36 cM - ***Yr16*** - 25cM - Da-Su-D		Worland et al. (1988)
Yr17	***Yr17*** closely linked to *Lr37*, *Sr38* and SCAR marker SC-Y15, developed from RAPD marker OP-Y15580, and to Xpsr150-2Nv, ***Yr17*** linked with *Vga1*, PCR assay based on Vga1 gene-like sequence was developed	SCAR ,RAPD	Robert et al. (1999), Seah et al. (2001)

APPENDIX 1 Continued

Yr gene	Mapping information[a]: genes and markers linked to *Yr* genes, flanking markers and genetics distance	Marker system[b]	References
Yr18 Cloned	***Yr18*** linked with *Bdv1, Lr34*, ***Yr18*** linked Xgm295.1 and Xwgm44, Xgwm120-7D - 0.9 cM - ***Yr18/Lr34/Pm38*** - 0.7 cM - Xgwm295-7D, 6 gene-based markers to ***Lr34/Yr18*** named cssfr 1 to 6 , ***Yr18*** completely linked with *Ltn*, *Pm38*, *Lr34*	SSR, gene based marker based on deletion and SNP	Singh (1993), Suenaga et al. (2003), Spielmeyer et al. (2005), Lagudah et al. (2009), McIntosh et al. (2009)
Yr21 (YrLem)	***Yr21*** - 0.3 cM - *YrRpsLem*	RGAP	Pahalawatta and Chen (2005)
Yr24 (YrCH42)	*Yr15* - 4 cM - ***Yr24***, Xbarc187-1B - 2.3 cM - ***Yr24*** - 1.6 cM - Xgwm498-1B, *Yr26*,***Yr24,*** *YrCH42* likely to be the same gene Gene order *Yr15* - *Yr24* - Xgwm11-1B	SSR	McIntosh and Lagudah (2000), Li et al. (2006a) Zakari et al. (2003)
Yr26 (YrCH42)	***Yr26*** - 1.9 cM - Xgwm11/Xgwm18-1B- 3.2 cM - Xgm413, Xwe177/20-1B1 - 0.3 cM - Xwe173 - 1.4 cM - ***Yr26*** - 6.7 cM - Xbarc181-1BL- 3.0 cM - Xwmc419	SSR, STS	Ma et al. (2001), Wang et al. (2008)
Yr27 (YrSk)	***Yr27*** closely linked with *Lr13*, *Lr23*, *Yr31*(see *Yr31*), ***Yr27***associated with Xcdo152-2B and Xcdo405-5B	RFLP	McDonald et al. (2004), Singh et al. (2003)
Yr28	***Yr28*** closely linked with Xmwg634-4DS	RFLP	Singh et al. (2000b)
Yr29	***Yr29*** completely linked with *Lr46*, Xwmc44-1B - 1.4 cM - Xbac24prot - 9.5 cM - ***Yr29/****Lr46* -2.9 cM - Xbac17R-1B, Xgwm44-1B - 3.6 cM - ***Yr29*** - 2.1 cM - XtG818/XBac17R-1B ***Yr29*** completely linked to *Lnt2*	AFLP, SSR	William et al. (2003), Rosewarne et al. (2006)
Yr30	***Yr30*** closely linked with *Sr2* and *Lr27*	-	McIntosh et al. (2008)
Yr31	***Yr31*** located in or near a cluster of resistance genes including *Lr13, Lr23, YrSp, Sr10* Recombination values: ***Yr31*** - *Yr27*= 0.148; ***Yr31*** - *Lr23*=0.295; ***Yr27*** - *Lr23* =0.131	-	Singh et al. (2003)
Yr32 (YrCV)	Xwmc198-2A- 2 cM - ***Yr32,*** ***Yr32*** coincident with AFLP marker M62/P29-156	AFLP, SSR	Eriksen et al. (2004)
Yr33	***Yr33*** flanking markers Xgwm111-7D and Xgwm437-7D	SSR	Nazari and Wellings (2008)
Yr34 (YrWA)	Xgwm410.2-5A - 8.2 cM - B1 - 12.2 cM - ***Yr34***	SSR	Bariana et al. (2006)
Yr35 (YRs8)	***Yr35*** linked with *Lr53* *Xgwm191-6B* – 18.9 cM – ***Yr35*** – 3 cM – *Lr53* – 1.1 cM – *Xcfd-6B* – 3.4 cM – *Xgwm50-6B*	SSR	Marais et al. (2005b) Dadkhodaie et al. (2011)
Yr36 Cloned	Xucw68-6B - **Xucw69-6B/Xbarc101-6B/*Yr36*** - Xucw66-6B, ***Yr36*** is 2-4 cM proximal to *Gpc-B1* ***Yr36*** maps between Xucw129 and Xucw148 (0.02 cM).	SSR, RFLP, INDEL	Uauy et al. (2005) Fu et al. (2009)
Yr37	***Yr37*** linked with *Lr54*	-	Marais et al. (2005a)
Yr38 (YrS12)	***Yr38*** linked with *Lr56*	-	Marais et al. (2006)
Yr39	***Yr39*** closely linked to RAPG markers Xwgp36 and Xwgp45	RAPG	Lin and Chen (2007a)
Yr40	***Yr 40*** completely linked with *Gsp*, *Lr57*, Xfbb276 and Xbcd873 CAPS marker *XLr57/****Yr40****-MAS-CAPS16*	RFLP, CAPs	Kuraparthy et al. (2007), Kuraparthy et al. (2009)
Yr41 (YrCN19)	***Yr41*** - 0.3 cM - Xgwm410, ***Yr41*** - 7.9 cM - Xgwm374, ***Yr41*** - 12.3 cM - Xwmc477, ***Yr41*** -21.2 cM - Xgwm382	SSR	Luo et al. (2009)
Yr42	Associated with Lr62	-	Marais et al. (2009)
Yr43	Xwms501-2B – 11.6 cM – Xwgp110-2B – 4.4 cM – ***Yr43*** – 5.5cM – Xwgp103-2B – 12.8 cM – Xbarc139-2B	SSR	Cheng and Chen (2010b)
Yr44 (YrZak)	XSTS7/8/***Yr5*** – 12.7 cM – ***Yr44*** – 3.9 cM – Xwgp100 – 1.1 cM– Xgwm501-2B	SSR,STS	Sui et al. (2009)
Yr45	Xbarc6-3D – 0.9 cM – Xwmc656-3D – 6.9 cM – Xwp118-3D – 4.8 cM – ***Yr45*** – 5.8 cM – Xwp115-3D	SSR	Li et al. (2010)
Yr46	Close linkage with Xcfd71-4D and Xbarc98-4D estimated at 4.4cM, and Xcfd23-4D at 5.2 cM (all on the same side of ***Yr46***) *Xgwm165-4D/Xgwm192-4D* – 0.4 cM – ***Yr46/Lr67*** *Pleiotropic with Lr37*	SSR	Hiebert et al. (2010) Herrera-Foessel et al. (2011)
Yr47	5 +/- 2 cM proximal to *Lr52*		{Bansal et al. 2011)
Yr48	Co-segregated with *Vrn2*, Be495011, Xcfa2149-5AL, Xgpw2181a-5AL, Xwmc74-5AL, and Xwmc410-5AL Xwmc727-5AL – 4.4 cM – ***Yr48*** – 0.3 cM – Xwms291-5AL	SSR	Lowe et al. (2011b)

APPENDIX 1 Continued

***Yr* gene**	**Mapping information[a] : genes and markers linked to *Yr* genes, flanking markers and genetics distance**	**Marker system[b]**	**References**
Yr49	*Xgpw7321-3D/**Yr49*** – 1 cM – *Xgwm161-3D*	SSR	McIntosh et al. (2011) Spielmeyer et al.,unpublished
YrAlp	Wwgp47 - 1.1 cM - ***YrAlp*** - 2.8 cM - Yr5 - 0.4 cM - 15.2 cM - Xwgp48 - 2.4cM - Xwgp49 - 1.9 cM - Xwgp50 - 4.9 cM - YrH52 - 2.8 cM - Xgwm18-1B - 1.1 cM - Xgwm11-1B	SSR, RGAP	Lin and Chen (2007a)
YrC591	Xcfa20-40-7B - 8.0 cM - ***YrC591*** - 11.7 cM - SC-P35M48	SSR, SCAR derived from AFLP	Li et al. (2009)
YrCK	***YrCK*** flanking markers Xgdm005(distal) and Xwmc190 (proximal).	SSR	Bariana et al. (2001)
YrC142	Located in the ***Yr24/Yr26*** region close to Xbarc187-1B and Xgwm273-1B	SSR	Wang et al. (2009)
YrExp1	Xwgp78-1B - 4.2 cM - ***YrExp1*** - 3.4 cM - Xwmc631-1D	SSR	Lin and Chen (2008)
YrExp2	Xgwm639-5B - 9.2 cM - Xwgp81-5B - 1 cM - ***YrExp2*** - 0.7 cM - Xwgp82-5B	SSR	Lin and Chen (2008)
YrH52	Yr15 - 9.6 cM - ***YrH52*** - 1.4 cM - Nor-1B - 0.8 cM - Xgwm264a - 0.6 cM - cM - Xgwm264a - 0.6 cM - Xgwm18, ***YrH52*** in interval Xgwm359b – 1 cM - P55M53b, Xgmw273a - 2.7 cM - ***YrH52*** - 1.3 cM - Xgwm413(Nor1and UBC212a)	SSR , RFLP, AFLP, RAPD	Peng et al. (1999), Peng et al. (2000)
YrMoro	S11M17 - 2.3 cM - *Lrk10* - 3.8 cM -***YrMoro/S26M47*** - 1 cM - S13M63, STS marker derived from AFLP fragment S26M47	RFLP,AFLP	Smith et al. (2002)
Yrns-B1	Xgwm493-3B -21 cM - ***Yrns-B1,*** ***Yrns-B1*** in interval Xgwm493-3B - 2.5 cM - Xgwm1329-3B	SSR	Borner et al. (2000), Khlestkina et al. (2007)
YrPS1	Xgwm429-2B – 1.8 cM – ***YrP81*** – 4.1 cM – Xwmc770-2B	SSR	Pu et al. (2010)
YrS2199	Xgwm120-2BL - 11.0 cM - ***YrS2199*** - 0.7 cM - Xdp269, ***YrS2199*** and *Yr5* are likely to be the same gene or allelic genes	SSR	Fang et al. (2008)
YrSpP	***YrSpP*** - 10.9 cM - Xwmc441-2B	SSR	Guan et al. (2005)
YrZH84	Xwmc276-7B - 0.6 cM - Xcfa2040-7B- 1.4cM - ***YrZH84*** - 4.8 cM - Xbarc32	SSR	Li et al. (2006b)

APPENDIX 2: List of QTLs for Yellow rust resistance published

Chr.	QTL name	Peak marker or QTL interval	Origin	Phenotype	R2	Type	*Suspected Yr gene*	Ref.
1A	Qyr.sun-1A	Xgwm164	Janz	APT	0.07	RS		e
1A	QYrid.ui-1A	X377889-XLMW1	IDO444	APT IT	0.09	HTAP		af
1A		Xfba118b	Recital	APT Sev AUDPC	0.09			g
1A	Qyr.sgi-1A	s15m19D-s23m18E	Kariega	APT Sev IT	0.06 - 0.12			u
1AL	QYrst.orr-1AL	wPt-4399	Stephens	APT IT	0.12			ac
1AL	QYr.caas-1AL	wPt-2406-Xwmc59	Naxos	APT Sev	0.08			w
1AL		wPT-6005-wPT-4709	Pastor	APT Sev IT	0.04			y
1B	Qyr.sun-1B	Xbarc80	Kukri	APT	0.04			e
1BL		Xgwm259	Pavon 76	APT IT	0.35		*Yr29*	ad
1BL	-	Xpsr305-P39/M38-2, P36/M36-1	CD87	APT	0.09	APR	*Yr29*	b
1BL	QPst.jic-1B	Xwmc735	Brigadier	APT	0.05			m
1BL	QYrex.	Xwmc631-Xwgp78	Express	APT AUDPC IF	0.09	HTAP		o
1BL	QPst.jic-1B	Xgwm818-Xgwm259	Guardian	APT Sev IT	0.22-0.45		*Yr29*	r
1BL		XP35/M55-LTN	Attila	APT Sev	0.12-0.17		*Yr29*	x
1BL	Yr46	csLV46-Xgwm818	Pastor	APT Sev IT	0.16		*Yr29*	y
1BL.1RS	QYr.caas-1BL.1RS	Xiag95	Shanghai	APT Sev	0.08			w
1BS		wPT-8168-wPT-6240	Pastor	APT Sev IT	0.05			y
1DS	QYr.caas-1DS	XUgwm353-Xgdm33b	Naxos	APT Sev	0.05	APR		w
2A	Qyr.sun-2A	117454	Kukri	APT IT	0.13	RS		e
2A	QYrtm.pau-2A	Xwmc407-Xwmc170	PAU14087(T. monococcum)	APT Sev and IT	0.14	APR		ag
2AL	QYR2	Gwm356–Gwm382	Camp Remy	APT Sev AUDPC	0.13			f
2AL		Xwmc 198a,Xwmc 170b,Xwmc 181	Solist and Wasmo	APT IT	0.4		*Yr32*	ah
2AL		Xgwm382c-XBarc122	Recital	APT Sev AUDPC	0.04			g
2AL	Qyr.inra-2AL	Xgwm382a-Xgwm359	Camp Remy	APT Sev AUDPC	0.26	APR		q
2AL		Xgpw4496	Taldor	APT Sev AUDPC	0.03			s
2AS	QYr.ufs-2A	wPt-733314-wPt0003	Cappelle-Desprez	APT Sev IT	0.37			a
2AS	QYrst.orr-2AS	wPt-0003	Stephens	APT IT	0.19			ac
2AS		Xwmc 407	Kris	APT IT	0.27		*Yr17*	ah
2AS	QYr.inra-2AS2	Xgwm512-Xgwm400b	Renan	APT Sev AUDPC	0.45	SDR	*Yr17*	g
2AS	QYr.inra-2AS1	XDupw210-Xcfd36	Recital	APT Sev AUDPC	0.09			g
2AS	QYr.osu-2A		Jagger	APT			*Yr17*	h
2AS	YrR61	Xbarc124-Xgwm359,	Pioneer 62R61	APT IT	0.56			k
2AS	QYr.ucw-2AS	wPt-5839	PI610750	APT Sev IT	0.02			p
2AS	QYr.inra-2AS	Xcfd36b-Xcnl127	Apache	APT Sev	0.24	SDR	*Yr17*	s

Appendix 2 continued

Chr.	QTL name	Peak marker or QTL interval	Origin	Phenotype	R2	Type	*Suspected Yr gene*	Ref.
				AUDPC				
2B	QYrid.ui-2B.1	wPt-9668-Xgwm429	IDO444	APT Sev and IT	0.13-0.25	HTAP		af
2B	QYrid.ui-2B.2	Xgwm429-Xbarc91	IDO444	APT Sev and IT	0.13-0.31	HTAP		af
2B	Qyr.sgi-2B.1	Xgwm148-s12m60A	Kariega	APT Sev IT	0.17 - 0.46			u
2B	Qyr.sgi-2B.2	Xpsp3030-s16m40A	Kariega	APT IT	0.07			u
2BL	-	P36/M43-5, P37/M53-1, P35/M38-5, P35/M37-4.	Katepwa	SDT	-	SDR	*Yr7*	b
2BL	-	P36/M49-4-P31/M50-1	Cranbrook	SDT	-	SDR	*Yr7*	c
2BL	QYR1	Gwm47–Gwm501	Camp Remy	APT Sev AUDPC	0.46		*Yr7*	f
2BL		Xwmc149, Xwmc317a	Deben	APT IT	0.21			ah
2BL	QYraq.cau-2BL	Xwmc175-Xwmc332	Aquilera	APT Stripe number	0.61			j
2BL	QYr.inra-2BL	Xwmc245-Xwmc175	Camp Remy	APT SDT	0.47	SDR	*Yr7*	q
2BL	QYr.inra-2BL	Xcfd267 -Xcfd73a	Apache	APT Sev AUDPC	0.52	SDR	*Yr7*	s
2BL	QTLAPR.2B	wPt-9190 -Xwmc175	Claire	APT Sev IT	0.08 - 0.24			t
2BL	QYr.caas-2BL	wPt-8460-wPt3755	Naxos	APT Sev	0.12			w
2BL		Xgwm619-Xgwm1027	Avocet S	APT Sev	0.06	RS		x
2BS	QYrst.orr-2BS.2	wPt-0408	Stephens	APT IT	0.11			ac
2BS	QYR3	Cdo405–bcd152	Opata85	APT Sev and AUDPC	0.31		*Yr27*	f
2BS	QYr.inra-2BS	Xfba70-Xfbb67c, Xgwm210a-Xfbb67c	Renan	APT Sev AUDPC	0.12			g
2BS	QYRlu.cau-2BS.1	Xwmc154-Xgwm148	Luke	APT IT	0.37	HTAP		j
2BS	QYrl.cau-2BS.2	Xgwm148-Xbarc167	Luke	APT IT	0.41	HTAP		j
2BS	QYrcaas-2BS	Xbarc13-Xbarc230	Pingyuan 50	APT Sev	0.05-0.09			n
2BS	QYr.ucw-2BS	wmc474	UC1110	APT Sev IT	0.05			p
2BS	Qyr.inra-2BS	Xgpw3032-Xcfd50a	Camp Remy	APT Sev AUDPC	0.475	APR		q
2BS		XP32/M62-XP88/M64	Attila	APT Sev	0.07		*Yr27*	x
2BS	Yr31	Yr31 (phenotype)	Pastor	APT Sev IT	0.16		*Yr31*	y
2D	QPst.jic-2D	Xgwm652-Xgwm692	Guardian	APT Sev IT	0.07-0.12			r
2DL		Xgwm349	Fukuhokomugi	APT IT	6.5 to 9.6			ab
2DL	QPst.jic-2DL	Xgwm320	Alcedo	APT SDT	0.37	SDR		m
2DL	QTLAPR.2Da	Xgwm301-Xwmc167	Claire	APT Sev IT	0.05 - 0.32		*Yr16*	t
2DL	QTLAPR.2Db	Xgwm539-EST22	Claire	APT Sev IT	0.08 - 0.23		*QTL Alcedo*	t
2DL	QYr.caas-2DL	wPt-6752-Xcfd47	Naxos	APT Sev	0.03		*QTL Guardian*	w
2DS	QYr.ufs-2D	Xgwm102-wPt-	Cappelle-	APT Sev IT	0.08		*Yr16*	a

Appendix 2 continued

Chr.	QTL name	Peak marker or QTL interval	Origin	Phenotype	R2	Type	*Suspected Yr gene*	Ref.
		664520	Desprez					
2DS	YrKat	Xwmc111-Xwmc025a	Katepwa	APT	0.12	APR		b
2DS	YrCK	Xgdm005-Xwmc190	Sunco	SDT	-	SDR temperature sensitive		d
2DS	QYr.inra-2DS	Xgwm102-gwm539	Camp Remy	APT Sev AUDPC	0.4	APR	*YR16*	q
3A		wPT-6422-wPT-7890	Avocet	APT Sev IT	0.02			y
3B	QYrid.ui-3B.1	X345897-wPt-3921	IDO444	APT IT	0.1	HTAP		af
3B	QYrid.ui-3B.2	X379646-Xgwm299	IDO444	APT Sev	0.09	HTAP		af
3B		wPT-2458-wPT-0036	Pastor	APT Sev IT	0.05		*Yr30*	y
3Bc		Xgwm131b-Xbcd131	Renan	APT Sev AUDPC	0.06			g
3BL	QYrex.wgp-3BL	Xgwm299-Xwgp66	Express	APT AUDPC IF	0.27	HTAP		o
3BS		Xgwm389	Oligoculm	APT IT	0.05-0.24		*Yr30*	ab
3BS		XPstAATMseCAC2	Pavon 76	APT IT	0.06		*Yr30*	ad
3BS	QYrAlt.syau	Xgwm389-Xbarc238	Alturas	APT IT	0.34-0.50	HTAP		ae
3BS	Qyr.sun-3B	wPt-6802	Kukri	APT IT	0.05			e
3BS	QYr.inra-3BS	Xgwm533	Renan	APT Sev AUDPC	0.07			g
3BS	QYr.uga-3BS.1	wPt-2557–Xbarc133	AGS 2000	APT IT	0.05			k
3BS	QYr.uga-3BS.2	wPt-730063–wPt-9579	AGS 2000	APT IT	0.07			k
3BS	QYr.uga-3BS.3	wPt-1612–wPt-7486	AGS 2000	APT IT	0.05			k
3BS	QYr.ucw-3BS	gwm533.1	UC1110	APT Sev IT	0.22			p
3D	QYR6	Cdo407–ksuA6	Opata85	APT Sev and AUDPC	0.12			f
3DS		Xgwm456-Xgwm55b	Recital	APT Sev AUDPC	0.08			g
3DS		XBarc125-Xgwm456a	Recital	APT Sev AUDPC	0.12			g
4A	QYrrb.ui-4A	wpt-2983-wPt-8275	Rio Blanco	APT Sev and IT	0.16-0.22	HTAP		af
4A	Qyr.sgi-4A.1	s21m40A-s22m55A	Kariega	APT Sev IT	0.08 - 0.24			u
4A	Qyr.sgi-4A.2	s13m94B-s18m47B	Avocet S	APT IT	0.24			u
4B	QYr.ufs.4B	Xgwm165-Xgwm495	Palmiet	APT Sev IT	0.09			a
4B	QYrrb.ui-4B	Xgwm165-Xgwm495	Rio Blanco	APT IT	0.07	HTAP		f
4B	QPst.jic-4B	Xgwm539-Xgwm349	Guardian	APT Sev IT	0.08 - 0.14			r
4B	QYr.inra-4B	Xgwm6	Apache	APT Sev AUDPC	0.15	APR		s
4BL		Xgwm538	Oligoculm	APT IT	0.02-0.12			ab
4BL	QYrpl.orr-4BL	312980	Platte	APT IT	0.09			ac
4BL		Xgwm495	Avocet S	APT IT	0.09			ad
4BL	QPst.jic-4BL	Xwmc692	Alcedo	APT	0.11			m
4BS	QYrst.orr-4BS	wPt-5265	Stephens	APT IT	0.11			ac

Appendix 2 continued

Chr.	QTL name	Peak marker or QTL interval	Origin	Phenotype	R2	Type	*Suspected Yr gene*	Ref.
4DL		Xwmc399	Oligoculm	APT IT	0.02-0.08			ab
4DL	QYr.caas-4DL	Xwmc331-Xgwm165	Bainong 64	APT Sev	0.08	APR	*Yr46*	v
4DS		wPT-4572-wPT-6880	Pastor	APT Sev IT	0.04			y
5A	QYR5	Fbb209–abg391	Opata85	APT Sev and AUDPC	0.15			f
5A	QYrtb.pau-5A	Xbarc151-Xcfd12	PAU5088 (*T. boeoticum*)	APT Sev and IT	0.24	APR		ag
5A	QPst.jic-5A	Xwmc752-Xgwm786	Brigadier	APT	0.04			m
5AL	QYr.osu-5A		Jagger	APT				h
5AL	QYr.caas-5AL	Xwmc410 -Xbarc261	Pingyuan 50	APT Sev	0.05-0.20			n
5AL	Yr48	cfa2149	PI610750	APT Sev IT	0.1		*Yr48*	p
5AL	QYr.caas-5AL.2	wPt-19035-AL-Xwmc757-5AL	Shanghai 3/Catbird	APT Sev	0.03			w
5AL		wPT-0837-wPT-5231	Pastor	APT Sev IT	0.05			y
5B	QYr.ufs-5B	wPt-7114-Xbarc74	Cappelle-Desprez	APT Sev IT	0.05			a
5B	Qyr.sun-5B	wPt-3030	Janz	APT IT	0.07			e
5B	QYrid.ui-5B	X63541-Xbarc59	IDO444	APT IT	0.07	HTAP		af
5B	QYr-term-5B.2	Xwmc604-Xwmc253	Flinor	SDT	0.33	SDR temperature sensitive		i
5B	QYr.uga-5B	wPt-665267–Xgdm152	AGS 2000	APT IT	0.05			k
5BL		Xwmc415	Oligoculm	APT IT	0.02-0.16			ab
5BL	QYr-term-5B.1	Xbarc89-Xgwm67	Flinor	SDT	0.37	SDR temperature sensitive		i
5BL	QYr.inra-5BL.1	Xgwm499-Xgwm639c	Camp Remy	APT Sev AUDPC	0.235	APR		q
5BL	QYr.inra-5BL.2	Xgwm604-Xgpw1082	Camp Remy	APT Sev AUDPC	0.315	APR		q
5BL	QYr.caas-5BL.3	wPt-2707-Xbarc275	Shanghai 3/Catbird	APT Sev	0.05			w
5DL		Xgwm583	Octane	APT Sev IT	0.6			l
6AL	QYrpl.orr-6AL	378849	Platte	APT IT	0.06			ac
6AL		Xgwm617	Avocet S	APT IT	0.08			ad
6AS	QYr.uga-6AS	wPt-671561–wPt-7840	Pioneeer 62R61	APT IT	0.07	HTAP		k
6AS	QYrex.wgp-6AS	Xgwm334-Xwgp56	Express	APT AUDPC IF	0.32	HTAP		o
6AS		wPT-2573-wPT-0959	Avocet	APT IT	0.02			y
6B	Qyr.sun-6B	wPt-8183	Janz	APT	0.05			e
6B	QYr.inra-6B	Xcdo270-Xgwm193	Renan	APT Sev AUDPC	0.04			g
6BL		XPstAGGMseCGA1-Xgwm58	Pavon 76	APT IT	0.14			ad
6BL		Xwmc397, Xwmc105b, Xwmc341	Wasmo, Deben, Kris	APT IT	0.29 to 0.25			ah
6BL		wPT-6329-wPT-5176	Pastor	APT IT	0.03			y
6BS		Xgwm935.1	Oligoculm	APT IT	0.04			ab

Appendix 2 continued

Chr.	QTL name	Peak marker or QTL interval	Origin	Phenotype	R2	Type	*Suspected Yr gene*	Ref.
6BS	QYr.caas-6BS	Xgwm361-Xbarc136	Pingyuan 50	APT Sev	0.05-0.08			n
6BS	QYr.caas-6BS.3	Xwmc487-Xcfd13	Bainong 64	APT Sev	0.05	APR		v
6BS	QYr.caas-6BS.2	Xwmc104-wPt-0259	Naxos	APT Sev	0.04			w
6BS	QYrst.wgp-6BS.	Xbarc10 1-Xbarc136	Stephens	APT IT	0.32-0.45	HTAP		z
6BS	QYrst.wgp-6BS.2	Xgwm132-Xgdm113	Stephens	APT IT	0.25-0.43	HTAP		z
6DL	QYr.ufs-6D	Xgwm325-Xbarc175	Cappelle-Desprez	APT Sev IT	0.06			a
6DL	QTR7	Bcd1510–ksuD27	W-7984	APT Sev and AUDPC	0.13			f
7A		Xbcd129b-Xfba127c	Recital	APT Sev AUDPC	0.08			g
7A	Qyr.sgi-7A	s19m89C-s18m47B	Kariega	APT IT	0.11			u
7AL		wPT-2260-wPT-2501	Avocet	APT IT	0.03			y
7AS	QYrst.orr-7AS	wPt-4319	Stephens	APT IT	0.15			ac
7AS	QYr.caas-7AS	Xbarc127-Xbarc174	Jingshuang 16	APT Sev	0.06	APR		v
7AS		wPT-4172-wPT-8149	Avocet	APT IT	0.02			y
7B	Qyr.sun-7B	wPt-3723	Kukri	APT	0.09			e
7BL		Xwmc166	Oligoculm	APT IT	0.02-0.09			ab
7BL		Xgwm611	Tiritea	APT Sev IT	0.42 to 0.26			l
7BL	QTLAPR.7B	wPt-1069-wPt-9925	Claire	APT Sev IT	0.07 - 0.13			t
7BL	QYr.caas-7BL.1	wPt-8106-Xbarc176	Shanghai 3/Catbird	APT Sev	0.09			w
7BL	QYr.caas-7BL.2	Xgwm577-wPt-4300	Shanghai 3/Catbird	APT Sev	0.06			w
7BL		XP32/M59-Xgwm344	Attila	APT Sev	0.03			x
7BL		wPT-3190-wPT-1475	Pastor	APT IT	0.07			y
7BS		Xgwm935.3	Oligoculm	APT IT	0.01-0.05			ab
7D	Qyr.sun-7D	wPt-3328	Janz	APT IT	0.12		*Yr18*	e
7D	Qyr.sgi-7D	Xgwm295-Ltn	Kariega	APT Sev IT	0.09 - 0.29	APR	*Yr18*	u
7DS	QTL-7DS	Xgwm295.1	Fukuhokomugi	APT IT	0.11-0.24		*Yr18*	ab
7DS		P36/M41-2-Xwmc405b	CD87	APT	0.15	APR	*Yr18*	b
7DS	QTR4	Wg834–bcd1438	Opata85	APT Sev and AUDPC	0.14		*Yr18*	f
7DS		Xgwm44	Octane	APT Sev IT	0.13 to 0.23		*Yr18*	l

Chr. chromosome, Ref. Reference for the QTL and mapping population: (a) Yr16DH70 (Cappelle Desprez derived line) x Palmiet RIL pop. (Agenbag et al., 2012), (ab) Fukuho-komugi × Israeli wheat Oligoculm DH pop. (Suenaga et al., 2003), (ac) Stephens x Platte RIL pop. (Dolores Vazquez et al., 2012), (ad) Avocet S x Pavon 76 RIL pop. (William et al., 2006), (ae) Alturas x Taichung 29F1,F2,F3 pop. (Zhao et al., 2012), (af) Rio Blanco x IDO444 RIL pop. (Chen et al., 2012), (ag) PAU14087 x PAU5088 RIL pop. (Chhuneja et

al., 2008), (ah) Kris x Deben, Krisx Wasmo, Solist x Deben DH pop. . Christiansen et al. (2006), (b) CD87 x Katepwa DH pop. (Bariana et al., 2001), (c) Cranbrook x Halberd DH pop. (Bariana et al., 2001), (d) Sunco x Tasman DH pop. (Bariana et al., 2001), (e) Kukri x Janz DH pop. (Bariana et al., 2010), (f) ITMI(Opata85 × synthetic W-7984) and Camp Remy × Michigan amber RIL pop. (Boukhatem et al., 2002), (g) Renan x Recital RIL pop. (Dedryver et al., 2009), (h) Jagger x 2174 RIL pop. (Fang et al., 2011), (i) Flinor x Ming Xian 169 F1, F3 pop. (Feng et al., 2011), (j) Luke x Aquilera F2,F3 pop. (Guo et al., 2008), (k) Pioneer 26R61 x AGS 2000 RIL pop. (Hao et al., 2011), (l) Octane x Tiritea DH pop. (Imtiaz et al., 2004), (m) Alcedo x Brigadier DH pop. . (Jagger et al., 2011), (n) Pingyuan 50 x Mingxian 169 DH pop. (Lan et al., 2010), (o) Express x Avocet S RIL pop. (Lin and Chen, 2009), (p) UC1110 x PI 610750 RIL pop. (Lowe et al., 2011b), (q) Camp Remy × Recital RIL pop. (Mallard et al., 2005), (r) Guardian x Avocet S F2, F3 pop. (Melichar et al., 2008), (s) Apache x Taldor DH pop. (Paillard et al., 2012), (t) Claire x Lemhi DH pop. . (Powell, 2010), (u) Kariega x Avocet S DH pop. . (Ramburan et al., 2004), (v) Bainong 64 x Jingshuang 16 DH pop. (Ren et al., 2012c), (w) Naxos x Shanghai 3/Catbird RIL pop. (Ren et al., 2012b), (x) Attila x Avocet S RIL pop. (Rosewarne et al., 2008), (y) Avocet S x Pastor RIL pop. (Rosewarne et al., 2012), (z) Stephens x Michigan Amber RIL pop. (Santra et al., 2008),

APPENDIX 3: Statistical analysis of historical data (Genstat output)

Output from Genstat obtained from model III.1 (test replicate)

- Model

  Variate: LOG10(x+1)

  Fixed terms: variety+trial+ trial.replicate

  Data point: 17248

- Residual variance model

Term	Estimate	s.e.
Residual	0.0933	0.00103

- Tests for fixed effects

Fixed term	Wald statistic	n.d.f.	F statistic	F probability
Variety	24325.54	438	55.54	<0.001
Trial	5169.7	204	25.34	<0.001
Replicate within trial	108.82	205	0.53	1

Output from Genstat obtained from model III.5 on the entire data set

- Model

  Variate: LOG10(x+1)

  Rom term:

  variety+year+variety.year+variety.isolate+variety.layout+year.isolate+year.layout+isolate.layout

  Data point: 12907

- Estimated variance components

Rom term	Component	S.e.
Variety	0.08781	0.00740
Year	0.00750	0.00449
Variety.year	0.02897	0.00183
Variety.isolate	0.02897	0.00099
Variety.layout	0.00777	0.00128
Year.isolate	0.00766	0.00117
Year.layout	0.00634	0.00279
Isolate.layout	0.00276	0.00097

- Residual variance model

Term	Estimate	s.e.
Residual	0.0277	0.00073

- Deviance:-2*Log-Likelihood

Deviance	d.f
-21178.06	12897

APPENDIX 4: Summary yellow rust evaluations on the YR panel lines genotype for *Rht1*, *Rht2, Ppd-D1*, 1BL.1RS translocation, *Yr17* (introgressed fragment 2NS.2AS)

The *Yr* gene postulations come from the following publications : (a) (Pathan et al., 2008), (b) (Singh et al., 2008), (c) (Hovmøller, 2007), (d) (Roelfs and Bushnell, 1985), (e) Wheat yellow rust UKCPVS reports 1989 to 2010, (f) (Hovmøller, 2001b), (g) (Mallard et al., 2005), (h) (Lewis, 2006), (i) (Johnson, 1992a), (j) (Bayles, 2001), (k) (Eriksen et al., 2004), (l) (McIntosh et al., 1995).

* Alchemy was wrongly diagnosed with the translocation 1BL.1RS using de Froidmont (1998) marker.

** the presence of the introgressed segment 2NS based on DArT markers is indicated in parenthesis to complement the SCAR marker genotype (Robert et al., 1999) or when contradictions were observed with DArT markers.

YR Panel line	Years in RL	Yellow rust HGCA scores Min-Max	Translocation 1BS or 1RS	Introgression 2NS.2AS with *Yr17***	Rht1(Rht-B1a/b)	Rht2(Rht-D1a/b)	Ppd-D1(no deletion)/Ppd-D1a	Infection score from historical data (adj.)	IF SDT Brock (adj.)	IF SDT Solstice (adj.)	AUDPCr 2010 (adj.)	AUDPCr 2011 (adj.)	Max severity 2010 (adj.)	Max severity 2011 (adj.)	Max HostR 2011 (adj.)	HostR 2011 end (adj.)	*Yr* genes postulated
Aarden			1B	? (Yr17)	Rht-B1a	?	Ppd-D1	3.7	2	-	-	0.1	-	12.4	-	0.5	
Aardvark			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.8	3	3.6	0.02	0.01	0.9	0	0.3	0.1	
Abbot	1997-1999	6-6	1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.3	3.5	6.3	0.28	0.11	54.6	10	0.7	0.6	*Yr17 (a)*
Abele			1R(Yr9)	?(no Yr17)	Rht-B1a	Rht-D1a	Ppd-D1	-	-	3.4	0.15	-	25	-	0.6	-	
Ac Barrie			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	15.2	6.4	7.4	1.01	1.12	89.3	92.9	0.9	0.7	
Access	2002-2007	3-4	1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	5.9	0.6	3.6	0.38	0.32	63.5	40.2	0.7	0.6	
Acclaim			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.9	1.1	7.6	0.44	0.28	59	34.1	0.6	0.6	
Admiral	1992-1995	4-4	1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	3	1.5	3.1	0.31	0.16	44.1	19.5	0.5	0.5	*Yr9(b)*
Adroit			1B	No Yr17	Rht-B1a	?	Ppd-D1	1.1	-	6.5	-	-	-	-	-	-	
Agami			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	3.6	1.2	8.3	0.82	0.58	87.2	65.3	1	0.6	
Alchemist			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	3.2	1.1	6.9	0.47	0.27	50.7	37.4	0.7	0.6	

YR Panel line	Years in RL	Yellow rust HGCA scores Min-Max	Translocation 1BS or 1RS	Introgression 2NS.2AS with *Yr17***	Rht1(Rht-B1a/b)	Rht2(Rht-D1a/b)	Ppd-D1(no deletion)/Ppd-D1a	Infection score from historical data (adj.)	IF SDT Brock (adj.)	IF SDT Solstice (adj.)	AUDPCr 2010 (adj.)	AUDPCr 2011 (adj.)	Max severity 2010 (adj.)	Max severity 2011 (adj.)	Max HostR 2011 (adj.)	HostR 2011 end (adj.)	*Yr* genes postulated
Alchemy	2006-2012	8.4-9	1B*	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.1	4.9	2.3	0.01	0.02	0	1.3	0.2	0.3	
Alsace			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.3	2	6	0.24	0.15	26.8	10	0.5	0.5	
Ambrosia	2005-2010	5-6	1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.4	2.9	6.7	0.35	0.32	52.9	34.8	0.7	0.6	
ante			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.4	3.5	8.6	0.25	0.11	63.2	6.3	0.7	0.3	*Yr17(b)*
Anglo			1B	Yr17	Rht-B1a	?	Ppd-D1	2.7	1.5	6.4	0.11	0.09	11.9	15.6	0.5	0.6	
Anvil			1B	?(no Yr17)	Rht-B1a+b	Rht-D1a+b	Ppd-D1	-	-	5.5	0.02	-	3.2	-	0.4	-	
Apollo	1988-1994	3-8	1R(Yr9)	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	2.6	1.2	7.2	0.79	0.18	84.4	28.1	0.8	0.6	*Yr9 (a,b)*
Apostle			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.2	1.5	1.9	0.01	0.01	0	0	0.2	0.1	
Aristocrat			1B	Yr17	Rht-B1a	Rht-D1a	Ppd-D1	0.8	4	4.9	-	0.02	-	3.2	-	0.6	
Ark			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.1	7.4	4.1	0.01	0.01	0	0	0.2	0.1	
Arlington			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.2	0.6	7.9	0.34	0.24	49.8	20.3	0.6	0.5	
Armada	1978-1985	4-7	1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	0.6	7.9	6.2	0.01	0.01	0.9	1	0.4	0.3	*Yr12,Yr27(a)*
Arminda			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	0.4	2.5	3.8	0.02	0.01	1.3	0	0.3	0	*Yr13(d)*
Arran			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.1	1.1	3.4	0.01	0.01	0.3	0	0.2	0	
Arriva			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.9	1.5	4.8	0.22	0.17	47.4	20.3	0.6	0.6	
Asagai			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.1	0.1	3.6	0.15	0.01	9.6	0	0.4	0.2	
Ashanti			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1	0.6	3.1	0.02	0.04	3.8	4.1	0.4	0.4	
Astron			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	5.8	-	6.7	0.31	-	54	-	0.6	-	
Atla			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.6	0.6	6.4	0.08	0.15	12.8	19.5	0.5	0.5	
Atlanta			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.4	1.1	6.2	0.29	0.1	57.2	22.2	0.7	0.6	
Atoll			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	3.2	4.9	6.9	0.28	0.16	50.9	17.8	0.6	0.5	
Atou	1973-1979	8-8	1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	2	4.9	6.9	0.39	0.09	63.2	7.9	0.6	0.5	*Yr3, Yr16(d)*
Avalon	1980-1992	4-8	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.4	7.4	4.5	0.34	0.22	63.2	28.9	0.7	0.5	*Yr4, Yr14(e)*

YR Panel line	Years in RL	Yellow rust HGCA scores Min-Max	Translocation 1BS or 1RS	Introgression 2NS.2AS with *Yr17***	Rht1(Rht-B1a/b)	Rht2(Rht-D1a/b)	Ppd-D1(no deletion)/Ppd-D1a	Infection score from historical data (adj.)	IF SDT Brock (adj.)	IF SDT Solstice (adj.)	AUDPCr 2010 (adj.)	AUDPCr 2011 (adj.)	Max severity 2010 (adj.)	Max severity 2011 (adj.)	Max HostR 2011 (adj.)	HostR 2011 end (adj.)	*Yr* genes postulated
Award			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.3	8.4	6.9	0.1	0.13	16.6	19.5	0.4	0.5	
Axial			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1a	2.9	3.5	7.4	0.6	0.35	93.3	46.8	1	0.7	
Axona	1985-2000	7-9	1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	0.7	2	4.5	0.05	0.05	17.1	1.2	0.4	0.3	
Baron			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	-	-	7.4	0.29	-	44.9	-	0.6	-	
Battalion	2007-2012	6-8	1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.5	1.5	2.4	0.23	0.07	48	12.4	0.7	0.5	
Beaufort	1995-1998	5-9	1R(Yr9)	Yr17	Rht-B1a	Rht-D1a	Ppd-D1	1.4	-	5.5	0.44	0.01	57.8	0.6	0.8	0.4	*Yr9, Yr17(a,b)*
Beaver	1990-1995	3-4	1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	4.1	0.6	6.4	0.38	0.16	46	17.8	0.7	0.5	*Yr9(b)*
Belter			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.8	3.5	5.5	0.24	0.05	38.6	10	0.5	0.6	
Benedict			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.5	0.6	3.8	0.01	0.15	0	25.3	0.2	0.6	
Bentley			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	3.6	4.9	6.7	0.33	0.41	47.8	46.8	0.6	0.6	
Biscay	2001-2003	4-5	1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	4.2	3	4.1	0.39	0.17	46.2	29.9	0.7	0.6	*Yr2,Yr3,Yr17(c)*
Blaze			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	8.5	1.1	6.4	0.5	0.45	63.5	39.7	0.5	0.6	
Bogart			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.9	-	-	-	-	-	-	-	-	
Boston			1B	No Yr17	Rht-B1b	Rht-D1a	Ppd-D1	0.3	1.5	1.2	0.02	0.01	0.1	0	0.2	0	*Yr15(c)*
Bounty	1979-1983	7-8	1B	?(no Yr17)	Rht-B1a+b	Rht-D1b	Ppd-D1	-	-	2.6	0.03	-	6.9	-	0.4	-	
Bouquet	1972-1980	8-8	1B	?	Rht-B1a	Rht-D1a	Ppd-D1	-	-	5.7	0.25	-	50.7	-	0.5	-	*Yr3, Yr14,Yr16?(d)*
Boxer			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	0.2	1.5	4	0.04	0.01	4.2	0.6	0.4	0.3	
Bro			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.7	1.5	5.7	0.24	0.21	38	29.9	0.6	0.5	
Brigadier	1993-1999	1-9	1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	7	0.6	4.3	0.74	0.54	84.6	65.3	0.8	0.7	*Yr9, Yr17(f)*
Brig	1979-1988	4-7	1B	?(no Yr17)	Rht-B1a+b	Rht-D1b	Ppd-D1	-	-	4.5	0.25	-	50.3	-	0.6	-	
Brock	1985-1991	2-8	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.2	8.4	1.9	0.01	0.04	0	2.6	0.2	0.4	*Yr7,Yr13(e)*
Broiler			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.5	2.5	2.6	0.11	0.05	23.6	4.1	0.5	0.6	

YR Panel line	Years in RL	Yellow rust HGCA scores Min-Max	Translocation 1BS or 1RS	Introgression 2NS.2AS with *Yr17***	Rht1(Rht-B1a/b)	Rht2(Rht-D1a/b)	Ppd-D1(no deletion)/Ppd-D1a	Infection score from historical data (adj.)	IF SDT Brock (adj.)	IF SDT Solstice (adj.)	AUDPCr 2010 (adj.)	AUDPCr 2011 (adj.)	Max severity 2010 (adj.)	Max severity 2011 (adj.)	Max HostR 2011 (adj.)	HostR 2011 end (adj.)	*Yr* genes postulated
Brompton	2005-2008	8-9	1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.2	1.1	7.2	0.05	0.1	10.7	10	0.4	0.4	
Brunel			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.2	1.1	8.1	0.33	0.1	33.4	11.5	0.7	0.5	
Bryden			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.9	1.1	3.3	-	0.01	-	0	-	0.1	
Buccaneer			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	5.2	8.4	7.9	0.41	0.24	69.2	20.3	0.8	0.6	
Buchan	1999-2003	3-4	1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	9.6	0.6	3.8	0.38	0.3	42.4	37.4	0.7	0.6	
Buster	1995-2000	9-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.2	0.6	2.4	0.05	0.01	3.4	0	0.4	0	*Yr1(c)/YrHVII(b)*
Cadenza	1994-1998	8-9	1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	0.2	2	2.1	0.01	0.01	0	0	0.2	0	*Yr7(b)/Yr6,Yr7(a)*
Camp Remy			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	2.8	7.9	2.2	0.01	0.01	0	0.2	0.2	0.2	*Yr7(g)*
Cantata			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.6	4.9	2.2	0.12	0.06	22.7	10	0.4	0.6	
Canterbury			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	4.9	1.5	5.7	0.26	0.13	30.5	22.2	0.6	0.6	
Caphorn			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1a	4.2	1.5	6.7	0.52	0.17	69.2	22.2	0.8	0.6	
Capnor			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	3.4	1.5	6.2	0.26	0.49	38.7	57.4	0.8	0.6	
Cappelle Desprez	1967 or before-1976	3-5	1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	-	7.4	6.7	0.28	0.08	60.1	7.9	0.6	0.5	*Yr3,Yr16(d)/Yr2,Yr3,Yr25(h)*
Caprimus			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	0.8	2.5	4.8	0.39	0.08	44.7	11.5	0.8	0.7	
Carlton			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	3.4	1.1	7.4	0.64	0.23	81.3	26.3	0.9	0.6	
Carstens V			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	1.7	8.4	7.2	0.22	0.3	50.9	28.1	0.7	0.6	*(Yr3,Yr4),Yr25,Yr32,YrSD(h)*
Caxton	1996-1998	7-8	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1	4	6.9	0.2	0.06	45.4	5.2	0.5	0.5	*Yr9,Yr17(b)*
Chardonnay			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	4	1.1	6.9	0.39	0.26	60.1	31.7	0.8	0.6	
Charger	1997-2004	7-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.3	3	6.7	0.2	0.16	33.4	22.2	0.7	0.5	*Yr6(b)/Yr3,Yr6,Yr32(c)*
Chatsworth			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.1	2	4.5	0.02	0.01	5.2	0	0.3	0	
Chaucer			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.5	3	7.4	0.68	0.86	88.6	79.6	0.7	0.8	
Chequer			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.6	1.1	5.5	0.4	0.27	66.4	31.7	0.8	0.5	
Chester			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.5	1.1	4.1	0.05	0.02	4	1.9	0.3	0.4	

YR Panel line	Years in RL	Yellow rust HGCA scores Min-Max	Translocation 1BS or 1RS	Introgression 2NS.2AS with *Yr17***	Rht1(Rht-B1a/b)	Rht2(Rht-D1a/b)	Ppd-D1(no deletion)/Ppd-D1a	Infection score from historical data (adj.)	IF SDT Brock (adj.)	IF SDT Solstice (adj.)	AUDPCr 2010 (adj.)	AUDPCr 2011 (adj.)	Max severity 2010 (adj.)	Max severity 2011 (adj.)	Max HostR 2011 (adj.)	HostR 2011 end (adj.)	*Yr* genes postulated
Chianti			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.7	1.5	6.9	0.54	0.29	62.7	47.4	0.8	0.6	*Yr17(b)*
Chicago			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.5	2	6.7	0.05	0.03	5.2	1	0.4	0.5	
Choice			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.8	2	3.1	0.14	0.14	25.8	16.9	0.6	0.6	
Claire	1999-2012	8.5-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.1	4.5	3.4	0.05	0.01	3.9	0.2	0.3	0.3	*Yr2,(Yr3), Yr4, Yr25, YrHVII(h)*
Clement			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	17.6	1.1	7.6	0.82	0.37	90.3	68.7	1	0.7	*Yr2, Yr9(i)*
Clove			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.4	1.1	4.5	0.28	0.3	47.4	27.8	0.6	0.5	
Comet			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.2	0.6	7	0.07	0.09	35.4	12.6	0.6	0.5	*Yr3,Yr6,Yr9,Yr32(c)*
Commodore			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.9	1.1	5.7	-	0.15	-	17.8	-	0.5	
Conqueror	2011-2012	5-6.5	1B	Yr17	Rht-B1b	Rht-D1a	Ppd-D1	3.9	8.4	7.4	-	0.06	-	3.2	-	0.3	
Consort	1995-2009	5-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.9	2.5	6.7	0.27	0.13	53.5	15.6	0.6	0.6	*Yr32 (l)*
Contender			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.3	0.6	3.8	0.02	0.01	0	0	0.2	0	
Context			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.8	1.5	4.8	0.14	0.03	30.5	3.2	0.6	0.5	
Convoy			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.6	2	6.7	0.07	0.07	11.8	10	0.4	0.4	
Copain	1980-1981	3-3	1B	?(no Yr17)	Rht-B1a	Rht-D1a	Ppd-D1	-	-	8.1	0.15	-	35.4	-	0.5	-	
Cordiale	2004-2012	5-8	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.9	7.4	1.7	0.01	0.12	0.9	17.8	0.4	0.6	
Cranley			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.3	1.1	-	-	0.07	-	6.3	-	0.4	
Crofter			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.4	1.5	5.7	0.2	0.05	11.9	1	0.4	0.4	
Cyber			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	2.3	2	1.7	0.01	0.01	0	0	0.2	0	
Dart			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.7	1.1	6.4	0.15	0.01	25.8	0.6	0.6	0.4	
Datum			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.2	0.1	6.9	-	0.04	-	2.5	-	0.4	
Dean			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.7	1.1	5.5	0.06	0.02	9.4	0.2	0.4	0.3	
Deben	2001-2009	9-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.1	6.9	4.8	0.01	0.01	1.7	0.2	0.3	0.1	
Defender			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.9	3.5	3.8	0.05	0.05	2.7	3.2	0.4	0.5	

YR Panel line	Years in RL	Yellow rust HGCA scores Min-Max	Translocation 1BS or 1RS	Introgression 2NS.2AS with *Yr17***	Rht1(Rht-B1a/b)	Rht2(Rht-D1a/b)	Ppd-D1(no deletion)/Ppd-D1a	Infection score from historical data (adj.)	IF SDT Brock (adj.)	IF SDT Solstice (adj.)	AUDPCr 2010 (adj.)	AUDPCr 2011 (adj.)	Max severity 2010 (adj.)	Max severity 2011 (adj.)	Max HostR 2011 (adj.)	HostR 2011 end (adj.)	*Yr* genes postulated
Denver			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.4	2	6.4	0.48	0.48	66.4	65.3	0.8	0.7	
Derwent			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.4	8.4	7.4	0.33	0.26	50.3	28.9	0.6	0.5	
Diablo			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	2.5	0.6	7.3	-	0.7	-	71.7	-	0.8	
Dickins			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.5	3	6.1	-	0.02	-	0.6	-	0.3	
Dickson	2004-2005	4-4	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.7	2	6.7	0.41	0.24	55.1	29.9	0.8	0.7	
Director			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.1	0.6	2.2	0.15	0.01	16.6	0	0.4	0.1	
Dorial			1R(Yr9)	No Yr17	Rht-B1a+b	Rht-D1b	Ppd-D1	0.1	0.1	7.9	0.39	0.23	48.2	25.3	0.7	0.6	
Dover			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.6	2.5	2.4	-	0.03	-	0.6	-	0.3	
Drake			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.5	1.1	6.4	0.26	0.15	34.6	17.8	0.6	0.5	
Duxford	2008-2012	4.7-8	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.8	2	2.4	0.18	0.18	45.5	27.8	0.7	0.5	
Dynamo	1995-1997	9-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.4	6.4	3.6	0.02	0.01	1.1	0.6	0.2	0.3	*Yr3,Yr4,Yr25(h)*
Eclipse			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.8	2	6	0.16	0.08	20.7	15.6	0.5	0.5	
Einstein	2003-2012	5-7	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.7	3	5.5	0.29	0.15	44.7	17.8	0.6	0.6	
Ekla			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	2.2	7.4	1.9	0.01	0.01	0	0	0.2	0.1	
Electron			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.1	3.5	6.2	0.24	0.01	19.5	0.6	0.4	0.3	
Encore	1995-1997	8-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.7	1.1	5	0.13	0.15	32.3	23.8	0.7	0.6	*Yr9(a)/Yr3,Yr6,Yr9,Yr32(b)*
Equator			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.2	1.1	5.5	0.18	0.06	25	6.3	0.5	0.5	
Equinox	1997-2004	4-6	1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.5	0.6	4.1	0.21	0.11	32.3	15.6	0.5	0.6	*Yr9,Yr17(a)/Yr6,Yr9,Yr17(c)*
Estica			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	0.7	1.1	2.6	0.04	0.01	16.6	0.2	0.4	0.2	
Exeter			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.3	6.4	4.8	0.02	0.01	2.3	0.6	0.3	0.2	
Explosiv			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.3	3	7.4	0.21	0.06	36.7	12.4	0.5	0.4	
Exsept			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.3	2.5	6.7	0.2	0.03	30.5	3.2	0.5	0.4	
Extend			1B	No Yr17	?	Rht-D1b	Ppd-D1	2.6	7.9	7.2	0.29	0.18	50.3	31.7	0.7	0.6	

YR Panel line	Years in RL	Yellow rust HGCA scores Min-Max	Translocation 1BS or 1RS	Introgression 2NS.2AS with *Yr17***	Rht1(Rht-B1a/b)	Rht2(Rht-D1a/b)	Ppd-D1(no deletion)/Ppd-D1a	Infection score from historical data (adj.)	IF SDT Brock (adj.)	IF SDT Solstice (adj.)	AUDPCr 2010 (adj.)	AUDPCr 2011 (adj.)	Max severity 2010 (adj.)	Max severity 2011 (adj.)	Max HostR 2011 (adj.)	HostR 2011 end (adj.)	*Yr* genes postulated
Falstaff			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.3	1.1	6.2	0.25	0.16	28.3	17.8	0.5	0.5	
Fastnet			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.4	0.6	2.2	0.02	0.01	2	0	0.2	0.1	
Feast			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	3.6	0	6.7	0.56	0.3	64.5	36.1	0.8	0.6	
Fenda			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.4	7.4	6.5	-	0.01	-	0.6	-	0.3	
Fender			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.2	4	2.5	0.01	0.01	0	0	0.2	0.1	
Fielder			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.7	0.1	4.1	0.31	0.21	50.7	24.1	0.7	0.5	
Flair			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	9.4	8.8	8.6	0.69	0.39	77.4	29.9	0.6	0.6	*Yr1(c)*
Flame	1994-1996	8-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.4	3.5	2.4	0.04	0.01	20	0.2	0.4	0.2	*Yr2,Yr4,Yr25(h)*
Flaxen			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.1	1.1	4.8	0.41	0.2	72.2	27.8	0.7	0.5	
Fletum			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	1.4	4.9	6.5	-	0.02	-	0.6	-	0.3	
Frelon			1B	Yr17	Rht-B1b	Rht-D1a	Ppd-D1a	0.6	6.4	4.1	0.14	0.01	17.2	0.6	0.5	0.2	
Fresco			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.2	6.9	7.2	0.33	0.05	63.2	7.9	0.8	0.6	*(Yr3,Yr4), Yr25,YrSD(h)*
Galahad	1984-1992	4-6	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.6	7.9	7.4	0.21	-	28.3	-	0.6	-	*Yr1(b)/(Yr1),Yr3,Yr4,Yr25(h)*
Galatea			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.5	7.4	8.1	0.24	0.12	31.1	14.2	0.6	0.5	
Gallant	2009-2012	4-6	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.8	7.4	4.8	-	0.16	-	28.9	-	0.6	
Gatsby	2006-2009	9-9	1R(Yr9)	No Yr17	Rht-B1b	Rht-D1a	Ppd-D1	0.1	0.1	2.6	0.03	0.01	2.5	0	0.2	0	
Genesis	1993-1996	5-5	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2	5.9	6.9	0.12	0.06	28.4	12.4	0.6	0.6	*YrHVII(b)*
Genghis			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.8	1.1	6.4	0.38	0.27	46.2	36.1	0.7	0.6	
Gladiator	2004-2011	8-9	1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.3	1.1	3.4	0.01	0.02	0.3	1	0.2	0.3	
Glasgow	2005-2011	4-5.2	1B	Yr17(no Yr17)	Rht-B1a	Rht-D1a	Ppd-D1	4.3	7.4	7.4	0.41	0.37	69.2	44.1	0.7	0.6	
Goldlace			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1	3.5	3.6	0.08	0.05	7.6	2.6	0.4	0.4	
Goodwill			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1	1.5	2.2	0.07	0.06	28.6	6.3	0.5	0.4	
Granta			1B	?(no Yr17)	Rht-B1a	Rht-D1a	Ppd-D1	-	-	2.4	0.06	-	9.1	-	0.5	-	

YR Panel line	Years in RL	Yellow rust HGCA scores Min-Max	Translocation 1BS or 1RS	Introgression 2NS.2AS with *Yr17***	Rht1(Rht-B1a/b)	Rht2(Rht-D1a/b)	Ppd-D1(no deletion)/Ppd-D1a	Infection score from historical data (adj.)	IF SDT Brock (adj.)	IF SDT Solstice (adj.)	AUDPCr 2010 (adj.)	AUDPCr 2011 (adj.)	Max severity 2010 (adj.)	Max severity 2011 (adj.)	Max HostR 2011 (adj.)	HostR 2011 end (adj.)	*Yr* genes postulated
Gulliver			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.3	0.6	1.9	0.01	-	0.3	-	0.4	-	
Harbour			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.1	2.5	7.4	0.28	0.12	55.3	15.6	0.7	0.6	
Harrier	1998-1998	4-4	1R(Yr9)	Yr17	Rht-B1a	Rht-D1a	Ppd-D1	5.7	1.1	7.2	0.38	0.21	62.7	27.8	0.6	0.5	
Harrow			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	4.2	0.6	7.4	0.23	0.13	22.7	15.6	0.7	0.5	
Haven	1990-1996	3-3	1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	4.7	1.1	4.8	0.42	0.11	70	12.4	0.7	0.4	*Yr6, Yr9, APR(f)/Yr9(b)*
Hereford			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.8	1.5	2	0.1	0.03	13	1.3	0.4	0.4	
Hereward	1991-2010	4-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.4	3	4.3	0.17	0.1	35.4	17.8	0.5	0.6	*Yr3,Yr32(c)/ Yr3,Yr4,Yr25,Yr32,YrSD(h)*
Heritage			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.4	2	6.9	0.29	0.19	38.7	29.9	0.6	0.7	
Hobbit	1977-1981	2-2	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	7.9	7.9	6.9	0.48	0.33	69.2	41.1	0.8	0.6	*YrHVII(b)*
Holster			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.9	3	8.1	0.25	0.14	51.4	28.9	0.8	0.6	
Hornet	1987-1991	2-9	1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	8.4	1.1	6.9	0.74	0.79	77.4	71.3	0.7	0.6	*Yr2,Yr6,Yr9(i)/Yr9(b)*
Hourra			1B	? (Yr17)	Rht-B1a+b	Rht-D1b	Ppd-D1	3.7	2	-	-	0.33	-	47.4	-	0.7	
Hudson			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.9	3.5	4.5	0.13	0.01	11.8	0.2	0.5	0.2	
Humber	2007-2011	7.5-9	1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.5	0.1	6.7	0.07	0.08	12.7	15.6	0.5	0.6	
Hunter	1993-1997	8-9	1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.4	0.6	2.6	0.06	0.01	14.1	0	0.5	0	*Yr9(a,b)/Yr3,Yr6,Yr9,Yr32(c)*
Hurley			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1a	0.1	6.4	5	0.01	0.01	0	0	0.2	0.1	
Hussar	1992-1999	5-9	1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.5	0.1	4.3	0.4	0.45	74.9	49.7	0.8	0.5	*Yr9, Yr17(f)*
Hustler	1978-1984	3-5	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	6.4	7.9	6.4	0.5	0.12	63.5	22.2	0.9	0.6	*Yr1(b)*
Hyperion	2006-2007	5-6	1B	Yr17(no Yr17)	Rht-B1a	Rht-D1b	Ppd-D1	3	1.5	6	0.23	0.08	38.3	12.4	0.8	0.6	
Impala			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.7	4	7.4	0.37	0.35	50.7	37.4	0.7	0.5	
Insight			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1	2.5	2.9	0.07	0.01	8.2	0.6	0.4	0.3	

YR Panel line	Years in RL	Yellow rust HGCA scores Min-Max	Translocation 1BS or 1RS	Introgression 2NS.2AS with *Yr17***	Rht1(Rht-B1a/b)	Rht2(Rht-D1a/b)	Ppd-D1(no deletion)/Ppd-D1a	Infection score from historical data (adj.)	IF SDT Brock (adj.)	IF SDT Solstice (adj.)	AUDPCr 2010 (adj.)	AUDPCr 2011 (adj.)	Max severity 2010 (adj.)	Max severity 2011 (adj.)	Max HostR 2011 (adj.)	HostR 2011 end (adj.)	*Yr* genes postulated
Isengrain			1B	No Yr17	Rht-B1b	Rht-D1a	Ppd-D1a	1.3	8.4	7.6	0.23	0.04	59	5.2	0.6	0.6	
Isidor			1B	?(no Yr17)	Rht-B1a+b	Rht-D1b	Ppd-D1a	3.2	8.8	-	-	0.42	-	47.1	-	0.5	
Istabraq	2004-2012	7.9-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.2	4.5	2.4	0.02	0.01	4.2	0.2	0.3	0.1	
Jacadi			1B	No Yr17	Rht-B1a	?	Ppd-D1	6.9	7.4	6.4	0.31	0.06	60.1	7.9	1	0.6	
JB Diego	2008-2011	6-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.6	1.1	4.4	0.05	0.02	8.2	1	0.4	0.5	
Joss Cambier	1968-1972	3-6	1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	3.1	7.9	8.1	0.69	0.32	81.3	44.1	0.8	0.6	*Yr2, Yr3, Yr11(d)*
Kador	1977-1981	4-8	1B	?(no Yr17)	Rht-B1a	Rht-D1a	Ppd-D1	-	8.8	8.1	0.29	0.06	57.8	7.9	0.5	0.5	
Kempt			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.2	1.1	7.2	0.52	0.54	68.7	65.7	0.7	0.6	
Ketchum	2009-2012	3.9-8	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	3.3	2	3.4	-	0.27	-	34.8	-	0.6	
Kinsman	1976-1980	3-4	1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	5.4	2	8.4	0.41	0.21	62.1	17.8	0.6	0.4	
Kipling			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.1	1.1	3.1	0.09	0.06	22.8	8	0.4	0.6	
Krakatoa			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.3	0.6	7.6	0.31	0.18	49.6	37.4	0.6	0.6	
KWS horizon			1B	No Yr17	Rht-B1b	Rht-D1a	Ppd-D1	2.2	4.9	4.8	-	0.14	-	19.6	-	0.6	
Lancelot			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.9	1.1	8.1	0.31	0.31	39.5	34.8	0.7	0.5	
Leo			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.9	2.5	6.9	-	0.06	-	10	-	0.7	
Limerick			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.1	2	2.4	0.14	0.04	40	7.9	0.5	0.5	
Longbow	1983-1989	3-6	1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	3.5	2	7.4	0.19	0.06	20	6.3	0.6	0.5	*Yr1(f) /Yr1, 2, 6(j)*
Lorraine			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1a	0.4	5.4	2.9	0.02	0.01	3.2	0	0.3	0.1	
Lynx (1)			1R(Yr9)	?(no Yr17)	Rht-B1a+b	Rht-D1b	Ppd-D1	-	-	7.6	0.92	-	84.4	-	1	-	
Lynx x (2)			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.4	0.6	5	0.33	0.14	57.8	15.6	0.7	0.6	*Yr6, Yr9, Yr17(f)/Yr9, Yr17(b)*
Macro			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.1	0.1	1.5	0.05	0.6	21.8	64.1	0.8	0.6	
Madrigal	1997-2003	3-4	1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	3.6	0.6	5.5	0.52	0.35	63.8	46.8	0.8	0.6	*Yr6, Yr9, Yr17(f)*

YR Panel line	Years in RL	Yellow rust HGCA scores Min-Max	Translocation 1BS or 1RS	Introgression 2NS.2AS with *Yr17***	Rht1(Rht-B1a/b)	Rht2(Rht-D1a/b)	Ppd-D1(no deletion)/Ppd-D1a	Infection score from historical data (adj.)	IF SDT Brock (adj.)	IF SDT Solstice (adj.)	AUDPCr 2010 (adj.)	AUDPCr 2011 (adj.)	Max severity 2010 (adj.)	Max severity 2011 (adj.)	Max HostR 2011 (adj.)	HostR 2011 end (adj.)	*Yr* genes postulated
Magellan			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.4	1.1	7.3	-	0.11	-	15.6	-	0.6	
Magnitude			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.7	4.5	3.4	0.02	0.01	1.1	1	0.2	0.5	
Malacca	1999-2009	8-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0	4.9	2.6	0.01	0.01	0.1	0	0.2	0	
Mallet			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.6	0.1	4.5	0.03	0.27	7.4	36.1	0.5	0.6	
Maris Beacon			1B	No Yr17	?	Rht-D1a	Ppd-D1	8.8	7.9	7	0.3	0.19	54	35.4	0.7	0.7	
Maris Freeman	1974-1979	3-3	1B	?(no Yr17)	Rht-B1a+b	Rht-D1a+b	Ppd-D1	-	2	7.6	0.24	0.14	56.7	14.5	0.5	0.5	
Maris Huntsman	1972-1983	4-6	1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	3.2	6.9	6.9	0.19	0.07	40.3	12.4	0.6	0.6	*Yr2, Yr3, Yr13 (Yr16) (d)*
Maris Templar			1B	No Yr17	?	Rht-D1a	Ppd-D1	4.2	2.9	6.9	0.33	0.01	52.9	0	0.6	0.1	
Marksman	2008-2010	5-8	1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.1	1.5	2.9	0.19	0.12	51.4	19.5	0.7	0.6	
Marshal			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	3.4	1.5	5.3	0.46	0.27	63.8	27.8	0.7	0.5	
Mascot	2006-2009	5-6	1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	5.2	2	7.4	0.39	0.22	53.5	32.9	0.7	0.6	
Maverick			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	8	5.4	5.3	0.43	0.28	54.9	37.4	0.5	0.5	
Mayfair			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.6	1.5	4.8	0.1	0.03	13.5	3.2	0.5	0.5	
Mayfield			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2	3	7.6	0.22	0.1	45.4	22.2	0.6	0.6	
Mega	1974-1977	5-6	1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	0.6	2.9	6	0.22	0.17	49.8	31.7	0.5	0.5	*Yr12*
Mercia	1986-1997	7-9	1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	0.7	5.4	2.2	0.02	0.02	4.3	1	0.4	0.4	
Milestone			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.1	1.1	2.6	0.23	0.04	44.6	5.2	0.5	0.5	
Monty			1R(Yr9)	No Yr17	Rht-B1b	Rht-D1a	Ppd-D1	5.6	1.1	7.6	0.49	0.24	68.4	21.1	0.7	0.6	
Monument			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.4	3.5	6	0.18	0.08	30.5	15.6	0.6	0.6	
Moulin	1985-1986	4-9	1B	?(no Yr17)	Rht-B1a+b	Rht-D1b	Ppd-D1	-	-	7.2	0.42	-	69.2	-	0.8	-	*Yr3,Yr4,Yr6(a)*
Napier	2000-2007	3-4	1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	3.7	0.6	3.1	0.27	0.32	54.2	37.4	0.7	0.5	
Newhaven			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.9	0.6	8.1	0.42	0.43	59.6	45.8	0.7	0.6	

YR Panel line	Years in RL	Yellow rust HGCA scores Min-Max	Translocation 1BS or 1RS	Introgression 2NS.2AS with *Yr17***	Rht1(Rht-B1a/b)	Rht2(Rht-D1a/b)	Ppd-D1(no deletion)/Ppd-D1a	Infection score from historical data (adj.)	IF SDT Brock (adj.)	IF SDT Solstice (adj.)	AUDPCr 2010 (adj.)	AUDPCr 2011 (adj.)	Max severity 2010 (adj.)	Max severity 2011 (adj.)	Max HostR 2011 (adj.)	HostR 2011 end (adj.)	*Yr* genes postulated
Nexus			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	3.8	1.1	4.5	0.38	0.42	60.1	43.6	0.7	0.6	
Nijinsky	2004-2008	8-8	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.2	2	2.3	0.21	0.02	46.1	2.6	0.6	0.3	
Norman	1981-1994	4-4	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	3.5	2	5.3	0.36	0.03	35.4	1	0.7	0.5	*Yr6(b)*
Oakley	2007-2011	1.8-6	1B	No Yr17	Rht-B1b	Rht-D1a	Ppd-D1	4.7	2	7.6	0.98	0.63	90.3	65.7	0.9	0.6	
Ochre			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.3	0.1	0.5	0.01	0.01	0	0	0.2	0	
Odyssey			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.2	1.1	3.4	0.45	0.12	63.5	12.4	0.7	0.5	
Option	2001-2006	9-9	1B	No Yr17	?	Rht-D1b	Ppd-D1	0.1	1.1	7.4	0.32	0.07	47.4	5	0.6	0.5	
Orestis			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	1.4	4.5	3.4	0.01	0.26	0.9	45.8	0.4	0.7	
Orton			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	5.2	0.6	7.9	0.42	0.27	50.7	34.8	0.6	0.6	
Ostara			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.7	1.1	5.3	-	0.01	-	0	-	0.1	
Oxbow			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	3.1	1.1	6.9	0.46	0.17	71.8	27.8	0.8	0.7	*Yr32 (e)*
Pagan			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.2	1.1	1.9	0.01	0.01	0	0	0.2	0.1	
Parade	1987-1989	9-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.4	2	2.2	0.01	0.01	0	0.2	0.2	0.2	
Pastiche	1989-1992	8-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.8	5.4	2.2	0.01	0.01	0	0	0.2	0	*YrHVII(b)*
Pennant			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0	0.1	2.6	0.01	0.01	0	0	0.2	0.1	
Phlebas			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.4	1.5	7.4	0.21	0.15	50.7	17.8	0.6	0.6	
Piranha			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	0.1	1.1	5.3	0.04	0.01	3.2	0.6	0.3	0.2	
Posit			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.4	1.1	8.1	0.42	0.27	62.1	46.8	0.7	0.6	
Potent			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.2	1.1	7.9	0.38	0.07	47.4	8	0.7	0.5	
PR21R60			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.5	2.5	7.3	0.62	0.46	72.2	56	0.9	0.7	
Predator			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.6	1.5	7.2	0.2	0.11	23.8	9.9	0.6	0.6	
Prophet			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.5	8.4	8.1	0.58	0.43	77.6	53.4	0.9	0.6	*Yr17(b)*
QPlus	2009-2010	6-9	1B	No Yr17	Rht-B1a	?	Ppd-D1	2.1	1.5	6.2	-	0.11	-	10	-	0.5	
Quest			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.7	1.5	5.7	0.27	0.16	41.7	24.4	0.6	0.6	

YR Panel line	Years in RL	Yellow rust HGCA scores Min-Max	Translocation 1BS or 1RS	Introgression 2NS.2AS with *Yr17***	Rht1(Rht-B1a/b)	Rht2(Rht-D1a/b)	Ppd-D1(no deletion)/Ppd-D1a	Infection score from historical data (adj.)	IF SDT Brock (adj.)	IF SDT Solstice (adj.)	AUDPCr 2010 (adj.)	AUDPCr 2011 (adj.)	Max severity 2010 (adj.)	Max severity 2011 (adj.)	Max HostR 2011 (adj.)	HostR 2011 end (adj.)	*Yr* genes postulated
Raglan			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.3	1.5	7.4	0.29	0.11	54	15.6	0.7	0.6	
Raleigh			1R(Yr9)	Yr17	?	Rht-D1b	Ppd-D1	0.4	1.1	6	0.3	0.11	42.4	9.9	0.6	0.4	
Rampart			1R(Yr9)	No Yr17 (Yr17)	Rht-B1a	Rht-D1b	Ppd-D1	3.5	0.6	8.1	0.5	0.39	65.9	39.7	0.8	0.6	
Ranger			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.7	0.1	4.1	0.28	0.21	55.5	34.1	0.5	0.6	
Reaper	1996-2000	3-4	1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	6.3	6.9	6	0.35	0.44	52.9	53.4	0.7	0.6	*Yr3,Yr17(c)/Yr17(a)*
Rendezvous	1987-1990	9-9	1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.8	5.9	6	0.31	0.34	60.1	43.6	0.7	0.5	*Yr17(e)*
Renown			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.2	4.9	-	-	0.03	-	1	-	0.6	
Reydon			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	5.1	0.6	5.7	-	0.34	-	34.8	-	0.5	
Rialto	1995-2001	4-6	1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	3.1	1.1	5	0.42	0.14	66.8	19.6	0.7	0.6	*Yr9(a,b)/Yr6,Yr9(c)*
Rib	1989-2008	4-7	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	4.3	7.4	6	0.17	0.03	13.1	1.9	0.4	0.5	*Yr6(b)*
Richmond	2003-2006	7-8	1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.2	3	2.4	0.02	0.01	3.4	1	0.4	0.4	
Ritmo			1B	?(no Yr17)	Rht-B1a	Rht-D1b	Ppd-D1a	6.4	-	6.7	0.52	0.24	65.9	28.9	0.9	0.6	*Yr1(f)*
Rivet			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.5	0.1	5.5	0.32	0.23	50.3	27.8	0.7	0.5	
Robigus	2003-2011	2-3	1B	No Yr17	Rht-B1b	Rht-D1a	Ppd-D1	5.7	2.8	6.9	0.89	0.57	93.3	68.7	0.9	0.6	*Yr2,Yr32(c)*
Rosario			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.2	2	7.9	0.36	0.42	54.2	52.9	0.7	0.6	
Rosette			1B	No Yr17 (Yr17)	Rht-B1a	Rht-D1b	Ppd-D1	0.9	2	7.4	0.52	0.47	75.8	56	0.9	0.6	
Rubens			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.9	7.4	7.2	0.41	0.29	66.5	49.3	0.8	0.6	
Russet			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.7	1.5	6.2	0.29	0.19	53.5	28.9	0.5	0.6	
Sabre			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	5.9	8.4	7.9	0.43	0.21	60.1	22.2	0.7	0.4	*YrHVII(b)*
Sahara			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.3	1.5	2.2	0.01	0.02	0	0.2	0.2	0.1	
Samurai			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.3	1.1	7.2	0.43	0.19	62.9	19.6	0.6	0.6	
Sancerre			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.7	5.4	2.4	0.01	0.01	0	0	0.2	0.1	

YR Panel line	Years in RL	Yellow rust HGCA scores Min-Max	Translocation 1BS or 1RS	Introgression 2NS.2AS with *Yr17***	Rht1(Rht-B1a/b)	Rht2(Rht-D1a/b)	Ppd-D1(no deletion)/Ppd-D1a	Infection score from historical data (adj.)	IF SDT Brock (adj.)	IF SDT Solstice (adj.)	AUDPCr 2010 (adj.)	AUDPCr 2011 (adj.)	Max severity 2010 (adj.)	Max severity 2011 (adj.)	Max HostR 2011 (adj.)	HostR 2011 end (adj.)	*Yr* genes postulated
Sarek			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1	7.4	4.9	-	0.07	-	10	-	0.5	
Savannah	1998-2006	4-5	1R(Yr9)	Yr17	Rht-B1a	Rht-D1a	Ppd-D1	6.7	1.1	3.1	0.28	0.08	29.4	7.9	0.5	0.4	*Yr1, Yr2,Yr3,Yr4, Yr9,Yr17(k)/Yr9,Yr17(c)*
Scia			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.3	2	5.5	0.3	0.12	44.7	22.2	0.6	0.6	
Scorpion 25	2003-2003	9-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.3	1.5	3.4	0.03	0.02	11.8	1.9	0.4	0.4	
Senator			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.6	0.6	8.6	0.49	0.28	60.4	37.4	0.7	0.6	
Shamrock	1999-2003	7-8	1B	Yr17 (No Yr17)	Rht-B1a	Rht-D1a	Ppd-D1	0.2	2.5	5.3	0.03	0.03	8.3	5	0.6	0.7	
Shango			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.5	2	8.1	0.34	0.08	44.1	12.6	0.6	0.5	*Yr4,Yr6(c)*
Shannon			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.6	1.5	6.9	0.11	0.06	17.1	7.9	0.6	0.5	
Slade			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.2	3.5	5.7	0.05	0.04	20.7	5	0.6	0.6	
Slejpner	1986-1991	2-5	1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	14.4	2	6.7	0.92	0.98	87.2	84	1	0.8	*Yr9(f)*
Smuggler	2004-2005	9-9	1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.2	3.5	3.8	0.01	0.01	0.3	0	0.2	0.1	*Yr1,Yr17(c)*
Soissons	1995-2009	5-8	1B	No Yr17	Rht-B1b	Rht-D1a	Ppd-D1a	1.6	8.8	7.4	0.05	0.1	19.5	13.7	0.5	0.6	
Soldier			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.8	0.6	4.9	0.47	0.33	72.2	56	0.8	0.7	
Soleil			1B	? (No Yr17)	Rht-B1a	Rht-D1a	Ppd-D1	-	-	1.7	0.01	-	0.9	-	0.2	-	
Solstice	2002-2012	3.5-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.3	1.5	5.7	0.71	0.29	72.2	46.8	0.8	0.6	
Spaldings prolific			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	0.6	2	2.2	0.01	0.05	0.4	10	0.3	0.5	*YrSP(l)*
Spark	1993-1999	6-8	1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	1.2	6.4	2.4	0.06	0.1	7.1	7.9	0.4	0.5	*Yr6(a,b)*
Spry			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.6	1.5	7.9	0.19	0.13	28.6	15.6	0.5	0.5	
Squadron			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.5	1.1	6.9	0.51	0.38	77.6	56	0.8	0.6	
Steadfast			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.2	0.6	6.7	0.54	0.12	62	19.5	0.7	0.6	
Stetson	1983-1984	9-9	1R(Yr9)	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	6.7	2	6	0.59	0.32	71.2	19.6	0.7	0.5	
Storm			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.9	1.5	6.4	0.2	0.1	56.7	15.6	0.5	0.4	

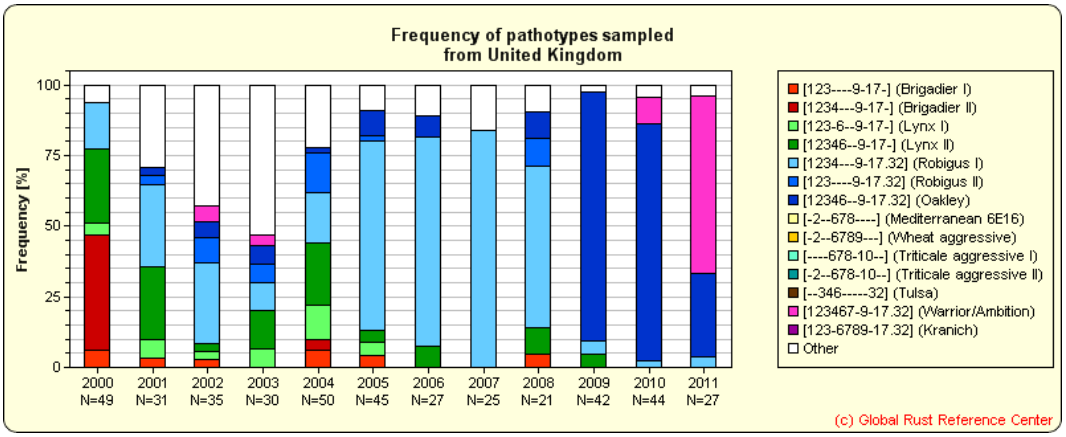
YR Panel line	Years in RL	Yellow rust HGCA scores Min-Max	Translocation 1BS or 1RS	Introgression 2NS.2AS with *Yr17***	Rht1(Rht-B1a/b)	Rht2(Rht-D1a/b)	Ppd-D1(no deletion)/Ppd-D1a	Infection score from historical data (adj.)	IF SDT Brock (adj.)	IF SDT Solstice (adj.)	AUDPCr 2010 (adj.)	AUDPCr 2011 (adj.)	Max severity 2010 (adj.)	Max severity 2011 (adj.)	Max HostR 2011 (adj.)	HostR 2011 end (adj.)	*Yr* genes postulated
SW Maxi			1B	? (No Yr17)	Rht-B1a	Rht-D1a	Ppd-D1	-	-	6.2	0.01	-	0.5	-	0.2	-	
SW Tataros			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.8	4.9	5.5	0.02	0.01	0.9	0	0.3	0	
Talon	1991-1992	9-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	5.4	2.5	6.4	0.4	0.19	62.1	25.3	0.5	0.4	
Tambor			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.5	6.9	7.2	0.06	0.1	4.9	7.9	0.4	0.5	
Tanker	2001-2005	5-7	1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	4.2	0.6	3.4	0.32	0.16	50.9	25.3	0.7	0.6	
Tara			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.3	1.5	3.8	0.01	0.01	0.9	0.2	0.4	0.2	
Tellus			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0	4.5	6.2	0.42	0.37	66.4	62.6	0.7	0.5	
Tempest			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.4	1.1	-	-	0.09	-	7.9	-	0.5	
Temple			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.9	5.9	5.3	-	0.28	-	50.3	-	0.7	
Thatcher			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	12.3	7.9	2.2	0.01	0.15	0.1	22.2	0.2	0.5	
Tilburi			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	6.9	4.5	3.8	0.53	0.46	78.3	66.8	1	0.8	
Tiller			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.7	-	-	-	-	-	-	-	-	
Timber	2007-2009	8-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.6	2.5	2.2	0.01	0.08	0.2	12.4	0.2	0.5	
Tommy			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	3.7	7.4	2.4	0.01	0.03	0	1.2	0.2	0.3	
Tonic	1985-1995	4-9	1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	0.4	2	2.2	0.01	0.01	0	0.8	0.2	0.2	*Yr7(b)*
Torfrida	1993-1994	9-9	1B	Yr17	Rht-B1a	Rht-D1a	Ppd-D1	0.5	4	4.1	0.28	0.41	39.9	62.6	0.7	0.7	*Yr17(b)*
Toronto			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	1	1.1	6.2	0.09	0.02	7.2	1	0.4	0.4	
Travix			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	9.7	1.1	6.2	0.44	0.39	66.4	37.4	0.7	0.6	
Trend			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.7	2.9	6.2	0.01	0.65	0	71.3	0.2	0.6	
Turpin			1R(Yr9)	Yr17	Rht-B1a	Rht-D1a	Ppd-D1	0.7	1.1	6.9	0.35	0.6	62.1	64.5	0.7	0.5	
Urban			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	4.8	8.4	7.2	0.3	0.1	59.2	8	0.8	0.6	
Vault			1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.2	7.9	3.5	0.01	0.01	0.3	0.6	0.2	0.3	
Vector			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0	0.1	1.9	0.01	0.01	0	0	0.2	0	
Verdon			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.4	6.9	-	-	0.07	-	12.4	-	0.5	

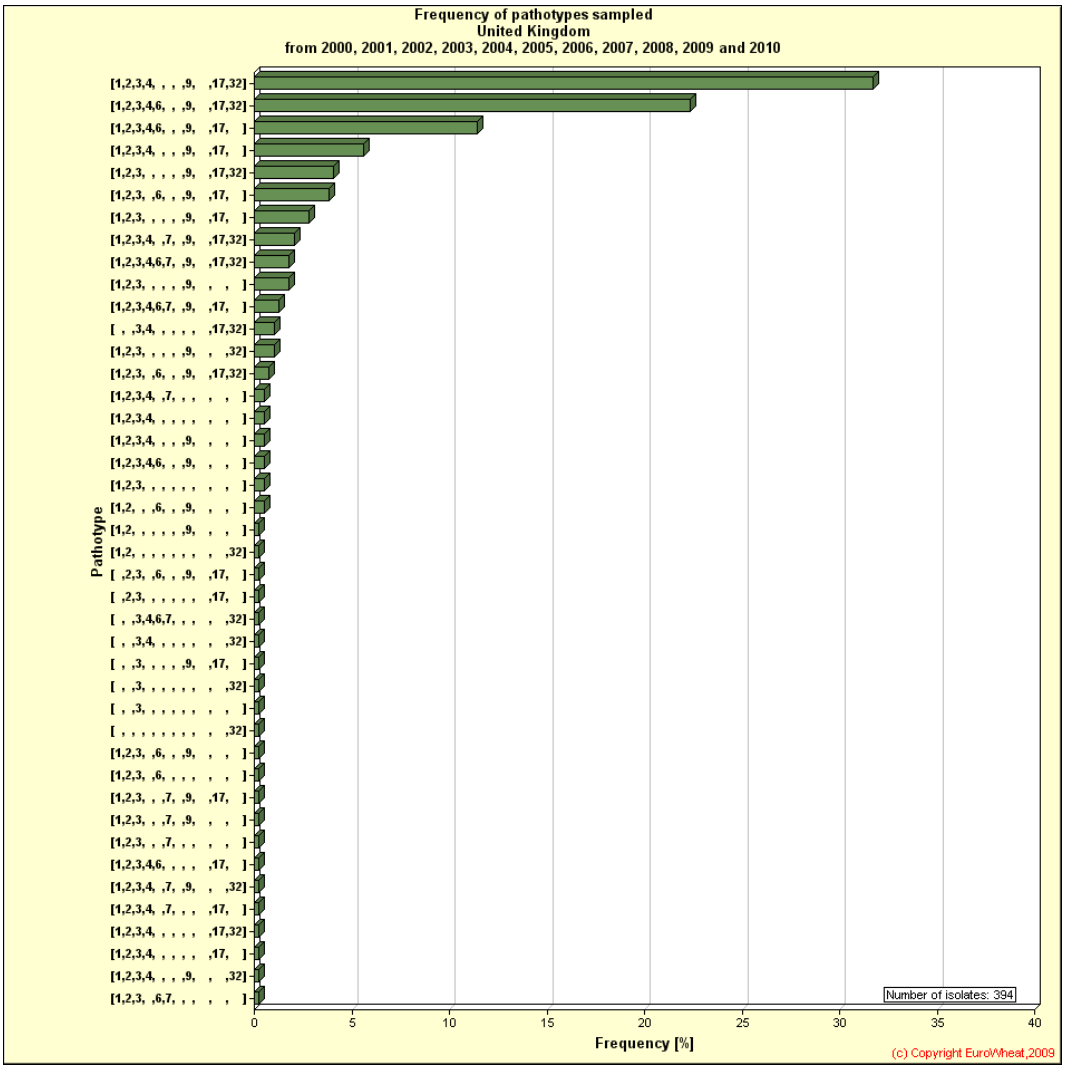
YR Panel line	Years in RL	Yellow rust HGCA scores Min-Max	Translocation 1BS or 1RS	Introgression 2NS.2AS with *Yr17***	Rht1(Rht-B1a/b)	Rht2(Rht-D1a/b)	Ppd-D1(no deletion)/Ppd-D1a	Infection score from historical data (adj.)	IF SDT Brock (adj.)	IF SDT Solstice (adj.)	AUDPCr 2010 (adj.)	AUDPCr 2011 (adj.)	Max severity 2010 (adj.)	Max severity 2011 (adj.)	Max HostR 2011 (adj.)	HostR 2011 end (adj.)	*Yr* genes postulated
Veritas			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1	1.1	-	-	0.02	-	1.9	-	0.5	
Vilmorin 27			1B	? (No Yr17)	Rht-B1a	Rht-D1a	Ppd-D1	-	7.4	2.4	0.14	0.02	32.9	0.2	0.5	0.2	
Virtue	1979-1985	3-4	1B	?(No Yr17)	Rht-B1a+b	Rht-D1b	Ppd-D1	-	-	5.5	0.39	-	62.1	-	0.8	-	
Virtuose			1B	Yr17	Rht-B1b	Rht-D1a	Ppd-D1	0.2	7.4	4.1	0.01	0.01	0	0	0.2	0.1	
Vivant			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.5	2	6	0.81	0.65	76	71.7	0.9	0.6	*Yr32(l)*
Vuka			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	11.5	8.2	8.2	0.66	0.26	76.5	22.3	0.9	0.5	
Warlock 24			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.2	1.5	3.1	0.03	0.02	5.6	3.2	0.4	0.3	
Warrior (1)			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1	1.1	6.9	-	0.62	-	65.7	-	0.6	
Warrior xxx(2)	2011-2012	8.4-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.7	1.2	4.1	-	0.01	-	0.8	-	0.3	
Wasp			1B	No Yr17	Rht-B1a	Rht-D1a	Ppd-D1	0.9	6.9	4.5	0.09	0.01	18.2	0	0.4	0.2	
Welford	2004-2008	6-7	1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.2	1.1	5	0.38	0.16	65.9	19.5	0.7	0.5	
Wellington			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	2.1	1.1	6.7	0.53	0.52	77.8	50.3	0.7	0.6	
Weston			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.5	1.1	6.4	0.57	0.71	84.4	77.4	0.8	0.6	
Wickham			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.8	0.6	5.5	0.44	0.42	72.2	53.4	0.8	0.5	
Windsor			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.3	2	7.4	0.96	0.54	81.3	68.7	0.7	0.7	*Yr2,Yr32(c)*
Wizard	2003-2006	4-5	1B	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.9	1.5	6.4	0.31	0.11	50.9	19.6	0.8	0.6	
Woburn			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.2	2	3.6	0.1	0.05	20.7	3.2	0.5	0.5	
Woodstock			1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.7	7.4	6.1	-	0.01	-	0	-	0.1	
XI19	2002-2010	9-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0	1.5	2.2	0.02	0.04	5	3.2	0.4	0.3	
Zaka			1R(Yr9)	Yr17	Rht-B1a	Rht-D1b	Ppd-D1	1.4	0.1	6.9	0.47	0.25	60.1	41.1	0.6	0.6	
Zebedee	2007-2010	9-9	1B	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.1	5.4	4.1	0.03	0.02	5.6	1.9	0.4	0.6	
Zodiac			1R(Yr9)	No Yr17	Rht-B1a	Rht-D1b	Ppd-D1	0.5	0.6	3.4	0.07	0.02	12.8	0.6	0.4	0.2	*Yr9(b)*

APPENDIX 5: Virulence frequency observed (%) from WYR isolates tested by the UKCPVS from 1977 to 2010.
(Adapted from UKCPVS reports from 1980 to 2010)

Virulence factor	Vir1	Vir2	Vir3	Vir4	Vir5	Vir6	Vir7	Vir8	Vir9	Vir10	Vir15	Vir17	Vir32	VirA	VirSd	VirSo	VirSP	VirRob	VirSol	VirClaire	VirCad	Number of isolates
2010	100	100	100	100	•	98	24	0	99	•	0	97	95	•	•	•	•	100	98	83	3	40
2009	100	100	99	98	•	90	0	0	93	•	0	87	94	•	•	•	•	81	69	43	0	42
2008	100	100	91	81	•	19	0	0	93	•	0	83	79	•	•	•	•	81	10	14	0	21
2007	•	•	•	100	•	4	8	0	94	•	0	88	92	•	•	•	•	84	•	4	0	25
2006	•	•	•	100	•	19	1	0	100	•	0	100	89	•	•	•	•	89	•	0	0	27
2005	100	100	100	87	•	10	8	0	95	•	0	97	85	•	•	•	0	79	0	23	0	39
2004	100	100	93	50	•	42	4	0	100	0	0	85	38	•	•	•	0	31	0	•	0	48
2003	100	100	100	86	•	50	36	0	93	0	0	93	64	•	100	93	7	•	7	•	0	14
2002	97	97	97	63	•	31	3	0	88	0	0	88	73	97	81	67	0	•	6	•	0	36
2001	100	100	100	74	•	39	0	0	90	0	0	77	42	90	84	77	0	•	•	•	0	31
2000	100	100	100	90	•	32	4	0	96	0	0	96	16	88	100	90	0	•	•	•	•	50
1999	99	99	100	87	•	21	10	0	99	•	•	100	4	91	98	91	0	•	•	•	•	97
1998	99	99	100	79	•	7	4	0	99	•	•	99	1	84	78	78	0	•	•	•	•	94
1997	100	100	100	47	•	1	7	0	99	•	•	99	13	•	•	•	•	•	•	•	0	138
1996	97	97	100	59	•	16	3	0	97	•	•	84	9	•	•	•	•	•	•	•	0	32
1995	90	99	100	67	•	35	0	0	95	•	•	57	55	•	•	•	•	•	•	•	0	83
1994	65	100	100	96	•	41	4	0	94	•	•	•	75	•	•	•	•	•	•	•	0	49
1993	89	98	100	89	•	68	8	0	84	•	•	•	•	•	•	•	•	•	•	•	2	63
1992	88	100	100	86	•	88	7	0	76	•	•	•	•	•	•	•	•	•	•	•	0	77
1991	91	100	100	86	•	64	19	0	88	•	•	•	•	•	•	•	•	•	•	•	•	42
1990	85	100	100	91	•	69	9	•	94	•	•	•	•	•	•	•	•	•	•	•	•	67
1989	62	100	100	97	•	57	2	•	99	•	•	•	•	•	•	•	•	•	•	•	•	156
1988	68	100	100	78	•	72	6	•	66	•	•	•	•	•	•	•	•	•	•	•	•	71
1987	87	100	100	47	•	89	8	•	5	•	•	•	•	•	•	•	•	•	•	•	•	52
1986	78	100	100	70	•	96	22	•	4	•	•	•	•	•	•	•	•	•	•	•	•	23
1985	76	100	100	45	•	90	3	•	3	•	•	•	•	•	•	•	•	•	•	•	•	29
1984	75	100	100	31	0	64	3	0	31	0	•	•	•	•	•	•	•	•	•	•	•	36
1983	85	100	100	20	0	26	0	0	23	0	•	•	•	•	•	•	•	•	•	•	•	63
1982	63	100	100	37	0	29	5	2	2	0	•	•	•	•	•	•	•	•	•	•	•	41
1981	71	100	95	29	0	31	5	0	5	0	•	•	•	•	•	•	•	•	•	•	•	42
1980	95	100	85	15	0	25	0	0	0	0	•	•	•	•	•	•	•	•	•	•	•	20
1979	83	100	100	17	0	17	0	0	0	0	•	•	•	•	•	•	•	•	•	•	•	30
1978	73	97	100	27	0	26	0	0	0	0	•	•	•	•	•	•	•	•	•	•	•	66
1977	73	100	100	24	0	16	8	4	0	0	•	•	•	•	•	•	•	•	•	•	•	26
1976	92	100	100	12	0	4	0	2	6	0	•	•	•	•	•	•	•	•	•	•	•	52
1975	69	99	100	35	0	62	1	0	1	0	•	•	•	•	•	•	•	•	•	•	•	68
1974	62	91	97	35	0	26	4	0	0	0	•	•	•	•	•	•	•	•	•	•	•	128
1973	40	97	98	59	•	47	1	•	•	•	•	•	•	•	•	•	•	•	•	•	•	157
1972	23	99	99	76	•	26	2	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
1971	24	91	97	71	•	3	0	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
1970	9	57	93	82	•	30	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
1969	24	34	85	72	•	19	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•

APPENDIX 6: Frequency of pathotype sampled in the United Kingdom
Source Eurowheat.org





APPENDIX 7: List of differential host varieties control tested in extended virulence tests of Pst isolates used in de novo phenotype

WYR official differential hosts tested

[(a)] Differential series described in (Chen, 2007) (Bayles et al., 2001). [(b)] *Yr* genes referring to (Chen and Line, 1992), (Chen and Line, 1993b), (Chen et al.),(Bayles, 2001),(Boshoff et al., 2002),(Eriksen et al., 2004),(Chen, 2007), (Lin and Chen, 2009).

Differential host	**Differential series [(a)]**	***Yr* genes [(b)]**
Chinese 166	Europe/ USA	*Yr1*
Kalyansona	Europe	*Yr 2*
Heines Kolben	Europe	*Yr2, Yr6*
Yamhill	USA	*Yr2,Yr4a, YrYam*
Heines VII	USA	*Yr2, Yr25,YrHVII*
Heines Peko	Europe	*Yr2, Yr6, Yr25*
Vilmorin 23	Europe	*Yr3a,Yr4a*
Nord Desprez	Europe	*Yr3a, Yr4a +*
Hybrid 46	Europe	*Yr3b, Yr4b*
T. spelta Album	Europe	*Yr5*
Fielder	USA	*Yr6, Yr20*
Lee	Europe/ USA	*Yr7, Yr22, Yr23*
Compair	Europe/ USA	*Yr8, Yr19*
Federation x4/Kavkaz	Europe	*Yr9*
Riebesel 47-51	USA	*Yr9*
Clement	Europe/ USA	*Yr2,Yr9,Yr25, YrCle*
Moro	Europe/ USA	*Yr10,YrMor*
Boston	UK	*Yr15*
VPM 1	Europe	*Yr17*
Hyak	USA	*Yr17*
Lemhi	USA	*Yr21*
Carstens V	Europe	*Yr32, Yr25*
Paha	USA	*YrPa1, YrPa2, YrPa3*
Produra	USA	*YrPr1,YrPr2*
Tres	USA	*YrTr1, YrTr2, Yr32*
Tyee	USA	*YrTye*
Strubes Dickkopf	Europe	*Yr25, YSd*
Spalding Prolific	Europe	*YrSp*
Express	USA	*YrExp1, YrExp2 +APR*

APPENDIX 7 continued

Reference varieties included in extended virulence test

*Adult plant resistance; + additional unnamed resistance factors present

Reference variety	Country	*Yr* genes/QTL	Reference
Cappelle Desprez	France	*Yr3a, Yr4a,Yr16* +*	(Agenbag et al., 2012)
Druchamp	France	*Yr3a,Yr4a,YrD, YrDru, YrDru2*	(Chen and Line, 1993b) (Chen et al., 1994) (Chen et al., 1996)
Minister	Belgium	*Yr3c, YrMin*	(Johnson, 1992b)
Madrigal	UK	*Yr6,Yr9,Yr17*	(Bayles and Stigwood, 2001)
Hornet	UK	*Yr6, Yr9*	(Bayles and Stigwood, 1991)
Tommy	France	*Yr7*	(Bayles and Stigwood, 1991)
Brock	UK	*Yr7, Yr14**	(Johnson, 1992a)
Guardian	UK	*Yr13*,Yr29**	(Melichar et al., 2008) (Bayles and Priestley, 1983)
Rendez-vous	UK	*Yr17*	(Bayles and Stigwood, 1995)
Opata 85	Mexico	*Yr18*,Yr27,Yr30**	(McDonald et al., 2004) (Singh et al., 2000b)
Cook	Australia	*Yr18*, YrCK*	(Navabi et al., 2005) (Park et al., 1992)
Talon	Germany	*Yr32*	(Hubbard and Bayles, 2011)
Oxbow	UK	*Yr32 +*	(Bayles et al., 2003)
Ciano T79	Mexico	*Yr27*	(McDonald et al., 2004)
Selkirk	Canada	*Yr27*	(McDonald et al., 2004)
Batavia	Australia	*Yr33,YrA,YrBat1,YrBat2*	(Nazari and Wellings, 2008) (Zahravi et al., 2003)
Alpowa (=WA 7677)	USA	*Yr39*, YrAlp,*	(Lin and Chen, 2007a)
IDO377s	USA	*Yr43*	(Cheng and Chen, 2010a)
ZAK	USA	*Yr44 (=YrZac)*	(Sui et al., 2009) (Cheng and Chen, 2010b)
PI181434 (=205)	Afghanistan	*Yr45*	(Li et al., 2010)
C 591	India	*YrC591*	(Li et al., 2009)
Daws	USA	*YrDa1, YrDa2*	(Chen et al., 1995b)
Cadenza	UK	*unknown*	(Hubbard and Bayles, 2011)
Claire	UK	*unknown*	(Hubbard and Bayles, 2011)
Robigus	UK	*unknown*	(Hubbard and Bayles, 2011)
Solstice	UK	*unknown*	(Hubbard and Bayles, 2011)
Timber	France	*unknown*	(Hubbard and Bayles, 2011)
Warrior (RAGT)	UK	*unknown*	(Hubbard and Bayles, 2011)
Alcedo	Germany	*QTL 2DL, QTL 4BL**	(Jagger et al., 2011)
Lalbahadur	India	*none*	(Singh et al., 1998)

APPENDIX 7 continued

List of near isogenic lines

(a) Differential series described in Chen (2007) Bayles et al. (2001). (b) Variable responses observed by different institutes worldwide presented by Collins Welling at the 2011 Borlaug Global Rust Initiative workshop.

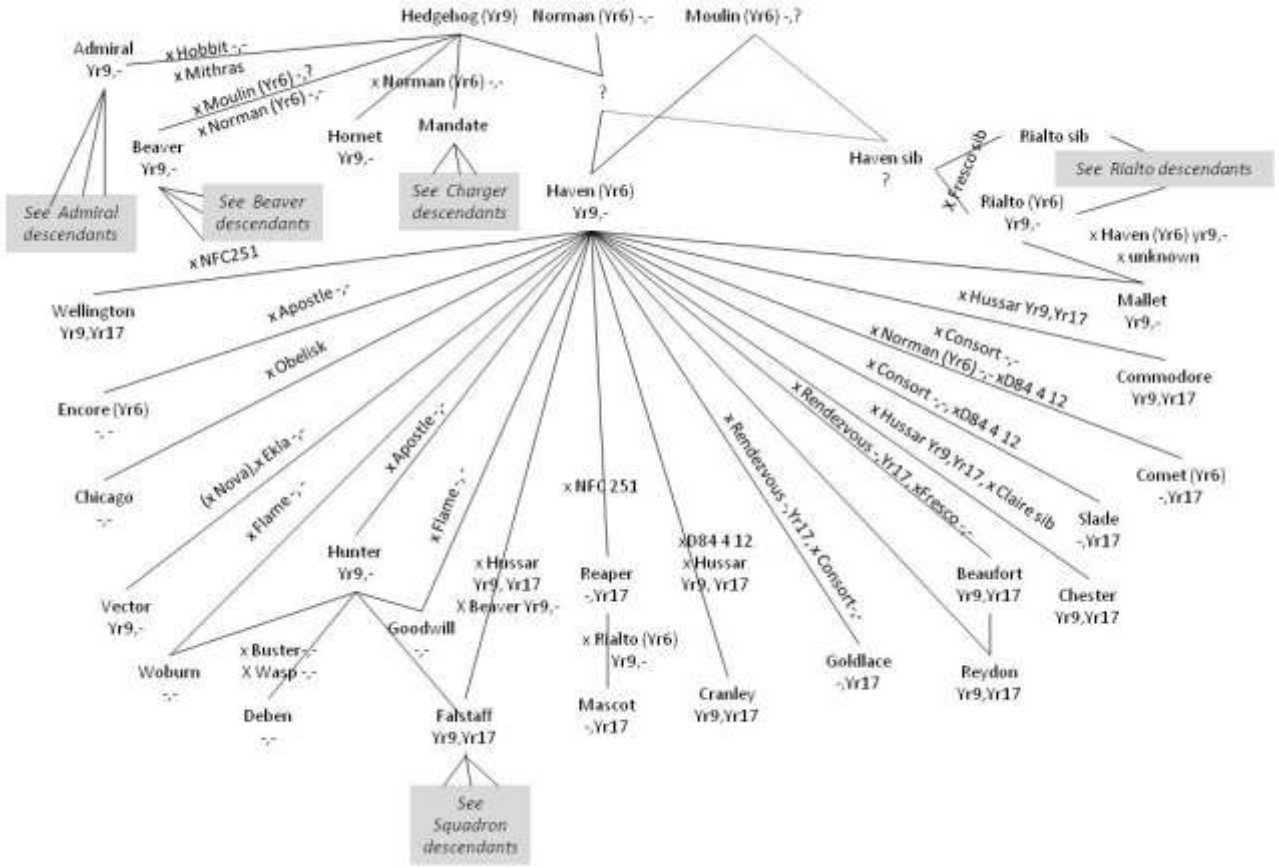
WYR near-isogenic line	Yr gene	Parent donor	Include in differential series (a)	Variable response observed (b)
Avocet S	*none*	-	-	-
Avocet R	*YrA*	-	Europe	-
Yr1/6* AvS	*Yr1*	Chinese 166	-	Yes (+*Yr18*?)
Yr5/6* AvS	*Yr5*	*Triticum spelta* Album	North America	Yes (+*Yr18*?)
Yr6/6* AvS	*Yr6*	Oxley	-	-
Yr7/6* AvS	*Yr7*	Lee	-	-
Yr8/6* AvS	*Yr8*	Compair	North America	-
Yr9/6* AvS	*Yr9*	Clement	North America	-
Yr10/6* AvS	*Yr10*	Moro	-	Yes (+*Yr18*?)
Yr15/6* AvS	*Yr15*	*T. dicoccoides* V763-251	Europe	-
Yr17/6* AvS	*Yr17*	VPM 1	-	Yes (+*Yr18*?)
Yr18/6* AvS	*Yr18*	Jupateco R	-	-
Yr24/6* AvS	*Yr24*	Meering2*//K733/ *T. tauschii* (CPI 18911)	-	-
Yr26/6* AvS	*Yr26*	*T. turigidum*	-	-
Yr27/6* AvS	*Yr27*	Opata 85	-	-
Yr32/6* AvS	*Yr32*	Carstens V	-	-
YrSP/6* AvS	*YrSP*	Spaldings prolific	-	Yes (+*Yr18*?)
Jupateco S	*none*	-	-	-
Jupateco R	*Yr18*	-	-	-

APPENDIX 8: Pedigree diagrams

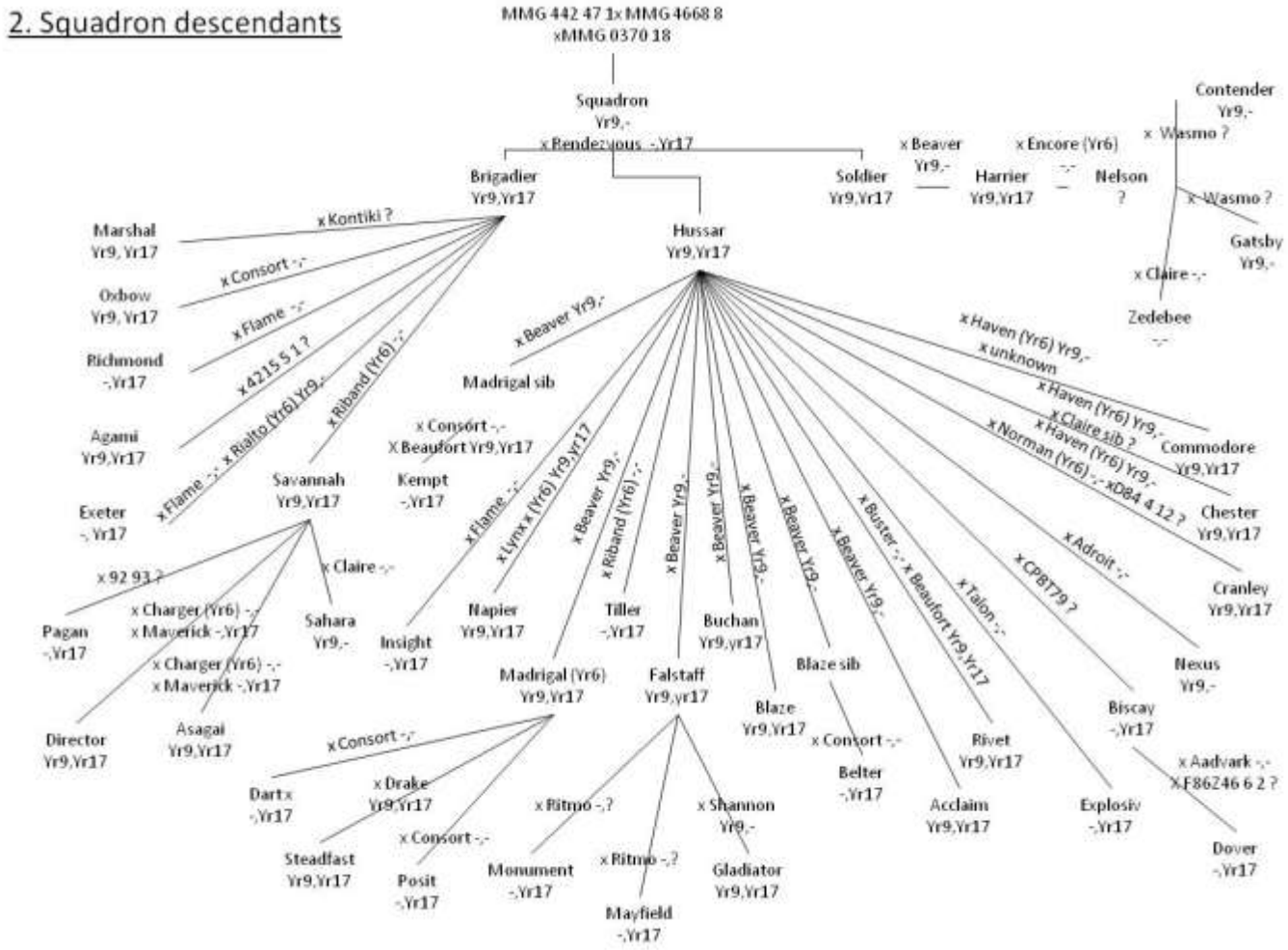
Presence/ absence of *Yr9 and Yr17* based respectively on DArT genotype and *Yr17* assay from Robert et al. (2000) are indicated as followed : "-,-" absence of *Yr9* *Yr17*, "-,Yr17" absence of *Yr9* presence of *Yr17*, "Yr9,-" presence of *Yr9* absence of *Yr17*, "Yr9,Yr17" presence of *Yr9* *Yr17*, ? for indeterminate; *Yr* genes in parentheses are sourced from several publications: Pathan et al. (2008), Hovmøller (2007), Singh et al. (2008), Hovmøller (2001b), Johnson (2001); *Yr* gene in red indicated an inconsistency between the pedigree the presence of the *Yr* gene

1- Hedgehog Haven descendants
2- Squadron descendants
3- Clement descendants
4- Benno descendants
5- Moulin descendants
6- Hobbit descendants
7- Apostle descendants
8- Armada Wasp descendants
9- Lynx x(2) descendants
10- Arminda descendants
11- Admiral, Fresco Consort descendants
12- Rendezvous descendants
13- Marksman (Maris marksman) descendants
14- Cadenza descendants
15- Thatcher descendants
16- Maris huntsman descendants
17- Norman descendants
18- Rialto descendants
19- Beaver descendants
20- Charger descendants

1.Hedgehog and Haven descendants

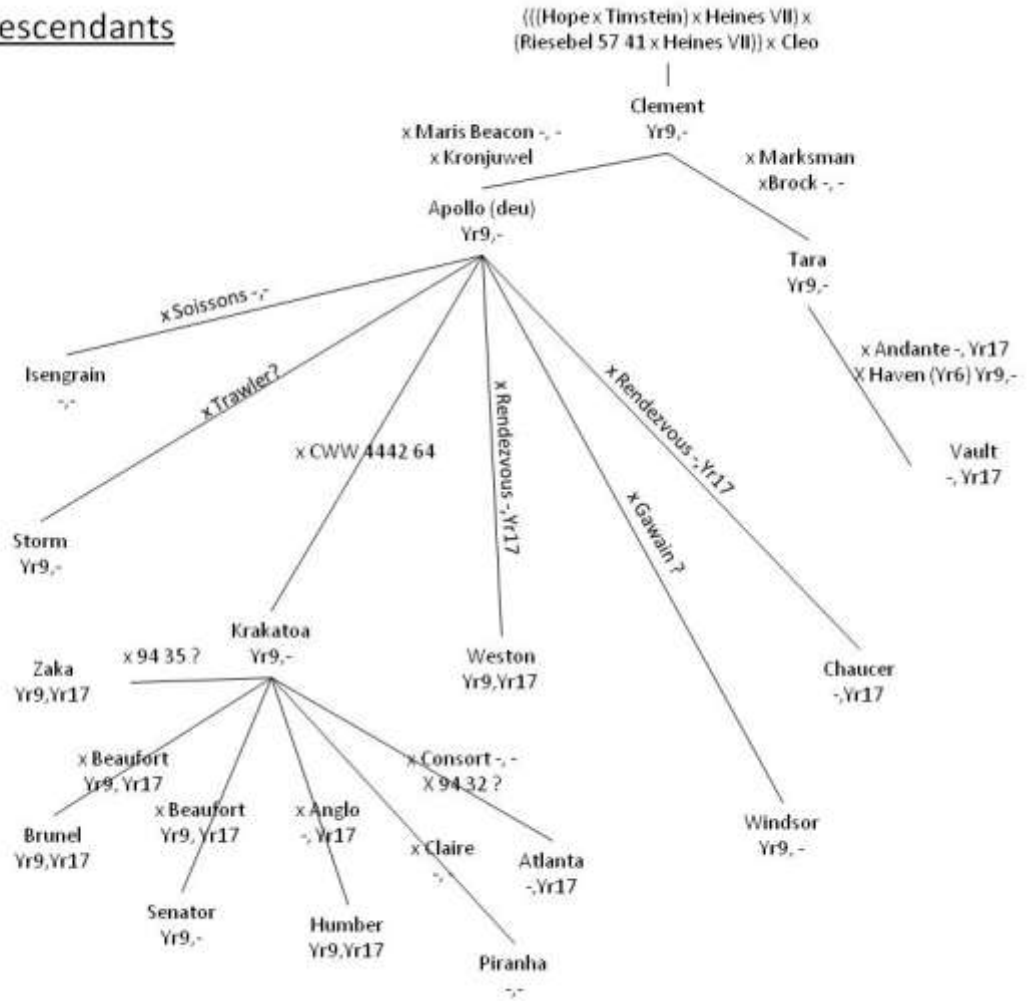


2. Squadron descendants

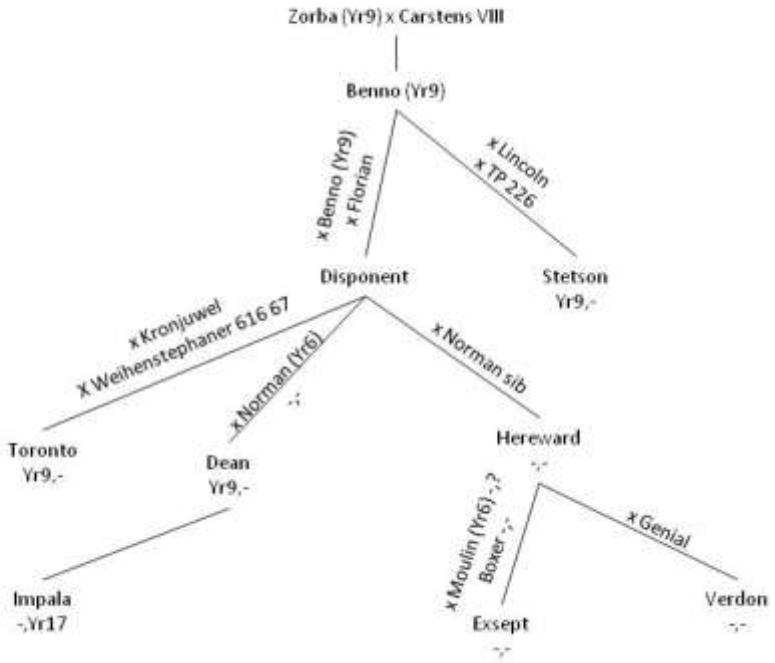


22-

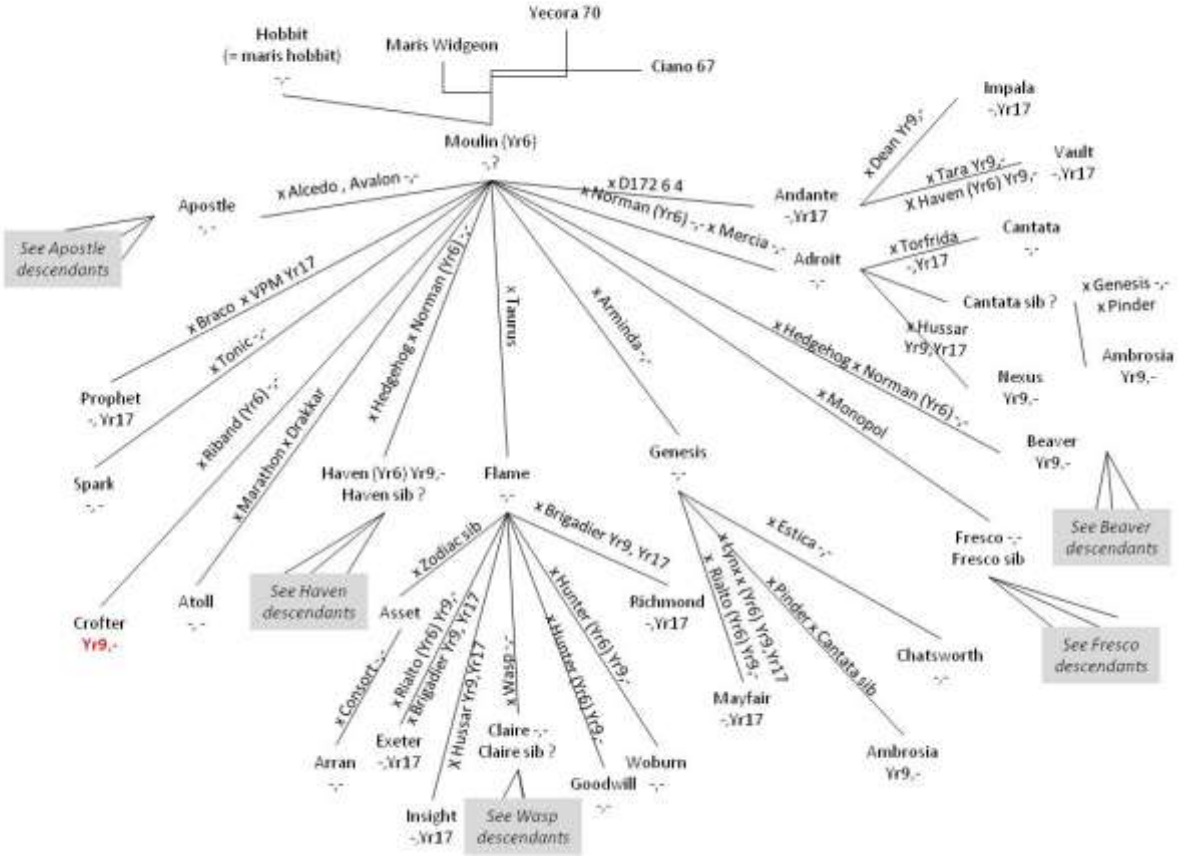
3. Clement descendants

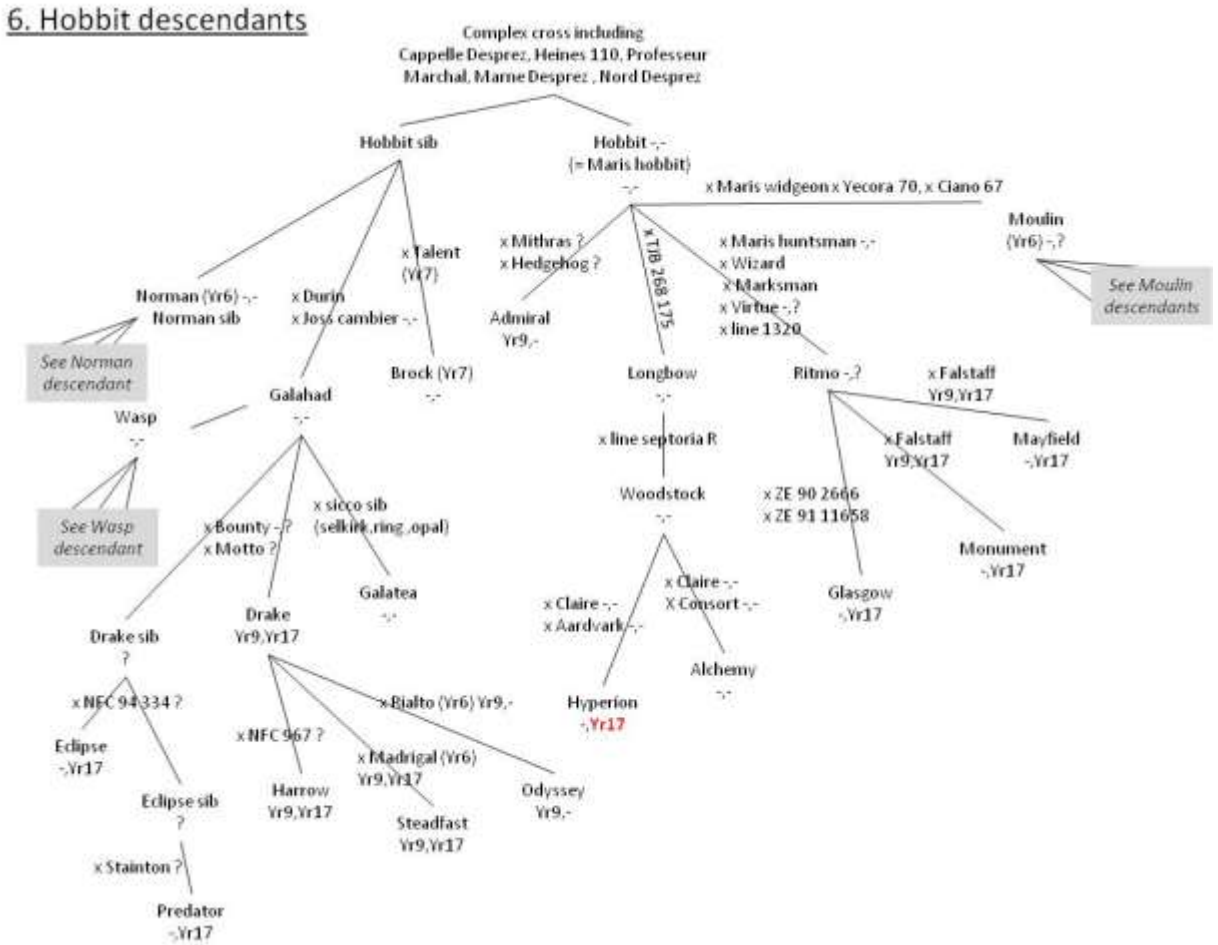


4. Benno descendants

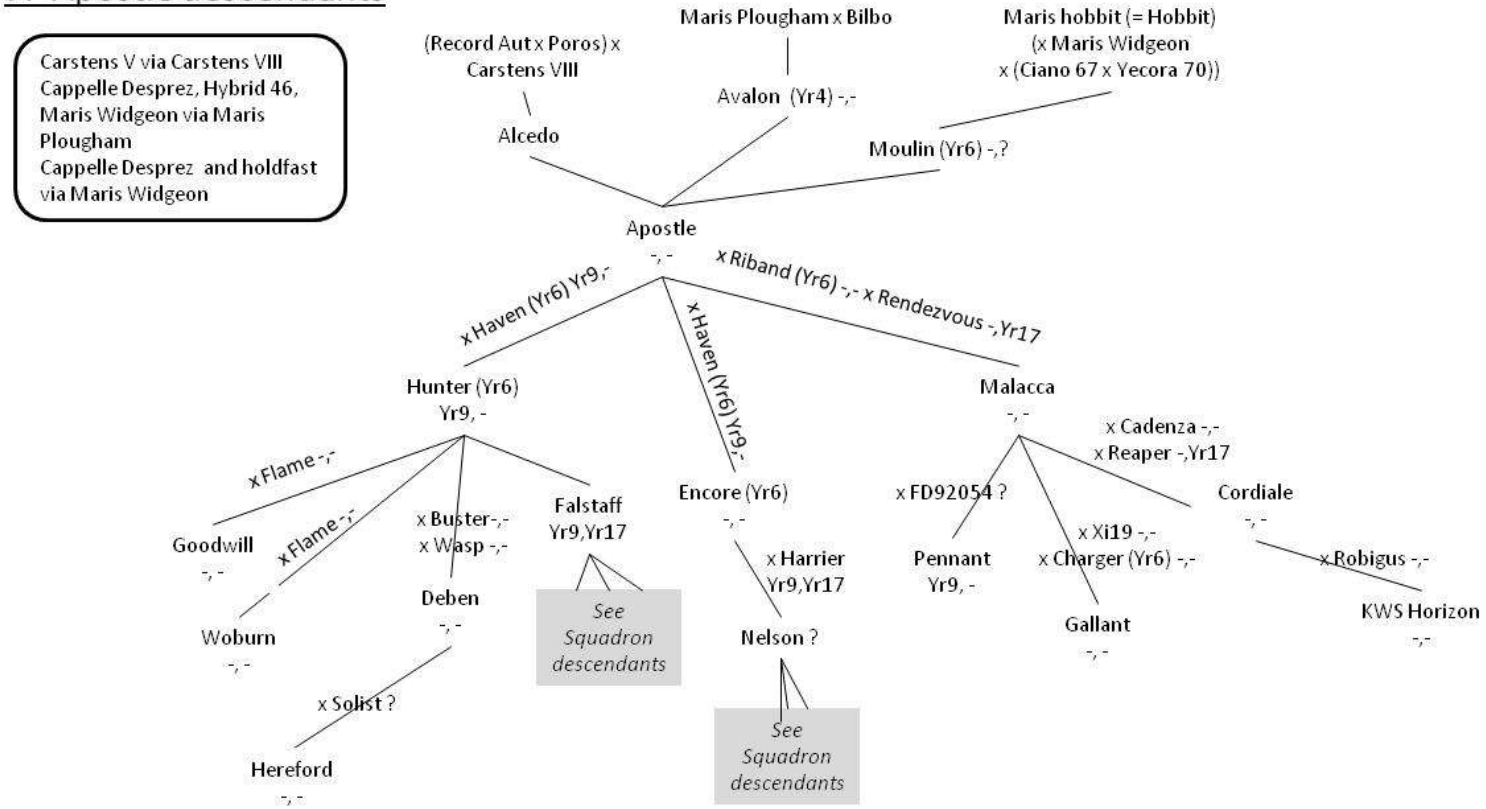


5. Moulin descendants

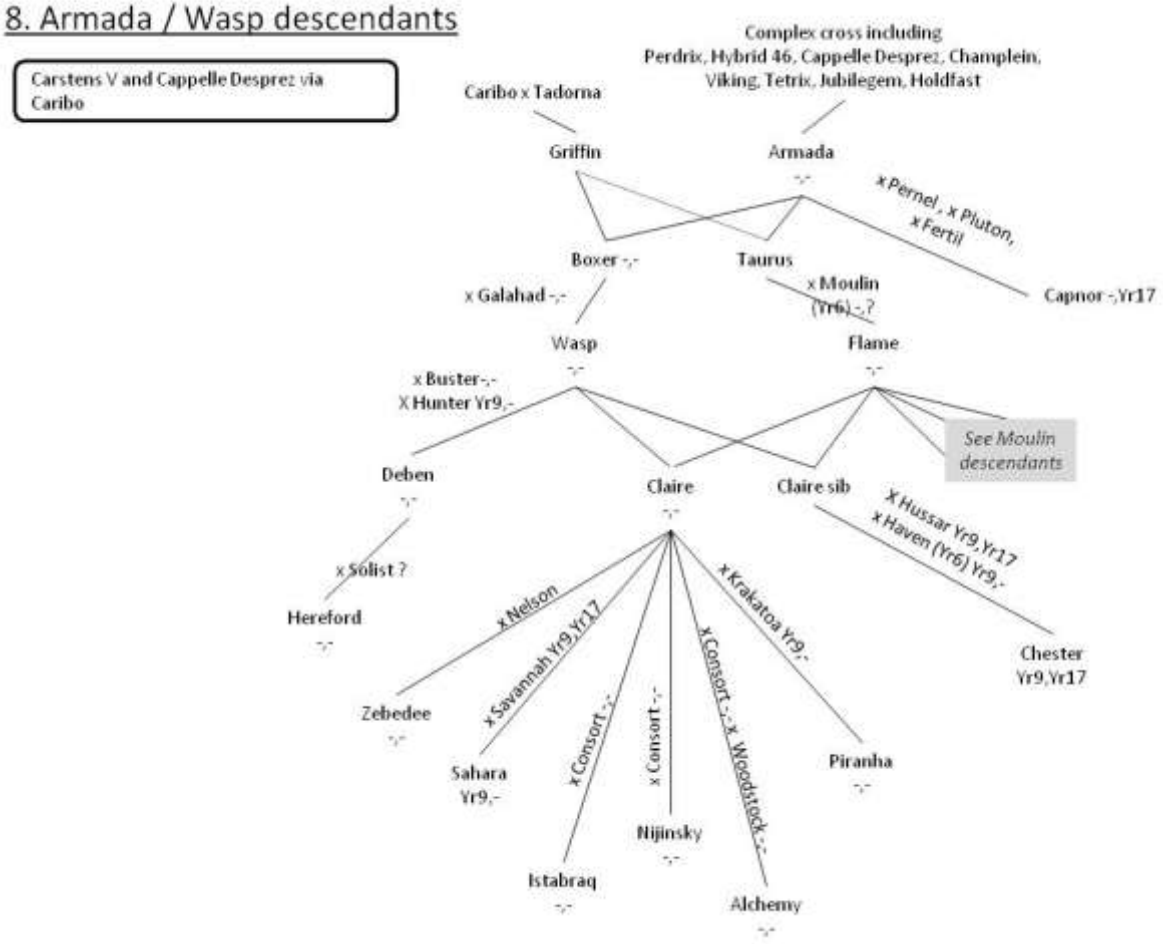



6. Hobbit descendants
Complex cross including
Cappelle Desprez, Heines 110, Professeur
Marchal, Marne Desprez , Nord Desprez
Hobbit sib
Hobbit -,-
(= Maris hobbit)
-,-
x Maris widgeon x Yecora 70, x Ciano 67
Moulin
(Yr6) -,?
See Moulin
descendants
x Mithras ?
x Hedgehog ?
x TIB 268 175
x Maris huntsman -,-
x Wizard
x Marksman
x Virtue -,?
x line 1329
x Talent
(Yr7)
Norman (Yr6) -,-
Norman sib
x Durin
x Joss cambier -,-
Admiral
Yr9,-
See Norman
descendant
Brock (Yr7)
-,-
Longbow
-,-
Ritmo -,?
x Falstaff
Yr9,Yr17
Galahad
-,-
Wasp
-,-
x line septoria R
x Falstaff
Yr9,Yr17
Mayfield
-,Yr17
See Wasp
descendant
x Bounty -,?
x Motto ?
x sicco sib
(selkirk ,ring ,opal)
Woodstock
-,-
x ZE 90 2666
x ZE 91 11658
Monument
-,Yr17
Galatea
-,-
x Claire -,-
X Consort -,-
x Claire -,-
x Aardvark -,-
Glasgow
-,Yr17
Drake sib
?
Drake
Yr9,Yr17
Alchemy
-,-
x NFC 94334 ?
x Rialto (Yr6) Yr9,-
Hyperion
-,Yr17
Eclipse
-,Yr17
x NFC 967 ?
x Madrigal (Yr6)
Yr9,Yr17
Harrow
Yr9,Yr17
Odyssey
Yr9,-
Steadfast
Yr9,Yr17
Eclipse sib
?
x Stainton ?
Predator
-,Yr17

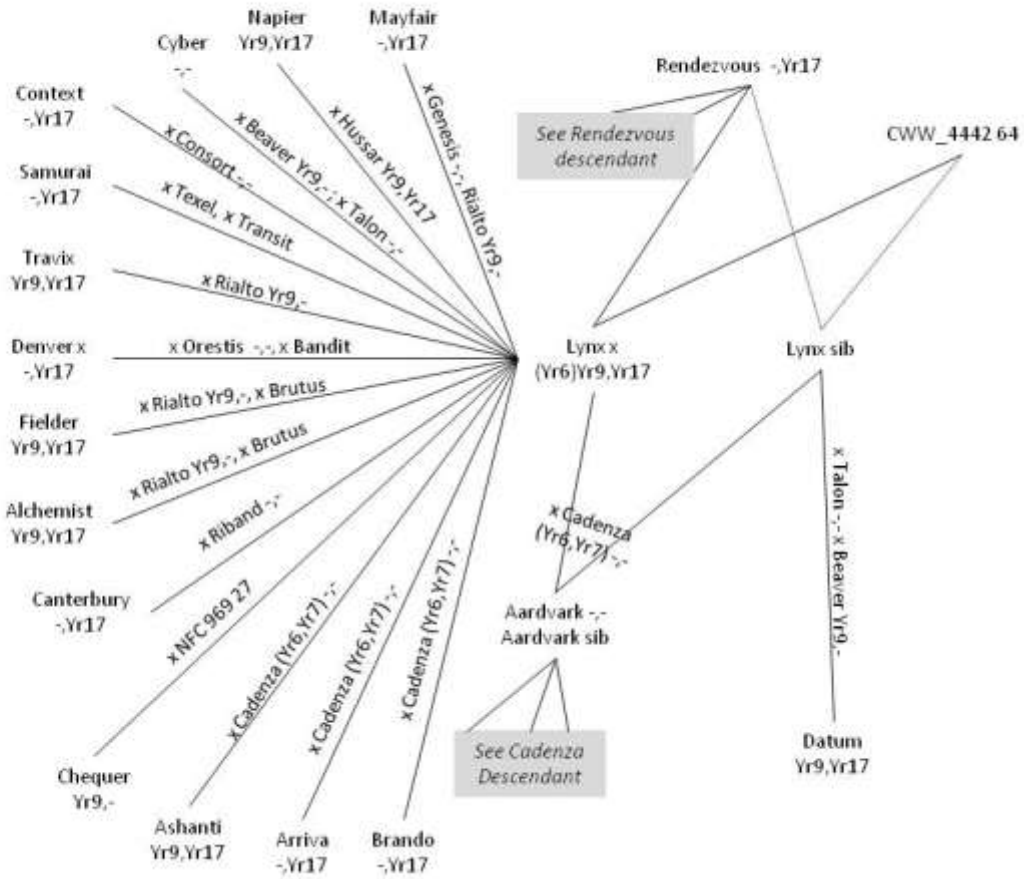
7. Apostle descendants



8. Armada / Wasp descendants

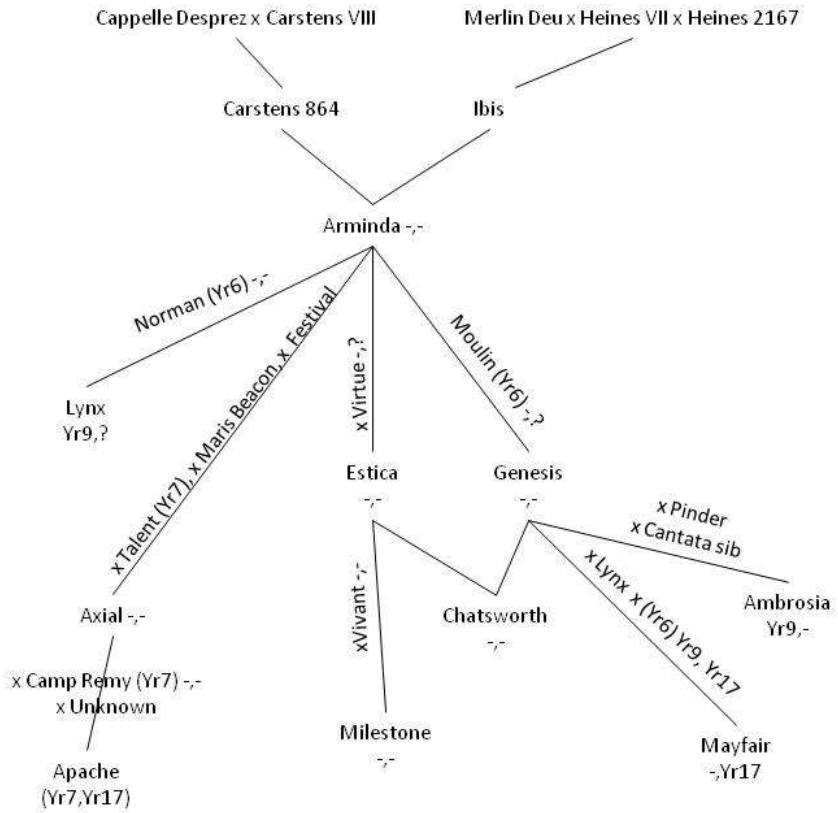


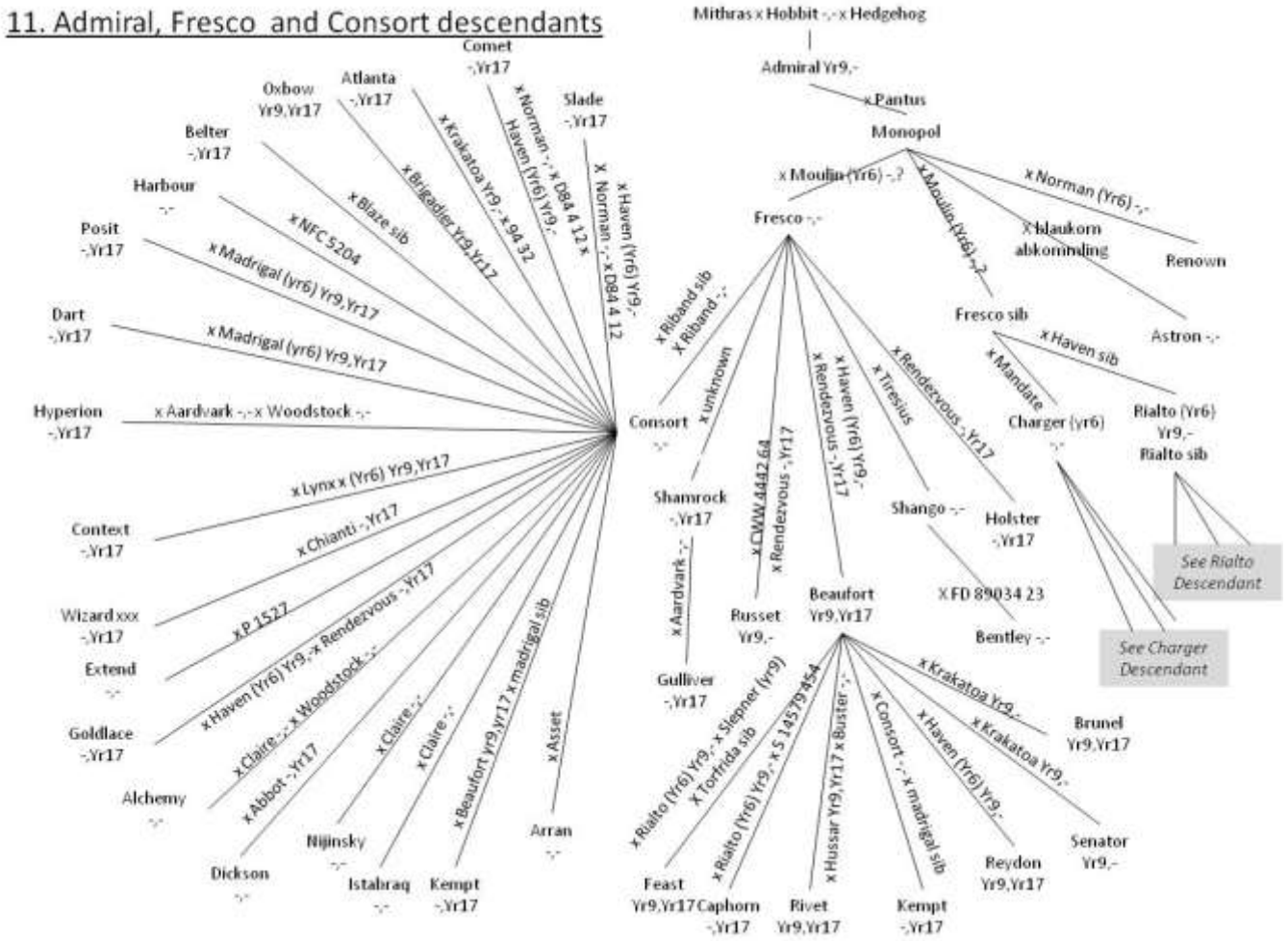
9. Lynx x (2) descendants



10. Arminda descendants

Carstens V via Carstens VIII

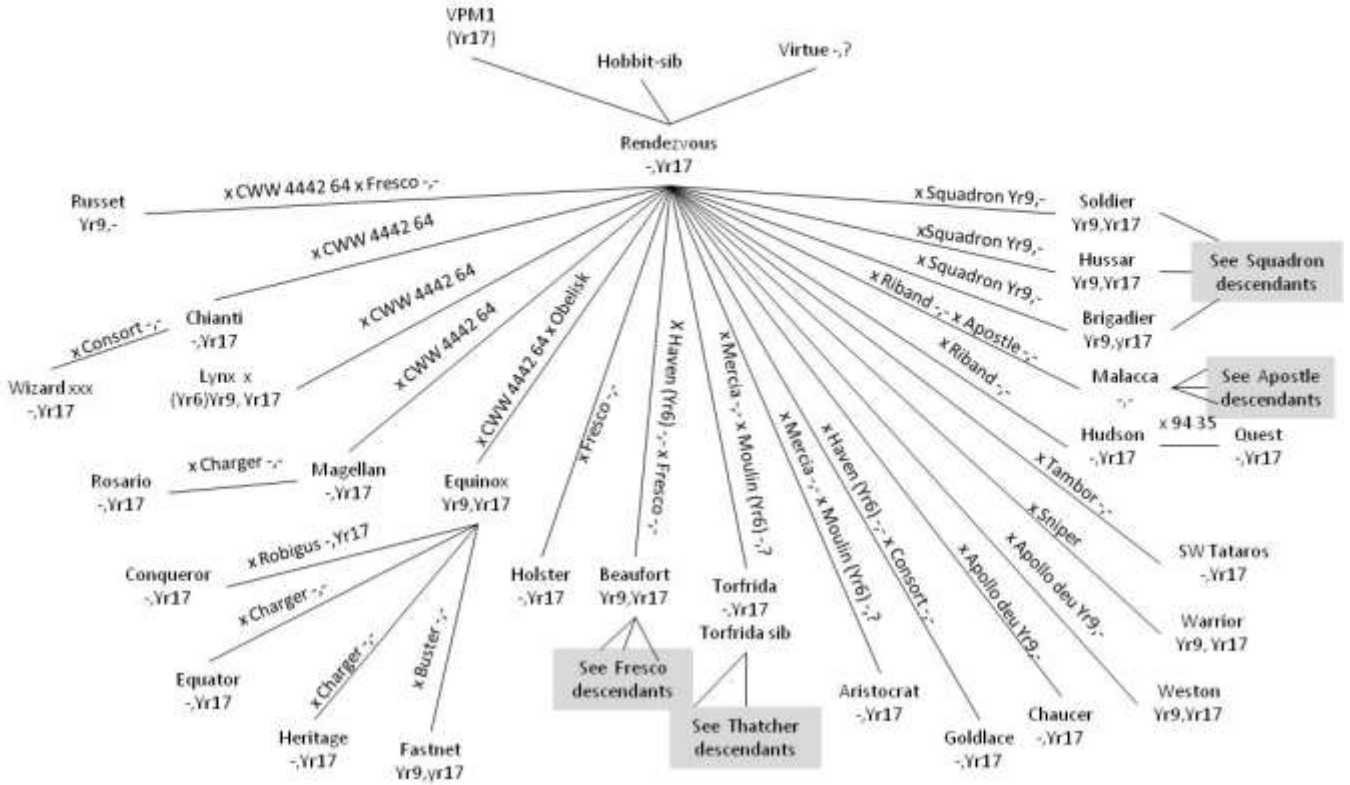



11. Admiral, Fresco and Consort descendants
Mithras x Hobbit -,- x Hedgehog
Admiral Yr9,-
x Pantus
Monopol
x Moulin (Yr6) -,?
x Moulin (Yr6) -,?
x Norman (Yr6) -,-
Fresco -,-
x Islaukorn abkömmling
Renown
Fresco sib
Astron -,-
x Haven sib
x Mandate
Charger (yr6) -,-
Rialto (Yr6) Yr9,- Rialto sib
See Rialto Descendant
See Charger Descendant
x Riband sib
x Riband -,-
Consort -,-
x unknown
x CWW 4442 64
x Rendezvous -,Yr17
x Haven (Yr6) Yr9,-
x Rendezvous -,Yr17
x Tiresius
x Rendezvous -,Yr17
Shamrock -,Yr17
Shango -,-
Holster -,Yr17
x Aardvark -,-
Russet Yr9,-
Beaufort Yr9,Yr17
X FD 89034 23
Bentley -,-
Gulliver -,Yr17
x Krakatoa Yr9,-
Brunel Yr9,Yr17
x Krakatoa Yr9,-
Senator Yr9,-
x Rialto (Yr6) Yr9,- x Siepner (yr9) x Torfrida sib
x Rialto (Yr6) Yr9,- x S 145794 54
x Hussar Yr9,Yr17 x Buster -,-
x Consort -,- x madrigal sib
x Haven (Yr6) Yr9,-
Feast Yr9,Yr17
Caphorn -,Yr17
Rivet Yr9,Yr17
Kempt -,Yr17
Reydon Yr9,Yr17
Comet -,Yr17
Atlanta -,Yr17
Slade -,Yr17
Oxbow Yr9,Yr17
Belter -,Yr17
Harbour -,-
Posit -,Yr17
Dart -,Yr17
Hyperion -,Yr17
Context -,Yr17
Wizard xxx -,Yr17
Extend -,-
Goldlace -,Yr17
Alchemy -,-
Dickson -,-
Nijinsky -,-
Istabraq -,-
Kempt -,Yr17
Arran -,-
x Norman -,- x Haven (Yr6) Yr9,-
x Krakatoa Yr9,- x 94 32 Brigadier Yr9,Yr12
x Norman -,- x D84 4 12 x Haven (Yr6) Yr9,-
x Blaze sib
x NFC 5204
x Madrigal (yr6) Yr9,Yr17
x Madrigal (yr6) Yr9,Yr17
x Aardvark -,- x Woodstock -,-
x Lynx x (Yr6) Yr9,Yr17
x Chianti -,Yr17
x P 1527
x Haven (Yr6) Yr9,- x Rendezvous -,Yr17
x Claire -,- x Woodstock -,-
x Abbot -,Yr17
x Claire -,-
x Claire -,-
x Beaufort yr9,yr17 x madrigal sib
x Asset

24-

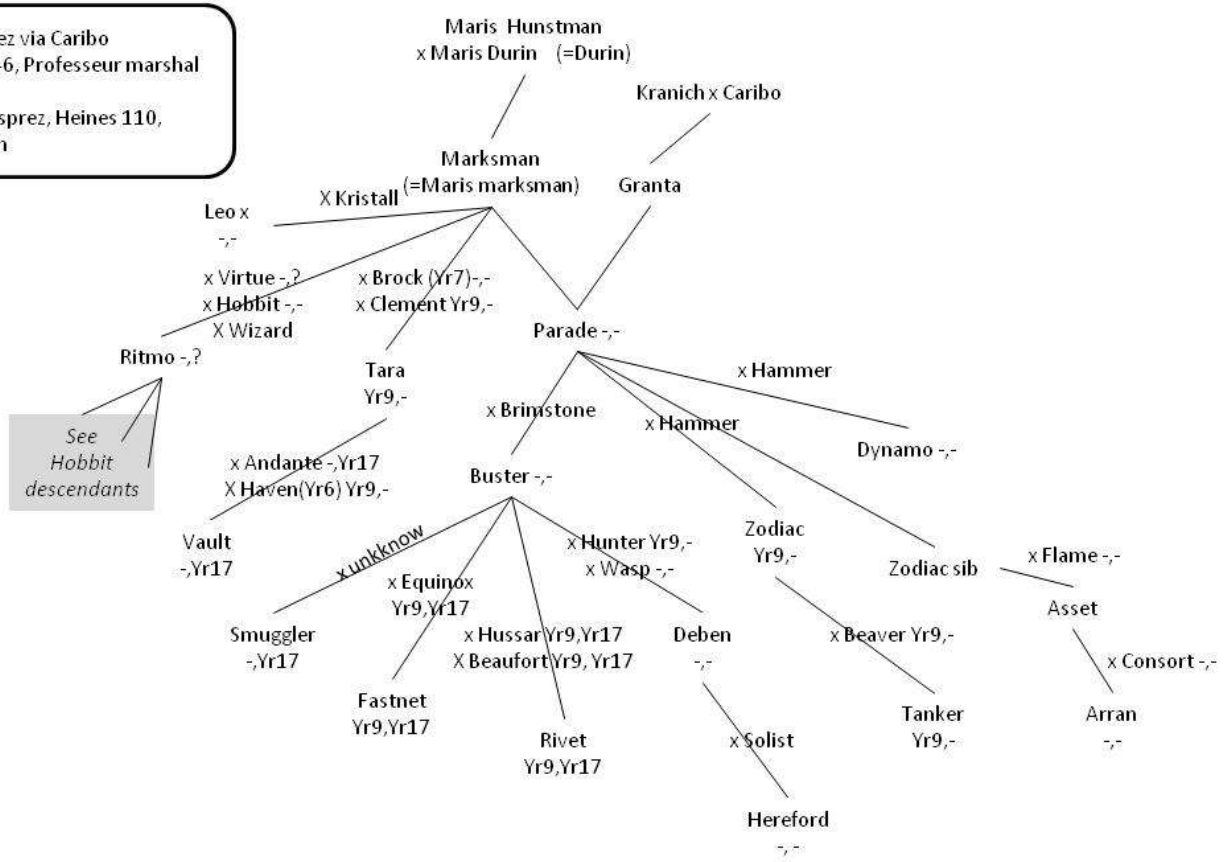
12. Rendezvous descendants

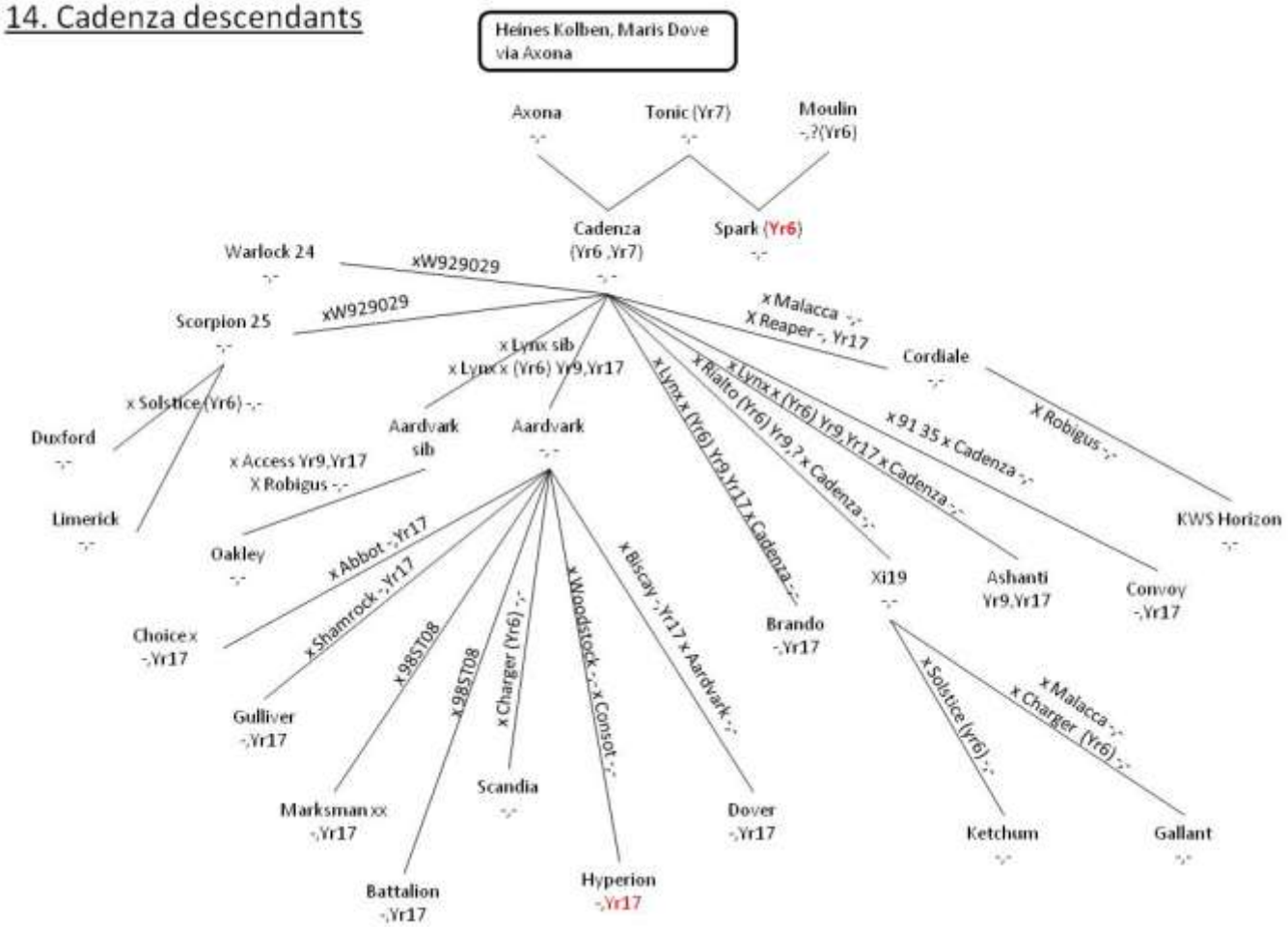
Cappelle Desprez , Hybrid 46, Vilmorin 23, Heines 110, Nord Desprez, Professeur Marshal via Virtue
Cappelle Desprez , Heines 110, Nord Desprez, Professeur marshal via Hobbit Sib



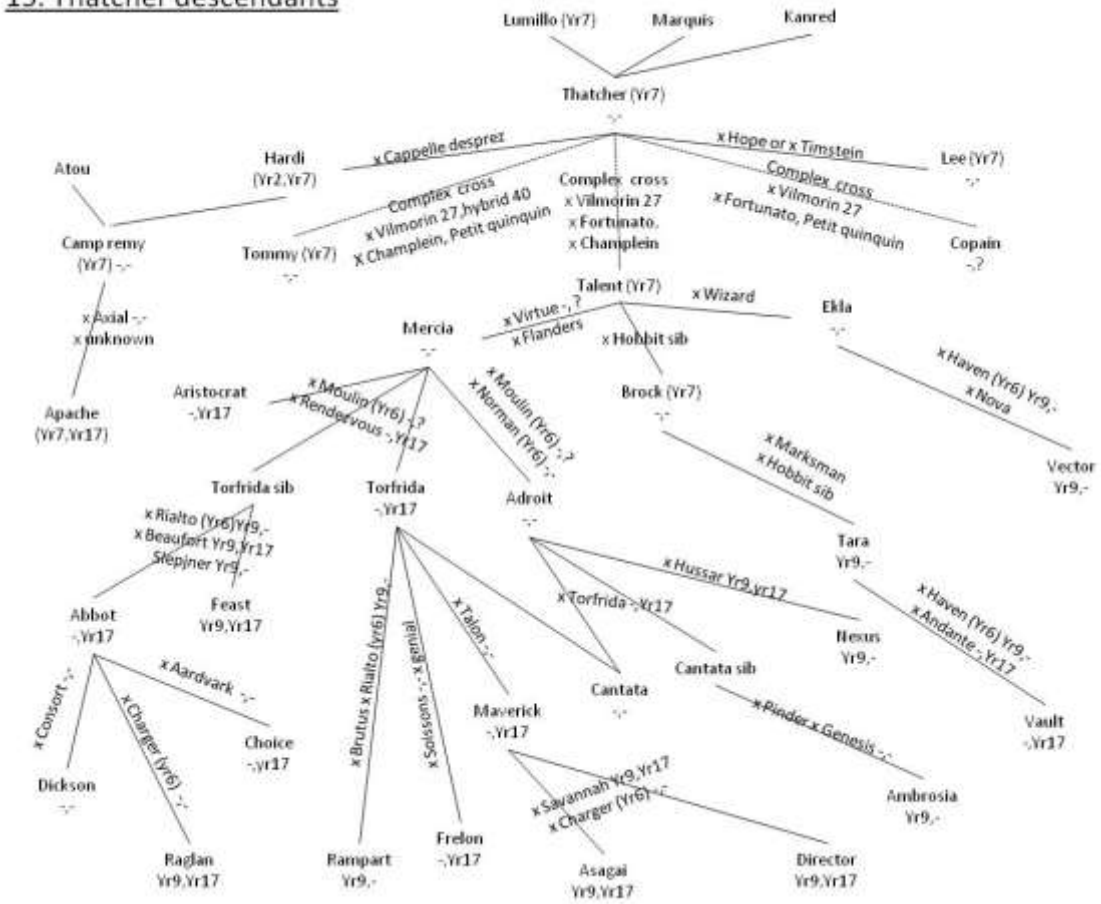
13. Marksman descendants

Carstens V, Cappelle Desprez via Caribo
Cappelle Desprez , Hybrid 46, Professeur marshal via Maris Hunstman
Cappelle Desprez , Nord desprez, Heines 110, Vilmorin 29 via Maris Durin

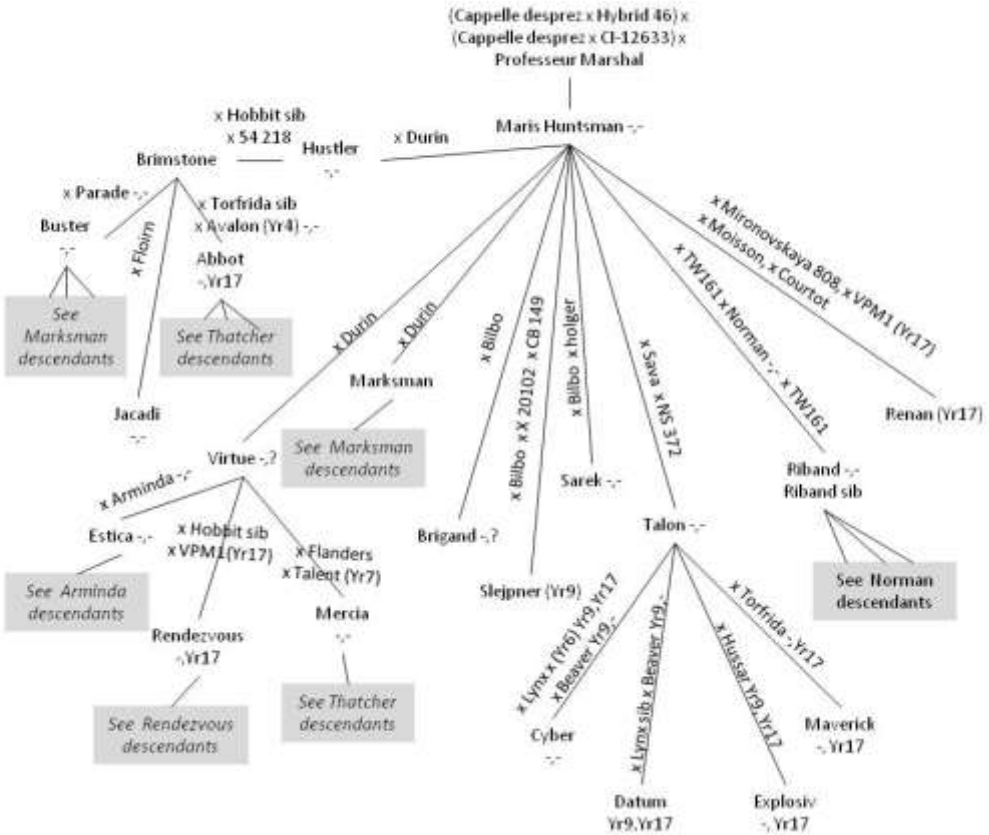


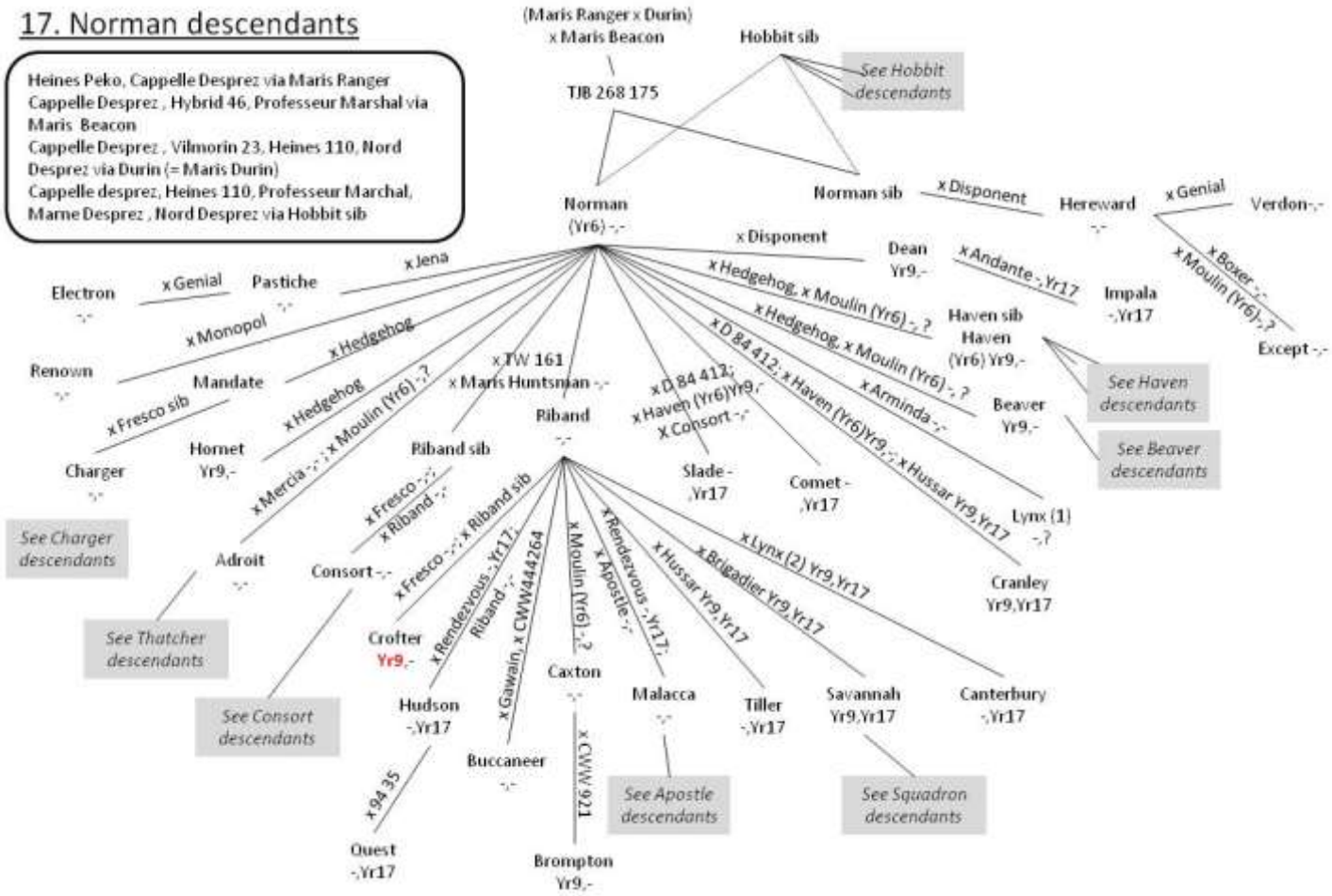

14. Cadenza descendants
Heines Kolben, Maris Dove via Axona
Axona -,-
Tonic (Yr7) -,-
Moulin -,?(Yr6)
Cadenza (Yr6 ,Yr7) -,-
Spark (Yr6) -,-
Warlock 24 -,-
xW929029
xW929029
Scorpion 25 -,-
x Malacca -,-
X Reaper -, Yr17
Cordiale -,-
x Lynx sib
x Lynx x (Yr6) Yr9,Yr17
x Lynx x (Yr6) Yr9,Yr17 x Cadenza -,-
x Rialto (Yr6) Yr9,? x Cadenza -,-
x 91 35 x Cadenza -,-
X Robigus -,-
x Solstice (Yr6) -,-
Duxford -,-
Limerick -,-
Aardvark sib
Aardvark -,-
x Access Yr9,Yr17
X Robigus -,-
Oakley -,-
KWS Horizon -,-
x Abbot -,Yr17
x Shamrock -,Yr17
Choice x -,Yr17
Gulliver -,Yr17
x 98ST08
x 98ST08
Marksman xx -,Yr17
Battalion -,Yr17
x Charger (Yr6) -,-
Scandia -,-
x Woodstock -,- x Consot -,-
Hyperion -,Yr17
x Biscay -,Yr17 x Aardvark -,-
Dover -,Yr17
Brando -,Yr17
Xi19 -,-
Ashanti Yr9,Yr17
Convoy -,Yr17
x Solstice (yr6) -,-
x Malacca -,-
x Charger (Yr6) -,-
Ketchum -,-
Gallant -,-

15. Thatcher descendants

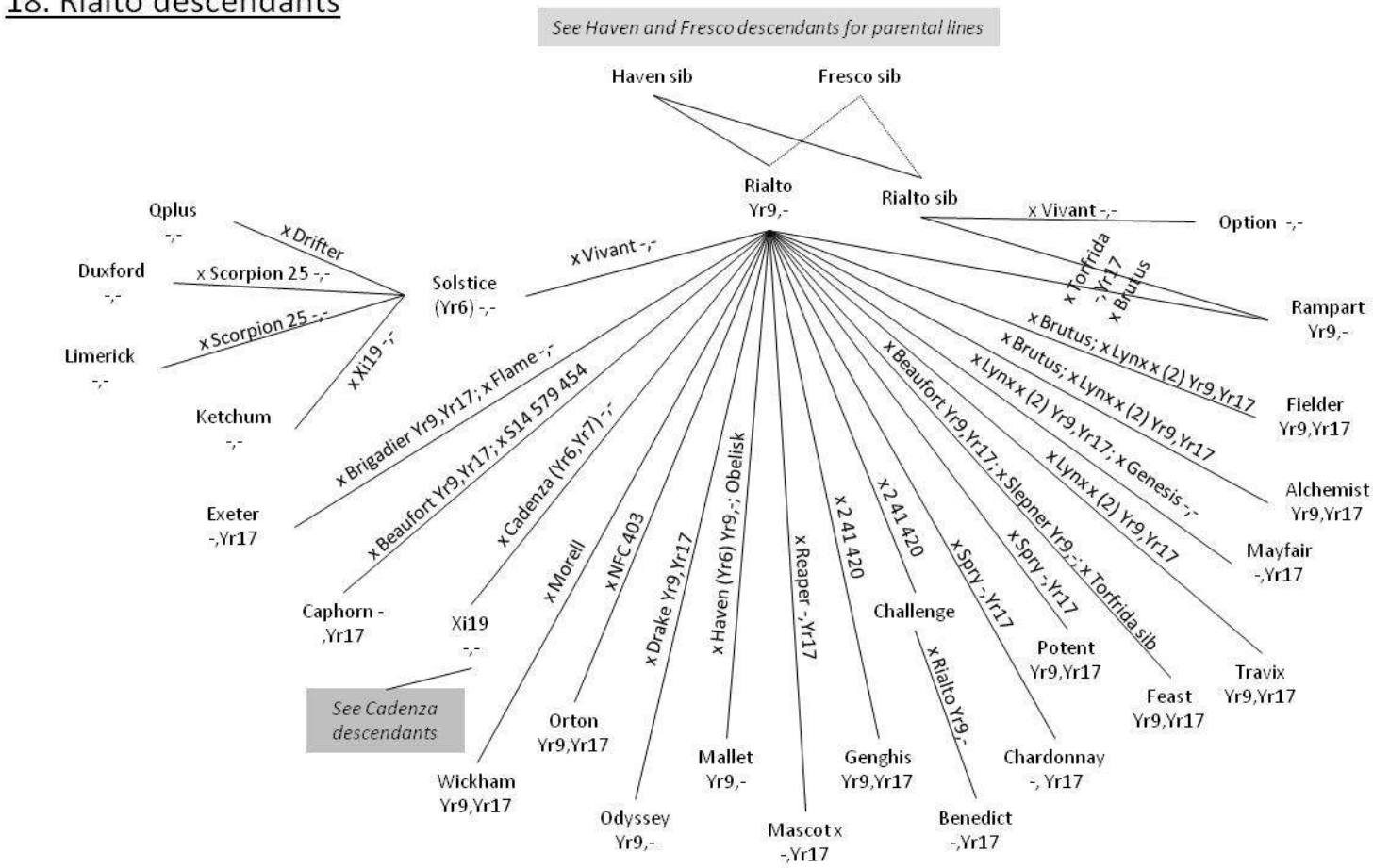


16. Maris huntsman descendants

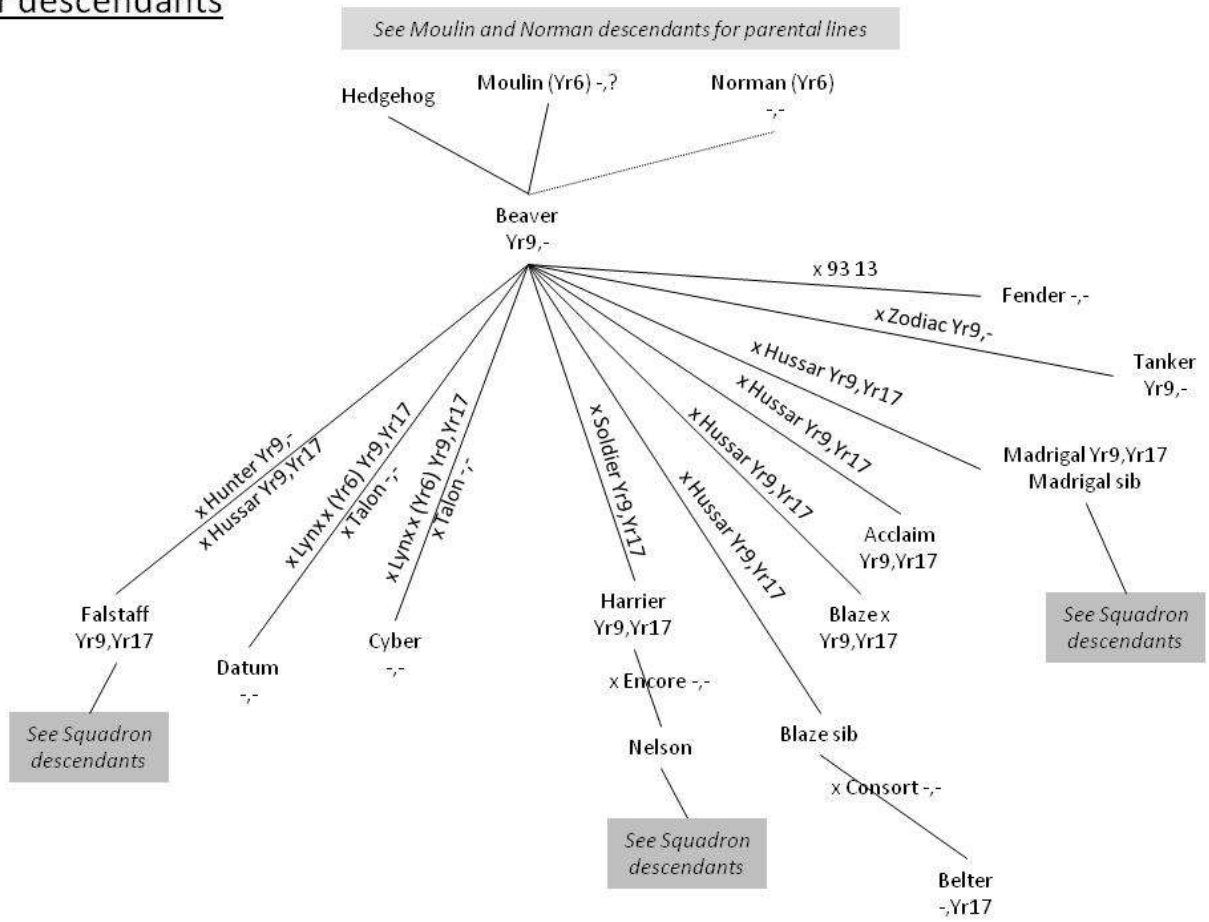



17. Norman descendants
Heines Peko, Cappelle Desprez via Maris Ranger
Cappelle Desprez , Hybrid 46, Professeur Marshal via Maris Beacon
Cappelle Desprez , Vilmorin 23, Heines 110, Nord Desprez via Durin (= Maris Durin)
Cappelle desprez, Heines 110, Professeur Marchal, Marne Desprez , Nord Desprez via Hobbit sib
(Maris Ranger x Durin) x Maris Beacon
TJB 268 175
Hobbit sib
See Hobbit descendants
Norman (Yr6) -,-
Norman sib
x Disponent
Hereward -,-
x Genial
Verdon-,-
x Boxer -,-
x Moulin (Yr6) -,?
Except -,-
x Disponent
Dean Yr9,-
x Andante -,Yr17
Impala -,Yr17
Haven sib
Haven (Yr6) Yr9,-
See Haven descendants
See Beaver descendants
Beaver Yr9,-
x Jena
Electron -,-
x Genial
Pastiche -,-
x Monopol
Renown -,-
x Hedgehog
Mandate -,-
x Fresco sib
Charger -,-
x Hedgehog
Hornet Yr9,-
x Mercia -,-; x Moulin (Yr6) -,?
x TW 161
x Maris Huntsman -,-
Riband -,-
Riband sib
x Fresco -,-; x Riband -,-
x Hedgehog, x Moulin (Yr6) -,?
x Hedgehog, x Moulin (Yr6) -,?
x D 84 412; x Haven (Yr6)Yr9,-; x Consort -,-
x D 84 412; x Haven (Yr6)Yr9,-; x Arminda -,-
Slade -,Yr17
Comet -,Yr17
Lynx (1) -,?
x D 84 412; x Haven (Yr6)Yr9,-; x Hussar Yr9,Yr17
Cranley Yr9,Yr17
See Charger descendants
Adroit -,-
See Thatcher descendants
Consort -,-
See Consort descendants
x Fresco -,-; x Riband sib
x Rendezvous -,Yr17; Riband -,-
x Gawain, x CWW444264
x Moulin (Yr6) -,?
x Apostle -,-
x Rendezvous -,Yr17; -
x Hussar Yr9,Yr17
x Brigadier Yr9,Yr17
x Lynx (2) Yr9,Yr17
Crofter Yr9,-
Hudson -,Yr17
Buccaneer -,-
Caxton -,-
Malacca -,-
Tiller -,Yr17
Savannah Yr9,Yr17
Canterbury -,Yr17
x 94 35
Quest -,Yr17
x CWW 921
Brompton Yr9,-
See Apostle descendants
See Squadron descendants

18. Rialto descendants

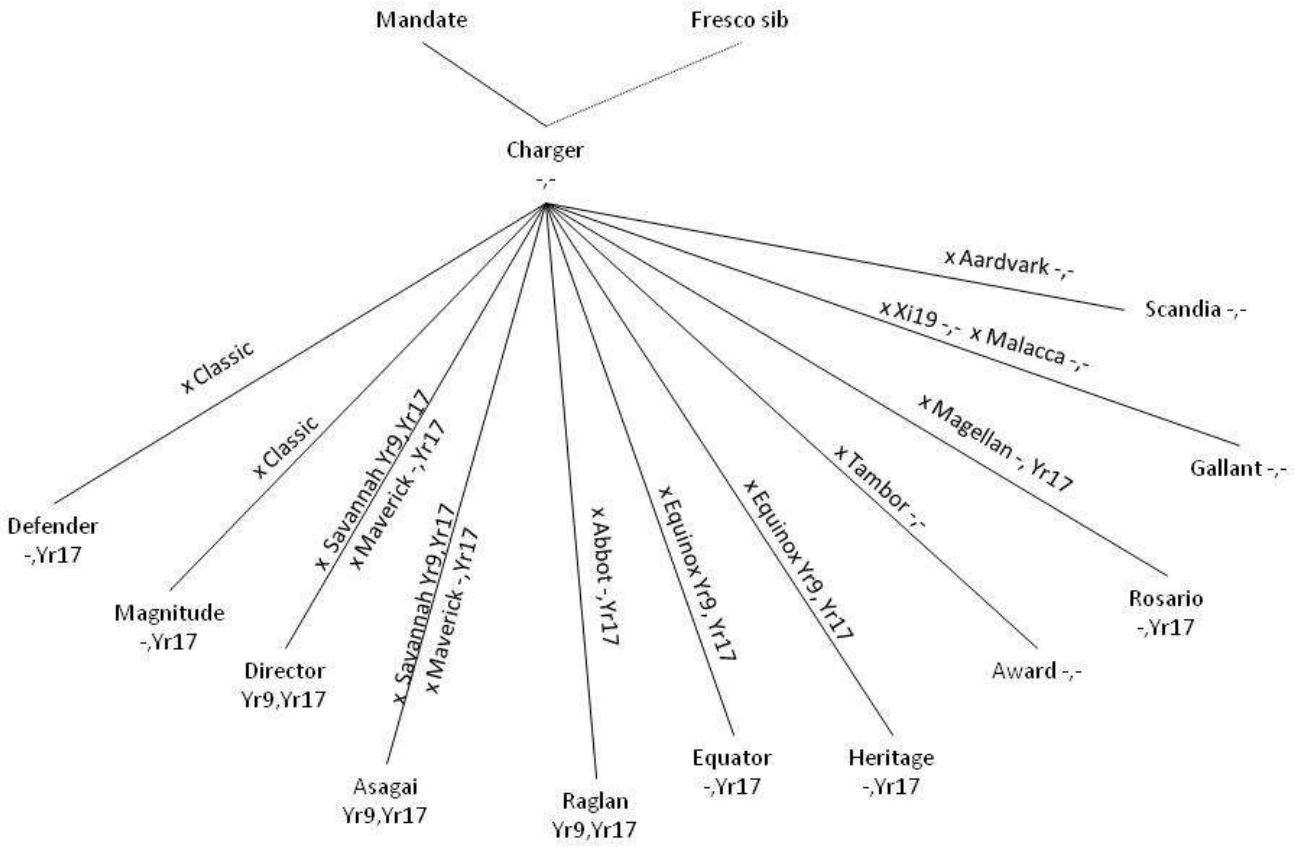


19. Beaver descendants



20. Charger descendants

See Haven and Fresco descendants for parental lines



APPENDIX 9: *Rht1 Rht2* assays from Wilhelm (2011)

Primers product expected for *Rht1 Rht2* assay

Locus/allele	Primer pair	Sequences 5'-3'	PCR products expected
Rht1/Rht-B1a (tall)	Rht-B-F1 Rht-B1a-R2	AGG CAA GCA AAA GCT TGA GA CCA TGG CCA TCT CCA GAT G	265 bp
Rht1/Rht-B1b (semi-dwarf)	Rht-B-F1 Rht-B1b-R2	AGG CAA GCA AAA GCT TGA GA CCC ATG GCC ATC TCC AGA TA	265 bp
Rht2/Rht-D1a (tall)	Rht-D-F5 Rht-D1a	GCT CGT TCT CCT CCC AGT TC ATG GCC ATC TCG AGC TGT TC	385 bp
Rht2/Rht-D1b (semi-dwarf)	Rht-D-F5 Rht-D1b-R2	GCT CGT TCT CCT CCC AGT TC CAT GGC CAT CTC GAG CTG TTA	385 bp

The PCR reactions was performed in 10µl volumes containing: 2 µl Green GoTaq reaction buffer (Promega) 5X, 0.2 µl dNTP (40mM), 0.3 µl Glycerol, 1 µl Forward primer, 1 µl Reverse primer, 0.25 µl *Taq* Polymerase 1 µl DNA (20ng/ µl). The reaction profile was 95°C for 5 min; followed by 40 cycles of 95°C for 30 s, 60°C for 30 s, 72°C for 30 s, with a final extension at 72°C for 10 min. Products were separated on 1.5% agarose gels in TE buffer visualized under UV light with ethidium bromide.

APPENDIX 10 : *Ppd-D1* assay from Beales et al. (2007)

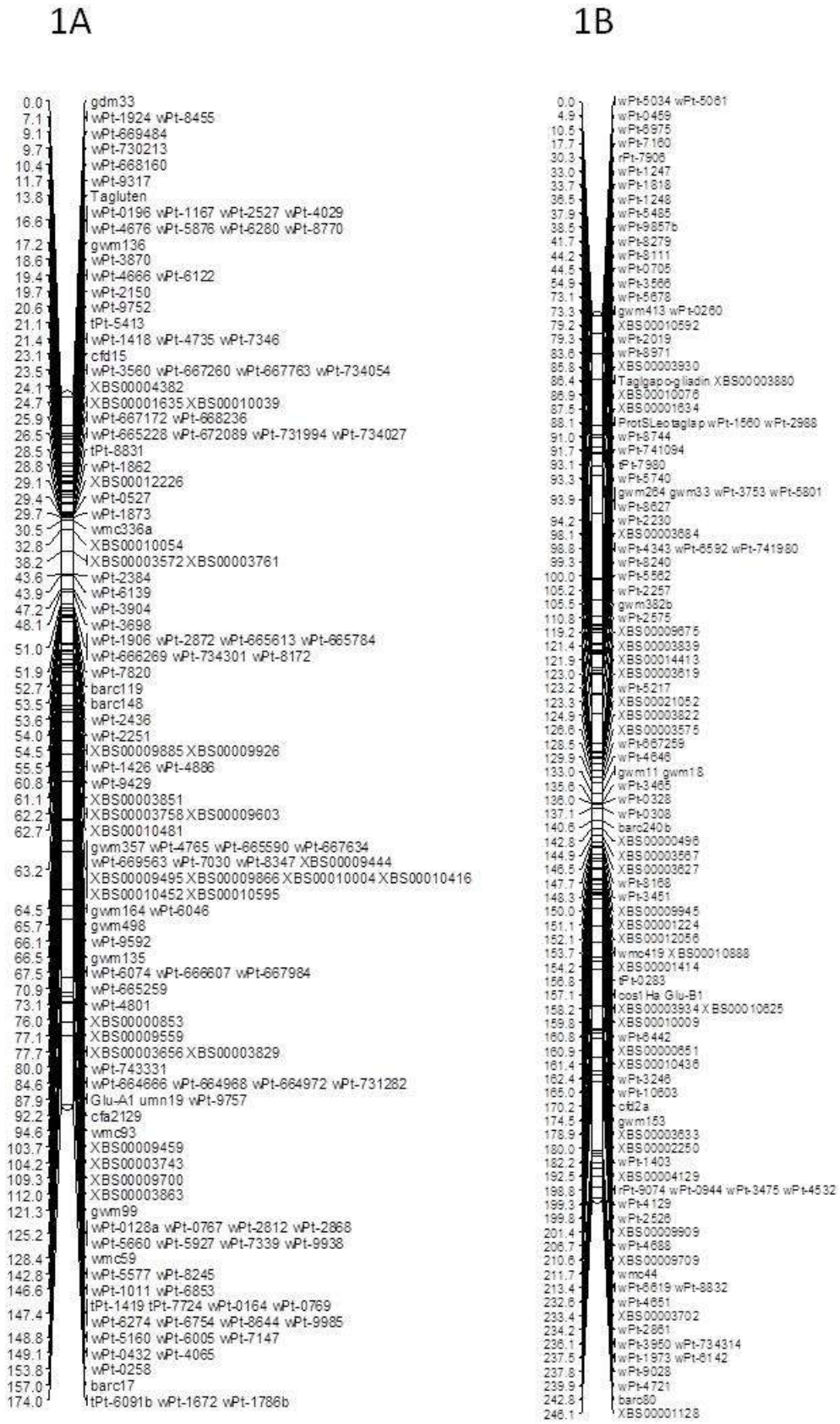
Primers product expected for *PPD-D1* assay

Locus/Target	Allele	Primer pair	Sequences 5'-3'	PCR products expected
PRR gene intact (photoperiod sensitive)	*Ppd-D1*	Ppd-D1_F Ppd-D1_R1	Acgcctcccactacactg gttggttcaaacagagagc	414 bp
PRR gene with 2kb deletion (photoperiod insensitive)	*Ppd-D1a*	Ppd-D1_F Ppd-D1_R2	Acgcctcccactacactg cactggtggtagctgagatt	288 bp

The PCR reaction was carried out in 10µl containing 1µM of each primers (a common forward primer Ppd-D1_F two specific reverse primers Ppd-D1_R1 Ppd-D1_R2), 200 µM of each dNTP, 0.5U of *Faststart Taq* (Roche), 1µl *Faststart* 10x buffer with $MgCl_2$ approximatly 10ng of DNA template. The reaction profile was 95°C for 8 min; followed by 40 cycles of 95°C for 30 s, 55°C for 30 s, 72°C for 40 s, with a final extension at 72°C for 10 min. Products were separated on 1.5% agarose gels in TE buffer visualized under UV light with ethidium bromide.

APPENDIX 11: Consensus map constructed in MergeMap based on genetic maps Avalon x Cadenza, Solstice x Robigus, Claire x Lemhi UC1110 x PI610750

The linkage groups views were produced in Mapchart 2.2 (Voorrips, 2002).



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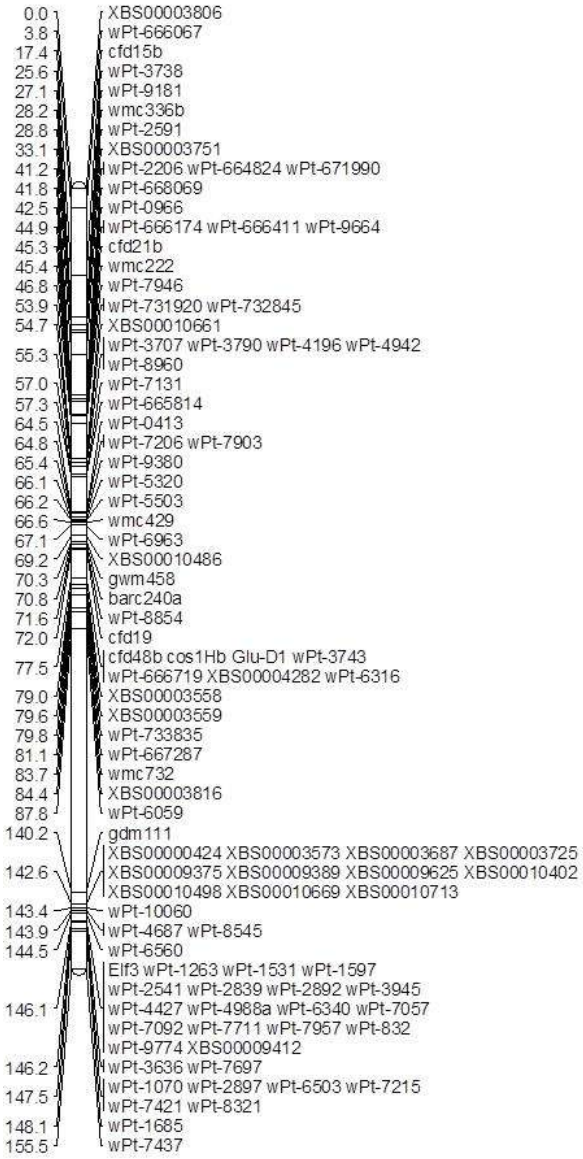
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33.1 XBS00003751
41.2 wPt-2206 wPt-664824 wPt-671990
41.8 wPt-668069
42.5 wPt-0966
44.9 wPt-666174 wPt-666411 wPt-9664
45.3 cfd21b
45.4 wmc222
46.8 wPt-7946
53.9 wPt-731920 wPt-732845
54.7 XBS00010661
55.3 wPt-3707 wPt-3790 wPt-4196 wPt-4942
wPt-8960
57.0 wPt-7131
57.3 wPt-665814
64.5 wPt-0413
64.8 wPt-7206 wPt-7903
65.4 wPt-9380
66.1 wPt-5320
66.2 wPt-5503
66.6 wmc429
67.1 wPt-6963
69.2 XBS00010486
70.3 gwm458
70.8 barc240a
71.6 wPt-8854
72.0 cfd19
77.5 cfd48b cos1Hb Glu-D1 wPt-3743
wPt-666719 XBS00004282 wPt-6316
79.0 XBS00003558
79.6 XBS00003559
79.8 wPt-733835
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83.7 wmc732
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87.8 wPt-6059
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142.6 XBS00000424 XBS00003573 XBS00003687 XBS00003725
XBS00009375 XBS00009389 XBS00009625 XBS00010402
XBS00010498 XBS00010669 XBS00010713
143.4 wPt-10060
143.9 wPt-4687 wPt-8545
144.5 wPt-6560
Elf3 wPt-1263 wPt-1531 wPt-1597
wPt-2541 wPt-2839 wPt-2892 wPt-3945
146.1 wPt-4427 wPt-4988a wPt-6340 wPt-7057
wPt-7092 wPt-7711 wPt-7957 wPt-832
wPt-9774 XBS00009412
146.2 wPt-3636 wPt-7697
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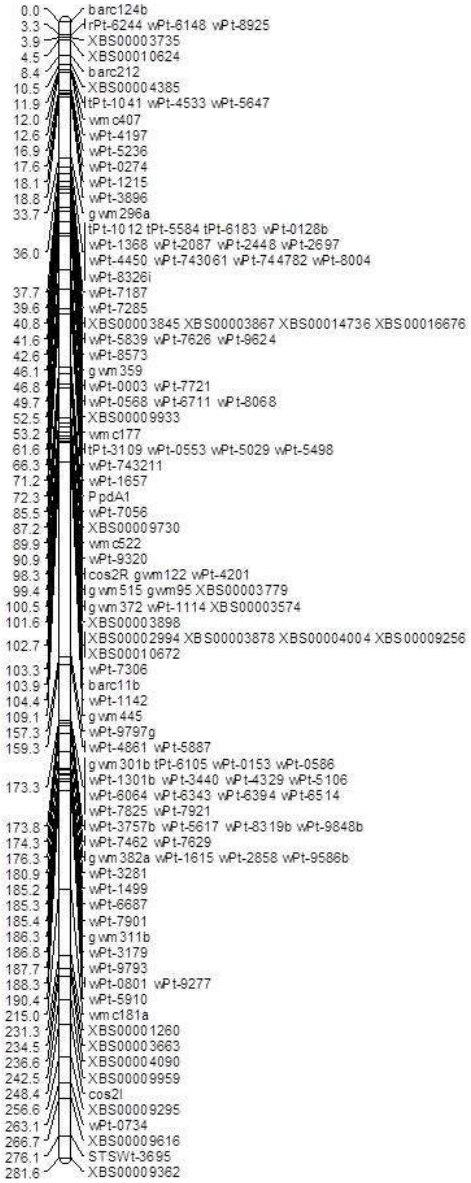
wPt-7421 wPt-8321
148.1 wPt-1685
155.5 wPt-7437

2A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12.0 wmc407
12.6 wPt-4197
16.9 wPt-5236
17.6 wPt-0274
18.1 wPt-1215
18.8 wPt-3896
33.7 gwm296a
tPt-1012 tPt-5584 tPt-6183 wPt-0128b
wPt-1368 wPt-2087 wPt-2448 wPt-2697
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wPt-8326i
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53.2 wmc177
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wPt-7825 wPt-7921
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174.3 wPt-7462 wPt-7629
176.3 gwm382a wPt-1615 wPt-2858 wPt-9586b
180.9 wPt-3281
185.2 wPt-1499
185.3 wPt-6687
185.4 wPt-7901
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234.5 XBS00003663
236.6 XBS00004090
242.5 XBS00009959
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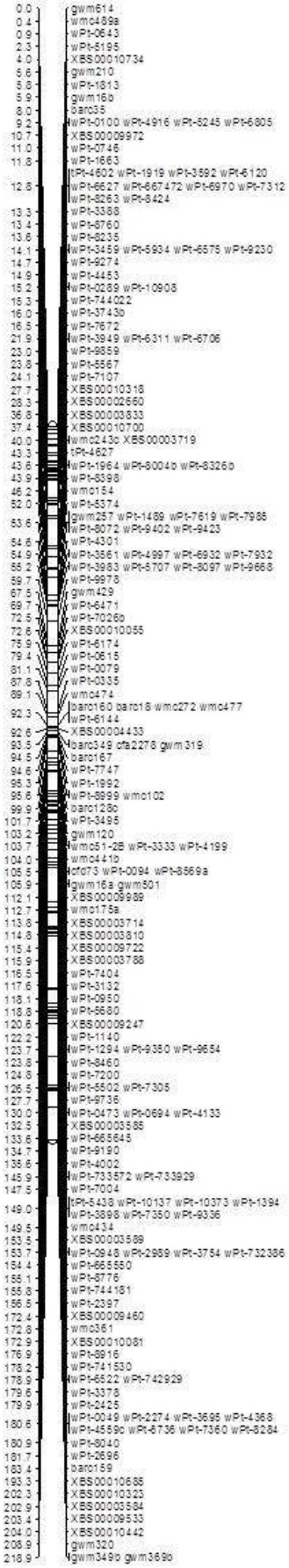
2B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37.4 XBS00010700
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94.5 barc167
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95.3 wPt-1992
95.6 wPt-8999 wmc102
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103.7 wmc51-2B wPt-3333 wPt-4199
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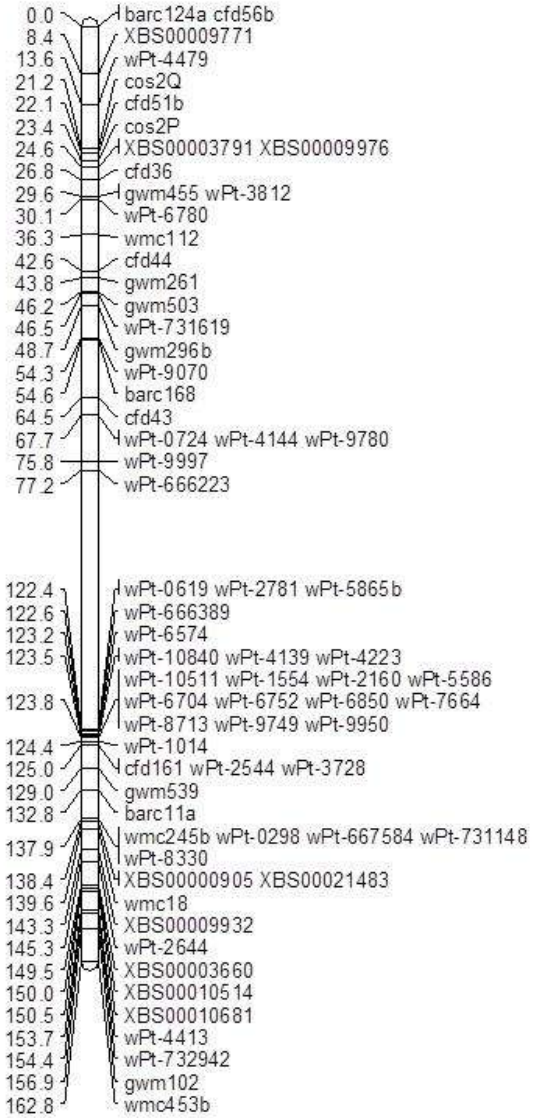
2D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139.6 wmc18
143.3 XBS00009932
145.3 wPt-2644
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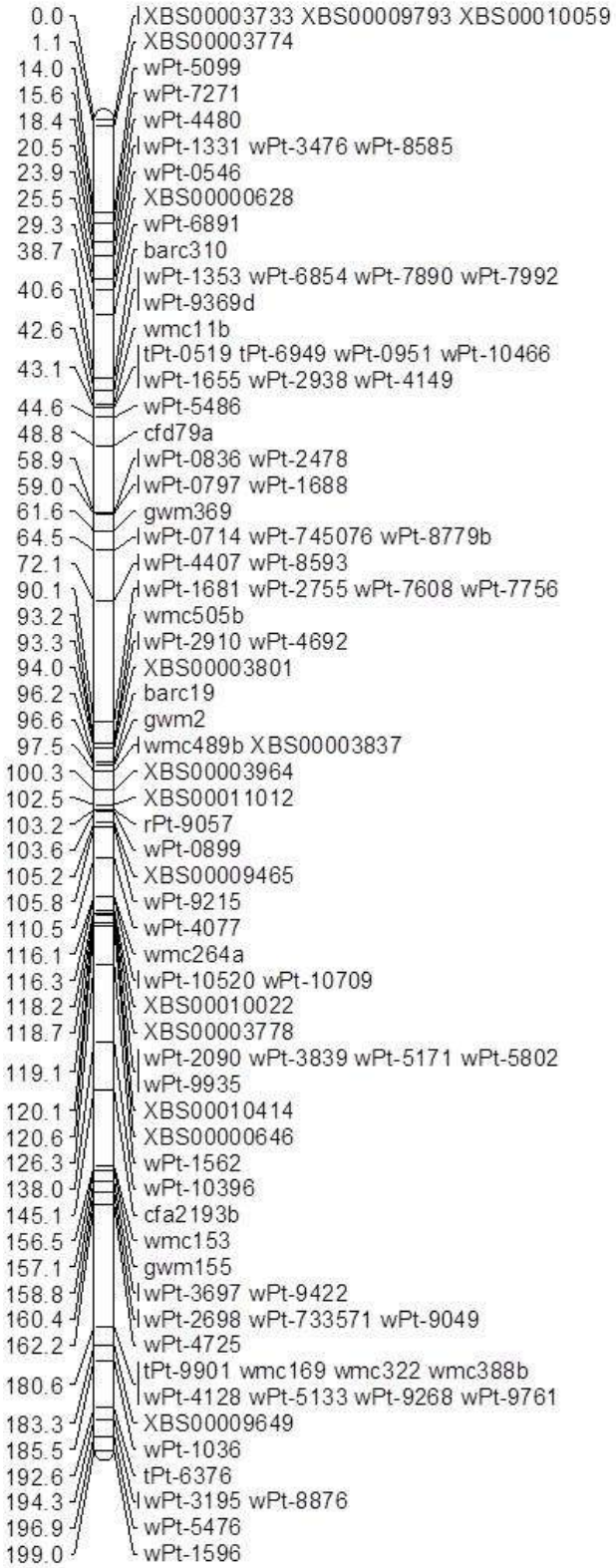
3A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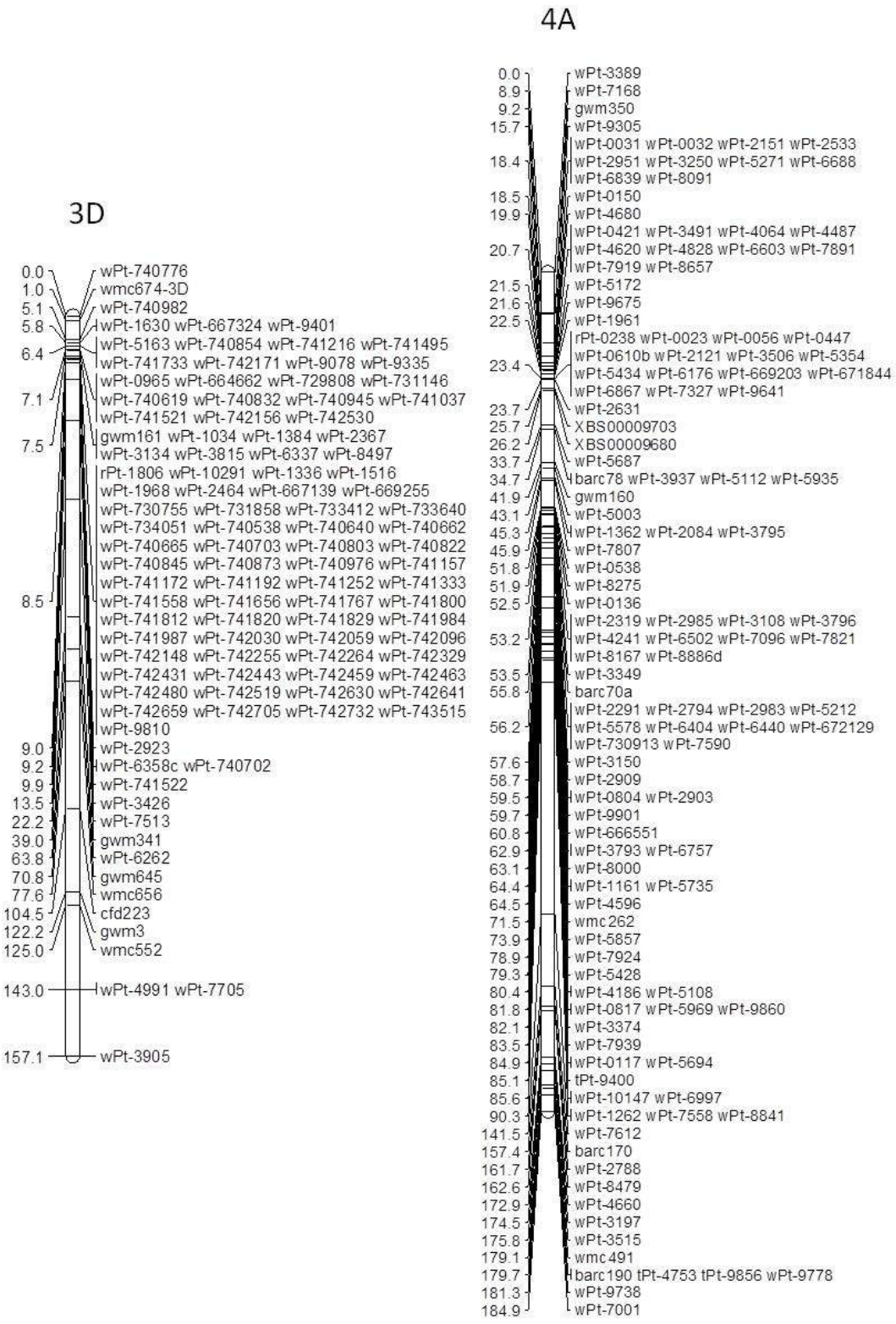
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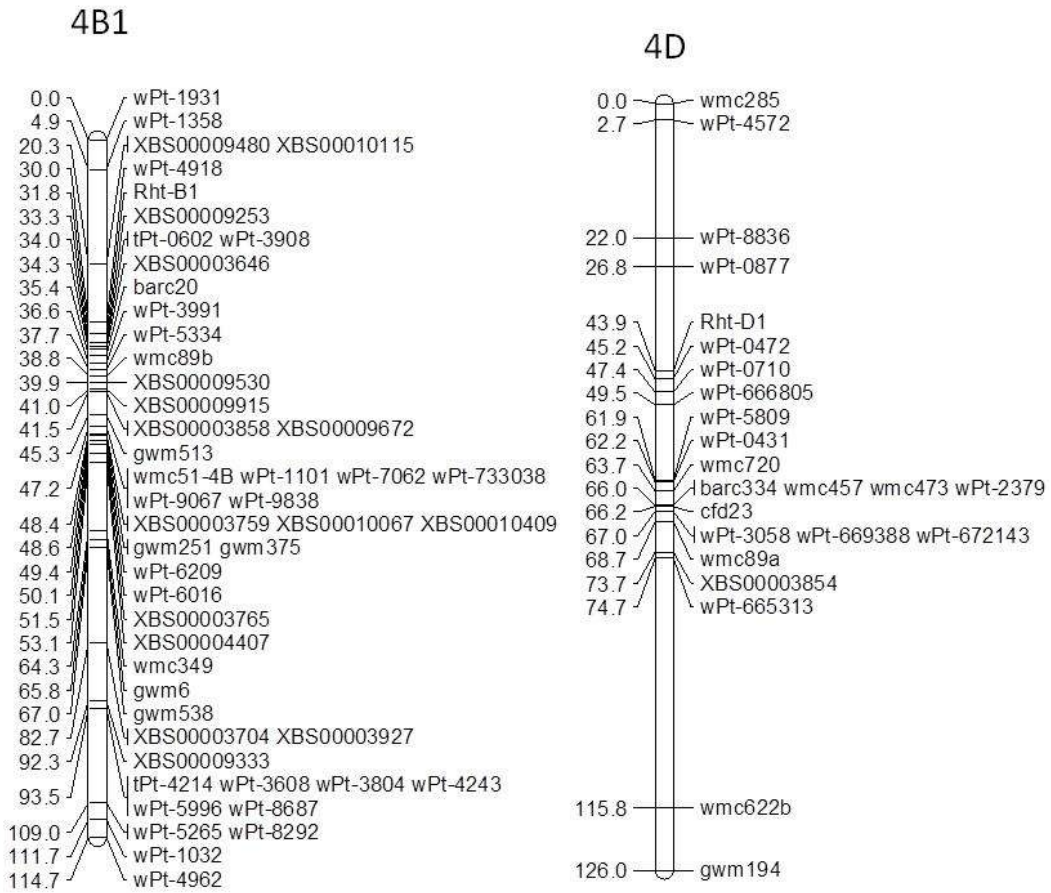
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38.8 wmc89b
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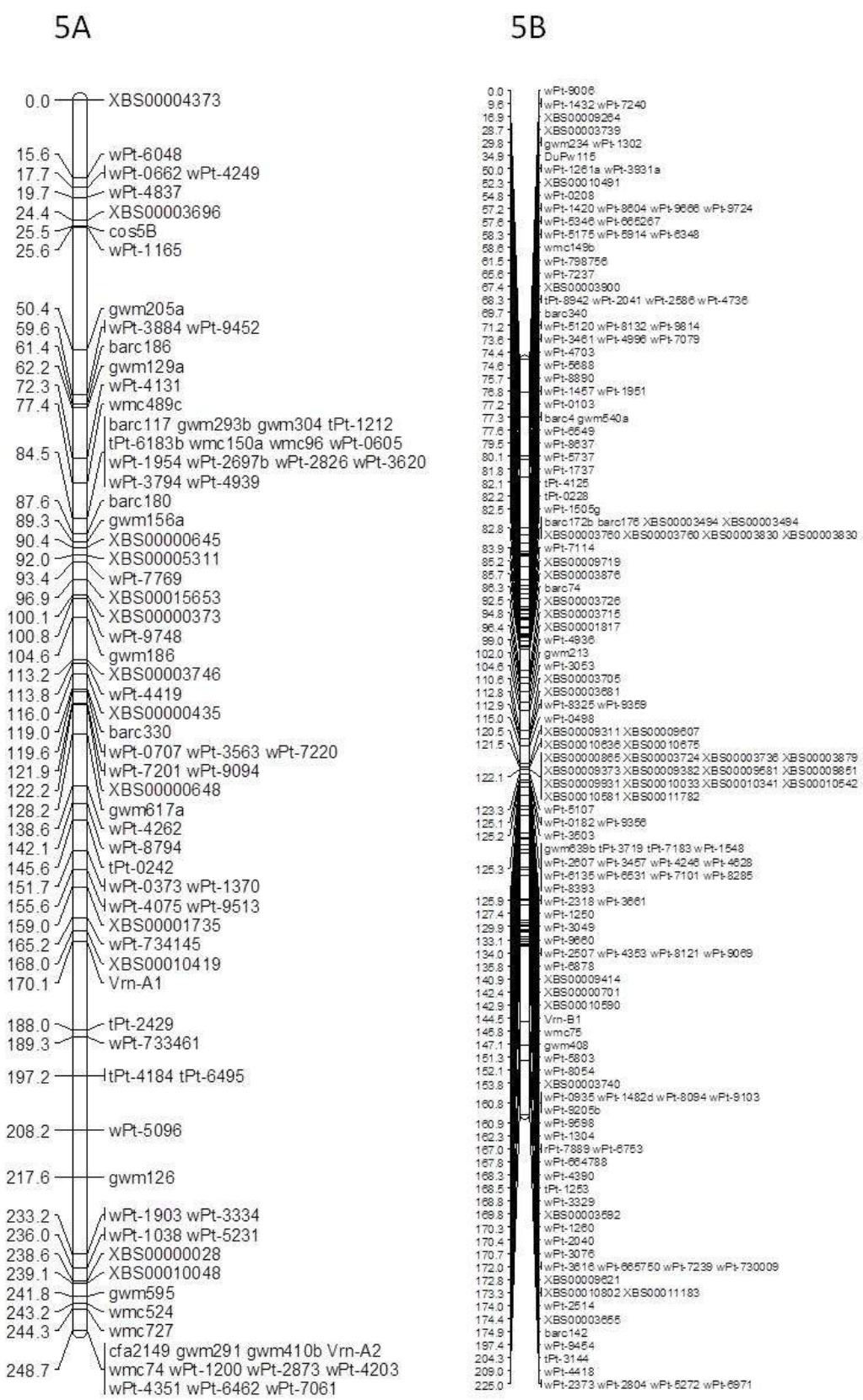
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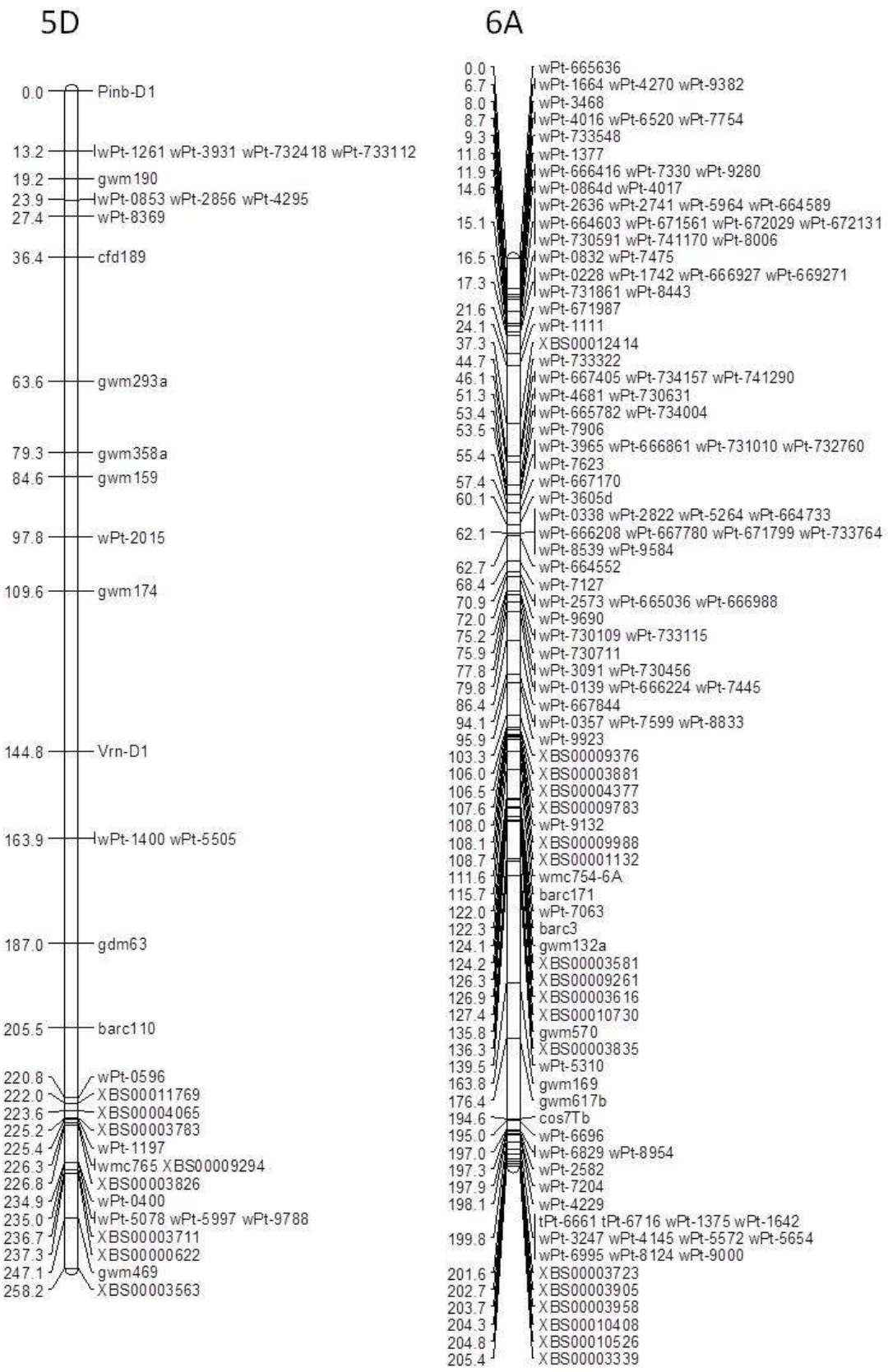
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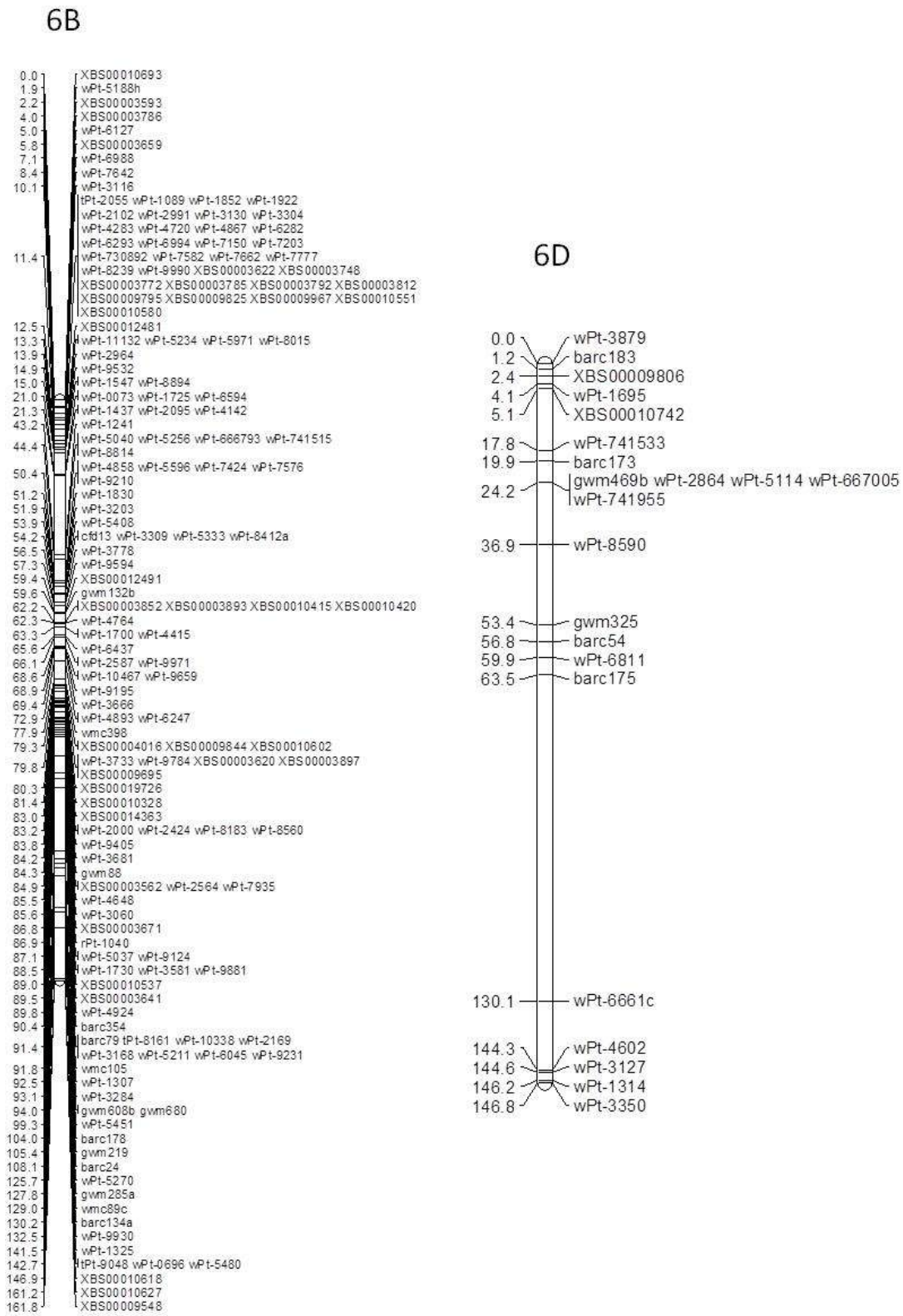
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88.5 wPt-1730 wPt-3581 wPt-9881
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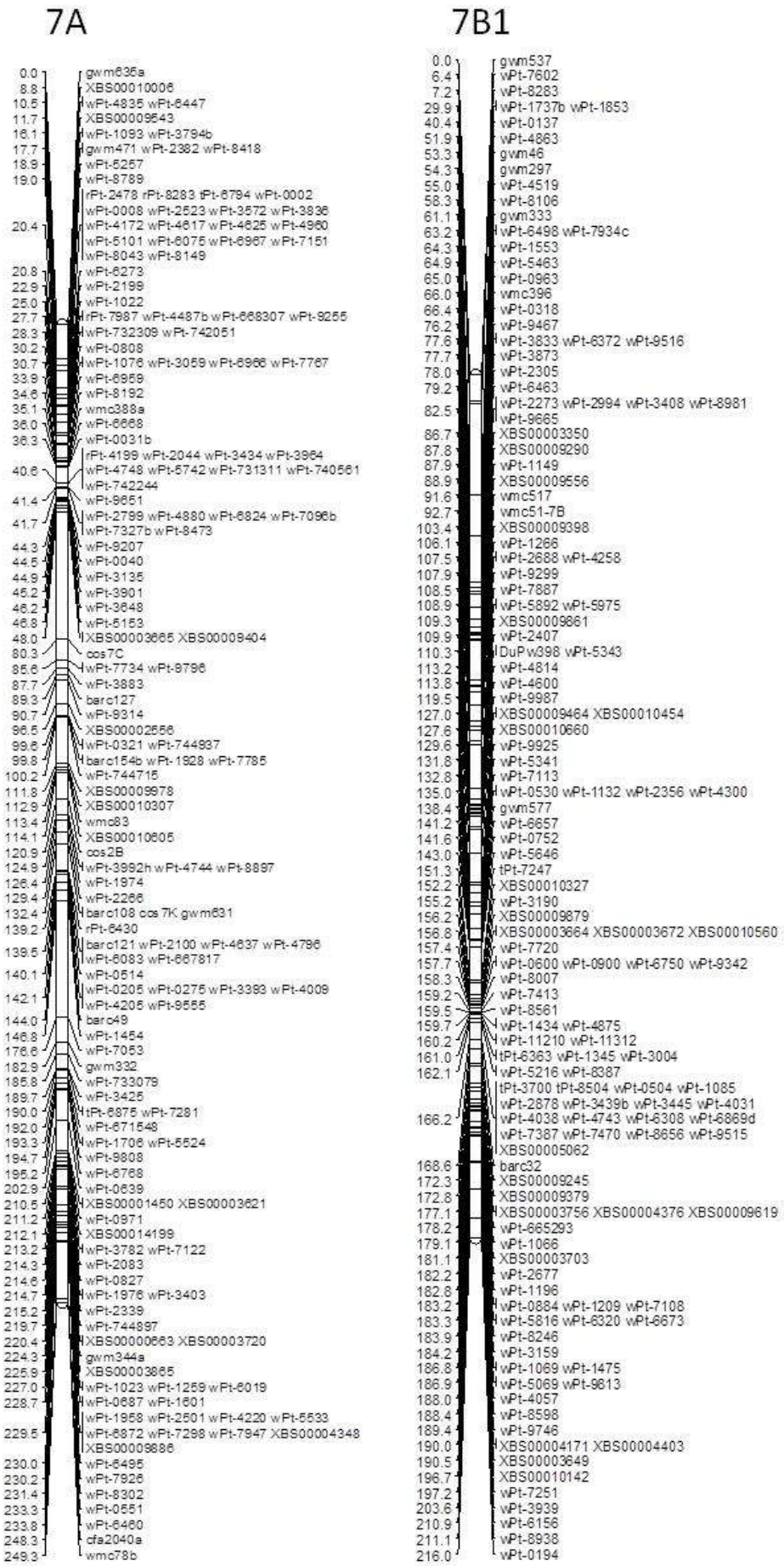
90.4 barc354
91.4 barc79 tPt-8161 wPt-10338 wPt-2169
wPt-3168 wPt-5211 wPt-6045 wPt-9231
91.8 wmc105
92.5 wPt-1307
93.1 wPt-3284
94.0 gwm608b gwm680
99.3 wPt-5451
104.0 barc178
105.4 gwm219
108.1 barc24
125.7 wPt-5270
127.8 gwm285a
129.0 wmc89c
130.2 barc134a
132.5 wPt-9930
141.5 wPt-1325
142.7 tPt-9048 wPt-0696 wPt-5480
146.9 XBS00010618
161.2 XBS00010627
161.8 XBS00009548
6D
0.0 wPt-3879
1.2 barc183
2.4 XBS00009806
4.1 wPt-1695
5.1 XBS00010742
17.8 wPt-741533
19.9 barc173
24.2 gwm469b wPt-2864 wPt-5114 wPt-667005
wPt-741955
36.9 wPt-8590
53.4 gwm325
56.8 barc54
59.9 wPt-6811
63.5 barc175
130.1 wPt-6661c
144.3 wPt-4602
144.6 wPt-3127
146.2 wPt-1314
146.8 wPt-3350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28.3 wPt-732309 wPt-742051
30.2 wPt-0808
30.7 wPt-1076 wPt-3059 wPt-6966 wPt-7767
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34.6 wPt-8192
35.1 wmc388a
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36.3 wPt-0031b
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40.6 wPt-4748 wPt-5742 wPt-731311 wPt-740561
wPt-742244
41.4 wPt-9651
41.7 wPt-2799 wPt-4880 wPt-6824 wPt-7096b
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44.5 wPt-0040
44.9 wPt-3135
45.2 wPt-3901
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46.8 wPt-5153
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80.3 cos7C
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87.7 wPt-3883
89.3 barc127
90.7 wPt-9314
96.5 XBS00002556
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113.4 wmc83
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120.9 cos2B
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126.4 wPt-1974
129.4 wPt-2266
132.4 barc108 cos7K gwm631
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139.5 barc121 wPt-2100 wPt-4637 wPt-4796
wPt-6083 wPt-667817
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142.1 wPt-0205 wPt-0275 wPt-3393 wPt-4009
wPt-4205 wPt-9555
144.0 barc49
146.8 wPt-1454
176.6 wPt-7053
182.9 gwm332
185.8 wPt-733079
189.7 wPt-3425
190.0 tPt-6875 wPt-7281
192.0 wPt-671548
193.3 wPt-1706 wPt-5524
194.7 wPt-9808
195.2 wPt-6768
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212.1 XBS00014199
213.2 wPt-3782 wPt-7122
214.3 wPt-2083
214.6 wPt-0827
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215.2 wPt-2339
219.7 wPt-744897
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224.3 gwm344a
225.9 XBS00003865
227.0 wPt-1023 wPt-1259 wPt-6019
228.7 wPt-0687 wPt-1601
wPt-1958 wPt-2501 wPt-4220 wPt-5533
229.5 wPt-6872 wPt-7298 wPt-7947 XBS00004348
XBS00009886
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233.3 wPt-0551
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7.2 wPt-8283
29.9 wPt-1737b wPt-1853
40.4 wPt-0137
51.9 wPt-4863
53.3 gwm46
54.3 gwm297
55.0 wPt-4519
58.3 wPt-8106
61.1 gwm333
63.2 wPt-6498 wPt-7934c
64.3 wPt-1553
64.9 wPt-5463
65.0 wPt-0963
66.0 wmc396
66.4 wPt-0318
76.2 wPt-9467
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77.7 wPt-3873
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79.2 wPt-6463
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87.9 wPt-1149
88.9 XBS00009556
91.6 wmc517
92.7 wmc51-7B
103.4 XBS00009398
106.1 wPt-1266
107.5 wPt-2688 wPt-4258
107.9 wPt-9299
108.5 wPt-7887
108.9 wPt-5892 wPt-5975
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109.9 wPt-2407
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113.8 wPt-4600
119.5 wPt-9987
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127.6 XBS00010660
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132.8 wPt-7113
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138.4 gwm577
141.2 wPt-6657
141.6 wPt-0752
143.0 wPt-5646
151.3 tPt-7247
152.2 XBS00010327
155.2 wPt-3190
156.2 XBS00009879
156.8 XBS00003664 XBS00003672 XBS00010560
157.4 wPt-7720
157.7 wPt-0600 wPt-0900 wPt-6750 wPt-9342
158.3 wPt-8007
159.2 wPt-7413
159.5 wPt-8561
159.7 wPt-1434 wPt-4875
160.2 wPt-11210 wPt-11312
161.0 tPt-6363 wPt-1345 wPt-3004
162.1 wPt-5216 wPt-8387
tPt-3700 tPt-8504 wPt-0504 wPt-1085
wPt-2878 wPt-3439b wPt-3445 wPt-4031
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wPt-7387 wPt-7470 wPt-8656 wPt-9515
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172.3 XBS00009245
172.8 XBS00009379
177.1 XBS00003756 XBS00004376 XBS00009619
178.2 wPt-665293
179.1 wPt-1066
181.1 XBS00003703
182.2 wPt-2677
182.8 wPt-1196
183.2 wPt-0684 wPt-1209 wPt-7108
183.3 wPt-5816 wPt-6320 wPt-6673
183.9 wPt-8246
184.2 wPt-3159
186.8 wPt-1069 wPt-1475
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188.0 wPt-4057
188.4 wPt-8598
189.4 wPt-9746
190.0 XBS00004171 XBS00004403
190.5 XBS00003649
196.7 XBS00010142
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210.9 wPt-6156
211.1 wPt-8938
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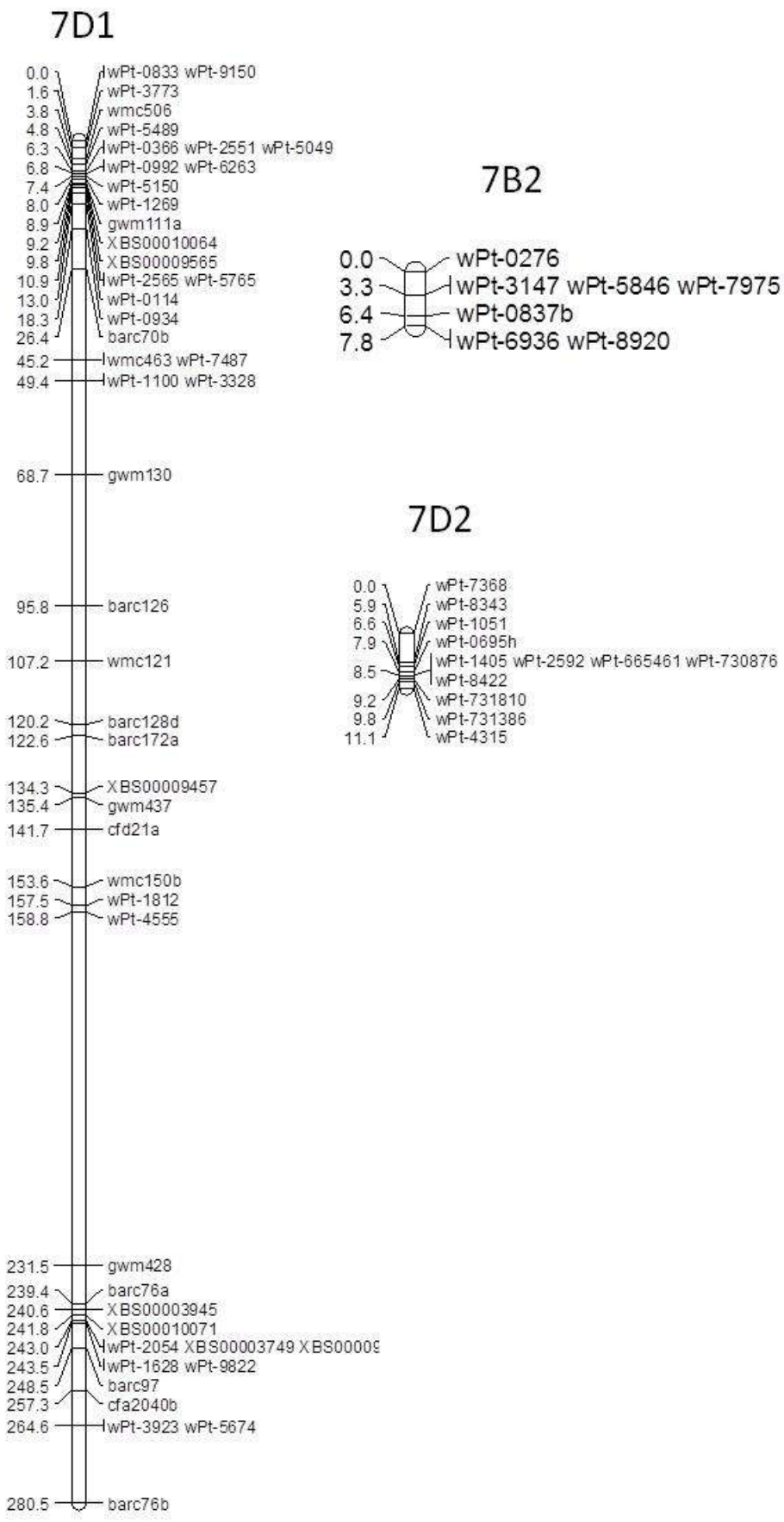
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9.8 XBS00009565
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13.0 wPt-0114
18.3 wPt-0934
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241.8 XBS00010071
243.0 wPt-2054 XBS00003749 XBS00009
243.5 wPt-1628 wPt-9822
248.5 barc97
257.3 cfa2040b
264.6 wPt-3923 wPt-5674
280.5 barc76b
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6.4 wPt-0837b
7.8 wPt-6936 wPt-8920
7D2
0.0 wPt-7368
5.9 wPt-8343
6.6 wPt-1051
7.9 wPt-0695h
8.5 wPt-1405 wPt-2592 wPt-665461 wPt-730876
wPt-8422
9.2 wPt-731810
9.8 wPt-731386
11.1 wPt-4315

APPENDIX 12: Main MTAs for height in YR panel

Level of significance:

- FDR: 10% FDR
- B: 5% Bonferroni
- * P-value<0.05
- ** P-value<0.01
- *** P-value<0.001

			r^2 level of significance						
Marker	**Chr.**	**Location (cM)**		**Model 3**		**Model 4**		**Model 5**	**Corresponding published QTL/gene**
wPt-665545	1AS	unmapped	2.1	*	3.2	***	2.2	**	?
SC-Y15	2AS	-	4.4	FDR, ***					(Griffiths et al., 2012)
wPt-733214	2B	unmapped			3.1	***	1.9	*	(Griffiths et al., 2012), (Neumann et al., 2011)
wPt-10874	3B	unmapped			3.5	***			?
wPt-730651	3D	7.5			3.9	***			None found
wPt-741202	3D	7.5			4.6	***			None found
wPt-741529	3D	7.5			3.1	***			None found
wPt-741949	3D	7.5			3.1	***			None found
wPt-741230	3D	7.5			3.6	***			None found
wPt-741598	3D	7.5			4.3	***			None found
wPt-666676	3D	7.5			3.1	***			None found
wPt-9067	4B	47.2			6.0	B***			*Rht1* (Ellis et al., 2002)
Rht2	4D	43.9	19.4	FDR, B,***	20.3	FDR, B,***	14.5	FDR, B,***	*Rht2* (Ellis et al., 2002)
wPt-4402	5B	unmapped	4.3	***			1.5	*	(Cui et al., 2011)
wPt-4577	5B	unmapped	3.4	***	3.4	***			(Cui et al., 2011)
wPt-6191	5B	unmapped	3.4	***	3.3	***			(Cui et al., 2011)
wPt-2707	5B	unmapped	3.3	***	3.4	***			(Cui et al., 2011)
wPt-671762	5D	235			3.5	***			None found
wPt-743645	7B LG2	3.0	3.8	***	3.9	***	2.7	**	(Cadalen et al., 1998; Ellis et al., 2005)
wPt-4220	multiple location	unmapped	4.6	***	1.6	*	3.5	***	?

APPENDIX 13: MTAs for yellow rust resistance and P-value for historical data GWA scans (Only selected markers from group 1BS-1RS, 2AS-2NS, 3D1 and 6D2 are included in the table)

Marker	Chr	cM		Group	1990-505	1993-24	1993-54	1994-519	1996-31	1996-502	1998-28	1998-96	1998-108	2000-41	2002-70	2002-84	no?	no7	no7no32	no4no8no7no32	virclaire	virrobavirsol	all histo
wPt-668160	1A	10.4	a	1A1	0.463	0.653	0.913	0.191	0.109	0.206	0.475	0.378	0.234	0.466	0.895	0.269	0.107	0.498	0.131	0.371	0.282	0.338	0.981	0.368
wPt-4676	1A	16.6	a	1A1	0.545	NA	NA	0.062	0.434	0.107	0.103	0.048	0.104	0.021	0.565	0.886	0.182	0.074	0.069	0.304	0.804	0.018	0.033	0.020
wPt-666776	1A	16.5	a,b	1A1	0.545	NA	NA	0.062	0.434	0.107	0.103	0.048	0.104	0.021	0.565	0.886	0.182	0.074	0.069	0.304	0.804	0.018	0.033	0.020
wPt-671790	1A	16.5	a,b	1A1	0.547	NA	NA	0.062	0.446	0.108	0.109	0.050	0.108	0.020	0.547	0.872	0.183	0.074	0.068	0.304	0.802	0.018	0.032	0.020
wPt-741357	1A	16.5	a,b	1A1	0.743	NA	NA	0.062	0.626	0.107	0.286	0.048	0.104	0.025	0.565	0.886	0.182	0.064	0.069	0.304	0.804	0.028	0.024	0.021
wPt-664778	1A	16.5	a,b	1A1	0.545	NA	NA	0.062	0.434	0.107	0.103	0.048	0.104	0.021	0.565	0.886	0.182	0.127	0.148	0.304	0.710	0.052	0.067	0.044
tPt-5413	1A	21.1	a	1A2	0.801	0.436	0.644	0.720	0.631	0.297	0.504	0.743	0.429	0.808	0.948	0.636	0.414	0.631	0.601	0.210	0.541	0.413	0.457	0.537
wPt-671596	1A	11.4	b	1A2	0.027	0.908	0.287	0.983	0.896	0.019	0.109	0.033	0.015	0.191	0.202	0.526	0.104	0.857	0.062	0.959	0.337	0.020	0.314	0.016
wPt-665174	1A	11.4	b	1A2	0.025	0.918	0.278	1.000	0.874	0.022	0.102	0.033	0.013	0.145	0.247	0.556	0.098	0.711	0.045	0.972	0.309	0.014	0.249	0.009
wPt-671823	1A	12.0	b	1A2	0.392	0.771	0.465	0.567	0.238	0.347	0.679	0.592	0.535	0.421	0.622	0.434	0.659	0.808	0.784	0.845	0.851	0.553	0.674	0.651
wPt-666607	1A	67.5	a	1A3	0.433	0.612	0.343	0.742	0.515	0.156	0.894	0.273	0.413	0.942	0.248	0.293	0.065	0.107	0.099	0.939	0.245	0.053	0.115	0.011
wPt-6074	1A	67.5	a	1A3	0.847	0.649	0.357	0.561	0.416	0.246	0.472	0.220	0.194	0.487	0.255	0.507	0.086	0.557	0.550	0.920	0.529	0.465	0.527	0.276
wPt-671483	1A	71.0	a,b	1A3	0.369	0.895	0.578	0.567	0.609	0.139	0.399	0.161	0.304	0.192	0.253	0.760	0.763	0.143	0.174	0.157	0.924	0.802	0.515	0.896
wPt-2694	1B	36.6	b	1B1	0.017	0.457	0.227	0.355	0.050	0.120	0.569	0.976	0.022	0.009	0.000	0.024	0.440	0.022	0.015	0.304	0.268	0.035	0.002	0.013
wPt-3465	1B	135.6	a	1B2	0.759	0.511	0.447	0.725	0.402	0.492	0.987	0.318	0.885	0.347	0.505	0.762	0.754	0.520	0.746	0.200	0.725	0.970	0.299	0.469
1BL.1RS marker	1BSor1RS	1.0	a,b	1BS-1RS	0.344	0.801	0.727	0.524	0.804	0.447	0.919	0.259	0.439	0.766	0.650	0.884	0.919	0.766	0.975	0.746	0.583	0.548	0.933	0.360
wPt-9883	1BSor1RS	1.0	a,b	1BS-1RS	0.444	0.884	0.695	0.513	0.958	0.397	0.532	0.263	0.506	0.957	0.694	0.814	0.779	0.929	0.778	0.779	0.546	0.764	0.778	0.488
rPt-7959	1BSor1RS	1.0	a,b	1BS-1RS	0.444	0.884	0.695	0.513	0.958	0.397	0.532	0.263	0.506	0.957	0.694	0.814	0.779	0.961	0.740	0.779	0.428	0.856	0.812	0.542
wPt-8930	1BSor1RS	2.0	a,b	1BS-1RS	0.444	0.884	0.695	0.396	0.958	0.352	0.532	0.179	0.487	0.969	0.694	0.814	0.697	0.901	0.877	0.779	0.801	0.453	0.811	0.286

APPENDIX 13 continued

Marker	Chr	cM		Group	1990-505	1993-24	1993-54	1994-519	1996-31	1996-502	1998-28	1998-96	1998-108	2000-41	2002-70	2002-84	no7	no5no7	no7no32	no5no7no32	no4no5no7no32	viticlaire	vitrobavirsol	all histo
wPt-5029	2A	61.6	a	2A1	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.799	0.126	0.340	0.152	0.498	NA	NA	0.320	0.805	NA
wPt-0553	2A	61.6	a	2A1	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.852	0.054	0.420	0.171	0.543	NA	NA	0.347	0.825	NA
wPt-5498	2A	61.6	a	2A1	NA	NA	NA	NA	NA	NA	NA	NA	0.075	0.005	0.320	0.286	0.167	0.075	0.313	NA	0.974	0.271	0.595	0.294
wPt-743211	2A	66.3	a	2A1	0.190	0.185	0.608	0.303	0.057	0.553	0.241	0.027	0.003	0.272	0.022	0.244	0.065	0.343	0.768	0.996	0.203	0.128	0.394	0.082
wPt-1657	2A	71.2	a	2A1	0.191	0.185	0.608	0.373	0.068	0.567	0.231	0.026	0.002	0.262	0.015	0.167	0.064	0.335	0.748	0.907	0.288	0.144	0.405	0.083
wPt-4021	2A		a, b	2A2	0.262	0.039	0.098	0.000	0.866	0.607	0.766	0.399	0.448	0.094	0.936	0.591	0.680	0.565	0.207	0.064	0.000	0.992	0.332	0.838
YR17	2A		a	2AS-2NS	0.106	0.010	0.006	0.119	0.751	0.920	0.713	0.981	0.577	0.556	0.818	0.218	0.480	0.857	0.310	0.401	0.179	0.591	0.574	0.328
wPt-744900	2ASor2NS	40.0	a, b	2AS-2NS	0.036	0.001	0.005	0.268	0.350	0.729	0.069	0.891	0.299	0.366	0.074	0.016	0.426	0.580	0.022	0.290	0.379	0.056	0.094	0.055
wPt-732882	2B		a, b	2B1	0.617	0.275	0.017	0.352	0.155	0.000	0.001	0.000	0.067	0.351	0.932	0.736	0.013	0.484	0.031	0.130	0.468	0.023	0.116	0.067
wPt-2293	2B	84.2	b	2B1	0.111	0.017	0.001	0.083	0.138	0.000	0.000	0.000	0.081	0.731	0.989	0.844	0.003	0.795	0.029	0.016	0.094	0.002	0.466	0.022
wPt-9402	2B	53.6	a	2B2	0.302	0.840	0.769	0.043	0.114	0.069	0.026	0.025	0.001	0.007	0.095	0.323	0.008	0.014	0.239	0.379	0.183	0.415	0.006	0.300
wPt-7619	2B	53.6	a	2B2	0.526	0.859	0.771	0.043	0.202	0.061	0.012	0.025	0.001	0.005	0.232	0.381	0.011	0.044	0.271	0.339	0.175	0.188	0.019	0.265
wPt-1489	2B	53.6	a	2B2	0.377	0.840	0.769	0.041	0.104	0.062	0.026	0.025	0.001	0.007	0.095	0.323	0.008	0.014	0.239	0.364	0.183	0.320	0.006	0.247
wPt-743630	2B	53.5	a, b	2B2	0.434	0.840	0.769	0.041	0.203	0.062	0.012	0.025	0.001	0.006	0.232	0.381	0.008	0.044	0.284	0.364	0.183	0.260	0.019	0.326
wPt-0950	2B	118.1	a	2B3	0.207	0.062	0.140	0.541	0.841	0.195	0.696	0.578	0.295	0.254	0.594	0.632	0.729	0.196	0.755	0.192	0.141	0.479	0.251	0.801
wPt-9350	2B	123.7	a	2B3	0.270	0.843	0.880	0.585	0.122	0.708	0.808	0.311	0.629	0.743	0.417	0.674	0.817	0.955	0.638	0.343	0.655	0.299	0.401	0.661
wPt-3378	2B	179.6	a	2B3	0.013	0.026	0.048	0.182	0.229	0.226	0.959	0.612	0.237	0.221	0.150	0.018	0.495	0.676	0.706	0.002	0.474	0.244	0.437	0.299
wPt-2274	2B	180.6	a	2B3	0.013	0.025	0.049	0.282	0.266	0.182	0.986	0.598	0.300	0.220	0.143	0.024	0.433	0.559	0.620	0.003	0.409	0.155	0.434	0.252
wPt-2724	2B	180.5	a, b	2B3	0.081	0.070	0.129	0.385	0.737	0.766	0.625	0.930	0.441	0.366	0.310	0.112	0.864	0.901	0.819	0.008	0.532	0.703	0.953	0.416
wPt-8693	2B	89.2	b	2B3	0.247	0.829	0.923	0.368	0.371	0.126	0.793	0.614	0.467	0.335	0.680	0.368	0.332	0.391	0.973	0.135	0.706	0.139	0.125	0.104

APPENDIX 13 continued

Marker	Chr	cM	Group	1990-505	1993-24	1993-54	1994-519	1996-31	1996-502	1998-28	1998-96	1998-108	2000-41	2002-70	2002-84	no7	no5no7	no7no32	no5no7no32	no4no5no7no32	viriclaire	virirobavirisol	all histo
wPt-9401	3D	5.8 [a]	3D1	0.209	0.216	0.120	0.939	0.109	0.051	0.004	0.040	0.001	0.033	0.004	0.001	0.031	0.013	0.013	0.345	0.354	0.005	0.163	0.175
wPt-2367	3D	7.5 [a]	3D1	0.871	0.469	0.185	0.554	0.207	0.138	0.013	NA	0.122	0.181	0.008	0.000	NA	0.350	0.111	0.288	0.139	0.023	0.587	0.344
wPt-1336	3D	8.5 [a]	3D1	0.533	0.594	0.251	0.915	0.289	0.043	0.004	0.038	0.001	0.033	0.004	0.001	0.028	0.018	0.017	0.343	0.376	0.006	0.163	0.186
wPt-8657	4A	20.7 [a]	4A1	0.006	0.014	0.187	0.916	0.008	0.217	0.472	0.916	0.296	0.423	0.001	0.001	0.455	0.737	0.291	0.387	0.658	0.296	0.374	0.107
wPt-734161	4A	20.5 [a, b]	4A1	0.006	0.014	0.187	0.916	0.008	0.217	0.472	0.916	0.296	0.423	0.001	0.001	0.422	0.773	0.282	0.387	0.658	0.324	0.399	0.118
wPt-0764	4A	20.5 [a, b]	4A1	0.006	0.014	0.187	0.916	0.008	0.217	0.472	0.916	0.296	0.423	0.001	0.001	0.455	0.737	0.291	0.387	0.658	0.296	0.366	0.125
RHT2	4D	43.9 [a, b]	4D1	0.303	0.593	0.621	0.048	0.011	0.058	0.041	0.006	0.010	0.713	0.019	0.154	0.241	0.083	0.952	0.049	0.012	0.005	0.055	0.000
wPt-731627	unknown		4D2	0.246	0.309	0.188	0.474	0.275	0.278	0.523	0.552	0.435	0.523	0.025	0.026	0.549	0.119	0.000	0.714	0.313	0.425	0.215	0.189
wPt-1165	5A	25.6 [a]	5A4	0.256	0.242	0.389	0.511	0.698	0.580	0.973	0.743	0.915	0.261	0.320	0.804	0.332	0.178	0.284	0.604	0.878	0.534	0.156	0.330
wPt-8157	5B	15.3 [b]	5B1	0.595	0.293	0.252	0.019	0.434	0.071	0.256	0.731	0.183	0.039	0.094	0.083	0.883	0.519	0.452	0.000	0.019	0.330	0.246	0.317
wPt-0708	5B	15.3 [b]	5B1	0.643	0.312	0.243	0.018	0.324	0.077	0.301	0.775	0.209	0.031	0.029	0.062	0.855	0.316	0.418	0.000	0.019	0.329	0.127	0.269
wPt-8604	5B	57.2 [a]	5B2	0.007	0.006	0.035	0.136	0.066	0.213	0.307	0.488	0.255	0.047	0.023	0.038	0.161	0.003	0.079	0.028	0.040	0.013	0.002	0.028
wPt-9666	5B	57.2 [a]	5B2	0.007	0.008	0.035	0.136	0.066	0.212	0.307	0.488	0.255	0.047	0.023	0.038	0.163	0.003	0.077	0.027	0.040	0.012	0.002	0.029
wPt-9724	5B	57.2 [a]	5B2	0.007	0.008	0.035	0.136	0.066	0.212	0.307	0.488	0.255	0.047	0.023	0.038	0.163	0.003	0.076	0.027	0.040	0.012	0.002	0.030
wPt-5346	5B	57.6 [a]	5B2	0.002	0.165	0.079	0.043	0.273	0.083	0.067	0.428	0.121	0.105	0.693	0.414	0.078	0.354	0.201	0.001	0.059	0.010	0.156	0.025
wPt-665267	5B	57.6 [a]	5B2	0.002	0.165	0.079	0.072	0.263	0.155	0.059	0.684	0.138	0.087	0.661	0.357	0.144	0.315	0.177	0.001	0.082	0.022	0.144	0.032
wPt-7237	5B	65.6 [a]	5B2	0.488	0.499	0.188	0.119	0.860	0.101	0.594	0.177	0.361	0.812	0.372	0.931	0.290	0.906	0.638	0.008	0.126	0.258	0.530	0.383
wPt-5928	5B	97.4 [b]	5B3	NA	NA	NA	NA	0.025	0.503	NA	0.516	0.644	0.698	0.005	0.001	0.881	0.026	0.972	NA	NA	0.120	0.013	0.142
wPt-5604	5B	97.4 [b]	5B3	NA	NA	NA	NA	0.025	0.577	NA	0.502	0.619	0.698	0.005	0.001	0.771	0.047	0.854	NA	NA	0.158	0.029	0.265
wPt-3763	5B	97.4 [b]	5B3	NA	NA	NA	NA	0.025	0.460	NA	NA	NA	0.511	0.005	0.001	0.753	0.098	0.750	NA	NA	0.132	0.063	0.196

APPENDIX 13 continued

Marker	Chr	cM	Group	1990-505	1993-24	1993-54	1994-519	1996-31	1996-502	1998-28	1998-96	1998-108	2000-41	2002-70	2002-84	no7	no6no7	no7no32	no6no7no32	virclaire	virtobavirsol	all histo
wPt-3695	2B	180.6 [a]	2B4	0.000	0.000	0.000	0.028	0.000	0.177	0.016	0.056	0.004	0.001	0.000	0.001	0.000	0.000	0.000	0.001	0.000	0.000	0.000
wPt-2135	2B	179.5 [a,b]	2B4	0.013	0.019	0.046	0.282	0.266	0.182	0.986	0.598	0.300	0.220	0.143	0.024	0.433	0.598	0.654	0.003	0.209	0.398	0.297
wPt-733641	2B	180.5 [a,b]	2B4	0.000	0.000	0.000	0.027	0.002	0.164	0.034	0.048	0.004	0.003	0.000	0.000	0.000	0.000	0.000	0.001	0.000	0.000	0.000
wPt-743307	2B	180.5 [a,b]	2B4	0.000	0.000	0.000	0.028	0.000	0.177	0.016	0.056	0.004	0.001	0.000	0.001	0.000	0.000	0.000	0.001	0.000	0.000	0.000
wPt-732666	2B	180.5 [a,b]	2B4	0.000	0.000	0.000	0.028	0.000	0.177	0.016	0.056	0.004	0.001	0.000	0.001	0.000	0.000	0.000	0.001	0.000	0.000	0.000
wPt-669273	2B	180.5 [a,b]	2B4	0.000	0.000	0.000	0.028	0.000	0.139	0.006	0.037	0.000	0.001	0.000	0.000	0.000	0.000	0.000	0.001	0.000	0.000	0.000
wPt-6419	2D	48.7 [b]	2D1	0.545	0.015	0.011	0.171	0.937	0.001	0.458	0.239	0.402	0.662	0.757	0.607	0.647	0.668	0.862	0.057	0.044	0.967	0.101
wPt-667054	2D	101.2 [b]	2D2	0.001	0.813	0.743	0.281	0.164	0.195	0.131	0.014	0.000	0.033	0.051	0.185	0.000	0.014	0.016	0.951	0.187	0.216	0.202	0.026
wPt-5476	3A	196.9 [a]	3A1	NA	0.189	0.470	0.229	NA	0.625	NA	NA	NA	NA	NA	NA	NA	NA	0.828	0.687	0.511	0.759	NA	0.484
wPt-3342	3B	125.2 [b]	3B1	0.708	0.003	0.009	0.063	0.034	0.112	0.151	0.182	0.567	0.773	0.212	0.076	0.017	0.864	0.081	0.046	0.098	0.078	0.468	0.208
wPt-8845	3B	125.5 [b]	3B1	0.113	0.003	0.058	0.430	0.142	0.372	0.982	0.676	0.964	0.817	0.890	0.637	0.342	0.632	0.260	0.265	0.636	0.065	0.604	0.123
wPt-8513	3B	128.8 [b]	3B1	0.408	0.006	0.014	0.972	0.119	0.790	0.304	0.953	0.361	0.541	0.285	0.191	0.797	0.325	0.420	0.451	0.971	0.710	0.743	0.424
wPt-6131	3B	131.5 [b]	3B1	0.477	0.008	0.007	0.727	0.837	0.810	0.705	0.466	0.699	0.280	0.902	0.434	0.684	0.811	0.994	0.068	0.589	0.583	0.846	0.681
wPt-5072	3B	131.5 [b]	3B1	0.396	0.008	0.006	0.729	0.197	0.974	0.497	0.483	0.607	0.461	0.214	0.142	0.788	0.575	0.504	0.043	0.737	0.428	0.573	0.314
tPt-7594	3B	131.5 [b]	3B1	0.245	0.001	0.010	0.852	0.185	0.553	0.557	0.449	0.523	0.771	0.148	0.074	0.514	0.472	0.401	0.141	0.983	0.681	0.803	0.698
wPt-7158	3B	132.5 [b]	3B1	0.291	0.005	0.007	0.918	0.092	0.685	0.628	0.849	0.734	0.628	0.235	0.170	0.644	0.216	0.367	0.325	0.945	0.955	0.540	0.270
wPt-6785	3B	210.3 [a]	3B2	0.300	0.153	0.184	0.517	0.171	0.124	0.105	0.421	0.140	0.433	0.079	0.063	0.298	0.204	0.002	0.294	0.175	0.115	0.079	0.147
wPt-664981	3B	210.0 [a,b]	3B2	0.271	0.420	0.183	0.853	0.580	0.179	0.112	0.574	0.710	0.452	0.308	0.216	0.233	0.303	0.008	0.328	0.331	0.214	0.174	0.350
wPt-672088	3B	210.0 [a,b]	3B2	0.269	0.420	0.183	0.737	0.600	0.146	0.055	0.491	0.733	0.623	0.374	0.218	0.242	0.474	0.011	0.230	0.274	0.271	0.265	0.434
wPt-10537	3B	272.2 [a]	3B3	0.742	0.228	0.321	0.031	0.645	0.626	0.662	0.473	0.986	0.072	0.610	0.706	0.402	0.780	0.593	0.000	0.031	0.303	0.924	0.470
wPt-0367	3B	281.5 [a]	3B3	0.516	0.865	0.902	0.518	0.838	0.339	0.819	0.149	0.991	0.202	0.368	0.790	0.203	0.977	0.926	0.010	0.376	0.433	0.942	0.880

APPENDIX 13 continued

Marker	Chr	cM	Group	1990-505	1993-24	1993-54	1994-519	1996-31	1996-502	1998-28	1998-96	1998-108	2000-41	2002-70	2002-84	no7	no7no32	no8no7	no7no32	no8no7no32	virclaire	virrobavirsol	all histo
wPt-1548	5B	125.3 [a]	5B4	NA	NA	NA	0.328	0.835	0.012	0.006	0.093	0.025	0.233	0.379	0.361	0.597	0.687	0.114	0.610	0.774	0.647	0.973	0.450
tPt-3719	5B	125.3 [a]	5B4	NA	NA	NA	0.299	0.643	0.011	0.005	0.093	0.025	0.237	0.527	0.467	0.494	0.569	0.114	0.498	0.973	0.835	0.794	0.618
wPt-7101	5B	125.3 [a]	5B4	0.358	0.156	0.026	0.102	0.566	0.048	0.313	0.221	0.001	0.711	0.586	0.679	0.518	0.517	0.018	0.747	0.119	0.756	0.788	0.570
wPt-6531	5B	125.3 [a]	5B4	NA	NA	NA	0.655	0.444	0.146	NA	NA	0.006	0.970	0.182	0.196	NA	0.930	0.462	NA	NA	NA	0.730	0.916
wPt-4628	5B	125.3 [a]	5B4	0.158	0.054	0.026	0.152	0.821	0.100	0.036	0.224	0.002	0.528	0.664	0.513	0.512	0.946	0.438	0.019	0.159	0.379	0.970	0.379
wPt-2607	5B	125.3 [a]	5B4	0.145	0.054	0.026	0.108	0.799	0.066	0.032	0.212	0.001	0.554	0.856	0.624	0.508	0.968	0.434	0.008	0.151	0.472	0.919	0.447
wPt-3457	5B	125.3 [a]	5B4	NA	NA	NA	0.963	0.664	0.218	NA	NA	0.006	0.970	0.203	0.222	NA	NA	0.763	NA	0.494	NA	0.595	0.960
wPt-1250	5B	127.4 [a]	5B4	0.117	0.027	0.014	0.107	0.775	0.018	0.002	0.064	0.002	0.367	0.387	0.104	0.462	0.769	0.227	0.003	0.132	0.170	0.610	0.164
wPt-666939	5B	128.0 [a,b]	5B4	0.067	0.062	0.032	0.108	0.769	0.066	0.017	0.212	0.001	0.360	0.377	0.252	0.683	0.800	0.307	0.008	0.201	0.265	0.644	0.170
wPt-733112	5D	13.2 [a]	5D1	0.813	0.869	0.962	0.073	0.113	0.593	0.937	0.974	0.723	0.388	0.746	0.225	0.756	0.521	0.527	0.658	0.026	0.289	0.499	0.247
wPt-732418	5D	13.2 [a]	5D1	0.843	0.869	0.962	0.076	0.132	0.560	0.993	0.970	0.837	0.333	0.815	0.253	0.810	0.580	0.542	0.650	0.028	0.314	0.542	0.273
wPt-0228	6A	17.3 [a]	6A1	0.736	0.784	0.851	0.172	0.246	0.575	0.896	0.693	0.429	0.158	0.081	0.084	0.286	0.049	0.227	0.214	0.058	0.207	0.037	0.037
wPt-734344	6A	2.6 [b]	6A1	0.710	0.869	0.974	0.375	0.260	0.533	0.368	0.739	0.259	0.187	0.830	0.542	0.372	0.287	0.685	0.325	0.205	0.697	0.229	0.403
wPt-669498	6A	3.9 [b]	6A1	0.759	0.773	0.885	0.255	0.364	0.697	0.316	0.746	0.246	0.198	0.788	0.520	0.364	0.316	0.677	0.329	0.124	0.580	0.237	0.315
wPt-3965	6A	55.4 [a]	6A2	0.108	0.932	0.965	0.030	0.069	0.052	0.745	NA	0.012	NA	0.115	0.311	0.072	0.005	0.180	0.037	0.002	0.006	0.001	0.018
wPt-9584	6A	62.1 [a]	6A2	0.105	0.932	0.965	0.030	0.604	0.176	0.932	NA	0.228	0.977	0.911	0.591	0.061	0.087	0.331	0.037	0.001	0.039	0.098	0.112
wPt-4893	6B	72.9 [a]	6B1	0.436	0.989	0.631	0.263	0.855	0.132	0.310	0.340	0.284	0.098	0.096	0.287	0.016	0.057	0.299	0.356	0.670	0.142	0.239	0.122
wPt-6247	6B	72.9 [a]	6B1	0.365	0.717	0.789	0.251	0.885	0.085	0.310	0.274	0.404	0.060	0.096	0.287	0.035	0.048	0.261	0.315	0.575	0.142	0.221	0.102
wPt-2424	6B	83.2 [a]	6B1	0.014	0.062	0.709	0.273	0.002	0.092	0.561	NA	0.009	NA	0.009	0.000	0.003	0.002	0.146	0.319	0.418	0.024	0.020	0.143
wPt-729979	6B	51.9 [a,b]	6B1	0.657	0.841	0.744	0.475	0.183	0.123	0.104	0.150	0.190	0.172	0.028	0.416	0.106	0.232	0.020	0.448	0.385	0.281	0.030	0.132
wPt-743974	6B	48.0 [b]	6B1	0.402	0.866	0.958	0.911	0.047	0.662	0.005	0.210	0.250	0.195	0.659	0.081	0.064	0.136	0.126	0.203	0.631	0.724	0.052	0.507
wPt-744302	6B	48.0 [b]	6B1	0.436	0.869	0.740	0.918	0.049	0.604	0.003	0.327	0.229	0.274	0.761	0.073	0.103	0.197	0.121	0.553	0.658	0.820	0.069	0.630
wPt-2218	6B	48.0 [b]	6B1	0.568	0.869	0.740	0.594	0.052	0.821	0.006	0.523	0.415	0.296	0.666	0.085	0.156	0.146	0.154	0.198	0.901	0.900	0.112	0.678

APPENDIX 13 continued

Marker	CHr	cM	Group	1990-505	1993-24	1993-54	1994-519	1996-31	1996-502	1998-28	1998-96	1998-108	2000-41	2002-70	2002-84	no7	no7no32	no8no7no32	no4no8no7no32	viriclaire	virtobavirisol	all histo
wPt-3207	6B	108.7 [b]	6B2	0.522	NA	NA	NA	NA	0.477	NA	0.677	0.703	0.895	NA	NA	0.705	NA	NA	NA	0.161	0.539	0.196
wPt-4164	6B	111.9 [b]	6B2	NA	NA	NA	NA	NA	0.365	NA	0.663	0.722	0.684	NA	NA	0.713	NA	0.175	NA	0.150	0.595	0.136
wPt-9952	6B	113.9 [b]	6B2	0.149	0.948	0.399	0.901	0.277	0.421	0.870	0.848	0.687	0.369	0.770	0.049	0.863	0.229	0.222	0.811	0.892	0.190	0.967
wPt-732061	6B	133.2 [b]	6B2	0.955	NA	NA	NA	NA	0.732	0.325	0.581	0.698	0.986	NA	NA	0.967	0.655	NA	NA	0.249	0.277	0.266
wPt-731311	7A	40.6 [a]	7A1	0.010	0.188	0.033	0.967	0.649	0.629	0.625	0.338	0.477	0.196	0.687	0.896	0.846	0.678	0.584	0.819	0.472	0.597	0.939
wPt-740561	7A	40.6 [a]	7A1	0.012	0.206	0.035	0.155	0.893	0.003	0.058	0.080	0.001	0.150	1.000	0.732	0.130	0.310	0.125	0.026	0.007	0.107	0.017
wPt-3147	7Bbis	3.3 [a]	7B1	0.212	0.783	0.501	0.559	0.343	0.346	0.384	0.679	0.131	0.206	0.918	0.793	0.448	0.137	0.251	0.039	0.893	0.367	0.350
wPt-743645	7Bbis	3.0 [a,b]	7B1	0.075	0.513	0.395	0.724	0.474	NA	0.454	NA	0.145	NA	0.849	0.841	0.662	0.618	0.857	0.273	0.484	0.714	0.028
wPt-4814	7B	113.2 [a]	7B2	0.008	0.547	0.274	0.019	0.261	0.584	0.105	0.523	0.092	0.001	0.009	0.372	0.723	0.013	0.297	0.013	0.044	0.158	0.115
wPt-9987	7B	119.5 [a]	7B2	0.030	0.850	0.733	0.001	0.033	0.981	0.230	0.707	0.254	0.002	0.010	0.054	0.968	0.035	0.471	0.018	0.003	0.012	0.101
wPt-5646	7B	143.0 [a]	7B2	0.358	0.230	0.239	0.001	0.465	0.689	0.984	0.098	0.075	0.009	0.395	0.901	0.055	0.010	0.485	0.109	0.002	0.012	0.439
wPt-741191	7B	109.0 [a,b]	7B2	0.399	0.142	0.467	0.411	0.021	0.132	0.110	0.232	0.052	0.438	0.010	0.003	0.020	0.142	0.006	0.756	0.698	0.049	0.032
wPt-0752	7B	141.6 [a]	7B3	0.419	0.232	0.332	0.000	0.943	0.323	0.863	0.755	0.421	0.256	0.292	0.342	0.746	0.169	0.834	0.000	0.000	0.199	0.134
wPt-7614	3B	[a,b]	multi	0.396	0.008	0.006	0.729	0.197	0.974	0.497	0.483	0.607	0.461	0.214	0.142	0.788	0.524	0.462	0.043	0.714	0.523	0.296
wPt-2559	3B	[a,b]	multi	0.433	0.008	0.006	0.729	0.244	0.974	0.304	0.483	0.607	0.369	0.339	0.245	0.788	0.643	0.666	0.043	0.714	0.625	0.345
wPt-6358	3D	8.6 [a,b]	multi	0.450	0.386	0.232	0.753	0.135	0.035	0.004	0.036	0.003	0.007	0.004	0.002	0.026	0.003	0.014	0.691	0.165	0.001	0.009	0.020
wPt-0610	Multiloc			0.041	0.022	0.248	0.260	0.550	0.024	0.346	NA	0.657	0.262	0.735	0.686	0.032	0.093	0.720	0.127	0.368	0.022	0.393	0.015
wPt-665027	unknown			0.367	0.554	0.196	0.896	NA	0.459	NA	NA	NA	NA	0.451	0.149	NA	NA	NA	0.565	0.544	0.052	NA	0.262
wPt-667413	unknown			0.367	0.554	0.196	0.896	NA	0.459	NA	NA	NA	NA	0.451	0.149	NA	NA	NA	0.565	0.544	0.052	NA	0.262
wPt-663727	unknown			NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.468	0.507	NA	0.969	0.146	NA	NA	0.173	0.555	0.101
wPt-732440	unknown			0.910	0.460	0.446	0.240	0.872	0.796	0.221	0.732	0.996	0.746	0.763	0.730	0.555	0.876	0.660	0.557	0.127	0.758	0.613	0.259
wPt-732734	unknown			0.917	0.504	0.419	0.280	0.977	0.976	0.289	0.809	0.922	0.981	0.709	0.688	0.962	0.858	0.581	0.593	0.130	0.527	0.807	0.226
wPt-667566	unknown			0.912	0.504	0.419	0.279	0.972	0.349	0.289	0.809	0.922	0.981	0.709	0.688	0.962	0.858	0.581	0.593	0.135	0.527	0.807	0.228
wPt-671831	unknown			0.933	0.504	0.419	0.268	0.945	0.322	0.310	0.886	0.877	0.999	0.678	0.738	0.937	0.824	0.468	0.593	0.116	0.680	0.877	0.304

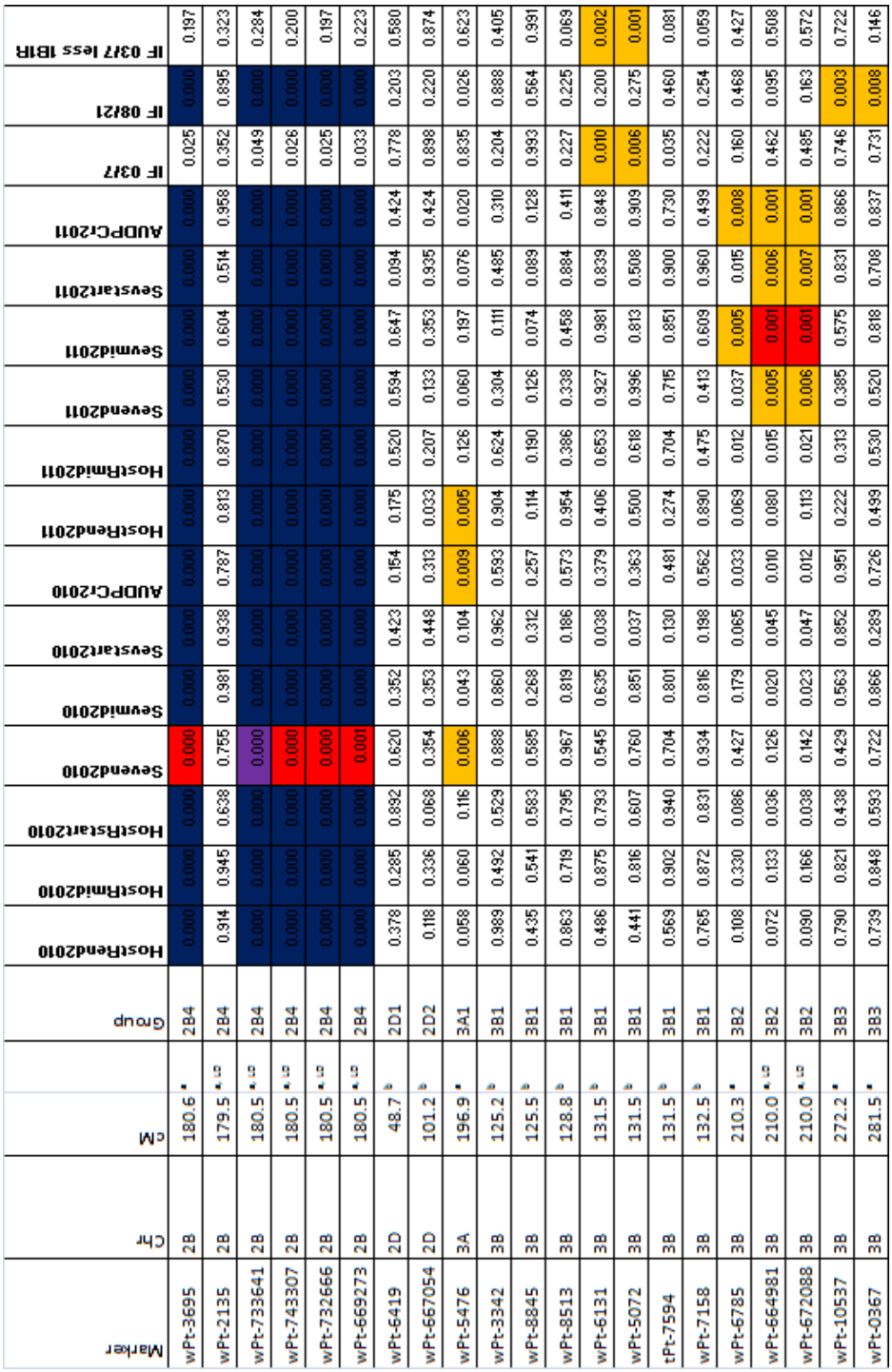
APPENDIX 14: MTA for yellow rust resistance and P-values for de novo phenotypes GWA scans (only selected markers from groups 1BS-1RS, 2AS-2NS, 3D1 and 6D2 are included in the table)

Marker	Chr	cM		Group	HostRend2010	HostRMid2010	HostRStart2010	Severd2010	Sevmid2010	Sevstart2010	AUDPCr2010	HostRend2011	HostRMid2011	Severd2011	Sevmid2011	Sevstart2011	AUDPCr2011	IF 03/7	IF 08/21	IF 03/7 less IBIR
wPt-668160	1A	10.4	a	1A1	0.572	0.498	0.831	0.889	0.954	0.592	0.211	0.053	0.005	0.037	0.013	0.059	0.013	0.661	0.824	0.708
wPt-4676	1A	16.6	a	1A1	0.004	0.004	0.001	0.000	0.000	0.100	0.002	0.053	0.079	0.377	0.338	0.493	0.933	0.000	0.094	0.014
wPt-666776	1A	16.5	a, b	1A1	0.004	0.004	0.001	0.000	0.000	0.100	0.002	0.053	0.079	0.377	0.338	0.493	0.933	0.000	0.094	0.014
wPt-671790	1A	16.5	a, b	1A1	0.004	0.004	0.001	0.000	0.000	0.101	0.002	0.058	0.084	0.389	0.343	0.493	0.934	0.000	0.095	0.014
wPt-741357	1A	16.5	a, b	1A1	0.002	0.005	0.002	0.002	0.000	0.201	0.005	0.096	0.074	0.483	0.426	0.568	0.961	0.000	0.136	0.012
wPt-664778	1A	16.5	a, b	1A1	0.010	0.020	0.006	0.007	0.001	0.202	0.010	0.053	0.079	0.377	0.338	0.493	0.933	0.000	0.265	0.019
tPt-5413	1A	21.1	a	1A2	0.084	0.015	0.008	0.035	0.015	0.444	0.094	0.169	0.050	0.373	0.300	0.308	0.399	0.629	0.398	0.776
wPt-671596	1A	11.4	b	1A2	0.003	0.010	0.058	0.000	0.004	0.428	0.030	0.042	0.151	0.158	0.239	0.159	0.086	0.725	0.357	0.578
wPt-665174	1A	11.4	b	1A2	0.003	0.009	0.047	0.000	0.004	0.381	0.030	0.047	0.157	0.193	0.303	0.207	0.119	0.738	0.360	0.570
wPt-671823	1A	12.0	b	1A2	0.015	0.031	0.356	0.010	0.135	0.997	0.389	0.628	0.670	0.422	0.484	0.970	0.320	0.683	0.816	0.951
wPt-666607	1A	67.5	a	1A3	0.014	0.308	0.011	0.042	0.001	0.007	0.000	0.014	0.046	0.036	0.018	0.004	0.006	0.313	0.271	0.719
wPt-6074	1A	67.5	a	1A3	0.063	0.402	0.268	0.298	0.138	0.173	0.075	0.004	0.037	0.074	0.282	0.100	0.270	0.484	0.777	0.957
wPt-671483	1A	71.0	a, b	1A3	0.145	0.028	0.010	0.097	0.209	0.882	0.551	0.310	0.057	0.255	0.168	0.427	0.553	0.610	0.046	0.309
wPt-2694	1B	36.6	b	1B1	0.072	0.007	0.036	0.028	0.125	0.050	0.090	0.052	0.014	0.024	0.025	0.023	0.101	0.144	0.018	0.183
wPt-3465	1B	135.6	a	1B2	0.586	0.853	0.886	0.966	0.825	0.590	0.570	0.725	0.737	0.997	0.303	0.202	0.440	0.001	0.565	0.124
1BL.1RS mark	1BSor1RS	1.0	a, b	1BS-1RS	0.683	0.754	0.682	0.879	0.902	0.929	0.552	0.204	0.487	0.862	0.665	0.220	0.178	0.000	0.286	NA
wPt-9883	1BSor1RS	1.0	a, b	1BS-1RS	0.332	0.754	0.939	0.193	0.592	0.881	0.446	0.313	0.826	0.923	0.479	0.212	0.135	0.000	0.541	NA
rPt-7959	1BSor1RS	1.0	a, b	1BS-1RS	0.307	0.687	0.892	0.162	0.498	0.934	0.389	0.339	0.767	0.915	0.463	0.191	0.126	0.000	0.542	NA
wPt-8930	1BSor1RS	2.0	a, b	1BS-1RS	0.810	0.279	0.341	0.757	0.575	0.482	0.866	0.027	0.251	0.407	0.834	0.648	0.430	0.000	0.034	NA

APPENDIX 14 continued

Marker	CT	cM		Group	HostRend2010	HostRMid2010	HostRStart2010	Sevend2010	Sevmid2010	Sevstart2010	AUDPCr2010	HostRend2011	HostRMid2011	Sevend2011	Sevmid2011	Sevstart2011	AUDPCr2011	IF 03/7	IF 08/21	IF 03/7 less IBIR
wPt-5029	2A	61.6	*	2A1	0.027	0.037	0.038	0.586	0.074	0.008	0.003	0.172	0.222	0.134	0.199	0.128	0.104	0.043	0.166	0.105
wPt-0553	2A	61.6	*	2A1	0.046	0.041	0.077	0.522	0.102	0.025	0.009	0.188	0.215	0.128	0.195	0.123	0.103	0.042	0.213	0.111
wPt-5498	2A	61.6	*	2A1	0.059	0.038	0.070	0.318	0.085	0.127	0.025	0.050	0.113	0.054	0.153	0.110	0.153	0.093	0.403	0.301
wPt-743211	2A	66.3	*	2A1	0.014	0.038	0.007	0.014	0.019	0.001	0.013	0.039	0.090	0.272	0.156	0.096	0.028	0.759	0.003	0.855
wPt-1657	2A	71.2	*	2A1	0.013	0.032	0.009	0.011	0.019	0.001	0.014	0.092	0.085	0.253	0.145	0.080	0.026	0.667	0.003	0.760
wPt-4021	2A		*, LD	2A2	0.253	0.658	0.276	0.220	0.834	0.742	0.842	0.269	0.341	0.980	0.745	0.845	0.846	0.925	0.430	0.826
YR17	2A		*	2AS-2NS	0.897	0.440	0.966	0.900	0.993	0.986	0.447	0.591	0.999	0.268	0.215	0.217	0.208	0.638	0.350	0.313
wPt-744900	2ASor2NS	40.0	*, LD	2AS-2NS	0.668	0.963	0.917	0.993	0.850	0.402	0.487	0.237	0.407	0.083	0.069	0.029	0.173	0.162	0.395	0.138
wPt-732882	2B		*, LD	2B1	0.226	0.099	0.206	0.115	0.133	0.919	0.356	0.285	0.342	0.183	0.148	0.134	0.267	0.016	0.411	0.010
wPt-2293	2B	84.2	b	2B1	0.283	0.394	0.579	0.120	0.421	0.230	0.359	0.866	0.684	0.646	0.450	0.265	0.587	0.004	0.195	0.000
wPt-9402	2B	53.6	*	2B2	0.026	0.003	0.040	0.014	0.007	0.112	0.039	0.033	0.003	0.008	0.006	0.094	0.173	0.356	0.062	0.757
wPt-7619	2B	53.6	*	2B2	0.018	0.002	0.023	0.008	0.002	0.082	0.025	0.037	0.003	0.006	0.004	0.118	0.126	0.258	0.086	0.872
wPt-1489	2B	53.6	*	2B2	0.032	0.005	0.045	0.017	0.008	0.126	0.046	0.046	0.004	0.010	0.007	0.111	0.187	0.435	0.088	0.639
wPt-743630	2B	53.5	*, LD	2B2	0.014	0.002	0.019	0.005	0.002	0.077	0.021	0.026	0.003	0.005	0.003	0.097	0.119	0.185	0.054	0.939
wPt-0950	2B	118.1	*	2B3	0.012	0.002	0.175	0.000	0.029	0.477	0.305	0.001	0.001	0.003	0.016	0.354	0.108	0.014	0.006	0.004
wPt-9350	2B	123.7	*	2B3	0.219	0.103	0.651	0.034	0.422	0.994	0.615	0.196	0.228	0.425	0.853	0.957	0.940	0.783	0.002	0.760
wPt-3378	2B	179.6	*	2B3	0.788	0.844	0.757	0.604	0.860	0.905	0.720	0.860	0.602	0.357	0.513	0.622	0.908	0.384	0.704	0.363
wPt-2274	2B	180.6	*	2B3	0.989	0.835	0.459	0.898	0.854	0.871	0.893	0.756	0.746	0.514	0.583	0.530	0.916	0.341	0.952	0.323
wPt-2724	2B	180.5	*, LD	2B3	0.961	0.876	0.905	0.742	0.560	0.869	0.436	0.714	0.897	0.272	0.234	0.916	0.606	0.287	0.709	0.385
wPt-8693	2B	89.2	b	2B3	0.021	0.060	0.073	0.018	0.006	0.133	0.015	0.112	0.160	0.058	0.059	0.133	0.286	0.238	0.307	0.250

APPENDIX 14 continued



Marker	Chr	cM		Group	HostRend2010	HostRmid2010	HostRstart2010	Sevend2010	Sevmid2010	Sevstart2010	AUDPC.2010	HostRend2011	HostRmid2011	Sevend2011	Sevmid2011	Sevstart2011	AUDPC.2011	IF 03/7	IF 08/21	IF 03/7 less IBIR
wPt-3695	2B	180.6	a	2B4	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.025	0.000	0.197
wPt-2135	2B	179.5	a,b	2B4	0.314	0.945	0.638	0.755	0.981	0.938	0.787	0.813	0.870	0.530	0.604	0.514	0.958	0.352	0.895	0.323
wPt-733641	2B	180.5	a,b	2B4	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.049	0.000	0.284
wPt-743307	2B	180.5	a,b	2B4	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.026	0.000	0.200
wPt-732666	2B	180.5	a,b	2B4	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.025	0.000	0.197
wPt-669273	2B	180.5	a,b	2B4	0.000	0.000	0.000	0.001	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.033	0.000	0.223
wPt-6419	2D	48.7	b	2D1	0.378	0.285	0.892	0.620	0.352	0.423	0.154	0.175	0.520	0.594	0.647	0.094	0.424	0.778	0.203	0.580
wPt-667054	2D	101.2	b	2D2	0.118	0.336	0.068	0.354	0.353	0.448	0.313	0.033	0.207	0.133	0.353	0.935	0.424	0.898	0.220	0.874
wPt-5476	3A	196.9	a	3A1	0.058	0.060	0.116	0.006	0.043	0.104	0.009	0.005	0.126	0.060	0.197	0.076	0.020	0.835	0.026	0.623
wPt-3342	3B	125.2	b	3B1	0.989	0.492	0.529	0.888	0.860	0.962	0.593	0.904	0.624	0.304	0.111	0.485	0.310	0.204	0.888	0.405
wPt-8845	3B	125.5	b	3B1	0.435	0.541	0.583	0.585	0.268	0.312	0.257	0.114	0.190	0.126	0.074	0.089	0.128	0.993	0.564	0.991
wPt-8513	3B	128.8	b	3B1	0.863	0.719	0.795	0.967	0.819	0.186	0.573	0.954	0.386	0.338	0.458	0.884	0.411	0.227	0.225	0.069
wPt-6131	3B	131.5	b	3B1	0.486	0.875	0.793	0.545	0.635	0.038	0.379	0.406	0.653	0.927	0.381	0.839	0.848	0.010	0.200	0.002
wPt-5072	3B	131.5	b	3B1	0.441	0.816	0.607	0.760	0.851	0.037	0.363	0.500	0.618	0.996	0.813	0.508	0.909	0.006	0.275	0.001
tPt-7594	3B	131.5	b	3B1	0.569	0.902	0.940	0.704	0.801	0.130	0.481	0.274	0.704	0.715	0.851	0.900	0.730	0.035	0.460	0.081
wPt-7158	3B	132.5	b	3B1	0.765	0.872	0.831	0.934	0.816	0.198	0.562	0.890	0.475	0.413	0.609	0.960	0.439	0.222	0.254	0.059
wPt-6785	3B	210.3	a	3B2	0.108	0.330	0.086	0.427	0.179	0.065	0.033	0.069	0.012	0.037	0.005	0.015	0.008	0.160	0.468	0.427
wPt-664981	3B	210.0	a,b	3B2	0.072	0.133	0.036	0.126	0.020	0.045	0.010	0.080	0.015	0.005	0.001	0.006	0.001	0.462	0.095	0.508
wPt-672088	3B	210.0	a,b	3B2	0.090	0.166	0.038	0.142	0.023	0.047	0.012	0.113	0.021	0.006	0.001	0.007	0.001	0.485	0.163	0.572
wPt-10537	3B	272.2	a	3B3	0.790	0.821	0.438	0.429	0.563	0.852	0.951	0.222	0.313	0.385	0.575	0.831	0.866	0.746	0.003	0.722
wPt-0367	3B	281.5	a	3B3	0.739	0.848	0.593	0.722	0.866	0.289	0.726	0.499	0.530	0.520	0.818	0.708	0.837	0.731	0.008	0.146

APPENDIX 14 continued

Marker	Chr	cM	Group	HostRend2010	HostRmid2010	HostRstart2010	Sevend2010	Sevmid2010	Sevstart2010	AUDPC.r2010	HostRend2011	HostRmid2011	Sevend2011	Sevmid2011	Sevstart2011	AUDPC.r2011	IF 0317	IF 0821	IF 0317 less IBIR
wPt-9401	3D	5.8 [a]	3D1	0.074	0.006	0.069	0.004	0.001	0.101	0.007	0.522	0.824	0.264	0.332	0.096	0.378	0.534	0.037	0.300
wPt-2367	3D	7.5 [a]	3D1	0.105	0.014	0.130	0.021	0.018	0.755	0.061	0.476	0.649	0.429	0.125	0.084	0.032	0.268	0.208	0.191
wPt-1336	3D	8.5 [a]	3D1	0.064	0.004	0.062	0.001	0.001	0.331	0.004	0.526	0.915	0.268	0.160	0.036	0.095	0.656	0.012	0.514
wPt-8657	4A	20.7 [a]	4A1	0.368	0.050	0.619	0.120	0.168	0.559	0.113	0.120	0.273	0.096	0.026	0.160	0.102	0.052	0.514	0.176
wPt-734161	4A	20.5 [a, b]	4A1	0.368	0.050	0.619	0.120	0.168	0.559	0.113	0.123	0.269	0.107	0.031	0.172	0.112	0.057	0.513	0.193
wPt-0764	4A	20.5 [a, b]	4A1	0.282	0.053	0.615	0.122	0.168	0.425	0.074	0.107	0.245	0.061	0.017	0.108	0.070	0.028	0.815	0.081
RHT2	4D	43.9 [a, b]	4D1	0.157	0.072	0.006	0.113	0.194	0.457	0.388	0.320	0.548	0.573	0.864	0.134	0.966	0.383	0.099	0.386
wPt-731627	unknown		4D2	0.543	0.650	0.913	0.862	0.874	0.532	0.585	0.374	0.238	0.563	0.867	0.958	0.796	0.646	0.674	0.518
wPt-1165	5A	25.6 [a]	5A4	0.005	0.022	0.047	0.006	0.016	0.524	0.040	0.013	0.003	0.002	0.008	0.013	0.013	0.994	0.412	0.976
wPt-8157	5B	15.3 [b]	5B1	0.374	0.361	0.420	0.488	0.212	0.446	0.264	0.064	0.338	0.053	0.036	0.060	0.004	0.520	0.332	0.167
wPt-0708	5B	15.3 [b]	5B1	0.467	0.432	0.423	0.548	0.246	0.529	0.312	0.048	0.273	0.037	0.025	0.040	0.003	0.460	0.379	0.139
wPt-8604	5B	57.2 [a]	5B2	0.086	0.088	0.059	0.045	0.080	0.295	0.157	0.025	0.012	0.171	0.392	0.294	0.741	0.392	0.045	0.146
wPt-9666	5B	57.2 [a]	5B2	0.086	0.087	0.062	0.043	0.078	0.302	0.159	0.024	0.012	0.168	0.376	0.308	0.714	0.404	0.048	0.155
wPt-9724	5B	57.2 [a]	5B2	0.086	0.087	0.062	0.043	0.078	0.302	0.159	0.024	0.012	0.166	0.385	0.302	0.718	0.406	0.049	0.146
wPt-5346	5B	57.6 [a]	5B2	0.056	0.035	0.089	0.024	0.021	0.181	0.079	0.035	0.023	0.049	0.041	0.136	0.112	0.408	0.169	0.985
wPt-665267	5B	57.6 [a]	5B2	0.054	0.041	0.121	0.029	0.026	0.206	0.098	0.029	0.020	0.073	0.077	0.176	0.201	0.555	0.190	0.878
wPt-7237	5B	65.6 [a]	5B2	0.652	0.830	0.981	0.577	0.349	0.828	0.612	0.961	0.953	0.551	0.600	0.708	0.673	0.156	0.622	0.083
wPt-5928	5B	97.4 [b]	5B3	0.852	0.257	0.923	0.751	0.406	0.033	0.212	0.290	0.622	0.591	0.680	0.485	0.831	0.870	0.026	0.301
wPt-5604	5B	97.4 [b]	5B3	0.845	0.256	0.906	0.752	0.400	0.035	0.214	0.363	0.747	0.664	0.808	0.660	0.687	0.860	0.061	0.186
wPt-3763	5B	97.4 [b]	5B3	0.768	0.433	0.777	0.902	0.421	0.043	0.224	0.653	0.924	0.925	0.943	0.410	0.728	0.964	0.318	0.296

APPENDIX 14 continued

Marker	Chr	cM		Group	HostRend2010	HostRMid2010	HostRStart2010	Sevend2010	Sevmid2010	Sevstart2010	AUDPC.r2010	HostRend2011	HostRMid2011	Sevend2011	Sevmid2011	Sevstart2011	AUDPC.r2011	IF 03/7	IF 08/21	IF 03/7 less IBIR
wPt-1548	5B	125.3	a	5B4	0.070	0.034	0.746	0.005	0.045	0.848	0.208	0.096	0.350	0.163	0.208	0.143	0.746	0.204	0.184	0.373
tPt-3719	5B	125.3	a	5B4	0.112	0.051	0.849	0.006	0.061	0.623	0.289	0.065	0.314	0.126	0.193	0.150	0.662	0.319	0.205	0.453
wPt-7101	5B	125.3	a	5B4	0.354	0.258	0.672	0.286	0.499	0.551	0.282	0.446	0.236	0.718	0.186	0.201	0.238	0.794	0.434	0.607
wPt-6531	5B	125.3	a	5B4	0.821	0.739	0.985	0.766	0.631	0.745	0.878	0.407	0.410	0.439	0.266	0.039	0.134	0.443	0.477	0.591
wPt-4628	5B	125.3	a	5B4	0.347	0.290	0.982	0.288	0.474	0.861	0.466	0.294	0.048	0.353	0.071	0.080	0.208	0.520	0.790	0.973
wPt-2607	5B	125.3	a	5B4	0.492	0.363	0.970	0.436	0.542	0.752	0.412	0.319	0.083	0.356	0.088	0.108	0.179	0.721	0.805	0.809
wPt-3457	5B	125.3	a	5B4	0.762	0.493	0.803	0.807	0.373	0.959	0.804	0.659	0.596	0.379	0.182	0.034	0.138	0.401	0.857	0.588
wPt-1250	5B	127.4	a	5B4	0.145	0.069	0.462	0.054	0.093	0.257	0.098	0.090	0.015	0.052	0.012	0.015	0.032	0.764	0.395	0.738
wPt-666939	5B	128.0	a b	5B4	0.110	0.070	0.475	0.031	0.090	0.286	0.120	0.085	0.012	0.049	0.007	0.009	0.028	0.493	0.547	0.901
wPt-733112	5D	13.2	a	5D1	0.002	0.001	0.099	0.015	0.045	0.234	0.098	0.689	0.138	0.815	0.475	0.737	0.566	0.277	0.597	0.239
wPt-732418	5D	13.2	a	5D1	0.003	0.001	0.096	0.016	0.044	0.246	0.101	0.708	0.139	0.822	0.465	0.754	0.569	0.272	0.627	0.240
wPt-0228	6A	17.3	a	6A1	0.018	0.126	0.043	0.100	0.007	0.007	0.000	0.248	0.135	0.064	0.025	0.002	0.010	0.929	0.002	0.467
wPt-734344	6A	2.6	b	6A1	0.012	0.024	0.061	0.177	0.018	0.024	0.009	0.888	0.224	0.504	0.163	0.041	0.041	0.973	0.002	0.836
wPt-669488	6A	3.9	b	6A1	0.013	0.024	0.058	0.176	0.020	0.025	0.009	0.893	0.214	0.367	0.126	0.021	0.027	0.906	0.002	0.781
wPt-3965	6A	55.4	a	6A2	0.045	0.019	0.167	0.044	0.093	0.038	0.008	0.051	0.042	0.118	0.075	0.034	0.032	0.316	0.030	0.518
wPt-9584	6A	62.1	a	6A2	0.069	0.021	0.151	0.073	0.065	0.029	0.013	0.065	0.045	0.161	0.084	0.115	0.028	0.085	0.046	0.593
wPt-4893	6B	72.9	a	6B1	0.002	0.003	0.008	0.005	0.006	0.004	0.002	0.038	0.080	0.086	0.187	0.347	0.183	0.264	0.023	0.807
wPt-6247	6B	72.9	a	6B1	0.005	0.003	0.005	0.002	0.004	0.006	0.002	0.029	0.100	0.110	0.199	0.279	0.202	0.420	0.023	0.682
wPt-2424	6B	83.2	a	6B1	0.672	0.820	0.441	0.338	0.631	0.003	0.039	0.154	0.360	0.621	0.928	0.909	0.651	0.909	0.643	0.771
wPt-729979	6B	51.9	a b	6B1	0.123	0.028	0.015	0.058	0.007	0.317	0.034	0.343	0.127	0.336	0.492	0.247	0.874	0.057	0.577	0.222
wPt-743974	6B	48.0	b	6B1	0.309	0.313	0.787	0.372	0.327	0.100	0.240	0.951	0.763	0.797	0.900	0.965	0.818	0.781	0.172	0.557
wPt-744302	6B	48.0	b	6B1	0.398	0.297	0.962	0.333	0.368	0.153	0.400	0.997	0.693	0.904	0.726	0.871	0.996	0.930	0.272	0.643
wPt-2218	6B	48.0	b	6B1	0.250	0.229	0.622	0.321	0.561	0.183	0.458	0.640	0.986	0.841	0.849	0.971	0.844	0.708	0.239	0.347

APPENDIX 14 continued

Marker	Chr	cM	Group	HostRend2010	HostRmid2010	HostRstart2010	Seven2010	Sevmid2010	Sevstart2010	AUDPCr2010	HostRend2011	HostRmid2011	Seven2011	Sevmid2011	Sevstart2011	AUDPCr2011	IF 03/7	IF 08/21	IF 03/7 less IBIR
wPt-3207	6B	108.7 [b]	6B2	0.030	0.015	0.071	0.034	0.075	0.370	0.132	0.028	0.000	0.114	0.031	0.013	0.092	0.001	0.055	0.003
wPt-4164	6B	111.9 [b]	6B2	0.027	0.036	0.051	0.053	0.185	0.337	0.121	0.044	0.001	0.133	0.028	0.012	0.113	0.001	0.149	0.005
wPt-9952	6B	113.9 [b]	6B2	0.868	0.855	0.820	0.778	0.415	0.407	0.326	0.322	0.102	0.629	0.196	0.256	0.627	0.009	0.232	0.008
wPt-732061	6B	133.2 [b]	6B2	0.067	0.057	0.203	0.122	0.206	0.544	0.233	0.053	0.001	0.210	0.077	0.030	0.139	0.001	0.149	0.005
wPt-731311	7A	40.6 [a]	7A1	0.139	0.250	0.307	0.022	0.024	0.662	0.456	0.651	0.505	0.483	0.327	0.634	0.808	0.821	0.013	0.818
wPt-740561	7A	40.6 [a]	7A1	0.570	0.379	0.897	0.506	0.683	0.004	0.053	0.411	0.242	0.997	0.486	0.050	0.131	0.385	0.806	0.199
wPt-3147	7Bbis	3.3 [a]	7B1	0.551	0.382	0.431	0.237	0.711	0.845	0.997	0.193	0.999	0.078	0.098	0.938	0.221	0.001	0.856	0.002
wPt-743645	7Bbis	3.0 [a, b]	7B1	0.978	0.782	0.724	0.682	0.823	0.690	0.548	0.639	0.208	0.722	0.605	0.492	0.641	0.000	0.649	0.001
wPt-4814	7B	113.2 [a]	7B2	0.276	0.143	0.337	0.665	0.733	0.649	0.412	0.068	0.024	0.020	0.048	0.059	0.211	0.024	0.982	0.002
wPt-9987	7B	119.5 [a]	7B2	0.559	0.472	0.478	0.383	0.259	0.156	0.168	0.822	0.977	0.732	0.712	0.324	0.791	0.436	0.789	0.050
wPt-5646	7B	143.0 [a]	7B2	0.620	0.713	0.965	0.576	0.833	0.465	0.640	0.465	0.441	0.751	0.720	0.353	0.374	0.791	0.393	0.511
wPt-741191	7B	109.0 [a, b]	7B2	0.430	0.428	0.293	0.198	0.969	0.444	0.838	0.148	0.052	0.141	0.134	0.239	0.110	0.629	0.066	0.753
wPt-0752	7B	141.6 [a]	7B3	0.505	0.323	0.928	0.854	0.738	0.883	0.700	0.370	0.375	0.396	0.570	0.436	0.785	0.001	0.329	0.000
wPt-7614	3B	[a, b]	multi	0.423	0.753	0.560	0.722	0.793	0.032	0.329	0.500	0.618	0.996	0.813	0.508	0.909	0.006	0.247	0.001
wPt-2559	3B	[a, b]	multi	0.563	0.770	0.629	0.792	0.903	0.045	0.402	0.860	0.880	0.758	0.970	0.620	0.990	0.008	0.399	0.001
wPt-6358	3D	8.6 [a, b]	multi	0.284	0.048	0.199	0.057	0.010	0.190	0.005	0.424	0.598	0.845	0.166	0.050	0.061	0.831	0.036	0.535
wPt-0610	Multiloc			0.010	0.012	0.067	0.033	0.036	0.025	0.017	0.012	0.007	0.001	0.001	0.037	0.030	0.365	0.079	0.380
wPt-665027	unknown			0.031	0.080	0.035	0.060	0.008	0.018	0.000	0.646	0.205	0.239	0.047	0.008	0.000	0.284	0.577	0.756
wPt-667413	unknown			0.031	0.080	0.035	0.060	0.008	0.018	0.000	0.646	0.205	0.239	0.047	0.008	0.000	0.284	0.577	0.756
wPt-663727	unknown			0.002	0.010	0.002	0.001	0.001	0.004	0.001	0.438	0.482	0.350	0.355	0.210	0.627	0.161	0.249	NA
wPt-732440	unknown			0.549	0.475	0.166	0.468	0.179	0.295	0.276	0.003	0.000	0.004	0.011	0.061	0.131	0.013	0.063	0.036
wPt-732734	unknown			0.261	0.371	0.089	0.448	0.070	0.528	0.171	0.004	0.000	0.001	0.004	0.049	0.055	0.027	0.069	0.092
wPt-667566	unknown			0.260	0.361	0.097	0.449	0.063	0.523	0.168	0.004	0.000	0.001	0.004	0.049	0.054	0.026	0.070	0.086
wPt-671831	unknown			0.355	0.433	0.105	0.568	0.105	0.599	0.226	0.007	0.000	0.002	0.006	0.069	0.101	0.035	0.085	0.117

APPENDIX 15 : List of markers included in MTA groups 1BS-1RS, 2AS-2AN, 3D1 and 6A2

P-values for markers underlined are available in Appendices 13 and 14

Markers in MTA group 1BS-1RS (101 markers with r^2 LD estimate>0.5 with 1BL.1RS marker from de Froidmont (1998)
rPt-0079, rPt-1217, rPt-1767, rPt-2869, rPt-3642, rPt-5341, rPt-6561, rPt-6965, rPt-7959, rPt-8894, rPt-9564, tPt-0136, tPt-0325, tPt-0734, tPt-1586, tPt-2076, tPt-2240, tPt-2326, tPt-2440, tPt-2550, tPt-3696, tPt-4566, tPt-5080, tPt-5515, tPt-5755, tPt-6015, tPt-7214, tPt-7559, tPt-7918, tPt-8109, tPt-8754, wPt-0014, wPt-0170, wPt-0320, wPt-0359, wPt-0729, wPt-0831, wPt-0974, wPt-0983, wPt-1116, wPt-1139, wPt-1251, wPt-1328, wPt-1684, wPt-1717, wPt-1781, wPt-1911, wPt-1997, wPt-2057, wPt-2261, wPt-2474, wPt-2577, wPt-2614, wPt-2654, wPt-2751, wPt-2762, wPt-2786, wPt-2999, wPt-3177, wPt-3787, wPt-4107, wPt-4605, wPt-4655, wPt-5065, wPt-5435, wPt-5506, wPt-5740, wPt-5765, wPt-5798, wPt-5800, wPt-6078, wPt-6434, wPt-664964, wPt-669404, wPt-6833, wPt-740789, wPt-740807, wPt-741274, wPt-741297, wPt-741612, wPt-741676, wPt-741749, wPt-741799, wPt-7422, wPt-742457, wPt-742513, wPt-742776, wPt-743523, wPt-7460, wPt-8177, wPt-8320, wPt-8338, wPt-8616, wPt-8884, wPt-8930, wPt-9472, wPt-9524, wPt-9562, wPt-9631, wPt-9883, wPt-9903
Markers in MTA group 2AS-2NS (16 markers with r^2 LD estimate>0.7 with 2AS.2NS marker from Robert et al. (1999)
wPt-2309, wPt-3565, wPt-3976, wPt-4533, wPt-6158, wPt-6207, wPt-6431, wPt-669721, wPt-733012, wPt-742886, wPt-744900, wPt-744943, wPt-8242, , wPt-8464, wPt-9712, wPt-9958
Markers in MTA group 3D1 (52 markers mapped to 5.8-8.6cM interval in the consensus map or in LD with markers wPt-2767, wPt-740662 (r^2>0.7))
wPt-10291, wPt-1336, wPt-2367, wPt-666676,wPt-666738, wPt-667139, wPt-669255, wPt-729808, wPt-730794, wPt-731146, wPt-733267, wPt-733640, wPt-740538, wPt-740640, wPt-740662, wPt-740665, wPt-740703, wPt-740803, wPt-740845, wPt-740945, wPt-740957, wPt-741189, wPt-741202, wPt-741230, wPt-741333, wPt-741440, wPt-741521, wPt-741529, wPt-741558, wPt-741598, wPt-741656, wPt-741683, wPt-741767, wPt-741800, wPt-741820, wPt-741829, wPt-741943, wPt-741949, wPt-741984, wPt-741987, wPt-742156, wPt-742222, wPt-742266, wPt-742405, wPt-742431, wPt-742448, wPt-742480, wPt-742519, wPt-742569, wPt-742630, wPt-9401
Markers in MTA group 6A2 (14 markers mapped to 55.0-62.7cM interval in the consensus map or in LD with markers from the group (r^2>0.7))
tPt-7399, wPt-2822, wPt-3965, wPt-664552, wPt-664733, wPt-666208, wPt-667780, wPt-671799, wPt-730729, wPt-731010, wPt-732760,wPt-733764, wPt-9131, wPt-9584

APPENDIX 16: Analysis of variance and Chi squared test for goodness of fit for infection type observed in Avalon x cadenza population

ANOVA outputs

d.f: degree of freedom, s.s. sum of squares, m.s. mean square, v.r. variance ratio, F pr. Probability based on F distribution, H^2 heritability

Score	Source	d.f	s.s.	m.s	v.r.	F pr.	H^2
IF 17DPI of AXC lines against 08/21	Line	201	3742.62	18.62	46.52	<0.001	0.978
	Block	1	14.14	14.14	35.32	<0.001	
	Residual	419	167.70	0.40			
	Total	621	3924.46	6.32			
IF 20DPI of AXC lines against 03/07	Line	201	165.50	0.823	48.69	<0.001	0.982
	Block	1	0.02	0.020	1.18	0.278	
	Residual	420	7.10	0.017			
	Total	622	172.62	0.278			

Segregation of the infection type observed at seedling stage in AxC population against isolates 08/21 and 03/07

Pop.	Pst isolate	Observed DH lines		Total	Expected Ratio R:S	χ^2	P-value
		R	S				
AxC	08/21[a]	102	99	201	1:1	0.045	0.83
AXC	03/07[b]	106	94	201	1:1	0.72	0.40

Pop.: population, R: resistant, S: susceptible, [a] Resistant lines, IF≤4; susceptible lines, IF≥4, [b] Resistant lines, IF≤3; susceptible lines, IF≥3

APPENDIX 17: Linkage group 2AL including marker STSwPt-3695 from Avalon x Cadenza mapping population