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**QTL mapping of resistance to two wheat rust fungi,
Puccinia triticina Eriks. and *Puccinia striiformis* Westend.
in a multiparental wheat (*Triticum aestivum* L.) population**

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LIST OF ABBREVIATIONS

°C	degree Celsius
ABC transporter	ATP-binding cassette transporter
AFLP	amplified fragment length polymorphism
ANOVA	analysis of variance
APR	adult plant resistance
ATP	adenosine triphosphate
Avr gene	avirulence genes
BC	backcrosses
BMWpop	Bavarian MAGIC Wheat population
bp	base pairs
CIM	composite interval mapping
cM	centrimorgan
CYP	cytrochrome P450
DH	double haploids
DMIs	14α-demethylation inhibitors
DNA	deoxyribonucleic acid
dt	decitons
ETI	effector triggered immunity
ETS	effector-triggered susceptibility
ff. spp.	formae speciales
GEBVs	genomic estimated breeding values
GS	genomic selection
GWAS	genome-wide association studies
ha	hectare
HCN	hydrogen cyanide
HR	hypersensitive response
HSP	heat shock proteins
LD	linkage disequilibrium
Lr genes	leaf rust resistance genes

List of abbreviations

LRR	leucine rich repeat
MAGIC	multiparent advanced generation intercross
MAMP	microbe-associated molecular pattern
MAS	marker-assisted selection
MIM	multiple interval mapping
NAM	nested association mapping
NB-LRR proteins	nucleotide binding site-leucine rich repeat proteins
NGS	next generation sequencing
NIL	near-isogenic lines
NLR proteins	nucleotide-binding and leucine-rich repeat proteins
PAMP	pathogen-associated molecular pattern
PCR	polymerase chain reaction
PDR	pleiotropic drug resistance
PRRs	pattern recognition receptors
PTI	pattern-triggered immunity
QTL	quantitative trait loci
QoIs	quinone outside inhibitors
R genes	resistance genes
RAPD	randomly amplified polymorphic DNA
RFLP	restriction fragment length polymorphism
RILs	recombinant inbred lines
ROS	reactive oxygen species
SDHI	succinate dehydrogenase inhibitors
SI	support interval
SIM	simple interval mapping
SNP	single nucleotide polymorphism
Sr genes	stem rust resistance genes
SSR	simple sequence repeats
START	steroidogenic acute regulatory protein-related transfer
Yr genes	stripe rust resistance genes

Summary

With a global production of 766 million tons in 2019, wheat is the world's second most important cereal, providing ≥ 20 % of calories and protein for the human diet. Wheat rusts such as *Puccinia striiformis* f. sp. *tritici* and *Puccinia triticina*, the causal agents of stripe rust and leaf rust respectively, are among the most important fungal pathogens in wheat with the potential to cause severe yield and quality losses up to 70 %. Use of resistant cultivars is the economically safest and most environmentally friendly approach to avoid yield losses and ensure food security. However, the continuous development of new races of stripe rust and leaf rust that are virulent against important resistance genes increases the need for new sources of resistance. In recent years, the identification of quantitative trait loci (QTL) has become the basis of targeted breeding approaches aiming at increased and durable resistance in modern wheat cultivars. In addition, multiparent advanced generation intercross (MAGIC) populations have proven to be a powerful tool to carry out such genetic studies.

In the framework of this thesis, the Bavarian MAGIC wheat population (BMWpop) was used to detect QTL conferring resistance against leaf rust and stripe rust. Seedling resistance was screened under controlled environmental conditions by using a detached leaf assay. Adult plant resistance was tested in multi-year's field trials at three locations in Germany. Phenotypic data, together with genotypic data from the 15 K + 5 K Infinium® iSelect® array containing 17,267 single nucleotide polymorphisms (SNP), were used to perform simple interval mapping (SIM) for stripe rust and leaf rust resistance. In total, 19 QTL corresponding to 11 different regions on chromosomes 1A, 4A, 4D, 5A, 6B, 7A and 7D were identified in independent SIM studies for leaf rust resistance. Six of these regions may represent putative new QTL, which have not been described earlier. For stripe rust, 21 QTL corresponding to 13 distinct chromosomal regions were detected, of which two may represent putatively new QTL located on wheat chromosomes 3D and 7D. Peak markers of the identified QTL were partly directly annotated with genes known to be involved in quantitative resistance to leaf and stripe rust. Additional promising gene annotations with different functions in relation to resistance responses were identified when considering ± 500 kb around each peak marker of a QTL.

The Bavarian MAGIC wheat population turned out to be well suited for the detection of QTL conferring resistance to leaf rust and stripe rust. Based on the phenotypic responses, RILs with increased resistance to both rust fungi were identified, which can be easily introduced into breeding programs due to their descent from elite parents.

Summary

ZUSAMMENFASSUNG

Mit einer weltweiten Produktion von 766 Millionen Tonnen im Jahr 2019 ist Weizen die zweitwichtigste Getreideart der Welt, die ≥ 20 % des Kalorien- und Proteinbedarfs der menschlichen Ernährung deckt. Weizenroste wie *Puccinia striiformis* f. sp. *tritici* und *Puccinia triticina*, die Erreger des Weizengelb- bzw. des Weizenbraunrosts, gehören zu den wichtigsten pilzlichen Krankheitserregern im Weizen, die Ertrags- und Qualitätsverluste von bis zu 70 % verursachen können. Die Nutzung von resistenten Sorten ist der wirtschaftlich sicherste und umweltfreundlichste Ansatz zur Vermeidung von Ertragsverlusten und zur Sicherung der menschlichen Ernährung. Durch die kontinuierliche Entwicklung neuer Rassen des Gelb- und Braunrosts, die gegen wichtige Resistenzgene virulent sind, ergibt sich die Notwendigkeit nach neuen Resistenzquellen zu suchen. In den letzten Jahren ist die Identifizierung von QTL zur Grundlage gezielter Züchtungsansätze geworden, die darauf abzielen, ein erhöhtes und dauerhaftes Resistenzniveau in modernen Weizensorten zu erreichen. Darüber hinaus haben sich MAGIC Populationen als hilfreiches Instrument für die Durchführung solcher genetischen Studien erwiesen.

Im Rahmen dieser Arbeit wurde die Bayerische MAGIC-Weizenpopulation (BMWpop) genutzt, um Resistenz QTL gegen Weizenroste zu identifizieren. Dabei lag der Fokus auf dem Braun- und Gelbrost. Die Keimlingsresistenz wurde unter kontrollierten Umweltbedingungen mit Hilfe spezifischer Blattsegmenttests geprüft. Die Adultpflanzenresistenz wurde in mehrjährigen Feldversuchen an drei Standorten in Deutschland getestet. Im Anschluss wurden die phänotypischen Daten zusammen mit den genotypischen Daten des 15 K + 5 K Infinium® iSelect® Arrays, der 17.267 Einzelnukleotid-Polymorphismen (SNP) enthält, genutzt, um eine Simple-Intervallkartierung (SIM) für die Resistenz gegen Gelb- und Braunrost durchzuführen. Insgesamt wurden in unabhängigen SIM-Studien 19 QTL für Braunrostresistenz identifiziert, die 11 verschiedenen Regionen auf den Chromosomen 1A, 4A, 4D, 5A, 6B, 7A und 7D entsprechen. Sechs dieser Regionen könnten mutmaßlich neue QTL darstellen und wurden bisher nicht beschrieben. Für Gelbrost wurden 21 QTL entdeckt, die 13 verschiedenen chromosomalen Regionen entsprechen, von denen zwei möglicherweise bisher nicht bekannte QTL darstellen und sich auf den Weizenchromosomen 3D und 7D befinden. Die Peak-Marker der identifizierten QTL konnten teilweise direkt mit Genen annotiert werden, von denen bekannt ist, dass sie an der quantitativen Resistenz gegen Gelb- und Braunrost beteiligt sind. Zusätzliche vielversprechende Genannotationen im Zusammenhang mit Resistenzreaktionen wurden bei Betrachtung eines fixen Intervalls von ± 500 kb um jeden Peak-Marker eines QTLs identifiziert.

Summary

Die bayerische MAGIC-Weizenpopulation erwies sich für den Nachweis von Resistenz QTL gegen Gelb- und Braunrost als gut geeignet. Anhand der phänotypischen Reaktionen wurden RILs mit erhöhter Resistenz gegen beide Rostpilze identifiziert, die aufgrund ihrer Abstammung von Elite-Elternmaterial leicht in Zuchtprogramme integriert werden können.

CHAPTER I | GENERAL INTRODUCTION

1. Bread wheat (*Triticum aestivum* L.)

Wheat (*Triticum* spp.) is one of the 'big three' cereals, along with maize (*Zea mays* L.) and rice (*Oryza sativa* L.), which together account for 90 % of the total cereal production (FAO 2021). Wheat is grown on 17 % of all crop areas from 69° North (temperate zone) to 45° South (subtropical zone) and represents a major source for food, feed and industrial raw materials (Charmet, 2011; Peng et al., 2011). In 2019, around 766 million tons of wheat were produced globally on 216 million hectares, putting wheat in second place behind maize in terms of production but first in terms of harvested area (FAO 2021). Bread wheat or common wheat (*Triticum aestivum* L.) accounts for approximately 95 % of all wheat types grown, with the remaining 5 % being durum wheat (*Triticum turgidum* subsp. *durum*) and other less important species (Shewry, 2009; Venske et al., 2019). Today, wheat provides ≥ 20 % of the calories and proteins for the human diet and additionally contributes essential amino acids, minerals, vitamins, as well as beneficial phytochemicals and dietary fibre components (Shewry, 2009; Braun et al., 2010). The world's three largest wheat producers are China, India and Russia, with an average production of 126.7, 93.3 and 61.5 million tons respectively over the last ten years on an area harvested of 24.3, 29.9 and 25.0 million hectares, respectively (FAO 2021). With 24.1 million tons of wheat on 3.2 million hectares, Germany still belongs to the top ten wheat producers worldwide (FAO 2021). Nevertheless, it is expected that the world's demand for wheat by an ever-growing population will increase to more than 900 million tons until 2050, which requires an annual increase of 0.9 % until 2050 (FAO 2006; 2021; Dixon, 2009). Since the 'Green Revolution' in 1966 until 1980, wheat production area continuously expanded worldwide. Additionally, average grain yield of wheat steadily increased due to the use of short-strawed higher yielding, and disease resistant varieties in combination with the development of improved agronomic practices (Ahrends et al.; Dixon, 2009). However, the decline in investment in agriculture in the post-'Green Revolution' period and environmental concerns such as climate change leading to rising temperatures, droughts and soil degradation have led to stagnated wheat productivity in many farming systems dominated by wheat (Dixon, 2009; Pingali, 2012). Compared to the period from 1962 to 2007, the average growth rate of wheat yield will be more than halved by 2050. Therefore, the annual increase of yield is projected to decline from 1.08 % between the period from 1987 to 2007 to 0.74 % by 2050 (Alexandratos and Bruinsma, 2012). A possibility to increase wheat productivity is the expansion of the acreage. However, area expansion is limited (Alexandratos and Bruinsma,

2012). Thus, increasing the yield potential per area is still one of the main objectives of wheat breeding to meet the requirements of continuous growing human population (Voss-Fels et al., 2019). In particular, improving photosynthetic capacity by increasing carbon fixation through the insertion of C4 plant genes may be an option (Reynolds et al., 2011). Nevertheless, in the context of climate change as a further future challenge, resistance against insects, viruses and fungi, as well as tolerance to abiotic stresses such as heat, drought and soil salinity become increasingly important in wheat breeding programs (Venske et al., 2019). Since the publication of the complete reference genome of wheat, a powerful tool is available for breeding and other genetic studies to enhance the understanding of wheat evolution, and to address the challenges for wheat production security (Appels et al., 2018).

Systematically, the wheat group *(Amblyopyrum*, *Aegilops*, and T*riticum*) is classified in the tribe Triticeae of the grass family Poaceae. The genus *Triticum* comprises in total six species at the diploid (*Triticum monococcum* L. and *Triticum urartu* Tum. Ex Grand.), and different polyploidy levels, from which the latter originated by hybridization between *Triticum* and the genus *Aegilops* (goatgrass): two tetraploid species, *Triticum turgidum* L. and *Triticum timopheevii* (Zhuk.) Zhuk., and two hexaploid species, *Triticum aestivum* L. and *Triticum zhukovskyi* Men. & Er. (Dvořák, 2001; Matsuoka, 2011; Feldman and Levy, 2015). Bread wheat is an allohexaploid species ($2n = 6x = 42$), that originated about 10,000 years ago in the Fertile Crescent, and more particularly in a region that nowadays surrounds the fertile alluvial plains of the Tigris and Euphrates rivers (Braidwood et al., 1969; Charmet, 2011; Venske et al., 2019). Two polyploidization events were involved in the evolution of bread wheat. First, natural hybridization between *Triticum urartu* (AA genome) and the donor of the BB genome (*Aegilops speltoides*-related species) formed *Triticum turgidum* ssp. *diccocoides* 0.5 million years ago. The second hybridization event took place between *Triticum turgidum* (AABB genome) and *Aegilops tauschii* (DD genome), forming *T. aestivum* (AABBDD), which through domestication and centuries of cultivation gave rise to the bread wheat cultivated today (Feldman et al., 1995; El Baidouri et al., 2017; Venske et al., 2019). Compared to most other crops (e.g. barley), wheat has a restricted genetic variability. The short period of time available for evolution is described to be one reason for this genetic narrowing, as there had not been sufficient time for mutations to accumulate or genes to be taken up by natural or artificial interspecific crossing processes (Cox, 1997; Venske et al., 2019). In addition, evolution only driven by polyploidization events and domestication, and generally, the emergence from only a few plants of ancestral species led to the restricted variability (Buckler et al., 2001; Dubcovsky and Dvorak, 2007; Charmet, 2011). Today, there are different strategies to broaden the genetic

diversity in wheat used in breeding programs. These strategies include traditional techniques as introgressions from the secondary and tertiary gene pools or mutagenesis, but also new molecular-based techniques as the genetic transformation or genome editing (Parry et al., 2009; Li et al., 2015; Crespo-Herrera et al., 2017; King et al., 2017; Wang et al., 2018).

2.   Wheat rusts

Wheat is exposed to several abiotic and biotic stressors leading to significant yield losses. Plant diseases are the most important biotic constraint on wheat production and threaten the global food supply. Around 40 fungal, 32 viral and 81 bacterial diseases of wheat are described (Bonjean and Angus, 2001). Among the fungal diseases, wheat rusts are the most economically important diseases (Gessese, 2019).

In general, rust fungi belong to the order Uredinales, which comprises more than 7000 species. The largest genera include the genus *Uromyces* with 600 species and the genus *Puccinia* with around 4000 species, causing damage to a wide range of important agricultural crops (Maier et al., 2003). Wheat rusts belong to the genus *Puccinia*, in which several special forms or species exist that differ in their ability to infect certain grasses and thus are classified as formae speciales (ff. spp.; Agrios, 2005; Figueroa et al., 2018). Furthermore, each formae speciales can be differentiated into many races or pathotypes that are only able to infect certain varieties of respective plant species (McIntosh et al., 1995; Gessese, 2019). Rust fungi belong to the fungal pathogens with an obligate biotrophic lifestyle and depend in consequence on living host cells to complete their life cycle and therefore form specialized infection structures to extract nutrients (Mendgen and Hahn, 2002; Voegele et al., 2009). Wheat rusts are heteroecious and macrocyclic and thus complete their life cycle on two taxonomically unrelated hosts by forming five distinct spore phases (Figure I.2-1, Kolmer, 2013). In successfully infected wheat, thousands of dikaryotic uredinospores (N+N) are produced in the uredium. In this asexual reproductive phase, the urediniospores produced are capable of leading to several re-infection cycles of wheat within one growing season. At the end of wheat growth, the uredinial infection transitions into the formation of telia, which produce thick-walled, durable dikaryotic teliospores (N+N). In spring and at the beginning of the sexual infection cycle, teliospores undergo nuclear fusion (karyogamy, NN) and meiosis to produce four binucleate, double haploid basidiospores (2N) that are forcibly released into the air to infect the secondary host. During successful infections, pycnia of two mating types are formed on the upper leaf surface with hyphae producing mating type-specific mononucleate haploid pycnospores (N). The pycnospores are carried by insects to other pycnia, where fusion of the pycnospores with

corresponding hyphae of an opposite mating type leads to plasmogamy and the development of aecia on the underside of leaves. This event of plasmogamy may occur several times within a single pycnium, giving rise to genetically distinct aecia. Dikaryotic aeciospores (N+N) are produced in a mature aecium, which are subsequently spread by wind and infect the primary host, e.g., wheat. There, uredinial infections develop and the sexual life cycle is completed (Kolmer, 2013; Schwessinger, 2017). There are three different wheat rust diseases caused by three distinct species: *Puccinia graminis* f. sp. *tritici*, *Puccinia striiformis* f. sp. *tritici* and *Puccinia triticina*.
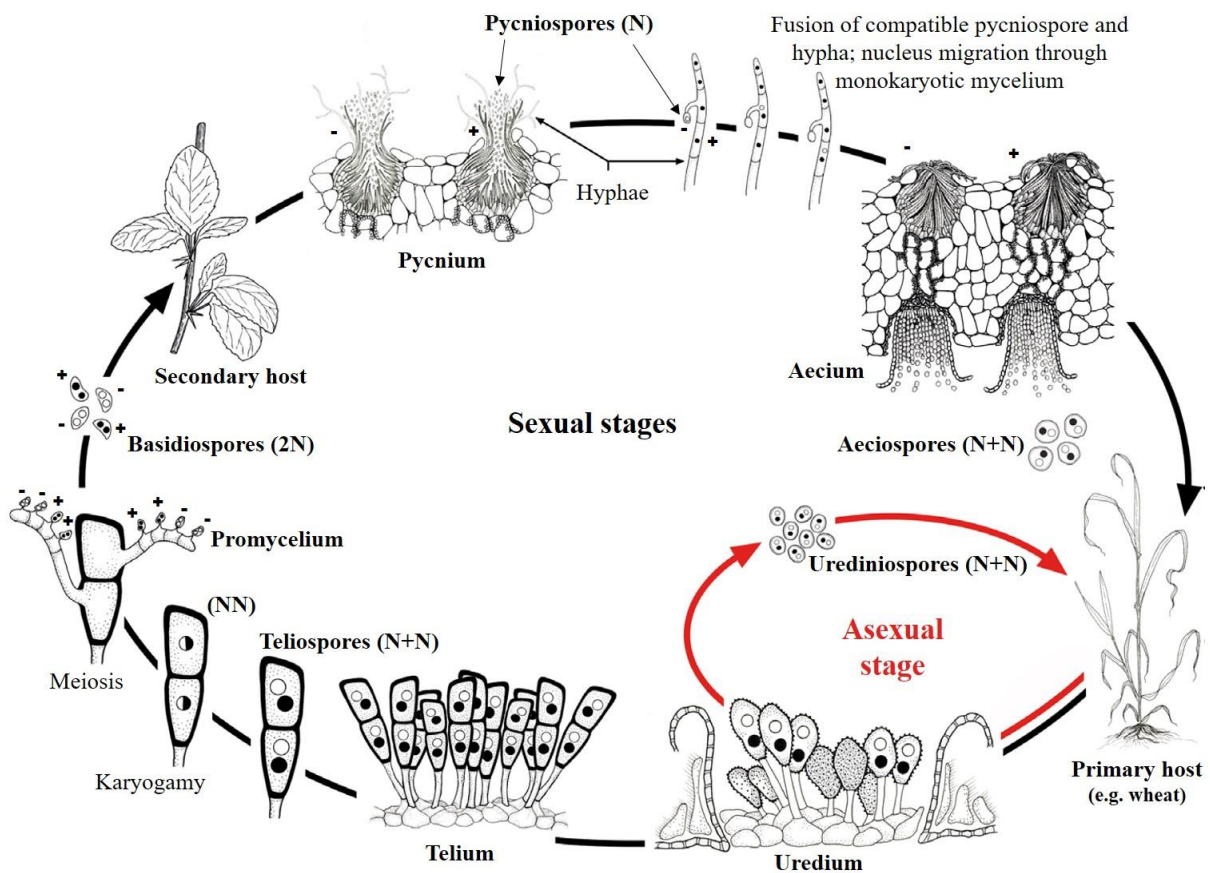
Figure I.2-1 | Life cycle of heteroecious, macrocyclic wheat rusts (modified according to Kolmer, 2013; original illustration from Jacolyn A. Morrison, USDA-ARS, USA)

Puccinia graminis f. sp. *tritici* Ericks and Henn is the causal agent of stem (black) rust, which has been one of the most devastating diseases of cereals and acted as a driving force during the Green Revolution (Figueroa et al., 2018; Gessese, 2019). Especially in Western Europe and North America, the elimination of barberry species acting as alternative hosts and the general distribution of semi-dwarf, high-yielding, resistant cultivars led to the successful control of stem rust in many parts of the world (Sharma, 2012). However, the emergence of the highly virulent 'Ug99' race in Uganda in 1998, its geographical spread across Africa and the Middle East, and the appearance of 'Ug99' variants signaled the return of this devastating disease (Singh et al., 2011; Singh et al., 2015). It is suggested that 90 % of the commercial wheat cultivars in the world are susceptible to 'Ug99' (Singh et al., 2011). In Addition, non-'Ug99' races such as the 'Digalu' race caused a devastating epidemic in Ethiopia in 2014 making stem rust currently the major concern in East Africa, which is also threatening wheat production in North and South Africa, Far East and West Asia, Australia, New Zealand, and South America (Olivera et al., 2015; Figueroa et al., 2018). Stem rust covers mainly the surface of leaves and stems by forming masses of redbrick urediniospores (Figure I.2-2, C). Susceptible varieties may also show infections on glumes and awns (Kolmer, 2005). Yield losses are associated with a reduction in the number of shoots and grains per ear, reduced grain size, as well as lodging of plants and may reach up to 100% depending on the susceptibility of the variety and favorable weather conditions (Roelfs, 1985a, 1985b; Leonard and Szabo, 2005). In Africa, the Middle East and South Asia, the estimated annual yield losses due to 'Ug99' stem rust race amount to approximately USD 3 billion (Singh, 2006).

Leaf rust is caused by *Puccinia triticina* Eriks. and is the most common and widespread rust disease of wheat. Compared to stem and stripe rust, leaf rust occurs more frequently and in most regions worldwide, representing one of the major constraints to wheat in North Africa, Asia (Central, South and Southeast), Europe, North and South Americas, Australia, and New Zealand (Bolton et al., 2008; Huerta-Espino et al., 2011). Common wheat, durum wheat and wild emmer are known as primary hosts of leaf rust. *Thalictrum speciosissimum*, *Isopyrum fumaroides* and *Anchusa azurea* are reported as secondary hosts needed to complete the whole life cycle (Bolton et al., 2008; Martinez-Moreno and Solís, 2019). Both primary and secondary hosts grow in the Fertile Crescent, where the sexual and asexual phases of *P. triticina* also exist. Therefore, it is assumed that leaf rust originated in this area (Bolton et al., 2008). However, the sexual phase contributes less to the direct inoculum, as the population of *P. triticina* is clonal in most parts of the world (Kolmer, 2005; Gessese, 2019). Urediniospores are widely spread by wind and infect host plants over several kilometers or even across countries (Kolmer, 2005).

Typical symptoms of infections caused by *P. triticina* are reddish-brown pustules of urediniospores distributed over the whole leaf (Figure I.2-2, A). A flag leaf infection of approximate 60-70 % at the time of ear emergence can lead to yield losses of 30 %. Under favorable conditions with temperatures of 20°C, however, leaf rust infection in the early stage may account for yield losses of more than 50 %. (Roelfs, 1992; Huerta-Espino et al., 2011). These yield losses are mainly associated with a reduction of the kernel weight and the number of grains per ear (Figueroa et al., 2018). Between 2003 and 2008, leaf rust caused yield losses of 2.5 dt ha^{-1} on average in Germany, with an average infestation rate of 19 %. (Jahn et al., 2012). Losses due to leaf rust in the USA from 2000 to 2004 were estimated at over 3 million tons, with a market value of over 350 million dollars (Huerta-Espino et al., 2011). Furthermore, Miedaner and Juroszek (2021) predict that due to climate change, the incidence and severity of wheat leaf rust in NW Europe will further increase during this century.

The causal agent of stripe rust (yellow rust) is *Puccinia striiformis* Westend. which causes infection on various grass genera by host specialization and comprises up to nine formae speciales (Eriksson, 1894; Chen et al., 2014). Of these, *Puccinia striiformis* f. sp. *tritici* Erikss. is responsible for stripe rust on common wheat, durum wheat, cultivated and wild emmer, as well as triticale and is considered to be the most common among the three rust fungi on wheat (Gessese, 2019). It is assumed that stripe rust was present long before wheat was cultivated and originated in the South Caucasus (Hassebrauk, 1965). First described in Europe in 1777, stripe rust is nowadays widespread in all temperate regions with cool and moist weather conditions and is common in over 60 countries on all continents (Carver, 2009; Chen et al., 2014; Waqar et al., 2018). As there were no alternative hosts known until 2010, *P. striiformis* f. sp. *tritici* was assumed to only produce dikaryotic uredial and telial stages on primary hosts (Chen, 2005). However, additional sexual stages are now reported on *Berberis* spp. and *Mahonia* spp. which act as alternative hosts and allow *P. striiformis* to complete its life cycle (Jin et al., 2010; Zhao et al., 2011; Wang and Chen, 2013; Zhao et al., 2013). On wheat, *P. striiformis* f. sp. *tritici* forms yellow to orange-colored urediniospores erupting from uredinia arranged in long, narrow stripes on the upper surface of leaves (Figure I.2-2, B). On susceptible plants, uredinia that are generally smaller than uredinia of stem or leave rust, may also be formed on leaf sheaths, glumes and awns. Under favorable conditions with temperatures of 10 - 12°C and depending on the susceptibility of the variety, the time of onset of the infection and the disease progress, stripe rust can cause yield losses up to 70 % (Carver, 2009; Begum et al., 2014). These yield and quality losses are mainly due to reduced grain size, number and weight of grains per ear and reduced dry matter (Wellings, 2011; Bux et al., 2012). Global yield losses caused by stripe

rust are estimated at USD 1 billion annually (Wellings, 2011; Beddow et al., 2015). *P. striiformis* f. sp. *tritici* is characterized by rapid emergence of virulent races and high pathogenic variability. This variability is due to the high reproducibility and genetic diversity as a result of sexual recombination, the ability to spread over long distances and adapt to different environments (Carmona et al., 2020). In the last two decades, new aggressive races of *P. striiformis* f. sp. *tritici* have emerged that are adapted to a climate with higher temperatures and have spread to regions in which stripe rust was previously less common (Ali et al., 2014). Considering this trend in the context of climate change and the general emergence of races showing virulence against resistances commonly present in cultivars, as e.g. the 'Warrior' races, stripe rust can be considered the economically most important wheat rust disease and threat to food security worldwide (Carmona et al., 2020; Miedaner and Juroszek, 2021).
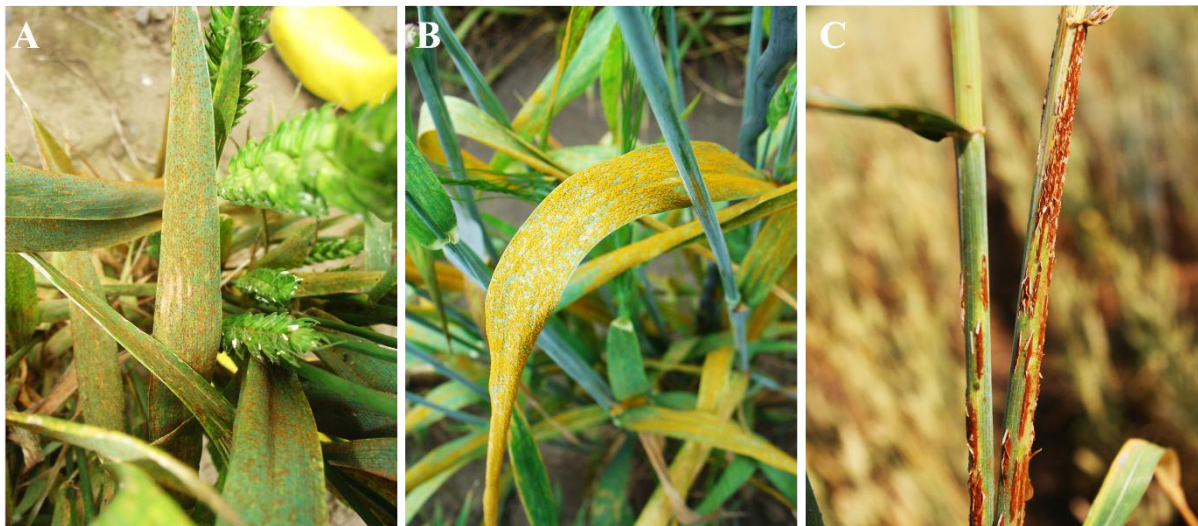
Figure I.2-2 | Characteristic symptoms of leaf rust (**A**, photo: Albrecht Serfling, JKI Germany), stripe rust (**B**, photo: Albrecht Serfling, JKI Germany) and stem rust (**C**, photo: Albrecht Serfling, JKI Germany) on wheat.

Due to the rapid spread and the frequency of development of new races, the management of cereal rust diseases is complex and it is recommended to use a combination of different methods to manage cereal rusts in wheat production (Singh et al., 2005). Rust disease control strategies include the use of different cultural practices, such as appropriate use of fertilizers, frequency and amount of irrigation, timing of seeding, and crop rotation (Roelfs, 1992; Neumann et al., 2004; Wan et al., 2007; Simón et al., 2011). In addition, the elimination of 'green bridges' between crops by tillage and the eradication of the alternative host help to control rust diseases by reducing the amount of endogenous inoculum that can infect wheat (Zadoks and Bouwman, 1985; Kolmer et al., 2007). A second line of defense is the chemical control of rust diseases by the use of fungicides, especially when new virulent races attack wheat varieties that were

previously resistant (Loughman et al., 2005). Fungicides such as quinone outside inhibitors (QoIs), 14α-demethylation inhibitors (DMIs) and more recently succinate dehydrogenase inhibitors (SDHI) have been approved and showed efficient control against wheat rusts (Oliver, 2014). In particular, QoIs and DMIs have maintained their performance and efficacy, either because rusts have an intron that prevents the G143A mutation that would confer robust resistance to QoIs, or because DMIs are low-risk resistance molecules (Carmona et al., 2020). However, not only chemical but also microbial bio-pesticides can be used to avoid rust epidemics. For example, the bacterial strain *Pseudomonas putida* has shown the ability to produce different types of antibiotics, siderophores and a low quantity of hydrogen cyanide (HCN), which inhibit the growth of *P. triticina* (Flaishman et al., 1996). Furthermore, the endophytic *Bacillus subtilis* strain E1R-j isolated from wheat roots inhibited the urediniospore germination of *P. striiformis* f. sp. *tritici* and demonstrated effective biological control in both greenhouse and field trials (Li et al., 2013). In another approach, the combined application of arbuscular mycorrhizal fungi and *Azospirillum amazonense* improved the growth, yield and quality of wheat plants, and reduced the severity of rust disease (Ghoneem et al., 2015; Savadi et al., 2018). Nevertheless, besides the use of early warning systems with regular pathogen monitoring and disease scoring, the use of genetic resistance has achieved resounding success in the management of various rusts in the past (Carmona et al., 2020). Use of resistant cultivars is the most effective, economic and environmentally friendly approach to manage cereal rusts. To date, more than 80 resistance genes to leaf rust (*Lr* genes), 82 stripe rust resistance genes (*Yr* genes) and over 61 stem rust resistance genes (*Sr* genes) are known with their respective chromosomal locations (Gill et al., 2019; McIntosh et al., 2019, 2020)

3.   Disease resistance mechanism against wheat rusts

Compared to animals, plant defense mechanisms against pathogen attacks are different because there is no intrinsic immune system in plants with antibodies that bind to foreign antigens to eliminate them. Nevertheless, plants have evolved complex defense mechanisms, in which each plant cell has an innate immune system that defends it against pathogen attack, and in which the plant uses systemic signals emanating from sources of infection (Jones and Dangl, 2006; Woloshen et al., 2011). The plant immune system is essentially divided into two branches, the basal disease resistance that forms the first line of defense, and effector-triggered immunity as the second line of defense (Figure I.3-1). In case a pathogen attacks a plant, microbe- or pathogen-associated molecular pattern (MAMP or PAMP), such as flagellin, are recognized by transmembrane pattern recognition receptors (PRRs) in the plant, triggering various events and

referred to as pattern-triggered immunity (PTI) (Jones and Dangl, 2006). However, pathogens are able to suppress PTI through the release of effectors capable of modulating host metabolism and defense responses, which lead to effector-triggered susceptibility (ETS, Prasad et al., 2019). In general, effectors are proteins that are secreted into the host tissue during infection of pathogens and influence cell functions of the host plant in different subcellular compartments (Kamoun, 2006). Thereby, effectors either act in self-defense to protect the pathogen from antimicrobial compounds produced by the host plant or interact with host targets proteins that are for example involved in the second line of plant defense (Rovenich et al., 2014). In this second plant defense line, effector proteins produced by avirulence (Avr) genes in the pathogen are recognized by nucleotide binding site-leucine rich repeat (NB-LRR) proteins, predominately encoded by corresponding resistance (R) genes in plants (Flor, 1956; Juliana et al., 2018). This results in an effector-triggered immunity (ETI) that usually initiate a hypersensitive response (HR) and leads to a localized programmed cell death preventing further colonization of biotrophic fungi in plants (Heath, 2000; Jones and Dangl, 2006). Several effector proteins have been characterized in plant pathogens, including Pst_12806 in the stripe rust fungus *P. striiformis* f. sp. *tritici*. Pst_12806 suppresses the host basal immunity by reducing callose deposition and the expression of defense-related genes in wheat (Xu et al., 2019). Furthermore, recent studies have identified additional effector proteins in *P. striiformis* f. sp. *tritici*, e.g. Pst18363 and PstGSRE1 (Qi et al., 2019; Yang et al., 2020). Pst18363 targets and stabilizes the wheat Nudix hydrolase 23 TaNUDX23, which in turn suppresses the effector Pst322-trigged cell death in wheat and thus supports colonization by *P. striiformis* f. sp. *tritici* (Yang et al., 2020). The effector PstGSRE1 compromises host immunity by disrupting the nuclear localization of the reactive oxygen species (ROS)-associated transcription factor TaLOL2 that suppresses ROS-mediated cell death induced by TaLOL2 (Qi et al., 2019).
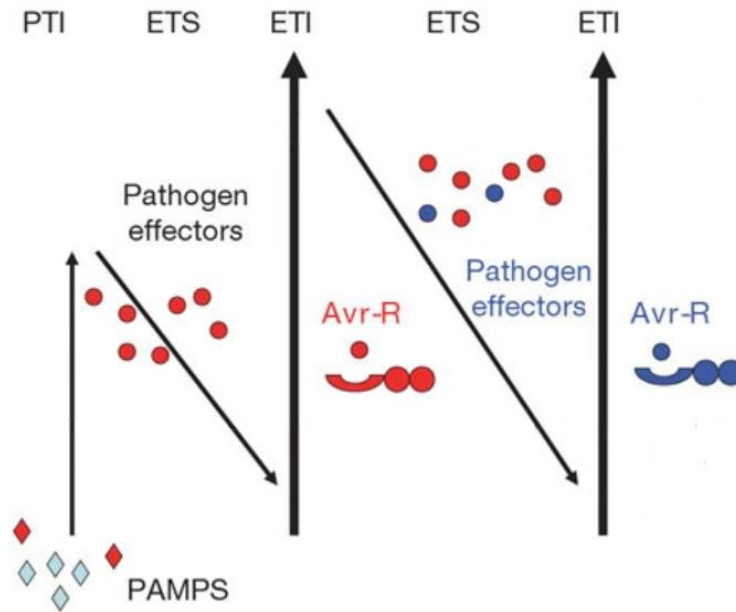
Figure I.3-1 | Exemplary illustration of the plant immune system (modified according to Jones and Dangl, 2006). The scheme is divided into four phases: 1) Plant detects microbe- or pathogen-associated molecular pattern (MAMP/PAMP; red diamonds) by pattern recognition receptors (PRRs) resulting in a pattern-triggered immunity (PTI); 2) Successfully colonized pathogen suppresses PTI through the secretion of effectors; 3) Individual effectors (marked in red) are recognized by nucleotide binding site-leucine rich repeat (NB-LRR) proteins, activating effector-triggered immunity (ETI), which might induce hypersensitive response (HR); 4) Pathogen races are selected, which gained new effectors suppressing the ETI. Selection favors new plant NB-LRR alleles, which in turn leads to ETI.

The genetic basis of wheat rust resistance is closely related to host-pathogen interaction and can therefore be classified as race-specific or non-race-specific resistance. Race-specific resistance also termed as qualitative or seedling resistance is monogenetically inherited, expressed throughout all growth stages and the underlying major genes are only effective against a subset of races of rust pathogens (Chen, 2005). Thus, race-specific resistance genes follow the classical gene-for-gene model, in which resistance responses are only facilitated n case the R protein of the plant recognizes the corresponding Avr gene product of the pathogen. If an Avr gene is either not present or mutated, the plant will be susceptible regardless of the presence of the corresponding R gene (Flor, 1956, 1971; Crute et al., 1997). Most of the 80, 81 and 61 designated leaf, stripe and stem rust resistance genes, respectively, are classified as race-specific since their very distinct phenotypic effects with high resistance values conferred by single genes can be rapidly detected in seedling tests, making R genes the first class of resistance genes to be genetically defined (Ellis et al., 2014). The use of race-specific resistance in plants by breeders is common over large geographical areas, leading to breakdown of major resistance genes according to the so-called boom-and-bust cycles (Figure I.3-2, McDonald and Linde, 2002). With increased popularity due to resistance and thus increased acreage of a

cultivar carrying a single resistance gene (boom), the selection pressure against the corresponding avirulence gene carried by a pathogen population also increases. Consequently, only one single mutation event at the avirulence locus can result in the development of a new virulent pathotype that overcomes the resistance (bust, Pink, 2002). For many race-specific resistance genes, e.g. leaf rust resistances *Lr26*, *Lr37*, or stripe rust resistances *Yr10*, *Yr24* and *Yr27*, it has already been reported that virulent races of *P. triticina* and *P. striiformis* f. sp. *tritici* emerged and overcame these resistance genes (Kolmer, 2005; Huerta-Espino et al., 2011; Hovmøller et al., 2017).
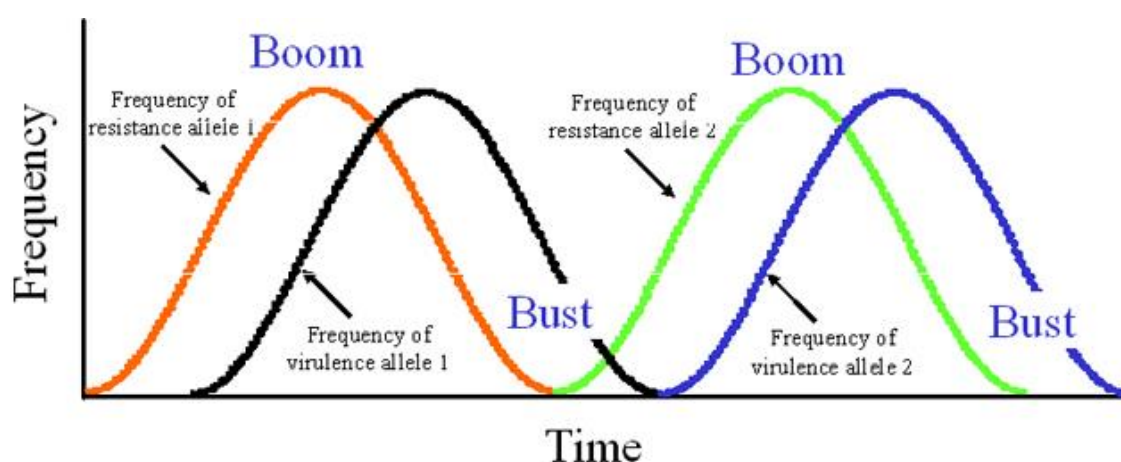
Figure I.3-2 | Boom and bust cycles related to resistance and virulence allele frequencies (www.apsnet.org).

Non-race-specific resistance operates against a broad range of races and is sometimes even effective against multiple pathogens. It is primarily inherited quantitatively, expressed at later growth stages and therefore termed quantitative or adult plant resistance (APR). Quantitative APR only confers partial resistance, in which the pathogen grows is less and slowed down and no necrotic response is triggered (Krattinger and Keller, 2016; Periyannan et al., 2017). However, it has been reported that the combination of individual quantitative APR genes showing different levels of partial resistance cause "near immunity" in adult plants grown in the field (Singh et al., 2014). In contrast to most NB-LRR-encoding R genes, quantitative APR genes appear to encode different proteins, such as ATP-binding cassette (ABC) transporters, protein kinases or hexose transporters, and are considered to be genetically durable (Ellis et al., 2014; Moore et al., 2015; Periyannan et al., 2017). Several studies have demonstrated that durability of resistance is more likely with quantitative APR than R genes, as in case of *Lr34/Yr18/Sr57/Pm38* (Singh, 1992), *Lr46/Yr29/Pm39* (Kolmer et al., 2015), *Lr67/Yr46* (Herrera-Foessel et al., 2014). Resistance is considered durable if it remains effective within a cultivar under cultivation for a significant number of years over a substantial area with favorable

conditions for the respective pathogen (Johnson, 1984). However, masking of APR by R genes with stronger resistance phenotypes prevents effective selection of quantitative APR, making APR breeding more complex than using R genes (Ellis et al., 2014). Pyramiding of a set of single race specific R genes conferring complete resistance with some non-race specific APR genes could be an efficient tool create a significantly broader durable resistance over a significant number of years in plants (Figlan et al., 2020).

To date, 25 wheat genes conferring resistance to stem rust (*Sr13*, *Sr21*, *Sr22*, *Sr33*, *Sr35*, *Sr45*, *Sr46*, *Sr50*, *Sr55*, *Sr57*, and *Sr60*), leaf rust (*Lr1*, *Lr10*, *Lr21*, *Lr22a*, *Lr34*, and *Lr67*) and stripe rust (*Yr5*, *Yr7*, *Yr10*, *Yr15*, *Yr18*, *Yr36*, *Yr46*, and *YrSP*) have been cloned (Mago et al.; Feuillet et al., 2003; Huang et al., 2003; Cloutier et al., 2007; Fu et al., 2009; Krattinger et al., 2009; Periyannan et al., 2013; Saintenac et al., 2013; Liu et al., 2014; Moore et al., 2015; Steuernagel et al., 2016; Thind et al., 2017; Zhang et al., 2017; Chen et al., 2018; Klymiuk et al., 2018; Marchal et al., 2018; Arora et al., 2019; Chen et al., 2020). Among these, *Lr1*, *Lr10*, *Lr21, Lr22a, Yr5, Yr7, Yr10, YrSP, Sr13, Sr21, Sr22, Sr33, Sr35, Sr45* and *Sr50* are race-specific genes encoding typical nucleotide-binding and leucine-rich repeat (NLR) proteins (Kim et al., 2020; Wang et al., 2020; Desiderio et al., 2021). However, *Lr34/Yr18/Sr57/Pm38* and *Lr67/Yr46/Sr55/Pm38* confer quantitative APR to various rust and powdery mildew fungi encoding an ABC transporter and hexose transporter, respectively. These multi-pathogen resistances are characterized by causing necrosis of leaf tips with accelerated senescence (Periyannan et al., 2017). In contrast, the stripe rust resistance genes *Yr15* and *Yr36* encode a protein with predicted kinase/pseudokinase domain and a protein with a kinase domain and a putative lipid-binding domain, respectively (Wang et al., 2020). In particular, *Yr36* encodes a chloroplast-localized protein with kinase and steroidogenic acute regulatory protein-related transfer (START) lipid-binding domains and is thought to reduce detoxification of reactive oxygen species by phosphorylating a thylakoid-associated ascorbate peroxidase, leading to enhanced defense responses (Gou et al., 2015; Periyannan et al., 2017).

4. Molecular Marker and QTL detection methods

In recent decades, great efforts have been made to identify regions of the genome that contain genes associated with a quantitative trait called quantitative trait loci (QTL) (Collard et al., 2005). As breeders before the 1990s were mostly limited to morphological markers, such as color, shape and size of flower, seeds or leaves, molecular markers have emerged in recent years as a powerful tool for analyzing genetic variation, allowing linkage of phenotypic and genotypic variation (Varshney et al., 2005; Grover and Sharma, 2016). Today, several marker

analysis methods have been developed, all of which must meet the following criteria: i) reliability through proximity of the markers to a locus under investigation; ii) polymorphisms to distinguish between different genotypes; iii) simple and fast technique; and iv) low need for genetic material to perform the analyses (Garrido-Cardenas et al., 2018). These molecular marker techniques can be divided into three categories depending on the method of analysis: non-PCR-based techniques, PCR-based techniques and sequence-based marker techniques (Lander and Botstein, 1989; O'Hanlon et al., 2000; Ganal et al., 2012). However, the decision for one or the other technique depends strongly on the size and composition of the plant population and the number of segregating genes in a population (Collard and Mackill, 2008; Garrido-Cardenas et al., 2018).

Restriction fragment length polymorphism (RFLP) is the only marker system representing hybridization-based markers. This involves the use of restriction enzymes and hybridization by labelling a target DNA fragment to be used as a probe in Southern blot analysis (Williams, 1989). RFLP markers are able to detect both alleles in a heterozygous sample resulting from either point mutation, DNA insertion, deletion or rearrangement. As RFLP are co-dominant, they are advantageous as a reliable marker for linkage analyses, QTL analyses and genetic fingerprinting. However, a large and high-quality quantity of DNA with known sequence is required, and labelling the probes with radioisotopes makes RFLP a time-consuming, expensive and dangerous technique, which is not in use anymore (Beckmann and Soller, 1983; Collard et al., 2005; Garrido-Cardenas et al., 2018). In contrast to this technique, the aim of using Randomly amplified polymorphic DNA (RAPD) markers is to obtain fragments of different sizes resulting from the random amplification of DNA sequences by PCR reaction (Williams et al., 1990). Thus, knowledge of the sequence is not necessary and less amount of DNA is sufficient. Disadvantages are that most of the markers are dominant and the bad reproducibility (Collard et al., 2005). Amplified fragment length polymorphism (AFLP) combines both the RFLP and RAPD techniques. It selects restriction fragments generated from a total digest of genomic DNA by PCR amplification. Again, no prior knowledge of the sequence is required and fingerprints can be made from any DNA regardless of its origin or complexity, resulting in high reproducibility. The disadvantage is that a large amount of DNA is required and the complex methodology makes the technique time-consuming and laborious (Vos et al., 1995). Simple sequence repeats (SSR) or microsatellites are short tandem repeats of DNA sequences of 1-10 base pairs that occur in both coding and non-coding regions of all eukaryotic and prokaryotic genomes (Vieira et al., 2016). These sequences are amplified using flanking primers in a PCR reaction, and the length of the products is subsequently determined by high-

resolution gel or capillary electrophoresis. The use of SSR markers is a simple, robust and reliable technique, and their codominant inheritance provides complete genetic information. The main drawback is the cost-, time- and labor-intensive development of new primers (Collard et al., 2005). Single nucleotide polymorphism (SNP) markers take advantage of detecting polymorphisms between individuals based on point mutations in single nucleotide positions. In other words, SNPs are a DNA sequence variation that occurs when a single nucleotide (A, T, G or C) differs between individuals of one species. Depending on the type of mutation, SNPs can be divided into three categories: (i) transversions with changes in nucleotides C/G, A/T, C/A and T/G; (ii) transitions involving changes in C/T or G/A; and (iii) indels resulting from insertion or deletion of a single nucleotide (Garrido-Cardenas et al., 2018). Due to their bi-allelic and codominant properties, they are extremely useful for a variety of analyses, as they can evaluate a large number of loci and efficiently distinguish between homozygous and heterozygous alleles. In contrast to multiallelic markers, the analysis of bi-allelic SNP markers can be completely automated. (Khlestkina and Salina, 2006; Garrido-Cardenas et al., 2018). Since the end of the 20th century, the introduction of microarrays (SNP arrays) has enabled the analysis of thousands of SNPs simultaneously in a single reaction (Kerr et al., 2000). In these arrays, thousands of genomic sequences are bound to a solid surface and hybridized with a corresponding biological sample, which has previously been fluorescently labelled. Each fluorescent signal is then detected individually, resulting in a hybridization map (Heller, 2002). Today, several genotyping SNP arrays are available for wheat, such as the 9K and 90K iSelect, as well as the 820K Axiom® array, developed for hexaploid wheat and its secondary and tertiary gene pool (Cavanagh et al., 2013; Wang et al., 2014; Winfield et al., 2016). These arrays were used to create genetic consensus maps that mapped e.g. 40,267 SNP markers from 81,587 SNPs based on eight biparental populations, providing an essential resource for diversity studies and wheat breeding in general (Wang et al., 2014; Wen et al., 2017).

One of the main applications of molecular markers is the construction of linkage maps for different plant species, reflecting the position and relative genetic distance between markers along the chromosomes and used to identify single genes controlling simple traits and QTL. The process of constructing linkage maps for QTL analyses is known as QTL mapping, in which molecular markers do not represent the target genes themselves, but act as a kind of landmark for the chromosomal region of interest (Collard et al., 2005). The principle of QTL mapping is based on the linkage of molecular markers and genes of interest during sexual reproduction, which allows analysis in the progeny (Paterson, 1996). Another prerequisite for QTL mapping is the generation of a segregating (mapping) population, which are mainly bi-

parental populations such as F_2, backcrosses (BC), doubled haploids (DH), recombinant inbred lines (RILS) or near-isogenic lines (NIL). For each such segregating population used for QTL mapping, an individual linkage map is generated based on genotypic data, and phenotypic data are generated for the trait of interest (Xu et al., 2017). The principle of the QTL analysis is based on the detection of an association between the phenotype and the genotype of the markers used. Based on the presence or absence of a particular marker locus, the corresponding mapping population is divided into different genotypic groups to determine whether significant differences exist between these groups with respect to the phenotypic data of the measured trait (Tanksley, 1993; Young, 1996; Collard et al., 2005). There are four mainly-used QTL mapping methods described: i) Single-marker analysis, ii) simple interval mapping, iii) composite interval mapping and iv) multiple interval mapping (Lander and Botstein, 1989; Tanksley, 1993; Zeng, 1993; Kao et al., 1999). Single-marker analysis is the simplest method using statistical procedures such as t-test, analysis of variance (ANOVA) and linear regression. The advantage is that no complete linkage map is required and the method can be performed with simple statistical software programs. However, the main disadvantage is the lower detection power and the underestimation of QTL effects (Tanksley, 1993). Simple interval mapping (SIM) is based on the maximum likelihood parameter estimation, uses linkage maps and simultaneously analyses the intervals between adjacent pairs of linked markers along chromosomes (Lander and Botstein, 1989). Thus, it is considered statistically more powerful and provides improved estimates of QTL effects compared to single marker analysis. The main drawback of this method is the increased computation time and the requirement for specifically designed software (Broman, 2001). Composite interval mapping (CIM) combines interval mapping with linear regression and includes additional marker loci as covariates. These markers serve as proxies for other QTL to increase the resolution of interval mapping by taking into account linked QTL and reducing residual variation (Zeng, 1993; Broman, 2001). However, the selection of the covariates is considered critical. Once the markers have been selected as covariates, CIM transforms the multidimensional QTL scan into a one-dimensional scan. Uncertainty in the selection of relevant marker covariates can then lead to an overly optimistic estimate of the accuracy of QTL localization (Broman and Sen, 2009). Multiple interval mapping (MIM) is the extension of standard interval mapping to a multiple QTL model. MIM allows the location of QTL to infer positions between markers, takes into account missing genotype data and can account for interactions between QTL. Besides the advantages of QTL mapping in which fewer markers are required and rare alleles can be identified, the major limitation is that only the diversity of segregating alleles between parents used to construct the

mapping population can be tested. In addition, the construction of a mapping population is time-consuming, and the use of bi-parental populations leads to a low number of recombination and thus to a lower mapping resolution (Mitchell-Olds, 2010; Alqudah et al., 2020).

In recent years, these limitations have been partly overcome by genome-wide association studies (GWAS), which rely on linkage disequilibrium (LD) structure throughout the genome of genetically diverse populations to calculate the association between markers and phenotypes of interest (Zhu et al., 2008). Thus GWAS offers three advantages compared to traditional linkage analyses: i) reduced time, ii) greater allele number and iii) increased mapping resolution (Yu and Buckler, 2006). However, the power of GWAS to detect associations strongly depends on the phenotypic variation, population size, allele frequency and the population structure (Alqudah et al., 2020). For example, outliers should be removed from the raw phenotypic data, as they lead to deviating from the normal distribution and thus limit the GWAS. Furthermore, only traits with medium and high broad-sense heritability should be considered. The population size is of great importance as it influences not only detection power, but also the possibility of detecting rare alleles and LD-decay, which is particularly important for mapping resolution. In general, a range between 100 to 500 individuals is considered suitable for performing GWAS. In natural populations used for GWAS, the relatedness between some individuals is closer than between other individuals. These specific differences in relatedness are the most limiting factor, can lead to non-functional, spurious associations and must therefore be taken into account by calculating population structure and kinship (Pritchard et al., 2000; Yu et al., 2006; Alqudah et al., 2020).

Regardless of the detection method used, molecular markers linked to agronomical important genes or associated with a trait of interest, respectively, can be used as a molecular tool for marker-assisted selection (MAS). MAS uses the presence or absence of a marker to replace or support phenotypic selection in a way that is more efficient, effective, reliable and cost-effective compared to conventional plant breeding (Collard et al., 2005). Furthermore, fully annotated and ordered genome sequences have promoted the development of systematic and time-efficient approaches to the selection and understanding of important traits, and facilitated the selection of candidate genes based on the predicted functions of the genes and gene ontologies (Borevitz and Chory, 2004; Appels et al., 2018). Thus, the recently published reference sequence Chinese Spring v1.0 is of great importance for wheat improvement through more reliable and accurate mapping of QTL (Appels et al., 2018). Another important milestone for the more precise identification of possible candidate genes is the international development of the pan-genome in wheat. A pan-genome describes a collection of all DNA sequences that

occur not only in one variety, but in one species. This improves the identification of genes and/or phenotypically consequential variants (Sherman and Salzberg, 2020).

5.   Multiparental populations

Thanks to new molecular techniques such as next generation sequencing (NGS), resulting in sequencing several thousand molecules of genetic material simultaneously, as well as the publication of the complete reference genome of wheat, marker information and its availability are no longer the limiting factor, but rather the phenotypic variation required to perform QTL analyses (Hall, 2007; Gibson, 2012; He et al., 2014; Appels et al., 2018). However, with the development of multiparental approaches such as nested association mapping (NAM) and multiparent advanced generation intercross (MAGIC) populations, attempts have been made in recent years to overcome this limitation while combining the advantages of QTL mapping and GWAS populations (Cavanagh et al., 2008; Yu et al., 2008; Huang et al., 2015). NAM bases on a multiparental crossing design in which F_1 plants arise from a series of bi-parental crosses between a common parental line and n other highly divergent founder lines. To produce the nested population, the F_1 progeny are subsequently backcrossed to the common parental line and then selfed several times, producing n RIL families (Maurer et al., 2015; Kidane et al., 2019). Thus, NAM offers the advantages of lower sensitivity to genetic heterogeneity and higher performance and efficiency in using the genome sequence or high marker density while maintaining high allelic richness due to different founders (Yu et al., 2008). The first NAM design has been applied in maize to dissect the genetic basis of complex quantitative traits (Yu et al., 2008). Nowadays, NAM populations are also used in other crops such as barley (*Hordeum vulgare*), rice, sorghum (*Sorghum bicolor*), wheat, and soybean (*Glycine max* L.) (Maurer et al., 2015; Bouchet et al., 2017; Fragoso et al., 2017; Jordan et al., 2018; Xavier et al., 2018). MAGIC populations were first exploited in mice for animals and in *Arabidopsis thaliana* for plants and typically derived from repeated intercrosses of four, eight or 16 parents in a balanced funnel scheme (The Complex Trait Consortium, 2004; Kover et al., 2009; Huang et al., 2015). Today, several MAGIC populations have been developed for different economical important crops, such as wheat, rice, maize, tomato (*Solanum lycopersicum*), faba bean (*Vicia faba* L.), barley, strawberry (*Fragaria* × *ananassa*), sorghum, and mustard (*Brassica juncea*) (Huang et al., 2012; Bandillo et al., 2013; Dell'Acqua et al., 2015; Pascual et al., 2015; Sallam and Martsch, 2015; Sannemann et al., 2015; Wada et al., 2017; Ongom and Ejeta, 2018; Yan et al., 2020). In general, the construction of a MAGIC population involves four steps: 1) selection of founder lines based on contrasting phenotypic and genotypic traits, 2) mixing of the selected

founders by intercrossing according to a funnel scheme, 3) random and sequential advanced intercrosses to produce recombinants in the population and 4) development of homozygous inbred lines through several steps of selfing. Compared to bi-parental populations, MAGIC populations capture increased genetic recombination and genetic variation, resulting in an increased power and resolution in QTL mapping (Samantara et al., 2021).

In wheat, there are a total of six MAGIC populations, of which the Bavarian MAGIC Wheat and WM-800 population were produced in Germany and are mainly based on German elite varieties (Huang et al., 2012; Mackay et al., 2014; Gardner et al., 2016; Milner et al., 2016; Sannemann et al., 2018; Stadlmeier et al., 2018). The Bavarian MAGIC Wheat population (BMWpop) comprises 394 diverse $F_{6:8}$ RILs, which are based on a simplified intercrossing design of the German and Danish winter wheat lines 'Event', 'Format', 'BAYP4535', 'Potenzial', 'Ambition', 'Bussard', 'Firl3565', and 'Julius' (Figure I.5-1).
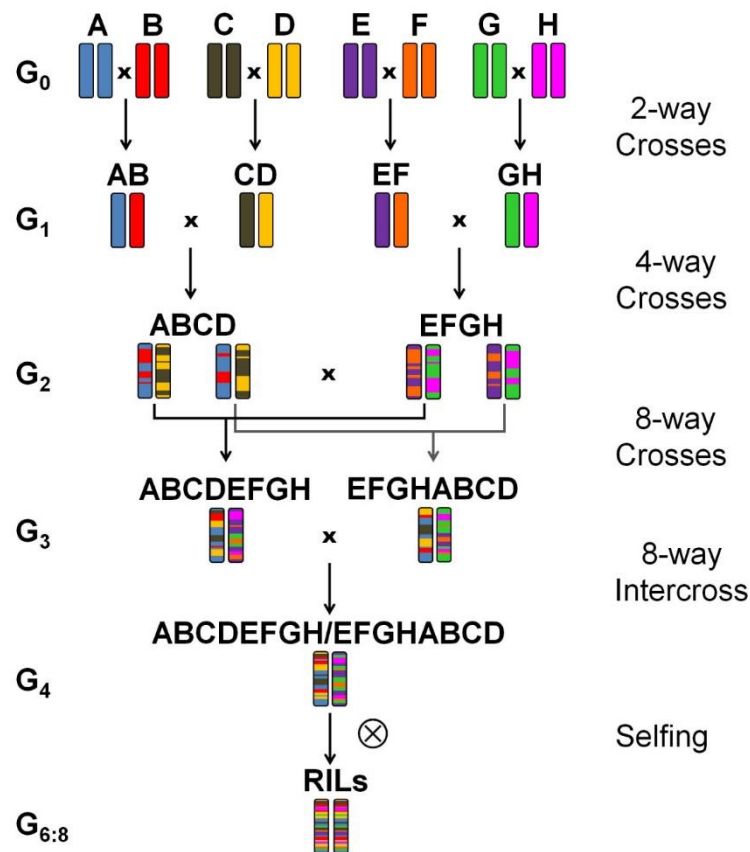
Figure I.5-1 | Crossing scheme of the eight-founder BMWpop. (A) 'Event', (B) 'Format', (C) 'BAYP4535', (D) 'Potenzial', (E) 'Ambition', (F) 'Bussard', (G) 'Firl3565', (H) 'Julius' (Stadlmeier et al., 2018).

Four two-way crosses (AB, CD, EF and GH) were performed to produce the F_1 progeny, which were further mated in 32 four-way crosses. Out of these, sixteen independently selected plants were subsequently crossed to obtain eight F_1 populations that involved four reciprocal cross combinations (ABCDEFGH and EFGHABCD). The four eight-way crosses were further

hybridized with the four reciprocal eight-way crosses to obtain 16 eight-way intercross combinations (ABCDEFGH/EFGHABCD). Next, the F_1 seeds were selfed to the F_6 generation via single seed descent followed by two generations of bulk propagation in the field (Stadlmeier et al., 2018). This makes BMWpop a valuable tool to conduct genetic studies for a wide range of economically important traits and uncover potential interactions related to breeding new wheat varieties.

6. Aim

The rust fungi stem rust (*Puccinia graminis* f. sp. *tritici* Ericks and Henn), stripe rust (*Puccinia striiformis* Westend.) and leaf rust (*Puccinia triticina* Eriks.) occur worldwide, pose a major obstacle to wheat production and thus threaten global food security. Known resistance genes to stem rust, stripe rust and leaf rust are present and described in many modern wheat varieties. However, according to the gene-by-gene hypothesis, these are restricted to certain races within the rust population and mainly show qualitative resistance. The emergence of new races of stem rust, stripe rust and leaf rust that possess virulence against common resistance genes has led to the breakdown of qualitative resistances and thus to an increased demand for more durable quantitative resistances. In science and industry, the search for new sources of durable resistance and the breeding of new resistant varieties is an ongoing process that has recently been forced by developments and advances in molecular markers and genotyping techniques. Furthermore, the development of multiparental-based approaches, such as MAGIC populations, combines the advantages of linkage analysis and association mapping to investigate genomic regions of interest with increased allelic variation and genetic resolution.

With a focus on leaf rust and stripe rust and based on the Bavarian MAGIC Wheat population, the following objectives were pursued in the present thesis: (i) genetic analyses of resistance to stripe rust and leaf rust in the context of multi-year field trials and macroscopic investigations in greenhouse experiments, (ii) identification of qualitative and quantitative resistances by QTL mapping, (iii) comparison of identified QTL to already known resistance loci in order to identify putative new resistances, and (iv) alignment of chromosomal QTL regions to the reference genome of wheat in order to find candidate genes in the background of resistances. Markers linked to these loci may be converted into KASP markers suitable for MAS in wheat breeding programs. In addition, the data set can also be used for genomic selection in the breeding material.

CHAPTER II| QTL MAPPING OF ADULT PLANT AND SEEDLING RESISTANCE TO LEAF RUST (*PUCCINIA TRITICINA* ERIKS.) IN A MULTIPARENT ADVANCED GENERATION INTERCROSS (MAGIC) WHEAT POPULATION

Sandra Rollar, Albrecht Serfling, Manuel Geyer, Lorenz Hartl, Volker Mohler, Frank Ordon

ORIGINAL ARTICLE

QTL mapping of adult plant and seedling resistance to leaf rust (*Puccinia triticina* Eriks.) in a multiparent advanced generation intercross (MAGIC) wheat population

Sandra Rollar[1] · Albrecht Serfling[1] · Manuel Geyer[2] · Lorenz Hartl[2] · Volker Mohler[2] · Frank Ordon[1]

Abstract

Key message **The Bavarian MAGIC Wheat population, comprising 394 F6:8 recombinant inbred lines was pheno-typed for Puccinia triticina resistance in multi-years' field trials at three locations and in a controlled environment seedling test. Simple intervall mapping revealed 19 QTL, corresponding to 11 distinct chromosomal regions.**

Abstract The biotrophic rust fungus *Puccinia triticina* is one of the most important wheat pathogens with the potential to cause yield losses up to 70%. Growing resistant cultivars is the most cost-effective and environmentally friendly way to encounter this problem. The emergence of leaf rust races being virulent against common resistance genes increases the demand for wheat varieties with novel resistances. In the past decade, the use of complex experimental populations, like multiparent advanced generation intercross (MAGIC) populations, has risen and offers great advantages for mapping resistances. The genetic diversity of multiple parents, which has been recombined over several generations, leads to a broad phenotypic diversity, suitable for high-resolution mapping of quantitative traits. In this study, interval mapping was performed to map quantitative trait loci (QTL) for leaf rust resistance in the Bavarian MAGIC Wheat population, comprising 394 $F_{6:8}$ recombinant inbred lines (RILs). Phenotypic evaluation of the RILs for adult plant resistance was carried out in field trials at three locations and two years, as well as in a controlled-environment seedling inoculation test. In total, interval mapping revealed 19 QTL, which corresponded to 11 distinct chromosomal regions controlling leaf rust resistance. Six of these regions may represent putative new QTL. Due to the elite parental material, RILs identified to be resistant to leaf rust can be easily introduced in breeding programs.

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Introduction

With approximately 219 million hectares worldwide and 30% of global major cereal crop production in 2017, wheat (*Triticum* spp.) belongs to the most important crops for human nutrition (Braun et al. 2010; FAO 2019). Leaf rust, caused by the obligate biotrophic fungus *Puccinia triticina* Eriks., is nowadays the most destructive and prevalent rust pathogen in wheat (Kolmer 2005). Due to its adaptation to a wide range of different environments, leaf rust occurs in many wheat-producing areas of the temperate zone, causing yield losses up to 70% (Aktar-Uz-Zaman et al. 2017; Herrera-Foessel et al. 2006; Marasas et al. 2004). Although the application of fungicides helps to avoid yield losses, the deployment of resistant cultivars is the most effective, economic, and environmentally friendly approach to manage this disease. For wheat leaf rust, both qualitative and quantitative resistances are known. Seedling/qualitative resistance is monogenically inherited and only effective

against a subset of races. Thus, it mainly follows the gene-for-gene concept, in which resistance depends on a specific genetic interaction between host-resistance genes and avirulence genes of the pathogen (Flor 1956, 1971). These major genes confer vertical resistance and tend to be expressed from seedling to adult plant stages. Genotypes carrying such resistances show a hypersensitive response or programmed cell death (Bolton et al. 2008). In contrast, quantitative resistance is based on minor genes encoding various resistance responses, which are not restricted to specific pathogen races. Quantitative resistances are effective at later growth stages and are therefore referred to as field resistance or adult plant resistance (APR, Krattinger and Keller 2016). To date, more than 80 resistance genes to leaf rust (*Lr* genes) have been identified in bread wheat, durum wheat, and diploid wheat species (Gill et al. 2019). While most of them show race-specific resistance at the seedling stage, genes like *Lr12*, *Lr13*, *Lr22a/b*, *Lr34*, *Lr35*, *Lr37*, *Lr46*, *Lr67*, *Lr68*, and *Lr77* confer resistance at the adult plant stage (Dakouri et al. 2013; McIntosh et al. 2013, 2017).

The identification of such resistance genes as well as of quantitative trait loci (QTL) has been mainly based on biparental crosses (Huang et al. 2012). The weakness of such populations is the narrow genetic variation and the fact that genetic recombination is limited, which leads to a lower map resolution (Bandillo et al. 2013). Nowadays, high-throughput marker systems are available and genetic marker information is no longer limiting (Bayer et al. 2017; Chen et al. 2014; Cui et al. 2017; He et al. 2014; Mammadov et al. 2012), but the genetic variation present in respective populations (Asimit and Zeggini 2010; Gibson 2012). Thus, complex experimental populations such as nested association mapping (NAM, Yu et al. 2008) and multiparent advanced generation intercross (MAGIC) populations have been developed to detect QTL with a better reliability (Cavanagh et al. 2008). First multiparental intermated populations were exploited in mice (Churchill et al. 2004) and *Drosophila melanogaster* (King et al. 2012). In plants, MAGIC populations were first developed and described in studies regarding *Arabidopsis thaliana* (Cavanagh et al. 2008; Kover et al. 2009). These experimental designs involved multiple intercrosses of inbred founders for several generations to combine the genetic variation of all parental lines in the resulting progeny (Huang et al. 2012). MAGIC populations have been widely used to conduct QTL mapping in several crop species, such as rice (Bandillo et al. 2013), maize (Dell'Acqua et al. 2015), tomato (Pascual et al. 2015), faba bean (Sallam and Martsch 2015), sorghum (Ongom and Ejeta 2018), barley (Sannemann et al. 2015), and wheat (Gardner et al. 2016; Huang et al. 2012; Mackay et al. 2014; Milner et al. 2016; Sannemann et al. 2018). There are two clear advantages of using multiparental populations. First, based on the choice of founders, more traits of interest from each founder can

be analyzed. Second, due to the higher genetic variability and recombination rate, QTL detection can be performed with increased precision and resolution (Bandillo et al. 2013; Cavanagh et al. 2008).

The Bavarian MAGIC Wheat population (BMWpop) is one of only two German MAGIC wheat populations, which are mainly based on adapted German elite cultivars. It captures 71.7% of the allelic diversity available in the German wheat breeding gene pool (Stadlmeier et al. 2018). These populations provide the potential to carry out genetic studies of important economical traits, such as plant height and resistance to powdery mildew (Sannemann et al. 2018; Stadlmeier et al. 2018). In addition, Stadlmeier et al. (2019) detected six, seven and nine QTL for resistance to important fungal pathogens, i.e., *Blumeria graminis*, *Zymoseptoria tritici*, and *Pyrenophora tritici-repentis*, respectively. The objectives of the current study were to (1) phenotype the BMW population for quantitative and qualitative leaf rust resistance in multi-environment field trials and an extensive seedling test and (2) genetically map QTL in order to develop closely linked molecular markers suitable for marker-assisted selection (MAS).

Material and methods

Plant material

The study is based on the multiparental BMW population comprising elite wheat cultivars (Stadlmeier et al. 2018). It consists of 394 diverse $F_{6:8}$ recombinant inbred lines (RILs), which were derived from a simplified eight founder MAGIC mating design with additional eight-way intercrosses. The founders 'Event', 'BAYB4535′, 'Potenzial', 'Bussard', 'Firl3565′, 'Format', 'Julius' and 'Ambition' originated from German and Danish wheat breeders and were selected on the criteria of (1) variation for agronomic, quality and disease resistance traits, (2) originating from different breeding programs, and (3) being important cultivars in the respective baking quality group. More detailed information about the development and the genetics of the BMW population is provided by Stadlmeier et al. (2018).

Phenotypic assessment of leaf rust resistance in field

Five field trials were performed, each using a randomized incomplete block design with two replications at three locations in Germany: Quedlinburg (QLB, 51° 46′ 21.45″ N 11° 8′ 34.8″ E) in Saxony-Anhalt, Soellingen (SOE, 52° 5′ 45.506″ N 10° 55′ 41.711″ E) and Lenglern (LEN, 51° 35′ 47.53″ N 9° 51′ 39.118″ E) in Lower Saxony. The 394 RILs, the eight founders, and the susceptible standard 'Schamane'

were evaluated in double rows under natural disease epidemics in SOE (2017 and 2018) and LEN (2018). In QLB entries were sown 2016/2017 and 2017/2018 in double rows of 1 m length with 30 plants per row and spacing of 0.2 m between rows. Additional infection stripes of susceptible varieties were arranged in regular intervals of every third plot. Growth regulator Medax® Top (BASF Agricultural Solutions, Germany, 1 L ha^{-1}) was applied twice (BBCH31, BBCH37) to reduce plant height and lodging. No selective fungicides were used. To ensure uniform infestation, the infection stripes were artificially inoculated at the beginning of flowering using the highly virulent *Puccinia triticina* isolate 77WxR (Tab. S1). For this, a spore suspension of 10 mg uredospores in 100 ml Isopar M (ExxonMobil Chemical Company, USA) was applied in a total amount of 10 ml suspension per m^2, using a hand-held spinning disc sprayer (Bromyard, U.K.). Phenotyping of the trials was carried out by scoring the average percentage of infected leaf area of the second and third youngest leaves in the two rows at two (SOE17, SOE18, LEN18), three (QLB18), and four (QLB17) subsequent dates according to Moll et al. (2010), starting at the time of clearly visible disease symptoms on the infection stripe or the susceptible standard, respectively. A time period of 1 to 2 weeks was chosen between the scorings.

Phenotypic assessment of leaf rust resistance in seedlings

All RILs, the parental lines, and the susceptible standard 'Borenos' were evaluated for resistance at seedling stage in a detached leaf assay (Douchkov et al. 2012). Seedlings were grown in 77-cell trays with mixed potting soil (Gebr. Patzer GmbH Co KG, Sinntal, Germany) using a randomized complete block design with five replications. Water agar (7 g L^{-1}) containing 45 mg L^{-1} benzimidazole (Sigma-Aldrich Chemie GmbH, Taufkirchen, Germany), used to delay senescence of leaf segments, was dispensed in 4 × 10 mL aliquots into nonsterile four-well polystyrene plates (8 × 12 x 1 cm, Greiner Bio-One GmbH, Frickenhausen, Germany). Ten days after sowing, when the second leaf was developed, 2.5-cm sections were cut from the middle of the primary leaves and placed into the plates, keeping the randomization. White polytetrafluoroethylene frames (eMachineShop, Mahwah, USA) were used to fix the leaves. Inoculation was performed by an infection tower with three seconds swirling duration and three minutes of settling time (Melching 1967). Due to space restrictions, plates were divided into two infection groups per replication. Each group was inoculated with leaf rust isolate 77WxR using a mixture of 30 mg uredospores and white clay (1:1 w/w, VWR International GmbH, Darmstadt, Germany) after application of a 0.01% Tween 20 (Sigma-Aldrich) solution

to support adhesion. For 24 h, the plates were covered by wet cotton paper to support spore germination in the dark and at high humidity. Inoculated leaf segments were subsequently incubated in greenhouse at night/day temperatures of 16 °C/18 °C with additional lighting (16 h/8 h day/night) for ten days. Quantitative scoring was conducted using a high-throughput phenotyping platform (Douchkov et al. 2012). Digital images with a resolution of 20 Megapixel and four wavelengths between 315 and 750 nm (UV, blue, green, and red) were taken automatically from every plate. Subsequently, the leaf area was calculated and compared to the area of uredospore pustules for analyzing the percentage of infected leaf area (Pi) using the software HawkSpex® (Fraunhofer IFF, Magdeburg, Germany). Additionally, all entries were visually evaluated for infection type (IT) using a 0–4 scale (McIntosh et al. 1995). To generate metric data, original IT data were converted to a 0 – 10 linear disease scale, modified according to Zhang et al. (2014) as follows: 0, 0;, − 1,1, + 1, − 2, 2, 2 +, − 3, 3, + 3 were coded as 0, 0.5, 1, 2, 3, 4, 5, 6, 7, 8 and 9, respectively. IT − 4 and 4 were coded as 10 and in case of special annotation code "C" for chlorosis, 0.5 was added to the linear scale.

Data analysis

The multiple scorings of the percentage of infected leaf area in field trials were taken to calculate the area under the disease progress curve (AUDPC) and the average ordinate (AO, Moll et al. 1996) for each RIL using the following equations:

$$AUDPC = \sum_{i=1}^{N_{i-1}} \frac{(y_i + y_{i+1})}{2} * (t_{i+1} - t_i) \quad \text{and} \quad AO = \frac{AUDPC}{T}$$

where y_i is the disease level at the ith observation, t_i is the time at the ith observation, N is the total number of observations and T is the total observation time from the first to the last scoring date in days. Out of percentage of infected leaf area, AUDPC, and AO, only AO values were used for subsequent statistical analysis. Different year–location combinations of all trials were referred to as "environment".

Analyses of all phenotypic data were carried out using *proc mixed* of the software package SAS 9.4 (SAS Institute Inc., NY, USA). In order to apply a mixed linear model, a log$_{10}$ data transformation of the AO, IT, and Pi values was performed. The factors genotype, environment, and the genotype × environment interaction of field data were set as fix effects, while the design effects of replication and block were set as random. To obtain variance components for calculation of the broad sense heritability, all model parameters were set as random. Heritability was estimated on a progeny mean basis according to Hallauer et al. (2010).

For analyzing IT and Pi scores from seedling test the model:

$$y_{ijk} = \mu + g_i + r_j + l_k(r_j) + e_{ijk}$$

was used, where y_{ijk} is the trait observation, μ is the overall mean, g_i is the fixed effect of the genotype, r_j is the fixed effect of the replication, l_k is the random effect of the infection group nested in the replication and e_{ijk} is the random residual error. Variance components were obtained by fitting the genotype as random to calculate the repeatability as the ratio of the genotypic variance and the sum of the genotypic and the residual error variance divided by the number of replications. For each trait, least-square means (lsmeans) were calculated and used for subsequent QTL analysis.

QTL mapping

The BMW population and the parental lines were genotyped using the 15 K + 5 K Infinium® iSelect® array containing 17,267 single nucleotide polymorphism (SNP) markers (TraitGenetics, Germany). The preparation of genotypic data and the construction of the linkage map used for QTL mapping were described in detail by Stadlmeier et al. (2018). QTL mapping was performed using the R (×32 3.2.5) package mpMap V2.0.2 (Huang and George 2011; R Core Team 2017). To conduct simple interval mapping (SIM), founder probabilities were calculated using the function 'mpprob'. These give information about the probability of each locus that the observed genotype was inherited from one of the eight founders and are based on multipoint haplotype probabilities (Broman et al. 2003). To determine the parental origin of an allele, the threshold was set to 0.7. For SIM, a genome-wide significant threshold of α < 0.05 was calculated for each trait. The thresholds were obtained from permutation of phenotypic data with 1000 simulation runs (Churchill and Doerge 1994). QTL detection was performed using the function 'mpIM', implemented in the mpMap package (Huang and George 2011). Phenotypic variance explained by individual QTL and additive QTL effects were estimated separately using the categorical allele information of the founders. QTL support intervals were determined using the function 'supportinterval' of the mpMap package. A QTL support interval was defined as the map interval surrounding a QTL peak at a $-\log_{10}(p)$ drop of one unit (Huang and George 2011).

In order to compare QTL identified in the present study with previously described QTL, overlapping QTL based on the support interval was merged together. Databases of the Triticeae Toolbox (https://triticeaetoolbox.org/wheat/genotyping/marker_selection.php), GrainGenes (https://wheat.pw.usda.gov/GG3/), as well as CerealsDB (https://www.cerealsdb.uk.net/cerealgenomics/CerealsDB/axiom_download.php) were used to obtain marker information. Physical positions were received by nucleotide BLAST (BLAST-n) of the marker sequences against the reference sequence

RefSeq v1.0 using the database of 10 + Genome Project (https://webblast.ipk-gatersleben.de/wheat_ten_genomes/). BLAST hits were considered significant if the percent identity was greater than 95%, and only the best hit was taken if multiple BLAST hits were detected (Gao et al. 2016). The start and end positions of peak marker sequences preceded by the chromosome name were taken to the URGI database to obtain functional gene annotations available from IWGSC (https://wheat-urgi.versailles.inra.fr/Seq-Repository/Annotations). Sequences of the closest related species, *Triticum urartu* (A-genome donor) and *Aegilops tauschii* (D-genome donor), were considered for the detection of orthologous genes.

Results

Phenotypic assessment

Leaf rust severity of field trials clearly varied between years and location, displaying in QLB 2017, SOE 2018, and LEN 2018 the lowest infestations of leaf rust (Fig. S1). Pearson correlation coefficient between the different environments ranged from 0.26 to 0.74 ($P < 0.001$). Nevertheless, after mixed model adjustment, a broad sense heritability (h^2) of 0.83 was estimated (Table 1). The mean phenotypic distribution of AOs was slightly right-skewed and indicated a broad variability within the population (Fig. 1a), ranging between 0.2 and 34.8% (mean 13.5%) leaf area diseased. However, single maximal AO scores up to 63.8% were observed within the population (Table 1). The average performance of parental lines was evenly distributed, resulting in a nonsignificant difference ($p < 0.05$) from the progeny mean. Founders 'BAYP4535′ and 'Bussard' were identified as the most resistant (4.5%) and most susceptible (22.9%) parental line to leaf rust, respectively. The analysis of variance showed significant differences concerning genotype, environment, and the interaction between genotype and environment (Table 2).

Scoring qualitative resistance in seedling test was performed twice—using an image analysis software to obtain the Pi and visually by assessing the IT (1–10). For both traits, phenotypic data revealed a large variability (Fig. 1b and c). The average IT ranged from 0.1 to 9.2 (mean 3.8). For Pi, the disease severity was on average between 0 and 28.3% (mean 8.5%). Phenotypic distributions of IT and Pi were slightly bimodal, with 131 and 185 RILs showing IT values smaller 2 (few areas with restricted sporulation) and Pi values below 5%, respectively. Maximal scores of 10 (IT) and 57.7% (Pi) were observed (Table 1). The population means of IT and Pi were not significantly different from the means of parental lines. According to the results of field trials, 'BAYP4535' and 'Ambition' were the most
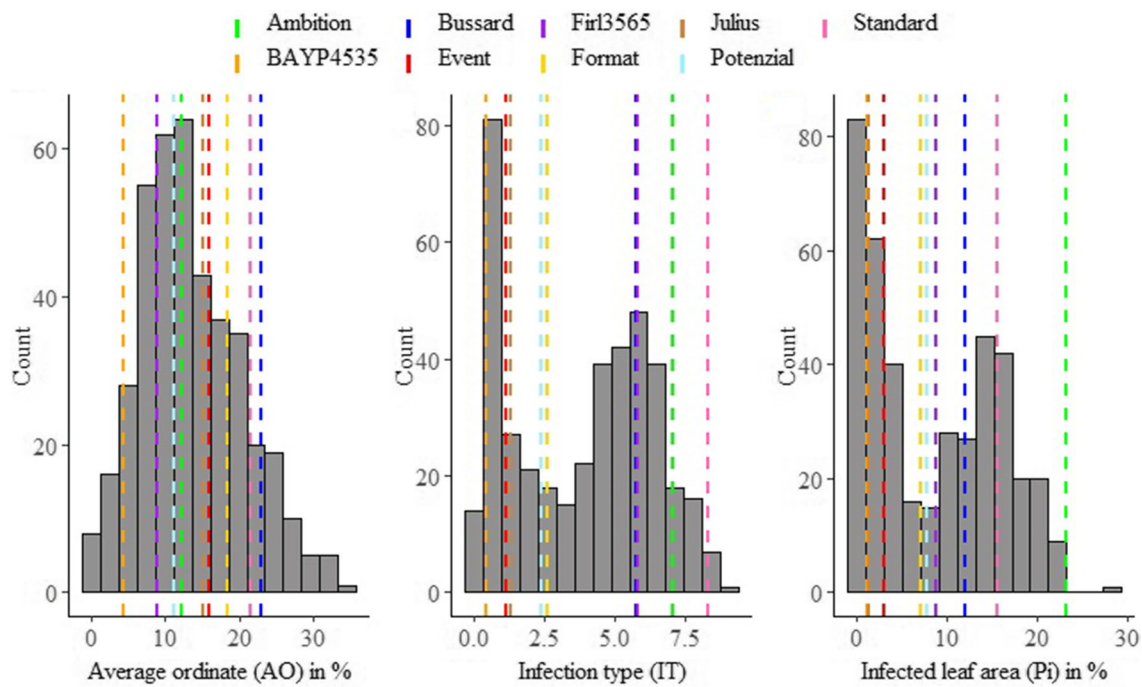
Fig. 1 Averaged phenotypic distribution of resistance to *Puccinia triticina* for field trials (A) and seedling test (B, C). Performance of each parental line is shown as vertical dashed line

Table 1 Descriptive statistics and heritability / repeatability for field trials (AO) and seedling test (IT and Pi)

Trait[a]	Mean founders	Mean population	Min[b]	Max[c]	$SE_{\pm}$[d]	CV[e]	h^2/rep
AO [%]	13.70	13.50	0	63.75	0.17	0.83	0.83[f]
IT [1–10]	3.32	3.84	0	10.00	0.06	0.96	0.93[g]
Pi [%]	8.06	8.47	0	57.73	0.18	0.72	0.91[g]

[a] Average ordinate (AO), infection type (IT), infected leaf area (Pi)

[b] Minimum

[c] Maximum

[d] Standard error

[e] Coefficient of variance

[f] Broad-sense heritability (h^2)

[g] Repeatability (*rep*)

resistant and susceptible founders, respectively, in the seedling inoculation test. Pearson correlation displayed a high correlation coefficient between both traits ($r = 0.91$; Fig S2 C). The qualitative traits IT and Pi and the quantitative scoring of AO showed weak correlations of $r = 0.27$ and $r = 0.24$ (Fig S2 A and B). For both traits, a significant genotype effect was observed, while for Pi also a significance of replication was found. Repeatability of both traits was high with $rep(IT) = 0.93$ and $rep(Pi) = 0.91$ (Table 1). From the parental lines, only 'BAYB4535 showed all stage resistance, whereas cv. 'Event', Format', 'Julius', 'Potenzial' only showed resistance at seedling and 'Firl3565' at adult plant stage, respectively. In total, 68 genotypes in the population expressed all stage resistance, 92 genotypes showed

resistance only at seedling stage and 44 genotypes were observed showing APR.

QTL mapping

Overall, SIM revealed 19 QTL located on chromosomes 1A, 4A, 4D, 5A, 6B, 7A, and 7D. Hence, five QTL were detected based on field data and seven QTL for seedling resistance, each for IT and Pi values (Table 3, Tab. S2).

The phenotypic variance (R^2) explained by the individual QTL detected in field trials ranged between 8 and 50%, with support intervals (SI) from 4 to 33 cM. The two strongest QTL, explaining 31% and 50% of R^2, were located on chromosome 4A with peak markers at 133 cM and 172 cM.

Table 2 Analysis of variance of $\log_{10}$-transformed data for leaf rust severity evaluated in field trials (AO) and seedling test (IT and Pi)

Trait[a]/factor	DF[b]	*F* value	*P* value
AO			
Genotype	402	18.98	< 0.0001
Environment	4	16.05	0.0049
Genotype × environment	1605	2.39	< 0.0001
IT			
Genotype	402	17.69	< 0.0001
Replication	4	0.94	0.5196
Pi			
Genotype	402	16.63	< 0.0001
Replication	4	6.66	0.0426

Significance level at $P < 0.05$

[a] Average ordinate (AO), infection type (IT), infected leaf area (Pi)

[b] Degrees of freedom

The largest allelic effects of these QTL were contributed by 'BAYP4535', reducing disease severity by 3.1% and 4.0%, respectively. Another QTL detected on chromosome 7D (at 18 cM) explained 28% of the phenotypic variance with 'BAYP4535′' as the most resistant founder, reducing infected leaf area by 3.2%. Remaining QTL on chromosomes 6B (at 22 cM) and 7A (at 368 cM) accounted for 8% and 7% of leaf rust variation. For these QTL, cv. 'Format' contributed the largest allelic effect reducing infected leaf area by 1.3% and 1.4%, respectively.

For IT, phenotypic variance explained by the seven QTL ranged from 1 to 28% with SIs ranging between 2 and 53 cM (Table 3). QTL on chromosomes 4A and 7D accounted for the highest R^2 i.e. 28% and 17% with peak markers at 170 cM and 22 cM. The largest allelic effect of both QTL was contributed by 'BAYP4535′', reducing disease severity by 2.6 and 1.8 scores, respectively. On chromosome 1A, one QTL was detected at 28 cM, explaining 11% of the phenotypic variance. A maximum effect of -1.0 score was detected for cv. 'Potenzial'. Furthermore, two QTL were detected on chromosome 5A with 8% (at 112 cM) and 7% (at 139 cM)

Table 3 QTL for resistance to *Puccinia triticina* in the BMW population detected in field trials and seedling tests

Trait	Chr.[a]	Pos.[cM][b]	SI [cM][c]	*P* value	R^{2d}	No. Env.[f]	Eff (A)[g]	Eff (B)[g]	Eff (C)[g]	Eff (D)[g]	Eff (E)[g]	Eff (F)[g]	Eff G)[g]	Eff (H)[g]
AO	4A	133	125–151	2.00E-22	0.31	4	− 0.17	− 3.12	+ 0.93	− 1.21	+ 0.90	+ 0.88	+ 0.86	+ 0,83
	4A	172	170–174	2.52E-58	0.50	4	+ 0.94	− 3.96	− 0.13	− 0.44	+ 2.04	− 0.18	+ 1.94	− 0,23
	6B	22	10–30	1.49E-05	0.08	1	+ 0.16	− 1.23	+ 1.70	+ 1.25	− 1.32	− 0.89	− 1.16	+ 1.47
	7A	368	346–379	1.52E-05	0.07	1	− 0.16	− 1.26	+ 1.10	− 1.39	− 1.42	+ 1.22	+ 0.94	+ 0.89
	7D	18	15–19	3.68E-32	0.28	4	na	− 3.16	na	na	+ 1.44	+ 0.94	+ 0.18	+ 0.58
IT	1A	28	0–34	1.55E-06	0.11		na	+ 0.76	− 0.27	na	− 0.75	− 0.98	− 0.67	+ 1.88
	4A	170	168–174	8.79E-23	0.28		0.00	− 2.57	− 1.32	+ 1.59	+ 1.15	− 1.10	+ 1.58	+ 1.12
	4D	69	59–86	2.57E-05	0.01		na	+ 0.01	na	na	+ 1.16	na	na	-1.98
	5A	112	102–152	1.56E-05	0.08		− 0.99	+ 0.21	+ 0.63	+ 0.06	− 2.06	+ 0.67	+ 0.87	+ 0.60
	5A	139	99–152	3.31E-05	0.05		− 0.26	+ 0.78	+ 1.29	− 1.29	− 1.44	+ 1.12	− 1.29	+ 1.07
	6B	249	248–250	2.18E-55	0.01		− 0.5	na	na	na	na	na	na	+ 0.5
	7D	22	15–30	6.14E-12	0.17		na	− 1.84	na	na	+ 0.61	+ 0.61	+ 0.02	+ 0.61
Pi	1A	26	0–34	8.11E-06	0.12		na	+ 0.60	+ 1.64	na	− 1.35	− 1.59	− 1.27	+ 1.98
	4A	171	168–174	1.11E-16	0.21		+ 0.33	− 4.14	− 1.33	+ 1.67	+ 1.47	− 0.88	+ 1.47	+ 1.42
	4D	72	59–86	4.27E-06	0.09		na	− 0.06	na	na	+ 1.6	na	na	− 1.54
	6B	249	247–250	1.76E-91	< .01		− 0.52	na	na	na	na	na	na	+ 0.53
	7A	65	54–87	7.02E-06	0.05		na	+ 1.05	+ 2.15	− 0.95	− 0.13	− 0.73	− 1.41	+ 0.03
	7A	99	94–111	6.12E-06	0.08		− 0.97	+ 1.38	+ 0.81	+ 0.08	+ 0.21	+ 0.23	− 2.38	+ 0.65
	7D	22	15–30	5.64E-09	0.14		na	− 2.78	na	na	+ 1.11	+ 1.11	− 0.27	+ 0.85

[a] Chromosomal position of QTL

[b] Position of peak marker based on Stadlmeier et al. (2018)

[c] Support interval

[d] Proportion of phenotypic variance explained by a single QTL

[f] Number of single environments in which QTL was detected

[g] Additive effects (±) of the founders Event (**A**), BAYP4535 (**B**), Ambition (**C**), Firl3565 (**D**), Format (**E**), Potenzial (**F**), Bussard (**G**) and Julius (**H**) relative to the population mean. Shown values are back-transformed to the original trait scale

Founder effects were reported as not available (na) if none of the RILs reached the probability threshold of 0.7

of the explained variance. SIs of these QTL ranged from 102 to 152 cM and from 99 to 152 cM, respectively. For both, 'Format' contributed the highest allelic effect (− 2.1 and − 1.4 scores). QTL located on chromosomes 4D (69 cM) and 6B (249 cM) explained only 1% of the phenotypic variance, each. By analyzing each environment separately, the two QTL on chromosomes 4A were also detected in LEN18, QLB17, QLB18 and SOE18, as well as LEN18, QLB18, SOE17 and SOE18, respectively. The QTL on chromosome 6B and 7D was detected in one (SOE18) and four (LEN18, QLB17, QLB18, SOE18) environments.

SIM of Pi values also revealed seven individual QTL with R^2 ranging from less than 1% to 21%. The support intervals varied between 2 and 34 cM. QTL regions on chromosomes 1A, 4A, 4D, 6B and 7D overlapped with QTL regions detected for IT (Table 3). Nevertheless, smaller R^2 of 21% (4A), 14% (7D) and < 1% (6B), as well as larger R^2 of 12% (1A) and 9% (4D) were calculated for individual QTL. The maximal reducing effect of the QTL on chromosomes 1A, 4A, 4D, and 6B ranged between 0.5% and 4.1%, while for 7D, only the founder 'BAYP4535′ showed a reducing allelic effect of 2.8%. Additionally, two QTL were detected on chromosome 7A at 65 cM and 99 cM, accounting for 5% and 8% of the phenotypic variance. SI ranged from 54 to 87 cM and from 94 to 111 cM, respectively. Founders 'Firl3565′ and 'Bussard' contributed the largest allelic effect, reducing the disease severity by 0.9% and 2.4%.

Based on support intervals of 19 QTL, detected in total for the different traits, 11 main QTL were identified (Fig. S3, Table 4). In silico annotations of peak markers revealed seven genes with known functions partly involved in resistance. Hence, marker *CAP8_c2448_355* on chromosome 1A referred to a DnaJ domain. A Protein kinase domain and a NB-ARC domain were identified for peak markers of *QLr.jki-4A.1* and *QLr.jki-4A.2* on chromosome 4A. Marker *AX-95126745* on chromosome 4D and *RAC875_c31670_389* on chromosome 5A referred to a cation/calcium exchanger 4 and ankyrin repeats, respectively. For peak markers of *QLr.jki-7A.1* and *QLr.jki-7A.1* on chromosome 7A, a pyridoxal-phosphate dependent enzyme and a sugar efflux transporter were annotated, respectively.

Discussion

Continuous evolution of leaf rust results in the emergence of new pathotypes virulent against single major resistance genes commonly present in cultivars. Many of these race specific *Lr* genes have been broken down in the past (Kolmer 2005; Serfling et al. 2013). Detection of effective leaf rust resistances is of essential importance to avoid rust epidemics. Therefore, experimental populations such as MAGIC populations provide powerful tools to discover, characterize,

and deploy QTL for complex traits including resistances (Cavanagh et al. 2008). Out of 80 designated *Lr* genes, it was reported, that only *Lr1, Lr3, Lr10, Lr13, Lr14a, Lr17b, Lr20, Lr24, Lr26, Lr34*, and *Lr37* were used individually or in combination in European varieties (Goyeau et al. 2006; Goyeau and Lannou 2011; Serfling et al. 2013). The BMW population emerged from crosses of eight elite parental lines originating from Germany and Denmark. Nevertheless, Stadlmeier et al. (2018) were able to show the potential of the BMW population to detect new QTL for resistance to powdery mildew, septoria tritici blotch, as well as tan spot, and in general the usefulness for further gene mapping studies (Stadlmeier et al. 2018, 2019).

In this study, phenotyping of 394 RILs from the BMW population resulted in a broad variability of resistance to *Puccinia triticina*. Despite an average correlation coefficient of 0.54 between the disease severities in five environments, a broad sense heritability of 0.83 was calculated which is in the range of previously published studies (Bemister et al. 2019; Gao et al. 2016; Zhang et al. 2017, 2019). This may hint to a quantitative inheritance due to QTL involved in slow rusting loci, which are characterized by relatively high heritabilities (Kolmer 1996). Phenotypic distribution for field trials was slightly right-skewed, while almost a bimodal distribution was observed for both IT and Pi values in seedling test. This may give hint that mostly horizontal (quantitative) or vertical (qualitative) resistances were scored, respectively. Calculation of correlation between field data and seedling test results showed *r* values of 0.27 (IT) and 0.24 (Pi), which are in accordance with correlations reported by Gao et al. (2016). Different virulence/avirulence patterns of leaf rust races may be an explanation for these low correlations (Gao et al. 2016). While a single highly aggressive race, with many virulence genes was used for artificial inoculation for seedling tests and field trials in QLB, field trials in SOE and LEN were conducted under natural infection pressure.

Overall, simple interval mapping detected 19 QTL, which corresponded to 11 distinct chromosomal regions (Table 4, Fig. S3). QTLs identified using the LSmeans over all environments were also identified by analyzing the single environments separately. Out of the 11 distinct chromosomal regions three QTL were detected at the adult plant stage. Six QTL conferred seedling resistance and two were active in both growth stages, indicating the presence of effective all-stage leaf rust resistance genes. In total, the regions were located on wheat chromosomes 1A, 4A, 4D, 5A, 6B, 7A and 7D. Peak markers of QTL could be partially annotated to genes, known to be involved in quantitative resistances to leaf rust, e.g. sugar efflux transporters, DnaJ domain belonging to heat shock protein family (Bekh-Ochir et al. 2013), a protein kinase domain, a NB-ARC domain and a cation/calcium exchanger. Such genes

Table 4 Main QTL for resistance to *Puccinia triticina* merged over all evaluated traits

QTL	Chr.[a]	Peak marker	Pos.[cM][b]	SI [cM][c]	Pos.RefSeq [bp][d] Start	End	Potential origin	Adjacent *T.aestivum* gene	Orthologous gene	Identity	Functional annotation
QLr.jki-1A.1	1A	CAP8_c2448_355	27.63	0–34	10069841	10069932	Potenzial	TraesCS1A01G020600	TRIUR3 04361[e]	93.63	DnaJ domain
		RAC875_c57939_78	26.12		11571831	11571931		TraesCS1A01G023400	F775 01617[f]	94.93	
QLr.jki-4A.1	4A	Kukri_rep_c109167_89	133.99	125–151	634737614	634737686	BAYP4535	TraesCS4A01G361100	TRIUR3 34719[e]	99.78	Protein kinase domain
									F775 31833[f]	99.28	
QLr.jki-4A.2	4A	BobWhite_c47168_598	171.04	168–174	726214891	726214991	BAYP4535	TraesCS4A01G461700			NB-ARC domain
		Excalibur_c46904_84	169.52		737340474	737340573		TraesCS4A01G481400	TRIUR3 03302[e]	96.59	
									F775 10262[f]	96.25	
QLr.jki-4D.1	4D	BS00023112_51	69.43	58–86	455763978	455764078	Julius	TraesCS4D01G285000	F775 08229[f]	100.00	Cation/calcium exchanger 4
		AX-95126745	71.96		464988433	464988533		TraesCS4D01G294600	F775 05351[f]	99.69	
QLr.jki-5A.1	5A	IAAV2363	111.77	98–152	481901324	481901524	Format	TraesCS5A01G271500	F775 15669[f]	96.01	Ankyrin repeats
		RAC875_c31670_389	138.69		514094550	514094650		TraesCS5A01G305200	F775 21555[f]	98.40	
QLr.jki-6B.1	6B	AX-94557244	21.83	10–30	25914587	25914687	Format	TraesCS6B01G041900			
QLr.jki-6B.2	6B	RAC875_c57692_88	249.34	247–250	712673112	712673182	Event	TraesCS6B01G456500			
QLr.jki-7A.1	7A	BS00011330_51	64.66	54–87	63112744	63112844	Firl3565	TraesCS7A01G102800	TRIUR3 02989[e]	99.72	Pyridoxal-phosphate dependent enzyme
									F775 27910[f]	95.24	
QLr.jki-7A.2	7A	wsnp_Ku_c26530_36497050	98.82	93–111	84772316	84772460	Bussard		TRIUR3 06012[e]	94.36	
QLr.jki-7A.3	7A	BS00011622_51	368.31	346–379	712309001	712309084	Format	TraesCS7A01G533900	TRIUR3 33918[e]	88.99	Sugar efflux transporter for intercellular exchange
									F775 06947[f]	87.19	
QLr.jki-7D.1	7D	AX-94930280	18.13	15–30	16119641	16119741	BAYP4535	TraesCS7D01G030600			
		IACX11794	21.65		12470235	12470390		TraesCS7D01G026100	F775 15174[f]	97.59	

[a] Chromosomal position of QTL

[b] Position of peak marker based on Stadlmeier et al. (2018)

[c] Support interval

[d] Position of peak marker at the reference genome RefSeq v1.0

[e] *Triticum urartu*

[f] *Aegilops tauschii*

show an increased expression during defense reactions in wheat-leaf rust (Sharma et al. 2018) and wheat-stripe rust interactions (Wang et al. 2020) and as response to environmental stresses.

In this study, *QLr.jki-1A.1* on chromosome 1A is based on the evaluation of IT and Pi in seedling tests and is physically located in a region between 1.3 Mbp and 12.5 Mbp (Table 5). Pinto da Silva et al. (2018) reviewed 11 QTL

Table 5 Comparison of physical positions of the QTL identified in the present study (bold) with those reported previously. Physical positions based on comparison of marker sequence data to the wheat reference genome (RefSeq1.0)

QTL	Marker interval	Physical position [Mbp]	Genetic material	References
QLr.jki-1A.1	IAAV3919–Tdurum_contig42479_3800	1.3–12.5	BMW population (RIL[a])	*Lr10*?
QLr.ccsu-1A.1	Xbarc263–Xcdo426	11.8–na[b]	Opata85 × W-7984 (RIL)	Kumar et al. (2013)
QLr.cau-1AS	gpw2246	7.7	Luke × AQ24788-83 (RIL)	Du et al. (2015)
MTA	IWA3182–IWA7191	7.1–13.7	Spring wheat collection	Elbasyoni et al. (2017)
Lr10		12.6		Feuillet et al. (2003)
QLr.jki-4A.1	AX-95253498–TA006348.0950	618.6–649.9	BMW population (RIL)	
MTA	IWA2816	641.5	Hexaploid Wheat Landraces	Kertho et al. (2015)
QLr.jki-4A.2	Tdurum_contig75819_1220–Excalibur_c33542_113	712.9–na	BMW Population (RIL)	
4A_t2	BobWhite_c47168_289	726.2	Elite spring wheat lines	Gao et al. (2016)
QLr.hebau-4AL	BobWhite_c15697_675–Excalibur_c2827_580	598.7–726.4	Zhou8425B × Chinese Spring (RIL)	Zhang et al. (2017)
QLr.jki-4D.1	AX-94793903–AX-94838884	130.9–479.7	BMW population (RIL)	Novel?
QLr.fcu-4DL	Xgdm61–Xcfa2173	na	TA4152-60 × ND495 (DH[c])	Chu et al. (2009)
QLr.hebau-4DL	AX-110476142–AX-111092299	381.2–428.6	Pingyuan50 × Mingxian169	Zhang et al. (2019)
QLr.sfrs-4DL	Xglk302b–Xpsr1101a	na	Forno × Oberkulmer (RIL)	Messmer et al. (2000)
Lr67	Xgwm165–Xgwm192	412.7	RL6077 × Avocet (RIL)	Herrera-Foessel et al. (2011)
QLr.jki-5A.1	AX-94732470–wsnp_Ex_c49211_53875600	444.6–na	BMW population	Novel?
QLr.cim-5AC	wPt-3187–wPt-7769	Na–464.7	Avocet-YrA × Kenya Kongoni (RIL)	Calvo-Salazar et al. (2015)
QLr.jki-6B.1	AX-94739546–TA003005.0339	19.3–34.3	BMW population	Novel?
QLr.caas-6BS.1	Xcfd13–Xwmc487	34.2–36.5	Bainong64 × Jingshuang16 (DH)	Ren et al. (2012)
QLr.wpt-6BS.2	wPt2175	na[b]	Winter wheat accessions	Gerard et al. (2018)
QLr.jki-6B.2	wsnp_Ex_c54772_57528275–Excalibur_c29748_954	710.1–719.7	BMW population	*Lr3*?
QLr.cim-6BL	277,143–1,234,305	714.3–na	Bairds × Atred#1 (RIL)	Lan et al. (2017)
6B_4	BobWhite_c43263_180–BS00011795_51	718.9–720.6	Elite spring wheat lines	Gao et al. (2016)
QLr.jki-7A.1	BobWhite_rep_c58252_112–wsnp_BF473884A_Ta_1_3	54.9–71.1	BMW population	Novel
QLr.jki-7A.2	RAC875_c75528_355–BS00024786_51	79.6–na	BMW population	Novel?
QLr.stars-7AS1	wsnp_Ex_c41150_48040078	78.4	Winter wheat accessions	Li et al. (2016)
MTA	IWA7192	81.1	Spring wheat collection	Elbasyoni et al. (2017)
Lr47		115		Helguera et al. (2000)
QLr.jki-7A.3	Tdurum_contig29240_206–wsnp_CAP11_c298_250917	702.4–724.1	BMW Population	*Lr20*?
MTA	IWA4175	717.1	Spring wheat accessions	Turner et al. 2017
QLr.jki-7D.1	TA016282.1180–AX-94883448	na–29.4	BMW Population	Novel
Lr34		47.4–51		Krattinger et al. (2009)

[a] Recombinant inbred line population

[b] marker information was not available or position could not be identified in the RefSeq v1.0

[c] Doubled haploid population

described in hexaploid wheat located on chromosome 1A. Based on available physical marker positions, *QLr.ccsu-1A.1* and *QLr.cau-1AS* identified in two different studies, were found to correspond to the region of *QLr.jki-1A.1* (Du et al. 2015; Kumar et al. 2013). While *QLr.ccsu-1A.1* is only 1.7 Mbp and 0.2 Mbp apart from our peak markers, the distance of the linked marker to *QLr.cau-1AS* is 2.4 Mbp and 3.9 Mbp, respectively (Tables 4, 5). Additionally, Elbasyoni et al. (2017) detected several marker-trait associations (MTAs) covering a region from 7.2 Mbp to 13.7 Mbp, which includes the region of *QLr.jki-1A.1*. Furthermore, the resistance gene *Lr10*, which is completely sequenced, is mapped at 12.6 Mbp, i.e. 2.5 Mbp and 1 Mbp apart from our peak marker (Table 4; Feuillet et al. 1997, 2003). Thus, and due to the fact that *Lr10*, *Lr1*, *Lr3a* and *Lr20* are the most prevalent genes used worldwide, *Lr10* is a promising candidate for the QTL aforementioned (Dakouri et al. 2013).

On chromosome 4A, two regions harboring leaf rust resistance were identified in this study (*QLr.jki-4A.1*, *QLr.jki-4A.2*, Table 4). To date, there are two *Lr* genes, *Lr28* originating from *Ae. speltoides* and *Lr30* from *T. aestivum*, and two QTL reported on chromosome 4A (Dyck and Kerber 1971; McIntosh et al. 2013; Pinto da Silva et al. 2018). Kertho et al. (2015) found one MTA at 641.5 Mbp, using the leaf rust race MCDL. Therefore, the marker is physically located within the region of *QLr.jki-4A.1*, but 6.8 Mbp apart from our peak marker. Due to the specific virulence pattern of the MCDL race, which is avirulent to *Lr30*, the MCDL-MTA might identify this *Lr* gene. However, to our knowledge, no mapping information for *Lr30* is available to allow a more precise comparison between *Lr30*, the MCDL-MTA and *QLr.jki-4A.1* detected in this study. Another significant MTA (*4A_t2*, Gao et al. 2016) was detected in the region of *QLr.jki-4A.2*, only 309 bp apart from the peak marker for this QTL (Table 4). *4A_t2* was mapped approximately at the position of the marker linked to *Lr28* (Bipinraj et al. 2011). This may be a hint that *QLr.jki-4A.2* also corresponds to *Lr28*, but further analyses have to be conducted. Furthermore, Zhang et al. (2017) reported a minor QTL for APR in Chinese Spring (*QLr.hebau-4AL*), which is physically located between 598.7 Mbp and 726.4 Mbp. This region includes both QTL on chromosome 4A detected in this study (Table 5).

In total, nine QTLs were detected on chromosome 4D so far, including the resistance gene *Lr67/Yr46/Sr55* (Herrera-Foessel et al. 2011; McIntosh et al. 2013; Pinto da Silva et al. 2018). In this study, *QLr.jki-4D.1* was detected for both IT and Pi in the seedling tests and mapped at the distal end of chromosome 4DL. Physically, it is located in a large interval from 130.9 Mbp to 479.7 Mbp (Table 5) with peak markers at 455.8 Mbp and 465 Mbp, respectively (Table 4). Chu et al. (2009) located a QTL (*QLr.fcu-4DL*) in double-haploid population 'TA4152-60×ND495′, mapped at a similar

position as *Lr67*, around 412.7 Mbp (Herrera-Foessel et al. 2011; Zhang et al. 2019). Another QTL on chromosome 4DL (*QLr.hebau-4D*) was located between 381.2 Mbp and 428.6 Mbp (Zhang et al. 2019). Considering the physical distances to our peak marker, it appears that *QLr.jki-4D.1* is independent from *QLr.fcu-4DL*, *QLr.hebau-4D*, and *Lr67* (Table 5). A higher similarity may exist with another QTL (*QLr.sfrs-4DL*) detected by Messmer et al. (2000). This QTL resulted in an APR and was mapped in the Swiss RIL population 'Forno×Oberkulmer' also at the distal end of chromosome 4DL. Since *QLr.jki-4D.1* has only been detected at the seedling stage, *QLr.sfrs-4DL* also seems to be located in a different region and with the available data, it is not possible to further determine whether it corresponds to our regions.

On chromosome 5A one QTL (*QLr.jki-5A.1*) was detected in seedling tests for IT (Table 4). To our knowledge, on chromosome 5A there is no designated *Lr* gene and only two QTL (*QLr.cim-5AC*, *QLr.cimmyt-5A*) are known (Calvo-Salazar et al. 2015; Rosewarne et al. 2012). *QLr.cimmyt-5A* was mapped on the long arm of chromosome 5A, closely linked to *Vrn-A1* at 587.0 Mbp (Rosewarne et al. 2012). *QLr.cim-5AC* was located in the centromeric region of chromosome 5A and flanked by markers *wPt-7769* and *wPt-3187*, of which the latter is located at 464.7 Mbp (Table 5). When comparing the physical positions of these three QTL, it is more likely that *QLr.jki-5A.1* corresponds to *QLr.cim-5AC* or is a novel QTL.

On chromosome 6B, two QTL were identified (*QLr.jki-6B.1* and *QLr.jki-6B.2*) in the present study (Table 4). *QLr.jki-6B.1* was mapped on the short arm of chromosome 6B, at 19.3—34.3 Mbp (Table 5). Up to now, 5 QTL have been described on chromosome 6BS, but only *QLr.caas-6BS.1*, derived from the wheat cultivar Bainong 64, was physically localized in the region between 32 and 34 Mbp (Gerard et al. 2018; Kankwatsa et al. 2017; Ren et al. 2012). Gerard et al. (2018) stated that another QTL (*QLr.wpt-6BS.2*) is genetically located in the same region as *QLr.caas 6BS.1*, whereas *QLr.wpt-6BS.2* was mapped close to the centromere, a region clearly distinct from *QLr.jki-6B.1* (Table 5). Therefore, further studies are required to confirm whether our QTL is located closely to these known QTL. The second QTL *QLr.jki-6B.2* was mapped at the distal end of chromosome 6BL, within a small interval encompassing 247 cM to 250 cM (710 – 720 Mbp). Out of six QTL already detected on chromosome 6BL, two QTL (*QLr.cim-6BL* and *6B_4*) were also located at the distal end of chromosome 6BL (Chu et al. 2009; Gao et al. 2016; Lan et al. 2017; Rosewarne et al. 2012; William et al. 2006). The DArTseq markers *1234305* and *2277143* flank *QLr.cim-6BL* detected by Lan et al. (2017). Marker *2277143* was converted into a diagnostic KASP marker, which is located at 714.3 Mbp, i.e. 1.6 Mbp distal from our peak marker of *QLr.jki-6B.2* (Tables 4 , 5). The results of Lan et al. (2017) indicated uniqueness of *QLr.*

cim-6BL, showing no relationship to other QTL on chromosome 6BL, as well as to *Lr3a* co-segregating with *Xmwg798* (Sacco et al. 1998). However, the second known QTL *6B_4* was physically mapped between 718.9 Mbp and 720.6 Mbp, and appeared to be in high linkage disequilibrium with *Lr3* (Gao et al. 2016). Regarding the similar physical regions, *QLr.jki-6B.2* may correspond to *QLr.cim-6BL* and *6B_4*, but further research is needed to come to a closer understanding of the relationship between these QTL and *Lr3*.

On chromosome 7A, the major resistance genes *Lr20,* forming a disease-resistance gene cluster with *Pm1,* and *Lr47*, which was transferred from chromosome 7S of *Ae. speltoides* have been reported (Dubcovsky et al. 1998; Neu et al. 2002). Additionally, three QTL on chromosome 7AL and several MTAs were detected (Pinto da Silva et al. 2018). In the present study, three QTL (*QLr.jki-7A.1* to *QLr.jki-7A.3*) were identified on chromosome 7A. The first two QTL were detected for Pi in the seedling test and their support intervals were separated from each other by a map distance of 7.1 cM on chromosome 7AS. *QLr.jki-7A.1* was physically mapped between 54.9 Mbp and 71.1 Mbp (Table 5). To our knowledge, no QTL have been reported in this region. Hence, *QLr.jki-7A.1* might be a novel QTL. The second QTL (*QLr.jki-7A.2*) on chromosome 7AS was located between 93 and 111 cM. The peak marker was mapped at 84.8 Mbp (Table 4). To date, there are two MTAs from different studies detected in similar regions as *QLr.jki-7A.2* (Elbasyoni et al. 2017; Li et al. 2016). The first MTA (*QLr.stars-7AS1*), associated with marker *IWA3760* was mapped at 78.4 Mbp, hence, it appears that *QLr.stars-7AS1* does not correspond to *QLr.jki-7A.2*. The second MTA (*IWA7192*) was detected by Elbasyoni et al. (2017) at 81.1 Mbp, and might be correspondent to the resistance gene *Lr47*. When comparing the physical position of a diagnostic marker for *Lr47* (around 115 Mbp), both *IWA7192*, and *QLr.jki-7A.2* seem to be different from this *Lr* gene (Helguera et al. 2000). Thus, *QLr.jki-7A.2* is likely a novel locus involved in resistance to *P. triticina*.

The third QTL (*QLr.jki-7A.3*) determined in field trials during this study was mapped between 346 and 379 cM on chromosome 7AL. This translates to a large physical distance between 702.4 Mbp and 724.1 Mbp, with the peak marker at 712.3 Mbp (Tables 4, 5). Out of five known regions on chromosome 7AL involved in leaf rust resistance (Kankwatsa et al. 2017; Li et al. 2016; Lu et al. 2017; Tsilo et al. 2014), only the MTA detected by Turner et al. (2017) may be localized within the region of *QLr.jki-7A.3*. The associated marker *IWA4175* was mapped at 717.1 Mbp, which is 4.8 Mbp apart from our peak marker. However, after Bonferroni correction, the marker was no longer significant ($P < 0.1$). The *Lr* gene *Lr20* is

genetically located in the distal part of chromosome 7AL (Neu et al. 2002), which may correspond to *QLr.jki-7A.3*. Based on the available data, investigations with diagnostic markers need to be conducted to gain further insights.

Finally, one QTL was detected on chromosome 7DS, based on phenotypic data from field trials and seedling test (Table 4). To date, out of 21 QTL reported on chromosome 7D, 19 correspond to the resistance gene *Lr34*, which confers race non-specific, partial, and slow rusting resistance to leaf rust (Lagudah et al. 2009; Pinto da Silva et al. 2018). *Lr34* has been physically located at 47.4 Mbp (Krattinger et al. 2009). Thus, *QLr.jki-7D.1* identified in our study does not correspond to the resistance gene *Lr34* and the 19 QTL reported (Table 5). The remaining two QTL *QLr.cim-7DS* and *QLr.hebau-7DS* on chromosome 7DS, which were detected in the two RIL populations 'Avocet-YrA × Francolin#1' and 'Shanghai3/ Catbird × Naxos', respectively, were located in different chromosome region (Lan et al. 2014; Zhou et al. 2014). Hence, *QLr.jki-7D.1* seems to be a novel locus.

The objective of this study was to identify QTL for resistance to leaf rust, using the Bavarian MAGIC Wheat population. We identified 19 leaf rust resistance QTL that were confined to 11 distinct chromosomal regions. To date, more than 249 leaf rust resistance QTL and 200 MTAs were reported covering all 21 chromosomes of hexaploid wheat (Pinto da Silva et al. 2018). These regions were identified in several mapping populations using different genotyping methods. Because of the absence of information on physical positions for many of these QTL, it is difficult to unequivocally determine the identity of newly described QTL. In the present study, six putatively new QTL were identified on chromosomes 4D, 5A, 6B, 7A and 7D. SNP markers linked to these regions may be converted into KASP markers suitable for MAS in wheat breeding programs (Neelam et al. 2013; Rasheed et al. 2016). This will enable stacking of the detected resistance loci to breed new varieties with an improved resistance to leaf rust.

Compliance with ethical standards

Conflict of interest The authors declare that there are no conflicts of interest in the reported research.

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CHAPTER III | QUANTITATIVE TRAIT LOCI MAPPING OF ADULT PLANT AND SEEDLING RESISTANCE TO STRIPE RUST (*PUCCINIA STRIIFORMIS* WESTEND.) IN A MULTIPARENT ADVANCED GENERATION INTERCROSS WHEAT POPULATION

Sandra Rollar, Manuel Geyer, Lorenz Hartl, Volker Mohler, Frank Ordon, Albrecht Serfling

Quantitative Trait Loci Mapping of Adult Plant and Seedling Resistance to Stripe Rust (*Puccinia striiformis* Westend.) in a Multiparent Advanced Generation Intercross Wheat Population

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Stripe rust caused by the biotrophic fungus *Puccinia striiformis* Westend. is one of the most important diseases of wheat worldwide, causing high yield and quality losses. Growing resistant cultivars is the most efficient way to control stripe rust, both economically and ecologically. Known resistance genes are already present in numerous cultivars worldwide. However, their effectiveness is limited to certain races within a rust population and the emergence of stripe rust races being virulent against common resistance genes forces the demand for new sources of resistance. Multiparent advanced generation intercross (MAGIC) populations have proven to be a powerful tool to carry out genetic studies on economically important traits. In this study, interval mapping was performed to map quantitative trait loci (QTL) for stripe rust resistance in the Bavarian MAGIC wheat population, comprising 394 $F_{6:8}$ recombinant inbred lines (RILs). Phenotypic evaluation of the RILs was carried out for adult plant resistance in field trials at three locations across three years and for seedling resistance in a growth chamber. In total, 21 QTL for stripe rust resistance corresponding to 13 distinct chromosomal regions were detected, of which two may represent putatively new QTL located on wheat chromosomes 3D and 7D.

Keywords: stripe rust, *Yr* genes, MAGIC population, simple interval mapping, QTL

INTRODUCTION

The biotrophic fungus *Puccinia striiformis* Westend. f. sp. *tritici* Eriks. is the causal agent of stripe rust and is one of the most important foliar diseases of wheat, which accounted for 25% of global cereal crop production in 2018 (Food and Agriculture Organization of the United Nations (FAO), 2020). Particularly prevalent in the temperate and maritime wheat growing regions, stripe rust can cause yield losses up to 70% mainly by reducing photosynthesis and taking assimilates from the host plant (Chen, 2005; Jagger et al., 2011; Rosewarne et al., 2012). In agricultural production systems, the application of fungicides, as well as the growing of resistant cultivars are currently used to control stripe rust, of which the latter is the most

economically safe and environmentally friendly approach to avoid yield losses. To date, about 82 stripe rust resistance genes (*Yr* genes) have been unequivocally identified, but a lot more temporary designated genes and quantitative trait loci (QTL) have been reported and mapped across the whole wheat genome (McIntosh et al., 2019; Yang et al., 2019a). Of these, *Yr5*, *Yr7*, *Yr10*, *Yr15*, *Yr18*, *Yr36*, *Yr46*, and *YrSP* have already been cloned and characterized as intracellular nucleotide-binding leucine-rich-repeat receptors (*Yr5*, *Yr7*, and *YrSP*), putative kinase-pseudokinase protein (*Yr15*), transporters (*Yr18* and *Yr46*), or wheat kinase start 1 (*Yr36*) (Fu et al., 2009; Krattinger et al., 2009; Liu et al., 2014; Moore et al., 2015; Klymiuk et al., 2018; Marchal et al., 2018). In addition, resistance genes, such as *YrAS2388R* derived from *Aegilops tauschii* and YrU1 derived from *Triticum urartu* have recently been cloned, encoding a nucleotide oligomerization domain-like receptor (NLR) and a coiled-coil-NBS-leucine-rich repeat protein with N-terminal ankyrin-repeat and C-terminal WRKY domains, respectively (Zhang et al., 2019; Wang et al., 2020).

Mainly two different types of resistance are described based on criteria, such as inheritance, specificity, plant growth stage, and temperature (Chen, 2013; Liu et al., 2018). The so-called all-stage resistance is detected at the seedling stage and is therefore also referred to as seedling resistance. Nevertheless, seedling resistance is in general expressed throughout all growth stages, leading to resistance in the seedling stage as well as in adult plants. It is monogenetically inherited, qualitatively expressed, and the underlying major genes are only effective against a subset of races (Chen, 2005; Feng et al., 2018). Thus, it mainly follows the gene-for-gene concept, in which the resistance depends on a specific genetic interaction between the host-resistance genes and the avirulence genes of the pathogen (Flor, 1971). Effectors produced by the pathogen are recognized by nucleotide binding site-leucine rich repeat (NB-LRR) proteins, predominately encoded by corresponding plant resistance genes (Flor, 1956; Juliana et al., 2018). This results in an effector-triggered immunity that usually initiates a hypersensitive response leading to a localized programmed cell death preventing further colonization, e.g., in the case of *Yr5*, *Yr7*, *Yr10*, and *YrSP* (Heath, 2000; Jones and Dangl, 2006). The use of race-specific resistance in plants is common in wheat, leading to a breakdown of major resistance genes according to the so-called boom-and-bust cycles (McDonald and Linde, 2002a). To date, most race-specific resistance genes against stripe rust, e.g., *Yr10*, *Yr24*, and *Yr27* have been overcome by virulent races leading to the demand for more durable resistance (Kolmer, 2005; Hovmøller et al., 2017; Wang and Chen, 2017). Adult plant resistance (APR), effective at later growth stages, is quantitatively inherited and based on minor genes encoding various resistance responses, which are not restricted to specific pathogen races (Krattinger and Keller, 2016). Thus, APR does not follow the gene-for-gene interaction and is generally considered as durable. A special type of APR to stripe rust is the high-temperature adult plant (HTAP) resistance that is additionally affected by temperature (Chen, 2013). However, the mechanisms of such durable resistances include an increased latency period, reduced uredinia size, reduced infection frequency, and reduced spore production to inhibit fungal infestation (Rosewarne et al., 2013). To improve the general stripe rust resistance in commercial cultivars, more genes and useful genetic markers are needed for increasing the level and durability of resistance by combining HTAP resistance with seedling resistance.

In the context of detecting new resistance genes and QTL, molecular markers are no longer the limiting factors due to the availability of high-throughput marker systems (Mammadov et al., 2012; Chen et al., 2014; He et al., 2014; Bayer et al., 2017; Cui et al., 2017), but rather the genetic variation present in the respective experimental populations that merge genomes of diverse founders *via* designed crosses (Asimit and Zeggini, 2010; Gibson, 2012). Such experimental populations are traditionally derived from crosses of two contrasting parents. Thus, only two alleles at a given locus segregate in such bi-parental populations (Han et al., 2020). In contrast, the strategy of multiparent advanced generation intercross (MAGIC) populations is to interrogate multiple alleles to achieve increased recombination and mapping resolution (Cavanagh et al., 2008). Prior to developing such MAGIC populations, founder lines have to be selected based on genetic and/or phenotypic diversity. The development itself includes three steps: (1) Selected parents are crossed with each other to form a broad genetic base. (2) To increase recombination events, advanced intercrosses among the mixed lines are performed. (3) Recombinant inbred lines (RILs) are created *via* single seed descent or by doubled haploid production (Huang et al., 2015). This procedure results in a high number of recombination events enhancing the mapping resolution (Han et al., 2020).

The Bavarian MAGIC wheat population (BMWpop) is one of the only two German MAGIC wheat populations, which are mainly based on adapted German elite cultivars (Sannemann et al., 2018; Stadlmeier et al., 2018). It captures 71.7% of the allelic diversity present in the German wheat breeding gene pool (Stadlmeier et al., 2018). Thus, the BMWpop provides a greater potential to detect new QTL for resistance to important fungal pathogens as has been shown for powdery mildew, septoria tritici blotch, tan spot, leaf rust, and additional important agronomic traits (Stadlmeier et al., 2018, 2019; Rollar et al., 2021). The objectives of the present study were to (i) phenotype the BMWpop for quantitative and qualitative stripe rust resistance in multi-environment field trials and an extensive seedling test and to (ii) map QTL for these resistances to develop closely linked molecular markers suitable for marker-assisted selection (MAS).

MATERIALS AND METHODS

Plant Material

The study is based on the multiparental BMWpop comprising eight elite wheat cultivars (Stadlmeier et al., 2018). It consists of 394 diverse $F_{6:8}$ RILs, which were derived from a simplified eight founder MAGIC mating design with additional eight-way intercrosses. The founders "Event", "Bayp4535", "Potenzial", "Bussard", "Firl3565", "Format", "Julius", and "Ambition" originated from German and Danish wheat breeding programs and were selected on the criteria described by Stadlmeier et al. (2018). Detailed information about the

development and the genetics of the BMWpop were described by Stadlmeier et al. (2018).

Phenotypic Assessment of Stripe Rust Resistance in Field Trials

Six field trials were performed, each using a randomized incomplete block design with two replications at three locations in Germany: Quedlinburg (QLB, 51° 46′ 21.45 ″N 11° 8′ 34.8″ E) in Saxony-Anhalt, Soellingen (SOE, 52° 5′ 45.506 ″N 10° 55′ 41.711″ E) and Lenglern (LEN, 51° 35′ 47.53 ″N 9° 51′ 39.118″ E) in Lower Saxony. The 394 RILs, the eight founders, and the susceptible standard "Akteur" were evaluated for stripe rust resistance in double rows under natural disease epidemics in SOE (2017 and 2018) and LEN (2018 and 2019). In QLB, entries were sown in 2016/2017 and 2017/2018 in double rows of 1 m length with 30 plants per row and a spacing of 0.2 m between rows. Additional spreader plots with susceptible varieties were sown in regular intervals of every third plot. To ensure uniform infestation, the spreader plots were artificially inoculated in spring at the time of stem elongation (BBCH30, Meier, 2018) using the highly virulent *Puccinia striiformis* isolate Warrior + YR27 (**Supplementary Table 1**). For this, a spore suspension of 10 mg uredospores in 100 ml Isopar M (ExxonMobil Chemical Company, USA) was applied in a total amount of 10 ml suspension per m², using a hand-held spinning disc sprayer (Bromyard, UK). Phenotyping of the trials was carried out by scoring the average percentage of infected leaf area of the second and third youngest leaf in two rows at two to four subsequent dates according to Moll et al. (2010). Scoring started at the time of clearly visible disease symptoms on spreader plots and/or when leaves of the susceptible standard "Akteur" showed ≥10% diseased leaf area and was conducted in 1-to-2-week intervals.

Phenotypic Assessment of Stripe Rust Resistance in Seedlings

All RILs, the parental lines, and the susceptible standard "Akteur" were evaluated for resistance at the seedling stage in a detached leaf assay (Lück et al., 2020). Seedlings were grown in 77-cell propagation trays with mixed potting soil (Gebr. Patzer GmbH Co KG, Germany) using a randomized complete block design with four replications. Water agar (7 g L^{-1}) containing 45 mg L^{-1} benzimidazole (Sigma-Aldrich Chemie GmbH, Germany) for delaying senescence of leaf segments, was dispensed in 4 x 10 mL aliquots into non-sterile 4-well polystyrene plates (8 × 12 × 1 cM, Greiner Bio-One GmbH, Germany). Ten days after sowing, when the second leaf was fully developed, 2.5 cM sections were cut from the middle of the primary leaves and placed into the plates according to the initial randomization. White polytetrafluoroethylene frames (eMachineShop, NJ, USA) were used to fix the leaves. Inoculation was performed by an infection tower with the swirling duration of 3 s and settling time of 3 min (Melching, 1967). Due to space restrictions, the plates were divided into two infection groups per replication. Each group was inoculated with stripe rust isolate Warrior + YR27 using a mixture of 50 mg uredospores and white clay (1:1 w/w, VWR International GmbH, Bruchsal, Germany) after the

application of a 0.01% Tween 20 (Sigma-Aldrich) solution to support adhesion. For the first 24 h of incubation, the plates were covered by wet cotton paper, and placed into a climate cabinet at 7°C to support spore germination. Next, inoculated leaf segments were incubated in a growth chamber at night/day temperatures of 16°C/18°C with additional lighting (16 h/8 h day/night) for 15 days. Quantitative scoring was conducted using the high-throughput phenotyping platform "Macrobot" (Lück et al., 2020). Digital images with a resolution of 20 megapixel and four wavelengths between 315 nm and 750 nm (UV, blue, green, and red) were taken automatically from every plate. Subsequently, the leaf area was calculated and compared to the area of uredospore pustules for analyzing the percentage of infected leaf area (Pi) using the software HawkSpex® (Fraunhofer IFF, Germany). Additionally, all entries were visually evaluated for infection type (IT) using a 0–4 scale (McIntosh et al., 1995). To generate metric data, original IT data were converted to a 0–10 linear disease scale, modified according to Zhang et al. (2014), as below: 0, 0, N, −1, 1, +1, −2, 2, +2, −3, 3, +3 were coded as 0, 0.5, 0.75, 1, 2, 3, 4, 5, 6, 7, 8, and 9, respectively. The values IT −4 and 4 were coded as 10.

Data Analysis

The multiple scorings of the percentage of Pi in field trials were taken to calculate the area under the disease progress curve (AUDPC) and the average ordinate (AO) (Moll et al., 1996) for each RIL according to Rollar et al. (2021). For subsequent statistical analysis, only the AO values were used. Different year-location combinations of all trials were referred to as "environment". The analyses of all phenotypic data were carried out using *proc mixed* of the software package SAS 9.4 (SAS Institute Inc., NC, USA). To apply a mixed linear model, a log$_{10}$ data transformation of the AO, IT, and Pi values was performed. The factors, such as genotype, environment, and the genotype × environment interaction of field data, were set as fixed effects, while the design effects of replication and block were set as random. To obtain variance components for calculation of the broad-sense heritability, all model parameters were set as random. Heritability was estimated on a progeny mean basis using the formula according to Hallauer et al. (2010):

$$h^2 = \frac{V_G}{\frac{V_E}{re} + \frac{V_{GE}}{e} + V_G}$$

Where V_G is the genotypic variance, V_E is the environmental variance, V_{GE} is the genotype × environment variance, and r and e are the number of replicates and environments, respectively. For analyzing IT and Pi scores from the seedling test, the following formula was used:

$$y_{ijk} = \mu + g_i + r_j + l_k(r_j) + e_{ijk}$$

Where y_{ijk} is the trait observation, μ is the overall mean, g_i is the fixed effect of the genotype, r_j is the fixed effect of the replication, l_k is the random effect of the infection group nested in the replication, and e_{ijk} is the random residual error. Variance components were obtained by setting the genotype as random to

TABLE 1 | Descriptive statistics of raw data and heritability/repeatability for field trials (AO) and seedling test (IT and Pi).

Trait[a]	Mean founders	Mean population	Min[b]	Max[c]	SE[d]	CV[e]	h^2/rep
AO [%]	4.23	8.04	0	98.13	0.21	182.98	0.94[f]
IT [1-10]	1.28	1.72	0	10.00	0.06	129.08	0.76[g]
Pi [%]	0.22	0.92	0	25.00	0.07	185.74	0.58[g]

[a]Average ordinate (AO), infection type (IT), infected leaf area (Pi). [b]Minimum.
[c]Maximum. [d]Standard error. [e]Coefficient of variance. [f]Broad-sense heritability (h^2).
[g]Repeatability (rep).

calculate the repeatability as the ratio of the genotypic variance and the sum of the genotypic and the residual error variance divided by the number of replications. For each trait, least square means (ls means) were calculated and used for subsequent QTL analysis.

QTL Mapping

The BMWpop and the parental lines were genotyped using the 15K + 5K Infinium® iSelect® array (TraitGenetics, Germany) containing 17,267 single nucleotide polymorphisms (SNPs). The preparation of genotypic data and the construction of the linkage map used for QTL mapping were described in detail by Stadlmeier et al. (2018). QTL mapping was performed using the R (x32 3.2.5) package mpMap V2.0.2 (Huang and George, 2011; R Core Team, 2017). To conduct simple interval mapping (SIM), founder probabilities were calculated using the function "mpprob". To determine the parental origin of an allele, the threshold was set to 0.7. For SIM, a genome-wide significant threshold of $\alpha < 0.05$ was calculated for each trait. The thresholds were obtained from permutation of phenotypic data with 1,000 simulation runs (Churchill and Doerge, 1994). QTL detection was performed using the function "mpIM", implemented in the mpMap package (Huang and George, 2011). Phenotypic variance explained by individual QTL and additive QTL effects were estimated separately using the categorical allele information of the founders. A QTL support interval (SI) was defined as the map interval surrounding a QTL peak at a $-\log_{10}(p)$ drop of one unit.

To compare QTL identified in the present study with previously described QTL, overlapping QTL were merged based on the support interval. Databases of the Triticeae Toolbox (https://triticeaetoolbox.org/wheat/genotyping/marker_selection.php), GrainGenes (https://wheat.pw.usda.gov/GG3/), as well as CerealsDB (https://www.cerealsdb.uk.net/cerealgenomics/CerealsDB/axiom_download.php) were used to obtain marker information. Physical positions were obtained by nucleotide BLAST (BLAST-n) of the marker sequences against the reference sequence RefSeq v1.0 (Appels et al., 2018) using the database of 10+ Genome Project (https://webblast.ipk-gatersleben.de/wheat_ten_genomes/, Deng et al., 2007). BLAST hits were considered as significant if the percent identity was greater than 95% and only the best hit was taken if multiple BLAST hits were detected (Gao et al., 2016). The start and end positions of peak marker sequences preceded by the chromosome name were taken to the URGI database to obtain functional

gene annotations available from IWGSC (https://wheat-urgi.versailles.inra.fr/Seq-Repository/Annotations). Furthermore, a fixed chromosomal region of ± 500 kb on both sides of the QTL peak markers was examined for additional gene annotations and the output retrieved from URGI database was listed. Sequences of the closest related species, *Triticum urartu* (A-genome donor) and *Aegilops tauschii* (D-genome donor), were considered for the detection of orthologous genes.

RESULTS

Phenotypic Assessment

Stripe rust infestation of field trials was highly correlated between the year-location combinations (**Supplementary Figure 1**). Pearson's correlation calculations between the different environments showed only slight differences with high correlations between $r = 0.75$ and $r = 0.86$ ($p < 0.001$). A high heritability of $h^2 = 0.94$ was calculated (**Table 1**). The mean phenotypic distribution of AOs was right skewed with 266 RILs showing an AO smaller than 5% (**Figure 1A**). However, the mean distribution ranging between 0.4 and 58.1% (mean 8.0%) diseased leaf area and single maximum AO scores up to 98.1% were observed within the population (**Figure 1A**, **Table 1**). Six of eight founders showed mean AOs below 5%, resulting in a nonsignificant difference ($p < 0.05$) from the progeny mean. Founders "Bayp4535" and "Event" were identified as the most resistant (0.7%) and most susceptible (15.1%) parental lines to stripe rust, respectively. The analysis of variance showed significant differences concerning the genotype, environment, and the interaction between genotype and environment (**Table 2**).

For IT and Pi assessed in the seedling inoculation test, the phenotypic data revealed a high degree of resistance (**Figures 1B,C**). Phenotypic distributions of IT and Pi were strongly right skewed, with 287 and even 388 RILs showing IT values smaller than 2 and Pi values below 5%, respectively. The average IT ranged from 0.1 to 7.8 (mean 1.7). For Pi, the disease severity was on average between 0 and 11.1% (mean 0.9%). Maximal scores of 10 (IT) and 25% (Pi) were observed (**Table 1**). The population mean for IT was not significantly different from the mean of the parental lines, while a significant difference between the population and founder mean for Pi was observed. For IT and Pi, respectively, the parental lines "Potenzial" and "Bayp4535" turned out to be the most resistant. "Firl3565" was the most susceptible founder in the seedling inoculation test. Pearson's correlation displayed a high correlation coefficient between both traits ($r = 0.82$; **Supplementary Figure 2C**). The traits IT and Pi and the scoring of AO showed moderate correlations of $r = 0.63$ and $r = 0.46$ (**Supplementary Figures 2A,B**). For both traits, a significant effect of the genotype was observed. Repeatability of IT was high with $rep(\text{IT}) = 0.76$, while a moderate repeatability for Pi was calculated ($rep(\text{Pi}) = 0.58$, **Table 1**).

QTL Mapping

Overall, SIM revealed 21 QTL located on chromosomes 1A, 1D, 2A, 2B, 2D, 3B, 3D, 6A, and 7D. Eight of these were
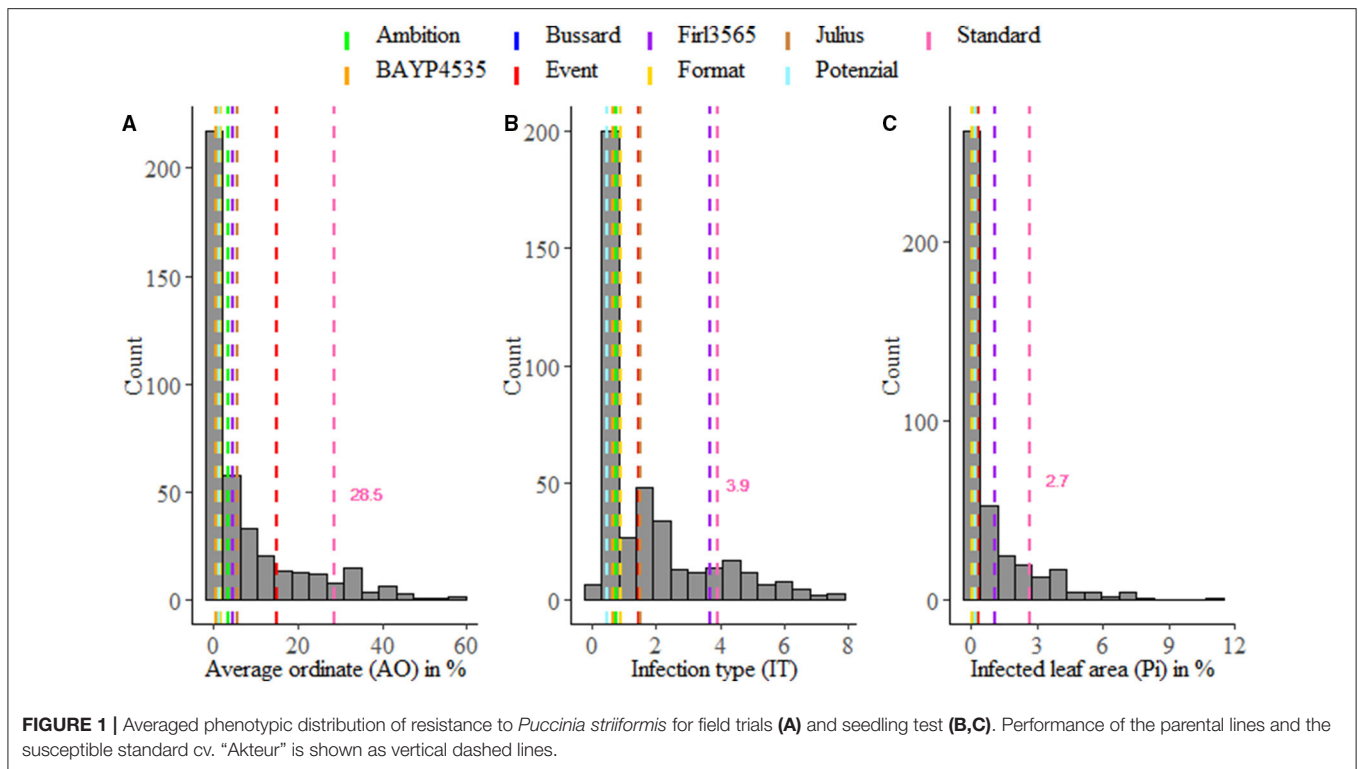
FIGURE 1 | Averaged phenotypic distribution of resistance to *Puccinia striiformis* for field trials **(A)** and seedling test **(B,C)**. Performance of the parental lines and the susceptible standard cv. "Akteur" is shown as vertical dashed lines.

TABLE 2 | Analysis of variance of $\log_{10}$-transformed data for leaf rust severity evaluated in field trials (AO) and seedling test (IT and Pi).

Trait[a]/factor	DF[b]	F value	P value
AO			
Genotype	402	58.16	<0.0001
Environment	5	101.57	<0.0001
Genotype × environment	2009	1.99	<0.0001
IT			
Genotype	402	4.29	<0.0001
Replication	3	1.54	0.3369
Pi			
Genotype	402	2.52	<0.0001
Replication	3	1.80	0.2917

[a] *Average ordinate (AO), infection type (IT), infected leaf area (Pi).* [b] *Degrees of freedom.*

detected based on field data averaged over six environments, seven QTL were found for IT, and six QTL for Pi (**Table 3**, **Supplementary Table 2**).

The phenotypic variance (R^2) explained by the individual QTL detected in field trials ranged between 1 and 29%, with SI from 6 cM to 81 cM. The three strongest QTL, explaining 23, 20, and 29% of R^2, were located on chromosomes 1A and 2B with peak markers at 16 cM, 106 cM and 172 cM, respectively. "Ambition", "Potenzial", and "Bayp4535" contributed to the largest allelic effects of these QTL, reducing disease severity (AO) by 2, 1.5, and 1.3%. Another QTL detected on chromosome 6A (at 259 cM) explained 16% of the phenotypic variance with

"Julius" as the most resistant founder line, reducing the Pi by 2.6%. On chromosomes 1A, 3B, and 7D, additional three QTL were detected at positions 62, 218, and 20 cM, respectively. The QTL accounted for 6% to 8% of stripe rust variation, while cv. "Bussard", "Julius", and "Potenzial" contributed to the largest allelic effects reducing the Pi by 1.8, 2.2, and 2.0%, respectively. The remaining QTL on chromosome 3D (4 cM) explained 1% of the phenotypic variance with "Firl3565" contributing to the highest allelic effect (-1.1%). All QTL detected over the mean of six environments were also identified by analyzing each environment separately (**Supplementary Table 2**). Hence, QTL located on chromosomes 1A, 1D, 2B, 3B, 3D, 6A, and 7D were identified in five (1A), two (1D), four (2B), five (2B), three (3B), four (3D), six (6A), and three (7D) environments, respectively (**Table 3**). However, on chromosome 4A, a QTL with a support interval (SI) between 159 cM and 200 cM was detected in LEN19, QLB18, QLB19, and SOE19, which was no longer significant when mean AO values across all environments were used (**Supplementary Table 2**).

For IT, the phenotypic variance explained by the seven QTL ranged from 1 to 16% with SIs between 5 and 34 cM (**Table 3**). QTL on chromosomes 2B and 6A accounted for the highest R^2, i.e., 16% each with peak markers at 164 cM and 260 cM, respectively. The founders "Bayp4535" and "Julius" reduced disease severity by 0.8 and 1.7 IT scores, respectively, contributing to the largest allelic effects. On chromosome 2D, one QTL was detected at 162 cM, explaining 9% of the phenotypic variance. A maximum effect of −1.1 IT scores was detected for the allele derived from cv. "Julius". Furthermore, two QTL were detected on chromosome 1A explaining 11% (at 12 cM) and 6%

TABLE 3 | QTL for resistance to *Puccinia striiformis* in the BMWpop detected in field trials (AO) and seedling tests (IT and Pi).

Trait	Chr.[a]	Pos.[cM][b]	SI [cM][c]	P value	R^{2d}	Eff (A)[e]	Eff (B)[e]	Eff (C)[e]	Eff (D)[e]	Eff (E)[e]	Eff (F)[e]	Eff (G)[e]	Eff (H)[e]
AO													
No. Env.[f]													
5	1A	16.37	0-34	2.47E-09	0.23	na	+0.97	−1.98	na	+2.02	−0.94	−0.06	na
2	1D	62.37	51-76	1.18E-05	0.06	−0.71	+0.70	+1.24	−1.76	+1.63	+0.31	−1.78	+0.38
4	2B	105.57	101-182	5.17E-13	0.20	+1.84	−1.38	na	na	na	−1.45	+0.35	+0.60
5	2B	163.5	158-167	1.33E-18	0.29	na	−1.27	na	na	na	+0.14	+1.13	na
3	3B	218.05	212-225	2.09E-05	0.07	+0.12	−0.97	−1.20	+2.21	+1.71	+1.38	−1.11	−2.17
4	3D	13.94	5-62	1.53E-05	0.01	+1.13	−0.49	na	−1.13	−0.53	na	na	na
6	6A	259.48	258-264	1.75E-23	0.16	−0.10	+1.80	+1.10	−1.28	+1.10	na	na	−2.62
3	7D	19.64	12-30	2.16E-06	0.08	na	+2.31	na	na	−0.57	−1.95	+0.07	0.12
IT													
	1A	11.77	0-34	6.14E-09	0.11	na	+0.53	−0.88	na	+1.56	−0.70	−0.49	na
	1A	210.75	197-215	0.0235	0.06	+0.45	−0.75	+1.55	+1.83	−0.73	−0.57	−1.00	−0.81
	2A	0.5	0-13	0.0039	<0.01	+0.19	−0.83	+1.22	−0.98	na	na	+1.22	−0.83
	2A	32.16	21-44	0.0377	0.01	+1.10	+0.05	−0.19	−0.10	−0.38	−0.41	+0.02	−0.05
	2B	163.5	155-167	1.33E-18	0.16	na	−0.82	na	na	na	0.25	0.56	na
	2D	161.57	144-166	0.0426	0.09	−0.03	na	na	na	+1.14	na	na	−1.10
	6A	259.98	258-263	6.57E-23	0.16	−0.15	+1.14	+0.98	−1.19	+0.88	na	na	−1.66
Pi													
	1A	204.48	191-215	0.0470	0.08	+0.22	−0.65	+1.37	+1.57	−0.69	−0.61	−0.63	−0.63
	2A	1.51	0-13	0.0041	<0.01	+0.73	−0.16	−0.22	−0.06	na	na	−0.10	−0.18
	2B	163.5	155-169	1.33E-18	0.12	na	−0.78	na	na	na	+0.29	0.50	na
	2B	197.5	184-217	8.11E-08	0.05	na	−0.54	na	na	+0.53	na	na	na
	2D	161.57	144-166	0.0426	0.07	−0.07	na	na	na	+1.10	na	na	−1.03
	6A	259.98	258-265	6.57E-23	0.10	−0.62	+0.76	+0.50	+0.38	+0.40	na	na	−1.40

[a]*Chromosomal position of QTL.* [b]*Position of peak marker based on the study by* Stadlmeier et al. (2018). [c]*Support interval.* [d]*Proportion of phenotypic variance explained by a single QTL.* [e]*Additive effects (±) of the founders Event (A), Bayp4535 (B), Ambition (C), Firl3565 (D), Format (E), Potenzial (F), Bussard (G), and Julius (H) relative to the population mean. Shown values are back-transformed to the original trait scale.* [f]*Number of single environments in which a QTL was detected. Founder effects were reported as not available (na) if none of the RILs reached the probability threshold.*

(at 211 cM) of the phenotypic variance. The cv. "Ambition" and "Julius" contributed to the highest allelic effect (−0.9 and −0.8 IT scores). Two QTL located on chromosomes 4D explained only 1% of the phenotypic variance each and were mapped at 1 cM and 32 cM.

QTL analysis of Pi values revealed six individual QTL with R^2 ranging from less than 1 to 12%. The SIs varied between 7 and 33 cM. QTL regions on chromosomes 1A, 2A, 2B, 2D, and 6A overlapped with QTL regions detected for IT (**Table 3**). The R^2 values of 12% (2B), 7% (2D), 10% (6A), 8% (1A), and <0.1% (2A) were calculated for individual QTL. The maximum reducing effects of each QTL for Pi ranged from 0.2 to 1.4%, contributed from different founders. Additionally, one QTL was detected on chromosome 2B at 198 cM, accounting for 5% of the phenotypic variance. A maximum effect of −0.5% was detected for the allele derived from the cv. "Bayp4535".

Based on SIs of 21 QTL detected in total for AO, IT, and Pi, 13 main QTL regions were derived, i.e., those detected for all estimated traits (**Supplementary Figure 3**, **Table 4**). *In silico* annotations of peak markers revealed seven genes with known functions partly involved in resistance. Marker *wsnp_Ex_c6488_11266589* on chromosome 1A referred to

CRS1-YhbY of *A. thaliana*, belonging to the chloroplast RNA splicing and ribosome maturation (CRM) domain-containing proteins. A dehydrogenase E1 component and a serine carboxypeptidase-like 19 were identified for peak markers for *QYr.jki-2A.1* and *QYr.jki-2A.2* on chromosome 2A. Markers *RAC875_c1226_652* and *AX-94388449* on chromosome 2B referred to BST_chr2B_nlr_143 and a formin-like protein 3, respectively. For the peak markers for *QYr.jki-2D* on chromosome 2D and *QYr.jki-3B* on chromosome 3B, GATA transcription factor 28 and a dual specificity phosphatase-catalytic domain were annotated. In addition, a fixed chromosomal region of ± 500 kb around each peak marker was examined. *In silico* annotations revealed additional gene annotations of different function on both sides of each QTL peak marker (**Supplementary Table 4**). On average, 24 gene annotations were identified within an interval of ± 500 kb on each side of the peak markers, including leucine-rich repeats for peak markers *AX-95080900* and *RAC875_c38756_141* of the QTL *QYr.jki-1A.1*, *wsnp_Ex_c28149_37293173* of QTL *QYr.jki-1A.2*, and *BobWhite_c13373_250* of *QYr.jki-2A.1*. In addition, NB-ARC domains were detected in the interval of peak markers *AX-95080900* and *wsnp_Ku_c23598_33524490* of QTL

QYr.jki-1A.1, *wsnp_Ex_c6488_11266589* of QTL *QYr.jki-1A.2*, *BobWhite_c13373_250* and *wsnp_Ku_c23598_33524490* of *QYr.jki-2A.1*, *AX-95177447* of *QYr.jki-2A.2*, *RAC875_c1226_652* of QTL *QYr.jki-2B.2*, *AX-94734962* of *QYr.jki-2D,* and *TA005377-1076* of *QYr.jki-7D*. Furthermore, protein kinase domains and/or ABC transporters were identified in the vicinity of peak markers *AX-95080900* and *RAC875_c38756_141* of QTL *QYr.jki-1A.1*, *BobWhite_c13373_250* and *wsnp_Ku_c23598_33524490* of *QYr.jki-2A.1,* and *AX-94526138* for QTL *QYr.jki-6A*. However, a minimum of four different resistance related gene annotations were identified in the interval of peak marker *AX 94388449* of the QTL *QYr.jki-2B.3*, while the maximum of 43 respective annotations were detected for *BobWhite_c13373_250* being the peak marker of QTL *QYr.jki-2A.1* (**Supplementary Table 4**).

DISCUSSION

Stripe rust occurs worldwide and is one of the most important pathogens in wheat cultivation. Known stripe rust resistances are present in many cultivars; however, their effectiveness is limited to certain races within the rust population in accordance with the gene-for-gene hypothesis (Flor, 1971). The emergence and selection of virulent pathotypes and their broad distribution results in considerable intraspecific variations in rust populations (Zetzsche et al., 2019). This in general causes the breakdown of qualitative resistances just a few years after their release (McDonald and Linde, 2002b; Kolmer, 2005). Thus, a continuous effort in wheat breeding programs is required to obtain a high degree of resistance to stripe rust by combining qualitative resistance genes with major effects and more durable APR. In this respect, the use of MAGIC populations in various QTL mapping studies turned out to be a powerful tool to detect both qualitative and quantitative resistance genes to different pathogens and other economically important traits (Pascual et al., 2015; Sallam and Martsch, 2015; Sannemann et al., 2015; Stadlmeier et al., 2019; Rollar et al., 2021).

In this study, more than 68% of the 394 RILs showed resistance to *Pucchinia striiformis*. A possible explanation for this can be found in the nature of the founder lines, of which almost all showed a high level of resistance to *P. striiformis* (**Figure 1**) suitable for the registration of varieties. Phenotypic data with many 0-values can lead to non-normally distributed residuals and thus affect the estimation of QTL effects in a regression-based QTL analysis. However, in this study, the phenotypic data were $\log_{10}$-transformed to ensure a normal distribution of the residuals for interval mapping. Thus, the right skewed distribution of the original phenotypic data did not affect the QTL detection results. With an average correlation coefficient of $r = 0.82$, minor differences between the disease severities in the six analyzed environments were observed. Additionally, a high broad-sense heritability of $h^2 = 0.94$ was calculated, which is in the range of previously published studies (Feng et al., 2018; Liu et al., 2018; Ma et al., 2019; Yang et al., 2019a). These results indicate that stripe rust resistance is highly heritable and that QTL detected in the different environments

were less affected by the occurrence of different *P. striiformis* races and/or different environmental conditions (Feng et al., 2018). Correlation between field data and seedling test results were as follows: $r = 0.63$ for IT and $r = 0.46$ for Pi, which are higher than the already reported correlations for leaf rust (Gao et al., 2016; Rollar et al., 2021). However, this observation may indicate similar scorings for seedling and adult plant resistance.

A method for linkage mapping in a MAGIC population was applied first by Xu (1996) based on the regression methods of Haley and Knott (1992). This method was used and subsequently improved based on parent probabilities by Mott et al. (2000), resulting in HAPPY. On this base, Huang and George (2011) finally developed the "mpMap" package, which was used in this study, by following a mixed-model context and including environmental and pedigree effects in the analysis. There are two main advantages of MAGIC populations: (1) Due to the crossing design of MAGIC populations, an increased genetic variation and recombination rate are achieved and (2) due to the increased genetic variation, QTL detection can be performed with increased precision and resolution (Cavanagh et al., 2008; Bandillo et al., 2013; Holland, 2015; Stadlmeier et al., 2019; and Rollar et al., 2021). This also comes along with smaller linkage blocks, a higher accuracy, and smaller SIs (Li et al., 2005; Stadlmeier et al., 2019). Overall, simple interval mapping in this study detected 21 QTL, of which only one QTL showed SI ≤ 5 cM. Nevertheless, Stadlmeier et al. (2019) successfully demonstrated the detection of QTL with small SIs in the BMWpop, which was supported by similar findings in other advanced intermated populations (Balint-Kurti et al., 2007; Huang et al., 2010). In the present study, 19% of the detected QTL showed SIs < 10 cM, and an average SI of 23 cM was calculated. Compared to double haploid (DH) lines, MAGIC populations are not completely homozygous. This residual heterozygosity can lead to problems, as heterozygotes for some markers cannot be distinguished in genotyping (Huang et al., 2015). This is particularly the case for polyploids and genotyping-by-sequencing (GBS) approaches (Elshire et al., 2011; Cavanagh et al., 2013). However, the mean proportion of heterozygous allele calls per RIL was described as 0.8% in the BMWpop (Stadlmeier et al., 2018).

The 21 QTL detected for AO, IT, and Pi correspond to 13 distinct chromosomal regions (**Table 4**, **Supplementary Figure 3**). QTL identified using the ls means across the six environments were also identified in the analyses of single environments (**Supplementary Table 2**). Additionally, a QTL for AO on chromosome 4A was detected in LEN19, QLB18, QLB19, and SOE19, describing 6% of phenotypic variance on average. Although this QTL was no longer significant by analyzing mean AO values across all environments, it may be of importance since there seems to be a relation to a QTL for leaf rust (*QLr.jki-4A.2*) mapped in a previous study (Rollar et al., 2021). At 13 distinct chromosomal regions, each of the five QTL was detected at the adult plant and seedling stages only. In contrast, three QTL were common to both growth stages, indicating the presence of effective all-stage stripe rust resistance genes. In total, the 13 QTL regions were located on wheat chromosomes 1A, 1D, 2A, 2B, 2D, 3B, 3D, 6A, and 7D.

TABLE 4 | Quantitative trait loci (QTL) resistance to *Puccinia striiformis* merged over all evaluated traits.

QTL	Chr.[a]	Peak markers for different traits	Determined by	Pos. [cM][b]	SI [cM][c]	Pos. RefSeq [bp][d] Start	End	Adjacent *T. aestivum* gene	Orthologous gene	Identity	Functional annotation
QYr.jki-1A.1	1A	AX-95080900	Field trials/ Seedling test	11.77	0–34	11893447	11893547	TraesCS1A01G017400LC			
		RAC875_c38756_141		16.37		7335009	7335109				
QYr.jki-1A.2	1A	wsnp_Ex_c28149_37293173	Seedling test	204.48	191–215	547965888	547966088	TraesCS1A01G370800	TRIUR3_02949[e] F775_06956[f]	99.85 95.27	
		wsnp_Ex_c6488_11266589		210.75		550613052	550613249	TraesCS1A01G376400	F775_01986[f]	98.66	CRS1-YhbY (CRM-domain)
QYr.jki-1D	1D	AX-94614313	Field trials	62.37	51–76	262248014	262248114	TraesCS1D01G294200LC			
QYr.jki-2A.1	2A	BobWhite_c13373_250	Seedling test	0.50	0–13	3962381	3962481	TraesCS2A01G010100	TRIUR3_01629[e]	97.70	Dehydrogenase E1 component
		wsnp_Ku_c23598_33524490		1.51		3447394	3447594	TraesCS2A01G007800	F775_30864[f] F775_31644[f]	97.24 98.22	Serine carboxypeptidase-like 19*
QYr.jki-2A.2	2A	AX-95177447	Seedling test	32.16	21–44	18165504	18165604				
QYr.jki-2B.1	2B	RAC875_rep_c109207_706	Field trials	105.57	101–182	69015103	69015203	TraesCS2B01G108000			
QYr.jki-2B.2	2B	RAC875_c1226_652	Field trials/ Seedling test	163.5	155–169	157693534	157693634	TraesCS2B01G182800			BST_chr2B_nlr_143
QYr.jki-2B.3	2B	AX-94388449	Seedling test	197.5	184–217	576083328	576083428	TraesCS2B01G406800	TRIUR3_14851[e]	98.97	Fomin-like protein 3*
QYr.jki-2D	2D	AX-94734962	Seedling test	161.57	144–166	636599900	636600000	TraesCS2D01G568600	F775_15392[f]	99.55	GATA transcription factor 28*
QYr.jki-3B	3B	BobWhite_c14365_59	Field trials	218.05	212–225	640059368	640059468	TraesCS3B01G404700	TRIUR3_12644[e]	98.84	Dual specificity phosphatase - catalytic domain
QYr.jki-3D	3D	Kukri_c3773_1450	Field trials	13.94	5–62	na	na				
QYr.jki-6A	6A	AX-94526138	Field trials, Seedling test	259.48	258–265	608502823	608502923	TraesCS6A01G598000LC			
		BS00067558_51		259.98		606439738	606439838	TraesCS6A01G391800	TRIUR3_27114[e] F775_21380[f]	98.15 95.94	
QYr.jki-7D	7D	TA005377-1076	Field trials	19.64	12–30	13295533	13295582	TraesCS7D01G027100	TRIUR3_33401[e] F775_32200[f]	96.45 100.00	

[a]Chromosomal position of QTL. [b]Position of peak marker based on the study by Stadlmeier et al. (2018). [c]Support interval. [d]Position of peak marker in the reference sequence RefSeq v1.0. [e]Triticum urartu. [f]Aegilops tauschii. *Information provided by https://www.cerealsdb.uk.net/cerealgenomics/CerealsDB/axiom_download.php.

Peak markers of QTL were partially annotated to genes, known to be involved in resistance mechanisms of plants. It was described that several serine carboxypeptidase-like proteins (QYr.jki-2A.2) catalyze the production of plant secondary metabolites involved in herbivory defense and UV protection (Fraser et al., 2005). Mugford et al. (2009) also reported a possible contribution of serine carboxypeptidase-like proteins in the synthesis of acylate plant defense compounds (avenacins) in oats. Peak marker wsnp_Ex_c6488_11266589 for QYr.jki-1A.2 was annotated to CRS1-YhbY, with a CRM protein domain. It was shown that CRM domain-containing proteins isolated from maize contribute to RNA binding activity (Barkan et al., 2007). Such RNA binding proteins are involved in various important cellular processes and in posttranscriptional regulation of gene expression, respectively. Thus, the RNA binding proteins play an important role in plant immune response regulation against pathogens, as they allow for a quick response to biotic and abiotic stress stimuli (Woloshen et al., 2011). A similar finding is the GATA transcription factor 28 for marker AX-94734962 on chromosome 2D. The GATA gene family is one of the most conserved families of transcription factors, playing a significant role in different aspects of cellular processes, e.g., in the abiotic stress signaling pathways (Gupta et al., 2017). The pyruvate dehydrogenase (E1) complex annotated for BobWhite_c13373_250 on chromosome 2A is involved in two interacting levels of control in plant cells. The first level is subcellular compartmentation contributing to tricarboxylic acid cycle and fatty acid biosynthesis, while the second level is the control of gene expression (Tovar-Méndez et al., 2003). The mean linkage disequilibrium (LD) decay for the genome in the BMW population is 9.3 cM, thus, considering a fixed interval of ± 5 Mb on both sides of a peak marker resulted in an excessive number of gene annotations (Stadlmeier et al., 2018). In this study, the fixed interval was reduced to ± 500 kb (1 Mb) based on several other studies in which the region on either side of the peak marker of a QTL was reduced to 100 kb (flax) (You and Cloutier, 2020), 2 kb (wheat) (Juliana et al., 2018), 2 kb (wheat) (Muqaddasi et al., 2020), or 100 kb (rice) (Hussain et al., 2020). However, examination of this interval led to the annotation of several leucine-rich repeats, NB-ARC domains, kinase domains, and ABC transporters. While leucine-rich repeats and NB-ARC domains are mainly involved in race-specific resistance responses, quantitative race unspecific resistance genes appear to encode different proteins, such as ABC transporters, protein kinases, and hexose transporters (Ellis et al., 2014; Moore et al., 2015; and Periyannan et al., 2017).

For the majority of the QTL detected in this study, the effect magnitudes were rather small as a high fraction of the population was highly resistant indicating that major stripe rust QTL were common to the founder lines. Two QTL were detected on chromosome 1A based on both field and seedling test data (QYr.jki-1A.1) and on seedling test data (QYr.jki-1A.2) only. QYr.jki-1A.1 is physically located in a region between 1.3 Mb and 12.5 Mb (Supplementary Table 3). To date, only one QTL for all-stage resistance to stripe rust was previously described in a similar region (Liu et al., 2018). QYrMa.wgp-1AS was mapped to the distal part of chromosome 1AS with the closest markers at 7.3 Mb (IWB57448) and 9.1 Mb (IWB5441). IWB57448 was also detected as peak marker for QYr.jki-1A.1 in this study (Table 4, Supplementary Table 3). Thus, the two QTL seem to be identical. QYr.jki-1A.2 was physically located at the distal end of chromosome 1AL between 540 Mb and 593 Mb. In the same region, there are two QTL (QYr.caas-1AL, QRYr1A.1) for APR to stripe rust (Ren et al., 2012; Rosewarne et al., 2012). These QTL were mapped at around 551 Mb and 575 Mb, respectively, but both were inconsistently detected across several environments. Another QTL (QYr.wsu-1A.2) detected at the adult plant stage and associated with marker IWA3215 was closely mapped to the distal end of QYr.jki-1A.2 around 593 Mb (Bulli et al., 2016). However, Jighly et al. (2015) described a QTL for seedling resistance that corresponds to QRYr1A.1 detected by Rosewarne et al. (2012) based on the linked DArT marker wPt-6005. Although QYr.jki-1A.2 was only detected in the seedling test, relationships between the aforementioned QTL previously described and QYr.jki-1A.2 based on physical positions might be possible.

On chromosome 1D, QYr.jki-1D was mapped in a large physical interval between 33 Mb and 366 Mb. However, the peak marker was located at 262 Mb. Furthermore, four QTL have been described at the distal end of chromosome 1DS, but none of these have been physically mapped near the region of QYr.jki-1D (Zwart et al., 2010; Vazquez et al., 2012; Hou et al., 2015; Naruoka et al., 2015). Maccaferri et al. (2015) reported the QTL QYr.ucw-1 D as a novel QTL independent of the aforementioned QTL. Its linked marker IWA980 is physically mapped at 36.3 Mb and is thus within the SI of QYr.jki 1D, but still far away from our peak marker (Supplementary Table 3). Ren et al. (2012) identified a QTL (QYr.caas-1D) flanked by markers Xgwm353 and Xgdm33b on chromosome 1DS in cv. "Naxos", but no physical marker information is available for a closer comparison (Supplementary Table 3). The resistance gene Yr25 was mapped on chromosome 1D and is one of the common Yr genes identified in European cultivars (McIntosh, 1988; Hovmøller, 2007). The stripe rust race Warrior + Yr27 used for inoculation in this study is virulent to Yr25 (Supplementary Table 1). This may give hint that QYr.jki-1D does not refer to this resistance gene.

QYr.jki-2A.1 and QYr.jki-2A.2 were both detected on chromosome 2AS based on the seedling test. To date, three designated Yr genes (Yr17, Yr56, and Yr69) and several QTL have been described on the short arm of chromosome 2A (Bariana and McIntosh, 1993; Hao et al., 2011; Lowe et al., 2011; Agenbag et al., 2012; Vazquez et al., 2012; McIntosh et al., 2014; Hou et al., 2016; Liu et al., 2018). QYr.jki-2A.1 was mapped between 3.1 Mb and 4.2 Mb, with peak markers at 3.4 Mb (Pi) and 3.9 Mb (IT, Table 4, Supplementary Table 3). Liu et al. (2018) located QYrMa.wgp-2AS around 2.7 Mb, corresponding to the region of Yr17, which was introgressed from Aegilops ventricosa to the hexaploid wheat line "VPM1" (Bariana and McIntosh, 1993). Based on the physical distance to our peak markers, it seems likely that QYr.jki-2A.1 corresponds to QYrMa.wgp-2AS and/or Yr17, respectively (Table 4, Supplementary Table 3). The second QTL QYr.jki-2A.2 was different from QYr.jki-2A.1 as the peak marker was mapped at 18.2 Mb. Nevertheless, QYr.jki-2A.2 was mapped in a large physical region from 5.7 Mb to

36.1 Mb, showing relationships with three QTL (*QYr.ufs-2A*, *QYr.uga-2AS*, *QYr.ucw-2AS*), as described previously. *QYr.ufs-2A* detected by Agenbag et al. (2012) was located in a region similar to *QYr.ucw-2AS* (Lowe et al., 2011) and *QYr.uga-2AS* (Hao et al., 2011). *QYr.ucw-2AS* was detected in an RIL population ("UC1110" × "PI610750") and is flanked by markers *wPt-5839* and *Xwmc177*, of which the latter was mapped at 33.7 Mb (Lowe et al., 2011). *QYr.uga-2AS*, which was derived from cv. "Pioneer26R61", was flanked by SSR markers *Xbarc124* (3.9 Mb) and *Xgwm359* (28.2 Mb) (Hao et al., 2011). Hence, all three QTL previously described are located in the chromosomal region of *QYr.jki-2A.2*, but further investigation is needed (**Supplementary Table 3**).

On chromosome 2B, QTL were detected based on field (*QYr.jki-2B.1*) and seedling test data (*QYr.jki-2B.3*) only, but also based on both data sets (*QYr.jki-2B.2*). QTL *QYr.jki-2B.1* was mapped to a large physical region between 69 Mb to 407 Mb, including the second QTL *QYr.jki-2B.2* (110.9 - 216.5 Mb). However, as the peak marker *RAC875_rep_c109207_706* was located at 69.0 Mb, *QYr.jki-2B.1* was designated separately and is assumed to be independent of *QYr.jki-2B.2* (**Table 4**, **Supplementary Table 3**). Chromosome 2BS is known to carry HTAP resistance that was detected in several wheat backgrounds (Ramburan et al., 2004; Guo et al., 2008; Carter et al., 2009; Chen et al., 2011). Chen et al. (2011) found *QYrid.ui-2B.1*, which was flanked by the markers *wPt-9668* and *Xgwm429*. The latter was physically mapped at 4.6 Mb proximal to the peak marker for *QYr.jki-2B.1*. As described by the authors, *QYrid.ui-2B.1* corresponds to two previously reported QTL: *QYr.sgi-2B.1* derived from cv. "Kariega" with the closest marker *Xgwm148* at 100.8 Mb (Ramburan et al., 2004) and *QYrlu.cau-2BS1* flanked by *Xwmc154* (36.4 Mb) and *Xgwm148* (100.8 Mb) (Guo et al., 2008). Based on these physical positions, *QYrid.ui-2B.1*, *QYr.sgi-2B.1*, and *QYrlu.cau-2BS1* appear to be located in the same region as *QYr.jki-2B.1* (**Supplementary Table 3**). For *QYr.jki-2B.2*, a similar conclusion can be drawn. In the study by Chen et al. (2011), a second QTL (*QYrid.ui-2B.2*) was identified, which was located in the same region as QTL *QYrlu.cau-2BS2Q* (Guo et al., 2008) and *Yrlo.wgp-2BS* (Carter et al., 2009). Together, the three QTL spanned a region from around 73.6 Mb to 448.7 Mb. The peak marker for *QYr.jki-2B.2* was mapped at 157.7 Mb, and thus is within the region of the three QTL described previously (**Supplementary Table 3**). The third QTL on chromosome 2BL (*QYr.jki-2B.3*) was detected for Pi values between 519 Mb and 724.5 Mb. Till date, there are seven designated *Yr* genes located on chromosome 2BL, of which *Yr5*, *Yr7*, and *YrSP* were already cloned between 615.8 Mb and 773.1 Mb (McIntosh et al., 2014; Marchal et al., 2018). Additionally, several QTL are described to be located at the long arm of chromosome 2B. One QTL was detected in the RIL population, "Camp Remy" × "Michigan Amber", and flanked by SSR markers *Xgwm47* (685.8 Mb) and *Xgwm501* (672.1 Mb) (Boukhatem et al., 2002). Another QTL (*QYraq.cau-2BL*) derived from cv. "Aquileja" was mapped between the markers *Xwmc175* and *Xwmc332* corresponding to 670.6–739.4 Mb (Guo et al., 2008). Guo et al. (2008) described that *QYraq.cau-2BL* corresponds to QTL which were previously detected by Mallard

et al. (2005) and Christiansen et al. (2006). These QTL in turn were assigned to the first-mentioned QTL detected by Boukhatem et al. (2002) and to resistance genes *Yr5* and *Yr7*, respectively (**Supplementary Table 3**). Although *QYr.jki-2B.3* seems to correspond to the aforementioned regions, the peak marker was mapped at 576.1 Mb, a physical distance of 94.5 Mb to the closest marker interval (**Table 4**, **Supplementary Table 3**). Thus, the relationship between *QYr.jki-2B.3* and the previously described QTL has still to be discussed. Furthermore, it is not clear whether *QYr.jki-2B.3* is related to the *Yr5*, *Yr7*, and *YrSP*.

QYr.jki-2D was mapped at the distal end of chromosome 2DL with the peak marker at 636.6 Mb. To date, there are six *Yr* genes (*Yr8*, *Yr16*, *Yr54*, *Yr55*, *Yr37*, and *YrCK*) known to be located on chromosome 2D. Unfortunately, no information on the physical positions is available for precise comparison. However, the APR gene *Yr16* was located in the centromeric region of chromosome 2D (Worland and Law, 1986; Ren et al., 2012), suggesting that this gene is different from *QYr.jki-2D*. Ren et al. (2012) reported a QTL on chromosome 2DL, flanked by the SSR marker *Xgwm539* (513.1 Mb) and *Xcfd44* (608.6 Mb). The authors assumed that this QTL is linked to two QTL as described previously, where both are closely linked to the marker *Xgwm349* (Suenaga et al., 2003; Melichar et al., 2008). This SSR marker is 7 bp apart from the peak marker of *QYr.jki-2D*. Hence, all three QTL may correspond to *QYr.jki-2D* (**Supplementary Table 3**).

On chromosome 3B, one QTL (*QYr.jki-3B*) was detected based on field trial data. The QTL SI spans a physical region from 581.3 Mb to 665.3 Mb, and is located on the long arm of chromosome 3B. There are many QTL previously reported that are partly summarized by Rosewarne et al. (2013) and Chen and Kang (2017). However, most of these are located on the short arm of chromosome 3B and do not correspond to *QYr.jki-3B*. In addition, the resistance genes *Yr4*, *Yr30*, and Yr57 were mapped on chromosome 3BS. Two QTL are detected on the long arm of chromosome 3B, *QYrex.wgp-3BL* (Lin and Chen, 2009) and *QYrid.ui-3B.2* (Chen et al., 2011). For both QTL, the SSR marker *Xgwm299* was reported as a flanking marker physically mapped at 804.8 Mb and does not correspond to the identified region of *QYr.jki-3B* (**Supplementary Table 3**). Recently, another QTL (*QYr-3BL*) was discovered in the durum wheat RIL population "Stewart" x "Bansi" flanked by the marker *IWB9451* (660.3 Mb) (Li et al., 2020). The authors associated this QTL with *Yr80*, a gene that is flanked by markers *KASP65624* and *KASP53113* spanning a physical region between 550.3 Mb and 605.4 Mb (Nsabiyera et al., 2018). Based on the physical positions, *QYr.jki 3B* may correspond to the resistance gene *Yr80*.

The quantitative trait locus *QYr.jki-3D* was mapped based on field data only. It is located at the distal end of chromosome 3DS between 19.8 Mb and 22.0 Mb. The two resistance genes *Yr49* linked to *Xgwm161* at 7.1 Mb, and *Yr66* linked to *IWB47165* at 2.6 Mb, as well as five QTL are described to be located on the arm of this chromosome (McIntosh et al., 2011, 2014; Basnet et al., 2013; Rosewarne et al., 2013). However, less marker information of QTL locations is available for precise comparison between *QYr.jki-3D* and QTL identified on chromosome 3DS by Boukhatem et al. (2002), Singh et al. (2000), and Basnet et al. (2013). Dedryver et al. (2009) found one QTL in cv.

"Recital" flanked by the markers *Xbarc125* (174.8 Mb) and *Xgwm456* (282.5 Mb). Another QTL was mapped between 309.9 Mb and 357.1 Mb, far away from the region identified in this study (Yang et al., 2013). Thus, neither the QTL nor the *Yr* genes correspond to *QYr.jki-3D*, which therefore seems to be novel.

Based on the field and seedling test data conducted in this study, a QTL (*QYr.jki-6A*) was detected on chromosome 6AL, with peak markers at 606.4 and 608.5 Mb. There are three regions conferring resistance to stripe rust which are all closely linked to SSR marker *Xgwm617* (William et al., 2006; Lillemo et al., 2008; Vazquez et al., 2012), which is 2.1 and 4.2 Mb away from our peak markers. William et al. (2006) reported the presence of *QYr.cimmyt-6A*, which corresponds to the QTL found by Lillemo et al. (2008), both contributed by the cv. "Avocet". It is likely that this QTL was derived from *Agropyron elongatum* due to a translocation in cv. "Avocet" (Lillemo et al., 2008). However, the third QTL (*QYrpl.orr-6A*) previously reported by Vazquez et al. (2012) was found in the RIL population "Stephens" × "Platte" and was also assigned to the QTL detected by Lillemo et al. (2008). A close relationship between these QTL and *QYr.jki-6A* can be assumed (**Supplementary Table 3**). Several additional QTL and major genes are reported to be located on chromosome 6A, including the resistance genes *Yr38, Yr42,* and *Yr81* (Marais et al., 2006, 2009; Prins et al., 2010; Cao et al., 2012; Rosewarne et al., 2012; Gessese et al., 2019). Unfortunately, the information provided was not sufficient to allow for further comparison.

The quantitative trait locus *QYr.jki-7D* based on data from field trials was located on the short arm of chromosome 7D. The QTL was physically mapped between 5.4 Mb and 29.4 Mb, with a position of the peak marker at 13.3 Mb. The five closest QTL already reported were linked to the SSR marker *Xgwm295* (53.6 Mb), which is 40.3 Mb apart from our peak marker (Ramburan et al., 2004; Navabi et al., 2005; Bariana et al., 2010; Yang et al., 2013). *Xgwm295* was found to be the closest microsatellite marker to the resistance complex *Lr34/Yr18* (Suenaga et al., 2003). In addition, Jighly et al. (2015) identified a QTL on chromosome 7DS linked to DaRT marker *wPt-668026*. The authors associated this QTL with the 7DS locus near the marker *Xbcd1438* described by Singh et al. (2000), which in turn was again associated with *Lr34/Yr18* (Jighly et al., 2015). This resistance gene has been functionally characterized and is already sequenced (Krattinger et al., 2009). However, due to the large distance between these QTL and the one detected in the present study, *QYr.jki-7D* seems to be a novel QTL (**Supplementary Table 3**).

The aim of this study was to use the Bavarian MAGIC wheat population to identify new sources of resistance to stripe rust, a fungal disease that causes devastating yield losses in wheat worldwide. The analyses resulted in 21 stripe rust resistance QTL that were confined to 13 distinct chromosomal regions. Eleven of these regions corresponded to QTL already described in previous studies. The increasing information on the physical map position of many stripe rust QTL, helped to infer the identity of the QTL found in the present study.

Two putatively new QTL were identified on chromosomes 3D (*QYr.jki-3D*) and 7D (*QYr.jki-7D*). SNP markers linked to these regions may be converted into KASP markers suitable for MAS in wheat breeding programs (Wu et al., 2017; Yang et al., 2019b). This will enable stacking of the detected resistance loci to breed new varieties with an improved resistance to stripe rust. Additionally, data and information generated in the present study can be used for weighted selection (Bernardo, 2014).

DATA AVAILABILITY STATEMENT

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

AUTHOR CONTRIBUTIONS

LH and FO planned and managed the project. LH and MG provided and characterized all RILs. MG contributed to the analyses of the results. AS, FO, MG, LH, and VM contributed to the interpretation and discussion of the results. SR conducted the field screenings and seedling test, analyzed the data, and wrote the manuscript. All authors contributed to the article and approved the submitted version.

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SUPPLEMENTARY MATERIAL

The Supplementary Material for this article can be found online at: https://www.frontiersin.org/articles/10.3389/fpls.2021.684671/full#supplementary-material

Supplementary Figure 1 | Pearson's correlation of stripe rust severity between different field trials. Diagonals are histograms for each environment (Lenglern LEN 2018-2019, Quedlinburg QLB 2017 2018, Söllingen SOE 2017 2018). *** denotes significance at α = 0.001.

Supplementary Figure 2 | Pearson's correlation (*r*) between averaged infection type (IT), infected leaf area (Pi) of seedling test and average ordinate (AO) of field trials **(A,B)**, as well as correlation between IT and Pi **(C)**. *** denotes significance at α = 0.001.

Supplementary Figure 3 | Simple interval mapping of resistance to *Puccinia striiformis* in field trials **(A)** and seedling test **(B,C)**. The x-axis shows the 21 wheat chromosomes. Positions are based on the genetic map, and the -log10(*p*) values of each marker are displayed on the y-axis (black line). The red horizontal line represents the significance thresholds. The seed index (SI) of the significant QTL detected in this study are colored in blue.

Supplementary Table 1 | List of virulences and avirulences of Puccinia striiformis isolate, Warrior + Yr27 used in field trials and seedling test. Brackets indicate

ambiguous results due to the differing symptom ratings between replications or moderate susceptibility (based on Zetzsche et al., 2019).

Supplementary Table 2 | Complete information of the quantitative trait loci (QTL) for stripe rust resistance in BMW population, evaluated in field trials (AO) and seedling test (IT and Pi).

Supplementary Table 3 | Comparison of the physical positions of the QTL identified in the present study (bold) with those reported previously.

Supplementary Table 4 | List of gene annotations for peak markers ± 500,000 bp, shown as output retrieved from URGI database (https://wheat-urgi.versailles.inra.fr/Seq-Repository/Annotations).

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CHAPTER IV | GENERAL DISCUSSION

1. Variation in disease severity within the population

Global agriculture is threatened by the rapid development and spread of pathogenic fungi that cause diseases such as rusts. For example, in recent years new races of wheat stem rust and stripe rust have emerged in parts of Africa, Asia and Europe, posing a threat to food security through their rapid spread and ability to overcome resistance in wheat varieties (Periyannan et al., 2017). Also, leaf rust is a serious disease because of its high diversity and adaptability to a wide range of climates (Figueroa et al., 2018).

The BMWpop is based on eight elite wheat lines, which were selected according to the criteria of multiple variation in agronomic, quality and disease resistance traits (Stadlmeier et al., 2018). The phenotypic variation in the BMWpop for leaf rust showed a high variability of resistance responses, indicating a high genetic diversity. Only a slightly right-skewed "normal" distribution was observed for the field data, while the seedling tests revealed an almost bi-modal distribution. These observations are in agreement with previously published studies and suggest a polygenic quantitative inheritance within the population as well as the presence of qualitative resistance conferred by a single gene (Lan et al., 2017; Aoun et al., 2019; Zhang et al., 2019). In contrast, phenotyping of stripe rust resistance in both field trials and seedling tests revealed a highly right-skewed distribution with more than 68 % of stripe rust resistant RILs. These results demonstrate that the variation within the population arises from the variation between the parental lines, as almost all of them showed high levels of resistance. Thus, the genetic variation of MAGIC populations is highly dependent on the prior selection of parental lines, for which the criteria must be carefully chosen. However, an important advantage of the BWMpop is that resistant RILs resulting from crossing elite material can be used directly in breeding programs without prior time-consuming backcrossing, compared to populations resulting from wild ancestors. A major concern in reviewing the studies was that many 0-values (resistant) could lead to residuals that are not normally distributed and thus affect QTL mapping. In all studies in this thesis, a $\log_{10}$-transformation of the phenotypic data was performed to ensure a normal distribution of the residuals for interval mapping. Furthermore, Vatter et al. (2018) have also shown efficient QTL detection using a NAM barley population that was highly resistant to *Puccinia striiformis* f. sp. *hordei* by ensuring a normal distribution of the residuals.

Successful infection and the extent of disease pressure are highly dependent on environmental conditions and thus influence the generation of reliable data in field trials

(McIntosh et al., 1995; Chen and Kang, 2017). In addition, simultaneous infection of trials with several different diseases can further complicate the evaluation of genotypic responses to a particular pathogen. In the field trials conducted to test the BMWpop for both leaf rust and stripe rust resistance, significant differences in genotype-environment interactions were observed. These differences may be due to different environmental conditions and due to different methods used at different locations, such as artificial or natural infection. However, for this reason, the focus was on the average performance of the genotypes across all environments by calculating model-adjusted LS means (Stigler, 1981; Govindarajulu et al., 2021). To demonstrate the validity of the data, correlations between environments and heritability were calculated. Moderate to high and significant correlations were found for all environment-environment combinations for both leaf rust and stripe rust. In addition, high heritabilities of 83 % and 94 %, respectively, were estimated, showing that disease severity depends mainly on genotype. Finally, simple interval mapping was performed for each environment separately, and it was generally found that the same QTL were found for the individual environments as for the mean data across environments. Overall, the results of this study highlight the suitability of the BMWpop for the detection of population-wide and parent-specific QTL conferring resistance to leaf and stripe rust. This is an additional advantage due to the previously established effective applicability for the detection of QTL for different traits by using multiparental populations (Sannemann et al., 2018; Stadlmeier et al., 2018; Stadlmeier et al., 2019).

In the present thesis, the set of 394 RILs of the BMWpop was investigated in field trials and seedling tests for resistance to the fungal pathogens *Puccinia triticina* and *Puccinia striiformis* f. sp. *tritici*, the causal agents of leaf rust and stripe rust, respectively. Based on their phenotypic response, RILs were identified that showed particularly increased resistance to these rust pathogens. A resistant phenotypic response was defined as disease severity of ≤ 15 % or infection response type of ≤ 2. Leaf rust was tested in five environments (LEN18, QLB17, QLB18, SOE17, SOE18) and stripe rust was tested in six environments (LEN17, LEN18, QLB17, QLB18, SOE17, SOE18). In addition, both leaf rust and stripe rust were evaluated in the seedling tests under controlled environmental conditions in five and four replications, respectively. Out of 394 RILs, 331 RILs showed a resistant phenotype to leaf rust in at least two environments (year-location combination). Of these, 98 RILs were also resistant at the seedling stage (Table A.3-1). Ninety RILs were resistant in all five test environments, so almost a quarter of the BMWpop had high resistance to leaf rust with a total average AO of 5.81%. Only twenty-four of these ninety RILs also expressed resistance responses at the seedling tests.

The remaining 103, 73 and 65 RILs showed resistance to leaf rust in four, three and two environments, of which forty, nineteen and fourteen RILs were also resistant at the seedling stage respectively. For stripe rust, 355 out of 394 RILs showed a resistant phenotype in at least two environments, of which 270 RILs were also observed to be resistant at the seedling stage (Table A.3-2). A total of 250 RILs were resistant in all six environments tested, for which an average AO of 1.51 % was calculated. 223 of these 250 RILs, i.e., almost 90 %, showed a resistance reaction in the seedling tests. The remaining 48, 25, 21 and 11 RILs showed resistance to stripe rust in five, four, three and two environments respectively, of which twenty-six, eleven, three and seven RILs were also resistant at seedling stage. Half of the BMWpop, about 200 RILs, showed resistance to both leaf and stripe rust.

2. Suitability of BMWpop for QTL mapping

Stadlmeier et al. (2019) evaluated the BMWpop for the first time for its suitability to detect QTL conferring resistance to fungal pathogens such as *Blumeria graminis*, *Zymoseptoria tritici*, and *Pyrenophora tritici-repentis*. For QTL detection, the analysis tool 'mpMap' implemented in R was used to conduct SIM, which was developed by Huang and George (2011) and shown by Stadlmeier et al. (2019) to be very effective for QTL detection in BMWpop. In general, 'mpMap' also offers the option to carry out CIM, but only SIM was conducted in the context of this thesis. CIM attempts to address the problem of epistasis, the genetic interaction between independent loci, and can thus increase the statistical power; however, the selection of covariates is crucial and determines whether CIM is superior to SIM. If the number of markers selected is too low or too high, the power of QTL detection will be compromised. Furthermore, the final QTL detection does not take into account the uncertainty in the selection of covariates and may lead to overly optimistic support intervals. Because of these drawbacks, Broman (2001), the developer of R/qtl and author of critical papers about interval mapping methodology generally recommends against the use of CIM (Broman and Sen, 2009).

A major concern with regard to the statistical power of linkage mapping in MAGIC populations is the possible reduction in the number of detectable QTL due to the complex genetic background (Keurentjes et al., 2011; Stadlmeier et al., 2019). While Kover et al. (2009) and Huang et al. (2011) each detected fewer QTL when using multiparental populations than linkage analyses with bi-parental populations for the same trait, other studies did not detect differences in the number of QTL (Gnan et al., 2014). Other studies reported the detection of new QTL, presumably due to increased genetic variation resulting from the use of multiple parents (Pascual et al., 2015; Sannemann et al., 2015). In this thesis, simple interval mapping

based on field trials and seedling tests revealed 19 and 21 QTL corresponding to in total 11 and 13 different chromosomal regions, respectively, controlling both leaf rust and stripe rust resistance. The comparison of these QTL with already described genes and QTL, known to be involved in resistance to rusts has already been discussed extensively in the context of Chapter II and Chapter III. In more detail, two genetic regions were identified on the short arm of chromosome 1A (1AS) and on the short arm of chromosome 7D (7DS), which are associated with both rust diseases. *QLr.jki-1A.1* and *QYr.jki-A.1* overlapped perfectly based on their support intervals (SI), which both span a chromosomal region between 0 and 30 cM. However, while *QYr.jki-A.1* was detected in field trial and seedling tests, *QLr.jki-1A.1* was only identified at the adult plant stage. Based on the positions of corresponding peak makers for *QLr.jki-1A.1* (*RAC875_c57939_78*) and *QYr.jki-A.1* (*RAC875_c38756_141*), QTL are still at least 9.75 cM apart from each other, which physically represents a distance of 4.2 Mbp. On chromosome 7DS, *QLr.jki-7D.1* and *QYr.jki-7D* were located within SI of 15-30 cM and 12-30 cM, respectively, with peak markers mapped between 18.1 cM to 21.7 cM. In addition, Stadlmeier et al. (2019) discovered a QTL (*QPm.lfl-7D*) associated with resistance to powdery mildew in the same chromosomal region of BMWpop, with an SI of 12 cM to 22 cM and a peak marker at 19 cM. The authors reported that *QPm.lfl-7D* corresponds to the multi-resistance gene *Lr34/Yr18/Sr57/Pm38*, even if the peak marker is 34 Mbp distal to the APR gene *Lr34* (Krattinger et al., 2009; Stadlmeier et al., 2019). Even though *QLr.jki-7D.1* and *QYr.jki-7D* were not associated with *Lr34* and *Yr18* respectively due to their physical position, there is strong evidence for the presence of *Lr34/Yr18/Sr57/Pm38* as resistance to leaf rust, stripe rust and powdery mildew is recorded in the same chromosomal region in three independent studies (Stadlmeier et al., 2019; Rollar et al., 2021b; Rollar et al., 2021a). An additional argument for this multi-resistance gene based on parental information, as it is also reported that the parental line 'Potenzial' possesses this gene (Serfling et al., 2011). However, all three QTL were still mapped far from corresponding resistance genes, and in addition, *QLr.jki-7D.1* was also detected at the seedling stage, suggesting that at least *QLr.jki-7D.1* is distinct from the APR gene *Lr34/Yr18/Sr57/Pm38* (Rollar et al., 2021b).

For plant breeders, the benefit of QTL conferring resistance depends on the percentage of phenotypic variance explained by the QTL. A phenotypic variance of at least 10 % to 20 % should be explained by a QTL to make MAS feasible (Miedaner and Korzun, 2012). In this work, QTL with high phenotypic variances of up to 50 % and 29 % for leaf rust and stripe rust, respectively, as well as phenotypic variances of less than 1 % were detected. However, especially for stripe rust, only small allele effects of less than 5 % or less than 3 scores were

calculated for the QTL. Mapping rare QTL variants in combination with small effects is already described as a challenge in MAGIC populations (Kover et al., 2009). This is associated with a small phenotypic variance, which in turn depends on both phenotypic and genetic variance. In MAGIC designs, there is a higher probability of markers with lower minor allele frequencies. In the case of a MAGIC design with eight parental lines, the minor allele frequency in the extreme case is 1/8. Since genotypic variance is a function of the QTL effect and the allele frequency of the QTL, a low minor allele frequency reduces the estimated proportion of explained phenotypic variance of a QTL regardless of the actual value of the phenotypic variance (Falconer and Mackay, 1996; Uemoto et al., 2015; Stadlmeier et al., 2019). However, minor QTL and QTL with moderate effects are necessary to achieve durable resistance, as major genes without any quantitative resistance are in general overcome quickly. Pyramiding of resistance genes or QTL, even with a minor effect, may be considered a successful strategy to increase the overall resistance level. Thus, the introgression of genes by MAS allows the combination of several genes/QTL into a single cultivar (Gupta et al., 2010). Today, multi-gene pyramiding strategy is successfully used in resistance breeding of crops such as rice (Liu et al., 2016), maize (Jiang et al., 2012), soybean (Wang et al., 2017) and wheat (Wang et al., 2001; Pietrusińska et al., 2011; Bai et al., 2012; Singh et al., 2017). More recently, the use of MAS has enabled the pyramiding of up to 12 genes/QTL for grain quality and rust resistance in wheat (Gautam et al., 2020). Nevertheless, in the context of complex quantitative traits, especially with small effects, practical breeding is shifting from MAS to genomic selection (GS), which seems to be more efficient (Heffner et al., 2009; Crossa et al., 2017). GS is a method to overcome the limitations of MAS for quantitative traits (Robertsen et al., 2019). Where MAS uses a defined subset of significant markers, GS takes advantage of all molecular markers for genomic prediction of the performance of the candidates for selection (Heffner et al., 2009; Crossa et al., 2017). In general, genomic selection is a form of MAS in which all locus, haplotype or marker effects across the genome are estimated simultaneously in order to calculate genomic estimated breeding values (GEBVs) (Meuwissen et al., 2001). By combining molecular and phenotypic data in a training population, GS determines the GEBVs of individuals in a testing population that have been genotyped but not phenotyped. In other words, both phenotypic and genotypic data of the training population are used to estimate GS model parameters to subsequently calculate GEBVs of selection candidates (testing population) of which only the genotypic data are available (Meuwissen et al., 2001; Heffner et al., 2009; Crossa et al., 2017). Thus, GS takes advantage of dense markers to quantify Mendelian sampling to avoid the need to extensively phenotype the progeny, making it the most promising

breeding method to accelerate the development and release of new genotypes (Crossa et al., 2017).

3. Annotation of putative candidate genes

In the present thesis, the BMWpop comprising 394 RILs that are based on elite wheat material was evaluated for resistance against the fungal pathogens *Puccinia triticina* Eriks. and *Puccinia striiformis* Westend., the causal agents of leaf rust and stripe rust. The RILs were phenotyped in field trials at three locations in Germany and in seedling test under controlled conditions. Subsequent simple interval mapping revealed 19 and 21 QTL corresponding to in total 11 and 13 different chromosomal regions conferring resistance to leaf rust and stripe rust. Gene annotation was performed for each peak marker by transferring the start and end positions to the IWGSC RefSeq v1.0 genome assembly of the URGI database (https://wheat-urgi.versailles.inra.fr/Seq-Repository/Annotations) (Alaux et al., 2018). However, the definition of a single candidate gene of the detected QTL only based on the peak markers is not sufficient also due to the fact that a mean LD-decay of 9.3 cM was calculated for the BMWpop (Stadlmeier et al., 2018). In turn, large support intervals were calculated for the different QTL, which also do not allow an adequate representation of potential candidate genes for the relevant QTL. Therefore, the search for candidate genes was limited to a defined resolution of ±500 kb around the peak markers, which has proven to be sufficient to identify candidates (Hussain et al., 2020; Muqaddasi et al., 2020; You and Cloutier, 2020). The number of predicted genes for identified QTL ranged between 10 to 38 genes for leaf rust and 4 to 43 genes for stripe rust resistance (Table IV.3-1). For leaf rust, 12 regions harbored leucine-rich repeats (LRRs) and/or NB-ARC domains (complete list of predicted genes for leaf rust is not published). Ten LRR/NB-ARC containing regions were found for stripe rust.

Table IV.3-1 | Number of predicted genes that are associated with disease recognition or defense located in identified QTL regions.

QTL	Marker	Chr.	Interval [Mb]	Total No of annotation	No leucine-rich repeats	No NB-ARC domains	No ABC transporter	No protein kinase domains
QLr.jki-1A.1								
	RAC875_c57939_78	1A	11.1 - 12.1	16	4	0	0	1
	CAP8_c2448_355		9.6 - 10.6	34	1	3	0	0
QLr.jki-4A.1								

QTL	Marker	Chr.	Interval [Mb]	Total No of annotation	No leucine-rich repeats	No NB-ARC domains	No ABC transporter	No protein kinase domains
	Kukri_rep_c109167_89	4A	634.2 - 635.2	10	0	0	0	1
QLr.jki-4A.2								
	Excalibur_c46904_84	4A	736.8 - 737.8	21	0	1	0	4
	BobWhite_c47168_598		725.7 - 726.7	29	3	4	0	0
QLr.jki-4D.1								
	BS00023112_51	4D	455.3 - 456.3	28	0	0	0	0
	AX -95126745	4D	464.5 - 465.5	18	0	0	0	1
QLr.jki-5A.1								
	IAAV2363	5A	481.4 - 482.4	17	0	1	0	0
	RAC875_c31670_389	5A	513.6 - 514.6	17	0	1	0	0
QLr.jki-6B.1								
	AX -94557244	6B	25.4 - 26.4	38	2	2	0	1
QLr.jki-6B.2								
	RAC875_c59968_234	6B	712.2 - 713.2	35	3	4	1	2
QLr.jki-7A.1								
	BS00011330_51	7A	62.6 - 63.6	25	0	4	0	0
QLr.jki-7A.2								
	wsnp_Ku_c26530_36497050	7A	84.3 - 85.3	28	2	1	0	0
QLr.jki-7A.3								
	BS00011622_51	7A	711.8 - 712.8	28	3	0	0	1
QLr.jki-7D.1								
	AX -94930280	7D	15.6 - 16.6	17	0	4	0	0
	IACX11794	7D	12.0 - 13.0	10	0	0	0	0
QYr.jki-1A.1								
	AX -95080900	1A	11.4 - 12.4	39	5	2	0	2
	RAC875_c38756_141	1A	6.8 - 7.8	39	2	1	0	2
QYr.jki-1A.2								
	wsnp_Ex_c28149_37293173	1A	547.5 - 548.5	20	1	0	0	0
	wsnp_Ex_c6488_11266589	1A	550.1 - 551.1	18	0	1	0	0
QYr.jki-1D								
	AX -94614313	1D	261.7 - 262.7	24	0	0	0	0
QYr.jki-2A.1								
	BobWhite_c13373_250	2A	3.5 - 4.5	43	10	2	2	1

QTL	Marker	Chr.	Interval [Mb]	Total No of annotation	No leucine-rich repeats	No NB-ARC domains	No ABC transporter	No protein kinase domains
	wsnp_Ku_c23598_33524490	2A	2.9 - 3.9	30	0	3	2	0
QYr.jki-2A.2								
	AX -95177447	2A	17.7 - 18.7	39	0	2	0	0
QYr.jki-2B.1								
	RAC875_rep_c109207_706	2B	68.5 - 69.5	24	0	0	0	0
QYr.jki-2B.2								
	RAC875_c1226_652	2B	157.2 - 158.2	10	0	1	0	0
QYr.jki-2B.3								
	AX 94388449	2B	575.6 - 576.6	4	0	0	0	0
QYr.jki-2D								
	AX 94734962	2D	636.1 - 637.1	23	0	5	0	0
QYr.jki-3B								
	BobWhite_c14365_59	3B	639.6 - 640.6	5	0	0	0	0
QYr.jki-3D								
	Kukri_c3773_1450	3D	19.3 - 22.5	55	0	0	0	2
QYr.jki-6A								
	AX -94526138	6A	608.0 - 609.0	42	0	0	1	3
	BS00067558_51	6A	605.9 - 606.9	9	0	0	0	0
QYr.jki-7D								
	TA005377 -1076	7D	12.8 - 13.8	19	0	1	0	0

LRRs play an important role in the immune system of plants, which uses immune receptors to recognize pathogens and trigger defense responses. Most of these plant immune receptors possess a LRR domain, a structure characterized by a conserved pattern of hydrophobic leucine residues. LRR domains have a broad interaction surface that can tolerate a high degree of variability. Therefore, LRRs exhibit different classes of immune receptors that serve to mediate protein-protein interactions and thus have a dual function as a guardian and activator in plant defense against pathogens (Padmanabhan et al., 2009). When LRR domains are fused with a central nucleotide binding domain, NB-LRR proteins are formed. The core nucleotide-binding fold in NB-LRR proteins is part of a larger entity called the NB-ARC domain because of its presence in APAF-1 (apoptotic protease-activating factor-1), R proteins and CED-4 (*Caenorhabditis elegans* death-4 protein) (van Ooijen et al., 2008). Thus, these protein

constructs are the first receptors to recognize MAMP/ and PAMP in PTI, and Avr proteins in ETI, respectively. The LRR domain is involved in auto-inhibition and/or effector recognition and the central NB-ARC domain acts as a regulatory domain determining whether the protein is in an active or inactive state (Bent et al., 1994; DeYoung and Innes, 2006; Steele et al., 2019). First active or induced defense response of plants is, e.g. the production of ROS (oxidative bursts) initiating HR (Almagro et al., 2009). By catalyzing the oxidoreduction of hydrogen peroxide, peroxidases (*AX-94557244*; *QLr.jki-6B.1*) are involved in ROS metabolism and thus play an important role in pathogen recognition and disease resistance. Furthermore, peroxidases are involved in the biosynthesis of the cell wall components, such as lignin and suberin (Hiraga et al., 2001; Almagro et al., 2009). Another enzyme involved in lignin and suberin biosynthesis and associated with increased cell wall strength, stress tolerance and disease resistance is o-methyltransferase (Lam et al., 2007; Novakazi, 2020). Two family proteins of these enzymes were identified within the fixed interval for *QLr.jki-4A.1* (*Kukri_rep_c109167_89*) and *QYr.jki-2D* (*AX-94734962*).

While race-specific resistance genes often encode NB-LRR proteins, race-nonspecific resistance genes encode ABC transporters as well as protein kinases, which are involved in various molecular mechanisms (Keller et al., 2005; Krattinger et al., 2011). For leaf rust and stripe rust, one (*QLr.jki-6B.2*; *RAC875_c59968_234*) and two regions (QYr.jki-2A.1; BobWhite_c13373_250/ wsnp_Ku_c23598_33524490; QYr.jki-6A; AX-94526138) respectively, were found to contain ABC transporters (Table IV.3-1). In general, transporters are integral membrane proteins present in all organisms and differ in the number of transmembrane domains and transmembrane helices. In plants, ABC transporters belong to one of five different transporter families involved in disease resistance. (Devanna et al., 2021). Originally, they were identified as transporters involved in detoxification processes. Today, it is known that ABC transporters transport a wide range of substrates involved in various processes required for organ growth, plant nutrition, plant development, response to abiotic stress, resistance to pathogens and the interaction of plants with the environment (Kang et al., 2011; Devanna et al., 2021). The ABC superfamily consists of eight subfamilies, designated ABCA to ABCI, with ABCH absent and ABCB and ABCG most abundant in plants. These subfamilies are further divided into three categories based on their structural domains, number of transmembrane domains and nucleotide binding domains (Hwang et al., 2016; Devanna et al., 2021). One of the most important quantitative resistance genes incorporated in wheat today is *Lr34/Yr18/Sr57/Pm38*, which encodes the pleiotropic drug resistance (PDR)-type ABCG transporter that enables the plant to limit the growth and development of various rust fungi as

well as powdery mildew (Krattinger et al., 2009; Krattinger et al., 2011). In Arabidopsis, a group of three ABC transporters (PDR, ABCG36 and PEN3) associated with glucosinolate-dependent defense mechanisms restricts the penetration of non-host fungal pathogens (Hwang et al., 2016).

In the fixed interval of *wsnp_Ex_c6488_11266589* (*QYr.jki-1A.2*) a heat shock factor was annotated. Heat shock factors are the transcription factors that regulate the expression of heat shock proteins (HSP) (Sorger, 1991). HSPs are divided into six classes, of which HSP70 and HSP90 are chaperones responsible for protein folding, translocation and degradation, but are also involved in the response to abiotic and biotic stress factors (Rajan and D'Silva, 2009; Al-Whaibi, 2011). Furthermore, HSP90 regulates cellular signaling, e.g. glucocorticoid receptor activity, while cytoplasmic HSP90 is responsible for pathogen resistance by reacting with pathogen signaling receptor and thus contributes to resistance of wheat to stripe rust (Pratt et al., 2004; Al-Whaibi, 2011; Wang et al., 2011). In contrast, HSP70 does not function alone, but binds to so-called J-proteins (DnaJ proteins), which belong to the HSP40 family and determine the specificity of HSP70 for function in various cellular processes (Rajan and D'Silva, 2009). Corresponding DnaJ domains were identified in the fixed intervals of *CAP8_c2448_355* (*QLr.jki-1A.1*), *Kukri_rep_c109167_89* (*QLr.jki-4A.1*) and *IAAV2363* (QLr.jki-5A.1). The function of HSP40 against viral pathogens has been well studied in various virus-plant interactions. For example, HSP40 was shown to cause HR-like cell death in soybean when overexpressed during infection with *Soybean mosaic virus*, while silencing resulted in increased susceptibility (Liu and Whitham, 2013; Park and Seo, 2015). However, Zhong et al. (2018) demonstrated increased ROS content and improved resistance of rice to the rice blast fungus *Magnaporthe oryzae* by silencing DnaJ OsDjA6.

In the interval of six and seven peak markers for leaf and stripe rust, respectively, on chromosomes 1A, 2A, 2B, 3D, 4A, 4D and 7D, cytochrome P450 was annotated. The cytochrome P450 (CYP) superfamily is the largest enzymatic protein family in plants, and has been found in different organisms such as animals, fungi, protists, archaea, bacteria and viruses (Xu et al., 2015). The members of this superfamily are involved in numerous metabolic pathways with diverse and complex functions and play an important role in a variety of reactions (Xu et al., 2015). In terms of resistance to biotic stress factors, CYPs are important enzymes involved in the synthesis of various metabolites such as alkaloids, cyanogenic glucosides and terpenoids, but also in the synthesis of various defensive signaling molecules such as oxidized fatty acids and hydroperoxides (Xu et al., 2015). Thus, CYP93G2 was found to function as a flavanone 2-hydroxylase providing 2-hydroxyflavanones for C-glycosylation,

while CYP93G1, the closest homologue of CYP93G2, is a flavone synthase II that catalyzes the direct conversion of flavanones to flavones in rice (Lam et al., 2014). In addition, CYP are also involved in the biosynthesis of important cell wall components. In wheat, the CYP98 family catalyzes the 3-hydroxylation step in the phenylpropanoid pathway leading to the biosynthesis of suberin phenol monomers (Morant et al., 2007).

Using the Bavarian MAGIC wheat population, already known and established sources of resistance against the two rust fungi *Puccinia triticina* and *Puccinia striiformis* were detected, but also putative new QTL. For almost all QTL discovered in this study, genes directly involved in plant immunity were detected. Thus, the BMWpop contains promising candidate genes within the QTL that are worth further analysis. SNP markers for QTL can be converted to KASP markers usable in breeding for improved rust resistance.

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APPENDIX

1. **Supplementary material of Chapter II | QTL mapping of adult plant and seedling resistance to leaf rust (*Puccinia triticina* Eriks.) in a multiparent advanced generation intercross (MAGIC) wheat population**

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Fig. S1 Pearson correlation of leaf rust severity between different field trials. Diagonals are histograms of each environment (Lengern LEN 2018, Quedlinburg QLB 2017 2018, Söllingen SOE 2017 2018). *** denotes significance at α = 0.001. Lowess curves were adjusted to the data points with a smoothing range of 0.75, based on the 'lowess' function implemented in the R-based 'stats' package

Fig. S2 Pearson correlation (r) between averaged infection type (IT), infected leaf area (Pi) of seedling test and average ordinate (AO) of field trials (A, B), as well as correlation between IT and Pi (C). *** denotes significance at α = 0.001

Fig. S3 Simple interval mapping of resistance to *Puccinia triticina* in field trials (A) and seedling test (B, C). The x axis shows the 21 wheat chromosomes. Positions are based on the genetic map, and the $\log10(p)$ values of each Marker are displayed on the y axis (black line). The red horizontal line represents the significance thresholds. SI of the significant QTL detected in this study are coloured in blue

Tab. S1 List of virulences and avirulences of *Puccinia triticina* isolate 77WxR used in field trials and seedling test. Brackets indicate ambiguous results due to the differing symptom ratings between replications or moderate susceptibility (based on Zetzsche et al. 2019)

Tab. S2 Complete information of the 19 QTL for leaf rust resistance in BMW population, evaluated in field trials (AO) and seedling test (IT and Pi)

2. Supplementary material of Chapter III | Quantitative trait loci mapping of adult plant and seedling resistance to stripe rust (*Puccinia striiformis* Westend.) in a multiparent advanced generation intercross wheat population

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https://www.frontiersin.org/articles/10.3389/fpls.2021.684671/full#supplementary-material

Supplementary Figure 1 Pearson's correlation of stripe rust severity between different field trials. Diagonals are histograms for each environment (Lenglern LEN 2018-2019, Quedlinburg QLB 2017 2018, Söllingen SOE 2017 2018). *** denotes significance at $\alpha = 0.001$.

Supplementary Figure 2 Pearson's correlation (r) between averaged infection type (IT), infected leaf area (Pi) of seedling test and average ordinate (AO) of field trials (A, B), as well as correlation between IT and Pi (C). *** denotes significance at $\alpha = 0.001$.

Supplementary Figure 3 Simple interval mapping of resistance to *Puccinia striiformis* in field trials (A) and seedling test (B, C). The x-axis shows the 21 wheat chromosomes. Positions are based on the genetic map, and the $-\log10(p)$ values of each marker are displayed on the y-axis (black line). The red horizontal line represents the significance thresholds. SI of the significant QTL detected in this study are colored in blue.

Supplementary Table 1 List of virulences and avirulences of *Puccinia striiformis* isolate Warrior + Yr27 used in field trials and seedling test. Brackets indicate ambiguous results due to the differing symptom ratings between replications or moderate susceptibility (based on Zetzsche et al. 2019)

Supplementary Table 2 Complete information of the QTL for stripe rust resistance in BMW population, evaluated in field trials (AO) and seedling test (IT and Pi).

Supplementary Table 3 Comparison of physical positions of the QTL identified in the present study (bold) with those reported previously.

Supplementary Table 4 List of gene annotations for peak markers ± 500,000 bp, shown as output retrieved from URGI database (https://wheat-urgi.versailles.inra.fr/Seq-Repository/Annotations)

3. Supplementary material of Chapter IV | General discussion

Table A.3-1 | List of RILs resistant against *Puccinia triticina* in at least two environments (location-year combinations), sorted in descending order.

RIL of BMWpop[a]	No. of environments	AO[b]	IT[c]	Pi[d]
2263	5	0.19	0.20	0.11
2268	5	0.26	0.25	0.05
2417	5	0.30	0.50	0.11
2059	5	0.98	0.60	1.15
2166	5	1.08	0.10	0.01
2241	5	1.14	0.60	0.76
2094	5	1.19	0.60	0.62
2291	5	2.05	0.55	0.40
2140	5	2.12	0.50	0.25
2258	5	2.30	2.10	3.07
2120	5	2.32	0.65	1.75
2164	5	2.36	0.55	0.01
2332	5	2.40	0.30	0.07
2170	5	2.71	2.90	3.87
2243	5	2.74	0.40	0.24
2248	5	2.92	3.50	3.60
2099	5	3.11	0.60	0.13
2086	5	3.19	0.30	0.00
2200	5	3.39	0.55	1.40
2492	5	3.42	3.50	5.82
2478	5	3.51	1.00	0.42
2449	5	3.70	0.65	1.80
2178	5	3.90	0.70	0.71
2334	5	3.93	0.30	0.31
2126	5	4.22	5.60	9.26
BAYP4535	5	4.54	0.45	1.14
2146	5	4.65	0.50	0.12
2168	5	4.70	4.80	9.22
2135	5	4.74	4.10	17.78
2384	5	4.88	6.10	16.24
2081	5	4.99	6.60	21.12
2042	5	5.02	6.30	15.25
2281	5	5.21	0.50	0.00
2266	5	5.23	3.20	3.06
2191	5	5.37	5.60	14.55
2494	5	5.38	4.50	6.41
2037	5	5.42	0.80	0.64
2234	5	5.42	6.10	13.09
2041	5	5.51	6.60	19.94
2267	5	5.52	4.80	4.49
2056	5	5.92	4.85	11.08
2255	5	5.94	7.20	14.84

RIL of BMWpop[a]	No. of environments	AO[b]	IT[c]	Pi[d]
2131	5	5.96	2.75	7.58
2175	5	6.01	4.60	13.32
2497	5	6.03	2.80	1.93
2512	5	6.10	0.70	0.27
2139	5	6.18	4.20	3.69
2100	5	6.33	2.10	1.42
2130	5	6.39	5.40	14.06
2296	5	6.41	0.45	0.21
2397	5	6.42	0.25	2.87
2195	5	6.44	5.35	3.55
2496	5	6.68	5.90	14.98
2013	5	6.78	7.70	16.63
2409	5	6.83	1.05	0.53
2302	5	6.86	1.10	1.68
2322	5	6.88	4.60	3.53
2439	5	6.88	3.31	1.42
2250	5	6.92	5.50	13.46
2224	5	6.95	0.35	0.11
2113	5	6.99	2.90	12.06
2027	5	7.11	1.20	0.71
2238	5	7.25	5.30	14.39
2328	5	7.25	3.40	6.05
2087	5	7.38	4.65	11.12
2167	5	7.42	4.10	9.92
2452	5	7.49	0.60	0.22
2387	5	7.57	4.10	6.53
2392	5	7.57	0.60	0.10
2366	5	7.68	2.40	7.15
2173	5	7.84	4.80	10.13
2134	5	7.88	5.20	3.78
2015	5	7.89	5.30	14.07
2209	5	7.90	1.30	2.36
2410	5	7.92	5.60	11.31
2515	5	8.03	1.70	1.24
2083	5	8.04	5.30	16.15
2048	5	8.18	5.50	12.14
2457	5	8.24	1.40	1.17
2336	5	8.37	6.40	16.51
2109	5	8.88	2.40	4.38
2363	5	8.96	5.60	15.48
2046	5	9.18	5.30	13.50
2358	5	9.40	1.40	4.00
2092	5	9.69	7.80	20.74
2008	5	9.78	5.20	14.30
2493	5	9.86	5.50	15.81
2309	5	9.88	4.30	2.25
2063	5	10.43	5.90	13.66

RIL of BMWpop[a]	No. of environments	AO[b]	IT[c]	Pi[d]
2445	5	10.98	6.70	16.56
2127	5	11.35	0.90	2.90
2190	4	0.07	0.45	0.18
2106	4	0.11	1.80	5.19
2082	4	0.28	0.40	0.90
2114	4	0.28	0.45	0.07
2090	4	0.42	0.65	0.56
2091	4	0.42	0.55	0.01
2498	4	0.46	0.55	2.19
2344	4	0.74	0.65	0.04
2372	4	0.74	0.10	0.12
2097	4	0.94	0.55	0.13
2442	4	1.16	0.60	0.56
2340	4	1.37	0.55	0.01
2108	4	1.41	0.30	0.36
2483	4	1.65	0.10	0.97
2077	4	2.89	0.60	0.48
2125	4	3.03	0.60	1.51
2045	4	3.19	7.00	16.09
2321	4	3.31	5.60	15.26
2275	4	4.13	5.60	8.42
2510	4	4.19	0.75	1.65
2347	4	4.29	6.20	13.04
2468	4	4.43	2.40	3.03
2323	4	4.77	0.85	0.21
2154	4	5.12	0.45	0.65
2047	4	5.14	4.80	4.56
2503	4	5.42	3.60	8.76
2376	4	5.52	5.50	12.05
2388	4	5.54	5.00	12.15
2165	4	5.75	3.70	10.02
2444	4	5.91	4.40	3.15
2354	4	5.97	3.10	6.67
2473	4	6.08	4.40	4.37
2276	4	6.14	6.90	15.07
2338	4	6.18	0.60	2.24
2075	4	6.40	3.90	1.98
2364	4	6.51	5.60	10.16
2112	4	6.56	5.00	5.62
2186	4	6.56	5.10	13.01
2284	4	6.62	5.40	12.52
Firl3565	4	6.63	5.80	8.81
2039	4	6.76	3.80	10.95
2425	4	6.85	1.05	0.44
2467	4	6.95	4.70	12.64
2413	4	6.97	0.25	0.10
2157	4	7.12	0.70	1.81

RIL of BMWpop[a]	No. of environments	AO[b]	IT[c]	Pi[d]
2071	4	7.15	5.20	15.09
2511	4	7.15	1.10	0.20
2237	4	7.24	5.30	12.80
2007	4	7.30	7.50	17.28
2310	4	7.43	0.70	0.59
2080	4	7.46	1.05	0.26
2304	4	7.53	1.60	1.53
2043	4	7.54	6.30	14.01
2356	4	7.55	6.70	18.19
2128	4	7.69	5.00	13.61
2447	4	7.72	0.70	0.05
2516	4	7.73	1.30	1.32
2485	4	7.93	0.70	1.62
2143	4	7.95	1.25	4.14
2156	4	7.99	3.40	2.60
2002	4	8.02	4.25	7.80
2141	4	8.04	4.80	8.47
2118	4	8.05	5.50	15.97
2489	4	8.15	5.10	11.82
2023	4	8.17	6.00	14.14
2219	4	8.23	2.25	3.10
2355	4	8.38	5.10	13.89
2145	4	8.48	4.90	10.41
2330	4	8.50	0.85	0.75
2353	4	8.54	5.60	13.51
2474	4	8.56	5.90	14.56
2095	4	8.58	0.75	0.40
2203	4	8.68	7.80	20.60
2038	4	8.70	0.50	0.10
2293	4	8.72	5.70	14.52
2272	4	8.82	5.90	12.93
2465	4	8.88	5.90	16.13
2438	4	8.90	4.80	9.67
2018	4	9.09	4.70	10.02
2394	4	9.21	0.90	0.15
2475	4	9.22	0.75	1.11
2230	4	9.27	0.85	1.32
2025	4	9.47	5.40	13.38
2220	4	9.48	3.70	1.45
2193	4	9.52	7.90	22.06
2031	4	9.65	5.70	15.99
2271	4	9.74	0.25	0.32
2205	4	9.75	6.30	14.97
2287	4	9.85	3.40	5.71
2052	4	9.90	2.70	8.95
2277	4	10.01	1.55	1.32
2507	4	10.05	5.60	13.50

RIL of BMWpop[a]	No. of environments	AO[b]	IT[c]	Pi[d]
2242	4	10.09	6.00	14.16
2369	4	10.21	3.10	1.78
2137	4	10.34	6.20	16.76
2179	4	10.55	2.90	3.22
2456	4	10.56	6.20	15.81
2504	4	10.86	6.20	14.87
2180	4	10.96	4.05	2.91
2231	4	11.23	4.70	7.76
2212	4	11.26	5.90	13.55
2034	4	11.28	3.70	10.86
2129	4	11.54	1.40	2.86
2024	4	12.31	3.90	6.76
2107	3	0.73	0.55	0.13
2202	3	3.82	8.00	16.02
2297	3	4.86	0.50	0.17
2264	3	5.01	5.90	13.34
2257	3	5.16	2.15	1.12
2361	3	5.37	6.40	13.77
2294	3	5.71	5.50	13.48
2121	3	5.90	4.50	15.93
2246	3	5.98	5.50	5.16
2054	3	6.02	5.30	9.72
2279	3	6.33	0.90	2.81
2217	3	6.38	0.70	1.12
2365	3	6.39	6.40	15.54
2124	3	6.61	6.10	13.39
2206	3	6.94	5.20	19.57
2443	3	6.97	0.60	0.00
2229	3	7.03	6.10	15.75
2028	3	7.15	6.50	11.09
2481	3	7.21	0.65	0.03
2490	3	7.21	4.70	14.39
2454	3	7.36	0.55	0.01
Potenzial	3	7.45	2.40	7.70
2245	3	7.52	4.80	3.69
2288	3	7.55	0.50	0.02
2207	3	7.57	2.05	0.78
2315	3	7.62	3.15	4.35
2396	3	7.72	4.60	12.12
2299	3	7.74	3.85	4.91
2003	3	7.78	5.30	14.09
2337	3	8.04	6.90	15.06
2368	3	8.06	7.20	18.14
2441	3	8.11	2.00	2.06
2435	3	8.13	1.50	2.24
2393	3	8.15	1.75	1.61
2265	3	8.20	6.20	11.60

RIL of BMWpop[a]	No. of environments	AO[b]	IT[c]	Pi[d]
2116	3	8.29	6.30	17.08
2352	3	8.34	6.60	14.25
2184	3	8.37	7.20	16.96
2252	3	8.37	8.40	21.56
2005	3	8.39	5.40	10.19
2429	3	8.55	0.70	1.54
2236	3	8.57	5.90	15.95
2030	3	8.63	6.10	18.05
2073	3	8.63	5.00	13.06
2218	3	8.71	2.40	3.26
2329	3	8.90	4.40	8.66
Ambition	3	9.01	7.10	23.21
2461	3	9.04	4.35	4.16
2398	3	9.06	6.30	15.17
2283	3	9.13	4.90	10.23
Event	3	9.16	1.15	3.12
2326	3	9.29	0.80	1.23
2437	3	9.31	0.65	0.02
Julius	3	9.35	1.30	1.34
2153	3	9.59	0.10	0.05
2286	3	9.71	0.65	1.50
2233	3	9.79	5.90	15.14
2111	3	9.98	6.70	19.39
2285	3	10.12	0.40	0.61
2320	3	10.20	2.60	2.97
2377	3	10.28	6.00	10.86
2020	3	10.29	6.00	10.76
2172	3	10.30	4.90	13.11
2162	3	10.51	4.60	11.28
2401	3	10.61	0.70	1.28
2261	3	10.72	3.50	2.93
2004	3	10.74	7.20	19.07
2386	3	10.92	7.20	15.73
2351	3	11.17	5.70	11.64
2226	3	11.25	1.50	0.85
2424	3	11.31	0.90	2.83
2244	3	12.33	3.60	10.84
2096	3	12.62	6.30	16.70
2274	3	12.88	2.25	4.78
2469	3	12.94	4.50	4.36
2472	3	13.16	3.30	3.90
2051	3	13.43	4.20	10.17
2455	2	3.15	1.00	3.38
2240	2	4.12	5.50	15.83
2152	2	4.42	0.60	0.17
2132	2	4.75	6.10	28.27
2459	2	5.13	2.20	1.75

RIL of BMWpop[a]	No. of environments	AO[b]	IT[c]	Pi[d]
2148	2	5.19	1.00	3.28
2314	2	5.39	5.70	9.35
2509	2	5.82	0.85	0.94
2325	2	6.00	0.75	0.21
2177	2	6.21	2.80	5.69
2029	2	7.26	1.20	0.71
2213	2	7.36	6.40	18.09
2446	2	7.39	6.00	14.49
2362	2	7.46	6.60	15.51
2360	2	7.72	6.70	18.22
2182	2	7.76	5.30	14.13
2144	2	7.82	1.70	0.95
2001	2	8.07	5.40	9.84
2151	2	8.10	4.25	10.79
2292	2	8.19	5.40	5.43
2421	2	8.21	6.10	13.32
2176	2	8.23	8.40	23.08
2036	2	8.37	4.30	7.58
2232	2	8.78	6.40	16.36
2300	2	8.81	5.60	22.44
2216	2	8.88	6.80	17.84
2440	2	8.91	4.35	3.90
2373	2	9.31	5.70	17.88
2426	2	9.32	1.50	3.33
2204	2	9.56	2.50	2.64
2197	2	9.58	2.40	0.76
2136	2	9.65	6.90	20.31
2305	2	9.86	2.00	3.87
2223	2	9.90	7.60	18.66
2066	2	9.98	7.50	17.79
2133	2	10.05	7.70	16.29
2402	2	10.13	7.20	18.84
2270	2	10.21	0.40	0.15
2278	2	10.26	5.90	20.06
2280	2	10.27	0.70	1.07
2289	2	10.27	1.75	0.97
2249	2	10.32	4.50	8.80
2423	2	10.35	0.35	0.01
2342	2	10.43	4.00	3.62
2044	2	10.52	5.60	20.04
2032	2	10.64	6.20	16.10
2399	2	10.67	7.10	18.99
2269	2	10.80	6.70	16.26
2026	2	10.88	3.70	6.87
2006	2	11.00	5.60	10.31
2295	2	11.08	6.30	18.36
2508	2	11.16	4.00	9.45

RIL of BMWpop[a]	No. of environments	AO[b]	IT[c]	Pi[d]
2098	2	11.19	7.10	16.26
2192	2	11.30	5.70	21.00
2061	2	11.53	8.60	20.24
2477	2	11.74	6.70	17.91
2247	2	12.22	4.30	3.70
2119	2	12.37	7.10	16.42
2419	2	12.69	4.45	15.19
2513	2	12.87	1.70	2.77
2406	2	13.03	4.30	11.85
2470	2	13.05	5.10	12.48
2282	2	13.42	1.65	0.47
2196	2	13.46	6.60	21.90
2378	2	13.67	6.20	17.43

[a] RILs highlighted in green were resistant in field trials and seedling tests. RILs in **bold** were resistant against both *Puccinia triticina* and *Puccinia striiformis* f. sp. *tritici*

[b] Mean value of the average ordinate (field trials) over the number of environments displayed in column 2

[c] Mean value of the visually evaluated infection type (seedling test) over five replications

[d] Mean value of the percentage of infected leaf area (seedling test) over five replications

Table IV.3-2 | List of RILs resistant against *Puccinia striiformis* f. sp. *tritici* in at least two environments (location-year combinations), sorted in descending order.

RIL of BMWpop[a]	No. of environments	AO[b]	IT[c]	Pi[d]
2175	6	0.37	0.63	0.00
2263	6	0.40	0.56	0.03
2250	6	0.44	0.50	0.00
2281	6	0.48	0.44	0.00
2516	6	0.48	0.56	0.00
2393	6	0.52	0.69	0.00
2469	6	0.52	0.56	0.00
2146	6	0.53	0.69	0.00
2154	6	0.53	0.31	0.00
2264	6	0.53	0.50	0.00
2326	6	0.55	0.56	0.00
2013	6	0.56	0.50	0.00
2288	6	0.56	0.44	0.01
2364	6	0.57	0.69	0.05
2202	6	0.58	0.63	0.01
2065	6	0.60	0.69	0.13
2166	6	0.61	0.75	0.03
2167	6	0.61	0.38	0.00
2467	6	0.61	0.13	0.00
2284	6	0.62	2.31	0.61
2296	6	0.62	0.25	0.02
2333	6	0.62	0.50	0.00

RIL of BMWpop[a]	No. of environments	AO[b]	IT[c]	Pi[d]
2452	6	0.62	0.63	1.08
2261	6	0.64	0.38	0.05
2262	6	0.64	0.44	0.00
BAYP4535	6	0.64	0.63	0.02
2177	6	0.65	1.56	0.12
2135	6	0.66	0.69	0.23
2173	6	0.66	0.56	0.00
2188	6	0.66	1.88	0.45
2289	6	0.66	0.44	0.03
2493	6	0.66	0.56	0.00
2140	6	0.68	0.75	0.03
2179	6	0.68	0.56	0.04
2168	6	0.69	0.38	0.00
2256	6	0.69	0.56	0.00
2427	6	0.69	0.69	0.02
2041	6	0.70	0.38	0.12
2088	6	0.70	0.88	0.06
2156	6	0.70	0.56	0.02
2192	6	0.71	0.38	0.00
2204	6	0.71	0.56	0.00
2508	6	0.72	0.44	0.00
2229	6	0.73	1.88	0.33
2325	6	0.74	0.38	0.00
2421	6	0.74	0.38	0.00
2141	6	0.75	0.38	0.02
2353	6	0.75	0.75	0.09
2386	6	0.75	0.63	0.03
2089	6	0.76	0.56	0.02
2504	6	0.77	0.44	0.00
2181	6	0.78	0.75	0.00
2028	6	0.79	0.81	0.03
2245	6	0.79	1.44	0.07
2398	6	0.79	0.31	0.00
2437	6	0.79	0.50	0.00
2151	6	0.80	1.56	0.12
2176	6	0.80	0.75	0.14
2494	6	0.80	0.38	0.00
2182	6	0.81	1.00	0.08
2422	6	0.81	0.63	0.03
2476	6	0.81	0.75	0.12
2457	6	0.82	0.50	0.00
2083	6	0.83	0.75	0.03
2294	6	0.83	0.38	0.00
2136	6	0.84	0.56	0.37
2336	6	0.84	0.63	0.00
2454	6	0.84	0.19	0.00
2269	6	0.85[b]	0.69	0.12

RIL of BMWpop[a]	No. of environments	AO[b]	IT[c]	Pi[d]
2205	6	0.86	0.56	0.00
2153	6	0.87	0.13	0.00
2449	6	0.87	0.56	0.00
2124	6	0.88	0.50	0.01
2165	6	0.88	1.56	0.21
2419	6	0.89	0.31	0.00
2223	6	0.90	0.38	0.00
2507	6	0.91	0.81	0.09
2119	6	0.92	0.25	0.00
2428	6	0.92	0.50	0.02
2515	6	0.92	0.69	0.00
2071	6	0.93	2.31	0.51
2226	6	0.93	0.69	0.31
2472	6	0.94	0.50	0.01
2034	6	0.95	0.56	0.00
2180	6	0.95	0.56	0.01
2255	6	0.96	0.63	0.00
2295	6	0.96	0.56	0.00
2392	6	0.96	5.13	3.01
2510	6	0.96	0.44	0.00
2109	6	0.97	0.69	0.00
2164	6	0.97	0.69	0.00
2187	6	0.97	1.69	0.36
2208	6	0.97	0.25	0.00
2212	6	0.97	1.31	0.18
2440	6	0.97	0.63	0.00
2023	6	0.98	0.56	0.00
2139	6	0.98	0.69	1.20
2365	6	0.99	0.69	0.00
2130	6	1.00	0.50	0.00
2238	6	1.00	0.63	0.00
2293	6	1.00	0.69	0.00
2069	6	1.01	0.38	0.00
2231	6	1.01	1.19	0.03
2438	6	1.01	0.56	0.01
2039	6	1.02	1.56	0.62
2234	6	1.02	0.69	0.00
2277	6	1.02	0.31	0.00
2423	6	1.02	0.56	0.00
2126	6	1.04	0.56	0.02
2144	6	1.05	2.31	0.76
2443	6	1.05	0.38	0.04
2465	6	1.05	0.69	0.00
2506	6	1.05	0.56	0.00
2222	6	1.07	0.31	0.00
2274	6	1.07	0.63	0.00
2399	6	1.07[b]	0.75	0.00

RIL of BMWpop[a]	No. of environments	AO[b]	IT[c]	Pi[d]
2051	6	1.08	0.94	0.19
2197	6	1.08	0.56	3.60
2058	6	1.10	0.75	0.30
2441	6	1.10	0.75	0.01
2361	6	1.11	1.63	0.43
2475	6	1.11	0.50	0.00
2094	6	1.12	2.38	0.81
2066	6	1.13	0.44	0.00
2162	6	1.13	1.94	2.81
2470	6	1.14	0.75	0.00
2025	6	1.15	0.38	0.00
2063	6	1.15	1.44	0.44
2087	6	1.16	0.44	0.00
2152	6	1.17	1.06	0.05
2015	6	1.18	0.56	0.08
2243	6	1.18	0.63	0.00
2458	6	1.18	0.38	0.03
2098	6	1.20	0.94	0.08
2099	6	1.20	0.50	0.01
2300	6	1.20	0.50	0.00
2447	6	1.20	0.50	0.18
2455	6	1.20	0.50	0.02
2199	6	1.21	0.75	1.29
2268	6	1.22	0.38	0.00
2059	6	1.23	1.38	0.14
2006	6	1.24	1.31	0.13
2178	6	1.24	0.69	0.00
2286	6	1.25	0.75	0.10
2512	6	1.25	0.69	0.20
2081	6	1.26	3.25	3.19
2219	6	1.27	0.56	0.00
Potenzial	6	1.27	0.44	0.17
2031	6	1.28	0.56	0.00
2492	6	1.28	0.69	0.02
2373	6	1.29	0.50	0.66
2497	6	1.30	0.63	0.00
2207	6	1.31	0.88	0.07
2312	6	1.31	0.56	0.00
2496	6	1.34	0.69	0.00
2128	6	1.36	0.50	0.01
2342	6	1.36	1.00	0.02
2409	6	1.36	1.00	0.08
2417	6	1.36	1.44	0.17
2191	6	1.38	0.63	0.05
2360	6	1.40	0.50	0.00
2413	6	1.40	1.56	0.12
2215	6	1.41[b]	1.50	0.19

RIL of BMWpop[a]	No. of environments	AO[b]	IT[c]	Pi[d]
2446	6	1.41	0.38	0.00
2240	6	1.42	0.56	0.06
2459	6	1.42	1.94	1.75
2077	6	1.43	0.38	0.00
2163	6	1.43	0.63	0.01
2048	6	1.45	0.69	0.06
2514	6	1.46	1.75	0.36
2265	6	1.47	0.94	1.90
2032	6	1.48	0.56	0.39
2061	6	1.48	0.75	0.26
2211	6	1.49	0.75	0.03
2237	6	1.49	0.63	0.03
2456	6	1.50	0.56	0.00
2451	6	1.51	0.63	0.01
2155	6	1.52	0.56	0.00
2275	6	1.56	0.56	0.00
2445	6	1.59	0.88	0.21
2394	6	1.62	2.00	1.21
2448	6	1.62	0.44	0.00
2272	6	1.63	0.38	0.00
Bussard	6	1.63	1.50	0.23
2131	6	1.64	0.44	0.00
2196	6	1.64	0.56	0.00
2022	6	1.65	0.69	0.00
2213	6	1.68	2.33	1.66
2280	6	1.68	1.19	0.30
2267	6	1.71	2.19	0.37
2276	6	1.71	0.63	0.00
2330	6	1.71	0.44	0.00
2290	6	1.73	1.00	0.03
2424	6	1.74	1.38	0.43
2251	6	1.75	0.63	0.01
2477	6	1.75	0.63	0.00
2331	6	1.78	2.44	4.61
2258	6	1.82	0.63	0.00
2228	6	1.84	3.06	0.54
2236	6	1.85	0.75	0.03
2145	6	1.89	0.13	0.00
2358	6	1.89	3.13	2.48
2002	6	1.94	0.50	4.72
2062	6	1.94	1.06	0.05
2367	6	1.97	0.81	0.02
Format	6	2.14	0.88	0.06
2387	6	2.15	2.50	1.09
2118	6	2.16	1.25	0.10
2172	6	2.16	0.31	0.04
2395	6	2.17[b]	1.44	0.72

RIL of BMWpop[a]	No. of environments	AO[b]	IT[c]	Pi[d]
2241	6	2.21	1.00	0.15
2242	6	2.22	0.63	0.00
2282	6	2.26	0.58	0.01
2038	6	2.27	1.50	0.16
2397	6	2.28	0.50	0.00
2200	6	2.30	1.44	0.12
2206	6	2.30	0.56	0.04
2198	6	2.31	0.31	0.00
2414	6	2.36	0.38	0.01
2509	6	2.38	1.94	1.02
2209	6	2.43	0.75	0.02
2186	6	2.57	1.44	0.45
2435	6	2.57	2.19	0.35
2003	6	2.58	5.00	4.55
2043	6	2.62	0.69	0.00
2354	6	2.63	6.00	4.34
2024	6	2.65	0.88	0.20
2073	6	2.71	2.25	0.37
2216	6	2.85	1.88	0.31
2426	6	2.95	1.25	0.21
2244	6	3.02	0.50	0.00
2001	6	3.09	0.69	0.00
2384	6	3.09	0.75	0.19
2402	6	3.09	1.38	0.14
2453	6	3.17	3.88	1.99
2362	6	3.26	0.63	0.07
2220	6	3.28	3.13	2.23
2026	6	3.46	2.13	0.19
Ambition	6	3.60	0.75	0.03
2328	6	3.62	0.63	0.00
2266	6	3.63	0.75	0.03
2170	6	3.93	0.75	0.00
2401	6	4.09	2.19	0.72
2217	6	4.16	0.81	0.00
2292	6	4.17	1.50	0.46
2503	6	4.20	2.25	0.62
2148	6	4.25	0.50	0.00
2511	6	4.33	1.17	0.00
Firl3565	6	4.67	3.69	1.05
2232	6	4.81	5.25	2.24
2020	6	4.82	1.44	0.08
Julius	6	5.13	1.50	0.22
2338	6	5.92	3.94	1.96
2439	6	5.99	2.06	1.63
2311	6	6.86	1.63	0.33
2097	6	7.06	4.50	2.42

RIL of BMWpop[a]	No. of environments	AO[b]	IT[c]	Pi[d]
2444	6	7.26	3.75	1.30
2060	5	0.24	1.25	0.29
2033	5	0.48	0.81	0.01
2239	5	0.57	0.88	0.14
2143	5	0.95	0.50	0.00
2195	5	1.15	1.50	0.43
2111	5	1.34	4.31	2.31
2036	5	1.77	0.38	0.01
2430	5	1.83	2.25	1.20
2029	5	1.99	0.69	0.00
2490	5	1.99	1.63	0.25
2418	5	2.04	0.56	0.01
2142	5	2.15	2.94	0.78
2227	5	2.64	4.25	2.61
2030	5	2.67	3.25	0.56
2310	5	2.74	0.75	0.52
2329	5	2.79	0.75	0.00
2468	5	2.97	0.94	0.40
2323	5	3.02	0.69	0.01
2218	5	3.20	3.00	1.00
2327	5	3.24	1.63	0.85
2481	5	3.39	1.50	0.35
2278	5	3.58	0.56	0.05
2485	5	3.67	0.69	0.00
2324	5	3.68	1.38	0.47
2306	5	3.88	0.69	0.00
2505	5	3.93	2.44	0.39
2461	5	3.97	2.69	0.56
2055	5	4.08	0.69	0.00
2489	5	4.11	2.69	0.66
2133	5	4.14	1.00	0.12
2478	5	4.38	0.81	0.06
2285	5	4.46	4.94	2.83
2350	5	4.64	1.94	0.58
2193	5	4.67	2.13	0.60
2368	5	4.84	4.50	2.28
2012	5	5.07	2.38	0.82
2320	5	5.09	0.69	0.72
2037	5	5.22	4.25	2.15
2137	5	6.19	0.75	0.02
2355	5	6.77	3.63	3.82
2376	5	6.96	5.13	7.36
2127	5	7.38	4.38	3.42
2283	5	8.18	6.00	4.07
2009	5	8.23	7.50	5.88
2248	5	8.41	0.81	0.25
2322	5	8.49[b]	4.25[c]	2.45

RIL of BMWpop[a]	No. of environments	AO[b]	IT[c]	Pi[d]
2132	5	10.27	6.38	11.07
2334	5	11.88	6.00	7.40
2471	4	1.23	0.69	0.04
2363	4	1.41	1.56	0.41
2184	4	1.93	6.00	3.66
2271	4	3.69	5.25	3.93
2050	4	4.05	3.69	1.22
2287	4	4.28	0.50	0.00
2203	4	4.48	5.25	7.01
2080	4	4.67	0.44	0.00
2042	4	4.90	4.25	4.07
2252	4	5.07	2.50	0.77
2270	4	5.09	0.63	0.01
2247	4	5.32	1.50	0.23
2309	4	5.83	3.56	2.52
2406	4	6.49	1.50	1.23
2100	4	6.76	4.25	4.03
2129	4	6.91	2.19	0.52
2513	4	7.25	0.63	0.00
2230	4	7.54	0.81	0.12
2190	4	7.64	0.63	0.08
2348	4	8.21	4.50	3.63
2351	4	8.38	5.25	1.03
2157	4	8.53	1.38	0.16
2233	4	8.96	3.75	1.75
2356	4	10.61	4.13	5.18
2120	4	11.64	3.25	5.34
2008	3	3.61	4.38	3.57
Event	3	3.73	1.44	0.35
2432	3	4.37	1.58	0.04
2464	3	4.76	2.19	0.28
2474	3	4.90	2.13	0.84
2304	3	5.57	1.50	1.75
2113	3	5.60	3.25	1.89
2369	3	5.79	4.75	3.76
2429	3	7.20	1.94	0.59
2068	3	7.74	2.88	0.03
2112	3	7.88	5.00	5.68
2095	3	8.42	3.50	1.26
2396	3	8.53	3.00	1.72
2046	3	8.76	2.88	1.92
2305	3	9.16	3.63	6.93
2134	3	9.70	4.67	1.85
2299	3	9.85	3.38	3.34
2378	3	9.91	5.50	5.14
2366	3	10.15	7.25	5.97
2347	3	10.45	6.75	8.14

RIL of BMWpop[a]	No. of environments	AO[b]	IT[c]	Pi[d]
2121	3	10.86	2.75	2.83
2005	3	10.89	4.25	2.04
2307	2	2.41	1.81	0.45
2372	2	3.31	1.75	0.56
2344	2	4.04	1.44	0.09
2075	2	5.60	2.38	2.75
2249	2	5.71	2.31	2.61
2410	2	8.04	5.67	2.52
2340	2	8.14	0.81	0.01
2044	2	9.27	1.25	0.32
2027	2	10.73	2.44	1.33
2096	2	11.77	1.56	0.52
2224	2	11.79	1.44	0.69

[a] RILs highlighted in green were resistant in field trials and seedling tests. RILs in **bold** were resistant against both *Puccinia striiformis* f. sp. *tritici* and *Puccinia triticina*

[b] Mean value of the average ordinate (field trials) over the number of environments displayed in column 2

[c] Mean value of the visually evaluated infection type (seedling test) over four replications

[d] Mean value of the percentage of infected leaf area (seedling test) over four replications

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ERKLÄRUNG

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S. Roller