Sources of resistance to yellow rust and stem rust in wheat-alien introgressions

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ABSTRACT

Wheat is the staple food and the main source of caloric intake in most developing countries, and thereby an important source in order to maintain food security for the growing populations in those countries. Stem rust *Puccinia graminis* f. sp. *tritici*, and yellow rust *P. striiformis* f. sp. *tritici* of wheat continues to cause severe damage locally and globally, thereby contributing to food insecurity. In this paper biology and taxonomy of stem rust and yellow rust, breeding for resistance, utilization of resistance sources from different gene pools, molecular characterization and genetic dissection of resistance to rusts are discussed.

PREFACE

Plant breeding is an important tool, as it contributes to providing improved knowledge when biotic and abiotic factors are addressed, where the food security is searched for by humans in a changing world. Thus, wheat breeding play an essential role in developing modern cultivars those are adapted to current and future adverse environments. Wheat-alien introgressions have been utilized and are playing an important role, through the fact that alien genomes have contributed with several desirable donor genes for wheat improvement.

This introductory paper reviews the opportunities to obtain resistances to stem rust and yellow rust, and also possible applications of molecular tools as well as of QTL analysis. The paper thereby tries to cover, the most recent advances and achievement applied in wheat breeding's to fight stem rust and yellow rust, the two most destructive diseases of wheat worldwide.

CONTENTS

INTRODUCTION

Wheat is one of the most important and significant cereal staple food crops in the world, both in terms of food production and for providing the total amount of food calories and protein in the human diet (Gupta et al., 2008). However, wheat production is constrained by various wheat diseases caused by fungal, bacterial, and viral pathogens. Of these, diseases caused by the rust fungi have since long been a major concern and problem for breeders, farmers and commercial seed companies (Wiese, 1977). Rust diseases of wheat are among the oldest known diseases and are important worldwide (Singh et al., 2005). Globally, yellow rust (*Puccinia striiformis* f. sp. *tritici*), stem rust (*Puccinia graminis* f. sp. *tritici*), and leaf rust (*Puccinia triticina*) are the most damaging diseases of wheat and other small grain cereals (Roelfs et al., 1992). Historically, yellow rust has caused and is presently causing significant and severe losses on susceptible wheat cultivars worldwide (Wellings, 2011). Moreover, the recent detection of the widely virulent race Ug99 in Uganda in 1998 challenged the misconception that stem rust was a conquered disease (Singh et al., 2006; 2008a). Now, up to 90% of world's wheat cultivars are considered stem rust susceptible (Singh et al., 2006; 2011), and the disease is threatening 120 million tons or 20% of the world's wheat in Central and North Africa, the Middle East and Asia, with a population of more than one billion people (Dixon et al., 2009).

To date more than sixty race-specific resistance genes (i.e. *Yr* genes) have been described for yellow rust, and more than fifty different stem rust resistance genes (*Sr* genes) have been genetically characterized and named (McIntosh et al., 1995; 2001; 2010; 2011). Due to the frequent emergence of new yellow rust and stem rust races, efforts to identify potentially new sources of effective resistance genes are of the highest importance. New sources of resistance genes can be obtained from various sources in the primary, secondary and tertiary gene pools of wheat. One promising source of new genes for wheat is the tertiary gene pool, which includes *Secale cereale*, *Agropyron* spp., *Leymus spp. Thinopyrum* spp. and *Hordeum vulgare* (Mujeeb-Kazi, 2006; Dundas et al., 2007). Rye has been among the most successfully used alien resources contributing against biotic and abiotic stresses for wheat (McIntosh, 1991; Dvorak and McGuire, 1991; Jiang et al., 1994; Stephen et al., 1995). Many promising traits, particular disease and pest resistances, yield and adaptation have been localized on the seven rye chromosomes (Rabinovich, 1998; Schlegel et al., 1998; Miroslaw and Chelkowski, 2004). Therefore, wheat-rye translocations/substitutions have been widely used in international and regional breeding programs (Rabinovich, 1998; Miroslaw and Chelkowski, 2004). Also,

Thinopyrum and *Leymus*, species within the wheatgrass genus, are rich sources of genes for wheat breeding and improvement. These grasses have shown resistance to diseases such as leaf and stem rust and powdery mildew etc. in its natural populations in different environments (McIntosh 1991; Merker and Lantai, 1996; Chen et al. 1999; Ellneskog-Staam and Merker, 2002a; 2002b). Thus, new and useful sources of disease and pest resistance are abundantly available in the wild relatives of hexaploid wheat (Zaharieva et al. 2001).

Global Economic Importance of wheat

Wheat plays an important role in everyday life of the world's population and provides over 21% of the food calories and 20% of the protein to more than 4.5 billion people, thereby playing a fundamental role in food security (Braun et al., 2010). Wheat was one of the miracle crops of the 20th Century playing a significant role in the Green Revolution led by Norman Borlaug, which dramatically reduced poverty, hunger and saved millions of lives worldwide (CIMMYT and ICARDA, 2011). The demands for food production will continually be expanding, in order to produce enough food to feed the growing world population (OECD-FAO, 2009). Due to land limitations, the enhancement of wheat production must come from higher absolute yields, which can only be met by the concerted action of scientists involved in diverse agricultural disciplines and in particular by increased efforts in plant breeding (Braun et al., 1998). In the future, the nutritional value of the world wheat supply will become extremely crucial, because the world demand for wheat as a source of calories and protein is increasing simultaneously, as the world wheat stocks continue to decrease (Dixon et al., 2009; FAO, 2009). The global wheat production has been increased largely over the past decades due to plant breeding research and improved production and reached 676 million tonnes in 2011 (http://www.fao.org/news/story/en/item/53813/icode/). Currently, the worldwide population is over 7 billion people and expected to reach more than 9 billion by 2050 (http://www.fao.org/fileadmin/templates/wsfs/docs/expert_paper/How_to_Feed_the_World_in_2050.pdf). Moreover, the worldwide demand for wheat in the developing world is projected to increase with 60% by 2050 and due to climate change a decrease of wheat production by 20-30% is expected, particularly in developing countries (Braun, 2011). Therefore, further research and breeding are necessary to develop high yielding wheat cultivars resistant to diseases, pests and abiotic stresses to secure wheat production.

BIOLOGY AND TAXONOMY OF STEM RUST AND YELLOW RUST

Stem Rust

Stem rust, caused by the fungus *Puccinia graminis*, is a serious disease of wheat, oats, barley, and rye, as well as of many cereal wild grasses (Kurt et al., 2005). The first detailed reports about the wheat stem rust fungus was given by Fontana, (1932) and Tozzetti, (1952). According to Chester (1946) the stem rust was named *Puccinia graminis* by Persoon in 1797. The proof of heteroecism (development of different stages of a parasitic species on various host plants) of *Puccinia graminis* on cereals and barberry was reported in 1880 and 1866 (Walker, 1976). Later, Chester, (1946) provided one of the first detailed publications on the rust diseases. Initially, the wheat leaf rust pathogen was not distinguished from the stem rust pathogen, however in (1815) de Candolle discovered that the leaf rust is caused by the fungus *Uredo rubigo-vera* (Chester, 1946; McIntosh et al., 1995; Singh et al., 2002). In 1894 Eriksson in Sweden defined *formae speciales* in order to reflect "special forms" of the wheat stem rust and yellow rust pathogens which showed specialization on different host species (Walker, 1976; McIntosh et al., 1995). Pandemic outbreaks of stem rust have been reported throughout history with significant events occurring in Southern Asia, China, Central Asia, Eastern and Central Europe, Northern America and elsewhere in the past 130 years (Saari and Prescott, 1985; Roelfs et al., 1992). The Nobel Prize Laureate Dr. Norman Borlaug led the battle against wheat stem rust that threatened farmers in Mexico. Through breeding of new wheat cultivars which were resistant to causative pathogens Dr. Norman Borlaug spurred a Green Revolution in wheat production, one of the greatest milestones in the history of world agriculture (Stokstad, 2009; Hovmøller et al., 2010). However in 1999, the race of Ug99 (TTKSK) was first identified in Uganda, to which most commercial wheat cultivars and breeding lines worldwide are susceptible (Pretorius et al., 2000; Singh, et al., 2006; 2011).

Taxonomy and Life Cycle of Stem Rust

Stem rust (black rust) is caused by the fungus *Puccinia graminis* which belongs to the phylum Basidiomycota, class Urediniomycetes, order Uredinales, and family Pucciniaceae. It contains 17 genera and approximately 4121 species, of which the majority belongs to the genus Puccinia (Kirk et al., 2008). For the fungus *Puccinia graminis* f. sp. *tritici* the primary host is wheat and the barberry is the main alternate host. The life cycle of *Puccinia graminis* f. sp. *tritici* mostly consists of continual uredinial generations (Singh et al., 2002). The fungus develops teliospores on the wheat plant in order to produce a secondary spore called basidiospore. The stem rust life cycle of *Puccinia graminis* f. sp. *tritici* occurs in the following

stages: basidiospores → pycniospores → aeciospores → urediniospores → teliospores (Roelfs, 1985a). The disease cycle starts with exposure of the new wheat crop to stem rust inoculum, and the sources of inoculum are different in different areas (Leonard, 2001; Leonard and Szabo, 2005). In warm areas, the wheat is planted in late fall and harvested in early summer, thereby the infected volunteer wheat plants serve as a green bridge or source of the primary inoculum to start a new cycle next fall. In the areas with cold winter, aeciospores are the main source of the primary inoculum for the wheat stem rust (Leonard and Szabo, 2005). The role of barberry plant is to serve as a source of the primary inoculum of *Puccinia graminis* f. sp. *tritici*. The fungus produces black thick walled, diploid teliospores and later produces haploid basidiospores. The basidiospores infect the barberry plant and produce a haploid mycelium. Thereafter from mycelium the pycnial will be formed and pycniospores are produced. Moreover, the aeciospores are released in the spring and infect the wheat plants (Roelfs, 1985a; Agrios, 2005) (Fig. 1).

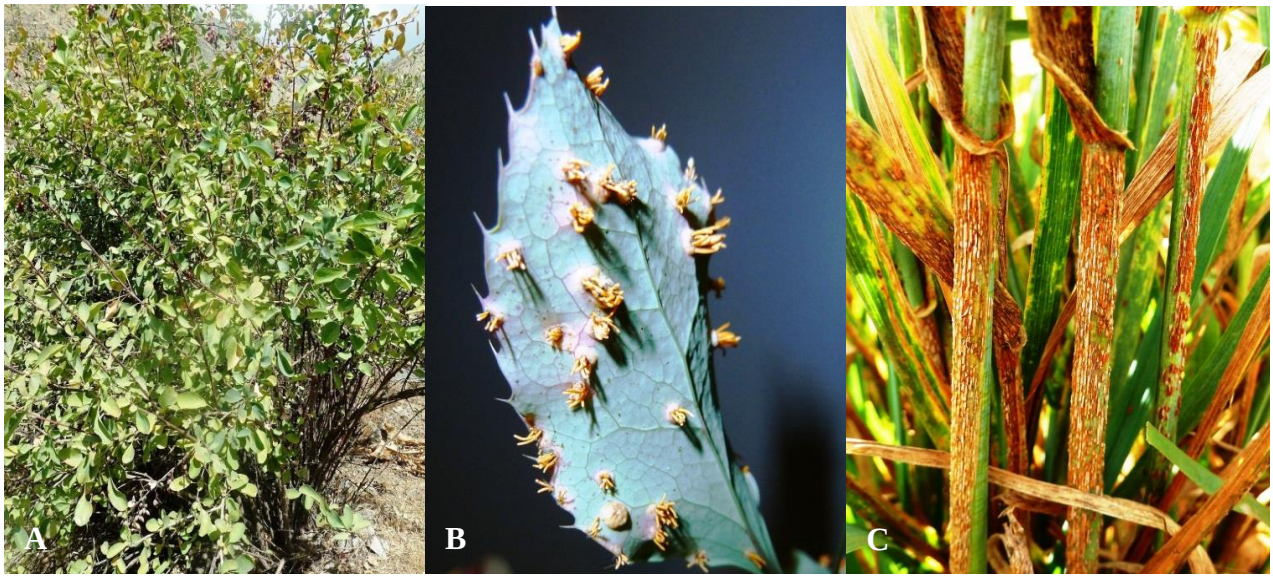

Figure 1. Symptoms *of Puccinia graminis* f. sp. *tritici* A) infected barberry bush in Tajikistan; B) Aecial infection in barberry leaf; C) infected wheat plants. *Photo: Mahbubjon Rahmatov*

Economic Importance

Stem rust is considered as the most destructive disease of wheat. The losses may reach 100% on susceptible wheat cultivars when conditions are favorable for the disease (Singh, et al., 2002). Stem rust can cause great damage to susceptible wheat crops over a broad number of geographical regions worldwide. A healthy crop before harvest can be destroyed by stem rust fungus, if sufficient inoculum arrives from infected fields. The nutrient flow in the plant is

interrupted at a severe infection on the stems leading to shriveling of spikes and grain. Besides that, infected stems are weakened, and therefore prone to lodging, leading to further loss of grain (Roelfs et al., 1992; Leonard and Szabo, 2005).

Epidemics of Stem Rust

Classical studies of epidemics of plant disease have been performed worldwide, including many examples involving the cereal rust diseases (Roelfs, 1985b). Historically, the first studies of plant disease epidemics on a regional basis took place when the wheat stem rust epidemics occurred in the United States in 1923 and 1925 (Stakman and Harrar, 1957; Roelfs, 1985b). The studies by Stakman and his colleagues became the concept for the area of phytopathology known as epidemiology (Roelfs, 1985b). The wheat stem rust was and still is the most severe disease of wheat and brings destructive damage on a periodic basis. Several, major wheat stem rust epidemics have occurred in the 20th century, leading to development of significant national and international mitigation and control efforts for rusts. Severe epidemics of stem rust occurred, with yield losses of 5-20% in Eastern and Central Europe in 1932, and 9-33% in Scandinavia in 1951 (Zadoks, 1963). The significant epidemics of stem rust in the first half of the 20th century led to massive damages across continents. Epidemics were also recorded in Central India in 1946-1947, estimated losses were 2 million tonnes or 20% of total production; Eastern Europe and Russia in 1932, losses 5-20%; North America in 1904 and 1954, a series of 5-6 devastating epidemics with losses from 1.3 to 3.7 million tonnes per epidemics; Mexico in 1947 – 1948, estimated losses 30%; Chile in 1951 40% losses; Australia in 1947 – 1948 losses 270 thousand tonnes in the warmer areas of Queensland and northern New South Wales (Roelfs et al.1992; Hodson, 2011). The stem rust epidemics were also the driver behind the breeding programs initiating the Green Revolution in 1960-1970. Stem rust resistance genes have been incorporated successfully into high yielding semi-dwarf wheat cultivars, with significant reduction of incidences of the stem rust disease globally (Hodson, 2011). The joint mitigation actions have played a great role for the global reduction of stem rust to near insignificant levels in the last 20-30 years (Hodson, 2011). However, in 1999 a new race of stem rust, Ug99, also called TTKSK was reported in central Africa, which is suggested as a major threat to the global wheat production (Pretorius, et al. 2000; Singh et al., 2008a).

Detection and Movement of Race Ug99/TTKSK

The widely virulent stem rust pathogen, Ug99 (aka isolate TTKSK), appeared in 1998 in Uganda and was classified in 1999 (Pretorius et al., 2000). It was designated as TTKS by

Wanyera et al., (2006), using the North American nomenclature system (Roelfs and Martens, 1988; Singh et al., 2006). When the fifth set of differential genotypes was added to further expand the characterization of the race, it was renamed to TTKSK (Jin et al., 2008). The race of Ug99/TTKSK has shown virulence for the gene *Sr31* which is located on the translocated 1BL.1RS chromosome (Singh et al., 2006). Thus, most genes originating form *Triticum aestivum* carry virulence spectrum to the Ug99 race (Singh et al., 2011). Variants of the disease with different virulences (i.e. for the genes *Sr24, Sr36* and *Sr38*) within the Ug99 lineage have also been detected in eastern Africa, complicating the resistance breeding efforts (Jin et al., 2009; Singh et al., 2011). The pathogen of stem rust, particularly of Ug99 is changing rapidly, and seven variants are now recognized as being part of Ug99 lineage. These Ug99 variants are having identical DNA fingerprints, but the avirulence and virulence profiles are slightly different (Szabo, 2007; Jin et al., 2009; Singh et al., 2011). The evolution of new virulences is appearing through mutation, migration and recombination of exciting virulence genes (Singh et al., 2008a). The race of Ug99 and its variants have spread across the African continent and have now established themselves in the Middle East. The uredospores of stem rust are highly adapted to long distance migration through wind irradiation and rain deposition (Rowell and Romig, 1966; Singh et al., 2006). Besides that, spread of spores may happen by accidental transport by means of contaminated clothing and goods. Since 1999, the migration of the Ug99 race has taken place from Uganda, to Kenya in 2001, to Ethiopia in 2003 and has been shown in most of the wheat production in those areas (Singh et al., 2006). In 2006, the Ug99 race was found in Sudan and Yemen, which was confirmed by race analysis (Singh et al., 2008b). The occurrence of the Ug99 race in Yemen was considered significant, as it provided a strong proof that the Ug99 race was migrating to the Middle East and Asia. In 2007 and in 2009 the Ug99 race was identified particularly in Iran (Nazari et al., 2009; Singh et al., 2011). According to Pretorius et al. (2010), in 2009 variants of the Ug99 (race PTKST virulence to *Sr31* and *Sr24*) were confirmed in South Africa (Figure 2). Distributions and confirmation of the seven variants of the Ug99 lineage from a number of countries have resulted in a naming by a five letter code as described by Jin et al., (2008), such as 1) TTKSK – Uganda in 1998, Kenya in 2001, Ethiopia in 2003, Sudan in 2006, Yemen in 2006, Iran in 2007 and Tanzania in 2009; 2) TTKSF – South Africa in 2000 and Zimbabwe in 2009; 3) TTKST – Kenya in 2006 and Tanzania in 2009; 4) TTTSK – Kenya in 2007 and Tanzania in 2009; 5) TTKSP – South Africa in 2007; 6) PTKSK – Ethiopia in 2007 and Kenya in 2009; 7) PTKST – Ethiopia in 2007, Kenya in 2008 and South Africa in 2009 (Singh et al., 2011).

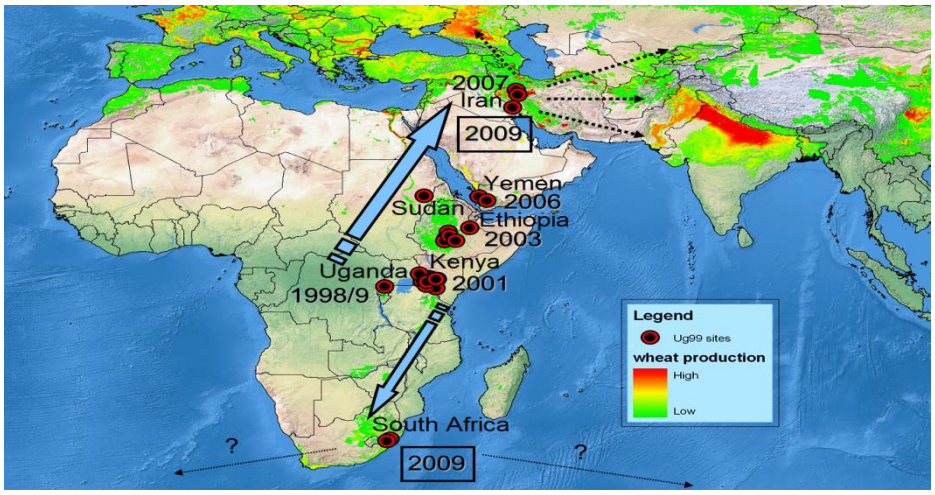

Figure 2. The spread of wheat stem rust race Ug99 lineage. **Source:** ***BGRI/CIMMYT/ICARDA***

Yellow rust

The yellow rust causative, *Puccinia striiformis* is a pathogen of cereal crops and grasses, and yellow rust is considered to be the most economically important disease (Roelfs et al., 1992). Initially yellow rust was described by Gadd and Bjerkander in 1777, and epidemically it was reported on rye in Sweden in 1794 (Eriksson and Henning, 1896; Singh et al., 2002). Schmidt, (1827) described the pathogen of yellow rust as *Uredo glumarum*, and later the yellow rust which was collected from rye (*Secale cereale*) was named *Puccinia striaeformis* (Westendorp, 1854). Also Fuckel (1860) studied rust and named it *Puccinia straminis*, but whether it was leaf rust or yellow rust is a doubt (Hassebrauk, 1965). Finally, yellow rust was shown being a separate rust disease of grasses and designated *Puccinia glumarum* (Eriksson and Henning, 1894). The name *Puccinia glumarum* was used until the pathogen was renamed as *Puccinia striiformis* Westend (Hylander et al. 1953; Cummins and Stevenson 1956; Manners, 1960), the name which is currently used. Common names of yellow rust have been Roya amarilla, Gelbrost, Rouille jaune, Gele roest, etc. (Eriksson and Henning, 1894; Humphrey et al., 1924; Stubbs, 1985). It thought that the center of origin for *Puccinia striiformis* is Transcaucasia i.e. Armenia, Azerbaijan and Georgia where the grasses were the primary host. Further, it is suggested that from the Transcaucasian countries, the pathogen has moved into Europe and along the mountain ranges to China and Eastern Asia (Humphrey et al., 1924; Stubbs, 1985; Line, 2002). The *Puccinia striiformis* has a center of origin in China and Central Asia, based on a high level of telia (pustule containing teliospores) production and high genetic diversity which is consistent with frequent recombination (Mboup et al., 2009; Ali et al., 2010). These telia produce teliospores which will germinate into aerial basidiospores and can infect the alternate or barberry host (Jin et al., 2010).

Taxonomy and Life Cycle of Yellow Rust

Yellow rust of grasses and cereal crops e.g. wheat, rye, barley and triticale is caused by different formae speciales of *Puccinia striiformis*, which is a fungus belonging to the order Uredinales of Basidiomycetes. Yellow or stripe rust of wheat is caused by *Puccinia striiformis* f. sp. *tritici*, and this biotrophic rust fungal group can be disseminated by airborne spores to a long distance (Zadoks, 1961; Brown and Hovmøller, 2002; Liu and Hambelton, 2010).

Figure 3. Symptom of yellow rust in A) Leaf; B) High infection of yellow rust in wheat, leading to fall down of spores; C) Infected *Elytrigia* species. *Photo: Mahbubjon Rahmatov*

The yellow rust life cycle consists of both dikaryotic uredial and telial stages, and the teliospores can also form haploid basidiospores. The *Puccinia striiformis* does not have any known alternate hosts for the basidiospores to infect, and the pycnial and aerial stages are unknown (Chen, 2005). The life cycle of *Puccinia striiformis* has similar as for most rust fungi damaging grasses and cereal crops been studied for more than a century, and still an alternate host of *Puccinia striiformis* was challenging to identify (Roelfs, 1992; Singh et al., 2002; Jin et al., 2010). In the late 19th century, only telial and uredinial stages of yellow rust were known while the alternate host was searched for among *Boraginaceae* species (Eriksson and Henning, 1894). However, recently the *Puccinia striiformis* pathogen was identified in barberry species, which were therefore suggested as alternate hosts, and sexual recombination was also found to play a role in the contribution to the pathogen variability (Jin et al., 2010). Figure 3 presents symptoms of the yellow rust in wheat and cereal grass.

Economic Importance

Yellow rust of wheat is a disease of great economic importance due to the severe damage caused on wheat and has therefore been subjected to extensive research (McIntosh et al., 1995). Historically, yellow rust epidemics have been significant in some locations, causing huge yield losses which require serious financial investment in order to manage the crops from loss (Wellings, 2007). Yellow rust decreases the yield, grain quality and forage value, and in most wheat producing regions the use of susceptible cultivars has resulted in yield losses of 10% – 70%. Severe epidemics of yellow rust are usually related with susceptible wheat cultivars, combined with favorable weather conditions for the disease like mild winters, as well as cool and wet springs and summers. Due to development or mutations of new races of *Puccinia striiformis*, varietal resistance can be overcome in a short period (Chen, 2005; Wellings, 2011; Hovmøller et al., 2011). Therefore cultivation of resistant cultivars is the most effective, economical and environmentally safe control measure of great value for the growers (Line and Chen 1995; Chen, 2005).

Epidemics of Yellow Rust

Historically, the epidemics of yellow rust have occurred in moderate regions with cool and wet spring and summer (Stubbs, 1985; Zadoks and Vandenbosch, 1995). The most and recent destructive epidemics have taken place in China, Northern and Eastern Africa, Western Asia, Central Asia and Middle East, and the epidemics may become even more aggressive with races that can tolerate and develop in higher temperatures (Hovmøller et al., 2010; 2011). According to Milus et al. (2009) the new races of yellow rust have significantly increased adaptation to warmer temperatures and therefore continue to cause disease epidemics.

Yield losses reported from yellow rust infection are $360 million in USA, in 2004, $100 million in Pakistan in 2005, $AUD127 million in Australia in 2009, $30 million in Morocco in 2009 and above 1 million tonnes in Syria in 2010 (Long, 2005; Duveiler et al., 2007; Murray and Brennan, 2009; Hodson, 2010; FAO, 2010). Five major epidemics of yellow rust have occurred in Central Asia in 1998, 2000, 2005, 2009 and 2010 (Ziyaev et al., 2011). In Tajikistan, the yellow rust is a serious disease with significant yield losses in susceptible cultivars. The severity of the yellow rust in 2010 resulted in damages up to 80-100% and yield losses of 30-50%, in widely grown farmers bread wheat varieties and 70% of the breeding lines showed susceptibility to yellow rust in the field trials (Rahmatov et al., 2011a; 2011b). Also, new aggressive races of yellow rust were found in Sweden and Denmark damaging triticale and affecting wheat, rye and barley (Jørgensen et al., 2010).

BREEDING FOR STEM RUST AND YELLOW RUST RESISTANCE

Since, new races of yellow rust and stem rust are moving and spreading throughout the worldwide wheat production regions, identification and transfer of novel sources of resistance genes are necessary. A number of wheat lines with transferred genetic material from related species are available such as wheat-rye, wheat-*leymus* and wheat-*thinopyrum* translocations/substitutions. The incorporation of genetic material from related species leads to wheat varieties adapted to the environment of interest, and to greater sustainability of the wheat production. The genetic resistance to stem rust and yellow rust can be characterized as qualitative and quantitative resistances. The qualitative resistance is classified into race-specific or vertical, seedling resistance, monogenic (major genes), hypersensitive, and the quantitative resistance is classified into the race-nonspecific or horizontal, adult plant resistance, slow rusting, polygenic (minor gene), durable etc. (Flor, 1956; McIntosh, 1988; 1995; Rajaram et al., 1988; Singh et al., 2000; Parlevliet, 2002; Chen, 2005; Clair, 2010; Lowe et al., 2011). In this paper 1) race-specific resistance/vertical resistance; 2) race-nonspecific resistance/or horizontal resistance; 3) seedling resistance; and 4) adult plant resistance (APR) are described.

Race-specific resistance

The gene-for-gene relationship states that for every resistance gene in the host plant there is a corresponding avirulence gene in the pathogen. However the ability of an avirulent gene to mutate to a virulent gene, no longer recognizable by the corresponding resistance gene, implies a type of resistance termed race-specific resistance (Flor, 1971). According to Dyck and Kerber (1985), a race-specific or vertical resistance signifies that the resistance to some pathogens is relatively simply inherited. The race-specific resistance is virulent only to particular races of a pathogen. Race-specific resistance is often based on genes that are effective at the seedling stage and remain effective at all post-seedling stages of the plants. Race-nonspecific resistances are mainly effective at the post-seedling and adult plant stages and adult plant resistance (APR) is often detected as field resistance (Johnson, 1992; Hovmøller et al., 2011). Most of the yellow rust and stem rust resistance genes are determined at seedling stages, and thus interact with specific races of the pathogen to confer resistance in a gene-for-gene relationship (Flor, 1971). Race specific resistance is usually governed by a hypersensitive response, controlled by major genes. The race-specific resistance is also known as monogenic resistance (resistance determined by a single gene), often led by a boom and bust cycle (Dyck and Kerber, 1985; Nagarajan and Joshi, 1985; Priyamvada and Tiwari, 2011)

Race non-specific resistance

Van der Plank (1968), described race non-specific resistance to be characterized by reduced apparent infection rate. Thus resistances that varied in a quantitative way and resulted in slow rusting were accepted to be supported by race non-specific resistance genes (Parlevliet, 1985). Race non-specific resistance conditioned by polygenes or quantitative genes is generally complex, as is its identification. Most of the race non-specific resistance tests have been carried out in adult plants (Roelfs et al., 1992). Thus, APR genes are considered to control race non-specific resistance, thereby contributing with partial resistance and being associated with a slow rusting resistance (Priyamvada and Tiwari, 2011). The stem rust resistance gene *Sr2* is considered to be one example of a gene contributing to partial or slow rusting resistance (McIntosh et al., 1995; Bansal et al., 2008). The race non-specific resistance is governed by minor genes and is therefore considered as a polygenetic resistance (resistance to parasites based on many genes). This type of resistance is often considered as durable and the genes are pyramiding. Most commonly, race non-specific resistance is characterized by durability, having a partially resistant phenotype, and being effective to a broad range of stem rust and yellow rust races with optimal level of expression at the adult plant stages (Parlevliet, 1985; McIntosh et al., 1995).

Seedling Resistance Test

Yellow rust and stem rust resistance genes are postulated or characterized based on seedling resistance test. The seedling resistance genes can be detected and are effective at the seedling stages, and they are characterized by the gene-for-gene interaction model (Flor, 1971). Generally, the seedling resistance genes are also active during the adult plant stage, and they are classified into race-specific resistance types (Chen, 2005; Lagudah, 2010). So far, above sixty yellow rust and fifty five stem rust race-specific resistance genes based on seedling resistance test have been identified (McIntosh et al., 2010; Singh et al., 2011). However, the seedling resistance genes are often broken down due to new and various races of the rusts pathogen (Chen and Moore, 2002). To characterize the seedling resistance genes of stem rust and yellow rust special scales have been developed and are demonstrated in Table 1 and 2 (McNeal, et al., 1971; McIntosh et al., 1995). Reaction of seedling on infection by stem rust and yellow rust is shown in Figure 4.

Table 1. Major infection type classes for stem rust infection type

Infection type	Host Response	Symptoms
0	Immune	No visible uredia
;	Very resistant	Hypersensitive flecks
1	Resistant	Small uredia with necrosis
2	Resistant to moderately resistant	Small to medium sized uredia with green islands and surrounded by necrosis or chlorosis
3	Moderately resistant/ moderately susceptible	Medium sized uredia with or without chlorosis
4	Susceptible	Large uredia without chlorosis

Table 2. Major infection type classes for yellow rust

Infection type		Host response	Symptoms
McNeal et al. (1971)	McIntosh et al., (1995)		
0	0	Immune	No visible uredia
1	**;**	Very resistant	Necrotic flecks
2	;N	Resistant	Necrotic areas without sporulation
3-4	1	Resistant	Necrotic and chlorotic areas with restricted sporulation
5-6	2	Moderately resistant	Moderate sporulation with necrosis and chlorosis
7-8	3	Moderately susceptible	Sporulation with chlorosis
9	4	Susceptible	Abundant sporulation without chlorosis

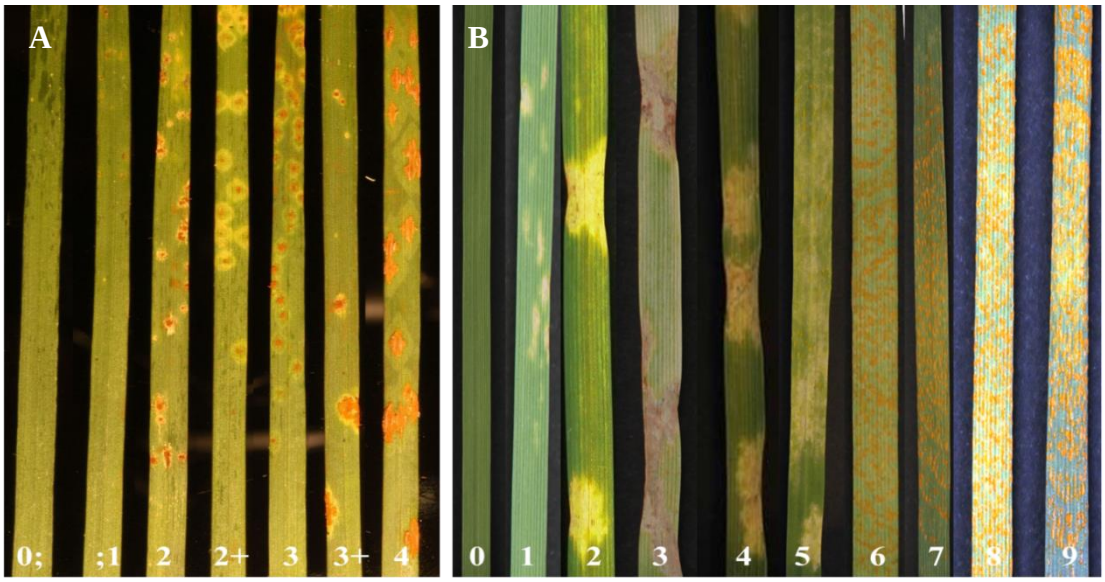

Figure 4. A) Stem Rust and B) Yellow Rust seedling reaction. Photo: Mahbubjon Rahmatov

Adult Plant Resistance Test

Wheat breeders and pathologists have always been concentrating on APR genes in order to identify and improve the level of resistances (Bansal et al., 2008). The detection of APR is usually conducted at the post-seedling stage, and is often characterized as field resistance (Van der Plank, 1982; Lagudah, 2010). APR genes are effective only in APR stages, but have been shown to be an important part of durable rusts resistance (Johnson, 1978; Priyamvada and Tiwari, 2011). The principle of APR may derive at any time during the post-seedling stage and also environmental factors (i.e. high and low temperature, climate change etc.) may interact for the APR gene expression (Bariana and McIntosh, 1995). The APR disease responses and severities based on the modified Cobb scale (Peterson et al. 1948), and the reaction types by Roelfs et al., (1992) are classified in Table 3.

Table 3. APR Disease response and severity for stem rust and yellow rust

Disease response	Disease severity, %	Host Response	Symptoms
R	0-5	Resistant	Resistant, no visible infection or some chlorosis or necrosis and no uredia
R-MR	10-20	Resistant to moderately resistant	
MR	20-30	Moderately resistant	Moderately resistant, small uredia present and surrounded by either chlorotic or necrotic areas
MR-MS	30-40	Moderately resistant to moderately susceptible	
MS	40-50	Moderately susceptible	Moderately susceptible, medium-sized uredia present and possibly surrounded by chlorotic areas
MS-S	50-70	Moderately susceptible to susceptible	
S	70-100	Susceptible	Susceptible, large uredia present, generally with little or no chlorosis and no necrosis

NOVEL SOURCES OF RESISTANCE GENES TO STEM RUST AND YELLOW RUST

Breeding for stem rust and yellow rust resistance always requires a constant inflow of novel sources of resistance genes, due to the appearance of new virulent pathogen races (i.e. Ug99) (Singh et al., 2011; Lowe et al., 2011). Resistance breeding might utilize novel stem rust and yellow rust resistance genes by means wheat-rye, wheat-*leymus* and wheat-*thinopyrum* introgression lines. Ultimately, these identified genes will be used to develop high yielding wheat cultivars, keeping in mind food security, environmental issues and human health.

Wheat Gene Pool

Bread wheat (*Triticum aestivum L.*) is a hexaploid species constituted of the AABBDD genome. The donors of the wheat genome are: AA *Triticum urartu*, BB *Aegilops speltoides* and DD *Aegilops tauschii* (Dvorak, 1998). Wheat belongs to the tribe *Triticeae* of the family *Poaceae.* According to crossability with hexaploid wheat, other related species are divided into three major gene pools: The primary gene pool; the secondary gene pool; and the tertiary gene pool (Mujeeb-Kazi and Rajaram, 2002). These gene pools can play an important role for present day wheat breeding when introducing novel sources of resistance to develop resistant cultivars toward yellow rust and stem rust. The source, origin and chromosomal location of stem rust and yellow rust race-specific resistance genes are presented in Table 3 and 4 (McIntosh et al., 1995; Tyrka and Chelkowski, 2004; Singh et al., 2011; http://www.shigen.nig.ac.jp/wheat/komugi/genes/symbolClassList.jsp; http://www.ars.usda.gov /Main/docs.htm?docid=10342).

Primary Gene Pool

The primary gene pool of bread wheat consist of species that have genomes homologues, with bread wheat, *Triticum aestivum* (AABBDD), e.g. hexaploid spelt (*Triticum spelta* AABBDD), tetraploid *Triticum turgidum* (AABB), diploid *Triticum monococcum* (AA), *Triticum dicoccoides*, *Aegilops tauschii* (DD), as well as landraces of hexaploid and tetraploid wheat (Mujeeb-Kazi and Rajaram, 2002). The desired genes within this group are possible to transfer via direct hybridization, homologous recombination, backcrossing, and selection (Friebe et al., 1996; Mujeeb-Kazi and Rajaram, 2002). Some sources of the resistance genes to stem rust and yellow rust are originating from the primary gene pools (Tables 3 and 4).

Secondary Gene Pool

The secondary gene pool of hexaploid wheat contains polyploid *Aegilops* and *Triticum* species that have one genome in common with *Triticum aestivum* e.g. *Triticum timopheevii* (AAGG) and *Triticum araraticum* (AAGG). Some *Aegilops* species share the evolution of wheat and have played an important role in wheat domestication. Examples of such *Aegilops* species include the Sitopsis section related to the B genome of hexaploid wheat, e.g. *Aegilops speltoides* and *Aegilops longissima* (2n=2x=14). Thus, the genus *Aegilops* represents the largest part of the secondary gene pool of wheat, and several species have been used by direct crossing, backcrossing, selection via chromosome recombination, embryo rescue and cytogenetic manipulations to enhance the recombination in wheat improvement programs (Mujeeb-Kazi and Rajaram, 2002; Mujeeb-Kazi, 2003; Kilian et al., 2011). The source of resistance genes from the secondary gene pool is presented in Tables 3 and 4.

Tertiary Gene Pool

Diploid and polyploid species, which are members of the tertiary gene pool of hexaploid wheat, have non-homologous genomes with hexaploid wheat. One promising source of novel genes for wheat is wheatgrasses and wild rye both being included in the tertiary gene pool. This gene pool has been successfully hybridized with wheat and genes have been incorporated into the bread wheat genome, representatives are from *Agropyron*, *Pseudoroegneria*, *Psathyrostachys*, *Thinopyrum*, *Elymus*, *Secale cereale, Hordeum vulgare* and *Leymus* species (Dewey 1984; Mujeeb-Kazi and Wang 1995; Wang and Jensen 2009). However, the tertiary gene pool species have been limitedly exploited in wheat, because the genomes of these species are non-homologous to those of wheat, and genetic transfers cannot be made by homologous recombination. In order to incorporate genome of these species, special techniques such as embryo rescue, irradiation etc., and further cytological manipulation are required (Friebe et al., 1996; Mujeeb-Kazi and Rajaram, 2002; Mujeeb-Kazi, 2003). Consequently, in this gene pool usually linkage drag is the effect, which could be associated with undesirable agronomic traits (Qi et al., 2007; Gill et al., 2011), and due to homoeology the linkage block might be inherited (Hanson, 1959a; 1959b; Pumphrey, 2012). Despite this, some of the stem rust and yellow rust resistance genes are originating from tertiary gene pool species (Tables 3 and 4).

Table 3. Origin and sources of resistance genes of stem rust

Sr Gene	Source/Origin	Chromosome	*Sr* Gene	Source/Origin	Chromosome
2	*Triticum turgidum*	3BS	25	*Thinoporum elongatum*	7DL
5	*Triticum aestivum*	6DS	26	*Thinoporum elongatum*	6AL
6	*Triticum aestivum*	2DS	27	*Secale cereale*	3A/3R
7a	*Triticum aestivum*	4BL	28	*Triticum aestivum*	2BL
7b	*Triticum aestivum*	4BL	29	*Triticum aestivum*	6DL
8a	*Triticum aestivum*	6AS	30	*Triticum aestivum*	5DL
8b	*Triticum aestivum*	6AS	31	*Secale cereale*	1BL/1RS
9a	*Triticum aestivum*	2BL	32	*Aegilops speltoides*	2AS, 2B
9b	*Triticum aestivum*	2BL	33	*Aegilops tauschii*	1DS
9d	*Triticum turgidum*	2BL	34	*Triticum comocum*	2A,2B
9e	*Triticum turgidum*	2BL	35	*Triticum monococcum*	3AL
9f	*Triticum aestivum*	2BL	36	*Triticum timopheevi*	2BS
9g	*Triticum turgidum*	2BL	37	*Triticum timopheevi*	4BL
10	*Triticum aestivum*	2B	38	*Triticum ventricosum*	2AS
11	*Triticum turgidum*	6BL	39	*Aegilops speltoides*	2B
12	*Triticum turgidum*	3BS	40	*Triticum araraticum*	2BS
13	*Triticum turgidum*	6AL	41	*Triticum aestivum*	4D
14	*Triticum turgidum*	1BL	42	*Triticum aestivum*	6DS
15	*Triticum aestivum*	7AL	43	*Thinoporum elongatum*	7D
16	*Triticum aestivum*	2BL	44	*Thinoporum intermedium*	7DS
17	*Triticum turgidum*	7BL	45	*Aegilops tauschii*	1DS
18	*Triticum aestivum*	1D	46	*Aegilops tauschii*	2DS
19	*Triticum aestivum*	2BS	47	*Aegilops speltoides*	2B = 2BL-2SL.2SS
20	*Triticum aestivum*	2BL	48	*Triticum aestivum*	2AL
21	*Triticum monococcum*	2AL	49	*Triticum aestivum*	5BL
22	*Triticum monococcum*	7AL	50 [R]	*Secale cereale*	1DL/1RS
23	*Triticum aestivum*	2BS	Tmp	*Triticum aestivum*	
24	*Thinoporum elongatum*	3DL	1A.1R	*Secale cereale*	1A.1R

Table 4. Origin and sources of resistance genes of yellow rust

Yr Gene	Source/Origin	Chromosome	*Yr* Gene	Source/Origin	Chromosome
1	*Triticum aestivum*	2A, 2AL	30	*Triticum aestivum*	3BS
2	*Triticum aestivum*	7B	31	*Triticum aestivum*	2BS
3	*Triticum aestivum*	Unknown	32	Carstens V	2AL
3a	*Triticum aestivum*	1B, 2B	33	Batavia	7DL
3b	*Triticum aestivum*	Unknown	34	WAWHT2046	5AL
3c	*Triticum aestivum*	1B	35	*Triticum dicoccoides*	6BS
4	*Triticum aestivum*	3BS	36	*Triticum dicoccoides*	6BS
4a	*Triticum aestivum*	6B	37	*Aegilops kotschyi*	2DL
4b	*Triticum aestivum*	6B	38	*Aegilops sharonensis*	6A
5	*Triticum spelta album*	2BL	39	Alpowa	7BL
6	*Triticum aestivum*	7B, 7BS	40	*Aegilops geniculata*	5DS
7	*Triticum turgidum*	2B, 2BL	YrCle	Clement	4B
8	*Aegilops comosa*	2D	YrD	Druchamp	6A
9	*Secalis cereale*	1B=1BL.1RS	YrH46	Hybrid 46	6A
10	*Triticum spelta*	1B, 1BS	YrHVII	Heines VII	4A
11	*Triticum aestivum*	Unknown	YrMin	Minister	4A
12	*Triticum aestivum*	Unknown	YrMor	Moro	4B
13	*Triticum aestivum*	Unknown	YrND	Nord	4A
14	*Triticum aestivum*	Unknown	YrS	Stephens	3BS
15	*Triticum dicoccoides*	1BS	YrTye	Tyee	6D
16	*Triticum aestivum*	2D	YrTr1	Tres	6D
17	*Aegilops ventricosa*	2AS-6M	Tres	Tres	3A
18	*Triticum aestivum*	7D, 7DS	YrYam	Yamhill	4B
19	*Triticum aestivum*	5B	YrV23	Vilmorin	2B
20	*Triticum aestivum*	6D	Yrns-B1	Lgst.79-74	3BS
21	*Triticum aestivum*	1B	YrSte		2B
22	*Triticum aestivum*	4D	YrSte2		3B
23	*Triticum aestivum*	6D	YrDa1		1A
24	*Triticum turgidum*	1BS	YrDa2		5D
25	*Triticum aestivum*	1D	YrA		Unknown
26	*Haynaldia 23illosa*	1BS, 1BL	YrDru		5B, 6B
27	*Triticum aestivum*	2BS	YrDru2		6A
28	*Aegilops tauschii*	4DS	YrH52		1BS
29	*Triticum aestivum*	1BL	YrCk		2DS

Genetics behind and evaluation of wheat-rye, wheat-*Leymus* and wheat-*Thinopyrum* alien introgressions

The first wheat-rye 5A (5R) chromosome spontaneous substitutions were reported by Katterman (1937) and O'Mara (1947). However, such substitution lines have non-homologous pairing of the chromosome 5 and therefore it was a complicated task to transfer desired traits (O'Mara, 1940). O'Mara therefore crossed and backcrossed the wheat and wheat-rye amphidiploid, to produce monosomic and disomic plants (O'Mara; 1940; 1951; Riley and Chapman, 1958a). Later 1B (1R) substitutions, 1BL.1RS and 1AL.1RS translocations have been identified in several widely grown wheat cultivars (Blüthner and Mettin, 1974; Mettin et al., 1973; Zeller, 1973; Schlegel and Korzun, 1997). The source of these alien substitution and translocation chromosomes has been intensively discussed in terms of the genetic background and historical basis. Basically four sources are supposed to exist, two in Germany, one in the USA and one in Japan (Schlegel and Korzun, 1997). The first, 1BL.1RS wheat-rye translocation with the 1RS chromosome/segment introduced into the wheat genome was obtained through the Petkus rye in 1950 in Germany (Mettin et al., 1973; Zeller, 1973; Schlegel and Korzun, 1997; Rabinovich, 1998). A number of useful genes for particular diseases (yellow rust *Yr9*, stem rust *Sr31*, leaf rust *Lr26* and powdery mildew *Pm8*) and pests (aphids, Hessian fly etc.) resistances have been localized on the seven rye chromosomes and transferred into the wheat genome (Schlegel et al., 1998; McIntosh et al., 1995; Friebe et al., 1996). Bread wheat has also been crossed with an octoploid triticale in Japan and the cultivar Salmon (1BL.1RS) was developed through this cross. The cultivar Salmon has thereafter been used to develop breeding lines and cultivars (Tsunewaki 1964; Rabinovich, 1998). The 1AL.1RS translocation originated through the Argentinian rye Insave and from there the cultivar Amigo was developed carrying the resistance gene *Sr1AL.1RS* to stem rust (Zeller and Fuchs, 1983; Lukaszewski, 1990; Singh et al., 2011). The 1DL.1RS wheat-rye translocation originated through the Imperial rye carrying stem rust resistance genes *Sr50* (*SrR*) (Mago et al., 2002). The 1RS translocation became spread worldwide through the cultivars Aurora and Kavkaz, which played a significant role in wheat breeding programs to develop new wheat cultivars (Rabinovich, 1998). In 2011, about 1.050 varieties were carrying the 1RS.1BL translocation, about 100 varieties the 1RS.1AL translocation, and about 30 varieties a 1R (1B) substitution as reported in the rye gene map database (http://www.rye-gene-map.de/rye-introgression/).

A significant number of different wheat-rye translocation and substitution lines have been developed in Sweden by late Professor Arnulf Merker, using hexaploid triticale and bread wheat. In this crossing procedure, spring wheat cultivars and lines such as Drabant, Prins, Sonett and SV 77328 and spring triticale cultivars Beagle and Drira were used. The obtained F_1 in these combinations were backcrossed with bread wheat and BC_1F_1 were produced (Table 5). In the BC_1F_3 generation C-banding analysis were performed to check the presence of rye chromosomes in the lines. The results showed that a number of different combinations of wheat-rye translocations and substitutions were obtained (Merker, 1984; 1992). Moreover, hexaploid winter triticale such as Sv856003, Sv876012, Sv876032, double wheat-rye 1R and 2R substitutions, wheat-*Leymus mollis* introgression lines (AD99), and Swedish winter wheat cultivars Goerzen, Holme and Kraka were used for crossing and backcrossing (Table 6). Thereafter, Fluorescent In-situ Hybridization, C-banding and powdery mildew resistance test were utilized to identify and confirm the presence of the introgressed chromosomes in the wheat genome (Forsstrom and Merker, 2001; Forsstrom et al., 2002).

Also *Leymus* and *Thinopyrum* species have been used for wide-crossing of wheat to enhance transfer of valuable traits into the wheat genome by introgression of alien chromosomes in order to control the effect of biotic and abiotic factors (Baum et al., 1992; Jiang et al., 1994; Ellneskog-Staam and Merker, 2001). Seven species of *Leymus (L. racemosus, L. arenarius, L. mollis, L. cinereus, L. triticoides, L. angustus* and *L. multicaulis)* have been successfully hybridized to wheat with the aim to transfer resistance to fungal and virus diseases, as well as to drought and salinity tolerance (Petrova, 1960; Mujeeb-Kazi and Rodriguez 1980; 1981; Mujeeb-Kazi et al.,1984; Plourde et al., 1989; 1992; Merker and Lantai, 1996;). The *Leymus* species belong to a polyploid genus, and *L. arenarius* (2n=56 octoploid), *L. mollis* and *L. racemosus* (2n=28 tetraploid) have been used for cultivation as perennial species and for breeding of amphiploids (hybrid which have a diploid set of chromosomes from each parental species) with wheat (Anamthawat-Jonsson et al., 1997). Spring hexaploid bread wheat-rye translocation 5RL.5BS lines have been crossed with *L. arenarius*, *L. mollis*, *L. racemouses* and *Thinopyrum junceiforme*. Also, three lines of the tetraploid wheat *T. turgidum* var. *carthlicum* (2n=28 AABB) were used for crossing with *L. arenarius*, *L. mollis*, *L. racemouses* and *Thinopyrum junceiforme*. However, in these combinations the hybrids were obtained through the use of embryo culture technique. Thereafter the hybrids from the *L. mollis* and *Thinopyrum junceiforme* were backcrossed to the 5RL.5BS wheat-rye translocation line, and the hybrids of the *Thinopyrum junceiforme* were also backcrossed to all three lines of the tetraploid wheat *T.*

turgidum var. *carthlicum* and BC_1F_1 were obtained (Merker and Lantai 1996; Ellneskog-Staam, and Merker, 2001; 2002a; 2002b). The BC_1F_2 were selfed, and in the BC_1F_3 and further generations the Genomic In-situ Hybridization was used to analyze the genomic compositions and resistance tests against leaf rust and powdery mildew were performed (Table 7; Ellneskog-Staam, and Merker, 2001; 2002a; 2002b). Wheat-*Leymus* and wheat-*Thinopyrum* lines have been used for cytogenetic analyses to elucidate their chromosome composition, meiotic stability and fertility (Ellneskog-Staam and Merker, 2001; 2002a). Furthermore, the stem rust *Sr24*, *Sr25*, *Sr26*, *Sr43* and leaf rust *Lr19* resistance genes were derived from *T. elongatum*, while the *Sr44* originated from *T. intermedium* (McIntosh et al. 1995; Singh et al., 2011).

Table 5. The crossing and backcrossing combinations of the hexaploid spring triticale and wheat cultivars by A. Merker

Crossing	Generation & Backcrossing		Note
Beagle x Drabant	F_1 x Prins	BC_1F_1	Selfed
		BC_1F_2	Selfed
		BC_1F_3	C-banding
Beagle x Sonett	F_1 x Sv 77328	BC_1F_1	Selfed
		BC_1F_2	Selfed
		BC_1F_3	C-banding
Drira x Sonett	F_1 x Sv 77328	BC_1F_1	Selfed
		BC_1F_2	Selfed
		BC_1F_3	C-banding
Drira x Sonett	F_1 x Sonett	BC_1F_1	Selfed
		BC_1F_2	Selfed
		BC_1F_3	C-banding
Drira x Drabant	F_1 x Prins	BC_1F_1	Selfed
		BC_1F_2	Selfed
		BC_1F_3	C-banding

Table 6. The crossing and backcrossing combinations of the hexaploid winter triticale and wheat cultivars by A. Merker

Crossing			Generation & Backcrossing				Note
Sv 856003	x	Holme	F_1	x	Kraka	BC_1F_1	Selfed
						BC_1F_2	Selfed
						BC_1F_3	Selfed
						BC_1F_4	Selfed
						BC_1F_5	Pm resiatnce test
Sv 876012	x	Holme	F_1	x	Kraka	BC_1F_1	Selfed
						BC_1F_2	Selfed
						BC_1F_3	Selfed
						BC_1F_4	Selfed
						BC_1F_5	Pm resiatnce test
Sv 876032	x	Holme	F_1	x	Kraka	BC_1F_1	Selfed
						BC_1F_2	Selfed
						BC_1F_3	Selfed
						BC_1F_4	Selfed
						BC_1F_5	Pm resiatnce test
Sv 876032	x	Holme	F_1	x	Goerzen	BC_1F_1	Selfed
						BC_1F_2	Selfed
						BC_1F_3	Selfed
						BC_1F_4	Selfed
						BC_1F_5	Pm resistance test
AD99	x	Kraka		x	Kraka	BC_1F_1	Selfed
						BC_1F_2	Selfed
						BC_1F_3	Pm resistance test
AD99	x	Kraka		x	Holme	BC_1F_1	Selfed
						BC_1F_2	Selfed
						BC_1F_3	Pm resistance test
AD99	x	Goerzen		x	Wheat	BC_1F_1	Selfed
						BC_1F_2	Selfed
						BC_1F_3	Pm resistance test
1R and 2R	x	Holme	F1		None		In F_5 generation the Pm resistance test were performed
1R and 2R	x	Kraka	F1		None		In F_5 generation the Pm resistance test were performed
1R and 2R	x	Goerzen	F1		None		In F_5 generation the Pm resistance test were performed

Note: Pm – Powdery mildew

Table 7. The crossing and backcrossing combinations of the hexaploid and tetraploid wheat with *Leymus arenarius, Leymus mollis, Leymus racemosus* and *Thinopyrum junceiforme*

Crossing	Generation & Backcrossing	Note
Tr. Cartlicum x *Lr* F_1 x *Tr. Cartlicum*	BC_1F_1	Selfed
	BC_1F_2	Selfed
	BC_1F_3	Pm resistance test and GISH analysis
Tr. Cartlicum x *Thj* F_1 x *Tr. Cartlicum*	BC_1F_1	Selfed
	BC_1F_2	Selfed
	BC_1F_3	Pm resistance test and GISH analysis
Hpph x *Lm* F_1 x Hpph	n.a.	n.a
Hpph x *Thj* F_1 x Hpph	n.a	n.a
Hpph x *Lr* F_1 x Hpph	n.a	n.a.
Hpph x *La* F_1 None	n.a.	n.a.

Note: ***La – Leymus arenarius; Lm – Leymus mollis; Lr – Leymus racemosus; Thj - Thinopyrum junceiforme***

The role of the *Ph1* gene and *Ph1b* mutant in developing wheat-alien introgressions

A transfer of genes from the tertiary gene pool via homologous recombination into the bread wheat is very rare, since the homologous chromosome pairing in wheat is strictly controlled by the *ph1* gene (Qi et al., 2007). Hexaploid wheat behaves as a diploid organism at meiosis with controlled pairing due to the *ph1* gene (Riley and Chapman, 1958b; Sears, 1976). The *ph1* gene is located in the 5B (5BL) of the hexaploid and tetraploid wheat chromosomes (Okamoto, 1957; Sears and Okamoto, 1958). Moreover, other *ph* genes having minor effect on the homoeologous pairing are located on the 3AS and 3DS chromosomes, and possible on the 4D chromosome, and also some other promoters suppress the pairing (Mello-Sampayo and Canas, 1973; Driscoll, 1973; Sears, 1976; 1977). X-ray irradiation has been used in hexaploid and tetraploid wheat for deletion of the *ph1* gene on the 5B (5BL) chromosome. A *Ph1b* mutant in hexaploid and *Ph1c* mutant in tetraploid wheat has been produced (Sears, 1977; Giorgi and Cwozzo, 1980; Giorgi and Barrerab, 1981). Furthermore, another gene called ph^I (inhibitor) was transferred from *Aegilops speltoides* to Chinese spring (Chen at al., 1994). The ph^I gene suppresses the influence of *Ph1* gene, and thereby allows the homoeologous recombination between alien and wheat chromosomes. The ph^I is a dominant gene that eases the transfer of alien chromosomes into the wheat genome, and thus contributes to homoeologous pairing in the F_1 generation (Chen et al., 1994). Several, wheat-alien introgression lines have been developed using *ph1b* and ph^I mutants (Sears, 1981; 1982; Koebner and Shepherd, 1985; Chen

et al., 1994; Lukaszewski, 2000; Qi et al., 2007). Thus novel sources of stem rust, yellow rust and leaf rusts resistance genes have been derived from alien species using *ph1b* and *ph*^{I} mutants (Sears, 1956; Sarbarzeh et al., 2002; Dundas et al., 2007; Mago et al., 2009; Niu et al., 2011). A strategy of the wheat-alien recombinant chromosomes using *ph1b* and *ph*^{I} mutants is demonstrated in Figure 5.

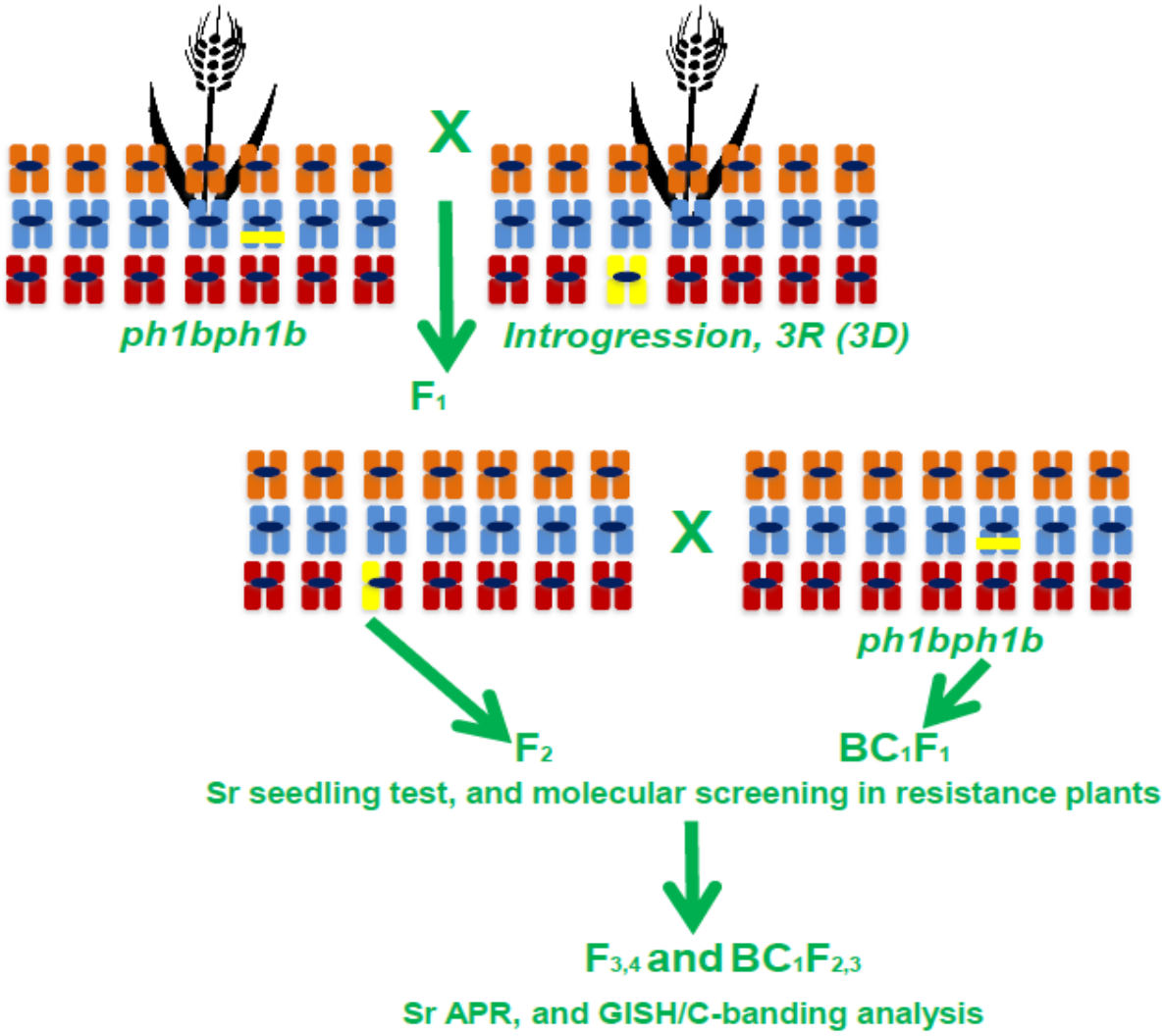

Figure 5. Developing of wheat-alien chromosome recombinant lines by using *ph1b* mutant for induced homoeologous recombination

Available molecular markers for stem rust and yellow rust resistance genes in wheat

Molecular markers are used to genetically map genes of interest in sexually reproducing organisms. During the last years, detailed genetic maps including more than 3000 molecular markers have been developed for wheat (Gill et al., 1991; Nelson et al., 1995; Röder et al., 1998; Somers et al., 2004). A number of different types of molecular markers are used e.g. Restriction Fragment Length Polymorphisms (RFLPs) (Gill et al., 1993; Nelson et al., 1995), Simple Sequence Repeats (SSRs) (Röder et al., 1998; Somers et al., 2004), Amplified Fragment Length Polymorphisms (AFLPs) (Peng et al., 2000), and Expressed Sequence Tags (ESTs) (Lazo et al., 2004; Qi et al., 2004; http://maswheat.ucdavis.edu/protocols/StemRust/index.htm; http://wheat.pw.usda.gov/NSF/). Both major genes and QTLs for different particular traits have been tagged in wheat (Varshney et al., 2006; 2007). Significant achievements have been reached as to mapping disease resistance genes, QTLs and major genes of particular importance for yellow rust and stem rust and available markers are summarized in tables 5 and 6.

]**Table 5. List of available stem rust markers**

Gene/ QTLs	Chromosome	Marker	Type	Sequence or Primer Pair	Reference
Sr1A/1R	1AL/1RS	Xbarc1048	SSR	F 5' ACGTGGTAATTAGTTGGGAGTCTGTA 3' R 5' TGACAACCCCCTTTCCCTCGT 3'	Yu et al., 2009; Saal and Wricke, 1999
		SCM9	SSR	F 5' TGACAACCCCCTTTCCCTCGT 3' R 5' TCATCGACGCTAAGGAGGACCC 3'	
		Xbarc028	SSR	F 5' CTCCCCGGCTAGTGACCACA 3' R 5' GCGGCATCTTTCATTAACGAGCTAGT 3'	
Sr2	3BS	Xqwm533	SSR	F 5' GTTGCTTTAGGGGAAAAGCC 3' R 5' AAGGCGAATCAAACGGAATA 3'	Hayden et al., 2004
		stm598tcac		F 5' GTTGCTTTAGGGGAAAAGCC 3' R 5' TCTCTCTCTCTCTCACACACAC 3'	
		Xgwm389	SSR	F 5' ATCATGTCG ATCTCCTTGACG 3' R 5' TGC CAT GCACATTAGCAGAT 3'	Röder et al., 1998
Sr6	2DS	Xwmc453	SSR	F 5' ACTTGTGTCCATAACCGACCTT 3' R 5' ATCTTTTGAGGTTACAACCCGA 3'	Tsilo et al., 2009; Yu et al., 2009
		Xcfd43	SSR	F 5' AACAAAAGTCGGTGCAGTCC 3' R 5' CCAAAAACATGGTTAAAGGGG 3'	
Sr9a	2BL	Xgwm47	SSR	F 5' TTGCTACCATGCATGACCAT 3' R 5' TTCACCTCGATTGAGGTCCT 3'	Röder et al., 1998
Sr13	6AL	Xwmc580	SSR	F 5' AAGGCGCACAACACAATGAC 3' R 5' GGTCTTTTGTGCAGTGAACTGAAG 3'	Simons et al., 2011
		Xdupw168	SSR	F 5' CGGAGCAAGGACGATAGG 3' R 5' CACCACACCAATCAGGAACC 3'	
Sr15	7AL	STS638	STS	F 5' GCGGTGACTACACAGCGATGAAGCAATGAAA 3' R 5' GCGGTGACTAGTCCAGTTGGTTGATGGAAT 3'	Neu et al., 2002
Sr17	7BL	wPt5343	DArT	F 5' TATTCTACAACGCTCCATCC R 5' CGCATGCAANCCATACCTTT	Crossa et al., 2007; Yu et al., 2009
		wPt0600	DArT	F 5' AGCTCGTACAATGGTGG R 5' CATGAAATAAGCTGCCACTT	
Sr19	2BS	wPt9402	DArT	F 5' ATTTTATATTGCCGTGCCAG R 5' ATGGCCAGCACGATAGAGAG	Crossa et al., 2007; Yu et al., 2009
Sr22	7AL	cfa2123	SSR	F 5' CGG TCTTTGTTTGCTCTAAACC 3' R 5' ACC GGC CATCTATGATGAAG 3'	Yu et al., 2010
		cfa2019	SSR	F 5' GACGAGCTAACTGCAGACCC 3' R 5' CTCAATCCTGATGCGGAGAT 3'	
		Xbarc121	SSR	F 5' ACTGATCAGCAATGTCAACTGAA 3' R 5' CCGGTGTCTTTCCTAACGCTATG 3'	
Sr24	3DL	Xbarc71	SSR	F 5' GCGCTTGTTCCTCACCTGCTCATA 3' R 5' GCGTATATTCTCTCGTCTTCTTGTTGGT T 3'	Mago et al., 2005; Yu et al., 2010
		Sr24#12	AFLP	F 5' CACCCGTGACATGCTCGTA 3' R 5' AACAGGAAATGAGCAACGATGT 3'	
Sr25	7DL	BF145935	EST	F 5' CTTCACCTCCAAGGAGTTCCA C 3'	Ayala-Navarrete et al.,

				R 5’ GCGTACCTGATCACCACCTTGAAGG 3’	2007
		Gb		F 5’ CAT CCT TGG GGA CCT C 3 R 5’ CCA GCT CGC ATA CAT CCA 3	Yu et al., 2010
Sr26	6AL	Sr26#43		F 5’ AATCGTCCACATTGGCTTCT 3’ R 5’ CGCAACAAAATCATGCACTA 3’	Liu et al., 2009; Yu et al., 2010
		BE518379		F 5’ AGCCGCGAAATCTACTTTGA 3’ R 5’ TTAAACGGACAGAGCACACG 3’	
Sr28	2BL	wPt7004-PCR	DArT	5' CTCCCACCAAAACAGCCTAC 3' 5' AGATGCGAATGGGCAGTTAG 3'	Rouse et al., 2012;
		wmc332	SSR	5' CATTTACAAAGCGCATGAAGCC 3' 5' GAAAACTTTGGGAACAAGAGCA 3'	
Sr31	1BL/1RS	1B-159		F 5' AGCGCAGATAATGTTTGAACC 3' R 5' AAGTCGAAACCACAGTTATC 3'	Mago et al., 2004;
		Iag95	STS	F 5' CTCTGTGGATAGTTACTTGATCGA 3' R 5' CCTAGAACATGCATGGCTGTTACA 3'	Mago et al., 2002;
		wpt8949	DArT	F 5' TGGGATGCGAGAATATCCGG R 5' TGCGATGCCTAAAGCCTCTC	Crossa et al., 2007; Yu et al., 2009
		wpt1328	DArT	F 5' GCGCCGGTCGGACAGACCGG R 5' GAACTACTAATTACTGTACA	
Sr32	2AS, 2B	STM773	SSR	F 5' AAACGCCCCAACCACCTCTCTC R 5' ATGGTTTGTTGTGTTGTGTGTAGG	Somers et al., 2004; Yu et al., 2009
		Xbarc55	SSR	F 5' GCGGTCAACACACTCCACTCCTCTCTC 3' R 5' CGCTGCTCCCATTGCTCGCCGTTA 3'	
Sr33	1DS	Abc156	STS	F 5' TTACGGGATCAAAGCTGAGGC R 5' GACAAGCAACACCAACCAAGC	Mago et al., 2002; Yu et al., 2009
Sr35	3AL	Xcfa2170	SSR	F TGGCAAGTAACATGAACGGA R ATGTCATTCATGTTGCCCCT	Yu et al., 2009; Zhang et al., 2010
		Xwmc559	SSR	F ACACCACGAATGATGTGCCA R ACGACGCCATGTATGCAGAA	
		Xcfa2076	SSR	F CGAAAAACCATGATCGACAG R ACCTGTCCAGCTAGCCTCCA	
		Xwmc169	SSR	F TACCCGAATCTGGAAAATCAAT R TGGAAGCTTGCTAACTTTGGAG	
Sr36	2BS	Xgwm319	SSR	F 5' GGTTGCTGTACAAGTGTTCACG 3' R 5' CGGGTGCTGTGTGTAATGAC 3'	Tsilo et al., 2008; Yu et al., 2010
		Xwmc477	SSR	F 5' CGTCGAAAACCGTACACTCTCC 3' R 5' GCGAAACAGAATAGCCCTGATG 3'	
		Xstm773-2	SSR	F 5' ATGGTTTGTTGTGTTGTGTGTAGG 3' R 5' AAACGCCCCAACCACCTCTCTC 3'	
Sr39	2B	Sr39#22r		F 5' AGAGAAGATAAGCAGTAAACATG R 5' TGCTGTCATGAGAGGAACTCTG	Mago et al., 2009
		Be500705		F 5' ATCTGTGGCAGTGTGCTCCT R 5' TCCTGCAAATGCTTGTCGTT	
		Sr39#50s		F 5' CCAATGAGGAGATCAAAACAACC R 5' CTAGCAAGGACCAAGCAATCTTG	

Sr40	2BS	Xgwm344,	SSR	F 5' CAAGGAAATAGGCGGTAACT 3' R 5' ATTTGAGTCTGAAGTTTGCA 3'	Yu et al., 2009; 2010
		Xwmc661	SSR	F 5' CCACCATGGTGCTAATAGTGTC R 5' AGCTCGTAACGTAATGCAACTG	
		Xgwm374	SSR	F 5' ATAGTGTGTTGCATGCTGTGTG 3' R 5' TCTAATTAGCGTTGGCTTGCC 3'	
		Xwmc474	SSR	F 5' ATGCTATTAAACTAGCATGTGTCG R 5' AGTGGAAACATCATTCCTGGTA	
Sr44	7DS	Wpt2565	DArT	F 5' TACTTTGATTTGGTCAGTTG R 5' TCGCGACCAAGCTCTACAAT	Crossa et al., 2007
		Cdo475	RFLP	F 5' GACACATTGACCGCATCTTA R 5' CCTTCACCTCGCTCCCTACC	Yu et al., 2009
Sr45	1DS	Xwmc222	SSR	F 5' AAAGGTGCGTTCATAGAAAATTAGA R 5' AGAGGTGTTTGAGACTAATTTGGTA	Yu et al., 2009
		Xcfa2158	SSR	F 5' TTTCGTCTTCAAAATGCACTG R 5' TGGTAGCTTACAAAGGTGCG	
Sr50 (R)	1DL/1RS	AW2-5		F 5' GAATCCCATTGTTCAGCAAGT 3' R 5' TAGCACTCCAGCAGACTCCAC 3'	Anugrahwati et al., 2008
		CI2F	RFLP	F 5' AGGGTCACACAGGCAATCTAA 3' R 5' CATTCTGGTTTTCCGCAGCAAC 3'	Mago et al., 2004
		1B-159		F 5' AGCGCAGATAATGTTTGAACC 3' R 5' AAGTCGAAACCACAGTTATC 3'	Mago et al., 2004
		1B-267		F 5' GCAAGTAAGCAGCTTGATTTAGC 3' R 5' AATGGATGTCCCGGTGAGTGG 3'	Mago et al., 2004
		Xmwg060	STS	F 5' CAACGATACAACAGGCTCAA R 5' CTGGATAGAGAAGCCATGGA	Mago et al., 2004
Sr52		BE497099-STS	STS	F 5' TTCGCTCCACCAGGAGTCTA 3' R 5' GTGTCTCGCCATGGAAGG 3'	Qi et al, 2011; Röder et al., 1998
		WMS570/ Xgm570	SSR	F 5' TCGCCTTTTACAGTCGGC 3' R 5' ATGGGTAGCTGAGAGCCAAA 3'	

Table 6. List of available yellow rust markers

Gene	Chromosome	Marker	Type	Sequence or Primer Pair	Reference
Yr1	2A, 2AL	Stm673acag		F5’ TAACTCACAACACGTTCTGGTCGT 3’ R5 ACACACACACACACAGAGAGAG3’	Bansal et al., 2009; Bansal, 2011
Yr4	3BS	Xbarc75	SSR	F5' AGGGTTACAGTTTGCTCTTTTAC 3' R 5' CCCGACGACCTATCTATACTTCTCTA 3'	Bansal et al., 2010; Bansal, 2011
		Cfb3530	SSR	F5’TTGTGCTTGTGCTACTATTACC 3’ R5’CAACATCTTACTGCTAACGTCC3	
Yr5	2BL	Xgwm501	SSR	F5' GGCTATCTCTGGCGCTAAAA 3' R5' TCCACAAACAAGTAGCGCC 3'	Röder et al., 1998; Sui et al., 2009
		Yr5STS7/8	STS	F5’ GTGTACAATTCACCTAGAG 3’ F5’ GCAAGTTTTCTCCCTAT 3’	Chen et al., 2003
		YrSTS9/10	STS	F5’ AAAGAATACTTTAATGAA 3’ R5’ CAAACTTATCAGGATTAC 3’	Bansal, 2011
Yr7	2B, 2BL	Xgwm526-2B	SSR	F5' CAATAGTTCTGTGAGAGCTGCG 3 R5' CCAACCCAAATACACATTCTCA 3'	Yao et al., 2006
Yr9	1B=1BL.1RS	Iag95	STS	F5' CTCTGTGGATAGTTACTTGATCGA 3' R5' CCTAGAACATGCATGGCTGTTACA 3'	Mago et al., 2002
Yr10	1B, 1BS	Xpsp3000	SSR	F5' GCAGACCTGTGTCATTGGTC 3' R5' GATATAGTGGCAGCAGGATACG 3'	Wang et al., 2002
Yr15	1BS	Xgwm11	SSR	F5' GGATAGTCAGACAATTCTTGTG 3' R5' GTGAATTGTGTCTTGTATGCTTCC 3'	Röder et al., 1998; Bansal, 2011
Yr17	2AS-6M	SC-372	SCAR	F5’ ATGTCCGCCCTTCCACAACTC 3’ R5’ CACTTGCCTATAAGCACAGAG 3’	Jia et al., 2011
		SC-385	SCAR	F5’ CTGAATACAAACAGCAAACCAG 3’ R5’ ACAGAAAGTGATCATTTCCATC 3’	
		2NS specific	VENTRIUP LN2	F5’AGG GGC TAC TGA CCA AGG CT 3’ R5’ TGCAGCTACAGCAGTATGTACACAAAA 3’	Helguera et al., 2003
		Xcmwg682-2A	STS	F5' GCTCACTGCTTCGGAAAACAACGAC 3’ R5' ATAGCACCTCCAAAATAAGAGCCTT 3’	
Yr18	7D, 7DS	Xgwm294	SSR	F5' GTGAAGCAGACCCACAACAC 3' R5' GACGGCTGCGACGTAGAG 3'	Spielmeyer et al., 2005
		CsLV34		F5' GTTGGTTAAGACTGGTGATGG 3' R’5' TGCTTGCTATTGCTGAATAGT 3'	Bansal, 2011
Yr24	1BS	Xbarc187	SSR	F5' GTGGTATTTCAGGTGGAGTTGTTTTA 3' R5' CGGAGGAGCAGTAAGGAAGG 3'	Li et al., 2005

MOLECULAR CHARACTERISATION AND GENETIC ANALYSIS

Mapping populations are various types of populations that show variation between population's phenotypes having a certain target trait. There are many types of mapping population which can be used for linkage mapping and QTL analyses, and some types of mapping populations are commonly utilized e.g. F_2 Populations, Backcrossing Populations, Recombinant Inbreed Lines (RILs) Populations and Double Haploid Populations (Collard et al., 2005; Scott, 2012). Moreover, the different types of molecular markers and their genetic behavior are given in Table 5.

F_2 population

F_2 population for mapping is produced by selfing or intercrossing of heterozygous F_1 which are developed from a cross between a resistant and a susceptible parent. The F_2 population is the most commonly utilized population in linkage mapping because of the short time required for production, and also such a population is easy to develop. The segregating ratios of an F_2 population are expected to be 3:1 for dominant marker and 1:2:1 for a co-dominant marker (Table 5). The disadvantage of the F_2 population is that the genetic constitution will change during sexual reproduction. Thus, the genetic structure of an F_2 population is difficult to maintain, and therefore F_2 populations cannot be used for replicated trial (Collard et al., 2005; Zhang, 2012).

Backcross populations

Backcross populations are generated by crossing the F_1 with either of the parents, and such populations are also widely used as mapping populations. A backcross population is similar to an F_2 population in terms of that the genetic constitution will change by selfing. However, the segregation will be for co-dominant marker 1:1 and dominant marker 1:0 (Collard et al., 2005; Kooke et al., 2012) (Table 5). Also, backcross populations cannot be used as repeated trials (Table 5).

Recombinant Inbred Lines (RILs)

RILs are produced by continuous selfing or sib mating of individual members of an F_2 population by single seed descent (SSD) until complete homozygosity is achieved. The major disadvantage of RILs is the need of six to eight generations for production of such lines. The major advantages of RILs are that they produce homozygous individuals that can be multiplied and reproduced without occurrence of the genetic change. Moreover, the genetic

distances based on RILs population is broader as compared to when F_2 is used. Accompanied to backcross and Double Haploid populations, many generations of selfing or sib mating will increase the chance of recombination. RILs can be used for replicated trial (Collard et al., 2005; Pollard, 2012).

Double Haploid (DH) Population

The DH population is an attempt to combine the advantages of homozygosity with the speed of creating an early generation population. Heterozygous F_1 will be used to produce gametes in which the chromosome numbers are artificially doubled by colchicine treatment and anther culture. DH populations are homozygous and can be self-pollinated to produce large numbers of materials. The expected ratio for marker will be 1:1 whether dominant or co-dominant (Matzk and Mahn, 1994; Collard et al., 2005) (Table 5).

Table 5. Genetics characterization of different markers in mapping population (Collard et al., 2005)

Marker type	Nature	Polymorphism	Cost	Segregation Ratio			
				$F_{2;3}$	RIL	DH	BC_1
SSR	Co-dominant	Medium	Medium	1:2:1	1:1	1:1	1:1
SNP	Co-dominant	Medium	Low-Medium	1:2:1	1:1	1:1	1:1
RAPD	Dominant	Low	Low	3:1	1:1	1:1	1:0
AFLP	Dominant	Medium-high	Medium	3:1	1:1	1:1	1:0
RFLP	Co-dominant	Medium-high	Medium	1:2:1	1:1	1:1	1:1

Molecular Markers in Plant Breeding

Conventional plant breeding methods have made a significant contribution to crop improvement, but conventional breeding has also been slow in targeting complex traits. Conventional plant breeding is dependent upon genetic variation and phenotypic identification and visual selection of agronomic traits. The past years developments of molecular marker tools have revolutionized the genetic analysis of crop plants. Furthermore, molecular tools have also successfully been applied in plant breeding for identification of targeted traits (Patnaik and Khurana, 2001). Molecular markers were developed to be utilized for improving the efficiency of conventional plant breeding by being linked to genes for the targeted traits. By utilizing the molecular marker approaches, breeders can save time, resources and energy to produce cultivars with improved characteristics and traits. According to Tanksley (1983) molecular markers are valuable in discriminating five inherit properties i.e. 1) genotypes can be determined by the molecular loci at any plant tissue and cellular levels; 2) a relatively large number of naturally occurring alleles can be found at the molecular marker loci; 3) deleterious effects are not associated with alternate alleles of a molecular marker; 4) alleles at most molecular/loci are co-dominant, to allow all possible genotypes to be distinguished in any segregating population; 5) few epistatic or pleiotropic effects are produced, thus a very large number of segregating markers can be monitored in a single population.

There are two main types of molecular markers 1) Isozyme markers and 2) DNA based markers. The term 'Isozymes', was proposed by Markert and Moller (1959) and this type of markers are used to describe different molecular forms of bands possible to visualize for the same specific enzyme. DNA based markers can be used to study genetic variation, association and linkage/genetic mapping and QTLs detection. DNA sequences and/or segments that are closely linked to a gene locus and/or to morphological or other characters of a plant can be detected and visualized by molecular techniques. DNA based markers can be classified in the following groups:1) hybridization based markers (e.g. RFLP); 2) PCR-based molecular markers (e.g. RAPD, SSR); 3) molecular markers based on PCR followed by hybridization (RAPD/MP-PCR); 4) sequencing and DNA chip based markers (SNPs); 5) Diversity array technology (DArT) is a novel type of DNA markers which employs a microarray hybridization; and 6) Expressed Sequence Tags (EST) (Paterson et al., 1991; Jones et al., 1997; Gupta, et al., 1999; Qi et al., 2004; Xu, 2010).

PCR-based molecular markers

Polymerase Chain Reaction (PCR) was developed in 1983 by the American biochemist Kary Mullis and has become an essential technique widely utilized in molecular plant breeding. The idea of PCR is a simple process in which a specific segment of DNA is synthesized repeatedly, resulting in the production of large amounts of a single DNA sequence (Saiki et al., 1985). PCR-based DNA markers such as random amplified polymorphic DNAs (RAPDs), can also be converted into sequence characterized amplified regions (SCARs). The other widely used types of molecular markers are: Simple sequence repeats (SSRs) or microsatellites, sequence-tagged sites (STS), amplified fragment length polymorphisms (AFLPs), inter simple sequence repeat amplification (ISSR), DNA amplification fingerprinting (DAF), cleaved amplified polymorphic sequences (CAPs) and amplicon length polymorphisms (ALPs). In this review, focus will be on SSR and SNP markers, because they are currently widely used in wheat breeding for mapping purposes.

Simple sequence repeats (SSR)

SSR, also known as microsatellites are tandemly repeated units of short nucleotide motifs (1-6 base pairs long) such as di-nucleotide $(CA)_n$, tri-nucleotide $(AAT)_n$ and tetra-nucleotide $(GATA)_n$ repeats, which are extensively disseminated throughout the genomes of plants and animals (Tautz and Renz, 1984; Xu, 2010). Tautz et al. (1986) observed that microsatellites show a high frequency of variation in the number of repeats in different organisms, possible due to slippage during DNA replication. Therefore, this type of polymorphism at particular loci is easy to discover with special primer pairs in the flanking regions using PCR to amplify microsatellite alleles (Litt and Luty 1989). SSR analysis is based on individual PCR amplification of DNA fragments with specific oligonucleotide primer pairs designed complementary to unique DNA sequences flanking the SSR sequence (Xu, 2010).

The polymorphism between different organisms is due to allelic variation in the number of repeat units, which are composed of 1-6 bp short DNA sequences e.g. di-nucleotide $(CA)_n$ and tri-nucleotide $(AAT)_n$ repeats (Li et al., 2002). SSR markers are defined by their hypervariability and the reproducibility is mostly co-dominant and multiallelic, which make them easily transferable between genetic maps of throughout crosses of related species (Xu, 2010). Hexaploid wheat has a large genome and discovers high level of polymorphism in SSR loci amplified with locus-specific primer pairs. Multiple alleles in the SSR loci are inherited co-dominantly. Microsatellite markers detect much higher levels of variability compared to the

markers used previously e.g. AFLPs, RFLPs, therefore a number of microsatellite markers have been made available for wheat (Röder et al., 1995; 1998). Furthermore, SSR have been found valuable as genome-specific genetic markers in hexaploid wheat and rye (Devos et al., 1995; Röder et al., 1998; Khlestkina et al., 2004).

Reason for the particular preference for the microsatellite markers in wheat is the genome specificity, which makes it possible to analyze the three homologous genomes A, B and D of bread wheat (Pestsova, et al., 2000). Moreover, the SSR markers are simply transferred among wheat mapping populations, since they are able to detect specific loci in various genetic backgrounds, which is a useful tool for determination of the chromosomal identity of unknown regions of linkage groups (Röder et al., 2004). Microsatellites can be obtained by screening sequences in different databases or by screening libraries of clones (Xu, 2010). Several SSR loci have been detected and mapped in bread wheat and rye (Saal and Wricke, 1999; Gupta et al., 2002; Miroslaw and Chelkowski, 2004; Hayden et al., 2006), and also the SSR markers have been used to tag several genes and QTLs in wheat and rye.

Single Nucleotide Polymorphism (SNPs)

Brookes, (1999) determined SNPs as being single base pair position genomic DNA at which different sequence alternatives (alleles) exist in normal individuals of a population, wherein the least frequent allele has an abundance of 1% or greater. SNPs can be biallelic, triallelic or tetra allelic polymorphisms genetic markers and exclude single base insertion/deletion variants (Brookes, 1999). The simple inheritance of SNPs is due to an individual nucleotide base distinction among two DNA sequences. Nucleotide substitutions are classified based at either transitions (C/T or G/A) or transversions (C/G, A/T, C/A, or T/G) (Edwards et al., 2007). For instance, sequenced DNA fragments from two different organisms can be AAG**C**TA to AAG**T**TA. Transitions such C/T constitute 67% of the SNPs observed in humans, and also more of less the same rate have been identified in plants (Rafalski 2002; Batley et al., 2003; Edwards et al., 2007).

SNPs have been discovered to appear with a frequency as high as one in every 202 base pairs in the genome of mouse and one in 1000 base pair in the human genome (Brookes, 1999; Lindblad-Toh et al., 2000). The development and use of allele-specific PCR-primers would be preferably due to its simplicity, low cost and reproducibility of genotyping SNP (Lee et al., 2004; Hayashi et al., 2004). By this approach, SNPs can be identified simply using allele-specific PCR primers designed by the 3’ terminal nucleotide of a primer corresponding to the

site of the SNPs. Genotyping individuals using SNPs needs a plus/minus assay permitting easier automation, and also available high density oligonucleotide arrays on DNA chips (Gupta, et al., 1999). PCR-amplified products can be run on a standard agarose gel (Hayashi et al., 2004; Lee et al., 2004). Through sequencing of PCR-amplified products from a number of diverse individuals, DNA polymorphisms can be detected in a more straight forward way compared to when other types of DNA markers are used. Most other types of DNA markers are based on an indirect detection of sequence-level polymorphisms (Rafalski, 2002). Designed PCR primers are either derived from known DNA sequences of genes available from public Gene Banks, or from expressed sequence tags (ESTs) (Rafalski, 2002). Suitable and available SNP markers from EST sequences can be selected from the NCBI and wheat SNP databases (http://www.ncbi.nlm.nih.gov/dbEST/dbEST_summary.html; http://wheat.pw.usda.gov/NSF/), to be used for linkage mapping and QTL analysis. The use of the SNPs system, has led to rapid advancement in the development of the human genetic map, and currently offers rapid and highly automated genotyping (Gupta et al., 1999). An Illumina iSelect genotyping array was developed with 9,000 SNPs for the advancement of wheat breeding in order to understand the complex traits (Cavanagh et al., 2013; Saintenac et al., 2013). However, genotyping-by-sequencing (GBS) has been also introduced more recently, which is a low cost and excellent method to explore the genetic diversity in plant breeding and genetics (Poland et al., 2012; Poland and Rife, 2012).

Bulk Segregant Analysis

The bulked segregant analysis (BSA) is an alternative method for linkage mapping and QTL analysis. BSA is developed for rapid identification of markers linked to a specific gene of a genome region. The advantages of using BSA are that the markers can be discovered in short time with reduced costs (Michelmore et al., 1991; Collard et al., 2005). However, to apply BSA, the availability of a mapping population is required (Collard et al., 2005). In the BSA, DNA pools of individuals of a crossing progeny are used, selected based on their phenotype and through screening for differences in molecular markers (Michelmore et al., 1991). First, the markers polymorphism between parents must be identified, and thereafter F_2 resistant and susceptible bulks are screened with selected markers. Once linkage is discovered, additional markers in the chromosomal region are evaluated for the development of a linkage map.

Linkage Map and Genetic Distance

Genotyping of mapping populations could be performed with any marker approaches, e.g. SSR, SNP etc. Linkage mapping is based on genetic distances in a map, delineated from recombinant frequencies and expected number of meiotic crossover events between any two loci. Genetic distances are measured in centiMorgan (cM) (Haldane, 1919; Kosambi, 1944; Collard et al., 2005). Two different mapping functions, the Haldane mapping function and the Kosambi mapping function, are commonly used. According to Ott (1985), the Haldane mapping function considers the occurrence of multiple crossovers, but the Kosambi mapping function is the phenomenon of one crossing over which is preventing the formation of another in its neighborhood. The linkage map based on genotyped markers and their pairwise recombination frequencies can be constructed using various statistical softwares, although the common one is MapMaker (Lander et al., 1987). The linkage between markers is usually calculated using odds ratios (Collard et al., 2005). Linkage analysis can be carried out by evaluating F_2 populations, backcrossing populations, double haploid lines, recombinant inbred lines etc., which the mapping population using the two-point analysis to identify linkage group at a logarithm of odds (LOD) score of 3.0 (Risch, 1992). Several maps based on SSRs and SNPs have been developed for wheat and rye (Saal and Wricke, 1999; Khlestkina et al., 2004; Varshney et al., 2007; Akhunov et al., 2009).

Quantitative Trait Loci (QTL) analysis

The major agronomic traits e.g. flowering time, root morphology, yield, biotic and abiotic stress tolerances etc., are regulated by several genes or QTLs. QTL analysis is a powerful tool for identifying genes with major and minor effects via genetic linkage mapping in order to locate their specific chromosomal regions (McMullen, 2003). The principle of QTL analysis is based on phenotypically evaluated traits that are compared with molecular markers using different statistical software (Collard et al., 2005). The determination of the position of the QTL underlying a trait has three substantial steps: 1) a segregation population is developed and genotyped with molecular analysis; 2) the individuals of the same population are phenotypically characterized for the traits under investigation; 3) the genotypic molecular characterization are analyzed for association with the phenotypic trait data by using statistical methods (Doerge, 2002).

There are several methods for detection of QTLs available: single-marker analysis; simple interval mapping; composite interval mapping; multiple interval mapping; Bayesian analysis

(Tanksley, 1993; Zeng, 1993; Liu, 1998; Kao et al., 1999; Yi and Shriner, 2008). The single-marker analysis is the simplest method for determining QTL associated with a single marker. QTL mapping could be associated with single marker analysis, which can be implemented as a single marker with t-test, ANOVA, linear regression and likelihood approaches to detect the genetic markers that are close to a QTL (Sax, 1923; Collard et al., 2005). However, using single marker analysis the QTL positions cannot be precisely determined, because the QTL effect and the location are confounded (Doerge, 2002). Simple interval mapping is used for the linkage mapping and characterization of intervals between adjacent pairs of linked markers in a single chromosome simultaneously, instead of analyzing of single markers (Collard et al., 2005). Lander and Botstein (1989), made available an interval mapping method using flanking markers that determines and localizes the QTL more precisely. Recently, multiple interval mapping and composite interval mapping have become more powerful and precise to find the significance of the two or three linked QTL (Zou, 2009; Xu, 2010). The composite interval mapping is aimed to fit genetic markers closely linked to other QTL across the genome as covariates. The multiple intervals mapping resembles the composite interval mapping, but they are completely different in terms of procedure. The multiple intervals mapping is aimed to fit estimated positions of other QTL rather than their closely linked genetic markers. Also, the multiple intervals mapping is fitted with multiple putative QTL effects and it is associated with epistatic influence (Xu, 2010; Silva et al., 2012). The Bayesian model in QTL mapping is adopted via the Markov Chain Monte Carlo algorithm, which has the potential to carry out linkage analysis with any number of marker loci, multiple trait loci and multiple genomic segments (Xu, 2010; 2013). The evidence for linkage to a QTL is measured by the logarithm of odds (LOD) score, to measure the strength of indication for the presence of a QTL at a special location (Blanco et al., 2006).

For statistical analyses several software are available e.g. Windows QTL Cartographer; R/qtlbim; QTL Network; SAS program (http://ibi.zju.edu.cn/software/qtlnetwork/; http://www.stat.wisc.edu/~yandell/statgen/software/biosci/qtl.html; http://www.statgen.ucr.edu/; http://statgen.ncsu.edu/qtlcart/WQTLCart.htm; http://www.qtlbim.org).

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