Review Article

Stripe Rust: A Review of the Disease, Yr Genes and its Molecular Markers

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Abstract | Wheat is the most essential food used by nearly 40% of the total population of the world. Yellow or stripe rust (produced by Puccinia striiformis), is a globally significant disease of wheat. Stripe rust was primarily considered a disease of cooler climate (2˚C - 15˚C), upper altitudes and northern latitudes, but current epidemics of the disease have confronted this supposition because fresh strains have greater adaptation to higher temperatures and countries closer to the equator. Crop damages can reach 50 - 100%, due to infected plants and shriveled grain. These problems can be overcome by knowledge about the disease, identifying resistance lines and subsequently develop resistant varieties with an aim to shorten the disease cycle. One of the quickest ways in this direction is the designing molecular markers for non-race-specific resistance genes. Use of molecular markers is efficient tool for screening diversity of rust genes in wheat germplasm and can facilitate the integration of multiple genes into wheat by pyramiding and transformation. This review discusses information regarding rust disease and resistance in wheat to tackle the disease through resistance breeding.

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Introduction

Wheat (*Triticum* spp.) belongs to family *Poaceae* and genus *Triticum*. There are two types of modern wheat species upon which the world wheat production depends. Normally bread wheat or common wheat with ploidy level hexa, and scientifically known as *T. aestivum*, having chromosome number 42 (42=2n =6x), with genomes AA, BB and DD (Goutamet al, 2013). Most of wheat genotypes (>80%) is attributed to this type (Stubbs, 1985). Another type is Durum wheat with ploidy level tetra and scientifically known as *T. turgidum* (2n=4x=28), with genomes AA and BB (Goutam et al, 2013).Wheat is considered as grain at the center of Indo-European civilization. It is eaten by almost 40% of the total world inhabitants (Goyal et al., 2010; Peng et al., 2011). Its nutritional composition helps in giving 21% of the total calories and nearly 20% of the protein to almost 4.5 billion human population on earth (Braun et al, 2010; Rahmatov, 2013).To overcome this huge diet requirement its cultivation and breeding is of great importance. Global trade of wheat is about US $50 billion each year. Wheat is grown on 215 million hectares each year, worldwide (CGIAR, 2017). This demand for wheat production would further expand with the in-

crease in the population (FAO, 2009). According to an estimate, the world population will touch nearly nine billion from existing 7 billion in 2-3 decades, for which an annual increase of 2% in wheat production is, must in order to ensure food security (Rahmatov, 2013). Retrospective of this increasing demand wheat stocks remain at decreased level (Dixon, 2009; FAO 2009). Due to land limitations, absolute higher yields must be ensured (Braun et al, 2010). As forecasted by climatologists, with the existing cultivars and practices, 30 % reduction in South Asia's wheat yield is expected (CGIAR, 2017).

The loss inflicted by wheat disease such as rusts that could be controlled by the joint efforts of scientists involved in diverse agricultural disciplines and plant breeding programs (Braun et al, 1998). Rust is a unanimous name for a group of disease, caused by *Puccinia* species (fungal pathogens) infecting cereal crops (wheat, rye, barley, and triticale) and grasses (Brown and Hovmoller, 2002). Documentation of rust disease on wheat is quite old. Depending upon the infection caused by *Puccinia* species, rust can be classified into three types (Table 1).

Table 1: *Rust types along with their causal agent.*

S.No.	Type	Causal agent
1	Leaf rust (Brown rust)	*Puccinia triticina*
2	Stem rust (Black rust)	*Puccinia graminis* f.sp. *tritici*
3	Stripe rust (Yellow rust)	*Puccinia striiformis*

The Rust fungus is abiotropic, obligate, parasitic organism that takes nutrition from their host plants (Chen et al., 2014) affecting the photosynthetic ability of the plant and consequently reducing plant height, floret set and grain yield. It also slow growth and low forage quality, poor seedling germination, foliar injury and shriveling of the grain (Roelfs et al., 1992; Chen, 2005). These characteristics make rust a major biotic production constraint for wheat (Singh et al 2004). Rust disease is highly prolific and its spores have the tendency to spread over long distances. It can grow anywhere on a suitable substrate (wheat species) under favorable conditions and can pose a persistent threat to all the sustainable wheat producing areas (Singh et al, 2004). Periodic epidemics of rusts in the 19th century caused famine in many parts of the world. About US $5 billion losses occur due to cereal rusts including wheat around the globe every year (Martinez et al., 2012). Yellow rust is caused by *Puccinia striiformis* f. sp. *Tritici* (*Pst*), which is one of the most eminent pathogen in several fragments of the globe including Pakistan (McIntosh, 1980; Singh et al.,2004). This review will discuss yellow rust in detail.

Types of pathogen

Black rust: Black or stem rust is caused by *Puccinia graminis* f.sp. *tritici*. Due to formation and presence of dark and shiny black spores at nearly start of stem, it is known as summer or black rust. Humid and wet warm season of about15˚C to 35˚C plays important role in developing and spreading of black rust. Black rust can cause 100% destruction by converting the susceptible crop in a dead mass of damaged and broken leaf and stems along with shriveled grains just a few weeks before the harvest (International Wheat Stripe Rust Symposium, 2011).

Brown rust (leaf rust): *Puccinia triticina* is the causal agent of brown rust. Brown rust is prevalent in major wheat grown areas. Favorable temperatures for leaf rust development ranges between10˚C and 30˚C. Leaf rust can cause up to 30% wheat yield losses (International Wheat Stripe Rust Symposium, 2011).

Stripe rust (yellow rust): Stripe rust (caused by *Pst*) of wheat is considered to be one of the most destructive disease. It is not limited to wheat only and can infect barley, rye and more than 50 grass species (Line, 2002). It has the potential to loss 100% wheat yield, if the susceptible cultivars become infected at an early stage and progress during the crop season. However, the level of losses is observed to fluctuate between 10-70 percent, depending upon the favorable weather conditions of an area, development of new races of pathogen, level of susceptibility of cultivar, initial infection at early growth stage, degree of disease progress and length of disease (Begum et al., 2014).The damage by stripe rust is inflicted in the form of decreased yield, grain quality and forage value (McIntosh et al., 1995).

Background of stripe rust: Although the existence of stripe rust is thought to be present long before human beings started to grow wheat as a food crop, in 1777 the first report on the disease was published by Gadd in Europe. In 1794, the disease was spread epidemically on rye in Sweden (Singh et al., 2002). Epidemics of stripe rust all around the world with massive yield limiting potential, makes it a globally known disease

marked with profound economic importance (Roelfs et al., 1992). Wheat stripe rust has shown its presence in nearly 60 countries except Antarctica (Stubbs, 1985; Singh et al., 2002). In Asia about 46% yield losses are due to the epidemics of stripe rust (Singh et al., 2004). It is therefore crucial to devise ways to control stripe rust. Cultivation of wheat cultivars that show resistance towards the stripe rust disease seems to be the most effective, inexpensive and environmentally harmless control measure for yellow rust epidemics (Line and Chen 1995; Zhang et al. 2009).

Classification of stripe rust

The pathogen is classified and placed in kingdom- Fungi, phylum- Basidiomycota, class- Urediniomycetes, order- Uredinales, family- *Pucciniaceae*, genus- *Puccinia* (Chen et al., 2014). The nomenclature for the wheat stripe rust pathogen underwent a series of changes before finally being named *Puccinia striiformis Westend (Ps)*

- In 1827, Schmidt described the pathogen infecting barley glumes as *Uredoglumarum*.
- Later in 1854 Westend named the pathogen as *Puccinia striiaeformis* (with reference from rye).
- In 1860 Fuckel named it *Puccinia straminis*.

Then leaf rust was confused with yellow rust.

- However in 1894, Eriksson and Henning identified yellow rust as a distinct rust disease and was termed as *Puccinia glumarum.*
- The term was reviewed again in 1953 and was changed to specials of *Puccinia striiformis*

Apart from wheat species, *Puccinia striiformis westend* (Ps) can infect members of rye, barley and 59 grass species (Line, 2002). Based on this knowledge *Puccinia striiformis westend* (Ps) is further divided into nine formae specials based on host response on various grass genera and species. Chen with his colleagues are the pioneer of reporting *P. striiformis* into five formae specials (Chen et al, 2014). Some of the main species with host (Table 2).

Table 2: *List of important P. striiformis with their host plant.*

S.No	Specie	Host specialization
1	*P. striiformis* f. sp. *tritici*	wheat (Causative agent of Yellow/Stripe rust)
2	*P. striiformis* f. sp. *Hordei*	Barley (Chen et al., 2014)
3	*P. striiformis* f. sp. *Secalis*	Rye (Chen et al., 2014)
4	*P. striiformis* f. sp. *Elymi*	*Elymus* spp. (Chen et al., 2014)
5	*P. striiformis* f. sp. *agropyron*	*Agropyron* spp (Chen et al., 2014)
6*	*P. striiformis* f. sp. *Dactylidis*	Orchard grass (Manners, 1960;Tollenaar, 1967)
7*	*P. striiformis* f. sp. *Poae*	Kentucky blue grass (Tollenaar, 1967)
8*	*P. striiformis* f. sp. *Leymi*	*Leymussecalinus* (Georgi) Tzvel (Niuet al., 1991).

** Later, three more form of a specials were reported*

Disease symptoms

Infection: Fungus such as *Pst* only infests the green parts of vulnerable plants: leaves, leaf sheaths, glumes and awns (Chen et al., 2014). It can infect wheat anytime from young single leaf plant to mature plant provided they stay green (Chen, 2005).

Urediniospores: The bright yellow to orange Urediniospores of the fungus is about 20 to 30 um in diameter with thick and echinulate walls. These spores have the ability of rapid germination provided that free moisture is available in surroundings and on leaf surface along with optimum temperature range 7 to 12˚C. With an increase in temperature or through the late growth phases of the host, Urediniospores production is usually followed by two-celled, dark brown, thick walled black teliospores (Alfredo et al., 2012) Figure 1.

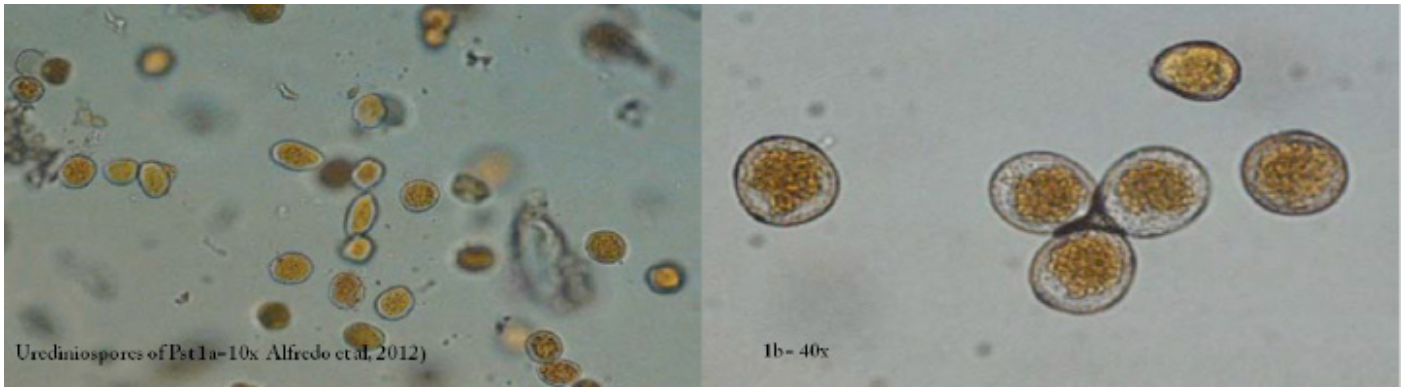

Figure 1: *Urediniospores under a microscope (Alfredo et al., 2012).*

Figure 2: *Pustules of Pst on wheat leaf (Alfredo et al., 2012).*

Uredia: (Urediniospores are contained in pustules (sori) Figure 2.

Primary symptoms of stripe rust: Chlorosis or Necrosis (hypersensitive response) results as a symptom of the disease in plants showing resistance (Line and Qayum, 1992; Chen, 2005). The intensity of response depends upon the level of plant resistance and temperature, with or without sporulation (Figure 3).

Life cycle : For more than a 100 years, life cycle of *Pst* is unknown (Jin et al., 2010). The *Pst* and basidiospores has four phases in its lifecycle which requires two hosts for its completion (Chen et al., 2014).

1. **Uredial and Telialphases:** which takes place in the primary host (wheat/ grasses).
2. **Pycnial and Aecial phases:** taking place in the alternate host (Berberis or Mahonia spp).

At the end of 19thcentury, uredinial and telial were the only stages of yellow rust which were understood. However, recently, Berberis spp. (*B. chinensis, B. holstii, B. koreana* and *B. vulgaris*) have been studied to find out an alternate host of stripe rust. Later on it was put under the category of rust fungus *Puccinia striiformis* which was confirmed by the molecular technique of DNA sequencing and real time PCR (Jin et al., 2010).

Favorable conditions for yellow rust: The development and spread of stripe rust depends upon three most significant features of weather which includes wind, moisture and temperature (Chen, 2005).

Moisture: Spore germination, infection, and survival of rust greatly depend upon the moisture content. Urediniospores ultimately requires at least 3 hours of continuous moistness on the surface of plant where it will grow and cause infection (Rapilly, 1979).Moist conditions with due formation in growing season provides favorable condition for development of stripe rust. High moisture induces spore germination, but on the other hand it can also badly affect the survival of the urediniospores as they lose viability more quickly under high-moisture environments than under dry

Figure 3: *Stripe rust symptoms (A-B), mature symptoms of Stripe rust (C) (Alfredo et al., 2012).*

conditions (Chen, 2005).They lack the ability of fungistasis and can germinate immediately after they are produced provided that moist and optimum temperature is present, because of this reason shipment and storage of wheat becomes quite a difficult task. Moisture also affects spore dispersal. High humidity increases the cluster dispersal of urediniospores rather than individual dispersal (Chen, 2005).

Temperature:
Temperature affects initially the germination and growth of these spores, their infection capability, sporulation, spore survival, host-plant resistance relation and latent period (Rapilly, 1979). Stripe Rust principally attack wheat grown in cooler climate; require temperate zone and parts of high altitude in tropical sections. But the recent attack of some species on the wheat grown in dry areas shows its adaptation to high temperature (International Wheat Stripe Rust Symposium, 2011). The minimum of 3˚C and the maximum of 20˚Ctemperature have been observed for the growth of pathogen (Line, 2002). Exceedingly low temperature in winters can reduce the survival of stripe rust pathogen by destroying the causal agent in the infected areas of plant (leaf). Moreover temperature lowers than –10 ˚C can completely stop the pathogen from growing (Rapilly, 1979). Knowledge of temperature at a specific location can give information to predict the presence or absence of stripe rust (Chen 2005).

Wind: Wind plays important role in scattering rust spores magnanimously. Wind maximizes the period of *P. striiformis* spore viability by drying Urediniospores (Roelfs et al., 1992). Wind is one of the important tools for spreading spores over wide range but timing holds the key and direction significantly affect the premature, scales and development rate of pathogen (Chen, 2005).

Environmental factors

Airborne: It is difficult to control yellow rust because its Urediniospores could be easily taken away by the wind to travel to long distances, with the possibility of migrating from continent to continent. Migration of pathogen can change the virulent nature according to the area of its establishment (Brown and Hovmoller, 2002). It is assumed that *Puccinia striiformis* is innate to Caucasus (Georgia, Armenia, and Azerbaijan) and subsequently dispersed to Europe, China and Eastern Asia (Humphrey et al., 1924; Stubbs, 1985; Line, 2002).

Temperature adaptive: Historically, the epidemics of yellow rust have been reported at high altitudes and moderate cold regions (Stubbs, 1985; Zadoks and Vandenbosch, 1995). In recent years, new races of yellow rust were able to adapt the environment significantly including warmer areas and they were tolerant and aggressive to high temperatures (Milus et al, 2009).

Variable virulence: Virulence is the capability of a pathogen to defeat a particular genetic resistance for any particular gene in plant and it may not be limited to single gene, it can be for multiple genes (Flor, 1971; Brown, 2003). Evolution of new races of *Puccinia striiformis (Ps)* through mutation, somatic recombination and sexual recombination, produces an obvious change in the virulence of the pathogen, making the disease pathogen more adaptive to overcome the plant defense (Stubbs, 1985; Jin et al., 2010; Roelfset al., 1992; Chen, 2005). Mutation played a pivotal role in the development of novel races of *Ps* and defeating the defense system of a variety a short period (Chen, 2005). The virulence studies of *Pst* have a long history showing eminent virulent spectra. Each of the factors involved in the pathogen variability is discussed below.

Mutation: Mutation is a naturally occurring unpredictable alteration in the genetic sequence of the living organism. Mutations can range from small single nucleotide change to larger chromosomal translocations, inversions, additions and deletions. The first report on the change of yellow rust virulence due to mutation was made in 1932 (Robbelen and Sharp, 1978).Surveys held in growing areas of wheat at different locations reveled that mutation is the main source for originating the new varieties of rust (Watson, 1981; McIntosh, 1988).

It is also called parasexualism and this phenomenon commonly occurs in stripe rust whose presence has been confirmed under greenhouse conditions (Braun et al., 1998; Stubbs, 1985). This phenomenon involves changes in the normal dikaryotic organization of the nuclei inside the cell. Somatic hybridization may result from the co-existence of genetically different nuclei in a cytoplasm, segregation and recombination at mitosis and fusion of unlike nuclei in the hyphae. This phenomenon gives rise to new variations influencing pathogenicity and host range. Research suggests that somatic hybridization or parasexualism arises when

specific races raise collectively on the host (Watson, 1981).

The sexual stage and the alternate hosts of *Pst* continued to be a secret for a long time. Berberis spp. was identified as an alternate host recently. The diversity in the virulence of stripe rust can be due to sexual fertilization in wheat growing areas along with susceptible barberry species (Jin et al., 2010). This phenomena majorly contributes to the yellow rust pathogen diversity but is not yet entirely understood. For controlling of stripe rust one must understand its process of sexual reproduction (Jin et al., 2010; Mboup et al., 2009).

Stripe rust races: The formae specials of *Pst* are further categorized into different forms depending on their virulence level to wheat cultivars or genotypes. Furthermore these types are grouped together by their way of infecting plant material. The occurrence of "specialized varieties" in *Ps* on the basis of host specificity were first reported by the Hungerford and Owens (1923), whereas the presence of these races in *Pst* established on specificity of wheat cultivars were first mentioned Allison and Isenbeck (1930).

Resistance through conventional breeding: The presence of genetic resistance in wheat towards *Puccinia striiformis* was resolved for the first time by Biffen in 1905. He described multiple resistant types for stripe rust. Chen (2013) grouped the types of resistance based on:

1. Growth stage (All stage [seedling] resistance, Adult plant resistance).
2. Testing condition (green house, field).
3. Specificity (Race specific, race non-specific).
4. Degree of resistance (Absolute, relative).
5. Sensitivity to pathogen infection (Hypersensitive, non-hypersensitive).
6. Speed of symptom/sign development (Fast rusting, slow rusting).
7. Response to temperature (Temperature sensitive, temperature non sensitive).
8. Inheritance (qualitative, quantitative).
9. Effect of genes (Major, minor).
10. Number of genes (Monogenic, Polygenic).
11. Molecular basis (NBS-LRR type resistance, non NBS-LRR type resistance).
12. Durability (Non-durable, durable).
13. Race specificity, Growth stage and temperature sensitivity (Race-specific all-stage resistance, non-race specific high-temperatureadult-plant (HTAP) resistance).

We can control stripe rust by taking advantage of naturally occurring resistance in wheat cultivars. Resistant variety can be developed for race specific or for broad spectrum multiple race resistance if none of the rust can be identified. One can develop qualitative or quantitative resistance against wheat. To date cultivation of resistant cultivars seems like the best approach to limit stripe rust epidemics. On the basis of inheritance resistance can be divided into two categories as shown in the Table 3 below (Flor, 1956; McIntosh, 1988; 1995; Rajaram et al., 1988; Singh et al., 2000; Parlevliet, 2002; Chen, 2005; Clair, 2010; Lowe et al., 2011):

Stripe rust resistance gene: In 1962 Lupton & Macer worked on seven wheat cultivars to understand the effect of seedling-expressed resistance and introduced the catalog of *Yr* genes i.e. assigned *Yr* symbols to stripe rust resistance genes. More than 70 genes have been named as *Yr* followed by a number, letter or symbol (Chen, 2005). Many reported stripe rust resistance genes need to be named (Chen et al., 1998; Chen, 2002).These *Yr* genes confer different types of resistances (Table 4).

To utilize the genetic diversity of a particular germplasm for the improvement of its crop, the knowledge of genetic diversity of its germplasm is important and must be investigated. Nowadays these molecular markers, because of their accuracy and reliability, have become one of the best tools for identifying the genetic diversity in many plant and animal genotypes. These can also provide detailed characterization of genetic resources (Zhang et al., 2001).

Resistance through molecular markers

DNA based molecular markers are short DNA sequences that can identify the location of a particular gene and could be used in plant breeding for identification of targeted traits. Once conventional plant breeding methods greatly contributed to the crop improvement, but it was slow in targeting complex traits as it was dependent upon phenotypic and visual selection of morphological characters. In recent decades the use and development in the field of molecular techniques and use of DNA based markers have reached a new high level and it has brought revolution in the field of genetic world and crop plant analysis (Patnaik and Khurana, 2001).

Table 3: *Types of resistance in wheat (Triticumaestivum) against yellow rust disease (Pucciniastriiformisf. sp. Tritici) on the basis of inheritance.*

Qualitative resistance	**Quantitative resistance**
Qualitative inheritance, it has two classes one susceptible and other resistant.	Quantitative inheritance, it has multiple classes ranging from complete resistance to complete susceptible.
Monogenic (major genes)	**Polygenic (minor gene)**
Only single gene controls the resistance with a large effect.	Multiple genes control the resistance with each gene having minor effect.
Hypersensitive	Nor hypersensitive
Resistance characterized by the localized induced cell defense response of host plant at the site of infection of a pathogen. Symptoms are minute flecks (necrotic tissue) that are indicative of high resistance.	Either completely resistant (immune) or reduced severity, but susceptible infection type (e.g. slow-rusting)
Non-durable resistance	**Durable**
Resistance is "broken-down" by some races	Resistance that remains effective in a cultivar during its prolonged and widespread use in an environment that favors the disease
Fast rusting	**Slow rusting**
Rust develops fast and quickly reaches the highest level.	Plants have a susceptible infection type but disease progressesslowly on them.
Race-specific or vertical resistance	**Race-nonspecific or horizontal**
The gene-for-gene relationship states that for every resistance gene in the host plant there is a corresponding virulence gene in the pathogen that can mutate to a virulent form. Mutated virulence of a specific race of a pathogen no longer recognizable by the resistant gene of plant can overcome the race-specific resistance (Flor, 1971). Race specific resistance is completely effective against some races, but not others. It is governed by a hypersensitive response, controlled by major genes. It is also known as monogenic resistance. (Dyck and Kerber, 1985; Nagarajan and Joshi, 1985;Priyamvada and Tiwari, 2011)	Resistance is effective against all races. It is characterized by reduced apparent infection rate (slow rusting) (Van der Plank, 1968). This type of resistance is inferred by polygenes or quantitative genes and is mostly carried out by adult plants (Roelfset al., 1992). It is a durable type of resistance (Parlevliet, 1985; McIntosh et al., 1995).
These two are major types of resistances to Pst in Wheat.	
All stage (seedling) resistance	**Adult plant (field) resistance**
Resistance can be detected in the seedling stage, but remains effective throughout all growth stages. This type of resistance is race specific and can lose easily due to emergence of new patho-types through mutation and recombination (Line and Qayoum, 1992; Line and Chen 1995, 1996; Jin et al., 2010).	Adult plants with resistance are susceptible in the seedling stage but can develop varying levels of resistance in late stages. Being non race specific it is considered more robust in terms of resistance. The resistance is implied by minor genes, which may not be overcome. High temperature adult plant resistance (HTAP) Resistance is effective against all races when plants grow old and temperature increases.

By using DNA, molecular markers we can easily detect the gene for resistance in plant even at seedling stage and can screen the plant for rust resistant gene and thus can ultimately cultivate genetically diverse and resistant varieties of wheat. Characterization of wheat genotypes for Yr genes by using molecular markers and subsequently utilizing these screened germplasm for genes pyramiding can be used to improve and enhance the rust (stripe) resistance (Begum et al., 2014). DNA based markers are useful for studying genetic differences, genetic association, linkage/genetic mapping and QTLs detection (Rahmatov, 2013).

Table 4: *Resistance genes for stripe rust, chromosomal locations, types of resistance and references (Rahmatov, 2013; Chen, 2005).*

Yr gene	Chromosomal location	Resistance type[a]	Reference
Yr1	2A	RS, AS	Lupton and Macer 1962
Yr2	7B	RS, AS	Lupton and Macer 1962
Yr3	Unknown		Stubbs 1985
Yr3a	1B, 2B	RS, AS	Lupton and Macer 1962
Yr3b	Unknown	RS, AS	Lupton and Macer 1962
Yr3c	1B	RS, AS	Lupton and Macer 1962
Yr4	3BS		Baylesand Thomas, 1984

Links Researchers

Yr4a	6B	RS, AS	Lupton and Macer 1962
Yr4b	6B	RS, AS	Lupton and Macer 1962
Yr5	2BL	RS, AS	Macer 1966
Yr6	7BS, 7B	RS, AS	Macer 1966
Yr7	2B, 2BL	RS, AS	Macer 1966
Yr8	2D	RS, AS	Riley et al. 1968
Yr9	1RS/1BL	RS, AS	Macer 1975
Yr10	1B, 1BS	RS, AS	Macer 1975
Yr11	Unknown	RS, AP	McIntosh 1988
Yr12	Unknown	RS, AP	McIntosh 1988
Yr13	Unknown	RS, AP	McIntosh 1988
Yr14	Unknown	RS, AP	McIntosh 1988
Yr15	1BS	RS, AS	Gerechter-Amitai et al. 1989
Yr16	2D	NRS, AP	Worland and Law 1986
Yr17	2AS-6M	RS, AS	Bariana and McIntosh 1993
Yr18	7D, 7DS	NRS, HTAP	Singh 1992
Yr19	5B	RS, AS	Chen et al. 1995b
Yr20	6D	RS, AS	Chen et al. 1995b
Yr21	1B	RS, AS	Chen et al. 1995b
Yr22	4D	RS, AS	Chen et al. 1995b
Yr23	6D	RS, AS	Chen et al. 1995b
Yr24	1BS	RS, AS	McIntosh et al. 1998
Yr25	1D	RS, AS	McIntosh et al. 1998
Yr26	1BS, 1BL	RS, AS	McIntosh et al. 1998
Yr27	2BS	RS, AS	McDonald et al. 2004
Yr28	4DS	RS, AS	Singh et al. 2000
Yr29	1BL	NRS, AP	McIntosh et al. 2001
Yr30	3BS	NRS, AP	McIntosh et al. 2001
Yr31	2BS	RS, AS	McIntosh et al. 2003
Yr32	2A	RS, AS	Eriksen et al. 2004
Yr33	7DL	RS, AS	McIntosh et al. 2004
Yr34	5AL	AP	McIntosh et al. 2004
Yr35	6BS	RS, AS	McIntosh 2004, personal communication
Yr36	6BS	NRS, HTAP	J. Dubcovsky 2004, personal communication
Yr37	2DL	RS, AS	McIntosh 2004, personal communication
Yr38	6A	-	Rahmatov, 2013
Yr39	7BL	-	Rahmatov, 2013
Yr40	5DS	-	Rahmatov, 2013
YrH52	1BS	RS, AS	Peng et al. 2000
Yrns-B1	3BS	NRS, AP	Börneret al. 2000
YrSP	2BS	RS, AS	McIntosh et al. 1995
YrA	Unknown	RS, AS	McIntosh et al. 1998
YrCle	4B	RS, AS	Chen et al. 1998a
YrDru	5B, 6B	RS, AS	Chen et al. 1998a
YrDru2	6A	RS, AS	Chen et al. 1998a
YrDa1	1A	RS, AS	Chen et al. 1998a
YrDa2	5D	RS, AS	Chen et al. 1998a
YrH46	6A	RS, AS	Chen et al. 1998a
YrHVII	4A	RS, AS	Chen et al. 1998a
YrMin	4A	RS, AS	Chen et al. 1998a
YrMor	4B	RS, AS	Chen et al. 1998a
YrND	4A	RS, AS	Chen et al. 1998a
YrSte	2B	RS, AS	Chen et al. 1998a
YrSte2	3B	RS, AS	Chen et al. 1998a
YrTye	6D	RS, AS	Chen et al. 1998a
YrTr1	6D	RS, AS	Chen et al. 1998a
yrTr2	3A	RS, AS	Chen et al. 1998a
YrYam	4B	RS, AS	Chen et al. 1998a
YrV23	2B	RS, AS	Chen et al. 1998a
YrJh1…	2A	RS, AS	Zhang et al. 2001
YrJh2	4D	RS, AS	Zhang et al. 2001
YrGui1	Unknown	RS, AS	Cao et al. 2004
YrGui2	Unknown	RS, AS	Cao et al. 2004
YrGui3	Unknown	RS, AS	Cao et al. 2004
YrJu1	Unknown	RS, AS	Zhao et al. 2004
YrJu2	Unknown	RS, AS	Zhao et al. 2004
YrJu3	Unknown	RS, AS	Zhao et al. 2004
YrJu4	Unknown	RS, AS	Zhao et al. 2004
YrA1	Unknown	NRS, HTAP	Chen et al. 1998a
YrA2	Unknown	NRS, HTAP	Chen et al. 1998a
YrA3	Unknown	NRS, HTAP	Chen et al. 1998a
YrA4	Unknown	NRS, HTAP	Chen et al. 1998a
YrA5	Unknown	NRS, HTAP	Chen et al. 1998a
YrA6	Unknown	NRS, HTAP	Chen et al. 1998a
YrA7	6BS	NRS, HTAP	Chen et al. 1998a
YrA8	Unknown	NRS, HTAP	Chen et al. 1998a
YrD	6A	-	Rahmatov, 2013: Review
YrS	3BS	-	Rahmatov, 2013: Review
Tres	3A	-	Rahmatov, 2013: Review
YrCK	2DS	-	Rahmatov, 2013: Review

[a]: *AS, All-stage resistance/seedling resistance;* **AP:** *adult-plant resistance;* **HTAP:** *high-temperature, adult-plant resistance;* **RS:** *race-specific resistance;* **NRS:** *non-race-specific resistance.*

Identification and mapping of Yr resistance genes using molecular markers

Detection of Stripe rust resistance genes in host plants can be mapped thorough *Yr* molecular markers. Marker-assisted selection of particular genotypes for *Yr* gene is particularly one of the most important and much needed research areas on rusts, especially stripe rust. As markers are characterized by their closeness to the resistant genes they help in marker-assisted selection of genotypes. Molecular markers for *Yr* gene allow us to screen the wheat germplasm for the presence /absence of *Yr* genes. The genetic diversity of *Yr*

genes in different wheat lines could then be utilized in gene pyramiding in attempt to improve the stripe rust resistance (Begum et al., 2014). Gene pyramiding is basically grouping of multiple genes which will ultimately give higher level of expression of almost all the genes or will give combination effect in a variety to give resistance in crop plant. This technique is becoming popular and utmost for developing and improving the output of breeding to get broad spectrum resistance capabilities.

Current status of stripe rust

It is obvious that *Pst* remained a noteworthy threat in most of the global wheat growing areas with possibility to impose consistent regional crop damages. These losses are in the range of 0.1 to 5%, occasional losses of 5–25%. The current susceptibility regions include USA (especially Pacific North West), East Asia (China- northwest and southwest), South Asia (India, Pakistan and Nepal), Oceania (Australia, New Zealand), East Africa (Ethiopia, Kenya), the Arabian Peninsula (Yemen) and Western Europe (east England). In old Pakistani wheat varieties i.e. Lyalpur-73 Barani-83, Inqilab-91, stripe rust resistance gene (*Yr18*) existed (Rehman et al., 2013). Recently, new scientific technologies have been implemented for monitoring of disease through aerial and space remote sensing (Wang et al., 2016).

Conclusions

Yellow rust is a threat to wheat cultivation in Pakistan. Due to the specific landscape, data on the dispersal of rust is lacking. Well-equipped greenhouses must be constructed to test the disease at seedling stage to control its spread outside in the field. Durable resistance genes must be identified in the land races and shall be incorporated in modern cultivars. Regular monitoring, complete epidemiological experiments and wide-ranging patho-type analysis of rust samples in close collaboration with neighboring countries to tolerate an effective management strategy is needed.

Table 5: *List of stripe rust resistance SSR markers.*

Sr no.	Markers	Sr no.	Markers	Sr no.	Markers	Sr no.	Markers
1	gwm136	26	wmc336	51	barc204	76	gwm11
2	barc302	27	gwm604	52	barc181	77	barc302
3	cfd92	28	cfd83	53	wmc429	78	gwm458
4	gdm111	29	barc220	54	barc124	79	gwm124
5	cfd233	30	barc201	55	barc114	80	barc45
6	gwm2	31	barc228	56	gwm539	81	barc228
7	wmc741	32	wmc41	57	gwm484	82	barc159
8	wmc650	33	wmc664	58	barc314	83	gwm533
9	wmc349	34	barc7	59	barc84	84	gwm181
10	barc301	35	gwm645	60	cfd9	85	gwm645
11	gwm415	36	barc170	61	wmc468	86	wmc262
12	gwm371	37	wmc238	62	gwm495	87	wmc710
13	gwm499	38	barc303	63	wmc705	88	barc303
14	gwm159	39	gwm304	64	barc319	89	barc151
15	gwm583	40	gwm604	65	wmc773	90	gwm268
16	barc178	41	gwm371	66	gwm604	91	xgwm181
17	wmc593	42	gwm205	67	barc286	92	wmc590
18	gwm276	43	barc320	68	barc118	93	wmc177
19	gwm295	44	cfd49	69	barc301	94	wmc124
20	barc126	45	barc154	70	psp3071	95	cfd56
21	gwm333	46	gwm332	71	cfd42	96	wmc539
22	gwm121	47	wmc488	72	gwm437	97	gwm148
23	psp3113	48	wmc396	73	gwm121	98	barc290
24	gwm400	49	gwm319	74	gwm292	99	xgwm3
25	gwm46	50	cfd49	75	gwm544	100	xfbb194

Basic information of the disease, its races (host specific and non-host specific), way of spreading and how to develop specific type of resistance (Table 3).

Recommendations

To contain the worst effects of *Pst*, young plant breeders and pathologists must be trained and equipped. Adequate measures should be taken for gene deployment across the region, use of molecular markers to follow the flow and the build-up of resistance in the wheat germplasm, monitor the genetic diversity in the rust populations across the region, and maintenance of an adequate level of host diversity in the breeding programs to stabilize resistance to the predominant cereal rust.

Author's Contribution

AW, TR and Rabia presented the idea and compilation and screened the research paper. SHK and SB generated main idea, helped in manuscript writing compilation of qualitative and quantitative resistance. AS helped in conventional breeding. WA helped in molding the article and writing the abstract. SSZ updated marker list used for SSR analysis and marker presence on each chromosome. IS helped in material and methods. GMA identified and modelled Yr resistance genes using molecular markers.

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