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Molecular Basis and Genetics of Stem Rust Resistance in Wheat

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Wheat is the staple food for many countries, therefore, it needs more attention as compared to other cultivated crops. Stem rust is one of the major wheat diseases. Fungus spores fall on the plant surface and starts to grow inside the plant tissues. Two groups of resistant genes(R & Avr) have been identified conferring host-specific and non-host specific resistance respectively. Resistance is either achieved through thickening of the cell wall or through the programmed cell death (hypersensitivity) type of response. Every pathogen has specific pathogen-associated molecular patterns PAMPs which are recognized by the receptor protein. i.e. pattern recognition receptors PRRs. Plants can activate separate defence pathways depending on the type of the pathogen encountered. Jasmonic acid (JA) and ethylene-dependent responses seem to be initiated by necrotrophs, whereas salicylic acid (SA) dependent response activated by biotrophic pathogens.

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1. INTRODUCTION

Wheat, the third most important cereal in the world behind two major crops; rice and maize. It fulfils 20% of proteins and calories consumed per capita [1,2]. Rice is mostly used by many countries of the world. In Asia, rice was the major part of the diet for most of the countries but after 2000 this trend started changing and rice is being replaced with wheat [3]. From the beginning, the staple food for the Pakistani people is wheat, while most of the other countries of Asia were dependent upon rice but they are now moving towards wheat. Being the staple food of the world's major population, it is necessary to maintain sustainable yield of wheat. So the protection of wheat from different biotic and abiotic stresses is inevitable. Black rust or Stem rust is one of the major wheat diseases and main type of rust that cause up to 100% yield losses followed by leaf rust which can cause up to 40% losses [4,5]. Stem rust is caused by fungus *Puccinia graminnis* tritici. Other hosts of this fungus include oat, barley, rye, triticale, and almost all grasses. Varieties in which resistance has been induced by the transfer of one gene lose their resistance after few years of their release because of evolving pathotypes against the gene that can overcome the resistant conferred by that single gene. "International Spring Wheat Nursery Program " was initiated by Bayles and Rodenhiser in 1950. Purpose of this program was to screen new genes of rust resistance at the global level [1,6,7].

Since 1960's Green Revolution, the evolution in the pathogen has nearly stopped because of reduced disease pressure and reduced pressure of pathogen races lacking capability against the germplasm of resistant gene at CIMMYT [1,8]. Ug99 is the most virulent strain of stem rust capable of overcoming a number of resistant genes including Sr 31 which is known for its durability. To date, eight races belonging to the Ug99 ancestrol group are known. Increased pathogen monitoring activities have led to the identification of other races in Africa and Asia with additional virulence to commercially important resistance genes [9,10,11]. Stem rust caused 80% loss in yield in Kenya. Yield losses due to stem rust were maximum in 2011 [12]. In Pakistan, Plain areas and foothills of Northern Punjab and KPK in 1994-1995 and 1995-1996 stem rust caused a loss of two billion rupees [9]. Three durum wheat and two bread wheat varieties have been released in Ethiopia. Focused Identification of Germplasm strategy (FIGS) approach can be used when there is a selection of landraces and crop's wild relatives is to be made for experiments being conducted with the aim to identify diversity in target traits (Endresen et al. 2012). There is an urgent need to find additional genes that confer resistance to the new races of the Ug99 race group and identify reliable markers that assist breeding programs in introgressing these genes in germplasm [13,14].

2. MECHANISM OF DISEASE SPREAD

Uredospore's produced by this fungus by growing on wheat plant affect the wheat plant. Mycelium of the fungus hibernate in the soil and restore activity in the next season. Studying the genome of the wheat plant using the molecular markers has helped us in the identification of various stem rust genes i.e. Sr2, Sr22, Sr24, Sr46, etc. [15,16]. To date, more than 187 rust-resistant genes (80 leaf rust, 58 stem rust, and 49 strip rust) have been derived from diverse wheat or durum wheat cultivars and the related wild species using different molecular methods. The fungus enters through the stroma of the chloroplast and then establishes its growth by growing its haustoria [17]. The biochemical study revealed that in resistant cultivars the growth of the fungal hyphae was restarted by the activity of catalase, peroxidase and more concentration of chlorophyll while susceptible cultivars exhibit more reactive oxygen species [18,19].

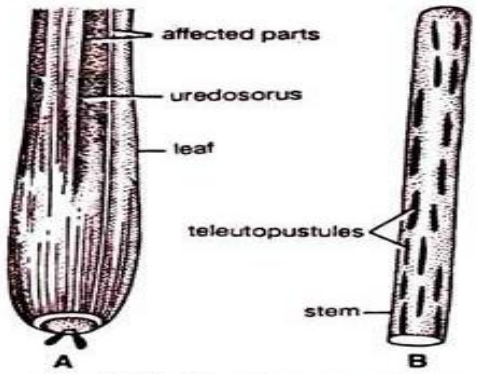

Fig. 1 (A, B). *Puccinia gaminis* tritici: Symptoms on wheat plant. (A) Uredosorus on leaf; (B) Teleutopustules on stem

Pujol et al. 2016 [15]

3. RESISTANCE MECHANISM

Resistance is induced by leading to death of the surrounding guard cells and epidermal cells i.e. hypersensitivity type of response. Wheat is the primary host of this fungus thus, mostly affected by it. Aeciospores which are produced on secondary host i.e. barberry are also a source of infection on wheat. Hyphae of the fungus penetrates through the stomata and obtain its food from surrounding cells. Thickening of the plant cell wall is also one of the mechanisms to restrict fungal entry inside the host. The resistant mechanism in a number of plants is associated with the local induction to produce polymeric compounds such as callose, lignin [20,21].

4. HOST-PATHOGEN INTERACTION

Stem rust (Black rust) of fungus is more devastating than all other rust types. Under favourable conditions, stem rust may cause up to 100% yield loss [22,23]. Recently discovered biotype of fungus in Uganda (1999) is Ug99 which can cause serious damages in future to crops in other countries. (Asmmawy et al. 2013). They also purposed that out of 56 designated stem rust resistance genes only 8 genes *(Sr13, Sr14, Sr22, Sr28, Sr33, Sr35*, *Sr42, and Sr45*) have potential to confer resistance against this biotype of fungus. This fungus can be controlled with the help of fungicides, crop rotations and through resistant cultivars. The use of fungicides have a negative economic role, so the breeding of resistant cultivars is necessary [24]. There are two types of host-pathogen interactions. One is a compatible type in which the host is lacking resistant gene and another is incompatible in which host has resistant genes against fungus [25]. Plants generally show two types of responses. One is constitutive type and another is inducible type. Constitutive type includes the phytotoxins which have some antimicrobial activities and other is inducible type response which is activated by elicitor molecules e.g. Glycoproteins released by the fungus [26].

5. GENETICS OF STEM RUST RESISTANCE

Until now a number of stem resistance genes have been discovered in wheat by the different scientist. Mainly there are two families of genes that resist fungal attack. One family includes R genes and other includes Adult Plant Resistant Genes (APR). R gene show race-specific resistance while the resistance induced by APR genes may be against many sub-species of fungus [27]. R genes also show gene for gene hypothesis of flour (1971) i.e. R genes in host show resistance by recognizing the elicitors molecules released by the corresponding Avr gene of the pathogen. In other words, the efficiency of the R gene is host Avr gene-dependent i.e. host specific. While the resistance of the APR is partial type i.e. slow rusting. Recent molecular techniques have revealed that these resistance genes encode for the plant immune system components that recognize and activate resistant mechanism against a specific pathogen [28], Akter et al. 2017, [29]. The resistance shown by the R gene is overcome by the new mutant type of that corresponding Avr gene, which is not recognized by the R gene products. One solution to make this R gene resistance durable is their pyramiding into single genotype. It will take a long time for pathogen to overcome this resistance but the efficiency of resistance gene pyramiding depends upon the association of molecular markers associated with them. The more tight linkage between resistance genes and molecular markers will be made easy by their pyramiding. The new biotype of *Puccinia graminis* mainly arises by the sexual recombination through the production of sexual spores on alternate host i.e. barberry. Success in America and Australia has been achieved through absence or near reduction of this alternation host [30,22,9].

The RPG gene family provides resistance against *Puccinia graminis* f. sp. tririci. Barley is the main source of this family. Rpg1, Rpg2, Rpg3 Rpg4, Rpg5, Rpg BH and Rpg6 are main members of this family, which provide resistance against *Puccinia graminis*.

5.1 Molecular Markers of Sr Genes

Resistant cultivars provide one of the best means for controlling stem rust. To date, nearly 60 stem rust (Sr) loci and many quantitative trait loci (QTLs) have been identified in wheat and its wild relatives against stem rust. Many studies have been carried out in order to identify and map genes for stem rust resistance in wheat. Tetraploid wheat (T. turgidum ssp.) in particular have contributed a number of important stem rust resistance genes such as Sr2, Sr9d, Sr9e, Sr9g, Sr11, Sr12, Sr13, Sr14, and Sr17. Once only the Sr2 gene was known to confer slow rusting APR; now, four more genes—Sr55, Sr56, Sr57, and Sr58—have been characterized and additional quantitative trait loci identified. Singh et al. 2015, [31,32].

The use of biparental mapping populations is a standardized approach to identify the chromosomal locations of plant disease resistance loci. Bulked Segregant Analysis (BSA) is a quick and relatively cheaper method to efficiently identify molecular markers associated with a trait response. The procedure consists of comparing two pooled DNA samples of individuals from a segregating population arising from a single cross. Within each bulk, the

Table 1. Pakistani stem rust resistant cultivars [1,36]

Resistant Cultivars	Anmol-91,Bahawalpur-97,Darwar-97,Faisalabad-2008,Fareed-2006,Kohistan-97,Kohinoor-83, Lasani-08, Manthar-3, Mehran-89, Moomal-2002, Parwaz-94, Pirsabak 2004, Punjnad-1, Saleem-2000, Saussi, Sehar-2006, Shaheen-94, Shahkar-95, Soorab-96, Takbeer,V-87094, Wafaq-01, Watan-94, Zarghoon, Zarlashata
Resistant	AS-2002,Auqab-2000,Bahawalpur-2000,Bahkhar-2002,Chakwal-86,Chakwal-97, Faisalabad-83,Khyber-87, Kirin-95, LU-26,Margalla-99, Mexipak-65, MH-97, Nowshehra- 96,Pasban-90,Pirsabak-2005,Punjab-96,Raskooh,Rohtas-90,Sariab-92,Sarsabz,SH-2002, Shafaq-2006, Sind-81, Soughat-90, Suleman-96, Tadojam-83, WL-711, Zardana

Table 2. Forward and reverse primers for stem rust resistance gene markers

Author's	Markers	Primer sequences
Xiaofeng et al. [37]	Xgwm5333(Sr2)	5'-GTTGCTTTAGGGGAAAAGCC 5'-AAGGCGAATCAAACGGAATA
	Sr24#12 (Sr24)	5'-CACCCGTGACATGCTCGTA 5'-AACAGGAAATGAGCAACGATGT
	Gb	5'-CATCCTTGGGGACCTC 5'-CCAGCTCGCATACATCCA
	Sr26#43	5'-AATCGTCCACATTGGCTTCT 5′-CGCAACAAAATCATGCACTA
	SCSS30.2$_{576}$	5′-GTCCGACAATACGAACGATT 5′-CCGACAATACGAACGCCTTG
	Iag 95	5′-CTCTGTGGATAGTTACTTGATCGA 5′-CCTAGAACATGCATGGCTGTTACA
	VENTRIUP	5′-AGGGGCTACTGACCAAGGCT 5′-TGCAGCTACAGCAGTATGTACACAAAA
Nzuve et al. [38]	Xcfa2019(Sr22)	5'-GACGAGCTAACTGCAGACCC 5'-CTCAATCCTGATGCGGAGATCGGTCTTTGTTTGCTCTAAAC CACCGGCCATCTATGATGAAG
	Sr24#50(Sr24)	5'-CCCAGCATCGGTGAAAGAA 5'- ATGCGGAGCCTTCACATTTT
	Sr26#43(Sr26)	5'-AATCGTCCACAT TGGCTTCT 5'-CGCAACAAAATCATGCACTA
	BE518379(Sr26)	5'-AGCCGCGAAATCTACTTTGA 5'-TTAAACGGACAGAGCACACG
	SCSS30.2(Sr31)	5'- GTCCGACAATACGAACGATT 5'-CCGACAATACGAACGCCTTG
	Sr39-I(Sr39)	5'-AGAGAGAGTAGAAGAGCTGC 5'-AGAGAGAGCATCCACGA
	Sr39-II(Sr39)	5'- GAGAGAGAGTAGAAGAGC 5'- AGAGAGAGAGCATCCACC
Bansal et al. [39]	sun218(Sr56)	5'-AAACCCAACATTTCAGTTTGCC 5'-ATCATCCCAACATGCCATCC
	sun221(Sr56)	5'-TTCCTTAAGACATGACAACC 5'-AATGGACTTCACTACTACGT
Mago et al. [40]	Barc71	5'-GCGCTTGTTCCTCACCTGCTCATA 5'-GCGTATATTCTCTCGTCTTCTTGTTGGTT

individuals are identical for the trait or gene of interest but are segregating randomly for all other genes. The two bulks that are contrasting for a trait such as response to disease are analyzed to find molecular markers that differentiate them. Therefore, the markers that are polymorphic between the pools will be linked genetically to the locus that is associated with the trait used to make the bulk [33,34,35].

6. MOLECULAR BASIS OF STEM RUST RESISTANCE

Puccinia graminis is obligate biotroph i.e. it cannot live without living cells. Although the fungus can be cultured with difficulty on artificial media, cultures grow slowly and upon subculturing they develop abnormal ploidy levels and their ability to infect host plants get varies [22]. It belongs to group phylum Basidiomycota of kingdom fungi. This fungus show alternation of generation between two hosts i.e. wheat and barberry. It is unique from other fungi of this group that it completes its life cycle in five spore stages i.e. uredospore, teliospores, basidiospores, and aeciospores. Uredospores cause infection on the wheat plant. The first microscopic symptom is usually a small chlorotic fleck, which appears a few days after infection. About 8-10 days after infection, a pustule several millimetres long and a few millimetres wide is formed by rupture of the host epidermis from the pressure of a mass brick red uredospore's produced in the infection [22]. The fungus gain entry through stomata of the leaves. Appressorium structure develops on stomata from which small pigs enter into the plant. After gaining entry, fungus establishes its growth [41]. Every pathogen has specific pathogen-associated molecular patterns PAMPs which are recognized by the receptor protein. i.e. pattern recognition receptors PRRs. Stimulation of PRRs leads to PAMP-triggered immunity [28]. Fungal plant pathogens, like rust-causing biotrophic fungi, secrete hundreds of effectors into plant cells to subvert host immunity and promote pathogenicity on their host plants by manipulating specific physiological processes or signal pathways, but the actual function has been demonstrated for very few of these proteins [42]. There are usually two types of plant mechanism response shown by the plants. Either the growth of the fungus is immediately stopped i.e. immune type of response or their colonies continue to grow i.e. intermediate type resistance. Haustoria of the fungus secrete some effectors which are recognized by the plant to activate the awake up resistance mechanism. Plants can activate separate defence pathways depending on the type of the pathogen encountered. Jasmonic acid (JA) and ethylene-dependent responses seem to be initiated by necrotrophs, whereas salicylic acid (SA) dependent response activated by biotrophic pathogens. The mechanism responsible for this differential recognition and response may involve crosstalk among these three different signals transduction pathways. i.e. Jasmonic acid, ethylene and salicylic acid [24,43]. Elicitors are the molecules which are secreted by the pathogens which induce defense mechanism in plants. Following elicitors perception, the activation of signal transduction pathways generally lead to the production of active oxygen species (AOS), phytoalexin biosynthesis, reinforcement of the plant cell wall associated with phenyl propanoic compounds, deposition of callose, synthesis of defense enzymes and the accumulation of pathogenesis-related (PR) protein, some of which possess antimicrobial properties [25] (Letta, 2018). The first mechanism that has been proposed is hypersensitivity response i.e. death of cells surrounding the fungal growth.

6.1. Hypersensitivity Response

HR constitutes one of the main mechanisms shown by the plants. The cells surrounding the fungal growth die in a coordinated way, so that the fungal growth may be stopped. HR type response is useful for obligate biotroph fungus. i.e. like *Puccinia graminis* with the nutrient supply of the cells cut off thus leading to the inhibition of the fungal growth. But this mechanism has not been used against necrotrophic fungus because they feed on dead tissues. Incompatible pathogens, whether fungi, viruses or bacteria, frequently provoke the accumulation of both free BA and SA and their respective glucoside conjugates, with the highest concentrations forming in the immediate vicinity of the infection site. The induction of these compounds is commonly associated with HR [44]. Salicylic acid also shows some antimicrobial activity. SA is derived from phenylpropanoid pathways but it can also be synthesized by the activity of enzyme BA-2H which converts benzoic acid into Salicylic acid. During HR type response the activity of this enzyme is also enhanced Leon et al. 1995. Thus in this incompatible R-Avr gene system salicylic acid may arrest the growth of the fungus (Singh and Ram., 1995). Besides SA, ethylene also plays an important role in programmed cell death. The experimental proof

was obtained by work on oat, where the application of the ethylene inhibitors. i.e. amino-oxyacetic acid (AOA) and silver thiosulphate (STS) on plant mesophyll cells leads to reduced cell death induced by victirin Singh and Ram., 2013, [15].

6.1.1 Salicylic acid and Jasmonic acid signalling

Salicylic acid is a key molecule for plants to show resistance to plant pathogens as its role in plant response has been known for the last 35 years. Salicylic acid binding proteins are still remained to be investigated. Any breakthrough regarding this may prove vital for understanding this signalling pathway. Recently identification of salicylic acid binding elements such as NPR3, NPR4 & NPR1 has created more interest in this field [45]. The interactions between SA and JA signalling appear to be complex, and there is evidence for both positive and negative interactions between these pathways. However, the primary mode of interaction between these pathways appears to be mutual antagonism. This has been proposed to be central to the plant's ability to fine-tune the induction of plant defences in response to different plant pests and pathogens [46,47].

6.2 Thickening of Cell Wall

The second type of response which is linked with HR in the plants in thickening of their cell wall to prevent the entry of fungus through the epidermal cell. Experiment evidence for this phenomena has been provided by (Sherwood and Vance. 1979) in which they noted that there was less number of infections when there were the incompatible host and pathogen infection. But when the plants were treated with cycloheximide solution (12ug/ml) which inhibit leaf responses, no lignification of the cell wall was observed so the number of infections was increased. Other experimental evidence was provided by Moerschbacher et al. [48] in near-isogenic lines of wheat where activities of the enzymes associated lignin biosynthesis were significantly increased in incompatible host-pathogen interaction. The work of these same scientists in (1990) provided the more experimental base to this type of resistance. They used three inhibitors of phenylalanine ammonia-lyase enzyme which is the first enzyme of lignin biosynthetic pathways i.e. namely a-aminooxy acetate, a- aminooxy-B-phenyl propionic acid a (1-amino-enzyme cinnamyl-alcohol dehydrogenase, namely N(O-amino phenyl) sulfinamoyl-tertiobutyl acetate and N(O-hydroxyphenyl) sulfinamoy-ltertiobutyl acetate. Treatment with these inhibitors significantly decreased the lignification process so the penetration rate of the fungus was increased [48,49].

6.2.1 Lignin deposition

Lignin is a polyphenolic polymer that strengthens and waterproofs the cell wall of specialized plant cell types. Lignification is part of the normal differentiation program and functioning of specific cell types, but can also be triggered as a response to various biotic and abiotic stresses in cells that would not otherwise be lignifying. It thickens the cell wall and retards further cell growth [50,33].

7. CONCLUSION

Stem rust is one of the major devastating diseases of the wheat. To feed double population till 2050 we have to cope with these biotic stresses. Fungicides are not a permanent solution as they have adverse impacts on our health. Resistant cultivars are the ultimate solution to handle all these problems. But to breed new cultivars it is important to identify new sources of resistance. Molecular markers have enabled us to identify and map those loci which are involved in the resistance mechanism. In this short review, we tried to briefly explain the genetics and molecular basis of this disease. Understanding the genetic and molecular interaction is prerequisite in developing resistant cultivars. Further tight linkage of molecular markers with resistant genes makes gene pyramiding more feasible. Molecular markers and biochemical analysis are becoming economically available these days so their use in research will enable us to identify new locus and molecules involved in stem rust gene resistance and to transfer those novel genes in our present successful cultivars more efficiently.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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