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Wheat rust research—then and now

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ABSTRACT

Wheat is an important constituent of human diet worldwide. India is the second largest producer of wheat in the world and wheat is directly related to the economic health of country. To meet the food requirements of the growing population, there is a need to increase wheat production. Wheat rusts caused by a fungus *Puccinia* species are the main biotic constraints in our efforts to sustain and boost production. Wheat rusts are historic and devastating pathogens worldwide. Their ability to spread aerially over the continents, production of infectious pustules geometrically in trillions and evolving new physiologic forms, makes the management of wheat rusts a very challenging task. To counter the threat of wheat rusts, efforts are going on worldwide. Identification of pathotypes, anticipatory breeding, evaluation for rust resistance and deployment of rust resistant cultivars is a time tested strategy to manage wheat rusts. There had been continuous efforts to increase the diversity for rust resistance. A list of more than 210 rust resistance genes and associated markers for many are available for the use of breeders. However, many of them have lost the effectiveness over the years. Introgression of rust resistance from rye and later on from other sources opened new vistas in research. However, the rust pathogens out smarted and new virulent pathotypes emerged which could overcome the novel rust resistance genes. Emergence of Ug 99 type of virulences threatened the cultivation of wheat in 40% of the world's acreage. DNA fingerprinting, sequencing of wheat and rust genomes were the milestone pieces of research in the 21st century. Efforts are still needed in studying the perpetuation of wheat rusts, epidemiology and inventing next generation techniques to break the yield barriers and manage wheat rusts. Role of *Berberis chinensis*, *B. holstii*, *B. koreana* and *B. vulgaris* as alternate hosts to *P. striiformis* (yellow rust of wheat) was an important discovery in this respect. Consolidated information on wheat rust research conducted over the years has been reviewed in this publication.

Key words: Axenic culture, Black rust, Brown rust, Chromosomes, Epidemiology, Genome, Interaction, Management of wheat rusts, Resistance genes, Yellow rust

Indian agriculture accounted for 14.1% of the GDP (GOI 2013) and has always played a pivotal role in the country's economy in imparting employment to 58.2% population. India is the second largest producer of wheat, next to China only. During 2013-14, total wheat production reached all time high 95.91 million tonnes **(**Anonymous 2014). To increase and sustain wheat yields, biotic constraints mainly rusts pose a constant challenge and have always been a priority for researchers as well as planners.

Wheat rusts are devastating diseases throughout the world. Yellow (stripe) rust is more devastating in West Asia, southern Africa, the Far East (China), South America and northern Europe. Brown (leaf) rust leads to more serious losses in South Asia, North Africa, south east Asia and South America. Black (stem) rust has traditionally been important in North America, Australasia, northern Africa, south Africa and, to some extent, Europe. Wheat rust diseases will continue to demand the attention of researchers and advisory personnel because of the dynamic nature of this relationship (McIntosh *et al.* 1995). In India, all the three rusts of wheat are important. In northern India, brown and yellow rusts are capable of causing loss to wheat production. These pathogens change forms very fast and evolve into more virulent forms. A resistant cultivar is rendered susceptible by a new pathotype, which makes this pursuit very challenging. The development of new wheat varieties is a continuous process as the pathogen keeps evolving correspondingly.

History of wheat rust research in India

The systematic investigation on cereal rusts in India was initiated by Rai Bahadur professor Karam Chand Mehta of Agra College around 1922. In 1930, with the assistance of Imperial Council of Agricultural Research (ICAR), he strengthened rust research program and other than Agra, three locations (Shimla, Almora and Murree, i.e. now in Pakistan) were selected for research work. Ultimately Shimla

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(Flowerdale) was found most suited site to maintain the cultures around the year (Nayar *et al.* 1994). His outstanding contribution in the discovery of the lifecycle of black rust of wheat in India and reported that barberry, an alternate host of wheat black rust pathogen in other countries, does not play any functional role in perpetuation of the rust fungus in India (Mehta 1940). The Flowerdale station founded by Prof. Mehta has now a repository of more than 127 rust pathogens, is responsible for identifying rust resistance in wheat material of India, identification of wheat rust pathotypes in India as well as neighbouring countries. In addition, the center is also responsible for supplying nucleus rust inocula elsewhere in India, pyramid resistance genes, conduct need based molecular studies and design wheat rust management strategies.

Losses due to wheat rusts

Both black and yellow rusts of wheat can cause 100% losses, whereas brown rust can incur 50% yield loss (Anonymous1982). Roelfs (1978) observed that the losses due to black and brown rusts in USA from 1918 to 1976 must have been to the extent of 50% or more during the epidemic years. The corresponding losses due to yellow rust which was restricted in distribution were observed to be around 70%.

Historical account of wheat rust epidemics in India has been given by Nagarajan and Joshi (1975). Epidemics were reported in Jabalpur as early as 1786 and subsequently in 1805, 1827, 1828-29, 1831-32. According to our estimates, meager 5% losses due to rusts , could result a loss of ₹ 39 200 million whereas 25% losses due to yellow rust in northern could lead to a loss of ₹ 10 000 million during 2007 in India.

Taxonomy, nomenclature, chromosomes, genome

Christiaan Hendrik Persoon (Persoon 1797) named *Puccinia graminis* to the causal organism of wheat black rust. Wheat rust pathogens belong to genus *Puccinia*, family Pucciniaceae, order Pucciniales, class Pucciniomycetes, subphylum Pucciniomycotina and phylum Basidiomycota (Bauer *et al.* 2006).

The recent morphological and genetic studies of the pathogen showed that that *P. recondita* is not the incitant of wheat leaf rust rather *P. triticina* should be the preferred name as shown by Savile (1984) and Anikster *et al.* (1997). Schmidt (1827) named the yellow rust fungus as *Uredo glumarum*. Westendorp (1854) used *Puccinia striiformis* for yellow rust collected from rye (Stubbs 1985). Later on, Eriksson and Henning (1896) showed that yellow rust resulted from a separate pathogen, which they named *P. glumarum*. This name of yellow rust pathogen was in the vogue until Hylander *et al.* (1953) revived the name *P. striiformis* Westend.

There were conflicting reports on the number of chromosomes in wheat rusts. McGinnis (1953) examined germinating sporidia of *P. graminis* during nuclear division and found at metaphase a haploid number of six chromosomes or total number of chromosomes as 12. Developing a complete genetic map with dominant markers such as RAPDs and AFLPs is difficult because *P. graminis* is dikaryotic. The fungus *P. graminis* f. sp. *tritici* is known to have 16-18 chromosomes (Boehm *et al.* 1992). Genome size of *P. graminis tritici*, *P. triticina* and *P. striiformis*is about 67mb (Backlund and Szabo 1993), 89mb and 53-73 mb (Cantu *et al.*2011), respectively.

Wheat rusts in India and their recurrence

Black rust of wheat

The disease is generally prevalent in summer wheat crop areas of Tamil Nadu, Himachal Pradesh and Jammu and Kashmir in the north. In favorable years it may be of concern in about 7 million hectares of Central and Peninsular India. *Puccinia graminis* Pers. f. sp. *tritici* Eriks. & Henn. is a macrocyclic, heteroecious rust. There are five stages in the life cycle of wheat rusts: 0-Pycnial stage, I-Aecidial stage, II-Uredial stage; III-Telial stage, IV-Basidiosporal stage. Pycnial stage or spermogonial stage is called stage - 0 because before 1927 the role of pycnial stage in the life cycle of rusts was not understood. Craigie (1927a) in Canada determined its function in the life cycle and in the variation of rust fungi. The pycnial stage (stage-0) and aecidial stage (stage-I) occur on alternate hosts for completion of life cycle of the pathogen. Its pycnial and aecial stages are produced on alternate hosts like *Berberis, Mahonia* and *Mahoberberis*, whereas uredial and telial stages occur on graminaceous host. Telio (Teleuto) spores germinate after a long dormancy period when exposed to freezing temperature. On germination a four celled promycelium is produced and each cell gives rise to a sterigma (pl. sterigmata) which bears basidiospore which infect wheat to produce pycnia. Pycnia are flask shaped and consist of spermatia (pycniospores) and receptive hyphae. Mating of opposite types in receptive hyhae and pycniospores result in aecia and aeciospores. Aeciospores infect wheat.

Since, under Indian conditions alternate hosts are not functional (Mehta 1929, 1940), the survival and perpetuation of the pathogen, therefore, occurs in the form of uredospores in the hills on self sown plants, summer crop being grown there. The non synchronization of vulnerable tender barberry leaves when the basidiospores are available, drastically, curtails the role of alternate host under Indian conditions. In fact, alternate hosts are of no consequence in the recurrence of stem rust in India (Nagarajan and Joshi 1985).

Brown (leaf) rust of wheat

The disease occurs wheever wheat is grown. Life cycle of *P. triticina* Eriks., causing wheat brown rust is like that of black rust. The fungus is known to survive on alternate hosts like *Thalictrum* spp (Jackson and Mains 1921), *Isopyrum fumarioides* (Chester 1946), *Clematis* spp (Sibilia 1960**)** and *Anchusa* spp (de Oliveira and Samborski 1966). However, under Indian conditions, none of the alternate hosts is functional, therefore, fungus perpetuates in the form of uredial stage only. An aecial stage observed on *Thalictrum*

javanicum in India is connected with *Agropyron semicostatum* Nees and is of *Puccinia persistens* but does not infect wheat. Few species of *Aegilops* are also susceptible to brown rust of wheat.

Yellow (stripe) rust of wheat

P. striiformis Westend. f. sp.*tritici*, causing wheat yellow rust, occurs in cooler wheat growing tracts of North western India and hilly areas of Tamil Nadu. Later on the yellow stripes turn black when teleutospores are formed and many people confuse it with black rust of wheat. The infection occurs on leaf, leaf sheaths, stems, glumes, awns and also at times on kernels.

Heteroecious nature of the yellow rust pathogen was not known until 2010. Inoculation of grasses using aeciospores from naturally infected *Berberis chinensis* and *B. koreana* resulted in infection on *Poa pratensis*, producing uredinia typical of yellow rust caused by *P. striiformis*. Analyses using real-time polymerase chain reaction and DNA sequence confirmed the rust fungus as *P. striiformis*. Wheat inoculated with aeciospores from *B. chinensis* resulted in uredinia, which demonstrated that *Berberis* spp. also serve as alternate hosts for the wheat yellow rust pathogen (Jin *et al*. 2010). In the absence of a functional alternate host, in India the primary inoculum for the Indo-Gangetic plains has been said to be coming each year from the array of Himalayan hills, where it survives on volunteer plants or summer crop in the form of uredospores or some other grasses/plants in the catchment areas. There is further scope to investigate epidemiology of wheat rusts in India.

Axenic culture of rust pathogens

Wheat rust pathogens are obligate parasites which need the living tissues of wheat or other species for their survival. However, wheat stem rust pathogen (*Puccinia graminis* f. sp. *tritici* race 126-ANZ-6,7) was grown on artificial media successfully (Williams *et al.* 1967).

Physiological specialization and nomenclature of pathotypes

The term *formae specialis (*f. sp.*)* coined by Eriksson to designate these pathogenically specialized group within species of a rust fungus. He gave experimental evidence for this through cross inoculation experiments in *Puccinia graminis* and recognized five special forms in this rust, viz. *tritici* on wheat, *avenae* (oat), *secalis* (rye), *agrostidis* (*Agrostis* spp.) and *poae* (*Poa* spp.). The variations within the *formae specialis* reported by Elvin Charles Stakman (1914) on the basis of the pathogenicity of these variants (pathotypes) on a set of host cultivars /or differentials. A set of 12 differential cultivars for identification of pathotypes/ or races of black rust was proposed by Stakman and Levine (1922). The occurrence of races in brown rust (*Puccinia triticina*) was first proved by Mains and Jackson (1926), later on, Johnston and Mains (1932) selected eight differentials for the identification of races in brown rust pathogen. Similarly, in yellow rust (*Puccinia striiformis* f. sp. *tritici*), Gassner and Straib (1932) initiated to identify races on the basis of 12 differential cultivars.

With the gene for gene theory of Flor (1956), it was realized that pathotypes identification system requires modification in order to use the virulence survey results in more meaningful purpose. Consequently numerous procedures using near isogenic lines were suggested around the world (Watson and Luig 1963, Johnson *et al.* 1972, Roelfs and McVey 1974). Genetic studies in hexaploid wheat led to the identification of genes for resistance. These were designated with an *Lr* prefix (Ausemus *et al.* 1946). A summary of *Lr* genes (from *Lr*1 to *Lr*29) was provided by Browder (1980), and updated lists are provided by McIntosh

Table 1 The revised composition of sets of differentials for the identification of pathotypes

Set 0	Set A	Set B
Brown rust (*Puccinia triticina*)		
IWP 94 (*Lr*23+)	*Lr*14a	Loros (*Lr*2c)
Kharchia Mutant	*Lr*24	Webster (*Lr*2a)
Raj 3765	*Lr*18	Democrat (Lr3)
PBW 343	*Lr*13	Thew (*Lr*20)
UP 2338	*Lr*17	Malakoff (*Lr* 1)
K 8804	*Lr*15	Benno (*Lr*26)
Raj 1555	*Lr*10	HP1633 (*Lr* 9+)
HD 2189	*Lr*19	
Agra Local	*Lr*28	
Black rust (*Puccinia graminis tritici*)		
*Sr*24	*Sr*13	Marquis (*Sr*7b+)
NI 5439	*Sr*9b	Einkorn (*Sr*21+)
*Sr*25	*Sr*11	Kota (*Sr*28+)
DWR 195	*Sr*28	Reliance (*Sr*5+)
HD 2189	*Sr*8b	Charter (*Sr*11+)
Lok 1	*Sr*9e	Khapli (*Sr*7a+*)*
HI 1077	*Sr*30	Tc*6/*Lr26* (*Yr*9)
Barley Local	*Sr*37	
Agra Local		
Yellow rust (*Puccinia striiformis*)		
		Wheat *Barley*
WH147	TDT	Chinese 166 (*Yr*1) Hybrid 46 (*Yr*4)
Barley local	Barley local	Lee (*Yr*7) Heines VII (*Yr*2+)
WH416	WH147	Heines Kolben (*Yr*6) Compair (*Yr*8)
PDW215	Heils Franken	Vilmorin 23 (*Yr3*) *T.spelta album* (*Yr*5)
HD2329	Fong Tien	Moro (*Yr*10) Tc*6/*Lr26* (*Yr*9)
HD2667	Himani	Strubes Dickkopf Sonalika (*Yr*2+)
PBW343	BHS 16	Suwon92 X Omar Kalyansona *Yr*2(KS)
HS240	Alfa 93	Riebesel47/51
Anza	Dolma	(*Yr*9+)

et al. (2008)**.** Various research institutions developed their own systems of analysis and race designation, but some joined in using a common system, in order to facilitate communication. Based on the experience on wheat rusts in India and systems proposed in other countries, to begin with, a system for the analysis of brown rust pathotypes in India was proposed by Nagarajan *et al.* (1983) and latest modification is given by Bhardwaj *et al.* (2011) as given in Table 1.The system has 3 sets of differentials.

Pathometry

Wheat rust interactionis recorded as per Stakman *et al.* (1962) as 0 to 4 with some modifications. For field observations Peterson *et al.* (1948) provided a range of diagrams of visible lesions and the percentage area covered by the lesions to measure rust intensity. At CIMMYT a uniform scale of 5,10,20,40,60 and 100 is followed for all the rusts.

Loegering (1959) gave a detailed outline for recording black and brown rusts on the basis of severity and response. Severity is recorded as visual observations. Below 5% severity, intervals used are traces to 2%. Usually 5% interval is used between 5-20 and 10 between 20-100% severity. The response is referred as type of infection as; 0= No infection (0.0), R= Resistant infection type (0.2); MR= Moderately resistant (0.4); X= Mesothetic (0.6) MS = Moderately susceptible (0.8); S= Susceptible (1.0), where value in brackets are the response values. Reading of severity and response are recorded together with severity first, e.g. 40R. For statistical analyses the reactions are converted into a co-efficient of infection as use by Loegering (1959) which can be calculated by multiplying the % of infection by response value for the concerned infection type, e.g. 10R=10×.2=2; 10MR=10×.4=4; 10X=10×.6=6; 10MS=10×.8=8; 10S=10×1=10.

Wheat rusts as international pathogens

Rusts are able to spread over long distances. Yellow rust of wheat was not known in Australia before 1979 (Wellings and McIntosh 1981) when pathotype 104E137 identical to the one that was found only in Europe was noticed. Yellow rust was subsequently reported from New Zealand next year (Beresford 1982). The introduction of yellow rust into Australia appears to be human aided, while introduction into New Zealand appears to be by way of dispersal of uredospores by wind (Wellings 2011). Studies revealed the long distance spread of *P. graminis tritici* uredospores from Australia to New Zealand across 2 000 km distance of ocean (McEwan 1969). Identical biochemical patterns and connective winds from Australia indicate long distance dispersal and deposition of viable uredospores across 5 000 km ocean from southern parts of Africa to that of Australia.

In 1990 *Yr*9 virulence was identified in Syria and in April 1994 the virulence attacking Veery#S cultivars such as Pak81, Pirsabak85, Seri82 possessing *Yr*9 became susceptible in Pakistan (Nagarajan and Saari 1995). In 1996 a virulent pathotype on *Yr*9 and another virulent to *Yr*9 and *Yr*27 in 2002 were identified from the bordering areas of Punjab (Prashar *et al.* 2007) and subsequently have been identified from Nepal and Bhutan.

Ug99 and BGRI

Many sources of resistance including the alien sources have been used for black rust resistance. Introduction of rye (*Secale cereale* L.) gene (1B/1R translocation or substitution) into bread wheat (Mettin *et al*. 1973, Zeller 1973) which carries *Lr*26/ *Sr*31/ *Yr*9, completely linked resistance gene has not only contributed 12–20% yield jump but also imparted resistance to major biotic and abiotic stresses (Cox *et al*. 1995). During many years, *Sr*31 and other resistance genes kept the black rust fungus under control. For nearly 60 years wheat rusts were not in the news. However, the occurrence of black rust virulence on 1BL.1RS translocation popularly known as *Sr*31 raised the alarm bells and reminded us of Dr. Norman Borlaug words that 'Rust never sleeps'. It endangered nearly 40% of the wheat area covered with resistance based on *Sr*31 in different parts of the world. William W. Wagoire first observed *Sr*31 virulence in an experimental wheat field at the Buginyanya Zonal Agricultural Research and Development Centre in Uganda in 1998, latterly, it was confirmed as a new race of *P. graminis* f. sp. *tritici* by Zacharias A. Pretorius in 1999, popularly designated as Ug99 (Pretorius *et al.* 2000). Under Ug99 lineage, till date 11 variants have been documented (Patpour *et al.* 2016).

Since the appearance of Ug99, it was widely publicized that Ug99 is a serious threat to Indian wheat production. A detailed epidemiological study on the relevance of Ug99 to the NWPZ of India has shown that Ug99 is not a threat to wheat production in the NWPZ or to India's food security (Nagarajan 2012, Bhardwaj *et al*. 2014).

Occurrence of a virulence on *Sr*31 in Uganda in 1998, entire world came to one platform and Dr. Borlaug became the champion behind the development of the Borlaug Global Rust Initiative. The new race of *Puccinia graminis* f.sp. *tritici*, TTKSK (Ug99) has further spread and caused catastrophic losses among smallholder farmers in Uganda, Kenya and Ethiopia. It was found to be virulent on more than 90 percent of the world's wheat crop and threatened to invade the wheat fields of the world's bread baskets in the Middle East, South Asia, and South Africa. In response and with the support and advice of Dr. Borlaug and the Rockefeller Foundation, CIMMYT called for what became known as a "Global Rust Summit," which was held in Nairobi in May 2005. Since then, BGRI has made a significant impact on world wheat productivity. It has significantly advanced rust research, improved the world's ability to withstand rust epidemics, increased wheat productivity, and grown the global rust community in numbers and capacity.

Genetics of wheat rust resistance

Wheat and rusts have co-existed and co-evolved hand

in hand for centuries, consequently, there is a close relation between the two. Based on the knowledge of definite host rust pathogen interaction, resistance breeding has become very precise and focused (Bhardwaj 2013). Wheat genotypes differ in their degree of resistance or susceptibility to rust infection. Incompatibility (resistance) is a recognition process which results from a resistant host and avirulent pathogen. The infection type produced as a result of pathogen attacking a host is a product of two genetic systems. There occurs specificity in combinations involving the genes that are normally dominant, R and P. Any one of the three other combinations, RR/pp, rr/PP, rr/pp results in susceptibility or compatible reaction.

Biffen (1905) working with yellow rust, provided the first evidence that resistance to a pathogen could be governed by a single recessive gene inheriting in Mendelian fashion. He demonstrated that resistance to yellow rust in Rivet wheat was controlled by one recessive gene.

The gene-for-gene relationship was discovered by Harold Henry Flor who was working with rust (*Melampsora lini*) of flax (*Linum usitatissimum*). Flor (1942) was the first scientist to study the genetics of both the host and parasite and to integrate them into one genetic system. In his initial studies he dealt with a gene pair in the pathogen corresponding to a gene pair in host (Flor 1947). The hypothesis says that 'for every gene for resistance in the host there is a corresponding gene for pathogenicity'. One is a plant gene called the resistance (*R*) gene. The other is a pathogen gene called the avirulence (*Avr*) gene. Plants producing a specific R gene product are resistant towards a pathogen that produces the corresponding *Avr* gene product.

Early studies of genetics of rust resistance were complemented with the application of aneuploid techniques, resulting in a progressive explanation of resistance loci, including their characteristic low infection types, chromosomal location and linkage to traits of interest to wheat breeders. Progress became more rapid with the application of molecular marker technology and the development of mapping populations that permitted replicated studies across a range of environments. Rust resistance genes have been identified progressively in wheat and currentlythere are 75, 76 and 59 genes for yellow, brown and black rust resistance that have been designated. In addition, many uncharacterized resistances await further genetic investigation. A large proportion of designated resistance genes have been shown to be pathotype specific, including seedling effective genes and adult plant resistance (APR). In general, genes for APR confer a partial, often slow rusting phenotype (Singh *et al.* 2011b). Life of effective race-specific resistance genes can be prolonged by using gene combinations, an alternative approach being implemented at CIMMYT is to deploy varieties that possess APR based on combinations of minor, slow rusting genes (Singh *et al.* 2014). APR genes individually provide low levels of resistance and combinations of three or more genes are essential to express commercially adequate levels of resistance (Bariana and McIntosh 1993).

Host pathogen interaction at molecular level

Two major classes of rust resistance (R) gene are the NBS-LRR genes (McHale *et al.* 2006) and the cell surface pattern recognition receptors (PRR) (Song *et al.* 1995). Most plant disease resistance (R) proteins contain a series of leucine-rich repeats (LRRs), a nucleotide-binding site (NBS), and a putative amino-terminal signaling domain. They are termed NBS-LRR proteins. The protein products of the PRRs contain extracellular, juxtamembrane, transmembrane and intracellular non-RD kinase domains. Genetically, the LRRs of plant R proteins are determinants of response specificity, and their action can lead to plant cell death in the form of the familiar hypersensitive response (HR). This class of resistance genes are pathogen race specific in their action, effective at all plant growth stages.

Genetic linkages

The rust resistance genes reported to be tightly linked or pleiotrpic are *Yr*9/*Lr*26/*Sr*31,*Lr*19/*Sr*25,*Lr*20/*Sr*15,*Lr*24/*Sr*24,*Lr*34/*Sr*57/*Yr*18,*Lr*37/*Sr*38/*Yr*17,*Yr*30/*Lr*27/*Sr*2,*Lr*46/*Sr*58/*Yr*29,*Yr*40/*Lr*57,*Yr*46/*Lr*67/*Sr*55,*Yr*47/*Lr*52. When a large number of markers are segregating simultaneously in a mapping population and these markers are to be placed on a linkage map, the first step is to group the markers into linkage groups. Linkage groups are established by considering all estimates of recombination frequencies based on LOD score (Logarithm of Odds). If two markers are significantly linked (by LOD value) they belong to the same linkage group.

Using infection type response to characterize rust resistance genes

Seedling or all stage resistance genes can be postulated by testing host genotypes against an array of pathotypes differing in virulence phenotypes (Browder 1973). In this method, the genotype/cultivar under gene postulation is planted alongwith isogenic lines having known resistance genes and inoculated separately at seedling stage with an array of virulences of a rust pathogen. Rust reactions are recorded as per Stakman *et al.* (1962). Postulation of resistance genes is inferred through gene matching technique (Burton *et al.*1969, Browder 1973). At times rust resistance genes can also be characterized through genetic linkage, molecular markers, cytogenetic analysis and morphological markers.

Genetics of rust resistance in Indian wheat

Nagarajan *et al.* (1987) documented rust resistance genes in wheat material, subsequently updates were also published (Nayar *et al.* 2001, Bhardwaj *et al.* 2010a). In between diverse information on genetics of wheat rust resistance has been added (Nayar 1989, Sawhney 1994, Tomar and Menon 2001, Nayar *et al.* 2001, Walia and Kumar 2008, Bhardwaj 2011). Based on the available information, it can be concluded that brown rust resistance of Indian wheat is based on *Lr*1, *Lr*3, *Lr*9, *Lr*10, *Lr*13, *Lr*14a, *Lr*17, *Lr*18, *Lr*19, *Lr*22, *Lr*23, *Lr*24, *Lr*26, *Lr*28,

*Lr*34,*Lr*46 and *Lr*49. Among these *Lr*26, *Lr*13, *Lr*23 and *Lr*34 have been characterized in many wheat lines. Presently *Lr*24, *Lr*25, *Lr*29, *Lr*32, *Lr*39, *Lr*45, *Lr*47 are resistant to all the pathotypes of *P. triticina* in India (Bhardwaj *et al* 2010b). *Sr*2, *Sr*5, *Sr*6, *Sr*7a, *Sr* 7b, *Sr* 8a, *Sr* 8b, *Sr* 9b, *Sr* 9e, *Sr*11, *Sr*12, *Sr*13, *Sr*17, *Sr*21, *Sr*24, *Sr*25, *Sr*30 and *Sr31* have been characterized in Indian wheat material. Among these *Sr*2, *Sr*11 and *Sr*31were very common in bread wheat whereas Sr7b, *Sr*9e and *Sr*11 conferred black rust resistance in many durum lines. *Sr*26, *Sr*27, *Sr31*, *Sr*32, *Sr*33, *Sr*35, *Sr*39, *Sr*40, *Sr*43 and *Sr*Tt3 (Jain *et al*. 2013) confer resistance against Indian population of *P. graminis tritici*. Yellow rust resistance of wheat in India is based on *Yr*2, *Yr*A, *Yr*9 and *Yr*18. *Yr*5, *Yr*10, *Yr*11, *Yr*12, *Yr*13, *Yr*14, *Yr*15, *Yr*16, Yr*sp*, Yr*sk* (Prashar *et al*. 2015) are resistant against *P. striiformis* in India.

DNA polymorphism and genome sequencing in wheat rusts

Traditional differential and subsequently Near Isogenic Lines (NILs), markers like biochemical were used to study the variability in pathogens. With the advancement in the field of molecular biology we have been able to virtually uncover unlimited number of DNA markers for their use in plant pathology (Singh and Hughes 2006). The most popular markers employed in the variability studies of *Puccinia* sp. include Restriction Fragments Length Polymorphism (RFLP), Random amplified polymorphic DNA (RAPD), Amplified Fragment Length Polymorphism (AFLP), Simple Sequence Repeats (SSR), Internal Transcribed Sequence (ITS) and Single Nucleotide Polymorphism (SNP) (Cooke and Lees 2004**,** McCartney *et al.* 2003). Markers used to characterize wheat rust polymorphism are as follows: Black rust (SSR, RFLP, RAPD, AFLP), brown rust (SSR, AFLP, RAPD), and yellow rust (SSR, RAPD, AFLP, RFLP, ISSR).

Whole genome sequencing is another aspect of molecular biology which has opened up research areas from where we can uncover a number of unsolved mysteries.The genome of *P. graminis* f. sp. *tritici* (Duplessis *et al.* 2011), *P. striiformis* (Cantu *et al.* 2011) and *P. triticina* have been sequenced at Broad Institute, USA.

Molecular markers for rust resistance genes in wheat

More than 210 genes conferring resistance to rust fungi (http://www.shigen.nig.ac.jp/wheat/komugi/genes/symbolClassList.jsp) have been catalogued. The size of this genome as well as the high percentage (over 90%) of non-coding sequences and three genomes A, B and D with 7 homoeologous chromosomes cause molecular identification and cloning of wheat resistance genes to be difficult. Each of 42 hexaploid wheat chromosomes has the average size of about 800 Mbp. Physical distance between crossing-overs (=1 cM) varies from 0.3 to 3 Mbp (Feuillet *et al.* 1995). Wild relative species of wheat usually have one genome in common with wheat, which makes them very helpful for searching and mapping new resistance genes. Because various wheat-related species carry different genomes (*Triticum* sp., genome B; *Aegilops speltoides*, genome S, similar to B; *Triticum boeoticum*, genome A; and *Aegilops squarrosa*, genome D), they have been and still are used as sources of resistance genes in breeding. A frequent way to transfer the resistance genes is using wheat lines with translocation of a chromosome fragment carrying the gene from a wild species. This was done in the case of genes *Lr* 19, *Lr* 24 and *Lr* 29 derived from *Agropyron elongatum* (Procunier *et al.* 1995, Schachermayr *et al.* 1995**,** Prins *et al.* 1996).

The first molecular STS marker was developed by Schachermayr *et al.* (1994**)** for the *Lr* 9 gene derived from *Aegilops umbellulata*, and soon results on the identification of several markers for other rust resistance genes were published. The development of new DNA-based assays has led to their application for designing direct and tightly linked markers – restriction fragments length polymorphism (RFLP), random amplified polymorphic DNA (RAPD), cleaved amplified polymorphic sequence (CAPS), sequence characterized amplified regions (SCAR), sequence tagged sites (STS) and simple sequence repeats (SSR) microsatellites to identify individual resistance genes in wheat accessions.

Intensive research on genomics over the last few decades has led to identification of an array of DNA-based markers, popularly called as "molecular markers". DNA marker systems can be grouped into three classes (Gupta *et al.* 1999): (i) Hybridization –based markers such as restriction fragment length polymorphism (RFLP), (ii) PCR-baesd markers: random amplification of polymorphic DNA (RAPD), amplified fragment length polymorphism (AFLP) and microsatellite or simple sequence repeat (SSR), and (iii) DNA chip and sequence-based markers: single nucleotide polymorphism (SNP).

Hybridization-based markers

These are also called restriction fragment length polymorphism (RFLP) markers and thesystem was initially developed to map the human genome (Botstein *et al.* 1980). It is based on hybridization of DNA sequence called probes (usually labelled with radioactive isotopes) to genomic DNA restricted with restriction enzymes. Variation in number and position of restriction sites among individuals defines polymorphism (Semagn *et al.* 2006a). Several rust resistance genes (*Lr*1, *Lr*24, *Lr*35, *Lr*57, *Sr*2, *Sr*22,*Yr*15and *Yr*40) were mapped using RFLP markers (Paull *et al.* 1994**,** Schachermayr *et al.* 1995**,** Nelson *et al.* 1995**,** Sun *etal.* 1997, Seyfarth *et al.* 1999, Ling *et al.* 2003, Kuraparthy *et al.* 2007). This marker system is not routinely used now.

PCR-based markers

Randomly amplified polymorphic DNA (RAPD)

Polymerase chain reaction (PCR) is used to amplify DNA fragments (Semagn *et al.* 2006a). The RAPD marker system involves the use of single primer (short single stranded DNA sequence) to amplify random regions throughout the genome (Williams *et al.* 1990). Rust resistance genes *Lr19, Lr24, Lr34, Sr22* and *Yr15* were tagged using RAPD markers (Schachermayr *et al.* 1995, Sun *et al.* 1997, William *et al.*

1997, Khan *et al.* 2005, Gupta *et al.* 2006). This system is not currently used because of lack of reproducibility, low levels of polymorphisms, amplification of multiple bands and dominant inheritance (Semagn *et al.* 2006a).

Amplified fragment length polymorphism (AFLP)

This marker system combines the properties of RFLP and RAPD marker systems. It involves selective PCR amplification of fragments from a pool of restricted genomic DNA and selective amplification primers are comprised of sequences complementary to adapters and one to three random nucleotides attached at the 32 end. Primers specifically bind to the fragments containing matching ends to selectively amplify different sized fragments (Vos *et al.* 1995). AFLP markers for rust resistance genes *Lr*3*, Lr*26*, Sr*30*, Sr*31*, Sr*39*, Yr*7*, Yr*9and *Yr*29 have been reported (Bariana *et al.* 2001**,** Mago *et al.* 2005**,** Dieguez *et al.* 2006**,** Rosewarne *et al.* 2006**,** Mago *et al.* 2009).

Sequence tagged site (STS)

Primers designed to amplify DNA sequence that are specific to a locus and found nowhere else in the genome are called STS markers (Gupta *et al.* 1999, Semagn *et al.* 2006b). STS markers for rust resistance genes *Lr*9, *Lr*20/ *Pm*1 and *Lr*24 (RAPD-derived, Schachermayr *et al.* 1994**,** Schachermayr *et al.* 1995**,** Neu *et al.* 2002); *Lr*35 and *Sr*22(RFLP-derived, Seyfarth *et al.* 1999**,** Periyannan *et al.* 2011); *Lr*19*, Lr*26*, Lr*28*, Lr*37*,Sr*R*, Sr*24*, Sr*26*, Sr*31*, Sr*38*, Sr*39*, Yr5* and *Yr*9 (AFLP-derived; Naik *et al.* 1998, Prin s*et al.* 2001, Mago *et al.* 2002, Mago *et al.* 2005, Smith *et al.* 2007, Mago *et al.* 2009) and *Lr*34, *Sr*13, *Sr*25 and *Sr*26 (EST-derived; Lagudah *et al.* 2006, Liu *et al.* 2010, Simons *et al.* 2011) were reported.

Microsatellites or simple sequence repeats (SSRs)

Tandem repeats of a few base pairs (1-6) throughout the genomes are also called microsatellites or SSRs (Tautz and Renz 1984). The number and type of repeats determine polymorphism among different individuals. Condit and Hubbel (1991) were the first to apply SSRs for plant genome analysis. High levels of polymorphism, lowcost and amenability for automation are the major advantages of this system (Hayden *et al.* 2006). SSR markers linked with rust resistance genes *Lr*19*, Lr*22a*, Lr*24*, Lr*34*/Yr*18*,Lr*39*, Lr*42*, Sr*2*, Sr*6*, Sr*22*, Sr*36*, Sr*35*, Sr*40*, Sr*Web*, Yr*5*, Yr*10*, Yr*10vav*, Yr*36*, Yr*CH42 and *Yr*ZH84 are available for marker assisted selection (Schachermayr *et al.* 1995**,** Raupp *et al.* 2001**,** Bariana *et al.* 2002**,** Sun *et al.* 2002, 2010, Spielmeyer *et al.* 2003, 2005, Khan *et al.* 2005, Uauy *et al.* 2005, Bossolini *et al.* 2006**,** Gupta *et al.* 2006**,** Li *et al.* 2006**,** Hiebert *et al.* 2007**,** Tsilo *et al.* 2008, 2009, Wu *et al.* 2009**,** Hiebert *et al.* 2010, Olson *et al.* 2010**,** Zhang *et al.* 2010).

Insertion site-based polymorphism (ISBP)

Transposable elements have unique insertion sites that are highly conserved betweendifferent cultivars of plants. ISBP markers were used as PCR based markers by Flavell*et al.* (1998). ISBP markers were also developed from the BAC end sequences ofchromosome 3B (Paux *et al.* 2006).

DNA chip and sequenced-derived markers

All the molecular markers described above are gel-based and are labour-intensive andtime-consuming. DNA-chip based methods are high throughput and highly efficient.

DArT markers

Diversity arrays technology (DArT) marker system was developed to provide a cost effective whole-genome fingerprinting tool and efficient for species which havecomplex genomes and lack prior DNA sequence information (Jaccoud *et al.* 2001, Wenzl *et al.* 2004). A single DArT assay is capable of typing of hundreds to thousands of single nucleotide polymorphism (SNPs) and insertion/deletion (indel) polymorphisms distributed throughout the genome. Details of this technology are provided on their website (http://www.diversityarrays.com/). DArT marker system hasnow evolved further and it is now referred to as DArTseq. It involves sequencing of the genomic representations on the Next Generation Sequencing (NGS) platforms. Many wheat populations have been mapped using DArT and DArTseq system. DArT markerslinked with *Lr*34*/Yr*18*/Pm*38*, Lr*46*/Yr*29*/Pm*39*, Sr*2*, Sr*6*, Sr*25 and *Yr*51are available for marker assisted selection (Lillemo *et al.* 2008, Tsilo *et al.* 2009, Yu *et al.* 2010, Randhawa *et al.* 2014*).*

Single nucleotide polymorphism(SNPs)

SNPs are single nucleotide variations in the DNA sequence of individuals. These are the most abundant molecular markers in the genome (Soleimani *et al.* 2003). SNP genotyping is highly efficient due to their amenability to automation. In wheat 9K SNP (Cavangah *et al.* 2013) and 90K SNP (Wang *et al.* 2014) chips were developed. Competitive allele specific primers (KASP) have been designed and sequences areavailable at the Cereals Database (http://www.cerealsdb.uk.net/). SNP markers linkedwith economic traits including resistance to rust diseases are preferred by breedingcompanies due to their amenability to high throughput.

Management of wheat rusts

A vigil is always required to combat wheat rusts. A combination of cultural management practices with disease resistance and perhaps fungicide applications (under emergency situation) will be the most effective measures of managing the wheat rust diseases.

Breeding for rust resistance

Genetic resistance is the most effective, least expensive and environmentally safe means of rust diseases management. When adequate genetic resistance is achieved in a cultivar, no other prophylactic measures are necessary. A few historic cultivars, such as Thatcher and Hope (Hare and McIntosh 1979) for black rust, Americano 25, Americano 44d,

Surpreza, Frontana and Fronteira (Perez and Roelfs 1989) for brown rust, and Wilhelmina, Capelle-Desprez, Manella, Juliana and Carstens VI (Stubbs 1985) for yellow rust, have maintained some resistance for many years. Eagle carrying *Sr*26 was released in Australia in 1971 and has remained resistant to black rust for long (McIntosh *et al*.1995). In most, if not all the cases, the failures have been due to the rush of releasing new varieties even when these do not conform the disease standarads or due the inadequate knowledge of the virulences present in the pathogen population. In other cases, mutations or perhaps a recombination of existing virulence combinations occurred and rendered host susceptible. There have been haphazard efforts to breed for rust resistance. With the clear differentiation between vertical and horizontal resistance (Van der Plank 1963), scientists have used various approaches to control plant diseases. Wheat has always been a focus of efforts and a model system for rust resistance breeding.

The key points for the management of wheat rusts has always been to avoid large scale planting of single genotype/ similar resistance and deploy varieties with diverse resistance, if possible then resistance based on more than one effective gene. Blend of seedling, race specific adult plant resistance and non race specific adult plant resistance/ slow rusting would be useful while identifying genes/varieties for deployment. It will not only avoid the epidemics of wheat rusts but also increase the self life of wheat varieties and discourage the evolution in pathogens.

Source of wheat rust resistance

Breeding for rust resistance always requires a constant inflow of novel sources of resistance genes, due to the appearance of new virulent pathogen races like Ug99. While most rust resistance genes originate from hexaploid wheat, some were introduced from related cereal species. According to crossability with hexaploid wheat, other related species are divided into 3 major gene pools: The primary gene pool; the secondary gene pool; and the tertiary gene pool (Mujeeb-Kazi and Rajaram 2002). Many sources of resistance including the alien sources have been used for black rust resistance. Landmark beginning was made by introgression of rye (*Secale cereale* L.) gene into bread wheat (1B/1R translocation or substitution) in 1973 (Mettin *et al*. 1973, Zeller 1973) which carries *Lr* 26/ *Sr* 31/ *Yr* 9. Sometimes yield reduction is associated with alien gene introgression in wheat cultivars. For instance, *Sr*26 showed 9% yield penalty associated with the original 6AS.6AL-6Ae#1L segment originally introgressed into the distal region of the long arm of hexaploid wheat chromosome 6A via an alien segment from *Agropyron elongatum* (syn. *Thinopyrum ponticum*) (Knott 1961). *Sr26* is one of the few known major resistance genes effective against the *Sr31*-virulent race Ug99 (TTKSK) and its *Sr24*-virulent derivative (TTKST). Subsequently, a number of resistance genes have been introgressed into wheat from the alien sources (McIntosh *et al*. 1995).

Durable, slow rusting, adult plant resistance to rusts

The durability of genetic resistance against rust diseases in cultivated wheat still remains a major challenge in modern agriculture and is of special concern to both plant breeders and farmers. Durable resistance to a disease is resistance that remains effective during its prolonged and widespread in an environment favourable to the disease. The association of durable resistance with both major and minor genes, depending on different host-pathogen systems and the parasitic behaviour of pathogens and their degree of host specialization, has been much discussed (Parlevliet 1993). Rather than displaying the immune phenotype, APR is most active in the mature plant, and tends to slow (rather than completely prevent) the development of the pathogen (Singh *et al.* 2005). This 'slow rusting' is thought to result from the host's ability to lengthen the time required for the pathogen to colonize and to reduce the pathogen's sporulation capacity. Based on number of pustules, uredial size, latent period, incubation period, uredospore production, slow rusting resistance is characterized. Slow rusting lines have low area under disease progress curves (AUDPC). It is also a complex trait and is supposed to be conditioned by many genes (Singh *et al.* 1991) and may not follow the Gene-for-Gene hypothesis religiously (Nayar *et al*. 2003). A simple formula:7*(1+2/2)+ 7*(2+3/2)+7*(3+4/2), where 1,2,3 are the disease co-efficients of 1st, 2nd, 3rd, 4th rust observations. Importantly, APR tends to be race non-specific, and therefore more durable than race-specific resistance.

Resistance gene *Sr*2*,* in addition to other unknown minor genes derived from cultivars Hope and H-44, provided the foundation for durable resistance to black rust in germplasm in different places like University of Minnesota in the USA and Sydney University in Australia, and in the spring wheat germplasm developed by Dr N E Borlaug as part of a program sponsored by the Mexican Government and the Rockefeller Foundation. Unfortunately, not much is known about the other genes in the *Sr*2 complex and their interactions. Knott (1989) has shown that adequate levels of multigenic resistance to black rust can be achieved by accumulating approximately five minor genes. In his studies the genes were different from *Sr*2. *Lr34, Lr46, Lr67* and other minor genes for durable resistance to brown rust.

Genetic analysis of Frontana and several CIMMYT wheats, possessing excellent slow rusting resistance to brown rust worldwide, has indicated that such adult plant resistance is based on the additive interaction of *Lr*34 and two or three additional slow rusting genes (Singh and Rajaram 1992). Brown rust severity observed in Mexico on most slow rusting cultivars was related to the number of minor genes they carry. When susceptible cultivars display 100% brown rust severity, cultivars with only *Lr*34 display approximately 40% severity; cultivars with *Lr*34 and one or two additional minor genes display 10-15% severity; and cultivars with *Lr34* and two or three additional genes display 1-5% severity. Brown rust may increase to unacceptable levels on cultivars carrying only *Lr*34, or *Lr*34 and one or two additional

genes. However, cultivars with *Lr*34 and two or three additional genes show a stable response in all environments tested so far, with final brown rust ratings lower than 10%. The presence of *Lr*34 can be indicated by the presence of leaf tip necrosis in adult plants, which is closely linked with it (Singh 1992a). At the cellular level this kind of resistance was seemingly operating effectively.

*Yr*18, *Yr*29 and other minor genes for durable resistance to yellow rust. Singh (1992b) and McIntosh (1992) indicated that the moderate level of durable adult plant resistance to yellow rust of the CIMMYT-derived US wheat cultivar Anza and winter wheats such as Bezostaja is controlled in part by the *Yr*18 gene. This gene is completely linked to the *Lr*34 and *Sr*57. The level of resistance it confers is usually not adequate when present alone. However, combinations of *Yr*18 and 3-4 additional slow rusting genes result in adequate resistance levels in most environments (Singh and Rajaram 1994). Genes *Lr*34, *Sr*57 and *Yr*18 occur frequently in germplasm developed at CIMMYT and in various countries. The recently identified slow rusting gene *Yr*29 is completely linked to gene *Lr*46, which confers moderate resistance to brown rust (William *et al.* 2003). Likewise *Lr*67/*Sr*55/*Yr*46 is also known to provide slow rusting/adult plant type of resistance to brown rust under Indian conditions. There had been statistically insignificant yield penulty due to brown, black and yellow rusts in the varieties based on *Lr*67 resistance. Durability of such resistance can be expected if the cultivar's low disease severity is due to the additive interaction of several (4 to 5) partially effective genes.

Relatively little efforts were made historically to characterize slow rusting resistance genes even though this kind of resistance is known to exist for a long time (McFadden 1930). The most studied, and possibly the most effective, now cloned slow rusting leaf rust resistance gene *Lr*34 located on chromosome arm 7DS, has maintained its moderate effectiveness for over 60 years of use (Krattinger *et al.* 2009). Gene *Lr*46, located on chromosome 1BL (William *et al.* 2003), was first identified in the CIMMYT-derived Mexican variety Pavon 76. This gene is widely distributed in germplasm from CIMMYT and other countries. It also confers slow rusting to yellow rust and slow mildewing to powdery mildew and is designated as *Yr*29 and *Pm*39, respectively. Though not designated, this gene also confers slow rusting to black rust (Bhavani *et al.* 2011). Herrera-Foessel *et al.* (2014) demonstrated that APR locus *Lr67*/*Yr46* has pleiotropic effect on black rust and powdery mildew resistance and is associated with leaf tip necrosis. Genes are designated as *Sr55*, *Pm46* and *Ltn3*, respectively.

Field assessments of breeding populations and parental lines have frequently described partial, slow-rusting and temperature-sensitive yellow rust resistance that can be measured as quantitative characters and thus referred to as quantitative trait loci (QTLs). Bariana and co-workers (2010) identified QTLs controlling APR to yellow rust in Kukri/Janz-derived doubled haploid (DH) population through molecular mapping and identified genotypes combining resistance from both parents.

Chemical measures

As an emergent tool for contolling wheat rusts, chemical method of wheat rust diseases management has been successfully used in Europe, permitting high yields (6 to 7 tonnes/ha) and where prices for wheat are supported (Buchenauer 1982). Chemicals were also used to control a brown rust epidemic in 1977 in the irrigated Yaqui and Mayo Valleys of Mexico (Dubin and Torres 1981). Elsewhere, chemicals have had limited use on high-yielding wheat in the Pacific Northwest of the United States for yellow and brown rust management. For controlling initial load of inoculums or under high yellow rust incidence in India, fungicides belonging to triazole group such as Propiconazole 25% EC (Tilt), Tebuconazole 25% EC (Folicur) and Triadimefon 25% EC (Bayleton) have been used effectively at the rate of 0.1% for the management of wheat rusts.

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