



Wheat Rust Research-Shifting Paradigms Globally

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1.1 Introduction

Many biotic and abiotic factors impede the efforts to increase wheat production worldwide. Among the biotic constraints, rusts are the major obstacles in wheat production. Wheat rusts have been devastating pathogens since time immemorial. There are records of their occurrence as early as 1300 BC (Kislev 1982). Even in the modern literature these have been categorized as historic pathogens, capable of causing huge losses to wheat crop worldwide (Stakman and Harrar 1957). Leaf rust (*Puccinia triticina*), stem rust (*P. graminis* f. sp. *tritici*) and stripe rust (*P. striiformis* f. sp. *tritici*) are wheat diseases of global importance. The epidemic losses caused due to rusts on wheat are the interplay of susceptible host, virulent pathotypes of rust pathogens, favourable environment, time duration and human indulgence (Fig. 1.1). Among these factors, matching virulences of rust pathogens, susceptible wheat cultivar, stage of crop and favourable environment are the key factors in causing epidemics. The stage of wheat crop at which the infection occurs is very crucial. The early infections lead to more loss than late ones. The stage of wheat crop is also important because the adult plant resistance is triggered after the third leaf formation. Thus, a variety which is susceptible in initial stages could become resistant later on. This is one of the reasons that wheat rust epidemics have become infrequent, as many unknown adult plant resistance genes have conferred field resistance to rusts.

Among the wheat rusts, leaf (brown) rust is the most common, occurring mostly on the leaves, leaf sheaths, leaf blades and spreads well if the temperature ranges from 10 to 30 °C. Losses to wheat due to leaf rust can be up to 50% (Anonymous 1982). Leaf rust occurs throughout the wheat-growing areas in the world. Stem

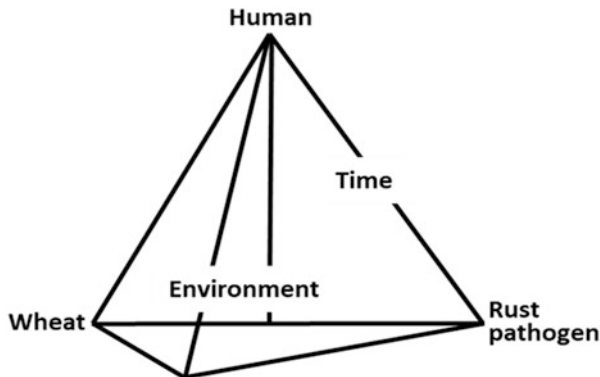
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Fig. 1.1 Variables of wheat rust epidemics



(black) rust of wheat generally occurs on stems, leaf sheaths and sometimes on leaves. It is favoured by warmer wheat-growing situations (15–35 °C) in the world and occurs in central and peninsular India. It appears late in the season; therefore, it is not a problem in the Northern hills zone, Northwestern and Northeastern India. Stem rust can cause up to 100% loss on susceptible wheat varieties. Stripe (yellow) rust of wheat is mainly a disease of cooler areas (2–15 °C); however, recently high temperature (up to 20 °C) loving stripe rust cultures have been observed in many countries. In many cases, the crop losses due to wheat stripe rust can be severe (50%) and under extreme epidemic conditions, it can lead to 100% losses (Roelfs et al. 1992). In India, wheat stripe rust is a major problem of Northern hills zone and Northern plains zone.

Wheat rusts have principally remained under control for quite some time, and largely it was thought that these have been managed. However, occurrence of *Yr9* virulences in Syria, Iran in 1990, 1992 and 1993 caused severe stripe rust epidemics in Western Asia had sent alarming bells. In April 1994, the *Yr9* virulences rendered Veery#5 cultivars such as Pak81, Pirsabak85 and Seri82 susceptible in the NWFP of Pakistan (Nagarajan and Saari 1995). Subsequently, *Yr9* virulences have created havoc in China, the United Kingdom, Africa and other parts of the world. In India, virulences on *Yr9* appeared in 1996 and 2001 (Prashar et al. 2007). Later on, the occurrence of *Sr31* virulences also called Ug99 in Uganda during 1998 (Pretorius et al. 2000) caused an international concern. It rendered gene *Sr31* ineffective after its use for about four decades. Consequently, about 40% of world's wheat material came under the threat of stem rust epidemic (Singh et al. 2011). During the last decade, 13 pathotypes in the lineage of Ug99 have been identified from 13 countries (CIMMYT 2019).

These events have reminded us of the adage of Dr. Norman Borlaug, 'Rust never sleeps'. There is always a continuous race for supremacy between wheat breeders and rust pathogens. Breeders develop new wheat varieties, whereas rust pathogens evolve new pathotypes/virulences which render resistant varieties susceptible. New virulences evolve through either sexual reproduction (in areas or countries where alternate hosts are functional) or mutations and somatic hybridization (where

alternate hosts are not functional). Mutation is a very common mode of evolution of new pathotypes especially when alternate hosts are non-functional. Virulence genes are mostly recessive. Recessive mutations are more frequent than the dominant ones. Frequency of recessive mutations is about 1×10^{-5} – 110^{-6} . The wheat rusts being dikaryotic would require a double mutant of the order of 1×10^{-10} – 1×10^{-12} . The urediniospores are produced in trillions, and therefore the probability of occurring a mutant and resultant infection on a previously resistant cultivar carrying single gene for resistance is very high. This phenomenon leads to the susceptibility of wheat varieties and succumbing of vertical resistance genes (Knott 1989) popularly called boom and bust cycle.

Hence, efforts of the scientists are required to remain few steps ahead of the pathogen. Therefore, a continual vigil on wheat rusts is required. Monitoring of pathotypes in early stages and evaluation for rust resistance, pre-emptive breeding and strategic deployment of wheat varieties using pathotype zonation have now become integral part of wheat rust management system (Bhardwaj et al. 2019). With the landmark invention of Craigie (1927), demonstrating the role of pycnia in the life cycle of stem rust fungus, our understanding of wheat rust fungi has increased. The transition from simple to refined science over these 100 years has been described on the succeeding pages.

1.2 Detection and Diagnosis

Precise diagnosis of plant diseases or detection of the pathogens causing these diseases is a pre-requisite for employing effective management strategies. Traditionally, wheat rusts are being diagnosed based on the symptoms and signs of these diseases on wheat. The characteristic symptoms of wheat rusts are as follows: (1) Stem rust: symptoms appear as small-brownish coloured pustules lower leaves and stem at early stages of infection. The urediospores are oval ($25\text{--}30 \mu \times 17\text{--}20 \mu$ approximate) in size. As the disease progresses, the pustules become larger and form lesions of dark-brown colour. Towards the end of the disease cycle, dark black coloured teliospores ($40\text{--}60 \mu \times 15\text{--}20 \mu$ approx.) are produced. (2) Leaf rust: at initial stage the disease or symptoms appear on leaf blade as light orange colour round to oval ($0.5\text{--}2.3 \text{ mm}$ approx.) pustules. Later on, these pustules appear in partially yellow brownish colour. At the advanced stage, the pustules turn brown. (3) Stripe rust: in case of stripe rust, the uredia are mostly linear, citron orange to yellow colour and typically narrow to form stripes on the leaves. The uredospores developed in case of stripe rust are round to ovate in shape ($25\text{--}35 \mu \times 20\text{--}35 \mu$ approx.). The disease appears on leaf, leaf sheaths, awns, etc. Diagnosis of these pathogens becomes slightly difficult when the symptoms of two or more wheat diseases including rusts are merged together. Another approach to diagnose these diseases is through the use of molecular markers, which have been utilized for precise forecasting of disease outbreak. Molecular markers could be utilized for estimating the initial inoculum load of the pathogen, and therefore, prophylactic disease control practices could be placed well before the occurrence of a disease

outbreak. Molecular marker-based diagnostic assays have been developed for the early detection of *Puccinia* spp. causing wheat rusts (Aggarwal et al. 2017; Lihua et al. 2008; Liu et al. 2014).

A range of image-based approaches assimilating image acquisition and analysis are described to have massive prospects for accurate disease diagnosis. These approaches include thermal, chlorophyll fluorescence, multispectral, hyperspectral and RGB (red, green and blue)-based sensors, some of which (hyperspectral imagery) have been utilized for wheat rusts' detection. Performance of ten spectral vegetation indices was evaluated for the identification of yellow rust infection on wheat leaves (Devadas et al. 2009). Likewise, diverse wheat rust diseases were detected based on hyperspectral measurements of vegetation indices. Some of the indices were able to successfully detect yellow rust infection (Ashourloo et al. 2014). Automatic embedded image processing system was developed for the detection of *Puccinia triticina* and grading disease diagnosis (Xu et al. 2017). These image-based approaches could be further customized and utilized as agricultural robot to inspect, diagnose and classify crop diseases in the field.

1.3 Variability in *Puccinia* Species on Wheat

A resistant wheat variety becomes susceptible after a few years of its cultivation. The detailed investigations of this cause have led to the identification of variants in a rust pathogen named races or pathotypes. Many scientists have used these terms interchangeably. Pathotype is a subdivision of a species or a *forma specialis* distinguished by common characters of pathogenicity or symptoms produced on varieties (differentials) of a host. Pathotype is used to describe the type (reference) culture of a pathovar. A list of 294 races of *P. graminis tritici* was given by Stakman et al. (1962). Likewise, an updated list of 226 variants in *P. triticina* was published by Johnston and Browder (1966) and 66 of *P. striiformis tritici* by Johnson et al. (1972). However, with the propounding of gene for gene hypothesis by Flor (1956), it was realized that pathotype identification systems are not on sound footings. Subsequently, pathotype identification systems based on Near Isogenic Lines (NILs) were put in to practice in different countries. The pathotype identification systems followed by various countries are different. Huerta-Espino et al. (2011) have discussed the systems for naming pathotypes of *P. triticina* being followed world over. There was a move internationally to have a common system for the identification of pathotypes of *Puccinia* spp. on wheat. However, it did not materialize. The main reason for the disagreement was differences in populations of various regions/countries. Few genes are effective in one but are ineffective in other countries. Therefore, common differentials for the identification of pathotypes could not come into existence. Consequently, different countries generally follow different differentials or identification systems. However, it is easy to decode every system and know the population structure of any country by knowing the avirulence and virulence structure of pathotypes.

1.4 Shifting Virulence Patterns in Population of *Puccinia* Spp. on Wheat

Wheat and rusts have coexisted and coevolved together for centuries. More variability and advanced populations of rust pathogens have been seen either around the centres of origins or in areas where more vibrant and dynamic wheat breeding programmes are going on. The evolution of rust pathogens is independent of wheat. However, a resistant wheat variety favours the selection of a virulent mutant. Introgression of new genes is countered easily by the wheat rust pathogens. Even the transgenic varieties developed through 1B.1R translocation, *Lr24/Sr24*, and many more were rendered susceptible, shortly after their commercialization. In some cases, virulent pathotypes of rust pathogens have evolved even before a wheat variety based on the corresponding resistance was developed or commercialized. Incorporation of a new resistance gene is easily matched by the dynamic rust pathogens. Consequently, the pathogen acquires virulence every time with one step gain in virulence and we are heading towards super races (Bhardwaj et al. 2019). Breeders have been called inciters of epidemics (Johnson 1961). Replacement of wheat varieties is readily coped up with the shift in virulence patterns and building up of matching pathotypes. Therefore, frequent shift in virulence patterns is a common phenomenon in wheat rust pathogens. The rapid increase in *Yr9* virulent pathotypes in NWPZ of India was due to the cultivation of PBW343 with unique resistance and clean foliage. A distinct shifting virulence pattern can be seen in stripe rust pathogen as new pathotypes have emerged almost every 5–6 years in northern parts of India (Prashar et al. 2007; Gangwar et al. 2017). The wheat cultivar PBW343 was released in 1995, adopted quickly by farmers of Punjab where it occupied more than 90% wheat area of this state, and about seven million ha area throughout the Indo-Gangetic Plains (Singh et al. 2017). Subsequently, a new pathotype 78S84 emerged in 2001 with matching virulence to PBW 343, and this pathotype became predominant.

Cultivation of PBW 343 had to be discontinued due to the build-up of pathotype 78S84 of *P. striiformis*. Later on, PBW343 was replaced with a number of wheat varieties, and pathotype 78S84 was replaced by 46S119. In 2014, three new pathotypes 110S119, 110S84 and 238S119 which possessed combined virulence to Strubes Dickkopf and Suwon92xOmar were detected (Gangwar et al. 2019), and now 238S119 has become most prevalent. It was observed that with the emergence of new pathotypes, old pathotypes get replaced due to the cultivation of new wheat cultivars which guide shifts in pathogen's virulence patterns. Competitive ability and fitness potential decide the predominance of pathotype as we have seen it in the case of different pathotypes of *Puccinia triticina* (Bhardwaj et al. 2014a; Gupta et al. 2018).

1.5 Alternate Hosts for Wheat Rust Fungi

It is well known that in off-season, wheat rusts complete a part of their life cycle on the alternate hosts. Prior to 2010, alternate hosts were not known for *P. striiformis*. In a landmark discovery, Jin et al. (2010) could prove that *Berberis chinensis*, *B. koreana*, *B. holstii* and *B. vulgaris* are alternate hosts for wheat yellow rust fungus. Later on, in China too, susceptible *Berberis* spp. were found which may facilitate in sexual cycle of *P. striiformis* f. sp. *tritici* and contribute to the diversity of the fungus (Zhao et al. 2013). The leaf rust pathogen (*Puccinia triticina*) completes its sexual life cycle on alternate hosts like *Thalictrum* spp., *Isopyrum fumarioides*, *Clematis* spp. and *Anchusa* spp. (Jackson and Mains 1921; de Oliveira and Samborski 1966). *Berberis*, *Mahonia* and *Mahoberberis* are the alternate hosts of *Puccinia graminis* f. sp. *tritici*. However, under Indian conditions, none of the alternate hosts is functional; therefore, fungus perpetuates as mycelia or uredial stage only. Consequently, we reinvestigated the role of alternate hosts in the life cycle of wheat rusts in India. Inoculations of aeciospores from *Berberis* spp. growing in Himachal, Uttarakhand and Nepal did not cause any infection on the wheat leaves. Therefore, in the absence of alternate hosts, drawing a parallel on survival of *Puccinia* spp. on grasses, it was concluded that wheat rusts remain dormant in some collateral, unknown alternate or some unknown hosts and produces infectious spores when the environment becomes favourable (Bhardwaj et al. 2019).

1.6 Epidemiology of Wheat Rusts

Alternate hosts play a definite role in the epidemiology of wheat rusts. However, in countries where *Berberis* spp. do not occur or alternate hosts are non-functional, it is a big question that, how the rusts recur every year? In addition to India, Europe, Australia, New Zealand, South Africa, Ecuador, Pakistan, Nepal, Bangladesh and Bhutan fall in this category. In most of these countries, wheat harvest is followed by harsh summer and in many rainy seasons also. There is no wheat crop in the off-season in many of these nations. Planting of wheat disease monitoring nurseries in Northern India also showed that wheat cannot be the host in off-season. In all the places wheat crop did not survive the rainy season except the one at Shimla. Among the rusts, only leaf rust appears at Shimla every year on disease trap nursery. In India, wheat growing in off-season is limited to wheat rust research centres in Wellington (Tamil Nadu), Dalang Maidan (Himachal Pradesh), very limited area in Kinnaur district of Himachal Pradesh and Ladakh area which do not play much role in the epidemiology of wheat rusts elsewhere in India. The pathotypes occurring in these areas are generally primitive and these do not occur in adjoining plains. In addition, wheat rusts appear in plains first whereas late in the hills. It is also noteworthy that new pathotypes have been observed initially in plains and subsequently 4–5 years later in the hills. Moreover, there is a lag period between harvest in hills and young wheat crop in the plains of India. Thus, the recurrence of wheat rusts every year in India or other countries is an area of future investigations (Bhardwaj et al. 2019).

1.7 Ug99 and BGRI

Wheat stem rust had become a disease of past for about 60 years. Most of the wheat varieties in the initial stages developed at CIMMYT, Mexico and different wheat-growing countries were bred for stem rust resistance. Later on, 1B.1R translocation (*Lr26/Sr31/Yr9*) conferred resistance to all the wheat rusts. Consequently, stem rust resistance got ignored inadvertently and over-dependence on *Sr31* to manage wheat stem rust occurred. However, in 1998, some of the wheat lines having *Sr31* showed susceptibility to stem rust in Uganda and that led to the identification of Ug99 or the *Sr31* virulence (Pretorius et al. 2000). It was observed to be a threat to wheat cultivation in about 40% of the world's acreage (Singh et al. 2011). The pathogen is very dynamic, evolving very fast and 13 new members have been added to the Ug99 lineage of *P. graminis tritici*. The Ug99 group of variants has been reported from 12 other countries. Another variant PTKSK was detected from South Africa in 2017. This group of races has spread to Kenya, Ethiopia, Yemen, Sudan, Tanzania, Mozambique, Zimbabwe, South Africa, Egypt and Iran (CIMMYT 2019). The risk area of Ug99 type of races could be to the tune of 50 million hectares which is around 25% of the world's area under wheat (Singh et al. 2008). The Indian wheat varieties PBW343, PBW373 and many others also became susceptible to Ug99 type of races and were withdrawn from the cultivation. Over the years, these virulences have moved to other wheat-growing areas. After the report of *Sr31* virulence of *Puccinia graminis* f. sp. *tritici* during 1998 in Uganda, Ug99 was widely publicized as a major challenge to wheat production in India. The detailed analyses of epidemiology of wheat rusts in context to the occurrence of Ug99 in main wheat belt of NWPZ in India have shown that Ug99 cannot be a threat to wheat crop in this zone (Nagarajan 2012; Bhardwaj et al. 2014b) as it has not appeared in this part of the world even after 23 years. Notwithstanding that, taking proactive measures, all the Indian wheat germplasm especially Advance Varietal lines were evaluated in Kenya and Ethiopia regularly. The information thus generated was used for deploying wheat varieties in stem rust-prone areas. These areas of India have almost 100% coverage with Ug99-resistant wheat varieties. Most of the wheat varieties under cultivation in the Peninsular and Central zone have resistance to Ug99 type of virulences.

Considering the circumstances, Dr. Norman Borlaug appealed for a joint international effort to tackle the rust menace. It led to the formation of the Borlaug Global Rust Initiative (BGRI) on September 9, 2005, at Nairobi, Kenya, with the aims to track the movement of Ug99 virulence, evaluate released cultivars and other lines for resistance against Ug99, share the generated information worldwide, and incorporate the seedling and adult plant resistance into acceptable agronomic background. Under the BGRI guidelines, the evolution and migration route of Ug99 group races were monitored so that an early warning system could provide alarms to all stakeholders in case of an epidemic. India has been actively involved in germplasm evaluation against Ug99 group races in Kenya and Ethiopia. As preparedness for combating the likely threat of Ug99, about 250,000 wheat genotypes which included advanced breeding lines from African and Asian countries were tested for resistance to stem rust Ug99 group races at Njoro, Kenya, and Kulumsa and Debre Zeit, Ethiopia

during 2005–2012 (Bhardwaj et al. 2014b). Institutions like BGRI, CIMMYT, Kenya Agricultural and Livestock Research Organisation, and Ethiopian Institute of Agricultural Research have imparted training to many researchers of the world on the identification of Ug99-resistant wheat germplasm. Fortunately, gene *Sr31* is still effective against all races of stem rust pathogen occurring in India and adjacent countries. The use of *Lr26/Sr31/Yr9* type of resistance has now come down to 18% from the 40% earlier. Moreover, we are proactive in keeping a vigil over and tracking stem rust pathogen spreads, survey and surveillance, and varietal deployment. Therefore, India has an effective preparedness to tackle such kinds of threats.

1.8 Deoxyribonucleic acid (DNA) Polymorphism and Rust Genome Sequencing

Molecular analysis was performed on Indian pathotypes of *Puccinia triticina* by using SSR markers. High genetic diversity among the pathotypes was observed, and these could be grouped into seven major clusters. These findings offer valuable information for framing suitable disease management strategies through appropriate region-specific gene deployment and improve the understanding of the population biology and evolution of brown rust fungus in the Indian subcontinent (Prasad et al. 2017; Prasad et al. 2018a, b). These investigations also suggest that Indian population of wheat rust pathogens is different from that of other continents.

The genome of *P. triticina* (wheat leaf rust fungus) was decoded to understand the molecular basis of variability in rust fungi. Based on this study, a highly variable *Puccinia triticina* Race77 and its 13 biotypes were compared with a stable Race106. Race106 has not mutated since its first detection in 1930 and preserved at ICAR-IIWBR, Shimla. However, Race77 which was first detected in 1954 from Pusa (Bihar) has evolved into 13 pathotypes (races) and affects wheat production in the country. Therefore, next-generation sequencing technology was used to decode the genomes of these races to understand the molecular basis for fast evolution, virulence and adaptability within Race77, and stability of Race106 genome. A high-quality draft genome sequence (~100 Mb) of Race77 group exhibited 27,678 protein-coding genes responsible for various functions. Genome-wide comparative analysis revealed that 37.49% and 39.99% genomes are repetitive in case of Race77 and Race106, respectively. Race77 is significantly distinct from Race106 at repeat elements, segmental duplication and SNP(Single Nucleotide Polymorphism)/InDel (Insertion or Deletion) levels. Some “hot spot regions” in the genomes of Race77 were located. These were responsible for fast evolution and variability in Race77. This study provided an insight into the genome structure and molecular basis of variation and pathogenicity of *P. triticina*. This information would be an important landmark research and would facilitate wheat improvement programme in India (Kiran et al. 2016). Likewise, the genome of three races (K, 31 and 46S119) of *P. striiformis* f. sp. *tritici* (wheat yellow rust fungus) (Kiran et al. 2017) and four *P. graminis tritici* (stem rust) races were also decoded (Kiran et al. 2021).

1.9 Wheat Rust Interaction

Wheat and rusts interact on the basis of gene for gene hypothesis and strictly follow the rules of Mendelian inheritance. Generally, wheat resistance to rust diseases is controlled by dominant genes and virulence in wheat rust pathogens by recessive genes. It implies that avirulence is controlled by dominant genes. Wheat rust interaction manifests on the host surface in the form of definite phenotype. The expression of phenotypes (infection types) may be influenced by intensity of light and temperature range. Resistance (incompatible reaction) is the result of interaction between a resistant host and an avirulent pathogen. Several gene interactions do occur between wheat and rust pathogens (Bhardwaj 2013).

Under host–pathogen interaction, an effort was made to compare the expression patterns of defense-responsive genes at early stages of infection in susceptible and resistant lines which differ in *Lr24*-based resistance. Gene *Lr24* confers resistance to all pathotypes of *Puccinia triticina* in India. The expression levels of aquaporin, endochitinase, β -1,3-glucanase, phenylalanine ammonia-lyase, Type 1 non-specific lipid transfer protein precursor and caffeic acid O-methyltransferase were significantly higher in resistant response. In the incompatible response, comparatively high expression of aquaporin and phenylalanine ammonia-lyase was observed at the pre-historical stage, whereas expression of lipid transfer protein and endochitinase was high during the post-historical stage. This study could infer that differential expression of defense-responsive genes plays a pivotal role in compatibility or incompatibility of wheat to leaf rust. This understanding would facilitate in devising novel strategies to manage wheat leaf rust (Prasad et al. 2018b). The function of salicylic acid and sugar-mediated resistance mechanisms was also studied during the early stages of infection of wheat by leaf rust pathogen. The expression of the main salicylic acid (SA) regulators (TaEDS1, TaSGT1, TaPAD4, TaNDR1, TaRAR1, TaHSP90, TaPAL, TaEDS5 and TaNPR1) and sugar (TaHTP, TaSTP13A) pathways were examined between two wheat NILs with *Lr24* and without *Lr24*. The expression profiles of candidate genes using reverse transcription quantitative real-time polymerase chain reaction (PCR) at different time points post-inoculation revealed stage-specific transcriptional reprogramming of these genes between susceptible NIL and resistant ones. The genes acting upstream of SA in the SA pathway (TaEDS1, TaPAD4, TaRAR1, TaSGT1, TaNDR1, TaHSP90, TaEDS5) showed strong expressions after 48 h post-inoculation (hpi) in the susceptible NIL compared to unchanged or slightly changed expressions in the resistant response. Contrarily, the genes involved in SA downstream signalling (TaNPR1), SA biosynthesis (TaPAL) and sugar transportation (TaHTP, TaSTP13A) expressed highly at mid-phase of infection between 6 and 24 hpi in the resistance compared to the susceptible NIL. These patterns of gene expressions suggest that TaNPR1 and TaPAL play a positive regulatory role in the SA-mediated resistance pathway, whereas TaHTP (*Lr67*) plays an important role in the sugar-mediated resistance pathway induced by the leaf rust resistance gene, *Lr24* (Savadi et al. 2018).

During host–pathogen interaction, small ribonucleic acid (sRNAs) generated by host and pathogens could impart resistance or susceptibility due to RNA interference

(RNAi) mechanism. In one study, sRNA libraries were sequenced using Illumina sequencing technology. A total of ~1–1.28 million potential sRNAs and two micro-RNA-like small RNA (mi-RNAs) candidates were identified. Expression analysis of 20 selected sRNAs, targeting host genes pertaining to metabolic processes, transporter, reactive oxygen species (ROS)-related disease resistance, apoptotic inhibitor and transcription factors along with two pt-mi-RNAs by qRT-PCR, showed distinct patterns of expression of the sRNAs in urediniospore-specific libraries. A pioneering work has been done in identifying the novel sRNAs in *P. triticina* including two pt-mi-RNAs that may play a pivotal role in biotrophic growth and pathogenicity (Dubey et al. 2019).

1.10 Rust Resistance in Wheat

Resistant wheat cultivars have been a classic mechanism to manage rust diseases. A few historic cultivars, Hope and Thatcher for stem rust, Surpreza, Americano 25, Frontana and Fronteira for leaf rust, and Manella, Juliana, Wilhelmina, Capelle-Desprez, and Carstens VI for stripe rust, carried considerable level of resistance for many years. Several cultivars have remained resistant for 5 years or even more, that is, an average agronomic lifespan of a cultivar. Sometimes, resistance may last longer. However, some cultivars had become susceptible even before those were grown on a fraction of the cultivated acreage. In most cases, cultivars have become susceptible because of inadequate knowledge of the virulences existing in a pathogen population. In other cases, mutations or somatic recombination among existing virulences have occurred and rendered the host susceptible.

Rust resistance genes are categorized into two classes. The first class is R (for resistance) genes, which are race-specific in their action and effective at all growth stages of wheat. The second class is adult plant resistance (APR) genes. These genes usually express at the third leaf stage. APR can be non-race-specific (partial) or race-specific (both of hypersensitive and non-hypersensitive type). The levels of resistance conferred by single APR genes are only partial and allows substantial rust development (Kilpatrick 1975; Ellis et al. 2014). The landmark success of rust breeding programme had been against stem rust. This is because of an exhaustive research on this pathogen for a larger number of years. A number of cultivars carrying *Sr26* have been released in Australia since 1971 and were grown on a large area without being affected by stem rust (Luig and Rajaram 1972). The adult plant resistance gene *Sr2*, derived from Hope, showed the absence of uredia on the internode tissues (Hare and McIntosh 1979). This has perhaps been the most commonly used *Sr* gene globally since the 1940s. Gene *Sr31*, imparted a high to moderate effective resistance to stem rust, was effective worldwide until 1998. Presently, it is common in many high-yielding wheat cultivars, including Burgus II, Lovrin 10, Aurora, Kavkaz, Riebesel, Siouxland, Alondra, Weique, Bartweizen, Nautica, Salzmuendu Clement, Pak 81, Faisalabad 85, Veery, Bobwhite and many thousands of germplasm lines or cultivated wheat lines.

Although 80 *Lr*, 59 *Sr* and 83 *Yr* genes have been designated worldwide (McIntosh Pers. comm.), however, virulence occurs for a majority of them. Therefore, the best control strategy would involve combinations of these race-specific and non-race-specific genes. Many spring wheat cultivars developed by CIMMYT have shown a slow rusting type of resistance (Rajaram et al. 1996). Gene *Lr34* along with the other undesignated slow rusting genes is believed to impart a durable resistance in Frontana and other wheat cultivars (Singh and Rajaram 1992). Gene *Lr46* in combination with an unknown slow rusting gene is onus for slow rusting resistance in cv. Pavon 76 (Singh et al. 1998). The usefulness or durability of resistance is not associated with the donor genera or species.

1.11 Evaluation of Germplasm for Rust Resistance

A foolproof screening system of germplasm assessment is necessary for identifying rust resistance sources. It is ensured that uniform rust epiphytotic is created by providing optimum conditions for infection of rusts. All the wheat germplasm developed under the umbrella of All India coordinated Wheat and Barley Improvement Project, and other materials are subjected to rigorous screening at seedling (all time), race-specific APR at IIWBR, Regional Station, Flowerdale, Shimla, H.P., and also at many coordinated centres for APR to different rusts. The screening involves challenging each wheat line against the most virulent and predominant pathotypes with different avirulence/virulence structures. Studies involve screening of wheat material at seedling stage against 70 different pathotypes of three rust pathogens under controlled conditions of temperature and light. At the same time, race-specific adult plant evaluation is undertaken at Shimla and other centres also. Similarly, non-race-specific screening of Advance Wheat Trial material is also carried out at many centres to calculate area under disease progress curve. At the same time, genetics of rust resistance is also investigated. Based on the gene matching technique, rust resistance genes are characterized in wheat material. All these aspects enable the selection of lines with diverse rust resistance. This information is used for the identification of new varieties and varietal deployment.

1.12 Gene Mining and Gene Discovery for Rust Resistance in Wheat

Efforts are on world over to search for novel and unexplored rust resistance genes available in old wheat varieties, present-day wheat material, local germplasm, alien sources and exotic accessions. Such genes have been identified to confer leaf or and stripe rust resistance in CIMMYT, Mexico, Australia and many other countries. In fact, the gene inventory was enriched more in the last 20 years than ever before. Recently, in India, a major rust resistance gene *Lr80* was identified from local wheat landrace, Hango-2 and mapped on 2DS chromosome (Fig. 1.2).

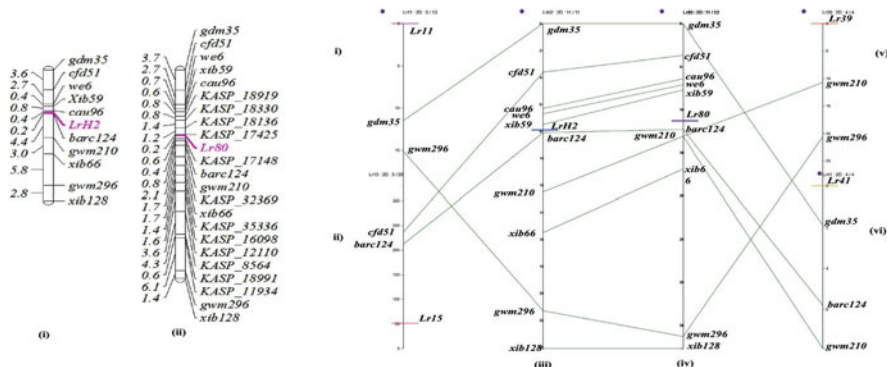


Fig. 1.2 Genetic linkage maps of Agra Local/Hango-2 population (1) *LrH2* present study SSR map, (2) *Lr80* SSR and SNP map

This gene confers resistance to all the predominant and virulent pathotypes of *P. tritricina* in India (Kumar et al. 2021). This landrace was collected from Hango, District Kinnaur, Himachal Pradesh, India. Hango-2 exhibits a low infection type (IT) to all Indian *P. tritricina* pathotypes, except the pathotype 5R9–7 which shows IT 3+. Pathotype 5R9–7(16–1) does not infect bread wheat cultivars and, however, is virulent on tetraploid wheat Khapli. Pathotype 5R9–7 is not prevalent in nature for the last 15 years. Genetic analysis based on Agra Local/Hango-2-derived F_3 families indicated monogenic control of leaf rust resistance, and the underlying locus was temporarily named *LrH2*. Bulked segregant analysis using 303 simple sequence repeat (SSR) markers located *LrH2* in the short arm of chromosome 2D and was formally designated as *Lr80*. Gene *Lr80* is flanked by markers *cau96* (distally) and *barc124* (proximally). The 90 K Infinium SNP array was used to identify SNP markers linked with *Lr80*. Markers *KASP_17425* and *KASP_17148* showed association with *Lr80* (Kumar et al. 2021). This gene is being used to develop rust-resistant genetic stocks and wheat varieties. It would help in creating diversity of rust resistance and management of leaf rust in India.

1.13 Anticipatory Wheat Breeding and Pyramiding of Resistance

With the discovery of Biffen (1905) that resistance to stripe rust in wheat is inherited in Mendelian way, it paved a way for rust resistance breeding. Subsequently, new wheat varieties were bred with an objective to manage rusts. Systematic breeding for rust resistance in wheat began in the early 1950s in India. Much has been achieved through the years in managing the rusts by planting resistant cultivars conferring diverse resistance. The genetic diversity among the cultivars has proved an effective strategy in managing the rust diseases, reducing the quantum and frequency of rust epidemics and understanding the rust epidemiology. The resistance gene *Lr26*

together with *Lr13*, *Lr23* and *Lr34* and *Lr24/Sr24* segment from *Agropyron* have played a pivotal role in imparting field resistance and enhancing wheat production.

Pre-emptive or anticipatory breeding is a breeding for resistance to future pathotypes, in terms of development of cultivars resistant to future threats. It requires a relevant knowledge of the pathogenicity of pathotypes and host resistance genes deployed over wheat-growing areas. Resistance breeding strategies are usually supported with the maintenance of genetic diversity to provide buffering against extreme crop losses in case of epidemics. It basically refers to devising strategies for tackling the future pathotypes. It is assumed that new pathotypes will evolve from the prevalent pathotypes through mutation with respect to single-host resistance genes. It is also anticipated that future pathotypes would have the ability to overcome one or more of the resistance genes, which are deployed in recently grown wheat cultivars. Under Indian conditions, mutation occurring in the rust pathogens is the prime cause of variability. Three factors, namely, genetic diversity, durability and effectiveness are considered the most important in pre-emptive breeding. Host genetic diversity is encouraged to provide a bulwark in case of a rust outbreak. Information on genetic diversity and recommended cultivars is made available to extension personnel and farmers to assist in decision-making processes and to issue an advisory.

Another important aspect which cannot be left behind in case of pre-emptive resistance breeding is the frequent pathotypic surveys in farmers' fields, hotspots and disease trap nurseries. A vigilant and relevant pathogenicity survey monitors variability in pathogen populations. If resistances are based on genes effective only at the adult plant stages, the adult plant resistance (APR) tests should be conducted every season at hot spots for the disease. Many surveys monitor an arbitrary array of seedling resistance factors, and the information usually is of limited use to wheat breeders. Our national-level surveys jointly done by ICAR institutes and State Agricultural Universities have two major objectives. First, pathogenicity genes are used as markers in monitoring the origins, movement and epidemiology of rust pathogen populations and second, they monitor the disease responses conferred by the available resistance genes actually deployed in the region. With relevant information on pathogenicity from surveys and genetic information from host studies, it is feasible to opt for a limited number of pathotypes for screening the selection nurseries. Genetic studies are most suitably conducted with single pathotype in order to avoid the complexities that occur when resistance to a pathotype mixture is due to gene combinations (McIntosh 1992). Another point to be considered for pre-emptive breeding efforts calls for detailed knowledge about pathogenic variation and factors responsible for it. Migration has been reported as a major factor for variability in pathogen population structure. There is a strong proof that anthropogenic activities also contribute to the dispersal of wheat yellow rust. For instance, European travellers first introduced yellow rust pathogen into Australia in 1979 (Wellings 2007). The introduction of inoculum followed by successful establishment of introduced pathotypes from outside the area has become a frequent problem. Earlier, only wind-borne entry was suspected (Nagarajan and Joshi 1985; Watson and de Sousa 1982). Once introduced, further introductions of identical or

similar pathotypes cannot be recognized. Pathogens that become established in one region having favourable conditions for its growth rapidly appear and try to establish in other regions adjusting to the changing requirements for its survival. Evolutionary pathways can be successfully constructed to show the occurrence of mutational changes over time with the help of bio-informatics.

The role of mutation and recombination in the evolution of pathogens to create variability cannot be ignored. *Puccinia striiformis* populations in some parts of the globe are highly clonal, especially in India, Australia and Europe. Since *Berberis* species do not play any role in completion of sexual life cycle and perpetuation of the stripe rust pathogen in India (Mehta 1940), probably, mutation and adaptation to popular regional cultivars are the primary mechanisms of evolution and selection of new pathotypes (Gangwar et al. 2016). Pathotype 46S119 evolved independently from the known pathotype 46S103 by gaining virulence to *Yr9* (Prashar et al. 2007). It appears that a genetic change occurred recently in pathotype 46S119 resulting in additional virulence to differentials Suwon92xOmar and Riebesel 47/51, and pathotypes 110S119 and 238S119 got evolved. Similarly, pathotype 78S84 gained additional virulence to cv. Strubes Dickkopf and gave rise to pathotype 110S84 (Gangwar et al. 2019). Thus, selection of pathotypes facilitated by the host genotypes with relevant resistance factors is a major driving force through which mutant newly or introduced pathotypes increase in frequency.

1.14 Rust Management Strategies

The genetic resistance is the most economic, ecologically safe and effective way to manage wheat rusts. Use of effective bio-pesticides or biological agents, chemicals particularly of triazole group, interventions in cultural practices, continual monitoring of rust diseases and deployment of rust-resistant varieties are the popular strategies for wheat rusts' management. A novel strain of *Alternaria alternata* was able to penetrate and colonize the urediniospores of yellow rust pathogen (Zheng et al. 2017). This hyperparasite seems to be less promising as biological control agent, because of rapid production of rust spores and their aerial dispersion. However, *Bacillus subtilis* strain E1R-j was reported effective in wheat stripe rust management (Li et al. 2013). Other biological control agents such as *Trichoderma* spp., *Bacillus* spp., *Streptomyces* spp. and arbuscular mycorrhizal fungi can also be used for managing wheat rusts (El-Sharkawy et al. 2018; Huang and Pang 2017; El-Sharkawy et al. 2015). Application of a fungicide is generally recommended when the cultivar is susceptible, and the disease has appeared at the early stage. For maximizing the cost–benefit ratio, fungicides must be applied between flag leaf formation and complete head emergence. Post-flowering application of fungicide would not economically feasible because a considerable damage to the flag leaf will have occurred by that time. Protecting the flag leaf is of utmost importance. Fungicides such as propiconazole 25% EC (Tilt), difenoconazole 25% EC (Score), azoxystrobin 25% SC (Amistar) and tebuconazole 25% EC (Folicur) could be used effectively (@ 0.1%) to manage wheat rust diseases. Fungicides tebuconazole

(Folicur 250 EC), tebuconazole + tridimenol (Silvacur 375 EC), tebuconazole (Orion 25 EW) and AmistarXtra 280 SC, individually or in combination, were highly effective in avoiding black rust epidemics in Kenya (Wanyera et al. 2009). Despite all the benefits associated with the application of fungicides, there is always risk of development of fungicide resistance in rust fungi, which results in reduction or loss of fungicide sensitivity in these pathogens (Arduim et al. 2012).

There is a need to devise more harmonized ways of managing the threat of plant diseases such as wheat rusts by integrating different plant breeding, chemistry, biotechnology and bioinformatic tools for a practical and sustainable crop protection (Lucas et al. 2015). Alternative novel approaches using biotechnological tools such as genomic selection, cis-genesis, intra-genesis, sequence-specific nuclease technology, oligonucleotide-directed mutagenesis, gene cassettes, RNA-dependent DNA methylation, pathogen effectors guided breeding and reverse breeding are being developed and employed for breeding broad-spectrum and durable disease resistance in crops (Prasad et al. 2019; Savadi et al. 2017).

1.15 Conclusion

Diversity is the keyword for wheat rust management. Much has been achieved in managing the wheat rusts through deploying resistant wheat varieties having diverse resistance in India. A blend of all time (seedling), slow rusting, adult plant rust resistance genes of both race-specific and non-race-specific types are considered for the deployment of wheat varieties. It would not only avoid the severe losses due to wheat rusts but also increase the self-life of wheat cultivars and discourage evolution in rust pathogens. Skillful deployment of wheat cultivars based on the knowledge of pathotypes distribution has led to the successful management of wheat rusts in India. In recent years, wheat has shown relatively higher production stability as compared to other cereal crops through strategic gene deployment (Tomar et al. 2014). A combination of cultural management practices, deployment of rust-resistant genotypes and fungicide application (in case of emergent situation) would be the most effective module for managing the wheat rust diseases.

References

- Aggarwal R, Sharma S, Gupta G, Manjunatha C, Singh VK, Kulshreshtha D (2017) Gene based analysis of *Puccinia* species and development of PCR based marker for the detection of *Puccinia striiformis* f. sp. *tritici* causing yellow rust of wheat. *J Gen Plant Pathol* 83:205–215
- Anonymous (1982) Annual report. Plant Breeding Institute, Univ. of Sydney, Sydney
- Arduim GDS, Reis EM, Barcellos AL, Turra C (2012) In vivo sensitivity reduction of *Puccinia triticina* races, causal agent of wheat leaf rust, to DMI and QoI fungicides. *Summa Phytopathol* 38:306–311
- Ashourloo D, Mobasheri MR, Huete A (2014) Evaluating the effect of different wheat rust disease symptoms on vegetation indices using hyperspectral measurements. *Remote Sens* 6:5107–5123
- Bhardwaj SC (2013) *Puccinia-Triticum* interaction: an update. *Indian Phytopathol* 66(1):14–19

- Bhardwaj SC, Gupta N, Sharma TR, Pal D, Prasad P (2014a) Competitive ability and fitness potential among the pathotypes of *Puccinia triticina* on wheat in India. *Indian Phytopathol* 67 (1):33–37
- Bhardwaj SC, Prashar M, Prasad P (2014b) Ug99-Future Challenges. In: Goyal A, Manoharachary C (eds) Future challenges in crop protection against fungal pathogens, fungal biology. Springer, New York, pp 231–248
- Bhardwaj SC, Singh GP, Gangwar OP, Prasad P, Kumar S (2019) Status of wheat rust research and progress in rust management in Indian context. *Agronomy* 9:892
- Biffen RH (1905) Mendel's law of inheritance and wheat breeding. *J Agric Sci* 1:4–48
- CIMMYT (2019) https://rusttracker.cimmyt.org/?page_id=22
- Craigie JH (1927) Discovery of the function of the pycnia of the rust fungi. *Letter Editor Nat* 120: 765–767
- de Oliveira B, Samborski DJ (1966) Aecial stage of *Puccinia reconidita* on Ranunculaceae and Boraginaceae in Portugal. *Proc Cereal Rusts Conf* 1964:133–150
- Devadas R, Lamb DW, Simpfendorfer S, Backhouse D (2009) Evaluating ten spectral vegetation indices for identifying rust infection in individual wheat leaves. *Precis Agric* 10:459–470
- Dubey H, Jaswal R, Kiran K, Bhardwaj SC, Sharma TR (2019) Discovery and profiling of small RNAs from *Puccinia triticina* by deep sequencing and identification of their potential targets in wheat. *Funct Integr Genomics* 19(3):391–407
- Ellis JG, Lagudah ES, Spielmeyer W, Dodds PN (2014) The past, present and future of breeding rust resistant wheat. *Front Plant Sci* 5:641
- El-Sharkawy HHA, Rashad YM, Ibrahim SA (2018) Biocontrol of stem rust disease of wheat using arbuscular mycorrhizal fungi and *Trichoderma* spp. *Physiol Mol Plant Pathol* 103:84–91
- El-Sharkawy HHA, Tohamy S, Khalil AA (2015) Combined effects of *Streptomyces viridosporus* and *Trichoderma harzianum* on controlling wheat leaf rust caused by *Puccinia triticina*. *Plant Pathol J* 14:182–188
- Flor HH (1956) The complementary genic systems in flax and flax rust. *Adv Genet* 8:29–35
- Gangwar OP, Kumar S, Bhardwaj SC, Kashyap PL, Prasad P, Khan H (2019) Characterization of three new *Yr9* virulences and identification of sources of resistance among recently developed bread wheat germplasm. *J Plant Pathol* 101(4):955–963
- Gangwar OP, Kumar S, Bhardwaj SC, Prasad P, Khan H, Savadi S, Sharma SK (2017) Detection of new *Yr1*-virulences in *Puccinia striiformis* f. sp. *tritici* population and its sources of resistance in advance wheat lines and released cultivars. *Indian Phytopathol* 70(3):307–314
- Gangwar OP, Kumar S, Prasad P, Bhardwaj SC, Khan H, Verma H (2016) Virulence pattern and emergence of new pathotypes in *Puccinia striiformis tritici* during 2011–15 in India. *Indian Phytopathol* 69(4s):178–185
- Gupta N, Bhardwaj SC, Sharma TR, Prasad P, Gangwar OP, Kumar S (2018) Population behaviour of predominant and virulent pathotypes of *Puccinia triticina* causing wheat brown rust in India. *Indian Phytopathol* 71(1):59–64
- Hare RA, McIntosh RA (1979) Genetic and cytogenetic studies of the durable adult plant resistance in 'Hope' and related cultivars to wheat rusts. *Z Pflanzenzüchtg* 83:350–367
- Huang S, Pang F (2017) Biocontrol agents for controlling wheat rust. *Methods Mol Biol* 1659:277–288
- Huerta-Espino J, Singh RP, German S, McCallum BD, Park RF, Chen WQ, Bhardwaj SC, Goyeau H (2011) Global status of wheat leaf rust caused by *Puccinia triticina*. *Euphytica* 179:143–160
- Jackson HS, Mains EB (1921) Aecial stage of the orange leaf rust of wheat, *Puccinia triticina* Erikss. *J Agric Res* 22:151–172
- Jin Y, Szabo LJ, Carson M (2010) Century-old mystery of *Puccinia striiformis* life history solved with the identification of *Berberis* an alternate host. *Phytopathology* 100:432–435
- Johnson R, Stubbs RW, Fuchs E, Chamberlain NH (1972) Nomenclature for physiologic races of *Puccinia striiformis* infecting wheat. *Trans Br Mycol Soc* 58:475–480
- Johnson T (1961) Man guided evolution in plant rusts. *Science* 133:357–362

- Johnston CO, Browder LE (1966) Seventh revision of the international register of physiologic races of *Puccinia recondita* f. sp. *tritici*. Plant Dis Rptr 50(10):756–760
- Kilpatrick RA (1975) New wheat cultivars and longevity of rust resistance 1971–1975. USDA, Agric Res Serv Northeast. ARS-NE 64 20
- Kiran K, Rawal H, Dubey H, Jaswal R, Bhardwaj S, Prasad P, Pal D, Devanna BN, Sharma T (2017) Dissection of genomic features and variations of three pathotypes of *Puccinia striiformis* through whole genome sequencing. Sci Rep 7:1–16
- Kiran K, Rawal HC, Dubey H, Jaswal R, Devanna BN, Gupta DK, Bhardwaj SC, Prasad P, Pal D, Chhuneja P, Balasubramanian P, Kumar J, Swami M, Solanke AU, Gaikwad K, Singh NK, Sharma TR (2016) Draft genome of the wheat rust pathogen (*Puccinia triticina*) unravels genome-wide structural variations during evolution. Genome Biol Evol 8(9):2702–2721
- Kiran K, Rawal HC, Dubey H, Jaswal R, Bhardwaj SC, Deshmukh R, Sharma TR (2021) Genome-wide analysis of four pathotypes of wheat rust pathogen (*Puccinia graminis*) reveals structural variations and diversifying selection. J Fungi 7(9):701. <https://doi.org/10.3390/jof7090701>
- Kislev ME (1982) Stem rust of wheat 3300 years old found in Israel. Science 216:993–994
- Knott DR (1989) The wheat rusts-breeding for resistance. Springer, Berlin Heidelberg, p 201
- Kumar S, Bhardwaj SC, Gangwar OP, Sharma A, Naeela Q, Kumaran VK, Khan H, Prasad P, Miah H, Singh GP, Sharma K, Verma H, Forrest KL, Trethowan RM, Bariana HS, Bansal UK (2021) *Lr80*: a new and widely effective source of leaf rust resistance of wheat for enhancing diversity of resistance among modern cultivars. Theor Appl Genet 134:849–858
- Li H, Zhao J, Feng H, Huang L, Kang Z (2013) Biological control of wheat stripe rust by an endophytic *Bacillus subtilis* strain E1R-j in greenhouse and field trials. Crop Prot 43:201–206
- Lihua C, Shichang X, Ruiming L, Taiguo L, Wanquan C (2008) Early molecular diagnosis and detection of *Puccinia striiformis* f. sp. *tritici* in China. Lett Appl Microbiol 46:501–506
- Liu TG, Wang X, Gao L, Liu B, Chen WQ, Xiang WS (2014) A FIASCO based approach for detection and diagnosis of *Puccinia graminis* f. sp. *tritici* in China. J Integr Agric 13:2438–2444
- Lucas JA, Hawkins NJ, Fraaije BA (2015) The evolution of fungicide resistance. Adv Appl Microbiol 90:29–92
- Luig NH, Rajaram S (1972) The effect of temperature and genetic background on host gene expression and interaction to *Puccinia graminis tritici*. Phytopathology 62:1171–1174
- McIntosh RA (1992) Pre-emptive breeding to control wheat rusts. Euphytica 63:103–113
- Mehta KC (1940) Further studies on cereal rusts in India. Imper Council Agric Res 14:224
- Nagarajan S (2012) Is *Puccinia graminis* f. sp. *tritici* – virulence Ug99 a threat to wheat production in the North West Plain Zone of India? Indian Phytopathol 65(3):219–226
- Nagarajan S, Joshi LM (1985) Epidemiology in the Indian subcontinent. In: Roelfs AP, Bushnell WR (eds) The cereal rusts, diseases, distribution, epidemiology and control. Academic Press, Orlando, pp 371–402
- Nagarajan S, Saari EE (1995) Repeat performance of yellow rust spread from Turkey to Indian sub-continent. Indian J Mycol Pl Pathol 25(1–2):68
- Prasad P, Bhardwaj SC, Gangwar OP, Kumar S, Khan H, Kumar S, Rawal HC, Sharma TR (2017) Population differentiation of wheat leaf rust fungus *Puccinia triticina* in South Asia. Curr Sci 112(10):2073–2083
- Prasad P, Bhardwaj SC, Savadi S, Kashyap PL, Gangwar OP, Khan H, Singh SB, Kumar S (2018a) Population distribution and differentiation of *Puccinia graminis tritici* detected in the Indian subcontinent during 2009–2015. Crop Prot 108:128–136
- Prasad P, Savadi S, Bhardwaj SC, Gangwar OP, Kumar S (2019) Rust pathogen effectors: perspectives in resistance breeding. Planta 250:1–22
- Prasad P, Savadi S, Bhardwaj SC, Kashyap PL, Khan H, Kumar S, Kumar R, Virupaksh P (2018b) Stage-specific reprogramming of defense responsive genes during *Lr24*-mediated leaf rust resistance in wheat. J Plant Pathol 101(2):283–293
- Prashar M, Bhardwaj SC, Jain SK, Datta D (2007) Pathotypic evolution in *Puccinia striiformis* in India during 1995–2004. Aust J Agric Res 58:602–604

- Pretorius ZA, Singh RP, Wagoire WW, Payne TS (2000) Detection of virulence to wheat stem rust resistance gene *Sr31* in *Puccinia graminis* f. sp. *tritici* in Uganda. *Pl Dis* 84(2):203
- Rajaram S, Singh RP, van Ginkel M (1996) Approaches to breed wheat for wide adaptation, rust resistance and drought. In: Richards RA, Wrigley CW, Rawson HM, Rebetzke GJ, Davidson JL, Brettell RIS (eds) Proc. 8th assembly wheat breeding Society of Australia. The Australian National University, Canberra, pp 2–30
- Roelfs AP, Singh RP, Saari EE (1992) Rust diseases of wheat: concepts and methods of disease management. CIMMYT, Mexico, D.F., 81 pp
- Savadi S, Prasad P, Bhardwaj SC, Kashyap PL, Gangwar OP, Khan H, Kumar S (2018) Temporal transcriptional changes in SAR and sugar transport-related genes during wheat and leaf rust pathogen interactions. *J Plant Growth Regul* 37:826–839
- Savadi S, Prasad P, Kashyap PL, Bhardwaj SC (2017) Molecular breeding technologies and strategies for rust resistance in wheat (*Triticum aestivum*) for sustained food security. *Plant Pathol* 67:771–791
- Singh RP, Hodson DP, Huerta-Espino J, Jin Y, Bhavani S, Njau P, Herrera-Foessel SA, Singh PK, Singh S, Govindan V (2011) The emergence of Ug99 races of the stem rust fungus is a threat to world wheat production. *Annu Rev Phytopathol* 49:465–481
- Singh RP, Hodson DP, Huerta-Espino J, Jin Y, Njau P (2008) Will stem rust destroy the world's wheat crop? *Adv Agron* 98:271–309
- Singh RP, Mujeeb-Kazi A, Huerta-Espino J (1998) *Lr46*: a gene conferring slow-rusting resistance to leaf rust in wheat. *Phytopathology* 88:890–894
- Singh RP, Rajaram S (1992) Genetics of adult-plant resistance to leaf rust in 'Frontana' and three CIMMYT wheats. *Genome* 35:24–31
- Singh RP, Srivastava P, Sharma A, Bains NS (2017) Bread wheat cultivar PBW 343 carries residual additive resistance against virulent stripe rust pathotype. *J Crop Improv* 31(2):183–191
- Stakman EC, Harrar JG (1957) *Principals of plant pathology*. Ronald Press, New York, p 581
- Stakman EC, Stewart DM, Loegering WQ (1962) Identification of physiological races of *Puccinia graminis* var. *tritici* USDA, ARS E617 53
- Tomar SMS, Singh SK, Sivasamy M, Vinod (2014) Wheat rusts in India: resistance breeding and gene deployment-a review. *Indian J Genet* 74:129–156
- Wanyera R, Macharia JK, Kilonzo SM, Kamundia JW (2009) Foliar fungicides to control wheat stem rust, race TTKS (Ug99), in Kenya. *Plant Dis* 93:929–932
- Watson IA, de Sousa CNA (1982) Long distance transport of spores of *Puccinia graminis tritici* in the southern hemisphere. *Proc Linnean Soc NSW* 106:311–321
- Wellings CR (2007) *Puccinia striiformis* in Australia: a review of the incursion, evolution, and adaptation of stripe rust in the period 1979–2006. *Aust J Agric Res* 58(6):567–575
- Xu P, Wu G, Guo Y, Chen X, Yang H, Zhang R (2017) Automatic wheat leaf rust detection and grading diagnosis via embedded image processing system. *Proc Comput Sci* 107:836–841
- Zhao J, Wang L, Wang ZY, Chen XM, Zhang HC, Yao JN, Zhan GM, Chen W, Huang LL, Kang ZS (2013) Identification of eighteen *Berberis* species as alternate hosts of *Puccinia striiformis* f. sp. *tritici* and virulence variation in the pathogen isolates from natural infection of barberry plants in China. *Phytopathology* 103:927–934
- Zheng I, Zhao J, Liang X, Zhan G, Jiang S, Kang Z (2017) Identification of a novel *Alternaria alternata* strain able to hyperparasitize *Puccinia striiformis* f. sp. *tritici*, the causal agent of wheat stripe rust. *Front Microbiol* 8:71