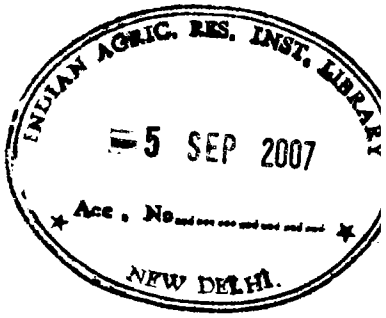
GENETIC ANALYSIS AND CHROMOSOMAL LOCATION OF RUST RESISTANCE GENES IN INTERSPECIFIC DERIVATIVES IN WHEAT (*Triticum aestivum* L.)

गेहूं के अन्तःविशिष्ट व्युत्पनों में रतुआ प्रतिरोधी जीनों का आनुवंशिक विश्लेषण और गुणसूत्रीय (क्रोमोजोमल) स्थिति

S. KUMARASAMY



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GENETIC ANALYSIS AND CHROMOSOMAL LOCATION OF RUST RESISTANCE GENES IN INTERSPECIFIC DERIVATIVES IN WHEAT (*Triticum aestivum* L.)

By

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CERTIFICATE

This is to certify that the thesis entitled "**Genetic analysis and chromosomal location of rust resistance genes in interspecific derivatives in wheat (*Triticum aestivum* L.)**", submitted to the Faculty of the Post Graduate School, Indian Agricultural Research Institute, New Delhi, in partial fulfilment of the requirements for the award of the degree of **Doctor of Philosophy in Genetics** by **S. Kumarasamy** embodies the results of *bonafide* work carried out by him under my supervision and guidance. No part of the thesis has been submitted by him for any other degree or diploma.

I further certify that any help or information received during the work on this thesis has been duly acknowledged.

Place : New Delhi

Date : 5th Aug., 2006

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Place : New Delhi
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Dedicated to

my

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INTRODUCTION

Wheat is one of the most widely cultivated crops in the world. In India, it is the second most important crop next to rice. It is mainly a *rabi* season crop in India. The advent of semi dwarf wheats and subsequently their introduction in India transformed Indian agriculture from subsistence farming to sustainable farming. Wheat has played a very vital role in stabilizing the food grain production in the country over the past few decades. Currently, our wheat grain production is around 73 million tones (ES : 2006-2007). The area under wheat cultivation is around 26 million ha in India. Today India is the second largest producer of wheat in the world.

The present level of yields in wheat has been attained largely due to breeding and release of high yielding varieties and their cultivation by the farmers under recommended agronomic practices. The yield gains noticed so far were primarily due to the introgression of genes such as *Rht's* and *Ppd's* and they were further sustained with the introduction of rye segment (IBL.IRS) into wheat. However, in recent times, it has been observed that production as well as productivity of wheat has reached to almost a plateau. Therefore, breeders have to mobililze genes from diverse sources including wild germplasm for restructuring the plant type, which responds to high inputs and stress environment.

Wheat, in India is attacked by several fungal diseases. Among which, rusts continue to pose a serious threat by inflicting heavy losses in grain production. All the three rusts of wheat i.e. black (stem) rust (*Puccinia graminis* Pers *f.sp.tritici* Ericks & Henn), yellow (stripe) rust (*Puccinia striiformis* Westend) and brown (leaf) rust (*Puccinia recondita* = *P. triticina*) are known to cause significant losses. The rusts are highly variable and each one of them is known to have many pathophysiological races or bio-types. Appearance of new pathotype(s), generally render resistant variety susceptible in a short span of time after their release. During the last thirty years concerted efforts for increasing the production and productivity have been made. Right from the beginning of wheat breeding in India, breeding for rust resistance has received major attention.

Estimates for losses in production due to rust epidemics have been made by various workers. Yield losses caused by leaf rust epidemic alone are estimated at around 40 per cent (Sawhney, 1995) and by stem and stripe rusts could be more if infection intensity is

higher. Historical account of rust epidemics in India was given by Nagarajan and Joshi (1975). During the recent past, epidemics of leaf rust and stripe rusts have caused significant losses. Isolated epidemics have occurred in some pockets of North India during 1971-1972 and a pandemic in 1972-1973. During 1993, a severe leaf rust epidemic with severity ranging between 60S-80S or more was recorded over an area of four million hectares in Uttar Pradesh, Punjab and Haryana (Nayar *et al.*, 1997). It is evident, therefore, that rusts may appear in epidemic proportions in any wheat growing areas and cause heavy yield losses.

Among different rust diseases of wheat, leaf rust is the most common in India and develops rapidly at temperatures ranging between 10 to 30^0C. It occurs in all wheat growing zones at various intensities depending on the time of appearance and environmental conditions. About 55 '*Lr*' genes providing resistance to leaf rust have been documented (McIntosh *et al.*, 2005). A large number of designated leaf rust resistance genes have their origin in *Triticum aestivum,* however, most of them have become ineffective to the prevalent Indian pathotypes (Sawhney, 1995; Tomar and Menon, 1998). The resistance gene introdgressed from wild progenitors and alien species confer a high degree of resistance to a broad spectrum of leaf rust pathotypes.

Stem rust is another important disease of worldwide concern including India. The humid condition and temperature range of 15 to 35^0C favours disease development. It is the most devastating disease in areas like North America, Australia and Africa (McIntosh *et al.*, 1995). In India, it usually appears in peninsular and central India, Nilgiri hills, rainfed area of Himachal Pradesh and Jammu and Kashmir, attacking both durum and bread wheats. Frequent spontaneous mutations lead to evolution of new virulent physiological races rendering the existing stem rust resistance genes ineffective. There are about 45 genes from divers sources, which have been identified for stem rust resistance (McIntosh *et al.*, 2005). Among which the genes, namely, *Sr24, Sr25, Sr26, Sr27, Sr28, Sr31, Sr33* and *Sr38* derived from related genera and species confer moderate to high degree of resistance to the prevalent Indian races of stem rust (Tomar and Menon, 2001).

Breeding for host-plant resistance is the most economical, suitable and sustainable method of rust control. Usually, the life span of a resistant wheat variety is short due to the co-evolution of new races of rust pathogen. The diversification of rust resistance genes in the varieties and thier strategic development in different agro ecological regions has been

instrumental in arresting the spread of rusts in major wheat growing areas of the country. Genetic diversity in crop population with cultivars differeing in resistance genes improves the durability of resistance and also imparts lower vulnerability against rusts. Gradually in the process of host-parasite interaction new virulent pathotypes evolve that renders the cultivars susceptible to rusts. The evolution of new virulent pathotype(s) necessitates the breeders to search for new sources of resistance continuously. Access to diverse genetic stocks possessing different rust resistnace gene(s) is a pre-requisite for a dynamic breeding programme. If the donor for resistance are not available in the cultivated or primitive genotypes, wild species are utilized as potential donors.

Wild progenitors of common wheat (*T. aestivum*), allied genera and related species constitute a vast reservior of potentially useful genes for disease resistance, particularly rusts (Dhaliwal *et al.,* 1986; Tomar and Kochumadhavan, 1996). Several useful genes have been transferred from these wild species to common wheat (Sharma and Gill, 1983; Sharma, 1995). The importance of secondary and tertiary gene pools of wheat to enhance the genetic potential of cultivated types of wheat had also been highlighted by Jiang *et al.* (1994) and Mujeeb-Kazi (1995). *Triticum timopheevi* (McIntosh and Gyarfas, 1971), *Aegilops speltoides* (Dvorak and Knott, 1990), *Agropyron intermedium* (Friebe *et al.,* 1992) *Aegilops ventricosa, T. persicum, T. urartu,* and *T. boeoticum* etc. are important sources of rust resistance genes (Bariana and McIntosh, 1993).

Plant breeders generally work within the primary gene pool where hybridization is easily accomplished. The production of hybrids between crop species and their wild and weedy relatives is generally referred to as wide hybridization. To accomplish susceptible genetic transfers from wild species to cultivated wheat, the cytogenetical techniques are employed for inducing homoeologous chromome farming. Both interspecific and intergeneric hybridization has been potentially used for improvement of crop plants. In such attempts, cytologically stable interspecific derivatives (2n = 42) of *T. aestivum x Aegilops speltoides, T. aestivum x T. militinae* and *T. aestivum x T. boeticum* have been selected which carry a high level of leaf rust and stem rust resistance. In addition, they possess some specific morphological markers such as pubescence of leaf and leaf sheath alongwith other desirable traits. The information on genetic behaviour of such derivatives is necessary if they are to be utilized in breeding programme. The knowledge about the mode of inheritance, identity

of genes and their location in specific chromosomes is useful and will facilitate their utilization in breeding programme.

Therefore, this study on genetic analysis and chromosomal location of rust resistance genes in interspecific derivatives in wheat was undertaken with the following objectives:

1. To study the mode of inhertiance of leaf rust and stem rust resistance.
2. Identification of gene(s) through test of allelism.
3. Linkage analysis and
4. Mapping of gene(s) on specific chromosome(s).

REVIEW OF LITERATURE

Bread or common wheat (*Triticum aestivum* L.) is cultivated over an area of 240 million hectares in the world with a total production of 595.15 million tonnes. In India, production of wheat rose from 12 million tonnes during 1964-65 to 73 million tonnes in 2005-06 with an increased productivity level from 9 q/ha to 27 q/ha during the same period. However, great efforts and sound policies are needed to improve total production and productivity to tap the real genetic potential of wheat crop. As the Nobel Laurate Norman E. Borlaug estimated that to meet projected demands by the year 2025 AD, an average cereal yield must increase by 80% over the average of 1990. The fluctuations in food production coupled with environmental stresses makes this formidable task (Serageldin, 2000).

The major constraints towards the realization of potential yield are attributed both to biotic and abiotic stresses. Diseases play a greater havoc when appear in epidemic proportions. The diversity in climatic conditions prevalent in India provides a conducive environment for epidemics of rust(s) in large part of the country. Rust epidemics of the past have been reviewed by Nagarajan and Joshi (1975) and it was estimated that the leaf rust incidence on Kalyansona during 1971-72 alone led to a loss of 0.8 million tonnes (Joshi *et al.*, 1975). The review of literature on various aspects is presented here.

Biffen (1905) conducted the first study on genetics of rust resistance. He reported that a single recessive gene governs the yellow rust resistance. Subsequently, several other studies showed that resistance is governed by duplicate and complementary gene interactions. Most of these studies were conducted without considering the physiological specialisation (pathotype differentiation or concept of race) of the pathogen (Stakman and Piemeisel, 1917). Later on Flor (1942, 1956) while working on flax rust proposed gene-for-gene hypothesis implying that for each gene conditioning rust reaction in the host there is a corresponding specific gene considering pathogenicity in the pathogen. In other words, for every resistance gene present in the host, the pathogen has a gene for virulence. Susceptible reaction would result only when the pathogen is able to match all the resistance genes present in the host with appropriate virulence genes. If one or

more resistance genes are not matched by the pathogen with the appropriate virulence gene, resistant reaction is the result. In most of the pathogens, virulence is recessive to avirulence.

Person (1959) extended the gene-for-gene hypothesis to cultivars possessing two or more genes. These studies provided the genetic basis for breeding and identification of genes for rust resistance. Since then a large number of resistance genes have been identified and located in specific chromosomes. This became possible only when a set of aneuploids were developed by Sears (1954).

McIntosh and Watson (1982) observed that a single gene or a combination of genes for resistance might provide durable resistance. Evidence suggest that the species belonging to primary, secondary and tertiary gene pools of common wheat have contributed several resistance genes that are used in wheat improvement (McIntosh, 1991). Sears (1956) for the first time transferred *Lr*9 for leaf rust resistance from *Aegilops umbellulata* into wheat.

Inheritance studies showed resistance genes of all categories being monogenic, digenic, dominant, recessive or showing even more complicated pattern. (Gorlatch (1937) and Wells and Swenson (1944) showed monogenic resistance for leaf rust at mature plant stage. Gurdev *et al.* (1988) reported duplicate gene interaction by resistance genes for leaf rust and stripe rust.

Field resistance controlled by a single recessive gene for stem rust was reported in cultivars like Kota (Clark, 1924), S227, S308 and E4853 (Sheopuria *et al.,* 1970). Two dominant duplicate genes in Sharbati Sonora determined seedling resistance to races 15, 17 and 194 and one dominant gene to race 2A-1 (Jag Shoran Rao, 1974) of leaf rust. Adhikari and McIntosh (1998) identified stem rust resistance genes in nine triticale cultivars and postulated *Sr*Lal, *Sr*La2, *Sr*Bj, *Sr*j, *Sr*Nin genes. Thirteen winter wheat genotypes were evaluated and genes *Sr*31, *Sr*35 and *Sr*36 were postulated by Manninger *et al.* (1998).

2.1 Identification of Variation in Wheat Rust Pathogens

The rust pathogens are highly variable and many pathotypes are known to occur in each one of them. A rust infected field of susceptible variety may have many pathotypes. New pathotypes arise through sexual cycles. However, in the absence of

Table 1. Standard differentials for the identification of black, brown and yellow rust races

Black rust	Brown rust	Yellow rust
Little club	Malakoff	Michigan Amber
Marquis	Carina	Ble Rouge d'Ecosse
Reliance	Brevit	Strubes Dickkopf
Kota	Webster	Webster
Aurnautka	Loros	Holzapfels Fruh
Mindum	Mediterranean	Vilmorin 23
Spelmar	Hussar	Heines Kolben
Kubanka	Democrat	Carstens V
Acme		Spalding Prolific
Einkorn		Chinese 166
Vernal		Rouge P. Barbu
Khapli		*Triticum dicoccum var. tricoccum*
		Fong Tien
		Heils Franken
		Pet Kuser Roggen
		Lee
		Richersberg 42
Additional differentials		
Charter	Thew	Kalyansona
Yalta	NI 5439	Sonalika
E535	IWP 94	

Table 2. The revised constitution of sets of differential

Set-0	Set-A	Set-B
Brown rust (*Puccinia recondita tritici*)		
IWP 94 (*Lr.23*$^{+}$)	*Lr14a*	Loros(*Lr 2c)*
Kharchia Mutant	*Lr24*	Webster (*Lr 2a)*
Raj 3765	*Lr18*	Democrat (*Lr 3)*
PBW 343	*Lr13*	Thew (*Lr 20)*
UP 2338	*Lr17*	Malakoff (Lr1)
K 8804	*Lr15*	Benno (*Lr 26)*
Raj 1555	*Lr10*	
HD 2189	*Lr19*	
Agra Local		
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*	
Barley Local	*Sr 37*	
Agra Local		
Yellow rust (*Puccinia striiformis*)		
WH 147	Chinese 166 (*Yr 1)*	Hybrid 46 (*Yr 4)*
Barley local	Lee (*Yr 7*)	Heines VII (Yr 2+)
WH 416	Heines Kolben (*Yr 6)*	*Compair (Yr 8)*
PDW 215	Vilmorin 23 *(Yr 3)*	*T. spelta album (Yr 5)*
HD 2329		Moro *(Yr 10),* Tc61 *Lr 26* *(Yr 9)*
HD 2667		Strubes Dickkopf
Sonalika *(Yr 2+)*		
PBW 343	Suwon 92 X Omar	Kalyansona *Yr 2 (KS)*
HS 240	Riebesel 47/51 *(Yr 9+)*	
Anza		

alternate hosts new pathotypes arise either through somatic recombination, parasexuality, mutation or through introduction. In India, new pathotypes arise mainly by one step mutation or introduction.

The production of new pathotypes render existing resistant varieties susceptible. The phenomenon of physiological specialization also termed as specialization of parasitism, was first observed by Schroeter (1879) in *Puccinia graminis.*

Each *formae speciale* of rust species contains enormous genetic variation. These variants called pathotypes (races) are differentiated on the basis of infection types produced on a set of hosts called "differential", the host pathogenicity technique is quite reliable and accurate method of identification of pathotypes. Specialization of parasitism in black rust was demonstrated and existence of races in black rust of wheat came to be known in 1917 (Stakman and Piemeisel, 1917).

Rust samples are inoculated on standard differential host to identify the races (Table 1). Infection types are graded as 0 (immune), 1 (very resistant), 2 (moderately resistant), 3 (moderately susceptible), 4 (very susceptible) and x (mesothetic). A comparison of the infection type of existing races of differential host confirms the designation. This system was being followed in India also.

With the gene for gene theory of Flor (1956), it was realized that race (pathotyoe) identification system requires modification in order to use the virulence survey, which results in more meaningful purpose. Consequently, numerous procedures using near-isogenic lines were suggested around the world (Watson and Luig, 1966; Roelfs and McVey, 1979). 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). The system has 3 sets of differentials. Set '0' contained popular cultivars of bread wheat and durum, a susceptible cultivar and a resistant line to act as a watchdog. Set 'A' comprised of 8 near-isogenic lines of different *Lr genes.* Set 'B' had six selected lines from old international differentials. Keeping into account a number of lines in three sets and near-isogenic lines, the system permits to evaluate more than 14 resistance genes. Recasting of set 0 of differentials was done by Nayar *et al.* (1997). Subsequently on similar pattern, pathotype analysis systems were developed for black rust (Bahadur *et al.,* 1985) and yellow rust (Nagarajan *et al.,* 1984) (Table 2).

2.2 Host Plant Resistance

Resistance can be defined as the ability of host plant to withstand, oppose or overcome the attack of a pathogen completely or in some degree. Hart (1931) reported three main types of resistance in wheat to stem rust as protoplasmic (hypersensitive), morphological and functional resistance. Later, Van der Plank (1963, 1968) classified the resistance into race-specific (vertical) and non-race specific (horizontal) and subsequently this classification received much attention in resistance breeding (Nelson, 1978; Robinson, 1980).

Another category of seedling and adult plant resistance was recognised where seedling resistance remains operative both in seedling and adult stages, while adult plant resistance expresses only in mature plants (Manners, 1969; Robinson, 1976). Based on studies with stripe rust of wheat, in which the resistance to a pathogen continues for a number of years, Johnson (1981) introduced the term 'durable resistance'.

Various approaches for the incorporation of such a resistance have been critically studied. The genetic basis of rate reducing resistance can be due to adult plant resistance (Nayar *et al.*, 1994), temperature sensitive genes (Nayar *et al.*, 1997), polygenic resistance, and so on. However, vertifolia effect arising out of wilful selection for vertical resistance can result in the erosion of rate reducing resistance. An appropriate blend of both vertical and horizontal resistance may lead to more durable resistance in varieties. Also, development of cultivars with more than one effective resistance gene(s) comparatively will give long-lasting resistance. Some of the resistance genes can be characterised on the basis of molecular/morphological markers (Nagarajan, 1984; Nayar *et al.*, 1997) and cytology (Bhardwaj *et al.*, 1990). Information generated on the genetic constitution can be effectively used for the deployment of resistance genes on the basis of pathotype distribution data (Nayar *et al.*, 1997, Nayar and Bhardwaj, 1997).

Breeding for resistance to rusts has been the major priority of the national wheat improvement programme, so that the yield gains achieved through the spread of 'green revolution' technologies are not only sustained but further increased even at higher levels of crop management practices. The major emphasis has so far been on the incorporation of vertical resistance following pedigree method and selection for resistant phenotypes along with grain yield and other traits, without any regard to incorporation

of some specified genes combinations. The release of several rust resistant wheat varieties from time to time has been successful in containing rust epidemics that the country has experienced in the past. This programme, however, is of continuous nature due to complex relationship between the host and the pathogen and appearance of new virulence(s).

Efficient management of the crop disease through such a strategy requires an understanding of the functioning of resistance genes in different genetic backgrounds. In wheat varieties that have combinations of race specific resistance genes, the genes usually respond independently to an avirulent pathotype, producing the infection type (IT) of the gene with lowest category (Dyck and Kerber, 1981). But a combination of two or more such genes may give higher resistance that is conferred by the individual genes (Samborski and Dyck, 1968). Some genes that are difficult to be detected in seedling stage may also confer partial adult plant resistance but of durable type in combination with other genes of similar nature. Studies conducted to find out the genetic basis of rust resistance in Indian cultivars show that many of the varieties, inadvertently, also possess genes for adult plant resistance in combination with other seedling resistance genes (Nayar *et al.* 1994; Sawhney and Sharma, 1996).

2.3 Alien Gene Transfers in Wheat

The leaf rust resistance genes native to *T. aestivum* are almost ineffective comparatively to the alien genes from related species of cultivated wheat that are found to be effective to Indian pathotypes (Sawhney and Sharma, 1996). To achieve this, several strategies for producing wheat-alien chromosome translocations were reviewed by many wheat workers (Sears, 1972, 1981; Gall and Miller, 1987 and Feldman, 1988). The first group of methods of inducing wheat-alien translocations is by exploiting homoeologous chromosome pairing which can be achieved by eliminating chromosome 5B (Sears, 1972), using the ph Ib mutant (Sears, 1981; Koebner and Shepherd, 1985), or suppressing the effect of the ph I gene (Riley *et al.,* 1968). Spontaneous wheat-alien chromosome translocations occur frequently and form a second group of methods for homologous gene transfers (Sears, 1972; Metin *et al.,* 1973; Zeller, 1973 and Jiang *et al.,* 1993). The third group of methods include the use of ionizing irradiation (Sears, 1956), or tissue culture (Lapitan *et al.,* 1984) etc. A detailed account on recent advances in alien gene transfer in wheat have been reviewed (Jiang *et al.,* 1994; Friebe *et al.,*

1996). The method for transferring genes from related species largely depends on the evolutionary distance between the species involved. Species belonging to primary gene pool of common wheat share homologous genomes. Evidences suggest that the species belonging to primary, secondary and tertiary gene pools of common wheat have contributed several resistance genes that are used in wheat improvement (McIntosh, 1991). For example, Sears (1956) for the first time transferred *Lr*9 for leaf rust resistance from *Ae. umbellulata* chromosome (6U#1). The genotype 'Agent', a spontaneous wheat *Agropyron elongatum* translocation line, carries resistance genes *Lr*24/*Sr*24 of *A. elongatum* origin (Smith *et al.*, 1968).

Adult plant resistance genes are difficult to postulate based on seedling resistance though a number of them are said to be widely prevalent in Indian wheat. Also, presence of some genes in combination enhance their expression especially those exhibiting adult plant resistance such as *Lr*34 (Dyck, 1991). Samborski and Dyck (1992) reported the enhanced resistance combinations of *Lr*13 + *Lr*16. *Lr*30 + *Lr*3ka, *Lr*30 + *Lr*11 and *Lr*33 + *Lr*34. Presence of both *Lr*27 and *Lr*31 is necessary for the complementary resistance (Singh and McIntosh, 1984). Presence of *Lr*34 the APR gene is postulated based on the temperature dependent infection types. Also, the progressive leaf tip drying along the leaf margin is considered as additional supportive evidence. Therefore, postulation of *Lr*34 is more through the circumstantial evidences since the available pathotypes of *P. triticina* do not resolve it clearly at seedling stage.

The inheritance studies made in recent past have suggested the presence of number of resistance genes in many lines *viz.* 2 *Lr* genes in DL 803-3, HPW 63, WL 711 and WH 569; 3 *Lr* genes in CPAN 2099, HD 2380, HPW 42, HS 280 and WH 542, 4 *Lr* genes in K 9006; 2 *Sr* genes in HD 2329 and VL 616; 3 *Sr* genes in Lok-1 and Swati and 2 *Sr* genes in WH 896.

Linked genes can sometimes be used as markers for the presence of a specific gene for resistance even in the absence of differential reactions. eg. *Lr* 26/ *Sr* 31/ *Yr* 9/ *Pm* 8, *Lr* 20/ *Sr* 15, *Sr* 24/ *Lr* 24, *Lr* 34/ *Yr*18. However such genes can be postulated only if they are tightly linked to a gene of interest. *Lr* 24 confers resistance to all the Indian pathotypes of leaf rust but its presence can be detected through the presence of tightly linked gene *Sr* 24 that produces differential reactions with black rust pathotypes. The genes *Lr* 26, *Sr* 31, *Yr* 9 and *Pm* 8 co-segregates and hence, the information generated

on *Lr* 26 can be effectively used for the presence of other genes of this linkage group (Nayar *et al.*, 2001).

2.4 Potential Donors of Disease Resistance in Wheat

Plant breeders use diverse sources of disease resistance in breeding programmes. The most common source include land races and modern cultivar(s) as the genes for resistance are easier to move from one varietal background to the other through conventional breeding methods. If the donors for resistance are not available in the cultivated or primitive genotypes, species and genera are utilised as donor. Progenitors of hexaploid wheat, allied genera and species constitute a vast reservoir of potentially useful genes for disease and pest resistance in particular and wheat improvement in general. Many wild relatives and related spieces can be successfully crossed with bread wheat and the useful traits can be transferred into wheat. Several agronomically important traits including resistance to diseases and pests, moisture stress and salt tolerance etc. have been transferred from these species to wheat (Zeller and Hsam, 1983; Jiang *et al.*, 1994) by adopting wide hybridisation technique. Important among them have been the genes for resistance to stem rust, leaf rust, stripe rust, powdery mildew and a few other foliar diseases.

Among the progenitors of the common wheat, einkorn wheat, *T. boeoticum* ssp. *aegilopoides* exhibited high degree of resistance to leaf rust, stripe rust and powdery mildew at adult plant stage, while ssp. *thoudar* was resistant to all the three rusts. The accessions of *T. boeoticum* have shown a high level of resistance in other geographical areas also (Zohary *et al.*, 1969; Gill *et al.*, 1985). Among the sitopsis group, *Ae. speltoides* showed a high level of resistance to stem rust, leaf rust and powdery mildew. The major gene pool represented by the sitopsis section remains largely untaped for disease resistance. Dhaliwal *et al.* (1986) evaluated a large collection of wild wheat and identified many accessions having genes for resistance to different diseases. A high frequency of resistance to powdery mildew and leaf rust occurred among the *Aegilops* species (Gill *et al.*, 1985; Tomar and Menon, 2001).

The tetraploid wheat species *T. timopheevi* Zhuk. and *T. militinae* Zhuk *et.* Migush. (2n=4x=28, genome AAGG) are well known for their complex resistance to diseases. *T. timopheevi* was found in western Georgia in 1926, while *T. militinae* was isolated as

a spontaneous mutant from collection plots of *T. timopheevi* in 1950 by Zhukovsky. Both of these species have attracted the interest of wheat breeders and geneticists on account of their exceptionally high immunity to several diseases of wheat (Zhukovsky, 1971).

The botanical variety, *T. militinae* var. *albimilitinae* arose naturally in a population of interspecific hybrids involving *T. timopheevi*. Mutational factors affected the genetic systems controlling ear compactness, ear colour, glume firmness and the reproductive system. *T. militinae* has resistance to the common fungal diseases and a high protein content that can be recommended as a donor in wheat breeding (Apel an Moeseev, 1982).

Tomar *et al.* (1988) evaluated 15 accessions of *T. timopheevi,* eight accessions of *T. araraticum* and one accession of *T. militinae* for disease resistance at Wellington, Tamilnadu, a hot spot for many wheat diseases and showed that *T. timopheevi* and *T. militinae* accessions were resistant to all the three wheat rusts as well as powdery mildew pathogen (*Erysiphe graminis*).

Genetic analysis of progenies of the crosses between common wheat (Saratovskaya 29) and tetraploid species (*T. timopheevi* and *T. militinae*) revealed monogenic control of resistance to *Puccinia recondita* and *Erysiphe graminis* f. sp. *tritici*. Some of these introgressed lines had an effective gene for resistance which was independent of genes *Lr9*, *Lr*19, *Lr*24 and was considered either identical to gene *Lr*28 or closely linked to it. The majority of lines carried new effective genes for resistance, which were different from *Lr*9, *Lr*19 and *Lr*24 and are yet to be designated. Monosomic analysis revealed the presence of a dominant gene for resistance to powdery mildew (*Erysiphe graminis*) derived from *T. timopheevi* located on chromosome 4A (Peusha *et al.*, 1996).

Tomar *et al.* (1997) carried out interspecific hybridisation between the wild accession of *T. militinae* and *T. aestivum* cvs. C306 and Sonalika. From backcrossing and further selections, cytologically stable derivatives viz. Sel. T216 and Sel. T2600 were obtained. These lines exhibited a high level of resistance at seedling as well as adult plant stages to most virulent pathotypes 77-2 and 77-5 of *P. recondita.*

The tetraploid and hexaploid *Aegilops* species carrying one set of D genome namely *Ae ventricosa* (DDUnUn or DDMM), *Ae cylindrica* (CCDD), *Ae crassa* (both 4x and 6x genomes DDMM and DDMMSS respectively), *Ae juvenalis* (DDMMUU) and *Ae crassa* var. *vavilovi* were found susceptible to leaf rust but showed low infection to stem rust (Tomar and Kochumadhavan, 1993). The study reveals that some accessions of *T. sphaerococcum* and *T. compactum* may possess valuable genes for high adult plant resistance to stripe rust (Tomar *et al.,* 1986) and *T. amplissifolium* for leaf rust.

Prasad (1947) observed the occurrence of uredinial and telial stages on leaves, stems and ears of *A. semicostatum* and *A. longearistatum* in the Shimla hills. These results indicated that the stem rust occurring on *Agropyron* spp. in natural condition is the same as that on wheat. It is likely that some of the *Agropyron* sp. serve as collateral host for stem rust of wheat. Vasudeva *et al.* (1958) reported that some of the *Agropyron* spp. take infection of *P. graminis tritici*. Brahma (1988) isolated rust from uredinial pustules occurring on leaves or stems of *Agropyron* spp. and cross inoculated on cv. Agra local (*T. aestivum*). Isolates from all the *Agropyron* spp. infected Agra local heavily. Further testing revealed that stem rust pathotypes 40A and 117A were common infecting both *T. aestivum* and *Agropyron* spp. and that *Agropyron* spp. are the collateral hosts of 40A and 117A pathotypes.

Kochumadhavan *et al.* (1988) and Tomar and Menon (1993) evaluated sixty six accessions of *Agropyron* for wheat rusts and powdery mildew under natural, epiphytotic conditions at Wellington. They found forty percent of the accessions including *A. elongatum* (2x) and *A. junceum* (2x) were resistant to all the three rusts.

2.5 Inheritance and Chromosomal Location of Rust Resistance Genes

Inheritance studies were started even before the rediscovery of Mendel's laws (Farrer, 1898) while location of a specific gene on individual chromosome started only after the creation of aneuploid stocks. Complete set of monosomics, nullisomics and tetrasomics for each chromosome in the background of Chinese Spring developed by Sears (1954) paved the way for extensive studies on chromosomal location of genes in wheat. Further, Sears and Sears (1979) isolated all the possible telocentrics, 41 in Chinese spring and one telo 7 DL in Canthatch. The various aneuploids especially monosomics and telo-centrics have been used extensively to identify the specific

chromosomes that carry resistance gene(s). Numerous reports are available on inheritance and chromosomal location of wheat genes by the scientists all over the world. A concise review on inheritance of resistance to leaf and stem rust was given by Ausemus *et al.* (1946). Inheritance studies showed resistance genes of all categories being monogenic, digenic, dominant, recessive or sharing even more complicated pattern. Of enormous data on inheritance of genes for resistance, only a few examples have been presented below under each category.

2.5.1 Leaf rust resistance

Independent inheritance of adult plant and seedling resistance was reported by Mains *et al.* (1926) and Wismer (1934). Gorlatch (1937) and Wells and Swenson (1944) showed monogenic resistance at mature plant stage. Mains *et al.* (1926) reported monogenic inheritance as dominant, partially dominant or recessive in accordance with parents and races used. Dominant monogenic inheritance in seedling stage was evident from studies carried out by Waterhouse (1930); recessive monogenic control by Caldwell and Compton (1947) and Martinez *et al.* (1953) and Shaalan *et al.* (1966).

Incompletely dominant gene *Lr*14a *in 'Spica'* (McIntosh *et al.*, 1967), *Lr*17 in Klein Lencero and *Lr*18 in Africa 43 (Dyck and Samborski, 1968) were reported. About thirteen Bulgarian wheat cultivars exhibited an incomplete disease resistant reaction against *P. recondita* pathogen (Todorova, 2000). McIntosh and Dyck (1975) observed that *Lr*23 behave recessive as well as dominant in different crosses and that dominance was greater at higher temperatures. Studies carried out by Nagarajan *et al.* (1984) revealed that *Lr*13, *Lr*17 and *Lr*31 are temperature sensitive.

Singh and McIntosh (1984a, 1984b) reported the complementary interaction of *Lr*27 and *Lr*31 genes. Dyck (1987) observed two complementary genes *Lr*T2 and *Lr*T3 in wheat varieties, Terenzio and Lageadinho of which *Lr*T2 has been later designated to be *Lr*34. Gene *Lr*34 interacts with seedling complementary genes *Lr*27 and *Lr*31 for enhanced and durable resistance to leaf rust (Sawhney, 1992). Durable resistance to leaf and stripe rust has often associated with the linked *Lr*34 and *Yr*18 genes. In South Africa, *Lr*34 can be detected in wheat seedling at low temperature but resistance is most effective in adult plant stage.

Duplicate gene interaction by resistance genes for leaf rust was exhibited by *Lr*7 and *Lr*8 (Fitzgerald *et al.*, 1957). Gurudev *et al.* (1988) reported similar interaction of genes for resistance to stripe rust and leaf rust. Slow rusting of leaf rust resistance in wheat is often observed by a few partially effective, non-hypersensitive type of genes with additive effects (Singh *et al.*, 2000).

2.5.2 Stem rust resistance

Dominant monogenic control for resistance to stem rust pathogen *P. graminis* f. sp. *tritici* was accounted in Kanred (Aamodt, 1922), in E 4870 (Sheopuria *et al.*, 1970) etc. Field resistance to stem rust controlled by a single recessive gene was reported in cultivars like Kota (Clark, 1924), S 227, S 308 and E4853 (Sheopuria *et al.*, 1970). The designated stem rust resistance genes viz. *Sr*30 (Knott and McIntosh, 1978) and *Sr*8b (Singh and McIntosh, 1986) were found to be recessive in nature. In a wheat-rye recombinant line, 'Selection 212', the resistance to stem rust races 122 and 40A was governed by a single recessive gene (Sharma and Singh, 2000). Chen Wan Quan *et al.* (2001) identified resistance genes in 11 out of 40 rust resistant cultivars tested in China and are yet to be designated.

Incomplete dominant gene imparting resistance was observed in Vernel to race 21 (Harrington and Smith, 1929). In Chhoti lerma, one dominant gene to races 15, 17, 34A and 194 and two recessive genes each to races 21A-1 and 122 conferred resistance. Similarly two dominant duplicate gene in Sharbati Sonora determined seedling resistance to races 15, 17 and 194 and one dominant gene to race 12A-1 (Jag Shoran Rao, 1974).

Waterhouse (1929) observed the loss of rust resistance in wheat and oat seedlings under warm summer conditions. Similar results were reported by Johnson and Newton (1937, 1941), Green and Johnson (1955), Forsyth (1956), Joshi (1962). Loegering (1966) and Luig and Rajaram (1972). The reverse change from susceptibility to resistance due to increased temperatures was noticed in *Sr*9b aznd *Sr*14 (Roelfs and McVey, 1979; Luig, 1988).

Adhikari and McIntosh (1998) identified stem rust resistance genes in nine triticale cultivars and postulated *Sr*La1, *Sr*La2, *Sr*Bj, *Sr*J, *Sr* Nin genes. Thirteeen winter wheat genotypes were evaluated for stem rust resistance and the genes *Sr*31, *Sr*35 and *Sr*36 were postulated (Manninger *et al.*, 1998) in them.

A comprehensive details of catalogued leaf and stem rust resistance genes, their origin, chromosomal location and features are mentioned in Tables 3 and 4, respectively.

2.6 Genetics of Morphological Characters

Maystrenko (1976) has identified and located genes controlling leaf hairiness in young plants of bread wheat. The varieties with marked hairiness of leaves, Saratov 29, Saratov 210 and Milturum 321 were crossed with Chinese spring (euploid and monosomic lines). Monosomic analysis revealed that the gene controlling hairiness, *Hl*, is located on chromosome 4A. Kuspira *et al.* (1957) concluded that only one major gene locus, with a multiple allelic series, appear to be involved in determining leaf pubescence in *T. monococcum.* Specifically pubescent leaves Hl^p is dominant to glabrous leaves *Hl*. He provided evidence for at least three alleles at *Hl* locus in *T. monococcum.* With the reversal of chromosome designation for 4A and 4B, this locus in *T. monococcum* cannot be allelic to the gene in *T. aestivum*. Neatby and Goulden (1930) indicated digenic control of this character. Laikova *et al.*(1980), clearly indicated that the mode of inheritance of pubescent leaves is monogenic.

Maystrenko (1976) and Laikova *et al.* (1980) showed that the gene for leaf pubescence in *T. aestivum* is on the β arm of chromosome 4A. Maystrenko (1976) showed that modifying genes in chromosome *1A, 1B, 2B, 4B, 6B, 2D* and *6D* in *T. aestivum* affect the expression of leaf pubescence.

Table 3 Origin, location, and other special features of leaf rust resistance (*Lr*) genes

Gene	Origin	Chromosome location	Special features	Reference
Lr1	*T. aestivum*	5DL	Effective in Italy	Ausemus *et aql.* (1946); McIntosh and Baker (1970); Dyck and Johnson (1983); Singh and Gupta (1991)
Lr2a	*T. aestivum*	2DS	Temperature sensitive recessive gene	Ausemus *et al.* (1946); Luig and McIntosh (1968); Dyck and Samborski (1974); Browder (1980)
Lr2b	*T. aestivum*	2DS	-	Dyck and Samborski (1974); Browder (1980)
Lr2c	*T. aestivum*	2DS	-	Dyck and Samborski (1974); Browder (1980)
Lr3	*T. astivum*	6BL	Usually incompletely dominant, may display disturbed genetic ratio	Ausemus *et al.* (1946); Heyne and Livers (1953); Luig (1964); Browder (1980); Singh and Gupta (1991)
Lr3bg	*T. aestivum*	6BL	-	Haggag and Dyck (1973); Dyck and Johnson (1983)
Lr3ka	*T. aestivum*	6BL	-	Haggag and Dyck(1973); Dyck and Johnson (1983)
Lr4 to *Lr8*	*T. aestivum*	-	-	Fitzgerald *et al.* (1957)
Lr9	*T. umbellulatum*	6BL	-	Soliman *et al.* (1963); Sears (1956, 1961, 1966); Browder (1980)
Lr10	*T. aestivum*	1AS	Epistatic to *Lr2* and Temperature sensitive	Choudhuri (1958); Browder (1980); Singh and Gupta (1991)
Lr11	*T. aestivum*	2A	Low Temperature	Soliman *et al.* (1964); Williams and Johnson (1965); Singh and Gupta (1991)
Lr12	*T. aestivum*	4B	Adult plant Resistance (ineffective in India)	Dyck *et al.* (1966); Dyck (1991); McIntosh, *et al.* (1995)

Gene	Origin	Chromosome location	Special features	Reference
Lr13	*T. aestivum*	2BS	High Temperature	Dyck *et al.* (1966); Hwwthorn (1984); Pretorius *et al.* (1984); Singh and Gupta (1991)
Lr14a	*T. aestivum*	7BL	Low Tempetarue	Law and Johnson (1967); Dyck and Samborski (1970); Dyck and Johnson (1983); McIntosh *et al.* (1995)
Lr14b	*T. aestivum*	7BL	Associated with with APR, Lower temperatgure	Dyck and Samborski (1970); Dyck and Johnson (1983); Sawhney *et al.* (1992)
Lr15	*T. aestivum*	2DS	-	Luig and McIntosh (1968); McIntosh and Baker (1968); Dyck and Johnson (1983)
Lr16	*T. aestivum*	2BS	Always associated with *Sr23*, high Temperature	Dyck and Samborski (1968); Dyck and Johnson (1983); Singh and Guipta (1991)
Lr17	*T. aestivum*	2AS	Closely linked with *Lr37/Sr38/Yr17* in a VPM derivative high temperatgure	Dyck and Samborski (1968); Dyck and Kerber (1977a); Dyck and Johnson (1983); Bariana and McIntosh (1993)
Lr18	*T. timopheevi*	5BL	Low temperature	Dyck and Samborski (1968); Browder (1980); McIntosh (1983)
Lr19	*Thinopyrum ponticum*	7DL	Linked with Yellow pigment of flour, *Sr25*	Sharma and Knott (1966); Browder (1972)
Lr20	*T. aestivum*	7AL	Low temperature linked with *Sr15*	Browder (1972);
Lr21	*T. tauschii* var. *meyeri*	1DL (also reported on 1DS)	-	Rowland and Kerber (1974); Johnes *et al.* (1990); Dyck and Johnson (1983)
Lr22a	*T. tauschii* var. *strangulata*	2DS	Adult plant Resistance	Rowland and Kerber (1974); Dyck and Johnson (1983)
Lr22b	*T. aestivum*	2DS	Adult plant Resistance (Only few Avirulent pathotypes)	Dyck (1979)

Gene	Origin	Chromosome location	Special features	Reference
Lr23	*T. turgidum*	2BS	Close linkage with with *Lr13*, recessive to partial dominance, greater dominance at higher temperature	McIntosh aznd Dyck (1975); Dyck and Johnson (1983); Sharma (1990); Sharma and Sawhney (1991); Singh and Gupta (1991)
Lr24	*Th. ponticum*	3DL	Closely linked with *Sr24*	Smith *et al.* (1968); McIntosh *et al.* (1976); Dyck and Johnson (1983)
Lr25	*Secale cereale*	4BS	Close linkage with *Pm7*	Driscoll and Anderson (1967); McIntosh (1988a)
Lr26	*Secale cereale*	1RS-1BL	Completely linked with *Sr31, Yr9, Pm8*	Mettin *et al.* (1973); Zeller (1973); McIntosh (1988a); Singh and Gupta (1991)
Lr27	*T. aestivum*	3BS	Complementary to *Lr31*	Singh and McIntosh (1984a&b) Singh and Gupta (1991)
Lr28	*Ae. speltoides*	4AL	-	McIntosh *et al.* (1982)
Lr29	*Th. ponticum*	7DS	-	McIntosh (1988a); Knott (1989)
Lr30	*T. aestivum*	4AL	Recesive gene	Dyck and Kerber (1981); Singh and Gupta (1991)
Lr31	*T. aestivum*	4BS	Complementary to *Lr27*	Singh and McIntosh (1984a&b) Singh and Gupta (1991)
Lr32	*T. tauschii*	3DS	-	Kerber (1987, 1988)
Lr33	*T. aestivum*	1BL	Linked with *Lr2b* and complementary to *Lr34*	Dyck *et al.*(1987)
Lr34	*T. aestivum*	7DS	APR gene linked with *Yr18* and leaf tip necrosis	Dyck (1987); Singh and Gupta (1991); Singh (1992a&b); Singh and Rajaram (1992)
Lr35	*Ae. speltoides*	2B	APR gene	Kerber and Dyck (1990)
Lr36	*Ae. speltoides*	6BS	-	Dvorak and Knott (1990)
Lr37	*T. ventricosum*	2AS	More effective at 17°C, linked to *Yr*17, *Sr*38, *Lr*17	Bariana and McIntosh (1993)

Gene	Origin	Chromosome location	Special features	Reference
Lr38	*A. intermedium*	-	-	Friebe *et al.* (1992)
Lr39	*T. tauschii*	-	-	McIntosh, Wellings *et al.* (1995)
Lr40	*T. tauschii*	-	-	McIntosh, Wellings *et al.* (1995)
Lr41	*T. tauschii*	1D	-	Cox *et al.* (1994)
Lr42	*T. tauschii*	1D	-	Cox *et al.* (1994)
Lr43	*T. tauschii*	7DS	-	Cox *et al.* (1994); Hussien *et al.* (1977)
Lr44	*T. spelta*	1B	-	McIntosh, Wellings *et al.* (1995)
Lr45	*Secale cereale*	2A	-	McIntosh, Wellings *et al.* (1995)
Lr47	*T. speltoides*	7AS	7S-7A translocation	Porter (1998)
Lr48	*T. aestivum*	4BL	-	McIntosh *et al.* (2005)
Lr49	*T. aestivum*	2AS	-	McIntosh *et al.* (2005)
Lr50	*T. armeniacum*	2BL	-	McIntosh *et al.* (2005)
Lr51	*Ae. speltoides*	1BL	-	McIntosh *et al.* (2005)
Lr52	*T. aestivum*	5BS	-	McIntosh *et al.* (2005)
Lr53	*T. dicoccoides*	6BS	-	McIntosh *et al.* (2005)
Lr54	*Ae. speltoides*	2DL	-	McIntosh *et al.* (2005)
Lr55	*Hardeum bulbosum*	1B	-	McIntosh *et al.* (2005)

Table 4 Origin, location and special features of stem rust resistance (Sr) genes

Gene	Origin	Chromosome location	Special features	Reference
Sr2	*T. turgidum* var. *dicoccum*	3BS	APR gene, recessive inheritance	Ausemus *et al.* (1946) Knott (1968); McIntosh (1988b)
Sr3	(Symbols abandoned)			
Sr4	(Symbols abandoned)			
Sr5	*T. aestivum*	6DS	-	Ausemus *et al.* (1946) Sears *et al.* (1957)
Sr6	*T. aestivum*	2DS	Dominant or recessive depending on patho-type temperature and genetic background, temperatue < 22°C	Knot and Anderson (1956); McIntosh and Baker (1968)
Sr7a	*T. aestivum*	4AL	Prominent yellow chlorosis/necrosis surrounding the uredia	Knott and Anderson (1956); Loegering and Sears (1966)
Sr7b	*T. aestivum*	4AL	-	Loegering andSears (1966)
Sr8a	*T. aestivum*	6AS	-	Knott and Anderson (1956); Singh and McIntosh (1986)
Sr8b	*T. aestivum*	6AS	Recesssive low temp.	Singh and McIntosh (1986)
Sr9a	*T. astivum*	2BL	Ineffective in India	Green *et al.* (1960)
Sr9b	*T. aestivum*	2BL	-	Green *et al.* (1960)
Sr9d (Syn Sr1)	*T. turgidum*	2BL	Ineffective in India	Knott (1966)
Sr9e	*T. turgidum*	2BL	-	McIntosh and Luig (1973a)
Sr9f	*T. aestivum*	2BL	Ineffective in India	Loegering (1975)
Sr9g	*T. turgidum* var. *durum*	2BL	Closely associated with *Yr7*	McIntosh and Luig (1973a)
Sr10	*T. aestivum*	2B	Low temperature	Knott and Anderson (1956) McIntosh, Hart *et al.* (1995)
Sr11	*T. turgidum*	6BL	Disturbed genetic ratios	Knott and Anderson (1956); Sears (1966)
Sr12	*T. turgidum* var. *durum*	3BS	Recessive, low temperature (<20°C)	Sheen and Snyder (1964) McIntosh *et al.* (1980)

Gene	Origin	Chromosome location	Special features	Reference
Sr13	*T. turgidum* var. *dicoccum*	6AL	High temperature (20-28°C)	Knott (1962)
Sr14	*T. turgidum* var. *dicoccum*	1BL	High light and high temperature	Knott (1962); McIntosh (1980)
Sr15	*T. aestivum*	7AL	Closely linked to *Lr 20* and *Pml,* Low temperature (15-18°C)	Watson and Luig (1966)
Sr16	*T. astivum*	2BL	-	Loegering and Sears (1966)
Sr17	*T. aestivum*	7BL	Recessive low temperature	McIntosh (1988a); McIntosh *et al.* (1967)
Sr18	*T. aestivum*	1DL	-	Baker *et al.* (1970)
Sr19	*T. aestivum*	2BS	-	Anderson *et al.* (1971)
Sr20	*T. aestivum*	2B	-	Anderson *et al.* (1971)
Sr21	*T.monococcum*	2AL	-	The (1973); The *et al.* (1979)
Sr22	*T. monococcum*	7AL	Low temperature	The and McIntosh (1975)
Sr23	*T. astivum*	2BS	Closely linked with *Lr16*	McIntosh and Luig (1973b)
Sr24	*Th. ponticum*	3DL	Closely linked with *Lr24*	McIntosh *et al.* (1976)
Sr25	*Th. ponticum*	7DL	Associated with *Lr19*	McIntosh *et al.* (1976)
Sr26	*Th. ponticum*	6AL	-	Knott (1961)
Sr27	*Secale cereale*	3A	-	Acosta (1963); McIntosh (1988a)
Sr28	*T. aestivum*	2BL	-	McIntosh (1978)
Sr29	*T. aestivum*	6DL	-	Dyck and Kerber (1977b)
Sr30	*T. aestivum*	5DL	Recessive	Knott and McIntosh (1978)
Sr31	*Secale cerale*	1RS,1BL	Linked to *Lr26, Yr9, Pm8*	Mettin *et al.* (1973); Zeller (1973)
Sr32	*T. speltoides*	Independent translocation on 2A, 2B, 2D	-	McIntosh (1988a); McIntosh, Hart *et al.* (1995)

Gene	Origin	Chromosome location	Special features	Reference
Sr33	*T. tauschii*	1DL	Linked with *Lr21* Low temperature	Kerber and Dyck(1979) McIntosh *et al.* (1982)
Sr34	*T. comosum*	2M	-	
Sr35	*T. monococcum*	3AL	-	McIntosh *et al.* (1984)
Sr36	*T. timopheevi*	2BS	-	McIntosh and Gyarfas (1971)
Sr37	*T. timopheevi*	4BL	-	McIntosh and Gyarfas (1971)
Sr38	*T. ventricosum*	2AS	Low temperature	Bariana and McIntosh (1993)
Sr39	*T. speltoides*	2B	Incompletely dominaznt	Kerber and Dyck (1990)
Sr40	*T. timopheevi*	2BS	-	Dyck (1992)
Sr41	*T. aestivum*	4D	-	McIntosh, Wellings *et al.*(1995)
Sr42	*T. aestivum*	6DS	-	McIntosh *et al.* (2005)
Sr43	*T. aestivum*	-	-	McIntosh *et al.* (2005)
Sr44	*Agrotrichum*	-	7A-1L, 7A-1S translocation	McIntosh *et al.* (2005)
Sr45	*Ae. tauschii*	1DS	-	McIntosh *et al.* (2005)

MATERIALS AND METHODS

The experimental materials used and methods adopted for the present investigation on genetic analysis and chromosomal location of leaf rust and stem rust gene(s) in interspecific derivatives of wheat (*Triticum aestivum* L.) are described hereunder.

3.1 Experimental Materials

3.1.1 Leaf and stem rust resistant genetic stocks Selection 2429 and Selection 4805-1

Selection 2429 (Sel. 2429) was selected from the cross wheat x *Aegilops speltoides*. This showed adult plant resistance to leaf and stem rusts. This genotype also has its leaf sheath pubescent. Similarly, Selection 4805-1 (derivative of wheat x *Triticum militinae*) showed adult plant resistance to both leaf rust and stem rust. This line has pubescence on leaves and leaf sheath. Both the stocks were tested in seedling stage with individual pathotypes as well as with a mixture of races of stem rusts and leaf rusts under artificial epiphytotic conditions in glasshouses and field respectively. Seedling tests were carried out at DWR Regional Station, Flowerdale, Shimla as well as at New Delhi.

3.1.2 Monosomic series

A complete set of monosomic lines (2n-1) differing for the deficient chromosomes in the background of Chinese Spring were used to assign resistance gene(s) in Sel.2429 to specific chromosomes.

3.1.3 Susceptible checks

In order to study the inheritance pattern of *Aegilops speltoides* and *Triticum militinae* derived stem rust and leaf rust resistance, the wheat cultivars viz. Agra Local (AL), Lal Bahadur (LB), WL 711 and NI 5439 were used as one of the parents. All these cultivars are known for their high degree of susceptibility to most of the Indian leaf rust and stem rust races/biotypes/pathotypes both at seedling and adult plant stages. Agra Local is widely used as susceptible check as well as for maintaining the various pathotypes of leaf rust and stem rust pathogens.

3.1.4 Genetic stocks used for test of allelism

Genetic stocks carrying leaf rust resistance genes of alien origin viz., *Lr*19 and *Lr*24 present in the backgrounds of HW 2043 and HW 2003 respectively were used to test the identity of leaf rust resistance in Sel. 2429 derived from *A. speltoides*. Both *Lr*19 and *Lr*24 have been derived from *A. elongatum* (= *Thinopyrum ponticum*).

3.1.5 Differential sets

The near-isogenic lines (NILs) and stocks carrying known genes in use as differentials in India for pathotype analysis of leaf rust pathogen *Puccinia triticina* (Nagarajan *et al.*, 1983) and stem rust pathogen *Puccinia graminis* f. sp. *tritici* (Bahadur *et al.*, 1985) were considered in the present study. The constitution of sets of differentials are presented in Tables 5 and 6.

3.1.6 Pathogen

Three pathotypes of *P. triticina* f. sp. *tritici* and two pathotypes of *P. graminis* f. sp. *tritici*, which are more prevalent, were used for multipathotype analysis. The tested races along with their new designations as proposed by Nagarajan *et al.* (1983) and Bahadur *et al.* (1985) are presented in Table 5 and 6. The initial inoculum of pure uredospores of respective races was obtained from DWR, Regional station, Flowerdale, Shimla and Division of Plant Patholoty, Indian Agricultural Research Institute, New Delhi. The inoculum of each race was multiplied and used after ensuring the purity using differentials concerned. The races 77-5, 77-6 and 77-7 of leaf rust pathogen and the races 117-6 and 40A of stem rust pathogens were used for genetic analysis of resistance gene(s) in Sel.2429 and Sel.4805-1. The race 77-5 of leaf rust pathogen was used in the study of chromosomal location of resistance gene(s) in Sel. 2429.

All experimental materials were provided by the Division of Genetics and Division of Plant Pathology, Indian Agricultural Research Institute, New Delhi. The field experiments and glass house screening were conducted at Division of Genetics, I.A.R.I., New Delhi (during *rabi* 2002, 2003 and 2004), I.A.R.I. Regional Station, Wellilngton, Tamil Nadu (during offseason, *Kharif* 2003).

Table 5. **Infection types of three pathotypes of *P. recondita* f. sp. *tritici* on differential sets A and B**

Pathotype differential	77-5 (121R63-1)	77-6 (121R554)	77-7 (121R127)
Set A			
Lr 14a	3+	3+	3+
Lr 24	;1	;1	0;
Lr 18	12	X+	;
Lr 13	3+	3+	3+
Lr 17	3+	X+3+	3+
Lr 15	3+	3+	3+
Lr 10	3+	X+3+	3+
Lr 19	0;	0;	0;
Set B			
Laros (*Lr* 2c)	3+	3+	3+
Webster (*Lr*)	3+	3+	3+
Democrate	3+	3+	3+
Thew (*Lr* 20)	3+	3+	3+
Malakoff (*Lr*)	3+	3+	3+
Benno (*Lr* 26)	3+	3+	3+
Lr 23	-	-	3+

Table 6. **Infection types of two pathotypes of *P. graminis* f. sp. *tritici* on differential sets A and B**

Pathotype differential	40A (62G29)	117-6 (37G19)
Set A		
Sr 13 Mq	12	3+
Sr 9b Mq	3+	2
Sr 11 Mq	3+	3+
Sr 28 Kota	3+	0;
Sr 8 Mq	3+	2
Sr 9e Verstein	3+	3+
Sr 30 Webster	2	2
Sr 37 line w	;1	2
Set B		
Marquis (*Sr*7b, *Sr* 18, *Sr*)	3+	3+
Einkorn (*Sr* 21)	;1	3+
Kota (*Sr* 7b, *Sr* 19, *Sr* 28)	4	0;
Reliance (*Sr* 5, *Sr* 16, *Sr* 18)	3+	0;
Charter (*Sr* 11)	3+	3
Khapli (*Sr* 7a, *Sr* 13, *Sr* 14)	10	2+

3.2 Methods

3.2.1 Cytological identification of monosomic plants

The complete set of 21 monosomic lines of Chinese Spring (CS) were raised in plots separately and monosomic plants were cytologically identified in each line at first meiotic metaphase stage (Metaphase I). Likewise the F_1 monosomic plants were also identified cytologically to obtain F_2 seeds (Plates 1-4). The identification procedure followed was as under :

Screening and fixing of anthers

The appropriate young spikes were selected just before their emergence from bootleaf and screened for pre-selection of anthers at desired stage of cell division of three anthers in a floret, only one was squashed between microscopic slide and coverslip after adding a drop of 2% acetocarmine strain and examined under the light microscope. The floret with anthers at first meiotic metaphase stage was identified and the remaining two anthers were fixed in modified Carnoy's fluid (3:1 ratio of ethanol and glacial acetic acid).

Pre-treatment and staining

Fixed anthers were hydrolysed in a glass tube using 1N HCl at 60°C for 12 minutes. After hydrolysis, 1 N HCl was discarded from the tube and added with carbolic-feulgen stain. Anthers were taken out as they turned deep violet in colour and squashed between slide and coverslip after adding a drop of acetocarmine (2%) as spreader. The slide was heated gently and tapped uniformly. Excess stain was removed using filter paper and by exerting a uniform pressure on cover slip in order to ensure a good spread of cells and separation of chromosomes. The PMC's were observed under microscope and chromosomes were counted. Thus plants which showed 41 (20"+1') chromosomes were selected in each line. Monosomic F_1 plants in each line were also identified and tagged in a similar manner.

3.2.2 Hybridization to make different crosses

To study the inheritance and chromosomal location of rust resistance gene(s), the following crosses were made.

(i) Agra Local x Sel. 2429

Plate 1: Disomic F_1 plant with 19 bivalents and 4 univalents at first meiotic metaphase

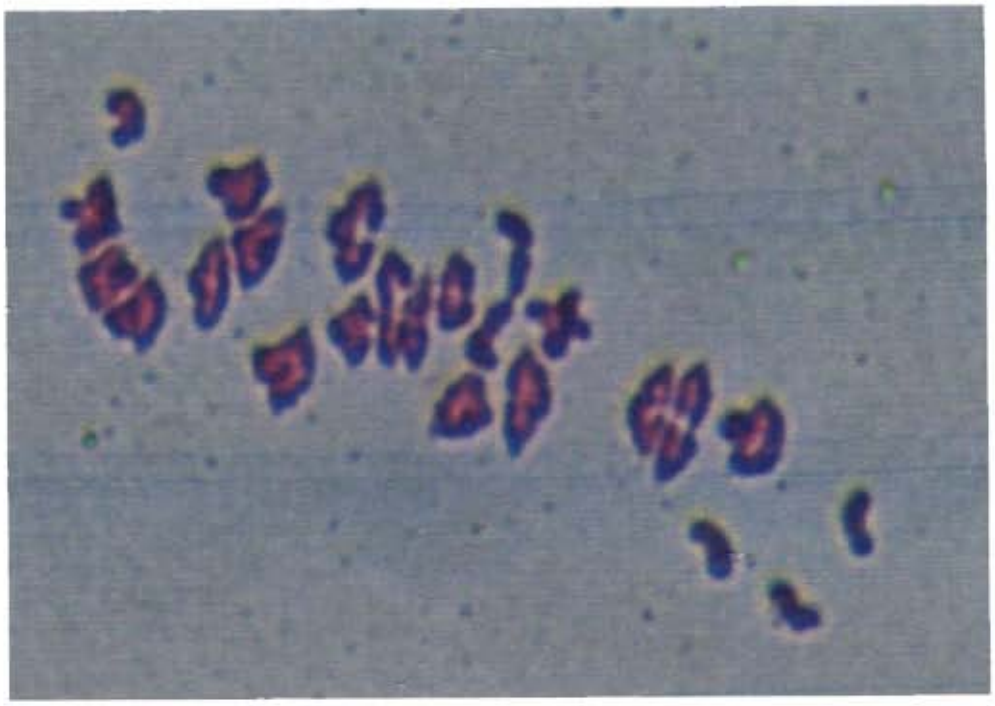


Plate 2: Monosomic F_1 plant with 17 bivalents and 7 univalents at first meiotic metaphase

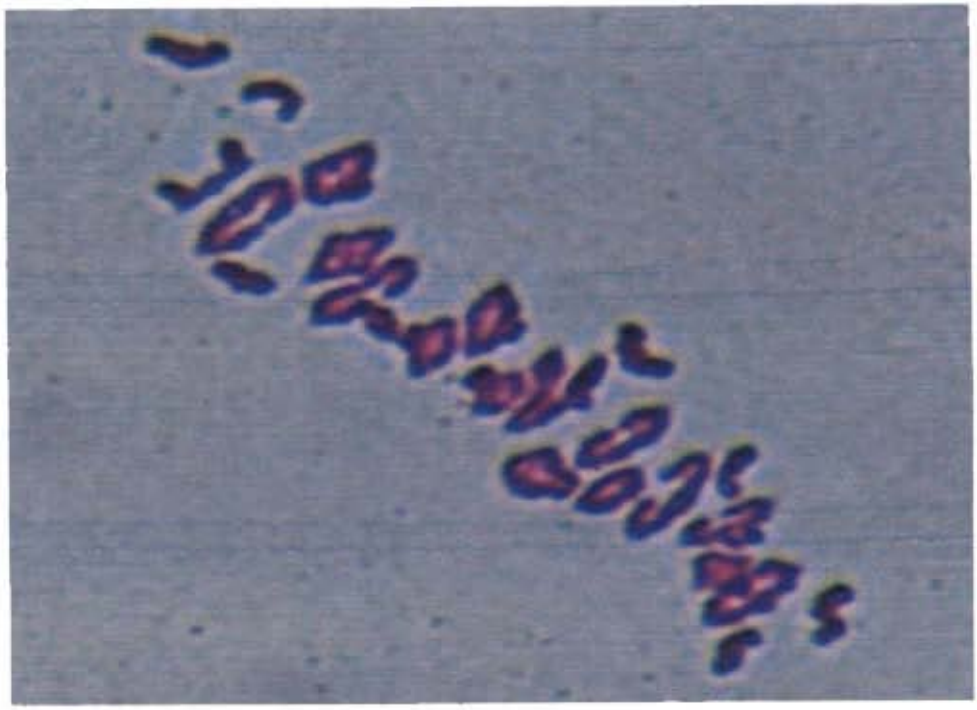


Plate 3: Monosomic F_1 plant with 19 bivalents and 3 univalents at first meiotic metaphase

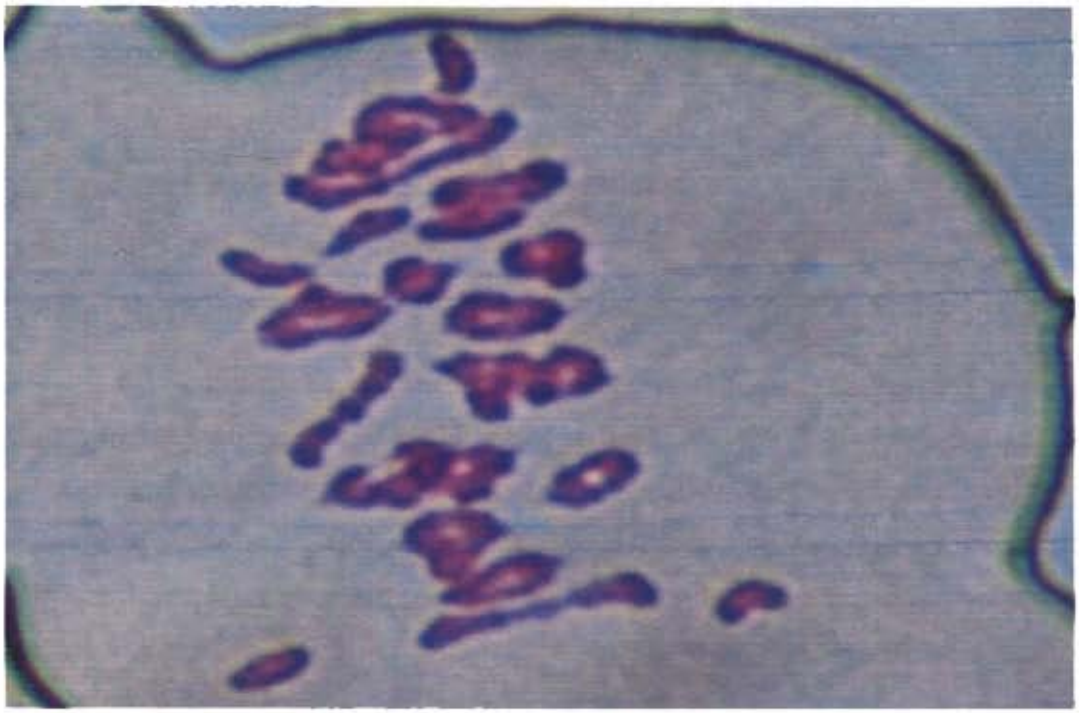


Plate 4: The F_1 plant of cross Agra Local x Sel. 2429 showing isochromosome at first meiotic metaphase

(ii) Lal Bahadur x Sel. 2429

(iii) WL 711 x Sel. 2429

(iv) HW 2043 (*Lr*19)x Sel. 2429

(v) HW 2003 (*Lr*24) x Sel. 2429

(vi) Agra Local x Sel. 4805-1

(vii) NI 5439 x Sel. 4805-1

(viii) All 21 monosomics of Chinese Spring x Sel. 2429.

All the genetic stocks were raised in pots and field plots and crosses were made following the 'go-go' method. Two earheads per female plants in each cross were emasculated and pollinated with pollens of respective male parent to obtain desired number of seeds for the experiment. The seeds from the above crosses were sown and all F_1 hybrid plants except those derived from monosomic x Sel. 2429 were strictly selfed to get F_2 seeds. The F_1 monosomic plants in each cross involving Sel. 2429 (cross no. viii) were cytologically identified and seeds from each cross were harvested separately and the F_2 generations were tested with rust pathotypes in glass house. Some of the F_1 plants derived from the crosses i, ii, iii, vi and vii were backcrossed with the susceptible parents in order to get the respective BC_1 F_1 populations.

3.2.3 Multiplication of inoculum

The pure nucleus inoculum of urediospores of selected pathotypes of *P. graminis* f. sp. *tritici* and *P. triticina* was obtained from DWR, Regional Station, Flowerdale, Shimla. The uredo-inoculum of each pathotype was multiplied on Agra Local (Plate 5), a susceptible variety, in the glasshouse following the standard procedure.

Soil

A well friable soil mixture was placed in 4" plastic pots for sowing the seeds. The plastic pots were selected for sowing because they are durable, easy to carry and retain moisture for a longer period.

Sowing

Seedlings were raised in a separate unit of the glasshouse, which is spore-proof and provide aseptic conditions. Approximately, 10-12 properly spaced seeds were sown

Plate 5: Inoculum multiplication on Agra Local seedlings

at ½" depth in pots. In order to get uniform germination, seeds were dibbled at equal depth with embryo downward.

Inoculation

Ten days old seedlings, with the primary leaf completely open, were selected for inoculation. The leaves were sprayed with tap water before inoculation and rubbed between moistened fingers to remove the thin layer of cuticle wax to provide uniform layer of moisture on leaf surface. The inoculum was applied on the leaf surface through spatula as per the procedure given by Joshi *et al.* (1988). The wet flat-end of lancet needle was loaded with urediospores and applied gently on the abaxial side of leaf as uniformly as possible. After inoculation, the seedlings were sprayed with tap water using sprayer to make fine dew on the leaf surface and kept in the moist glass chamber under diffused light. The inoculated trays were taken out after 48 h of incubation and kept on the benches in the glasshouse. The temperature of glasshouse varied between 15 to 25^{0}C. Strict precautionary measures were adopted to avoid contamination and ensure purity of pathotypes by inoculating one pathotype at a time. Hands were washed with lysol and disinfected by rectified spirit before and after inoculation. The incubated pots with different pathotypes were kept in separate isolated chambers and their purity was tested on differential sets before their utilisation.

Collection of dust

The urediospore dust was collected on butter paper after 10-15 days of incubation and stored for further testing. The dust of a single pathotype was collected at a time to avoid contamination. Clipping of secondary and tertiary leaves was done regularly just after taking the dust. Clipping was done to harness more dust from the seedlings.

Storage of inoculum

The dust was collected on a butter paper by tapping the infected leaves with the help of lancet needle. The butter paper was then folded to make the packet. Type of rust, name of pathotype and date of collection was marked on each packet. Such packets were kept on the glasshouse bench for 24 h for air drying and then stored in a refrigerator at 5^{0}C.

3.2.4 Grading of host/pathogen interaction

The infection types were recorded according to standard procedure. The differential sets were recorded after 15 days of incoulation when the disease developed. The grading system is mentioned below (plates 6 and 7).

I. Resistant

0 (naught)	=	Immune	No uredia nor any other symptoms
; (fleck)	=	Nearly immune	No uredia but hypersensitive fleck is present
0;(naught fleck)	=	Very resistant	No uredia plus hypersensitive flecks are present
1	=	Resistant	Uredia minute surrounded by distinct necrotic areas
2	=	Moderately resistant	Uredia small or medium with chlorosis or necrosis

II. Susceptible

3	=	Moderately susceptible	Uredia medium in size usually without necrotic or chloretic areas
4	=	Susceptible	Pustules large, often coalescing, no necrosis but chlorosis may be present under favourable conditions.

III. Mesothetic

X	=	Mesothetic	Uredia variable, including all infection types on the same leaf, no mechanical separation possible.

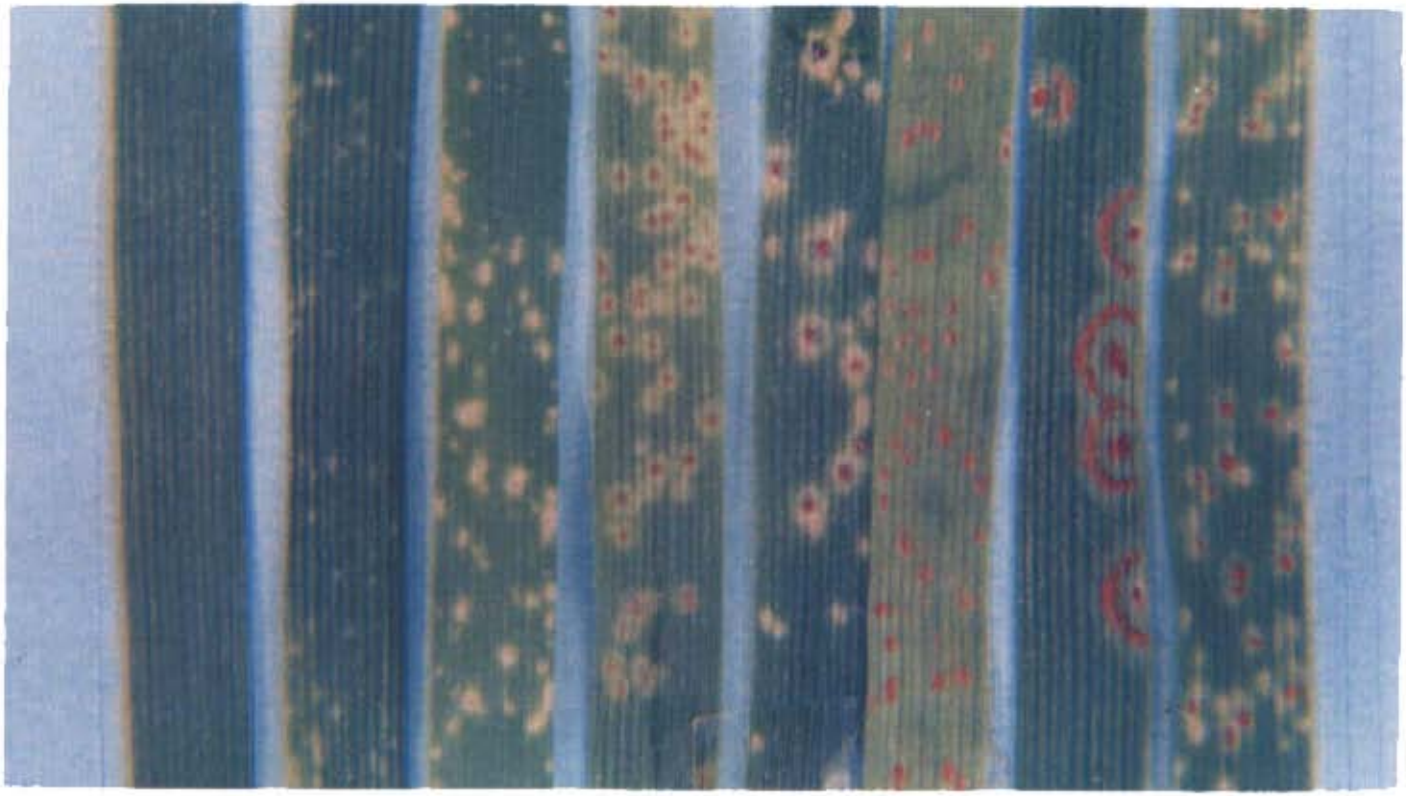
Plus (+) and minus (-) signs were used to indicate minor variations in the infection types. The sign '+' indicates infection type higher, while '-' indicates lower than the normal categories. Symbol 'N' was used to record necrosis of the leaf.

3.2.5 Screening for rust resistance at seedling stage

3.2.5.1 Multipathotype test

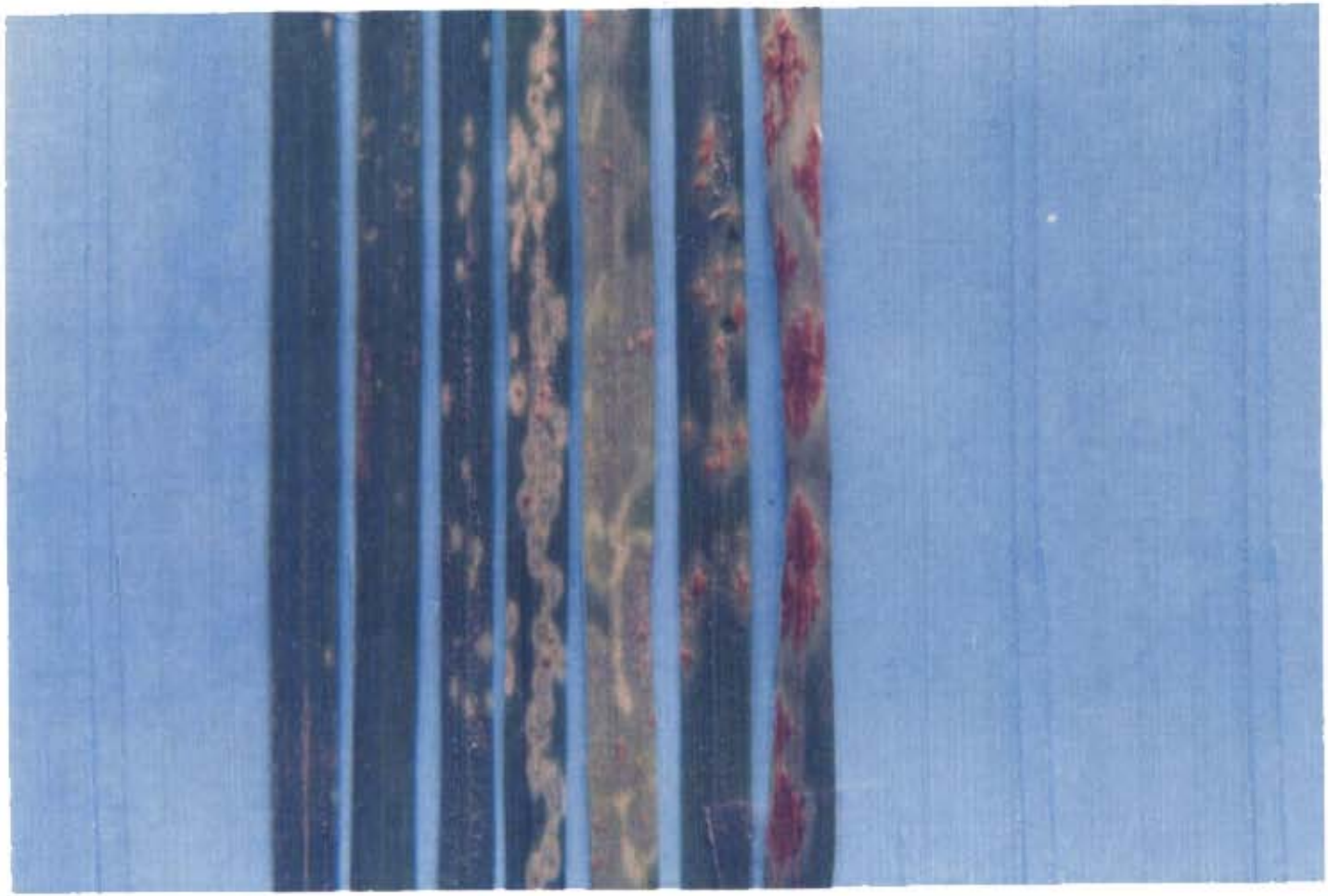
The resistant stocks Sel. 2429 and Sel. 4805-1 along with the background susceptible cultivars viz., WL 711, NI 5439 and Lal Bahadur, universal susceptible check Agra Local and differentials were tested with many pathotypes of *P. triticina* and

Plate 6: Infection types (ITs) of *Puccinia recondita*



0 0; ; 1 2 3 4 X

Plate 7: Infection types (ITs) of *Puccinia graminis* f. sp. *tritici*



0 0; ; 1 2 3 4

P. graminis f. sp. *tritici* (Table 7). This multipathotype test provided information on effectiveness of the resistance in interspecific derivatives Sel. 2429 and Sel. 4805-1.

3.2.5.2 Testing of segregating population and analysis

The mode of inheritance and the number of resistance genes were determined by testing F_1 and F_2 generation of crosses involving Sel. 2429, Sel. 4805-1 and susceptible checks viz., WL 711, NI 5439, Agra Local and Lal Bahadur at seedling stage (Plates 8 and 9). The F_2 results were interpreted according to various Mendelian ratios as mentioned below:

No. of gene(s)	Nature of gene(s)	F_2 ratio R : S	F_3 ratio R : Seg : S
One	Dominant	3 : 1	1 : 2 : 1
	Recessive	1 : 3	1 : 2 : 1
Two	Dominant	15 : 1	7 : 8 : 1
	Complementary	9 : 7	1 : 8 : 7
	1 dominant + one rccessive	13 : 3	7 : 8 : 1
	Recessive	7 : 9	7 : 8 : 1
	Recessive duplicate	1 : 15	1 : 8 : 7
Three	Dominant	63 : 1	37 : 26 : 1
	2 dominant + 1 recessive	61 : 3	37 : 26: 1
	1 dominant + 2 recessive	55 : 9	37: 26 : 1

To determine the identity of possible resistance genes, the F_1 and F_2 population of crosses involving Sel. 2429 and genetic stocks carrying known resistance genes were tested with a selected pathotype avirulent on Sel. 2429. The appearance of susceptible segregants in F_2 generation would suggest that the resistance gene in the tested stock is different to that of present in the known genetic stock.

For locating the gene(s) for resistance on specific chromosome of Sel. 2429, the F_1's of all 21 different CS monosomics x Sel. 2429 and F_2 population derived from cytologically identified monosomic F_1 of respective crosses were tested with selective

Plate 8: Seedlings inoculated with leaf rust race 77-5

Plate 9: Seedlings inoculated with stem rust race 40A

leaf rust pathotype 77-5 at seedling stage. Specific chromosome carrying resistance gene(s) was identified by 'critical line analysis'. The F_2 population designed from non-critical monosomic lines could exhibit a normal disomic inheritance while the F_2's of critical line (chromosome carrying resistance gene) would lead to a significantly distorted segregation ratio from normal ratio.

3.2.5.3 Scoring of morphological markers

The resistant lines Sel. 2429 and Sel. 4805-1 were scored for their morphological markers to find out their inheritance pattern and also to find any linkge between these markers and rust resistance. The Sel. 2429 derivatives were scored for leaf sheath pubescence, whereas Sel. 4805-1 derivatives were scored for leaf and sheath pubescence.

3.2.6 Statistical analysis

3.2.6.1 F_2 segregation for detection of inheritance pattern

The chi-square (χ^2) test for testing goodness of fit, as described by Panse and Sukhatme (1976) was used to establish validity of observations in relation to expected values in segregating population on the basis of Mendelian segregation.

$$\chi^2 = \frac{\Sigma (O-E)^2}{E}$$

Where

Σ = Summation

O = Observed frequency

E = Expected frequency

For complete agreement with the hypothetical distribution, the value of χ^2-score will be zero, but chance deviation would occur.

3.2.6.2 Detection of linkage

If two characters segregate independently, the F_2 is expected to show 9:3:3:1 segregation ratio. The χ^2 value for the four classes segregation is calculated by taking deviation from independent assortment. This would represent χ^2 due to linkage with 3 d.f. if segregation of individual characters is strictly as per expectation. But this is

hardly ever the case and the deviation from the theoretical ratios of individual characters as well as experimental error also contribute to the calculated value of χ^2 deviating from the above (Panse and Sukhatme, 1985). So their contributions were removed from the gross value of χ^2 to get χ^2 for linkage alone, which will have 1 d.f.

Thus total χ^2 for 9:3:3:1 ratio with 3 d.f. was partitioned into χ^2 due to segregation of plants for the first genetic marker (designated as χ^2_A) into 3 dominant forms : 1 recessive form with 1 d.f. : χ^2 for segregation of the other genetic marker (designated as χ^2_B) into 3 dominant forms : 1 recessive form with 1 d.f. and χ^2 for linkage (denoted as χ^2_L) with 1 d.f. The three values of χ^2 were calculated according to the procedure given by Mather (1951) as under :

$$\chi^2_A = \frac{[a_1 + a_2 - 3\,(a_3 + a_4)]^2}{3\,n}$$

$$\chi^2_A = \frac{[a_1 + a_3 - 3\,(a_2 + a_4)]^2}{3\,n}$$

$$\chi^2_A = \frac{[a_1 + 3a_2 - 3a_3 + 9a_4)]^2}{9\,n}$$

Where a_1, a_2, a_3 and a_4 are observed frequencies of four classes expected in ratio 9:3:3:1 and n is the number of individuals in the family.

In case where one marker trait was expected to segregate in 3 : 1 ratio but the other marker trait was governed by two genes which segregated in 15:1 ratio, whereas the F_2 was expected to segregate in the ratio of 45:15:3:1. The χ^2 value were calculated using the formulae :

For 3:1 ratio :

$$\chi^2_X = \frac{[a_1 + 3a_2 - a_3 + 3a_4)]^2}{3n}$$

For 15 : 1 ratio :

$$\chi^2_v = \frac{[a_1 + a_2 - 15a_3 + 15a_4)]^2}{15n}$$

and

$$\chi^2_L = \frac{[a_1 + 3a_2 - 15a_3 + 45a_4)]^2}{45n}$$

In case of disturbed segregation, where one of the two factors deviates from the expected 3 : 1 ratio, the following formula was applied for detection of linkage (Mather, 1951).

$$\chi^2_L = \frac{(a_1a_4 - a_2a_3 - \frac{1}{2}n)^{2n}}{(a_1 + a_2)(a_3 + a_4)(a_1 + a_3)(a_2 + a_4)}$$

The calculated χ^2s were compared with the Table values for 1 d.f. The null hypothesis for linkage test assumes that there is no linkage, i.e. the marker traits are segregating by independent assortment. Existence of linkage was demonstrated if χ^2_L was significant and χ^2_A and χ^2_B are non-significant.

3.2.6.3 Comparison of mean number of bivalents and univalents in different disomic and monosomic plants

Let $\bar{X}$ be the mean of a sample of size N, and Z, the standard deviation. It is to be tested whether $\bar{X}$ is the same as population mean (or hypothetical mean) μ (Chandel, 2004).

$$\text{Here, } t = \frac{\bar{X} - \mu}{\left(\frac{S}{\sqrt{N}}\right)} = \frac{\bar{X} - \mu}{\text{S.e. of X}} \text{ , where, } S = \sqrt{\frac{S(X - \bar{X})^2}{N - 1}}$$

D.F. = N - 1

The significant value of t indicates that the population from which the sample has been drawn is not the same as the hypothetical population with mean μ.

3.2.6.4 Comparison of mean number of bivalents in monosomic and disomic plants

In experimental work generally it becomes necessary to test whether the two samples differ from one-another significantly in their means, or whether they may be regarded as belonging to the same population.

Suppose, we have got two samples, $X_{11}, X_{12}, \ldots X_{1n_1}$ and $X_{21}, X_{22}, \ldots X_{2n_2}$. The following statistics will be calculated for testing the significance of the difference between their means.

$$X_1 = \frac{1}{n_1} \Sigma X_1, \qquad S_1^2 = \frac{1}{n_1 - 1} \Sigma (X_1 - X_1)^2$$

$$X_2 = \frac{1}{n_2} \Sigma X_2 \qquad S_2^2 = \frac{1}{n_1 - 1} \Sigma (X_2 - X_2)^2$$

$$\sigma_c^2 = \frac{\Sigma (X_1 - X_1)^2 + \Sigma (X_2 - X_2)^2}{(n_1 - 1) + (n_2 - 1)} \qquad \ldots\ldots\ldots (1)$$

$$\text{also } \sigma_c^2 = \frac{(n_1 - 1) S_1^2 + (n_2 - 1) S_2^2}{(n_1 - 1) + (n_2 - 1)} \qquad \ldots\ldots\ldots (2)$$

Where S_1^2 and S_2^2 are the estimated variances from two samples and σ_c^2 is a pooled estimate of the population variance which is estimated by pooling the sums of square $\Sigma (X_1 - \bar{X}_1)^2$ and $\Sigma (X_2 - \bar{X}_2)^2$ and then dividing by the total number of degrees of freedom $[(n_1 - 1) + (n_2 - 1)]$ contributed by the two samples, as given by equation (1). It can also be obtained by taking the weighted average of the two sample variances S_1^2 and S_2^2, weighted by the corresponding degrees of freedom $(n_1 - 1)$ and $(n_2 - 1)$ as given by equation (2).

Now, t is given by the following equation

$$t = \frac{X_1 - X_2}{\sqrt{\sigma_c^2 \left(\frac{1}{n_1} + \frac{1}{n_2} \right)}} \qquad \ldots\ldots\ldots (3)$$

$$\text{D.F.} = (n_1 - 1) + (n_2 - 1) \qquad \ldots\ldots (4)$$

The denominator of the expression given in equation (3) is the standard error of $(\bar{X}_1 - \bar{X}_2)$. It is something different from that obtained in large samples.

EXPERIMENTAL RESULTS

The present investigation was carried out to identify *T. militinae* and *Ae. speltoides* derived rust resistance gene(s) in common wheat. Studies were conducted to establish the identity of resistance gene(s), to localize the identified gene(s) on specific chromosomes and to study linkages with morphological traits. Results on genetic and cytological analysis and rust observations are presented in this chapter.

4.1 Multi pathotype test

The rust resistant genetic stock Sel.2429 and Sel.4805-1, the parental lines, NI5439, WL711, Lal Bahadur, *T. militinae* and *Ae. speltoides* and susceptible check Agra Local were tested with different leaf rust (77-5, 77-6 and 77-7) and stem rust (40A and 117-6) pathotypes individually at seedling stages under glass house conditions. The result obtained for multipathotype tests are presented in Table 7. The respective differential sets included in each experiment and their infection type (IT) are presented in Tables 5 and 6.

4.1.1 Leaf rust resistance

4.1.1.1 Sel. 2429

Sel. 2429 showed resistant reaction in seedling stage to all the three leaf rust pathotypes included in the study (Table 7). The range of infection type recorded was '0;' to ';1'. The parental genotype *Ae. speltoides* showed resistant reaction (IT = '0') while Agra Local exhibited susceptible reaction (IT = 33^+) to all the pathotypes tested.

4.1.1.2 Sel.4805-1

The multipathotype test of Sel.4805-1 and its parental lines tested with three leaf rust pathotypes are presented in Table 7. Sel. 4805-1 showed resistant raction (IT : '0;' to ';') in seedling stage to the selected pathotypes mentioned above. The parental genotypes, *T. militinae* and WL 711 exhibited immune (IT : '0;') and susceptible reactions (IT : 3^+ to 33^+), respectively. The susceptible check Agra Local showed infection type of 3^+ to 4 to all the pathotypes used in the study.

Table 7. **Infection types of selected pathotypes of *P. recondita* f. sp. *tritici* and *P. graminis* f. sp. *tritici* on materials under investigation**

Genotype	Pathotype				
	Leaf Rust			Stem rust	
	77-5	77-6	77-7	40A	117-6
Sel. 4805-1	;1	;	0;	;1	;
Sel 2429	0;	0;	;1	;	0;
T. militinae	;	0;	;	0;	0;
Ae. speltoides	0;	0;	;	0;	0;
NI 5439	3+	3+	3+	3+	3+
Agra Local	3+	3+	3+	4	4
Lal Bahadur	3+	3+	3+	3+	3
WL 711	3+	3+	3+	3+	3+

4.1.2 Stem rust resistance

4.1.2.1 Sel.2429

Sel.2429 showed resistant reaction in seedling stage to both stem rust pathotypes included in the study (Table 7). The range of infection on types recorded was '0;' to ';1'. The parental lines *Ae. speltoides* and Agra Local exhibited resistant (IT = 0;) and susceptible (IT = 33^{+}) reactions, respectively.

4.1.2.2 Sel.4805-1

The results of testing of *T. militinae* derivative Sel. 4805-1 with two stem rust pathotypes viz., 40A and 117-6 are presented in Table 7. Genetic stock Sel.4805-1 and one of its parent *T. militinae* exhibited resistant reaction (IT = '0;'), while NI5439 involved in the pedigree showed susceptible reaction (IT : 3^{+}). Agra Local exhibited high degree of susceptibility (IT : 3^{+} to 4) in seedling stage against all the races included in the present study.

4.2 Inheritance studies

The number and nature of rust resistance gene(s) in genetic stocks can be conclusively determined by genetic analysis of different filial generations.

4.2.1 Inheritance of leaf rust resistance

4.2.1.1 Selection 2429

The inheritance studies for leaf rust resistance to the most virulent rust pathotypes viz. 77-5, 77-6 and 77-7 were carried out at seedling stage and results are presented in Tables 8, 9, 10 and Sel. 2429 was crossed with susceptible genotypes Agra Local, Lal Bahadur and WL 711. The parents F_1, F_2 and BC_1 populations were tested with the above mentioned three most virulent Indian leaf rust pathotypes.

The resistant genetic stock Sel. 2429 and all F_1 plants of the cross Agra Local x Sel. 2429 exhibited resistance to the selected pathotypes, 77-5 and 77.7. The infection type obtained in F_1 seedlings determined the dominance of resistance. The F_2 population derived from the cross AL X Sel. 2429 segregated into 172 resistant and 58 susceptible plants when tested with 77-5. While against 77-7, 147 F_2 segregants were resistant and 39 susceptible. The observed data in both the crosses fit well into expected ratio of 3

Table 8. Segregation of seedlings in F_2 and BC_1F_1 generations of different crosses tested with leaf rust pathotype 77-5

Parent/cross	Parent/ generation	Number of seedlings			Expected Ratio	χ^2	P- value
		Resistant (IT:0-2)	Susceptible (IT:3-4)	Total			
Agra Local (AL)	P_1	0	33	33			
Sel. 2429	P_2	29	0	29			
AL/Sel. 2429	F_1	31	0	31			
	F_2	172	58	230	3R:1S	0.006	0.95-0.90
AL/Sel. 2429//AL	BC_1F_1	15	18	33	1R:1S	0.273	0.70-0.50
Lal Bahadur (LB)	P_1	0	23	23			
Sel. 2429	P_2	35	0	35			
LB/Sel. 2429	F_1	29	0	29			
	F_2	89	35	124	3R:1S	0.688	0.50-0.30
LB/Sel. 2429//LB	BC_1F_1	23	27	50	1R:1S	0.320	0.70-0.50
WL 711	P_1	0	28	28			
Sel. 2429	P_2	37	0	37			
WL 711 x Sel. 2429	F_1	39	0	39			
	F_2	168	48	216	3R:1S	0.889	0.50-0.30
WL711/Sel. 2429//WL 711	BC_1F_1	33	28	61	1R:1S	0.409	0.70-0.50
Pooled over three crosses	F_2	429	141	570	3R:1S	0.021	0.90-0.80
	BC_1F_1	71	73	144	1R:1S	0.028	0.90-0.80

($\chi^2 = 3.841$; P = 0.05)

Table 9: Segregation of seedlings in F_2 and BC_1F_1 generations of different crosses tested with leaf rust pathotype 77-6

Parent/cross	Parent/ generation	Number of seedlings			Expected Ratio	χ^2	P- value
		Resistant (IT:0-2)	Susceptible (IT:3-4)	Total			
Agra Local (AL)	P_1	0	32	32			
Sel. 2429	P_2	31	0	31			
AL/Sel. 2429	F_1	0	26	26			
	F_2	19	67	86	1R:3S	0.388	0.70-0.50
AL/Sel. 2429//AL	BC_1F_1	22	27	49	1R:1S	0.510	0.50-0.30
Lal Bahadur (LB)	P_1	0	28	28			
Sel. 2429	P_2	29	0	29			
LB/Sel. 2429	F_1	0	37	37			
	F_2	25	90	115	1R:3S	0.652	0.50-0.30
LB/Sel. 2429//LB	BC_1F_1	18	14	32	1R:1S	0.500	0.50-0.30
WL 711	P_1	0	42	42			
Sel. 2429	P_2	38	0	38			
WL 711 x Sel. 2429	F_1	0	40	40			
	F_2	29	93	122	1R:3S	0.098	0.90-0.80
WL711/Sel. 2429//WL 711	BC_1F_1	26	31	57	1R:1S	0.439	0.70-0.50
Pooled over three crosses	F_2	73	250	323	1R:3S	0.992	0.50-0.30
	BC_1F_1	66	72	138	1R:1S	0.261	0.70-0.50

($\chi^2 = 3.841$; P = 0.05)

Table 10. Segregation of seedlings in F_2 and BC_1F_1 generations of different crosses tested with leaf rust pathotype 77-7

Parent/cross	Parent/ generation	Number of seedlings			Expected Ratio	χ^2	P- value
		Resistant (IT:0-2)	Susceptible (IT:3-4)	Total			
Agra Local (AL)	P_1	0	18	18			
Sel. 2429	P_2	29	0	29			
AL/Sel. 2429	F_1	36	0	36			
	F_2	147	39	186	3R:1S	1.613	0.30-0.20
AL/Sel. 2429//AL	BC_1F_1	29	35	64	1R:1S	0.563	0.50-0.30
Lal Bahadur (LB)	P_1	0	28	28			
Sel. 2429	P_2	42	0	42			
LB/Sel. 2429	F_1	31	0	31			
	F_2	115	49	164	3R:1S	2.081	0.20-0.10
LB/Sel. 2429//LB	BC_1F_1	52	59	111	1R:1S	0.441	0.70-0.50
WL 711	P_1	0	19	19			
Sel. 2429	P_2	24	0	24			
WL 711 x Sel. 2429	F_1	35	0	35			
	F_2	123	48	171	3R:1S	0.859	0.50-0.30
WL711/Sel. 2429//WL 711	BC_1F_1	46	55	101	1R:1S	0.802	0.50-0.30
Pooled over three crosses	F_2	385	136	521	3R:1S	0.339	0.70-0.50
	BC_1F_1	127	149	276	1R:1S	1.754	0.20-0.10

($\chi^2 = 3.841$; P = 0.05)

resistant : 1 susceptible in F_2 generations with χ^2 value 0 .006 (P value : 0.95 - 0.90) for race 77-5 and χ^2 of 1.613 (P value : 0.30 - 0.20) for race 77-7 (Table 8 and 10). In BC_1 population 15 plants were resistant, while 18 susceptible, when tested with 77-5, however against the pathotype 77-7, 29 plants were resistant while 35 susceptible fitting well into 1R : 1S ratio supporting the hypothesis proposed in the F_2 generation.

Similarly, the F_1 hybrids of the crosses Lal Bahadur x Sel. 2429 and WL 711 x Sel. 2429 showed resistant reaction when tested with 77-5 and 77-7 pathotypes indicating the dominance of resistance. In F_2 population of the former cross 89 seedlings were categorised as resistant and 35 susceptible, whereas in latter cross 168 segregants showed resistance and 48 plants exhibited susceptibility when tested with leaf rust pathotype 77-5. The F_2 seedlings in both the above mentioned crosses, when subjected to 77-7 pathotype, 115 seedlilngs were resistant and 49 susceptible in LB x Sel. 2429, whereas in the cross WL 711 x Sel. 2429 123 F_2 seedling showed resistant reaction and 48 exhibited susceptibility. The observed frequency of resistant and susceptible seedlings in both the crosses fit well into expected ratio of 3R : 1S indicating that the resistance in Sel. 2429 is controlled by a single dominant gene against the leaf rust pathotypes 77-5 and 77-7. The value of χ^2 calculated in both the crosses were 0.688 and 0.889 for the pathotypes 77-5 and 2.081 and 0.859 for race 77-7 respectively. In pooled analysis of F_2 data for all three crosses against both the pathotypes, 77-5 and 77-7 also depicted a non-significant χ^2 value 0.021 (P : 0.90 - 0.80) for race 77-5 and χ^2 : 0.339 (P : 0.70 - 0.50) for 77-7 for the expected ratio of 3R : 1S.

The F_1 plants were backcrossed with the respective susceptible parents to get BC_1F_1 generation. The BC_1F_1 population in both the crosses showed a similar segregation pattern with goodness of fit for the expected frequency of 1 resistant : 1 susceptible for both the races 77-5 and 77-7. The value of χ^2 calculated were 0.320 and 0.409 for race 77-5 and 0.565, 0.441 and 0.802 for race 77-6 respectively. The non-significant value for χ^2 supported the hypothesis proposed in F_2 generation. Pooled analysis of BC_1-F_1 data also fit well into an expected ratio of 1R : 1S further supporting the results of genetic analysis of F_2 data that the resistance to 77-5 and 77-7 pathotypes is indeed governed by a single dominant gene (Table 8 & 10)

The F_1 plants of the crosses, namely, AL x Sel 2429, LB x Sel. 2429 and WL 711 x Sel. 2429 exhibited susceptible reaction (IT 3^+) to 77-6 pathotype indicating that

susceptibility is dominant over resistant. The F_2 population derived from the crosses segregated into 19 resistant and 67 susceptible plant in AL x Sel.2429; 25 resistant and 90 susceptible in LB x Sel. 2429 and 29 resistant and 93 susceptible in WL 711 x Sel. 2429 all fitting well into an expected ratio of 1R : 3S with non-significant χ^2 value of 0.388, 0.652 and 0.098 respectively. The data in F_2 generation was pooled over the crosses and the genetic analysis revealed that the resistance in Sel. 2429 against 77-6 pathotypes is governed by a recessive gene. The F_1 plants were backcrossed with the respective susceptible parents in above mentioned crosses to get BC_1F_1 progenies. All the BC_1F_1 populations were tested with 77-6 which showed a segregation pattern corresponding to 1 resistant : 1 susceptible ratio. The value of χ^2 for the data which were non-significant supported well the above proposed hypothesis based on F_2 genetic analysis that resistance in Sel. 2429 is controlled by a single recessive gene in respect to 77-6 pathotype of leaf rust. The pooled analysis of BC_1F_1 data over the crosses also supported the inference drawn on the basis of F_2 analysis (Table 9).

4.2.1.2 Selection 4805-1

To study the inheritance of resistance in Sel. 4805-1 against leaf rust pathotypes, 77-5, 77-6 and 77-7 the seedlings in F_1, F_2 and BC_1F_1 generations of the crosses Agra Local x Sel. 4805-1 and NI5439 x Sel. 4805-1 were tested with the above mentioned selected pathotypes (Table 11, 12 and 13) under glass house conditions.

The F_1 plants in both the crosses showed resistant reaction against 77-5 pathotype of leaf rust indicating that resistance is dominant. The F_2 populations derived from each cross segregated into resistant and susceptible plants. Out of 240 F_2 plants in AL x Sel. 4805-1, 177 were resistant and 63 susceptible when tested with this pathotype. Similarly, the F_2 population in the cross NI 5439 x Sel. 4805-1 segregated into resistance (228 plants) and susceptibility (83 plants). The observed frequency of the F_2 segregants fit well into the expected ratio of 3 resistant : 1 susceptible with non significant chi-square values of 0.200 and 0.473 in both the crosses, namely AL x Sel. 4805-1 and NI 5439 x Sel. 4805-1 respectively. These results indicated that resistance in Sel. 4805-1 is controlled by a single dominant gene. The pooled F_2 analysis of both the crosses also depicted the similar ratio of 3R : 1S with non-significant χ^2 value of 0.659 (P : 0.50-0.30) for the expected ratio indicating the goodness of fit.

Table 11. Mode of segregation for resistance to leaf rust pathotype 77-5 in Sel. 4805-1 at seedling stage

Parent/cross	Parent/ generation	Number of seedlings			Expected Ratio	χ^2	P- value
		Resistant (IT:0-2)	Susceptible (IT:3-4)	Total			
Agra Local (AL)	P_1	0	32	32			
Sel. 4805-1	P_2	35	0	35			
AL/Sel. 4805-1	F_1	27	0	27			
	F_2	177	63	240	3R:1S	0.200	0.70-0.50
AL/Sel. 4805-1//AL	BC_1F_1	68	58	126	1R:1S	0.794	0.50-0.30
NI 5439	P_1	0	49	49			
Sel. 4805-1	P_2	37	0	37			
NI 5439/Sel.4805-1	F_1	33	0	33			
	F_2	228	83	311	3R:IS	0.473	0.50-0.30
NI5439/Sel. 4805-1//NI5439	BC_1F_1	49	60	109	1R:1S	1.110	0.30-0.20
Pooled over two crosses	F_2	405	146	551	3R:1S	0.659	0.50-0.30
	BC_1F_1	117	118	235	1R:1S	0.004	0.95-0.90

($\chi^2 = 3.841$; P = 0.05)

Table 12. Mode of segregation for resistance to leaf rust pathotype 77-6 in Sel. 4805-1 at seedling stage

Parent/cross	Parent/ generation	Number of seedlings			Expected Ratio	χ^2	P- value
		Resistant (IT:0-2)	Susceptible (IT:3-4)	Total			
Agra Local (AL)	P_1	0	25	25			
Sel. 4805-1	P_2	23	0	23			
AL/Sel. 4805-1	F_1	26	0	26			
	F_2	115	79	194	9R:7S	0.723	0.50-0.30
AL/Sel. 4805-1//AL	BC_1F_1	12	31	43	1R:3S	0.194	0.70-0.50
NI 5439	P_1	0	17	17			
Sel. 4805-1	P_2	19	0	19			
NI 5439/Sel.4805-1	F_1	14	0	14			
	F_2	87	63	150	9R:7S	0.187	0.70-0.50
NI5439/Sel. 4805-1//NI5439	BC_1F_1	6	21	27	1R:3S	0.111	0.80-0.70
Pooled over two crosses	F_2	202	142	344	9S:7S	0.854	0.50-0.30
	BC_1F_1	18	52	70	1R:3S	0.019	0.90-0.80

($\chi^2 = 3.841$; P = 0.05)

Table 13. Mode of segregation for resistance to leaf rust pathotype 77-7 in Sel.4805-1 at seedling stage

Parent/cross	Parent/ generation	Number of seedlings			Expected Ratio	χ^2	P- value
		Resistant (IT:0-2)	Susceptible (IT:3-4)	Total			
Agra Local (AL)	P_1	0	25	25			
Sel. 4805-1	P_2	24	0	24			
AL/Sel. 4805-1	F_1	20	0	20			
	F_2	111	35	146	3R:1S	0.082	0.80-0.70
AL/Sel. 4805-1//AL	BC_1F_1	29	24	53	1R:1S	0.472	0.50-0.30
NI 5439	P_1	0	21	21			
Sel. 4805-1	P_2	18	0	18			
NI 5439/Sel.4805-1	F_1	27	0	27			
	F_2	98	32	130	3R:1S	0.010	0.95-0.90
NI5439/Sel. 4805-1//NI5439	BC_1F_1	41	35	76	1R:1S	0.474	0.50-0.30
Pooled over two crosses	F_2	209	67	276	3R:1S	0.077	0.80-0.70
	BC_1F_1	70	59	129	1R:1S	0.938	0.50-0.30

($\chi^2 = 3.841$; P = 0.05)

A large number of BC_1F_1 plants derived from the F_1 x susceptible parents segregated into 1R : 1S ratio when tested with 77-5 pathotype. The values of χ^2 recorded were 0.794 in AL/Sel.4805-1//AL and 1.11 in NI 5439/Sel. 4805//NI5439. Pooled analysis of BC_1F_1 data also support the hypothesis proposed on the basis of F_2 analysis with non-significant χ^2 value of 0.004.

The plants in AL x Sel.4805-1 and NI 5439 x Sel.4805-1 crosses showed resistance, when tested with 77-7 pathotype of leaf rust indicating that resistance is dominant. The F_2 plants segregated into 111 resistant and 35 susceptible in the former cross whereas in latter cross 98 plants were resistant and 32 susceptible. These results fit well into the expected ratio of 3R : 1S with non-significant chi-square values of 0.082 and 0.010. The BC_1F_1 population derived from AL/Sel.4805-1//AL produced 29 resistant and 24 susceptible plants while 41 resistant and 35 susceptible plants were observed in NI 5439/Sel.4805-1//NI5439 derived population. The observed frequencies of resistant and susceptible plants fit well into an expected ratio of 1R : 1S with non-significant χ^2 values of 0.472 and 0.474 respectively. The pooled analysis over two crosses in F_2 generation also indicated that resistance is governed by a single dominant gene as the pooled observed frequencies of resistant and susceptible plants fit well into 3R : 1S ratio with non-significant χ^2 value of 0.079 (P = 0.80 - 0.70). Similarly, the pooled BC_1F_1 population gave a good fit to 1R : 1S ratio with χ^2 value of 0.938 supporting the F_2 hypothesis.

The F_1 seedlings in both the crosses, AL x Sel.4805-1 and NI5439 x Sel.4805-1 showed resistance to leaf rust pathotype 77-6 (Table 12). In the cross AL x Sel. 4805-1, 115 plants were resistant while 79 susceptible. The observed frequencies fit well into expected ratio of 9R : 7S with non-significant χ^2 value of 0.723. These results indicated that the resistance is governed by two complementary dominant genes. The BC_1F_1 population of this cross segregated into 1R : 3S ratio (12 plants resistant and 31 plants susceptible) with non-significant χ^2 of 0.194 supporting the hypothesis proposed in F_2 analysis. Similarly, the F_1 plants in NI 5439 x Sel.4805-1 cross showed resistance to 77-6 pathotype of leaf rust indicating the dominance of resistance. The F_2 plants segregated into 87 resistant and 63 susceptible and the observed frequency fit well into 9R : 7S ratio with non-significant χ^2 value of 0.187. Twenty seven BC_1F_1 plants in the

cross NI 5439/Sel.4805-1//NI 5439 segregated into an expected ratio of 1R : 3S as there were plants observed. The non-significant χ^2 value of 0.111 proved the goodness of fit. The results of genetic analysis carried out in F_2 and BC_1-F_1 generations were also supported by the pooled analysis of F_2 (χ^2 value 0.854) and BC_1 F_1 data (χ^2 value of 0.019) over the crosses.

4.2.2 Inheritance of stem rust resistance

4.2.2.1 Selection 2429

The results obtained in the study of mode of inheritance of stem rust resistance in Sel. 2429 are presented in Tables 14 and 15. Three crosses namely, AL x Sel. 2429, LB x Sel. 2429 and WL 711 x Sel. 2429 were analysed genetically.

The F_1 plants in all the above mentioned crosses showed resistance to stem rust pathotypes 40A and 117-6. The reaction pattern in F_1's indicated that resistance is of dominant nature. Out of 206 F_2 plants in the cross AL x Sel. 2429, 147 were resistant and 59 susceptible. The observed frequency fit well into an expected ratio of 3R : 1S with non-significant χ^2 value of 1.456 against the 117-6 pathotype. The results indicated that the resistance is governed by a single dominant gene in Sel.2429. These results were well supported by the segregation in BC_1F_1 as 46 plants were observed to be resistant and 40 susceptible. The frequency in two categories fit well into a ratio oif 1R : 1S with non-significant χ^2 value of 0.419 (P = 0.70 - 0.50).

One hundred and thirty six F_2 plants in the cross LB x Sel. 2429 were resistant while 38 susceptible. Similarly in the cross WL 711 x Sel. 2429, 97 plant produced resistant reaction whereas 28 showed susceptibility. The observed frequencies in resistant and susceptibility categories in F_2 population of both the crosses fit well into an expected ratio of 3R : 1S with non significant χ^2 value of 0.927 and 0.451 respectively. This indicated that rersistance to 117-6 in Sel.2429 is controlld by a single dominant gene. The BC_1F_1 generations derived from these crosses segregated into an expected ratio of 1R : 1S as there were 21 resistant and 17 susceptible plants in LB / Sel.2429//LB cross and 22 resistant and 14 susceptible plants in WL711/Sel.2429//WL711 with non significant χ^2 values of 0.421 and 1.778 respectively. The results of BC_1-F_1 analysis supported the F_2 hypothesis that the resistance in Sel.2429 is governed by a single dominant gene to the pathoype 117-6. The pooled analysis of F_2 data over the crosses

Table 14. Segregation of seedlings in F_2 and BC_1F_1 generations of different crosses tested with stem rust pathotype 40-A

Parent/cross	Parent/ generation	Number of seedlings			Expected Ratio	χ^2	P- value
		Resistant (IT:0-2)	Susceptible (IT:3-4)	Total			
Agra Local (AL)	P_1	0	25	25			
Sel. 2429	P_2	29	0	29			
AL/Sel. 2429	F_1	38	0	38			
	F_2	172	72	244	3R:1S	2.645	0.20-0.10
AL/Sel. 2429//AL	BC_1F_1	81	71	152	1R:1S	0.658	0.50-0.30
Lal Bahadur (LB)	P_1	0	32	32			
Sel. 2429	P_2	34	0	34			
LB/Sel. 2429	F_1	19	0	19			
	F_2	102	30	132	3R:1S	0.364	0.70-0.50
LB/Sel. 2429//LB	BC_1F_1	22	13	35	1R:1S	2.314	0.20-0.10
WL 711	P_1	0	26	26			
Sel. 2429	P_2	36	0	36			
WL 711 x Sel. 2429	F_1	30	0	30			
	F_2	145	59	204	3R:1S	1.673	0.20-0.10
WL711/Sel. 2429//WL 711	BC_1F_1	45	35	80	1R:1S	1.250	0.30-0.20
Pooled over three crosses	F_2	419	161	580	3R:1S	2.354	0.20-0.10
	BC_1F_1	148	119	267	1R:1S	3.150	0.10-0.05

($\chi^2 = 3.841$; P = 0.05)

Table 15. Segregation of seedlings in F_2 and BC_1F_1 generations of different crosses tested with stem rust pathotype 117-6

Parent/cross	Parent/ generation	Number of seedlings			Expected Ratio	χ^2	P- value
		Resistant (IT:0-2)	Susceptible (IT:3-4)	Total			
Agra Local (AL)	P_1	0	29	29			
Sel. 2429	P_2	24	0	24			
AL/Sel. 2429	F_1	34	0	34			
	F_2	147	59	206	3R:1S	1.456	0.30-0.20
AL/Sel. 2429//AL	BC_1F_1	46	40	86	1R:1S	0.419	0.70-0.50
Lal Bahadur (LB)	P_1	0	32	32			
Sel. 2429	P_2	39	0	39			
LB/Sel. 2429	F_1	28	0	28			
	F_2	136	38	174	3R:1S	0.927	0.50-0.30
LB/Sel. 2429//LB	BC_1F_1	21	17	38	1R:1S	0.421	0.70-0.50
WL 711	P_1	0	28	28			
Sel. 2429	P_2	41	0	41			
WL 711 x Sel. 2429	F_1	34	0	34			
	F_2	97	28	125	3R:1S	0.451	0.70-0.50
WL711/Sel. 2429//WL 711	BC_1F_1	22	14	36	1R:1S	1.778	0.20-0.10
Pooled over three crosses	F_2	380	125	505	3R:1S	0.017	0.90-0.80
	BC_1F_1	89	71	160	1R:1S	2.025	0.20-0.10

($\chi^2 = 3.841$; P = 0.05)

also fit well into 3R : 1S ratio with non significant χ^2 value of 0.017. The BC_1F_1 data was also subjected to pooled analysis which fit well into 1R : 1S ratio with non significant χ^2 value of 2.025.

The F_1 plants in all the three crosses AL x Sel. 2429, LB x Sel. 2429 and WL 711 x Sel. 2429 were tested for the pathotype 40A and showed resistance. The seedling reactions observed in F_1 indicated that the resistance is dominant. AL x Sel. 2429 derived population segregated into 172 resistant and 72 susceptible. The observed frequency fit well into an expected ratio of 3R : 1S with non significant χ^2 value of 2.645, indicating that the resistance to 40A pathotype of stem rust in Sel.2429 is controlled by a dominant gene. One hundred and fifty two BC_1 plants of this cross, 81 resistant and 71 susceptible fitting well into 1R : 1S ratio with non-significant χ^2 value of 0.658. This supported the F_2 analysis

The F_2 plants in the crosses, LB x Sel.2429 segregated in resistance (102 plants) and susceptibility (30 plants) and 145 were resistant while 59 susceptible in WL 711 x Sel.2429. The observed frequencies in resistant and susceptible categories fit well into 3R : 1S ratio with non significant χ^2s of 0.364 and 1.673 respectively. These results indicated that the resistance to the pathotype 40A in Sel.2429 is controlled by a single dominant gene. The BC_1F_1 populations of the cross LB / Sel. 2429//LB produced 22 resistant plants 13 susceptible while in the cross WL 711/Sel.2429//WL711 plants were resistant and susceptible. The observed frequency fit well into 1R : 1S with non-significant χ^2 values of 2.314 and 1.250 respectively supporting the hypothesis proposed in F_2 genetic analysis.

The pooled F_2 data over the crosses was also analysed, which fit well into 3 : 1 ratio (non significant χ^2 value of 2.354) and upon pooling BC_1F_1 data a non significant χ^2 value of 3.150 was obtained for 1R : 1S ratio. This further supported the genetic analysis that a single dominant is determining the resistance to 40A pathotype in Sel.2429.

4.2.2.2 Selection 4805-1

The mode inheritance of stem rust resistance in Sel.4805-1 was studied and the results are presented in Tables 16 and 17. Two crosses viz., AL x Sel.4805-1 and NI 5439 x Sel.4805-1 were made and the F_1 hybrids, F_2 and BC_1F_1 generations were tested with two pathotypes viz. 117-6 and 40A of stem rust. The results are discussed below:

Table 16. Mode of segregation for resistance to stem rust pathotype 40A in Sel. 4805-1 at seedling stage

Parent/cross	Parent/ generation	Number of seedlings			Expected Ratio	χ^2	P- value
		Resistant (IT:0-2)	Susceptible (IT:3-4)	Total			
Agra Local (AL)	P_1	0	18	18			
Sel. 4805-1	P_2	27	0	27			
AL/Sel. 4805-1	F_1	22	0	22			
	F_2	68	25	93	3R:1S	0.176	0.70-0.50
AL/Sel. 4805-1//AL	BC_1F_1	17	12	29	1R:1S	0.862	0.50-0.30
NI 5439	P_1	0	14	14			
Sel. 4805-1	P_2	17	0	17			
NI 5439/Sel.4805-1	F_1	26	0	26			
	F_2	48	20	68	3R:1S	0.706	0.50-0.30
NI5439/Sel. 4805-1//NI5439	BC_1F_1	15	21	36	1R:1S	1.000	0.50-0.30
Pooled over two crosses	F_2	116	45	161	3R:1S	0.747	0.50-0.30
	BC_1F_1	32	33	65	1R:1S	0.015	0.95-0.90

($\chi^2 = 3.841$; P = 0.05)

Table 17. Mode of segregation for resistance to stem rust pathotype 117-6 in Sel. 4805-1 at seedling stage

Parent/cross	Parent/ generation	Number of seedlings			Expected Ratio	χ^2	P- value
		Resistant (IT:0-2)	Susceptible (IT:3-4)	Total			
Agra Local (AL)	P_1	0	18	18			
Sel. 4805-1	P_2	15	0	15			
AL/Sel. 4805-1	F_1	22	0	22			
	F_2	85	24	109	3R:1S	0.517	0.50-0.30
AL/Sel. 4805-1//AL	BC_1F_1	24	18	42	1R:1S	0.857	0.50-0.30
NI 5439	P_1	0	19	19			
Sel. 4805-1	P_2	24	0	24			
NI 5439/Sel.4805-1	F_1	21	0	21			
	F_2	78	28	106	3R:1S	0.113	0.80-0.70
NI5439/Sel. 4805-1//NI5439	BC_1F_1	18	15	33	1R:1S	0.273	0.70-0.50
Pooled over two crosses	F_2	163	52	215	3R:1S	0.076	0.80-0.70
	BC_1F_1	42	33	75	1R:1S	1.080	0.30-0.20

($\chi^2 = 3.841$; P = 0.05)

The F_1 seedlings of both the crosses showed resistant reaction to the pathotype 117-6 indicating that resistance is dominant. Out of 109 F_2 plants tested in AL x Sel.4805-1 cross, 85 were resistant and 24 susceptible. The observed frequencies of two categories fit well into an expected ratio of 3R : 1S with non-significant χ^2 value of 0.517. The results indicated the presence of a single dominant gene for resistance to the race 117-6 in Sel. 4805-1. The BC_1F_1 population segregated into 24 resistant and 18 susceptible plants which fit well into an expected ratio 1R : 1S with non significant χ^2 value of 0.857.

The F_2 plant generation of the second cross NI 5439 x Sel.4805-1 produced 78 resistant and 28 susceptible plants, which fit well into a ratio of 3R : 1S with non-significant χ^2 value of 0.113. Out of 33 BC_1F_1 plants derived from NI 5439/Sel.4805-1//NI5439 cross 18 showed resistance and 15 susceptibility. The observed frequency fit well into 1R : 1S ratio with χ^2 value of 0.273 which is non-significant supporting the hypothesis that resistance against 117-6 in Sel. 4805-1 is determined by a single dominant gene.

The data in F_2 and BC_1F_1 were pooled separately and analysed genetically. The pooled observed frequency in F_2 generation as well as in BC_1F_1 population fit well into an expected ratios of 3R : 1S ($\chi^2 = 0.076$) and 1R : 1S respectively supporting the above hypothesis

In order to study the mode of inheritance the F_1 plants of the crosses, AL x Sel.4805-1 and NI5439 x Sel.4805-1 were also tested with 40A pathotype of stem rust. The resistance reaction in F_1 indicated the dominance of resistance. Out of 93 F_2 plants in the cross AL x Sel.4805-1, 68 were observed to be resistant and 25 susceptible. The F_2 data fit well into an expected 3R : 1S ratio with non significant χ^2 value of 0.176. These results demonstrated that the resistance in Sel.4805-1 is controlled by a single dominant gene. The BC_1F_1 population derived from the AL/Sel.4805-1//AL segregated into resistance (17) and susceptibility (12) fit well into an expected ratio of 1R : 1S with non-significant χ^2value 0.862 further confirming the F_2 hypothesis that resistance in Sel.4805-1 is indeed determined by a single dominant gene. Similarly, the F_2 plants in the cross NI 5439 x Sel.4805-1 segregated into 48 resistant and 20 susceptible. The data observed in F_2 generation fit well into a ratio of 3R : 1S with χ^2-0.706 which is non-significant. The F_2 analysis indicated that resistance against stem rust pathotype

40A is controlled by a single dominant gene. The BC_1F_1 population from NI 5439/ Sel.4805-1//NI5439 cross segregated into 15 resistant and 21 susceptible plants fitting well into an expected ratio of 1R : 1S ratio (χ^2-1.000) supporting the hypothesis that resistance in Sel.4805-1 is indeed determined by a single dominant gene..

The presence of a single dominant gene for resistance to the pathotype 40A in Sel.4805-1 was confirmed from the genetic analysis of pooled F_2 and BC_1-F_1 data as the pooled observed frequencies of resistant and susceptible plants fit well into 3 : 1 (χ^2 value = 0.747) and 1 : 1 (χ^2 value = 0.015) respectively.

4.3 Test of Allelism

The F_1 and F_2 seedlings of two crosses viz., HW 2043 (*Lr*19) x Sel.2429 (*Lr*24) and HW 2003 x Sel.2429 (Table 18) were tested with leaf rust pathotypes, 77-5 and 77-7 to ascertain the identity of resistance genes carried by the genetic stock Sel.2429. The derivative Sel.2429 was crossed with stocks carrying known genes for leaf rust resistance *Lr*19 and *Lr*24. The F_2 population of crosses HW 2043 (*Lr*19) x Sel.2429 and HW 2003 (*Lr*24) x Sel. 2429 segregated into resistance and susceptibility. Out of 91 seedlings 83 were resistant while 8 susceptible in the former cross whereas 105 plants were resistant and 12 susceptible to 77-5 pathotype of leaf rust. These observed frequencies of plant fit well into an expected ratio of 15R : 1S ratio. Similarly, when F_2 generations in both the crosses tested with 77-7 pathotype of leaf rust, 163 plant were resistant and 15 susceptible from HW 2043 x Sel.2429 whereas 88 F_2 plants showed resistant reaction and 10 susceptible in HW 2003 x Sel.2429 cross. The observed data in both the crosses also fit well into 15R : 1S ratio indicating the presence of one dominant gene in Sel.2429 conferring resistance to 77-7 pathotype.

Based on the F_2 analysis it is presumed that resistance in Sel.2429 is controlled by a single dominant gene to both the races 77-5 and 77-7 and these genes are presumbaly different than *Lr*19 and *Lr*24 present in HW 2043 and HW2003 respectively.

4.4 Chromosomal location of leaf rust resistance gene(s)

For the purpose of chromosomal location of leaf rust resistance, a complete set of monosomic series in the background of Chinese Spring was used. Individual monosomic line in the series were identified cytologically and crossed with the resistant genetic stock Sel. 2429. The F_1 monosomic plants were also cytologically identified

Table 18. Segregation pattern in F_2 generation of resistant x resistant parents against leaf rust pathotypes, 77-5 and 77-7

Cross	Number of seedlings			Expected Ratio	χ^2	P- value
	Resistant (IT:0-2)	Susceptible (IT:3-4)	Total			
(i) Tested with leaf rust pathotype 77-5						
HW 2043 (*Lr 19*) x Sel. 2429	83	8	91	15R:1S	1.003	0.50-0.30
HW 2003 (*Lr 24*) x Sel. 2429	105	12	117	15R:1S	3.205	0.10-0.05
(ii) Tested with leaf rust pathotype 77-7						
HW 2043 (*Lr 19*) x Sel. 2429	163	15	178	15R:1S	1.350	0.30-0.20
HW 2003 (*Lr 24*) x Sel. 2429	88	10	98	15R:1S	2.615	0.20-0.10

($\chi^2 = 3.841$; P = 0.05)

Table 19. Segregation of F_2 plants derived from F_1 monosomic hybrids between monosomic series of var. Chinese Spring and Sel. 2429 for resistance to leaf rust pathotype 77-5 in seedling stage

Cross	Resistance	Susceptible	Total	Expected ratio	χ^2	P-value
CS1A^m/Sel. 2429	34	18	52	3R : 1S	2.5641	0.10-0.05
CS1B^m/Sel. 2429	19	7	26	3R : 1S	0.0513	0.90-0.80
CS1D^m/Sel. 2429	28	10	38	3R : 1S	0.0351	0.90-0.80
CS2A^m/Sel. 2429	36	16	52	3R : 1S	0.9231	0.50-0.30
CS2B^m/Sel. 2429	33	18	51	3R : 1S	2.8824	0.01-0.05
CS2D^m/Sel. 2429	125	10	135	3R : 1S	22.2840	<0.001
CS3A^m/Sel. 2429	26	14	40	3R : 1S	2.1333	0.20-0.10
CS3B^m/Sel. 2429	60	28	88	3R : 1S	2.1818	0.20-0.10
CS3D^m/Sel. 2429	82	36	118	3R : 1S	1.9096	0.20-0.10
CS4A^m/Sel. 2429	27	12	39	3R : 1S	0.6923	0.50-0.30
CS4B^m/Sel. 2429	36	17	53	3R : 1S	1.4151	0.30-0.20
CS4D^m/Sel. 2429	38	18	56	3R : 1S	1.5438	0.30-0.20

Table 19 contd...

Cross	Resistance	Susceptible	Total	Expected ratio	χ^2	P-value
CS5A^m/Sel. 2429	92	38	130	3R : 1S	1.2410	0.30-0.20
CS5B^m/Sel. 2429	20	6	26	3R : 1S	0.0513	0.90-0.80
CS5D^m/Sel. 2429	103	43	146	3R : 1S	1.5434	0.30-0.20
CS6A^m/Sel. 2429	42	22	64	3R : 1S	3.1071	0.10-0.05
CS6B^m/Sel. 2429	25	15	40	3R : 1S	3.1333	0.10-0.05
CS6D^m/Sel. 2429	45	23	68	3R : 1S	2.8235	0.10-0.05
CS7A^m/Sel. 2429	34	13	47	3R : 1S	0.1773	0.70-0.50
CS7B^m/Sel. 2429	44	23	67	3R : 1S	3.1095	0.10-0.05
CS7D^m/Sel. 2429	37	16	53	3R : 1S	0.4610	0.50-0.30

($\chi^2 = 3.841$; P = 0.05)

to confirm their 2n-1 number. The F_2 population of each cross along with the parents and normal disomics were tested with the leaf rust pathotype 77-5 in seedling stage under glass house conditions with temperature ranging from 18^0 to 27^0C.

The segregation pattern of F_2 generation derived from 21 F_1 monosomic plant(s) invloving individual lines of Chinese Spring monosomic series and Sel.2429 (Table 19) showed non-significant chi-square values ranging from 0.035 to 3.133 with P-value of 0.90- 0.05 in all the crosses except CS 2D mono/Sel.2429. The significant chi square value of 22.284 (P value :<0.001) was observed in the exceptional cross CS 2D mono/ Sel.2429, which deviated completely from the monogenic (3:1) ratio.

The statistical significance and non-significance was calculated in all the lines as against the tabulated value of 3.84 at one degree of freedom and 0.05% level of significance with the expected ratio of 3R : 1S.

4.5 Inheritance of morphological traits

4.5.1 Leaf sheath pubescence (*Sp*) in Selection 2429

Contrasting parents differing in leaf sheath pubescence and its absence [Pubescence (P) Vs. Glabrous (G)] were crossed. The F_1 phenotype of the cross AL (G) x Sel.2429 (P) was pubescent (Table 20), which indicated the dominant nature of sheath pubescence. Out of 172 F_2 plants, the leaf sheath of 137 plants was pubescent while 35 produced non pubescent. The observed data in F_2 generation fit well into an expected ratio of 15 pubescent : 1 glabrous (χ^2 = 1.985, P-value : 0.20 - 0.10). These results suggested that leaf sheath pubescence is under digenic control.

In the BC_1F_1 population derived from the cross AL/Sel.2429//AL, 29 plants showed pubescent leaf sheath while 27 were glabrous. The observed frequencies in BC_1F_1 fit well into an expected ratio of 1 pubescent : 1 glabrous. The value of χ^2 recorded was 0.071. The BC_1F_1 results supported the hypothesis proposed in F_2 analysis that the leaf sheath pubescence is controlled by a single dominant gene.

4.5.2 Leaf sheath pubescence (*Sp*) and leaf pubescence (*Lp*) in selection 4805-1

The inheritance pattern of leaf pubescence and leaf sheath pubescence in Sel. 4805-1 was studied by analysing the F_1, F_2 and BC_1F_1 generations derived from the crosses NI5439 x Sel.4805-1 and F_1 x NI5439. The results are presented in Table 21.

Table 20. Segregation for leaf sheath pubescence in F_2 and BC_1F_1 generations

Parent/cross	Parent/ generation	Number of progenies in different classes		Total	Expected Ratio (P:G)	χ^2	P- value
		Pubescent (P)	Glabrous (G)				
Agra Local (AL)	P_1	0	23	23			
Sel. 2429	P_2	27	0	27			
AL/Sel. 2429/	F_1	29	0	29			
	F_2	137	35	172	3:1	1.9815	0.20-0.10
F_1 x AL	BC_1F_1	29	27	56	1:1	0.0714	0.80-0.70

($\chi^2 = 3.841$; P = 0.05)

Table 21. Segregation for leaf sheath and leaf pubescence in NI 5439 x Sel.4805-1 F_2 and (NI5439 x Sel.4805-1) x NI 5439 BC_1F_1 generations

Character	Parent/ generation	Number of progenies in different classes		Total	Expected Ratio (P:G)	χ^2	P- value
		Pubescent (P)	Glabrous (G)				
Leaf sheath pubescence	F_2	388	136	524	3:1	0.255	0.70-0.50
	BC_1F_1	55	40	95	1:1	2.368	0.20-0.10
Leaf pubescence	F_2	483	41	524	15:1	2.217	0.20-0.10
	BC_1F_1	71	24	95	3:1	0.004	0.98-0.95

($\chi^2 = 3.841$; P = 0.05)

The parent NI 5439 is glabrous (non-pubescent) while Sel. 4805-1 produces pubescence on leaf and leaf sheath. In F_2 generation, 388 plants produced pubescence on leaf sheath while 136 were non pubescent. The observed data fit well into an expected ratio of 3P : 1G with non-significant value of χ^2 (0.255). The data in BC_1F_1 generations with non-significant chi-square values supporting the hypothesis also fit well into expected ratio of 1P : 1G.

The trait leaf pubescence was also genetically analysed in Sel.4805-1. The F_1 plants in the cross NI 5439 x Sel.4805-1 showed presence of hairyness on upper surface of the leaf indicating that pubescence is dominant. Out of 524 plants, 483 produced pubescence in leaf while 41 did not. These observed frequencies of plants fit well into an expected ratio of 15P : 1G with non significant χ^2 value. In BC_1-F_1 generation 71 segregants were having pubescent leaves while 24 plants showed globrous leaves and the data fit well into 1 : 1 ratio for presence and absence of pubescence on leaves. The non-significant χ^2 value indicated that F_2 hypothesis is correct and that the pubescence on leaves is determined by two duplicate dominant genes.

4.6 Linkage analysis

4.6.1 Linkage between leaf sheath pubescence and rust resistance in Sel.2429

Sel.2429 possesses pubescence on leaf sheath and exhibited resistance to leaf rust and stem rust. Therefore, the linkage between leaf sheath pubescence and rust resistance (both leaf rust and stem rust resistance) was calculated by using F_2 data in the crosses AL x Sel.2429 and the results are presented in Table 22. The parent Agra Local is highly susceptible to rusts and does not produce any pubescence on leaf sheath.

Joint segregation of the gene(s) *Sp* and *Lr* (Leaf rust Resistance) was analysed in crosses AL x Sel.2429 to detect linkage between *Sp* and *Lr* genes. The value recorded for χ^2 was 0.332 with P value ranging from 0.70-0.50, which revealed the absence of linkage between leaf sheath pubescence and leaf rust resistance in Sel.2429.

Similar observations were made for leaf sheath pubescence and stem rust resistance (*Sr*) with non-significant chi square value 2.279 which indicated the absence of linkage between leaf sheath pubescence and stem rust resistance in Sel.2429.

Table 22. Joint segregation of leaf sheath pubescence (SP) with rust resistance in Sel. 2429 derivatives

Character	Phase	Gene pair	F_2 segregation					χ^2			P-value
			a	b	c	d	Total	Locus 1	Locus 2	Joint segregation	
Sheath pubescence (SP) Leaf rust resistance (Lr)	C	Sh-Lr	330	107	25	10	472	1.094 (15:1)	0.011 (3:1)	0.332 (45:15:3:1)	0.70-0.50
Sheath pubescence (SP) Stem rust resistance (Sr)	C	Sh-Sr	334	103	23	12	472	1.094 (15:1)	0.102 (3:1)	2.279 (45:15:3:1)	0.20-0.10

($\chi^2 = 3.841$; P = 0.05) C - Coupling phase

a = double dominant; A-B-
b = dominant recessive A-bb
c = recessive dominant aaB –
d = double recessive aabb

Table 23. Joint segregation of leaf and leaf sheath pubescence with leaf rust resistance in Sel. 4805-1

Character	Phase	Gene pair	F_2 segregation					χ^2			P-value
			a	b	c	d	Total	Locus 1	Locus 2	Joint segregation	
Leaf pubescence (Lp) Leaf rust resistance (Lr)	C	Lp-Lr	371	127	18	8	524	1.484 (15:1)	0.163 (3:1)	0.271 (45:15:3:1)	0.70-0.50
Sheath pubescence (Sp) Leaf rust resistance (Lr)	C	Sp-Lr	290	98	99	37	524	0.255 (3:1)	0.163 (3:1)	0.217 (9:3:3:1)	0.70-0.50
Leaf pubescence (Lp) Sheath pubescence (Sh)	C	Lp-Sp	368	130	20	6	524	1.484 (15:1)	0.255 (3:1)	0.115 (45:15:3:1)	0.80-0.70

($\chi^2 = 3.841$; P = 0.05) C - Coupling phase

a = double dominant A-B-
b = dominant recessive A-bb
c = recessive dominant aaB–
d = double recessive aabb

4.6.2 Linkage between leaf rust resistance with other morphological characters in Selection 4805-1

Joint segregation analysis of leaf rust resistance gene with leaf and leaf sheath pubescence was carried out in the cross AL x Sel.4805-1 (Table 23). The results analysed in F_2 population of the above mentioned cross were used to determine the linkage between *Lp-Lr* genes (Table 23). The genetic analysis revealed no linkage between *Lp* (leaf pubescence) and *Lr* (leaf rust resistance) genes, as the χ^2_L value was non significant (χ^2 = 0.271 : P value 0.70 - 0.50).

Similarly, no linkage could be established between the gene pair *Sp* (Sheath pubescence) and *Lr* (Leaf rust resistance) from joint segregation analysis of F_2 data in the cross AL x Sel.2429 (Table 23). The calculated χ^2_L was 0.217 with P-value ranging from 0.70-0.50.

4.6.3 Linkage between leaf pubescence (*Lp*) and sheath pubescence (*Sp*) in Sel. 4805-1

Joint F_2 segregation analysis was carried out in the cross AL x Sel.4805-1 to detect linkage between the genes *Lp* and *Sp* (Table 23). The genetic analysis revealed non significant value of χ^2_L for the gene pair *Lp-Sp* (χ^2 = 0.115; P value : 0.80 - 0.70). These results (Table 23) indicated the absence of linkage between the gene pair *Sp* (Sheath pubescence) and *Lp* (Leaf pubescence) in Sel. 4805-1.

4.7 Comparison of number of bivalents and univalents observed in F_1 plants of CS monosomic series x Sel. 2429.

Normally a disomic wheat plant produces 21 bivalents at 1st meiotic metaphase and a monosomic plant is identified by 20 bivalents and a single univalent. In a cytological analysis of disomic and monosomic F_1 plants derived from 21 lines of CS monosomic series x Sel.2429, variation in the number of bivalents and univalents was recorded. However, no significant differences were observed between bivalents of disomic plants but the differences between bivalents of a few monosomic lines were noticed (Table 24). In a number of lines precocious separation of a bivalent was seen and in disomics derived from CS1D x Sel.2429 and CS4A x Sel.2429 univalents upto 6 in number were also noticed indicating the effect of *A. speltoides* derived segment present in certain chromosome(s). The frequency of univalents was quite high in monosomic plants derived particularly from the crosses CS4B x Sel.2429, CS5A x

Table 24. Number of bivalents (II) and univalents (I) observed at first meiotic metaphase in disomic and monosomic F_1 plants in CS monosomic series x Sel.2429

Cell No.	CS1A x Sel.2429				CS1B x S2429				CS1D x S2429			
	Disomic		Monosomic		Disomic		Monosomic		Disomic		Monosomic	
	II	I	II	I	II	I	II	I	II	I	II	I
1	19	4	17	7	19	4	20	1	19	4	19	3
2	20	2	17	7	20	2	20	1	18	6	19	3
3	19	4	18	5	20	2	19	3	19	4	19	3
4	19	4	18	5	20	2	19	3	19	4	19	3
5	19	4	18	5	21	0	19	3	18	6	19	3
6	20	2	18	5	21	0	19	3	20	2	18	5
7	20	2	19	3	21	0	19	3	20	2	18	5
8	20	2	19	3	20	2	19	3	20	2	18	5
9	20	2	17	7	19	4	20	1	20	2	19	3
10	20	2	18	5	20	2	20	1	19	4	19	3
11	20	2	17	7	21	0	20	1	19	4	19	3
12	20	2	18	5	21	0	19	3	20	2	18	5
13	19	4	17	7	20	2	19	3	20	2	18	5
14	19	4	18	5	20	2	20	1	18	6	20	1
15	20	2	18	5	21	0	20	1	18	6	20	1
16	20	2	18	5	21	0	19	3	20	2	20	1
17	20	2	18	5	19	4	19	3	19	4	20	1
18	20	2	19	3	20	2	19	3	19	4	19	3
19	20	2	19	3	20	2	19	3	20	2	19	3
20	19	4	17	7	20	2	19	3	20	2	19	3
Mean	19.65	2.70	17.90	5.20	20.20	1.60	19.35	2.30	19.25	3.50	18.95	3.10
S.D.0.49	0.98	0.72	1.44	0.70	1.40	0.49	0.98	0.79	1.58	0.69	1.38	
S.E.	±0.11	±0.22	±0.16	±0.32	±0.16	±0.32	±0.11	±0.22	±0.18	±0.36	±0.15	±0.30

Table 24 contd...

Cell No.	CS2A x Sel.2429				CS2B x Sel.2429				CS2D x Sel.2429			
	Disomic		Monosomic		Disomic		Monosomic		Disomic		Monosomic	
	II	I	II	I	II	I	II	I	II	I	II	I
1	19	4	20	1	20	2	19	3	18	6	20	1
2	20	2	20	1	20	2	19	3	18	6	19	3
3	20	2	19	3	20	2	20	1	19	4	17	7
4	20	2	19	3	20	2	20	1	19	4	17	7
5	20	2	19	3	20	2	20	1	19	4	17	7
6	20	2	19	3	20	2	19	3	17	8	17	7
7	20	2	19	3	20	2	19	3	17	8	19	3
8	20	2	19	3	20	2	19	3	19	4	19	3
9	20	2	20	1	20	2	19	3	19	4	20	1
10	20	2	20	1	20	2	20	1	19	4	19	3
11	20	2	19	3	20	2	20	1	20	2	19	3
12	19	4	19	3	20	2	20	1	20	2	19	3
13	19	4	19	3	20	2	20	1	20	2	20	1
14	20	2	20	1	20	2	19	3	18	6	20	1
15	20	2	20	1	20	2	19	3	18	6	20	1
16	20	2	19	3	20	2	19	3	18	6	19	3
17	20	2	19	3	20	2	19	3	18	6	17	7
18	19	4	19	3	20	2	19	3	19	4	17	7
19	19	4	20	1	20	2	19	3	19	4	19	3
20	20	2	19	3	20	2	19	3	19	4	19	3
Mean	19.75	2.50	19.35	2.30	20.00	2.00	19.35	2.30	18.65	4.70	18.65	3.70
S.D.	0.44	0.88	0.49	0.98	0.00	0.00	0.49	0.98	0.88	1.76	1.18	2.30
S.E.	±0.10	±0.20	±0.11	±0.22	±0.00	±0.00	±0.11	±0.22	±0.20	±0.40	±0.27	±0.54

Table 24 contd...

Cell No.	CS3A x Sel.2429				CS3B x Sel.2429				CS3D x Sel.2429			
	Disomic		Monosomic		Disomic		Monosomic		Disomic		Monosomic	
	II	I	II	I	II	I	II	I	II	I	II	I
1	19	4	19	3	20	2	20	1	19	4	18	5
2	20	2	18	5	20	2	20	1	19	4	18	5
3	20	2	19	3	20	2	20	1	19	4	19	3
4	20	2	19	3	21	0	19	3	20	2	19	3
5	20	2	18	5	21	0	19	3	19	4	19	3
6	20	2	20	1	21	0	19	3	20	2	20	1
7	20	2	20	1	21	0	19	3	20	2	20	1
8	20	2	18	5	21	0	19	3	20	2	19	3
9	20	2	18	5	20	2	19	3	20	2	19	3
10	20	2	19	3	20	2	19	3	19	4	19	3
11	19	4	19	3	20	2	20	1	19	4	18	5
12	19	4	19	3	20	2	20	1	19	4	18	5
13	19	4	19	3	21	0	20	1	20	2	18	5
14	20	2	18	5	21	0	20	1	20	2	19	3
15	20	2	20	1	21	0	20	1	20	2	20	1
16	20	2	20	1	20	2	19	3	20	2	20	1
17	19	4	20	2	20	2	19	3	20	2	20	1
18	20	2	20	1	20	2	20	1	20	1	19	3
19	19	4	19	3	21	0	19	3	19	4	19	3
20	20	2	19	3	20	2	20	1	20	2	18	5
Mean	19.70	2.60	19.05	2.90	20.45	1.10	19.50	2.00	19.60	2.80	18.95	3.10
S.D.	0.47	0.94	0.76	1.42	0.51	1.02	0.51	1.02	0.50	1.00	0.76	1.42
S.E.	±0.11	±0.22	±0.17	±0.34	±0.11	±0.22	±0.2	±0.24	±0.11	±0.22	±0.17	±0.34

Table 24 contd...

Cell No.	CS4A x Sel.2429				CS4B x Sel.2429				CS4D x Sel.2429			
	Disomic		Monosomic		Disomic		Monosomic		Disomic		Monosomic	
	II	I	II	I	II	I	II	I	II	I	II	I
1	20	2	20	1	18	6	17	7	20	2	19	3
2	20	2	20	1	20	2	17	7	20	2	19	3
3	20	2	19	1	20	2	17	7	20	2	19	3
4	21	0	20	1	19	4	17	7	21	0	19	3
5	21	0	20	1	19	4	17	7	20	2	19	3
6	21	0	20	1	19	4	19	3	20	2	20	1
7	21	0	20	1	19	4	19	3	20	2	20	1
8	21	0	20	1	18	6	18	5	20	2	20	1
9	21	0	19	3	18	6	18	5	20	2	20	1
10	20	2	20	1	18	6	18	5	21	0	20	1
11	20	2	20	1	18	6	18	5	21	0	20	1
12	20	2	19	3	20	2	18	5	21	0	20	1
13	20	2	19	3	20	2	18	5	21	0	19	3
14	20	2	19	3	20	2	17	7	20	2	19	3
15	20	2	19	3	20	2	19	3	20	2	19	3
16	21	0	19	3	19	4	19	3	20	2	20	1
17	21	0	19	3	19	4	19	3	21	0	20	1
18	21	0	20	1	19	4	19	3	21	0	19	3
19	21	0	20	1	20	2	19	3	21	0	20	1
20	21	0	20	1	20	2	17	7	20	2	20	1
Mean	20.55	0.90	19.65	1.70	19.15	3.70	18.00	5.00	20.40	1.20	19.55	1.90
S.D.	0.51	1.02	0.49	0.98	0.81	1.62	0.86	1.72	0.50	1.00	0.51	1.02
S.E.	±0.11	±0.22	±0.11	±0.22	±0.18	±0.36	±0.19	±0.38	±0.11	±0.22	±0.11	±0.22

Table 24 contd...

Cell No.	CS5A x Sel.2429				CS5B x Sel.2429				CS5D x Sel.2429			
	Disomic		Monosomic		Disomic		Monosomic		Disomic		Monosomic	
	II	I	II	I	II	I	II	I	II	I	II	I
1	20	2	17	7	20	2	19	3	20	2	19	3
2	20	2	17	7	20	2	19	3	21	0	19	3
3	19	4	18	5	20	2	19	3	21	0	19	3
4	19	4	18	5	20	2	19	3	21	0	19	3
5	19	4	18	5	19	4	19	3	21	0	19	3
6	19	4	18	5	19	4	19	3	21	0	19	3
7	18	6	18	5	19	4	19	3	20	2	19	3
8	18	6	17	7	19	4	19	3	20	2	20	1
9	18	6	19	3	20	2	19	3	20	2	20	1
10	19	4	19	3	20	2	19	3	20	2	20	1
11	19	4	20	1	20	2	19	3	20	2	20	1
12	20	2	20	1	20	2	19	3	20	2	20	1
13	20	2	17	7	19	4	19	3	20	2	19	3
14	20	2	17	7	19	4	19	3	21	0	19	3
15	19	4	17	7	19	4	19	3	21	0	20	1
16	19	4	19	3	20	2	19	3	21	0	20	1
17	20	2	19	3	20	2	19	3	21	0	20	1
18	20	2	19	3	19	4	19	3	20	2	19	3
19	18	6	17	7	20	2	19	3	20	2	19	3
20	18	6	18	5	20	2	19	3	21	0	19	3
Mean	19.10	3.80	18.10	4.80	19.60	2.80	19.00	3.00	20.50	1.00	19.40	2.20
S.D.	0.79	1.58	1.02	2.04	0.50	1.00	0.00	0.00	0.51	1.02	0.50	1.00
S.E.	±0.18	±0.36	±0.23	±0.46	±0.11	±0.22	±0.00	±0.00	±0.12	±0.24	±0.11	±0.22

Table 24 contd...

Cell No.	CS6A x Sel.2429				CS6B x Sel.2429				CS6D x Sel.2429			
	Disomic		Monosomic		Disomic		Monosomic		Disomic		Monosomic	
	II	I	II	I	II	I	II	I	II	I	II	I
1	19	4	18	5	21	0	19	3	20	2	19	3
2	19	4	18	5	21	0	19	3	20	2	20	1
3	18	6	18	5	21	0	19	3	20	2	20	1
4	18	6	18	5	20	2	19	3	20	2	19	3
5	18	6	19	3	20	2	19	3	20	2	19	3
6	19	4	19	3	21	0	20	1	20	0	19	3
7	19	4	19	3	21	0	20	1	19	4	19	3
8	19	4	19	3	21	0	20	1	19	4	20	1
9	18	6	18	5	20	2	20	1	19	4	20	1
10	18	6	18	5	20	2	20	1	19	4	19	3
11	18	6	19	3	19	4	19	3	18	6	19	3
12	19	4	19	3	19	4	19	3	18	6	20	1
13	19	4	18	5	19	4	20	1	18	6	20	1
14	19	4	18	5	19	4	20	1	19	4	19	3
15	19	4	19	3	21	0	20	1	19	4	20	1
16	18	6	19	3	21	0	19	3	20	2	20	1
17	18	6	19	3	21	0	19	3	20	2	20	1
18	19	4	19	3	20	2	20	1	20	2	20	1
19	19	4	19	3	20	2	20	1	20	2	19	3
20	19	4	18	5	20	2	20	1	18	6	20	1
Mean	18.60	4.80	18.55	3.90	20.25	1.50	19.55	1.90	19.30	3.40	19.55	1.90
S.D.	0.50	1.00	0.51	1.02	0.79	1.58	0.51	1.02	0.80	1.60	0.51	1.02
S.E.	±0.11	±0.22	±0.11	±0.22	±0.18	±0.36	±0.11	±0.22	±0.18	±0.36	±0.11	±0.22

Table 24 contd...

Cell No.	CS7A x Sel.2429				CS7B x Sel.2429				CS7D x Sel.2429			
	Disomic		Monosomic		Disomic		Monosomic		Disomic		Monosomic	
	II	I	II	I	II	I	II	I	II	I	II	I
1	19	4	19	3	18	6	17	7	20	2	19	3
2	19	4	19	3	18	6	17	7	21	0	19	3
3	19	4	19	3	18	6	17	7	20	2	19	3
4	19	4	19	3	18	6	17	7	21	0	19	3
5	19	4	19	3	18	6	18	5	20	2	19	3
6	19	4	19	3	19	4	18	5	20	2	19	3
7	19	4	19	3	19	4	18	5	20	2	20	1
8	19	4	19	3	19	4	19	3	20	2	20	1
9	19	4	19	3	18	6	19	3	20	2	20	1
10	19	4	19	3	19	4	17	7	20	2	20	1
11	19	4	19	3	19	4	18	5	21	0	20	1
12	19	4	19	3	19	4	19	3	21	0	19	3
13	19	4	19	3	18	6	18	5	21	0	19	3
14	19	4	19	3	19	4	19	3	20	2	20	1
15	19	4	19	3	19	4	17	7	21	0	21	1
16	19	4	19	3	18	6	17	7	21	0	19	3
17	19	4	19	3	18	6	17	7	20	2	19	3
18	19	4	19	3	19	4	19	3	20	2	19	3
19	19	4	19	3	19	4	19	3	21	0	19	3
20	19	4	19	3	18	6	18	5	21	0	20	1
Mean	19.00	4.00	19.00	3.00	18.50	5.00	17.90	5.20	20.45	1.10	19.40	2.20
S.D.	0.00	0.00	0.00	0.00	0.51	1.02	0.85	1.70	0.51	1.02	0.50	1.00
S.E.	±0.00	±0.00	±0.00	±0.00	±0.12	±0.24	±0.19	±0.38	±0.11	±0.22	±0.11	±0.22

Table 25. **Average number of bivalents and univalents observed at first meiotic metaphase in disomic and monosomic F_1 plants in CS monosomic series x Sel 2429**

Cross	Disomic plants		Monosomic plants	
	Average number of bivalents	Average number of univalents	Average number of bivalents	Average number of univalents
CS1A x Sel.2429	19.65	2.70	17.90	5.20
CS1B x Sel.2429	20.20	1.60	19.35	2.30
CS1D x Sel.2429	19.25	3.50	18.95	3.10
CS2A x Sel.2429	19.75	2.50	19.35	2.30
CS2B x Sel.2429	20.00	2.00	19.35	2.30
CS2D x Sel.2429	18.65	4.70	18.65	3.70
CS3A x Sel.2429	19.70	2.60	19.05	2.90
CS3B x Sel.2429	20.45	1.10	19.50	2.00
CS3D x Sel.2429	19.60	2.80	18.95	3.10
CS4A x Sel.2429	20.55	0.90	19.65	1.70
CS4B x Sel.2429	19.15	3.70	18.00	5.00
CS4D x Sel.2429	20.40	1.20	19.55	1.90
CS5A x Sel.2429	19.10	3.80	18.10	4.80
CS5B x Sel.2429	19.60	2.80	19.00	3.00
CS5D x Sel.2429	20.50	1.00	19.40	2.20
CS6A x Sel.2429	18.60	4.80	18.55	3.90
CS6B x Sel.2429	20.25	1.50	19.55	1.90
CS6D x Sel.2429	19.30	3.40	19.55	1.90
CS7A x Sel.2429	19.00	4.00	19.00	3.00
CS7B x Sel.2429	18.50	5.00	17.90	5.20
CS7D x Sel.2429	20.45	1.10	19.40	2.20
Mean	19.650	2.700	18.990	3.030
SE	0.145	0.291	0.128	0.255
μ	19.500	2.500	18.500	2.900
Calaculated 't'	1.033	0.678	3.843*	0.509

* Significant at 5% for 20 degrees of freedom.

Table 26. Comparison of number of bivalents observed at first meiotic metaphase between disomic and monosomic F_1 plants in CS monosomic series x S2429

Cross	Pooled estimate of variance (σ^2c)	't' value
CS1A x Sel.2429	0.380	8.757*
CS1B x Sel.2429	0.365	22.110*
CS1D x Sel.2429	0.550	1.247
CS2A x Sel.2429	0.217	17.518*
CS2B x Sel.2429	0.120	51.419*
CS2D x Sel.2429	1.083	0.000
CS3A x Sel.2429	0.399	15.461*
CS3B x Sel.2429	0.260	34.686*
CS3D x Sel.2429	0.414	15.355*
CS4A x Sel.2429	0.250	34.174*
CS4B x Sel.2429	0.698	15.650*
CS4D x Sel.2429	0.255	31.649*
CS5A x Sel.2429	0.832	11.411*
CS5B x Sel.2429	0.125	45.584*
CS5D x Sel.2429	0.255	40.958*
CS6A x Sel.2429	0.255	1.861
CS6B x Sel.2429	0.442	15.037*
CS6D x Sel.2429	0.450	5.275*
CS7A x Sel.2429	0.000	0.000
CS7B x Sel.2429	0.491	11.598*
CS7D x Sel.2429	0.255	39.089*

* Significant at 5% for 38 degrees of freedom.

Sel.2429 and CS7B x Sel.2429 indicating the major influence of *Ae. speltoides* derived segment present in Sel.2429.

Normally, both disomic and monosomic F_1 plants different in respect of bivalents as they produce 21 bivalents and 20 bivalents respectively. Exceptionally there were no differences between the bivalents produced in CS2D x Sel.2429 and CS7A x Sel.2429 as the pooled estimated variance has zero.

DISCUSSION

The production and productivity of wheat crop is greatly influenced by several diseases but rusts are more prominent in causing damage. The high intensity of leaf rust and stem rust infection in rust prone regions is mainly attributed to the frequent changes in race flora due to evolution of new races/biotypes of the pathogen by spontaneous mutations. This situation necessitates the wheat breeders to search for useful new sources of resistance continuously. Of the designated resistance genes, majority of them are native to *T. aestivum* and *T. durum, T. dicoccum* and other sub-species, which have become ineffective against new virulence(s) of rust pathogen. Only a few genes of alien origin such as *Lr*9 from *Ae. umbellulata* (Sears, 1956), *Lr19/ Sr25* and *Lr24/Sr*24 from *A. elongatum* (Sharma and Knott, 1966 and McIntosh *et al.,* 1977), *Lr*28 (Riley *et al.,* 1968), *Lr*35 and *Sr39* (Kerber and Dyck, 1990) from *Ae. spletoides* and *Lr*37 (Bariana and McIntosh, 1994) from *Ae, ventricosa* are effective against the prevalent Indian races of leaf rust and stem rust pathogens (Tomar and Menon, 2001). Due to host-parasite interaction, the genes such as *Lr9* and *Lr19* have been overcome by new virulence(s) in India though they are not prevalent.

The wheat species, *T. militinae* a mutant of *T. timopheevi* (genome, AAGG) and *Ae. speltoides* are well known for their resistance to common fungal diseases that attack wheat (Zhukovsky, 1971 and Dhaliwal *et al.,* 1986). Several attempts have been made to transfer disease resistance from *T. timopheevi* to common wheat (Peusha *et al.,* 1996 ; Enno *et al.,* 1998; Tomar *et al.,* 1997). Several genes such as *Lr*28, *Lr*35 and *Lr*36 conferring resistance to leaf rust have been transferred from *Ae. speltoides* to common wheat. Both the diverse species still offer an excellent source of resistance to rusts. Cytologically stable hexaploid selections were isolated from the wide crosses. One such derivative of *T. militinae* Sel. 4805-1 exhibited a high level of resistance to rusts and powdery mildew. Similarly a derivative 'Sel. 2429' from Sonalika/*Ae. speltoides* showed a high degree of resistance to stem rust and leaf rust in adult stage.

Investigations were, therefore, carried out to find out the nature and number of gene(s) controlling *Ae, speltoides* and *T. militinae* derived resistance to leaf rust and

stem rust in Sel.2429 and Sel.4805-1. The present study also aimed at locating gene(s) for leaf rust resistance in Sel.2429 on specific chromosome(s) and also to study the linkage between the morphological traits and rust resistance. The objectivewise results are discussed hereunder.

5.1 Multipathotype Test

Screening the genetic stocks for resistance to different prevalent pathotypes of rusts is essential for their use in resistance breeding programme. Such screening also provides an understanding on degree and range of resistance to a spectrum of pathotypes. Derivatives of wheat x *Ae.speltiodes* (Sel. 2429) and wheat x *T. militinae* (Sel. 4805-1) and their parental lines *Ae. speltiodes* and *T. militinae* were tested with three pathotypes of leaf rust (*P. triticina*) and stem rust (*P. graminis tritici*) for determining the pattern of resistance. Both the derivatives, Sel. 2429 and Sel.4805-1 conferred a high degree of resistance to 77-5, 77-6 and 77-7 of leaf rust and 40A and 117-6 pathotypes of stem rust. A high degree of field resistance to leaf rust and stem rust in *Ae. speltoides* and *T. militinae* and their derivatives, both under controlled and field conditions, was reported by Tomar *et al.* (1997). The *T. aestivum* parents involved in the evolution of these inter-specific derivatives exhibited susceptible reaction to the rust pathotypes as mentioned in the results. Although the source of rust resistance in the derivatives was different but the pattern of reactions or infection type (IT) exhibited by Sel.2429 (IT '0;' -';1') and Sel.4805-1 (IT '0;' - ';1') to all the three selected pathotypes of *P. triticina* was, however, not much different. The little difference in pattern of reaction could be manifested by the effect of background of recipient cultivars employed in genetic transfer and narrow diversity revealed among the derivatives could also be attributed to less number of pathotypes of rusts to which they were subjected in seedling tests.

The genetic stocks, Sel. 2429 and Sel.4805-1 when tested with selected pathotypes of stem rust produces ITs ranging from '0;' to ';1'. A similar explanation of narrow range in reaction pattern among the stocks could also be extended for stem rust resistance as only two pathotypes of stem rust were selected for the study. The seedling reaction of the two donor species with three pathotypes of *P. triticina* and two pathotypes of *P. graminis tritici* were of IT '0;". In order to identify the resistance

gene(s) in derivatives contributed by each donor species, all the 5 pathotypes belonging to both the pathogens were considered for inheritance studies. All these pathotypes are virulent on Agra Local, Lal Bahadur, WL 711 and NI 5439.

5.2 Inheritance of resistance to leaf and stem rusts

The nature and number of gene(s) conferring resistance to leaf rust and stem rust in Sel.2429 and Sel.4805-1 were determined through genetic analysis. Different segregating generations derived from the crosses involving resistant genetic stocks Sel. 2429 and Sel.4805-1 and susceptible cultivars viz., Agra Local, Lal Bahadur, WL711 and NI5439 were tested with selected pathotype of leaf and stem rusts. However, the genetic analysis of resistance was restricted to only the most virulent and prevalent pathotypes 77-5, 77-6 and 77-7 of *P. triticina* and 40A and 117.6 of *P. graminis* f.sp. *tritici.*

5.2.1 Leaf rust resistance

Genetic analysis on resistance in Sel.2429 to leaf rust pathotypes 77-5, 77-6 and 77-7 were carried out based on three crosses each involving a different susceptible parent viz., Agra Local, Lal Bahadur and WL711. The resistant reaction to 77-5 and 77-7 pathotypes in all F_1 plants of the cross Agra Local x Sel.2429 indicated that the resistance is dominant. The segregation of F_2 population of the above mentioned cross fits well into an expected ratio of 3 resistant : 1 susceptible indicating the monogenic dominant nature of resistance to 77-5 and 77-7 pathotypes. The BC_1F_1 population segregated into 1R : 1S ratio with good fit further validated the hypothesis proposed on the basis of genetic analysis in F_2. A similar trend was observed in F_1, F_2 and BC_1F_1 populations of the other two crosses Lal Bahadur x Sel. 2429 and WL711 X Sel. 2429 (Tables 8 and 10) for both the pathotypes 77-5 and 77-7 of leaf rust indicating monogenic dominant control of leaf rust resistance in Sel. 2429. These results indicated that the parents in each cross differed at only one locus governing the resistance to the pathotypes 77-5 and 77-7 of *P. triticina.*

The seedling reaction to 77-6 pathotype in all F_1 plants from the crosses, Agra Local x Sel. 2429, Lal Bahadur x Sel.2429 and WL 711 x Sel. 2429 showed susceptibility indicating that resistance is recessive. The segregation in F_2 population into 1 resistant : 3 susceptible monogenic ratio confirmed recessive nature of resistance.

The BC_1F_1 population derived from two crosses segregated into 1R : 1S ratio with good fit validated the hypothesis that resistance in Sel. 2429 against 77-6 pathotype is controlled by single reccessive gene. Three leaf rust resistance genes viz. *Lr*28, *Lr*35 and *Lr*36 have been transferred into wheat from *Ae. speltoides*, however, each one of them exhibited a different pattern of reaction in seedling stage (Tomar and Menon, 2001). The resistance identified in Sel. 2429 seems to be different than that imparted by these genes.

T. militimae derived stock Sel. 4805-1 was subjected to genetic analysis for rust resistance. The F_1 seedlings of the crosses, Agra Local x Sel. 4805-1 and NI5439 x Sel.4805-1 showed resistance when tested with 77-5, 77-6 and 77-7 pathotypes of *P. triticina.* The reaction pattern in F_1 seedlings indicated that resistance is dominant. The observed frequency of resistant and susceptible plants in F_2 generation of both the crosses when tested with 77-5 and 77-7 pathotypes fit well into 3R : 1S with a non-significant χ^2 value. The data indicated that resistance in Sel. 4805-1to the above mentioned pathotypes is controlled by a single dominant gene. The monogenic inheritance of resistance to 77-5 and 77-7 leaf rust pathotpyes was further confirmed by analysing BC_1F_1 generation. The data in BC_1F_1 generation fitting perfectly well into an expected ratio of IR : 1S with non-significant χ^2 value. It can therefore, be concluded that the resistance against leaf rust pathotypes 77-5 and 77-7 is indeed governed by a single dominant gene.

In a similar study carried out by Sibikeev *et al.* (1994), Peusha *et al.* (1996) and Enno *et al.* (1998) in *T. militinae* derivatives, a single dominant gene imparting resistance to leaf rust pathotypes was reported. However, the plant materials as well as the races of *P. triticina* used were different in their studies. The screening of wheat x *Triticum timopheevi* derivatives and a few other genetic stocks against leaf rust pathotype 77-5 (121R-63-1) was carried out by Saritha *et al.* (2005), the study revealed that Sel. T3171 exhibited resistance in seedling and in adult stage. The study on inheritance indicated that resistance against 77-5 pathotype governed by a single dominant gene.

Although the F_1 plants in AL x Sel.4805-1 and NI5439 x Sel.4805-1 crosses exhibited seedling resistance to the leaf rust pathotype 77-6, the F_2 population when tested with the same pathotypes segregated a ratio of 9 resistant : 7 susceptible seedlings.

The genetic analysis indicated digenic dominant nature of resistance (Table 12) against 77-6 pathotype in Sel. 4085-1. Complimentary resistance to leaf rust controlled by two dominant genes has been reported earlier by Fitzgerald *et al.* (1957) and to strip rust by Gurdev *et al.* (1988).

5.2.2 Stem rust resistance

Both the resistant lines, Sel.2429 and Sel.4805-1 showed resistance to pathotypes 40A and 117-6 of *P. graminis* f.sp. *tritici.* Genetic analysis was, therefore, carried out to find out the nature of inheritance and number of genes governing resistance to these pathotypes 40A and 117-6 of stem rust.

All the F_1 plants of the crosses, Agra Local x Sel.2429, Lal Bahadur x Sel. 2429 and WL711 x Sel.2429 showed resistant reaction in seedling stage indicating that the resistance to stem rust pathotypes 40A and 117-6 in Sel.2429 is dominant. The genetic analysis in F_2 generation in all the three crosses revealed a dominant monogenic resistance as the observed frequency of F_2 segregants fit well into 3R : 1S ratio. In the BC_1 generation derived from the crosses of all F_1 with susceptible parent(s), the resistant and susceptible seedlings fit well into 1 : 1 ratio with non-significant χ^2 value. This confirmed the dominant nature of resistance and also indicated that Sel.2429 carried a single dominant gene for resistance against 40A and 117-6 pathotypes of stem rust. *Ae. speltoides* derivatives possessing *Sr*32 show a high degree of resistance in adult stage (Menon and Tomar 2001). It was also reported that the line W 3531 carrying *Sr*32 also exhibited excellent mature plant resistance.

In order to understand the inheritance of resistance to stem rust pathotypes 117-6 and 40A in Sel.4805-1 the segregating generations of two corsses viz., Agra Local x Sel. 4805-1 and NI5439 x Sel.4805-1 were analysed genetically. The F_1 seedlings in all these crosses exhibited resistance revealing dominant nature of resistance. The segregation pattern of resistant and susceptible seedlings in F_2 generation fit well to the expected ratio of 3 : 1. The data indicated that resistance to 40A and 117-6 pathotype of stem rust is controlled by a single dominant gene. In BC_1F_1 generation, the frequency of resistant and susceptible plants fit well into an expected ratio of 1 : 1 confirming the F_2 hypothesis that resistance is indeed governed by a single dominant gene. The genetic analysis of different crosses reverealed that Sel.4805-1 also carried a single

dominant gene for resistance to 40A and 117-6 pathotypes of stem rust. The analysis for resistance in both the selections viz. Sel 2429 and Sel. 4805-1 derived from *Ae. spletoides* and *T. militinae* respectively indicated the dominant monogenic inheritance.

Majority of the already designated stem rust resistance genes are of dominant nature and under monogenic control (McIntosh *et al.*, 1995). Further, Sinha *et al.*, 2001 reported a similar genetic control for stem rust resistance introgressed from *T. militinae* into common wheat.

5.3 Test of allelism

The nature of leaf rust resistance in Sel. 2429 was under monogenic dominant control against the pathotypes, 77-5 and 77-7. To know whether Sel. 2429 carried resistance conferred by some of the alien genes which are effective against Indian leaf rust pathotypes. Therefore, Sel. 2429 was crossed with genetic stocks carrying known alien genes viz. *Lr*19 and *Lr*24 (Table 18) that are effective against a wide spectrum of races of *P. triticina* prevailing in India. The F_1 hybrids of both the crosses, HW 2043 (*Lr*19) x Sel. 2429 and HW 2003 (*Lr*24) x Sel. 2429 showed resistance in seedling stage against both the selected pathotypes. The F_2 population was tested with the pathotypes 77-5 and 77-7 of *P. triticina* that are more prevalent and virulent under Indian conditions.

The observed frequency of resistant and susceptible F_2 segregants in both the crosses HW2043 (*Lr*19) x Sel.2429 and HW2003 (*Lr*24) x Sel.2429 fit well into expected ratio of 15 : 1 ratio for the leaf rust pathotypes 77-5 and 77-7. The genetic analysis revealed that Sel. 2429 carries a different gene than that of *Lr*19 and *Lr*24, which also confer resistance against both the above mentioned leaf rust pathotypes.

Most of the native wheat genes are ineffective against a virulent and most prevalent pathotype 77-5 of leaf rust. The gene *Lr*9 from *Aegilops umbellulata* is overcome by 77-6 while *Agropyron elongatum* derived gene *Lr*19 has been rendered ineffective by 77-8 biotype of leaf rust. Only a few genes viz. *Lr*24, *Lr*28 and *Lr*32 which are occurring in Indian wheat varieties are effective against the pathotype 77-5 (Tomar and Menon, 2001). The gene *Lr*28 produces immune reaction to 77-5 in seedling stage while *Lr*32 exhibits infection type of 1, 2 in comparison to Sel. 2429

which showed 1 IT. It is presumed, therefore, the resistance present is Sel. 2429 in diverse of *Lr*24, *Lr*28 *and Lr*32.

Prabhu *et al.* (2003) developed a SCAR molecular marker tagged to *Agropyron elongatum* derived leaf rust resistance conferred by *Lr*24. While validating the marker on a pair of NILs carrying *Lr*19 from Sunstar*6/C80-1 and *Lr*24 from TR 380-14*7/ 3Ag# 14 revealed that each NILs pair possessed the same gene i.e. *Lr*24. These results were further confirmed by test of allelism between the donors. Presuming that HW 2043 used in present study, carried *Lr*19, therefore, it cannot be conclusively inferred that Sel.2429 imparts resistance which is diverse of *Lr*19. However, *Lr*19 has recently been overcome by a new virulence 77-8 in seedling as well as in adult stage (V.C. Sinha and S.M.S. Tomar unpublised data). It can, therefore, be assumed that Sel.2429 carried resistance, which is diverse than that of the genes, namely, *Lr*19, *Lr*24, *Lr*28 and *Lr*32.

5.4 Chromosomal location of leaf rust resistance in Sel. 2429 to the pathotype 77-5

Aneuploid analysis using complete set of monosomic lines in the background of Chinese Spring was carried out to assign the gene(s) for resistance to leaf rust on specific chromosomes. The resistance to pathotype 77-5 in Sel.2429 was controlled by a single dominant gene as evident from the present investigation. If a single dominant gene confers resistance, then all the cytologically identified monosomic F_1 plants including the critical line will be resistant. In other words the F_1 plants will be hemizygous resistant (R-). Selfing of these cytologically identified monosomic F_1 plants including the hemizygous ones will give rise to F_2 plants that are homozygous (RR), hemizygous (R-) and nullisomic (- -). Due to unequal gametic transmission rates, about 3 to 4 percent of the population are of nullisomics that show suscepcibility. As a result the frequency of resistant plants will be much higher than expected in the ratio favouring resistant category (McIntosh, 1987; Knott, 1989; Hussien *et al.,* 1997).

The segregation of F_2 generation derived from monosomic F_1 plants with significant chi-square value in the critical cross, CS 2D mono x Sel. 2429 ($P<0.001$) and non significant value in rest of the non-critical lines ($P>0.01$) indicated that the leaf rust resistance in Sel. 2429 is located on chromosome 2D (Table 19). The genes

already located for leaf rust resistance on chromosome 2D are *Lr*2a (Ausemus *et al.* 1946; Luig and McIntosh, 1968; Dyck and Samborski, 1974; Browder, 1980), *Lr. 2b* (Dyck and Samborski, 1974; Browder, 1980), *Lr*2c (Dyck and Samborski, 1974; Browder, 1980, *Lr*IS (Luig and McIntosh, 1968; McIntosh and Baker, 1968; Dyck and Johnson, 1983), *Lr*22a (Rowland and Kerber, 1974; Dyck and Johnson, 1983), *Lr*22b (Dyck, 1979) and *Lr*54 (McIntosh *et al.*, 2005). Further it will be useful if the location of the resistance is confirmed by molecular markers.

It appears contradictory that the source of resistance in Sel.2429 is *Ae speltoides* which carries SS genome and the resistance factor persent in the derivative is located in 2D chromosome. The occurrence of homoeologus pairing between S and D genome is likely in the presence of PhI genes which suppresses the gene Ph on chromosome 5B of wheat. The accession of *Ae speltoides* used in wide crossing might be possessing the PhI gene and hence the resistance source from S genome got translocated presumably to D genome.

5.5 Mode of Inheritance of Morphological Traits

Information on inheritance pattern and linkage between different morphological traits is useful for plant breeders, geneticists and cytogeneticists, as it facilitates the efficient utilisation of marker traits in wheat improvement programme. Further, the study on marker traits can provide information for locating the specific genes influencing quantitative traits. Although morphological markers are very few in wheat yet the accumulation of information regarding genetic markers in crops, particularly wheat, enables the development of well defined linkage maps which are indispensable tools for any breeder. The hairy leaf character is generally not found among cultivated tetraploid and hexaploid wheats. However, *Aegilops speltoides* derivative Sel. 2429 possesses hairy leaf sheath like that of its diploid parent and *Triticum militinae* derivative Sel. 4805-1 possesses hairy leaf and leaf sheath like that of its tetraploid parent. In the present study, leaf sheath pubescence in Sel.2429 and leaf and leaf sheath pubescence in Sel.4805-1 were studied to understand their genetic behaviour and their linkage with rust resistance. The results obtained during the investigation are discussed hereunder.

Sel.4805-1 with leaf sheath and leaf pubescence and Sel. 2429 with leaf sheath pubescence were crossed with Agra Local and NI5439 with contrasting character of not producing pubescence either on leaf or on leaf sheath.

The F_1 plant derived from the crosses of non pubescent (Glabrous) x pubescent leaf sheath showed presence of pubescence on sheath. In F_2 generation the observed frequency of pubescent sheath and non pubescent plants fit well into 3 : 1 ratio indicating that leaf sheath pubesence is determined by a single dominant gene in Sel.2429. This hypothesis was completely supported by the genetic analysis of BC_1F_1 (Sel.2429/ AL/ /AL) populations as the data fits well into 1P : 1G ratio.

Similarly, the F_2 generation of the cross Sel.4805-1 x NI5439 segregated into 3 pubescent : 1 glabrous ratio indicating dominant monogenic inheritance. Lange and Jochem Sen (1987) reported that hairy leaf sheath is controlled by a single dominant gene in *T. dicoccoides*. Also certain hexaploid wheats derived from common wheat x *T. dicoccoides* G25 showed hairy leaf sheath which is also controlled by a single dominant gene; complementary genes also determined hairy leaf sheath trait in certain accessions of *T. dicoccoides* (McIntosh *et al.* 1998).

The leaf pubescence character in Sel.4805-1 was found to be controlled by two duplicate dominant genes. The F_1 plants derived from the cross between non pubescent (G) x pubescent (Hairy leaf =Pb) showed pubescence on leaves indicating the dominance of the trait. In F_2 generation the observed number of plants fit well into an expected ratio of 15P : 1G and the data in BC_1F_1 generation was in agreement with the ratio of 3P : 1G (Table 21). The genetic analysis revealed that hairy leaf character is governed by two dominant duplicate genes. However, Maystrenko (1976) identified only one dominant gene controlling hairiness of leaves in hexaploid wheat Militurum 321, which was located on chromosome 4A. Kuspira *et al.* (1989) concluded that only one major gene locus with a multiple allelic series appears to be involved in determining the pubescence in *T. monococum*. He provided evidence for at least three alleles at this locus in *T. monococcum* but with the reversal of chromosome designation for 4A and 4B, this locus in *T. monococcum* cannot be allelic to the gene in *T. aestivum*. Digenic control of leaf pubescence was also recorded by Neatby Goulden (1994) and Dhakate (2002). The results obtained in present study indicate that there are two duplicate dominant gene governing leaf pubescence in Sel.4805-1 derived from

T. militinae a mutant of *T. timopheevi*. The difference in intensity of leaf pubescence was, however, noticed which revealed that they could be independent of each other.

5.6 Linkage analysis

For genetic mapping detection of linkage between different genes is a pre-requisite. The analysis of linkage between genes provide an indication for assigning different markers to a specific linkage group. For practical purposes the knowledge of linkage among various traits may be useful in selection. Therefore, linkage analysis has important significance in all the crops. However, very few reports are available on linkage between morphological characters and the characters of economic value.

Linkage analysis was carried out between leaf pubescence and sheath pubescence in Sel.4805-1 (Table 23). The non-significanct χ^2 indicates that there is absence of linkage between leaf pubescence and leaf sheath pubescence in Sel. 4805-1.

Detection of linkage was also carried out between the morphological traits (leaf and leaf sheath pubescence) and rust resistance in Sel.2429 and Sel.4805-1 (Tables 22 and 23). In all the cases non-significant χ^2 value was observed in individual crosses. The genetic analysis clearly indicated the absence of linkage between them. Howes (1986) reported linkage between leaf rust resistance gene *Lr*10 with two endosperm proteins and hairy glumes in hexaploid wheat. Panin and Netsvetaev (1986) found some genetic control of glidine and the spike morphology in durum wheat. Generally, in translocations when alien segment is transferred to receipient variety, the segment is usually found to carry more than one gene. For example, the resistance gene *Lr*19, *Lr*24, *Lr*37 are linked with *Sr*25, *Sr*24, *Sr*38 respectively (Sharma and Knott, 1966; McIntosh *et al.*, 1977; Bariana and McIntosh, 1993).

5.7 Comparison of number of bivalents and univalents observed in F_1 plants of CS monosomic series x Sel. 2429.

The frequency of univalents in monosomic F_1 plants of 4B x Sel.2429, 5A x Sel.2429 and 7B x Sel.2429 was comparatively very high ranging from 1 to 7 chromosomes per cell indicating some influence of Sel.2429 chromosomes on homologous chromosome pairing. It is presumed that the segment of *Aegilops speltoides* present in Sel.2429 affects the homologous chromosome pairing. It is also likely that the segment

is restricted to affect only chromosome pairing and induces desynapsis. It is also presumed that the segment translocated from *Aegilops speltoides* to wheat may carry either of desynaptic or gametocidal gene as some of the F_1 hybrids were partially male sterile. Genes inducing desynapsis are known in *T. timopheevi*. In the presence of gametocidal genes the transmission of certain gene(s) is affected in heterozygous condition, however, the degree of distortion is determined by the genetic background. Gametocidal genes have also been identified in several species belonging to sitopsis group of *Aegilops*. Gametocidal genes also suppresses the activity of 3C chromosome gametocidal gene of *Aegilops triuncialis* (Tsujimoto and Noda, 1988). This alien gametocidal factor also promotes chromosome breakage (Tsujimoto and Tsumewaki, 1985).

SUMMARAY AND CONCLUSIONS

Genetic diversity serves a useful purpose the way it can be explored in wheat breeding. Progenitors of wheat, allied and alien species of wheat constitute potential reservoir for desirable genes for wheat improvement. A number of genes providing resistance to wheat foliar diseases, particularly rusts, have been transferred from related species to wheat. The stability in wheat production has been achieved through sustained efforts in bringing about varietal diversity for resistance to biotic and abiotic stresses. In this endeavour the role of breeding for disease resistance has been the most important as the wheat crop is always vulnerable to new virulence(s) of pathogens appearing frequently and posing threat to yield stability. Though several native and alien gene sources have been extensively used for incorporating resistance against rusts. To incorporate desirable alien gene(s), specific cytogenetical techniques are needed, while exploitation of species, having common genome(s) with common wheat can be transferred with relative ease. In one such effort, Division of Genetics has exploited two related species, namely, *Aegilops speltoides* and *T. militinae* for leaf rust and stem rust resistance in common wheat. As a result two genetic stocks namely Sel.2429 and Sel.4805-1 were developed involving above mentioned species and common bread wheat. The above mentioned genetic stocks were found resistant to leaf rust and stem rust under natural conditions at hot spot location, Wellington, the Nilgiris, South India.

In addition to resistance, Sel.2429 possesses pubsescent leaf sheath and Sel.4805-1 produces both pubescent leaf and its sheath. In the present study, the identification of resistance genes, their mode of inheritance with respect to leaf rust pathotypes viz., 77-5, 77-6 and 77-7 and stem rust pathotypes namely, 40A and 117-6 has been investigated. The location of leaf rust resistance gene on specific chromosome was also studied. Mode of inheritance of morphological traits viz., sheath pubescence in Sel.2429 and leaf and leaf sheath pubescence in Sel.4805-1 were also studied. Investigation on linkage between morphological traits and rust resistance was also carried out. The salient findings of the study are summarized hereunder.

Inter-specific derivatives viz., Sel.2429 and Sel.4805-1 and the parental lines, namely, *Ae. speltoides, T. militinae and T. aestivum* were tested with three pathotypes 77-5, 77-6 and 77-7 of *Puccinia triticina* and two pathotypes, 40A and 117-6 of *Puccinia graminis tritici* for determining the infection spectrum. Both the derivatives alongwith donors conferred resistance to all the selected pathotypes of stem rust and leaf rust pathogenes and infection type ranged from IT '0;' to IT '1", while *T. aestivum* stocks showed susceptible infection type 3 to 4. The infection type in derivatives was recorded "1" as compared to donor species.

Segregating generations viz., F_2 and BC_1F_1 of crosses involving Sel. 2429 and Sel.4805-1 with susceptible lines Agra Local, Lal Bahadur, WL 711 and NI5439 were tested with selected pathotypes of leaf rust and stem rust pathogens.

- Seedling tests of Sel.2429 with leaf rust pathotypes 77-5 and 77-7 indicated the presence of one dominant gene for resistance.
- The results of seedling test revealed that Sel.2429 possess one recessive gene for resistance against 77-6 pathotypes of leaf rust.
- Sel.2429 was tested with two stem rust pathotypes 40A and 117-6. The segregation pattern in F_2 and BC_1F_1 generation indicated that this derivative also carries a dominant gene for resistance to aforesaid pathotypes of stem rust.
- *T. militinae* derivative Sel.4805-1 was tested with three leaf rust pathotypes. A single dominant gene controlling resistance against 77-5 and 77-7 pathotypes was identified while two genes governing resistance against 77-6 were determined.
- Sel. 4805-1 carries a single dominant gene for resistance to stem rust pathotypes 40A and 117-6.
- Results of test of allelism revealed that the leaf rust restitance identified in Sel.2429 to the pathotypes 77-5 and 77-7 is different than that of alien genes *Lr*19 and *Lr*24.
- Aneuploid analysis using complete sets of monosomic series in the background of Chinese Spring revealed that the resistance in Sel. 2429 against most virulent leaf rust pathotype 77-5 is located on chromosome 2D.

- The analysis of leaf sheath pubescence in Sel.2429 revealed that it is controlled by a single dominant gene.
- There is no linkage observed between leaf sheath pubescence and leaf rust and stem rust resistance in Sel.2429.
- The resistance identified in the interspecific derivatives is diverse, hence useful in wheat breeding.
- Genetic studies in Sel.4805-1 revealed that leaf sheath pubescence is controlled by a single dominant gene and leaf pubescence is controlled by two duplicate dominant genes.
- No linkage was found between leaf rust resistance and leaf and sheath pubescence in Sel. 4805-1.
- The cytogenetic analysis of F_1 plants of cross CS monosomics x Sel.2429 revealed that there is significant difference in number of bivalents observed between disomic and monosomic plants. The segment carried by Sel. 2429 affects the chromosome pairing.
- Significant differences were also observed in number of bivalents present in Ist meiotic metaphase among different monosomic F_1 plants.

The presence of resistance genes for leaf rust and stem rust in interspecific derivatives would be of immense value in diversifying the genetic base for resistance to rusts in wheat. The judicious use of the diverse genes for resistance to stem rust and leaf rust may provide protection to wheat crop from rust epidemics in order to minimize the loss caused due to rust infection. It is suggestd that molecular markers linked to newly identified genes should be developed for rapid breeding for rust resistance in wheat.

ABSTRACT

Wheat is one of the most important cereal crop in India. Its production and productivity are affected by both biotic and abiotic stresses. Among biotic stresses, fungal diseases, especially rusts cause severe damage to wheat crop. The genetic analysis of rust resistance was carried out in two interspecific derivatives namely, Sel. 2429 derived from wheat X *Aegilops speltoides* and Sel. 4805-1 selected from wheat X *Triticum militinae* crosses. Both these derivatives exhibited high degree of resistance to leaf rust and stem rust in seedling as well as in adult stage under natural and artificial epiphytotic conditions. In addition, these derivatives carry pubescence on leaf and leaf sheath. Three pathotypes of *Puccinia triticina* viz., 77-5, 77-6 and 77-7 and two of *Puccinia graminis tritici* namely, 40A and 117-6 were selected for determining the resistance against each rust.

The seedling resistance to the leaf rust pathotypes 77-5 and 77-7 in Sel.2429 is determinied by a single dominant gene while against 77-6 pathotype the resistance was controlled by a single recessive gene. Similarly, *Triticum militinae* derivative Sel. 4805-1 showed monogenic dominant resistance to leaf rust pathotypes 77-5 and 77-7 and digenic dominant resistance against the pathotype 77-6. Both the lines, Sel. 2429 and Sel.4805-1 showed monogenic dominance resistance against the stem rust pathotypes 40A and 117-6.

Results of test of allelism indicated that the resistance genes present in Sel. 2429 to the leaf rust pathotypes 77-5 and 77-7 is different than that of *Lr* 19 and *Lr* 24. Also the reaction pattern to the selected pathotypes indicated that these derivatives do not carry the genes *Lr* 9, *Lr* 20 *Lr* 23 and *Lr* 26, since these genes *Lr* 23 and *Lr* 26 are ineffective against 77-5; *Lr* 20, *Lr* 23 and *Lr* 26 are ineffective to 77-6 and *Lr* 9, *Lr* 23 and *Lr* 26 are over come by 77-7. The aneuploid analysis using Chinese Spring monosomic series revealed that the resistance gene to leaf rust pathotype 77-5 in Sel 2429 is located on the chromosome 2D. One dominant gene is identified for leaf sheath pubescence in Sel.2429. This leaf sheath pubescence is not linked with stem rust and leaf rust resistance in Sel.2429. Studies on morphological traits in Sel.4805-1 revealed that leaf sheath pubscence is controlled by a single dominant gene while leaf pubescence is controlled by two duplicate dominant genes. No linkage was found btween leaf rust resistance and leaf and sheath pubescence in Sel.4805-1.

सार

गेहूं भारत की सबसे महत्त्वपूर्ण फसल है। इसका उत्पादन और उत्पादकता दोनों ही जैविक और अजैविक बाधाओं से प्रभावित होते हैं। जैविक बाधाएं जैसे फफूंद रोग, विशेष रूप से रतुए से, इस फसल को गंभीर क्षति होती है। रतुए की प्रतिरोधिता का आनुवंशिक विश्लेषण दो अन्तःविशिष्ट व्युत्पन्नों नामतः गेहूं × *एजीलॉप्स स्पेल्टोइडस* से व्युत्पन्न Sel 2429, तथा गेहूं × *ट्रिटिकम मिलिटिनी* से व्युत्पन्न Sel 4805-1 से किया गया। इन दोनों ही व्युत्पन्नों ने प्राकृतिक और कृत्रिम पादप अवस्थाओं के तहत पौद अवस्था के साथ-साथ प्रौढ़ अवस्था में भी पत्ती रतुआ और तना रतुआ दोनों के प्रति उच्च प्रतिरोधिता प्रदर्शित की। इसके अलावा इन दोनों व्युत्पन्नों में पर्ण और पर्ण आच्छद रामिलता (लीफ शीथ प्यूबीसैंस) भी होती है। *पक्सीनिया ट्रिटीसीना* के तीन रोगप्ररूपों, नामतः 77–5, 77–6 और 77–7 तथा *पक्सीनिया ग्रेमिनिस ट्रिटिकी* के दो रोगप्ररूपों नामतः 40A और 117–6 का चयन प्रत्येक रतुए के प्रति प्रतिरोधिता निर्धारित करने के लिए किया गया।

पत्ती रतुए के, Sel 2429 में रोगप्ररूपों 77–5 और 77–7 का निर्धारण एक एकल प्रभावी जीन द्वारा होता है, जबकि रोगप्ररूप 77–6 के प्रति प्रतिरोधिता एक एकल अप्रभावी जीन से नियंत्रित होती है। इसी प्रकार Sel 4805-1 के व्युत्पन्न *ट्रिटिकम मिलिटिनी* द्वारा पत्ती रतुआ रोगप्ररुपों 77–5 और 77–7 के प्रति एकलिंगी प्रबलित प्रतिरोधिता तथा रोगप्ररुप 77–6 के प्रति द्विलिंगी प्रबलित प्रतिरोधिता प्रदर्शित की गई। दोनों ही वंशक्रमों Sel.2429 और Sel.4805-1 ने तना रतुआ रोगप्ररुपों 40A और 117–6 के प्रति एकलिंगी प्रबलित प्रतिरोधिता प्रदर्शित की।

युग्मविकल्पता (एलेलिज़्म) के परीक्षण के परिणामों से संकेत मिला कि Sel.2429 में उपस्थित पत्ती रतुआ रोगप्ररुपों 77–5 और 77–7 के प्रतिरोधी जीन *Lr19* और *Lr24* से भिन्न है। इसके अतिरिक्त चुने गए रोगप्ररुपों के प्रतिक्रिया रुझान से यह संकेत मिला कि इन व्युत्पन्नों में *Lr 9, Lr 20, Lr 23* और *Lr 26* जीन नहीं होते हैं क्योंकि *Lr 23* और *Lr 26* जीन 77–5 के प्रति अप्रभावी; *Lr20, Lr23* और *Lr26* जीन 77–6 के प्रति अप्रभावी होते हैं तथा *Lr9, L23* और *Lr26* जीन 77–7 द्वारा अतिक्रमित हो जाते हैं। चाइनीज़ स्प्रिंग मोनोसोमिक श्रृंखला से यह स्पष्ट हुआ कि Sel 2429 में पत्ती रतुआ रोगप्ररुप 77–5 का प्रतिरोधी जीन 2D गुणसूत्र पर स्थित होता है। Sel.2429 में पर्ण आच्छद रामिलता के लिए एक प्रभावी जीन को पहचाना गया है। यह पर्ण आच्छद रामिलता Sel.2429 में तना रतुआ और पत्ती रतुआ प्रतिरोध से संबंधित नहीं है। Sel.4805-1 में आकृतिविज्ञानी विशेषकों संबंधी अध्ययन से यह स्पष्ट हुआ है कि पर्ण आच्छद रामिलता इकहरे प्रभावी जीन द्वारा नियंत्रित होता है, जबकि पत्ती रामिलता दो डुप्लीकेट प्रभावी जीनों द्वारा नियंत्रित होता है। पत्ती रतुआ प्रतिरोध तथा पत्ती और आच्छद रामिलता के बीच Sel.4805-1 के बीच कोई संबंध नहीं पाया गया।

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