

# **Enhancing the Genetic Diversity and Durability of Leaf Rust Resistance in Durum Wheat**

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## Abstract

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The importance of leaf rust, caused by *Puccinia triticina*, has increased dramatically in recent years in durum wheat (*Triticum turgidum* ssp. *durum*) worldwide. Little is known on the occurrence and nature of resistance in this crop. Thirty durum wheat lines derived from the International Maize and Wheat Improvement Center (CIMMYT) were characterized for their resistance to the Mexican *P. triticina* race BBG/BN which was identified in 2001 and caused susceptibility of a large number of the world's durum wheat cultivars.

Ten genotypes with race-specific resistance displayed low to intermediate seedling reactions to leaf rust. In the field, eight genotypes were immune and two displayed moderate levels of resistance. The slow rusting resistant lines displayed a range of disease severity responses indicating genetic diversity.

The yield protection conferred by race-specific and slow rusting resistance was investigated in yield loss trials under high leaf rust pressure in the field. Race-specific resistance provided effective protection against yield losses caused by leaf rust. Yield losses for slow rusting resistant lines were higher than for immune race-specific resistant ones, but significantly lower than for the susceptible checks. Slow rusting lines with high resistance levels and reduced yield losses were identified.

The slow rusting components; latent period, uredinium size and receptivity, were determined in greenhouse experiments, and associations of these components with leaf rust progress in the field were calculated. The results indicated that predominantly uredinium size contributed to slow leaf rust progress in durum wheat.

The genetic basis and diversity of race-specific resistance was also determined in progenies from crosses of nine durum wheat lines with a leaf rust susceptible parent, and from intercrosses among the resistant parents. Five distinct sources of resistance were identified, four of which involved single partially, or completely, dominant genes, of which two were closely linked, and a pair of partially dominant complementary genes. Using molecular tools, the two linked genes were located on the long arm of chromosome 6B.

The best slow rusting resistant lines and the five distinct race-specific resistance sources can be used for enhancing the diversity and durability of leaf rust resistance in durum wheat.

Keywords: *Triticum turgidum* ssp. *durum*, *Puccinia triticina*, *Puccinia recondita* f. sp. *tritici*, genetic resistance, control, yield losses, inheritance studies, mapping.

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*Para mi familia con todo mi cariño*

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- II. Herrera-Foessel, S.A. Singh, R.P., Huerta-Espino, J., Crossa, J., Djurle, & A., Yuen, J. 2007. Evaluation of slow rusting resistance components to leaf rust in CIMMYT durum wheats. *Euphytica* (accepted).
- III. Herrera-Foessel, S.A. Singh, R.P., Huerta-Espino, J., Yuen, J., & Djurle, A. 2005. New genes for leaf rust resistance in CIMMYT durum wheats. *Plant Disease* 89: 809-814.
- IV. Herrera-Foessel, S.A. Singh, R.P., Huerta-Espino, J., William M., Rosewarne, G., Djurle, A., & Yuen, J. 2007. Identification and mapping of *Lr3* and a linked leaf rust resistance gene in durum wheat. *Crop Science* (accepted).

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## Introduction

Wheat is on a global basis the most widely grown cereal grain and occupies 17% (220 million hectares) of the total cultivated land in the world. It is the main staple food for 35% of the total world's population. The two most common types of wheat are bread wheat and durum wheat. Durum wheat is grown on approximately 17 million hectares in the world and about half of the area is in developing countries where durum wheat is used for making a range of products (CIMMYT, 2006). The wheat rusts (leaf rust, yellow rust and stem rust) have historically been diseases of great importance and they have significantly influenced the development of human civilisation (Roelfs *et al.*, 1992; McIntosh *et al.*, 1995). Early records of devastations of rust have for example been described in the Bible and in early Greek and Roman literature (Roelfs *et al.*, 1992; McIntosh *et al.*, 1995). Host plant resistance is the most effective way to protect wheat from losses due to rust diseases. However, breakdown of the resistance to leaf rust in durum wheat was recently reported in several countries. For example leaf rust epidemics during 2001 to 2003 severely affected a durum wheat production area of 250,000 hectares in north-western Mexico causing estimated losses of at least US\$32 million (Singh *et al.*, 2004). The principal source of durum wheat germplasm for the developing world is from the International Maize and Wheat improvement Center (CIMMYT), in Mexico. The main objective of this study was to reduce genetic vulnerability of durum wheat to leaf rust epidemics in developing countries by enhancing the genetic diversity and durability of leaf rust resistance in CIMMYT durum wheat germplasm.

## Background

### *The host: general genetic aspects of durum wheat*

Durum wheat (*Triticum turgidum* var. *durum*) is grown on 8-10% of the total cultivated wheat area worldwide (Mac Key, 2005; Kantety *et al.*, 2005). The durum wheat production in the developing world is concentrated in the Middle East, Central India, and the Mediterranean region of West Asia and North Africa (WANA). Other production areas include Ethiopia, Argentina, Chile, Russia, Kazakhstan and Mexico. In developing countries, durum wheat is produced in areas where it plays an important role for food security and livelihoods of millions of resource-poor farmers and their families (Ammar *et al.*, 2006). During 1991-1997 more than 90% of the durum wheat cultivars released in developing countries were introduced, or derived from, germplasm developed at CIMMYT (Pfeiffer & Payne, 2005). Durum wheat is used for the production of pasta products, couscous, bulgur, frekeh, leavened and flat bread and other regional dishes in WANA and the Mediterranean basin. It is also used for making other products such as chapatis in the Indian subcontinent, leavened bread in Caucasus, and tortillas and mote in Central and South America (Pena & Pfeiffer, 2005).

Wheat belongs to the grass family Poaceae (=Gramineae) which includes approximately 10,000 different species (Levy & Feldman, 2002), and the

subgroup or tribe, *Triticeae* Dumort (Mac Key, 2005). The classification of the genus *Triticum* and other related genera within the tribe have been under discussion, particularly in combining the species of *Aegilops* into *Triticum* (Mac Key, 1966; Gupta & Baum, 1986). Several species with different ploidy-levels are grouped in the genus *Triticum*. In Table 1 the members of the *Triticum* genera (*sensu stricto*) are presented according to Feldman (2001).

The development of wheat into different polyploidy series is a classical example of allopoloidization. Allopolyploids (or amphiploids) contain two or more diverged homoeologous genomes derived from hybridization of species (Levy & Feldman, 2002). The hybrid that results from such a cross is fertile only through chromosome doubling (Levy & Feldman, 2002).

Durum wheat (*T. turgidum* ssp. *durum* Desf.) is an allotetraploid ( $2n = 28$ , AABB) that arose from hybridization followed by chromosome doubling of a cross between *Triticum urartu* (genome AA) and a species related to *Aegilops speltoides* (genome BB). The true origin of the B genome has been under discussion for a long time and still remains elusive (Levy & Feldman, 2002; Huang *et al.*, 2002). Bread wheat is an allohexaploid ( $2n = 42$ , AABBDD) and evolved through a cross of tetraploid *Triticum turgidum* (AABB) and the diploid *Aegilops tauschii* (= *Ae. squarrosa*) (genome DD), which was followed by chromosome doubling (Levy & Feldman, 2002).

Allopolyploids are characterized by their bivalent pairing and disomic inheritance. Pairing at meiosis only occurs between truly homologous chromosomes and very seldom across homoeologous ones (Mac Key, 2005). A great discovery was that bivalent pairing was determined mainly by one major suppressor gene, *Ph1*, located on chromosome 5BL (Riley & Chapman, 1958; Sears & Okamoto, 1958). The control of meiotic pairing was later found to depend on the balance of several suppressor and modifying genes (Kimber & Sears, 1987).

The tetraploid wheats can be divided in two groups of species, the emmer group ( $2n=28$ , AABB) and the *Timopheevi* group ( $2n=28$ , AAGG). The domestication of wild types *T. araraticum* and *T. dicoccoides* led to the development of *T. timopheevi* and *T. dicoccum*, respectively (Bozzini, 1988). The cultivated areas of *T. timopheevi* remained limited geographically to Armenia and Transcaucasia, whereas *T. dicoccum* spread from the Near East to large areas of the Mediterranean and Middle East, Egypt and Ethiopia (Bozzini, 1988). Durum wheat is the most important cultivated tetraploid wheat today and is grown in many countries.



Table 1. Species, subspecies and common names of *Triticum* members

Diploids $2n=2x=14$	Tetraploids $2n=4x=28$	Hexaploids $2n=6x=42$
<p><b><i>Triticum monococcum</i> L. (AA)</b>  <i>ssp. aegilopoides</i> (Link) Thell. - Wild einkorn  <i>ssp. monococcum</i> - Cultivated einkorn or small spelt</p>	<p><b><i>Triticum timopheevii</i> (Zhuk.) Zhuk (AAGG)</b>  <i>ssp. armeniacum</i> (Jakubz.) Van slageren - Wild timopheevii  <i>ssp. timopheevii</i> - Cultivated timopheevii</p>	<p><b><i>Triticum zhykovski</i> Men. &amp; Er. (AAAAGG)</b></p>
<p><b><i>Triticum urartu</i> Tum. Ex Gand. (AA)</b></p>	<p><b><i>Triticum turgidum</i> L. (Thell) (AABB)</b>  <i>ssp. dicoccoides</i> (Korn. Ex Asch. &amp; Graebn.) Thell - wild emmer wheat  <i>ssp. dicoccon</i> (Schränk) Thell. (= <i>dicoccon</i>) - cultivated emmer  <i>ssp. paleocolchicum</i> (Men.) A. Love &amp; D. Love  <i>ssp. parvicoccum</i> Kislev - Georgian wheat</p>	<p><b><i>Triticum aestivum</i> L. (AABBDD)</b>  <i>ssp. spelta</i> (L.) Thell. - Dinkel or large spelt  <i>ssp. macha</i> (Dek. &amp; Men) MK  <i>ssp. aestivum</i> - Common or bread wheat  <i>ssp. compactum</i> (Host) MK - Club wheat  <i>ssp. sphaerococcum</i> (Percival) MK - Indian dwarf or shot wheat</p>
	<p><b><i>ssp. durum</i> (Desf) Husn. Macaroni, durum or hard wheat</b>  <b><i>ssp. turgidum</i> - Rivet, cone or pollard wheat</b>  <b><i>ssp. polonicum</i> (L.) Thell. - Polish wheat</b>  <b><i>ssp. turanicum</i> (Jakubz.) A. Love &amp; D. Love - Khorassan wheat</b>  <b><i>ssp. carthlicum</i> (Nevski) A. Love &amp; D. Love - Persian wheat</b></p>	

Modified after Feldman (2001)

The discovery in the 1930s that colchicine can induce chromosome doubling opened the possibilities of further studying wheat evolution by mimicking polyploidization events (Feldman, 2001). The tetraploidization event for the formation of wild emmer wheat (*Triticum turgidum* ssp. *dicoccoides*, genome AABB), the progenitor of durum wheat (*Triticum turgidum* ssp. *durum*, genome AABB), occurred a few hundred thousand to half a million years ago (Huang *et al.*, 2002; Levy & Feldman, 2002). *T. timopheevii* (genome AAGG) was formed later than wild emmer wheat as it has shown less genetic variation than the emmer group (Mori *et al.*, 1995). The hybridization event that led to the formation of bread wheat (*T. aestivum*) was more recent and occurred approximately 8,000 to 10,000 years ago (Huang *et al.*, 2002; Levy & Feldman 2002).

The domestication of diploid wheat occurred in the northern Levantine Corridor of the Fertile Crescent in the Near East, while domesticated tetraploid wheat originated at the watershed of the Jordan River in the southern Levantine Corridor. The first indication of the cultivation of wild emmer wheat is in the Prepottery Neolithic A period around 10,300-9,500 years ago. Domesticated non-brittle as well as naked forms of emmer wheat appeared in the Prepottery Neolithic B period, 9,500-7,500 years ago (Feldman, 2001).

The geographical origin of hexaploid wheat, where hybridization occurred between domesticated tetraploid wheat and *Ae. tauschii*, is southwest of the Caspian Sea, in western Iran (Feldman, 2001; Levy & Feldman 2002). Other centers of variation or diversity for tetraploid wheat is the Ethiopian plateau, the Mediterranean basin and the Transcaucasia (Feldman, 2001). Ethiopia was considered by Vavilov (1951) to be the centre of origin of tetraploid wheat, but was later changed to be the centre of diversity (Feldman, 2001).

The genome sizes differ for the members of the grass family, from 450 Mb for rice to 16,000 Mb for hexaploid wheat (Arumuganathan & Earle, 1991). The genome sizes of diploid and tetraploid wheat are estimated to be 5,600 Mb and 13,000Mb, respectively (Arumuganathan & Earle, 1991). This variation in the grass family is in part due to differences in ploidy level but mainly due to the amount of repetitive DNA (Keller, 2005). The size of the bread wheat genome is five times larger than the human genome (Keller, 2005).

#### *The pathogen: general aspects of Puccinia triticina*

Leaf rust caused by *Puccinia triticina* Erikss. is the most common and widely distributed of the wheat rusts and continues to pose a major threat to wheat production in many countries (Knott, 1989). Total crop losses due to leaf rust are rare but yield reductions up to 62% are reported under conditions favorable for disease build-up (Sayre *et al.*, 1998). On a world wide basis leaf rust causes more damage than the other wheat rusts (Samborski, 1985).

Table 2. Uredinial-telial and pyrenial-aecial hosts of *P. trititica* and *P. recondita*

Group	Uredinial-telial host		Pyrenial-aecial host	
	Principal	Additional	Family	Principal Additional
<b>I.</b> <i>Puccinia trititica</i>	<i>T. aestivum</i>	<i>Aegilops longissima</i> etc.	Ranunculaceae	<i>Thalictrum speciosissimum</i> <i>Thalictrum</i> spp.
	<i>T. turgidum</i> ssp. <i>durum</i>			
	<i>T. turgidum</i> ssp. <i>dicoccoides</i>			
	Triticale			
<b>II.</b> <i>Puccinia recondita</i>	Type A.	<i>T. aestivum</i> <i>T. turgidum</i> ssp. <i>dicoccoides</i>	Boraginaceae	<i>Anchusa italica</i> None known
	Type B.	<i>Aegilops biuncialis</i> etc.	Boraginaceae	<i>Echium glomeratum</i> <i>Anchusa undulata</i> , <i>Lycopsis arvensis</i>
	<i>Aegilops ovata</i>			
	Type C.	<i>Aegilops longissima</i> etc.	Boraginaceae	<i>Anchusa aggregata</i> <i>Anchusa undulata</i> etc.
	Type D.	<i>Secale montanum</i>	Boraginaceae	<i>Lycopsis arvensis</i> <i>Anchusa undulata</i>

Modified after Annikster et al. (1997)

The wheat rusts belong to the genus *Puccinia*, the family Pucciniaceae of the order Uredinales and class Basidiomycetes (Knott, 1989). The rust fungi are obligate biotrophs. *P. triticina* has a complex life cycle with sexual and asexual spore stages (basidial, pycnial, aecial, uredinial and telial) and host alternation (Roelfs *et al.*, 1992).

For all wheat rust fungi the urediniospores play the most important role in the propagation of disease. Large numbers of urediniospores are produced by the uredinia over a period of several weeks causing rapid multiplication of inoculum (Roelfs *et al.*, 1992). The uredinial cycle repeats every 8 to 20 days depending on temperature and other environmental conditions (Chester, 1946). Urediniospores are wind-borne and germinate in the presence of free water. They have a great dispersal capacity and can be carried by wind over long distances (Roelfs *et al.*, 1992).

Durum wheat is the host of two different species causing leaf rust; *Puccinia triticina* Eriks. and *P. tritici-duri* = *P. recondita* f. sp. *tritici*, type A (Anikster *et al.*, 1997) (Table 2). The main telial-uredinial host of *P. triticina* is *T. aestivum* and *T. turgidum* ssp. *durum* although its host range is broad and includes cultivated and wild wheats, triticale, rye and wild barley (Anikster *et al.*, 1997). The main pycnial-aecial host of *P. triticina* is *Thalictrum speciosissimum* from the Ranunculaceae family. The scientific name *P. recondita* f. sp. *tritici* was previously used for *P. triticina*, but the wheat leaf rust pathogen has been shown morphologically distinct and genetically isolated from the leaf rust species normally attacking rye (*P. recondita*) (d'Oliveira & Samborski, 1966; Savile, 1985; Anikster *et al.*, 1997).

*P. tritici-duri* has *Anchusa italica* as its principal pycnial-aecial host and *T. turdigum* ssp. *durum* as its principal telial-uredinial host but is also compatible with *T. aestivum* (Table 2). *P. tritici-duri* is geographically limited to areas where the alternate host occurs and has been found in Morocco and Portugal (d' Oliveira & Samborski, 1966; Anikster *et al.*, 1997). *P. triticina* has, on the other hand, been successfully escorted by cultivated wheats around the world ahead of the limits of *T. speciosissimum* and its sexual cycle (Anikster *et al.*, 1997).

#### *Host – pathogen interaction*

The most efficient and environmentally friendly method to reduce yield losses due to the leaf rust pathogen is to use resistant cultivars (Knott, 1989). Chemical control is not justified under low yielding and low priced circumstances such as those found in many developing countries. Several rust resistance genes have been identified and used in breeding for resistance (McIntosh *et al.*, 1995a), but variants of the pathogen (referred to as races) that can overcome the resistance in one or several of these resistant cultivars can evolve.

A race or pathotype can be defined as an individual or group of biotypes that give the same combination of high (susceptible response) and low (resistant response) infection type responses on a specified set of differential host cultivars

(Browder, 1971). New races of the rust fungi evolve constantly in nature (Samborski, 1985) through mechanisms such as mutation, genetic drift, gene flow, sexual and asexual recombination, and selection (McDonald & Linde, 2002). Mutation is considered to be the most important source of variation and evolutionary force in rust fungi (Samborski, 1985). Mutation coupled with efficient directional selection (widespread deployment of a resistance gene) contributes to a rapid increase of virulent races in a population (McDonald & Linde, 2002). Other mechanisms that may contribute to evolution, but are not thought to play a major role at least for *P. triticina*, are sexual recombination on the alternate host and somatic hybridization (asexual recombination via anastomosis of germ tubes and hyphae) (Samborski, 1985). Introduction of new genotypes by migration of spores is an important mechanism that allows exchanges between geographically separated populations (McDonald & Linde, 2002).

Stripe rust was the first disease for which host resistance was shown to be an inherited trait that follows the rules of Mendelian genetics (Biffen, 1905). Stakman & Levine (1922) later demonstrated physiological specialization of *P. graminis* (causing stem rust) and that the resistance to stem rust can be overcome by variants of the pathogen. Flor (1956) studied inheritance of pathogenicity in the pathogen and inheritance of resistance in the host using flax (*Linum usitatissimum*)-flax rust (*Melampsora lini*) as a model system and concluded that “for every gene that conditions resistance in the host there is a corresponding gene in the parasite that conditions pathogenicity”. This concept or model is known as the “gene-for-gene theory” (Flor, 1971) and has been demonstrated in other systems such as for wheat and wheat rusts (Luig & Watson, 1961). In this model it is presumed that the host genes for resistance are dominant and the genes for virulence (the ability of the pathogen to overcome the host gene for resistance) are recessive. Low infection type response occurs only when the host carries a gene for resistance for which the pathogen does not carry the corresponding gene for virulence. Hence, the recognition of an avirulent pathogen by a resistant host leads to incompatibility, i.e. resistance. According to this model, susceptibility, or compatibility, occurs either because the pathogen carries the corresponding gene for virulence or the host does not carry the gene for resistance to which the pathogen is virulent or avirulent. Most of the leaf rust resistance genes (*Lr*) in Table 3 have been demonstrated to follow the gene-for-gene relationship (McIntosh *et al.*, 1995a), but some exceptions have also been reported (Vanderplank, 1968; Parlevliet, 1985; Kolmer & Dyck, 1994; Kolmer, 1996).

#### *Race-specific and slow rusting resistance*

Two different types of resistance are often described in the literature against specialized fungi that parasitize living cells have been described: race-specific resistance, which is also known as vertical or hypersensitive resistance; and race-non specific, or horizontal, non-hypersensitive, partial or slow rusting resistance.

Table 3. Chromosomal location, origin, authors of cytogenetic and molecular mapping, and marker type used for the molecular characterization of identified leaf rust resistance genes

<i>Lr</i> gene	Location	Origin	Author of cytogenetic mapping	Author of molecular mapping	Marker type
<i>Lr1</i>	5DL	<i>T. aestivum</i>	McIntosh <i>et al.</i> (1965); McIntosh & Baker (1970)	Feuillet <i>et al.</i> (1995)	RFLP, RAPD, STS
<i>Lr2a, 2b, 2c</i>	2DS	<i>T. aestivum</i>	Luig & McIntosh (1968)		
<i>Lr3a, 3ka, 3bg</i>	6BL	<i>T. aestivum</i>	Heyne & Livers (1953)	Sacco <i>et al.</i> (1998)	RFLP
<i>Lr9</i>	6B	<i>Ae. umbellulata</i>	Sears (1961)	Schachermayr <i>et al.</i> (1994)	RFLP, RAPD
<i>Lr10</i>	1AS	<i>T. aestivum</i>	Dyck & Kerber (1971)	Nelson <i>et al.</i> (1997)	RFLP
<i>Lr11</i>	2A	<i>T. aestivum</i>	Solisman <i>et al.</i> (1964)		
<i>Lr12</i>	4B	<i>T. aestivum</i>	McIntosh & Baker (1966)		
<i>Lr13</i>	2BS	<i>T. aestivum</i>	Singh & Gupta (1991)	Seyfarth <i>et al.</i> (2000)	RFLP, SSR
<i>Lr14a, 14b</i>	7B	<i>T. aestivum</i> <i>Lr14a T. turgidum, Lr14b T. aestivum</i>	McIntosh <i>et al.</i> (1967); Law & Johnson (1967)		
<i>Lr15</i>	2DS	<i>T. aestivum</i>	Luig & McIntosh (1968)		
<i>Lr16</i>	2BS	<i>T. aestivum</i>	McIntosh <i>et al.</i> (1995a)	McCartney <i>et al.</i> (2005)	SSR
<i>Lr17a, 17b</i>	2AS	<i>T. aestivum</i>	Dyck & Kerber (1977); Singh <i>et al.</i> (2001)		
<i>Lr18</i>	5BL	<i>T. timopheevii</i>	McIntosh (1983)	Yamamori (1994)	N-band
<i>Lr19</i>	7DL	<i>Agropyron elongatum</i>	Sharma & Knott (1966)	Autrique <i>et al.</i> (1995)	RFLP, AFLP
<i>Lr20</i>	7AL	<i>T. aestivum</i>	Sears & Briggie (1969)	Neu <i>et al.</i> (2002)	RFLP
<i>Lr21</i>	1DS	<i>Ae. tauschii</i>	Rowland & Kerber (1974)	Spielmeier <i>et al.</i> (2000); Huang and Gil. (2001)	RFLP, RGA clones
<i>Lr22a, 22b</i>	2DS	<i>Lr22a Ae. tauschii, Lr22b T. aestivum</i>	Rowland & Kerber (1974); Dyck (1979)	Raupp <i>et al.</i> (2001)	SSR
<i>Lr23</i>	2BS	<i>T. turgidum</i> ssp. <i>durum</i>	McIntosh & Dyck (1975)	Nelson <i>et al.</i> (1997)	RFLP
<i>Lr24</i>	3DL	<i>Agropyron elongatum</i>	Sears (1973)	Autrique <i>et al.</i> (1995); Schachermayr <i>et al.</i> (1995)	RFLP and RAPD
<i>Lr25</i>	4BS	<i>Secale cereale</i>	Driscoll & Anderson (1967)		
<i>Lr26</i>	1BL	<i>Secale cereale</i>	Zeller (1973)	Hsam <i>et al.</i> (2000)	AFLP, RFLP

<i>Lr27 + Lr31</i>	3BS +	<i>T. aestivum</i>	Singh & McIntosh (1984)	Nelson <i>et al.</i> (1997)	RFLP
<i>Lr28</i>	4BS	<i>T. speltaoides</i>	McIntosh <i>et al.</i> (1982)	Naik <i>et al.</i> (1998)	RAPD, STS
<i>Lr29</i>	4AL	<i>Agropyron elongatum</i>	Sears (1977)	Procunier <i>et al.</i> (1995)	RAPD
<i>Lr30</i>	7DS	<i>T. aestivum</i>	Dyck & Kerber (1981)	Autrique <i>et al.</i> (1995)	RFLP
<i>Lr32</i>	4AL	<i>Ae. taushii</i>	Kerber (1987)		
<i>Lr33</i>	3D	<i>T. aestivum</i>	Dyck <i>et al.</i> (1987)		
<i>Lr34</i>	1BL	<i>T. aestivum</i>	Dyck (1987)	Nelson <i>et al.</i> (1997)	RFLP
<i>Lr35</i>	7DS	<i>T. aestivum</i>	Dyck & Dyck (1990)	Seyfarth <i>et al.</i> (1999)	RFLP, STS
<i>Lr36</i>	2B	<i>T. speltaoides</i>	Kerber & Dyck (1990)		
<i>Lr37</i>	6BS	<i>T. speltaoides</i>	Dvorak & Knott (1990)		
<i>Lr38</i>	2AS	<i>Ae. ventricosa</i>	Bariana & McIntosh (1993)	Robert <i>et al.</i> (1999)	RAPD, SCAR
<i>Lr39 (Lr41)</i>	6DL	<i>Agropyron intermedium</i>	Friebe <i>et al.</i> (1992)	Friebe <i>et al.</i> (1992)	Isozyme analysis
<i>Lr40 (allelic to Lr21)</i>	2DS	<i>Ae. taushii</i>	Raupp <i>et al.</i> (2001)	Raupp <i>et al.</i> (2001); Singh <i>et al.</i> (2004c)	SSR, RFLP
<i>Lr42</i>	1DS	<i>Ae. taushii</i>	Cox <i>et al.</i> (1994)	Huang & Gill (2001)	RFLP, STS
<i>Lr43</i>	1DS	<i>Ae. taushii</i>	Cox <i>et al.</i> (1994)		
<i>Lr44</i>	7DS	<i>Ae. taushii</i>	Hussein <i>et al.</i> (1997)		
<i>Lr45</i>	1BL	<i>T. aestivum ssp. spelta</i>	Dyck & Sykes (1994)		
<i>Lr46</i>	2A	<i>Secale cereale</i>	McIntosh <i>et al.</i> (1995b)		
<i>Lr47</i>	1B	<i>T. aestivum</i>	Singh <i>et al.</i> (1998)	William <i>et al.</i> (2003)	AFLP
<i>Lr48</i>	7A	<i>T. speltaoides</i>	Dubkovsky <i>et al.</i> (1998)	Dubkovsky <i>et al.</i> (1998)	RFLP
<i>Lr49</i>		<i>T. aestivum</i>	Saini <i>et al.</i> (2002)		
<i>Lr50</i>	2BL	<i>T. timopheevii ssp. armeniacum</i>	Saini <i>et al.</i> (2002)		
<i>Lr51</i>	1BL	<i>T. speltaoides</i>	Brown-Guedira <i>et al.</i> (2003)	Brown-Guedira <i>et al.</i> (2003)	SSR
			Dvorak & Knott (1980)	Helguera <i>et al.</i> (2005)	RFLP, CAPS

Race-specific, or vertical, resistance is a type of resistance that is effective against some races but ineffective against others (Vanderplank, 1963; 1968). This type of resistance is based on a post-haustorial mechanism, which involves a hypersensitive reaction with rapid cell-death of invaded and neighboring cells resulting in a necrotic lesion (Parlevliet, 1975; 1994). Parlevliet (1994) reviewed the different types of resistance against biotrophic and hemi-biotrophic fungi and concluded that hypersensitive resistance is often governed by genes that follow the gene-for gene relationship, has large effects and is controlled by major genes which are race-specific and often dominant. These genes usually occur in large numbers, and more than 50 race-specific genes have been documented in wheat (Table 3).

The vulnerability of wheat to leaf rust epidemics has increased with the tendency to grow large areas of genetically homogeneous cultivars with resistance based on single race-specific resistance genes (Samborski, 1985). New virulent races have evolved and breeding for rust resistance has been characterized by the so called boom-and-bust syndrome where new cultivars with new effective resistance genes incorporated had to be released continuously (Kilpatrick, 1975). Race-specific resistance is therefore usually not considered durable, especially if the resistance is based on deployment of single genes. One way to prolong the effectiveness of these genes is to 'pyramid' or combine several effective race-specific genes into a single cultivar. The pathogen is then forced to undergo a sequence of mutations corresponding to each resistance gene reducing the probability of break-down of resistance (McDonald & Linde, 2002).

Partial, or horizontal, resistance was described by Vanderplank (1963; 1968) as race-non specific with a non-differential interaction between the host and the pathogen and evenly effective against all races of the pathogen. Hence, it does not follow the gene-for-gene relationship. Parlevliet (1994) showed that there were small differential interactions for partial resistance to barley leaf rust isolates and did therefore choose not to call it race-nonspecific. Caldwell (1968) described this type of resistance to the wheat rust fungi as general or slow rusting resistance because it was manifested as slow development of disease on a cultivar compared to a specific check cultivar despite a compatible host-pathogen interaction. This type of resistance was shown to be based on a pre-haustorial mechanism, which did not involve rapid cell death and necrotic lesions (Rubiales & Niks, 1995; Martinez *et al.*, 2001). The same chromosome regions were shown to be effective against other diseases (yellow rust, powdery mildew, barley yellow dwarf virus) (Singh, 1993; Parlevliet, 1994). Slow rusting resistance to leaf rust in barley and wheat was found to be the result of the collective effect of a longer latent period, smaller uredinium size, reduced infection frequency (receptivity), and reduced spore production (Vanderplank, 1963; Parlevliet, 1975; Ohm & Shaner, 1976; Parlevliet, 1985; Wilcoxson, 1981). This type of resistance is considered to be durable.

Genetic studies have indicated that slow rusting to leaf rust is determined by few to several genes with moderately high heritability (Parlevliet, 1978; Bjarko & Line, 1988a; Singh & Rajaram, 1992; Navabi *et al.*, 2003; Das *et al.*, 2004).



Additive gene effects are predominant for this type of resistance but other types of interactions are also reported (Bjarko & Line, 1988b; Das *et al.*, 1992). Of the over 50 known wheat leaf rust resistance genes (Table 3) only *Lr34* and *Lr46* have been classified as genes conferring slow rusting or partial resistance. The slow rusting genes *Lr34* and *Lr46* have often been used in combinations with other slow rusting genes (Singh *et al.*, 2005). Singh *et al.* (2000) showed that bread wheat lines that were nearly immune to leaf and stripe rust could be developed by accumulating 4-5 slow rusting genes through intercrossing lines that carried intermediate disease levels followed by selection under high rust pressure. Several near-immune slow rusting bread wheat lines for release in developing countries have since been produced by using these lines as a source of resistance for breeding (Singh *et al.*, 2004b). Genetic studies have shown that at least 10 to 12 different slow rusting genes are involved in conferring slow rusting resistance in CIMMYT bread wheats (Singh *et al.*, 2005).

#### *Different P. triticina populations occur on bread and durum wheat*

Several studies have shown that the *P. triticina* populations predominant on durum wheat are different from those predominant on bread wheat. The genes effective against predominant races of durum and bread wheat rust pathogens are therefore also expected to be different. Huerta-Espino & Roelfs (1992) conducted a global survey of *P. triticina* collections from both durum and bread wheat and found that the races attacking these two species were different when tested with the North American differential-set (Long & Kolmer, 1989). Leaf rust cultures isolated from durum wheat were seldom virulent to the most susceptible bread wheat cultivars, while the same cultures were often virulent to durum wheat cultivars tested (Huerta-Espino & Roelfs, 1992). Similarly, the cultures from bread wheat that were highly virulent on bread wheat were often avirulent on durum wheat. Huerta-Espino & Roelfs (1992) found that a majority of the leaf rust collections from durum wheat belonged to the physiological race BBB following the nomenclature system of Long & Kolmer (1989). The virulence frequency to *Lr1*, *Lr2a*, *Lr2c*, *Lr3* and *Lr26* was much lower in the leaf rust populations isolated from durum wheat. While 54% of the cultures from durum wheat were avirulent to *Lr1*, *Lr2a*, *Lr2c* and *Lr3*, only 6% were avirulent when collected from bread wheat. Leaf rust isolates from durum wheat were often virulent to *Lr10* and *Lr23* (Roelfs & Huerta-Espino, 1992). All of the over 1,000 leaf rust isolates from bread wheat were virulent to the universally susceptible bread wheat Thatcher (carrying adult plant resistance gene *Lr22b*) compared to only 48% of the 201 isolates from durum wheat (Roelfs & Huerta-Espino, 1992).

Differences in races that occur on bread and durum wheat were also reported in studies from Ethiopia (Dmitrev & Gorshkov, 1980; Kuzmichev, 1984) and from Mexico (Singh, 1991). Dmitrev & Gorshkov (1980) and Kuzmichev (1984) reported that leaf rust collected from durum wheat in Ethiopia affected durum wheat more severely than bread wheat, and leaf rust isolates from bread wheat affected bread wheat more severely. Some durum wheat leaf rust races were also reported avirulent to all leaf rust resistance (*Lr*) genes tested. Singh (1991) conducted a leaf rust survey in Mexico during 1988 and 1989 and identified 23

different *P. triticina* races. An interesting feature was that the predominating races isolated from durum wheat were different from those from bread wheat, even when the fields were adjacent. The *P. triticina* race BBB/BN predominated in durum wheat, while the most frequently occurring races in bread wheat fields were TCB/TD and TBD/TM. Singh (1991) used the nomenclature system according to Long & Kolmer (1989) for race determination with two supplementary Mexican sets for a complete description of variation. Race BBB/BN was virulent to few of the known wheat leaf rust resistance genes and would have been classified as physiologic race 1 according to the historical race nomenclature key of Johnston & Levine (1955).

Ordóñez *et al.* (2004) analyzed *P. triticina* from durum wheat collected from several countries and distinguished at least two different groups of *P. triticina* adapted to durum wheat. They were distinct from the isolates found on bread wheat in their virulence pattern based on 36 near-isogenic lines of Thatcher. Leaf rust isolates from durum wheat originating from Argentina, France, Mexico, Spain and California showed high infection type responses (susceptible response) on 24 durum wheat cultivars tested and on differentials carrying *Lr10, 14b, 20, 23, 33, 41* and *Lr44*. Few isolates collected from durum wheat fields in Chile and northern USA had virulence specificities similar to leaf rust races from bread wheat and had low infection types (resistant response) on the durum wheat cultivars. Several isolates from Ethiopia had low infection types on the universally susceptible bread wheat cultivar Thatcher, and all Ethiopian isolates had high infection type responses on durum wheat cultivars tested in the study.

Huerta-Espino & Roelfs (1992) pointed out that even if leaf rust races from durum wheat may appear similar based on bread wheat differentials they can be very different when tested on different durum wheat lines.

#### *Resistance to leaf rust in durum wheat*

Information is limited on the nature and the genetic basis of leaf rust resistance in durum wheat. Of the known designated leaf rust resistance genes only *Lr14a* and *Lr23* are reported to have originated from durum or emmer wheat. Other genes that have been suggested to be present in durum and emmer wheat are *Lr3* (Singh *et al.*, 1992), *Lr10* (Aguilar-Rincon, 2001), *Lr13* (Singh *et al.*, 1992), *Lr16*, *Lr17a* (Zhang & Knott, 1990) and *Lr33* (Dyck, 1994). These genes have been postulated to be present in durum wheat by comparing the reaction pattern to different *P. triticina* races with bread wheat differentials carrying known *Lr* genes. If the pattern of the seedling reaction to a diverse collection of races is different from the pattern exhibited by the known resistance genes then it can indicate that such resistance is conferred by an unidentified gene(s). Dissimilar patterns of reaction have often been observed in durum wheat when tested with a range of races (Zhang & Knott, 1990; Singh *et al.*, 1992; Bai & Knott, 1994). Different behavior of durum wheat and bread wheat to different races have been reported in studies from Italy (Paradies, 1980; 1981) and India (Pandey & Rao, 1984; Sharma *et al.*, 1986) and have also led to the conclusion that the resistance in durum wheat is different.

While extensive inheritance studies have been conducted to investigate the genetic basis of resistance in bread wheat, only a few have been reported from durum wheat. The genetic studies for leaf rust resistance in durum wheat are summarized in Table 4. Most genetic studies reported (Table 4) on durum wheat were on seedling resistance under greenhouse conditions. The resistance in several durum wheat lines was conferred by recessively inherited genes. In bread wheat, recessive resistance is not so common. Only a few of the known designated genes identified in bread wheat, such as *Lr14b* (Dyck & Samborski, 1970), *Lr30* (Dyck & Kerber, 1981) and *Lr3*, in certain backgrounds (Sacco *et al.*, 1998) were reported to be recessively inherited. The recessive and dominant nature of resistance may change depending on the test conditions and genetic background as shown in bread wheat (Dyck & Kerber, 1985; Kolmer, 1996).

Pathogen collections from bread wheat have often been used for conducting genetic studies on durum wheat. The relevance of the information generated with the use of improper races can be considered low if the objective is to improve resistance in durum wheat. Mishra (1996) therefore studied the inheritance of resistance in durum wheat by using leaf rust isolates collected from durum wheat fields from several countries (Chile, Ethiopia, India, Israel, Italy, Morocco, Mexico, Pakistan, Romania and Turkey). Inheritance studies were conducted on F<sub>1</sub> and F<sub>2</sub> seedlings in controlled greenhouse conditions. In all, 27 dominant and 21 recessive resistance genes were identified among 15 cultivars. Based on differences in specificity, resistance phenotypes, and inheritance, 21 of the dominant genes and all of the 21 recessive genes were thought to be unique and different from previously identified genes from bread wheat. However, their allelic relationships were not investigated and the results were not verified using F<sub>3</sub> families. In general the inheritance was simple and often conditioned by one or two genes. Resistance was either incompletely dominant or recessive. A characterizing feature for the leaf rust -durum wheat interaction was the high frequency of mesothetic (random distribution of variable-sized uredinia on a single leaf) infection type responses. In particular Y and Z infection type responses could be observed, with greater compatibility towards the leaf tip or towards the leaf base, respectively. Of the known leaf rust resistance genes only *Lr11* and *Lr37* confer such reaction (Y) and none of these genes were thought to be present in durum wheat material investigated by Mishra (1996).

Singh *et al.* (1993) studied the genetic basis of resistance in nine CIMMYT-derived durum wheat lines, including the most commonly grown Mexican cultivar Altar C84 at that time. The Mexican *P. triticina* race BBB/BN prevalent during that period on durum wheat was used in their study. The inheritance studies were based on rust evaluations conducted on seedlings and/or adult plants of F<sub>1</sub>, F<sub>2</sub> populations and F<sub>3</sub> lines. A partially dominant gene conferred seedling resistance in Altar C84 as well as in three other durum wheat lines which seemed to interact in an additive manner with two other partially effective slow rusting genes.

Table 4. Summary of genetic studies of leaf rust resistance in durum wheat

Durum wheat	Race	Population	Type of resistance	Condition	Genes	Authors
Gaza (E.931)	Race mix	F <sub>1</sub> , F <sub>3</sub>	Adult (low IT)	Field	2 rec. ( <i>LrGaz</i> )	Murty & Lakhani (1958); Mathur <i>et al.</i> (1964)
St. 464	Race mix	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Adult (low IT)	Field	2 rec. ( <i>LrGaz</i> )	Mathur <i>et al.</i> (1964)
C.L. 7809	135D,68C,64A	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Seedling	Greenhouse	1 dom ( <i>LrT<sub>1</sub></i> ) + 2 rec. ( <i>LrT<sub>2</sub></i> + <i>LrT<sub>3</sub></i> )	Ataullah (1969)
P.L. 109593	135D,64A	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Seedling	Greenhouse	2 rec. ( <i>LrT<sub>1</sub></i> + <i>LrT<sub>3</sub></i> )	Ataullah (1969)
Gerardo 512 (Valgerardo)	12,233	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Seedling	Greenhouse	2. dom ( <i>LrG1</i> + <i>LrG2</i> to race 12 and <i>LrG1</i> to race 223)	Zitelli (1972)
Leeds	UN 1, UN 2	F <sub>1</sub> , F <sub>2</sub> , and F <sub>3</sub>	Seedling	Greenhouse	2 rec.	Statler (1973); Abdallah (1971)
Ramsay	UN 1, UN 2	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub> , BCF <sub>1</sub> , BCF <sub>2</sub>	Seedling	Greenhouse	2 rec.	Rashid <i>et al.</i> (1976); Abdallah (1971)
D561	UN 1, (UN 2)	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub> , BCF <sub>1</sub> , BCF <sub>2</sub>	Seedling	Greenhouse	1 rec. (2 rec.)	Rashid <i>et al.</i> (1976); Abdallah, 1971)
D6733	UN 1	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub> , BCF <sub>1</sub> , BCF <sub>2</sub>	Seedling	Greenhouse	1 rec.	Rashid <i>et al.</i> (1976)
Cocont-71	77	F <sub>1</sub> , F <sub>2</sub> , BCF <sub>2</sub>	Seedling	Greenhouse	1 partially dom. ( <i>LrCoc</i> )	Ortiz <i>et al.</i> (1976)
Jori C-69	77	F <sub>1</sub> , F <sub>2</sub> , BCF <sub>3</sub>	Seedling	Greenhouse	1 rec. ( <i>LrJori</i> )	Ortiz <i>et al.</i> (1976)
Crane s.	77	F <sub>1</sub> , F <sub>2</sub> , BCF <sub>4</sub>	Seedling	Greenhouse	2 rec. ( <i>LrCra</i> + )	Ortiz <i>et al.</i> (1976)
Stork s. D. Dwarf S. 15-Cr s.	77	F <sub>1</sub> , F <sub>2</sub> , BCF <sub>5</sub>	Seedling	Greenhouse	1 rec. ( <i>LrCra</i> )	Ortiz <i>et al.</i> (1976)
Bijaga Red	77, 162A	F <sub>1</sub> , F <sub>2</sub> , BCF <sub>6</sub>	Seedling	Greenhouse	1 rec. ( <i>LrCoc</i> )	Ortiz <i>et al.</i> (1976)
ED 155	12A,77,104B	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Seedling/adult (low IT)	Greenhouse	2 rec. + 4 dom.	Kadam <i>et al.</i> (1983)
ED 1096	12A,77,104B	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Seedling/adult (low IT)	Greenhouse/Field	2 dom. ( <i>LrED1551</i> + <i>LrED1552</i> )	Mishra <i>et al.</i> (1989)
ED 404	12A,77,104B	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Seedling/adult (low IT)	Greenhouse/Field	2 dom. ( <i>LrED10961</i> + <i>LrED10962</i> )	Mishra <i>et al.</i> (1989)
Stewart 63	15, (UN 2)	F <sub>1</sub> , F <sub>2</sub> , BCF <sub>2</sub>	Seedling/adult (low IT)	Greenhouse/Field	2 rec. ( <i>LrED4041</i> + <i>LrED4042</i> )	Mishra <i>et al.</i> (1989)
Lloyd	15	F <sub>1</sub> , F <sub>2</sub> , BCF <sub>2</sub>	Seedling/adult (low IT)	Greenhouse/Field	1 dom. ( <i>LrE?</i> ) + 1 rec. ( <i>LrS?</i> ) to race 15, (2 rec. to UN 2)	Zhang & Knott (1990, 1993); Abdallah (1971)
Wakoona	15	F <sub>1</sub> , F <sub>2</sub> , BCF <sub>2</sub>	Seedling/adult (low IT)	Greenhouse/Field	1 rec. ( <i>LrLb</i> ) + 1 dom. adult	Zhang & Knott (1990, 1993)
Quilfen	15	F <sub>1</sub> , F <sub>2</sub> , BCF <sub>2</sub>	Seedling/adult (low IT)	Greenhouse/Field	1 rec. ( <i>LrHk</i> )	Zhang & Knott (1990, 1993)
Medora	15	F <sub>1</sub> , F <sub>2</sub> , BCF <sub>2</sub>	Seedling/adult (low IT)	Greenhouse/Field	<i>Lr17?</i>	Zhang & Knott (1990, 1993)
			Seedling/adult (low IT)	Greenhouse/Field	1 rec. ( <i>LrMdl</i> ) + 1 dom. ( <i>LrMdl2</i> )	Zhang & Knott (1990, 1993)

Pelissier	15	F <sub>1</sub> , F <sub>2</sub> , BCF <sub>2</sub>	Seedling/adult (low IT)	Greenhouse/Field	1 rec. ( <i>LrP</i> ) + 1 rec. adult	Zhang & Knott (1990; 1993)
Golden Ball	15	F <sub>1</sub> , F <sub>2</sub> , BCF <sub>2</sub>	Seedling	Greenhouse	1 rec. ( <i>LrGB</i> )	Zhang & Knott (1990)
Vic	15	F <sub>1</sub> , F <sub>2</sub> , BCF <sub>2</sub>	Seedling	Greenhouse	1 dom. or 1 dom.+ 1 rec.	Zhang & Knott (1990)
Malvika	1, 77A, 108	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Seedling	Greenhouse	1 dom. to race 108; 2 dom. ( <i>Lr23?</i> +) to race 1 and race 77A	Gupta <i>et al.</i> (1992)
CPAN 6051	1, 77A, 108	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Seedling	Greenhouse	1 dom. to race 1 and 108; 2 dom. ( <i>Lr23?</i> +) to race 77A	Gupta <i>et al.</i> (1992)
Mexicali 75	BBB/BN	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Adult or slow rusting	Greenhouse/Field	2 additive adult ( <i>LrHum</i> + 1)	Singh <i>et al.</i> (1993)
Yavaros 79	BBB/BN	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Adult or slow rusting	Greenhouse/Field	2 additive adult ( <i>LrHum</i> + 1)	Singh <i>et al.</i> (1993)
Altar C84	BBB/BN	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Seedling/adult	Greenhouse/Field	1 partially dom. ( <i>LrAlt</i> ) + 2 additive adult ( <i>LrHum</i> + 1)	Singh <i>et al.</i> (1993)
Carcomun	BBB/BN	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Seedling/adult	Greenhouse/Field	1 partially dom. ( <i>LrAlt</i> ) + 2 additive adult ( <i>LrHum</i> + 1)	Singh <i>et al.</i> (1993)
Diver	BBB/BN	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Adult or slow rusting	Greenhouse/Field	2 additive adult ( <i>LrHum</i> + 1)	Singh <i>et al.</i> (1993)
Kingfisher	BBB/BN	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Adult or slow rusting	Greenhouse/Field	2 additive adult ( <i>LrHum</i> + 1)	Singh <i>et al.</i> (1993)
Morus	BBB/BN	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Seedling/adult	Greenhouse/Field	1 partially dom. ( <i>LrAlt</i> ) + 2 additive adult ( <i>LrHum</i> + 1)	Singh <i>et al.</i> (1993)
Somorguho	BBB/BN	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Adult or slow rusting	Greenhouse/Field	2 additive adult ( <i>LrHum</i> + 1)	Singh <i>et al.</i> (1993)
Totanus	BBB/BN	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Seedling/adult	Greenhouse/Field	1 partially dom. ( <i>LrAlt</i> ) + 2 additive adult ( <i>LrHum</i> + 1)	Singh <i>et al.</i> (1993)
lumillo	BBB/BN	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Adult or slow rusting	Greenhouse/Field	2 additive adult ( <i>LrHum</i> + 1)	Singh <i>et al.</i> (1993)
CI7181	15	F <sub>1</sub> , F <sub>2</sub> , BCF <sub>2</sub>	Seedling	Greenhouse	1 dom. ( <i>LrC171</i> )	Bai & Knott (1994)
( <i>dicoccoides</i> )						
PI133134	15	F <sub>1</sub> , F <sub>2</sub> , BCF <sub>2</sub>	Seedling	Greenhouse	1 dom.	Bai & Knott (1994)
( <i>dicoccoides</i> )						
PI164582	15	F <sub>1</sub> , F <sub>2</sub> , BCF <sub>2</sub>	Seedling	Greenhouse	1 dom.	Bai & Knott (1994)
( <i>dicoccoides</i> )						
PI197483	15	F <sub>1</sub> , F <sub>2</sub> , BCF <sub>2</sub>	Seedling	Greenhouse	1 dom. ( <i>LrC171</i> )	Bai & Knott (1994)
( <i>dicoccoides</i> )						
PI254159	15	F <sub>1</sub> , F <sub>2</sub> , BCF <sub>2</sub>	Seedling	Greenhouse	1 partially dom.	Bai & Knott (1994)
( <i>dicoccoides</i> )						
PBW 34	1, 77A, 108	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Seedling	Greenhouse	1 dom. to race 1 and 108; 2 dom. ( <i>Lr23?</i> +) to race 77A	Gupta <i>et al.</i> (1995)
DWL 5023	1, 77A, 108	F <sub>1</sub> , F <sub>2</sub> , F <sub>3</sub>	Seedling	Greenhouse	1 dom. to race 108; 2 dom. ( <i>Lr23?</i> +) to race 1 and race 77A	Gupta <i>et al.</i> (1995)
15 durum wheat	Durum leaf					
lines	rust races	F <sub>1</sub> , F <sub>2</sub>	Seedling	Greenhouse	1-2 genes per isolate	Mishra (1996)

Information on slow rusting resistance in durum wheat is limited. In the same study by Singh *et al.* (1993) the genetic basis of five CIMMYT-derived durum wheats lines; Mexicali 75, Yavaros 79, Diver, Kingfisher and Somorguho, showing compatible (susceptible) responses to *P. triticina* race BBB/BN were investigated. Based on these studies resistance in these lines was conferred by two additive genes, of which at least one was common in all durum wheat parents.

The importance of leaf rust in durum wheat has increased after the recent breakdown of resistance in various important cultivars in several countries. Singh *et al.* (2004a) reported severe leaf rust epidemics during 2001, 2002 and 2003 in durum wheat fields in north-western Mexico due to a new *P. triticina* race, later identified as BBG/BN. Most of the cultivars grown in Mexico, including Altar C84, as well as the majority of durum wheat cultivars from 31 different countries were susceptible to this new race. In addition, almost 90% of the entire CIMMYT durum wheat collection was also susceptible. The resistance in Altar C84 had remained effective for 16 years before becoming ineffective to race BBG/BN. Adjacent bread wheat fields were not affected by this new race. Singh *et al.* (2004a) also reported that CIMMYT derived durum wheat cultivars in recently became susceptible in Chile, southern France, Spain and Syria, and that durum wheat lines that were resistant to the Mexican race BBG/BN also were resistant in these countries. Recent studies have confirmed that the durum wheat leaf rust races in Spain and France have similar virulence specificity to the predominant Mexican race when tested on bread wheat differentials (Martinez *et al.*, 2005; Goyeau *et al.*, 2006). Singh *et al.* (2004a) identified several durum wheat lines that conferred either race-specific or slow rusting resistance to the new *P. triticina* race in Mexico after screening thousands of lines. A new cultivar, Jupare C2001, resistant to the new *P. triticina* race, has today replaced Altar C84 in north-western Mexico. The seedling resistance gene to which race BBG/BN is virulent and that was present in Altar C84 as well as in most CIMMYT durum wheat germplasm remains undesignated.

#### *Mapping leaf rust resistance genes in bread and durum wheat*

Chromosome location is a basic step for identifying and understanding the allelic relationships of resistance genes (McIntosh *et al.*, 1995a). Most of the known designated resistance genes to leaf rust were identified through cytogenetic analysis using aneuploids (plants that do not have the normal chromosomal number) (McIntosh *et al.*, 1995a).

Monosomic analysis (a monosomic lacks one chromosome of the chromosome pairs) is the most commonly used cytogenetic method in bread wheat (Knott, 1989) and is the most suitable method for locating dominant genes (McIntosh *et al.*, 1995a). Until recently, the chromosome locations of most of the mapped leaf rust resistance genes were determined using monosomic analysis and telocentric (chromosomes with the centromere in the terminal end) mapping (Table 3). Sears (1954) developed the complete set of 21 monosomics for each chromosomes using the bread wheat cultivar Chinese spring. The monosomic analysis involves crossing 21 monosomics as the female parent with the resistant cultivar carrying

the unknown resistance gene. The F<sub>1</sub> plants with 41 chromosomes are allowed to self pollinate and the F<sub>2</sub> plants are evaluated for rust resistance. The monosomic that coincides with the chromosome location of the gene gives a distorted segregation in the F<sub>2</sub> and subsequent segregating generations (Knott, 1989). Sears (1953; 1954) also produced chromosome substitution lines and other types of aneuploids (nullisomics, trisomics, tetrasomics) which were also used in mapping. Monosomic analysis is often combined with telocentric mapping where ditelosomics (a plant with one chromosome represented by a homologous pair of telocentric chromosomes) developed by Sears (1966) are used for determining the chromosome arm location and recombination distance of the gene from the centromere. These cytogenetic stocks were also used in recent years to determine the location of molecular markers.

In durum wheat, the use of aneuploid analysis has not been as successful as in bread wheat (Joppa, 1987; Knott, 1989). Aneuploidy is not as well tolerated in the tetraploids compared to hexaploids (Joppa, 1987). Reductions in chromosome numbers are more detrimental in tetraploids than increases in chromosome number. The monosomics in tetraploid wheats often show poor fertility and instability and were therefore not used in durum wheat for mapping of traits of interest (Joppa, 1987). Joppa & Williams (1983) produced a complete set of 14 different disomic-substitution lines with 13 pairs of durum wheat (cv. Langdon) chromosomes and a pair of D-genome chromosomes substituting the homeologous chromosome pair from the A or B genome. A complete set of double-ditelosomics for the A- and B-genome chromosomes were also developed in Langdon durum wheat that can be used for determining gene to centromere distances (Joppa, 1987).

Recent studies have shown that the aneuploids developed by Joppa & Williams (1983) and Joppa (1987) can effectively be used to find the chromosome location of traits in durum wheat (Hussein *et al.*, 2005; Singh *et al.*, 2006). Hussein *et al.* (2005) used the Langdon durum wheat D-genome disomic-substitution lines to determine the location of two adult plant leaf rust resistance genes in durum and emmer wheat. The two novel genes were temporarily designated *Lrac104* and *Lrac124* and were located on chromosome 6B and 4A, respectively. Bhagwat *et al.* (2004) used trisomics (one extra chromosome) to determine the chromosomal location of a recessive gene on chromosome 2B in durum wheat HD 4502.

The chromosome location of a few leaf rust resistance genes originating from durum or emmer wheat have been determined (Table 3). One of the alleles of *Lr14*, designated as *Lr14a*, is thought to have originated from the emmer wheat cultivar Yaroslav and then been transferred to the *T. aestivum* cultivars Hope and H-44 which were used in mapping (McIntosh *et al.*, 1967; McIntosh *et al.*, 1995a). *Lr23* was transferred to hexaploid wheat from *T. turgidum* ssp. *durum* cv. Gaza (Watson & Stewart, 1956) and localized to chromosome 2BS by McIntosh & Dyck (1975). Marais *et al.* (2005) determined the location of a gene that was introduced to bread wheat from *T. dicoccoides* to chromosome 6BS. Dyck (1994) transferred two leaf rust resistance genes from *T. turgidum* ssp. *dicoccoides* to hexaploid wheat, one of which was identical to *Lr33*. Resistance originating from

durum wheat sometimes is not expressed when transferred to bread wheat due to the effect of suppressor genes (Bai & Knott, 1992; Roelfs & Huerta Espino, 1992; Nelson *et al.*, 1997).

Molecular markers have been used in the last decades for chromosome mapping of genes that determine simply inherited traits or for finding quantitative trait loci (QTL) often associated with more complex traits. Another major application of molecular techniques is finding molecular markers closely linked, or co-segregating, with the trait of interest for the use in marker assisted selection (MAS) (Gupta *et al.*, 1999). Mapping of leaf rust resistance in bread wheat has also proven possible using molecular techniques (Friebe *et al.*, 1992; Nelson *et al.*, 1997; Brown-Guedira *et al.*, 2003; William *et al.*, 2003). The most commonly used molecular technique for the initial mapping of leaf rust resistance genes was RFLP (Table 4). However, markers that are tightly linked to the gene of interest and PCR-based markers (STS, SSRs, ISSRs) that are easy to manage have later been identified and developed for several of these genes (Huang & Gill, 2001). William *et al.* (2003) used bulked segregant analysis (BSA) with amplified fragment length polymorphisms (AFLP) (Vos *et al.*, 1995) and partial linkage mapping to map the slow rusting leaf rust resistance gene *Lr46*. The BSA approach was developed by Michelmore *et al.* (1991) for identifying markers in a specific region of the genome. The procedure involves preparing two bulked DNA samples from individuals selected from a segregating population of a cross that are contrasting in respect to the trait of interest. Polymorphic markers that distinguish the two samples are searched for and then used in the full population to determine the linkage status of the molecular marker with the trait.

Mapping of leaf rust resistance genes in durum wheat is almost non-existent. Nelson *et al.* (1997) mapped *Lr23* present in the durum wheat cultivar Altar C84 and its suppressor gene *SuLr23* in 2D using a cross between a synthetic hexaploid developed from Altar C84 and a *T. taushii* and the bread wheat cultivar Oyata. Zhang *et al.* (2005) recently transferred *Lr19* into durum wheat to improve leaf rust resistance and yellow pigment and they used RFLP markers to characterize the alien chromosome segment that carried the gene.

## Objectives

The objective of the work presented here was to reduce genetic vulnerability of durum wheat (*Triticum turgidum* ssp. *durum*) in farmers' fields against leaf rust (*Puccinia triticina*) epidemics in developing countries. This was to be achieved by enhancing the genetic diversity and durability of leaf rust resistance in CIMMYT durum wheat germplasm. The following research activities were undertaken:

- 1) Characterization of CIMMYT durum wheat for the type of resistance; viz. race-specific or slow rusting resistance (Paper I, II, III, IV).



- 2) Assessment of the yield protection obtained from race-specific and slow rusting genes through yield loss trials (Paper I).
- 3) Investigation of the slow rusting components; latent period, uredinium size and receptivity, in durum wheat and assessment of the relationship between these components and field disease parameters (Paper II).
- 4) Determination of the genetic basis and genetic diversity of race-specific resistance through genetic analysis (Paper III).
- 5) Identification of the chromosome location of race-specific resistance genes (Paper IV).

## Materials and methods

Singh *et al.* (2004a) evaluated approximately 30,000 tetraploid wheat lines for resistance to the *Puccinia triticina* race BBG/BN that was detected in north-western Mexico during the crop season 2000-2001. Durum wheat lines carrying race-specific resistance but also lines showing slow disease progress in the field despite a compatible host reaction were identified. From these initial screenings thirty advanced CIMMYT durum wheat lines and cultivars (listed in papers I to IV) were characterized further for the type of resistance in the greenhouse and in the field. Ten of these possessed race-specific resistance, eighteen were slow rusting and two were susceptible.

All thirty durum wheat lines were included in yield loss protection trials (Paper I). Seven of those showing slow rusting resistance and two susceptible durum wheat lines were used for the study on slow rusting resistance components (Paper II). Nine of those that showed race-specific resistance and one susceptible durum wheat were included for the inheritance studies (Paper III). Chromosome mapping involved two race-specific resistant and one susceptible line (Paper IV).

Two different sites in Mexico were used for field experiments, El Batán and Ciudad Obregón. The El Batán research station (and CIMMYT headquarter) is located northeast of Mexico City at 2230 meters above sea level (masl), with a wheat crop season from mid-May to mid-October. Ciudad Obregón is situated in the State of Sonora, in north-western Mexico at 39 masl with a wheat crop season from mid-November to late April. The main durum wheat production areas in Mexico are located in the north-western part of the country.

The avirulence/virulence formula for the *P. triticina* race BBG/BN (Singh *et al.*, 2004a) used in all studies is:  
*Lr1,2a,2b,2c,3,3bg,3ka,9,12,13,14a,15,16,17,18,19,21,24,25,26,27+31,29,30,32,34,35,36,37/10,11,14b,20,23,33.*

Artificial leaf rust epidemics were initiated in all field trials (Papers I-IV) by inoculating 'spreaders' consisting of the susceptible durum wheat cultivar Atil

C2001. Inoculations were conducted by spraying with urediniospores suspended in light mineral oil (Soltrol 170). To obtain a uniform leaf rust pressure the spreaders were sown as borders around the entire experiment and as small hills at one end of each plot.

In the field, leaf rust severity and host reaction were evaluated at weekly intervals (Papers I, II). Leaf rust severity (% rusted tissue) was recorded according to the modified Cobb Scale (Peterson *et al.*, 1948). Host reaction was recorded using four categories (resistant, moderately resistant, moderately susceptible and susceptible) as described in Roelfs *et al.* (1992). The area under the disease progress curve (AUDPC) was determined by using an Excel based program following the formula described in Roelfs *et al.* (1992) (Papers I, II).

All greenhouse studies were conducted at El Batán, in Mexico, where a collection of Mexican *P. triticina* races is preserved. In the greenhouse, plants were inoculated with *P. triticina* race BBG/BN by spraying with urediniospores suspended in Soltrol oil using an atomizer. After inoculations plants were transferred to a dew-chamber overnight to assure germination and infection of the pathogen. Greenhouse evaluation of rust infection type responses followed the 0-4 scale described in Roelfs *et al.* (1992) (Papers I-IV).

A paired split plot design was used for establishing the two yield protection trials (differing in sowing dates) with fungicide (Tebuconazole)-protected and rust infected (non-protected) treatments (Paper I). Grain yield as well as agronomic and yield traits were estimated in each plot according to Sayre *et al.* (1998). Percentage losses were calculated by comparing the mean values from the protected plots with that from the unprotected. The genotypic correlations were calculated between all measured parameters. Different statistical models were used for the analysis of data (such as spatial analysis) (Papers I, II) using ASREML (Gilmour *et al.*, 2002). The genotypic correlations were computed using SAS (SAS Institute Inc, 1999).

The slow rusting components; latent period, receptivity, and uredinium size, were evaluated on flag leaves in three repeated greenhouse experiments (Paper II) according to Das *et al.* (1993), Lee & Shaner (1985) and Singh & Huerta-Espino (2003), respectively. A randomized complete block design was used for the greenhouse tests and the field trial at El Batán. Disease data from the yield protection trials were also used for this study. Phenotypic correlations between field parameters, the final disease severity and area under the disease progress curve (AUDPC), and slow rusting components were calculated and a multiple regression analysis conducted using Excel and SAS (SAS Institute Inc, 1999), respectively. Different models in SAS were used for analysis of data depending on the association of repeated measurements for each component.

For inheritance studies (Paper III), the nine race-specific resistant parents were crossed with the susceptible cultivar Atil C2001 as the female parent. Individual F<sub>1</sub> plants were harvested and allowed to self in order to obtain F<sub>2</sub> populations. Approximately 98 space sown F<sub>2</sub> plants grown under disease-free conditions were

harvested individually to obtain F<sub>3</sub> lines/families for each cross. For the allelism test, the resistant parents were also intercrossed and F<sub>2</sub> populations obtained by individually harvesting F<sub>1</sub> plants. Approximately 200 F<sub>2</sub> plants per cross were individually evaluated and grouped into two categories; resistant and susceptible. The 98 F<sub>3</sub> families were grown as plots and individually evaluated into three categories; homozygous resistant, segregating and susceptible. The F<sub>2</sub> phenotypic and F<sub>3</sub> genotypic frequencies were compared with expected frequencies using  $\chi^2$ -analyses.

Two F<sub>3</sub> populations (Atil C2000 × Camayo; Atil C2000 × Storlom) characterized in the inheritance studies, and the parents were used for bulked segregant analysis to map resistance genes (Paper IV). The molecular approach used was similar to that by William *et al.* (2003). The bulked segregant analysis approach according to Michelmore *et al.* (1991) was used to search for amplified fragment length polymorphisms (AFLP) (Vos *et al.*, 1995) that were linked to the leaf rust resistance genes. Publicly available linkage maps from bread wheat (Roeder *et al.*, 1998) were utilised for detecting the chromosomal location of AFLP markers with the help of Map maker (Lander *et al.*, 1987). One sequence tagged site (STS) marker, generated from a RFLP marker that co-segregates with *Lr3* (Sacco *et al.*, 1998) and various known microsatellite markers (SSRs) were also used for mapping. Molecular and phenotypic characterisation of 197 F<sub>3</sub> families generated from individually harvested F<sub>2</sub> plants from the Camayo × Storlom cross were undertaken to establish the allelic relationship between genes in both parents. Responses of the parents were also compared to the bread wheat near-isogenic lines carrying *Lr3a*, *Lr3ka*, and *Lr3bg*.

## Results and discussion

### Effect of leaf rust on grain yield and yield traits of durum wheats with race-specific and slow rusting resistance to leaf rust (Paper I)

The thirty durum wheat lines included in the study were evaluated for leaf rust responses, grain yield and related traits, under high leaf rust pressure with or without fungicide protection in two replicated trials, differing in sowing dates at Ciudad Obregón, Mexico. In the late sown trial, plants were exposed to leaf rust at an earlier development stage than in the trial with the normal planting date. Most durum wheat lines with race-specific resistance were immune in the field. Durum wheat lines with slow rusting resistance showed a range of severity responses, but the severities were significantly lower than those of the susceptible checks. Leaf rust caused grain yield losses of up to 71% in susceptible durum wheat lines in non-protected plots when compared to protected plots. Lines with race-specific resistance had negligible or low yield losses. Yield losses for the slow rusting resistant durum wheat material varied and were high for several genotypes. The grain yield losses were attributed to reductions in biomass, harvest index, kernels per square meter, kernel weight, number of kernels per spike, and number of

spikes per meter square. Test weight, which has implications on the end-use quality of durum wheat, was also significantly reduced for the susceptible and most of the slow rusting durum wheat lines.

This study demonstrated the effectiveness of race-specific resistance in protecting grain yield from leaf rust. Several slow rusting lines with reduced severity responses and/or with low yield losses were also identified. The results presented here indicate that yield losses in durum wheat for the same severity level are higher compared to previously reported losses for bread wheat.

### **Evaluation of slow rusting resistance components to leaf rust in CIMMYT durum wheats (Paper II)**

Achieving durability by selecting slow rusting or partial resistance should be an important breeding objective to reduce genetic vulnerability and to provide a long term control of leaf rust. The slow rusting components and their effects on leaf rust in durum wheat had not been studied previously.

Across the three experiments the mean uredinium size was significantly smaller and the latent period significantly longer for slow rusting material than the susceptible checks. The mean receptivity was not lower for all slow rusting resistant lines compared to the susceptible checks as would have been expected. Several slow rusting lines had higher mean receptivity than the two susceptible lines. The uredinium size was the only component with a non-significant genotype  $\times$  experiment interaction, indicating that the ranking of the lines were similar in each experiment. Repeated measurements of the uredinium size did not alter the ranking of the durum wheat genotypes.

Disease progress in the field, measured as mean AUDPC and final disease severity (FDS), in each trial and across trials, was significantly lower for the slow rusting durum wheat lines than for the susceptible checks. Rankings of genotypes were not consistent in different field environments due to a significant genotype  $\times$  field trial interaction.

Latent period and particularly uredinium size showed strong associations with disease parameters in the field. The phenotypic correlation coefficients ( $r$ ) between uredinium size and AUDPC and FDS ranged between 0.86 and 0.90 depending on field trial. The  $r$ -values for the latent period and the above field disease parameters ranged between -0.60 and -0.80. Receptivity was not associated with disease parameters in the field. The three slow rusting components explained 62 and 66% of the variability for AUDPC and FDS, respectively; only uredinium size had a significant regression coefficient.

Our results are in agreement with similar studies on bread wheat showing that slow leaf rusting resistant genotypes possess smaller uredinium sizes and longer latent periods than susceptible genotypes, but this association was not found for receptivity. No association was found between receptivity and the other

components or with field disease parameters. Receptivity has shown to be more prone to environmental variation and experimental errors in studies on bread wheat and barley (Parlevliet, 1986; Das *et al.*, 1993). Unavoidable fluctuations in the amount of inoculum deposited on the leaves could have affected the results on the mean receptivity.

The level of slow leaf rusting resistance in the field was best predicted by the size of uredinia in the greenhouse. Similar results were also obtained for bread wheat by Das *et al.* (1993) and Singh & Huerta-Espino (2003). In other studies, latent period has instead been found to be the most reliable component and best predictor for slow disease progress in the field in bread wheat and barley (Parlevliet, 1985; Broers, 1989a; 1989b; Singh *et al.*, 1991; Zadoks, 1971).

The slow rusting durum wheat lines evaluated in this study show variations for individual components as well as for resistance levels in the field indicating that there should be genetic diversity for genes determining such resistance.

### **New genes for leaf rust resistance in CIMMYT durum wheats (Paper III)**

Searching for different sources of resistance for the enhancement of genetic diversity and for pyramiding of effective race-specific genes are important steps towards reducing the risks of rapid break-down of resistance due to evolution and selection of new virulence alleles in the pathogen population.

Five different sources of resistance in durum wheat to *P. triticina* race BBG/BN were identified in this study. These genes were temporarily designated as *LrJup1* + *LrJup2*, *LrLlar*, *LrGuay*, *LrCam* and *LrStor*. The same pair of partially dominant complementary genes *LrJup1* + *LrJup2* conferred resistance in the currently popular Mexican durum wheat cultivar Jupare C2001 and the lines Hulita and Pohowera. Four distinct partially, or completely, dominant single genes conferred resistance in the remaining material. The resistance in Somateria and the Chilean cultivar Llaretta INIA was due to the same dominant gene *LrLlar* whereas resistance in the sister lines Guayacan 2 and the Chilean cultivar Guayacan INIA was conferred by the same partially dominant gene *LrGuay*. Another partially dominant gene *LrCam* present in Camayo was linked in repulsion to a distinct gene *LrStor* in Storlom. Based on CIMMYT international nursery data and recently published information on races (Ordonez *et al.*, 2004; Martinez *et al.*, 2005; Goyeau *et al.* 2006), all identified genes confer or are expected to impart effective protection to the predominant races of durum *P. triticina* in Mexico, US, Chile, Spain and France and can therefore be used in enhancing genetic diversity for leaf rust resistance. To prolong their durability these genes should be deployed in combinations.

A mesothetic infection type response in seedlings of durum wheat is a frequent feature and was in agreement with earlier published results (Mishra, 1996). Of the nine race-specific resistant durum wheat parents used for the crosses, five

displayed a mesothetic response in seedlings that varied from 'X-' to 'X+'. Another characteristic feature of race-specific resistance in durum wheat was that infection type responses observed in the greenhouse tended to become lower, or plants became more resistant, as developmental stages advanced, reaching immunity in the field. For example, Jupare C2001 displayed the infection type response 'X-' in seedlings, ';1-' in adult plant tests in the greenhouse and immunity (no visible symptoms) in the field. A similar tendency was observed in the F<sub>1</sub> plants of the susceptible × resistant crosses. In adult plant tests in the greenhouse the F<sub>1</sub> plants of some crosses were susceptible indicating that the resistance was of a recessive nature. However, in the field the F<sub>1</sub> plants from the same crosses showed intermediate levels of resistance which indicates partial dominance. The recessive and dominant nature of resistance may therefore change depending on the test conditions and genetic background as has been shown in bread wheat (Kolmer, 1996). Due to the higher responses in the greenhouse, classification of F<sub>2</sub> plants from some susceptible × resistant crosses was sometimes difficult, making the field evaluations more reliable. It is therefore recommended that future inheritance studies of leaf rust resistance in durum wheat carried out in the greenhouse should be complemented with field observations.

Complementary genes conditioning leaf rust resistance with a partially dominant interaction have not previously been reported in durum wheat. The genes *Lr27* and *Lr31* are the only known complementary genes that impart leaf rust resistance in bread wheat (Singh & McIntosh, 1984a; 1984b). Although the presence of two complementary genes is necessary for the expression of resistance, the pathogen can mutate from avirulence to virulence at a single locus. The host-pathogen interaction is therefore not different from a single resistance gene.

### **Identification and mapping of *Lr3* and a linked leaf rust resistance gene in durum wheat (Paper IV)**

Determination of the chromosome location of genes identified in our study would reveal important information for their efficient deployment in durum wheat and for further breeding. It would also reveal whether any of these genes are already known designated genes identified previously in bread wheat or if they are novel.

Three AFLP markers were found to be associated with the leaf rust resistance gene present in Camayo; the nearest AFLP marker (P33/M48<sub>352</sub>) being at a distance of 1.1 cM. One of the linked AFLP markers mapped to the long arm of chromosome 6B in the ITMI population (derived from the cross of a synthetic wheat with the bread wheat cultivar Opata) for which a dense molecular map is publicly available (Roeder *et al.*, 1998). The genomic location of the leaf rust resistance gene present in Camayo was thereby also mapped to the long arm of chromosome 6B.

Of the already known wheat leaf rust resistance genes located in chromosome 6B (McIntosh *et al.*, 1995a), *Lr3* was the most likely candidate to be present in durum wheat. Three different alleles, *Lr3a* (*Lr3*), *Lr3bg*, and *Lr3ka* (Haggag &

Dyck, 1973; McIntosh *et al.*, 1995) have been described at the *Lr3* locus. The RFLP marker, *Xmwig798*, had previously been reported to co-segregate with *Lr3a* in the bread wheat cultivar Sinvalocho MA (Sacco *et al.*, 1998). In the study presented here the sequence tagged site (STS) version of the same marker, *Xmwig798*, generated by Kunzel *et al.* (2000) in the Atil C2000 × Camayo and Atil C2000 × Storlom F<sub>3</sub> populations was used for validating if any of the two genes could be *Lr3a*.

The marker *Xmwig798* was completely associated with the resistance gene in Storlom indicating that this gene is most likely *Lr3a*. This marker was absent in Camayo. The bread wheat near-isogenic lines carrying each of the alleles at the *Lr3* locus, i.e. *Lr3a*, *Lr3bg* and *Lr3ka*, were also tested for the presence of *Xmwig798*. Since *Xmwig798* detected all three alleles, it turned out to be a locus-specific rather than allele-specific marker. We therefore cannot exclude that gene present in Storlom is *Lr3bg* rather than *Lr3a*. The infection type response of *Lr3ka* is very different from the infection type of Storlom, and therefore leaves that option out.

The allelic relationship between the two genes was further investigated using approximately 200 F<sub>3</sub> families from the Camayo × Storlom cross. Phenotypic evaluation for rust reaction and molecular characterization of the AFLP markers and *Xmwig798* indicated that the two genes were most likely very closely linked in repulsion. The AFLP marker most closely associated to the gene in Storlom was located at a distance of 1.4 cM to *Xmwig798*. Only one of the 200 F<sub>3</sub> families evaluated had susceptible plants. The remaining had the leaf rust responses of either one of the parents or had plants with a combination of both. This apparent recombinant and the fact that Camayo does not have *Xmwig798* supports the hypothesis that these genes are not alleles of the same locus but are in fact very closely linked. The phenotypic response of Camayo to *P. triticina* race BBG/BN is also different from the responses of any of the near-isogenic bread wheat lines carrying *Lr3a*, *Lr3bg* or *Lr3ka*. Further studies on race-specificity with races avirulent and virulent to *Lr3a*, *Lr3bg* and *Lr3ka* could reveal additional information on the allelic relationship between the two genes. Since both Camayo and Storlom also carry an additional common resistance gene which is effective to all Mexican *P. triticina* races except BBG/BN, identification of allelic relationship based on host-pathogen interaction studies were not possible.

The leaf rust resistance genes identified in the genetic and mapping studies can be effectively deployed in countries where prevalent durum wheat *P. triticina* races are avirulent on these genes. Leaf rust resistance gene *Lr3a* is considered to have a low value in bread wheat because virulent races are common worldwide (McIntosh *et al.*, 1995a). However, the *P. triticina* races from durum wheat are known to be avirulent on most of the known leaf rust resistance genes (Huerta-Espino & Roelfs; 1992; Ordonez *et al.*, 2004). Many of these known *Lr* genes, such as *Lr3*, would therefore be useful for durum wheat especially if present together with other genes such as the one that conferred resistance to the durum wheat cultivar Altar C84 for 16 years until succumbing to race BBG/BN. This still undesignated gene in Altar C84 has played an important role to protect CIMMYT

durum wheat germplasm from *P. triticina* races of bread wheat at least in Mexico, where *P. triticina* races from bread wheat did not evolve for virulence to this gene.

The protection against *P. triticina* conferred by the known race-specific genes in bread wheat has turned out to be short-lived. Therefore, the same could be expected in durum wheat. Use of identified race-specific genes in combinations would enhance their longevity.

## Conclusions

From the work presented and discussed, the following general conclusions can be drawn:

- 1) Losses due to leaf rust can be effectively reduced to negligible levels by the deployment of resistant durum wheat cultivars. Race-specific resistance protected the crop most effectively against severe leaf rust epidemics. A high level of slow rusting resistance identified in this study was associated with reduced yield losses. The best slow rusting resistant lines can be utilized by breeding programs for generating durable resistance.
- 2) Uredinium size determined in the greenhouse was the best predictor for slow rusting resistance in durum wheat under field conditions. Among slow rusting resistance components, uredinium size is the easiest to measure and it had no significant genotype  $\times$  environment interaction.
- 3) Five distinct sources of race-specific resistance were identified in CIMMYT durum wheat germplasm. Although these genes demonstrate diversity for resistance their longevity can be enhanced if utilized in combinations in commercial cultivars.
- 4) Molecular techniques proved to be effective for mapping leaf rust resistance genes in durum wheat. Two closely linked genes were mapped to the long arm of chromosome 6B and one of the genes was identified as *Lr3a*. Although *Lr3a* is known to occur in bread wheat, it was confirmed for the first time in durum wheat. The other gene appears to be novel.

## Future perspectives

Inheritance studies for slow rusting resistance in durum wheat are needed to enhance the knowledge of the genetic basis and genetic diversity of such resistance. Eight slow rusting resistant parents were crossed with a susceptible durum wheat line in order to determine the genetic nature and basis of such resistance in CIMMYT durum wheat germplasm. These slow rusting lines were also intercrossed to investigate the genetic diversity for this type of resistance.



Complex crosses involving three and four slow rusting resistant parents were also made to accumulate slow rusting genes for the development of durum wheat cultivars with higher resistance and negligible yield losses under high leaf rust pressure.

A set of differentials with known *Lr* genes and unknown leaf rust resistance genes from durum wheat origin must be developed for determining variation in populations of *P. triticina* from durum wheat. These differentials can also serve for testing for the presence (postulating) of resistance genes in durum wheat. Highly susceptible semi-dwarf durum wheat lines that lack any known or unknown race-specific resistance genes were recently developed at CIMMYT as the first step to achieve this objective (Huerta-Espino & Singh, pers. com.).

Chromosome mapping is necessary for further characterization of the remaining race-specific resistance genes identified in this study and for mapping genes that confer slow rusting resistance. Molecular markers can be used for mapping as was shown from the study described here. In addition, PCR based molecular markers tightly linked to race-specific resistance genes should be developed for marker assisted gene pyramiding in breeding programs to enhance the longevity of resistance.

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