CHAPTER FIVE

Will Stem Rust Destroy the World's Wheat Crop?

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Race Ug99, or TTKSK, of fungus *Puccinia graminis tritici*, causing stem or black rust disease on wheat (*Triticum aestivum*), first identified in Uganda in 1998 has been recognized as a major threat to wheat production. Its spread in 2006 to Yemen and Sudan and further spread towards North Africa, Middle East and West-South Asia is predicted -aided by predominant wind currents and large areas of wheat varieties that are susceptible and grown under environments favorable for survival and multiplication of the pathogen. This has raised serious concerns of major epidemics that could destroy the wheat crop in these primary risk areas. Detection in Kenya of a new variant TTKST in 2006 with virulence to gene *Sr24*, which caused severe epidemics in 2007 in some regions of Kenya and rendered about half of the previously known Ug99-resistant global wheat materials susceptible, has further increased the vulnerability globally. Rigorous screening since 2005 in Kenya and Ethiopia of wheat materials from 22 countries and International Centers has identified low frequency of resistant materials that have potential to replace susceptible cultivars. Diverse sources of resistance, both race-specific and adult-plant type, are now available in high-yielding wheat backgrounds and are being used in breeding. The proposed strategy is to deploy spring wheat varieties possessing durable, adult plant resistance in East Africa and other primary risk areas to reduce inoculum and selection of new virulences capable of overcoming undefeated race-specific resistance genes. Race-specific resistance genes can then be deployed in secondary risk areas preferably in combinations. We believe that Ug99 threat in most countries can be reduced to low levels by urgently identifying, releasing and providing seed of new high yielding, resistant varieties.

1. Introduction

Wheat, one of the most important staple food crops, is grown on about 225 million ha worldwide from the equator to latitudes of 60°N and 44°S and at altitudes ranging from sea level to more than 3000 m. Approximately 600 million tons of wheat is produced annually, roughly half of which is in developing countries (Aquino *et al.*, 2002). The only limitation to production is humid and high-temperature areas in the tropics and high-latitude environments where fewer than 90 frost-free days are available for crop growth. The world's largest producers of wheat are China, India, and the USA, producing annually 100, 70, and 64 million tons with productivities

of 3.8, 2.6, and 2.9 t/ha, respectively (Aquino *et al.*, 2002). Only 10% of total wheat produced is sold on the export market, the primary exporting countries are USA, Canada, Australia, and France, and developing countries consume most of the wheat sold on the export market (Aquino *et al.*, 2002). In some countries, such as those in North Africa, per capita consumption of wheat is as high as 240 kg (FAO, 2001).

Globally important fungal diseases of wheat, caused by biotrophs (obligate parasites), include the three rusts, powdery mildew, and the bunts and smuts; whereas, those caused by hemibiotrophs (facultative parasites) include *Septoria tritici* leaf blotch, *Septoria nodorum* blotch, spot blotch, tan spot, and Fusarium head blight (scab). The obligate parasites are highly specialized and significant variation exists in the pathogen population for virulence to specific resistance genes. Evolution of new virulence through migration, mutation, recombination of existing virulence genes and their selection has been more frequent in rust and powdery mildew fungi. Therefore, enhancement of the knowledge on the genetic basis of resistance and breeding-resistant cultivars to these diseases has received larger attention.

Stem or black rust of wheat, caused by fungus *Puccinia graminis* Pers. f. sp. *tritici* Eriks. & E. Henn., historically is known to cause severe devastation periodically and was most feared disease in various countries in all continents where wheat is grown. The fear from stem rust is understandable because an apparently healthy looking crop of a susceptible cultivar about 3 weeks prior to harvest could reduce to a black tangle of broken stems and shriveled grains by harvest. There are several major wheat production areas worldwide in which stem rust can cause severe losses due to environments that are conducive to disease development. According to Saari and Prescott (1985), stem rust was historically a major problem in all of Africa, the Middle East, all of Asia except Central Asia, Australia and New Zealand, Europe, and the Americas (both North and South). Although the last major stem rust epidemics occurred in Ethiopia during 1993 and 1994 (Shank, 1994) when a popular wheat variety ''Enkoy'' suffered major losses, the rest of the world has practically remained unhurt from stem rust for over three decades. Because worldwide epidemics of other two rust diseases, leaf (or brown) rust caused by *P. triticina* and stripe (or yellow) rust caused by *P. striiformis*, were more frequent in recent years, there has been a major shift in priority and resources away from stem rust research and breeding to such an extent that in some countries testing and breeding for stem rust resistance have been suspended. Consequently, it is not surprising to find new wheat pathologists and breeders who have not even seen stem rust infection in the field.

In 1998, severe stem rust infections were observed on wheat in Uganda, and a race, designated as Ug99 with virulence on *Sr31*, was detected (Pretorius *et al.*, 2000). Race Ug99 was subsequently detected in Kenya and Ethiopia in 2005 (Wanyera *et al.*, 2006), and in Sudan and Yemen in 2006 (Jin *et al.*, unpublished data). A new variant of this race with virulence

to *Sr24* was detected in Kenya in 2006 (Jin *et al.*, 2007a). It is predicted that these races will migrate to North Africa, Middle East, Asia, and beyond and challenge wheat scientists and policy makers to identify, develop, and replace most of the wheat varieties currently grown in these areas that are susceptible. Although major losses to stem rust have already occurred in Kenya during the 2007 crop season in the ''Narok'' area and fungicides had to be used heavily to protect the wheat crop in other later-sown areas, the authors strongly believe that the major threat of stem rust once again destroying the vast span of wheat crops in its predicted migration path can be mitigated. This optimism arises from the fact that wheat scientists and policy makers worldwide have begun to respond positively to the alarm raised by Noble Laureate Dr. Norman E. Borlaug in 2005 (CIMMYT, 2005), which resulted in the formation of the ''Global Rust Initiative'' (www.globalrust.org).

2. Stem Rust Disease, Pathogen, and Epidemiology

Stem rust is caused by fungus *P. graminis* Pers. f. sp. *tritici* Eriks & E. Henn and belongs to one of several formae speciales in *P. graminis*. Stem rust appears as elongated blister-like pustules, or uredinia, most frequently on the leaf sheaths of a wheat plant, but also on true stem tissues, leaves, glumes, and awns. Stem rust pustules on leaves develop mostly on the lower side, but may penetrate and produce limited sporulation on the upper side. On the leaf sheath and glumes, pustules rupture the epidermis and give a ragged appearance. Masses of urediniospores produced on the pustules are brownish red in color, and easily shaken off the plants. As infected plants mature, uredinia convert into telia, changing color from red into dark brown to black, thus the disease is also called black rust. Teliospores are firmly attached to plant tissue.

The fungus is heteroecious, alternating between a telial host in Poaceae and an aecial host in Berberidaceae, and macrocyclic, with five spore states that are distinct in morphology and function. Crop species as primary hosts include bread wheat, durum wheat, barley, and triticale. There are a large number of species in *Berberis* and *Mahonia* that are susceptible to *P. graminis* (Roelfs, 1985), but the common barberry, *B. vulgaris*, is considered to be the most important alternate host. Aeciospores arising from an alternate host can be a source of inoculum. Historically, the source of inoculum from *B. vulgaris* was important in North America and northern and eastern Europe. This source of inoculum has generally been eliminated or greatly reduced by removal of common barberry from the proximity of wheat fields.

Urediniospores disseminate to newly emerged tissues of the same plant or adjacent plants to cause new infections, or can be transported through wind in long distances. Long-distance transport through prevailing winds is known to occur across the North American Great Plains (Roelfs, 1985), from Australia to New Zealand, and rarely to a distance of about 8000 km from southern Africa to Australia (Luig, 1985). In the case of long-distance dispersal, spore depositions on crops in a new area are often associated with rain showers. Stem rust urediniospores are rather resistant to atmospheric conditions if their moisture content is moderate (20–30%). The minimum, optimum, and maximum temperatures for urediniospore germination are 2, 15–24, and 30 °C; and for sporulation 5, 30, and 40 °C (Roelfs *et al.*, 1992), thus providing a vast range of favorable environmental conditions. Urediniospores initiate germination within 1–3 h of contact with free moisture over a range of temperatures. In field conditions, 6–8 h of dew period or free moisture from rains is required for the completion of infection process.

After two devastating stem rust epidemics in North America in 1904 and 1916, an important finding came from the pioneering work of E. C. Stakman (Stakman and Piemeisel, 1917) who showed that the stem rust pathogen had various forms or races. These races varied in their ability to infect different wheat varieties which later were found to carry distinct resistance genes or combinations thereof. At present wheat scientists use wheat lines that usually carry a single race-specific resistance gene to determine avirulence/virulence characteristics of a race. Mutation toward virulence in existing populations followed by selection on susceptible hosts is at present considered to be the most important evolution mechanism for stem rust pathogen to acquire new virulence to overcome resistance conferred by race-specific resistance genes. Where an alternate host is present, it is possible to have new combinations of virulences through sexual recombination; however, it is limited at present to few areas of the world. Rare asexual recombination is also known to occur through exchange of nuclei between conjugating hypha of two races that have by chance infected same tissues.

Wheat rust pathogens are biotrophs and therefore need living wheat plants or other secondary hosts for survival in the absence of alternate hosts. They produce large numbers of urediniospores during the crop season and wind dispersion transmits these urediniospores onto the same or new host plants in the vicinity or distantly. Typically, most spores will be deposited close to the source (Roelfs and Martell, 1984); however, long-distance dispersal is well documented with three principal modes of dispersal known to occur. The first mode of dispersal is single event, extremely long-distance (typically cross-continent) dispersal that results in pathogen colonization of new regions. Dispersion of this type is rare under natural conditions and by nature inherently unpredictable. It is also difficult to specifically attribute long-distance dispersal. However, rusts are one pathogenic group with reasonably strong evidence for unassisted, long-distance dispersal under

natural airborne conditions. Several examples of long-distance dispersal have been described by Brown and Hovmøller (2002), including the introduction of sugarcane rust into the Americas from Cameroon in 1978 and a wheat stem rust introduction into Australia from southern Africa in 1969. Both these examples provide strong evidence for being unassisted natural long-distance wind-borne dispersals. The enabling factor in this mode of dispersal for rusts is the robust nature of spores ensuring protection against environmental damage (Rotem *et al.*, 1985). Deposition in new areas is primarily through rain-scrubbing of airborne spores onto susceptible hosts (Rowell and Romig, 1966).

Assisted long-distance dispersal, typically on travelers clothing or infected plant material, is another increasingly important element in the colonization of new areas by pathogens. Despite strict phytosanitary regulations, increasing globalization and air travel both increase the risk of pathogen spread. Evidences strongly support an accidental introduction of wheat stripe rust into Australia in 1979, probably on travelers clothing, from Europe (Steele *et al.*, 2001). More recently, concerns over nonaccidental release of plant pathogens as a form of ''agricultural bio-terrorism'' have arisen, with wheat stem rust considered one pathogen of concern (Hugh-Jones, 2002) primarily due to its known ability to cause devastating production losses to a major food staple (Leonard, 2001).

The second major mode of dispersal for pathogens like rusts is stepwise range expansion. This typically occurs over shorter distances, within a country or a region, and has a much higher probability than the first described dispersal mode. This probably represents the most common or normal mode of dispersal for rust pathogens. A good example of this type of dispersal mechanism would include the spread of a *Yr9*-virulent race of *P. striiformis* that evolved in eastern Africa and migrated to South Asia through the Middle East and West Asia in a stepwise manner over about 10 years, and caused severe epidemics along its path (Singh *et al.*, 2004b).

The third mode of dispersal, extinction, and recolonization, could perhaps be considered a sub-mechanism of stepwise range expansion. This mechanism occurs in areas that have unsuitable conditions for year-round survival. Typically these are temperate areas where hosts are absent during winter or summer. A good example of this mechanism is the ''*Puccinia* pathways'' of North America—a concept that arose from another pioneering work of Stakman (1957) in which rust pathogens over winter in southern USA or Mexico and recolonize wheat areas in the Great Plains and further north following the prevailing south-north winds as the wheat crop season progresses. The second well-documented extinction–recolonization example is that of wheat stripe rust survival and spread from mountains in the Gansu province of China (Brown and Hovmøller, 2002) and wheat rusts in the Himalayas and Nilgiri Hills in northern and southern India, respectively (Nagarajan and Joshi, 1985) where susceptible hosts can be found year

round and environmental conditions are favorable for the pathogen to survive. Urediniospores from these areas are then blown to wheat fields in other areas and initiate disease.

3. Breeding for Resistance

3.1. Historical account

It was not until the beginning of twentieth century and soon after the rediscovery of Mendel's laws, that Biffen (1905) demonstrated that inheritance of resistance to wheat stripe rust followed Mendel's laws. Strong emphases to identify resistance to stem rust and to breed resistant wheat varieties were initially given in the USA, Canada, Australia, and Europe. Although the major epidemic of 1916 in the USA and Canada had already triggered extensive research on stem rust, efforts in the USA, Canada, and Australia were intensified further with subsequent epidemics in the following decades. Although resistance present in some hexaploid wheat sources were used in breeding during early years, the most successful control of stem rust came when H. K. Hayes in the University of Minnesota and E. S. McFadden in South Dakota State University transferred the stem rust resistance from tetraploid sources ''Iumillo'' durum and ''Yaroslav'' emmer, respectively, into bread wheat that gave rise to hexaploid wheat varieties ''Thatcher'' and ''Hope'' (Kolmer, 2001). Although several race-specific genes are present in Hope and Thatcher, the most effective component of the resistance in these two varieties is due to adult plant resistance. Thatcher and Hope, Hope sib ''H44–24a,'' and other varieties derived from these parents such as ''Selkirk'' and ''Chris'' that combined resistance to stem rust from other sources including gene *Sr6* found to be present in a plant selection by J. McMurachy in 1930. ''Kenya 58'' and other Kenyan varieties carrying the same gene *Sr6* were also used extensively in Australia by I. A. Watson and in Mexico by N. E. Borlaug. Efforts to find a solution to the stem rust problems facilitated global collaboration amongst wheat scientists who shared, grew, and evaluated wheat germplasm in the quest of finding different sources of resistance to stem rust. Resistant wheat materials developed at Njoro, Kenya through the support from Canadian scientists in 1960s and 1970s contributed substantially to international breeding efforts. Resistance from Hope and Chris formed the foundation of the high-yielding, semi-dwarf wheat varieties that led to ''Green Revolution'' in the 1970s.

3.2. International cooperation

Although germplasm exchange was common among wheat scientists, the International Spring Wheat Rust Nursery Program, initiated in 1950 by B. B. Bayles and R. A. Rodenhiser of USDA-ARS (United States

Department of Agriculture—Agricultural Research Services), Beltsville, formed the basis of a true international collaboration and operated continuously until the mid 1980s. The objectives of the program were (1) to find new genes or combinations of genes in wheat which condition field resistance to rusts throughout the world, and (2) to test new varieties and promising selections of wheat developed by plant breeders and pathologists for resistance to rusts. The germplasm and information generated were made available to the global wheat community. This nursery was the foundation of numerous other international nurseries and led to global cooperation to achieve resistance to diseases and pests of several crops. CIMMYT (International Maize and Wheat Improvement Center) and several other international research centers continue to use this methodology to not only distribute improved germplasm they develop but also to evaluate their performance for agronomic and disease resistance attributes.

3.3. Spread of semidwarf wheats with resistance to stem rust

The semidwarf wheat varieties developed by Dr. N. E. Borlaug in Mexico during early 1960s under the program sponsored by the Mexican Government and the Rockefeller Foundation were also resistant to stem rust and earlier in maturity compared to tall varieties grown previously. The two semidwarf "Green Revolution" mega-varieties, "Sonalika" and "Siete Cerros," continue to have moderate levels of resistance to race Ug99 even today; however, they were mostly replaced as they succumbed to leaf and yellow rusts and better varieties became available. These semidwarf varieties significantly reduced stem rust incidence in many areas, which is often attributed to a combination of resistance and early maturity that avoided stem rust inoculum buildup (Saari and Prescott, 1985). The tall variety "Yaqui 50," released in Mexico during the 1950s, and other *Sr2*-carrying semidwarf varieties released since then had stabilized the stem rust situation in Mexico and possibly in many other countries where modern semidwarf wheats were adopted. Changes in stem rust races have not been observed in Mexico for almost 40 years and natural infections are nonexistent.

Successful transfers and utilization of alien resistance genes *Sr24* and *Sr26* from *Agropyron elongatum* (*Thinopyrum ponticum*), *Sr31* located in the 1BL.1RS translocation from "Pektus" rye and an undesignated gene on 1AL.1RS translocation from "Insave" rye, *Sr36* from *T. timopheevi* and more recently *Sr38* from *T. ventricosum* further reduced stem rust incidence in various countries around the world in 1970s and 1980s. The alien resistance gene *Sr31* has been used in agriculture on the largest scale since 1980s in spring, facultative and winter wheat breeding programs worldwide except Australia. Its use in CIMMYT wheat improvement resulted in the release of several popular cultivars worldwide. The use of 1BL.1RS translocation was initially associated with increased grain yields and resistance to all three rusts

and powdery mildew as it carried resistance genes for all these diseases on the same translocation. Large-scale deployment of *Sr31* surprisingly did not result in its breakdown until the detection of race Ug99 in Uganda. In fact this gene probably further reduced the already low stem rust survival to almost nonexistent levels in most wheat growing regions to the extent that stem rust started to become a forgotten curse.

The decrease in incidence of stem rust to almost nonsignificant levels by the mid-1990s throughout most of the wheat producing areas worldwide were coincident with a decline in research and breeding emphasis to such a level that in many countries breeding was done in the absence of this disease. CIMMYT scientists continued to select for stem rust resistance in Mexico using artificial inoculation with six *P. graminis tritici* races of historical importance. New stem rust races have rarely occurred since the ''Green Revolution'' in Mexico (Singh, 1991). Moreover, a majority of wheat lines selected in Mexico remained resistant at international sites either due to absence of disease, inadequate disease pressure, or presence of races that lacked necessary virulence for the resistance genes contained in CIMMYT wheat germplasm.

Frequency of 1BL.1RS translocation went up to ~70% at one stage in CIMMYT's spring wheat germplasm but has declined to about 30% in more recent advanced lines. Such alien chromosome segments on the one hand are very useful for controlling multiple diseases, but on the other hand could lead to ''vertifolia'' or a masking effect (Vanderplank, 1963) resulting in decrease in frequency or even loss of other useful genes, especially minor types, in breeding materials. All wheat lines of CIMMYT origin evaluated in Kenya since 2005, irrespective of the presence or absence of 1BL.1RS translocation, were highly resistant to stem rust in Mexico and remain highly resistant in other parts of the world, indicating that the high frequency of this translocation in 1980s and 1990s cultivars explains only a portion of the current susceptibility of wheat germplasm to race Ug99 in Kenya. Jin and Singh (2006) compared seedling reactions of US wheat cultivars and germplasm with highly virulent races present in the USA and race Ug99. Several wheat lines, especially spring wheat that were highly resistant to US races and did not carry the1BL.1RS translocation, were also found to be susceptible to Ug99. This further supports the hypothesis that race Ug99 carries a unique combination of virulence to known and unknown resistance genes present in wheat germplasm. The major susceptibility is due to the specific nature of avirulence/virulence combination that Ug99 possesses, which had led to the susceptibility of many wheat materials irrespective of where they were developed.

3.4. Current knowledge of resistance to stem rust

At present 46 different stem resistance genes are catalogued and multiple alleles are known for three gene loci (Table 1). There are a few additional resistance genes that need further research before they can receive designation

Table 1 Origin and usefulness of designated *Sr*-genes in conferring seedling and/or adult plant resistance to Ug99 race of stem rust pathogen *Puccinia graminis* f. sp. *tritici*

Origin of *Sr* genes	Stem rust resistance (*Sr*) genes	
	Ineffective	Effective
Triticum aestivum	5, 6, 7a, 7b, 8a, 8b, 9a, 9b, 9f, 10, 15, 16, 18, 19, 20, 23, 30, 41, 42, Wld-1	28[a], 29[b], Tmp[a]
Triticum turgidum	9d, 9e, 9g, 11, 12, 17	2[b], 13[a,b], 14[a]
Triticum monococcum	21	22, 35
Triticum timopheevi		36[a], 37
Triticum speltoides		32, 39
Triticum tauschii		33[b], 45
Triticum comosum	34	
Triticum ventricosum	38	
Triticum araraticum		40
Thinopyrum elongatum		24[a], 25, 26, 43
Thinopyrum intermedium		44
Secale cereale	31	27[a], 1A.1R[a], R

[a] Virulence for the gene is known to occur in other races.
[b] Level of resistance conferred in the field usually not enough.

(McIntosh *et al.*, 1995). Several of these genes were incorporated into wheat from alien wheat relatives (Table 1). All designated genes, except *Sr2*, are race specific and are expressed in both seedling and adult plants. Race specificity derives from the gene-for-gene relationship between the host plant resistance gene and corresponding avirulence genes in the pathogen. With avirulent races a majority of stem resistance genes allows formation of tiny- to medium-sized uredinia, with limited sporulation, which are surrounded by a necrosis or chlorosis (McIntosh *et al.*, 1995). Genes that allow development of only microscopic or macroscopic hypersensitive reactions include *Sr5*, *Sr17*, *Sr27*, *Sr36,* and *Sr6* at cooler temperatures.

The adult plant resistance gene *Sr2* confers slow rusting (Sunderwirth and Roelfs, 1980). Combination of *Sr2* with other unknown slow rusting resistance genes possibly originating from Thatcher and Chris, commonly known as the "*Sr2*-Complex," provided the foundation for durable resistance to stem rust in germplasm from the University of Minnesota in the United States, Sydney University in Australia, and the spring wheat germplasm developed by Dr. N. E. Borlaug (McIntosh, 1988; Rajaram *et al.*, 1988). Unfortunately, not much is known about the other genes in the *Sr2* complex and their interactions. Knott (1988) has shown that adequate levels of multigenic resistance to stem rust can be achieved by accumulating approximately five minor genes.

US wheat cultivar Chris, which is not known to carry *Sr2* but possesses several seedling resistance genes including *Sr7a* (Singh and McIntosh, 1987) displayed adequate level of resistance to Ug99 in the field in Kenya. Preliminary studies of inheritance of seedling resistance to Ug99 in Chris indicated that Ug99 resistance in Chris is controlled by two complementary recessive genes (Jin, 2007), and the same seedling resistance is present in AC Barrie (a Canadian spring wheat cultivar), Thatcher, and Bonza 65 (a CIMMYT-derived cultivar). Singh and McIntosh (1987) indicated the possibility that the adult plant resistance to *Sr7a*-avirulent Australian races may involve interaction of the moderately effective gene *Sr7a* and other unknown adult plant resistance genes. Seedling tests indicated that Ug99 is virulent on the *Sr7a*-tester line (Jin *et al.*, 2007b) although Chris did show seedling resistance. Singh and McIntosh (1987) indicated that resistance conferred by *Sr7a* is difficult to evaluate both in seedlings and adult plants when the gene is present alone. Therefore, at this stage we cannot determine the role *Sr7a* may have played in resistance of ''Chris'' observed in Kenya. Even though seedling tests indicate that *Sr23*, another gene whose expression is difficult to evaluate in seedlings and adult plants when present alone, may be ineffective against Ug99, adequate resistance in ''Selkirk'' may involve interactions of moderately effective genes *Sr2* and *Sr23* (linked to leaf rust resistance gene *Lr16*) and perhaps additional unknown adult plant resistance genes. These observations, although they still require validation through genetic analyses, indicate that complex resistance to stem rust present in some tall cultivars developed in the 1960s and 1970s continue to remain effective.

4. Race Ug99 and Why it is a Potential Threat to Wheat Production

4.1. Avirulence/virulence genes in Ug99

Race Ug99, that emerged in Uganda in 1998 and was identified in 1999 (Pretorius *et al.*, 2000), is the only known race of *P. graminis tritici* that has virulence for gene *Sr31* known to be located in the translocation 1BL.1RS from rye (*Secale cereale*). It was designated as TTKS by Wanyera *et al.* (2006) using the North American nomenclature system (Roelfs and Martens, 1988) and more recently as TTKSK after a fifth set of differentials was added to further expand the characterization (Jin *et al.*, 2008). The most striking feature of race Ug99 is that it not only carries virulence to gene *Sr31* but also this unique virulence is present together with virulence to most of the genes of wheat origin, and virulence to gene *Sr38* introduced into wheat from *Triticum ventricosum* that is present in several European and Australian cultivars and a small portion of new CIMMYT germplasm (Table 1, Jin *et al.*,

2007b). This virulence combination might have accounted for the widespread Ug99 susceptibility in wheat varieties worldwide. A variant of Ug99 with added virulence to *Sr24* was detected in 2006 in Kenya. It is anticipated that mutation toward more complex virulence will likely occur as the fungal population size increases and selection pressure is placed on the population by resistant varieties.

4.2. Current distribution of race Ug99

As described by Singh *et al.* (2006) Ug99 was first identified in Uganda in 1998, although there is some evidence indicating that the race may have been present in Kenya since 1993, and had spread to most of the wheat growing areas of Kenya and Ethiopia by 2003. In 2005, Ethiopian reports confirmed its presence in at least six dispersed locations (Fig. 1). The East African highlands are a known ''hot-spot'' for the evolution and survival of new rust races (Saari and Prescott, 1985). The favorable environmental conditions and the presence of host plants year-round favor the survival and buildup of pathogen populations. Available evidence emerging from

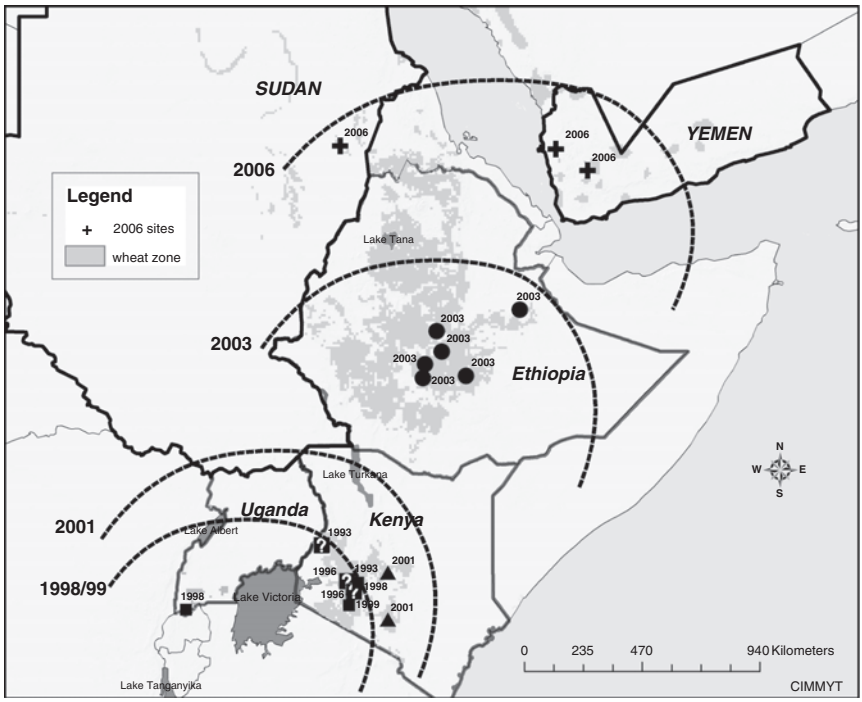

Figure 1 Regional wheat production areas and known distribution of stem rust pathogen race Ug99 as of September 2007.

the East African countries indicates that Ug99 has exhibited a gradual stepwise range expansion, following the predominant west-east airflows.

The confirmed range of Ug99 continues to expand, with new sites being recorded beyond the previously confirmed three East African countries Uganda, Kenya, and Ethiopia. In early 2006 (February/March), stem rust—tentatively caused by the Ug99 race—was reported from a site near New Halfa in eastern Sudan. Later the same year (October/November), reports were obtained from at least two sites in western Yemen (Fig. 1). Subsequent race analysis of samples from these sites, undertaken by the USDA-ARS Cereals Disease Laboratory, St. Paul, MN, USA confirmed the presence of Ug99 in these countries. The observed expansion into new areas is in-line with previous predictions on the likely movement of Ug99 (Hodson *et al.*, 2005; Singh *et al.*, 2006) and fits the stepwise dispersal model following prevailing winds as outlined by Singh *et al.* (2006). The exact route taken by Ug99 to reach Yemen is unknown, but neither the possibility of transfer from south-eastern/eastern Ethiopia on the fringes of the southwestern monsoon system nor the transfer from eastern Sudan/Eritrea/northern Ethiopia can be excluded (Fig. 2).

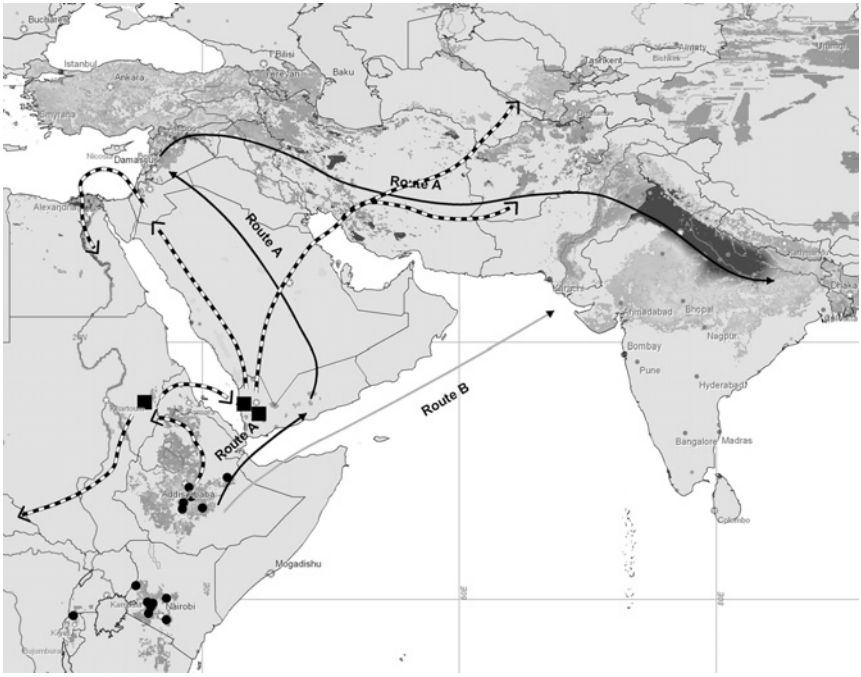

Figure 2 Updated potential migration routes of Ug99 based on historical precedence and recent studies of actual wind movements.

4.3. Predicting Ug99 migration to other wheat areas

Crossing of the Red Sea into Yemen by Ug99 is regarded as being particularly significant, as the pattern of regional airflows, combined with historical recorded migration of *Yr9*-virulent stripe rust race (Singh *et al.*, 2004b), both support the potential for onward movement from Yemen into significant wheat production areas of the Middle East and West-South Asia. On the basis of airflow patterns, Fig. 2 updates the potential migration routes A and B described in Singh *et al.* (2006). Nothing in the current observed spread of Ug99 indicates any basis for changing this hypothesis. Given this situation, the buildup of significant levels of Ug99 urediniospores in Yemen would be a cause for concern.

More detailed analysis of further potential onward movements undertaken using the HYSPLIT (HYbrid Single-Particle Lagrangian Integrated Trajectory) airborne particle trajectory model developed by NOAA (Draxler and Rolph, 2003) supports the hypothesis that Yemen could be a staging post for onward movement into the Middle East and Asia. Figure 3 illustrates 72-h airborne particle trajectories, derived from HYSPLIT, using

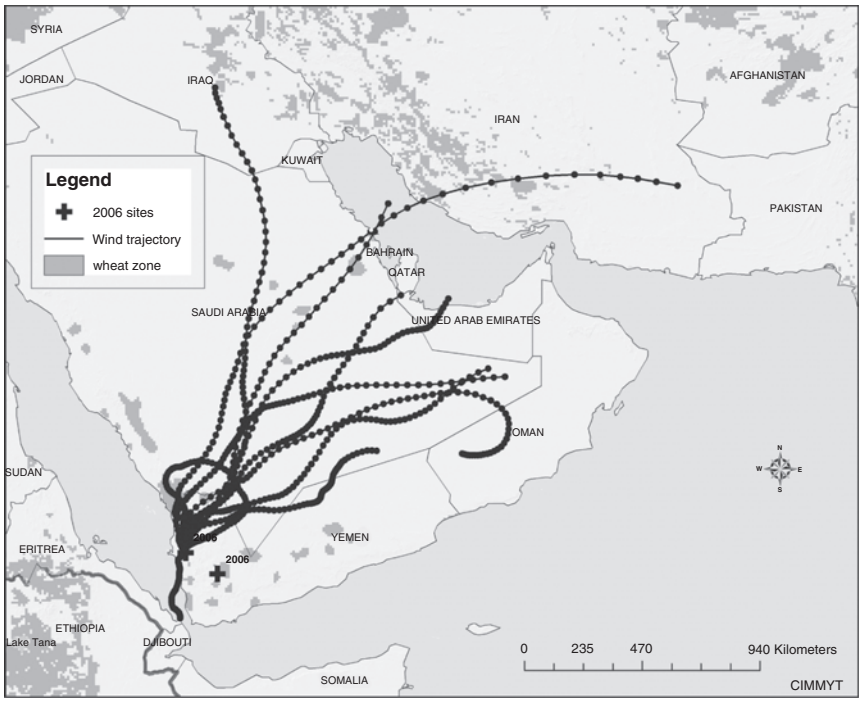

Figure 3 Air-borne particle trajectories, derived from the HYSPLIT model, originating from the confirmed Ug99 site of Al Kedan, Yemen (trajectories represent weekly 72-h movements for the period 1st December 2006 to 28th February 2007).

the confirmed Ug99 site Al Kedan in Yemen as a source. The trajectories shown are for weekly intervals during the period 1st December 2006 to 28th February 2007—a period in which wheat would be present and at a potentially susceptible growth stage in areas north of Yemen. During this period there was a clear tendency for airborne trajectories, originating at Al Kedan, to follow a north-easterly routing heading toward the wheat producing areas of Saudi Arabia, Iraq, and Iran. Similar results were obtained from an identical analysis covering the period 1st December 2005 to 28th February 2006, supporting the notion that the possibility of onward movements from sites in Yemen in the direction of key wheat areas occurs on a regular basis.

Immediate onward movements from eastern Sudan are potentially less problematic as airflow models indicate that direct movements in a northerly direction into the important wheat areas of the Nile valley are unlikely. However, given the uncertainty and complexity of airflows in this region the possibility of spores reaching these areas can never be totally excluded. In addition, there is a very real risk that spores could move northwards up the Arabian Peninsula from Yemen, enter the Nile Delta and then cycle back south down the Nile Valley. The *Yr9*-virulent stripe rust race did reach Egypt soon after its detection in Yemen (Singh *et al.*, 2004b). Sudan had escaped stripe rust because wheat is grown under relatively warm conditions, which is unfavorable for stripe rust survival.

At present, no known long-distance, single event ''random jump'' type movement (assisted or natural) has been recorded for Ug99. But with an expanding known range for the pathogen and the high mobility of people both regionally and internationally, there is a clear need for continued monitoring and surveillance in wheat areas beyond the immediate at risk region. Presence of the *Sr24*-virulent variant of Ug99 first identified in Kenya in 2006 has not yet been detected beyond Kenya, even though it was widespread in epidemic form in Kenyan highlands on the *Sr24* carrying variety ''Kenya Mwamba.''

4.4. Resistance/susceptibility of current wheat germplasm

Reynolds and Borlaug (2006) estimated that the potential area under the risk from Ug99 along the natural migration path in North Africa, Middle East and Asia (excluding China) might amount to 50 million ha of wheat, that is, about 25% of the world's wheat area and accounting for an estimated 19% of global production amounting to about 117 million tons. An estimated 1 billion people live within these wheat production areas.

Extensive screening of global wheat varieties for resistance to Ug99 has been undertaken at key sites in Kenya and Ethiopia (principally Njoro, Kenya and Kulumsa, Ethiopia) and results summarized by Singh *et al.* (2006). Available screening data has been linked via known pedigrees to databases

on areas planted to known varieties (CIMMYT, unpublished data). Prior to 2006, data were available for 10 countries in the Africa/Asia region with limited susceptibility ratings for Ug99 recorded for wheat varieties covering an estimated 44 million ha. By the end of 2006, the screening dataset had been extended to include germplasm from 18 countries in the region, including China, with more detailed resistance/susceptibility ratings obtained on varieties covering an estimated 75 million ha (Fig. 4). A summary of the area by susceptibility rating data is given in Table 2. Varieties exhibiting any observed resistance to Ug99 only account for 5% of the total estimated area in the 18 countries. The huge areas observed in India and Pakistan result from the predominance of ''mega-cultivars'' ''PBW343'' and ''Inqualab 91'' in the two countries, both of which are susceptible to Ug99. Further screening of additional varieties from 22 countries undertaken in Kenya during 2007 indicated a similar low frequency of resistant materials; however, the database mentioned above has not been updated yet.

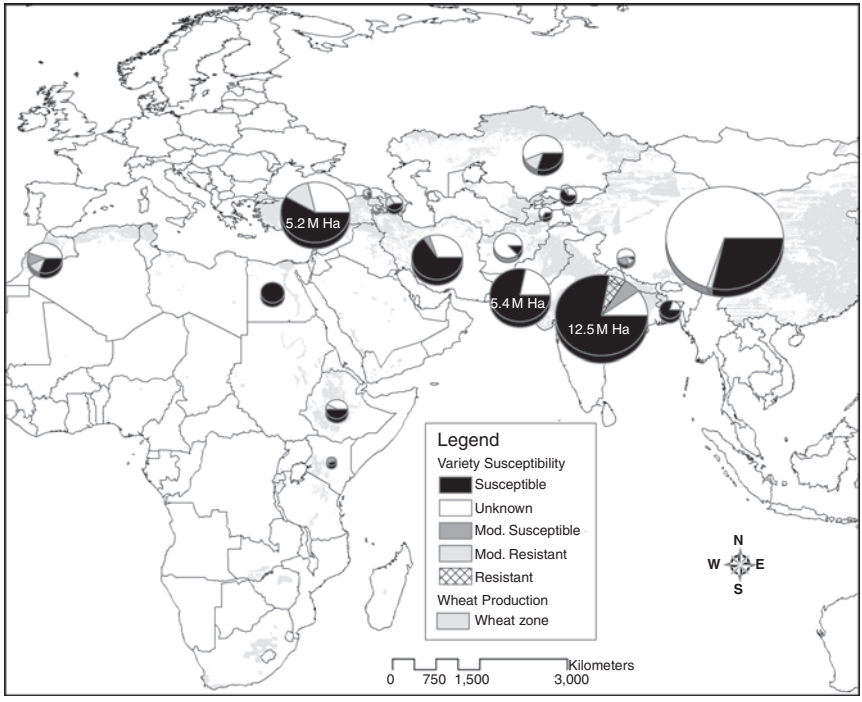

Figure 4 Summary of areas planted to known wheat varieties from 18 countries by their resistance/susceptibility groupings to Ug99 from screening results in Kenya and Ethiopia during 2005 and 2006 (symbols are scaled according to recorded areas by country).

Table 2 Estimated areas planted to wheat varieties in 18 African and Asian countries by resistance/susceptibility grouping to Ug99, based on screening data 2005, 2006 in Njoro, Kenya

	Estimated area	
Resistance/susceptibility group	million ha	%
Unknown	30.71	41.1
Susceptible	38.54	51.6
Moderately susceptible	1.73	2.3
Resistant	1.04	1.4
Moderately resistant	2.72	3.6

The seemingly favorable environmental conditions, coupled with the extensive coverage of susceptible wheat varieties is a grave cause of concern if Ug99 does spread unchecked. Conditions favorable for outbreaks of epidemics currently exist in the migration path of highest probability. If an epidemic from Ug99 does occur, extremely large numbers of wheat farming families may be seriously affected—especially those who have few alternative livelihoods. In these circumstances, landless laborers dependent on agricultural jobs would also be seriously affected, and one could anticipate an increase in the rural–urban migration of landless laborers and small farmers. If large production losses occur there would be significant implications for rural and national economic growth rates in seriously affected countries, and could even affect global wheat markets. Given the serious implications, there is a clear need for improved monitoring, surveillance, and early warning to directly support the efforts of wheat researchers and national policy makers working to prevent the spread of the disease and to alleviate its impacts.

4.5. Can a Ug99 pandemic be predicted?

A frequently asked question these days is when will Ug99 cause a major epidemic? However, the answer is very difficult because combination of factors occurring together is necessary for any epidemic to build up, and these could be different in different areas. Obviously susceptible host, virulent pathogen races, and a conducive environment are primary factors, but other more important factors in many areas are where and how much inoculum survives during the off-season, and when disease establishes in commercial fields.

Let us take a few examples to illustrate this further. In Kenya, where stem rust is endemic, that is, it is present throughout the year in the vicinity of wheat crops sown at different times and at different elevations, it took only one year from the detection in 2006 of the TTKST variant of Ug99 to cause

epidemics in 2007 on a previously resistant variety ''Kenya Mwamba'' and other varieties that were already susceptible to the original Ug99. Weather conditions during the 2006/07 period, October–April, were unexpectedly favorable for stem rust due to higher than normal rainfall, which allowed multiplication of race TTKST not only on wheat crop but also on off-season voluntary wheat plants. The specific combination of both changing virulence patterns and favorable weather conditions may have been the drivers of the observed epidemic.

Conversely, a Ug99 epidemic has not occurred in Ethiopia even though it was first detected in 2003 and sporadic infections were found on the commercial wheat crop. Huerta-Espino (1992) reported the presence of a *Sr36*-virulent race from samples collected in 1987 in Ethiopia; however, not until 1993 did an epidemic occur on the variety Enkoy that was previously protected by gene *Sr36*. The 1993 epidemic probably left enough inoculum to survive during the off-season on susceptible voluntary plants to cause a repeat second epidemic during 1994. Occurrence of epidemics had stopped by 1995, as the area under Enkoy was significantly reduced.

Stakman (1957) summarized the difficulties he faced in preventing stem rust epidemics in North America. It took 11 years for race 15B, first identified in collections from barberries in Iowa in 1939, to cause a pandemic in 1950 in Mexico, and USA, and Canada in 1953, 1954. However, it may not be necessary for a rust pathogen to take such a long time to cause epidemics. In northwestern Mexico, an exotic race of leaf rust detected in 2001 on durum wheat was able to cause an epidemic during the same year and the two subsequent years until the susceptible cultivar was removed from cultivation (Singh *et al.*, 2004a).

It is therefore difficult to predict when, or even if, an epidemic will occur once Ug99 is detected in a country. Since the ''Green Revolution,'' agronomic practices have changed in many parts of the world; creating a more favorable environment for stem rust buildup due to the higher use of nitrogen fertilizers and irrigation. Expansion of wheat cultivation into new areas and the expansion of conservation agriculture, allowing survival of more volunteer plants in the off-season, are all likely to change stem rust epidemiology that we know at present.

5. Breeding Strategies to Mitigate the Threat from Ug99 and Achieve a Long-Term Control of Stem Rust

Reducing the area planted to susceptible cultivars in ''Primary Risk Areas'' of East Africa, Arabian Peninsula, North Africa, Middle East, and West-South Asia is the best strategy if major losses are to be avoided.

The ''Global Rust Initiative'' (www.globalrust.org), launched in 2005, is using the following strategies to reduce the possibilities of major epidemics: (1) monitoring the spread of race Ug99 beyond eastern Africa for early warning and potential chemical interventions, (2) screening of released varieties and germplasm for resistance, (3) distributing sources of resistance worldwide for either direct use as varieties or for breeding, and (4) breeding to incorporate diverse resistance genes and adult plant resistance into high-yielding adapted varieties and new germplasm.

The best long-term strategy to mitigate the threat from Ug99 is to identify resistant sources among existing materials, or develop resistant wheat varieties that can adapt to the prevalent environments in countries under high risk, and release them after proper testing while simultaneously multiplying the seed. An aggressive strategy to promote these resistant varieties in farmers' fields is the only viable option as resource-poor as well as commercial farmers in most of Africa, Middle East, and Asia cannot afford chemical control or may not be able to apply chemicals in the event of large-scale epidemics due to their unavailability for timely application. A reduction in disease pressure in East Africa and Yemen will likely reduce chances of migration beyond these areas to other primary risk areas; however, it is unlikely that further range expansion of Ug99 can be stopped at this stage. Reduction of susceptible varieties throughout the primary risk area should reduce wind dispersal of spores from these areas to ''Secondary Risk Areas.''

For a long-term control, we like to discuss strategies that are already implemented or can be applied to identify, develop, and deploy varieties with race-specific resistance genes or with adult plant resistance.

5.1. Prevalence of *Sr24* and its breakdown

A high frequency of the highly resistant wheat materials from South America, Australia, USA, and CIMMYT identified from 2005 to 2006 screening with Ug99 in Kenya possess *Sr24* indicating it as an important resistance gene especially due to its presence in adapted genetic backgrounds. *Sr24* is located on the *Thinopyrum elongatum* translocation on chromosome 3DL together with leaf rust resistance gene *Lr24*. There are three distinct *Sr24* carrying translocations: the original one linked to a gene for red grain color, the shorter segment with white grain, and a third segment where a very small segment has been retranslocated onto chromosome 1BS. In all three segments both *Sr24* and *Lr24* are present together. Therefore, selection for *Lr24* with avirulent leaf rust isolates can be used as an indirect selection strategy.

Virulence for *Sr24* is known in South Africa (Le Roux and Rijkenberg, 1987) and India (Bhardwaj *et al.*, 1990) in local races and it arose from the deployment of this gene. Detection of race TTKST with *Sr24* virulence in

Ug99 lineage during 2006 in low frequency (Jin *et al.*, 2007a) resulted in rapid buildup to cause an epidemic on *Sr24* carrying variety Kenya Mwamba in 2007, which occupied about 30% of the Kenyan wheat area. Three *Sr24*-based resistant varieties, ''ETBW19,'' ''ETBW21,'' and ''ETBW22'' were multiplied under the emergency program in Ethiopia during the 2006 and 2007 to obtain several tons of seed. However, these varieties are now susceptible to the *Sr24*-virulent race TTKST present in Kenya indicating their genetic vulnerability if they occupy significant areas of rust-prone areas in Ethiopia in coming years.

The situations described above have once again questioned and reminded us of the consequence of dependence on single race-specific genes in the control of stem rust in areas where rust is endemic. Adoption of new varieties is a very slow process in many of the target countries and often the change of varieties is triggered by rust epidemics as happened in Ethiopia after the 1993 and 1994 stem rust epidemics on wheat variety Enkoy, which once was a leading variety but was soon replaced by other available varieties such as ''Pavon 76,'' ''Kubsa,'' and others. A similar effect was seen in Pakistan where stripe rust epidemics in northwestern Pakistan in 1995 and 1996 on varieties ''Pak 81'' and ''Pisabak 85'' led to the almost complete replacement by ''Inqualab 92,'' which was resistant to stripe rust at that time.

5.2. Race-specific resistance genes effective to Ug99

Resistance gene *Sr25* is located on a *Th. elongatum* translocation together with leaf rust resistance gene *Lr19* on chromosome 7DL. Despite the fact that this translocation is known to enhance yield potential (Singh *et al.*, 1998), it was not used widely because it is linked to a gene associated with the accumulation of undesirable levels of yellow pigment. A white floured mutant of the translocation, developed by D.R. Knott (1980), was recently transferred into some Australian and CIMMYT wheat backgrounds. *Sr25* conferred high level of resistance only in some genetic backgrounds, especially when the adult plant resistance gene *Sr2* was also present, for example, lines ''Super Seri#1'' (yellow flour), ''Wheatear'' (white flour), and several lines derived by crossing Wheatear. Virulence to *Sr25* was detected in the Nilgiri Hills of India during 2007 (M. Prashar, personal communication).

Gene *Sr26*, also of *Th. elongatum* origin, translocated to chromosome 6AL, has been used successfully in Australia and remains effective despite its large-scale deployment in the 1970s and 1980s (McIntosh, 1988). It is not known to be present in cultivars from other countries and the translocation used initially may confer a yield penalty (The *et al.*, 1988). The size of this translocation has been reduced by I. Dundas, University of Adelaide, and work is currently underway in Australia to determine if the negative effects were also removed.

Gene *Sr27* of rye origin has not been used in wheat improvement. Its deployment in triticale in Australia resulted in a rapid evolution of virulence (McIntosh *et al.*, 1983). This gene has also become ineffective in South Africa. Strategically, this gene should be left for triticale improvement in areas where virulence is not known.

Gene *Sr36*, derived from *T. timopheevi*, exhibits almost an immunity (no symptoms) to race Ug99 at both seedling and adult plant stages (Jin *et al.*, 2007b). This gene occurs in a high frequency in the US soft winter wheat (Jin and Singh, 2006) and in some Australian wheat varieties. Although races with virulence to *Sr36* are common worldwide including East Africa, this gene is effective to Ug99. Susceptible pustules were seen during 2007 in Kenya on wheat lines known to carry this gene indicating that Ug99 has evolved further, which was later confirmed.

Genes *Sr22* and *Sr35*, derived from *T. monococcum* and located on chromosomes 7AL and 3AL, respectively are also highly effective and can be backcrossed to modern wheats. Virulence to *Sr35* was identified in a laboratory culture in Australia (McIntosh *et al.*, 1995). Although race Ug99 is avirulent on gene *Sr28*, numerous races virulent to this gene are known to occur worldwide. Genes *Sr29, Sr32, Sr33, Sr37, Sr39, Sr40,* and *Sr44* have not been tested widely for their effectiveness to other races and also not used in breeding. Attempts at CIMMYT to transfer gene *Lr35* linked to *Sr39* in four spring wheat backgrounds resulted in a 15–20% reduction in grain yield potential (Singh, unpublished data). Sizes of alien chromosome segments must be reduced before *Sr32, Sr37, Sr39, Sr40,* and *Sr44* can be used successfully.

The undesignated resistance gene *SrTmp* from ''Triumph 64'' is present in some US wheat cultivars (Jin and Singh, 2006) and can be used in breeding. However, virulence to it is known in North America (Jin and Singh, 2006). An additional undesignated resistance gene, *Sr1A.1R*, located in rye chromosome translocation 1AL.1RS, is present in some US winter wheats such as ''Amigo,'' ''TAM107,'' ''TAM200,'' ''Nekota,'' ''Prairie Red,'' and other hard red winter wheat cultivars (Jin and Singh, 2006), and can also be used as it confers moderate resistance to Ug99 (Jin and Singh, 2006). Virulence to *Sr1A.1R* was detected in a sample collected in Yemen but not in the Ug99 lineage (Jin, unpublished data). Translocation carrying resistance gene *SrR*, introduced to wheat from ''Imperial'' rye in chromosomes 1BL.1RS and 1DL.1RS (Mago *et al.*, 2004) is also likely to be effective against race Ug99 as the original chromosome addition line ''TAF 2'' was found to be resistant. The allelic relationship between the stem rust resistance genes *Sr1A.1R*, *SrR,* and *Sr31* is unknown. The *Sr1A.1R* translocation is present in a few CIMMYT spring wheat lines derived from the crosses with TAM200.

Certain hexaploid synthetic (*Triticum turgidum* x *Aegilops tauschii*) wheat-derived advanced lines, some lines where certain Chinese cultivars such as

"Shanghai#7" and "Chuanmai 18" are parents, and a few more lines where resistance genes can be tracked to the US line "ND643" and a CIMMYT cross "HUW234+*Lr34*/Prinia" also have shown adequate level of resistance. Resistance in synthetic wheat-derived lines can be due to the presence of *Sr13* and *Sr14* in chromosome 6AL and 1BL, respectively, originating from durum wheat parents of synthetic wheats and *Sr33* and a newly designated resistance gene *Sr45* on chromosomes 1DL and 2DS, respectively deriving from *T. tauschii* parents. Genes *Sr13, Sr14,* and *Sr33* confer only moderate levels of resistance (Jin *et al.*, 2007b) and they will be useful in areas where stem rust pressure remains at moderate levels. Virulence to both *Sr13* and *Sr14* are known among races different from Ug99 (McIntosh *et al.*, 1995). Response of *Sr45* with Ug99 in seedling and field conditions is yet to be determined.

5.3. Strategy to use race-specific resistance genes in wheat improvement

The fastest way to reduce the susceptibility of important wheat cultivars and the best new germplasm is to systematically incorporate diverse sources of resistance through limited or repeated backcrossing. Because most of these Ug99-effective genes are of alien origin, co-segregating molecular markers for some of them are already available (Mago *et al.*, 2005; Prins *et al.*, 2001) and can aid selection. Where the alien stem rust resistance genes are linked to leaf rust resistance genes, screening for leaf rust in seedlings or adult plants can also be practiced in countries where Ug99 is absent.

To avoid fast breakdown, the best strategy is to use race-specific resistance genes in combinations. Molecular markers provide a powerful tool to identify plants that carry combinations of resistance genes. Table 3 lists available molecular markers that can be used in marker-assisted breeding. Markers for other genes need to be developed to facilitate their utilization. To transfer two or more effective resistance genes into an adapted cultivar the better crossing strategy would be to first cross the resistance sources and then cross the F_1 plants with the adapted cultivar. Molecular markers can then be used to select top-cross plants that have desirable agronomic features and carry the targeted resistance genes. Because such plants are expected to be in a low frequency, it is desirable to maintain large family size of ~400, which can be obtained by emasculating and pollinating 20 spikes. Further backcross on selected plants will help to restore the characteristics of the recurrent parent.

One major issue remains that various currently effective resistance genes are already present in some advanced spring breeding materials that are being tested in various countries to mitigate the immediate threat from Ug99. Should they not be deployed until their combinations are developed, is a difficult issue to resolve with wide range of opinions.

Table 3 PCR (polymerase chain reaction)-based markers associated to stem rust resistance genes effective to *Puccinia graminis* f. sp. *tritici* Race Ug99

Sr gene	Chromosome	Marker	Size (bp)	Marker sequence	Reference
Sr2	3BS	gwm533	120	F 5′ GTTGCTTTAGGGGAAAAGCC 3′ R 5′ AAGGCGAATCAAACGGAATA 3′	Hayden *et al.* (2004)
		stm598tcac	61	F 5′ GTTGCTTTAGGGGAAAAGCC 3′ R 5′ TCTCTCTCTCTCTCACACACAC 3′	Hayden *et al.* (2004)
		stm559tgag	85	F 5′ AAGGCGAATCAAACGGAATA 3′ R 5′ TGTGTGTGTGTGTGAGAGAGAG 3′	Hayden *et al.* (2004)
Sr22	7AL	cfa2123	245	F 5′ CGGTCTTTGTTTGCTCTAAACC 3′ R 5′ ACCGGCCATCTATGATGAAG 3′	Khan *et al.* (2005)
		cfa2019	234	F 5′ GACGAGCTAACTGCAGACCC 3′ R 5′ CTCAATCCTGATGCGGAGAT 3′	Khan *et al.* (2005)
Sr24/ Lr24	3DL/1BS	Sr24#12	500	F 5′-CACCCGTGACATGCTCGTA -3′ R 5′- AACAGGAAATGAGCAACGATGT -3′	Mago *et al.* (2005)
		Sr24#50	200	F 5′- CCCAGCATCGGTGAAAGAA -3′ R 5′- ATGCGGAGCCTTCACATTTT -3′	Mago *et al.*, 2005
		barc71	85, 103	F 5′- GCGCTTGTTCCTCACCTGCTCATA -3′ R 5′- GCGTATATTCTCTCGTCTTCTTGTTGGTT -3′	Mago *et al.* (2005)
Sr25/ Lr19	7DL	STSLr19–130	130	F 5′- CATCCTTGGGGACCTC -3′ R 5′- CCAGCTCGCATACATCCA -3′	Prins *et al.* (2001)
		wmc221	190	F 5′- ACGATAATGCAGCGGGGAAT -3′ R 5′- GCTGGGATCAAGGGATCAAT -3′	H. Bariana (personal communication)
Sr26	6AL	Sr26#43	207	F 5′- AATCGTCCACATTGGCTTCT -3′ R 5′- CGCAACAAAATCATGCACTA -3′	Mago *et al.* (2005)
Sr36	2BS	gwm271	171	F 5′ CAAGATCGTGGAGCCAGC 3′ R 5′ AGCTGCTAGCTTTTGGGACA 3′	Bariana *et al.* (2001)
		stm773	195	F 5′ ATGGTTTGTTGTGTTGTGTGTAGG 3′ R 5′ AAACGCCCCAACCACCTCTCTC 3′	Bariana *et al.* (2001)

(*continued*)

Table 3 (*continued*)

Sr gene	Chromosome	Marker	Size (bp)	Marker sequence	Reference
Sr39/ Lr35	2BS	Sr39/Lr35 (Sr39F2/ R3)	900	F 5′- AGA GAG AGT AGA AGA GCT GC -3′ R 5′- AGA GAG AGA GCA TCC ACC -3′	Gold *et al.* (1999)
Sr1A1R	1AL.1RS	R173.R (Paw S5/ Paw S6)	230, 310	F 5′ AACGAGGGGTTCGAGGCC 3′ R 5′ GAGTGTCAAACCCAACGA 3′	Mater *et al.* (2004)
SrR	1BL.1RS	IB-267	200–300	F 5′ GCAAGTAAGCAGCTTGATTTAGC 3′ R 5′ AATGGATGTCCCGGTGAGTGG 3′	Mago *et al.* (2002)
		IB-262	200–300	F 5′ GTAGGTAATGTATCAGAGTTGTAC 3′ R 5′ GTCTTTGTGCTCGGTAGCTCC 3′	Mago *et al.* (2004, 2005)

Devoting resources during the next 4–6 years to develop gene combinations knowing that these genes are already in hands of many wheat breeding groups, with no legislation to stop the release of cultivars carrying single resistance genes has provoked the CIMMYT wheat improvement group to focus their breeding effort toward breeding minor genes-based adult plant resistance, especially for areas considered to be under high risk and where survival of the pathogen for several years is expected due to the presence of susceptible hosts and favorable environmental conditions. It is thought that this strategy will allow other areas of the world, especially facultative and winter wheat growing regions to use race-specific resistance genes more successfully in their breeding program.

5.4. Adult plant resistance to Ug99 in old and new wheat

Durable stem rust resistance of some older US, Australian, and CIMMYT spring wheats is believed to be due to the deployment of *Sr2* in conjunction with other unknown minor, additive genes that could have originated from Thatcher and Thatcher-derived line Chris. *Sr2* can be detected through its complete linkage with pseudo-black chaff phenotype, which can be prominently expressed under certain environments leading to its elimination in some breeding programs. However, under the same environmental conditions negligible to high expression of pseudo-black chaff is observed in advanced breeding materials indicating that it is possible to select lines with *Sr2* with negligible pseudo-black chaff. On wheat lines that displayed pseudo-black chaff, we observed varying degrees of disease severity in Kenya ranging from traces to about 60–70% compared to 100% severity for highly susceptible materials. Reaction types varying from MR to S (moderately resistant to susceptible) on the same internodes of *Sr2* bearing plants clearly indicated that *Sr2* did confer at least some resistance.

Sr2 was detected in several highly resistant old, tall Kenyan cultivars, including ''Kenya Plume'' (Singh and McIntosh, 1986), and CIMMYT-derived semidwarf wheats ''Pavon 76,'' ''Parula,'' ''Kritati,'' and ''Kingbird.'' Pavon 76 and Kiritati were resistant since the initiation of rigorous screening in 2005 at Njoro, Kenya with maximum disease scores of 20MR-MS. Kingbird, a new advanced line, is at present the best known source of adult plant resistance in semidwarf wheat with maximum score recorded to be 5 MR-MS during the same period. Because these wheats are susceptible as seedlings with race Ug99, their resistance is speculated to be based on multiple additive genes where *Sr2* is an important component.

With the exception of *Sr2*, little is known on the genes involved in durable adult plant resistance; however, earlier work done by Knott (1982), knowledge on durable resistance to leaf and yellow rusts (Singh *et al.*, 2004b), and observations made on breeding materials and a F_6 mapping population involving Pavon 76 all indicate that the rate of rust progress is a

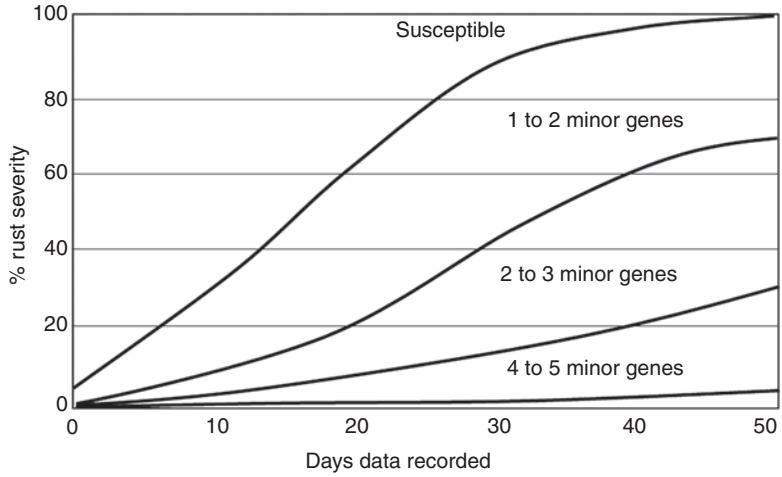

Figure 5 **Graphical representation of the additive effects from estimated number of minor genes in retarding rust progress in the field.**

function of the cumulative effect of the number of minor genes present in a genotype and individual effects of each gene (Fig. 5). Accumulation of between 4 and 5 genes is therefore expected to retard disease progress to rates that result in negligible disease levels at maturity under high disease pressure, described as ''near-immunity'' by Singh *et al.* (2000).

Accumulating such complex resistance will be cumbersome but not impossible in the absence of disease pressure caused by race Ug99 at most breeding sites and lack of molecular markers associated with genes contributing to resistance. Molecular markers linked to the slow rusting resistance gene *Sr2* are known and can be used in selection; however, this gene can also be identified in the field under most environments from its linkage with pseudo-black chaff phenotype. *Sr2* is present in about 60% of the current CIMMYT spring wheat germplasm including some of the most recent high-yielding wheats that have high level of resistance to leaf and stripe rusts and desirable end-use quality characteristics.

5.5. High-yielding wheat lines with adult plant resistance to stem rust

Stem rust screening of CIMMYT advanced breeding materials in Kenya since 2005 has resulted in the identification of 15–20% lines that carried adequate level of resistance under heavy disease pressure. Table 4 summarizes the information on resistance to stem rust in resistant spring wheat germplasm distributed worldwide by CIMMYT during 2006 and 2007, through the newly initiated 1st and 2nd Stem Rust Resistance Screening Nurseries (1st and 2nd SRRSN), and those currently under multiplication

Table 4 Stem rust resistance of entries included in 1st and 2nd SRRSN (Stem Rust Resistance Screening Nursery), and candidate entries under multiplication for inclusion in 3rd SRRSN

Resistance	1st SRRSN		2nd SRRSN		3rd SRRSN candidates	
Category	Number	%	Number	%	Number	%
Adult plant[a]						
R (5–10% severity)	4	4	0	0	6	4
R-MR (15–20% severity)	19	18	26	20	42	28
MR (30% severity)	6	6	22	17	36	24
MR-MS (40% severity)	2	2	15	12	0	0
MS (50–60% severity)	0	0	17	13	0	0
S (70–100% severity)	0	0	4	3	0	0
Race specific						
Sr24	39	38	0	0	0	0
Sr25	17	17	0	0	18	12
Sr36 (+*Sr24*)	0	0	0	0	5	3.3
Sr1A.1R (+*Sr24*)	2	2	0	0	0	0
SrTmp	0	0	25	20	11	7.3
SrSynt	4	4	8	6	6	4
SrSha7	9	9	8	6	8	5.3
SrND643	0	0	0	0	12	8
SrUnknown	1	1	3	2	6	4
Total	103		128		150	

[a] Adult plant resistance categories include lines that are susceptible in seedling greenhouse tests and with highest rating recorded during multiple years/seasons testing when the susceptible entries annihilated following 100% stem rust severity based on the modified Cobb Scale (Peterson *et al.*, 1948).

to form the 3rd SRRSN for distribution in 2008. A total of 29 (28%), 48 (37%), and 84 (56%) lines in these three nurseries, respectively have shown from high to moderate levels (up to 30% stem rust severity when the susceptible materials show annihilation following 100% severity) of resistance in at least two seasons of evaluation under high disease pressure in Kenya. Entries included in the 2nd and 3rd SRRSN have high yield potential in combination with various other desirable traits. These improved wheat materials have the potential to be released directly or be used by breeding programs worldwide.

Frequency of high-yielding spring wheat materials with stem rust resistance for distribution through other international yield and screening nurseries, such as Elite Spring Wheat Yield Trial (ESWYT) and International Bread Wheat Screening Nursery (IBWSN), is also rising (Table 5). Of the 190 entries included in 29th ESWYT and 41st IBWSN being prepared for growing in 2008–2009 crop seasons, 50 (26%) lines have adequate adult plant resistance. A total of 111 lines that have shown adequate stem rust resistance are being evaluated further for yield performance in Mexico and seed is being multiplied simultaneously during the 2007–2008 crop season to form the 30th ESWYT and 42nd IBWSN. Frequency of entries with race-specific resistance genes is much lower than those with adult plant resistance (Table 5).

5.6. Mexico-Kenya shuttle to breed high-yielding spring wheat with near-immune level of adult plant resistance

Because a large portion of CIMMYT high-yielding spring wheat germplasm does not carry effective race-specific stem rust resistance genes to Ug99 and several lines were identified to carry at least moderate levels of resistance, this

Table 5 Stem rust resistance of entries included in 29th ESWYT and 41st IBWSN, and entries being multiplied for possible inclusion in 30th ESWYT and 42nd IBWSN

Resistance	29th ESWYT and 41st IBWSN		Multiplied 30th ESWYT and 42nd IBWSN	
Category	Number	%	Number	%
Adult plant[a]				
R (5–10% severity)	0	0.0	9	2.4
R-MR (15–20% severity)	13	6.8	38	10.3
MR (30% severity)	37	19.5	64	17.4
MR-MS (40% severity)	14	7.4	72	19.6
MS (50–60% severity)	65	34.2	83	22.6
S (100% severity)	44	23.2	59	16.0
Race specific				
Sr25	1	0.5	26	7.1
SrTmp	10	5.3	6	1.6
SrSynt	3	1.6	2	0.5
SrSha7	1	0.5	0	0.0
SrND643	0	0.0	3	0.8
SrUnknown	2	1.1	6	1.6
Total	190		368	

[a] Adult plant resistance categories include lines that are susceptible in seedling greenhouse tests and with highest rating recorded during multiple years/seasons testing when the susceptible entries annihilated following 100% stem rust severity based on the modified Cobb Scale (Peterson *et al.*, 1948).

was viewed as a perfect opportunity to reconstitute high levels of adult plant resistance in newer wheat materials. In the absence of molecular markers for adult plant resistance genes and the absence of Ug99 race in Mexico, a shuttle breeding scheme between two Mexican sites and Njoro, Kenya was initiated in 2006 to transfer adult plant resistance identified in semidwarf CIMMYT wheats to a range of important wheat germplasm. Two crop seasons per year in Mexico and Kenya will accelerate the breeding. The ''single-backcross, selected-bulk'' breeding approach (Singh *et al.*, 2004b) is being applied for transferring multiple minor genes to adapted backgrounds. Simple and three-way crosses, where one or more parents carry adult plant resistance, are being used to breed new high-yielding, near-immune wheat materials.

In the single-backcross approach, we crossed resistance sources with the adapted high-yielding wheats and then a single backcross was made with the recurrent parent to obtain about 400 BC_1 seeds. BC_1 plants were then selected for desired agronomic features and resistance to leaf and stripe rusts, and harvested as bulk. F_2 plants derived from BC_1, simple and three-way crosses with desired agronomic features and resistance to leaf and stripe rusts were selected for agronomic traits and resistance to other diseases at CIMMYT research stations in Ciudad Obregon in northwestern Mexico or Toluca in the highlands near Mexico City and harvested as bulk. If F_2 populations were grown in Ciudad Obregon, where quarantine disease ''Karnal bunt'' is known to occur, the F_3 populations are grown at Toluca for another round of selection. About 1000 seeds of each of the F_3 and F_4 populations obtained from harvesting materials at Toluca were grown densely in Njoro, Kenya for selection under high stem rust pressure during the off-season. After removing tall plants, the remaining populations were bulk harvested and about thousand plump grains selected to grow F_4 and F_5 populations during the main season in Kenya under high disease pressure. Because stem rust affects grain filling, we expect that plants with insufficient resistance will have shriveled grains (Fig. 6). About 400 plump seeds harvested from the selected plants were sent back to Mexico for final selection as individual plants in the F_5 and F_6 generations at Ciudad Obregon. Individual plant selections will also be made in Kenya. This is the current status as of the 2007–2008 crop season. Selected plants in Ciudad Obregon with good characteristics will be grown as small plots in Toluca and El Batan field sites in Mexico and selected lines will be grown in Kenya for stem rust screening. Selected plants in Kenya with good grain characteristics will be grown in F_6 as hill plots or short rows in Kenya as well as small plots in Mexico for final selection. Finally, the resistant F_6 plots will be harvested for conducting yield trials in the following crop season in Ciudad Obregon and simultaneously evaluated for stem rust resistance in Kenya. The single-backcross, selected-bulk scheme is also being applied to transfer resistance from old, tall Kenyan cultivars into adapted semidwarf wheats. A shuttle breeding scheme

Figure 6 Effect of stem rust pathogen race Ug99 on wheat spikes and grains of a susceptible (left) and a resistant (right) variety in Njoro, Kenya during 2007.

was also implemented in 2006 between Aleppo in Syria and Kulumsa, or Melkasa, in Ethiopia by the bread wheat improvement program of ICARDA (International Center for Agricultural Research in the Dry Areas).

We expect that the frequency of advanced lines which carry high yield potential, maintain wide adaptation, end-use quality characteristics, and high level of resistance to all three rusts will increase over time through the use of the Mexico–Kenya shuttle. Moreover, the proposed approach is expected to rebuild the durable resistance in modern wheat germplasm. Genetic analyses are underway to understand the number and type of resistance genes involved in sources contributing the slow rusting, adult plant resistance. Genomic locations of minor, additive resistance genes, determined through molecular mapping, is expected to not only result in molecular markers for some of the slow-rusting genes but also will be useful to establish and enhance genetic diversity for such genes in the global spring wheat germplasm and will allow their incorporation in facultative and winter wheat materials.

5.7. Reducing the world's wheat area under susceptible cultivars

Potential epidemics following the spread of Ug99 or its variants can be avoided if current susceptible cultivars occupying most of the wheat areas in the primary risk areas in the predicted migration path are reduced. Screening in Kenya during 2005, 2006, and 2007 has identified a few resistant released varieties or advanced breeding materials at various stages of testing in most of the countries that submitted their materials for screening.

One strategy is to find ways to ensure that the best, high-yielding resistant materials occupy at least 5% of total wheat area distributed throughout the wheat region and are readily available. This might be via seed supply through procurement in the case that Ug99 establishment is evident in a particular country. However, it will be very difficult to promote resistant varieties on a large scale if they are inferior to the current popular varieties or because farmers have not seen stem rust. Moreover, growing inferior, resistant varieties is not an option as it will affect wheat production at a time when global wheat supply is at its lowest level causing sharp increases in wheat prices. Wheat production must continue to increase at the rate of 2% annually to meet predicted demand of about 800 million tons by 2020 from a current production of about 600 million tons (Fig. 7).

Identification and promotion of new stem rust resistant varieties that have significantly enhanced yield potential than current varieties, in conjunction with other desirable traits is probably the best strategy to ensure their fast adoption and thus to succeed in replacing the existing popular but susceptible varieties. This is an achievable objective as most of the current popular varieties were developed during early-to-mid-1990s and yield potential of current CIMMYT spring wheat germplasm has progressed significantly since then (Singh *et al.*, 2007).

Yield performances of 14 new high yielding, Ug99 resistant wheat lines together with local check and 15 additional high-yielding entries were determined in replicated yield trials, 2nd Elite Bread Wheat Yield Trial (2nd EBWYT), planted during the 2006–2007 crop season at 27 sites in India, Pakistan, Nepal, Afghanistan, Iran, Egypt, Sudan, Syria, and Mexico. Results from seven sites in India for 10 lines derived from different crosses with high yield potential and adequate resistance for their successful deployment in the northwestern Gangetic Plains, the wheat basket of India, are given in Table 6. The best performing entry Waxwing*2/Kiritati,

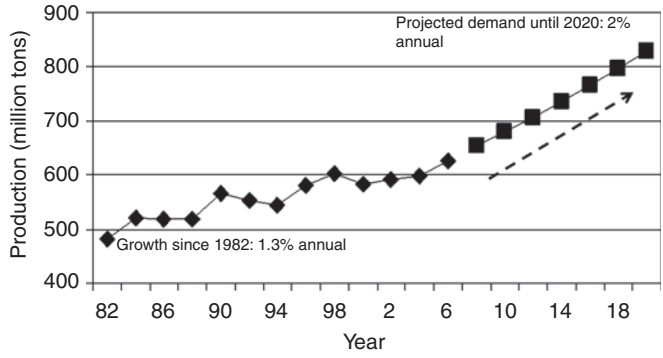

Figure 7 Historical and projected future wheat production requirements to meet the demand by year 2020.

Table 6 Mean grain yield performance for entries included in the 2nd EBWYT grown at seven sites in northwestern India during 2006–2007 crop season and stem rust response evaluated on three dates at Njoro, Kenya during 2007

		Mean grain yield			Stem rust response[a]			Resistance
Entry no.	Cross	kg ha^{-1}	Rank	% Check	25/09/2007	1/10/2007	11/10/2007	Gene/type
501	Local check	4472.2	27	100.0	–	–		–
510	Weebill_1*2/Kiritati	4688.6	19	104.8	5MR-MS	20MS-S	30MS-S	APR
514	Oasis/SKauz//4*Bcn/3/2*Pastor	5055.9	4	113.1	5RMR	10RMR	10MR	*Sr25*
516	Pfau/Seri.1B//Amadina/3/Waxwing	5074.4	3	113.5	20MR-MS	40MR-MS	40MR-MS	APR
517	Weebill_1*2/Brambling	4590.8	23	102.7	5MR-MS	15MR-MS	40MR-MS	APR
518	Munia/Chto/3/Pfau/Bow//Vee#9/4/Chen/Ae. Sq.//Bcn/5/Babax/Lr42//Babax	4953.3	8	110.8	10MS-S	20MR-MS	20MR-MS	APR + (*Sr24*)

519	Babax/LR42// Babax*2/3/Vivitsi	5208.2	2	116.5	15R-MR	30MR-MS	40MR-MS	*SrTmp*
521	Waxwing*2/Kiritati	5222.4	1	116.8	5MR-MS	20MS-S	30MS-S	APR
527	Hpo/Tan//Vee/3/ 2*Pgo/4/Milan/ 5/SSeri1	4737	18	105.9	5MS	10MS	20MS-S	APR
528	Pfau/Weaver*2// Kiritati	4596.6	22	102.8	1MS-S	5MS-S	15MR-MS	APR
530	SKauz/Bav92	4605.9	21	103.0	5RMR	10RMR	15RMR	*Sr25*
	LSD, $P = 0.05$	*410.1*						
	CV (%)	*11.1*						

[a] Stem rust response has two components: severity based on modified Cobb Scale (Peterson *et al*., 1948) and host reaction as described in Roelfs *et al*. (1992). Host reactions are R, resistant; MR, moderately resistant; MS, moderately susceptible; and S, susceptible.

numbered 521 in the trial, had16.8% mean grain yield advantage over the local check cultivars PBW502 or mega-cultivar PBW343. PBW502 is a stripe rust resistant descendent of PBW343 and has begun replacing PBW343. Because the adult plant resistance of this entry involves the ''*Sr2*-complex'' derived from ''Kiritati'' parent, and it also has excellent end-use quality characteristics and adult plant resistance to leaf and stripe rusts (data not presented), it could be an excellent option as a potential replacement cultivar. The 2nd and 4th best performing entries numbered 519 and 514 with grain yield advantages of 16.5 and 13.1% over the check have resistance based on race-specific resistance genes *SrTmp* and *Sr25*, respectively. Similar results were also seen in other countries where trials were grown indicating that replacement of current cultivars with new Ug99 resistant materials should also result in enhancing productivity even in the absence of stem rust. Yield performances of 27 additional high yielding, stem rust resistant materials are being evaluated as 3rd EBWYT at 50 sites in many more countries in the primary risk area during the 2007–2008 crop season.

5.8. Efforts to identify and develop resistant wheat varieties in secondary risk areas

Identifying existing varieties or advanced breeding materials through screening in Kenya or Ethiopia is the top priority at present for developing and developed countries where spring, facultative and winter wheats are grown without the use of fungicides. Developed countries that routinely test their materials for stem rust in Kenya or Ethiopia are the USA, Canada, and Australia. Characterization of a small number of spring wheat materials from Switzerland and Sweden in Kenya showed that they were moderately to highly susceptible. Resistance in materials from other European countries is yet to be characterized; however, intense use of modern broad spectrum fungicides in most of these countries to control other important diseases will also likely control stem rust.

Screening of US materials in Kenya are coordinated by the USDA-ARS. Resistance is also identified in important materials through seedling tests with Ug99 (TTKSK) and its variant race TTKST with *Sr24* virulence at the USDA-ARS Cereal Disease Laboratory, St. Paul, MN during winter months in quarantine greenhouses. A similar effort is also underway at the Cereal Research Center, Winnipeg, Canada. The resistance status of US materials was described by Jin and Singh (2006). Most breeding programs in the USA and Canada are using this information and diverse sources of resistance in their crossing program.

Since the creation of the ''Australian Cereal Rust Control Program'' in 1973 following the major stem rust epidemic, it is a routine exercise to incorporate new resistance genes in Australian varieties and breeding materials and determine their agronomic suitability (The *et al.*, 1988). This effort

has enhanced genetic diversity for resistance in Australian wheat germplasm and therefore new varieties are likely to be identified to replace those susceptible to Ug99.

Resistance to Ug99 in spring, facultative and winter wheats from China, Russia, Turkey, Iran, and some countries from Central Asia is also limited. Breeding efforts have been initiated to incorporate diverse race-specific resistance genes in important cultivars and promising advanced wheat breeding materials through marker-assisted backcrossing. A simultaneous effort to introduce adult plant resistance is also planned to be undertaken in China through close collaboration between the ''CIMMYT-CAAS Facultative and Winter Wheat Breeding Program'' and breeding programs of various other academies, and the ''Turkey-CIMMYT-ICARDA Winter and Facultative Wheat Breeding Program'' based in Turkey to target winter and facultative wheats grown in Turkey, Iran, Afghanistan, and other countries of Central Asia.

Spring wheat germplasm developed by CIMMYT in Mexico are well adapted in most of Central and South America and several South American breeding programs heavily use CIMMYT materials in their breeding program. This will allow identification of adapted stem rust resistant materials for deployment in the near future.

6. Conclusion and Future Outlook

Considering the progress made in identifying stem rust resistance in existing varieties and advanced breeding materials, though in low frequency, and high priority to incorporate diverse or durable adult plant resistance by various wheat breeding programs including those of CIMMYT and ICARDA, it is unlikely that stem rust race Ug99 or its descendents will destroy the world wheat crop. However, localized epidemics as observed in Kenya during 2007 cannot be ruled out. Migration of Ug99 is being monitored carefully through field surveys, monitoring nurseries and GIS tools to provide an early warning, which could allow chemical interventions if necessary and guide decision making. Reducing the area currently occupied by susceptible varieties in the primary risk areas of Africa, Arabian Peninsula, Middle East, and West-South Asia with resistant ones should become an immediate priority. It is highly advisable to release in these areas varieties that have durable, adult plant resistance, or have effective race-specific resistance genes in combinations, to prevent further evolution and selection of new virulences leading to ''Boom-and-Bust'' cycles. This will also allow reduction of inoculum in high-risk areas and thus reduce risks of its spread to secondary risk areas.

The emergence of Ug99 race of stem rust pathogen as a global threat to wheat production has highlighted the need and benefits of public-funded

wheat research and improvement where germplasm carrying useful traits and information can be readily shared to ensure a sustainable production of staple food crops at low cost and with the least negative environmental impacts. Response to the alarm raised by Dr. N. E. Borlaug of the global threat from Ug99 has been positive and effective from wheat scientists, research leaders, and importantly the donor community who came forward to support the global effort to mitigate the threat. However, to succeed much needs to be done, especially to ensure that seed of high yielding, stem rust resistant materials is made available to as many farmers as possible to ensure that their wheat crop is not destroyed by stem rust.

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REFERENCES

Aquino, P., Carrion, F., and Calvo, R. (2002). Selected wheat statistics. *In* "CIMMYT 2000–2001 World Wheat Overview and Outlook: Developing No-Till Packages for Small Scale Farmers" (J. Ekboir, ed.), pp. 52–62. CIMMYT, Mexico, D.F.

Bariana, H. S., Hayden, M. J., Ahmed, N. U., Bell, J. A., Sharp, P. J., and McIntosh, R. A. (2001). Mapping of durable adult plant and seedling resistances to stripe rust and stem rust diseases in wheat. *Aust. J. Agric. Res.* **52**, 1247–1255.

Bhardwaj, S. C., Nayar, S. K., Prashar, M., Kumar, J., Menon, M. K., and Singh, S. B. (1990). A pathotype of *Puccinia graminis* f. sp. *tritici* on *Sr24* in India. *Cereal Rusts Powdery Mildews Bull* **18**, 35–37.

Biffen, R. H. (1905). Mendel's laws of inheritance and wheat breeding. *J. Agric. Sci.* **1**, 4–48.

Brown, J. K. M., and Hovmøller, M. S. (2002). Aerial dispersal of pathogens on the global and continental scales and its impact on plant disease. *Science* **297**, 537–541.

CIMMYT (2005). "Sounding the Alarm on Global Stem Rust." http://www.globalrust.org/uploads/documents/SoundingAlarmGlobalRust.pdf (accessed on November 29, 2007). CIMMYT, Mexico, D.F.

Draxler, R. R., and Rolph, G. D. (2003). "HYSPLIT (HYbrid Single-Particle Lagrangian Integrated Trajectory)." http://www.arl.noaa.gov/ready/hysplit4.html (accessed on November 29, 2007). NOAA Air Resources Laboratory, Silver, Spring, MD.

FAO (Food and Agriculture Organization of the United Nations). (2001). "Food Outlook." FAO, Rome, Italy.

Gold, J., Harder, D., Townley-Smith, F., Aung, T., and Procunier, J. (1999). Development of a molecular marker for rust resistance gene *Sr39* and *Lr35* in wheat breeding lines. *Electron. J. Biotechnol.* **2**, 35–40.

Hayden, M. J., Kuchel, H., and Chalmers, K. J. (2004). Sequence tagged microsatellites for the Xgwm533 locus provide new diagnostic markers to select for the presence of stem rust resistance genes *Sr2* in bread wheat (*Triticum aestivum* L.). *Theor. Appl. Genet.* **109**, 1641–1647.

Hodson, D. P., Singh, R. P., and Dixon, J. M. (2005). An initial assessment of the potential impact of stem rust (race Ug99) on wheat producing regions of Africa and Asia using GIS. *In* "Abstracts. 7th International Wheat Conference", p. 142. November 27–December 2, 2005, Mar del Plata, Argentina.

Huerta-Espino, J. (1992). "Analysis of Wheat Leaf and Stem Rust Virulence on a Worldwide Basis." Ph.D. thesis, University of Minnesota, USA.

Hugh-Jones, M. E. (2002). Agricultural bioterrorism. *In* "High-Impact Terrorism: Proceedings of a Russian-American Workshop," pp. 219–232. National Academy Press, Washington, DC.

Jin, Y. (2007). Resistance to race TTKS of *Puccinia graminis* f. sp. *tritici* in Chris and related spring wheat. *Phytopathology* **97**, S162(Abstract).

Jin, Y., and Singh, R. P. (2006). Resistance in US wheat to recent eastern African isolates of *Puccinia graminis* f. sp. *tritici* with virulence to resistance gene *Sr31*. *Plant Dis.* **90**, 476–480.

Jin, Y., Pretorius, Z. A., and Singh, R. P. (2007a). New virulence within race TTKS (Ug99) of the stem rust pathogen and effective resistance genes. *Phytopathology* **97**, S137(Abstract).

Jin, Y., Singh, R. P., Ward, R. W., Wanyera, R., Kinyua, M., Njau, P., Fetch, T., Pretorius, Z. A., and Yahyaoui, A. (2007b). Characterization of seedling infection types and adult plant infection responses of monogenic *Sr* gene lines to race TTKS of *Puccinia graminis* f. sp. *tritici*. *Plant Dis.* **91**, 1096–1099.

Jin, Y., Pretorius, Z. A., Singh, R. P., and Fetch, T., Jr (2008). Detection of virulence to resistance gene *Sr24* within race TTKS of *Puccinia graminis* f. sp. *tritici*. *Plant Dis.* **92**: In press.

Khan, R. R., Bariana, H. S., Dholakia, B. B., Naik, S. V., Lagu, M. D., Rathjen, A. J., Bhavani, S., and Gupta, V. S. (2005). Molecular mapping of stem and leaf rust resistance in wheat. *Theor. Appl. Genet.* **111**, 846–850.

Knott, D. R. (1980). Mutation of a gene for yellow pigment linked to *Lr19* in wheat. *Can. J. Genet. Cytol.* **22**, 651–654.

Knott, D. R. (1982). Multigenic inheritance of stem rust resistance in wheat. *Crop Sci.* **22**, 393–399.

Knott, D. R. (1988). Using polygenic resistance to breed for stem rust resistance in wheat. *In* "Breeding Strategies for Resistance to the Rusts of Wheat" (N. W. Simmonds and S. Rajaram, eds.), pp. 39–47. CIMMYT, Mexico, D.F.

Kolmer, J. A. (2001). Early research on the genetics of *Puccinia graminis* stem rust resistance in wheat in Canada and the United States. *In* "Stem Rust of Wheat: From Ancient Enemy to Modern Foe" (P. D. Peterson, ed.), pp. 51–82. APS Press, St. Paul, MN.

Leonard, K. J. (2001). Stem rust—Future enemy? *In* "Stem Rust of Wheat: From Ancient Enemy to Modern Foe" (P. D. Peterson, ed.), pp. 119–146. APS Press, St. Paul, MN.

Le Roux, J., and Rijkenberg, F. H. J. (1987). Pathotypes of *Puccinia graminis* f. sp. *tritici* with increased virulence for *Sr24*. *Plant Dis.* **71**, 1115–1119.

Luig, N. H. (1985). Epidemiology in Australia and New Zealand. *In* "Cereal Rusts, Vol. II: Diseases, Distribution, Epidemiology, and Control" (A. P. Roelfs and W. R. Bushnell, eds.), pp. 301–328. Academic Press, Orlando.

Mago, R., Spielmeyer, W., Lawrence, G. J., Lagudah, E. S., Ellis, J. G., and Pryor, A. (2002). Identification and mapping of molecular markers linked to rust resistance genes located on chromosome 1RS of rye using wheat-rye translocation lines. *Theor. Appl. Genet.* **104**, 1317–1324.

Mago, R., Spielmeyer, W., Lawrence, G. J., Ellis, J. G., and Prior, A. J. (2004). Resistance genes for rye stem rust (*SrR*) and barley powdery mildew (*Mla*) are located in syntenic regions on short arm of chromosome. *Genome* **47**, 112–121.

Mago, R., Bariana, H. S., Dundas, I. A., Spielmeyer, W., Lawrence, G. J., Pryor, A. J., and Ellis, J. G. (2005). Development of PCR markers for the selection of wheat stem rust resistance genes *Sr24* and *Sr26* in diverse wheat germplasm. *Theor. Appl. Genet.* **111**, 496–504.

Mater, Y., Baenzinger, S., Gill, K., Graybosch, R., Whitcher, L., Baker, C., Specht, J., and Dweikat, I. (2004). Linkage mapping of powdery mildew and greenbug resistance genes on recombinant 1RS from 'Amigo' and 'Kaukaz' wheat-rye translocations of chromosome 1RS.1AL. *Genome* **47**, 292–298.

McIntosh, R. A. (1988). The role of specific genes in breeding for durable stem rust resistance in wheat and triticale. *In* "Breeding Strategies for Resistance to the Rust of Wheat" (N. W. Simmonds and S. Rajaram, eds.), pp. 1–9. CIMMYT, Mexico, D.F.

McIntosh, R. A., Luig, N. H., Milne, D. L., and Cusick, J. (1983). Vulnerability of triticales to wheat stem rust. *J. Plant Pathol.* **5**, 61–69.

McIntosh, R. A., Wellings, C. R., and Park, R. F. (1995). "Wheat Rusts: An Atlas of Resistance Genes." CSIRO Publications, Victoria, Australia.

Nagarajan, S., and Joshi, L. M. (1985). Epidemiology in the Indian subcontinent. *In* "The Cereal Rusts, Vol. II: Diseases, Distribution, Epidemiology, and Control" (A. P. Roelfs and W. R. Bushnell, eds.), pp. 371–402. Academic Press, Orlando.

Peterson, R. F., Campbell, A. B., and Hannah, A. E. (1948). A diagrammatic scale for estimating rust intensity of leaves and stem of cereals. *Can. J. Res. Sect. C* **26**, 496–500.

Pretorius, Z. A., Singh, R. P., Wagoire, W. W., and Payne, T. S. (2000). Detection of virulence to wheat stem rust resistance gene *Sr31* in *Puccinia graminis* f. sp. *tritici* in Uganda. *Plant Dis.* **84**, 203.

Prins, R., Groenewald, J. Z., Marias, G. F., Snape, J. W., and Koebner, R. M. D. (2001). AFLP and STS tagging of *Lr19*, a gene conferring resistance to leaf rust in wheat. *Theor. Appl. Genet.* **103**, 618–624.

Rajaram, S., Singh, R. P., and Torres, E. (1988). *In* "Current CIMMYT Approaches in Breeding Wheat for Rust Resistance. Breeding Strategies for Resistance to the Rust of Wheat" (N. W. Simmonds and S. Rajaram, eds.), pp. 101–118. CIMMYT, Mexico, D.F.

Reynolds, M. P., and Borlaug, N. E. (2006). Applying innovations and new technologies from international collaborative wheat improvement. *J. Agric. Sci.* **144**, 95–110.

Roelfs, A. P. (1985). Wheat and rye stem rust. *In* "The Cereal Rusts, Vol. II: Diseases, Distribution, Epidemiology, and Control" (A. P. Roelfs and W. R. Bushnell, eds.), pp. 3–37. Academic Press, Orlando.

Roelfs, A. P., and Martell, L. B. (1984). Uredospore dispersal from a point source within a wheat canopy. *Phytopathology* **74**, 1262–1267.

Roelfs, A. P., and Martens, J. W. (1988). An international system of nomenclature for *Puccinia graminis* f. sp. *tritici*. *Phytopathology* **78**, 526–533.

Roelfs, A. P., Singh, R. P., and Saari, E. E. (1992). "Rust Diseases of Wheat: Concepts and Methods of Disease Management." CIMMYT, Mexico, D.F.

Rotem, J., Wooding, B., and Aylor, D. E. (1985). The role of solar radiation, especially UV, in the mortality of fungal spores. *Phytopathology* **75**, 510–514.

Rowell, J. B., and Romig, R. W. (1966). Detection of urediospores of wheat rusts in spring rains. *Phytopathology* **56**, 807–811.

Saari, E. E., and Prescott, J. M. (1985). World distribution in relation to economic losses. *In* "The Cereal Rusts, Vol. II: Diseases, Distribution, Epidemiology, and Control" (A. P. Roelfs and W. R. Bushnell, eds.), pp. 259–298. Academic Press, Orlando.

Shank, R. (1994). Wheat stem rust and drought effects on Bale agricultural production and future prospects. Report on February 17–28 assessment. *In* "United Nations Emergencies Unit for Ethiopia." http://www.africa.upenn.edu/eue_web/Bale_mar.txtaccessed on November 29, 2007. Addis Ababa, Ethiopia.

Singh, R. P. (1991). Pathogenicity variations of *Puccinia recondita* f. sp. *tritici* and *P. graminis* f. sp. *tritici* in wheat-growing areas of Mexico during 1988 and 1989. *Plant Dis.* **75**, 790–794.

Singh, R. P., and McIntosh, R. A. (1986). Genetics of resistance to *Puccinia graminis tritici* and *Puccinia recondita tritici* in Kenya plume wheat. *Euphytica* **35**, 245–256.

Singh, R. P., and McIntosh, R. A. (1987). Genetics of resistance to *Puccinia graminis tritici* in 'Chris' and 'W3746' wheats. *Theor. Appl. Genet.* **73**, 846–855.

Singh, R. P., Huerta-Espino, J., Rajaram, S., and Crossa, J. (1998). Agronomic effects from chromosome translocations 7DL.7Ag and 1BL.1RS in spring wheat. *Crop Sci.* **38**, 27–33.

Singh, R. P., Huerta-Espino, J., and Rajaram, S. (2000). Achieving near-immunity to leaf and stripe rusts in wheat by combining slow rusting resistance genes. *Acta Phytopathlogica Hungarica* **35**, 133–139.

Singh, R. P., Huerta-Espino, J., Pfeiffer, W., and Figueroa-Lopez, P. (2004a). Occurrence and impact of a new leaf rust race on durum wheat in the northwestern Mexico from 2001–2003. *Plant Dis.* **88**, 703–708.

Singh, R. P., William, H. M., Huerta-Espino, J., and Rosewarne, G. (2004b). Wheat rust in Asia: Meeting the challenges with old and new technologies. *In* "New Directions for a Diverse Planet: Proceedings of the 4th International Crop Science Congress," http://www.cropscience.org.au./icsc2004/symposia/3/7/141_singhrp.htm (accessed on November 29, 2007). September 26–October 1, 2004. Brisbane, Australia.

Singh, R. P., Hodson, D. P., Jin, Y., Huerta-Espino, J., Kinyua, M., Wanyera, R., Njau, P., and Ward, R. W. (2006). Current status, likely migration and strategies to mitigate the threat to wheat production from race Ug99 (TTKS) of stem rust pathogen. *CAB Reviews: Perspectives in Agriculture, Veterinary Science, Nutrition and Natural Resources* **1**, 54.

Singh, R. P., Huerta-Espino, J., Sharma, R., Joshi, A. K., and Trethowan, R. M. (2007). High yielding spring bread wheat germplasm for global irrigated agro-ecosystems. *Euphytica* **157**, 351–363.

Stakman, E. C. (1957). Problems in preventing plant disease epidemics. *Am. J. Bot.* **44**, 259–267.

Stakman, E. C., and Piemeisel, F. J. (1917). A new strain of *Puccinia graminis*. *Phytopathology* **7**, 73.

Steele, K. A., Humphreys, E., Wellings, C. R., and Dickinson, M. J. (2001). Support for a stepwise mutation model for pathogen evolution in Australasian *Puccinia striiformis* f. sp. *tritici* by use of molecular markers. *Plant Pathol.* **50**, 174–180.

Sunderwirth, S. D., and Roelfs, A. P. (1980). Greenhouse characterization of the adult plant resistance of *Sr2* to wheat stem rust. *Phytopathology* **70**, 634–637.

The, T. T., Latter, B. D. H., McIntosh, R. A., Ellison, F. W., Brennan, P. S., Fischer, J. A., Hollamby, G. J., Rathgen, A. J., and Wilson, R. E. (1988). Grain yield of near isogenic lines with added genes for stem rust resistance. *In* "Proceedings of the 7th International Wheat Genetics Symposium" (T. S. Miller and R. M. D. Koebner, eds.), pp. 901–906. Institute of Plant Science Research, Cambridge, UK.

Vanderplank, J. E. (1963). "Plant Diseases: Epidemics and Control." Academic Press, New York and London.

Wanyera, R., Kinyua, M. G., Jin, Y., and Singh, R. P. (2006). The spread of stem rust caused by *Puccinia graminis* f. sp. *tritici*, with virulence on *Sr31* in wheat in Eastern Africa. *Plant Dis.* **90**, 113.