Institut für Agrar- und Ernährungswissenschaften
der Naturwissenschaftlichen Fakultät III
der
Martin-Luther-Universität Halle-Wittenberg

Analysis of pathogen virulence and cultivar resistance to yellow rust, *Puccinia striiformis* f. sp. *tritici*, in Ethiopia

Dissertation

zur Erlangung des akademischen Grades
doctor agriculturarum (Dr. agr.)

vorgelegt von

Diplomagraringenieurin Woubit Dawit Bedane
geb. am 26.05.1974 in Äthiopien

Gutachter: Prof. Dr. W. E. Weber

Prof. Dr. Thomas Miedaner

Priv.-Doz. Dr. Andreas Börner

Verteidigung am: 26.09.2008

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Aus dem Institut für Agrar- und Ernährungswissenschaften
(Geschäftsführender Direktor: Prof. Dr. Klaus Eder)

der Naturwissenschaftlichen Fakultät III
(Dekan: Prof. Dr. Peter Wycisk)

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Dedication

To my beloved parents (Mr. Dawit and Mrs. Tiwab)

And

Husband Dr. Selamyihun Kidanu

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LIST OF ABBREVIATIONS

CIMMYT: International Maize and Wheat Improvement Center

cM: Centi Morgan

CSA: Central Statistical Authority

dNTP: Deoxyribonucleic triphosphate

dUTP: Deoxyuridine triphosphate

IEAR: Institute of Ethiopian Agricultural Research

FDRE: Federal Democratic Republic of Ethiopia

HAR: Holleta Agricultural Research

IAR: Institute of Agricultural Research

KARC: Kulumsa Agricultural Research Center

PCR: Polymerase chain reaction

SSR: Simple sequence repeats (Microsatellite markers)

1. INTRODUCTION

Ethiopia is a tropical country located in northeastern Africa between 3^0 and 15^0 latitude, and 33^0 and 48^0 longitude. The diverse physiographic conditions ranging from -125 m (Danakil depression) to 4620 m (Ras Dejen) above sea level coupled with contrasting climate have made possible the presence of diverse fauna and flora resources (FDRE 1997). Smallholder farm household production system where livestock plays a key role, is predominant producing over 90% of the total agricultural outputs (Shiferaw and Helden 1999). In the crop production subsystem, six major cereal crops (teff, wheat, barley, maize, sorghum, and millet) cover about 80% of the land under arable crop cultivation while contributing 77% of the country's annual gross grain production (CSA 2002). The first three crops are primarily cool-weather crops cultivated in the highlands at altitudes above l500 m above sea level.

Ethiopia is the second largest wheat producer (*Triticum* spp.) in the sub-Saharan Africa (White et al. 2001). Both bread (*T. aestivum* L.) and durum (*T. turgidum* var. *durum*) wheat are important food crops in Ethiopia. More than one million hectares of land is allocated for wheat production per year, making up 13% of the total crop production. About 85% of bread wheat is grown in south and southeast highlands (Arsi, Bale and Southeast Shoa), whereas durum wheat is produced predominantly on the central and northwestern highlands of the country (i.e., North Shoa, Gojam, Gonder and Tigray regions) (Fig. 1, page 6) (Hailu 1991). Though wheat is widely produced in the highlands and mid-altitudes, there is a considerable irrigated wheat production potential in lowlands (White et al. 2001).

Bread wheat occupies nearly 60% of the total wheat area and its cultivation is being expanding rapidly due to its high yield and wide adaptability (Bekele and Tanner 1995, White 2001). The indigenous durum wheat with few pocket areas of emmer wheat production

accounts for the remaining 40% of the total wheat area. The average wheat yield in the country is around 1.4 t/ha which is lower than the attainable yield over 5 t/ha (Hailu 1991). The yield gap of over 3 t/ha suggests the potential for increase wheat production in the country.

Over the last 40 years more than 30 wheat fungal pathogens have been identified in the major wheat producing areas of the country (Eshetu 1985). Yellow rust or stripe rust caused by the obligate pathogen *Puccinia striiformis* f. sp. *tritici*, is the most economically important disease. It causes severe yield losses when resistant commercial varieties tend to become susceptible due to changes in the pathogen-host interaction (Eshetu 1985, Badebo et al. 2001 and Dereje 2003). The geographical distribution of this pathogen overlaps with all major wheat producing areas of the country that range from 2150 to 2850 m above sea level. In this part of the country low temperatures with humid rainy days are common throughout the wheat growing season which favours the development of the disease (Mengistu et al. 1991, Bekele et al. 2002).

Four major stripe rust epidemics were reported between 1994 and 2004 that occurred at three to four years intervals in the central, south, and southeast Ethiopia. During these epidemics, previously resistant bread wheat cultivars were attacked to an extent that cultivars such as Dashen were completely put out of production after only a few years of deployment (Badebo et al. 2001, Bekele et al. 2002, Dereje 2003). Grain yield losses in susceptible common wheat cultivars ranged between 30 to 69% (Eshetu 1985, Bekele et al. 2002, Dereje 2003). Chen (2005) also reported a yield loss of 10 to 70% in most wheat producing areas of the world which varies with the susceptibility of the cultivar, initial infection time, rate of disease development, and duration of the disease. *Puccinia striiformis* f. sp. *tritici* is known to infect tetraploid wheat (Stubbs 1985) but an epidemic level of attack has never been experienced in Ethiopian highlands. Nevertheless, severe infection rates that could easily lead to epidemics

have been observed very recently on durum cultivars (Dereje and Chemeda 2006), suggesting the possibility of virulence changes in the pathogen population.

Although stripe rust can be effectively controlled by fungicide application, this is no way for small-scale farmers who produce the bulk of wheat production (95%) due to prohibitive costs in the local market. Thus the most environmentally and farmer friendly strategy for reducing yield losses is the use of rust resistant cultivars. However, this method primarily depends upon the availability of resistant donor parent genotypes. The knowledge of the genetic basis of resistance to stripe rust in wheat varieties is useful to understand the pathogen race composition and distribution.

With the discovery of genetic resistance by Biffen (1905), physiological specialization in the rust by Stakman and Levine (1992), and the gene for gene interaction by Flor (1959), the use of race specific types of resistance has been predominating in wheat improvement. However, resistance based on single race-specific genes often becomes ineffective within few years producing "boom and bust" cycles in wheat production (Zhengi 2002). The "bust" cycles (when rust resistance has been overcome) are caused by pathogens mutating to acquire virulence, virulent races migrating into a new region or races favoured by selection pressure from the existing pathogen population.

Rust pathogens migrate from one region to another by wind movement. For example, virulence in *P. striiformis* for the *Yr9* gene evolved in East Africa and migrated to North Africa, West Asia, and South Asia all the way to Nepal over a nine years period (Singh and Huerta-Espino 1996) causing major epidemics in Ethiopia, Turkey, Iran, Afghanistan, and Pakistan (Singh and Huerta-Espino 2001). Early detection of new virulences in pathogen populations combined with knowledge of resistance sources in cultivars would have been

made possible to replace susceptible cultivars with resistant cultivars in time to avoid large-scale epidemics.

Bonthuis (1985) noted that different planting dates and volunteer's wheat fields provide continuous sources of inoculum within or between East African countries. In addition, the mutation rate of the pathogen is expected to be high as high latitudes in these regions have intensive UV light exposure in the natural environment which increases the mutability of stripe rust isolates (Johnson et al. 1978). The phenomenon that single genes lose there effectiveness, is the reason to look for alternative resistance management strategies such as gene pyramiding, gene deployment, partial, and slow rusting (Danial 1994).

Annual virulence surveys, practices of gene postulation, and genetic analysis would greatly help to understand the status of the current host – pathogen interaction and thereby offer the possibility to predict the appearance of new virulence combinations (McIntosh and Brown 1997). For example, when the current situation indicates that the wheat cultivars grown in an area have only a single gene for resistance or when wheat cultivars are of similar genetic background for stripe rust resistance, it would be imperative to expect selection pressure to take place in the pathogen population (Steele et al. 2001).

Race specific resistance genes can be identified by comparing the reaction of the cultivars to a set of pathogen isolates with those displayed by testers carrying known *Yr* genes (Dubin et al. 1989, Hovmøller 2007). At present, 40 wheat stripe rust resistance genes have been catalogued (McIntosh et al. 1998 and 2007, Chen 2005). Most of these genes are unique as indicated by different chromosomal locations and differential response to races (Chen 2005). These resistance genes were identified from wild and related wheat species. Several researchers have employed the gene postulation technique for identifying *Yr* genes in a group

of wheat genotypes. For instance, Sharma et al. (1995) identified *Yr* genes *Yr2, Yr6, Yr8, Yr9, Yr10, Yr15, YrA,* and *YrSu* from 52 advanced emmer wheat lines; Hovmøller (2007) identified *Yr1, Yr2, Yr3, Yr4, Yr6, Yr9, Yr15, Yr17, Yr25,* and *Yr32* genes from 98 Danish wheat cultivars. Xia et al. (2007) identified stripe resistance genes *Yr2, Yr3a, Y4a, Y6, Yr7, Yr9, Yr26, Yr27, YrSel,* and *YrSd* from 72 Chinese wheat cultivars and advanced lines.

Tagging and mapping of resistance genes by molecular markers are efficient, reduce effort and expense of detection and allow pyramiding of major genes into wheat cultivars of interest. Application of molecular markers for mapping wheat stripe rust resistance genes has been demonstrated by several authors for *Yr5* (Sun et al. 2002, Yan et al. 2003, Chen et al. 2003), *Yr9 (*Shi et al. 2001*), Yr10* (Frick et al. 1998, Shao et al. 2001, Bariana et al. 2002, Smith et al. 2002, Wang et al. 2002), *Yr15* (Chague et al. 1999, Peng et al. 2000), *Yr17* (Robert et al. 1999, Seah et al. 2001), *Yr18* (Suenaga et al. 2003), *Yr24* (Zakari et al. 2003), *Yr26* (Ma et al. 2001), *Yr28* (Singh et al. 2000), *Yr32 (*Eriksen et al. 2004), *Yr33* and *Yr34* (McIntosh et al. 2004), *YrH52* (Peng et al. 2000), and *Yrns-B1* (Börner et al. 2000).

The present study was conducted to analyse the genetic structure of wheat cultivars and *P. striiformis* f. sp. *tritici* populations in the major wheat growing regions of Ethiopia. Possible ways are suggested to improve breeding of wheat cultivars with resistance against stripe rust.

2. MATERIALS AND METHODS

2.1. Population structure of *Puccinia striiformis* f. sp. *tritici*

2.1.1. *Puccinia striiformis* isolates

2.1.1.1. Sample collection

Collection of stripe rust samples was carried out in the 2005 growing season in Ethiopia. Stripe rust infected leaves were collected from breeders' variety trial plots, large commercial farms and small farmers' fields in Gonder, Gojam, Wello, Shoa, Arsi and Bale administrative regions, which represent the major wheat production areas of the country (Fig. 1). These were selected in such away that represent the diverse edaphic, climatic and wheat production systems in the country.

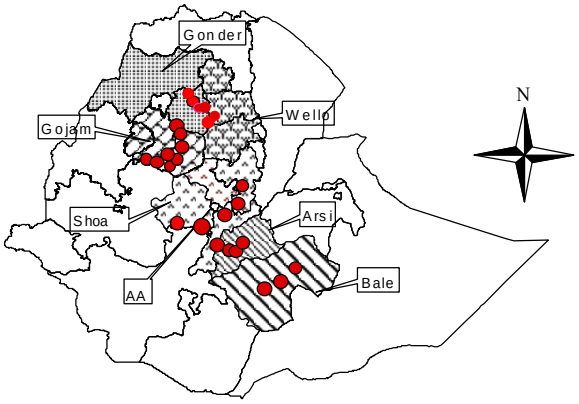

Fig. 1. Wheat stripe rust sampling location in Ethiopia, 2005

In the farmers and commercial fields, the collection was made in 20 km distances along the wheat belt of the country. Depending on the field size 2-3 samples were collected per field. A total of 230 samples were collected. Sporulating leaves were wrapped with plastic bags and put inside on ice-box at about 4^{o}C. In the laboratory, leaves were taped on plates, sporulating side up, and placed in a plastic bag with 100% humidity at 8^{o}C. After 12 h, spores were collected using a micro spore collector, desiccated on glycerine gel, and stored in a refrigerator at about 4^{o}C. After desiccation samples were stored at -80^{o}C for further use.

2.1.1.2. Isolate development

Isolate development and maintenance were carried out following the procedure developed by Villaral et al. (2002). To develop single-pustule isolates, 10-12 days old seedlings of the susceptible cultivar Morocco were inoculated with 5 mg spores mixed with 25 times talc powder using a 50 x 50 cm wide and 1 m high settling tower. Before inoculation, spores were taken out of -80^{o}C and heat shocked in a 40^{o}C water bath for 5 min. Inoculated plants were incubated in separate booths with water at the bottom, covered with plastic film, in a dark dew chamber for about 24 h at 8^{o}C and 100% relative humidity. Plants were then put back to the greenhouse in conditions, similar to those before inoculation, except for 20^{o}C and 80% relative humidity. Daylight was supplemented to 16 h with high-pressure sodium vapour lamps. After a week, unsporulated lesions were observed for each successful infection. One leaf bearing a single lesion was chosen from each pot, and the remaining leaves were removed. Pots were then replaced in individual booths for 7 to 9 days. When the isolated lesions were fully developed, spores were used to inoculate new plants for spore multiplication. A total of 107 monopustule isolates were developed from a total of 49 collected viable samples (Appendix 1).

2.1.1.3. Multiplication and maintenance of isolates

For spore multiplication seeds of the susceptible cultivar Morocco were sown in an 8 x 8 cm plastic pot and placed in a room with 16^{o}C. When seedlings reached a height of 3 cm, 15 ml of maleic acid (0.25 g L^{-1}) per pot were added to slow down seedling growth. Inoculation was performed by rubbing the leaves of 10–12 days old seedlings with sporulating leaves. Inoculated seedlings were incubated and grown as described in section 2.1.1.2. Eighteen days after inoculation, spores were collected by gently shaking the plants, dried in desiccators at 4°C for 3 days, and stored at -80°C until use.

2.1.1.4. Grouping of isolates

The stripe rust isolates were collected from Ethiopia largely differing in environmental conditions and production systems. Thus to gain insight into the regional diversity of the pathogen population in the country, four sub-groups were made based on the regions of collection. North Ethiopia (Wello, Gojam, and Gonder) formed the first region of collection with 24 isolates. The second region of collection, central Ethiopia (Shoa), consisted of 31 isolates. In north and central highlands of Ethiopia durum is the dominant wheat crop, which is primarily grown on poorly drained valley bottoms soils with altitude range of 2000 to 2400 m above sea level (Hailu 1991). South (Arsi) and southeast (Bale) are the third and the fourth regions which each consisted of 26 isolates. These regions are known for their bread wheat production; about 85% of bread wheat is grown in south and southeast highlands. Well-drained soil with a slightly higher elevations (>2400) m above sea level is characterstic for the growing regions.

2.1.2. Virulence assessment

For determination of the virulence spectra of the isolates, differential host genotypes with known seedling resistance genes were used. These include entries from the European and world sets (Johnson et al. 1972), and near-isogenic lines with the Avocet S background (Wellings et. al. 2004) (Table 1). All isogenic lines were kindly provided by Dr. C. Wellings, Plant Breeding Institute, University of Sydney, Australia. These differential genotypes allowed to detect the virulences matching the resistance genes *Yr1, Yr2, Yr3, Yr4, Yr5, Yr6, Yr7, Yr8, Yr9, Yr10, Yr15, Yr17, Yr24, Yr26, Yr27, Yr32, YrCv, YrA, YrSu, YrSd,* and *YrSp.*

Virulence assessment was carried out using the method developed by Stubbs (1988). Five seeds of each differential genotype and the susceptible cultivar Morocco were grown as described in section 2.1.1.2. Spores of each isolate mixed in mineral oil (FC-43) at the ratio of 2-3 mg/ml were used to inoculate 10-12 days old seedlings of the differentials and the susceptible cultivar. After inoculation seedlings were incubated at 10^{o}C for 24 h and then transferred at 20^{o}C for a couple of week as describe in section 2.1.1.2.

The infection types (IT) of *P. striiformis* on the wheat genotypes were scored using a 0 to 9 scale 15-18 days after inoculation as described by MacNeal et al. (1971) and Stubbs (1988) (Table 2). They were classified into resistant (0-5) and susceptible (6-9) types.

Table 1. Differential genotypes used for virulence assessment.

No.	Differential genotype	*Yr* genes
1	*Yr1*/6*Avocet S	*Yr1*
2	Heines VII	*Yr2+*
3	Vilmorin 23	*Yr3a*
4	Nord Desprez	*Yr3b*
5	Hybrid 46	*Yr4a*
6	*Yr5*/6* Avocet S	*Yr5*
7	*Yr6/6**Avocet S	*Yr6*
8	Heines Peko	*Yr6+2*
9	*Yr7/6**Avocet S	*Yr7*
10	*Yr8*/6*Avocet S	*Yr8*
11	*Yr9*/6*Avocet S	*Yr9*
12	Clement	*Yr9+2*
13	*Yr10*/6*Avocet S	*Yr10*
14	*Yr15*/6*Avocet S	*Yr15*
15	Rendezvous	*Yr17*
16	*Yr24*/6*Avocet S	*Yr24*
17	*Yr26*/6*Avocet S	*Yr26*
18	*Yr27*/6*Avocet S	*Yr27*
19	*Yr32*/6*Avocet S	*Yr32*
20	Carstens V	*YrCv*
21	Anza/Avocet ´R´	*YrA*
22	Suwon 92 x Omar	*YrSu*
23	Strubes Dickkopf	*YrSd*
24	*YrSp*/6*Avocet S	*YrSp*

Table 2. Infection types of *Puccinia striiformis* f. sp. *tritici* isolates on wheat.

Description of infection	Index value
Immune/no symptom	**0**
Hypersensitive flecks without uredia	**1**
Necrotic/Chlorotic flecks without uredia	**2**
Necrotic/Chlorotic stripes without uredia	**3**
Necrotic/Chlorotic stripes with trace uredia	**4**
Necrotic/Chlorotic stripes with light uredia	**5**
Necrotic/Chlorotic stripes with intermediate uredia	**6**
Necrotic/Chlorotic stripes with moderate uredia	**7**
Chlorotic stripes with abundant uredia	**8**
Abundant uredia without necrosis/chlorosis	**9**

2.1.3. Designation of pathotype

The pathotypes were named according to Gilmour (1973). This was by dividing the 24 differentials into eight triplets. The binary values 0 (avirulence, resistance) and 1 (virulence, susceptebility) of each triplet were transformed into an octal number with the range of 0-7(Table 3). The sequence of the differentials in the set is given in Table 1. The number derived then serves as a race name for that particular pathotype and translation back to the binary code reveals the spectrum of virulences. HaGis (Habgood-Gilmour) spreadsheet was used for data entry and analysis (Herrmann et al. 1999).

Table 3. Transformation of triple codes into octal notation to name isolates.

Virulence per triplet	Differential genotype			Octal number
	A	B	C	
None	0	0	0	0
One	1	0	0	1
	0	1	0	2
	0	0	1	3
Two	1	1	0	4
	1	0	1	5
	0	1	1	6
Three	1	1	1	7

Source: Gilmour (1973)

2.1.4. Population structure

2.1.4.1. Virulence frequency

The virulence frequency expresses the proportion of virulent isolates per gene. Based on the calculated value of virulence gene frequency, the level of effectiveness of the resistance genes was classified using the grouping system developed by Felsenstein and Jaser (2000) (Table 4).

Table 4. Classification of the effectiveness of stripe rust resistance genes as a function of virulence frequency in the pathogen population.

Virulence gene frequency (%)	Resistance level
0-10	Very high
>10-20	High
>20-50	Moderate
>50	Low/no

Source: Felsenstein and Jaser (2000)

2.1.4.2. Virulence complexity

The virulence complexity of each pathotype was obtained by the sum of its virulences to the genes in the differential set (Welz 1986). Thus it gives information about the isolate ability to overcome the resistance genes of the host plant. However, the interpretation of the complexity value is primarily depending on the size and choice of the set of differential genotypes (Löwer 1999). Hence in the present study cutting points of 0-5, 6-12, and 13-24 were taken to represent a low, moderate and high virulence, respectively.

2.1.4.3. Pathotype abundance

Pathotype abundance is the proportion of individual pathotypes in the population.

2.1.4.4. Population diversity

The phenotypic diversity within populations (locations) was characterized using Shannon, Simpson and Gleason diversity indices.

The Shannon index (H) (Shannon and Weaver 1949) was used to determine the similarities of the frequencies and evenness of the different pathotypes in a set of isolates.

$$H = -\sum_{i=1}^{s} \left(\frac{ni}{N}\right).\ln\left(\frac{ni}{N}\right)$$

Simpson's diversity index (S) (Simpson 1949) used to determine the concentration of different pathotype in the population (richness). The formula is:

$$S = 1 - \sum_{i=1}^{s} \frac{ni(ni-1)}{N(N-1)}$$

where n_i = the number of isolates of the i^{th} pathotype, s = number of different pathotypes in the sample, and N = total number of isolates. The value of ´S´ ranges between 0 and 1. 0 represents no diversity and 1 represents infinitive diversity.

Gleason index (G) (Groth and Roelfs 1987) was used to detect the number of distinct pathotypes. It is considered less sensitive to the sample size and is calculated by the following formula:

$$G = \frac{(n-1)}{\ln(N)}$$

where n is the number of pathotypes, N is the total number of isolates in the sample and 1n = natural logarism.

The Kosman diversity index (H_{KW}), proposed by Kosman (1996) is used to determine the genetic relationship of pathotypes within the population. The value of H_{KW} for population A is calculated as follows:

$$H_{KW} = \frac{1}{nk}.ASS_{max}(A, A)$$

where n refers to the number of isolates, k = number of differentials, and ASS_{max} (A, A) = the maximum number of matched pairs of isolates. The value ranges from 0 to 1; $H_{KW} = 0$ for a population that is limited to a single pathotype. The maximum diversity score is assigned to a population which consists of pairs of isolates with absolutely different response patterns on the entire set of differentiating factors (differentials).

Diversity between populations was estimated by Rogers´ index (R) and Kosman´s distance (H_{KB}). Roger's index (Rogers 1972) provides a comparison between two populations on the basis of the frequencies of phenotypes that occur in the populations, regardless of how many shared virulences occur.

$$R = \frac{1}{2}\sum_{i=1}^{n}|P_{Ai} - P_{Bi}|$$

where P_{Ai} and P_{Bi} are frequencies of the i^{th} pathotype in populations A and B, respectively and n is the total number of different pathotypes in both populations. Rogers´ index varies from 0, for two populations with identical pathotype structure, to 1, for populations with no pathotype in common.

Kosman´s distance (H_{KB}) (Kosman 1996) takes into account both pathotype frequencies and degrees of similarity among distinct pathotype. The Kosman distance between two populations A and B of n individuals is defined as follows:

$$H_{KB} = \frac{1}{n_p k} . ASS_{min} (A, B)$$

where Ass_{min} (A, B) = minimum value of sum of distances between matched pairs of isolates, n_p = number of matched pairs isolates, and k = number of differentials. If the samples of isolates from the two populations are identical, then H_{KB} =0; if the samples are different, then $0 < H_{KB} \leq 1$.

2.1.4.5. Stastical analysis

Virulence complexity, phenotypic and genetic diversity indices data were subjected to statistical analysis. Pair wise comparisons between the regions were tested with the t-test. The preliminary variance equality test was made using SPSS statistical package (SPSS 2004). The Simpson (S), Kosman diversity index (H_{KW}), Rogers (R), and Kosman´s distance (H_{KB}) were found to have equal variance so that a common pooled variance could be used. Virulence complexity and Gleason index had unequal variances, so that the degrees of freedom were calculated using the approximation of Satterthwaite (1946). Following Jain et al. (1975) the same approximation was used for the Shannon index. In all cases the significance level for differences between means was P = 0.05.

To determine the relationship between indices 2-tailed pearson correlations test was employed. The square of the correlation coefficient (r^2) was used as a measure of the amount of variability in one variable that is explained by the other. The significance level for the

correlation between two variables was P=0.05. The sign of correlation coefficient (+, -) was used to differentiate whether the relationship between the two variables was positive or negative.

2.1.4.6. Cluster analysis

To examine the similarity of pathotypes between sampling regions, cluster analysis was performed by using complete linkage method using SAS 8.1 software (SAS INSTITUTE Inc. 1999) (Proc Cluster). A dendrogram was developed for the clustering of isolates.

2.2. Resistance test of Ethiopian wheat cultivars

2.2.1. Gene postulation

2.2.1.1. Stripe rust isolates

Twenty stripe rust isolates which had various combinations of avirulence and virulence with respect to *Yr1, Yr2, Yr3, Yr4, Yr6, Yr7, Yr8, Yr9, Yr10, Yr17, Yr24 , Yr27, Yr32, YrA, YrSu, YrCv, YrSd,* and *YrSp* were used to inoculate the wheat differentials and cultivars. Three French isolates were kindly provided by Dr. Claude de Vallavieille-Pope, INRA Epidémiologie Végétale, Thiverval-Grignon, France. Two isolates were obtained from the Research Institute of Plant Protection (IPO), Wageningen, the Netherlands. Six of the Ethiopian isolates where selected from stripe rust isolate sample collection of 2005. Nine isolates were obtained from the German isolate collection of the Julius Kühn-Institute in Braunschweig. The isolates were selected based on their virulence spectrum, and not for their country of origin. The list of the isolates, source and reaction on the differentials are indicated in Table 5.

Table 5. List, source and virulence profile of isolates used for gene postulation.

Isolate	Source	Virulence profile	No. of virulence factors
6E18	France	*Yr6+2*, *7*, and *32*	3
6E22	Germany	*Yr2*, *3a*, *6+2*, *7*, *8*, *27*, *32*, *Su*, and *A*	9
32E0	Germany	*Yr32* and *A*	2
33E32	Germany	*Yr1*, *32*, *Cv*, *Su*, and *Sd*	5
39E134	Germany	*Yr1*, *2*, *3a*, *3b*, *6+2*, *6*, *7*, *32*, *Cv*, *Su*, and *Sd*	11
41E168	Germany	*Yr1*, *2+*, *3a*, *3b*, *27*, *32*, *Cv*, *Su*, *Sd*, and *Sp*	10
44E204	Germany	*Yr2*, *3a*, *3b*, *4a*, *6+2*, *7*, *32*, *A*, *Cv*, *Su*, *Sd*, and *Sp*	12
45E204	France	*Yr1*, *2*, *3a*, *3b*, *6+2*, *6*, *32*, *Cv*, *A*, and *Sd*	10
108E141	Germany	*Yr2*, *3a*, *3b*, *4a*, *6+2*, *6*, *27*, *32*, *A*, *Cv*, and *Sd*	11
109E141	Germany	*Yr1*, *2*, *3a*, *3b*, *4a* ,*6+2*, *6*, *27*, *32*, *A*, *Su*, and *Sd*	12
169E136	Germany	*Yr1*, *2*, *3a*, *3b*, *4a*, *9+2*, *9*, *17*, *27*, *32*, *A*, *Cv*, and *Sd*	13
232E137	Netherlands	*Yr1*, *2*, *3a*, *3b*, *4a*, *6+2*, *6*, *9+2*, *9*, *27*, *32*, *A*, *Cv*, *Su*, and *Sd*	15
237E141	Netherlands	*Yr1*, *2*,, *3a*, *3b*, *4a*, *6+2*, *6*, *9+2*, *9*, *32*, *A*, *Cv*, *Su*, and *Sd*	14
32E01	France	*Yr3b*, *6*, *27*, *32*, *A*, *Cv*, and *Sd*	7
230E150	Ethiopia	*Yr2*, *3a*, *6+2*, *6*, *7*, *8*, *9+2*, *9*, *24*, *27*, *32*, *Su*, and *Sd*	13
70E16	Ethiopia	*Yr2*, *6*, *7*, *8*, *24*, *27*, *A*, and *Su*	8
232E158	Ethiopia	*Yr2*, *3a*, *3b*, *6+2*, *6*, *7*, *8*, *9+2*, *9*, *27*, *32*, *A*, *Cv*, *Su*, and *Sd*	15
86E30	Ethiopia	*Yr2*, *3b*, *6+2*, *6*, *7*, *8*, *9*, *10*, *A*, *Cv*, and *Su*	11
230E158	Ethiopia	*Yr2*, *3b*, *6+2*, *6*, *7*, *8*, *9+2*, *9*, *27*, *A*, *Su*, and *Sd*	12
70E158	Ethiopia	*Yr2*, *3a*, *3b*, *6+2*, *6*, *7*, *8*, *9+2*, *9*, *27*, *A*, and *Su*	12

2.2.1.2. Plant materials

A total of 64 wheat genotypes (24 differentials, 25 bread and 15 durum wheat cultivars) were tested for their reaction to infection by 20 isolates of *P. striiformis* isolates. The list and pedigree of the wheat cultivars and differential genotypes investigated are given in Table 6 and 14 (page 44). The bread and durum wheat cultivars represent Ethiopian commercial and donor parents. They are obtained from the National Wheat Program, IEAR, Ethiopia.

2.2.1.3. Seedling resistance test

For resistance tests, five seeds of each genotype were sown in a separate hole in a plastic tray of 30 x 15 cm. The test was carried out in three replications in a room with 16^0C and 80% relative humidity. Each genotype was challenged with 20 isolates (Table 5). The method of inoculation and incubation and disease assessments were made as outlined in section 2.1.1.2 and 2.1.2.

2.2.1.4. Interpretation of results

The stripe rust resistance genes in the cultivars were postulated by comparing the infection types displayed by the cultivars with those displayed by the known *Yr* gene carrying testers. The reaction patterns were interpreted according to Hovmøller and Justesen (2007), in terms of most likely *Yr* genes and/or *Yr* genes combined with additional sources, based on infection types produced on the differential host genotypes. Dubin et al. (1989) also reported a brief set of steps for postulating the presence of genes.

2.2.2. Adult plant resistance test

To investigate the presence of adult plant resistance (APR) in the cultivars, field and greenhouse tests were carried out at the adult stage.

Table 6. List of Ethiopian bread and durum wheat genotypes used for gene postulation.

Genotype	Code	Pedigree[1]
Bread wheat		
Pavon 76	HAR 437	VCM/CNO "S"/7C/3/KAL/BB
Mada walabu	HAR 1480	TI/3/Fn/Th/Nar 59 *2/4/Bol'S'
Mitike	HAR 1709	FSYR20.6/87 BOW28//RBC (ET1297)
Simba	HAR 2536	PRL/VEE//MYNA/VUL
KBG-01	FH6-1-7°	(300/SM+501M)/HAR 1709
Dodota	HAR 2508	BJY/COC//PRL/BOW
K 6295-4A	HAR 515	ROMANY x GB-GAMENYA
Suf-Omer	HAR 1889	4777(2)//FKN/GB/3/PVN"S"
Bobicho	HAR 1889	PEG/PF70354/KAL/BB/ALD/3/MRNG
Kubsa	HAR 1685	ND VG9144//KAL/BB/3/YACO´´S´´/4VEE#5´´S´´
Wetera	HAR 1920	MON´´S´´BUC´´S´´
Tusie	HAR 1407	COOK/VEE//DOVE/SERI
Abola	HAR 1522	BOW/BUC
Galama	HAR 604	4777(2)//FKN/GB/3/PVN
Sirbo	HAR 2192	VS73.600/MRL/3/BOW//YR//TRF
ET-13	-	UQ105 Sel X ENKOY
Wabe	HAR 710	MRL"S"-BUC"S"
Kater	HAR 1899	COOK/VEE//DOVE/SERI/3/BIY/COC
Tura	HAR 1775	ARO YR SEL. 60/89
Dereseligh	-	(300/SM+501M)/HAR 1709
Magal	HAR 710	F371/TRM//BUC´´BUC´´S´´/3/LIRA´´S´´
Hawii	HAR 2501	CHIL/PRL
Shinna	HAR 1868	GOV9AZ//MUS/3/R37/GHL121//KAL/BB/4/ANI
K6290-Bulk	-	BJY/COC//PRL/BOW
Dashen	HAR 408	VEE 17 KUZ-BUHO "S"/KAL-BB
Durum wheat		
Boohai		Na
DZ-04-118		Developed by mass selection
Foka		Na
Kilinto		Na
Cocorit-71		Na
Gerardo		VZ 466/61-130xGII''s'', CM9605
Tob-66		Na
Assasa		Na
Ld-357		Na
Bichena		Na
Quamy		Na
Robe		Na
Ginchi		Na
Yerer		Chen/Tez/3/Guil//CII
Ude		Chen/ALTAR 84//Aid

[1]Na: not available

2.2.2.1. Field adult plant resistant test

The field experiment was laid out in randomised complete block design (RCBD) in four replications. Each variety was planted on 60 x 80 cm plots. The experiment was conducted in the main growing season for three years, 2005-07. To generate a sufficient disease infection pressure, a mixture of susceptible wheat varieties were used as spreader rows between the test plots along the main wind direction at a distance of 3-4 m. Disease was provocated by artificiall inoculation of the spreader rows at tillering (growth stage of BBCH 21-23) using a micro sprayer and an oil spore suspension (appr. 120 mg spores per 100 m of spreader row, light oil `Isopar M´ or `Fluorinert FC-43´). A mixture of four virulent races (6E22, 39E134, 44E204, and 232E137) which were virulent on the cultivars at the seedling stage was used to investigate the presence of adult plant resistance in the field. To get enough dew and 10^{o}C that is required for yellow rust infection, inoculation were done only early in the morning. The percentage of leaf area infection as designated by Peterson et al (1945), was used to assess the yellow rust severity three times in the growing season starting from heading to maturity stage of the crop.

2.2.2.2. Greenhouse adult plant resistant test

Although field data of disease severities and race composition can indicate the presence of adult plant resistance, greenhouse tests, using isolates virulent to the seedlings of a genotype under controlled temperature (30^{o}C) conditions were conducted for confirmation. For this, five seeds of each genotype were sown in a 15 cm diameter plastic pot and grown in a room with 16^{0}C and 80% relative humidity. The experiments were conducted in four replications. A mixture of the same isolates listed above was used to investigate the presence of APR in the greenhouse. For inoculation, 400 mg of spores (100 mg of each isolates) mixed with 500 ml of mineral oil (FC-43) were used to inoculate plants using a micro sprayer. Inoculation was done twice. The first inoculation was carried out at the begnning of stem elongation stage

(growth stage of BBCH 29), the second was done two weeks after the first inoculation. After inoculation plants were incubated for 24 h in the dark at 10°C and 100% relative humidity. Then after transferred to a room that have a similar condition before inoculation, except 30°C. Disease severity assessments were made as outlined in section 2.2.2.1.

2.2.2.3. Interpretation of results

The level of resistance under greenhouse and field conditions, when challenged by virulent isolates (defined at the seedling stage) was categorized low (>10%) and high (≤10%) resistance. Cultivars in the category, which had ≤10% leaf area covered, were considered to possess components of effective APR (Hovmøller 2007).

2.3. Mapping of *Yr* genes using SSR markers

Molecular markers were used to map the stripe rust resistance genes from cultivars Suf-Omer (HAR 1889), Wetera (HAR 1920), Tusie (HAR 1407), and Wabe (HAR 710). Wabe and Tusie were resistant to all isolates tested, whereas Suf-Omer and Wetera were susceptible only to two isolate at the seedling stage. For mapping, simple sequence repeat (SSR) markers were applied. This type of molecular marker is genome-specific, appears to be evenly distributed over the wheat genome and shows a higher level of polymorphism compared to any other marker system (Röder et al. 1998).

2.3.1. Development of F_2 populations

F_2 mapping populations were derived by selfing of F_1 seeds from the crosses the four cultivars Suf-Omer, Wetera, Tusie and Wabe resistance to stripe rust isolate 41E168 with the susceptible German variety Thasos.

2.3.2. Resistance test of the F_2 populations

F_2 plants were grown in the greenhouse in the same way as described in section 2.1.1.2. Spores of the isolate 41E168, which were avirulent to the donor parents and virulent to the susceptible parent, mixed with mineral oil (FC-43) at the ratio of 2-3 mg/ml, were used to inoculate 10–12 days old seedlings of the F_2 populations. Disease assessment was made 15 to 18 days after inoculation with similar methods as described in section 2.1.2.

2.3.3. DNA extraction and bulk segregant analysis

DNA was extracted from the tertiary seedling leaves using the Doyle's method (Doyle and Doyle 1990) with minor modifications. To provide maximum coverage of the A, B, and D genome, 257 SSR wheat markers (six to ten per chromosome) were selected from the mapped SSR markers of wheat (Röder et al. 1998) and tested for polymorphism between the resistant and susceptible parents. Markers associated with the resistance locus were identified by bulk segregant analysis (BSA) (Michelmore et al. 1991) by separate pooling of equal amounts of DNA (3 µl) from 10 resistant and 10 susceptible plants from the segregating F_2 populations. The candidate markers were further screened on 10 resistant and 10 susceptible single F_2 plants grouped individually for clear linkage analysis. The markers for which linkage to the target gene was confirmed were tested on all genotypes on the respective mapping populations.

2.3.4. PCR amplification

Polymerase chain reactions (PCR) were performed in Perkin-Elmer (Norwalk, CT) thermocyclers in a total volume of 25 µl containing 50-100 ng each template DNA, 250 nM cy5-labelled forward primer, 250 nM unlabelled reverse primer, 0.2 mM dNTPs, 2.5 µl PCR buffer (10x), 1.5 mM $MgCl_2$, and 1U *Taq* DNA polymerase. After 3 min at 94^0C, 45 cycles

were performed with 1 min at 94^0C, 1 min at either 50, 55, 60^0 C (depending on the individual microsatellite), 2 min at 72^0C, and a final extension step of 10 min at 72^0C. Fragments were detected by an Automated Laser Fluorescence (ALF express) sequencer (Amersham Biosciences) and fragment sizes were calculated using the computer program Fragment Analyser 1.02 (Amersham Biosciences) by comparison with internal size standards. In case of weak or no fragment products, PCR amplifications were repeated to exclude failed PCR reactions as cause of null allele. PCR products were denatured at 94^0C for 2 min and placed on a cold block until use. Each sample of 6 µl (4 µl PCR product and 2 µl internal markers) and one external standard marker of 6 µl were loaded in the preheated gel.

2.3.5. Statistical analysis

Chi-square (χ^2) test was applied to the data to test goodness of fit to postulated ratios. Map construction and recombination rates between stripe rust and SSR markers were calculated using Joinmap 3.0 (van Ooijen and Voorrips 2001) regarding infection types 0-5 as resistant and 6-9 as susceptible. Logarithmic odds ratios (LOD) scores of 3.0 or greater were chosen for declaration of linkage. The Kosambi´s (1944) mapping function was used to convert recombination frequencies to map distances in centi-Morgans (cM). No SSR marker data were available for Tusie x Thasos.

3. RESULTS

3.1. Population structure of *Puccinia striiformis* populations in Ethiopia

3.1.1. Diversity of virulence

Pathogenecity of 107 isolates of *Puccinia striiformis* f. sp. *tritici* collected from four major wheat growing regions of Ethiopia was tested using 24 differential genotypes. The isolates belonged to 39 different pathotypes and all were virulent on a differential genotype carrying resistance gene *Yr7* and avirulent on the four genotypes that possess *Yr1, Yr5, Yr15,* and *YrSp* genes (Table 7). The pathotypes differed from each other by virulence on 19 differential genotypes carrying resistance genes *Yr2*, *Yr3a*, *Yr3b*, *Yr4a*, *Yr6, Yr6+2, Yr8, Yr9*, *Yr9+2, Yr10, Yr17, Yr24, Yr26, Yr27, Yr32, YrCv, YrA, YrSd,* and *YrSu.*

Table 7. Thirty nine pathotypes from 107 isolates found in 2005 and their reaction on the corresponding *Yr* resistance genes possessed by 24 wheat differentials.

Pathotype	No. of isolates	*Yr1*	*Yr2+*	*Yr3a*	*Yr3b*	*Yr4a*	*Yr5*	*Yr6+2*	*Yr6*	*Yr7*	*Yr8*	*Yr9+2*	*Yr9*	*Yr10*	*Yr15*	*Yr17*	*Yr24*	*Yr26*	*Yr27*	*Yr32*	*YrCv*	*YrA*	*YrSu*	*YrSd*	*YrSp*	Complexity
21650040	4	-	+	-	+	-	-	-	+	+	+	-	+	-	-	-	-	-	-	-	-	+	-	-	-	7
00611701	1	-	-	-	-	-	-	+	+	+	-	-	-	+	-	-	+	+	+	-	-	-	+	-	-	8
00411351	1	-	-	-	-	-	-	-	+	+	-	-	-	+	-	-	+	+	+	-	-	+	+	-	-	8
21650450	4	-	+	-	+	-	-	-	+	+	+	-	+	-	-	-	-	-	+	+	-	+	-	-	-	9
00611741	3	-	-	-	-	-	-	-	+	+	+	-	-	+	-	-	+	+	+	-	-	-	+	+	-	9
01411351	1	-	-	-	+	-	-	-	-	+	+	-	-	+	-	-	+	+	-	+	-	+	+	-	-	9
21770440	6	-	+	-	+	-	-	+	+	+	+	-	+	+	-	-	-	-	+	-	-	+	-	-	-	10
21650461	3	-	+	+	-	-	-	-	+	+	+	-	+	-	-	-	-	-	+	-	+	+	+	-	-	10
20750461	1	-	-	+	-	-	-	+	+	+	+	-	+	-	-	-	-	-	+	+	-	+	+	-	-	10
21650063	2	-	+	-	+	-	-	-	+	+	+	-	+	-	-	-	-	-	-	-	+	+	+	+	-	10
01644473	1	-	-	-	+	-	-	-	+	+	-	-	+	-	-	+	-	-	+	+	+	+	+	+	-	11
21750461	1	-	+	-	+	-	-	+	+	+	+	-	+	-	-	-	-	-	+	-	+	+	+	-	-	11
20650553	1	-	-	+	-	-	-	-	+	+	+	-	+	-	-	-	+	-	+	+	-	+	+	+	-	11
21650361	1	-	+	-	+	-	-	-	+	+	+	-	+	-	-	-	+	+	-	-	+	+	+	-	-	11
00651751	7	-	-	-	-	-	-	-	+	+	+	-	+	+	-	-	+	+	+	+	-	+	+	-	-	11
01654463	6	-	-	-	+	-	-	-	+	+	+	-	+	-	-	+	-	-	+	-	+	+	+	+	-	11
01650473	7	-	-	-	+	-	-	-	+	+	+	-	+	-	-	-	-	-	+	+	+	+	+	+	-	11
03750361	1	-	-	-	+	+	-	+	+	+	+	+	-	-	-	-	+	+	-	-	+	+	+	-	-	12
01651751	2	-	-	+	-	-	-	-	+	+	+	-	+	+	-	-	+	+	+	+	-	+	+	-	-	12
21650473	8	-	+	-	+	-	-	-	+	+	+	-	+	-	-	-	-	-	+	+	+	+	+	+	-	12
20770453	4	-	+	-	-	-	-	+	+	+	+	+	+	-	-	-	-	-	+	+	-	+	+	+	-	12
21650553	1	-	+	-	-	+	-	-	+	+	+	-	+	-	-	-	+	-	+	+	-	+	+	+	-	12
01754473	3	-	-	-	+	-	-	+	+	+	+	+	-	-	-	+	-	-	+	+	+	+	+	+	-	13
23650473	2	-	+	-	+	+	-	-	+	+	+	-	+	-	-	-	-	-	+	+	+	+	+	+	-	13
03750473	1	-	-	-	+	+	-	+	+	+	+	-	+	-	-	-	-	-	+	+	+	+	+	+	-	13
21654473	5	-	+	-	+	-	-	-	+	+	+	-	+	-	-	+	-	-	+	+	+	+	+	+	-	13
21750473	3	-	+	-	+	-	-	+	+	+	+	-	+	-	-	-	-	-	+	+	+	+	+	+	-	13
21670473	1	-	+	-	+	-	-	-	+	+	+	+	+	-	-	-	-	-	+	+	+	+	+	+	-	13
61750453	1	-	+	+	+	-	-	+	+	+	+	-	+	-	-	-	-	-	+	+	-	+	+	+	-	13
21750553	1	-	+	-	+	-	-	+	+	+	+	-	+	-	-	-	+	-	+	+	-	+	+	+	-	13
61770453	3	-	+	+	+	-	-	+	+	+	+	+	+	-	-	-	-	-	+	+	-	+	+	+	-	14
23750473	1	-	+	-	+	+	-	+	+	+	+	-	+	-	-	-	-	-	+	+	+	+	+	+	-	14
21651771	1	-	+	-	+	-	-	-	+	+	+	-	+	+	-	-	+	+	+	+	+	+	+	-	-	14
21750573	2	-	+	-	+	-	-	+	+	+	+	-	+	-	-	-	+	-	+	+	+	+	+	+	-	14
21770473	13	-	+	-	+	-	-	+	+	+	+	+	+	-	-	-	-	-	+	+	+	+	+	+	-	14
21670773	1	-	+	-	+	-	-	-	+	+	+	+	+	-	-	-	+	+	+	+	+	+	+	+	-	15
61770473	1	-	+	+	+	-	-	+	+	+	+	+	+	-	-	-	-	-	+	+	+	+	+	+	-	15
23770473	1	-	+	-	+	+	-	+	+	+	+	+	+	-	-	-	-	-	+	+	+	+	+	+	-	15
21751773	1	-	+	-	+	-	-	+	+	+	+	-	+	+	-	-	+	+	+	+	+	+	+	+	-	16

* + = compatible reaction (IT 7-9); - = incompatible reaction (IT 0-6)

3.1.2. Virulence frequency

The average virulence frequency of *Yr6, Yr8, Yr9*, *Yr27*, and *YrA* genes were near the upper limit (91-99%) while that of *Yr7* gene was 100% (Fig. 2 and Table 8). The virulence frequency of *Yr2+, Yr3b, Yr32, YrCv, YrSu,* and *YrSd* varied between 65 to 87% with a mean value of 75%. Differentials with the *Yr3a* and *Yr4a* resistance genes showed the second and third lowest average virulence frequency of 5% and 6%, respectively. *Yr6+2, Yr9+2, Yr10, Yr17, Yr24,* and *Yr26* genes showed intermediate virulence frequencies, which ranged between 14 to 41%.

The virulence revealed variation among locations. Virulence to resistance gene *Yr3a* was absent in central Ethiopian isolates, whereas it was present in isolates from the other three locations. The population mean was 5% and ranged from 4% (in south and southeast) to 13% (north). Virulence to *Yr17* was recorded in all regions except those from the south and southeast. Likewise, *Yr4a* virulence was not detected in the central and north region. The frequency of *Yr4a* gene was comparable with that of *Yr3a* (population mean of 6%) and varied from 4% (south) to 19% (southeast). The frequency of *Yr17* was also low but higher than *Yr3a* (population average was 14%) and ranged from 19% (central) to 38% (north) (Table 8).

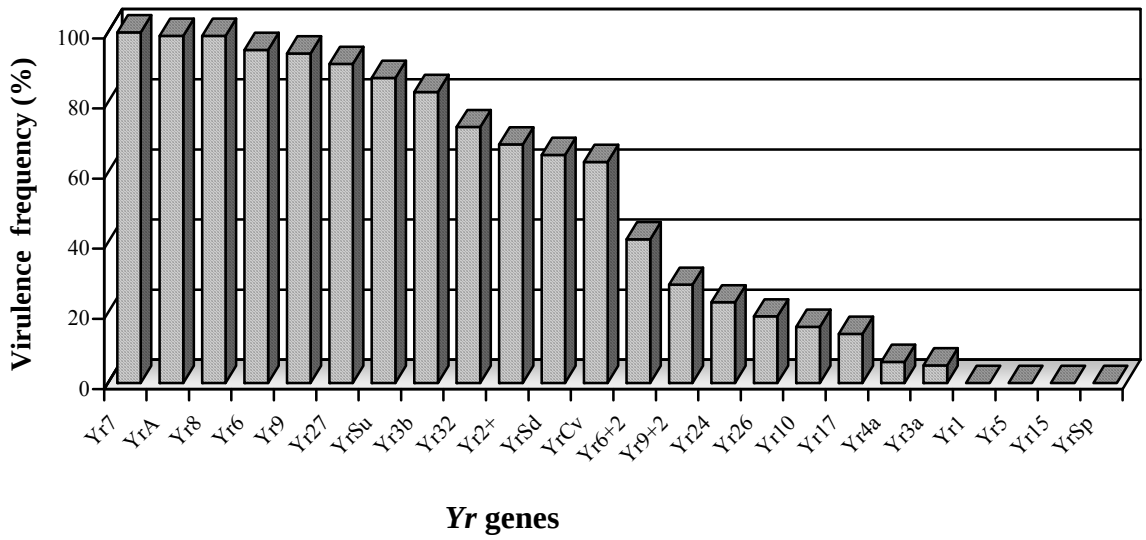

Fig. 2. Distribution of virulence frequency to stripe rust of wheat across the country to the samples collected in 2005.

Table 8. Virulence frequencies (%) of 107 isolates of *Puccinia striiformis* f. sp. *tritici* collected from north, central, south and southeast Ethiopia in 2005.

Differential genotype	*Yr* gene	Location				
		North (n = 24)	Central (n = 31)	South (n = 26)	Southeast (n = 26)	Total (n = 107)
Yr1/6*Avocet S	*Yr1*	0	0	0	0	0
Heines VII	*Yr2+*	46	68	73	85	68
Vilmorin 23	*Yr3a*	13	0	4	4	5
Nord Desprez	*Yr3b*	79	65	100	92	83
Hybrid 46	*Yr4a*	0	0	4	19	6
Yr5/6* Avocet S	*Yr5*	0	0	0	0	0
*Yr6/6**Avocet S	*Yr6*	92	100	100	100	95
Heines Peko	*Yr6+2*	13	42	54	54	41
*Yr7/6**Avocet S	*Yr7*	100	100	100	100	100
Yr8/6*Avocet S	*Yr8*	96	100	100	100	99
Yr9/6*Avocet S	*Yr9*	75	100	100	100	94
Clement	*Yr9+2*	13	32	27	38	28
Yr10/ 6*Avocet S	*Yr10*	25	23	8	8	16
Yr15/6*Avocet S	*Yr15*	0	0	0	0	0
Rendezvous	*Yr17*	38	19	0	0	14
Yr24/6*Avocet S	*Yr24*	25	23	23	23	23
Yr26/6*Avocet S	*Yr26*	25	23	12	15	19
Yr27/6*Avocet S	*Yr27*	92	87	100	85	91
Yr32/6*Avocet S	*Yr32*	50	68	100	73	73
Carstens V	*YrCv*	46	32	92	85	63
Anza/Avocet ´R´	*YrA*	96	100	100	100	99
Suwon 92 x Omar	*YrSu*	83	65	100	100	87
Stubes Dickkopf	*YrSd*	50	45	96	73	65
YrSp/6*Avocet S	*YrSp*	0	0	0	0	0

KEY: Gray colours indicate ranges 0-100 as shown below

51-100%	21-50%	0-20%

3.1.3. Virulence complexity

Table 9 shows the distribution and frequency of pathotypes and corresponding isolates according to the number of virulence genes (virulence complexity). The pathotypes contained 7 to 16 virulences (Fig. 3). The most frequent isolates were those with 11 virulences (24 isolates); 20

isolates contained 14 virulences and 17 isolates 13 virulences. The lowest complexity value of seven was contained by four isolates. Pathotype 21751773 had the highest complexity value of 16. The virulence complexity of isolates in individual populations was 10.54 at north, 10.94 at central, 12.54 at southeast, and 12.92 at south regions (Table 11). The mean virulence complexity for the whole population was 11.72. The maximum theoretically possible value in this study would be 24.

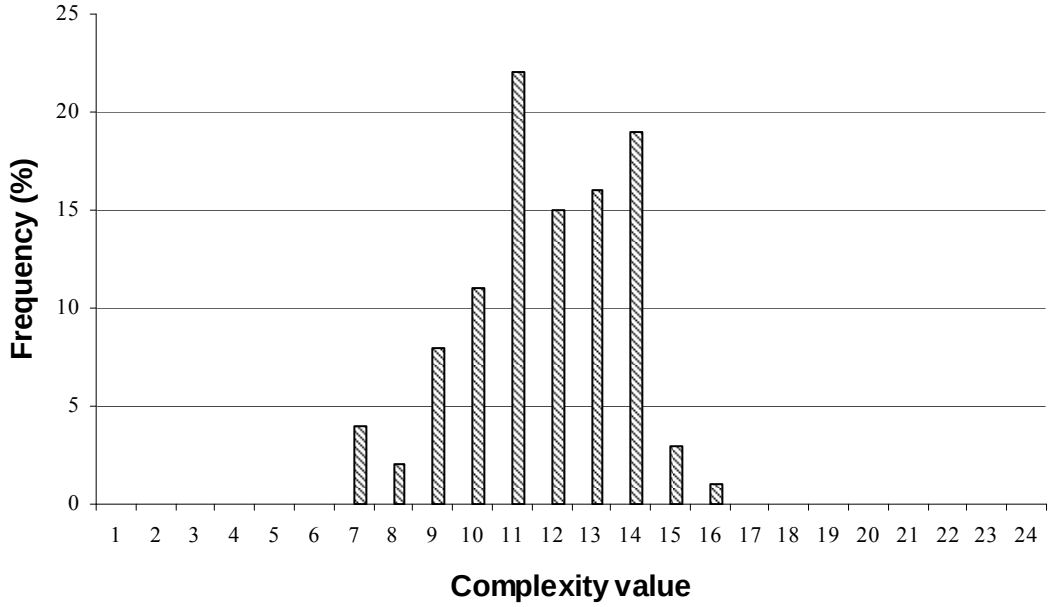

Fig. 3. Distribution of complexity (number of virulence loci per isolate) for the samples collected from north, central, south and southeast Ethiopia in 2005.

Table 9. Pathotype frequency (%) of *Puccinia striiformis* f. sp. *tritici* from Ethiopia in 2005 grouped according to the number of virulence gene and frequency of occurrence.

Number of virulence genes									
7	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**
						21654473(5)			
				20650553(1)		01754473(3)			
				01654463(6)		21750473(3)			
				00651751(7)	20770453(4)	21670473(1)	21770473(13)		
			21650063(2)	01650473(7)	21650473(8)	61750453(1)	21750573(2		
		21650450(4)	21650461(3)	01644473(1)	01651751(2)	21750553(1)	21651771(1)	23770473(1)	
	00611701(1)	00611741(3)	21770440(6)	21750461(1)	03750361(1)	03750473(1)	23750473(1)	61770473(1)	
21650040(4)	00411351(1)	01411351(1)	20750461(1)	21650361(1)	21650553(1)	23650473(2)	61770453(3)	21670773(1)	21751773(1)

*Number of isolates are indicated in parenthesis

3.1.4. Pathogen abundance

Thirty one, 24, 26 and 26 isolate were analyzed from central, north, south and southeast regions and grouped into 7, 10, 12 and 15 distinct pathotypes, respectively (Tables 10). The most abundant pathotype in the central region was 00651751 representing 22.6% of the total isolates in that region. In the north region pathotype 01654463 represented 25.0% of the isolates. The most abundant pathotype in south (01650473) and southeast (21770473) regions account for 26.9% and 30.8% of the total isolates in their respective regions. Over all, isolate 21770473 (virulent to *Yr2, Yr4a, Yr6+2, Yr6, Yr7, Yr8, Yr9+2, Yr9, Yr27, Yr32, YrCv, YrA, YrSu,* and *YrSd*) was the most dominant pathotype and account for 12.1% of the total pathotype population (Tables 10). The ten most abundant pathotypes comprised 59.8% of the total sample.

Table 10. Numbers of pathotypes found in central, north, south and southeast regions of Ethiopia in 2005.

Pathotypes	Central	North	South	Southeast	Total	Percent
21770473			5	8	13	12.1
21650473	4		2	2	8	7.5
00651751	7				7	6.5
21654473	3	2			5	6.5
01650473			7		7	6.5
21770440	6				6	5.6
01654463		6			6	5.6
20770453	4				4	3.7
21650040	4				4	3.7
21650450		4			4	3.7
01754473	3				3	2.8
21650461		2		1	3	2.8
61770453		3			3	2.8
00611741		3			3	2.8
21750473			3		3	2.8
21750573			2		2	1.9
23650473				2	2	1.9
01651751				2	2	1.9
21650063				2	2	1.9
01644473		1			1	0.9
00611701		1			1	0.9
00411351		1			1	0.9
01411351		1			1	0.9
21670473			1		1	0.9
23750473			1		1	0.9
21751773			1		1	0.9
21651771			1		1	0.9
61750453			1		1	0.9
21750553			1		1	0.9
21670773			1		1	0.9
03750473				1	1	0.9
61770473				1	1	0.9
21750461				1	1	0.9
20750461				1	1	0.9
21650553				1	1	0.9
20650553				1	1	0.9
21650361				1	1	0.9
03750361				1	1	0.9
23770473				1	1	0.9
	7/31*	10/24	12/26	15/26	39/107	100

* indicate number of pathotypes / isolates

3.1.5. Population diversity

3.1.5.1. Diversity within population

The values of Simpson, Shannon, Gleason and Kosman diversity indices are presented in Table 11. With the exception of Simpson's diversity, which is similar in all regions, pairwise t-test comparisons among the four locations showed that pathotype populations from these regions differed substantially (P = 0.05) from each other. However, the different measures do not yield the same rank order of diversity. Gleason's and Shannon's diversity indices classified the central and southeast regions as systems having lowest and highest pathotype diversity, respectively. However the diversity indices from the southeast region and north as well as central regions have the lowest and highest pathotype diversity, respectively when these regions were compared using Kosman diversity index.

The correlation coefficients between diversity indices Shannon, Simpson, and Gleason were positive and highly significant (Table 13). Shannon's, Gleason's, Simpson's and Kosman´s diversity indices were correlated with virulence complexity between 68 to 88%. However, none of these correlations were statistically significant.

Table 11. Phenotypic and genetic diversity within populations (locations) of *Puccinia striiformis* f. sp. *tritici* in Ethiopia, 2005.

Location	No. of isolates	Complexity	Phenotypic diversity			Genetic diversity
			Simpson (S)	Shannon (H)	Gleason (G)	Kosman diversity (H_{KW})
North	24	10.54 b	0.823±0.003	1.900± 0.013c	2.240± 0.027c	0.331± 0.003a
Central	31	10.94 b	0.814±0.003	1.781± 0.010d	1.783± 0.015d	0.301± 0.002a
South	26	12.92 a	0.824±0.003	1.945± 0.015b	2.492± 0.034b	0.210± 0.003b
Southeast	26	12.54 a	0.831±0.005	2.067± 0.021a	2.958± 0.047a	0.128± 0.002c

- Diversity indices (mean ± standard deviation) with different letters are significantly different at P = 0.05.

3.1.5.2. Diversity between populations

To analyze the diversity between the four populations (regions), phenotypic index (Rogers) and genetic index (Kosman distance) were calculated for all possible pairs (Table 12). Comparisons with t-test detected significant differences in both phenotypic and genetic diversity indices (Table 12). With few exceptions the phenotypic and genetic diversity indices measured with Roger's and Kosman´s distance resulted in a similar rank order of diversity.

The calculated correlation coefficient between Rogers and Kosman distance indices was positive and highly significant (Table 13). However, these indices accounted only 30 to 40% of the variance associated with virulence complexity and were not statistically significant (Table 13).

Table 12. Average phenotypic and genetic diversity values between regions.

Between regions[1]	Phenotypic diversity[2]		Genetic diversity[2]	
	Rogers (R)	Rank	Kosman distance (H_{KB})	Rank
C and N	0.940 ± 0.004b	4	0.131 ± 0.002b	2
C and SE	0.972 ± 0.003a	5	0.169 ± 0.003a	5
C and S	1.00 ± 0.000a	6	0.174 ± 0.002a	6
N and SE	0.937 ± 0.005b	3	0.136 ± 0.002b	3
N and S	0.931 ± 0.004b	2	0.154 ± 0.002a	4
SE and S	0.770 ± 0.068c	1	0.088 ± 0.001c	1

[1] N= north, C = central, S = south, and SE = southeast

[2] Diversity indices (mean ± standard deviation) with different letters are significantly different at P = 0.05. Data summarized from all possible pair comparisons of the four collection regions.

Table 13. Pearson correlations between virulence complexity, phenotypic and genetic diversity of *Puccinia striiformis* f. sp. *tritici* populations in Ethiopia, 2005.

				Diversity within population				**Diversity between regions**	
				Phenotypic			**Genetic**	**Phenotypic**	**Genetic**
	Mean	**SD**	**Complexity**	**Simpson (S)**	**Shannon (H)**	**Gleason (G)**	**Kosman diversity ($H_{KW)}$**	**Rogers (R)**	**Kosman distance ($H_{KB)}$**
Complexity	11.73	1.170	+1.000	+0.603	+0.680	+0.720	-0.875	+0.414	+0.319
Simpson	0.823	0.006		+1.000	+0.991**	+0.985**	-0.843	-0.349	-0.559
Shannon	1.920	0.119			+1.000	+0.998**	-0.843	-0.299	-0.479
Gleason	2.370	0.491				+1.000	-0.865	-0.299	-0.429
Kosman diversity	0.243	0.091					+1.000	+0.032	+0.041
Rogers	0.925	0.083						+1.000	+0.952**
Kosman distance	0.141	0.343							+1.000

** Correlation is significant at the 0.05 level (2-tailed), SD = standard devation

3.1.6. Cluster analysis

A dendrogram, which was constructed based on similarity matrices, did not differentiate the pathotypes in respect to the regions of collection (Fig. 4). However, group similarity was observed among isolates from the southeast: E06, E07, and E13. Likewise, isolates E01, E02, and E03, as well as E08 and E09 were similar. Four pathotypes from this region had pair similarity with isolates from the south. For instance isolates, S10 and E05, S12 and E15, as well as S09 and E08 were very similar. High distance was observed among pathotypes N07, N08, and N09 which represent the isolates from the northern region.

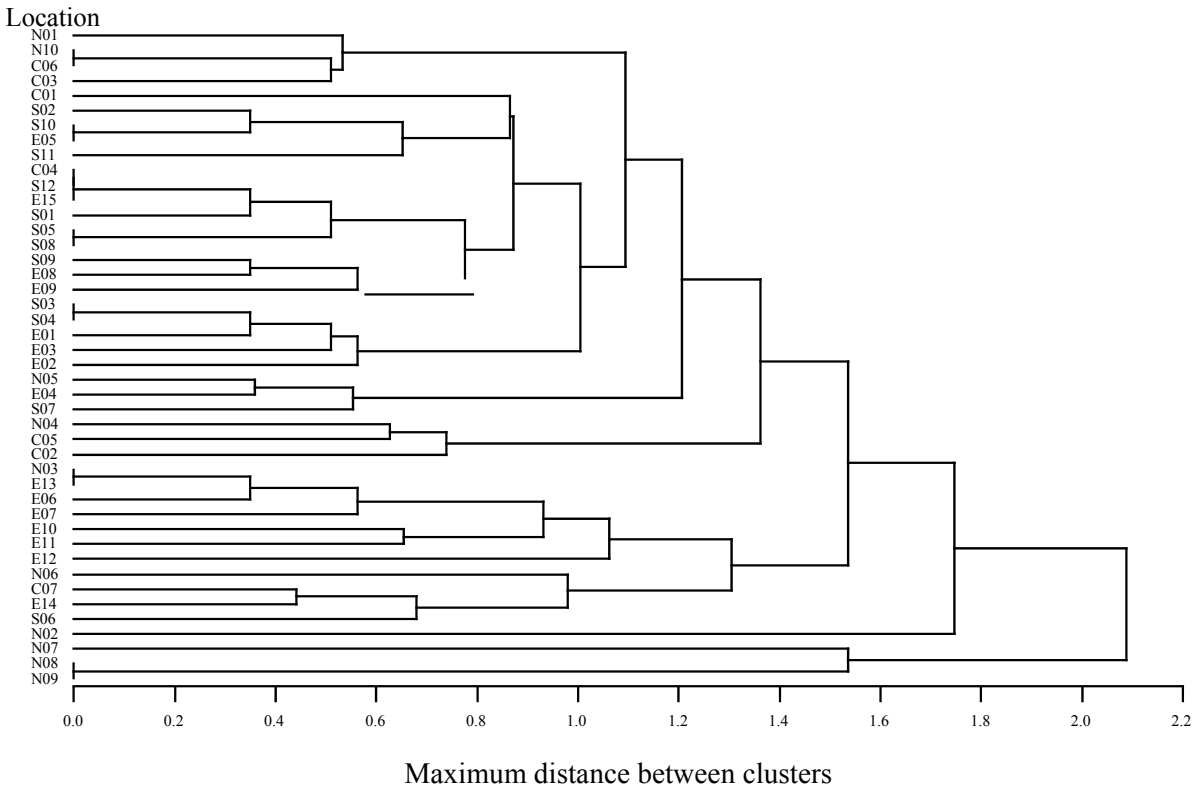

Fig. 4. Cluster analysis of pathotypes collected from the northern (N), central (C), south (S) and southeast (E) region of Ethiopia in 2005. A total of 7, 10, 12 and 15 pathotypes were used for northern, central, south and southeast regions, respectively.

3.2. Resistance of Ethiopian wheat cultivars

3.2.1. Gene postulation

The differentials, bread and durum cultivars investigated are listed in Table 14, 15, and 16 (pages 44-46), which also contain information about the resistance response spectrum and level of disease resistance under inoculated greenhouse and field conditions. All isolates were avirulent on *Yr5, Yr15* and *Yr26,* hence 21 response spectra were produced by the differential host genotypes. In addition, the cultivars gave rise to 10 additional spectra (six on bread and four durum wheats) differing from the spectra already produced by the differential genotypes which are combinations of resistance genes of the differentials.

The interpretation of the results for each cultivar was based on the isolate-specific response on the differential genotypes. Some spectra appeared to represent single *Yr* resistance genes where as others represented two or more genes. 18 out of 25 bread wheat cultivars tested were postulated to have different combinations of stripe rust resistance genes *Yr2, Yr3, Yr4, Yr6, Yr7, Yr8, Yr9, Yr27, Yr32, YrA,* and *YrSu* (Table 15) while five durum wheat cultivars were assumed to have different combinations of *Yr8, Yr32,* and *YrA* genes (Table 16). Gene *Yr2* was the most commonly encountered stripe rust resistance gene in the bread wheat cultivars. This gene was found in nine bread wheat cultivars as single gene or in combinations with other *Yr* genes. *Yr* genes *Yr1, Yr10, Yr17, Yr24, YrSd,* and *YrSp* were not found in any of the investigated cultivars. Bread wheat cultivars Wabe, Tusie, Sirbo and ET-13 were resistant to all isolates tested. Hence it was not possible to identify their source of stripe rust resistance genes with the isolates used in this study. In addition, due to the absence of matching response spectra to the tested differential genotypes, the *Yr* genes for three of the bread wheat cultivars (Megal, Suf-Omer, and Wetera) and 10 of the durum wheat cultivars could not be determined. Only two isolates were virulent on cultivars Suf-Omer and Wetera. The response spectra and the identified *Yr* genes are presented as follows.

1. ***Pavon 76:*** This cultivar consistently gave high infection types to the races that were virulent to differential genotypes *Yr6/6**Avocet *S* and *Yr7/6**Avocet *S* which are known to possess resistance genes *Yr6* and *Yr7*. However low infection types were characteristic for this cultivar when one or both of these virulence genes were absent in the isolates used for inoculation. Gene composition: *Yr6* and *Yr7*.

2. ***Mada Walabu:*** This cultivar gave high infection types whenever challenged with races that carrying combined virulence to *Yr6*, *Yr7,* and *Yr32*. Low infection types were commonly expressed in the absence of any of these genes. Gene composition: *Yr6, Yr7,* and *Yr32*.

3. ***Mitike: :*** Disease reaction of this cultivar was the same as of the differential Nord Desprez (*Yr3b*). It has compatible reaction for 14 isolates that have virulence to Nord Desprez (*Yr3b*) and incompatible reaction to six of the isolates which are avirulent on it. Gene composition: *Yr3b.*

4. ***K 6295-4A:*** The disease reaction pattern of this cultivar was the same as that of Mitike except that the three isolates which were virulent to Mitike were avirulent to K 6295-4A. The isolates were avirulent when the resistance genes *Yr2+* (Heines VII) and *Yr3b* (Nord Desprez) were present together. This implies that cultivar K 6295-4A posses *Yr2+* in addition to the gene *Yr3b* that was identified in cultivar Mitike. Gene composition: *Yr2+* and *Yr3b.*

5. ***Dereseligh and Shina:*** These two cultivars had the same disease reaction pattern as the differential genotype Kalyansona (*Yr2*). They exhibited compatible reactions to 16 races that have virulences to Kalyansona (*Yr2*) and non-compatible reactions to four of the isolates that are avirulent. Gene composition: *Yr2.*

6. ***Kubsa:*** Kubsa had the same disease reaction pattern as Dereseligh and Shina except it had avarulent reactions to the three of the isolates which were avirulent on Heines VII (*Yr2+)* and virulent on Kalyansona (*Yr2*). Gene composition: *Yr2+*

7. ***Dodota:*** This cultivar gave similar reaction pattern to the differential genotype Hybrid 46 (*Yr4a*). It has compatible reactions to six of the isolates having virulence to *Yr4a* (Hybrid 46) and low infection to 14 of the isolates which are avirulent on it. Gene composition: *Yr4a*.

8. ***K6290-Bulk and Simba:*** The infection pattern observed by these two cultivars were similar to that of the differential genotype Heines Peko (*Yr6+2*). Both cultivars showed compatible reaction to 14 isolates and avirulence to six isolates as Heines Peko (*Yr6+2*). Gene composition: *Yr6+2.*

9. ***Tura:*** Tura had high infection types to isolates which had virulent reaction to the resistance genes *Yr6+2* and *Yr9* and low infection types in the absence of one or both of the genes. Gene composition: *Yr6+2* and *Yr9.*

10. ***Abola and KBG-01:*** These two cultivars exhibited high infection type to isolates which were virulent to genes *Yr9+2* and *Yr27* and low infection type in the absence of one the genes. Gene composition: *Yr9+2* and *Yr27.*

11. **Bobicho and Hawii*:*** These cultivars exhibited compatible reactions to isolates which had combined virulence on genes *Yr4a, Yr9* and *Yr27* and non-compatible reactions to isolates that do not have combined virulence on these genes. Gene composition: *Yr4a, Yr9,* and *Yr27.*

12. Kater: Kater had the same infection pattern as the differential genotype Suwon92 x Omar (*YrSu*). It showed high infection types to 15 isolates which were also virulent on *YrSu* and low infection to five isolates which were avirulent on *YrSu*. Gene composition: *YrSu*

13. Galama and Dashen: These cultivars had the same disease reaction as the differential genotype that posses *Yr8*. The gene composition: *Yr8*

14. . Megal, Suf-Ome, and Wetera: The *Yr* genes could not be identified for these cultivars because of the non-matching reaction patterns to any of the differential genotypes.

15. Tusie, Sirbo, ET-13, and Wabe: The *Yr* genes present in these cultivars could not be postulated because of the noncompatibility of the cultivars to all the isolates tested.

B. Durum wheat

1. ***Ude:*** This cultivar had high infection types to isolates which were virulent to the resistance genes *Yr3b* (Nord Desprez) and *YrA* (Anza/Avocet ´R´) and low infection type in the absence of one of the gene. Gene composition: *Yr3b* and *YrA*.

2. ***Gerardo:*** This cultivar was susceptible to isolates which had virulence on *Yr8* and *Yr27* and resistance to all other isolates in the absence of one or both genes. Gene composition: *Yr8* and *Yr27*.

3. **Foka:** This cultivar exhibited high infection types to isolates which were virulent to *Yr32* and *YrA* and low infection type in the absence of one or both genes. Gene composition: *Yr32* and *YrA*

4. ***Bichena:*** This cultivar exhibited compatible infection types to 16 isolates which were virulent on *Yr32* and exhibited non-compatible infection types to four of the isolates which were avirulent on *Yr32*. This cultivar exhibited the same diseases reaction pattern as the differential genotype carrying *Yr32*. Gene composition: *Yr32*.

5. ***Quamy:*** This cultivar exhibited a similar reaction pattern to *Yr8* except that it was resistant to isolate 230E158. This isolate is the only isolate which indicated for the presence of *YrA*. Gene composition: *Yr8* and *YrA.*

6. ***Tob-66, DZ-04-118, Yerer, Kilinto, Ginchi, Assassa, Ld-357, Cocorit-71, Robe, and Boohai:*** These cultivars had non-matching infection patterns to any of the *Yr* genes possessed by the differential genotypes. Thus the stripe rust resistance genes of these cultivars could not be identified.

3.2.2. Adult plant resistance

To support the interpretation of the resistance genes present in the cultivars at the seedling stage all cultivars were challenged with a mixture of virulent isolates at the adult plant stage. At least one isolate was virulent to the cultivars at the seedling stage with the exception of the fully resistance cultivars (Tusie, Sirbo, ET-13, and Wabe) and Wetera. Under greenhouse conditions, the five bread wheat cultivars (Tusie, Sirbo, ET-13, Wabe and Wetera) that were resistant at the seedling stage remain resistant as expected when challenged by the same panel of isolates at the adult stage (Table 15).

Due to the prevailing non-conducive environmental condition in the season, low disease severity was observed in 2006-07. Thus, only one year data (2005) was used to asses the presence of APR in the field. The disease severity of the tested cultivars as measured by the infected leaf

area ranged between 0 to 50%. Among the bread wheat cultivars, Simba had the highest disease severity score of 50% under inoculated greenhouse test. The bread wheat cultivars: Mada walabu, Dodota, Abola, KBG-01, Bobicho, Hawii, Kater, Galama, Megal, and Suf-Omer had ≤10% disease severity under field and greenhouse conditions. Among the durum wheat cultivars, the highest terminal disease severity of 35% was recoded for Gerardo. Nine out of 15 tested durum wheat cultivars had ≤10% disease severity. These include Ude, Foka, Bichena, Tob-66, Yerer, Kilinto, Ginchi, Cocorit-71, and Robe.

Table 14. Seedling infection type (IT) of differential genotypes inoculated with 20 *Puccinia striiformis* f. sp. *tritici* isolates.

Differential genotypes	*Yr* genes	6E18 F	6E22 G	32E0 G	33E32 G	39E134 G	41E168 G	44E204 G	45E140 F	108E141 G	109E141 G	169E136 G	232E137 N	237E141 N	32E01 F	230E150 E	70E16 E	232E158 E	86E30 E	230 E30 E	70E1580 E
Yr1 / 6*Avocet S	*Yr1*	-	-	-	+	+	+	-	+	-	+	+	+	+	-	-	-	-	-	-	-
Kalyansona	*Yr2*	-	+	-	-	+	+	+	+	+	+	+	+	+	-	+	+	+	+	+	+
Heines VII	*Yr2+*	-	+	-	-	+	+	+	+	+	+	+	+	+	-	+	-	+	-	-	+
Nord Desprez	*Yr3b*	-	-	-	-	+	+	+	+	+	+	+	+	+	+	-	-	+	+	+	+
Hybrid 46	*Yr4a*	-	-	-	-	-	-	+	-	+	+	+	+	+	-	-	-	-	-	-	-
Yr5 / 6* Avocet S	*Yr5*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Heines Peko	*Yr6+2*	+	+	-	-	+	-	+	+	+	+	-	+	+	-	+	-	+	+	+	+
*Yr6 / 6*Avocet S	*Yr6*	+	+	-	-	+	-	-	+	+	+	-	+	+	+	+	+	+	+	+	+
*Yr7/ 6*Avocet S	*Yr7*	+	+	-	-	+	-	+	-	-	-	-	-	-	-	+	+	+	+	+	+
Yr8/ 6*Avocet S	*Yr8*	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	+	+	+
Yr9/ 6*Avocet S	*Yr9*	-	-	-	-	-	-	-	-	-	-	+	+	+	-	+	-	+	+	+	+
Clement	*Yr9+2*	-	-	-	-	-	-	-	-	-	-	+	+	+	-	+	-	+	-	+	+
Yr10/ 6*Avocet S	*Yr10*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-
Yr15/ 6*Avocet S	*Yr15*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Rendezvous	*Yr17*	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	+
Yr24/6*Avocet S	*Yr24*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-
Yr26/ 6*Avocet S	*Yr26*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Yr27/ 6*Avocet S	*Yr27*	-	+	-	-	-	+	-	-	+	+	+	+	-	+	+	+	+	-	+	+
Yr32/ 6*Avocet S	*Yr32*	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	-	+	-	-	-
Anza/Avocet ´R´	*YrA*	-	+	+	-	-	-	+	+	+	+	+	+	+	+	-	+	+	+	+	+
Carstens V	*YrCv*	-	-	-	+	+	+	+	+	+	-	+	+	+	+	-	-	+	+	-	-
Suwon 92 x Omar	*YrSu*	-	+	-	+	+	+	+	-	+	+	-	+	+	-	+	+	+	+	+	+
Stubes Dickkopf	*YrSd*	-	-	-	+	+	+	+	+	+	+	+	+	+	+	+	-	+	-	+	-
YrSp/ 6*Avocet S	*YrSp*	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-
Morocco (standard)	*None*	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+

- +´ represents 7-9 compatible and ´-´ represents 0-6 incompatible reactions; Standard differentials (Johnson et al. 1972) and Avocet NILs (Wellings et al. 2004); E, F, G and N represent isolates from Ethiopia, French, Germany and Dutch.

Table 15. Infection pattern, identified seedling resistance genes and level of adult plant resistance of Ethiopian bread wheat cultivars.

Bread wheat	Code	6E18	6E22	32E0	33E32	39E134	41E168	44E204	45E140	108E141	109E141	169E136	232E137	237E141	32E01	230E150	70E16	232E158	86E30	230 E158	70E150	Identified *Yr* genes	Level of adult plant resistance when challenged by virulent isolates	Field disease severity (%)	Disease severity (%) under greenhouse condition
Pavon-76	HAR 437	+	+	-	-	+	-	-	-	-	-	-	-	-	-	+	+	+	+	+	+	*Yr6, Yr7*	Low	15	20
Mada walabu	HAR 1480	+	+	-	-	+	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	*Y6, Yr7, Yr32*	High	3	0
Mitike-1709	HAR 1709	-	-	-	-	+	+	+	+	+	+	+	+	+	+	-	-	+	+	+	+	*Yr3b*	Low	10	15
K 6295-4A	HAR 515	-	-	-	-	+	+	+	+	+	+	+	+	+	-	-	-	+	-	-	+	*Yr2+, Yr3b*	Low	15	10
Dereseligh	-	-	+	-	-	+	+	+	+	+	+	+	+	+	-	+	+	+	+	+	+	*Yr2*	Low	15	20
Shina	HAR1868	-	+	-	-	+	+	+	+	+	+	+	+	+	-	+	+	+	+	+	+	*Yr2*	Low	40	10
Kubsa	HAR 1685	-	+	-	-	+	+	+	+	+	+	+	+	+	-	+	-	+	-	-	+	*Yr2+*	Low	30	20
Dodota	HAR 2508	-	-	-	-	-	-	+	-	+	+	+	+	+	-	-	-	-	-	-	-	*Yr4a*	High	10	10
K6290-Bulk	-	+	+	-	-	+	-	+	+	+	+	-	+	+	-	+	-	+	+	+	+	*Yr6+2*	Low	15	20
Simba	HAR 2536	+	+	-	-	+	-	+	+	+	+	-	+	+	-	+	-	+	+	+	+	*Yr6+2*	Low	15	50
Tura	HAR 1775	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	+	+	+	+	*Yr6+2, Yr9*	Low	15	20
Abola	HAR 1522	-	-	-	-	-	-	-	-	-	-	+	+	-	-	+	-	+	-	+	+	*Yr9+2,Yr27*	High	10	10
KBG-01	FH6-1-7A	-	-	-	-	-	-	-	-	-	-	+	+	-	-	+	-	+	-	+	+	*Yr9+2, Yr27*	High	10	5
Bobicho	HAR 2419	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	*Yr4a, Yr9, Yr27*	High	3	0
Hawii	HAR 2501	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	*Yr4a, Yr9, Yr27*	High	5	5
Kater	HAR 1899	-	+	-	+	+	+	+	-	+	+	-	+	+	-	+	+	+	+	+	+	*YrSu*	High	1	0
Galama	HAR 604	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	+	+	+	*Yr8*	High	1	0
Dashen	HAR 408	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	+	+	+	*Yr8*	Low	15	30
Megal	HAR1868	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	+	+	+	*Unknown*	High	0	5
Suf-Omer	HAR 1889	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	-	-	-	*Unknown*	High	3	5
Wetera	HAR 1920	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	*Unknown*	-	0	0
Tusie	HAR 1407	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	*Unknown*	-	0	0
Sirbo	HAR 2192	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	*Unknown*	-	0	0
ET-13	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	*Unknown*	-	0	0
Wabe	HAR 710	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	*Unknown*	-	0	0
Standard	-	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	*None*	No	60	90

- +´ represents 7-9 compatible and ´-´ represents 0-6 incompatible reactions.

Table 16. Infection pattern, identified seedling resistance genes and level of adult plant resistance of Ethiopian durum wheat cultivars.

Durum wheat	6E18	6E22	32E0	33E32	39E134	41E168	44E204	45E140	108E141	109E141	169E136	232E137	237E141	32E01	230E150	70E16	232E158	86E30	230 E158	70E150	Identified *Yr* genes	Level of adult plant resistance when challenged by virulent isolates	Field disease severity (%)	Disease severity (%) under greenhouse condition
Ude	-	-	-	-	-	-	+	+	+	+	+	+	+	+	-	-	+	+	+	+	*Yr3b, YrA*	High	1	5
Gerardo	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	-	+	+	*Yr8, Yr27*	Low	10	35
Foka	-	+	+	-	-	-	+	+	+	+	+	+	+	+	-	-	+	-	-	-	*Yr32, YrA*	High	1	10
Bichena	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	-	+	-	-	-	*Yr32*	High	5	5
Quamy	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	+	+	*Yr8, YrA*	Low	10	15
Tob-66	-	-	+	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	*Unknown*	High	1	10
DZ-04-118	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	*Unknown*	Low	15	30
Yerer	-	+	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	+	-	-	*Unknown*	High	1	0
Kilinto		+	+	-	-	-	-	-	-	+	-	+	-	+	-	-	-	+	-	-	*Unknown*	High	5	10
Ginchi	-	+	+	-	-	-	-	-	+	+	-	-	-	-	-	-	-	+	-	-	*Unknown*	High	0	10
Assassa	-	+	+	-	-	-	+	+	+	+	-	+	+	+	+	+	+	+	+	+	*Unknown*	Low	15	20
Ld-357	-	+	+	-	+	-	+	+	-	+	-	+	-	-	-	-	+	+	-	-	*Unknown*	Low	25	25
Cocorit-71	-	+	+	+	-	+	+	+	+	+	-	+	+	+	-	-	+	-	+	-	*Unknown*	High	5	10
Robe	-	-	+	-	+	-	+	-	-	+	-	+	+	+	-	-	+	-	+	+	*Unknown*	High	5	10
Boohai	-	+	+	+	+	+	+	-	+	+	-	+	-	-	-	-	-	-	+	-	*Unknown*	Low	10	20
Standard	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	*None*	No	60	90

• +´ represents 7-9 compatible and ´-´ represents 0-6 incompatible reactions.

3.3. Mapping of *Yr* genes using SSR markers

3.3.1. Inheritance of stripe rust seedling resistance genes

Figure 5 shows the distribution of the infection types of the four F_2 populations and their parents. The segregation ratios of resistant and susceptible F_2 seedling plants after being tested with stripe rust isolate 41E168 in the greenhouse are given in Table 17. All the four populations showed monogenic dominance for resistance. The calculated chi-square values given in Table 17 were not significantly different from the expected 3:1 at P = 0.05. The observed bimodal segregation patterns of the F_2 populations of the crosses between Suf-Omer x Thasos, Wetera x Thasos and Tusie x Thasos (Fig. 5A, 5B, and 5C), supported also the monogenic inheritance of the resistance genes. No clear segregation pattern of the F_2 population was observed in the cross between Wabe and Thasos (Fig. 5D).

Table 17. Segregation of resistance to susceptibility of the F_2 populations tested with the stripe rust isolate 41E168.

Cross	Number of F_2 plants			
	Resistant Infection type 0-5	Susceptible Infection type 6-9	Total	$\chi^2_{3:1}$
Suf-Omer x Thasos	76	23	99	0.17
Wetera x Thasos	126	38	164	0.29
Tusie x Thasos	89	31	120	0.04
Wabe x Thasos	103	39	142	0.46

*$\chi^2_{0.05} = 3.84$.

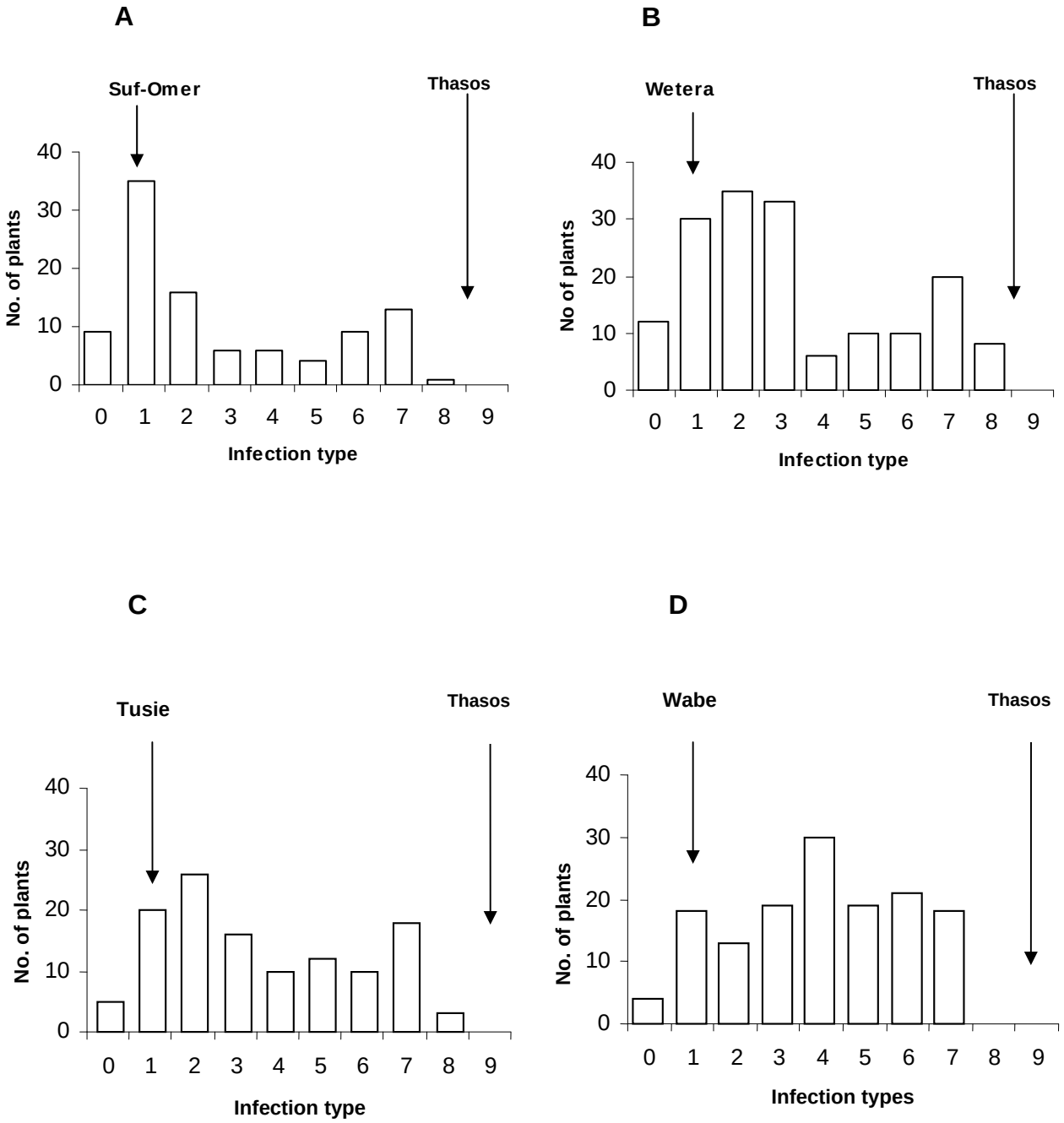

Fig. 5. Segregation patterns of F_2 plants for seedling resistance test of stripe rust isolate 41E168 Suf-Omer x Thasos, (**B**) Wetera x Thasos, (**C**) Tusie x Thasos and (**D**) Wabe x Thasos. The arrows indicate the infection types of the resistant and susceptible parents (0-5 = resistant and 6-9 = susceptible).

3.3.2. Linkage analysis and gene mapping

Polymorphism tests of the resistant and susceptible parents showed that, depending on the cross, 20-27% of markers were polymorphic. Tests on the susceptible and resistant DNA pools did not result in clear linkages. Therefore, polymorphic markers were individually screened on 10 resistant and 10 susceptible single F_2 plant groups. The markers for which linkage to the target gene was confirmed were genotyped on the respective entire mapping populations. The genetic map and the recombination values between the *Yr* genes and the SSR markers are given in Fig. 6 and Table 18, respectively. In the cultivar Suf-Omer markers loci *Xgwm181*, *Xgwm340* and *Xgwm247* on chromosome 3BL were linked to the *Yr* gene with recombination rates of 4.4%, 7.8% and 15.2%, respectively. Markers loci *Xgwm759* and *Xgwm131* of chromosome 1BL were linked to the *Yr* gene in the cultivar Wetera with recombination rates of 4.2% and 6.8%, respectively. Marker *Xgwm577* was linked with a recombination rate of 14.7% to the *Yr* gene in cultivar Wabe, this marker is located on the long arm of chromosome 7B. The other linked marker was *Xgwms238* which has a recombination frequency of 28%.

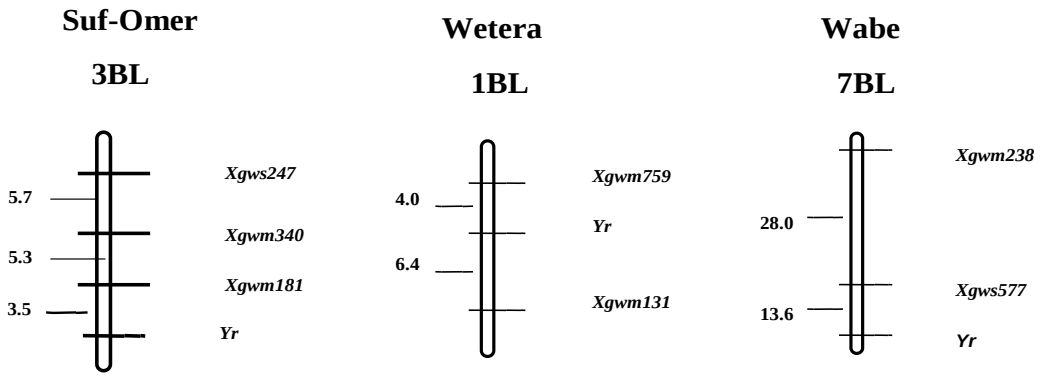

Fig. 6. Genetic maps of chromosomes 3BL (Suf-Omer), 1BL (Wetera) and 7BL (Wabe) of the F_2 populations. Locus names and the corresponding locations on the genetic map are indicated on the right side. Kosambi map distances (cM) are shown on the left side.

Table 18: Recombination rates of stripe rust resistance with respect to SSR loci.

Markers	Cross	Chromosome	Recombination rate	LOD score
Xgwm181	Suf -Omer x Thasos	3BL	0.044	13.80
Xgwm340	Suf -Omer x Thasos	3BL	0.078	10.70
Xgwm247	Suf -Omer x Thasos	3BL	0.152	6.07
Xgwm759	Wetera x Thasos	1BL	0.042	25.69
Xgwm131	Wetera x Thasos	1BL	0.068	21.07
Xgwm577	Wabe x Thasos	7BL	0.147	9.59
Xgwm238	Wabe x Thasos	7BL	0.288	4.37

4. DISCUSSION

4.1. Population structure of *Puccinia striiformis* f. sp. *tritici*

Ethiopian pathotypes carrying virulence to one or more of the following known stripe rust genes were identified in this study: *Yr2+, Yr3a, Yr3b, Yr4a, Y6, Yr6+2, Yr7, Yr8, Yr9, Yr9+2, Yr10, Yr17, Yr24, Yr26, Yr27, Yr32, YrA, YrCv, YrSd,* and *YrSu.* Virulence to *Yr17* had not been detected in Ethiopia until 2005, although virulence frequency close to 100% has been reported for this gene in some Northern European countries (Villaral et al. 2002). In the present study 14% mean virulence frequency to *Yr17* was observed. Genes *Yr8, YrCv* and *YrSu* had high virulence frequency, although they were characterised as effective source of resistance against stripe rust in Ethiopia in previous studies (Yahyaoui et al. 2000). This difference suggests drastic pathotype shift that occurred between 2000 and 2005. Other isolates carrying virulence to *Yr2+, Yr3a, Yr3b, Yr4a, Y6, Yr6+2, Yr7, Yr8, Yr9, Yr9+2, Yr10, Yr24, Yr26, Yr27, Yr32, YrA,* and *YrSd* identified in this study were not unique to Ethiopia. For instance, virulence to *Yr2, Yr6*, *Yr7,* and *Yr9* occur in most wheat producing areas of the world (Chen et al. 2002). In Germany virulence has been reported to *Yr* genes *Yr2*, *Yr3*, *Yr4*, *Yr6, Yr7, Yr8, Yr9, Yr17*, *YrCv*, *YrSu*, and *YrSd* (Flath and Bartels 2002).

Yr1, Yr5, Yr15, and *YrSp* stripe rust resistance genes were found effective against all isolates collected across the major wheat growing regions of Ethiopia. The findings were in agreement with the work of Chen (2005), Afsharri (2008) and Chunmei et al (2008) who reported that virulences to *Yr5* and *Yr15* genes rarely occur in most wheat producing areas of the world as a rare phenomenon. *Yr5* showed high resistance against all or most of the yellow rust isolates in China, North America, and Turkey (Macer 1966, Wang et al. 1996, Zeybek and Fahri 2004, Chen 2005).

On the contrary virulence close to 100% was observed to *Yr1* in Europe (Flath and Bartels 2002, Woz´niak-Strzembicka 2003). The use of these resistance genes alone or in combinations may help to reduce yield losses due to stripe rust in Ethiopia. However, the gene pyramiding approach has a clear advantage to develop commercial cultivars with durable resistance (Jacobs and Parlevliet 1993). Genes *Yr3a, Yr4a, Yr10, Yr17*, and *Yr26* confer high resistance to the tested Ethiopian isolates. *Yr* genes *Yr6+2, Yr9+2,* and *Yr24* provide moderate resistance and still can be considered for gene pyramiding in the future breeding programme. However, their low protective efficiency to the existing stripe rust population underline the urgent need to search for novel sources of resistance.

Mutations, migrations, recombinations, and direct selections can change the pathogen virulences of the rust population. By classifying pathotypes and grouping them according to the number of virulence genes present in each isolate (Tables 7 and 9),high similarity between pathotypes is observed. Thus, the pathotype with 16 virulences (21751773) differs from the pathotype with 15 virulences (21670773) by the presence of virulence to gene *Yr10.* The pathotype with 15 virulences (61770473) differs from the pathotype with 14 virulences (21770473) by the presence of the *Yr3a* virulence. The number of virulences (14) in 21770473 and 23750473 are increased by the addition of virulences to *Yr*2+9 and *Yr4a* in to pathotype 21750473 (13), respectively. Similarly the most frequent pathotype 21650473 in the sub-group with 12 virulence genes differs from pathptype 01650473 with 11 virulences by having virulence to *Yr2+*. This pathatotype differ from pathotype 21654473 with 13 virulence by having virulence to *Yr*17. Likewise, pathotype 21750473 increased to 14 by the addition of virulence to *YrCv* in 21750573. The difference among pathotypes by a single *Yr* gene might explains a single step mutation pattern.

There were remarkable differences in the composition of pathotypes in different regions (Table 10). Out of the 39 pathotypes, only four were detected in two or three locations. The most

frequent pathotype in the south (01650473) was not found at any otherregion. Similarly the two most frequent pathotypes in the central region (00651751 and 21770440) were not detected in the other threeregions. The most frequent pathotype in the north (01654463) was not identified at any other location. The most abundant pathotype (21770473) with 12.1% was found in the south and southeast regions. The regional differences in the composition of pathotypes indicate that the wheat improvement programme may capitalize on strategies that target these regional pathotype populations' differences to developed commercial cultivars for specific regions. However, in view of small sample size used further studies with more locations is essential. In addition the composition of pathogen populations can change through time and this can also be an important consideration for breeding programs. In this regard future studies should focus on special and temporal dynamics of stripe population within and between years in the country.

In plant pathogen populations that are subject to continuous directional selection from increasing virulence, it is inevitable that the most complex pathotypes will eventually predominate if no other selective factors are present. In the present study pathotype with a high complexity value of 14 is more frequent. The lowest complexity value of 0 to 6 is not contained by any pathotypes (Table 9). These results are in agreement with the work of Chen (2005) who found in recent years the predominance of *P. striiformis* f. sp. *tritici* with a wide virulence spectrum compared to those with a narrow virulence spectrum in North America. However, this phenomenon does not support the general concept that isolates with fewer virulence genes are more aggressive and have better fitness than isolates with more virulence genes (Vanderplank 1963, Line and Qayoum 1992). This concept was later demonstrated with *P. striiformis* f. sp. *hordi* in North America (Chen 2004). The predominance of a wider or narrow virulence spectrum is probably more influenced by selection pressure from the genotypes of the host population at a specific location. Chen (2005) reported that, if the host population contains relatively few resistance genes, races with only the virulence genes to match these resistance genes should be favoured by

selection and therefore, should tend to be predominant. In the contrary, if the host population contains many resistance genes, races capable of overcoming more of these genes should become predominant.

Significant phenotypic and genetic differences were detected within and between populations of different regions (Table 11 and 12). However, within populations, the genetic index (Kosman diversity) was negatively correlated with the phenotypic diversity indices (Shannon's, Simpson's and Gleason's) and hence did not yield the same rank order of diversity. The southeast region which had the highest Shannon and Gleason diversity indices was rated as lowest divers system with Kosman diversity. Similarly the isolates from the north and central regions which were characterized as the most diverse with Kosman diversity index received the lowest Shannon's and Gleason's diversity values. A general lack of significant correlations between phenotypic and genetic diversities within populations is in part attributed to the differences in procedures followed to determine each diversity index. For instance all phenotypic indices are calculated from occurrence and frequency of different pathotypes in each population, regardless of how many virulence factors the phenotypes share in common. The Kosman diversity index on the other hand take into account the number of virulence factors the isolates share in common in addition to the relative frequencies of different pathotypes in each population. Our results are in agreement with the work of Manisterski et. al. (2000) who found high correlation between Shannon´s and Simpson index values but not to the Kosman diversity index. In a similar study, Andrivon and de Vallavielle Pope (1993) found a general lack of significant correlation between differences in complexity and diversity as measured by the Shannon diversity in three populations of *Erysiphe gramins*, the causal agent of powdery mildew on barley in France. Andrivon and de Vallavielle Pope (1995) also found a general lack of correlation between virulence complexity and diversity for other rust and mildew diseases.

´Within´ diversity comparison, pathotypes from the north and central regions had higher genetic diversity (Kosman diversity index) than pathotypes from the south and southeast regions. This may be attributed to the cultivation of both indigenous landrace cultivars and commercial durum wheat cultivars along with bread wheat cultivars in the central and northern regions of the country. Durum wheat genotypes in these regions are known to have high genetic diversity (Tessema and Belay 1991) and hence, isolates with high complimentary genetic diversity co-exist with a wide range of genotypes. Manisterski et. al. (2000) have noted strong influence of genes in cultivated wheat cultivars, landraces and wild and relative species on diversity of virulence in a pathogen population of *Puccinia recondita* f. sp. *tritici* in Israel.

Unlike the north and central regions, genetically low diverse pathotypes were observed from the south and southeast regions. This could be ascribed to the cultivation of wheat cultivars with a low degree of genetic diversity. In these regions, the large-scale of semi-commercial bread wheat monocropping system is the dominating mode of production and thus time has played a significant role in reducing crop diversity in farmers' fields (Ensermu et al. 1998). A recent disease survey in the south and southeast regions showed that 56% of the small-scale farmers grow two major bread wheat cultivars, Kubsa and Galama (Temesgen, Pers. comm.). These regions account for more than 75% of the total area under bread wheat cultivation in the country (Ensermu et al. 1998). Most of the bread wheat cultivars in the country lack adequate genetic variation against stripe rust resistance because they have the same genetic background (Gebre-Mariam 1991, Badebo 2002). Selection by growing cultivars with a similar genetic background induces shift of pathotypes and increases pathogen complexity (Müller et al. 1996).

A maximum Rogers index value of 1.00 between the central and the south regions indicates that no pathotype was common between these two regions. Unlike the durum wheat belt of the central region the south wheat belt is commonly occupied by bread wheat cultivars. The lowest

Rogers index value of 0.77 was observed for populations from the south and southeast which have two pathotypes in common (Table 10). The lowest genetic distance was also measured with Kosman distance index was obtained from these two locations. This was also demonstrated by the cluster analysis that indicated group similarity within and between pathotypes from these two regions. This may suggest that the stripe rust population present in these two regions have high adaptation to *T. aestivum* commercial cultivars which are grown under both small-scale and commercial state farmers.

4.2. Gene postulation

Resistance-specificity of the host, as expressed by distinct qualitative disease reactions on seedlings, i.e. infection types (IT), when challenged by a series of pathogen isolates, has often formed the basis for genetic analysis and gene postulation of both the host and the pathogen (Day 1974, Jensen et al. 1992, Johnson and Knott 1992). To postulate the genes from the cultivars, in the present study cultivars and differential genotypes were challenged with 20 stripe rust isolates at the seedling stage. The set of the isolates represents different origins and virulence spectra, and proven to be effective in discriminating different sources of resistance, thereby providing useful information for the current wheat breeding programmes. The 18 bread wheat cultivars tested were postulated to have stripe rust resistance genes *Yr2, Yr3a, Yr4a, Yr6, Yr7, Yr8, Yr9, Yr27, Yr32, YrA,* and *YrSu* in different combinations while the five durum wheat cultivars were assumed to have genes *Yr3a*, *Yr8, Yr32, Yr27*, and *YrA.* The most commonly encountered stripe rust resistance gene in the bread wheat cultivars was *Yr2.* This gene was present in eight of the bread wheat cultivars alone or in combinations with additional genes. Gene *Yr2* introduced through ´Kalyansona´, is widely present in the CIMMYT derived germplasm (Rajaram et al. 1983). According to this study, *Yr2* is known not to provide enough protection against a wide range of pathogen genotypes indicating the need for searching more effective stripe rust resistance genes to be incorporated in Ethiopian bread wheat cultivars.

Although *Yr* genes *Yr1, Yr5, Yr15*, and *Yr17* found to be effective under Ethiopian conditions, there is no indication for the presence of these genes in Ethiopian wheat cultivars. By contrast, Hovmøller (2007) identified genes *Yr1, Yr15,* and *Yr17* in European wheat cultivars.

The validity of host-pathogen interaction data strongly depends on large prerequisites, which include correct identity and purity of the seed stocks, pure and well-characterized pathogen isolates, appropriate experimental conditions for temperature, humidity and light, to which the yellow rust/wheat interaction may be sensitive (Stubbs 1967), and correct interpretation of the disease reaction in terms of compatibility or incompatibility. Here, the purity of the seed samples was insured by carrying out homogeneity test using 6 SSR markers that were selected based on their amplifications on the different regions of the wheat chromosomes (Appendix 4). There was no perfect uniformity in 5 out of 8 varieties for at least one marker. In addition the identified genes *Yr6* and *Yr7* in the cultivar Pavon-76 that was confirmed also by earlier work of Dubin et al (1989) and Badebo et al. (1990) demonstrated the validity of the gene postulation. (Where is that shown ???)

The stripe rust resistance genes *Yr5, Yr15*, and *Yr26* were non-compatible to all the isolates tested which makes it difficult to postulate these genes in the cultivars Tusie, Sirbo, ET-13, and Wabe which had also resistance genes effective against all isolates tested. However the molecular analysis revealed that the mapped *Yr* genes on chromosomes 7BL in cultivars Wabe confirmed that the resistance in this cultivar is governed by different resistance gene other than *Yr5* (2BL), *Yr15* (1BL), and *Yr26* (1BS). The resistance genes were not identified for three of the bread (Wetera, Megal, and Suf-Omer) and ten of the durum wheat cultivars due to the lack of matching reaction patterns with any of the differential genotypes used for the gene postulation.

4.3. Adult plant resistance (APR)

In the case of *P. striiformis* on wheat, where other sources of host resistance are expressed at the adult plant stages (Johnson 1984), percentage of leaf area infected in single-isolate inoculated field trails have been used as a basis for describing the presence of adult plant resistance (Priestley et al. 1984). In the present study all the cultivars were challenged with a mixture of four virulent isolates. At least one isolate was virulent to the cultivars at the seedling stage except to the four cultivars that were non-compatible to all the isolates tested. The high disease severity (60-90%) which was observed on the susceptible check both in the greenhouse and field, indicates that the infection level was high enough to asses the resistance in the cultivars tested. Cultivars that had ≤10% leaf area infection were regarded as caryying adult plant resistance genes (Johnson 1993). In the present study, the bread wheat cultivars Mada walabu, Dodata, Abola, KBG-01, Bobicho, Hawii, Kater, Galama, Megal, and Suf-Omer had ≤10% disease severity, indicats for the presence of APR genes in these cultivars. The bread wheat cultivars Dereseligh (*Yr2*), Kubsa (*Yr2+*), K6290-Bulk (*Yr6+2*), Simba (*Yr6+2*), Tura (*Yr6, Yr9*) and Dashen (*Y8*) exhibited 20% or more disease severity. This threshold level indicates, APR in these cultivars does not play a major role in keeping the seedling stage resistance at the adult stage.

On the other hand, the durum wheat cultivars were susceptible at seedling stage to most of the races tested but about 60% of the cultivars have less than 10% disease severity when artificially inoculated with the same set of isolates at adult stage. This may suggest unlike bread wheat cultivars that stripe rust resistance in durum wheat cultivars is mainly governed by adult plant resistance genes.

4.4. Mapping of *Yr* genes using microsatellite markers (SSR)

Following discovery of the Flor´s gene-for-gene relationship between the hosts and the pathogen genotypes, breeding and use of resistant varieties underwent enormous progress. Using this method new stripe rust race-specific resistance genes were identified and transferred to breeding populations. Recently, resistance genes have been identified and mapped from the host plants using molecular markers. Molecular markers linked with stripe rust resistance genes were reported for *Yr5* (Chen et al. 2003), *Yr9 (*Shi et al. 2001*), Yr10* (Wang et al. 2002), *Yr15* (Peng et al. 2000), *Yr17* (Seah et al. 2001), *Yr18* (Suenaga et al. 2003), *Yr24* (Zakari et al. 2003), *Yr26* (Ma et al. 2001), *Yr28* (Singh et al. 2000), *Yr32 (*Eriksen et al. 2004), *Yr33* and *Yr34* (McIntosh et al. 2004), *YrH52* (Peng et al. 2000), and *Yrns-B1* (Börner et al. 2000). Closely linked markers can be used for marker-assisted selection (Chen 2005). In the present investigation, stripe rust resistance genes were mapped on the long arms of wheat chromosomes 3B (Suf-Omer), 1BL (Wetera) and 7BL (Wabe) using SSR markers. The only seedling resistance gene for stripe rust resistance so far mapped on chromosome 3B is *YrSte2*. The chromosome arm of *YrSte2* which is identified from Stephens has not been determined (Chen 2005, McIntosh et al. 2006).

Yr3a, Yr3b and *Yr3c* were identified on chromosome 1B although their specific positions were not yet determined. Macer (1975) and Peng et al. (2000) were able to map seedling resistance genes *Yr9* and *Yr15* on the long arm of chromosome 1B for the wheat genotype Riebesel and *T. dicoccoides,* respectively. Chen et al. (1995) and Singh et al. (2005) mapped adult plant resistance gene *Yr21* and *Yr29.* The chromosomal location for *Yr21* was not given while *Yr29* was mapped on the long arm of 1B. In the current study, the races that were virulent on *Yr3a, Yr3b,* and *Yr9* at the seedling stage, were avirulent on Wetera. On the contrary, two of the races which were virulent on Wetera were avirulent to *Yr15.* Hence the *Yr* genes identified in Wetera might be different from these genes. However, allellism test with the donor parents Capelle-Desprez (*Yr3a*), Hybrid 46 (*Yr3b*) and *Yr9*/6*Avocet S (*Yr9*) is a must to confirm this.

Chen (2005) reported the seedling resistance gene *Yr6* and an adult plant resistance gene *Yr39* on the short and long arm of chromosome 7B, respectively. In a similar study, Lupton and Macer (1962) as well as Chen et al. (1995) identified the seedling resistance gene *Yr2* on chromosome 7B from the variety Heines VII. According to the present seedling resistance test, all the isolates which were virulent on Heines VII were avirulent on Wabe. Therefore, the identified *Yr* gene from this cultivar in the long arm of chromosome 7B might be different from the mapped *Yr2* gene. However, it is suggested to conduct further allelism test, by crossing the source cultivars of the present study with the donor parent of the *Yr2* (Heines VII) to further differentiate the genes.

The identified resistance genes from cultivar Suf-Omer, Wetera, and Wabe confer high resistance to the tested isolates. However, further validation of the identity potential value of these genes is crucially important. The markers *Xgwm181* and *Xgwm340* closely linked to *Yr* gene in Suf-Omer may be useful in pyramiding this gene with other stripe rust resistance genes. Molecular markers for *Yr5* and *Yr15* are currently being used to combine these two genes, each of which confers resistance to all *P. striiformis* f. sp. *tritici* races in North America (Chen 2005). The recombination frequency between the markers and resistance genes in Wabe are too large to be used in marker-assisted selection.

5. SUMMARY

Yellow rust, caused by the obligate pathogen *Puccinia striiformis* f. sp. *tritici*, is one of the most important diseases of wheat worldwide. In Ethiopia, in years of epidemics which occur at intervals of four to five years, the grain yield loss in susceptible common wheat cultivars ranges between 30 to 69%.

Despite frequent stripe rust epidemics in Ethiopia, there have been not enough studies on the genetic basis of resistant wheat cultivars and the structures of pathogen population. In the present study genetic structure of wheat cultivars and *P. striiformis* population that naturally occur in the major wheat growing regions of Ethiopia were analyzed and results were discussed in the context of wheat breeding to control stripe rust in the country. The summary of the major findings are outlined as follows.

1. Virulence and diversity of the Ethiopian stripe rust population were studied using differential genotypes with known *Yr* genes. The total 107 isolates were classified into 39 different pathotypes (physiological races). There were marked differences in the composition of individual sub-populations in the four tested regions of Ethiopia. Out of the 39 different pathotypes, only four of them occurred in more than one region.

2. Significant phenotypic and genetic differences were detected within and between populations in respect to the regions of collections. No common pathotype was shared by these regions. This regional variation of the pathotypes might indicate that wheat cultivars could have been developed and released for production on a regional basis.

3. The virulence gene frequency, which is the measure of the effectiveness of resistance genes, ranged from 0 to 100%, while the virulence complexity (number of the virulent genes out of 24) varied from 7 to 16.
    - *Yr1, Yr5, Yr15,* and *YrSp* stripe rust resistance genes were found to be effective against all isolates. The use of these genes alone or in combination in the future breeding programme could help to reduce yield losses due to stripe rust. However, the gene pyramiding approach has a clear advantage to develop cultivars with durable resistance.
    - Genes *Yr3a, Yr4a, Yr10, Yr17*, and *Yr26* confer high resistance, genes *Yr6+2, Yr9+2,* and *Yr24* provide moderate resistance and can still be considered for the gene pyramiding in the future breeding programme. However, the existence of virulent pathotypes in the stripe rust population underline the urgent need to search for other sources of resistance.
    - Genes *Yr8, YrCv,* and *YrSu*, which have been characterised as effective source of resistance against stripe rust in Ethiopia in 2000 showed high virulence frequency (63-99%) which may suggest drastic shift of pathotype within the population that occurred between 2000 and 2005.

4. The stripe rust resistance genes from 40 Ethiopian commercial bread and durum wheat cultivars were identified following both the classical and molecular approach. In the classical approach, the *Yr* genes from the cultivars were postulated by comparing their reaction patterns with those of the differential genotypes to 20 stripe rust isolates.
    - The 18 bread wheat cultivars tested were postulated to have stripe rust resistance genes *Yr2, Yr3b, Yr4, Yr6, Yr7, Yr8, Yr9, Yr27, Yr32, YrA,* and *YrSu* singly or in

different combinations while the five durum wheat cultivars were assumed to have genes *Yr3b, Yr8, Yr32, Yr27,* and *YrA.*

- The *Yr2* gene was the most commonly encountered stripe rust resistance gene either as a single or in combination with other *Yr* genes in nine of the bread wheat cultivars investigated. This gene, however, provided insufficient protection against a wide range of pathogen isolates collected across the major wheat growing regions of the country. Thus, replacing these cultivars with more effective genes is necessary.
- Virulent pathotypes have been already observed to most of the *Yr* genes used in Ethiopian wheat cultivars. Thus, searching for new effective sources of stripe rust resistance genes is necessary to cope with changes in the host-pathogen interaction.
- Microsatellites (simple sequence repeats, SSRs) are applied to map the resistance genes from three resistant bread wheat cultivars.
- The *Yr* gene in cultivar Suf-Omer is closely linked to markers *Xgwm181* and *Xgwm340* on chromosome 3BL. Marker loci *Xgwm759* and *Xgwm131* of chromosome 1BL are closely linked to the *Yr* gene in the cultivar Wetera. *Xgwm577* and *Xgwm238* on chromosome 7BL are linked the *Yr* gene in cultivar Wabe.
- The identified resistance genes confer high resistance to the tested isolates. However, further validation of the identity potential value of these genes is necessary.
- The identified closely linked markers *Xgwm181* and *Xgwm340* to the *Yr* gene in Suf-Omer may be helpful as detectable markers in pyramiding this gene with other stripe rust resistance genes.

6. ZUSAMMENFASSUNG

Gelbrost wird durch das obligate Pathogen *Puccinia striiformis* f. sp. *tritici* verursacht und ist eine der bedeutendsten Weizenkrankheiten weltweit. In Äthiopien treten Gelbrostepidemien alle vier bis fünf Jahre auf und verursachen bei anfälligen Weizensorten Kornertragsverluste zwischen 30 und 69%.

Trotz häufiger Gelbrostepidemien gibt es in Äthiopien nicht genügend Untersuchungen über die genetische Basis resistenter Weizensorten und die Struktur von Pathogenpopulationen. In der vorliegenden Arbeit werden die genetische Struktur von Weizensorten und die in den äthiopischen Hauptanbaugebieten natürlich vorkommenden *P. striiformis*-Populationen analysiert. Die Ergebnisse werden im Zusammenhang mit der Weizenzüchtung zur Bekämpfung von Gelbrost diskutiert und wie folgt zusammengefasst:

1. Virulenz und Diversität der äthiopischen Gelbrostpopulation wurden mit Differenzialgenotypen untersucht, die über bekannte *Yr*-Gene verfügen. Die insgesamt 107 Isolate wurden 39 unterschiedlichen Pathotypen (physiologischen Rassen) zugeordnet. Es zeigten sich erhebliche Unterschiede in der Beschaffenheit der vier getesteten Regionen Äthiopiens. Von den 39 unterschiedlichen Pathotypen traten nur vier in mehr als einer Region auf.

2. Signifikante phänotypische und genetische Unterschiede hinsichtlich der Regionen, in denen gesammelt wurde, konnten sowohl innerhalb als auch zwischen den Populationen beobachtet werden. Es gab keinen gemeinsamen Pathotyp in diesen Regionen. Aufgrund dieser Unterschiede wären regionalspezifische Gelbrost-Zuchtprogramme erwägenswert.

3. Während die Virulenzhäufigkeit als Maß für die Effektivität von Resistenzgenen zwischen 0 und 100% variierte, betrug die Virulenzkomplexität (Anzahl Virulenzgene pro Isolat) zwischen 7 und 16.

   - Die Gelbrostresistenzgene *Yr1*, *Yr5*, *Yr15* und *YrSp* erwiesen sich als resistent gegen alle Isolate. Die Nutzung dieser Gene und deren Kombinationen im zukünftigen Zuchtprogramm könnte die durch Gelbrost verursachten Ertragsausfälle reduzieren. Dabei hat die Nutzung von Genkombinationen entscheidende Vorteile hinsichtlich der Entwicklung kommerzieller Sorten mit dauerhafter Resistenz.
   - Die Gene *Yr3a, Yr4a, Yr10, Yr17* und *Yr26* bewirken hohe Resistenz, die Gene *Yr6+2, Yr9+2* und *Yr24* mäßige Resistenz und können zur Genpyramidisierung im zukünftigen Zuchtprogramm verwendet werden. Das Vorhandensein virulenter Pathotypen in der Gelbrostpopulation unterstreicht jedoch die dringende Notwendigkeit, nach neuen Resistenzquellen zu suchen.
   - Die Gene *Yr8, YrCv* und *YrSu*, die in Äthiopien im Jahr 2000 als wirksame Gelbrostresistenzquellen beschrieben wurden, zeigten im Zeitraum von 2001 bis 2005 hohe Virulenzhäufigkeiten (63-99%), die auf drastische Veränderungen der Pathotypen innerhalb der Population in diesem Zeitraum hindeuten.

4. Die Gelbrostresistenzgene von 40 äthiopischen Aestivum- und Durumweizensorten wurden sowohl mit klassischen als auch mit molekularen Methoden analysiert. Bei der klassischen Methode wurden die *Yr*-Gene der Sorten dadurch bestimmt, dass ihre Reaktionsmuster gegenüber 20 Gelbrostisolaten mit denen der Differenzialgenotypen verglichen wurden.

- Bei den 18 Aestivumweizensorten wurden die Resistenzgene *Yr2, Yr3, Yr4, Yr6, Yr7, Yr8, Yr9, Yr27, Yr32, YrA* und *YrSu* einzeln oder in unterschiedlichen Kombinationen nachgewiesen, die Durumweizensorten enthielten die Gene *Yr3a, Yr8, Yr32, Yr27* und *YrA*.
- Das Gen *Yr2* wurde am häufigsten, sowohl einzeln als auch in Kombination mit anderen *Yr*-Genen, in neun der untersuchten Aestivumweizensorten nachgewiesen. Dieses Gen bietet jedoch nur einen unzureichenden Schutz gegen die Mehrzahl der Isolate, die in den Hauptanbaugebieten des Landes gesammelt wurden. Deshalb ist es notwendig, Sorten mit diesem Gen durch Sorten mit wirksameren Resistenzgenen zu ersetzen.
- Für die Mehrzahl der in äthiopischen Weizensorten genutzten *Yr*-Gene wurden bereits virulente Pathotypen festgestellt. Deshalb ist es notwendig, nach neuen, wirksamen Gelbrost-Resistenzquellen zu suchen, um Veränderungen in der Wirt-Pathogen-Interaktion zu beherrschen.
- Für die Kartierung der Resistenzgene in drei resistenten Aestivumweizensorten wurden Mikrosatelliten (simple sequence repeats, SSRs) genutzt.
- Das *Yr*-Gen der Sorte Suf-Omer ist eng gekoppelt mit den Markern *Xgwm181* und *Xgwm340* auf Chromosom 3BL. Die Marker *Xgwm759* und *Xgwm131* auf Chromosom 1BL sind eng an das *Yr*-Gen der Sorte Wetera gekoppelt. *Xgwm577* und *Xgwm238* auf Chromosom 7BL sind mit dem *Yr*-Gen der Sorten Wabe gekoppelt.
- Die identifizierten Resistenzgene bedingen wirksame Resistenz gegen die getesteten Isolate, jedoch sind weitere Untersuchungen zur Identifizürung dieser Gene erforderlich.

- Die identifizierten, eng an das Gen von Suf-Omer gekoppelten Marker *Xgwm181* and *Xgwm340* könnten als diagnostische Marker für die Pyramidisierung dieses Gens mit anderen Gelbrostgenen eingesetzt werden.

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10. APPENDIX

Appendix 1. Number of isolates tested per sample and host cultivars used to characterize *Puccinia striiformis* population structure in 2005.

Sample no.	No. of isolates tested per sample	Location	Group	Host cultivar
1	4	Shoa	Central	LCSR20MG (BW)
2	5	Shoa	Central	BW (EWTN Rep-1 #87)
3	3	Shoa	Central	DW
4	3	Shoa	Central	DW
5	4	Shoa	Central	BW
6	4	Shoa	Central	BW
7	3	Shoa	Central	BW
8	5	Shoa	Central	BW
9	1	Arsi	South	PBW65/2*SERI.IB (BW)
10	1	Arsi	South	DW
11	1	Arsi	South	SERI.1B *2/3/Kauz *2/BOW//KAUZ
12	1	Arsi	South	HAR 1706 (BW)
13	1	Arsi	South	HAR 1018 (BW)
14	1	Arsi	South	HAR 627 (BW)
15	2	Arsi	South	Morocco (BW)
16	1	Arsi	South	HAR 1706 (BW)
17	1	Arsi	South	Abola (HAR 1522)
18	1	Arsi	South	Dashen (BW)
19	1	Arsi	South	Bonde (BW)
20	1	Arsi	South	Bonde (BW)
21	1	Arsi	South	ISR16RA (BW)
22	1	Arsi	South	W2691SR15NK (BW)
23	1	Arsi	South	Vernstein (BW)
24	1	Arsi	South	BW
25	1	Arsi	South	FHB-2-2 (BW)
26	1	Arsi	South	BW
27	1	Arsi	South	BW
28	1	Arsi	South	Abola (HAR 1522) (BW)
29	1	Arsi	South	Kubsa (BW)
30	1	Arsi	South	Abola (HAR 1522)
31	1	Arsi	South	Tropy (BW)
33	1	Arsi	South	DW
34	1	Arsi	South	Morocco (BW)
35	3	Gojam	North	BW
36	4	Gojam	North	BW
37	2	Gojam	North	Dashen (BW)
38	4	Gonder	North	Dashen (BW)
39	3	Gonder	North	BW
40	4	Wello	North	BW
41	2	Wello	North	BW (Gumede)
42	2	Wello	North	BW
43	2	Bale	Southeast	Morocco (BW)
44	2	Bale	Southeast	K1-161 (DW)
45	2	Bale	Southeast	K1-161 (DW)
46	2	Bale	Southeast	K1-161 (DW)

Sample no.	No. of isolates tested per sample	Location	Group	Host cultivar
47	2	Bale	Southeast	Avocet-S
48	3	Bale	Southeast	Kalyansona (BW)
49	1	Bale	Southeast	HAR 2505

Appendix 2. All possible pair-wise comparison of phenotypic and genetic diversity values within populations.

Locations	Complexity		Simpson		Shannon		Gleason		Kosman	
North	10.54	}^{NS}	0.823	}^{NS}	1.900	}**	2.240	}**	0.331	}^{NS}
Central	10.94		0.814		1.781		1.783		0.301	
North	10.54	}**	0.823	}^{NS}	1.900	}*	2.240	}**	0.331	}**
South	12.92		0.824		1.945		2.492		0.210	
North	10.54	}**	0.823	}^{NS}	1.900	}**	2.240	}**	0.331	}**
Southeast	12.54		0.831		2.065		2.958		0.128	
Central	10.94	}**	0.814	}^{NS}	1.781	}**	1.783	}**	0.301	}**
South	12.92		0.824		1.945		2.492		0.210	
Central	10.94	}**	0.814	}^{NS}	1.781	}**	1.783	}**	0.301	}**
Southeast	12.54		0.831		2.067		2.958		0.128	
South	12.92	}^{NS}	0.824	}^{NS}	1.945	}**	2.492	}**	0.210	}**
Southeast	12.54		0.831		2.067		2.958		0.128	

NS = statically non significant; statistically different at P< 0.05 (*) and P< 0.1 (**).

Appendix 3. Computed t-test for diversity indices between populations.

Between population[1]	Phenotypic Diversity					Genetic Diversity				
	Rogers (S)		P -Value			Kosman distance (KBm)		P -Value		
			Calculated	P < 0.1	P < 0.5			Calculated	P < 0.1	P < 0.5
CxN CxSE	0.940 0.972	**	6.4	2.68	1.93	0.131 0.169	**	10.53	2.64	1.98
CxN CxS	0.940 1.00	**	20	2.68	2	0.131 0.174	**	11.92	2.64	1.98
CxN NxSE	0.940 0.937	NS	0.05	2.68	2	0.131 0.136	NS	0.21	2.68	2.00
CxN NxS	0.940 0.931	NS	1.59	2.62	1.96	0.131 0.154	**	8.13	2.63	1.98
CxN SExS	0.940 0.770	*	2.5	2.68	2	0.131 0.088	**	19.23	2.65	2.00
CxSE CxS	0.972 1.00	**	9.3	2.68	2	0.169 0.174	NS	1.39	2.64	2.00
CxSE NxS	0.972 0.931	**	8.2	2.65	1.96	0.169 0.154	**	4.16	2.64	1.98
CxSE SExS	0.972 0.770	*	2.9	2.68	1.96	0.169 0.088	**	25.61	2.64	1.98
CxS NxSE	1.00 0.937	**	12.6	2.68	2	0.174 0.136	**	8.49	2.65	2.00
CxS NxS	1.00 0.931	**	17.25	2.68	2	0.174 0.154	**	7.07	2.63	2.00
CxS SExS	1.00 0.770	*	3.4	2.68	2	0.174 0.088	**	30.41	2.63	2.00
NxSE NxS	0.937 0.931	NS	0.9	2.68	2	0.136 0.154	*	3.18	2.63	2.00
NxSE SExS	0.937 0.770	*	2.4	2.68	2	0.136 0.088	**	21.47	2.65	2.00
NxS SExS	0.931 0.770	*	2.4	2.68	2	0.154 0.088	**	32.96	2.68	2.00

[1]N= North, C = Central, S = South, and SE = South east

NS = statically non significant; statistically different at P = 0.05 (*) and P = 0.01 (**).

Appendix 4. Assessment of uniformity of wheat cultivars at DNA microsatellite loci.

Variety	Xgws003			
	Allel A	Allel B	Heterozygous	Total no of allel
B5	24	0	0	1
B8	24	0	0	1
B11	24	0	0	1
B14	24	0	0	1
B15	23	0	0	1
B16	21	0	0	1
B17	24	0	0	1
D7	24	0	0	1
	Xgws46			
B5	23	0	0	1
B8	24	0	0	1
B11	12	2	9	2
B14	24	0	0	1
B15	24	0	0	1
B16	24	0	0	1
B17	24	0	0	1
D7	17	5	0	1
	Xgws95			
B5	24	0	0	1
B8	24	0	0	1
B11	24	0	0	1
B14	24	0	0	1
B15	13	7	4	2
B16	13	7	4	2
B17	24	0	0	1
D7	24	0	0	1
	Xgws155			
B5	24	0	0	1
B8	24	0	0	1
B11	24	0	0	1
B14	24	0	0	1
B15	24	0	0	1
B16	24	0	0	1
B17	17	4	3	2
D7	22	1	1	2
	Xgws261			
B5	13	4	7	2
B8	24	0	0	0
B11	21	1	2	2
B14	24	0	1	2
B15	24	0	0	1
B16	24	0	0	1
B17	22	1	1	2
D7	Null allel			
	Xgws325			
B5	24	0	0	1
B8	24	0	0	1
B11	24	0	0	1
B14	24	0	0	1
B15	24	0	0	1
B16	24	0	0	1
B17	24	0	0	1
D7	24	0	0	1
	Xgws389			

B5	11	7	6	2
B8	24	0	0	1
B11	24	0	0	1
B14	24	0	0	1
B15	19	1	4	2
B16	22	1	1	2
B17	24	0	0	1
D7	20	1	3	2
Xgws631				
B5	24	0	0	1
B8	24	0	0	1
B11	24	0	0	1
B14	22	1	1	2
B15	17	3	4	2
B16	24	0	0	1
B17	20	2	2	1
D7	24	0	0	1
Xgws680				
B5	24	0	0	1
B8	24	0	0	1
B11	20	3	1	2
B14	21	2	1	2
B15	24	0	0	1
B16	24	0	0	1
B17	24	0	0	1
D7	Null allel			

*B5 = KBG-01, B8 = Suf-Omer, B11 = Wetera, B14 = Galama, B16 = ET-13, B17 = Wabe, and D7 = Tob-66

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Erklärung

Hiermit erkläre ich, dass ich die Arbeit selbständig angefertigt und keine anderen als die angegebenen Hilfsmittel benutzt habe.

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