

Virulence of wheat yellow rust races and resistance genes of wheat cultivars in Ecuador

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Abstract Virulence factors of the yellow rust, *Puccinia striiformis*, populations in bread wheat were studied in Ecuador between 1973 and 2004. The number of virulence factors has increased markedly from very few in the early seventies to 16 at the end of the 90s. Isolates belonging to race OE0 seem to be the ancestor of a rapid virulence evolution of yellow rust in Ecuador. This evolution can be explained by a single step mutation pattern. Virulence to the resistance genes *Yr1*, *Yr2*, *Yr2+*, *Yr3V*, *Yr3ND*, *Yr4+*, *Yr6*, *Yr6+*, *Yr7*, *Yr7+*, *Yr9*, *Yr9+*, *Yr11*, *Yr12*, *Yr18*, *Yr24*, *Yr26* and those in the cultivars Carstens V (*YrCV*) Strubes Dickkopf (*YrSD*), Suwon92/Omar (*YrSU*), Spaldings Prolific (*YrSP*), Anza (*YrA+*) and Selkirk (*YrSK*), was identified. Virulence to *Yr5*, *Yr8*, *Yr10*, and *Yr15* was not found. Postulation of resistance genes at the seedling stage of 14 Ecuadorian wheat cultivars indicated that these cultivars carry alone or in combinations the resistance factors *Yr1*, *Yr2*, *Yr3*, *Yr6*, *Yr9* and/or other undesigned resistance factors. Yellow rust

evolution in Ecuador has been associated with deployment of these resistance genes. None of these deployed *Yr* resistance genes are effective to the present yellow rust population in Ecuador.

Keywords *Puccinia striiformis* · Quantitative resistance · Seedling resistance · Virulence pattern · Wheat · Yellow rust

Introduction

Bread wheat (*Triticum aestivum* L.) is cultivated in Ecuador mainly by small-scale farmers along the Andean highlands above 2,500 masl. The cultivated area declined from over 100,000 ha in 1969 (MAG, 1994) to 22,000 ha at the start of the 21st century (SICA et al. 2002). This is mainly due to the frequent occurrence of severe yellow rust (*P. striiformis* f. sp. *tritici* Westend) epidemics and the lack of resistant cultivars. In highly susceptible cultivars, yellow rust severity can reach 100% and can cause yield losses of up to 96 % (Ochoa 1997). In commercial plantings, the most common cultivar Cojitambo sometimes reaches disease severity levels up to 70%. Variable planting dates and presence of volunteer plants ensure presence of inoculum early in the crop cycle throughout the year. These epidemiological factors together with the cool climate of the highlands are very conducive for yellow rust development (Stubbs 1988).

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Resistance to yellow rust in most of the released cultivars is based on major resistance genes. The Ecuadorian yellow rust population has developed virulence to the corresponding *Yr* resistance genes in the commercial cultivars so far released.

In the present study we report evidence that the adaptation of the pathogen to these *Yr* genes can be explained through single step mutations.

Materials and methods

Virulence studies

Analysis of virulence was conducted in the Plant Protection Institute (IPO, now PRI), Wageningen-The Netherlands and in the Santa Catalina Experimental Station (EESC) of the National Institute of Agricultural Research (INIAP) at Quito, Ecuador. At IPO, virulence studies were conducted from 1973 to 1992, and at INIAP from 1974 to 1977 and from 1995 to 2004. In the period 1973–2001 race genotypes were identified by using the World and European differential sets as described by Johnson et al. (1972). The cultivars Kalyansona (*Yr2*), Federat*⁴/Kavkas (*Yr9*) and Anza (*YrA*) were used as supplementals between 1985 and 2001. The differentials *Triticum spelta album* and *Triticum diccoides*, carrying *Yr5* and *Yr15*, respectively, were included in the supplemental set at IPO since 1976, while at EESC these two differentials were included from 1996. In the period 2001–2004 a differential set composed of near isogenic lines (NILs) was used to identify yellow rust virulences at the EESC. These NILs carried *Yr1*, *Yr5–Yr12*, *Yr15*, *Yr17*, *Yr18*, *Yr24*, *Yr26*, *YrSP*, *YrSK* and *YrA*. The *Yr* genes 11, 12 and 18 are adult plant resistance genes and were evaluated at the adult plant stage from 2001 and 2004. This differential set did not include most of undesignated (+) genes. The isolines resulted from five to six backcrosses to the susceptible cultivar Avocet.

In 1991, at the IPO, the study was conducted under fully controlled conditions as described by Stubbs et al. (1988). Ten to 15 seedlings of each differential were grown in pots measuring 4 × 4 cm. Seedlings of each pot were inoculated

with one isolate. At INIAP, about 10 seedlings of each differential cultivar were raised in a green house with a light regime of 14 h and at an average temperature of 20°C ± 8. The 10 seedlings from each differential were inoculated 10 days after sowing and then incubated for 16 h in dark conditions at an average temperature of 14°C ± 6. After incubation the seedlings were transferred to a greenhouse with similar conditions as in the greenhouse for raising the seedlings. Both at IPO and INIAP the infection type (IT) was assessed twice using a 0–9 scale (McNeal et al. 1971). These assessments were conducted between 16 and 20 days after inoculation. Infection types were classified as Resistant (IT = 0–3), Intermediate (IT = 4–6) and Susceptible (IT = 7–9). An isolate was considered to carry virulence when the IT was a susceptible one. Depending on the number of virulences carried, the isolate got a race identification according to the binomial notation proposed by Johnson et al. (1972), from 1973 to 2001. The differentials composed of NILs were used to identify the virulence spectrum between 2001 and 2004.

Postulation studies

Resistance gene postulation was conducted in 14 bread wheat Ecuadorian cultivars. Seven races with different virulence patterns were used to inoculate the cultivars at the seedling stage. The work was conducted partly at IPO in 1991 and partly at EESC between 1996 and 2001. Plant raising, inoculation and IT evaluation were conducted as described above for race identification, at both IPO and EESC. Postulation of resistance genes was carried out as described by Dubin et al. (1989).

Results and discussion

Virulence studies

Sixty three races were identified from the 129 samples collected between 1973 and 2001, and coded according to the Johnson et al. (1972) binary notation. Virulence developed by the Ecuadorian yellow rust population during the seventies, eighties, nineties and early two

Table 1 Virulences developed in the yellow rust population of Ecuador during the decades of seventies, eighties, nineties and early 2004

| Decade | No. isolates collected | No. of races identified | <i>Yr</i> virulences | Number of virulences |
|------------------------|------------------------|-------------------------|-------------------------------------------------------------|----------------------|
| 1970s | 35 | 17 ^a | 2+, 3N, 3V, 4+, 6, 6+, 7, CV, SP, SU | 10 |
| 1980s | 31 | 10 ^a | 2, 3N, 3V, 6, 6+, 7, 7+, 9, A+, SP, SU | 11 |
| 1990s | 63 | 38 ^{a,b} | 1, 2, 2+, 3N, 3V, 4, 6, 6+, 7, 7+, 9, 9+, A+, SD, SP, SU | 16 |
| 2001–2004 ^c | 20 | - | 1, 6, 7, 9, 11, 12, 18, 24, 26, SK, A | 11 |

^aRaces identified at IPO—The Netherlands

^bRaces identified at EESC (INIAP)—Ecuador

^cEvaluation done using the NILs differential set. This differential set does not carry virulences to the *Yr* genes 2, 3, SD, SU, CV and the undesignated genes present in Johnson et al. (1972) differential sets

thousands are given in Table 1. In the seventies it was not possible to examine virulence to *Yr2*, *YrA* and *Yr9* independently from an additional unknown virulence indicated by +. The differentials that could discern these additional virulences became available in the late seventies. Virulence to the *Yr* resistance genes 2, 3N, 3V, 6, 6+, 7, SP and SU has been common since the seventies. Virulence to *YrA*+ was present in all the races studied after 1985 while virulence to *Yr9* was highly frequent since 1989. Virulence to *Yr1* was identified in 1991 and detected again in 1999 and 2001. Virulence to Strubes Dickkopf (SD) was observed in 1996 in one sample. In addition, virulence to *Yr2*+, *Yr4*+ and *YrCV* was observed only during the seventies. The differential NILs set indicated occurrence of virulence to the *Yr* genes 1, 6, 7, 9, 11, 12, 18, 24, 26, SK and A between 2001 and 2004. This differential set does not include isolines for *Yr2*, *Yr3*, *YrSD*, *YrCV* and *YrSU* nor for the undesignated genes carried by the Johnson et al. (1972) differential set. Identification of virulences to the *Yr* genes 11, 12, 18, 24, 26 and SK were possible because of the NILs differentials. These virulences might have been present already in the Ecuadorian yellow rust population before. In none of these studies virulence to *Yr5*, *Yr8*, *Yr10*, *Yr15* and *Yr17* has been identified so far in Ecuador.

The virulence patterns based on the differential set of Johnson et al. (1972) of the more common races identified in Ecuador are shown in Table 2. During the early seventies very simple races were identified and then a rapid evolution has taken place. A postulated evolutionary tree of

the Ecuadorian yellow rust population since 1973 is shown in Fig. 1. A single step mutation pattern appears to explain the development of virulences in the yellow rust population in Ecuador. Race 0E0 seems the starting point for the evolution towards complex races. During the seventies the races carrying only one virulence 2E0, 4E0, 8E0, 64E0, 0E64, 0E32 and 32E0 and their single derivatives (races with one additional virulence) were predominant. Later the virulence evolution concentrated primarily in two evolutionary branches derived from races 4E0 and 64E0 (Fig. 1). This evolutionary process has rapidly resulted in very virulent races such as 198E10 which was frequent in 1996, but it appeared to be flexible as well as the relatively simple race 7E0 evolved in 1999. Race predominance in Ecuador appears mainly associated with virulence development irrespective of isolate complexity.

Postulation studies

The resistance factors that were postulated in 14 Ecuadorian cultivars are shown in Table 3. Cultivar 150 was susceptible to most races, only race 0E0 indicates the presence of a race-specific incomplete resistance. The Cv. Miramar seems to carry *Yr2*, as it was susceptible to all races carrying virulence to *Yr2*. Cv. Antisana is assumed to carry *Yr6* due to its compatibility with races carrying virulence to *Yr6*. Cv. Tungurahua was susceptible to races carrying *Yr3* and *Yr6*; it therefore is assumed to carry *Yr3* and *Yr6*. Cv. Cotapaxi most likely carries *Yr1*, as it showed susceptible reactions to races carrying virulence

Table 2 Infection types of the 16 most frequent wheat yellow rust races evolved in Ecuador since 1973 on the 19 differential wheat cultivars and their *Yr*-factors

| Differential cultivar | <i>Yr</i> | Race/year of collection | | | | | | | | | | | | | | | |
|----------------------------------|-----------|--------------------------|-------------|-------------|--------------|--------------|--------------|--------------|---------------|---------------|---------------|---------------|---------------|----------------|-----------------|----------------|-------------|
| | | 0E0 ^a 1973 | 2E0 1974 | 6E0 1985 | 66E0 1985 | 70E0 1986 | 6E64 1990 | 6E64 1991 | 66E64 1991 | 70E64 1991 | 70E64 1991 | 14E78 1991 | 15E78 1991 | 14E142 1991 | 110E207 1996 | 198E10 1996 | 7E8 1999 |
| World set ^c | | | | | | | | | | | | | | | | | |
| Chinese 166 | 1 | R ^b | R | R | R | R | R | R | R | R | R | R | S | R | R | R | S |
| Lee | 7 | R | S | S | S | S | S | S | S | S | S | S | S | S | S | S | S |
| Heines Kolben | 6 | R | R | S | R | S | S | S | R | S | S | S | S | S | S | S | S |
| Vilmorin 23 | 3V | R | R | R | R | R | R | R | R | R | R | S | S | S | S | I | R |
| Moro | 10 | R | R | R | R | R | R | R | R | R | R | R | R | R | - | R | R |
| Strubes | SD | R | R | R | R | R | R | R | R | R | R | R | R | R | S | R | R |
| Dickkopf | | | | | | | | | | | | | | | | | |
| Suwon 92 x Omar | SO | R | R | R | S | S | R | R | S | S | S | R | R | R | S | S | R |
| Clement EUR. SET ^c | 9+ | * | * | R | R | R | R | R | R | R | R | R | R | R | R | S | R |
| Hybrid 46 | 4 | R | R | R | R | R | R | R | R | R | R | R | R | R | S | I | R |
| Reichersberg 42 | 7+ | R | R | R | R | R | R | R | R | R | R | S | S | S | S | S | R |
| Heines Peko | 6+ | R | R | R | R | R | R | R | R | R | R | S | S | S | S | R | R |
| Nord Desprez | 3N | R | R | R | R | R | R | R | R | R | R | S | S | S | S | S | S |
| Compair | 8 | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R |
| Carstens V | CV | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R |
| Spalding Prolific | SP | R | R | R | R | R | S | S | S | S | S | S | S | S | R | S | I |
| Heines VII | 2+ | R | R | R | R | R | R | R | R | R | R | R | R | S | S | I | R |
| Supplement Set | | | | | | | | | | | | | | | | | |
| Federat ⁴ /Kavkaz | 9 | * | * | * | * | * | R | S | R | R | S | R | R | R | S | S | S |
| Kalyansona | 2 | * | * | S | S | S | S | S | S | S | S | S | S | S | R | S | S |
| Anza | A+ | * | * | S | S | S | I | S | S | S | S | S | S | S | - | S | S |

^aRace designation according the binary notation proposed by Johnson et al. (1972)

^bScoring according to the 0–9 McNeal scale (1971); 0–3 resistant (R), 4–6 intermediate (I) and 7–9 susceptible (S)

^cSets proposed by Johnson et al. (1972)

*Differentials were not included yet at time of scoring

-Missing value

to *Yr1*. Cv. Cotacachi, released in 1999, seems also to carry *Yr1* as it is compatible with race 7E8, although the reaction to 15E78 is unknown. Cv. Cojitambo showed intermediate to susceptible reactions to races with virulence to *Yr9*, indicating that it carries an extra gene different from *Yr9*. The remaining cultivars appear to carry other unknown *Yr* resistance factors.

Cultivars Crespo and Amazonas grown before 1970 carry *Yr* factors different to those formally designated so far (Table 3). Isolate 0E0 used in this study was virulent on these cultivars, indicating that some 0E0 isolates identified during the early seventies carry virulences to *Yr* genes carried by local cultivars. Nevertheless, 0E0 isolates appear simple and pioneer of the virulence evolution of yellow rust in Ecuador. After the

seventies, yellow rust evolution appeared mainly associated with the formally designated *Yr* genes, although the *Yr* resistance factors carried by cvs. Chimborazo, Altar, Cojitambo and Quilindaña (Table 3) also seem to have directed the evolution of yellow rust in Ecuador. This virulence evolution evidently took place in Ecuador. Race migration from Colombia and Peru is less likely. In Colombian races virulence to *Yr2+* and *Yr4+* were very frequent in the nineties (IPO, unpublished data) while these virulences were observed in Ecuador only during the early seventies. Wheat areas in Peru are far away from Ecuadorian wheat areas which make establishment of Peruvian races unlikely.

Most of the bread wheat cultivars released in Ecuador derive from CIMMYT germplasm.

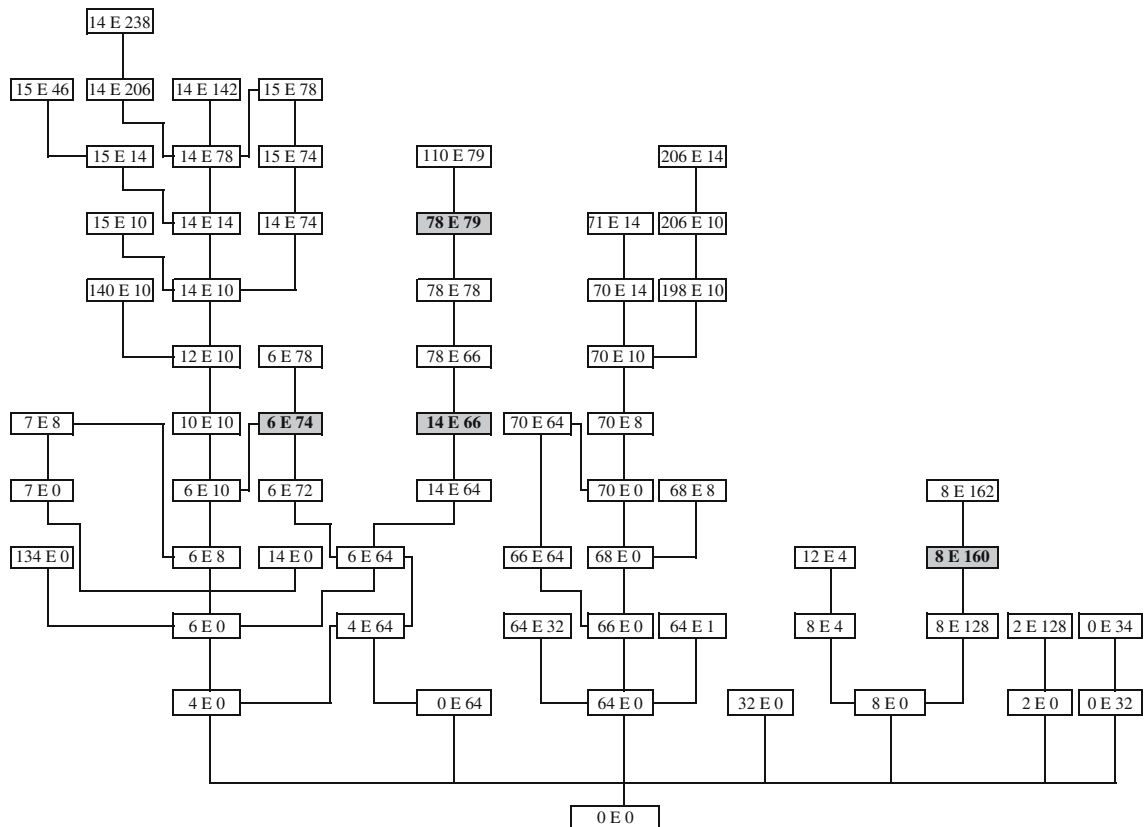


Fig. 1 Virulence evolution pattern of the Ecuadorian wheat yellow rust populations since 1973. Isolates identified as race 0E0 appeared the ancestor of all races evolved in Ecuador. Races were identified by binary notation according to Johnson et al. (1972). Races in bold and

shaded most likely escaped sampling. All races below the line with races 7E8 to 70E8 were collected during the 70's, all below the line 140E10 to 198E10 were collected during the 80's and all from the line 7E0 to 8E162 upward were collected during 90's and early 2000's

Resistance genes such as *Yr2*, *Yr3*, *Yr6*, *Yr7*, *Yr9* and *YrA+* were widely used by CIMMYT during the seventies and eighties (Dubin et al. 1989; Babebo et al. 1990; Danial et al. 1994) and have been deployed in commercial cultivars in Ecuador. *Yr9* resistance derived from rye has been introduced in CIMMYT material through the 1B/1D translocation (Zeller 1973). It has been effective in various parts of the world (Rajaram et al. 1983). In Ecuador *Yr9* lasted only a very short period like in other areas conducive to yellow rust such as in Kenya and in North Western Europe (Daniel et al. 1994). *Yr1* occurs in Ecuador only in the cvs. Cotopaxi and Cotacachi (Table 3). The presence of *Yr1* in Cotopaxi was also previously postulated by Ochoa et al. (1998).

All *Yr* resistance genes so far deployed in commercial cultivars in Ecuador are at present ineffective. The pathogen population has adapted to the deployment of *Yr* resistance genes in a very efficient way. The two most recent races found in Ecuador, 198E10 and 7E8, are collectively virulent to all resistance *Yr* genes carried by the cultivars released in Ecuador (Table 3). Such a fast pathogen adaptation will most likely continue to occur if new *Yr* genes will be deployed in the future.

Deployment of such monogenic, race specific resistance is apparently not a good solution. However, in all regions where yellow rust in wheat is a serious problem, cultivars have been found with a resistance that lasted for an extended period of time, despite being grown on a

Table 3 Yellow rust infection types (IT) for 14 cultivars released in Ecuador against seven Ecuadorian yellow rust races and their postulated *Yr* resistance gene(s)

| Cultivar | Year of release | Yellow rust races | | | | | | | <i>Yr</i> gene |
|-------------|-----------------|-------------------|-------------------|-------------------|--------------------|--------------------|---------------------|------------------|----------------|
| | | 0E0 ^a | 0E64 ^a | 4E64 ^a | 70E64 ^a | 15E78 ^a | 198E10 ^b | 7E8 ^b | |
| 150 | Lv | I ^c | S | S | S | S | S | S | – |
| Barba negra | Lv | I | S | R | S | S | S | n.d | + |
| Miramar | Lv | R | S | S | S | S | S | S | 2 |
| R. Pizan | Lv | R | R | R | R | S | S | I/S | + |
| Crespo | 1963 | S | S | S | R | S | S | S | + |
| Amazonas | 1969 | S | R | R | R | I | S | I | + |
| Antisana | 1978 | R | R | R | S | S | S | I | 6 |
| Chimborazo | 1978 | R | R | S | S | R | S | R | + |
| Tungurahua | 1982 | R | R | R | R | S | S | R | 3,6 |
| Altar | 1982 | R | R | S | S | S | I | I | + |
| Cotopaxi | 1988 | R | R | R | R | S | R | S | 1 |
| Cojitambo | 1992 | R | R | R | I | R | I | S | 9, + |
| Quilindaña | 1994 | (–) | (–) | (–) | (–) | (–) | S | R | + |
| Cotacachi | 1999 | (–) | (–) | (–) | (–) | (–) | R | S | 1 |

Race 0E0 has no known virulence; race 0E64 with virulences to *Yr* 2 and *Yr* SP; Race 4E64 with virulences to *Yr*2, *Yr*6+, *Yr*SP, *Yr*A+ and *Yr*SO; race 70E64 with virulences to *Yr*2, *Yr*6, *Yr*7, *Yr*9, *Yr*SU, *Yr*SP, *Yr*A+ and *Yr*SO; race 15E78 with virulences to *Yr*1, *Yr*2, *Yr*3V, *Yr*3N, *Yr*6, *Yr*6+, *Yr*7, *Yr*SP, *Yr*A+ and *Yr*SO; race 198E10 with virulences to *Yr*2, *Yr*3V, *Yr*3ND, *Yr*6, *Yr*7, *Yr*7+, *Yr*9, *Yr*9+, *Yr*SU and *Yr*A+; race 7E8 with virulences to *Yr*1, *Yr*2, *Yr*3N, *Yr*6, *Yr*7, *Yr*9 and *Yr*A+

^aEvaluation done at IPO. Wageningen-The Netherlands

^bEvaluation done at INIAP. Quito-Ecuador

^cIT based on a 0–9 scale (McNeal et al. 1971) whereby 0–3 (R), 4–6 (I), 7–9 (S)

Lv—Local cultivars presumably introduced by Spanish

n.d. Not tested as there was not enough seed of this old cultivar available for all tests

– no postulated factor, + = undesignated resistance factors

(–) Cultivars not available at time of evaluation

considerable area. Johnson (1981) defined that as durable resistance. One of these durably resistant cultivars, Capelle Desprez, was shown to carry resistance factors on several chromosomes (Johnson 1981). In the Netherlands the cultivar Felix remained resistant to yellow rust for over 15 years. It appeared to carry two resistance genes, *Yr*3 and *Yr*CV (Stubbs 1985). Another Dutch cultivar, Manella, kept its resistance for over 18 years (Anonymus 1958–1992). Its resistance is assumed to be complex (Johnson 1981). Van Dijk et al. (1988) investigated 15 Dutch old cultivars with durable resistance. They were released between 1901 and 1935. The level of resistance was invariably moderate to fair, but never very high. ‘Wilhelmina’ and its daughter ‘Julia’ both were grown over very large acreages for a period of nearly 50 years. The resistance never eroded. Their conclusion was that the durable resistance was of a quantitative type. In

Kenya too several cultivars remained resistant over extended periods (Danial et al. 1994). Such cultivars should be used in breeding programs. Danial et al. (1994) also observed that the cultivars of which the introduced major resistance gene became ineffective never got as susceptible as the very susceptible cultivar Morocco. There was in all these cultivars a residual resistance, which was in some cultivars of a considerable level. Apparently here too a quantitative resistance appeared widely present. Breeding for durable resistance to yellow rust should be based on such quantitative types of resistance.

Conclusion

The wheat yellow rust population in Ecuador showed a wide variation and a clear increase in the number of virulence factors. The erosion of

resistant genes to this pathogen was rapid and the formation of highly complex races was evident. The evolution of yellow rust virulence has been closely associated with *Yr* resistance gene deployment in new cultivars. Gene postulation can be a useful tool to help breeders to identify the resistance factors in the seedling stage.

Because of the complex nature of the yellow rust races, breeders are advised to select for quantitative resistance. By using cultivars with reasonable levels of quantitative resistance and cultivars with proven durable resistance in the breeding program one may obtain genotypes with good levels of durable resistance.

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References

- Anonymus (1958–1992) Beschrijvende rassenlijst voor landbouwgewassen (Descriptive lists of varieties for arable crops) nrs. 33–67 (1958–1992), Leiter-Nypels, Maastricht
- Babebo A, Stubbs RW, Van Ginkel M, Gebeyehu G (1990) Identification of resistance genes to *Puccinia striiformis* in seedlings of Ethiopian and CIMMYT bread wheat varieties and lines. *Neth J Plant Pathol* 96:199–210
- Daniel DL, Stubbs RW, Parlevliet JE (1994) Evolution of virulence patterns in yellow rust races and its implication for breeding for resistance in wheat in Kenya. *Euphytica* 80:165–170
- Dubin HJ, Stubbs RW, Johnson R (1989) Postulated genes for resistance to stripe rust in selected CIMMYT and related wheat. *Plant Dis* 73:472–475
- Johnson R (1981) Durable disease resistance. In: Jenkyn JF, Plumb RT (Eds.) *Strategies for the control of cereal disease*. Blackwell Scient. Publ, Oxford, London, Edinburgh, pp. 55–63
- Johnson R, Stubbs RW, Fuchs E, Chamberlain NH (1972) Nomenclature for physiologic races of *Puccinia striiformis* infecting wheat. *Trans Br Mycol Soc* 58:475–480
- McNeal FH, Konzak CF, Smith EP, Tate WS, Russel TS (1971) A uniform system for recording and processing cereal research data. USDA, ARS Bulletin 34–121
- Ministerio de Agricultura y Ganadería (MAG) (1994) Primer Compendio Estadístico Agropecuario del Ecuador. Granos y Cereales. División de Estadísticas Agropecuarias. Quito, Ecuador
- Ochoa J (1997) La roya amarilla del trigo en el Ecuador; aspectos epidemiológico y de resistencia. In: Daniel DL (Ed.) *Primer Taller de Resistencia Duradera en Cultivos Altos en la Zona Andina*. PREDUZA, Quito, Ecuador, pp. 45–52
- Ochoa J, Lowers J, Broers L (1998) Analysis of virulence and evolution of the Ecuadorian population of stripe rust (*Puccinia striiformis* f. sp. *tritici*) in wheat (*Triticum aestivum* L.). *Fitopatología* 33 (3):160–164
- Rajaram S, Mann ChE, Ortiz-Ferrera G, Mujeeb-Kasi A (1983). Adaptation, stability and high yield potential of certain 1B/1R CIMMYT wheats. *Proceeding sixth International. Wheat Genetics Symposium, Kyoto, Japan*, pp. 613–621
- Roelfs AP, Sing RP, Saari EE (1992) Rust diseases of wheat, Concept and methods of disease management. Mexico, D.F., CIMMYT
- SICA, INEC, MAG (2002) II Censo Nacional Agropecuario. Resultados Nacionales y Provinciales. Cultivos Transitorios. Ecuador
- Stubbs RW (1985) Stripe rust. In: Roelfs AP, Bushnell WR (Eds.) *The cereal Rusts, vol. II: diseases, distribution, epidemiology and control*. Academic Press, Orlando, London, pp. 61–101
- Stubbs RW (1988) Pathogenicity analysis of yellow (stripe) rust of wheat and its significance in a global context. In: Simmonds NW, Rajaram S (Eds.), *Breeding strategies for resistance to the rust of wheat*. Mexico, D.F., CIMMYT, pp. 23–38
- Van Dijk P, Parlevliet JE, Kema GHJ, Zeven AC, Stubbs RW (1988) Characterization of the durable resistance to yellow rust in old winter wheat cultivars in The Netherlands. *Euphytica* 38:149–158
- Zeller FJ (1973) IB/IR wheat rye chromosome substitutions and translocation. *Proc fourth Internat Genetics Symp* pp 209–222. Columbia