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Research Article

New resistance genes in the *Zea mays* - *Exserohilum turcicum* pathosystem*

Juliana Bernardi Ogliari[1], Marco Antônio Guimarães[2], Isaías Olívio Geraldi[3], and Luis Eduardo Aranha Camargo[3]

[1]*Universidade Federal de Santa Catarina, Departamento de Fitotecnia do Centro de Ciências Agrárias, Florianópolis, SC, Brazil.*
[2]*Monsanto do Brasil Ltda, Uberlândia, MG, Brazil.*
[3]*Universidade de São Paulo, Escola Superior de Agricultura "Luiz de Queiroz", Piracicaba, SP, Brazil.*

Abstract

The use of monogenic race-specific resistance is widespread for the control of maize (*Zea mays* L.) helminthosporiosis caused by *Exserohilum turcicum*. Inoculation of 18 Brazilian isolates of *E. turcicum* onto elite maize lines containing previously identified resistance genes and onto differential near-isogenic lines allowed the identification of new qualitative resistance genes. The inoculation of one selected isolate on differential near-isogenic lines, F_1 generations and a BC_1F_1 population from the referred elite lines enabled the characterization of the resistance spectrum of three new genes, one dominant (*HtP*), one recessive (*rt*) and a third with non-identified genetic action. Three physiological races of the pathogen were also identified including two with new virulence factors capable of overcoming the resistance of one of the resistance genes identified here (*rt*).

Key words: Northern leaf blight, genetic inheritance, resistance genes, races, maize.

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Introduction

Helminthosporiosis is one of the main leaf diseases of maize and is caused by *Exserohilum turcicum* Leonard & Suggs [*Helminthosporium turcicum* Pass.], which is the teleomorph of *Setosphaeria turcica* (Lutterell) Leonard & Suggs (Frederiksen, 1991) [*Trichometasphaeria turcica* Lutterell]. Temperatures between 20 and 25 °C, relative humidity from 90 to 100%, and low luminosity (Bentolila *et al*., 1991) favor the disease. Severe but sporadic epidemics occur most frequently in the southern and western regions of Brazil where they cause severe losses in yield (Esteves, 1989). Such losses may exceed 50% as a consequence of extensive leaf damage during the grain-filling period and of the greater predisposition of diseased plants to stem rot caused by *Diplodia* maydis (Raymundo and Hooker, 1981).

The genetic control of *E. turcicum* in maize can be achieved both by qualitative and quantitative resistance, used separately or together. Most of the qualitative genes such as Ht_1, Ht_2, Ht_3, *HtM*, and *HtN* (Gevers, 1975; Hooker 1961, 1963a, 1963b, 1965, 1975, 1977, 1978; Hooker, 1981; Robbins and Warren, 1993) are dominant or partially dominant but there is one example of a recessive gene identified by Carson (1995) and Carson and Wicks (1993) in a maize synthetic.

The qualitative maize resistance genes referred above have great phenotypic effect, but might be overcome by virulence genes present in specific races of the pathogen. Following a classification system proposed by Leonard *et al*. (1989), *E. turcicum* races are defined based on their phenotypic reactions when inoculated onto a set of differential maize lines. In this system, *E. turcicum* race designations are assigned according to the maize resistance genes that their virulence matches, *e.g. E. turcicum* race 0 is ineffective (avirulent) against all *Ht* genes described above whereas *E. turcicum* race 1 is only effective (virulent) against Ht_1. Both races are effective against maize genotypes lacking all resistance genes. So under this nomenclature system, the designation of these races are Ht_1, Ht_2, Ht_3, *HtN*/0 and Ht_2, Ht_3, *HtN*/Ht_1, respectively.

In North America *E. turcicum* races 0 and 1 predominate whereas 2N e 23N rarely occur (Berquist and Masias, 1974; Fallah Moghaddam and Pataky, 1994; Jordan *et al*., 1983; Lipps and Hite, 1982; Pieczarka, 1980; Smith and Kinsey, 1980; Thakur *et al*., 1989; Windes and Pedersen, 1991). In Brazil *E. turcicum* populations seems to be more diverse in terms of race composition. Gianasi *et al*. (1996), for instance, observed a predominance of race 0 but also de-

Send correspondence to Juliana Bernardi Ogliari. Rodovia Admar Gonzaga 1346, Bairro Itacorubi, 88034-001 Florianópolis, SC, Brazil. E-mail: jbogliar@cca.ufsc.br. *Part of the doctoral thesis presented by first author at Universidade de São Paulo, Escola Superior de Agricultura "Luiz de Queiróz", Piracicaba-SP, Brazil.

tected races capable of overcoming resistance conferred by Ht_1 (races 1N, 12N, and 123N).

This study was carried out to characterize the qualitative resistance genes of unknown origin from two elite maize lines. For this, the resistance reactions of these lines were compared to those of differential lines after inoculation with 18 *E. turcicum* isolates. The results allowed us to identify both new qualitative resistance genes and consequently new corresponding virulence factors.

Material and Methods

Plant material

Inbred lines L30R and L40, developed by Sementes Agroceres S/A (currently Monsanto do Brasil Ltda) possess monogenic resistance to *E. turcicum*. The L30R line was generated after six backcross cycles to the susceptible L30S line and two terminal self-pollinations (BC_6S_2) using the American L10 resistant line as donor of the resistance gene. The L30S line was developed under central Brazilian conditions (a tropical region) through self-pollinations and selections on a synthetic population of inbred maize lines derived from dent endosperm varieties from the International Maize and Wheat Improvement Center (CIMMYT). The L40 line was developed in northern Paraná state (a subtropical/tropical transition region) by self-pollinations and selections on a synthetic of intercrosses between Brazilian commercial maize hybrids and can, therefore be considered a subtropical line. A third form of L30 from the cross of L30R with L30S (L30R x L30S), the derived F_1 generations [(L30R x L40) and (L30S x L40)], and the BC_1F_1 population [(L30R x L40) x L40] were obtained to study the genetic basis of the L30R and L40 resistance. A set of near-isogenic lines each containing a different *Ht* gene was developed by A.L. Hooker from the University of Illinois and these constitute the set of differential lines used in our study. The differential inbred maize lines Pa91Ht_1Ht_1, Pa91Ht_2Ht_2, Pa91Ht_3Ht_3, and Pa91*HtNHtN* which are susceptible to *E. turcicum* races 1, 2, 3, and N, respectively, and the maize line Pa91, which is susceptible to all races (including races with no virulence factors) were used to identify the virulence genes present among the collection of *E. turcicum* isolates.

Isolation of *E. turcicum*

Pathogenic *E. turcicum* was isolated from 18 samples of infected maize leaf tissue collected in different Brazilian maize growing regions. Small dehydrated segments of diseased leaf tissue were rinsed in 70% (v/v) aqueous alcohol, transferred to 1% sodium hypochloride solution (v/v) for two minutes and then rinsed in sterile distilled water. Leaf pieces were transferred to water agar medium and incubated for five days. The pathogen was then transferred to lactose casein hydrolysate medium (LCH) (Dhingra and Sinclair, 1995). Monoconidial cultures were obtained from the LCH isolates by transferring single conidium to potato dextrose agar medium (PDA). Inoculum was produced by inoculating sterile sorghum seeds, previously embedded in water (1.5 seed:1 water; v/v) with the monoconidial isolates followed by incubation in the dark at 24 °C for three weeks.

Screening of *E. turcicum*

Eighteen *E. turcicum* isolates were inoculated onto L30R, L30S, (L30R x L30S) and L40 maize plants grown in the field in order to identify isolates capable of detecting qualitative resistance genes present in L30R and/or L40 according to lesion type. Each maize line was sown in 1m-row plots spaced 1m apart (five plants per meter) and inoculated at the four to six leaf stages by placing about 20 sorghum seeds colonized by the pathogen into the leaf whorl (Carson, 1995). Plants were evaluated for lesion type between 20 and 30 days after inoculation using the score scale described by Esteves (1989), where plants were considered resistant if they displayed chlorotic-necrotic lesions or did not display any symptoms at all and susceptible if they displayed olive green necrotic lesions.

Identification of *E. turcicum* virulence factors

Selected *E. turcicum* isolates were re-inoculated onto maize lines L30R, L30S, (L30R x L30S), and L40 as well as onto the hybrids from the crosses (L30R x L40) and (L30S x L40) and on the set of near-isogenic lines Pa91, Pa91Ht_1Ht_1, Pa91Ht_2Ht_2, Pa91Ht_3Ht_3, and Pa91*HtNHtN*. Inoculations were performed on three plants of each genotype grown in pots placed in a greenhouse under controlled humidity and temperature conditions. Inoculum production and inoculation were carried out as described above except that after inoculation, plants were incubated for one night in a humidity chamber (over 85% relative humidity) at 25 °C and then kept under natural photoperiod conditions at a temperature that varied from 13 °C up to 29 °C, this temperature range being the ideal average for race identification experiments (Leonard *et al.*, 1989). Lesion type was assessed 20 to 30 days after inoculation using the method of Esteves (1989) and the results of this assessment allowed us to characterize the virulence factors of the *E. turcicum* isolates using the classification system of Leonard *et al.* (1989).

Genetic basis of L30R and L40 resistance

The maize hybrids from the crosses (L30R x L40) and (L30S x L40) and the backcross population from [(L30R x L40) x L40] were inoculated with one isolate avirulent on L30R and L40. This experiment was carried out in the greenhouse under similar conditions as described above. In this case, inoculum preparation and inoculation were carried out with an adaptation of the method described by Simcox and Bennetzen (1993). Twenty days after inoculation, 138 BC_1F_1 plants were evaluated for lesion type each week for one month (Esteves, 1989). The disease reactions

of the BC_1F_1 plants were compared to the reactions of the near-isogenic lines of the Pa91 group as well as to the reactions of the lines L30R, L30S and L40 lines and the hybrids (L30R x L40) and (L30S x L40).

Results and Discussion

Three *E. turcicum* isolates (isolate 18 from Santa Cruz das Palmeiras and isolate 47 from Cravinhos, both from São Paulo, and isolate 49 from Bandeirantes, Paraná) were selected from the preliminary tests carried out in the field with the 18 *E. turcicum* isolates from Brazil and the L30R, L30S, (L30R x L30S) and L40 lines based on their ability to discriminate qualitative resistance genes controlling lesion type in this set of lines. The different performance of the four maize lines inoculated with the three *E. turcicum* isolates indicated the involvement of at least three resistance genes (Table 1).

Since the pathosystem studied fits the gene-to-gene model proposed by Flor (Lim *et al.*, 1974), the genetic resistance basis of L30R and L40 could be evaluated from the reaction of the maize lines Pa91, Pa91Ht_1Ht_1, Pa91Ht_2Ht_2, Pa91Ht_3Ht_3, Pa91*HtNHtN*, by the maize set L30 [L30R, L30S, and (L30R x L30S)], by the L40 line, as well as by the F_1(L30R x L40) and F_1(L30S x L40) maize hybrids when inoculated with *E. turcicum* isolates 18, 47 or 49 (Table 1; Figure 1). The disease reactions of L30R, L30S, (L30R x L30S), L40, F_1(L30R x L40), and F_1(L30S x L40) differed from the ones presented by the set of near-isogenic lines Pa91 when inoculated with isolates 18, 47 and 49. In addition, the reactions of L30R and L30S also differed from that of line L40. This is evidence not only that the genes involved in the resistance of maize lines L30R and L40 are different from one another but also involves genes other than the Ht_1, Ht_2, Ht_3 and *HtN* genes (Table 1).

Individual analysis of each *E. turcicum* isolate showed that isolate 18 produced necrotic lesions of the susceptible type in the lines Pa91, Pa91Ht_1Ht_1, Pa91Ht_2Ht_2, Pa91Ht_3Ht_3 and Pa91*HtNHtN* but no lesions in the F_1 hybrid (L30R x L40) and chlorotic-necrotic resistant-type lesions in the lines L30R, L30S, (L30R x L30S), and L40 and F_1 hybrid (L30S x L40). The resistance reaction of the L30R, L30S and (L30R x L30S) maize lines and the susceptibility reaction of the maize Pa91 set of differentials suggests the presence of at least one resistance gene common to the three maize lines of the L30 group [L30R, L30S, and (L30R x L30S)] but different from the L10 gene introduced into the L30R maize line and the other dominant genes (Ht_1, Ht_2, Ht_3, and *HtN*) present in the maize Pa91 set. Resistance of L40 to isolate 18 in addition to the differential

Table 1 - Resistance or susceptibility reaction of maize lines and F_1 hybrids inoculated with *Exserohilum turcicum* isolates 18, 47 and 49.

	E. turcicum isolates		
Maize genotypes	18	47	49
Differential lines			
Pa91	S	S	S
Pa91Ht_1Ht_1	S	S	R
Pa91Ht_2Ht_2	S	S	S
Pa91Ht_3Ht_3	S	S	S
Pa91*HtNHtN*	S	R	R
Near-isogenic lines			
L30R	R	R	R
L30S	R	S	S
L30R x L30S	R	R	R
Line			
L40	R	R	S
F_1 simple hybrids			
(L30Rx L40)[a]	R	R	R
(L30S x L40)[b]	R	S	S
E. turcicum virulence	123N	123x	23rx

[a](L30R x L40) and [b](L30S x L40) are F_1 simple hybrids, R = resistant plants with R_1, R_2, R_3 or R_4 reactions; S = susceptible plants with S_5 or S_6 reactions (Esteves, 1989; see Legend to Figure 1 for reaction types.).
The *E. turcicum* isolates 18 and 47 came from the Brazilian state of São Paulo and isolate 49 from the Brazilian state of Paraná.

Figure 1 - Reaction of the maize Pa91, Pa91Ht_1, Pa91Ht_2, Pa91Ht_3, Pa91*HtN* near-isogenic lines (differential lines), L30R converted (L30*HtpHtpRtRt*), L30S recorrent (L30*htphtpRtRt*), (L30R x L30S) (L30*HtphtpRtRt*) and L40 (L40*htphtprtrt*) lines and of the F_1(L30R x L40) and F_1(L30S x L40) maize hybrids, according the type of lesion (Esteves, 1989) after inoculation with *Exserohilum turcicum* isolates 18, 47 and 49. Reaction type: no lesion = R_1; chlorotic points or small round chlorotic-necrotic lesions = R_2; narrow chlorotic-necrotic lesions at initial phase of development = R_3; necrotic lesions with no chlorotic halo circumscribed with a dark border at the edge = S_5; necrotic lesions with no circumscription with dried and shrunken edge of the leaves = S_6. Both susceptible plants (S_5/S_6) produce straw colored olive green necrotic lesions.

reaction of this line and the Pa91 differential lines to isolates 47 and 49 indicated the presence of a third gene, different from the resistance factor common to the L30 maize set, as well as the *Ht1*, *Ht2*, *Ht3* and *HtN* genes of the Pa91 group and the L10 gene present in maize lines L30R and (L30R x L30S). These results suggest the virulence formula *x*,*y*/*Ht1,Ht2,Ht3*,*HtN* (ineffective/effective pathogen genes, respectively) for *E. turcicum* isolate 18, where *x* corresponds to the resistance factor in the L30 maize set and *y* to the resistance factor in the L40 maize line. In this way, isolate 18 can also be classified as race 123N based on the nomenclature proposed by Leonard *et al.* (1989).

The analysis of *E. turcicum* isolate 49 showed that it produced susceptible lesions in maize lines Pa91, Pa91*Ht2Ht2*, Pa91*Ht3Ht3*, L30S, L40 and F_1(L30S x L40) and resistant lesions in lines Pa91*Ht1Ht1*, Pa91*HtNHtN*, (L30R x L30S), L30R and F_1(L30R x L40). Such behavior suggests that isolate 49 not only overcome the resistance conferred by the maize *Ht2* and *Ht3* genes but also has unknown factors capable of circumventing the resistance conferred by the *y* resistance factor in the L40 maize line and the *x* resistance factor present in the L30S maize line, although not by the resistance gene present in the (L30R x L30S) maize line or the L30R line derived from maize line L10. This behavior indicates that the L30R gene is dominant and that isolate 49 cannot circumvent the protective action of this gene. In this case, the formula *z,Ht1,HtN/Ht2,Ht3,x,y* represents the virulence spectrum of *E. turcicum* isolate 49, which could also be classified as *E. turcicum* race 23xy. It should be noted that *z* corresponds to the L30R resistance gene derived from the L10 line which has an unknown spectrum of genes conferring race-specific maize resistance against *E. turcicum*.

The last *E. turcicum* isolate used in this study, isolate 47, produced typically susceptible lesions in maize lines Pa91, Pa91*Ht1Ht1*, Pa91*Ht2Ht2* and Pa91*Ht3Ht3*, L30S and F_1(L30S x L40) and resistant lesions in lines Pa91*HtNHtN*, L30R, (L30R x L30S), L40 and F_1(L30R x L40), indicating its ability to overcome the resistance conferred by the *Ht1, Ht2, Ht3* genes and the *x* resistance factor but not the resistance conferred by either *HtN* or the *z* and *y* resistance factors (Figure 1). Based on these considerations, isolate 47 can be classified as race 123x and the its virulence formula represented by *z, y, HN*/*Ht1*, *Ht2*, *Ht3*, *x*. The presence of susceptible lesions in L30S and resistant lesions in L30R and (L30R x L30S) further suggests the dominance of the L10 resistance gene and also indicates the inefficiency of the *x* resistance factor common to the L30R/(L30R x L30S)/L30S set of maize lines.

The presumed involvement of a resistance gene in maize line L40 different to the *z* resistance factor in maize line L30R, the *Ht1, Ht2, Ht3,* and *HtN* genes from the Pa91 set and the *x* resistance factor common to group L30 is further supported by analysis of segregation of disease reaction types resulting from the inoculation of *E. turcicum* isolate 47 onto the BC_1F_1 plants. Based on the susceptible reactions of L30S and F_1(L30S x L40), on the resistance reactions of L30R, (L30R x L30S), L40, F_1 (L30R x L40), and the segregation ratios of resistant and susceptible plants in the BC_1F_1 population (106 resistant and 32 susceptible) it was not possible to infer about the genetic constitution of the *x* resistance factor common to the L30 group. However, it was possible to infer the presence of a single recessive gene, corresponding to the *y* resistance factor, and named by us as *rt* gene, in the L40 maize line and a single dominant gene in the L30R maize line, corresponding to the *z* resistance factor and named by us the *HtP* gene. Thus although the race classification of *E. turcicum* isolates 47 and 49 has remained the same, their virulence spectrums are better represented by the formulas *HtP, HtN*, *rt*/*Ht1*, *Ht2*, *Ht3*, *x* (*E. turcicum* race 123x) and *HtP, Ht1*, *HN*/*Ht2, Ht3*, *rt*, *x* (*E. turcicum* race 23rx), respectively.

This genetic hypothesis presupposes that maize line L30R is homozygous for the *HtP* and *Rt* genes (L30*HtPHtPRtRt*), so that the joint effect results in the resistance reaction observed with isolate 47. The segregation observed in BC_1F_1 plants suggests that maize line L40 carries contrasting genes to those present in maize line L30*HtPHtPRtRt* for the two loci. In this case, the resistance of line L40 to *E. turcicum* isolate 47 would come from the expression of the *rtrt* genotype (L40*htphtprtrt*) since the *htphtp* condition conferred susceptibility to the same *E. turcicum* isolate. Based on these considerations the BC_1F_1 plants [(L30*HtPHtPRtRt* x L40*htphtprtrt*) x L40*htphtprtrt*] can have four different genotypes (*HtPhtpRtrt* : *HtPhtprtrt* : *htphtprtrt* : *htphtpRtrt*) with each genotype representing 1/4 of the total possible genotypes, thus fitting a hypothetical model where the *HtP* and *rt* genes segregate independently. The χ^2 test applied to the expected proportions of 3/4 resistant (*HtPhtpRtr* : *HtPhtprtrt* : *htphtprtrt*) to 1/4 susceptible (*htphtpRtrt*) was not significant, indicating that the deviations between the expected and observed frequencies did not justify the rejection of the hypothesis of independent segregation of *HtP* and *rt*.

Carson and Wicks (1993) have already identified one effective maize recessive resistance gene against *E. turcicum* races 0, 1, 23 and 23N located close to the centromere on the short arm of maize chromosome 1 (Carson, 1995). Our data indicates that, similar to Carson and Wicks (1993), the maize *rt* resistance factor described above provides protection against the *E. turcicum* virulence factors 1, 2, 3 and N present in isolate 18 (race 123N). Furthermore, the *rt* gene conferred resistance to the factor or factors present in *E. turcicum* isolate 47 (race 123x) and susceptibility to the corresponding r factor present in *E. turcicum* isolate 49 (race 23rx). Even though the expression of the resistance conferred by the gene identified by Carson and Wicks (1993) was different from the resistance presented by the *rt* gene, additional co-segregation analyses should still be carried out in order to confirm the identity of

the *rt* resistance factor as a new maize gene with recessive resistance.

The analysis of the evolution of parasitism involves knowledge of the population dynamic of the pathogen through time and space and such knowledge allows the identification of changes in pathogen virulence and the appearance of new races that could be produced by new mutations on avirulence genes and/or new combinations between preexisting virulence genes. Although our data could suggest evolution of *E. turcicum* races it was not possible to analyze population dynamics of the *E. turcicum* races through time and space. To answer such questions further studies are needed with *E. turcicum* strains from different regions of Brazil.

The identification of *E. turcicum* race 23rx and *E. turcicum* race 123x carrying two unknown virulence factors (*r* and/or *x*) is an important finding for defining genetic breeding strategies for maize resistance to helminthosporiosis. Our data demonstrates that the presence of the dominant *HtP* gene is capable of conferring wide resistance to *E. turcicum* races such as 123x and 23rx that combine multiple virulence factors. Thus, maize line L30R represents a good source of resistance to this pathogen.

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